A case of life-threatening wide QRS tachycardia related to flecainide toxicity

Flecainide acetate is a potent class Ic anti-arrhythmic drug with major sodium channel blocking agent. Flecainide acetate intoxication can be observed during therapy in patients with renal failure. Flecainide toxicity can cause myocardial impairment and precipitates circulatory collapse. We report a successful management of flecainide toxicity induced cardiac failure using extracorporeal membrane oxygenation (ECMO), hemoperfusion, slow low efficiency dialysis (SLED), maintaining alkaline acidity. A 72-year-old female had a history of paroxysmal atrial fibrillation (AF), end stage renal disease due to hypertensive nephrosclerosis and had received hemodialysis 3 times per week. She had been receiving flecainide 50mg per day for 2 years. She was admitted to emergency department because of dyspnea, chest discomfort with paroxysmal AF. Initial chest radiograph showed mild pulmonary edema. Treatment with conventional hemodialysis and volume control resulted in improvement of her chest radiograph and dyspnea and we considered discharge. Flecainide toxicity caused QT prolongation, wide QRS tachyarrhythmia and refractory shock, which did not respond to conventional treatment. She was intubated and treated with mechanical ventilator care, vasopressor, heparinization, ECMO, hemoperfusion, SLED. She was weaned from ECMO within 72 hours after its initiation and required continuous renal replacement therapy. EKG showed AF rhythm and heart rate was 70 to 90. After cardioversion 200J, EKG was converted to sinus rhythm. Sedative agents and inotropics were stopped, pulmonary congestion was improved. She was extubated and finally made a full recovery. She was discharged from the hospital without neurologic sequelae. This case of life-threatening flecainide intoxication in a female with renal failure underscores the importance of dose adjustment of flecainide acetate in a patient with renal failure and proper management of cardiovascular collapse with ECMO, hemoperfusion, maintaining alkaline acidity.

A case of cardiovascular beriberi with reversible pulmonary hypertension

Cardiovascular beriberi is caused by thiamine deficiency and usually presents a high cardiac output heart failure associated with predominantly right sided heart failure and refractory shock. We present a case of cardiovascular beriberi with severe pulmonary hypertension. A 50 years old man with chronic heavy alcoholics was referred to our department for dyspnea with mental change. On physical examination he presented blood pressure of 148/82 mmHg, respiratory rate of 26 breaths per minute, heart rate of 86 beats per minute. Initial electrocardiogram showed ventricular tachycardia and after administration of intravenous amiodarone, it changed to normal sinus rhythm with negative T waves in leads V1 to V3 and QT prolongation. Central venous pressure was elevated at 25 cm H2O. Laboratory test revealed renal and hepatic failure with hyperlactemia (lactic acid 9.7 mmol/L). Marked increased in pro-B type natriuretic peptide level was noted (23,251 pg/mL). Echocardiography showed marked right ventricular dilatation and flattening of the interventricular septum with a D-shaped deformation of the left ventricle. Left ventricular ejection fraction was 55% and calculated cardiac output was increased at 7.5 L/min. Moderate tricuspid regurgitation was found and estimated right ventricular systolic pressure was 62 mmHg. There was no evidence of pulmonary thromboembolism on contrast-enhanced chest computerized tomography. Cardiovascular beriberi (wet beriberi) is suspected and intravenous thiamine was administered. Within 1 week, his renal and hepatic function was normalized and mental status was improved. Follow up echocardiography showed improvement of right ventricular function and estimated right ventricular systolic pressure was decreased at 13 mmHg. Thiamine deficiency can cause reversible pulmonary hypertension, and high index of suspicion is needed for diagnosis.