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Mycobacterium chelonei Iatrogenic Infections

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We report on two outbreaks of *Mycobacterium chelonei* subsp. *abscessus* cutaneous infections, which occurred between June 1974 and April 1975 in a series of 24 patients (15 studied bacteriologically) subjected to venous stripping for varicose veins. The source of infection was the aqueous solution of merbromin used in presurgical care.

In revaluating dermatological infections by *Mycobacterium tuberculosis*, which have become much less frequent, Feldman and Hersfield (3) emphasize the presence of cutaneous infections by other mycobacteria such as *M. ulcerans*, *M. marinum*, and *M. fortuitum* and less often by *M. kansasii* and *M. intracellulare*. Appearance of postinfection abscesses and cutaneous and ganglionar infections due to *M. chelonei* has been reported (6, 7).

The outbreaks that we observed in Barcelona and the two outbreaks in North Carolina and Colorado in February-April and April-May 1976, respectively, as reported by Hines et al. (4) suggest that this type of infection may be more frequent than was supposed.

In this report we describe two outbreaks of *M. chelonei* subsp. *abscessus* cutaneous infections seen in Barcelona in patients subjected to venous stripping for varicose veins. The first, which took place between June and December 1974, included 13 patients (11 women and 2 men); of them, only 4 were studied bacteriologically. The second outbreak, January-April 1975, included 11 women, all studied bacteriologically. Both outbreaks happened in two different Barcelona hospitals, under the care of two surgical teams.

In all cases, symptoms started within 20 to 90 days after the stripping, with appearance over the line of the removed vein, and occasionally near the incision, of hard oval nodes surrounded by an area of inflammation. These nodes softened later on and fluctuated, and some opened spontaneously to drain a thick pus.

There were remarkable differences in the number and severity of the lesions as well as in the clinical evolution of the patients. Some presented only one or two nodes, which healed for good 2 or 3 weeks after drainage. The others developed disseminated lesions on the leg, with multiple nodes in different evolutive stages and with appearance of new ones after some of the first had been cured. In most of these patients the lesions went on for months, losing intensity and with new abscesses appearing with diminishing frequency. Healing was obtained, although in two of the patients new nodes still appear occasionally 2.5 years after the start of the process.

No clear-cut effects have been observed following institution of systemic antituberculous therapy with streptomycin, isoniazid, and *p*-aminosalicylic acid, nor with topical application of iodine solution.

Pathological studies were performed in two patients. One of the specimens consisted of a small sample of dermal and epidermal tissue in which only a nonspecific inflammatory infiltrate was evident. The other sample showed an invasion of the dermis by pseudotuberculous nodes with epithelioid and Langhans cells and with a polymorphonuclear infiltrate, but without signs of caseation.

The microscopic examination of the pus, either drained or obtained by aspiration, of the 15 patients studied showed many polymorphonuclear leukocytes. In all of the specimens, Ziehl-Neelsen staining showed acid-fast bacilli with features typical of mycobacteria. By inoculation of this material on blood agar and Löwenstein-Jensen medium we obtained, in the 4 specimens of the first outbreak and in 10 of the 11 of the second, growth of gram-positive, acid-fast bacilli between 2 and 4 days.

These 14 isolates were subjected to differential studies for rapidly growing mycobacteria. The tests used, which were according to the techniques of Vestal (10) except the niacin test, for which we used niacin test strips from Difco, and the results obtained appear in Table 1. The 14 strains gave identical results and can be identified as *M. chelonei* subsp. *abscessus* (8).

Although a number of items were investigated

TABLE 1. *Biochemical characteristics of the 26 strains of mycobacteria studied*[a]

Test	Result			
	Patient isolates (14 strains)	Merbromin isolates[b] (2 strains)	Other merbromin isolates[c]	
			Group A (5 strains)	Group B (5 strains)
Pigment	N	N	N	N
Growth on MacConkey agar	+	+	+	+
Arylsulfatase (3 day)	±/1+	±/1+	±/1+	2+/3+
Nitrate reduction	−	−	−	−
Iron uptake	−	−	−	−
Growth on 5% NaCl	+	+	+	+(weak)
Tellurite reduction	+	+	+	+
Tween hydrolysis	−	−	−	−
Catalase (drop method)	+	+	+	+
Niacin	−	−	−	−

[a] +, Positive; −, negative; N, nonchromogenic.
[b] Isolated from merbromin solutions in use in the hospitals where the outbreaks took place.
[c] Isolated from merbromin solutions of other origins.

as possibly contaminated with mycobacteria, the source of infection was ultimately traced to aqueous solution of 2′,7′-dibromo-4′-hydroxymercury fluorescein (merbromin), which was being used as a skin disinfectant in presurgical care.

The microscopic study of the sediment of merbromin solutions used in the two operating theaters showed presence of acid-fast bacilli resembling those seen in the pus from the patients' lesions. Culture of the merbromin solutions on blood agar and Löwenstein-Jensen medium was positive for gram-positive, acid-fast bacilli in 3 to 6 days. These two strains were also identified as *M. chelonei* subsp. *abscessus* (Table 1).

We also isolated 10 additional strains of *M. chelonei* subsp. *abscessus* from merbromin solutions in use in medical centers other than those involved in the reported outbreaks and from commercial bottles. In the differential studies, five of these strains (Table 1, group A) showed the same pattern as the strains isolated from the patients' pus and from merbromin solutions in use in the operating theaters where the outbreaks took place. The remaining five (Table 1, group B) showed slight variations in the arylsulfatase test and in their growth on 5% NaCl.

We studied the susceptibility to *p*-aminosalicylic acid, isoniazid, streptomycin, rifampin, ethionamide, and ethambutol of the 26 strains of mycobacteria by following the technique of Canetti et al. (2), and all of them turned out to be resistant to the six drugs. This may explain the patients' poor response to antituberculous therapy. We have also studied their susceptibility to 25 other antimicrobial agents (by diffusion technique on Mueller-Hinton blood-agar plates), and all of them showed only slight susceptibility to kanamycin and erythromycin, the same two drugs that showed some effect in the outbreaks reported by Hines et al. (4).

Since the use of merbromin was banned, new cases have not been observed by either of the two surgical teams.

Although the possibility of contamination of the solution during the preparation cannot be excluded, the multiplicity of sources of merbromin, quite apart from those within the city, strongly suggests that the initial source could well be the merbromin powder itself.

The antiseptic activity of merbromin has been reported to be quite weak (9), and to some extent it is not surprising that an aqueous solution could harbor an agent such as *M. chelonei* if not properly sterilized. In fact, the growing of pathogenic bacteria in several kinds of antiseptics has been described (1, 5). However, the unfortunate series of cases reported here should perhaps be regarded not as a microbiological curiosity but as a further warning against a naive reliance on the microorganism-killing capacity of many antiseptics.

ADDENDUM

These observations were communicated in May 1975 at a meeting of the Clinical Microbiology Group of the Spanish Microbiology Society. At that time we did not know the correct identification of the mycobacteria isolated, or their susceptibility to the drugs.

LITERATURE CITED

1. **Ayliffe, G. A. J., D. R. Barry, E. J. L. Lowbury, M. J. Roger-Hall, and W. M. Walker.** 1966. Postoperative infection with *Pseudomonas aeruginosa* in an eye hospital. Lancet i:1113-1117.
2. **Canetti, G., N. Rist, and J. Grosset.** 1963. Mesure de la sensibilité du bacille tuberculeux aux drogues antibacillaires par la méthode des proportions. Rev. Tuberc. Pneumol. 27:217-272.
3. **Feldman, R. A., and E. Hersfield.** 1974. Mycobacterial

skin infection by an unidentified species. A report of 29 patients. Ann. Intern. Med. **80**:445–452.

4. **Hines, M. P., R. Waggoner, and T. M. Vernon.** 1976. Atypical Mycobacteria wound infections—Nortn Carolina, Colorado. Morbid. Mortal. Week. Rep. **25**:238, 243.
5. **Lowbury, E. J. L.** 1951. Contamination of cetrimide and other fluids with *Pseudomonas pyocyanea*. Br. J. Ind. Med. **8**:22–26.
6. **Morris, C. A., G. H. Grant, P. H. Everall, and A. T. Myres.** 1973. Tuberculous lymphadenitis due to *Mycobacterium chelonei*. J. Clin. Pathol. **26**:422–426.
7. **Pattyn, S. R., J. Vandepitte, F. Portaels, and A. de Muynck.** 1971. Cases of *Mycobacterium borstelense* and *M. abscessus* infection observed in Belgium. J. Med. Microbiol. **4**:145–149.
8. **Runyon, E. H., L. G. Wayne, and G. P. Kubica.** 1974. Family II. *Mycobacteriaceae* Chester 1897, 63, p. 681–701. *In* R. E. Buchanan and N. E. Gibbons (ed.), Bergey's manual of determinative bacteriology, 8th ed. The Williams & Wilkins Co., Baltimore.
9. **Spaulding, E. H., and D. H. M. Gröschel.** 1974. Hospital disinfectants and antiseptics, p. 852–857. *In* E. H. Lennette, E. H. Spaulding, and J. P. Truant (ed.), Manual of clinical microbiology, 2nd ed. American Society for Microbiology, Washington, D.C.
10. **Vestal, A. L.** 1975. Procedures for the isolation and identification of Mycobacteria. Center for Disease Control publ. no. 75.8230. U.S. Department of Health, Education and Welfare, Washington, D.C.