The world’s first case of *Serratia liquefaciens* intravascular catheter-related suppurative thrombophlebitis and native valve endocarditis

*Serratia liquefaciens* is an organism rarely encountered in clinical practice. It belongs to the genus *Serratia* and the family Enterobacteriaceae [1]. It is widely distributed in nature, including river water [2], mineral, spring and table water [3], domestic sewage [4], fish, minced meat and pasteurized milk or cream [5]. It has been reported as a cause of mastitis in a dairy herd [6]. In humans, it has rarely been reported as a cause of nosocomial infections, including urinary tract infection [7], pneumonia [8], post-sternotomy mediastinitis [9], neonatal meningitis [10] and septicemia resulting from transfusion of contaminated blood products [11]. A few reports of *S. liquefaciens* causing community-acquired infections include peritonitis associated with continuous ambulatory peritoneal dialysis [12], pustulosis and costochondritis in heroin addicts [13], fistulous pyoderma [14], septic arthritis [15] and ocular infections resulting from contamination of contact lens cases [16].

I recently encountered a patient who developed an intravascular central catheter-related suppurative thrombophlebitis and endocarditis due to *S. liquefaciens*. He was a 55-year-old man who had developed short gut syndrome as a complication of chronic pancreatitis and multiple bowel resections. He had been dependent on parenteral nutrition for the preceding 6 months. He presented with fever, malaise and pain in the right side of the neck, along the tunnel of the indwelling intravenous central catheter. On physical examination, he had no heart murmur or peripheral signs of endocarditis. Four blood cultures grew *S. liquefaciens* within 48 h of inoculation. Duplex ultrasound revealed an occlusive clot in the right internal jugular vein surrounding the central venous catheter. Transosophageal echocardiography showed a mobile thrombus originating in the superior vena cava extending into the right atrium, measuring 5.2 x 0.9 cm, and all heart valves appeared normal. After a therapeutic anticoagulation was achieved, the central venous catheter was removed. He subsequently underwent resection of the right atrial mass, which on histological examination was confirmed to be a thrombus with secondary suppurative endocarditis. The organism was susceptible to multiple antibiotics, including piperacillin, trimethoprim-sulfamethoxasole, third-generation cephalosporins, ciprofloxacin and imipenem-cilastatin. He was treated with ceftriaxone, 2 g daily for 4 weeks, for ease of administration with concomitant parenteral nutrition.

To date, there have been no prior reports of suppurative thrombophlebitis or endocarditis caused by *S. liquefaciens* in the world’s literature. This organism is differentiated from *S. marcescens* by its relatively weak phospholipase activity [17] and the lack of fermentation of L-arabinose by the latter [1]. Antimicrobial susceptibility is also somewhat different, with *S. liquefaciens* being more resistant to aztreonam, ceftazidime and amikacin than *S. marcescens* [18].

Endocarditis caused by *S. marcescens* is seen most commonly in intravenous drug users and in one report it caused 14% of all addict-associated endocarditis [19]. Among these patients, most cases of right-sided endocarditis were cured by antibiotics alone and most cases of left-sided endocarditis treated medically did not survive. Most other cases of endocarditis due to *S. marcescens* have occurred in patients with prosthetic heart valves. Only two cases of native valve endocarditis due to *S. marcescens* were reported in the literature [20,21]. Both had right-sided involvement and indwelling intravenous catheters at the time of diagnosis and both were successfully treated medically. An experimental endocarditis rabbit model using *S. marcescens* confirmed the significance of the presence of an indwelling catheter in the development of endocarditis [22].

The classic presentation of suppurative thrombophlebitis involves the internal jugular vein in association with acute oropharyngeal infections – Lemierre’s syndrome. However, several other veins can be affected by the same problem, such as the pelvic veins with puerperal sepsis, the portal vein with inflammatory bowel disease and the dural venous sinuses with contiguous infections. Suppurative thrombophlebitis of the central veins as a complication of central venous catheters is a common problem in patients receiving cancer chemotherapy or hyperalimentation. Unlike peripheral vein suppuration, surgical excision of central veins may not be feasible. Medical management with intravenous antibiotics, catheter removal and anticoagulation is a reasonable alternative, but in cases such as ours, surgical excision of a deep-seated focus of infection is recommended [23].

**REFERENCES**


Pericarditis caused by Corynebacterium urealyticum

Corynebacterium urealyticum, formerly known as Corynebacterium group D2 or CDC group D2 [1], is an aerobic, catalase-positive, Gram-positive bacillus which shows resistance to multiple antibiotics. It has been associated mainly with infections of the urinary tract [2], and the isolation of this organism in other infections is very unusual. We report the first case, to our knowledge, in which C. urealyticum is associated with pericarditis.

A 55-year-old woman with a medical history of diabetes mellitus was admitted to the emergency room with a 2-week history of lower retrosternal pain and dyspnea. On examination she was pyrexial (38 °C) and tachypneic. There were no pericardial or pleural rubs. The patient was alert and oriented. Results of laboratory studies were as follows: hemoglobin level, 10.4 g/dL; white blood cell count, 9910/mm³ (79% neutrophils, 13% lymphocytes, 6.9% monocytes and 0.9% eosinophils); erythrocyte sedimentation rate, 56 mm/h. The urea and electrolyte values were normal. The plasma glucose was elevated at 296 mg/dL. The electrocardiogram (ECG) and chest X-ray were normal. Two-dimensional echocardiograms revealed mild cardiomegaly with normal myocardial contractility, and a big pericardial effusion with fibrinous bands; no echocardiographic signs of cardiac tamponade were observed. A diagnosis of acute pericarditis was made, and pericardiocentesis was prescribed. Seven hundred milliliters of a purulent pericardial fluid were obtained. Histologic examination and Gram stain of the fluid only showed many polymorphonuclear cells. Cultures were performed on blood and chocolate agar, McConkey agar, Sabouraud agar and Brucella agar. An aerobic, catalase-positive bacillus with the typical appearance of Corynebacterium species to antimicrobial agents. J Chemother 1990; 2: 79–81.


tion, *C. urealyticum* rarely causes infection outside of the urinary tract. A limited number of non-urinary infections caused by this organism have been reported: some cases of endocarditis [4–6], bacteremia [7–11], postsurgical osteomyelitis [12], peritonitis in outpatients with chronic peritoneal dialysis [13], necrotic infection of soft tissue [14] and wound infections [8]. Most of these infections were hospital-acquired, the patients had been previously treated with antibiotics, and the infection, in many cases, was related to previous surgery or invasive manipulations.

To our knowledge, this is the first case of a pericarditis caused by *C. urealyticum*. In a literature search for the period 1984–99 via MEDLINE, no other case was found. Our case involved pericarditis in a diabetic patient who had not been previously treated with antimicrobial agents and had not acquired the infection in the hospital, and on whom no invasive diagnostic procedure or surgery had been previously performed. Due to the unusual localization of this infection, establishing the pathogenicity of this microorganism may be difficult. *C. urealyticum* was the only organism isolated from the pericardial fluid, and since the symptoms resolved after the organism was eliminated by antibiotic treatment, we believe that this bacterium was the etiologic agent of the infection. Diabetes was the only underlying predisposing factor that may contribute to the pathogenicity of this strain.

### References


### Infective endocarditis due to *Clostridium histolyticum*

Endocarditis caused by anaerobic and micro-aerophilic bacteria accounts for 7–10% of all cases of infectious endocarditis [1].

The most frequently reported causes of anaerobic endocarditis are *Bacteroides* (particularly the *B. fragilis* group), and anaerobic *Streptococcus*, *Clostridium*, *Peptostreptococcus*, *Fusobacterium*, *Propionibacterium* and *Lactobacillus* species occasionally cause endocarditis [2]. Only 25 cases of clostridial endocarditis have been reported to date, and the most common species was *C. perfringens* [3–7]. To our knowledge, this case is the first occurrence of *Clostridium histolyticum* endocarditis reported in the literature.

An 18-year-old woman was admitted to the Turgut Ozal Medical Center (Malatya, Turkey) following a 20-day history of cough, yellowish-green sputum, and fever; however, she had not experienced dyspnea. She denied smoking and use of drugs. On physical examination, the patient appeared toxic, with an axillary temperature of 39 °C; pulse rate of 85/min, blood pressure of 90/60 mmHg, and respiratory rate of 20 min. A widespread systolodiastolic murmur could be heard on chest auscultation. Auscultation of the lungs revealed crepitant rales over the basal areas, but no pleural rub was noticed. No other anomalies were detected, and pelvic examination did not reveal any foreign body. The results of a neurologic exami-
nation were normal. Laboratory data included a WBC count of \(11 \times 10^9/L\), a hemoglobin level of 8.7 g/dL, and an erythrocyte sedimentation rate of 80 mm/h. The results of the platelet count, prothrombin time, partial thromboplastin time, bleeding time, blood urea nitrogen and serum creatinine, glucose, bilirubin, calcium, protein, albumin, globulin, electrolytes, aspartate, aminotransferase, alanine transaminase and alkaline phosphatase and urine sediment were within normal ranges. Lactate dehydrogenase was 723 IU/L. Serologic tests for HIV, HBV and HCV performed upon admission were all negative. A chest X-ray and abdominal ultrasonography did not reveal any abnormality. The electrocardiogram showed changes due to left ventricular hypertrophy. An echocardiogram showed aortic regurgitation, prominent thickening and a large vegetation on the aortic valve, a finding suggestive of infective endocarditis. Two subsequent aerobic blood cultures, sputum cultures and urine cultures were all negative. Anaerobic blood culture was not performed. The patient received treatment with penicillin 4 million units every 4 h plus 80 mg of gentamicin every 8 h empirically.

Five days after admission, cardiac surgery was planned due to congestive cardiac failure and large mobile vegetations on the aortic valve. Surgery also revealed a large vegetation on the aortic valve. A microbiological specimen from the valve tissue sent to the laboratory for culture was inoculated on Brucella blood agar plates for anaerobic culture and tryptic soy-asblood agar plates for aerobic culture. Gram-positive rods were seen on Gram staining of the direct preparation. Growth occurred after 48 h under anaerobic conditions. The aerotolerance test, cellular morphology, susceptibility to kanamycin, colistin and vancomycin special potency disks for anaerobic bacteria and spore tests revealed a Clostridium sp. [8]. Species identification and antibiotic susceptibility tests were performed in the Anaerobe Reference Laboratory in Helsinki. The organism was identified as C. histolyticum, and its susceptibilities to penicillin, metronidazole, clindamycin and imipenem were determined by means of the E test (AB Biodisk, Solna, Sweden), and the following MICs were obtained: penicillin G, 0.016 mg/L; metronidazole, 0.094 mg/L; clindamycin, 0.016 mg/L; and imipenem, 0.012 mg/L. These MICs are below the breakpoint values [4]. Treatment with penicillin G plus metronidazole was started in the immediate postoperative period. After a 5-week treatment period, laboratory and clinical findings were all normal.

C. histolyticum is known as one of the histotoxic clostridia, and is most commonly involved in myonecrosis [9]. This species of Clostridium has not been previously isolated from endocarditis or bacteremia patients.

There is a propensity for anaerobic endocarditis to produce large vegetations. Diagnosis of anaerobic endocarditis is not different from that of other forms of endocarditis and usually involves demonstration of continuous bacteremia and a valvular lesion that is consistent with endocarditis [2]. In our case, the echocardiogram showed a large vegetation on the aortic valve which was also reported by the surgeon. In the histologic examination, valvular endocardial tissue showed mild plasma cell infiltration with scattered eosinophils. Small foci of fibrous vegetations were also present on the valvular surface. All of these findings were consistent with valvular endocarditis which was also previously hyalinized.

Of the 25 cases reported in the literature, 14 have detailed clinical information for the major characteristics of clostridial endocarditis. Two were associated with intravenous drug abuse, and subacute presentation was documented in six. Carcinoma of the colon or cervix was documented in two other cases. Peripheral emboli were reported in four cases, coexistent atypical pneumoniae and cardiac failure was documented in one case [5], emboli were documented in one case [6], and hemiplegia was reported in one case [4]. An echocardiogram indicated vegetations on the tricuspid valve [3], on the aortic wall at the junction with a dacron prosthesis [4], and below the mitral annulus of the left ventricular wall [6]; also, in the other study, an echocardiogram showed a para-aortic abscess [7]. Vegetations were revealed at autopsy on the tricuspid and pulmonary valves in another case [5].

Involvement of the tricuspid valve, the mitral valve, the aortic valve or both was documented in all other cases; infection of prosthetic valves was reported in four cases [4,5,7]. Negative aerobic blood cultures are sometimes associated with fungal or anaerobic endocarditis [9]. Confirming our results, the aerobic blood cultures were all negative.

Using both aerobic and anaerobic blood culture bottles together will successfully increase the rate of detection of anaerobic etiologic agents of bacteremia or infective endocarditis.

Although anaerobic blood culture was not performed, isolation of C. histolyticum from valvular tissue specimens and histology of the surgically resected valvular tissue indicated that this bacterium was the etiologic agent of this patient’s infective endocarditis, confirming the clinical findings.

ACKNOWLEDGMENTS

We thank Dr Hannale Somer-Jousimies and her colleagues in the Anaerobe Reference Laboratory in Helsinki for identification to species level and antibiotic susceptibility testing of Clostridium histolyticum.

REFERENCES


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Susceptibility pattern of Streptococcus pneumoniae in outpatients in Germany

During a 1992–97 German surveillance study, Reinert et al [1] found no penicillin-resistant Streptococcus pneumoniae strains among blood culture isolates (n = 200). We report the susceptibility pattern of recent S. pneumoniae strains (only one per patient) isolated from outpatients seen in general practice or in our Outpatient Department. The majority of the S. pneumoniae strains (n = 89) were cultured from material sent to a laboratory serving general practitioners in the Frankfurt area (EJKR) in 1998. Only eight strains (three of them in winter 1999) were cultured from patients who consulted the Outpatient Department of our Hospital. The sources of the strains were: upper respiratory tract (63); lower respiratory tract (17); blood/cerebrospinal fluid (6); ear (5); others (6). Minimum inhibitory concentrations (MIC) were determined for penicillin (PEN), erythromycin (ERY), doxycycline (DOXY), clindamycin (CLIN), levofloxacin (LEV) and moxifloxacin (MOX) using the E test method. Three S. pneumoniae strains, with a penicillin MIC of 0.5, 2.0 and 4.0 mg/L, respectively, provided by Dr Bryskier, France were used as quality control strains.

The MIC distribution of the 97 strains tested is given in Table 1. The rate of resistance at break-points defined in the Alexander Study [2] to PEN is 4.1%, to ERY is 15.5%, and to DOXY is 15.5%. As recommendations from an independent source such as DIN (Deutsches Institut fuer Normung eV) are still missing, we used the break-points suggested by several German pharmaceutical companies: MOX, ≤ 1 mg/L for sensitivity, ≥ 4 mg/L for resistance; LEV, ≤ 2 mg/L for sensitivity, ≥ 8 mg/L for resistance. At these admittedly high break-points for the newer quinolones none of the strains was classified as resistant (Table 2).

Four out of 97 strains which we collected in 1998–99 were resistant to penicillin. We believe this is the highest incidence of penicillin-resistant pneumococci reported so far from Germany. In the present study we found 15.5% (15/97) erythro-

<table>
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PEN, Penicillin G; ERY, erythromycin; DOXY, doxycyclin; CLIN, clindamycin; MOX, moxifloxacin; LEV, levofloxacin.
mycin resistance, which is markedly higher than the 3% (only six out of 200 strains collected between 1992 and 1996) reported recently by Reinert et al [3] for Germany, and reflects the trend reported by Kresken et al [4] who observed an increase in erythromycin resistance (break-point $\geq 1 \text{mg/L}$, which is lower than the one used in the Alexander Project [2]) from 3.6% in 1992 to 11.3% in 1997. Frankfurt (if not Germany) seems to have joined the ‘club of countries with penicillin-resistant pneumococci’.

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These results were presented at 21st ICC, Birmingham, 1999. Poster NO:190.

REFERENCES

Table 2 Breakpoints, R I S distribution, geom. mean and range of 97 pneumococcal strains

<table>
<thead>
<tr>
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PEN, Penicillin G; ERY, erythromycin; DOXY, doxycyclin; CLIN, clindamycin; MOX, moxifloxacin; LEV, levofloxacin.

Localized Mycobacterium avium complex infection in a patient on HAART

The increases in CD4 T-lymphocytes count induced by antiretroviral therapy provide an anti-infective protection such that primary prophylaxis against opportunistic infections can be stopped. There is, however, concern that CD4 count augmented by drug therapy may not include cells specifically active against some of the frequent opportunistic infections, including atypical mycobacterial infections [1].

We report a case of Mycobacterium avium intracellulare occurring in a 55-year-old retired bank manager who had been unwell for about 9 months with repeated respiratory chest infections and loss of weight. He was thought initially to have an underlying neoplasm and had undergone extensive investigations including a whole body CT (computed axial tomographic) scan at the referring hospital. He was divorced in 1978 and during the 1980s had multiple homosexual contacts. During the 2 weeks prior to being referred he had become progressively short of breath associated with a dry cough. Physical examination was essentially normal apart from a temperature of 37.5 °C. His chest X-ray revealed bulky hilar lymphadenopathy and there were patchy opacities of both the mid-zones.

Blood gas analysis revealed $pO_2$ 10.6 kPa (4.5–6.1) and $pCO_2$ 4.1 kPa (12.0–14.0). Pulmonary function tests showed that the TLCO was 6.77 mmol/min/kPa (62% of expected) and $kCO$ was 1.18 mmol/min/kPa/L (84% of expected). Although induced sputums taken for indirect immuno£uorescent antibody stain against Pneumocystis carini were negative, a subsequent broncho-alveolar lavage examination was strongly positive. Acid-fast bacilli were not demonstrated. He was commencing on high dose cotrimoxazole (120 mg/kg per day) with prednisolone. He was counselled and consented to be tested for HIV and this was shown to be positive (ELISA Abbot (Abbot Laboratories, Chicago, USA); ELISA Organon (Organon Teknika Ltd, Boxtel, the Netherlands) and INNO-LIA Blot test (Ghent, Belgium)). The HIV load was noted to
be 750,000 copies/mL (Roche Amplicor) with the CD4 count of 63 cells/μL (600–1100). He symptomatically improved with treatment but became very confused and disoriented on completion of the therapy for PCP, and a CT scan of the brain revealed a generalized cerebral atrophy. A lumbar puncture showed the protein elevated at 1.44 g/L (0.18–0.5) but there were no cells or organisms seen and the cryptococcal antigen (Latex agglutination test) was negative. He was commenced on anti-retroviral therapy of zidovudine, lamivudine and indinavir. He was discharged home but had an episode of mania requiring treatment with haloperidol and lorazepam with good response.

He clinically improved but 2 months later he developed a dry, unproductive cough with sweats and a fever. He was noted to be anemic with the hemoglobin of 9.9 (13.5–18.0 g/dL). A repeat chest X-ray showed a nodular opacity at the apex of the right lower lobe. A CT scan of the thorax (Figure 1) demonstrated mediastinal lymphadenopathy and a patchy area of confluent nodular opacification with a cavity in the apical segment of the right lower lobe. Bronchoalveolar fluid examination showed presence of acid-fast bacilli by Ziel-Neilsen stain, and a trans-bronchial biopsy of the lesion showed granulomatous inflammation with central necrosis containing polymorphs. Subsequent culture (MB\BacT Organon Teknika Ltd) of the lavage fluid grew *M. avium* complex. Three sets of blood cultures did not show any growth. At this stage the HIV viral load was reduced significantly to 1850 copies/mL with a CD4 count of 250 cells/μL. The polymerase chain reaction (PCR) for *M. tuberculosis* was negative. The patient was commenced on treatment with azithromycin and rifabutin. The organism was multiresistant to isoniazid, ethambutol, rifampicin, pyrazinamide, ciprofloxacin, azithromycin and streptomycin (resistance ratio method). His symptoms resolved and he was discharged home.

Our case demonstrates the ever-increasing spectrum of clinical manifestations associated with opportunistic infections in HIV-infected individuals in the era of HAART (Highly Active Anti-Retroviral Therapy). Initiation of combination anti-retroviral therapy results in the dramatic decrease of the HIV viral load with a concomitant restoration of cell-mediated immune function [1]. This enhanced immunity may lead to a marked inflammatory reaction and to altered clinical manifestations of pre-existing latent infections. A recent study reported the incidence of pathogen-associated inflammatory diseases occurring in 143 HIV-infected patients after treatment with potent anti-retroviral therapy. Thirty-three responders (25%) had one or more episodes of opportunistic infections. Five had localized MAC disease, six had exacerbation of CMV retinitis, nine had mucocutaneous herpes, eight had dermatomal zoster, four had myelitis and/or encephalitis with presumptive HSV infection, three had HCV-associated hepatitis, two had MTB-associated lymphadenitis/pneumonitis or cerebritis and six had inflamed molluscum contagiosum or warts. These diseases occurred during the first 2 months of therapy and a low baseline CD4 T-cell count was the only predictor of their onset [2].

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There have been a number of reports of a clinical syndrome of MAC-related inflammatory lymphadenitis on initiating...
HAART. Race et al recognized this on initiation of a protease inhibitor, indinavir, in patients with advanced HIV-1 disease [3]. A retrospective study conducted on 52 patients with mycobacterial lymphadenitis in HIV showed 12 patients developed the infection within 12 weeks of initiating combination anti-retroviral therapy. The clinical presentation, which may vary, was in these cases of localized disease and was associated with a relatively high CD4 count. All patients showed a good immunological and virological response to HAART [4].

A further analysis identified five patients who developed mycobacterial lymphadenitis within 8 weeks of initiating HAART. All patients had fever, weakness and splenomegaly. Four of the five patients were anemic with Hb < 10 g/dL and demonstrated lymphadenopathy. This was cervical in three, submandibular in two, mediastinal in three and preauricular in one [5]. Interestingly, our patient presented with a primary lung lesion (Figure 1). We are not aware of any other literature describing cavitary lung lesions, due to localized MAC infection, relating to HAART therapy in HIV infection.

MAC infections may reflect restoration of pathogen-specific immune responses rather than reactivation of the initiating infection. Partial immune reconstitution, as demonstrated by an increase in the CD4 counts on anti-retroviral therapy is likely to be responsible for the localized MAC infection. This reconstitution may lead to an unmasking of subclinical infection and an enhanced immune response to mycobacterial antigens. There is evidence to show that immune reconstitution due to HAART occurs in two stages and the initial CD4 lymphocyte proliferation is primarily of the memory subsets [6–8]. Thus this marked memory T-cell-mediated and antigen-specific inflammatory reaction may then lead to tissue suppuration and granuloma formation, the hallmarks of mycobacterial disease.

The occurrence and severity of illness in our patient with localized but untreated MAC infection after the start of HAART suggests that patients with advanced HIV disease should be assessed for active mycobacterial infection before the initiation of such therapy. It is also important to consider that dual opportunistic infections may occur in advanced disease as our patient demonstrated. Therefore, patients with severe immune dysfunction should be monitored very carefully, in particular during the first weeks after initiating HAART. Indeed, it may be important in the first few months of HAART to maintain patients on prophylaxis for opportunistic infections.

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Endocarditis due to Gemella haemolysans in a patient with hemochromatosis

Gemella haemolysans is a facultative anaerobic Gram-positive coccus that can be a commensal of the upper respiratory tract and oral cavity of humans. The clinical spectrum of infections produced by this microorganism includes endocarditis, septicaemia and meningitis. We report a case of endocarditis due to G. haemolysans in a patient with hemochromatosis.

A 77-year-old man with a long history of hemochromatosis with chronic liver disease and arterial hypertension was admitted to hospital because of malaise, anorexia, weight loss and progressive dyspnea over the previous 4 months. On admission he was afebrile and the physical examination revealed a grade 2 diastolic ejection murmur in the left sternal border and hepatomegaly. Laboratory studies disclosed the following data: leucocyte count 12 000/mm³ (80% neutrophils, 17% lymphocytes), haematocrit 48%, erythrocyte sedimentation rate of 54 mm/h, iron 20(0) μg/dL, ferritin 2277 U
mL, AST 64 U/mL and ALT 57 U/mL. Radiographs of the chest were normal. Echocardiography revealed a vegetation on the aortic valve with moderate valvular insufficiency. *G. haemolysans* was isolated in the three blood cultures taken on admission. The identification was performed on the basis of Gram stain, catalase activity and biochemical characteristics (API 20 STREP, BioMérieux, France). The isolate was susceptible to penicillin (MIC: 0.06 mg/L). The patient was treated with penicillin G (24 MU IV daily, divided in six doses) and tobramycin (100 mg twice a day IV) for 2 weeks whereupon he showed improvement of his clinical status as well as sterilization of the blood cultures. In a follow-up visit 30 days after discharge, the echocardiogram did not show any vegetation.

*G. haemolysans* was first described in 1938 by Thjotta and Bøe [1] as *Neisseria haemolysans* but was reclassified into a new genus, *Gemella*, following demonstration of biochemical differences with other *Neisseria*. This species is easily decolorized during Gram staining and may therefore appear as Gram-variable or even Gram-negative. It can be misidentified as a viridans streptococcus or remain unidentified. An accurate identification can be made with commercial biochemical tests such as the API 20 STREP system.

To the best of our knowledge, 15 cases of endocarditis due to *G. hemolysans* have been reported so far [2–14]. The available information is shown in Table 1. Mean age was 53.2 years and ranged from 20 to 77 years. Of 16 patients, only two were women (12.5%). All but two cases occurred in patients with underlying mitral or aortic valve disease (including prosthetic valves) and/or poor dentition [5,10]. Infected valves were aortic and mitral, both of them with the same percentage (50%). The outcome was completely favorable after antibiotic treatment, although some patients needed valve replacement [2–4,9,10,12,13]. In our patient, aortic valve insufficiency was the only risk factor identified for endocarditis but, interestingly, he had hemochromatosis. The association of *G. haemolysans* with hemochromatosis has not been reported to date and in this case probably represents a casual relationship between two uncommon conditions. However, the increased availability of iron in patients could have enhanced the virulence of *G. haemolysans* as has been demonstrated with other bacterial species such as *Yersinia* spp., *Vibrio*

### Table 1 Summary of patients with reported cases of *G. haemolysans* endocarditis

<table>
<thead>
<tr>
<th>Case/Reference</th>
<th>Age/Sex</th>
<th>Underlying conditions or source of infection</th>
<th>Infected valve</th>
<th>Treatment</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/1</td>
<td>62/M</td>
<td>Dental manipulation</td>
<td>Mi</td>
<td>Cefamandole, gentamicin, benzylpenicillin, amoxicillin</td>
<td>Survived</td>
</tr>
<tr>
<td>2/1</td>
<td>48/M</td>
<td>Poor dentition, respiratory viral syndrome</td>
<td>Ao</td>
<td>Benzylpenicillin, gentamicin</td>
<td>Survived</td>
</tr>
<tr>
<td>3/1</td>
<td>56/M</td>
<td>Mi insufficiency, dental manipulation</td>
<td>Mi</td>
<td>Benzylpenicillin, gentamicin</td>
<td>Survived</td>
</tr>
<tr>
<td>4/2</td>
<td>68/M</td>
<td>Ao insufficiency</td>
<td>Ao</td>
<td>Benzylpenicillin, streptomycin</td>
<td>Survived</td>
</tr>
<tr>
<td>5/3</td>
<td>47/M</td>
<td>Prosthetic aortic valve, periodontal disease</td>
<td>Ao</td>
<td>Erythromycin, rifampin</td>
<td>Survived</td>
</tr>
<tr>
<td>6/4</td>
<td>62/F</td>
<td>None</td>
<td>Mi</td>
<td>Benzylpenicillin, tobramycin, clindamycin</td>
<td>Survived</td>
</tr>
<tr>
<td>7/5</td>
<td>74/M</td>
<td>Mi prolapsed</td>
<td>Mi</td>
<td>Benzylpenicillin, gentamicin, amoxicillin</td>
<td>Survived</td>
</tr>
<tr>
<td>8/6</td>
<td>42/M</td>
<td>Ao insufficiency, wound</td>
<td>Ao</td>
<td>Vancomycin, fusidic acid, amoxicillin, gentamicin</td>
<td>Survived</td>
</tr>
<tr>
<td>9/7</td>
<td>71/M</td>
<td>Colonic carcinoma</td>
<td>Mi</td>
<td>Amoxicillin, clavulanic acid, gentamicin</td>
<td>Survived</td>
</tr>
<tr>
<td>10/8</td>
<td>53/F</td>
<td>Mi regurgitation</td>
<td>Mi</td>
<td>Benzylpenicillin, gentamicin</td>
<td>Survived</td>
</tr>
<tr>
<td>11/9</td>
<td>20/M</td>
<td>None</td>
<td>Ao</td>
<td>Benzylpenicillin, gentamicin</td>
<td>Survived</td>
</tr>
<tr>
<td>12/10</td>
<td>34/M</td>
<td>Prosthetic valve, endocarditis, dental sepsis</td>
<td>Ao</td>
<td>Cefuroxime, tobramycin, ciprofloxacin, erythromycin</td>
<td>Survived</td>
</tr>
<tr>
<td>13/11</td>
<td>48/M</td>
<td>Prosthetic Ao and Mi valves</td>
<td>Mi</td>
<td>Benzylpenicillin, gentamicin</td>
<td>Survived</td>
</tr>
<tr>
<td>14/12</td>
<td>63/M</td>
<td>Chronic obstructive bronchitis, poor dentition</td>
<td>Mi</td>
<td>Amoxicillin, amikacin</td>
<td>Survived</td>
</tr>
<tr>
<td>15/13</td>
<td>26/M</td>
<td>Down syndrome, Ao insufficiency, ventricular septal defect</td>
<td>Ao</td>
<td>Benzylpenicillin, gentamicin</td>
<td>Survived</td>
</tr>
<tr>
<td>Present report</td>
<td>77/M</td>
<td>Ao insufficiency, hemochromatosis</td>
<td>Ao</td>
<td>Benzylpenicillin, tobramycin</td>
<td>Survived</td>
</tr>
</tbody>
</table>

Note: Sex (M, Male; F, Female); Valves (Ao, Aortic valve; Mi, Mitral valve).
spp., and some mycobacteria among others [15]. In summary, our case suggests that hemochromatosis may be a risk factor for endocarditis due to G. haemolysans.

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Unusual high-performance liquid chromatography profile of a strain of Mycobacterium avium

Mycobacterium avium complex (MAC) is the most frequently detected non-tuberculous mycobacterium, not only in AIDS patients, where it is responsible for disseminated and localized infections, but also in non-immunocompromised subjects. Among the latter, it is frequently responsible for pulmonary disease in elderly people with various predisposing conditions, and for cervical lymphadenitis in the children [1].

Several different approaches can be applied for the identification of MAC: conventional tests reveal well-defined biochemical and cultural features; highly specific commercial DNA probes (AccuProbe, USA) react with a species-specific 16S rRNA stretch; and finally, high-performance liquid chromatography (HPLC) of cell wall mycolic acids provides an easily recognizable pattern characterized by three clusters of peaks (Figure 1b).

Herein, we report the characterization of two strains isolated from sputum collected at 5-month intervals from the same immunocompetent elderly subject. Both isolates had the phenotypic features of MAC, and both reacted with AccuProbe M. avium, but not with AccuProbe M. intracellulare.

Figure 1 Mycolic acid patterns obtained by HPLC: (a) profile of the first strain (atypical); (b) profile of the second strain (MAC-typical). LMWIS and HMWIS are low and high molecular weight internal standards, respectively.

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The IS1245-based restriction fragment length polymorphism analysis [2,3] of both isolates yielded different patterns (Figure 2), thus demonstrating the double origin of the infection.

Surprisingly, the HPLC profiles of the two strains were completely different (Figure 1): the one obtained from the second strain (b) is like that established for *M. avium* and for MAC in general [4], whereas the HPLC pattern of the first strain (a) is unique, never seen in our experience with over 180 MAC strains characterized with HPLC and AccuProbe, and never reported for *M. avium*.

The above data therefore add to our present knowledge of the HPLC profile of MAC; the same typical three-clustered profile is shared by strains assigned, on the basis of hybridization with specific AccuProbes, to the species *M. avium* or *M. intracellulare*, and by the members of the so-called MAC intermediates or MAI-X group [5], which react with AccuProbe MAC, but not with either AccuProbe – *M. avium* or Accuprobe – *M. intracellulare*.

A similar anomaly of HPLC profiles has been previously reported in a few isolates of mycobacteria reacting with the MAC probe but not with the species-specific ones [6]; in that case it was, however, possible to establish genetic sequences unrelated to MAC and very close to *M. simiae*. In the present case, hypervariable regions A and B of 16S rDNA [7] yielded a perfect match with the signature sequences of *M. avium* for both isolates.

This is therefore the first report of an *M. avium* strain in which the 16S rDNA species-specific markers do not correlate with the chromatographic pattern typical of this species.

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