An Unusual Presentation of Fatal Acute Haemorrhagic Pancreatitis in a Young Adult

Naveen A, Sahu MR, Padhi KS, Sharma M, Sahu SS, Mohanty MK

Department of Forensic Medicine and Toxicology

All India Institute of Medical Sciences (AIIMS),

Bhubaneswar, India.

Corresponding Author

Manas Ranjan Sahu

Department of Forensic Medicine and Toxicology,

All India Institute of Medical Sciences (AIIMS),

Bhubaneswar, India.

E-mail: fmt_manas@aiimsbhubaneswar.edu.in

Citation

Naveen A, Sahu MR, Padhi KS, Sharma M, Sahu SS, Mohanty MK. An Unusual Presentation of Fatal Acute Haemorrhagic Pancreatitis in a Young Adult. 2024;86(2):241-4.

ABSTRACT

Acute haemorrhagic pancreatitis is a medical emergency and the most severe form of Acute Pancreatitis. It is characterized by severe epigastric pain that radiates to the back and is associated with vomiting. If not diagnosed and managed promptly, it may result in sudden, unexpected, unexplained deaths which fall within the medicolegal domain. In such cases, the role of an autopsy is of paramount importance to determine the cause of death. Here we report a young adult, who presented to the local hospital with vague abdominal discomfort and vomiting following alcohol intake and referred to our tertiary care center for further management. But he was received dead on arrival at our hospital. The diagnosis of acute haemorrhagic pancreatitis was made only after the post mortem examination. Awareness of the physicians about the unusual symptoms in acute haemorrhagic pancreatitis and the need for pancreas examination at autopsy of all sudden deaths is emphasised.

KEY WORDS

Acute pancreatitis, Autopsy, Autolysis, Haemorrhagic pancreatitis, Sudden death, Young adult

INTRODUCTION

Acute Pancreatitis (AP) is a medical emergency. It occurs predominantly in males of the 30–40 years age group.[1,2] Gall stone and alcoholism are reported to be the major causative factors of AP.[3] Though AP is a self-limiting disorder of the pancreas in the majority (80%) cases, fatal in the remaining (20%) cases.[4]

Acute haemorrhagic pancreatitis (AHP) is the most severe form of AP that occurs in 1.2 to 14.5% of AP. The haemorrhage occurs as a consequence of digestion of the surrounding vessels by released pancreatic enzymes following necrosis. Splenic artery, portal vein, spleen, and unspecified peripancreatic vessels are the major source of bleeding.[5] Clinically, Abdomen pain is the cardinal symptom, which is characterized by acute onset of persistent and severe epigastric pain, often radiating to the back, and associated with vomiting.[6]

Timely diagnosis of AHP is crucial in determining morbidity and mortality. At times, the patient may present clinically with nonspecific or mild symptoms.[7] This could prevent a physician to make a possible diagnosis of impending AHP on time. This missed or delayed diagnosis often results in sudden, unexpected, unexplained deaths which fall within the medicolegal domain. Moreover, establishing the cause of death in such cases may pose a challenge for autopsy surgeons due to various reasons. Firstly, deaths due to AHP are very rarely encountered in routine autopsy practice. Secondly, the examination of the pancreas is often overlooked or missed because of its retroperitoneal location. Thirdly, post mortem autolysis of the pancreas.

Here we present a case of a young adult, who presented to the local hospital with non-specific abdominal symptoms and was later referred to our tertiary care center for further management. However, he was received dead on arrival at the ED. The diagnosis of acute haemorrhagic pancreatitis was established only at the time of autopsy.

CASE REPORT

Mr. X was a 32-year-old male, presented to the local hospital with symptoms of nausea, vomiting, and vague abdominal discomfort following ingestion of alcohol at his home in the evening. His medical and surgical history was unremarkable except for chronic alcoholism for 8 years. He refrained from drinking alcohol for the last 6 months following admission at the local hospital for severe gastritis. However, he started drinking again last week.

He was treated symptomatically with antacids and antiemetics. However, his symptoms were not subsided. Therefore, he was referred to this tertiary care center for further management. Unfortunately, he was received dead on arrival at the ED. The body was referred for the post mortem examination because of its sudden, unexpected, and unexplained nature of death.

The post mortem examination was conducted after 18 hours of death. On external examination, no injuries were found all over the body. Rigor mortis was developed all over the body. Post-mortem lividity was present over dependent parts of the back except over bony prominences. All the natural orifices were intact and free from discharge. The face was congested. On internal examination, the brain was intact and congested, no signs of haemorrhage were present. The heart was intact and the weight was 382 g. Multiple petechial haemorrhages were present over the posterior surface of the left atrium. All coronaries were patent and the left ventricular wall thickness was 1.6 cm. Both lungs were intact, congested, and oedematous.

On opening the abdomen, about 800 ml of blood was found inside the retroperitoneal cavity. A thorough examination was conducted to locate the source of haemorrhage after draining the blood. The pancreas was oedematous and haemorrhagic throughout its external surface (Fig. 1). After removal and washing, the weight was 220 g. The pancreas was fixed in 10% formalin for histopathological examination (HPE). On serial sections, extensive haemorrhage was present over the entire parenchyma of the pancreas from head to tail parts of the pancreas (Fig. 2). Microscopy findings of from the pancreas revealed autolysis.

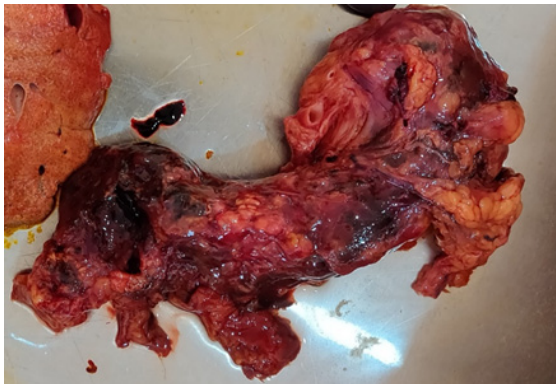

Figure 1. Gross examination of pancreas at autopsy shows diffuse haemorrhage along with focal areas of fat necrosis.

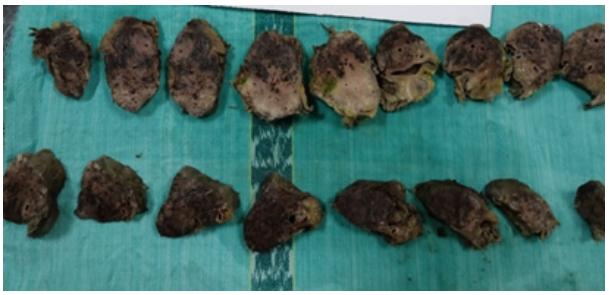

Figure 2. Serial transverse sections of pancreas show extensive hemorrhage that spread across the gland from head to tail.

The liver was intact and enlarged. Weight: 1880 g. The surface was soft, greasy, and pale yellow. On microscopy, multiple micro and macro vesicular fatty changes were observed. The gall bladder was intact and contained bile. There was no gall stone found in the gall bladder and biliary tract. Both kidneys were congested and the cortico-medullary junction was indistinct. The stomach was intact, contains about 100 ml of yellowish fluid which emitted a faint fruity odour. The stomach mucosa was congested in places. The oesophagus was explored till the lower end to look for any lesions including varices but no abnormality was detected. The trachea was intact and no findings suggestive of aspiration were present.

Toxicological analysis of blood was positive for ethyl alcohol. In consideration of the history, inquest paper, and autopsy findings the cause of death was opined as shock consequent upon acute haemorrhagic pancreatitis.

DISCUSSION

Acute pancreatitis is a reversible, inflammatory disorder of the pancreas which is precipitated by premature activation of the trypsin.[4] It occurs predominantly in males of the 30-40 years age group.[1,2] Age of the present case was 32 years which corresponds to the previous studies.

Though gallstone was reported to be the predominant cause for AP, studies from Ohio (USA), Germany, and India found alcohol abuse as the major causative factor in 80%, 70% and 60% cases of acute pancreatitis, respectively.[2,8,9] Various mechanisms by which alcohol can induce pancreatitis are elevation in exocrine function, contraction of the sphincter of Oddi, direct acinar cell damage by generating oxidative stress, and duct obstruction by protein plug formation.[4] In the reported case, he had a history of chronic alcoholism for 8 years. He started developing symptoms immediately after consumption of alcohol in the evening. Acute ingestion of a large amount of alcohol could have precipitated pancreatitis in this case.[10] Other most common causes are hypertriglyceridemia, endoscopic retrograde cholangiopancreatography (ERCP) particularly after biliary manometry, blunt abdominal trauma, drugs (for example, azathioprine, oestrogens, tetracycline) and hereditary following PRSS1, SPINK and CFTR gene

mutations.[1,11]

Acute pancreatitis is diagnosed clinically when two of the following three criteria are present (Revised Atlanta Classification 2012): 1) Typical abdominal pain, (persistent, severe epigastric pain, usually radiating to back) (2) serum lipase (or amylase) level ≥ 3 times the upper limit of normal, and 3) characteristic findings on contrast-enhanced computed tomography (CECT) and less commonly MRI or transabdominal USG.[6] Acute pancreatitis with normal serum amylase and lipase levels are also reported in the literature.[12,13]

In our case, the nonspecific complaints (vague abdominal discomfort and vomiting) at admission did not raise the suspicion of acute pancreatitis. Furthermore, he was referred to this tertiary center without any preliminary investigations. Hence, it was not diagnosed until the post mortem examination. It must be stressed here that the symptoms could be masked or obscured in an alcoholic or patients with antipsychotic medications.[14] A similar case of severe acute haemorrhagic pancreatitis with mild or vague symptoms reported from Thailand by Srettabunjong et al.[7] They reported a case of a 38-year-old male, who had symptoms of dizziness, nausea, and fatigue without significant abdominal pain lasted 1 week before death. Besides, he had lower extremity weakness, intense thirst for the last 2 days, and collapsed before he was received dead at the ED. They concluded that the cause of death was extensive severe haemorrhagic acute pancreatitis secondary to cholelithiasis.[7]

Treatment is mainly supportive. Aggressive fluid resuscitation following nil per oral (NPO) to rest the pancreas, intravenous analgesics (narcotics) is the cornerstone of treatment.[11] The present case was treated symptomatically with antacids and antiemetics. Early suspicion coupled with intensive care would have made the prognosis better.

The mortality of AP is usually < 1% for mild form (oedematous pancreatitis) and ranging from 10 to 30% for a severe form of AP (haemorrhagic pancreatitis).[15] This fatality might be influenced by multiple local and systemic complications that arise after an acute attack. These early complications (onset to three days) of AP are systemic in nature16 and they may include shock, respiratory failure, acute renal failure, DIC, hyperglycaemia, and hypocalcemia.[3] In the present case, the damage was fatal due to necrosis and extensive haemorrhage within the pancreatic substance and extra pancreatic tissues. Locally spreading necrosis with enzymatic digestion of the blood vessels and rupture of pseudoaneurysms are known to cause initial haemorrhage In AP.[5]

Post mortem autolysis of the pancreas is usually mediated by the release of stored enzymes from the duct of the pancreas after death. Grossly, the autolyzed pancreas may appear to be soft with loss of lobular architecture.[17] In our case, the pancreas was soft, oedematous, and haemorrhagic across the entire gland with intact lobular architecture at autopsy. For histopathological examination, post mortem sampling of the pancreas should be preferably done within 8 hours of death to observe the features of antemortem pathology such as acute inflammatory cell infiltrate, fat necrosis, and calcium deposition.[15,18,19] Although history and gross findings were suggestive of acute haemorrhagic pancreatitis, (Fig. 1) this report fails to report microscopic findings of acute haemorrhagic pancreatitis likely due to the accelerated autolysis of the pancreas in pancreatitis and the longer sampling interval (18 hours).[20]

In conclusion, acute haemorrhagic pancreatitis is the fatal complication of acute pancreatitis and represents high morbidity and mortality. This case report raises awareness among the treating physicians on unusual symptoms of acute haemorrhagic pancreatitis at admission to make a prompt diagnosis and early intervention. Although rare, the autopsy surgeon should have a high index of suspicion for acute haemorrhagic pancreatitis and examine the pancreas in all sudden, unexpected, and unexplained deaths.

ACKNOWLEDGMENT

The authors express sincere thanks to all faculties and residents of the Department of Forensic Medicine and Toxicology at All India Institute of Medical Sciences, Bhubaneswar, Odisha, India.

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