

Case Report

Cystic artery pseudoaneurysm, a rare complication of duodenal ulcer

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ABSTRACT

In this case report, we would like to discuss a rare case of cystic artery pseudoaneurysm. Multiple aetiology has been described in literatures. One of the rarest is duodenal ulceration causing erosion into the cystic artery, leading to pseudoaneurysm formation. Here, we presented a case of ruptured cystic artery pseudoaneurysm with duodenal ulcer presenting as lower gastrointestinal bleeding and its subsequent management.

Keywords: Cystic artery aneurysm, Perforated duodenal ulcer, Visceral aneurysm

INTRODUCTION

Cystic artery pseudoaneurysm is a very rare condition with very few cases reported.^{1,2} Diagnosing it is still a challenge as it has multiple etiologies and there are no specific symptoms that would clearly suggest a cystic artery pseudoaneurysm. In this case study, we would like to share our findings and further discuss how we reached the diagnosis and what are the treatment options.

CASE REPORT

A 49-year-old lady presented with 9 days history of worsening right sided abdominal pain, associated with fever and malaise. She had no known medical illness or prior surgical intervention.

Clinically, she was septic looking, tachycardia, febrile and dehydrated. She had abdominal tenderness, mainly at right hypochondrium. Investigation revealed leukocytosis, raised CRP and normocytic normochromic anemia with hemoglobin level of 9.7 g/dl. Total bilirubin level was elevated with predominant direct bilirubin level of 57.4 u/l and amylase level of 499 u/l.

Contrast enhanced CT abdomen showed hemoperitoneum and haemobilia, with evidence of pooling of contrast on arterial phase within the gallbladder, from a cystic artery pseudoaneurysm. The findings were confirmed by mesentery angiogram, and the pseudoaneurysm were successfully embolized by coiling. Despite the successful angioembolization, the patient developed melena. Esophagogastroduodenoscopy revealed a visible vessel at first part of duodenum (D1) and blood clots within the lumen, with no evidence of active bleeding. Her hemoglobin level was static at day 2 of admission. Despite that, her condition worsened with worsening leukocytosis (38×10⁹ /l).

Reassessment CT abdomen was done with the suspicions of gallbladder infarct post embolization. It showed perforation at first part of duodenum with pneumoperitoneum. Gallbladder appeared emphysematous with surrounding rim enhancing collection. She underwent laparotomy, in which we identified a 3 cm perforated D1 ulcer, with perforated necrotic gallbladder. Otherwise, no active bleeding seen.

We performed cholecystectomy, primary repair of duodenal perforation, pyloric exclusion with gastrojejun

bypass (Billroth II). She developed another episode of melena at day 5 post surgery which successfully arrested by embolization. Mesenteric angiogram revealed active blush from branch of gastroduodenal artery. The patient made an uneventful recovery since then and was discharged with eradication therapy.

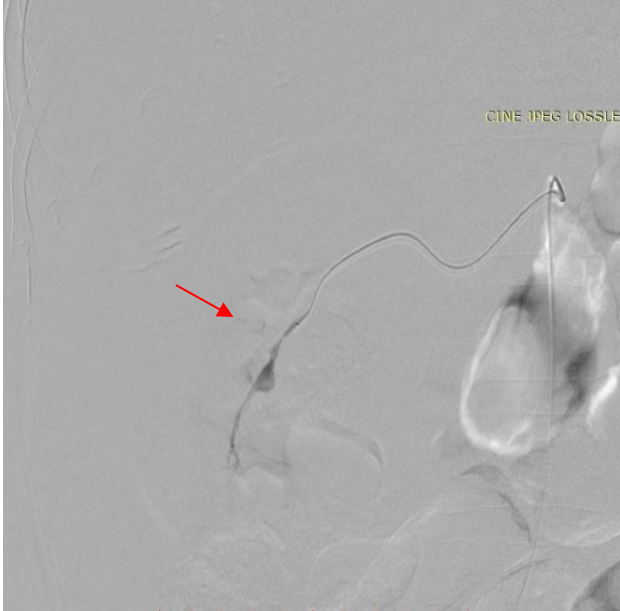


Figure 1: Cystic artery pseudoaneurysm pre-embolisation (red arrow).



Figure 2: Cystic artery pseudoaneurysm post-embolisation (yellow arrow).

DISCUSSION

Pseudoaneurysm of visceral vessels are extremely rare, where in most cases due to adjacent localised inflammation or direct trauma. In infrequent cases they may be due to vascular diseases or tumours.³⁻⁵ Cystic artery pseudoaneurysm secondary to duodenal ulcer are

caused by inflammation from the anterior part of duodenum towards the arterial wall, leading to the wall necrosis, subsequently leads to localised weakness of the arterial wall.

The pseudoaneurysms are prone to rupture, and they may bleed into the biliary system resulting in haemobilia, and patients may present with gastrointestinal bleeding. They may also present with right hypochondrium pain, and jaundice. Our case suggested that penetration of the cystic artery may occur with anterior or lateral post bulbar duodenal ulcers.

The recommended managements of cystic artery pseudoaneurysms are still controversial with scarce in existing clinical evidence. Previous published reports showed haemostasis can be achieved by emergent surgical intervention, or embolization with subsequent cholecystectomy.⁶ Because the cystic artery is an end artery, obstruction of the blood flow may lead to gallbladder infarction. Hence, subsequent cholecystectomy was suggested prior to embolization by certain literatures. However, as suggested by Mullen et al embolisation of the pseudoaneurysm without cholecystectomy was feasible, especially in high-risk patient was a viable option of treatment. They revealed that not all patients develop gallbladder infarct post embolisation, as there may be blood flow to gallbladder from other small proximal branches of the cystic artery.⁷ Patients may be monitored closely, and reimaging if presence of any clinical evidence to suggested gallbladder ischemia before proceeding with cholecystectomy. In the event of deterioration, definitive option of treatment will be cholecystectomy. In our presented case, the duodenal ulcer progressed into perforation despite the bleeding arrested with angioembolisation. Hence, we proceeded with surgical intervention-ulcer repair with cholecystectomy.

CONCLUSION

Cystic artery pseudoaneurysm of a rare clinical condition and henceforth the diagnosis is challenging. Earlier detection of the disease is very crucial and awareness of this potential diagnosis is vital. Option of treatment should be depending on patient's clinical condition. Due to high risk of rupture, angioembolisation of the cystic artery pseudoaneurysm is thought to be the preferred option of treatment.

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