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BRUGADA SYNDROME UNMASKED BY AMITRIPTYLINE

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Objectives: Brugada syndrome is a genetically determined sodium channel dysfunction characterised by ECG abnormalities in anterior chest leads V1-3 with normal structure of the heart. We report a patient who developed Brugada syndrome while possibly taking an overdose of amitriptyline and developed seizures, ventricular tachycardia and syncope.

Methods: A case report of a patient who was taking amitriptyline developed Type 1 Brugada Syndrome.

Results: A46-year-old man presented with a history of witnessed seizure, GCS 5 (response to painful stimuli only), cyanosis, agonal respiration and bradycardia (Day 0). Empty packets of unknown drugs were noted. He has a history of heavy alcohol use, hepatitis B and C and alcohol related seizure. He was managed with midazolam, calcium gluconate and sodium bicarbonate by the paramedics. In the Emergency Department, his GCS was 3, HR 128, BP 90/70 and has a lactic acidosis (lactate=14 mmol/L). Blood ethanol level was negative. ECG showed a broad complex tachycardia with a coved type ST segment elevation in anterior chest leads. He was managed with a further bolus dose of sodium bicarbonate with narrowing of the broad complex tachycardia, intubated and hyperventilated to pH 7.5. Urine drug screen was negative for amphetamine or cocaine but positive for opiates and benzodiazepines. He has intermittent jerking movement which was managed with midazolam bolus and infusion. Echocardiogram showed normal left and right ventricular function with abnormal septal motion likely related to bundle branch block. CT brain was normal. He developed short self-limiting episodes of ventricular tachycardia overnight. He was extubated next day with resolution of the ECG changes by the second day of admission.

He was commenced on 50 mg daily amitriptyline about 1 month before admission for the management of his peripheral neuropathy. He denied taking any overdose of medications. An amitriptyline level taken on the 3rd day of admission was 0.31 µmol/L (0.27-0.72) and its metabolite, nortriptyline has a

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concentration of 0.55 µmol/L (0.19-0.57) suggesting that amitriptyline concentration would have been much higher on admission. EEG performed on day 4 of admission showed no epileptiform activity.

Conclusion: This patient developed type 1 Brugada syndrome induced by amitriptyline which blocks the cardiac sodium channels and developed ventricular tachycardia and syncope. The possibility of amitriptyline overdose cannot be excluded.