CORRESPONDENCE

Aspergillus Infection in Patients with Hyperimmunoglobulin E Syndrome

Sir—In their case report on pulmonary aspergillosis in a child with hyperimmunoglobulin E syndrome (HIE), Wolach et al. [1] state that aspergillus infections in patients with this syndrome are extremely rare. They were able to find only one case of pulmonary aspergillosis and HIE in the literature.

On the basis of our experience, we tend to disagree about the rarity of this occurrence. In a series of five Dutch patients with HIE that was reported in 1985, two patients with this complication were included [2]. One of them, a woman, died of massive hemoptysis caused by multiple pulmonary aspergillomas at the age of 20. A second female patient was treated surgically for a pulmonary aspergilloma in the right lower lobe when she was 8 years old. Twelve years later, she had a pulmonary abscess and recurrent hemoptysis refractory to embolization; a lobectomy of the right upper lobe was performed. During the postoperative period, the patient died of respiratory insufficiency.

Recently, we saw a third patient (a 16-year-old girl with HIE) who developed a pyopneumothorax and a cavitary lesion of the right lower lobe [3]. Cultures of pleural fluid yielded Aspergillus fumigatus. After drainage and treatment with intravenous amphotericin B and itraconazole, resection of the diseased lobe was performed. The patient recovered. Currently, she is receiving prophylaxis with fluocoxacinil and itraconazole.

Each of these three patients had classical HIE with high serum levels of IgE, the typical physionomy, dermatitis, recurrent skin abscesses, and pulmonary infections (mainly caused by Staphylococcus aureus). In all three patients, the aspergilloma arose in a preexisting pneumatocele, which was a consequence of previous suppurative pulmonary infections. Thus, aspergillus infections are not that rare in patients with HIE, especially when cavitary lesions are present.

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References


Reply

Sir—We appreciate the interest in our article [1]. Our statement that aspergillus infection is extremely rare in patients with hyperimmunoglobulin E syndrome (HIE) was based on an extensive search of the English-language literature, which yielded only one case of an adult patient with HIE and aspergillus infection [2]. We were also surprised by the paucity of reports describing the association of the above-mentioned entities. The three cases cited by van der Meer et al. in their correspondence were reported in Dutch [3, 4] and therefore were overlooked. Even with the additional three cases, aspergillosis in patients with HIE is still uncommon. Its occurrence emphasizes that pulmonary infection with opportunistic fungi can be found in patients with HIE. It is of interest that a lesion in the right lung was reported in all four cases [1–4].

We thank van der Meer et al. for bringing to our attention the additional cases of aspergillosis in patients with HIE.

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References


Is the Free-Living Ameba Hartmannella Causing Keratitis?

Sir—In a letter recently published in Clinical Infectious Diseases we assert the fact that there is no evidence that the free-living ameba Hartmannella is a human parasite [1]. In the meantime, a brief report appeared on the possible involvement of Hartmannella in a mixed infection with Acanthamoeba causing keratitis [2]. Acanthamoeba is a proven etiologic agent of keratitis in humans, but we have demonstrated recently that in all previously reported cases in which Hartmannella was isolated, the pathogenicity of the isolate was not proven [3]. Therefore, as this ameba is widespread in the environment, Hartmannella isolates could be merely contaminants. Since several Hartmannella strains have been reported to be isolated from human dis-

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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 ameba 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.

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References


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Clinical Infectious Diseases 1998;27:1337–8  © 1998 by the Infectious Diseases Society of America. All rights reserved. 1058-4838/98/2705–0053$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).

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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.

References


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