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 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. Adjutant systemic therapy was initiated to treat residual metastatic disease. After the failure of three consecutive chemotherapeutic agents (interpheron-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 documented normal left ventricular systolic and diastolic function, and normal valvular structures. A week after the procedure, he was discharged home. 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 chemotherapeutic drugs.

Emre Gürel, Zeki Yüksel Günaydın1, Müge Karaoğlanoğlu*, Tuncay Kirş
Departments of Cardiology and *Oncology, Ordu State Hospital; Ordu-Turkey
1Department of Cardiology, Ordu University Hospital; Ordu-Turkey

References

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 crepitations reaching to upper middle zone. Consequently the patient was transferred to the coronary intensive care unit with the diagnosis of an acute pulmonary edema. Invasive 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 ethiology of acute pulmonary edema. 25 mg of metoprolol, 25 mg of spironolactone, 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