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Hypoglycemia-induced silent myocardial ischemia in a patient with diabetes

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Abstract

Myocardial ischemia can occur under various conditions. We report a diabetic patient with silent myocardial ischemia, which was manifested during hypoglycemia. A 72-year-old man with insulin-dependent diabetes was brought to the emergency room of Matsushita Memorial Hospital because of unconsciousness. The glucose level was 20 mg/dl and the patient became alert immediately after dextrose injection.

Electrocardiography showed mild ST-segment elevation on presentation, followed by terminal T wave inversion on the next day, without any chest symptoms or cardiac enzyme elevation. These electrocardiographic findings disappeared within five days. A critical stenosis in the left anterior descending coronary artery was later confirmed by coronary angiography, which was treated with stenting.

Keywords

diabetes; electrocardiography; hypoglycemia; silent myocardial ischemia

Introduction

Myocardial ischemia can develop in the absence of angina or anginal equivalents, especially in diabetic patients, under various conditions [1-3]. Several diagnostic procedures are available to detect silent myocardial ischemia, such as ambulatory electrocardiographic monitoring or stress testing with or without imaging [1-3]. We report a diabetic patient with silent myocardial ischemia that was recognized by hypoglycemia-induced electrocardiographic changes.

Case Report

A 72-year-old man was transferred to the emergency room of Matsushita Memorial Hospital because of loss of consciousness. The patient had been well until 3 days before admission, when general fatigue and appetite loss developed. His oral intake had been decreasing but he did not reduce or stop insulin for type 1 diabetes. The conscious level had deteriorated a few hours before admission, and he was found to have lost consciousness at home. The patient had a history of putamen hemorrhage, poorly controlled hypertension, and chronic kidney disease. Diabetes had been controlled with insulin self

administration: regular insulin three times a day (9 units, 7 units, and 7 units at meals) and long-acting insulin twice daily (5 units at breakfast and 9 units before sleep). Other medications included losartan 25 mg per day and hydrochlorothiazide 12.5 mg per day. He did not smoke, drink alcohol, or use illicit drugs.

On examination, his consciousness was impaired (Japan Coma Scale, 30). The blood pressure was 181/95 mmHg, the pulse was 87 beats per minute and regular, the body temperature was 35.7ºC, and the oxygen saturation was 100% while he was breathing ambient air. The heart sound and respiratory sound were normal. The capillary blood glucose level was reduced to 20 mg/dl. His consciousness became clear immediately after an intravenous bolus administration of 50% dextrose (16 g). Neurological examination revealed unremarkable findings. The patient was admitted for the adjustment of insulin dosage.

An electrocardiogram on admission showed normal sinus rhythm with a heart rate of 78 beats per minute; slight ST-segment elevation, along with abnormal Q waves, in leads V1 to V2 (Figure 1), although the patient did not report any chest symptoms. A chest radiograph showed a cardiothoracic ratio of 50% without pulmonary congestion. The blood levels of creatine and urea nitrogen were 5.79 mg/dl and 86 mg/dl. Lactate dehydrogenase and creatine kinase levels were also elevated to 331 IU/l and 581 IU/l, respectively. The reminder of the routine blood examination was normal. Computed tomography of the brain without the administration of contrast material showed no evidence of an acute intracranial process.

On the next day, electrocardiography showed newly developed terminal T wave inversion in V3 and V4 leads (Figure 1). Cardiology consultation was obtained. Echocardiography showed reduced contractility in the anteroseptal and apical walls of the left ventricle and the ejection fraction was calculated to be 48%. The level of creatine kinase increased (726 U/l), but the MB isoenzyme was within a normal range (23 U/l). The terminal T wave inversion improved 2 days after the hypoglycemic attack and completely disappeared 5 days later (Figure 1). No chest symptom had been observed in the clinical course.

One month after discharge, hemodialysis was initiated and coronary angiography was undertaken. A critical stenosis was detected in the mid portion of the left anterior descending coronary artery (Figure 2), which was treated with stenting.

Discussion

Serial changes in electrocardiographic findings developed after a hypoglycemic attack in our patient with diabetes. Given the severe coronary stenosis and left ventricular wall motion abnormalities, it is acceptable that a diagnosis of silent myocardial ischemia was made in our case. Furthermore, we may safely consider that the electrocardiographic changes were associated with hypoglycemia, although coronary angiography or myocardial perfusion imaging was not performed before the hypoglycemic attack or during the acute phase of hypoglycemia in our case.

Myocardial ischemia develops when oxygen supply (i.e., coronary blood flow) becomes inadequate to meet oxygen demand (i.e., myocardial oxygen consumption) [4,5]. The precise mechanism of hypoglycemia-induced myocardial ischemia remains uncertain in our case, but myocardial ischemia due to increased oxygen demand seems less likely because the heart rate and rate-pressure product were not significantly elevated (i.e., 87 beats per minute and 15,747 bpm-mmHg, respectively) during the

hypoglycemic attack, although the blood pressure was high (181/95 mmHg) that was unchanged from previous values on routine visits.

One mechanism of decreased oxygen supply may be explained by the fact that an extremely low level of glucose itself can be a direct cause of myocardial ischemia. In a study of 72-hour continuous glucose monitoring along with simultaneous cardiac Holter monitoring for ischemia in patients with diabetes and known coronary artery disease [6], myocardial ischemia was related to hypoglycemia defined as a blood glucose of <70 mg/dl. Furthermore, in canine experiments, hypoglycemia – approximately blood sugar of 30 to 50 mg/dl – increased myocardial ischemic injury following coronary artery occlusion, as assessed by electrocardiographic, enzymatic, and histological analyses [7]. Given the current patient's blood sugar level of 20 mg/dl on admission, it is intuitive that myocardial ischemia due to hypoglycemia could develop.

It should be noted that myocardium can utilize not only glucose but also other substrates as an energy source — free fatty acids, lactate, ketones, amino acids, etc [8,9]. It has been assumed that the multiple source availability protects the heart from direct energy deprivation caused by severe hypoglycemia [10]. Nonetheless, glucose is a major energy substrate in the presence of severe coronary disease [10-12]. Thus, it is reasonable to consider that limited glucose metabolism diminished the energy supply and ischemic electrical responses was accelerated by hypoglycemia in our case.

Other possible mechanisms of hypoglycemia-induced myocardial ischemia include endothelial responses, inflammatory mediators, and cytokines. It is reported that acute insulin-induced hypoglycemia increased plasma endothelin concentrations in patients with Type 1 diabetes, leading vasoconstriction and possible precipitation of myocardial ischemia [13] because endothelins are peptides that account for nearly all the resting tone in atherosclerotic coronary arteries [14]. Acute hypoglycemia also has provoked upregulation and release of vasoactive substances in adults with and without type 1 diabetes [15]. Furthermore, insulin-induced hypoglycemia deeply influences platelet function, causing an increase of platelet sensitivity to aggregating agents in vitro [16].

Figure

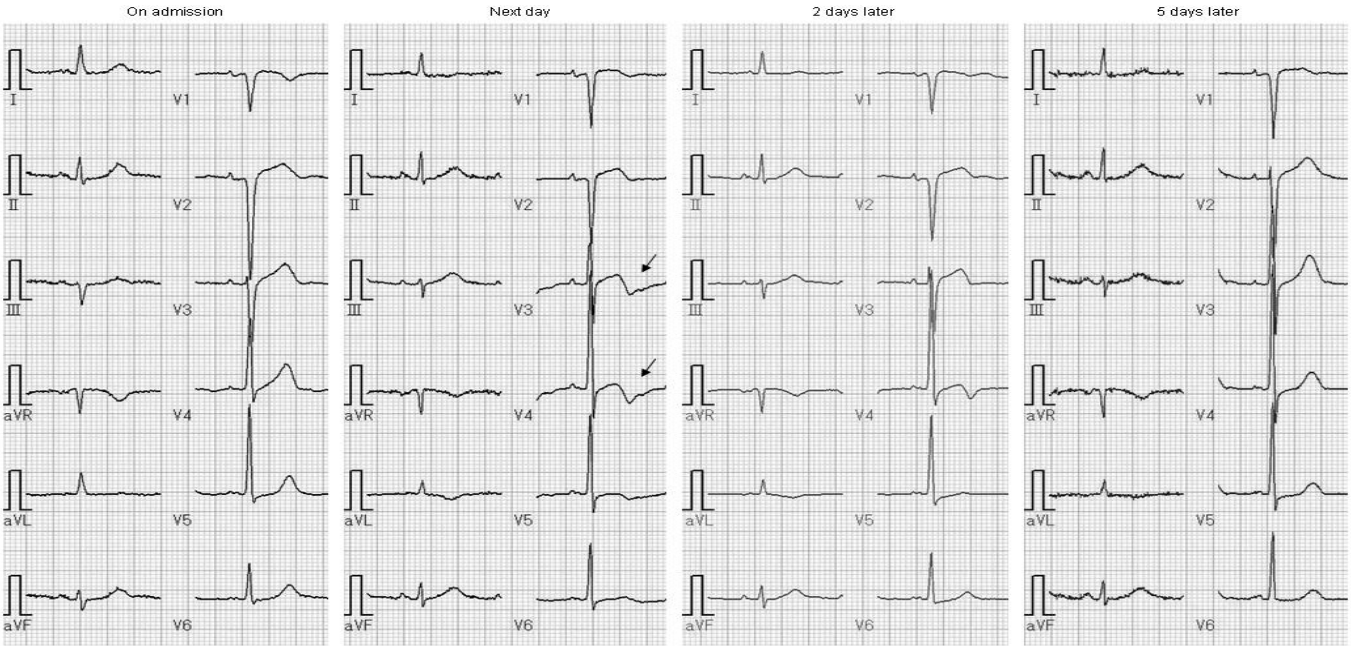

Figure 1: Serial changes in electrocardiography.

An electrocardiogram on admission shows mild ST-segment elevation in leads V1 to V2 with abnormal Q waves. Terminal T wave inversion, which was not observed on admission, is present on the next day (arrows). These findings improved two days after admission, and completely disappeared five days after admission.

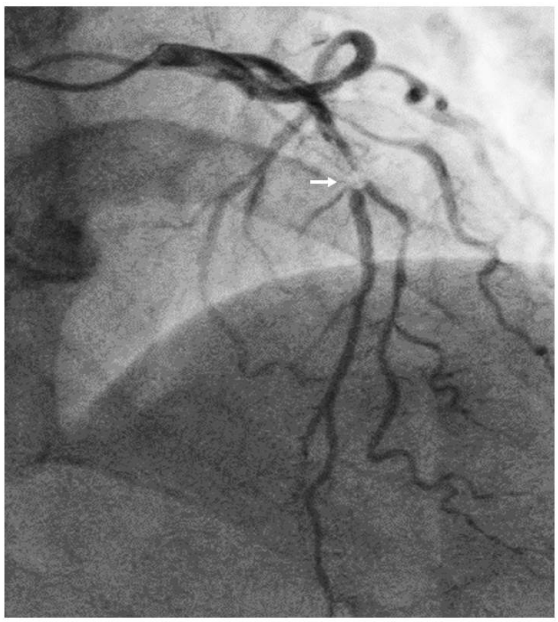

Figure 2: Coronary Angiography.

A critical stenosis is shown in the mid portion of the left anterior descending coronary artery involving the bifurcation of the first diagonal branch (arrow).

Conclusion

In conclusion, we experienced a case of diabetes, in which myocardial ischemia was clearly observed during a hypoglycemic attack. Our case highlights the importance of keeping in mind that hypoglycemia could cause myocardial ischemia in the management of patients with diabetes.

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