Superior Mesenteric Vein Thrombosis due to Hyperhomocysteinemia

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We describe a case of thrombosis of superior mesenteric veins due to hyperhomocysteinemia with no other risk factor.

A 38 year lady presented with abdominal pain since 3 days. She had abdominal distension since 2 days with vomiting, constipation with no other medical or surgical history. On presentation, patient had tachycardia with normal blood pressure. Abdomen was guarded and tender without any liver dullness obliteration. Plain radiograph of erect abdomen revealed bowel with gas. Ultrasonography of the abdomen was normal. CECT abdomen revealed thrombosis of superior mesenteric vein with ischemia of the small bowel with no free gas in peritoneum.

Patient was investigated for hypercoagulable state which revealed raised serum homocysteine levels of 70. In view of stable vital parameters and maintained urine output, patient was treated with conservative management with nasogastric aspiration, adequate hydration, and low molecular heparin.

Patient continued to be vitally stable, and gradually abdominal distension settled over 4 days. Patient passed liquid stools on day 5 with no Malena. Patient was started on liquids on day 6 with folic acid, homocheck and titrated warfarin dosage. She was maintained on liquid diet for 3 weeks followed by soft diet. Serum homocysteine was repeated after 2 months which showed marginally raised levels. A repeat CECT abdomen after 3 months revealed recanalization of the SMV.

This is the rare reported case of concomitant thrombosis of the superior mesenteric veins with hyperhomocysteinemia, no other risk factors. Prompt and sustained anti-coagulation with vitamin B_{12}, together with homocysteine normalization, prevent progression to bowel gangrene.[1,2] This report highlights the fact that hyperhomocysteinemia itself as a significant cause to lead to lethal multiple abdominal vein thrombosis.[3] It underscores the need to (1) consider Mesenteric thrombosis in the differential diagnosis of epigastric abdominal pain, (2) perform a complete thrombotic work-up to elucidate metabolic abnormalities that could be contributing to a pro-thrombotic state.

References

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