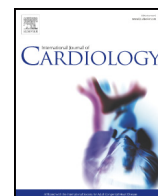




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Letter to the Editor

Kounis syndrome caused by aspirin-induced asthma

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Introduction. Kounis syndrome is known as an acute coronary syndrome such as coronary spasm, acute myocardial infarction, and stent thrombosis caused by allergy or hypersensitivity, as well as anaphylactic or anaphylactoid reactions, to various allergens and several conditions [1,2]. For example, causes of Kounis syndrome are drugs (antibiotics, analgesics, corticosteroids, anticoagulants), various conditions (bronchial asthma, urticarial, food allergy), and environmental exposures [3]. Herein, we report a case of Kounis syndrome secondary to asthma induced by aspirin intake which was intended to treat angina pectoris.

Case report. A 51-year-old male had an allergy to aspirin and a history of asthma, and was treated with beta2 agonists and inhaled corticosteroids. He had gone to another hospital due to chest pain, and had received aspirin because of the suspected coronal atherosclerosis change on the CT. Several days later, he was admitted to our hospital with chest compression and severe dyspnea 20 min after taking 100 mg of aspirin. His physical examination revealed acute respiratory failure as follows, the heart rate was 93 per minute, the blood pressure was 117/58 mm Hg, the respiratory rate was 45 breaths per minute, and the oxygen saturation was 98% under 10 L/min supplemental oxygen. His chest examination showed diffuse wheezing, and chest X ray indicated no remarkable sign of heart failure. His electrocardiogram showed ST-segment elevation in leads III and aVF with ST-depression in leads I, aVL and V1–5 (Fig. 1). Laboratory studies showed WBC count 12,000/mm³ (reference: 4000–8000), an absolute eosinophil count 708/mm³ (reference: 70–450) CK 288 IU/L (reference: 35–170), CK-MB fraction 26 IU/L (reference: 0–25). After the administration

of sublingual nitroglycerin and intravenous corticosteroids (100 mg), his complaint improved with the resolution of electrocardiographic abnormalities, so an urgent coronary angiography was not performed. He was treated in an intensive care unit, and received continuous intravenous nitroglycerine and heparin. However, his respiratory condition worsened, and he required mechanical respiratory support. At that time, his CK (1866 IU/L) and CK-MB fraction (148 IU/L) levels were elevated, and transthoracic echocardiography revealed asynergy in posterior and inferior segments at the basal level, so a coronary angiography was performed. It revealed no organic lesion in coronary arteries, and left ventriculography revealed severe hypokinesia of inferior segment with an ejection fraction of 42.8%.

He was suspected to have coronary spasm secondary to aspirin. He was treated with continuous intravenous nicorandil, intravenous theophylline and dexamethasone under mechanical ventilation. At first, his peak air way pressure was over 40 cm H₂O due to asthma attacks, but it gradually improved, and extubation was done on the fourth day. 123I-beta-methyl-iodophenyl-pentadecanoic acid (BMIPP) and 201 thallium (TI) dual myocardial SPECT indicated a mismatch in inferior segments, on the 7th day (Fig. 2).

He was discharged from hospital with nicorandil, benidipine, theophylline, leukotriene receptor antagonist, and inhaled corticosteroids. After discharge, he had not had angina pectoris and asthma attacks.

Discussion. Kounis syndrome is characterized by the concurrence of acute coronary syndrome with the activation of inflammatory mediators such as histamine, neutral protease, arachidonic acid products, platelet activating factor and a variety of cytokines and chemokines [1–3]. Our patient was diagnosed with coronary spasm triggered by aspirin because of elevated cardiac enzyme and ST segment change in ECG at the event without coronary organic lesions.

Aspirin-induced-asthma (AIA) is a distinct clinical syndrome, and the Samter's triad (asthma, aspirin sensitivity, and nasal polyp) is well-known [4]. It usually starts with chronic rhinitis with the development of nasal polyps, and its hallmark is precipitation of asthma and rhinitis by aspirin or other non-steroidal anti-inflammatory drugs (NSAIDs) [5,6]. In our patient, the diagnosis of AIA could be made because of the history of acute asthma attack after taking aspirin.

He developed dyspnea again 6 h after the arrival, when 100 mg of hydrocortisone sodium succinate was administered intravenously. Corticosteroid allergy is more commonly seen in asthmatics with a history of aspirin allergy, and hydrocortisone, prednisone, and methylprednisolone are often reported as the causes [7], so we should have administered other kinds of corticosteroids. Indeed, asthma attack was

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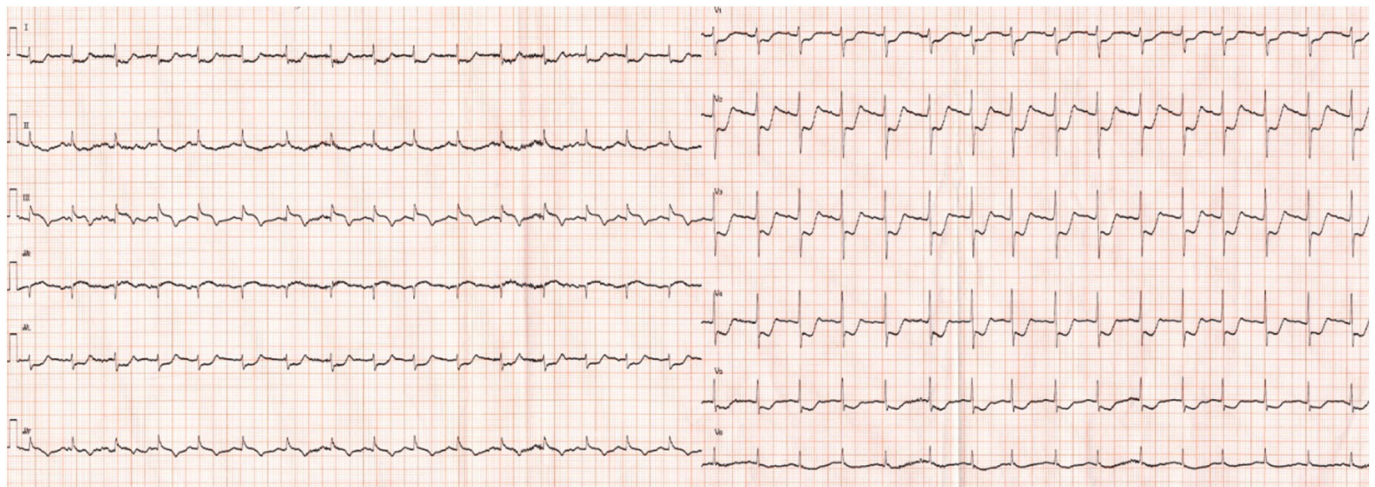


Fig. 1. The initial ECG at presentation showing ST elevation in leads II, III, and aVF, and ST depression in leads I, aVL, and V1–V4.

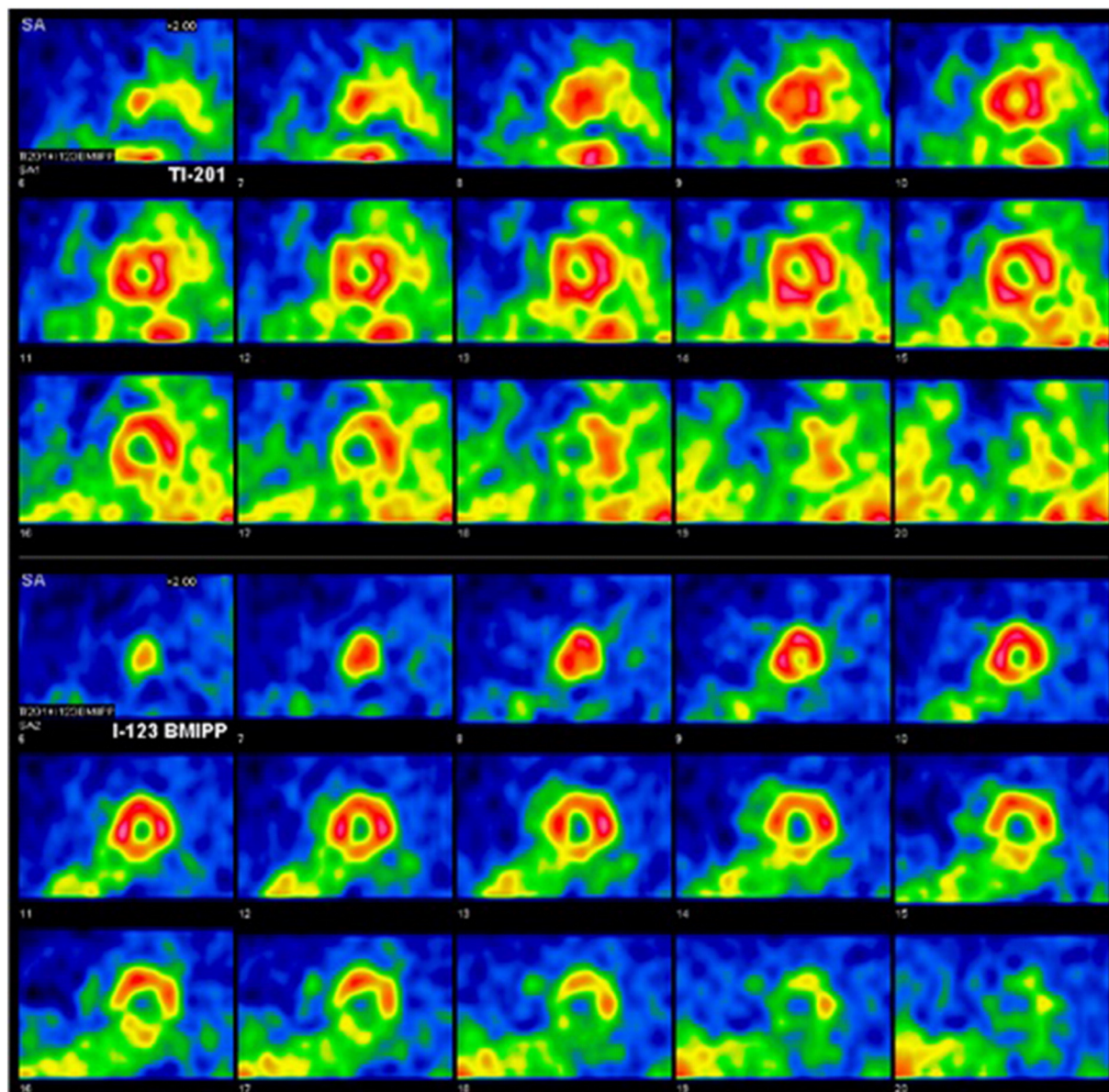


Fig. 2. BMIPP-TI dual myocardial SPECT indicating an inferior myocardial infarction.

significantly suppressed after the intravenous dexamethasone in our case, which suggested that the coronary spasm was induced by aspirin allergy. The treatment is very challenging, since it needs to consider both cardiac and allergic syndromes simultaneously [8]. In general, for the management of cardiac syndromes, aspirin, nitroglycerin, heparin, beta-blockers, and calcium-channel blockers are recommended. For the management of allergic syndrome, H-1 blockers, H-2 blockers, corticosteroids, epinephrine, and mast cell stabilizers are recommended.

Reports of coronary spasm cases induced by AIA are few. When patients are suspected as acute coronary syndrome, aspirin will be given soon, though aspirin could be dangerous sometimes like in this case. It is sure aspirin is needed to ACS, but there exists some cases of aspirin-induced allergic reactions. It doesn't happen so often, but we should keep in mind this, and should check patients' past medical histories and allergies. Kounis syndrome is really unique and is often under-diagnosed, so it should be kept in mind.

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The authors of this manuscript have certificated that they comply with the Principles of Ethical Publishing in the International Journal of Cardiology.

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