Delayed haemoperitoneum post-paracentesis: Still an enigma

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SUMMARY

Paracentesis or colloquially known as abdominal tapping is a bedside clinical procedure commonly performed to aid in the diagnosis of the underlying cause as well as to relieve symptoms attributable to ascites. In practice, therapeutic paracentesis for symptomatic ascites remains as one of the commonest indications for this procedure, principally undertaken among patients with decompensated liver cirrhosis. Despite the inherent coagulation aberrations among the later, paracentesis-associated haemorrhagic complications remains scarce especially for non-large volume paracentesis (LVP). In this vignette, we describe an unusual case of non-LVP associated delayed haemoperitoneum which occurred in a middle-aged gentleman with co-existing advanced chronic kidney disease and newly diagnosed liver cirrhosis. This report serves to remind the clinicians to be vigilant about such complication among patients with concomitant liver cirrhosis and advanced renal disease.

INTRODUCTION

Paracentesis is a common clinical procedure routinely undertaken to obtain a peritoneal fluid sample or to drain symptomatic ascites for both diagnostic or therapeutic purposes.[1] It is generally regarded as a safe and simple procedure that can be performed bedside with little risk of major complications.[2,3] Nevertheless, there has been anecdotal evidence to suggest that such procedure carries a remote possibility of causing haemorrhagic complication, with delayed haemoperitoneum representing the rarest form of complications.[4,5] In this report, we describe a patient with co-existing advanced chronic kidney disease (CKD) and newly diagnosed liver cirrhosis who developed delayed haemoperitoneum following its inaugural diagnostic and therapeutic paracentesis. To the best of our knowledge, this is the first case of haemoperitoneum induced by non-large volume paracentesis with a delayed onset.

CASE PRESENTATION

A 54-year-old man with underlying advanced CKD, type 2 diabetes mellitus, hypertension and dyslipidaemia was referred to emergency department (ED) from nephrology daycare clinic for uraemia and fluid overload due to advanced CKD. He reported progressive abdominal swelling for 3 months which was associated with bilateral lower limb swelling and orthopnea. At the presentation in ED, his BP was 187/102 mmHg, and heart rate was 71 beats/minute with peripheral saturation of 99% under room air. Physical examination revealed bilateral lung crepitations and clinical findings in keeping with gross ascites. Laboratory results demonstrated significant renal function derangement with urea and creatinine levels of 49.5 mmol/L and 907 µmol/L, respectively. Subsequently, he was admitted to a medical ward for urgent haemodialysis (HD) and paracentesis.

At day 4 of admission, therapeutic and diagnostic paracentesis was performed in a single attempt at a right lower quadrant of the abdomen, which successfully drained 2.4 L of straw-coloured peritoneal fluid. Before the procedure, he had an international normalised ratio (INR) of 1.37 with aPTT and PT times of 42.6 seconds and 18.3 seconds, respectively. On the other hand, full blood count demonstrated haemoglobin (Hb) of 7.4g/dL and platelet of 128×10^9/L.

Despite previous paracentesis and ultrafiltration during haemodialysis, the ascites had reaccumulated rapidly, and examination showed gross ascites with prominent fluid thrill. Therapeutic paracentesis was repeated on day 6 of admission; however, it drained haemoserous fluid, and the procedure was abandoned after draining 500 ml of haemoserous fluid due to concern of possible intraabdominal injury. Nevertheless, reevaluation with bedside ultrasound (USG) refuted this as it showed gross ascites and large pockets of ascites. Furthermore, the size of the branula used was only 16G. During observation, he remained well and did not exhibit signs of haemodynamic instability or peritonism. In retrospect, in order to mitigate the risk of bleeding, heparin-free HD had been prescribed since the day before the inaugural paracentesis.

Alarmingly, his haemoglobin dropped acutely from 7.0 g/dL to 5.6 g/dL within a span of 3 days after the second paracentesis which yielded haemoserous fluid. Consequentially, he was transfused with two pints of packed cells. An urgent Contrast Enhanced Computed Tomography (CT) of the Abdomen and Pelvis was performed following the precipitous drop in haemoglobin. The CT revealed complex ascites with features consistent with liver cirrhosis. There was no evidence to suggest active bleeding. To substantiate our suspicion of probable haemoperitoneum, USG-guided paracentesis was repeated at day 13 of admission, which

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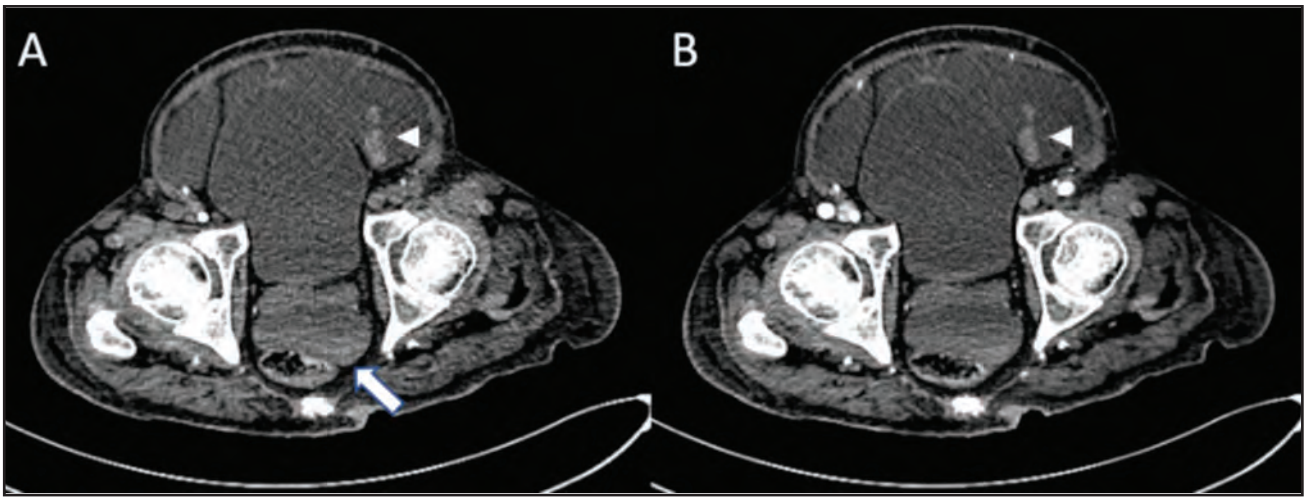

Fig. 1: Axial CT abdomen images in unenhanced (A) and arterial (B) phases at the level of the pelvis. These images demonstrate lobulated, non-enhancing hyperdensities at the left iliac fossa suggestive of blood clots (white arrowheads). Presence of hyperdense layering at the dependent region of vesicorectal pouch (white arrow) is suggestive of layering of blood products within the gross ascites. There was no evidence of active arterial bleed demonstrated in this study

drew bloody ascites and thus confirmed haemoperitoneum. In order to rule out intraabdominal bleeding, an emergent CT angiography (CTA) of the abdomen was performed, which did not show evidence of active bleeding, besides the presence of hyperintensities seen at the left iliac fossa suggestive of blood clots. In addition, there were no collateral intraabdominal varices seen (Figure 1).

In view of the findings of haemoperitoneum as well as the presence of liver cirrhosis, he was referred to the surgical team and hepatology team for co-management. To note, the Child-Pugh Score was B (8) with Model for End-Stage Liver Disease (MELD) score of 24 on admission. As screening for viral hepatitis B and C, and subsequently, autoimmune panel were all negative, the cause of the liver cirrhosis was believed to be due to metabolic associated fatty liver disease (MAFLD). During the multidisciplinary discussion, it was decided that the haemoperitoneum would be drained via percutaneous pigtail. On day 15 of admission, a peritoneal pigtail catheter was inserted under USG guidance over the largest pool area at a right lower quadrant, which drained bloody ascites fluid. A total of 15 L ascitic fluid was drained over 2 weeks, and human albumin replacement was given to prevent post-paracentesis hypotension. Later, the peritoneal fluid investigation and tumour markers ruled out suspicion of malignancy. A low serum-ascites albumin gradient (SAAG) of 0.8 g/dL, together with a low ascitic protein of 2.4 g/dL could suggest a renal predominant cause of the ascites. The pigtail catheter was removed on day 29 of admission, and he was discharged well the following day. At the time of writing, he remained well and had been undergoing regular haemodialysis. To note, there was no subsequent admission for symptomatic ascites in the last 3 months after discharge.

DISCUSSION

Paracentesis is the most common bedside procedure utilised by physicians in treating patients with ascites. This procedure is primarily carried out with the intent to relieve the symptoms of tense ascites or to ascertain the aetiology of the ascites, and both could be due to a multitude of pathologies.[1] Despite being predominantly performed among liver cirrhosis patients who commonly have abnormal coagulation parameters and thrombocytopaenia, the procedural complications are infrequently reported.[2] According to published literature, haemorrhagic complications related to paracentesis are extremely rare with an incidence rate of less than 1%.[2,3] Among the reported haemorrhagic complications were abdominal wall haematoma, pseudoaneurysm and haemoperitoneum, with later portending the worst survival outcomes.[4]

Delayed haemoperitoneum defined as onset of haemoperitoneum after 24 hours of paracentesis represents the rarest paracentesis-associated complications. As a rule of thumb, haemorrhagic transformation of peritoneal fluids on the following peritoneal tapping dictates a close monitoring for haemoperitoneum as it could be the precursor of an ongoing occult intraperitoneal bleed. In our case, the bloody ascitic fluid which clinched the diagnosis of haemoperitoneum happened 10 days after the first paracentesis in the absence of abdominal symptoms. In contrast, Arnold et al.[6] reported a case series of acute haemoperitoneum manifesting with shock and abdominal pain leading to the suspicion of such complications. The two extreme timeline variation for haemoperitoneum manifestation highlights both spectra of haemoperitoneum and underscores the perils of delayed onset haemoperitoneum which commonly lack overt symptoms till the late stage.

Several mechanisms have been theorised to explain the occurrence of delayed haemoperitoneum. The causal link has been commonly attributed to the abrupt splanchnic circulation decompression as a result of sudden shift in the intra-abdominal pressure after large-volume paracentesis

(LVP), defined as removal of more than 5 L of ascitic fluid. This would lead to a rise in the portosystemic blood flow via collaterals resulting in dilation and rupture of friable mesenteric varices.[6,7] Yet, our case did not corroborate with this theory as the inaugural peritoneal tapping only drained 2.4 L of ascitic fluid. In addition, the large pockets of ascites confirmed by ultrasound abate the likelihood of intra-abdominal organ or vessel puncture. This was further supported by the abdomen CTA findings, which did not identify any active bleeding organ or vessel. While the actual source of bleeding in our case remains an enigma, there is a common patient characteristic that coincides with several case reports. Co-existing liver cirrhosis and advanced renal disease elevate the risks of procedural-related bleeding complications in these reports.[4,8] To note, heparin-free haemodialysis had been prescribed prior to the inaugural paracentesis, which aims to mitigate the bleeding risk.

The diagnostic strategy to determine the aetiology of haemoperitoneum is contingent on the patient's haemodynamic status. In general, imaging in the forms of abdomen ultrasound or CT, and subsequently CTA if the former was negative, should be performed emergently following diagnostic paracentesis in order to exclude bleeding vessels or organ. In addition, it could also assist in planning the therapeutics subsequently after uncovering the cause of bleeding.[9] A multidisciplinary discussion should be made on the detection of haemoperitoneum, especially in the case of unstable patients. This is crucial because an exploratory laparotomy might be warranted to diagnose and to ligate the active bleeding vessels in the later. Yet, there is a possibility that the source of bleeding might not be identified during laparotomy. Under such circumstances, an unstable patient may decompensate further post-operation leading to hepatorenal syndrome or hepatic encephalopathy where liver transplantation might be required.[5,6] Considering all these, multidisciplinary evaluation is warranted and especially the risk and benefits of invasive approach must be carefully discussed with patients or the next-of-kin.

Due to the rarity of delayed haemoperitoneum post-paracentesis, most of the treatment approach is based of anecdotal evidence or expert opinion. Majority of the published cases ascribe the causes to intraabdominal variceal bleeding that are discovered via either abdominal CTA, exploratory laparotomy, transjugular intrahepatic portosystemic shunt or autopsy.[4-6,8] Therefore, all treatment strategies revolve around the ligation or embolisation of bleeding varices depending on the identification of the culprit varices. Haemoglobin and coagulopathy optimization, when necessary via judicious blood transfusion is also pivotal. In the case series published by Arnold et al, they identified sources of bleeding originated from either bleeding mesenteric varices, colon transversum varices, small bowel varices or variceal bleed at multiple sites. Furthermore, the fatality rate was 50% and the survivors (2/4) had been treated with variceal ligation or embolisation. It is noteworthy that 1 of the fatal cases succumbed due to disseminated intravascular coagulopathy (DIVC) within 1 week after the right hemicolectomy, which was performed to stop the colon transversum variceal bleeding.[6]

Clinicians should apprise themselves of the caveat of CTA examination in the detection of bleeding sites. According to a systematic review by Garcia-Blazquez, the overall sensitivity of CT angiography for detecting active acute gastrointestinal haemorrhage was 85.2 % (95 % CI 75.5 % to 91.5 %) influenced by the severity of bleeding.[10] In our case, we postulate that the source of bleeding most likely originated from venous bleeds, which are subtle and difficult to be detected via conventional CTA. This is supported by the absence of intraabdominal varix on the CT scan, as well as oesophageal or gastric varix during oesophagogastroduodenoscopy assessment done around the time of the incidence. Moreover, it is believed that the intraabdominal bleeding could have been stopped at the point of CT examination. This is evidenced by the Hb trend where the Hb stabilised following initial packed cells transfusion.

Lastly, exploratory laparotomy would be controversial due to the potential risk of hepatic decompensation or even a non-diagnostic surgical exploration as the bleeding could have ceased. Additionally, with a baseline coagulopathy and thrombocytopenia in cirrhotic patients coupled with a possibility of DIVC as alluded earlier, invasive procedures can be perilous. In light of this, a conservative approach was adopted in this case and the haemoperitoneum was drained via pigtail catheter inserted under ultrasound guidance in order to prevent abdominal compartment syndrome and peritonitis. The favourable 3 months survival at the time of writing provides evidence that conservative approach would be a viable treatment option in haemodynamically stable individuals with no active CT or clinical evidence of ongoing intraabdominal bleeding.

CONCLUSION

Delayed haemoperitoneum, a complication of paracentesis, is a substantial and challenging clinical problem, albeit its rarity. Patients with co-existing advanced renal disease and liver cirrhosis warrant active monitoring for such complication, as they could occur even in the event of non-large volume paracentesis. The management strategy should be contingent on patient's haemodynamic and premorbid status underpinned by multidisciplinary collaboration. A decisional balance would dictate either a conservative approach or high-risk exploratory laparotomy with the intent of ligating the bleeding varices if identifiable to be the best course of treatment.

ETHICAL APPROVAL

This case report has obtained approval from the National Medical Research Register (NMRR), Ministry of Health Malaysia: NMRR-23-00155-YPX

ACKNOWLEDGEMENTS

We would like to thank Director General of Health Malaysia for his permission to publish this article.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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