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ASCITES: INITIAL MANAGEMENT

Ascites is the pathologic accumulation of fluid within the peritoneal cavity. It is the most common decompensating event in cirrhosis, with 5% to 10% of patients with compensated cirrhosis per year developing this complication.[1] The development of ascites is associated with reduction in 5-year survival from 80% to 30%.[2] Hyponatremia, low arterial pressure, impaired kidney function and low renal sodium excretion are independent predictors of mortality in cirrhosis with ascites.[3] Patients with cirrhosis who develop clinically significant ascites and related complications should be referred for liver transplant evaluation.[2] This article discusses ascites due to cirrhosis, however it should be noted that other causes of ascites include cancer, heart failure, tuberculosis, hemodialysis, pancreatic disease and other rare diseases.

The key steps in the pathogenesis of ascites in cirrhosis is the effective arterial underfilling as a result of splanchnic vasodilation which leads to the activation of sodium retaining systems, such as the renin-angiotensin-aldosterone system and sympathetic nervous system. This promotes positive fluid balance and extracellular fluid volume expansion. The resulting portal hypertension, leading to increased sinusoidal hydrostatic pressure along with architectural changes in hepatic sinusoids, contributes to the ascites formation.

Patients typically report progressive abdominal distension that may be painless or associated with abdominal discomfort, weight gain, early satiety and dyspnea resulting from fluid accumulation and increased abdominal pressure. Initial patient evaluation should include history, physical examination, abdominal ultrasound, laboratory assessment of liver and renal functions as well as an analysis of the ascitic fluid. Diagnostic paracentesis is indicated in all patients with new ascites and in those admitted to the hospital for any complication of cirrhosis. Ascitic fluid albumin and total protein concentration, cell count and culture should be always assessed.[2] The serum albumin-ascites gradient (SAAG) is calculated by subtracting the ascitic fluid albumin from the serum albumin in simultaneously obtained samples. A SAAG ≥1.1 g/ dL is highly suggestive of portal hypertension, usually caused by liver disease, with an accuracy of approximately 97%. SAAG <1.1 g/dL suggests other causes of ascites. A high ascitic fluid protein (>2.5 g/dL) supports a cardiac source for ascites. A neutrophil count above 250/mm3 denotes spontaneous bacterial peritonitis (SBP). Other tests, such as amylase, cytology, cholesterol, carcinoembryonic antigen or culture for mycobacteria should be guided by clinical presentation.

Ascites can be graded according to the amount of fluid accumulated in the abdominal cavity. Grade 1 is mild ascites only detectable by ultrasound examination. Grade 2 is moderate ascites, manifested by symmetrical distension of the abdomen. Grade 3 is large ascites leading to marked abdominal distension. No treatment is recommended for grade 1 ascites, as there is no evidence that it improves patient outcomes. Management of patients with Grade 2 and 3 ascites depends on the presence of an underlying cause of cirrhosis as well as the presence of superimposed complications (e.g. renal failure, hyponatremia and SBP). Moderate restriction of sodium intake (90mmol/ day, corresponding to 2g of salt) can be considered in uncomplicated grade 2 ascites. This can lead to the resolution of ascites in only around 10% of patients. In most patients, diuretic therapy is necessary. Aldosterone antagonists (e.g., spironolactone) and loop diuretics (e.g., furosemide, torsemide, bumetanide) are the mainstay of diuretic treatment.[4] The recommended initial dose of spironolactone is 100 mg/day, which can be progressively increased up to 400 mg/day. The recommended initial dose of furosemide is 40 mg/day and may be progressively increased to160 mg/day. During the first weeks of treatment patients should undergo frequent clinical and biochemical monitoring. Once ascites has largely resolved, the dose of diuretics should be reduced to the lowest effective dose. The treatment of choice for the management of patients with grade 3 ascites is large volume paracentesis (LVP) combined with albumin infusion. After LVP, patients should receive the minimum dose of diuretics necessary to prevent re-accumulation of ascites. Ascites that cannot be mobilized or recurs after LVP despite dietary sodium restriction and diuretic therapy is termed refractory ascites. Trans-jugular intrahepatic portosystemic shunts (TIPS) can be considered in well selected patients with refractory ascites. Patients with ascites may develop complications such as SBP, hyponatremia, acute kidney injury, umbilical hernias, and hepatic hydrothorax. The management of these patients is typically multidisciplinary.

Referral to a hepatologist is recommended for patients with chronic liver disease. At Eastern Pennsylvania Gastroenterology & Liver Specialists (EPGI), Dr. Adam Peyton, Dr. She-Yan Wong and I are board certified transplant hepatologists and offer expert care for patients with cirrhosis including those who have decompensated liver disease. We also partner with the University of Pennsylvania to provide access to liver transplantation.

IN THIS EDITION

BONNIE PATEK, DO

BEYOND POST-PARTUM PELVIC SYMPTOMS THAT AFFECT THE GASTROINTESTINAL TRACT

Pregnancy not only leads to major perinatal physical changes that can affect the GI tract, such as heart burn, nausea and constipation; but many women will experience significant changes long after the delivery of their bundle of joy that may not be apparent at the initial post-partum check with the Obstetrician. This leaves the burden to primary care providers to investigate seemingly vague rectal or pelvic pains associated with new and frustrating bowel habits like constipation or fecal incontinence. In addition, some women experience more troublesome symptoms that are often difficult to illicit due to embarrassment such as urinary incontinence, dyspareunia or altered perianal sensation.

Within the pelvic floor, the main muscles that are associated with assistance in bowel function include the levator ani muscle group (made up of the iliococcygeal, pubococcygeal and puborectalis muscles) and the anal sphincter complex.[1] As a result of pregnancy, increased progesterone causes global slowing of the GI tract leading to changes such as constipation and laxity of ligaments resulting in stretching of the pelvic floor in association with the growing uterus and increasing weight on the pelvic floor structures. Other causes of pelvic floor laxity include excessive weight gain during pregnancy (or not), long second phase of labor, advancing age, previous pelvic surgery or traumatic injuries to the pelvis. The stretching of the muscles can also lead to potential damage to nerves, most notably the pudendal nerve, causing pudendal neuralgia.1

Pudendal neuralgia/nerve entrapment has many different presentations. Other than vaginal delivery, which results in further stretching of the pelvic floor compared to C-section, other causes include previous pelvic surgery, trauma to the back of buttocks region, chronic constipation, excessive bicycling or prolonged sitting.2 Nerve entrapment is a more likely post-operative complication, such as after a hysterectomy. Presentation includes pain, numbness within the region of genitalia or rectum, sexual dysfunction, dyssynergic defecation or fecal incontinence. Because of the autonomic innervation of the pudendal nerve, occasional sympathetic reactions such as diaphoresis, pupil dilation or tachycardia may accompany pain. Diagnosis is challenging but Nantes Criteria can be helpful (pain in anatomical area of pudendal nerve, pain with sitting, pain does not wake patient from sleep, no sensory loss, relief with pudendal nerve block).[2] Therapy includes physical therapy (stretches like knee to chest or happy baby pose), neuromodulator medications (like TCA or gabapentin), avoidance of sitting, engaging in a more active lifestyle or pudendal nerve block/surgical decompression in the setting of nerve entrapment.

Functional anorectal disease includes pain in the anus/rectum that lasts for more than 30 minutes (most often described as a dull ache or pressure made worse with sitting), for at least the last 3 months with the start of symptoms occurring over 6 months ago (based on the Rome IV criteria).[3] This is felt to be related to chronic tension or spasm in the pelvic floor, with child birth being the most common precipitating factor along with pelvic surgery.[4] Exclusion of structural pathology must be done and a digital rectal exam is necessary to determine pain with posterior traction of the puborectalis muscle and asymmetric pain (generally more on the left side, for unclear reasons).[3]

Chronic proctalgia is recurring pain in the anal canal or rectum. It is a type of functional anorectal disease that can be subdivided into two categories: levator ani syndrome or unspecified functional anorectal pain (this is the same as levator ani syndrome with the exception that there is no tenderness during posterior placed traction on the puborectalis muscle during digital rectal exam). Chronic proctalgia can be associated with coccygodynia, despite the pain not being in the coccyx.[4] This syndrome should be entertained post-partum if a patient continues to have perianal pain with prolonged sitting, especially if there was a vaginal birth. A coccygeal fracture/bruise can occur from blunt trauma, a fall in the seated position or during vaginal childbirth. The initial pain is severe and sharp with sitting, can occur with bowel movements and sexual intercourse. Patients typically will require anti-inflammatory medication, offloading with the use of donut cushion, stool softeners to prevent constipation and it can take 8-12 weeks to heal.[4] However, if a patient continues with these symptoms after this initial period, consider the levator ani syndrome as the pelvic floor musculature that attaches posteriorly to the coccyx may become hypertrophied, thus continuously pulling on the coccyx and leading to continued issues with dyspareunia and constipation.

Treatments of anorectal functional pain syndromes include pelvic floor physical therapy that may utilize electro-galvanic stimulation, biofeedback training or stretching/yoga. Medications like muscle relaxants have been tried in addition to sitz baths and digital massage of the pelvic floor.[3]

One last thing to keep in mind is that these pelvic disorders are not solely female, post-partum issues. They can occur in women who were never pregnant and also in men. There are many other causes for these symptoms to develop, including obesity and a sedentary life style. Many of these symptoms are incredibly embarrassing and debilitating to patients who have likely been dealing with these issues for several years without relief. Having an open discussion about pelvic floor symptoms is the first step toward resolution of these issues.

ERIC NELLIS, MD

"WHAT SHOULD I EAT?": AN EVIDENCE-BASED GUIDE TO TALKING DIET WITH GERD PATIENTS

"It happens whenever I eat spicy food." Like many healthcare providers, I commonly hear this refrain from patients as they describe the factors that may exacerbate their heartburn symptoms. However, when it comes to making firm evidence-based recommendations with regard to diet and Gastro-Esophageal Reflux Disease (GERD), the literature is rather conflicting. GERD is exceedingly prevalent in the United States, with up to 44% of individuals reporting symptoms at least once monthly. For many patients, a dietary intervention may be one of the simplest and preferred approaches to disease management. A recent study in one hundred patients with GERD symptoms showed that 85% were able to identify a dietary trigger to their symptoms. Furthermore, elimination of said dietary triggers led to a marked reduction in symptoms (heartburn decreased from 93 to 44% and regurgitation from 72 to 28%). While by no means comprehensive in nature, below we will look to highlight studies assessing the association between GERD and a few of the more common foods that patients may ask about.

Coffee

A staple of the American diet, coffee is consumed by 64% of adult Americans with 11% having more than four cups per day. A 2013 review article of fifteen epidemiological studies noted a correlation between GERD and coffee intake in only five with the majority of studies finding no association. More recently a prospective trial in Taiwan looking at 1837 patients undergoing EGD showed no association between coffee intake and clinically symptomatic reflux or findings of erosive esophagitis on endoscopy. On the contrary, in the prospective Nurses' Health Study II looking at 48,308 women aged 42-62, the highest intake drinkers of coffee (>6 servings/day) had a hazard ratio of 1.34 (95% CI, 1.13-1.59) for GERD symptoms at least once weekly. When discussing coffee consumption it is also important to differentiate between caffeinated and decaffeinated intake. A double-blind study objectively comparing these options in seventeen patients with a pH monitor 3 hours after consumption showed 18% esophageal acid time in those consuming regular coffee compared to 3.1% exposure in the decaf group. For patients who do notice GERD symptoms associated with coffee intake, the suggestion of less excessive use or switching to decaf may provide some benefit although outright elimination of coffee from the diet seems to have less merit.

Tomato and Derivatives

Tomato and its derivative products can be a potent trigger for GERD symptoms. The high levels of organic acids (including citric and malic acids) in tomatoes produce an acidic environment which can worsen heartburn symptoms during regular physiologic relaxations of the lower esophageal sphincter. Survey data has shown GERD patients, in comparison with healthy individuals, to have a higher consumption of foods containing tomato. One cross-sectional study done in Saudi Arabia showed an odds ratio of 5.77 for symptomatic GERD among the group with frequent consumption of tomato-based foods. Unfortunately, there is less data is available on the physiologic impacts of tomato products on the esophagus nor prospective trials comparing exposure to non-exposure.

Chocolate

Chocolate is commonly cited by patients as a trigger for GERD. Manometric studies suggest that this association has some validity as it has been demonstrated that chocolate ingestion leads to a statistically significant decrease in lower esophageal sphincter pressure. Further, in a small case series of seven patients with typical GERD symptoms, the ingestion of chocolate in comparison with a meal of similar calories led to greater esophageal acid exposure time. Like tomato-based foods, prospective trials are lacking. Based on the available data, if patient's symptoms have a clear association with chocolate then removing it from the diet may be reasonable.

Alcohol

Alcohol increases the symptoms of GERD through multiple mechanisms including increased gastrin stimulation, decreased resting lower esophageal sphincter pressure with increased frequency of transient relaxation and impaired luminal motility. In healthy patients without GERD, modest alcohol intake has been shown to induce reflux symptoms and increase esophageal acid time. An epidemiologic study in China showed reflux symptoms in 43% of heavy alcohol users compared with only 16% of non-users. However, a more recent 2017 meta-analysis only showed a slightly higher but statistically insignificant pooled prevalence of GERD in alcohol users compared with non-users (20.3 vs. 18.1%). In addition, a prospective case-control study in which symptomatic GERD patients with alcohol use underwent six months of abstinence failed to show improvements in objective esophageal pH so the association is not fully clear.

In many of these cases, although prospective data is lacking, there is circumstantial evidence to suggest that eliminating specific dietary components can help to improve GERD. A good general approach for patients would be to keep a food diary to help identify obvious triggers and then attempt to reduce these precipitants. Beyond targeted dietary elimination for patients who are overweight or obese, subscribing to a low-fat, low-calorie diet to work towards a healthy weight might have the biggest impact on improving their GERD symptoms.

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Ascites: Initial Management

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