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Case Report

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A Rare Case of Portal Vein Thrombosis Due to Portal Vein Aneurysm. Case Report and the Review of the Literature

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ABSTRACT

A portal vein aneurysm (PVA) is an abnormal dilatation of the portal vein measuring more than 19 mm in patients with portal hypertension and more than 15 mm in individuals with normal portal venous pressure. PVA is rare accounting for only 0.06% to 3.0% of the visceral vessels' aneurysms. This case report is about a 20-year-old female who presented with portal vein (PV) and superior mesenteric vein (SMV) thrombosis with PVA. She was managed with anticoagulation. At 8 years of follow-up, she was symptom-free and did not have evidence of portal hypertension.

Keywords: Portal vein aneurysm, portal vein thrombosis, portal vein development

INTRODUCTION

Portal vein aneurysm (PVA) is an abnormal dilatation of the portal vein of more than 19 mm in patients with portal hypertension and more than 15 mm in patients with normal portal venous pressure (1). PVA is very rare accounting for only 0.06% to 3.0% of the visceral vessels aneurysms and it was first described in 1956 (2), (3), (4). Only a few cases are reported in the literature. On a Google search by using the keywords "portal vein aneurysms", and "case report", only 64 cases were found. This case report is on a patient who presented with portal vein (PV) and superior mesenteric vein (SMV) thrombosis with PVA.

CASE REPORT

A 20-year-old female experienced sudden onset abdominal pain in the past. A computed tomographic (CT) scan of the abdomen demonstrated a PVA with an acute PV and SMV vein thrombosis (Figure 1, Figure 2). The PVA was a saccular aneurysm at the PV and the splenic vein confluence. In addition, the SMV was distended. There were no features of chronic pancreatitis. There were no features of venous congestion or venous gangrene of the bowel. Thrombophilia screening tests were negative. Anticoagulation was started. The pain resolved with anticoagulation. She was tolerating oral feeding. Since the pain resolved and she was tolerating oral feeding, no further interventions were done. The patient was started on life-long oral anticoagulation. At 8 years of follow-up, she did not have abdominal pain. Her weight was maintained.

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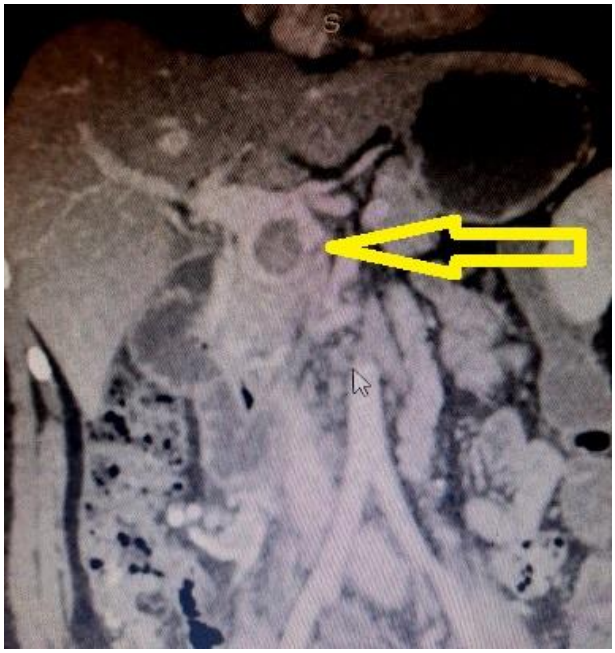

Figure 1: Computed tomographic (CT) scan showing portal vein aneurysm (Arrow) and thrombosis

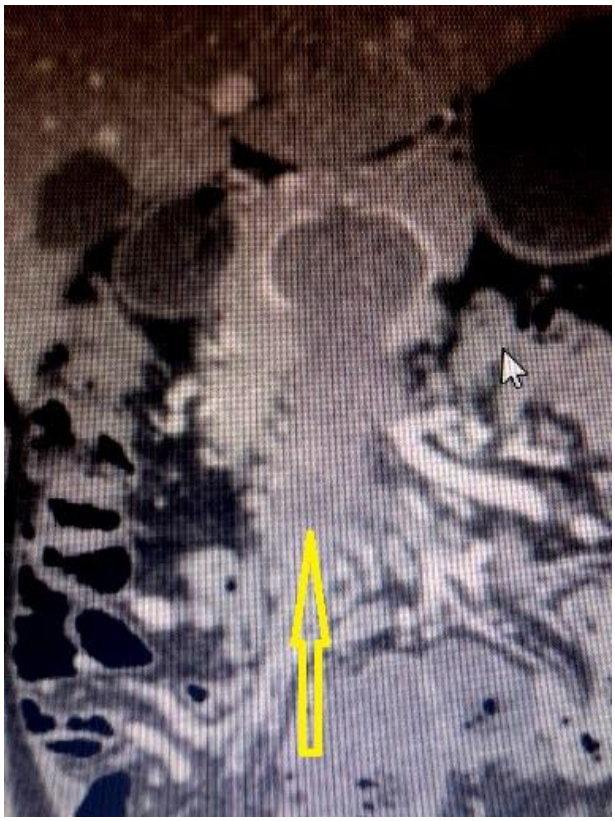

Figure 2: Computed tomographic scan showing portal vein aneurysm and portal vein (PV) and superior mesenteric vein (SMV) (arrow) thrombosis

DISCUSSION AND CONCLUSIONS

The portal vein is formed by the confluence of the SMV and the splenic veins, anterior to the inferior vena cava and posterior to the neck of the pancreas. It divides into right and left branches and supplies the liver.

In an autopsy study of 92 cases, the PV diameter was found to be 0.64–1.21 in patients without evidence of cirrhosis or portal hypertension (5). In another ultrasound-based study the maximum diameter of the PV was found to be 15 mm on normal patients and 19 mm in cirrhotic patients (1). Therefore, when the portal vein diameter is more than 19 mm in patients with portal hypertension and more than 15 mm in patients with normal portal venous pressure it is called aneurysmal (1).

The causes for portal vein aneurysm are divided into congenital and acquired. During the embryonic development, two vitelline veins originate from the yolk sac. The proximal parts of the vitelline veins lie on the right and left sides of the developing duodenum. These vitelline veins become interconnected by three transverse anastomoses. The superior mesenteric and splenic veins develop separately and join the left vitelline vein (Figure 3).

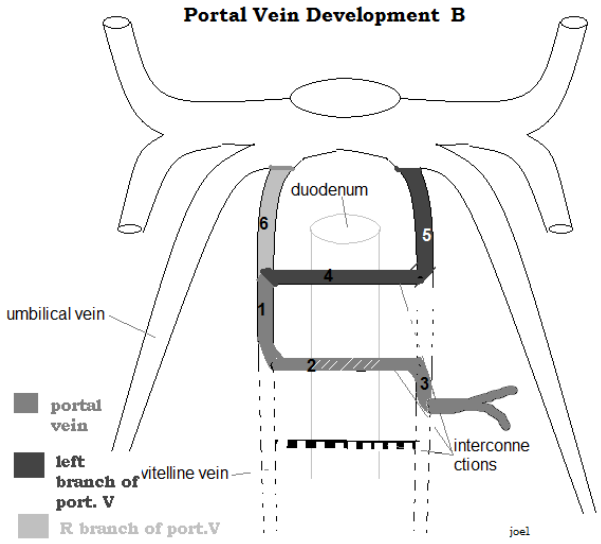

Figure 3: Portal vein development

During further development, parts of the vitelline veins and the caudal ventral anastomosis disappear. The rest forms the portal vein and its division.

The areas where these branches join or incomplete regression of the embryonic veins, result in aneurysm formation (Congenital) (6). Congenital aneurysm formation concept is supported by the development of PVA in children with normal portal venous pressure as in the above-described case. Acquired causes of PVA include portal venous hypertension, pancreatitis and trauma. The abovementioned patient did not have any of the above factors.

The patients with PVA can be asymptomatic. Others present with nonspecific abdominal pain. About 10% present with symptoms due to compression by the aneurysm e.g. bile duct compression and jaundice. Acute presentation following rupture of PVA has also been reported (7), (8), (9). The portal vein thrombosis occurs in about 20% to 30% of cases with PVA (2), (10), (11),

Indications for interventions for PVA include; Presence of symptoms, complicated aneurysm (rupture, thrombosis, compression of adjacent structures, etc.), and aneurysms larger than 3 cm (12). The management options of PVA include conservative, surgical and endovascular.

In addition, patients who developed thrombosis are treated with anticoagulation. Reports describing endovascular techniques for PVA management are lacking in the literature. About 20% of the patients with PVA require surgical interventions (13).

The technique of surgical intervention depends on the presence of cirrhosis and portal hypertension, presence of thrombosis and the type of aneurysm (Saccular or fusiform). (9), (10). In patients without portal hypertension aneurysmorrhaphy or aneurysm excision and repair are performed (10). When cirrhosis and portal hypertension are present, a portosystemic shunting with splenectomy or liver transplantation may be needed (13).

In the above-mentioned patient considering that the patient was asymptomatic and she maintained the weight, a conservative approach with anticoagulation was considered. She did not have evidence of portal hypertension probably due to partial recanalisation and development of collateral circulation.

Therefore, PVA as a cause of portal vein thrombosis should be considered especially in a young patient with normal liver and normal portal venous pressure. Therefore, to diagnose the PVA, it is necessary to be aware of the condition and a thorough evaluation of the CT scan should be done.

Author declaration

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Conceptualization and Design: JA; Data Collection and Patient Care: JA; Manuscript Writing: JA; Critical Review and Editing: JA.

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Written informed consent was obtained from the patient before they participated in the case, ensuring their understanding of the purpose, procedures, and potential implications involved.

Statement on data availability:
All data generated during this study are included in this published article.

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