

CASE REPORT

Case of Severe Acute Necrotizing Pancreatitis with Abdominal Compartment Syndrome with Splenic Infarction

Prabhat Jha^{1*}, Raju Bhandari¹, Bijendra Dhoj Joshi²

¹Department of General Surgery, Alka Hospital Private Limited, Pulchowk, Lalitpur, Nepal

²Department of Surgery, Kathmandu Model Hospital, Nepal

ABSTRACT

Background Abdominal compartment syndrome and splenic complications are common in patients with acute severe infected necrotizing pancreatitis. These conditions increase the morbidity and mortality in patients with acute severe pancreatitis. **Case presentation** this case report describes a patient with acute severe infected necrotizing pancreatitis who developed abdominal compartment syndrome and diffuse splenic infarction. Patient was managed with laparotomy with sequential debridement and Bogota bag closure and splenectomy. **Conclusion** Abdominal compartment syndrome is a life threatening condition occurring in patients with severe acute pancreatitis. Laparotomy and decompression can be lifesaving in these patients. Besides, splenic infarction can also occur in these patients. Splenectomy should be done in patients with diffuse splenic infarction.

INTRODUCTION

The morbidity and mortality of patients with Severe Acute Pancreatitis (SAP) remain high despite significant improvement in treatment due to the better understanding of the pathophysiology of disease, early aggressive fluid resuscitation, timely surgical intervention, permanent monitoring, and organ supporting intensive care. The major predictors of unfavorable outcome are development of persistent organ failure and presence of bacterial infection of necrosis [1].

Abdominal Compartment Syndrome (ACS) in patients with SAP is a sign of severe disease with high risk of adverse outcomes [2, 3, 4, 5]. ACS is defined as a state of serious organ dysfunction resulting from sustained increase in intra-abdominal pressure [6]. Very recently published systematic review on this topic showed that the mortality rate in patients who developed ACS during SAP was 49%, while it was 11% in patients without this complication [7].

The development of organ failure in SAP is in correlation with presence of Intra-Abdominal Hypertension (IAH) [3, 4, 8, 9]. Measurement of Intra-Abdominal Pressure (IAP)

is recommended in all patients in intensive care unit as it predicts mortality [6]. The management of ACS during SAP is controversial and there are no definite guidelines. The guidelines and definitions published by the World Society of Abdominal Compartment Syndrome (WSACS) are general for ACS due to any cause and it is not known if these can be applied to patients with SAP. The treatment, indications, timing and interventional techniques for the treatment of ACS during pancreatitis remains unresolved [3, 10, 11, 12, 13].

Pancreas is closely related with splenic artery, splenic vein and spleen. So, splenic complications are frequently seen in patients with acute pancreatitis [14]. Splenic infarction is rare in inflammatory diseases of the pancreas [15]. Most reported cases are of focal infarction, and treatment is mostly conservative but cases with diffuse infarction have been reported [15]. Diffuse splenic infarction is a rare complication of severe acute pancreatitis. Diagnosis is straight forward but management can vary from conservative to surgical. Conservative treatment should be considered as the first line of management but surgical treatment might be needed if spleen cannot be salvaged.

CASE PRESENTATION

A 35 year old male presented with complaints of pain abdomen and vomiting multiple episodes for one day. On investigations he had increased total white blood cell counts, serum amylase and serum lipase level. Patient had history of laparotomy for hollow viscus perforation three years back. Besides he was admitted one year back with diagnosis of acute alcoholic pancreatitis. Patient is a regular alcohol consumer. With a diagnosis of acute mild alcoholic pancreatitis patient was admitted. The patient was initially

Received December 13th, 2019 - Accepted February 20th, 2020

Keywords Pancreatitis; Abdominal Compartment Syndrome; Splenic Infarction

Abbreviations ACS Abdominal Compartment Syndrome; SAP Severe Acute Pancreatitis; IAP Intraabdominal Pressure; IAH Intra-Abdominal Hypertension; WSACS World Society of Abdominal Compartment Syndrome; ICU Intensive Care Unit; VAC Vacuum Assisted Closure

Correspondence Prabhat Jha

General Surgery, Alka Hospital Private Limited,
Pulchowk, Lalitpur, Nepal

Tel +977-9851208714

Fax +00977-1-5547196

E-mail prabhatkaushaljha@gmail.com

managed with intravenous fluid administration, feeding as tolerable and analgesics.

On third day of admission he was transferred to ICU because of decreased oxygen saturation and decreased urine output. He was intubated. His urinary bladder pressure was persistently high. Urine output was nil and patient was started on hemodialysis with sedation and neuromuscular blockade. USG abdomen showed moderate ascites and one litre of fluid was drained. Patient was on maximum noradrenaline support but was not maintaining blood pressure. After twelve hours of transfer to ICU his bladder pressure increased to 45 cm H₂O. So he was planned for laparotomy. Midline laparotomy extending from xiphisternum up to pubic symphysis was done. At laparotomy bowel loops was grossly edematous, marked peripancreatic inflammation with greater omentum necrosis with approximately 2.5 L of hemorrhagic black fluid was present in peritoneal cavity. Pancreas was grossly swollen. Fluid was drained, omentectomy was done, drains were placed in the lesser sac, right and left paracolic gutters and pelvis. Abdomen was closed over a Bogota bag (urobag) (**Figure 1**). Patient was kept in ICU under sedation and neuromuscular blockade. Dialysis was continued daily. Continuous irrigation of pancreatic bed was done with normal saline through drains kept in pancreatic bed. Nutrition was maintained with total parenteral nutrition. Patient was started on imipenem-cilastatin, Polymixin-B and Tigecycline were added later during the course of admission. Patient was extubated on post-operative day. Dialysis was continued for seven days.

On 7th post-operative day debridement of necrotic pancreatic tail up to fresh bleeding with Bogota bag exchange was done. On 10th post-operative day patient had fecal discharge from the drain. Per operatively jejunal perforation 0.5 × 0.5 cm and 2 × 2 cm approximately 120 cm and 140 cm distal to duodenojejunal flexure was noted (**Figure 2**). Resection and anastomosis with peritoneal lavage with necrosectomy with Bogota bag change was performed. On 17th post-operative day laparotomy with partial pancreatic necrosectomy and Bogota bag change was done (**Figure 3**). Anastomotic site was intact. On 26th postoperative day necrosectomy was performed. Per operatively approximately 200 mL pus with necrotic pancreas and greater omentum was present.

On 36th post-operative day patient underwent laparotomy due to increased drain output and increasing counts with fever. Per operative findings revealed pancreatic necrosis with diffuse splenic infarction with necrotic omentum. Spleen wasn't salvageable and it was considered to be the source of infection along with pancreatic necrosis. Splenectomy with pancreatic necrosectomy was done (**Figure 4**). Splenic infarction wasn't suspected preoperatively and it was detected during laparotomy only. He was vaccinated with pneumococcal, meningococcal and H. influenza vaccines. Patient was started on aspirin following splenectomy due to rebound thrombocytosis. Besides octreotide was

used for 10 days to decrease drain output. On 55th post-operative day patient underwent final debridement with tension suturing. Primary closure of wound was possible. Patient underwent re-exploration six times following the index operation.

Patient was discharged with drain in splenic fossa after 79 days of admission. Tension sutures and drain were removed on the follow up visit.



Figure 1. Laparotomy wound closed over a Bogota Bag.



Figure 2. Bowel perforation.



Figure 3. Necrotic pancreas.



Figure 4. Necrotic spleen.

DISCUSSION

Abdominal compartment syndrome and necrotizing pancreatitis

IAP during SAP occurs due to visceral edema, ascites, acute peri-pancreatic fluid collections, paralytic ileus, duodenal obstruction causing gastric dilatations, aggressive fluid retention, capillary leakage due to intra-abdominal inflammation, and decreased abdominal wall compliance due to edema. Our patient also had grossly dilated bowel loops, ascites, and peri-pancreatic fluid collection.

Respiratory failure occurs in IAH because the diaphragm is pushed up causing reduced chest wall compliance. Besides there is decreased venous return causing circulatory failure. Decreased renal perfusion contributes to oliguria and renal failure. Also there is decreased perfusion to the pancreas contributing to pancreatic necrosis [8, 16].

For the management of ACS regular monitoring of bladder pressure, ICU management, neuromuscular blockage with artificial respiration, pain relief, and urine output monitoring is important. Besides, nasogastric/colonic decompression should be done. Haemodialysis, diuretics and haemodialysis are also helpful. Our patient was managed in Intensive Care Unit with vasopressor support noradrenaline, mechanical ventilator support and haemodialysis for decreased blood pressure, decreased oxygen saturation and acute renal failure respectively. Besides six hourly bladder pressure monitoring, nasogastric decompression was done.

Interventional measures such as percutaneous catheter drainage under radiological guidance can be used if conservative measures fail [17, 18, 19]. Drainage of ascitic fluid under ultrasound guidance was done in our patient.

According to WSACS guidelines, in patients with persisting ACS despite percutaneous procedure performed, decompressive laparotomy with temporary abdominal

closure is recommended [6]. Early debridement is associated with a high incidence of retroperitoneal bleeding [8]. Different surgical decompressive methods such as full-thickness midline laparotomy, full-thickness transverse subcostal bilateral laparotomy, and subcutaneous linea Alba fasciotomy can be performed. Bogota bag and Vacuum Assisted Closure (VAC) can be used for temporary abdominal closure [10].

Our patient underwent laparotomy with Bogota bag closure. No attempt was made for debridement of necrotic pancreatic tissue in the first setting. Debridement was done in each setting till fresh bleeding.

Splenic infarction and necrotizing pancreatitis

The close anatomic relationship between pancreas, splenic vessels and spleen predisposes to different splenic complications during the course of both acute and chronic pancreatitis [20, 21, 22, 23, 24].

In a series of 159 CT scans performed on 100 consecutive patients with acute pancreatitis, Heider et al. found splenic infarcts in 10 patients and sub-capsular haemorrhage in two patients. In another series of 238 patients with pancreatic pseudocysts, 14(5.9%) patients had splenic parenchymal involvement [25]. Most reported cases are of focal infarction, and treatment is mostly conservative. Ray et al. reported a case of diffuse splenic infarction in a 17 year old boy with severe acute pancreatitis who presented with massive upper gastrointestinal bleeding and was treated with splenectomy [26]. A study reported two cases of acute pancreatitis with splenic complications. In the first case narrowing of splenic artery due to acute pancreatitis was suspected and in second cases hypercoagulability due to pancreatitis was suspected. Both cases were managed conservatively with serial CT follow up. Close monitoring of clinical parameters and serial CT scans is necessary for diagnosis of splenic complications. Our patient had diffuse splenic infarction and underwent splenectomy.

CONCLUSION

In conclusion abdominal compartment syndrome and diffuse splenic infarction can occur in patients with severe acute necrotizing pancreatitis. Early recognition and management may help in increasing the chances of survival of patient.

Acknowledgement

The authors would like to thank the patient and patient party who stood tough and gave permission for the publication of this case report.

Conflicts of Interest

All named authors hereby declare that they have no conflicts of interest to disclose.

REFERENCES

1. Working Group IAP/APA Acute Pancreatitis Guidelines, "IAP/APA evidence-based guidelines for the management of acute pancreatitis," *Pancreatology* 2013; 13:e1-e15. [PMID: 24054878]
2. Gecelter G, Fahoum B, Gardezi S, Schein M. Abdominal compartment syndrome in severe acute pancreatitis: an indication for a decompressing laparotomy? *Dig Surg* 2002; 19:402-404. [PMID: 12435913]
3. Chen H, Li F, Sun JB, Jia JG. Abdominal compartment syndrome in patients with severe acute pancreatitis in early stage. *World J Gastroenterol* 2008; 14:3541-3548. [PMID: 18567084]
4. Al-Bahrani AZ, Abid GH, Holt A, McCloy RF, Benson J, Eddleston J, et al. Clinical relevance of intra-abdominal hypertension in patients with severe acute pancreatitis. *Pancreas* 2008; 36:39-43. [PMID: 18192879]
5. Dambraszkas Z, Parseliunas A, Gulbinas A, Pundzius J, Barauskas G. Early recognition of abdominal compartment syndrome in patients with acute pancreatitis. *World J Gastroenterol* 2009; 15:717-721. [PMID: 19222096]
6. Kirkpatrick AW, Roberts DJ, De Waele J, Jaeschke R, Malbrain ML, De Keulenaer B, et al. Intra-abdominal hypertension and the abdominal compartment syndrome: updated consensus definitions and clinical practice guidelines from the World Society of the Abdominal Compartment Syndrome. *Intensive Care Med* 2013; 39:1190-1206. [PMID: 23673399]
7. van Brunschot S, Schut AJ, Bouwense SA, Besselink MG, Bakker OJ, van Goor H, et al. Abdominal compartment syndrome in acute pancreatitis: a systematic review. *Pancreas* 2014; 43:665-674. [PMID: 24921201]
8. Keskinen P, Leppaniemi A, Pettila V, Piilonen A, Kemppainen E, Hynninen M. Intra-abdominal pressure in severe acute pancreatitis. *World J Emerg Surg* 2007; 2.
9. Mentula P, Hienonen P, Kemppainen E, Puolakkainen P, Leppaniemi A. Surgical decompression for abdominal compartment syndrome in severe acute pancreatitis. *Arch Surg* 2010; 145:764-769. [PMID: 20713929]
10. Deng ZG, Zhou JY, Yin ZY, Peng YY, Wang FQ, Wang XM. Continuous regional arterial infusion and laparotomic decompression for severe acute pancreatitis with abdominal compartment syndrome. *World J Gastroenterol* 2011; 17:4911-4916. [PMID: 22171133]
11. Davis PJ, Eltawil KM, Abu-Wasel B, Walsh MJ, Topp T, Molinari M. Effect of obesity and decompressive laparotomy on mortality in acute pancreatitis requiring intensive care unit admission. *World J Surg* 2013; 37:318-332. [PMID: 23052814]
12. Boone B, Zureikat A, Hughes SJ, Moser AJ, Yadav D, Zeh HJ, et al. Abdominal compartment syndrome is an early, lethal complication of acute pancreatitis. *Am Surg* 2013; 79: 601-607. [PMID: 23711270]
13. Malbrain ML, Chiumello D, Pelosi P, Bihari D, Innes R, Ranieri VM, et al. Incidence and prognosis of intraabdominal hypertension in a mixed population of critically ill patients: a multiple-center epidemiological study. *Crit Care Med* 2005; 33:315-322. [PMID: 15699833]
14. Arenal Vera JJ, Said A, Guerro JA, Otero M, Gil I. Splenic infarction secondary to acute pancreatitis. *Rev Esp Enferm Dig* 2008; 100:300-303. [PMID: 18662085]
- 15.
16. Ray S, Mridha AR, Ahammed M. Diffuse splenic infarction in a case of severe acute pancreatitis. *Am J Surg* 2011; 201:e23-e25. [PMID: 21367360]
17. Tao HQ, Zhang JX, Zou SC. Clinical characteristics and management of patients with early acute severe pancreatitis: experience from a medical centre in China. *World J Gastroenterol* 2004; 10:919-921. [PMID: 15040047]
18. Sun ZX, Huang HR, Zhou H. Indwelling catheter and conservative measures in the treatment of abdominal compartment syndrome in fulminant acute pancreatitis. *World J Gastroenterol* 2006; 12:5068-5070. [PMID: 16937509]
19. Reed SF, Britt RC, Collins J, Weireter L, Cole F, Britt LD. Aggressive surveillance and early catheter-directed therapy in the management of intra-abdominal hypertension. *J Trauma* 2006; 61:1359-1363. [PMID: 17159677]
20. Reckard JM, Chung MH, Varma MK, Zagorski SM. Management of intraabdominal hypertension by percutaneous catheter drainage. *J Vasc Interv Radiol* 2005; 16:1019-1021. [PMID: 16002511]
21. Siu TL. Percutaneous drainage of spontaneous subcapsular haematoma of the spleen complicating chronic pancreatitis. *Surgeon* 2004; 2:52-55. [PMID: 15570808]
22. Thompson JE Jr, Ashley SW. Subcapsular hematoma of the spleen associated with acute pancreatitis. *Surgery* 1997; 121:231-233. [PMID: 9037239]
23. Mortelet KJ, Mergo PJ, Taylor HM, Ernst MD, Ros PR. Splenic and perisplenic involvement in acute pancreatitis: determination of prevalence and morphologic helical CT features. *J Comput Assist Tomogr* 2001; 25:50-54. [PMID: 11176293]
24. Lankisch PG. The spleen in inflammatory pancreatic disease. *Gastroenterol* 1990; 98:509-516.
25. Hastings OM, Jain KM, Khademi M, Lazaro EJ. Intrasplenic pancreatic pseudocyst complicating severe acute pancreatitis. *Am J Gastroenterol* 1978; 69:182-186. [PMID: 665640]
26. Heider R, Behrns KE. Pancreatic pseudocysts complicated by splenic parenchymal involvement: results of operative and percutaneous management. *Pancreas* 2001; 23:20-25. [PMID: 11451143]
27. Ray S, Mridha AR, Ahammed M. Diffuse splenic infarction in a case of severe acute pancreatitis. *Am J Surg* 2011; 201:e23-e25. [PMID: 21367360]