Letter To Editor

Hyperhomocystenimia with Inferior vena cava thrombosis.

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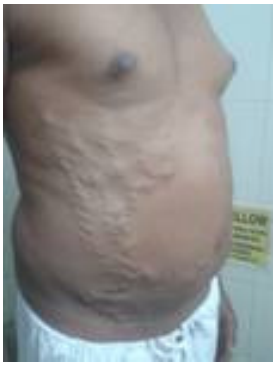
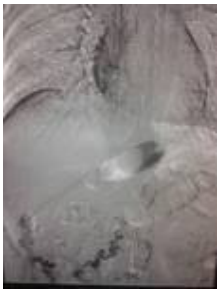
Dear Sir,

Case 1: A 35 year old man presented to us with chief complains of swelling in both lower limbs on and off since 6 months. There was no history of dyspnea, PND, orthopnea, cough, urinary complaints, claudication. He was non alcoholic, non hypertensive and non diabetic. His general physical examination was normal. Per abdominal examination revealed dilated veins predominantly on the lateral aspect of the abdominal wall with a flow below upwards suggestive of inferior venae caval obstruction. (Photograph 1) On investigations, routine hemogram, coagulation profile was normal. His Liver and kidney function tests were normal.



Photograph 1

X ray chest and USG abdomen was normal. On Doppler ultrasound venous collaterals were noted in hepatorenal area. IVC venogram revealed partial thrombosis of common iliac vein with chronic occlusion of infra renal IVC with multiple collaterals draining lower limbs into SVC and directly in to right heart. (Photograph 2) A routine thrombophilia profile was normal. Serum homocystine level was 29.65 µmol/L. (normal < 15 µmol/L). CT abdomen was not done because of low affordability of patient. The patient was treated with with pyridoxine, folic acid, and vitamin B12 supplementation and tablet Warfarin 5 mg per day. Patient was discharged with a INR of 2.5 and is awaiting follow up after 2 weeks.



Photograph 2

Case 2: A 23-year old patient who presented with an IVC / bilateral iliac vein thrombosis thought to be secondary to a retroperitoneal haematoma of benign origin. Case-B was a 25-year old patient diagnosed with a significant IVC / bi-iliac / bi-femoral venous thrombus secondary to a previously undiagnosed congenital IVC malformation. Treatment options in the case of IVC thrombus without anatomical variance include anticoagulation, mechanical thrombectomy, systemic thrombolytic therapy, transcatheter regional thrombolysis, pulse-spray pharmacomechanical thrombolysis and angioplasty1,31. There is no specific literature describing the ideal duration of anticoagulation in these instances, however, case evidence identifies a trend toward treatment for a minimum of one year with the interplay of hypercoagulability disorders needing to be factored into any decision. Surgical reconstruction of the IVC and bypass of an aberrant section are both recognised modalities reserved for the most severe cases and are associated with morbidity and mortality risk32. Endovascular stent placement in combination with angioplasty is recommended in the cases of residual stenosis and chronic IVC occlusion32.

In the case of IVC thrombus associated with an aberrant IVC, with no other predisposing factors, treatment involves anti-coagulation. The duration of this treatment is widely debated with no extensive literature to provide an evidence-based approach. Dean *et al* take a similar view to us, that a caval anomaly is a permanent risk factor for venous stasis and thrombosis and that anticoagulant treatment should be lifelong21.

Discussion: The etiology of inferior vena cava (IVC) thrombosis are myriad. The most important possibilities that should be considered are, hypercoagulability related to haematological or neoplastic abnormalities, venous stasis secondary to extraluminal pressure from tumours or inflammatory processes and vessel injury due to trauma. [1]

The clinical history is usually vague. Patients complain of lower back and limb pain, swelling of the lower limbs, and even pyrexia, which can mislead to diagnose vertebral disease, arterial disease or even cardiac condition. Dilatation of superficial abdominal veins especially in the lateral aspect of the abdominal wall with a flow below upwards is the clinical hallmark, that distinguishes it from portal hypertension, where the flow will be hepatofugal.

IVC thrombosis is associated with a significant acute and chronic morbidity. A high index of suspicion is warranted. When IVC thrombosis is diagnosed in young patients,detailed evaluation for thrombophilia profile for , Factor V Leiden, prothrombin gene mutation, low protein S levels, high homocysteine concentration,

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methylenetetrahydrofolate reductase gene mutation and antiphospholipid antibodies is required, along with ultrasonic search for the status of other venous channels especially femoral and iliac veins. Usually these veins are also affected. CT or preferably MRI imaging, are required to delineate IVC anatomy and ascertain extent of the thrombus. Although invasive therapeutic modalities exist, long-term and commonly life-long anticoagulation is often required. [2,3] Hyperhomocysteinemia was the risk factor in our case. A high level of homocysteine causes endothelial injury, which leads to vascular inflammation, which in turn may lead to atherogenesis.[4] Deficiencies of the vitamins pyridoxine (B_6), folic acid (B_9), or B_{12}can lead to high homocysteine levels. Supplementation with pyridoxine, folic acid, B_{12}, or trimethylglycine (betaine) reduces the concentration of homocysteine in the bloodstream.

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