

CASE REPORT

Coronary artery spasm induced by carotid sinus massage

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Abstract

A 60 year old man with a history of frequent episodes of chest pain and dizziness was referred for evaluation of coronary artery disease. He had no significant coronary artery stenosis at baseline coronary angiography. A carotid sinus massage was performed for evaluation of carotid sinus hypersensitivity in the patient. Both heart rate and blood pressure decreased a little, and returned to baseline level immediately after carotid sinus massage. However, 2.5 minutes after carotid sinus massage, ECG showed ST segment elevation in leads II, III, and aVF. Four minutes after carotid sinus massage, he had chest pain with a progressive elevation in the ST segment in the same leads, when he had 99% focal spasm in the right coronary artery. The vasospasm induced by carotid sinus massage was reproducible over several minutes and resolved spontaneously. Coronary artery spasm may be provoked by the enhanced vagal activation due to carotid sinus massage.

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Coronary vasospasm may be provoked by a supersensitivity of the vascular smooth muscle cells, which may be associated with various stimuli.^{1,2} It has been reported that autonomic imbalance may often be a component of the mechanisms leading to spontaneous coronary vasospasm.^{1,3,4} Carotid sinus massage is known to provoke cardiac vagal activation,⁵ which may be involved in the genesis of coronary artery spasm.

A 60 year old man with a history of frequent episodes of chest pain and dizziness was referred for evaluation of coronary artery disease. Resting 12 lead ECG showed flattened T waves in leads II, III, and aVF, and precordial leads V4 to V6. The patient showed no abnormalities on cross sectional or Doppler echocardiography. No spontaneous chest pain or ischaemic ST segment changes were documented on ambulatory 24 hour Holter monitoring and exercise testing. Two days after admission, he had spontaneous chest pain during the night. Then, the ECG showed ST segment elevation in leads II, III, and aVF, which disappeared with subsidence of the attack after administration of sublingual nitroglycerin.

Coronary arteriography was performed four days after admission. The patient had no significant coronary artery stenosis at baseline angiography. We performed carotid sinus massage for evaluation of carotid sinus hypersensitivity in the patient, which was often accompanied by dizziness. His baseline heart rate and blood pressure were 68 beats per minute and 132/70 mm Hg, respectively. During carotid sinus massage on the right side of the neck, heart rate decreased a little to 50 beats per minute, and returned to baseline level immediately after carotid sinus massage. Blood pressure decreased to 100/56 mm Hg and increased to 120/70 mm Hg less than 10 seconds after carotid sinus massage. However, 2.5 minutes after carotid sinus massage, ECG showed ST segment elevation in leads II, III, and aVF. Four minutes after carotid sinus massage, he had chest pain with a progressive elevation in the ST segment in leads II, III, and aVF, when he had 99% focal spasm in the distal portion of the right coronary artery (figs 1 and 2). The

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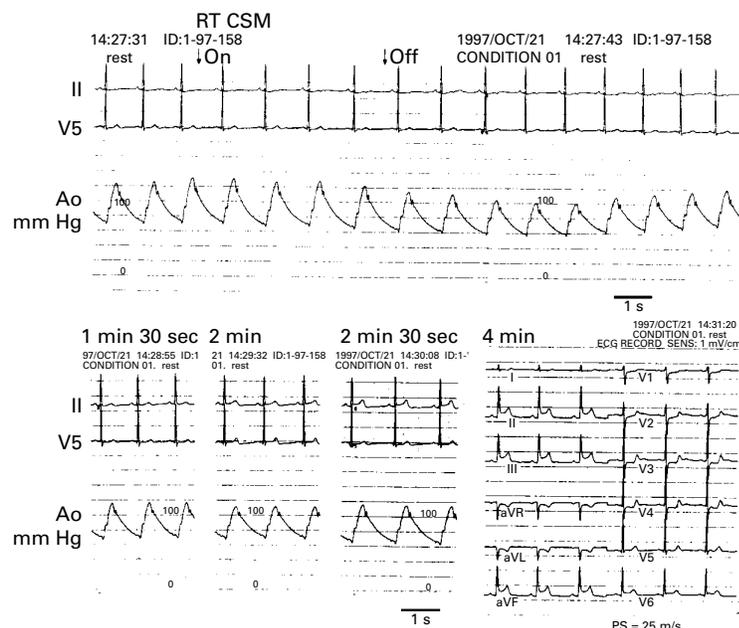


Figure 1 ECG at baseline and after carotid sinus massage. Ischaemic ECG change with marked ST segment elevation in leads II, III, and aVF were recorded four minutes after carotid sinus massage.

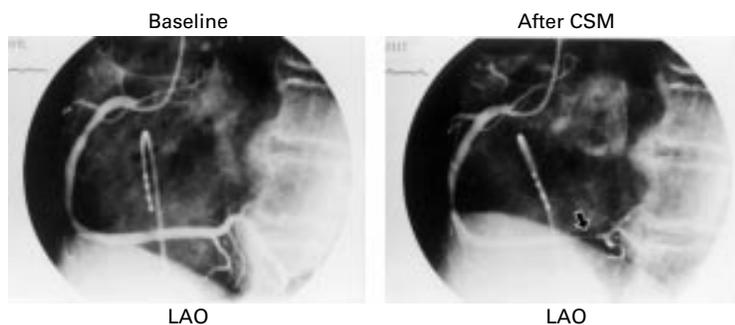


Figure 2 Coronary arteriography at baseline and after carotid sinus massage. Subtotal occlusion in the distal portion of the right coronary artery was induced by carotid sinus massage. CSM, carotid sinus massage; LAO, left anterior oblique.

vasospasm induced by carotid sinus massage was reproducible over several minutes and resolved spontaneously. Total vasoconstriction in the proximal portion of the left anterior descending artery was detected in response to intracoronary acetylcholine administration, and was completely relieved by the injection of isosorbide dinitrate.

Previous studies have shown that coronary vasospasm depends on the presence of autonomic imbalance.¹ Yasue *et al* reported that coronary vasospasm was induced by intracoronary injection of acetylcholine, the effect of which was blocked by premedication with atropine injection.³ Therefore, the vagal activation has been shown to play an important role in the pathogenesis of coronary spasm. However, Lanza *et al* showed that vagal withdrawal may frequently be a component of the

mechanism that can lead to or predispose to spontaneous coronary spasm.⁴

We report the first case of vasospastic angina, where vasospastic attack was induced by carotid sinus massage. In an animal study, coronary vasodilation has been reported to be provoked by activation of parasympathetic dilator fibres mediated with a graded baroreceptor, due to carotid sinus hypertension, and to disappear almost completely within 20 seconds.⁵ More than two minutes elapsed from carotid sinus massage to the occurrence of coronary spasm in this case. Heart rate decreased during carotid sinus massage and returned to baseline level less than 10 seconds afterwards, suggesting little residual vagal effect of carotid sinus massage. Therefore, stimulating the sympathetic nerve in response to enhanced activity of the parasympathetic nervous system, rather than residual parasympathetic activity, may affect coronary artery vasomotor tone.^{1,4}

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- 5 Kalsner S. Cholinergic constriction in the general circulation and its role in coronary artery spasm. *Circ Res* 1989;65:237-57.