



CASE REPORT

Hyponatremia Presenting as Cardiac Conduction Defect

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Abstract

Hyponatremia, the most common electrolyte disorder in hospitalized patients is usually asymptomatic. Clinical cardiac toxicity associated with hyponatremia has not been previously described, Although it is usually difficult to single out hyponatremia as the cause of conduction defects thus, we describe a case that developed reversible cardiac conduction defect temporally associated with hyponatremia.

Key Words

Heart Block, Hyponatremia, Atrio-ventricular Block, Left Bundle Branch Block

Introduction

Hyponatremia, the most common electrolyte disorder in hospitalized patients is usually asymptomatic (1). When clinical manifestations do occur they are usually related to central nervous system dysfunction; confusion, convulsions, coma and death (2, 3). Clinical cardiac toxicity associated with hyponatremia has not been previously described, although in patients with congestive heart failure the frequency of ventricular premature beats has been shown to correlate with the severity of hyponatremia. Although it is usually difficult to single out hyponatremia as the cause of conduction defects (4). In this report we describe a case that developed reversible cardiac conduction defect temporally associated with hyponatremia or its correction.

Case Report

A 75 year old female with hypertension, COPD, non-diabetic, was on antihypertensive treatment, bronchodilators and multivitamins. Because of inadequate control of blood pressure hydrochlorothiazide and amiloride was added. She was admitted with a single chest pain, vomiting, and loss of consciousness. On examination she was afebrile, oriented but drowsy. The pulse was 48/minute and blood pressure recorded was 100/60mmHg. There was no pallor, cyanosis, clubbing, pedal edema and JVP was not raised. Chest was clinically clear and cardiac examination was unremarkable. Neurological examination showed no focal neurological deficit. Electrocardiogram showed first degree

atrioventricular block with PR-interval of 240msec. and left anterior hemiblock (LAHB) (Fig1). A few hours later she became confused with heart rate of 36/mt. on examination and no change in blood pressure. On ECG complete AV-block was documented (Fig2), requiring insertion of temporary pacemaker. Pacemaker insertion did not alter mental status. The laboratory investigations found that complete blood count, renal function test, liver function test, lipid profile, thyroid hormone profile were all within normal limits. Troponin-T test was done by quantitative method which was negative. The cardiac enzymes sent revealed CK- 337 U/L, LDH- 432 U/L, AST- 69U/L, and ALT- 35 U/L. Chest X-ray showed mild cardiomegaly with cardio-thoracic ratio of 60%. ABG analysis at the time of AV-block revealed pH of 7.44, sodium of 110mmol/Lt. and potassium of 4.12mmol/Lt. (Table1). Hyponatremia was corrected by hypertonic saline. When serum sodium reached 125mmol/Lt., the patient became oriented and the ECG showed 2:1 AV-block. A day later ECG showed normal sinus rhythm and complete left bundle branch block. The left bundle branch block disappeared when serum sodium concentration was 130mmol/Lt. Transthoracic echocardiography revealed an ejection fraction of 50 % with normal valves, no regional wall motion abnormality and without any visible mass or thrombus. 24 hour Holter monitoring showed few premature ventricular complexes with no couplets or triplets or significant pauses. Coronary angiography was

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done on fifth day which showed normal coronaries with TIMI-111 flow in all the three vessels. Electrophysiological test was done on the same day and revealed normal HV-interval, sinus node conduction time, sinus node recovery time and corrected sinus node recovery time. The temporary pacemaker was removed on the 7th day and patient was discharged on antihypertensive medications (CCBs, ACEs, statins and salbutamol inhalations) and is on regular follow up without complications.

Discussion

Hyponatremia has not been previously associated with cardiac conduction defects, although co-occurrence of hyponatremia and sinoatrial block was described in one patient induced by carbamazepine therapy (5). Theoretically, reduction of the extra cellular concentration of sodium should slow cardiac pacemaker activity. In animal models, wide QRS complexes, either through hyperkalemia or quinidine administration, has been documented (6). Documentation of the effects of various sodium concentrations on the human myocardial conduction system is scarce. Garcia-Palmieri described four patients with hyperkalemia and hyponatremia who had wide QRS complexes. Three of these patients also had a moderate to severe metabolic acidosis. The patients received hypertonic saline with reversal of the ECG changes. This can be either attributed to a change in serum potassium, or to a modification of the cardiac effects of the hyperkalemia (7). In the specific hyponatremic patient, ascribing conduction defect to hyponatremia is usually complicated by other coexisting conditions which may have a direct deleterious effect on the conduction system; primary cardiac disease, diuretic and antiarrhythmic drugs, or other electrolyte abnormalities (8). We presented a case of thiazide-induced hyponatremia, which typically occurs in elderly women a few days after thiazide therapy is started. The unusual appearance of conduction defects could not be explained solely by a diseased conduction system, or by

Table 1. Course of Electrolyte and Electrocardiographic Changes

Hospital Day	Serum Na+ (mmol/l)	Serum K+ (mmol/l)	Serum Bicarb. (mmol/l)	Electrocardiogram
1	110	4.1		~LAHB & 1st degree AV- block
2	116	4.8	18	~Complete AV-block
3	125	5.3		~2:1 AV-block
4	131	5.1	24	Normal sinus rhythm
5	128	4.9		Normal sinus rhythm
6	135	5.3	22	Normal sinus rhythm
7	140	5.0	24	Normal sinus rhythm



Fig. 1 ECG Showing LAHB with 1st Degree Heart Block



Fig. 2 ECG Showing Complete Heart Block

myocardial ischemia. The close temporal association between the totally reversible conduction defect and hyponatremia strongly suggests that hyponatremia played a role in the pathogenesis of the conduction defect.

Conclusion

The association between cardiac conduction defects and hyponatremia is far from established. Future clinical experience with increased index of suspicion will help to establish the relation between these abnormalities.

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