Case Report

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Do not ignore Walzel's sign with Acut Severe Pancreatititis: A Mortal Chain of Complications with Multiple Organ Dysfunction Syndrome and Disseminated Intravascular Coagulation

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Abstract

This case report describes a patient with severe acute pancreatitis (AP) who presented with livedo reticularis (Walzel's sign), a rare cutaneous finding. A 51-year-old woman presented to the emergency department with reticular skin lesions starting from the lower extremities and spreading throughout the body. Laboratory and radiologic investigations showed that she developed severe AP, multiple organ failure and disseminated intravascular coagulation. The patient's clinical course deteriorated rapidly and he died in cardiac arrest within approximately 10 hours. This case is presented to contribute to the literature and to point out that Walzel's sign characterized by livedo reticularis, a rare skin finding in severe AP, may be a clinical sign that may lead to early diagnosis. It is envisioned that early diagnosis in patients with severe AP with aggressive course may have a favorable effect on the clinical course and prognosis.

Keywords: Acute pancreatitis, acute renal failure, disseminated intravascular coagulation, livedo reticularis, walzel's sign

Introduction

Acute pancreatitis (AP) is an inflammatory condition that begins with damage to the acinar cells of the pancreas and triggers a systemic inflammatory response. Patients usually present with gastrointestinal symptoms such as severe abdominal pain starting in the epigastric region and radiating to the back, nausea and vomiting. However, extra-pancreatic findings should not be ignored (1). Although most cases of AP have a mild course, approximately 20% may require intensive care unit (ICU) hospitalization with a severe clinical picture. Morbidity and mortality rates may reach upto 30% in this patient group (2). Severe AP may be further complicated by conditions such as multiple organ failure and vascular complications, making the diagnosis difficult. The association of livedo reticularis rash, which is one of the skin lesions associated with AP, is also known as Walzel's sign and may be guiding in the diagnostic process. The 10-hour clinical course of this atypical case is presented in the light of the literature to draw attention to the importance of skin findings such as livedo reticularis in cases presenting with severe AP.

Case Report

A 51-year-old female patient with no known comorbidities presented to the emergency department with a reticular, net-like rash that initially appeared on the lower extremities and rapidly spread to the trunk and upper limbs without mucosal involvement (Figure-1). She denied any history of smoking, alcohol or intravenous drug use, trauma, recent cholecystitis, or other systemic diseases. A routine serum β-hCG test was negative, and she had a history of two previous deliveries.

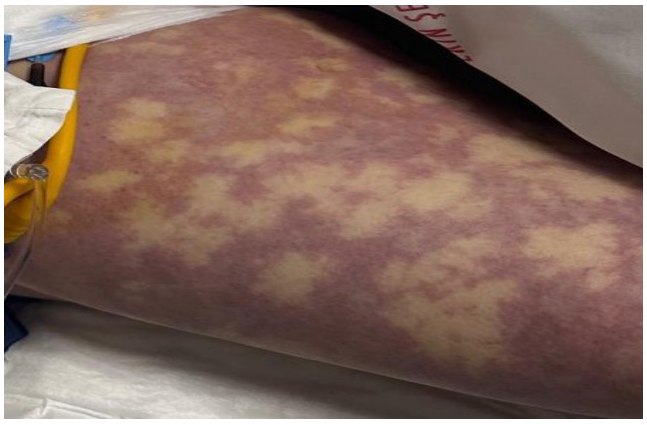

Figure 1. Livedo reticularis rash (Walzel's sign)

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On admission, the patient's general condition was poor, and her Glasgow Coma Scale (GCS) score was 12. Vital signs were as follows: body temperature 37.5 °C, heart rate 110 bpm, respiratory rate 38 breaths/min, blood pressure 167/82 mmHg, and oxygen saturation 97% on room air.

Initial laboratory results revealed elevated liver enzymes (ALT 133 U/L, AST 201 U/L), LDH 925 U/L, amylase 210 U/L, lipase 187 U/L, calcium 6.4 mg/dL, andurea 58 mg/dL. Complete blood count showed WBC $33.2\times10^{9/L}$, hemoglobin 7.3 g/dL, hematocrit 22.4%, MCV 134.9 fL, MCH 89.2 pg, and MCHC 66.1 g/dL. Coagulation parameters were markedly abnormal: aPTT 73.1 s, PT 24.9 s, INR 2.18, D-dimer> 8 µg/mL, and fibrinogen 0.61 g/L. Arterial blood gas analysis revealed a high anion gap metabolic acidosis with elevated lactate (pH 7.18, lactate 8.1 mmol/L, BE −7). C-reactive protein (CRP) was within normal limits, while the erythrocyte sedimentation rate (ESR) was> 140 mm/h.

The patient received fluid resuscitation and correction of metabolic abnormalities, after which she was admitted to the intensive care unit (ICU). In the ICU, her hemodynamic status remained unstable. The mean arterial pressure (MAP) target was maintained at ≥ 65 mmHg using norepinephrine infusion titrated between 0.2 and 0.5 µg/kg/min. Despite administration of approximately 3 L of crystalloids within the first six hours, adequate circulatory response was not achieved. Blood urea and creatinine levels progressively increased, and urine output fell below 0.5 mL/kg/h, followed by oliguria; hemodiafiltration was recommended.

Arterial blood gas analysis showed progressive lactate elevation (14.7 → 16 → 19 mmol/L). Coagulation tests demonstrated prolonged aPTT, PT, and INR with elevated D-dimer and decreased fibrinogen. Platelet count decreasedto $120\times10^3/\mu L$. Peripheral smear revealed erythrocyte hemagglutination without atypical cells. After hematology consultation, blood grouping and transfusion planning were completed. Considering disseminated intravascular coagulation (DIC) and acute kidney injury, transfusion thresholds were set at hematocrit< 25% and platelet < 50,000/µL, and red blood cell suspension, cryoprecipitate (1 unit/10 kg), and fresh frozen plasma were administered.

Approximately two hours after admission, the patient developed respiratory failure and was intubated. Pressure-controlled mechanical ventilation was initiated (FiO_2 0.6, PEEP 6 cmH_2O, tidal volume 6 mL/kg). Post-intubation, the PaO_2/FiO_2 ratiowas 120, and plateau pressure was maintained below 30 cmH_2O. Empiric carbapenem therapy was started and later adjusted according to culture results. As the patient's overall condition was critical, exploratorylaparotomy was deferred until hemodynamic stabilization, and intensive care management was continued.

In the following hours, signs of multiple organ failure became evident. Despite maximal norepinephrine support, hypotension became refractory. Respiratory function rapidly deteriorated, and despite FiO_2 100%, the PaO_2/FiO_2 ratio dropped below 80. The patient became anuric, and continuous renal replacement therapy (CRRT) was initiated at hour 8. Laboratory tests revealed pH 7.03, lactate 24 mmol/L, markedly elevated D-dimer, and thrombocytopenia ($120\times10^3/\mu L$), consistent with DIC. Despite aggressive supportive therapy, cardiovascular collapse ensued, and the patient experienced cardiac arrest at hour 10 and could not be resuscitated.

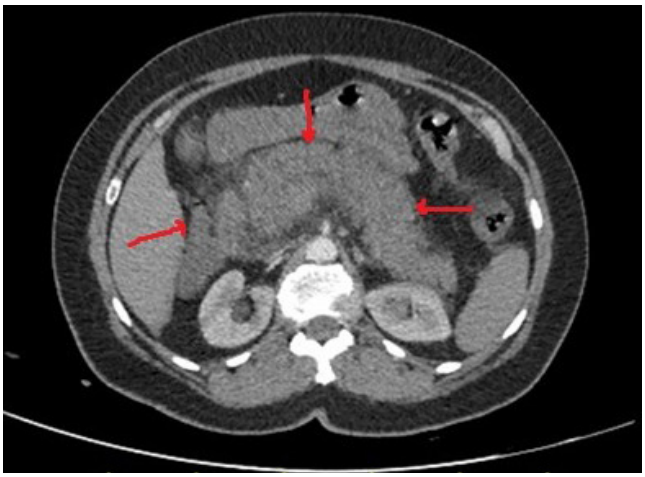

Figure 2. Appearance of the pancreas on contrast-enhanced abdominal CT image

At presentation, the livedo reticularis-type lesions were limited to the lower extremities but progressively worsened during follow-up, spreading to the trunk and upper limbs (Figure-1). Dermatology consultation confirmed the diagnosis of livedo reticularis and recommended etiological evaluation. Despite hemodynamic optimization and supportive therapy, no regression was observed in the skin lesions. The reticular pattern persisted until the terminal stage and became diffuse overmost of the body before death.

Contrast-enhanced abdominal computedt omography (CT) revealed an edematous pancreas with irregular contours, heterogeneous peripancreatic fats tranding, and peripancreatic fluid accumulation (Figure-2). The gall bladder wall was markedly thickened and edematous, with focal pancreatic necrosis but no peripancreatic collection. The Balthazar score was assessed as grade D. Emergency abdominal ultrasonography (USG) demonstrated minimal pericholecystic free fluid; however, due to bowel distension and overlying gas, the pancreas could not be clearly visualized. Brain and thoracic CT scans were unremarkable. Abdominal examination revealed distension without guarding or rebound tenderness.

Discussion

This case of severe and atypical acute pancreatitis (AP) is presented to highlight its association with a rare cutaneous manifestation and to emphasize the clinical significance of mortal complications, supported by literature.

Although most patients with acute pancreatitis present with classic gastrointestinal symptoms such as abdominal pain, nausea, and vomiting, extra-pancreatic manifestations may provide early diagnostic and therapeutic clues. In this patient, livedo reticularis was the initial presenting sign. The purplish, reticular rash, initially prominent in the lower extremities, rapidly progressed to involve the entire body within hours. Livedo reticularis is characterized by a violaceous, net-like skin pattern caused by impaired cutaneous blood flow.

Differential diagnosis includes vasculitis, antiphospholipid antibody syndrome, cholesterol embolism, cryoglobulinemia, and severe sepsis. In this case:

- Vasculitis was excluded due to negative inflammatory markers.
- Tests for antiphospholipid antibody syndrome (ANA, anti-dsDNA, antiphospholipid antibodies) were negative.
- There was no history of vascular intervention or atherosclerotic risk factors suggestive of cholesterol embolism.
- Laboratory findings did not indicate cryoglobulinemia, and cultures were negative for sepsis.

The abrupt onset, rapid progression, and concurrent severe AP support a diagnosis of pancreatitis-associated livedo reticularis. Reports in the literature of livedo reticularis associated with acute or chronic pancreatitis are very rare. Daniel et al. reported that in cases of acute pancreatitis, the cutaneous lesions resolved with fluid therapy and antibiotic treatment as the underlying disease improved (3). Gould et al. described recurrent rashes that appeared during pancreatitis exacerbations and resolved completely after the episodes subsided (4). Cutaneous findings often reflect systemic inflammation and microcirculatory dysfunction and are associated with poor prognosis (5). Early recognition may serve as an early warning of worsening pancreatitis and may influence prognosis.

AP results from premature activation of pancreatic enzymes within the pancreas, leading to inflammation ranging from edema to necrosis. Severe AP is associated with various etiologic factors, including hyperlipidemia, which is thought to trigger inflammation through increased free fatty acids in the pancreas. The release of pro-inflammatory cytokines such as TNF-α and IL-1, along with lipolytic and proteolytic enzymes, disrupts tissue integrity, allowing enzymes to entersy stemic circulation, causing endothelial injury and activation of the coagulation cascade (5).

In this case, the patient's organ dysfunction, evaluated using the Modified Marshall Scoring System, was 4 (PaO_2/FiO_2 ≤100, creatinine>439 μmol/L, systolic blood pressure <90 mmHg, pH <7.2), classifying the AP as severe according to the Atlanta Classification. Mortality may reach 35% in 20–25% of moderately severe cases. According to the World Health Organization, AP is the 14th leading cause of death worldwide, with an incidence of 0.90–1.10 per 100,000 (6). Accurate assessment of disease severity is critical for guiding treatment and predicting prognosis.

Elevated arterial lactate is associated with increased 28-day mortality in severe AP (7). The activated partial thromboplastin time (aPTT) test is used to detect endogenous coagulation disorders. Prolonged aPTT is linked to more severe organ failure in sepsis and higher ICU mortality. In patients with prolonged aPTT in AP, the incidence of disseminated intravascular coagulation (DIC) is higher than in those with normal values (8). Hematologic abnormalities in severe AP may include decreased hematocrit, hypercoagulability due to clotting factor defects, DIC, thrombotic thrombocytopenic purpura/hemolyticuremic syndrome, leukocytosis, abnormal erythrocytes, increased reticulocyte count, acute hemolytic anemia, and thrombocytopenia.

Livedo reticularis may reflect DIC and microvascular perfusion disturbances in severe pancreatitis. Fibrin microthrombi and impaired capillary circulation in DIC contribute to its characteristic appearance. Therefore, the presence of livedo reticularis may serve as an early indicator of multiple organ dysfunction syndrome and provide prognostic information as a clinical correlate of hemodynamic deterioration.

At presentation in the emergency department, arterial lactate was 8.1 mmol/L, rising to 17 mmol/L during follow-up. aPTT, PT, and INR were prolonged, fibrinogen was decreased, and other coagulation parameters were impaired, indicating the development of DIC.

Acute kidney injury (AKI) in AP arises from local inflammation triggered by acinar cell injury, leading to systemic inflammatory response and cytokine activation. AKI occurs in 15–69% of patients with AP and is a serious complication, increasing mortality ten fold (9,10). Patients with renal involvement have longer hospital stays, higher ICU admission rates, increased mortality, and higher treatment costs. Early recognition, aggressive therapy, and prompt ICU transfer are therefore critical.

While elevated pancreatic enzymes, abdominal distension, and systemic inflammatory response are expected in AP, livedo reticularis as the initial presenting sign in this case was unexpected and atypical. This rare dermatologic finding, associated with DIC and AKI, reflects the severity of systemic inflammation and vascular involvement. Recognition of such atypical cutaneous signs may facilitate early diagnosis of severe AP and improve clinical outcomes.

Conclusion

This case highlights the clinical significance of Walzel's sign, characterized by livedo reticularis, in severe acute pancreatitis. Recognition of this early dermatologic manifestation may provide valuable guidance for early

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diagnosis and management, contributing to the literature. Given the potential for rapid progression to multiple organ failure and DIC in severe AP, prompt identification and aggressive treatment strategies may improve prognosis.

Ethics Informed Consent: Written informed consent required for the case report as obtained from the patient.

Conflict of Interest: No conflict of interest was declared by the authors.

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