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CASE REPORT

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Upper gastrointestinal bleeding due to hepatic artery pseudoaneurysm in a patient with systemic lupus erythematosus

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Abstract

Inflammation of the vessels can result in the development of arterial aneurysms in patients with systemic lupus erythematosus (SLE). We present case of a 46 years-old women with SLE who developed three episodes of upper gastrointestinal (GI) bleeding due to hepatic artery pseudoaneurysm. Upper GI endoscopy showed a pulsating submucosal protrusion in the duodenal bulb. Abdominal ultrasound showed cystic lesions located at the hylus of the liver. On day 3 of admission, she had massive hematemesis again. Within the 24 hours hemoglobin level dropped from 10,5 g/dl to 5,5 g/dl despite several blood transfusion. Angiography of the celiac artery showed a 2x3 cm aneurysm originating from the common hepatic artery and the bleeding arose from rupture of a common hepatic artery aneurysm. Patient transfered to surgery for treatment and performed ligation of artery ligation. She died on the 10th postoperative day.

Keywords: Systemic lupus erythematosus, hepatic artery pseudoaneurysm, upper gastrointestinal bleeding

Introduction

Systemic lupus erythematosus (SLE) is a connective tissue autoimmune disease, where vasculopathy is one of the typical symptoms. Lupus vasculopathy is usually seen in cutaneous vessels, in renal glomeruli, coronary and brain vessels, the brain, lung alveoli and less often in the gastrointestinal tract [1]. Involvement of the hepatic and splenic arteries is uncommon. Hepatic artery pseudoaneurysm can result in hemorrhage into the gastrointestinal tract when an abnormal communication is established between the vessel and the enteric part involve [2]. We report a case of upper gastrointestinal bleeding due to hepatic artery pseudoaneurysm by diagnosed angiography in a patient with SLE.

Case report

A 46 years-old women was admited to emergncy department with hematemesis and melena. One day earlier, she had had abrupt onset of abdominal pain. She described three times upper gastrointestinal bleeding with one week intervals in the last two month. She denied taking aspirine or non-steroidal anti-inflammatory drug. She had fifteen year history of SLE which had been treated with chloroquine and prednisolone for ten years. She had chronic renal failure which was undergoing haemodialysis for nine years. Upper gastrointestinal endoscopy on the day after hospitalization had showed the swollen of duodenal bulb but revealed no distinct source of bleeding. She was hemodynamically stable with hemoglobin 8.5 mg/dL, blood pressure 140/70 mmHg and pulse rate 100/min. During the physical examination the patient was faint, the abdomen was soft, slightly tender in the epigastrium. There was no rebound tenderness, hepatomegaly or splenomegaly. In laboratory evaluation the values of the biochemical parameters were as follows: AST: 14 IU/L, ALT: 9 IU/L, LDH: 197 IU/L, GGT: 60 IU/L, ALP: 238 IU/L, total bilirubin: 1.2 mg/dL, direct bilirubin: 0.6 mg/dL, BUN: 43 mg/dL, creatinine: 4.9 mg/dL, Na:139 mg/dL, K: 4.6 mg/dL, total protein: 4.6 gr/dL, albumin: 2.8 gr/dL, PT: 14 second, APTT: 24.9 second. In the complete blood count; hemoglobin was 8.2 gr/dL, hemotocrit 23.3 %, white blood cells 7200 /mm^3, and platelets 175000 /mm^3, erythrocytes sedimentation rate 92 mm/h. In further evaluation; hepatitis markers were negative. Elevated anti-double-stranded DNA (56 IU/mL, normal <7) and a positive antinuclear antibody titer (ANA) of 1:320, homogenous pattern.

Abdominal ultrasound and computed tomography showed cystic lesions located at the hilus of liver in our patient. On day 3 of admission, she had massive hematemesis again, endoscopic examination was repeated which showed a

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large amounth of fresh blood within the stomach, probably originating from the duodenal bulb. However; bleeding site could not be identified. Within the 24 hours hemoglobin level dropped from 10,5 g/dl to 5,5 g/dl despite several blood transfusion. Emergency angiogram of the celiac artery showed a 2x3 cm aneurysm originating from the common hepatic artery (Figure 1) and the bleeding arose from rupture of a common hepatic artery aneurysm.

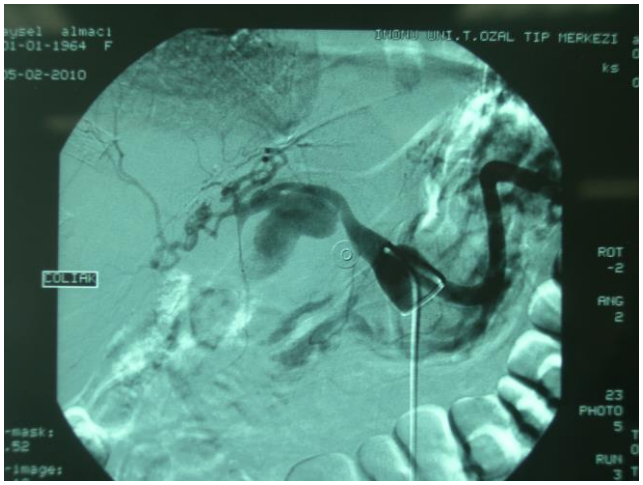

Figure 1: Selective angiography of the celiac axis shows a hepatic artery aneurysm

Patient transfered to surgery for treatment

Discussion

We report a patient with SLE presenting with multiple episodes of GI bleeding secondary to hepatic artery aneurysms. Pseudoaneurysm of the hepatic artery is a rare and potentially life threatening entity. Pseudoaneurysms of the hepatic artery are the second most common in the visceral artery pseudoaneurysms, [3]. Causes include abdominal trauma, liver and bilier surgery, and percutaneous interventional procedures involving the liver. Pancreatitis may also cause pseudoaneurysms of the hepatic artery. In our case; there was no pancreatitis findings and surgical procedure history. Our patient had 15-year history of SLE.

Visceral pseudoaneurysms usually present abdominal pain but there are patients having ruptured pseudoaneurysms without any clinical symptoms [4]. Other unusual presentations include upper gastrointestinal hemorrhage or obstructive jaundice. Our patient presented both

abdominal pain and repeated upper gastrointestinal bleeding but no jaundice. Ultrasound, computed tomography, and angiography are the methods of choice for aneurysm detection. Abdominal ultrasound showed cystic lesions located at the hylus of liver in our patient. An upper gastrointestinal endoscopy, in most cases, does not reveal any lesions in the stomach and duodenum. In our case, emergency upper gastrointestinal endoscopy showed a pulsating submucosal protrusion in the duodenal bulb.

A selective hepatic artery angiography is the diagnostic modality of choice when a pseudoaneurysm is suspected. It helps in making the diagnosis, provides anatomical details of the visceral arteries important for the preoperative planning and it can help to avoid the need to operate [2]. In our patient, emergency angiography of the celiac artery showed a 2x3 cm aneurysm originating from the common hepatic artery and the bleeding arose from rupture.

Treatment of a specific aneurysm depends on its location, the regional vascular anatomy, aetiology of the aneurysm and any associated or coexisting conditions. Common hepatic artery aneurysms can be treated by surgical ligation. This is possibly owing to the considerable collateral circulation available to the liver from the gastroduodenal right gastric arteries. Same cases may require resection of the involved segment of liver or transplantation. Embolization and endovascular stenting of the aneurysm are the preferred approaches to hepatic artery aneurysms [5]. Transcatheter arterial embolization has a success rate of 67%-100%, morbidity rate of 14%-25%, and mortality rate of 0%-14%. In these studies required re-embolization either for rebleeding or recanalization of the vessels in 37% of patients [6-8]. There are very few cases of pseudoaneurysm developed due to lupus that has been reported in the literature. And after surgery procudere, most of these patients died.

In conlusion, pseudoaneurysm of the hepatic artery is a rare but serious complication. Early diagnosis can be difficult even in symptomatic patients. Angiography and transarterial embolization are the means used for diagnosis and hemostasis. Operative ligation of the pseudoaneurysm remains, in some cases, the treatment of choice.

Conflict of Interest

The all authors declared no conflicts of interest involved in this study.

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