Advances in Surgical Sciences
2025, Vol. 13, No. 2, pp. 17-21
https://doi.org/10.11648/j.ass.20251302.11

Research Article

Acute Necrotic Haemorrhagic Pancreatitis (ANHP): Two Cases in a Hospital with Limited Resources (CHU de Conakry, Guinea), and a Review of the Literature

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Abstract

Introduction: Severe forms of acute pancreatitis are defined by the appearance of visceral failure or a local complication (necrosis, pseudocyst, abscess). *Purpose:* To discuss two cases of difficult diagnosis of acute necrotic-haemorrhagic pancreatitis (ANHP) discovered *intraoperatively in a clinical context of acute generalised peritonitis. Observation 1:* 23-year-old patient admitted with abdominal pain, vomiting, cessation of feces and gas, abdominal distension and fever. Course: 72 days. History: epigastralgia. No history of alcohol or tobacco intoxication. *Examination:* Painful symmetrical abdomen, maximum in the epigastrium. Generalized parietal defensiveness. Decreased tenderness of flanks. Rectal examination: Douglas bulging and painful. After median laparotomy: 3.5 litres of blackish liquid, 'candle flows' on the greater omentum and transverse colon. No gastric or duodenal perforation. Pancreatic necrotic-haemorrhagic lesions present. Omentectomy. Necrosectomy, cleansing, drainage. Follow-up: oedema of the lower limbs, parietal then deep suppuration, evisceration. Surgical revision: total necrosis of the pancreas. Necrosectomy, peritoneal cleansing. Death on the operating table. *Observation 2:* 60-year-old patient admitted with abdominal pain, nausea, cessation of bowel movements and gas, fever. Evolution 22 hours. Notion of chronic alcohol-smoking. *Examination:* Abdomen painful throughout, maximum in the umbilical region, epigastrium, parietal defence. Pre-hepatic fat retained. After median laparotomy. Aspiration of 200 ml of haematic fluid. No gastro-duodenal perforation. Presence of 'candle stains', necrotic and haemorrhagic zones in the pancreas. Necrosectomy. Peritoneal cleansing and drainage. Good post-operative outcome. *Conclusion:* Drainage of infected necrosis was achieved by surgical necrosectomy in our observations.

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Received: 29 September 2024; **Accepted:** 1 July 2025; **Published:** 22 July 2025

Keywords

Necrotic-haemorrhagic Pancreatitis, Peritonitis, Diagnosis, Surgery

1. Introduction

Assessment of severity is an essential step in the management of acute pancreatitis [1]. The prognosis differs between acute oedematous forms (mortality <1%) and severe forms (mortality 25 to 35%) [1]. Severe forms of pancreatitis are defined by the appearance of visceral failure or a local complication (necrosis, pseudocyst, abscess) [2]. Acute necrotizing-hemorrhagic pancreatitis (ANHP) may be discovered preoperatively or intraoperatively and requires early and effective management.

Purpose: To discuss two cases of diagnosis and difficult therapeutic management of acute necrotic-haemorrhagic pancreatitis (ANHP) discovered intraoperatively in a clinical context of acute generalised peritonitis in the visceral surgery department of the Donka CHU National Hospital in Conakry.

2. Observation 1

23 year old patient, mechanic, admitted with abdominal pain, vomiting, cessation of fluid and gas, abdominal distension, fever. Progression: 72 hours.

History: epigastralgia. Traditherapy (use of decoctions).

On examination: patient agitated, dry lips, hypocoloured integuments and mucous membranes. Blood pressure (BP) = 80/60 mm Hg, temperature = 38.1 ℃, pulse = 122. Painful abdomen, maximum in the epigastrium. Generalized parietal defensiveness. Decreased tenderness of flanks. Inaudible peristalsis. Rectal examination: Douglas bulging and painful.

Emergency workup: white blood cells 13 giga/litre, haemoglobin 11 g/dl, blood glucose 0.97 g/l.

Preoperative diagnosis: Acute generalised peritonitis due to probable perforation of peptic ulcer + shock.

After midline laparotomy. Aspiration of 4.5 litres of odourless blackish fluid. Examination showed thickened, infiltrated large omentum and transverse colon. There were no perforations in the stomach or duodenum. Access to the omental bursa by opening the gastrocolic ligament: necrotic-haemorrhagic lesions of the pancreatic lodge infiltrating the supramesocolic stage (Figure 1). We performed:

(1). omental biopsy for histopathological examination, sampling of peritoneal fluid for bacteriological examination. (2). Necrosectomy, cleansing and drainage of the pancreatic and subhepatic cavities.

Postoperative diagnosis: Acute necrotic-haemorrhagic pancreatitis.

Postoperative care: ceftriaxone 2 g/24H, Tramadol 100 mg/8H, perfusable Metronidazole 500 mg/8H, Omeprazole 40 mg injectable /24H. Rehydration (0.9% saline, 5% glycosylated serum, Ringer lactate, Hemacel).

Post-operative biological check-up: haemoglobin 9 g/dl, lipasemia: 180 U/L, blood glucose: 1.2 g/l, blood calcium: 3 mmol/l.

Post-operative follow-up: oedema of lower limbs, fever to 39 ℃, parietal then deep suppuration, evisceration. At 30 days post-op acute generalised peritonitis + evisceration of necrotic-haemorrhagic pancreatitis. Intraoperatively we noted total necrosis of the pancreas and deposits of false membranes in places. We performed a necrosectomy and peritoneal lavage. During these procedures, the patient died on the operating table from probable septic shock, with multiple organ failure.

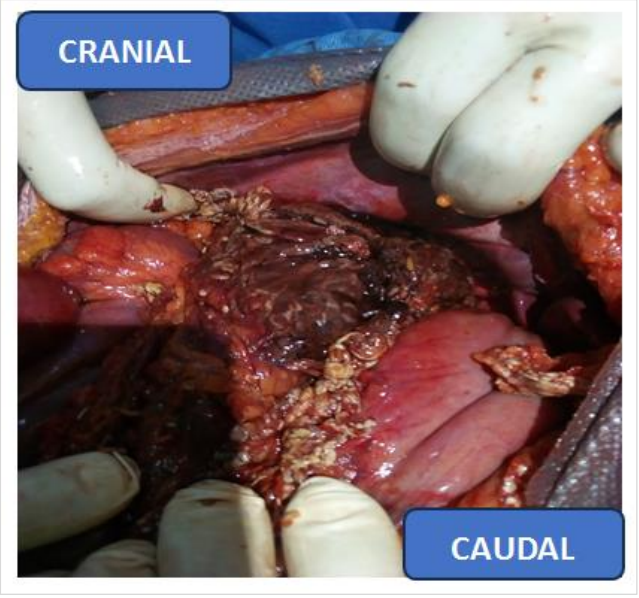

Figure 1. *Operating view Pancreatic necrosis.*

3. Observation 2

60 year old patient, sailor, admitted for sudden epigastric pain after a meal, radiating to the bar and back, nausea, cessation of bowel movements and gas, fever. Progression 22 hours. Chronic alcoholic-smoker.

On examination: BP =110/70 mmHg, pulse = 86, temperature = 38.5 ℃, respiratory rate =24 cycles/minute. General condition satisfactory, normal-coloured integument and mu-

cous membranes, active posture. Abdomen only slightly involved in breathing, painful overall with a maximum in the umbilical region, epigastrium, right hypochondrium and right flank with parietal defence. Prehepatic dullness preserved. No sloping flank fat. Free hernial orifices. Elsewhere no particularities.

ASP: diffuse aerocolia with pneumatisation of the distal portion of the digestive tract without hydroaeric images.

Abdominal ultrasound: moderate peritoneal fluid effusion in the perivesicular, intervesical and Douglas areas, suggesting probable peritonitis due to gastric perforation. Homogeneous hepatomegaly with no abnormalities of the bile ducts or portal trunk. Absence of pancreatic and spleno-renal morphological lesions.

Biology: hyperleukocytosis of 12.8 giga/litre, positive retroviral serology, haemoglobin level: 14 g/litre, blood glucose: 1.31 g/litre, blood group B+.

Preoperative diagnosis: acute generalised peritonitis due to perforation of a gastroduodenal ulcer, probably? in a human immunodeficiency virus (HIV) infection.

After median supra and sub umbilical laparotomy. Aspiration of 200 ml of haematic fluid. Investigation showed local 'candle spot' lesions and necrotic and haemorrhagic areas in the pancreatic cavity (Figure 1). Peritoneal lavage with 4 litres of 0.9% SS. Placement of a drain in the omental bursa through the omental foramen, brought out by a counter incision under the right rib. Parietal closure.

Postoperative diagnosis: Acute necrotic-haemorrhagic pancreatitis with HIV (Human immunodeficiency Virus).

The postoperative course was favourable under treatment: rehydration with 1.5 litres of fluids (LR, 0.9% saline, 5% glycosylated serum) / 24 hours, atropine 0.5 mg, perfusable paracetamol 1 g, ceftriaxone 2 g/24H, metronidazole 500 mg perfusable /8H, omeprazole 40 mg injectable /24H, fat-free refeeding. Post-operative follow-up at 11 days post-op revealed genital herpes which was treated, after a dermatological consultation, with aciclovir 200 mg, Betadine scrub and dermal betadine. The patient was seen again at 20 days with good clinical progress. The surgical wound was healing well. A biological check-up: lipasemia, glycaemia, transaminases, was unremarkable.

4. Discussion

Severe acute pancreatitis is one of the most serious digestive disorders. It is a medical and surgical emergency [2]. Severe forms of acute pancreatitis are defined by the occurrence of visceral failure or necrosis and superinfection of the necrosis, or pseudocysts or abscesses [1]. In our two (2) cases, the necrotic-haemorrhagic form was discovered in the context of an acute peritoneal syndrome caused by perforation of a gastroduodenal ulcer.

The probable causes of the acute necrotic-haemorrhagic pancreatitis were alcoholic (observation 2) and unknown (observation 1).

Terrain is a prognostic factor. Elements of severity are: age > 50 years, obesity (Body Mass Index BMI >30), visceral failure (respiratory, cardiac, renal), hypovolaemic shock (haemodynamic visceral failure) [1, 2].

In the first case, our patient was young (under 50 years of age), not obese, and had no particular history suggesting a pancreatic origin. However, he was admitted in a state of shock, reflecting very marked visceral failure. The risk factors are correlated with the aetiologies, the main ones being alcohol and lithiasis [1].

The clinical investigation in the first case did not reveal any alcoholic or lithiasis-related aetiology. It was an idiopathic necrotic-hemorrhagic pancreatitis. Only 5-10% of cases of PANH in adults remain unexplained [1].

In the second case, the patient was aged 60 (over 50). The main risk factor was alcohol intoxication [1, 2] for more than 20 years in a specific HIV setting. He had been on antiretroviral therapy (Emtricitabine/Tenofovir Disoproxil) for about 10 years.

The etiologies were dominated by biliary lithiasis (45%) and alcohol intoxication (35%) [1, 2].

From a pathophysiological point of view, early organ failure associated with hypovolaemia determines late necrotic superinfection, due to the existence of mesenteric hypoperfusion associated with bacterial translocation [1].

However, the clinical features (diffuse pain with a maximum in the epigastrium, vomiting, cessation of bowel movements and gas) led to the diagnosis of acute generalised peritonitis due to perforation of a gastroduodenal ulcer in both cases.

Biological tests showed a hyperleukocytosis with neutrophilic polymorphonuclear cells.

In both cases, the unprepared abdomen X-ray was non-contributory. Abdominal computed tomography was not performed because it was not available urgently in our context. The standard techniques (unprepared abdominal X-ray and chest X-ray) are still of definite value in diagnosing complications (peritonitis, pleurisy, etc.): rule out pneumoperitoneum due to perforation or intestinal ischaemia [1, 3]. PSA may show evidence of PANH (calcium-toned image in the vesicular or pancreatic area).

Abdominopelvic CT scans can identify changes in the pancreas (homogeneous enlargement of the pancreas, areas of necrosis, peripancreatic flows) [2, 3].

Infection of the necrosis occurs after the first week or two. It is the cause of multiple visceral failures and, in the absence of effective eradication, the majority of deaths [4]. The presence of air bubbles within the necrosis on CT scan is a good sign, supporting the diagnosis of infection of the necrosis when it reaches 30% of the gland [3]. This necrosis is an important prognostic factor [1, 4].

Computed tomography (CT) is the reference examination that provides the most information about the severity of pancreatitis and predicts the occurrence of complications and mortality [3, 4].

The diagnostic approach has 3 objectives: - to make a positive diagnosis on the basis of the characteristics of the epigastric pain, the lipasemia assay (essentially), and radiological investigations;

1) Assess the initial severity (bioclinical and radiological scores);
2) look for local, loco-regional and systemic complications [1, 4, 6].

In both cases, the diagnosis of necrotic-haemorrhagic pancreatitis was made intraoperatively by the discovery of:

1) candle stains on the omental apron, (Figure 2).
2) necrotic-haemorrhagic lesions of the pancreatic cavity in a context of acute generalised peritonitis (Figure 1).
3) Peritoneal fluid effusion reflects visceral failure, which is responsible for systemic haemodynamic, respiratory, renal, haematological and metabolic complications (shock) [5, 6], as in our observations.

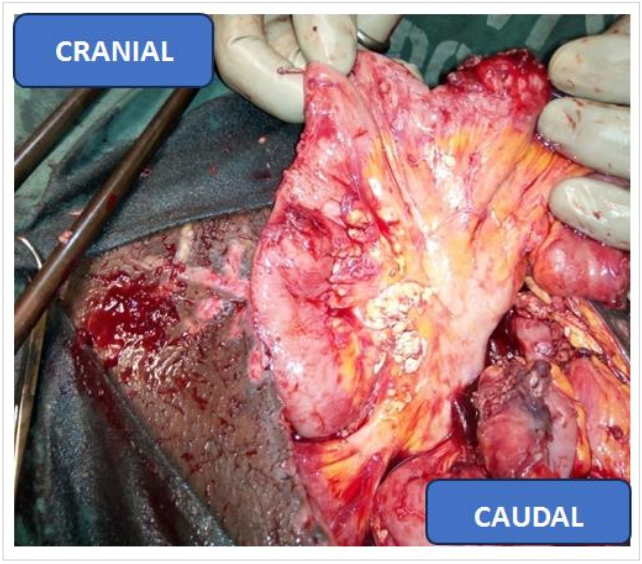

Figure 2. *Operating view Candle stains.*

Local complications include pancreatic necrosis. Superinfection of pancreatic necrosis has a decisive influence on the local course and prognosis [1, 4, 6]. Diagnosis of the severity of PANH depends on the aetiology, the patient's condition, associated pathologies and pancreatic lesions, particularly necrosis and superinfection [2, 4-6].

The surgical indications for PANH are: superinfection of necrosis (a serious factor), pseudocysts, haemorrhage, perforation, and treatment of the aetiology [1, 2, 5, 7].

In our context, the indication was, a fortiori, pancreatic necrosis in both cases (Figure 3).

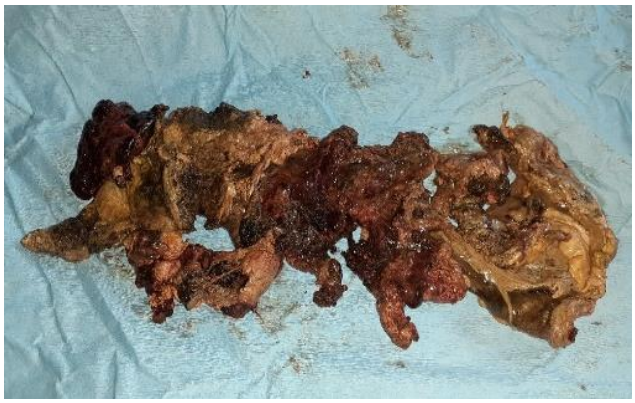

Figure 3. *Surgical specimen of pancreatic necrosis.*

Management was multidisciplinary (anaesthesia-intensive care, surgery, radiology and microbiology): surgery for sterile necrosis, indication for biliary surgery and endoscopic retrograde cholangiopancreatography (ERCP) [5, 7].

Treatment was surgical. Treatment consisted of drainage, omental biopsy (necrosis) and bacteriological sampling of the peritoneal fluid. Bacteriological analysis isolated ceftriaxone-sensitive Staphylococcus aureus.

Treatment of infection of the necrosis is based on transparietal radiological drainage [7], surgical necrosectomy [8, 9] (Figure 3) or endoscopic necrosectomy [10, 11]. Appropriate antibiotic therapy, in terms of bacteriology and pharmacokinetics, may be justified early on in severe cases [1, 4, 12]. In both cases, drainage was surgical, combined with antibiotic therapy (ceftriaxone 2 g/24hrs, metronidazole 500 mg 1.5 g/24hrs) and rehydration (9% saline, Ringer lactate) 2 L/24hrs.

The other therapeutic options (radiological and endoscopic) are not commonly used in emergencies in our context.

In the first observation, the post-operative course was riddled with septic complications on postoperative day 12: purulent fluid flow through the drains, into the pancreatic cavity and the subhepatic cavity, through the median wound responsible for evisceration, fever, progressive weight loss. Superinfection of the necrosis may occur early (1-2 weeks) in 25-30% of cases, but the maximum risk is observed during the 3rd week [1, 5, 6]. This risk increased during the 2nd week in our first observation [1, 2]. Superinfection of the necrosis is a serious complication from the 2nd week onwards, revealed by a septic syndrome that can be confirmed by radioguided puncture + bacteriological examination [13].

Post-operative biological monitoring revealed: lipasemia > 3N, anaemia at 8 g/L (a blood transfusion was carried out). Iterative clinical controls led to an indication for repeat surgery for post-operative generalised acute peritonitis plus Evisceration. The total necrosis of the pancreas found in the first observation may reflect the severity of the superinfection, a consequence of the delay in management [5, 6, 12].

Secondary complications such as diabetes and exocrine pancreatic insufficiency are correlated with the extent of

necrosis [1, 13]. This makes the prognosis more guarded [1, 5].

Lipasemia is produced and secreted exclusively by the pancreas. It is the best, most specific and most sensitive biological test for diagnosing acute pancreatitis [1, 2, 5].

Surgical treatment of complications (superinfection of necrosis, haemorrhage) remains cumbersome, with a high morbidity and mortality rate [14]. In the first observation, death occurred intraoperatively. It was caused by septic shock, probably due to superinfection of the total necrosis of the pancreas (poly-visceral failure). The main cause of death is (infection of the necrotic tissue, which is associated with a poor prognosis [5, 12]. In our case, the duration of the pancreatitis (72 hours) and the use of traditional decoctions would have been factors in the poor prognosis. Mortality varies from 11 to 39% [1]. Mortality due to PANH remains high (25 to 35%) compared with oedematous forms [6]. From the point of view of evolution, the two main causes of death associated with acute pancreatitis, which are of equivalent importance, are organ failure in the first few days and infection of the pancreatic necrosis between the 2nd and 4th week of the evolution of AP [1, 2, 5, 12], as was the case in our first observation.

In the 2nd observation, in contrast to the first case, the post-operative course was favourable, with healing in the first instance notwithstanding the HIV infection. Morbidity was 40% [1]. It is manifested by fistulas, endocrine sequelae and ventrations [4, 6, 14].

5. Conclusion

The necrotic-haemorrhagic pancreatitis in our cases was very serious. In all cases it presented as a pseudo-peritoneal form simulating ulcer perforation. Treatment consisted of drainage of the infected necrosis and surgical necrosectomy. The prognosis was good in one case and poor (death) in the other. The latter was due to systemic complications and local anatomopathological lesions of the pancreas.

Early diagnosis and multidisciplinary therapeutic management can improve the prognosis of patients suffering from acute necrotic-haemorrhagic pancreatitis.

Conflicts of Interest

The authors declare that there is no conflict of interest.

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