Flecainide/warfarin/traditional-Chinese-medicine interaction

OXS

Various toxicities: case report

A 75-year-old woman developed various toxicities following concomitant administration of flecainide, warfarin and traditional-Chinese-medicine. This drug–herb interaction also caused warfarin overdose and a life-threatening pleomorphic arrhythmia associated with flecainide toxicity.

The woman was admitted to the emergency room because she had fainted several times. Her past medical history was significant for a mitral valve replacement with abi-leaflet prosthetic valve 3 years previously, which was anticoagulated with warfarin with constant INR between 2.5−3.5. She had been taking flecainide 100mg twice daily for the treatment of atrial extra systoles which was started 2 years prior to the admission. She reported that for the last 2 weeks, she had been taking a traditional-Chinese-medicine (concentrated Chinese herbal tea made of goji berries (GB)) for the prevention of COVID-19, drinking 1–2 glasses per day. Upon initial examination, she was unresponsive; her BP was 85/60mm Hg, with a respiratory rate of 20 rpm. Two days before, she had been complaining of dizziness, nausea and extreme fatigue. Cardiac auscultation showed a closing click at the apex, there were no audible murmurs, lungs were clear, and there was no oedema or other signs of congestion. An electrocardiogram (ECG) showed a very wide QRS complex tachycardia at a rate of 160 b.p.m.with a left bundle branch block (LBBB) morphology and northwest axis deviation, accompanied by periodic bursts and a cyclical increase in the QRS amplitude with repeated sequences of 5, 7 and 9 beats with apparent P-wave-like deflection before each burst. On the basis of her history and the ECG findings, flecainide toxicity was suspected.

The woman was treated with sodium bicarbonate, amiodarone and magnesium sulfate. This resulted in a 140b.p.m. regular monomorphic wide QRS tachycardia. As her pulse remained feeble, a 200J electrical cardioversion was delivered and resulted in sinus rhythm with immediate onset of an atypical atrial flutter with a very wide P wave, LBBB morphology, and prolonged QTc interval and improvement in consciousness and haemodynamic status. Laboratory findings were remarkable: AST 152 U/L, ALT 92 U/L, lactate 797 U/L, troponin 57.7 ng/mL, prothrombin time 77.4s, partial thromboplastin time 46.7s, and INR 7.18. In view of elevated INR, vitamin K was administrated. An echocardiogram showed normal left ventricular systolic function without segmental wall motion abnormalities, mild pulmonary hypertension and left ventricular hypertrophy. Mechanical valve revealed no abnormalities. After 8h, her mental status improved. Control laboratories showed an INR of two. A repeated ECG revealed an atypical flutter with narrow QRS associated with slow repolarization, QTc 530 ms and asymmetric T-wave inversion in the anterior wall. She was started on enoxaparin sodium and was later bridged to warfarin. She was discharged home 48h after admission with amiodarone and warfarin. A pre-discharge ECG revealed sinus rhythm at a rate of 87 b.p.m., atrial extrasystoles, and resolution of all morphological patterns associated with flecainide toxicity. One week after discharge, she visited to the outpatient cardiology clinic with a new INR determination for monitoring and adjustment of warfarin dose. Finally, it was determined that the flecainide toxicity developed secondary to CYP2D6 inhibition by GB with the consequent increase in serum levels of flecainide" "The increase in INR secondary to warfarin overdose was also associated with inhibition of the CYP2C9 by GB.

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