

Cardiovascular Complications in Diabetic Ketoacidosis

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Diabetic Ketoacidosis (DKA) is one of the two important metabolic complications of diabetes, the other being hyperglycaemic hyperosmolar non-ketotic state (HHNS). DKA consists of the triad of hyperglycaemia, ketosis and acidosis; the diagnostic criteria being blood glucose level > 250 mg/dl, arterial pH < 7.3, bicarbonate level < 15 mEq/L and a moderate degree of ketonaemia and ketonuria. There has been little change in the mortality rate associated with DKA despite great improvements in our understanding of its pathophysiology and treatment. The cardiovascular problems in DKA include

1. Arrhythmias due to electrolyte imbalance.
2. Adverse effects of acidosis.
3. Acute myocardial infarction.
4. Pulmonary oedema.

DKA and Arrhythmias

Fluid and electrolyte imbalance is very common in DKA and typically manifests as loss of 5-8 litres of water, 400-700 mEq of sodium, 250-700 mEq of potassium and 30-50 mEq of magnesium. However, the relative loss of water is in excess of the electrolytes and hence most patients will have normal or even elevated levels of sodium, potassium and magnesium at the time of presentation, even though the body stores of these elements are almost always less than normal[3].

Potassium deficit is one of the most important of electrolyte imbalances seen in DKA, as it can lead to fatal arrhythmias. Initial levels of potassium may be normal or elevated, but by 12 hours of therapy most patients would become hypokalemic in the absence of replacement potassium, serial EDG's would be helpful in early detection of hypokalemia or hyperkalemia. Hypokalemia is associated with ECG abnormalities and cardiac arrhythmias, especially when the serum potassium level is below 3 mEq/L. The most common and perhaps the earliest ECG finding in hypokalemia is a prominent U wave, usually evident in leads II and III AVF. ST depression and T inversion can also occur, resembling myocardial ischaemia. The most common cardiac arrhythmias are atrial premature contractions, atrial tachycardia with or without AV block, SVT and ventricular premature contractions. Less common are AV junctional tachycardia or escape rhythm and AV block[5].

Hyperkalemia occurs in the early stages of DKA and the correlation between serum potassium levels and arrhythmias is more accurate in hyperkalemia than in hypokalemia. The earliest ECG change is a tall, tent-shaped peaked T-wave, which is often symmetrical with a narrow base; this occurs at potassium levels of 5.5-7.5 mEq/L and is best seen in leads II, III and V2-V5. At potassium concentrations of 7.5-10 mEq/L, there is reduction in amplitude of P-wave, prolongation of PR interval, depression of ST-segment and late disappearance of P-wave. With increasing hyperkalemia, potassium concentrations above 10-12 mEq/L, the QRS complex uniformly widens and at levels above 12 mEq/L, ventricular tachycardia or fibrillation, sine wave, slow ventricular escape rhythm or ventricular stand still occurs. Intraventricular conduction defects can occur which is usually diffuse and uniform and when associated with flat P-waves, closely resembles ventricular tachycardia. Intraventricular conduction defect is usually non-specific but patterns resembling RBBB, LBBB, LAHB or LPHB can also occur. Unlike conventional RBBB and LBBB in hyperkalemic RBBB and LBBB prolongation of the QRS complex occurs both in the initial and terminal portions. Hyperkalemia has no significant effect on the SA node, while varying degrees of AV block, though uncommon can occur. However, in the absence of discernible P-waves, it becomes difficult to diagnose AV block[5].

Occasionally, the ECG changes in hyperkalemia in DKA can mimic acute myocardial infarction viz. tall T-waves, QRS widening and bundle branch block. Hyperkalemia can also induce a current of injury called 'dialyzable current of injury', which can cause ST-segment elevation and thus be mistaken for acute infarction. Treatment of hyperkalemia reverses these changes.

Effect of Acidosis

The effect of acidosis on the heart depends upon the pH level. In mild acidosis, there is increased catecholamine release which is compensated by increased inotropy, chronotropy, cardiac output and peripheral vascular resistance. When acidosis is severe, i.e. pH is less than 7.2, the H⁺ ions have a direct cardiac depressant action. They cause negative inotropy, bradycardia, reduced cardiac output, peripheral vasodilatation and severe shock. Bicarbonates are usually not given in DKA; the only indications being pH less than 6.9 and arrhythmias due to hyperkalemia.

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DKA and Acute Myocardial Infarction

Acute myocardial infarction is not only increased in frequency in diabetics but is also associated with higher morbidity and mortality. This is due to the greater size of the infarct, the greater frequency of congestive heart failure and cardiogenic shock, arrhythmias and conduction abnormalities and the complication of ketoacidosis in diabetes. The relationship between diabetes and infarction is related mainly to the development of accelerated atherosclerosis and plaque rupture seen in diabetics. It is well known that factors leading to atherosclerosis and plaque rupture include hyperlipidaemias, hypertension, hyperinsulinaemia and abnormal blood viscosity; all of which are especially prevalent in diabetics. In addition, diabetes also favours intraluminal thrombosis which is related to abnormalities of coagulation, abnormally elevated levels of fibrinogen, increased levels of plasminogen activator inhibitor, abnormal endothelial function and abnormalities of platelet function with increased synthesis of thromboxane A₂ by platelets favouring platelet aggregation and increased platelet β thromboglobulin. They tend to prevent or retard spontaneous thrombolysis or increase the risk of reocclusion after thrombolysis. There are also metabolic abnormalities, in the form of increased catecholamine and cortisol secretion which causes lipolysis and increases the level of non-esterified fatty acids. Under normal conditions, 60-90% of myocardial energy requirement is met by oxidation of free fatty acids. During ischaemia, the heart shifts from aerobic to anaerobic metabolism and thus uses glucose instead of fatty acids as the primary fuel source. In DKA, insulin deficiency and high levels of ketones and free fatty acids inhibits glucose uptake by the cells and thus deprives the myocardium of energy utilisation during ischaemia. There is also excessive production of free radicals which enhance damage to the myocardium. An excess of catecholamines reduces the insulin secretory reserve and causes lipolysis and increases myocardial uptake of free fatty acids, which are toxic to the myocardial cells.

On the other hand, acute MI can be one of the major precipitating factors for DKA, along with infection, omission or inadequate insulin and intercurrent illnesses. DKA occurs in approximately 4% of diabetic patients with infarcts and may be the presenting symptoms. DKA complicating acute MI increases mortality which may approach 85%[7].

Acute MI complicating DKA is associated with a higher mortality predominantly due to pump failure.

DKA and Pulmonary Oedema

Rarely, pulmonary oedema in the absence of left ventricular failure has been reported in DKA. This is probably due to alteration in the alveolar capillary membrane permeability and may be a variant of adult respiratory distress syndrome (ARDS). The aetiology may be pulmonary vascular microangiopathy seen in diabetics. Vigorous fluid therapy can precipitate this condition[8].

A diagnostic dilemma is a patient of DKA with acute onset of dyspnoea. The differential diagnosis include acidotic breathing due to acidosis, acute left ventricular failure due to AMI, pulmonary oedema or ARDS. The initial approach would be a quick clinical examination followed by an ECG, an arterial blood gas analysis and a chest X-ray. ECG would show presence of AMI and ABG would show the severity of acidosis and of hypoxia. If the diagnosis is still in doubt, haemodynamic monitoring with a Swan-Ganz pulmonary artery catheter may be useful. ARDS can be diagnosed on the basis of the following features: diffuse alveolar infiltrates on X-ray chest, arterial/alveolar oxygen ratio less than 0.3, pulmonary capillary wedge pressure less than 18 mm Hg and total thoracic static compliance less than 40 ml/cm H₂O.

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