

Impetigo of Bockhart – A Case Report

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Abstract:

The most common superficial form of infectious folliculitis is called impetigo of Bockhart (BI) and is caused by *Staphylococcus aureus*. Clinically it is characterized by 1-6mm erythematous follicular-based papules or fragile pustules that may rupture and leave a yellow crust. The pustule is often pierced by hair that is easily extracted from the follicle. A case of BI is presented.

Keywords: *Staphylococcus*, folliculitis, Bockhart

Introduction:

A 40-year-old boy presented with pustular lesions on both his legs. The lesions were folliculo-centric and had been recurrent for the past five years with temporary short periods of remissions. The lesions were tender, painful, and pruritic. They began with an inflammatory follicular papule or pustule located over the front of both legs and accompanied by a burning sensation. Gradually, more pustules developed and eventually coalesced into scaly patches or plaques studded with pustules (Figures 1 & 2)



Figure 1: Showing folliculo-centric papules and pustules



Figure 2: Coalescence of individual lesions to form plaques

Diagnosis:

impetigo of Bockhart

Pathogenesis:

Bacterial folliculitis is characterized histologically by inflammatory cells within the wall and ostia of the hair follicle, associated with bacterial colonization, most commonly by *Staphylococcus aureus*. The inflammation can be limited to the superficial aspect of the follicle, primarily involving the infundibulum, or the inflammation can affect both the superficial and deep aspect of the follicle ¹.

Description/ Clinical Picture:

Lesions are most commonly located on the head and neck, trunk, extremities, buttocks, and axillae. The lesions can be tender, painful, or simply pruritic. Because of the superficial nature of the process, scarring is uncommon. Up to 20% of patients with bacterial folliculitis experience recurrent disease, which may be the result of community-type methicillin-resistant *Staphylococcus aureus* ².

Furuncles are deep folliculitis that typically involves the entire hair follicle and the surrounding subcutaneous tissue. Coalescence of several furuncles results in a carbuncle or subcutaneous abscess. Patients present with a tender, erythematous nodule, often with a central pustule that may drain on its own³.

The differential diagnosis includes other forms of folliculitis that arise from fungal infections (*Pityrosporum* folliculitis, tinea barbae, and Majocchi's granuloma), herpes, Demodex, or gram-negative bacteria. In addition, drug-induced folliculitis, acute generalized pustulosis, pseudofolliculitis barbae, eosinophilic folliculitis, acne, keratosis pilaris, impetigo, miliaria, and an id reaction should be considered in the differential.

These entities can be sorted out by bacterial, viral, or fungal cultures (or polymerase chain reaction [PCR]); combined with skin biopsies, Gram stains for bacteria, periodic acid-Schiff (PAS) stains for fungi, or a Tzanck preparation (with or without direct immunofluorescence) for herpes.

Management:

For recurrent uncomplicated superficial folliculitis, the use of antibacterial soaps and good handwashing may be all that is needed. For refractory or deep lesions secondary to gram-positive organisms, empiric treatment with topical and/or oral antibiotics may be beneficial^{4,5}.

Since *S aureus* is the most common pathogen, systemic therapy must cover this organism and other gram-positive bacteria. *S. aureus* is penicillin-resistant; therefore,

dicloxacillin or a cephalosporin is the first line of treatment. Methicillin-resistant organisms are becoming more common, and treatment may require clindamycin, trimethoprim-sulfamethoxazole, minocycline, or linezolid. The newest medications that have been approved for methicillin-resistant *S. aureus* (MRSA) infections are ceftaroline, dalbavancin, oritavancin, and tedizolid.

If a patient does not improve with a standard course of antibiotics, other causes of folliculitis must be investigated.

For recurrent and recalcitrant folliculitis, mupirocin ointment in the nasal vestibule twice a day for five days may eliminate the *S. aureus* carrier state. Family members also may be nasal carriers of *S. aureus*; mupirocin ointment in the nasal vestibule twice a day for five days and/or 600mg/day of rifampin orally for ten days is recommended.

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