

ECG Wave Changes and Non- ST Segment Elevation of Ischemic Heart Disease, Diabetes Mellitus Case Report

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ABSTRACT: Being best known for its neurological consequence, hypoglycemia is an important and not uncommon side effect with insulin or oral anti-diabetic therapy in patients with diabetes mellitus. However, hypoglycemia-induced myocardial infarction should be also taken into clinical consideration despite being rarely reported, especially non-ST segment-elevation myocardial infarction. Herein, we report a 68-year-old lady who underwent an episode of hypoglycemia manifested as reversible conscious disturbance and acute non-ST segment-elevation myocardial infarction, which was documented by typical ECG changes and dynamic cardiac-specific enzyme elevation, during long-term insulin therapy. The ST segment depression completely resolved as soon as the hypoglycemia was corrected. Subsequent coronary angiography showed no significant coronary stenosis responsible for the patient's ECG changes, suggestive of the strong relationship between hypoglycemia and myocardial infarction. This case is unique because of the unusual clinical manifestations.

KEYWORDS: Hypoglycemia _ Ischemia _ Non-ST-segment elevation myocardial infarction.

I. INTRODUCTION

Although its neurological consequence is common, hypoglycemia per se may cause cardiovascular complications, including bradycardia,1 atrial fibrillation,2 ventricular

arrhythmia,3 and rarely, myocardial infarction.4,5 There is limited information regarding hypoglycemia-induced myocardial infarction. To the best of our knowledge, hypoglycemia--induced non-ST segment-elevation myocardial infarction without significant coronary stenosis has never been reported in the literature. Herein, we report an unusual cardiovascular manifestation of hypoglycemia in a diabetic patient treated with insulin

II. CASE REPORT

A 68-year-old lady with past medical history of end-stage renal disease under regular hemodialysis, hypertension, and diabetic mellitus under insulin therapy for 10 years. However, she took a bath instead of taking meal immediately after insulin administration, about 4 hours before arriving at the emergency room. While dressing after bath, she felt general weakness, unable to Hypoglycemia-Induced Myocardial Infarction, and then lost consciousness. She was found lying on the floor by her family about 1 hour before admission. She was then sent to our emergency department by ambulance. On examination, the patient's body temperature was 36.7 °C, pulse rate was 84 beats/min, and blood pressure was 152/87 mmHg. Physical examination revealed bilateral fine rales over lung bases, bruise on the right forehead, and wet clothes due to sweating. Capillary blood glucose was 29 mg/dl. 12-lead electrocardiogram (ECG) revealed ST segment depression over leads II, III, aVF, and V3-6 (Figure 1A). The patient soon became conscious after intravenous infusion of 80 ml of 50% glucose water. She didn't complain of chest tightness throughout the treatment course. Therefore, physicians did not

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prescribe nitroglycerin for sublingual use. Follow- up ECG (Figure 1B) after hypoglycemia was corrected showed complete resolution of the previous ST segment changes irrespective of initial plasma levels of electrolytes. Laboratory data revealed white blood cell

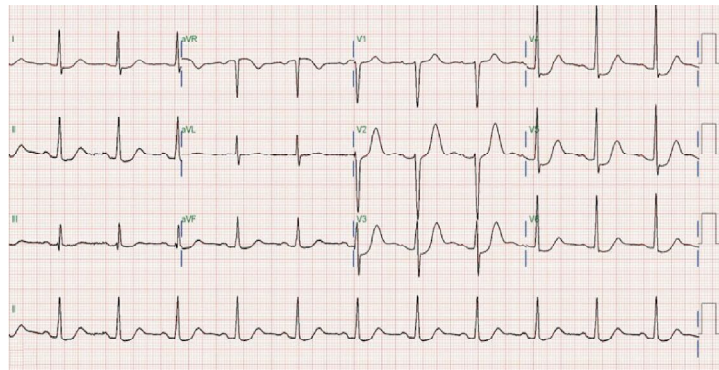
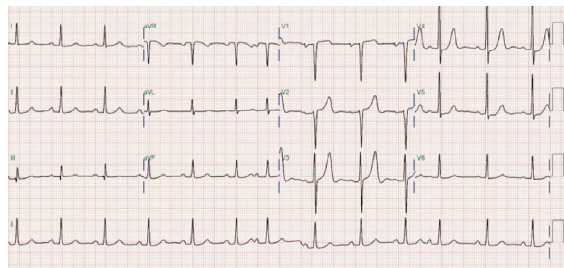


Figure 1. (A) Electrocardiograms revealed ST segment depression over anterolateral and inferior leads when the patient arrived at the emergency department;



B) Follow-up electrocardiograms showed complete resolution of the previous ST segment changes after hypoglycemia had been corrected.

Bio chemical levels of ischemic heart disease

S.No	Parameter	Values
1	White blood cell count	14.5 _ 103 cell/_L
2	hemoglobin level	10.9 g/dL,
3	platelet count	276 _ 103 cell/_L,
4	, potassium	2.9 mmol/L
5	calcium I	11.1 mg/dL
6	phosphate	4.6 mg/dL
7	magnesium	2.5 mg/dL
8	initial creatine kinase	109.3 U/L
9	CK level	1188 U/L;
10	CK-MB level	55.84 U/L
11	CK level	585 U/L
12	troponin T	13.55 ng/mL

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count of 14.5×10^3 cell/ μ L, hemoglobin level of 10.9 g/dL, platelet count of 276×10^3 cell/ μ L, potassium level of 2.9 mmol/L, calcium level of 11.1 mg/dL, phosphate level of 4.6 mg/dL, and magnesium level of 2.5 mg/dL. The initial creatine kinase (CK)-MB level was 109.3 U/L, and CK level was 1188 U/L; both were peaking at the presentation. The initial cardiac troponin T was 10.57 ng/ml. Follow-up cardiac biomarkers 9 hours later revealed CK-MB level of 55.84 U/L, CK level of 585 U/L, and troponin T level of 13.55 ng/mL. Echocardiography showed adequate global LV performance without regional wall motion abnormality at that time.

The patient was treated as acute non-ST segment-elevation myocardial infarction. Subsequent coronary angiography two days later revealed only non-obstructive coronary atherosclerosis without any evidence of unstable lesion over the left anterior descending artery, left circumflex artery, and right coronary artery (Figure 2). The patient was managed medically. Her hospital course was smooth, and she was discharged 6 days after admission.

III. DISCUSSION

Hypoglycemia can be associated with cardiovascular disease, including myocardial ischemia⁶⁻¹¹ and ST segment-elevation myocardial infarction,⁵ as well as QT interval prolongation and arrhythmia.^{12,13} However, hypoglycemia-induced non-ST segment-elevation myocardial infarction without obstructive coronary stenosis has never been reported in the literature. In our patient, myocardial infarction was diagnosed based on the universal definition of acute myocardial infarction. Hypoglycemia-induced non-ST segment-elevation myocardial infarction was documented because anterolateral and inferior lead ST segment depression on initial ECG resolved completely and immediately after hypoglycemia was corrected irrespective of mild electrolyte imbalance, and subsequent coronary angiography revealed only non-obstructive coronary atherosclerosis. None of them appeared as the culprit vessel for this index myocardial infarction. The pathophysiology of myocardial ischemia is an imbalance between myocardial oxygen demand and supply. Hypoglycemia may result in increased myocardial oxygen demand by causing sympathetic surge, releasing counter-regulatory hormones, such as epinephrine, norepinephrine, cortisol, glucagon, and growth hormone, that have immediate adverse cardiovascular effect by increasing afterload, inotropic and chronotropic status of the myocardium.

These adrenergic hormones also cause coronary vasoconstriction, impairing coronary blood flow and myocardial oxygen supply.¹⁴ In addition, hypoglycemia and coronary vasoconstriction limit the delivery of substrate (glucose and free fatty acids) to the myocardium, which further deteriorates the imbalance of myocardial energy supply and demand. Finally myocardial ischemia or eventually myocardial infarction ensue. Previous case reports regarding hypoglycemia-induced myocardial infarction mostly focused on ST segment elevation myocardial infarction. It is presumed that hypoglycemia-induced counter-regulatory hormone surge will cause coronary spasm and will react with betaadrenergic receptors on ventricular myocardium with resultant apical ballooning cardiomyopathy.¹⁵ Our case's development of non-ST segment-elevation myocardial infarction was probably not mediated by the aforementioned mechanisms. In patients treated with insulin, as seen in our case, some reports suggested that betaadrenergic sensitivity was lower than that of normal individuals or patients treated with oral anti-diabetic therapy. Therefore, we speculated that coronary spasm or stress cardiomyopathy cause by elevated counter-regulatory hormone was theoretically less frequent in insulin-treated diabetic patients. Furthermore, it has been suggested that prolonged myocardial ischemia will alter energy metabolism in myocardium, shifting from energy-efficient free fatty acid utilization toward oxygen-efficient glucose consumption to reduce oxygen depletion. However, hypoglycemia per se will inhibit this protective mechanism, and deteriorate myocardial ischemia resulting in myocardial infarction, just like in our patient, who had undergone prolonged hypoglycemia for more than one hour. In conclusion, hypoglycemia-associated non-ST segment elevation myocardial infarction should be included in the differential diagnosis of diabetic patients presenting with hypoglycemic coma.

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