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Hypocalcemic heart block and torsade de pointes

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Torsade de pointes is a polymorphic ventricular tachycardia that occurs in the setting of QT prolongation, most commonly caused by electrolyte abnormalities like hypokalemia, various drugs and congenital long QT syndromes. Hypocalcemic torsade de pointes is a rare occurrence, with only a single reported case [1]. Hypocalcemia-induced second and third degree atrioventricular (AV) block has been described in the neonatal and pediatric populations [2]. We describe here the first case of an adult patient with both hypocalcemic torsade de pointes and second degree AV block.

An 82 year old patient with metastatic prostate cancer presented with upper extremity tetany. His serum potassium was 4.2 meq/l (normal 3.4––4.7 meq/l), magnesium was 1.7 meq/l (normal 1.3––1.9 meq/l), calcium was 4.9 mg/dl (normal 8.5––10.0 mg/dl), ionized calcium was 3.0 mg/dl (normal 4.74–5.2 mg/dl) and he had normal renal function. Electrocardiography on admission showed variable AV block including Wenckebach second degree AV block (Fig. 1) and transient third degree AV block. His QT interval was 0.73 s and QTc was calculated as 0.60 s. He was treated with calcium supplements and calcitriol. He had normal thyroid function, an elevated parathyroid hormone level of 96.3 pg/ml (normal 0.0–55.0 pg/ml) and a depressed vitamin D level of 8 ng/ml (normal 10–55 ng/ml). His hypocalcemia was thought to be secondary to hungry bone syndrome from metastatic prostate cancer. On hospital day 6 he developed a short tachycardia of torsade de pointes when his calcium was 7.3 mg/dl and

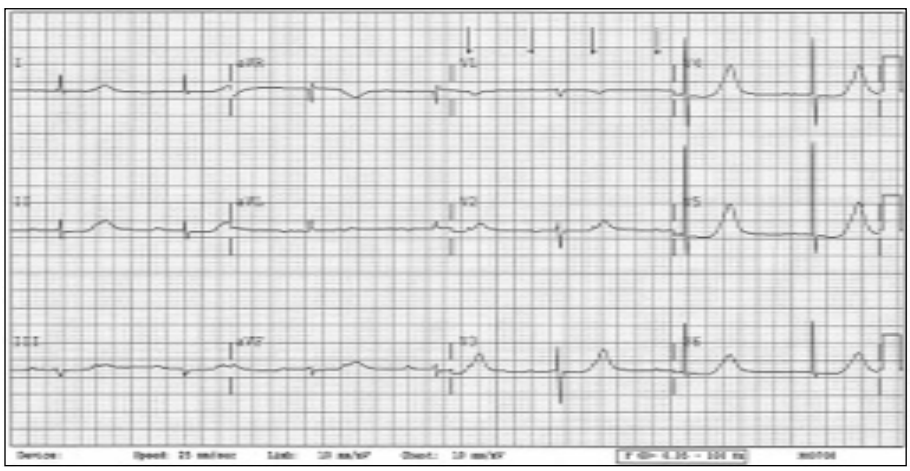

Figure 1. 12 lead electrocardiogram showing Wenckebach 2:1 atrioventricular block. P-waves in V1 chest lead are indicated by arrows.

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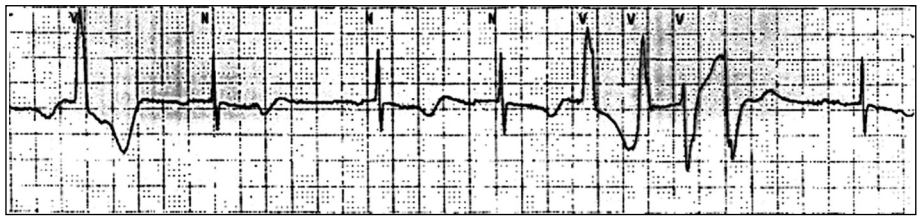

Figure 2. Telemetry strip showing torsade de pointes.

ionized calcium was 3.1 mg/dl (Fig. 2). His hypocalcemia was corrected and 24 hour holter monitoring revealed no recurrence of torsade de pointes. He was discharged home in sinus rhythm with calcium supplementation.

The occurrence of AV block in the presence of severe hypocalcemia is predicted by the contribution of both calcium and sodium ions in the genesis of the AV nodal action potential [3]. We believe this to be the first reported case of hypocalcemia induced second degree AV block as well as torsade de pointes occurring in the same patient. Awareness of torsade de pointes and AV block induced by hypocalcemia may lead to the identification of more such cases.

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