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SYSTEMIC COMPLICATIONS LEADING TO MODS IN A TRAUMA PATIENT: A CASE-BASED PATHOPHYSIOLOGIC ANALYSIS

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Abstract

The surgical and medical management of organ damage and metabolic complications following high-energy multi-trauma presents a significant clinical challenge. Factors such as severe physiological stress, sympathetic nervous system activation, blood and fluid loss, elevated intracranial pressure, and systemic inflammation can precipitate profound metabolic disturbances, leading to liver and kidney dysfunction, as well as cardiac cell injury, even in the absence of direct trauma to these organs. Despite successful bleeding control and aggressive fluid and electrolyte resuscitation, patients may enter a vicious cycle of metabolic deterioration. Cardiac dysfunction and arrhythmias can develop rapidly, often culminating in sudden death. Early recognition of the pathophysiological mechanisms underlying clinical deterioration and timely intervention are crucial to improving survival. This study analyzes the literature and presents a case-based discussion to explore optimal strategies for managing metabolic complications associated with isolated splenic lacerations resulting from high-energy trauma, with a particular focus on the development of Multiple Organ Dysfunction Syndrome (MODS).

Keywords: *Multi-organ failure, spleen trauma, metabolic complications, MODS, trauma management*.

Introduction

High-energy multi-trauma is a leading cause of morbidity and mortality, especially in young individuals. The spleen, due to its anatomy, is highly prone to injury in blunt abdominal trauma. While non-operative approaches like splenic artery embolization (SAE) are preferred in stable patients, splenectomy remains life-saving in unstable cases. Despite improvements, Multiple Organ Dysfunction Syndrome (MODS) remains a major cause of death in severe trauma.[1]

MODS involves progressive failure of multiple organs following physiological insults. Its pathogenesis includes inflammation, sympathetic overactivation, ischemia-reperfusion, and microvascular dysfunction.[2]

Traumatic brain injury (TBI), common in such traumas, triggers autonomic dysregulation, catecholamine surges, and neuroinflammation, leading to dysfunction in the heart, kidneys, and liver.[3]

This report analyzes a case of splenic trauma with rapid MODS progression, focusing on the pathophysiological mechanisms behind cardiac, renal, and hepatic failure, supported by current literature.[5,6,7]

Case Report

A previously healthy 16-year-old male suffered high-energy multi-trauma following a motor vehicle accident. He was ejected from a car traveling at over 150 km/h without wearing a seatbelt. He was transported to the emergency department (ED).

Upon arrival, the patient was conscious with a Glasgow Coma Scale (GCS) score of 15. Vital signs revealed a blood pressure(BP) of 110/70 mmHg and a pulse rate (HR) fluctuating between 100 and 120 beats per minute. Full body examination identified left temporal and frontal bruising, dermal bulging of the left temporal region without raccoon eyes or periorbital edema, left abdominal abrasions, and a fractured left arm.

Comprehensive imaging studies were performed, including a non-contrast cranial computed tomography (CT) and contrast-enhanced thoracic and abdominal CT scans.

Preoperative Course

On admission, the patient had stable vital signs and GCS of 15. Labs showed normal liver and renal function (ALT: 27 U/L, AST: 44 U/L, Cr: 0.9 mg/dL), Hb 15.4 g/dL, Hct 45.8%, PLT 302,000/μL, and INR 1.84.

Imaging revealed:

- Cranial CT: minimal edema, diffuse hyperdensity (possible DAI), no hemorrhage.
- Thoracic CT: left lower lobe contusion.
- Abdominal CT: grade III (WSES) / IV (AAST) splenic laceration with subcapsular hematoma, no active bleeding.(Figures 1 and 2)

Vitals remained stable initially (BP 110/70 mmHg), but Hb dropped to 11.2 g/dL, prompting transfusion (1 unit fresh frozen plasma (FFP) + 1 erythrocyte suspension (ES)).

En route to Pediatric İntensive Care Unit (PICU), the patient deteriorated: BP 80/50 mmHg, HR 130, GCS 8, and raccoon eyes suggesting intracranial hypertension. Emergency surgery was decided.

Blood gases showed severe acidosis (pH 7.11, HCO_3^- 15.6, lactate 8 mmol/L), Hb 6.3 g/dL, Hct 19%. Resuscitation included fluids, 4 ES, 3 FFP, bicarbonate, calcium, tranexamic acid, and vasopressors. Despite transient stabilization (BP 100/50 mmHg), worsening acidosis persisted (pH 7.02, HCO_3^- 8.3), prompting urgent laparotomy.

Figure 1: Middle pole laceration and intraperitoneal haemorrhage leakage area behind the splen-ic vein

Figure 2: Upper pole laceration and subcapsullar hematoma

Operative Findings

During anesthesia induction, the patient experienced cardiovascular collapse with hypotension (60/40 mmHg), bradycardia (40 bpm), and pulseless VT. CPR was initiated, restoring rhythm after 10 minutes of advanced life support (ALS).

Following stabilization, a midline laparotomy revealed massive intra-abdominal hemorrhage and compartment syndrome. Bleeding was controlled, and a complete splenectomy was performed due to upper pole venous injury. No other injuries were found.

Intraoperative labs showed severe anemia (Hb 5.6 g/dL), acidosis (pH 7.20, lactate 15.75 mmol/L). Two more units of ES and FFP were given. Post-op labs revealed Hb 9.4 g/dL, Hct 30%, platelets 103,000/μL, profound acidosis (pH 6.83, HCO_3^- 5.8), and lactate 18.52. Bilateral facial edema and pulsatile nasal bleeding required nasal tamponade.

Postoperative Course
Following surgery, the patient was admitted to the ICU with BP 100/50 mmHg, HR 90 bpm, requiring triple inotropic support despite no active bleeding. Labs revealed:

- Severe liver injury (AST >913 U/L, ALT >942 U/L)
- Renal dysfunction (Cr 2.99 mg/dL)
- Hyperkalemia (K^+ 6.6 mmol/L)
- Hypocalcemia (Ca^{2+} 5.9 mg/dL)
- Metabolic acidosis (pH 6.84, lactate 16.4 mmol/L) and oliguria, consistent with early AKI

As illustrated in Table 1, multi-organ failure progressed despite intensive interventions, including fluid resuscitation, vasopressors, diuretics, and blood transfusions. The patient remained unresponsive, cranial imaging could not be obtained, and cardiac arrest occurred prior to the initiation of dialysis. Resuscitation efforts were unsuccessful, and the patient was pronounced dead.

Table 1: Sequential Timeline of the Patient's Clinical Course from ED Admission to Postopera-tive ICU

Time Point	Vital Signs	Laboratory Findings	Intervention
ED Admission	BP: 110/70 mmHg, HR: 100–120 bpm, GCS: 15	Hb: 15.4 g/dL	Imaging, monitoring
+2 hours	BP: 110/70 mmHg	Hb: 11.2 g/dL	1 unit FFP, 1 unit ES
Pre-PICU	BP: 80/50 mmHg, HR: 130 bpm, GCS: 8	Hb: 6.3 g/dL, pH 7.11, Lactate: 8 mmol/L	Emergency surgery decision
Intraoperative	BP: 60/40 mmHg, Bradycardia 40 bpm, VT	Hb: 5.6 g/dL, pH 7.20, Lactate: 15.75 mmol/L	CPR, splenectomy
Post-op ICU	BP: 100/50 mmHg, HR 90 bpm	AST >913 U/L, Cr: 2.99 mg/dL, Lactate: 16.4 mmol/L	Inotropic support, fluids, diuretics

Discussion

Pathophysiology and Clinical Insights into Trauma-Induced MODS
This case illustrates the rapid progression of multi-organ dysfunction syndrome (MODS) despite aggressive resuscitation and raises several critical questions:

- Why did a previously healthy patient deteriorate so quickly?
- What accounts for the early surge in liver enzymes and the sudden onset of acute kidney injury (AKI) despite only transient hypotension?
- Could an undiagnosed traumatic brain injury (TBI) have triggered the systemic response?

These adverse outcomes, occurring despite effective surgical control of bleeding, underscore significant gaps in our understanding of trauma-induced systemic dysfunction. The rapid deterioration observed in this patient was likely multifactorial, with hemorrhagic shock and an unrecognized severe TBI acting synergistically to precipitate MODS.

Traumatic Brain Injury and Its Role in MODS
Severe traumatic brain injury (TBI) is a major contributor to multi-organ dysfunction syndrome (MODS). Following TBI, sympathetic overactivation and neuroinflammation cause systemic disturbances. Autonomic dysregulation leads to a catecholamine surge, resulting in myocardial stress, renal hypoperfusion, and endothelial dysfunction.[4,6]
One cardiac effect of TBI is contraction band necrosis (CBN), marked by hypercontracted sarcomeres and inflammatory infiltration, often linked to arrhythmias, decreased cardiac output, and sudden death.[3,6,7]
TBI also contributes to acute kidney injury (AKI) via renal vasoconstriction and use of high-chloride hyperosmolar therapies[6], while hypotension and AKI in TBI patients correlate with higher mortality.[10]
Additionally, traumatic coagulopathy—marked by platelet dysfunction and hyperfibrinolysis—further worsens prognosis.[11,12] In this case, raccoon eyes, neurological decline, acidosis, and nasal bleeding despite normal labs suggest undiagnosed severe TBI, contributing to MODS and fatal outcome.

Fulminant Liver Failure without Direct Hepatic Injury
Fulminant liver failure (FLF) without direct liver injury is increasingly observed after trauma. Key mechanisms include ischemia-reperfusion, systemic inflammation (SIRS), and catecholamine-mediated hepatocellular injury.[5]
In high-energy trauma, global hypoperfusion and reperfusion trigger oxidative stress and mitochondrial dysfunction, leading to "ischemic hepatopathy" with markedly elevated AST/ALT, despite normal imaging.[4]
Catecholamine surges during TBI exacerbate liver injury by causing vasoconstriction and microvascular dysfunction. Neuroinflammation increases cytokines like TNF-α and IL-6, amplifying endothelial and hepatocellular damage.[6]
In this case, liver enzymes rose sharply within 24 hours, despite no visible parenchymal injury, supporting ischemic and catecholamine-related hepatotoxicity. Massive transfusion may have further increased oxidative stress, worsening liver failure and accelerating MODS and death.[5]

Neuroinflammatory Influence on Hepatic Function
TBI-induced neuroinflammation likely played a key role in hepatic injury by amplifying systemic inflammation, increasing catecholamine release, and triggering endothelial and microvascular damage[12]. Even without direct trauma, cytokines released during TBI can impair distant organs like the liver, underscoring the systemic nature of trauma-induced MODS.

Fulminant Liver Failure and Its Systemic Impact

FLF is a rare but critical complication in trauma, often linked to lactic acidosis and coagulopathy.[8] As the liver regulates detoxification and metabolism, its failure disrupts systemic homeostasis.

In this case, elevated lactate suggested hepatic hypoxia, which worsened renal function. This vicious cycle of acidosis and organ failure highlights the need for early interventions like renal replacement therapy and plasmapheresis.[5]

Role of Catecholamine Surge in Liver Injury

Catecholamine surges from trauma or TBI can induce hepatic vasoconstriction, worsening ischemia-reperfusion injury and hepatocellular damage.[11]

This mechanism is especially relevant in neurogenic shock or excessive sympathetic activation. Managing catecholamine levels, correcting metabolic imbalances, and initiating early dialysis or plasmapheresis may improve outcomes in trauma-related liver failure.

Renal Dysfunction Following High-Energy Trauma: Pathophysiology and Management Considerations

High-energy trauma triggers a surge of catecholamines (adrenaline, noradrenaline, angiotensin II), leading to renal vasospasm and reduced renal perfusion[7]. This is worsened by rhabdomyolysis and cytokine storms, further impairing kidney function.[9]

Factors like hemorrhage, hypovolemia, and massive transfusions contribute to renal tubular injury, hyperkalemia, and metabolic acidosis.[10]

If urine output remains inadequate despite fluids and diuretics, early hemodialysis is crucial[19]. Since AKI in trauma often results from a mix of SIRS, transfusions, and systemic inflammation, plasmapheresis alongside dialysis should be considered to prevent MODS.[19,20]

Cardiac Dysfunction Following High-Energy Trauma: Mechanisms and Management

High-energy trauma triggers a systemic inflammatory response, marked by sympathetic activation, catecholamine and angiotensin surges, electrolyte imbalances, and cytokine release, leading to myocardial dysfunction.[13] This can result in tachycardia, coronary vasospasm, and life-threatening arrhythmias.[12]

Key management includes correcting electrolyte and acid-base imbalances, improving cardiac preload, and preventing vasospasm. Milrinone or dobutamine may enhance cardiac output, while angiotensin blockade can improve stability.[13]

Even without direct cardiac injury, systemic inflammation may cause severe dysfunction. When inflammation and renal failure persist, early dialysis and plasmapheresis should be considered.[17]

- ACE Inhibitors: Reduce vascular resistance, support natriuresis, and prevent adverse remodeling. Shown to improve cardiac function post-ischemia.[14]
- Milrinone: Inotropic and vasodilatory, with anti-inflammatory effects in TBI. Useful for cardiac and cerebral support.[15,16]
- Amiodarone: Effective for managing ventricular arrhythmias in trauma, particularly in shock-resistant VF/VT.[18]

Combined use of ACE inhibitors, inotropes, and antiarrhythmics may stabilize cardiac function and reduce mortality in severe trauma.

Conclusion

High-energy multi-trauma requires prompt, multidisciplinary management. This case (see Table 1 for clinical timeline and key interventions) demonstrates that end-organ dysfunction can develop rapidly, even when initial vitals are stable, particularly in patients with suspected tra-umatic brain injury (TBI). Key lessons and practical recommendations include:

1. Continuous monitoring and repeat imaging are essential to detect delayed organ injuries.
2. Early recognition and management of sympathetic overactivation and catecholamine surges can prevent cardiac, hepatic, and renal complications.
3. Aggressive correction of acidosis and electrolyte imbalances, along with careful transfu-sion practices, reduces the risk of MODS.
4. Early initiation of renal replacement therapies (hemodialysis) and plasmapheresis may mitigate AKI and systemic inflammation.
5. Targeted cardiac support using ACE inhibitors, inotropes (e.g., milrinone or dobutami-ne), and antiarrhythmics (e.g., amiodarone) can stabilize hemodynamics and prevent life-threatening arrhythmias.

In summary, rapid identification of evolving organ dysfunction, guided by continuous monitoring and supported by timely, multidisciplinary interventions, is critical to improving outcomes and preventing irreversible deterioration in trauma patients.

Conflict of Interest

The authors declare that there is no conflict of interest.

Compliance of Ethical Statement

Written informed consent for publication could not be obtained due to the patient’s death. The case has been anonymized to protect patient privacy, and all efforts were made to ensure ethical reporting standards.

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Author Contributions

Y.B.: Study Design, Data Collection, Literature Review, Writing; A.H.E.: Critical Revision

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