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Case Report

A case report of successful splenic artery embolization for atraumatic splenic rupture secondary to Epstein Barr virus infection in a haemodynamically unstable patient ☆,☆☆

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ABSTRACT

Splenic rupture in haemodynamically unstable patients has traditionally been managed with splenectomy. This case report discusses the successful management of atraumatic splenic rupture, a rare but life-threatening complication of Epstein-Barr virus (EBV) infection, in a hemodynamically unstable patient. The patient, diagnosed with infectious mononucleosis (IM) secondary to EBV, presented with severe abdominal pain and a syncopal episode. Imaging revealed an American Association for the Surgery of Trauma (AAST) grade III splenic injury, which was subsequently upgraded to a grade IV injury on repeat imaging. The patient's condition deteriorated even with initial resuscitation, leading to splenic angioembolization. The procedure was successful and the patient was discharged after 5 days. This case highlights the efficacy of splenic artery embolization (SAE) in haemodynamically unstable patients with atraumatic splenic rupture, particularly in centers with interventional radiology resources, offering an alternative to splenectomy and its associated complications.

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Introduction

Epstein-Barr virus (EBV) is a common viral pathogen, typically presenting as Infectious Mononucleosis (IM) with symptoms such as fever, malaise, pharyngitis, and lymphadenopathy [1]. It affects 90% of adolescents and young adults before the age of 30 worldwide. Splenic involvement is a hallmark of EBV pathogenesis, with splenomegaly resulting in an increased splenic rupture risk.

Although rare, atraumatic splenic rupture is a life-threatening complication of EBV. Splenic rupture classically presents with left upper quadrant pain radiating to the left shoulder tip (Kehr sign) due to diaphragmatic irritation from the hemorrhage[3]. Management of splenic rupture historically involved operative management with splenectomy. In recent years, nonoperative management with splenic artery embolization (SAE) has become increasingly more prevalent in haemodynamically stable patients [2–5]. This report presents a case of the successful management of atraumatic splenic rupture in a hemodynamically unstable patient.

Case report

A 27-year-old male presented to the emergency department (ED) of a regional hospital following a conscious collapse in the hospital car park. He had driven to hospital with severe abdominal pain on the background of being unwell with a sore throat, myalgia and arthralgia for 3 weeks. The patient had a consultation with his general practitioner (GP) a few days prior to the hospital presentation and was commenced on oral phenoxymethylpenicillin. A diagnosis of infectious mononucleosis (IM) secondary to Epstein Barr Virus (EBV) infection was made on the basis of serology tests which were sent off during the consultation with the GP.

The morning of the ED presentation, the patient had developed epigastric pain and nausea. Later in the afternoon, the pain progressively worsened and was radiating to the left shoulder tip. He subsequently had a syncopal episode immediately prior to driving to the hospital. The patient denied any recent trauma or any significant previous medical history.

Upon presenting to ED, the patient had a heart rate of 105 beats per minute (BPM) and blood pressure of 96/73 mmHg. He was afebrile and the respiratory rate and oxygen saturations were within normal limits. He was focally tender in the left upper quadrant (LUQ) and underwent a point of care ultrasound scan which showed free fluid in the LUQ around the spleen. Venous blood gas results were significant for a pH of 7.30, HCO3- of 27 mmol/L, pCO2 55 mmHg and lactate 3.9 mmol/L. The formal hemoglobin was 134 g/L. A unit of packed red blood cells was commenced and a contrast enhanced computed tomography (CT) scan of the abdomen and pelvis was performed urgently. The scan demonstrated an American Association for the Surgery of Trauma (AAST) grade III splenic injury with a large peri-splenic and subcapsular haematoma, without obvious contrast extravasation (Fig. 1). The General Surgery team were consulted and a decision was made for nonoperative management. The patient was promptly transferred to a tertiary center with interventional radiology facilities for consideration of splenic angioembolization.

Upon transfer to the tertiary center, the patient remained alert and oriented. However, his condition hemodynamically and biochemically had worsened. His heart rate at the time was 115 BPM and blood pressure was 80/50 mmHg. Biochemically, the formal haemoglobin was 88 g/L, and venous blood gas demonstrated a pH 7.27, pCO2 54 mmHg, HCO3 24 mmol/L, lactate 4.1 mmol/L. The patient was resuscitated with further packed red blood cells and a repeat CT abdominal angiogram was urgently performed. The repeat scan performed 7 hours after the initial CT scan, showed increasing volume of splenic hemorrhage with a potential contrast blush on the delayed phase at the lateral aspect of the splenic mid-pole (Fig. 2).

The injury on repeat CT was consistent with a AAST grade IV injury, a higher-grade injury when compared with the initial CT scan. The patient was discussed between the General Surgery team and the Interventional Radiologist on Call and a decision was made to proceed with splenic angioembolization. The patient was urgently transferred to the angiography suite. The right common femoral artery was accessed using a 5-french (Fr) sheath. The celiac axis was cannulated with a 5-Fr C2 catheter (Cook Medical). Angiography confirmed a large parenchymal defect in the central spleen with abnormally slow flow in an arterial branch within the defect. This vessel was selectively cannulated and embolized with 3-4 mm micro-coils (Concerto, Medtronic). Given the severity of the splenic injury, the proximal splenic artery was embolized with a 7 mm Amplatz vascular plug to reduce splenic arterial perfusion pressure. Completion angiography demonstrated no ongoing hemorrhage and the catheter was withdrawn (Fig. 3).

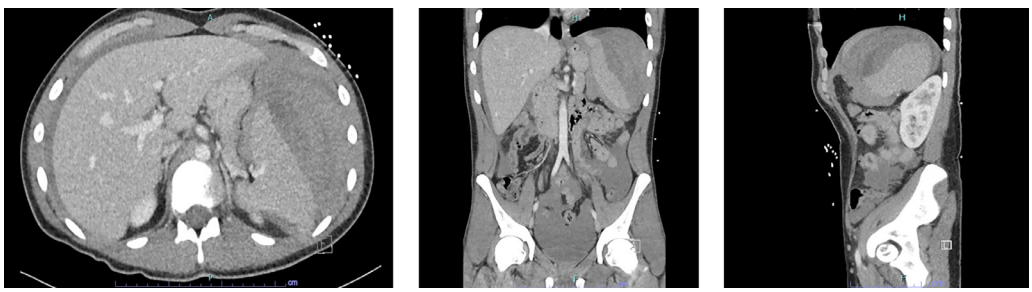

Fig. 1 – Axial, coronal and sagittal CT imaging showing AAST grade III splenic injury with a large subcapsular and perisplenic haematoma.

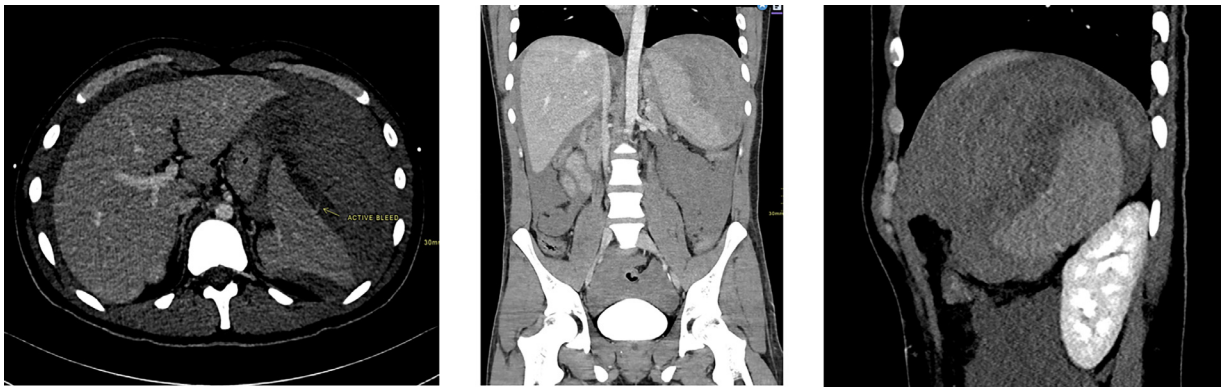

Fig. 2 – Axial, coronal, and sagittal CT imaging showing an AAST grade IV splenic injury with a potential area of contrast extravasation just lateral to the splenic midpole.

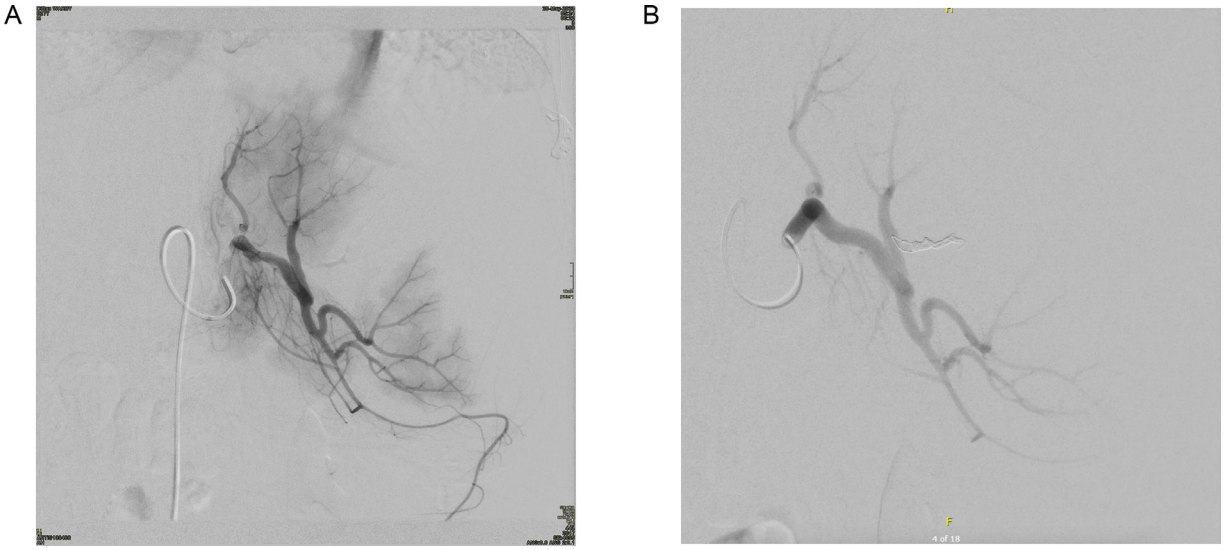

Fig. 3 – (A) Ruptured spleen with a large cleft in the center (B) Imaging post-embolization demonstrates coil embolization of a splenic arterial branch and peripheral 'pruning' of splenic parenchymal enhancement.

The patient was admitted to the intensive care unit for observation. There were no peri-procedural complications and the patient was discharged home 5 days post admission. The patient was followed up by his GP 4 weeks post embolization, during which blood tests including a full blood count with film were performed. The haemoglobin levels were stable and Howell-Jolly bodies were not detected. The patient was considered to have no evidence of hyposplenism. Follow-up imaging is not routinely conducted by the tertiary level health service in the absence of clinical concern.

Discussion

Splenic rupture, even-though rare, is the most common cause of mortality in Infectious Mononucleosis (IM) and occurs in less than 0.5% of cases [6]. The pathophysiology resulting in splenic rupture is considered to be a result of the splenomegaly caused by lymphocytic infiltration, stretching of the splenic capsule, and weakening of the trabeculae [7]. A systematic review of IM associated splenic rupture case reports revealed a patient population with an average age of 22 years and majority being male (70%). The average time between onset of symptoms and splenic rupture ranged from 2-8 weeks. Interestingly, a preceding history of trauma was only reported in 14% of patients [8]. Given the risk of mortality from splenic rupture in IM, it is important that patients with IM are counselled appropriately. Current guidelines recommend abstaining from contact sports for 1 month after onset of symptoms and until asymptomatic [9] (Table 1).

The grading of splenic injury is based on the American Association for the Surgery of Trauma (AAST) classification. The initial CT demonstrated a AAST grade III injury due to the presence of a subcapsular haematoma, involving >50% of the surface area. The presence of active bleeding in the second CT study resulted in the upgrading to a grade IV splenic injury [10]. Traditionally, splenectomy formed the basis of the man-

Table 1 – American Association for the Surgery of Trauma (AAST) splenic injury grading scale.

Grade	Injury	Description
I	Haematoma	Subcapsular, <10% surface area
	Laceration	Capsular tear, <1 cm parenchymal depth
II	Haematoma	Subcapsular, 10%-50% surface area
		Intraparenchymal, <5 cm diameter
	Laceration	Parenchymal laceration 1-3 cm
III	Haematoma	Subcapsular, >50% surface area or expanding
		Ruptured subcapsular or intra-parenchymal haematoma > 5 cm
	Laceration	Parenchymal laceration >3 cm in depth
IV	Vascular	Any injury in the presence of a splenic vascular injury (pseudoaneurysm or arteriovenous fistula) or active bleeding confined to the splenic capsule
	Laceration	Laceration of segmental or hilar vessels producing major devascularisation (>25% of spleen)
V	Laceration	Shattered spleen
	Vascular	Any injury in the presence of a splenic vascular injury with active bleeding extended beyond the spleen into the peritoneum

agement of splenic injury [11]. In recent years, splenic artery embolization (SAE) is advocated in hemodynamically stable patients [12]. However, surgical management with splenectomy is still carried out for patients presenting with hemodynamic instability despite resuscitation and evidence of splenic injury on contrast CT. The evidence to support splenic angioembolization in haemodynamically unstable patients is limited. A retrospective study of 1052 hypotensive patients presenting with blunt splenic injury, 95% of patients underwent splenectomy and 5% of patients were treated with SAE. The mortality rates of patients who underwent SAE were similar to those who underwent splenectomy [13]. In contrast, splenectomy is associated with a longer hospital stay and increased lifetime risk of infection with encapsulated bacteria [14].

Splenic injury of AAST grade III or higher, evidence of active contrast extravasation and pseudoaneurysm formation are indications for SAE. SAE promotes hemostasis by reducing flow in the main splenic artery while preserving lower pressure flow to the splenic parenchyma by collateral vessels [5]. While preserving splenic function, SAE has achieved high success rates in managing splenic injuries from trauma in both children and adults [15]. Nevertheless, the rates of SAE as a modality of treatment in atraumatic splenic rupture remain low compared to splenectomy even in haemodynamically stable patients [6,8].

Conclusion

This case highlights the success of SAE in treating a patient with atraumatic splenic rupture secondary to EBV infection even in the setting of haemodynamic instability. The patient was able to avoid the long-term effects of asplenia and avoid the risk of reduced immune function. Operative management with splenectomy remains the standard of care for haemodynamically unstable patients with splenic rupture. However, in centers where interventional radiology resources are readily available, there may be a role for SAE as an intervention without an increased risk of mortality. Furthermore, this case also highlights the utility of SAE as a treatment option in atraumatic splenic rupture.

Patient consent

Written informed consent was obtained from the patient included in the study and a copy is available for the Editor-in-chief by request.

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