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Pseudo Myocardial Infarct – Electrocardiographic Pattern in a Patient with Diabetic Ketoacidosis

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ABSTRACT

Diabetic ketoacidosis is an extremely serious complication of diabetes mellitus. It arises because of a complex disturbance in glucose metabolism. There is usually a precipitating cause such as sepsis or myocardial infarction. If not recognised and appropriately treated, it can have devastating consequences. This is a case report of a patient with severe diabetic ketoacidosis and interesting electrocardiographic findings. The initial electrocardiographic (ECG) findings were suggestive of an acute myocardial infarction. The ECG changes normalised remarkably following initial management of the diabetic ketoacidosis. There have been only occasional reports of hyperkalemia causing electrocardiographic changes, closely resembling those of acute myocardial infarction.

Keywords: diabetic ketoacidosis, hyperkalemia

INTRODUCTION

Diabetic ketoacidosis is an extremely serious complication of diabetes mellitus that results from a complex disturbance of glucose metabolism. This syndrome occurs secondary to an absolute or relative insulin deficiency and is characterised by hyperglycaemia, ketonaemia, metabolic acidosis, electrolyte depletion and other derangements. There is usually a precipitating cause such as sepsis or myocardial infarction (Fig 1). If not recognised and appropriately treated, it can have devastating consequences. This is a case report of a patient with severe diabetic ketoacidosis and interesting electrocardiographic findings. The initial electrocardiographic (ECG) findings were suggestive of an acute myocardial infarction. The ECG changes responded remarkably to initial management of diabetic ketoacidosis. There have been only occasional reports of hyperkalemia causing electrocardiographic changes closely resembling those of acute myocardial infarction.

In a diabetic patient, ketoacidosis may be a complication of an acutely stressful event such as myocardial infarction or sepsis. One must not however, ignore the severe electrolyte derangements that may accompany diabetic ketoacidosis and be aware of how these derangements may effect the management of associated or triggering conditions.

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CASE REPORT

A 59-year-old female, NAP, presented to the Department of Emergency Medicine in an obtunded state. It was learnt from her relatives that she had been a diabetic for a number of years and was on daily insulin therapy as follows: a morning dose of subcutaneous Actrapid(R) insulin 20 units and Monotard(R) insulin 20 units and a nocturnal dose of subcutaneous Monotard(R) insulin 20 units.

On the day before presentation, she had experienced a few episodes of vomiting. She had not complained of chest or abdominal discomfort, diaphoresis or difficulty in breathing. On the morning of attendance she was difficult to arouse and was experiencing laboured breathing. She had no recent known febrile illness. We were not able to obtain any further history from her relatives and the patient.

Clinical examination at the Emergency Department revealed the patient's general condition to be poor. She was confused and drowsy with an axillary temperature of 36.7°C, a blood pressure of 77/39 mmHg as measured by the Dinamap(R) automatic blood pressure monitor and a pulse rate of 117 per minute. Her breathing was deep and laboured and she was extremely dehydrated. Her heart sounds were otherwise normal. The lungs were clear with equal breath sounds bilaterally and absence of crackles and wheezes. Her abdomen was soft, scaphoid and non-tender with no visceromegaly. Pedal oedema was not noted.

An urgent blood sugar by a Reflolux II(TM) reflectance glucometer revealed an extremely high reading above the maximum measurable limit (viz "HHH"). A blood specimen was immediately dispatched to the biochemical laboratory for full blood counts and urea/electrolyte/sugar determination. Her urine specimen collected tested strongly positive for ketones and glucose (both 3 +). A rapid arterial blood gas analysis by the Automatic Blood Gas Analyser (AVL 995 model)(M) done with the patient on an oxygen mask showed the following: blood pH 6.742, Pa Co_2 22.8 mmHg, Pa 0_2 125.1 mmHg, base excess – 34.1 mmol/L, standard bicarbonate 3.0 mmol/L and oxygen saturation 87.9% (Table I).

An electrocardiogram was done (Fig 1) and showed sinus tachycardia of 125/min, pathological Q-waves with markedly raised ST-segments in limb leads V1, V2 and V3. A chest X-ray done revealed a normal-sized heart shadow with no pulmonary congestion and no evidence of chest infection. A diagnosis of

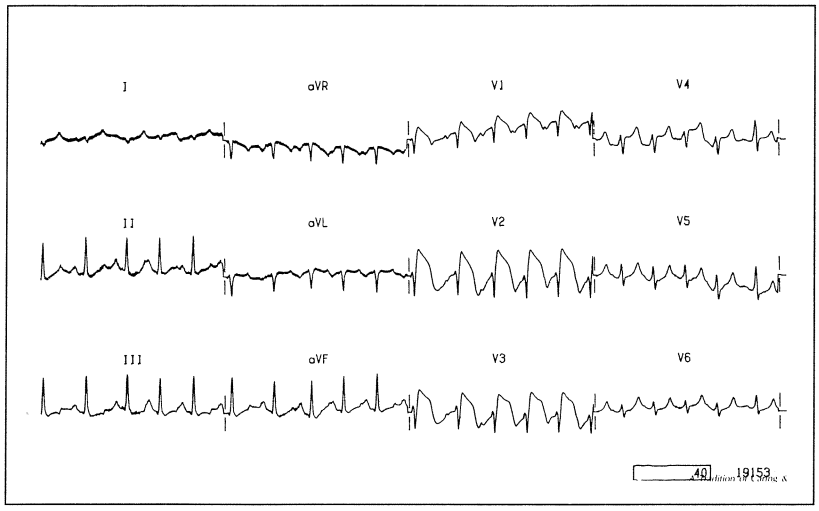

Fig 1 – The initial ECG done at the Emergency Department.

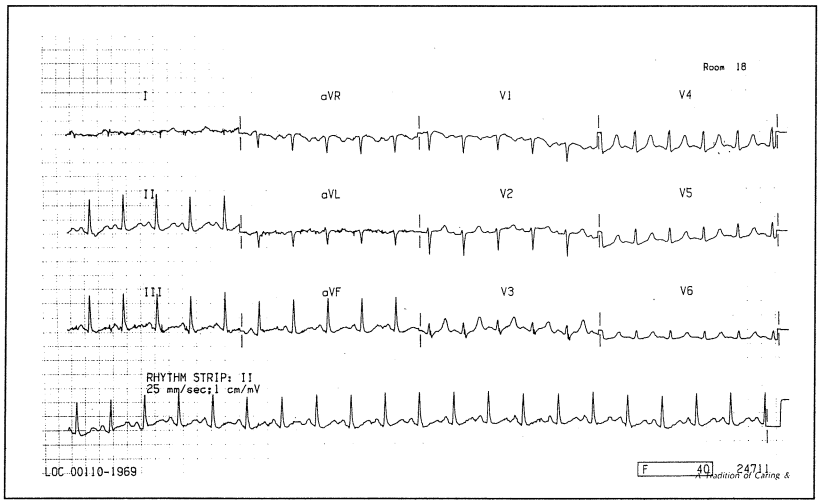

Fig 2 – The ECG done 3 hours after admission to the Intensive Care Unit.

Table I – Biochemistry and arterial blood gas results

	At ED (1520 hrs)	At ICU (1630 hrs)	Units	Normal range
Urea	17.1	30.3	mmo/L	2.8 – 7.7
Sodium	133	145	mmo/L	135 – 145
Potassium	7.2	4.8	mmo/L	3.3 – 4.9
Creatinine	93	102	μmol/L	44 – 141
Bicarbonate	< 5.0	< 5.0	mmo/L	19 – 31
Glucose	56.7	44.0	mmo/L	3.1 – 10.00
PH	6.741	6.905		7.35 – 7.45
PCO_2	22.8	15.5	mmHG	35.0 – 45.0
PO_2	125.1	118.2	mmHG	75.0 – 100
BE	– 34.1	– 28.5	mmo/L	– 2.0 to 2.0
0_2 Sat	87.9	95.5	%	95.0 – 100
SBC	3.0	5.1	mmo/L	2.8 to 7.7

Table II – Serum creatinine kinase level*

Date	22 May 94	23 May 94	25 May 94
CK levels	70 U/L	362 U/L	75 U/L
CKMB levels	-	32 U/L	-

* CKMB levels are not done if CK levels are < 164 U/L

acute myocardial infarction with diabetic ketoacidosis was made.

Fluid resuscitation with normal saline was commenced immediately and 100 mLs of 8.4% $HaHCO_3$ was infused. An intravenous insulin drip at 5 units/hr was set up. Altogether 1 litre of normal saline, 10 units of insulin and 100 mLs is of 8.4% $NaHC0_3$ were infused before the patient reached the Intensive Care Unit.

Her blood haematology and biochemistry results were as follows: Hb 11.5 g/dL white cell count 27,000/dL, blood urea 17.1 mmol/L, sodium 133 mmol/L, potassium 7.2 mmol/L, creatinine 93 μmol/L, bicarbonate < 5.0 mmol/L, glucose 56.7 mmol/L and creatinine kinase 70 μ/L (Table I). No haemolysed cells were reported in the blood specimen. A repeat arterial blood gas done 70 minutes after the first one is as shown in Table I. Her blood potassium repeated at the second instance was 4.8 mmol/L. A repeat electrocardiogram three hours after admission revealed sinus tachycardia with resolution of the ST-T wave changes back to normal (Fig 2). Apart from the echocardiogram revealing a normal sized chamber, no segmental wall-motion abnormalities, a left ventricular ejection fraction (calculated) of 65%, no intracardiac clots and normal antegrade flow were noted. Her serial creatinine kinase levels were as shown in Table II.

The patient stayed for three days in the Intensive Care Unit and was then transferred to the general ward and was discharged subsequently on the tenth hospitalisation day after an uneventful recovery. The precipitating event for the diabetic ketoacidosis was found to be sepsis from urinary tract infection rather than an acute myocardial infarction.

DISCUSSION

Acute myocardial infarction (AMI) is a known precipitating factor for diabetic ketoacidosis (DKA) in diabetics[1,2]. This is presumed to be owing to the relative insulin deficiency, especially in patients similar to this case. The acute stress of an acute myocardial infarction or infection increases insulin requirements in such patients. It is therefore a standard practice for an ECG to be done for a patient presenting with DKA. DKA has been known as an important contributing cause of death among diabetics who develop AMI[2,3]. The infarction may, however, be relatively silent and can be overshadowed by ketoacidosis.

Hyperkalemia occurs frequently with DKA. However, severe hyperkalemia is uncommon and is likely to be a consequence of severe dehydration, pre-renal azotemia and renal potassium retention.

In a patient presenting with DKA and with an electrocardiograph (ECG) resembling acute myocardial infarction, it is appropriate to make an initial diagnosis of AMI.

The classical ECG changes of hyperkalemia may be graded as follows[4]:

Serum K^+ Level (on U/Litre)	ECG Changes
5.5 – 6.5	Peaking of T waves
> 6.5	QRS widens
> 7	P wave amplitude diminished and P-R interval increases
> 8	P wave disappears
12 – 14	VF or Asystole

Hyperacute ECG changes in a patient with acute myocardial infarction are usually described as follows[5]:

- At first (first few hours) the ST segment is elevated. This segment becomes convex upwards.
- Giant pointed T wave, positive sign of subendocardial ischaemia.
- Q wave of necrosis which may be small at the onset.

Suppression of P waves has not been noted previously as part of the ECG complex of early acute myocardial infarction.

The patient we described had "P" waves present (though of low amplitude in most leads), but markedly elevated ST segments with T waves pointing upwards without a convex curving. Previous reports of ECG changes resembling acute myocardial infarction in hyperkalemic patients[4-9] have all showed similar changes. It is this combination of changes that are slightly different from the early hyperacute changes of AMI that should prompt suspicion of hyperkalemia.

We postulate that the ECG changes seen in the hyperkalemia of DKA are a result of a combination of the following:

i) Coronary insufficiency owing to severe dehydration.
ii) Effect of severe acidosis on intraventricular conduction.
iii) Effect of severe hyperglycaemia on myocardial tissues.
iv) Effect of severe hyperkalemia per se.

CONCLUSION

It is not known if metabolic acidosis per se or very high levels of blood sugar alone are associated with changes in the electrocardiogram. What is likely at least from this patient described is that such ECG changes appear principally as an effect of high potassium level.

All these have a bearing on priorities in the management of very sick diabetic patients presenting to the Emergency Department; viz any sick diabetic presenting either with difficulties in breathing, nausea, vomiting or other severe manifestation of illness requiring at least a 12-lead ECG, an immediate blood sugar test, a rapid electrolyte assay and an immediate arterial blood gas analysis.

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