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CASE IMAGE

Extensive epidural pneumatosis and pneumomediastinum combined with diabetic ketoacidosis

Akiko Hirata | **Naoki Yonezawa** | **Miho Fukasawa** | **Michiko Fujisawa**

Department of Emergency and Critical Care Medicine, Yokohama City Minato Red Cross Hospital, Yokohama, Kanagawa, Japan

Correspondence
Naoki Yonezawa, Department of Emergency and Critical Care Medicine, Yokohama City Minato Red Cross Hospital, 3-12-1 Shinyamashita, Naka-ku, Yokohama, Kanagawa 2318682, Japan.
Email: nyone139@gmail.com

Key Clinical Message

Epidural pneumatosis and pneumomediastinum are rare, benign complications of diabetic ketoacidosis. As they can mimic serious conditions including esophageal rupture, diagnostic evaluation, and attentive monitoring are crucial.

Abstract

Diabetic ketoacidosis can rarely present with epidural pneumatosis and pneumomediastinum, possibly due to forceful vomiting and Kussmaul breathing. Recognizing these pneumocomplications is crucial, as they can mimic severe conditions, including esophageal rupture. Consequently, diagnostic workup and vigilant monitoring are critical, even though these pneumocomplications are typically benign and self-resolving.

KEYWORDS

diabetic ketoacidosis, epidural pneumatosis, Hamman syndrome, pneumomediastinum

A 14-year-old girl in shock and coma presented to the emergency department following 7 days of fatigue, fever, vomiting, and chest pain, after 1 month of polydipsia, polyuria, and weight loss. She preferred soft drinks and had no medical history except for a positive urinary glucose result 3 months earlier. Her body mass index was 18 kg/m^2. Upon examination, the patient had a Glasgow Coma Scale of 3 points; blood pressure, 49/22 mmHg; pulse, 130 beats/min; temperature, 40.7°C; and oxygen saturation, 95% on room air. She displayed dehydration signs, Kussmaul breathing at a rate of 30 breaths/min, and no neck stiffness. Her abdomen was soft and flat, with normal bowel sounds. Subcutaneous crepitations were not palpable. Arterial blood gas analysis showed metabolic acidosis with pH 6.95, $PaCO_2$ 31 mmHg, bicarbonate 6.4 mmol/L, lactate 1.6 mEq/L, sodium 132 mEq/L, chloride 108 mEq/L, and potassium 3.3 mEq/L. Laboratory results revealed a leukocyte count of $16.3 \times 10^3/\mu L$; hemoglobin, 14.3 g/dL; platelets, 317,000/μL; C-reactive protein, 1.3 mg/dL; creatinine, 3.16 mg/dL; glucose, 1409 mg/dL, and; hemoglobin A1C, 12.3%. The plasma concentrations of acetoacetic and 3-hydroxybutyric acids were 2960 μmol/L and 6960 μmol/L, respectively.

A diagnosis of diabetic ketoacidosis (DKA) was made; however, unexpectedly, a chest radiograph revealed cervical subcutaneous and mediastinal emphysema (Figure 1A). Following fluid resuscitation, a chest computed tomography (CT) scan was performed to further assess pneumocomplications. The scan revealed air in the epidural cavity between C1 and T7 (Figure 1B), but no hydropneumothorax or pleural effusion. The cerebrospinal fluid analysis was unremarkable, except for a glucose level of 814 mg/dL. Although esophageal rupture and mediastinitis were not completely ruled out, DKA-associated epidural pneumatosis and pneumomediastinum were considered.[1]

With close monitoring, the patient's DKA treatment with intravenous fluid, insulin, and potassium quickly improved

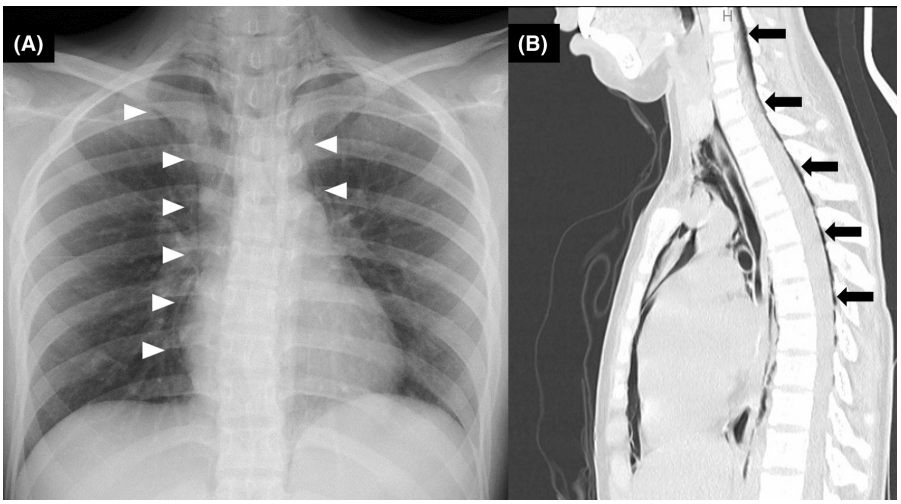

FIGURE 1 (A) Chest radiograph displaying cervical subcutaneous and mediastinal emphysema. (B) Sagittal view of chest computed tomography scan on admission, uncovering extensive air distributed across the epidural canal (black arrows), in the pulmonary window setting.

her hemodynamic status, hyperglycemia, and metabolic acidosis, allowing her to regain consciousness shortly after. She was not given antibiotics. Approximately 24 h later, she remained hemodynamically stable, with an arterial pH of 7.46 and a glucose level of 211 mg/dL. As esophageal rupture or mediastinitis appeared unlikely, drinking water was started,[2] and with no adverse events, oral intake was started on day 3. Blood, urine, and cerebrospinal fluid cultures were all negative on admission. Follow-up CT on day 9 confirmed the disappearance of the epidural pneumatosis and pneumomediastinum. The patient was diagnosed with acute-onset type 1 diabetes mellitus after testing positive for autoantibodies against glutamic acid decarboxylase and tyrosine phosphatase-related islet antigen 2. After diabetic education, she was discharged home.

Although the exact mechanism behind these pneumocomplications in DKA remains unclear, increased pressure from forceful vomiting combined with Kussmaul breathing may cause alveolar rupture and subsequent air dissection to the mediastinum and epidural canal via fascial planes and intervertebral spaces.[1,3] Although awareness of these pneumocomplications is critical, it is also important to understand that they can mimic other more serious conditions, including esophageal rupture, which may have similar symptoms and CT findings.[4] Consequently, diagnostic workup and close monitoring are critical, even though these pneumocomplications are typically benign and self-resolving.[1]

AUTHOR CONTRIBUTIONS

Akiko Hirata: Conceptualization; data curation; investigation; writing – original draft. **Naoki Yonezawa:** Conceptualization; investigation; writing – original draft; writing – review and editing. **Miho Fukasawa:** Conceptualization; data curation; writing – original draft. **Michiko Fujisawa:** Supervision; writing – review and editing.

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CONFLICT OF INTEREST STATEMENT

The authors declare that they have no conflict of interest.

DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no datasets were generated or analyzed during the current study.

ETHICAL APPROVAL AND INFORMED CONSENT

Published with the written consent of the patient.

ORCID

Naoki Yonezawa https://orcid.org/0000-0003-2557-5698

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