

Case Report

Surgical management of blunt pancreatic trauma: A modus operandi or individualized therapy?

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Abstract

The overall rate of blunt pancreatic trauma observed in level one trauma centers is rather low compared to other injuries, with a reported prevalence of 0.4 per 100,000 hospital admissions. The situation may be further complicated by the presence of associated major visceral injuries in these patients. A number of previous reports indicate that blunt pancreatic trauma carries high morbidity and mortality rates, especially when diagnosis is delayed or inappropriate surgery is attempted. Many mandate prompt surgical explorations for organ-specific diagnosis on CT; however other literature and upcoming studies prove otherwise. Over the years, several technologic advances have increased the sophistication of non-operative management. In our case, a period of careful observation followed by surgical intervention did not adversely effect the outcome. The period of observation resulted in stabilization of other solid organ injuries with focus on pancreas during surgical exploration.

Introduction

Pancreatic injuries continue to be an ordeal for trauma surgeons. The relatively infrequent incidence, the complexity in making an apt diagnosis and high morbidity and mortality, justify the unease these injuries provoke. The management of blunt pancreatic injuries has been controversial, with some suggesting selective observation and others advocating immediate exploration to prevent the delay-induced escalation in morbidity and death.^{1,2} The situation may be further complicated by the presence of associated major visceral injuries in these patients. Here is one such case with forthcoming alternative treatments.

Case Report

A 22 years old male presented to our emergency room about 4 hours after sustaining a pedestrian injury with a fast moving vehicle of unknown velocity. The front wheel of the vehicle ran over his chest and upper abdomen. After initial resuscitation at a local hospital, he was shifted to our hospital. Primary survey of the patient was unremarkable except that he was tachycardiac with a pulse of 125 beats per minute and his blood pressure was 110/50 mm of Hg. He was fully conscious and oriented with a Glasgow Coma

Scale score of 15/15. Secondary survey revealed superficial abrasions on the left mid chest, along with the tyre mark going across his right lumbar quadrant to about 10 cms below his left nipple. There was bilaterally equal air entry on chest auscultation. Abdominal examination revealed superficial abrasions in the right half of the abdominal wall. Abdomen was mildly distended but moving equally with respiration. On palpation, there was some tenderness in the epigastric region with no definite peritoneal signs. Rest of the systemic examination including head and neck, cardiovascular system, and pelvis was unremarkable. There was no injury to all the four limbs.

After initial resuscitation and laboratory investigations, an ultrasound FAST (focused abdominal sonogram for trauma) was performed, which revealed free fluid in the hepatorenal pouch, between the bowel loops and the pelvic cavity. In view of his relatively stable haemodynamic status, he was planned for a CT scan of the abdomen and pelvis. CT scan with oral and I/V contrast revealed that this patient had injuries to multiple abdominal organs. There was a grade III laceration involving segment VI of the liver measuring 4.7cm and a grade II laceration over the caudate lobe of liver measuring 1.7 cm along with multiple other small lacerations in the right lobe. There was a full thickness laceration involving the mid and lower part of spleen measuring 2.8cm with normal pedicles. A bruise was noted at the junction of the body and tail of pancreas, but ductal continuity could not be commented upon by CT scan. There was a laceration involving the superior pole of right kidney and upper pole of the left kidney was completely shattered without any hilar injury. There was gross haemoperitoneum and retrohaemoperitoneum. Major vessels including aorta and inferior vena cava were unremarkable. There was evidence of bilateral mild pleural effusion with lung contusion in the right basal region. The hollow viscera including stomach, bowel loops and bladder were reported as unremarkable. In view of presence of fluid in peritoneal cavity, a diagnostic aspirate of the fluid was done under ultrasound guidance to exclude bowel perforation. The analysis of the fluid revealed no faecal matter or bowel contents, but amylase level was raised along with the leukocyte count. His other investigations revealed that his haemoglobin was 9.6 g/dL and liver

function tests were mildly deranged.

Despite having a trauma score of 12/12, it was decided to manage him conservatively in view of his stable haemodynamic status. There was a high suspicion of pancreatic injury because of CT findings and raised amylase content of peritoneal fluid, but it was decided to delay exploration in this patient because of anticipated troublesome bleeding from multi-organ involvement. He was started on somatostatin analogue, total parenteral nutrition and other supportive treatment. During his hospital stay he remained vitally stable with no signs of peritonitis over the next seven days.

However, one week after the initial injury, the patient started complaining of mild pain in left upper abdomen, which progressively increased over the next three days. He also started having high grade fever, with increasing leukocyte counts. A repeat CT scan of the abdomen and pelvis was performed which revealed a large 9.9 x 9.3 cms fluid collection in the lesser sac at the site of previous injury to the body of pancreas. Other organs including liver and spleen revealed significant improvement in the previously noted lacerations. In view of worsening abdominal symptoms, a decision was made to explore the patient. On exploration, the patient was found to have complete pancreatic disruption at the junction of body and tail leading to fluid collection and some peri-pancreatic necrosis. After thorough abdominal wash-out, distal pancreatectomy and splenectomy was performed.

Post operatively the patient made an uneventful recovery and was discharged home on 15th day of admission. On follow up visits, he was found to be asymptomatic and recovering well.

Discussion

The overall rate of blunt pancreatic trauma observed in level one trauma centers is rather low compared to other injuries, with a reported prevalence of 0.4 per 100,000 hospital admissions.^{3,4} Only a third of these admissions for pancreatic injuries occur as a result of blunt trauma.¹ A number of previous reports indicate that blunt pancreatic trauma carries high morbidity and mortality rates, especially when diagnosis is delayed or inappropriate surgery is attempted.⁵ Based on these reports, many authors mandate prompt surgical explorations for organ-specific diagnosis on CT;⁶ however other literature and upcoming studies prove otherwise.

Over the years, several technologic advances have increased the sophistication of non-operative management. More recently, the medical literature supports observation in selected patients with blunt pancreatic trauma. Blunt abdominal trauma patients with hyperamylasaemia who present with a reliable, benign abdominal examination are carefully observed and the serum amylase level is

reassessed after several hours. If these patients remain clinically stable, a trial of total parenteral nutrition (TPN) or elemental diet through a feeding jejunostomy and somatostatin analogues may result in decreased drainage and closure of minor injuries.⁴ On the other hand, any patient with blunt abdominal trauma who continues to have abdominal pain or who develops symptoms of pancreatic injury should be thoroughly reassessed for pancreatic injury.⁶ Persistent abdominal symptoms or elevation of serum amylase levels mandates further evaluation, which may include abdominal CT scanning, ERCP⁴ or surgical exploration. A pancreatic injury severity scale devised by the American Association for the Surgery of Trauma has been delineated in Table.

Table:** American Association for the Surgery of Trauma (AAST) Pancreatic Injury Severity Score.

Grades	Injury Description	AIS score*
I	Small haematoma without duct injury; superficial laceration without duct injury	2
II	Large haematoma without duct injury or tissue loss; major laceration without duct injury or tissue loss	2;3
III	Distal transaction or parenchymal laceration with duct injury	4
IV	Proximal transaction or parenchymal laceration involving ampulla	4
V	Massive disruption of pancreatic head	5

Abbreviations: AIS*: Abbreviated Injury Score

Derived from**:

Souba WW. ACS: principles and practice. 2004. Page 928.

In a retrospective study of 154 paediatric patients with blunt pancreatic trauma, Keller et al reported that about 80% of the patients with grade I or II pancreatic injuries were successfully managed conservatively without any need for intervention.⁷ On the other hand, 52% of the patients with grade III and above injuries required surgical intervention. They concluded that clinical deterioration and major ductal injury were the main indicators of surgical intervention. They also expressed the opinion that the ultimate outcome of these patients, and selective conservative treatment may be justified in patients with blunt pancreatic trauma.

Similar reports indicate that initial selection of patients with isolated pancreatic trauma for observation or surgery can be based on the determination of main pancreatic duct integrity.¹ Early ERCP showing intact pancreatic ducts, including the secondary and tertiary radicals, without any extravasation permits nonoperative therapy if no associated injuries are present. The major difficulty in this management scheme is to determine which patients warrant early ERCP, as noninvasive tests of pancreatic duct disruption including serum amylase level and CT scan of abdomen, are often not very helpful.

The major limiting factor with this selective approach is that patients requiring delayed surgical intervention after an unsuccessful period of observation or a subsequent operation due to undetected main pancreatic duct injury, demonstrate a higher rate of pancreas-specific mortality and morbidity.^{1,2} This over-riding morbidity has to be carefully balanced against the overzealous use of exploratory laparotomy, as pancreatic trauma itself is associated with poor outcome and high rate of post-operative complications. A recently published study suggests that despite higher rate of failure of non-operative management for blunt pancreatic trauma, the timing of operation in solid organ injury was irrelevant and not detrimental.⁸

Another complicating factor in blunt pancreatic trauma is the presence of associated abdominal and extra-abdominal injuries. Current literature supports non-operative management of blunt spleen and liver injuries, with extension of this practice to appropriate patients with multiple injuries. This practice of conservative treatment may be extended to select patients with pancreatic trauma. In our case, a period of careful observation followed by surgical intervention did not adversely affect the outcome. The period of observation resulted in stabilization of other solid organ injuries with focus on pancreas during surgical exploration.

In conclusion, the management of patients with blunt pancreatic injuries should be individualized. Selected

patients with stable abdominal signs without pancreatic ductal injuries may be carefully observed. Any deterioration of clinical situation or demonstration of pancreatic ductal injury should mandate an exploratory laparotomy. The treatment has to be tailored to individual situations, especially in patients with severe concomitant injuries.

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