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Embolisation of an enlarging paraumbilical vein aneurysm

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Abstract

Whilst recanalised Paraumbilical Veins (PUVs) are common pathways for decompression in patients with portal hypertension, aneurysmal change is rare, with only a handful of previously reported cases. A 47-year-old man with cirrhosis and portal hypertension was found to have an asymptomatic, but rapidly enlarging PV aneurysm. This was successfully treated with embolisation and this is the first time that this has been described.

Keywords: Paraumbilical vein; Aneurysm; Embolisation; Portal hypertension.

Introduction

Recanalised Paraumbilical Veins (PUVs) represent common pathways of decompression in patients with Portal Hypertension (PH). Drainage is from the left portal vein into the superior and inferior epigastric veins and/or into small subcutaneous vessels. Aneurysmal change of a PUV is a rare entity, having been recognised in only 5 cases previously [1-4].

Here we describe a case of a rapidly enlarging PUV aneurysm that was electively embolised in order to prevent rupture. To our knowledge, this is the first report of embolisation of a PUV aneurysm. Ethical approval is not required for publication. Consent was obtained from the patient for publication.

Case report

An 47-year-old man with alcohol-related chronic liver disease and PH presented with multiple right-sided rib fractures due to repeated falls. The patient was also encephalopathic on admission and this was successfully treated medically. The admission trauma Computed Tomogram (CT) showed a saccular PUV aneurysm measuring 4.7 cm (Figure 1). This had enlarged from 2.6 cm just 12 months ago.

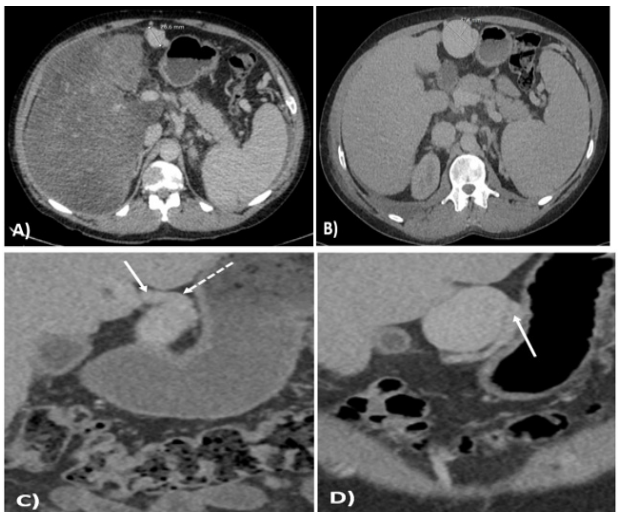

Figure 1: A) An axial portal venous phase computed tomogram (CT) from 12 months prior illustrating the paraumbilical vein (PUV) aneurysm (arrowed). B) The pre-treatment axial portal venous phase CT illustrating growth to 4.7 cm. C) A coronal reconstruction portal venous phase CT illustrating the afferent vein to the aneurysm from the left portal vein (solid arrow) and the narrow neck (dashed arrow). D) A more anterior coronal image showing the efferent vein (solid arrow).

The case was discussed at the hepatology Multidisciplinary Team (MDT) meeting and due to the rapid increase in size over a relatively short period of time, the decision was made to offer the patient embolisation. The patient had a recurrent right pleural effusion that was initially exudative and related to trauma, before becoming transudative and potentially due to hepatic hydrothorax. At the time of the MDT, the effusion was complex and septated and was being managed by the cardiothoracic surgeons. Transjugular Intrahepatic Portosystemic Shunt (TIPS) was not therefore not concurrently performed.

At the elective embolisation, 6 weeks after the trauma CT, ultrasound guidance was used to place an 11 cm 4 Fr sheath (Brite-Tip, Cordis, Miami Lakes, Fl, USA) into the intraperitoneal efferent vein just deep to the anterior abdominal wall. Direct portal pressure was 22 mmHg. Embolisation was performed across the neck of the aneurysm with multiple Ruby coils (Penumbra, Alameda, California, USA) (Figure 2). Haemostasis was achieved by manual compression.

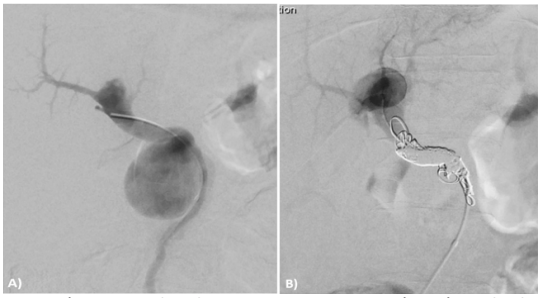

Figure 2: A) A Digital Subtraction Venogram (DSV) with the catheter in the afferent vein of the narrow-necked PUV aneurysm illustrating hepatofugal flow and reflux into small left intrahepatic portal vein branches. B) A DSV post coil placement with the catheter placed past the coil pack in the afferent vein. There is successful embolisation and improved left intrahepatic portal vein filling.

Follow-up ultrasound and CT confirmed successful embolisation of the aneurysm and he had an uncomplicated recovery. The right pleural effusion has decreased in size. At 6-month follow-up he has not experienced any further complications of PH and has had no further encephalopathic episodes.

Discussion

Whilst recanalised PUVs are common pathways of decompression in PH, aneurysmal change is rare. All 5 of the previously described cases were asymptomatic, incidental imaging findings and were managed conservatively [1-4]. One of the PUV aneurysms reported by Ohhira et al. was stable in size out to five years, but overall there is no meaningful long term data on which to base management decisions [4]. Furthermore, none of the previously cases describe such a rapid rate of enlargement. Given this brisk enlargement in size, relatively simple access to the aneurysm and the suitability of the narrow neck to embolisation, the decision to treat the aneurysm in our case was made.

The mechanism of PUV aneurysm formation is not known. There is a reported traumatic case, in a cirrhotic patient who had experienced blunt trauma to the abdomen, although the PUV aneurysm may well have been pre-existing [5]. Portal vein aneurysms are more widely recognised and can be congenital or acquired due to portal hypertension, pancreatitis or malignancy (the latter two causes should strictly be considered to cause pseudoaneurysms) [6]. Even then, it is unclear why only a small number of patients with portal hypertension develop portal venous aneurysms. All of the described cases of PUV aneurysms are in patients with chronic liver disease and portal hypertension. The fact that the PUVs and not the umbilical vein recanalise due to PHT is thought to be due to the fibrous constraints of the ligamentum teres surrounding the umbilical vein [7]. It seems highly likely that PUV aneurysms develop as a result of portal hypertension, presumably in the context of a predisposing factor that result in abnormality of the venous wall.

The management of portal vein aneurysms is controversial, but it is likely that the majority of cases can be managed conservatively, with intervention reserved for the 20% of cases that are symptomatic or complex (ruptured, thrombosed and rapidly expanding) [6]. Aside from active surveillance, other strategies in our case would have been creation of a TIPS in order to reverse and reduce the flow in the PUV aneurysm, as well as surgery to tie off the shunt. As with embolisation of other portosystemic shunts, it is important to consent and monitor for worsening of other manifestations of portal hypertension such as variceal bleeding or hepatic hydrothorax. None are proving problematic with our patient currently.

Conclusion

In conclusion, PUV aneurysms are rare entities, with only a handful of previously reported cases. Due to their rarity, there is a paucity of evidence on which to base management decisions. Due to rapid size expansion, embolisation was performed here and this the first reported case to be treated in this manner. It is however likely that many PUV aneurysms can be simply monitored, but the publication of long-term data is required to definitively justify this and MDT discussion on management is required.

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