

Spontaneous atraumatic splenic rupture secondary to infectious mononucleosis

P. Sweeney¹, K. McKevitt², J. Ní Mhuirheartaigh², Y. Abdeldaim¹.

1. Department of Surgery, University Hospital Limerick, St Nessian's Rd., Dooradoyle, Co. Limerick, Ireland.
2. Department of Radiology, University Hospital Limerick, St Nessian's Rd., Dooradoyle, Co. Limerick, Ireland.

Abstract

Presentation

A healthy 23-year-old man developed sore throat and fever with positive EBV Infectious Mononucleosis test, followed by abdominal pain, vomiting, and haemodynamic collapse, without trauma.

Diagnosis

Bedside ultrasound suggested intraperitoneal free fluid. Urgent contrast-enhanced CT demonstrated large haemoperitoneum, perisplenic and subcapsular haematoma, and active splenic contrast extravasation.

Treatment

Emergency laparotomy revealed massive haemoperitoneum, and confirmed splenic bleeding; splenectomy was performed. Recovery was uncomplicated, with post-splenectomy antibiotics and vaccinations. He was well at three-month follow-up.

Discussion

EBV-associated atraumatic splenic rupture is rare yet life-threatening. CT imaging confirms injury and bleeding. Management depends on physiology; unstable patients require prompt surgical management, whereas stable cases may merit spleen-preserving strategies and close monitoring.

Introduction

Infectious mononucleosis (IM) secondary to Epstein–Barr virus (EBV) is usually self-limiting, however splenic enlargement predisposes patients to atraumatic splenic rupture (ASR) – an uncommon yet life-threatening complication reported in approx. 0.1 – 0.5% of IM cases. Most cases occur within the first month of illness, and are more common among men. Prompt recognition and haemorrhage control are critical. Decisions between spleen-preserving strategies and splenectomy hinge primarily on haemodynamic stability and imaging evidence of active bleeding¹⁻³.

Case Report

A previously healthy 23-year-old man presented to his GP after two days of sore throat and fever. Monospot test with the GP was positive for IM. The following day he developed abdominal pain and vomiting; by day four he attended the emergency department with severe diffuse abdominal pain, vomiting, fever, and malaise. He denied trauma or recent contact sports. Examination revealed a soft, but diffusely tender abdomen with guarding and rebound. Initial laboratory tests showed haemoglobin 13.8 g/dL, WCC 28.8 x10⁹/L with neutrophilia and lymphocytosis, elevated liver enzymes (ALT 677 U/L, ALP 271 U/L, GGT 167 U/L), lactate was 3.8 mmol/L, renal profile was normal. Peripheral smear showed numerous plasmacytoid lymphocytes. He received intravenous fluids and analgesia.

On subsequent surgical review, he deteriorated acutely with pallor, clamminess, syncope, hypotension (85/50 mmHg), and tachycardia (122 bpm). Bedside ultrasound suggested intraperitoneal free fluid. Urgent contrast-enhanced CT demonstrated large-volume haemoperitoneum, a >10 cm perisplenic and subcapsular haematoma, and active contrast extravasation from the spleen surface, with pooling inferiorly, consistent with splenic rupture.

He underwent emergency exploratory laparotomy. A massive haemoperitoneum was evacuated, active splenic bleeding was confirmed and splenectomy performed with ligation of the splenic vessels. Other intra-abdominal organs were unremarkable. Histopathology showed preserved splenic architecture with focal subcapsular and perisplenic haemorrhage consistent with rupture. No evidence of malignancy was observed. Recovery included a short ICU stay before ward transfer. He received routine post-splenectomy antibiotics and vaccinations as per local practice, and was well at three-month follow-up.

Discussion

EBV-related ASR is rare but can be catastrophic. Incidence estimates in IM range from 0.1% to 0.5%. Most reported ruptures occur within 21 days of symptom-onset. Reported mortality ranges from 4–9% in modern case series²⁻⁴. EBV causes lymphoid hyperplasia, capsular thinning, and splenic congestion, rendering the spleen friable and susceptible to rupture in the absence of trauma³.

Clinical recognition can be challenging as systemic IM symptoms may mask evolving intra-abdominal manifestations. Contrast-enhanced CT is the diagnostic standard when ASR is suspected—identifying haemoperitoneum, and active splenic bleeding².

Management is dictated by physiology. Unstable patients or those with imaging evidence of ongoing haemorrhage require urgent definitive surgical intervention—typically splenectomy, as in this case. Whereas stable patients may be considered for spleen-preserving strategies, including close observation or splenic artery embolization, ideally in centres with rapid access to surgery should non-operative measures fail^{5, 6}.

Post-splenectomy, patients should receive guideline-directed immunisations for asplenia (pneumococcal, MenACWY, MenB, Hib, plus annual influenza vaccines), with timing generally 4 weeks before elective splenectomy or 2 weeks after emergency splenectomy. Post-splenectomy antibiotic prophylaxis should also be offered according to local guidelines, with counselling on the risk of overwhelming infection and the importance of prompt care for febrile illness⁷.

Finally, as rupture risk is highest early, activity restriction after EBV infection is prudent. Current recommendations suggest at least 3 weeks without athletic activity from symptom onset, with some experts advising longer restriction (4–6 weeks) for contact sports^{4, 8}.

This case emphasises that sudden decompensation in mononucleosis should prompt immediate imaging and surgical consultation; in unstable patients with active bleeding, definitive surgical management remains lifesaving³.

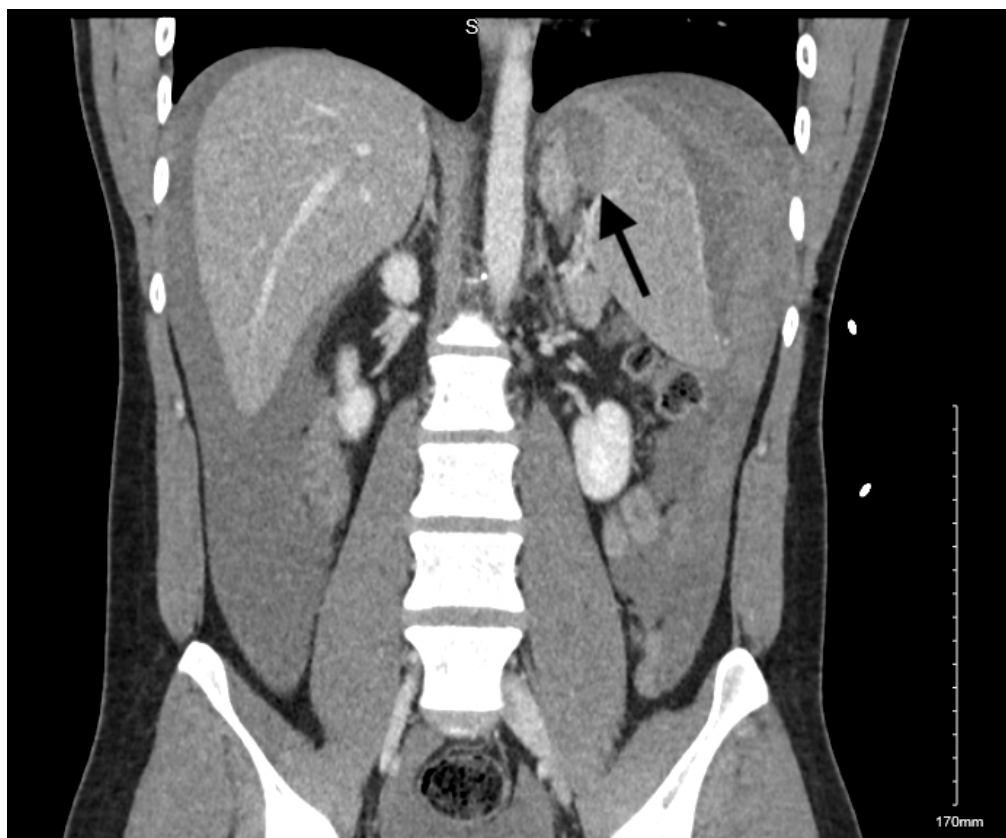


Figure 1: Coronal CT image with arrow illustrating haematoma, and active contrast extravasation from the spleen.



Figure 2: Axial CT image with arrow illustrating haematoma, and active contrast extravasation from the spleen.

Declarations of Conflicts of Interest:

None declared.

Corresponding author:

Patrick Sweeney
Department of Surgery,
University Hospital Limerick,
St Nessian's Rd.,
Dooradoyle,
Co. Limerick,
Ireland.

E-Mail: patricksweney24@rcsi.ie

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