ment of endothelial function, or direct protection of the myocardium also plays a role in extracellular matrix modulation (3, 6). These effects may contribute to the prevention of AF of statins. Therefore, we suggest that routine administration of statins is useful in patients undergoing elective CABG for prevention of postoperative AF. In our study, CRP levels were significantly lower in patients without AF versus those with AF. CRP levels on the 14th postoperative day were significantly lower in the statin group compared with those in the non-statin group.

Atorvastatin undergoes rapid absorption when taken orally, with an approximate time to reach maximum plasma concentration (Tmax) of 1-2 h. The absolute bioavailability of the drug is approximately 14%; however, the systemic availability for HMG-CoA reductase activity is approximately 30% (7). Thus, based on pharmacokinetics, the drug should be active and effective during the first postoperative day. Each extubated patient was given 40 mg of atorvastatin per day, which was started on an average of 6 h after the operation. All patients are able to take oral statin. Therefore, we suggest that if preoperative statin therapy is not administered to patients, statin therapy should be started in a short time postoperatively to obtain the beneficial effects of statins.

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Frequently occurring Torsades de pointes attacks in an old patient on solifenacin therapy and management strategy

To the Editor,

The most frequent reason for the acquired long QT syndrome and associated Torsades de pointes (TdP) is drugs. Solifenacin, an antimuscarinic drug, causes QT prolongation by decreasing the activity of potassium channels in phase 3 of the action potential (1). In these patients, temporary pacemaker (PM) implantation is a life-saving therapeutic approach (2).

An 84-year-old male patient was admitted to the emergency department with complaints of short-term episodes of loss of consciousness and cyanosis in the hands. The patient has been on treatment with 10 mg/day solifenacin for 15 days because of the urinary incontinence. In addition, he has been taking metformin (2000 mg/day) and atorvastatin (10 mg/day) for type 2 diabetes mellitus and hyperlipidemia. The patient's consciousness was clear in the first examination in the emergency department. Moreover, it was determined that the patient had a blood pressure of 120/80 mm Hg, heart rate of 72 bpm, and blood glucose of 140 mg/dL. During evaluations, he developed a sudden loss of consciousness. A sustained ventricular tacycardia (VT) attack was observed in the electrocardiogram (ECG) monitor, and synchronized cardioversion was applied with 50 J. After cardioversion, a normal sinusal rhythm was established, and the patient's consciousness normalized again. A 12-lead ECG was obtained just after the VT episode. There were no ischemia-related alterations in 12-lead ECG; however, QT prolongation was determined. The corrected QT (QTc) interval was calculated as 548 ms. Cardiac enzymes and electrolytes were found to be in normal ranges. Despite the fact that the level of K+ was 4 mE/L, parenteral K^+ and peroral magnesium treatment were provided to the patient because of the existence of VT resulting from QT prolongation. Echocardiographic examination of the patient demonstrated normal echocardiographic findings with an ejection fraction of 63%.

Frequent VT attacks reappeared after approximately 4h. Short-term episodes of a loss of consciousness accompanied those attacks. Parenteral magnesium therapy was initiated. Because of the unresponsiveness of VT attacks to the parenteral magnesium treatment, synchronised cardioversion was applied 8 times. Sustained VT episodes were reappearing in almost 5-10 min after cardioversion. A temporary VVI-PM was implanted in the patient. The heart rate was set as 110 bpm in PM. The VT attacks did not recur after PM implantation. In addition, coronary angiography was performed to rule out coronary artery disease. The angiography demonstrated no significant obstructive lesion in epicardial coronary arteries.

When PM was stopped after 8 h, QTc was determined to be 450 ms. Serum electrolytes were also in normal ranges at that moment. On the next day, solifenacin therapy was discontinued. TdP attacks did not recur. Temporary PM was removed after 24 h of observation. The patient was discharged after 3 days of hospitalization. He had no complaints in the outpatient controls, and the QT interval was measured as 420 ms.

In conclusion, patients taking QT prolonging drugs should be monitored in the hospitals for few days in case of TdP development. After documenting the first TdP attack, temporary PM should be immediately inserted with a ventricular rate of 110-120 bpm to shorten the QT interval.

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Coronary artery embolization after left ventriculography: A rare cause of myocardial infarction

To the Editor,

Coronary artery occlusion and myocardial infarction (MI) secondary to embolization of intracardiac masses such as thrombi or vegetation is a rare clinical entity (1, 2). Here we present a case of coronary artery embolization (CAE), which occurred after left ventriculography (LVG) in a patient with inferior MI.

A 62-year-old woman was admitted to our emergency department with typical chest pain for 6 h. Twelve-lead ECG showed ST segment elevation in D2, D3, and aVF, which was compatible with acute inferior MI. She received 300 mg acetyl salicylic acid and 600 mg clopidogrel and was transferred to the catheter laboratory for primary percutaneous intervention (PCI). Total occlusion in the middle portion of the right coronary artery (RCA) and a critical lesion (80% stenosis) in the mid left anterior descending (LAD) artery were detected. Intravenous heparin was administered, and direct stent implantation was performed, which restored TIMI 3 flow in RCA. Elective PCI was planned for the LAD lesion. Transthoracic echocardiography showed moderate mitral valve regurgitation (MR) and severe hypokinesia at the mid portion of inferior left ventricular (LV) wall, with LV EF of 45%. An intracardiac mass was not detected. On the third day of hospitalization, she developed dyspnea, and intravenous diuretic treatment was commenced. Control echocardiography was performed, which was consistent with the first examination. After consultation with the cardiovascular surgeons, we decided to perform control coronary angiography and ventriculography on the fourth day of hospitalization. First, LAD injections were administered, which showed a critical lesion (80% stenosis) in the LAD artery. LVG showed mild MR and hypokinesia of the inferior wall. Just after LVG, the patient described sudden onset chest pain, and ST segment elevation was observed on the monitor. The LAD artery was cannulated, and control angiograms were obtained. The LAD artery was found to be occluded with a huge thrombus at the site of the stenosis. Intravenous heparin and tirofiban infusion were administered, and PCI was performed. A 3 x 20 mm stent was implanted after balloon angioplasty, and distal TIMI 3 flow was restored. The rest of the hospitalization was uneventful.

Even in the modern era of intensive pharmacotherapy, including anticoagulant and antiagregant medications, embolic complications may occur after MI (3). In our case, CAE probably occurred secondary to micro-thrombi formed near the hypokinetic segments of LV during the early phase of MI. Dislodgement of micro-thrombi during catheter manipulation or contrast injection may have caused LAD occlusion. The diagnosis of micro-thrombi is challenging, and as in our case, transthoracic echocardiography may not have enough resolution for the detection of micro-thrombi. Other imaging modalities such as transoesophageal echocardiography may be safer for the evaluation of LV thrombi, aneurysm, and the severity of valvular disease in this patient population.

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