International Journal of Science and Research (IJSR)
ISSN: 2319-7064
SJIF (2020): 7.803

Portal Venous Varix: Case Report

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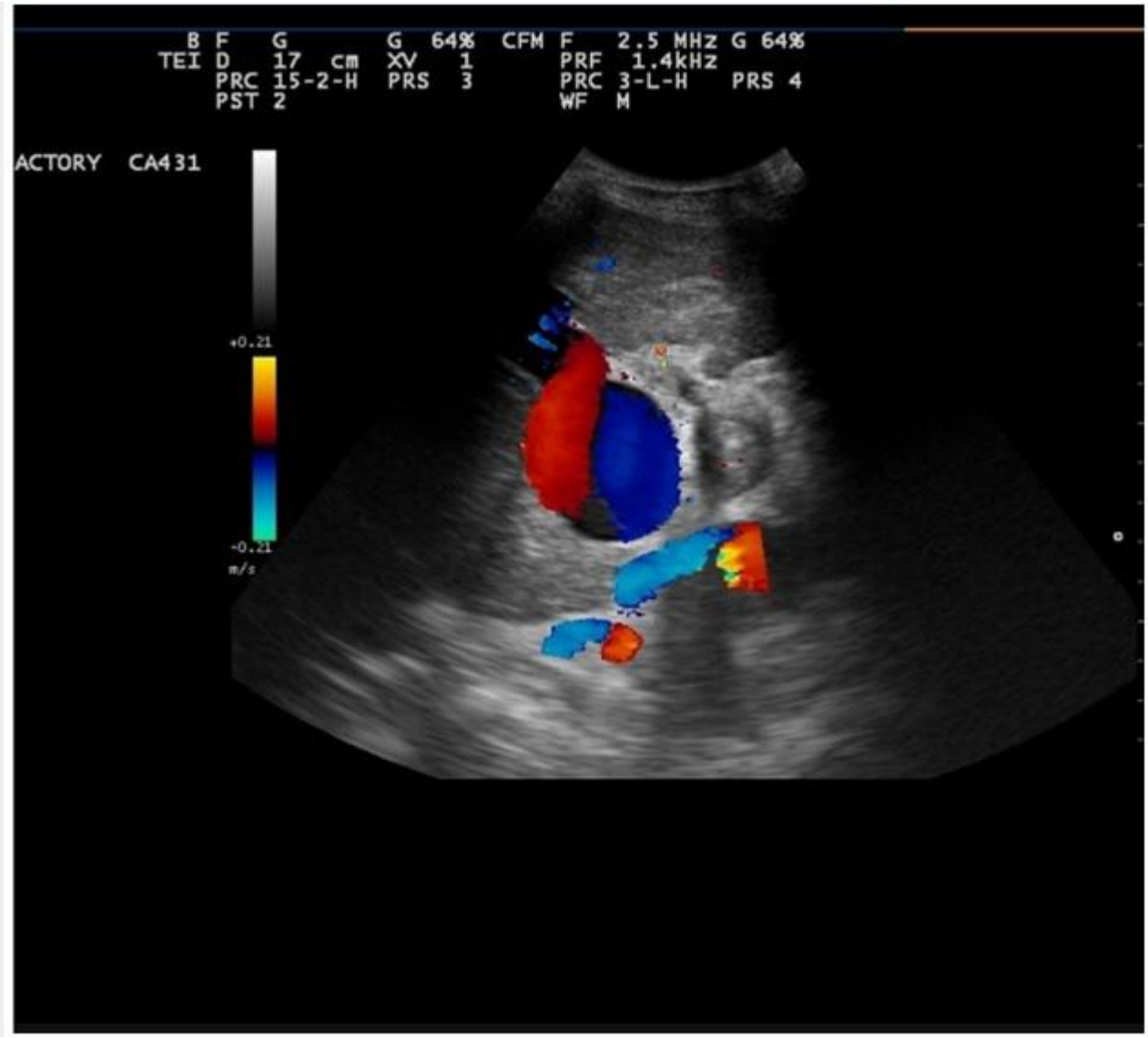
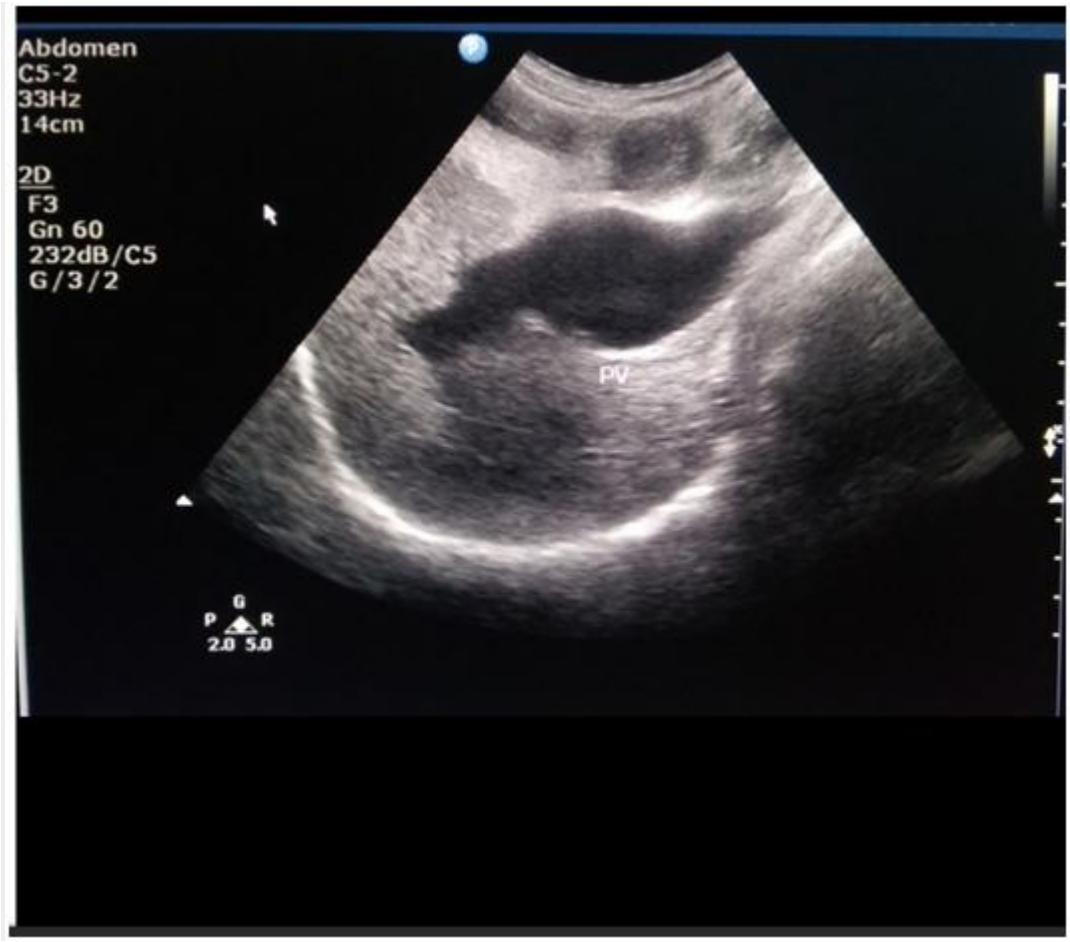
1.Presentation

A 35 year old female, who had been diagnosed as HCV+ 2 years back, presented with a history of two episodes of haematemesis in the past five days and malena for two days. Massive splenomegaly was present. No signs of chronic liver disease stigmata or signs of portal hypertension were noted.





On ultrasound, fusiform non calcified dilation of intrahepatic portal vein was seen with no thrombosis. The flow was hepatopetal, monophasic, non pulsatile with "yin yang" sign.

2.Case Discussion

Portal venous aneurysm are quite a rarity with a reported incidence of approx 0.43% [1, 2] Here we present a case of a HCV positive, cirrhotic woman with intrahepatic portal venous aneurysm.

Intrahepatic portal venous aneurysm are the rarer type of portal venous aneurysms, with the cases contributing only 23.1 % of all the portal venous aneurysms.[2]

The patient being cirrhotic also makes this case a rarity, as portal venous aneurysms are rarely reported in cirrhotic patients.[3, 4]

Most aneurysmal dilatation of the venous system occur in the popliteal, jugular, or saphenous veins, with the rarest being those affecting the femoral, caval, forearm, or portal veins[5]. Portal venous system aneurysm are defined as a focal saccular or fusiform dilatation of the portal venous system which includes portal vein (PV), superior mesenteric vein (SMV), and splenic vein (SV) in the region of spleno-portal junction.

Portal venous system aneurysm were first described in 1956 [6] and are the commonest of the visceral venous aneurysms. Earlier studies based on sonographic findings showed the incidence to be 0.6 per 1, 000 patients [7] while newer CT based studies show the incidence to be as high as 4.3 per 1, 000 patients [1] This rise in the more recent studies is attributed primarily to the increasing use of radiological investigations, and advent of new imaging techniques. Approximately 170 cases of portal venous aneurysms had been described till a few years back [8]

Aneurysms of the portal venous system occur mainly at sites of bifurcation or confluence, and can be intrahepatic or extrahepatic. Aneurysms in the small or peripheral intrahepatic portal branches are rare but have been reported in association with portal-hepatic venous shunts.[9]

Ultrasonography with doppler is a reliable modality with the aneurysm appearing as a cystic structure with nonpulsatile monophasic flow pattern within the lesion.

Contrast CT and MRA are useful in equivocal cases, for the differential diagnosis of thrombosis of a portal venous aneurysm, and for clear anatomic delineation for surgical intervention.

Portal venous system aneurysms could be either congenital or acquired in origin. The portal venous system develops from vitelline and umbilical veins. The right primitive vitelline vein normally regresses, however the regression failure can lead to portal venous aneurysms. The diverticular remnant of the vitelline vein enlarges to form a saccular aneurysm of the portal vein.[10] Other

researchers attribute the development of a portal vein aneurysm to an inherent weakness of the vein wall.[10]

The acquired portal venous system aneurysms occur mainly in cases of portal hypertension which leads to intimal thickening and compensatory medial hypertrophy. The media is replaced by fibrous tissue which weakens the tensile strength of the vein wall, and hence makes it susceptible to aneurysmal dilatation.[11]

Portal hypertension secondary to chronic liver disease is considered the most common cause of acquired portal vein aneurysms.[11] Other causes of acquired portal vein aneurysm include severe pancreatitis causing digestion and inflammation of the portal vein, trauma, invasion of the portal vein by various malignancies.1[2] and after making a potocaval shunt which causes arterialisation of the portal vein.[13, 14]

Few other possible and suspected causes include portal venous thrombosis, thrombophillia defect, Cutis laxa, and Psoriasis. In patients with a thrombophilic defect, thrombosis of the portal vein can cause portal venous system aneurysm formation. In Cutis laxa, there is inherent weakness of the vessel wall, which can lead to aneurysm formation.

Portal venous aneurysms are rarely reported in cirrhotic patients.[3, 4]

Most patients with portal vein aneurysms are asymptomatic or have unrelated gastrointestinal complaints that prompt abdominal imaging. The clinical aspects of portal venous system aneurysms are related to their size. Small aneurysms often produce no symptoms. In a study it was revelaed that the patients symptomatic due to portal venous aneurysms always exhibited aneurysms that were thrombosed, usually had larger aneurysms than those of asymptomatic patients, and often had multiple aneurysms.[1] Complications of portal vein aneurysms include thrombosis, rupture, and symptoms from pressure on adjacent structures. Acute thrombosis of the portal vein aneurysm can result in severe, life threatening portal hypertension.[10] Recurrent or chronic thrombosis can result in the complete occlusion of the portal vein and manifestations of extrahepatic pulmonary hypertension such as varices and bleeding.[10] Rupture of portal vein aneurysms is not commonly seen because of low portal venous pressure, but the risk of rupture rises in the presence of portal hypertension.[6] Large extrahepatic portal vein aneurysms can cause symptoms by pressing on adjacent viscera. Deviation, compression, and obstruction of the common bile duct can cause recurrent abdominal pain, cholestasis, and obstructive jaundice. Obstruction of the duodenum, compression of the portal vein by the aneurysm causing thrombosis and portal hypertension, and compression of the vena cava have also been reported.[10]

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