**CASE REPORT**

**Gram-negative folliculitis caused by**

**Morganella morganii**

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**Key words:** folliculitis; gram-negative bacteria; Morganella morganii.

**INTRODUCTION**

*Morganella morganii* (MM) is an anaerobic gram-negative enteric bacterium belonging to the Enterobacteriaceae family. Urinary and hepatobiliary tracts, blood, and skin are the main ports of entry. The microorganism is an opportunistic pathogen usually presenting as a nosocomial infection involving the urinary tract and surgical wounds. However, MM can also present as bacteremia, meningitis, ecthyma gangrenosum, spontaneous bacterial peritonitis, chorioamnionitis, septic arthritis, endophthalmitis, and cutaneous abscess. Here we present a gram-negative folliculitis (GNF) caused by MM.

**CASE REPORT**

A 30-year-old man with unremarkable medical history referred to our clinic with a 1-year history of resistant nodulocystic lesions on his face and neck. The patient had already been treated with several courses of antibiotics including doxycycline, azithromycin, and clindamycin with only partial improvement. His medication history was also notable for 1-year intermittent use of a potent topical steroid on facial lesions. On physical examination, multiple pustular and nodulocystic lesions were noted mainly on his cheeks and neck (Fig 1).

Using a sterile technique, lesions were sampled, and obtained pus was sent for Gram staining and culture. Gram staining was positive for gram-negative rods. Three culture media including blood agar, MacConkey agar, and eosin methylene blue agar were used. After 24 hours, monomorphous gram-negative, catalase-positive, oxidase-negative, urease-positive, H2S-negative bacillus grew similarly on all media. Finally, biochemical standard methods confirmed the isolation of MM. Antibiogram found susceptibility to ciprofloxacin, cotrimoxazole, imipenem, and ceftriaxone; relative susceptibility to gentamycin; and resistance to tobramycin and azithromycin.

With the final diagnosis of *Morganella morganii*-induced gram-negative folliculitis (MMGNF), the patient was started on oral ciprofloxacin, 500 mg twice a day. In addition, weekly abscess drainage was done up to 3 weeks, and at 4 weeks, the lesions had resolved completely (Fig 2). Ciprofloxacin was continued for 4 more weeks and stopped. Finally, lesions did not recur over the 2-year course of follow up.

**DISCUSSION**

In 1968, Fulton et al 3 first described GNF in a group of acne vulgaris patients whose disease was resistant to conventional therapies. GNF mainly occurs in acne patients who have been using broad-spectrum antibiotics for a long time.

Generally, GNF caused by long-term antibiotic use has 2 different presentations. Commonly, it
presents with superficial pustular lesions mostly on the perinasal area (type I), which is mainly caused by infection with lactose-fermenting gram-negative rods including *Escherichia*, *Klebsiella*, and *Serratia*. Type II presentation, however, includes nodulocystic lesions developing from infection with *Proteus* species. Clinical presentation of this case was similar to that of *Proteus*-induced GNF (ie, nodulocystic lesions). This can be because both MM and *Proteus* belong to the tribe III of the Enterobacteriaceae, which could explain their similar presentation of infecting deeper layers of skin.

It is suggested that immunologic factors might play a role in the development of GNF. Given that MM is found mostly in immunosuppressed individuals, the fact that we found it in an immunocompetent person may, in part, be explained by the patient’s frequent topical corticosteroid use.

In our review of the literature, we did not find any report regarding MMGNF. This case presentation, adds *Morganella morganii*, another member of the Enterobacteriaceae family, to the list of other already-known pathogens that cause GNF. In addition, MMGNF can be treated with a similar approach used to treat GNF caused by other pathogens.

![Fig 1. Initial presentation of multiple pustular and nodulocystic lesions on the cheeks and neck.](image1)

![Fig 2. A, Partial improvement after 2 weeks of treatment with oral ciprofloxacin. B, Complete improvement after 4 weeks of treatment.](image2)

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