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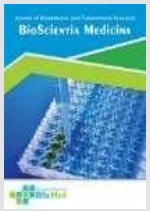

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Emergency Splenectomy for Blunt Splenic Rupture Complicated by Post-Traumatic Acute Kidney Injury and Multiple Organ Dysfunction Syndrome: A Case Report

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A B S T R A C T

Background: Multiple organ dysfunction syndrome is a leading cause of mortality after major blunt trauma. The spleen is the most frequently injured intra-abdominal solid organ, and when rupture occurs, it may precipitate haemorrhagic shock, systemic inflammatory response, and cascading failure of the kidneys, lungs, and coagulation system. Early recognition, rapid haemorrhage control, and coordinated multidisciplinary support are essential to limit progression and improve survival. **Case presentation:** A 30-year-old male motorcyclist presented to the emergency department after a high-velocity road traffic accident without helmet protection. On arrival, he was hypotensive (72/57 mmHg), tachycardic (140 bpm), apathetic with a Glasgow Coma Scale of 12, and had left upper quadrant tenderness with muscular defence. Focused assessment with sonography for trauma demonstrated free intraperitoneal fluid consistent with splenic rupture. Haemoglobin fell from 12.8 to 5.9 g/dL within hours and leukocytes rose to 37.8 $\times 10^3/\mu L$. An emergency splenectomy was performed for haemostasis. During the five-day intensive care course, he developed progressive oliguric acute kidney injury (peak creatinine 5.6 mg/dL, urea 114 mg/dL) requiring intermittent haemodialysis, hypercapnic respiratory failure (pH 6.969; pCO_2 124.6 mmHg) requiring mechanical ventilation, stress hyperglycaemia (441 mg/dL) managed with insulin, and transient thrombocytopenia. Multidisciplinary care by trauma surgery, critical care, nephrology, and internal medicine resulted in haemodynamic stabilisation, recovery of renal function, and successful weaning from ventilation. **Conclusion:** Prompt haemorrhage control through emergency splenectomy, goal-directed resuscitation, early initiation of renal replacement therapy, metabolic support, and coordinated multidisciplinary intervention were decisive in mitigating trauma-induced multiple organ dysfunction syndrome in this patient and are proposed as pillars of management in similar presentations.

1. Introduction

Blunt splenic injury remains the most common visceral injury following blunt abdominal trauma and is an important contributor to the early mortality of road traffic accidents worldwide. Contemporary systematic reviews of high-grade (AAST III–V) blunt splenic trauma demonstrate that splenic angioembolization has broadened the spectrum of non-operative management, yet up to a third of high-grade injuries still require surgical haemostasis when haemodynamic instability persists or when angiographic control fails.[1] Validated grading tools,

such as the World Society of Emergency Surgery classification remain the operational backbone for triage in trauma bays and correlate well with the need for intervention in resource-limited settings.[2] Complementary radiological scoring systems, including the modified Federle score, have been shown to outperform injury-grade alone in predicting the need for splenectomy in patients with isolated blunt splenic trauma, underscoring the importance of combining physiology, imaging, and injury pattern in real-time decision making.[3]

Despite improvements in imaging and endovascular therapy, the failure rate of non-operative management in the haemodynamically marginal patient is non-trivial. Contemporary analyses predict angioembolization failure on the basis of injury grade, contrast blush, and systolic pressure, and identify a clinically important subset that ultimately requires splenectomy even after initial attempts at vessel-preserving control.[4] Real-world data from multicentre cohorts confirm that splenic artery embolization has acceptable technical and clinical success, but outcomes deteriorate rapidly when shock physiology is established on arrival.[5] When presentation is extreme — exemplified by reports of traumatic cardiac arrest secondary to splenic rupture — a damage-control approach with emergency laparotomy and splenectomy is often the only intervention compatible with survival.[6]

Beyond local haemostasis, the patient with high-grade splenic injury is vulnerable to the full spectrum of trauma-induced multisystem disease. Post-traumatic acute kidney injury (AKI) occurs in approximately a quarter of trauma intensive care unit admissions and is strongly associated with increased morbidity and prolonged length of stay. Severe haemorrhagic shock, solid organ injury, massive transfusion, and delayed surgical control converge on tubular ischaemia, oxidative stress, and renal microvascular dysfunction, producing a clinical picture that frequently evolves into multiple organ dysfunction syndrome (MODS). Early recognition of organ dysfunction and a structured multidisciplinary response are accordingly the defining features of modern trauma care.

The present report describes a young adult male who sustained severe blunt splenic injury complicated by profound haemorrhagic shock, evolving AKI requiring intermittent haemodialysis, hypercapnic respiratory failure needing mechanical ventilation, stress hyperglycaemia, and transient thrombocytopenia. The novelty of this case lies in the integrated depiction of the cascade from splenic rupture to MODS in a single patient managed at a regional Indonesian hospital, and in the explicit mapping of clinical decisions — damage-control splenectomy, goal-directed resuscitation, timing of renal replacement therapy, glycaemic control, and protective ventilation — to published evidence. The aim of this report is to document the clinical course and to contextualise it against current literature so that it may serve as a teaching example of how prompt, coordinated multidisciplinary management can convert a near-fatal presentation into functional recovery.

2. Case Presentation

Written informed consent for publication of clinical details and anonymised images was obtained from the patient. A 30-year-old, previously healthy male was brought by bystanders to the emergency department of PKU Muhammadiyah Wonosobo Hospital on 2 October 2025 following a single-vehicle motorcycle crash. The rider had skidded on a rain-slicked highway while not wearing a helmet and briefly lost consciousness at the scene. On regaining awareness, he reported retrograde amnesia for the impact itself, complained of severe left upper abdominal pain, and vomited once with dark, food-stained content. There was no antecedent medical illness, no regular medication, and no previous surgery. The patient's demographic characteristics, mechanism of injury, and initial vital signs are detailed in Table 1.

Table 1. Patient demographic profile, injury mechanism, and initial vital signs on emergency department arrival.

Parameter	Value
Age	30 years
Gender	Male
Mechanism of injury	Motorcycle crash, unhelmeted, skid on wet highway
Time from injury to ED arrival	Approx. 45 minutes
Glasgow Coma Scale (E/V/M)	3 / 4 / 5 = 12 (apathetic)
Blood pressure	72 / 57 mmHg
Heart rate	140 beats/min
Respiratory rate	24 breaths/min
Peripheral oxygen saturation	99% on nasal cannula 2 L/min
Temperature	36.8 °C
Capillary refill time	>2 seconds
Blood group	B Rh-positive
HBsAg/HIV	Non-reactive/Non-reactive

On arrival the patient was apathetic with a Glasgow Coma Scale of 12 (E3V4M5), hypotensive at 72/57 mmHg, tachycardic at 140 beats per minute, tachypnoeic at 24 breaths per minute, afebrile at 36.8°C, and maintaining oxygen saturation of 99% on 2 L/min via nasal cannula. The head was normocephalic with multiple facial abrasions. Conjunctivae were not pale and the sclerae were not icteric. The oral mucosa was dry. The chest was clear with vesicular breath sounds bilaterally and no adventitious sounds. Cardiac examination demonstrated regular heart sounds without murmurs or gallops. Abdominal inspection showed seat-belt type abrasions; palpation revealed muscular defence and focal tenderness over the left hypochondrium. Bowel sounds were diminished. The extremities were cool with capillary refill exceeding two seconds and non-palpable dorsalis pedis pulses bilaterally, consistent with Class III to IV haemorrhagic shock.

Focused assessment with sonography for trauma performed within minutes of arrival demonstrated anechoic collections in the hepatorenal and perisplenic spaces, a positive Morison's pouch sign, and additional free fluid in the pelvis and perirenal space; these findings strongly suggested a ruptured abdominal solid organ, most likely the spleen. A supine chest radiograph was unremarkable for pneumothorax or haemothorax, and a non-contrast head computed tomography showed no intracranial haemorrhage but incidental mild maxillary and ethmoidal sinusitis. Laboratory investigation drawn at 20:00 on the day of admission demonstrated leucocytosis, progressive anaemia on serial sampling, and mild creatinine elevation indicative of early renal impairment in the setting of shock. Marked hyperglycaemia was also noted. The evolution of haematological, renal, and metabolic parameters over the subsequent intensive care stay is summarised in Table 2, and the trend in respiratory and metabolic derangement is captured by the arterial blood gas data shown in Table 3.

Initial resuscitation comprised 1000 mL of intravenous 0.9% saline delivered as a rapid bolus followed by maintenance infusion at 20 drops per minute, together with supplemental oxygen by nasal cannula. Symptomatic measures included intravenous ketorolac, ranitidine, ondansetron, and tranexamic acid, and broad-spectrum coverage was initiated with ceftriaxone 2 g. Because the patient remained hypotensive and demonstrated biochemical and sonographic evidence of ongoing intra-abdominal haemorrhage, damage-control laparotomy was undertaken without delay; a total splenectomy was performed to secure haemostasis. Three units of packed red cells were transfused in the perioperative period. Once stabilised intraoperatively, the patient was transferred to the intensive care unit, intubated and mechanically ventilated, with a right internal jugular triple-lumen catheter and a surgical drain in situ. Damage-control resuscitation followed published

principles of integrated haemorrhage control, crystalloid restraint, and early blood product delivery to avoid the lethal triad of acidosis, hypothermia, and coagulopathy.[7]

Table 2. Serial haematological, renal, electrolyte, and metabolic parameters during the first week after admission.

Parameter (unit)	ER D0	ICU D3	ICU D4	ICU D5	Reference
Leukocytes (×10³/µL)	24.85 → 37.82	18.31	21.54	16.36	4.0–10.0
Haemoglobin (g/dL)	12.8 → 5.9	10.1	9.6	9.5	12.0–16.0
Haematocrit (%)	35.6 → 17.5	28.0	28.4	27.9	40.0–54.0
Platelets (×10³/µL)	209–324	60	32	63	150–450
Urea (mg/dL)	28	114	82	—	10–50
Creatinine (mg/dL)	1.4	5.6	2.7	—	0.1–1.1
Sodium (mEq/L)	135.7	—	142.2	—	135–145
Potassium (mEq/L)	3.65	—	3.34	—	3.5–5.5
Chloride (mEq/L)	105.7	—	109.1	—	96–106
INR	1.16	—	—	1.07	0.8–1.2
aPTT (s)	29.9	—	—	18.3	23–45
Random glucose (mg/dL)	441	—	—	—	80–120

***** Values at the emergency department are shown as sequential readings, where trended (arrow indicates direction of change on serial sampling). —, not measured on the indicated day.

Table 3. Arterial blood-gas evolution during the first 48 hours of intensive care.

Parameter	ICU admission (nadir)	ICU D2 (post-resuscitation)	Reference
pH	6.969	7.142	7.35–7.45
pCO_2 (mmHg)	124.6	78.5	35–45
pO_2 (mmHg)	—	135.2	80–105
Lactate (mmol/L)	—	1.0	0.36–0.75
Urea (mg/dL)	—	114	10–50
Creatinine (mg/dL)	—	5.6	0.1–1.1

† Severe combined respiratory and metabolic acidosis prompted immediate mechanical ventilation and initiation of intermittent haemodialysis.

During the first 72 hours in intensive care, the leukocyte count, haemoglobin, and serum creatinine followed a trajectory that is illustrated graphically in Figure 1. The leukocyte count, which had already reached 37.8 ×10³/µL at presentation, gradually declined toward 16–21 ×10³/µL over the subsequent days, still above normal but trending in the expected direction for a post-splenectomy patient with controlled haemorrhage. Haemoglobin stabilised around 9.5–10.1 g/dL after transfusion, and platelets transiently fell to 32 ×10³/µL on the second post-operative day before recovering. Endocrine stress was evident: random glucose peaked at 441 mg/dL and prompted a sliding-scale insulin regimen using rapid-acting analogue therapy with twice-daily capillary glucose checks. Thyroidal and adrenal responses to shock are recognised in the literature and may contribute to both the catabolic state and to the transient leucocyte and platelet dynamics seen after splenectomy.[8] Experimental modelling of traumatic haemorrhagic shock in animal systems reproduces similar patterns of acidosis, oxidative injury, and inflammatory cell mobilisation, providing a mechanistic context for the observed trajectory.[9]

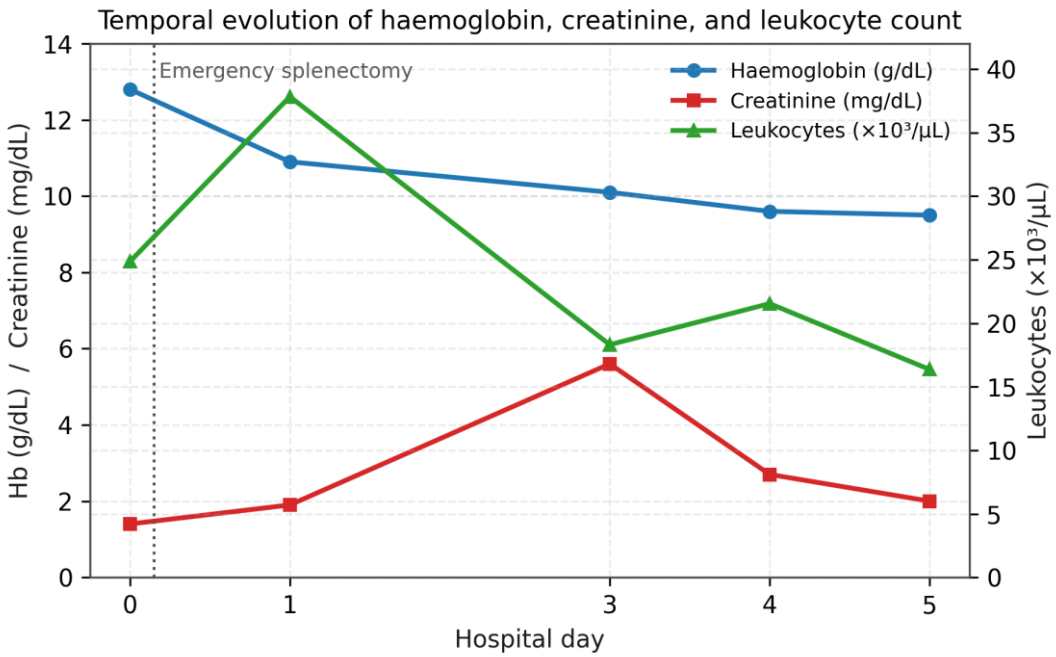

Figure 1. Temporal trajectory of haemoglobin, creatinine, and leukocyte count during the first five hospital days. The dotted vertical line marks the timing of emergency splenectomy; progressive anaemia and rising creatinine triggered transfusion and initiation of renal replacement therapy on ICU day three.

Renal function deteriorated in parallel. The initial creatinine of 1.4 mg/dL had risen to 5.6 mg/dL by intensive care unit day three, with urea climbing from 28 to 114 mg/dL; this satisfied KDIGO stage 3 criteria for AKI. Intermittent haemodialysis was instituted after discussion with nephrology, using a standard three-hour session focused on clearance and cautious ultrafiltration to avoid hypotensive episodes. Advanced haemodynamic monitoring devices, including compact arterial pressure monitors of the type validated in swine REBOA models, are increasingly used to guide resuscitation in such patients and may reduce the frequency of dialysis-related hypotension.[10] Serial arterial blood gas sampling demonstrated severe acidaemia with pH as low as 6.969 and hypercapnia up to 124.6 mmHg on the first ICU day, prompting lung-protective mechanical ventilation. The follow-up chest radiograph performed on the fourth hospital day confirmed correct positioning of the endotracheal tube, chest drain, and central venous catheter, and demonstrated patchy bilateral infiltrates compatible with pulmonary contusion together with bilateral subcutaneous emphysema extending to the supraclavicular regions.

Coagulation indices at presentation were within normal limits (prothrombin time 12.7 s; international normalised ratio 1.16; activated partial thromboplastin time 29.9 s) and remained compensated throughout admission, effectively ruling out disseminated intravascular coagulation. Over the subsequent five days, the patient was weaned from vasopressors, extubated after normalisation of gas exchange, and transitioned from intermittent haemodialysis to conservative renal support as creatinine declined toward 2.7 mg/dL. Nutritional support was resumed enterally. Post-splenectomy immunisation against Streptococcus pneumoniae, Haemophilus influenzae type b, and Neisseria meningitidis was planned for day 14 in accordance with the hospital protocol, and the patient and his family received structured counselling on lifelong

infection risk, fever action plans, and the importance of annual influenza vaccination. On the sixth intensive-care day he was transferred to the surgical ward, and subsequently discharged in stable condition with outpatient follow-up arranged for surgical, internal medicine, and nephrology clinics.

3. Discussion

The clinical tableau of hypotension, tachycardia, altered mental status, peripheral vasoconstriction, and rapidly declining haemoglobin in this patient is the textbook presentation of Class III–IV haemorrhagic shock.[7] Damage-control resuscitation consolidates four principles — permissive hypotension during active bleeding, restrictive crystalloid use, early balanced blood product transfusion, and immediate surgical or endovascular haemorrhage control — and its application in our patient was decisive. Even in resource-constrained settings, a protocolised approach to haemorrhage control improves outcome, as shown by cohort analyses in orthopaedic polytrauma that integrate cardiopulmonary support, haemorrhage control, and damage control as a single bundle.[7] Adjunctive responses of the endocrine system to profound shock — including transient thyroid changes and catecholamine surge — are increasingly recognised and, although they did not require specific pharmacological correction here, they highlight the systemic nature of shock physiology.[8] Preclinical work in haemorrhagic-shock animal models demonstrates that correction of perfusion alone is insufficient if mitochondrial dysfunction and oxidative injury are allowed to propagate, providing a physiological rationale for early mixed colloid/crystalloid and blood product resuscitation.[9] Adjunctive monitoring tools, including compact invasive arterial monitors validated in swine REBOA studies, are increasingly available to provide beat-to-beat assessment of resuscitation adequacy at the bedside.[10]

Intravenous tranexamic acid was administered in the emergency department prior to surgical control, consistent with contemporary recommendations based on the CRASH-2 and military experience. A large analysis of tranexamic acid use in the United States military population found a modest but acceptable rate of associated thromboembolic events, without a clinically meaningful increase in pulmonary embolism when the drug was given within three hours of injury.[11] Military combat trauma cohorts further showed that early tranexamic acid use is associated with reduced 24-hour and 30-day mortality in the most severely injured casualties.[12] Despite this evidence, contemporary audits at civilian trauma centres have demonstrated inconsistent compliance with tranexamic acid protocols, with only a minority of eligible patients receiving the drug within the recommended window.[13] Survey data from trauma surgeons and emergency physicians identify cost perception, fears of thrombosis, and lack of institutional protocols as the principal barriers to uptake, arguing for explicit pathway integration in emergency departments handling polytrauma.[14] In our patient, early tranexamic acid delivered before splenectomy likely contributed to the stable coagulation parameters observed throughout the ICU course and the absence of delayed bleeding complications.

Post-traumatic AKI is driven by the confluence of pre-renal insults (hypovolaemia, hypotension), intra-renal ischaemia-reperfusion injury, rhabdomyolysis, transfusion-associated pigment nephropathy, and systemic inflammation. Our patient demonstrated KDIGO stage 3 AKI within 72 hours, with creatinine rising from 1.4 to 5.6 mg/dL and urea climbing fourfold. Large paediatric and mixed trauma cohorts confirm that transfusion exposure in the context of sepsis and shock is independently associated with AKI, and that volume of transfusion correlates with renal replacement therapy requirement.[15] The temporal profile of renal function in this patient is faithfully reflected in Figure 1 and in Table 2, with creatinine peaking on the second post-operative day and falling after two sessions of intermittent haemodialysis. Clinicians must remain vigilant for delayed vascular complications of abdominal trauma

that may present as recurrent AKI or flank pain weeks after the index injury; reports of renal-segmental pseudoaneurysm detected late after blunt mechanisms remind us that follow-up imaging has value even after functional recovery.[16] The timing of renal replacement therapy remains debated; early initiation in the context of rising urea, hyperkalaemia, and fluid overload is supported in trauma populations, whereas late initiation risks metabolic acidosis and worsening lung function. The cautious ultrafiltration strategy used here — emphasising solute clearance without aggressive volume removal — was chosen to avoid exacerbating post-splenectomy hypotension and to preserve end-organ perfusion.

Non-operative management of blunt splenic injury has become the default in haemodynamically stable patients, supported by a robust evidence base and by refined endovascular techniques.[1] However, haemodynamic instability, transfusion requirement exceeding four units, expanding haemoperitoneum on follow-up imaging, and failure of angiographic control all constitute indications for splenectomy, as articulated by the WSES classification used in our institution.[2] Our patient met several of these criteria simultaneously, making splenectomy the safest option. Minimally invasive alternatives such as laparoscopic selective clipping of splenic artery branches have been described in isolated blunt splenic trauma and offer the potential of spleen preservation in haemodynamically stable patients; their role in unstable physiology remains limited.[17] Preserving splenic function has clear immunological value: retrospective cohorts of patients managed non-operatively demonstrate conservation of specific antibody responses and of memory B-cell populations, underscoring the long-term immunological cost of total splenectomy.[18] Paediatric series further demonstrate that conservative treatment of abdominal solid-organ injuries in stable children can achieve excellent outcomes with organ preservation rates approaching 95%, although the threshold for operative conversion must remain low in any patient exhibiting shock.[19] For the patient reported here, the potential immunological disadvantage of total splenectomy was accepted as the necessary cost of haemorrhage control; a structured vaccination and education programme was initiated to mitigate the consequences.

Acute trauma-induced coagulopathy is distinct from classical disseminated intravascular coagulation. Recent phenotype-guided frameworks argue for the classification of hypocoagulopathic and hyperfibrinolytic trauma phenotypes, each requiring different transfusion strategies and different monitoring tools.[20] Our patient maintained a compensated coagulation profile (INR 1.07–1.16; aPTT 18.3–29.9 s), and no cryoprecipitate or fresh frozen plasma beyond the transfusion of three units of packed red cells was required. The transient platelet nadir of 32 $\times 10^3/\mu L$ on the second post-operative day most likely reflects consumption within the splenectomy bed, dilution, and sequestration in the pulmonary vascular bed after contusion; it resolved without platelet transfusion. Cyclone-related disaster medicine reports provide a useful reminder that trauma patients remain at elevated risk of venous thromboembolism for weeks after injury; chemical thromboprophylaxis should therefore be reintroduced as soon as haemorrhage is controlled and the patient is haemodynamically stable.[21] Emerging basic science research further suggests that inflammatory pathways such as AKT/mTOR/STAT3 signalling contribute to coagulopathy in the septic-trauma interface, pointing toward future adjunctive therapies beyond classical transfusion.[22]

Pulmonary contusion, hypercapnic respiratory failure, and mechanical ventilation

Bilateral pulmonary contusion, evident on the follow-up chest radiograph, contributed materially to the profound acidaemia and hypercapnia documented in the arterial blood gas data in Table 3. Lung-protective ventilation with tidal volumes of 6–8 mL/kg of predicted body weight, moderate positive end-expiratory pressure, and permissive hypercapnia targets were employed, with careful attention to

plateau pressure. Subcutaneous emphysema extending to the supraclavicular region was treated conservatively and resolved without surgical intervention. The potential for pulmonary contusion to evolve into acute respiratory distress syndrome is well established, and conservative fluid strategies, prone positioning when indicated, and early physiotherapy are cornerstones of prevention. Contusion physiology differs from alveolar oedema in several important respects: extravasation of red cells into the interstitium and alveolus is the primary lesion, intrapulmonary shunt peaks 48 to 72 hours after injury, and ventilation-perfusion mismatch is typically refractory to simple increases in inspired oxygen. For that reason, a protective strategy that minimises barotrauma, avoids fluid overload, and permits moderate hypercapnia is generally preferred to aggressive recruitment manoeuvres in the first days of care. In this patient, lung compliance improved in parallel with renal function once ultrafiltration was instituted, underscoring the physiological linkage between extravascular lung water, cardiac output, and successful liberation from mechanical ventilation. Serial arterial blood-gas sampling, as summarised in Table 3, guided the stepwise reduction in minute ventilation and formed the basis for a spontaneous breathing trial on the fifth intensive-care day, after which extubation was successful without need for reintubation.

Multiple organ dysfunction syndrome is best conceptualised as the clinical manifestation of a dysregulated host response to severe injury. In the classical paradigm, a proinflammatory phase dominated by cytokine release, endothelial activation, and neutrophil priming is followed or paralleled by a compensatory anti-inflammatory response that, when exaggerated, produces immunoparalysis and predisposes to secondary infection. In trauma, this response is amplified by tissue factor release from injured parenchyma, by damage-associated molecular patterns such as mitochondrial DNA and high-mobility group box 1 protein, and by the microcirculatory consequences of haemorrhagic shock. The sequential organ failure assessment (SOFA) score formalises organ dysfunction along six axes — respiratory, renal, hepatic, cardiovascular, haematological, and neurological — and allows standardised longitudinal description of the process. In our patient, peak SOFA was driven by a creatinine rise requiring renal replacement therapy (renal subscore), by severe hypercapnia and hypoxia requiring mechanical ventilation (respiratory subscore), by thrombocytopenia (haematological subscore), and by Glasgow Coma Scale reduction during presentation (neurological subscore). Each of these axes improved on serial assessment, indicating that the trajectory had reversed within 72 hours of definitive haemorrhage control. This reinforces a key learning point: control of the inciting surgical lesion is the single most effective intervention for MODS in trauma.

Stress hyperglycaemia with random glucose of 441 mg/dL was managed with sliding-scale insulin and twice-daily capillary glucose testing targeting 140–180 mg/dL, mirroring contemporary ICU practice. Tight glucose control is important because hyperglycaemia impairs neutrophil function, exacerbates oxidative stress, and is an independent predictor of infection and mortality in critically ill patients. The pathogenesis of stress hyperglycaemia in trauma is multifactorial: sympathetic surge drives hepatic glucose production through glycogenolysis and gluconeogenesis; cortisol and growth hormone promote peripheral insulin resistance; and inflammatory cytokines such as tumour necrosis factor alpha and interleukin-6 further downregulate insulin signalling in skeletal muscle and adipose tissue. A glucose value exceeding 400 mg/dL in a patient without known diabetes therefore reflects an extreme neurohormonal response rather than a primary endocrine disorder, but must be corrected promptly because persistent hyperglycaemia is associated with impaired wound healing, increased rates of ventilator-associated pneumonia, and delayed weaning from mechanical ventilation. In our patient, serial capillary glucose measurements returned to the

140–180 mg/dL target within 48 hours of insulin initiation, and this coincided with improvement in acidaemia and in leukocyte trend. Outpatient follow-up with formal oral glucose tolerance testing was planned to exclude underlying type 2 diabetes.

Post-splenectomy immunology is a chronic concern that persists well beyond the acute admission. Loss of the spleen's filtering and antibody-producing functions leaves the patient at lifelong risk of overwhelming post-splenectomy infection (OPSI), particularly from encapsulated organisms such as Streptococcus pneumoniae, Haemophilus influenzae type b, and Neisseria meningitidis. Although rare, OPSI carries a mortality exceeding 50% when established, and its onset can be fulminant, with the patient transitioning from apparent wellbeing to septic shock within hours. International guidelines therefore recommend structured peri-splenectomy vaccination (ideally administered 14 days after surgery where possible), annual influenza immunisation, a low threshold for antibiotic therapy in unexplained fever, and the issuance of a patient-held splenectomy card. Non-operative management data further suggest that even a preserved but injured spleen may not restore full immunological competence, so surveillance must continue regardless of operative strategy.[18] In our patient, a full vaccination schedule and written patient-education material were initiated before discharge, and the family was counselled to seek care within six hours of any febrile illness.

The outcome in this case was a direct product of multidisciplinary integration — trauma surgery, anaesthesiology and critical care, internal medicine, and nephrology — orchestrated under a unified resuscitation plan. This model mirrors evidence-based descriptions of integrated resuscitation and damage-control bundles in orthopaedic trauma, where the presence of structured team roles, shared checklists, and explicit escalation criteria is associated with reduced mortality and fewer omissions of therapy.[7] At the systems level, our experience reinforces the importance of protocol-driven tranexamic acid administration, early blood banking coordination, and clear criteria for theatre activation; gaps in any of these pathways have been shown to delay definitive care and worsen outcomes.[13,14]

Several recent reports allow benchmarking of the present case against the published experience of severe blunt abdominal trauma. Gu and colleagues described a polytrauma patient who sustained recurrent traumatic cardiac arrest due to splenic rupture and was successfully resuscitated after emergency laparotomy and splenectomy in a dedicated trauma resuscitation unit; their case underscores the narrow therapeutic window in unstable splenic injury and supports the doctrine of immediate surgical haemorrhage control that we applied.[6] In contrast to that case, our patient never developed pulseless arrest, allowing the same surgical principles to be delivered with a broader physiological safety margin. Sengupta and colleagues reported atraumatic splenic rupture in a patient in his seventies presenting with acute abdomen and shock, highlighting how splenic pathology can mimic or compound trauma presentations and arguing for a systematic imaging protocol in any patient with unexplained abdominal pain and hypotension.[23] Proa-Arriaga and colleagues reported a grade V AAST intestinal vascular injury after blunt abdominal trauma; although the injured organ differed, the case emphasised that the combination of haemorrhagic shock, high AAST grade, and delayed definitive care drives morbidity in a manner superimposable on our experience.[24] Real-world multicentre Italian data on splenic artery embolization show that endovascular control is feasible in about 80% of appropriately selected patients, but the remaining 20% — those with haemodynamic instability or high-grade injury — still require urgent splenectomy, a group into which our patient fell.[5] Taken together, these published cases situate the present report within an emerging picture: even in centres with advanced endovascular capability, a subset of patients with severe blunt splenic trauma will continue to require damage-control splenectomy followed by critical-care management of AKI and MODS, and the structured

pathway described here can serve as a practical template for those cases. What distinguishes our report is the integrated trajectory from splenic rupture to hypercapnic respiratory failure to dialysis-dependent AKI, managed end-to-end at a single regional Indonesian hospital without recourse to an endovascular suite; the outcome suggests that careful application of basic damage-control principles, evidence-based pharmacological adjuncts, and multidisciplinary cooperation remains transferable across healthcare settings.

Limitations

This report is constrained by its single-patient nature and by the observational framework of routine clinical care. Volumetric quantification of splenic injury by computed tomography was not available because of the haemodynamic instability that mandated immediate surgery, and thromboelastographic monitoring of coagulation was not performed. Long-term immunological follow-up was planned at outpatient review but is not yet available. These limitations, however, do not materially diminish the internal consistency of the clinical findings and the management lessons drawn from them.

The case reinforces several practical lessons. First, haemodynamic physiology should trump imaging grade in the decision to proceed to splenectomy; a transiently responding patient who relapses despite initial resuscitation should be returned to theatre without hesitation. Second, goal-directed resuscitation with restrained crystalloid use and early balanced blood-product delivery is compatible with the resources available in regional hospitals, provided that blood banking and theatre pathways are activated in parallel rather than sequentially. Third, the threshold for initiating renal replacement therapy should be low when oliguric AKI and metabolic acidosis coexist; cautious ultrafiltration strategies that prioritise solute clearance over aggressive volume removal minimise haemodynamic disturbance in the freshly splenectomised patient. Fourth, glycaemic control using rapid-acting insulin analogues with twice-daily capillary glucose testing is a pragmatic approach to stress hyperglycaemia in this setting and should not be postponed until frank diabetic ketoacidosis develops. Fifth, structured post-splenectomy immunisation and patient education are as important as the index operation in determining long-term outcome, and should be documented in the discharge summary with explicit booster schedules. Finally, deliberate debriefing of the resuscitation team after every major trauma case, using a shared checklist of time-to-theatre, tranexamic acid timing, and transfusion ratios, drives continuous improvement of the departmental trauma system and mitigates the common error of omission that drives preventable mortality in low-volume centres.

4. Conclusion

Blunt splenic rupture complicated by haemorrhagic shock, post-traumatic AKI requiring renal replacement therapy, hypercapnic respiratory failure, stress hyperglycaemia, and transient coagulopathy represents a quintessential substrate for MODS. The patient described here recovered functionally because four principles were observed simultaneously: immediate haemorrhage control through damage-control splenectomy; goal-directed resuscitation grounded in restrained crystalloid and balanced blood product use; timely initiation of renal replacement therapy with careful attention to haemodynamic tolerance; and coordinated multidisciplinary intervention across surgery, critical care, internal medicine, and nephrology. Attention to stress hyperglycaemia, trauma-induced coagulopathy, and post-splenectomy immunology further supported recovery. The case demonstrates that contemporary evidence can be translated into effective care even in resource-constrained regional hospitals, provided pathways are protocolised and teams are aligned. We recommend that institutions caring for trauma patients formalise haemorrhage-control bundles, tranexamic acid pathways, dialysis-readiness criteria, and post-splenectomy vaccination

and education as standing protocols, and that subsequent reports systematically capture the long-term immunological and renal follow-up of such patients to refine the evidence base.

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