Idiopathic thrombosis of portal, splenic and mesentric vein leading to gut gangrene:

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ABSTRACT

Portal and splenic vein thrombosis are uncommon and often asymptomatic. Sometimes catastrophic consequences occur when it involves mesenteric vein leading to mesenteric ischemia and subsequently gut gangrene. It poses surgical and vascular emergency. Early diagnosis and intervention is the key to prevent gut gangrene and mortality. We report an unusual case of 38 year man with idiopathic thrombosis of superior mesentric, splenic and portal vein with gangrenous changes in the jejunum. Diagnosis was established by colour doppler ultrasonography and CECT abdomen. Thorough evaluation of the patient revealed no etiological factor leading to thrombosis. Patient was successfully treated with surgery and was put on anticoagulants in postoperative period.

Keywords: thrombosis, mesenteric ischaemia, gut gangrene

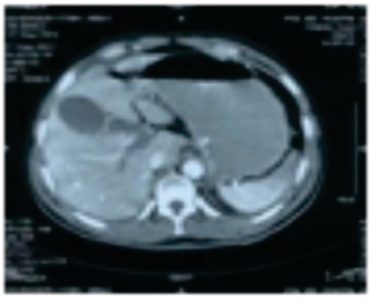
INTRODUCTION

Mesenteric vein thrombosis once believed to cause a few cases of mesenteric ischaemia and diagnosed during laparotomy or at autopsy. With advent of highly specific and sensitive imaging modalities, the scenario is changed. It is responsible for 5-15% of all cases of acute mesenteric ischaemia.[1] In majority of the cases, thrombosis is believed to be due to some underlying causes such as hypercoagulable states due to neoplasm, polycythemia vera, protein C & S deficiency, antithrombin III deficiency and abdominal surgery. However, 20-40% of the cases are considered to be idiopathic.[2] It has a non specific clinical presentation and symptoms are out of proportion to the physical finding. It must be suspected when acute abdominal symptoms develop in a patient with prior thrombotic episode, known thrombophilic disorder or other causes of acute abdomen have been ruled out. Early diagnosis and rapid aggressive treatment is plays a key role in avoiding bowel necrosis and subsequent mortality.

CASE REPORT

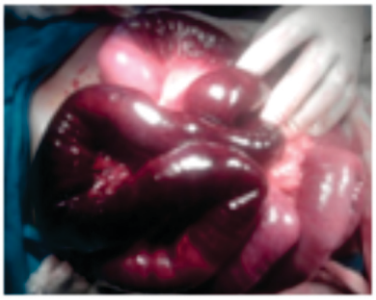
A 38 years-old man presented with complains of worsening upper abdominal pain and billous vomiting for three days. His personal and family history was noncontributory. There was no history suggestive of DVT, or known thrombophilic disorder or previous surgery. There were no signs and symptoms suggestive of inflammatory bowel disease, or cirrhosis. On examination patient was conscious and oriented. His vitals were: temperature 99.4°F, pulse rate 114/ minute, respiratory rate 19 breaths/minute, BP 108/60 mm Hg. Abdominal examination shows guarding and rebound tenderness with hypoactive bowel sounds. Lab results showed leucocytosis (13× 10^9cells/L). Other investigations including serum electrolytes, amylase, lipase, and liver and renal function tests were within normal limits. ECG and chest X-ray were also normal. Colour Doppler ultrasonography showed thickening of gut loops in the region of small intestine with echogenic thrombus into SMV, portal vein and splenic vein. CECT abdomen showed echogenic thrombus in left branch of SMV, in splenic and portal vein (Fig.1).

Fig.1:CECT Image showing intrahepatic portal vein thrombosis



Loops of jejnum were dilated, thick walled and nonenhancing. Spleen also shows an area of hypodensity. A provisional diagnosis of gut gangrene was made and immediate explorative laparotomy was planned. At laparotomy, there was approximately four and half feet of gangrenous jejunal and adjacent ileal segment 10 cm distal to DJ junction (Fig.2).

Fig.2: Intraoperative photographs showing gangrenous jejunal loops



500 ml of altered blood was present in the peritoneal cavity. After through peritoneal lavage, gangrenous segment of gut was resected and jejuno-ileal anastomosis with feeding jejunostomy was done. Patient was started on low molecular weight heparin (parnaparin) with a dose of 7000 I.U/day from the immediate postoperative period followed by oral anticoagulation (Nicoumaolone 2mg/day) with target INR 2-3. Patient was investigated for cause of thrombosis. Thrombophilic screening including PT and aPTT, complete blood count, bleeding time, clotting time, protein C & S, antithrombin-III, Leiden factor-V, homocystiene level, antiphospholipid antibody and peripheral blood film was ordered and it reflects all these parameters within normal limits. So a diagnosis of idiopathic mesenteric vein, portal and splenic thrombosis leading to gangrene of the jejunum and adjacent ileum was made. The patient had an uneventful recovery. On follow-up the patient is on routine evaluation of the PT, aPTT and international normalized ratio to ensure the adequacy of oral anticoagulation therapy. He is on oral anticoagulation with Nicoumaolone 2mg/day and is well after a follow up of six months.

DISCUSSION

Combined thrombosis involving mesenteric vein and the portal vein is rare; difficult to diagnose and can be fatal.[3] Stricture and bowel necrosis with peritonitis due to transmural intestinal infarction may complicate the course. Common causes include liver disease, pancreatitis, inflammatory bowel disease, cancer, sepsis, an underlying myeloproliferative disorder, surgery or trauma, and systemic thrombophilia.[4]

Virchow's triad of stasis, epithelial injury and hypercoagulability is a prerequisite for Mesenteric vein thrombosis but ischaemic changes in gut depends on location, extent & rapidity of thrombus formation because of collateral develop in chronic Mesenteric vein thrombosis. Therefore chances of gut infarction are less common as compared with acute Mesenteric vein thrombosis. Acute thrombus formation may be due to local factors, splenectomy, pancreatitis which generally involve large splanchnic veins or due to hypercoagulable state which involve intramural venules, vasa recta or venous arcade. Whatever may be the mechanism, but infarction due to

mesenteric vein thrombosis require involvement of venous arcade and vasa recta which leads to complete occlusion of venous return. Because of inadequate venous return, intestinal walls become engorged with blood, cyanotic, ischaemic and ultimately progress to transmural infarction.[5]

A clinical feature depends on location and timing of thrombus formation. Acute form is suggested by colicky abdominal pain which is initially out of proportion to the physical examination of the abdomen.[6] Signs of peritonitis is a late feature and suggests intestinal infarction. No signs or symptoms are specific for acute form. A high index of suspicion is necessary for early diagnosis before the infarction occurs. The most common site involved is the ileum or the jejunum, followed by colon and duodenum.[1,7] Small gut involvement is more common than the large gut because of collateralization in the systemic circulation via left renal vein and hemi azygous system.[2]

Mesenteric vein thrombosis should be diagnosed before intestinal infarction develops so that gut can be salvaged. Earlier diagnostic angiography or laparotomy were used for diagnosis in high index suspicion cases but now with advent of newer non invasive diagnostic modality the task has become easy. CECT abdomen is the investigation of choice to diagnose this condition at an early stage. Although accuracy of angiography is better than CT, it is reserved for patients with non conclusive CT results, yet a clinical suspicion of Mesenteric vein thrombosis.

The early initiation of anticoagulation using unfractionated heparin or low molecular weight heparin has been shown to minimize the risk of serious complications,[3] nonetheless spontaneous resolution of extensive superior mesenteric and portal vein thrombosis has been also reported.[8] The aim of treatment is to prevent the development of gut infarction and if the infarction has already developed, resection of infected segment is the only choice left. Symptomatic patients with early diagnosis and without any evidence of gut infarction can be treated with thrombolysis by using streptokinase, urokinase or tissue plasminogen activator.[9] Mechanical thrombectomy is a non pharmalogical alternative for patients who pose high risk for thrombolytic therapy. A combined approach using catheter-directed thrombolysis and mechanical thrombectomy has also been tried successfully.[10] But when intestinal infraction is suspected by clinical or radiological findings, immediate exploration of abdomen is mandatory and once after exploration diagnosis is confirmed intra operative heparinistaion should be done. Short non-viable segments of intestine are resected. But if viability of long ischemic segments is questioned, a planned 'second-look' operation during the first 12–48 postoperative hours, such that any intestinal segments of questionable viability may be reassessed, thereby limiting the extent of the initial resection.[11] If the cause of thrombosis is a thrombotic disorder or idiopathic the duration of anticoagulation will be life long. Otherwise, it may be limited to six months to one year.[12]

CONCLUSION

A strong clinical suspicion remains the key to early diagnosis which is confirmed by CT scan. Early use of anticoagulant therapy and early decision can improve the outcome and decrease mortality and morbidity. It is also concluded that all patients presenting with apparently unexplained combined thrombosis of portal,

splenic and mesenteric vein should be investigated for the presence of a hypercoagulable state. Anticoagulation should be considered in all patients with this diagnosis and should be a lifelong therapy in those with an underlying thrombophilia or idiopathic thrombosis.

AUTHOR NOTE

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