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A Case of Superior Mesenteric Artery Occlusion without causing Non-Cirrhotic Portal Hypertension

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1. Case Summary

Non-cirrhotic portal hypertension is a potential consequence of superior mesenteric artery (SMA) occlusion. We reported the case of a 72-year-old female with an incidental finding of a critical SMA occlusion which did not result in non-cirrhotic portal hypertension due to the development of collaterals.

2. Case Details

A 72-year-old female was hospitalized following a sudden onset of left lower quadrant abdominal pain on a background of chronic constipation and overflow diarrhea.

During the admission, she had an abdominal CT, showing faecal loading and rectal distension with mild inflammatory stranding consistent with stercoral colitis. It promptly resolved after administering gastrograffin. Incidentally, an SMA occlusion was also found on CT.

The origin of the SMA demonstrates high-grade stenosis with post-stenotic dilatation of 6 mm and the formation of collaterals distally. There was no evidence of infarction. The SMA and collaterals can be visualized below [Figure 1].

An SMA occlusion can be acute, leading to acute mesenteric ischemia, or chronic, leading to non-cirrhotic portal hypertension. Non-cirrhotic portal hypertension commonly presents with esophageal varices, and less commonly ascites. However, there were no signs of either in our patient. Laboratory findings also show that the liver's synthetic and excretory functions were normal.

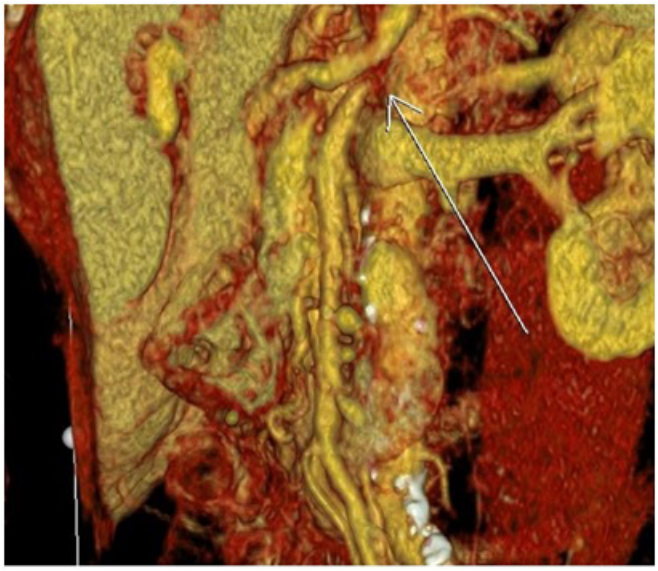

Figure 1: CT image demonstrates the SMA and the collaterals formed distally

Ultrasound of the liver showed a normal liver size, smooth surface, normal portal vein diameter, and ante grade portal vein flow, suggesting no portal hypertension. This is likely due to collaterals compensating for the SMA stenosis.

Further investigations were performed to find any pre-disposition to the occlusion. Common causes are atrial fibrillation, inherited thrombophilia, occult malignancy, atherosclerosis, and calcification [1]. A recent MRCP found a likely malignant tumor within the distal common bile duct at the ampulla of Vater, which causes an acquired thrombophilia. Thromboembolic disease is a common

complication of pancreatic cancer, with incidences ranging from 17-57% [2], portal vein thrombosis being a common clinical manifestation. This is likely the cause of the occlusion. Other common causes have been ruled out.

Treatment for a critical narrowing would be a trans-jugular intrahepatic portosystemic stent, with follow-up with a hepatologist [3]. However, despite the critical stenosis, our patient had a normal liver function with no portal hypertension and can be discharged for outpatient follow-up.

3. Conclusion

Not every patient with an SMA occlusion develops non-cirrhotic portal hypertension, due to collaterals. This is an important factor to consider for follow-up planning as the presence of collaterals may be a reassuring sign and may not require follow-up as intense as if they were absent.

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