

Introduction:

Hypoglycemia is a very common side effect of insulin therapy and oral hypoglycemic agents. Occurrence of cardiac arrest as a result of severe hypoglycaemia has not been documented much in literature. We describe a interesting case of a type 2 diabetic patient admitted in intensive care unit following traumatic brain injury having cardiac arrest resulting from hypoglycaemia. A 59 years old type2 diabetic patient was transferred to our intensive care unit from a nearby hospital with history of traumatic brain injury for which decompressive craniotomy followed by duroplasty had already been done. His random blood sugars were uncontrolled for which he was on Insulin infusion as per our hospital protocol. He suffered cardiac arrest (asystole) at night for which no apparent cause could be identified except very low random blood sugar (<30mg/dl).

Case report:

A 59 years old type 2 diabetic and hypertensive patient was transferred to our intensive care unit from a nearby hospital with history of traumatic brain injury for which decompressive craniotomy followed by duroplasty had been done. Tracheostomy was already done in the previous hospital but he needed ventilator support. At admission his GCS was E4M1VT. Otherwise he was hemodynamically stable. His random blood sugars were uncontrolled for which he was on continuous Insulin infusion as per our hospital protocol. After two days of admission at around 5:45 AM he developed cardiac arrest and CPR was started according to ACLS protocol. The blood sugar were found to be very low(<20 mg/dl) . Patient revived after getting 100 ml 25% dextrose, 1 mg Epinephrine. Post cardiac arrest care was provided. During the post cardiac arrest analysis no apparent cause could be found except Low plasma glucose. So the cardiac arrest was attributed to hypoglycaemia.

Discussion:

Acute hypoglycaemia provokes pronounced patho- physiological responses, the important consequences of which are to maintain the supply of glucose to brain and promote hepatic production of glucose. Blood flow is increased to the myocardium, splanchnic circulation and the brain. Hypoglycemia and the rapid changes in blood glucose have been shown to increase counter-regulatory hormones such as epinephrine and nor-epinephrine, which may induce vasoconstriction and platelet aggregation, thereby precipitating myocardial ischemia. Autonomic activation

principally of the sympatho-adrenal system, results in end-organ stimulation and the profuse release of epinephrine which precipitates hemodynamic changes like tachycardia, increased peripheral systolic blood pressure, decreased central blood pressure and increased myocardial contractility with an increased ejection fraction³. The catecholamine induced myocardial contractility may induce ischemia in the myocardium in patients with CAD. The greater oxygen demand is not met because of not only the rigid vessels, but also endothelial dysfunction with failure to vasodilate.

There have been multiple case reports associating angina with hypoglycaemia. ECG changes, including ectopic activity, flattening of T-wave, ST depression, ventricular tachycardia, and atrial fibrillation, have been reported in cases of low plasma glucose¹.

Evidence is accumulating that severe hypoglycaemia can provoke adverse cardiovascular outcomes such as myocardial ischemia or cardiac arrhythmia like torsade 2. Episodes of severe hypoglycaemia are common during intensive therapy in type-1 and type-2 diabetes in the out-patient as well as in-patient setting. The challenge to the physician is to lower blood glucose to normal values to decrease the risk for long-term complications and at the same time minimize hypoglycaemia and hypoglycaemia-associated morbidity and mortality.

Conclusion:

Although not included in AHA guidelines as a cause of cardiac arrest (5H&5T), we believe hypoglycaemia can cause cardiac arrest independently and in this case after thorough investigation no other cause could be found except hypoglycaemia. So we strongly suggest hypoglycaemia can independently cause cardiac arrest and should be considered as a cause of cardiac arrest.

References:

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