

Traumatic Aorto-Mesenteric-Portal Fistula: Percutaneous management

Case Report

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الناصور الأبهري المساريقي الباطني: العلاج عن طريق الجلد تقرير حالة

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الملخص: بعد شهرين من جرح قاطع في البطن جاء الى معهد كل الهند للعلوم الطبية رجل عمره 35 سنة يعاني من تقيؤ دموي وصدمة مع فرط ضغط الدم الباطني. التصوير المقطعي المحوسب للبطن وتصوير الأوعية البطنية كشفت ناسورا كبيرا يربط بين أم دم كاذبة في الأبهري البطني و فرع من الوريد المساريقي العلوي. اغلق الناسور عن طريق الجلد حيث تم تثبيت الضغط الدموي. ومن ثم إجراء عملية نهائية لاستئصال الناسور واصلاح العيب الأبهري.

مفتاح الكلمات: الأبهري. أم الدم. تصوير الأبهري. عمليات التدخل. تقرير حالة. الهند.

ABSTRACT Two months after a stab injury to the abdomen, a 35-year-old male presented at the All India Institute of Medical Sciences with haematemesis, shock and portal hypertension. Computed tomography of the abdomen and abdominal angiography revealed a large fistulous communication between an abdominal aortic pseudoaneurysm and a branch of the superior mesenteric vein. The fistula was occluded percutaneously, which allowed the patient to stabilise haemodynamically and, finally, to undergo a definitive surgical excision of the pseudoaneurysm and repair of the aortic defect.

Keywords: Aorta; Trauma; Aneurysm; Aortography; Interventional procedures; Case report; India.

POST-TRAUMATIC ABDOMINAL AORTIC aneurysms are relatively uncommon. We report a patient with post-traumatic pseudo-aneurysm of the abdominal aorta, which presented with life-threatening complications due to its communication with the portal venous system. This patient is important to report because of the unusual problems encountered during his management, especially during attempts at percutaneous embolisation of the fistula.

CASE REPORT

A 35-year-old man was stabbed in the right upper quadrant of the abdomen. An exploratory laparotomy

revealed a tear in the left lobe of the liver that was sutured, and a retroperitoneal haematoma in the upper abdomen which was left undisturbed. His postoperative course was uneventful and he was discharged on the tenth postoperative day. Five days later, he had a small amount of haematemesis which stopped spontaneously. After that, he had recurrent episodes of haematemesis once or twice a week. None of these was associated with hypotension, nor did he require hospitalisation or blood transfusions. Two months after the initial injury, he presented at the Department of Gastrointestinal Surgery and Liver Transplantation of the All India Institute of Medical Sciences with a history

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Table 1: Liver and renal function tests after embolisation

	Days after embolisation					
	0	1	2	5	12	15
Urea (mg/dl)	212	209	201	117	12	12
Serum creatinine (mg/dl)	2.3	3.2	3.4	1.7	0.6	0.6
Serum bilirubin (mg/dl)	0.8	0.8	0.7	0.7	0.7	0.7
Serum alkaline phosphatase (u/L)	332	330	412	504	653	290
Aspartate aminotransferase (u/L)	481	5730	2090	147	52	36
Alanine aminotransferase (u/L)	436	2080	1295	365	106	23

of progressively increasing abdominal distension for one week, decreased urine output for three days and a large bout of haematemesis just prior to admission.

At the time of examination he was anxious and anaemic. He was tachycardiac (140/minute), hypotensive (systolic 70mmHg), and tachypnoeic (36/minute). There were tense ascites and an upper abdominal bruit. All his peripheral pulses were palpable and equal in volume. A diagnostic paracentesis revealed straw-coloured fluid, and the nasogastric aspirate showed fresh blood.

His haemoglobin was 7 g/dl, blood urea 241 mg/dl, serum creatinine 3.5 mg/dl, serum sodium 129 mEq/dl and serum potassium 6.6 mEq/dl. The serum bilirubin was 0.7 mg/dl, alkaline phosphatase 313 U/L, serum aspartate aminotransferase/serum alanine aminotransferase (AST/ALT) 97/108 U/L [Table 1], and the prothrombin time was markedly prolonged (more than 60 seconds; control of 12 seconds). Blood gas analysis showed metabolic acidosis.

He was resuscitated with intravenous crystalloids and blood transfusions. Endoscopy showed congested and oozing gastric mucosa, but no gastro-oesophageal varices. A large volume paracentesis was done to improve the patient's respiratory status. Despite this, he remained oliguric, acidotic, tachypnoeic and haemodynamically unstable. The ascites re-accumulated rapidly (within hours). Administration of fresh frozen plasma improved his prothrombin time to within 5 seconds of the control value.

A contrast enhanced computed tomography scan showed massive ascites. There was a dilated, tortuous vascular channel anterior to the aorta at the level of the origin of the renal arteries. This structure (pseudoaneurysm) was in direct communication with a branch of the superior mesenteric vein (SMV). The draining superior mesenteric and portal veins were di-

lated (2.5cms diameter) and showed marked enhancement with contrast [Figure 1]. The liver was markedly hyperdense compared to the spleen, which showed little enhancement. The kidneys showed poor enhancement and the aorta below the site of abnormality had a smaller lumen, indicating a shunt from the aorta into the portal venous system, resulting in 'functional' portal hypertension.

An abdominal angiogram confirmed the presence

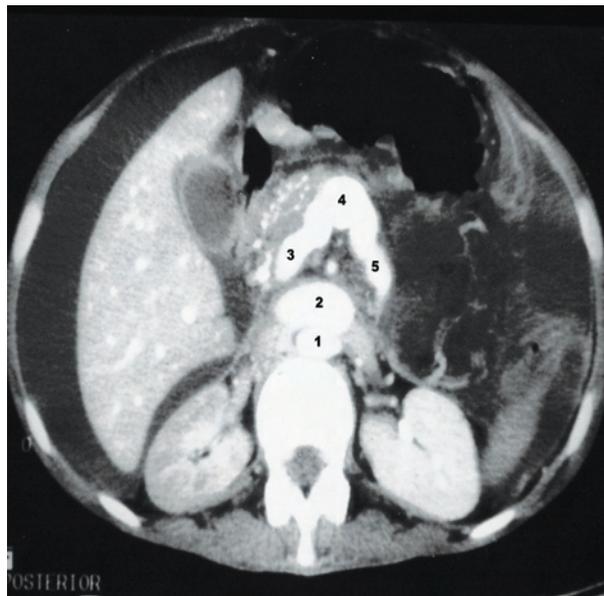


Figure 1: Contrast enhanced scan of the abdomen at the level of the renal vessels shows the following structures. [1] - aorta, [2] - a large, vascular channel (pseudoaneurysm) just anterior to the aorta, [3] - the draining branch of the SMV, [4] - dilated SMV, and [5] - retrograde flow into splenic vein. Note the presence of gross ascites and the differential enhancement of the liver and spleen. The punctate high-density structures in the head of the pancreas represent dilated venules secondary to high-pressure retrograde flow in the mesenteric-portal venous system

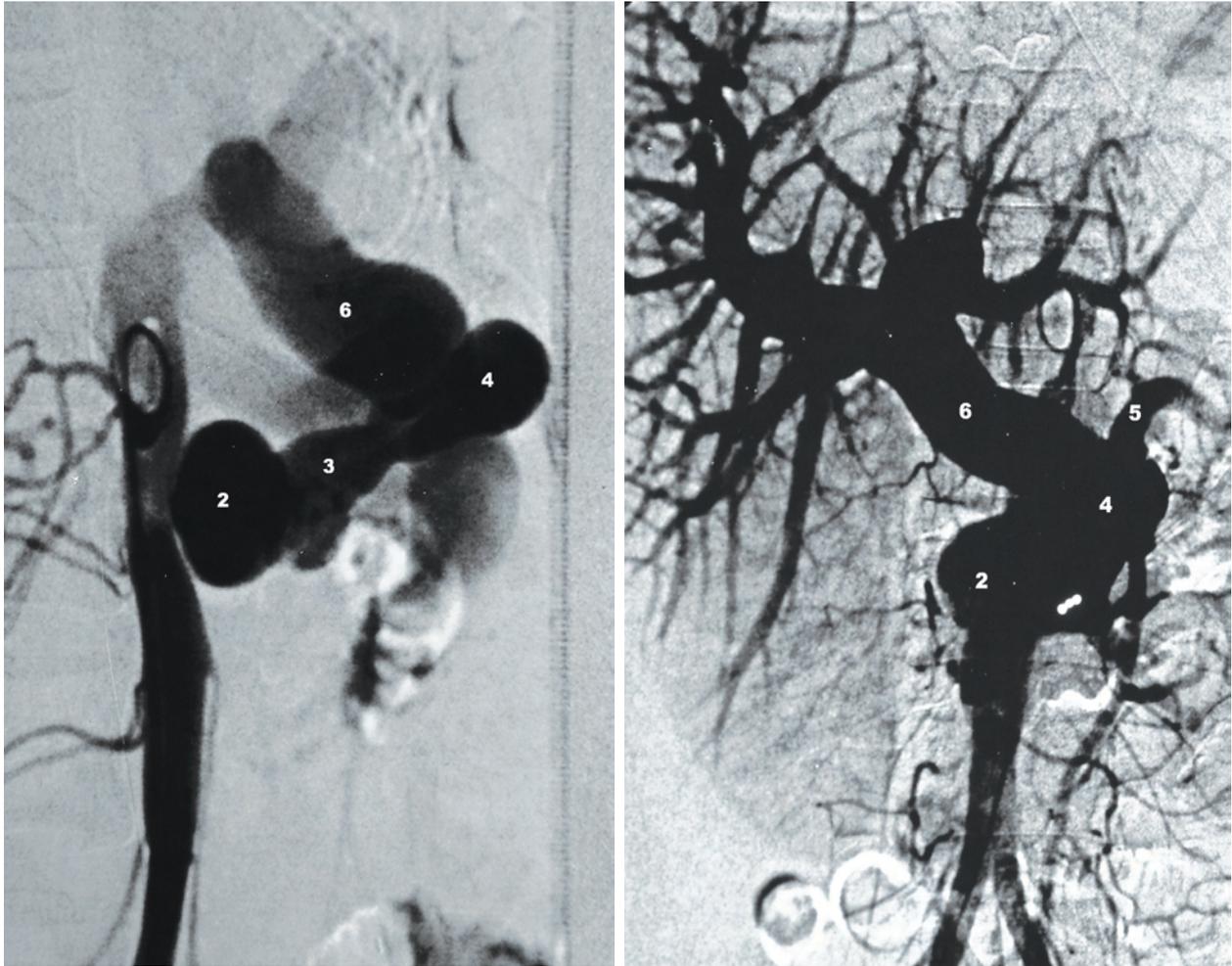


Figure 2: Abdominal aortogram, catheter tip at L1 vertebral level - (a) lateral, and (b) anteroposterior projections. Angiogram demonstrates a large fistulous communication between the aorta and a branch of the SMV, at the L2 vertebral level. Most of the injected contrast is diverted into the portal venous system, with hardly any flow into the renal arteries. The superior mesenteric and portal veins are grossly dilated. Numeric labelling in the figures indicates the same structures as in Fig. 1. ([6] - portal vein)

of an 8mm wide communication between the aorta (at the level of the origins of the renal arteries) and a large pseudoaneurysm. The latter, in turn, was draining through a 1cm wide defect into a dilated (1.5cm) branch of the SMV [Figures 2a and 2b].

As an emergency measure, it was decided to attempt an occlusion of the aortic opening of the pseudoaneurysm, with the possibility of encouraging thrombosis of the abnormal vascular channel. An 8mm diameter balloon catheter was placed across the aortic end of the pseudoaneurysm, and the balloon gently inflated until a waist was identified, to block the aortic opening temporarily [Figure 3], and with the hope of inducing thrombosis of the pseudoaneurysm. Over the next 6-8 hours the patient became normotensive, his urine output increased, he did not require further paracentesis

and his respiratory parameters improved.

A follow-up angiogram after 12 hours revealed a patent fistulous tract. An embolisation of the fistula was attempted using large diameter coils. Two 12mm diameter 3.5-turn platinum coils were placed at the junction of the branch vein and the SMV. This was followed by release of multiple steel coils into the pseudoaneurysm [Figure 4a]. This resulted in cessation of the blood flow through the fistula into the SMV [Figure 4b]. The abdominal bruit disappeared and the patient had a rapid clinical recovery with resolution of the ascites. The serum biochemistry also showed a gradual but sustained improvement [Table 1]. As the ascites disappeared, a small, ill-defined, non-expansile lump became palpable in the epigastric region.

Ten days later, the abdominal bruit reappeared.

Angiography demonstrated recurrence of a small amount of flow across the fistula and into the SMV. Release of more steel coils into the pseudoaneurysm resulted in cessation of flow into the SMV, but a small amount of flow persisted through the aortic opening into the pseudoaneurysm. Therefore, the patient was offered a surgical repair. He, however, refused surgery at that time due to social reasons. He was discharged from the hospital with normal renal and liver function tests.

He was readmitted 3 weeks later and had remained well during this period. The abdominal lump and bruit were still present but there was no ascites. The haemogram, renal and liver function tests were within normal limits. An ultrasound Doppler study confirmed the persistence of blood flow across the aortic opening into the pseudoaneurysm. However, the superior mesenteric and portal venous blood flow was normal with no evidence of arterial pulsations.

The patient underwent surgical exploration through a midline laparotomy. A 4 x 5cm size retroperitoneal mass was palpated. The mass was densely adherent to the SMV. The abnormal communication was successfully excised with repair of the aorta and the SMV.

Postoperatively, the patient had an uneventful recovery. At follow-up clinical examinations 2 and 4 months after the surgical procedure, his renal and liver functions were normal, and Doppler studies on both occasions did not demonstrate any recurrence of the fistula.

DISCUSSION

A number of patients with aortic injury exsanguinate prior to reaching a hospital. The mortality has been reported to be up to 70% even among those who survive long enough to undergo surgery. These injuries are often associated with other visceral and vascular injuries of the abdomen that may obscure the diagnosis. In some patients, a fistulous communication may occur between the aorta and a major vein, either as a result of trauma or spontaneous rupture of a pseudoaneurysm. This may control the aortic bleeding and might lead to the diagnosis being missed at the initial laparotomy.¹ Such patients usually survive the initial injury and may present weeks to months later, with manifestations of portal hypertension or congestive heart failure depending on the site of communication - the portal system or the vena cava.²

We came across only four previous reports in the



Figure 3: Angiographic balloon dilatation catheter inflated across the aortic opening. The site and size of the opening are identified by the waist of the balloon (arrow). The distal part of the balloon, tip and coiled guidewire are across the aortic opening within the pseudoaneurysm

literature of traumatic aorto-portal fistulae. In one patient, a preoperative angiogram demonstrated the abnormality, which was corrected surgically.³ Two patients presented with signs of portal hypertension, 14 and 18 months after the initial laparotomy. The aorto-portal fistulae in these patients were managed surgically,⁴ and with angiographic embolisation,⁵ respectively. The fourth patient had presented one year after the initial laparotomy, with a two months' history of melaena, intermittent haematemesis and signs of right heart failure, and was treated surgically.⁶

In our patient, the aortic injury was overlooked at the initial laparotomy. The recurrent gastrointestinal bleeding and the gradually progressive abdominal distension preceding his presentation at our institution suggested a progressive increase in the blood flow across the fistula leading to severe 'functional portal

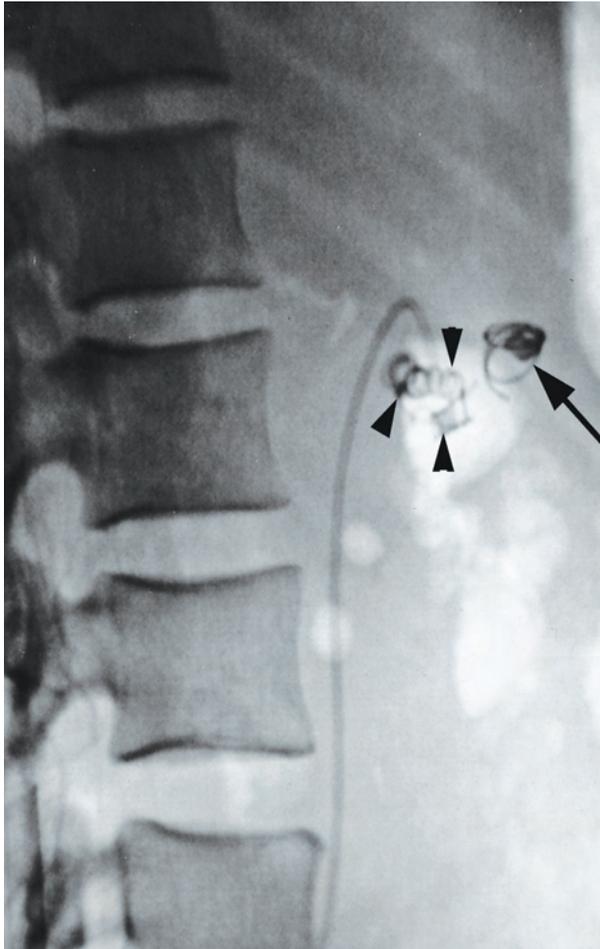


Figure 4a: Lateral projection shows large diameter platinum coils occluding the junction of the branch vein with the SMV (arrow), and multiple steel coils within the pseudoaneurysms (arrowheads)

hypertension, and prerenal azotaemia compounded by hypovolaemia due to multiple episodes of haematemesis. The gastrointestinal bleeding was the result of marked congestion of the gastric mucosa - a picture akin to severe portal hypertensive gastropathy. However, the absence of varices suggests that there was a rapid increase in the blood flow across the fistula over a few days prior to his presentation to us.

Angiographic management of the aorto-mesenteric-portal fistula presented an interesting challenge. The ideal technique for occlusion of a similar fistula in a more peripheral location would have been placement of a covered metallic stent graft. However, in this case, the site of the fistula at the level of renal arteries, and in close proximity to the origin of the superior mesenteric artery, precluded the use of a covered stent graft. The next option could have been a detachable balloon, but a balloon large enough to occlude the

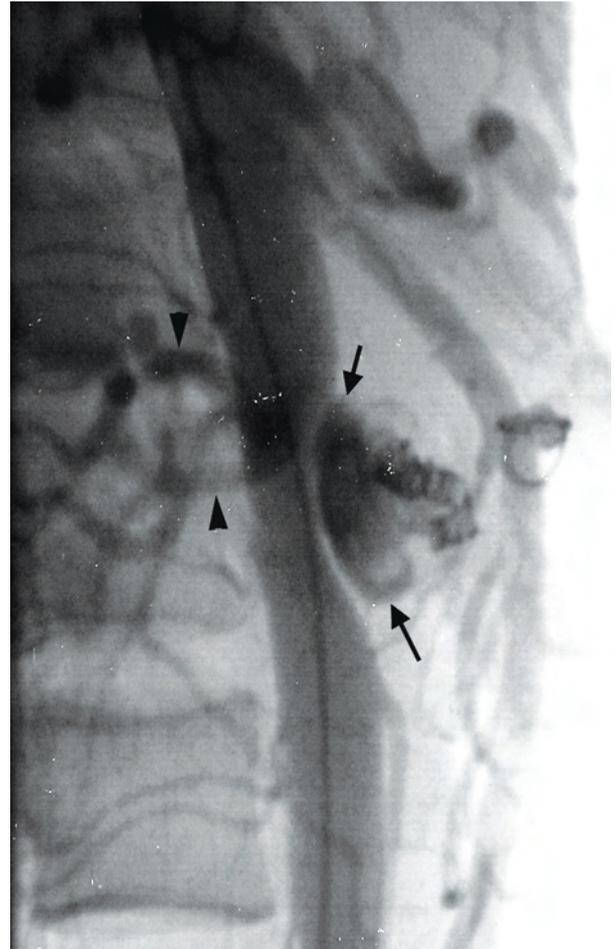


Figure 4b: Abdominal aortogram performed after embolisation of the fistula with steel coils, showed good flow in the distal aorta, with filling up of the major branches. The pseudoaneurysm (arrows) showed minimal filling from the aortic opening. There was no flow of contrast into the portal venous system. Good flow of contrast is now shown in both renal arteries (arrowheads)

4cm diameter pseudoaneurysm was not available. The presence of rapidly accumulating large volume ascites prevented pre-embolisation percutaneous transhepatic occlusion of the junction of the branch vein with the SMV. Therefore, to tide over the immediate crisis, we inflated a balloon catheter across the aortic opening of the fistula. This was successful in occluding the opening, and the patient's condition stabilised.

Subsequently, the pseudoaneurysm was successfully embolised with multiple metallic coils and the patient improved rapidly. However, the aortic end of the pseudoaneurysm could not be fully obliterated due to the rapid flow and its large size. This resulted in persistent blood flow into the pseudoaneurysm (but not into the mesenteric vein) with reappearance of a

bruit necessitating surgical repair. Though we did not attempt this, an injection of thrombin solution has been used successfully to effect complete thrombosis of pseudoaneurysms.⁷

In retrospect, two alternative techniques could have been employed for occlusion of the fistula. The first was the placement of an atrial septal defect occluder across the aortic opening; the potential drawbacks of this technique would have been the possibility of occluding the renal artery origins, and prevention of any subsequent surgical repair of the aortic defect. Theoretically, it should also have been possible to perform a transjugular transhepatic approach into the SMV (similar to a transjugular intrahepatic portosystemic stent shunt [TIPSS]) followed by placement of a large diameter covered stent across the opening of the branch vein into the SMV, prior to transaortic embolisation of the pseudoaneurysm; however, we did not have the expertise for performing TIPSS at our institution. Also, this approach would have resulted in a permanently dilated (2.5cm diameter) segment of the SMV (due to the implanted metallic stent), with the potential for thrombosis and infection.

After successful embolisation, the serum transaminases increased [Table 1] and then gradually normalised. This probably happened due to a relative ischaemia secondary to the disruption of arterial blood flow through the portal vein. The subsequent normalisation suggested that the hepatic arterial flow had returned to normal volume.

Unlike the previous case of aorto-portal fistula treated successfully by embolisation,⁵ in our patient angiographic embolisation did occlude the fistula, but could not block the large aortic opening of the pseudoaneurysm. However, it helped to stabilize the patient, restore normal liver function and allowed a delayed elective surgical procedure.

To conclude, post-traumatic aorto-portal fistula is a rare complication. This patient presented with secondary functional portal hypertension, and shock due to the steal phenomenon because of the large diameter, high-flow aorto-mesenteric-portal fistula. Unconventional percutaneous techniques had to be employed to occlude the fistula, which subsequently permitted elective surgery.

CONCLUSION

This report describes the rare condition of post-traumatic aorto-mesenteric-portal fistula with functional portal hypertension and its emergent radiological management as a bridge to definitive surgical therapy. The techniques involved in the successful radiological management were dictated by the location of the large fistulous communication in proximity to origins of important vascular structures, which could not be occluded, hence the difficulty encountered in using standard occlusion devices. This in turn prompted the use of unconventional equipment and procedures in an emergent setting.

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