

Original Article

Superior Mesenteric Venous Thrombosis: Riyadh Central Hospital Experience

Mohammed K Alam¹, Abdelrahman El-Kayali¹, Abdulmajeed Abdelhamid Mohammed²,
Radwan Taha Galul²

¹Department of Surgery, King Khalid University Hospital, Saudi Arabia

²Department of Surgery, Riyadh Central Hospital, Saudi Arabia

The Kuwait Medical Journal 2001, 33 (1): 18-21

ABSTRACT

Objective: The objective of this study was to analyze our experience with MVT (Mesenteric Venous Thrombosis) with a particular reference to identify the risk factors in our population.

Methods: A retrospective study was conducted on 68 patients treated for mesenteric venous thrombosis at Riyadh Central Hospital (Saudi Arabia) between 1988-1998. The data were collected and analyzed for this study.

Results: There were 68 patients (58 males - 85%) whose final diagnosis was acute mesenteric venous thrombosis (MVT). Their presenting symptoms were pain (n = 68), vomiting (n = 52), constipation (n = 30), haematochezia (n = 11), diarrhea (n = 8), and hematemesis (n = 2). The average duration of pain before hospital admission was 3-5 days. Abdominal tenderness (n = 65) and abdominal distension (n = 33) were the common physical signs. The diagnosis was suspected preoperatively in 24 patients. Contrast enhanced CT scan (n = 24) established diagnosis in all (24 patients). Duplex ultrasound, done on 15 patients was able to establish diagnosis in nine patients.

Of the 61 patients who underwent surgery, 24 were diagnosed preoperatively. The remainder were diagnosed as acute MVT at surgery. Sixteen patients underwent a second-look operation.

All patients received anticoagulation therapy after surgery. Seven MVT patients were treated conservatively with anticoagulation therapy. The majority of our patients (63%) had liver cirrhosis as the main co-morbid factor. There were 18 deaths (26%) due to multiorgan failure, septicemia, pulmonary embolism and DIC (disseminated intravascular coagulation).

Conclusions: MVT should be suspected when a patient presents with vague but persistent acute abdominal symptoms and signs. Suspicion should be high if the patient has liver cirrhosis or prior thrombotic episodes. Contrast enhanced CT is the most sensitive diagnostic test. Most patients present late and will require bowel resection, although MVT can occur without gangrene of bowel. Anticoagulation therapy decreases the recurrence of the disease and improves survival. Mortality is high among these patients.

KEYWORDS: abdominal disease, intestinal ischemia, mesenteric venous thrombosis

INTRODUCTION

Mesenteric venous thrombosis (MVT) is an infrequent but distinct form of intestinal ischemia. Although Elliot^[1] first described intestinal gangrene secondary to mesenteric vascular occlusion in 1895, only since the development of the new imaging techniques have the various forms of the condition been recognized.

MVT makes up 5-15% of all acute mesenteric thrombosis cases^[2,3,4]. Despite modern diagnostic tools and methods, delay in diagnosis is frequent and contributes to the 15-40% mortality^[5,6,7]. The primary reasons for delay in diagnosis are the non-specific abdominal signs and symptoms of MVT^[5,7]. Recent discoveries, however, have altered our concepts of the disorder, and we now recognize that thrombosis of superior mesenteric vein can develop slowly with pain but no

intestinal infarction or acutely with the classic presentation^[6].

The increasing use of CT scan and Duplex ultrasound in the evaluation of abdominal disease has facilitated diagnosis of MVT. These imaging studies have also revealed MVT as incidental findings in patients without acute symptoms of bowel ischaemia^[8,9,10]. Options for treatment may range from observation to urgent exploration with resection of the infarcted bowel.

Anti-coagulation is suggested by most authors^[4,7,11]. Experience at any one institution with the management of MVT is limited, only case reports or small series exist in the literature^[3,12,13]. We present our experience in the diagnosis and management of patients treated for mesenteric venous thrombosis in the past 10 years at this hospital.

Address correspondence to:

Dr. Mohammed K. Alam, FRCS, Associate Professor and Consultant General Surgeon, Department of Surgery, King Khalid University Hospital, PO Box 2925, Riyadh 11461, Kingdom of Saudi Arabia.

PATIENTS AND METHODS:

We retrospectively reviewed the clinical records of 68 patients treated at Riyadh Central Hospital, during 1988-1998 with a diagnosis of MVT. The records included patients who were admitted with a clinical diagnosis of mesenteric ischaemia or were subsequently found to have MVT at surgery.

Patients whose bowel ischaemia were not due to MVT were excluded from the study. The data collected in this study included age, gender, clinical presentations, investigations, operative findings, post-operative courses, complications and final outcomes. Analysis of these data forms the basis of this study.

RESULTS

During a ten-year period (1988-1998) 68 patients were treated for MVT. Their ages ranged from 20-81 years with a mean age of 44.6 ± 11 years. There were 58 males (85%) and 10 females.

All patients (n = 68) presented with abdominal pain. The duration of pain before admission ranged from 1 day to 4 weeks with an average duration of 3-5 days. Vomiting was the next most common symptom (n = 52). Other presenting symptoms were constipation (n = 30) and diarrhea (n = 8) (Table 1). Abdominal tenderness was present in all, while distension was noted in 33 patients. Rectal examination was positive for blood in 11 patients. Most patients (n = 48) were febrile ($>37.4^\circ\text{C}$) upon admission (Table 1). Abdominal X-ray showed air fluid levels in eight patients. Leukocytosis ($>15,000/\text{cmm}^3$) was present in 40 patients. Contrast enhanced abdominal CT scan was done on 24 patients; all were positive for MVT. CT findings suggestive of MVT were small bowel wall thickening, thrombus or filling defect in superior mesenteric vein or portal vein (Figs. 1 & 2).

Ultrasound with or without color duplex evaluation of the mesenteric and portal veins was obtained in 15 patients. Thrombus or absence of flow in the mesenteric vein or portal vein was demonstrated in nine patients. A total of 61 patients were operated; 24 with a preoperative diagnosis of MVT, and the others with the diagnosis of peritonitis (n = 20), acute appendicitis (n = 6), intestinal obstruction (n = 8) and perforated duodenal ulcer (n = 3). Sixteen patients had a second look surgery. A total of 77 surgical procedures were done. No recurrence of thrombosis was seen on second look surgery. Seven patients (with no features of peritonitis) were treated conservatively with heparin and oral anticoagulation.

At operation, the findings suggestive of MVT were presence of abundant sero-sanguinous ascitis, congestion and edema of the intestine, segmental

Table 1

Presenting features of mesenteric venous thrombosis (n = 68)

Symptoms	No. of Patients	(%)	Signs	No. of Patients	(%)
Pain	68	100	Abdominal tenderness	68	100
Vomiting	52	76	Temperature ($>37.4^\circ\text{C}$)	48	71
Constipation	30	44	Abdominal distension	33	49
Hematochezia	11	16	Guarding and rebound tenderness (Peritonitis)	11	16
Diarrhea	8	12	Bleeding per rectum (P/R exam)	11	16
Hematemesis	2	3			

Table 2

Associated diseases in patients with MVT

Associated Diseases	No. of Patients	(%)
Liver cirrhosis	* 43	63
Diabetes Mellitus	12	18
Heart diseases	7	10
Deep venous thrombosis	3	4
Chronic renal failure	1	1

* 11 patients previously had Hassab's gastro-esophageal devascularization for bleeding esophageal varices.

involvement of bowel, and thrombosis of superior mesenteric vein arcades with patent superior mesenteric artery. The estimated length of infarction of the intestine ranged from 30 cm to 1 meter. The mid-portion of the small bowel was the most commonly involved segment. All patients received I.V. heparin after surgery followed by warfarin for 3-6 months. Liver cirrhosis was the most commonly associated disease in 63% (n = 43) of the patients, 16% (n = 11) of whom had previously undergone Hassab's gastro-esophageal devascularization. The list of other associated diseases is presented in Table 2. Hospital stay ranged from 2 to 28 days.

There were eighteen deaths (26%) in the study group, two of whom had second look operations. There was no mortality in the conservatively treated group. Most of the deaths were due to multiorgan failure, septicaemia, chest infection and DIC. Four patients were suspected to have died from pulmonary embolisms.

DISCUSSION

Mesenteric venous thrombosis is an infrequent surgical emergency. The number of patients with primary MVT is continuously decreasing. An increasing number of patients are now diagnosed secondarily as MVT due to the increased awareness of disorders predisposing to MVT^{16,14}. Co-morbid states, such as hyper-coagulability, cirrhosis, splenomegaly, cancer, trauma, pancreatitis, or

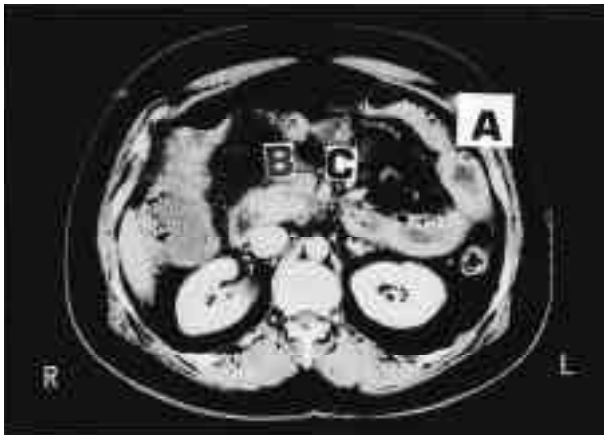


Fig. 1: CT scan showing (a) Thickened wall with free gases in small bowel, (b) Filling defect in SMV, (c) Superior mesenteric artery

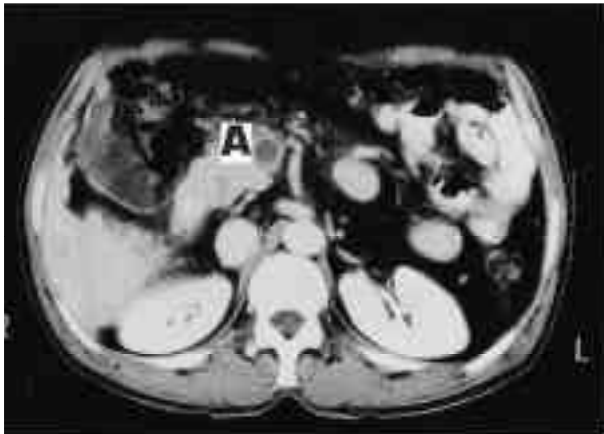


Fig. 2: CT scan showing filling defect (a) In SMV.

diverticular disease, are responsible for most cases of MVT^[14]. Deficiency of physiologic anticoagulants, such as antithrombin III, protein C, and protein S are known to be etiologic factors of MVT. Protein C and S production can be decreased with any form of hepatic insult and as a result of consumption^[15,17].

Most patients in our study ($n = 43$, 63%) had liver cirrhosis as the main predisposing factor. Eleven of them had developed MVT at varying intervals following Hassab's gastro-esophageal devascularization and splenectomy for bleeding esophageal varices. However, Abdu et al^[5] found that in 81% of their patients, MVT was secondary to diseases such as thrombophlebitis (15%), inflammatory visceral disease (14%), post abdominal surgery (12%), and liver cirrhosis (11%).

The physiopathologic mechanism of primary mesenteric venous thrombosis involves proximal propagation of thrombus originating in the distal veins of the vascular arcades with ultimate occlusion of all collateral venous channels involving the arcuate and the *vasa recta* and resulting in hemorrhagic infarction of intestine^[6]. The time of occurrence of these events correlates

well with the characteristic insidious progression of the disease. This explains why the symptoms of acute MVT are usually less severe than those of acute mesenteric arterial occlusion and, hence, the delay in diagnosis^[18].

Usually patients with MVT present with abdominal pain^[5,12,19,20]. This can be sudden in onset in a few cases but frequently begins insidiously and becomes progressively worse^[5,12,19-21]. In our study, the mean duration of pain before presentation to hospital was 3-5 days. This was similar to the series of Boley, et al^[6]. Others have reported this interval to be two weeks^[18]. Matthews and White^[19] reported that 27% of their patients had pain for more than one month before presenting to hospital. Uncommon presentations such as hematemesis and lower gastrointestinal bleeding can also delay in diagnosis, as seen in some patients of this study.

Physical findings in acute mesenteric venous thrombosis at initial presentation may vary greatly, reflecting the different stages of the disease and different degrees of ischaemic injury. We observed that the initial presenting symptoms were usually out of proportion to the physical signs. Similar to other reports,^[5,6,22] abdominal distension was the most frequent sign present in our patients (50%). Laboratory tests are also less contributory towards making a specific diagnosis of MVT^[6]. Leucocytosis ($>15,000 \text{ cmm}^3$) was present in 60% of our patients. This, along with progressive but vague abdominal symptoms and signs, should raise suspicion of the possibility of intestinal ischaemia due to MVT.

In the past, an objective diagnosis of MVT was difficult to establish. Recently, contrast enhanced abdominal CT scanning has been shown to accurately detect MVT in more than 90% of cases^[23,24]. In our series, contrast enhanced CT scan diagnosed MVT in all patient (100%) who underwent this test.

Miller and Besland^[25] found abdominal duplex scanning as effective as CT scan. In our study, Duplex ultrasound was less sensitive and diagnosed MVT in 9 out of 15 patients (60%). Selective mesenteric arteriography can establish a definitive diagnosis before bowel infarction. It can differentiate venous thrombosis from arterial ischaemia and can provide access for the administration of intra-arterial vasodilators in specific patients. However, we feel that this invasive investigation may be more useful in arterial ischaemia. We did not use this modality during the period of this study.

The decision of surgical exploration is guided by clinical presentation. Patients with a clear picture of peritonitis should have prompt surgery, followed by anticoagulant therapy. A small group of our patients ($n = 7$) who were diagnosed MVT by ultrasonography or CT scan, but had no physical

signs of intestinal infarction, were treated by anticoagulant therapy only. They were closely observed for abdominal signs of ischaemia during conservative therapy. Follow-up ultrasound showed complete recanalisation of mesenteric and portal vein. Boley et al^[6] reported that heparin significantly prolonged survival and lowered recurrence. In this study, all patients received anticoagulant therapy for 3-6 months after surgery.

Sixteen patients in our study group had second surgeries (second look) within 2-3 days of primary operation. Our indications for second look were suspicious viable margins of saved small bowel at initial operation in patients with extensive bowel gangrene and clinical suspicion of recurrent disease. None of these patients needed further resection due to recurrence of the disease. Rhee et al^[22] also recommends second look surgery in selected patients. Mortality in our group was 26% and no recurrent disease, which compares favorably to the 25% recurrence rate and a mortality rate of 50% in some series^[5]. We feel that this improvement may have been due to anticoagulation therapy.

We conclude that MVT is more common than arterial ischaemia in our hospital. Most of these patients are cirrhotics, presenting with 3-5 day histories of persistent but vague abdominal symptoms and signs with leucocytosis. This should raise the suspicion of MVT and an urgent contrast enhanced CT will establish the diagnosis. Most will need surgery, but early cases can be managed by anticoagulation therapy. All patients after surgery will also need anticoagulation therapy. Some of these patients will need nutritional support, such as parenteral nutrition, for long term care.

ACKNOWLEDGMENT

We are grateful to all the Consultant Surgeons, Riyadh Central Hospital for allowing us to study their patients. We are also thankful to Mrs. Cora Rivera for her secretarial help in the preparation of this manuscript.

REFERENCES

1. Elliot JW. The operative relief of gangrene of intestine due to occlusion of the mesenteric vessels. *Ann Surg* 1895; 21:9-23.
2. Kairaloum MI, Karkola P, Heikkinen E et al. Mesenteric infarction. *Am J Surg* 1977; 133:188-193.
3. Levy PJ, Krausz MM, Manny J. The role of second-look procedure in improving survival time for patients with mesenteric venous thrombosis. *Surg Gynecol Obstet* 1990; 170: 287-291.
4. Kitchens CS. Evolution of our understanding of the pathophysiology of primary mesenteric venous thrombosis. *Am J Surg* 1992; 163: 346-348.
5. Abdu R, Zakhour BJ, Dallis DJ. Mesenteric venous thrombosis from 1911 to 1984. *Surgery* 1987; 101:383-388.
6. Boley SJ, Kaleya RN, Brandt LJ. Mesenteric venous thrombosis. *Surg Clin North Am* 1992; 72:183-201.
7. Carr N, Jamison MH. Superior mesenteric venous thrombosis. *Br J Surg* 1981; 68:343-344.
8. Grieshop RJ, Dalsing MC, Ckirit DF, Lalka SG, Sawchuk AP. Acute mesenteric venous thrombosis: Revisited in a time of diagnostic clarity. *Am J Surg* 1991; 57:573-578.
9. Shaff MI, Powers TA, Gilpin LB, Faulstich ME. Abdominal case of the day. *Am J Radiol* 1992; 158:1363-1367.
10. Rahmouni A, Mathieu D, Golli M et al. Value of CT and sonography in the conservative management of acute spleno-portal and superior mesenteric venous thrombosis. *Gastrointest Radiol* 1992; 17:135-140.
11. Kispert JF Jr, Kazmers A. Acute intestinal ischaemia caused by mesenteric venous thrombosis. *Semin Vasc Surg* 1990; 3:157-171.
12. Naitove A, Weismann RE. Primary mesenteric venous thrombosis. *Ann Surg* 1965; 161:516-523.
13. Scully RE, Mark EJ, McNeely WF, McNeely BU. Weekly clinicopathological exercises (Case records of the Massachusetts General Hospital). *N Engl J Med* 1991; 324:613-623.
14. Font VE, Hermann RE, Longworth DL. Chronic mesenteric venous thrombosis: Difficult diagnosis and therapy. *Clev Clin J Med* 1989; 56:823-828.
15. Bemelman WA, Butzelaar RMJM, Khargi K, Keeman JN. Mesenteric venous thrombosis caused by deficiency of physiologic anticoagulants: Report of a case. *Neth J Surg* 1990; 42:161-169.
16. Wilson C, Walker ID, Davidson JF, Imrie CW. Mesenteric venous thrombosis and antithrombin III deficiency. *J Clin Pathol* 1987; 40:906-908.
17. Tollefson DFJ, Friedman KD, Marlar RA, Bandyk DF, Towne JB. Protein C deficiency; a cause of unusual or unexplained thrombosis. *Arch Surg* 1988; 123:881-884.
18. Sack J, Aldrete JS. Primary mesenteric venous thrombosis. *Surg Gynecol Obstet* 1982; 154:205-208.
19. Matthews JE, White RR. Primary mesenteric venous occlusive disease. *Am J Surg* 1971; 122:579-583.
20. Clavien PA, Durig M, Harder F. Venous mesenteric infarction: a particular entity. *Br J Surg* 1988; 75:252-255.
21. Brokmans AW, van Rooyen W, Westerveld BD. et al. Mesenteric vein thrombosis as presenting manifestation of hereditary protein S deficiency. *Gastroenterology* 1987; 92:240-242.
22. Rhee RY, Gloviczki P, Mendonca CT et al. Mesenteric venous thrombosis: still a lethal disease in the 1990's. *J Vasc Surg* 1994; 20:688-697.
23. Vogelzang RL, Gore RM, Anschuetz SLet al. Thrombosis of the splanchnic veins: CT diagnosis. *Am J Roentgenol* 1988; 150:93-96.
24. Harward TRS, Green D, Bergan JJ et al. Mesenteric venous thrombosis. *J Vas Surg* 1989; 9:328-333.
25. Miller VE, Berland LL. Pulsed Doppler duplex sonography and CT or portal vein thrombosis. *Am J Roentgenol* 1985; 145:73.
26. Lanthier P, Lepot M, Mahiue P. Mesenteric venous thrombosis presenting as a neurological problem. *Act Clin Belg* 1984; 29:92-95.