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Case Report

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CASE SERIES: PORTOSYSTEMIC COLLATERALS

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ABSTRACT

Portal hypertension is defined as pathological increase in portal venous pressure. Increase in portal venous pressure can be caused by increased resistance to blood flow either at the level of the portal vein (pre-hepatic), hepatic sinusoids(hepatic) or hepatovenous outflow (post-hepatic). This results in formation of portosystemic collateral pathways between the splanchnic circulation and the systemic circulation. Isolated gastric and esophagogastric collaterals are another important collateral pathways to be mentioned, because they may cause massive gastrointestinal hemorrhage if goes untreated. These pathways are numerous, widespread and varied in appearance. Knowledge of these common and less-common portosystemic collateral pathways on CT /MRI at each anatomic site is important implication for clinicians and interventional radiologists to plan their further management.

KEYWORDS: Portal hypertension, Extra hepatic portal vein obstruction, Hepatic venous pressure gradient, Transjugular intrahepatic portosystemic shunt.

INTRODUCTION

Normal portal venous pressure is between 5 to 10mm Hg while normal pressure gradient between portal vein and inferior vena cava i.e. hepatovenous pressure gradient is 1 to 5mm Hg.

Portal hypertension is defined when portal venous pressure greater than 12 mm Hg.

Portosystemic collaterals are abnormal communications between the portal system and the systemic circulation, and such shunts can be congenital or acquired. Congenital shunts can be intrahepatic or extra hepatic. The more severe and more prolonged the portal hypertension, the higher are the number of portosystemic pathways.

Among these collateral pathways, Gastric fundus and/or esophageal varices are one of the most severe complications. However, esophageal varices are the most important clinically because it may cause massive hemorrhage of upper gastrointestinal tract. The clinical management of these gastric and esophageal varices is related to their hemodynamics and location, which can be determined with the help of MDCT imaging.

In this article, we present four different cases related to portal hypertension. All these 4 cases had different findings.

CASE REPORT

CASE 1:

A 50 year old male, known case of EHPVO (extra hepatic portal vein obstruction) with portal hypertension, presented with multiple episodes of blood in vomiting associated with jaundice and pain in abdomen. He was then referred to imaging department for USG. USG revealed cirrhotic liver with multiple portosystemic collaterals. Then, we decided to go ahead with CT.

CT showed few esophagogastric collaterals in (Fig 1A) some of them protruding intraluminally (Fig 1B), outflow being left renal vein which has retroaortic course. (Fig 1C). Additional findings of cirrhotic liver with multiple other porto-systemic collaterals were also noted.

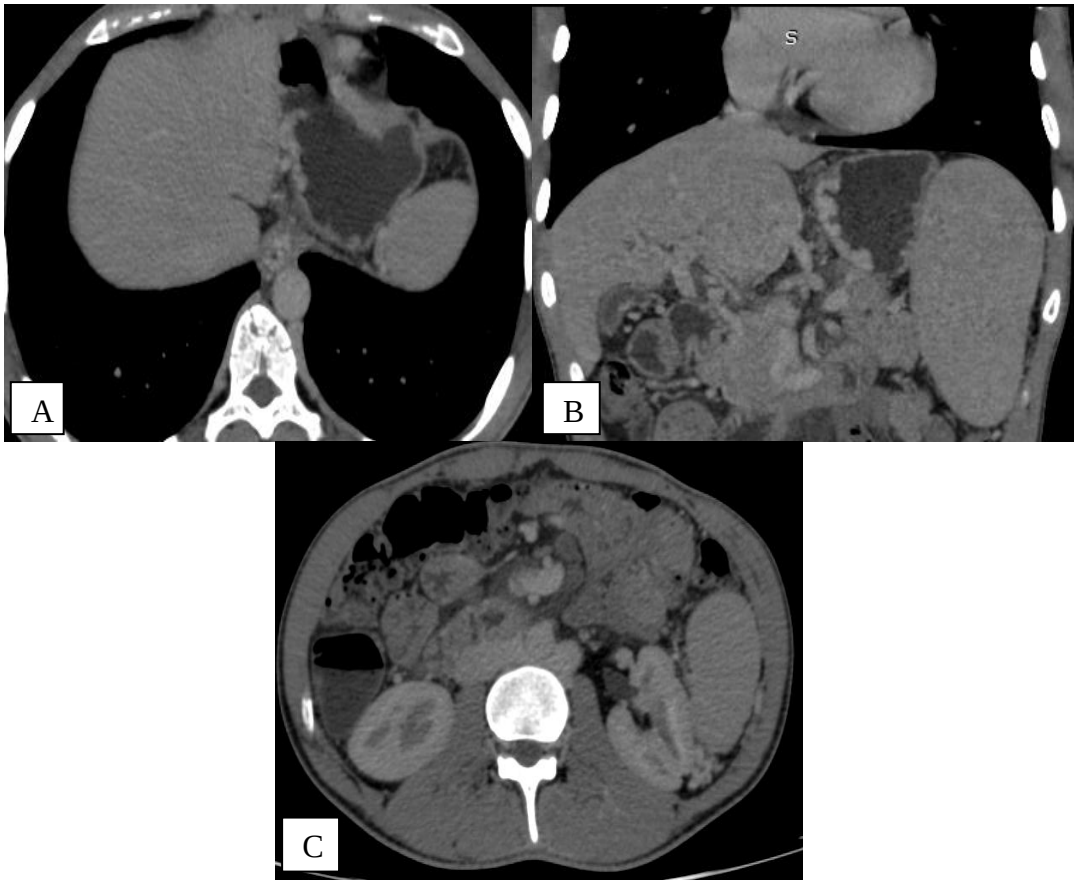

Figure 1: Multidetector computed tomography with contrast enhanced axial and reformed coronal images showing few esophagogastric collaterals, some of them protruding intraluminally, outflow being left renal vein which has retroaortic course.

CASE 2:

A 58-year-old male who was a known case of alcoholic cirrhosis, who presented with intermittent pain in abdomen, abdominal distension since 15 days. On examination, no specific diagnosis was made. Biochemical abnormality revealed raised levels of serum alpha-fetoprotein. He was then referred to imaging department for USG. USG revealed cirrhotic liver with multiple portosystemic collaterals. Then, we decided to go ahead with CT.

CT showed multiple lienorenal and anterior abdominal wall collaterals. (Fig 2A, B); and extramural and intramural gastric collaterals (Fig 2C, D).

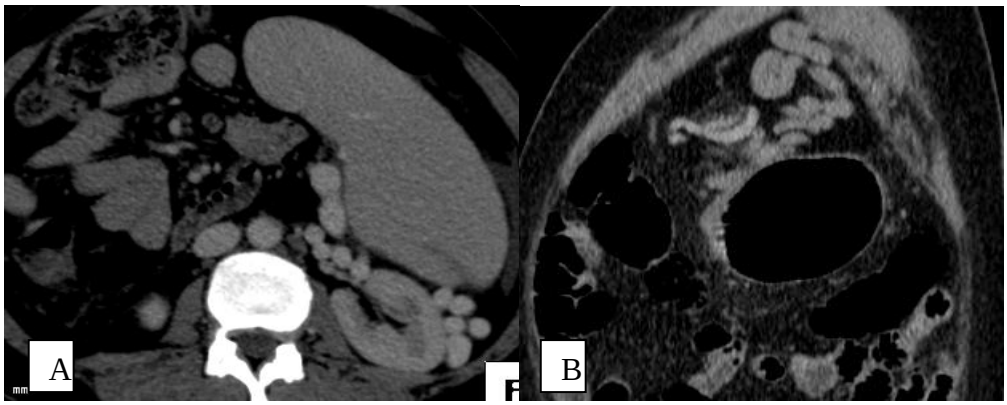

Figure 2: Multidetector computed tomography with contrast enhanced axial and reformed coronal images showing multiple lienorenal and anterior abdominal wall collaterals.

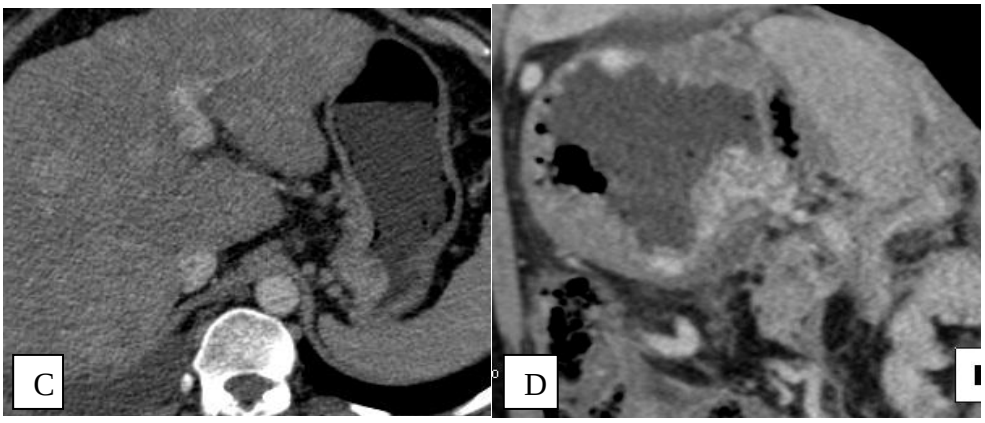

Figure 3: Multidetector computed tomography with contrast enhanced axial and reformed coronal images showing extramural and intramural gastric collaterals.

CASE 3:
A 64-year-old male who was a known case of EHPVO with portal hypertension, who presented with continuous pain in abdomen with jaundice since 1 month. He was then referred to imaging department for USG. USG revealed gross splenomegaly, multiple Porto systemic collaterals and dilated portion of proximal and mid CBD, Distal CBD was not well appreciated. So, the clinician advised to carry out MRI abdomen with contrast for the same.

Dynamic contrast enhanced MRI images of abdomen showed esophagogastric varices (Fig 3A,) with inflow being from left gastric vein (which ms 9 mm)(Fig 3B) and outflow being lienorenal collaterals(Fig 3C).

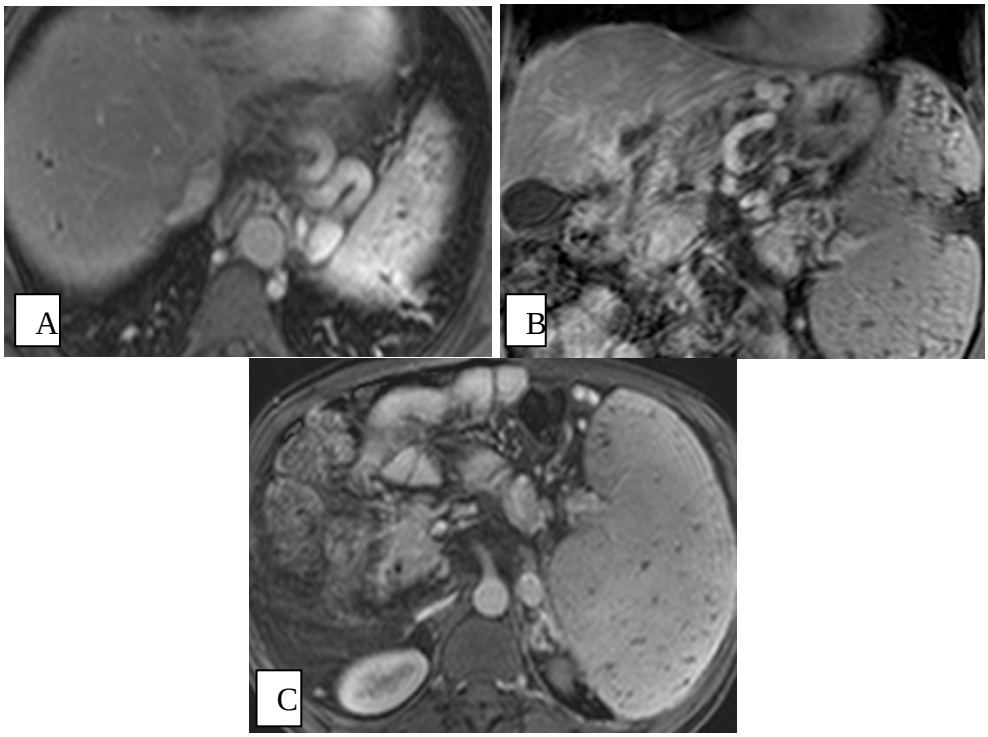

Figure 4: Respiratory gated DCE-MRI (dynamic contrast enhanced) abdomen axial images reveal esophagogastric varices with inflow being from left gastric vein (which ms 9 mm) and outflow being lienorenal collaterals.

CASE 4:
A case of 19 year young adult male who was a known case of chronic calcific pancreatitis diagnosed 1 year back, who presented now with dull aching pain in abdomen since 10-15 days month. He was then referred to imaging department for USG. USG revealed findings of calcific pancreatitis, multiple portosystemic collaterals, distal splenic vein thrombosis with mild splenomegaly. So, the gastroenterologist advised to carry out MRI abdomen with contrast for the same.

Dynamic contrast enhanced MRI images of abdomen showed multiple gastric varices with predominantly extramural component (Fig 4A, B). No significant gastrorenal shunt is noted. (Saad Caldwell classification Type 2A).

Additional findings of chronic thrombosis of distal splenic vein with multiple mesenteric peripancreatic, splenic hilar and perigastric collaterals were also seen with multiple collaterals noted between SMV and terminal SV near hilum.

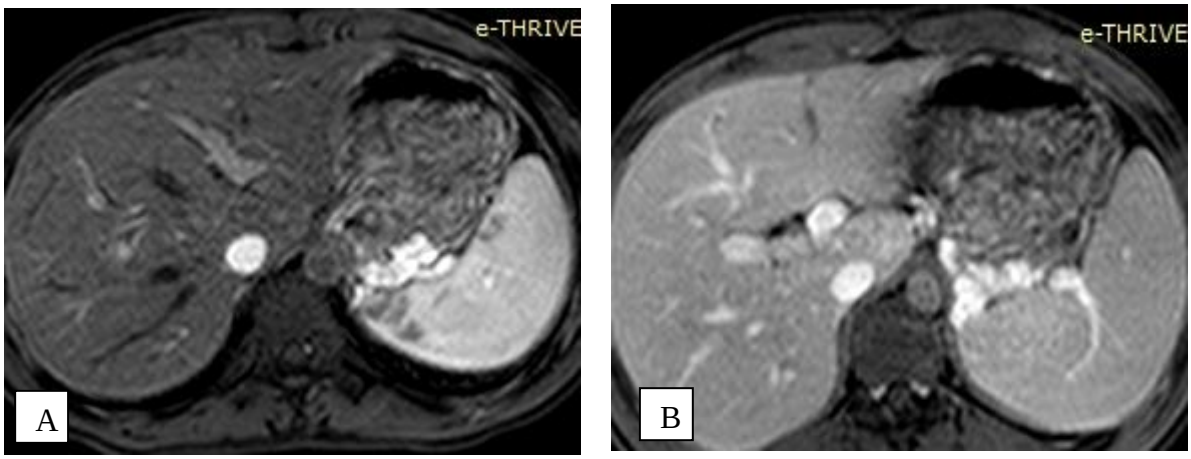

Figure 5: Respiratory gated DCE-MRI abdomen axial images reveal multiple gastric fundic varices with predominantly extramural component.

DISCUSSION

Portosystemic collaterals are one of the most severe complications. It may cause severe hemorrhage of the upper gastrointestinal tract. So, prompt evaluation of portosystemic collaterals is important.

Multidetector contrast-enhanced computed tomography can determine the extent and location of portosystemic collaterals in patients with portal hypertension. In cirrhotic patients with portal hypertension, some blood in the portal venous system may reverse its flow and pass through the portosystemic anastomoses in the systemic venous system. As a result, major hepatofugal collateral pathways can develop in patients with portal hypertension. Multidetector contrast enhanced computed tomography can also determine gastric varices (intramural and extramural), esophageal varices, paraesophaeal varices, esophagogastric, isloted gastric varices as well as their association with gastrorenal and splenorenal shunts.

In our CASE 1 and CASE 2:
There were multiple porto-systemic collaterals, esophagogastric as well as gastric varices. Knowledge of exact location of these varices is important for a gastroenterologist as well as for an interventional radiologist to plan their further management.
--Intramural gastric collaterals are treated by endoscopic glueing of fundic varices.
--Extramural gastric collaterals are treated on the basis of hepatic venous pressure gradient (HVPG). If HVPG is more than 12 mm Hg, treatment is TIPS (Transjugular Intrahepatic Portosystemic Shunt). If HVPG is less than 12mm Hg, treatment is BRTO.(Balloon occluded retrograde transvenous obliteration).

CASE 3: Knowledge of afferent and efferent pathways for porto-systemic collaterals as well as shunts is also important. Whether there are single or multiple pathways should be determined.
Some of these afferent-efferent pathways are as follows:
---Esophageal collaterals: left gastric as afferent for esophageal varices and efferent as azygous and hemiazygous veins.
---Gastric collaterals: afferents are left gastric and short gastric veins and efferent as splenic vein.
---Rectal and perirectal collaterals: afferent being Inferior mesenteric vein and iliac vein, efferent being middle and inferior rectal veins.
---Paraumbilical vein: afferent being left portal vein and efferent being anterior abdominal wall veins.
--- Gastrorenal shunt: afferent being Gastric varices or posterior or short gastric veins and efferent being Left renal vein.
---- Splenorenal shunt: afferent being splenic vein and efferent as Left renal vein.

*CASE 4: (*case of chronic calcific pancreatitis with thrombosis of splenic vein).
Important is to determine are these varices are isolated gastric varices or esophagogastric varices, and whether they are associated with shunts (gastrorenal, splenorenal) and these are accordingly planned for further line of management by clinician/ gastroenterologist as per SADD-CALDWELL classification of gastroesophgeal varices with its correlation endoscopic Sarrin classification of gastroesophgeal varices.

CONCLUSION

Portal hypertension is a common clinical problem. It has a variety of presentations, as do portosystemic shunts in imaging. MDCT contrast enhanced can accurately demonstrate majority of these collateral pathways. The role of interventional radiologist as well as gastroenterologist is to have knowledge of various patterns of collateral pathways for detecting and diagnosing portal hypertension, for image guided interventions to treat complications of portal hypertension and prevention of accidental vascular injury.

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