Skin Lesions Associated with E. Coli Sepsis in a Patient with Acute Leukemia

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The clinical course of a patient with acute leukemia and metastatic intradermal abscesses secondary to Escherichia coli sepsis is described. E. coli organisms, cultured first from the blood and later from the lesions, had an identical antibiotic susceptibility and biotyping profile. This complication has not been previously described. Granulocytopenia and varicose veins may have been critical predisposing factors. With more widespread use of progressive immunsuppressive regimens, unusual manifestations of common infections will be recognized more frequently.

Skin lesions are known to be associated with gonococemia, pseudomonas sepsis, and disseminated candidiasis (1). They are often the only clue of systemic infection. In this report, we describe a patient with acute granulocytic leukemia who developed intradermal abscesses as a complication of Escherichia coli sepsis. Except for a description by Fisher, et al (2) of infected subepidermal bullae, a distinctly different lesion from the one we describe, cutaneous lesions associated with E. coli sepsis have not been reported.

Case Report

A 43-year-old white woman was admitted to Henry Ford Hospital with chills and fever (41°C). Previously, she had been treated for acute granulocytic leukemia, now in relapse following a one-year remission induced with adriamycin, vincristine, cytosine arabinoside, and prednisone. With each reinduction of chemotherapy treatment over the preceding three months, she had developed episodes of urinary tract infection with or without bacteremia, due to E. coli. One week before admission, she received ifosfamide (a cyclophosphamide analog) and adriamycin in an effort to re-induce remission.

On admission she was hypotensive and toxic. Her white blood count was 300 m/l with 38% segmented and band neutrophils and 65% lymphocytes. Platelet count was 15,000/ml (3). She was immediately started on intravenous cephalothin, 2.0 gm every 6 hours, and gentamicin, 90 mg every 8 hours, after cultures had been obtained. Blood and urine cultures subsequently grew E. coli with identical sensitivity and biochemical profiles by the analytab method. Thirty-six hours after she was admitted, the patient developed simultaneously two painful, indurated, subcutaneous 2 by 2 cm nodules, one on the left lateral thigh and the other on the right calf. Within 24 hours these lesions enlarged to involve the entire dermis and measured 7 by 7 and 10 by 10 cm, respectively, with a central 2 by 2 cm area of intense erythema in each lesion (Fig. 1).

By the next day, her blood pressure stabilized, but she continued to spike temperatures up to 40°C. The induration around the leg lesions remained constant. Meanwhile, the erythema spread, became purpuric, and developed bullous centers with clear fluid containing E. coli but no granulocytes. These organisms had sensitivities and biochemical profiles identical to those previously isolated from blood and urine. The patient received granulocyte transfusions for three consecutive days, after which the bullous fluid became cloudy, containing neutrophils. Subsequent blood cultures were negative, but the skin lesions continued to grow E. coli for the next five days. On the tenth day, ampicillin was substituted for cephalothin and gentamicin. At this time, the lesion on the left leg had begun draining thin, serosanguinous fluid, and the lesion on her right leg developed a central eschar. The patient's white count rose to 3,000 m/l. Both lesions underwent slow resolution with draining fistulae formation during the third hospital week. She was discharged on the 22nd day, the drainage of sterile necrotic debris having significantly decreased.
Appearance of metastatic cutaneous lesions 72 hours after sepsis began in (A) right popliteal fossa and (B) left lateral thigh. Note surgical scars from previous vein stripplings on each leg.

Discussion
The development of cellulitis during the course of systemic E. coli infection is an unusual complication which has not been described in two recent reviews (3,4). However, there are factors which may have been responsible for this complication in this particular patient. Teplitz suggests that, at least in Pseudomonas infection, tissue invasion occurs when local tissue neutropenia is present (5). Our patient had fewer than 300 granulocytes per ml and finally required granulocyte transfusions to treat her persistent infection. Aspirates of the lesions contained no granulocytes until after her transfusions. Also, she had varicose veins in her legs, and vein stripping procedures had been performed several years previously (Fig. 1). Her venous disease may have provided the site for bacterial tissue invasion. The loss of endothelial integrity probably secondary to thrombocytopenia also predisposes to bacterial dissemination in this clinical setting; but, at present, we can only speculate about a firm cause and effect relationship between thrombocytopenia in neutropenic patients and tissue invasion by microorganisms. The unusual manifestations of common Gram-negative infections, such as skin lesions, should be recognized and treated promptly in these patients.

References