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Case Report

Successful treatment of prolonged cardiopulmonary arrest of Kounis syndrome during coronary angioplasty

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ABSTRACT

We experienced a case of Kounis syndrome with cardiopulmonary arrest and severe coronary spasm. A 70-year-old man with cardiac pacemaker and chronic dialysis was treated for angina pectoris of the right coronary artery. After diagnostic coronary angiography of the right coronary artery, optical coherence tomography was performed with contrast medium and low-molecular-weight dextran. The patient's blood pressure unexpectedly dropped to 40 mmHg and erythema of the breast was noted. Electrocardiogram showed remarkable ST elevation in II, III, aVF leads. Coronary angiography showed total occlusion of the proximal right coronary artery. Although intracoronary infusion of sodium nitrate did not dilate the coronary artery promptly, coronary balloon angioplasty recovered the artery flow. Since severe anaphylaxis-related shock was contemplated, methyl prednisolone and epinephrine were administered intravenously. We could not introduce percutaneous cardiopulmonary support due to kinking of the vein. After 1 hour of cardiopulmonary resuscitation with frequent ventricular fibrillation and direct current shock, the sinus rhythm and blood pressure recovered. Following 2 months of intensive care treatment for other complications, including infection, the patient was discharged from hospital without any residual disability.

<**Learning objective:** An anaphylactic reaction is one of the causes of sudden deterioration of a patient's condition observed during interventional procedures. Kounis syndrome is a rare and not yet well known important concept that deals with the reaction. Therefore, we report a severe case of Kounis syndrome with cardiopulmonary arrest.

Introduction

Kounis syndrome is an anaphylactic reaction that causes either coronary spasm (type I) or acute coronary syndrome due to plaque rupture or coronary erosion (type II) [1,2]. Usually a skin reaction and reduced blood pressure are accompanying symptoms. We report a case with an abrupt and prolonged cardiac arrest due to severe coronary spasm and reduction in blood pressure by vessel dilatation of the whole body after intracoronary infusion of low-molecular-weight dextran.

Case report

A 70-year-old man with cardiac pacemaker was admitted to our hospital for percutaneous coronary intervention (PCI) of the right coronary artery (RCA).

He was admitted to the hospital 1 month before the present admission with dyspnea and diagnosed with complete atrioventricular block with a heart rate of 33/min. He had diabetes mellitus and hypertension and was administered several drugs. Cardiac echocardiography showed no wall motion abnormality with ejection fraction of 83% as well as abnormal relaxation (E/A ratio 0.79) and mildly elevated end-diastolic pressure (E/E′ 15.8) and pulmonary systolic pressure (48 mmHg). Before DDD pacemaker implantation, coronary angiography was performed and 75% diameter stenosis at the distal RCA was detected. No allergic reaction was observed after coronary angiography (CAG). He

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gradually recovered from heart failure after DDD pacemaker implantation and was discharged from hospital 1 month prior to the present admission. His prescription medications were azilsartan 20 mg, amurodipine 5 mg, glimepiride 1 mg, linagliptin 5 mg, and metformin 500 mg per day. He showed no abnormal finding on physical examination. Blood pressure was 138/64 mmHg and the heart rate was 86/min. The electrocardiogram (ECG) showed pacemaker rhythm (A sensing and V pacing, Fig. 1A). The white blood cell count was 5610/μL. The red blood cell count was 454×10^4/μL. Platelet count was 16.2×10^4/μL. The proportion of eosinophils was 2.6% and was less than 2% during hospitalization. IgE level was not measured. Blood urea nitrogen level was 26.9 mg/dL and serum creatinine level was 0.77 mg/dL. Na, K, and Cl levels were 142, 4.4 and 107 mEq/L, respectively. Blood glucose level was 158 mg/dL and hemoglobin A1c was 7.9% indicating poor diabetic control. Other blood data showed no abnormalities. Chest radiography showed cardiothoracic ratio of 46%. On the second day, PCI was performed. A 6Fr Launcher SAL1.5SH (Medtronic, Inc., Minneapolis, MN, USA) guiding catheter was engaged for the RCA. CAG of the RCA was performed once in the left anterior oblique position (Fig. 2A). A guide wire was introduced into the RCA and optical coherence tomography (OCT, St Jude Medical, Inc., St Paul, MN, USA) was performed. A mixture of contrast media and low-molecular weight-dextran (1:1, 18 mL) was injected into the RCA for removal of blood. Suddenly, the patient complained of bilateral arm pain and dyspnea. Systolic blood pressure dropped to 40 mmHg. Broad redness on the surface of his breast was observed. ECG showed remarkable ST elevation at II, III, aVF leads and ST depression at V1-4 leads (Fig. 1B). His consciousness became Japan coma scale 300 and breathing stopped. Subsequently, cardiopulmonary resuscitation was performed. Anaphylactic shock was assumed and 125 mg methyl prednisolone was injected intravenously and 0.5 mg epinephrine was injected intramuscularly followed by intubation. CAG showed total occlusion at the proximal RCA (Fig. 2B). We did not perform CAG of the left coronary artery because of cardiopulmonary arrest and risk of contrast medium allergy. Since intracoronary infusion of 2.5 mg isosorbide dinitrate did not dilate the coronary artery promptly, a percutaneous coronary balloon angioplasty was performed with a 3-mm balloon catheter (Fig. 2C). After the procedure, both of the RCA coronary flow and the ST elevation at II, III, and aVF were regained (Fig. 2D). However, cardiac arrest continued and 1 mg epinephrine was administered intravenously twice with a 4-min interval. Repetitive ventricular fibrillation occurred and biphasic direct current (DC) shocks with 150 J were performed (Fig. 1C). We could not introduce percutaneous cardiopulmonary support (PCPS) due to kinking of the left iliac vein. Moreover, we did not introduce intra-aortic balloon pump (IABP) because of prolonged introduction of PCPS. After 1-hour cardiopulmonary resuscitation with frequent ventricular fibrillation and DC shocks, a sinus rhythm and systolic blood pressure of 100 mmHg was recovered (Fig. 1D). OCT showed a partial lipid-rich plaque at the spastic site and fibrous plaques at other sites (Fig. 3). Tryptase on the day of PCI was 39.1 μg/L and the next day was 23.8 μg/L (normal range 2.6–9 μg/L) (Fig. 4). Kounis syndrome was diagnosed. Troponin T level was 4.99 ng/mL ($N < 0.014$) on day 3. Maximal creatine kinase (CK) level was 20,230 IU/L and maximal CK-MB level was 227 IU/L on day 4. There was a large discrepancy between these values mainly because of vigorous chest compression and electrical defibrillation. The causal antigen was considered as either low molecular dextran or contrast media. The drug-induced lymphocyte stimulation test (DLST) was negative for either agent and for xylocaine. The patient was complicated by bacteremia and was treated with antibiotics. After intensive care for treatment of the infection and cardiac

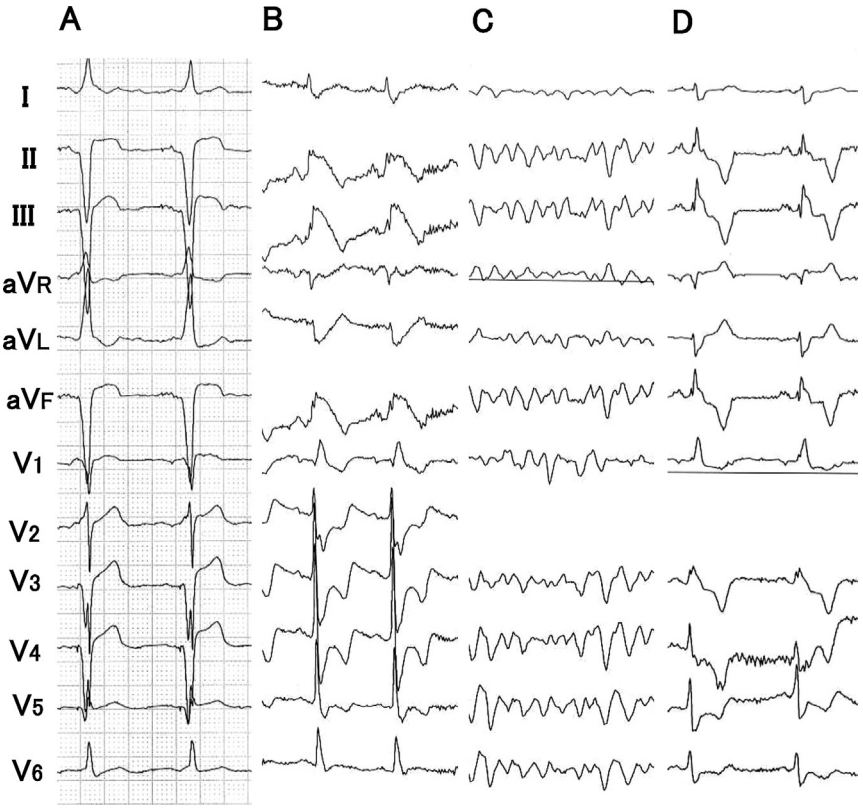

Fig. 1. Electrocardiogram during percutaneous coronary intervention. (A) Before coronary angiography. (B) Occurrence of ST change at II, III, aVF, and V1-4 leads. (C) Ventricular fibrillation. (D) Recovery to sinus rhythm.