Staphylococcus haemolyticus Urinary Tract Infection in a Male Patient

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Urinary tract infections caused by staphylococci are usually attributed to Staphylococcus epidermidis or S. saprophyticus. The case study reported here describes a persistent urinary tract infection caused by S. haemolyticus in a 38-year-old male whose infection was ultimately resolved through the use of the antibiotic trimethoprim-sulfamethoxazole.

Urinary tract infections (UTIs) are caused by a variety of bacterial species, but the most common is the gram-negative bacterium Escherichia coli (2, 14). Although coagulase-negative staphylococci are among the dominant organisms colonizing the urethra and periurethra in males and females, they typically account for fewer than 10% of all UTIs in the United States (2, 14). Most of these coagulase-negative staphylococcal UTIs are caused by the two species Staphylococcus epidermidis and S. saprophyticus (14). This report describes a case of acute cystitis that was caused by another species of coagulase-negative staphylococci, S. haemolyticus.

The 38-year-old male patient reported to our outpatient clinic with a several-week history of frequency, urgency, and dysuria upon urination. These symptoms progressively worsened during the week preceding medical consultation. There was no recent history of fever, chills, abdominal pains, or hemorrhia. Eight years earlier, the patient was diagnosed and treated for prostatitis and was vasectomized without complications. Four years earlier, the patient passed a urethral stone, but no further urinary tract problems ensued until the present infection was diagnosed.

A midstream urine specimen was collected during the patient’s first (day 1), second (day 6), and third (day 18) visits to the clinic. S. haemolyticus (>10^8 CFU/ml) was isolated by routine culture from each specimen. Antibiotic therapy was not prescribed during the first visit. The patient continued to be symptomatic and during the second visit was treated with erythromycin. The patient continued to be symptomatic until day 18. A third urine specimen was collected, and oral trimethoprim-sulfamethoxazole was prescribed on the basis of disk diffusion susceptibility data reported for the isolates cultured on day 1 (strain CNS-1a) and day 6 (strain CNS-1b), both of which were susceptible to trimethoprim-sulfamethoxazole and erythromycin. The patient responded to this treatment regimen and was asymptomatic when last seen on follow-up day 32 of his illness. The patient refused further urological evaluation.

Each urine specimen was inoculated to sheep blood agar by using a calibrated-loop (0.001-ml) technique, and the plates were incubated for 18 to 24 h at 35°C. All three strains proved to be coagulase-negative staphylococci on the basis of their susceptibility to lysostaphin, production of acid from glucose, and inability to produce coagulase (2, 8, 18). Each strain was identified to the species level by using the API Staph-Ident system (Analytab Products, Plainview, N.Y.), conventional methods, and criteria described elsewhere (12, 18).

Strain CNS-1a had an API Staph-Ident profile number of 0440. The two other strains, CNS-1b and CNS-1c, had an API profile of 0040. All three were identified by the API computer center as S. aureus. All three strains fit the description of S. haemolyticus by conventional characteristics and tests (12, 18). The strains weakly produced slime by the tube adherence test described elsewhere (3). Because of the improbability of culturing S. aureus, a colonizer of the human ear canal, from urine, each strain was sent to W. E. Kloos, North Carolina State University, Raleigh, for identification. He identified all three as S. haemolyticus.

Antimicrobial susceptibility testing was done by the Kirby-Bauer disk diffusion method (16). All three strains were susceptible to amikacin, cephalosporin, chloramphenicol, clindamycin, erythromycin, gentamicin, novobiocin, trimethoprim-sulfamethoxazole, tetracycline, and vancomycin but were resistant to ampicillin, sulfisoxazole, and penicillin. Mixed results were obtained with methicillin: strains CNS-1a and CNS-1b were resistant and CNS-1c was susceptible to the antibiotic.

UTIs caused by coagulase-negative staphylococci are rare among healthy males in an outpatient setting (13, 14). Based upon the rarity of UTIs caused by coagulase-negative staphylococci and upon reports attributing most of these infections to S. epidermidis sensu stricto or S. saprophyticus (1, 6, 14), the finding of S. haemolyticus as a causative agent of acute cystitis in our male patient was unexpected. This species causes fewer than 10% of the nosocomial and community-acquired infections that are attributed to coagulase-negative staphylococci (6, 7, 10). It has been implicated as the primary etiologic agent in diseases such as cystitis (6), abscesses (6), continuous ambulatory dialysis peritonitis (7), prosthetic valve endocarditis (10), and septicemia (6).

S. haemolyticus grows in moderate numbers on human glabrous skin (10, 11) and is one of only a few coagulase-negative staphylococcal species that colonizes the urethra or periurethra of males and females. In fact, colonization numbers of this and other coagulase-negative staphylococcal
species often exceed those of gram-negative bacilli, which cause 80% of all UTIs (2, 13). Strains of *S. haemolyticus* produce a hemolysin (5), cytolysin (5), and enterotoxin (17) and are often multiply antibiotic resistant (6, 7, 13). Although these characters suggest a potential pathogenic role for this organism, its ability to initiate infection in an infant mouse model for testing the virulence of coagulase-negative staphyloccocal species is reportedly poor compared with the infective abilities of *S. epidermidis* (15). However, larger clinical and laboratory studies are needed to fully assess the virulence for humans and laboratory animals, since preliminary (unpublished) studies in our laboratory suggest that fresh clinical isolates of *S. haemolyticus* are more virulent for infant mice than was reported previously (15).

Recurrent infections follow as many as 10% of all UTIs caused by coagulase-negative staphylococci, especially among patients with indwelling catheters and prosthetic devices (7, 10). To our knowledge, our patient was not afflicted by any underlying clinical disease and was healthy. Persistence of the organism during the 12 days of therapy with erythromycin can be attributed partially to poor in vivo response of the organism to this bacteriostatic drug even though all three strains were susceptible in vitro to the antibiotic. Resolution of the illness immediately followed the change in therapy to trimethoprim-sulfamethoxazole.

The results of this study and those of others suggest that an epidemiological advantage may often be gained by identifying coagulase-negative staphylococci when they are cultured in large numbers from some types of clinical specimens (1, 4, 6, 7, 9, 10). This is especially applicable to specimens obtained from problematic patients suspected of having endocarditis and to specimens from patients who are being evaluated for possible nosocomial infections (7, 10, 13). The significance of coagulase-negative staphylococci that are cultured from these types of patient specimens is typically recognized with some difficulty because of their frequent colonization of human skin, a site from which they are inadvertently selected as contaminants during collection of transcutaneous specimens (11). Thus, the laboratory must occasionally distinguish among isolates which are obtained at different times or from different sites in a patient, among strains which may represent fresh or treatment failure infections, and among isolates which may represent contaminants or infective strains (1, 9, 10). Diagnostic criteria are available for distinguishing such strains and include biotyping techniques, such as antibiograms, biochemical profiles, and growth characteristics (6–8, 12, 18).

With our patient, multiple isolation of *S. haemolyticus*, coupled with a high colony count, suggested that the initial infection was not resolving with erythromycin therapy and that a change in therapy was in order. In the absence of a high colony count, a coagulase-negative staphylococcus isolated from a urine specimen of a similar patient might have been dismissed as a contaminant. In this case, other etiologic agents of urethritis in males, e.g. chlamydiae, might have been suspected. This scenario, in which the significance of coagulase-negative staphylococci cultured from urine is underestimated, may occur more frequently than is generally believed, since studies have shown that many staphyloccocal UTIs are accompanied by colony counts far lower than 10⁵ CFU/ml (14). Thus, in a clinical case of symptomatic UTI with low accompanying urinary colony counts of coagulase-negative staphylococci, biotyping a patient’s coagulase-negative staphylococcal isolates could aid the physician in determining the significance of the organism and could guide the selection of the appropriate course of chemotherapy for the patient. However, the increased workload and cost that result from such laboratory work preclude such biotyping on a routine basis. The need to do it rests on clinical evidence that implies that such studies are useful in treating the patient.

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**LITERATURE CITED**


