Rhodotorula glutinis-Related Meningitis

Rhodotorulae are members of the family *Cryptococcaceae*, subfamily *Rhodotorulodeae*; although several species have been described, *Rhodotorula glutinis* and *Rhodotorula rubra* are the most common. *Rhodotorula* spp. are common saprophytes, widespread in nature. They have accidentally been isolated in human biological materials without pathological significance: they have been found in sputum and urine, especially in patients with severe debilitating diseases, in blood from patients with permanent intravenous devices, and in saliva obtained from denture wearers.

Only rarely has an etiological role been attributed to these yeasts, and in all cases infection was clearly opportunistic (1–6). The following case is a R. glutinis-related meningitis in an immunocompetent patient that was successfully treated with amphotericin B.

The patient was a 69-year-old white male (retired farmer) who was in his usual state of good health until 5 days before admission, when he developed persistent fever and headache. At physical examination, clear meningeal signs were observed, while the first hematochemical tests were not significant. Three separate blood cultures from a peripheral vein and a lumbar puncture (LP) were performed. Blood specimens were inoculated into Bactec Plus Aerobic/F and Bactec Anaerobic/F vials, which were placed in the BACTEC 9249 instrument and held for 8 days. The LP yielded clear cerebrospinal fluid (CSF), with a protein content of 0.82 g/liter, normal glucose concentration (and normal glycemia), and 30 lymphocytes/µl. A Gram stain of the CSF was negative, as was a search for capsular antigens for Neisseria spp., pneumococcus, Haemophilus spp., and Cryptococcus neoformans; cultures of CSF for bacteria, mycobacteria, and fungi were also obtained. Empirical therapy with ceftriaxone, ampicillin, and acyclovir was started. Serologic tests for herpes, parotitis, enterovirus, human immunodeficiency virus, and treponemes were negative. Moreover, the Mantoux test, the chest x-ray, and a cerebral computerized tomography scan had no significant findings. After 3 days, both blood and CSF culture results were still negative, with the exception of the growth of one colony of R. glutinis in CSF. R. glutinis grew on Sabouraud dextrose agar containing chloramphenicol and gentamicin and was identified by the API 32-C System Biomerieux (API 32-C Bio-Merieux). The reported organism was considered a contaminate and therefore neglected. Because of the persisting symptoms, on day 5 of the hospital stay, a second LP was performed. The CSF analysis revealed a clear fluid, proteins at 0.9 g/liter, normal glucose (and normal glycemia), and 40 lymphocytes/µl. Once again, after 3 days of incubation at 37°C, the Sabouraud dextrose agar showed the growth of three colonies of R. glutinis. On day 8 of the hospital stay, the previous therapy was stopped and the

patient was treated intravenously with amphotericin B at dose of 1 mg/kg/day. In 3 days, the patient's temperature was back to normal, with a progressive improvement of clinical condition. When the CSF was examined 10 days after the beginning of the antifungal treatment, the patient's condition was entirely recovered. A total dose of 1.5 g of amphotericin B was administered without any significant adverse reaction.

The possible connections between *Rhodotorula* spp. and human pathology have been discussed in the past, with considerable difficulty in identifying lesions pathognomonic for the organism, even in the case of postmortem examinations.

Furthermore, there have been reports of *Rhodotorula*-related diseases resolved without the use of an antifungal treatment (4).

In our patient, it may be difficult to deny the pathologic role of this yeast; the clinical picture, the mycological findings, and the prompt response to therapy fit well together.

Interestingly, no predisposing diseases were present (with the exception of old age, a predisposing factor in the broad sense). Further clinical studies will be necessary to establish whether *R. glutinis* may be considered an emergent pathogen.

REFERENCES

- 1. Donald, F. E., J. F. Sharp, J. L. Firth, J. L. Crowley, and P. Ispahani. 1988. *Rhodotorula rubra* ventriculitis. J. Infect. 16:187–191.
- Eisenberg, E. S., B. E. Alpert, R. A. Weiss, N. Mittman, and R. Soeiro. 1983. *Rhodotorula rubra* peritonitis in patients undergoing continuous ambulatoryperitoneal dialysis. Am. J. Med. 75:349–352.
- Fanci, R., P. Pecile, R. L. Martinez, A. Fabbri, and P. Nicoletti. 1997. Amphotericin B treatment of fungemia due to unusual pathogens in neutropenic patients: report of two cases. J. Chemother. 9:427–430.
- Pien, F. D., R. L. Thompson, D. Deye, and G. D. Roberts. 1980. Rhodotorula septicemia: two cases and a review of the literature. Mayo Clin. Proc. 55:258– 260.
- Wong, V., L. Ross, L. Opas, and E. Lieberman. 1988. *Rhodotorula rubra* peritonitis in a child undergoing intermittent cycling peritoneal dialysis. J. Infect. Dis. 157:393–394. (Letter.)
- Young, R. C., J. E. Bennet, G. W. Geelhoed, and A. S. Levine. 1974. Fungemia with compromised host resistance. A study of 70 cases. Ann. Intern. Med. 80: 605–612.

Massimiliano Lanzafame Giovanna De Checchi Antonino Parinello Unit of Infectious Diseases Legnano, Italy

Marco Trevenzoli Anna Maria Cattelan* Division of Infectious Diseases General Hospital of Padua Azienda Ospedaliera Via Giustiniani, 2 35100 Padua, Italy

*Phone: 39-049-8213763 Fax: 0039-049-8213768 E-mail: aacattelan@hotmail.com