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Is it APLA Causing Portal Vein Thrombosis with Portal Cavernoma ?

KEYWORDS: portal vein thrombosis Antiphospholipid antibody syndrome

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INTRODUCTION :

Portal vein thrombosis (PVT) is the blockage or narrowing of the portal vein by a thrombus. It is relatively rare and has linked with presence of underlying liver disease or prothrombotic disorders. (APLA –antiphospholipid antibody syndrome).However, no cause is identified in more than 25% of patients. Since lot of patients are asymptomatic and diagnosis is usually made in presence of complications. Abdominal pain is an interesting presentation of portal vein thrombosis, such as the patient presented in following case report.

CASE HISTORY:

A 39 year old female presented with complaints of abdominal distension & pain & black colored stool for 9 months and with no comorbidities. Past history of operation done for renal stones. No history of any abortions. she is well evaluated with history, physical examination, and various investigations which included BT,CT,PT/INR, usg abd ,Doppler, CT abd, UGI scopy , acl antibodies IgG and IgM,β2GP1-IgM,ANA.

EXAMINATION:

NO pallor, icterus, cyanosis,clubbing,lymphadenopathy,edema. JVP-normal
BP : 120/80mmHg

PR: 80/min

RR : 18/min

Temperature : afebrile

CVS: s1+ s2+ No murmur

P/A: Tenderness present in Right hypochondrium & epigastrium

no organomegaly,

RS :normal vesicular breath sounds heard bilaterally

No added sounds

CNS : NFND

INVESTIGATIONS:

ULTRASONOGRAMG ABDOMEN : showing portal vein replaced by multiple collaterals (peripancreatic & omental collaterals, splenorenal, splenoepigastric colleraterals) & multiple dilated & tortuous vessels noted in the portahepatis region proceeded by portal vein.

PORTAL VENOUS DOPPLER :

showed the caliber of main ,right & left portal veins are significantly decreased with absence of flow due to chronic thrombosis & periportal collaterals seen & largest collateral measuring 8mm in diameter & velocity 15cm/sec.

CECT ABDOMEN:

chronic thrombosis of the portal vein & superior mesenteric vein with cavernous malformation of the portal vein & multiple collaterals and atrophy of lateral segment of left lobe of liver.

UPPER GASTROINTESTINAL SCOPY:
erythematous gastritis, no varices .
ANA: negative,
PROTEIN S AND C : normal
APLA IGM & B2-GPI-IGM : positive.
Repeat APLA IgG and IgM, β2GP1-IgM are positive after 12 weeks.

Medical gastroenterology opinion obtained- suggested chronic Portal vein thrombosis $2^0$to APLA & advised to maintain INR between 2 to 3 & patient got discharged with T.pantoprazole 40 mg OD & syrup mucaine gel 5ml TDS & T.warfarin 3mg OD (at the time of discharge INR is 2.4)

DISCUSSION :

This is a case report of APLA causing portal vein thrombosis with portal cavernoma. APLA causing thrombosis incidence is 11%. In patients with APLA & venous thrombosis INR must be maintained 2-3.

Portal vein thrombosis refers to the development of thrombosis within the extra-hepatic portal venous system draining into the liver. It has been classified into 4 anatomic groups:

(1)thrombosis confined to the portal vein beyond the confluence of the splenic and superior mesenteric vein (SMV);
(2)extension of thrombus into the SMV but with patent mesenteric vessels;
(3)diffuse thrombosis of splanchnic venous system but with large collaterals;
(4)extensive splanchnic venous thrombosis but with only fine collaterals

Based on this classification, our patient had category 2 portal vein thrombosis.

- In patients developing heparin induced thrombocytopenia drugs such as fondaparinux 7.5 mg s/c daily (or) rivaroxaban 10mg OD can be given.
- Portal vein thrombosis is a rare occurrence of APLA.

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- It is important that serum antiphospholipid antibodies should be investigated in patients with portal vein thrombosis of unexplained etiology and also in patients with unexplained signs of intestinal angina.
- Portal vein thrombosis develops in a variety of hypercoaguable states like protein C Protein S, or antithrombin III deficiency , including APLA

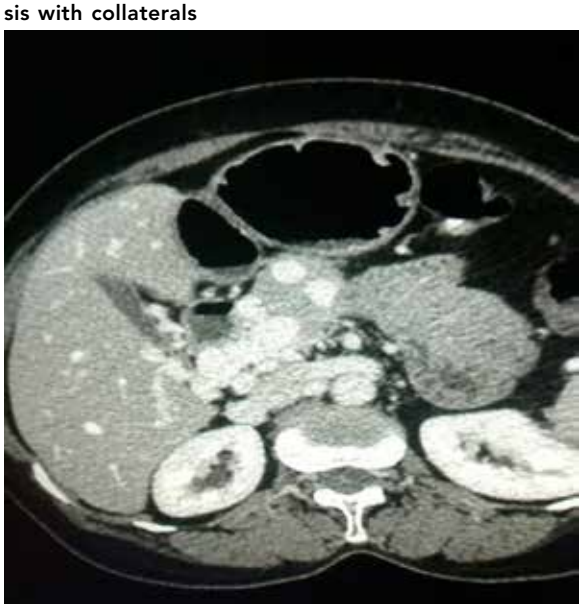
Treatment of patients with APLA and portal vein thrombosis is difficult because of risk of bleeding and recurrent thrombosis if they do not receive appropriate life long term anticoagulation therapy

Treatment of associated risk factors like HTN, hypercholesterolemia and avoidance of prolonged immobilization should be taken care.

CONCLUSION:

Portal vein thrombosis is a rare disease, but our understanding of this disorder has improved during the last few years. The presence of PVT should be considered as a clue for prothrombotic disorders, liver disease, and other local and general factors that must be carefully investigated. Early anticoagulation seems to restore the vascular permeability in the majority of the cases. The management of possible complications like varices, portal hypertension, and biliary complications via endoscopic surveillance is key. It is hoped that this case report will help increase awareness of the complexity associated with portal vein thrombosis among the medical community.

figure - CECT abdomen -showing portal vein thrombosis with collaterals



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