Episode Book

EPISODE #5719

When Bile Burns Your Stomach Lining

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SYNOPSIS

After gallbladder removal, bile drips continuously into the stomach — and for some patients, it chemically burns the lining. This episode breaks down the mechanism of bile reflux gastritis, the longitudinal studies tracking gastritis rates after cholecystectomy, and why an empty stomach is the worst place for bile to sit. Then it gets practical: small frequent low-fat meals, what ursodeoxycholic acid and zinc-L-carnosine actually do (and don't do), and how to build meals that buffer bile while supporting mucosal repair.

TRANSCRIPT



Corn

A study tracked a hundred and thirty-three patients through gallbladder surgery and endoscoped them afterward. Before the operation, about a third had gastritis. Six months later, half did. The moderate-to-severe cases doubled.



Herman

And none of that is infection. That's the part people get wrong.



Corn

Which is exactly where Daniel's question starts. He's had this since his own gallbladder came out, and he's written in with something dense. His framing is that bile gastritis is common after cholecystectomy, it's extremely hard to treat, and the bile itself does the corrosive work. He wants it separated from other chemical gastritis, the NSAID kind, because the stressor here isn't an external drug you can just stop taking. It's a body fluid the liver keeps producing whether you want it to or not.



Herman

That's the whole hinge of it.



Corn

Then four questions. Why is skipping meals the worst thing you can do. Whether the folk wisdom around supplements and nutrients actually holds up. And he wants that integrated into recipes you'd actually enjoy, not swallowed as standalone remedies. Plus the constraint: the post-gallbladder population processes fat badly, so low fat isn't optional.



Herman

So the ask is a list of meals that hit four objectives at once.



Corn

Nutritious, tissue-repair building blocks for the stomach lining, something that buffers the bile, and low fat. He wants options, plural.



Herman

That's a real brief. Let's start with what bile gastritis actually is, because the mechanism is what makes every other answer make sense.



Corn

Go.



Herman

Also called alkaline reflux gastritis, biliary reflux gastritis, or duodenogastric reflux. It's a chemical gastropathy. Duodenal contents, chiefly bile acids plus pancreatic and intestinal secretions, reflux backward through the pylorus into the stomach. The injury is chemical. Not infectious, not autoimmune.



Corn

And the histology is what unifies it with the other chemical gastropathies.



Herman

Right. Foveolar hyperplasia, vascular congestion, lamina propria edema, prominent smooth muscle fibers, and very little inflammatory infiltrate. That's why it's called reactive rather than inflammatory. The tissue isn't under immune attack. It's being chemically burned.



Corn

Which is why the treatment logic is different from an ulcer.



Herman

Completely. An ulcer is often an infection or a drug, and you treat the cause. Here the cause is a normal body fluid arriving in the wrong place. You can't eradicate bile. You can only change how long it sits there and how hostile it is when it does.



Corn

Now, why the post-surgical population specifically. The gallbladder concentrates and stores bile and releases it in pulses when you eat. Remove it and bile drips continuously from the liver into the duodenum whether you're eating or not. No reservoir. More patulous pyloric dynamics, more bile available to reflux.



Corn

So the reservoir is gone, and the plumbing that used to gate it is looser.



Herman

Both. The gallbladder was a storage tank with a valve. Take the tank out and the supply line just runs. And the pylorus, the gate between the stomach and the duodenum, tends to be more open after surgery. So you've got more bile arriving and a looser gate holding it back.



Corn

Daniel's exact framing. A body fluid produced constantly whether the person wants it or not.



Herman

And the stakes aren't just comfort. Chronic bile reflux is linked to intestinal metaplasia and is a recognized long-term concern for gastric and esophageal carcinogenesis.



Corn

That's the sentence that should sit under everything else we say today.



Herman

It should. Which is why the reason this hits the post-surgical population so hard comes down to what bile does to the lining. Let's get into the mechanism.



Corn

How does it actually burn? Bile is a detergent. I assume that's the short version.



Herman

That's close to the short version. Soluble bile acids enter mucosal cells in their non-ionized, lipophilic form and accumulate up to about eight times the luminal concentration inside the cell. They injure cell membranes and tight junctions from the inside.



Corn

Eight times.



Herman

And the uptake is pH-dependent. It's worse at acidic pH. So the more acid in the stomach, the more efficiently bile gets into the cells that line it.



Corn

That is a counterintuitive piece of physiology.



Herman

It gets better. Bile acids also inhibit gastric carbonic anhydrase. That's an enzyme that matters for cytoprotection, part of how the lining defends itself. Cholate was the most potent inhibitor of any bile acid they tested.



Corn

So it doesn't just physically assault the lining. It disables one of the lining's own defense enzymes.



Herman

Exactly that. Two mechanisms running at once. The governing equation for injury is concentration times pH times mucosal exposure time.



Corn

Hold on exposure time.



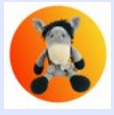
Herman

Hold onto it hard, because that's the variable the patient actually controls, and it sets up the entire fasting argument.



Corn

Before we get to that, the direct evidence in the post-cholecystectomy population. You mentioned the cohort at the top.



Herman

Guerra Cordeiro, Obesity Surgery, this year. A hundred and thirty-three patients followed longitudinally. Gastritis prevalence went from thirty-three point nine percent to fifty-one point four percent after cholecystectomy. Moderate-to-severe cases rose from eleven point nine to twenty-three percent. Both significant. And the authors don't hedge on the cause. They attribute it to enhanced enterohepatic bile circulation and increased alkaline reflux.



Corn

Then the earlier one. The eighty-five patients.



Herman

Atak and colleagues, twenty twelve. Eighty-five laparoscopic cholecystectomy patients endoscoped before surgery and again six months after. None of them had chemical gastritis before. Seven were diagnosed after. Bile in the fasting gastric fluid increased, endoscopic gastritis findings rose, and intestinal metaplasia went from six patients to twenty.



Corn

Six to twenty. That's the number that should alarm people.



Herman

And H. pylori colonization actually fell, from sixty-four to fifty-two. So this isn't an infection story. It's the exact opposite of one.



Corn

Then there's Shenouda.



Herman

Twenty eighteen, a bariatric cohort. Bile gastritis incidence was thirty percent, and gastric-aspirate bilirubin level correlated significantly with mucosal inflammation. Bilirubin above twenty milligrams per deciliter was associated with severe esophagitis, erosive gastritis, and metaplasia.



Corn

Which gives you a measurable marker. The concentration of bile in the stomach tracks the damage to the lining.



Herman

It does. Bile is not an abstraction. You can aspirate it and measure it.



Corn

Now the distinction Daniel drew. This versus NSAID chemical gastritis.



Herman

Both are chemical gastropathies. Same histologic signature. But the source of the stressor is fundamentally different. Bile is endogenous and continuous. NSAIDs are exogenous and intermittent. You cannot stop bile production the way you can stop a drug.



Corn

And that changes the treatment logic entirely.



Herman

It does. But here's the nuance that complicates the clean distinction, and it matters practically. Aspirin and NSAIDs themselves increase duodenogastric reflux. And bile salts markedly enhance NSAID-induced damage.



Corn

So they feed each other.



Herman

Bile plus acid plus NSAIDs are synergistic. Far more injury than either alone. A post-cholecystectomy patient taking NSAIDs is getting a double hit.



Corn

That's a strong practical argument. If you've had your gallbladder out, the NSAID question is not a casual one.



Herman

It isn't. And there's one more tradeoff worth flagging, because it's where standard practice gets muddy. PPIs help. They promote cellular restitution and reduce oxidative damage, and they're commonly used. But they raise gastric pH, and bile acid injury is worse at acidic pH.



Corn

So you're trading one thing for another.



Herman

The picture is mixed. Acid suppression is standard practice and it is not a clean fix for a chemical, non-acid injury. That's not an argument against PPIs. It's an argument against expecting them to solve this by themselves.



Corn

So that's the injury. The question Daniel actually asked is what you do about it. And the first answer is counterintuitive, which is that the thing you'd think of as resting your stomach is the thing making it worse.



Herman

Fasting. Which is the part he's already figured out on his own.



Corn

Walk through why, point by point, because it's worth being precise.



Herman

First. Bile flows continuously post-cholecystectomy. But food is what triggers duodenal transit and gastric emptying. An empty stomach has nothing to dilute or sweep the bile. So the bile sits, and exposure time lengthens, and exposure time is one of the determinants of injury.



Corn

Which is the variable he controls.



Herman

Second point. Fasting gastric fluid is precisely where bile is most detectable. That's what Atak found. They were aspirating fasting fluid and finding bile in it.



Corn

The stomach at rest is the bile reservoir.



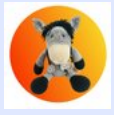
Herman

Third. Meals stimulate the mucosa's own defenses. Food and the resulting meal stimulus promote mucus and bicarbonate secretion, prostaglandin release, and mucosal cell renewal. Eating literally feeds the defense system.



Corn

The stomach is not a passive bag. It's actively maintaining itself, and it needs the signal of food to do it.



Herman

Fourth. Food physically buffers and dilutes refluxed bile and gives you a substrate to bind and adsorb bile acids. Fifth. Regular small meals keep the stomach from ever becoming a stagnant bile reservoir.



Corn

That's the entire argument in five beats. So the translation is small, frequent, low-fat meals and snacks. Never long fasts. Five to six mini-meals a day. Never skip breakfast.



Herman

And that's the single most defensible behavioral intervention in this whole episode. More defensible than any supplement we're about to discuss.



Corn

Then let's do the supplements honestly. Sorted by evidence tier, not by what's popular.



Herman

Start with the strongest pharmacologic option, which is ursodeoxycholic acid. UDCA. And the key thing to understand is that it works by changing the composition of refluxed bile, not by healing the mucosa directly.



Corn

Say more.



Herman

It raises the proportion of UDCA to about fifty percent of gastric bile acids, displacing the more toxic cholic and deoxycholic acids. So the bile that refluxes is a gentler bile.



Corn

And the evidence?



Herman

Stefaniwsky, nineteen eighty-five. A thousand milligrams a day profoundly reduced pain and nearly abolished nausea and vomiting. But the mucosa looked unchanged endoscopically and histologically.



Corn

So symptoms improved and the tissue didn't.



Herman

That's a real split, and it's worth sitting with. Then Jang, twenty twenty-four, a secondary analysis of the PEGASUS-D randomized trial. Five hundred and twenty-one patients post-gastrectomy. Three hundred milligrams of UDCA, not six hundred, significantly reduced bile reflux and gastritis grade at twelve months. Odds ratios of zero point four four and zero point five zero. But symptom differences were not significant.



Corn

So the dose matters and the symptom benefit isn't reliable.



Herman

Modest, dose-specific, and not a symptom cure. And it's prescription, not over the counter.



Corn

Next tier. The best-supported supplement for actual mucosal repair.



Herman

Zinc-L-carnosine. Polaprezinc, or PepZin GI. Mahmood and colleagues in Gut, two thousand seven. It stimulated epithelial migration and proliferation about threefold, reduced gastric injury by seventy-five percent and small-intestinal injury by fifty percent in animal models. And in a human randomized trial it prevented indomethacin-induced increase in gut permeability.



Corn

That's a real human endpoint.



Herman

It is. Hewlings and Kalman, twenty twenty, reviewed it and supported safety and efficacy for restoring the gastric lining. It's approved in Japan for gastric ulcers. Hiraishi and Nishiwaki both showed antioxidant activity, protecting gastric cells from hydrogen peroxide, ethanol, and monochloramine injury, and accelerating healing.



Corn

Caveat.



Herman

One veterinary randomized trial, Baan, twenty eleven, found no benefit versus placebo for aspirin injury in dogs. So it's not a slam dunk. But the human and mechanistic evidence is favorable, and it's the best-supported supplement in this category by a distance.



Corn

Next.



Herman

Sucralfate. This is a prescription mucosal protectant that directly adsorbs bile salts and forms a protective barrier over damaged mucosa. Graham, Digestive Diseases and Sciences, nineteen eighty-four. It's less effective than cholestyramine at bile binding and doesn't deplete bile salts at neutral pH, but it's the closest thing to a buffering drug you can get.



Corn

Worth a conversation with a gastroenterologist.



Herman

Worth a conversation. Then the bile acid sequestrants. Cholestyramine, colesevelam, colestipol. These target the downstream problem, bile acid diarrhea and fat processing, not gastric reflux directly. But they're highly relevant post-cholecystectomy. Seventy to ninety-six percent of chronic diarrhea patients with bile acid malabsorption respond to cholestyramine.



Corn

That's a huge response rate.



Herman

Barkun, Canadian Journal of Gastroenterology, twenty thirteen. And post-cholecystectomy diarrhea responds dramatically. Arlow, Sciarretta. Prescription again.



Corn

Now the folk wisdom tier, because Daniel specifically asked whether it holds up.



Herman

DGL, deglycyrrhizinated licorice, slippery elm, marshmallow root, aloe. There is no direct evidence for bile gastritis specifically. Mechanistically plausible, since mucilage coats and buffers, but anecdotal.



Corn

So reasonable to try, indefensible to present as proven.



Herman

That's the honest position. And general alkalinizing or bile-thinning supplements have no evidence base at all. I'd put them in the same bucket as the mucilaginous herbs, except with less mechanistic plausibility.



Corn

Then the recipes, which is what he actually asked for.



Herman

Four design principles. Buffer and adsorb bile. Build the lining. Low fat. Small and frequent. And the key insight is that soluble fiber and mucilaginous foods are doing the same job as cholestyramine, just in food form.



Corn

Soluble fiber is the food-based bile sequestrant.



Herman

That's the whole strategy in one line. Oats, barley, psyllium, okra, chia, flax, legumes, apples, pears, carrots. They bind bile acids in the gut.



Corn

And the second objective, building the lining.



Herman

Protein, especially glutamine- and arginine-rich foods, plus zinc and vitamins A, C, and E. Those support epithelial proliferation and repair. And low fat is the third constraint, not just because of the fat processing problem, but because fat triggers CCK and bile release. Small amounts of fat spread across meals beat a fat bolus every time.



Corn

Small and frequent is the fourth.



Herman

Five to six mini-meals. Never skip breakfast, never go long without eating.



Corn

Give me the list.



Herman

Breakfast first, and never skip it. Oatmeal with sliced banana and a spoon of chia or ground flax. The beta-glucan in the oats binds bile acids, banana is gentle and alkaline, chia adds mucilage. Low fat.



Corn

Next.



Herman

Low-fat Greek yogurt with cooked pear or applesauce. Protein plus pectin, which is another bile binder. Soft-cooked egg with plain toast and applesauce. Modest fat, good protein, bland.



Corn

Lunch and dinner.



Herman

Poached or baked skinless chicken or white fish with steamed carrots, zucchini, and a small portion of white rice or peeled potato. Lentil or split-pea soup, well-cooked, low-fat, no cream, with a little turmeric and ginger. Barley and vegetable soup with shredded chicken. Steamed or poached salmon, small portion, around three ounces, with quinoa and green beans. Tofu and soft-cooked vegetable stir-fry over rice with minimal oil, using broth or water instead. Mashed sweet potato or squash with a lean protein.



Corn

The sweet potato is doing double duty.



Herman

Soft, alkaline, and the vitamin A supports mucosal repair. That one's close to a perfect food for this condition.



Corn

Snacks. Because the whole point is preventing bile pooling between meals.



Herman

Applesauce, banana, melon. Plain rice cakes or a small bowl of oatmeal. A small handful of cooked and cooled potato. Low-fat yogurt or kefir. And psyllium in water if you tolerate it, which is the most direct food-based bile binder you can take.



Corn

Beverages.



Herman

Chamomile, ginger, or licorice-root tea. Note the licorice root can raise blood pressure, so DGL tea is the safer form. Warm water with a little honey. Avoid coffee, alcohol, strong citrus, and carbonation.



Corn

And the repair-plus-buffer list to keep in your head.



Herman

Soluble fiber from oats, barley, psyllium, legumes, okra, chia, flax, apples, pears, carrots. Lean protein from skinless poultry, white fish, egg whites, tofu, low-fat dairy, legumes. Zinc from lean meat, legumes, small amounts of pumpkin seeds, fortified cereals. Glutamine from eggs, lean meat, cabbage, beets, legumes, low-fat dairy. Vitamins A, C, and E from cooked carrots, squash, sweet potato, cooked spinach, bell peppers.



Corn

Foods to limit.



Herman

Fried and greasy foods, fatty cuts, full-fat dairy, cream sauces, butter, large fat loads in one sitting. Very spicy or very acidic meals. Alcohol. And NSAIDs, for the double-hit reason we covered.



Corn

Put a sample day together so it's concrete.



Herman

Breakfast, oatmeal with banana and chia. Mid-morning, applesauce or a small yogurt. Lunch, lentil soup with plain toast. Afternoon, a rice cake or a banana. Dinner, poached white fish with steamed carrots and zucchini and a small rice portion. Evening, chamomile tea and a few cooked potato slices if you're hungry.



Corn

That's a day where nothing is more than a couple of hours from the last thing, and no meal is a fat load.



Herman

It's a day designed around exposure time.



Hilbert

I spent about a year of my life pumping the stuff out of a rendering floor.



Corn

Say that again.



Hilbert

It came down a channel maybe eight inches wide and it moved. That was the whole job. Keep it moving and keep it diluted. If it sat, it set. Not hard like concrete. Hard like wax, and once it set you were chipping it off a steel floor with a scraper, and it took the paint with it.



Herman

That's the detergent quality doing exactly what we described to the lining.



Hilbert

It doesn't care what's under it. That's what I keep thinking about listening to you two. The equation you kept coming back to, concentration, acid, time. Time was the one we could do something about. Put water in it and keep pushing it along.



Corn

So the fasting point, from the other direction.



Hilbert

From the floor. Whatever sits, sits on living tissue in your case, and we were dealing with steel that at least didn't have to repair itself. My brother-in-law took the second pump when the company sold the building. He's still got it in a shed. Doesn't use it. Just didn't want to see it go.



Herman

And the chipping.



Hilbert

The chipping was the job, is the thing. Nobody tells you that when you take it. The pump is easy. The scraper is the job.



Corn

That's a physical picture you don't get from odds ratios.



Herman

The patient is managing a substance, not an abstraction. That's what the exposure-time argument actually means in practice.



Hilbert

Anyway. Your levels drifted a little on the last segment. I'll nudge it in the edit.



Corn

If you take one thing from this, it's that the one variable the patient controls is time. Not the bile, not the liver, not the surgery you already had. How long the bile sits there.



Herman

And the behavioral answer, small frequent low-fat meals, is also the most defensible thing in the episode. The supplement evidence is mostly mechanistic or small-trial, and the strongest options, UDCA and sucralfate, are prescription. The rest is a conversation with a gastroenterologist.



Corn

The metaplasia piece is the reason none of this is just about comfort. Chronic bile reflux is worth taking seriously as a long-term risk, not just a daily annoyance.



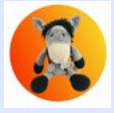
Herman

And the population is large and growing. Which means the meal strategy matters more, not less.



Corn

Review us if you've got a minute, it helps. Otherwise, my weird prompts dot com.



Herman

Or email us at show at my weird prompts dot com.



Corn

Thanks to our producer, Hilbert Flumingtop, who will be nudging the levels.



Herman

This has been My Weird Prompts. We'll be back soon.



Corn

Take care of your stomach.