ECG QUIZ

NGAI-SHING MOK
Department of Medicine & Geriatrics, Princess Margaret Hospital, Kowloon, Hong Kong

PING-TIM TSUI

CHEE-WO WU

Follow this and additional works at: https://www.jhkcc.com.hk/journal

Recommended Citation

This ECG Quiz is brought to you for free and open access by Journal of the Hong Kong College of Cardiology. It has been accepted for inclusion in Journal of the Hong Kong College of Cardiology by an authorized editor of Journal of the Hong Kong College of Cardiology.
Our patient was a 61-year-old lady who had a known history of hypertension, diabetes mellitus, old cerebral infarcts and epilepsy. In February 2000 she was admitted to Princess Margaret Hospital after a fall which resulted in fracture neck of femur. In ward she was witnessed to suffer from cardiac arrest. External electrical defibrillation with 200 Joules successfully resumed her vital signs.

**Questions**

1. What were the ECG abnormalities?
2. What was the most likely underlying cause for her cardiac arrest?

---

**Figure 1.** Rhythm strip showing patient's cardiac rhythm during cardiac arrest.

**Figure 2.** 12-lead ECG recorded immediately following successful defibrillation.
Comments

The first rhythm strip showed a polymorphic ventricular tachycardia (250 beats/min).

On the second ECG, pacing spikes of large amplitude were seen suggesting a unipolar pacemaker system. They were however not followed by any P wave or QRS complex implying a failure to capture. Pacemaker sensing function was normal (constant QRS to pacing spike interval of 1000 ms). There was bradycardia (heart rate 40 bpm) with variable PR intervals. A closer look at long lead II will find that some P waves were buried within QRS complexes or T waves and there was atrioventricular (AV) dissociation diagnostic of complete AV block. The QRS complexes were wide (136 ms) and the T waves were bizarre-looking which were wide and deeply inverted. The QT (736 ms) and QTc (588 ms) intervals were grossly prolonged.

The constellation of clinical and ECG findings in this patient suggested that her cardiac arrest was due to bradycardia dependent torsades de pointes (TdP) despite she already had a pacemaker implanted for her complete AV block.

Discussion

This patient first presented in 1998 with syncope. ECG confirmed complete AV block and so a permanent VVI pacemaker was implanted in her right pectoral region with pacing rate set at 60 beats/min. All along there was no documentation of TdP. The pacing and sensing thresholds have been good and stable.

TdP is an important complication of complete AV block. This case illustrated that it may not be totally avoided even in patients who already have permanent pacemaker implanted. Strasberg et al. compared patients with complete AV block with TdP to those without TdP, and found that the QT interval in patients with TdP was longer than in those without TdP, whereas heart rate and QRS interval during the escape rhythm were not significantly different. Kurita et al. studied 14 patients with complete AV block with TdP (TdP[+] group) or without TdP (TdP[-] group) before and after pacemaker implantation. They found that the QT and QTc intervals were significantly longer in the TdP[+] group than the TdP[-] group before pacemaker implantation (Figure 3). After pacemakers were implanted, the QT interval in the TdP[+] group was significantly prolonged compared with the TdP[-] group when the pacing rate was decreased to ≤ 60 beats/min. The authors concluded that complete AV block with TdP had a bradycardia-sensitive repolarization abnormality. The critical heart rate that induced abnormal QT prolongation in the TdP[+] group was ≤ 60 beats/min and therefore the pacemaker rate in these patients must be set at ≥ 70 beats/min to avoid recurrence of TdP.

All along this patient had good pacing and sensing thresholds of her pacemaker. The battery of the pacemaker was not depleted and lead impedance was normal. She did not take any anti-arrhythmic drug which might have raised the pacing threshold. It was therefore unlikely that her TdP was due to bradycardia following pacemaker failure. Rather, she had a propensity to develop bradycardia dependent TdP that occurred while she was being paced at a rate of 60 beats/min which was not sufficiently fast to correct the underlying repolarization abnormality. Transient loss of capture by the pacemaker due to an acute rise in pacing threshold has been frequently reported following both external and internal defibrillation. One possible reason may be due to the damage of the myocardium at the myocardium-electrode interface as a result of high-energy currents being shunted to the pacemaker circuit during the defibrillation.

Other causes of or contributing factors to TdP should also be considered in this patient. Her serum potassium, calcium and magnesium levels were normal. She did not take any drug which might prolong her QT interval or have any known myocardial disease. One important differential diagnosis to consider in this patient is intracranial haemorrhage (especially subarachnoid haemorrhage) which might present with cardiac arrest due to TdP accompanied by QT prolongation and giant inverted T waves (neurogenic pattern) on ECG (during sinus rhythm). CT brain in this patient did not show any haemorrhage.
Figure 3. ECG during the escape rhythm in 2 local patients with complete AV block with TdP (A) and without TdP (B). QT and QTc intervals in (A) were not prolonged and T waves looked normal. However, the QT and QTc intervals were markedly prolonged in (B). The T waves exhibited an abnormal and bizarre contour.

References