BroadcastMed | Update in the Treatment of Chronic Pancreatitis

ANDREAS: Good afternoon, and thank you for joining us today. I am Andreas Gelrud. I am the director of the Pancreatic Disease Center and Islet Transplant Program here at the University of Chicago. I'm also the Director of Therapeutic Endoscopy at the Center of Endoscopic Research and Therapeutics. Today I am going to be talking about an update on the treatment of chronic pancreatitis We're going to start with some definitions. And then we're going to go into some clinical cases to better illustrate some of the work that is being done, and that we're doing here in our institution. So to start, let's make sure that we're talking all about the same condition. One of the problems that we have in the pancreas world is that all of the different types of pancreatitis that we have are called pancreatitis. But we have to distinguish acute, versus recurrent acute, versus chronic pancreatitis, to make sure that we're talking about the same medical condition. Each of them has a completely different clinical implication. And each of them may run a different course. Today we're going to talk exclusively about chronic pancreatitis. But I want to define acute, recurrent acute, and chronic. So for those of you follow the medical literature, particularly in the pancreas, I want to remind you about this paper in the "American Journal of Gastro-" from September, 2013, where the new guidelines for acute pancreatitis were published. Remember that acute pancreatitis is defined by a patient that comes in meeting two out of the three criteria. The first one is a patient having the typical pain, which is severe pain in the epigastric area frequently triggered by food, frequently triggered by fat and dissipation, actually, that's coming in with bad pain, usually 10 over 10 in intensity, radiates to the back in a band-like manner-- very bad pain. The patient is coming in because he or she cannot handle any more of the pain. So that's number one, severe pain. Number two, it's an amylase pain or lipase elevation of three times the upper limit of normal. It has to be three times upper limit of normal because there's many other clinical conditions that will give you an elevation of amylase pain or lipase, but just a little bit lower. So that would be number two. And number three is radiological evidence of pancreatic inflammation that we don't always need to get it. But frequently we do, and particularly by the time that we're called from the emergency department. So again, two out of the three makes a diagnosis of acute pancreatitis. By definition, acute pancreatitis is one episode and never again. When you have a patient that comes back with a second episode of documented acute pancreatitis, now we're talking about a patient with recurrent acute. Again acute is one. Recurrent acute, it's two or more. In between the episodes this patient is completely symptom-free. And then we have chronic pancreatitis patients. Chronic pancreatitis is a patient that has damage in the gland. Chronic pancreatitis is a patient that may be coming in with exocrine and/or endocrine insufficiency. But by far the reason why we see these patients the most is because of constant pain that frequently can be very debilitating. So again, today we're going to be focusing our attention on chronic pancreatitis. So what are the etiologies that we see for these? I personally like to follow the TIGAR-O classification because it's a very easy and simple way to remember and to scan all of the most likely and frequently seen etiologies that may lead to chronic pancreatitis. So T stands for Toxic Metabolic. Here we're talking about alcohol. We're talking about alcohol abuse-- long-term alcohol use. Tobacco-- a patient that is smoking frequently for many years-- hyperlipidemia-- hypercalcemia-- some medications-- extremely important to ask-- toxins, which are rare, but we sometimes see them. I-- for Idiopathic. There's always a piece of the pie that is idiopathic. If you look at the old books, you're going to see that this pie was very big, and as I'm about to show you, this piece of the pie actually, it's shrinking as we are learning more and more about different genetic conditions that lead to chronic pancreatitis. G-- it's for Genetic Factors. We have learned a lot about hereditary pancreatitis-- the PRSS1 gene mutation-- cystic fibrosis mutation-- over 1,500 mutations. Some of which may lead not only to the classic form of chronic, of cystic fibrosis, but it can also lead to what is called single organ disease, and what we see frequently in our clinic, expressions of single organ disease in the form of recurrent acute or chronic pancreatitis. We have SPINK1, which we call it more a modifier, expressing in approximately 2% of the general population, depending where you read. And we frequently see these big mutations together with another one in patients that have full blown disease. Others including Alpha 1-antitrypsin-- CPRC mutations. And of course, we have to mention others, because I'm sure there's more mutations that are to come as we're learning more and more about genes. Then we're going to the A from TIGAR-O which is Autoimmune Conditions. Autoimmune pancreatitis rarely leads to chronic pancreatitis, but it's very important to keep it in mind. I think it's a new kid in the block. We're learning more and more about it. We're checking more, and the more that we check, the more that we see. We treat it with steroids. A subset of patients are going to have elevations in ITG4. Some of them, actually the majority, don't. So it may be a difficult condition to diagnose, but I think we're doing a very good job in making the early diagnoses and preventing sending some of these patients to the OR like we used to in the past. Other conditions like Sjogren's, rheumatoid arthritis, PSC, and of course, other conditions that may lead to a chronic pancreatitis, but rare. We have R for Recurring, or severe acute pancreatitis. These are patients that have recurrent acute as defined before that after so many hits the pancreas, to put it in simple words, the pancreas may give up and lead to an organ disease. Again, frequently we see this with underlying genetic mutations. If there's a treatable cause, we should be treating these and, of course, preventing the recurrent attacks. But in some genetic conditions we tend to see this progression of starting with recurrent acute and then leading to chronic pancreatitis. Severe acute pancreatitis-- we don't always see it-- but we always have this subset of patients that come to the hospital very late, very sick, and we get an image of the pancreas by CT or other modes, and we see that the pancreas, it's basically gone out of how severe this necrotizing pancreatitis was. And sometimes these patients end up requiring insulin and pancreatic enzyme supplementation for the rest of their lives. Obstructive-- very important-- post-traumatic-- duct obstruction-- we're talking about tumors, and here we're talking about slow growing tumors. We're talking also about adenocarcinoma that may lead, actually, to exocrine insufficiency, pancreatitis cancers, IPMN, and rarely pancreas divisum. There's a lot of papers out there, including some from our group, showing that rarely pancreas divisum on its own will lead to chronic pancreatitis. And we think that a subset of these patients with divisum have truly an underlying genetic mutation, and that what led to the chronic pancreatitis is not that the divisum, per se, but we think it's an anatomical variant. But what led to the chronic pancreatitis was the underlying genetic mutation. There's a lot of people with different thoughts about this. But in general this is how we approach these patients. This is a slide that I am borrowing from Dr. Wickham where we showed that multiple etiologies lead to chronic pancreatitis. And as we were saying before, a very large piece of this pie of idiopathic-- it's shrinking and shrinking more as we are learning more about different genetic mutations that lead to pancreatitis. So we now know, and we're able to recognize that mutations may lead to the development of recurrent acute and chronic pancreatitis. So how do we make the diagnosis of chronic pancreatitis? As all of you know, it's very easy when a patient comes with a full-blown disease in a pancreas that looks very abnormal with multiple classifications, a CT scan that you can see ductal dilation, and [? so. ?] But in reality the ones that we see in our clinic can be very difficult to make the diagnosis, particularly when the gland looks relatively normal. Extremely important to get a very good clinical history. Does the patient have any major risk factors? How long have they been having symptoms? What age did they start at? When was the first episode of pancreatitis diagnosed? How has been the progression of their disease? Very important also to ask about a family history. Anybody in the family with a history of pancreatitis? Recurrent pancreatitis? Chronic pancreatitis? Anybody in the family dying from pancreatic cancer? Extremely important-- because having chronic pancreatitis and smoking, for example, may lead to pancreatic cancer. Some mutations may predispose the patient also to develop pancreatitic cancer. Very, very important to get a good family history. Is there anybody in the family with a history of cystic fibrosis? Extremely important, including male infertility. Anyhow, how do we make the diagnosis? Good clinical history. Does the patient have any evidence of exocrine or endocrine insufficiency? These are things that we must ask. Again, obtaining good quality imaging nowadays, so we're able to pick up very subtle changes-- very important. Frequently we get patients coming to our clinical already with a good quality pancreatic protocol CT scan that shows their disease. MRI, it's being used more, and more, and more, to make the early diagnosis. And here we're talking about patients that don't even have calcifications or strictures, or a very dilated duct. MRI, with gadolinium-- very good tool. We can always do an MRCP also to get a very good anatomical view of the duct as well as the biliary system. We do a lot of endoscopic ultrasound, particularly when you have somebody with a lot of expertise and a lot of training-- somebody that is doing a lot of these USs, and can really give you a good sense of how good or how bad the pancreas looks. And occasionally abdominal x-rays that you can see calcifications. And, believe it or not, sometimes these patients come to our clinic with an x-ray that show calcifications, and they're being referred to us for further work up. Then we have test of functions that we do in a small subset of patients when we're not really sure if this is what the patient has or not. And this is what it is called-- the secretin stimulation test-- that we do the [? Grayling ?] tube, or you can do the endoscopic test as well. You see these done in centers like ours. Centers that have pancreatic disease centers of excellence.

And in occasion we're getting genetic testing. Please keep in mind that if you're going to be doing genetic testing, you want to have a genetic counselor with you. You want to make sure that they see a genetic counselor before you start to get this information, so the patients can understand better what the implications are of checking genes, particularly when one genetic mutation may come back positive. So this is always very good to obtain before we get the blood samples, before we do the genetic testing. Remember that nowadays we can tell the patient this is why you have the disease, but unfortunately we don't have genetic therapies, at least in the year 2014. I'm sure soon, or in the next couple of years, we will. But you have to be careful when you're checking tests that we don't have treatments for, and remember to get your genetic counselor involved-- very good idea. This is a good example of a patient where you see the arrow in the middle of the abdomen, of a x-ray, of a patient that had a calcification. This patient actually had malabsorption, was losing weight, and had some nonspecific abdominal pain. Long story short, this patient ended up having a chronic calcific pancreatitis that was picked up with this KUB. This is a different patient that you can see severe calcific disease, particularly in the head of the pancreas. You can see the calcifications with proximal dilations in this CT scan. This other image, again, you see some calcifications in the head of the pancreas, and again, it's different modalities showing the same calcifications, biliary ductal dilations, and so, that will help us make the diagnosis. So once the diagnosis is made, how do we treat these patients? And this is the challenging part. Number one, if your patient is smoking, or drinking, you have to convince them to stop, and as I always tell the patients, it's very easy to tell somebody to lose weight. It's very easy to tell somebody, "Stop smoking. Stop drinking," particularly when they have been doing this for many, many years. So I don't know how, but we have to convince the patients to stop smoking and drinking if that's the case. We need a very motivated patient, and you need a patient that really wants to change and to try, actually, to get better. Anyhow, stop smoking, stop drinking, extremely important. Pain medications, we always start with NSAIDs. We try to avoid the use of narcotics. If you're going to be using them, try using long-acting narcotics and not the short-acting. You want to avoid those peaks and valleys. You want to give more like a longstanding pain relief so the patient to be back functional. Very, very careful with addiction. Be also very careful with the patients creating resistance to the medications, and we're just increasing, increasing the dosage. And we may be creating a nightmare for these patients. So be careful. Get always your pain service involved, or if you don't have pain service, refer your patient to a pancreatic disease center that we actually can approach these patients in a multidisciplinary way. And hopefully try to prevent these complicated problems, particularly addiction from narcotic use. Antioxidants, we have positive studies. We have negative studies. Some patients we start them on them, and some others we don't. If you want to use it, I think nobody will blame you. They are good, antioxidants actually, are good anti-inflammatories. So we think that this may decrease the pancreatic inflammation. If they don't work, they won't do any harm, and that's what I tend to tell my patients. Nutrition, extremely important to be well hydrated. The pancreas likes to be surrounded by water. Hydration alone will decrease somehow the amount of pain. I have no data to show you what I just said. But this is an observation that I have had. And I think many and pancreatologists will agree with that-- this comment. If a patient has a lot of pain, low fat diet-- very important. Fat is a stronger stimuli, so we try to keep their patients in low fat. I try to tell the patients not to go too crazy with no fat. There always has to be a little bit. But what we do also-- it's to get our nutritionist, our dietitians, involved so they can see our patients. And we try to have the patients see us in the office, and the same day see our nutritionist so they don't have to be coming and going. Endoscopic therapy, it's something that we're doing more and more. Or I should say, less and less, depending on the patient, depending on the age, depending on the comorbid conditions, depending on how good or how bad the pancreas looks. Stones, no stones, dilation, strictures, that may benefit from endoscopic therapy. Or they may benefit from going straight to the OR. And again, there's plenty of data on randomized studies showing that maybe going straight to the OR may be the way to go. After five, six years we start to see that endotherapy and surgical therapy may [? calibrate. ?] So endoscopic therapy continues to be a very good option for a subset of patients. I have to tell you from personal experience frequently our surgical colleagues are telling us that this patient is not a surgical candidate, that we should try endotherapy and see how they do. Again, working together with your surgical colleagues, working together with interventional radiologies. And I have to say that it's often extreme help to be able to have a weekly, or every other week, or depending how much patients you see with pancreatitis, and to have a multidisciplinary pancreas conference where you can actually present your cases. Go over and discuss with your surgeons, IR, and radiology, what the best treatment option will be for your patient. So what about steatorrhea? Steatorrhea, it's one of the symptoms that we see in a subset of patients at the initial presentation, particularly in those that have minimal pain or those that have had pain and have been taking medications that sometimes we don't know exactly how they got them. But by the time that we see them, they are losing weight. For the patient to have steatorrhea, approximately 90% of the output of lipase, pancreatic enzymes, has to be gone. So more or less 90% of the pancreas has to be severely diseased before they develop steatorrhea. Steatorrhea suppressions will start actually in the stool. And I am about to show you a picture that was sent to me by a patient that depicts actually what steatorrhea really means. So what we should do is when you have a patient with chronic pancreatitis, you want to make the early diagnosis before they develop a full-blown steatorrhea One of the problems is that some of the symptoms that these patients develop are very nonspecific. Some abdominal pain, bloating, gasiness. This may be nonspecific and this may represent a milieu of other medical conditions. So again, when you have a patient that is losing weight, which should be always on major red flag. So when you're thinking about abdominal pain, gas, bloatiness, and weight loss, you must think about some type of pancreatic disease. Remember also that patients with steatorrhea, you want to make the diagnosis way before this happens so you can prevent the weight loss. By the time they develop steatorrhea and weight loss, there's a significant degree of malnutrition. So this is something that it's very, very important to keep in mind so we can treat the patients soon. How do we make the diagnosis? Every single patient that we make the diagnosis of chronic pancreatitis, we're getting in our clinic a baseline fecal elastase. Monoclonal antibody-- it can be checked. Actually, remember it has to be a well-formed or semi-solid bowel movement. It cannot be watery. And this is a very good, an easy way for the patient to make a diagnosis of exocrine insufficiency. We're checking fecal elastase Elastase is made together with the other enzymes, and it's a very good way to make the early diagnosis. 72-hour fecal bag collection-- if you're going to be doing this, make sure the patient has 100 gram fat diet per day. I can tell you that my patients, the ones that have done it, or when we do it in clinical trials, they are not too happy about connecting stool for three days-- even worse if they have to do it at home. This has to be refrigerated. So you can imagine where the stool it's going-- together with the rest of their food. They are not happy. I can tell you also that our technicians are not happy to be processing all of this amount of stool. So we try to stay away from that, and we try to make our patient's life easier. You can also do a test with pancreatic enzymes and see what the clinical response is. I don't want to encourage you to do this, but this is something that you can do. And this is something that is very well accepted in the surgical world. So if you have a patient that underwent some type of pancreatic resection for benign or malignant disease, and they are having a little bit of diarrhea, increased bowel movement, gas, bloating, FSO. They actually, by their guidance, can give pancreatic enzymes and see how they do. This is something actually that we also doing in a subset of patients-- elevated 13C, mixed [? gas ?] glyceride breath test. I just want to mention this because there's two breath tests that are out there not clinically available, but there's a lot of research being done. I don't think it's going to become available in our clinics for the next couple of years. But I do want to make sure that you know what's coming in the pipeline. Remember, vitamins A, D, K, and E, are fat soluble, which means that if you have exocrine insufficiency, you won't be absorbing these enzymes, these vitamins. I am sorry. Very, very, very important to check particularly a bone density scan in your patients that are coming with malnutrition, weight loss. There's plenty of data, including data coming from Brigham, from Dr. Peter Bank's group, showing with a very large number of patients how patients with chronic pancreatitis are at a very high risk of bone fractures mainly because we in the pancreas world have done, I should say, up a poor job educating the rest of the primary care doctors and gastroenterologists about the high risk that these patients have of developing metabolic bone disease. So check bone density scans in your patients, particularly above the age of 40, particularly if they're smokers. And particularly if they're coming in already with significant weight loss and steatorrhea that you know they have been malabsorbing all of these fat soluble enzymes for a significant amount of time. So with this I would like to change gears here for a second and just illustrate three cases of chronic pancreatitis to present it in a different way. And what we have done also to treat these patients. So I want to start with the first patient, a 55-year-old man referred to our pancreas center for evaluation and possible therapy of chronic pancreatitis and pruritis. The patients has a history of alcohol abuse. The patient has a history of cirrhosis, refractory ascites, varicocele bleeding-- has been sober for four weeks. The patient states that he stopped completely drinking four weeks ago-- has documented recurrent acute pancreatitis. Labs show an AST/ALT that were elevated, [? alcohol ?] is elevated, and a

bilirubin, actually you don't see that direct, but it was mainly at expense of the direct. This patient is obstructed. So what should we do next? What you can see here-- you can get an abdominal CT scan. If you're going to do with this, I would get a pancreatic protocol so you can also rule out tumors. At the same time, you can look at the liver and adjust in structures. MRI, MRCP is another good option. Can you go straight for an ERCP? You're going to be doing an ERCP on this patient for sure, but I would like to see a little bit better the anatomy. And I would like to make sure that there's no other conditions that may be hiding like a pancreatic tumor. Can you do an EUS ERCP? Abosolutely, yes. Surgical consult-- I also think it's a good idea. What you see with this patient that by the time that I saw the patient in the clinic, you see a CT scan of a patient with ascites cirrhosis. You can actually see in the left-lower quadrant, multiple calcifications, particularly in the head of the pancreas, with proximal dilation. You see a little bit of atrophy, and again, in the lower-right you see more calcifications, mainly in the head of the pancreas. If any, actually, I would expect this patient to have much more pain than what the patient is having. Again, by the time that I'm seeing the patient an MRCP has been performed, and as you can see here, the patient has an almost 3 centimeter long distal common bowel duct stricture, very likely this is fibrosis from the gland compressing the common bowel duct, leading to an obstruction. Remember this happens in 20 to 30% of patients with chronic pancreatitis, so it's something to keep in mind. So what should we do next? I don't think we need more imaging. I think we have ruled out other malignancies, for example, other conditions. And I think it's fine to go straight for an ERCP. Yes, you can get a surgical consult, but I doubt that our surgeons are going to be too keen on getting near this patient. As you can see in this ERCP, the left-lower image of the ERCP, you see literally what we just saw in the MRCP-- distal common bowel duct stricture, some proximal dilation. We got up and [? cladogrammed ?] the lower-right image. And you can see a dilated main pancreatic duct with multiple side branches. There's no doubt this patient also has chronic pancreatitis. How did we treat this patient? Over the past couple of years, two, or three, we have been placing fully-covered metal stents. We'll leave them there for 10, 11 months, and then we can do a stent exchange. The patient was having some abdominal pain, pancreatic in origin-- the reason why I also placed the stent that you see fluoroscopically and endoscopically, the blue one in the main pancreatic duct. And that's the endo- and fluoroscopic view. Side note-- if you would like to put multiple stents, that's acceptable. And that's another treatment that we can do for these type of patients. Again, we'll put one. Bring them back in three months. We put two. Bring them back in three months. And then you can put 4,5 until this is fully dilated. Bilirubin, completely normalized. The pain significantly improved-- still requiring narcotics. Unfortunately, we saw the patient recently, and he gossiped me that he's drinking again. Again, this is a very bad habit to break, but the patient overall is doing better. Let's switch for the second case, I just have three cases that I would like to show you. So this second patient, it's a 67-year-old, African-American female, that was referred to us for the evaluation of possible pancreatitis and steatorrhea. The patient was started on pancreatic enzymes at a good dose, and he just wasn't getting better. The patient had a history of alcohol abuse-- has been sober for 40 years-- never had pancreatitis-- never had abdominal pain. Very unusual presentation-- real case. The patient actually sent us a picture that I'm about to show before she even came saying, "Can you help me? If you can, I'll go." Past medical history-- hypertension, COPD. The patient is taking a diuretic, beta blockers, pancreatic enzyme replacement therapy every eight hours-- not with meals-- every eight hours, by the clock. Five alcoholic beverages per day for eight years-- sober for four. One pack of cigarettes per day for 30 years-- still smoking-- not good. Family history-- non-contributory. Weight-- has been relatively stable, three to four bowel movements per day. And depending what she eats, she's going to see some fat in the stool. And depending how much she eats at nighttime, she's going to have to wake up in the middle of the night to have a bowel movement. Again, very suggestive of steatorrhea and malabsorption. Thin female-- otherwise the physical exam is normal. Labs-- very unremarkable. Low albumin and very low pre-albumin. Next step-- again, do you want to admit a patient like these for a 72-hour collection? I don't think so. If you want to do it at home, that's fine. I would check on fecal elastase. But option one, it's possible. Check stool for Sudan stain-- nonspecific-- it's going to show fat most likely. Secretin stimulation test-- you can do it. I don't think it's needed. Fecal elastase-- it's a good idea, but let's wait and see how this stool is looking. Should we adjust pancreatic enzyme replacement therapy? Remember, she taking enzymes every three hours. I think that would be a good idea. Other suggestions are open, and you can fill in the blank. There's multiple things we can do. Very interesting CT scan that this lady had. You can see in the upper-left the classifications in the head of the pancreas. Upper-right extending towards the neck. Lower-left extending into the body, and lower-right with a very, very atrophic gland. No pain. Remember what I said before? No history of pancreatitis Very different, very different presentation. So here you can see the picture that this patients sent. I think this picture truly gives you the sense of what steatorrhea is. Steatorrhea is fat droplets mixed with water that you can see circles, the way that they are projecting here. You can see actually in the side of the toilet also some of the fat droplets that are just getting stuck. You can imagine that this is not going to flush easily, and you see the stool that is pale, frequently foul smelling, and it's not the diarrhea that we tend to think these patients are going to have. This is the classical picture of steatorrhea. So the question is what we do next? And again, we have got a very good history on this patient. And we know the patient is taking by the clock their enzymes every eight hours. So I'm sure you had the right answer. Based on the study from Dr. Enrique Munoz, Dominguez-Munoz, from Spain, 2005. We learned that if we give the enzymes in the middle and at the end of each meal, that coefficient of fat absorption is going to be the best, meaning they are going to be absorbing as much fat as possible. So the idea is that we should give this patient enzymes in the middle and at the end, and you can give enzymes between 40,000 to 72,000, depending where you read. But you want to make sure that you mix them with meals, so that way there's more absorption and these patients hopefully are going to do better. Remember as a side comment other etiologies that may present with steatorrhea. Patients with pancreatic surgery, if you remove a tumor, if you remove a significant amount of the pancreas, you should expect to see some degree of steatorrhea. So give enzymes, supplement them with what they don't have. Pancreatic obstruction, tumors-- you frequently see a tumor with proximal ductal dilation and pancreatic parenchyma atrophy. So if you are removing a tumor, you are leaving a trophic gland, then most likely they are going to need enzyme replacement. Patients with bad pancreatitis-- patients with cystic fibrosis-- and just keep in mind that a subset of patients with type I diabetes, even type II, patients with inflammatory bowel disease, celiac disease, patients with gastronoma, may develop steatorrhea. I don't want to go into too much detail, but just keep it in mind. When the patients are taking the enzymes, the way that they should, as we just mentioned, if they are not responding, think about bacteria overgrowth, think about compliance with medication, very important. Think about under-dosing of pancreatic enzyme replacement therapy. And also think that the patient may have some type of infection-- like giardia cysts or others. So it's very important to rule these. And just to end, let me end with this third case of a patient that is 52 years old-- history of alcohol abuse. Sober for one year. Big time smoker for many years. Now down to one pack per day. Referral to our pancreatic disease center for further evaluation and treatment. Over the past four months the patient has developed bad, severe abdominal pain, constant weight loss, mainly because he's afraid of eating. Every time he eats the pain-- it gets worse-- and increased frequency of bowel movements. Past medical history-- known. Medications-- enzyme replacement. He's taking them the right way. No allergies. He used to be an alcoholic. He is now sober-- continues to smoke, unfortunately, one pack of cigarettes per day. Father with recurrent acute pancreatitis-- also an alcoholic. Paternal aunt with very likely a history of pancreatitis. You can see in these CT scans disease predominant to the head. You can see in the lower-right and lower-left, a lot of calcifications in the head of the pancreas with proximal dilation of the main pancreatic duct. Prior to referring the patient to our pancreatic disease center, there were four experienced therapeutic endoscopies that tried to do ERCPs on this patients. But it was impossible to pass-- bypassing the stones that you just saw by CT scan. So the question is what we do next? Do we refer the patient for surgical decompression? Reattempt ERCP? Laser therapy? Lithotripsy? Meaning going in the pancreas and trying by laser to remove the stone-- break up the stone, which is something that we do on occasion-- extra-corporeal shockwave lithotripsy? Or do you have any other suggestions? So let me tell you. In this case it's a young man. He's otherwise healthy-- very productive. He's still working. I sent him to our surgeons to see if they could decompress his duct and remove the stent because I'm sure that long-term he was going to do well. And he wasn't going to need multiple endoscopic interventions. Long story short, the patient's other surgeons, for different reasons, he wanted to wait, and he declined surgical intervention at this time. So what I offered the patient was to do extracorporeal shockwave lithotripsy, and I'm happy to say that this is something that we're frequently doing in our center here at the University of Chicago. You can see the machine in the left part of the slide. This time actually we did it in the operating room. Nowadays we do it in our new endoscopy unit. You can see actually in the upper-right the water drum. That's actually where the shockwave is being generated and it's being fired towards the patient. In this case you can see after the procedure that we gave 6,000 shocks, and you can see the different settings. What you see here, it's the same day of the ESWL. I

was trying to see if I could pass a wire. I was trying to see if I could put a catheter to give a little bit of infusion of saline at the same time that we were doing the procedure. Again, I tried to follow the protocols from India that they have extensive experience doing ESWL. Anyhow you can see here that it was impossible. We have actually very good fluoroscopic images. This is the machine actually. You can see to the left before treatment, and to the right, the same patient, the same image, the same angle. And you can see that he has actually gone very, very light. This is actually very good. This goes along with fragmentation and destruction off these hard stones. You can see that immediately after we were able actually to pass a wire. You can see a very dilated duct consistent with chronic pancreatitis. You see filling defects, and I hope that's projecting well in your views, and you can see here after we were done, and we left a main pancreatic duct stain. Here you can the size actually of the stones that we were removing in on this gentleman. So in this case it was very, very effective. I wish I could say that as well was as effective in every patient, which it's not-- operator-dependent, machine dependent. So you also have to get some experience and feel comfortable so you can help the operator, which is usually a technician that comes in together. You can see actually here the skin of this patient where the shocks were being delivered. We frequently see these. Depending who you talk to, this may be a good sign that maybe that a lot of waves, a lot of shocks, were transmitted, and they were successful. Again, you see a close up here of the main pancreatic duct of this patient two months later when we're bringing the patient to remove, to do the stent exchange. You can see how beautiful this has decompressed. You see mainly no filling defects in the main pancreatic duct. We were able actually to clear up a little bit more of the stones. In a follow-up six months after stent removal, the patient continues to be completely pain-free. The patient is taking pancreatic enzymes. The patient is gaining weight, trying to quit smoking. Unfortunately, he continues to smoke. But this comes to show you other treatments available for the treatment of these patients with bad calcific pancreatitis, and the different options that we have other than surgery. Again, with this, I would like to end, and if there's any questions, I would like to take them. Thank you.