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Case Report

An elderly patient with dementia presenting euglycaemic ketoacidosis due to oesophageal stricture-associated eating habits

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Abstract

An 85-year-old man with dementia was referred to our department because of problematic eating habits, which made it difficult to manage his diabetes mellitus. The patient exhibited euglycaemic ketoacidosis, with a clinical course and biochemical parameters more indicative of starvation ketoacidosis rather than diabetic ketoacidosis. After correcting the acid–base imbalance with dextrose-containing fluids, he attempted to resume oral intake; however, throat noises consistent with reflux were noted during food ingestion. Imaging and pathological studies revealed a stricture with wall thickening in the mid-oesophagus attributed to non-specific chronic oesophagitis. After receiving hypercaloric fluids to calm oesophageal inflammation, an endoscopic bougienage was performed, enabling oral intake and achieving appropriate glycaemic control with oral glucose-lowering agents. This case illustrates the importance of closely monitoring eating behaviours, which is crucial for identifying comorbid organic abnormalities, without being limited by cognitive bias in clinical reasoning that problematic dietary changes are highly prevalent in dementia patients.

Keywords: dementia; eating habits; diabetes mellitus; euglycaemic ketoacidosis; oesophageal stricture; gastroesophageal reflux disease

Introduction

The incidence of dementia is predicted to double every twenty years worldwide [1]. Patients with dementia frequently change their eating habits or food preferences [1, 2]. As the disease progresses, they may resist or neglect to eat, which can lead to dysphagia, frailty, weight loss, and aspiration pneumonia, which are becoming more familiar with Japan's ageing population [3]. The growing number of elderly Japanese individuals living alone poses challenges for dietary management and monitoring swallowing abilities within the community (https://www.ipss.go.jp/index-e.asp). In these circumstances, other coexisting organic diseases that have clinical manifestations and courses similar to dementia may go unrecognised and overlooked.

In healthy individuals, it typically takes 14 days of starvation to reach maximum circulating ketoacid levels [4]. However, dehydration, low glycogen storage, and excessive alcohol consumption can accelerate this catabolic process, leading to the early onset of euglycaemic ketoacidosis, such as starvation ketoacidosis (SKA) and alcoholic ketoacidosis (AKA) [4]. Nursing facility residents have malnutrition and dehydration at the rates of 85% and 51%, respectively [2], indicating a higher susceptibility to SKA. Patients taking sodium-glucose co-transporter-2 inhibitors (SGLT-2i) are predisposed to developing euglycaemic diabetic ketoacidosis (DKA), which may be difficult to distinguish from SKA without a comprehensive understanding of the clinical context, particularly in the setting of pre-existing diabetes mellitus (DM) [4, 5].

This report describes an elderly patient initially suspected of having abnormal eating habits due to dementia, but close monitoring of his eating behaviour revealed oesophageal stricture accompanied by SKA.

Case report

An 85-year-old man living alone was referred to our department for the management of his DM treatment, which had become complicated by dietary issues. He had a history of gastroesophageal reflux disease (GERD) in his 30s and angina pectoris requiring stent placement in his 70s. The patient was diagnosed with DM in his 40s and commenced insulin therapy in his 60s. He had been treated with multiple daily insulin injections and oral glucose-lowering agents, including empagliflozin, which had maintained his glycated haemoglobin in the 7% range over the past year until three months before his initial visit to our department. There was no history of gastroesophageal surgery or radiation therapy. He had abstained from alcohol and cigarettes throughout his life.

Two months earlier, the patient underwent craniotomy for a meningioma in the parasphenoid region. Postoperatively, he developed moderate cognitive impairment (the revised Hasegawa

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Table 1. Laboratory characteristics of the patient on arrival.

Variable	Result	Reference
Serum albumin (g/dL)	3.5	4.1–5.1
Serum urea nitrogen (mg/dL)	26.5	8.0–20.0
Serum creatinine (mg/dL)	0.84	0.46–0.79
Serum uric acid (mg/dL)	9.2	3.7–7.8
Serum sodium (mEq/L)	143	134–147
Serum potassium (mEq/L)	3.6	3.6–5.0
Serum chloride (mEq/L)	107	96–107
pH	7.430	7.350–7.450
$PaCO_2$ (mmHg)	29.9	32.0–46.0
PaO_2 (mmHg)	104.6	74.0–108.0
HCO_3^- (mEq/L)	19.5	21.0–29.0
Base excess (mEq/L)	−4.1	−2.0 – 2.0
Anion gap (mEq/L)	17	10–14
Total ketone bodies (μmol/L)	4720	26–122
Acetoacetic acid (μmol/L)	1080	13–69
β-Hydroxybutyric acid (μmol/L)	3640	≤76
Fasting blood glucose (mg/dL)	183	70–109
Glycated haemoglobin (%)	7.6	4.6–6.2
Glycated albumin (%)	22.2	11.0–16.0
Anti-GAD antibody (U/mL)	< 0.4	< 5
C-peptide immunoreactivity (ng/mL)	1.0	0.6–1.8
Urinary glucose	+	−
Urinary ketone bodies	3+	−

Abbreviations: $PaCO_2$, carbon dioxide partial pressure; PaO_2, oxygen partial pressure; HCO_3^-, bicarbonate ion; GAD, glutamic acid decarboxylase.

dementia scale: 15/30 points) and was admitted to a nursing home. According to his patient's caregiver, he was dissatisfied with life in the facility, refused meals, and left to return home of his own volition two days prior to his initial consultation. A representative from the comprehensive community support centre urged him to seek medical attention as he had not eaten anything and suspended all DM treatment regimens since leaving the nursing home.

Upon arrival, his vital signs were as follows: blood pressure 116/50 mmHg, pulse rate 90 beats/min, and respiratory rate 20 breaths/min. Laboratory tests revealed hypoalbuminaemia and elevated serum urea nitrogen, creatinine, and uric acid levels (Table 1). The anion gap, serum β-hydroxybutyric acid, and glucose levels imply euglycaemic ketoacidosis. A compensatory mechanism by respiratory alkalosis prevented the development of acidaemia despite the underlying metabolic acidosis. Anti-glutamic acid decarboxylase antibody titre and serum C-peptide level indicated a non-insulin-dependent state of type 2 DM.

The patient experienced several days of dietary deficit, leading to a diagnosis of SKA rather than DKA (Table 2). After admission, SKA and dehydration were corrected with an infusion of dextrose-containing balanced crystalloid solutions, resolving the acid–base imbalance by the second hospital day. Oral intake was then attempted; however, food-refluxing noises came from the throat during food ingestion. A barium meal study revealed that a tapered narrowing in the mid-oesophagus prevented solid objects from passing despite excellent swallowing function (Fig. 1A). A computed tomography scan showed circumferential oesophageal wall thickening at the site of the stricture (Fig. 1B–D) and a hiatus hernia (Fig. 1E). Neither mediastinal mass lesion nor lymph node enlargement was observed. The gastroesophageal endoscopy confirmed the stricture 30 cm from the incisors (Fig. 2A). Oesophageal biopsy specimens revealed a non-specific chronic oesophagitis (Fig. 2B and C). Mucosal cultures showed no evidence of aerobic bacteria or fungi.

Table 2. Differences in laboratory findings, clinical history, and management between starvation, diabetic, and alcoholic ketoacidosis.

Variable	Starvation ketoacidosis	Diabetic ketoacidosis	Alcoholic ketoacidosis
Blood glucose	low or normal (< 100 mg/dL, variable if having diabetes)	elevated (≥ 250 mg/dL, variable if taking SGLT-2i)	normal or low (sometimes mildly elevated)
Urinary glucose	low or negative	positive	negative
Serum insulin	low or normal	low to undetectable	normal or low
Serum potassium	low	normal to elevated	variable, often low
Serum ketone bodies	mildly elevated	elevated	mildly elevated
β-HB/AcAc ratio	≈ 3	≈ 3	4 to 7
Clinical history	History of prolonged fasting or malnutrition	History of diabetes, often with infection, stress, missed insulin,or taking SGLT-2i	History of chronic alcohol use,recent binge drinking, vomiting, or poor nutrition
Ketoacidosis management	Glucose administration with 5–10% dextrose	Fluid resuscitation with ~0.9% NaCl and iv insulin infusion (*variable if taking SGLT-2i*)	Glucose administration with 5–10% dextrose

Abbreviations: β-HB, β-hydroxybutyric acid; AcAc, acetoacetic acid; SGLT-2i, sodium-glucose co-transporter-2 inhibitor; 0.9% NaCl, isotonic saline; iv, intravenous.

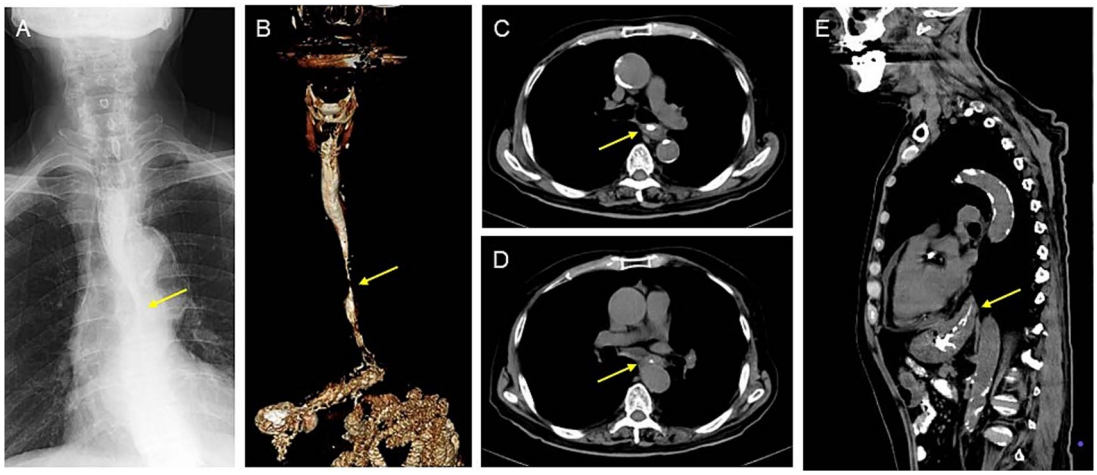

Figure 1. (A) A barium meal examination and (B) a three-dimensional computed tomography (CT) scan revealed a stricture with gradual narrowing of the calibre in the mid-oesophagus (*arrows*). (C, D) circumferential wall thickening in the mid-oesophagus and (E) a hiatus hernia are confirmed on the CT images (*arrows*).

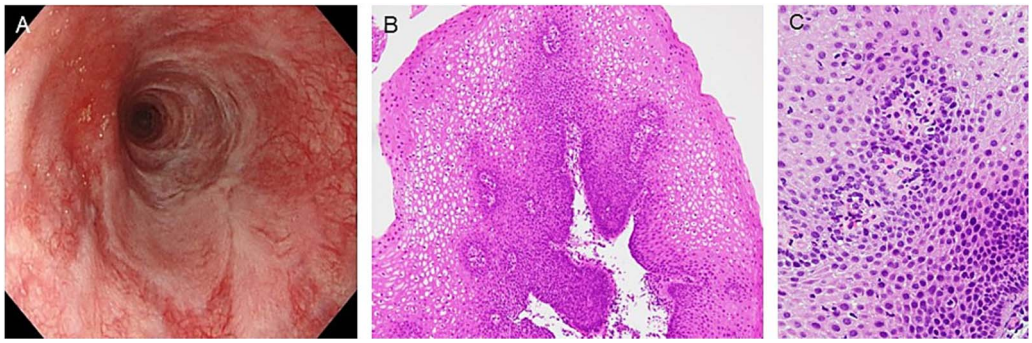

Figure 2. (A) Upper gastroesophageal endoscopy shows oesophagal stricture with mucosal oedema and breaks. (B) Oesophageal biopsy samples revealed thickening of the lamina propria papillae and dilation of blood vessels within the epithelium (× 100). (C) Hyperplasia of basal and parabasal cells and infiltration of inflammatory cells are observed, with no significant disruption in cell arrangement (× 400).

Oral intake was restricted, and a high-calorie infusion was implemented to calm oesophageal inflammation and restore nutritional status. The patient subsequently underwent endoscopic bougienage, which enabled the resumption of oral intake. His DM was managed with metformin, sitagliptin, repaglinide, and voglibose, achieving adequate glycaemic control without the need for insulin therapy.

Discussion

In Japan's ageing society, a significant issue is that many dementia patients with dysphagia occupy a large number of hospital beds for extended periods post-treatment [6, 7], which is partly driven by the lower actual cost of hospitalisation under the universal healthcare system. This problem is especially prevalent in small to medium-sized hospitals that provide community-based medical and emergency care for elderly individuals and that also suffer from a blurred line between medical care and the caregiving role. Although problematic dietary changes are highly expected in dementia patients, coexisting organic abnormalities may be identified through closely monitoring eating behaviours. However, these facilities, constrained by limited resources and high patient volumes per physician, risk delivering more standardised care without fully considering individual patient needs [8]. This case report underscores the importance of avoiding cognitive biases–such as anchoring, availability, and hassle biases–when making clinical reasoning, regardless of the working environment. Care must be personalised to each patient's circumstances in collaboration with paramedical staff.

Several benign conditions can cause oesophageal strictures, including peptic oesophagitis, eosinophilic oesophagitis, ingestion of caustic substances, radiation exposure, and post-surgical complications [9, 10]. Similar to dementia, benign oesophageal strictures are common in individuals over 60 and exhibit symptoms including dysphagia, poor appetite, and weight loss [9, 10], which can be alleviated through dilation procedures such as endoscopic ballooning, bougienage, topical steroids, stenting, incisions, and surgery [11]. In this case, the patient presented with a mid-oesophageal stricture, likely due to chronic peptic oesophagitis associated with GERD. GERD accounts for 60%–80% of all causes of benign oesophageal strictures and is most typically found in the lower segment [12]. The patient's hiatal hernia may have modified peptic acid reflux, explaining why the stricture occurred in a relatively uncommon location.

SGLT-2i are among the first-line oral glucose-lowering agents for DM. Beyond glycaemic control, SGLT-2i have gained attention for their organ-protective effects, especially in the treatment of chronic kidney disease and heart failure, regardless of DM status [13]. The increasing use of SGLT-2i warrants more awareness of the risk of developing euglycaemic DKA [5]. Accurate diagnosis of euglycaemic ketoacidosis is crucial to prevent hypoglycaemia and expedite recovery from acid–base imbalance [4, 5]. This patient was predisposed to developing euglycemic DKA due to the discontinuation of insulin therapy and a history of SGLT-2i use. In this

case, the patient's serum ketoacids levels and fractions helped identify the subtype of euglycaemic ketoacidosis. The ratio of β-hydroxybutyric acid to acetoacetic acid is generally around 3 in the DKA and SKA, compared with 4 to 7 in AKA [14]. Total ketone body levels were relatively lower in SKA than in DKA, consistent with this patient's finding upon admission. Therefore, oesophageal stricture-related starvation was likely the primary factor in the acid–base imbalance.

Consent

Written informed consent was obtained from the patient to publish this case report.

Guarantor

The corresponding author, Hiroaki Iwasaki, is nominated as the guarantor of this case report.

Contribution

Hiroaki Iwasaki and Seie Morita were involved in the patient's clinical care at Minamiyamato Hospital. Hiroaki Iwasaki conducted the case description and literature review and wrote the article, while Seie Morita reviewed and provided feedback on the manuscript.

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Conflict of interest

The authors declare no conflicts of interest associated with the present case report.

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Ethical Approval

The Ethics Committee of Minamiyamato Hospital approved the present case report (No. MYHP 2024–001).

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