

Acute myocardial infarction induced by axitinib

To the Editor,

Axitinib is a novel tyrosine kinase inhibitor which is a second-line option for the treatment of metastatic renal cell carcinoma with progression after previous therapy (1, 2). We present the first reported case of acute myocardial infarction in a patient receiving axitinib.

In July 2010, a 40-year-old male with no history of smoking, hypertension, diabetes or hypercholesterolemia, and no family history of coronary artery disease, underwent right nephrectomy due to renal cell carcinoma. Chest computed tomography, at the time of diagnosis, revealed the presence of multiple nodules in both lung areas, the largest of which was in the right middle lobe measuring 1.2 cm. Pathologic examination of a transbronchial lung biopsy showed metastatic clear-cell type renal cell carcinoma. Abdominal magnetic resonance imaging detected no metastatic lesion. Normal bone scan was observed in technetium-99m methylene diphosphonate scintigraphy. Whole-body fluorodeoxyglucose positron emission tomography imaging exhibited increased uptake in proven metastatic pulmonary lesions while the rest of the body showed physiological distribution. Transthoracic echocardiography documented normal left ventricular systolic and diastolic function, and normal valvular structures. Adjuvant systemic therapy was initiated to treat residual metastatic disease. After the failure of three consecutive chemotherapeutic agents (interferon-alpha for 3 months, everolimus for 2 years, sunitinib for 1 year, consecutively), treatment with oral axitinib was started at Ordu State Hospital, in November 2013. One week after beginning axitinib, he developed chest pain with sudden onset. The electrocardiogram (ECG), which was recorded during chest pain, demonstrated ST segment elevation in leads II, III, aVF and V3 to V6, reciprocal ST depressions in lead I, aVL, and third-degree atrioventricular block. On physical examination, there were no abnormal findings. The patient was diagnosed with acute myocardial infarction of inferolateral wall, and transthoracic echocardiography showed mildly hypokinetic myocardium (involving the right coronary artery territory), with an estimated left ventricular ejection fraction of 55%. After pretreatment with clopidogrel (600 mg of oral loading dose), aspirin (300 mg, oral) and heparin (10000 U, intravenous), he was immediately transferred to the catheter laboratory for a primary percutaneous coronary intervention. Coronary angiography revealed that the right coronary artery (RCA) was totally occluded by a thrombus in the proximal segment, while the left main, the left anterior descending and the circumflex artery showed no significant stenosis. After successful wire crossing in the RCA, the totally occluded lesion was pre-dilated with a 2.5 x 15 mm balloon at 10 atms. Subsequently, 3.0 x 20 mm bare-metal stent was implanted at 15 atms and thrombolysis in myocardial infarction (TIMI) 3 flow was achieved. The patient's symptoms were relieved, and ST elevations on ECG regressed. A week after the procedure, he was discharged from the hospital in a stable condition, with the prescription of clopidogrel 75 mg, aspirin 300 mg, metoprolol 25 mg and atorvastatin 10 mg (all once a day). Axitinib was discontinued immediately after the diagnosis of myocardial infarction and the patient was referred to oncology department of our hospital following discharge, for the arrangement of his chemotherapy drugs.

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Peripartum cardiomyopathy associated with triplet pregnancy

To the Editor,

A 22-year-old puerpera after c/section for a triplet at thirty sixth week of gestation was admitted to obstetric clinic in our institution. Pregnancy record for his triplet did not show any problem. After an elective cesarean section under epidural anesthesia, three healthy babies were born. Before pregnancy there was no documented cardiac disease. Progressive dyspnea and orthopnea began within two hours following birth. Arterial blood pressure was 100/60 mm Hg and heart rate was 110/ min. On auscultation S3 heart sound was heard. Arterial blood gas analysis revealed, O₂ saturation: 87%, PaO₂: 85 mm Hg, PaCO₂: 40 mm Hg, pH: 7.30. Lung examination showed bilateral crackles reaching to upper middle zone. Consequently the patient was transferred to the coronary intensive care unit with the diagnosis of an acute pulmonary edema. Intravenous nitroglycerin, furosemide and continuous positive airway pressure (CPAP) therapy decreased the shortness of breath. On transthoracic echocardiography, left ventricular ejection fraction (LVEF) was 23% and left ventricular diastolic diameter (LVDD) was 61 mm. We considered peripartum cardiomyopathy (PPCMP) as an etiology of acute pulmonary edema. 25 mg of metoprolol, 25 mg of sprinolactone, 40 mg of furosemide and 2.5 mg of ramipril, all with oral use, were added to the daily treatment of the patient after positive response to the acute treatment. Patient followed up 2 weeks in our intensive care unit and orthopnea gradually decreased. LVEF was measured 25% at the time of discharge. 1 month after discharge, LVEF was 30% with NYHA class 1 functional status. 2 months later the LVEF was advanced to the 45% while LVDD regressed to 55 mm. However, sixth month control echocardiography demonstrated LVEF of 60% and LVDD of 49 mm and that the patient was symptom free. Twelfth month control was also showed normal left ventricular systolic function.

Classical risk factors of PPCMP include race (African origin), advanced maternal age, twin pregnancy, preeclampsia, hypertension, infections and long-standing tocolytic therapy (1, 2). Although twin pregnancy is a known risk factor, PPCMP associated with triplet pregnancy has not been described previously in the literature. The pathogenesis of