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SHORT CASES IN CARDIOLOGY

Primary hyperparathyroidism presenting as torsades de pointes

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A previously well 47 year old woman presented to hospital complaining of syncopal episodes over the preceding 24 hours and of anorexia, nausea, polyuria, polydipsia, and generalised aches and pains for 2 weeks. She lost consciousness and her electrocardiograph showed short runs of multiform ventricular tachycardia (figure). These worsened, degenerating into ventricular fibrillation that required repeated DC cardioversion. The arrhythmia was refractory to intravenous lignocaine, magnesium, and amiodarone and stabilised only after a transvenous pacemaker set to 100 beats/min was inserted. Clinical examination was unremarkable. Serum biochemistry showed hypokalaemia (3.1 mmol/l) and hypercalcaemia (3.12 mmol/l). Twenty four hour urinary calcium was increased (13.5 mmol) and a parathormone assay (INTACT IRMA) showed a concentration of 517 pg/ml (normal range 5-15 pg/ml) that confirmed the diagnosis of primary hyperparathyroidism. A parathyroid adenoma weighing 2.79 g was located and excised and the patient made an uneventful recovery.

Hypokalaemia, a well recognised precipitant of torsades de pointes¹ is noted in approximately 5% of cases of primary hyperparathyroidism.² It is probably a result of proximal (type II) and, to a lesser extent, type I renal tubular acidosis. Secondary hyperaldosteronism resulting from intravascular volume depletion can also occur.

Hypercalcaemia also may have contributed. The increases in heart rate and contractility caused by parathormone are thought to be the result of direct induction of calcium influx into myocardial cells and the release of endogenous myocardial adrenaline.³ Hypercalcaemia shortens the plateau phase of the action potential, leading to a shorter QT interval. Re-entry is one proposed electrophysiological mechanism underlying torsades de pointes and in this context the positive chronotropic and QT-shortening effect of parathormone might be expected to protect against the arrhythmia by decreasing intraventricular dispersion of refractoriness. Calcium administration reduced the propensity to ventricular fibrillation by shortening the QT interval in dogs.⁴

There is, however, also evidence that an increased calcium concentration might have an arrhythmogenic effect by enhancing early afterdepolarisations, which have more recently been hypothesised to underlie torsades de pointes.¹

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Electrocardiograms recorded shortly after admission. (A) Short run of multiform ventricular tachycardia follows a long RR interval of the dominant cycle, starting with an R on T ventricular extrasystolic beat. (B) U waves (indicated by arrow) and a run of multiform tachycardia with typical axis changes and variable rate. ST depression may be the result of hypokalaemia and/or hypercalcaemia.