Management of prosthetic valve endocarditis: a clinical challenge

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The decision over whether to treat prosthetic valve endocarditis medically or surgically continues to challenge clinicians

Prosthetic valve endocarditis (PVE) is a very serious disease. PVE is usually classified as early PVE (that is, acquired perioperatively), and late PVE (resulting from infections unrelated to the valve operation). Early PVE has a much worse prognosis than late PVE. Differences in infective organisms and in the clinical setting explain the dismal prognosis in early PVE and the almost universal need for surgical treatment in this type of PVE. PVE is an uncommon disease, but its frequency seems to be increasing, ranging from 0.1–2.3 per patient-year. This increased frequency occurs in late cases because preoperative antibiotic prophylaxis and improved manipulation both during the operation and in the postoperative period have significantly contributed to a decrease in the number of early PVE cases.

Transoesophageal echocardiography is an extremely useful diagnostic tool in PVE because it allows early recognition of vegetations that could not be visualised by transthoracic echocardiography; it is also the best tool to investigate periprosthetic damage and prosthetic dysfunction. Improvements in surgical techniques have also been reported in recent years. However, even if these diagnostic and therapeutic advances have occurred they do not seem to have had a dramatic impact on the prognosis of the disease. Reported mortality rates are still quite high and have not shown significant improvements in recent years. Also the most appropriate treatment approach, either medical or surgical, is under discussion.

Retrospective analysis

In this issue of Heart, Akowuah and colleagues present a retrospective analysis of 66 patients with PVE, mainly late cases, treated at a single institution in the 1990s. In this study overall mortality was 33%, 46% from the medical group and 24% from the surgical group. The authors considered that seven patients from the medical group were surgical candidates, but surgery was not attempted because they were too sick to have an operation. Therefore they conclude that mortality in those patients in whom medical treatment was chosen was 29%, and therefore similar to the mortality from the surgical group. These results compare with a recent paper from our group on 59 patients with late PVE: overall mortality was 25% and 42 patients (70% of the series) were treated by antibiotics alone. In our study mortality did not differ between medical and surgical patients (24% and 29%, respectively). However, during follow up a high percentage of medically treated patients needed an operation because of progressive prosthetic dysfunction. One should not be greatly impressed by comparing these rates: crude comparisons of mortality rates between medically and surgically treated patients are meaningless as there are wide differences in clinical features and prognosis between these patient groups.

Another remarkable aspect of the study by Akowuah and colleagues is that in their series antibiotics were given intravenously only for two weeks and were followed by oral antibiotics for a total of at least six weeks; most guidelines recommend 4–6 weeks of intravenous antibiotics in patients with PVE.

The present study confirms that there is still not a universally accepted treatment for PVE. Although most series suggest that surgical treatment has better results than medical treatment alone, it is very difficult to select the most appropriate treatment for the individual patient. PVE is a very complex disease. Its outcome is influenced not only by the disease itself—that is, the infective organism, vegetation size, degree of periprosthetic damage, degree of prosthetic dysfunction, presence of endocarditis complications such embolism or heart failure—but also by individual characteristics of the patient such as age, number of previous heart operations, state of left ventricular function, and other non-cardiac morbidities that can determine the clinical outcome and surgical results as much as the endocarditis itself. Therefore the decision whether or not to operate on a patient with PVE is not an easy one, and all these factors should be carefully considered.

Evidence for surgery

The present study, and others from the literature, confirm that there is no clear evidence that all patients with PVE should be treated surgically on a systematic basis. Nevertheless, in all cases of PVE surgical treatment has to be considered very early, at the time of diagnosis, and both the cardiologist and the surgeon should be involved in the therapeutic decision. It is likely that a majority of patients with PVE will eventually benefit from early or delayed surgery.

All patients with PVE should be evaluated by transoesophageal echocardiography as soon as they reach the hospital. In cases with very aggressive organisms (mostly non-streptococcal)
or who fail to respond immediately to antibiotics, and cases with large periprosthetic leaks or abscesses, surgical treatment should be considered on an urgent basis—provided the judicious assessment the clinical condition of the patient (age, comorbidities) favours this therapeutic option. It must be emphasised that the decision to operate, and the operation itself, should not be delayed. Most surgeons would agree that surgical mortality relates to the amount of anatomical damage, and it is well known that anatomical destruction relates both to the aggressiveness of the microorganism and the duration of the disease. Surgeons claim that patients should be sent to them earlier.

However, in selected cases cure by medical treatment can be achieved. Patients in whom the diagnosis has been made very early in the course of the disease, patients with streptococcal disease or with prompt antibiotic response, and patients with reassuring echocardiographic findings such small or absent vegetations, no periprosthetic damage, and no severe prosthetic dysfunction are candidates for conservative management. In these cases considered for medical treatment the clinical course should be carefully monitored and reconsideration of the surgical indication performed if any complication arises. Therefore, only in patients at both ends of the prognostic spectrum (those with specific findings of less severe disease and those too ill to undergo an operation) is medical treatment likely to be the best choice. Furthermore, even if cure is achieved by medical treatment, close follow up is mandatory, to rule out progressive prosthetic dysfunction.

**REFERENCES**


**IMAGES IN CARDIOLOGY**

**Misplaced pacemaker wire as a cause of mitral regurgitation**

In 1990, a 73 year old woman received a VVI pacemaker for second degree heart block. The patient first returned to the pacemaker clinic in 1996 for a routine evaluation. A loud systolic murmur (III/IV) was noticed at the lower left parasternal border. The clinical diagnosis was presumed to be tricuspid regurgitation caused by the cardiac pacemaker. The patient declined further investigation. She remained asymptomatic but finally consented in September 2001 to an echocardiogram. The four chamber view is shown below (left). This shows the pacemaker wire crossing the foramen ovale and lodging into the mitral valve apparatus. This has produced a pronounced inflammatory/sclerotic response at the level of the mitral valve which was associated with severe mitral regurgitation (estimated effective regurgitant orifice 0.6 cm²). The regurgitant jet was directed along the pacemaker wire medially. There was also moderate tricuspid regurgitation with a pulmonary artery pressure of 90 mm Hg.

In retrospect, the diagnosis of a misplaced pacemaker wire could have been suspected earlier as the paced ECG has a right bundle branch pattern (below right). So far, the patient has refused further investigations and treatment of these conditions.

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Misplaced pacemaker wire as a cause of mitral regurgitation

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