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Open splenectomy for Varicella zoster induced spontaneous splenic rupture

Mark Christopher Sykes[a,*], Bilal Azhar[b], Laurence John[c], Salman Bokhari[c]

[a] Imperial College London, South Kensington Campus, London SW7 2AZ, United Kingdom
[b] London Postgraduate School of Surgery, Russell Square Stewart House, WC1B 5DN, United Kingdom
[c] Northwick Park Hospital, Watford Rd, Harrow, HA1 3UJ, United Kingdom

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ABSTRACT

INTRODUCTION: Here we present a case of atraumatic splenic rupture secondary to varicella infection requiring emergency splenectomy. The presentation was as would be expected for epstein barr virus (EBV) related splenic injury, which is well documented in the literature. Dermatological findings however suggested varicella zoster, and viral serology subsequently confirmed the diagnosis.
PRESENTATION OF CASE: A young Romanian male presented to the emergency department with peritonism without preceding trauma. Free fluid on USS was aspirated as frank blood and cross-sectional imaging demonstrated a ruptured spleen. He underwent emergency splenectomy and recovered well. During his presentation he was noted to have an erythematous rash with different rates of evolution raising the suspicion for Varicella Zoster. This was subsequently confirmed on viral serology.
DISCUSSION: A number of precedents have been identified for spontaneous splenic rupture, however Varicella Zoster has only been reported a handful of times. A number of surgical options are available for splenic rupture, and guidelines exist for traumatic splenic injury. There is limited guidance on the most effective surgical management for spontaneous splenic ruptures with haemodyamic compromise.
CONCLUSION: Atraumatic splenic rupture should be considered as an important differential in those presenting with abdominal pain and peritonism without a history of preceding trauma. Haematological and infectious diagnoses should be sought to identify causation for the splenic rupture.

1. Introduction

Splenic rupture typically presents as left upper quadrant pain, peritonism, and haemodynamic compromise. The diagnosis is made with clinical suspicion being confirmed on ultrasound and/or computed tomography (CT). Reflecting the aetiology of the injury, splenic ruptures are considered either traumatic or atraumatic. Abdominal injury leading to immediate and delayed traumatic splenic rupture is a well recognised event [1]. Whilst less common [2], atraumatic rupture of the spleen is also widely documented in the literature and is associated with a pathological precedent in 93% of cases [3]. Infection precedes 27.3% of atraumatic splenic ruptures and is commonly related to Epstein-Barr virus and malaria [3–5].

Varicella zoster (VZV) is a highly infectious member of the human herpes viruses, often presenting as chickenpox in childhood leading to approximately 90% immunity amongst adults [6]. Following primary infection VZV remains dormant in dorsal root ganglia where it can reactivate in adulthood as herpes zoster. Systemic complications of VZV such as pneumonitis and encephalitis are rare and largely associated with the immunosuppressed population [7]. Here we present a rare case of atraumatic splenic rupture secondary to VZV presenting to a large district general hospital with emergency surgery capabilities.

Note this case report has been constructed in accordance with the SCARE guidelines [17].

2. Presentation of case

A 32 year old male presented to the accident and emergency department complaining of a 2 day history of worsening abdominal pain and chest pain. There were no other associated symptoms; notably his bowels had opened normally, he had no urinary symptoms, and there was no stigmata of systemic viral disease such as a sore throat nor rash. He could not recall any recent traumatic injury. There was no past medical/surgical history and he had no known unwell personal contacts.

His observations at presentation were a temperature of 36.8° centigrade, oxygen saturations 100% in room air, respiratory rate 16, blood pressure 164/99, and heart rate 108, giving him a national early warning score of [8].

Physical examination demonstrated an alert, acutely unwell adult male. He had a distended abdomen with generalised tender-

* Corresponding author at: Charing Cross Hospital Urology Department, Imperial College Healthcare Trust, Fulham Palace Road, London, W6 8RF, United Kingdom.
E-mail address: marksykes@nhs.net (M.C. Sykes).

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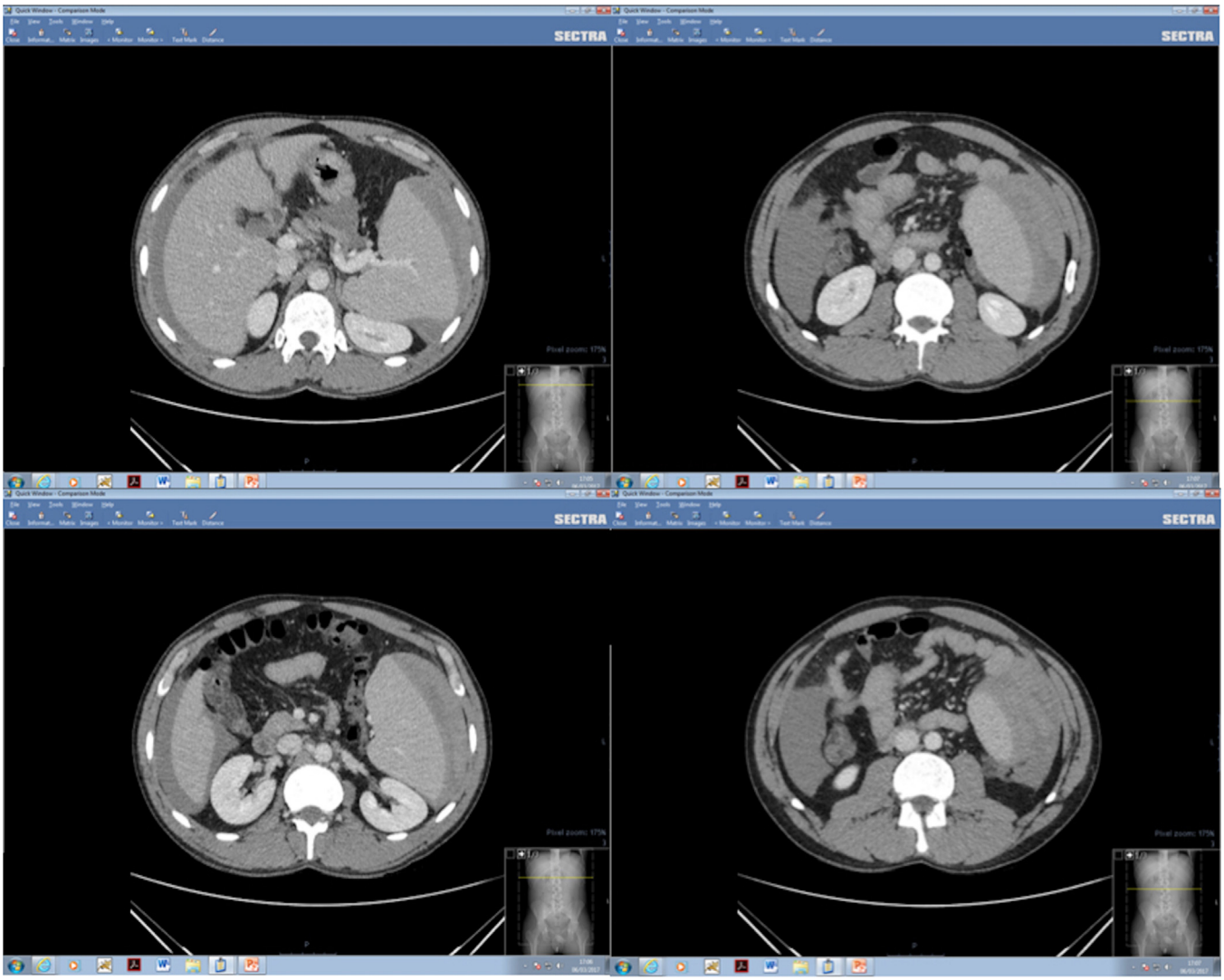

Fig. 1. CT abdomen (axial) demonstrating sub-capsular splenic haematoma.

ness, involuntary guarding throughout and evidence of peritonism. There was no palpable organomegaly at time of examination. He had no palpable lymphadenopathy or evidence of enlarged or exudative tonsils. Cardiorespiratory examination was normal.

Laboratory investigations revealed: Hb 120 g/L, WCC 5.5 × 10*9, platelet count 109 × 10*9, CRP 59.2, Creatinine 116, Urea 10.1, Amylase 37, lactate 0.8. A repeated haemoglobin test demonstrated a fall in levels to 104 g/L on full blood count.

The patient subsequently underwent a computed tomography (CT) scan of abdomen and pelvis with contrast (Figs. 1 and 2). This demonstrated an enlarged spleen with a contained collection lateral to the spleen, consistent with a substantial sub-capsular haematoma. Free-intra abdominal fluid was also demonstrated which was subsequently aspirated under USS guidance and confirmed as blood. A CT mesenteric angiogram (Figs. 1 and 2) was performed which demonstrating irregularity in the superior anterolateral corner of the spleen, confirming splenic rupture. No arterial aneurysm were identified including those of the splenic artery.

The patient was initially resuscitated and an emergency open splenectomy was performed. A ruptured splenic capsule with a large adherent haematoma was identified at laparotomy (Fig. 3). The spleen was medialised and the splenic vessels controlled and ligated at the hilum, allowing safe excision of the spleen

In light of the atraumatic splenic rupture in the presence of splenomegaly a viral screen was performed. The initial screen did not include varicella. Immunoglobulin quantification and autoantibody testing including a direct antiglobulin test were also performed:

CMV IgG positive (evidence of previous infection)
CMV IgM negative
EBV VCA IgG/IgM negative
EBV EBNA IgG positive (evidence of EBV infection over 8 weeks ago)
Heb B SAg non reactive
Hep C abs non reactive
HIV-1/-2 abs non reactive

The patient was reviewed by infectious diseases team who commented on a new rash with erythematous clusters with different rates of evolution. They also reported a palpable spleen. Based on this finding the impression was a varicella induced systemic illness, with subsequent spontaneous splenic rupture. He was commenced on acyclovir, and varicella and herpes simplex serology was sent which confirmed this suspicion:

Varicella Zoster DNA detected
Herpes simplex type 1 not detected
Herpes simplex type 2 not detected