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Case Report

Spontaneous pneumoperitoneum and diabetic ketoacidosis in fulminant type 1 diabetes: a case report

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Abstract

A 41-year-old woman was referred to our emergency department with a 3-day history of upper abdominal pain. We diagnosed her with diabetic ketoacidosis (DKA) after laboratory tests indicated a blood glucose level of 569 mg/dL, positive urine ketone bodies and metabolic acidosis. Plain computed tomography (CT) scan revealed free gas surrounding the porta hepatis and gastric pylorus, which disappeared on the subsequent contrast-enhanced CT scan. Upper gastrointestinal endoscopy demonstrated no perforations; therefore, we assumed that the free gas was caused by spontaneous pneumoperitoneum. The patient had fulminant type 1 diabetes mellitus, as evidenced by her glycated hemoglobin A1c level of 6.9%, reduced insulin secretion and negative islet-specific autoantibodies. Pneumoperitoneum did not recur with conservative treatment, and DKA improved with intravenous fluids and insulin administration. Conservative management of DKA with spontaneous pneumoperitoneum may be considered if the patient's general condition is stable and there are no signs of peritoneal irritation.

INTRODUCTION

Spontaneous pneumomediastinum, known as Hamman's syndrome, can be secondary to diabetic ketoacidosis (DKA) [1–3]. However, there are few reports of spontaneous pneumoperitoneum accompanying DKA, except in cases of mediastinal emphysema migrating into the abdominal cavity in Hamman's syndrome [4]. In addition, although there are some reports of cases of non-occlusive mesenteric ischemia (NOMI) combined with DKA resulting in free gas in the abdominal cavity [5], there have been no reports of pneumoperitoneum combined with DKA without abnormalities in the abdomen. Here, we present the case of a patient with fulminant type 1 diabetes mellitus who developed DKA and spontaneous pneumoperitoneum.

CASE REPORT

A 41-year-old Chinese woman with no pre-existing medical conditions, including diabetes mellitus or positive urine glucose, was referred to the emergency department with symptoms of upper abdominal pain, nausea and vomiting that persisted for 3 days.

She weighed 45 kg at the time of arrival, measured 150 cm in height and had a body mass index of 19 kg/m^2. She was mildly disorientated, and her respiratory rate increased to 34 breaths/min. She had a dry oral cavity and mild tenderness from the epigastric region to the right hypochondrium, but Murphy's sign was negative. We diagnosed her with DKA after laboratory testing (Table 1) revealed hyperglycemia with a blood glucose of 569 mg/dL and ketoacidosis, based on observations of urine ketones (4+), pH 7.084 and HCO_3^- 2.7 mmol/L. The glycated hemoglobin A1c (HbA1c) level was 6.9%, and insulin secretion was depleted when she was admitted to our hospital and even after completing the acute treatment for hyperglycemia. Both anti-glutamic acid decarboxylase and anti-islet antigen-2 antibodies were negative. Additionally, pancreatic enzymes, such as pancreatic amylase, lipase and elastase, were elevated. She was diagnosed with fulminant type 1 diabetes mellitus because she met the following diagnostic criteria: ketoacidosis within 1 week of symptom onset, blood glucose level > 288 mg/dL, HbA1c level < 8.7%, decreased urinary C-peptide concentration at onset and no response according to the glucagon test result. She was negative for anti-GAD and anti-IA-2 antibodies, as is usually observed in fulminant type 1 diabetes mellitus [6].

Plain computed tomography (CT) of the trunk (Fig. 1A and B) showed free gas in the periportal area of the porta hepatis and gastric pylorus. Meanwhile, no gas was observed in the mediastinum in the chest CT scan. Contrast-enhanced CT scan (Fig. 2A and B)—performed 2 h after the plain CT scan—revealed that the free gas in the same area had disappeared, and there was no evidence of increased pancreatic fatty tissue density, swelling of the pancreas, intestinal necrosis. There were also no gallstones, enlargement of the gallbladder or dilation of the bile duct. We chose conservative treatment based on these findings and the absence of any peritoneal irritation symptoms.

On day 5 of admission, when the patient's health improved, an upper gastrointestinal endoscopy was performed, which indicated no perforation or ulcer in the stomach or duodenum. We diagnosed her with spontaneous pneumoperitoneum, and she resumed eating, with no recurrence of abdominal pain.

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Table 1. Laboratory testing of the patient.

Urine analysis	**Blood chemistry**	**Sodium**	**128 mEq/L**		
Urinary glucose	4+	Albumin	5.6 g/dL	Chloride	104 mEq/L
Urinary ketone body	4+	C-reactive protein	0.04 mg/dL	Potassium	6.2 mEq/L
		Aspartate aminotransferase	27 U/L	Calcium	8.4 mg/dL
Complete blood cell count	Alanine aminotransferase	31 U/L	Inorganic Phosphorus	2.9 mg/dL	
White blood cells	14 540/μL	Lactate dehydrogenase	403 U/L	Glucose	569 mg/dL
Hemoglobin	15.3 g/dL	Alkaline phosphatase	73 U/L	Glycated albumin	27.0%
Platelet	31.4 × 10^4/μL	γ-glutamyl transpeptidase	22 U/L	Glycated hemoglobin A_{1c} (HbA_{1c})	6.9%
		Amylase(AMY)	110 U/L	Serum osmolarity	309 mOSM/kg H_2O
Arterial blood gas	P-AMY	94 U/L	Thyroid stimulating hormone	0.46 μIU/mL	
pH	7.084	Lipase	113 U/L	Free T4	1.09 ng/dL
pO2	120.6 mmHg	Elastase	508 ng/dL	C-peptide immunoreactivity	<0.20 ng/mL
pCO2	9.2 mmHg	Urea nitrogen	13 mg/dL	Anti-GAD antibody	<5.0 U/mL
HCO3	2.7 mmol/L	Creatinine	0.70 mg/dL	Anti-IA-2 antibody	<0.6 U/mL
BE	−24.8 mmol/L	Uric acid	7.0 mg/dL		

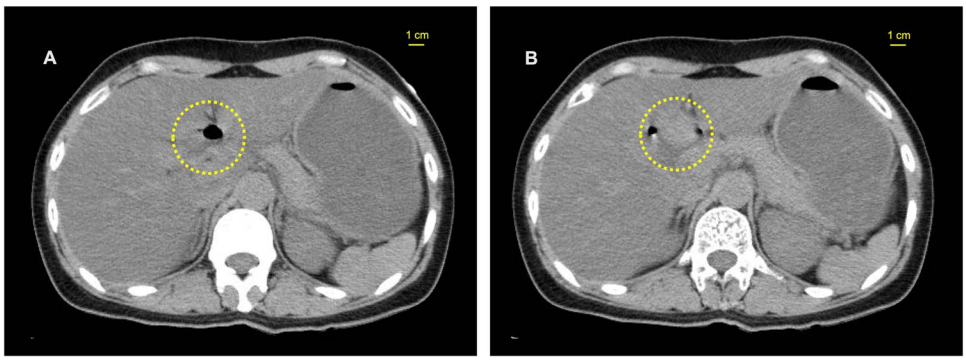

Figure 1. Plain CT of the trunk showing free gas surrounding the porta hepatis (**A**) and the pyloric side of the stomach (**B**). The dotted circles indicate the free gas. CT, computed tomography.

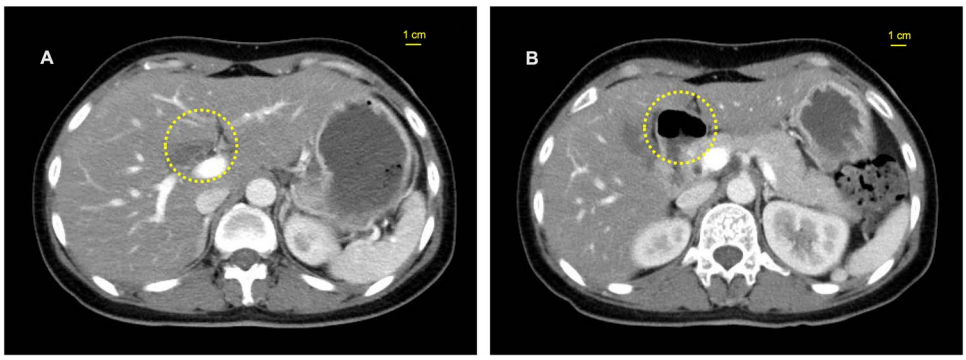

Figure 2. Contrast-enhanced computed domography (CT) scan of the trunk showing the disappearance of free gas detected in plain CT scan surrounding the porta hepatis (**A**) and the pyloric side of the stomach (**B**) with no signs of intestinal necrosis. The dotted circles show the area where the free gas disappeared.

Moreover, her metabolic acidosis and hyperglycemia improved with intravenous fluids and insulin administration, and subcutaneous intensive insulin therapy was initiated. Her blood glucose levels returned to normal range, and then she was discharged on day 17.

DISCUSSION

Spontaneous pneumomediastinum, known as Hamman's syndrome, can be secondary to DKA; however, spontaneous pneumoperitoneum accompanied by DKA is rare, and to our best knowledge, this is the first report of peritoneal emphysema complicated by DKA that resolved spontaneously with conservative treatment. Meanwhile, there are several reports of DKA with abnormal abdominal free gas, but these are frequently caused by non-occlusive mesenteric ischemia (NOMI) [5]. Most of these instances required surgical treatment with bowel resection and rarely resolved spontaneously.

Pneumoperitoneum is known to be caused by thoracic or gynecological factors in addition to abdominal organ abnormalities

and medical interventions. However, the CT scan of the trunk revealed no gynecological disease, and she had no history of abdominal surgery. Therefore, it was unlikely that these were caused by pneumoperitoneum [7]. A case involving a patient with DKA, reported another mechanism of abdominal free gas development—pneumomediastinum migration into the abdominal cavity [4, 8]. Another study reported that hypoperfusion caused by dehydration induced NOMI, which was alleviated by restoring the circulatory dynamics through rehydration therapy [9].

The reason for the abdominal free gas development in our patient is unclear, but the following possibilities are considered based on previous reports:

(i) There was pneumomediastinum, which was not detectable through CT imaging, and air leaked into the abdominal cavity.
(ii) DKA-induced mild circulatory failure caused a decrease in organ blood flow, resulting in NOMI, which was relieved by conventional medical treatment.
(iii) Repeated vomiting damaged the wall of the gastrointestinal tract, allowing air to leak through small perforations.

In this case, the pneumoperitoneum improved with careful follow-up without surgery. No reports have indicated the criteria for selecting between conservative and surgical treatment for pneumoperitoneum; however, a low sequential organ failure assessment (SOFA) score ($\leq$ 3 points) is reportedly a useful indicator for conservative treatment in NOMI cases [10]. In our case, although it is unclear whether the pneumoperitoneum was due to NOMI, the SOFA score was 1 point, indicating that conservative treatment was appropriate. According to some reports, if symptoms are mild, localised in the upper abdomen, and hemodynamic status is stable, conservative treatment with frequent and thorough monitoring should be considered.

DKA with pneumoperitoneum can be a severe condition requiring laparotomy. However, if a patient lacks signs of peritoneal irritation and is in a stable health condition, as in this case, it may improve spontaneously, and a careful follow-up may help avoid unnecessary surgery.

In summary, we described a case of spontaneous pneumoperitoneum in conjunction with DKA in a patient with fulminant type 1 diabetes, who was successfully treated with conservative measures. Knowledge of this rare disease and an accurate diagnosis based on clinical findings might help avoid unnecessary surgery.

ACKNOWLEDGEMENTS

None.

CONFLICT OF INTEREST STATEMENT

None declared.

FUNDING

None.

ETHICAL APPROVAL

No ethical approval was required.

CONSENT

We explained the possibility of publishing a paper to the patient and obtained verbal consent. We were unable to track her after discharge and could not obtain written consent. We have taken patient anonymity into full consideration in accordance with the journal policies (https://academic.oup.com/omcr/pages/Policies).

GUARANTOR

Kenji Toyoshima.

DATA AVAILABILITY

Data sharing is not applicable to this article as no data sets were generated or analyzed in this article.

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