Case Report

A Therapeutic Challenge of STEMI with concurrent DKA in Emergency Department

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Abstract

Diabetic ketoacidosis (DKA) with acute ST-elevation myocardial infarction (STEMI) is rare but associated with significantly increased mortality and morbidity. We report a case of a 58-year-old woman with poorly controlled diabetes mellitus (HbA1c 18.1%) who presented with chest pain and was found to have inferior STEMI and DKA. Despite thrombolysis, she developed ventricular fibrillation and succumbed before rescue percutaneous coronary intervention (PCI) could be performed. This case illustrates the diagnostic and therapeutic challenges in managing coexisting DKA and STEMI, particularly in resource-limited settings. High HbA1c, lactic acidosis, and volume overload were contributing factors to poor outcomes. Fluid resuscitation, a mainstay of DKA management, must be balanced against the risk of exacerbating cardiac dysfunction. Early recognition, multidisciplinary coordination, bedside ultrasound for fluid assessment, and timely access to PCI-capable centres are essential for improving survival. Policy efforts should aim to expand STEMI network service hours to provide equitable care for critically ill patients presenting outside standard operational times.

Keywords: *DKA, STEMI network, thrombolysis*

INTRODUCTION

The concurrent incidence of diabetic ketoacidosis (DKA) in patients with acute ST-elevation myocardial infarction (STEMI) is estimated to be 0.5%. This combination is associated with a high mortality rate, with patients with both STEMI and DKA being five times more likely to die than those with STEMI alone. It is also linked to several other complications, such as cardiogenic shock, cardiac arrest, renal failure, prolonged ICU stay, increased length of hospital stay, and increased healthcare costs.[1] Various factors can trigger DKA, the most common being the omission or inadequacy of insulin. Other potential causes include infections, burn injuries, polytrauma, and acute medical conditions, such as cardiovascular events (myocardial infarction (MI) and stroke) and gastrointestinal issues (bleeding and pancreatitis). Additionally, surgeries and certain medications, such as corticosteroids, beta-blockers, diuretics, anticonvulsants, and antipsychotics, may also precipitate DKA.[2,3] When DKA and STEMI coexist, it is challenging to determine which condition occurs first, as STEMI is a known trigger for DKA, and DKA may also precede STEMI. Management is further complicated by the lack of formal guidelines and limited access to interventional cardiology units offering percutaneous coronary intervention (PCI).

CASE PRESENTATION

A 58-year-old woman presented to our emergency department (ED) at 4 a.m. with complaints of ongoing chest pain for 4 hours, with a pain score of 9/10 on a numeric rating scale associated with diaphoresis and multiple episodes of vomiting. Her medical history included hypertension, hyperlipidemia, and poorly controlled diabetes mellitus, with a recent glycated haemoglobin A1c (HbA1c) of 18.1%. On arrival, she was alert, with a Glasgow Coma Scale score of 15. Her blood pressure (BP) was 98/49 mmHg, and her heart rate was 43 beats per minute (bpm). She was not tachypnoeic and had an oxygen saturation of 95% on room air. Breath sounds were equal on chest auscultation, with no crepitations or rhonchi. Her heart sounds were S1 and S2, not muffled, with no murmur. Her abdominal examination was unremarkable, and she had no lower limb oedema. An immediate 12-lead electrocardiogram (ECG) revealed a second-degree AV nodal block Mobitz type I heart block, with a heart rate of 48 bpm, along with ST elevation in leads II, III, and aVF with posterior and right-sided involvement (Fig. 1).

Bedside cardiac ultrasound revealed moderate to poor left ventricular (LV) contractility and minimal pericardial effusion, with an aortic root measuring 2.8 cm. Bilateral lung ultrasound (US) revealed an A-shaped profile, and her inferior vena cava (IVC) was plethoric, measuring 2.0 cm. The intravenous (IV) administration of 1 mg of atropine improved her heart rate; however, her BP remained persistently low, with a mean arterial pressure (MAP) less than 65 mmHg, necessitating vasopressor infusion. She was then started on an acute coronary syndrome treatment protocol and thrombolysed with 35 mg of IV tenecteplase, which was dosed according to her body weight.

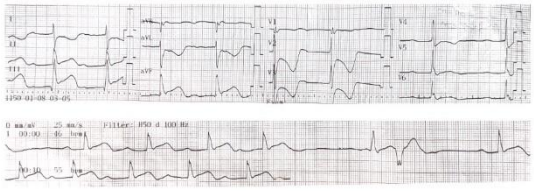

Figure 1: Standard 12 lead ECG on arrival

She was diagnosed with DKA when her random blood sugar (RBS) was found to be 46 mmol/L, with a ketone level of 3.3 mmol/L. Her venous blood gas revealed compensated high anion gap metabolic acidosis, with a pH of 7.37, bicarbonate (HCO3) of 11 mmol/L, and a base excess (BE) of -11.6 mmol/L, along with lactic acidosis at 6.6 mmol/L. Her anion gap was 24 mmol/L. The initial full blood count and renal profile were unremarkable. However, her liver function tests revealed deranged liver enzymes, with aspartate transaminase (AST) at 1055 U/L and alanine transaminase (ALT) at 306 U/L, suggesting ischemic hepatitis. Table 1 summarizes her initial blood parameters. A semierect anteroposterior chest radiograph (Fig. 2) revealed cardiomegaly, with a cardiothoracic index of 0.57.

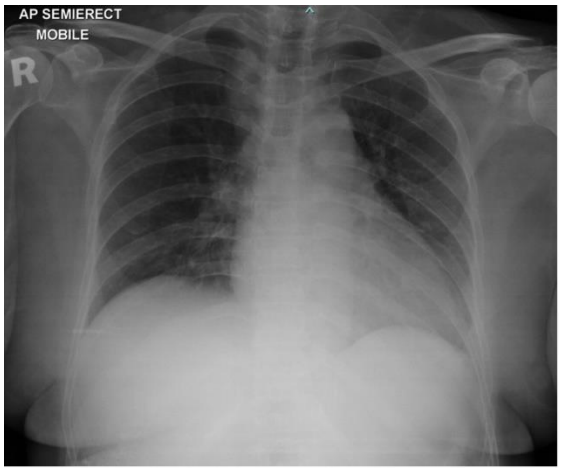

Figure 2: CXR on arrival

For her DKA, we administered a 250 mL bolus of normal saline (NS) over one hour. Upon reassessment, her IVC was further dilated. A normal saline drip at 1.3 mL/kg/hour and an IV insulin infusion at a fixed rate of 0.1 U/kg/hour were initiated. A subsequent repeat ECG (Fig. 3) revealed worsening ST elevation, indicative of fibrinolytic failure, despite her being free of pain. Rescue PCI was planned. However, her condition deteriorated further when she developed ventricular fibrillation, necessitating defibrillation and intubation. Her venous blood gas after spontaneous circulation returned revealed severe metabolic acidosis (pH 6.80, HCO3 5.4 mmol/L, BE -28.6 mmol/L) and lactic acidosis (19.4 mmol/L). Her blood glucose level remained persistently high at over 30 mmol/L, despite her ketone level decreasing to 0.4 mmol/L. Owing to her unstable condition, interfacility transfer for rescuing PCI was not feasible. She ultimately passed away.

Table 1: Summary of relevant initial blood investigation

Blood investigations		Results
Full blood count	WCC (10^3/ μL)	8.50
	Hb (g/dL)	11.5
	HCT (%)	34.5
	Plt (10^3/ μL)	309
Renal profile	Urea (mmol/L)	5.8
	Na (mmol/L)	131
	K (mmol/L)	3.5
	Creatinine (μmol/L)	80.7
	Cl (mmol/L)	95.6
Liver function test	Total Bilirubin (μmol/L)	20.1
	Albumin (g/L)	36.9
	ALT (U/L)	306
	AST (U/L)	1054
	ALP (U/L)	175
Venous blood gas	pH	7.37
	pCO_2 (mmHg)	19
	HCO_3 (mmol/L)	11
	BE (mmol/L)	-11.6
	Lactate (mmol/L)	6.6
Biochemistry	CK (U/L)	94
	RBS (mmol/L)	46

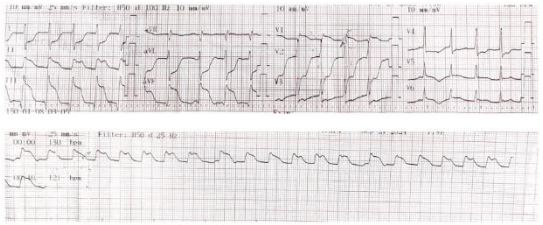

Figure 3: Standard 12 lead ECG post fibrinolytic treatment

DISCUSSION

Many factors increase mortality in patients with both DKA and STEMI. One factor identified in this case was significantly elevated HbA1c, which is a known risk factor for increased all-cause mortality and cardiovascular events. The optimal HbA1c range for minimizing mortality in people with diabetes mellitus is between 6.0% and 8.0%.[4] A higher HbA1c value is associated with increased severity of coronary heart disease (CHD) and DKA, where levels above 9% are more likely to have severe CHD and a 12-fold increase in DKA.[5,6] Diabetes accelerates atherosclerotic plaque formation and rupture, especially with hypertension, leading to myocardial infarction (MI) classified as Type 1 based on the Fourth Universal Definition of Myocardial Infarction.[7,8] Although there is a possibility of Type 2 MI precipitated by acute severe illness such as DKA, symptoms of myocardial ischemia with regional ST elevation (inferior leads with lead III higher than II, posterior leads and reciprocal changes) suggest primary coronary events involving right coronary artery occlusion rather than oxygen supply–demand mismatch, as seen in Type 2 MI.[8,9]

The pathophysiology of DKA is understood to be caused by decreased insulin secretion with concurrent increases in counter-regulatory hormones such as glucagon, catecholamines, cortisol, and growth hormone. Hyperglycaemia is sustained by increased gluconeogenesis, increased glycogenolysis, and decreased glucose utilization. Increased catecholamines, along with insulin deficiency, promote lipolysis, resulting in the production of excess glycerol and free fatty acids (FFAs). FFAs then feed into the ketogenic pathway, producing ketone bodies.[3] These typically unmeasured excess ketone bodies result in high-anion-gap metabolic acidosis. Lactic acidosis is also common in critically ill patients and may contribute to systemic acidaemia. Rodrigez-Villar et al. reported that a reduced arterial pH, where the threshold is 7.28, negatively impacts cardiac function and contractility.[10]

DKA is typically associated with marked dehydration with hypovolemia, where volume loss averages up to 10%.[2] NS has been most commonly used as the resuscitative fluid. Compared with NS, the use of balanced crystalloids (such as lactated Ringers) has no effect on the time to resolution of DKA, major adverse kidney events, or the incidence of hypokalaemia but may reduce post-resuscitation chloride levels.[11] Nevertheless, specific studies directly comparing fluid resuscitation strategies in DKA and STEMI patients are lacking. The severity of dehydration may be masked by pulmonary congestion due to acute heart failure (AHF) through several mechanisms. Reduced cardiac output activates the sympathetic nervous system, increasing vascular tone and redistributing blood from the venous reservoir to the effective circulatory volume. Activation of the renin–angiotensin–aldosterone system further promotes sodium and water retention.[12]

Replenishing fluid lost through osmotic diuresis while managing the potential for worsening congestion in the setting of AHF is challenging. Early multidisciplinary involvement (cardiology, endocrinology, and anaesthesiology), combined with the judicious use of ultrasound, can help tailor treatment strategies to address the competing demands of DKA and STEMI. US is widely available, non-invasive, and easy to use for guiding fluid status assessment. A recent SHoC-IVC study by Robert Dunfield et al. demonstrated that IVC point-of-care ultrasound (PoCUS) is feasible for spontaneously breathing patients. A dilated IVC > 2.5 cm with minimal or absent collapsibility is a highly specific and sensitive predictor of volume-overloaded fluid status in hypotensive patients.[13]

Even without DKA, patients with diabetes and STEMI have increased 30-day mortality. These patients frequently have a lower left ventricular ejection fraction and are more likely to develop cardiogenic shock and heart failure. Primary PCI is more effective than fibrinolysis, offering better survival outcomes despite a higher prevalence of multivessel disease and a greater degree of stenosis in patients with diabetes.[14] STEMI network services have been established in the Klang Valley, connecting non-PCI-capable centers with PCI-capable centers to provide primary PCI, which is available during weekday office hours.[15] There should be at least two transport team members in the patient compartment of the transport vehicle, who are fully equipped for oxygen and respiratory support, vital sign monitoring equipment, and medications with delivery devices. The lead escort must be competent in advanced cardiac life support (ACLS) and airway management and able to analyse hemodynamic data with subsequent adjustments of therapies.[16,17] The transport team must be equipped to provide intensive care equivalent to that of an intensive care unit setting throughout the journey.

CONCLUSION

STEMI with concurrent DKA is a challenging condition to manage in the ED. It is associated with a high mortality rate due to multiple factors can negatively affect cardiac function. Opting for PCI as a reperfusion strategy can increase the likelihood of successful reperfusion and improve patient outcomes. While an around-the-clock STEMI network might not be feasible

at present, policymakers should consider expanding the operating hours of STEMI network services to ensure availability at all times for selected high-risk cases.

CONFLICT OF INTEREST

We have no conflicts of interest to disclose.

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