

Splenic Rupture, Pancreatic Laceration, and Post-Traumatic Diaphragmatic Hernia Presenting as Hemorrhagic Shock: A Case Report

Sosa-Medellin Miguel Ángel^{1*}, González-Naranjo Eduardo Enrique¹, Trejo-Roldan Suridday¹, Estrada-Lugo Diana Evelin¹, Sanchez-Garcia Sonia Isanami¹, Sosa-Lumbreras Mario², Rocha-Contreras Jesús Daniel² and Cifuentes-Quiroga Inés Teresita³

¹Department of Intensive Care, High-Specialty Medical Unit, Traumatology and Orthopedics Hospital No. 21, Mexican Social Security Institute, Monterrey, Nuevo León, Mexico.

²Department of General Surgery, High-Specialty Medical Unit, Traumatology and Orthopedics Hospital No. 21, Mexican Social Security Institute, Monterrey, Nuevo León, Mexico.

³Department of Anesthesiology, High-Specialty Medical Unit, Traumatology and Orthopedics Hospital No. 21, Mexican Social Security Institute, Monterrey, Nuevo León, Mexico.

*Correspondence:

Miguel Angel Sosa Medellin, Department of Intensive Care, High-Specialty Medical Unit, Traumatology and Orthopedics Hospital No. 21, Mexican Social Security Institute, Monterrey, Nuevo León, Mexico.

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ABSTRACT

We present the case of a 62-year-old male patient who was admitted to the emergency department following a motor vehicle collision, presenting with severe hemodynamic instability due to hemorrhagic hypovolemic shock. Initial clinical and tomographic evaluation identified a massive hemopneumothorax and splenic injury, while the traumatic diaphragmatic hernia and pancreatic laceration were confirmed through emergency surgical exploration. An exploratory laparotomy, splenectomy, and diaphragmatic repair were performed under a massive transfusion protocol. The patient showed a favorable clinical evolution during his stay in the intensive care unit, achieving hemodynamic stability, mechanical ventilation weaning and discharge to the general surgery ward 48 hours after the surgical intervention.

Keywords

Splenic rupture, Pancreatic laceration, Traumatic diaphragmatic hernia.

Introduction

Blunt thoracic and abdominal trauma represents one of the leading causes of morbidity and mortality in emergency departments worldwide. Despite having properly established and widely known advanced management protocols, uncontrolled bleeding remains the primary cause of preventable death following trauma. Sudden deceleration forces can cause an abrupt increase in intra-abdominal pressure, which can rise to 200 cmH₂O, transferring significant kinetic energy that results in severe organ injuries. Within the abdominal cavity, the spleen stands out as one of the most frequently injured solid organs following blunt trauma [1-3].

Traumatic diaphragmatic hernia is a relatively uncommon entity, with a global incidence ranging from 1-15% in trauma patients, representing approximately 2-6% of all combined thoracic and

abdominal trauma cases. Its prevalence varies according to the mechanism of injury, occurring in 1-7% of blunt trauma compared to 10-15% observed in penetrating trauma. Anatomically, lesions in the left hemidiaphragm are significantly more common, accounting for up to 75% of cases in blunt trauma, due to congenital weakness in muscular fusions and the protective effect exerted by the liver over the right hemidiaphragm. Despite its rarity, traumatic diaphragmatic hernia carries a high mortality rate ranging between 30-45%, which increases considerably with late diagnosis [3,4]. In this same context, pancreatic trauma is a rare event that occurs in only 2-4% of patients suffering abdominal injuries, yet it harbors catastrophic potential. Protected by its retroperitoneal position, the existence of pancreatic trauma implies the presence of a massive energy impact, which explains why it presents almost exclusively with concomitant damage to other intra-abdominal organs, vastly complicating its diagnosis through conventional imaging modalities. Hemorrhagic shock secondary to these combined injuries requires an immediate multidisciplinary approach, integrating massive transfusion protocols and timely damage

control surgery [5].

This case report shows the diagnostic and therapeutic challenge of severe hemorrhagic hypovolemic shock associated with complex multivisceral displacement, highlighting the importance of timely surgical treatment in hemodynamically unstable patients.

Case Presentation

A 72-year-old male without relevant medical history was admitted to the emergency department after suffering a motor vehicle collision. The mechanism of injury consisted of a frontal vehicular impact that caused direct thoracic trauma against the steering wheel.

Upon arrival the patient was neurologically intact with a Glasgow Coma Scale score of 15. Nevertheless, he presented with hemodynamic instability characterized by a blood pressure of 70/30 mmHg, a heart rate of 110 beats per minute, and a capillary refill time of 5 seconds. Physical examination revealed hypoventilation in the left hemithorax, with an oxygen saturation of 88%. Abdomen findings included both deep and superficial epigastric pain alongside board-like rigidity.

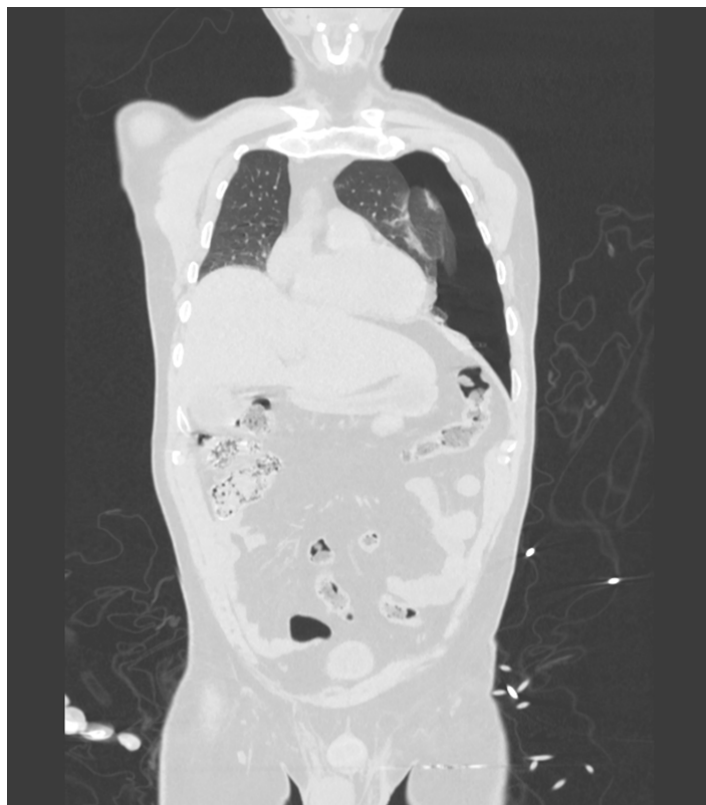


Figure 1: Coronal computed tomography image showing traumatic rupture of the left hemidiaphragm with herniation of abdominal viscera into the left hemithorax and associated hemopneumothorax.

The initial diagnostic approach via chest radiography and thorax tomography documented a massive left hemopneumothorax (Figure 1), managed through a chest tube, which initially

drained 860 mL of serosanguinous fluid. Contrast-enhanced abdominal computed tomography revealed pneumoperitoneum in the left parietocolic gutter and a splenic injury. An emergency transfusion protocol was immediately initiated with two red blood cells concentrates alongside the administration of one gram of intravenous tranexamic acid.

Given the signs of hypovolemic shock despite initial fluid and transfusion treatment, the patient was urgently transferred to the operating room. Under general anesthesia, a surgical approach was performed via a midline supra and infraumbilical incision. During tissue dissection into the abdominal cavity, a defect in the left hemidiaphragm measuring approximately 12 cm in diameter (grade 4 diaphragmatic injury) was identified. Herniating through this defect into the ipsilateral hemithorax were the stomach, spleen, and tail of the pancreas with no arterial or venous injury involvement.

Following careful reduction of the visceral contents back into the abdominal cavity, a grade 3 splenic injury was evidenced, featuring active bleeding refractory to conventional hemostatic maneuvers. Consequently, a splenectomy was performed through clamping and sectioning of the splenic hilum. The diaphragmatic defect was primarily repaired using a continuous 2-0 nylon suture. Additionally, a superficial laceration was detected in the tail of the pancreas (grade 2 pancreatic injury) without evident ductal involvement. Pancreatic hemostasis was successfully achieved through manual compression and polypropylene sutures. A systematic review of the cavity, including the entire length of the small intestine from the ligament of Treitz to the ileocecal valve, as well as inspection of the liver and colon, revealed no further abnormalities. After confirming hemostasis in the left upper quadrant an open drain was placed in the left flank.

Overall intraoperative bleeding of 2500 mL warranted the initiation of a massive transfusion protocol throughout the procedure including 8 red blood cells concentrates, 4 fresh frozen plasmas, and 1 platelet apheresis.

Subsequently, the patient was admitted to the intensive care unit, where the massive transfusion protocol continued, completing the 1:1:1 ratio for blood components transfusion. Neurologically, the patient was initially maintained under sedation and analgesia targeting a Richmond Agitation-Sedation Scale (RASS) score of -4 using intravenous benzodiazepines and opioids. The initial ventilation strategy used pressure-limited assist-control modality, achieving adequate lung protection. Hemodynamically, early stabilization was achieved without the need for norepinephrine, maintaining the mean arterial pressure within perfusion ranges, sinus cardiac rhythm, and normalized capillary refill.

24 hours after admission into the intensive care unit, having verified the lack of active bleeding, absence of tissue hypoperfusion markers, and hemodynamic stability, the mechanical ventilation weaning protocol was initiated. Highly favorable extubation predictors were documented, including a negative inspiratory

force of -40 cmH₂O, a positive cuff leak test, and a peak cough flow of -70 L/min. The spontaneous breathing trial was successful, leading to complication free elective extubation.

The patient was transferred to the general surgery ward in a stable clinical condition; achieving hospital discharge 5 days after the event.

Discussion

The convergence of extensive diaphragmatic disruption, splenic rupture, and pancreatic laceration in a single traumatic event turns into a multivisceral injury phenotype that challenges conventional resuscitation and damage control algorithms. Physiopathologically, the loss of continuity in the left hemidiaphragm eliminates the normal anatomical barrier and subjects the abdominal organs to a continuous and extreme pleuroperitoneal pressure gradient. This phenomenon facilitated the acute herniation of the stomach, spleen, and pancreas into the thoracic cavity, which not only collapsed the adjacent pulmonary parenchyma but also caused mechanical distortion of the mediastinal structures. Such intrathoracic compression severely compromises venous return and cardiac output, synergistically exacerbating tissue hypoperfusion in a patient already experiencing active hemorrhagic shock secondary to splenic parenchymal and vascular injury.

The diagnostic challenge in these combined injuries is superlative. Diaphragmatic injuries, like pancreatic trauma, are often silent in their initial stages and present a significant diagnostic omission rate even with the use of high-resolution computed tomography. In this scenario, the magnitude of the multivisceral herniation and the pancreatic laceration could only be diagnosed and characterized through direct surgical exploration, reaffirming that an initial imaging evaluation with subtle or negative findings should not rule out clinical suspicion in the face of high-energy mechanisms [3,4].

The principles of therapeutic management were based on early hemostatic resuscitation and the use of timely damage control surgery. In accordance with international consensus for massive hemorrhage management, the early administration of tranexamic acid and restrictive fluid resuscitation under transient permissive hypotension regimens were fundamental to avoiding the lethal triad of trauma and achieving stabilization prior to anesthetic induction. The transabdominal approach via an exploratory laparotomy constitutes the gold standard in acute settings, as it allows for the anatomical reduction of the herniated viscera, exhaustive inspection of the peritoneal cavity, and primary repair of the diaphragm using tension-free non-absorbable suture material [2,4,6].

Although the contemporary surgical trend in splenic trauma prioritizes organ preservation through non-operative management or arterial angioembolization, the profound hemodynamic instability of the patient and refractory active hemorrhage dictated

the imperative need to perform an emergency open splenectomy. Similarly, upon confirming the integrity of the main pancreatic duct during exploration of the omentum (grade 2 injury), a conservative management of the parenchyma was chosen with mechanical hemostasis, primary closure, and closed drainage placement, avoiding a distal pancreatectomy that would have drastically increased morbidity and surgical time in a critically compromised patient. Finally, close surveillance in the intensive care unit, focused on the optimization of tissue perfusion and early weaning from invasive mechanical ventilation, proved vital in preventing catastrophic complications such as nosocomial or ventilation associated pneumonia, diaphragmatic weakness, the formation of intra-abdominal abscesses, and the development of pancreatic fistulas [2,5,6].

Conclusion

Traumatic diaphragmatic hernia complicated by multivisceral herniation and associated blunt injuries to the spleen and pancreas constitutes a surgical emergency of extreme complexity. This case illustrates that, in the face of high-energy trauma, the highest index of clinical suspicion must be maintained to identify diaphragmatic disruptions and retroperitoneal damage that can easily go unnoticed in initial evaluations. Urgent exploratory laparotomy remains the indispensable diagnostic and therapeutic approach in patients with hemodynamic instability. Therapeutic success and patient survival depend absolutely on the timely integration of massive transfusion protocols, the precise execution of damage control surgical techniques, and meticulous postoperative management in the intensive care unit.

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