azoles [14] have been shown to be synergistic against *Scedosporium* spp. Local therapy alone has been shown to be ineffective [5]. This particular antifungal susceptibility pattern makes prompt diagnosis of great importance, since immunocompromised hosts may present a rapidly deteriorating clinical course without appropriate therapy [15]. The optimal duration of therapy remains to be defined, although most authors have given antifungal drugs for a minimum of 3–4 weeks, associated with surgical debridement when indicated. Shorter courses have been associated with relapses [3].

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Pseudomonas mendocina **as a cause of chronic infective endocarditis in a patient with situs inversus**

Pseudomonas mendocina occurs in water and soil, like other pseudomonads [1,2], but is rarely recovered as a human pathogen. Its pathogenic role was first documented less than one decade ago as the infective agent causing mitral valve endocarditis in a 63-year-old man from Argentina [3]. We have recently isolated *P. mendocina* from three blood cultures from a woman with a tricuspid endocarditis [4], possibly lasting several years.

A 28-year-old woman with situs inversus was admitted to hospital in September 1999 because of abdominal pain, dyspnea, intermittent influenza like-symptoms, tricuspid stenosis and suspected endocarditis.

She had had three operations in 1979, 1982 and 1983 because of double-outlet right ventricle, ventricular septal defect (VSD) and pulmonary stenosis. An intraventricular baffle conducting blood from the left ventricle to the aorta was created. The VSD was closed by a Dacron patch, and the pulmonary cusps were resected.

In 1994, the patient was admitted to hospital because of 1 week of intermittent fever and suspected endocarditis. No signs of endocarditis could be found by echocardiogram at that time. However, she had an elevated hypersedimentation rate of 48 mm/h and elevated C-reactive protein of 900 mg/L. The antibody titers against staphylococci, streptococci or small Gram-negative rods, which often cause endocarditis, were all within the normal range.

After admission to hospital in September 1999, three sets of blood cultures were obtained and treatment with penicillin and gentamicin was begun because of suspected endocarditis. A transthoracic echocardiogram revealed significant tricuspid stenosis and fluttering vegetations on the systolic and the anterior tricuspid valves. After 48 h, Gram-negative bacilli were grown in all blood cultures and were identified as *P. mendocina*, based on biochemical reactions discussed previously [2,3,5]. The therapy was subsequently changed to ampicillin and gentamicin, due to

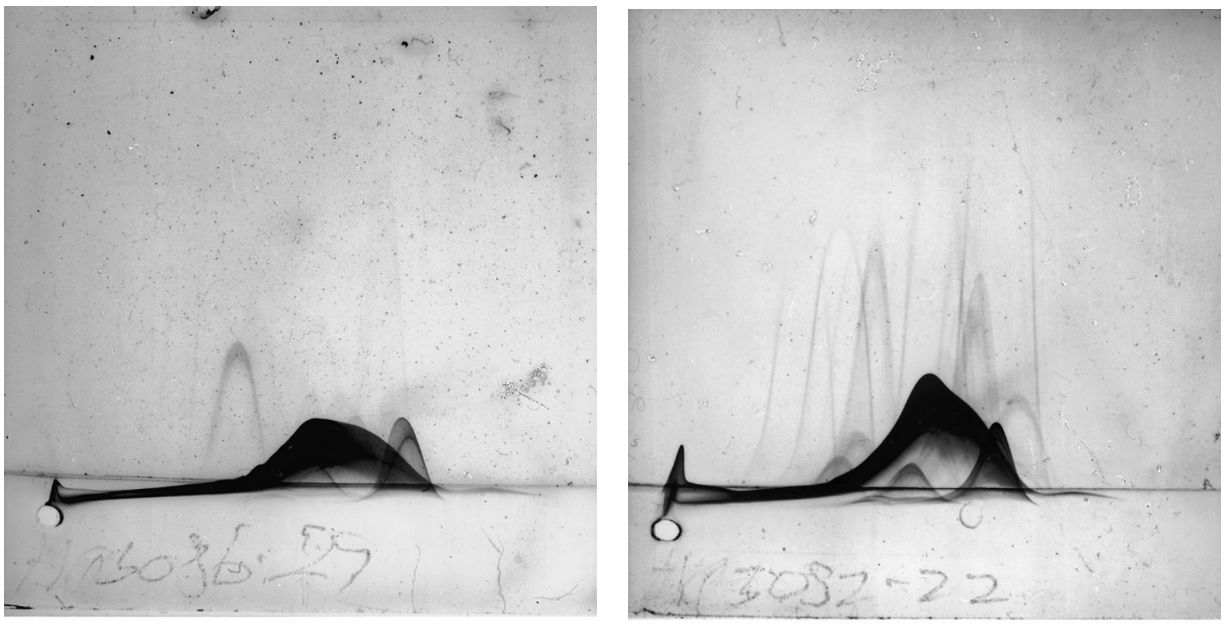
lower MIC, and to ciprofloxacin, due to signs of drug allergy. After 7 weeks, the patient was discharged from the hospital in healthy condition.

In February 2000, catheterisation of the left side of the heart revealed significant tricuspid stenosis; clinically, the patient developed increasing right-sided heart failure and was readmitted to hospital for heart surgery. The Dacron patch was removed, as an abscess cavity was revealed surrounding the patch. No bacteria were cultured. A thin VSD membrane was left intact. A pulmonary homograft was implanted, and a tricuspid valve repair was performed. Subsequently, the patient was treated successfully with meropenem and ciprofloxacin for 6 weeks.

In our laboratory, as elsewhere, serodiagnosis of endocarditis has been developed [6–9]. A blood sample from the patient from 1994 and one from 1999 were investigated for precipitating antibodies against the patient's own *P. mendocina* isolate (water-soluble antigens obtained by sonication) used as antigen by crossed immunoelectrophoresis (XIE) (normal: 0–1 precipitates, variation coefficient 11%, significant increase $\geq$20% increase of number of precipitating antibodies) [10,11]. She had 16 precipitating antibodies in 1994 and 24 in 1999 (Figure 1), whereas no significant antibody response was found against other bacteria causing endocarditis. It is therefore possible that she had been suffering from tricuspid endocarditis or an abscess caused by *P. mendocina* for several years.

The MICs of the *P. mendocina* isolate were meropenem 0.125 mg/L, ciprofloxacin 0.023 mg/L, ampicillin 1.0 mg/L and gentamicin 2.0 mg/L by the E test (AB Biodisk, Solna, Sweden). The isolate did not produce β-lactamase (tested with nitrocefin disks, Cefinase, BBL Microbiology Systems, Cockeysville, MD, USA).

This seems to be the second case in which *P. mendocina* has been isolated from human blood cultures [3]. All the clinical, serologic, echocardiographic and microbiological evidence suggests that our patient's endocarditis was also caused by *P. mendocina*. During the cardiac operation, an abscess cavity was discovered around the ventricle septum patch. Unfortunately, we were not able to culture any bacteria from the samples which were removed during the operation. This is probably due to the antibiotic treatment of the endocarditis given 4 months previously. We can only speculate that *P. mendocina* could have been introduced during or after one of the cardiac operations performed in 1979, 1982 or 1983, or maybe accidentally via the bloodstream before the 1994 episode, and formed a silent biofilm on the Dacron patch. This would be in accordance with the prolonged development of the antibody response (Figure 1).



(a) (b)

Figure 1 Crossed immunoelectrophoresis of 2 μL of sonicated water-soluble antigens from the patient's own *P. mendocina* isolate against 300 μL of serum from the patient with tricuspid valve endocarditis. Size of the plate 5 × 5 cm. (1) Dimension electrophoresis, anode to the right. (2) Dimension electrophoresis, anode to the top. (a) 16 precipitins from 1994. (b) 24 precipitins from 1999. Normal: 0–1 precipitates.

In the first published case, *P. mendocina* was also of low virulence and low pathogenicity, since it was treated without any sequelae [3].

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