staff practices much more defensive medicine, American style, than do others, and that the staff possibly obtains many samples for unjustified blood cultures.

Our patient case mix is definitely different from that of Módol et al. [2]. Our hospital admits substantial numbers of geriatric patients and nursing home residents, many of whom harbor chronic, debilitating illnesses and associated infections. On the other hand, our emergency department cares for very few patients who abuse injection drugs or who have alcoholism, cirrhosis, or AIDS, which are uncommon in our country. In this respect, the conclusion of Módol and colleagues—namely, that occult bacteremia correlates with specific underlying conditions that are prone to develop bacteremia—is important. This information should lead to heightened alertness to possible cases of bacteremia among clinicians treating patients with those risk factors.

We would like to accentuate what is common rather than what differs between these studies. No death or serious complication was reported for any of the patients with occult bacteremia who were discharged from the emergency department. These observations indicate that, although careful clinical evaluation in the emergency department may not prevent discharge of an occasional patient with occult bacteremia, these patients can be safely recalled and retreated without undue complications. The data of Módol et al. [2] provide further support for the practice of obtaining blood samples for culture in the emergency department when clinically indicated, although some of these patients can be subsequently discharged if they are deemed clinically stable. This is reassuring from both clinical and medicolegal viewpoints.

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Colistin: An Antimicrobial for the 21st Century?

Sir—The emergence of multidrug-resistant, gram-negative bacteria that cause nosocomial infection is a growing problem worldwide. Strains of Pseudomonas aeruginosa that are resistant to all commercially available drugs have been reported to cause infection in intensive care units [1]. Our department has a special interest in the treatment of infections associated with orthopedic devices. Recently, we have seen the emergence of resistance to each class of antibiotics used against these infections [2]. In our hospital, the first strain of P. aeruginosa that was resistant to all commercially available antibiotics (except colistin) was isolated in 1999. In the past 6 months, an outbreak occurred in which >30 such strains were isolated from clinically significant sites. We evaluated the use of colistin for the treatment of 3 patients who had infections associated with their orthopedic devices. One patient had suppurative tibial pseudarthrosis after an open fracture was stabilized with an osteosynthetic plate, and 2 patients had chronic infections associated with their hip prostheses. Isolates of P. aeruginosa that were resistant to all antibiotics except colistin were recovered from all 3 patients. Orthopedic implants could not be removed, and the patients were treated with colistin (1 million U iv t.i.d.) for 3–6 months; all were definitely cured.

Colistin was first introduced in 1952 and was used until the early 1980s for treatment of infections caused by gram-negative bacilli [3]. Colistin was no longer preferred when second- and third-generation cephalosporins became available, mainly because of colistin’s toxicity [1, 4]. Presently completely abandoned, colistin is no longer recommended in reference manuals of clinical microbiology and infectious diseases for treatment of deep infections. Nevertheless, in vitro, colistin has had excellent activity against a variety of gram-negative rods, including those that are resistant to other classes of antimicrobials (e.g., penicillins, cephalosporins, quinolones, aminoglycosides, and carbapenems) [4]. The precise mechanism of action of colistin is not known, but it is thought to involve disruption of the bacterial cell membrane by binding of the drug to phospholipids [5]. Nephrotoxicity is the most important adverse effect, which occurs more frequently in patients with preexisting impairment of renal function. Doses must be adjusted in patients with renal insufficiency, because colistin is excreted principally by the kidneys, and elevated levels of the drug in blood may further impair renal function [1]. Despite the extended duration of therapy, no adverse effects that required discontinuation of therapy were noted in any of our patients.

The emergence of multidrug-resistant bacteria that cause nosocomial infection poses a serious therapeutic problem. Because no fundamentally new anti-infective drugs are currently available, it appears that we need to reevaluate the “old” colistin in additional, larger clinical and pharmacokinetic studies, to determine whether it might be born again as an antimicrobial option in the 21st century.

CORRESPONDENCE • CID 2002;35 (1 October) • 901
Stevens-Johnson Syndrome Associated with Abacavir Therapy

Str—Adverse cutaneous reactions to drugs are commonly observed in HIV-infected patients [1]. We describe what is, to our knowledge, the first case of Stevens-Johnson syndrome in an HIV-1-infected patient that is associated with use of abacavir, a nucleoside reverse-transcriptase inhibitor.

A 37-year-old man was admitted to the hospital in October 2001 because of neurologic disorders and dyspnea. Diagnosis of toxoplasmic encephalitis and Pneumocystis carinii pneumonia was made. At this time, the patient had HIV-1 infection diagnosed. The plasma HIV-1 RNA level was 118,000 copies/mL, and the CD4 cell count was 1 cell/mm³. Treatment with pyrimethamine-sulfadiazine was started. Ten days after he began receiving therapy, the patient presented with a febrile maculopapular rash; sulfadiazine was switched to clindamycin (for treatment of toxoplasmic encephalitis) and atovaquone (for treatment of P. carinii pneumonia), and pyrimethamine therapy was continued. Cutaneous manifestations resolved completely within 2 days. The patient was discharged from the hospital 15 days after the switch without neurological or pulmonary manifestations.

In December 2001, the patient began receiving antiretroviral treatment with zidovudine (300 mg b.i.d.), lamivudine (150 mg b.i.d.), and abacavir (300 mg b.i.d.). Thirteen days after he began receiving antiretroviral therapy, the patient presented with a nonfebrile, generalized maculopapular rash. At admission to the hospital, his temperature was 37°C, his pulse was 86 beats/min, and his respiration rate was 16 breaths/min. The patient’s blood pressure was 130/60 mm Hg. Physical examination revealed a disseminated cutaneous eruption of discrete dark-red macules on 90% of the body surface area, a detachment of 5% of the epidermis, genital ulcerations, erosive stomatitis, and conjunctival lesions with hyperemia, and a pseudomembranous formation occurred without keratitis or corneal erosions. Nikolsky’s sign was noted.

Hematologic test findings, blood chemistry findings, enzyme values, and chest radiograph findings were normal. No infectious agent was found. Histopathologic evaluation of skin biopsy specimens yielded findings compatible with Stevens-Johnson syndrome. The results of immunofluorescence studies were negative. Antiretroviral treatment was stopped, and therapy with pyrimethamine, clindamycin, and atovaquone was continued. A week later, the epidermis began to regrow, and the condition resolved completely within 3 weeks. The patient was rechallenged with zidovudine and lamivudine and commenced therapy with ritonavir and indinavir without recurrence of clinical manifestations.

These findings clearly suggest that there is a link between abacavir use and Stevens-Johnson syndrome. In patients with HIV infection, cases of Stevens-Johnson syndrome have been reported in association with other antiretroviral agents, such as nevirapine [2]. Until now, to our knowledge, only hypersensitivity reactions to abacavir have been reported [3]. Abacavir should now be added to the list of antiretroviral agents associated with Stevens-Johnson syndrome.

References

Successful Treatment of Enterococcus faecalis Prosthetic Valve Endocarditis with Linezolid

Str—Linezolid possesses a broad spectrum of activity against gram-positive organisms, including Enterococcus faecalis [1]. Like other agents used for the treatment of E. faecalis endocarditis, linezolid possesses only bacteriostatic activity [2].

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