2008

UWOMJ Volume 78, Supplement S1, Summer 2008

Western University

Editorial: A New Perspective

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A stack of Canadian Medical Association Journals (CMAJ) sits on my kitchen table. I have big plans to read and understand each article, to expand my knowledge, to feel like a real doctor. But more often than not those CMAJs simply serve as a place mat for the novel or newspaper that I lay over top. It is always with a twinge of guilt that I delay reading these journals. It feels like they are something I ***should*** read, that I have a ***responsibility*** to read. This guilt is made all the more poignant by the question that always seems to accompany my decision to leave the CMAJ closed- why don't I ***want*** to read it? Didn't I spend the better part of the last decade doing everything I could to get into medical school? Didn't I experience an almost embarrassing level of excitement when that first issue was delivered to my mailbox?

That question has nettled me all year as the stack grew ever taller. But this summer my clinical elective preceptor helped me to answer that question. She said that the CMAJ isn't necessarily relevant to me at this point in my training. We simply haven't learned enough to either completely understand every article nor to find it always interesting. Much like a book on skydiving would be mildly intriguing to an earth-bound reader, it doesn't become riveting until you've taken that first leap. This summer, many of our classmates leapt for the first time. We left behind our textbooks and lectures and faced the real world of medicine.

The Summer Supplement is a record of those experiences in the real world. This issue of the UWOMJ is considerably different from the regular print issues. The purpose of the articles in this issue is not just to inform, but to inspire and celebrate. These are the true experiences of our classmates; they remind us of our own summer experiences, encourage us to move beyond the familiar, and commemorate the successes of our peers. By capturing these experiences in an article for the Summer Supplement, we are able to leave a record of all the things we accomplished this summer, be it research, clinical electives, or travel.

In addition to different goals, the perspective of this issue is remarkably different. Normally the authors' gaze is in to the literature; the Summer Supplement has challenged us to raise our eyes up from our textbooks, beyond the walls of our classrooms and to see how medicine exists in the real world. This perspective is simultaneously exciting, overwhelming, and inspiring. Suddenly we saw that diseases that were described in our notes happen to real people, and it is now up to us to dredge up all the facts that we painstakingly learned throughout the year and apply them to actual practice.

A written discourse tells the world how you want to be remembered. It is a record of your thoughts and perceptions, your level of knowledge, your interests. That is what makes this issue of the UWOMJ different from the regular print issues and from publications like the CMAJ. This compilation of articles is all about perspective. Authors were encouraged to write an article that would appeal to the medical student, both in terms of knowledge level and interest. While the goal was also to describe common presentations, diagnostic tools, and treatments, the primary aim of this issue was to create a record of medicine through the eyes of the Classes of 2010 and 2011.

It is immediately apparent that each article in this collection has a slightly different structure. The reason is that the authors were encouraged to be creative. Since perspective plays such a pivotal role in this collection, each author was encouraged to choose a style that worked for their topic, their case, their style of writing.

Given this emphasis on perspective, I hope this collection of articles will not suffer the same fate as my CMAJs. The subsequent articles are each relevant to this point of our education, simply by virtue of the fact that they were written by members of the intended audience. The diverse compilation that follows will hopefully help you to broaden your perspective, to consider medicine as an entity outside of the classroom, and to inspire you to approach your studies in such a way that will prepare you for your own leap into the real world of medicine.

Right Total Shoulder Replacement with Reverse Delta Prosthesis, Complicated by a Brachial Plexus Injury

Amin Madani, Hon. BSc. (Meds 2011), Dr. Richard M. Holtby, MD, MB, BS, FRCSC

Abstract/Introduction

When conventional total shoulder replacements are not considered to be an effective surgical management to improve patients' quality of life, a reverse total shoulder arthroplasty is considered. The reverse total shoulder replacement is where a prosthesis with a convex articular surface is fixed to the lateral aspect of the glenoid, and a prosthesis with a concave articular surface is fixed to the proximal aspect of the humerus (Figure 1).[1] This procedure was initially indicated for patients with rotator cuff arthropathy, for whom a traditional total shoulder replacement has a relatively high rate of failure.[1,2]

Today, they are also indicated for patients with failed arthroplasty, or for selected patients with shoulder fractures whose prosthesis has failed.[3] Despite its high success rate, this procedure is also prone to complications.[1,2,3,4] It is estimated that 1-4% of shoulder arthroplasty cases produce neurologic complications.[1,2] In this type of patient who has a history of rotator cuff arthropathy and a full-thickness rotator cuff tear that was repaired and subsequently degenerated, it is shown that a total shoulder arthroplasty with a reverse prosthesis can be used to provide improved function and quality of life. It is furthermore shown that patient recovery and functional outcome can be complicated by radial nerve palsy.

Figure 1. Delta prosthesis used for reverse total shoulder arthroplasty.
http://www.jnjgateway.com/home.jhtml

Case

Mrs. W is a 74-year-old woman seen in the clinic for assessment of her right shoulder. She has a thirteen-year history of pain, where she was diagnosed with a rotator cuff tear and had surgical repair nine years ago. Although she felt this helped significantly, there was gradual increase in pain and weakness of the shoulder. Examination showed that she was able to get her arm to 150^{o} of flexion with some catching and pain. External rotation was 45^{o} with internal rotation to the thoracolumbar junction. She had pain, crepitus coming from the subacromial space, and significant weakness of forward flexion. X-rays were also done, which showed degenerative changes at the glenohumeral joint with marked superior migration of the humeral head with respect to the glenoid fossa, consistent with rotator cuff arthropathy.

Rotator cuff arthropathy is characterized by irreparable loss of the rotator cuff, severe osteoarthritis of the glenohumeral joint, pain, and minimal function of the shoulder.[1,2,3] It was felt that a reverse prosthesis would not only effectively help her pain and provide more function, but unlike the traditional shoulder replacements, would resist superior translation of the humeral head due to a lack of stability of the joint from degeneration of the supraspinatus rotator cuff muscle.

Surgical Technique

The shoulders were examined with the patient under general anaesthesia, where the range of motion of the right side was noted to be sub-optimal (Table 1).

Table 1. Pre-Operative Range of Motion of the Right Shoulder

Forward Flexion	150^o
Abduction	90^o
External Rotation (with 0^o abduction)	40^o
External Rotation (with 90^o abduction)	40^o
Internal Rotation (with 90^o abduction)	20^o

With the patient in the beach position, draping was done to expose the shoulder with the arm free, allowing the surgeon and his assistants to manoeuvre the arm in different positions. The incision was a saber-cut incision, which is made 4 cm from the superior border of the anterior and middle deltoid fibers, passing just above the acromioclavicular joint, extending 5 cm inferiorly on the posterior side and creating a reverse U-shape.[1,2] A deep deltoid incision was subsequently done to get access to the joint. Operative findings included a massive rotator cuff tear of the supraspinatus estimated at 5 cm medial to lateral and 5 cm anterior to posterior, bone erosion and eburnation of bone on the articular surface of the humeral head and glenoid consistent with osteoarthritis, degeneration of subscapularis and teres minor muscles, and rupture of the long head of the biceps tendon.

Initial neck cut was made removing the head component at the surgical neck of the humerus, providing exposure of the glenoid with a fork retractor. The capsule was then released around the articular margins, and the labrum and biceps tendon remnant removed. Once the centre of the glenoid was demarcated, a drill hole was made into the glenoid medullary cavity. This allowed the insertion of the glenosphere reamer and metaglene (glenoid components of the prosthesis), which were hammered into place, and fixated with four screws.

After drilling a canal for the humeral component's stem, trial humeral components were inserted to determine the correct size of the prosthesis. After appropriate trial reductions, the canal was prepared for irrigation, filled with cement, and the humeral stem cemented into place. Finally, the definitive glenosphere and humeral polyethylene components were inserted, and the joint reduced, which showed stability with good range of motion. After a routine closure, the patient returned to the recovery room with a simple blue sling and in satisfactory condition. Ancef was given as prophylactic antibiotic with two doses given intra-operatively.

Post-Operative Results and Discussion

Specimen sent with fragments of bone and soft tissues from the joint, as well as post-operative x-rays revealed no apparent complications (Figure 2).
However, the day following surgery she was noted to have a right finger and wrist drop with numbness in the posterior arm, despite regaining significant range of shoulder movement. Examination revealed absent right brachioradialis and triceps reflex with normal biceps reflex. There was marked weakness of right shoulder external rotation and abduction, almost no triceps function, no visible brachioradialis function and only slight wrist extension with total finger drop. Sensation to pin was also reduced in the posterior hand, forearm and upper arm.

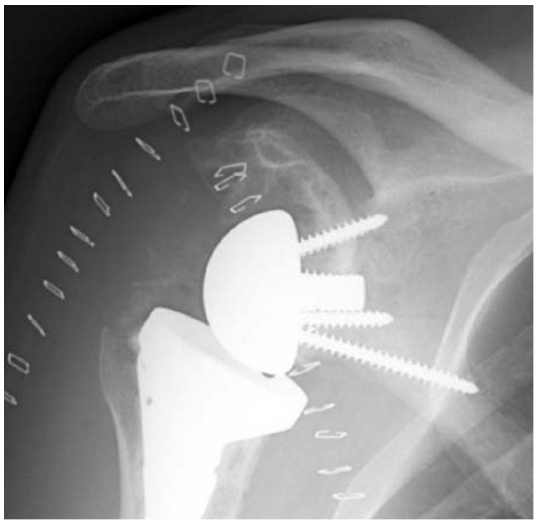

Figure 2. Example of a post-operative anteroposterior radiograph from a reverse total shoulder arthroplasty.[1]

Electromyography workup showed normal right median and ulnar nerve conduction. The radial sensory response was absent and lateral cutaneous nerve of forearm normal. Needle electrode examination revealed marked and moderate denervations of the infraspinatus and deltoid muscles, respectively, and severe denervation throughout the radial nerve distribution. Although musculocutaneous, median and ulnar nerve function were normal, there was partial denervation in suprascapular, axillary and mostly radial nerve distribution. As there were some surviving neurons, the nerves appeared to be in continuity but with axonal deterioration. It is likely that the lesion was at the border of the teres muscle or more proximally in the superior trunk. With severe axonal loss in the radial nerve distribution, recovery proceeded slowly over a number of months with appropriate physiotherapy, and the patient eventually regained brachial plexus function with total reinnervation.

Acknowledgements

Amin Madani would like to thank Dr. Richard M. Holtby, Orthopaedic Surgeon at Sunnybrook Health Sciences Centre, Orthopaedic & Arthritic Centre, and Assistant Professor of Surgery at the University of Toronto, for his assistance throughout the case study.

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Inspiration
Location: Sunnybrook Health Sciences Centre, Orthopaedic & Arthritic Centre in Toronto, ON.
Program: Non-credit elective.

The Consequences of Poorly Controlled Diabetes in a Young First Nations Man

Dax Biondi, BSc, MSc (Meds 2011) and Dr. Michael Kirlew, Northern Medical Practice, Meno Ya Win Health Centre, Sioux Lookout

The consequences of poorly controlled diabetes, including blindness and renal failure, are exemplified in a young man from an isolated Northwestern Ontario community. The nurse in charge reported during the summer of 2008 that 25% of community members have diabetes. There are many social and medical reasons for a patient's failure to control his or her blood glucose. The claim that financial barriers prevent First Nations people from eating a diet that is high in fruits and vegetables, complex carbohydrates and protein and low in fat and sugar is addressed in the present study. It is well known that morbidity can result from poorly controlled diabetes; Derek's story sadly illustrates its devastating toll.

Derek is 27 this year but as Dr. Michael Kirlew mentioned, chronic poor diabetes control has aged his blood vessels to the equivalent of 47 years. Dr. Kirlew is a family physician, one of several who has provided care for Derek since he was diagnosed with type II diabetes mellitus at the age of 13. His sister, who also has diabetes as do Derek's mother and all his maternal aunts, has taken care of him for the last 3 years. Derek worked in renovations in his community until diabetic complications rendered him incapable. Clinic notes over the past 3 years detail the toll that high blood glucose has taken on Derek's body. Despite consistent medical advice and support, Derek has failed to accept the invitation to become involved in the management of his disease. At such a young age, his condition is now nearly palliative.

The backbone of type II diabetes management is well-controlled blood glucose; maintaining hemoglobin glycosylation below 7% is protective against microvascular complications[1]. It is not surprising that it was difficult to convince a rebellious 17 year old of the potential consequences of his disease. As a teenager, Derek felt fine and in the absence of consistent family support, Derek did not attend most of his diabetes education classes. Free of charge, he was given the opportunity to attend education camps and counseling but he did not take advantage. Five years later, at the age of 22, Derek's doctor dictated a letter to Derek's boarding school principal. His doctor urged the principal to assist Derek with his blood glucose control as its high levels where likely impairing his ability to study. The proportion of HbA1c that is glycosylated reflects what a patient's average blood glucose has been over the proceeding 3 months[2]. **Figure 1** illustrates that the glycosylation of Derek's HbA1c between 2006 and 2008 was consistently elevated.

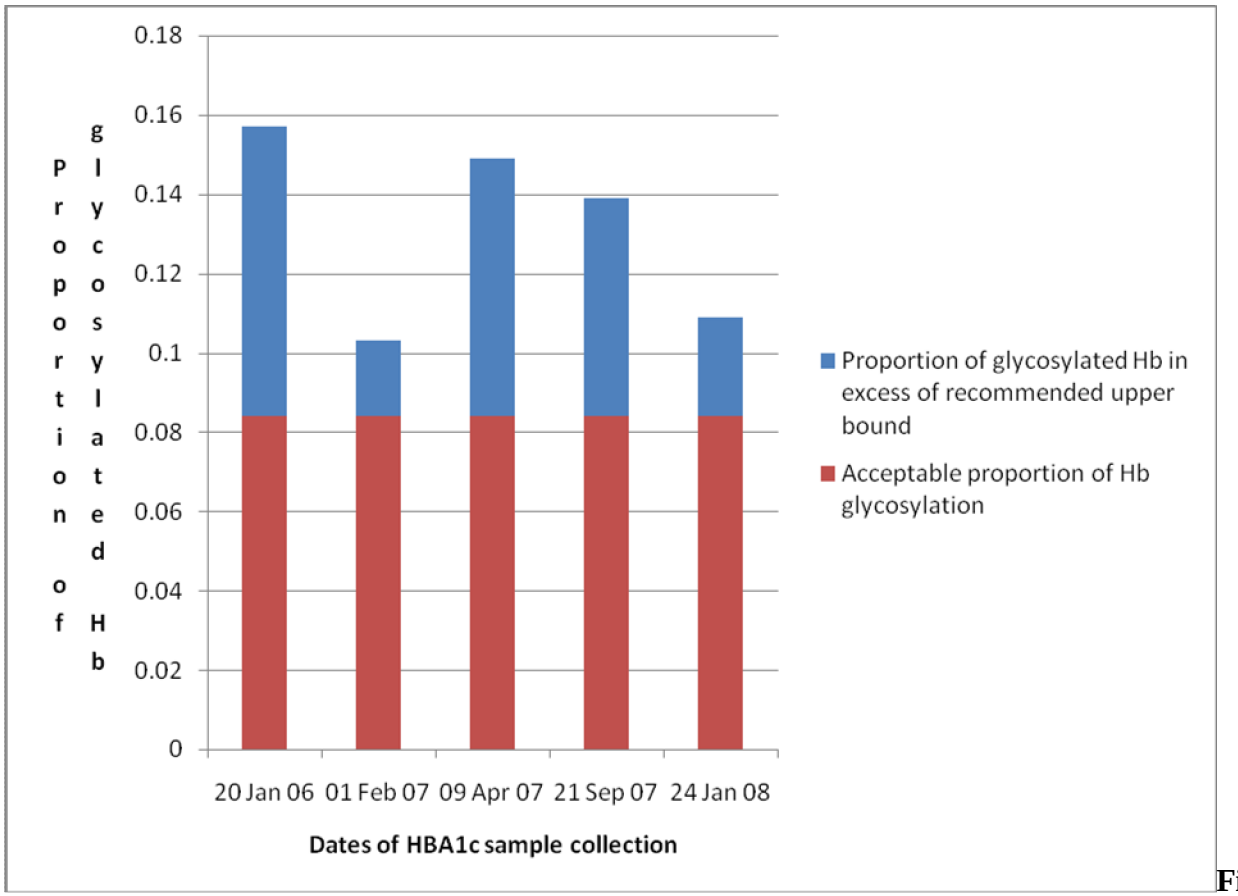

Figure 1. Derek's HbA1c measurements taken over 2 years demonstrate that his blood sugar was consistently toxically elevated.

Pharmacotherapy of Derek's disease floundered. At 23 years, he was supposed to be taking Glyburide, Metformin and insulin to control his blood sugar. However, Derek was forgetting to take his Glyburide and he chose to stop taking his Metformin when an increase in dose began to cause him gastrointestinal upset. Shortly after, he developed diabetic foot ulcers due to neuropathy that rendered his feet less sensitive. Yet, his feet were found to have good pulses, colour and sensation and fortunately, his ulcers were not infected. It was assumed that his ulcers were due to wearing work boots.

At 24 years, Derek began to appreciate the gravity of his disease and was willing to take insulin. However, 11 years of poorly controlled diabetes had already damaged his kidneys and urinalysis indicated that there was protein in his urine. He reported at that time that he did not drink any alcohol nor take any illegal drugs.

Around 25 years, Derek discontinued taking his insulin and Glyburide and made it known he did not care to check his blood sugar at home. This coincided with the onset of infected foot ulcers for which treatment in Sioux Lookout, a community 400km to the south, had to be sought. Diabetes is a

vascular disease and necessitates cardiovascular protective pharmacotherapy. Despite blood pressures as high as 165/102, we cannot be certain this 25 year old was taking his Ramipril regularly.

Almost 13 years of nearly subclinical, consistently high blood glucose levels began to show evidence of destruction during Derek's 26th year. At an appointment in which his morning blood glucose was found to be 15 mmol/L and post lunch, 16-18, he stated that he had blurred vision and bilateral decrease in sensation below the knees. Despite these symptoms, he failed to attend an appointment with an ophthalmologist in the city of Winnipeg because he did not qualify for an escort. Given that escorts are an important part of care for patients in isolated communities, the government pays for the cost of transportation for a friend or family member to travel to one of the regional centres to enable the patient to access required care. Derek was frustrated that he did not qualify for such a companion and for that reason, he missed his appointment.

About a month after missing the ophthalmology appointment, Derek presented with a fixed dilated pupil due to a dense vitreous hemorrhage of his right eye; this caused neovascular glaucoma, which rendered that eye blind. Shortly thereafter, he developed bilateral diabetic peripheral neuropathy and diffuse eczema on his lower legs, presumably from venous stasis. In the fall of that year, he began to experience tunnel vision in his left eye and further tests indicated proteinuria.

I had the opportunity to meet Derek in his home clinic this past summer. At 27 years of age, he presented with leg pain that was not responding to Tylenol Number 3. He had a large ulcer on the dorsum of his right foot, for which his doctor was concerned about underlying osteomyelitis. He had a deep vein saddle thrombosis, which arched from his right femoral vein to his left and he was in kidney failure. He was blind and he could no longer walk.

To facilitate dialysis, Derek will move to Thunder Bay in the near future. He is aware of the irreversible toll that high blood glucose has taken on his body and he is trying to prepare himself for the consequences.

Many social and medical factors have contributed to Derek's rapid and debilitating course with diabetes. Genetic predisposition has unarguably contributed to Derek's condition as have many social predisposing factors. It is argued that the high food costs in northern communities may be one such social determinant of health. How does the cost of food in Derek's isolated community compare to that in the nearest regional centre?

The financial burden of eating well in Derek's community was documented by the present author during August of 2008 at one of the two local grocers, the *Northern* store. It was found that it costs a person about 2 times as much to stock his or her fridge and pantry as someone living in Sioux Lookout. **Figure 2** shows that by food group, most everything is double the price, be it processed foods like fries and pizza

pops or fresh vegetables, whole grain breads and milk

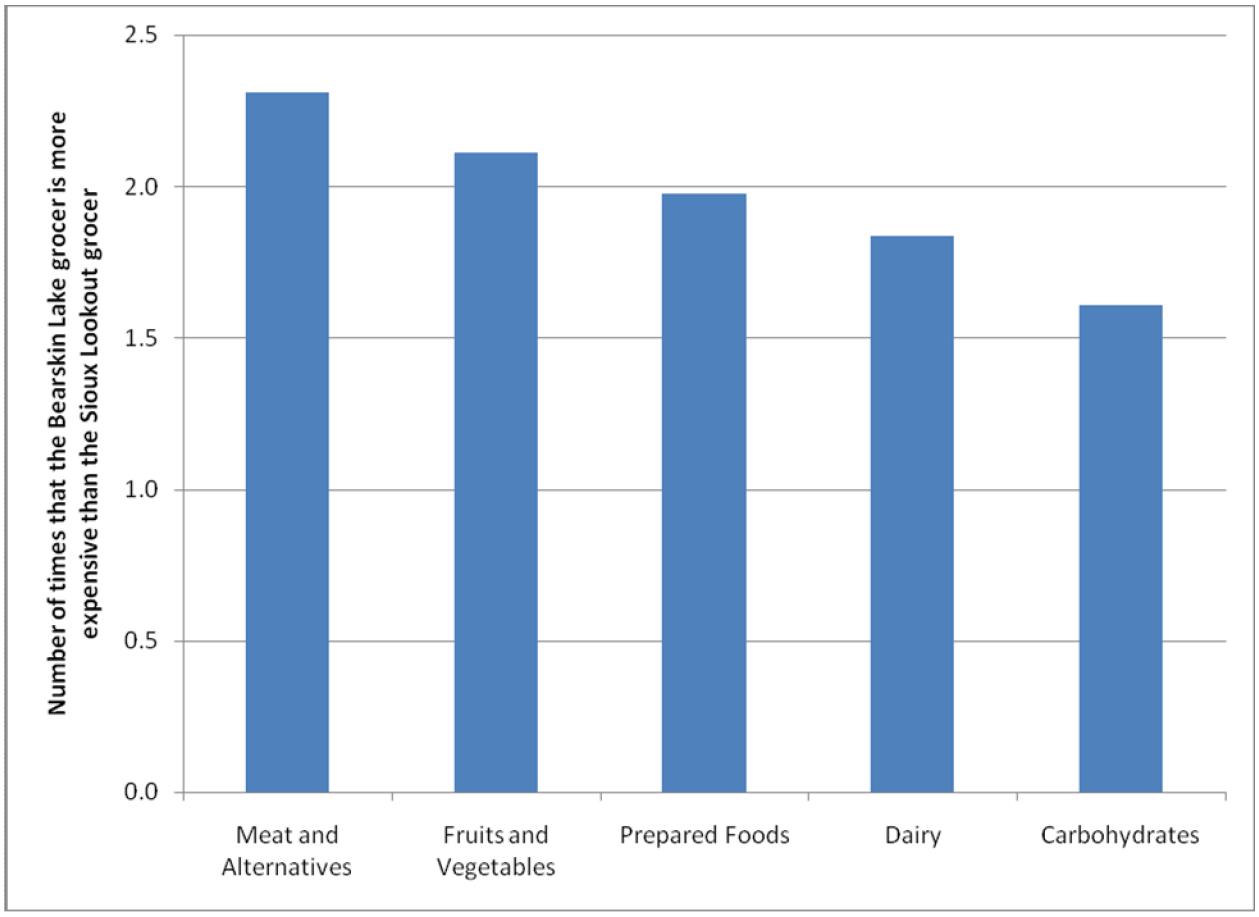

Figure 2. The number of times groceries are more expensive in Derek's community in comparison to Sioux Lookout by food group.

This food price comparison is a look at August food availability only and in other months, supply may be less due to less food being available in the off seasons or more due to it being less expensive to bring in food by ice road during the winter. It was found that many fresh and healthy foods were available for sale in town; physical availability does not seem to be the cause of poor diet. If expense were the main barrier to eating a healthy and balanced diet then price subsidization would be the answer to preventing diabetes morbidity. It is likely naïve, however, to deem that this would resolve the problem. Skeptical of this solution, I ponder the following questions, "Is it not reasonable to assume that the lower cost of living enjoyed by First Nations people on reserves should off-set the high cost of food? Are people making poor choices in terms of how to prioritize their financial resources rendering their diets inadequate while fulfilling other desires?"

The reality of the price disparity between the north and south cannot be

dismissed but I suspect it does not entirely explain the high burden of diabetes and its comorbidities. Interventions which have set out to educate First Nations people on how and why one must eat a balanced diet and get adequate exercise have shown promise at reducing the burden of obesity and diabetes[3]. Perhaps these ingredients for health are met with apathy stemming from complex social issues including normalization of obesity[4], boredom, poverty, rebellion, and as Rock, M. writes about, distress and societal suffering?[5]

Investigation into the following questions may shed further light on what impairs some persons with diabetes from maintaining low blood glucose: "What foods do you usually buy at the grocery store? What percentage of your money do you spend on groceries each week? How many dependents do you care for and how old are they? What is the body mass index (BMI) of your dependents? Do you think that healthy foods at the store are too expensive? Do you know the elements of a balanced diet?" Ho et al. asked similar questions and concluded that "Increasing knowledge and outcome expectations about healthier food preparation and selection could help reduce fat and sugar intake as well as increase fiber intake."[6]

The present discussion leaves many questions in terms of the social determinants, which led to Derek's condition. However, the case does give striking support to the research on the debilitating consequences of poorly controlled diabetes[7], particularly in genetically susceptible individuals[8]. Whether or not the cost of groceries in Derek's community prohibited him from following dietetic counsel is not known. However, it is reasonable to conclude that First Nations people in isolated communities in Northwestern Ontario must allocate more money for groceries than those in the south.

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Inspiration The young man whose identity has been protected by the name Derek.

Location: Meno Ya Win Health Centre, Sioux Look and Derek's community nursing station (the name of which is being withheld to protect Derek's identity).

Program: Medicine 5010 Non Credit Summer Elective

Balloon brachytherapy in the treatment of invasive ductal carcinoma of the breast in a rural setting

Kevin Mitchell, Hon. BSc (Meds 2011) and Valerie Jefford, MD

Case

A pleasant 61-year-old woman presented to her family doctor after her good friend was diagnosed with breast cancer. She suffers from hypertension and Raynaud's, but denies any cardiovascular disease or diabetes. At the time of her surgical consultation, she was taking Diovan 80/12.5mg, Adalat 30mg XL, and ASA 80mg OD. The patient had an aunt who was diagnosed with breast cancer in her sixties, with no other significant family history. The patient was morbidly obese, but otherwise had an unremarkable physical exam with the exception of a small palpable lump in the lower outer quadrant of the right breast, which was firm and mobile. No lymphadenopathy was detected. She underwent her first mammogram, serving as her baseline mammogram, and a small abnormality was detected in the inferolateral aspect of the right breast (Figure 1). A follow-up ultrasound and biopsy were performed (Figure 2). Histological examination of the biopsied tissue indicated that the patient had invasive ductal carcinoma of the right breast.

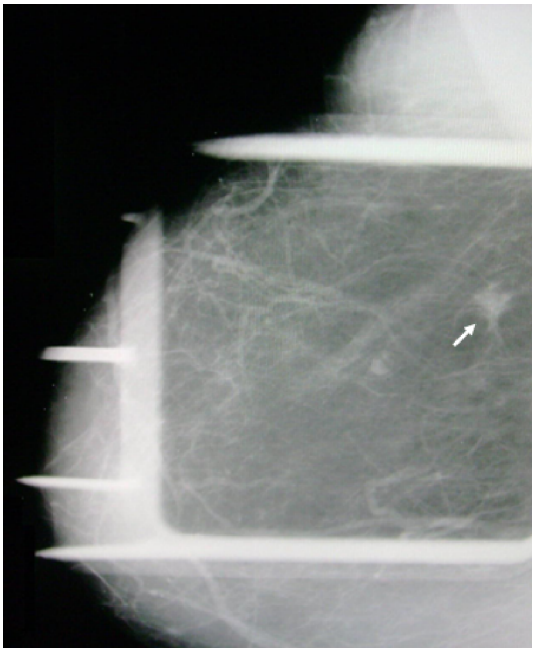

Figure 1. Mammogram of the right breast. White arrow indicates tumour location.

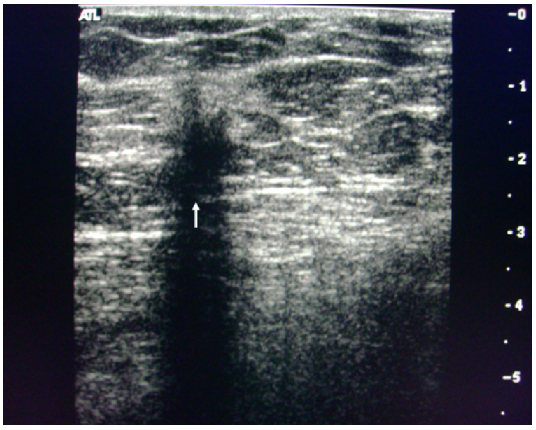

Figure 2. Ultrasound of the right breast. White arrow indicates lesion.

The patient was counselled regarding her surgical options. She was offered a right partial mastectomy followed by radiation treatment, or a total mastectomy. She was also informed about the sentinel lymph node biopsy procedure, as well as the axillary lymph node biopsy that may be necessary if the sentinel lymph node biopsy was positive for metastasis. The patient was also educated on the sequence of events following surgery, including some routine investigations and a referral to medical radiation oncology. The patient decided to proceed with a partial mastectomy, understanding that radiation treatment must follow surgery.

The surgery was performed and the tumour was determined to be 1.5 centimetres in diameter with a Grade I Nottingham score. Sentinel lymph node biopsy showed no metastasis. The distance of the closest margin of the tumour was less than 1mm from normal tissue, so the anterior margin was re-excised a few days later as a cautionary measure. The final pathology report showed fibrofatty tissue with a few small ducts and one larger duct with hyperplastic epithelium. Also present were small clusters and tubules of bland atypical cells. Haematoxylin and Eosin stains were performed, as well as Immunoperoxidase stains. The results were compatible with low grade invasive carcinoma of mammary ductal origin.

As an alternative to whole breast radiation, the patient was informed of balloon brachytherapy using a MammoSite balloon catheter. The patient opted to undergo this procedure, which entailed implanting a silicone balloon connected to a catheter with an inflation channel and a port for the insertion of a high-dose brachytherapy source. Radiation treatment for this type of procedure generally consists of an ^{192}Ir source connected to a computer-controlled high-dose-rate remote afterloader being inserted into the balloon to deliver the prescribed dose of radiation.[1] The balloon catheter was implanted into the patient and was

inflated to its maximum capacity of 70mL with normal saline (Figure 3). The surgery was performed at Western Memorial Regional Hospital in Corner Brook, Newfoundland, after which the patient was transferred to the Health Sciences Centre in St. John's, Newfoundland to receive a 5-day course of radiation.

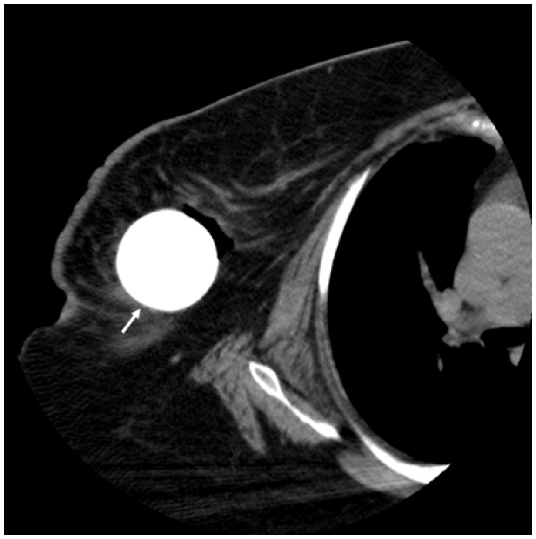

Figure 3. Post-operative CT scan of right breast. White arrow indicates brachytherapy balloon filled with normal saline.

Discussion

Breast conservation therapy (BCT) is an attractive treatment option for patients with Stage I and II breast cancer.[2] BCT for early-stage breast cancer involves the surgical removal of the tumour followed by whole-breast radiation.[3] Long-term studies have indicated that disease-free survival for breast conservation therapy is similar when compared to mastectomy.[4,5] Based on the cosmetic advantages and the similar survival rates, balloon brachytherapy is an ideal treatment option for patients wishing to avoid a total mastectomy.

Patient selection criteria for balloon brachytherapy as determined by the American Society of Breast Surgeons is patient age of 50 years or over, invasive ductal carcinoma or ductal carcinoma in situ, 2 cm tumour size, negative microscopic margins with surgical margins of excision of at least 2 mm, and N0 nodal status.[1]

In comparison to multi-catheter brachytherapy, balloon brachytherapy has several advantages. Balloon brachytherapy has been shown to be superior to interstitial high-dose radiation brachytherapy in planning target volume coverage.[6] While both therapies deliver high-dose radiation to the patient in one week or less, interstitial implants using multiple catheters require additional expertise and training. Furthermore, numerous puncture sites are required for multi-catheter brachytherapy, which can lead to suboptimal cosmetic outcome and infection.[1]

Balloon brachytherapy confines radiation treatment to a limited volume of breast tissue adjacent to the lumpectomy cavity, which allows for a higher dose of radiation per fraction, decreasing treatment time.[6] This makes balloon brachytherapy an ideal treatment option for patients from smaller

communities. Conventional radiation therapy following lumpectomy generally takes 5 weeks to complete. For a patient from a small town, this requires undergoing daily radiation treatments in a larger centre, potentially far away from the patient's place of residence. The patient treated in this case resided over 600 km from the closest breast cancer radiation treatment centre. Balloon brachytherapy enabled the patient to undergo a 5-day course of radiation treatment as an alternative to 5 weeks of radiation. In the context of the patient's illness experience, instead of having to relocate to St. John's, Newfoundland for 5 weeks, the patient was able to stay in St. John's for 5 days to complete her radiation treatment and then return home. Ultimately, this treatment option allows for less time away from home and a higher quality of life during treatment.

Conclusion

BCT with balloon brachytherapy is an excellent treatment option for patients fitting the selection criteria. This therapy has very good cosmetic results along with a reduced post-operative radiation treatment time while maintaining the efficacy of a total mastectomy. This allows for patients to receive treatment while limiting the interference in their everyday lives that comes with 5-week whole-breast radiation courses, resulting in an increased quality of life. Where patients from rural communities once may have chosen a total mastectomy instead of undergoing BCT due to the five weeks of radiation, balloon brachytherapy and the associated reduced treatment time enables these patients to opt for BCT in lieu of a total mastectomy.

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Inspiration

Location: Western Memorial Regional Hospital in Corner Brook, Newfoundland.
Program: Non-credit elective.

Peripheral Vascular Disease: A Tale of Ulcers and Cans of Tomatoes

Laura Hinz BSc (Meds 2011), Dr. Laura Heemskerk, MD and Dr. Ernie Janzen, MD

Case

This is a case of peripheral vascular disease, but it is not a story about a toe. Rather, it is a story about a can of tomatoes. A can that is perched precariously on the counter in the home of a 92-year old woman, Mrs. S. Six months ago, that can fell onto the first toe of her left foot. Five months ago, Mrs. S began to experience increasing discomfort in that toe and began a home treatment regimen of Polysporin (bacitracin and polymyxin) and Bactroban.

Mrs. S is accustomed to managing her own health concerns, as she is currently on medication for hypothyroidism (Synthroid), hypertension (Adalat and Inhibace) and atrial fibrillation (Coumadin). She has peripheral vascular disease that was treated with bilateral surgical revascularization twenty years ago yet she has absent popliteal and pedal pulses. Her medical history was notable for migraines, angina, varicosities, macular degeneration, and profound hearing loss in the left ear.

When it became clear that her home treatments were making slow progress, Mrs. S presented to her internist's office. The can of tomatoes had done considerable damage- the sole of the left great hallux was marked by a 1.5 cm ulceration that extended to the bone. There was no necrosis or obvious signs of infection except a slight yellow drainage. Mrs. S needed to wear her shoes at all times in order to maintain her balance- meaning the wound was under constant pressure.

The internist informed Mrs. S that in order to prevent amputation of the toe and possibly the foot, she would need to be admitted to hospital, a treatment plan that was less than appealing to Mrs. S. She had a home and a dog to care for and was reluctant to relinquish her independence, yet she eventually consented to enter the hospital. The wound was diagnosed as an ischaemic ulcer secondary to peripheral vascular disease and a plastic surgery consult was ordered. The x-ray revealed no fractures or dislocations but significant vascular calcification between the first and second metatarsals. She was treated with a silver sulfadiazine dressing (Flamazine), Amoxicillin and Clavulin orally. Mrs. S spent 27 days in hospital and was discharged with forefoot offloading shoes.

Peripheral Vascular Disease: Manifestations

Peripheral vascular disease is an occlusive disorder caused by atherosclerosis. More than 50% of patients are asymptomatic whereas others present with claudication, ischaemic ulcers, or gangrene when blood supply is unable to meet metabolic demand (Figure 1).[1,2] PVD is more common in the

elderly, with a 5.6% prevalence in the 38-59 age range versus 26% in those 81 and older.[1] A study in *Wound Repair and Regeneration* found that 26.2% of patients admitted from nursing homes had pressure ulcers compared to only 4.8% from other living situations.[3] Ulcers are especially prevalent among patients with diabetes mellitus, with up to 25% of diabetics developing an ulcer.[4] Risk factors for PVD include smoking, hypothyroidism, diabetes, hypertension, dyslipidemia, elevated plasma homocysteine.[1] Besides endangering the viability of peripheral tissue, PVD can also increase the risk of adverse cardiovascular events such as MI or stroke.[1]

PVD most often manifests as leg ulcers, which can be classified as arterial, venous, or neuropathic:[5]

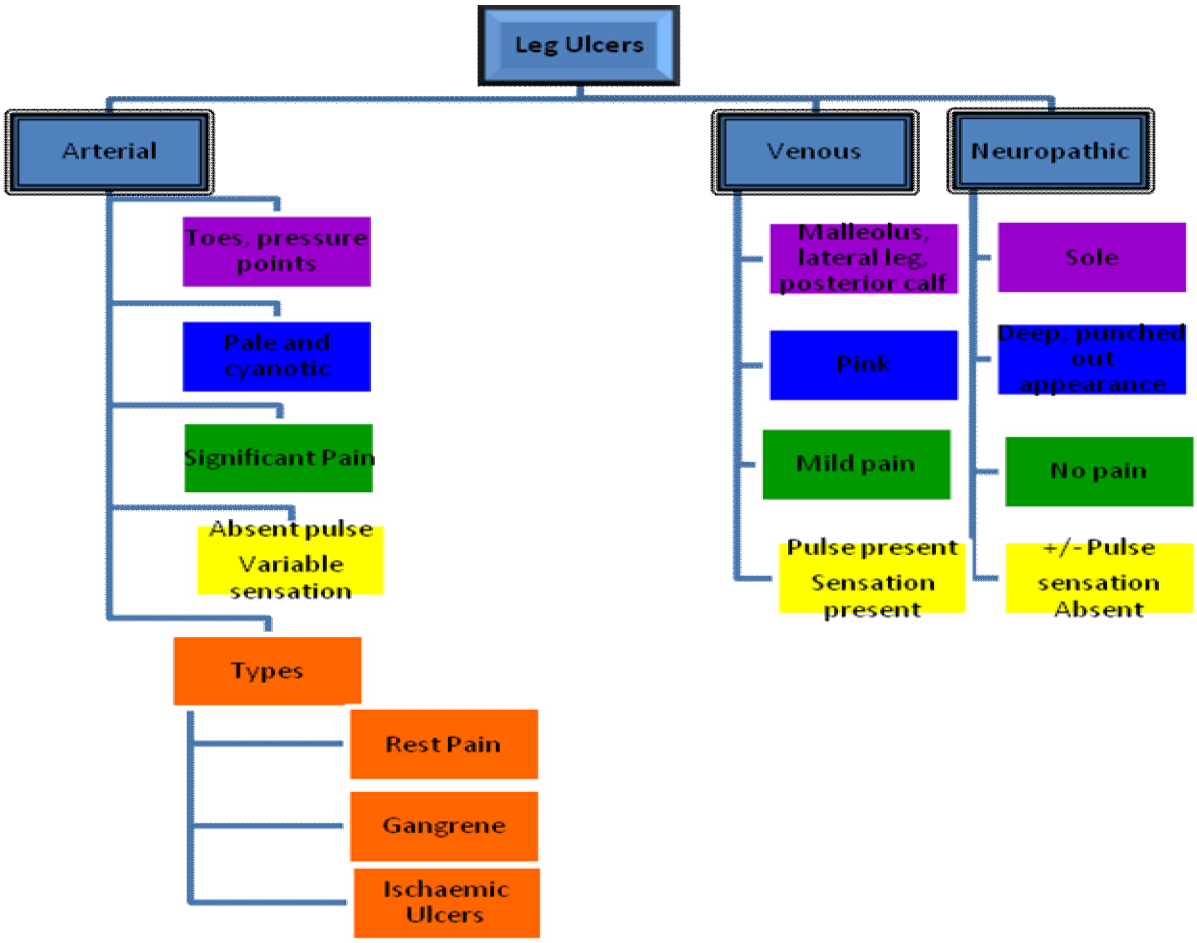

Figure 1. Comparison of the manifestations of leg ulcers in terms of location, colour of wound, presence of pain/pulse/sensation, and subclassification.[1,2]

PVD: Diagnosis

The single most useful tool in assessing the manifestations of PVD, according to the Ontario Venous Leg Ulcer Community Care Protocol, is the ankle-brachial pressure index (ABPI), a ratio of the blood pressure in the arm relative to the leg.[6] Normal ABPI is 0.9 to 1.2, venous ulcers have an ABPI greater than 0.8, mixed ulcers between 0.5 and 0.8 and arterial ulcers less than 0.5.[6,1] This distinction determines management as arterial ulcers often require referral to a vascular specialist while venous ulcers may be treated with compression bandages and topical antibiotics.[6] Also helpful in the diagnosis of PVD are segmental Doppler pressures, volume plethysmography, duplex imaging, magnetic resonance angiography, and contrast angiography.[5]

As in the case of Mrs. S, the manifestations are easy to diagnose once they are observed but the challenge is often the identification step. Despite frequent access to quality care, ulcers may go unnoticed for a myriad of reasons- they are overshadowed by more pressing health concerns or they are unnoticed even by the patient (such as in cases of diabetic neuropathy). In a study of patients receiving home health care, 7-12% had pressure ulcers.[7] This study recommended an educational program for home healthcare workers in order to promote early recognition of ulcers.

PVD: Treatment

To promote healing, the ulcerated area must be offloaded. This can be achieved through footwear modification or activity restriction.[8] The challenge is to remove pressure from the wound without compromising balance. Options for footwear modification include insoles (Sorbothane or polyurethane), plantar moulding, forefoot offloading shoes, and rocker bottom shoes. However these modifications have been shown to reduce activity level by up to 90%.[8]

Treatment of leg ulcers often involves topical or oral antibiotics with more severe cases requiring amputation. Treatment of PVD focuses on control of risk factors- smoking cessation, diabetic control, statins for dyslipidemia, antiplatelet therapy, ACE inhibitors, and exercise.[1]

Discussion

The diagnosis and treatment in this case was fairly straight forward- a known history of peripheral vascular disease, a clear mechanism of injury, recognition of the need to remove sources of pressure to promote healing and the use of antibiotics to prevent infection. The unique features of the case pertain to the can of tomatoes.

Two immediate questions are why the injury occurred in the first place and why it went untreated for six months. The answer to the first question may be attributed to advances in internal medicine. According to Dr. Hazel, a plastic surgeon in Lethbridge, Alberta, “internal medicine has

become too good- people no longer die of their organ problems; now we have to be aware of peripheral issues." In the USA, 60,000 patients die of peripheral ulcer complications annually.[7] Mrs. S had a past history riddled with internal complaints but all were well controlled with medication, thus allowing her to remain in her own home at the age of 92. So while the internists may congratulate themselves on the arsenal of effective medications at their disposal, they must be aware of the new Frankenstein their success has created. Increasing attention must be paid to issues outside of the head and thorax. The Ontario Leg Ulcer Community Care Protocol suggests that monitoring is the single best intervention in regards to peripheral ulcers.[9] Simple tests such as the ankle-brachial index are quick and easy to complete at each visit and when combined with a physical exam that extends to each digit may lead to earlier diagnosis of arterial ulcers. Care of PVD and subsequent ulcers requires a multidisciplinary approach.[10]

The response to the second question is twofold- the ulcer was not noted because Mrs. S's caretakers and physicians did not see it and because she chose not to display it. Why did she choose to conceal a bone-depth ulceration? It is likely because she knew that in hospital tomatoes came not in cans, but only in the plastic compartment of her meal tray. The can represented her independence, her ability to care for herself. This is in contrast to what Morgan and Moffatt described as the "social ulcer"- a wound that the patient hopes will not heal in order to maintain contact with the medical profession and thus the outside world.[11] The complication in this case was not the diagnosis or treatment of the ulcer, but the struggle of an elderly woman to choose between staying in a place where tomatoes came in cans and entering a place that would promote the viability of her toe. A study in *International Wound Care* identified practitioner focus on the wound rather than the whole person as one of the greatest barriers to ulcer care.[11]

Struggles such as this take time to contemplate- time that most internists do not have. Enter the medical student. We often worry that we don't have the knowledge or experience to effectively treat patients.[12] But we are also lacking something else that serves in our favour- pagers. A medical student is at liberty to spend an hour with patients like Mrs. S, working out details such as who would care for her dog.[2] So the next time you feel under qualified or obtrusive as a medical student, remember that both the physician and the patient will be grateful for your pager-less ability to acknowledge the importance of the can of tomatoes, not just the ulcer.

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Inspiration

Location: Lethbridge Regional Hospital in Lethbridge, AB

Program: One-month Internal Medicine Observership through the Alberta Rural Physician Action Plan

Coronary Artery Disease: Medical Management vs. Percutaneous Coronary Intervention

Samir Raza, BESc Candidate (Meds 2010) and Jaffer Syed, MD, FRCP

Case

Mrs.G, a 55 year old female, presented to her family doctor one year ago with chest pain on exertion. The pain was described as sharp in nature, radiating to both arms and was not accompanied by shortness of breath. Symptoms initially appeared following weight lifting at the gym. Currently, she is able to carry out all activities of daily living (ADLs) without limitation. Chest pain is elicited by brisk walking or carrying groceries up stairs, placing her in Canadian Cardiovascular Society (CCS) class I-II. She has a blood pressure of 130/72, heart rate of 60, and BMI of 27. She is a non-smoker with well controlled dyslipidemia. Upon investigation, MIBI stress testing demonstrated moderate-sized inferior wall ischaemia consistent with right coronary artery (RCA) disease. Her current medications include ECASA, atorvastatin, ezetimibe (antilipemic), bisprolol (beta-blocker), perindopril (ACE inhibitor), and a nitroglycerine spray. Following discussion with the patient, it was decided that coronary angiography and possible percutaneous coronary intervention (PCI) was not indicated at this time as symptoms were mild and a satisfactory quality of life was maintained. Mrs.G was encouraged to use her nitroglycerine spray prophylactically.

Discussion

Coronary Artery disease (CAD) is the leading cause of death in the developed world with 1/3 of all deaths attributed to CAD[1]. Blood flow through the coronary arteries becomes impaired by two mechanisms:

1. Stenotic lesions
2. Endothelial dysfunction

Fixed atherosclerotic plaques cause a narrowing of the coronary arteries. This stenosis causes an increase in vascular resistance and thus, a decrease in coronary blood flow. Atherosclerotic plaques may also be associated with endothelial dysfunction and paradoxical coronary vasoconstriction. During times of physical or emotional stress, a mismatch in myocardial oxygen supply and demand (myocardial ischemia) may result from both epicardial stenosis and endothelial dysfunction. Myocardial ischemia often (but not universally) manifests clinically as angina pectoris [2].

Signs and Symptoms

Angina pectoris is the hallmark symptom of CAD. It is frequently described by patients as a diffuse retrosternal tightness or pressure in the chest lasting for a few minutes. In women it is not uncommon for the pain to be described as sharp. It commonly radiates to the shoulders and arms. Patients often hold a clenched fist over their chest to describe the pain, this is known as "Levine's sign". Anginal symptoms are elicited by physical/emotional stress, eating, and exposure to cold[3].

Diagnosis

In addition to the clinical findings mentioned, there are a variety of tests available to aid in the diagnosis of CAD. Table 1 lists the different modalities.

Table 1. Diagnostic studies and corresponding positive findings for CAD[4].

Modality	Finding
ECG / Exercise Stress Test	ST depression, T wave inversion
Nuclear studies (MIBI scan)	Poor radionuclide accumulation in ischemic areas. Radionuclide accumulates in proportion to degree of perfusion of viable myocardial cells.
Exercise Echocardiography	Stress-induced regional wall-motion abnormality
Coronary Angiography	Stenotic coronary artery seen on fluoroscopy during injection of contrast agent

CAD can lead to and/or exacerbate heart failure. The New York Heart Association (NYHA) Functional Classification system is commonly used to classify the severity of heart failure. This classification system is based upon the degree of physical limitation experienced by the patient. Table 2 lists the NYHA system[5].

Table 2. NYHA Functional Classification system for heart failure[5].

Class	Definition
I	Patients with no limitation of activities; they suffer no symptoms from ordinary activities
II	Patients with slight, mild limitation of activity, they are comfortable with rest or mild exertion
III	Patients with marked limitation of activity; they are comfortable only at rest
IV	Patients who should be at complete rest, confined to bed or chair; any physical activity brings discomfort and symptoms occur at rest

Medical Management vs. PCI

Treatment of CAD involves medical management to control symptoms of ischemia as well as control of coronary risk factors, and occasionally revascularization (PCI or CABG). The goals of treatment are threefold:

1. Decrease frequency and severity of anginal episodes
2. Prevent myocardial infarction
3. Prolong survival

Medical management of ischemic symptoms of CAD often involves a combination of three anti-ischemic medications: nitrates, beta blockers, and calcium channel blockers[4]. Nitrates are

primarily used prophylactically and as needed for acute symptom relief. For secondary prevention purposes, ASA, statins, and ACE-inhibitors or angiotensin-receptor blockers (ARB) are strongly recommended.

Revascularization involves either percutaneous coronary intervention (PCI) or coronary artery bypass grafting (CABG). Traditionally, PCI has been indicated for discrete single or double vessel disease whereas CABG has been the mainstay of treatment for left-main or multi-vessel disease[6]. However, advances in interventional techniques and the advent of drug eluting stents have resulted in increasing numbers of patients with complex, multi-vessel and left main disease being treated via PCI[7]. Current literature suggests that PCI in stable CAD offers improved symptom relief as compared to medical therapy alone, but it does not prolong survival[8]. In the case of Mrs.G, her nuclear MIBI findings suggested single vessel CAD, and PCI could potentially be appropriate for relief of unacceptable symptoms. However, given the additional risks associated with any invasive procedure, angiography and revascularization (CABG or PCI) is generally indicated in only three situations:

1. Medical therapy has not adequately improved patient's symptoms
2. Unacceptable adverse effects associated with medication
3. Patients who are suspected to have high-risk CAD[4]

The cornerstone of management of CAD is aggressive medical therapy. When this fails, or non-invasive testing suggests a high-risk pattern of CAD, coronary angiography is indicated. Angiographic findings, patient age, co-morbidities, and patient preferences, are some main factors considered when deciding upon the appropriate route of revascularization.

Conclusion

Mrs.G presented with a one year history of chronic stable angina. Symptoms were well-controlled on medical therapy alone, and non-invasive testing did not suggest a prognostically significant pattern of CAD. Based on these considerations, continuance of medical therapy was recommended. Prophylactic use of nitroglycerin spray was encouraged, as was continued coronary risk factor reduction.

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Inspiration

Location: University Hospital in London, ON.
Program: Non-credit elective.

Adolescent hallux valgus; a novel case report and review

Anthony Main, BSc (Meds 2011) and Peter Clark, MD, FRCSC

Case

A 12-year-old female presented with a four year history of bilateral hallux valgus (HV) which caused her no pain, but a significant cosmetic deformity as seen in figure 1. Her condition had been treated conservatively by a podiatrist for 4 years with splinting, toe gel spacers, and use of anti-pronation running shoes. However, her HV deformity showed no improvement with conservative management. Her parents, concerned that the deformity would become more severe over time, consulted their family physician, who arranged for radiographic imaging studies and referred her for an orthopedic consult for surgical management.

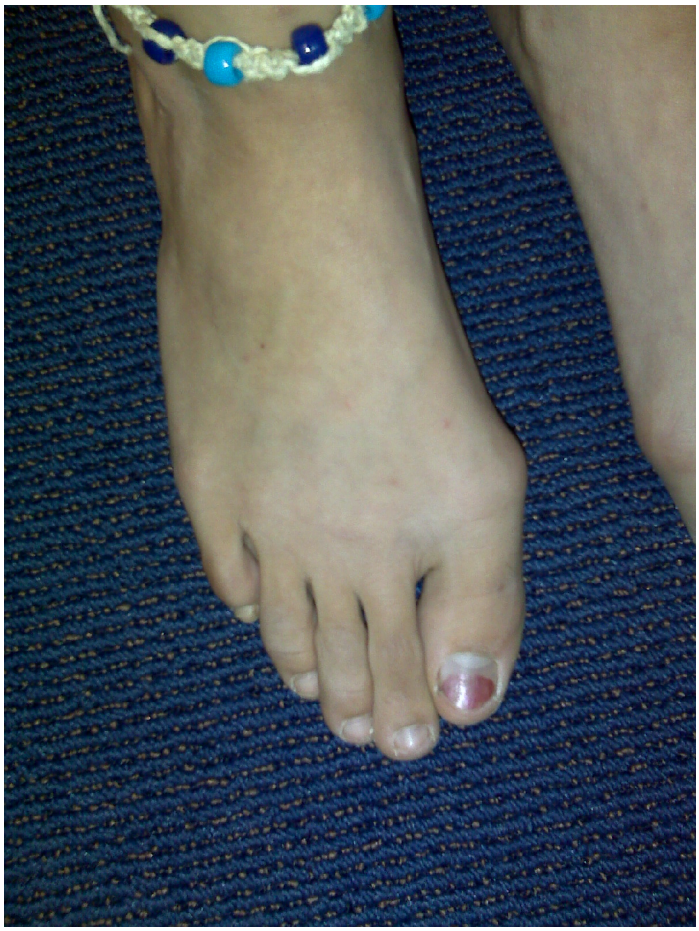

Figure 1. Juvenile hallux valgus deformity of right foot in an asymptomatic 12-year-old girl. Deformity is bilateral with a second hallux valgus deformity on the left foot.

On examination, the patient was found to have a 20° HV angle pes planus and short first metatarsal. Her medical history was non-contributory. She was born at term by cesarean section and had a fraternal twin brother.

Bilateral foot radiographic examination was undertaken with AP, lateral and oblique views of the patients foot. The patient was found to be skeletally immature with open physes, and the HV deformity was worse on the right than the left. Radiographs also revealed a congenitally short first metatarsal, HV angle of 20° and an abnormal metatarsophalangeal (MTP) joint which was cone shaped with a ring shaped epiphysis at the base of the proximal phalanx, as seen in Figures 2 and 3. To our knowledge, this joint pattern is a unique presentation of adolescent HV and has not been reported in the literature.

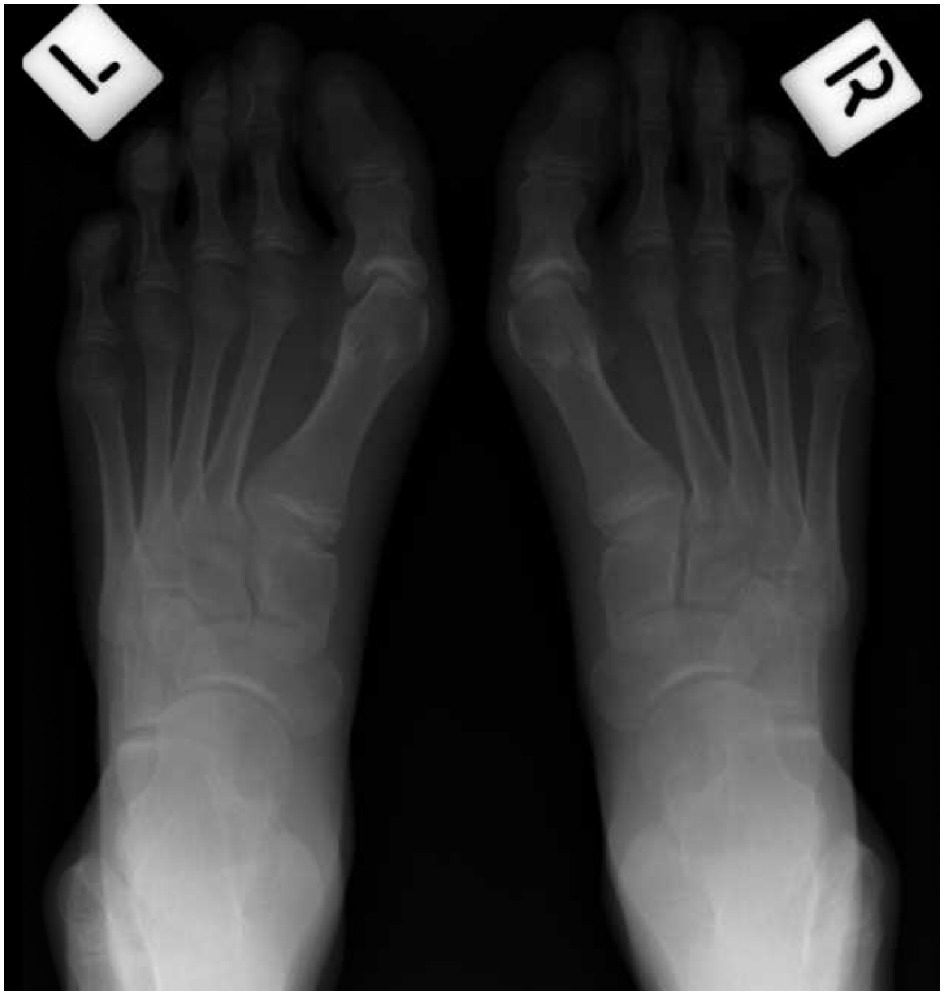

Figure 2. AP bilateral radiograph of hallux valgus deformity showing unique cone-in-cup MTP joint deformity.

As the patient was asymptomatic and experiencing no pain, conservative treatment of observation and follow up were arranged.

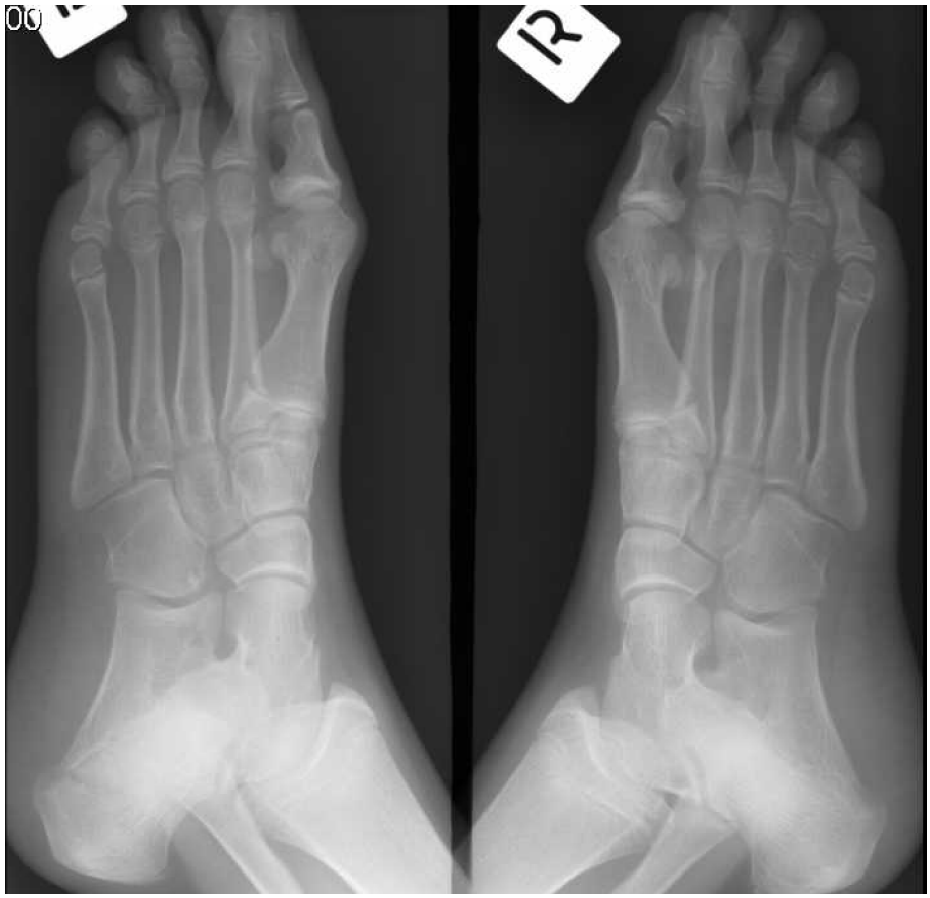

Figure 3. Oblique view of bilateral radiograph of hallux valgus deformity showing unique cone-in-cup MTP joint deformity.

Discussion

Adolescent hallux valgus, associated with a bunion, is a lateral deviation of the great toe in the transverse plane of the MTP joint. It generally affects females twice as often as males, and incidence varies depending on the population being studied and cultural practices, such as footwear use.[1] HV has a multifactorial etiology including causes such as inflammatory joint disease, genetic predisposition, abnormal mechanics or anatomy of the metatarsophalangeal (MTP) joint or the first ray. It has been proposed that HV is caused by poor footwear. Footwear is thought to be an aggravating factor rather than a causal factor as not all individuals who wear poor footwear go on to develop HV.[2]

Adolescent HV can be a serious finding as it can indicate fibrodysplasia ossificans progressiva (FOP), when found in combination with short thumbs. FOP is a very rare yet deleterious genetic disease characterized by extraskeletal bone formation. Pressure

on soft tissue causes new born formation and can lead to a 'stone statue' disease, where an individual slowly loses muscle function and freezes into one position.[3,4]

Adolescent HV is thought to be caused by metatarsus primus varus (MPV). Taken together, HV and MPV are the most common foot deformities in adolescents. As many as 50% of adolescent HV patients will also have flexible flat feet and ligamentous laxity.[3]

Diagnosis of HV can be made from patient history and clinical examination, but may require radiographic studies, especially to assess the articulating surfaces of the MTP joint and for surgical planning. Physical findings associated with HV include bursitis of the MTP joint covering, synovitis of the MTP joint, hammertoe deformity of the lesser toe, medial dorsal cutaneous nerve entrapment also known as Morton's neuroma, callus formation under the second metatarsal head due to altered weight-bearing and the development of osteoarthritis of the MTP joint.[5,6,7]

There are a number of radiographic criteria used to assess the degree of HV. First, the status and condition of the articulating surfaces of the MTP joint should be inspected. Second, the HV angle, which is the angle made between the articulating surfaces of the metatarsal and the proximal phalanx should be measured. An angle greater than 20° is considered abnormal. Thirdly, the intermetatarsal (IMT) angle, which is the angle between the first and second metatarsal bones should normally be less than 9°.[8]

Multiple treatment options exist for HV and choice depends on the amount of discomfort expressed by the patient, need for joint salvage and physician preference. There is also great debate on whether bilateral or unilateral repair is best, and whether joint fusion or bunion excision is best.[9,10]

Conclusion

We have presented a novel description of an adolescent hallux valgus in a 12 year old girl and review of current hallux valgus literature.

Conservative management of HV can include wearing of wide shoes, stretching, icing after activity, and NSAID medications. Foot orthoses have been used to prevent joint dysfunction and relieve pain. In one study of rheumatoid arthritis (RA) patients, orthoses were shown to prevent progression of the hallux valgus angle in 90% of the treatment group vs. 75% of the control group, however there was no significant difference in pain experienced.[11] Splinting is also used to treat HV, most commonly non-weight bearing night splints, however a clinical trial that examined the effects of splints on HV was not able to demonstrate any improvements in reducing pain.[12]

Surgical management is based upon clinical and radiographic findings. A surgeon must take into account a patient's level of pain and ability to ambulate before they decide to proceed with any surgery. There are well over one hundred different operations available to treat HV, but all procedures include some combination of the following. Arthrodesis is a surgery where the MTP joint is fused resulting in loss of ROM but also decrease in pain symptoms. Osteotomy with distal soft tissue correction, where the metatarsal bone is cut and realigned, is the most common surgical option. In some cases, excision of the bunion with soft tissue correction is the only operation necessary. In rare cases, a primary joint fusion of the MTP joint is required. In adolescent hallux valgus, the periosteum is very active and the osteotomies remodel very well. The choice of surgery depends on degree of angulation on radiograph, the status of the articulating surface and the patients' symptomatology.

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Inspiration

Location: Thunder Bay Regional Health Sciences Centre and Lakehead University Sports Medicine Clinic in Thunder Bay, ON.

Program: Northern Ontario School of Medicine Summer Elective.

Cavitating pulmonary lesions and hemoptysis in a case of Wegener's granulomatosis

Wang Xi, BMSc (Meds 2011) and Louise Moist, MSc, MD, FRCPC

Case

A 49-year old gentleman was admitted for investigation of multiple cavitating pulmonary lesions on CT and hemoptysis (Figure 1)[1]. He developed a dry, non-productive cough a month prior to admission with fevers, chills, and night sweats, but no shortness of breath and no chest pain. Half a month prior to admission, he was diagnosed with pneumonia by chest X-ray and prescribed azithromycin. Without symptomatic relief after one week on antibiotics, his treatment regimen was changed to gatifloxacin and metronidazole for ten days. Three days later, the patient's symptoms worsened to include a productive cough yielding yellow sputum, hemoptysis, muscle aches, epistaxis, and hematuria. He had also lost ten pounds the preceding month. The patient was deteriorating which prompted CT investigation showing multiple cavitation lesions.

The patient had quit smoking three years ago after a 30 pack-year history. He had an unremarkable medical history, and no family history of lung disease or vasculitis. He had no known previous exposures to farming, mining, or tending to birds. He had no significant travel history or exposure to tuberculosis.

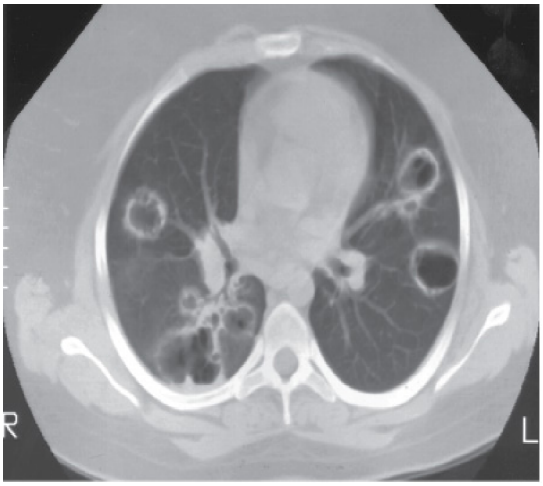

Figure 1. Multiple, bilateral cavitating lesions on CT (adapted from *Harrison's Principles of Internal Medicine*)[1].

On examination, the patient had a temperature of 38.9°C, a heart rate of 110 beats/min, blood pressure 127/72, and a respiratory rate of 16/minute, saturating 92-94% oxygen on room air. Examinations of cardiovascular and pulmonary systems were unremarkable. No finger clubbing was present. His hemoglobin was low at 89 g/L and creatinine high at 132 µmol/L. Urinalysis was positive for blood and protein, but negative for casts. Bacterial and fungal cultures were requested, as well as an initial vasculitis screen with serum ANA, rheumatoid factor, C-ANCA, P-ANCA, C3, C4, CH50, ESR, and CRP. The C-ANCA titre (Proteinase 3, PR3) was positive at 154 (> 30), strongly suggesting a diagnosis of Wegener's granulomatosis (WG). He was

started on pulse prednisone and oral cyclophosphamide.

While in hospital, his renal function declined, with his creatinine rising to over 600 µmol/L. A trial of plasma exchange was initiated with five treatments over ten days. Renal function improved and he was fortunate to avoid hemodialysis. Hemoptysis and hematuria improved with time. No renal biopsy was performed. He was started on oral Septra as prophylaxis against *Pneumocystis carinii* pneumonia and also given a no-salt-added diet. He was discharged 1 month after admission after he became stable. At discharge, his hemoglobin was low at 93 g/L and creatinine high at 407 µmol/L. He was to be reassessed and advised not to return to work.

Discussion

WG is a systemic vasculitis that is characterized by granulomatous lesions in the upper and lower respiratory tracts and focal necrotizing glomerulonephritis. All of the body's small and medium-sized vessels can be affected resulting in many potential clinical manifestations (Table 1)[2]. These manifestations may suggest several vasculitic etiologies and diagnosis may prove to be a challenge. In this case, a common presentation of WG is discussed, where a supposed pneumonia, as shown on chest X-ray, does not resolve after treatment with multiple antibiotics. Combined with further symptoms and/or investigations, the circumstances point toward an alternate diagnosis (Table 2)[3].

Table 1. Common Clinical Findings of Wegener's Granulomatosis[2]

Systemic	Fever, lethargy, weakness, weight loss, night sweats, skin lesions (rash and skin sores), arthralgias
Lungs	Pulmonary infiltrates, pulmonary nodules, hemoptysis, pleuritis
Kidney	Glomerulonephrtitis, hematuria, proteinuria
Ear, Nose, Throat	Sinusitis, nasal membrane ulceration and crusting, saddle nose deformity, epistaxis, hearing loss, ear pain, oral lesions
Eyes	Conjunctivitis, eye pain, visual loss
Nervous System	Peripheral neuropathy

The serum auto-immune anti-nuclear cytoplasmic antibody (ANCA) level is useful in both diagnosis and understanding the pathophysiology of WG. Patients with active WG are seen to have an auto-immune, antigen-dependent, inflammatory background, which is corroborated by CD4+ T cell and monocyte activation in active disease. Next, neutrophils are activated through the production of C-ANCA or P-ANCA by B-cells targeting either Proteinase 3 (PR3) or Myeloperoxidase (MPO), respectively[4]. However, the ANCAs are present in 85% of cases of active WG and are not pathognomic for the disease. Other ANCA-positive vasculitides are microscopic polyangiitis (MPA, 70% positive) and Churg-Strauss syndrome (50% positive)[4]. WG is mostly C-ANCA positive while MPA

is mostly P-ANCA positive, but this differentiation is not clinically significant as treatment for both conditions is similar. Churg-Strauss presents a different clinical picture and generally includes asthma in 95% of patients, among other atopic allergic symptoms[5].

Table 2. Typical Differential Diagnosis for Hemoptysis with Cavitating Lung Lesions[3]

Neoplasms
Infectious
Tuberculosis
Histoplasmosis, Aspergillosis, or Coccidiomycosis
Klebsiella or Staphylococcal pneumonia
Lung abcess
Septic emboli
Autoimmune
Wegener's granulomatosis
Rheumatoid arthritis
Environmental exposures
Silica

The hypothetical etiology of WG is attributed to combinations of infectious, genetic, or environmental factors; however, efforts in identifying specific factors have proved to be difficult[6]. The disease is equally distributed between men and women, presenting most commonly among the fourth and fifth decades of life[7].

The initial presentation of WG may be followed by an indolent, mild, or aggressive illness. Serum ANCA levels are seen to vary with the extent, severity, and activity of the disease. A limited form of the disease can restrict itself primarily to the lungs and is less likely to be ANCA positive, but this form may still eventually progress to the kidneys[8]. Poorer survival is associated with older age and the need for dialysis[9].

Treatment of WG has advanced greatly since the disease was first described in 1936, when 1-year mortality was 100% with median survival of 5 months[10, 11]. Treatment with corticosteroids in the 1960s procured a median survival time of 8 months[12]. The most significant medical breakthrough was the introduction of cyclophosphamide in the early 1970s that prolonged survival and relieved symptoms.

Overall, the treatment plan is two-fold: to induce remission in patients with active WG and to maintain remission once the goal has been achieved. The strategy in treating WG is primarily immune suppression given the inflammatory nature of the disease. Patient symptoms, disease activity, organ involvement, and lab test results are all determinants of a patient's particular treatment plan. In severe WG with kidney involvement, cyclophosphamide and prednisone are generally prescribed, providing symptom relief in 91% of patients and inducing remission in 75% of patients[2]. Due to the immunosuppressive nature of cyclophosphamide, it is preferred to switch to methotrexate or azathioprine after three to six months. Induction can be achieved in patients with mild disease with methotrexate and prednisone. During treatment, prophylactic therapy with Septra should be used to prevent *Pneumocystic carinii* pneumonia. Septra has also been associated with a decreased recurrence of WG. The

presence of renal failure in a WG patient increases the risk of end-stage renal disease and mortality. For patients with creatinine over 500 µmol/L, plasma exchange has been shown to reduce this risk[13].

Even with treatment, WG can recur or maintain a low activity level in 60-80% of patients, leading to organ damage and chronic morbidity[2, 14]. Important risk factors for relapse were C-ANCA positivity and disease within the lower or upper respiratory tracts[15]. Overall mortality for WG patients is four times the rate of the mortality of the general population[16]. Most (93%) of the morbidity and mortality was due to disease-related activity rather than treatment-related causes[9].

Table 3. Key Points

- Wegener's granulomatosis is a complex vasculitis with many potential systemic manifestations
- Suspect alternate etiologies in suspected cases of pneumonia that does not resolve with multiple antibiotics
- Goals of management are induction and maintenance of remission
- The patient on immunosuppressants for WG should be given prophylaxis and be vigilant for infections
- Reduce cardiac risk factors as vasculitic diseases increase the risk of an adverse cardiovascular event

Conclusion

WG is a systemic vasculitis with many potential clinical manifestations and is challenging to manage in the clinic. The diagnosis of WG has potentially devastating impacts on patients' lives given the high risks of organ damage and recurrence of the disease. Treatment of WG reduces mortality and morbidity but does not eliminate these risks altogether. Consequently, patients should be given regular follow-up to monitor disease remission and organ function.

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Inspiration

Location: Victoria Hospital in London, ON.
Program: Non-credit elective.