CLINICAL VIGNETTE

Post Laparoscopic Cholecystectomy Ascites: Rare Case Presentation and Discussion

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Case Report

A 44-year-old male with a past medical history of alcohol abuse with cirrhosis (sober for 2 years), chronic pancreatitis, ESRD secondary to IgA nephropathy on hemodialysis (3 times per week and awaiting kidney transplant), and treated latent tuberculosis had a laparoscopic cholecystectomy for cholelithiasis with recurrent biliary colic. The patient's gallbladder was removed to eliminate a possible source of future infections in anticipation of requiring immunosuppression once he received his renal transplant. His intra-operative course was unremarkable, and he was discharged home on post-operative day one in stable condition. One week post-operation, the patient was admitted to an outside hospital with severe sepsis and new spontaneous bacterial peritonitis. Peritoneal fluid samples showed WBC 1450 with PMN 68%, amylase 18, and albumin 1.7. He was treated with IV cefepime, metronidazole, and levofloxacin. HIDA scan did not reveal any biliary leakage. He was discharged to a nursing home for continued IV antibiotic treatment.

On the day of discharge to the nursing home, the patient presented to our facility for continued nausea, abdominal pain, and shortness of breath. He denied fevers, chills, and night sweats. Vital signs: afebrile, pulse 121, blood pressure 120/77, respiratory rate 20, and oxygen saturation 99% on room air. He was thin, pleasant, and in no acute distress on admission with mild crackles at lung bases and mildly distended abdomen with diffuse tenderness primarily at the right upper quadrant. There were no focal neurological deficits or pedal edema. Initial labs revealed a creatinine of 7.63, WBC 11.3 (PMN 68.8%, Lymphocytes 16.4%), total protein 4.7, albumin 1.6, total bilirubin 0.4, AST 39.9, ALT 23, Alk Phos 321, GGT 64.8, LDH 124, PT 15.9, PTT 33.6, and INR 1.5.

Hospital Course

Patient was treated for SBP and was initially started on ceftriaxone. Parenteral vancomycin and metronidazole were added for enterococcus and anaerobe coverage on day 3. He was transitioned to ciprofloxacin on day 21. Initial peritoneal fluid was a hazy straw color: albumin 1.3, amylase 16, glucose 104, LDH 87, protein 3.4, WBC 1390, RBC 1050, PMN 55%, lymphocytes 27%, monocytes 16%, and ADA 8.9. Peritoneal gram stain and culture were negative. The patient continued to have rapidly accumulating ascites requiring a paracentesis every 3-4 days. CT abdominal imaging was clear without masses; however, large ascites were present with bilateral pleural effusions without loculations or abscesses. The pancreas showed multiple calcifications and dilated ducts compatible with chronic pancreatitis. The kidneys were atrophic. Repeat liver panel was largely normal except for low albumin and mildly elevated INR (Protein 4.4, Albumin 1.5, TBili 0.5, AST 26.9, ALT 15, Alk Phos 196, GGT 37.2, LDH 92.6, PT 15, INR 1.4, PTT 34); peritoneal fluid collected on the same day revealed a SAAG score of <1 (Straw colored and cloudy, Alb 1.7, protein 3.3, LDH 92, WBC 194: PMN 28% and Lymphocytes 68%). IV albumin was administered to replete serum albumin starting on day 5. The differential for low SAAG score included: biliary leakage from recent surgery, infection (TB, fungal), chronic pancreatitis, serositis, nephrotic syndrome, protein-losing enteropathies, protein malnutrition, and malignancy (peritoneal cancer).

Infection workup was negative for peritoneal AFB smear and culture, fungal culture, bacterial culture, MTB PCR, ADA, HIV, serum crytococcal Ag, cocci Ab, histoplasmosis urine Ag, and hepatitis A, B and C.

Elevated ESR level of 18 and CRP of 171 prompted an autoimmune serositis/peritonitis evaluation; however, RF, ANA, smooth muscle Ab, and anti-mitochondria were all negative. Other tests including ceruplasmin and alpha-1 antitrypsin were within normal limits.

Therapeutic paracentesis removed approximately 4 L every 3 days (See Table 1). Peritoneal fluid WBCs were downtrending indicating that antibiotic therapy was effective and no clinical suspicion for peritonitis was present.

Table 1: Peritoneal Fluid.

Hospital Day	Peritoneal WBCs (/uL)	PMN (%)	Serum Albumin (g/dL)	Peritoneal Albumin (g/dL)	Fluid Removed
-4	1450	68%		1.7	Unknown
2	1390	55%		1.3	65mL
5	194	28%	1.5	1.7	65mL
7	1440	65%		1.2	4L
10	430	25%	1.6	1.1	4L
15	438	11%	2.5	2.1	6L
17	205	3%		1.8	4L
26	17	4%		2.0	4L

CA-125 was elevated at 354. Further imaging with FDG body PET/CT scan was unremarkable. CT pancreatic protocol showed no discrete pancreatic mass. MRCP was unrevealing and ERCP was not performed (LFTs: Alk phos 196, ALT 15, AST 26.9, TBili 0.5, Total protein 4.4, Alb 2.8, LDH 92.6, INR 1.7, and Cr 9.10). Abdominal ultrasound and doppler yielded the same impression as previous CT study revealed a cirrhotic appearing liver and a patent portal vein. Repeat HIDA scanned was again negative for biliary leak. Cardiac ECHO was unremarkable. Surgical consultation brought up the possibility that exploration may be needed to see if surgical material was left behind and contributing to the source of the infection not seen on imaging; however, diagnostic laparoscopy was not pursued given the significant associated peri-operative morbidity/mortality and eventual decreasing ascites accumulation. On day 28, the patient was able to tolerate weekly paracentesis and discharged on PO ciprofloxacin with close follow-up. He continues hemodialysis 3 times per week.

Discussion

This case was interesting for its broad differential diagnosis, which made it hard to conclude that post-surgical infection was the etiology despite the onset of SBP 1 week post-laparoscopic cholecystectomy. Moreover, the tremendously fast accumulating ascites (up to 1.5 L per day) that was culture negative with the low SAAG score (<1) and without peripheral edema was an unusual presentation. The patient's recent surgery, prior TB, alcohol abuse history, and ESRD secondary to IgA nephropathy required ruling out biliary leakage from recent surgery, infection (especially TB), chronic pancreatitis, serositis, nephrotic syndrome, protein-losing enteropathies, protein malnutrition and malignancy (peritoneal cancer) as possible causes of his recurrent, rapidly accumulating ascites.

The low SAAG score was not consistent with portal hypertension secondary to cirrhosis in which we would expect SAAG >1. Similarly, other protein losing conditions such as nephrotic syndrome, malnutrition, and enteropathies would also not fit. A low SAAG score could suggest TB, especially given the patient's prior history of TB. TB peritonitis in a patient with cirrhosis would generally have low SAAG but high ascitic fluid LDH >90.[1] However, cultures and labs were negative for TB. Given the ESRD and IgA nephropathy, nephrotic syndrome was determined to be unlikely given that the patient was only urinating ~200cc/day – not enough to have significant protein loss. Other diagnostic possibilities for low SAAG score were each ruled out with respective imaging and lab studies (pancreatitis, infectious, rheumatologic and malignancies). The ascitic fluid was within normal limits for bile, and HIDA scans were negative eliminating bile leak from the differential.

Nephrogenic ascites (also known as idiopathic dialysis ascites) was considered given that the patient had been on hemodialysis for 2 years and has decreased urine output. The clinical presentation and laboratory findings almost exactly replicate a previous nephrogenic ascites case report.[2] Nephrogenic ascites incidence is unknown, but it is uncommon with modern dialysis techniques and equipment. Vaguely, the diagnostic criteria includes straw colored ascites, high protein 2.5, and SAAG <0.9; however, it is a diagnosis of exclusion, which requires a peritoneal biopsy that was not performed in this patient.

By day 15, it became clearer that complication from the post-laparoscopic cholecystectomy was most likely responsible for the fast accumulating ascites. The culture negative ascitic fluid may be due to treatment with antibiotics prior to culture collection. The prolonged antibiotic course may have treated a peritonitis/serositis and been the reason for the eventual decrease in ascitic fluid accumulation. Prior reports of post laparoscopic ascites in cirrhotic patients (Child-Pugh class A and B) have been described, but the rate of ascitic fluid accumulation was not specified.[3]

We speculate that the patient developed SBP because he was immune compromised given his long-standing ESRD and cirrhosis.[4,5] Chronically high-serum ammonia despite regular hemodialysis could have impaired the patient's immune system to predispose him to iatrogenic infection. It is harder to speculate the cause for his fast accumulating ascites at an astounding rate of 1.5 L per day, but it is most likely exacerbated by a constellation of factors including the ESRD, cirrhosis, and chronic dialysis, which modulated the post-surgery infection.

Conclusion

This case is an unusual presentation of post laparoscopic cholecystectomy ascites with low SAAG score and fast accumulating ascitic fluid. The patient had a Child-Pugh score of 6 pre-op making him Class A (Alb 3.5, INR 1, TBili 0.7, no ascites, and no hepatic encephalopathy), thus he was a

candidate for the procedure. Previous literature suggests that laparoscopic cholecystectomy is safe and effective in Child-Pugh class A and B cirrhotic patients. However, it would appear that mild cirrhosis with ESRD requiring dialysis may be a contraindication to the surgical procedure given the compounded detrimental effect on the immune system.

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