Hyperkalemia-Induced Conversion of Chronic Atrial Fibrillation to Normal Sinus Rhythm; A Case Report

P. K. Mathew, M.D.
ROCHESTER, NEW YORK

Abstract

The electrocardiographic manifestations of hyperkalemia have been well described. We report a patient with chronic congestive heart failure and atrial fibrillation for 6 years, who converted to sinus rhythm during an episode of hyperkalemia. To our knowledge, this is the first report of hyperkalemia-induced conversion of chronic atrial fibrillation to sinus mechanism.

Case Report

An 80-year-old woman with a history of coronary artery disease and previous myocardial infarctions was admitted to the hospital on 7/23/76 with severe congestive heart failure and cellulitis of the right leg. The patient had been hospitalized recurrently for congestive heart failure, and at the time of admission she was on digoxin, 0.25 mg, furosemide, 200 mg, and spironolactone.

Her electrocardiogram showed atrial fibrillation with a ventricular rate of 65/minute, multiple VPCs, and complete left bundle branch block (LBBB) (Figure 1). The LBBB and the atrial fibrillation had been apparent in her ECGs since 1970. Her BUN was 47 mg%, serum sodium was 139 mEq, and potassium was 4.8 mEq. Study of the arterial blood gases revealed a pH of 7.38, a PCO_2 of 31.5 mm Hg, a PO_2 of 61 mm Hg, and 20mEq of bicarbonate.

In the next 1 week her cardiac status improved slightly, but her ECG continued to show atrial fibrillation and an LBBB pattern. The serum K on 7/31/76 was 5.2 mEq. On 8/1/76 the ECG monitor showed conversion of atrial fibrillation to regular sinus rhythm and this was confirmed by 12-lead ECG (Figure 2A). The P waves were clearly visible with a PR interval of 0.36 second. The LBBB pattern persisted, but the QRS complex was more widened to 0.28 second. The T wave morphology was not markedly different from the previous ECG. The serum potassium during this time was 7.6 mEq. Her BUN was 67 mg%, sodium 138 mEq, and the arterial blood gases showed a pH of 7.34, a PCO_2 of 32 mm Hg, a Po_2 of 65 mm Hg, and 18 mEq or bicarbonate. The serum digoxin level was 1.3 ng/L.

The patient was treated with Kayexalate enemas, and the serum potassium returned to 5 mEq in the next few hours. She continued to show an LBBB and regular sinus rhythm, but the PR interval shortened to 0.28 second and QRS returned to the original duration (Figure 2B). Although the hyperkalemia was controlled, the patient's cardiac status and renal functions deteriorated, and she died.

Discussion

Elevation of K+ concentration reduces the resting potential and, the rate of increase and amplitude of the action potential, and accelerates the rate of repolarization. Gradually increasing plasma K+ induces a biphasic, more or less reproducible electrical sequence of initial increase, and then pro-

From the Department of Medicine, University of Rochester School of Medicine and Dentistry, and Saint Mary's Hospital, Rochester, New York.

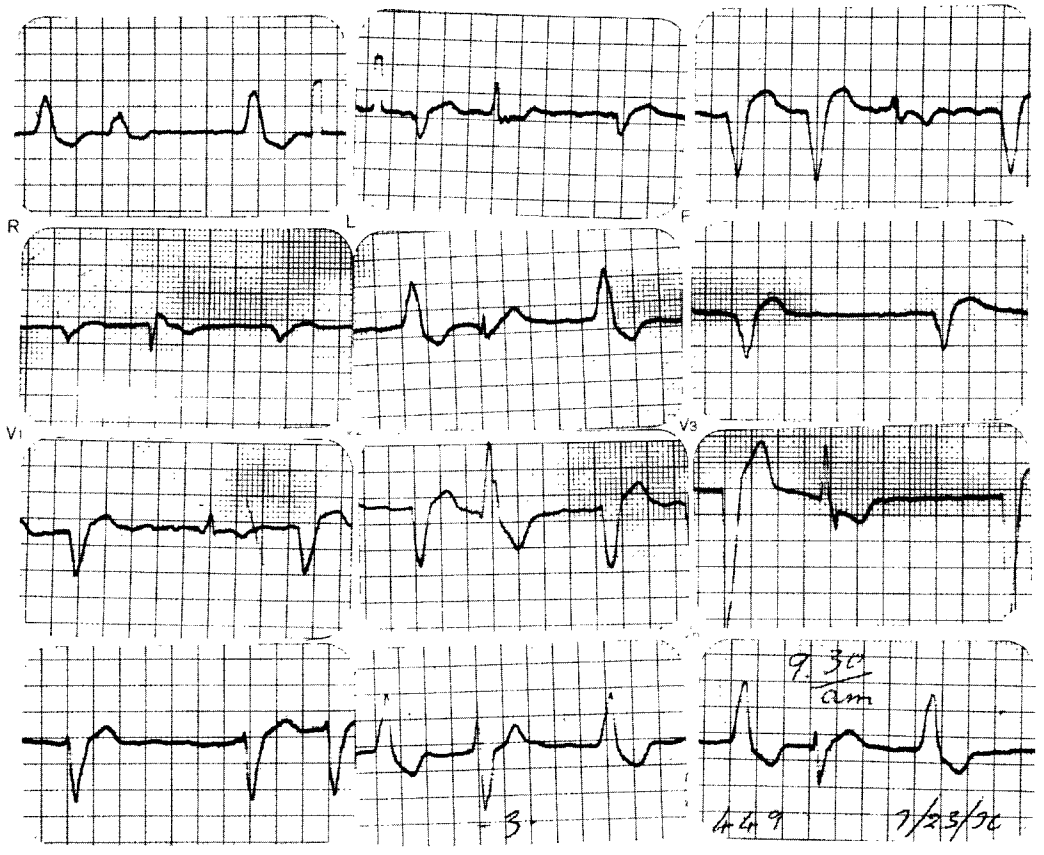

FIG. 1. The electrocardiogram made on admission shows atrial fibrillation, a ventricular response of 65 per minute, multiple VPCs, and left bundle branch block.

found depression in excitability, conduction, and automaticity.[1] The heart is composed of fibers of different functional properties which react quantitively differently to changes in potassium levels, although the qualitative effects on different fibers are similar. The work by De Mello and Hoffman[2] established that atrial muscle frequently appears to be more sensitive than ventricular muscle to elevated potassium, whereas specialized fibers such as SA node, internodal tracts, and His bundle are practically resistant.

The electrophysiologic mechanism by which hyperkalemia terminated atrial fibrillation in our patient is unknown. As suggested by Ettinger et al.,[3] it is logical to assume that a transient acceleration of conduction at a modest increase in K+, or that a decrease in conduction at higher K+, may abolish a re-entrant circuit in the atria. Alternatively, higher K+ levels depress excitability and tend to suppress an ectopic irritable focus in the atrium. Since the SA node is relatively resistant to hyperkalemia, the high K+ level tends to provide an optimal condition for re-establishment of the sinus mechanism, either by abolishing a re-entrant circuit or by suppressing an ectopic focus in the atrium. The relative infrequency of this finding in clinical practice probably indicates that a potassium level in the range of significant hyperkalemia is a prerequisite of this effect.

A review of the literature shows that hyperkalemia-induced conversion of chronic atrial fibrillation to regular sinus rhythm has not been previously reported. Bettinger et al.[4] reported the case of a 51-year-old man with arteriosclerotic heart disease and bronchopneumonia who presented with atrial fibrillation, which reverted to regular sinus rhythm after infusion of potassium chloride. However, it is not known whether the patient

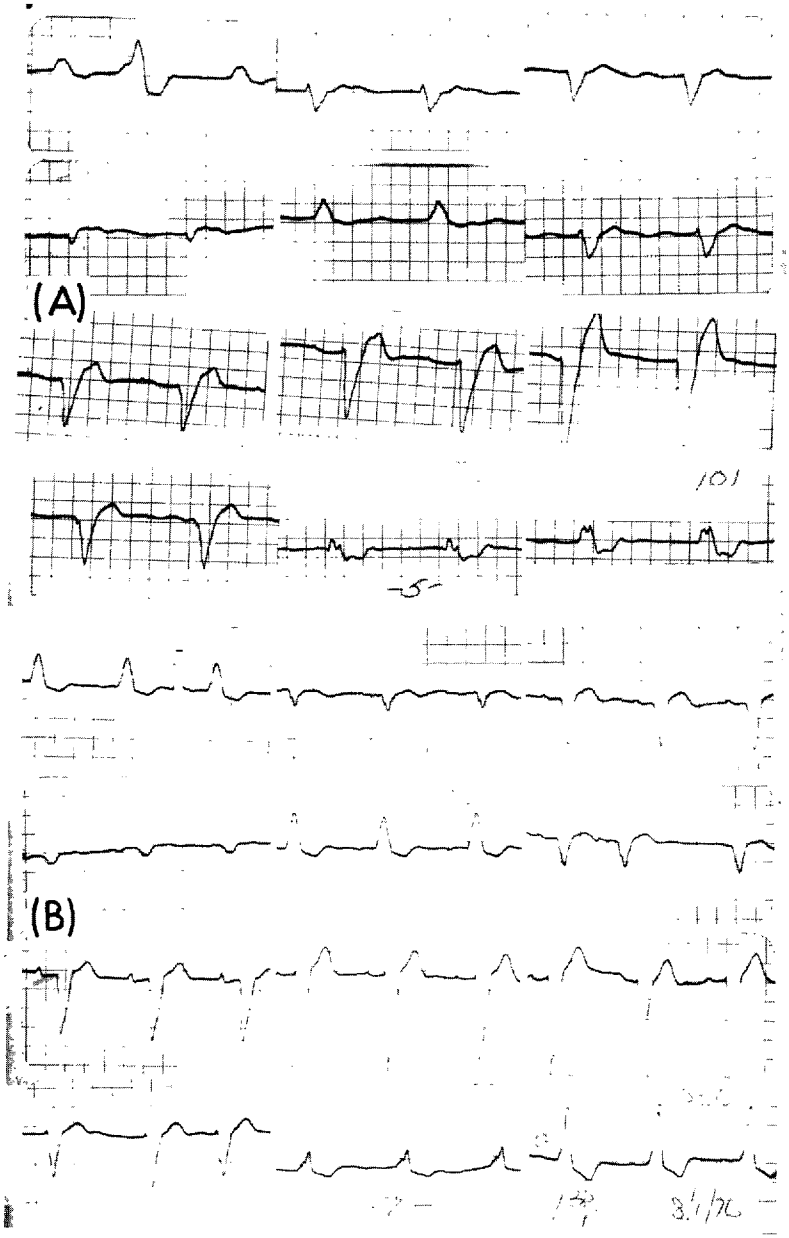

FIG. 2. (A) The electrocardiogram recorded when the serum K+ was 7.6 mEq. The P wave morphology is identifiable and the PR interval is 0.36 second. The LBBB pattern persists, but the QRS complexes are more widened. (B) The electrocardiogram done a few hours after the first tracing, when the serum K+ returned to normal. The patient remains in sinus rhythm, the PR interval is 0.28 second, and the QRS complex returns to its original duration.

had recent or chronic atrial fibrillation, and the patient's serum potassium was not in the hyperkalemic range. Two other cases are reported[5,6] in which atrial fibrillation converted to sinus rhythm after K+ infusion, but again the duration of atrial fibrillation is unknown and the serum K+ levels were not certainly in the hyperkalemic range.

Even though one can postulate that the change in acid base, electrolyte, and blood

gas status might have contributed to the abolition of atrial fibrillation in this patient, this is probably unlikely, because except for hyperkalemia, there were no significant measurable new changes in any of these parameters at the time of rhythm change.

This case report emphasizes the fact that, in addition to the well-described electrocardiographic features of hyperkalemia, conversion of atrial fibrillation to regular sinus rhythm may occur as a manifestation of hyperkalemia.

P. K. Mathew, M.D.
St. Mary's Hospital
89 Genessee Street
Rochester, New York 14611

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