

Case Report
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Small Bowel Obstruction with Post Operative Ascites

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A 25 year old male with a prior history of abdominal exploration for a gunshot wound presented with bilious emesis, abdominal pain and obstipation. Work up was consistent with small bowel obstruction and the patient did not respond to initial conservative management (Figure 1). At exploration multiple small bowel adhesions were present. The point of obstruction was the proximal ileum. A segmental resection of the area of obstruction was performed followed by a stapled side-to-side anastomosis. Several small bowel serosal tears were imbricated and the abdomen was closed using a running looped PDS suture. Staples were used for the skin. The pathology report of the resected specimen revealed extensive mucosal necrosis and adhesions. 4 days later there was serosanguinous drainage from the incision and the patient was returned to the operating room for local wound exploration. Although the fascial closure was intact overall there was some slackening of the PDS suture. This area was reinforced with an interrupted 1-0 vicryl suture. A drain was placed subcutaneously in the region of the umbilicus and the incision was closed. Post-operatively a large volume of clear fluid began to drain from the JP drain. His serum albumin dropped to 1.5. The ascitic fluid was evaluated and the creatinine of this fluid was 1.0. The serum creatinine was 0.9. The serum ascites albumin gradient was not determined. Drainage volume varied from 500 cc's to 1 liter per day. Intravenous hyperalimentation and intravenous albumin replacement was initiated. Eventually the patient was able to tolerate oral intake and the hyperalimentation was discontinued. A post-operative CT scan was done (Figure 2). No evidence of superior mesenteric venous or arterial obstruction from a thrombus was seen. Small bowel ileus was evident (Figure 3). Once the serum albumin increased to 2.7 the drainage stopped. The patient ambulated and began to tolerate soft food. The drain was removed and he was discharged to home in good condition.

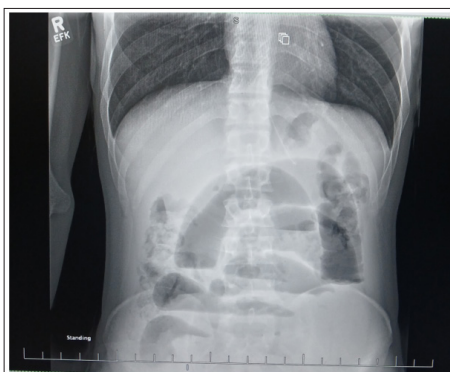

Figure 1

Figure 2

Figure 3
Discussion

The indication for abdominal exploration in the patient described was straightforward. Failure of a trial of non-operative therapy mandates exploration [1]. Large volume ascites is unusual after abdominal exploration for small bowel obstruction. The differential diagnosis should include cirrhosis, congestive heart failure, chylous ascites and iatrogenic injury to the ureter, bladder or bowel [2]. The fluid was clear (thus excluding chylous ascites) and the creatinine level of the fluid was not consistent with a urinoma. There was no clinical evidence of congestive heart failure and his liver was normal on operative examination and by imaging.

The serum ascites albumin gradient has been used to differentiate ascites caused by portal hypertension. A gradient greater than 1.1 is consistent with cirrhosis and associated portal hypertension. A gradient less than 1.1 is usually associated with inflammatory or malignant conditions [3]. Hypoalbuminemia remains a risk factor for morbidity after an abdominal exploration. It is not a benign condition as post-operative outcomes can be adversely impacted with albumin levels less than 3.4 [4]. In our case, the patient had local wound exploration because of serosanguinous drainage from a previously dry wound. This clinical finding should always raise the specter of fascial dehiscence [5]. Although overall intact, the fascial closure revealed some slackening and was reinforced. Even with these measures, large volume ascitic fluid drainage occurred. The serum albumin dropped to very low levels. Once albumin replacement achieved partial resolution to 2.7, the ascites resolved.

The physiologic driver of third space fluid includes Starling forces. Capillary hydrostatic pressure is the forward driving force and plasma oncotic pressure helps to maintain fluid in the capillary space. When compensatory mechanisms including lymphatic function are surmounted, third space fluid (ascites, pleural effusion, tissue edema) occurs. In the setting of cirrhosis, several theories have been proposed to explain the development of ascites. One involves lower circulating volume with sequestration of fluid in the mesentery. This stimulates the renin-angiotensin pathway resulting in fluid retention. There was no evidence of cirrhosis in the patient described but there is laboratory evidence in a mouse model to support hypoalbuminemia contributing to ascites via sodium and water retention [6]. Another novel theory suggests that protein is lost intraluminally from the small bowel because of a capillary leak secondary to inflammation [7].

In our patient partial correction of the serum albumin level was equivalent to restoring plasma oncotic pressure tilting the physiology away from third space fluid accumulation. In summary, we describe the case of a young man with small bowel obstruction secondary to adhesions who developed post-operative ascites in the setting of significant hypoalbuminemia. Once the albumin partially corrected (using albumin infusions and central hyperalimentation) the ascites stopped. He was discharged to home in good condition.

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