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DOI:10.14744/AnatolJCardiol.2016.7389

Author`s Reply

To the Editor,

We are much pleased with the authors' interest in our article entitled "SYNTAX score predicts postoperative atrial fibrillation in patients undergoing on-pump isolated coronary artery bypass grafting surgery" (1), as published ahead of print for the Anatol J Cardiol 2015 Nov 18, and we would like to thank them for their contributions.

Firstly, definitive diagnosis of postoperative atrial fibrillation (PoAF) is not found in the relevant guidelines. In our study, PoAF was defined as it has been in previous studies (2). In the literature there are many controversial definitions of PoAF (3, 4). In our study, patients were followed with continuous telemetry for between 72 and 96 hours. A 12-lead electrocardiography (ECG) was obtained from the patients every 12 hours or 24 hours at the intensive care and in-patient units, respectively. Rhythm monitoring was continued until patients were discharged from the hospital. If patients had complaints such as dyspnea, palpitation, or angina, 12-lead ECG was taken during hospitalization. Incidence of PoAF could increase beyond the 72 to 96-hour window observed with continuous telemetry. The rate of PoAF may be underestimated in our study.

Drug use, including beta blockers, renin angiotensin aldosterone blockers, and statins before surgery could affect incidence of PoAF. In our study, percentage of beta blocker, angiotensin-converting enzyme inhibitor, and angiotensin receptor blocker use was 100% and 98.9%, respectively. There was no difference in drug use between the 2 groups. Obstructive sleep apnea and obesity were not included in our study as independent parameters because of low number of instances.

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Kounis syndrome presenting with acute inferior wall myocardial infarction and cardiogenic shock secondary to intravenous ampicillin/sulbactam administration

To the Editor,

Kounis syndrome (KS) is induced by allergic and anaphylactic reaction, and is considered a rare cause of coronary artery spasm (1) A 44-year-old male patient was admitted to our center with complaint of severe chest pain lasting for 1 hour. He was administered treatment of 1 g intravenous ampicillin/sulbactam with diagnosis of upper respiratory tract infection. He did not have history of allergy or traditional risk factors for coronary artery disease. Ten minutes after the injection, he felt severe, squeezing retrosternal chest pain. On physical examination, he was pale. He did not have pruritus or rash. His blood pressure (BP) and heart rate were 77/48 mm Hg and 104 bpm, respectively.

Electrocardiogram (ECG) showed ST segment elevation in leads D2, 3, aVF, V4R, V5R, and V6R. Echocardiogram revealed moderate reduction in left ventricular ejection fraction (45%), inferior and inferoseptal wall hypokinesia, and right ventricular systolic dysfunction. Coronary angiography (CAG) revealed diffuse constriction of left anterior descending artery and left circumflex artery, and total occlusion of right coronary artery. All constrictions were considered coronary artery spasm, and despite deep hypotension, intracoronary nitroglycerin was administered to confirm spasms. Subsequently, BP rose to 108/73 mm Hg. By this time, chest pain was alleviated and follow-up CAG showed complete normalization of coronary artery spasm. Combination treatment of intravenous 25 mg prednisolone, 50 mg ranitidine, and 50 mg diphenhydramine was administered with diagnosis of KS. He felt better and chest pain was completely resolved over the next 15 minutes. Second ECG showed resolution of ST segment elevation. Isosorbide-5-mononitrate and ciprofloxacin 400 mg once a day were administered to manage KS. Patient's eosinophil count was in normal range (120/μL), and cardiac biomarkers were elevated as follows: troponin I: 3 ng/dL, creatine kinase-MB: 52 U/L. Additionally, serum tryptase level was elevated at 146 ng/mL (reference range: <11.4 ng/mL). Patient was observed closely over the next 2 days and did not develop further chest pain or complication. There are 3 types of KS; type I variant is described as coronary artery spasm in patients with normal coronary arteries without traditional risk. In these patients, coronary artery spasm is triggered by acute release of inflammatory mediators (1–3). Although cardiac marker elevation is not expected in type I variant, our patient suffered severe myocardial infarction due to crucial diffuse coronary artery spasm. Ampicillin/sulbactam-induced KS is very rarely reported. First intravenous antibiotic administration resulted in catastrophic complication in our patient. Although adrenalin is a traditional drug used for anaphylactic shock, it causes both coronary vasodilatation and myocardial oxygen demand by direct inotropic and chronotropic effects (4). Due to serious side effects of adrenalin, we administered antihistaminic and corticosteroid combination. In selected patients, intracoronary nitroglycerine may be used to reverse vasospasm. In patients with coronary vasospasm related to allergic reaction, treatment with vasodilators such as nitrates and calcium channel inhibitors is choice of treatment for case of coronary vasospasm (5).

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DOI:10.14744/AnatolJCardiol.2016.7335

Renal artery stenting of chronic kidney disease patient with resistant hypertension

To the Editor,

Resistant hypertension is a clinical entity presenting with uncontrolled blood pressure (BP) despite use of 3 or more antihypertensive drugs, including diuretic. Reno vascular hypertension related to renal artery stenosis (RAS) occurs in etiology of hypertension and affects up to 5% of all hypertensive patients (1). Fifty-five-year-old male patient was admitted to our clinic with uncontrolled BP. He was using several antihypertensive drugs, including diuretic. He had history of chronic kidney disease and untreated renal artery stenosis. He had residual amount of 500 mL daily urine output. We wanted to evaluate his residual renal function with diuretic administration. We increased daily urine output up to 1500 mL with furosemide and this encouraged us to pursue renal artery intervention. Renal angiography revealed moderate stenosis of right renal artery and severe stenosis of left renal artery. We implanted a 4.0x15 mm bare metal stent in left renal artery. BP responded immediately after intervention and we were able to discontinue antihypertensive drugs. Daily urine output increased up to 1000 mL without diuretic. Hemodialysis sessions were decreased to 2 days per week.

RAS primarily causes significant reduction in renal blood flow and is notable factor in development of progressive kidney failure. Atherosclerotic RAS patients present with persistent and progressive reduction in glomerular filtration rate, treatment resistant severe hypertension, and recurrent episodes of flash pulmonary edema. Pathogenesis of chronic kidney disease progression due to RAS is assumed to be more complex than just arterial narrowing. Different cytokines and chemokines related to stimulation of