

Case Report

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# Delayed presentation of isolated ductal rupture of pancreatic head from blunt abdominal trauma managed conservatively: A case report



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ARTICLE INFO	A B S T R A C T	
A R T I C L E I N F O Keywords: Isolated duct rupture MRCP Pancreatic head Trauma Octreotide Case report	Introduction: and Importance: Blunt abdominal injury causing significant and isolated major pancreatic injury is rare in adolescents and young adults, with a controversial approach to its management. <i>Case presentation:</i> We present our experience of diagnosis and management of the ductal injury of the pancreatic head (Grade III) in the setting of blunt abdominal trauma in a 20-year-old male diagnosed by a series of various tests including magnetic resonance cholangiopancreatography (MRCP) and managed by pigtail drainage and octreotide alone; contrary to the previous recommendations of management of high-grade pancreatic trauma through surgical approach or endoscopic retrograde cholangiopancreatography (ERCP) and stenting. <i>Clinical discussion:</i> Isolated ductal rupture of the pancreatic head can have delayed presentation within a window of time and can be diagnosed by a series of tests including hematological, biochemical, and radiological investigations. Conservative treatment is generally recommended for Grade I and II whereas a surgical approach is preferred for higher grade pancreatic injury. <i>Conclusions:</i> Pancreatic ductal injury must be kept in mind when present with vague symptoms in the setting of blunt abdominal trauma. Magnetic resonance cholangiopancreatography (MRCP) is the investigation of choice for the diagnosis of pancreatic ductal injury. Even higher-grade pancreatic injury (grade III) can be managed with a conservative approach with pigtail drainage and an appropriate dosage of occreotide.	

## 1. Introduction

The pancreas is one of the least common injured organs following blunt trauma abdomen with an overall incidence of blunt pancreatic trauma of just about 0.2%–0.3% [1]. In addition, isolated traumatic injuries to the retroperitoneal located pancreas without accompanying other solid organ injuries are extremely unusual and diagnosis may be difficult due to subtlety of symptoms and delay in presentation. Delayed presentation or clinical deterioration of the patient may in some instances be the first clue of an underlying occult or undetected injury. This delay can be associated with significant toil especially in a low-resource country as traumatic pancreatic injuries have a high grade of morbidity (39–60%) and mortality (10–30%) [2,3].

It is graded into five grades ranging from minor contusion or small laceration without duct injury to a major pancreatic duct or head disruption and transaction according to the American Association for the Surgery of Trauma-organ injury scale (AAST-OIS) [4]. The management of pancreatic trauma is debatable, however, on the basis of the AAST-OIS classification, it ranges from simple conservative treatment to various surgical procedures depending on the grade of pancreatic injury [4].

Herein, we report a case of a 20-year-old male who suffered a blunt abdominal injury in a motorbike accident in whom the delayed diagnosis of pancreatic ductal injury was made after a myriad of investigations. Despite having high-grade pancreatic injury (Grade III); he was successfully managed conservatively with pigtail drainage and octreotide injection. This case has been reported in line with SCARE criteria [5].

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Abbreviations: AAST-OIS, American Association for the Surgery of Trauma-Organ injury scale; ATT, Antitubercular therapy; ADA, Adenosine deaminase; ERCP, Endoscopic retrograde cholangiopancreatography; MRCP, Magnetic resonance cholangiopancreatography; SAAG, Serum-ascites albumin gradient.

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## 2. Presentation of case

A 20-year-old male with no significant medical/surgical history presented to emergency in a local health facility with a complaint of pain and cut injuries of the upper eyelid, pain in the left shoulder, and right forearm abrasions after suffering a fall injury from a motorbike. On arrival in ER, vitals were stable, general condition was fair, Glasgow coma scale (GCS) 15/15, with no significant findings on respiratory, cardiovascular, neurological, and abdominal examination. To exclude potential and life-threatening injuries, trauma series including X-rays of cervical and dorsolumbar spine, pelvis, chest, bilateral proximal upper and lower limb, computed tomography (CT) scan of the head, and relevant blood investigations were done which were normal (Fig. 1). Moreover, the ultrasonography (USG) of the abdomen and pelvis showed normal findings with normal size and outline of the pancreas with normal echotexture, no space-occupying lesion or calcification, no dilation of the main pancreatic duct, and without ascites. Thus, he was managed conservatively for the pain and regional injuries with metoclopramide, ketorolac, injection of tetanus toxoid, and ceftriaxone.

However, fifteen days after the initial trauma, he presented with complaints of multiple episodes of loose stool, vomiting, generalized abdominal pain for two days, an episode of fever, and gradual distention of the abdomen. On examination, he was ill-looking, with bilateral pitting edema of the lower limbs. Abdominal examination revealed a soft, slightly distended, tender abdomen with normal bowel sounds. Blood examination revealed leukocytosis (34,900/mm<sup>3</sup> with 90% neutrophils), thrombocytosis (760000/mm<sup>3</sup>), low albumin (2.7 gm/dl) with normal renal function tests, and liver function test. X-ray abdomen erect and supine showed bilateral pleural effusion (Fig. 2). USG abdomen and pelvis showed moderate thick echogenic free fluid with septations in the pelvic and peritoneal cavity. Plain CT of the abdomen showed a hypodense collection of fluid density in the right subphrenic, perihepatic, subhepatic region, right paracolic gutter, thick enhancing fibro-collagen membrane shifting bowel loops to the left side. Similar collections were



Fig. 1. Initial X-ray shows no evidence of pleural effusion.



Fig. 2. Chest x-ray obtained later showing evidence of bilateral pleural effusion.

also present in the perisplenic region extending into the left paracolic gutter and pelvic cavity with mild dilated and reactive thickening of bowel wall-possibility of encapsulating peritoneal sclerosis and bilateral pleural effusion (Right > Left). The pancreas was normal in size, outline, and attenuation with no dilatation of the main pancreatic duct (Figs. 3 and 4). Thus, with a provisional diagnosis of chronic hemoperitoneum in the setting of blunt abdominal trauma along with abdominal tuberculosis (TB) based on prior history of contact with TB patients, he was planned to start on anti-tubercular therapy (ATT). With these complaints, the patient visited our center. At the time of his presentation to our center, he was ill-looking, pale, and tachycardic. The abdomen was tense, tender, and distended with guarding. Examination of other organ systems was grossly normal. Hematological investigations revealed persistent leukocytosis, low Hb, elevated erythrocyte sedimentation rate (ESR-52mm/hr), increased serum amylase, deranged liver function test, and increased adenosine deaminase (ADA) (Table 1).

A diagnostic ascitic fluid tapping was done, the evaluation of which revealed increased amylase, lymphocytosis, and low albumin. Pleural fluid analysis showed leukocytosis with 60% lymphocytic predominance. No growth was observed on peritoneal and pleural fluid. Serumascites albumin gradient (SAAG) was calculated to be less than 1.1 gm/ dl. Cytopathology was negative for malignant cells. Upper gastrointestinal endoscopy was normal. In order to drain ascitic fluid, a drain was placed in the pelvic and subhepatic regions. Drain fluid examination showed leukocytosis, raised amylase level, and a high ADA of 214. However, the acid-fast bacilli stain and Mantoux test were negative (Table 2).

Based on this, still, a provisional diagnosis of ascites with pleural effusion under evaluation to rule out tubercular sepsis was made. However, on the basis of increased amylase level in ascitic and drain fluid with abdominal distension, magnetic resonance pancreatography (MRCP) was done as USG and a CT scan of the abdomen did not reveal any specific pancreatic abnormality. On MRCP, the pancreatic duct wasn't visualized on the head. An approximately 4.4cm  $\times$  2cm x 1.5cm



Fig. 3. Initial computed tomography (CT) scan of the abdomen showing normal pancreatic structure with ascites (arrow).



Fig. 4. Plain CT pelvis obtained later shows fluid collection in peritoneal and pelvis cavity.

sized T2 high signal intensity fluid collection was seen anterior to the head, likely pseudocyst communicating with the pancreatic duct (Fig. 5). However, the pancreatic duct in the body and tail were normal. On the basis of these findings, finally, a diagnosis of traumatic ductal rupture of the pancreatic head (grade III pancreatic injury) with pseudocyst and pancreatic ascites was made. He was planned for ERCP and stenting along with octreotide and drainage by the team of gastroenterologists. However, the condition of the patient was not suitable to undergo ERCP and was managed with pigtail drainage and injection of octreotide (50 mcg subcutaneous three times a day) for two weeks. During this time, the condition of the patient improved significantly, and thus the conservative management was continued with octreotide in the dose of 100mcg along with other symptomatic management for another four weeks. During the subsequent follow-up, he had improved clinically with no abdominal discomfort and distension.

#### 3. Discussion

Being an immobile retroperitoneal injury, compression of the pancreas to the vertebral column during a high-velocity impact injury may cause transaction of the gland and even rupture of the duct and can present acutely or months later [2]. Majority of pancreatic injuries involve the body and the neck [6]. The retroperitoneal location of the pancreas makes signs and symptoms typical of intra-abdominal injuries less obvious, and the diagnosis and management can be challenging. Concurrent small bowel lesions occur in approximately 90% of patients with pancreatic injury [6]. As presented in this case, isolated injury to the pancreas by blunt abdominal trauma is very rare.

Early symptoms may be extremely subtle being limited to mild epigastric pain while in delayed cases it may present as peritonitis or with complications as subsequently discussed [7]. Thus, the diagnosis of

#### Table 1

Table showing the hematological parameters at the time of presentation.

Parameters	Result	Reference Range
Hematology		
Total Leukocyte Count (TLC)	16,000/mm <sup>3</sup> (80%	4000–11,000
	Neutrophils)	
Hemoglobin	8.7 gm%	12–18
Platelets	816,000/mm <sup>3</sup>	150000-400000
Biochemistry		
Amylase	19600U/L	28-100
Total bilirubin	13.0uMol/L	3–21
Direct Bilirubin	3.0uMol/L	
SGPT/ALT	17.0U/L	<42
SGOT/AST	27.0U/L	<37
Alkaline phosphatase	151.0U/L	30–90
Total protein	62.0 gm/l	60-80
Serum Albumin	19.0 gm/l	38–49
Gamma GT	78.0U/L	11-50
ADA test	80.0U/L	0–30
Urea	4.3mmol/L	1.6-7.0
Creatinine	75.0uMol/L	60-115
Sodium	130.0mEq/l	135-145
Potassium	4.2mEq/l	3.5-5.2
PT/INR	15.0/1.25secs	10-12/1.0-1.3
ESR	52mm/hr	0–9

Table	2
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Results of various fluid analysis.

ASCITIC FLUID				
Test	Result	Reference Range		
TLC	16,270/mm <sup>3</sup> (15% polymorph, 85%	0–5		
	monomorphs)			
Amylase	>10000 U/L	similar to serum		
		level		
sugar	1.6mmol/L	2.3-4.6		
albumin	2.1g/dL	3.8-4.9		
protein	2.2g/dL			
Pleural fluid				
TLC	200/mm3 (40% polymorph, 60% lymphocyte)	0–5		
Sugar	5.9mmol/L	2.3-4.6		
Protein	33.2g/L			
Drain Fluid				
TLC	7000/mm <sup>3</sup> (10% polymorph, 90% lymphocyte)	0–5		
Sugar	2.2mmol/L	2.3-4.6		
Protein	13.3g/L			
Amylase	>4000 Units			
ADA	214.0U/L	0–30		

isolated ductal rupture in the pancreatic head in blunt abdominal trauma can be tricky and it requires multiple investigations including blood reports, physical examinations, and radiological tests [7]. In addition, pancreatic trauma may frequently be overlooked or not readily appreciated during initial clinical examination and investigation. Serum levels of lipase and/or amylase have low sensitivity in such cases [8]. However, a raised pancreatic enzyme level in the background of blunt abdominal trauma should raise a suspicion of pancreatic injury. As the pancreatic enzymes were not measured initially at another center and the symptoms were very vague, this might be one of the factors for the delayed presentation in our case.

Identification of subtle radiologic findings and the use of multimodality imaging may be necessary for reaching a diagnosis, given the nonspecific clinical findings. As the diagnostic accuracy of CT is lowest for the detection of traumatic injury of the pancreas as well as the pancreatic duct, MRI/MRCP or even ERCP may be necessary to further define the parenchymal injuries, evaluate main pancreatic duct (MPD) integrity, and confirm the injury grade and identify the ductal rupture [6,9,10]. ERCP and MRCP can detect ductal injury as well as detect late complications [1,11]. Whenever there is a suspicion of pancreatic ascites, diagnostic paracentesis should be performed. Pancreatic ascites is characterized by an amylase level over 1000 IU/L and protein level greater than 3 g/dL, serum-ascites albumin gradient (SAAG) normally less than 1.1 g/dL, and lymphocytosis. These features distinguish pancreatic ascites from ascites secondary to portal hypertension [12].

The management of pancreatic injury depends on hemodynamic status, time to presentation, degree of pancreatic parenchymal injury, the status of MPD, and associated injuries. Ductal status is an important predictor of outcome in pancreatic trauma and is essential for establishing the basis for treatment decisions [8]. For hemodynamically stable patients, conservative treatment/non-operative management (NOM) is considered in low-grade injuries (grade I-II), and surgical treatment is mostly required for high-grade (grade III, IV, and V) pancreatic injuries due to blunt abdominal trauma [4]. Although NOM is a feasible option in most cases of low-grade pancreatic injuries without pancreatic duct injury and is increasingly reported for pancreatic trauma with ductal injury, the failure of this approach will require subsequent surgery or delayed surgical intervention due to initially missed main pancreatic duct injury leading to higher pancreas-specific mortality and many irremediable situations [10,13].

Although the Eastern Association for the Surgery of Trauma (EAST) guidelines recommend operative treatment for high-grade pancreatic trauma, NOM with the appropriate drainage may be a promising treatment for grade III or IV trauma, especially at facilities with expertise in interventional radiology and endoscopy [14]. Also, based on data in a systematic review it is suggested that early ERCP and ductal stenting may lead to the resolution of symptoms and healing of the injured duct in selected cases (30–100%), even for grade III injuries, thus avoiding major laparotomy and resection [15]. Endoscopic transpapillary stenting of the MPD promotes healing of duct disruptions by blocking the leaking duct and bridging the disruption or by ablating the pancreatic sphincter converting the high-pressure pancreatic duct system to a low-pressure system with the preferential flow to the duodenum [16].

If there is duct injury or late-onset complications, the placement of a transpapillary stent by means of ERCP or a nasopancreatic drain tube favors healing, especially if the placement is done in the early stages [17]. Delay in diagnosis and treatment for more than 24 hours causes an increase in complications in 1/3rd of cases such as pancreatic pseudocysts (PPC), abscesses, hemorrhage, fistulas, ductal stricture, or sepsis with multi-organ failure [2]. PPC may be discovered very late, and may even pose the diagnostic possibility of a cystic tumor of the pancreas. But PPC usually becomes evident within weeks following the traumatic incident with classic signs (gastric emptying disorder, pain, elevated lipase). Treatment decisions depend on whether or not the PPC is symptomatic, its size, and its position relative to other organs. A small PPC (<5 cm in diameter) without clinical signs of infection requires only simple monitoring especially when the patient is young [18]. In addition, pancreatic ascites is a rare complication of traumatic pancreatitis due to the peritoneal leakage from the disrupted pancreatic duct [12]. The management of pancreatic ascites can be medical or surgical depending on the wide etiology of the ascites. Generally, surgical therapy for pancreatic ascites is usually recommended early when the etiology is due to trauma [19]. Medical treatment includes withholding oral feedings, total parenteral nutrition, paracentesis, and administering octreotide [20]. Our patient was successfully managed conservatively as mentioned.

## 4. Conclusion

Pancreatic injuries are frequently overlooked as clinical signs are vague, laboratory tests and imaging modalities are not specific in the early period of injury, and require a high index of suspicion to diagnose and manage these injuries. As new-onset ascites can mislead delaying the diagnosis, especially in a low-resource country, emphasis on knowing the mechanism of injury and a multidisciplinary approach is essential for the diagnosis and management of pancreatic injuries.



Fig. 5. MRCP obtained later shows non-visualization of the pancreatic duct in pancreatic head along with pancreatic fluid collection (red circle). (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

#### Provenance and peer review

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#### Author contribution

Hari Sedai- Study concept, data collection and analysis and surgical treatment of the patient. Elisha Poddar- Study concept, data collection and analysis and surgical treatment of the patient. Suraj Shrestha- Study design, data analysis and writing the paper. Dinesh Koirala- Study design, data analysis and writing the paper. Abishkar Gautam- Study design, data analysis and writing the paper.

## **Registration of research studies**

- 1. Name of the registry:
  - 2. Unique Identifying number or registration ID:

3.Hyperlink to your specific registration (must be publicly accessible and will be checked):

#### Consent

Written informed consent was obtained from the patients for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

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## Declaration of competing of interest

None to declare.

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