

A CASE REPORT ON LIVER ABSCESS CAUSING INFERIOR VENA CAVA THROMBOSIS

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KEYWORDS :

INTRODUCTION:

Amoebic liver abscess is the most common extra-intestinal manifestation of *Entamoeba histolytica* infestation and it is associated with significant morbidity and mortality. The common complications of liver abscess include rupture into peritoneal cavity and pleural cavity, rarely vascular complications are seen in liver abscess in the form of thrombosis or compression resulting in either hepatic venous outflow obstruction or inferior vena cava obstruction. I am presenting a case of liver abscess where patient presents with signs of IVC thrombosis and radiological confirmation of the IVC obstruction by thrombosis and/or external compression.

CASE REPORT:

A 30 year old male patient presented at civil hospital Ahmedabad in emergency surgical department on 13th October 2018 with chief complaint of pain in epigastric region and fever from 6 days and loose stool for 2 days. Vitals were pulse-86/m, blood pressure-130/86 mmHg. On physical examination abdomen mildly distended and mild epigastric tenderness present. In blood investigations; HB-12 gm/dl, WBC-37000, PLT-91000, INR-1.52, S. BILIRUBIN-Total-5.98, direct bilirubin-1.52, s. creatinine-4.78. Chest x-ray within normal limit, abdominal x-ray within normal limit. USG (Abdomen+Pelvis)-liver appears enlarged in size and bright in echotexture and shows 144 cc sized partially liquefied abscess in right lobe.

On day of admission USG guided aspiration was done, approximately 30cc encovy sauce pus aspirated, post tapping chest x-ray normal. After 2 days of admission patient developed abdominal distention and bilateral pedal edema. Ryles tube inserted and catheterization done, and further evaluation done. Chest x-ray showing bilateral cp angle blunted s/o-minimal pleural effusion. Expert USG done-s/o-280 cc solidified liver abscess and supra hepatic IVC thrombosis. CVTS reference done, they advised conservative management with short acting heparin for 10 days and overlapped with tab warfarin on day 8. While starting heparin INR was 1.6 and after starting tab warfarin INR was 2.10. Expert medicine reference done, they advised CT pulmonary angiography+abdomen and pelvis. CT S/O-liver abscess extending in hepatic IVC causing thrombosis in hepatic IVC and right atrium. No evident pulmonary thromboembolism. 2DECHO was normal. Patient shifted in ICU and kept on BIPAP mode for 2 days then shift to O₂ mask. Pedal edema and abdominal distension reduced on day 8 of giving short acting heparin. Follow up USG(A+P) done s/o-Approx. 120cc sized predominantly solidified abscess is noted in right lobe of liver. Wall of IVC appears thickened and shows normal color flow

within p/o- Recanalization of IVC thrombosis. Patient started orally and shift to ward. Patient was discharged in stable condition after 25 days with tab warfarin (5 mg) for 6 months according to INR report with proper advice to see for gum bleeding, bleeding per rectum. Follow up after 15 days patient was alright and INR was 2.3. Then patient follow up every month and follow up USG(abdomen+pelvis) s/o-Recanalisation in IVC thrombosis.

Figure 1

Figure 2

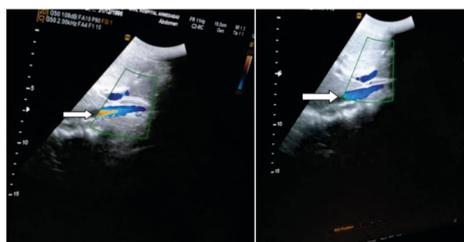
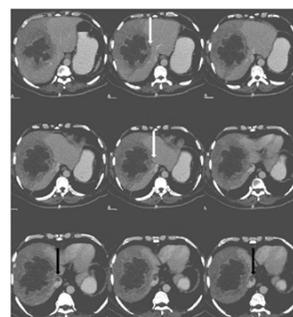


Figure 1: Showing IVC thrombosis

Figure 2: Showing recanalization

CT plates showing IVC thrombosis



DISCUSSION

Though amoebic and pyogenic liver abscesses are commonly encountered in India, IVC thrombosis secondary to liver abscess is quite rare. As IVC thrombosis is life-threatening, it requires high clinical suspicion followed by CECT in appropriate clinical setting. Rupture of the abscess into a vascular IVC, predisposed to localized luminal thrombosis which may act as a source of pulmonary emboli and septicemia. Vascular complications in liver abscess should be considered in patients with fever, tender liver or signs of portal hypertension, more so in patients with diabetes. CECT is an excellent method for diagnosing liver abscess as well as its complications. Ultra-sonography may also be helpful in detection and characterization of venous extension, especially

for follow up. Echocardiography is useful in assessment in cases of intracardiac extension. Treatment includes conservative management with antibiotics, aspiration, anticoagulant. Venography is important when transluminal interventional treatment such as filter insertion or venoplasty is considered.

CONCLUSION:

Whenever a liver abscess is seen adjacent to a vascular channel or in the caudate lobe, it is imperative for the radiologist to look for vascular complications, as venous involvement may not be very symptomatic in some cases. Treatment of the abscess improves the recanalization of thrombosed vessels with anticoagulants. If abscess is drained then it improves the recanalization of thrombosed vessels even without anticoagulation.

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