Paroxysmal narrow complex tachycardia secondary to hypokalaemic periodic paralysis

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Introduction

Hypokalaemia is a known trigger of atrial arrhythmias (Weiss et al, 2017). Common causes of hypokalaemia include fluid loss, medications and endocrine disorders. Hypokalaemic periodic paralysis is a rare channelopathy resulting in episodic hypokalaemia, commonly presenting with sudden severe muscular weakness or total paralysis, and responding to potassium replacement. Cases of arrhythmia associated with hypokalaemic periodic paralysis have been described, but always in the context of severe concomitant weakness (Shires, 1978; Canpolat et al, 2012). This article describes a case of hypokalaemic periodic paralysis presenting predominantly with tachycardia, without significant neurological involvement.

Case report

This woman first presented at the age of 21 years with recurrent episodes of palpitations with associated breathlessness. These episodes lasted up to a day at a time, requiring frequent hospital admission. In between episodes, she was asymptomatic. Initially, the frequency of the episodes was one to two per year. Her medical background at presentation included asthma, for which she took a regular inhaled long-acting β2-adrenergic agonist and inhaled salbutamol as required. There was no significant family history. Electrocardiography during the symptomatic episodes revealed a regular, narrow complex tachycardia, with loss of the isoelectric baseline, morphologically consistent with atrial flutter (Figure 1). She remained haemodynamically stable during these episodes. Clinical examination did not reveal evidence of decompensated heart failure. She reported mild generalised muscle weakness during the episodes, although neurological examination showed near-normal muscle strength in the upper and lower extremities. Echocardiography, repeated on several occasions, showed good biventricular systolic function, with no significant valvular disease.

During the symptomatic attacks, her serum potassium levels were consistently between 2.3 and 3.3 mmol/litre (normal range 3.5–5.1 mmol/litre). In between episodes, her serum potassium levels were between 3.5 and 5.0 mmol/litre. The tachyarrhythmias did not respond to intravenous adenosine or beta-blockade, but reverted to sinus rhythm following correction of the hypokalaemia. While occasionally her symptom onset coincided with recent salbutamol use, this was not consistently the case. She had no features suggestive of gastrointestinal loss of potassium, and no laxative or diuretic use. Serum magnesium levels, thyroid function tests and early morning cortisol levels were normal, and urinary electrolytes confirmed normal potassium excretion. Urinary pH and serum bicarbonate level were normal, making a renal tubular acidosis unlikely.

By the age of 40 years, the tachyarrhythmia had become more frequent, with up to a dozen episodes per year. Regular antiarrhythmics, including flecainide and amiodarone, were unsuccessful in reducing the frequency of the episodes. A trial of spironolactone also failed to prevent the attacks. At 42 years of age, she underwent an electrophysiology study. There was no inducible tachycardia with a standard stimulation protocol, no evidence of an accessory pathway, and no evidence of dual atrioventricular node physiology.

At the age of 45 years, because of her unexplained recurrent hypokalaemia, she was referred to a nephrologist in the authors' hospital, who suspected a diagnosis of hypokalaemic periodic paralysis. This was subsequently confirmed by a specialist in the condition in a separate centre. Genetic testing for common channelopathies associated with hypokalaemic periodic paralysis (including CACNAS1S, SCN4A and KCNJ2) was negative. She was started on acetazolamide (a carbonic anhydrase inhibitor), uptitrated to a dose of 1 g daily. Over the next 4 years, the frequency of her attacks reduced to one to two per year. These continue to be treated with intravenous potassium replacement, with rapid resolution of her tachycardia and symptoms.

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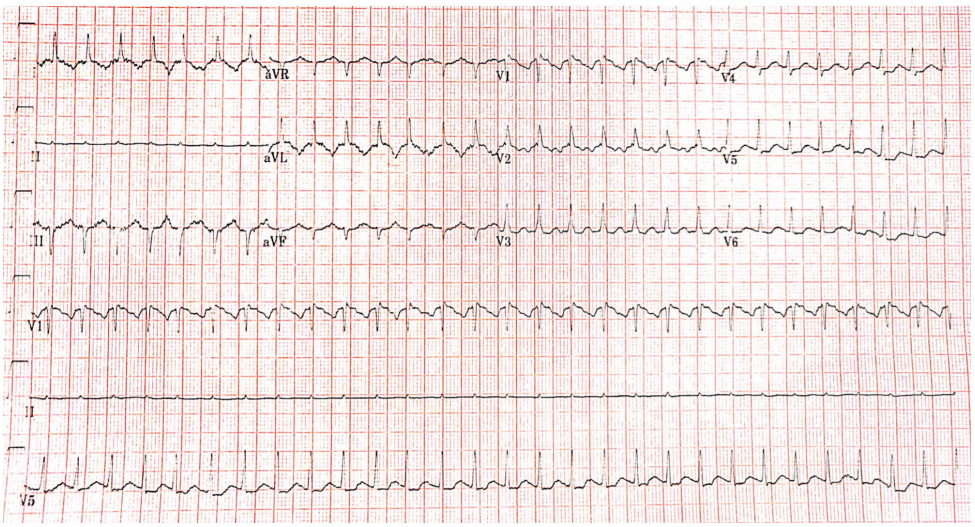

Figure 1. An illustrative 12-lead electrocardiogram, performed when the patient was 31 years old, demonstrating a regular narrow complex tachycardia, with ventricular rate of 171 bpm and loss of isoelectric baseline.

Discussion

This case is the first description of a patient with paroxysmal episodes of supraventricular tachycardia associated with recurrent and sudden hypokalaemia as a result of hypokalaemic periodic paralysis, in the absence of concomitant severe muscular weakness. According to the supportive diagnostic criteria of hypokalaemic periodic paralysis (Statland et al, 2018), the suggestive features of hypokalaemic periodic paralysis in this case are:

1. Onset in second decade of life
2. More than two attacks with documented serum potassium <3.5 mmol/litre
3. Attack duration longer than 2 hours
4. Improvement with potassium intake
5. Exclusion of other causes of hypokalaemia (renal, adrenal or thyroid dysfunction, renal tubular acidosis, diuretic or laxative abuse).

Electrolyte imbalance, particularly hypokalaemia, is associated with increased risk of bradyarrhythmias and tachyarrhythmias. Hypokalaemia is responsible for increased abnormal automaticity (Sohinki and Obel, 2014) and re-entry circuits (Osadchii, 2010) resulting in arrhythmogenicity. These mechanisms are caused by increased resting membrane potential, increased duration of the action potential and increased duration of the refractory period secondary to hypokalaemia. Hypokalaemia has been associated with triggered arrhythmias such as torsades de pointes, polymorphic ventricular tachycardia and ventricular fibrillation (Nordrehaug et al, 1985).

In this case, a diagnosis of hypokalaemic periodic paralysis was made after two decades. The delay in establishing a diagnosis was multifactorial. First, absence of severe episodic weakness made the diagnosis of hypokalaemic periodic paralysis less obvious. There was also no family history suggestive of the condition. Furthermore, her presentations during the first few years often coincided with recent salbutamol administration, and it was initially thought that this was inducing the hypokalaemia. However, she subsequently experienced many episodes without associated salbutamol use. While β2-agonist therapy may have occasionally exacerbated a drop in serum potassium levels, it cannot, in isolation, explain the recurrent episodes. The normal thyroid function tests ruled out the possibility of secondary hypokalaemia periodic paralysis.

Conclusions

Hypokalaemic periodic paralysis, which usually presents with significant neurological involvement, can present with predominant cardiac involvement, manifesting as recurrent narrow complex tachycardia. In this case, long-term treatment with acetazolamide reduced the frequency of tachyarrhythmias, and rapid treatment with intravenous potassium successfully reverted the tachyarrhythmia to sinus rhythm.

Learning points

- Hypokalaemia can be a trigger for atrial arrhythmias.
- Hypokalaemic periodic paralysis can present predominantly with palpitations and narrow complex tachycardia, without significant neurological involvement.
- Long-term treatment with acetazolamide may reduce the frequency of tachyarrhythmias in the context of hypokalaemic periodic paralysis.

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