Ventricular arrhythmias complicating weight reduction therapy in a patient with a prolonged QT interval

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Summary: Serious ventricular arrhythmias are known to occur in patients with long QT intervals. We describe a case of torsade de pointes occurring in a patient with a prolonged QT interval while taking a 1000 calorie diet for 3 weeks; bendrofluazide 5 mg/d and diethylpropion hydrochloride (Tenuate Dospan) and bendrofluazide. In patients with long QT intervals, hypokalaemia and drugs which further delay repolarization may facilitate the development of life threatening arrhythmias.

Case report

A 35 year old housewife presented with a 24 h history of brief episodes of loss of consciousness. She had been taking a 1000 calorie diet for 3 weeks; bendrofluazide 5 mg/d and diethylpropion hydrochloride 75 mg/d were added for 10 d prior to admission. On admission the pulse was irregular and the blood pressure was 120/80 mm Hg. Cardiovascular and neurological findings were otherwise normal. Biochemical investigations revealed a reduced serum potassium of 2.4 mmol/l (normal range 3.5–4.5 mmol/l). All other biochemical and haematological tests were normal. The electrocardiogram revealed sinus rhythm, with frequent unimor- phological ventricular ectopics. The measured QT interval (QTm) was 0.4 s during a heart rate of 100 beats/min, and the corrected QT interval (QTc) according to Bazett (1920) was 0.52 s (normal less than 0.44 s). The QRS and ST segments were normal and U waves were present. Chest radiograph and echocardiogram were normal.

Cardiac monitoring showed episodes of ventricular tachycardia, of a torsade de pointes morphology (Figure 1), associated with episodes of loss of consciousness. Treatment included the withdrawal of bendrofluazide and diethylpropion hydrochloride, potassium replacement and an intravenous infusion of mexiletine which suppressed ventricular arrhythmias. Two days later when the patient was normokalaemic, the QTc was prolonged at 0.50 s (QTm 0.42 s, heart rate 85 beats/min) and U waves were absent (Figure 2).

Three months later investigations were performed when the serum potassium was 3.9 mmol/l. The electrocardiogram revealed that QTc was persistently prolonged to 0.48 s (QTm 0.42 s, heart rate 77 beats/ min). No ventricular arrhythmias were induced by maximal treadmill exercise testing. An intracardiac electrophysiological study revealed normal conduction times and no ventricular arrhythmias were induced with programmed right ventricular stimulation, either before or after intravenous isoprenaline (7 μg/min for 5 min). The QTm during identical rates of atrial pacing was unchanged following the isoprenaline infusion. Subsequently, 100 mg/d of atenolol has shortened the QTc to 0.42 s (QTm 0.43 s, heart rate 51 beats/min) and the patient has remained asymp-

Figure 1 Torsade de pointes.

Figure 2 Shows prolonged QT interval (QTc 0.50 s) after correction of hypokalaemia.

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tomachic during 8 months follow up. In addition 6 first
degree relatives were studied and found to have
normal QT intervals.

Discussion

Torsade de pointes is an atypical ventricular tachycar-
dia which has been described in patients with congen-
ital long QT syndromes and acquired prolonged QT
intervals (Kossman, 1978; Krikler & Curry, 1976;
Soffer et al., 1982). Its association with sudden death is
well known (Jervell & Lange-Nielsen, 1957; Stubbs et
al., 1976). This patient’s markedly prolonged QT
interval on admission shortened following the with-
drawal of drugs and correction of hypokalaemia.
However, in the absence of metabolic abnormalities
and any drugs, the QT interval remained moderately
prolonged 3 months later. This suggested that the
patient had a long standing prolonged QT. It has been
previously reported that the QT interval prolongs
during identical atrial pacing following isoprenaline in
patients with congenital long QT syndromes (Milne
et al., 1982). In contrast, patients with acquired and
idiopathic prolonged QT intervals have unaltered QT
intervals after isoprenaline (Milne et al., 1982). The
diagnosis of idiopathic prolonged QT in this patient
was suggested by the response of the paced QT
interval to isoprenaline and the absence of affected relatives.

Rapid weight loss in patients using liquid protein
near starvation diets, has been reported to prolong the
QT interval and induce torsade de pointes and sudden
death (Brown et al., 1978; Isner et al., 1979). The
pathogenesis remains unknown. This patient’s weight
loss was not rapid, and torsade de pointes has not been
associated with less rigorous weight reduction diets.

Torsade de pointes has been described after the use
of several drugs which prolong the QT intervals,
including class 1 antiarrhythmic drugs and phenothia-
azines (Fowler et al., 1976; Meltzer et al., 1978).
Although diethylpropion hydrochloride has sympa-
thetic actions it has not been reported to prolong
the QT interval (Schreiber et al., 1968). The drug has a half
life of 16 h and it is unlikely to have caused persistent
QT interval prolongation 3 months later. Furthermore,
as sympathetic stimulation does not prolong the QT
interval in patients with idiopathic long QT syndrome, it is unlikely that diethylpropion hydrochloride contributed to the patient’s markedly
delayed repolarization at admission. However, sympa-
thetic stimulation with isoprenaline has been repor-
ted to further prolong repolarization in patients with
congenital long QT syndromes and diethylpropion
hydrochloride may further delay repolarization in this
particular group of patients (Milne et al., 1982).

Hypokalaemia, hypomagnesaemia and, rarely, hy-
pocalcaemia have been associated with the induction of
torsade de pointes in patients with QT abnorm-
alities (Stubbs et al., 1976; Curry et al., 1976; Khan et
al., 1981). Hypokalaemia, due to a variety of aetologies, has been found to cause a high incidence of
arrhythmias when the plasma-potassium falls below
3.2 mmol/l, but there are few reports of life threatening
arrhythmias (Curry et al., 1976). The incorrect use of
diuretics and the development of hypokalaemia may
have facilitated the induction of torsade de pointes in
this patient with an underlying prolonged QT interval
and a predisposition to ventricular arrhythmias.

Following the correction of hypokalaemia and
withdrawal of drug, there was no spontaneous or
inducible ventricular arrhythmia. Therefore great cau-
tion is required in the use of drugs which cause
hypokalaemia or further delay ventricular repolariza-
tion in patients with long QT intervals.

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