# Case Report of Acute Splenic and Superior Mesenteric Vein Thrombosis and its Successful Medical Management

E Foo, \*FRCS (Glas), R Sim, \*FRCS (Glas), FRCS (Edin), M Med (Surg), B K Ng, \*\*FRCS

### Abstract

We report a case of a 27-year-old pregnant patient who presented with severe colicky abdominal pain, diarrhoea and fever. She was initially treated for gastroenteritis. She later requested a termination of the pregnancy. Abdominal X-rays showed small bowel dilatation. A dynamic computed tomographic scan was performed and showed a splenic and superior mesenteric vein thrombosis. This was confirmed by colour duplex scanning and angiography. Anticoagulation with heparin was associated with dramatic relief of the symptoms and complete recanalisation of both veins. Surgical intervention was avoided.

Ann Acad Med Singapore 1996; 25:755-8

Key words: Anticoagulation, CT scan, Duplex scan, Pregnancy

# Introduction

Mesenteric vein thrombosis (MVT) is a less frequent but definite cause of intestinal ischaemia. Although Elliot' recognised intestinal gangrene secondary to mesenteric venous occlusion almost 100 years ago, it was only in 1935 when Warren and Eberhard<sup>2</sup> reported 2 personal cases and 73 others collected from the literature that it became a recognised clinical entity.<sup>3</sup>

In the past, patients often presented acutely and required operation when the diagnosis was made. In recent years, with the advent of newer diagnostic modalities, cases with less acute presentations have been diagnosed and earlier non-operative treatment instituted. However, cases presenting acutely and successfully treated non-operatively with heparin alone or with thrombolytic agents are still rare.<sup>4,5</sup>

## **Case Report**

A 27-year-old pregnant patient complained of crampy abdominal pain, watery diarrhoea and fever of one day's duration. She was six weeks amenorrhoeic and had 5 other children. She had no medical or family history of note. Her surgical history included an appendicectomy and haemorrhoidectomy 8 years prior to this admission.

On physical examination, she was found to have a temperature of 38.5°C. Her abdomen was soft and tender to deep palpation in the epigastrium. There was no rebound tenderness and bowel sounds were active.

Rectal examination revealed brown stools which were guaiac negative.

Laboratory tests showed a white cell count of 13 700  $/mm^3$ , haematocrit of 37.8% and platelet count of 278  $000/mm^3$ . Urea and electrolytes were within normal range as were the liver function tests and serum amylase level. An ultrasound was the only radiological test obtained at admission which showed some sludge in the gallbladder. The pancreas and bile ducts were normal.

She was diagnosed and treated as having gastroenteritis. However, her pain increased in frequency and severity, being especially worse after meals. In addition, her stool was occasionally stained with blood. As she had completed her family, she requested termination of her pregnancy which was performed at the end of her first week of hospitalisation.

After termination of the pregnancy, an abdominal roentgenogram showed dilated loops of the small bowel. An upper gastrointestinal series was done and was essentially normal. A computerised tomographic (CT) scan, (Fig. 1) however, showed thromboses of the splenic and superior mesenteric veins. This was confirmed by a colour duplex scanning (Fig. 2). A superior mesenteric angiogram was performed which showed normal arterial patterns. The venous phase showed thromboses of the splenic and superior mesenteric veins with extension into the portal vein. There was collateral venous drainage via the gastric, gastroepiploic and inferior mesenteric veins.

Alexandra Hospital

Address for Reprints: Dr Edward Foo, Department of Surgery, Alexandra Hospital, 378 Alexandra Road, Singapore 159944.

<sup>\*</sup> Registrar

<sup>\*\*</sup> Clinical Associate Professor and Head Department of Surgery



Fig. 1. A computed tomographic scan showing thrombus within the splenic vein (arrow).



Fig.3. A repeat computed tomographic scan performed 2 weeks after therapy showing recanalisation of the splenic vein (arrow)

The patient's abdomen remained soft although mildly tender. Intravenous heparin was started at 1000 U/h to obtain clotting times twice the normal values. Over the next two days, her pain subsided dramatically and she was essentially pain-free by the third day. Coumadin was started after the patient had been on heparin for a week.

A repeat CT scan, (Fig. 3) obtained about 2 weeks after anticoagulation was started, showed complete recanalisation of her splenic and superior mesenteric veins. She was discharged on coumadin at 3 mg/day which maintained her clotting time at twice the normal value.

Her haematological work-up showed normal levels of antithrombin ITT, protein C and protein S. The antinuclear antigen test was negative. Anticardiolysin tests for immunoglobulin G and immunoglobulin M were negative. Although serum protein electrophoresis showed elevated immunoglobulin G, these were non-monoclonal and therefore not indicative of any myeloproliferative disease



Fig.2. Colour duplex scan (reproduced in black and white) showing thromboses in the superior mesenteric vein and splenic vein (SPL V). SMA: superior mesenteric artery; IVC: inferior vena cava.

#### Discussion

The incidence of MVT, once thought to be as high as  $41\%^6$  of all cases of mesenteric ischaemia, has been reported to be as low as  $5\%^7$  in more recent literature. This could be attributed to the more widespread use of angiography leading to more accurate diagnoses.

Amongst patients with MVT, predisposing conditions have been found in more than 80%.<sup>8</sup> Fewer cases of primary<sup>9,10</sup> or ""idiopathic" MVT have been reported now that newer conditions such as antithrombin III,<sup>11</sup> protein C<sup>12</sup> and S<sup>13</sup> deficiencies are known. The known predisposing conditions include peripheral deep vein thrombosis,<sup>14,15</sup> neoplasms,<sup>10</sup> antithrombin III deficiency,<sup>11</sup> protein C<sup>12</sup> and S<sup>13</sup> deficiency, oral contraceptive use,<sup>16</sup> thrombocythaemia,<sup>4</sup> polycythaemia vera,<sup>17</sup> cirrhosis,<sup>4\*</sup> following sclerotherapy,<sup>19</sup> pancreatitis,<sup>20</sup> sepsis,<sup>18</sup> trauma,<sup>10</sup> and even decompression sickness.<sup>21</sup> As there was no clinical or biochemical evidence of the above conditions in our patient, our case, we believe, is the first reported where the only association is pregnancy.

The most common presenting symptom is pain, which as in the case of our patient, is typically out of proportion to the physical findings.<sup>7,10,22</sup> Interestingly, this patient had a good result from anticoagulation although this treatment was started 2 weeks after the onset of pain. This was attributed to the development of good collaterals which prevented early intestinal infarction. Experimental<sup>23</sup> and clinical<sup>24</sup> evidence demonstrates that more gradual occlusion of the mesenteric vein did not lead to immediate intestinal infarction. However, infarction may result from propagation of the clot into smaller branches of the veins<sup>18</sup> unless this is prevented by anticoagulation. Moreover, even if the ischaemic injury is reversible, a patient may develop chronic MVT leading to portal hypertension, bleeding varices and hypersplenism.<sup>2,20</sup> **Other symptoms and signs** including nausea, diarrhoea, abdominal tenderness, fever and leukocytosis are also described although they are less frequent features of the disease.<sup>21</sup>

Diagnosis of MVT clinically is difficult. Until recently, 50% of cases were diagnosed at laparotomy. Radiological studies have become the best means to establish a definitive diagnosis.<sup>25</sup> Plain radiographs of the abdomen in our patient had shown some dilated loop5 of the bowel but this was not indicative of infarction. Small bowel studies<sup>26</sup> have been reported to be useful by some authors but this was not helpful in our patient. Positive findings include thickening of the bowel wall, and "thumbprinting" of the mucosa not unlike that found in Crohn's disease.

Ultrasonography,<sup>4,27,28</sup> especially colour duplex scan ning, has been reported to be useful in some cases. In our patient, before termination of her pregnancy, it was the only means available. However, it is highly operator dependent and should be accompanied by a high index of suspicion. Therefore, while an initial ultrasound failed to detect the lesion, a subsequent ultrasound easily detected the thrombus in the superior mesenteric and splenic vein.

CT scan<sup>4,25,28,29</sup> of the abdomen is possibly the best modality for diagnosing MVT, its sensitivity being as high as 90% in some series. Some authors have had success with magnetic resonance imaging<sup>5</sup> but this modality is not widely available.

Selective mesenteric angiography<sup>14,30</sup> can also establish a definitive diagnosis. Although it is invasive, it has the advantage of differentiating MVT from arterial forms of ischaemia and allowing the infusion of intra-arterial dilators, eg. papaverine.<sup>31</sup> This treatment has been proposed based on experimental evidence furnished by Polk<sup>32,33</sup> and Laufman<sup>23</sup> that MVT is associated with arterial spasm. Clinical experience with infusion of intra-arterial dilators however, has been limited. This was not attempted in our patient.

Anticoagulation remains the cornerstone of therapy once the diagnosis of MVT is established. This includes patients who require bowel resections with<sup>34</sup> or without surgical thrombectomies. This treatment was initially proposed based on experimental evidence supplied by Nelson and Kremen<sup>35</sup> that heparinisation before mesenteric venous occlusion prevented deaths in 6 out of 8 dogs by preventing the propagation of the thrombotic process. All unheparinised dogs died. However, it was only recently that a clear benefit has been demonstrated from clinical studies. In one study, heparinised patients had a recurrence rate of only 13% compared to 25% for patients who did not receive anticoagulation after surgery.<sup>8</sup> Patients diagnosed to have a congenital hypercoagulable state should probably receive longterm, perhaps even life-long anticoagulation.<sup>10,14,25,36</sup> The **plan** in **our patient was to continue** anticoagulation **for only** 6 **months as the only detectable risk factor was her pregnancy.** 

#### REFERENCES

- Eliiot J W. The operative relief of gangrene of the intestine due to occlusion of the mesenteric vessels. Ann Surg 1895; 21:9-23.
- 2 Warren S, Eberhard I Mesenteric venous thrombosis. Surg Gynecol Obstet 1935; 61:102-20
- 3 Donaldson JK, Stout BF Mesenteric thrombosis Arterial and venous types as separate clinical entitles: a clinical and experimental study. Am J Surg 1935; 29:208-12.
- 4 Verbanck JJ, Rutgeeris W, Haerens MH, Tytgat JH. Segaert MF, Lytgat H J, et al Partial splenoportal and superior mesenteric venous thrombosis. Early sonographic diagnosis and successful conservative management Gastroenterology 1984; 86:949-52
- 5 Al Karawi M 4, Quaiz M, Clark D Hilali A Mohamed A E, Jawdat M, Mesenteric vein thrombosis: Non-invasive diagnosis and follow-up (US + MRI) and non invasive therapy by streptokinase and anticoagulants. Hepatogastroenterology 1990; 37:507-9.
- Trotter CBG. Embolism and Thrombosis of Mesenteric Vessels. Cambridge: Cambridge Univ Press, 1913.
- 7 Kaleya R N, Boley S J Mesenteric venous thrombosis In: Najarian J S, Delaney J P, editors. Progress in Gastrointestinal Surgery. Chicago: Y eai Book Medical Publishers, 1989:417-25.
- Abdu R A Zafkour B 5, Dallis D J Mesenteric venous thrombosis---1911 to 1984. Surgery 1987;101:383-8.
- Anane-Sefah J C, Blair E, Reckler S. Primary mesenteric venous occlusive disease. Surg Gynecol Obstet 1975; 151:740-3.
- Sack I, Aldrete J S. Primary mesenteric venous thrombosis. Surg Gynecol Obstet 1982; 154:205-8.
- 11 Gruenberg J C, Smallridge R C, Rosenberg R D Inherited antithrombin III deficiency causing mesenteric venous infarction: A new clinical entity. Ann Surg 1975; 181:791-4.
- 12 Green D, Ganger DR, Blair A T. Protein C deficiency and splanchnic venous thrombosis. Am J Med 1987; 82:1171-4
- Broeekmans A W van Rooyen W, W estet veld B P Mesenteric vein thrombosis as presenting manifestation of hereditary protein S deficiency. Gastroenterology 1987; 92:240-2.
- 14 Clavien PA, Durig M, Harder F. Venousnesenteric infarction: A particular entity Bi J Surg 1988; 75 252-5.
- 15 C ruickshank A H Venous thrombosis in internal organs associated with thrombosis or leg venus. J Pathol Bacteriology 1956; 71:383-90.
- 16 Nesbit J R R, Deweese J A, Mesenteric venous thrombosis and oral contraceptives South Med J 1977; 70:360-2
- 17 Ostermiller J W, Carter R. Mesenteric venous thrombosis secondary to polycythemia vera. Ann Surg 1969; 35:407-9.
- Johnson C C, Baggenstoss A 11 Mesenteric venous occlusion: Study of 99 cases of occlusion of venus. Mayo Clin Proc 1949; 24:628-36.
- 19 Thatcher B S, Sivak M V. Ferguson D R Mesenteric venous thrombosis as a possible complication of endoscopic selerotherapy. A report of two cases. Am J Gastroenterol 1986, 81:126-9.
- Warshaw A L, Gongliang J, Ottinger L W. Recognition a n d clinical implications of mesenteric and portal vein obstruction in chronic pancreatitis. Arch Surg 1987; 122:410-5.
- 21 Boley S, Kaleya R, Brandt L. Mesenteric venous thrombosis Surg Clm North Am 1992; 72:183-200.
- 22 Matthews P, White R R Primary mesenteric venous occlusive disease Am JSurg 1571; 122:579-83.
- 23 I aufman H. Gradual occlusion of the mesenteric vessels: Experimental

study. Surgery 1941; 13:406-14.

- 24. Laufman H, Scheinberg S. Arterial and venous mesenteric occlusion: Analysis of 44 cases. Am J Surg 1942; 58:84-92.
- Harward T R S, Green D, Bergan J J, Rizzo R J, Yao J S T. Mesenteric venous thrombosis. J Vasc Surg 1989; 9:328-33.
- Clemett A R, Chang J. The radiologic diagnosis of spontaneous mesenteric venous thrombosis. Am J Gastroenteroll975; 63:209-15.
- Kidambi H, Herbert R, Kidami A V. Ultrasound demonstration of superior mesenteric and splenoportal venous thrombosis. J Clin Ultrasound 1981; 14:199-203.
- Matos C, van Gansbeke D, Zalcman N I. Mesenteric venous thrombosis: Early CT and ultrasound diagnosis and conservative management. Gastrointest Radio1 1986; 11:322-5.
- Vogelzang R L. Thrombosis of the splanchnic veins: CT diagnosis. Am J Radio1 1988; 150:93-6.
- 30. Clark A Z, Gallant T E. Acute mesenteric ischemia: angiographic spectrum.

Am J Radio1 1984; 142:555-62.

- Lanthier I', Lepot M, Mahieu P. Mesenteric venous thrombosis presenting as a neurological problem. Acta Clin Belg 1984, 29:92-5.
- 32. Polk H C. Studies in experimental mesenteric venous occlusion I: The experimental system and its parameters. Surgery 1964; 108:693-8.
- Polk H C. Experimental mesenteric venous occlusion III: Diagnosis and treatment of induced mesenteric venous thrombosis. Ann Surg 1966; 163:432-8.
- Bergentz S, Ericsson B, Hedner U, Leandoer L, Nilsson I M. Thrombosis in the superior mesenteric and portal veins: Report of a case treated with thrombectomy. Surgery 1974; 76-286-90.
- Nelson L, Kremen A. Experimental occlusion of the superior mesenteric vessels with special reference to the role of intravascular thrombosis and its prevention by heparin. Surgery 1950; 28:819-29.
- Umpleby H C. Thrombosis of the superior mesenteric vein. Br J Surg 1987; 74:694-6.