Dear Sir,

We have reported a rare case which presented with tonic convulsion associated with sinus arrest due to hyperkalemia in a chronic hemodialysis patient. Long-term electrocardiographic (ECG) recording clearly demonstrated a close association of tonic convulsion with sinus arrest due to hyperkalemia.

Case Report

A 55-year-old man with a 2-year history of maintenance hemodialysis due to IgA nephropathy was found by his wife at about 10 a.m. on October 18, 1994. He had lost consciousness with tonic convulsions, legs stretched, arms flexed and eyes rolled upwards. He regained consciousness after several minutes and promptly consulted his family physician. No abnormal findings were noted except for slight ECG abnormality of a tall T wave. He was sent home with Holter monitoring. At 2:40 p.m. on the same day he lost his consciousness again and was brought to our emergency room. He was already alert again on arrival. Physical examination revealed no neurologically abnormal findings. Blood pressure was 152/92 mm Hg, pulse 62 beats/min and regular. Blood laboratory data were as follows: hematocrit 19.4%, blood urea nitrogen 65.4 mg/dl, creatinine 10.8 mg/dl, sodium 137 mEq/l, potassium 7.6 mEq/l, chloride 105 mEq/l, calcium 3.9 mEq/l. Arterial blood gas analysis showed pH 7.32, pO2 90.8 mm Hg, pCO2 32.4 mm Hg and HCO3 16.2 mmol/l. A 12-lead ECG tracing revealed a tall, peaked, symmetrical T wave with a narrow base seen in leads II, III, aVF and V2-6 which was consistent with that of hyperkalemia.

Fig. 1. A Holter ECG tracing before and at the time of convulsion.

arrest followed by brady-junctional rhythm, and two episodes of asystole with a maximum duration of 14 s detected at the same time when he lost consciousness; at 2:33 brady-junctional rhythm and ectopic sinus bradycardia with SA block followed by junctional escape. Neither head CT nor electroencephalogram showed any abnormality which might cause a tonic convulsion. Echo-cardiography revealed no cardiac function

Hemodialysis was performed immediately and resulted in a reduction of serum potassium to 4.3 mEq/l without detection of arrhythmias on ECG monitoring. Analysis of Holter ECG monitoring disclosed the following abnormalities (fig. 1): at 1:30 p.m. normal sinus rhythm,
60 beats/min and a tented T wave; at 2:29 p.m. ectopic sinus bradycardia with SA block followed by junctional escape, 28 beats/min; at 2:32 sinus.

Our patient developed sinus arrest at serum K levels of about 7.5 mEq/l before demonstrating other typical hyperkalemic ECG changes such as prolongation of the PR interval, disappearance of the P wave and widened QRS complex with a deep S wave. In humans, as in animals, SA block, either Wenckebach (type I) or Mobitz (type II) followed by junctional or ventricular escape rhythms, may occur [1], but hyperkalemia-induced sinus arrest which may cause convulsion is reported to be unusual [2], because the fibers of sinoatrial (SA) node and bundle of His are more resistant to hyperkalemia than atrial or ventricular myocardium [3]. The effects of K upon the ECG are greatly modified by other electrolyte disturbances, especially hyponatremia and hypocalcemia [4]. These electrolyte disturbances did not appear to have contributed to the development of sinus arrest, because neither hyponatremia nor hypocalcemia were observed in our patient. Our case suggests that hyperkalemia may induce heart block to cause a convulsion in chronic hemodialysis patients.

References

Convulsion and Hyperkalemia
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