More than merely potassium: an uncommon cause for sine waves in renal failure

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An 68-year-old man with a background of end-stage kidney disease on haemodialysis due to diabetic nephropathy presented to hospital with increasing fatigue, deteriorating mobility and subjective generalised weakness. He had a diagnosis of Wolff-Parkinson White and had continued on flecainide-controlled release 200mg daily long-term for related atrial fibrillation, commenced when his renal function was normal. He had been receiving renal replacement therapy with haemodialysis for two years and had a degree of residual renal function, usually passing small volumes of urine multiple times daily. Flecainide levels had not been taken over this time, although an empiric dose reduction to 100mg daily was recommended at the time of commencing haemodialysis; community prescribing records show that this never occurred.

Due to his symptoms the patient was unable to attend his usual haemodialysis at a satellite unit on the morning of presentation, and he subsequently disclosed that his residual urine output had ceased three days prior. At the time of initial review, the patient was in an irregular bradycardia at 40bpm, and hypertensive at 183/66mmHg with a raised jugular venous pressure. Cardiovascular exam was otherwise unremarkable and the chest was clear to auscultation. Neurological examination was normal.

An ECG on arrival (Figure 1) demonstrated an irregular broad complex bradycardia with a single P wave. Initial blood tests revealed a mild hyperkalemia of 5.3mmol/L similar to usual pre-dialysis levels, sodium of 131mmol/L, and normal calcium, magnesium and phosphate. Plasma pH was normal and flecainide drug levels

Figure 1: Presenting ECG demonstrating broad-complex bradycardia with QRS prolongation and dominant terminal R wave in aVR (marked by arrow).
were initially unavailable. The patient was initially monitored overnight on telemetry and his flecainide withheld; this revealed a deterioration in rhythm with periods of a sine-wave pattern (Figure 2). By the following morning, his ECG demonstrated ongoing broad-complex bradyarrhythmia with marked QRS prolongation (Figure 3, left), and given a repeat potassium level of 5.8mmol/L, empiric treatment for flecainide toxicity with intravenous sodium bicarbonate was initiated. This resulted in near-immediate narrowing of the QRS (Figure 3, right). The patient was thereafter transferred to a tertiary centre for inpatient haemodialysis while continuing an intravenous bicarbonate infusion. A flecainide level taken on arrival returned at 4,280nmol/L, over twice the upper limit of the therapeutic range (2,100nmol/L). He was subsequently treated with pacemaker insertion to treat intermittent complete heart block, cessation of flecainide, and ultimately made a full recovery.

Discussion

This case demonstrates a prototypical case of flecainide toxicity, including the classical ECG findings. It demonstrates a number of important points regarding the use of this drug.

Flecainide is a Vaughan-Williams Class 1c anti-arrhythmic that slows cardiac conduction through sodium channel blockade.1 It is licenced for use in the treatment of paroxysmal tachyarhythmia including atrial fibrillation and flutter, supraventricular tachycardia, and ventricular tachycardia in patients with structurally normal hearts and no history of ischaemic heart disease due to its association with increased mortality outside of these settings.1,2 Flecainide is primarily renally excreted with a highly variable elimination half-life that averages 20 hours, although this can extend beyond 50 hours in the setting of end-stage kidney disease.1,3 Less than 1% of the drug is removed by haemodialysis.1 Avoidance of the drug is recommended in significant renal impairment unless benefit is thought to outweigh risk and careful drug monitoring is undertaken.1

This case demonstrates the classical ECG findings of flecainide toxicity, and by extension sodium channel blockade. There is marked prolongation of the QRS with subsequent degeneration of rhythm into a sine-wave pattern, and a dominant terminal R wave in aVR. Prompt recognition of these features facilitates emergent management of which intravenous sodium bicarbonate is the mainstay, with the use of intravenous lipid emulsion, pacing and ECMO also described.4–6 The rapid narrowing of QRS duration that follows intravenous sodium bicarbonate therapy, thought mediated by the combination of alkalinisation and sodium ion load, is also demonstrated in this case.7

Ultimately the vast majority of patients who present with flecainide toxicity will survive; acute intoxication has been documented historically to have a mortality rate of 10%.8 Whether this rate accurately extrapolates to the setting of chronic cumulative exposure seen in our case remains unknown.
Figure 3: Twelve lead ECG immediately before (above) and after (below) the administration of intravenous sodium bicarbonate, demonstrating narrowing of the QRS complex with this treatment.

Competing interests:
Nil.

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