Arthritis and Meningitis

— The First Manifestations of Bacterial Endocarditis in 2 Patients —

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We have encountered 2 patients in whom the first manifestations of bacterial endocarditis were arthritis (in 1 case septic arthritis and in the other nonseptic arthritis) and bacterial meningitis. These presentations were followed by acute heart failure due to aortic valve destruction, although the patients showed no significant cardiovascular manifestations on admission. Aortic valve replacement was performed in each case and the patients' postoperative course was comfortable. We would like to emphasize the following points. (1) Arthritis and meningitis are uncommon in patients with bacterial endocarditis. However, it is necessary to consider the possibility of bacterial endocarditis when these clinical manifestations present together. Such a combination can cause rapid valve destruction. When more than 2 rare complications of bacterial endocarditis coexist, surgery should be considered as soon as the definite diagnosis of bacterial endocarditis is established, even if congestive heart failure has not yet developed. (2) Arthritis associated with bacterial endocarditis might be truly septic rather than mediated by circulating immune complexes as is commonly believed.

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Nonspecific musculoskeletal complaints, such as myalgia and arthralgia, are common in patients with bacterial endocarditis, but arthritis is relatively rare (occurring in about 13% of cases)^{1,2}. Neurologic complications, mainly stroke or encephalopathy secondary to septic cerebral embolism, occur in around one-third of cases^{3,4}. Meningitis is the first manifestation in fewer than 5% of cases^{3,4}. Until now there has been no case report of bacterial endocarditis accompanied both by septic or nonseptic arthritis and by meningitis as the first manifestations. We have encountered 2 patients with acute bacterial endocarditis first manifested by arthritis and meningitis, and followed by acute heart failure due to aortic valve destruction within a month. They were treated with aortic valve replacement in the acute phase of heart failure.

Case Reports

Case 1

A 65-year-old man was admitted to our hospital on 9 May 1995 because of chills associated with high fever and arthralgia in the right shoulder, the left wrist, and the right ankle joints. On admission no cardiovascular sign was detected. Blood culture yielded group B streptococcus. Carbapenem (1.0 g×2/day iv) and piperacillin (2 g×3/day iv) were administered. A week after admission the patient became confused, he developed a rigid neck and his cerebrospinal fluid was purulent. Group B streptococcus could again be cultured from the cerebrospinal fluid and from synovial fluid aspirated from the left wrist joint.

The arthritis and the meningitis gradually resolved with antibiotic treatment (Fig 1). However, on the 24th hospital day, severe congestive heart failure suddenly occurred. The patient's heart rate was 130/min,

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Case 1

Fig 1. Clinical course in case 1. Although the inflammatory findings gradually resolved, acute heart failure developed on the 24th hospital day.

Fig 2. Chest radiograph from case 1, taken in the sitting posture at development of heart failure. Pulmonary edema is apparent. The cardiothoracic ratio is 63.3%.

his respiratory rate 40/min and his blood pressure 160/60 mmHg. S3 gallop was audible but heart murmur was not. Chest radiography revealed cardiomegaly and acute pulmonary edema (Fig 2). Arterial blood gas analysis yielded values of pH 7.40, PaCO₂ 28.5 mmHg, PaO₂ 54.1 mmHg, HCO₃⁻ 17.5 mmol/L and base excess of -5.4 mmol/L with the patient breathing

10 L/min oxygen through a face mask. Heart failure was barely controlled with medical treatment, including aggressive furosemide iv and dopamine iv.

On the next day a to-and-fro murmur (grade 2/6) was audible at the heart base. A mobile vegetation attached to the right coronary cusp and diastolic buckling of the cusp were detected on echocardiography (Fig 3). The diameter of the vegetation was 12 mm. Aortic regurgitation was of grade 4/4, and was accompanied by mitral valve flutter. The ejection fraction was 61%. The left ventricle was dilated (internal diameter 60.9 mm in diastole and 40.4 mm in systole). Cardiac output was 4.88 L/min. Pulmonary arterial pressure (PA) was 32/17 (mean 27) mmHg and pulmonary arterial wedge pressure (PAWP) was 17 mmHg on right cardiac catheterization. The aortic valve was replaced by a Karbo-Medics 25A valve 3 days later. Severe destruction of the right coronary cusp with vegetation (15 mm in diameter) and a perforated non-coronary cusp were observed during surgery. Penicillin G (8,000,000 U×3/day iv) and gentamicin (40 mg×2/day im) were administered for 2 weeks after surgery. The postoperative course was uneventful.

Case 2

A 67-year-old man developed high fever (39.0°C) and arthritis in the metacarpophalangeal joint of the left index finger and the left knee joint and diffuse cellulitis at the left thigh 3 days before admission. The following day he developed gait disturbance and

Fig 3. Echocardiogram from case 1. A mobile vegetation 12 mm in diameter is attached to the right coronary cusp and diastolic buckling of the cusp (arrow) is visible. Aortic regurgitation was of grade 4/4.

Case 2

Fig 4. Clinical course in case 2. The inflammatory findings resolved on administration of antibiotics. Nevertheless, acute heart failure still developed.

became stuporous. Aphasia and hearing difficulty developed progressively. He was admitted to our hospital on 30 May 1995. Blood culture and spinal tap were performed and *Streptococcus pneumoniae* could be cultured from both specimens. Carbapenem ($0.5 \text{ g} \times 4/\text{day}$ iv) and aminobenzyl penicillin ($2 \text{ g} \times 4/\text{day}$ iv)

treatment was instituted immediately. A to-and-fro murmur (grade 2/6) was audible on the 9th hospital day. Moreover, echocardiography showed a high-echoic lesion of 6 mm in diameter at the left coronary cusp suggestive of vegetation and aortic regurgitation (grade 2/4). Our experience from case 1 led us easily

to conceive of a diagnosis of bacterial endocarditis. Antibiotics were then replaced by high-dose penicillin G (6,000,000 U $\times$ 4/day iv). Joint swelling and pain and nuchal rigidity gradually subsided (Fig 4). The patient's level of consciousness also improved; however, he developed dyspnea on the 30th hospital day. His temperature was 37.9°C, and his pulse was regular at a rate of 82/min. The patient's respiratory rate was 28/min and blood pressure 144/80 mmHg. Chest radiography revealed cardiomegaly (cardiothoracic ratio 63%) and acute pulmonary edema (Fig 5). The

Fig 5. Chest radiograph from case 2, taken in the sitting posture at the time of heart failure. The stag-horn sign and bilateral pleural effusion can be detected.

grade of aortic regurgitation was 4/4 on echocardiography (Fig 6). Right cardiac catheterization revealed postcapillary pulmonary hypertension [PA 40/22 (mean 30) mmHg, mean PAWP 20 mmHg]. Cardiac output was 5.3 L/min. Surgery was performed 2 days later. The aortic valve was replaced by Karbo-Medics 25A. After surgery, penicillin G was administered for 15 days (6,000,000 U $\times$ 2/day iv), and the patient's postoperative course was comfortable except for deafness.

Discussion

These 2 cases are similar in some respects. First, in each case the initial manifestation was high fever accompanied by acute arthritis on admission, followed by meningitis a few days later. Secondly, the same organism (group B streptococcus in case 1, *Streptococcus pneumoniae* in case 2) was cultured from blood and cerebrospinal fluid. Thirdly, neither patient showed any significant cardiovascular manifestations on admission, but developed congestive heart failure due to bacterial endocarditis about 1 month later when the arthritis and meningitis were almost under control. Finally, the aortic valves were destroyed in both patients and aortic valve replacement was performed in the acute phase of heart failure, followed by an uneventful course.

Neither patient exhibited any cardiovascular manifestations on admission, but we cannot rule out the possibility that they were suffering from subclinical, degenerative valvular defects that could act as a nidus for initiation of endocarditis. Even if minor valvular defects were absent, it is generally known that both *Streptococcus pneumoniae* and group B streptococci

Fig 6. Echocardiogram from case 2. A high-echoic lesion on the aortic valve is suggestive of vegetation (arrow). The grade of aortic regurgitation is 4/4.

can attack normal valves and cause widespread metastatic foci. *Streptococcus pneumoniae*, in particular, has a predilection for the aortic valve, and infection with this organism runs a fulminant course with rapid valvular destruction. Our 2 patients exhibited no signs of heart murmur early in their clinical course, although both showed signs of 2 serious complications. It is possible that in some circumstances the clinical course might not parallel the ability of micro-organisms to cause valve destruction. In our 2 patients arthritis and meningitis may have developed before cardiovascular manifestations became significant.

It is relatively rare for bacterial endocarditis to be accompanied by arthritis or meningitis. To our knowledge, there has been no case report of bacterial endocarditis preceded by arthritis and meningitis in the English language medical literature. We emphasize that bacterial endocarditis must be considered when these 2 manifestations coexist, and that, if complicated by bacterial endocarditis, severe heart failure may rapidly develop despite the usual antibiotic therapy⁵.

Valve replacement is not without problems (eg emboli, bleeding from anticoagulation, valve degeneration, prosthetic valve endocarditis). However, mortality from endocarditis with moderate to severe heart failure in patients with aortic regurgitation is extremely high. Immediate valve replacement is essential. Any delay to surgery, especially in the presence of heart failure, may convert an elective procedure with a relatively low mortality rate into an emergency procedure with a high mortality rate. When more than 2 rare complications of bacterial endocarditis coexist, surgery should be considered as soon as the definite diagnosis of bacterial endocarditis is established, even if congestive heart failure has not

yet developed.

It is commonly considered that arthritis associated with bacterial endocarditis is due to circulating immune complexes as Churchill et al¹ and Myers and Commerford² reported synovial fluid to be sterile. In one of our patients, arthritis was truly septic. Thus, the principal cause of arthritis associated with bacterial endocarditis might be sepsis rather than immune complexes⁶⁻¹⁰.

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