Escherichia hermannii as Sole Isolate from a Purulent Conjunctivitis

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Short title: Escherichia hermannii as Sole Pathogen

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*E. hermannii* was isolated in pure culture from a case of 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.

**Key words:** sole pathogen, acute conjunctivitis, isolation, case report, *E. hermannii*
**Case.** A 38-year-old male was presented with painful red eye, burning, photophobia, purulent discharge and eyelid edema. The symptoms had appeared four days before his admission and his condition had gradually deteriorated. The patient was an otherwise healthy individual without 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 McConkey 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 gram-negative rods. Based on the gram-stain results, antibiotic treatment was started with oral cefuroxime (500 mg bid) 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 from all culture media, in large numbers. The colonies of the microorganism were yellow and their color was more apparent on blood agar. No other bacteria were detected. *Chlamydia trachomatis* antigen was not detected using an enzyme linked fluorescent assay (ELFA, Vidas Chlamydia, BioMérieux, Marcy l’Étoile, France). The isolated microorganism was motile. It was identified as *Escherichia hermannii* using the MicroScan automated system (Dade Behring Inc., West Sacramento, CA) and the API 20E system (99.4% confidence; T-value=1.00; bioMérieux, Marcy l’Étoile, France). Specifically, the microorganism fermented glucose, arabinose, mannitol and rhamnose, and was positive for ornithine decarboxylase and indole production. It gave negative reactions for lactose, mellibiose and sorbitol fermentation, arginine dihydrolase, H₂S and urease production, lysine decarboxylase, Voges-Proskauer and citrate.
Biochemical identification was confirmed by sequencing of the 16S rRNA gene. Using universal primers an amplicon of 1,401 bp (from position 08 to position 1408 of the 16S rRNA gene, *E. coli* numbering) was produced (12). Nucleotide sequencing of both strands of the PCR amplicon was performed using an ABI Prism 377 DNA sequencer (Perkin-Elmer, Applied Biosystems Division, Foster City, CA). The sequenced product was 100% identical with 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 E-test method (AB Biodisk, Solna, Sweden) according to the CLSI MIC interpretative standards (2). The isolate was found resistant to penicillin, amoxicillin, ticarcillin and susceptible to amoxicillin/clavulanate, ampicillin/sulbactam, piperacillin/tazobactam, cefaclor, cefuroxime, cefotaxime, ceftazidime, aztreonam, imipenem, ciprofloxacin, tobramycin, amikacin, tetracycline and trimethoprim/sulphamethoxazole. The susceptibility pattern was in accordance with previous data, suggesting that *E. hermannii* produces a chromosomal class A \( \beta \)-lactamase, which confers resistance to aminopenicillins but not to \( \beta \)-lactam/inhibitor combinations and 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 three days and completely resolved within five days of treatment.

**Discussion.** *E. hermannii* is a distinct species within the genus of *Escherichia*. It was initially considered as an *Escherichia 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 high DNA relatedness of strains within the species and low relatedness (35-45%) with *E. coli*, the most clinically relevant species of the genus. Furthermore biochemical tests, electrophoretic enzyme
polymorphism (6) and ribosomal DNA restriction fragment length polymorphism patterns (9) supported this classification.

*E. hermannii* is isolated mainly from environmental sources, like drinking-water distribution systems (13). It is also known by its capacity to accumulate metals in industrial waste (7). In humans, the microorganism has been sporadically recovered from clinical specimens such as wounds, sputum 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, more pathogenic coexisting bacteria (4, 5, 8, 11). Although in a case of cephalomatoma *E. hermannii* was the sole pathogen recovered, the prior use of antibiotics was considered a confounding factor, because they may have inactivated other coexisting microorganisms (3). In accordance with this clinical data, animal studies have shown that *E. hermannii* is not capable of causing persistent wound infection and is not lethal following intaperitoneal 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 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 conjunctivitis in an infant (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 to an associated pathogen only, may cause infection as a sole pathogen,
particularly when there is a history of epithelial damage. Specialists should also be aware of *E. hermannii* 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


