CASE REPORTS

SPONTANEOUS RUPTURE OF A PAPILLARY MUSCLE OF THE HEART

BY

G. A. STIRLING and B. F. LAMBERT

From the University College of the West Indies

Rupture of a papillary muscle of the heart is a rare condition. The subject has been reviewed by Davison (1948) who summarized 26 previously reported cases and added 3 of his own, and by Askey (1950) who reported 8 additional examples. Up to 1953 only 44 cases had been recorded. The most common cause was cardiac infarction affecting the base of a papillary muscle. In a few instances rupture has been ascribed to ulcerative endocarditis, syphilis, trauma, abscess formation, and periarteritis nodosa, and in three patients the cause was unknown.

In the present case there was no evidence of cardiac infarction, a condition rarely met with in Jamaica, and the cause was only revealed by the examination of serial sections of the papillary muscles.

Case History

The patient was a man aged 56 years who was first seen at the University College Hospital, Jamaica, in September, 1958. He had hypertension, but had been well otherwise until three months before when he complained of dyspnoea and retrosternal pain aggravated by exertion and had coughed up frothy sputum.

On examination he was well nourished and showed no signs of congestive cardiac failure. His pulse was 80 a minute and regular. The blood pressure was 190/140. The apex beat was in the sixth left intercostal space in the anterior axillary line. There was no palpable thrill. Auscultation revealed triple rhythm but no murmur. No abnormal signs were found in the lungs. The liver, which was palpable two finger breaths below the right costal margin, was firm but not tender. The spleen was not palpable.

An X-ray of the chest showed gross cardiac enlargement; an electrocardiogram showed right bundle-branch block, and left ventricular hypertrophy and strain. The V.D.R.L. test was positive in a dilution of 1 in 8.

Four months later he was admitted to hospital with signs of cardiac asthma. His blood pressure had fallen to 140/50. Although he did not look unduly distressed and his condition gave no cause for alarm, he died suddenly on the same day.

Necropsy

The heart was enlarged weighing 620 g. The left ventricle was 18 mm. thick and the right 6 mm. The tip of the posterior papillary muscle of the left ventricle was necrotic and completely ruptured (Fig. 1). The surface of the anterior muscle was hæmorrhagic but there was no necrosis. The papillary muscles on the right side of the heart were normal. The coronary arteries were patent and showed only a mild degree of atheroma. The myocardium was healthy and showed no evidence of infarction. The chordæ tendineæ of both ventricles and the mitral and tricuspid valves were normal. The aorta showed longitudinal wrinkling suggestive of syphilitic aortitis in the ascending portion of the arch. In the abdominal aorta there was conspicuous atherosclerosis. All the lobes of both lungs were congested and œdematous.

Histology. Posterior Papillary Muscle. A small infarction involved the tip of the papillary muscle. The endocardial surface of the affected muscle was covered by blood clot. Random

section revealed no cause for the infarction since the vessels, though thickened, were patent. However, the study of serial sections revealed an aneurysm extending for 3 mm. before being lost to view in the torn end of the muscle (Fig. 2). The artery concerned was centrally situated and was the largest in the papillary muscle. Proximal to the aneurysm the vessel showed a hyaline thickening similar to that present in other vessels in the papillary muscles of both ventricles. The thickening was periodic-acid-schiff negative and did not stain for amyloid. The transition from thickened vessel to aneurysm was abrupt and occurred immediately beyond a point where a branch was given off. The lumen was occluded by recently formed coagulum and the adjacent myocardial muscle was necrotic and infiltrated with polymorphonuclear leucocytes. Sections stained by Warthin's method revealed no spirochætes. At the point of rupture a second torn but otherwise healthy artery was seen.

FIG. 1.—The ruptured posterior papillary muscle indicated by arrows.

FIG. 2.—Aneurysm of a small artery in the posterior papillary muscle. At this point the vessel was only partially occluded. H. & E. $\times 190$.

Anterior Papillary Muscle. This showed a small area of degenerate muscle fibres. The arteries were thickened. In some vessels the wall was changed to a structureless swollen eosinophilic material. The lumina were narrowed and in one instance almost obliterated. The elastica was split and increased in amount. *Papillary Muscle from Right Ventricle.* A few degenerate fibres were seen. The arteries were patent.

Myocardium. Fibrosis was seen in the left ventricle but no evidence of either old or recent infarction. No remarkable feature was seen in the branches of the coronary arteries.

Aorta. There was scarring and vascularization of the media. Destruction of elastic tissue and foci of chronic inflammatory cells were present. The appearances were those of syphilitic aortitis but no spirochætes were seen in the sections stained by Warthin's method.

Discussion

It is almost certain that the sequence of events in the present case was aneurysm formation with occlusion of the aneurysmal sac followed by infarction and rupture of the posterior papillary muscle. The pathogenesis of the aneurysm and therefore of the rupture is not certain. Infection

was believed to be responsible for the cases of ruptured papillary muscle reported by Bertin (1824) and Spanton (1865). In both cases vegetations on the valves and chordæ tendineæ were described. More recently Hackel and Kaufman (1953) described a case in which *Entamoeba coli* septicæmia following acute pyelonephritis resulted in a myocardial abscess and rupture of the left anterior papillary muscle. There was no vegetation or abscess in the present case. Periarteritis nodosa leading to myocardial infarction and rupture of a papillary muscle has been reported by Askey (1950). In the patient under discussion the aneurysm was similar to those encountered in periarteritis nodosa but it was an isolated lesion. Trauma has been implicated in cases described by Kleberger (1920), Glendy and White (1936), and Payne and Hardy (1937). No history of trauma was elicited in the present case and the patient was at rest in bed when the rupture occurred.

In the absence of myocardial infarction and the more uncommon causes mentioned above of ruptured papillary muscle, the presence of a positive V.D.R.L. test and evidence of aortitis suggested syphilis as the ætiology, although no spirochætes were demonstrated and a syphilitic aneurysm in a small vessel must be unusual. Syphilis as a cause of ruptured papillary muscle has been substantiated in only one case when Spaulding and Glahn (1921) found spirochætes in the ruptured ends of a papillary muscle of a man dying with congestive heart failure.

The posterior papillary muscle of the left ventricle is more frequently ruptured than the anterior muscle. Rarely are the muscles of the right ventricle involved. Only one report exists of the rupture of two papillary muscles (Nicod, 1834). In the present case, in addition to a ruptured posterior papillary muscle, there was an area of degeneration in the anterior papillary muscle. No aneurysm was found in this muscle; by contrast the arterioles showed a curious thickening which reduced the lumen of one vessel to a tiny slit.

Rupture of a papillary muscle is generally followed by sudden collapse and death as in the present case. Nevertheless Askey (1950) has recorded two patients who survived two and six months. In the latter the ruptured end of the papillary muscle was covered by endothelium. A number of patients survive for a short time and in these the problem of diagnosis is encountered. According to Askey (1950) the possibility of ruptured papillary muscle must be considered when an apical systolic murmur suddenly appears in a patient with cardiac infarction, sepsis, endocarditis, or syphilis. The condition is to be differentiated from ruptured interventricular septum. In the latter condition, according to Askey (1950), a systolic thrill is frequently met with, but in ruptured papillary muscle this sign is absent. On the other hand Hackel and Kaufman (1953) noted an apical systolic thrill after rupture of a papillary muscle and felt that this criterion must be considered of less significance than was previously thought.

Summary

An unusual case of ruptured papillary muscle of the heart is described. The study of serial sections revealed a small aneurysm in the ruptured papillary muscle. The cause of the aneurysm was not certain but it was probably syphilitic.

We wish to thank Professor G. Bras for reading the manuscript.

References

- Askey, J. M. (1950). *Amer. J. Med.*, **9**, 528.
 Bertin R. J. (1824). *Traité des Maladies du Cœur et des Gros Vaisseaux*. Baillière, Paris.
 Davison, S. (1948). *J. Mt. Sinai Hosp.*, **14**, 941.
 Glendy, R. E., and White, P. D. (1936). *Amer. Heart J.*, **11**, 366.
 Hackel, D. B., and Kaufman, N. (1953). *Ann. intern. Med.*, **38**, 825.
 Kleberger, K. (1920). *Virchows Arch. path. Anat.*, **228**, 1.
 Nicod, J. (1834). *Hebdomen des Progres des Sciences Medicales*, **4**, 42.
 Payne, W. C., and Hardy, H. H. (1937). *New Orleans med. surg. J.*, **89**, 373.
 Spanton, W. D. (1865). *Trans. Path. Soc. London*, **16**, 73.
 Spaulding, E. D., and Von Glahn, W. C. (1921). *Bull. Johns Hopkins Hosp.*, **32**, 30.