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Diagnosis: Recurrent Ascites and Lower Extremity Edema in a 67-year old Female

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Symptoms

"I feel so bloated." The 67-year-old female could feel herself getting hot as she tried to maneuver herself into an upright position. She had gone through this 3 months prior, and wondered why it was happening to her again. Her abdomen was now completely distended. She explained that her ascites had been drained 3 months ago, and she promised that she had been sober. Exasperated, she brought herself to her feet. She had no one in the room with her, and looked apologetic as her eyes scanned the room, eventually resting on a distant roof garden.

As her eyes fell to her legs, she explained how they had gradually ballooned over the past 12 weeks, despite her use of diuretics. With the swelling methodically moving up from her feet to her thighs, her slender frame was now completely hidden. She was exhausted; she said she had never felt so tired in her life. She could not get up quickly enough to reach the restroom, and the uneasy decision was made to place a Foley catheter. She looked down over her belly, unable even to see her feet, bewildered by her own condition.

Investigation

My resident assigned me this patient, emphasizing that it would be a great way to cement my understanding of hepatic pathophysiology, a textbook case of cirrhosis. My patient was a 67-year old female with a past medical history including cervical cancer (status post radiation) 15 years prior, with a resultant rectovaginal fistula, and a 40-year history of alcohol dependence. She was single, and gravida 0. She was a non-smoker, and denied any IV drug use in the past.

Her labs revealed a normocytic anemia; she had started folate and multivitamins during her last admission. This explained some of her fatigue, but was there some underlying chronic disease? The private attending following her planned a bone marrow biopsy. The medicine team did not work up the anemia right away, instead focusing their attention on the worsening ascites and lower extremity edema.

Records from the patient's last hospitalization included a CT scan demonstrating a cirrhotic liver. No liver biopsy had been performed. There was no documentation as to whether the cirrhosis was of viral or toxic etiology, or possibly both. GI was consulted, and serology was sent for hepatitis panels. Her liver function tests were abnormal, as expected. My resident was fairly certain that this was another routine case of cirrhotic decompensation triggered by alcoholic hepatitis, but her AST: ALT ratio (aspartate aminotransferase: alanine aminotransferase) was not 2:1. Moreover, I felt the patient had no reason to lie about her sobriety, since she had been forthcoming about her alcohol history. Hepatitis B and C virology returned negative.

The patient's serum albumin was low, at 2.0 grams per deciliter (normal being 3.5-5 g/

dl). She wasn't spilling any protein into her urine, ruling out a nephrotic syndrome. Her low albumin was most likely due to a combination of chronic malnutrition and alcoholic hepatitis. Renal was consulted and SPA (serum poor albumin) treatment was initiated to pull the escaping fluid back into her intravascular space. SPA was of negligible benefit, with the patient still in overt pain and discomfort. My resident and intern performed a therapeutic tap. The paracentesis removed 3 liters of ascitic fluid, alleviating, but not resolving, the patient's abdominal distention.

My resident assigned me with calculating the serum-ascites albumin gradient (SAAG), anticipating that it would support a cirrhotic etiology for the ascites. The SAAG was 1.0; by definition, a SAAG greater than or equal to 1.1 would have suggested portal-hypertension related ascites. However, the patient's SAAG was *less than* 1.1, meaning my patient's ascites were possibly nonportal-hypertension related. My resident held firm to his belief that the ascites were portal-hypertension related, noting that a SAAG of 1.0 could be considered borderline. Moreover, as cirrhosis was the cause of eighty-one percent of portal-hypertension related ascites, he reasoned that this was likely the case with our patient. However, because our patient had known cirrhosis, meaning an expected SAAG beyond 1.1, and her SAAG was still less than 1.1, I wondered if we should spend more time considering other etiologies for her ascites.

The physician's aphorism played in my mind, "If you hear hoof-beats, look for horses, not zebras," reminding me of the practice of pursuing more common, rather than exotic, diagnoses. However, as a medical student with a paucity of clinical experience and a relative excess of time, looking for zebras and following stringent SAAG cutoffs was more intuitive, and interesting, than looking for horses. Alternative diagnoses included peritonitis, pancreatitis, vasculitis, bowel obstruction or infarct, hypoalbuminemic states (nephrotic syndrome or a protein-losing enteropathy), or Meig's syndrome.

In the absence of any amylase or lipase elevations, I eliminated pancreatitis. Peritonitis did not fit, as the ascitic fluid showed PMNs, white blood cells indicative of acute infection, to be less than 250, and a white count less than 500. Moreover, she was afebrile, and had no abdominal tenderness. There was no evidence of any vasculites or bowel obstruction. This left hypoalbuminemia or Meig's syndrome. However, based on the failure of SPA treatment, it didn't seem as if her ascites could have been solely due to a hypoalbuminemic state. Meig's syndrome typically presents as a triad: ascites, pleural effusion, and ovarian tumor. A possible two out of three seemed reasonable, so I texted my resident, "What about Meig's?"

Resolution

Renal had re-initiated daily diuretics to drain the remaining fluid and lessen the patient's lower extremity edema, the standard furosemide 40 and spironolactone 100. The patient's private attending, an oncologist, had ordered a slew of tumor markers: AFP (alpha-fetoprotein), CEA (carcinoembryonic antigen), CA-125 (an antigen on nonmucinous ovarian cancers), and CA-19-9 (a monoclonal antibody against certain GI carcinomas). In reviewing her day's labs, her CA-125 had returned; it was elevated. This threw weight behind Meig's, or any gynecologic malignancy. As a transvaginal ultrasound was scheduled, I left the team for my next rotation.

A few days later, I re-visited my patient. She told me how horrible the transvaginal ultrasound had been, and said she had been told it was to test for ovarian cancer. She showed me the soaps and lotions a friend had brought her, sliding them under my nose, and pointed out the bouquet of cattails she had added to her windowsill garden of sunflowers. I reassured her, and wished her good luck. She kissed me on the cheek and thanked me. As I left her room, I did not know whether she had a benign fibroma or a malignant tumor, or whether the CA-125 was leading us astray. I wondered whether our path to diagnosis had been achieved, not knowing ultimately to what the hoof-beats belonged.

Five months later, I did a double take as I saw my former patient being admitted. From afar, I could see that her face had become unsettlingly gaunt, her belly more distended, and her legs unusually swollen juxtaposed against her twig-like arms. She was no longer my patient, and I was no longer on the medicine team. The medical record number that I had once typed by memory had receded from my mind. And unfortunately, I was unable to learn her final diagnosis before she was moved to another floor.