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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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

renin-angiotensin-aldosterone system (RAAS) may play a role in further development of renal ischemia (2). Recent studies have indicated that medical treatment should be mainstream choice for management of RAS patients. No difference was found in renal or cardiovascular adverse events between "medical therapy and renal artery stenting" and "medical therapy alone" groups in Cardiovascular Outcomes in Renal Atherosclerotic Lesions study; however, this study investigated medically under control, normotensive renal artery stenosis patients (3). Indications for renal artery stenosis intervention in chronic kidney failure in cases of uncontrolled resistant hypertension are debatable. Resistant hypertension is a commonly seen problem in chronic kidney disease patients and cardiovascular outcomes of these patients are poor. Residual kidney mass may be source of RAAS stimulation and chemokine release. Bilateral nephrectomy is best known way to control resistant hypertension and to decrease adverse cardiovascular event rates (4). Nephrectomy is well-known choice of treatment for resistant hypertension in chronic kidney disease; however, this is surgical procedure with its own risks related to operation. We thought that if potential of residual kidney tissue could be evaluated it would clear out the benefit of renal artery revascularization (5).

Resistant hypertension is a problematic clinical entity closely related to poor cardiovascular outcomes in chronic kidney disease patients. Renal artery stenting can be a good choice instead of bilateral nephrectomy in selected patients.

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Medication adherence and quality of life in coronary artery bypass grafting patients, results of retrospective cohort study

To the Editor,

Studies have shown that medication non-adherence is related to greater morbidity and mortality in chronic disease, including coronary heart disease patients (1). In addition, patients who experience impaired quality of life (QOL) have reported low medication adherence. The purpose of the present study was to evaluate relationship between QOL and patient symptoms and compliance. This is a retrospective cohort study of 196 patients who underwent coronary artery bypass grafting (CABG) 5 years prior. Medication and follow-up visit adherence, post-CABG symptoms and events, and QOL were assessed using study checklist and 36-item health related QOL questionnaire. Five-year survival rate of discharged patients was 87% (SE: 0.032). Kaplan-Meier survival curves did not show difference between men and women (men: 89%, women: 82%; p=0.3). Frequency of rehospitalization for cardiac reasons, re-angiogram, and percutaneous coronary intervention in CABG cohort during 5-year period was 18.8%, 7.3%, and 3.1%, respectively. Medication and follow-up visit non-adherence rates were 10.7% and 51.5%, respectively. Logistic regression analysis showed compliance with follow-up visits in patients with chest pain, dyspnea on exertion, and New York Heart Association (NYHA) Functional Classification III/IV were increased 1.7, 1.8, 1.5 times compared to those without symptoms ($p<0.05$). Mean score of physical and mental components were statistically different in patients with and without symptoms ($p<0.05$). Linear regression analysis after adjustment for age and sex indicated lower QOL was related to more symptoms. Physical and mental components of QOL were negatively associated with medication (B:-0.18, p:0.04; B:-0.29, p:0.02, respectively) and follow-up visit observance (B:-0.3, p:0.01; B:-0.3, p: 0.01, respectively).

QOL scores in physical and mental components among our study population were equivalent to general elderly population (2). Chest pain, dyspnea, or poor NYHA classification was trigger for seeing doctor, greater medication adherence, and worse QOL. Perhaps taking large number of pills or doses per day may influence QOL, especially mental component. Angina and dyspnea can cause activity limitation and thereby decrease level of QOL. Also, more reported medication and follow-up visit adherence were related to lower QOL score. According to systematic review of chronic obstructive pulmonary disorder patients, increased QOL may trigger medication non-compliance (3). Studies like that of Loopen et al. (4) have shown patient QOL was improved immediately after sur-