Chest pain, normal coronaries: whose problem?

Patients with symptoms of angina pectoris which do not respond adequately to medical treatment are likely to be referred in due course for coronary angiography. In 10–20% of cases this will show ‘normal coronary arteries’—that is, there will be no stenosis > 50% of vessel diameter in any of the major coronary vessels. The likelihood of this outcome is increased if the patient is female, or if a previous exercise test has been negative or equivocal. Patients with angiographically normal coronary arteries have a good prognosis in terms of future cardiovascular mortality or risk of myocardial infarction.¹ ² Patients are always delighted to hear this, but simply communicating the information does not necessarily alleviate the symptoms. Indeed, there is a real danger that patients can become confused and even hostile as a reaction to an apparent conflict between the reality of their symptoms and their perceived ‘dismissal’ by the angiographer. In the last issue, Potts and Bass underlined the clinical problem, namely the tendency towards persistence of symptoms and poor functional recovery, in the context of a follow-up period of more than ten years.³

Some cardiologists use the term ‘Syndrome X’ to describe the phenomenon of myocardial ischaemia in the presence of normal coronary arteries, but though this may be a convenient shorthand for research it is seldom helpful to the patient.

The first reaction to a finding of normal coronary arteries in a patient with chest pain must be to search for and if possible to document an alternative cause. The most likely candidates are musculoskeletal chest pain, oesophageal dysfunction, and, less commonly, aortic disease. Menopause-associated chest pain may, when it becomes better characterized, also join the list. Musculoskeletal pain should always be a positive rather than a negative diagnosis: the pain should be reproducible by palpation or by particular postures or movements. Radiography or more sophisticated studies such as magnetic resonance imaging are unhelpful in the absence of physical signs.

Oesophageal dysfunction is both a mimicker⁴ ⁵ and a provoker⁶ of myocardial ischaemia. Pain originating in the oesophagus can be misinterpreted as of cardiac origin, and in addition, acid stimulation of the lower oesophagus may induce coronary vasoconstriction.⁷ The potential for confusion is increased because both calcium antagonists and long-acting nitrates, widely used in the treatment of angina, tend to increase gastrooesophageal acid reflux.⁸ Oesophageal pain can be provoked by exercise, but characteristically occurs on recumbancy or when seated. If this is misinterpreted as angina at rest or decubitus angina, the likelihood of referral for coronary angiography is increased. Patients with ‘decubitus angina’ but good exercise tolerance are more likely to have oesophageal than coronary disease.

Neither a barium swallow nor endoscopy alone are reliable in excluding oesophageal pathology. Full investigation may need to include oesophageal manometry, ambulatory pH studies, and a Bernstein test (placing dilute acid in the lower oesophagus). Empirical therapeutic tests such as raising the head of the bed and a limited trial of a proton-pump inhibitor can also be useful.

Very occasionally, patients presenting with ‘angina’ turn out to have either an aneurysm of the ascending aorta or a chronic aortic dissection. Both are more common in hypertensive patients. The chest radiograph is usually helpful, but sometimes a computerised tomographic or magnetic resonance scan is diagnostic. The menopause is well recognized as the point from which the incidence of coronary disease in women begins to converge with that for men. It is less commonly appreciated that episodic chest tightness, which may superficially mimic angina, is sometimes a perimenopausal symptom, along with hot flushes and palpitation. A careful history, backed if necessary by measurement of follicle stimulating hormone and luteinizing hormone, may raise the suspicion, and hormone replacement therapy may provide relief.

What if all investigations are negative and the patient continues to suffer symptoms? Psychological abnormalities and hyperventilation have been implicated, but it remains uncertain whether such factors are causative or secondary. Some patients undoubtedly do have ‘syndrome X’, though its

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mechanism continues to elude us and there is no
definite agreement on effective treatment. If conven­
tional anti-anginal drugs are symptomatically helpful,
they should be continued, if not, they can be with­
drawn. Patients are not immune from progressing to
classical coronary disease and advice about smoking,
diet and exercise is often appropriate. Biofeedback
and pain relief clinics can sometimes help, and psy­
chological assessment or psychiatric treatment may
sometimes be of value.

Paradoxically, even if it is possible to identify an
alternative source of chest pain, it may be difficult for
the patient to accept this. An overconfident initial
diagnosis of cardiac pain, followed by refutation and
apparent rejection of the patient, is a potent recipe
for reducing trust in medical advice. Above all, these
patients need consistency of advice and continuity of
care. Whether this is to be provided by cardiologist,
general physician or general practitioner can be
debated, but in the context of ‘procedure related care’
and ‘finished consultant episodes’, it is a decision
which must not be neglected.

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