BANTI'S SYNDROME: A RARE CHRONIC PANCREATITIS COMPLICATION

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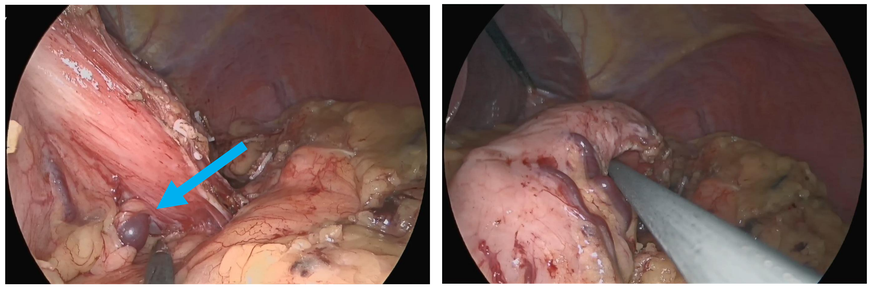
INTRODUCTION

Splenic vein thrombosis is a rare complication of chronic pancreatitis. It can lead to a portal hypertension of the splenic territory with splenomegaly, pancytopenia (hypersplenism), and esophageal varices. This syndrome was first discovered by Guido Banti in 1882 and is known as Banti's syndrome or idiopathic portal hypertension. Esophageal varices represent the main clinical presentation of this syndrome. The absence of cirrhosis is a notable finding in these cases.

CASE REPORT

A 46-year-old man from Brazil with a long history of alcohol consumption. The patient presented with asthenia, dizziness, and episodic large-volume hematemesis on hospital admission. Endoscopy revealed medium-sized varices in the gastric fundus, with red spots and signs of recent bleeding (IGV-1). A CT scan showed an unremarkable liver, splenomegaly, cysts in the tail of the pancreas; and prominence of venous vessels in the periesophageal region, greater and lesser curvature of the stomach. Laboratory tests revealed severe anemia, negative results for malaria and schistosomiasis, and normal liver function tests. All other causes of hypersplenism were ruled out, and idiopathic splenomegaly and portal hypertension of the splenic region and the diagnosis of Banti's syndrome was made. The patient underwent a laparoscopic azygos-portal disconnection. A splenectomy was not performed because of the presence of numerous pseudocysts in the pancreatic tail. Splenic artery ligation and an antireflux procedure (Dor technique) were performed. The patient presented an uneventful recovery, and he was discharged on the third day after the procedure.

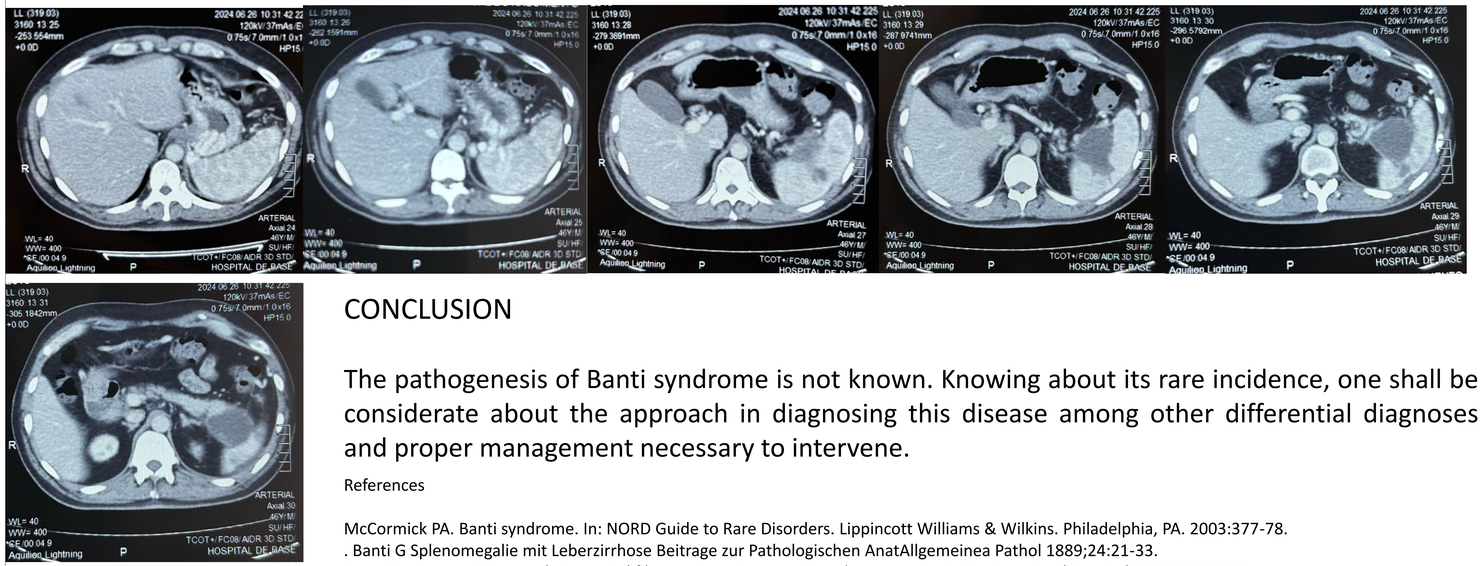
FIGURE 1 – INTRAOPERATIVE VIEW – LEFT GASTRIC VEIN; GREAT CURVATURE VEINS



DISCUSSION

Banti's syndrome (also known as Banti's disease, hypersplenism, idiopathic congestive splenomegaly, or IPH) is a chronic congestive enlargement of the spleen, resulting in blood cell destruction.
The major symptoms in patients with Banti's syndrome are gastrointestinal tract hemorrhage and splenomegaly, which were found in our patient. Hematemesis and melena, associated with the condition, can make the anemia more profound.
Banti's syndrome also causes noncirrhotic portal hypertension, often leading to ascites.
Beta-blockers are advised for portal hypertension prevention, and splenectomy may be needed for severe anemia and recurrent bleeding.
Endoscopic sclerotherapy and endoscopic variceal band ligation can be performed in the emergency setting.

FIGURE 2 – CT SCAN



CONCLUSION

The pathogenesis of Banti syndrome is not known. Knowing about its rare incidence, one shall be considerate about the approach in diagnosing this disease among other differential diagnoses and proper management necessary to intervene.

References

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