CASE REPORT

A case of Takotsubo cardiomyopathy induced by accidental hypothermia and diabetic ketoacidosis

Yoichi Katayama,¹ Toru Hifumi,² Junichi Inoue,² Yuichi Koido²

¹Department of Emergency Medicine, Sapporo Medical University Hospital, Sapporo City, Hokkaido, Japan

²Department of Critical Care Medicine and Trauma, National Disaster Medical Center, Tachikawa, Tokyo, Japan

Correspondence to
Dr Yoichi Katayama,
yoichikata@gmail.com

SUMMARY

Takotsubo cardiomyopathy is known to be triggered by some stress. We report the first case of Takotsubo cardiomyopathy induced by hypothermia and diabetic ketoacidosis (DKA). A 59-year-old woman was brought to our emergency department. Upon examination, she was found to be lethargic and disoriented. The body temperature was 30.9°C, blood pressure was 86/68 mm Hg, blood glucose level was 1018 mg/dl. Urinalysis revealed the presence of ketones and arterial blood gas analysis revealed pH 6.87. She was diagnosed with DKA and accidental hypothermia. An ECG demonstrated a J wave (V₄₋₅) and ST-segment elevation (V₃₋₅), and an ultrasound cardiogram revealed contractile failure of the left ventricular wall at the apex with hypercontraction of the basal segment. Coronary artery disease was ruled out by coronary angiography, and the diagnosis of Takotsubo cardiomyopathy was confirmed. Physicians should keep in mind that Takotsubo cardiomyopathy can be induced by accidental hypothermia and DKA.

BACKGROUND

Takotsubo cardiomyopathy is known to be triggered by emotional and physical stress; however, we report the first case of Takotsubo cardiomyopathy induced by hypothermia and diabetic ketoacidosis (DKA).¹⁻³

DKA is serious acute complications of diabetes. The earliest symptoms of marked hyperglycaemia are polyuria, polydipsia and weight loss. As the degree or duration of hyperglycaemia progresses, neurological symptoms, including lethargy, and focal signs, which can progress to coma in later stages, can be seen.⁴ Furthermore, there is a report that Takotsubo cardiomyopathy is one of the DKA complications.⁵

Hypothermia is defined as a core temperature below 35°C and is precipitated by some causes; cold exposure, hypothyroidism, adrenal insufficiency, DKA, sepsis, alcohol and substance abuse.⁶ Patients with DKA are known to have the potential of developing hypothermia due to the inability of adequate amounts of glucose to enter cells,⁷ and it is reported that stress of hypothermia develop Takotsubo cardiomyopathy.⁸

To our knowledge, this is the first case of Takotsubo cardiomyopathy induced by accidental hypothermia and DKA.

CASE PRESENTATION

A 59-year-old woman was brought to our emergency department with impaired consciousness and

low blood pressure. She felt chest pain a day before admission. The patient had no history of major illness or disability. However, her family found her lying unresponsive in her room on one particular summer day. On presentation, her Glasgow Coma Scale score was 10/15 (E4V2M4). The vital signs were as follows: body temperature 30.9°C; blood pressure 86/68 mm Hg; heart rate 69 bpm and respiratory rate, 23 breaths/min.

Heart sounds were clear and the rhythm was regular without murmurs. She was severely dehydrated and her tongue was dry. Blood gas analysis demonstrated significant metabolic acidosis with the following readings: pH 6.875; pCO₂ 8.6 mm Hg; pO₂ 203 mm Hg; FiO₂ 0.45; HCO₃ 1.6 mmol/l; base excess -30.4 mmol/l and anion gap 35.8 mmol/l. Laboratory test findings were as follows: blood glucose 1018 mg/dl; creatine kinase level 1951 IU/l and troponin I 7.94 ng/ml. Urinalysis showed ketonuria, and an ECG demonstrated a J wave (V₄₋₆) and ST-segment elevation (II, III, aVF, V₃₋₅; figure 1). The patient was in shock and this condition was diagnosed as hypovolaemia, which was induced by DKA. Warmed crystalloid fluid, regular insulin and ceftriaxone (2 g/day) were administered. The patient was intubated and mechanically ventilated for airway protection. The patient's body temperature was maintained at 35°C with blanket and infusion of warmed crystalloid fluid. A cardiologist was consulted because the patient's condition did not improve even with the infusion of 3500 ml of acetate-Ringer solution. Contractile failure of the left ventricular wall at the apex was detected by transthoracic echocardiography (figure 2). Acute myocardial infarction was ruled out by coronary angiography (figure 3), and a diagnosis of Takotsubo cardiomyopathy was confirmed. The patient was haemodynamically stabilised with an intra-aortic balloon pump and administration of catecholamines.

OUTCOME AND FOLLOW-UP

The patient's hospital course was uncomplicated. Level of troponin I lowered gradually after admission. On day 2, the ECG demonstrated no J wave or ST-segment elevation. On day 4, she was discharged from the intensive care unit. On day 13, a balloon-like left ventricular wall motion abnormality had disappeared.

DISCUSSION

We discuss the pathogenesis of this case of Takotsubo cardiomyopathy induced by hypothermia and DKA.

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Figure 1 ST-segment elevation in leads II, III, aVF, V₃₋₅, and J wave in lead V₄₋₆.

Figure 2 Contractile failure of the ventricular wall at the apex.

DKA is known to be induced by various forms of physical stress (infection, discontinuation of insulin therapy, acute pancreatitis, new-onset type 1 diabetes).⁴ This patient did not have a history of elevated levels of blood glucose; however, she was diagnosed with new-onset type 1 diabetes during her hospital stay. In DKA, decreased effective insulin concentrations and

increased counter-regulatory hormones concentration (catecholamines, cortisol, glucagon and growth hormone) lead to hyperglycaemia (the inability to consume glucose), the release of free fatty acids and ketosis.⁴ The mechanism of hypothermia in DKA is probably related to the inability of glucose to endocytose because of the insulin deficit, which leads to a lack of substrate for cellular heat production.⁷

Hypothermia is induced by a variety of factors including adrenal dysfunction, thyroid dysfunction, alcohol consumption, intracranial disease and drug intoxication.⁶ Because the patient did not have a history of exposure to cold, adrenal dysfunction, thyroid dysfunction, intracranial disease, inability to consume glucose and glucose availability were finally considered as causes of hypothermia in this patient.

Takotsubo cardiomyopathy is followed by illness excessive secretion of catecholamine, such as pheochromocytoma,⁹ higher levels of plasma catecholamine are observed in patients with Takotsubo cardiomyopathy. Direct injury to the myocardium due to excessive concentration of catecholamines is associated with the onset of Takotsubo cardiomyopathy.³ Patients with postoperative hypothermia are also reported to have increased catecholamine concentrations.¹⁰

Sudip *et al* reported that DKA can coexist with Takotsubo cardiomyopathy⁵ and indicated that myocyte injury due to the acidosis of DKA is associated with Takotsubo cardiomyopathy.

This patient developed hypothermia due to DKA, and then developed Takotsubo cardiomyopathy following myocyte injury due to acidosis associated with DKA and catecholamine secretion induced by hypothermia.

Figure 3 The figure shows no coronary artery disease.

Learning points

- ▶ Takotsubo cardiomyopathy can be induced by diabetic ketoacidosis and hypothermia.
- ▶ Takotsubo cardiomyopathy may be associated with direct injury to the myocardium due to excessive catecholamine concentration.
- ▶ In the event of patients presenting with an unknown source of shock, Takotsubo cardiomyopathy could be one of the causes.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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