Pseudo torsade de pointes in Parkinson’s disease

Drs Erisa Ito, Martina Mason and James Adams report a case of a patient with Parkinson’s disease with marked Parkinsonian tremor and possibly epileptiform activity, whose electrocardiogram led to the misdiagnosis of torsade de pointes. They highlight the complexities of interpreting electrocardiograms and the importance of recognising artefacts in order to avoid unnecessary interventions.

It is known that body movements, poor skin-electrode contact, recorder malfunction and electromagnetic interference can cause electrocardiogram (ECG) recording artefacts and lead to the misdiagnosis of life threatening arrhythmias. Patients with Parkinson’s disease often have abnormal movements that can induce ECG artefacts mimicking ventricular and supraventricular tachycardias.

Torsade de pointes is a cardiac arrhythmia that may cause blackouts or even sudden death in a patient with a structurally normal heart. The phrase ‘torsade de pointes’ is French, meaning literally ‘twisting of the points’. This ventricular arrhythmia was so named due to the characteristic change in amplitude and twisting of the QRS complexes about the isoelectric axis. It usually resolves spontaneously but it often recurs and has the potential of degenerating into ventricular tachycardia and/or fibrillation, so it is very important to recognise it early and initiate appropriate treatment. Here is a case illustrating how Parkinson’s tremor simulating torsade de pointes could have subjected the patient to unnecessary interventions.

Case report (used with permission)
An 82-year-old gentleman was admitted to Southampton General Hospital with recurrent syncopal episodes. He had a history of poorly controlled Parkinson’s disease and hypothyroidism, and was taking Sinemet® 62.5mg six hourly, levobyroxine 75 mcg once daily and Calcichew D3 Forte® two tablets once daily.

Initial clinical examination was unremarkable, apart from marked parkinsonian features. ECG revealed sinus rhythm with no acute ischaemic changes; chest x-ray imaging and blood results — including thyroid function test — were within normal limits. Differential diagnosis included cardiac arrhythmia, under-treated Parkinsonism and postural hypotension. Parkinsonian treatment was increased to 125mg of Sinemet® six hourly and a 24-hour tape was requested. The following day, the patient had improved remarkably, with better control of his parkinsonian symptoms.

Two days later, however, he experienced another syncopal episode. The patient was cardiovascularly stable with blood pressure (BP) 130/80mmHg and pulse 78/minute, ECG showed sinus rhythm and the neurological system was intact, apart from twitching of the right arm which resolved spontaneously. The diagnosis of a possible fit was raised and he was started on sodium valproate. Subsequent CT of the head revealed age-related volume loss only and an electroencephalogram showed no diagnostic epileptiform activity.
In the early hours of the following day, the duty doctor was called to review the patient for possible further fits. On arrival, the patient was fully alert, cardiovascularly stable with a BP of 129/80mmHg, pulse 92/minute and the clinical examination was unremarkable apart from upper limb shaking. An ECG taken at this time is shown in Figure 1.

The diagnosis of torsade de pointes was made and while awaiting appropriate treatment, a further ECG was obtained (Figure 2), which showed sinus rhythm. Cardiology opinion was sought and the patient was not on any medications causing prolongation of QT (the measure of time between the start of the Q wave and the end of the T wave; the T-wave on the ECG is caused by the repolarisation of the ventricle). There was no evidence of long QT on the baseline ECG and the QRS complexes seen on the trace (arrows, Figure 1) were normal (QRS complex on the ECG is caused by the contraction of the ventricles). It was thought the most likely cause of the ECG appearance was artefact caused by the involuntary movement, rather than torsade de pointes as originally suspected. His 24-hour tape was non-diagnostic and the patient was discharged few days later. Diagnosis of epilepsy was established on clinical grounds and he continues on antiepileptic treatment.

Discussion

The ECG taken during the syncopal episode shows coarse repetitive regular waveforms occurring at a frequency of 3.3Hz, which may be consistent with parkinsonian-like tremor. The ECG suggests asymmetry of the tremor, involving predominantly the right arm, as lead three of the ECG is the least affected by the artefact (Figure 3, see page 32). The jerky movements were also involving the torso as all the precordial leads were showing the abnormal waveforms. It is difficult to be sure whether the artefact is purely caused by parkinsonian tremor or by other abnormal movements. The fact the ECG taken a few minutes after the syncopal episode showed baseline artefact (the patient’s Parkinsonian resting tremor causing fine fluctuations in the ECG baseline recordings) only, and the fact the patient responded clinically to antiepileptic treatment, would suggest these movement artefacts may have been caused by epileptiform activity.

It is easy to misinterpret electrocardiographic artefact as ventricular tachycardias and an interesting study by Knight et al in 2000 demonstrated this by surveying 766 physicians. They found 94 per cent of general physicians, 58 per cent of cardiologists and 38 per cent of electrophysiologists failed to recognise artefacts simulating wide-complex tachycardia. They also found the majority of physicians that had misdiagnosed the rhythm strip, had then gone on to recommend an invasive procedure for further evaluation or therapy. In 2001, they reported 12 cases where misdiagnosis resulted in patients undergoing unnecessary interventions, which included cardiac catheterisation (three patients) and unnecessary medical therapy (nine patients). In one case, the patient underwent a permanent pacemaker implantation after misdiagnosis of torsade de pointes.

The case report here along with these studies therefore demonstrate the importance of recognising the possibility of ECG artefacts in patients otherwise haemodynamically stable and asymptomatic. Our patient did not have to undergo any further invasive investigations or treatment, thanks to easy access to early expert cardiology opinion from a consultant with special interest in electrophysiology.
Body movements can cause ECG artefacts that may be mistaken for life-threatening arrhythmias.

Evidence suggests that, in general, physicians are poor at identifying ECG artefacts.

It is important for all physicians to be aware and to have some ability to recognise ECG artefacts in order to prevent unnecessary investigations and interventions.

However, the following are some characteristics which would help differentiating artefacts from ventricular arrhythmias:
- absence of haemodynamic compromise;
- normal QRS complexes within the artefact (arrows, Figure 1);
- unstable baseline on the ECG before and/or after the event;
- the relation of the arrhythmia with patient’s body movements.

This case highlights the complexities of interpretation of ECGs and the importance of the physicians’ awareness and ability to identify artefacts. The ‘take home’ message is that the ECG should always be interpreted in the context of the patient’s condition and other factors present at the time of recording.

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