Vomiting, hyperkalaemia and cardiac rhythm disturbances

A 51-year-old man presented with profuse vomiting and epigastric discomfort. The patient was known to suffer from a bipolar affective disorder with previous hospitalization for psychotic depression and attempted suicide. An ECG tracing showed normal sinus rhythm, inverted T waves in leads II, III and AVF and depressed ST segments in V4–V6 (Figure 1). Laboratory investigations revealed a serum potassium of 6.4 mmol/l, urea 28 mg/dl and a CPK level of 313 IU/l (normal 15–195). An initial diagnosis of acute coronary insufficiency was made and the patient treated with isosorbide dinitrate spray to no effect. Hyperkalaemia was managed by the administration of a glucose/insulin infusion and oral sodium polystyrene sulphonate resin. Although serum potassium level declined to 4.8 mmol/l, over the ensuing 24 h rhythm disturbances (as shown by the serial ECGs in Figures 2–4) developed culminating in refractory polymorphic ventricular tachycardia and the patient’s demise.

Questions

What is your diagnosis? What laboratory investigation would be most helpful? What treatment is indicated?
Answers to quiz on preceding page

The key to this patient’s diagnosis lies in the association of vomiting with hyperkalaemia in the presence of normal renal function. Under ordinary physiological circumstances severe vomiting will result in hypokalaemic, hypochloraeemic metabolic alkalosis. When faced with hyperkalaemia in such a situation, one must invoke either a tubular abnormality of potassium excretion or an inhibition of the Na-K ATPase pump. In the absence of any known renal disease and a normal blood gases analysis, the latter option seems logical. Given this patient’s history of depression and suicidal tendency, the ingestion of a pharmacological agent producing Na-K ATPase inhibition is more than likely. Of the commonly available drugs possessing such action, cardiac glycosides are probably the most frequent. Digitalis toxicity should, therefore, have been foremost in mind. A serum digoxin level (although belatedly taken in our case was >6 ng/ml) would best serve to clinch the diagnosis. Appropriate treatment in this patient with life threatening digoxin intoxication should have been the administration of digoxin specific Fab antibody fragments.

Discussion

Acute digitalis poisoning is a medical emergency with a reported mortality rate of 3–25% [1,2]. The most important step in the management of toxicity due to cardiac glycosides is its recognition. Serum digoxin levels do not always correlate well with symptomatic intoxication [3,4]. Furthermore, electrocardiographic abnormalities such as ventricular or atrial arrhythmias with or without some degree of concurrent atrioventricular block, often also occur in patients with congestive heart failure and/or underlying coronary atherosclerosis who are not receiving a cardiac glycoside. Added to these, is the currently much reduced usage of digitalis preparations.

In severe overdosage, the Na-K ATPase pump is markedly inhibited leading to elevation of the serum potassium [5,6]. Hyperkalaemia, increasing age, male sex and AV block have been reported to be adverse prognostic factors in acute digitalis poisoning [2].

Conventional therapy of digoxin toxicity consists of maintenance of serum potassium levels within the normal range, intravenous magnesium which is a cofactor of the Na-K ATPase transport system [1], antiarrhythmics and temporary pacemaking. In life threatening situations, management has been made much more effective by the availability of purified Fab fragments of anti-digoxin antibodies [3,4]. This agent provides a rapidly acting, safe antidote applicable to all commonly used digitalis preparations.

Suggested reading

5. Rosen MR. Cellular electrophysiology of digitalis toxicity. J Am Coll Cardiol 1985; 5 [Suppl A]: 22A–34A

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