Rupture of Papillary Muscles: Occurrence of Rupture of the Posterior Muscle in Posterior Myocardial Infarction

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Introduction

Rupture of a papillary muscle of the heart has been considered an unusual complication of myocardial infarction. Although first recognized as early as 1808, only 56 cases have been reported to date.

The literature was reviewed by Davison in 1948. He added three cases to 26 previously reported. Other instances have been described by Glendy and White; Payne and Hardy; Askey; Smith, Schwartz and Canelli; Hope and Askey; Craddock and Mahe; Briggs; Ritama and Heino; Segall and Sharp; Oeser; and Reuter.

Our main purpose in presenting five more cases is to emphasize the typical combination of rupture of the posterior papillary muscle following simultaneous infarction of that muscle and of the posterior wall of the left ventricle. We should also like to note that in our experience, rupture of a papillary muscle in general has not been too rare an occurrence. We saw the five cases reported here in a relatively short period of time, and we know of an additional five cases which were not included because of insufficient clinical data.

Case 1: E. P., a 78 year old man, entered the hospital on August 30, 1964, complaining of retrosternal pain radiating into both arms. The pain started the night before admission and had never been present before. He had been treated for bronchial asthma during the previous six months. Examination revealed a weak, lethargic, and drowsy man, who appeared younger than the stated age. The blood pressure was 100/70 mm. Hg., and the pulse was 60 per minute and regular. Scattered expiratory wheezes were heard. The heart was not enlarged. No thrills or murmurs were found. The electrocardiogram showed an acute posterior myocardial infarction. The sedimentation rate was slightly elevated. He was placed in an oxygen tent and started on dicumarol. He did well during the first 17 days of hospitalization and had only an occasional bout of pain. During the afternoon of the 18th hospital day he suddenly became dyspneic, cold, and sweaty. His pulse increased to 120 and his blood pressure was unobtainable. For the first time a moderately loud, medium-pitched, systolic murmur was heard at the apex with transmission to the axilla. No thrill was felt. The diagnosis of a ruptured papillary muscle was made at this time. He expired three hours later in circulatory collapse.

On autopsy the posterior papillary muscle showed a complete fresh rupture located 1.5 cm. below the cranial end. The upper portion of the muscle was firm and had a yellowish surface. In the vicinity of the tear the tissue was softer and grayish-

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Cases 1, 2 and 5 are from General Rose Memorial Hospital; Case 3 from Colorado General Hospital; and Case 4 from Porter Sanitarium and Hospital.
The latter case was reported previously with regard to pulmonary changes (E. F. Geever, Karl T. Neuburger and E. K. Rutledge. Atypical pulmonary inflammatory reactions, Dis. Chest, 19:325, 1951).
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pink. The lower portion exhibited accentuated discoloration and softness. It showed sub-endocardial hemorrhage. The upper half of the posterior wall of the left ventricle had a mild aneurysmal bulge. The myocardium in this field showed decreased consistency and yellow to light tan discoloration. There was recent fibrinous hemorrhagic pericarditis over the posterior wall. Old fibrosis was found in the anterior wall. Coronary sclerosis was severe, and the right coronary contained an older thrombus which measured 6 cm. long. The lungs were edematous.

Case 2: E. K., a 61 year old man, was admitted to the hospital on December 12, 1954, with chest pain of 12 hours duration. He had apparently been in good health before this admission. He had been a heavy smoker most of his life and had suffered from chronic cough and some wheezing for several years. On examination the blood pressure was 134/82, and the pulse was 82 and regular. The heart sounds were distinct and a faint apical systolic murmur was heard. The lungs contained numerous wheezes bilaterally. The electrocardiogram showed acute posterior myocardial infarction. The white blood cell count was 12,600 per cu. mm. with 91 per cent polymorphonuclears. The sedimentation rate was moderately elevated. He was placed in an oxygen tent and immediately started on heparin and dicumarol. On the second hospital day the heart rate increased; the wheezing became worse and was associated with some dyspnea. Because of impending failure he was digitized with some improvement. He did fairly well until the morning of the eighth hospital day when he suddenly became quite dyspneic and showed evidence of shock. His condition gradually deteriorated and he died eight hours later. During this time, his chest was so noisy due to wheezes, rhonchi, and moist rales that it was impossible to hear the heart sounds.

The moderately enlarged heart was flabby on autopsy. The epicardial surfaces, especially over the posterior wall of the left ventricle, were covered with shaggy masses of fibrin. There was a sight aneurysmal bulge of the posterior wall. The left posterior papillary muscle was ruptured. A 1.6 cm. gap separated the upper and lower portions which respectively measured 3 and 2 cm. in length. The muscle was soft and yellow-gray. The bulk of the posterior ventricular wall and a small adjacent portion of the septum were similar and mottled with bright yellow patches. Calcific sclerosis, with narrowing of all branches, was found in the coronaries. The right coronary had a near-total occlusion of a 12 mm. segment with constriction of the wall, intimal ulceration and presence of soft brownish thrombotic material. The lungs showed a moderate degree of edema.

Case 3: H. S., a 76 year old butcher, was admitted to the hospital on October 18, 1954, with chest pain of 24 hours duration. He had been treated for basal cell carcinoma of the lips in June, 1954, when an electrocardiogram was normal and no heart murmur was heard. Examination revealed a thin well developed man in acute pain. The blood pressure was 80/70 mm. Hg., and the pulse was 88. The heart was not enlarged. The heart sounds were distant. A low pitched systolic murmur was heard in the aortic and apical regions with maximum intensity at the apex. No thrill was felt. The electrocardiogram showed an acute posterior myocardial infarction. The sedimentation rate was moderately elevated. The white blood cell count was 17,500 with 92 per cent polymorphonuclears.

He was immediately given oxygen, demerol, wyamine, heparin, and dicumarol. He did fairly well for two days but on the third hospital day his blood pressure began to fall in spite of intravenous wyamine. He rapidly went into deep shock and died on the morning of the fourth hospital day.

At autopsy the heart weighed 460 grams. The posterior wall of the left ventricle and the interventricular septum were yellow-red, mottled, softened and flabby. The base of the posterior papillary muscle was torn, ragged, and covered by an adherent blood clot. The proximal fragment of the muscle was entangled in the chordae tendineae. The left coronary artery was almost occluded by calcific plaques. The right coronary artery had numerous plaques and was occluded by a reddish-brown thrombus 4 cm. from its origin. The lungs exuded abundant foamy fluid from the cut surface.

Case 4: N. W., a 63 year old gardener, was admitted to the hospital on September 11, 1947, with severe substernal pain of 24 hours duration. There had been no previous attack. Examination revealed an apprehensive man with moderate severe chest pain. The blood pressure was 150/100 and the pulse was 98 and regular. Respirations were 26 per minute. Medium and fine rales were heard in the upper lung field on both sides. The heart was slightly enlarged and no murmur was heard. The electrocardiogram showed inverted T-waves in Leads I and II and V6 suggesting a lateral infarction, probably acute. The white blood cell count was 13,500, with 84 per cent polymorphonuclears. The sedimentation rate was normal.

He was placed in oxygen and given aminophyllin and morphine. His chest pain decreased in a few days. An electrocardiogram on the fifth hospital day showed much less T-wave inversion. However, the patient developed a viral pneumonia which did not respond to antibiotic treatment. He declined steadily and on the 18th hospital
day suddenly became worse. His blood pressure fell to 100/70 and his pulse became rapid and irregular. A high-pitched systolic murmur of maximum intensity at the apex was noted for the first time. He expired two days later.

At autopsy the moderately enlarged heart showed several small areas of fibrosis and numerous patches of fresh necrosis in the lateral aspect close to the surface. The posterior papillary muscle was completely torn at its base. The latter was covered was a 4 cm. grayish-red thrombus with soft yellow center. The chordae tendinae were twisted, coiled, and entangled. The right coronary artery was essentially free of sclerosis. The left coronary artery showed thickening of the wall with calcification and large, coalescing, yellowish plaques which greatly narrowed the lumen. Thrombosis was not observed. The lungs showed the picture of atypical pneumonia.

**Case 5:** P. K., a 67 year old man, entered the hospital on September 15, 1953, complaining of intermittent substernal chest pain of one week's duration. Examination revealed a well-developed man in no apparent distress. The blood pressure was 120/76 and the pulse was 100. The left border of the heart was percussed at the mid-clavicular line. No thrill was noted. There was a low-pitched, systolic murmur of maximum intensity at the apex. A few rales were heard in the right lung base. The electrocardiogram showed changes indicative of an acute anterolateral infarction. The sedimentation rate was moderately elevated. During hospitalization he had occasional bouts of substernal and left chest pain. He died quietly early in the morning of his fifth hospital day.

Autopsy revealed the distended pericardial sac containing a large amount of fresh-clotted blood. It weighed 800 grams. There was a 15 x 5 mm. rupture of the lateral aspect of the left ventricular wall. The surrounding myocardium was necrotic and hemorrhagic. The anterior papillary muscle was torn completely. Its upper portion was 2.6 cm. long and the lower portion 2 cm. The surfaces facing the tear were ragged and hemorrhagic. The tissue of the muscle was soft and yellow-gray to tan. The left circumflex branch showed severe sclerosis with subtotal obliteration of the lumen but without thrombosis. The other branches showed relatively mild changes.

**Discussion**

**Etiology and Pathogenesis:** In the great majority of patients, rupture of the papillary muscle is a complication of coronary atherosclerosis and myocardial infarction. It occasionally occurs as a complication of other conditions, such as periarteritis nodosa, syphilis, vegetating valvulitis, and trauma (see Table I).

Infarction in the absence of demonstrable thrombosis is usually explained as resulting from a combination of decreased arterial flow due to arterio-

<table>
<thead>
<tr>
<th>TABLE I</th>
<th>CAUSES OF PAPILLARY MUSCLE RUPTURE</th>
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<tbody>
<tr>
<td></td>
<td>Cases</td>
</tr>
<tr>
<td>Myocardial infarction with coronary occlusion</td>
<td>42</td>
</tr>
<tr>
<td>Myocardial infarction without coronary occlusion</td>
<td>5</td>
</tr>
<tr>
<td>Myocardial infarction with normal appearing coronary arteries</td>
<td>1</td>
</tr>
<tr>
<td>Periarteritis nodosa</td>
<td>1</td>
</tr>
<tr>
<td>Syphilis</td>
<td>1</td>
</tr>
<tr>
<td>Probably syphilis</td>
<td>1</td>
</tr>
<tr>
<td>Vegetating valvulitis</td>
<td>2</td>
</tr>
<tr>
<td>Trauma</td>
<td>6</td>
</tr>
<tr>
<td>Unknown</td>
<td>2</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td><strong>61</strong></td>
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sclerosis plus increased oxygen demand by the myocardium, resulting from some factor such as tachycardia; arteriolar and capillary lesions may also be significant (Barboni and Tumiotto).9

Infarction and rupture of the posterior muscle is usually associated with posterior myocardial infarction. Since this type of infarction is variously reported as being from one-half to one-fourth as common as anterior infarction, the posterior muscle would appear to be six to 12 times as susceptible to rupture as the anterior one. The tendency of the posterior muscle to rupture seems to be related to its increased susceptibility to ischemia. Necrosis of this muscle has been demonstrated in 20 per cent of all posterior infarcts,7 and subendocardial lesions seen in coronary insufficiency were found in greater numbers in the same location.1 The vulnerability of the papillary muscles in general to inadequate blood supply has been known for a long time. The frequency of scarring in the anterior muscle was referred to by Mönckeberg in 1924.10 One of us has often seen patches of necrosis and fibrosis in the papillary muscles of young epileptics in correlation with functional disturbances of the circulation and insufficient blood supply to these muscles incident to epileptic attacks and, as a rule, unaccompanied by organic lesions in the arterial tree. The papillary muscles also tend to be involved in carbon monoxide poisoning. The increased susceptibility of the posterior papillary muscle to ischemia may perhaps be explained on the basis of its anatomy and physiology. The blood supply to this area is derived from the right coronary artery11 or from both main arteries.24, 25 The vessels that actually supply the posterior muscle, however, are relatively smaller and more remote from their main source than the vessels to the anterior muscle. The demands of the greatly burdened papillary muscles for steady blood supply are considerable. When there are circulatory disturbances, the predominance of necrosis and rupture in the posterior muscle may be explained on the basis of its greater distance from its blood source.

Clinical Picture: The clinical picture of a patient who has just ruptured a papillary muscle is often quite dramatic. A few days or weeks following myocardial infarction, there is a sudden turn for the worse. Breathing becomes labored, the lungs rapidly are filled with moist rales and rhonchi,
and death frequently follows within a few hours to days or even within a few minutes. Associated with this sudden change in the clinical picture is the appearance of a loud apical systolic murmur which was not present before. The murmur is often of a rough or vibratory nature and is usually heard over the entire precordium, but is loudest at the apex and is transmitted toward the axilla. Murmurs were described in only 62 per cent of the reported cases. Their absence in many instances may be attributed to the suddenness of death or to the presence of loud wheezes and rales which obscure heart sounds. However, Askey¹ has cited patients who were closely examined shortly before death and in whom heart sounds were present but no murmur was heard.

Diastolic murmurs have been observed in a few instances. A “friction rub” has been described in five cases. These, however, have not met all of the criteria of a true pericardial friction rub and autopsies have not disclosed evidence of pericarditis. A thrill associated with the murmur has been reported in only one instance and this was a case of incomplete papillary muscle rupture.³

In the presence of the clinical picture described, electrocardiographic evidence of a posterior myocardial infarction furnishes some evidence pointing to a ruptured posterior papillary muscle. Conduction disturbances in the electrocardiogram are rare.

Diagnosis of a rupture of the papillary muscles depends upon three factors: 1. Presence of heart disease, most commonly posterior myocardial infarction; 2. Sudden appearance of either a new murmur at the apex or a considerable intensification of an old apical murmur; and 3. Sudden change in the clinical course with the rapid onset of intractable pulmonary edema. To date, only seven of the 61 reported cases in the literature have been diagnosed antemortem including one case in this paper.

### TABLE III
SURVIVAL TIME FOLLOWING PAPILLARY MUSCLE RUPTURE

<table>
<thead>
<tr>
<th>Cases</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Sudden death, less than one hour</td>
<td>11</td>
</tr>
<tr>
<td>Less than twenty-four hours</td>
<td>22</td>
</tr>
<tr>
<td>Less than one week</td>
<td>1</td>
</tr>
<tr>
<td>Less than two weeks</td>
<td>4</td>
</tr>
<tr>
<td>Less than two months</td>
<td>4</td>
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<tr>
<td>Four months</td>
<td>1</td>
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<tr>
<td>Six months</td>
<td>1</td>
</tr>
<tr>
<td>Ten months</td>
<td>1</td>
</tr>
<tr>
<td>Twenty months</td>
<td>1</td>
</tr>
<tr>
<td>(Survival unknown)</td>
<td>5</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td>61</td>
</tr>
</tbody>
</table>
Prognosis: Following the rupture of a papillary muscle the outlook is extremely grave. More than 50 per cent of the patients died within the first 24 hours. One-third of them died almost immediately. Less than 20 per cent survived the second week, and only four lived beyond two months (Table III). The only survival beyond one year is Mérat's patient who died 20 months following rupture. In this patient, however, rupture was not due to myocardial infarction.

The cause of death in the majority of these patients is acute left heart failure with intractable pulmonary edema. This is caused by the additional load of severe mitral insufficiency suddenly superimposed upon an already weakened left ventricle. The regurgitated blood markedly increases the left atrial pressure and the blood backs up into the pulmonary circulation. The result is massive pulmonary edema followed shortly by death.\textsuperscript{13} Pulmonary edema was a significant finding in four of our cases. In the fifth, it was difficult to evaluate the amount of edema due to the extensive bronchial pneumonia.

An unusual type of death was presented in our fifth case. While rupture of the heart wall was stated to be the cause of death in 9 per cent of all deaths from acute myocardial infarction,\textsuperscript{14} such rupture coincident with rupture of a papillary muscle must be extremely rare. We have not been able to find any mention of this in the literature.

Differential diagnosis: After myocardial infarction, rupture of a papillary muscle is most likely to be confused with acute cardiac dilatation with mitral insufficiency. The pictures are quite similar and the sudden onset of failure is likely to be considered as the result of an extension of the infarction. With failure the increase in heart size is often progressive rather than abrupt and the murmur increases in intensity over a period of a few days. Similarly the onset of symptoms in failure is likely to be more gradual than in the case of rupture of a papillary muscle. If infarction is posterior, one should suspect a ruptured papillary muscle in any case where there is a sudden deterioration in the clinical picture of the patient.

Perforation of interventricular septum may resemble rupture of a papillary muscle in its clinical appearance on occasion. Both of these complications occur in the first week or two following the infarction. The murmur of the perforated septum, however, is best heard along the left sternal border in the fourth or fifth interspace. The murmur of a ruptured papillary muscle is that of mitral insufficiency and is best heard at the apex. It is transmitted toward the axilla. A systolic thrill over the left sternal border is present in more than half of the septal perforations, while a thrill has been reported only once in papillary muscle rupture. Anterior myocardial infarction is found in 75 per cent of the cases of septal perforation, while posterior infarction is just as common among cases of papillary muscle rupture. Conduction defects, particularly right bundle branch block, are seen in over one-third of the septal perforations and are rare with papillary muscle rupture. The course following rupture of a papillary muscle is usually rapid with intractable pulmonary edema and left heart failure.
Septal perforation may follow a similar pattern, but is more likely to be associated with a gradually developing failure.\textsuperscript{15}

SUMMARY

1. Five cases of papillary muscle ruptured are presented.
2. Rupture of the posterior papillary muscle of the left ventricle is much more common than rupture of the anterior one. The syndrome of simultaneous infarction of the posterior wall of the left ventricle and the posterior papillary muscle with rupture of the latter is described and discussed.
3. Diagnosis of rupture of the papillary muscle should be considered in the presence of acute myocardial infarction, most commonly posterior, when there suddenly develops a loud apical systolic murmur with sudden onset of dyspnea and pulmonary edema.
4. The prognosis is extremely poor.
5. The coincidental occurrence of rupture of the left ventricular wall and of the anterior papillary muscle is reported.

RESUMEN

1. Se presentan cinco casos de ruptura de los músculos papilares.
2. La ruptura del músculo papilar posterior del ventrículo izquierdo es mucho más común que la ruptura del anterior. El síndrome de infarto simultáneo de la pared posterior del ventrículo izquierdo y del músculo papilar posterior con ruptura de éste se describen y se discuten.
3. El diagnóstico de ruptura del músculo papilar debe plantearse en presencia de infarto agudo del miocardio, más a menudo posterior, cuando se presenta de pronto un murmullo sistólico elevado, apical con repentino principio con disnea y edema pulmonar.
5. Se refiere la coincidencia de ruptura de la pared ventricular izquierda y del músculo papilar anterior.

RESUME

1. Les auteurs présentent cinq cas de rupture d'un pilier.
2. La rupture du pilier postérieur du ventricule gauche est plus fréquente que celle du muscle antérieur. Le syndrome d'infarctus simultané de la paroi postérieure du ventricule gauche et du pilier postérieur avec rupture de celui-ci est décrite et discutée.
3. On devrait avoir présent à l'esprit le diagnostic possible de rupture du pilier en présence d'infarctus du myocarde, le plus souvent postérieur, lorsque se développe brusquement un souffle systolique de pointe, avec apparition soudaine de dyspnée et d'oedème pulmonaire.
4. Le pronostic est extrêmement grave.
5. Les auteurs rapportent un cas de rupture de la paroi du ventricule gauche coïncidant avec la rupture du pilier antérieur.
RUPTURE OF PAPILLARY MUSCLES

1. Fünf Fälle von Ruptur eines Papillarmuskels werden vorgestellt.
4. Die Prognose ist äusserst ungünstig.
5. Bericht über gleichzeitiges Auftreten einer Ruptur der Wand des linken Ventrikels und des vorderen Papillarmuskels.

REFERENCES