Rare Cause Of A Common Clinical Presentation: Case Report And Review Of Relevant Literature

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Citation

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Abstract

Pain in the right iliac fossa is a common presenting complaint in the A & E department. A significant number of cases have appendicitis, but occasionally some cases spring surprises. We present a case of delayed presentation of splenic rupture presenting as right iliac fossa pain. The diagnosis can be challenging especially if the history of trauma is not forthcoming from the patient or is of trivial nature and is ignored by the clinician, in the face of clinical findings.

CASE REPORT

20-year-old football player was brought in to A & E by ambulance with 2-hour history of acute abdominal pain. He was apyrexial with a pulse of 54/min, blood pressure of 110/70 mm Hg, RR of 18/min, SaO2 of 98% on air and a BM of 6.7 mmol/lit. He gave a history of gradual onset of central abdominal pain, which later became more acute in the right iliac fossa. The pain was sharp in nature, constant, non-radiating and worse on movement. He gave a past history of infectious mononucleosis 2 years back, which had completely resolved. He was a county football player and had recently been practising with his team.

On examination, he looked quite well built and fit. There was no evidence of any pallor or lymphadenopathy. Abdominal examination revealed localised tenderness with guarding and rebound in the right iliac fossa. Blood results showed an elevated white cell count and raised CRP. Chest X ray was normal. A diagnosis of acute appendicitis was made and he was listed for an emergency appendecectomy. On opening the peritoneum thru a Lanz incision, free peritoneal blood was found. The operation was converted into a full exploratory laparotomy through a liberal midline incision. On exploration, 1.5 lit of blood was aspirated from the abdominal cavity. The offending organ was found to be the spleen. It had a full thickness tear in the posterolateral aspect. Though a clot had formed in the tear there was active oozing from the margins. A standard splenectomy was carried out. Other solid organs were found to be normal.

In the postoperative period, the patient was further questioned with regards to history of trauma, even if trivial. After much thought, the patient gave history of being tackled while playing football 2-3 days back. He had been elbowed in his ribs on the left side and had fallen to the ground after that. After a few minutes, he had continued to play and had completely forgotten about it.

Grossly the spleen measured 110 X 90 X 65 mm and weighed 250 grams. Cut section revealed a 40 mm haematoma centrally within the specimen. Microscopy, showed a large blood clot within the parenchyma with surrounding neutrophilic infiltrate. In areas the sinuses were dilated but there was no definite evidence of a haemangioma. There was no evidence of infection such as infectious mononucleosis or malignancy. Two pathologists confirmed these findings.

He had an uneventful postoperative period and was discharged after 5 days. On follow up after 6 weeks, he had made a complete recovery.

DISCUSSION

The syndrome of delayed splenic rupture after blunt abdominal trauma, has fascinated surgeons since first description of delayed splenic rupture at the end of the 19 th century by Evans [$_1$].

In 1932, McIndoe estimated the incidence of delayed rupture of spleen to be 14% [$_2$]. He suggested that the term ‘delayed rupture of spleen' was imprecise, and instead favoured ‘delayed haemorrhage after traumatic rupture of the spleen' as more accurate and descriptive of the process. In 1970's

when diagnostic peritoneal lavage (DPL) was widely used for evaluation of blunt trauma the incidence dropped to 1 % to 2 % [3]. This suggested that true delayed rupture of spleen is an unusual sequel to blunt splenic trauma and that most patients thought to have delayed rupture of the spleen represent instead delayed recognition of splenic rupture.

To resolve the confusion surrounding the term ‘delayed splenic rupture', Kluger proposed four categories of delayed splenic rupture [1]. Category 1 was termed ‘true delayed splenic rupture' and consisted of the following criteria: history of blunt abdominal trauma, the absence of clinical evidence of intra abdominal injury (peritonitis) and normal findings on the CT scan of the abdomen on initial imaging. Category II is represented by our patient and was termed ‘delayed presentation of splenic injury' and was associated with the following clinical criteria: history of blunt abdominal trauma, the absence of clinical evidence of intra – abdominal injury, and a lack of CT imaging at initial evaluation (or negative DPL). In each of these first two categories, it was required that clinical deterioration occurs more than 48 hours after the initial traumatic injury. Category III delayed splenic ruptures were described as a ‘failure of non surgical management of splenic injury' and required early CT documentation of splenic injury followed by delayed haemorrhage and the need for surgical intervention. Category IV delayed ruptures were those that failed early surgical intervention including spleenorrhaphy or partial Splenectomy, only to be followed by recurrent haemorrhage leading to a second operation.

Allen et al have argued that the technological limits of radiographic studies should not be the core determining criteria for diagnosis of delayed rupture of the spleen, as the diagnostic accuracy and precision of imaging is constantly improving [4]. Thus, by rigid adherence to the classification system proposed by Kluger, the incidence of ‘true delayed rupture of the spleen' would change as our ability to detect the injury by radiographic means changes [4]. Indeed, review of English literature found only 11 cases of true delayed splenic rupture [1,5,6,7,8,9,10].

Two mechanisms have been postulated as possible causes for delayed rupture of the spleen and /or its delayed presentation. A gradual increase in the volume and tension of a subcapsular haematoma, probably due to clot lysis, resulting in increased oncotic pressure and subsequent free rupture, was the chief postulate for many years [9]. However, Black et al managed non-operatively 23 patients with CT diagnosed splenic subcapsular haematoma, none of which ruptured subsequently or required a delayed operation [11]. The second postulated theory is that a capsular tear and perisplenic haematoma is formed at the time of injury and is tamponaded by surrounding organs, free rupture into the peritoneal cavity occurring at a later date [1]. However, from recent studies and case reports it appears that delayed rupture/presentation is caused by a large spectrum of hilar or parenchymal injuries, with or without associated subcapsular hematomas [12].

Occasionally the trauma may be trivial or the individual may ignore it as part of sport injury due to competitive gesture or due to tolerance of pain. Present case did not seek any relief from pain or injury sustained during playing of football and only reported after 2 days when the blood had trickled down to right iliac fossa and given rise to features suggestive of localized peritonitis. A similar case of delayed splenic rupture presenting as acute appendicitis has been previously reported [13].

Spontaneous rupture of spleen is a rare but potentially fatal complication of infectious mononucleosis (IM), occurring in 0.1% to 0.5% of patients with proven active IM majority of cases occuring within 3 weeks of illness [14]. In order to make a diagnosis of spontaneous splenic rupture in infectious mononucleosis, the following criteria should be met: (1) no history of recent trauma, (2) hematological and serologic evidence of infectious mononucleosis, (3) recent clinical symptoms of infectious mononucleosis, and (4) histological examination of the spleen should show changes consistent with infectious mononucleosis [15]. Our patient had history of IM one year back, which had completely resolved. He did not have any lymphadenopathy on clinical examination and finally the histological examination of the spleen did not reveal any evidence of atypical lymphocytes characteristic of IM.

There are several considerations that can be taken from this unusual case of delayed presentation of splenic rupture. The history that we elicit from the patient remains our most valuable tool in determining the ultimate diagnosis and yet this simple history is often quite elusive in the A and E department. A history incompletely given or solicited will result in misdiagnosis. Persistent and repetitive questioning finally elicited the history of trauma in our patient, although appropriate therapy was instituted without the full history.

CONCLUSION

In conclusion we report an interesting case of a rare entity of

delayed presentation of splenic rupture, which may occur in 1% of blunt abdominal trauma victims. Such delayed ruptures can present atypically and after minor trauma that may be interpreted by the patient to be insignificant, and therefore forgotten. In these instances, continued clinical vigilance and early appropriate imaging will likely decrease morbidity.

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