

Reversible Cardiomyopathy and Acute Respiratory Distress Syndrome Masked by Severe Diabetic Ketoacidosis: A Diagnostic Pitfall for Primary Care Physicians

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Abstract

Diabetic ketoacidosis (DKA) is one of the most common metabolic emergencies family physicians see in practice. Although cardiac complications are rare, missing them can seriously worsen outcomes. A 53-year-old woman with poorly controlled type 2 diabetes presented with community-acquired pneumonia, severe DKA, and acute respiratory distress syndrome. Despite standard DKA management, she developed refractory hypotension and atrial tachycardia. Bedside echocardiography revealed severe global left ventricular dysfunction with an ejection fraction of 20%. She required Veno-arterial extracorporeal membrane oxygenation (VA-ECMO) and continuous renal replacement therapy. Her cardiac function recovered to 35-40%, allowing ECMO decannulation. She was discharged in stable condition after surgical repair of access-site bleeding. When a patient with DKA remains unstable despite fluids and insulin, think about reversible stress cardiomyopathy. Early cardiac evaluation and timely escalation to critical care can be lifesaving.

Categories: Family/General Practice, Cardiology, Emergency Medicine

Keywords: acute respiratory distress syndrome [ARDS], cardiogenic shock, case report, : diabetic ketoacidosis, echocardiography, pneumonia, primary care, primary care physicians, stress cardiomyopathy, va-ecmo

Introduction

Diabetic ketoacidosis (DKA) is among the most common and serious acute complications of diabetes mellitus. Primary care physicians often serve as the first point of contact for patients with diabetes, and early recognition of DKA is essential to prevent progression to life-threatening illnesses. The classic presentation includes polyuria, polydipsia, dehydration, Kussmaul respiration, and altered mental status. Standard care centers on fluid resuscitation, intravenous insulin, electrolyte replacement, and treatment of the precipitating cause, most often infection. This case is reported in accordance with the CARE guidelines: consensus-based clinical case reporting guideline [1], Gagnier, et al.

Severe metabolic stress and systemic inflammation can trigger transient myocardial dysfunction. As stated by Combes, et al. [2], clinicians may see patients who fail to improve despite appropriate DKA therapy, and recognizing the possibility of cardiogenic shock can save lives. Similar to findings of Evans, et al. and Gagnier, et al. [3,4], this report describes a patient with severe DKA and pneumonia who developed reversible cardiomyopathy, underscoring the need to widen the differential diagnosis when clinical response is slower than expected.

Case Presentation

A 53-year-old woman with a five-year history of type 2 diabetes mellitus presented to the emergency department with fever, confusion, and respiratory distress. She had not taken her oral hypoglycemic agents for two weeks. On examination, she was tachycardic, hypotensive, and tachypneic. She rapidly required endotracheal intubation and mechanical ventilation.

Laboratory investigations (Table 1) showed: blood glucose 28 mmol/L; arterial pH 6.95; bicarbonate 8 mmol/L; anion gap 28 mmol/L; and serum ketones strongly positive. Chest computed tomography showed extensive right upper and middle lobe consolidation consistent with severe community-acquired pneumonia. The diagnosis was severe DKA precipitated by respiratory infection.

Management included aggressive intravenous fluid resuscitation, continuous insulin infusion, potassium replacement, broad-spectrum antibiotics, and lung-protective ventilation. Despite these measures, she remained hypotensive and developed new-onset atrial tachycardia. Electrical cardioversion failed, and intravenous amiodarone was started.

Bedside echocardiography performed by the critical care team showed severe global left ventricular systolic dysfunction with an estimated ejection fraction of 20% (Figure 1). No valvular disease, regional wall motion abnormalities, or chronic structural changes were seen. The working diagnosis was mixed cardiogenic and septic shock with multi-organ dysfunction.

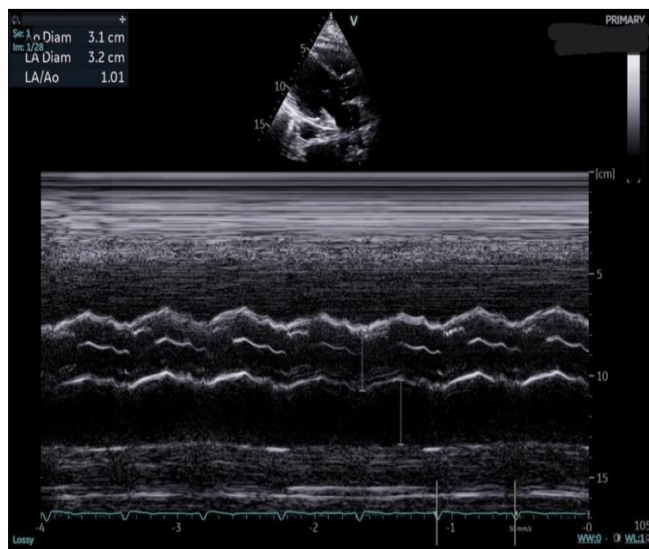
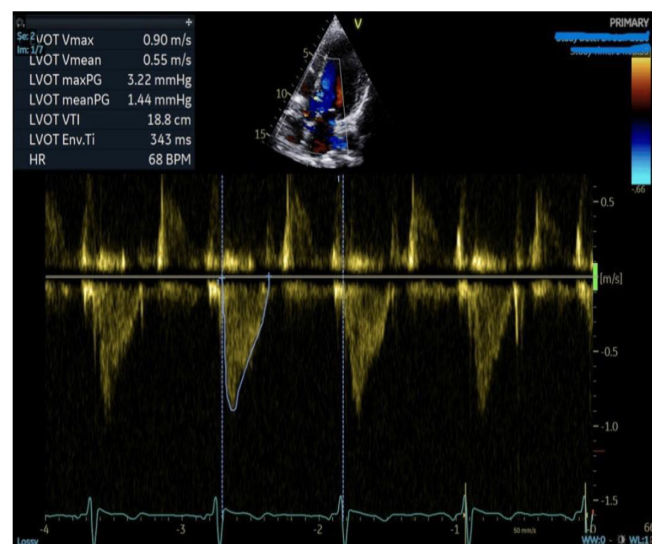
She was commenced on Veno-arterial extracorporeal membrane oxygenation (VA-ECMO) and continuous Veno-venous hemodiafiltration. Over the following 72 hours, her hemodynamic parameters improved markedly. Repeat echocardiography demonstrated recovery of left ventricular ejection fraction to 35-40%, and ECMO was successfully decannulated (Figure 2).

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Table 1: Lab test findings at admission.

Lab Marker (Normal Range)	Day 0 (04:22)	Day 1 (13:20)	Day 1 (14:38)	Primary Care Clinical Interpretation
pH (7.35-7.45)	7.380	7.347	7.333	Worsening acid levels in the blood due to poor tissue perfusion
PCO ₂ (35-45 mmHg)	40.5	38.7	41.7	Weak breathing response; loss of protective respiratory compensation
PO ₂ (83-108 mmHg)	129	289	173	High oxygen levels caused by the initial ventilator settings.
HCO ₃ (21-28 mmol/L)	24.0	21.2	22.1	Depleted buffering system due to heavy metabolic acid loading.
Hemoglobin (11.0-15.0 g/dL)	8.9	8.0	8.3	Severe baseline anemia due to the infection.
SpO ₂ (95-99 %)	98.5	100.7	98.9	Steady oxygen saturation while on the mechanical ventilator.
Potassium (3.4-5.0 mmol/L)	4.6	5.2	4.9	High risk of cell shifts balanced out by acute kidney strain.
Sodium (134-143 mmol/L)	137	138	137	Normal sodium balance maintained throughout.
Ionized Calcium (1.15-1.29 mmol/L)	1.16	1.15	1.14	Low calcium trend, which weakens heart pump power if left untreated.
Glucose (60-100 mg/dL)	174	285	269	Stubbornly high blood sugar showing metabolic escape under stress.
Lactate (0-4 mmol/L)	0.9	1.0	1.0	Completely normal lactate level, which falsely suggests the patient is stable.

Figure 1: Transthoracic ECHO Findings on Admission.

Figure 2: Transthoracic ECHO (TTE) Findings at Discharge.


Post-decannulation, the patient developed femoral access-site hemorrhage requiring surgical repair and transfusion. She was subsequently weaned from mechanical ventilation, transitioned to oral hypoglycemic therapy, and discharged in stable condition after a 21-day hospital stay.

Discussion

This case illustrates an uncommon but important complication of severe DKA. Family physicians are often the first to evaluate patients with acute diabetic complications, and awareness of atypical presentations can speed up appropriate referral. In this patient, persistent hypotension and arrhythmia despite adequate fluid resuscitation and insulin therapy were the key clues that prompted cardiac evaluation. She had no chronic heart disease, her ventricular function recovered rapidly (Figure 2), and the clinical context was one of severe metabolic stress and sepsis - all of which pointed to reversible stress or sepsis-associated cardiomyopathy rather than acute coronary syndrome very similar to clinical progression stated by Lyon, et al and McDonagh, et al, [5,6].

Several mechanisms may contribute to myocardial dysfunction in this setting: catecholamine excess from stress and acidosis exerts direct toxic effects on cardiomyocytes; pro-inflammatory cytokines suppress contractility; metabolic derangements including acidosis and electrolyte disturbances disrupt excitation-contraction coupling; and sepsis-induced cardiomyopathy is itself a distinct, reversible entity McDonagh, et al. [7].

The clinical picture resembles Takotsubo syndrome as described by Lyon, et al. [6], in which extreme emotional or physical stress triggers transient but severe left ventricular dysfunction. In this patient, the combined DKA and the inflammatory response to pneumonia probably produced a similar transient myocardial injury. Current heart failure guidelines stress that reversible cardiomyopathy must be distinguished from chronic systolic dysfunction. The key lesson is that not all shock in DKA is hypovolemic or septic. Myocardial dysfunction should be considered when a patient remains unstable despite appropriate rehydration and insulin therapy. Primary care physi-

cians should aim for early bedside echocardiography and prompt involvement of critical care and cardiology teams as recommended by Umpierrez, et al. and Marik, et al. [6,8].

Conclusion

Severe diabetic ketoacidosis may be complicated by profound but reversible myocardial dysfunction, resulting in refractory shock. Although hypovolemia and sepsis are common causes of hypotension in DKA, this case highlights the importance of considering stress- or sepsis-associated cardiomyopathy when patients fail to stabilize despite guideline-directed therapy. Bedside echocardiography was essential in identifying cardiogenic shock and enabling timely escalation to advanced hemodynamic support, leading to recovery of cardiac function.

Strengths and limitations: This report provides clinically relevant insight for frontline practice; however, as a single case, the findings are descriptive and cannot be generalized or used to guide definitive management strategies.

Learning Points: 1) Hypotension in diabetic ketoacidosis is not always attributable to hypovolemia or sepsis. 2) Stress- or sepsis-associated cardiomyopathy should be considered in patients with DKA who remain unstable despite appropriate therapy. 3) Bedside echocardiography is a valuable tool for early identification of cardiogenic shock in this setting. 4) Prompt multidisciplinary escalation and hemodynamic support can result in favorable and reversible cardiac outcomes.

Additional Information Author Contributions:

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Acquisition, analysis, or interpretation of data: Sajitha Prasad, Sangeeta Sharma, Pranav S. Prasad

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