The demon drink

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CASE REPORT

A 42-year-old woman was brought in by ambulance to our accident and emergency department with a short history of blackouts. Her neighbours had alerted the emergency services after they heard her screaming late at night. On the way to hospital she appeared to faint for short spells broken by screaming on recovery. Initially, it was felt that she may have been fitting or even psychiatrically disturbed. One of the staff remarked that she looked as if she was 'possessed by demons'.

Later, it was discovered that she had a long problem of alcohol abuse, drinking upwards of five bottles of vodka a week. She lived alone and no longer worked. She had no history of renal or cardiac disease. About a month before, she had been admitted with a urinary tract infection and alcohol withdrawal. She had continued to drink fitfully since. She was on no medications, prescribed or otherwise.

On observing her first 'blackout' in the accident and emergency department, the patient was attached to a monitor. This showed a run of ventricular tachycardia during which she lost consciousness. She was cardioverted with 200J to sinus rhythm. On regaining consciousness, she screamed loudly and was extremely distressed, as previously described. She had further episodes of ventricular tachycardia which self-terminated. A bolus dose of 100 mg lignocaine was given intravenously followed by a continuous infusion without any effect.

A 12-lead electrocardiogram (ECG) showed a markedly long QT interval of 0.7 seconds. A diagnosis of torsade de pointes was made and the patient was given a bolus dose of magnesium followed by a continuous infusion with an immediate beneficial effect. There were no further runs of ventricular tachycardia and all ectopic activity vanished. Electrolytes subsequently showed a deranged picture of hypomagnesaemia (0.44 mmol/l), hypocalcaemia (1.94 mmol/l corrected) and hypokalaemia (3.0 mmol/l). Magnesium supplements were continued over the following few days, but there were no further rhythm abnormalities. All other electrolyte problems reversed without intervention. The ECG subsequently returned to normal. Before discharge, we reiterated the importance of abstinence, but unfortunately she failed to attend follow-up counselling.

DISCUSSION

Torsade de pointes is an amorphous ventricular tachcardia, first defined in the 1960s, showing a characteristic electrocardiographic (ECG) pattern of a continuous twisting of the QRS axis around the baseline (Dessertenne, 1966). Prolongation of the QT interval is seen consistently in the beat preceding the arrhythmia and often in the 12-lead ECG. The runs of ventricular tachycardia usually self-terminate but may degenerate into ventricular fibrillation. Congenital forms are rare, and the majority of acquired forms are caused by drugs that delay repolarization, such as tricyclic antidepressants.

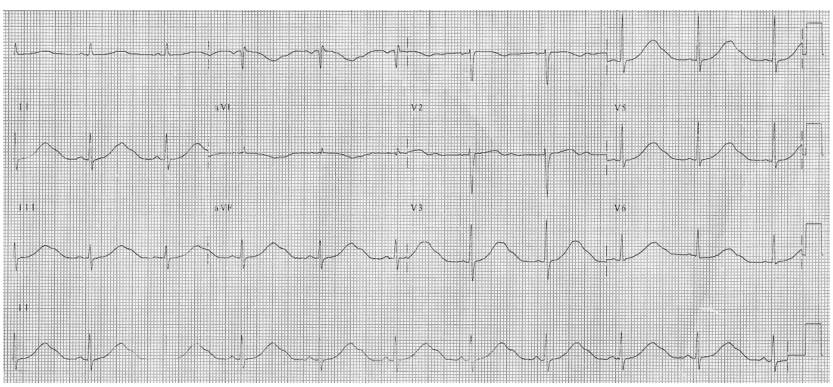

Figure 1. Original abnormal electrocardiogram.

Torsades can also be caused by a variety of electrolyte disturbances, e.g. hypomagnesaemia and hypokalaemia (Napolitano et al, 1994). Alcohol abuse has been shown to lead to a variety of electrolyte disturbances in the presence of normal liver and glomerular function (De Marchi et al, 1993). De Marchi et al showed that transient defects in renal function are common in long-term alcoholics on abstaining, and may contribute to electrolyte derangement. These defects are independent of liver or glomerular function and, in most cases, disappear within 4 weeks.

In this case, the patient had a long-term alcohol problem and presented with recurrent blackouts caused by runs of ventricular tachycardia. Lignocaine treatment was unsuccessful but subsequent intravenous magnesium gave immediate results. Lignocaine, like other antiarrhythmic agents, may delay repolarization and not only be unhelpful, but possibly deleterious.

Magnesium has been shown to be effective in suppressing drug-induced torsades de pointes, even in the presence of normal magnesium levels (Tzivoni et al, 1984). It has also been shown to abolish ventricular arrhythmias associated with hypomagne-

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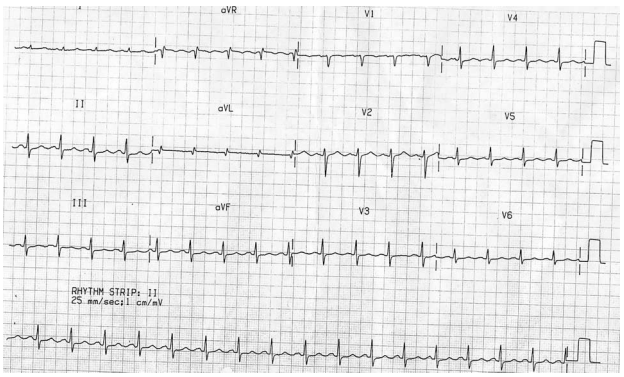

Figure 2. Electrocardiogram taken 4 years before presentation.

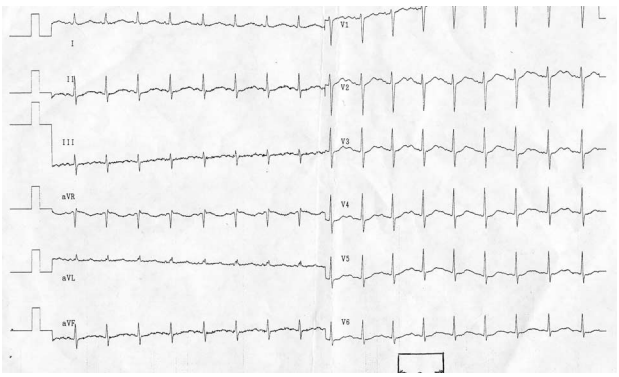

Figure 3. Electrocardiogram several months later, showing reversal of changes.

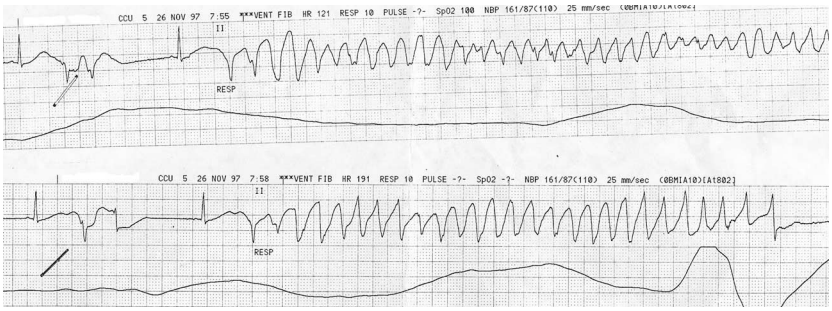

Figure 4. Rhythm strips of patient.

saemia (Iseri et al, 1975). The exact mechanism of its action is unknown.

Our patient responded promptly to treatment in the presence of severe hypomagnesaemia and other electrolyte disorders. This diagnosis should be considered in all alcoholics who present with blackouts, and clinicians should be alert to the high prevalence of electrolyte abnormalities in this group. **HM**

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Dessertenne F (1966) Un chapitre nouveau d'electrocardiographie: les variations progressives de l'amplitude de l'electrocardiogramme. *Actual Cardiol Angeiol Int (Paris)* **15:** 241–58

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Napolitano C, Priori SG, Schwartz PJ (1994) Torsade de Pointes. mechanisms and management. *Drugs* **47(1):** 51–65

Tzivoni D, Keren A, Cohen AM et al (1984) Magnesium therapy for torsades de pointes. *Am J Cardiol* **53:** 528–30