Episode Book

EPISODE #5692

When Bile Reflux and Bloating Collide After Gallbladder Surgery

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SYNOPSIS

Daniel had his gallbladder out years ago and has been carrying bile reflux, bloating, and a stack of diagnoses ever since. After a holiday dinner of red wine and curry, he spent the night walking his apartment with acid in his throat and his teeth aching. This episode pulls apart what's actually happening: a chemical and motility problem downstream of losing an organ, plus a somatic one in how the diaphragm and abdominal wall move together — two different problems that keep getting treated as one. We look at why the PPI isn't failing so much as aimed elsewhere, why a negative gastric emptying test doesn't rule out postprandial distress syndrome, and what questions are worth bringing to the next appointment.

TRANSCRIPT



Corn

Before we get into this one, the usual thing: nothing on this show is medical advice. This is two of us thinking out loud about a situation a friend described. That's it.



Herman

And it's a real situation, so we're going to take it seriously.



Corn

Daniel wrote in at length. Years ago he had his gallbladder out, and you know from us talking about him that that surgery has never fully sat right with him. One of the things he's carried since is bile reflux. He tried cholestyramine for it, and he describes it as incredibly hard to find and completely impractical to use. Sachets, several times a day, and every other medication he took had to be spaced around it. That alone tells you something about what living with this has been like.



Herman

Keep going, because the dinner is the important part.



Corn

Last night was a Jewish holiday. Hannah cooked, there was red wine, there was curry, and they went out to try to catch some of the Simchat Torah celebrations. Almost immediately he felt bloated. Visibly distended. His words were that it felt like carrying a heavy pillow on top of his stomach. Uncomfortable enough that holding Ezra was hard, and Ezra is fifteen months old and wants to be held. So they stayed out twenty minutes and came home.



Herman

And then the night.



Corn

He went to bed early with the baby, woke in the middle of the night with regurgitation, burning in the chest, and the sensation of acid pooling in his throat, which means constant throat clearing. He took Tums, but he'd already taken his omeprazole on schedule, so there was nothing else to reach for. His teeth ached from the acid. He couldn't lie down, because lying down makes it worse, so he walked the apartment. Sometimes it takes an hour before the throat clearing runs its course. On the nights it happens, it can happen a second or third time. Most nights it doesn't happen at all, but when it does, that's the shape of it.



Herman

That's a very specific pattern, and I want to flag right now that I don't think it's random.



Corn

He's collected some diagnoses along the way. A gastroenterologist told him abdominophrenic dyssynergia, postprandial distress, functional dyspepsia. He did the egg test for gastroparesis, negative. He tried low-dose amitriptyline, which gave him brutal fatigue and didn't help the bloating. He was told to eat smaller meals, and even when he does, the events still come. His own read is that his system gets backed up processing what he takes in, and it reacts with distension and then regurgitation, and food is a definite trigger.



Herman

Which is a better description of the mechanism than a lot of people manage.



Corn

Here's what he actually asked for. He's going back to his family doctor next week. Last time went nowhere, and he wants to walk in with specific questions and specific directions worth exploring. He says the cumulative effect on his quality of life is large. So let's take this apart, because there are two or three separate things happening here and my strong suspicion is they've been getting treated as one.



Herman

Let me put the thesis out and then we can build on it. Daniel is carrying three diagnostic labels that sound like a pile of jargon, but underneath them there are two different problems. One is chemical and motility-based, downstream of losing an organ. The other is somatic and behavioral, in how his diaphragm and his abdominal wall move together. Those two problems need different treatments.



Corn

And the reason nothing has worked is that he's been taking treatments aimed at one and applying them to the other.



Herman

Right. Start with the surgery. The gallbladder did two things: it stored bile, and it concentrated it, sometimes by a factor of ten. Take it out and you lose both. What you're left with is dilute hepatic bile dribbling continuously into the intestine instead of being released in a bolus when fat arrives.



Corn

Continuous instead of on demand.



Herman

That breaks a feedback loop. Bile in the intestine normally suppresses cholecystokinin. With constant bile delivery, CCK signaling gets disrupted and levels stay elevated, which relaxes the sphincter of Oddi, the valve at the bottom of the bile duct. Relaxed sphincter, plus a stomach that isn't protected the way it used to be, and bile goes where it shouldn't: up into the stomach, and sometimes higher.



Corn

Give me the scale on that, because I want listeners to hear it's not a fringe outcome.



Herman

A retrospective cohort found bile reflux gastropathy in just under sixty-two percent of post-cholecystectomy patients. Roughly six in ten. It is a common consequence of a very common surgery, and it is nonetheless badly under-treated.



Corn

Which is the frustration. Mayo Clinic's own language on bile reflux is that it's harder to treat than acid reflux and that there's little evidence determining the effectiveness of the treatments. That's their position, not a complaint from a patient forum.



Herman

So when Daniel says last time he pushed and got nowhere, part of that is the state of the field. He isn't failing at treatment. The treatment landscape is thin.



Corn

Alright. Take the first problem then, the mechanical one, because I think that's the one that's been most misunderstood. Abdominophrenic dyssynergia. Explain what's actually happening, because the name makes it sound psychological and it isn't.



Herman

It's a somatic behavioral response. The diaphragm and the anterior abdominal wall are supposed to work as a pair. When you inhale, the diaphragm descends and the belly comes out. In APD, that relationship inverts. The diaphragm contracts down while the abdominal wall relaxes, and the contents of the abdomen get pushed downward and outward. The result is a belly that visibly distends, in real time, in response to a meal.



Corn

So the distension is structural. It's the wall moving, not gas arriving.



Herman

There's a study of a hundred and thirty-nine patients with APD. Girth increased in a hundred and thirty-eight of them. In ninety-nine of those patients, intestinal gas volume stayed within three hundred milliliters of baseline. Three hundred milliliters is nothing. That's the cleanest possible demonstration that distension is not gas.



Corn

Which matters enormously for Daniel, because every piece of bloating advice he's been given assumes gas. Cut the FODMAPs. Take the simethicone. Watch the beans. If the distension is a hundred and thirty-eight out of a hundred and thirty-nine, and the gas is flat, all of that advice is aimed at the wrong target.



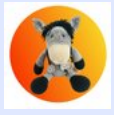
Herman

That's the point. And there's a timing signature he can check himself. APD distension typically peaks around forty-five minutes after a meal. If his belly is at its worst forty-five minutes in, that's a clue he can bring to the appointment. If it's worst at ten minutes, that's a different story and it points elsewhere.



Corn

That's a useful thing to hand someone. Not "track your symptoms," but a specific clock.



Herman

Now the second label. Postprandial distress syndrome and functional dyspepsia. Functional dyspepsia is a disorder of gut-brain interaction with no structural abnormality. PDS is the subtype dominated by postprandial fullness and early satiation, the feeling that you've eaten far more than you have, or that you can't finish what's on the plate.



Corn

At this point I want to correct something I think a lot of people have wrong, and I suspect Daniel may have had it implied to him. The mechanism for PDS is not delayed stomach emptying.



Herman

Impaired gastric accommodation. The stomach is supposed to relax and expand to hold a meal. That reflex is blunted, so a normal volume of food sits in a stomach that hasn't made room for it, and the person feels full and distended. Delayed gastric emptying turns up in only about thirty percent of functional dyspepsia patients, and it doesn't correlate with symptom severity.



Corn

Say that last part again, because it's the hinge of the whole appointment.



Herman

Delayed emptying appears in about thirty percent of FD patients and does not correlate with symptoms. Which means a negative egg test does not rule out PDS. His egg test coming back clean is entirely compatible with the diagnosis he already has.



Corn

So if anyone along the way treated that negative result as closing the book, that's the error worth naming. A normal emptying test wasn't evidence against PDS. It was information he already knew.



Herman

The other thing the research says plainly is that a negative emptying study doesn't mean there's nothing there. It means the mechanism isn't emptying. Which leaves accommodation, which nobody has measured.



Corn

And nobody has measured that for him.



Herman

Nobody has measured that for him. That's a question worth asking directly.



Corn

The amitriptyline failure. I don't think it means what people assume it means.



Herman

It doesn't. TCAs are best supported for epigastric pain syndrome, the burning and pain cluster. They have much less support for postprandial fullness and distension. So the drug may have been mismatched to the symptom on top of causing the fatigue. He isn't necessarily a person who failed neuromodulators. He's a person who was given the wrong one for the cluster he has.



Corn

What's the alternative in that class?



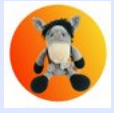
Herman

Mirtazapine has a different side-effect profile. It's sedating in a way that some patients find useful, and it does cause weight gain, and I don't want to gloss that. But it isn't the same drug wearing a different name. It's a different mechanism.



Corn

And there's a whole category he may not have been offered at all. Prokinetics.



Herman

Metoclopramide, domperidone, prucalopride, itopride. Those have evidence for postprandial distress and gastroparesis, particularly for nausea and early satiation. If he's never been offered that category, that's a gap worth naming. I'll add the caveat that these aren't free of side effects and metoclopramide in particular has a real risk profile, but as a category they're missing from his list, and that's the thing to raise.



Corn

So that's the mechanism. Now let's talk about what happens at night, because the pattern Daniel describes has a name.



Herman

It does, and it isn't just bad acid reflux. What he's describing is laryngopharyngeal reflux. In ordinary GERD, refluxate stays in the esophagus and you get heartburn. In LPRD, the refluxate crosses the upper esophageal sphincter and reaches the throat and larynx. And the material isn't just acid. It's pepsin and bile salts too. The classic presentation includes throat clearing, a sensation of something pooling in the throat, a hoarse or irritated voice, dental erosion.



Corn

And often without much heartburn, which is why it gets missed.



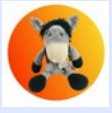
Herman

He does get the burning in the chest, but the throat clearing and the teeth aching are the LPRD features and those are the ones he mentions repeatedly.



Corn

So what was the meal doing? Because I don't think this was bad luck.



Herman

This was a predictable pharmacological event. Red wine relaxes the lower esophageal sphincter and irritates the esophageal lining directly. Curry is spicy and usually high-fat, and both of those properties relax the LES and slow gastric emptying, which keeps volume sitting higher for longer. Then he went out, came home, lay down. Lying down removes gravity from the equation. It's the exact sequence that produces the symptom.



Corn

Nothing about that is mysterious. He put three separate LES-relaxing inputs into a stomach that already empties unpredictably, then lay flat.



Herman

And he took his omeprazole on schedule, which is worth saying out loud, because acid suppression doesn't touch bile. If bile salts are the irritating agent, you can suppress acid all day and still get the mucosal damage.



Corn

That's the piece I don't think he's been told. The PPI isn't failing. It's aimed at a different thing.



Herman

Right. Which is why the diagnostic he'd want is a HEMII-pH study. That's the reference test for laryngopharyngeal reflux specifically, and it can phenotype the reflux: acidic, weakly acidic, non-acidic, gaseous. It tells you what is actually coming up. He has never had it. That's a concrete, nameable test he can ask whether it's appropriate for him.



Corn

Which brings us to the drug he couldn't get and couldn't tolerate, and there's a detail about it that changes the whole story.



Herman

Cholestyramine is a bile acid sequestrant. It binds bile acids in the gut so they get excreted instead of recycling. The problem is that Mayo's own guidance notes that bile acid sequestrants can cause severe bloating as a side effect.



Corn

His single worst symptom.



Herman

So it's entirely possible the drug he struggled to obtain and couldn't tolerate was making the thing he was trying to fix worse. That reframes the cholestyramine chapter from "I failed the drug" to "the drug was a poor fit." Which is a very different thing to tell a doctor.



Corn

And the supply problem was not in his head.



Herman

Upsher-Smith discontinued the four gram, five and a half gram Prevalite powder in May of last year, and they called it a business decision. Supply has been inconsistent since, and it's still inconsistent now. That's not a local pharmacy failing to stock something. That's a supply chain issue, and it's worth him hearing that so he doesn't think he did something wrong.



Corn

And the practical burden was real. Multiple sachets a day, and it delays or reduces absorption of a long list of drugs. Warfarin, thyroid replacement, tetracycline, digoxin. Which explains why he was spacing everything.



Herman

If someone had explained that up front, it might not have felt like his own failure. It's a demanding drug on a good day, and it's a demanding drug when you can find it.



Corn

What's the alternative landscape, then? Because there is one.



Herman

Ursodeoxycholic acid is the most interesting. A systematic review and meta-analysis published last year, fourteen studies, found UDCA significantly improved bile reflux gastritis outcomes versus conventional therapy. An earlier placebo-controlled trial showed



Herman (continued)

eighty percent symptomatic improvement with no endoscopic change. That gap between symptom improvement and no visible change is worth holding onto, because it tells you the benefit may be real and measurable to the patient while being invisible on a scope.



Corn

That's an honest framing. Symptom relief is a real outcome even when the tissue looks the same.



Herman

Sucralfate forms a protective coating and has been studied against post-cholecystectomy alkaline gastritis. Baclofen reduces lower esophageal sphincter relaxation, which limits how much can come up. And alginates have a specific role in weak-acid and non-acid reflux, reducing postprandial and nocturnal reflux, which is exactly the pattern he has. Alginate is the one you can buy over the counter, and it's the one a lot of clinicians never mention.



Corn

And that's where the research gets interesting, because there's a trial for the mechanical half that I don't think is well known.



Herman

Thoracoabdominal wall motion guided biofeedback. Forty-two patients with meal-triggered distension, randomized, placebo-controlled. Three sessions over four weeks, training them to move the chest down and the abdomen out.



Corn

The opposite of what feels natural when you're bloated.



Herman

Completely opposite. The instinct is to suck in. The training is to let go. And the numbers are striking. Intercostal activity fell eighty-two percent. Anterior abdominal wall activity rose ninety-seven percent. Clinical symptoms improved by about sixty-six percent. At six-month follow-up the benefit held. Out of the biofeedback group, one patient didn't respond.



Corn

One out of the treatment arm.



Herman

One. Now here's the caveat, and it's a real one. That trial excluded patients on neuromodulators and patients with psychiatric comorbidity. Daniel has already failed amitriptyline, so he wouldn't have been eligible. The clean result may not transfer to him directly.



Corn

So the honest version is: the protocol is cheap, it's teachable, it's low risk, and the evidence for it is strong in a narrower population than him.



Herman

Which is still a much better ask than "eat smaller meals." He's already tried that. Chest down, abdomen out, five minutes before and after meals is a specific instruction with a number attached.



Corn

Put the appointment questions together, then, because that's what he actually asked for.



Herman

Ask about HEMII-pH monitoring, and whether it's appropriate for the nighttime pattern. Ask whether the negative egg test was over-read, given what's known about emptying in functional dyspepsia. Ask whether impaired gastric accommodation has ever been assessed, since that hasn't been measured. Ask about prokinetics as a category, since he may never have been offered one. Ask about mirtazapine as an alternative to amitriptyline, and whether the drug was mismatched to his symptom cluster. Ask about ursodeoxycholic acid and sucralfate as bile reflux options, since both are supported and both are cheap. Ask about alginate for the nocturnal component. And ask whether thoracoabdominal biofeedback is available locally, or whether a physiotherapist could teach the protocol.



Corn

What he should not do is walk in and say he's tried everything and nothing works. He hasn't tried everything. He's tried one drug in one class badly matched to one symptom, and a piece of generic dietary advice.



Herman

One more thing about the night itself, and I don't want this to sound like blame. He walked around for an hour and that helped. Upright posture is doing real work there. It's worth saying that the hour of walking wasn't wasted suffering. It was the correct intervention.



Herman

He needs somebody to look at the two halves separately, and he needs to know the evidence base for the chemical half is thin. Mayo says so directly. That's a difficult thing to hear and a useful thing to know. The gap between "nothing has worked" and "the evidence is weak" is where this conversation should start. If he walks in expecting a clean answer, he'll be disappointed again. If he walks in with eight specific asks, the odds are much better.



Corn

I think the diaphragm piece is the one that surprised me most. The biofeedback trial changes what "treatment" looks like here.



Corn

Hold on.



Corn

Sixty-two percent. That number keeps sitting with me. This isn't a rare edge case, it's a predictable outcome of a routine surgery, and it gets treated as an obscure complaint.



Herman

The surgical consent conversation for cholecystectomy almost never mentions bile reflux. That's a separate failure and not one to chase right now.



Corn

Go on, because there's something in the structure of this that I want to say before we wrap.



Herman

The thing that strikes me is that his own description is more accurate than the labels he's been given. He says the system gets backed up and reacts with distension and regurgitation. That's accommodation failure followed by retrograde flow. He described the mechanism before anyone named it for him.



Corn

Which means the labels, helpful as they are, may have obscured the fact that there are two problems, not one.



Herman

A patient carrying four diagnostic names can easily assume they're all describing the same illness. They aren't.



Corn

I think that's the most useful thing to come out of this: the chemical problem from the surgery, and the somatic behavioral problem in how his diaphragm and abdominal wall move. Two problems, two sets of treatments, and they've been fused into one.



Corn

Anyway. The evidence base for the chemical half is thin, and Mayo will say so to your face, which is why approaching this as a set of specific asks is the way in. Expecting one clean answer is not.



Herman

Go in with the list.



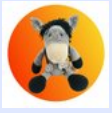
Corn

Our producer Hilbert Flumingtop puts this together every day. The producing, the desk, the timing, all of it.



Corn

This has been My Weird Prompts. If something in here sounded like your own situation, send it our way. You can email us at show at my weird prompts dot com.



Herman

We will be back soon, everyone.