An Unusual Case of Acute Abdomen

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ABSTRACT

Acute superior mesenteric vein thrombosis is an uncommon but life-threatening disease condition that is not often diagnosed early. This is a case of a 28-year-old male who presented with a 4 day history of nausea, vomiting, episodic abdominal pain and melena. Tenderness and diminished intestinal sounds were the prominent abdominal physical findings. Computed tomography confirmed superior mesenteric vein thrombosis. He was initiated on heparin and then later transitioned to anticoagulants. Surgical resection, site-directed thrombolysis and thrombectomy are other options of treatment for acute superior mesenteric vein thrombosis. Early diagnosis and subsequent treatment resulted in resolution of the thrombus with no long-term sequelae.

Key words : Venous thrombosis, Superior mesenteric vein, portal vein, intestinal ischemia

Introduction :

Thrombosis of the superior mesenteric vein (MVT) is an uncommon but potentially life-threatening disorder. It's cause may be clear, as in patients with adjacent neoplasm or inflammation or in patients with cirrhosis or portal vein thrombosis. Alternatively, the etiology and hence therapy may be more difficult to determine if no definite cause is seen. Mesenteric venous thrombosis (MVT) is a rare but potentially lethal form of mesenteric ischemia. MVT must be distinguished from arterial and nonocclusive types of mesenteric ischemia and accounts for 5% to 15% of all cases of mesenteric ischemia. Patients may have evocative signs, such as abdominal pain, nausea, or vomiting. However, a clinical diagnosis is often difficult because abdominal symptoms are nonspecific.[1] Primary MVT accounted for 25% to 55% of cases in early studies, but recent reports show a decline in primary MVT because of improvements in the diagnosis of hypercoagulable states.[2] Advances in new imaging techniques also have enabled early recognition of this disease without or before laparotomy.[3-5] There is no consensus about initial treatment in the management of MVT. Some authors have proposed an aggressive surgical approach[6] whereas others have advocated medical therapy.[7]

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Case Report :

A 28-year-old male presented with 4 days history of abdominal pain, vomiting, and melena. Abdominal pain was acute in onset localized to umbilical and left hypochondriac region, severe episodic colicky type and worsened with eating. He denied any fever or chills. He had persistent melena during the hospital stay. There was no history of fever, bleeding per rectum, hematemesis, jaundice or any urinary complaints. Past medical history was not contributory. The patient was an occasional alcoholic. Family history was not significant.

On examination Pulse was 98/min regular, normal volumic, peripheral pulsation well felt, respiratory rate was 24/min , BP 130/80 mmHg, the abdomen was soft but tender in epigastric, umbilical and left hypochondriac region. There was no guarding, rigidity, organomegaly or free fluid in abdomen. The patient was provisionally diagnosed as acute abdomen ? Gastritis ?? acute pancreatitis. Patient was investigated as CBC : TLC - 16,700 Hb - 13.5 g/dl platelet - 1.91 lakh; LFT, KFT were normal; S. amylase - 51 U/L; S.lipase - 250 U/L; INR - 1.1; Stool for occult blood was positive; USG abdomen was s/o Portal vein thrombosis with periportal collaterals with mild splenomegaly with splenic vein thrombosis and perisplenic collaterals along with oedematous small bowel loops with minimal vascularity in left hypochondriac and mild ascites; ANA was 0.98 (negative); Homocysteine was high - 7.7 (normal < 30). CT abdomen was s/o Thrombosis in right and left branch of portal, main portal, proximal splenic and proximal superior mesenteric

vein associated with periportal, peripancreatic, perisplenic collaterals With early ischemic changes involving jejunal loops in the form of long segment bowel wall thickening and decreased wall enhancement. Hepatic veins, arteries, IVC, aorta and its branches were normal. There was mild splenomegaly mild ascites. Thrombophilia profile was not done as the patient immediately started on anticoagulants.

The final diagnosis was superior mesenteric vein thrombosis with portal and splenic vein thrombosis with early jejunal wall ischemia. The patient was managed with heparin and later on switched to nicoumalone with higher antibiotics meropenem, analgesic tramadol, I.V. fluids. The opinion of Intervention radiologist, general surgeon, cardiovascular and thoracic surgeon, Gastroenterologist was sought and all advised conservative management with anticoagulants and no intervention. During the course of illness, patient had frequent episodic pain and melena. Initially, bowel was rested and later on with the resolving bowel wall edema and ischemia patient was started on liquid supplements. Melena subsided with normal stools. Patient was discharged on nicoumalone and advised regular monitoring of INR

Discussion :

Acute MVT is defined as a thrombus of the mesenteric vein with bowel infarction, regardless of its severity. MVT has been variably estimated to account for 0.002-0.06% of all inpatient admissions,[8,9] 0.01% of all emergency surgical admissions,[10] and less than one in 1000 laparotomies for 'acute abdomen'.[11]

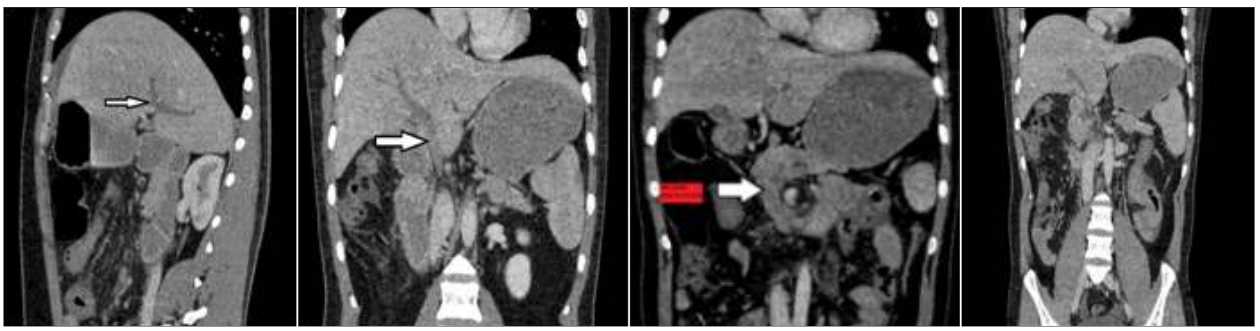
The location, extent and rapidity of thrombus formation, however, determine the severity of intestinal injury. Different pathophysiologic mechanisms have been postulated for acute thrombus formation because of local factors, systemic hypercoagulable states. Whichever mechanism is invoked, however, infarction from MVT requires the involvement of the venous arcades and vasa recta leading to complete occlusion of venous return[12]. Risk factors may be divided broadly into heritable and acquired thrombophilias (e.g. prothrombin G 20210 mutation, MPD); hypercoagulable states related to systemic disorders (e.g. nephrotic syndrome, malignancy); and local intra-abdominal processes (e.g. splenectomy, diverticulitis, trauma).

Doppler ultrasound is widely available, rapidly performed and non-invasive has good specificity (100%) but poor sensitivity (70-90%) for MVT.[13]

CT of the abdomen with adequate portal venous phase contrast is a highly sensitive and specific, readily available test which has emerged as the most reliable modality to diagnose MVT.[14]

Immediate heparinization upon diagnosis of MVT - intraoperatively and even for patients who present with bleeding because of MVT-induced ischemia - is currently accepted as standard therapy.[15]

Over the last two decades, various systemic and percutaneous thrombolytic techniques have been applied to the treatment of acute MVT, albeit mostly in case reports and small series.[16] When acute large vessel thrombosis is identified, thrombectomy is useful,[17] as mechanical thrombectomy may result in a rapid and durable venous patency without the need



A and B : Noncontrast opacification of right and left branches of the portal vein.
C and D: Noncontrast opacification of SMV

for prolonged thrombolytic therapy. When intestinal infarction is suspected by virtue of clinical decompensation or radiologic findings, immediate exploration of the abdomen either by laparotomy or laparoscopy is mandated, and once the diagnosis is confirmed, intraoperative heparinization with intestinal resection should be initiated.

MVT has a better prognosis than do other forms of AMI. In a large systematic review of almost 3700 cases of AMI seen between 1966 and 2002, the overall mortality rate of patients with MVT was 44% compared with 66-89% for patients with arterial embolism, non-occlusive ischemia, or arterial thrombosis.[18] Just as with other forms of AMI, mortality rates in acute MVT are primarily determined by the presence or absence of intestinal infarction.

Conclusion :

Although acute MVT is responsible for only about 3-15% of cases of AMI, a high index of suspicion is needed for accurate diagnosis because of the non-specific nature of its presenting signs, symptoms and laboratory tests. Evidence of infarction mandates emergent surgical consultation and abdominal exploration and / or aggressive intravascular therapy in order to save as much intestine as possible. Patients with thrombophilia should be defined, and long-term anticoagulation tailored to the patient's individual needs. Short-term mortality largely depends on whether infarction is present and has improved over time because of earlier diagnosis and aggressive therapy.

References :

1. Rhee RY, Gloviczki P, Mendonca CT, Petterson TM, Serry RD, Sarr MG, et al. Mesenteric venous thrombosis: still a lethal disease in the 1990s. Journal of vascular surgery. 1994;20(5):688-97.
2. Grieshop RJ, Dalsing MC, Cikrit DF, Lalka SG, Sawchuk AP. Acute mesenteric venous thrombosis. Revisited in a time of diagnostic clarity. The American surgeon. 1991;57(9):573-7; discussion 8.
3. Abdu RA, Zakhour BJ, Dallis DJ. Mesenteric venous thrombosis--1911 to 1984. Surgery. 1987;101(4):383-8.
4. Harward TR, Green D, Bergan JJ, Rizzo RJ, Yao JS. Mesenteric venous thrombosis. Journal of vascular surgery. 1989;9(2):328-33.
5. Hickman A, Wilson SK, Stein M, Lewis J. Portal and superior mesenteric venous thrombosis following splenectomy. Journal of the Tennessee Medical Association. 1991;84(7):329-30.
6. Loring LA, Panicek DM, Karpeh MS. Portal system thrombosis after splenectomy for neoplasm or chronic hematologic disorder: is routine surveillance imaging necessary? Journal of computer assisted tomography. 1998;22(6):856-60.
7. Yu JS, Bennett WF, Bova JG. CT of superior mesenteric vein thrombosis complicating periappendiceal abscess. Journal of computer assisted tomography. 1993;17(2):309-12.
8. Ottinger LW, Austen WG. A study of 136 patients with mesenteric infarction. Surgery, gynecology & obstetrics. 1967;124(2):251-61.
9. Boley SJ, Kaleya RN, Brandt LJ. Mesenteric venous thrombosis. The Surgical clinics of North America. 1992;72(1):183-201.
10. Hansen HJ, Christoffersen JK. Occlusive mesenteric infarction. A retrospective study of 83 cases. Acta chirurgica Scandinavica Supplementum. 1976;472:103-8.
11. Harnik IG, Brandt LJ. Mesenteric venous thrombosis. Vascular medicine. 2010;15(5):407-18.
12. Johnson CC, Baggenstoss AH. Mesenteric vascular occlusion; study of 99 cases of occlusion of veins. Proceedings of the staff meetings Mayo Clinic. 1949;24(25):628-36.
13. Haddad MC, Clark DC, Sharif HS, al Shahed M, Aideyan O, Sammak BM. MR, CT, and ultrasonography of splanchnic venous thrombosis. Gastrointestinal radiology. 1992;17(1):34-40.
14. Morasch MD, Ebaugh JL, Chiou AC, Matsumura JS, Pearce WH, Yao JS. Mesenteric venous thrombosis : a changing clinical entity. Journal of vascular surgery. 2001;34(4):680-4.
15. Amitrano L, Guardascione MA, Scaglione M, Pezzullo L, Sangiuliano N, Armellino MF, et al. Prognostic factors in noncirrhotic patients with splanchnic vein thromboses. The American journal of gastroenterology. 2007;102(11):2464-70.
16. Karawi MA, Quaiz M, Clark D, Hilali A, Mohamed AE, Jawdat M. Mesenteric vein thrombosis, non-invasive diagnosis and follow-up (US + MRI), and non-invasive therapy by streptokinase and anticoagulants. Hepato-gastroenterology. 1990;37(5):507-9.
17. Kumar S, Sarr MG, Kamath PS. Mesenteric venous thrombosis. The New England journal of medicine. 2001;345(23):1683-8.
18. Schoots IG, Koffeman GI, Legemate DA, Levi M, van Gulik TM. Systematic review of survival after acute mesenteric ischaemia according to disease aetiology. The British journal of surgery. 2004;91(1):17-27.