# **Case Report**

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# A case with missed pancreatic injury with massive pancreatic ascites and severe traumatic pancreatic necrosis: a case report and literature review

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#### **ABSTRACT**

Despite the major advances in the field of trauma management. High grade Blunt pancreatic injury remains a disease with both high mortality and morbidity specially in cases of delayed diagnosis. Herein we will present a case report of 31 years old Saudi male who was involved in motor vehicle accident with a delayed diagnosis of pancreatic injury associated with other multiple injuries. He was diagnosed late on 12th day of admission during laparotomy where a missed high-grade pancreatic fracture at the neck of pancreas, massive pancreatic ascites and extensive pancreatic necrosis was found. Open abdominal closure technique with mesh mediated fascial closure and Vacuum assisted closure was used to improve both respiratory function and abdominal disease through repeated debridement with peritoneal lavage and sequential approximation of fascia. He responded well gradually and offered definitive closure of fascia and feeding jejunostomy. Unfortunately, he had a sudden stroke followed by rapid deterioration then expired 48 hours later.

**Keywords:** Blunt pancreatic injury, Pancreatic ascites, Pancreatic necrosis

#### **INTRODUCTION**

Pancreatic trauma presents a major challenge to the surgeon in terms of both diagnosis and management. Despite the improvements in the pre-hospital care, intensive care therapy, and operative techniques, the mortality and morbidity associated with pancreatic injury remain high specially in cases of delayed diagnosis.

Diagnosis needs a high index of suspicion as symptoms and signs are often poor. Biochemistry, Contrast enhanced computerized scan (CECT), Endoscopic retrograde cholangiopancreatography (ERCP), and MRCP with conventional magnetic resonance imaging (MRI) can help to diagnose the situation. Treatment is

either conservative or surgery specially for higher grades of injury. The most important prognostic factor is pancreatic duct disruption and, once ductal disruption has been confirmed, early surgical intervention should be considered and specialist HPB surgeons should be consulted.

# **CASE REPORT**

A 31 years old Saudi male was admitted through Emergency Department as a victim of a motor vehicle accident. On arrival, he was comatosed and had convulsions. He was resuscitated according to ATLS protocol and intubated immediately. His vital signs were stable and maintained O<sub>2</sub> saturation. Clinically, he had a

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deformed mandible and diminished air entry bilaterally, extensive contusion over the right side of chest and abdomen and pelvis. No apparent long bone fractures but he had Hematuria. CXR showed moderate bilateral lung contusions and FAST ultrasonography showed a minimal to moderate intra-peritoneal fluid; increased to mild to moderate after 6 hours. X-ray pelvis showed a major stable fracture of the right side of the pelvis. Pan body computerized scan; with contrast enhanced CT of abdomen and pelvis (CECT) showed revealed bilateral lung contusion, the fracture of mandible and pelvis, low grade renal laceration with no extravasation and retroperitoneal hematoma, low grade splenic injury, mild to moderate free intrapertoneal fluid with no gross intracranial injuries.

He was shifted to ICU for conservative treatment under, general surgery, neurosurgery, thoracic surgery, urology, orthopedic, maxillofacial specialties and maintained stable vital signs and O2 saturation. Over the next few days, he maintained stable vital signs and had a failed trial for extubation. He was cleared from General surgery specialty on 5th day of admission He was followed by neurosurgery, thoracic surgery, urology, orthopedic and maxilla-facial specialties. On the 9th day of admission, he was referred again to general surgery because his Hemoglobin dropped 4 grams and developed abdominal distension. Clinically, he had a blood pressure of 130/70 mmhg, pulse: 100/min, SaO<sub>2</sub>: 93%, afebrile, intubated with increasing PEEP support with progressive worsening of ventilation settings as he developed ARDS. No inotropic support was started. The abdomen was tense, distended, with large sided contusion over the right side of chest and abdomen. Sluggish bowel sounds, and empty rectum were noted.

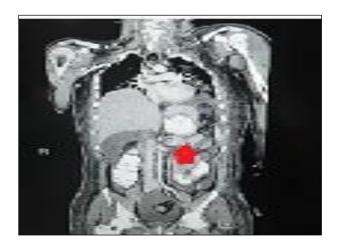


Figure 1: CECT showing a missed fracture of pancreas shown with massive ascites.

Urine was clear. Bedside USS revealed mild to moderate free intra-peritoneal collection. Patient did receive 2 units PRBCS since admission. Lab works showed Hb of 9.5, WBC 25,000, normal PT, PTT, INR, ABG, renal and liver functions and serum amylase was 254 units /L.



Figure 2: Intraoperative findings with severe traumatic pancreatic necrosis and wide spread inflammation and saponification.

The situation was discussed with other specialties including ICU and family were refusing a follow up CECT and agreed on 11th day and report showed only a mild to moderate intraperitoneal free collection; the pancreatic injury was missed (Figure 1). A decision was taken for decompressive laparotomy and exploration, but the family firstly refused then agreed later. Patient was taken to OR on 12th day of admission. Laparotomy through a long midline incision was done and a 4.5 L of free pancreatic ascites, extensive pancreatic necrosis and a fracture of pancreas at neck, inflammation and necrosis of omentum and areas of saponification dilation of whole gut (Figure 2). No solid organ injuries were detected. Samples were taken from ascetic fluid for cytology, biochemistry, and culture and sensitivity. Debridement was done and drainage of lesser sac pelvis together with through lavage with several liters of saline. The abdomen was left open and a Bogota bag placed (Figure 3). Postoperatively, S C octreotide and TPN were started. Amylase was uncountable in peritoneal fluid sample. Patient was taken in 48 hours for second look laparotomy; where minimal debridement needed as he showed improvement of inflammatory process, saline lavage and a double face mesh fixed to the fascia; after removal of Bogota bag for a mesh mediated closure. Vacuum assisted closure (VAC) system was used. He was taken to the theatre twice after 2; and then after 3 days later.



Figure 3: VAC system installed for open abdomen with underlying mesh.

Once the diagnosis of severe pancreatic injury has been made, a referral to a hepatobiliary center was done; but unfortunately, no reply was obtained. Each time, the abdomen was entered through the mesh at the midline. Exploration revealed gradual improvements of inflammation with improvement of edema of bowel. The volume of drain fluid and amylase and lipase levels were coming down gradually from 1500 ml to around 150 ml and the level of enzymes from uncountable to just two folds the serum level which was lastly 298 units /l. Gradual fascial approximation with mesh gradual plication after lavage with saline then closure of the mesh with running prolene no. 1 suture (Figure 4).

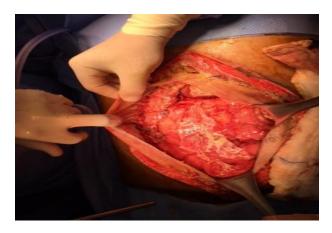


Figure 4: Exploring the abdomen through the mesh with improvement of inflammation and bowel dilatation.

Lastly, he was booked for definitive fascial closure together with a feeding jejunostomy to stop TPN as he showed improvements of ventilation settings, and vital signs were normal. WBC came back to normal. Unfortunately, he had a cerebral stroke with rapid deterioration with increasing inotropic and maximum PEEP support returned then; he died 48 hours later.



Figure 5: Great improvement and almost disappearance of ileus and inflammation with more fasial approximation without tension. Also, sponges of VAC shown after removal. 4 drains are shown here; upper 2 at bed of pancreas and lower most in the pelvis.

#### **DISCUSSION**

Pancreatic injury is uncommon, accounting for less than 7% of penetrating and 5% of blunt abdominal trauma.<sup>1,2</sup> These injuries rarely occur alone and are often associated with other intra-abdominal injuries.<sup>3</sup> Blunt isolated pancreatic trauma is even rarer, and its diagnosis, detection and treatment still remains a challenge. Physical signs and symptoms are often poor and laboratory findings non-specific, making diagnosis more difficult and easily missed. This delay may result to severe complications with an increase in morbidity and mortality rates.<sup>1,4</sup>

The retroperitoneal position of the pancreas influences the site and type of injury. The proximity of major vessels and surrounding viscera adds to the complexity of pancreatic injuries. Leakage of pancreatic exocrine secretions in cases of duct disruption augments the mechanical effects of local direct pancreatic injury, with peri-pancreatic oedema and tissue and fat necrosis. <sup>5,6</sup> Blunt midline upper abdominal trauma results in posterior compression of the anterior abdominal wall against the spine, with injury to the intervening pancreas overlying or to the left of the portal vein and superior mesenteric vessels. <sup>7</sup>

Isolated injuries to the pancreas are uncommon. 90% of cases have associated injuries, which cause most of the morbidity and mortality in patients with pancreatic trauma. The organs most commonly injured are the liver (42%), stomach (40%), major vessels (35%), thoracic viscera (31%), colon and small bowel (29%), central nervous system and spinal cord, skeleton and extremities (25%) and duodenum (18%).<sup>8-13</sup>

A high index of suspicion is required to diagnose a possible pancreatic injury. History and examination are non-specific. Laboratory tests are of little value in the early diagnosis of pancreatic trauma. An increase in serum amylase is time dependent and was found to be elevated in all patients with pancreatic injury after three hours; yet over 30% of patients with severe pancreatic trauma have serum amylase levels within normal parameters. <sup>14-16</sup> Triple phase CT in haemodynamically stable patients has been shown to have a sensitivity and specificity as high as 80%. <sup>17</sup> It has been demonstrated that 20-40% of pancreatic injuries are missed if CT is performed within 12 hours of injury. <sup>18</sup>

Pancreatic duct injury is poorly characterized on CT and it is often detected during laparotomy. Laceration less than 50% of the diameter of the pancreas usually indicates no ductal injury. Magnetic resonance cholangiopancreaticography (MRCP) or endoscopic retrograde cholangiopancreaticography (ERCP) can be used for further assessment although these modalities are not suitable in the acute stage. 19,20 American Association for the Surgery of Trauma grading include: grade I-minor contusion or laceration with no duct injury, grade II-

major contusion or laceration with no duct injury, grade III-transection or major laceration with duct disruption in distal pancreas, grade IV-transection of proximal pancreas or major laceration with associated injury to the ampulla, grade V-Massive disruption of the pancreatic head.<sup>21</sup>

The principles of management of pancreatic trauma include the need for early diagnosis and accurate definition of the site and extent of injury in order to facilitate optimal surgical intervention, otherwise, serious complications if the injury is underestimated or treated.<sup>1,2,11</sup> Major inappropriately complications including pancreatic fistula, pseudocyst, abscess or haemorrhage occur in one-third of surviving patients.<sup>1,10,11</sup> Delay in diagnosis and intervention is the most important cause of increased morbidity and mortality, mostly, the diagnosis is made laparotomy. 12,15,16

Blunt trauma to the pancreas causing mostly minor contusions or lacerations of the pancreatic substance with intact duct requiring only external drainage using a closed silastic suction drain. Determining the presence and extent of a pancreatic injury intraoperatively requires recognition of the features indicating a potential pancreatic injury, adequate exposure of the pancreas, definition of the integrity of the pancreatic parenchyma and determination of the status of the major pancreatic duct. The commonest major injury is a prevertebral laceration of the proximal body or neck of the pancreas which requires a distal pancreatectomy. Major fractures to the right of the portal vein with an intact bile duct are similarly best treated by distal resection.

Prognosis is determined by the cause of the injury, the extent of blood loss, the presence or absence of shock, rapidity of resuscitation, magnitude of associated injuries and nature and site of the pancreatic injury. Early mortality is due to uncontrolled or massive bleeding from associated vascular or visceral injuries. Late mortality is a consequence of infection and multiple organ failure. Neglect of major duct injury may lead to serious complications including fistulas, pseudocyst formation, sepsis, pancreatitis, and bleeding. Distal injury with duct disruption Injury to the neck, body or tail of the pancreas with major lacerations or transections and associated pancreatic duct injury is best treated by distal pancreatectomy with or without pancreaticoenteric anastomoses. 13,23

Pancreaticoduodenectomy is reserved for severe high-grade injuries.<sup>2</sup> With careful assessment of the injury by inspection, pancreatic complications can be reduced without the need for complex resections, enteric diversions and pancreaticoenteric anastomoses.<sup>22,23</sup>

The most common specific complication following pancreatic injury is a pancreatic fistula. This occurs in 10-20% of major injuries to the pancreas as in our case. Most

fistulas are minor and resolve spontaneously within 1 or 2 weeks of injury, provided adequate external drainage has been established. High-output fistulas (>700 ml/day) usually indicate major pancreatic duct disruption. Nutritional support is standard important, and the role of somatostatin and octreotide is unproven.<sup>24</sup>

Intra-abdominal sepsis requires guided aspiration to obtain fluid for bacteriology and amylase content. Empiric parenteral antibiotic therapy should bused till results of cultures are obtained. Percutaneous aspiration or catheter drainage is usually effective in patients with accessible unilocular collections and no evidence of pancreatic necrosis. Surgery with debridement of necrotic tissues with external catheter drainage is indicated in the presence of necrosis. A secondary serious haemorrhage from the pancreatic bed or surrounding vessels rarely occur.<sup>1,2</sup>

Pancreatic ascites is an exudative ascites caused by nonmalignant disease of pancreas characterized by very high fluid amylase level over 1000U/L and ascetic fluid protein concentration more than 3gm/dl.<sup>26-28</sup> The fluid is caused by leakage of pancreatic juice through a pseudocyst or a disruption in the pancreatic duct which is secondary to chronic pancreatitis and rarely after acute pancreatitis or trauma as in our case.<sup>29</sup> Treatment options differ according to the underlying cause. They include conservative treatment with nutritional support, octeriotide, endoscopic stenting or surgery.<sup>30,31</sup>

#### **CONCLUSION**

The diagnosis of pancreatic trauma requires a high index of suspicion and detailed imaging studies. Grading pancreatic injury is important to guide operative management. The most important prognostic factor is pancreatic duct disruption and, once ductal disruption has been confirmed, early surgical intervention should be considered and specialist HPB surgeons should be involved.

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