

Original Research Article

SPLENECTOMY AT A TERTIARY CARE TEACHING HOSPITAL: A CASE SERIES OF TEN PATIENTS

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ABSTRACT

Background: Splenectomy remains an essential surgical procedure despite advances in spleen-preserving strategies and non-operative management. It continues to play a crucial role in the treatment of haemodynamically unstable splenic trauma, refractory splenic infections and selected haematological disorders associated with hypersplenism. This case series describes the diverse indications and operative approaches of splenectomy performed at a tertiary care teaching hospital.

Case Report: This case series includes ten consecutive patients who underwent splenectomy for varied traumatic and non-traumatic splenic pathologies. Splenectomy was performed for a broad spectrum of traumatic and non-traumatic splenic disorders. Emergency splenectomy was indicated in patients with haemodynamically unstable blunt splenic trauma associated with significant hemoperitoneum. Elective splenectomy was undertaken for symptomatic hypersplenism secondary to β -thalassaemia major, hereditary spherocytosis, and sickle cell disease, as well as for splenic abscesses refractory to conservative management, including one post-traumatic abscess. Contrast-enhanced computed tomography was the principal diagnostic modality for traumatic and infective lesions. Open splenectomy was performed in nine patients, whereas laparoscopic splenectomy was successfully completed in one patient with a localized post-traumatic splenic abscess. Appropriate post-splenectomy vaccination and counselling were provided according to institutional protocol.

Conclusion: Splenectomy remains a safe and effective treatment for carefully selected patients with both traumatic and non-traumatic indications. Individualized surgical decision-making based on clinical presentation, imaging findings, haemodynamic status and underlying pathology enables favourable short-term outcomes. This series highlights the continued relevance of splenectomy across a broad spectrum of splenic diseases, particularly in resource-constrained settings.

Keywords: AAST Splenic Injury, Hemolytic Anemia, Hypersplenism, Splenectomy, Splenic Trauma

INTRODUCTION

The spleen is the largest secondary lymphoid organ in the human body and performs essential immunological, hematological, and reticuloendothelial functions, including filtration of senescent erythrocytes, clearance of encapsulated microorganisms, immunoglobulin production, and

regulation of both innate and adaptive immune responses.^[1] Owing to its highly vascular architecture and central role in erythrocyte sequestration, the spleen is susceptible to a wide spectrum of pathological conditions ranging from traumatic injury and infective processes to congenital and acquired hematological disorders. Although advances in critical care, diagnostic imaging, and

interventional radiology have substantially increased the success of spleen-preserving strategies, splenectomy remains an indispensable surgical procedure for selected patients in whom conservative management is either inappropriate or unsuccessful. Worldwide, blunt abdominal trauma remains the leading indication for emergency splenectomy, whereas elective splenectomy is commonly performed for disorders such as β -thalassemia major, hereditary spherocytosis, sickle cell disease, immune thrombocytopenia, and selected splenic infections.^[2]

The management of splenic diseases has undergone a significant paradigm shift over the past three decades. The widespread use of contrast-enhanced computed tomography, implementation of standardized injury grading systems, and increasing availability of angiographic embolization have resulted in a marked increase in non-operative management of blunt splenic trauma, with reported splenic salvage rates exceeding 80% in haemodynamically stable patients.^[3] Nevertheless, splenectomy continues to represent the definitive treatment for patients presenting with haemodynamic instability, extensive splenic parenchymal disruption, failed non-operative management, persistent haemorrhage, or associated intra-abdominal injuries requiring laparotomy. Similarly, elective splenectomy remains an established therapeutic intervention in patients with symptomatic hypersplenism, transfusion-dependent haemoglobinopathies, hereditary red blood cell membrane disorders, and splenic abscesses refractory to medical therapy. Consequently, careful patient selection and individualized clinical decision-making remain fundamental to achieving optimal surgical outcomes.

The indications for splenectomy in routine clinical practice are highly varied and include both emergency and elective conditions. High-grade splenic trauma remains a common cause of emergency abdominal surgery. This is especially true in developing countries, where road traffic accidents and blunt abdominal injuries are major contributors to trauma-related morbidity and mortality.^[4]

Inherited haemolytic disorders form another important group. Conditions such as β -thalassemia major, hereditary spherocytosis, and sickle cell disease often lead to progressive splenomegaly and hypersplenism due to chronic sequestration of red blood cells and increased reticuloendothelial activity. Many of these patients eventually need splenectomy to reduce transfusion requirements and improve hematological parameters. Splenic abscess, although uncommon, is another important indication for splenectomy because delayed diagnosis and inadequate treatment are associated with significant morbidity.

Despite being a well-established procedure, splenectomy is associated with important perioperative and long-term considerations. Immediate surgical complications include haemorrhage, pancreatic injury, subphrenic collections, and injury to adjacent viscera, whereas

long-term complications include portal venous thrombosis, thromboembolic events, and overwhelming post-splenectomy infection (OPSI), a potentially fatal condition resulting from impaired immune function against encapsulated bacteria.^[5] These risks have led to the development of standardized perioperative protocols incorporating preoperative vaccination, antibiotic prophylaxis, meticulous surgical technique, and structured postoperative follow-up. Furthermore, laparoscopic splenectomy has become the preferred approach for most elective benign splenic disorders because of its association with reduced postoperative pain, shorter hospitalization, lower wound morbidity, and earlier functional recovery. However, open splenectomy continues to be the procedure of choice in patients with massive splenomegaly, complex splenic pathology, or haemodynamically unstable trauma requiring rapid haemorrhage control.

Although numerous studies have evaluated splenectomy for individual disease entities, relatively few reports have comprehensively described the diverse indications, operative approaches, and early clinical outcomes of splenectomy across both traumatic and non-traumatic pathologies within a single tertiary care institution, particularly in resource-constrained healthcare settings. Existing literature is largely confined to specific disease populations, thereby limiting the understanding of the broad clinical spectrum encountered in general surgical practice. Moreover, institutional experiences describing the management of uncommon conditions such as post-traumatic splenic abscess alongside traumatic injuries and inherited haemolytic disorders remain limited. Therefore, the present case series was undertaken to describe the clinical presentation, radiological characteristics, operative management, and short-term postoperative outcomes of ten consecutive patients undergoing splenectomy for varied indications at a tertiary care teaching hospital.

CASE SERIES

Case 1: Laparoscopic splenectomy for a splenic abscess developing after blunt abdominal trauma

A 51-year-old woman presented with a two-day history of abdominal pain that was insidious in onset and without identifiable aggravating or relieving factors. There was a history of accidental fall from bed approximately eight days earlier. There was no fever, vomiting, jaundice, or alteration in bowel habits. Her past history was significant only for bilateral tubal ligation, and she had no known comorbidities. Her obstetric history was P2L2, and she reported amenorrhoea for the preceding two months.

On examination she was conscious and oriented, afebrile, and haemodynamically stable (pulse 78/min, blood pressure 110/70 mmHg, SpO₂ 98% on room air, random blood sugar 112 mg/dL). There was no pallor or icterus. The abdomen was soft and non-

distended, with no tenderness, guarding, or organomegaly, and normal bowel sounds; cardiovascular and respiratory examinations were unremarkable.

Ultrasonography of the abdomen demonstrated a normal-sized spleen (9.5 cm) containing an irregular, heterogeneously hypoechoic collection measuring approximately $4.7 \times 5.4 \times 4.4$ cm (estimated volume 50–60 cc) involving the upper pole, with no free intraperitoneal fluid. Contrast-enhanced computed tomography of the abdomen was performed for further characterisation and demonstrated a rounded, well-defined, hypodense collection with a peripherally enhancing wall at the superior pole of the spleen, without diaphragmatic or peritoneal breach and without free intraperitoneal fluid. The liver, gallbladder, kidneys, and bowel loops were unremarkable [Figure 1].

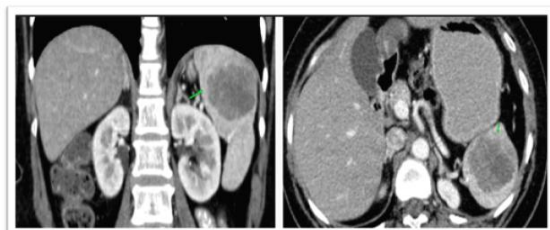


Figure 1: Contrast-enhanced CT of the abdomen (coronal and axial images) demonstrating a rounded, well-defined, hypodense collection with a peripherally enhancing wall at the superior pole of the spleen (arrows), consistent with a splenic abscess. No free intraperitoneal fluid was present.

In the context of the antecedent blunt trauma, the findings were consistent with a splenic abscess, likely arising within an infected post-traumatic collection. The patient was initially managed conservatively with intravenous ceftriaxone, metronidazole, a proton-pump inhibitor, analgesics, and fluids. As there was no satisfactory clinical improvement after five days, surgical intervention was planned following informed written consent.

Laparoscopic splenectomy was performed under general anaesthesia (ASA grade I) in the right lateral decubitus position. Pneumoperitoneum was established through a 10-mm subumbilical port, with an additional epigastric working port. Exploration confirmed a lesion of the superior pole. The gastrosplenic ligament was divided to enter the lesser sac; after hilar dissection, the splenic artery was secured with Hem-o-lok clips and divided, followed by ligation and division of the splenic vein. After medial-to-lateral mobilisation, the specimen was retrieved through a Pfannenstiel incision and sent for histopathology, and a left subphrenic drain was placed.

Recovery was uneventful. The patient remained afebrile with progressively decreasing drain output, tolerated oral feeds from the first postoperative day, and was discharged on the fourth postoperative day on oral antibiotics.

Case 2: Emergency splenectomy for AAST Grade III splenic injury (blunt trauma)

A 48-year-old man was brought to the emergency department following blunt trauma to the head and abdomen. On arrival he was conscious but in distress and was shifted to the surgical intensive care unit. Primary survey revealed haemodynamic compromise with tachycardia and signs of peritoneal irritation in the left upper quadrant. Abdominal examination showed guarding and tenderness, predominantly in the left hypochondriac and lumbar regions, with clinical suspicion of intra-abdominal injury. There was no external penetrating wound.

Investigations revealed haemoglobin 11.1 g/dL with leucocytosis (total leucocyte count $14.54 \times 10^3/\mu\text{L}$) and a platelet count of $349.70 \times 10^3/\mu\text{L}$. Liver function tests showed a mildly elevated SGOT (42 IU/L) with total bilirubin 1.09 mg/dL, total protein 5.02 g/dL and albumin 3.01 g/dL. Renal function was preserved (creatinine 1.3 mg/dL, urea 28 mg/dL). Resuscitation with intravenous analgesia, calcium gluconate, and urgent blood transfusion was initiated. Contrast-enhanced CT of the abdomen and pelvis demonstrated a large, irregular parenchymal laceration involving the upper and mid poles of the spleen measuring approximately 6.0×5.0 cm, extending deep into the parenchyma with surrounding intraparenchymal haematoma and disruption of the splenic capsule at the site of injury. Moderate perisplenic haematoma with adjacent fat stranding was noted. Moderate hemoperitoneum was present, with hyperdense free fluid in the perisplenic region, left paracolic gutter, perihepatic space, and dependent pelvis. The splenic vascular pedicle was intact, with no active contrast extravasation or pseudoaneurysm, and no additional solid-organ injury was identified. The findings were consistent with an AAST Grade III splenic injury [Figure 2A].

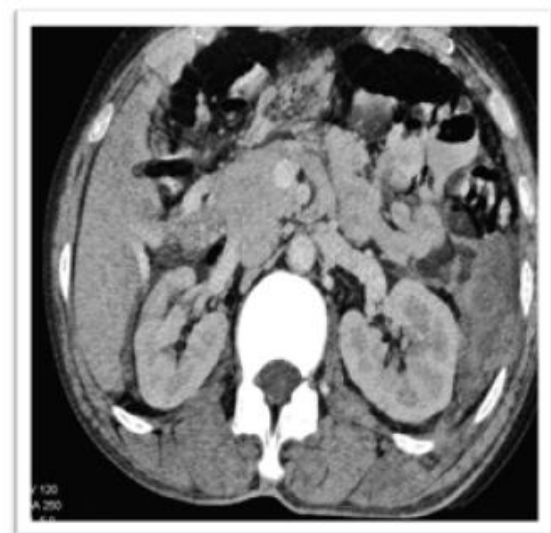


Figure 2 (A): Contrast-enhanced CT of the abdomen showing an AAST Grade III splenic laceration (approximately 6.0×5.0 cm) involving the upper and mid poles, with perisplenic haematoma and moderate hemoperitoneum.

In view of haemodynamic instability and a Grade III injury with significant hemoperitoneum, emergency exploratory laparotomy was undertaken. Intraoperatively, a severely lacerated spleen with gross hemoperitoneum was confirmed, and splenectomy was performed with adequate haemostasis. The excised spleen showed extensive parenchymal disruption corresponding to the radiological findings. The postoperative period was uneventful, and the patient was discharged in satisfactory condition [Figure 2 B].



Figure 2 (B): Intraoperative photograph of the excised spleen demonstrating the Grade III laceration with associated haematoma.

Case 3: Elective open splenectomy for hypersplenism in beta-thalassaemia major

A 7-year-old girl, a known case of transfusion-dependent beta-thalassaemia major, was admitted electively for symptomatic massive splenomegaly. She had been on regular follow-up and presented with increasing pallor, fatigability, and progressive abdominal distension. The indication for surgery was symptomatic Grade 3 splenomegaly with transfusion-dependent hypersplenism. On examination she appeared pale and malnourished, with massive splenomegaly extending to the umbilicus and a palpably enlarged liver; there was no lymphadenopathy.

Preoperative investigations revealed severe anaemia (haemoglobin 5.0 g/dL) and thrombocytopenia (platelet count $56 \times 10^3/\mu\text{L}$), consistent with hypersplenism, with a total leucocyte count of $5.0 \times 10^3/\mu\text{L}$. Red cell indices showed an elevated RDW-CV (19.6%) and RDW-SD (61.7 fL), reflecting marked anisocytosis, with a Mentzer index of 15. Ultrasonography confirmed hepatosplenomegaly, the spleen measuring 16.5 cm craniocaudally with homogeneous echotexture and normal Doppler flow; the liver measured 14.8 cm, with minimal free fluid in both iliac fossae.

The patient underwent elective open splenectomy under general anaesthesia. Through a midline incision, the peritoneum was opened and a massively enlarged spleen visualised. The lesser sac was entered through a window in the gastrosplenic ligament, and the gastrosplenic and splenorenal ligaments were divided using a harmonic energy device. The splenic artery was identified, doubly ligated with silk and haemoclips, and divided, followed by ligation and division of the splenic vein. After division of the splenophrenic ligaments the spleen was delivered. Accessory splenic tissue (splenunculi) identified along the gastrosplenic ligament was excised. Haemostasis was confirmed, and the abdomen was closed in layers using absorbable sutures for the fascia and a subcuticular skin closure; the specimen was sent for histopathology [Figure 3].



Figure 3: Intraoperative photograph of the excised, massively enlarged spleen.

Postoperatively, the patient was shifted to the surgical intensive care unit, where she remained haemodynamically stable (blood pressure 120/80 mmHg, pulse 80/min, SpO₂ 98% on room air), conscious and oriented. She made an uneventful recovery and was discharged in satisfactory condition, with advice for post-splenectomy immunisation.

Case 4: Emergency splenectomy for AAST Grade V (shattered) splenic injury following a road traffic accident

A 35-year-old woman was brought to the casualty department following a road traffic accident, having sustained blunt abdominal trauma after a fall from a motorcycle. She was shifted to the surgical intensive care unit. On primary survey she was haemodynamically compromised with tachycardia. Abdominal examination revealed generalised tenderness with guarding and rigidity, most marked

in the left upper quadrant, raising strong suspicion of intra-abdominal haemorrhage.

Emergency investigations revealed haemoglobin 8.7 g/dL, haematocrit 28%, RBC count $2.43 \times 10^6/\mu\text{L}$, and platelets $143 \times 10^3/\mu\text{L}$, with leucocytosis (WBC $10.98 \times 10^3/\mu\text{L}$, 85% granulocytes). A repeat sample after resuscitation showed haemoglobin 9.5 g/dL. Liver function showed a mildly elevated SGOT (77 IU/L) consistent with hepatic contusion, with total bilirubin 0.44 mg/dL, albumin 3.52 g/dL, and total protein 6.12 g/dL. Renal function was normal. Mild hyponatraemia (131.7 mmol/L) and hypocalcaemia (ionised calcium 1.01 mmol/L) were noted.

Contrast-enhanced CT of the abdomen and pelvis revealed a mildly bulky spleen (11.4 cm) with multiple irregular, non-enhancing parenchymal lacerations extending from the capsular surface to the hilum, involving approximately 50–60% of the parenchyma (maximum laceration length 4 cm) with involvement of segmental splenic vessels. No active arterial extravasation or pseudoaneurysm was seen. A significant amount of hyperdense free fluid was present in the perisplenic, splenorenal, perihepatic, hepatorenal, paracolic, and pelvic regions, consistent with moderate-to-significant hemoperitoneum. The liver was normal, and an incidental non-obstructive left renal calculus was noted. The findings were consistent with an AAST Grade V (shattered) splenic injury [Figure 4].

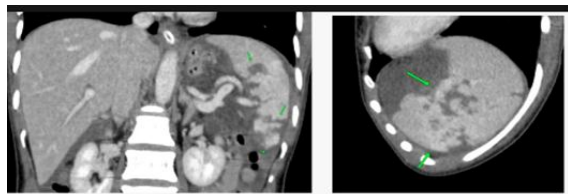


Figure 4: Contrast-enhanced CT of the abdomen showing an AAST Grade V (shattered) spleen with multiple non-enhancing lacerations extending to the hilum, involving 50–60% of the parenchyma, with significant hemoperitoneum.

Given haemodynamic instability and a Grade V injury, emergency exploratory laparotomy and splenectomy were performed. Intraoperatively, a shattered spleen with massive hemoperitoneum was confirmed; haemostasis was secured and the abdomen closed. The patient recovered well and was discharged in satisfactory condition.

Case 5: Elective open splenectomy for hereditary spherocytosis

A 3-year-6-month-old boy presented with pallor, progressive abdominal distension, and recurrent fatigability over several months. He was a diagnosed case of hereditary spherocytosis with associated splenomegaly, and splenectomy was indicated for symptomatic splenomegaly with ongoing haemolytic anaemia. On examination he was pale and icteric with a grossly distended abdomen and massive splenomegaly extending well below the umbilicus; vital signs were stable, with no lymphadenopathy or ascites.

Preoperative investigations revealed haemoglobin 6.9 g/dL with a reticulocyte count of 2.0%, consistent with compensated haemolytic anaemia. The total leucocyte count was $10.37 \times 10^3/\mu\text{L}$, platelets $153 \times 10^3/\mu\text{L}$, and RBC count $2.60 \times 10^6/\mu\text{L}$; serum urea was 75 mg/dL and sodium 139.7 mmol/L. The diagnosis of hereditary spherocytosis was established on the basis of the clinical picture, haematological findings, and a peripheral smear demonstrating spherocytes with polychromasia.

After preoperative optimisation including correction of anaemia, elective open splenectomy was performed under general anaesthesia. Through a left subcostal transverse incision, the peritoneal cavity was entered and an enormously enlarged spleen encountered. The organ was elevated, and the splenocolic and gastrosplenic ligaments were systematically dissected; the short gastric vessels were ligated with silk and divided. The splenic vessels were identified at the hilum, ligated with silk and haemostat clips, and divided, and the pancreatic tail was carefully dissected free from the hilum. The spleen was removed in toto and sent for histopathology [Figure 5].



Figure 5: Intraoperative photograph demonstrating the enormously enlarged spleen in a young child with hereditary spherocytosis prior to excision.

Haemostasis was secured and the wound closed in anatomical layers. Postoperatively the patient was monitored in the surgical ward. Abdominal sonography on the first postoperative day confirmed absence of splenic tissue without any intra-abdominal collection, with mild hepatomegaly (liver span 11 cm). The child made an uneventful recovery and was discharged in stable condition on the fourth postoperative day.

Case 6: Emergency splenectomy for AAST Grade V (shattered) splenic injury following assault

A 61-year-old woman was brought to the emergency department, referred from a rural hospital. She gave an alleged history of blunt assault resulting in injury to the left lower chest and left upper abdomen earlier the same day. Her past history was notable for an

abdominal surgical procedure approximately 30 years previously.

On examination she was pale with tachycardia. Abdominal examination revealed tenderness with guarding and rigidity, predominantly in the left hypochondrium and left flank, with features consistent with hemoperitoneum. Resuscitation was initiated with intravenous fluids, broad-spectrum antibiotics, and analgesics, and a nasogastric tube and urinary catheter were placed. Investigations revealed haemoglobin 8.0 g/dL, total leucocyte count $12.75 \times 10^3/\mu\text{L}$ with neutrophilia (91.3%), and platelets $187 \times 10^3/\mu\text{L}$. Renal function was normal. Liver function showed total bilirubin 0.98 mg/dL, SGOT 34 IU/L, and SGPT 22 IU/L. LDH was markedly elevated (542 IU/L) and CRP was raised, reflecting significant tissue trauma and a systemic inflammatory response. Contrast-enhanced CT of the abdomen and pelvis demonstrated a bulky spleen with multiple linear and patchy non-enhancing lacerations involving 60–70% of the parenchyma and extending to the hilum with segmental vessel involvement. A subcapsular haematoma extended inferiorly along the splenic flexure to the paracolic gutter and pelvis. Significant hyperdense free fluid was present in the perihepatic, hepatorenal, perisplenic, paracolic, and pelvic regions, consistent with moderate hemoperitoneum. No pneumoperitoneum, active extravasation, or other organ injury was identified; incidental bilateral minimal pleural effusion was noted. The findings were reported as an AAST Grade V (shattered) splenic injury [Figure 6A].

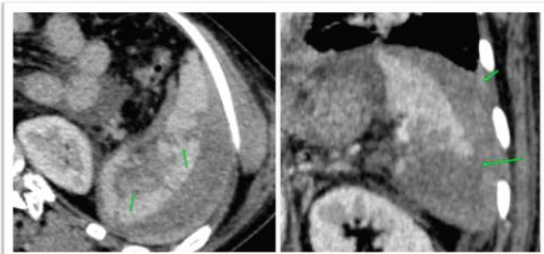


Figure 6A: Contrast-enhanced CT of the abdomen showing an AAST Grade V (shattered) spleen with multiple non-enhancing lacerations extending to the hilum, segmental vessel involvement, and moderate hemoperitoneum.



Figure 6 B: Intraoperative photograph of the excised shattered spleen.

Case 7: Elective open splenectomy for beta-thalassaemia major with transfusion-acquired hepatitis C

A 13-year-old girl, a known case of beta-thalassaemia major, was admitted with progressive Grade 4 splenomegaly and hepatomegaly. She had a longstanding history of transfusion-dependent anaemia with recurrent admissions for packed red cell transfusions since early childhood. Splenectomy was indicated for transfusion-dependent hypersplenism with symptomatic massive (Grade 4) splenomegaly. On presentation she appeared pale and chronically ill, with massive splenomegaly extending well into the lower abdomen and associated hepatomegaly.

Preoperative serological screening revealed reactive hepatitis C virus status, consistent with transfusion-acquired hepatitis C — a recognised complication of repeated transfusions in thalassaemia major; HBsAg was non-reactive. Routine and special haematological investigations were completed, and informed consent was obtained from the guardian after counselling.

Elective open splenectomy was performed under general anaesthesia. Through a midline incision the peritoneum was opened, revealing an enlarged liver and a massively enlarged spleen. The liver was retracted superolaterally. The splenocolic and splenophrenic ligaments were divided using bipolar diathermy and scissors, followed by division of the gastrosplenic ligament with division of the short gastric vessels. The splenorenal ligament was divided, and the splenic vessels were doubly ligated and divided, with careful preservation of the pancreatic tail. A tortuous mass of dilated vascular structures was noted superoposterior to the body of the pancreas, representing dilated splenic vessels. The spleen was delivered and sent for histopathology [Figure 7].



Figure 7: Intraoperative photograph of the excised spleen with an accessory splenunculus identified and excised from the greater omentum. Operative findings included a tortuous mass of dilated splenic vessels superoposterior to the pancreatic body.

A thorough search for accessory splenic tissue identified a splenunculus in the greater omentum, which was excised. Haemostasis was confirmed, the greater omentum was used to fill the splenic fossa, and the abdomen was closed in layers. Postoperative recovery was uneventful, and the patient was discharged in healthy condition, with advice for post-splenectomy immunisation.

Case 8: Elective splenectomy for splenomegaly with splenic abscess

A 49-year-old man presented to the outpatient department with left hypochondriac pain, fever, and generalised weakness, and was admitted for evaluation. On examination he was febrile and mildly pale, with tender splenomegaly. A provisional diagnosis of splenomegaly with splenic abscess was made, and he was started on broad-spectrum antibiotics, analgesics, antiemetics, and supportive care.

Preoperative investigations revealed haemoglobin 12.1 g/dL, total leucocyte count $7.00 \times 10^3/\mu\text{L}$, and platelets $134 \times 10^3/\mu\text{L}$ (mild thrombocytopenia consistent with hypersplenism), with a normocytic picture (MCV 84.4 fL). Serum bilirubin and liver enzymes were within acceptable limits, blood group was A positive, and renal parameters were normal.

Abdominal ultrasonography demonstrated splenomegaly (14 cm) with a $5.0 \times 5.7 \times 5.3$ cm hypoechoic lesion with irregular margins which raised the suspicion of a splenic abscess. Contrast-enhanced CT of the abdomen and pelvis confirmed splenomegaly with a well-defined, rounded, hypodense lesion (approximately 6.0 cm in maximum dimension) with fuzzy margins in the anterior pole, consistent with a large splenic abscess. Additional small hypodense foci were noted within the spleen, and a 2.6×2.8 cm hypo-enhancing collection was seen adjacent to the splenic hilum near the pancreatic tail. Mild perisplenic free fluid, a mildly enlarged liver, and minimal left pleural effusion with subsegmental atelectasis were also noted.



Figure 8: Intraoperative photograph of the excised spleen containing a large abscess.

Given a large splenic abscess unresponsive to conservative management with persistent septic features, elective splenectomy was undertaken.

Intraoperatively, the spleen was enlarged with a large intrasplenic abscess; splenectomy was completed with concurrent drainage of the intra-abdominal purulent collection, and the specimen was sent for histopathology [Figure 8].

Postoperatively, the patient recovered well and was discharged in healthy condition with advice for review.

Case 9: Emergency splenectomy for AAST Grade V (shattered) splenic injury following blunt abdominal trauma.

A 45-year-old man presented to the emergency department following blunt abdominal trauma with suspected splenic rupture. On presentation patient was haemodynamically unstable and was found to have abdominal tenderness and guarding. Simultaneous resuscitation with intravenous fluids, broad-spectrum antibiotics, and analgesics was initiated.

Investigations demonstrated haemorrhagic anaemia (haemoglobin 7.8 g/dL, haematocrit 25.1%), leucocytosis (total leucocyte count $19.37 \times 10^3/\mu\text{L}$ with 92% neutrophils), and thrombocytopenia (platelets $97 \times 10^3/\mu\text{L}$), indicating an acute physiological stress response to major haemorrhage; the picture was normocytic (MCV 95.0 fL). Biochemistry showed creatinine 0.98 mg/dL, urea 23 mg/dL, total bilirubin 1.05 mg/dL, SGOT 24 IU/L, SGPT 17 IU/L, total protein 4.88 g/dL, and albumin 3.19 g/dL.

Contrast-enhanced CT of the abdomen and pelvis demonstrated a bulky spleen with multiple irregular, non-enhancing hypodense lacerations predominantly involving the lower and mid poles, affecting approximately 50–60% of the parenchyma, with extension to the hilum and involvement of segmental splenic vessels. A subcapsular haematoma extended along the mid and lower poles to the splenic flexure. No pseudoaneurysm or active extravasation was seen. A significant amount of hyperdense free fluid was present in peritoneal cavity consistent with hemoperitoneum. The liver was normal, and minimal bilateral pleural effusion with subsegmental atelectasis was noted. The findings confirmed an AAST Grade V (shattered) splenic injury without pneumoperitoneum or additional organ injury [Figure 9].

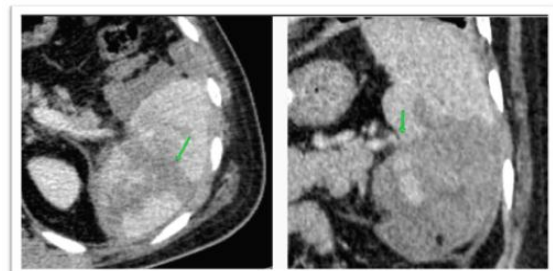


Figure 9: Contrast-enhanced CT of the abdomen showing an AAST Grade V (shattered) spleen with non-enhancing lacerations involving 50–60% of the parenchyma, segmental vessel involvement, and hemoperitoneum.

Given a Grade V injury with haemodynamic instability, the patient was taken up for emergency splenectomy on the day of admission. The postoperative course was uneventful, and the patient was discharged in healthy, ambulatory condition five days after surgery.

Case 10: Elective open splenectomy for sickle cell disease with hypersplenism

An 11-year-old girl who was a known case of sickle cell disease was brought to the paediatric emergency department with acute leg pain and lethargy, and reduced appetite. A provisional diagnosis of sickle cell disease with severe anaemia and suspected vaso-occlusive crisis was made.

Initial investigations revealed severe anaemia (haemoglobin 5.0 g/dL) and a total leucocyte count of $5.46 \times 10^3/\mu\text{L}$ with neutrophilia (63%). There was also marked thrombocytopenia (platelets $85 \times 10^3/\mu\text{L}$) indicating significant hypersplenism. Indirect bilirubin was markedly elevated (7.07 mg/dL) with direct bilirubin 0.43 mg/dL consistent with severe haemolytic jaundice. SGOT was mildly elevated (57 IU/L), likely secondary to haemolysis, and total protein was 7.89 g/dL; renal function and electrolytes were largely normal.

Abdominal ultrasonography demonstrated gross splenomegaly (17.8 cm) with a hyperechoic, heterogeneous parenchyma consistent with sickle cell-related splenic disease. The gallbladder contained multiple calculi (largest 2×1.5 cm) without pericholecystic inflammation, confirming pigment cholelithiasis — a recognised complication of chronic haemolysis.

The patient was managed initially with packed red cell transfusion, hydration, analgesia, and supportive care for the vaso-occlusive crisis. Haemoglobin improved to 5.8 g/dL, though the platelet count declined further to $59 \times 10^3/\mu\text{L}$, reflecting progressive hypersplenism. In view of gross splenomegaly with hypersplenism unresponsive to conservative management, the patient was transferred to the surgical unit and taken up for elective open splenectomy under general anaesthesia; the specimen was sent for histopathology (Figure 10).



Figure 10: Intraoperative photograph of the excised, massively enlarged spleen.

Postoperatively, repeat haematology showed haemoglobin 10.0 g/dL and reactive thrombocytosis (platelets $559 \times 10^3/\mu\text{L}$) — a recognised post-splenectomy phenomenon. The patient was discharged in stable condition, with advice for post-splenectomy vaccination.

DISCUSSION

The present case series shows that splenectomy remains relevant in the management of both traumatic and non-traumatic splenic diseases today. Advances in imaging, intensive care, interventional radiology, and minimally invasive surgery have greatly expanded the scope of non-operative management. Even so, splenectomy remains the definitive treatment for selected patients. These include patients with life-threatening splenic trauma, refractory splenic infections, and haematological disorders with symptomatic hypersplenism. The range of indications in this series included blunt splenic trauma, splenic abscess, haemoglobinopathies (β -thalassemia major, hereditary spherocytosis, and sickle cell disease). This reflects the broad spectrum of splenic pathologies seen at tertiary care centres.

Blunt splenic trauma was one of the leading causes for emergency splenectomy in this study. Current international guidelines recommend non-operative management for haemodynamically stable patients, regardless of injury grade. However, this depends on the availability of intensive monitoring and angioembolization facilities.^[6] Surgery remains mandatory when patients have persistent haemodynamic compromise, ongoing haemorrhage, or fail conservative management. Moore et al,^[7] established the AAST splenic injury grading system, which still forms the basis for radiological classification and surgical decision-making. The good outcomes in our trauma patients support a well-established principle: prompt splenectomy is a life-saving procedure in unstable patients with high-grade splenic injuries. This is particularly true in resource-constrained settings, where access to advanced endovascular interventions may be limited.

Splenic abscess is an uncommon but potentially fatal condition that accounted for two cases in the present series, including one secondary to infected post-traumatic splenic haematoma and another presenting as a primary splenic abscess. The incidence of splenic abscess remains low, but delayed diagnosis is associated with considerable mortality owing to overwhelming sepsis and rupture.^[8] The widespread availability of contrast-enhanced computed tomography has substantially improved diagnostic accuracy and facilitated earlier therapeutic intervention. While image-guided percutaneous drainage has emerged as a treatment option for carefully selected unilocular abscesses, splenectomy continues to represent the treatment of choice in patients with multiloculated collections, multiple

abscesses, associated splenic destruction, or failure of antimicrobial therapy. Chang et al,^[9] reported excellent clinical outcomes following definitive splenectomy in patients unsuitable for conservative management, findings that are consistent with the favourable postoperative recovery observed in both patients included in the present series.

Elective splenectomy was performed in patients with β -thalassemia major, hereditary spherocytosis, and sickle cell disease, all of whom presented with symptomatic massive splenomegaly and hypersplenism. Chronic haemolytic anaemias result in progressive splenic enlargement secondary to increased erythrocyte sequestration and reticuloendothelial hyperactivity, ultimately leading to worsening anaemia, thrombocytopenia, leukopenia, and escalating transfusion requirements. In transfusion-dependent β -thalassemia major, splenectomy has been shown to significantly reduce transfusion burden and improve cytopenias in appropriately selected patients.^[10] Taher et al,^[11] demonstrated that splenectomy decreases annual transfusion requirements and improves quality of life in patients with severe hypersplenism, although careful patient selection is essential because of the increased long-term risk of thrombosis and infection. Similarly, splenectomy remains an accepted therapeutic option in selected patients with sickle cell disease who develop recurrent splenic sequestration, persistent hypersplenism, or symptomatic splenomegaly despite optimal medical management. The marked postoperative improvement in haematological parameters observed in our patients is consistent with previously published evidence supporting the efficacy of splenectomy in carefully selected haemoglobinopathies.

Hereditary spherocytosis remains the most common inherited disorder of the erythrocyte membrane for which splenectomy is indicated. The child included in this case series had severe haemolytic anaemia and massive splenomegaly requiring definitive surgical management after appropriate preoperative optimization. Current international recommendations advocate total or partial splenectomy in patients with moderate-to-severe hereditary spherocytosis after completion of recommended immunization schedules, preferably beyond five years of age whenever clinically feasible.^[12] Bolton-Maggs et al,^[13] demonstrated that splenectomy produces sustained correction of haemolysis, improves haemoglobin concentration, reduces reticulocytosis, and significantly enhances long-term quality of life. Although one of our patients required surgery at a younger age because of severe symptomatic disease, comprehensive perioperative vaccination and postoperative counselling were undertaken in accordance with current recommendations to minimize the lifelong risk of overwhelming post-splenectomy infection (OPSI).

The operative approach in this series was tailored according to the underlying pathology, splenic size, and clinical presentation. In this case series

laparoscopic splenectomy was successfully performed in a patient with localized post-traumatic splenic abscess. On the other hand, open splenectomy was performed in patients with massive splenomegaly and those requiring emergency surgery for traumatic splenic rupture. Targarona et al,^[14] demonstrated that laparoscopic splenectomy offers significant advantages over the open approach in elective benign splenic disorders. However, open splenectomy continues to represent the procedure of choice in haemodynamically unstable trauma patients, patients with giant spleens, or situations requiring rapid vascular control. Importantly, no major perioperative complications or mortality were encountered in the present series, reflecting careful patient selection, meticulous surgical technique, and standardized perioperative care.

An equally important aspect of splenectomy is long-term postoperative management. Loss of splenic immune function predisposes patients to overwhelming post-splenectomy infection, particularly due to encapsulated organisms such as *Streptococcus pneumoniae*, *Haemophilus influenzae* type b, and *Neisseria meningitidis*. Di Sabatino et al,^[15] emphasized that adherence to standardized vaccination protocols and structured follow-up significantly reduce the incidence of serious infections after splenectomy. In the present series, all elective patients were counselled regarding post-splenectomy immunization and long-term preventive measures, while emergency trauma patients were vaccinated during the postoperative period according to institutional protocol.

The principal strength of this case series lies in the fact that it included a broad range of splenic pathologies which were managed within a single tertiary care institution. Although limited by its retrospective design, small sample size, and absence of long-term follow-up, this series contributes valuable institutional data from a resource-limited setting where timely surgical intervention continues to play a central role in the management of complex splenic diseases. Larger prospective multicentre studies are warranted to further evaluate long-term outcomes, postoperative complications, and quality-of-life measures following splenectomy across different pathological conditions.

CONCLUSION

Splenectomy continues to be an indispensable surgical procedure for carefully selected patients with both traumatic and non-traumatic splenic disorders. In this case series, individualized management based on haemodynamic status, underlying pathology, and imaging findings resulted in favourable short-term outcomes without major perioperative complications. Although spleen-preserving strategies remain preferable whenever feasible, timely splenectomy remains life-saving in unstable trauma and provides effective treatment for refractory splenic infections

and symptomatic hypersplenism in selected haematological disorders, particularly in resource-limited settings.

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