PAIN ABDOMEN AT EXTREME ALTITUDES : SINISTER IMPLICATIONS

Lt Col AC ANAND ^{VSM*}, Lt Col A SAHA ⁺,
Lt Col V SHARMA [#], Lt Col HS NANDA ^{**},
Col CM ADYA ^{VSM⁺⁺}, Maj Gen VIRENDRA SINGH ^{##}

ABSTRACT

2 cases of portal, splenic and superior mesenteric vein thromboses related to prolonged stay at high altitude are presented. Both presented initially with innocuous appearing vague pain in abdomen with no physical signs. Later hepatosplenomegaly, and pleural effusion (left) was also detected. Diagnosis was based on CT scan and colour Doppler study showing thrombosed veins and porto-systemic collateral. 1 patient developed a large splenic haematoma requiring splenectomy. Both were managed with early anticoagulation and have done well in the short follow up.

MJAFI 2001; 57 : 158-160

KEY WORDS :High altitude; Mesenteric vein thrombosis; Portal vein thrombosis; Splenic vein thrombosis.

Introduction

Superior mesenteric vein thrombosis, a rare form of acute mesenteric ischaemia, is an infrequent disease. Previously, the diagnosis was made only at laparotomy or autopsy, but now imaging techniques allow it to be recognised during life [1]. Aetiology of this condition is usually a hypercoagulable state [2,3]. So far this complication has never been described due to stay at high or extreme altitude. We describe here two young officers who presented with innocuous looking pain abdomen after a few months stay at extreme altitude.

Case Report -1

YS, a 35 year old male officer was inducted into extreme altitude area in May 99 and stayed for variable periods at altitudes of 14000 to 22000 ft. He was stationed at 20000 ft for a period of over a month when symptoms started. On 01 Jan 2000, he developed pain in epigastrium. Pain was of acute onset, moderately severe, felt mainly in upper abdomen. Over next seven days the pain spread to left hypochondrium and in another couple of days to whole abdomen. Eating, and change of posture aggravated pain, but there was no effect on defecation. Along with pain he also developed loss of appetite and even vomited once on 3rd January. On 7th January, he also noticed black coloured motions, which continued over next two weeks. He also felt feverish but temperature could not be recorded till 17th, when it was found to be 38°C. There was no history of haematemesis, icterus, syncope, trauma, or drug/toxin exposure. There was a past history of brief episode of pain epigastrium in June 99 during Operation Vijay, when he was given some antacids with good relief in a few days. He is a non-smoker, and teetotaler by habit. He could not be evacuated from central glacier (> 19000 ft) and started trekking back on 7th January, reaching base camp on 12 Jan (18000 ft), from here he was heli-lifted to a medical post on 16 Jan at 12000

feet and later to a field ambulance on 17 Jan at 10000 ft. He was moved to a tertiary care centre by aircraft on 19 Jan 2000. Initial diagnosis at the field ambulance was acute pancreatitis and was managed with intravenous fluids, and ranitidine. Investigations at this stage showed Hb 15.5 gm%, TLC 26000/cmm with P84%, and Serum amylase 168 i.u./l. He reached this hospital on 19 Jan 2000, and examination here showed height 176 cm, wt-60 kg. Pulse rate - 110/min regular, BP-160/96 mm of Hg, respiratory rate-24/min regular, and temperature-37°C. There was no edema, pallor, cyanosis, icterus or lymphadenopathy. JVP was not raised. Examination of abdomen showed that there was diffuse distension and tenderness. There was an impression of a vague lump in upper abdomen more to left than right side with local guarding. Liver and spleen were not palpable. There was dullness in the flanks and test for shifting dullness was positive. Examination of other systems was within normal limits. Investigations showed Hb 14 gm%, TLC 12,300/cmm, DLC P87%, L-6%, M-1%, E-6%, platelet count 188000/cmm, Serum bilirubin 2.20 mg%, SGOT/SGPT 49/43 i.u./l, USG abdomen showed presence of ascites, a large splenic SOL (?haematoma), and splenic vein thrombosis. SMV and PV were not visualised. Pancreas appeared normal. Urgent UGIE was within normal limits and did not show any cause of bleeding. Peritoneal fluid appearance was haemorrhagic, specific gravity was 1.018, RBCs were 28000/cmm, WBC was 11,600/cmm, predominant cell was lymphocytes, serum proteins were 3.80g%, peripheral blood smear showed leucocytosis and presence of toxic granules in WBC. CECT abdomen showed portal vein and splenic vein thrombosis, superior mesenteric vein thrombosis (Fig-1), splenomegaly with large splenic haematoma thickened small bowel wall, presence of ascites, bilateral pleural effusion. Pancreas was normal. Subsequently, a power Doppler study confirmed these findings and showed multiple collateral at the porta-hepatais. No evidence of thrombosis was seen in the leg veins. Further investigations ruled out deficiency of antithrombin-III, protein C or S and presence of Antiphospholipid syndrome. He was kept on a regimen consisting of nil by mouth and partial parenteral nutrition (with a mixture of amino acids and fat emulsion). Intravenous antibiotics were given (cefotaxime, amikacin

*Gastroenterologist, *Gastrointestinal Surgeon, #Radiologist, **Anaesthesiologist, ++ Senior Adviser (Medicine), ##Commandant. Command Hospital (Western Command), Chandimandir -134 107.

Fig. 1: A CT-scan frame showing thrombosis of the superior mesenteric vein (arrow) in case number 1

and metronidazole) and he was cautiously heparinised keeping a close watch on gastro intestinal bleeding. He improved gradually and oral feeds were started on 30 Jan. Haematoma in spleen showed no reduction in size and his pain over that area persisted. On 02 Feb, he was electively taken up for splenectomy. At surgery extensive omental adhesions were noted in left upper abdomen. Within hours of achieving haemostasis, heparin was started to avoid recurrence of thrombosis. Patient stabilized over next few days and was placed on oral anticoagulants and was discharged on 14 Feb.2000.

Case Report - 2

SK, a 33 year old male medical officer, staying at extreme altitude for over 90 days, developed pain abdomen on 17.03.2000. Pain was poorly localised, dull boring/aching in character and constantly present in umbilical, epigastric and right hypochondrium area. Pain was aggravated on eating food and on walking and even lying down. Examination showed pulse 100/mt, BP 110/90. Abdomen was soft, but there was tenderness all over. 2 cm soft and non-tender hepatomegaly was noted and clinically a possibility of Amoebic Hepatitis was considered. There was no relief with medications such as antacids, Tinidazole, Norfloxacin, Baralgin, Spasmoproxylon and later Pethidine; hence he was evacuated to Command Hospital, Chandimandir. Investigations showed Hb 17.5 g/dl, TLC 8100.Cmm, P63%, L3%, M2%, E4%. Serum bilirubin 0.6 mg/dl, urea 25 mg/dl, sugar 90 mg/dl, stool for occult blood was positive. On 28/03/00, serum bilirubin was 2.8 mg/dl, albumin/globulin 4.1/3.1 g/l, SGOT/SGPT:46/71 U/l, serum alkaline phosphate 216 U/l, prothrombin time (control/test) 12/15 seconds, and serum amylase 138 SU. At Command Hospital, ultrasound abdomen showed liver 15.5 cm, spleen 13.4 cm, portal vein was 16-17 mm at formation, both portal and splenic vein contained echogenic material. Ascites was present. Small amount of pleural fluid was seen on left side. Colour Doppler showed findings suggestive of splenic, superior mesenteric and portal venous thrombosis with evidence of portal venous hypertension. Liver parenchyma was normally preserved showing normal hepatic venous and hepatic artery flows. CT scan also showed similar finding. UGI endoscopy showed grade 1 oesophageal varices, fundal varices and erosive gastritis. Antiphospholipid antibody and anti-thrombin-III showed no abnormality. Tests for protein C, S and antithrombin-III were normal. He was managed with antibiotics and anticoagulation initially with heparin and later with Sintrom. Propanolol was added to reduce portal pressure. Gradually he stabilized, became asymptomatic and was dis-

Fig. 2: A CT scan frame showing that almost two-thirds of the spleen is replaced by a hematoma in case number 1 (low attenuation area marked by 'o'). There is also a sub capsular collection anteriorly.

charged.

Discussion

This is possibly the first report of thrombosis of superior mesenteric artery, portal vein and splenic vein occurring in relation to high altitude. The diagnosis was not suspected till the patients were evacuated to a tertiary care hospital. This is not surprising as such a possibility has never been discussed in any forum. Although pulmonary vein thrombosis [4] and cerebral vein thrombosis [3] have been described, thrombosis of abdominal veins in relation to high altitude is virtually unheard of in literature. The diagnosis in these cases was also delayed, because initial symptom of pain was poorly localised and there were no physical signs, which are a common features of this disease. In fact mean duration of pain before hospital admission is described as 5-14 days [5] and in one report 27% patients had pain for over a month [6]. By the time, these patients were evacuated to this hospital (approximately 2-3 weeks from the onset of pain), they had already started developing varices/alternative channels at the porta-hepatis. In portal vein thrombosis due to other etiologies, the cavernomatous varices at porta have been noted to develop after 5 to 12 weeks [7]. These patients had none of the usual precipitating factors associated with venous thrombosis such as trauma, prolonged immobilisation or dehydration. Moreover, no thrombosis was noted in the veins of the legs. Presence of splenic haematoma is a unique finding for which we have no clear explanation. Acute splenic vein thrombosis or trivial trauma not remembered by the patient may be a cause. Disorders of coagulation during ascent to high altitude in the unacclimatized are well recognised. Initial prporthrombotic phase, compounded by increased capillary fragility

and increase in bleeding time have been described [8]. Another study describes increase in D-Dimer on exposure to chronic hypoxia (during a stay at 6542 m), which seems to correspond to an activation of coagulation. The study also found a decrease of F.VIII R. Cof/F. VIII R. Ag ratio, which suggests an endothelial cell damage [9]. Recognition of thrombosis as a complication of high altitude stay has also stimulated interest in rheological changes resulting from hypobaric hypoxia. A recent study noted significant and sustained elevation in platelet numbers within 48 h of ascent to high altitude. It was not related to any increase in thrombopoietin levels though an etiological role of erythropoietin was not ruled out [10]. Existence of additional genetic procoagulant factors remains an important consideration [11]. Though none was found in this case as yet.

References

1. Brandt LJ, Smithline AE. Ischaemic lesions of the bowel. In Feldman M, Sliesenger MH, Scharschmidt BF, Klein S editors. *Gastrointestinal and Liver Disease*, 6th ed. 1998;2:2009-24.
2. Abdu RA, Zakour BJ, Dallis DJ. Mesenteric venous thrombosis : 1911-84. *Surgery* 1987;101:383-8.
3. Harward RTS, Green D, Bergan JJ et al. Mesenteric venous thrombosis. *J Vasc Surg* 1989;9:328-32.
4. Cucinell SA, Pitts CM. Thrombosis at mountain altitudes. *Aviat Space Environ Med* 1987 Nov;58(11):1109-11.
5. Sack J, Alderete JS. Primary mesenteric venous thrombosis. *Surg Gynecol Obstet* 1982;154:205-11.
6. Mathews J, White RR. Primary mesenteric venous occlusive disease. *Am J Surg* 1971;122:579-84.
7. Schafer DF, Sorrell MF. Vascular Diseases of the liver. In Feldman M, Sliesenger MH, Scharschmidt BF, Klein S editors. *Gastrointestinal and Liver Disease*, 6th ed. 1998;2:1188-98.
8. Doughty HA, Beardmore C. Bleeding time at altitude. *J R Soc Med* 1994;87(6):317-9.
9. Le Roux G, Larmignat P, Marchal M, Richalet JP. Haemostasis at high altitude. *Int J Sports Med* 1992 Oct;13Suppl 1:S49-51.
10. Hudson JG, Bowen AL, Navia P, Rios-Dalenz J, Pollard AJ, Williams D, Heath D. The effect of high altitude on platelet counts, thrombopoietin and erythropoietin levels in young Bolivian airmen visiting the Andes. *Int J Biometeorol* 1999;43(2):85-90.
11. Boulos P, Kouroukis C, Blake G. Superior sagittal sinus thrombosis occurring at high altitude associated with protein C deficiency. *Acta Haematol* 1999;102(2):104-6.