Fatal Blunt Severe Hemorrhagic Pancreatitis Trauma: A Case Report

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1. Introduction

Pancreatic trauma, while relatively uncommon compared to injuries to other abdominal organs, poses a significant clinical challenge due to its potential for severe complications and high mortality rates. The pancreas, a retroperitoneal organ with both exocrine and endocrine functions, is vulnerable to injury from various mechanisms, including blunt and penetrating trauma. Blunt pancreatic trauma, often resulting from motor vehicle accidents, falls, or assaults, can lead to a spectrum of injuries ranging from minor contusions to complete transection of the pancreas. The severity of pancreatic injury can vary widely, and the clinical presentation may be nonspecific, making prompt diagnosis and appropriate management crucial for optimal outcomes. One of the most devastating consequences of blunt pancreatic trauma is severe hemorrhagic pancreatitis (SHP). This life-threatening condition is characterized by extensive pancreatic necrosis, hemorrhage, and a systemic inflammatory response. The pathophysiology of SHP involves the activation of pancreatic enzymes within the gland, leading to autodigestion of the pancreatic parenchyma. This process triggers a cascade of events, including inflammation, vascular damage, and the release of inflammatory mediators, which can result in multi-organ dysfunction and death. The diagnosis of pancreatic trauma can be challenging due to the retroperitoneal location of the pancreas and the nonspecific nature of its clinical presentation. Patients
may present with abdominal pain, nausea, vomiting, and signs of shock, which can be attributed to various other causes. Imaging modalities such as computed tomography (CT) scans play a crucial role in the diagnosis and assessment of pancreatic injuries. CT scans can identify pancreatic lacerations, contusions, hematomas, and fluid collections, aiding in the grading of the injury and guiding management decisions. However, in some cases, the diagnosis may only be confirmed during surgical exploration or autopsy.1-3

The management of blunt pancreatic trauma depends on the severity of the injury. In mild cases, conservative management with pain control, bowel rest, and close monitoring may be sufficient. However, in severe cases, such as those involving major ductal disruption, extensive necrosis, or significant hemorrhage, surgical intervention is often necessary. Surgical options may include drainage of fluid collections, debridement of necrotic tissue, pancreatic resection, or even total pancreatectomy in extreme cases. The timing of surgery is also critical, as delayed intervention can lead to increased morbidity and mortality. Several factors can influence the prognosis of patients with blunt pancreatic trauma. These include the severity of the injury, the presence of associated injuries to other organs, the timeliness of diagnosis and intervention, and the overall health status of the patient. Early recognition of pancreatic injury, prompt resuscitation, and appropriate surgical management are essential for improving outcomes in these patients. In addition to the clinical challenges associated with pancreatic trauma, there are also forensic implications. In cases of assault or homicide, pancreatic injuries can provide crucial evidence regarding the mechanism and cause of death. Forensic pathologists play a vital role in identifying and documenting pancreatic injuries during autopsies, which can aid in legal investigations and prosecutions.4-6 This case report presents a unique and tragic instance of fatal blunt severe hemorrhagic pancreatitis resulting from an assault. The victim, a 21-year-old male student, sustained multiple punches to the abdomen, leading to extensive pancreatic damage, hemorrhage, and ultimately, death. This case highlights the potential severity of blunt pancreatic trauma, even in seemingly healthy young individuals. It also emphasizes the importance of raising awareness of pancreatic trauma among healthcare professionals, law enforcement agencies, and the public. Early recognition of the signs and symptoms of pancreatic injury can lead to timely intervention and potentially improve outcomes for victims of blunt abdominal trauma.

2. Case Presentation

On November 18th, 2022, at approximately 10:00 PM, local authorities discovered a 21-year-old male student unconscious in his dormitory room. The initial investigation revealed that the victim had been involved in a physical altercation earlier that evening with a group of 13 individuals, during which he sustained multiple blows to the abdomen. Witnesses reported that the victim was repeatedly punched in the abdomen before being rendered unconscious and left on the sidewalk outside the dormitory. Emergency medical services were promptly contacted, and the victim was transported to the nearest hospital. Upon arrival, the victim was unresponsive, exhibiting signs of shock, including hypotension and tachycardia. Despite aggressive resuscitative efforts, the victim tragically succumbed to his injuries and was pronounced dead shortly after arrival. Given the circumstances surrounding the victim’s death, a comprehensive post-mortem examination was requested by the authorities to ascertain the cause of death and assess the extent of the injuries sustained during the assault. The external examination of the victim’s body revealed extensive bruising on the abdomen, consistent with blunt force trauma. The bruising was predominantly located in the upper abdomen, extending from the epigastric region to the left and right hypochondriac regions. The bruises varied in size and color, suggesting that the victim had sustained multiple impacts to the abdomen at different times during the assault.

The internal examination of the victim’s body revealed a cascade of severe injuries resulting from the blunt abdominal trauma. The cardiovascular system exhibited signs of stress, with enlargement of the heart.
and dilation of the coronary arteries. The abdominal cavity showed significant damage, including colonic vasodilation, dilated stomach arteries with mesocolonic ecchymosis, and copious blood on the surface of the pancreatic tissue. The spleen was wrinkled and presented with deep lacerations, edema, dilatation, and necrosis on the upper anterior portion of the diaphragm. Additionally, there was evidence of blood absorption in the left chest skin, although no fractures were observed. The examination of the respiratory system revealed left lower lung ecchymosis, edema, and congestion, suggesting that the victim may have experienced difficulty breathing due to the abdominal trauma. However, further examination ruled out pneumothorax and air embolism in all four ventricles of the heart, indicating that these conditions were not contributing factors to the victim’s death. To investigate the possibility of intoxication as a contributing factor, toxicological tests were conducted on the victim’s blood and urine samples. These tests included analyses for common drugs of abuse, alcohol, psychoactive substances, cyanides, pesticides, and insecticides. The results of all toxicological tests were negative, ruling out the possibility of intoxication as a cause or contributing factor to the victim’s death. The extensive damage to the pancreas observed during the internal examination prompted a more detailed histological analysis of the pancreatic tissue. This analysis revealed severe necrosis, characterized by widespread cell death and tissue destruction. Multiple sites of cytoplasmic necrosis were observed, indicating that the pancreatic cells had undergone irreversible damage. Furthermore, there was widespread hemorrhagic hyperemia of the peripancreatic tissue, indicating extensive bleeding and inflammation in the tissues surrounding the pancreas.

Based on the collective findings of the external examination, internal examination, toxicological tests, and histological analysis, the cause of death was determined to be severe hemorrhagic pancreatitis resulting from blunt force trauma to the abdomen. The multiple punches to the abdomen had inflicted severe damage to the pancreas, leading to a cascade of events that ultimately resulted in the victim’s untimely demise. The blunt force trauma to the abdomen caused direct damage to the pancreatic tissue, disrupting the pancreatic ducts and causing leakage of pancreatic enzymes into the surrounding tissues. These enzymes, which are normally involved in digestion, initiated a process of autodigestion, leading to extensive necrosis and hemorrhage within the pancreas. The inflammatory response triggered by the pancreatic injury further exacerbated the damage, leading to systemic complications and ultimately, the victim’s death. The severity of the pancreatic injury in this case was likely due to the repeated and forceful nature of the blows to the abdomen. The cumulative effect of multiple impacts caused extensive damage to the pancreatic tissue, overwhelming the organ’s ability to repair itself. The retroperitoneal location of the pancreas, while providing some protection from external forces, also made it more vulnerable to severe injury in the event of blunt abdominal trauma.

Figure 1. Pancreas. (a) Copious blood on the surface of the pancreatic. (b) On histologic analysis showed severe necrosis, several cytoplasmic necrosis sites, and widespread hemorrhagic hyperemia of peripancreatic tissue.
3. Discussion

This case serves as a stark reminder of the potential severity and life-threatening consequences of blunt pancreatic trauma. The pancreas, an organ nestled deep within the abdomen, plays a crucial role in digestion and glucose regulation. However, its anatomical location, tucked behind the stomach and other organs in the retroperitoneal space, makes it particularly vulnerable to injury from blunt force trauma. The retroperitoneal space, while offering some protection to the pancreas, also poses a diagnostic challenge. Unlike injuries to organs located in the peritoneal cavity, which often present with immediate and localized pain, pancreatic injuries may manifest with vague or nonspecific symptoms. This can lead to delays in diagnosis and treatment, significantly impacting the patient's prognosis. In the case of the 21-year-old male student, the initial presentation of unconsciousness and hypotension could be attributed to various causes, making it difficult to pinpoint the exact source of the problem. The extensive bruising on the abdomen, a telltale sign of blunt force trauma, raised suspicion of internal injuries. However, the specific diagnosis of severe hemorrhagic pancreatitis was only confirmed during the post-mortem examination. This highlights the importance of maintaining a high index of suspicion for pancreatic injury in cases of blunt abdominal trauma, even in the absence of specific signs or symptoms.

The mechanism of injury in this case, multiple punches to the abdomen, underscores the vulnerability of the pancreas to blunt force trauma. The repeated blows likely caused direct damage to the pancreatic tissue, leading to a cascade of events that ultimately proved fatal. The pancreas is a delicate organ, and even seemingly minor trauma can trigger a series of pathological processes that can quickly escalate. The initial insult to the pancreas, in this case, likely involved the disruption of the pancreatic ducts, the channels through which pancreatic enzymes are secreted into the small intestine. These enzymes, essential for digestion, are highly potent and can cause significant damage if they leak into the surrounding tissues. The leakage of pancreatic enzymes triggers an inflammatory response, leading to the release of cytokines and other inflammatory mediators. This inflammatory cascade, in turn, can cause vasodilation, increased vascular permeability, and edema, further compromising the blood supply to the pancreas and surrounding organs.

As the inflammatory process progresses, the pancreatic tissue begins to undergo necrosis, or cell death. This necrosis can be focal or diffuse, depending on the extent of the injury. In severe cases, such as the one presented here, the necrosis can involve the entire pancreas, leading to massive hemorrhage and the release of toxic substances into the bloodstream. This can trigger a systemic inflammatory response syndrome (SIRS), characterized by fever, tachycardia, tachypnea, and leukocytosis. If left untreated, SIRS can progress to multiple organ dysfunction syndrome (MODS) and death. The diagnosis of pancreatic trauma relies on a combination of clinical, laboratory, and imaging findings. In the acute setting, patients may present with abdominal pain, nausea, vomiting, and signs of shock. Elevated serum amylase and lipase levels, markers of pancreatic injury, may be present but are not always reliable indicators. Imaging modalities such as computed tomography (CT) scans are essential for visualizing the pancreas and assessing the extent of the injury. However, in some cases, the diagnosis may only be confirmed during surgical exploration or autopsy.

The management of blunt pancreatic trauma depends on the severity of the injury. In mild cases, conservative management with pain control, bowel rest, and close monitoring may be sufficient. However, in severe cases, such as the one presented here, surgical intervention is often necessary to control bleeding, debride necrotic tissue, and prevent further complications. Early diagnosis and prompt surgical intervention are crucial for improving outcomes in patients with severe pancreatic trauma. In this case, the victim's presentation with unconsciousness and hypotension likely precluded a timely diagnosis and intervention. The rapid progression of the pancreatic injury, from initial trauma to severe hemorrhagic pancreatitis and ultimately death, highlights the importance of early recognition and aggressive management of this condition. The post-mortem
examination played a crucial role in confirming the diagnosis and providing valuable insights into the pathophysiology of blunt pancreatic trauma. This case also serves as a stark reminder of the devastating consequences of violence. The victim, a young student, lost his life due to a senseless act of aggression. This case underscores the need for continued efforts to prevent violence and promote peaceful conflict resolution. It also highlights the importance of raising awareness of the potential dangers of blunt abdominal trauma, particularly among young people who may be more likely to engage in physical altercations.14-16

In this tragic case, the mechanism of injury involved a series of complex pathophysiological events triggered by blunt force trauma to the abdomen. The multiple punches delivered to the victim's abdomen initiated a cascade of events that ultimately culminated in severe hemorrhagic pancreatitis and subsequent death. The primary impact of the punches was direct damage to the pancreatic parenchyma. The pancreas, a retroperitoneal organ, is relatively protected from external forces due to its location deep within the abdomen. However, the repeated and forceful nature of the blows in this case overwhelmed this natural protection, causing direct injury to the pancreatic tissue. This direct injury likely resulted in contusions, lacerations, and disruptions of the pancreatic parenchyma, leading to immediate functional impairment. One of the critical consequences of the direct pancreatic injury resulting from the blunt force trauma was the disruption of the pancreatic ducts. The pancreas, a complex glandular organ nestled within the abdomen, plays a crucial role in digestion by producing and secreting a variety of enzymes that break down carbohydrates, proteins, and fats. These enzymes are synthesized within the acinar cells of the pancreas and are transported through a network of ducts that converge into the main pancreatic duct. The main pancreatic duct, also known as the duct of Wirsung, runs the length of the pancreas and joins with the common bile duct before emptying into the duodenum, the first part of the small intestine.15-17

The pancreatic ducts are lined with epithelial cells that form a protective barrier, preventing the leakage of pancreatic enzymes into the surrounding tissues. However, in the case of blunt pancreatic trauma, the forceful impact of the punches to the abdomen likely caused significant damage to the pancreatic parenchyma, including tearing or rupture of the pancreatic ducts. This disruption of the ductal system led to the leakage of pancreatic enzymes, including proteases, lipases, and amylases, into the surrounding tissues. The leakage of pancreatic enzymes into the surrounding tissues initiated a cascade of events that ultimately led to the development of severe hemorrhagic pancreatitis. Proteases, enzymes that break down proteins, began to autodigest the pancreatic tissue itself, causing extensive necrosis and inflammation. Lipases, enzymes that break down fats, caused fat necrosis in the surrounding tissues, further contributing to the inflammatory response. Amylases, enzymes that break down carbohydrates, also played a role in the inflammatory process. The inflammatory response triggered by the leakage of pancreatic enzymes led to the release of various inflammatory mediators, including cytokines, chemokines, and prostaglandins. These mediators attracted immune cells to the site of injury, further amplifying the inflammatory response. The inflammation caused vasodilation and increased vascular permeability, leading to edema and hemorrhage within the pancreas and surrounding tissues. The combination of pancreatic enzyme autodigestion, inflammation, edema, and hemorrhage resulted in the development of severe hemorrhagic pancreatitis. The extensive necrosis and hemorrhage within the pancreas disrupted the organ's normal function, leading to a systemic inflammatory response syndrome (SIRS). SIRS is a life-threatening condition characterized by a dysregulated inflammatory response that can lead to multiple organ dysfunction syndrome (MODS) and death.

In this case, the severe hemorrhagic pancreatitis caused by the blunt pancreatic trauma ultimately led to the victim's death. The extensive damage to the pancreas and surrounding tissues, combined with the systemic inflammatory response, overwhelmed the victim's physiological reserves and resulted in irreversible organ failure. The post-mortem
examination revealed the devastating consequences of the pancreatic duct disruption, highlighting the critical role of these ducts in maintaining the integrity and function of the pancreas. The disruption of the pancreatic ducts is a significant event in the pathophysiology of blunt pancreatic trauma. The leakage of pancreatic enzymes into the surrounding tissues triggers a cascade of events that can lead to severe hemorrhagic pancreatitis, a life-threatening condition. The case presented here underscores the importance of recognizing the potential for pancreatic duct injury in cases of blunt abdominal trauma and the need for prompt medical intervention to mitigate the devastating consequences of this injury.16-18

The pancreas, a vital organ nestled within the abdomen, plays a crucial role in digestion by producing a potent cocktail of enzymes. These enzymes, including proteases, lipases, and amylases, are typically secreted into the duodenum, the first segment of the small intestine, where they aid in breaking down proteins, fats, and carbohydrates, respectively. However, in the unfortunate event of pancreatic injury, such as in the case of blunt abdominal trauma, the delicate balance within the pancreas can be disrupted, leading to a catastrophic cascade of events known as leakage of pancreatic enzymes and autodigestion. The pancreas is a complex organ composed of two main types of cells: acinar cells and ductal cells. Acinar cells are responsible for producing and storing digestive enzymes in an inactive form within small granules called zymogen granules. These zymogen granules are then transported to the apical surface of the acinar cells, where they fuse with the cell membrane and release their contents into the pancreatic ducts. Ductal cells, on the other hand, line the pancreatic ducts and secrete bicarbonate-rich fluid, which helps to neutralize the acidic chyme entering the duodenum from the stomach and create an optimal environment for the pancreatic enzymes to function. Under normal physiological conditions, the activation of pancreatic enzymes is tightly regulated. The enzymes are synthesized and stored in their inactive forms within the zymogen granules, preventing them from prematurely digesting the pancreatic tissue itself. Activation of the enzymes occurs only when they reach the duodenum, where they encounter specific enzymes and an alkaline environment that trigger their conversion into active forms. This intricate regulatory mechanism ensures that the powerful digestive enzymes are unleashed only when and where they are needed, safeguarding the integrity of the pancreas and surrounding tissues.17-19

However, in the setting of pancreatic injury, this delicate balance can be disrupted, leading to the leakage of pancreatic enzymes into the pancreatic parenchyma and surrounding tissues. Blunt abdominal trauma, such as the multiple punches sustained by the victim in this case, can cause direct damage to the pancreatic tissue, disrupting the acinar cells and zymogen granules. This disruption can result in the premature activation of pancreatic enzymes within the pancreas itself, initiating a process of autodigestion. The proteases, lipases, and amylases released from the damaged acinar cells begin to break down the pancreatic tissue itself, as if it were food in the digestive tract. Proteases, such as trypsin and chymotrypsin, cleave proteins into smaller peptides and amino acids, causing extensive damage to the cellular structures and extracellular matrix of the pancreas. Lipases, such as pancreatic lipase, hydrolyze fats into fatty acids and glycerol, leading to the accumulation of fatty acids within the pancreatic tissue and further exacerbating the inflammatory response. Amylases, such as pancreatic amylase, break down carbohydrates into simple sugars, contributing to the overall metabolic dysregulation associated with pancreatic injury.16,17

The autodigestion process not only causes direct damage to the pancreatic tissue but also triggers a robust inflammatory response. The release of pancreatic enzymes and cellular debris activates the immune system, leading to the recruitment of inflammatory cells, such as neutrophils and macrophages, to the site of injury. These inflammatory cells release cytokines, chemokines, and other mediators that amplify the inflammatory response, leading to further tissue damage and dysfunction. The combined effects of autodigestion and inflammation can have devastating consequences for the pancreas.
and surrounding organs. The extensive tissue damage can lead to pancreatic necrosis, a condition characterized by the death of pancreatic cells and the formation of necrotic debris. The necrotic debris can serve as a breeding ground for bacteria, leading to the development of pancreatic abscesses and sepsis. The inflammatory response can also extend beyond the pancreas, affecting other organs such as the lungs, kidneys, and liver, leading to multiple organ dysfunction syndrome (MODS) and potentially death. In the case of the victim presented in this report, the blunt abdominal trauma sustained during the assault caused significant damage to the pancreas, leading to the leakage of pancreatic enzymes and the initiation of autodigestion. The post-mortem examination revealed extensive necrosis of the pancreatic tissue, multiple sites of cytoplasmic necrosis, and widespread hemorrhagic hyperemia of the peripancreatic tissue. These findings are consistent with the devastating consequences of pancreatic enzyme leakage and autodigestion. The severity of the pancreatic injury in this case was likely due to the repeated and forceful nature of the blows to the abdomen. The cumulative effect of multiple impacts caused extensive damage to the pancreatic tissue, overwhelming the organ’s ability to repair itself. The retroperitoneal location of the pancreas, while providing some protection from external forces, also made it more vulnerable to severe injury in the event of blunt abdominal trauma. The lack of immediate medical attention further exacerbated the injury, allowing the autodigestion and inflammatory processes to progress unchecked. The leakage of pancreatic enzymes and autodigestion is a catastrophic event that can occur in the setting of pancreatic injury. The premature activation of pancreatic enzymes within the pancreas itself leads to the breakdown of pancreatic tissue, triggering a robust inflammatory response. The combined effects of autodigestion and inflammation can have devastating consequences, including pancreatic necrosis, abscess formation, sepsis, and multiple organ dysfunction syndrome. The case presented in this report highlights the importance of recognizing the potential severity of blunt abdominal trauma and the need for prompt medical attention to prevent the devastating consequences of pancreatic enzyme leakage and autodigestion. The inflammatory response to the pancreatic injury involved the release of various inflammatory mediators, such as cytokines and chemokines, which attracted immune cells to the site of injury. This influx of immune cells, while intended to repair the damage, further contributed to the inflammatory cascade, leading to increased vascular permeability, edema, and hemorrhage. The hemorrhage, in turn, compromised the blood supply to the pancreatic tissue, further promoting necrosis and tissue damage.18,19

The combination of direct injury, autodigestion, inflammation, and hemorrhage resulted in extensive necrosis of the pancreatic tissue. Necrosis, or cell death, is a hallmark of severe hemorrhagic pancreatitis (SHP). The pancreatic acinar cells, responsible for producing digestive enzymes, are particularly vulnerable to injury. In this case, the repeated blows to the abdomen likely caused direct damage to these cells, leading to the release of activated pancreatic enzymes into the surrounding tissue. These enzymes, including trypsin, chymotrypsin, and elastase, initiated a process of autodigestion, breaking down the pancreatic parenchyma and surrounding tissues. As the pancreatic cells died, they released additional inflammatory mediators, such as cytokines and chemokines, into the bloodstream. These mediators triggered a systemic inflammatory response syndrome (SIRS), a life-threatening condition characterized by widespread inflammation, organ dysfunction, and circulatory failure. SIRS is a common complication of severe hemorrhagic pancreatitis and is associated with high morbidity and mortality rates. In this case, the SIRS progressed to multiple organ dysfunction syndrome (MODS), as the inflammatory response and circulatory failure affected multiple organ systems. The victim’s respiratory system was compromised, as evidenced by the left lower lung ecchymosis, edema, and congestion observed during the post-mortem examination. These findings suggest that the victim may have experienced acute respiratory distress syndrome (ARDS), a common complication of SIRS and MODS. ARDS is characterized by widespread

93
inflammation in the lungs, leading to impaired gas exchange, hypoxia, and respiratory failure. The cardiovascular system was also affected, with enlargement of the heart and dilation of the coronary arteries, indicating significant stress and dysfunction. The circulatory failure associated with SIRS and MODS can lead to myocardial ischemia, arrhythmias, and ultimately, cardiac arrest. In this case, the combination of respiratory and cardiovascular dysfunction, along with the extensive pancreatic necrosis and hemorrhage, proved fatal.\textsuperscript{15-17}

The rapid progression of the victim's condition from initial injury to death highlights the aggressive nature of severe hemorrhagic pancreatitis. The retroperitoneal location of the pancreas can make early diagnosis challenging, as the signs and symptoms may be nonspecific or masked by injuries to other organs. In this case, the victim presented with unconsciousness and hypotension, which could be attributed to various causes. The extensive bruising on the abdomen raised suspicion of internal injuries, but the specific diagnosis of severe hemorrhagic pancreatitis was only confirmed during the post-mortem examination. The absence of other significant injuries, such as fractures or internal bleeding from other organs, suggests that the pancreatic injury was the primary cause of death. The negative toxicological tests further support this conclusion, ruling out the possibility of drug or alcohol intoxication as contributing factors. The histological findings of severe necrosis, cytoplasmic necrosis, and hemorrhagic hyperemia of the peripancreatic tissue provide definitive evidence of the extensive pancreatic damage and the resulting inflammatory response. The mechanism of injury in this case involved a complex interplay of factors, initiated by blunt force trauma to the abdomen. The repeated punches caused direct damage to the pancreas, leading to disruption of the pancreatic ducts, leakage of pancreatic enzymes, and subsequent autodigestion of the pancreatic tissue. This process triggered a cascade of events, including inflammation, hemorrhage, necrosis, SIRS, and MODS, ultimately resulting in the victim's death. This case highlights the potential severity of blunt pancreatic trauma and the importance of prompt diagnosis and aggressive management to prevent fatal outcomes.\textsuperscript{19,20}

4. Conclusion

This case report presents a fatal instance of blunt severe hemorrhagic pancreatitis resulting from an assault. The victim, a 21-year-old male student, sustained multiple punches to the abdomen, leading to extensive pancreatic damage, hemorrhage, and ultimately, death. This case highlights the potential severity of blunt pancreatic trauma and the importance of prompt diagnosis and treatment. The retroperitoneal location of the pancreas can make diagnosis challenging, and delays in care can have devastating consequences.

5. References


