Facial Folliculitis Due to Clostridium perfringens in a Patient Infected with Human Immunodeficiency Virus

The most frequent bacterial skin diseases associated with HIV infection include common pyogenic infections due to Staphylococcus aureus and Streptococcus pyogenes as well as syphilis, mycobacterial infections, and bacillary angiomatosis [1]. However, as patients with HIV infection live longer with more prolonged periods of immunosuppression, the spectrum of organisms leading to infection and the spectrum of diseases caused by these organisms continue to expand. This circumstance is illustrated by the following case report in which we describe a patient with folliculitis due to Clostridium perfringens.

A 60-year-old woman with AIDS was hospitalized in February 1996 because of fever, diarrhea, abdominal pain, asthenia, and weight loss. Her HIV status had been known since 1988 (she had received a blood transfusion in 1978), and AIDS was diagnosed in May 1995, secondary to Pneumocystis carinii pneumonia. In addition, she had a history of rosacea and hepatitis C infection, without evidence of chronic hepatitis. Since May 1995, she had been treated with zidovudine, lamivudine, and trimethoprim-sulfamethoxazole (TMP-SMZ) (one double-strength tablet daily).

Three days after she was hospitalized, cytomegalovirus retinitis was diagnosed. Her CD4+ cell count was 13 × 10^6/L and treatment with iv foscarnet (6 g b.i.d.) was initiated. Six days later, because of the onset of oral candidiasis and dysphagia, treatment with oral fluconazole (100 mg/d) was instituted.

Ten days later, a pustular eruption appeared on her face. Non-specific superficial folliculitis was suspected, and the area was treated with local antiseptics. However, despite this treatment, the pustular eruption progressed to involve the entire face. Three days after the onset of the eruption, cutaneous examination revealed a predominant mediofacial follicular and nonfollicular pustular eruption (figure 1). These lesions coalesced to form a honey-colored surface crust on the chin. With the exception of one pustular lesion on the left shoulder, there were no other cutaneous lesions. The clinical examination revealed only a recurrence of fever (temperature, 39°C). A WBC count revealed an increase to 4 × 10^9/L, from a level of 1.7 × 10^9/L 3 days earlier. Blood cultures were negative, as was a urinalysis, and findings on a chest radiograph were within normal limits.

Figure 1. Folliculitis due to Clostridium perfringens appearing as a facial pustular eruption with honey-colored crust on the chin in a woman with AIDS.
replaced with metronidazole, secondary to the onset of an adverse drug reaction in the form of a cutaneous eruption. Results of a colonoscopy were normal. The duration of therapy with antimicrobial agents was 21 days, and the patient’s condition resolved.

To our knowledge, ours is the first case of folliculitis due to \textit{C. perfringens}, an anaerobic, gram-positive, spore-forming bacillus that is usually responsible for three distinct soft-tissue infections: crepitant cellulitis, suppurative myositis, and gas gangrene [2]. On the basis of our case, \textit{C. perfringens} is also responsible for folliculitis, at least in immunocompromised patients. Other diagnoses that might be considered in immunocompromised patients with folliculitis include superficial bacterial infection due to \textit{S. aureus}, \textit{S. pyogenes}, \textit{Pseudomonas aeruginosa}, and other organisms; widespread fungal infection due to \textit{Candida albicans}, dermatophytes, \textit{Malassezia furfur}, and other organisms; demodicidosis; and HIV-associated cosinophilic folliculitis [1]. For our patient, such diagnoses were ruled out, given the histological results for the cutaneous biopsy, the negative results of color staining of the cutaneous biopsy specimen, and the results of bacteriologic cultures.


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**References**


**Isolation of Legionella pneumophila Serogroup 1 from Human Feces with Use of Amebic Cocultures**

Legionnaires’ disease is a serious pneumonic infection, caused mainly by the protozoan bacterium \textit{Legionella pneumophila}. Thirty to fifty percent of cases also develop diarrhea, but significant disease of the intestinal tract is rare [1–3]. Erythromycin, the antibiotic used most frequently for treating legionnaires’ disease, can cause diarrhea, but in one review [4], 16% (38%) of 42 cases had diarrhea as a presenting symptom. In some cases the diarrhea was of such severity that a diagnosis of salmonella or shigella gastroenteritis was considered [4]. The cause of diarrhea in untreated cases of legionnaires’ disease is unknown.

Twenty-two fecal specimens from 14 patients with legionnaires’ disease and 14 specimens from 14 other individuals initially suspected of having, but later shown not to have, legionnaires’ disease were cultured on a selective medium for legionellae (MWY; Oxoid, Basingstoke, Hants, England). The feces were also cultured on lawns of live \textit{Klebsiella aerogenes} NCTC (National Collection of Type Cultures) 7427 at 35°C for recovery of free-living amebas. No legionellae or amebas were isolated. The same fecal specimens were cocultured in tissue culture flasks with 10 mL of washed suspension of \textit{Acanthamoeba polyphaga} Linc Ap1 trophozoites (10^{7}/mL) at 35°C. The protocol used was based on previous experience [5]. Specimens from five patients with community-acquired cases of legionnaires’ disease (table 1) were then found to be culture positive for \textit{L. pneumophila} serogroup 1 (SG1). The strains from the five cases were subtyped by the Respiratory and Systemic Infection Laboratory (RSIL) (Public Health Laboratory Service [PHLS] Central Public Health Laboratory, Colindale, London); all had different restriction fragment length polymorphism (RFLP) types. Rare bright and moderately bright bacilli, consistent with legionellae, were detected by immunofluorescence in four of the five culture-positive feces (table 1). Four of the five culture-positive feces were diarrheal.

\textit{L. pneumophila} SG1 antigen was detected by ELISA (at RSIL) in urine samples from all the copropositive cases (table 1). The positive feces were obtained before three of the five patients produced antibodies to \textit{L. pneumophila} SG1 (table 1). Four of the cases produced antibody to \textit{L. pneumophila} SG1, but the remaining patient was still seronegative 15 days after onset of the infection. The coculture-negative feces obtained from the other nine cases were obtained mainly after the patients had begun to produce antibodies to \textit{L. pneumophila} and had received antibiotics for several days. Two of the culture-confirmed cases fulfilled the current PHLS Communicable Disease Surveillance Centre criteria for serologically confirmed cases of legionnaires’ disease. The other three cases did not even fulfill the serological criteria for presumptive cases. However, in two instances convalescent sera were not obtained.

\textit{L. pneumophila} SG1 was also isolated by direct plating and via coculture from the sputa of the two oldest cases (table 1). One sputum sample was obtained on the same day as the feces sample (case 4), and the other was obtained 2 days later (case 5). The sputum and feces isolates from each case were indistinguishable by monoclonal antibody subtyping and RFLP typing. The isolates from case 4 were of the same type as isolates from previous cases associated with the same hotel in Benidorm, Spain. The fecal isolate from case 1 was indistinguishable from a sputum isolate from another female case who stayed at the same holiday apartments in Salou, Spain. The other cases acquired their infections in the Dominican Republic (case 2), Majorca (case 3), and northern England (case 5).

Patient isolates are very important in legionellosis case-and-outbreak investigations. Coculture of early fecal specimens provides a means of isolating legionellae from patients who do not produce sputum (case 2) or from whom no sputa are available (cases 1 and 3). Stool samples should be collected carefully to avoid contamination with water that might contain legionellae. It is not known how long legionellae survive in human feces, but