Letter to the Editor

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Variant angina associated with myocardial bridging and obstructive sleep apnea syndrome after lumbar spine surgery

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Variant angina is caused by transient and recurrent coronary spasms leading to repetitive episodes of transmural myocardial ischemia. Although the precise mechanism of coronary spasm remains unclear, various stimuli, such as hyperventilation, exercise, mental stress and administration of ergonovine, dobutamine or acetylcholine, can trigger coronary spasms [1].

A 31-year-old male patient without previous cardiovascular event, coronary risk factors or underlying disease was admitted for posterior decompression at the lumbar 4–5 level. General anesthesia was induced with fentanyl 100 μg and propofol 120 mg with no abnormal cardiac symptoms or signs. The operation was performed successfully. Postoperatively, the patient experienced lower back pain (LBP) with a score of 7 points on a numeric rating scale. Patient controlled analgesia and additional analgesics were administered, but the patient experienced sleep disturbance because of LBP. Five days later, he suffered from dizziness, dyspnea and severe chest pain for approximately 15 minutes, subsequently losing consciousness without seizure-like motion for 5 minutes.

To determine the cause of these postoperative symptoms, several tests were performed. Levels of cardiac markers were analyzed (CK-MB: 0.6 ng/ml, Troponin I: < 0.01 ng/ml, Myoglobin: 12.7 ng/ml, BNP: 17 pg/ml) and were found to be normal. Twenty-four-hour Holter electrocardiogram (ECG) monitoring

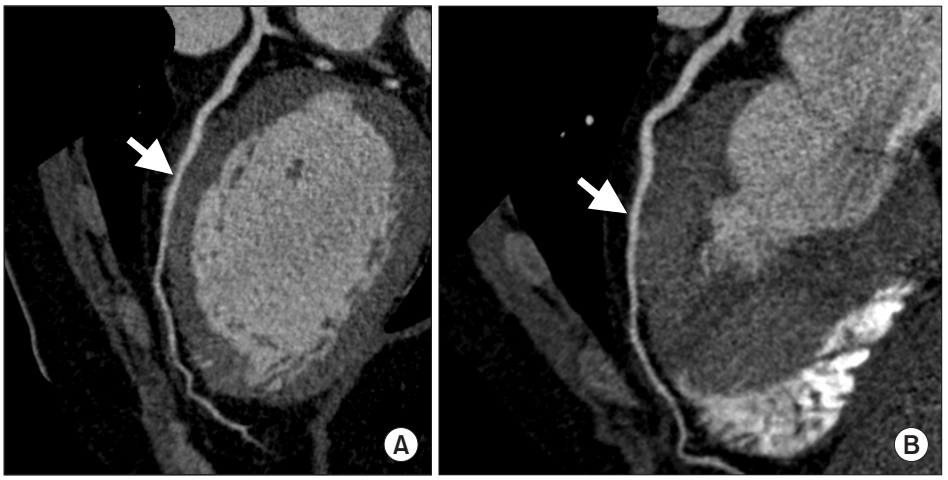

Fig. 1. Coronary CT angiography reveals high-grade myocardial bridging (arrow) in the mid-segment of the left anterior descending coronary artery. (A) Coronal view, (B) axial view.

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was performed, revealing subtle ST segment depression in the aVF lead. The patient also experienced chest discomfort at midnight. Coronary CT angiography revealed high-grade myocardial bridging at the mid-segment of the left anterior descending coronary artery (Fig. 1), which is known to have a 33% chance of abnormal diastolic contraction. Transthoracic echocardiogram revealed no regional wall motion abnormalities. The patient was diagnosed with variant angina and started on calcium channel blockers (diltiazem) 90 mg bid. The symptoms decreased in intensity and frequency, but intermittent chest pain persisted. The results of brain MRI were within normal limits, and no epileptiform discharges were seen in the electroencephalogram waking and sleep test. In the autonomic nervous system function test, normal responses were seen in the sympathetic skin response test for both hands and feet. Blood pressure and heart rate responses to postural changes were normal; however, abnormal variation was seen on heart rate variability analysis, which suggests dysfunction of the parasympathetic nervous system. On polysomnogram, sleep variables (apnea-hypopnea index [AHI]: 9.2 /hr, respiratory disturbance index [RDI]: 18.1 /hr, and arousal index: 27.8 /hr) were found to be abnormal, and the patient was diagnosed with obstructive sleep apnea syndrome. His body weight and height were 88.8 kg and 1.77 m, respectively. The patient's body mass index was calculated to be 28.25 kg/m^2.

Two months later, he was admitted for tonsillectomy, uvulopalatopharyngoplasty and septoturbinoplasty in order to relieve chest pain. The patient arrived at the operating room without premedication. His preoperative ECG showed normal sinus rhythm with a rate of 72 bpm, and his blood pressure was 129/65 mmHg. General anesthesia was induced with fentanyl 100 μg, propofol 70 mg and an infusion of nitroglycerine 0.5 μg/kg/min. After propofol injection, the patient complained of chest pain, but became gradually sedated. The ECG revealed sinus arrhythmia without ST segment depression or elevation. The patient's blood pressure was 113/68 mmHg, and his heart rate was 68 bpm. Nitroglycerine infusion was increased to 1.0 μg/kg/min, and the ECG subsequently returned to normal sinus rhythm. After 10 minutes, the patient was fully awake and did not complain of chest pain. The standard cardiac markers were checked and were found to be within normal range. After consultation with a cardiologist, general anesthesia was induced again with slow injection of propofol 100 mg and infusion of nitroglycerine 1.0 μg/kg/min while monitoring patient's response and ECG changes closely. There was no chest pain or ECG changes during the second induction.

In the early postoperative period, the patient complained of intermittent chest pain with ECG revealing ST segment elevation in leads V_1 to V_3. The symptoms subsided within 10 min without medication, and ECG revealed normal sinus rhythm.

One month later, the patient visited the cardiac outpatient department. His chest pain had diminished while taking medication continuously. On postoperative polysomnogram, sleep variables (AHI: 2.5 /hr, RDI: 8.6 /hr, and arousal index: 13.9 /hr) were decreased.

Myocardial bridging, which refers to a segment of the major coronary artery that runs intramurally through the myocardium, has been traditionally considered a benign congenital coronary abnormality. Its true incidence is not known, but has a wide range from 16% on angiography to 80% at autopsy [2]. Focal coronary spasm has been shown to result from endothelial dysfunction that cannot maintain a balance between flow-mediated vasodilatation and nonspecific vasoconstrictor stimuli in vascular smooth muscle [3].

Obstructive sleep apnea syndrome may be a predisposing factor for coronary spasm as a result of increased sympathetic activity during sleep [4]. Repetitive cycles of hypoxia and reoxygenation increase reactive oxygen species and reduce the availability of endothelial nitric oxide, resulting in endothelial dysfunction, playing an important role in the pathogenesis of coronary spasms [5].

The patient was definitively diagnosed with obstructive sleep apnea syndrome shortly after undergoing lumbar spine surgery and suffering from chest pains. Finally, chest pain diminished after undergoing upper airway surgery, and sleep variables were decreased (AHI, RDI, and arousal index) on polysomnogram.

After upper airway surgery, the patient complained of intermittent chest pain, and ST segment elevation was seen in leads V_1–V_3. ECG changes in leads V_1–V_3 seemed to be associated with myocardial bridging that was identified in the mid-segment of the patient's left anterior descending coronary artery, as the territory supplied by the left anterior descending coronary artery corresponds with leads V_1–V_3. On the other hand, many cases of coronary artery spasm during regional or general anesthesia have been reported. During induction of anesthesia for upper airway surgery, chest pain occurred within a few minutes of administration of propofol and fentanyl. It is possible that these agents provoked coronary artery spasm via activation of the autonomic nervous system.

In conclusion, we described a case of variant angina associated with myocardial bridging after a lumbar spine surgery. Obstructive sleep apnea syndrome and disorder of the parasympathetic nervous system induced by lumbar spine surgery are the most likely causative factors.

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