Paroxysmal Ventricular Fibrillation in Two Patients with Hypomagnesemia

Treatment by Transvenous Pacing

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SUMMARY

Paroxysmal ventricular fibrillation unassociated with heart block occurred in two patients with hypomagnesemia. In neither patient were other causes of the arrhythmia apparent. Temporary transvenous pacing successfully suppressed the episodes after drug therapy failed. Prolongation of the Q-T interval was a prominent electrocardiographic feature in both patients and is postulated to have resulted from a loss of intracellular potassium secondary to hypomagnesemia.

Additional Indexing Words:
Adams-Stokes attacks Electrocardiographic features Potassium deficiency

Paroxysmal ventricular fibrillation is a common cause of Adams-Stokes attacks in patients with complete heart block and has been observed occasionally in patients without heart block. Two such cases are reported herein. Each patient presented with recurrent seizures due to ventricular fibrillation refractory to usual modes of therapy, but controlled by catheter pacing. Hypomagnesemia was documented in both patients and suggests a mechanism for the arrhythmias.

Report of Cases

Case 1

B. W., a 29-year-old Negro female, was admitted to the hospital because of convulsions beginning 1 day prior to admission following several weeks of excessive intake of alcohol and malnutrition. The patient had had “asthma” since childhood and had been addicted to alcohol for several years. She had no past history of cardiac or neurological disease.

On physical examination, the patient was poorly nourished, but alert, cooperative, and in no apparent distress. Blood pressure was 110/60 mm Hg; pulse was 90 beats/min and regular; temperature was normal. Examination of the heart revealed findings consistent with minimal aortic regurgitation. The remainder of the physical examination was unremarkable except for evidence of pelvic inflammatory disease. Lumbar puncture yielded normal cerebrospinal fluid. On admission the hemoglobin value was 8.7 g per 100 ml. Levels of blood glucose, urea nitrogen, albumin, globulin, protein-bound iodine, and amylase were normal. The concentration of serum potassium was 3.5 mEq/L; sodium 126 mEq/L; chloride 89 mEq/L; and calcium 9.6 mg/100 ml. The serum magnesium concentrations were 0.5 mEq/L on admission, 0.6 mEq/L the following day, and 0.7 mEq/L 6 days later. Additional studies including blood cultures, Kahn test, L.E. preparation, ASO titers, and antibody titers to selected viral antigens were negative.

Hospital Course

Serial electrocardiograms recorded over the next several hours (fig. 1) revealed a pattern...
PAROXYSMAL VENTRICULAR FIBRILLATION

B.W. am. 5/27/65

LEAD II

Figure 1

Case 1. Lead II of the electrocardiogram recorded from 12:04 to 1:22 a.m. (a-d) Sinus rhythm with progressive increase in the Q-T interval is seen. There is no evidence of heart block. In (e) bigeminal rhythm appears and reappears (in f) following a brief episode of ventricular fibrillation. (g and h) A longer episode of ventricular fibrillation was associated with a grand mal seizure. Following the seizure sinus rhythm ensues (j) and the Q-T interval again increases (k-r) until bigeminal rhythm reappears (s-u).

categorized by sinus rhythm, followed by bigeminal rhythm, multifocal ventricular complexes, and ventricular fibrillation terminating spontaneously in sinus rhythm. It was noted that the Q-T interval lengthened progressively prior to the appearance of bigeminy but returned to a near normal duration immediately following ventricular fibrillation. Although several brief periods of ventricular fibrillation were unaccompanied by clinical sequelae, longer episodes were observed during
which the patient lost consciousness and developed tonic convulsions. Therapy including procainamide, diphenylhydantoin, potassium, calcium chloride, and 50% solution of glucose failed to prevent the arrhythmia. Although each episode appeared to terminate spontaneously, the possibility of a single fatal episode prompted institution of artificial cardiac pacing as a means of controlling the rhythm. A bipolar pacing catheter was inserted without difficulty and pacing from the right ventricle appeared to be effective in preventing further episodes of ventricular fibrillation. Yet, several hours after pacing had been started, the patient again lost consciousness and convulsed. An electrocardiogram revealed absence of artificial pacing which was found to be due to battery failure. Upon battery replacement, pacing was reinstated and continued for 24 hours during which no further arrhythmia or seizures occurred. Pacing was then discontinued, and sinus rhythm without ventricular ectopic beats ensued. Recovery was uneventful.

Prior to discharge, myocardial biopsy was performed at open operation which revealed only minimal edema of the myocardium on light microscopy. When last seen, the patient felt well, cardiac findings were unchanged, and the electrocardiogram showed a normal Q-T interval. The serum magnesium concentration was 1.0 mEq/L.

Case 2

F. R., a 24-year-old Negro female, was admitted because of loss of consciousness associated with convulsive seizures. Prior to admission the patient had been taking digoxin and hydrochlorothiazide for what was considered to be mild rheumatic heart disease. On the day of admission she had felt well except for slight fatigue after returning from a shopping trip. On awakening from a brief nap, the first of several seizures occurred. Ingestion of more than her daily dosage of digoxin was denied.

Examination revealed an alert, anxious Negro female who was hyperventilating and vomiting.

![F. R. Lead 1](image_url)

Figure 2

Case 2. Lead I of the electrocardiogram. (a and b) A continuous tracing taken shortly before pacing. Sinus rhythm is present and the Q-T interval measures 0.52 sec. A few multifocal ventricular complexes lead into ventricular fibrillation which after a few seconds terminates spontaneously with resumption of sinus rhythm. Similar but longer episodes were associated with seizures. (c) Recorded during a clinic visit 6 months later. The Q-T interval measures 0.4 sec.
Blood pressure was 110/40, pulse 80 with bigeminy, and rectal temperature 100 F. Auscultation of the heart disclosed a loud apical third heart sound and murmurs characteristic of mitral and aortic regurgitation. Physical examination was otherwise negative. Since digitalis intoxication was suspected, potassium chloride was administered parenterally prior to withdrawal of blood for electrolyte determination. Several hours later, values for the serum potassium were 3.9 mEq/L, sodium 137 mEq/L, and chloride 100 mEq/L. The serum magnesium concentration was 0.7 mEq/L, increasing to 1.6 mEq/L by the sixth hospital day. Serum glutamic oxaloacetic transaminase (SGOT) values were 38, 78, 38, and 26 units and values for lactic dehydrogenase (LDH) were 525, 1,575, and 930 units. Hemoglobin, white blood cell count, urea nitrogen, glucose, and icteric index were normal. Two L.E. preparations were negative, and an ASO titer was 166 Todd units.

Hospital Course

Shortly after admission and coincident with the appearance of ventricular fibrillation on the electrocardiogram, the first of many grand mal seizures was observed. Between episodes, the rhythm was sinus and regular or bigeminal. Treatment with potassium chloride, procainamide, and diphenylhydantoin did not prevent the episodes (fig. 2a and b). Transvenous pacing was instituted, and ventricular capture was accomplished by pacing at 120/min preventing further arrhythmia. After 16 hours of pacing, the pacing unit was turned off, and normal sinus rhythm ensued. Although the Q-T interval remained prolonged for several days, the patient was asymptomatic and made an uneventful recovery.

When last seen, although episodes of “nervousness” and “hyperventilation syndrome” continued, she had no cardiac symptoms and was no longer taking digoxin or diuretics. Serum magnesium levels have been consistently normal since discharge. An electrocardiogram taken 6 months after discharge (fig. 2c) showed a Q-T interval of 0.4 sec.

Discussion

The management of patients with recurrent ventricular arrhythmias unassociated with heart block is frequently difficult. When factors such as electrolyte imbalance, hypoxia, or hypotension are present, their correction may suffice. In other instances ventricular irritability may be suppressed by drugs. All too frequently, however, these methods are either ineffective or require dangerous dosages of myocardial depressant drugs. Artificial cardiac pacing has recently been found effective in preventing recurrent ventricular arrhythmias in patients who, like our two patients, had repeated episodes not responsive to conventional drug therapy.14-20 Although the mechanisms by which pacing is able to suppress ventricular arrhythmias unassociated with heart block are obscure, increasing the heart rate appears to be an important factor.20 In addition, since rapid atrial pacing has been as effective as rapid ventricular pacing,15,18 changing the direction of myocardial depolarization probably plays no role.

In neither of our patients could the paroxysms of ventricular fibrillation be explained by their mild valvular lesions, and in neither was there evidence of active carditis. Both patients had hypomagnesemia without significant abnormalities of other serum electrolytes.

In case 1, the low serum magnesium can be explained by alcoholism and malnutrition.21-28 In case 2, the cause of hypomagnesemia is less certain, but may have been related to thiazide therapy.29 Since potassium was given to this patient before determination of serum electrolytes, hypokalemia could not be excluded; however, prolongation of the Q-T interval persisted after the serum potassium concentration was known to be normal. Anxiety and hyperventilation were prominent features of case 2, and both may be associated with ventricular fibrillation.6,80

Although neuromuscular abnormalities are commonly associated with clinical hypomagnesemia,31,32 consistent cardiovascular manifestations have not been described. In nine hypomagnesemic patients, Randall and associates26 found it difficult to interpret electrocardiographic changes because of associated diseases and other electrolyte deficiencies. Nonspecific ST-segment and T-wave abnormalities were noted and, in two instances, these disappeared after magnesium replacement. In Fankushen and co-workers,23 studies of hypomagnesemic alcoholics, one patient's electrocardiograms showed a prolonged Q-T
interval persisting after all serum electrolytes except magnesium had returned to normal. Electrocardiographic changes found in experimental magnesium deficiency have included a decrease in the P-R and QRS intervals and inverted T waves.\textsuperscript{33, 34} Seta and associates\textsuperscript{35} found T-wave peaking to be an early manifestation of magnesium depletion, but when magnesium deficiency was prolonged, the electrocardiographic changes resembled those of hypokalemia. These investigators also found that by making animals magnesium deficient, the duration of digitalis intoxication could be prolonged.\textsuperscript{36}

Any relationship between hypomagnesemia and the ventricular arrhythmias occurring in our patients remains speculative. In both patients prolongation of the Q-T interval was a prominent electrocardiographic feature. Others\textsuperscript{12, 17, 37, 38} have noted association between a prolonged Q-T interval and ventricular arrhythmias, but no single mechanism satisfactorily explains these observations. In the absence of overt hypokalemia or hypocalcemia, a prolonged Q-T interval might be explained by abnormalities in the relationship between intracellular and extracellular potassium. Failure of potassium to reenter the depolarized cell (delayed repolarization) or a diastolic leak of potassium from cells already repolarized (early depolarization) could, if asymmetrical, be recorded by the surface electrocardiogram as a prolongation of the Q-T interval. Asynchronous repolarization is thought by Palmer\textsuperscript{39} to promote aberrant conduction, reentry phenomenon, and ventricular fibrillation. The role played by magnesium in the above scheme is derived from its influence upon cell membrane permeability. In magnesium deficiency a reduction in the activity of the magnesium dependent enzyme (adenosine-triphosphatase) leads to a loss of intracellular potassium.\textsuperscript{36, 40, 41} Thus, the magnesium deficiency in our patients may have caused a loss of intracellular potassium leading to prolongation of the Q-T interval, increased vulnerability, and paroxysms of ventricular fibrillation.

References


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