Delayed Hyperdense Ascites in a Peritoneal Dialysis Patient after Contrast Injection

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Ascites refers to the accumulation of fluid in the peritoneal cavity. Transudative ascites can be the result of fluid overload or it could have a cardiogenic cause, as reflected by computed tomography (CT) attenuation of -20 and 20 Hounsfield units (HU). When ascitic fluid contains protein, blood or white cells, a higher attenuation is recorded on CT [2]. High attenuation of peritoneal fluid on CT may signify a more serious cause [1]. Benedetti et al. [1] highlighted the phenomenon of delayed-contrast enhancement of ascitic fluid on CT when the cause of high attenuation may be attributed to benign delayed-contrast enhancement. Cooper and colleagues [2] showed a relationship between serum creatinine levels and the likelihood of delayed-contrast enhancement.

Duchenne and Becker muscular dystrophy accounts for 80% of all the muscular dystrophies and are inherited in an X-Linked pattern. Becker muscular dystrophy is considered less severe, with milder symptoms and later onset [3]. We present a case of marked benign delayed-contrast enhancement in a patient with Becker muscular dystrophy and peritoneal dialysis who presented with signs of intestinal obstruction.

PATIENT DESCRIPTION

Institutional review board (Helsinki Committee) approval was granted for this retrospective case report. A 36 year old male patient with Becker muscular dystrophy and an extensive medical history including a heart transplant and peritoneal dialysis for end-stage renal disease presented following 2 days of vomiting and constipation and 1 day of obstipation. A week prior to admission the patient presented at our emergency department (ER) with abdominal signs suggestive of spontaneous bacterial peritonitis (SBP). On examination the abdomen was distended, with tenderness noted in the left lower quadrant around the dialysis catheter entry site. An ascitic tap revealed a white cell count of 0.29 K/µl with a negative culture. SBP was suspected and the patient was treated on an outpatient basis with intraperitoneal vancomycin.

The patient returned to the ER 9 days following the suspected SBP. Examination revealed signs of bowel obstruction and a tender distended abdomen. An abdominal X-ray displayed markedly distended loops of small bowel suggestive of obstruction. In order to determine the cause of the suspected small bowel obstruction, a CT scan was performed with an iodine-based contrast agent administered intravenously (100 mg iohexol 350 mg/ml) and orally (iohexol 350 mg/ml 52 ml diluted in 2 L water). The CT showed markedly distended small bowel loops with air-fluid levels and collapsed distal small and large bowel loops. Oral contrast material propagated only to the proximal small bowel. A clear-cut transition zone could not be identified. A small pneumoperitoneum was also identified and was attributed to peritoneal dialysis. Ascitic fluid was noted surrounding the liver and between bowel loops and had a water density with measured attenuation of -3.00 HU. Because no obvious site of obstruction was identified, laboratory tests demonstrated a normal serum white cell count of 5.49 K/µl. The patient was hemodynamically stable. A decision was made to treat the patient with intravenous ceftazidime 1.5 g bolus and IV cefemezin 2.0 g and he was admitted to the surgical ward for observation. Serum creatinine levels of 2.09 mg/dl were measured at admission.

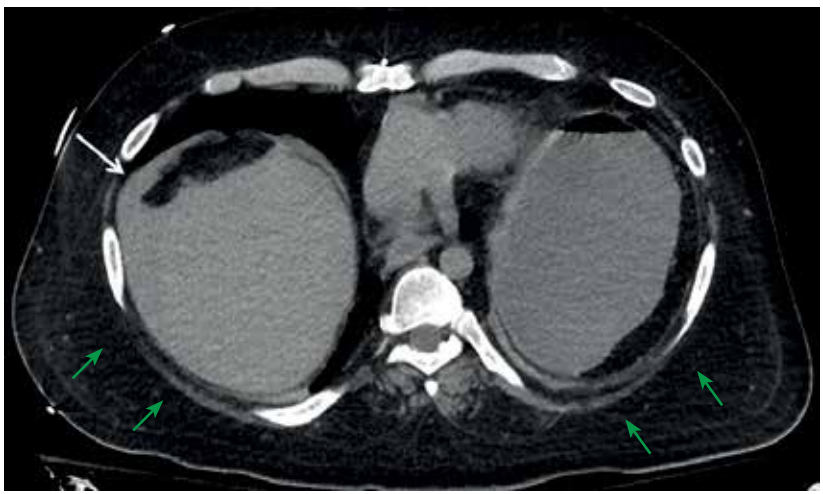
The following day the patient appeared well and improvement was noted on examination. A follow-up non-contrast CT was performed, yielding particularly interesting findings. The small bowel was still distended, but to a lesser extent; the ascitic fluid was now markedly hyperdense, with attenuation of 77 HU, almost indistinguishable from that of the liver and spleen [Figure 1]. Given the high attenuation of ascitic fluid, the question was raised that perhaps the patient had suffered intestinal perforation leading to leakage of oral contrast into the peritoneal cavity; however, this did not fit with the patient's clinical appearance. The management protocol was therefore continued. The patient showed significant improvement and was discharged after 7 days of observation.

The patient presented 13 months later with serious pneumonia, which was unfortunately complicated by sepsis leading to his death.

COMMENT

Although delayed-contrast enhancement was described previously by Cooper et al. in 1993 [2] and investigated by Benedetti et al. in 2009 [1], a formal definition has yet to be proposed. The presented case highlights an array of pathological processes that may have contributed to the markedly

Figure 1. Axial CT image showing hyperdense fluid near the liver with a measured HU of 77 (white arrow), nearly indistinguishable from the liver. Notice also the extreme skeletal muscle atrophy (green arrows)



high attenuation of ascitic fluid following administration of contrast material. In accordance with the findings of Benedetti and co-authors [1], our patient had end-stage renal disease. Therefore, poor renal function may be highlighted as a risk factor for delayed-contrast enhancement.

While Cooper and colleagues [2] state that the exact mechanism of delayed-contrast enhancement remains unclear, they write, "the phenomenon probably results from increased vascular-peritoneal permeability." Examples have been proposed, such as peritoneal carcinomatosis and loculated ascites causing an increase in permeability [1]. Hammerman et al. [4] conducted a case series, presenting eight cases of delayed-contrast enhancement; pathologies included viral cirrhosis, metastatic colon carcinoma, metastatic ovarian carcinoma, complicated pancreatitis, metastatic lung carcinoma, and alcoholic cirrhosis. Delayed-contrast enhancement of ascites has also been demonstrated in liver metastasis [5].

The present case shows delayed enhancement of ascites to the magnitude of 80 HU taken 14 hours post-contrast administration. The suspected diagnosis of SBP in the week prior to admission may have led to diffuse inflammation of the peritoneum, thus damaging the peritoneal membrane and increasing vascular-peritoneal permeability. Another contributing factor may be the marked distension of the small bowel at presentation, postulated to be secondary to ileus caused by SBP, further affecting the vascular-peritoneal integrity in accordance to LaPlace's Law, which states that wall tension is proportional to the luminal diameter.

In conclusion, in the present case, both markedly delayed high attenuation of ascites and free peritoneal air were present due to the patient's end-stage renal disease. Peritoneal dialysis led to the free peritoneal air; and increased gut wall permeability, presumably caused by the combination of renal failure, SBP and markedly distended bowel loops, caused the hyper-attenuated fluid. As both hyper-attenuated fluid and pneumoperitoneum can be signs of life-threatening bowel perforation, clinicians should be aware of the delayed fluid hyper-attenuation mechanism, especially in patients undergoing peritoneal dialysis, as knowledge of this phenomenon can avert unnecessary surgery.

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