Disseminated *Mycobacterium chelonae* infection: Complicating a case of hidradenitis suppurativa

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ABSTRACT

Mycobacteium chelonae is a rapidly growing atypical mycobacteria known to be pathogenic in humans. We report a case of Hidradenitis Suppurativa (HS) with diabetes complicated by infection of the lesions with *Staphylococcus aureus* and *M. chelonae* leading to non-healing and discharging lesions. HS is a rare, insidious and debilitating disease characterized by swollen, painful, inflamed lesions in the axillae, groin, and other parts of the body that contain apocrine glands. Discharge from HS lesions are often found to be sterile, however, polymicrobial bacterial colonization commonly occurs within sinus tracts which can lead to offensive smelling discharge, infection, cellulitis, and superinfection. The incidence of HS is very low and the association with *M. chelonae* makes it a rare and interesting case.

Key words: Diabetes, hidradenitis suppurativa, *Mycobacterium chelonae*

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INTRODUCTION

Mycobacterium chelonae is a rapidly growing non-tuberculous mycobacterium that is a saprophyte and causes infection following incidental environmental inoculation.[1] Hidradenitis Suppurativa (HS) is a distressingly chronic, disabling, suppurative, and cicatricial follicular disease that primarily affects apocrine gland-bearing skin sites such as the axilla and ano-genital areas with a prevalence rate of 1 in 3,000.[2] Clinically, the disease often presents with tender subcutaneous nodules beginning around puberty. The nodules may spontaneously rupture or coalesce, forming painful, deep dermal abscesses and eventually, fibrosis with the formation of extensive sinusoidal tracts results.[3] It is now more or less evident that HS is not a true infectious process and bacterial infection if any is only secondary.[2] *M. chelonae* is an atypical "fast-growing Mycobacteria" that is a rare cause of human infection. Although *M. chelonae* can be found in many cutaneous sites, infection occurs most commonly after skin trauma, surgery, injections, or minor injuries.[4] Though many bacteria have been reported as the causative agents of secondary infection in HS, infection due to *M. chelonae* is not documented. We report a rare case of HS in a patient with diabetes complicated by secondary bacterial infection with *M. chelonae* and *Staphylococcus aureus*.

CASE REPORT

A 28-year-old, married, male presented with complains of pain, discomfort and purulent discharge from both axilla and inguinal regions since 4 years duration with intermittent partial regression and relapse. He had been treated by general practitioners for all these years with systemic and local antibiotics without much relief. He also complained of low-grade intermittent fever, weight loss and weakness since 6 months.

On examination, suppurative nodules, plaques, and discharging sinuses were seen over both the axilla and inguinal region with involvement of the scrotum [Figures 1-3]. The discharge consisted of cream colored pus without granules or any specific odour. In some areas there were healed lesions with scarring and fibrosis. Axillary and inguinal group of lymph nodes were palpable and non-tender. He had normal pulse rate, blood pressure, chest was clear and no organomegaly was seen. Laboratory investigations revealed hemoglobin 11.6 g%, TLC 9,000/mm^3, Differential

count showed N 64%; L 32%; E 3%; M 1%; ESR 60 mm in 1st hour and fasting blood sugar 230 mg/dl. Mantoux test was negative. Serum for VDRL and HIV tests was non-reactive. Chest X-ray was normal. Gram staining of the purulent discharge from the axilla and inguinal region showed the presence of Gram positive cocci in clusters [Figure 5]. Zeihl Neelsen (ZN) staining revealed the presence of acid fast bacilli [Figure 4]. The pus sample from all the four sites were inoculated separately in blood agar, MacConkey agar, Lowenstein-Jensen (LJ) media in duplicate, one covered with black paper to rule out scotochromogens and in SDA slants. After 48 h of incubation two types of colonies were seen on MacConkey agar media, which was found to be Gram positive cocci in clusters and acid fast bacilli. On the 6th day non-pigmented colonies were seen in LJ media which were found to be acid fast [Figure 6] No growth was seen in SDA slants after 4 weeks of incubation. Both the organisms were identified as *S. aurues* and *M. chleonae* based on preliminary staining, growth characteristics and biochemical findings. *M. chelonae* and *S. aureus* were isolated from all the four sites from where samples were collected. Biopsy of the lesion showed foreign body type granulomas around the hair follicle with inflammatory cell infiltrate, fibrosis and destruction of appendageal structures suggestive of HS. Differential diagnosis of furnculosis, scrofuloderma, actinomycosis, lymphogranuloma venereum and granuloma inguinale were ruled based on microscopic findings of the pus, absence of genital ulcer as well as history of contact, no spread from bone or lymph node, or on any bony prominence, chronicity and bilateral nature of the lesions.

The lesions were drained after excision to provide local relief to the patient. The infection was treated with clarithromycin (500 mg) twice daily for 12 weeks, injection tobramycin (16 mg) twice daily for 2 weeks along with local application of mupirocin. The discharge was sterile on culture after 2 weeks of treatment. Isotretinoin (40 mg daily in two divided doses), dapsone (100 mg daily) and zinc gluconate (200 mg daily) were given for 6 months. He was also given insulin as his

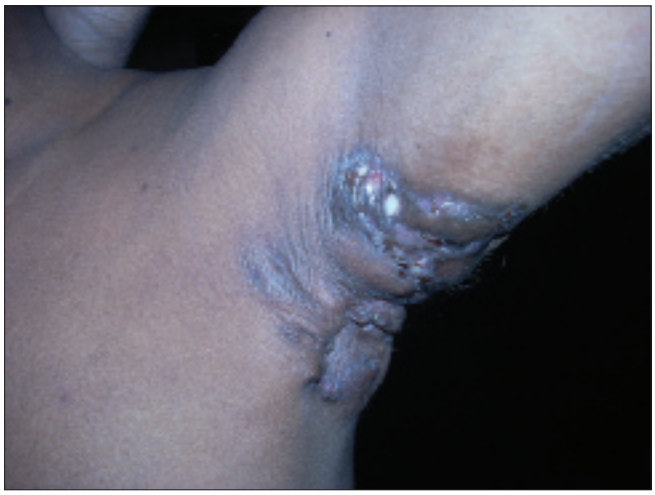

Figure 1: Plaques, nodules, discharging sinuses and healed scars over the left

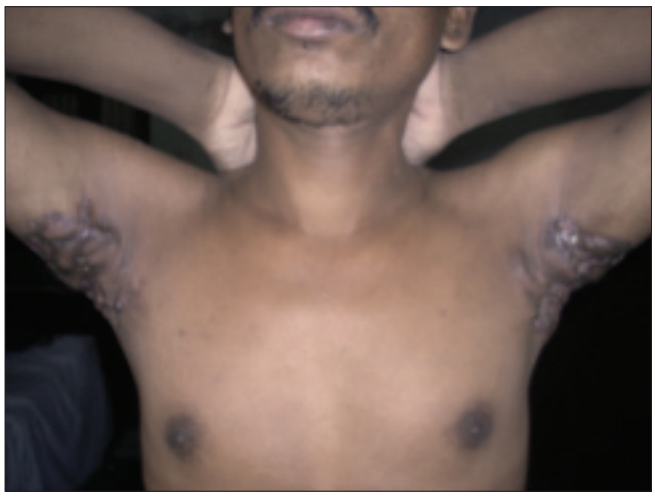

Figure 2: Involvement of both axillae

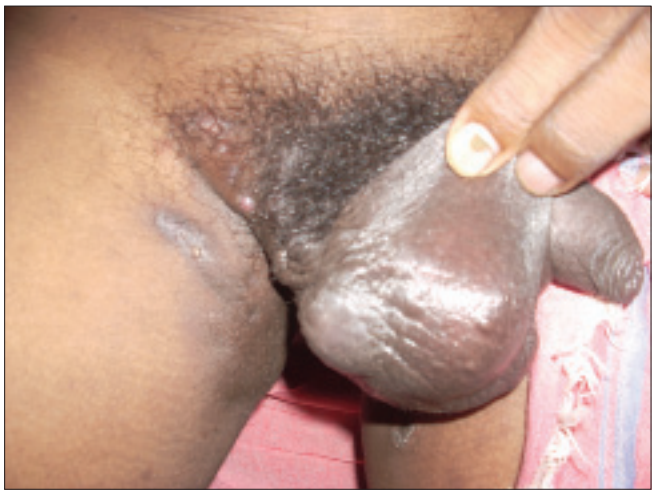

Figure 3: Discharging nodules, plaques and scaring over the right inguinal region

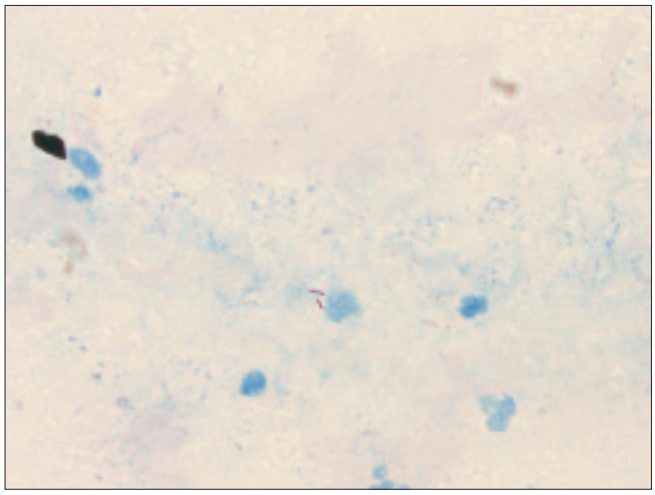

Figure 4: Zeihl Neelsen staining of the discharge showing acid fast bacilli

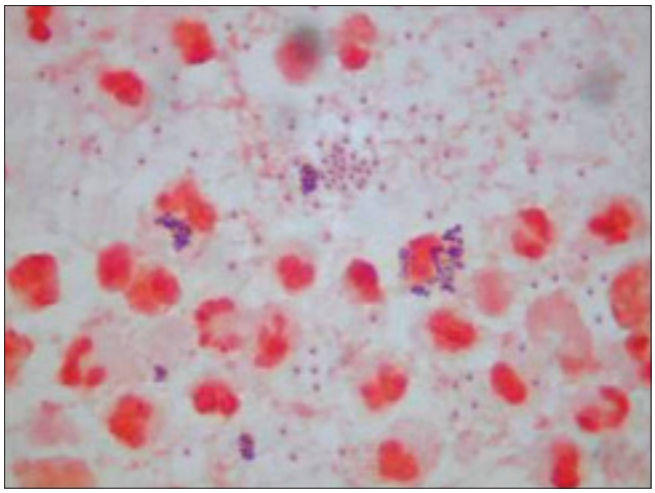

Figure 5: Gram staining of the discharge showing presence of Gram positive cocci

fasting blood sugar was high. The lesions showed signs of regression with disappearance of the distressing symptoms like tenderness, swelling and discharge.

DISCUSSION

M. chelonae has been implicated as pathogen in skin, soft tissue, postoperative wound infection and keratitis. It is a rapidly growing, non-tuberculous mycobacterium that is ubiquitous in the environment and present word-wide. They have been found in soil, marshes, rivers, municipal water supplies and in marine and terrestrial life forms. The organism is classified as a Runyon group IV mycobacterium that forms non-pigmented colonies.[1] Over the last two decades, there has been a rise in the proportion of mycobacterial infections caused by non-tuberculous species like *M. chelonae*.[5] It is a rapidly growing atypical mycobacteria that can cause both systemic and cutaneous infections. Organisms gain entry into the host by inoculation into the skin and the subcutaneous tissue during trauma, injection, surgery or through animal contract. The characteristic presentation in immunocompromised patients is a disseminated cutaneous infection whereas localized cellulitis or abscess is typical in immunocompetent patients.[4]

HS is caused by follicular occlusion, which in turn, occludes the apocrine glands and causes perifolliculitis. It is confined to areas of the body that contain apocrine glands. These areas are the axillae, areola of the nipple, groin, perineum, perianal, and periumbilical regions. HS is painful and can be disabling but is rarely fatal, except when it progresses to overwhelming systemic infection in an immunocompromised patient. Extensive disease can prevent patients from performing normal work and engaging in normal social activities. In some patients, especially those with severe disease, the condition creates significant psychological problems, particularly regarding sexual relationships. The lesions in HS begin as

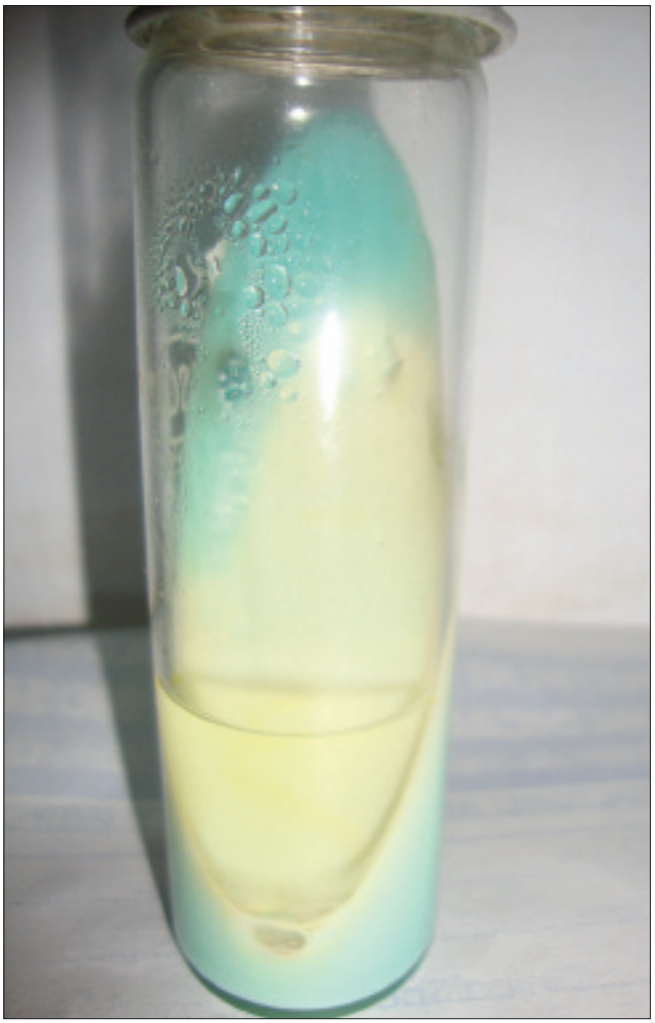

Figure 6: LJ medium showing growth of *Mycobacterium chelonae*

boils or tender nodules that are painful and do not point readily. Within hours to days the abscesses grow in size, and if untreated, break through the overlying skin discharging purulent or seropurulent discharge gradually leading to the formation of chronic sinuses. Secondary infection however may occur when the abscesses rupture on to the surface. Healing occurs with fibrosis, with the formation of band-like scars. Recurrences are common and complete healing of resolving abscesses never takes place.[2] However, several treatment options are available including preventive, medical, surgical and psychological modalities. As the disease manifests in a variety of forms, treatment should be based on the patient's presentations and circumstances.[6]

In this case the patient was suffering from diabetes mellitus rendering him immunocompromised. The patient may have acquired the infection from the environment and gained its entry through the sinusoidal openings caused by HS. The organisms may have disseminated as the patient was immunocompromised and was not treated successfully due to delay in diagnosis. He exhibited the classic presentation of HS with secondary

multibacterial infection caused by *M. chelonae* and *S. aureus*. There have been no reports of HS with secondary infection due to *M. chelonae* to the best of our knowledge. HS is an insidious and debilitating disease often misdiagnosed and under-managed. All patients of HS with abscesses or discharging sinuses should be subjected to microbiological evaluation to rule out secondary infections which accounts for distressingly chronicity and supportive nature of lesions. Mycobacterial causes should be kept in mind in diagnosing such cases.

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