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INTERNATIONAL JOURNAL OF ADVANCED RESEARCH (IJAR)

Article DOI:10.21474/IJAR01/15282
DOI URL: <http://dx.doi.org/10.21474/IJAR01/15282>



RESEARCH ARTICLE

CASE REPORT: HEPATIC ARTERY ANEURYSM.A RARE VISCERAL ANEURYSM

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Manuscript Info

Manuscript History

Received: 27 June 2022
Final Accepted: 30 July 2022
Published: August 2022

Abstract

Hepatic artery aneurysm (HAA) is a rare disease (0.002%– 0.4%) but is a clinically important phenomenon [1]. HAAs are traditionally the second most common visceral aneurysms with an incidence of 20% and have the highest (44%) reported rate of rupture [2]. The clinical manifestations depending on the size of the aneurysm include epigastric pain, obstruction of biliary tract, rupture and death [3]. Imaging modalities like computed tomography (CT) and CT angiography have a valuable role in the early detection of HHA, its complications, and selecting appropriate treatments depending on the size and location of the aneurysms [4].The etiology of visceral artery aneurysms is mostly atherosclerotic. Trauma and inflammation may cause pseudoaneurysm formation, such as in case of an acute pancreatitis causing periarterial inflammation or vessel erosion from an adjacent pseudocyst[5,6]. Other conditions which are associated with hepatic artery aneurysms are medial degeneration, fibromuscular dysplasia and vasculitis [7]. Atherosclerotic aneurysms are typically extrahepatic while traumatic aneurysms or pseudoaneurysms are more commonly intrahepatic [8].

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Introduction:-

Case presentation:

A female of 36 years old presented with recurrent abdominal pain for around 9 months and upper abdominal mass mainly at right hypochondrium region. Her pain was controlled with simple analgesia. She was pale not jaundice and hemodynamically stable. The mass was pulsatile 10 cm and easily noticed. Abdominal ultrasound showed Saccular aneurysm of the hepatic artery with intra-luminal thrombus and mural plaques which are calcified and non-calcified.

Interruption of hepatic artery by the aneurysm with distal run off via collateral arteries with moderate fatty hepatomegaly

The patient was admitted and A CT Abdomen with Angiogram was done which showed A rounded lesion which is rather rounded and of soft tissue attenuation value is seen at the level of pancreatic head and fairly well defined and

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exerts significant mass effect on the surrounding structures causing indentation and elevation of the inferior hepatic surface and significantly compressing the portal vein, common bile duct and hepatic artery as well as compressing, splaying and indenting the posterior gastric wall, splaying proximal duodenal segments, compressing, splaying and stretching the head and body of pancreas.

- The lesion also significantly compresses the splenic and superior mesenteric veins.
- After contrast administration the lesion shows gradual enhancement not matching with early arterial phase and delayed contrast wash out in comparison to porto-venous phase.
- The hepatic branch of coeliac artery is seen in direct continuity with lesion being tangential to its dome as well as spurt of contrast via a small opening on later arterial phase. The lesion is seen exactly located prior to division of the hepatic artery, where the right branch is distally enhancing, however, the proximal aspect of left hepatic artery is seen and its distal segment or distal branches could not be outlined.
- After contrast administration the lesion shows significant enhancement yet a central area of relatively low attenuation value within its center is noted denoting blood clot or thrombus. It shows peripherally and irregular wall where multiple fine projections towards the center are seen some of which show calcifications. It measures 80 x 90 x 70 mm in maximum ML, AP and CC maximum dimensions is seen. No contrast extravasation detected.
- Numerous small, and tortuous arteries were enhanced in the late arterial phase with no definite arterial communications seen.
- No venous communication with the mass lesion detected.
- Furthermore, the antero-lateral aspect of the superior mesenteric artery shows an antero-lateral protrusion from its wall prior to a side branch. The lesion is significantly enhancing matching with phasic enhancement of the superior mesenteric artery and its branches denoting its arterial nature. The lesion measures 8 x 9 x 7 mm in maximum ML, AP, and CC dimensions.

Liver

- It is relatively mildly enlarged with smooth outlines and intact capsule; however, it shows heterogeneous attenuation value being predominantly of low attenuation and heterogeneous enhancement pattern. Nonetheless, no focal lesion of abnormal attenuation value or abnormal enhancement detected.
- Mild dilatation of proximal intra-hepatic biliary radicles are seen.
- The portal vein is dilated, splayed, partly compressed, however, homogeneously and fairly enhanced.

Gall bladder

- It shows average capacity with no evidence of radio dense gall stones detected.

Pancreas

- Despite being compressed and splayed it shows normal matrix, size and homogenous attenuation pattern with no evidence of related focal lesion, abnormal enhancement, or calcifications detected.
- No pancreatic duct dilatation identified.

Spleen

- It is of normal size and tissue characters.
- No focal lesions or calcifications detected.
- Intact splenic capsule.

Kidneys

- Both shows small hepatic veins draining into the IVC, another separate right renal veins draining into right external iliac veins and a separate left renal vein draining into right common iliac vein.
- Both show normal size, shape, orientation, position, adequate bilateral contrast uptake, concentration and excretion with no evidence of dense renal stones, expanding lesions or back pressure changes identified.
- No evidence of renal focal solid or cystic lesions detected.
- Both renal arteries and veins are of average calibers, patent and well opacified.

Diagnostic CT scan and angiography:

Figure 1:-Huge saccular aneurysm compressing the liver, portal vein and hepatic artery.



Figure 2:- Sagittal plan showing mass effect of the hepatic artery aneurysm and showing clots, calcification and significant enhancement.

We endorsed the patient to vascular team, and they prepared for angiography and endovascular intervention.

A selective catheterization of the coeliac trunk was done followed by angiography which showed the huge hepatic aneurysm. The aneurysm was selectively catheterized followed by insertion of long 7Fr sheath and advanced until the aneurysm. Attempts were performed to catheterize selectively the intrahepatic arteries (aneurysm arterial outflow) using different techniques and catheters with no success. The intrahepatic arteries were successfully catheterized using microcatheter. Then, embolization was done to the intrahepatic bifurcation (aneurysm arterial outflow) using a microvascular plug of 6.5mm. Afterward, coils were deployed into the aneurysmal sac to enhance thrombosis. Then, the proper hepatic artery (aneurysm arterial inflow) was embolized using microvascular plug of 9.2mm. Follow up angiogram showed good results. SMA angiogram showed filling of the intrahepatic arteries by collaterals.

The procedure outcome ended with complete thrombosis of hepatic artery and discharged on good condition. However, she is still having chronic pain due mass effect, raised liver enzymes and low HB due to chronic anemia.

Follow up Doppler USS after angiography:

Large aneurysm at the hepatic artery measuring 8 x 7.7 cm shows hyper echogenicity with no obvious doppler signal due to thrombosis. Both the inflow and outflow plugs of the hepatic aneurysm were visualized. Good doppler signals were seen at the celiac trunk as well as at the intrahepatic arteries

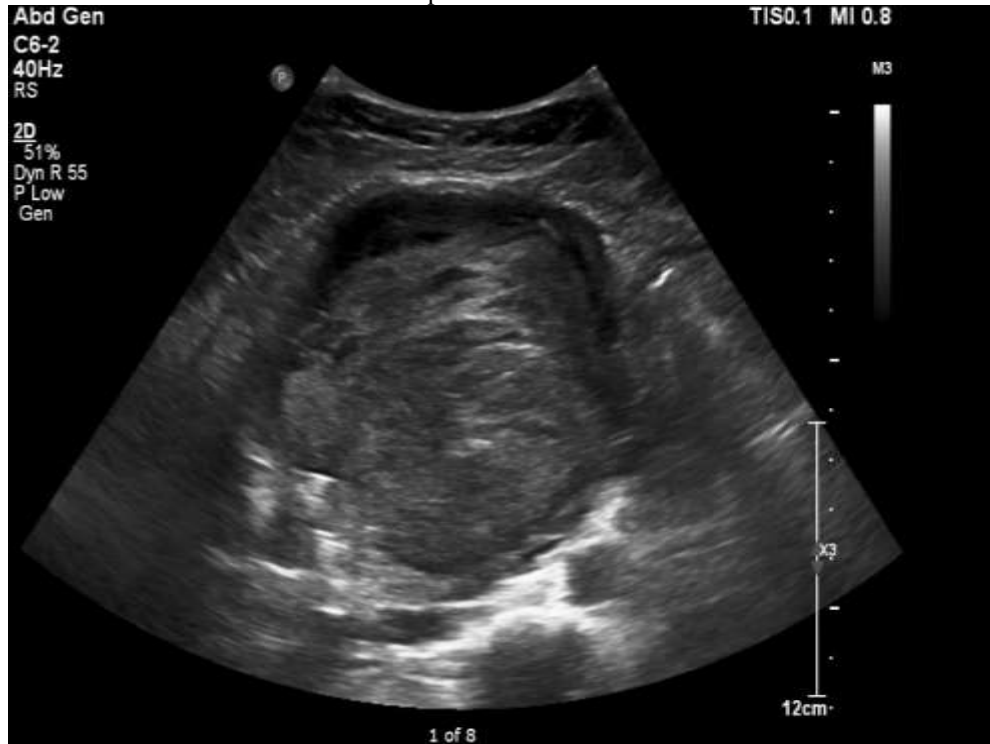


Figure 3:- Doppler USS showed no signal due to thrombosis of hepatic artery aneurysm 5 days post-procedure.



Follow up CT abdominal aortogram: post procedure, showing thrombosis of the hepatic artery aneurysm.

Discussion:-

Although very rare, hepatic artery aneurysms are the second commonest visceral aneurysms, after splenic artery aneurysms [5]. They usually are asymptomatic and discovered as an incidental finding during diagnostic imaging procedures performed for other reasons. Some patients, however, experience abdominal pain, and others are diagnosed during surgery for rupture. These patients usually are in hemorrhagic shock and reported survival rates are as low as 50%.² The risk of rupture appears to be related to the size of the aneurysm. The size threshold at which treatment becomes advisable is controversial, although it has been suggested that aneurysms less than 2 cm may not require treatment [6].

The clinical presentation of a hepatic artery aneurysm may be non-specific and variable. The classic triad is epigastric pain, haemobilia and obstructive jaundice, although only one-third of patients with hepatic artery aneurysm present with all three symptoms [5].

Open surgical treatment of visceral artery aneurysms is safe and effective, and offers satisfactory early and long-term results [9].

Nevertheless, the minimal invasive character of endovascular repair might provide a benefit in both elective and emergency interventions [10]. Endovascular exclusion may be accomplished by coil embolization or the selective use of N-butyl-2-cyanoacrylate[8]. Coil embolization has been used in anatomically difficult cases due to its relative simplicity. Stent-grafting offers a more physiologic repair in its ability to maintain blood flow through the affected artery[6].

In Conclusion:-

Visceral aneurysms such as hepatic artery aneurysm should be considered as a rare but important differential diagnosis of acute abdominal pain. All emergency physicians and surgeons should keep this diagnosis in their mind, as it can prevent from its life-threatening complications.

Management of hepatic artery aneurysm if discovered before rupture is either surgical resection or endovascular embolization and stenting depending on size and underlying causes.

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