



Case Report

An irregular, extremely broad QRS complex rhythm

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ABSTRACT

We report a case of a 74-year-old lady admitted to the emergency department with a very broad QRS complex caused by flecainide intoxication due to acute renal failure. Appropriate recognition of the ECG changes and symptoms provoked by flecainide intoxication permitted quick directed treatment with intravenous administration of high dose sodium bicarbonate, which resolved the QRS elongation.

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1. Introduction

Flecainide overdose can induce life threatening ventricular arrhythmias and cardiogenic shock [1]. Noncardiac symptoms include nausea, vomiting, seizures and altered level of consciousness [2]. Involuntarily flecainide intoxication, though rare, is associated with mortality up to 22,5% [3]. Despite being a rare cause of QRS widening, a prompt diagnosis in the emergency department allows the physician to reverse the intoxication and prevent further harm.

2. Case

A 74-year-old lady was brought to the emergency department because of repetitive falls. The patient was conscious, had a known mild cognitive impairment and complained of extreme fatigue with nausea. Her vitals were stable with a blood pressure of 114/74 mmHg, heart rate of 86 bpm, respiratory rate of 18 bpm and a body temperature of 37 °C. Clinical examination revealed a pale patient with prolonged capillary refill time. Auscultation revealed bilateral pulmonary crackles and irregular heart sounds. There were no clear signs of sustained trauma from falling and the neurological examination was normal. Her surface ECG showed an irregular, broad QRS complex rhythm without any discernible atrial activity. QRS morphology was most compatible with a very wide right bundle branch block and left anterior fascicular block, explaining the left axis deviation (Fig. 1). Blood analysis showed: serum creatinine 3,12 mg/dL (eGFR 14 mL/min/1,72m²); sodium 121 mmol/L; potassium 4,2 mmol/L and bicarbonate 19 mmol/L. The

fractional excretion of sodium was 0,6%, suggesting renal hypoperfusion.

Her husband, being in charge of the medication, stated that no errors were made. She took levothyroxine 100 µg, pantoprazole 20 mg, rosuvastatine 10 mg, perindopril 2,5 mg, and indapamide 0,625 mg. Her cardiologist recently started flecainide 150 mg, bisoprolol 2,5 mg and apixaban 5 mg b.i.d. because a Holter monitoring revealed paroxysmal atrial fibrillation. Afterwards, her general practitioner associated bumetanide 1 mg to treat peripheral edema.

Clinical and lab examination excluded hyperkalemia, hypercalcemia and hypothermia as potential cause of QRS widening, rendering drug toxicity the most probable cause. In fact, the ECG changes are typical for sodium blocker intoxication as seen with tricyclic antidepressants and flecainide [4]. Drug panel was negative for tricyclic antidepressants, narrowing the diagnosis down to flecainide accumulation due to acute renal failure.

The patient received a 150 mEq sodium bicarbonate bolus followed by continuous infusion of 8,4% sodium bicarbonate titrated for pH 7,5. After the initial infusion, a spectacular change in QRS duration was noted (Fig. 2). During the first hours, the patient developed several episodes of non-sustained ventricular tachycardia with immediate and spontaneous recuperation. In the following days, the QRS normalized and she was discharged in good clinical condition (Fig. 3).

3. Discussion

Here we present a patient with an irregular wide QRS complex rhythm. Given the absence of tachycardia, ventricular tachycardia (VT) and supraventricular tachycardia (SVT) with aberrant conduction can be ruled out. Distinction must then be made between accelerated idioventricular rhythm (AIVR), sinoventricular rhythm (when the

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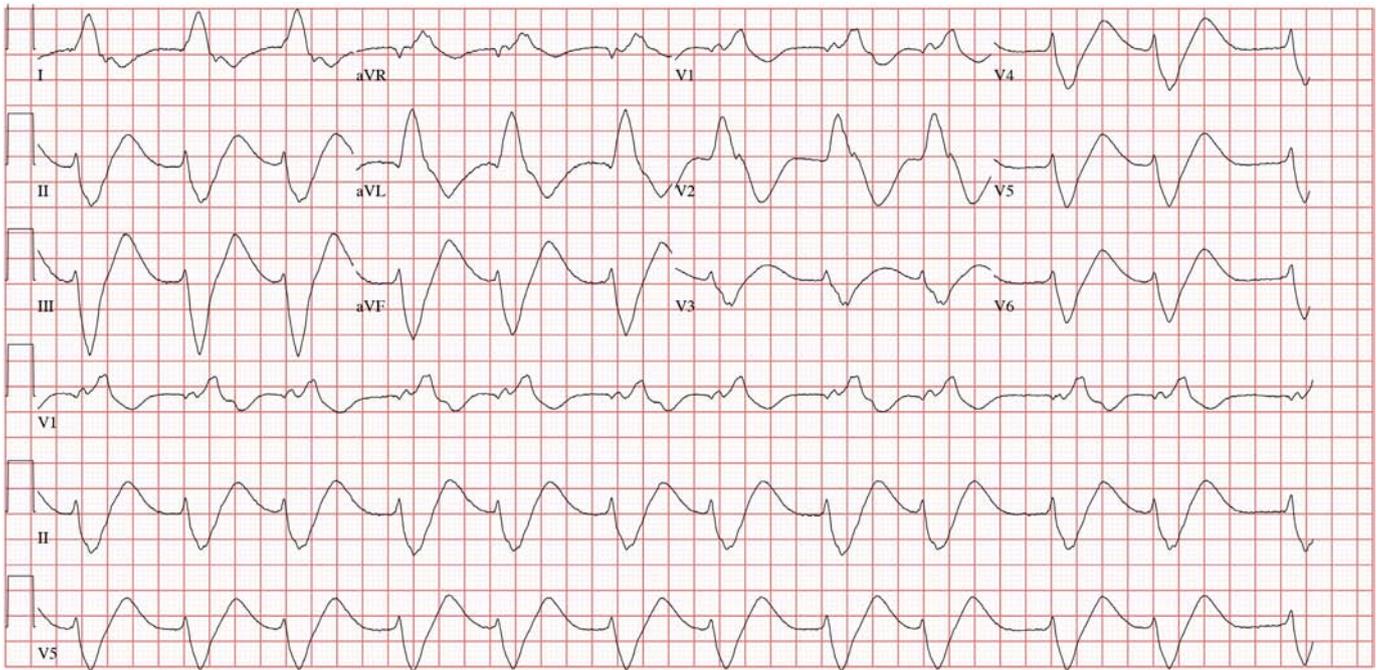


Fig. 1. Surface ECG on admission showing an irregular, broad QRS complex. The QRS interval measures 260 ms.

sinus node continues to propagate electrical activity to the ventricles without depolarizing the atrial muscle, a hallmark of severe hyperkalemia) and ECG changes induced by a toxin or medication.

Accelerated idioventricular rhythm (AIVR) can be excluded because the broad complex rhythm is irregular and it is mostly seen in some specific clinical circumstances such as after myocardial reperfusion in acute coronary syndrome, in digoxin intoxication or after return of spontaneous circulation (ROSC) following cardiac arrest. Serum potassium is normal. Another possible explanation for the QRS widening is sodium channel blocker intoxication. Since a drug panel is negative for tricyclic antidepressants, flecainide intoxication is the most likely diagnosis.

Flecainide is a class Ic anti-arrhythmic agent most often used in the treatment of supraventricular tachycardia. It depresses the upstroke of the cardiac action potential through inhibition of fast sodium channels in a dose-dependent manner, resulting in slower conduction in the atria, ventricles and his-purkinje conduction system with QRS widening [1,5,6]. Flecainide has a long elimination half-life (10–24 h), that is prolonged in cases of renal failure, liver insufficiency, heart failure and urinary alkalization. [2]

Knowledge on the treatment of flecainide intoxication is based on case reports. The therapeutic aims are (1) detoxification through reduction of absorption, (2) antagonizing the effect and (3) treatment of

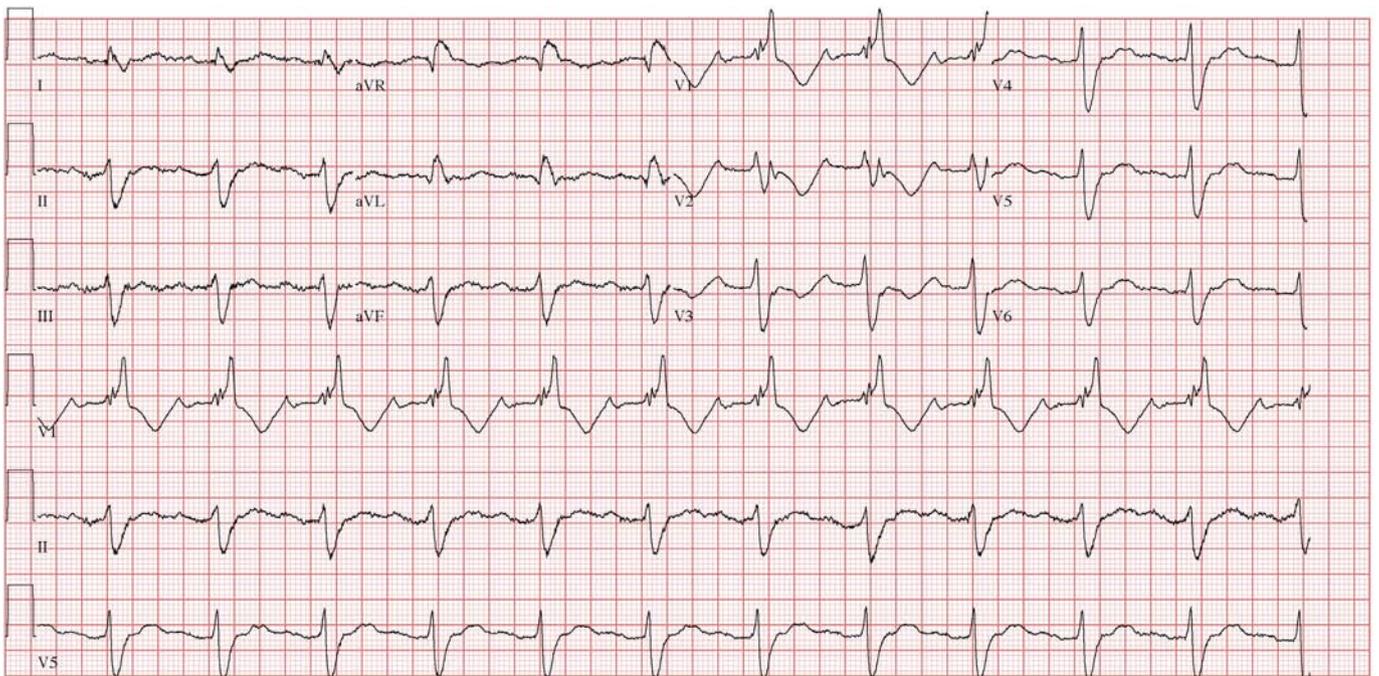


Fig. 2. 2 h after admission and sodium bicarbonate infusion, ECG changes were spectacular. The QRS is markedly narrowed and measures 200 ms.

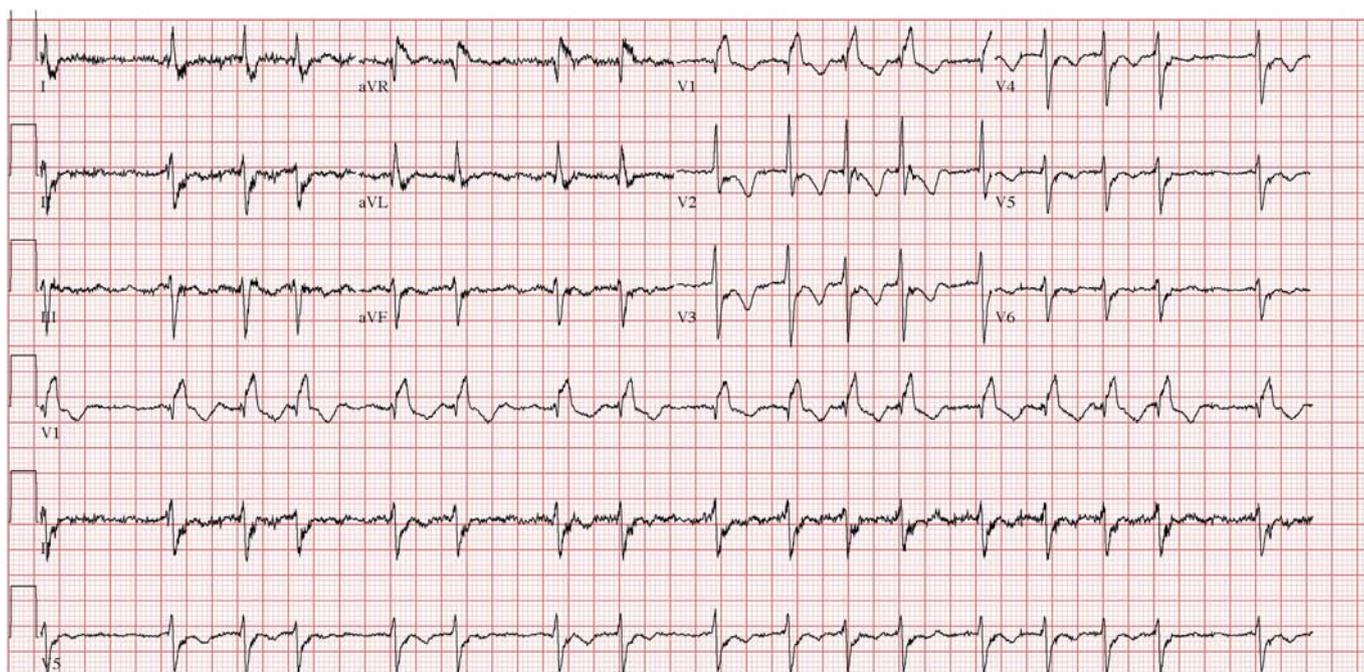


Fig. 3. At discharge the QRS interval measured 140 ms which was comparable with older ECG registrations. Note the recurrence of atrial fibrillation.

shock with maintaining adequate perfusion to vital organs while elimination occurs. Detoxification can be achieved by active charcoal, though solely in the hours directly after ingestion. The contribution of extracorporeal detoxification methods is little, due to a low extraction ratio and a large distribution volume. [2,7] Furthermore, anti-arrhythmic drugs such as lidocaine and amiodarone as well as fat emulsion therapy have been used anecdotally but were not used in our patient.

The standard therapeutic option to antagonize flecainide, is high dose intravenous sodium bicarbonate since high sodium levels compete with flecainide at the high voltage gated sodium channels. Furthermore, sodium bicarbonate alkalinizes the serum preventing flecainide binding through electrostatic repulsion thereby facilitating dissociation from the sodium channel [8]. Sodium bicarbonate administration should be initiated as early as possible, though the precise dose is unclear. Experts advise 50–100 mEq boluses followed by a continuous infusion targeting an alkaline pH of 7.50–7.55 [2]. While the effect can be dramatic and instantaneous, hypernatremia is a necessary evil and should be accepted.

4. Conclusion

This case highlights the importance of prudent use of anti-arrhythmic drugs in the poly-medicated elderly. The acute renal failure in our patient caused accumulation of flecainide leading to the typical ECG appearance of sodium blocker intoxication with extreme QRS

broadening and potentially lethal conduction disturbances. The concomitant hyponatremia, most likely a consequence of the thiazide diuretics, worsened the conduction abnormalities. High dose of intravenous sodium bicarbonate proved to be an effective and safe treatment.

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