Mitral obstruction in bacterial endocarditis

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SUMMARY Mitral obstruction due to a large vegetation of acute Streptococcus viridans endocarditis occurred in a 47 year old man. Serial echocardiography documented the rapid growth of the vegetation and its haemodynamic effects. Emergency mitral valve replacement was performed too late to reverse the course of fatal septic shock.

Bacterial endocarditis of the mitral valve usually results in mitral regurgitation. Since 1967 we have been able to trace only seven cases of mitral obstruction as a complication of infective endocarditis in English language reports. We report the preoperative diagnosis and the surgical management of this rare and lethal lesion.

Case report

A 47 year old man was admitted to hospital with a pyrexia of 39°C, with chill and rigor, nausea, anorexia, and dysuria for two weeks. His symptoms had started 24 hours after extraction of a tooth. He had treated himself with dipyrone tablets only. His hypersensitivity to penicillins was recognised six years ago when he was operated on for a lumbar disc lesion. He had no history of rheumatic fever.

Physical examination on admission showed cachexia, a blood pressure of 130/80 mm Hg, and sinus tachycardia of 130 beats/minute. A grade 3/6 diastolic rumble and a grade 1/6 systolic murmur were heard at the apex. The chest radiograph showed mild cardiomegaly with prominence of the right border of the heart. The electrocardiogram confirmed sinus tachycardia with normal axis. There was polymorphonuclear neutrophilic leucocytosis with a total white cell count of 12.0×10^9/l. Urine analysis showed 1–2 white cells and occasional red cells per high power field, and the sample was sterile on culture. Blood cultures were also sterile. An echocardiogram (Fig. 1) showed a mildly stenotic thickened mitral valve with minimal regurgitation and an enlarged left atrium. No valvar or atrial vegetation was seen.

He was initially treated for urinary tract infection with gentamycin, which controlled the fever. An intravenous pyelogram did not provide any additional information. Three weeks later he developed atrial fibrillation, when a second echocardiogram was recorded. It showed severe mitral obstruction with minimal regurgitation due to a huge echogenic mass 2 cm × 4 cm in size, which nearly occluded the mitral orifice. The aortic valve was normal. The left ventricle was of normal size and contracted well (Fig. 1). Blood cultures at this stage grew Streptococcus viridans. The patient developed congestive heart failure. Conservative treatment with vancomycin, digoxin, quinidine, and frusemide for bacterial endocarditis and cardiac failure was started in view of his critical clinical condition. Nevertheless, he did not respond to medical management and gradually became hypotensive and required intubation and mechanical ventilation. Ten days later he was referred for cardiac surgery in a state of septic shock.

At operation a large pericardial effusion was noted and 4000 ml of straw coloured fluid was drained off both pleurae. The heart was enlarged with a very tense main pulmonary artery. A large greyish brown vegetation 2 cm × 4 cm in size was arising from the anterior mitral leaflet and the adjacent left atrial wall near the anterolateral commissure and almost occluded the mitral orifice (Fig. 2). It was removed, and the valve was replaced with a size 31 Björk-Shiley prosthesis. Postoperatively, the septic shock remained uncontrollable despite all measures, and pulmonary hypertension continued at 52/35 mm Hg. Renal failure supervened from the second postoperative day, and he died of septic shock on the fifth day.

Discussion

Mitral obstruction complicating bacterial endocarditis
is very rare and mortality is high. If this case is added to the seven reported cases already mentioned only three of eight (37.5%) patients have survived (Table). Of these eight, five underwent mitral valve replacement. The three survivors\(^5\)\(^6\)\(^8\) were operated on promptly after the diagnosis of endocarditic mitral obstruction had been made by echocardiography or cardiac catheterisation or both. Among the non-survivors one patient\(^7\) was operated on in a rapidly deteriorating haemodynamic state and with congestive failure. He died seven days later from septic shock and a cerebrovascular accident. Our patient was similarly referred in a moribund state to surgery after 30 days in hospital and too long a trial of conservative management. He too succumbed to septic shock five days after the operation. Of the three other reported cases, one was a necropsy finding in a patient who died four months after a normal delivery from a full-term pregnancy.\(^3\) The other two who were treated conservatively had a very rapid downhill course and died after 10 and eight days respectively.\(^4\)\(^9\)

Cerebrovascular events occurred in four patients,\(^3\)\(^5\)\(^7\) possibly due to embolism. In another patient, who died in coma and renal failure, at necropsy bacterial vegetations were also noticed beneath all three aortic cusps.

Echocardiography is important for prompt, accurate, early diagnosis\(^10\)\(^11\) since emergency mitral valve replacement early in the clinical course may become mandatory. It may detect vegetations as small as 2 mm in diameter. Bacterial vegetations mimicking mitral stenosis have been found.\(^12\) In an M mode
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The echocardiogram the posterior mitral leaflet is difficult to differentiate from an echogenic mass obstructing the mitral orifice in later diastole. Nevertheless, dense echoes occurring soon after mitral valve opening would indicate an obstructing echogenic mass rather than valvar mitral stenosis. Left atrial myxoma may also resemble this lesion, but then there will be a mass of echoes from the body of the left atrium continuous with the atrial septum in the cross sectional echocardiogram. A sterile left atrial thrombus is difficult to differentiate by echocardiography from an obstructing vegetation due to mitral endocarditis, but the clinical picture of the latter should help in diagnosis. Our serial study is unique in showing rapid growth of a vegetation so that in three weeks it became a giant mass which occluded the mitral orifice. This finding has not been previously reported. The cross sectional echocardiographic evidence was diagnostic and correlated with the operative findings. Nevertheless, contrary to our finding, Sheikh et al. reported little or no change in the echocardiographic size of vegetations in 27 patients over a period of six weeks unless systemic embolisation occurred.13

Two types of vegetations are found in the mitral valve: (a) a globular or elongated mass as seen in five cases and (b) multiple verrucae occluding the mitral orifice as seen in two. The vegetations showed no predilection for site in the mitral apparatus. The posterior leaflet was the site in two patients, whereas the anterior leaflet was the primary area in three cases. The site of the vegetation was not mentioned in two cases. Signs of earlier rheumatic disease were noted in six.

In conclusion, mitral obstruction due to bacterial endocarditis is a dangerous complication with a rapidly deteriorating course often causing cerebrovascular accidents. Prompt diagnosis with M mode and cross sectional echocardiography alone should lead to emergency mitral valve replacement. Mitral valve replacement in bacterial endocarditis has usually been advocated in congestive heart failure due to mitral regurgitation or systemic embolisation.14 We consider that the documentation of the rare possibility of mitral obstruction by vegetations should also indicate valve replacement early in the clinical course. Cardiac catheterisation is unnecessary,11 and may increase the risk of embolism. Delayed surgical intervention may not be adequate to reverse the course of fatal septic shock.

References


Table  Previously reported cases of mitral obstruction in bacterial endocarditis

<table>
<thead>
<tr>
<th>Author and year</th>
<th>Age/sex</th>
<th>Signs of rheumatic disease</th>
<th>Echocardiography or cardiac catheterisation or both</th>
<th>Surgery/necropsy findings</th>
<th>Organism</th>
<th>Treatment</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Roberts et al13</td>
<td>21/F</td>
<td>Present</td>
<td>Not done</td>
<td>Vegetations from LA filled MV orifice</td>
<td>β Haemolytic streptococcus</td>
<td>MVR (porcine xenograft)</td>
<td>Died after 1 mth</td>
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<tr>
<td>1967</td>
<td></td>
<td></td>
<td></td>
<td>Multiple vegetations on AML, aortic cusps involved</td>
<td>Staphylococcus aureus</td>
<td></td>
<td>Died after 10 days</td>
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<tr>
<td>Reeve et al2</td>
<td>36/M</td>
<td>Present</td>
<td>Not done</td>
<td>A large vegetation occluding MV orifice</td>
<td>Staphylococcus aureus</td>
<td>MVR (porcine xenograft)</td>
<td>Survived</td>
</tr>
<tr>
<td>1974</td>
<td></td>
<td></td>
<td></td>
<td>Multiple obstructive vegetations, each 2 mm x 3 mm; MV orifice 1-5 cm²</td>
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<tr>
<td>Matula et al54</td>
<td>51/M</td>
<td>Present</td>
<td>Multiple dense mitral echoes; EF slope less than 10 mm/s; RV enlarged; catheterisation confirmed MS; severe PH</td>
<td>Soft brownish globular mass from PML, 4 cm in diameter</td>
<td>β Haemolytic streptococcus</td>
<td>MVR (porcine xenograft)</td>
<td>Died after 7 days from CVA, septic shock</td>
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<td>1975</td>
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<td>Survived</td>
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<tr>
<td>Copeland et al14</td>
<td>52/F</td>
<td>Present</td>
<td>Decreased motion of AML; multiple adjacent echoes from LA aspect of AML; catheterisation confirmed MS</td>
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<tr>
<td>1979</td>
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<tr>
<td>Alam et al7</td>
<td>65/F</td>
<td>Absent</td>
<td>Reduced EF slope; increased mass of echoes in latter 3/4 of diastole behind AML; CSE showed obstructing mass in diastole adherent to MV in systole; no fibrinous continuity with septum</td>
<td></td>
<td></td>
<td></td>
<td>Died after 7 days from CVA, septic shock</td>
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<td>1979</td>
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<td></td>
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<td>Survived</td>
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<tr>
<td>Waller et al14</td>
<td>37/M</td>
<td>Present</td>
<td>Typical MS in M mode echo; catheterisation showed mean diastolic mitral gradient of 26 mm Hg</td>
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<td></td>
<td></td>
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<tr>
<td>1982</td>
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<tr>
<td>Grenadier et al10</td>
<td>40/M</td>
<td>Absent</td>
<td>Giant globular mass from PML; several abscesses in myocardium</td>
<td></td>
<td></td>
<td></td>
<td>Died suddenly after 8 days</td>
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<tr>
<td>1983</td>
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<tr>
<td>Ghosh et al1</td>
<td>42/M</td>
<td>Present</td>
<td>Brown large mass 2 cm x 4 cm from AML and LA wall at ALC</td>
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<td></td>
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<td>Died after 5 days from septic shock</td>
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<td>(present report)</td>
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MV, mitral valve; AML, anterior mitral leaflet; LA, left atrium; PML, posterior mitral leaflet; RV, right ventricle, MVR, mitral valve replacement; CSE, cross sectional echocardiography; MR, mitral regurgitation; MS, mitral stenosis; ALC, anterolateral commissure; PH, pulmonary hypertension.
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