Syncope in association with Prinzmetal variant angina

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A case of Prinzmetal variant angina with transient complete atrioventricular block and syncopal episodes following an anteroseptal myocardial infarction is described. The syncopal attacks were not prevented by demand cardiac pacing and were presumably caused by transient severe ischaemia of the left ventricle, with a consequent reduction in cardiac output. The left ventriculogram showed a large anterior dyskinetic area corresponding to the high grade proximal obstruction in the left anterior descending artery demonstrated by coronary angiography. All other coronary vessels appeared free of disease and it is suggested that the anginal episodes were caused by transient proximal segmental spasm of the right coronary artery. The anginal episodes were successfully prevented by a regimen of two-hourly coronary arterial vasodilator therapy.

In 1959 Prinzmetal and co-workers (1959) described a variant form of angina in which the chest pain occurred at rest and was not related to physical exertion or emotion. The characteristic feature of this syndrome is the occurrence of an electrocardiographic current of injury pattern during chest pain rather than ST segment depression which is the usual pattern of classical angina. Prinzmetal's variant angina also differs from classical effort-induced angina in that haemodynamic changes do not precede the anginal attacks though impairment of left ventricular function occurs during the actual episode of chest pain and correlates well with the degree of electrocardiographic abnormality (Guazzi et al., 1971).

This paper describes a patient with Prinzmetal variant angina who developed a pronounced reduction in cardiac output and syncope during anginal episodes.

Case report
A 50-year-old female patient was admitted to the Coronary Care Unit of the Royal Melbourne Hospital in June 1973, with a 4-hour history of severe crushing retrosternal chest pain. She had had hypertension for 12 years and a 12-month history of chest pain at rest but not on exertion. The hypertension had been well controlled with methyldopa and hydrochlorothiazide and she was on no other medication. Physical examination revealed a blood pressure of 140/90 mmHg (18.6/12.0 kPa) and a pulse rate of 96. Examination of the lung fields revealed fine basal crepitations. Her heart sounds were normal and there was no evidence of thyrotoxicosis or of any neurological abnormality. The electrocardiogram showed extensive anteroseptal infarction.

Twenty-four hours after admission she developed further chest pain lasting 5 minutes, and during this time the electrocardiogram showed complete atrioventricular block, with a ventricular rate of 60 per minute. She returned to sinus rhythm upon cessation of the pain.

On the fourth day after admission she had another episode of chest pain associated with transient loss of consciousness. Over the next 24 hours several episodes of chest pain followed by second (Mobitz I) (Fig. 1, panel C) and third degree atrioventricular block, with narrow QRS complexes and a ventricular rate of between 40 to 60 beats per minute, were documented. Each time, sinus rhythm returned immediately after relief of the pain. During these episodes, despite a ventricular rate of 40 per minute or more, the patient became drowsy. A temporary pacing wire was inserted and a His bundle electrocardiogram was obtained during sinus rhythm. The AH and HV intervals were 90 and 40 ms, respectively. Atrial pacing to a rate of 120 beats per minute did not induce chest pain or atrioventricular block and the AH interval increased to 120 ms.

In view of the recurrent episodes of complete heart block, a permanent demand pacemaker (Elema 153) was inserted and the patient was given practolol 100 mg twice a day.

She was discharged from hospital, but 2 weeks later was readmitted after an episode of severe chest pain and transient loss of consciousness with generalized convulsions and incontinence of urine. On admission she was in sinus rhythm and serial electrocardiograms and enzyme studies did not reveal any evidence of myocardial infarction. She was continuously monitored and on the second day after admission a typical attack was recorded (Fig. 1). She developed chest pain which was preceded by significant ST segment elevation in the inferior leads (Fig. 1 panel B). The PR interval progressively lengthened and the onset of a higher degree of atrioventricular block...
was interrupted by the implanted demand pacemaker. Despite a regular pacing rhythm (Fig. 1, Panel D), she became unconscious and the blood pressure could not be recorded. The episode was transient, the patient regained consciousness spontaneously, the blood pressure rose to normal levels, sinus rhythm returned, and the ST segment returned to the isoelectric line.

Several such episodes occurred under observation, each episode lasting between 3 and 5 minutes. The episodes could be aborted by prompt administration of sublingual nitroglycerine immediately the chest pain developed.

A provisional diagnosis of Prinzmetal variant angina was made and it was postulated that this was due to a critical lesion in the right coronary artery, resulting in atrioventricular block and severe myocardial pump failure. At cardiac catheterization the left ventriculogram showed a moderate sized ventricular cavity, with a large dyskinetic segment of the anterior wall. The left ventricular end-diastolic pressure was 13 mmHg (1.7 kPa) from the midchest level.

The left coronary angiogram showed a 90 per cent stenosis of the anterior descending artery just beyond the first diagonal branch (Fig. 2, Panel A). There were no other obstructive lesions in the left coronary system. The right coronary artery was the dominant vessel and was free of any significant obstructive lesion (Fig. 2, Panel B).

The artery to the atrioventricular node was well opacified and was normal. During the procedure the patient did not experience any chest pain.

Segmental spasm of the right coronary artery was then postulated as the cause of the anginal episodes and the patient was given isosorbide nitrate sublingually every 2 hours and was observed for a further 10 days. During this period no further chest pain occurred and she was discharged on this medication. She remained free of chest pain on this regimen for a further 6 months, after which she was electively admitted to hospital to observe the effects of reduction of her therapy. Twenty-four hours after withdrawal of the coronary vasodilator therapy the patient developed a further episode of chest pain and syncope and the 2-hourly isosorbide nitrate was immediately resumed, and since then the patient has remained free of attacks.

**Discussion**

The diagnosis of Prinzmetal variant angina in this patient was supported by the occurrence of angina at rest with preservation of exercise tolerance and the presence of marked ST segment elevation during the chest pain. Since the original description of this syndrome by Prinzmetal and co-workers, there has been considerable debate as to the underlying mechanism of ischaemic pain. Both postmortem (Prinzmetal et al., 1959; Peretz, 1961; Meriel et al., 1966; Dorra et al., 1968; Rizzon et al., 1969; Silverman and Flamm, 1971) and coronary angiographic studies (Dorra et al., 1968; Gianelly, Mugler, and Harrison, 1968; Whiting et al., 1970;
Cheng et al., 1972; Bobba et al., 1971; Dhurandhar et al., 1972) have indicated that most patients with this syndrome have a local area of narrowing in a major coronary artery. The degree of obstruction, however, is often insufficient to account for the symptoms at rest (Gianelly et al., 1968), particularly as exercise tolerance is preserved. Furthermore, there have been several reports of Prinzmetal angina occurring in association with normal coronary arteries (Ross and Gorlin, 1968; Demany, Tamba, and Zimmerman, 1968; Cheng et al., 1972; Whiting et al., 1970; Christian and Botti, 1972). For these reasons, coronary artery spasm has been postulated as the precipitating mechanism in this form of variant angina, but verification of this concept has been difficult. Recently, however, there have been 3 reports of Prinzmetal variant angina in which spasm of the right coronary artery during chest pain was documented by coronary arteriography (Oliva, Potts, and Pluss, 1973; Schroeder, Silverman, and Harrison, 1974; Kerin and Macleod, 1974). In all 3 cases the spasm appeared unrelated to the catheter tip and when the angiograms were performed in the absence of chest pain, the right coronary artery was normal in 2 cases and had minimal disease in the other case. The hypothesis that coronary artery spasm is the precipitating factor in Prinzmetal variant angina is further supported by the observation of Guazzi and co-workers, that continuous haemodynamic monitoring in 4 patients with spontaneous variant angina did not detect any circulatory changes preceding the anginal attacks (Guazzi et al., 1971).

In our patient, the occurrence of transient complete heart block, with narrow QRS complexes and the presence of ST segment elevation in the inferior leads during chest pain, suggested a critical lesion in the right coronary artery. The coronary angiogram, however, showed a dominant right coronary artery which appeared free of any obstructive lesion. The left anterior descending artery, as expected, had a proximal high grade obstruction accounting for the anteroseptal infarction and the corresponding dyskinetic area on the left ventriculogram. Thus in this patient, the right coronary artery supplied the major portion of the remaining viable myocardium as well as the atrioventricular node. Therefore, we postulate that this patient had intermittent spasm of the proximal right coronary artery producing severe ischaemia of both the left ventricle and the atrioventricular node, resulting in important reduction in cardiac output with consequent hypotension and syncope. Guazzi and co-workers (Guazzi et al., 1971), using sophisticated haemodynamic monitoring methods, have demonstrated transient impairment of left ventricular function including a reduction in cardiac output during attacks of variant angina, but this is usually
not severe enough to produce clinical manifestations (MacAlpin, Kattus, and Alvaro, 1973). In our case, however, left ventricular function had already been compromised by a large anterior wall infarction and, therefore, a sudden reduction in blood flow in the right coronary artery could be expected to produce a pronounced fall in the cardiac output, with consequent syncope. The diagnosis of coronary artery spasm in this patient is further supported by the satisfactory response to coronary artery vasodilators and the recurrence of symptoms on withdrawal of this therapy.

Transient atioventricular block during classical angina is unusual, but has been reported to occur in association with Prinzmetal variant angina where there has been involvement of the right coronary artery (MacAlpin et al., 1973). There have been three reports where a permanent pacing system has been implanted because of high grade atioventricular block and syncope during episodes of Prinzmetal angina (Bodenheimer et al., 1974; Gianelly et al., 1968; Whiting et al., 1970). In all cases, atioventricular conduction between attacks was normal, as was the situation in our patient even when the conducting system was stressed with atrial pacing. Chiche, Hait, and Steff (1974) have described two patients who developed syncope during episodes of classical (Heberden's) angina. Both patients had evidence of conduction disturbances between anginal episodes and in both patients the syncopal episodes were successfully treated by permanent pacing.

Our case illustrates the fact that, in Prinzmetal angina, syncopal episodes are not necessarily prevented by cardiac pacing even when high grade atioventricular block during an attack of chest pain has been documented. Therefore, we recommend that patients with syncope and atioventricular block should be monitored in hospital with a temporary pacing electrode and several anginal episodes observed before a decision to pace an individual patient permanently is made.

Reference


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