Biventricular tachycardia induced by herbal medicine containing ephedra sinica

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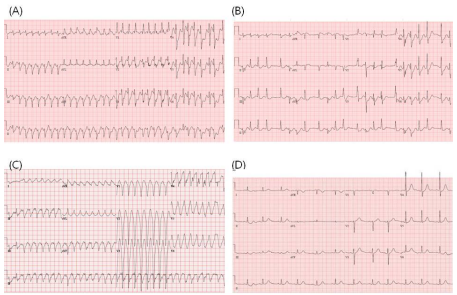
Ephedra is a potent sympathomimetic agent with cardiovascular effects including tachyardia, positive inotropy, arterial vasoconstriction and hypertension. We report 1 case of biventricular tachycardia after taking herbal medicine containing ephedra sinica. A 77-year-old male with a 30-year-history of COPD prsented with acute onset chest discomfort and dyspnea. He was precribed herbal medicines containing ephedra sinica for abdominal discomfort 2 days ago. Initial electrocardiography (ECG, Fig. A) showed hemodynamically unstable monomorphic ventricular tachycardia with RBBB pattern in percordial leads, which was terminated by electrical cardioversion. ECG after cardioversion, atrial fibrillation was observed (Fig. B). About 1 hour later, hemodynamically unstable fast monomorphic ventricular tachycardia with LBBB pattern was spontaneously induced (Fig. C). Because of hemodynamic instability, additional electrical cardioversion was performed and sinus rhythm was restored (Fig.D). Electrolytes and arterial blood gas analyses were normal. To rule out structural heart disease, echocardiography was done and it revealed normal ejection fraction and no significant abnormalities. Coronary angiography showed a 80% stenosis in the proximal circumflex coronary artery and a 70% stenosis in the posterior descending artery. Percutanous coronary intervention was done to left circumflex artery. The patient was discharged on clopidogrel, warfarin, ACE inhibitor, beta-blocker, statin. His follow up was uneventful.



A case of Non-ST elevation myocardial infarction caused by critical subclavian artery stenosis

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We presented a case of a 49-year-old female patient with end stage renal disease (ESRD) from diabetes mellitus on peritoneal dialysis who complained of resting chest pain and left arm pain after CABG surgery using a left internal thorasic artery. The initial electrocardiography showed a new ST segment depression on lateral leads (V4-6) and serum troponin I level was elevated (19.75 ng/mL), consistent with non-ST segment elevation myocardial infarction involving lateral wall. Brachial systolic blood pressure measured in the left arm was about 30 mmHg lower than that measured in the right arm. Urgent coronary angiography and bypass graft angiography did not reveal any critical stenosis in CABGs connected with LAD or OM. However, aortic arch angiography showed subtotal occlusion at the ostium of the left subclavian artery with sluggish flow into the LITA. Because the significant left subclavian artery stenosis was not demonstrated during preoperative evaluation, non ST elevated myocardial infarction was caused by de novo left subtotal subclavian artery stenosis proximal to the LITA. ESRD and poor glycemic control might cause rapid progression of native left subclavian artery stenosis. After subclavian artery intervention, her chest pain and arm pain subsided and ST depression was improved. Blood pressure measured in both arms also became approximated (right arm 130/70 mmHg, left arm 128/70 mmHg). Short-term echocardiography follow-up after 2 weeks showed significant improvement of left ventricular wall motion and systolic function with ejection fraction of 40%. In conclusion, careful physical evaluation including blood pressure measurement in both arms and meticulous evaluation of overall pathway from the aorta to the CABGs cannot be over-empathized for the patients who had undergone CABG surgery in order to render the clues for uncommon and unexpected culprit lesion beyond CABGs.