Atypical Mycobacterial Infection: A Case Report

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ABSTRACT

A 58 years old lady, a known case of Rheumatoid arthritis involving large as well as small joints of the body since 27 years presented with widespread skin ulcers all over the body since 2 months. Prior to the onset of the ulcers she had been treated with oral steroids and methotrexate for rheumatoid arthritis in varying doses and duration. After the onset of ulcers too she had been treated with steroids on suspicion of rheumatoid vasculitis being the cause for the ulcers. She had been on antidiabetic drugs for previous 8 months due to elevated blood sugar levels.

Dermatological examination revealed multiple tender soft nodules and abscesses breaking down to form ulcers over both lower limbs, hip girdles, and upper limbs. Ulcer bases were necrotic and purulent discharge was evident from most ulcers. Some of the nodules were breaking down to form ulcers. No petechiae or purpura was seen. No cribiform scars were seen.

Patient was started on parenteral antibiotics pending bacteriology of the pus discharge from the ulcers. ZN stain of the pus revealed strongly acid fast thin beaded filamentous bacteria in large numbers suggestive of Atypical Mycobacterial Infection. Pending the culture and species identification, patient was started on parenteral antibiotics targeted towards atypical mycobacteria.

Over a few days the general condition of patient deteriorated as she de-saturated and had to be ventilated in the ICU. Tracheal aspirate also grew atypical mycobacteria and in addition Acinetobacter from wound swab. Despite best combination of antibiotics targeting both the infections, she continued to deteriorate into sepsis and succumbed to the illness.

Aim of presentation is to highlight the rarity of the case, importance of atypical mycobacteria as a cause of ulcers in immunocompromised patients, and resistance to chemotherapy of atypical mycobacteria resulting in the demise of the patient.

Keywords: Atypical mycobacterial infection, Mycobacterium abscessus, Rheumatoid arthritis, Immunodeficiency

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INTRODUCTION

Atypical mycobacteria are opportunistic, acid-fast and ubiquitous organisms. With increasing immunosuppression due to Human Immunodeficiency Virus and immunosuppressants usage in malignancies, collagen vascular disorders and transplant patients, incidence of atypical mycobacterial infection is rising, outnumbering M.tuberculosis\(^1,2\). Here, we report one such case where atypical mycobacterial infection mimicked multiple cutaneous vasculitic ulcers in a patient of Rheumatoid arthritis.

CASE REPORT

A 58 year old female farmer, with Rheumatoid Arthritis since 27 years, was on immunosuppressants with Methotrexate, oral steroids and hydroxychloroquine for 8 months prior to presentation. Treatment for Diabetes Mellitus was initiated two months back with oral hypoglycemic agents. She presented with multiple crops of painful raised nodular lesions over the hips, gluteal area, lower and upper limbs since 2 months. These nodules spontaneously ulcerated within 3-4 days with profuse pus discharge. She also had high grade fever with chills since 2 months, significant weight loss of 10kg in 3 months associated with loss of appetite and was bed bound.

Examination showed multiple, tender, well-defined, punched out ulcers of varying sizes with profuse discharge over upper limb, lower limbs and gluteal area. Ulcers had necrotic base, undermined edge, livid hue around the margins (Figures 1, 2). A differential diagnosis of rheumatoid vasculitis and Pyoderma gangrenosum was considered. Investigations revealed anemia (Hemoglobin-10gm/dl), random blood sugar-355mg/dl, and HIV negative. Ziehl Neelsen stain of pus discharge from ulcers showed long filamentous, beaded acid fast bacilli resembling Atypical Mycobacteria (Figure 3). Culture on Lowenstein Jensen medium showed growth of buffy colored colonies in 5 days. PCR test for \textit{M.tuberculosis} was negative.

Treatment was started with intravenous Inj Amikacin 375mg q12h, Inj Clarithromycin 500mg q12h and Inj Imipenem 1gm q6h. After an initial favorable response, the patient started desaturating, requiring ventilation. HRCT lung revealed bilateral lower lobe consolidation, and bronchoscopic guided tracheal aspirate showed the same organism (Figure 4).

A diagnosis of Disseminated Atypical Mycobacterial infection was made, possibly by rapid growers, pending specific tests. \textit{Acinetobacter} superinfection was also detected from the wound cultures. Despite aggressive antibiotic therapy including Tigecycline cover for \textit{Acinetobacter}, patient succumbed to overwhelming sepsis from both the infections.

Sub-typing of the atypical mycobacteria by biochemical tests (Nitrate reductase, iron uptake, Tween 80 hydrolysis– negative; arylsulphatase – positive) identified the organism to be \textit{Mycobacterium Abscessus}. 
DISCUSSION

The index case had disseminated cutaneous ulcers all over the body (25-30 ulcers) with pulmonary infection in the setting of immunosuppression from repeated steroid intake for treatment of rheumatoid arthritis and diabetes mellitus. The atypical mycobacterium was demonstrated both in skin and tracheal aspirate cultures confirming the diagnosis of disseminated infection. Despite exhibiting the patient to the cocktail of antibiotics known to treat *M. abscessus*, the patient succumbed to the infection suggesting the severity of infection and chemoresistant nature of this organism.

*Mycobacterium Abscessus* is a rapidly growing mycobacterium first described by Moore and Frerichs in 1953. *M. abscessus* was formerly considered to be a subspecies of *Mycobacterium chelonae* (*M. chelonae* subsp. *abscessus*), but on the basis of DNA homology studies it has been shown to be genetically distinct and has thus been elevated to a separate species status.

The major threat posed by this species is mainly due to its resistance to antibiotics. Indeed, *M. abscessus* is one of the most resistant organisms to chemotherapeutic agents. *M. abscessus* produces enzymes that potentially degrade or modify antibiotics, which can result in their inactivation. It contains an aminoglycoside 2 – N – acetyltransferase and phosphotransferase that can confer aminoglycoside resistance.

*M. abscessus* is also known to be of three types of which one is relatively more chemoresponsive than the other two. Treatment of infections due to *M. abscessus* complex may benefit from molecular identification within the complex since *M. massiliense* appears more susceptible than *M. abscessus sensu stricto* and *M. boletii*. If the index case was infected with any of the chemoresistant serovars of *M. abscessus* it may explain the failure to respond to known antibiotics. However the facility to do subclassification of *M. abscessus* was not readily available at the time.

In conclusion, atypical mycobacterial infection poses both a diagnostic as well as a therapeutic challenge especially in patients with collagen vascular disorders on immunosuppression. Multiple cutaneous ulcers in a bedridden, known Rheumatoid arthritis patient are most commonly treated for vasculitic causes or for pyoderma gangrenosum with oral or parenteral steroids, which can further aggravate any infection. Hence a simple bedside ZN Stain or Gram stain is a must to exclude any bacterial infection before treatment is started with steroids.

Since the clinical presentation and treatment of subtypes of atypical mycobacteria vary, it is essential to obtain subtyping early in course of the illness to initiate appropriate treatment. The treatment of a patient with atypical mycobacteria remains a challenge even today due to the variable response to antibiotics.

Aim of presentation is to highlight a rare infective cause for ulcers in an immunocompromised patient and the poor outcome in disseminated atypical mycobacterial infections despite antibiotic therapy.
Figure 1: Multiple non healing ulcers over right gluteal region and thigh showing ragged and undermined edges

Figure 2: Multiple large ulcers on right upper limb and elbow of similar nature
Figure 3: ZN stain of pus swab showing numerous long filamentous acid fast bacteria (x 1000)

Figure 4: ZN stain of tracheal aspirate also showing numerous acid fast beaded bacteria (x 1000)
REFERENCES