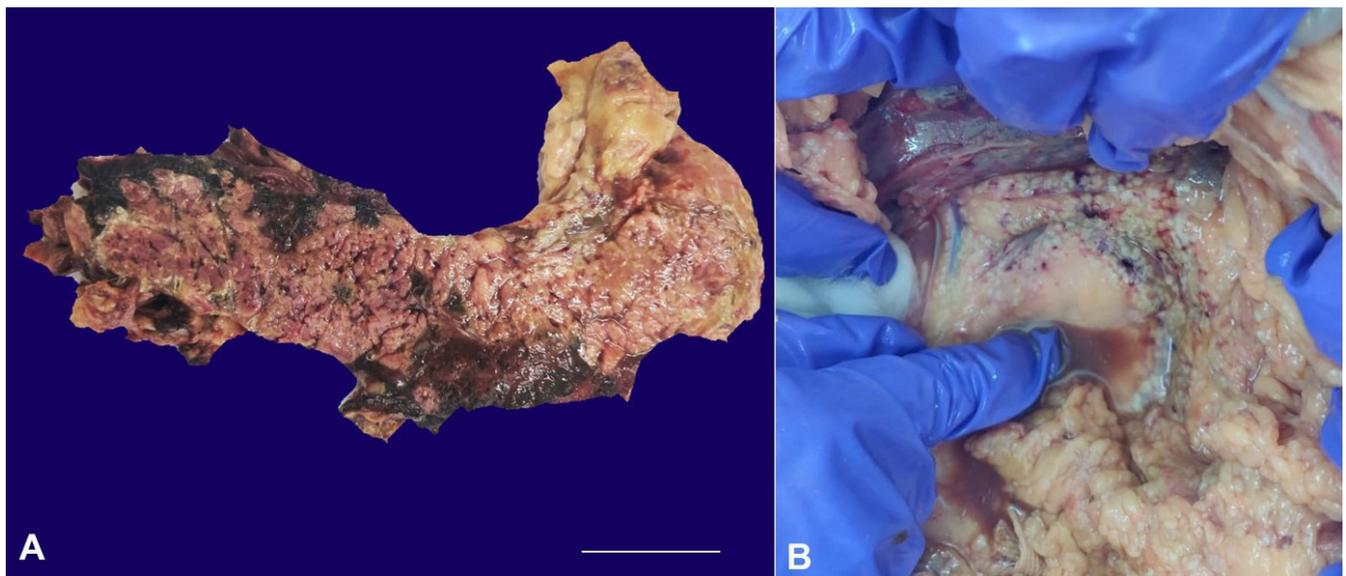


## Acute necrotizing pancreatitis

Deepika Phogat<sup>1</sup> , Mukul Bajpai<sup>1</sup> , Pranshu Agrawal<sup>1</sup> , Prosenjit Ganguli<sup>2</sup> 

How to cite: Phogat D, Bajpai M, Agrawal P, Ganguli P. Acute necrotizing pancreatitis. *Autops Case Rep* [Internet]. 2020; 10(4):e2020215. <https://doi.org/10.4322/acr.2020.215>



**Figure 1.** Gross view of the pancreas and peritoneal fat tissue. **A** – Cut surface of pancreas showing extensive parenchymal necrosis accompanied by hemorrhage within the gland; **B** – Foci of fat necrosis in the form of small white chalky deposits in the peritoneal cavity along brown tinged fluid with globules of fat.

Acute pancreatitis (AP) is an inflammatory condition of the pancreas that results from inappropriate intrapancreatic activation of enzymes like trypsin with downstream activation of other proenzymes into the interstitial space (rather than the acinar lumen) that causes the autodigestion of the pancreatic parenchyma, fat tissue and damages elastic fibres of blood vessels along with a marked inflammatory response.<sup>1</sup> The most common etiological factors are alcohol misuse, cholelithiasis, drugs, trauma, hyperlipidemia and post retrograde cholangiopancreatography.<sup>1,2</sup> Smoking and diabetes mellitus seem to increase the risk for acute pancreatitis.<sup>3,4</sup> The incidence of AP ranges between

13 to 45 per 100,000 people.<sup>5</sup> Chronic alcohol consumption results in the secretion of protein-rich pancreatic fluid leading to the deposition of protein plugs and obstruction of the pancreatic ducts besides the direct effects of alcohol on the pancreatic acinar and stellate pancreatic cells. In the biliary pancreatitis, the toxic effect of bile acids within the acinar cells increase intra-acinar calcium concentration triggering the inflammatory response.<sup>1,6</sup> Histologically, acute pancreatitis is classified as interstitial edematous and necrotizing subtypes.<sup>7</sup> The necrotizing form is associated with high mortality.<sup>8</sup> Among the grading scores of the severity of acute pancreatitis, like the

<sup>1</sup> 151 Base Hospital, Department of Lab Sciences, Guwahati, Assam, India

<sup>2</sup> Command Hospital Eastern Command, Department of Lab Sciences, Kolkata, West Bengal, India

Ranson Score (that is based on the clinical and lab investigation parameters), those based upon the computed tomographic imaging findings, like the Revised Atlanta Classification Score are more accurate with >90% accuracy.<sup>7,9</sup>

The above picture refers to the case of a 33-year-old male, known alcohol abuser, presented with sudden onset acute epigastric pain over the last six hours. His laboratory investigations revealed serum lipase level of 3412 U/L (RR:73-393IU/L); serum amylase level of 358 IU/L (RR: 85-115IU/L), serum calcium of 6.1 mg/dl (RR:8.5-10mg/dl), blood urea of 66mg/dL (RR;10-50mg/dl) and creatinine of 2.5mg/dL (RR;0.8-1.0mg/dl). The abdominal computed tomography revealed features of acute pancreatitis with the Revised Atlanta Classification score of 8/10. Despite aggressive management, the patient developed features of Systemic Inflammatory Response Syndrome and succumbed to his condition. On Autopsy, the pancreas was grossly enlarged and edematous with loss of surface lobulations, weighing 500gm (mean RR; 110 g). The cut surface showed extensive parenchymal necrosis accompanied by hemorrhage intermingling the substance of the gland (Figure 1A). Foci of fat necrosis in the form of small white chalky deposits were present in the peritoneal cavity adjacent to the pancreas (Figure 1B). Histopathological examination revealed areas of parenchymal necrosis surrounded by foci of shadowy outlines of necrotic fat cells with basophilic calcium deposits. Microbiological analysis of the necrotic pancreatic tissue did not reveal the presence of any bacterial organisms.

Severe acute pancreatitis is associated with an overall mortality of 30%, with 5% deaths occurring within the first week of illness.<sup>10,11</sup> The laboratory findings include marked elevation of serum amylase levels during the first 24 hours, followed by a rising serum lipase level by 72 to 96 hours after the beginning of the attack. Hypocalcemia may occur from precipitation of calcium soaps in necrotic fat. Direct visualization of enlarged, inflamed pancreas by CT scanning is useful.

The key to the acute pancreatitis management is resting the pancreas by total restriction of oral intake and by supportive therapy with intravenous fluids and analgesics. Acute respiratory distress syndrome and acute renal tubular necrosis are ominous complications. In 40-60% of patients with acute necrotizing pancreatitis, the necrotic debris becomes

infected, usually by gram-negative organisms from the alimentary tract, further complicating the clinical course. Infected Necrosis has a mortality of 100%.<sup>12,13</sup> Systemic organ failure and necrosis in the pancreas are both poor prognostic findings.

## ACKNOWLEDGEMENTS

The authors would like to acknowledge the lab technicians and support staff for their timely effort, sincerity, and dedication towards the work.

## REFERENCES

1. Lankisch PG, Apte M, Banks PA. Acute pancreatitis. *Lancet*. 2015;386(9988):85-96. [http://dx.doi.org/10.1016/S0140-6736\(14\)60649-8](http://dx.doi.org/10.1016/S0140-6736(14)60649-8). PMID:25616312.
2. Dumonceau JM, Andriulli A, Deviere J, et al. European Society of Gastrointestinal Endoscopy (ESGE) Guideline: prophylaxis of post-ERCP pancreatitis. *Endoscopy*. 2010;42(6):503-15. <http://dx.doi.org/10.1055/s-0029-1244208>. PMID:20506068.
3. Sadr-Azodi O, Andrén-Sandberg Å, Orsini N, Wolk A. Cigarette smoking, smoking cessation and acute pancreatitis: a prospective population-based study. *Gut*. 2012;61(2):262-7. <http://dx.doi.org/10.1136/gutjnl-2011-300566>. PMID:21836026.
4. Girman CJ, Kou TD, Cai B, et al. Patients with type 2 diabetes mellitus have higher risk for acute pancreatitis compared with those without diabetes. *Diabetes Obes Metab*. 2010;12(9):766-71. <http://dx.doi.org/10.1111/j.1463-1326.2010.01231.x>. PMID:20649628.
5. Yadav D, Lowenfels AB. The epidemiology of pancreatitis and pancreatic cancer. *Gastroenterology*. 2013;144(6):1252-61. <http://dx.doi.org/10.1053/j.gastro.2013.01.068>. PMID:23622135.
6. Perides G, van Acker GJ, Laukkarinen JM, Steer ML. Experimental acute biliary pancreatitis induced by retrograde infusion of bile acids into the mouse pancreatic duct. *Nat Protoc*. 2010;5(2):335-41. <http://dx.doi.org/10.1038/nprot.2009.243>. PMID:20134432.
7. Bradley EL 3rd. A clinically based classification system for acute pancreatitis: summary of the International Symposium on Acute Pancreatitis, Atlanta, Ga, September 11 through 13, 1992. *Arch Surg*. 1993;128(5):586-90. <http://dx.doi.org/10.1001/archsurg.1993.01420170122019>. PMID:8489394.
8. Beger HG, Rau B, Mayer J, Pralle U. Natural course of acute pancreatitis. *World J Surg*. 1997;21(2):130-

5. <http://dx.doi.org/10.1007/s002689900204>. PMID:8995067.
9. Balthazar EJ, Freeny PC, van Sonnenberg E. Imaging and intervention in acute pancreatitis. *Radiology*. 1994;193(2):297-306. <http://dx.doi.org/10.1148/radiology.193.2.7972730>. PMID:7972730.
10. Kumar V, Abbas A, Aster J, Robbins S, Cotran R, Perkins, J. Robbins & Cotran pathologic basis of disease. London: Elsevier Health Sciences; 2015.
11. Imrie CW. Underdiagnosis of acute pancreatitis. *Adv Acute Pancreat*. 1997;1:3-5.
12. Banks PA. Infected necrosis: morbidity and therapeutic consequences. *Hepatogastroenterology*. 1991;38(2):116-9. PMID:1855766.
13. Baron TH, Morgan D. Acute necrotizing pancreatitis. *N Engl J Med*. 1999;340(18):1412-7. <http://dx.doi.org/10.1056/NEJM199905063401807>. PMID:10228193.

This work was carried out at 151 Base Hospital, Department of Lab Sciences, Guwahati, India.

**Authors' contributions:** All authors equally contributed to the manuscript conception, collectively proofread the final version and approved it for publication.

**Ethics statement:** The authors retain informed consent signed by the deceased's next-of-kin authorizing the data publication.

**Conflict of interest:** none.

**Financial support:** none.

**Submitted on:** July 22<sup>nd</sup>, 2020

**Accepted on:** July 24<sup>th</sup>, 2020

### Correspondence

Deepika Phogat  
151 Base Hospital, Department of Lab Sciences  
Kamrup -781029, Guwahati, Assam, India  
Phone: +91 (84) 3690-1520  
[deepika.phogat@gmail.com](mailto:deepika.phogat@gmail.com)