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Spontaneous splenic rupture associated with influenza B virus

*Corresponding Author: Rishi Chavda Email: rishi.chavda@nhs.net

Abstract

Atraumatic Splenic Rupture (ASR) is rare relative to traumatic cases, being referred to as a spontaneous rupture in the presence of a histologically proven normal spleen. ASR carries with it a risk of mortality which has been reported to be as high as 12.2%, given the life-threatening potential a prompt diagnosis and subsequent management is critical. However, the aetiology of ASR is wide ranging and often and the clinical diagnosis can be obscured by signs and symptoms being attributed to the underlying disease process, thus requiring a high index of clinical suspicion. We present the first documented case of a spontaneous splenic rupture associated with Influenza B virus in English literature.

Keywords: Spontaneous splenic rupture; Atraumatic splenic rupture; Influenza B virus.

Introduction

Atraumatic Splenic Rupture (ASR) is a rare pathological entity relative to traumatic cases, being referred to as a spontaneous rupture in the presence of a histologically proven normal spleen, conversely nomenclature stipulates it is referred to as a pathological rupture in diseased spleens [1]. ASR carries with it a risk of mortality which has been reported to be as high as 12.2% [2], given the life-threatening potential a prompt diagnosis and subsequent management is critical. However, the aetiology of ASR is wide ranging and often and the clinical diagnosis can be obscured by signs and symptoms being attributed to the underlying disease process, thus requiring a high index of clinical suspicion.

Case presentation

A 41 year old female with no past medical history of note presented to the emergency department with a 3 day history of coryzal symptoms, a productive cough of yellow sputum, exertional shortness of breath, left sided pleuritic chest pain, myalgia and confusion. She was a previous cigarette smoker with a 9 pack year history and was currently vaping. Initial observations on admittance to hospital revealed an Early Warning Score **Rishi Chavda*; Vijay Chavda; Neil Bhardwaj** Department of HPB Surgery, Glenfield Hospital, Leicester, UK.

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(EWS) of 1 for a blood pressure of 106/64 mmHg, clinical examination was unremarkable beyond scanty left basal crepitations on auscultation, biochemical anomalies were a c-reactive protein of 83, platelet count 80x10⁹/L, prothrombin time 11.5 seconds and a lymphocyte count 0.36x10⁹/L with a normal overall white cell count. A chest x-ray revealed left lower zone patchy infiltrates and a respiratory viral screen returned positive for influenza B virus. She was commenced on intravenous antibiotics and oral oseltamivir for viral pneumonia with suspected secondary bacterial infection, being transferred to a respiratory ward. On day 2 of her admission she complained of worsening left sided chest/hypochondriac pain and continued to be hypotensive, presumed to be secondary to the underlying infection, before rapidly deteriorating later in the evening with an EWS of 10. A venous blood gas revealed a metabolic acidosis with a lactate of 5.6 mmol/L and a Haemoglobin (Hb) 7.8 g/dl. She was treated initially for septic shock before a repeat blood gas shortly after boluses of intravenous fluid showed a falling Hb to 4.4 g/dl, confirmed on laboratory results. The major haemorrhage protocol was declared with administration of blood products to also correct clotting before transfer to radiology for an urgent CT chest, abdomen and pelvis with a mesenteric angiogram. Imaging findings revealed a subcapsular splenic haematoma, with extensive intraperitoneal extension and active contrast extravasation consistent with ongoing haemorrhage confirming ASR with an American Association for the Surgery of Trauma (AAST) grade 4 splenic injury. This was alongside scattered patchy consolidative changes throughout the lungs, with atypical appearances, sparing the dependant areas being predominantly limited to the peri-hilar and anterior segments of the upper lobes as well as the right middle lobe and left lingula. The patient underwent an emergency open splenectomy later that evening. She made an uneventful recovery and was discharged home at day 7. Macroscopically the specimen was of normal appearances apart from the obvious haemarrhage. Microscopically the specimen retained architecture of the white pulp with normal immunohistochemistry, there was some expansion of the red pulp indicating a degree of mild congestion but no specific cause of the splenic rupture was identified.



Figure 1: CT-1.



Figure 2: CT-2.



Figure 3: CT-3.

Table 1: Examples of aetiology.

Aetiology	Examples
Neoplatic	Hodgkin's symphoma and primary/secondary neoplas- tic disorders such as angiosarcoma and lung cancer
Infectious	Viral, bacterial, fungal or protozoal in nature such as malaria
Inflammatory	Pancreatitis, systemic lupus erythematosus
Treatment related	Haemodialysis, oral anticoagulants
Mechanical disorders	Pregnancy, congestive splenomegaly secondary to liver cirrhosis and portal hypertension

Discussion

The management of ASR can be broadly divided into surgical with either a total splenectomy or organ preserving surgery and non-surgical with arterial embolization or conservative "watchful waiting". Whilst there remains no generalised consensus, the decision is influenced by a number of factors including, but not limited to, the degree of haemodynamic instability and ultimately the clinical condition of the patient, grade of splenic injury, availability of specialist interventional radiology services for arterial embolization and the suspected cause, with malignant causes favouring total splenectomy [3]. In a large systematic review of ASR Renzulli et al found that of 774 patients, 85.3% underwent surgery within 24 hours and of the remainder managed non-surgically, 17% eventually underwent secondary

splenectomy due to rebleeding and haemodynamic compromise [2]. However the benefits of non-surgical management must not be disregarded due to the spleens fundamental role in immunity and conferring protection against encapsulated organisms [4]. The underlying aetiological association of ASR is expansive and can be broadly categorised into distinct subgroups [2,5].

Of the above sub-groups Renzulli et al. found that malignant haematological, viral infectious and inflammatory disorders accounted for 42.1% of the 845 patients [2]. In a systematic review of ASR in 613 patients, Aubrey-Bassler et al found that the most common associated pathological processes were infectious in nature [6]. ASR may present with an array of clinical symptoms and signs obfuscating an accurate and time critical diagnosis. This is further compounded by the wide variety of aetiological conditions which may contribute to it. Given the associated high mortality, and can be attributed to any number of the associated aetiological conditions. Patients may present with abdominal pain, not solely in left upper quadrant as commonly depicted in textbooks as a result of hemoperitoneum thus imitating commoner causes of an acute abdomen. They may also present in the absence of any pain and instead with vague symptoms of shoulder tip pain referred to as Kehr's sign, chest pain, nausea and vomiting as well as weakness [7].

Conclusion

To summarise we have outlined the first documented case of a spontaneous splenic rupture associated with Influenza B virus in English literature. Although the majority of patients with ASR will undergo an emergency splenectomy, non-surgical management such as splenic artery embolization should be considered in clinically appropriate cases to preserve splenic function and thus avoid the need for vaccinations and lifelong antibiotic therapy. The associated pathological entities of ASR are vast and can present in a manner which strays from a textbook presentation, given the associated mortality a high index of clinical suspicion is required and this case presentation serves as a gentle reminder for clinicians to not overlook ASR in their list of differential diagnoses.

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