Preventing Amputations in Diabetes

Presented by

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Introduction and Overview

My name is Greg Caputo. I am on the faculty and staff at Penn State Hershey Medical Center, and each year I spend some time at the Cherokee Indian Hospital as well in North Carolina. I am always very inspired when I spend time at Cherokee and I am inspired by what I have seen and heard thus far today, and I am inspired by all of you and what you do.

I wanted to give a very brief special thanks to two colleagues, Dr. Jan Ulbrecht, and Dr. Peter Cavanagh, real experts in this field of diabetic foot care, who have really been mentors for me during my career and any wisdom that's imparted in this presentation is a credit to them and other experts in the field and any inaccuracies are entirely my responsibility.

So as far as my own expertise, I'll harken back to this definition of an expert that some of you may know. Someone that travels a great distance and brings a PowerPoint, and in order to bolster my credentials, I have this map of the US just to show you just how far it is from Central Pennsylvania to New Mexico.

Finally, by way of introduction I just wanted to make it known that all the case vignettes, and there will be a number of them, are composites and don't contain any specific information on actual individuals.

I always like to start out by going over the takeaway messages and set the context for the discussion. Now, I hope this is a discussion and an interactive presentation and then circle back to these takeaway messages as we conclude. This discussion will focus on primary care clinicians, should have relevance to other clinicians who care for patients with diabetes, but these next two slides show the major takeaway points.

A primary role for primary care clinicians is to identify high-risk patients with an annual foot examination, and to enter those higher risk individuals into some structured prevention program. We will talk about that in some detail.

For primarily neuropathic ulcers that manage and includes effective pressure relief and debridement. These are the two elements of care that we most often see missing from the care of patients with longstanding neuropathic ulcers. So it's particularly important that we be mindful of that and emphasize that. For primarily ischemic ulcers, a bedside evaluation, noninvasive evaluation, that we will discuss is fundamental, and also specialty consultation, as we will talk about in a minute and the reasons why.

We will talk some about infection in the diabetic foot, complicating ulcers and it's key to be able to recognize at the bedside clues to more severe infection, to recognize that milder infection is -- one can usually treat those infections as an outpatient with close followup, but for more severe infection admission of course with immediate surgical consultation and IV antibiotics.

The next takeaway message if that for the acute charcot foot, it's critical for primary care clinicians to be very well versed in the clinical presentation of the warm, swollen foot in a patient with neuropathy. We will talk a little bit about the differential diagnosis, but how important it is to keep charcot foot in the differential of that syndrome. And then finally, to use a multidisciplinary approach and to address this focused issue with the patient's lower extremities in the context of the whole person.

I think we all have a good sense about what the costs are of the diabetic foot complications. We know that diabetes is the leading cause of non-traumatic lower extremity amputations in our country, that the treatment of diabetes and its complications is horrifically expensive in monetary terms, that at

least 33% of these costs are linked to treatment of foot ulcers. And that as you know better than anyone that these issues impact native people to a far greater degree than they do others. But we also know that these statistics don't even come close to measuring the true cost, the human cost, the cost in human suffering. So, it's really that that we'll keep our emphasis on as we talk about these issues today.

Screening and Prevention

So here is a case vignette of a 45-year-old man who had a 20-year history of Type 2 diabetes, had a foot ulcer on the plantar surface for about eight months, had no pain, no drainage, and his physician had prescribed a series of courses of antibiotics, multiple different types of dressings, and told him, that is, told him, "Stay off your feet." We've heard that story, haven't we?

So here is what the foot exam look like (refers to slide # 9 in handout).

As you can see, by this image, there has been some debridement of a callus around the ulcer, but the ulcer is how it appeared in the office that day. So let me ask you, what is it that you see there? Can you describe this? And is this lesion, can this heal after eight months of repeated efforts to get this lesion to heal? And it really has made no progress. First, what do you see there?

So I see some granulation tissue centrally. So this is a wound that has pretty good looking tissue, I agree with that. There is good looking tissue, well-perfused tissue at the base of this ulcer. That's an important finding, one of the hallmarks of a neuropathic ulcer that is being well-perfused.

The white looks like there is some maceration, and there was some macerated callus around that, and that actually is what was left after there was some sharp debridement of that devitalized tissue around there. So there was some surrounding callus and macerated tissue, a healthy appearing wound. I can tell you that - although again, there is some Betadine around there to prep the ulcer- there were no real signs of inflammation or drainage in this wound and the patient had excellent dorsalis pedis and posterior tibial pulses.

So what do you think about the ability of this wound to heal after all that time (refers to slide #10 in handout)? This is something that clearly when managed in somewhat of a different way, taking a look at it in a somewhat different way, implementing some of those key issues that we talked about, I mentioned in the introduction, this is something that can heal.

So how is it that diabetes ends up causing foot ulcers? And I think you are well familiar with this concept of Loss of Protective Sensation (LPS), a critical early step in the formation of neuropathic foot ulcers in diabetes. That is sensory neuropathy, severe enough that skin injury is no longer painful.

Loss of protective sensation as you see here leads to a foot ulcer as you see here by starting off with an inability to feel pain(refers to slide #12 in handout). And then this is an image of a footprint that shows you these high peak pressure areas under the metatarsal heads. And so that combination of sensory neuropathy with high plantar pressure results in calluses as you see here. Calluses which are a result of the pressure and contribute even more to the pressure at those focal areas. It's like walking on a stone.

So it begins to injure the tissue, you start to see some tissue damage, as you see here in the pre-ulcer phase with some flex of a hemorrhage into that callus, and then finally, a full thickness ulcer occurs. So that is really the pathogenesis beginning with loss of protective sensation to neuropathic ulceration.

So I'll review with you some of the recent most current ADA foot care recommendations which acknowledge that fact.

So for all patients with diabetes, the ADA recommends that we perform an annual comprehensive foot exam to identify those risk factors that are predictive of ulceration and amputation, and that includes inspection as you see here, an assessment of foot pulses, a test for loss of protective sensation, and we'll talk a little about that monofilament as I am sure again you are familiar with.

But for the first time, the ADA is now also recommending that we implement one of these other tests, I won't go through them each in detail, but that we do loss of protective sensation with a monofilament and then include one of these tests to determine whether or not they are at risk for a neuropathic ulcer.

This is the technique for doing monofilament testing (refers to slide #14 in handout). Is everyone familiar with monofilament testing? I would imagine that you are quite familiar with the 10-gram monofilament. But it's a very useful and quick and effective way to identify loss of protective sensation. You see the technique here, in the upper panel here, place the monofilament against the skin, apply pressure until it buckles, and hold it in place for a second, release it. The patient's eyes close, you ask them, do they feel that pressure?

You should test the highlighted sites with the patient's eyes closed, and if there are any misses, so to speak, if the patient cannot feel any one of those sites, then they should be considered to have loss of protective sensation, early loss of protective sensation, they will be patchy, which is why we check a variety of areas. You don't want to test obviously over an area of callus.

So this shows in photographic form a demonstration of using the monofilament (refers to slide #15 in handout).

The ADA also recommends that we provide general foot-self care education for all patients with diabetes, that as I mentioned earlier, we use a multidisciplinary approach for patients who are at high risk, who have foot ulcers clearly, someone who has had a prior foot ulcer amputation. The ADA recommends that we refer patients to foot care specialists for ongoing preventive care, surveillance those who have high risk by virtue of their initial exam, smokers, those who have prior lower extremity complications. So again as you see here, our role in primary care is to identify those high risk patients. Those who are not at risk by those tests can be reassured, given some education and screen again in a year. Those that who have a risk factor, we need to engage them in a preventive care program, which we'll mention in just a minute.

In terms of vascular disease, clearly take a history regarding vascular arterial insufficiency symptoms, check pedal pulses as I mentioned. They recommend that we consider obtaining an ankle-brachial index, pressure at the ankle, pressure in the arm, comparing those. Many patients with PAD, they note are asymptomatic, and then to refer those patients who have claudication or a positive ABI less than 1 for further vascular assessment.

So this is an algorithm I think is reasonably practical (refers to slide #18 in handout), and the goal today is to give some practical, usable, day-to-day information in your day-to-day practices. So screening annually, a careful history of foot exam as we discussed, checking for loss of protective sensation, a vascular assessment, if normal, educate, continue that annual screening. If there is higher risk, then enter into a prevention program of self-care, some professional care, podiatric care for ongoing callus and nail care, for example, and footwear, which is critically important. Most of us as primary care physicians are not well-versed in that. We are not going to talk about the nuances of foot care, but it's clear that those patients who are neuropathic need to have a professional recommending

appropriate footwear, because that's a contributing factor to high pressure, and if a patient can't feel tight shoes, doesn't have the support they need in footwear, then that can just accelerate the pathway.

Neuropathic Ulcer Care

So what about the patient who develops an ulcer like the case #1? What are the principles of management and many of my colleagues are fond of saying, it's not what you put on a wound, it's what you take off, referring specifically to pressure relief and debridement.

56-year-old man, foot ulcer for two years plus, multiple surgeries, skin grafts, courses of antibiotics, every dressing and potion and lotion that you can imagine known to humankind, and this wound had failed to heal (refers to slide # 20 in handout). Can this wound heal? Same thing, good pulses, completely insensate, good-looking tissue at the base, generally healthy tissue. This is far more advanced than that one I showed you in the first -- is this one that can heal? Depends. So the point was, is the diabetes controlled? So do we have close enough, tight enough glycemic control to facilitate healing? Any other factors that depends on? So whether or not there is some local ischemia. Thankfully again, it looks like the base of that wound is well-perfused, but certainly you have to at least be thinking along those lines for somebody who hasn't healed. Are they staying off of it? So that really gets to the key issue. Are they staying off of it, meaning has pressure been adequately relieved from the side of the ulceration, because we talked about how important pressure was in creating this ulcer. And this ulcer will not heal without effective, innovative, creative ways to try to relieve pressure. A very important tool is the Total Contact Cast.

So this is the technique that we used and it's about three months later this lesion healed. It was really the absence of effective pressure relief that caused this lesion to be present for two full years. I think this is one of the best examples I've seen of how important pressure relief is.

Now this Total Contact Cast as you maybe able to see has a heel on it at the bottom here. The patient is fully enclosed. Because all of this tissue was debrided, we did some test to make sure there wasn't osteomyelitis etcetera. But at the end of the day it was effective pressure relief while allowing this patient to be reasonably ambulatory, at least walking around the house with a cane, and that allowed this to heal.

So Total Contact Casting, this isn't something that anyone expects that a primary care clinician is going to do in their office, but it's so important that we recognize these tools that are available, and that we somehow when we see that pattern of failure to heal, good vascular supply but no infection but failure to heal. We are thinking about how do we tap into our community resources to get pressure off this wound.

So the Total Contact Cast, the hypothesis is that if you intimately mold the cast material around the entire surface of the foot and ankle and lower leg, this is a cutaway version (refers to slide #21 in handout) on the bottom just to show what it looks like. It equilibrates the loading away from that focal point and the rest of the foot and ankle and even calf bears the weight. These casts are changed weekly and sometimes will bi-valve it. If we are concerned we'll make a Clamshell Device and wrap it around with some Velcro, so that the visiting nurse, so that we can have the patient come back and take a look at that wound on every few days at least for the first couple of weeks.

But there are other offloading devices that are clinically effective. You don't have to have Total Contact Cast resource in your community to be mindful and attentive to pressure relief. A CAM Walker as you see here (refers to slide #22 in handout), a wedged half-shoe, if the patient has got a small forefoot ulcer. The Instant TCC, I think this is an ingenious invention, colleagues David

Armstrong and others in Arizona. So you put the CAM Walker on, as you see towards the left and then you just wrap with plaster or a cohesive bandage so the patient can't get out of it because really if they can get out they oftentimes will. And we tell patients that a single step a day is enough to prevent healing, a single unprotected step a day is enough to prevent healing.

So it's not just going to the bathroom and back, that's way too much. Not standing in the shower, you can't stand in the shower unprotected. We engage them in creative ways to help them figure out how they can prevent weight-bearing, unprotected weight-bearing. Dr. Ulbrecht is just fantastic at this. He will sort of go home with the patient in his mind with the patient, and sort of strategize, well, what are you doing during the day? What are the pitfalls, where are the landmines that you're going to end up with that tendency to bear weight if they're not in a fully enclosed cast? So that's really what you need to do to help creatively get pressure off this. Removable Cast Walkers, there are not a lot of devices, and again, the key is to engage those people in your community that are well-versed in these. Yes.

The fully enclosed Total Contact Cast has traditionally been thought of as sort of the Gold standard, but frankly there is not robust data on comparing these various methods.

And there is a large NIH study that's enrolling patients to compare these. There are some smaller studies, but we really need some better evidence to really determine what the effectiveness of the variety of devices are.

The other thing as I mentioned before one patient used a cane. You should consider some gait-stabilizing devices as appropriate, because these things can insert some gait instability. So if the person is just walking in one of those wedged sandals without a sole in the forefoot they can tend to fall forward. So you want to make sure that they are having some stabilizing device as appropriate, and as always, and again, this is sort of nuts-and-bolts that I'm sure you are well aware of and adhere to, but just clearly documented that the patient understands and accepts these benefits and risks that may not be critical from your standpoint because you may not be actually putting these casts on. But again it's just a matter of reinforcing and making sure the patient understands what the plan is.

We mentioned Debridement. This is a patient who came into our clinic with this (refers to slide #25 in handout) - in fact, the Physical Therapist called me and said that there is this patient with this callus and some flecks of hemorrhage in the center. And she said, she thought that there was probably an ulcer underneath, so I went down and saw this patient and just basically debrided that callus, but there was an ulcer underneath here as you see.

She knew that, she was suspicious that that was going to be the case and there was. Again, a clean appearing, very healthy appearing tissue there. A little bit of surrounding erythema, wondered whether that was from pressure or whether there was some early cellulitis. But the bottom-line is that we debrided that and most experts I think believe that sharp debridement of that devitalized tissue is important in preparing that wound to heal. But there is frankly limited evidence, so I wanted to make sure that you were aware of that; that even though it has become standard practice in general to do sharp debridement of these wounds, there really isn't great evidence for that. There are some studies, but most authorities I think believe it's an important component of management. So again, it's not what you put on these wounds in general, it's what you take off, offload the pressure and remove the devitalized tissue.

This is probably the shortest discussion in any -- you will hear on local care. For what sort of dressings to put on. This gets entire lectures at diabetic foot meetings, entire conferences on local dressings, etcetera. I am deemphasizing it some because I think there is not a lot of evidence that more advanced therapies and dressings are a major advance over simple gauze dressings that are moist. But it is clear that as I mentioned here in these four bullet points, healing is accelerated with

occluded moist wounds. I don't believe there is any clear-cut evidence for one type of dressing product or another. Now there are some excellent wound care centers and physical therapists, wound care nurses that have a regimen that they use and I don't try to discourage that if they're using these cautiously for selected patients. But my point is that the answer isn't the best dressing as you saw before with that dramatic case. We put that man in the cast and that thing healed. We weren't putting a lot of different advanced biological therapies etcetera. One thing we do avoid is antiseptics and detergents. So we don't put a Betadine dressing, a gauze soaked in Betadine on the wound. There is some evidence that antiseptics, detergents etcetera might delay healing. Some of these advanced therapies as I mentioned and as listed there, there are ongoing studies and they may have a role in selected patients, but I don't think they're the core blocking and tackling part of managing these wounds.

A very nice review here shown on the slide (refers to slide #27 in handout), consensus recommendations on advancing the standard of care. Much of what I have talked about and much more is found for those who are interested in a more -- a very expert summary of wound care management.

So the bottom lines for neuropathic ulcers, they require pressure relief in order to heal, limited evidence for sharp debridement, but it's grown to be part of the standard management, moist wound environment in a multidisciplinary approach.

Icshemic Ulcer Care

So here is the next case (refers to slide #29 in handout), a 78-year-old woman Type 2 diabetes, coronary disease, nicotine dependence, hypertension, the other co-morbidities that are listed there. Hospitalized for diverticulitis, gets pneumonia while in house and these are the lesions that we are seeing in the office.

So I will ask you the same question that I did before, what do you see here (refers to slide #30) and can this heal? Any thoughts on what you see? No granulation. What do you see in place of granulation? Necrosis, an eschar, a dark blackened eschar, and that's a hallmark of?
Tissue death. Tissue death. Likely from?

Ischemia, exactly! So this is tissue that is not well-perfused. Again I can tell you that this patient's pedal pulses were absent, and as you see here a very well-demarcated eschar on top of this wound. So this is a very different condition than what we've just been talking about for the past 15 minutes or so. This is ischemia, arterial insufficiency is really driving this process.

So critical ischemia could be defined as at rest pain, ulcers, or gangrene, in one or both legs attributable to objectively prove an arterial occlusive disease, and that's what this man had. He had ulcers and some localized gangrene that was -- we found -- we had high suspicions that this was due to arterial insufficiency and we confirmed that with some testing.

This is something (refers to slide #32 in handout) that to go along with the interdisciplinary theme that we are discussing. It's something that requires some initial wound management, that's very cautious frankly. It's basically just to protect this wound and to make sure we don't go on that path of intervention that we were just talking about with neuropathic ulcers. We are not going to put moist dressings on this wound. We are certainly not going to debride that off. We are not going to put this patient in a cast. That's just not going to work. You can relieve all the pressure you want here and we should try to protect the wound and keep pressure off of it, but that's not going to heal this. Really, this is not likely to heal without restoration of pulsatile flow. So this is a setting in which, again, reaching out to the community where your expertise is to find the expertise with vascular consultation. It doesn't

imply that every patient is a candidate for surgical reconstruction. We all know that many of these patients are elderly, they've got a lot of co-morbidities, they may not tolerate surgery. But most of the patients who get reconstructive surgery or reperfusion surgery bypass have these co-morbidities and with modern techniques this can be a limb-saving intervention. So again we are not going to take a deep dive into arterial reconstructive surgery. The points are that we should be able to recognize at the bed side the two ends of the extremes there that we want to protect this wound until we can get a specialist to weigh in on whether this patient is a candidate.

Of course, you were going to be weighing in with the patient. The patient is going to be having the most say in all of this. We know that. This is where the patient is the most important person on this team, and so it's vital for you as the primary care clinician to be understanding, advocating, representing and listening to their views on this and their perspective.

So I will show you some ulcers (refers to slide #33 in handout) and maybe you can give me your sense about whether this is primarily neuropathic in etiology or primarily ischemic in etiology. What about this one? Neuropathic - it goes along with those hallmarks that we talked about before. Generally, a healthy appearing tissue, it's got that rim of callus around it from pressure. Again, good pulses that you can't see in the image obviously, but it doesn't have that eschar that we just saw in that ischemic wound.

What do you think-ischemic? This dramatic black eschar (refers to slide #34 in handout). There clearly in all likelihood was some pressure, probably from a tightly fitting shoe on the medial aspect of this man's hallux. You can see there is one on the tip of the second toe as well and this is from shoes in a patient with arterial insufficiency.

This is the one that I showed you earlier (refers to slide #35 in handout), one of the ones I showed you earlier, again healthy appearing tissue.

Neuropathic, sure. I heard ischemic and I agree that this is one with that dramatic black eschar that is unlikely to heal without restoration of blood flow. Not impossible, but nearly so (refers to slide #36 in handout).

So we've talked about these contributing factors to amputation. Neuropathy, the pathway we talked about at first with pressure on an area of the foot. A callus that generally then leads to a skin ulcer, and about 85% of the time it's that pathway that's the primary driver of the amputation. About 15% of the time it's the ischemic pathway that drives amputation. Now we know that very frequently these conditions coexist. There is some ischemia and some neuropathy that are coexisting and contributing. But again typically at the bedside we can get a reasonably good assessment of what the primary driver is as we've just discussed.

Infection

Let's talk a little bit about infection and note that infection can complicate and can actually accelerate either one of these pathways that we just talked about. It's important to realize again, these are patients you will see in your office that the diagnosis of infection is made by carefully listening to the patient, by carefully examining the patient and it's not really made in the microbiology lab by doing cultures.

This is something (refers to slide #39 in handout) that at bedside you should have a good sense of whether or not this wound is infected. So this is what, these are lesions that didn't have any signs of a local erythema or drainage or warmth that would characterize an infected wound. This as I think I

mentioned before there's some local erythema. So this we were convinced was showing some signs of some mild or some early infection. This on the other hand a much deeper wound, more extensive erythema. This is a more severe infection.

Dr. Ben Lipsky collaborated with a large group to define an international consensus on the management and prevention of diabetic foot complications, and I think these clues still hold true today that if these factors are present clearly there is going to be – the infection is going to be at the more severe end of the spectrum, certainly penetration into the subcutaneous tissues. If there are signs of systemic illness, leukocytosis, fever, chills etcetera, that's clearly going to put it into the more severe category, that's obvious. Complicating features such as a foreign body, a puncture wound, an abscess draining, venous insufficiency, that's more likely going to be a severe progression despite appropriate antibiotics or apparently appropriate antibiotics and ischemia. All of those would define the more severe end of the spectrum, and the small ulcer fairly superficial with just a little bit a few centimeters of local erythema would be at the other end of the spectrum. It requires clinical judgment. These are not hard and fast rules but these are the sort of guidelines at the bedside that could help you make those distinctions. And I did want to mention and remind ourselves that systemic signs and symptoms in diabetes maybe absent, even in severe infection. So you wouldn't want to wait until those were happening before you say this is somebody that's got a severe infection needs to be in the hospital.

I am going to go over some details related to infection. The microbiology of mild versus severe is different. At the mild end of the spectrum it's usually staph and strep, sometimes MRSA, but it's usually fairly straightforward microbiology. You really don't need to do cultures and empiric treatment from the antibiotic standpoint is usually a Cephalosporin like Cephalexin or occasionally Amoxicillin, Clavulanate, Augmentin occasionally levofloxacin in a patient who has penicillin allergy, severe allergy, occasionally Clindamycin though it's not used too much. If MRSA is thought to be present, then Trimethoprim Sulfa or Doxycycline and usually treat for a week or two and include the management for the pressure ulcer, for the ulcer that we just talked about.

In areas where MRSA has high prevalence even in the outpatient setting, then using an agent that has predictable activity against MRSA in your community, if that's Clindamycin then that's reasonable. Trimethoprim Sulfa or Doxycycline may or may not be active, they tend to be active but there maybe local resistance patterns that prevents you from relying on those. Clearly we need to be mindful of what our local resistance patterns and our local pathogens are.

For more severe infection again just a reminder that systemic symptoms might be absent even in quite severe infection, and for patients who have more severe infection then you do need to cover polymicrobial organisms.

And the empiric treatment that we would typically use would be a beta-lactam, beta-lactamase inhibitor, I like Piperacillin, a Tazobactam, Timentin or others or Clindamycin plus a gram-negative agent like a fluoroquinolone or a third generation cephalosporin. And in areas where MRSA is going to be predictably present then adding Vancomycin to that patient when they are in the hospital would be routine. If not, then you can give the other empiric treatment and then add a Vancomycin as needed.

Empiric antimicrobial therapy at one end of the spectrum typically gram-positives usually staph and strep, a first generation Cephalosporin or an anti-MRSA drug if that's needed. At the other end of the spectrum the patient is admitted to the hospital broad spectrum antibiotics, add Vancomycin for MRSA if you need to.

Complications and Summary

I think I am going to note that diagnosis of osteomyelitis is controversial. I am not going to again take a deep dive into this. There is a lot of controversy, lot of debate, sometimes that debate sheds more heat than light. But this is a controversial area and I think this was an important paper in JAMA in 2008. Butalia and colleagues "Does this patient have osteomyelitis in the lower extremity?" And the key findings in that review paper were that an ulcer area larger than two centimeters, positive probe to bone test - they're taking a probe and gently seeing if there is a track that already exists that leads to bone that could be palpable - highest sed rate, abnormal plain radiograph were helpful. A negative MRI makes the diagnosis much less likely when the above findings were absent but that no single historical feature of physical examination finding reliably excludes ostia. And the diagnostic utility of a combination of findings is unknown. So again when this is being strongly considered, again we have to tap into what local expertise we have to try to get our heads together and make a judgment as to whether this patient has osteomyelitis.

And so in recognizing as I mentioned before that both neuropathy and ischemia contribute to many foot ulcers and diabetes. If we can at the bedside make a judgment as to what the primary driver is, we have found this type of algorithm to be helpful in managing patients at the bedside. So the patient with this ulcer, if it's neuropathic, we will look for local signs of infection. If there is no apparent infection we'll implement the treatment regimen that we mentioned before, debridement, local care and some pressure relief with close follow-up. If it looks like there is some mild infection then we would implement those foot ulcer treatment elements but add some oral antibiotics and closely follow that patient, because if they are going in the wrong direction then we're going to need to reconsider admission or broadening the antibiotics. If there are signs of severe infection or moderate-to-severe infection then that's the patient that we'll admit to the hospital for surgical drainage and debridement IV antibiotics. The essentially ischemic ulcer that's where we will again reach out and get some consultation to see if this patient is a candidate for revascularization and if this wound is infected, then we will admit that patient to the hospital because that's a little bit threatening process there. For all cases we're going to have to be considering out whether Osteomyelitis is present, do a plain film to start out, sed rate, and then consider some of those more advanced imagings in a subset of patients. And as I mentioned before and we've highlighted to use an interdisciplinary approach.

One final case, a 35-year-old woman (refes to slide #47 in handout), Type 1 diabetes, sensory neuropathy, three days of the swollen foot in the lower leg. What do you see there? Charcot is suggesting us to the underlying cause and certainly that ended up being what this patient had and we see a swollen foot with some localized erythema. There was some concern in this patient for local cellulites or deep cellulites or abscess. There was concern about whether this patient had a DVT because the edema actually extended up into the calf. But as you mentioned, this patient ended up having an acute Charcot foot densely neuropathic patient. You see here in the bottom right that the left foot there is the ankle and lower leg is swollen compared to the right.

Charcot doesn't really have a consistent definition in my view in the literature, but I think, general and my colleague Dr. Ulbrecht would say that it's a process leading to dislocation. There may be fracturing and fragmentation, resorption of the bone around a nueropathic joint. It's usually associated with fluoride signs of inflammation, as we said, swelling in erythema, warmth, but essentially without pain. The initial management, again we are not going to go into detail, but it's critical to understand that the initial management once the diagnosis is made is pressure relief and some compression, usually in a cast, there is some variety of approaches, but there are no randomized trials that I am aware of for various pressure relief measures.

And so again, it's important that once our role is to make sure that we are including Charcot in the differential. I don't think anyone would argue with the fact that we should be considering the possibility of infection in this foot. Or that we should be considering the possibility of DVT, venous thrombophlebitis there. But what we need to make sure of is that we are also considering the acute Charcot foot because the x-rays usefully by this time are suggestive if not diagnostic. Sometimes they are normal and you need to repeat the x-rays over a few days. But the point is, if we are highly suspicious and we've got a patient with an acute Charcot, we need to bring in a consultation with folks who have some expertise in managing this, and as we just discussed.

So let's circle back to the take-home messages that I started with, and say that our role as primary care clinicians is to among other things as we will talk about identify high-risk patients, we talked about the ADA current guidelines, identify high-risk patients with an annual comprehensive foot exam, and then enter those patients into a structured prevention program if they are at high-risk. Engaging the patient, education, we heard about motivational interviewing, professional foot care, callus care, nail care as indicated, appropriate footwear. If the patient is unfortunate enough to have a nueropathic ulcer, as we have shown in several cases, the management must include effective pressure relief and debridement. As I mentioned before the evidence for debridement is less striking clearly than it is for pressure relief, but the debridement thought to be an important part of care.

For the ischemic ulcer we need to be able to recognize that those findings at the bedside. If we need to do non-invasive evaluation absolutely. But specialty consultation early in the process, I think quite promptly, to give that patient the benefit of that expertise of at least considering whether or not revascularization is something that's appropriate to them.

For infection, we talked about the clues to severe infection, core competency, I think for all of us in primary care. Milder infection treated as an outpatient typically with close follow-up, mobilizing the support structure that we can to help that patient deal with this. More severe infection, those patients come in with a consultation for that debridement of devitalized tissue making sure there is not an abscess there and IV antibiotics. We talked about recognizing the clinical syndrome of a Charcot. And if you are practicing in an emergency department or primary care setting, that acute swollen, red, warm foot, a nueropathic patient, certainly consider infection DVT, but we want to make that Charcot, acute Charcot is in our differential.

And then finally, as I know is just characteristic of many practitioners that I have had the privilege of meeting and working some within the caring for Native Americans in the IHS, an interdisciplinary team-based approach is I think one of your signature strengths and vary admirable and I find inspiring.

Thank you!