Variant angina induced by biliary colic

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SUMMARY  A 65 year old woman with gall stones presented with crushing chest pain after an attack of biliary colic. The electrocardiogram showed ST segment elevation in leads I, aVL, and V1-V3 while leads II, III, and aVF showed ST segment depression. Cardiac enzyme activity remained within the normal range. During the next three weeks attacks of epigastric and right hypochondrial pain preceded by crushing chest pain with identical electrocardiogram changes occurred with decreasing frequency. Coronary arteriography showed 60% obstruction of the left anterior descending coronary artery and good left ventricular function. During the next three years the patient complained both of mild abdominal pain, probably biliary colic, and mild effort related angina pectoris without a relation between the two symptoms.

It is suggested that the attack of variant angina was triggered by biliary colic through sympathoadrenal discharge causing vasospasm.

Variant angina pectoris is characterised by recurring attacks of chest discomfort at rest associated with ST segment elevation on the electrocardiogram. ST segment changes disappear as the pain subsides and there is no evidence of myocardial necrosis. Various stimuli have been reported to induce variant angina; we describe a case of variant angina pectoris precipitated by biliary colic.

Case report

A 65 year old woman was admitted to our department with crushing left chest pains radiating to the neck. These followed intense spasmodic pain in the epigastrium and right hypochondrium radiating to the right shoulder. During the year before admission she had occasional attacks of mild right upper quadrant pain precipitated by fatty meals which were diagnosed as biliary colic; oral cholecystography showed gall stones.

On admission the patient was pale and sweating; her blood pressure was 140/80 mm Hg, the pulse was regular 80 beats/min, and she weighed 82 kg. There was acute tenderness at the upper abdomen; a fourth heart sound was audible on the precordium; the remainder of the examination was unremarkable. Chest x ray was normal. The electrocardiogram showed sinus rhythm, T wave inversion in leads V2-V4 (fig 1a). Laboratory data were as follows: creatine kinase 2·4 μkat/l (normal value < 1·6), alanine aminotransferase 0·66 μkat/l (normal < 0·37), aspartate aminotransferase 0·37 μkat/l (normal < 0·42), serum bilirubin 36 μmol/l (indirect 13·7 μmol/l), haemoglobin 142 g/l, haematocrit 43%, and a white cell count 10 500 with a normal differential count.

The day after admission she complained of spasmodic pain in the epigastrum and right hypochondrium; some while after, the pain worsened and radiated to the right posterior region of the thorax: the electrocardiogram showed sinus rhythm and biphasic T waves in leads V2-V4 (fig 1b). About 20 minutes later an intense oppressive chest pain developed, radiating to the neck, while the electrocardiogram showed sinus rhythm 74 beats/minute, ST segment elevation in leads I, aVL, and V1-V3 with ST segment depression in leads II, III, and aVF (fig 1c). The spasmodic epigastric and hypochondrical chest pain gradually disappeared after half an hour and the electrocardiographic pattern returned to that observed on admission (fig 1d); twelve hours later an electrocardiogram revealed biphasic T waves in leads V2-V4 (fig 1e). Cardiac enzyme activities remained in the normal range.

She was started on nifedipine (60 mg daily). The next day she had another attack of biliary colic angina with similar ST segment elevation on the electrocardiogram and papaverine (80 mg three times a day and verapamil (80 mg) three times a day were added to the treatment. In the next three weeks she complained of other attacks, but these declined in duration and intensity and the degree of ST seg-

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ment elevation also lessened. Cardiac enzyme activities remained in the normal range.

A month after admission the T waves in leads V2–V4 became positive and cardiac catheterisation revealed 60% obstruction of the left anterior descending coronary artery and good left ventricular function. The patient felt well and was discharged.

In the next three years she continued to complain of angina pectoris on effort (functional group New York Heart Association II) and of mild pain in the right upper quadrant, which was probably biliary. The occurrence of these two symptoms was unrelated. The patient refused cholecystectomy and continued taking calcium antagonists.

Discussion

In our patient the onset of the chest pain at rest, the typical electrocardiographic changes, and the lack of evidence of myocardial necrosis justify the diagnosis of variant angina pectoris; however, the presence of a 60% narrowing in the left anterior descending coronary artery suggests that it was mixed rather than classic variant angina. In this patient biliary colic caused a cramping pain in the right upper quadrant that radiated to the right posterior region of the thorax, whereas cardiac ischaemia caused a crushing pain in the left chest that radiated to the neck. Moreover, the pattern of the electrocardiogram during the biliary colic and before the onset of the chest discomfort was similar to that recorded during the pain free period. The temporal relation suggests that the onset of biliary colic triggered the variant angina.

Coronary artery spasm causes variant angina pectoris; but the mechanism by which it occurs remains unclear. It usually occurs at the site of an atherosclerotic lesion and the normal mechanism
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by which the endothelium counteracts vasospasm has been compromised.4

Coronary arteries are innervated by both sympathetic and parasympathetic nerves, both \( \alpha_1 \) (vasoconstrictor) and \( \beta_1 \) (vasodilator) adrenoceptors are present in the large vessels but the \( \beta_1 \) adrenoceptors predominate functionally; in the branch arteries only \( \beta_1 \) adrenoceptors are present: \( \alpha_2 \) adrenoceptors and muscarinic receptors are present on the sympathetic nerve endings of the coronary arteries; their activation reduces the release of noradrenaline and hence lessens the dilatation of these vessels.

Both parasympathetic5 or \( \alpha \) adrenergic6,7 stimulation can precipitate coronary vasoconstriction. A relation between spasm of the coronary artery and spasm of the oesophagus was found in some patients and the stimulation of \( \alpha \) adrenergic receptors was suggested as the common pathogenetic mechanism.8

We think that in our patient the sympathoadrenal discharge accompanying biliary colic could have triggered the \( \alpha \) adrenoceptors causing coronary vasospasm that resulted in an attack of angina.

References

4 Shepherd JT, Van Houtte PM. Mechanism responsible for coronary vasospasm. J Am Coll Cardiol 1986;8:50A-4A.