Two Outbreaks of *Flavobacterium meningosepticum* Type E in a Neonatal Intensive Care Unit

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Two separate outbreaks due to *Flavobacterium meningosepticum* type E occurred in a neonatal intensive care unit in March-April and July 1975. The first outbreak involved all five infants in the unit. Two infants developed meningitis, one had bacteremia, and two were colonized. During the second outbreak, five of seven infants were colonized but none developed disease. The upper respiratory tract was colonized first in most instances, and the organism persisted at this site for a mean of 17.3 days. Duration of colonization was more prolonged in infants receiving antibiotics than in untreated infants. Extensive environmental surveillance failed to demonstrate a reservoir; however, *F. meningosepticum* was recovered from three nasendotracheal tubes and from an aerosol tube before colonization of four infants. The organism was resistant to most antimicrobial agents tested and developed resistance to others during the treatment course of one infant. Although *F. meningosepticum* was not recovered from cultures of transport vehicles, several other gram-negative bacteria were isolated and were also resistant to multiple antibiotics.

Flavobacterium meningosepticum, a nonmotile, oxidative, gram-negative bacillus, was first classified by King in 1959 (6). It has been incriminated as a cause of serious infections in newborns (1-3, 7-9, 14, 18) and occasionally in adults (13, 19). In newborns, meningitis is the most common disease caused by this organism. Among the various serotypes of *F. meningosepticum* (A to F), type C has been the cause of most reported epidemics (2, 6).

Between March and July 1975, two separate *F. meningosepticum* type E outbreaks occurred in the neonatal intensive care unit (NICU) at our hospital. This report describes the epidemiological and bacteriological investigations of these outbreaks, with primary emphasis on the site sequence of colonization with the organism and on environmental culturing.

MATERIALS AND METHODS

Human and environmental cultures. Cultures from human subjects were obtained with sterile swabs. Swabs were premoistened in sterile tryptic soy broth to obtain environmental cultures. Fluids (50-ml samples) were filtered through 0.45- μ m membrane filters (Millipore Corp., Bedford, Mass.). The surface of the filter was swabbed and used for culture. Specimens were planted on sheep blood and MacConkey agar plates.

Identification of flavobacteria. The flavobacteria are oxidase-positive, nonmotile, oxidative, gram-negative bacilli. Since the biochemical reactions are

delayed and acid production is slight, these organisms were identified by using the buffered substrates designed for the identification of nonfermentative, gram-negative bacilli. Substrates were inoculated heavily and incubated at 35 to 37°C for 24 to 72 h. By using the charts and tabulations supplied by the substrate manufacturers (Key Scientific Products Co., Los Angeles, Calif., and Callabs, North Hollywood, Calif.), differentiation of the flavobacteria into groups I, II, and III was made. Flavobacteria that were resistant to 2 U of penicillin and oxidized D-lactose and D-fructose were designated group I. Flavobacteria that were resistant to 2 U of penicillin and oxidized D-fructose but not D-lactose fell into group II. Group III consisted of flavobacteria that were susceptible to 2 U of penicillin.

F. meningosepticum and *Flavobacterium* IIb are included in group II. Although the biochemical reactions of these two organisms were similar, differentiation between the two was not difficult (16). Isolates of *Flavobacterium* IIb produced a marked yellow pigment, did not oxidize D-mannitol, and took 3 or more days to liquefy gelatin. *F. meningosepticum* isolates had a pale-yellow pigment that developed only after the cultures were held at room temperature for several days. The isolates also produced acid from D-mannitol and liquefied gelatin in 24 h. Both *F. meningosepticum* and *Flavobacterium* IIb were indole positive and neither grew on MacConkey agar in 48 h. For the purpose of this paper, flavobacteria other than these two species will be referred to as *Flavobacterium* sp.

Antibiotic susceptibilities. The method used for the antibiotic susceptibility testing of the isolates was the modified Kirby-Bauer technique currently recommended by the U.S. Food and Drug Administration

(4, 5) and the National Committee for Clinical Laboratory Standards (10). Mueller-Hinton agar was inoculated with an actively growing culture, the turbidity of which had been adjusted to match that of a no. 0.5 MacFarland standard. Standard disks were applied, and the plates were incubated aerobically at 35 to 37°C for 16 to 18 h. A template and transmitted light were used to facilitate measurement of the zone diameters. Inhibition zones were compared to interpretive standards (10). In addition, representative isolates from each patient were tested at the Antimicrobics Investigations Section, Bacteriology Division, Center for Disease Control, Atlanta, Ga., by the modified Kirby-Bauer and tube dilution susceptibility methods.

RESULTS

Bacteriological findings. The epidemic strain of *F. meningosepticum* was a nonmotile, oxidative, gram-negative bacillus. Colonies were smooth, glistening, and mucoid on 10% sheep blood agar. A pale-yellow pigment was discernible only after the cultures were held at room temperature for 5 days. There was no growth on MacConkey agar in 24 to 48 h, although slight growth was apparent after 5 to 7 days. Oxidase and catalase were produced. Growth on triple sugar iron agar revealed an alkaline slant, with no change in the butt. The strain produced acid from D-glucose, D-mannitol, and D-maltose; acid was not produced from D-lactose, D-xylose, and D-sucrose. Nitrate was not reduced and citrate was not utilized. Litmus milk was peptonized and esculin hydrolysis was positive. Urea and H₂S were negative. Gelatin was liquefied within 24 h.

The isolates produced small amounts of indole, and identification of the organisms was delayed because of a false-negative indole test. Since the marked yellow pigment of the other flavobacteria is not produced by *F. meningosepticum*, the indole test is extremely important in differentiating *F. meningosepticum* from other oxidase-positive, nonfermentative and weakly fermentative, gram-negative bacilli. We used two different indole substrates (Key Scientific Products and Callabs) and three different reagents (r/b Kovacs reagent, Corning Medical Diagnostics, Roslyn, N.Y.; API Kovacs reagent, Analytab Products, Inc., Plainview, N.Y.; and Ehrlich reagent, Harleco, Gibbstown, N.J.); a strongly positive indole test resulted when Ehrlich reagent was added to the Key Scientific Products indole substrate.

Identification of the initial isolates as *F. meningosepticum* was facilitated through the expeditious service of the Special Bacteriology Section, Bacteriology Division, Center for Disease Control. Representative isolates of *F. meningosepticum* from each case were also referred to the same Section and were all identified as *F.*

meningosepticum type E.

General description of the first outbreak. The first appearance of *F. meningosepticum* type E occurred on 24 March 1975, from a nasoendotracheal (NET) tube culture of an infant in the NICU. Because this isolate was resistant to ampicillin, carbenicillin, cephalothin, gentamicin, kanamycin, tetracycline, colistin, chloramphenicol, and streptomycin, the medical and nursing staff of the unit were alerted and the patient was put in strict isolation. The infant had no evidence of infection at the time. On 6 April, a blood culture from another premature infant in NICU grew *F. meningosepticum* type E with an identical antibiogram.

The NICU and the premature-newborn nursery occupy the same hospital wing, but are separated by a common corridor (Fig. 1). Each unit has an additional access via another corridor not shared by the other unit. In an effort to limit the possible spread of the organism, the NICU was closed to admissions on 9 April. Center corridor exits from room A and the nurses' station in the NICU were closed. Traffic to and from the unit was limited to the outside corridor through rooms B and C. There was no exchange of personnel between the NICU and other areas of the hospital, including the nursery. Five infants remained in the NICU. All were subsequently found to be colonized with *F. meningosepticum* type E. Further details about these infants will be presented later in this paper.

By 6 May, only one infected infant remained in the NICU. He was relocated alone in the overflow area (Fig. 1) and kept in strict isolation. The NICU was thoroughly cleaned and reopened to admissions.

General description of second outbreak. After the reopening of the NICU, all infants admitted to this unit were monitored by bi-weekly oropharyngeal and rectal cultures. No *F. meningosepticum* was recovered until 14 July, when an NET-tube culture of a patient admitted on 26 June yielded the organism. Previous cultures of the NET tube on this patient were negative. The patient was placed in strict isolation.

FIG. 1. Floor plan of wing containing NICU and nursery.

tion, and the NICU was again closed to admissions on 16 July. At the time, seven infants were in NICU, five of whom were colonized.

The second outbreak ended on 30 July, and the NICU was again opened to admissions. Since then there has been no reappearance of the organism.

Colonization and clinical observations of affected infants. Intensive monitoring of infants in the NICU after identification of the index cases during the two outbreaks provided an opportunity to investigate the site sequence of colonization with *F. meningosepticum*. Frequent cultures, often daily, of the oropharynx, rectum, and NET tubes were obtained. Blood and cerebrospinal-fluid cultures were obtained from all patients twice a week, or as indicated. Table 1 summarizes the colonization data on the 10 affected infants. Infants were initially colonized from 1 to 20 days after admission (mean, 9.9 days). *F. meningosepticum* was first isolated from NET tube cultures in four of eight infants who were intubated. In addition, the organism was isolated from an aerosol tube (case 4) 3 days before its recovery from other sites in that infant. The respiratory tract was the first

site colonized in seven infants, and was eventually colonized in all but one infant (case 10). Sequential observations were available in nine cases, and in eight of these the organism persisted in the respiratory tract for a mean of 17.3 days. In case 10, colonization was restricted to the gastrointestinal tract. The gastrointestinal tract was colonized in seven of the nine infants, for a mean of 4.7 days. In case 3, *F. meningosepticum* was repeatedly isolated from cerebrospinal fluid over a 39-day period despite intensive antimicrobial therapy.

Of the 10 infants affected, only 3 in the first outbreak exhibited disease directly related to *F. meningosepticum* (Table 1). The remaining two infants in March-April and all five infants in July were colonized but not infected. The three diseased infants (cases 2, 3, and 4) had positive blood cultures. Two developed meningitis; one died within 6 days (case 2), and the other survived but developed hydrocephalus requiring a ventriculoperitoneal shunt (case 3). The one patient who did not develop meningitis had only a transient bacteremia, was treated with appropriate antibiotics, and eventually was discharged home (case 4).

TABLE 1. Initial and subsequent colonization with *F. meningosepticum* type E in 10 infants during both outbreaks

Case	Date first positive	Days between admission and initial colonization	First site positive	Days between first and subsequent site	Subsequent site sequence	Days of colonization	Outcome
1	24 March	18	NET tube	1	Oropharynx, rectum	36	Died ^a
2	6 April	6	Blood	1	Meninges, oropharynx	6	Died of meningitis
3	9 April	3	Oropharynx and rectum	1	Blood, NET tube, meninges	39	Hydrocephalus
4 ^b	9 April	1	Aerosol tube	3	Oropharynx, rectum, blood	20	Well
5 ^b	12 April	11	Oropharynx	2	Rectum	17	Well
6	14 July	19	NET tube		None	10	Well
7	16 July	8	NET tube	8	Rectum	9	Well
8	17 July	6	NET tube	8	Oropharynx	11	Well
9 ^b	22 July	7	Rectum	3	Oropharynx	15	Well
10	28 July	20	Rectum		None	7	Well

^a Death unrelated to *F. meningosepticum*.

^b No NET tube used.

Environmental surveillance. Attempts were made during both outbreaks to recover *F. meningosepticum* from sources other than the infants in the NICU. Cultures from patients in the nursery, NICU and nursery personnel, and of various inanimate objects were obtained.

All infants admitted to the NICU from the time of recognition of the index cases until closure of the unit were identified. The families of patients who had been discharged were contacted by telephone, and all infants were seemingly well. Infants transferred from the NICU to the adjacent nursery and all other nursery patients were cultured. Specimens for culture were obtained from the oropharynx, rectum, and umbilicus of each infant. The infants were monitored biweekly and on discharge. None of the cultures yielded *F. meningosepticum*.

Oropharyngeal, rectal, and hand cultures were obtained from all personnel in the NICU and the nursery. Ancillary personnel who had patient contact in the NICU were similarly cultured. In total, 180 cultures were obtained, and all were negative for *F. meningosepticum*.

Cultures of inanimate objects in the NICU were collected. These included sink drains and traps, water faucets, sink basins, ice machine, oxygen and air outlets, floors, walls, solutions, medications, lubricating jelly, clean diapers, plants, formula, catheters, soap dispensers, and unused aerosol and NET tubing. *F. meningosepticum* was not isolated from any of these cultures.

Since patient monitoring revealed colonization of NET and aerosol tubes with *F. meningosepticum*, concentrated efforts were directed at investigating respiratory therapy equipment. The organism was not isolated from saline or water bottles used by inhalation therapists. In five of eight patients requiring respiratory equipment, the organism was first recovered from NET or aerosol tubing, and subsequent coloni-

zation occurred in four of these infants. Review of records of all patients admitted to the NICU revealed that colonization with *F. meningosepticum* occurred only in patients who had some form of respiratory assistance. In addition, cultures of nebulizer water in use in two infants yielded *F. meningosepticum* 2 and 3 days after the infants were colonized with the organism. The organism was also recovered on one occasion from suction fluid of an affected patient; however, this was judged to be secondary to the patient's colonization.

All infants admitted to the NICU were transferred from other institutions. Review of admission records of the 10 affected infants indicated that they were transferred from eight separate hospitals. Efforts were then directed to determine whether transport vehicles could be the source of the offending organism. Our hospital transport vehicle and three commercial ambulances were used to transport the infants. Cultures of suction apparatus, air vents, isolettes, and surface of inanimate objects were obtained from all vehicles. The hospital transport vehicle contains a sink with a water reservoir and stock supplies. Cultures of the water from the reservoir, sink drain and trap, bar soap, saline, and water bottles were obtained. Although *F. meningosepticum* was not isolated from any source, several gram-negative organisms were recovered (Table 2). One commercial ambulance was free of these potentially pathogenic organisms.

Antibiotic therapy and susceptibility patterns. The antibiotic susceptibility patterns of all *F. meningosepticum* type E isolates in the first and second outbreaks were identical. The isolates were susceptible to sulfisoxazole, erythromycin, vancomycin, minocycline, rifampin, trimethoprim-sulfamethoxazole, and clindamycin, but were resistant to penicillin G, carbenicillin, cephalothin, gentamicin, tetracycline, colistin, chloramphenicol, streptomycin, spectinomycin,

TABLE 2. *Gram-negative bacteria recovered from transport vehicles*

Vehicle	Source	Organisms recovered ^a
Commercial ambulance no. 1	Humidifier in isolette	<i>Flavobacterium</i> sp. Unidentified gram-negative rod
Commercial ambulance no. 2	Humidifier sponge in isolette	<i>Flavobacterium</i> IIb
Children's Hospital transport vehicle	Suction machine	<i>Flavobacterium</i> IIb <i>Pseudomonas pickettii</i>
	Water reservoir	<i>Flavobacterium</i> IIb <i>Alcaligenes faecalis</i>
	Sink trap	<i>Flavobacterium</i> IIb <i>Pseudomonas diminuta</i> <i>Pseudomonas</i> sp.
	Tap water	<i>Pseudomonas</i> sp.

^a All isolates were confirmed by the Special Bacteriology Unit, Center for Disease Control.

neomycin, and tobramycin. Susceptibility patterns were determined for the gram-negative organisms recovered from the transport vehicles. These organisms were resistant to most antibiotics, and the patterns were almost identical to that of *F. meningosepticum* type E.

Figure 2 shows the colonization pattern in one of the diseased infants (case 3) with *F. meningosepticum* type E. Also depicted is a summary of the antimicrobial agents used to treat the patient. Susceptibility to minocycline, rifampin, and trimethoprim-sulfamethoxazole was not determined till late in April, hence the delay in using these drugs. Noteworthy is the observation that in this patient the organism developed resistance to sulfisoxazole, erythromycin, and vancomycin shortly after or during the administration of these drugs. All antibiotics were administered intravenously. Vancomycin and erythromycin were also administered intraventricularly for 5 days each. Resistance developed to clindamycin although it was not used in the treatment of this patient. The change in susceptibility to the antibiotics as measured by the modified Kirby-Bauer method was confirmed by determining the minimum inhibitory concentrations against two representative isolates. One isolate on 9 April (susceptible) and another on 6 May (resistant) were compared (Table 3).

FIG. 2. Colonization of various sites with *F. meningosepticum* type E in case 3. Five patterns of susceptibility (S) or resistance (R) of the organism to certain antimicrobial agents and the duration of administration ($\leftrightarrow$) of these agents are shown.

TABLE 3. Minimum inhibitory concentrations (MIC) against *F. meningosepticum* before and after development of resistance

Antibiotic	MIC (μ g/ml) of:	
	Susceptible strain	Resistant strain
Clindamycin	0.5	8.0
Erythromycin	4.0	64.0
Vancomycin	8.0	64.0

The effect of antimicrobial therapy on the duration of colonization with *F. meningosepticum* was determined. Four colonized infants did not receive specific therapy (cases 6, 7, 8, and 10). The mean duration of colonization was 9.3 days. In cases 1, 5, and 9, who were colonized and received specific antibiotic treatment, the mean duration of colonization was 22.7 days.

DISCUSSION

Infections due to *F. meningosepticum* have been reported primarily in neonates (1, 2, 6, 9, 14, 18). Meningitis is the primary manifestation and occurs equally in premature and full-term infants (3). In previous reports, serotype C was predominant (2, 6), although disease due to types A, B, and F has been recognized (6, 18, 19). To our knowledge, only one case of neonatal meningitis due to type E has been reported previously (20).

Both outbreaks in this report were confined to the NICU and involved only infants receiving respiratory assistance. The respiratory tract was the primary colonization site in most instances. In five infants, cultures from four NET tubes and an aerosol tube were the first to yield the organism. However, extensive surveillance failed to identify a human or an environmental reservoir. Although respiratory equipment was not the reservoir for *F. meningosepticum*, there was substantial evidence to incriminate such equipment as an important link in the perpetuation of the outbreaks. Before these outbreaks, the respiratory equipment on intubated patients was changed every 72 h. For patients who were not intubated but required respiratory assistance, the change interval for oxygen hoods, masks, and tubings was not specified but commonly occurred every 5 to 7 days. Therefore, a revised policy was instituted for respiratory equipment maintenance at our hospital. Equipment concerned with humidifying or administering gases is changed every 24 h. For intubated patients, this includes all tubing, humidifiers, nebulizers, connectors, and various other equipment in contact with the airway. For nonintubated patients, items such as oxygen hoods, bag and mask, nebulizers, humidifiers, tent canopies, and aerosol tubing are also changed every 24 h. Since institution of this revised policy, there has been no recurrence of *F. meningosepticum*.

Reservoirs of *F. meningosepticum* have been identified on some occasions. Cabrera and Davis isolated the organism from a sink drain in the anteroom of a premature nursery (2). A rubber hose attached to a faucet in a delivery room was another reservoir (17). *F. meningosepticum* was also recovered from saline solution used to rinse infants' eyes after silver nitrate application

(14). In another epidemic involving eight adults, the organism was grown from multiple-dose vials of drugs (11). Indwelling arterial catheters have been described recently as a source of nosocomial bacteremia caused by *Flavobacterium* IIb (15). Outside the hospital environment, *F. meningosepticum* has been isolated from brook water and soil (12). The recovery of *F. meningosepticum* from respiratory equipment has not been documented previously.

In our search for reservoirs of *F. meningosepticum*, potentially pathogenic, gram-negative bacteria in transport vehicles were found. This suggests a possible hazard that may exist in such vehicles and their life-sustaining equipment. The water reservoir and sink in the Children's Hospital transport vehicle were therefore eliminated. Suction equipment and the isolette are now removed and thoroughly cleansed after each use. The owners of the commercial ambulances were informed of the contamination found in their isolettes and were advised on methods for decontamination.

The changing pattern of antimicrobial susceptibility of the organism in case 3 was a most unusual observation. The organism from cerebrospinal fluid developed resistance to sulfisoxazole, erythromycin, vancomycin, and clindamycin. The observations made in our laboratory were confirmed by the Antimicrobics Investigation Section at the Center for Disease Control. Several other gram-negative bacteria were isolated from environmental cultures and from the respiratory and gastrointestinal tracts of the infants in the NICU. Some of these bacteria were resistant to many antibiotics. It is tempting to postulate that *F. meningosepticum* acquired a resistance factor from some other organism in the environment. This assumption cannot be confirmed, however, since erythromycin, vancomycin, and clindamycin are not ordinarily included as part of the antibiogram of gram-negative bacilli, and the original isolates are not available for further testing.

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LITERATURE CITED

1. Brody, J. A., H. Moore, and E. O. King. 1958. Meningitis caused by an unclassified gram-negative bacterium in newborn infants. *Am. J. Dis. Child.* **96**:1-5.
2. Cabrera, H. A., and G. H. Davis. 1961. Epidemic meningitis of the newborn caused by *Flavobacterium*. I. Epidemiology and bacteriology. *Am. J. Dis. Child.* **101**:289-295.
3. Eykens, A., E. Eggermont, R. Eeckels, J. Vandepitte, and J. Spaepen. 1973. Neonatal meningitis caused by *Flavobacterium meningosepticum*. *Helv. Paediatr. Acta* **28**:421-425.
4. Federal Register. 1972. Rules and regulations. Antibiotic susceptibility discs. *Fed. Regist.* **37**:20525-20529.
5. Federal Register. 1973. Rules and regulations. Antibiotic susceptibility discs correction. *Fed. Regist.* **38**:2576.
6. King, E. O. 1959. Studies on a group of previously unclassified bacteria associated with meningitis in infants. *Am. J. Clin. Pathol.* **31**:241-247.
7. Lapage, S. P., and R. J. Owen. 1973. *Flavobacterium meningosepticum* from cases of meningitis in Botswana and England. *J. Clin. Pathol.* **26**:747-749.
8. Maderazo, E. G., H. P. Bassaris, and R. Quintiliani. 1974. *Flavobacterium meningosepticum* meningitis in a newborn infant. *J. Pediatr.* **85**:675-676.
9. Madruga, M., U. Zanon, G. M. N. Pereira, and A. C. Galvão. 1970. Meningitis caused by *Flavobacterium meningosepticum*. The first epidemic outbreak of meningitis in the newborn in South America. *J. Infect. Dis.* **121**:328-330.
10. National Committee for Clinical Laboratory Standards. 1974. Performance standards for antimicrobial disc susceptibility tests. Philadelphia, Pa.
11. Olsen, H. 1967. An epidemiological study of hospital infection with *Flavobacterium meningosepticum*. *Dan. Med. Bull.* **14**:6-9.
12. Olsen, H. 1969. *Flavobacterium meningosepticum* isolated from outside hospital surroundings and during examination of patient specimens. *Acta Pathol. Microbiol. Scand.* **75**:313-322.
13. Olsen, H., W. C. Frederiksen, and K. E. Siboni. 1965. *Flavobacterium meningosepticum* in 8 non-fatal cases of postoperative bacteraemia. *Lancet* **i**:1294-1296.
14. Plotkin, S. A., and J. C. McKittrick. 1966. Nosocomial meningitis of the newborn caused by a *Flavobacterium*. *J. Am. Med. Assoc.* **198**:194-196.
15. Stamm, W. E., J. J. Colella, R. L. Anderson, and R. E. Dixon. 1975. Indwelling arterial catheters as a source of nosocomial bacteremia. An outbreak caused by *Flavobacterium* species. *N. Engl. J. Med.* **292**:1099-1102.
16. Tatum, H. W., W. H. Ewing, and R. E. Weaver. 1974. Miscellaneous gram-negative bacteria, p. 270-286. In E. H. Lennette, E. H. Spaulding, and J. P. Truant (ed.), *Manual of clinical microbiology*. American Society for Microbiology, Washington, D.C.
17. Vandepitte, J., G. Beeckmans, and R. Butiaux. 1958. Quatre cas de méningite à *Flavobacterium* chez des nouveau-nés. *Ann. Soc. Belge Med. Trop.* **38**:563-570.
18. Watson, K. C., J. G. Krogh, and D. T. Jones. 1966. Neonatal meningitis caused by *Flavobacterium meningosepticum* type F. *J. Clin. Pathol.* **19**:79-80.
19. Werthamer, S., and M. Weiner. 1972. Subacute bacterial endocarditis due to *Flavobacterium meningosepticum*. *Am. J. Clin. Pathol.* **57**:410-412.
20. Yabuuchi, E., A. Ohya, H. Takeda, M. Sugiyama, and S. Kono. 1970. *Flavobacterium meningosepticum* from neonatal meningitis. *Jpn. J. Microbiol.* **14**:241-242.