Case reports

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Atrial infarction leading to rupture

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SUMMARY In the case described, necrosis of the atrial musculature led to rupture of the chamber. This was overlooked for 4 days until the patient died of ventricular rupture. This unusual complication of atrial infarction may be amenable to surgical repair if recognised in time.

The high gain recording of P waves in the presence of AV block made it possible to evaluate the P wave changes during atrial infarction: (1) changes in the configuration and width of the P wave, probably resulting from intra-atrial conduction defect; (2) depression of the PR segment (0.04 to 0.07 mV); and (3) a previously unrecorded convexity in the first portion of the PR segment, in the leads in which the depression of the PR segment was observed.

Atrial infarction is a poorly defined and rarely recognised entity. Its clinical diagnosis is masked by the usually concomitant ventricular infarction, whereas the electrocardiographic diagnosis is based on vague signs such as 'abnormal displacement of the PR segment' or 'abnormal P wave configuration' (Wilson and Knudson, 1954; Freundlich and Sereno, 1959; Liu *et al.*, 1961; Flowers and Horan, 1966; Sivertssen *et al.*, 1973). Pathological diagnosis is also difficult and made only after careful search in the atrial musculature (Cushing *et al.*, 1942; Soderstrom, 1948; Wartman and Hellerstein, 1948; Liu *et al.*, 1961; Sivertssen *et al.*, 1973).

However, clinical and experimental studies show that atrial infarction influences the clinical course of myocardial infarction, leading to supraventricular arrhythmias, mural thrombi, and far less commonly the rupture of the chamber (Abramson *et al.*, 1938; Kohn *et al.*, 1954; James, 1961; Liu *et al.*, 1961; Levine *et al.*, 1972; Sivertssen *et al.*, 1973; Cristal *et al.*, 1975).

The purpose of this report is to describe a case in which the diagnosis of atrial infarction was made before death, with the aid of a method that allows the high gain recording of P waves (Cristal and Freidberg, 1974). The occurrence of atrial rupture was overlooked until necropsy examination.

Case report

A 71-year-old woman was admitted to the coronary

care unit on 11 June 1974, complaining of epigastric pain, cold sweating, and recurrent vomiting of 4 hours' duration. Three similar but shorter episodes had occurred during the previous month. She had been known to suffer from arterial hypertension for the past five years, but had received no treatment.

On admission, her blood pressure was 200/110 mmHg. No other abnormal findings were noted upon physical examination. The electrocardiogram showed signs of acute inferior wall myocardial infarction. A significant rise in serum levels of cardiac enzymes confirmed the diagnosis of myocardial necrosis. The chest x-ray film was normal.

On the third day in hospital, a variable degree of AV block developed. The heart rate ranged from 40 to 60 beats per minute and responded satisfactorily to intravenous administration of atropine. The atrium, described below, showed signs of atrial infarction.

On the sixth day, the patient's condition deteriorated while the heart rate remained unchanged (40-60 beats/minute). She became confused and incontinent of urine. The blood pressure fell to 130/70 mmHg and the venous pressure rose for the first time. During an attempt to place a temporary pacemaker through the right subclavian vein, the patient became restless and uncooperative, and cried out; the procedure had to be stopped before the electrode reached the right atrium. Immediately afterwards, the blood pressure dropped to 70/50 mmHg but rose spontaneously to 110/70 mmHg.

A chest x-ray film showed widening of the upper mediastinum on the right side, not seen previously and attributed to intramediastinal haemorrhage, probably caused by tearing of the subclavian vein. The haemoglobin dropped from 13.0 to 9.5 g/dl and the patient received 2 units of packed cells. Her condition remained stable for the next 3 days, despite an episode of praecordial pain with electrocardiographic confirmation of extension of the infarction to the lateral wall on the seventh day.

On the ninth day she complained of praecordial pain and dyspnoea. She suddenly lost consciousness and the blood pressure dropped to zero, while her heart rate was stable (80 beats/minute). She was treated with oxygen, digoxin, glucagon, and fluids, and gradually her blood pressure rose to 100/70 mmHg. She regained consciousness and passed 300 ml urine within a one-hour period. Venous angiography was performed and, with the diagnosis of perforation of the subclavian vein, the patient was transferred to the operating room. At operation no mediastinal haemorrhage was found, but there was a large amount of blood in the pericardium. While opening the pericardium, there was a sudden massive haemorrhage of at least 3 litres and the patient went into cardiac arrest. Two tears were noted in the myocardium: one in the inferior wall of the left ventricle and the other in the posterior wall of the right atrium. The patient received 8 units of blood under pressure. All attempts to suture the ventricular rupture failed and the patient died on the operating table.

NECROPSY FINDINGS

At necropsy the heart weighed 400 g. Numerous focal haemorrhages were seen on the anterior and posterior walls of the left ventricle, the posterior wall of the left atrium near the interatrial septum, and on the posterior wall of the right atrium. A complete rupture of the myocardium, 4.5 cm long, was seen in the posterior wall of the left ventricle near the interventricular septum. Another rupture, 1.5 cm long, was seen in the posterior part of the interatrial septum, extending to the posterior wall of the right atrium. In the proximal part of the right coronary artery there was an organised thrombus with recanalisation.

Histological sections from both atria showed foci of myocardial infarction in the posterior wall of the right atrium, the left atrium, and the interatrial septum; infarction 7 to 8 days old and more recent infarction (3 to 4 days old) were seen. Histological sections from the posterior wall of the left ventricle also showed foci of infarctions, 7 to 8 days old, 3 to 4 days old, and 24 to 28 hours old.

Fig. Four consecutive atrigrams—see text.

COMMENTS ON ATRIGRAMS

The atrigram shows P waves enlarged tenfold, recorded simultaneously with the standard electrocardiogram at a paper speed of 50 mm/s (Cristal and Freidberg, 1974). The Figure shows four consecutive tracings and the most important measurements of the atrigrams are shown in the Table. Atrial infarction was thought to be present because of

Table Measurement of some relevant features in the atrigrams

Date	P wave width (ms)	Components			PR depression (mm)	PT _a * (atrial depolarisation) (ms)
		1st (ms)	2nd (ms)	3rd (ms)		
12.6.74	120	45	35	40	4 (II, aVF)	—
14.6.74	105	45	20	40	—	380
16.6.74	105	45	20	40	—	340
18.6.74	115	50	30	35	5 (II, III, VI) 7 (aVF)	400

*PT_a was measured in isolated P waves during AV block.

changes in the shape and width of the P wave, changing displacement of the PR segment in leads II, III, and aVF, and the development of a convexity in the PR segment in the same leads.

(a) Changes in shape and width of the P wave were present and mainly related to the appearance and disappearance of a deflection in its middle portion.

(b) Depression of the J point (the junction between the end of the P wave and the take-off of the PR segment) was present in the atriogram of 12 June 1974 in leads II and aVF; this depression returned towards the isoelectric line during the following days, but reappeared 6 days later.

(c) A convexity was recorded in the first part of the PR segment in leads where the J point was depressed (more clearly visible during the recording of isolated P waves on 14 June 1977). This convexity, with its mirror-image in aVR, evolved progressively in the leads where it was present.

Discussion

The diagnosis of atrial infarction is based largely on the electrocardiogram. The diagnostic features agreed by all authors (Wilson and Knudson, 1954; Freundlich and Sereno, 1959; Liu *et al.*, 1961; Flowers and Horan, 1966; Sivertssen *et al.*, 1973), are the abnormal configuration of the P wave and the displacement of the PR segment. More precise criteria relating to the extent, direction, and duration of the electrocardiographic changes have not been defined and, not infrequently, recognition of atrial infarction on the standard electrocardiogram depends upon the sharp eyes of an experienced cardiologist.

In the present case, the tenfold magnification of the P waves and the fortuitous occurrence of complete AV block enabled us to study more closely the change during depolarisation and repolarisation of the atria in the course of an anatomically-proven infarction of the atrial chambers. The changes in the configuration of the P wave were transient, as in other reported cases (Liu *et al.*, 1961; Sivertssen *et al.*, 1973). We can therefore assume that atrial infarction does not necessarily change the P wave by producing a stable 'wave of necrosis' like the Q wave of the ventricular infarction, but rather it changes its configuration by altering the spread of depolarisation. Displacement of the PR segment is thought to be a crucial sign in the diagnosis of atrial infarction (Liu *et al.*, 1961; Sivertssen *et al.*, 1973) and depression of the J point is far more frequent than its elevation (Sivertssen *et al.*, 1973). In the present case, a depression of 0.04 mV was present in the first atriogram in leads II and aVF, returned

toward the isoelectric line on the next atriogram, and reappeared on the atriogram recorded 6 days later. The occurrence of two separate ischaemic events within the atria was confirmed by the pathological findings. The presence of a convexity in the first part of the PR segment has never been previously described, and we are inclined to believe that this is an abnormal finding, since the normal atrial repolarisation vector must be in the opposite direction to the depolarisation vector. It is possible that this convexity is the equivalent of the ST elevation characteristic of ventricular infarction.

In retrospect, it seems likely that the infarcted right atrium ruptured 4 days before the ventricular rupture, during the unsuccessful attempt to insert an electrode through the right subclavian vein. The rise in the right atrial pressure which developed during the procedure, when the patient in the Trendelenburg position became distressed and cried, was probably an important contributing factor to the rupture. The bleeding into the pericardial sac during the first hours led to a drop in blood pressure and haemoglobin, but, later, increased intrapericardial pressure resulted in a decrease or even cessation of the bleeding. The clinical condition of the patient then remained stable for the next 5 days, until she became shocked after ventricular rupture.

Ventricular rupture is the cause of death in 8 to 11 per cent (Meurs *et al.*, 1970; Björck *et al.*, 1972; Van Tassel and Edwards, 1972) of patients dying of myocardial infarction. By contrast, rupture of the atrial chambers is a rare complication of myocardial infarction and since the case reported by Kohn *et al.* (1954), we found no further instance reported in the publications available to us. Of the 79 cases of atrial rupture collected at that time, 15 per cent survived more than 24 hours; the authors attributed this to the fact that the bleeding into the pericardium was not as massive as in ventricular rupture. In this series, the diagnosis was never made before death. The concurrent development of atrial and ventricular rupture has never been reported before, but the occurrence of more than one ventricular rupture in the same patient has been noted in a few cases who survived the first rupture (Björck *et al.*, 1972).

Atrial infarction is an overlooked entity and further studies are necessary in order to define more reliable criteria for its diagnosis. The recording of high gain P waves during myocardial infarction should contribute to this. In the present report, we draw attention again to the occurrence of this unusual complication of myocardial infarction and stress the fact that a patient with atrial rupture can survive long enough for it to be possible to attempt

surgical repair, provided that the diagnosis can be established.

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