Small Bowel Post Endoscopy Approach

Speaker 1:
Uh, and we don't consider the the frequencies, um, frequency events, uh, measured to standard rate of, of, of the CMS. Today, uh, I'll be talking about, uh, context to special text to API agreement, uh, some time. Today I'll be talking on an unusual talk, how to approach post-model analysis. Like, many times we would have heard, like, uh, endoscopy is normal, colonoscopy is normal. Get patient is having symptoms. What to do with these patients? We may not know. So it can be a small world. These are the indice patients. These are the endoscopy and colonoscopy are normal. So today's, uh, focus is on how to approach these images, approach to situations with small volume surface. So when small intestine really is not really small, it's actually a really long, uh, it is almost 5 to 7 meters in length, uh, it is it has lots of small, small. But by the name is because of the diameter. Large intestine is almost 5 to 7 centimeters, while small intestine is almost 3 centimeters. Coming to a small recap on physiology of dilation. So after eating food, uh, carbohydrates by salivary analysis, they get converted into polysaccharides. When these polysaccharides from pancreas juices get converted into maltose disaccharides, and from, uh, at the level of intestinal villa, the epithelium, these, uh, disaccharides are converted into monosaccharides. Protein in the stomach, uh, proteins by pepsin, by gastric pepsin, they get converted into polypeptides, and at the level of intestine epithelium, they get converted into small peptides, and amino acids where they are subsequently absorbed. And fat digestion, fat globules, Listen, and they get, uh, uh, uh, they become further small. They get converted into dry grasslands, protein-coating dry tile microns, and then from there into the lymphatic venous system. And also protein absorption happens. In simple terminology, explain digestion system means the process which starts with the right hand and ends with the left hand.

Speaker 2:
Yes.

Speaker 1:
Joe's, Joe's a Catholic. Catholic is geology, like, uh, they are so many liminal core factors and mucosal factors which cause small intestinal, uh, bowel disorders. So what happens at the level of the, in the, in the inside the, uh, small bowel lumen? So there can be small bowel overgrowth. There can be filament Uh, no, it's a basin activity in India, tropical streams, and small industrial factory overflow. So there is a concern, right? There is a rise in grants, last month we had the world IBDA IBDA, so they are very, uh, multiple factors why IBD is rising in India. Grants in 2000, in 2000, the instance was only 4.3%, now the instance is almost 22%, that is a rapid rise, maybe because of Westernized diet, urbanization, antibiotic overuse, and also hygiene advertises. The other things are less important because small industry has dimension inflammation, grants, celiac, SIBO, it can be obstruction because of structures, tumors, and the other part is bleeding, maybe because of bleeding for illusion ulcers and microscopy. Clinical features. So whenever a patient has anemia, central abdominal pain, diarrhea, this abdominal distension and emaciation, think of small bowel disease. So this is a small histology image showing normal will and damaged will, so when small bowel, uh, small intestinal will are damaged, there is no absorption, then they'll have malabsorption, they can have flatulence, diarrhea, spiritualia, weight loss, anemia, hypodeficiencies. So what is flatulence? This is a principle water, which happens the greatest picture pass, contains very bad smell, that is because of hydrogen sulfide, diethyl sulfide, or methanol. Spiritualia, these patients, because of malabsorption, can have spiritualia. Classically described as bulky, voluminous foods, which are made of very healthy, uh, gray-colored, yellowish, they are greasy or oily in appearance, foul-smelling with bulky portion. Clinical features, the most common symptom is diarrhea, patients especially have chronic diarrhea, sometimes we have enthusiastic patients, they'll be telling, "I know every toilet on the road from Kuala Lumpur to Chennai will be straightly with them because they usually suffer a lot because of frequent, uh, frequent use foods." When we take history, we need to know whether it is a pathology in the small bowel or large bowel. There are some important factors. For example, the reaction. The presence was with greasy stools, excessive flatulence, protein malabsorption, even edema, pain, foul-smelling stools, with the vitamin deficiency can be a cause of small intestinal disorder. So we should, uh, take proper history in these patients. For example, this is a cut surface of the intestinal lumen, we have all the layers. In the post mucosa, submucosa, muscle layer, and the xylosa. So what are the functions of each layer? Mucosa, we know, it is important for absorption, secretion, and diarrhea difference, because, uh, when this is altered, when there is mucosal defect or mucosal ulcers, uh, they can develop diarrhea, malabsorption, bleeding, anemia, nutritional deficiencies, and weight loss. This can happen in celiac or early IBD. When submucosa is involved, which contains more of lymphatics and blood vessels, there can be overt bleeding, and hypoalbuminemia or edema. Then muscle layer is involved, there can be colicky pain, vomiting, distension, and they can present with strictures, obstruction, and when the outermost layer, xylosa, is involved, there are fistula, abscess, perforation, which can be seen in the retrograde or advanced growth stages. So when we are seeing these patients, we should, uh, take some time to do physical examination, because there are so many signs. For example, vitamin B complex, B12 deficiency, vitamin C, or folate deficiency can cause all these glossitis, chelosis, or amyloid stomatitis. Vitamin A can cause night blindness, xerophthalmia, follicular keratosis, or bite of spots, they can be ecchymosis, easy bruisability, and there is vitamin K and

vitamin C deficiency. Vitamin D can cause osteopenia and osteoporosis. Vitamin E deficiency can cause ataxia, neurological symptoms. And there are various neurological symptoms which patients can present, these are all, especially we see more of B12 deficiencies, when they have malabsorption, they can have peripheral neuropathy, myelopathy, and vitamin E can cause ataxia, and all this. So frank nutrient deficiency is not very common, but subtle signs we should never miss them in our patients. We should, when we are doing general examination, we should look for clinical clues, they can be white dot spots because of vitamin A deficiency. This is episcleritis in a patient of IBD, then they can be glossitis, amyloid stomatitis, oral ulcerations, lymphadenopathy, cutaneous, they can be follicular hyperkeratosis because of vitamin A deficiency, this is called as well dermatitis, herpetoconus, which can, which we don't see over here, but can be seen in patients with celiac, nail changes like oleonychia, either edema, because of protein loss, vitamin B deficiency can cause bone changes, osteopenia, and ataxia, and there is vitamin E deficiency, and B12 can cause peripheral bleeding. So all these are very important, so before coming to a diagnosis for these patients, uh, rarely we can diagnose small bowel disorders with only single test. We should get clues from history, examination, laboratory evidence, imaging, and endoscopy. All this is very important. So how to diagnose these, uh, these individuals with small bowel problem? There are tests to assess the small intestine function, they are called as functional tests, and tests to assess small intestinal structure. Imaging and endoscopy. When doing functional test, these are all, everything is very, very useful in these patients, especially B12, fecal analysis, stool for fatty acids, all that will be helpful. Whether there is any inflammation, CRP, fecal calprotectin, when there are simple blood tests which are available. And to assess structure, we can do radiological investigations, or endoscopic evaluation. Before we, uh, we, uh, diagnose these patients, we should do some, some tests at least. Uh, how many general medicine degrees are here? So there are some clues, like when we do, they can be, uh, uh, they can be anemia, this is a reference we are showing microcytic hyperpigmented anemia, this is a small, they compare with the lymphocytes, this is smaller than, uh, this is smaller than a lymphocyte, this is an early B12 deficiency which causes hyperpigmented neutrophils, and the lymphocytes and the, uh, this is almost the same, same size. And this is a test of neuroplastic anemia, now the lymphocytes are very small, this is available. So all this is important, stool, uh, inspection is also important, and fecal calc analysis can be useful, but r Readiness is we should ask for the, you know, special same modified icon stain for we should
ask for the acid, uh, modified acid stain for microscope area. We should ask for the modified icon stain. So always request for special staining for this. This is a histology bench which was small, uh, uh, proposals of relation with the malabs option, and this is in a immunocompromised individual, isospora. It is special staining. protecting is an excellent test, but, uh, somehow it is underutilized.
That's what, uh, we feel. This is a simple non-invasive test. It's a marker of, uh, intestinal inflammation. So it calprotectin is there in the macrophages, and when there is excess inflammation, this, uh, calprotectin can be, uh, uh, measured in the, uh, spoon sample. And it is especially useful when we are, uh, dealing with the case of IBD to monitor the disease individuals and whether it is for organic or functional disorder. Also it can, uh, guide us. Whenever people have protectin is less than 100, we do not worry, but more than 100 or even 1000s, we have to do further tests for patients, especially if we are suspecting IBD. It can be a case of IBD. The specific test for confirming malabs option. This is a very simple test. We will not need xylose. Just give them 25 grams of xylose orally, and try to collect the urine sample over 5 hours. If the xylose is not absorbed, the value will be less than the urine, and it assesses malabs option. Uh, but it can only tell whether there is malabs option or not. It cannot diagnose what is the cause for malabs option. Cause for malabs option, we need the tissue diagnosis sometimes. Simple breath test like, uh, uh, we have tests available for carbohydrate malabs option, but unfortunately we don't have tests for fat or protein malabs option because these are very expensive, and they are only for research, uh, uh, setting. Principal for this breath test is just, uh, the intestinal bacteria can metabolize carbohydrate substrates producing hydrogen or methane, and this can be measured by the breath. So just give them carbohydrate oral sugar, and try to do a breath test. If any value more than 20 ppm, it can be a case of malabs option. So as I mentioned, these are very simple tests, non-invasive and easy to use. Any value more than 20 ppm is diagnostic of, uh, malabs option. So when we are using glucose, which with 100 grams, that was 50 grams. Lactulose and fructose we want to test. We can test the, uh, so at our unit we use wet form from USA. In the between suspect lactose intolerance, just give, uh, 50 gram of lactose powder, and if the value is more than 20, it means there's, uh, lactose, uh, malabs option. The specific test for, uh, cycles small intestinal bacterial overflow. We prefer to use glucose because lactulose, even though it is a good test, it can increase the, uh, uh, colonic transit. Intestinal transit. So, uh, glucose is much, uh, more preferred. So this is a normal glucose breath test, and in case there's malabs option, there can be a rise in, uh, hydrogen in the breath. As shown in this diagram. For lactulose, we can see we can this is a, uh, a test result showing double peak. When small intestine bacteria are more, the, uh, uh, lactulose is converted into hydrogen. This is showing the intestinal part, and this is showing the colonic part. So in any patient when they are having low intense symptoms of bloating, abdominal pain, altered bowel habits, think of SIBO, and you can do a small breath test if it is positive. Just for attachment for solar attachment for 5 pmg twice daily. For minimum 14 days, it will help these patients. To assist the structure of the small bowel, we have we can do imaging. Uh, radiology, capsule endoscopy, and microscopy. Radiology, we have N number of tests, but each has its advantages and disadvantages. You can do a brain X-ray. Here there's a, uh, dilated bowel moves in the brain X-ray. You can these we were doing earlier small bowel follow-through, intestinal ultrasound very good for, uh, follow-up of the patients with diabetes. We can do CT endography or we can put a massive gastric loop and do CT

endoplasis and if patients with the osteogenetic we can, uh, if we don't know where is the cause for bleeding, we can do a RBC tax or angiogram. All these are helping patients but there is a challenge, uh, especially small bowel image can be challenging why this happens is when we are doing ultrasound like the entire colon the lumen of the small bowel can have gas and this happens in ultrasound. Lumen of the small bowel is usually collapsed. So even if we need to do contrast, we need to do a large volume of contrast and the. Sometimes if there's a pathology over here, it becomes difficult when you when doing the CT endography whether the pathology is right in this segment or in the adjacent segment. That becomes challenging sometimes. So that's .

kept them in a submarine, and they injected into a human body. This is a world movie, science fiction movie. They are from outside, they are in the submarine, it's going to the IV line, and from outside it is going like we will have this scene, where they are exactly they want to go inside and create an image of. And these are all the IVCs. All these scientists are amazed to see all these visuals. And people thought this is only a science fiction movie, can this happen in real life? But to our surprise, capsule endoscopy came. Any idea how this capsule endoscopy came into medical field? Well, during lockdown I was living this book called Startup Nation, and I was surprised to see something about medical technology written in this book. So this is the person, he's called as he's not a doctor, he's a rocket scientist for Israel Defense Forces. He specialized in electro-optical devices. He had a novel idea how we can use missile technology, we can put this camera into a vein which can transmit pictures, no one encouraged him, but he had perseverance, he persisted and succeeded. Then in 2001, two camp was first time introduced in the world. So this is a old picture, late Jayaleka, and Dr. Vijiman Prasad, subsequently they were the first people to launch capsule endoscopy in the world. There is GBSERV. So this is an example of technological transfer, like optics, electronics, ample of technological transfer, like optics, electronics, Good abdominal pain, bronchitis, and multiple ulcers. There can be small ulcers; stenotic septum potential ulcers can cause strictures later on. And ulcerative stenosis. All this can be diagnosed by capsule endoscopy. Comparing the capsule endoscopy, this is an excellent investigation. It outperformed MR and intraoperatively. And it detects more small bowel lesions than CT intraoperatively as well, you see. And it can identify effectively the bleeding sources in patients with extra-GI bleed. We will start capsule endoscopy; it's a very good technology, but some of only we can see the we can only visually diagnose the patients, but we cannot actually take a biopsy. So now new technology is getting incorporated into capsules; we don't know if it's very interesting. This is a magnetic capsule. We are trying to manipulate from the outside; they're trying to take it left and right. For technology. So for Apache and Zardas, we have invested in lower GMV at the wellness level. We need more sophisticated technology especially for evaluation of the small power. Because even though push entrance copy video capsule entrance copy are available, they have some limitations. Push entrance copy to some extent we consider small one, but entire small one will not be able to evaluate. It is a very big investor push to entrance copy. This is a very senior gastroenterologist performing push entrance copy. So even though multiple attempts he's trying to push into the enteroscope, it's a long it takes time-consuming procedure. Why this with routine electron CM images are shown and when they are taking too much of time the technician everyone gets annoyed. When is this person going to leave me there within me? Despite so many attempts, they're not able to advance push entrance code. I just show the CM image. So what happens over here is this is the inverse of fixed field inverse of. Even though we use so much of force just doesn't give transmitted to the tip of the inverse code. So whatever he's trying to push or pull because of the small power nomenclature it has a tendency of loop. That's why it doesn't advance. So this is a difficulty. So subsequently to put it into the deeper small power and they came with something called a deep entrance copy. So they attach a small wire to the tip of the entrance code or a manual stylus or motorized entrance copy. Then with this technology we are able to see more part of the small intestine. So the same long entrance code will get a small balloon and this will help to clean the small intestine. So they are the single balloon entrance copy and small balloon entrance copy is now available. Then came how about putting something like articular tip of the entrance copy? So what is this cleaning? Same like for pile jam of the clothes and just show this video. Only if we push it will not go the thread. So we need to put some either a pen or something at the tip of the rope. So after doing that we'll be able to clean it and we'll be able to go further. The same technology. They kept a motorized stylus at the tip of the entrance code. And after doing that within minutes we can go inside and we can actually evaluate the entire small power unit. So this is a very good technology. It was available earlier. Now it is undergoing some modifications. So what happens is this is a regular endoscope and there is a small stylus with the tip of the endoscope. And within minutes we'll be able to go into the deeper parts of the small intestine. And the main advantage is we can see the lesions and actually we can treat the lesions while doing the procedure itself. So we launched this technology in 2020. We can use lots of accessories while doing the procedure. There is a polyp we can remove. There is a bleeding area we can actually treat for regularly. So multiple therapeutic applications of stylus entrance copy. This is our publication in video GI series. There we could reach target lesions in most of the patients using stylus entrance copy. So before I close a few case scenarios. This is we had a 64-year-old male patient which presented with different abdominal pain and vomiting. CT and probably showed large segment circumferential wall thickening. Involving the proximal ileum. So we did stylus endoscopy and colonoscopy were normal. We did stylus enteroscopy and we saw these are circumferential ulcerated areas in the small bowel. And we took biopsy and it turned out to be a case of pancreasis. Later on after treatment he was doing very well. This is another case who presented with a HP of liquid in shock. Hemoglobin was less than one. We thought he will not survive. Somehow we could save this

individual. After multiple transfusions we stabilized his hemoglobin. And colonoscopy showed endoscopy was normal. Colonoscopy lots of altered blood inside the large bowel. So we did CT and probably in this patient which showed wall thickening involving the internal loops. So we went ahead and did stylus enteroscopy. We are doing deep enteroscopy using the stylus tube. And at the distal junction of we could see a large ulcerated area and this first was in bleeding. So we did a dual therapy. We did injected epinephrine and then we clipped the lesion. And at the end we sprayed collagen which is a hemostatic powder. And this patient was doing very well. He's on follow-up with us for pancreasis and he's doing very well. The stabilized and later on discharged. So this is an interesting finding when we were doing the stylus enteroscopy. As we are advancing further we got confused. There are two lumens. Should we enter right or we don't know. It's a patient with massive lower GI bleeding. It's a patient with Mickels diverticum. The right side we could see the diverticum. And our subsequently we posted the procedure. Future perspectives. Well endoscopy is involving in a very big way. Now I have a robotic endoscopy is coming in a big way. When motorized capsule endoscopies are getting available now their number is such long. This is something called NPTAGs. What they do is it goes inside your stomach. It's like a moving they can actually like how we used to play video game. We can actually navigate capsule inside the human being. This is like a torpedo. How they are propulsion devices at the end. Using a simple remote we can actually manipulate the capsule. And future is very interesting. Now there's something called a slang robot. Future this may become available in medical technology. If someone scholars doctor graduate by mistake this slang robot tuning them we can build. It will just go inside and help in that way. And without doing any procedure patient can get discharged. So this is very interesting. And who knows later on they might develop a small robot like this like a spider like this which can be kept into a capsule. So a patient has abdominal pain. Just give this capsule. Robot will go inside. It will keep searching what is happening inside. So here there's a perforation. So immediately it sees and it starts seeing everything. All this may not be available now but maybe it available in the future. Before I close so there are so many small bowel disorders like inflammatory Crohn's. Malabsorption syndromes. All these are there. So to diagnose this small intestinal disorder it is we need to do more tests. One test may not be clear enough. Peripheral history clinical examination provides first two to localize and proceed for further evaluation in these patients. Combination of tests like functional assessment and structural evaluation is required. Every unexplained anemia or chronic area weight loss or obscure GI bleeding. This has a journey to the small bowel. Thank you very much.

Speaker 2:
And I will just say that this is actually just the itself is also so far supposed to suggest this is our supplements updates. Reviews. Studies on conditions of liver then they thought it is very important then it's also on liver and for the hepatic liver to cirrhosis continue whatever it is it must go into 2026. Still there is Dr. CGC attached which I must welcome sir. Dr. CGC please.

Speaker 3:
Thank you sir. Sir.

Speaker 2:
And the next session I request from sir Dr. Virakesari to chair this session. Yes sir.

Speaker 4:
Thank you very much dear friends. The session is next session is it's important one. How we are normal fatty liver is converted in the cirrhosis liver. Fatty liver is very simple. Accumulation of fat. More than 5% of body weight. Liver structure is maintained but cirrhosis is here. Gradual process. In the liver structure structure take a long time. I say a difficult time. How he is going to see children going to delivery chair. How he is a fatty liver will be converted into cirrhosis. Different structure. I welcome sir. Thank you.

Speaker 2:
Thank you sir. Thank you to the APA chapter of Coimbatore. Thank you to all the senior faculty here. And Sun Pharma for giving you the giving me the opportunity to share a few minutes with you all. On the outset endoscopy brilliant preservation Dr. Vamsi. So that's that should be an inspiration. I've been we all are physicians. Primarily but the passion for intervention took us towards subspecializing into gastroenterology. But our core is always medicine. So we all are physicians at True Blood. So when it comes to fatty liver this is a topic which like out of 10 four people are affected. That's how common it is. So we are room of

Speaker 1:
Becomes a big void, and finally somebody hits on it and ends up losing their life over a big accident, right? That's how fatty liver is. We say, "I've sat in diabetic OPD and saying fatty liver . ." They've said that, right? But over the decade, now things are changing; we're worried. Because my neighbor, who's a 10th standard kid, brings a report and says, "I have fatty liver, uncle, what should I do?" That's scary. Your grandkid comes and says, "I have fatty liver," that's where

we are living, because of Swiggy, Zomato, you can get everything within minutes, and you can sometimes get a doctor also. So that's where we live. So over to the topic. 6 objectives, right right? Basically, fatty liver, the name has changed, because it's become much more inclusive. We have no longer calling it non-alcoholic fatty liver. We'll go into the reasons why. I'm sure you would have gone through it. Let's go to the step 2, because most of the patients come and sit in front of us giving one thyroid care or a wireline or some lab report saying, "Doctor, I left , but ultrasound showing fatty liver." What next? I'm sure they would have gone to AI and got that also, checked, and they'll be conducting Vaiva with this. So you need to know what more can be done as non-invasive tools, point 2. 3, okay, fine, we have got the test, we have done the scores, but how do you stratify? How do you say, "This is where you need to stay, this is where you need to go and meet somebody, a hepatologist or a specialist," and you counsel them? And before going to the counseling, you need to risk stratify, right? So that comes into point number 3. 4, interventions, pharmacotherapy, we're all great at it. I'm sure all of us practice very well. 5th, most important thing: when do we initiate? Sometimes when patient has landed up in advanced cirrhosis, when should we say, "You are in cirrhotic range, but you're still not decompensated"? When do you intervene on time? So that will be the 5th objective. Last will be all the GLP-1, semaglutides, and all the things which have come up in the market and that will be the least of the options. If we stick to the first 4 or 3, I think our job is done. So we'll stick with that. So the revolution of renaming is not something like a old wine and a new bottle, right? Technically it is. It is fatty liver, but the emphasis is towards the word metabolic, right? It is always marshally, mash, and everything metabolic, right? So whenever we say metabolic dysfunction, people have started looking at it much more holistically than just saying, "I don't drink alcohol, it's non-alcoholic fatty liver." So the stigmatizing and the non-labeling is in a better way, in a better connotation, right? So second point being, but alcohol is still alcohol. It's inclusive. The framework has changed over 2023, but it has absorbed most of the things. There is MET-ALD, where you have moderate alcohol use, you have alcohol use also, and you've got metabolic dysfunction also. You call it MET-ALD. You no longer call them alcoholic, non-alcoholic fatty liver, right? So you call them MET-ALD. And you also bring in autoimmune causes and say, "So you have fatty liver, you have an autoimmune disease, bring them together." Say, and then you call them MAFLD with AAH, combination. Those things bring into a more inclusive

Speaker 2:

Speaker 1:
it's like an umbrella term, right? That's how we have to proceed towards it. Let's not ignore and say, "You're non-alcoholic, henceforth." And the emphasis again and again and again to all the young colleagues, it's metabolic, you need to use the word metabolic. Emphasize the word insulin resistance. I'm sure we all read, but the emphasis is more crucial because metabolic doesn't stay with sugars, fat, and liver. It's composite from head to toe, from stroke to heart attacks, to kidney diseases, and retinal dysfunctions. We're going to have it all. So start early and liver disease might be just the one tip of it. There's a whole bunch of it. So what are the cardiometabolic risk factors? You need to elaborate to the physicians. I mean, the patients. So a high BMI, especially in India, the BMI is set on the lower side. When compared as Southeast Asians, we have set at 23. The fasting glucose and impact fasting glucose, even at the impact stage, we need to pay attention to them because it could signify an insulin resistance very young. And of course, BP targets, triglycerides, weight circumference. Everything done in our OPD desks, right? In our physician desk, this can be done. The step 1 goes later. Most of the patients come with ultrasound or some fibro scan or things like this. Histology is almost getting phased out. We don't do, I'm sure Dr. Wangsi also will agree, none of us do liver biopsy for fatty liver these days. And it's always, always, always sorry, any pathologist involved? Was it me?

Speaker 2:
It's you.

Speaker 1:
Oh God, okay. This is how sometimes it works, right? Okay. , . So histology is no more the criteria. Exam going, please read. Let's stop it, just leave. But basically, when you take into all the cardiometabolic activities, basically this was the syndrome mix, right? The metabolic syndrome. So the metabolic syndrome is still the golden thumb rule. Identify this first rather than going into the step 1 of getting an ultrasound and finding an imaging. Most important, in this criteria, you will never find an LFT. Most important, right? There's nothing called LFT here in this report, right? It's always cardiometabolic risk factors. You get an imaging, and that stays, right? A normal LFT does not mean the patient has got no fatty liver. So why the fatty liver starts with steatosis, goes on with no fibrosis stage, but there is still mild hepatitis, and then goes on to a fibrosis stage, then goes for a compensated cirrhosis, and finally the decompensated cirrhosis, right? So, but majority, majority are still very early. It's still in the state of stage, stroke. 80% of your patients will still be just having a fatty liver without developing any hepatitis, fibrosis, or anything. But the point is to find out those 20 patients, right? So but the global incidence of NAFLD is rising crazily. Like how IBD is rising, almost we say 1 in 4, sorry, 4 out of 10, but

the rising of the global incidence, especially in India, Tamil Nadu, we see every 4 to 5 patients having a fatty liver problem. At the level of steatosis, luckily we have a low incidence of hepatitis, we dig deeper, we get so many other things. Sometimes drug-induced plays a role, sometimes alcohol plays a role, sometimes autoimmune disease plays a role, sometimes drug intake plays a role, and all those things play a role. We are really concerned about the Leading cause of liver transplant across the world, it's number one in the Western world. We are getting it almost closer in our world, and the biggest concern is the rising HCC incidence, right? The surveillance which was never, ever a factor for a simple start like fatty liver, is becoming an increasing trend where HCCs are diagnosed with fatty liver disease at the end, right? But the gold standard—what do we read? We read about wire hepatitis, we read about tattooing, we read about all those things, but seldom we do place importance for HCC rising etiology as fatty liver. So the disease spectrum has to be thought in this spectrum; it should never be fatty liver diagnosed, treated, get cured, never. It's always a trend; it's always a continuum. And as physicians, you need to break the cycle right here in F0 or F1, never let the patient get into advanced cirrhosis. If we do that, we are successful. Histological markers—I'll skip theoretical—hardly we do these days. But these are done in certain centers who, like the centers I work with, they do an incidental—they do a cholecystectomy, they find a fatty liver, they find a simple cirrhotic changes, microneedle changes, they do take a liver biopsy. In the same setting, when they come back, we can say exactly where do they stand, but most of them have a normal LFT, still most of them have a normal imaging too. So these are things which are gray lines, but still, more than the histological grading, our emphasis of F0, F1, F2, F3 are all with non-invasive markers, never with invasive markers, okay? So when a patient comes to you—any one of you here, right—don't push them to do a fibro scan. Don't push them to do fancy stuff. Don't push them to do costly ELF scores. No. Whatever happens, settle down, stick to the basic simple criteria, calculate a FIB4 score. All you need is age, all you need is the AST and ALT of the patient, and the platelet score. Most of us will have a CBC, LFT, in our table with our routine screens, right? Anything less than 1.3 is a low-risk patient. Anything higher than 2.6 or 3, you're dealing with some patient who has an elevated transaminitis or sometimes a platelet dysfunction which can be slightly subclinical, but still, one portion of—very important portion—it is less accurate in elderly age group. So don't take—because age as a factor will multiply too many factors along with this, so it'll give a negative value or a false positive value. So stick to the golden chunk, the 20 to 50s, and target them, and just do this FIB4 score.

has the FIB4 score as a part of the package. Just go and see them. The patients will come and say, "What is this FIB4 score?" Basically, tell them, "This is for fatty liver, and this is given in a such a large social initiative like Santhi Social Service in Coimbatore," right? FIB4 score is there, right? So it's—then it becomes mainstream, right? A place like that gives your report; we all need to know, simple. Okay? Second line: when do you get a fibro scan? Pharmas does. Centers does. There are individual centers who have fibro scans, one or two machines. But when do we refer? Only when you have a high FIB4 score, then you refer the patient as a second line for a fibro scan, right? Fibro scan is nothing but how stiff is your liver. You get two values: one is your fat level, which is a CAP score; second level is your fibrosis level. So you get two scores out of it. You pay attention to both. I request you to see both the values. Don't just stick to the fibrosis value, right? Sometimes the drugs you treat are to make sure both the fat comes down and the fibrosis comes down. Both the targets have to be achieved, right? You can't eat rassam without the rice. You have to eat rassam with the rice, then it becomes rassam rice, right? So you can't do both. So fatty liver, treat the steatosis along with the fibrosis. If you remember this, you'll get the end of the continuum straight. And you'll prevent a cirrhosis, right? LF score is a composite value which hyaluronic acid, the type 3 collagen, and the metyloproteinase are being measured, and then you get into factors. This is more a prognostic marker than a detecting marker. We use this in our center as a tertiary care center. So this is what we use it for: prognosticating and following up the patients rather than starting the screening with this. So LF comes the last, FIB4 comes the first. So never do the reverse order. Okay. So we have identified the patient with any cardiometabolic disease, clear? You calculate the FIB4 score, already your reports will have. If you don't know to calculate, please put in your phones. The phones are smart enough to calculate the FIB4 score because it's got a square root and algorithms and things like that. So simple calculators will work. Once your risks are defined, treat yourself, then refer whenever you have a high FIB4 score. Keep it as 3. Don't complicate 2.6 and all that. Keep it as 3. Anything above 3, you refer. Anything less than 1.3, you keep it yourself. No problem, right? So treatment will keep it at that, right? That's not the paradigm. Weight loss, diet, exercise. ,

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. , 5% ,
, ? .
, " 10%." We very, very categorical.
, we need to be having a very clear targets, right? When you say diabetic management, you want the HPMC to be a certain level, right? You want your cholesterols to be a certain level. And we strict about your weight loss targets. Please say, "You need to lose more than 10%, then you have a chance of improvement of fibrosis." You lose more than 15%, you can even reverse the cirrhosis. Most of the cases, this is a fundamental philosophy where semaglutide works, bariatric surgery works, and all those care works when your percentage weight loss is higher, right? Body is a miracle tool. When you reverse your cardiometabolic risk factors, your liver heals. It's a regenerative organ. And it heals. Diet, it's fluctuating. I don't want to go into the depth of it. Mediterranean diet is the only published data, but our South Indian diet is good enough. You need to just tell them, "You just need to reduce and coordinate the ratio of carbohydrates, emphasize the fact

fatty liver is never due to the fact you consume. It's because of the carbs, and if they have an inherent insulin resistance, to check on the carbs and the regular molecules." Ultra processed food, all those things, use it for your social media and patient awareness. Coffee is again black coffee, never our bias which we call as filter coffee, with the coffee milk and sugar and everything on the planet Earth. So coffee is black coffee, which is hypoprotective. Avoid alcohol. If patient says, "You want to drink alcohol," tell them, "Just have a bit of rat poison along with it, taste it along with the alcohol, you'll be great," right? We sarcastic, but be brutal, right? Sometimes you save a patient, but tell them, "Alcohol is poison for you, stop," right? And doctors, please don't drink in your medical conferences. So exercises, you need to be very clear. Resistance training is more important. Aerobic activity is balanced. Simple walking, even if it's walking, beyond 40 minutes is where the aerobic activity kicks in. So they can't go around the race course and say, "I have a coffee and bail in coffee," and say, "I've done my walk and whatever." That doesn't work. Be very brutal and say, "You need to do a certain amount of minutes and time for you to get in a better shape," right? Okay. Coming back to pharmacotherapy, very important. We don't have non-UDCA here, despite the panel, right? Not used in fatty liver, right? So Arsacol enough, not useful in NA15, right? First thing out, huh? Sorry to some pharma. No evidence. Right? Second, Resmedirom is FDA approved. Semaglutide is FDA approved. There's a peptide FDA approved. These are drugs meant for advanced fibrosis, advanced F2 to F3 fibrosis. When you start with going back to PPAR agonist, Saraglutisab, most of the physicians have experience with using it as a diabetic drug for decades. And it has been very useful for fatty liver. So use these therapy with judicial judgment. The Semaglutide has good evidence in the essence trial. As of 2024, you can go up to a dose of 2.4 mg per week. And continue the therapy. The endpoint is still unclear. So most of the time, it goes on. And especially in India, Semaglutide is given a license, never for fatty liver. It's still in evolution. It's still given for obesity and diabetes. Obesity also is a gray shade. It's only for diabetes. We have stuck to an anecdotally, I could say, we have stuck to microdosing the patients till 1 mg. And we continue with 1 mg. We are never gone to 2.4. They don't tolerate. Centers might have a different opinion according to their protocol which they follow. There's a peptide also, a GLP-1, GLP-2, and a lot. We have Synergy trials, which shows a good MASH resolution, also the fibrosis resolution. Why are these drugs emphasized rather than non-UDCA, Saraglutisab, and all those molecules? Because we are looking at fibrosis reversal. We were never having drugs beyond metformin and vitamin E. We are looking at fibrosis reversal. So these as physicians, we should be aware of these molecules which are available. The Resmedirom is a beautiful drug. It's a thyroid hormone receptor agonist, which works in F2 to F3 fibrosis. It's still yet to come mainstream in India. Metropharma has it. But they're yet to come into mainstream. But has great improvement at 52 weeks. Improvement. The Obitacolic acid is one molecule which 2 weeks. Improvement. The Obitacolic acid is one molecule which 2 weeks. Improvement. The Obitacolic acid is one molecule which Good luck. But please play a composite role; liver is never a single organ with its function, right? So always when you see the LFT, don't go with LFT; check for your albumin, check for your platelets, check for your splenomegaly, check for your portal vein diameter. Keep your eyes and ears open towards a fatty liver when is this fatty liver becoming a decompensated one, right? We play so much attention to this and we can easily prevent most of the patients from getting decompensated. I mean, to avoid becoming decompensated. Okay, head CC. All MAFLD patients with their four fibrosis, regardless of etiology, are at risk of head CC, Hepatocellular Carcinoma. The highest risk seems to be the diabetic fatty liver combination; the hepatitis combination with the fatty liver, again, plays a huge role. Surveillance is simple; ultrasound every 6 months is the standard of care, regardless of hepatitis or MAFLD. When you add AFP, the alpha-fetoprotein value detection, you still get a better outcome, right? But you can't recommend this for everybody at 6 months, so ultrasound is good enough. MRI only for indeterminate lesion, but use it with IV contrast. Triphasic CT, don't do it for every patient; avoid radiation and only when you have a strong suspicion at the USG level, please push it towards it. The special situation is something which I want to mention of lean NASH. This is a great group of patients where our people will be looking lean; they'll have a low BMI, but you all would have seen they have terrible sugars to control. These are the patients who have terrible insulin resistance despite normal weight. These are patients who develop sarcopenia. I want to emphasize on sarcopenia because muscle is the powerhouse of mitochondria in our body. Unless your muscle health is good, the sarcopenia will kick in, and your entire metabolic health goes for a bad zone, right? So a lean patient with uncontrolled sugars and bad insulin resistance has a much, much higher severity and has a much more severity to progress to worse decompensated disease later on in his life. So catch hold of this patient especially. Don't keep leaning only on the BS physicians; we should play more attention to our lean FLD along with the obese FLD and the diabetic FLDs. Bariatric surgery and even endobariatrics has come in a big way where obesity can be managed endoscopically, not just with balloons; endosuturing is available. Most of the centers have the facilities and I'm not diving into it. So the take-home message: the name is reframed; it's no more non-alcoholic fatty liver. Screen all patients for fatty liver; we said Santi Social has 4 score, so we all should have 4 score in our phones. MD Calc is a good app; just put in 4 score, you'll get the values. Please write it in your case sheets; please write it in your notebooks. Please check for yourself, right? How many of you checked your own 4 scores? Do that, right? To begin at home, right? Check for yourself, check for your mother, father, sister, wife, son, daughter; do it. Please do it this weekend. Please check, and again, stratify, right? Stratify yourself; where do you stand? Do you have just a fatty liver? Do you have a fibrosis? If you have fibrosis, which fibrosis are you looking at? Do you have F0, F1, F2? You still have F4 fibrosis, which are almost cirrhosis. You still have an opportunity to reverse it with the newer drugs, and a lot more drugs have come up in the pipeline. And 4 score, 4 score, 4 score. Normal LFT doesn't mean LFT of liver is normal. Thank you for

patient listening.

Speaker 2:
So thank you, sir. Please explain how is cirrhotic—I mean, fatty liver converted into a cirrhosis liver—and explain the role of fibrosis. So when you subject the patient for fibrosis scan,

Speaker 1:
I would say avoid because there are two conditions where patients come into our room saying, "I have got a high fibrosis score. I mean, fibrosis scan value." They'll have an abnormal LFT, sir. First, an abnormal LFT with the fibrosis fibrosis scan showing a high value, literally means nothing, sir, because you got hepatitis and they are going on to do a fibrosis scan, which will show a terrible value, and they'll come and say, "Do I have cirrhosis?" Again, they get confused. First, 4 score is good enough, sir, which we can do. Refer to a fibrosis scan only if you have a very strong doubt of fatty liver. You have high 4 score, refer sir. Otherwise, stick to the 4 score.

Speaker 2:
Thank you, sir. One more question to answer that. You are doing capsule endoscopy anyway. Any disorder in this, any complication, capsule endoscopy?

Speaker 1:
AFP CT doesn't pick up small bowel stricture; it can get stuck over there. The only way to remove is either doing an endoscopy again. They can—if there is a stricture, obstruction, but if there is a stricture, you should not do it. But sometimes imaging may not pick up. We had two cases in almost 15 years. Those cases we somehow removed using an endoscope.

Speaker 2:
Thank you. Any other question? In the audience?

Speaker 3:
One of Sarajevo.

Speaker 1:
Sarajevo, sir, can be used for patients with LFT abnormalities. We have a abnormalities, not for regular patients who don't have an LFT abnormality. Diabetic, along with fatty liver, with the LFT change, with the, let's say, high 4 score, you still can start on Sarajevo, sir. 4 MG per day is good. The maximum studied outcome has been two years, sir. After two years, we discontinue. The endpoint will be normalization of LFT scores. With the normal normalization of fibrosis scan values, that will still help.

Speaker 4:
I am Dr. Vishwanathan, professor of medicine and senior consultant at KMCH. Excellent lectures, sir. The one thing is, you have very well said that RSO deoxycholic acid has got no role at all. And even the latest edition of Aricin says that UDCA betaine dipeptidyl peptidase and also silymarin all four drugs have got absolutely no role in MASH. And the only thing is lifestyle, lifestyle, lifestyle. And the other thing is other things are respectable and semaglutide receptor.

Speaker 1:
Thank you, sir.

Speaker 4:
Thank you. And also excellent lectures, sir. One thing I would like to know is, do you encounter a lot of tropical food here?

Speaker 1:
Yes, sir. We do encounter, sir, but it's less, sir. 100 diagnosed, that's what we see. CMC, they had a huge publication. They have a follow-up with patients. The only thing is we need to treat them with long course of antibiotics.

Speaker 4:
Antibiotics. And do you get subtotal villus atrophy in that?

Speaker 1:
Many times in our endoscopy histology biopsies, we do this, sir. Thank you very much, Dr. Speaker, Dr. Wamsi, and Dr. Sridhar for relating concern on small bowel disorder and this metabolic dysfunction associated with fatty liver diseases. And I thank the audience also for your overwhelming response on this Saturday evening. Very traffic for attending this evening session. Thank you very much, sponsor. Sponsor is very difficult to harness any program. Some seconds. Thank you very much. Do you want

to tell anything? Please invite us for dinner. Otherwise, we will not join.

Speaker 4:
Thank you, sir. Wamsi, sir, and Sridhar, sir, for wonderful talk. And thank you, Ramkumar sir, Virke sir, sir, for the challenging position, sir. Please join us for dinner. Dinner arranged in the restaurant, sir. Please join with us.