Prinzmetal's angina

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A 23-year-old man had Prinzmetal's variant angina. After strenuous exercise and isoprenaline infusion the electrocardiogram remained normal and the patient asymptomatic. Coronary angiography gave normal results which remained normal after administration of ergometrine. An injection of methacholine induced an attack.

In 1959 Prinzmetal and co-workers described a 'variant form of angina pectoris' (Prinzmetal et al., 1960). The essential features of this syndrome are chest pain at rest unrelated to exertion and an electrocardiogram characterised by a current of injury pattern during attacks. Occlusion or spasm of a major coronary artery with important reduction of the coronary blood flow has thought to be the cause of the syndrome.

We report the youngest patient, to our knowledge, with this variant form of angina pectoris in whom a complete haemodynamic and angiographic study did not show any obstruction in his coronaries. The ergometrine test failed to show any significant degree of coronary spasm, though it was possible to reproduce an attack by administration of methacholine.

Case report

A 23-year-old postman was admitted to the General State Hospital of Athens because of severe chest pain which woke him from sleep. The pain started in the midsternal region, radiated to the left chest, and was associated with a moderate degree of dyspnoea. The episode lasted for 15 minutes and disappeared, only to reappear later in the day. The patient had had two similar episodes during the night 7 months earlier.

The family history was non-contributory. The patient had smoked two packets of cigarettes daily for the past three years. Physical examination showed a well-developed man, with a blood pressure of 130/80 mmHg and a heart rate of 64 beats per minute and regular. The jugular venous pressure was not raised and the carotid pulse was normal. The left ventricle was not enlarged clinically and there was no murmur. There was a fourth heart sound heard along the left sternal border.

Laboratory data

The haemoglobin was 16.4 g/dl, with haematocrit 48.2 and WBC 7900/mm³, with normal differential. A biochemical screening survey, including CK, SGOT, was within normal limits with cholesterol 180 mg/100 ml (4.7 mmol/l). The triglycerides were normal 54 mg/100 ml (0.6 mmol/l). Multiple determinations of aspartate transaminase were within normal limits. VDRL test was normal. A chest x-ray film on admission was normal. The electrocardiogram during one of these attacks (Fig. 1A) showed ST elevation in leads I and aVL. Five minutes later when the chest pain had subsided, the electrocardiogram of the Fig. 1B was recorded: this shows ST-T abnormalities mainly in the anterior wall. The electrocardiogram recorded one hour after the attack was normal.

The patient underwent a multistage exercise test on a treadmill. He was able to reach the VII stage (6 m.p.h., 22° incline) without any complaint, electrocardiographic abnormalities suggestive of myocardial ischaemia, or arrhythmia. His heart rate increased normally and reached the expected maximal heart rate for his age (190 beats/min).

Isoprenaline infusion at a rate of 25 μg/min did not reproduce the attack and the electrocardiographic response was normal.

Retrograde left heart catheterisation showed pressures in the left ventricle of 120/9 mmHg, and in the ascending aorta of 120/60 mmHg. Selective right and left coronary angiography in many projections indicated normal coronary arteries (Fig. 2). Ergometrine maleate was then administered intravenously in doses of 0.1, 0.2, and again later 0.3 mg. At no time after the administration of this drug did the patient develop chest pain or electrocardiographic abnormalities. Opacification of each coronary artery after the administration of the drug did
Fig. 1  Electrocardiogram (A) during attack. (B) 5 minutes later.

Fig. 2  Right and left coronary arteriography in the left anterior oblique projection indicates normal coronary arteries.
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not reveal any significant spasm, either diffuse or localised, of the coronary vessels.

A typical anginal attack was elicited 12 minutes after subcutaneous administration of methacholine (0.13 mg/kg) and it was associated with ST segment elevation mainly in leads I and aVL. The pain was relieved immediately after sublingual administration of nitroglycerin.

Discussion

In this young patient all the episodes of pain occurred during the night at rest, a time when myocardial oxygen demand is reduced, and they were not precipitated by strenuous exercise, isoprenaline infusion, or administration of ergometrine maleate.

Cardiac catheterisation done when the patient was not experiencing an attack showed normal coronary arteries and a normally functioning left ventricle. This finding is common in patients with variant angina (Gianelly et al., 1968; Dhurandhar et al., 1972; Cheng et al., 1973; MacAlpin et al., 1973; Endo et al., 1975; Masahiro et al., 1975) as is that of stenosis no greater than 50 per cent in any of the major coronary arteries (Endo et al., 1976). There have been reports of aortocoronary bypass grafts on patients with this syndrome in whom, though the grafts were proved to have good patency after the operation, attacks still continued (Dhurandhar et al., 1972; King et al., 1973; Endo et al., 1976). These observations indicate that an atherosclerotic stenosis or occlusion of a major coronary artery is not always involved in this syndrome.

MacAlpin and his co-workers (1973) were the first to propose the 'spasm theory' as the cause of the symptoms. This suggestion has been supported by several recent reports (Dhurandhar et al., 1972; Oliva et al., 1973; Cheng et al., 1973; Endo et al., 1975, 1976) showing arteriographically that severe coronary arterial spasm was associated with the attacks.

Yasue et al. (1974) first reported that they could induce an attack of Prinzmetal's angina by methacholine. Though the methacholine test was not performed on our patient during cardiac catheterisation, the prompt development of typical chest pain and electrocardiographic abnormalities after the administration of the drug suggest coronary spasm as the possible mechanism of the attack. This has been shown lately by Endo et al. (1976) during coronary angiography.

The possibility of inducing severe spasm of the coronary arteries and pain in patients with Prinzmetal's type angina with a parasympathomimetic drug suggests that the autonomic nervous system is deeply involved in the pathogenesis of this syndrome.

References


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