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Case Report

Rupture Spleen as Initial Presentation in A Patient of Chronic Myeloid Leukaemia: A Case Report

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Abstract

A 22 years female student presented with sudden severe abdominal pain in left upper quadrant followed by abdominal distension. She attended in surgery unit of a tertiary health care center. On examination she was in shock and the abdomen was distended, tender. She was evaluated as surgical acute abdomen and emergency laparotomy revealed rupture of spleen and splenectomy was done. Her CBC showed gross leucocytosis with predominance of neutrophils and significant number of myelocytes and the PBF showed the features of chronic phase of chronic myeloid leukaemia. BCR-ABL was positive from bone marrow that confirms the diagnosis. Splenomegaly is a common presentation of CML. However spontaneous rupture of spleen is a very rare presentation of CML. [*Journal of Science Foundation, 2015;13(2):52-54*]

Keywords: Rupture spleen; chronic myeloid leukaemia; abdominal pain

Introduction

Spontaneous splenic rupture is a rare clinical event with serious consequences, if unrecognized and untreated (Torricelli et al., 2001). It mostly occurs in pathologically enlarged spleens but cases of spontaneous rupture in a histologically proven normal spleen have been reported. Spontaneous splenic rupture has been reported as the presenting symptom in patients with lymphoma and acute myeloid leukaemia. We report the first case of a Philadelphia positive chronic myeloid leukaemia in Dhaka Medical College Hospital, Dhaka, Bangladesh which presented with spontaneous splenic rupture as the initial manifestation of the disease.

Case Presentation

A 22-year female student presented with sudden onset of severe left upper abdominal pain for 4 days. It was aggravated by coughing, deep breathing, even with posture change. There was no relieving factors and not associated with vomiting. Later pain became generalized with gradual abdominal distension .She also complains of low grade fever. She had no history of trauma. On clinical examination, he was hypotensive with a blood pressure of 80/60 mm Hg and a tachycardia of 130 beats/min. Her respiratory rate was 26. She was severely anaemic and her temperature was

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101^0F. There was no lymphadenopathy, no bony tenderness. Her abdomen was distended, generalized tender and rigid. Abdominal organs could not be assessed clinically due to rigidity and tenderness. Percussion note was dull all over the abdomen and ascites was evidenced by positive shifting dullness. Bowel sound was absent. Digital rectal examination revealed pouch of Doglus was bulged and tender. Her level of consciousness was rapidly deteriorating. Haematological investigations revealed low haemoglobin at 7 g/dl. WBC was 244×109/Land platelet count was 500×109/L. Differential. There were 5% promyelocytes, 13% myelocytes, 77% neutrophil, 3% lymphocytes,1% basophil and 1% monocytes . Prothrombin time was 13 sec and INR 1.02. USG of whole abdomen showed huge dense ascites with left sided mild pleural effusion, hugely enlarged liver (17cm) with uniform parenchymal echotexture, hugely enlarged spleen (20cm). Splenic outline was irregular, a splenic tear is likely. An echo-free area (13cm×10cm) was noted near upper pole suggests a haematoma. Rest shows uniform appearance. Two tiny enlarged lymph nodes are seen in splenic hilum. Plain X-ray abdomen showed distended loops of bowel with multiple air-fluid level. Liver function test, renal function test, electrolytes were normal. Patient was underwent emergency laparotomy with midline incision with left sub-costal extension. Per operative finding were massive haemoperitoneum, huge splenomegaly and hepatomegaly. Posterior pole of spleen was lacerated and covered with huge clot. Splenectomy was done with thorough peritoneal toileting. Spleen was weighted 2150 gm. Her postoperative recovery was uneventful. Histology of resected spleen showed focal collections of immature granulocytes with varying stages of maturation with nucleated red bllod cells and eosinophilic myelocytes. Ischaemic necrosis was also noted. Features were consistent with granulocytic sarcoma. Peripheral blood film was done again with bone marrow aspiration. Both reports were consistent with chronic phase of chronic myeloid leukemia. Bone marrow specimen for BCR-ABL translocation assay was positive which is the confirmatory. She was started with Imatinib mesylate subsequently. It is now 6 months since the diagnosis and she has achieved complete haematological response to treatment.

Discussion

Splenomegaly is a very common presentation of chronic myeloid leukaemia. Usually the progression of splenomegaly in chronic myeloid leukaemia is usually slow and insidious that many patients may not be aware of it until spleen has become huge in size. However spontaneous rupture of spleen is a very rare presentation of CML (Bauer et al., 1981).

Trauma is the most common cause of splenic rupture, while non-traumatic splenic rupture is a rare condition. Spontaneous non traumatic splenic rupture is a potentially life threatening complication. Spontaneous rupture usually occurs in a diseased spleen and is called a pathologic rupture1. Infection, malignancy, metabolic disorders, as well as vascular and hematological diseases are the usual reasons2. Disorders cause congestion or pathological infiltration of the spleen. Most commonly malignant cells of lymphoproliferative or myeloproliferative origin infiltrate the spleen directly.

From the review of the literature, we have been able to identify 136 cases of pathologic splenic rupture in haematological disorders since 1861; 34% have occurred in acute leukemias, 34% in non-Hodgkin's lymphomas, and 18% in chronic myelogenous leukemia. Total 11 chronic myeloid leukaemia cases with spontaneous splenic rupture have been reported in English literature. Of them 10 were male and the median age 49 years. Spontaneous splenic rupture as the initial presentation occurred in four of them. Seven of them could only be diagnosed at post mortem examination4. The sheer volume exceeds the capacity of the relatively non-distensible splenic capsule, causing splenic rupture and splenic haemorrhage. In our patient probably both the leukaemic infiltration and infarction were the causes of splenic rupture. Although blood coagulation abnormality is also a minor cause for splenic rupture, our patient’s coagulation profile was normal.

The choice of treatment for spontaneous splenic rupture is not only determined by haemodynamic stability, amount of blood products used but also by the underlying pathology (Giagounidis et al.,

1996). Due to a substantial risk of post-splenectomy infections, there has been a shift towards non-operative management in haemodynamically stable patients (Nix et al., 2001). Splenic artery embolization may be used as an adjunct to non-surgical management of splenic injury in haemodynamically stable patients. Giagounidis et al (1996) also reported that the mortality rate of spontaneous splenic rupture in haemalological malignancies was 37% in operated case and while 93.0% in non-operated cases.

Splenectomy exposes the patient to the dangers of post-operative complications (Ching-Liang et al., 1996). Post splenectomy sepsis is a potential lethal complication of splenectomy with a mortality of 50 to 80% (Asgaria and Begos 1997). The postoperative recovery was uneventful in our patient. 4 units of whole blood were transfused in perioperative period. She was immunized later with the vaccines. She didn't develop any sepsis till now. Her platelet count was raised up to 1010x109/L in immediate post operative days but no thrombo-embolic manifestation was evidenced. It is now within normal range.

Conclusion

Splenic rupture is a very dangerous condition. Careful and proper management is very much essential for a splenic rupture case.

Reference

Asgaria MM, Begos DG. Spontaneous splenic rupture in infections monomucleosis: A review. Yale J Biology Medicine. 1997;70:175–182

Bauer TW, Haskins GE, Armitage JO. Splenic rupture in patients with hematologic malignancies. Cancer 1981; 48: 2729-27335

Ching-Liang H, Yeu-Chinchen, Chao Tsue-Yi. Spontaneous splenic rupture in chronic myeloid leukaemia: A case report and review of literature. J Chinese Oncol Sac 1996;12(1):1–6

Giagounidis AA, Burk M, Meckenstock G, Koch AJ, Schneider W. Pathologic rupture of the spleen in hematologic malignancies: two additional cases. Ann Hematol. 1996;73(6):297-302

Nix JA, Costanza M, Daley BJ, et al. Outcome of the current management of splenic injuries. J Trauma 2001;50:835-42

Torricelli P, Coriani C, Marchetti M, Rossi A, Manenti A. Spontaneous rupture of the spleen: report of two cases. Abdom Imaging 2001; 26: 290-293