

NITIN GARG: All the people who come to you as of now have talked to you about what's new and what's cutting edge and what we are doing and how can we just keep pushing the envelope. My talk is going to take you back to pretty much the basics. There's not going to be anything new. If you have any questions, I'd think this-- I would be fine if you want to make this a more interactive session so you guys don't go to sleep [INAUDIBLE]. Nothing to disclose.

We're going to talk about some of the ideologies for leg pain, leg edema, what is vascular, what is not vascular, because there's a lot of confusion, a lot of things that come into play when we see patients in clinic, both at a primary care level and as a specialist. So this patient of mine is a 46-year-old female who works as a manager in an apartment building complex. And she complains of pain in the leg whenever she walks between the buildings, which she thinks is very disabling for her.

She is an obese lady. She's heavy smoker, about 1 and 1/2 packs a day, has coronary disease, and at a very young age already had an MI and stents put in. So she is on anti [INAUDIBLE] therapy, beta blocker, and [INAUDIBLE] therapy. So these are the questions that would come to you when the patient presents to you.

What is the next step? Do you go on and do an invasive study? Do you go on and get a cross-sectional study to see what their problem is? Do you just try and manage them and see what's going on?

Do you start with the medication therapy? Or do you address something completely different, which is their diabetes or potential for diabetes or their heart? So when I'll see a patient like this in clinic, I think the first step is, I want more information. This is just not enough what she describes to me. The first and foremost is, what is the character of her pain?

Is this pain like a shooting pain, bursting pain? Or is this pain an aching pain or-- almost the patients uniformly describe it my leg gives out or my leg just aches. It just is uncomfortable.

It's not really a pain sensation they describe to you. And the next is, where is this pain located? Is it in your shin? Is it in your calf?

Is it in your heel? Is it something else? And those things really make a difference in how we evaluate patients and how we distinguish it. Is it a vascular problem or is it something completely unrelated and they've just not been diagnosed for the underlying diseases?

How do they get relief? Do they just get better by standing? Do they always have to sit down? Or do they just keep going and things get better on their own?

Does the pain start somewhere else and radiate towards their legs or their toes? Or does it start on their toes and goes all the way up? Do they have any symptoms at night?

Do they have problems sleeping at night? Do their feet wake them off? Do they have cramping at night? Is this pain reproducible?

I walked three miles yesterday. I never any trouble. But today, I just was walking from your parking lot to your clinic and my leg is killing me.

So, reproducibility is very, very important when you're talking about vascular biology. Does the pain come in even if they're sitting like this like you guys have been sitting all morning? When we're talking about differential diagnosis for leg pain, the four most common things are peripheral arterial disease, which would be something that would most commonly be taken care of back to specialist, neurogenic leg pain, which is very easily confused with vascular etiology, venous claudication-- it's kind of a little bit of a misnomer because claudication typically is related to arterial side of the problem.

And Venous claudication is not very well recognized. We'll talk a little bit more about it in the later part of the talk. And obviously, degenerative joint disease, which is a very, very common problem, is either related to your spine, back, or hip, knee, or any kind of a joint you can imagine. Probably I would include fasciitis and foot problems in this category.

Peripheral arterial disease is basically defined as a flow limiting atherosclerotic stenosis or occlusion which will not allow you to get adequate perfusion to the distal-most part of your body. It can be asymptomatic. It could be lifestyle limiting or it could be limb threatening or we would call it critical limb ischemia. The key is, it is strongly related to other cerebral vascular disorders, be it stroke, be it coronary artery disease, be it renal [INAUDIBLE] disease.

So it's not a disease which occurs in isolation. It is a whole gamut of complex atherosclerotic problems that we see. Why is PAD so important and so close to us and why do we so rarely really pay so much attention to it? It's not just the fact that it is a marker for other disease processes. It is an expensive problem

We talk about this very commonly within our division and our department as nobody pays attention to how much money we spend on taking care of PAD. You talk about cancer care. You talk about colon cancer or breast cancer.

It receives so much public attention. And same with aneurysm disease, in fact, nowadays. But those things don't even have a fraction of the expenditure or cost to the society as compared to peripheral arterial disease.

And if you look at any published data, in fact, PAD will rank higher than coronary artery disease care or CHF in terms of the expenditure. And that does not include associated problems like I was talking about-- CAD or stroke that comes along with PAD. What are the risk factors for peripheral arterial disease?

If you go right towards the right side of the slide, that means that the higher-- there's a much higher association of that problem with the disease. If it is touching the line on the left side, that means it's [INAUDIBLE]. Now, being a male as compared to a female, it does have a one to two times risk of developing PAD. The reason you will see more of a close proximity of the left side of the line is primarily because there's an increasing prevalence of smoking in women.

But if you look at the far right side of the slide, the two biggest contributing factors are diabetes and smoking. So the odds ratio for somebody who is a diabetic or smoker is at least three to four times in any study population you see for the developing peripheral arterial disease as compared to people who are nonsmoker and non-diabetes. And it's not just the development of PAD. It also affects the severity, as we'll look at it in the next slide.

So, how do people present with PAD? There's people who will be asymptomatic, that they really don't have any problems. That usually doesn't get addressed because if they're asymptomatic, nobody would even diagnose it. If they don't complain of it, then nobody evaluates for it in most situations.

The problem is that the asymptomatic could be from either they have very developed collateral perfusion-- so if they have an occlusion stenosis of one of the main vessels, they have collateralized around it. But more commonly, like [INAUDIBLE] was talking this morning, they just don't walk enough to realize that they actually have problem. So they have either congestive heart failure or they have COPD. Their shortness of breath catches up before their legs will catch up with them.

Or they're just sedentary like a lot of patients we see. So they don't even realize they have symptoms from it. Claudication is typically defined as a reproducible pain in the muscular area of the leg after walking or after exertion.

So the reproducible part of it is very critical. If you have claudication, most of the times, reproducible does not mean that you have to walk 10 yards every single time. It is dependent on the incline, dependant on the weather, depending on what they were doing before but reproducibility means within a certain variation, if they have reproducible pain in the muscular compartments of the leg after exertion, that would be consistent with claudication. The other key part of it is always relieved by rest.

It does not carry on if you stop walking. And the relief time is variable depending on how severe the disease is. It could get better within two minutes, within five minutes. Again, it's a marker of the severity of the disease.

And by rest, we would mean just by standing. Patients do not have to sit down to get the relief from their pain. The biggest part of this or the most concerning segment is the critical limb ischemia segment in this patient population. It could be defined as rest pain, tissue loss, or ulcers.

Ulcers and tissue loss are basically in a way synonyms. But when we look at patients with arterial disease, ulcers and tissue loss can be very different. As I will explain to students a lot of times, if you have to keep your skin alive, you just need this much blood flow.

To walk, you need about this much blood flow. And to heal something, you need about this much blood flow. So even if you did not have ischemia to an extent to cause ulcers, you develop an ulcer for some other reason, you may not heal it because you have underlying ischemia. So a tissue loss because of underlying disease like diabetic entropathy or venous ulcers that may not heal from arterial disease as an underlying cause is very different from arterial ulcers. We can touch base on that.

So, a natural history of intermittent claudication is actually very, very, very favorable. And that's where I think I alluded to in the beginning, old is new. We knew this for a long time.

And that's the reason we are so conservative in taking care of patients with peripheral arterial disease especially, who are claudicants, even if are lifestyle-limiting claudicants, their national history in terms of any adverse outcomes as related to the limb is one of the safest things that we can follow in the entire gamut of things that we do is if you have asymptomatic coronary disease, they have a much higher risk for stroke as compared to limb loss in this population. 1% to 3% is definitely on the lower side of any risk factors that we evaluate patients for.

Most of these patients will actually remain stable for many years to come-- five years, 10 years. I have had patients-- I've not been in practice that long to have them five years. But I've had patients for three years that I know that have not advanced in terms of their severity of disease or symptoms even if they have not gotten any interventions done. There is a quarter of the patients who would progress to critical limb ischemia.

And that is a subset of population which is definitely a concerning population. Unfortunately, the patients who progress to critical limb ischemia are not because they were not adequately addressed or they were not intervened upon. It is because of some other risk factors. The highest rate of progression is early on. So once you see a patient who has lifestyle limiting claudication, we definitely want to follow them early on.

We want to follow them within six months, three to six months time period. And once we make sure that they are stable claudicant, then we could definitely make a follow-up a little bit less stringent and follow them every one to two years. Because if they don't progress quickly in the first time after they start their symptoms, they have a much better natural history. The other part why we are so conservative about these patients is it's not actually the limbs or the claudication which is going to really get them in trouble.

Any study-- and this spans over the last few decades and we have really not made any changes to this data. You follow patients who have claudication and peripheral arterial disease for 10 years. About half of them, give or take 10%, would be dead in 10 years. And most of them are not dead because of their legs.

They are dead from either their heart or they had a disabling stroke or they went on dialysis because of renal artery problems or there's something else which-- or cancer, actually, is the second or third most common cause of death in our patients. So it's basically-- the legs of the least of their problems. It's everything else that needs to be addressed in these patients.

So when I was talking about the quarter of the patients who progress to critical limb ischemia, the big risk factors for progression are diabetes and smoking just like I talked about. These are not the risk factors that cause the disease only. They're also very critical in ongoing progression of these patients.

Patients who basically are much severe presentation on initial presentation-- so AVIs less than 0.6 are much, much higher likelihood of progression as compared to the patients who are 0.8 or 0.9. Obviously, patients [INAUDIBLE] smoker or continue to mismanage their diabetes are going to be much higher risk. Now, I gave you a very favorable outlook on claudication or early symptoms of peripheral arterial disease. What happens if patient comes into this last bucket of critical limb ischemia.

Either they have rest pain or they have ulcers. That is a bad problem to have. That is, of all the problems you can imagine, as I think the mortality would approximate close to pancreatic cancer or esophageal cancer, which are on a scale of things, the highest mortality rates cancers that I know off.

So if you have-- now, the left side of the pie chart will tell you what would be done on these patients. And regardless of what is done, if you follow all these patients for a year out, this is the outcome that you see is the second pie chart on the right side-- about 40% limb loss at six months, about a 20% mortality at six months, and about a 30% or more mortality at one year as compared to a 40% mortality at 10 years, a 30% mortality one year, which is much more than CHF patients or advanced CHF patients like the [INAUDIBLE].

So this is a very, very bad problem to get into. The reason is, patients who get into critical limb ischemia, it is not just an isolated, local disease process. It is a marker of what we call Piss Poor Protoplasm, PPP. That's the common mnemonic we use.

So once you get to the critical limb ischemia stage, we have to be very aggressive in treatment. We are very conservative as long as you're not in this fourth category or third category, depending on which classification you use. But once you get to the critical limb ischemia, we really, really are very aggressive in trying to see what we can do to get them functional again.

How do we evaluate peripheral arterial disease? I think we touched mostly upon the history and the exam standpoint. We talked about patients who are either symptomatic, like we talk about the typical symptoms, or patients who have risk factors as listed here should be investigated with what you would call ankle break indices or non-invasive vascular studies. I'm sure all of you guys have seen what an ankle [INAUDIBLE] is.

Basically, it's a pressure differential between the leg and the upper extremity. Now, there is a few presumptions that come with this measurement. You have to make sure that the upper extremity pressure is normal.

It is not uncommon to have subclavian artery disease in patients who have lower extremity. So if your upper extremity pressure is artificially lower, you may still get an index of one. But that does not mean you have normal or extremity perfusion. The other one is this study is usually done at best. And resting study can miss significant disease in almost a third of the patients.

There have been studies done from a previous institution. If you do exercise studies on everybody, that you basically stress them after walking them two to three minutes on a treadmill, about a third of the patients will drop. The reason we don't do exercise studies on everybody is, one, it's time consuming. The second part is it does not change management in most patients.

Even if you find patients who are dropping their ABIs on exercise and they have risk factors, you're going to manage the risk factors. Now, if you clinically suspect somebody to have peripheral arterial disease and their resting study is normal, yes, that patient should get an exercise study. But the flip side does not always hold true. It is not cost effective to do exercise study on everybody because it's not going to change your management.

So beyond your first type of non-invasive vascular study, which is very basic-- it's blood pressure testing, can be done in the office. But most of us like to see just more than the pressure because pressures are unreliable in patients with diabetes or [INAUDIBLE] disease. But beyond the basic study of wave forms and blood pressure measurement, you really do not need to do any more investigations unless you plan to intervene on the patient.

Just because you can find a blockage based on CT or an arteriogram is not a reason to find one. If you're not going to fix there, if there is no indication to treat the patient, you don't need to put them through an invasive evaluation. So, the next few slides are going to be mostly a consensus statement that includes AHA, ACC, Society of Vascular Surgery, most of the societies which treat vascular disease.

This is one of the older classifications, I think, or the older recommendations set. The new recommendations set-- I think there has been a little bit of argument within some societies because Vascular Society wants to continue to be more conservative. They want to go by patient symptoms and functional outcomes.

And I think ACC has been little more towards an aggressive side. And they want to treat based on lesions and angiographic appearance. And so I don't think a consensus statement is going to come out at this time. It's probably going to be different statements that come out from different societies.

But I think this is a very, very good document to review. And this is mostly what we would go by as vascular specialists in our clinic when we see patients. So like any other category of levels of recommendation, level A is strong data, really something everybody should follow.

Level B is halfway reasonable data. I think the data can be better. But still, it's considered adequate quality data. Level C is somebody just made it up sitting in the committee. There's really not enough evidence.

This slide essentially tells the exact-- almost everything that you want to know about claudication or peripheral arterial disease treatment. So I'm hoping everybody can read this adequately. It should be in your handout. But again, on that little small slide, it's going to be very small to read. If you see a patient with peripheral arterial disease, the first part is risk factor modification.

We'll touch base on individual risk factors that are listed here in the next few slides. Once you have modified the risk factors, then you say, OK, do these patients have limitations that are affecting their lifestyle or their job or something related to that? If not, there's no reason to intervene, no reason to investigate further.

If they do have limitations, then you want to investigate further. You want to really see how bad the limitation is, and really how they're doing on that lifestyle modification before you start intervening on these patients. If they have a proximal lesion-- proximal would be considered above the groin. So iliac lesion, aortic lesion-- we tend to be a little bit more aggressive in intervening on those early on because conservative management does not have as much benefit. As compared to intervening on a lesion, conservative management does have better benefit.

And at the same time, aggressive intervention does not have very good long term data or long term outcome. We have plenty good data. The aggressive intervention does not have long good long term outcome. So if you have better outcome medically managing certain group of patients and not as good outcome surgically managing those patients, so it'll obviously makes sense to try and do medical management.

On the other hand, if you're above the groin, medical management does not do as well. Surgical management outcomes are much improved. So we tend to lean towards aggressive intervention. For [INAUDIBLE] disease, you talk about supervised exercise therapy for medical management, which is [INAUDIBLE], essentially the only drug we use nowadays. We used to have other medications.

And then based on symptom improvement, then you go on to the next step. So this is the big box on the top. Smoking cessation is probably the biggest part of lifestyle modification in our patient.

I don't know if I've ever see a patient from some of you people. But if you see a patient back from what I've seen for claudication, I just blatantly refuse to operate on them unless they stop smoking. My response to them is if it is so limiting for you, you need to change your lifestyle to change your symptoms before you want me to intervene on you. So if you have patients who are claudicants and they have lifestyle limiting claudication, they need to really change their lifestyle so the claudication can get better. Otherwise, I don't think we are doing a patient any service if they continue to smoke and we say, OK, yes, we can do an intervention.

We can fix your artery. We can put a stent in. We can do a bypass and, sure, you can walk better for six months.

Smoking is not just important in terms of peripheral arterial disease in terms of the progression like we talked about. It is definitely shown to be, in multiple studies that you can see-- there's six studies and I can't fit any more on this slide-- it improves survival, long term survival, not just related to claudication. It decreases the risk for major amputation.

It decreases the risk for failure of intervention and it decreases the need for revascularization. So all in all, it's a game winner if you consider smoking cessation for our patient population. Lipid lowering therapy, I think [INAUDIBLE] talked about this this morning.

It's has really been a big, big player in vascular disease over the last 10 years. When statins were introduced, we really did not think this is going to change our management of patients so drastically. We started with an LDL goal of 100.

Then, we started off with the LDL as normal. We want to put in statins. Then we started lowering the LDL to 70.

Now, we have come to a point where we say it doesn't matter what your LDL is. If you have a diagnosis peripheral arterial disease and you can tolerate statins-- and again, the toleration part is a little bit difficult because about 10% of patients would have significant [INAUDIBLE] with it. Statin is a must on all patients, regardless of their cholesterol level.

This just does not have plaque stabilizing effect or lipid loading effect. It has a lot of pleiotropic effects, we call them, which means a decrease of inflammation. Statins have been shown to decrease the risk of DVTs after the first event of DVT, it obviously has shown to decrease the risk for coronary artery disease. So this really has become a first line treatment for our patients. And I think in the next statement that comes out, this will all become a level A recommendation instead of the level B recommendation that you see on this current version.

So I think we talked about most of this. Statins have also been shown to improve patency both in endovascular and open surgical revascularization patients. So this was one of the large studies that evaluate the effect of similar statin on patients.

And the thin black line in the middle basically means no difference. And you can see every single outcome that's studied is on the left side of that thin black line, which shows that statins help with everything. As people will say, you just need to put them in water, basically.

We don't know how they work and some of the other factors apart from the lipid loading effects. But they have a lot of pleiotropic effects we just don't know of. Diabetes management is-- and we've been blessed that we have a very good endocrinology team who helps us with management in patients with diabetes.

But this is a very important part, again, reason being diabetic patients really do progress to critical limb ischemia from PAD very quickly. And diabetic management is very important, not just from PAD standpoint, but diabetes is very closely related to [INAUDIBLE] disease in these patients. And if you can manage their diabetes and you can avoid them getting into a CKD or end stage-- or dialysis problem, you decrease their limb loss significantly.

Hypertension management-- again, like any other atherosclerotic problem, hypertension management is very critical for them, both for PAD and for renal artery stenosis. Antiplatelet therapy-- we use aspirin extensively. We use Plavix very frequently.

We dabble into some of the newer antiplatelet agents on a regular basis. Most of the antiplatelet data does not really have a strong relation to peripheral arterial disease or lower extremity peripheral arterial disease. Most of the data comes from either stroke prevention or coronary arterial disease prevention.

So just because patients who have peripheral arterial disease have those two diseases is a good enough reason to put them on antiplatelet. But there is not adequate data to say that, yes, every PAD patient is going to help, as far as their legs are concerned, from antiplatelet therapy. Exercise therapy-- and this is another critical part of a claudication patients.

Most of the patients will ask me, can I walk? Is it going to hurt if I keep walking? Is it going to damage something?

To the contrary, the response is, it is actually going to help you if you keep walking. Regimented exercise for 30 to 40 minutes every day or at least three times a week has been shown to improve walking distance. And the principle behind that is people develop collateralization. I would explain to patients it's just like training for some kind of a big athletic event.

The more you train, the more stamina you build. If you keep walking, there is definitely collateral circulation that improves. If your main highway is clogged, the [INAUDIBLE] will open up. Every single study that has been done for regimented exercise program for claudication patients has shown about a 180% to 200% improvement in walking distance after three to six months of supervised exercise or compliant exercise program. The problem is, how do you get patients to be compliant?

So this is just the results of the study I was talking about. The one big thing you have to tell patients-- it is going to take at least three months, probably close to six months, before you realize a noticeable difference. If they're going to be in-patient, they're going to call you every two weeks.

I have not changed. I have not changed. It does not work.

But if they are willing to spend some time, and there are a fair number of patients, once I discuss this with them, who will say, you know, I really don't want to get an intervention which is going to fail in one or two years. I would rather just exercise and see how things go. And they just do fine.

Supervised exercise program definitely is much better than patient initiated exercise program. The biggest problem with that is, how do we pay for it? Insurances don't cover for it currently.

We're hoping at some point, when all this newer Affordable Care Act comes in, they will have some better coverage, just like we have for cardiac rehab now. And there are patients who are in cardiac rehab for some kind of a cabbage or some other disease process they've have had surgery for. And we just utilized the same program. And they tend to do better because they're on a supervised program as compared to a patient initiated program.

Pharmacological therapy, when I was a resident, we used to use pentoxifylline, or Trental, pretty extensively. Trental does not have as good of a data. It had a very modest benefit, maybe to 40%.

Pletal, or cilostazol, is definitely much better data in terms of benefit and the degree of benefit. I would tell patients that if you have infrainguinal disease, there is usually about a 60% improvement in walking distance with Pletal. Now, the problem is it does not help everybody.

And part of that data is because if you give Pletal to people who have [INAUDIBLE] disease or iliac disease, they usually do not tend to benefit as much. So that's what I was pointing to medical therapy is much, much better for infrainguinal disease or below the groins. And we tend to be more aggressive for above the groin disease.

You have to be careful. There is a black box warning for CHF in Pletal. You cannot give it to patients who have CHF.

There's no direct studies for Pletal and CHF. It's data that was extrapolated from related drugs. So I think if I have to see a patient who I think would really, really benefit, I would bite the bullet and give it even if they have CHF. You just have to be careful and explain to the patient that there are medical implications for that.

Revascularization-- you'll see study after study after study which will tell you revascularization does not offer any long term benefit as compared to all the other things you talked about for patients of claudication. They would feel better. It's almost synonymous to the study that came out for coronary artery stenting versus medical management for people who have stable angina.

There is adequate data to suggest that people who have stable angina, stenting does not improve survival. Stenting does have a little bit better functional outcome, but really not that much of a difference, considering the amount of money that is spent on it. But still, we have a lot of coronary interventions that are done for stable angina.

And the same thing applies to claudication. It has a lot of interventions that are done for claudication regardless of the fact that really, we do not have much better long term outcomes. Once we go down the path of interventions, we actually do have increased problems because once their intervention fails, they quickly get from a stable claudicant stage to a critical limb ischemia stage.

If we did not do an intervention and it never failed, they would have probably stayed in the claudicant stage for a long, long time. If you're going to intervene on somebody, you have to make sure they're well, well-managed medically. You've got to make sure they've done all the things you've talked about before.

You have to make sure they understand that it's not going to be a fix for life, especially on the younger side. It is going to fail within their lifetime. Risk for failure depends on what the intervention is, how severe the disease is. But they have to be counselled beforehand rather than just saying, OK, we're going to fix it and you'll be just back to normal like you started at age 16.

Surgical treatment versus endovascular treatment. There was a large trial in the UK again, a lot of controversy any time a randomized trial comes out, a large trial [INAUDIBLE] which came out about two years ago from UK which randomized people to endovascular versus surgical management. It did not show much difference between surgical and endovascular treatment.

Where's the next slide? But there is definitely a significant benefit for open surgical treatment versus endovascular treatment if you're looking at long term limb salvage. So for claudication or for walking pain, endovascular and surgery are all the same.

But if you have critical limb ischemia, surgery definitely tends to do better. Surgery definitely tends to do better with less risk for reintervention as compared to endovascular intervention. Here's just a couple of cases. This is an example of iliac artery disease, significant disease that you notice on the right [INAUDIBLE] artery.

Easy to read with stents, would be considered a task A lesion in most of the classifications. That is the classification we go by, task A to D. B is a very severe disease. Usually, we will say better off doing an open intervention versus an endovascular intervention.

But this would be a good patient if they have lifestyle-limiting disease despite modification of the risk factors to treat endovascular. Bottom slide obviously is a little bit more invasive, just maybe about 20% more invasive as compared to the top slide. But that's a distal bypass.

We call it a far away bypass. So it's to the level of the knee-- to the level of the ankle to the posterior tibial artery. These bypasses actually tend to do very well if you're doing them for a limb salvage situation. Their patency is excellent.

And again, you have to be willing to put the time in. These are not straightforward, short cases. These are about a six hour to eight hour case if you're going to do them right.

We talked about a lot of leg pain claudication to question. I just wanted to bring this to everybody's attention. You know, we always say that if you have vascular disease, you have leg pain or vice versa. If you have leg pain, you have vascular disease.

But there are patients that we will see who have no pain but definitely have a lot of vascular disease that needs to be addressed early on and taken care of. So this is not an uncommon patient in our clinic setting. Between the six of us, I would say we probably see at least two or three of these every week in outpatient and probably another one or two that could transfer to the hospital as an in-patient.

This would be a typical diabetic neuropathic ulcer or either diabetic or some other kind of neuropathy. And patients don't complain of pain in these feet surprisingly. You look at it, it hurts you. But the patients don't have much pain because they have so much sensory loss, they don't complain of any problems.

And a lot of these patients may not complain of claudication. They may not complain of ischemic rest pain because they have not walked enough to have claudication. And again, like I was telling you in the beginning, to heal this ulcer, they need a lot more blood flow than just to walk and hurt.

So these are the patients we have to really to be aggressive with. And I personally have a philosophy that if you see a diabetic foot ulcer, you need to treat it like an emergency, like a symptomatic aneurysm, not something you need just say, OK, yeah, let's try this. Let's try wound care for six months.

Let's try what happens. I think once they get to this stage, regardless of how much blood you can get to them, limb salvage becomes very, very challenging because they develop so much capillary thrombosis. So on the outside, all you see is this ulcer and some [INAUDIBLE].

On the inside, this has extended at least to the heel of this patient. I can guarantee you. So this is very, very crucial.

If you're going to see a patient who has diabetes and have any ulcer on the foot, even if it is early on, you absolutely have to make sure their perfusion is OK and get them help quickly instead of saying, OK, we'll put you in the wound care center. We'll try this. We'll try this, try this for a few weeks, and then see what the response is.

Yes, there is a fair number of patients who would not have any vascular etiology. But even if they don't, they're better off getting that ruled out as compared to two or three patients if they're missed over a month who would end up with an amputation just because they did not get caught early on. As compared to the diabetic foot ulcer, I think I alluded to this early on.

Patients who have arterial insufficiency ulcers would have painful ulcers. And their ulcers would be usually at the very distal part of their foot at the tip of the toe because that's farthest from the heart. So arterial ulcers are very uncommon as compared to ulcers or tissue loss that does not heal because of underlying arterial insufficiency.

And that's a very, very difficult distinction to make. Even our fellows would not think about it. All ulcers with arterial disease are the same.

That's not the case. I think all ulcers with arterial disease are not the same, the reason being if you have a diabetic foot ulcer and they don't have ischemic rest pain, you provide them with perfusion. Even if you provide them with perfusion for three months or six months, they heal their ulcer, they're fine. As can be to a patient who has an arterial ulcer, you have to do something which is going to be durable. Because as soon as you do something which is not durable, that thing fails in three months in six months, they are going to have the ulcer right over again.

But if you have a diabetic ulcer, you are able to heal their ulcer and you provide them excellent foot care, they may not have an ulcer recurrence. So even a limited procedure which is going to help them heal may be of benefit rather than doing a big procedure, which is going to be obviously with a lot of risk. Ulcer treatment, just basics.

We're going to switch gears. I'm looking at the clock here. I think we're OK.

So we talk about lower extremity edema, which is the other part of our big vascular practice. I think-- we talked about aneurysm or disease last year. So I did not want to kind of repeat the same topic.

This is a patient actually I just saw yesterday. So I updated some of the slides to give you a fresh patient scenario. So this is a young female who came to our clinic yesterday. She actually did not come primarily for swelling and pain.

She has actually gotten used to living with it. She thinks that's just a part of her life. She came in because somebody prescribed her a hormone replacement therapy.

And on the box, it said, risk for deep vein thrombosis. And she has DVT in the past. She wanted to talk to me and see if she should be on hormone replacement therapy or not.

But you talk to her, she has a history of DVT after her second childbirth 20 years before. And she has had swelling in her leg. and pain after prolonged periods of standing or even sitting for those 20 years. And it's progressively getting worse and nobody has ever told her anything. She just thinks this is part and parcel of her life now.

So she does not do any kind of compression therapy. She takes an aspirin. And she's morbidly obese. So the next step in this patient's care, if you talk to people outside, any of these options will be considered. Yes, sure, we can do that.

Again, like the arterial patient, before you jump into any of these things, you really need to know what is wrong with the patient. And this patient, I'm going to end up-- present at the very end and see what would the people in the audience do about this patient. When we talk about [INAUDIBLE] edema, we talk about systemic causes.

We talk about local causes. Systemic causes are something which are nothing to do with the vascular system but everything to do with everything else. Systemic causes, three main systems-- cardiogenic, liver-related, or nephrogenic. So if you have fluid overload for any reason or if you have right heart failure or if you have liver failure, if you have hyper [INAUDIBLE], you are going to have edema.

The key factor to distinguish this is almost always going to be bilateral. It's almost always going to be symmetric. It's usually defendant.

It is much better after the patient is laying in bed at night. And most patients, you can get a history of their underlying disease. Either they have some kind of cardiac problem.

They may not have told anybody. They may not carry a diagnosis. But if you question them directly, they will tell you that they have it.

This is almost always pitting edema because it is venous edema in a way. It's related to venous hypertension. The treatment is compression, leg elevation, and obviously treatment of the underlying cause.

That's not something that you go and chase veins about or fix the veins to help with this edema. And this brings into some local or systemic or local causes of edema. And as you see, there's five major categories. And there's definitely overlap. It's not uncommon to have patients who would have lipedema, lymphedema, or just plain, simple, old obesity all combine and put together in the same patient.

So how do we distinguish between these different kinds of edema? I think, as I say, a picture is worth a thousand words. This is probably the much better way of me explaining to you what these things are. So lipedema is usually symmetric, usually on the lower extremities, very rarely on the upper extremities. As we can see on the far left, this patient has a very slim torso but a very significant fat distribution in the upper thighs.

That would be considered a stage 1 lipedema. It is abnormal deposition of fat cells. We really don't know why it happens as of now. We really don't have a good treatment for it.

But we just treat her like a regular edema, which is wrong. But again, for lack of a better option, that's what do you do. Weight loss is not really a very good option for these patients.

Some people would say liposuction is a good treatment for them, especially lymph vessel spitting liposuction. We don't do those. I really don't know if there is any expert in this area who does those. But this is a difficult patient problem to deal with.

Fortunately, we don't see very many of these patients who have isolated lipedema. Most of these patients have something else going on and not just lipedema. This is lymphedema.

This is actually not a very extreme case of lymphedema. You'll see some less extreme cases of lymphedema. I have seen plenty of patients who present like this. So, lymphedema is basically related to obstruction of lymphatic flow. And as you all know, lymphatic flow contributes to about 10% of your residue of fluid drainage from whatever the veins don't pick up. And some patients, if they have venous problem and they have lymphatic problems superimposed on that, then the lymphatics obviously have to play a greater role.

The key factor in lymphedema is if you can notice this box-like appearance of any part of the body you see. It's almost like a rectangular box, just a squaring, if you call it-- a squaring of the leg, squaring of the toe. Lymphedema will almost always affect the forefoot and the toes.

As compared to most of the edema that we evaluate, it does not affect the forefoot and the toes. If you look at this patient's leg, there is really no box-like appearance and the ankles are slim. So that's a very critical distinction point between lymphedema and lipedema.

Lymphedema can be for many reasons. It could be pathologic. People have cancer.

They have lymph vessel obstruction from that. People have parasitic disease, fortunately not very common in this part of the world. But mostly in Africa, they have filariasis or some muscular bond parasitic diseases which would obstruct their lymph vessels. And then people would have what we call lymphedema [INAUDIBLE] or congenital lymphedema.

That's usually unilateral. Again, the treatment for this is manual massage compression and a lot of patient education. Surgical treatment for lymphedema is really, really very difficult, not very good long term outcomes. Patients' leg on the left side is sometimes, you have to do decompression surgery for them.

But we say the operation is worse than the disease sometimes. It takes forever to heal these sounds-- forever. So we would just leave them alone, leave them alone.

They will keep crying, keep saying we need something done. You try and do everything else and then do surgery, but it is something you have to be very careful about. You make an incision on that leg, it is not going to heal quickly.

This is typical venous edema. So not all patients present with a middle picture. Middle picture is obviously what we'll consider very advanced venous disease.

You see we have hyper pigmentation not only in the ankle area called the gaiter area, but also extending all the way up to the knee. Gaiter obviously comes from the fishing. I don't fish, but that's what I've been explained that's where it comes from.

So on the left side, you can see some varicosities on the patient. Now, if this patient was obese, you cannot always see the varicosities. But you can always get an idea of this patient's venous edema. Venous edema is always, always splitting.

It is not uncommon to have one leg worse than the other leg. And if it is going to be prolonged, it is not uncommon to have hyper pigmentation. In very advanced disease, people would have ulcers on their legs. And this is just pre-ulcer stage. This would be considered lipodermatosclerosis, which is thickening hyper-pigmented skin that happens because of capillary leaks and macrophage inflammation.

Treatment options for various kinds of edema, as I said, every disease is treated a little bit different. Compression is almost always the mainstay. Lifestyle modification, leg elevation, patients-- a lot of patients we see as basically sitting on the desk or standing. A lot of patients I see are males who are contractors who are standing on concrete all day.

A lot of them actually are landscapers. You name it and I've seen the occupation that requires long period of standing. I was telling my fellow my legs have started to have some spider veins just noted last couple of months.

So that's just the kind of occupation you're in. If you're going to be on a long period of standing, you are going to be at risk. So, try to avoid long standing. So what I'm doing is if I'm doing surgery, I will just basically sit down and operate.

Actually, I'm kidding. I don't do that. But whatever you can do-- I mean, obviously, there are limitations. You cannot just walk on your head.

You cannot expect patients to quit their job. But whatever you can do to change their lifestyle, [INAUDIBLE] with their legs in between-- if they're sitting, try and put their legs on stool-- are very, very critical steps. It really makes a big difference in the long run.

Skin care is very critical, especially people with lymphedema and people with venous disease. They tend to develop cellulitis or ulcers, so skin care is very critical in these patients. Weight loss-- a lot of patients with any of these diseases have obesity and weight loss exacerbates all these [INAUDIBLE] problems.

It affects venous disease very much so as much as lipedema as much as so. So it's very, very crucial that you educate them. It's a sensitive topic to talk to the patient about, but if you do the hard conversation, they are very appreciative of that.

And I've never had a patient except for one who got mad at me because I told her that she needs to lose weight. But they're very appreciative once you do the conversation. It's still hard but somebody has to do it.

And you have to just tell them, you're not their friend. You're their doctor. And most of the times, they'll listen to you.

Surgical treatment is based on how bad their disease is, what is the underlying pathology, and we have various options available. These are not the kind of patients I see. But every time you look at a venous poster, this is what you see.

And I am not sure which of these women actually have had a vein operation. My guess is none of them ever needed one. But again, what you see on the boards are not always what you get.

So venous reflux is probably the most common pathology that we treat with an intervention. There's multiple treatment modalities available. Vein stripping is a good old fashioned surgery. I tell the patients-- I call it the butcher operation.

We try not to do it anymore. We occasionally have patients who just don't have any other option because they have had other treatments before. But you basically put a catheter in-- I think I have a picture here. You put basically a probe from one side of the vein, take it out the other side of the vein, and you just pull the vein out.

This is a patient that was on our service not long ago who had severe superficial thrombophlebitis. And this is his native [INAUDIBLE] vein. As you can see, this is the normal size of the vein. This is about six or eight times larger because it had acute thrombus in it.

This is very, very painful. It is probably one of the most painful problems people have, even more than arterial disease. So if you can treat these patients, most of the times, you're trying to [INAUDIBLE] management with heart compresses, [INAUDIBLE], sometimes anti-coagulation. They tend to do pretty good with it.

But in this patient, obviously that was not an option. This is not going to get better with all those things we tried. So he needed a vein excision. But vein stripping for just pure reflux not without phlebitis, essentially the same principle.

Again you don't get this kind of ugly looking vein. You get a vein looking like this, but same principle. Then you can ablate the perforators. You can ablate the saphenous vein. You can do phlebectomies.

This is how ablation has done. There's two main modalities-- radio frequency and laser. This is laser ablation. You basically just access the vein percutaneously.

You put a laser fiber. The way it works is it induces heat-induced damage in the endothelium. And because of that, the vein will close off.

Not uncommon to have some phlebitis because of the heat-induced damage because you're doing what phlebitis will do to your vein. This causes inflammation. But it's usually much better than what phlebitis will do on its own.

Long term results are not bad, but there is about a good 12%, 20% failure rate at about two years. So again, not something you want to do for everybody. It's a much less invasive procedure.

This is phlebectomy. This is one of the patients I just did recently. This is a post-op [INAUDIBLE] appearance. Here there's a small saphenous reflux. I'm a little bit more conservative about ablating the small saphenous just because the sural nerve which runs right next to the small saphenous is much more disabling if you have any paresthesias as compared to the saphenous nerve, which is much better tolerated. So we did a saphenous vein stripping on him.

This is his incision from the saphenous vein stripping. And these are the incisions from phlebectomies. So you can see they heal-- this is only two weeks after surgery and they heal beautifully. patients really get better.

And he's a veteran. He was not complaining regardless of how many years. And here, the vein is about a centimeter and a half wide.

And he was so happy after he came back to see me in clinic. He was like, I wish I had got this done before. Now, before you start doing venous reflux procedure, there are some patients you absolutely, absolutely have to think twice or more than twice before you put them through a procedure.

Unfortunately, it's not uncommon that I've seen patients who have already had their vein ablated without realizing that they have something more than just venous reflux. Venous reflux can be primary. That is just valve abnormality without any underlying problem.

Venous reflux could be secondary when patients have underlying obstruction and their saphenous vein or the superficial veins actually becomes a very good outflow mechanism. And that is what they're dependent on. And that refluxing vein is not what you want to treat because that is their lifeline.

You treat that refluxing vein, you have basically burnt out their outflow and they're going to have horrible edema. They will hate you for the rest of their life. So patients have venous claudication symptoms, which is patients complain of bursting pain after a significant exertion.

It's not different than patients complaining of arterial claudication. But this is more like a constricting or circumferential pain that would affect both the thigh and the lower leg, mostly the thigh as compared with the lower leg as compared with the arterial, they are much more distal first and then progress to the thigh or the buttocks. So if patients have venous claudication symptoms, you always have to be careful.

Do they have an underlying obstruction? Patients who have had a DVT in the past, very, very, very uncommon that we would treat their superficial system even if they have significant reflux. Because if their duplex shows that their DVT is resolved, it is more or less just an insensitivity of the duplex because most patients would have residual [INAUDIBLE] disease after a DVT.

If patients can recall a specific event after which their veins popped up, that usually means that something is wrong. So you do not really-- varicose veins or venous reflux happens over a period of years and patients really don't happen to notice a specific inciting event. If you have limb length discrepancy or port-wine stains-- looks like this.

So the left leg is much bigger than the right leg. It's longer, it's bigger. We call it limb hypertrophy, limb length discrepancy. And these port-wine stains, which usually start in the buttock area and kind of progress down, they could vary in the amount of pigmentation from much lighter than this to much darker than this and usually are presented both.

Used to be called port-wine stains. We call it venous malformation stain, name it strawberry stains. You have seen all kinds of names for these.

But this is a hereditary disorder when people have venous malformations, very high risk for [INAUDIBLE] agenesis or [INAUDIBLE] problems from birth, you do not want to treat these superficial veins. The other reason is they have a very, very high risk for deep vein thrombosis. So if you're going to do anything to them, you have to be very aggressive with [INAUDIBLE] or prophylaxis because they do have a much higher risk for DVTs as compared to normal population perioperatively.

So those are the things you have to be very careful about when treating patients. When you're dealing with surgical with venous obstruction, again, like anything else, we do endovascular. We do venous bypasses. We try and be very creative sometimes and do a combination of both.

Not a lot of patients need that. This is kind of a patient of mine who had an iliac vein obstruction. So you can notice if you're looking at the patient face on, this is actually not that rare. This is the inferior vena cava.

And you inject contrast on the left side. All this contrast is going from collateral circulation. And none of this is the main external or common iliac vein. All of that is obstructed and they're trying to flow through this.

And we stented this patient with a long stent all the way from the IVC and this all the collateral disappear because that becomes an preferential perfusion. And the leg seems to get significantly better overnight. And a patient who had-- this was a young man who had a foot replaced after a DVT for some reason.

And his fleet were occluded and he had inclusion of his entire IVC. As you can see here, both his iliac veins external and internal and he has these large collaterals. We stented him.

This is the most number of stents I put in one patient in one day. I think we used 13 stents in this patient. And this is what his neo [INAUDIBLE] iliac system looks like. This guy lost 20 pounds in a week and he came to me and is like, thank you so much.

I could see my ankles for the first time in the last 15 years. So there are patients who go unnoticed with these kind of problems for many, many, many years, are miserable. The problem is, they're usually young.

They're not like my arterial patients. They're easy to take it off, even if they're non-compliant. They're hard to deal with. They're just pain in the butt literally.

You just say, OK, I got to take care of them, but three or four years, he'll probably be dead. That's the natural history of the disease. So it's not something that you have to put them through over and over and over again.

This population of venous disease, obstructive venous disease, is our workforce. These are 30, 35, 40 year old patients who are working moms or working women, working men. This guy used to travel for work and for business all the time. So they are patients who are very miserable and they really, really benefit from aggressive evaluation and treatment sometimes.

Some open interventions. This patient has basically right iliac obstruction. You just use the saphenous vein, flip it on to the other side, connect it, and that becomes your outflow from one leg to the other leg called the Palma procedure.

If you don't have a good saphenous vein, you can use an artificial bypass graft shown here. And it does not really work as well. Bypasses in the venous system overall don't work very well because of the low flow, low pressure state.

And we tend to give patients on anti-coagulation. But the prosthetic Palma definitely is not as good as the good vein Palma procedure. I just wanted to bring to your attention.

This here is a old-fashioned IVC occlusion device called a [INAUDIBLE] clip. Well, I just saw a patient about two months ago with this. We see about maybe one patient every two or three years who has had this. This is before the advent of the inferior vena cava filter.

This is another patient who had an IVC filter. Filter lead to occlusion of the entire iliac system and needed a bypass because the left leg was very symptomatic-- multiple ulcers. [INAUDIBLE] a bypass from the femoral being all the way to the inferior vena cava. Got a [INAUDIBLE] to keep this bypass open just until the bypass endothelializes.

This is one of the really, really problematic filters. In fact, I was just talking to a resident. We have a patient in the hospital right now who got cancer [INAUDIBLE] who got a trapeze filter last month. And his entire [INAUDIBLE] system is occluded now because the filter caused thrombosis of that.

So I don't think I'm going to go into filter talk today, but those are really problematic things. So for this lady, we talked about-- if you want to read the question and just tell me, she has reflux in her right sathanas vein and she has symptoms which are very problematic for her. First step would be obviously to do lifestyle changes.

I'm not going to give you that choice. I'm hoping that everybody would have picked that. You would ask her to lose weight. Absolutely, absolutely she has to get to at least a halfway reasonable overweight situation if not less than 25 BMI. And she has to wear compression stockings.

And then after you have done all that, what will you do for this patient? So, anybody would do one? Thank God, I have achieved my best way, I think.

Anybody would say we should do an open venous bypass for this patient? Good. Anybody would want to put a stent in this patient?

OK, anybody want to put an IVC filter in this patient? Excellent. So the fourth would be malpractice in my view. But unfortunately, I've had the displeasure of seeing a lot of patients who have got an IVC filter placed for problems like this.

It is a nightmare. They don't get treated with anti-coagulation properly. Filter occludes and then it is a lot more problematic than what you started with.

Before, it was one leg. Now, you've got two legs and the IVC, which is problematic. So if I was taking care of this patient after this patient had done all their lifestyle changes, I would evaluate them, if they've lost weight.

And if they still are symptomatic, which I do suspect he will be, with the fact that he has very large [INAUDIBLE] varicosities, these are varicose veins which you notice the super pubic region. And these are literally this big. The woman would not want to take her pants down because she was so scared that you're going to see those veins.

Nobody has noted this. Nobody has told her what this is. This is basically collateral perfusion that her right iliac obstruction is trying to pass through just like we saw on the venogram.

So after she has done everything and she's a compliant patient, I would strongly consider iliac vein stent in this patient. That would be my first option. Now, if this patient does not have option for an iliac vein stent, she feels it, then an open venous bypass, yes, would be an

Option but, again she has to absolutely lose weight because you put an open venous bypass and a stent in an obese patient, you are just dooming them for a failure. So with that, I'll close. I'm happy to take questions. I'm sorry I did not show you any sexy pictures or devices. We see a new device come out in market every three months on average [INAUDIBLE] I am probably between some of the [INAUDIBLE]