

A Rupture without a Blow: Atraumatic Splenic Rupture with Haemoperitoneum Revealing Inflammatory Splenomegaly and Profound Thrombocytopenia in a Young Adult

A. Mandek Farah^{1*}, H. Ezzaki², F. Riahi², A. Benfaida², N. Chouaib², S. Jidane², K. Mounir², A. Belkouch²

¹Emergency Department, Mohammed V Military Teaching Hospital, Rabat, Morocco

²Faculty of Medicine and Pharmacy of Rabat, Mohammed V University, Rabat, Morocco

DOI: <https://doi.org/10.36347/sjmc.2026.v14i10.001>

| Received: 29.07.2026 | Accepted: 21.09.2026 | Published: 01.10.2026

*Corresponding author: A. Mandek Farah

Emergency Department, Mohammed V Military Teaching Hospital, Rabat, Morocco

Abstract

Case Report

Atraumatic splenic rupture is a rare but potentially lethal cause of haemoperitoneum which, in more than nine out of ten cases, is the first manifestation of an underlying splenic or systemic disease. We report the case of a 28-year-old man with no medical history who was admitted to the emergency department for acute pain of the left hypochondrium occurring without any trauma, fall, sporting activity or recent invasive procedure. He was haemodynamically stable, with a blood pressure of 130/60 mmHg and a heart rate of 90 beats per minute, and had no peritoneal signs. Contrast-enhanced thoraco-abdomino-pelvic computed tomography (CT) demonstrated splenomegaly measuring 17 cm in long axis with a heterogeneous parenchyma, several lacerations more than 3 cm deep, an intraparenchymal haematoma of the upper pole, foci of splenic infarction, and a dense intraperitoneal effusion in the perisplenic space, the paracolic gutters and the pelvis, consistent with haemoperitoneum. The blood count on admission showed a haemoglobin concentration of 14.1 g/dL contrasting with profound thrombocytopenia at 5,000/mm³, leucocytosis of 22,100/mm³, C-reactive protein of 295 mg/L and ferritin of 4,943 ng/mL. An extensive aetiological work-up comprising viral serologies, blood cultures, autoimmune screening and protein electrophoresis identified no infectious or autoimmune cause; Epstein-Barr virus serology showed a profile of past rather than acute infection. Because the patient remained haemodynamically stable with no evidence of active bleeding, non-operative management was chosen, combining intensive monitoring, correction of the thrombocytopenia with the haematology team, multimodal analgesia and serial blood counts. Follow-up CT showed stable splenic lesions, no active bleeding and regression of the haemoperitoneum, and the patient was discharged on day 10 with his spleen preserved. Haemodynamic stability allows spleen-preserving management even when a profound platelet abnormality coexists; the rupture must then be read as the first sign of a disease still to be identified.

Keywords: Atraumatic splenic rupture; spontaneous splenic rupture; haemoperitoneum; splenomegaly; severe thrombocytopenia; non-operative management; spleen preservation; emergency medicine; case report.

Copyright © 2026 The Author(s): This is an open-access article distributed under the terms of the Creative Commons Attribution 4.0 International License (CC BY-NC 4.0) which permits unrestricted use, distribution, and reproduction in any medium for non-commercial use provided the original author and source are credited.

INTRODUCTION

Atraumatic splenic rupture (ASR), also referred to as spontaneous splenic rupture, is defined as rupture of the splenic parenchyma and/or capsule in the absence of any identifiable traumatic event. It is a rare condition whose true incidence remains unknown, because it is reported almost exclusively in the form of case reports and small series [1,2]. Its clinical presentation may mimic any cause of acute abdomen, and the diagnosis is frequently established only on imaging, sometimes after haemorrhagic shock has already developed.

Atraumatic rupture is far more often a revealing event than an isolated accident. In the largest systematic review available, covering 845 patients reported in 632 publications, Renzulli et al. showed that the spleen was pathological in 93.0% of cases: neoplastic disorders accounted for 30.3%, infectious causes for 27.3%, non-infectious inflammatory diseases for 20.0%, drug- and treatment-related causes for 9.2% and mechanical causes for 6.8%, while a strictly normal spleen was found in only 7.0% of patients [1]. Reviewing 613 cases occurring in patients with no previously known disease, Aubrey-Bassler and Sowers found a similar hierarchy, dominated by infection (143 cases), haematological disease (84

Citation: Mandek Farah, A., Ezzaki, H., Riahi, F., Benfaida, A., Chouaib, N., Jidane, S., Mounir, K., & Belkouch, A. (2026). A Rupture without a Blow: Atraumatic Splenic Rupture with Haemoperitoneum Revealing Inflammatory Splenomegaly and Profound Thrombocytopenia in a Young Adult. *Sch J Med Case Rep*, 14(10), 2112-2118. <https://doi.org/10.36347/sjmc.2026.v14i10.001>

cases), medical procedures (112 cases, of which 87 were colonoscopies), non-haematological neoplasms (48 cases) and drugs (47 cases) [2].

The prognosis remains serious. Overall mortality reached 12.2% in the series of Renzulli *et al.*, and was significantly increased by splenomegaly, age above 40 years and an underlying neoplastic disorder [1]. Historically, treatment consisted almost invariably of total splenectomy, performed in 84.1% of patients, whereas conservative measures were used in only 14.7% [1]. In parallel, in blunt splenic trauma, non-operative management has become the standard of care for haemodynamically stable patients irrespective of injury grade, with success approaching 90% in experienced centres [3].

Transposing non-operative management to atraumatic rupture is attractive, because it preserves the immune functions of the spleen and avoids the lifelong risk of overwhelming post-splenectomy infection; it is, however, far less well codified, particularly when a severe haemostatic abnormality coexists. Through this observation we report an atraumatic splenic rupture with haemoperitoneum occurring in a young adult whose platelet count was 5,000/mm³, successfully managed without surgery, and we discuss the diagnostic and therapeutic reasoning that made splenic preservation possible.

CASE PRESENTATION

A 28-year-old man with no relevant medical or surgical history, and taking no medication in particular no antiplatelet agent and no anticoagulant was admitted to the emergency department in August 2026 for acute abdominal pain predominating in the left hypochondrium. He reported no trauma of any kind: no fall, no road traffic accident, no sporting activity and no

recent endoscopic or other invasive procedure. There was no personal or family history of splenomegaly, haematological disease or bleeding disorder, and no recent febrile or pharyngeal illness.

On admission the patient was conscious, alert and cooperative. He was haemodynamically stable, with a blood pressure of 130/60 mmHg and a regular heart rate of 90 beats per minute. He was afebrile and eupnoeic. Abdominal examination found marked tenderness of the left hypochondrium without generalised guarding or rebound tenderness, and no mucocutaneous haemorrhagic signs were observed despite the profound thrombocytopenia identified subsequently. Given the intensity of the pain and its unexplained character, contrast-enhanced thoraco-abdomino-pelvic computed tomography (CT) was performed without delay, during the night of admission.

CT demonstrated an enlarged spleen measuring 17 cm in long axis, with a heterogeneous parenchyma containing several lacerations more than 3 cm deep and an intraparenchymal haematoma of the upper pole, together with foci of infarction predominating at the lower pole and discrete stranding of the perisplenic fat (Figures 1-3). A dense intraperitoneal effusion was present in the perisplenic space, along both paracolic gutters and in the pelvis, consistent with haemoperitoneum (Figures 1, 4). No active contrast extravasation was identified. The liver was of normal size and homogeneous, with a tongue of left hepatic parenchyma extending over the spleen. The pancreas, kidneys, adrenal glands and bowel loops were normal, and there was no intra-abdominal lymphadenopathy on this first examination. At the thoracic level, only a small right basal atelectasis was found, with no pleural or pericardial effusion and no suspicious pulmonary nodule.

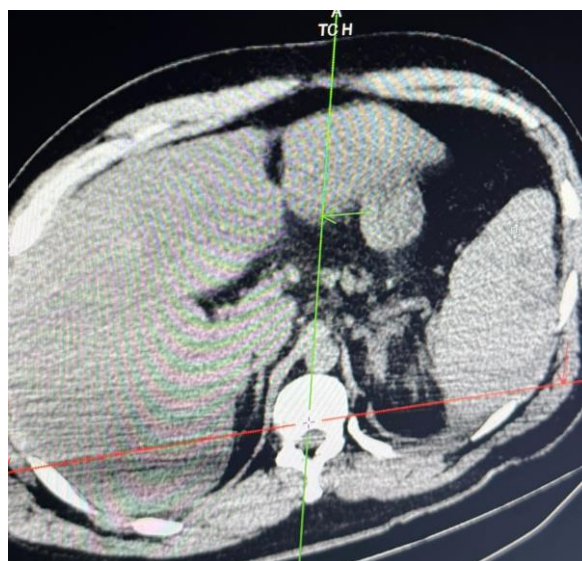


Figure 1: Axial contrast-enhanced CT of the upper abdomen at the time of admission

The spleen is enlarged and heterogeneous, with hypodense areas corresponding to lacerations and infarcts. A dense perisplenic and perihepatic effusion is visible, consistent with haemoperitoneum. The green

arrow indicates the tongue of left hepatic parenchyma extending over the splenic region. Patient-identifying data have been masked. CT: computed tomography.

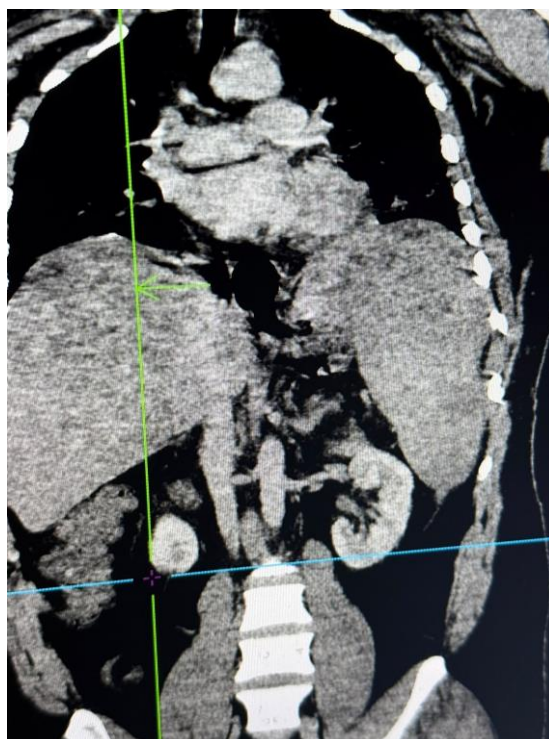


Figure 2: Coronal reformation of the contrast-enhanced abdominal CT

Splenomegaly measuring 17 cm in long axis with a heterogeneous parenchyma is demonstrated, together with the relationship between the spleen, the

liver and the left kidney. The green arrow indicates the hepato-splenic interface and the blue line the level of the corresponding axial section. CT: computed tomography.



Figure 3: Reformatted image centred on the spleen

The splenic parenchyma is heterogeneous and is surrounded by a crescentic hypodense collection

corresponding to the perisplenic haematoma, with discrete stranding of the adjacent fat.

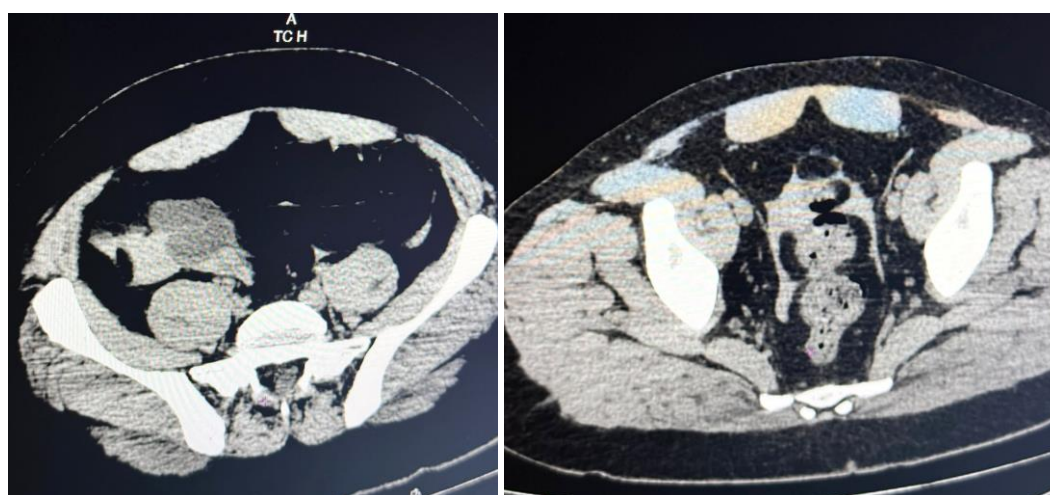


Figure 4: Axial CT sections of the lower abdomen (upper panel) and of the pelvis (lower panel).

Dense free intraperitoneal fluid is seen between the bowel loops, in the paracolic gutters and in the pelvic recesses, including the pouch of Douglas, consistent with haemoperitoneum. CT: computed tomography.

The blood count obtained on admission showed a haemoglobin concentration of 14.1 g/dL, leucocytosis of 22,100/mm³ with 16,300 neutrophils/mm³, and profound thrombocytopenia at 5,000/mm³. After correction of the thrombocytopenia in collaboration with the haematology team, the platelet count had risen to 17,900/mm³ on the sample drawn later the same day, at

which time the haemoglobin concentration was 13.0 g/dL. Haemostasis showed a prothrombin time of 56.1%, an international normalised ratio of 1.39, a normal activated partial thromboplastin time ratio of 0.83 and a raised fibrinogen concentration of 5.70 g/L. A major inflammatory syndrome was present, with C-reactive protein of 295.1 mg/L, an erythrocyte sedimentation rate of 48 mm in the first hour, ferritin of 4,942.8 ng/mL, hypoalbuminaemia of 27.8 g/L and a serum protein electrophoresis profile consistent with inflammation. The complete results are presented in Table 1.

Table 1: Laboratory results on admission and later on the same day

Laboratory test (unit)	On admission	Same day, second sample	Reference interval
Haemoglobin (g/dL)	14.1	13.0	13.0-17.0
Haematocrit (%)	41.6	39.2	40-54
Red blood cells ($\times 10^{12}/L$)	5.41	4.98	4.3-6.0
Mean corpuscular volume (fL)	76.9	78.7	78-98
White blood cells (/mm ³)	22,100	22,900	4,000-11,000
Neutrophils (/mm ³)	16,300	18,200	1,500-7,000
Lymphocytes (/mm ³)	2,750	1,690	1,000-4,500
Platelets (/mm ³)	5,000	17,900	150,000-450,000
Prothrombin time (%)	66.9	56.1	70-100
International normalised ratio	1.25	1.39	-
Activated partial thromboplastin time ratio	-	0.83	< 1.2
Fibrinogen (g/L)	-	5.70	2.0-4.5
Erythrocyte sedimentation rate (mm/1st h)	-	48	< 15
C-reactive protein (mg/L)	-	295.1	< 5
Ferritin (ng/mL)	-	4,942.8	21-274
Total serum protein (g/L)	-	55.7	64-83
Albumin (g/L)	-	27.8	40.2-47.6
Gammaglobulins (g/L)	-	7.2	8.0-13.5

The first sample was obtained on arrival in the emergency department and the second later on the same day, after correction of the thrombocytopenia in collaboration with the haematology team. Serum sodium, potassium, chloride, creatinine and liver enzyme concentrations were within their respective reference intervals. Dashes indicate tests that were not performed on the corresponding sample.

An extensive aetiological work-up was undertaken in parallel with the surveillance of the splenic lesions. Serological testing for human immunodeficiency virus, hepatitis B surface antigen and hepatitis C virus was negative. Cytomegalovirus and Epstein-Barr virus serologies showed profiles of past infection, with in particular positive anti-EBNA and anti-VCA IgG antibodies and negative anti-VCA IgM

antibodies, formally excluding acute infectious mononucleosis. Toxoplasma and leishmaniasis serologies were negative. Aerobic and anaerobic blood cultures remained sterile after seven days of incubation, and urine culture was sterile. Autoimmune screening was negative, with antinuclear antibodies below a titre of 1/100, negative anti-double-stranded DNA antibodies, negative anticardiolipin and anti- β 2-glycoprotein I

antibodies of both IgG and IgM isotypes, normal C3 and C4 complement fractions and a rheumatoid factor below 20 IU/mL. Screening for a lupus anticoagulant showed a raised dilute Russell viper venom time ratio of 1.68, but the mixing study corrected, so that the profile was interpreted as reflecting a coagulation factor deficiency rather than a true lupus anticoagulant. These results are summarised in Table 2.

Table 2: Aetiological work-up performed during hospitalisation

Investigation	Result
HIV p24 antigen and anti-HIV 1/2 antibodies	Negative
Hepatitis B surface antigen	Negative
Anti-hepatitis C virus antibodies	Negative
Cytomegalovirus IgG / IgM	Positive / negative (past infection)
Epstein-Barr virus anti-EBNA / anti-VCA IgG / anti-VCA IgM	Positive / positive / negative (past infection)
Toxoplasma gondii IgG / IgM	Negative / negative
Leishmania serology	Negative
Aerobic and anaerobic blood cultures	Sterile after 7 days of incubation
Urine culture	Sterile
Antinuclear antibodies	Negative (titre < 1/100)
Anti-double-stranded DNA antibodies	Negative
Anticardiolipin IgG / IgM antibodies	Negative
Anti- β 2-glycoprotein I IgG / IgM antibodies	Negative
Rheumatoid factor	< 20 IU/mL (negative)
Complement fractions C3 / C4 (g/L)	1.48 / 0.330 (normal)
Lupus anticoagulant (dRVVT screen / confirm / mixing study)	Ratio 1.68 / 1.26 / corrected: no true lupus anticoagulant
Serum protein electrophoresis	Inflammatory profile, hypoalbuminaemia, hypogammaglobulinaemia

dRVVT: dilute Russell viper venom time; EBNA: Epstein-Barr nuclear antigen; HIV: human immunodeficiency virus; VCA: viral capsid antigen.

Because the patient was haemodynamically stable, had no peritoneal signs and showed no evidence of active bleeding on CT, a collegial decision involving the emergency, visceral surgery, intensive care and haematology teams was taken to proceed with non-operative management. He was admitted to a multipurpose intensive care unit for continuous haemodynamic monitoring, strict bed rest and multimodal analgesia. The thrombocytopenia was managed jointly with the haematology team, and haemoglobin concentration and platelet count were measured repeatedly. Surgical management was held in reserve, with an operating theatre immediately available in case of haemodynamic deterioration or of a significant fall in haemoglobin.

The course was uneventful. The patient remained haemodynamically stable throughout admission, with no clinical sign of active bleeding and no significant fall in haemoglobin on serial testing. A follow-up contrast-enhanced abdominal CT performed on day 9, compared with the initial examination, showed a spleen that was relatively stable in size containing scattered non-enhancing hypodense areas consistent with ischaemic change, a stable intraparenchymal haematoma of the upper pole, a decrease in the volume of the pelvic

haemoperitoneum with a few residual layers of blood persisting in the pouch of Douglas, and no sign of active bleeding. A 10 mm precaval lymph node was newly described, together with multiple coeliomesenteric and lumbo-aortic nodes of non-suspicious appearance. In the absence of any fall in haemoglobin, and given the clinical, haemodynamic and radiological stability, splenectomy was not indicated.

The patient was discharged on day 10, conscious, in good general condition, afebrile, haemodynamically stable, with no clinical sign of active bleeding, a stable haemoglobin concentration and a corrected platelet count. He was advised to rest and to avoid physical exertion and activities carrying a risk of abdominal trauma, to undergo clinical and laboratory follow-up with serial full blood counts, to continue specialist haematological follow-up for the thrombocytopenia and its aetiology, to attend a visceral surgery outpatient review, and to seek emergency care immediately in the event of sudden or increasing abdominal pain, faintness, pallor, tachycardia or any other sign suggestive of recurrent bleeding.

DISCUSSION

The principal lesson of this observation is that atraumatic splenic rupture is rarely an isolated accident. In the systematic review by Renzulli *et al.*, the spleen was pathological in 93.0% of the 845 reported patients, and a strictly normal spleen accounted for only 7.0% of cases [1]. Our patient, who had a spleen measuring 17 cm, therefore belongs to the large majority of cases in which the rupture reveals an underlying condition. Splenomegaly is the main mechanical substrate of this phenomenon: an enlarged spleen has a capsule that is thin relative to the parenchymal volume it contains, extends beyond the protection of the rib cage and is subjected to greater parenchymal tension, so that everyday strain which usually goes unnoticed coughing, sneezing, straining at stool or simply turning over in bed may be sufficient to tear it.

The diagnostic pitfall is that the absence of trauma tends to steer clinical reasoning away from a haemorrhagic cause. Pain in the left hypochondrium, sometimes radiating to the left shoulder through irritation of the diaphragm (Kehr sign), is readily attributed to a gastric, colonic, renal or musculoskeletal origin, particularly in a young patient with no comorbidity who arrives with normal vital signs. In our patient, blood pressure and heart rate were strictly normal on admission, and only the disproportionate intensity of the pain prompted immediate CT. Contrast-enhanced CT is the reference investigation: it confirms the haemoperitoneum, characterises the splenic lesions, detects any active extravasation and guides the therapeutic strategy [3]. Applied to our patient, lacerations more than 3 cm deep combined with an intraparenchymal haematoma correspond to at least grade III of the American Association for the Surgery of Trauma organ injury scale, a grade at which non-operative management nevertheless remains the rule in haemodynamically stable patients [3,7].

The profound thrombocytopenia observed here, at 5,000/mm³ on admission, deserves a double reading. It is plausibly a contributing factor to the haemorrhage, by amplifying and prolonging bleeding from lacerations that might otherwise have remained contained; and it is, above all, a pointer to the underlying disease. Its association with splenomegaly, major leucocytosis, C-reactive protein of 295 mg/L and hyperferritinaemia above 4,900 ng/mL indicates an active systemic process, which the rupture merely brought to light.

The aetiological work-up was conducted in that spirit and was informative mainly through what it excluded. Epstein-Barr virus infection is the classical cause of atraumatic splenic rupture in young adults: in a systematic review of 186 reported ruptures associated with infectious mononucleosis, approximately 80% occurred within three weeks of the onset of symptoms, only 9.1% were preceded by any trauma, splenectomy

was performed in 84% of severe cases, and mortality reached 4.8% [4]. In our patient, the serological profile positive anti-EBNA and anti-VCA IgG antibodies with negative anti-VCA IgM antibodies indicated past infection and formally ruled out this hypothesis, as did negative cytomegalovirus IgM, sterile blood cultures and negative leishmaniasis serology. Autoimmune screening was likewise negative, and the isolated abnormality of the dilute Russell viper venom time corrected on mixing, excluding a true antiphospholipid syndrome.

The combination of splenomegaly, profound thrombocytopenia and marked hyperferritinaemia may legitimately raise the possibility of a reactive haemophagocytic syndrome, which the HScore was developed to quantify [5]. Several elements argue against this diagnosis in our patient: the absence of anaemia and of leucopenia, cytopenia being limited to the platelet lineage, and a fibrinogen concentration that was raised at 5.70 g/L rather than reduced. A haematological cause in the first instance a lymphoproliferative disorder therefore remains the leading hypothesis, supported by the lymph nodes described on the follow-up CT and by the hypogammaglobulinaemia found on electrophoresis. This is precisely why the rupture must be regarded as a symptom, and why haematological follow-up was maintained after discharge.

The therapeutic decision was the second key point of this observation. In blunt splenic trauma, the World Society of Emergency Surgery recommends that an initial attempt at non-operative management be made in every haemodynamically stable patient without other abdominal injuries requiring surgery, irrespective of the injury grade, provided that intensive monitoring, immediate access to angioembolisation, an immediately available operating theatre and blood products are guaranteed [3]. By analogy, and in the absence of a dedicated guideline for atraumatic rupture, these criteria guided our decision. Preserving the spleen has a lasting benefit, since splenectomy exposes the patient to lifelong risk of overwhelming post-splenectomy infection an argument that carries particular weight in a 28-year-old patient.

It must nonetheless be emphasised that the transposition is not automatic. In the atraumatic setting, conservative measures were used in only 14.7% of the patients reviewed by Renzulli *et al.*, while total splenectomy was performed in 84.1% [1]. Two features specific to atraumatic rupture explain this caution: the parenchyma is diseased and therefore fragile, so that the rupture may extend spontaneously, and the causal disease persists after the bleeding has stopped. The profound thrombocytopenia made this choice still more demanding, and it was accepted only because the platelet count could be corrected rapidly with the haematology team, because monitoring took place in an intensive care unit and because surgery remained immediately available.

The follow-up strategy is the point on which our management departs most clearly from published recommendations. The 2022 World Society of Emergency Surgery consensus document suggests contrast-enhanced ultrasonography or CT 48 to 72 hours after admission in moderate and severe splenic injuries managed non-operatively, and considers mobilisation safe once three successive haemoglobin measurements taken eight hours apart are within 10% of one another [6]. In our patient the follow-up CT was performed later, on day 9, clinical and laboratory monitoring having been judged sufficient in the interval. The favourable outcome does not validate this delay, and earlier imaging would have been preferable in a patient who combined deep splenic lacerations with a major platelet abnormality.

This work has several limitations. It reports a single case and therefore permits no generalisation. The aetiology of the splenomegaly and of the thrombocytopenia remained undetermined at the time of discharge, and the follow-up available is short. Finally, preserving the spleen, which was the therapeutic objective, also deprived us of the histological examination that a splenectomy specimen would have provided, so that the diagnosis rests entirely on the continuation of the haematological work-up. The value of this observation lies in the reproducible nature of the sequence described: unexplained pain of the left hypochondrium, immediate CT, a haemodynamically stable patient, a decision not to operate, and a spleen preserved despite a platelet count of 5,000/mm³.

CONCLUSIONS

Atraumatic splenic rupture is rare, potentially lethal and, above all, rarely isolated: in more than nine out of ten cases it reveals an underlying splenic or systemic disease. It should be considered in any patient presenting with spontaneous pain of the left hypochondrium, and contrast-enhanced CT should be obtained without waiting for haemodynamic deterioration, since normal vital signs on admission in no way exclude a substantial haemoperitoneum.

In a haemodynamically stable patient with no peritoneal signs and no active bleeding on imaging, non-operative management can preserve the spleen even in the presence of profound thrombocytopenia, provided that monitoring is intensive, that haemostasis is corrected jointly with the haematology team and that surgery remains immediately available. Sparing the spleen, however, does not spare the diagnosis: the rupture must be treated as the first sign of a disease that is still to be identified, and the aetiological work-up must be pursued well beyond discharge.

Additional Information

Disclosures

Human subjects: Informed consent was obtained from the patient for the publication of this case report and of the accompanying images. All patient-identifying data have been removed.

Conflicts of interest: In compliance with the ICMJE uniform disclosure form, all authors declare the following: Payment/services info: All authors have declared that no financial support was received from any organisation for the submitted work. Financial relationships: All authors have declared that they have no financial relationships at present or within the previous three years with any organisations that might have an interest in the submitted work. Other relationships: All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

REFERENCES

1. Renzulli P, Hostettler A, Schoepfer AM, Gloor B, Candinas D: Systematic review of atraumatic splenic rupture. *Br J Surg*. 2009, 96:1114-1121. <https://doi.org/10.1002/bjs.6737>
2. Aubrey-Bassler FK, Sowers N: 613 cases of splenic rupture without risk factors or previously diagnosed disease: a systematic review. *BMC Emerg Med*. 2012, 12:11. <https://doi.org/10.1186/1471-227X-12-11>
3. Coccolini F, Montori G, Catena F, *et al.*, Splenic trauma: WSES classification and guidelines for adult and pediatric patients. *World J Emerg Surg*. 2017, 12:40. <https://doi.org/10.1186/s13017-017-0151-4>
4. Toti JMA, Gatti B, Hunjan I, *et al.*, Splenic rupture or infarction associated with Epstein-Barr virus infectious mononucleosis: a systematic literature review. *Swiss Med Wkly*. 2023, 153:40081. <https://doi.org/10.57187/smw.2023.40081>
5. Fardet L, Galicier L, Lambotte O, *et al.*, Development and validation of the HScore, a score for the diagnosis of reactive hemophagocytic syndrome. *Arthritis Rheumatol*. 2014, 66:2613-2620. <https://doi.org/10.1002/art.38690>
6. Podda M, De Simone B, Ceresoli M, *et al.*, Follow-up strategies for patients with splenic trauma managed non-operatively: the 2022 World Society of Emergency Surgery consensus document. *World J Emerg Surg*. 2022, 17:52. <https://doi.org/10.1186/s13017-022-00457-5>
7. Kozar RA, Crandall M, Shanmuganathan K, *et al.*, Organ injury scaling 2018 update: spleen, liver, and kidney. *J Trauma Acute Care Surg*. 2018, 85:1119-1122. <https://doi.org/10.1097/TA.0000000000002058>