"TORSADE DE POINTES" — A UNIQUE VENTRICULAR ARRHYTHMIA
REPORT OF EIGHT CASES AND REVIEW OF THE LITERATURE

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SYNOPSIS

Eight cases of torsade de pointes seen over a period of 3 years are described. The morphological features, aetiology and treatment of this unique arrhythmia are reviewed. It is emphasized that recognition of torsade de pointes is not an academic matter because it requires urgent treatment which are quite different from the classical ventricular tachycardia.

INTRODUCTION

"Torsade de pointes" is a unique ventricular tachycardia characterised by distinctive electrocardiographic abnormalities and aetiological causes which differ from the usual type of ventricular tachycardia. The diagnosis of this uncommon cardiac arrhythmia is extremely important because the therapeutic approach is completely different from the usual way of treating ventricular arrhythmias. We describe in this paper our experience with eight cases of torsade de pointes which we encountered in the last three years highlighting the importance of early recognition and the role of cardiac pacing in the treatment of this arrhythmia.

CASE REPORTS

Case 1

A 60 year old asymptomatic Chinese woman was referred for bradycardia. The ECG showed complete heart block with narrow QRS complexes and a ventricular heart rate of about 45 beats per minute. Whilst being monitored in the CCU, she developed recurrent syncope which was found to be related to bursts of ventricular tachycardia which exhibited the characteristic features of torsade de pointes (Figure 1). The serum potassium was normal. Temporary right ventricular cardiac pacing was performed and this completely abolished the ventricular tachycardia. The patient refused permanent cardiac pacing. She was maintained on oral Sarventrine and has remained well after temporary cardiac pacing was stopped.

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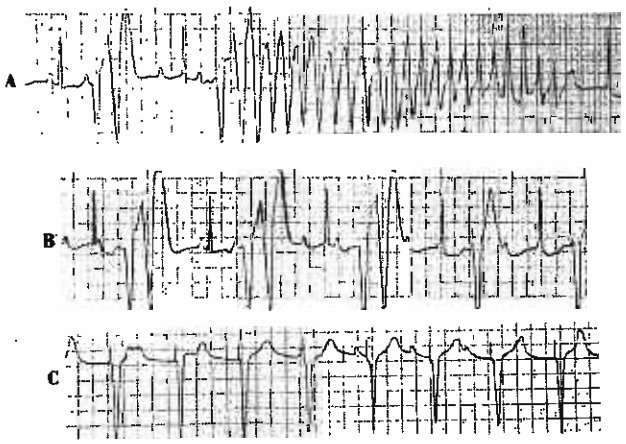

Fig. 1 (A) ECG showing complete heart block and short burst of torsade de pointes
(B) ECG showing frequent ventricular ectopic beats
(C) ECG showing suppression of ventricular arrhythmias with cardiac pacing.

Case 2

A 65 year old Chinese man presented with recurrent syncope. His ECG showed complete heart block with a slow ventricular rate, frequent ventricular ectopic beats and bursts of torsade de pointes (Figure 2). He has refused permanent cardiac pacing and still suffers from recurrent syncope.

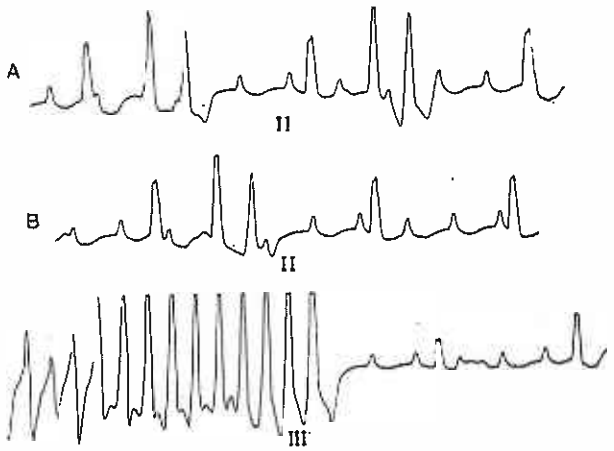

Fig. 2 ECG showing complete heart block and torsade de pointes (ECG strips A & B are continuous).

Case 3

A 60 year old man was monitored in the CCU for extensive anterior myocardial infarction. He developed complete heart block with a ventricular rate of 40/min. Soon after, bursts of torsade de pointes was noted. Temporary right ventricular pacing completely abolished the arrhythmia. He died two days later of cardiogenic shock.

Case 4

A 55 year old Chinese man suffered an anteroseptal transmural myocardial infarction. Whilst being monitored in the CCU, he developed on the second day recurrent bursts of ventricular tachycardia. This frequently degenerated into ventricular fibrillation which required electrocardioversion. The serum potassium was 4.2 milliequiv/litre. The ECG showed a slow junctional rhythm of about 42 beats/min and despite intravenous atropine the heart rate could not be accelerated. The QTc was 0.36 sec. Over the next 4 hours he exhibited extreme ventricular irritability as evidenced by multiple episodes of torsade de pointes and fifteen episodes of ventricular fibrillation, despite maximal dosages of intravenous Lignocaine (Fig. 3). Temporary coronary sinus pacing completely suppressed the ventricular irritability and ventricular fibrillation (Figure 3). Cardiac pacing was stopped three days later and the patient made an uneventful recovery subsequently.

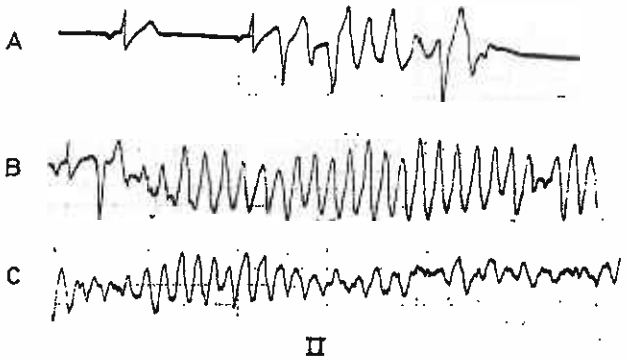

Fig. 3 ECG showing slow junctional rhythm and multiple episodes of torsade de pointes degenerating into ventricular fibrillation (ECG strips A, B & C are continuous).

Case 5

A 60 year old man with chronic coronary artery disease was treated with oral Quinidine (600mg daily) for several months because of frequent ventricular ectopic beats. On follow up, a routine ECG showed a markedly prolonged QTc of 0.52 sec and short bursts of torsade de points (Figure 4). The serum potassium was normal. Quinidine toxicity was diagnosed and this drug was immediately stopped. The patient refused admission to hospital. On follow up one week later, the ECG showed a normal cardiac rhythm. Oral Quinidine was recommenced at a lower dosage.

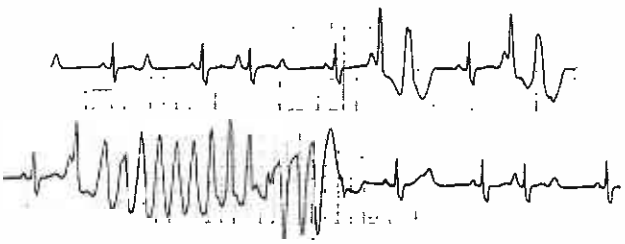

Fig. 4 ECG showing markedly prolonged QTc and torsade de pointes (ECG strips continuous).

Case 6

This patient has been previously described in full (1). In summary, she was a 73 year old Chinese woman with chronic ischaemic heart disease and was treated with 600mg oral Disopyramide for frequent ventricular ectopic beats. Seven months later, she presented with syncope and continuous ECG monitoring revealed a markedly prolonged QTc of 0.52 sec and about 25 attacks of ventricular tachycardia and/or ventricular fibrillation which were unresponsive to maximal doses of intravenous Lignocaine. The serum potassium

was normal. The electrocardiogram showed typical torsade de pointes. With temporary right ventricular pacing all arrhythmias were completely suppressed and she made an uneventful recovery subsequently.

Case 7

A 68 year old Chinese woman underwent laparotomy for haematemesis one week after partial gastrectomy for chronic duodenal ulcer. At operation, refashioning of the partial gastrectomy was done. Post operatively, whilst being monitored in the ICU, she exhibited multiple episodes of self-limiting torsade de pointes which was unresponsive to intravenous Lignocaine (Figure 5). At this time the serum potassium was noted to be 2.1 milliequiv/litre. After correction of the hypokalaemia by intravenous potassium chloride, the ventricular arrhythmias were abolished. However, she subsequently died because of septicaemia.

Case 8

A 50 year old man was monitored in the CCU for recent myocardial infarction. On the evening of the second post infarct day, he exhibited a burst of torsade de pointes progressing to a short period of ventricular flutter which reverted spontaneously (Figure 6). The serum potassium was normal and the QTc was 0.40 sec. The medical officer on call elected not to institute treatment because of the transient nature of the arrhythmia. Subsequent monitoring did not reveal any further cardiac arrhythmias and he made an uneventful recovery.

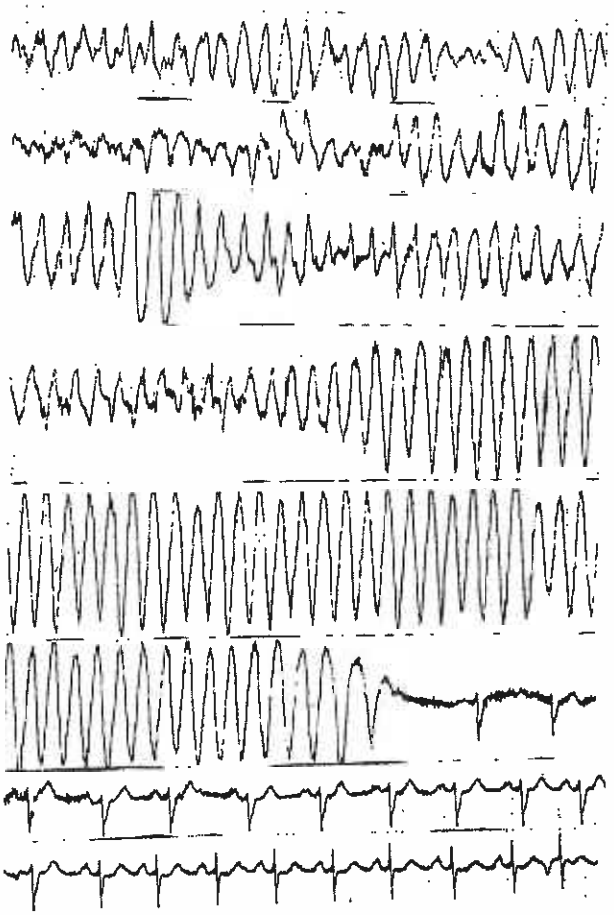

Fig. 6 ECG showing torsade de pointes progressing to a short run of ventricular flutter which reverted spontaneously (ECG strips continuous).

DISCUSSION

Although torsade de pointes was first described by Dessertenne in 1966 (2), and has been a well recognised entity amongst the French cardiologists in the last decade, this cardiac arrhythmia is still relatively unknown amongst the cardiologists of the English speaking world. The name torsade de pointes describes the appearance of the unique electrocardiographic findings which resemble the twisting (torsade) of the QRS complexes along an imaginary iso-electric point (de pointes). The arrhythmia consists of paroxysms of ventricular tachycardia in which the QRS axis undulates over runs of 5 to 20 beats with definite changes in direction. In contrast, attacks of the usual ventricular tachycardia are characterised by

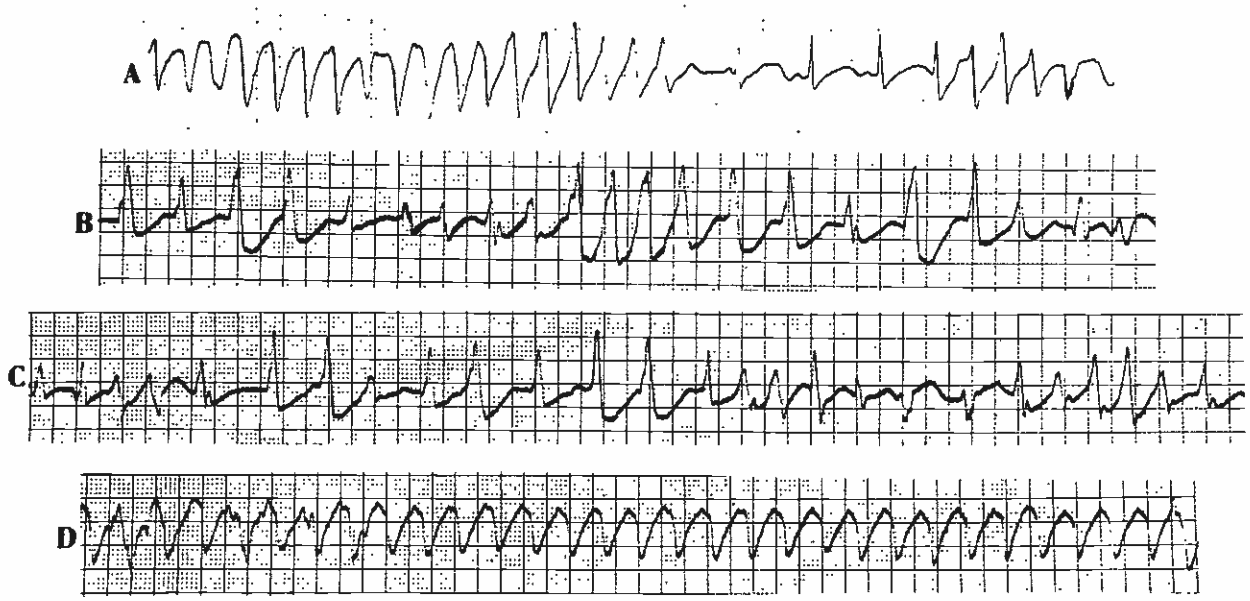

Fig. 5 Non-continuous ECG strips A, B & C showing torsade de pointes. ECG strip D shows ventricular tachycardia.

QRS complexes of similar morphology (Figure 7). Bidirectional ventricular tachycardia, another rare arrhythmia which is nearly always due to digitalis intoxication typically presents with ventricular beats which alternate in their morphology and without the undulating feature which is the hallmark of torsade de pointes (Figure 8). A variety of other names have been applied to this arrhythmia and they include "atypical ventricular tachycardia", "polymorphous ventricular tachycardia", "paroxysmal ventricular fibrillation", "cardiac ballet" etc. (3-5) The attacks may be brief or prolonged and may progress to ventricular fibrillation as was seen in our cases no. 3 and 5.

Table 1 summarises the aetiological causes of torsade de pointes. It is clear that these aetiological factors are different from those which cause the usual ventricular tachycardia. Of great importance is the fact that many of the drugs which are employed to suppress the usual type of ventricular tachycardia can cause torsade de pointes. Failure to diagnose this arrhythmia and exhibition of antiarrhythmic drugs worsens the clinical situation. The aetiology in our case no. 4 is most likely Quinidine toxicity and this is supported by the markedly prolonged QTc of 0.52 sec.

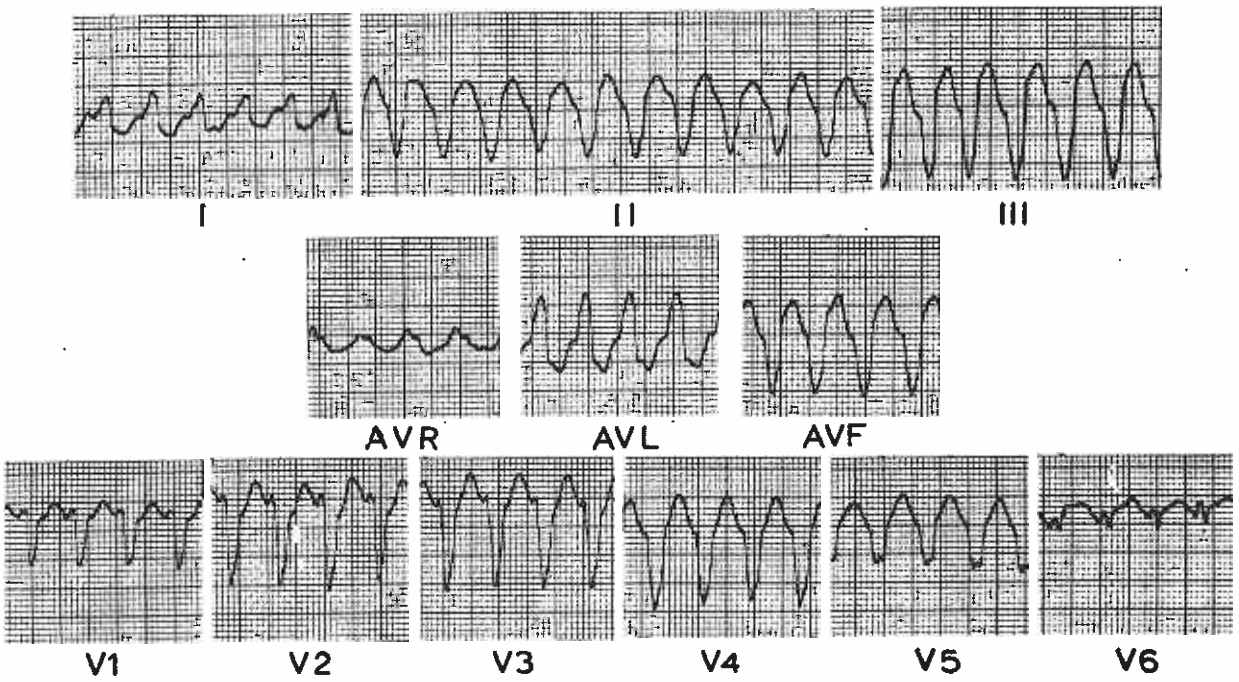

Fig. 7 ECG showing classical ventricular tachycardia.

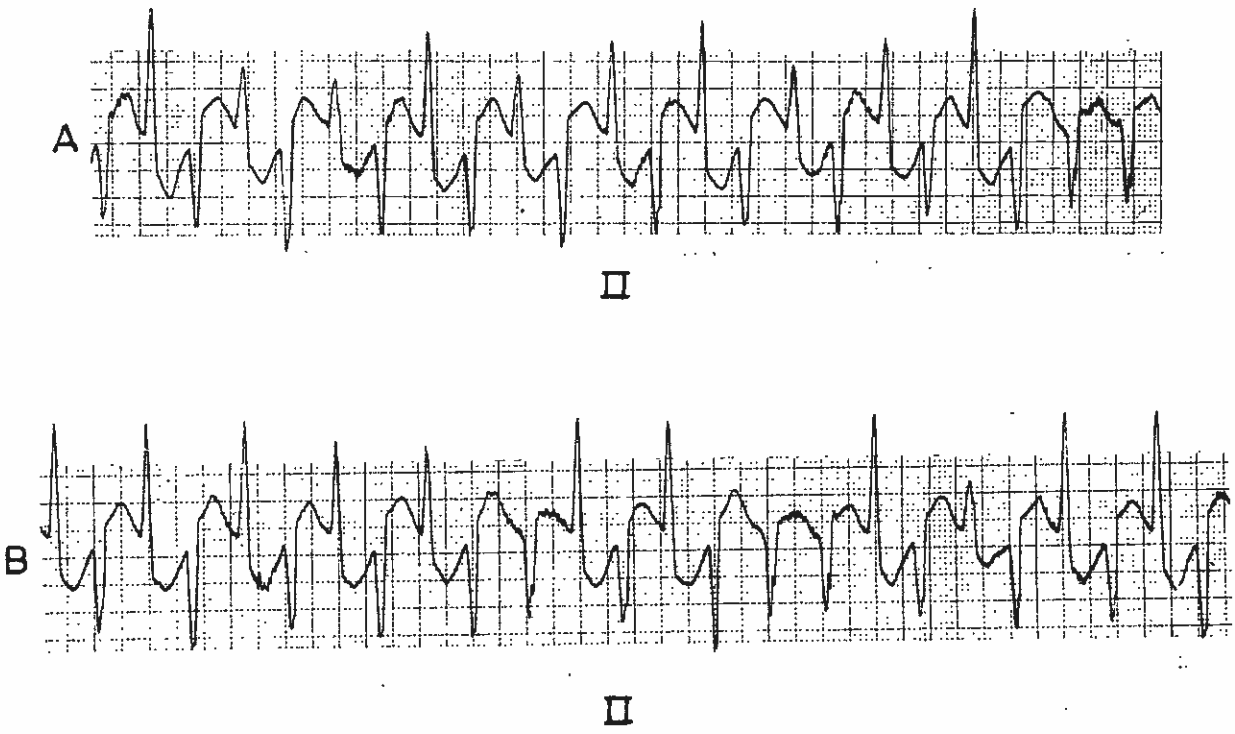

Fig. 8 ECG showing bi-directional tachycardia.

TABLE 1
Recognized causes of torsade de pointes (Krikler 1976) (6)

1) Slow basic rhythm
   a) Sinoatrial depression/disease
   b) High-degree AV Block
2) Electrolyte deficit(s)
   a) Potassium
   b) Magnesium
3) Congenital QT prolongation syndromes
   a) Overt, with deafness
   b) Forme fruste
   c) Concealed
4) Drugs
   a) Cardioactive agents: quinidine, lignocaine, procaineamide, prenylamine, etc
   b) Psychotropic agents: phenothiazines, tricyclic antidepressants, ? other major transquillizers
5) Cardiac ischaemia
6) Myocarditis

Case no. 5 is particularly interesting because torsade de pointes resulted from oral Disopyramide given in the conventional dosages. With the increased use of Disopyramide, it has been suggested that ventricular tachyarrythmias as a complication should be looked for, and the drug should perhaps be stopped in the face of a markedly prolonged QTc to anticipate this complication. The other drugs which have been reported to produce torsade de pointes are Lignocaine, Procainamide, Prenylamine, the Phenothiazines and the Tricyclic anti-depressants. Bradycardia as a cause was present in four of our seven patients. In cases no. 1, 2 and 3, it was due to complete heart block and in case no. 4, it was due to a very slow junctional rhythm. Hypokalaemia has also been reported to be an aetiological cause and was responsible in our case no. 6. The arrhythmia did not respond to Lignocaine, but with the correction of hypokalaemia, it was suppressed. In the rare congenital syndromes in which the QT interval is prolonged (Jervell and Lange Nielsen or Ramano Ward) torsade de pointes has been found to be an important complication. Lastly, torsade de pointes is reported to be a rare complication of myocardial infarction as was seen in our case no. 7.

Of crucial importance to the treatment is the recognition and diagnosis of the arrhythmia and the clinical setting in which it occurs. Clearly the underlying situation e.g. hypokalaemia or quinidine intoxication, must be corrected. In some instances where the arrhythmia is transient, this measure alone is sufficient. It is also extremely important to realise that anti-arrhythmic drugs usually aggravate the situation and thus contraindicated. This is clearly apparent in our cases no. 3 and 5, where maximal doses of Lignocaine had absolutely no effect on the arrhythmia. Isoprenaline infusion to shorten repolarization has been used with success and is being recommended. A major advance in treatment is the use of cardiac pacing to control the arrhythmia. This can sometimes be life saving as was evident in our cases no. 1, 3 and 5. In our opinion, temporary cardiac pacing should always be performed if the arrhythmia is prolonged and causes haemodynamic problems or degenerates into ventricular fibrillation. Meanwhile, all attempts should be made to remove any aetiological cause which may be present.

In conclusion, torsade de pointes is a unique ventricular arrhythmia with important aetiological, diagnostic and therapeutic implications. It needs to be widely known and its recognition is not an academic exercise because it requires urgent treatment which are quite different from the classical ventricular tachycardia.

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