eased tissue, it would be worthwhile to test the pathogenic properties of these strains in experimental animals. Until the pathogenicity of these *Hartmannella* strains is proven, species of this genus should be treated as harmless commensals.

We were surprised to read that a species, called “*H. varini,*” was used as a reference strain in the investigation of Aimard et al. [2]. To our knowledge, this species has not been described in the literature, and it is not listed in any of the definitive naked amoeba identification keys reported by Page [4–6]. In addition, it does not correspond to either of the two new *Hartmannella* species that have recently been described [7, 8]. Furthermore, there is no reference strain of a species of this name held by either the Culture Collection of Algae and Protozoa (Ambleside, United Kingdom) or the American Type Culture Collection (Rockville, MD). The use of “*H. varini,*” as a reference strain appears to have been invalid. We would be interested to know from where Aimard et al. [2] obtained this strain and where (if at all) it was described in the literature.

Johan F. De Jonckheere and Susan Brown
Protozoology Laboratory, Scientific Institute of Public Health—Louis Pasteur, Brussels, Belgium; and Culture Collection of Algae and Protozoa, Institute of Freshwater Ecology, Ambleside, Cumbria, United Kingdom

References


Reprints or correspondence: Dr. Johan F. De Jonckheere, Protozoology Laboratory, Scientific Institute of Public Health—Louis Pasteur, J. Wytsmanstraat 14, B-1050 Brussels, Belgium.

Clinical Infectious Diseases 1998;27:1337–8

© 1998 by the Infectious Diseases Society of America. All rights reserved. 1058-4838/98/2705–0051$03.00

Reply

Sir—We read with great interest the letter of De Jonckheere and Brown about our recent brief report [1]. We agree with De Jonckheere and Brown that *Hartmannella* should not be considered a pathogen without testing its pathogenic properties in experimental animals. However, culture of a corneal biopsy specimen yielded *Hartmannella* cysts and *Acanthamoeba* cysts and confirmed results of a histological microscopic examination. The conditions used to process the sample excluded the possibility of contamination with *Hartmannella*. Moreover, this ameba was not present on the superficial layers of the corneal biopsy specimen since scrapings were negative. In addition, the *Hartmannella* isolate found in the cornea was related to failure of hexamidine therapy, which may raise the question about the pathogenicity of *Hartmannella* as was previously suggested [2, 3].

“*H. varini,*” which was used as a reference strain, corresponded to *Hartmannella vermiformis* and was provided by the Culture Collection of Algae and Protozoa (Ambleside, United Kingdom; reference 1534/7).

Laurence Aimard, Philippe Brasseur, Loïc Favennec, Daniel Perrine, Laure Wat, and Gérard Brasseur
Département d’Ophtalmologie and Laboratoire de Parasitologie, Hôpital Charles Nicolle, Rouen, and Laboratoire de Parasitologie, Faculté de Pharmacie, University of Caen, Caen, France

Pneumococcal Pericarditis Since 1980

Sir—Saenz et al. [1] reported a case of purulent pericarditis caused by a highly resistant strain of *Streptococcus pneumoniae* that was successfully treated with vancomycin. With the prevalence of penicillin-resistant *S. pneumoniae* isolates approaching 16% in a New York City Department of Health survey from January to September 1997 (of which 10% had intermediate-level resistance and 6% had high-level resistance), one may expect more of such cases (written communication, New York City Department of Health).

A 75-year-old woman with cirrhosis and breast cancer who was being treated with tamoxifen presented with fever, chills, cough, and pleuritic chest pain. Physical examination revealed a pulsus paradoxus and a pericardial friction rub. A chest radiograph demonstrated right-lower-lobe pneumonia; a two-dimensional echocardiogram showed a large pericardial effusion, and cardiac tamponade was revealed with use of a Swan-Ganz catheter. Pericardiocentesis was done, and 300 mL of fluid was aspirated; the WBC count in the pericardial fluid was 265,000/mm³. A pericardial window was formed, and an external drain was placed.

Reprints or correspondence: Dr. L. Aimard, Laboratoire de Parasitologie, Hôpital Charles Nicolle, 1 Rue de Germont, F-76031 Rouen Cedex, France.

Clinical Infectious Diseases 1998;27:1338

© 1998 by the Infectious Diseases Society of America. All rights reserved. 1058-4838/98/2705–0052$03.00
Table 1. Summary of data on 15 cases of pneumococcal pericarditis from 1980 to 1998.

<table>
<thead>
<tr>
<th>Reference</th>
<th>Age (y)/sex</th>
<th>Culture specimen(s)</th>
<th>Risk factor(s)</th>
<th>Source</th>
<th>Medical treatment</th>
<th>Surgical treatment</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>[1]</td>
<td>78/F</td>
<td>Blood, PCF</td>
<td>Diabetes</td>
<td>Lung</td>
<td>Vancomycin</td>
<td>Pericardiocentesis</td>
<td>Recovered</td>
</tr>
<tr>
<td>[3]</td>
<td>64/M</td>
<td>Blood, PLF, CIE* PCF</td>
<td>Felty’s syndrome</td>
<td>Lung</td>
<td>Cephapirin sodium</td>
<td>Chest tube, pericardial window, pleural decortication</td>
<td>Recovered</td>
</tr>
<tr>
<td>[3]</td>
<td>60/M</td>
<td>Blood, PLF, sputum, CIE* PCF</td>
<td>Renal insufficiency, cardiac disease, alcoholism</td>
<td>Lung</td>
<td>Penicillin</td>
<td>Chest tube, pericardial window, pleural decortication</td>
<td>Recovered</td>
</tr>
<tr>
<td>[4]</td>
<td>31/M</td>
<td>Blood</td>
<td>Alcoholism</td>
<td>None</td>
<td>Penicillin</td>
<td>Pericardial drainage (surgical)</td>
<td>Died</td>
</tr>
<tr>
<td>[5]</td>
<td>19/F</td>
<td>PCF</td>
<td>Common variable immunodeficiency</td>
<td>Lung</td>
<td>Penicillin</td>
<td>Pericardial catheter drainage, chest tube</td>
<td>Recovered</td>
</tr>
<tr>
<td>[6]</td>
<td>88/F</td>
<td>Blood</td>
<td>None</td>
<td>Lung</td>
<td>Penicillin</td>
<td>Pericardial catheter drainage, chest tube</td>
<td>Recovered</td>
</tr>
<tr>
<td>[8]</td>
<td>47/M</td>
<td>PCF</td>
<td>None</td>
<td>Lung</td>
<td>Penicillin</td>
<td>Pericardial window, pericardiocentesis</td>
<td>Recovered</td>
</tr>
<tr>
<td>[9]</td>
<td>78/F</td>
<td>PCF</td>
<td>NA</td>
<td>NA</td>
<td>Antibiotics</td>
<td>Intrapericardial streptokinase, pericardiocentesis</td>
<td>Recovered</td>
</tr>
<tr>
<td>[10]</td>
<td>29/M</td>
<td>PCF</td>
<td>None</td>
<td>Lung</td>
<td>Antibiotics</td>
<td>Pericardiosentesis</td>
<td>Died</td>
</tr>
<tr>
<td>[11]</td>
<td>3.5/M</td>
<td>Blood, PCF, peritoneal, middle ear exudate</td>
<td>None</td>
<td>Otitis media</td>
<td>Penicillin</td>
<td>Pericardiosentesis, pericardial window, laparotomy</td>
<td>Recovered</td>
</tr>
<tr>
<td>[12]</td>
<td>57/F</td>
<td>Blood, PCF</td>
<td>Sarcoïdosis, steroid therapy</td>
<td>Lung</td>
<td>Penicillin, clarithromycin</td>
<td>Pericardiocentesis</td>
<td>Recovered</td>
</tr>
<tr>
<td>[PR]</td>
<td>75/F</td>
<td>Blood, PCF</td>
<td>Breast cancer, cirrhosis</td>
<td>Lung</td>
<td>Penicillin</td>
<td>Pericardiocentesis, pericardial window</td>
<td>Died</td>
</tr>
</tbody>
</table>

NOTE. CIE = counterimmunoelectrophoresis; I & D = incision and drainage; NA = not available; PCF = pericardial fluid; PLF = pleural fluid; PR = present report; TB = tuberculosis.

* Organism detected using CIE; cultures were nondiagnostic, probably secondary to prior antibiotic therapy.

Cultures of both blood and pericardial fluid yielded *S. pneumoniae* susceptible to penicillin. Despite therapy, she died after 4 weeks.

In an autopsy series of cases of pericarditis before 1943 [2], pneumococcal pericarditis occurred in about 51% of cases, followed by staphylococcal pericarditis (19%), pericarditis due to other streptococci (10%), and pericarditis due to gram-negative bacilli (2%). These cases occurred primarily in children and young adults secondary to pneumonia. After 1943, there was a significant change in the etiology of pericarditis, with gram-negative bacilli, staphylococci, other streptococci, and pneumococci accounting for 32%, 22%, 13%, and 9% of the cases, respectively. The change in the microbiological spectrum was attributed to the advent of antibiotics, thoracic surgery, renal dialysis, and chemotherapy. The average age of a patient with pericarditis was 49 years; pericarditis was more likely to be seen in a debilitated adult with chronic disease, malignancy, or a history of thoracic surgery.

We reviewed cases of pneumococcal pericarditis since 1980 to see if age, risk factors, and prognosis were any different in the past 2 decades. A MEDLINE search revealed an additional 14 cases (table 1). The average age of the patients was 49 years (range, 3.5 to 88 years). Risk factors included alcoholism, HIV infection, diabetes, renal insufficiency, common variable immunodeficiency, sarcoidosis, Felty’s syndrome, and steroid therapy. The source of pericarditis was as follows: pneumonia or empyema, pneumococcal pericarditis occurred in about 51% of cases, followed by staphylococcal pericarditis (19%), pericarditis due to other streptococci (10%), and pericarditis due to gram-negative bacilli (2%). These cases occurred primarily in children and young adults secondary to pneumonia. After 1943, there was a significant change in the etiology of pericarditis, with gram-negative bacilli, staphylococci, other streptococci, and pneumococci accounting for 32%, 22%, 13%, and 9% of the cases, respectively. The change in the microbiological spectrum was attributed to the advent of antibiotics, thoracic surgery, renal dialysis, and chemotherapy. The average age of a patient with pericarditis was 49 years; pericarditis was more likely to be seen in a debilitated adult with chronic disease, malignancy, or a history of thoracic surgery.

Three patients died and 12 recovered. Ten blood cultures and nine pericardial fluid cultures yielded *S. pneumoniae*. Two pericardial cultures were negative, but counterimmunoelectrophoresis was positive for *S. pneumoniae* antigen.

There has been a dramatic decrease in the number cases of purulent pericarditis. The diagnosis is often delayed because classic signs of pulsus paradoxus, friction rub, distended neck veins, and distant heart...
Although it was quite interesting to read the report of lepromatous leprosy in a renal transplant recipient [1], I was surprised that Mushatt et al. were permitted to speculate that the patient might represent a case of contact with armadillos, since he apparently grew up and lived in an area where leprosy is well known to be endemic—New Orleans and Baton Rouge in south Louisiana. In contrast, northern Louisiana has not been associated with the transmission of endemic leprosy until recently [2]; there have been virtually no other cases in these parishes where lifelong residents—with no contact with patients with leprosy—developed leprosy. In northern Louisiana, the argument is that armadillos are the only obvious source of *Mycobacterium leprae*. In contrast, it has been known for >200 years that living in south Louisiana poses some risk for the acquisition of leprosy without travel and without contact with patients with diagnosed cases. The risk predates armadillos in Louisiana [2, 3].

In addition, the case report by Mushatt et al. nicely included HLAs (human leukocyte antigens) and showed once again that the risk of developing leprosy is associated with class II antigens. The analysis that we conducted, which included the six cases previously reported from northern Louisiana and a meta-analysis of data in the literature at that time, showed an association between lepromatous leprosy and HLA-DR2 and HLA-DQw1 [4]. Concerns about the pathogenesis, epidemiology, endemic transmission, and diagnosis of leprosy (including the acquisition of endemic disease) should continue to receive educational and research efforts, particularly in countries where it is endemic, such as the United States [5].

Burton C. West

Department of Medicine, Meridia Huron Hospital, Cleveland, Ohio

References


Reprints or correspondence: Dr. Burton C. West, Department of Medicine, Meridia Huron Hospital, 13951 Terrace Road, East Cleveland, Ohio 44112.

Clinical Infectious Diseases 1998;27:1340
© 1998 by the Infectious Diseases Society of America. All rights reserved. 1058-4838/98/2705-0054/00.00

Reply

Sir—In his letter responding to our recent case report of leprosy in a renal transplant recipient [1], West expresses surprise that we were “permitted to speculate that the patient might represent a case of contact with armadillos. . . .” On the contrary, we stated that there was no direct contact with armadillos and that exposure