

CASE REPORT

Pancreatico-Vesical Fistula as a Result of Severe Acute Necrotising Pancreatitis

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ABSTRACT

Context This is very rare and unreported complication of necrotising pancreatitis. **Case report** We report an unusual case of patient who developed pancreatic necrosis as a sequelae of severe acute pancreatitis extending in retroperitoneum up to pre and para-vesical space resulting in enzymatic auto-digestion of urinary bladder wall leading to pancreatico-vesical fistula. Patient excreted significant amount of infected pancreatic necrosis through urine. **Conclusion** Patient was treated successfully with pigtail catheter drainage of infected necrosis initially, followed later by endoscope assisted minimally invasive pancreatic necrosectomy along pigtail catheter tract via retroperitoneal approach along with prolonged bladder decompression.

INTRODUCTION

Twenty five year old male patient with severe acute necrotising pancreatitis was transferred to us. He was detected to have pancreaticovesical fistula as a result of complication of necrotising pancreatitis. This complication following pancreatitis is unheard of and is never reported in literature to the best of our knowledge. It was treated by drainage initially by image guided percutaneous catheter and later by endoscope assisted minimally invasive pancreatic necrosectomy along with prolonged bladder decompression with good result.

CASE REPORT

Twenty-five-year-old male patient was referred from a community hospital with history of severe acute pancreatitis since last 3 weeks after initial supportive and conservative management. Patient had history of sudden, severe aggravation of abdominal pain, distention of abdomen, recent onset inability to tolerate enteral feeds and mild to moderate fever.

On admission, patient was febrile (38°C), had pulse rate of 110/min, blood pressure of 110/80 mm of Hg and respiratory rate of 22/min. Abdomen had generalised distension, severe tenderness over left flank extending till hypogastrium and there was obvious fullness in pre-pubic area. Patient's Hb was 8 gm% and he had leucocytosis of 20,000/c mm. His liver and renal function biochemical

parameters were within normal range. Blood gas analysis was showing mild metabolic acidosis. Referral note by previous hospital mentioned presence of renal and respiratory organ failure which settled after one week.

Provisional diagnosis of infected pancreatic necrotic collection was made. Broad spectrum antibiotics were started. On per urethral catheterisation turbid brownish urine with some solid necrotic debris which eventually settled at the bottom of the urine bag was noticed (**Figures 1, 2**). On further enquiry, patient gave history of passing similar coloured urine with debris since last 2 days before which urine was normal in colour without any debris. Above finding raised suspicion of pancreatico-vesical fistula.

CECT was performed which showed partially walled off pancreatic necrosis extending from pancreatic bed in retroperitoneum to track behind the Gerota's fascia lifting up left kidney, retro-colic region reaching till pre and paravesical region which corresponded to fullness in pre-pubic region (**Figures 3, 4**). In view of evidence of infection (air bubbles) in necrosis image guided percutaneous pigtail catheter drainage of the same was performed by access through retroperitoneum behind lower pole of left kidney, using 10 Fr. catheter (**Figure 3**). It drained around 750 ml of thick brownish fluid of the same colour and characteristics as the urine, further supporting suspicion of pancreatico-vesical fistula.

Urine amylase levels were raised marginally. To demonstrate the fistula we instilled 50 ccs of methylene blue solution through the pigtail catheter which started appearing in urine within few minutes giving light blue-green colour to urine (**Figure 5**), thus proving the presence of pancreatico-vesical fistula. In view of inability of pigtail catheter to provide effective drainage to thick residual necrosis and complication of fistulisation with bladder,

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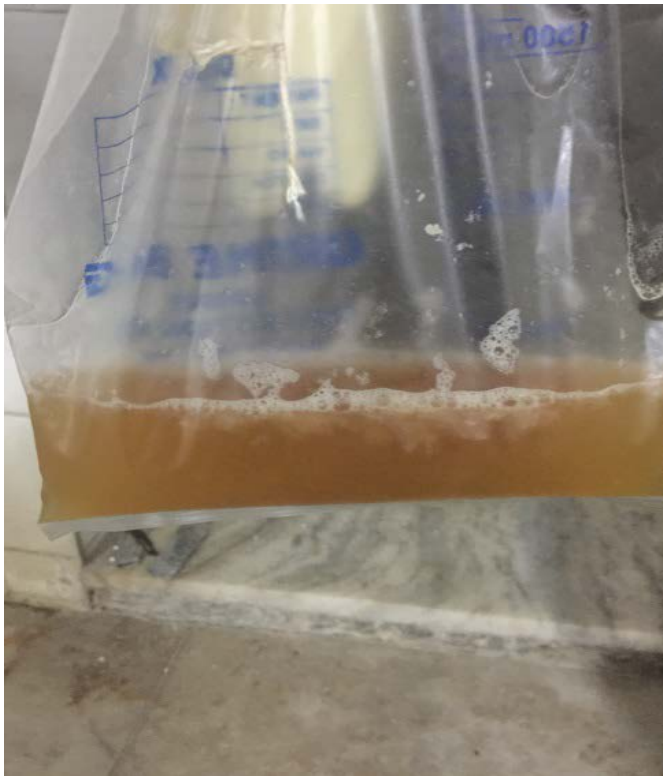


Figure 1. Urine bag showing turbid urine with necrotic debris due pancreatico-vesical fistula.

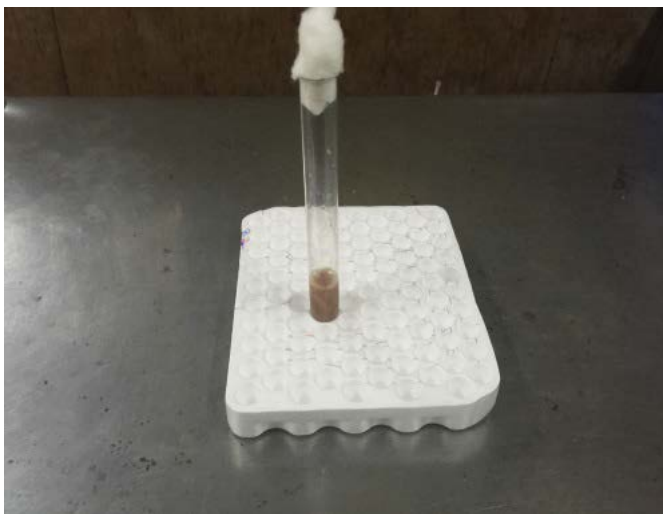


Figure 2. Showing turbid urine with pancreatic necrosis taken in a test tube.

patient was subjected to endoscope assisted minimally invasive pancreatic necrosectomy along the tract of pigtail catheter by retroperitoneal approach to drain the solid necrotic collection followed by sump drainage. Urinary bladder was kept decompressed for prolonged time. With this approach patient improved dramatically and recovered from sepsis. Necrotic collection resolved completely over 14 days and fistula closed spontaneously. Cystogram performed 8 weeks later did not show any extravasation.

DISCUSSION

Pancreatic necrosis is a frequent accompanant of severe acute pancreatitis, incidence being about one-third of the patients with severe acute attack [1]. Pathophysiologically,

necrosis occurs due to thrombosing vasculitis of small arteries, arterioles and capillaries [2]. Another important mechanism involved in pancreatic and peripancreatic tissue necrosis is premature pancreatic intra-acinar cell activation of digestive zymogens to their active forms like trypsinogen to trypsin which is mediated by lysosomal hydrolase enzyme Cathepsin B. Colocalisation hypothesis [3] explains Cathepsin B mediated premature zymogen activation wherein both the enzymes mix in the large cytoplasmic vacuoles in pancreatic acinar cells. These activated enzymes escape through membrane bound organelles in cytoplasm and degrade cell proteins. This process has been called “autodigestion”. These active enzymes initiate cascading vicious cycle of enzyme activation. Disruption of acini and ductules including the major pancreatic duct of Wirsung due to process of autodigestion causes further leakage of autodigestive enzymes in the necrotic collections and peripancreatic fluid collections. These enzymes enter peripancreatic tissues to bring about necrosis in surrounding tissues. Clinical spectrum of fate of this necrotic collection is very wide. It can remain sterile without clinically significant consequences [4]. Or it can get infected and produce local as well as systemic manifestations , sometimes leading to life threatening sepsis, multiorgan-dysfuncion syndrome and death [1,5]. If it persists for more than four weeks, It derives fibrous wall from surrounding retroperitoneal structures when it is called as “walled off pancreatic necrosis or WOPN” [5]. WOPN can remain sterile or might get infected. Source of infection of necrotic collections is from gastrointestinal tract as suggested by profile of organisms isolated from necrosis [1].

Extravasated pancreatic secretions and necrotic collections have rapid access to peripancreatic retroperitoneal space and the process of tracking of these necrotic collections is determined by the amount of collections and anatomy of retroperitoneal fascial planes. Peripancreatic necrotic collections track mostly on left side as most of the gland is situated on left of midline. It can spread over anterior surface of pancreas in anterior perirenal and

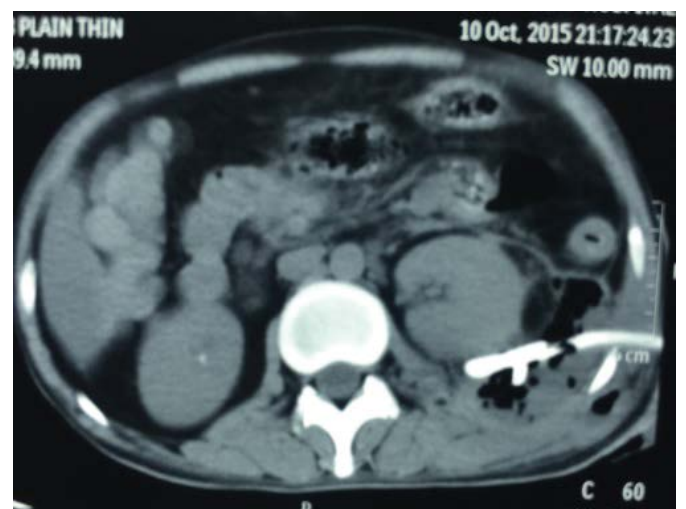


Figure 3. CECT showing pigtail catheter drainage of pancreatic necrotic collection tracking around the gerota's fascia.



Figure 4. Pancreatic necrosis tracking all the way in the retroperitoneum till pre and para vesical space with air specks within.



Figure 5. Change of urine colour to greenish blue due to leakage of methylene blue dye instilled in pancreatic necrosis cavity through pigtail catheter, thus proving presence of pancreatico-vesical fistula.

retromesenteric or intermesenteric and mesocolic plane. Superiorly it can extend in lesser sac till diaphragm and thorough the esophageal hiatus in posterior mediastinum. Inferiorly and laterally, these necrotic collections can have easy access to lateroconal fascial plane, retrorenal and retrocolic space and reach down to occupy pelvic peritoneal space. These retroperitoneal interfascial planes represent weak barriers to the spread of collections which assume the path of least resistance. Rarely this collection may even extend to scrotum through the inguinal canal [6]. Pelvic retroperitoneal space consist of lateral paravesical spaces , prevesical space and retro rectal space. It is continuous antero-superiorly with anterior pre-peritoneal space [7]. This pelvic retroperitoneal space contains extraperitoneal parts of urinary bladder and rectum.

Depending on the extent of spread of this peripancreatic necrotic collections in retroperitoneal interfascial planes,

Ishikawa classified severe acute pancreatitis in I to V grades and correlated extent to the severity of disease [8]. Grades were decided by pattern and extent of spread of necrotic collections and higher the grade more was the morbidity and mortality. Our patient with extension of necrotic collection till pelvis can be classified as Ishikawa grade IV.

Our patient was treated for initial 3 weeks at other hospital and as per referral note had prolonged (more than 48 hours) renal and respiratory organ failure; hence was classified as ‘severe acute pancreatitis as per Revised Atlanta Classification, [9]. Patient did not undergo any intervention in the form of drainage by percutaneous pigtail catheter at that hospital as described in step up approach. As a result, necrotic collections accumulated in large amount and spread in retroperitoneal fascial planes as described above to reach upto pelvic retroperitoneal space. It occupied pre and para-vesical space in the pelvis reaching laterally till lateral pelvic walls. This collection was in direct contact with extraperitoneal parts of urinary bladder where it caused injury to bladder wall by mechanism of “autodigestion” as described above leading to fistulisation of bladder with necrotic collection. Another less likely mechanism of fistulisation is ischemic necrosis of the bladder wall due to adjacent perivesical necrotic collection and sepsis with resultant thrombosis of bladder wall vasculature. This case emphasises the importance of timely intervention to drain necrotic collections which are large in amount to restrict their spread to distant areas and to avoid resultant local complications due to autodigestive process.

Various local complications of acute necrotising pancreatitis include internal or external fistulisation and pseudoaneurysm formation from splenic, gastroduodenal or pancreaticoduodenal arteries due to degradation of vessel wall due to pancreatic enzymes [10]. Leakage of autodigestive proteolytic enzymes from the disrupted pancreatic duct or from glandular parenchyma as a result of acute pancreatitis, is the factor largely responsible for its local complications [11]. It keeps on burrowing or eroding in retroperitoneum and surrounding organs by the process of ‘autodigestion’ and is known to develop fistula with peritoneum, pleura, colon, stomach or exterior [11]. Another important mechanism of formation of internal fistulae is ischemic necrosis of adjacent visceral wall due to shock and thrombosis [12]. At times mere large size of necrosis and fluid collections with severe compression can produce pressure necrosis and fistulisation [13]. Spontaneous drainage of necrosis through such fistulas internally in GI tract and passing per rectally, or to the exterior via pancreatico-cutaneous fistula is also known.

In our patient, pancreatico-vesical fistula provided effective egress route for excretion of necrotic collection and large amount of collection was excreted via urine. We adopted step up approach by draining necrotic collection through percutaneously placed pigtail catheter initially followed by minimally invasive endoscope assisted

retroperitoneal pancreatic necrosectomy along the pigtail catheter sinus tract to effectively drain the pelvic necrotic collection to halt autodigestive process and promote healing of bladder fistula. Effective and prompt drainage of pelvic necrotic collection alongwith bladder decompression was enough in this patient to heal fistula and formal surgical closure was not necessary.

One observation worth noting was development of pancreatiko-vesical fistula did not result in very severe life threatening sepsis and organ failure as compared to pancreatiko-colonic fistula where possibility septic complications and organ failures including mortality is very high (upto 30%)[14]. This can be explained by relative sterility of urine compared to colonic contents which is the source of infection of necrotic collections in colonic fistulas leading to life threatening sepsis.

To the best of our knowledge there is no reported case of pancreatic fistula with urinary bladder and necrosis being excreted in urine as was the case in our patient.

Common etiologies of vesical fistulas include postoperative like post hysterectomy, reversal of hartman's procedure, contiguous spread of disease in diverticulosis, crohn's colitis or advanced malignancies [15]. This is a unique case of vesical fistula secondary to necrotising pancreatitis.

Conflict of interest

The authors have no conflict of interest to declare.

References

1. Slavin J, Ghaneh P, Sutton R, Hartley M, Rowlands P, Garvey C, Hughes M, et al. Management of necrotising pancreatitis. *World J Gastroenterol* 2001; 7:476-81. [PMID: 11819813]
2. Nordback I, Lauslahti K. Clinical Pathology of acute necrotising pancreatitis. *J Clin Pathol* 1986; 39:68-74. [PMID: 3950033]
3. Steer ML. The early intra acinar cell events during acute pancreatitis: The Frank Book Memorial Lecture. *Pancreas* 1998; 17:31-37.
4. Warshaw AL. Pancreatic necrosis: to debride or not to debride-that is the question. *Ann Surg* 2000; 232:627-629. [PMID: 11066132]
5. Stamatakos M, Stefanaki C, Kontzoglou K, Stergiopoulos S, Giannopoulos G, Safioleas M. Walled Off Pancreatic Necrosis. *World J Gastroenterol* 2010; 16: 1707-1712. [PMID: 20380001]
6. Kalia S, Gupta R, Shenvi SD, Kumar H, Gupta R, Kang M, Rana SS, et al. Inguinoscrotal region as an unusual site of extra-pancreatic collections in infected pancreatic necrosis. *Gastroenterol Rep (Oxf)* 2015; 1-5. [PMID: 25649933]
7. Molmenti EP, Balfe DM, Kanterman RY, Bennett HF. Anatomy of the retroperitoneum: observations of the distribution of pathologic fluid collections. *Radiology* 1996; 200:95-103. [PMID: 8657951]
8. Ishikawa K, Idoguchi K, Tanaka H, Tohma Y, Ukai I, Watanabe H, Matsuoka T, Yokota J, et al. Classification of acute pancreatitis based on retroperitoneal extension: application of the concept of interfascial planes. *Eur J Radiol* 2006; 60:445-452. [PMID: 16891082]
9. Banks PA, Bollen TL, Dervenis C, Gooszen HG, Johnson CD, Sarr MG, Tsiotos GG, et al. Classification of acute pancreatitis--2012: revision of the Atlanta classification and definitions by international consensus. *Gut* 2013; 62:102-111. [PMID: 23100216]
10. Karakayali FY. Surgical and interventional management of complications caused by acute pancreatitis. *World J Gastroenterol* 2014; 20:13412-423. [PMID: 25309073]
11. Cameron JL, Kieffer RS, Anderson WJ, Zuidema GD. Internal pancreatic fistulas: pancreatic ascites and pleural effusions. *Ann Surg* 1976; 184:587-593. [PMID: 984927]
12. Ito K, Igarashi Y, Mimura T, Kishimoto Y, Kamata I, Kobayashi S, Yoshimoto K, et al. Acute pancreatitis with complicating colonic fistula successfully closed. *Case Rep Gastroenterol* 2013; 7:314-321. [PMID: 23904844]
13. Gardner A, Gardner G, Feller E. Severe colonic complications of pancreatic disease. *J Clin Gastroenterol* 2003; 37:258-62. [PMID: 12960726]
14. Gupta M, Dhar S, Dinesh BV, Lalwani S, Singla P, Yadav A, Kumaran V, et al. Colonic fistulae complicating severe acute necrotising pancreatitis. *The Ganga Ram Journal* 2011; 1:3-7.
15. Khanbhai M, Hodgson C, Mahmood K, Parker MC, Solkar M. Colo-vesical fistula: Complete healing without surgical intervention. *Int J Surg Case Rep* 2014; 5:448-450. [PMID: 24973524]