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A puzzling electrocardiogram

Received: 21 June 2003
Accepted: 22 October 2003
Published online: 17 December 2003
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Sir: A 36-year-old man was admitted to the intensive care unit in a deep coma requiring mechanical ventilation 14 h after heroin inhalation. The patient was regularly receiving lithium, chlorpromazine and diazepam for an on-going psychotic disorder. His family reported occasional recreational taking of heroin and other drugs. There was no history of previous cardiac disease or syncope, or familial cardiac abnormality.

On admission systolic arterial pressure was 98 mmHg and pulse rate was 100/min, rectal temperature was 36.6°C. Examination showed a deeply comatose patient (Glasgow coma scale 4) without focal deficit, the pupils were equal in diameter, intermediate and reactive. Heart sounds were normal without murmur. Lungs were clear. Abdominal examination found dullness in the supra-pubic region. No pressure sore was observed. Electrocardiogram showed prominent coved ST-elevation displaying J-wave amplitude ≥ 2 mm in the right leads V1–V3, followed by negative T-wave with no isoelectric separation, associated with right incomplete bundle branch block evocative of type 1 Brugada ECG pattern [1].

Blood tests, performed before bladder catheterisation, showed creatinine 213 $\mu\text{mol/L}$, urea nitrogen 10.6 mmol/L, kalaemia 8 mmol/L, creatine kinase isoenzymes 19,477 U/L ($N < 195$) and calcium 1.83 mmol/L. The patient was immediately given 250 ml of 42% sodium bicarbonate intravenously. After evacuation of 700 ml of urine (urinary potassium concentration 99 mmol/L), kalaemia reached 5.9 mmol/L. Simultaneously, the ECG showed regression of the Brugada pattern (Fig. 1) which completely resolved in 24 h, and 48 h later creatininaemia was 69 $\mu\text{mol/L}$. A search for cyclic antidepressants was negative, a urine search for cocaine was slightly positive on admission and remained positive 4 days after. Transthoracic echocardiography was normal. The clinical course led to a persistent vegetative state due to severe anoxia.

The Brugada syndrome is defined as electrocardiographic abnormalities in right precordial leads, associated with syncope

and high risk of sudden death related to ventricular fibrillation [1, 2]. This syndrome is unrelated to ischemia, electrolyte disturbances, drugs or obvious structural heart disease. A Brugada electrocardiographic pattern can be observed with class IA and IC antiarrhythmic drugs or cyclic antidepressants. Hyperkalaemia can lead to ST-segment elevation in the right precordial leads and hyperkalaemia-associated Brugada pattern has been recently reported [3, 4].

Chlorpromazine, diazepam and lithium are not known to induce ST-segment elevation in the right precordial leads. Heroin can cause prolonged QT interval, prominent U-wave or bradyarrhythmia but the Brugada pattern has not been described [5]. Cocaine is an inducer of the Brugada pattern, however, the complete Brugada pattern disappearance while the urine search for cocaine remained positive argues against a prominent role of cocaine in this ECG aspect.

Simultaneous normalisation of ECG and kalaemia highly suggests that hyperkalaemia was the main cause of this transient pattern. This ECG pattern is more likely related to hyperkalaemia occurring with a healthy heart rather than a Brugada syndrome unmasked by transient hyperkalaemia. In the absence of a pharmacological test, the latter hypothesis, even if unlikely, cannot be ruled out and a facilitating action of cocaine also cannot be ruled out.

Physicians should be aware of this particular electrocardiographic pattern in patients with severe hyperkalaemia.

Fig 1 Electrocardiograms showing type 1 Brugada pattern on admission. Note the dynamic ECG change with type 2 Brugada

pattern on H+4. Arrows denote the J-wave. Calibrations are 10 mm/mv and 25 mm/s

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