A Young Boy with Abdominal Pain

Pages with reference to book, From 284 To 286

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Introduction

At times, acute diffuse abdominal pain can be a diagnostic dilemma, especially when the symptoms appear to be out of proportion to the findings on physical examination. The case of a young boy with abdominal pain is presented.

Case Report

A 16 yearold student presented to the Emergency Room with a history of pain in the abdomen for the last one month. The pain was generalized and colicky in character. It gradually increased in severity, was episodic and aggravated severely over the last one week. About three years ago in 1990, he developed swelling of the left leg and was referred to a vascular surgeon. He was diagnosed to have deep vein thrombosis and treated with Tablet Aspirin for three, months. There was a history of seizure, forwhicha CT scan of the brainwas done and reported to be normal. On examination, his pulse was 100/minute and regular. His blood pressure was 140/90 mmHg. Chest examination revealed normal vesicular breathing. On abdominal examination, he had tenderness all over the abdomen but there was 110 rebound tenderness and no rigidity was noted. His liver, spleen and kidneys were not palpable and gut sounds were audible. Cardiovascular examination was essentially normal. Neurological examination was also normal.

Laboratory Investigations

Hemoglobin was 13.5 gm/dl, WBC 9.8xl0^{9}fL. Platelet count 227x10^{9}IL. His ESR was 16, RBS 126 rng/dl, BUN 14 mg/dl, serum creatinine 1 mg/dl, serum sodium 139 mm/I, serumpotassium4.2, serumchloride 108 and serumbicarbonate 18.5 mm/I. Total bilirubin was 0.3 mg/dl and alkaline phosphatase was 111 IU/L. Chest X-ray was unremarkable. Plain X-ray of the abdomen showed a small white density along the right ureter. Urine analysis was normal. Prothrombin time was 14.3 seconds with a control of 12 seconds and APTT was 33 seconds with a control of 30 seconds. Urinary porphyrins were negative. Ultrasound of the abdomen showed splenomegaly with thickened wall of the small intestinal loops. There was no evidence of ascites. The Brucella antibody litres were negative. Small bowel enema was performed which showed irregular thickened mucosal folds in the jejunum with fixation of distal ileal loops and thickened ileo-caecal valve. These findings were suggestive of gross inflammatory changes, most likely tuberculosis of the intestine. The patient was discharged over the weekend to be re-admitted for a colonoscopy and biopsy for confirmation of the histological and microbiological diagnosis before starting any treatment. After about 48 hours the patient reported again to the Emergency Room with severe abdominal pain over the last 12 hours. At the present admission the patient developed vomiting and bloody diarrhoea. On examination, his pulse was 92/minute and a blood pressure 120/90 mrnHg. He had tenderness involving whole abdomen but there was no rigidity. His bowel sounds were audible. On rectal examination the fingerstall was blood stained, and sigmoidoscopy showed the mucosa coated with blood streaks. In view of the clinical and laboratory findings, the decision to conservatively manage the patient with IV antibiotics and metronidazole was made. On investigation, his hemoglobin was 14.6 gm/dl with a WBC count now of 37.3x10 /L with 88% neutrophils. Microscopic examination of stool showed blood with RBCs>20/HPF and leucocytes

12/HPF. No ova, cysts or parasites ‘were identified. Entamoeba IHA litre was <1:16. He underwent an upper GI endoscopy which showed evidence of reflu.x esophagitis with no evidence of a bleeding site. A colonoscopy was also performed which showed only altered blood. Repeat plain abdominal film showed generalized small bowel distension with multiple air fluid levels and edema of part of the bowel wall; there was no gas under the diaphragm. He was reviewed by a surgeon who advised passage of a double lumen nasogastric tube as well as suctioning. The patient’s condition deteriorated, with rebound tenderness and rigidity of the abdomen, a drop in hemoglobin of about 1 g/dl and absence of gut sounds. The antibiotics metronidazole and ofloxacin were continued and the patient was transferred to the surgical unit for observation regarding the need for exploratory laparotomy.As the patient’s condition continued to deteriorate, an exploratory laparatomy was performed which showed ischernic bowel about 25 cm in length, 50 cm proximal to the ileo-caecal valve. About 40 cm of small bowel was resected. Thrombosis was found in the mesenteric veins; however, arteries were found to be patent. An ileostomy and a mucous fistula was made. The histopathology of the lesion revealed transmural infarction with large areas of hemorrhage. The margins of resected bowel were unremarkable. Mesenteric blood vessels were extensively dilated and congested. There was no evidence of granulomata or malignancy. The patient was followed up at regular intervals in the surgical outpatient clime for the care of ileostomy and colostomy sites and made a good recovery.
His serum anti-phospholipid antibody titre was elevated; IgG 10.3 GPL/ml (0-10.0) and IgM. 44.0 MPL/ml (0-20.0). Thus this patient most probably developed mesenteric venous thrombosis due to primary Anti-Phospholipid Antibody Syndrome (APAS). The past history of venous thrombosis in our patient may be anindicatorofa hypercoagutable state, although there was no family history.

Discussion

Ischemic bowel disease often presents in a ratherbenign manner, yet has significant morbidity and mortality if not diagnosed and managed promptly. Mesenteric venous thrombosis, presenting as ischcrnic bowel disease has the same poor prognosis if not aggressively diagnosed and managed. The cause of mesenteric venous thrombosis (MVT) can be categorized into trauma; mechanical, infection and hematological disorders[1]. MVT has occurred in women taking oral contraceptives[2]. Blunt abdominal trauma may result in MVT. Mechanical problems include volvulus, intussusception and post- operative abdominal sepsis. Associated conditions that authors have implicated as contributory factors include cirrhosis, previous abdominal sur;ery, congestive heart failure and congestive splenomegaly[3,4]. In the absence of the above mentioned identifiable factors, hypercoagulable states, either intrinsic or induced, account for most cases of MVT. This is particularly so in patients with recurrent episodes, as in our patient who had a history of deep vein thrombosis. The causes include polycytheniia, thrombocytosis, anti-thrombin III deficiency, protein C or protein S deficiency and anti-phospholipid antibody syndrome. Antiphospholipid antibodies are autoantibodies that bind a variety of phospholipids. In addition to thrombosis, recurrent abortions and thrombocytopenia, reported clinical features include livedo reticularis and possibly other skin lesions, heartvalve abnormalities, and a variety of neurological disorders. Mesentenc venous thrombosis is more common in the sixth and seventh[5] decades. The superior mesenteric vein is most commonly involved[6]. The large bowel is rarely involved. The hallmark of MVT is generalised severe crampy abdominal pain that is out of proportion to the physical findings. Most patients have a history of intermittent abdominal pain before presentation. A history of previous occurrence of spontaneous venous thrombosis should raise suspicion. Whether abdominal distension, vomiting, hematemesis, me lena or hematochezia is found, depends on the stage of the disease. Patients seem to have no problem with eating or with bowel movements in the early stage. Typically there is abdominal pain and on examination no rebound or guarding initially. This lack of distinguishing findings contributes to a delay in diagnosis. Stools were

found positive for blood in 80-90% in three separate studies[6]. Fever is usually absent or low grade. Laboratory analysis reveals hemoconcentration and leucocytosis with a left shift. Serum amylase and phosphorus levels are elevated only when ischemia and necrosis are advanced. Metabolic acidosis is common with elevation of lactic acid and a concomitant increased anion gap. Routine abdominal radiographs are of little diagnostic help; non-specific ileus, ascites, bowel wall thickening, niucosal irregularity and thumb printing have all been observed. Ultrasonography has been used for early diagnosis in non-obese patients[7]. If available, mesenteric artenography should be done early in the diagnostic evaluation. Patency of arteries and opacification of bowel wall with failure to visualise the mesenteric veins are diagnostic of MVT. Contrast enhanced computed tomography (CT) showing a dense venous wall surrounding a central lucency was diagnostic in a report involving six cases of MVT[8]. The use of Xenon 133 in nonnal saline injected into the peritoneal cavity has proved to be a promising method for early diagnosis[9]. Time is important in reducing mortality in MVT; therefore clinical and surgical evaluation should take priority over radiologic procedures. Because serosanguinous fluid is usually present in the peritoneal cavity, abdominal paracentesis has been found to be uniformly diagnostic[3]. Some authors have concluded that peritoneoscopy is the best and quickest diagnostic method[3]. Management includes laparatomy with identification of infarcted bowel and mesentery. A thrombectomy is mandatory, along with resection of all necrotic small bowel and mesentery. Intraoperative treatment with heparin should be started and continued for 7 to 10 days. Warfarin therapy should be started postoperatively and continued for at least 3 months an4 possibly indefinitely, depending on the underlying cause. Massive volume support and broad spectruLn antibiotics are also needed postoperatively, as sepsis is a common sequel. A “second look” operation performed 24 to 48 hours after the first one is recommended because of a 60% recurrent rate of thrombosis-and ischemia[6]. The mortality rate is 100% without treatment: even with surgery the mortality rate is 30-45%. Studies suggest that if long tenn anticoagulants are not used, 25% of patients will have anotherepisode of MVT. In patients without an obvious cause for MVT, evaluatioii for an intrinsic hypercoagulable disorder is warranted. This is important not only for determining the duration of oral anti- coagulant treatment, but because these deficiencies are inherited in anautosomal dominant pattern. The risk of venous thrombosis (including pulmonary) from these disorders increases with age.

Conclusions

Mesenteric venous thrombosis is relatively rare. A high index of suspicion is required for diagnosis when a person complains of generalized severe abdominal pain that is out of proprotion to physical findings.

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