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Gastric Ulcer - A Cause of Portal Cavernoma and Upper Gastrointestinal Bleeding: Case Report

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Abstract

Gastric ulcers, with a long duration of the disease, can lead to an inflammatory process in the upper abdomen (supramesocolic floor), with repercussions on the surrounding structures. Such ulcers can penetrate the gastric wall, toward the pancreas and hepatic hilum, the inflammatory process can lead to splenic vein trombosis and teh appearance of a portal cavernoma. A complication of the portal cavernoma and the portal hypertension is the formation of esophageal varices. This paper reports the case of a 58 years old patient with multiple episodes of upper gastrointestinal bleeding, determined by both, esophageal varices and existing gastric ulcers. This patient was initially diagnosed with portal cavernoma and the esophageal varices were considered the cause of gastrointestinal bleeding. A spleno-renal shunt was proposed, but intraoperative it was found that this was not necessary because the portal vein was thrombosed and the bleeding was probably caused by the gastric lesions. We performed a distal spleno-pancreatectomy associated with a cuneiform resection of the gastric lession, as well as the resection of the hepatic tumor. The patient had a favorable postoperative outcome.

Keywords: Cavernoma; Ulcer; Varices; Bleeding

Introduction

It is known that upper gastrointestinal bleeding has many causes, esophageal and gastric lesions being the most frequent. Esophageal varices, the most common esophageal disease involved in the appearance of gastrointestinal bleeding, are due to portal hypertension, which is generated by different causes, including portal cavernoma [1,2]. It is also known that portal cavernoma can have many causes. One of these is represented by a chronic inflammatory process located near the portal vein. A long evolving posterior gastric ulcer can determine an inflammatory process in the adjacent area [3], affecting the pancreas, the splenic artery and vein and even the liver and the hepatic hilum. Although portal vein thrombosis is a rare entity in non-cirrhotic patients [4], this paper intends to present the case of a non-cirrhotic patient with esophageal varices secondary to portal cavernoma and gastric ulcer, who developed multiple episodes of gastrointestinal bleeding.

Case Report

We report the case of a 58 years old, non-cirrhotic patient, diagnosed 3 years ago with portal cavernoma, with multiple episodes of upper gastrointestinal bleeding, who underwent therapeutic endoscopical ligature of second degree esophageal varices. He was admitted to the Gastroenterology Clinic, due to a recurrence of the upper gastrointestinal bleeding, presenting hematemesis and melena. On admission, the patient was in obvious distress, but with normal cardiovascular parameters. He presented anemia, with a hemoglobin level of 7.8 g/dl. The other biochemical tests showed low iron levels (21 μg/dl), normal values of white blood cells (8200/μl), transaminases (ALT=15U/l, AST=18U/l), blood creatinine (0.9 mg/dl) and a serum lipase of 209U/l. The emergency upper gastrointestinal endoscopy showed multiple esophageal ulcerations, with two residual rings after ligation; the stomach contained clear fluid and the duodenal bulb had a superficial ulceration covered with fibrin. The abdominal ultrasound examination confirmed the presence of a portal cavernoma and showed an enlarged spleen, with a diameter of 140 mm, and the absence of ascites. Treatment was started and after an initially favorable evolution, the gastrointestinal bleeding recurred, with decreasing level of hemoglobin (7.3 g/dl), seven days after admission and intensive treatment. A new endoscopy was performed, showing small post-ligation varices and small ulcerations. The stomach contained bilious fluid and a blood clot, which was washed out. On the posterior surface of lesser curvature, a fibrin covered linear ulcer of 2 cm was identified. The mucosa covering the gastric body was "snake skin" like. Another fibrin covered ulcer of 2 mm was identified on the posterior surface of the duodenal bulb. It worth mentioning that the patient had no history of ulcerous disease and that the gastric lesion was never seen in previous endoscopical examinations. Given these facts, surgical treatment was indicated and the patient was referred to Surgical Department.

On admission in Surgery Clinic, the patient presented a hemoglobin level of 7.1 g/dl and an elevated level of serum amylase (526 U/l). Treatment was started and several units of blood were transfused. Due to patient's history of portal cavernoma, the opportunity of a spleno-renal shunt was discussed. Two days after admission in Surgical Department, massive upper gastrointestinal bleeding reoccurred and the hemoglobin level decreased to 4.9 g/dl. Given these facts, an emergency surgery was decided, making impossible further imagistic explorations (Doppler ultrasound, CT or MRI) in order to prepare spleno-renal shunt. For a proper operatory field, a bisubcostal incision was performed. We found a tumoral block in the upper abdomen, containing the posterior wall of the stomach, the pancreatic tail and the splenic hilum. Because the tumoral block was localized posterior to the stomach, clinical and ultrasound examination could not identify it. After dissecting the block, we identified a tumoral lesion generating in the gastric wall and

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penetrating the pancreatic tail. This lesion was not clearly described on endoscopic examination, but it may be assigned to "snake skin" like aspect of the mucosa in the gastric body. We also found a thrombosis of the splenic vein. The spleen was enlarged, measuring approximately 150 mm, with an irregular outline, and the veins tributary to the portal system were dilated, turgid, but permeable, excepting splenic vein. We identified the portal cavernoma in the hepatic hilum. The tumoral block invaded the liver segment 3, the rest of the liver being macroscopically normal. A distal splenopancreatectomy, at the level of the pancreatic penetration, was performed, associated with a cuneiform resection of the gastric lesion and a hepatic tumorectomy. It was considered that the last episodes of gastrointestinal bleeding were secondary to the gastric lesion and we considered that a spleno-renal shunt was unsuitable, due to the splenic vein thrombosis. The patient had a favorable outcome and was discharged 12 days after the surgery. A treatment consisting of Propanolol 2x20 mg/day and a proton pump inhibitor drug was prescribed at discharge.

Histopathological examinations showed: splenic parenchyma without tumoral lesions; lymph nodes with histiocytosis, without carcinoma metastasis; pancreatic parenchyma with marked interstitial inflammatory infiltrate; gastric wall with infiltration of granulocytes, eosinophiles, and limpho-plasmocytes; trasmural inflammatory necrosis and suppurative foci; no malignant lesions were found in the gastric specimen; liver parenchyma with biliary stasis and moderate chronic inflammatory infiltrate in the portal areas.

Discussions

Penetrating ulcers with chronic evolution can lead to inflammatory blocks, affecting adjacent structures [5]. Furthermore, the bleeding may be violent [6], due to arterial or venous fistulas, even in the case of small ulcers surfaces, without bleeding marks by the endoscopic examination [7]. Moreover, a chronic inflammatory process, as seen in chronic ulcers, can cause the appearance of a portal cavernoma [1,2], leading to portal hypertension, esophageal varices and gastrointestinal bleeding. In this particular case, a vicious circle appeared, leading to recurrent gastrointestinal bleeding. The endoscopy could not precisely identify the main cause of gastrointestinal bleeding (esophageal varices or gastric ulcer) and the final diagnosis was established during the surgical procedure made. The surgical procedure we performed was indicated because the gastric ulcer had a long evolution and the macroscopic appearance of the surrounding inflammatory block could not exclude a malignant lesion. In this particular case, we performed the excision of all the structured contained in the inflammatory block. The histopathological examination excluded a malignant lesion but confirmed a chronic inflammatory process in all the resected specimens. Although portosystemic shunt is indicated and can be performed with good results in noncirrhotic patients with portal hypertension [8], the procedure could not be performed in this particular case, because of the splenic vein thrombosis and the necessity of removing the entire tumoral block. Primary local risk factors for extrahepatic portal vein thrombosis are cirrhosis, hepatobiliary malignancies and pancreatitis [9] and the major systemic factors are myeloproliferave disorders [10], but none of these factors was identified in our patient. In this case, elevated levels of serum amylase were rather attributed to ulcerous penetration. Given the facts, portal vein thrombosis can only be attributed to splenic vein thrombosis, secondary to gastric lesion.

Conclusions

Gastric ulcers may have an unpredictable outcome, penetrating the surrounding structures and generating a chronic inflammatory process, that may lead to structural changes in the adjacent organs (portal cavernoma, splenic vein thrombosis, tumor-like lesions in the liver). The only clinical expression may be represented by gastrointestinal bleeding, being difficult to determine if gastric lesion or esophageal varices are its main cause. Endoscopy can diagnose and treat esophageal and gastric lesions causing upper gastrointestinal bleeding, but in some cases, associated lesions, minor at first glance, can lead to clinical manifestations and the definite diagnosis will be set during the surgical procedure.

Conflict of interest

Authors have no conflict of interest to disclose.

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