Coronary artery spasm induced by carotid sinus stimulation during neck surgery


Department of Anesthesiology, Seoul National University Hospital, Seoul National University College of Medicine, 28 Yongon-Dong, Chongno-Gu, Seoul 110-744, Korea

*Corresponding author. E-mail: limyjin@snu.ac.kr

We observed four transient episodes of marked ST-segment elevation in a 58-yr-old man with no history of coronary artery disease undergoing resection of a metastatic neck mass under general anaesthesia. Elevations of the ST segment were abrupt, with no change in arterial pressure or heart rate, and resolved spontaneously. When the carotid sinus was compressed directly, ST-segment elevation was noted 1 min after the onset of stimulation. After surgery, coronary angiography showed diffuse, slight narrowing of the distal bed of the posterolateral branch of the right coronary artery. Ergonovine caused total occlusion of the posterolateral branch of the right coronary artery with chest pain and ST-segment elevation, confirming the diagnosis of variant angina. The coronary artery spasm seems to have been provoked by vagal activation from carotid sinus stimulation during general anaesthesia.

Br J Anaesth 2003; 90: 391–4

Keywords: anaesthesia, general; surgery, cardiovascular; complications, coronary vasospasm

Accepted for publication: October 9, 2002

Vasospastic or variant angina is a well-known clinical condition first described by Prinzmetal and colleagues and is characterized by coronary artery spasm (CAS), which may occur in angiographically normal and diseased coronary arteries. The cause of variant angina is localized hyperresponsiveness of the vascular smooth muscle cells caused by non-specific stimuli of vasoconstriction. Autonomic imbalance can be one of the mechanisms of...
spontaneous vasospasm, and sympathetic or parasympathetic stimulation can induce CAS.2–7

The carotid sinus, located at the bifurcation of carotid arteries, has an important role in the baroreceptor reflex. Carotid sinus massage can cause cardiac vagal activity, which can cause CAS.8 We present a case of CAS during an operation for modified radical neck dissection under general anaesthesia, which recurred in association with carotid sinus stimulation. We present ECG and coronary angiogram results that confirmed this diagnosis. To our knowledge, this is the first case report to describe CAS evoked by carotid sinus stimulation during general anaesthesia.

Case report

A 58-yr-old man, height 167 cm and weight 74 kg, was to undergo resection of a 10×10 cm metastatic left laryngeal mass. The mass was firmly fixed to the mid-portion of the internal jugular vein. There was no personal or family history of hypertension and ischaemic heart disease. The patient had a smoking history of 30 pack-years, but had stopped smoking 7 months previously. Preoperative laboratory findings, including chest x-ray, ECG and pulmonary function tests, showed no abnormalities.

In the operating room, the patient’s arterial pressure (AP) was 145/75 mm Hg and heart rate (HR) was 75 beats min−1. Anaesthesia was induced with fentanyl 100 μg, propofol 120 mg and vecuronium 10 mg. After intubation, anaesthesia was maintained with nitrous oxide (50%), oxygen (50%) and isoflurane (0.5–2.0 vol %). Ninety minutes after induction, ST-segment elevation in lead II, not preceded by changes in HR or AP (HR 85 beats min−1, AP 120/70 mm Hg), was noted suddenly on the ECG monitor. The ST-segment alterations subsided completely in 5 min without treatment. Forty minutes later, an identical ECG change of marked ST-segment elevation, lasting only 1 min (HR 90 beats min−1, AP 110/69 mm Hg), was noted suddenly on the ECG monitor. The ST-segment alterations subsided completely in 5 min without treatment. Forty minutes later, an identical ECG change of marked ST-segment elevation, lasting only 1 min (HR 90 beats min−1, AP 110/69 mm Hg), occurred again during retraction of the tissues surrounding the mass. PaCO2 (42 mm Hg), PaO2 (203 mm Hg), haematocrit (36%) and plasma potassium concentration (4.2 mmol litre−1) were within normal limits. We suspected that the ST-segment elevation and carotid sinus stimulation were related. The carotid sinus was compressed for 10 s by steady finger pressure after removal of the neck mass. About 1 min later, the same ECG change of ST-segment elevation, lasting only 1 min (HR 90 beats min−1, AP 110/69 mm Hg), occurred again during retraction of the tissues surrounding the mass. PaCO2 (42 mm Hg), PaO2 (203 mm Hg), haematocrit (36%) and plasma potassium concentration (4.2 mmol litre−1) were within normal limits. We suspected that the ST-segment elevation and carotid sinus stimulation were related. The carotid sinus was compressed for 10 s by steady finger pressure after removal of the neck mass. About 1 min later, the same ECG change of ST-segment elevation, lasting only 1 min (HR 90 beats min−1, AP 110/69 mm Hg), occurred again during retraction of the tissues surrounding the mass. PaCO2 (42 mm Hg), PaO2 (203 mm Hg), haematocrit (36%) and plasma potassium concentration (4.2 mmol litre−1) were within normal limits.

We suspected that the ST-segment elevation and carotid sinus stimulation were related. The carotid sinus was compressed for 10 s by steady finger pressure after removal of the neck mass. About 1 min later, the same ECG change of ST-segment elevation, lasting only 1 min (HR 90 beats min−1, AP 110/69 mm Hg), occurred again during retraction of the tissues surrounding the mass. PaCO2 (42 mm Hg), PaO2 (203 mm Hg), haematocrit (36%) and plasma potassium concentration (4.2 mmol litre−1) were within normal limits. We suspected that the ST-segment elevation and carotid sinus stimulation were related. The carotid sinus was compressed for 10 s by steady finger pressure after removal of the neck mass. About 1 min later, the same ECG change of ST-segment elevation, lasting only 1 min (HR 90 beats min−1, AP 110/69 mm Hg), occurred again during retraction of the tissues surrounding the mass. PaCO2 (42 mm Hg), PaO2 (203 mm Hg), haematocrit (36%) and plasma potassium concentration (4.2 mmol litre−1) were within normal limits. We suspected that the ST-segment elevation and carotid sinus stimulation were related. The carotid sinus was compressed for 10 s by steady finger pressure after removal of the neck mass. About 1 min later, the same ECG change of ST-segment elevation, lasting only 1 min (HR 90 beats min−1, AP 110/69 mm Hg), occurred again during retraction of the tissues surrounding the mass. PaCO2 (42 mm Hg), PaO2 (203 mm Hg), haematocrit (36%) and plasma potassium concentration (4.2 mmol litre−1) were within normal limits. We suspected that the ST-segment elevation and carotid sinus stimulation were related. The carotid sinus was compressed for 10 s by steady finger pressure after removal of the neck mass. About 1 min later, the same ECG change of ST-segment elevation, lasting only 1 min (HR 90 beats min−1, AP 110/69 mm Hg), occurred again during retraction of the tissues surrounding the mass. PaCO2 (42 mm Hg), PaO2 (203 mm Hg), haematocrit (36%) and plasma potassium concentration (4.2 mmol litre−1) were within normal limits. We suspected that the ST-segment elevation and carotid sinus stimulation were related. The carotid sinus was compressed for 10 s by steady finger pressure after removal of the neck mass. About 1 min later, the same ECG change of ST-segment elevation, lasting only 1 min (HR 90 beats min−1, AP 110/69 mm Hg), occurred again during retraction of the tissues surrounding the mass. PaCO2 (42 mm Hg), PaO2 (203 mm Hg), haematocrit (36%) and plasma potassium concentration (4.2 mmol litre−1) were within normal limits.


Discussion

The typical ECG changes during an attack of variant angina are sudden ST-segment elevation in the lead overlying the ischaemic region, associated with ST-segment depression in the reciprocal leads. Characteristic ECG changes allow diagnosis of CAS. In this patient, we noted four episodes of marked ST-segment elevation. During surgery, marked ST-segment elevation and the QRS widening in the fourth episode were thought to be caused by ischaemic conduction delay, which can lead to potentially lethal ventricular arrhythmia. Thus lidocaine was used and the ECG changes

Fig 2 Coronary angiogram and ECG supporting the diagnosis of variant angina. (A) Coronary angiogram showed diffuse, slight narrowing of the distal bed of posterolateral branch of the right coronary artery. (B) The posterolateral branch of the right coronary artery was occluded totally after i.v. ergonovine, and (C) the spasm was relieved after intracoronary nitroglycerine. (D) The patient experienced chest pain with ST-segment elevation in leads II, III and aVF, and reciprocal ST-segment depression in leads I and aVL.
resolved. The ECG changes were interpreted later as ST-segment elevation by an experienced cardiologist. We are not sure whether lidocaine had a direct effect on the spastic vessel; it may have been given at the time of spontaneous resolution. The angiographic findings strongly suggest that spasm of the posterolateral branch of the right coronary artery caused the repeated ischaemic changes seen during surgery.

General anaesthesia can be a triggering event for CAS, although CAS during anaesthesia is a rare event.9±11 This case is unique in that the typical ECG changes of variant angina were provoked by surgical manipulation of the carotid sinus during general anaesthesia.

Sympathovagal imbalance caused by sudden increases in parasympathetic tone was thought to be the triggering factor for CAS in this patient. Previous studies have shown that the increased parasympathetic activity can trigger the development of coronary vasospasm.3±5 The carotid sinus reflex is a baroreceptor-mediated reflex that responds rapidly, a maximum response often being observed even in the first cardiac cycle after carotid sinus stimulation.12 The reflex does not continue for more than 20 s because of parasympathetic withdrawal and the return of sympathetic discharge. Nishizaki and colleagues8 showed that carotid sinus massage in an awake patient without significant coronary artery stenosis could provoke CAS on angiography in approximately 2.5 min with a short period of bradycardia. In our case, about 1 min elapsed from the carotid sinus stimulation to the appearance of ST-segment elevation, and bradycardia was not noted. Thus, in our case, involvement of reflex sympathetic activity in response to enhanced parasympathetic activity3±8 could have been a predominant mechanism.

In summary, we describe a case of CAS associated with carotid sinus stimulation during general anaesthesia in a patient with variant angina. Because of the possibility of fatal arrhythmias and myocardial ischaemia, early recognition of CAS-induced ST-segment elevation and prompt initiation of nitroglycerine infusion is indicated.

References