In April 2003, a 66-year-old man living in Marseille indicated a history of fever for 8 days. He had returned from Vietnam 1 month earlier, where he had stayed for 4 weeks in the Ho Chi Minh City area and 2 days in the region of his visit. He had returned to Marseille 3 weeks after he returned to Vietnam and included fever (temperature 38°C), asthenia, chills (1 day), moderate dyspnea during exercise, transient bilateral pain of the testes, and an episode of hemopermia. He was referred by his family doctor to the Infectious and Tropical Diseases Unit, North Hospital.

On admission, the patient's temperature was 38°C. Physical examination of the patient, including the testes, was normal except for a systolic heart murmur (preexisting and known to the patient), and clinical signs of left pleural effusion. The effusion was subsequently confirmed by chest x-ray, which also showed a discrete diffuse bilateral lung infiltrate. Results of routine laboratory tests conducted on blood samples were normal except for an elevated eosinophil count of 5.2 x 10^9/L. Blood smears for plasmodia and microfilaremia were negative. Urologic examination, including echography and prostate-specific antigen, showed no abnormalities except a prostatic adenoma (preexisting and known to the patient). No eggs or parasites were detected by microscopic examination in stools or in urine, although both sedimented and centrifuged urine specimens were studied and filtration techniques were used. After transthoracic aspiration of 100 mL of pleural effusion, cytologic examination showed an eosinophil count of 5,800/L without parasites. Bacterial culture, including mycobacteria, was negative. On day 4, the patient was afebrile and was discharged. On admission, results of a first set of examinations involving reactivities to schistosomiasis, paragonimiasis, strongyloidiasis, cysticercosis, trichinosis, gnathostomiasis, filariasis, and toxocariasis were negative.

One month later, the eosinophil count of our patient had decreased to 1.8 x 10^9/L. He was afebrile, and his only complaint was asthenia. A new set of serologic examinations was conducted. The Western blot assay for gnathostomiasis conducted at the Swiss Tropical Institute (Socinstrasse 57, CH-4002, Basel, Switzerland) was positive, showing immunoglobulin G reactivity to four specific bands including the 24-kDa band, considered pathognomonic for the diagnosis of *Gnathostoma* infection (1). The seroconversion confirmed the diagnosis of gnathostomiasis. All other serologic tests remained negative, except an increase of antibodies against *Acanthocheilonema vitae* used as antigen for unspecific serologic screening for filariasis (Laboratoire Marcel Merieux, Lyon, France). After a 21-day course of albendazole and a single dose of ivermectin, the eosinophil count of our patient decreased to 0.8 x 10^9/L.

Three aspects of gnathostomiasis as an emerging imported disease can complement the findings of Moore et al. (1). First, the clinical findings in our case are very unusual. Hemospermia is often benign with predominant causes including prostatic and seminal vesicle disease. Infections, including mainly schistosomiasis and tuberculosis, have been associated with these symptoms (2). Although our patient had a prostatic adenoma, this is the first time that hemospermia has been associated with gnathostomiasis. Because of the anxiety hemospermia caused, this symptom was the main reason that our patient consulted our center. Secondly, eosinophilic pleural effusion is also unusual in gnathostomiasis. Although reported as a potential cause in reference books (3), a Medline search (key words: gnathostomiasis and eosinophilia and pleural effusion or pleuritis or lung) disclosed only two references to pleural effusion as the main symptom of gnathostomiasis (4,5).
The eosinophilic pleural and pulmonary response may be elicited by the larvae of helminths carried hematogenously into lungs and pleura in an aberrant fashion (3). The last point we stress is that, as shown by Moore et al. (1), patients returning from disease-endemic areas, mainly Southeast Asia and Central and South America, should be tested systematically for gnathostomiasis. Although some patients show a typical cutaneous form of gnathostomiasis associated with eosinophilia (6,7), most atypical forms are probably underdiagnosed, and severe neurologic involvement may occur if treatment is not given (1). However, until recently specific serologic tests for gnathostomiasis were available only in Asia, mainly in Thailand and Japan. Some laboratories in Europe currently provide testing for gnathostomiasis, which would be a valuable aid in evaluating patients returning from the tropics.

Philippe Parola,* Gerard Bordmann,† Philippe Brouqui,* and Jean Delmont*

*Hôpital Nord, Marseille, France; and †Swiss Tropical Institute, Basel, Switzerland

References


Address for correspondence: Philippe Parola, Service des Maladies Infectieuses et Tropicales, Hôpital Nord, AP-HM, Chemin des bourrelys, 13015, Marseille, France; fax: 33-4-91-96-89-38; email: philippe.parola@medecine.univ-mrs.fr

Methicillin-resistant Staphylococcus aureus, Pakistan, 1996–2003

To the Editor: This letter is written in response to the article titled “Co-trimoxazole-sensitive, methicillin-resistant Staphylococcus aureus, Israel, 1988–1997” (1). We found the authors’ findings most interesting. As the authors pointed out, methicillin-resistant Staphylococcus aureus (MRSA) infections have become a major problem worldwide. The problem is not restricted to industrialized countries. The last decade has seen an alarming increase in MRSA infections in Pakistani hospitals (2). Pakistan’s Armed Forces Institute of Pathology provides laboratory services to a 1,500-bed tertiary-care hospital in Rawalpindi and is the main reference laboratory in northern Pakistan. According to our computerized database, the frequency of MRSA among all nosocomial isolates of S. aureus increased from 39% (212/543) in 1996 to 51% (516/1,018) in 2003 (p < 0.0001). Most of the isolates were obtained from pus and pus swab specimens (153 in 1996 and 394 in 2003), while the rest were obtained from blood (20 in 1996 and 37 in 2003), intravenous catheter tips and surgical drainage tubes (14 in 1996 and 31 in 2003), various body fluids (9 in 1996 and 19 in 2003), respiratory secretions (8 in 1996 and 18 in 2003), tissue (4 in 1996, 9 in 2003), throat swabs (2 in 1996, 6 in 2003), and urine (2 in 1996, 5 in 2003).

During the last 7 years, resistance in MRSA isolates has steadily increased to most of the antimicrobial drugs such as gentamicin (69% in 1996 and 88% in 2003), ciprofloxacin (87% in 1996 and 94% in 2003), clindamycin (60% in 1996 and 70% in 2003), and rifampicin (20% in 1996 and 60% in 2003). However, resistance to co-trimoxazole and doxycycline has decreased. In 1996, 15% (32/212) of our MRSA isolates were susceptible to co-trimoxazole, whereas in the first 9 months of 2003, 43% (222/516) of the isolates were susceptible (p < 0.0001). Similarly, susceptibility to doxycycline increased from 34% in 1996 to 49% in 2003 (p = 0.0005). Antimicrobial drug susceptibility of the isolates was tested by the modified Kirby-Bauer technique and results were interpreted according to the National Committee for Clinical Laboratory Standards criteria (3). Methicillin resistance was tested by using 1 µg oxacillin disks (Oxoid, Basingstoke, Hampshire, UK) on Mueller-Hinton agar containing 4% sodium chloride. Plates were incubated at 35°C for 24 hours. We agree with Bishara et al. (1) that the increase in susceptibility is likely due to decreased use of these antimicrobial drugs for staphylococcal infections in clinical practice. The use of co-trimoxazole in our hospital decreased from 48 daily doses per 1,000 hospital days in 1996 to 35 daily doses in 2003, while use of doxycycline decreased from 12 daily doses per 1,000 hospital days in 1996 to 9 daily doses in 2003 (4). These antimicrobial drugs offer an inexpen-