Electrocardiogram as a Guide to Potassium Replacement in Diabetic Ketoacidosis

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SUMMARY

Twenty-three patients in severe diabetic ketoacidosis were followed by continuous electrocardiographic monitoring using Lead II of the electrocardiogram throughout their course of treatment. Frequent serum potassium determinations were carried out and correlated with the ECG changes. On admission to hospital the electrocardiogram of ketoacidotic patients showed varied T wave patterns often with little correlation to the serum potassium, but the two patients who were initially hypokalemic were identified from the ECG appearances. Following the infusion of fluids alone the serum potassium fell with accompanying T wave and ST segment changes while the administration of potassium reversed these abnormalities. Although the electrocardiogram is not a substitute for serum potassium determinations, it is a useful guide to potassium replacement in diabetic ketoacidosis. In the successful management of this diabetic emergency the achievement and maintenance of a normal ECG, by means of early and continuous administration of potassium in the infused fluids, should be an important objective of treatment. DIABETES *23*:610-15, July, 1974.

The successful management of diabetic ketoacidosis calls for constant medical supervision and for immediately available biochemical facilities to monitor changes in fluid and electrolyte balance. Once treatment starts, potassium balance alters rapidly,[1] and it is often difficult to follow the rapidly changing serum potassium; the veins are usually collapsed in severely dehydrated patients, making repeated venipunctures difficult; frequent requests for serum potassium determinations are a strain on the routine biochemical service during the day, and results take longer to obtain than is desirable in this diabetic emergency; at night, electrolyte determinations are not always easily accessible. In 4 to 9 per cent of ketoacidotic patients[2,3] hypokalemia is already present on admission to hospital, and a delay in obtaining accurate serum potassium results and instituting appropriate therapy can lead to a fatal outcome. Similarly, treatment with fluids, insulin and alkali can uncover large potassium deficits[4-6] which need immediate recognition and correction.

The present study was undertaken to evaluate continuous electrocardiographic monitoring as a guide to potassium replacement in diabetic ketoacidosis.

METHODS

Twenty-three patients in diabetic ketoacidosis, all of whom survived, were the subjects of this study —one other patient was excluded because of asymptomatic acute inferior infarction. Their ages varied between sixteen and eighty years. In all cases the arterial pH was less than 7.2 and the standard bicarbonate less than 10 mEq./L. On the arrival of a ketoacidotic patient in the Casualty Department, a full ECG was performed and immediate admission to the Diabetic Unit, in which all of them were treated by the same team of physicians, was arranged.

After preliminary work, Lead II of the electrocardiogram was selected as the monitoring lead of choice. The advantages of this lead are that the P wave can easily be seen, the T wave is an upright deflection and there is less base line swing with respiration than with a chest lead, which is an important consideration in patients who are hyperventilating. The disadvantages

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are that the U waves are often better seen in the V leads and a leg lead has to be used.

The patients received treatment with insulin, saline and varying amounts of bicarbonate. It is known that a total body deficit of 300 mEq. of potassium or more exists in ketoacidosis[7] and that during treatment large quantities of potassium are lost in the urine.[8] Potassium administration was determined by the rate of infusion of fluid, the serum potassium and the electrocardiogram. The aim was to keep the serum potassium within normal limits and to achieve, as well as maintain, a normal electrocardiogram. In our recent study[8] potassium requirements averaged between 30 and 40 mEq./L. of fluid infused.

The electrocardiogram of ketoacidotic patients was studied during three phases of hospital attention: (a) On admission to hospital before treatment was started; (b) following the administration of intravenous fluids without potassium; (c) following the administration of intravenous fluids with potassium.

RESULTS

ECG before treatment was commenced. Figure 1 shows the ECG patterns recorded on admission to hospital. At this stage the serum potassium is usually normal or high, but may be low. A large T wave taller than its accompanying QRS complex (a 'giant' T wave) was only encountered in three patients in all of whom the serum potassium was > 6.5 mEq./L. (figure 1a). The other extreme of a flat or inverted T wave with ST segment depression was seen in two patients who both had serum potassium < 3.5 mEq./L. (figure 1f).

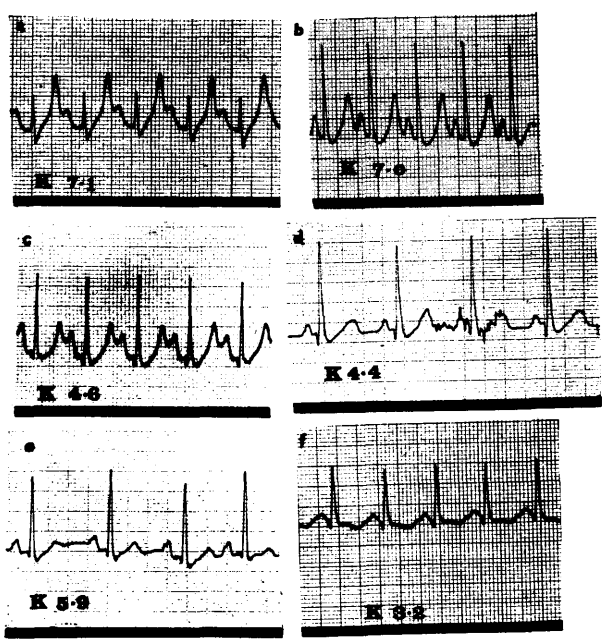

FIG. 1. The different ECG patterns (Lead II) of ketoacidotic patients on admission to hospital in relation to their serum potassium.

T waves of various sizes between these two extremes covered a wide range of serum potassium; representative recordings are shown in figure 1. A tall peaked T wave occurred with a serum potassium of 7 mEq./L. (figure 1b), a smaller T wave with a serum potassium of 4.6 mEq./L. (figure 1c). Less prominent T waves were observed with serum readings of 4.4 mEq./L. and 5.9 mEq./L. respectively (figures 1d and 1e). On admission the QT interval was frequently prolonged, but it was often found difficult to measure due to merging of the T wave with the following P wave.

ECG following the administration of fluids without potassium. In the second phase of study* the infusion of fluids led to a fall in the serum potassium and electrocardiographic changes in the T wave. Figure 2 shows these changes in a patient who had a 'giant' T wave on admission to hospital and a serum potassium of 7.1 mEq./L. Following administration of 1.5 L. of saline, the serum potassium fell to 5.9 mEq./L. and the T wave lost its peaking. After another 500 cc. of 1 per cent sodium bicarbonate the serum potassium fell to 3.8 mEq./L. and the T wave diminished in size with some sagging of the ST segment.

Another patient (figure 3) had a serum potassium of 6.1 mEq. and a prominent T wave on admission. Following the administration of 2 L. of saline, the serum potassium fell to 4.8 mEq./L. and the T wave became flatter. After another 500 cc. of saline the T wave became inverted and the serum potassium reading was 3.7 mEq./L.

ECG following the administration of fluids with potassium. The third phase of study shows the correction of electrocardiographic appearances following the administration of potassium in the infused fluid. Serial ECG recordings of a patient with an initial serum potassium of 3.2 mEq./L. and an electrocardiogram showing inversion of the T waves are presented in figure 4. Following administration of 150 mEq. of potassium chloride the serum potassium rose to 4.6 mEq./L. and the previously inverted T waves became flat. On being given another 30 mEq. of potassium, the serum potassium rose to 4.8 mEq./L. and the electrocardiogram began to show a positive T wave.

*Insulin was withheld during this stage in patients who were fully conscious and cooperative, not unduly distressed and while they remained under constant medical supervision.

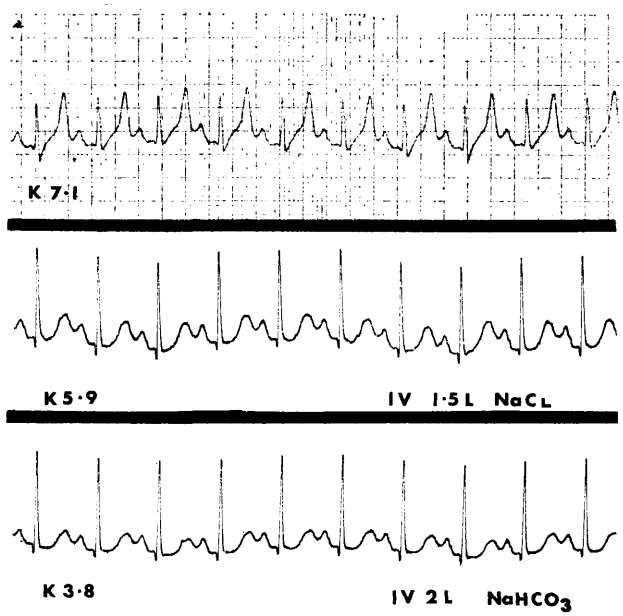

FIG. 2. This patient with a 'giant' T wave and an initial serum potassium of 7.1 mEq./L dropped his serum potassium, lost his peaked T waves and developed ST segment sagging following the infusion of saline and bicarbonate.

This positive deflection increased in amplitude as more potassium was given and after a total of 250 mEq. the T waves were normal in appearance. At this stage, the serum potassium was 4.6 mEq./L.

Despite the administration of 230 mEq. of potassium to another patient (figure 5) the ECG still showed ST segment sagging and small T waves and the serum potassium was 3.3 mEq./L. A normal ECG appearance was only restored when a total of 310 mEq. of potassium had been infused.

In this study the electrocardiogram was also found a useful guide to the rate of potassium replacement in the treatment of diabetic ketoacidosis, as illustrated in figure 6. In this patient the serum potassium was 4.5 mEq./L. and the T waves were relatively normal. During potassium infusion the T waves suddenly became peaked and the amplitude of the QRS diminished, indicating that the potassium was being infused too quickly.[9] Within a couple of minutes of slowing down the infusion of potassium, the ECG returned to normal with a serum potassium of 4.6 mEq./L. Similar changes were observed in other patients when potassium was being infused too quickly.

DISCUSSION

The electrocardiographic abnormalities observed in this study during the treatment of diabetic ketoacidosis were noted by earlier workers. In 1937, Bellet and Dyer[10] reviewed the literature and described the ECG changes in seventeen of their own patients, namely depression of the ST segment, lengthening of the QT interval and alterations in the amplitude and direction of T waves. A strong correlation between small T waves and a low serum potassium was noted by Martin and Wertman[11] and when Holler[12] originally described the severe clinical effects of hypokalemia in diabetic ketoacidosis, the electrocardiogram in his hypokalemic patient showed ST segment sagging and flat T waves, the appearances returning to normal following potassium replacement. Further work[13,14] indicated that although the ECG did not always reflect quantitatively the serum potassium, sequential ECG recordings were a valuable means of following the changes in the serum potassium during the treatment of diabetic ketoacidosis as confirmed in the present study.

The importance of potassium in the maintenance of normal cardiac function was first shown by Ringer.[15] However, the basic cause of the electrocardiographic signs of hypokalemia has remained a matter for debate. Surawicz[16] initially favored the potassium gra-

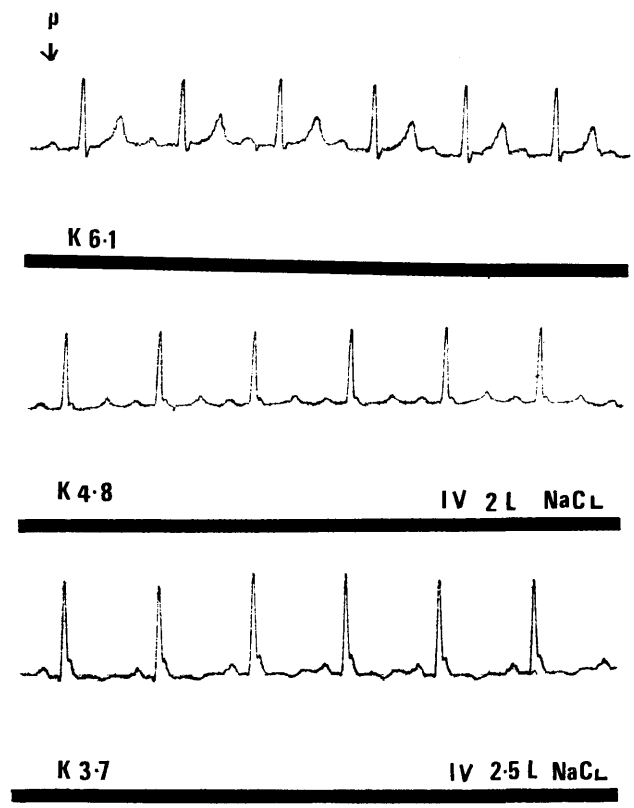

FIG. 3. The infusion of saline alone led to a fall in the serum potassium from an initial level of 6.1 mEq./L and changed the T wave from a peaked to an inverted appearance.

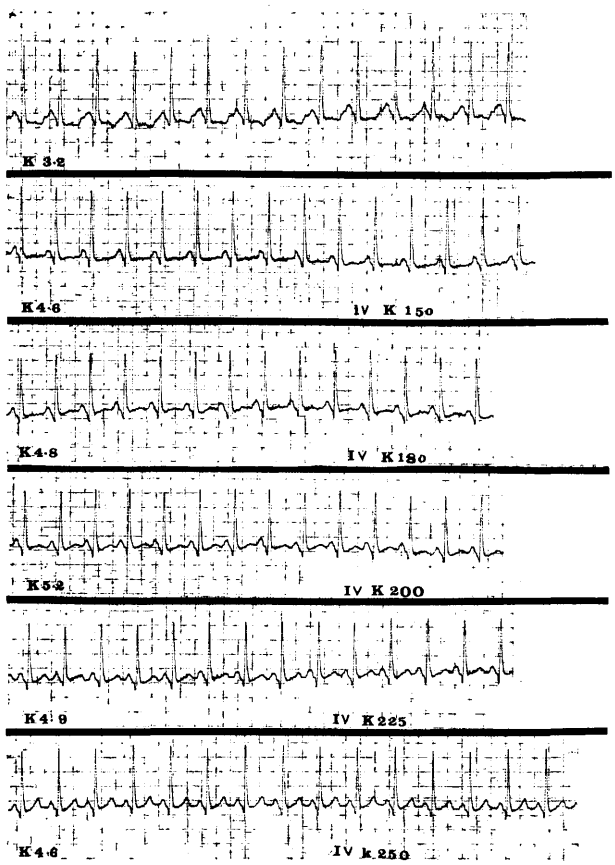

FIG. 4. T wave inversion and ST segment sagging in a patient with hypokalemia on admission to hospital gradually reversed following potassium replacement and a normal ECG appearance finally restored following the infusion of 250 mEq. of potassium.

dient across the cell membrane, but later[17] considered the serum potassium level to be the important factor, either directly or by its large effect on the ratio of extracellular to intracellular potassium concentration. On the other hand, during experiments in dogs who were acutely depleted of potassium by hemodialysis, sequential ECG changes continued at a time when the serum potassium concentration remained unaltered, suggesting that the ECG changes were due to losses of intracellular potassium.[18] From a detailed study of the serum potassium and ECG changes in diabetic ketoacidosis[19] it was concluded that the electrocardiogram is more likely to reflect intracellular potassium changes and the findings in the present study —that correction of the ECG abnormalities following potassium infusion can occur despite a fall in the serum potassium—would support this view (figure 4). Moreover, in fully recovered ketoacidotic patients with a normal pH, we have observed delays in a return to normal ECG appearance despite a serum potassium in the normal range, when insufficient potassium has been administered to correct existing deficits and presumably intracellular potassium. The use of sodium bicarbonate in diabetic ketoacidosis increases potassium requirements,[8] leads to a restoration of intracellular potassium,[20] and it is our impression that, if adequate potassium replacements are used, a normal ECG follows sooner compared to cases treated without alkali.

The acidosis associated with uncontrolled diabetes may interfere with the interpretation of the ECG changes due to potassium depletion.[21] As early as 1939, experiments on human volunteers showed that acidosis is accompanied by an increase in the size of T waves.[22] Acid-base disturbances produced in dogs[23,24] also led to ECG changes; if a metabolic acidosis was induced the ECG appearances seen in hyperkalemia followed with or without elevation of the serum potassium. Isolated ECG recordings in acidotic patients may, therefore, lead to imprecise information about the state of the serum potassium. However, in the work of Surawicz et al.[17] alkalosis and acidosis did not prevent the appearance of the typical ECG changes of hypokalemia, which were also seen in the two ketoacidotic patients with initial hypokalemia included in our study. In our investigation the emphasis was on sequential ECG changes and the accompanying changes in the serum potassium, which were shown both in patients treated with bicar-

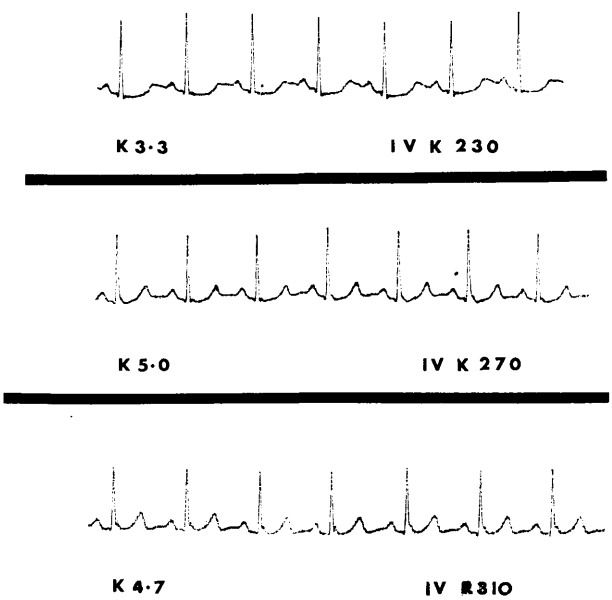

FIG. 5. Hypokalemia with accompanying ECG changes (ST segment sagging and small T waves) occurring during the treatment of diabetic ketoacidosis and corrected following further administration of potassium.

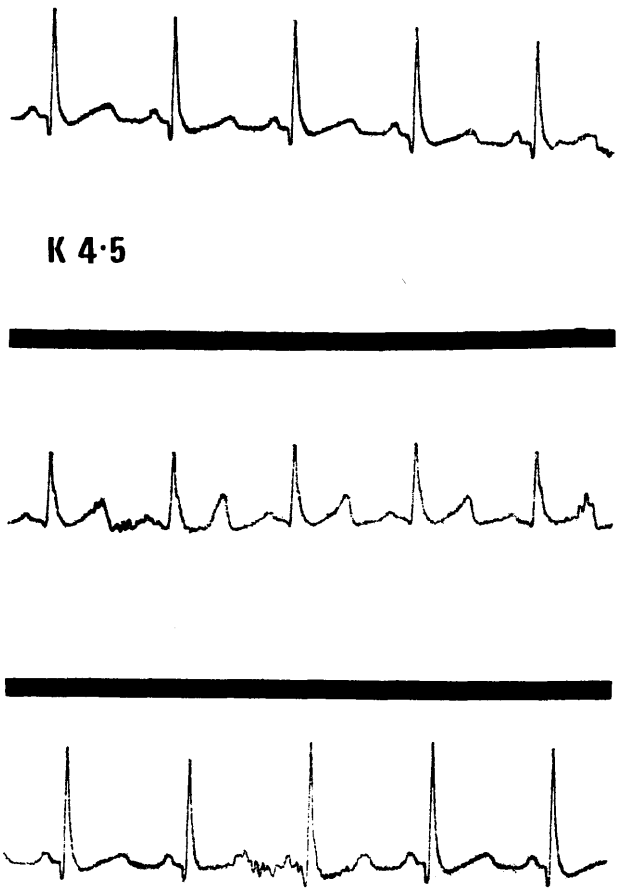

FIG. 6. During the administration of potassium, peaking of the T wave and loss of amplitude of the R wave indicated too rapid infusion of potassium. The appearances were reversed by slowing down the infusion.

bonate and in patients given an infusion of saline. Clearly the acid-base change resulting from infusion of alkali may lead to ECG changes, but we were unable to separate them from the changes due to the falling serum potassium which invariably accompanied the use of bicarbonate. On the other hand, the administration of intravenous saline to ketoacidotic patients without alteration of the pH (figure 3) also produced a decrease in the serum potassium and ECG changes. The final restoration of a normal ECG appearance following the administration of potassium may have been influenced, to a certain extent, by the concomitant use of sodium bicarbonate which eliminated the effect of acidosis and encouraged intracellular potassium repletion. Other patients who only received saline infusions retained less potassium[8] and a normal ECG was attained after a longer period of treatment.

It has been argued that as the electrocardiogram does not always reflect accurately the serum potassium, its use is of limited value during the treatment of diabetic ketoacidosis.[25,26] However, in the individual patient, serial ECG records when compared with tracings at known levels of serum potassium give a useful working guide to the rapidly changing serum potassium. In addition, since the ECG is a better indicator of the seriousness of potassium depletion than the determination of serum potassium,[16] the achievement and maintenance of a normal ECG must be an important goal during replacement of the potassium deficits of diabetic ketoacidosis. Cardiac arrhythmias may also complicate diabetic coma.[10,19] While acidosis lowers the cardiac threshold to arrhythmias[27] hypokalemia is a recognized cause of a wide spectrum of atrial, junctional and ventricular arrhythmias.[28] Recent reports confirm that hypokalemia can lead to fatal arrhythmias in diabetic ketoacidosis.[3,29] Continuous ECG monitoring not only leads to early detection of arrhythmias before disaster ensues, but also gives an immediate record of their response to treatment.

In our work on diabetic coma, continuous ECG monitoring using Lead II of the electrocardiogram has been employed in over a hundred cases.[3] The method is a simple and readily available bedside monitor, but has certain limitations. The ST and T-wave changes in patients with ischemic heart disease or on treatment with digoxin invalidate the ECG for monitoring serum potassium changes. Sinus tachycardia, which is frequently present in untreated ketoacidotic patients, may be associated with ST segment depression, but our studies indicate that sequential changes in the T wave can still be followed and the heart rate usually slows down as dehydration is corrected. Although the ECG of acidotic patients on admission to hospital does not always reflect accurately the serum potassium, in our experience patients who are already hypokalemic can be identified from the ECG before the serum potassium is available. It is now our practice to commence potassium administration immediately; a fluid infusion is set up in all ketoacidotic patients except those with an initial ECG showing T waves taller than their accompanying QRS complexes, indicating a very high serum potassium (> 6.5 mEq./L.). A poor urine output is not a contraindication to potassium administration, but caution is necessary when oliguria persists. On the other hand, some authorities,[30] also advocates of early aggressive potassium replacement, prefer to delay commencement of intravenous potassium therapy until the first or second liter of fluid is infused. In diabetic ketoacidosis potassium requirements are usually between 30 and 40 mEq./L. of fluid

infused[8]—average 8 L. in twenty-four hours—but for patients with initial hypokalemia 60 to 80 mEq./L. may be necessary.[30] The ECG is the only reliable monitor of the rate of potassium administration, peaking of the T waves and loss of amplitude of the R waves indicating that potassium is being infused too quickly.[9] If potassium replacement is commenced early and continued throughout the course of treatment the use of large amounts of parenteral potassium, in short bursts, with its attendant dangers, should only rarely be necessary to correct unexpected deficits; in these cases ECG monitoring is mandatory. While continuous ECG monitoring is a useful addition in the intensive treatment of diabetic ketoacidosis[3] and is of inestimable value in critical situations, such as hypokalemia, impaired renal function and cardiac arrhythmias, it is not a substitute for frequent serum potassium determinations which remain essential for the successful management of this diabetic emergency.

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