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When an arterial blood gas analysis can save an unnecessary cardiac catheterization

Rakshya Poudyal, Saroj Lohani

ABSTRACT

Introduction: Diabetic Ketoacidosis can mimic electrocardiogram (EKG) changes of myocardial ischemia or infarction. The EKG changes of myocardial ischemia or infarction usually disappear after resolution of metabolic and electrolyte changes of DKA. We present a case in which a timely arterial blood gas analysis (ABG) prevented an unnecessary cardiac catheterization. Case Report: An 82-year-old female with past medical history of Type 2 Diabetes Mellitus was brought to Emergency Department (ED) after she was found with agonal breathing. Electrocardiogram (EKG) en route to ED showed ST segment elevation in V1-V3.Troponin was mildly elevated. Bedside Echocardiogram did not reveal any wall motion abnormalities. ABG revealed severe metabolic acidosis. Labs showed diabetic ketoacidosis (DKA) and hyperkalemia. Her EKG changes were thought to secondary to metabolic cause rather than a cardiac etiology, and cardiac catheterization was cancelled. She was treated with intravenous fluids and insulin. EKG done the next morning revealed resolution of ST segment changes. Conclusion: EKG changes mimicking MI can rarely be seen in DKA without myocardial damage and is called "pseudoinfarction". The EKG changes resolve after resolution of metabolic and electrolyte abnormalities in DKA. Various theories have been proposed for pseudoinfarction pattern in DKA. The most commonly cited one is secondary to hyperkalemia in DKA. Other proposed mechanisms are secondary to metabolic acidosis or coronary artery spasm resulting from severe acidosis. Pseudoinfarction pattern in DKA is critical to recognize, since systems of care now prioritize rapid triage in treatment of STEMI that often includes bypass of emergency care and direct transfer to catheterization labs.

Keywords: Acidosis, Diabetic ketoacidosis, Electrocardiogram, Hyperglycemia

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INTRODUCTION

Diabetic Ketoacidosis (DKA) is a serious complication of diabetes mellitus, characterized by ketoacidosis and hyperglycemia leading to serious complications [1]. Myocardial Infarction (MI) is a well-known precipitating cause and the leading coexisting cause of death in DKA [2]. DKA can mimic electrocardiogram (EKG) changes of myocardial ischemia or infarction. Types of EKG changes in DKA that can mimic MI include ST segment elevation [3], peak T waves [3], ST depression and T wave inversion [4]. The EKG changes of myocardial ischemia or infarction usually disappear within 24 hours after resolution of metabolic and electrolyte changes of DKA. It is important to understand this entity as misdiagnosis can lead to unnecessary procedure and treatment which can lead to complications. We present a case in which a timely arterial blood gas analysis (ABG) prevented an unnecessary cardiac catheterization

CASE REPORT

An 82-year-old female with past medical history of type 2 Diabetes Mellitus was brought to the Emergency Department (ED) of a tertiary hospital with chief complaint of altered mental status. As per the ED records, her welfare manager was visiting the patient at home and found her lying in bed with agonal breathing. EKG en route to the ED showed ST elevations in V1-V3 (Figure 1). ST elevation MI alert was activated and cardiac catheterization lab was informed immediately. On arrival to the ED, she was responsive to painful stimuli and was subsequently intubated. Vital signs included temperature of 98° F, pulse of 110/minute and blood pressure of 88/60 mm Hg. An internal jugular triple lumen catheter was inserted and she was started on norepinephrine infusion for low blood pressure. Physical examination revealed dry mucus membranes. Bedside Transthoracic Echocardiogram revealed no wall motion abnormalities. Arterial Blood Gas Analysis (ABG) was done which showed metabolic acidosis with pH of 6.9 (normal 7.35–7.45) and bicarbonate of 5 meq/L (normal 22-28 meq/L). Other pertinent labs included blood glucose of 600 mg/dL, anion gap of 45 meq/L (normal 7-17 meq/L), beta-hydroxybutyrate of 34.7 mg/dL (normal <3.02 mg/dL), potassium of 7.8 meq/L (normal 3.5-5.2 meq/L, troponin of 0.07 (normal <0.03) and creatinine of 2.3 mg/dL (normal 0.6–1.3 mg/dL). With these laboratory results, the patient was diagnosed with diabetic ketoacidosis.. With significant metabolic derangement and unremarkable echocardiogram and only mildly elevated troponin, her EKG changes were thought to secondary to metabolic cause rather than a cardiac etiology, and cardiac catheterization was cancelled. She was resuscitated with intravenous fluids in the ED and started on insulin drip. She was transferred to Intensive Care Unit (ICU) for further management. Her blood glucose improved to 200mg/dl after 2.5 L of intravenous fluids and insulin drip. EKG done following morning revealed resolution of ST elevation (Figure 2). Hyperkalemia and acidosis resolved with serum potassium of 3.8 mEq/L and serum bicarbonate 18 mEq/L the next morning.

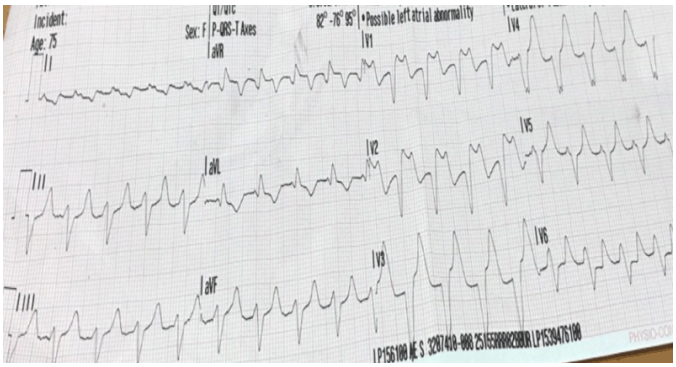

Figure 1: Electrocardiogram en route to emergency department.

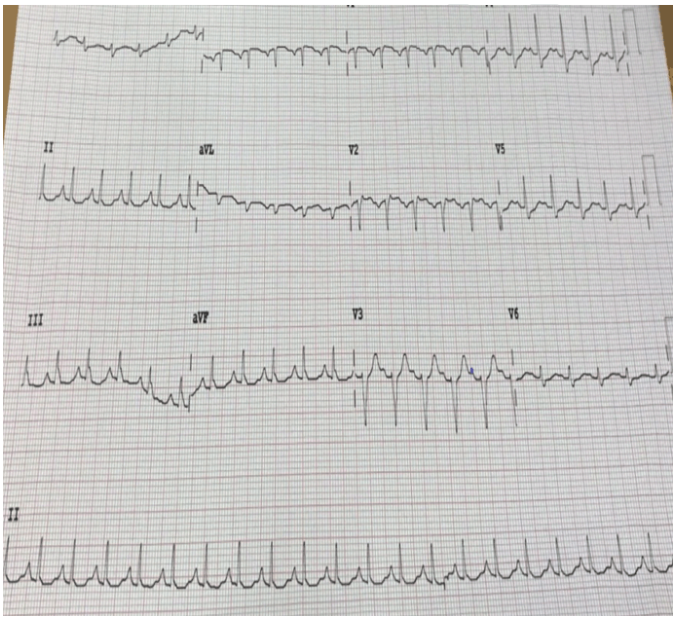

Figure 2: Electrocardiogram the next morning.

DISCUSSION

Diabetic ketoacidosis (DKA) is a serious complication of diabetes mellitus. Various factors can precipitate DKA such as infection, non-compliance with insulin, medications such as steroids and thiazide diuretics; myocardial infarction is among one of the precipitants. However EKG changes mimicking MI may be present in patients with DKA without myocardial damage and this entity is called “pseudoinfarction” [3, 5, 6].

Various theories have been proposed for pseudoinfarction pattern in DKA. The most commonly cited one is secondary to hyperkalemia in DKA. Various case reports have been published in which pseudoinfarction pattern was seen in patients with DKA and hyperkalemia; in these cases, as in ours, EKG changes resolved after potassium level returned to normal [3, 5]. However in one case report published by Aksakal et al. [6] pseudoinfarction pattern was seen with normokalemia (but also resolved after treatment of ketoacidosis). It is not clear whether ST elevation in hyperkalemia is a primary repolarization abnormality or artifact caused by merging of terminal R portion of QRS with T wave [5]. Hyperkalemia can cause the EKG changes through direct myocardial damage or through other mechanisms such as anoxia and acidosis [7].

Other mechanisms have also been proposed to explain pseudoinfarction pattern in DKA patients. ST segment elevation may arise from metabolic acidosis or other metabolic abnormalities in DKA [5]. Furthermore, severe acidosis can trigger coronary spasms which can lead to EKG changes which resolve with treatment of DKA [8].

Pseudoinfarction pattern in DKA is critical to recognize, since systems of care now prioritize rapid triage in treatment of STEMI that often includes bypass of emergency care and direct transfer to catheterization

labs [9]. Recognition of this STEMI mimic all out for rapid re- triage and intensive treatment of ketoacidosis.

CONCLUSION

Electrocardiogram changes in Diabetic ketoacidosis can mimic those of myocardial ischemia or infarction. It is important to recognize this entity in someone with risk factors as it can prevent inappropriate cardiac catheterization and complications secondary to inappropriate treatment.

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Author Contributions

Rakshya Poudyal – Substantial contributions to conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published

Saroj Lohani – Substantial contributions to conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published

Guarantor of Submission

The corresponding author is the guarantor of submission.

Source of Support

None.

Consent Statement

Written informed consent was obtained from the patient for publication of this case report.

Conflict of Interest

Authors declare no conflict of interest.

Data Availability

All relevant data are within the paper and its Supporting Information files.