Escherichia hermannii as the Sole Isolate from a Patient with Purulent Conjunctivitis

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Escherichia hermannii was isolated in pure culture from a patient with acute purulent conjunctivitis after a minor ocular injury. This is the first report of E. hermannii isolated as the sole pathogen from an infected site without prior antibiotic exposure, confirming the pathogenic potential of the microorganism.

CASE REPORT
A 38-year-old male presented with a painful red eye, burning in the eye, photophobia, purulent discharge, and eyelid edema. The symptoms had appeared 4 days before his admission and his condition had gradually deteriorated. The patient was an otherwise healthy individual without any history of systemic disease. Slit lamp examination revealed acute bilateral follicular-hemorrhagic conjunctivitis, pseudomembranes, purulent exudates, and diffuse stromal opalescence. Also, a very thin wood splinter was discovered and removed from the limbus of his right eye. The patient mentioned that the day before the onset of symptoms, he was cutting firewood, which was stored in his house’s shed.

 Conjunctival smears were collected from both eyes and cultured using thioglycolate broth, Columbia blood agar incubated aerobically and anaerobically, chocolate agar, and MacConkey agar. Two Sabouraud agar plates were also incubated at 25°C and 37°C. Microscopic examination of Gram-stained smears of the purulent secretions revealed many white cells and gran-negative rods. Based on the Gram stain results, antibiotic treatment was started with oral cefuroxime (500 mg twice a day) and eye drops of ciprofloxacin every 1 h for the first 6 h and then every 2 h. Following overnight incubation, the same gram-negative rod was recovered in large numbers from all culture media. The colonies of the microorganism were yellow, and their color was more apparent on blood agar. No other bacteria were detected.

 The isolate was found resistant to penicillin, amoxicillin, and ticarcillin and susceptible to amoxicillin-clavulanate, ampicillin-sulbactam, piperacillin-tazobactam, cefaclor, cefuroxime, cefotaxime, cefazidime, aztreonam, imipenem, ciprofloxacin, tobramycin, amikacin, tetracycline, and trimethoprim-sulfamethoxazole. The susceptibility pattern was in accordance with previous data, suggesting that E. hermannii isolates are generally sensitive to quinolones.

 Biochemical identification was confirmed by sequencing of the 16S rRNA gene. By use of universal primers, an amplicon of 1,401 bp (from position 8 to position 1408 of the 16S rRNA gene; E. coli numbering) was produced (12). Nucleotide sequencing of both strands of the PCR amplon was performed using an ABI Prism 377 DNA sequencer (Perkin-Elmer, Applied Biosystems Division, Foster City, CA). The sequenced product was 100% identical to the 16S rRNA E. hermannii GenBank entry (X80675) in a region of 1,347 determined base pair positions.

 Antimicrobial susceptibility testing of the microorganism was determined by the Etest method (AB Biodisk, Solna, Sweden) according to the CLSI MIC interpretative standards (2). The isolate was found resistant to penicillin, amoxicillin, and ticarcillin but susceptible to amoxicillin-clavulanate, ampicillin-sulbactam, piperacillin-tazobactam, cefaclor, cefuroxime, cefotaxime, cefazidime, aztreonam, imipenem, ciprofloxacin, tobramycin, amikacin, tetracycline, and trimethoprim-sulfamethoxazole. The susceptibility pattern was in accordance with previous data, suggesting that E. hermannii produces a chromosomal class A β-lactamase which confers resistance to aminopenicillins but not to β-lactam–inhibitor combinations or cephalosporins (14). Based on the susceptibility results, the patient’s treatment was not modified, and he completed a 10-day course of antibiotics after the initial consultation. Symptoms were alleviated within the first 48 h of treatment. The hemorrhagic and mucopurulent discharge was reduced by 50% in 3 days and completely resolved within 5 days of treatment.

E. hermannii is a distinct species within the genus Escherichia. It was initially considered as an E. coli-like biogroup and was classified as enteric group 11 by the Enteric Section, Centers for Disease Control (CDC). The name of this yellow-pigmented microorganism was proposed by Brenner et al. (1) based on the high DNA relatedness of strains within the species and the low relatedness (35 to 45%) with E. coli, the most clinically relevant species of the genus. Furthermore, biochemical tests, electrophoretic enzyme polymorphism (6), and ribo-
somal DNA restriction fragment length polymorphism patterns (9) supported this classification.

*E. hermannii* is isolated mainly from environmental sources, such as drinking water distribution systems (13). It is also known for its capacity to accumulate metals in industrial waste (7). In humans, the microorganism has been sporadically recovered from clinical specimens such as wound specimens, sputa, and stools (1, 10, 13). However, it was not the primary pathogen, as it was isolated from mixed infections together with large numbers of other pathogenic bacteria (10). *E. hermannii* has also been considered as an associated pathogen in a few invasive infections, which were mostly attributed to other coexisting bacteria that were more pathogenic (4, 5, 8, 11). Although *E. hermannii* was the sole pathogen recovered from a patient with cephalohematoma, the prior use of antibiotics was considered a confounding factor, because they may have inactivated other coexisting microorganisms (3). In accordance with these clinical data, animal studies have shown that *E. hermannii* is not capable of causing persistent wound infection and is not lethal following intraperitoneal injection (10). Therefore, the pathogenicity of the microorganism remains undetermined.

This report presents the first case where *E. hermannii* was the sole pathogen isolated from an infected site without previous administration of antimicrobial treatment. The microorganism was isolated in large numbers and caused an acute purulent eye infection. We believe that the unnoticed ocular injury by the wood splinter compromised corneal integrity and increased the susceptibility of the conjunctiva to *E. hermannii* infection in our patient. The fact that *E. hermannii* is frequently found in water and soil samples may explain how the wood, which was stored in a moist environment, was contaminated with the bacterium. To our knowledge, this is also the first report of ocular *E. hermannii* infection. A previous report had described *E. hermannii* isolation from an infant patient with conjunctivitis (10). However, in that case the infection was not attributed to *E. hermannii* but to *Staphylococcus aureus*, also recovered in large numbers. In conclusion, our report highlights the fact that *E. hermannii*, previously shown only to be an associated pathogen, may cause infection as a sole pathogen, particularly when there is a history of epithelial damage. Specialists should also be aware of *E. hermannii*’s propensity to infect the conjunctiva, given the fact that produced β-lactamase may inactivate commonly administered topical antimicrobials such as ampicillin, leading to treatment failure.

**REFERENCES**


