Case Report Section

Cardiac Dysfunction in Severe Hyperkalemia*

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The clinical manifestations of hyperpotassemia are well described in the current literature, and the electrocardiogram is one of the most important diagnostic aids in establishing this syndrome. This paper is primarily concerned with the management of the often dramatic electrocardiographic abnormalities seen in patients with hyperkalemia.

The electrocardiograph was first used to demonstrate the effects of abnormal serum levels of potassium in the animal laboratory in 1938 by Winkler 1 and again by Chamberlain 2 in 1939. Since that time many investigators 3-7 have described similar alterations in the electrocardiograms of human beings with hyperpotassemia.

The progressive electrocardiographic changes seen with rising serum potassium have been summarized by Burch and Windsor 8 as follows:

1. Increased magnitude of T waves.
2. Depression of the ST segment.
4. Increased duration of the QRS complex.
5. Increased PR interval.
6. Distortion of P wave with a decrease in magnitude.
7. Prolongation of QRS to produce a pattern of bundle branch block.
8. Auricular standstill.

The most characteristic change seen in peaking of T waves and increase in the duration of the QRS complex.

Attempts at correlation between serum levels of potassium and electrocardiographic alteration are difficult, since it is the intracellular potassium which is important in determining the cardiac muscle response. Tarail 9 pointed out that with serum levels of 6.8-7.6 mEq/liter, the electrocardiogram showed inconstant changes, but when the serum level was over 7.8 mEq/liter, the changes were consistently present.

The following case of acute glomerulonephritis demonstrates many of the typical abnormal electrocardiographic features of hyperkalemia.

The patient was an 18 year old airman who had streptococcal pharyngitis in October, 1955 which was treated with penicillin. Early in November, 1955, he had recurrent sore throat but did not seek medical attention. In December he was hospitalized with complaints of pain and swelling of both knees and wrists and a rash on the lower legs. Past history was negative.

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Physical examination revealed an acutely ill, well developed, well nourished man. Blood pressure was 115/70, temperature 99.8° F., pulse 92, respirations 20. The examination of the head, including eyes, ears, nose, and throat, was negative except for slight infection of the pharynx. His neck was supple and his chest and lungs normal. The heart rhythm was regular and a grade I systolic murmur was heard at the apex. There was no palpable thrill or friction rub heard. The pulmonic second sound was slightly accentuated. The abdomen, back and genitalia were normal. The joints and extremities exhibited slight swelling of ankles but no erythema or increase in temperature was noted. Extensive purpuric lesions were present over both lower extremities.

On admission the white blood cell count was 15,000 with 82 polymorphonuclear leukocytes, hematocrit 45 per cent, sedimentation rate 36 mm. per hour. The urine was negative for sugar, albumin, and microscopic findings. A chest x-ray film and electrocardiogram were normal. A throat culture was negative for Beta hemolytic streptococcus. Platelet count, bleeding and clotting time, clot retraction, prothrombin time, and capillary fragility tests were all normal.

In the first month, the illness was that of the rheumatic state. The laboratory and electrocardiographic studies gave confirmation of active myocarditis. During this

FIGURE 1: Base line electrocardiogram. Low voltage is present in Lead I. The auricular and ventricular conduction times are normal. The voltage and form of the T waves were considered to be within normal limits.
time abdominal pain was a frequent complaint. Radiographic studies of the gastrointestinal tract were not helpful. The arthritis and purpura gradually cleared but fever persisted.

In the fifth week of hospitalization the urine exhibited microscopic red blood cells, albumin and casts. During the course of the next week, he developed pleural effusion, weight gain of 20 pounds, peripheral edema, and elevation of blood pressure to 180/100. The laboratory findings demonstrated early uremia and mild acidosis. A urine culture showed the presence of Escherichia coli, and he was treated effectively for this with chloromycetin.

The disease progressed, and with the urine output gradually dropping, he became more uremic and acidotic. The urea nitrogen was 60 mg. per cent, hematocrit 30 per cent, CO₂ combining power 41 vol. per cent. A supportive regimen including restricted salt intake, digitalization, and small blood transfusions was carried out, without improvement.

FIGURE 2: Serum potassium recorded 8.6 mEq./liter at this time. Electrocardiogram exhibiting peaked T waves, diminution of the R wave. A-V dissociation with widening of the QRS, depression of ST segment and merging of the QRS and T waves into a sine wave.
At the end of the eighth week of hospitalization, the serum potassium rose to 7.0 mEq./liter. The use of retention enemas with potassium-removing resins effected temporary reduction to 6.4 mg. On February 13 and again on February 22, he was dialyzed by Dr. Arthur MacNeill and staff of the University of Buffalo, using the MacNeill Mark XI-b dialyzer. The second dialysis produced a good chemical response with the blood urea nitrogen dropping from 170 mg. per cent to 90 mg. per cent. Subjectively he was improved, though renal function remained severely impaired.

On March 3, he developed acute hyperkalemia with serum potassium of 8.6 mEq./liter. Clinically this was manifested by Cheyne-Stokes respirations, marked lethargy and dramatic alteration of the electrocardiogram. Figure 1 shows the electrocardiogram taken on December 29, 1955. At this time regular sinus rhythm was present with normal auricular and ventricular conduction times. The T waves are not considered grossly abnormal and electrolyte imbalance is as yet not evident from our laboratory procedures. Figure 2 shows the electrocardiogram at the time of the increase in serum

FIGURE 3: The progressive response to intravenous glucose and insulin at the time. The fourth and fifth strips show A-V dissociation with more normal ventricular complexes. Sinus mechanism was established after three and one-half hours of glucose and insulin administration.
potassium. Peaked T waves are noted in V3 and V4; the diminution of R wave, absence of P wave, widening of the QRS complex, and merging of the QRS and T into a sine wave, are all demonstrated at this time. Figure 3 shows short sections of Lead II before 1,000 cc. of 10 percent glucose and 40 units of regular insulin were administered, and then at 10 minutes, 20 minutes, three and one-half hours, and six and one-half hours after the start of glucose administration. The reversion to normal rhythm is demonstrated in the electrocardiogram taken the following day (Figure 4), but voltage is considerably reduced. On March 6, 1956, the electrocardiogram was very near in appearance to the one prior to the abrupt rise in serum potassium. Because of the persistence of uremia and anuria, dialysis was again performed on March 6, 1956. The procedure was tolerated well, even though severe acidosis was present.

Four hours after termination of the dialysis the patient convulsed, and in the next 10 hours he had 20 more convulsions. The blood pressure rose to 210/100. Magnesium sulfate, heavy intravenous sedation, and hypertonic fluids were used without response, and he expired on March 7, 1956. Post mortem examination demonstrated the renal findings seen with acute glomerulonephritis.

FIGURE 4: Tracing taken on succeeding day. Persistence of sinus mechanism with delayed A-V conduction and low voltage.
Treatment Differential

The management of this case presented several problems, of which the control of hyperkalemia is frequently the most difficult. Unfortunately, there is no specific treatment for the primary pathology in acute glomerulonephritis; but the complications of uremia, acidosis, and hyperkalemia which produce the fatality can frequently be controlled.

The aim of current therapy is to forestall these complications while waiting for the kidney to resume its normal functions. Finch, Meroney, and their associates demonstrated that temporary lowering in serum potassium can be accomplished by the use of simple physiological saline solution. They point out that the addition of calcium, particularly when serum levels of this ion are concomitantly depressed, further aids by directly antagonizing the effects of potassium on the heart muscle. Danowski and Elkinton report the successful reduction of serum potassium through the use of cation exchange resins. Employment of various dialysis techniques has been demonstrated by Kolff. In our case the MacNeill blood dialyzer, which is of unique design and highly effective function, was used in the later stages of the illness.

Recently the use of carbonic anhydrase has been described by Mosely. The resultant decrease of available hydrogen ion without blockage of potassium ion transfer from intracellular position in the renal tubules to the tubule lumen, increases potassium excretion in the urine. The favorable response of patients with hyperpotassemia to exchange transfusion technique has been reported by Goldbloom.

Bellet and associates have shown the effectiveness of molar sodium lactate in the control of heart block secondary to hyperkalemia as well as to other etiologies. The mechanism of action is still under investigation. The current concept is that return of abnormal electrolyte patterns to a more physiological state results in an increase in cardiac rhythmicity.

The use of hypertonic glucose and insulin was initially demonstrated by Darrow and again more recently by Goldbloom. The net effect is the deposition of glycogen similar to that occurring in the treatment of diabetic acidosis. Concurrently potassium is transferred into the cell at a rate that Darrow estimated at 0.36 millimoles of potassium per gram of glycogen. The insulin acts to stimulate the deposition of glycogen, though it is not considered to have a specific potassium reducing effect in itself. The latter form of therapy was used to correct the cardiac arrhythmias in this case.

REFERENCES

Vol. XXXIV CARDIAC DYSFUNCTION IN HYPERKALEMIA 221


