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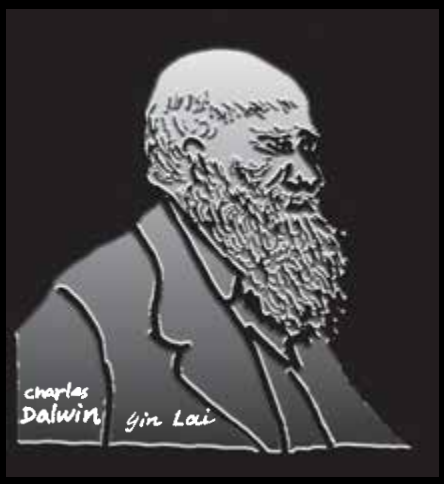
The Journal of Southern California CLINICIANS



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Our Mission and Purpose

Southern California Clinicians is established by the Medical Staff of Pomona Valley Hospital Medical Center in Pomona, California. It provides a journal for modern California clinicians to publish articles to share their clinical experiences and opinions with other physicians, show their academic achievements in medical practice, and keep a permanent record of valuable case studies and case reports from all departments and all specialities in the modern era.

This journal invites all clinicians in southern California to contribute interesting articles and reviews, including new developments in clinical skills and techniques, or new procedures applied during their medical practice.

In order to maintain the highest quality, accuracy and academic dignity, we reserve the right to peer review all articles. Articles will be reviewed by our editorial board and special consultants.

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Guidelines for Authors

On behalf of the editorial staff of PVHMC's new medical journal *Southern California Clinicians*, I would like to extend an invitation to you to contribute articles for publication. Articles that pertain to your medical practice, any case reports you may have, or past clinical experiences are welcome. Articles for publication in the next journal are due no later than April 30th, 2012.

1) Use a single page to show your full name, your academic degrees and affiliations, and your current address, phone number, fax, e-mail.
2) All articles must be titled.
3) Please write your article with Microsoft Office Words, double spaced and save to a CD.
4) Length is flexible, from 1 page to 10 pages.
5) You may include a short abstract and conclusion as you wish. Slides, tables, figures, photos or pictures are welcome. Most important is a list of references numbered in the order in which you marked in the text.
6) All articles have to be original, never been published before, reflecting your own experience, knowledge and opinion.
7) All articles, once accepted, will be peer reviewed, corrected or revised and will be sent back to you for your approval.
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Preface for this Edition

Yin H. Lai, M.D.
Editor-in-Chief

ECONOMY, LAW AND MEDICINE

As the national economy becomes so poor today, while the medical care expenses keep rising, the Obama administration has no choice other than to use law makers to launch a health care reform.

One of the main issue is the law's mandate on Americans to carry health insurance. On August 12, 2011, at the U.S. Court of Appeals for the 11th Circuit in Atlanta, the majority agreed with two lower courts that said Congress overreached when it required most Americans to carry insurance or pay a penalty. The ruling was made by Judge Frank Hull (appointed by Bill Clinton) and Judge Joel Dubina (appointed by George W. Bush). Judge Hull has found part of the patient Protection and Affordable Care Act unconstitutional.

However, the Atlanta appellate judges said other provisions, except for individual mandate part, should remain "legally operative." That means the law will leave in place except for the mandate. That also suggests the entire health insurance market will be thrown into turmoil. Why? By the new law, all health insurers are required to accept all prospective customers, even those who are already ill. These insurers are willing to accept the provision only if they were guaranteed an influx of millions of new customers through the coverage mandate.

By deleting the mandate, the law could encourage people to wait until they are so sick to acquire coverage.

We have two more federal appellate courts to rule on the health law: (1) The Fourth Circuit Court in Richmond, Va. (2) D.C. Court of Appeal (in September). Then, the Supreme Court will put the final resolution, possibly after November 2012 president election.

What do all these mean to us? Nothing! A doctor is a doctor. Yes, we are working like an engineer, a carpenter, a healer. But we have one thing the other professions do not have: a "loving heart." We take care of our patients with a loving heart. No matter what the law will change, we shall never lose our "loving hearts".

With this loving heart, we love to take care of sick people. We also love to meet each other in the hospital to discuss new, advanced skills in patient care. The hospitals also like to work with doctors in harmony. They provide a comfortable facility for patients and doctors. They provide CME for doctors to improve medical knowledge. A smart administrator always knows what the doctors and the patients need. He also understands how to improve the reputation of his hospital.

Even with poor economy, I am still experiencing a hearty support to this medical journal from all nearby hospitals, medical teaching facilities, doctors and even pharmacies. This medical journal provides new knowledge, introduces all clinicians to know each other and create a harmony in medical community. It increases doctor's writing skills. It also raises the academic level of all hospitals since they own so many excellent medical staffs who can write these creative and high quality medical articles to be published in this one and only medical journal. The entire editorial board of "Southern California Clinicians" thanks to all of you as supporters!

–Yin H. Lai
Editor

THE DEVELOPMENT AND IMPLEMENTATION OF CHI:
An Overview

Dr. Gary Selnow and Dr. Suellen Crano

Received on 4/6/2011

The Problem

Access to health information and other tools of disease prevention is a basic human right. Remarkably, prevention is often overlooked, perhaps because it isn't glamorous or profitable. And yet the need for life-saving information is compelling: health knowledge can save billions of dollars, millions of lives, and lifetimes of anguish.

Giving people knowledge about healthy practices, teaching them about harmful conditions, and showing them signs and symptoms of illness and disease can go a long way to improve health and avoid or reduce the need for costly treatments.

Several years ago Dr. Lee Jong-wook, then Director-General of the World Health Organization (WHO), reflected on the grievous outcomes of unequal access to medical care around the world. We agree with Dr. Jong-wook's observations and add that developing and medically underserved populations also face unequal access to information that enables them to understand and contribute to their own health. Educating communities to safeguard and improve their health is efficient and effective. WiRED International has dedicated its organizational resources to improving health information equity in war-torn and developing regions.

WiRED International is a non-profit organization in operation for more than 10 years. This volunteer-driven organization focuses on health information and education for medical professionals and grassroots populations in developing countries, using information technology (IT). WiRED, acting alone and in collaboration with U.S.-based medical schools, has provided medical information and training in Iraq, the Balkans, Central America, and Africa.

WiRED's work on community health began under an NIH developmental grant in Kenya. That program has led to the creation of a comprehensive e-library of interactive, training tools for grassroots populations. The organization holds that community health starts with knowledge, and its goal is to provide information and training that fortify community health knowledge. WiRED has been funded by the National Institutes of Health, the U.S. Department of State, the Medtronic Foundation, Pfizer, Rotary International, and a number of family foundations. WiRED is the recipient of the UC Berkeley School of Public Health 2009 Organizational Public Health Hero Award.

The Community Health Information (CHI) Program

The CHI program provides community health training and illness prevention for grassroots communities. It develops and maintains interactive training libraries designed for people with no medical background.

WiRED defines health broadly. In addition to a standard array of health topics on communicable and non-communicable illnesses, WiRED includes women's health, family planning, diabetes, cancer, and a range of environmental health issues. These include programs on clean water, low-fuel cooking methods, food safety, sanitation, and hygiene. In addition, WiRED offers targeted programs for caregivers, community health workers, students, and patients.

The community training programs are disseminated through several mechanisms. The most comprehensive of them–the turn-key package–includes the purchase and set-up of computers, the installation of complete health libraries and training tools, staff training and ongoing management of the facility. In all cases, WiRED enters into collaborative arrangements with local community-based organizations, clinics, schools, and other established institutions in the operation of the CHIC facility.

The organization's primary interest is to distribute the training programs as widely as possible to grassroots populations, and this does not require a CHIC infrastructure. Other non-government organizations and institutions have computers and need only professionally developed health content. Consequently, WiRED is making its complete CHI e-libraries available to any non-profit organization that serves under-resourced populations. Such interactive, health training programs are available through commercial sources, but they cost tens of thousands of dollars or more—far beyond the budgets of most small non-profits. Driven by the belief that health information is a human right, WiRED will charge little or nothing for its material in order to make it as widely available as possible. WiRED is attempting to fund this activity with support from donors interested in community and global health.

Health information, such as provided by the CHI program, can save billions of dollars in treatment costs globally when ordinary people understand and act on prevention and basic health matters. At present, WiRED works only in developing countries; however, it is preparing to distribute this material within the poorest regions of the United States. This will be its first venture into the domestic arena.

HIS HANDS ARE ALMOST ALWAYS RIGHT!

By Suellen L. Crano, PhD.

Suellen Crano, Ph.D.
After earning her Ph.D. in Higher Education Administration/Student Personnel in 1984 from Michigan State University, Dr. Crano served in administrative roles at Texas A&M University, The George Washington University, The University of Arizona, the State of Michigan Department of Higher Management Systems, Wyeth Laboratories, Inc., and most recently, at Western University of Health Sciences. She is a member of the Boards of WiRED International, the Arizona Cancer Center, and the College of Graduate Nursing at Western University of Health Sciences. Not satisfied by underwriting WiRED's VideoVisit program, which enables families to communicate with their children undergoing life-saving medical treatment in western countries, Dr. Crano helped coordinate the launch from a children's cancer ward in the Hospital of Pisa. Her role as a WiRED director enables her to continue a lifelong passion of helping people rebuild devastated lives. She is actively involved in developing materials and relationships for WiRED's educational programs and the International Telemedicine Network. In addition, since becoming a cancer patient herself, Dr. Crano has become an advocate for people with cancer who need assistance.

In the spring of 1999, I arrived at my G.P.s office with a terrible earache. I had only recently moved to southern California and had limited contact with Dr. Right , who had come to southern California from Taiwan. My insurance company assigned me to him and I knew little about him, other than he took his time with his patients and if you weren't the first patient in the morning, you wound up waiting for a long time.

Dr. Right examined me thoroughly; asked me many lifestyle questions, especially about diet, exercise, and environment; and then prescribed antibiotics, decongestants, and asked if there was anything else I needed. I told him I needed a referral to a gynecologist as it was about time for my annual physical. Dr. Right knew I was a Vice President of a medical university and very busy as he was involved with our faculty at the nearby hospital. He told me that in Taiwan, he trained as an Ob-Gyn, but when he came to the U.S., his family made the decision for him to become a family practice physician. Dr. Right offered to do the pelvic exam right then and there to save me the hassle of another doctor's visit. I took him up on his kind offer.

Dr. Right's medical assistant came into the room as he readied the stirrups and the necessities for the pelvic exam. He poked, prodded, grunted and groaned, and then looked at me and said, "Something is very unusual and these hands are almost always right! I feel a mass."

Dr. Right was humble but convinced that something was untoward. His assistant and I looked at each other and then back at him. He started writing orders for a pelvic ultrasound and various blood tests, including a CA-125. Given his concern, I rushed to complete the tests.

Long story short, after drinking what seemed like gallons of water and nearly wetting the exam table, the ultrasonographer looked at me as if I were crazy and said the doctor must have mistaken some bowel matter for a mass. They sent me home and the report came back negative. Dr. Right did not believe it—they are wrong. The CA-125 results came back at 42—high normal, but normal as such. Dr. Right looked at me at the follow up exam and said, "These Taiwanese hands are usually right! I will follow you every three months with a CA-125 and other imaging."

A few months later, I was living temporarily in northern California and discovered a breast lump. I went immediately to a Gynecologist to check it out and when I saw a diploma from a reputable U.S. medical university on the wall, I told him about Dr. Right's hands always being right and the results that I had received. I asked if he would examine me as well. After completing his exam, he called in his PA to do the same.

They both shook their heads and she looked at me and said, "Something feels unusual." Next stop was pelvic

and vaginal ultrasound. Again, I got a negative result. They called me back and told me not to worry. These tests are always right. I must just be different. Dr. Right was now Dr. Wrong…or was he?

I sent the results to Dr. Right, and at the end of the third month, he told me to have another CA-125 test. The results came back at 55. Dr. Right sent me for a CT-scan based on those results. The CT came back normal. Dr. Right was not impressed. He examined me again and looked me in the eye and said, "I know something is wrong. These hands are usually right. We are not going to stop until we figure this out." And he didn't stop. Every few months, my CA-125 went up steadily and every type of imaging showed nothing wrong, just ovarian cysts that changed sides depending on the month of the exam.

Finally, when my CA-125 was over 100, Dr. Right suggested I contact a former colleague and friend in another state who was a famous oncologist to get his opinion. My colleague was alarmed about the CA-125 and sent me to a renowned gynecological oncologist at a well-known cancer center in southern California. I was seen immediately, and after considerable imaging, which showed nothing unusual, the verdict was that all measures have variability and unreliability, but nothing seemed untoward at the time. The prescription was to continue with regular follow-ups and wait and see.

Fast forward to January, 2001. My CA-125 was in the mid 100's, and for the first time ever, my CT-scan showed a bit of ascites, in addition to a small cyst this time on my right ovary, but nothing else. Dr. Right said, "See—my hands are usually right. Hurry and go to the cancer center." I followed Dr. Rights's advice and was scheduled for an exploratory laparoscopy. I was assured that it all seemed pretty benign, as I was physically healthy. I had just spent the previous two weeks skiing with my 17-year old son. I couldn't have done that if I were ill. There were no symptoms, no pain, no weight loss, no change in bowel habits, no night sweats, nothing. The surgeon said that there was probably just a bit of infection on the cyst. I was told that if there was something else, I would be cut through the midline and would be given a complete hysterectomy, oophorectomy, and whatever else was necessary. I signed the requisite release forms.

I woke up many hours later in the recovery room. I only felt a twinge of pain on my right side—no apparent midline incision. I asked the recovery room nurse what the outcome was. She told me the surgeon would be in soon to give me the report. I said that it felt like I had only my right ovary removed and she confirmed that. I said to myself, "YES!" and felt a smile come over my entire body. I had secretly been in great fear as my family was riddled with cancer, but primarily colon and breast cancer. Maybe Dr. Right was over-reacting. Maybe Dr. Right's hands were, in fact, sometimes wrong?

Or, maybe not? The doors of the recovery room burst open. My son, who had been waiting impatiently, came rushing toward me. His face said it all. I looked at him and said, "I have cancer don't I?" He burst into tears, hugging me like I had never been hugged before. Nurses came flying in after him, trying to get him out of the area, to no avail. The surgeon and my husband arrived. I had never seen three men look so grim. My morphine-clouded brain worked hard to comprehend what I was being told. Even with the hallucinogenic effects of the morphine, I could comprehend that it wasn't just an infected cyst.

I had stage 3B metastatic cancer. It was all over my abdomen and pelvis, from the hemidiaphragm on and around all of my organs. The cancer seedlings were so small and diffuse they did not show up on imaging. I was going to have 6 rounds of chemotherapy, both intravenous and intraperitoneal. Then, I would have surgery to remove what was left and continue with chemotherapy. The prognosis was poor, but I was strong, young, and had a strong will to live. Dr. Rights's hands did not lie. He was right. Something was very wrong.

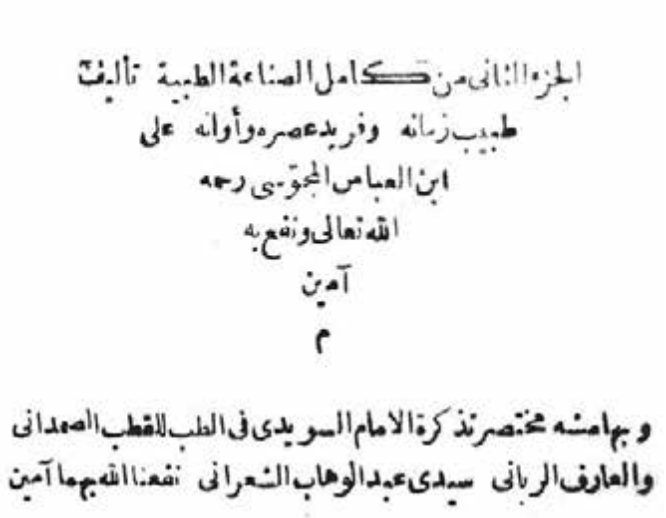
Had Dr. Right not been so convinced of his clinical exam findings and been so tenacious about following up with me, I would not be here today to tell this story. I also would not be here had I not had the best gynecologic oncology surgeon on earth and a colleague in Arizona who was the tops in gynecologic pharmacology. I was diagnosed with diffuse Peritoneal Mesothelioma, generally a death sentence with a life expectancy of 6-18 months from time of diagnosis. Luckily for me, Dr. Rights's magic hands, his acute diagnostic tool, were there for me. Being diagnosed so early in the progression of the disease has led to excellent outcomes. As this article is being published, I am celebrating my tenth anniversary year with mesothelioma. Thank you Dr. Right and your hands that do not lie.

Moral of the story: Believe in and follow your clinical judgments.

MANAGEMENT OF THE MANIFESTATIONS IN CHILDREN (PART I OF II)

Eugene L. Mahmoud, MD — Staff Neonatologist; Pomona Valley Hospital Medical Center; Pomona, California

Hikmat Sous, RN — Staff NICU Nurse; Pomona Valley Hospital Medical Center, Pomona, California


الجزء الثانى من كامل الصناعة الطبية تأليف
طبيب زمانه وفريد عصره وأوانه على
ابن العباس المجوسى رحمه
الله تعالى ونفع به
آمين
م
وبهامشه مختصر تذكرة الامام السويدى فى الطب للقطب الصعدانى
والعارف الربانى سيدى عبد الوهاب الشعرانى نفعنا الله بهما آمين

Kamaal Al Sanaa't Al Tibbiyat (The Complete Art of Medicine of Medicine/The Royal Book) was produced by Ali Ibn Al Abbaas Al Majoosi (Haly Abbas) in the year 980 AD.

Introduction

The book was produced to answer the need for a less voluminous, yet concise practical encyclopedia of the Practice of Medicine. Even with incomplete Latin translation, it was adapted as Liber Pantegni by Constantinus Africanus, which became a founding text of the Medical School in Salerno, Italy. After translated into Latin, it was widely used in many parts of Europe. Since there has never been a complete translation into the Modern English Language of this work, it is our delight to examine the English Translation of Chapter 20, Management of manifestations in children, from Book One of the two volume work.

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Twentieth Chapter: The management of manifestations in children

Once the baby is born, the baby should be sprinkled on the skin with salt and crushed roses to strengthen the skin against the air, when the skin of the baby is still with plenty of moisture. Thereafter, by using one of the baby's finger to dip in the honey and to rub honey to the baby's palate. Then the baby's ears, are well sucked.

Then the baby is fed with smoothly crushed sugar with sesame seed oil for two days. The skin is rubbed with sesame seed oil on separate parts morning and evening. And separated parts of skin are stretched out to meet the joints of baby's feet and hands, while the crushed myrtle and the roses are placed on all the joints. Also like that between the thighs. Afterwards stretched out his hands and his feet while swaddled, swaddling well. And if the head scaling or it has plenty swelling was eased and let be put underneath a solid object, be it fear or prayer, whether that charitable place of arrival with rag. Perhaps it pains. And bind to forehead with the binding, while tightened a little bit. Every two to three days the baby is washed with fresh lukewarm water cooked with myrtle and roses. And ears are sucked at the time of washing to get water out of them.

While covers its face and put it to sleep together used with them gentle movement. With companion

also composed for them good melodies. So gave it delightful good songs which are softer just as the perfectly delightful. In doing so was human propensity for love of movement and love of melodies. Then calm overtakes it

Page 53

was found from embrace and brings to it sleep. And not slept in bright place because tender eyes of the child are weak and bright light dispersed. But gathered canopy light and strengthens sight.

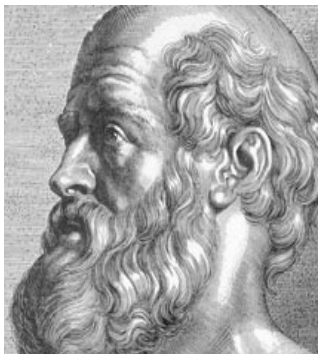
And if the newborn is a baby boy, his skin will be rubbed with liniment strongly for four months because much oiled skin will become firm and strong. It is believed that a baby body needs more oil rubbing than a baby girl will get. If it is a baby girl, then her skin will be rubbed softly with violet oil for two months. Afterwards, it was cut off because a little gentle rubbing with oil will moisten the body while too much forced rubbing will make her skin coarse.

At times one should inspect the baby's cries and search what harms them by guess and sought protection from persons who are already trained in bringing up the children. A baby does not cry, unless things will harm it, because it is not able to complain. A baby gets hurt either from outside or from inside. Any hurt from the outside can be due to the heat and cold. Whether flies or bugs and anything similar to it, those causes should be cleared. Any hurt from inside can be due to hunger and thirsty, or retention of urine and feces. Unless because of pain in some of the separate parts. As far as the hunger and thirsty is concerned, the baby should be provided with plenty of food, milk and water. As for urine retention, the baby should be given pulp of the watermelon seed assembled in the julep. If the baby gets wet, the nurse should bathe the baby in warm water instead of humbling the baby in hot water. Then the skin should be rubbed with oil of gillyflower or iris. And as for withholding of nature, then should be carried the healing of defecation gushed forth whether easy to bear matter from foreheads seen, until the greatest edible plants work as emptying and dropping. Or from the salt marshmallow. And fed the wet-nurse vegetables with pure olive oil softening the belly. While opposed with vinegar and dried figs and pears accompanied the pulp of watermelon.

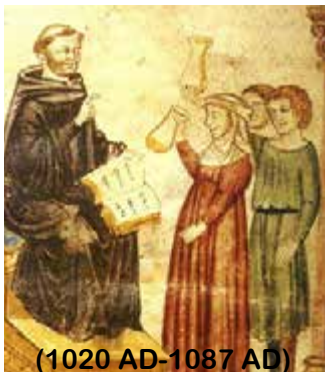
For the illness presented in some of the organs, one should try to treat first with its antidote and continue to observe any symptoms which is characteristic of organ specific illness. Some illness have already been mentioned by Hippocrates in "The Seasons", where he said that children can easily expose to thrush, vomiting, sleeplessness, fears, inflammation of navel, and the moisture of the ears. And as soon as the time of their teeth growth, swelling took place in the gums and went away with fevers and spasms. And in different occasions especially as characteristic teeth grow, children may be exposed to inflammation of the throat, itch in the ears, and conjunctivitis.

And convulsions may occur together with illness of boys. One should also examine the abdomen and other part of body pending on the illness and symptoms.

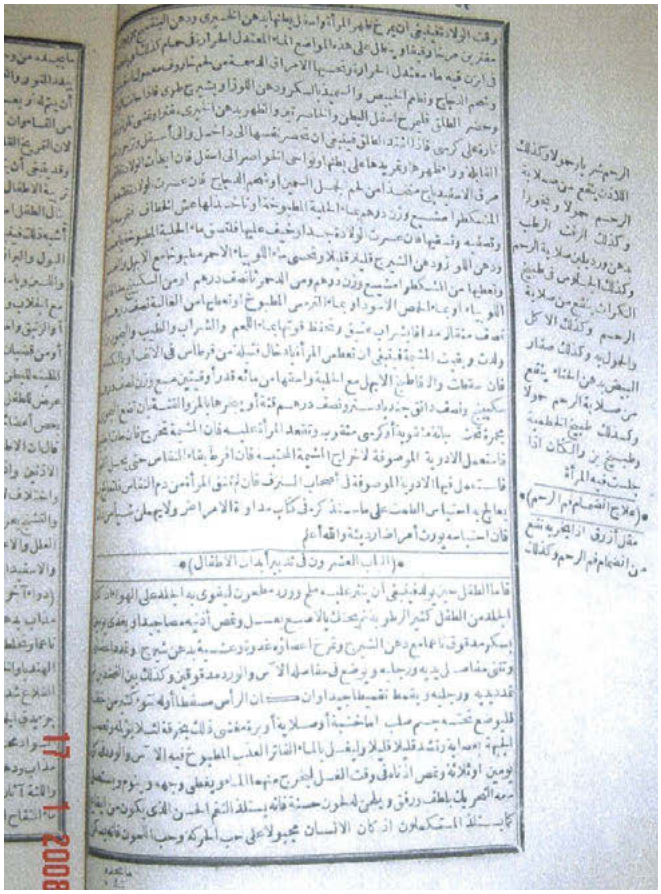
'Ali Ibn Al Abbas Al Majusi



Hippocrates



Constantine the African



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VAGINAL BLEEDING IN A PATIENT WITH INFLAMMATORY BREAST CARCINOMA

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Abstract

Metastatic carcinoma involving the uterus and cervix is relatively rare, presenting clinically with vaginal bleeding and most often originating from the breast. We present a case of a 42 year old female with advanced inflammatory carcinoma of the breast on chemotherapy, metastatic to the endometrium, myometrium, cervix, ovaries and fallopian tubes. The hysterectomy specimen consisted of a uterus including the cervix, fallopian tubes, and ovaries with no grossly discernable abnormalities.The histopathologic sections revealed only isolated malignant neoplastic cells haphazardly sprinkled throughout the female reproductive system. The gross and microscopic morphologic features in patients with metastatic breast carcinoma involving the uterus and adnexal structures can be very deceptive requiring careful review of multiple sections and immunohistochemical studies for confirmation. Endocervical glandular neoplastic or dysplastic changes, found in our case, may accompany the metastatic carcinoma and can additionally complicate or confound the clinicopathologic presentation in arriving at or rendering an appropriate diagnosis. Immunohistochemical staining is essential for definitive diagnostic evaluation.

Case Presentation

A 42-year-old gravida 5 para 5 female presented to her primary care physician with the chief complaint of daily vaginal bleeding over the previous several months. Her past medical history included a inflammatory left breast carcinoma diagnosed three years earlier. She had undergone left breast mastectomy and received chemotherapy, which consisted of trastuzumab, doxorubicin, cyclophosphamide, and paclitaxel. After completion of chemotherapy, she was given hormonal therapy. Five months prior to the current presentation,

a dilatation and curettage was performed because of vaginal bleeding which revealed a poorly differentiated adenocarcinoma, partially infiltrating and replacing the endometrial stroma.

A PET scan, performed two months earlier, revealed focal areas of hyperbolic activity in the uterus and in the region of the cervix. There was an interval increase in size and intensity of these findings from those studied one year ago suggestive of interval progression of neoplastic pathology. There were new hyperbolic pathologic findings in the right femur, the right liver, and the left axilla consistent with metastatic lymphadenophy. At the time, she continued to receive monthly trastuzumab.

Preoperative pelvic exam revealed a normal vulva, perineum, & perianal area. The vagina and rectum were normal. The cervix and uterus were slightly enlarged. The parametria were free. There was no ascites or palpable abdominal or pelvic masses. The patient was concerned about the disability from the severe daily bleeding, and surgical consultation was requested. The patient underwent total abdominal hysterectomy with bilateral salpingooopherectomy.

Pathology

The hysterectomy specimen consisted of a uterus with attached cervix, both fallopian tubes and ovaries. The uterus weighed 150 g and appeared grossly essentially normal (figures 1 and 2). It was 9.0 cm long and 7.0 x 5.0 cm in diameter across a smooth pink white symmetrical fundus. The uterine cavity was lined by relatively smooth to slightly irregularly bosselated pink white mucosa. The endometrium varied from 0.1 to 0.2 cm in thickness. The myometrium measured 2.5 cm in thickness. The right and left fallopian tubes measured 4.0 x 0.7 cm and 4.5 x 0.7 cm, respectively with no grossly discernible lesions. The right and left ovaries measured 3.5 x 2.0 x 1.0 cm and 4.5 x 2.0 x 1.5 cm in greatest dimensions respectively. A specimen labeled as "right peri-metrial lymph node" was also received.

Histopathologic sections of the cervix, endometrium, myometrium, and the adnexal structures revealed variable numbers of isolated large malignant neoplastic cells, exhibiting round hyperchromatic nuclei with prominent nucleoli scattered deceptively throughout the stromal components of virtually all the specimens evaluated (figures 3-10). The endocervical mucosa was covered by variably atypical to dysplastic columnar epithelial cells exhibiting some loss of polarity, nuclear hyperchromasia, high nuclear cytoplasmic ratio, and occasional mitoses (figure 3 and 4). Sections of the endometrium consist of oval to slightly elongated glands lined by relatively orderly pseudostratified columnar epithelium. Sections of the tissue specimen labeled as "right peri-metrial lymph node" consisted of portions of lobular adipose tissue that showed patchy infiltration of the fibrovascular stroma by malignant neoplastic cells cytologically similar to those described previously (figure 11). There was no evidence of lymphatic tissue in the specimen.

The neoplastic cells infiltrating the stroma showed immunoreactivity with CK7, mammoglobin (figure 12) and GCDFP-15. There was focal positivity with CEA, few cells with S100, and rare cells with CA125 .The hormone receptor markers ER and PR were negative. The neoplastic cells were also negative with: CK20, TTF-1, WT-1, CDX2, VIM, and HMBE-1.

Discussion

Metastasis to the female genital tract from an extragenital neoplasm is uncommon. The most common site for metastasis is the ovary (1), while the uterus accounts for less than 10% of all cases of metastases to the female genital tract from extragenital cancers (2). Out of 198 cases of metastasis to the uterus from extragenital cancers, breast carcinoma was the most common (56.6%) origin followed by gastric (16.7%) (3). In terms of anatomical distribution, a review of 52 cases showed that metastasis to the myometrium was the most frequently involved (96.2%), and involvement solely of endometrium

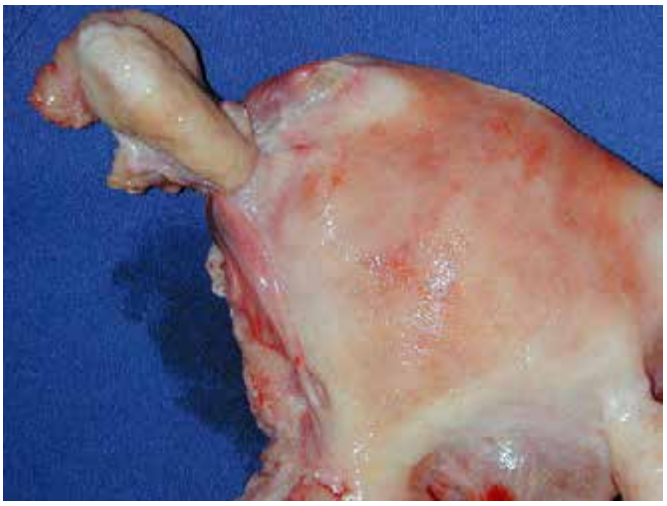

Figure 1: Essentially normal uterus on gross inspection

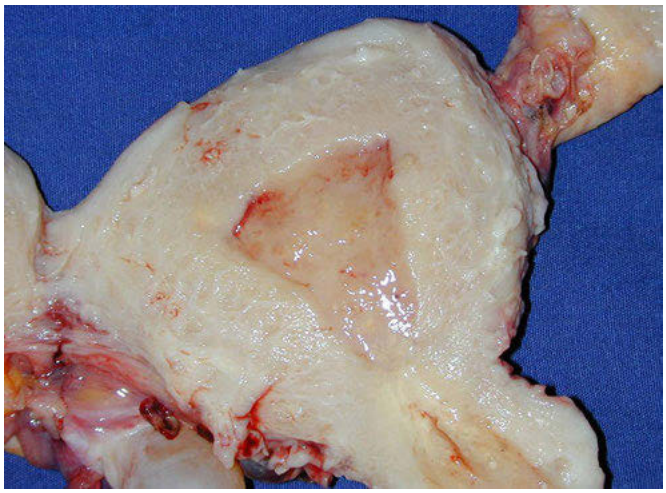

Figure 2: Cut-surface of the uterus with no grossly discernible lesions.

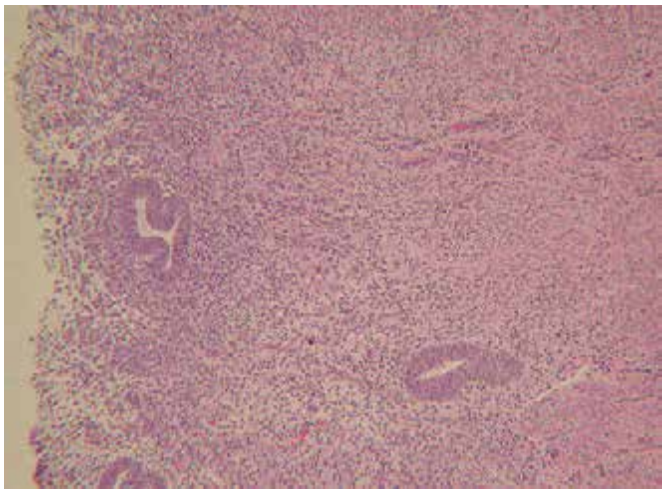

Figure 3: Endometrial at low magnification appearing essentially normal.

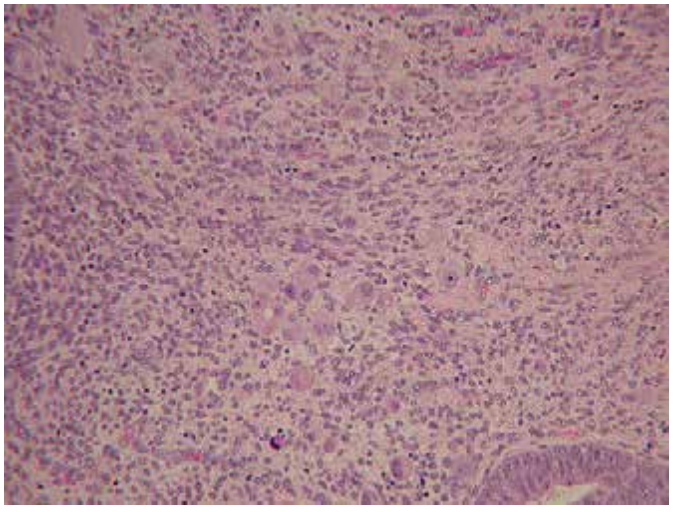

Figure 4: Endometrium at higher magnification revealing atypical cells within the stroma

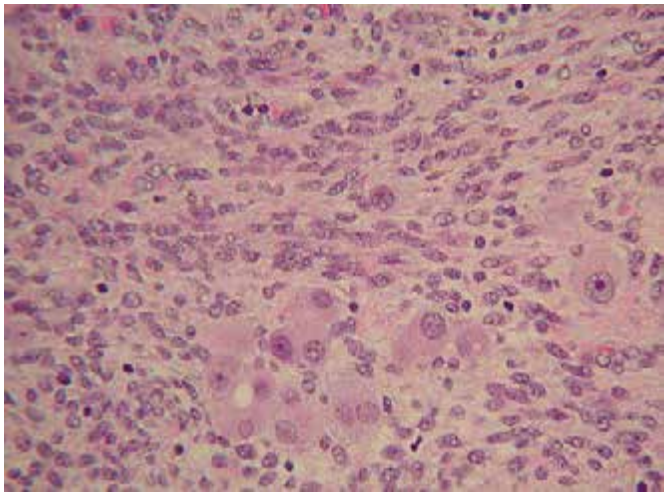

Figure 5: isolated malignant cells infiltrating the endometrium.

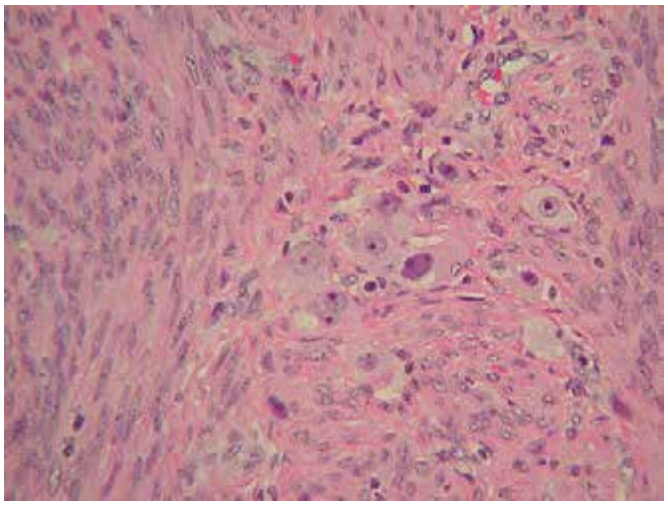

Figure 6: isolated malignant cells infiltrating the myometrium.

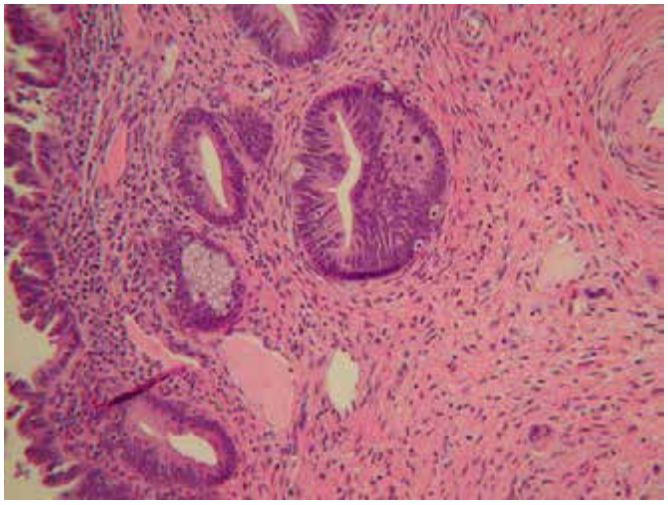

Figure 7: Endocervical mucosa with dysplastic glands and isolated malignant cells on the right lower corner within the stroma.

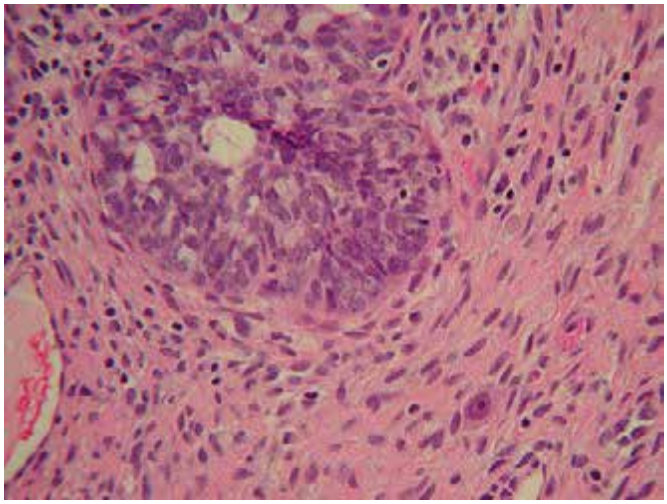

Figure 8: A dysplastic endocervical gland and isolated malignant cells on the right lower corner within the stroma.

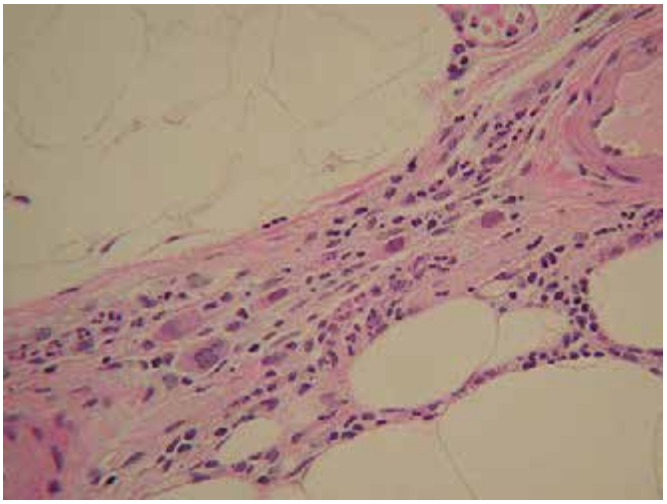

Figure 11: isolated malignant cells within the mesenteric fibrous stroma.

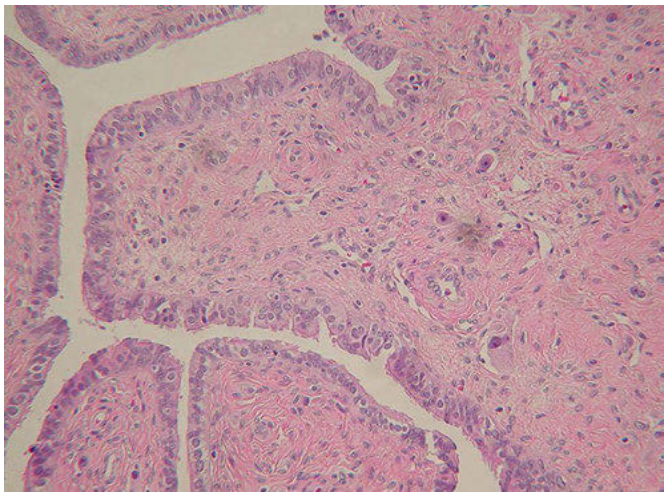

Figure 9: fallopian tube mucosa with isolated malignant cells within the stroma.

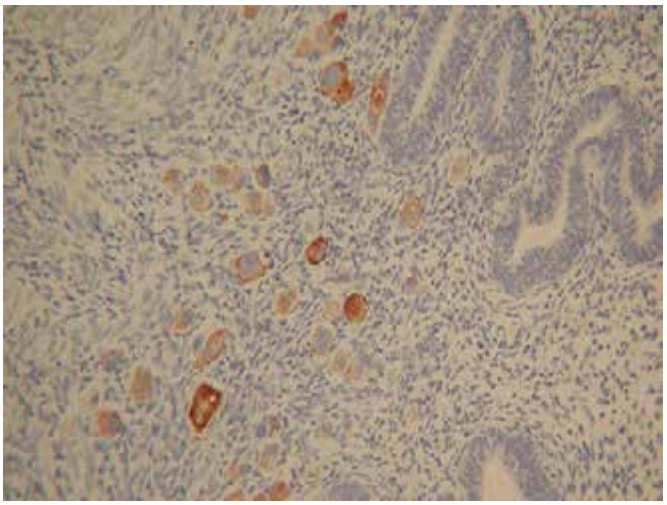

Figure 12: immunohistochemical stain reactivity of malignant cells with mammoglobin.

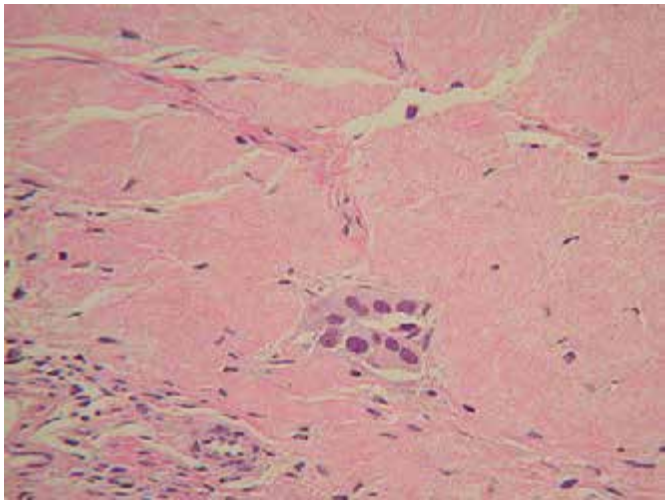

Figure 10 ovarian stroma with an isolated malignant gland within corpus albicans.

was very uncommon (3.8%) (4). Among breast carcinoma, lobular type is the most common to metastasize to the uterus (5). Although the most common first sign was vaginal bleeding, as in our case, asymptomatic cases have also been reported (6,7)

Primary inflammatory breast cancer (IBC) accounts for 0.5 to 2 percent of invasive breast cancers, though its incidence has increased (8). Interestingly, even though less than five percent of breast cancers diagnosed by mammography screen are stage III tumors, locally advanced breast cancer represents thirty to fifty percent of newly diagnosed breast cancers in medically underserved populations in the United States and in other countries (9,10). Primary IBC has the following biological characteristics: 1) rapidly progressive, 2) highly angiogenic and angioinvasive, which account for high metastatic potential, 3) the aggressive behavior and

angiogenicity are intrinsic to the tumor (11). Due to these characteristics, primary IBC is rapidly progressive and has a high propensity for early metastatic spread with the lungs, bone, and liver being the most common sites for metastasis. The majority of IBCs are hormone receptor-negative (12, 13) and approximately sixty percent of IBCs overexpress the epidermal growth factor receptor (EGFR) and the type EGFR, also called HER2 (14), which this patient's breast cancer histology followed.

More recent therapies against HER2 provide another therapeutic option, either as monotherapy or in combination with endocrine therapy or chemotherapy. Specifically, trastuzumab is a humanized monoclonal antibody (MoAb) that binds to a specific epitope

of the HER2 protein on the cell surface, which causes inhibition of signal transduction induced by peptide growth factors and their associated receptors. Overall this causes cellular growth inhibition (15-17).

To our knowledge, there has not been a reported case of uterine metastasis in patients with inflammatory breast cancer receiving Herceptin therapy. There are several cases with reported uterine metastasis from patients on tamoxifen (1,3, 24-26). Tamoxifen, useful for its anti-estrogenic properties for breast cancers, is also a weak estrogenic agonist and has been shown to produce histological changes on the endometrium including polypoid lesions, hyperplasia, and adenocarcinoma (18, 19). Because the patient had not received tamoxifen (?) and was on monthly Herceptin at the time of vaginal bleeding, metastasis to the uterus from breast carcinoma was highly suspected.

The immunohistochemical profile of the uterus in this case was reactive to GCDFP-15, as reported in other cases (1,3). GCDFP-15 is positive in 65 to 80 percent of breast cancer cases and is rarely positive in skin adenexal tumors, endometrial cancers, and salivary gland tumors (20-22). Another marker, mammaglobin, was reactive as well and though it is more sensitive, it has less specificity (20). Generally both GCDFP-15 and mammoglobin are used together. CEA is a sensitive marker for adenocarcinomas of the breast, lung, and gastrointestinal tract, but cannot distinguish origin site. Expression of cytokeratins (CK) can assist with differentiation. The presence of CK7 and absence of CK20 suggests breast cancer (20), as was seen in this case.

Patient with metastatic breast cancer are unlikely to be cured and in the absence of curative treatment, the goals of therapy typically shift from cure to palliative, symptomatic control for improved quality of life and prolongation of survival. The decision for surgical resection may be indicated in symptomatic women, which in this case was daily vaginal bleeding for months that was detrimental to the patient's quality of life. In one report of 59 patients who underwent surgery for breast cancer metastases to the abdomen and pelvis, five-year survival from time of surgery was 24 percent (23). Median survival was improved if debulking to <2cm of residual disease could be accomplished and if the disease-free interval was >5 years after initial diagnosis (23).

Conclusion

Metastasis to the uterus from a primary breast carcinoma is uncommon. However, the presentation of vaginal bleeding in a breast cancer patient should prompt investigation of breast cancer metastasis to the uterus.

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A CASE REPORT OF ECTOPIC EXTRA-HEPATIC RETRO-PERITONEAL SUPRA-RENAL GALL BLADDER

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Abstract

Retro-peritoneal ectopic position of the gall bladder is a very rare finding. If encountered the surgical management will test the patience and the skills of the surgeon. There is only one reported such case in the English literature (1), and that case was not surgically operated but only radio-logically proved. Here is the first reported case of such surgically removed retro-peritoneal ectopic gall bladder situated above and in front of the right kidney, extra-peritoneally and extra-hepatically.

Case Report

The patient is a 66-year-old male with a long history of heavy ethanol abuse who comes to the Pomona Valley Hospital Medical Center's emergency room on 12/14/2010 complaining of severe epigastric pain with nausea and vomiting. The pain radiates to the back. The CT scan, Nuclear scan and MRI of abdomen(figs:1-6) revealed pancreatitis, acute cholecystitis, cholelithiasis, choledocholithiasis. His past history included heavy ethanol abuse, tobacco use,and of rheumatoid arthritis. He is morbidly obese. He had multiple incisional hernia surgeries with meshes.

His medications were Methotrexate,Tylenol,Dilaudid, and Folic acid.

Because of choledocholithiasis, an ERCP with sphincterotomy and removal CBD stones and a biliary stent was placed. The large stones from the CBD could not be removed.

A decision was made to do exploratory laparotomy and lysis of extensive intestinal adhesions and cholecystectomy with CBD exploration with removal of stones. As expected the exploratory laparotomy through right subcostal incision extensive intestinal adhesions secondary to previous multiple incisional hernia repair surgeries were found. There were extensive adhesions of the small bowel and the large bowel,the transverse colon and the liver to the anterior abdominal wall. All these adhesions were meticulously lysed and the liver was exposed from the falciform ligament to the entire right lobe of the liver. The gallbladder was not seen in its usual position. Hence, a possibility of intrahepatic gallbladder was entertained. The intra-operative Ultra Sound was performed. No intrahepatic gallbladder was identified. However the gallbladder was found with the help of intra-operative Ultrasound in an abnormal position,ectopically situated, in retro-peritoneal, extra-hepatic position, in a very posterior location behind and below the right lobe of the liver lying horizontally rather than lying in a vertical position underneath the liver, above and anterior to the kidney. Once the gallbladder was identified, the exposure was facilitated with help of a Bookwalter retractor to access the most posteriorly and abnormally located gallbladder. The gallbladder was dissected retrograde fashion and brought down to

the common bile duct area, where the common bile duct was exposed and incision was made for removal of the common bile duct stones. Because of the common bile duct inaccessibility, it was decided to leave the stent in place. The common bile duct was sutured with 3-0 Vicryl sutures placing interruptedly. After obtaining complete hemostasis, the gallbladder was removed. The common bile duct was sutured with 3-0 Vicryl sutures. The Jackson-Pratt drain was placed and was brought out through a separate stab wound. The abdominal subcostal incision was closed in the usual fashion and the patient was transferred from the operating room to the recovery room in a satisfactory condition.

Discussion

The liver, extra-hepatic bile ducts, the gallbladder, and the ventral pancreas develop from a hepatic diverticulum off the primitive mid gut at 4th week of intra uterine life. The complex rotation and canalization and migration of the gallbladder bud by 28th week should result in the normal anatomical position of the gallbladder.(ref.10) The ectopic position of the gallbladder is the result of the embryonic developmental malformation.

The anomalies of the gallbladder are related to (ref:1-12):

1: Number :
Agenesis (incidence:0.38% is associated with triploidy), double, bi-lobed OR the multiseptated gallbladder

2: Shapes:
Phrygian cap(ref:12),hourglass,diverticulum, cystic,hypo-plastic,rudimentary gallbladder

3: Position:
Left sided,retro displaced, transverse position, floating,intrahepatic gallbladder. Wandering gallbladders(lesser omentum,retro-duodenal,within the falciform ligament, within the abdominal wall muscles,and intra-thoracic, retro-hepatic, & retro-peritoneal.

Conclusion:

Ultrasound is one of the cheapest ways to identify the anomalous gallbladder. Along with the other radiological modalities which are also essential in the appropriate interpretation, it will lead to correct and safe surgical intervention. If intra-operative Ultrasound is employed for identification of the ectopic gallbladder, it will prove to be an invaluable tool for the surgeon in identifying the anomalous gallbladder.

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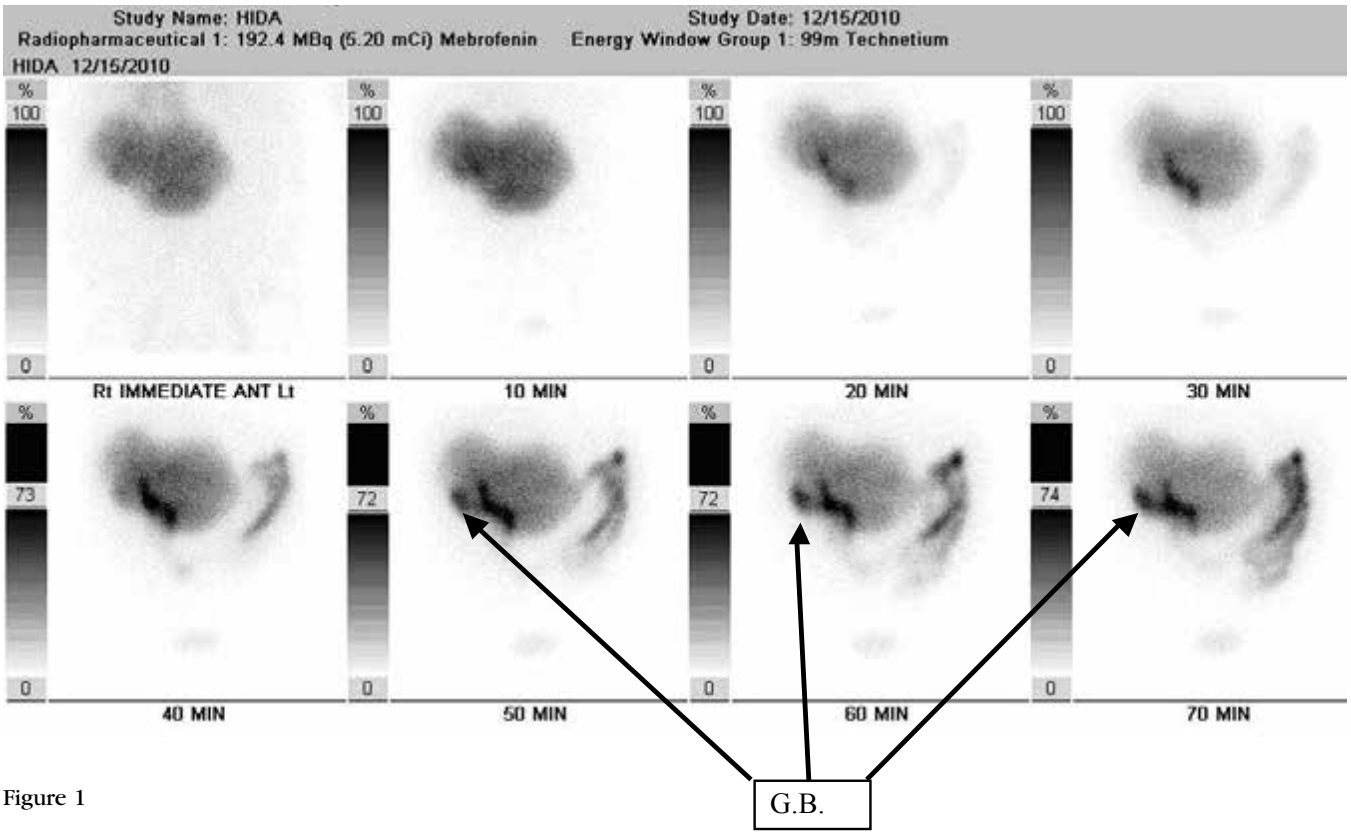


Figure 1

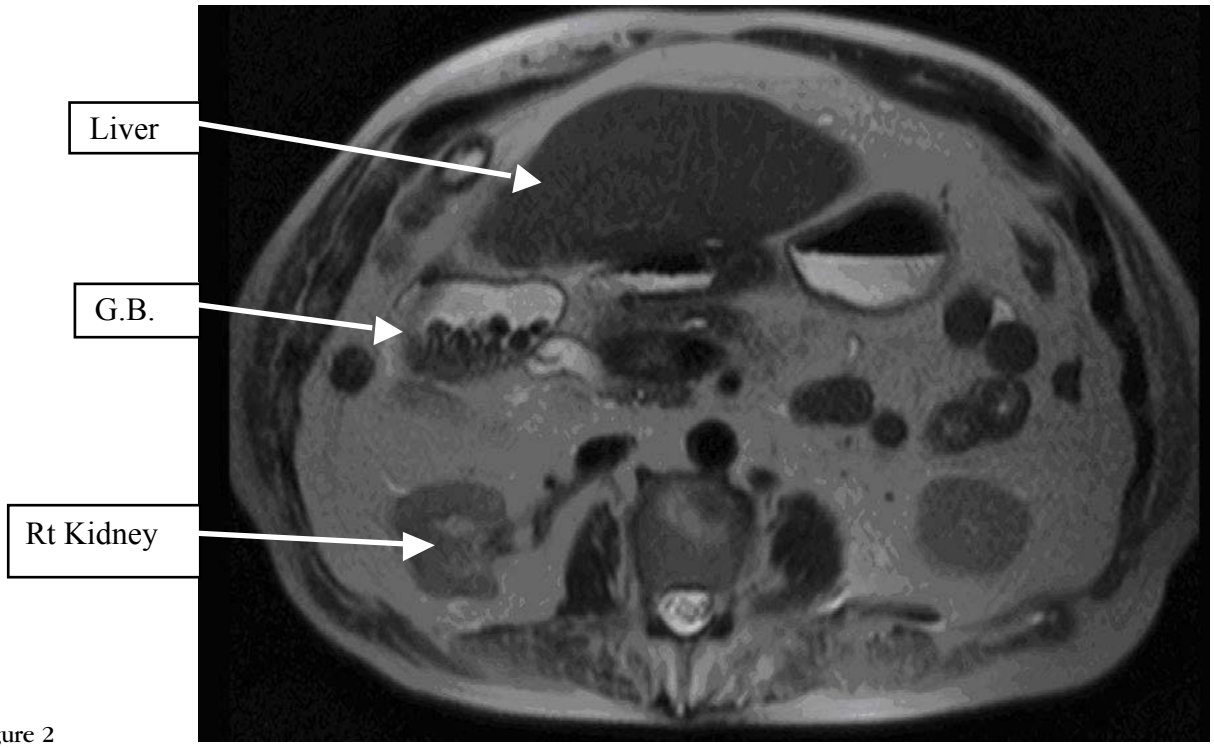
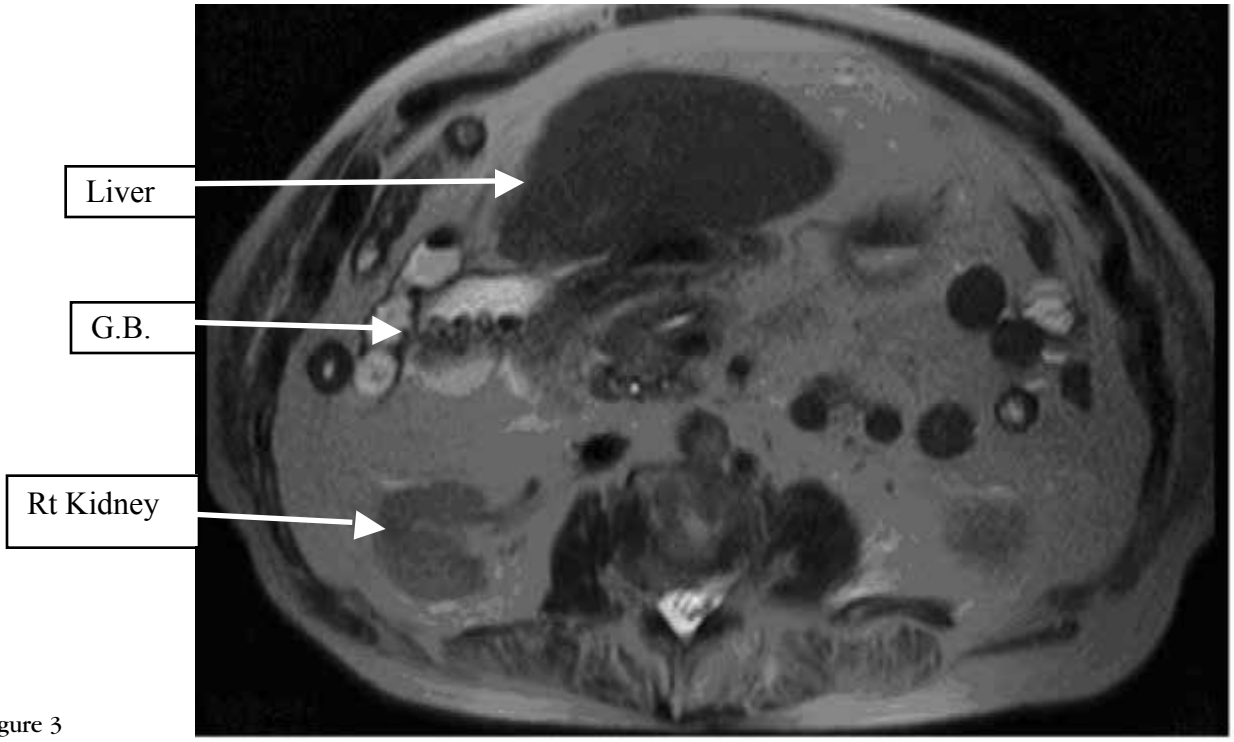


Figure 2



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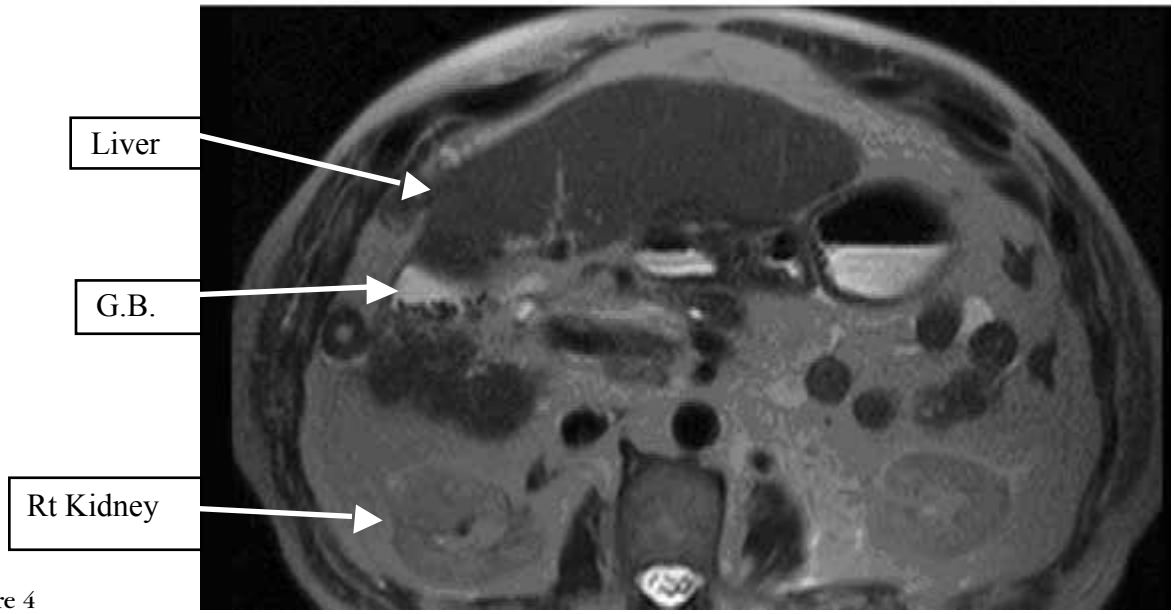

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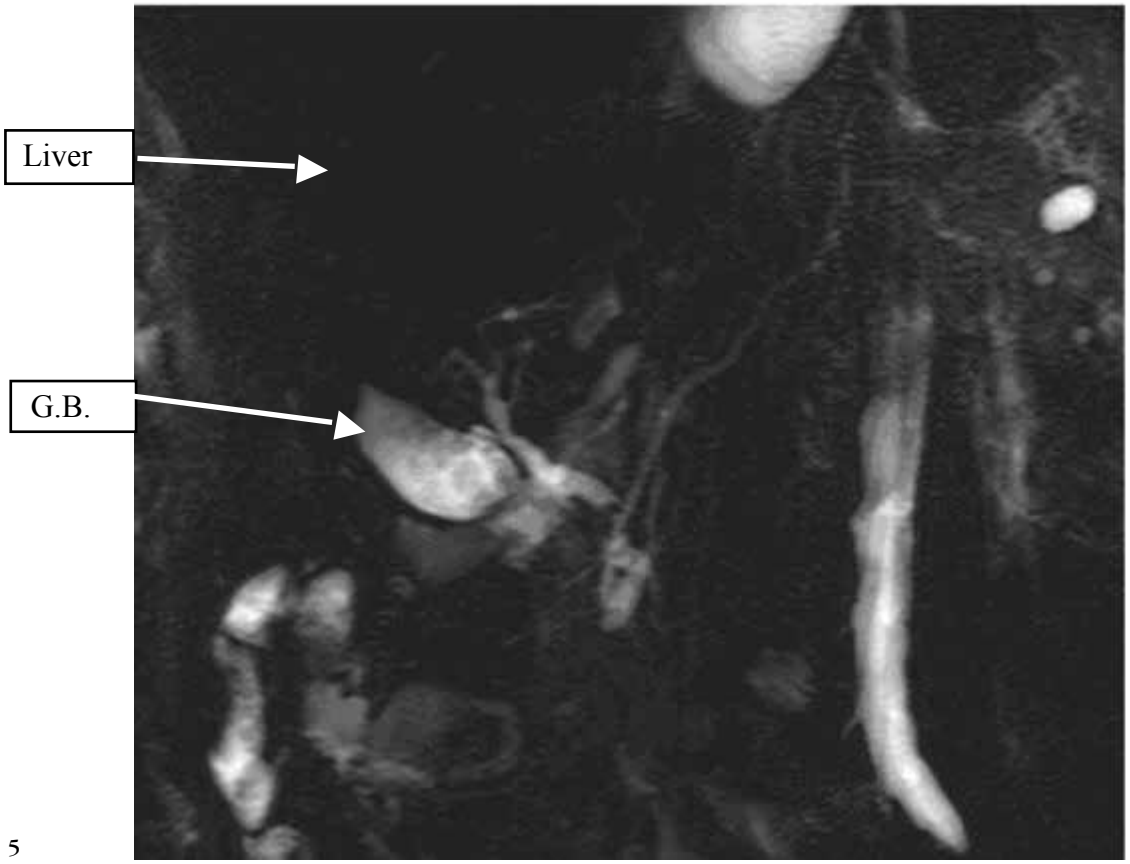

Figure 5

MEASURING THE EFFICACY OF REHABILITATION FOR LOW BACK PAIN

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Introduction

Medical rehabilitation has placed an increasing emphasis on the need to evaluate outcomes, with the goal of reliable and valid measures which treatment efficacy can be assessed. Implementation and analysis of both generic health-related and disease-specific outcomes in clinical practice have the potential to improve quality, help set clinical guidelines, and impact clinical decisions regarding each individual patient [1, 2]. The use of condition specific, standardized outcome measures provides a quantitative assessment of change in an individual's level of function and their adjustment to disability for attributes most relevant to a specific disease or condition. Correlating change in these outcomes with various therapeutic interventions allows clinicians to better understand and optimize their care. This article will illustrate one example of outcome collection in an outpatient Neurosurgical and Orthopedic rehabilitation setting. Low back pain was chosen as it is the most common musculoskeletal condition and comprises a significant percent of the caseload in orthopedic rehabilitation [3].

Low back pain can be caused by injury to many different structures of the spine including muscles, ligaments, facet joints, discs, nerves, and bones. In addition low back pain can be the result of congenital disorders, metabolic and metastatic diseases (for review see [4, 5]). Low back pain is the second leading cause for medical visits in the United States and the fifth most common reason for hospital admissions [3]. It is estimated that 80-90% of people in U.S. will be affected by some form of low back pain during their lifetime with 1-2% requiring surgery, 5-10% suffering from chronic low back pain, and 1% of that total will be disabled [3]. Psychological issues such as distress, depression, somatization, and fear avoidance may also play a role in low back pain and the ensuing disability that results from low back pain [6, 7].

Recent clinical guidelines published in the Journal of the American Academy of Orthopaedic Surgeons recommend physical therapy as an early option for treatment of low back pain [8]. Strengthening the core muscle groups, which include both the abdominal wall and lumbar musculature, has been suggested to be an important component in the treatment of low back pain [9]. In addition, exercise has been shown to improve physical function and decrease pain in adult patients with chronic low back pain [10-12]. In order to determine the efficacy of physical therapy for low back pain in our outpatient rehabilitation hospital, we implemented a number of standardized outcome measures to be used with all patients admitted for low back pain. In this article we will summarize the outcome measures chosen and provide an initial assessment with a small pilot population.

Outcome Measures

To effectively assess low back pain, standardized outcome measures were chosen that would attempt to cover the range of functional impairments that may manifest in individuals with low back pain. Patient-reported and clinician-documented measures were chosen to capture both the patient's perceived level of change and the more objective, clinician-

documented functional change. All outcomes measured were collected three times, at the initial evaluation, the mid-point of treatment, and at discharge.

Patient-reported outcome measures

Patient reported outcome measures provide a unique assessment of an individuals perceived level of ability or disability, and the changes they recognize in themselves following a treatment or intervention. The Oswestry Disability Questionnaire (OD) and Fear Avoidance Belief Questionnaire (FABQ) have both been used clinically to evaluate individuals with low back pain (Table 1). The OD is a well-documented diagnosis-specific tool that measures disability in patients with low back pain in relation to activities of daily living [13-16]. The OD has been used extensively in both research and clinical settings due to its good specificity, validity, and test-retest reliability [13, 16]. A lower score on the OD signifies less disability and is correlated with increased functional ability as well as improved quality of life [2, 17-19]. The FABQ was designed to quantify an individual's pain-related fear of movement, specifically, fears and beliefs about the need to change behavior in an attempt to avoid pain [20, 21]. It is divided into two sections, one pertaining to physical activity and the other related to work, which can be completed independent of each other. A high score on the FABQ has been shown to be correlated with an exaggerated perception of pain and a higher level of self-reported disability suggesting that this measure may be used in part to predict an individual's perceived disability [22-24].

While the OD and FABQ are specific for back pain, the SF-12 Quality of Life measure (SF-12) was developed as a generic heath status measure [25, 26]. The SF-12 and its longer version the SF-36 are widely used and accepted tools to assess self-reported aspects of health related quality of life [26-28]. The SF-12 is a 12-item subset of the original 36 items in the SF-36 with the same 8 domains being examined including physical function, physical role, bodily pain, general health, social functioning, vitality, emotional roles, and mental health [26, 27]. Two summary scales are reported from the SF-12, the physical and mental component summary scores (PCS and MCS). The SF-12 has been found to be reliable and sensitive in several different paradigms and conditions including longitudinal studies, stroke, pancreatitis, fibromyalgia, and low back pain [1, 2, 18, 19, 29-32].

Physical Functioning Outcomes

As mentioned previously, low back pain can be the result of injury, congenital disorders, or disease (for review see [4, 5]) with disability resulting from low back pain varying depending on the etiology and the individual. To assess functional change related to low back pain several objective clinician-reported measures were chosen including lumbar range of motion (ROM), neural tension, manual muscle test (MMT) of the lower extremity, gait velocity, the 30 second sit to stand measure, and a number of items looking at core stability (Table 1). Previous studies have observed restricted lumbar ROM in individuals with low back pain [33, 34]. Lumbar ROM can also be indicative of the type of injury resulting in back pain. For example, patients' with disc protrusion or extrusion in the lumber spine may show limited ROM depending where the lesion is located. Similarly, limited extension ROM or pain with extension ROM may be seen in patients with low back pain as a result of spinal stenosis.

Another potential cause of low back pain, as well as weakness and pain in the lower extremities, is compression or irritation of a nerve in the spine, which may lead to neural tension. Neural tension refers to the nerve's ability to move within the body. When there is tension on the nerve, the ability and freedom for it to move is constrained. In addition to pain, neural tension can lead to a loss of spinal and lower extremity range of motion, which can in turn alter gait pattern. Often with derangements involving a lower lumbar disc, such as a posterior or lateral protrusion or extrusion of the disc, a patient may show a positive neural tension sign secondary to compression of the nerve root [35]. Neural tension can be tested using the straight leg test, which has been shown to be a highly specific test [36]. The straight leg test is performed in the supine position with legs extended. The examiner lifts the leg flexing at the hip while keeping the knee extended [35]. If pain is increased in the lumbar region or the legs after 30 degrees of hip flexion the test is positive for probable herniated disc [37, 38].

Another sign of nerve involvement is weakness in a specific nerve root distribution. Patients with low back pain may show weakness in their lower extremities when there is damage to the nerve

supplying a muscle or group of muscles. Other patients, especially chronic pain patients, may have weakness secondary to being deconditioned. A useful measure in determining the involvement of nerve damage and lower extremity weakness is manual muscle testing (MMT) (for review see [39]). Additional measures that can signify nerve root compression are the inability to heel and toe walk, which affect L4-L5 and L5-S1, respectively.

Trunk Stabilization and strength is understood to play an important role to prevent as well as rehabilitate low back injuries although more research is needed in this area [9]. Muscle groups that are involved in trunk stabilization include the rectus abdominus, external obliques, erector spinae muscles, transversus abdominus, and lumbar multifidi. Measures that monitor trunk stabilization strength and function include the ability to perform abdominal bracing, the ability to perform a partial sit up and hold contraction for 10 seconds, the ability to bridge, the ability to hold neutral spine in quadruped, and the ability to perform the bird dog exercise. Abdominal bracing, defined as the ability to pull the navel to the spine performing an isometric contraction without changing the position of the pelvis or spine, is a basic exercise in core strengthening that mainly recruits the transverse abdominus muscle. The partial sit up, ability to lift one's head and scapula off the mat, recruits mainly the rectus abdominus and external obliques and is often used to determine overall fitness level and can also be an indicator of core strength. One of the main core muscles often affected in patients with low back pain is the multifidus which is the main muscle activated in the bridge exercise (Figure 1A). The bird dog exercise, which is the ability to stabilize in a quadruped position and extend opposite, requires the stabilization, coordination, and recruitment of the core muscles especially external obliques [40]. The ability to perform these core strengthening measures and the correlation between these measures and other clinician-documented and patient-reported measures will provide valuable information helping to further define the role of trunk stabilization in low back pain.

Low back pain can cause both severe back and lower extremity pain that has been shown to cause gait dysfunction [41-43]. Gait velocity, which is the ratio between stride length and step length, is an important indicator of functional status, social

A. B.

Figure 1. Illustration of a Bridge (A) and Sit to Stand test (B)

participation, and has been shown to predict mobility impairments [44-46]. Research suggests that with low back pain there is a change in the rotation and coordination of the pelvis and thorax during gait leading to slower gait velocity [47]. Exercise programs for patients with chronic low back pain have been shown to improve gait velocity [48]. Measuring gait velocity using the 5M-walk test provides an easy way to administer and retest gait with good reliability and norm values for age. There are also documented values that define pathological gait and the velocity needed for common community gait activities such as crossing a street. Gait velocity has been shown to predict adverse outcomes in well functioning elderly persons [49, 50].

Another assessment tool used to measure functional strength is the 30 second sit-to stand test, which simply measures the number of times a person can perform the transition from sitting to standing in a 30 second time period (Figure 1B). Moving from a sitting to standing position is an activity that is performed daily and if impaired, significant functional limitations can occur [46]. Impaired ability on this task has been found to predict further disability and correlate with greater difficulty in performing activities of daily living [51]. The 30-second sit-to-stand test is easy to administer, has good test retest reliability, normative values for age, and has been shown to be sensitive to change in individuals with low back pain [52].

Table 1: Outcome measures

Domain
Patient Reported Outcome measures
Oswestry Disability Questionnaire (OD)
SF-12 Quality of Life (SF-12)
Fear Avoidance Belief Questionnaire (FABQ)
Pain 0-10/10
Physical Functioning outcomes
Range of motion – Lumbar
Straight Leg Raise (SLR) and crossed SLR
Ability to bridge
Ability to maintain neutral pelvis in quadruped
Ability to maintain neutral pelvis in quadruped with opposite arms/legs
Ability to perform abdominal bracing
Ability to lift head and scapula off mat and hold >10
Lower extremity strength
Ability to toe walk
Ability to heel walk
5 meter walk (gait velocity)
consecutive sit to stand in 30 seconds

Analysis of Outcome Measures

To illustrate the use of the above outcome measures in an outpatient rehabilitation setting a select pilot population of 12 patients with no surgical history and a diagnosis of low back pain were used. The selected population is comprised of 6 males and 6 females with a mean age of 59.8 years and range from 30 to 84 years (Table 2). This large range in age highlights the extent to which low back pain is a widespread problem effecting old and young. It also demonstrates the ability of these measures to capture a wide variety of individuals with varying degrees of disability and not encounter large issues with floor or ceiling effects. The average number of visits for these patients was 17 but ranged between 8 and 37 visits. Therapy services are referred by a physician and will vary according to patient needs but can include a traditional land-based only physical therapy program and/or aquatic therapy with treatment aimed to decrease pain and improve function. An educational piece is also issued to each patient at admission discussing proper body mechanics and posture. Participation in these physical therapy services depends on the patient's abilities and is recommended by the physical therapist or referring physician. In this population we had 42% of the patients participating in land-based physical therapy only, 16% in aquatic therapy, and 42% receiving a combination of both (Table 2).

Table 2: Subject Characteristics (n=12)

Characteristic	Total (%)
Sex	50% M, 50% F
Age (yrs)	
Mean ± SD	59.8 ± 14.8
Range	30 - 84
Number of visits	
Mean ± SD	17.0 ± 8.3
Range	8 - 37
Type of therapy	
Land	5 (42%)
Pool	2 (16%)
Both	5 (42%)

As stated above, patient-reported outcome measures were used to provide an assessment of the patient's perceived level of functional ability and disability. A patient's self-reported pain level is reported at each therapy visit using a scale of 0-10 with 0 = no pain and 10 = highest levels of pain. In this pilot population a significant decrease in pain was reported between the initial evaluation and mid-way through treatment (Figure 2). Correlating with this reduction in pain were significant changes in patient-reported disability using the OD (Figure 1). These changes represent a clinically meaningful reduction in disability. Using the FABQ we observed a trend towards improved understanding about pain and its impact on physical function, suggesting effective patient education. Taken together, the observed changes in overall pain, the OD, and the FABQ support the role of physical therapy in the treatment of low back pain. Interestingly, when patients were grouped according to the type of therapy services they received (land-based only, aquatic-based only, or both) a difference in the mean scores between groups was observed with the aquatic-based patients starting with higher levels of pain and more disability. While the starting mean does appear to be different, the slope of change between admission and follow-up appears to be fairly consistent between the groups, thus identifying different therapy

needs initially but with similar overall outcomes. With a larger data set this trend can be further defined.

While the SF-36 and SF-12 are known as generic health-related quality of life measures, previous studies have found these outcomes to be effective and sensitive in individuals with low back pain [2, 53]. In this small sample we did not observe significant changes using the SF-12 however, the physical component score of the SF-12 did show a trend towards improved quality of life (Figure 2). Further, a correlation was observed between the OD and the physical component score with a decrease in patient-reported disability predicting an increase in quality of life. Interestingly, we also observed a correlation between admission FABQ scores and a patient's mental component score on the SF-12, signifying a potential relationship between individuals' beliefs about pain and movement and how it affects their quality of life.

The clinician-documented physical function outcome measures that we chose were intended to provide a more objective assessment of a patients' level of functioning. While not all effects were significant or can be defined by numeric change, several improvements in physical function were noted and suggest meaningful changes in a patients functioning as a result of their participation in physical rehabilitation. In two widely used outcome measures the 5-meter walk and the 30-second sit-to-stand test we observe changes in gait, lower extremity strength, and mobility (Figure 3). These functional measures correlate with one another suggesting a previously reported relationship between low back pain, lower extremity strength, and gait speed [41, 44, 47]. Of particular interest was the correlation between patient reported pain levels and the 5M walk, underlying the relationship between pain and gait velocity. With a larger data set these measures can be used to correlate with quality of life, disability, pain, and perhaps even predict overall levels of functioning. In addition, we hope to be able to determine correlations between lumbar ROM, neurotension, lower extremity weakness, functional level, gait velocity, patient-reported pain level and disability and overall outcome. We did observe interesting trends when looking at range of motion in this population, with 80% of patients increasing their lumbar range of motion and 63% increasing in lumbar extension range of motion. When lumbar range of motion was correlated with the OD and FABQ a relationship was observed between restricted range of motion and higher scores on the OD and FABQ, suggesting greater disability. Trunk stabilization, strength and function, was improved in 100% of the patients who originally experienced difficulties in these areas. While our small sample prevents definitive conclusions regarding trunk stabilization and low back pain, these data suggest a meaningful relationship, which needs to be studied further. Taken together, these functional outcome measures provide a nice basis of a patient's level of functioning and how it correlates to their level of pain, disability, and overall well-being.

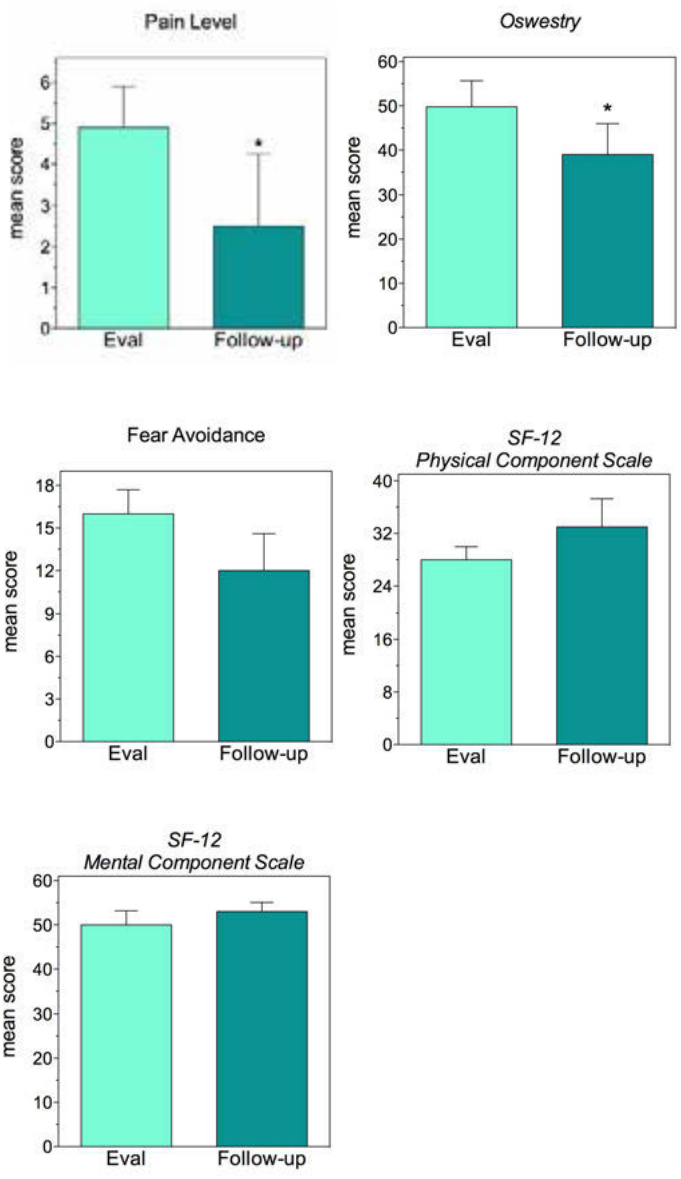

Figure 2: Patient-Reported Outcome Measures. Significant changes were observed in the level of pain patients were experiencing and in the amount of pain-related disability as measured by the Oswestry Disability Questionnaire. Data show mean scores for each measure at initial evaluation and upon follow-up during physical therapy services (+ SEM). *$p < 0.05$ in comparison to the eval scores.

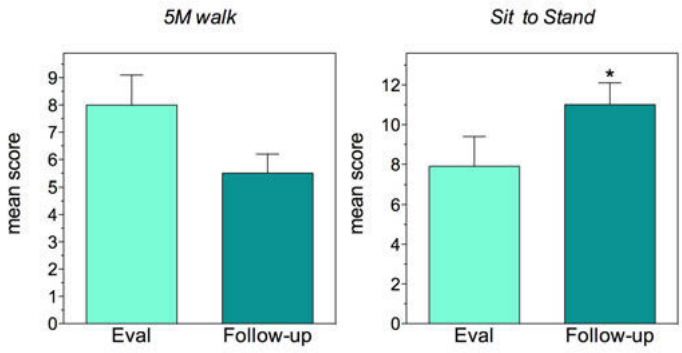

Figure 3: Physical Functioning Outcome Measures.
A trend towards increased gait velocity was observed using the 5M-walk test. Significant changes were observed in the number of sit-to-stands patients were able to do in 30 second. Data show mean scores for 5M walk and sit-to-stand at initial evaluation and upon follow-up during physical therapy services (+ SEM). *$p < 0.05$ in comparison to the eval scores.

Conclusion

Utilization of standardized quantifiable outcome measures for a diagnosis such as low back pain helps to assure effective treatments with positive patient outcomes. To effectively assess low back pain in an outpatient neurosurgical and orthopedic rehabilitation setting, patient-reported and clinician-documented measures were implemented to capture both patient perceived changes and more objective clinician-documented changes, which cover the range from quality of life to functional ability. Findings in this small sample suggest these measures are useful and valid for this population. Our results are supported by the numerous research publications documenting the utility of these outcome measures. We plan to continue this research with a larger data set including a surgical population so the impact of surgery can be evaluated as well as other variables that may underlie a patient's condition and their overall outcomes. We predict that this research will yield important findings supporting the use of these assessment tools for outcome measures in the clinical setting and a role of physical therapy for the treatment of low back pain.

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GENETIC HYPERCOAGULOBILITY STATE

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Introduction

Many patients with hypercoagulobility state are born with mutations of one or more genes associated with coagulation. Common genetic mutations are Factor V Leiden, Prothrombin G20210A varient, Methrylentetrahydrofolate reductase (MTHFR) mutation, Deficiencies of Protein C, Protein S and Antithrombin III. Less common genetic hypercoagulobility conditions involve mutations in Fibrinogen chains, Factor XIII, Platelet Glycoproteins, Plasminogen Activator Inhibitor-1, and Thrombin Activatable Fibrinolysis Inhibitor (1).

Interestingly, people with blood groups other than type O are at a two- to fourfold relative risk.Those with type O have lower levels of the blood protein von Willebrand factor as well as factor VIII, which confers protection from thrombosis (3).

Well known environmental or acquired factors associated with hypercoagulobility state include oral contraceptives, hormone replacement therapy,Antiphospholipid syndrome, Lupus anticoagulant, pregnancy, smoking, nephritic syndrome, connective tissue disease such as Systemic Lupus Erythematosus or Rheumatoid Arthritis, Myeolproliferative disorders such as Polycythemia Vera or Essential Thrombocythemia, and solid organ malignancy.

Despite well established hereditary genetic conditions associated with high risk of thromboembolism, current management of the venous thromboembolism especially recommended by American College of Chest Medicine (2) does not take those genetic conditions into consideration, which can result in potential harm or even fatal consequence to patients.

We are presenting 2 patients who developed venous embolism including pulmonary embolism due to their genetic mutations.

Case 1

P.Y is a 49 year-old White man who developed Deep Vein Thrombosis (DVT) of the left leg 7 years ago, and he had been treated with Enoxaparin (Lovenox®) followed by Coumadin for 6 months. However, 1 year after he stopped the Coumadin, he had pulmonary embolism. Since then he was taking the Coumadin. However, due to his traveling job situation, he did not take it for the past 1 ½ years. Recently he developed painful swelling of the left leg and the Ultrasound on 3/22/2011 revealed a thrombus in the left greater saphenous vein. Now he is back on the Coumadin therapy.

Family history included his father died of prostate cancer

at age 63, but no significant blood or clotting disease or premature coronary artery disease reported.

He is a long term cigarette smoker (30 year-pack history), and drinks whiskey. He has fatty liver.

Physical examination showed BP 130/84, not icteric or pale eyes, red face, no peripheral lymphadenopathy, few wheezing, normal heart sound, no hepatosplenomegaly, 1+ leg edema, no neurological deficits.

Laboratory tests were done.

CBC on 4/1/2011 showed WBC 9.4 k/uL, Hb 17.8 g/dl, Hct 52.2%, RBC 5.03 mil/uL, MCV 103.8, Platelet 161 k/uL. Prothrombin Time (PT) 11.2/International Normalization Ratio (INR) 1.1, Partial Thrompoplastin Time (PTT) 28 seconds.

Comprehensice Metabolic Panel is normal except total Bilirubin 3.0, AST 48 U/L.

Hypercoagulobility tests: Protein C, S and Antithrombin III levles are all normal. Anticardiolipin Ab (-), Phosphatidyl serine Ab (-), Prothrombin A20210G mutation (-).

Homocycteine level 18.5 umol/L (elevated), Factor V Leiden one copy (+), MTHFR/A1298C mutation 2 copies (+).

Lupus anticoagulant is presumably negative because of the normal PTT.

Chest X-ray showed findings suggestive of emphysema.

The patient was diagnosed as having hypercoagulobility state due to the heterozygous Factor V Leiden mutation as well as the homozygous MTHFR/A1298C mutation. The hypercysteinemia is the resultant of the MTHFR mutation. The high hematocrit was considered as secondary polycythemia from the heavy smoking.

Case 2

E.M is a 45 year-old Hispanic woman with a history of Rheumatoid Arthritis who developed acute dyspnea. CT scan revealed bilateral pulmonary emboli in the arterial branches to the right middle lobe as well as to the lingula. Apparently she was treated with Lovenox, and was discharged with an oral anticoagulation drug.

During her hospitalization, Lab tests included: WBC 9.2, Hb 14.1, Hct 42%, Plt 376 k/uL; unfortunately, baseline PT and PTT are not done; normal CMP; Factor V Leiden (-); Homocycteine 6.3 umol/L (normal); normal levels of Protein S and Antithrombin III; Protein S activity was initially slightly low at 43%; Anticardiolipin IgM (+); Rheumatoid Factor 148(+); Phosphatidylserine IgM(+). She has been treated with Remicade®, methorexate and prednisone for Rheumatoid Arthritis. Her mother has a history of miscarriage.

Physical examination showed BP 100/60, not icteric or pale eyes, no peripheral lymphadenopathy, normal heart and lung sounds, no hepatosplenomaegaly, trace left leg edema. Repeated Protein C and antigen done as an outpatient turned out to be normal at 79% and 144% respectively. Prothrombin A210200G was (-); MTHFR genetic test showed that she is homozygous for C677T mutation.

Discussion

Factor V Leiden is an autosomal dominant condition which results in a factor V variant which is resistant to degradation by activated Protein C (APC). When factor V remains active without being degraded by APC, it facilitates overproduction of thrombin, leading to excess fibrin generation and excess clotting. This mutation is a single nucleotide polymorphism (SNP) located in exon 10 in which as a missense substitution it changes amino acid of the gene from arginine to glutamine. About 5% of Caucasians in North America have factor V Leiden. However, this condition is less common in Hispanics and African-Americans and is extremely rare in Asians (4).

Up to 30% of patients who present with deep vein thrombosis (DVT) or pulmonary embolism have this condition. Inheriting one copy of the mutation from a parent (heterozygous) increases by 4-fold to 8-fold the chance of developing venous thrombosis. People who inherit two copies of the mutation (homozygous), one from each parent, may have up to 80 times the usual risk. About 1% of Factor V Leiden patients are homozygous who develop a more severe clinical condition (4).
The presence of acquired risk factors for venous thrombosis, including smoking, hormonal therapy for postmenopausal women, a use of oral contraceptives and immobilization such as recent surgery, further increase the chance of developing DVT. Similarly, co-existence of other genetic mutations further compound the risk of venous thromboembolism as seen in this case report.

Women with factor V Leiden have a substantially increased risk of venous thromboembolism. They also may have a small increased risk of miscarriage and stillbirth as well as preeclampsia. However, many of these women go through pregnancies with no difficulties, while others may repeatedly have pregnancy complications.

Methylenetetrahydrofolate reductase (MTHFR) is an enzyme encoded by the MTHFR gene. MTHFR catalyzes the conversion of 5,10-methylenetetrahydrofolate to 5-methyltetrahydrofolate, a cosubstrate for homocysteine remethylation to methionine. Therefore, the weak MTHFR activities due to the genetic mutation results in hyperhomocysteinemia. It has been reported that the elevated plasma Homocycteine increases the risk of venous and arterial thromboembolism and causes premature atherosclerosis.

It is also reported that MTHFR mutation is associated with complication of pregnancy such as miscarriage and preeclampsia, migrane headache, and cognitive disorders such as Alzheimer's disease (3,5,7).

Because the reduced MTHFR activity with C677T mutation increases the methotrexatete toxicity, the dosage of the methotrexate was modified for the patient of the case 2. This patient also has positive anticardiolipin IgM antibody and IgM phosphatidylserine antibody, which can be seen in patients with active Rheumatoid arthritis as well as with circulating lupus anticoagulant. In this case, a prolonged baseline PTT not corrected by mixing normal plasma would have confirmed the presence of the circulating lupus anticoagulant. Unfortunately the baseline PTT was not available.

Some research data showed that only C677T mutation is associated with hyperhomocysteinemia (8). However, the patient of the case 1 has a very high homocycteine level with A1978C mutation while the other patient in the case 2 has normal homocysteine level despite homozygous C677T mutation. Therefore, the research of this MTHFR mutation appears to be incomplete and not comprehensive. Certainly more clinical research studies are needed to answer many questions regarding MTHFR mutation especially in terms of the management of patients with this mutation.

The patient of the case 2 had low Protein C activity in the blood specimen drawn before the anticoagulation therapy. However, its level became normal when it was repeated later. The Protein C activity level can be influenced by other factors. For example, warfarin therapy decreases the Protein C activity because the Protein C is vitamin K dependent. Therefore, a patient taking warfarin can have a falsely low Protein C activity. Furthermore, in acute stage of thromboembolism, the Protein C activity can be falsely low because the Protein C is consumed during the active clotting process as shown in the patient of the case 2. Patients with liver disease have low Protein C activity due to impaired synthesis of the Protein C (5).

The Protein C has a very short half-life of 6-8 hours and is therefore one of the first decreased coagulation protein with warfarin therapy, resulting in temporary hypercoagulobility condition. Therefore, warfarin should be given to patients with thromboembolism only after the initiation with heparin or low-molecular heparin. Protein C deficiency also increases the risk of the warfarin-induced skin necrosis (5).

Because of the history of the life- threatening pulmonary embolism, both patients are required to be on a life-long anticoagulation therapy. Life style modification such as cessation of smoking, exercise and lowering cholesterol should be accompanied to lower the risk of the blood clotting. Furthermore, it is advised that the siblings and children be tested for those genetic mutations.

Current guideline by American College of Chest Medicine (2) does not take those genetic conditions into consideration in the management of venous thromboembolism. Nevertheless, clinicians are encouraged to carefully look for possible underlying genetic hypercoagulobility conditions when treating patients with thromboembolism.

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A NEEDLE AS THIN AS THE MOSQUITO'S MOUTH

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As vividly as yesterday, I remember what happened 20 years ago.

Bill was a 50 year-old man with lung cancer metastatic to the brain. The brain lesions had recurred despite conventional radiation therapy. At the University of Southern California he had finished another round of radiation by gamma knife, the new method of treatment at that time. Because of deep vein thrombosis of the leg, he had been admitted to the hospital in Upland, California, and I was just walking out his room after the round when he called out to me. He was trying to hand something to me. I took it and bent my head to see it. To my surprise, it was a check for $10,000.

"I don't know how to express my gratitude to you," he said in a weak and slightly hoarse voice. On his trembling lips a shadowy smile appeared.

Tweenty years ago this $10,000 check was hardly a small amount of money, especially to someone who had started a new medical practice and was, of course, in need of financial stability. Not only because it was rude to reject his offer but also because I honestly wanted to have that money, I took it. But thinking that I was hesitating, he said, "You don't need to do it. Thank you so much." Bill had accumulated great wealth by working as a construction contractor. Just three months later he died.

About three years ago, my fasting blood glucose level was high at 135 mg/dl. I had diabetes. By following a low carbohydrate diet and getting some more exercise, I was able to bring my blood glucose levels down to normal without taking any hypoglycemic drugs. I continue to check my blood glucose level at home.

But checking my level meant puncture pain from those big needles. I became increasingly resistance to the daily finger pricks that those checks required. I tried every available lancet on the market – the OneTouch from Johnson & Johnson's LifeScan, the Accu-Chek Multiclix and Softclix from Roche, the FreeStyle from Abbott, and the BD lancet from Becton, Dickinson. But all those lancets were more or less the same in the amount of puncture pain they caused.

Necessity is the mother of invention, and I came up with an idea of a painless lancet. What if I made the needle as thin as the mosquito's mouth? If the blood coming out from the skin were not enough for glucose testing, could two or three needles in a bundle mounted on a lancing device body produce two or three times the amount of blood amount as a single needle? So I contacted a patent attorney to file a patent application. Then, I asked a Japanese engineer in Tokyo to make a prototype lancet with two 3mm-long 38-gauge needles mounted on the end of a lancing device.

As I hoped, the first prototype lancet that the engineer made did not cause puncture pain. However, I found out that a 3mm long 38-gauge needle could bend when it hits hard, calloused skin. Furthermore, someone else had already patented the idea of multiple needles mounted on a lancing device. I fell into deep despair because I had spent a good deal of money and time on it the prototype lancet.

Still, the amount of blood from a single 38-gauge needle was enough to test blood glucose levels with the new glucose meters on the market. So having two or three needles was unnecessary.

Nevertheless, I remained busy with my practice. A patient named Doris with newly diagnosed laryngeal cancer was referred to me for treatment. She was a 78 year-old woman who I later found was the older sister of the late Bill, who had died 17 years earlier having left such a strong memory in my mind and $10,000 in my bank account. I wondered if they carried cancer genes, although they both smoked, and it is well known that smoking is the most common cause of both lung and laryngeal cancer. Fortunately, a combination of chemotherapy and radiation therapy put her cancer into complete remission, which repeated laryngoscopy and PET/CT scans confirmed.

Around that time I had a strange dream. In the dream Doris was writing a check. She showed it to me, which reminded me of Bill giving me a large check many years earlier. In the dream I thought that Doris simply wanted to show me the wealth she possessed. Since she drove a Mercedes, and didn't seem like someone who showed off, I assumed that she had written a check for several million dollars. But to my great surprise, I saw in my dream that she had added two or three more zeros, making it a $300,000,000 or $3,000,000,000 check.

When I woke up, I still felt the dream was real. Later, when I told Doris of my dream, she laughed out loud and said she wished that she had that kind of money.

Despite my setbacks with making better needles, I was still devising and studying them whenever I could make time during my busy medical practice. I felt some urgency, since I had seen newborn babies in the hospital crying sharply in a shrill, fretful tone obviously in pain when those ordinary 28- or 30-gauge needles punctured their heels. I also found that no lancets were suitable for children, since lancets with the same old thick and long needles were used for adults and children alike.

Finally, I came up with a lancet that satisfied my conditions. It had to be painless, produce enough blood for testing, not intimidating visually, mass producible, compatible with currently available lancing devices, and patentable.

I found the secret. The needle had to cut only the capillary complexes in the papillary layers of the dermis, while not touching or irritating the free nerve fibers beneath. This lancet had to predictably and consistently penetrate no deeper than the papillary layer, regardless of skin conditions. Additionally, it had to minimize the dwell time between the needle and the skin.

The measured depth level of the papillary layer of the fingertip is about 0.6-0.8mm, although it may vary depending on the thickness of the epidermis. For example, a heavily callused finger has a 1mm-thick epidermis. Therefore, I devised a lancet having a needle of 38-gauge and of 0.75mm thickness mounted on a pedestal-shaped structure at the distal end of the lancing device.

Using the lancets that I invented, I conducted a randomized clinical study with about 40 people with diabetes to test whether my invention really was painless while producing enough blood for glucose testing. I published the results in a peer-reviewed medical journal, Clinical Medicine Insights: Endocrinology and Diabetes.

Recently, the International Diabetes Federation invited me to present my clinical study at its meeting in Busan, South Korea. In the United States, the Food and Drug Administration has approved the sale of my lancets under the name "TiniBoy." They are available through www.Tiniboy.com and www.amazon.com. This year, the Korea Intellectual Property Office issued a patent for these lancets, and I am preparing to sell them there before the end of 2011.

I will continue to develop the TiniBoy lancets in hopes that someday they will achieve great success. To date, however, I have had considerable development expenses and haven't yet made any money from them.

Actually, I remain somewhat hopeful, because of the Doris dream. My true dream is to build and operate charity hospitals and cancer centers wherever people need them.

It has been said that one's fate is sealed and predetermined to run a certain course as soon as it was set into motion regardless of one's desire and effort. It could be my fate that I will fail despite my vigorous efforts. It is Doris's choice whether or not to give me the check, regardless of my desire.

Realistically speaking, the odds of success in this business are low. Start-up companies reportedly succeed only about 3 to 5 percent of the time.

So far, the only comfort I get is the many thank-you letters from parents of children with diabetes whose pain I have been able to greatly ease by using my painless TiniBoy lancets. At times, in fact, I consider this business as a charity, because I have been spending

my money and time only to help others without any guarantee of success.

I was once convinced by this theory of fate, because I learned that I could not change the fate of many cancer patients. No matter how hard I tried, having seen those cancer patients die against my and their will, I could not do anything except to humbly accept God's will.

However, it looks sublime and even holy to see the extraordinary efforts of doctors and patients together trying to save patients' lives. Furthermore, I occasionally have seen cancer patients who survived against the odds when doing their best and not giving up easily, making me wonder if the theory of fate is always true.

Suppose there is a poor Mexican woman with ovarian cancer living in Mexico where no adequate cancer treatment is available and is surely destined for an early death there with the chance of long-term survival of less than 5 percent. In the U.S. ovarian cancer is treatable with a high chance of survival, even at an advanced stage. Then she decides to cross the border to go to San Diego for treatment, as American hospitals treat anyone with an emergency medical condition regardless of their immigration status or ability to pay. Then, her chance of survival goes up to 50 percent as soon as she makes that determination to cross the border, when she could get caught and turned away. But if she makes it across, her chance of survival goes to more than 90 percent in the hospital's emergency room.

Regardless of Doris's initial intention not to give me her big check, when I have a deep desire and determination to get it, I might persuade her to give it to me. Then the theory of self-determination of fate becomes true, like it would be for the hypothetical Mexican woman. One can change the course of the fate, succeeding with determination, vigorous effort, and faith, raising the probability of success from a mere 5 percent to 90 percent.

The dream of Doris only showing the check to me may be interpreted as the Tiniboy business having the unfortunate fate of not making a great deal of money. However, even if that interpretation is correct, if I can make my dream come true through the great success of the TiniBoy business with my best effort, just as the Mexican woman changed her fate, the theory of self-determination, not the theory of fate will be proven right.

NEUROPATHIC FOOT ULCERS IN THE DIABETIC PATIENT: REVIEWING THE STANDARDS

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Introduction

Neuropathic ulcers in the diabetic patient have become a common complication. Approximately 50-60% of Type II diabetic patients develop peripheral neuropathy (1). The prevalence of peripheral neuropathy increases with the duration of diabetes with the onset of symptoms usually occurs on average 10 years after the development of diabetes in the Type II patient. The onset is variable in the Type I diabetic patient. Foot ulcers occur in 15 - 25% of diabetic patients (2, 3), which equates to slightly more than 2% annually and between 5.0 - 7.5% of those patients with neuropathy (4, 5). Foot ulceration is the one of the leading causes of hospitalization in the diabetic patient.The average cost of treating a foot ulcer in the United States per episode has been reported to be $13,179. (6) The overall cost of foot ulcers to the United States health care system is approximately 1/3 of the $116 billion spent on diabetes care and related complications in 2007. (7)

The sequela of the ulcers equates to the personal cost. Most patients who heal their ulcers go on to develop ulcers in the future. Additionally, it has been estimated that 15% of diabetic foot ulcers result in lower extremity amputations and 85% of diabetic patients who undergo lower extremity amputations had an ulcer prior to amputation. (8, 9) The amputation then creates additional complications such as increased morbidity and mortality. Five-year mortality rates in diabetic patients with a neuropathic ulcer have been reported to be 45%. (10) There are a number of recent advancements in treatment that have been used to successfully heal these patients, however, a detailed assessment of these patients is critical to determining the proper treatment. Ultimately, prevention is the key to success.

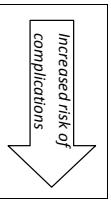
Etiology

A diabetic ulceration typically evolves due to a combination of underlying causes. The triad of peripheral neuropathy, deformity, and trauma are the most common causes. Reiber found that more than 63% of the patients in his multi-centered, retrospective cohort study of 148 patients had all three of these causes. (11)

Peripheral neuropathy is an independent risk factor, and likely the most significant risk factor, in diabetic foot ulcers. The presence of deformity is also critical secondary to the increased plantar peak pressures on the foot. The deformity may result from abnormal biomechanics, such as bunions, hammertoes, and equinus. However, deformities like Charcot neuroarthropathy or hammertoes from wasting of the intrinsic musculature can result from the peripheral neuropathy itself. Not all deformities are visible upon inspection. Examination may reveal limited joint mobility which causes undue stress distal to the affected joint. It is important to recognize that the deformity alone will not cause ulcerations, but when combined with neuropathy the ulcer may develop. (12) Lavery found a 12-fold increased risk for ulceration in patients

Table 1. Diabetic Foot Risk Classification System

Risk Classification	Definition of Class
Group 0	(-) neuropathy, (-) peripheral arterial disease
Group 1	(+) neuropathy, (-) peripheral arterial disease
Group 2	(+/-) neuropathy, (+) peripheral arterial disease
Group 3	(+) ulcer history
Group 4	(+) amputation history



with neuropathy and deformity compared to deformity alone. (13) The trauma involved in ulcer development is frequently minor, repetitive episodes. The constant cycle of micro-traumatic events usually relates to deformities. Lastly the deformities cause areas of irritation with shoe wear, which is critical to the prevention of other injuries in the insensate foot. Irritation from inappropriate footwear is the most common cause of the trauma associated with diabetic foot ulcers. (14)

Other factors have also been associated with the risk of ulcer development. The diabetic foot risk classification system predicts the at-risk patient. Peripheral arterial disease was strongly implicated in ulcer development and is one of the most common lower extremity complications in the diabetic foot. In general, a significant increase in the number of ulcerations, infections, amputations and hospitalizations was noted as the at–risk groups progressed. (15) However, ischemic ulcers may also occur in the diabetic patient regardless of the neuropathic status of the patient, so the etiology of the ulcer is important to determine to achieve successful healing.

Assessment

If prevention is not an option and the patient presents with an ulcer, the initial evaluation is the most critical appointment. A comprehensive history is imperative, taking care to assess the patient's general health and the ulcer history. Many of these patients have co-morbidities which may delay wound healing and also result in poor nutritional status, clinical depression, and crutches in their social lives such as tobacco or alcohol.

The patient's health regarding their diabetes history and management is critical. Hyperglycemia is thought to be important regarding wound healing although there is a lack of direct evidence linking hyperglycemia and inadequate healing. Nephropathy is reported to have a three-fold increase in the risk of lower-extremity amputation in the diabetic patients. (9)

Factors that affect macro-vascular status such as hyperlipidemia, coronary artery disease, cerebral vascular accident, or intermittent claudication certainly increase the risk of delayed wound healing. Micro-vascular status is also a concern and a history of neuropathy, nephropathy, retinopathy, gastroparesis, and sexual dysfunction can be indicative of this process. (16) A nutritional assessment is also important whereby hypo-albuminemia is also an indicator of delayed wound healing. Pre-albumin is a better indicator of protein malnutrition than albumin, which is likely a better indicator of nephropathy than wound healing complications. Tobacco has been shown to increase the rate of incisional wound infections (17), but there is little evidence demonstrating impaired wound healing in the neuropathic ulcer. However, tobacco does negatively impact endothelial and smooth muscle microcirculation to the skin. (18) Alcohol can impair the patient leading to an increase in non-compliant behavior and in clinical depression, which is already present in many of the neuropathic diabetic patients. (9)

Information regarding the ulcer history is valuable since the duration of the ulcer can affect the healing potential. The longer the ulcer remains open the greater the risk of infection, including osteomyelitis, which has a significant impact on the treatment necessary. Prior treatment therapies and history of previous ulcers will likely provide information regarding healing potential.

The physical examination must include a thorough evaluation of the wound. The presence of infection is likely the most critical local issue while the presence of vascular disease is the most pressing issue proximal to the ulcer. The size of the wound, wound grade, and duration of the wound has been implicated as factors affecting wound healing. (19) The evaluation of the ulcer base and margins determines the presence of necrotic tissue or fibrous tissue impedes healing. Sinus tracts and undermining may indicate exposed bone or osteomyelitis, while probing to bone has a positive predictive value of 89% for osteomyelitis. (20) A negative probe-to-bone test however has a negative predictive value of 56% and does not necessarily indicate the absence of osteomyelitis. Therefore a negative result should be verified with further diagnostic testing such as radiographs, magnetic resonance imaging (MRI), or bone scans if MRI is contra-indicated. (21) Ultimately the definitive diagnosis of osteomyelitis is made via bone biopsy.

Arterial supply should be assessed beginning with palpation of pedal pulses. An ankle-brachial index and/or

Table 2. Wagner Classification

Wagner Classification System	
Grade	Lesion
0	No ulcer but pre-ulcerative due to deformity or callus
1	Superficial ulcer
2	Deep ulcer to tendon or joint capsule
3	Deep ulcer with abscess, osteomyelitis or joint sepsis
4	Local gangrene – forefoot or heel
5	Gangrene of the entire foot

Table 3. University of Texas at San Antonio Diabetic Wound Classification System.

Presence of infection or ischemia	Grade	Wound Depth			
		0	1	2	3
	A	Pre- or post-ulcerative lesion with complete epithelialization	Superficial ulcer; no tendon, bone or capsule involvement	Ulcer penetrates to tendon or capsule	Ulcer involves bone or joint
	B	A + INFECTION →			
	C	+ ISCHEMIA →			
	D	+ INFECTION & ISCHEMIA →			

toe-brachial index should also be performed. If suspicion of vascular insufficiency remains then additional testing such as transcutaneous oxygen pressures, segmental pressures and skin perfusion pressures are done. Lastly, angiography or magnetic resonance angiography with a vascular consultation is necessary should insufficiency be diagnosed or suspicion remain. (22) Ulcers can be graded on a number of classification systems. The most commonly used is the Wagner classification, which is sometimes necessary for reimbursement purposes. (Table 2) However the classification fails to account for some independent risk factors and lacks prognostic value. The University of Texas Diabetic Foot Ulcer Classification accounts for these faults in the Wagner classification and its validity as related to prognosis has been verified. (23) (Table 3)

Assessment of the patient's foot from a biomechanical standpoint is important to assess deformities and causes of increased plantar foot pressures, such as the presence of a cavus foot, Charcot foot, or an equinus deformity. Callus patterns are a good indicator of areas of abnormal pressure and therefore of areas future risk of ulceration.

Treatment

The primary goal is to achieve rapid wound closure to prevent the increased risk of co-morbidities, infections, hospitalization, and amputations. (22) The main tenets of treatment are off-loading of the foot to decrease plantar pressures and eradication of infection. (9) Through deceleration of the foot for ground contact and shortening the time of foot contact with the ground, plantar pressure and strain rate decrease, which in turn eliminates further micro-trauma and undue pressure on the neuropathic area. The gold standard for accomplishing this is the total contact cast (TCC), which is unfortunately cumbersome and time consuming to apply. The instant TCC, which is a modified removable walker, is certainly easier but less compliance and therefore decreased results are observed. Alternative methods relieve plantar pressures to varying degrees but there is little evidence to support their use.

Eliminating infection is also critical to success. Without proper control of infection ulcer healing will not occur. It is important to keep in mind that deep cultures are recommended when there are clinical signs of infection or sensitivities are needed for directing antibiotic therapy. (22) Clinical signs of infection may be absent secondary to the potential of an impaired host response. Therefore secondary signs such as exudates, delayed healing, wound breakdown and friable or discolored granulation tissue are more likely to aide in the diagnosis of infection. The Infectious Diseases Society of America classification has been validated and serves as a guide for infection severity and treatment protocols. (24, 25) Osteomyelitis frequently underlies infected diabetic foot ulcers and greatly increases the challenges in treatment and successful healing. Debridement of the infected bone is ideal; however, IV antibiotics may be used if the patient's health precludes surgical intervention.

Debridement, while intuitive and predominantly supported anecdotally, is done routinely and recommended as the standard of care. (22) The purpose of debridement is to remove devitalized tissues, biofilms, and wound margins to stimulate growth factors necessary for healing. Weekly surgical debridement is recommended since healing is more rapid than less frequent debridement. (26)

A moist wound environment has traditionally been considered the conventional therapy and is frequently used as the control group in studies evaluating the efficacy and safety of new therapies. Moist wound

therapy (MWT) is most commonly accomplished with a wet-to-dry saline gauze, which unfortunately causes non-selective destruction of the tissue. Other dressings used to create this environment consist of hydrogels, alginates, and other dressings to affect the cytokines, biofilms, and matrix matelloproteinases that negatively impact cell proliferation and growth factors necessary for re-epithelialization.

Advanced therapies have become a newer means of treatment over the past five to ten years. These developments have typically been reserved for severe, complex wounds and those not healing. Unfortunately, they are not employed as part of the treatment regimen frequently or early enough in many cases. If a wound is less than 50% healed after 4 weeks, the likelihood of healing is significantly reduced (27, 28). With this in mind it is recommended that after four weeks with less than 50% wound area reduction, advanced therapies should be considered. (22) Examples of advanced therapies human skin equivalents, recombinant growth factors, vacuum-assisted closure, hyperbaric oxygen therapy, and electrical stimulation.

The advanced therapies with the highest levels of evidence are the recombinant growth factor PDGF and two human skin equivalents. One is a bi-layered skin equivalent (Apligraf®, Organogenesis) and the other a human derived fibroblast dermal substitute (Dermagraft®, Advanced Biohealing). All three of these therapies have demonstrated significant success in expediting ulcer healing and increased percentage of patients achieving complete re-epithelialization. (29, 30, 31) Negative pressure wound therapy (NPWT), or vacuum assisted closure, has recently demonstrated greater efficacy with fewer secondary amputations that traditional MWT in a multi-centered, randomized controlled trial. (32) Additionally NPWT has shown a decrease in resource utilization with fewer surgeries and dressing changes than MWT as well as decreased cost of care. (33)

Conclusion

Diabetic foot ulcers are most commonly the result of a combination of factors, with peripheral neuropathy at the center of the problem. While prevention is the best means of treatment, proper control of this chronic condition and reliance on a collaborative effort of specialists is critical. A thorough assessment of the patient and the ulcer allows for proper management and rapid healing of the ulcer. Should any delay in healing occur, a number of advanced therapies should be used to increase the likelihood of healing this complex and challenging problem that can have devastating consequences.

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SHINGLES IN IMMUNE COMPETENT PATIENTS

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Abstract:

Shingles is an infectious disease caused by the Varicella Zoster virus.(1) During an initial infection, Varicella Zoster appears as chicken pox. After the resolution of the disease, the virus lays dormant in the human body for many years.(1) However, it can reactivate affecting the body as a painful vesicular rash in a dermatomal fashion, usually unilateral.(1) In this article, we examine a case of an 8 year old boy in which he presented with a four day history of a burning sensation of the right hip followed by a vesicular eruption in the same region. It is important to remind health care practitioners that with such a rash, Shingles must be included in the differential diagnosis and workup. It is also important to reassure patients that as long as there is no underlying immune compromise, the process is generally benign and is treatable.

Case Study:

Our patient t is an 8 year old Hispanic boy brought in by mother who presents with a four day history of a rash. The patient stated that four days prior to presentation, he noted a burning sensation on his lateral right hip which later became pruritic. Patient stated that a rash developed on the affected area the night before he presented to clinic. Mother denied any previous episodes of this presentation. She also denied any history of allergies or contact dermatitis in her son. Mother also denied any diagnosed immunologic compromising diseases such as AIDS or frequent infections in the child. His past medical history is only remarkable for a chicken pox infection while less than one year old. History is unremarkable for organ transplant, use of any immune suppressants by the child, or any family members who use immune suppressants that could have lead to an accidental ingestion by the child. The patient also denied history of trauma in the affected region.

The child's vital signs were within reference ranges. Upon physical examination, a 6 cm x 6 cm erythematous vesicular pustular rash was noted slightly below his inguinal ligament on the right hip (figure 1). Physical examination revealed a well-developed, well-nourished adolescent male. Examination of the patient's right hip revealed no motion restriction, muscle strength of 5/5 in lower extremities, and intact light touch sensation bilaterally intact in upper and lower extremities. There were no eccyhmosis or evidence of hip trauma. A CBC was ordered along with a Varicella anti IgM/IgG titers, and an HSV1/2 anti IgG titer to rule out a herpes simplex infection. Biopsy of the lesion was not done.

Figure 1 – A 6 cm x 6 cm diameter maculopapular rash with an erythamatous base and herpetic characteristics. Rash is 1 day old and found on a 8 year old boy's right hip.

A diagnosis of shingles was made, the mother was reassured that the rash is benign, and her son was sent home on a ten day course of acyclovir. Five days later, the mother returned with her son for follow up. Upon examination, the rash was still 6 cm x 6 cm and exhibited dry ruptured vesicles (figure 2). Lab results were discussed with the mother. She was instructed to return to clinic if symptoms worsen or fail to resolve.

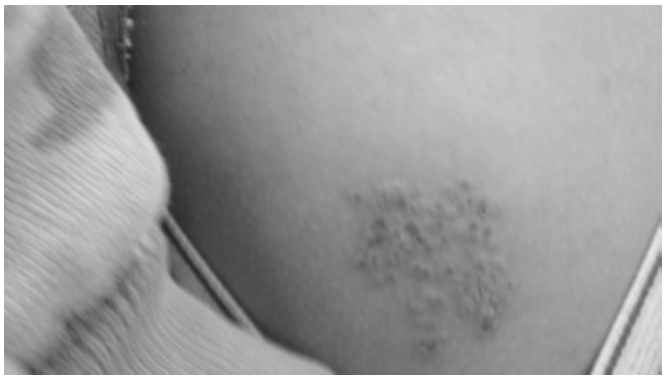

Figure 2 –A 6 cm x 6 cm maculopapular rash with an erythamatous base and herpetic characteristics. Rash is 6 days old in the picture with dry, ruptured vesicles noted on the right hip of an 8 year old boy.

Lab Results:

WBC (x1000 / ul)	9.6
RBC (x 10^6 / ul)	4.69
Hgb (g/dl)	12.3
Hct (%)	37%
Neutrophils	61.5%
Lymphocytes	27%
Monocytes	7.1%
Eosinophils	3.8%
Basophils	0.6%

Antibody Titer	Value	Reference
VZV Anti IgG	2.61	< 0.90 Neg, 0.91 – 1.09 equivocal, > 1.10 pos
VZV Anti IgM	0.12	< 0.90 Neg, 0.91 – 1.09 equivocal, > 1.10 pos
HSV 1 Anti IgG	0.27	< 0.90 Neg, 0.91 – 1.09 equivocal, > 1.10 pos
HSV 2 Anti IgG	0.10	< 0.90 Neg, 0.91 – 1.09 equivocal, > 1.10 pos

Discussion:

Epidemiology and Risk Factors:

It is believed that one million people are affected by shingles annually in the United States. (2) The incidence has been constant over the past twenty years. (3) shingles is a condition that can affect up to 34% of those who were previously infected with chicken pox. (4) Risk factors include: advanced age, a recent Varicella vaccine, the use of immune suppressants, TNF – alpha for Pediatric or adult Rheumatoid Arthirits, AIDS or pathologic immune compromise, maternal infection while in utero, or chickenpox infection before 1 year of age. (5)

Although rare in immune-competent children, an estimated 0.04 out of 10,000 cases of shingles occur in the pediatric population from ages 0-14. (5) Literature shows that the relative risk of shingles arising in children who had chickenpox before during the first year in life can be up to 20.9 times higher than children who had it after turning one year of age. (6) In such cases, shingles appears later in life in preadolescent years and up to twenty years of age. (6) The study also showed the majority of cases involved the thoracic dermatomal distribution (65.2%), while only 11.1% involved the lumbar region; this trend is also found in the adult population although there is a higher incidence of cranial nerve involvement among adults at 13.1% compared to only 5.2% in the pediatric population. (6)

Genetics can play a role in the development of Shingles. In one case-control study, it was shown that 35% of those infected with shingles have recalled a family member that developed shingles, as compared to 11% who have not reported a family history. (7)

Pathophysiology:

After an initial infection with Varicella Zoster, the virus travels along the nerves of the affected area of the skin. (1, 8) It then lays dormant in the cytoplasm of dorsal root ganglia cell bodies. (1, 8) Upon reactivation, the virus replicates in the nucleus leading to inflammation and possible necrosis of the dorsal root ganglion. (1, 8) The virus then travels to the skin area specific to the affected dorsal root ganglion. (9) Therefore, the rash appears in a contained area on a patient's skin, usually unilateral, and follows a specific dermatome. (1)

Even though shingles can signify an underlying immune

compromised state, it can also affect those with an intact immune system. (1) Cell-mediated immunity maintains the virus in a latent phase. (8) Literature shows that patients affected by hypogammaglobinemia had no increase in the incidence of shingles which minimizes humoral immunity role. (8) Advanced age, or any weakening of the immune system such as stress can activate the infection (1).

As a result of an acute immune response to the reactivation of Varicella Zoster, anti-IgM varicella antibodies are produced. (4) A positive IgG antibody indicates previous immunity or infection, but could also be indicative of active infection. (4) According to lab results, our patient tested positive for Varicella IgG but negative for IgM. Our patient presented during an early phase for shingles, therefore the patient's immune system has not produced enough IgM to be detected on laboratory results. Literature has shown that it takes roughly 10 to 14 days for levels of IgM to be detected. (4) The patient's CBC results including the differential rule out potential immune deficiency that would affect humoral response.

Pediatric shingles appears with the same clinical symptoms as adult shingles with nearly similar risk factors. The rash usually appears in a specific (or part of) a specific dermatome. (1,5,9) It is preceded usually by a pruritic and burning sensation in the same area anywhere from four to seven days before where the vesicular rash appears. (9) The rash undergoes the usual stages of herpetic lesions: preceding burn and itching followed by a pustular and vesicular rash with an erythematous base. The vesicles could either form bullae, or rupture resulting in serous fluid leakage or scar formation. (1,8) Scars can persist for weeks before the affected area heals, and in some cases scars can remain for prolonged periods of time (9). In twenty-five percent of cases, a post-herpetic neuralgia appears in the same nerve affected by the virus. (5)

Prognosis & Treatment:

Pediatric Shingles follows a more benign course, is less symptomatic, and the prognosis is much more favorable compared to adult shingles (10). This in turn, decreases the chance of developing post herpetic neuralgia. (10) Treatment standards suggest using acyclovir for seven to ten days, although no studies have been shown to demonstrate a cost/efficacy ratio for the duration of treatment using acyclovir. (11) For patients who develop a post-herpetic neuralgia, gabapentin has been shown to help with symptoms. (10) In a study of 859 patients of all age groups, only twelve percent developed long term complications (12). Those complications included a post herpetic neuralgia, bacterial infection, ophthalmalgia including uveitis and keratitis (if trigeminal nerve involved), motor neuropathy, and meningitis (12). Although greater than 50% of complications arose in patients greater than sixty years old (12), no studies of complication incidence in children has been performed.

Conclusion:

Shingles is common in the United States and affects up to one million people annually. (3) The disease presents as a vesicular rash that is preceded by few days of neuralgia, itching, or burning sensation in the affected area. (1) Our patient presented with several days of neuralgia followed by a 5 cm x 5 cm erythematous vesicular rash. It is important for a health practitioner to have a high index of suspicion when a dermatomal rash appears with a sudden onset, particularly in those who were previously infected with Varicella Zoster. Even though Shingles generally affects adults, the same index of suspicion should be utilized in a pediatric patient. In our case, the patient had chicken pox prior to one year of age, which is a risk factor for a pediatric patient. Accordingly, if a patient presents with shingles, it is important to offer reassurance to patients that the disease is relatively benign, as it could be worrisome for the patient. The standard current regimen for treating shingles is a ten day course of acyclovir, (11) and gabapentin for post herpetic neuralgia. (10)) The same standard applies for the pediatric population although more research is needed to justify cost/efficacy ratio of acyclovir. (11) There is limited literature available about the prevalence of shingles in the pediatric population including long term complications. This case serves to examine the available data in order to emphasize the necessity of greater research surrounding shingles and as a reminder to suspect shingles when patients present with a rash similar to our case.

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ON CONTINUITY OF CARE

Which Continuity of Care Model should the U.S. Adopt? An Examination from a Cost Effectiveness Perspective

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Abstract

The purpose of this study was to explore the cost effectiveness of continuity of care in the chronically ill geriatric population, and to propose methods to increase the continuity of care within the U.S. healthcare system. Results of the literature review were that continuity of care is cost effective for this population. Given these results, it is imperative to institute a continuity of care model within the U.S. at the current time. The type of model to be instituted and the methods of implementation are discussed.

Introduction

In the current political climate of health reform, the focus of healthcare has changed towards a more efficient and effective system. Although the United States has made a large economic investment of 16.2% of the GDP in healthcare, it has obtained a poor return on investment as shown through its health outcomes, which rank it 25th in the world, when compared to other developed countries.(1) One solution to this problem is to focus on disease management of chronic conditions as a vehicle for achieving the goal of more cost effective health care.

Disease management is defined as "an approach to patient care that emphasizes coordinated, comprehensive care along the continuum of disease and across health care delivery systems".(1) As such, disease management has the dual capacity to improve health outcomes and to provide an economic benefit. In the meta analysis performed by Ofman and colleagues, 45% of the studies illustrated that disease management helped to improve the control of chronic disease as well as patient adherence to the treatment regimen.(1) Krause and colleagues examined the economic impact of disease management, through the study design of meta-analysis, and found that disease management programs were moderately effective in terms of economics, with an average effect size of 0.3.(2) How, then, can disease management be improved to render it more cost effective?

A concept that is integral to disease management is coordination, or continuity of care. Continuity of care, as defined here, refers to a growing knowledge of the patient by those providing the care. Such knowledge is obtained is from the other health professionals who have provided health care to the patient, in which case it is classified as informational continuity. (3) Given this definition of continuity of care, it makes sense, therefore, to turn to this facet of disease management as an opportunity for improvement to render it more cost effective. Indeed, in their report Crossing the Quality Chasm, the Institute of Medicine explicitly referred to the importance of continuity of care, through their recommendation that "clinicians and institutions should actively collaborate and communicate to ensure an appropriate exchange of information and coordination of care". (4)

Statement of the Problem

Given that coordination of care is critical to disease management, there appears to be a significant

problem with the current state of care coordination. Davis and her colleagues at the Commonwealth Fund found that the average score across five indicators of care coordination for the U.S. decreased from 72 to 71 from the years 2006 to 2008.(5) Bodenheimer and colleagues also confirmed this finding : for example, they found that coordination between primary care physicians and emergency department was ineffective, as 30% of adults seen in the emergency department in 2004 reported that their regular physician was not informed about the care that they received.(6) Coordination between hospital based physicians and primary care physicians was also problematic according to Bodenheimer and colleagues, as only 3% of primary care physicians were involved in discussions with hospital physicians about patient's discharge plans, while only 20% were notified that the patient had been discharged and received a discharge summary.(6)

Given the problems with continuity of care, what impedes our ability to fix this issue? The answer is that barriers to continuity of care are abundant. One of the primary barriers to continuity of care is overstressed primary care. With large panels of patients, and a growing number of tasks to perform, primary care physicians can no longer provide high quality, short term, long term and preventive care during a fifteen minute visit, let alone perform care coordination functions. A second barrier that builds upon the first is dysfunctional financing.(6) Most dollars are paid to physicians on the basis of quantity rather than quality and on face to face visit time, rather than the between visit time required for care coordination. Therefore, physicians have no financial incentive to offer the discharge care needed to smooth the transition between hospital and home. A third barrier is lack of interoperable computerized records, which make it easier and faster to transfer patient information between providers.(6) Lastly, a lack of integrated systems of care makes it more difficult to coordinate care. According to Bodenheimer and colleagues, 47% of private physicians work in practices of one or two physicians, rather than in a group of twenty or more physicians. The likelihood of working with the same physician who treated your patient in the emergency department is low, thus making it more geographically difficult to coordinate care.(6)

What populations are most affected by lack of care coordination? According to Nutting and colleagues, in a study of 4,454 outpatient visits, the higher the number of chronic conditions and medications, the higher the value placed on continuity ($p < .001$).(7) Studying the chronically ill also makes sense from the point of view of prevalence, mortality and economic burden. According to Johns Hopkins University, in 2005, 133 million Americans had one or more chronic conditions, a number which is projected to increase by more than one percent each year through 2030.(8) However, chronic conditions are not only common, but have a significant impact on long term health. According to the CDC data from 2005, the age adjusted mortality rates for liver disease is 8.9, congestive heart failure is 94.5, coronary artery disease is 154, diabetes is 76.7, chronic obstructive pulmonary disease is 250.4, and end stage renal disease is 14.3.(9) The cost of the chronic disease burden is not readily discounted; according to Johns Hopkins University, 83% of health care spending in 2001 was attributable to the care of people with one or more chronic conditions.(8) Consequently, it is key to examine not only coordination of care as a mechanism to improve cost effectiveness of health care but to place it in the proper context of chronic conditions.

Purpose of the Study

This study intends to explore the cost effectiveness of continuity of care in the chronically ill and to propose methods to increase the continuity of care within the U.S. healthcare system.

Importance of the Study

Research in the area of continuity of care for the chronically ill suggests that it has comprehensive benefits. Bennett, Fosbinder and Williams first examined this issue in 1997.(9) The study population was patients coded in two DRG groups, either craniotomy or cerebral hemorrhage during the acute phase of neurologic treatment. The intervention was coordination of care as performed by a nurse. Bennett and colleagues found that the intervention group experienced a lower length of stay at the six month intervention mark that was two weeks shorter than the control group, and saved $28,000 overall compared to the control group.

In 2005, Naylor and colleagues at the University of Pennsylvania examined this topic in a population with congestive heart failure.(10) The intervention

was continuity of care as provided by an advance practice nurse. Naylor and colleagues found that increased continuity of care, as provided by an APN, resulted in 13.7% lower rate of rehospitalization, 110 more days of event free survival as well as a

cost savings of $4845 per patient. Thus, it is evident that the hypothesis of a positive correlation between continuity of care and cost effectiveness of care is supported.

Scope, Limitations, and Delimitations of the Study

This study will analyze which continuity of care models are most economical and effective, and will propose which models should be applied to the U.S. healthcare system. The major delimitation of this study is that the population studied is limited to those over age 65 with chronic diseases such as congestive heart failure, and coronary artery disease. Another delimitation is the time period of this study, which will be limited to the past twenty years, as studies prior to this time can be deemed less relevant to the U.S. because of the differing sociopolitical and economic influences present.

Review of the Literature

This review of literature will focus on the impact of continuity of care models on the economics and outcomes of patient care, and, then, will analyze which models will best improve the level of continuity of care within the U.S.

The first model of continuity of care involves telephonic intervention in addition to a home visit by a care provider. Rich and Beckham first examined this topic at Jewish Hospital in 1996 through examining readmission, quality of life and costs of care for 282 patients, 70 years of age or older with congestive heart failure over three months.(11) Data was collected between July 1990 and June 1994. Continuity of care was defined as by intensive follow-up with the hospital's home care team, supplemented by individualized home visits and telephone contact with members of the study team. The study design was case control, with the intervention defined as continuity of care. Rich and Beckham found that high continuity of care reduced the number of readmissions by 56.2% in the treatment group; In addition, in a subgroup of 126 patients, quality of life scores improved more from baseline for patients in the treatment group (p=.001). In terms of costs of care, continuity of care resulted in a cost reduction of $460 per patient in the treatment group. In 2005, Anderson, Deepak and Zarich investigated the effect of continuity of care on patient health outcomes and costs of care using a case control design.(12) They recruited 276 patients with congestive heart failure, and studied them over six months. Continuity of care was provided by an experienced cardiac nurse educator, who interviewed patients by telephone within two weeks of hospital discharge, and by home health care nurses, who visited patients twenty times over six months; each visit consisted of a formal cardiac evaluation, patient education regarding their disease, diet and activity, and continual assessment of patient compliance with diet, medication and weight monitoring. The intervention group with high continuity of care experienced a marked reduction in six month readmission rates from 44.2% to 11.4% (p=.01). The average total cost savings for each subject in the intervention group was $1541, based on the decreased utilization of both skilled nursing services and home health care during outpatient follow-up.

The next study done in the area of telephonic intervention and home care visits, was that of Coleman, Parry, and Chalmers, who examined the effects of continuity of care in 750 elderly adults with one or more chronic conditions from September 2002 to August 2003.(13) The design of the study was case control . The intervention consisted of tools to promote cross communication and guidance from a transition coach, seventy two hours after discharge. The results were that the intervention group had lower rehospitalization rates at thirty days of 8.3 % vs. 11.9% for the control, and the lower mean hospitalization costs at 180 days of $2058 for intervention patients compared to control at $2546. Again, it is clearly evident that the second model of continuity of care, telephone follow up and home visits, is cost effective.

Yet another type of continuity of care model includes only home visits by a qualified provider. The first study in this area was performed in 1998, by Stewart and Horowitz.(14) This study was initiated within a tertiary referral hospital in Australia. The study population was 97 patients with NYHA Class II, III or IV congestive heart failure. The study period was six months. The continuity of care intervention consisted

of a single home visit by a nurse and a pharmacist. The results were that patients in the intervention group had fewer unplanned readmissions, (p=.03) and few our of hospital deaths (p=.11). Patients in the intervention group also had fewer days of hospitalization (p=.05). In addition, the mean cost of hospital based care tended to be lower for the intervention group at 3200 Australian dollars compared to 5400 Australian dollars for the control group. Annttila and Huhtala further elucidated this topic in a study performed in 2000.(15) The population examined was 204 elderly patients with chronic conditions observed from 1992 to 1994. The study design was a case control type. The intervention was follow-up care provided by home health nurses two and four weeks post discharge. The results show no differences in admissions to nursing homes, but a 52% decrease in cost of care for the intervention group.

The next study done in the area of home visits was performed by Naylor and Brooten in 2004. (16) The study design was a randomized controlled trial conducted over 52 weeks. The setting was academic and community hospitals in Philadelphia, where 239 patients with congestive heart failure were recruited for participation. The intervention was a three month advanced practice nursing directed follow up protocol. The results were that the intervention group had fewer readmissions (p=.047) and lower mean total costs of $7636 compared to $12481 for the control group (p=.002). However, intervention patients also demonstrated some short term improvements for quality of life over twelve weeks (p<.05) and patient satisfaction over six weeks (p<.001). Overall, the continuity of care model which consists of home visits is cost effective. The only study which questioned this premise in terms of the model's effect on health outcomes is that of Annttila and Huhtala in 2000, which showed no effect on clinical outcomes.(15) However, since this study was done in Finland, the quality of care there is significantly different from that of the U.S.; therefore, had the same study been conducted in the U.S., differences between the two groups may have become evident. The only study which questioned the premise that this model has a positive economic effect was that of Stewart and Horowitz in 1998, who found the economic differences between the intervention and control groups to be statistically insignificant.(14) However, since the sample size Stewart and Horowitz utilized was small, this lack of statistical significance can be attributed to the lack of power of the study. In short, then, enough evidence exists that this model has a positive effect on both the economics and clinical aspects of patient care.

Another more complex and common model of continuity of care is that of case management. Case management is a trifold system of health assessment, delivery, and coordination that is designed to meet patients' health needs.(17) The goal of case management is to provide continuous high level care that will enhance the patient's quality of life while simultaneously containing costs.

A key study examining the case management model was that of Riegel and Carlson in 2002, who performed a randomized controlled trial, recruiting 358 patients with heart failure from two southern California hospitals.(18) The study period was six months. The intervention was case management via telephone. Patients were first phoned within five days of discharge, and thereafter at a frequency guided by case manager judgment. On average, patients received seventeen phone calls from the case manager over the study period of six months. The results were as follows: the heart failure hospitalization rate was 45.7% lower in the intervention group than the control at three months (p=.03) and 47.8% lower at six months (p=.01). The number of hospital readmissions for heart failure was 36% lower in the intervention group at six months (p=.03). The average numbers of days spent in the hospital was 46% lower for the intervention group at six months as well (p=.03). Lastly, the inpatient costs for the intervention group was 45.5% lower at six months (p=.04), resulting in a cost savings of $1000 per patient for the intervention group. It seems clear, then, that the case management continuity of care model has a positive impact on both the economics and clinical outcomes of patient care.

"Guided care" is yet another complex alternative to the case management continuity of care model. Guided care is provided by a practice based team that includes a registered nurse, 2 to 5 physicians, and the other members of the office staff. This team provides eight clinical services to patients.

The primary study of relevance done in this area was conducted in 2009 by Leff and Reider.(19) The type of study was a cluster randomized controlled trial which followed 904 elderly chronically ill patients

from 14 primary care teams through hospitalizations. The time period of the study was June 2007 through February 2008. The intervention was Guided Care after discharge. The results were that the intervention group experienced on average 24% fewer hospital days, and 15% fewer ED visits. These differences translate into a cost savings per patient of $1364. Thus, although Guided Care is more complex to implement, it seems to be worth the risk, for it pays dividends in terms of decreasing cost and increasing effectiveness of health outcomes.

Which model is the best? It is necessary to turn back to an overview of all of the models in order to be better equipped to make such a decision. There were six models of continuity of care that were discussed. The first model was telephone follow-up in addition to a home visit. The second model was home visits alone. The third model was case management. The fourth model was guided care. The only model that had conflicting evidence of its validity in terms of economic and clinical benefit was the second model. Again, the study done by Stewart and colleagues, which questioned this hypothesis, had too small of a sample size, to render it of statistical significance.(14) The other study which questioned this hypothesis was that of Annttila, who showed that home visits were not positively correlated with improved clinical outcomes.(15) However, these results can be interpreted with skepticism, given that the study was conducted in Finland, where the standards for quality of care differ greatly from the U.S., thereby raising the issue that a change of location to the U.S. may have changed the results. However, as there is a preponderance of evidence supporting this model, it is still valid to consider it as a viable model of continuity of care. Therefore, there are four models of continuity of care that appear to be cost effective in terms of patient care.

Conclusion

As this literature review illustrates, continuity of care has a beneficial effect on both the cost and effectiveness of health care for the geriatric population. However, four models of continuity of care exhibit this effect: therefore, which models should be implemented in the U.S., and in what manner should the implementation occur?

In order to effectively answer this question, it is necessary to turn to an examination of best practices in coordination of care. According to the evidence compiled by Chen and colleagues in 2000 regarding "best practices", successful care coordination models were of two types: case management and disease management.(20) Case management programs were non disease specific programs, and targeted patients at high risk of suffering hospitalizations because of complex social and medical vulnerabilities. Disease management programs, on the other hand, targeted patients whose main health problem was a single diagnosis.

Both sets of programs had three steps in common, which were:

1. Assess and plan.
2. Implement and deliver.
3. Reassess and adjust.

Each step was further broken down into a set of tasks.

The first step, Assess and Plan was broken down into four smaller tasks:

1. Uncover all important problems.
2. Address all important problems and goals.
3. Draw from a comprehensive arsenal of proven interventions.
4. Produce a clear, practical plan of care, with specific goals.

The second step, Implement and Deliver, was broken down into four smaller components as well:

1. Build ongoing relationships with the PCPS and with other providers.
2. Build ongoing relationships with patients and families.
3. Provide excellent patient education.
4. Make certain that planned interventions get done.

The third step, Reassess and Adjust, was broken down into five components:

1. Perform periodic reassessments.
2. Be accessible.
3. Nurture the relationship with PCPs and providers.
4. Nurture the relationship with patient and family.
5. Make prompt adjustments to the plan of care as needed.

In the first step, in both the disease management and the case management programs, the interventions used consisted of at least a home visit by a trained provider such as a nurse in addition to other methods. Telephonic intervention by itself was ineffective. As such, any of the continuity of care models which incorporated home visits seem to be effective, under the guidelines for best practice.

While best practice identifies the methods of how to implement continuity of care models, it does not answer which model would fit best in the U.S. It is necessary to turn to Rosenbach and Young to flesh this issue out further.(21)

Rosenbach and Young identified three main care coordination models:

1. A centralized team model, in which team members are located at a central office, and coordinate services for all patients.
2. A regionalized model that coordinates services only for patients identified within geographic boundaries;
3. A provider based model that coordinates care for all patients served by a specific provider entity.

Given that this model would have to be implemented on a national level, a provider based model appears to be the most efficient. When considering the models of continuity of care previously discussed, one model obviously fits this mold: the Guided Care Model. The Guided Care Model is provided by a practice based team that includes a registered nurse, 2 to 5 physicians, and the other members of the office staff. This team provides eight clinical services to patients.

For each patient, the guided care nurse performs the following eight services:

1. Creates an evidence based care guide
2. Performs a comprehensive assessment at home
3. Monitors and coaches the patient monthly
4. Coordinates the efforts of all the patients' providers
5. Smooths the patient's transitions between sites of care
6. Promotes patient self-management
7. Educates and supports family caregivers
8. Facilitates access to appropriate community resources.

A "Guided Care" Model overcomes one of the primary stumbling blocks to care coordination: namely, the inability to contact the treating physician, because they are already incorporated into the existing team. A second obstacle to overcome in care coordination is the ability of the coordinator to identify patients' clinical problems and choose effective interventions. (21) Once again, the guided care model overcomes this obstacle because the care coordinator under this model is a nurse.

Wagner's Chronic Care Model from 1996 further elucidates how to implement care coordination for chronic conditions in the U.S. across a variety of healthcare environments.22 What becomes most evident from this model that providers must be paid for performance. This change provides the driving incentive to achieve coordination of care on a national level. Secondly, electronic health records must be adopted universally to facilitate transmission of medical information to providers separated by geography.

This scenario appears to be readily facilitated by the passage of health reform. Under President Obama's plan, Medicare plans receive bonuses based upon quality ratings, and Medicare payments are reduced to hospitals for preventable readmissions. Both of these actions have the same effect: they create a forceful incentive for high quality care. This in turn, would create a "push" for continuity of care, as evidence based medicine has shown the impact of continuity of care in improving quality of care. President Obama has even specifically noted the need for continuity of care in his new proposal by establishing the Community based Collaborative Care Network Program to support health care providers in coordinating and integrating health care services.

With the national stage set for acting as an impetus to coordinate care, the only question remains: will the continuity of care models work in practice? The only existing completely integrated model on a state level that is similar to the Guided care model is that of Wisconsin.23 The Wisconsin Partnership program is characterized by a close working relationship among members of the interdisciplinary care team who manage access to the full range of primary, acute and long term care services. Nurse practitioners lead the team and serve as the liaisons between the team and physician. The nurse practitioner accompanies the patient to the office visits and discusses the care plan

with the physician. A social worker is also part of the team, and provides information about benefits and services. A review of this program has found a very low incidence of emergency room visits and hospital admissions for ambulatory care sensitive conditions. Furthermore, for patients enrolled in this program, number of hospital days dropped from 5 per 1000 to 2.1 after participation in this program.23 As such, it is self-evident that the practical implementation of an integrated continuity of care program supports the existing evidence from clinical trials. Therefore, it is imperative for the United States to move forward on the path to becoming more efficient and effective by adopting a guided care model to enhance continuity of care as soon as possible. Then, our healthcare system will truly be on its way towards maximizing its potential.

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THE NEUROLOGIC EFFECTS OF NOISE POLLUTION ON HEALTH

A Year Long Case Study from and Osteopathic Perspective

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"I have never found a companion that was so companionable as solitude."
—Henry David Thoreau

A. T. Still, M.D., D.O., the founder of osteopathic medicine, understood the importance of environment on health, preaching that fresh air, good slumber and healthful environment all influenced health of an individual. He says it best in his autobiography "Through the darksome night I lay enchained by slumber's thrall, but with the first faint flushing of the dewy morn I arose and wandered forth. All Nature seemed to wait in hushed expectancy. With the iron hand of will I barred the gates of memory, shut out the past with all its old ideas. My soul took on a receptive attitude, my ear was tuned to Nature's rhythmic harmony" Through such poetic prose AT Still expressed the foundation of sleep for a healthful life. In 1908 when this was written, the automobile was just gaining in fashion, and the machine age had not yet hit its stride. What A.T. Still didn't know was how that machine age would grow into a menacing health threat. Noise as an environmental pollutant has become an increasing threat in industrialized nations. Aside from the obvious effects of noise pollution, such as annoyance and hearing loss over time, the constant exposure to urban noise produces negative effects on the nervous system and health. To date very few studies are being done on the health impact of noise pollution in urban environments, especially with a "noise naïve" population. My intention is to underscore the need to assess environment in a thorough history and physical as a contributing factor to health of the patient.

Personal experience would soon allow me to understand the environmental impact to which Dr. Still referred. Through this article I would like to use my experience as an example, as it was quite profound in understanding the influence of urban congestion, noise and high density living on an individual not acclimatized to such an environment.

In 2010 I had a major shift in my career which involved leaving my family and my small rural community practice to become a professor at a university. In order to be trained for my new job, I moved to an urban setting in a high density living situation for a year. This was not where my permanent employment would be and after a year I would leave this environment. During the last 10 years I have lived in a rural setting in which the major noise was coyotes and owls at night, although I have often visited and spent time in urban centers with no ill effects. Surely I would adapt to the environment.

In addition I chose to live without a car in this environment, and to live in a higher density complex close enough to be able to walk to my employment, complicating further the access to transportation to get away from the constant noise exposure. I was, in essence, becoming a "lab rat" to the noise effect. This particular urban setting had an average to slightly increased crime rate for an urban setting

of population greater than 1 million. To give perspective, my previous residence was located on 5 acres of land with no drive- thru traffic, and no urban noise and a population density of 26,000.

The urban setting was also close to a major thoroughfare in which police, ambulance and fire vehicles would traverse, often hourly. In addition, it was in the flight path of 2 major airports, adding jet engine noise to the mix. My apartment was one block from the train tracks where passenger and freight trains passed by several times daily. There was also the "typical noise" of high density living, such as music playing too loudly, heavy footsteps in the apartment above as well as loud conversations in a communal courtyard. Landscaping equipment added additional daily noise into the equation with the popular "back pack blowers" and lawnmowers. The area was frequented by police helicopters as well, usually with increase on the weekends when crime was worse.

Noise pollution is now recognized as a major factor in overall wellbeing and health. In The American Journal of Preventive Medicine, Moudon found evidence for increase in aggressive behavior and increase in myocardial infarction, as well as reduced performance with sustained exposure. The World Health Organization has recommended a daytime level no greater that 55 dB and a nighttime level of no greater than 40 dB in order to prevent ill effects from noise pollution. To put this in perspective, a jet taking off is approximately 100 db, while motorcycle or heavy truck in traffic is 90 dB (from 25 feet). A passenger car going 65 mph or a vacuum cleaner is 70 db. Noise in a quiet rural area at night is 32-35 dB. In another study in by Paunovic et al, studying aircraft noise and blood pressure in children, found increase in blood pressure with increasing noise after controlling for confounders. Deepak Prasher studied the immunologic effects of noise and found "The acute and chronic responses of the HPA system are variable across individuals to noise as well as other stressors, but it is clear that sustained activation of the HPA system causes imbalance of the hormonal system and promotes disorders of many organs and the brain."

Neurologically it makes sense as increasing noise triggers our sympathetic nervous system into a fight or flight response. Both acute and chronic exposure to noise affects cortisol levels which are made worse by lack of sleep. Over time the effects of the sympathetic stimulation adversely affect health thru increased release of stress hormones. These wear on the organ systems over time and have ill effects on immunity. In a 2011 article on the effects of long term psychological stress, Seldenrik et al, found increase in coronary artery calcification with increased cortisol levels, underscoring this fact.

In my own experience, before I even realized the adverse effects of noise pollution as more than an annoyance, it had already affected my health. Over the course of the year, I experienced the exacerbation of one previously stable chronic condition, and the development of 4 acute conditions. Although it is difficult to rule out confounding factors, such as job and relocation stress or poor air quality, the noise exposure creating sleep disturbance I believe was a key factor. Multiple sleep deprivation studies have correlated lack of sleep to adverse health effect.

Why is noise impact not included in the typical history and physical exam when working up health risk? In my opinion it has to do with the insidious nature of noise. Just like air pollution, it is difficult to assess its impact on health. In addition, the brain becomes acclimatized to noise, or lack of noise. In my case, to move from an area where the average noise is 35dB to an area where the average is 70-100dB is more difficult than moving from one urban setting to another. In addition, certain individuals are more "noise sensitive" and noise impact is variable among individuals. How can the physician adjust for all the confounding variables?

In this instance had I known the effects of noise pollution on my physical health I would have started much earlier to counter them. Alvarsson et al, demonstrated thru skin conductance levels that nature sounds facilitate recovery from sympathetic activation after psychological stressors. Nature sounds, noise cancelling head phones, ear plugs and "white noise" can all reduce the ill effects on health. . It is up to us as physicians to ask the questions about environment when we see a patient in a downward health spiral. As physicians we must expand our knowledge of what health effects the environment has on body, mind and spirit. Population dense areas need to seek new ways to counterbalance the ill effects of noise pollution thru better regulation, and all individuals need to do their part for a healthier future. In the words of A.T. Still, "We say disease when we should

say effect; for disease is the effect of change in the parts of the physical body. Disease in the abnormal body is just as natural as health when all parts are in place." These words are as true today as they were when the sage author wrote them, words for the wise.

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SACROILIAC JOINT PAIN AND DYSFUNCTION:
A Forgotten Cause of Low Back Pain

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Introduction

Although most doctors are very familiar with herniated disc or spinal stenosis as causes of low back pain (LBP), some may not be familiar with sacroiliac joint (SIJ) dysfunction as a cause of LBP. In fact, some studies maintain that up to 25% of low back pain comes from the sacroiliac joint. However, due to the lack of effective and long lasting treatments in the past, along with controversy about the SIJ being a cause of LBP by various pain experts, SIJ pain as a cause of LBP has been neglected.

Fortunately, there has been rapid progression, especially in the last few years, on the treatment of this less known cause of LBP. In fact, there are long lasting treatment advances both in the interventional pain management and surgical fields.

In my pain practice, the SIJ as a cause of LBP is part of my differential diagnosis of many patients presenting with mechanical LBP. My routine examination of the LBP patient includes inspection of the SIJ.

Anatomy

The pelvic area includes the wedge shaped sacrum, a combination of 5 fused sacral vertebrates. The sacrum connects with the 5 lumbar vertebrates superiorly and coccyx inferiorly. The whole weight of the upper body is transferred from the sacrum to the ilium via the sacroiliac joint to the lower body. The SIJ is the largest axial joint in the body. The SIJ itself is a true synovial joint, with encapsulated synovial fluid, adjacent bones connected by ligaments, and surfaces that allow a small amount of motion. However, the SIJ is generally made for stability. There are multiple ligaments that hold the adjacent bones together to give the SIJ stability.

The SIJ is commonly innervated by L4, L5, S1, S2, and S3, although there are variants in many individuals. It is these variations that make diagnosis and treatment of SIJ pain so difficult.

Common Causes

Although most cases of sacroiliac joint dysfunction or sacroiliitis are due to osteoarthritis of the joint, some classic injuries leading to SIJ pain include falling on the buttocks, heavy and/or prolonged lifting and bending, motor vehicle accident, and repetititive motions sustained in sports such as figure skating or golfing.

Interestingly lumbar surgery such as lumbar fusion can itself create SIJ pain. It makes sense, as fixation of the lumbar spine causes more movement of the SIJ and pelvic girdle, thus stressing the SIJ further.

Pregnancy is another time in which SIJ pain increases. Physiologically, during pregnancy because of hormonal changes, the woman's pelvic girdle is made more mobile due to relaxation of ligaments and connective tissues in preparation for childbirth so that the baby can pass through the birth canal. Unfortunately, this relaxation of the pelvic girdle increases the like-hood of sacroiliitis and sacroiliac joint pain. There are specialized supportive belts, sometimes called "Trochanteric Belts" or "Sacroiliac Belts," that can be used to alleviate some of the pain.

Symptoms and Signs

Pain from the SIJ is often confused with lumbar herniated disc, spinal stenosis, facet disease, and even sciatica. Furthermore, many times, patients have these other concomitant conditions, further confusing the diagnosis. Classically, SIJ pain is localized to the buttock area, and may radiate down the thigh to the

knee, as well as the groin and hip regions. However, there are rare instances in which the pain radiates down to the leg and foot and is even associated with numbness and weakness, similar to sciatica or lumbar radiculopathy. It is common for the patient to complain of pain in the buttock and in fact, may prefer not to sit on the affected side.

Physical Examination

There are numerous physical exam maneuvers to diagnosis sacroiliac joint pain, but none are very specific or sensitive. There are too many tests to review in this short article. In a busy clinical setting, a "good test" for SIJ pain may be as simple as asking the patient to point to the pain with one finger. This is called the Fortin's Finger Test and the result is positive if the finger is within 1 cm of the posterior superior iliac spine (PSIS).

The clinician can also confirm by palpating the SIJ and asking the patient if that is painful. In my experience, most patients with sacroiliac joint pain will let you know instantly when you palpate the affected SIJ.

Another common and relatively simple test to perform is Patrick's Test or the "FABRE" sign, in which the patient affected SIJ is tested by having the hip Flexed, ABducted, externally Rotated, and Extended. This test is commonly performed by me in the pain clinic. Unfortunately, if the patient has hip pain or trochanteric pain, they may also have a positive response.

Differential Diagnoses

As mentioned earlier, hip and trochanteric related pain can mimic sacroiliac joint pain and vice versa. Other less common pathology to rule out include pelvic fractures, infection, spondylarthropathies (e.g. Rhematoid Arthritis or Ankylosing Spondylitis), tumors, and of course, lumbar herniated disc, lumbar spinal stenosis and facet DJD can all have the same pain distribution.

Definitive Diagnosis

Usually, imaging studies, including X-rays, CTs and MRIs are not helpful and a waste of medical resources and money. In fact, up to 25% of asymptomatic persons may have positive findings of SIJ degeneration on CT scan.

Many pain management specialists and anesthesiologists believe that the **best way to diagnose and treat SIJ pain** is with one or more diagnostic intra-articular injections directly into the sacroiliac joint under fluoroscopic guidance using contrast dye. Generally, most clinicians favor a combination of local anesthetic and a long acting steroid. The local anesthetic would afford immediate relief, which is the diagnostic component. The steroid effect will afford long term relief (e.g. months to even years), which is the therapeutic component.

At this point, most pain clinicians advocate injection of the SIJ under fluoroscopic or other imaging modalities (e.g. CT or ultrasound). Fluoroscopic guidance is most often used because it is relatively inexpensive and many interventional pain doctors are well trained in its usage. The joint is too deep to be properly injected using anatomical landmarks or palpation techniques. Most "blind" injection techniques will lead to ligamentous, muscular, and/or myofascial injections only.

Treatments

In terms of treatment, SIJ pain follows the same algorithm used for most other causes of mechanical low back pain.

Conservative treatment with a combination of NSAIDs and perhaps a muscle relaxant, as well as physical therapy, is the initial treatment plan. Physical therapy emphasizes strengthening and stabilization of the pelvic girdle as well as the lumbar spine and lower extremities. Postural and gait abnormalities are also diagnosed and corrected by the physical therapist, as they often cause or contribute to SIJ pain. Ice or heat may also be used.

When conservative treatments fail, the next step is diagnostic and therapeutic intra-articular injections as detailed above.

If local anesthestic/steroid injection combinations are effective but short-lasting, there have been cases of radiofrequency ablation of the nerves innervating the SIJ. Usually, radiofrequency ablation is a neurolytic technique involving lesioning of the nerves at a high temperature (e.g. 80 degrees Celsius). To prevent lesioning of motor nerves and to confirm lesioning of

the correct (painful) sensory nerves, both motor and sensory testing is performed before radiofrequency ablation occurs. Currently there is more than one company offering radiofrequency ablation equipment. However, because of inconsistent results and non-coverage by many insurance companies including Medicare, this modality has not been widely adopted by interventional pain physicians.

Until recently, surgical options have been limited. Traditional open surgery SIJ fusion involved a large incision and recovery was often 4-5 days post-operatively. The results were also not impressive. Traditional open surgery was reserved for intractable and disabling SIJ pain.

Currently, there is a new surgical fixation technology on the market, performed by local neurosurgeons and spine surgeons. It involves making a 3 cm incision and the surgical procedure lasts about 1 hour. Recovery is quick, as it can even be done in the same-day surgery centers. The technology, called "I-Fuse" and manufactured by SI-Bone, is relatively simple. It requires the surgeon to fuse the SIJ (ilium and sacrum) by placing 3 small rods via the 3 cm incision. According the company's literature, patients can weight bear on the affected SIJ at 3 weeks post-op. One criterion for this new surgical fusion technique is successful response to intra-articular injection first.

Conclusion

In summary, it is important to remember the SIJ when a patient presents with mechanical low back pain. The SIJ is a commonly overlooked cause of mechanical low back pain, although studies show that up to ¼ of low back pain may arise from the SIJ. Oftentimes, sacroiliitis or SIJ pain will not be the sole cause, but a contributing cause of low back pain, along with other more common causes (e.g. lumbar herniated disc). However, to give your patient optimal pain relief, it is imperative that you identify as many pain generators as possible. Fortunately, for the SIJ, the treatment options are better now than ever before.

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ACUTE MOUNTAIN SICKNESS

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Family Physician and Surgeon

Acute mountain sickness (AMS) represents a collage of signs and symptoms brought on by acute exposure to high altitudes. The spectrum of disorders ranges from mild nausea, headaches and insomnia to coma and death.[15] The first written description of AMS, composed sometime between 37 and 32 BC, is attributed to To Kan, a Chinese government official who noted that "a man's face turns pale, his head aches and he begins to vomit"[11], [14] when crossing the Himalayan Kilak Pass.

Broadly interpreted, high altitude is defined as anywhere above 5,280 foot (1609 m) in elevation. At such high elevations, almost everyone is affected to some degree by AMS. Effects vary from individual to individual and cover a variety of symptoms. The most classical of these are headache, insomnia, anorexia, nausea and dizziness.[14] AMS is perhaps best characterized as "feeling hung over." In more severe cases, symptoms may also include vomiting, dyspnea, muscle weakness, oliguria, irregular respiration, incoordination, lethargy, thirst, indigestion, excess flatulence, cough, chest discomfort, nightmares, peripheral edema and retinal hemorrhaging.[7],[18]

Other factors related to AMS are decreases in the oxygen content of air and decreased humidity at high elevations. In the Colorado Rockies at elevations between 8,000 and 10,000 ft, for example, there is approximately 45% less oxygen and approximately 50-80% less humidity compared to sea level. These account for the symptoms of a dry mouth, nausea, restlessness, and shortness of breath upon rapid ascent to high altitudes. The increased rate of respiration and the dryness of the ambient air increase insensible water loss and may cause acute dehydration.

Progressive dyspnea with tussive attacks, associated with dependent edema and other signs and symptoms consistent with fluid accumulation in the lungs requires prompt medical attention. High altitude pulmonary edema (HAPE) is estimated to occur in 0.5 to 15% of individuals who ascend too rapidly to high elevations. When the signs and symptoms of pulmonary edema are recognized early and treatment is promptly provided, reversal of the situation can occur, thereby reducing morbidity and mortality from high altitude sickeness.[11] Finally, HAPE can lead to symptoms of rales, cyanosis, ataxia and other complications such as high altitude cerebral edema (HACE), which affects approximately 1% of travelers to altitudes above 12,000 ft.[1]

Pathophysiology:

Hypoxia is the primary insult delivered by rapid ascent to a high altitude environment. The lowered barometric pressure of the ambient atmosphere rests in diminished alveolar oxygen tension.[13] As a consequence, arterial partial pressures of oxygen (PPO_2) drops from 100 mm/Hg at sea level to about 38 mm/Hg at 15,000 ft.[4] Although the exact pathway from hypoxemia to AMS, HACE and HAPE is unclear, a number of physiological processes appear to be involved, all related to a drop in PaO_2. Due to the rapid ventilator response to the drop in PaO_2, both respiration rate and dieresis increase, thereby increasing body fluid loss and leading to dehydration. Individuals whose respiratory or renal responses to

high altitudes are diminished are more likely to suffer AMS.[6]

The diagnosis of AMS may be hindered due to the clinical setting and complications of the hypoxic state and the pressures produced by peers due to their tight schedule for "having fun."[7]

Physicians should advise travelers going to high altitudes to suspect significant altitude illness in themselves if they have a headache and "hung over" feeling, dyspnea and a respiratory rate above 20/ minute at rest, anorexia, vomiting, ataxia or unusual fatigue while walking. They should suspect significant altitude illness in their companions if they note that a member of the party is skipping meals, exhibiting anti-social behavior, begins to stumble or is having the most difficulty with whatever the group's current activity is. A relatively simple field test of coordination is to ask the individual to walk on a narrow pathway, on a string or piece of wood placed close to or on the ground.

Other conditions implicated in the differential diagnosis of AMS should include dehydration, substance abuse, hypothermia, carbon monoxide poisoning (from using a stove in an enclosed tend), infection, exhaustion and an exacerbation of some pre-existing condition.[16]

Prevention:

The initial complaints of AMS should disappear as the body adjusts to the lowered oxygen content and dryness of the air. This may take several days to as much as a week. Upon arrival to high elevations, patients should be advised to avoid alcohol, excessive stress and increase fluid intake for the first day or two. Alcohol appears to intensify AMS through overexertion, dehydration, the sedative effects of alcohol and its interaction with other drugs in assn with depression of respirations.[7] Travelers over the age of 35 can prepare for their ascent by visiting the gym to prepare for their trip. Patients with pre-existing illnesses such as cardiac disease, circulatory lung disease or cerebral diseases should have a mandatory physical examination prior to departure.[17]

Patients should be urged to allow flexibility in their schedule of activities to allow members of the group to acclimatize. Drinking plenty of fluids is routinely recommended. In addition, a high carbohydrate diet increases the respiratory quotient and improves the body's use of oxygen at very high altitudes. The diet should also be low in fat and salt. These dietary changes should result in less altitude illness and better performance.

Slow ascent is most important on trips above 10,000 ft. One should ascend approximately only 1000 ft per day after reaching the 10,000 ft (3050 m) level.[1] Hypothermia is exacerbated at high altitudes so appropriate clothing is also necessary.

Acetazolamide, by inhibiting carbonic anhydrase in the kidney and lung, promotes the excretion of bicarbonate, producing a slight metabolic acidosis.[5] This effect counters the hypocapnic alkalosis produced by hyperventilation, thereby reducing symptoms of AMS by hastening the acclimatization process and decreasing susceptibility to AMS. Acetazolamide increases minute ventilation oxygen saturation and decreases periodic breathing at night.[9] It is also thought to reduce the incidence of HAPE and HACE.[19] Acetazolamide is especially important for those altitudes approaching 10,000 ft, but is also beneficial on slower ascents and especially for those who tend to get altitude illness.

Acetazolamide should be carried by everyone going to high altitudes to treat possible symptoms and thereby accelerate the acclimatization process. The maximum dose is 5 mg/kg per day in two or three doses. About 125 mg a day, twice a day, three times a day or once at bedtime may be adequate.[4]

Dexamethasone is also effective as a prophylactic for AMS, but it is not routinely recommended because of significant dysphoria and the likelihood of rebound altitude illness when it is discontinued.[12] Other modes of action of Dexamethasone may be its ability to decrease fluid leak from the microvasculature. Most specifically, Dexamethasone is used to treat cerebral symptoms, but it is routinely recommended for prophylaxis. Doses of 4 mg every 6 hours have been purported as acceptable prophylaxis for AMS.

Finally, patient advice should include appropriate rest. Patients should be advised to take a nap when sleepy and to get a good night's sleep after an exertive day at high altitudes. Patients should again be reminded to eat lightly, drink plenty of fluids and avoid alcohol for the first 48 hours.

Treatment:

The acute treatment of AMS consists of reducing the activity level of the patient to more modest levels, mild analgesics, acetazolamide (125 to 250 mg bid, beginning with a dose at bedtime) and Prochlorperazine, 10-25 mg dose, for nausea and vomiting. Severe AMS calls for immediate descent, oxygen and acetazolamide. Dexamethasone, 4 mg PO every 6 hours, should be given for severe AMS when descent is underway.[18]

AMS AND CHRONIC DISEASES: For patients with chronic diseases going to high altitudes, special care should be given. They should plan an itinerary that allows for easy access and easy ascent and descent (if not for the patient, then at least for potential rescue teams). Prudence and planning a secondary itinerary in case the first becomes too demanding will probably be the most beneficial advice. In general, one should preferably stay under the 8,000 ft level and know where local medical services are obtainable on rapid notice.

Deaths at high altitudes are more common with individuals having known cardiac disease or a significantly altered hemodynamic status. One should consider increasing the dosage of anti-anginal medications and recommend rest for a minimum of 72 hours upon arrival to the high altitude. Many asthmatics report improvement in their condition at high altitudes, perhaps due to less dust, lower air density and fewer inhaled allergens.[3] Such individuals should use inhaled bronchodilators before and after exercise and/or exposure. Diabetics may experience problems regulating their insulin dosage due to the varying energy expenditure and food intakes. More frequent insulin dosing and blood glucose determinations would be advised for those undertaking these activities.[17] The presence of sickle cell disease or other hemoglobinopathies is considered a contraindication to ascent to high altitudes. There is a 60% increased risk of crisis with sickle cell disease.[2]

In conclusion, the successful treatment of acute mountain sickness lies in early recognition of the signs and symptoms of it and of related disorders. Supplemental O_2 is indicated as temporary relief; however, descent to lower altitudes is curative. Patients should be aware of the various health-associated problems that may be encountered at higher elevations. Self-diagnosis and treatment is the usual course at such remote locations and patients should be strongly urged to plan realistic itineraries and avoid peer pressure that may lead them to undertaking activities that will exceed the individual capacities.

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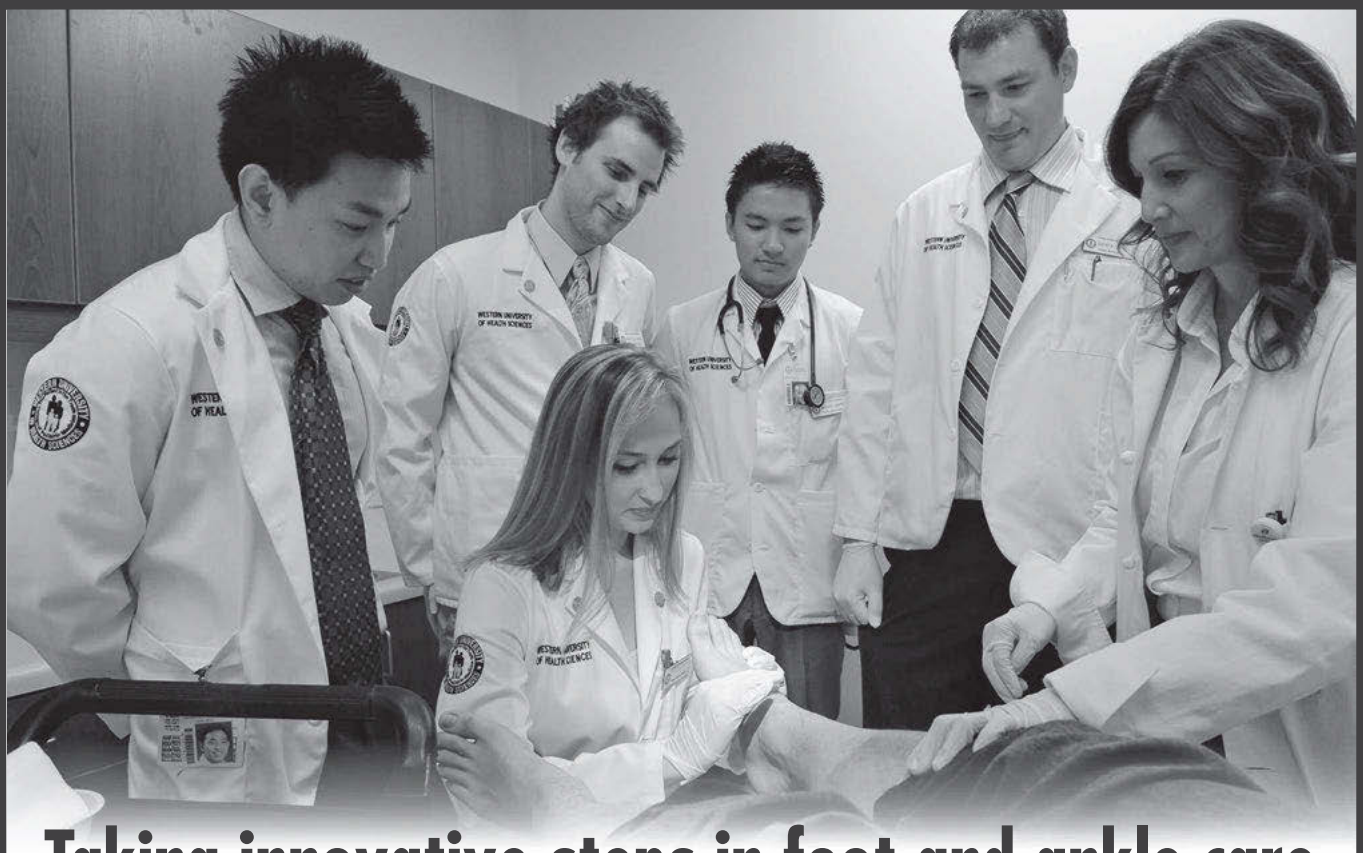

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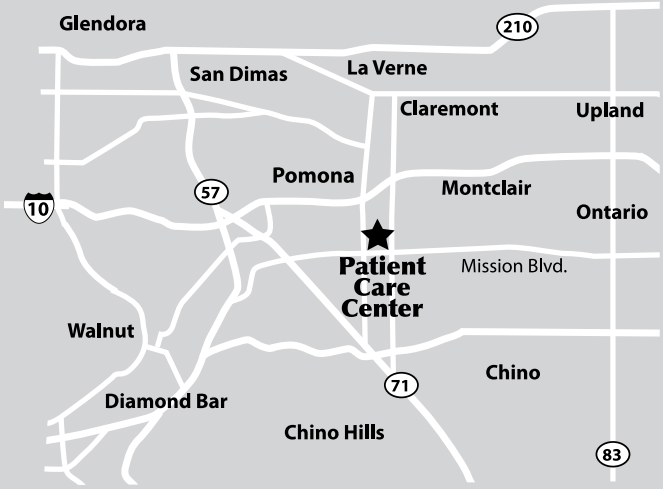

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