Letter to the editor

ANTIBIOTIC-RESISTANT GRAM-NEGATIVE BACTERIA IN DEEP TISSUE CULTURES

Past studies have shown that antibiotic-resistant Gram-negative organisms (AR GNO) colonise the surface of chronic wounds (1–4). We performed a chart review to determine the prevalence of AR GNO in deep tissue of non healing and infected wounds after sharp debridement had been performed.

We reviewed records of bacterial wound cultures for all patients seen over a 6-month period at an urban wound healing programme. For each patient, all cultures that had been obtained at our institution were reviewed. Cultures were included in analysis if they were derived from deep tissue obtained after sharp excisional debridement of all grossly necrotic and non viable tissue. Clinical indications for wound debridement included infection or failure to heal in a non ischaemic wound.

Antibiotic resistance was defined as resistance to carbapenems in acinetobacter strains (5), resistance to third-generation cephalosporins in klebsiella strains and resistance to fluoroquinolones, third-generation cephalosporins, or carbapenems in pseudomonas strains. (6) Intermediate sensitivity was considered to be resistant. Diphtheroids and coagulase-negative staphylococcus were not included in assessment of polymicrobial infection.

Thirty of 336 total patients (8·9%, 95% CI: 8·7–9·2%) had deep tissue cultures with antibiotic-resistant acinetobacter, pseudomonas and/or klebsiella. Four patients had more than one of these organisms. There were 7 patients with resistant acinetobacter, 8 with klebsiella and 19 with pseudomonas. Of the 19 patients with antibiotic-resistant pseudomonas, 12 had resistance to fluoroquinolones, 5 to carbapenems and 15 to third-generation cephalosporins. Eleven patients had resistance to more than one of these classes, and two patients had resistance to all three classes. Of the patients with carbapenem-resistant acinetobacter, all isolates were resistant to cephalosporins and fluoroquinolones. No resistance to polymixin was observed.

Of the 30 patients, 12 had venous stasis ulcers; 7 ischaemic wounds; 5 pressure ulcers; 5 diabetic foot ulcers and 1 pyoderma. Twenty-five cultures were obtained in the operating room and five in the outpatient or bedside setting.

Of 10 patients with resistant Gram-negative organisms in bone cultures, 9 had pathology specimens, of which 5 showed histopathology consistent with osteomyelitis. Twenty-two cultures (65%) were polymicrobial infections. Of the 30 patients with positive cultures, 50% were 65 years or older, 30% lived in a nursing home and 23% were bedbound. There was a high rate of chronic disease: 63% of patients were diabetic and 9% were immunosuppressed from human immunodeficiency virus, post-transplant medications or chemotherapy. Patients had been hospitalised on the wound programme's inpatient unit an average of 3·5 times over the previous 3 years.

This study documents the presence of AR GNO in deep tissue after wound debridement. Antibiotic regimens for resistant bacteria often require intravenous administration and have higher rates of adverse effects. We hypothesise that wound debridement decreases the risk of developing resistant organisms by surgically removing the infected tissue. We further hypothesise that, in the presence of resistant organisms, tailored antibiotic regimens lead to faster resolution of infection and increase healing rates.

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