Hypoglycaemia induced myocardial ischaemic ECG changes-a case report.

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ABSTRACT

A 76 year old female patient a diabetic for 15 years with coronary artery disease-on oral hypoglycaemic agents(sulfonylurea and metformin) and cardiac drugs(antiplatelets, statins, beta-blockers) was brought to the emergency department with unconsciousness, CBG was found to be 45mgs/dl. The initial ECG reveals a significant ST segment depression, more than 1mm in the anterolateral leads, compared with the old ECG of the same patient. The above ST segment changes resolved with correction of hypoglycaemia with 50% dextrose solution intravenously. No ST segment changes noted during her hospital stay. Hypoglycaemia-induced ECG changes are rarely documented. An altered balance between energy demand and supply in myocardial tissue has been postulated as possible cause of these ECG changes. So it is advisable to record an ECG to all patients who are presenting with symptoms of hypoglycaemia.

Keywords: hypoglycaemia, ECG, myocardial
INTRODUCTION

Hypoglycaemia related neuroglycopenic, autonomic complications are common and well documented. Hypoglycaemia may cause bradycardia (1), atrial fibrillation (2), ventricular arrhythmia (3) and rarely myocardial ischaemia (4). There is very little information regarding hypoglycaemia induced myocardial ischaemia/injury. Hence, we reported this cardiac manifestation of hypoglycaemia in a diabetic patient.

CASE DETAILS:

A 76-Years old lady with past medical history of diabetes for 15 years and coronary artery disease on cardiac drugs and oral hypoglycaemic drugs was found unconscious by her family members and brought to emergency department on 24.12.2015 at 11.30pm.

The day before admission this patient took all medication with poor food intake. Since 3 pm on the day of admission patient has profuse sweating, left sided chest pain, shivering, reduced verbal response.

On examination the patient was semi-conscious with a GCS OF 7/15. temperature was 36.7°C, pulse rate was 79 beats/minute, regular and blood pressure 140/70 mm of Hg on supine posture-left upper limb. capillary blood glucose was 45 mgs/dl. 12-lead electrocardiogram (ECG) revealed ST segment depression in lead I, II, AVL, V4, V5, V6 more than 1 mm (fig 1), which were new changes when compared with the recent old ECG of that patient. The patient soon becomes conscious after intravenous infusion of 55ml of 50% dextrose solution. There was no chest pain following correction of hypoglycaemia. Follow-up ECG (FIG 2) after hypoglycaemic correction showed complete resolution of the ST segment changes. Laboratory investigations showed total WBC of 11570 cells/mL with neutrophilic predominance, ESR - 60 mm at 1 hr, Haemoglobin - 11.2 gms, urea - 46mg/dl, creatinine - 1.5mg/L, serum electrolytes (Na - 136mEq/L, K - 5.7mEq/L). Calcium - 9.6mg/dl. Cardiac enzymes were not elevated. ECHO showed moderate left ventricular dysfunction (EF =). After an uneventful period of 3 days patient was discharged.

Figure 1: ECG at presentation – st depression)
Hypoglycaemia can be associated with cardiovascular disease including myocardial ischaemia and ST segment elevation myocardial infarction, as well as QT interval prolongation and arrhythmias. However hypoglycaemia 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. Hypoglycaemia induced non ST segment elevation was documented because anterolateral ST segment depression on initial ECG resolved completely and immediately after hypoglycaemia was corrected irrespective of mild electrolyte imbalance.

The pathophysiology of myocardial ischaemia is an imbalance between myocardial oxygen demand and supply. Hypoglycaemia may results in increased myocardial oxygen demand by causing sympathetic surge, releasing counter regulatory hormones, such as epinephrine, nor-epinephrine, cortisol, glucagon and growth hormone, that have immediate adverse cardiovascular effect by increasing afterload, ionotropic and chronotropic status of the myocardium. These adrenergic hormones also cause coronary vasoconstriction, impairing coronary blood flow and myocardial oxygen supply. In addition, hypoglycaemia 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 ischaemia or myocardial infarction ensue. Previous case reports regarding hypoglycaemia induced myocardial infarction are mostly focused on ST segment elevation myocardial infarction. It is presumed that hypoglycaemia induced counter regulatory hormone surge will cause coronary spasm and will react with beta-adrenergic receptors on ventricular myocardium with resultant apical ballooning cardiomyopathy.

CONCLUSION

Being best known for its neurological manifestations, hypoglycaemia is an important and not uncommon side effect with insulin or oral anti diabetic therapy in patients with diabetes mellitus. However hypoglycaemia induced myocardial infarction should be also taken in to clinical consideration despite being rarely reported, especially non ST segment elevation myocardial infarction.

REFERENCES