Nocardial endocarditis of an aortic valve prosthesis

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SUMMARY The organism responsible for endocarditis of a prosthetic aortic valve was identified as Nocardia asteroides. The patient was treated with intravenous amikacin (250 mg four times a day) and intravenous imipenem (1·5 g four times a day). The valve was replaced under this new antibiotic regimen. This is the first report of survival after prosthetic valve nocardiosis.

Two cases of endocarditis of a prosthetic valve resulting from Nocardia asteroides infections have been reported. Both patients died before specific treatment could be established. We report a case of nocardial endocarditis that followed aortic valve replacement.

Case report

A 61-year-old man with severe calcific aortic valve disease was given a Medtronic Hall valve prosthesis in March 1984. Fever developed after operation and he was treated with the antibiotic cefadroxil because an exacerbation of chronic emphysematous bronchitis was suspected. The patient was transferred to the medical department 21 days after operation. Physical examination was normal and the prosthetic heart sounds were clear. A chest X-ray indicated emphysema without pulmonary infiltrates; the heart was of normal size. Abdominal sonography and cholangiography showed small stones in the gall bladder and in the cystic duct. The erythrocyte sedimentation rate (56 mm per hour), the neutrophil count (5·7 × 10⁹/l) (with an increased proportion of unsegmented neutrophils (11%)), serum iron (<25 µg/dl), and copper (240 µg/dl) were compatible with a systemic infection. When antibiotic treatment was withdrawn the patient’s oral temperature rose to 40°C. After two weeks of incubation at 37°C blood cultures showed growth of a Gram positive filamentous micro-organism. This organism was identified as biovar B of the Nocardia asteroides complex or Nocardia farcinica. Sputum cultures were always negative for nocardiae and urine cultures contained no micro-organisms.

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TREATMENT

Intravenous treatment with amoxycillin (2 g three times a day) was started followed by a combination of mezlocillin (5 g three times a day) and tobramycin (80 mg twice a day) because blood cultures remained negative for two weeks. When Nocardia asteroides was identified as the aetiological agent treatment with sulphadiazine 12 g a day was started. But the dose of 24 tablets per day produced unacceptable gastrointestinal side effects. Intravenous treatment with amikacin and a combination of amoxycillin plus clavulanic acid (Augmentin) was started at dosages of 250 mg two to four times a day and 5·5 g three times a day (5 g amoxycillin + 0·5 g clavulanic acid) respectively. In vitro susceptibility tests showed that the Nocardia strain was sensitive to amikacin at a minimal inhibitory concentration (MIC) of 3 µg/ml and moderately sensitive to amoxycillin plus clavulanic acid (MIC 8–16 µg/ml) and the thiamycin derivative, imipenem (MIC 8 µg/ml). Oral dosages of imipenem (1·5 g four times a day) and amikacin (500 mg four times a day) were chosen to maintain the bactericidal concentrations in the serum that were obtained during infusion of both drugs. This regimen reduced fever to 37–38°C and reduced the proportion of unsegmented neutrophils.

Six weeks after admission, moderate signs of cholangitis were seen and a cholecystectomy was performed in the eighth week. Cultures from the removed gall bladder, the bile, and blood were negative. Body temperature declined and remained normal for four weeks and the patient was discharged on the thirteenth postoperative day without antibiotic treatment.

The patient was readmitted to the medical department on 30 July 1984 because of recurrent septic fever. Blood cultures again produced Nocardia asteroides (N farcinica) which was still susceptible.
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ceptible to amikacin, imipenem, and co-trimoxazole in vitro. Clinical investigation, especially prosthetic valve sounds and laboratory tests including chest x-ray, bronchoscopy, abdominal sonography, and bone marrow histology did not indicate any systemic malignant disease. On the second day of amikacin treatment (500 mg four times a day) and imipenem (1 g four times a day) fever levelled off and remained at 36.5–37.5°C. The patient was transferred to the department of surgery. At operation an intact aortic valve prosthesis was found but there was an aneurysm of the non-coronary sinus of Valsalva. Cultures from the removed valve produced Nocardia asteroides (Nfaricinica). The sinus of Valsalva and the base for the valve were locally disinfected and a Medtronic Hall aortic valve prosthesis was implanted. Postoperative recovery was good. Amikacin had to be withdrawn because of rapid deterioration of hearing. Treatment with imipenem (1 g four times a day) was continued for three weeks after operation. Then the patient was treated with oral trimethoprim and sulphamethoxazole (160 and 800 mg twice a day respectively). Before discharge transmission computed tomography and scintigraphy with indium-111 labelled leucocytes did not detect any cranial abscesses. The patient remains well without any laboratory evidence of infectious disease.

Discussion

Nocardiosis is a rare disease with an unfavourable prognosis which occurs predominantly in severely compromised patients with systemic malignant disease or in those on immunsuppressive treatment. So far two cases of nocardiosis of prosthetic valves have been reported. Vlachakis and associates reported the death of a 34 year old women four months after mitral valve replacement. Falk and coworkers reported the death of a 64 year old woman from nocardial endocarditis six months after replacement of an aortic valve. Both patients had multiple abscesses and in the absence of other sites of entry the lungs were the most likely portal for systemic nocardiosis. In our patient body temperature declined to normal immediately after cholecystectomy; however, an infectious agent was not cultured from the resected gall bladder and cholangitis or cholecystitis have not been reported as primary manifestations of nocardiosis. The possibility of false negative cultures must be considered; however, the mode of infection remains unclear. Possibilities include intraoperative infection and activation by the operation of an infection that was acquired preoperatively.

We thank Professor Gerhard Schroeter, Institute of Hygiene and Professor Olaf Elert, Department of Thoracic and Cardiovascular Surgery, University of Würzburg for their cooperation.
References