Chromobacterium violaceum Infection of the Deep Neck Tissues in a Traveler to Thailand

Chromobacterium violaceum is frequently found in soil and water in tropical and subtropical regions, but rarely in temperate regions [1]. Infection often occurs after exposure of damaged skin to stagnant water or soil [2]. Progression to systemic infection is often rapid, with the development of multiple metastatic abscesses that involve the liver, spleen, lung, or skin [2–5]. We report, to our knowledge, the first case of C. violaceum infection in the deep tissues of the neck.

A previously healthy 27-year-old male presented after he developed fever, pharyngitis, and odynophagia while traveling in Thailand. One week earlier he had cut his left leg on coral. A pustule developed adjacent to the wound 4 days before the onset of his illness. He was admitted to the hospital and received a 6-day course of antibiotics. His condition did not improve, and he underwent bilateral tonsillectomy for presumed quinsy. He was discharged with instructions to take ofloxacin (100 mg b.i.d.) and amoxicillin/clavulanic acid (500 mg/125 mg t.i.d.), and he returned to New Zealand.

When he was examined 1 day later, bilateral tonsillar exudate was present, but he was well, and the antibiotic therapy was stopped. Eight days later he was readmitted to the hospital with fevers and increasing left-sided neck pain. His temperature was 37.5°C. He had bilateral tonsillar exudates. There was local tenderness over the bony origin of the sternomastoid. Findings on physical examination were otherwise normal.

Laboratory investigations showed a hemoglobin level of 94 g/L, a WBC count of 9.9 × 10^9/L, and cholestatic derangement of his liver enzymes; serology for HIV was negative. Blood cultures were sterile.

A diagnosis of retropharyngeal infection was made, and treatment with iv amoxicillin/clavulanic acid (1.0/0.2 g t.i.d.) was started. A neck CT scan did not show any fluid collection. The left internal jugular vein was nonenhancing and was presumed to be occluded. The patient remained unwell, with temperature spikes to 40°C. He developed pleuritic chest pain. A repeated CT scan on hospital day 5 showed a left prevertebral fluid collection and an opaque mastoid. Emergent left-jugular-vein ligation and excision, mastoidectomy, thrombectomy from the sigmoid sinus and jugular bulb, and drainage of the prevertebral abscess were performed.

Within 36 hours, C. violaceum was isolated from mastoid tissue and tracheal aspirates. The young colonies were smooth, convex, and 1 mm in diameter. The older colonies were β-hemolytic. The isolate did not decarboxylate either lysine or ornithine but hydrolyzed arginine. The Baxter Microscan identification system (Baxter Laboratories, West Sacramento, CA) gave a biotype number of 20355370405–150, which identified the isolate as C. violaceum; in addition, it was noted that the isolate’s purple pigment production did not diffuse into clear nutrient agar plates. Disk susceptibility testing showed that the isolate was resistant to amoxicillin, cefuroxime, cefoxitin, ceftazidime, piperacillin, and ticarcillin but susceptible to chloramphenicol, gentamicin, amikacin, cotrimoxazole, tetracycline, imipenem, and ciprofloxacin. The MICs of gentamicin, amikacin, and ciprofloxacin were 2.0 mg/L, 16 mg/L, and <0.03 mg/L, respectively.

After the isolate was identified, iv therapy with amikacin (500 mg q8h) and chloramphenicol (1.2 g q8h) was begun. The patient initially remained grievously ill; there was radiographic evidence of septic emboli to his liver, and metastatic lesions were noted on his skin. However, by day 13, 6 days after the operation, his condition had clearly improved. The antibiotic regimen was changed to iv ciprofloxacin (200 mg q12h). Treatment with oral ciprofloxacin (750 mg b.i.d.) was started on day 17. He was discharged after 20 days. He completed 3 months of treatment and remains well 12 months later.

Systemic infection with C. violaceum is rare, and we did not consider this organism in the differential diagnosis for our patient. This is the first reported case of C. violaceum infection of the deep tissue of the neck. Our management of this case was complicated by the unusual spread of the infection; while quinolyl can occasionally spread to the adjacent parapharyngeal space, spread to the prevertebral space is extremely rare. The focus of this infection was in the prevertebral space and adjacent skull base, with secondary effects on the mastoid and sigmoid-jugular system. Deep neck infections are associated with significant morbidty and mortality [6, 7], and the development of jugular thrombophlebitis with septic pulmonary emboli is a well-known complication [6]. Presumably, hematogenous seeding to the deep-neck-tissue spaces occurred in our patient some time after the infection developed on his leg.

Mortality rates of 60% have been described for patients with C. violaceum infection [5]. The optimal antibiotic regimen (the drug, mode of administration, or duration of administration) is not known. Aminoglycosides [2–4, 8] were used in four of the five survivors of C. violaceum infection for whom the antibiotic regimens were described. However, the MICs of gentamicin reported for three isolates [5, 8] and herein ranged from 1 mg/L to 5 mg/L. Clearly, C. violaceum is not extremely susceptible to gentamicin; our isolate was similarly not extremely susceptible to amikacin. These laboratory data cast doubts on the routine recommendation of aminoglycosides for this infection. Recently, because of relapses [2, 3], oral therapy has been given for 2 to 3 months after initial treatment with iv ciprofloxacin.

A quinolone and ceftazidime [9], with or without an aminoglycoside, could be initial therapeutic choices for patients from tropical regions who present with fulminating sepsis; C. violaceum, Burkholderia pseudomallei, and the usual bacterial pathogens would be covered by such a regimen.

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References
Clinical and Pathophysiological Aspects of Immune Complex Glomerulonephritis Associated with Entamoeba histolytica Abscess of the Liver

Various bacterial or parasitic infections are associated with glomerulonephritis. Detection of microbial antigens within glomerular lesions during the course of infection-associated glomerulonephritis suggests a direct link between the infectious process and glomerular damage. We describe a patient who presented with an amebic liver abscess in association with proliferative glomerulonephritis; “humplike” deposits were present in some glomeruli.

Fifteen days after a trip to Yemen, a 62-year-old woman presented to our hospital with fever and a pain in the left hypochondrium. Physical examination showed an enlarged liver. Laboratory values were as follows: erythrocyte sedimentation rate, 118 mm after 1 hour; hemoglobin level, 8.9 g/dL; WBC count, 10,660/ mm³; serum creatinine level, 1.1 mg/dL; serum urea level, 124 mg/dL; and levels of alkaline phosphatase and transaminases, twice the normal values. An abdominal ultrasonogram showed an intrahepatic image compatible with an 11-cm abscess. An EIA with Entamoeba histolytica antigen and hemagglutination was strongly positive (titers, 1,350 and 1,024, respectively), confirming the diagnosis of amebic liver abscess. Treatment with intravenous metronidazole (500 mg three times daily) and oral tilbroquinol (1 g/d) for 10 days was initiated, and within 4 days, the fever and abdominal pain had attenuated. The abscess was punctured on day 8 to prevent its spontaneous rupture. This procedure provided complete relief of the patient’s symptoms.

The patient developed mild, generalized edema 3 days after treatment was started. Laboratory studies showed the following values: albumin concentration 23 g/L; serum creatinine level, 1 mg/dL; serum urea level, 82 mg/dL; and proteinuria, 5.8 g/d without leukocyturia or hematuria. Type 2 cryoglobulinemia was detected (monoclonal IgG-κ level, 0.23 g/L). No circulating immune complexes were detected. The levels of total hemolytic complement, C3, C4, and B were normal. Findings on an ultrasonogram of the kidneys and urinary tract were normal. A renal biopsy was performed, and examination of the glomeruli showed mesangial hypercellularity and presence of polymorphonuclear cells. Some glomeruli showed endomembranous and extramembranous “hump-like” deposits. The interstitium and vessels were normal.

Direct immunofluorescence revealed granular deposits of IgG and C3 along the glomerular basement membranes and in the mesangium. Electron microscopic investigation showed mesangial proliferation and mesangial, endomembranous, and extramembranous humplike deposits (figure 1). Attempts to detect the 170-kD immunodominant protein of E. histolytica with use of monoclonal antibodies yielded negative results. As these histopathological data were consistent with postinfectious glomerulonephritis, no treatment was prescribed. The renal abnormalities spontaneously disappeared within 2 months, the albumin concentration returned to normal, and the level of proteinuria decreased to 350 mg/d. At the last evaluation, the patient was free of renal symptoms.

In the present case, which resembles the case reported by Westendorp et al. [1], three findings suggested a causal link between the patient’s visceral amebiasis and glomerulonephritis: the onset of glomerulonephritis during the course of the infection, the concomitant mixed cryoglobulinemia [2], and the presence of extramembranous humplike immune complex deposits. Microbial agents can be found in glomerular immune complex deposits in patients with infection-associated glomerulopathies [3]. Immune complex deposition, which leads to the production of the C5b9 component (the so-called membrane attack complex [MAC]), results in glomerular basement membrane injury [4]. The inactivation of CD59, an inhibitor of C5b9, has been shown to worsen glomerular damage [5].

Figure 1. Transmission electromicroscopy shows extramembranous “hump-like” deposits (arrows) along the glomerular basement membrane in a patient with a liver abscess due to Entamoeba histolytica (original magnification, ×16,000).