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Escherichia vulneris as a cause of bacteremia in a patient with chronic lymphocytic leukemia

Escherichia vulneris is a recently identified environmental organism that can colonize humans and animals. It has mainly been recovered from human wounds.[1] Very few infections with *E. vulneris* have been reported. Pien et al.[2] described a series of 12 Hawaiian patients who had *E. vulneris* isolated from soft tissues. It now seems clear that these organisms can cause invasive infections in immunocompetent and immunodepressed patients, as is shown in the following case.

A 70-year-old man, treated two years previously for chronic lymphocytic leukemia and followed in the Department of Hematology, was admitted to the hospital because of rigors, fever and left gonalgia. He was allergic to beta-lactams and received no medications as prophylaxis. On physical examination, his temperature was 39 °C, and there was a tenderness of the knee, where a subcutaneous mass was detected with fluctuation. He had a blood pressure of 100/80 mmHg, and the remainder of the examination was unremarkable. Laboratory examination revealed hemoglobin of 12.7 g/l, a white blood cell count of 7.8×10^9/l, and a platelet count of 335×10^9/l. Routine blood chemistry and coagulation tests were normal, except for a C-reactive protein level of 191 mg/l. Blood cultures yielded Gram-negative bacilli, identified as *E. vulneris* by Vitek 1 (bioMerieux). Susceptibility testing was performed according to the NCCLS disk diffusion method. The same organism was isolated in the pus of a subcutaneous abscess after drainage. The organism was susceptible to ampicillin, cephalosporins, aminoglycosides (gentamicin, amikacin and streptomycin), quinolones (ciprofloxacin, ofloxacin and levofloxacin) and cotrimoxazole. Surgical debridement was not recommended. The patient was treated with a combination of ciprofloxacin and gentamicin for one week, and then ciprofloxacin for seven days. He was discharged on day 15 of treatment, with almost complete resolution of the soft tissue infection; he continued the ciprofloxacin treatment for a further 15 days. At follow-up 2 weeks after completion of treatment, he was well without any symptoms.

Since 1982, *E. vulneris* has been classified in the family of *Enterobacteriaceae* in Enteric Group 1, on the basis of DNA relatedness studies and biochemical reactions. It has been isolated from animals, the environment, potable water and humans,[1,3] where it can colonize many sites, especially wounds.[2] *Escherichia vulneris* has been identified in many invasive infections, including osteomyelitis,[4] bacteremia,[5] urosepsis,[6] septic shock[7] and meningitis.[8] More recently, a peritonitis in the setting of peritoneal dialysis has been reported[9] in an elderly female patient. Our patient presented with a colonization of his wound causing a local sepsis, and because of immunosuppression due to a lymphoproliferative disorder, bacteremia occurred with the same agent. It is unclear whether hematologic malignancies, such as myeloproliferative or lymphoproliferative disorders, play a role in the pathogenesis of such infections.

In wounds with *E. vulneris* infection, co-infection with other bacteria has been reported, and this may contribute to the extensive tissue injury seen in such cases.[7] Clinical isolates of *E. vulneris* are slightly more susceptible to the commonly used antibiotics than *Escherichia coli* or *Escherichia hermannii*, especially aminoglycosides and ticarcillin.[10] A reduction in the susceptibility of some strains may suggest a nosocomial source of the disease.[8]

In conclusion, to our knowledge, this is the first reported case of *E. vulneris* bacteremia in a patient with chronic lymphocytic leukemia in Tunisia, demonstrating that the organism is a true pathogen.

Conflict of interest: No conflict of interest to declare.

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Gastric and cutaneous dissemination of visceral leishmaniasis in a patient with advanced HIV

Visceral leishmaniasis (VL) is a well-recognized opportunistic infection in patients with HIV-1 infection. Several reports indicate a rising trend in VL/HIV co-infection. The majority of HIV/*Leishmania* co-infected cases show classic features of VL. Atypical features such as dissemination to the gastrointestinal tract and to the skin may also occur.

We describe herein a patient with advanced HIV-1 infection residing in a non-endemic area for leishmaniasis. The diagnosis of HIV had been made 4 years prior to this admission. Unfortunately, the Centers for Disease Control and Prevention (CDC) stage at the time of diagnosis was not known. He presented with atypical features of visceral leishmaniasis. In particular, the patient had dissemination to the esophagus, the stomach, and to the skin.

A 50-year-old Omani male presented with progressive dysphagia, diarrhea with vague abdominal pain, severe anorexia, and weight loss of 20 kg over 6 months. A history of intermittent episodes of high-grade fever, chills, night sweats, weakness, and occasional headaches was elicited. The patient had also noticed the appearance of multiple pigmented plaques over his face and neck during the same period. He had lost the vision in his left eye 3 months earlier. The patient denied any history of travel outside Oman.

Examination showed a sick looking patient with severe wasting and marked pallor. He was febrile and had neck stiffness with intact sensorium and no focal deficit. Skin examination revealed multiple pigmented plaques of 3 × 1 cm over the right mandible and left base of the neck (Figure 1). Similar lesions were seen over the hard palate. Multiple enlarged cervical lymph nodes were noted with no hepatosplenomegaly. The patient had no light perception in the left eye; visual acuity in the right eye was limited to finger counting. Fundoscopy showed changes consistent with cytomegalovirus (CMV) retinitis in both eyes.

Laboratory findings were as follows: full blood count revealed anemia (Hb 9.6 g/dl), leukopenia (white blood cell count: 2.97 $\times 10^9$/l), and a normal platelet count (276 $\times 10^9$/l). ELISA and Western blot for HIV-1 was positive. The HIV-1 viral load was 480 000 RNA copies/ml, with an absolute CD4 count of 3.0/μl, CD4/CD8 ratio of 0.005:1.

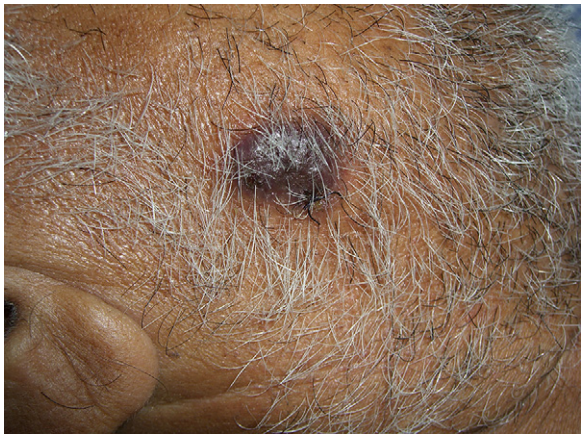

Figure 1 Dissemination of visceral leishmaniasis to the skin.