RUPTURED AORTIC VALVE WITH MYCOTIC ANEURYSM DUE TO ACUTE BACTERIAL ENDOCARDITIS

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Although mycotic aneurysms due to bacterial endocarditis are a recognized cause of rupture of one or more cusps of the aortic valve, the following case is of interest owing to the absence of fever.

CASE REPORT

Mrs. R. W., aged 48, was seen on September 16, 1940, with Dr. Bessie Brown. Two weeks previously she had begun to have attacks of shivering. Two days later she was taken ill in a cinema, and on her way home in the black-out collided with a lamp post. This gave rise to pain in the front of the chest on the right side, but there was no bruising. On the following day she was forced to remain in bed on account of retching associated with a dry, irritating cough and with shortness of breath. During the few days prior to examination streaks of blood had been coughed up, and dyspnoea had become increasingly severe; she had lost all desire for food and the tongue had been furred. The illness was afebrile throughout.

Examination revealed a stout, apprehensive woman, looking rather pale. The pulse was intermittent at a rate of about 90. The blood pressure was 150/30 mm. The apex impulse was not palpable, owing to the thick chest wall. The first sound at the apex was clear, but the second sound was followed by a rough diastolic murmur, which could be traced up the left border of the sternum and was audible at the second right space; there was no thrill. Crepitations were present at the bases of both lungs; they extended further up the back on the right side than on the left. The liver was not enlarged. There were no petechiae; no Osler’s nodes; and the spleen was not palpable.

The combination of the rapid onset of left ventricular failure with the murmur of aortic incompetence and a greatly increased pulse pressure suggested a ruptured aortic valve, and she was admitted to hospital on the following day for investigation regarding the possibility of a bacterial endocarditis.

The blood showed a moderate secondary anaemia (haemoglobin, 70 per cent, red cells, 3-6 millions). The leucocytes numbered 22,900 (polymorphs, 82 per cent). The Wassermann reaction was negative. The urine contained albumen, a few red cells, and a trace of pus. The temperature remained sub-normal throughout, and on this account no blood culture was made.
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The electrocardiogram on admission showed a 2:1 A-V block, with an auricular rate of 124 and a QRS of 0.08 sec. (Fig. 1), but six hours later the auricular rate had risen to 148 and the QRS was 0.12 sec. Digoxin (0.25 mg.) was given four hourly and led to considerable clinical improvement, but the dose was halved after 12 tablets (3 mg.) had been given, since the ventricular complexes

![Fig. 1.—17/9/40. 2:1 A-V block (A, 124; V, 62): QRS, 0.08 sec.]

![Fig. 2.—19/9/40. 2:1 A-V block in leads I and II (A, 106; V, 53) with right bundle branch block (QRS, 0.12 sec.). There is some depression of the S–T interval. In lead III there is an admixture of 2:1 block and 1:1 rhythm (P–R, 0.56 sec.).]
had become those of a type B right bundle branch block with depression of the S-T interval, although the auricular rate had fallen to 106 (Fig. 2). Four days later 1:1 rhythm returned at a rate of 74 and the ventricular complexes were becoming normal. They were quite normal in another four days (Fig. 3).

**Fig. 3.—26/9/40.** 1:1 rhythm at a rate of 74 (P-R, 0·50 sec.). A sinus irregularity gives rise to an apparent variability in the A-V block.

Sept. 26); the P-R interval was 0·50 sec., although a sinus irregularity gave rise to an apparent variability in the A-V block. On September 30, pulmonary oedema set in with further haemoptysis, and she gradually sank and died on October 2, fifteen days after her admission to hospital. Normal ventricular complexes and 1:1 rhythm were still present a few hours before death.

**Autopsy**

**Heart.** The heart weighed 425 g., the relative thickness of the right and left ventricular walls being normal. The aortic valve was grossly incompetent, owing to a complete rupture of the right posterior cusp (Fig. 4). Around the site of the rupture, and particularly behind the damaged valve, there was much vegetative proliferation, which on the whole was firmly adherent and extended deeply into the membraneous part of the ventricular septum. There were further wart-like vegetations at the base of the left posterior cusp. The remaining valves were normal. There was no evidence of congenital abnormality.

On opening the right auricle a tumour was seen situated immediately above the junction of the anterior and medial cusps of the tricuspid valve (Fig. 5). The tumour was cone-shaped, with a height and basal diameter of about 1 cm.
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It was of the same colour as the auricular wall, was of a firm consistency, and had a smooth surface; it could not be moved on its base. The tumour emerged from the membraneous part of the septum, and on section proved to be an aneurysmal sac that communicated directly with the excavating cavity round the ruptured valve.

Other Systems. There was some oedema and congestion of the lungs,
Fig. 5.—Right side of the heart showing the aneurysm.

(A) Aneurysm projecting into right auricle.
(B) Right auricle.
(C) Right ventricle.
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especially at the right apex. There was a small right hydronephrosis. The spleen was normal.

HISTOLOGY

Sections were prepared from the vegetations around the aortic valve and the adjacent myocardium, and also from the centre of the auricular tumour. These were stained with hematoxylin, eosin, and Gram’s stains.

1. Aortic Vegetation. Whilst a little of the tissue consisted of platelet thrombus, the greater part was composed of a loose stroma, which was grossly infiltrated with round cells and polymorphs: numerous red blood cells were also present.

2. Wall of the Aneurysm. The aneurysmal wall consisted of three distinct layers. The outer layer was composed of dense fibrous tissue, the middle layer of muscular tissue together with a few fibroblasts and lymphocytes, whilst the inner layer was chiefly platelet thrombus containing a few fibroblasts and polymorphs.

3. Heart Muscle. There was no evidence of rheumatic disease.

The diagnosis after autopsy was a ruptured aortic valve with a mycotic aneurysm due to acute bacterial endocarditis.

DISCUSSION

Except for the first two days of malaise the patient was under observation in bed and the temperature never rose above normal. Horder (1908) noted that fever was absent in five out of his one hundred and fifty cases, but he thought it was unlikely that these cases were afebrile throughout, and that they had probably come under observation during an afebrile phase. Perry (1936) had one case in which the temperature was normal for thirteen weeks. Possibly the rapid course of the illness with death from heart failure four weeks after the onset did not allow time for fever to develop.

It is uncertain if digitalis exercises any effect upon intra-ventricular conduction. White (1937) thought that it did to some extent, but Comeau and Hamilton and White (1938) could find no evidence of such an action. The situation of the aneurysm made some interference with conduction inevitable, and complex changes in conduction have been recorded in a similar case by Wishaw (1940). The widening of the QRS began before digitalis was given, during a period of increasing heart failure, and this association has been noted frequently in transient bundle branch block (Comeau, Hamilton, and White, 1938). On the other hand, the right branch block increased while clinical improvement was taking place, and did not regress until four days after the digitalis dosage had been reduced; neither did it recur, although death took place from pulmonary oedema. In the absence, however, of evidence from other sources of a similar action of digitalis it would appear more likely that the transient right branch block was occasioned by the heart failure rather than by digitalis.
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SUMMARY

A case of rupture of the aortic valve with a mycotic aneurysm due to bacterial endocarditis has been described. The temperature was sub-normal throughout the illness. Death took place quickly with signs of left ventricular failure.

REFERENCES

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