An Unusual Presentation of Cutaneous Tuberculosis for Surgeons—Review Of Literature

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Abstract Tuberculosis is one of the oldest diseases on earth. With rising trends of immunosuppressive states, the extrapulmonary manifestations of tuberculosis have emerged and are flourishing. Most often it is the systemic dissemination of the bacilli from the primary infective foci during the periods of low immunity. Cutaneous manifestation of tuberculosis in the form of injection abscess or traumatic inoculation is well known; however, sporadic occurrence of it in the form of abscess with no underlying lesions is quite rare. This would caution the treating units in the endemic areas about the possibility of tuberculosis as the disease in any inflammatory disorder of skin with acute presentations with no systemic symptoms. Only a high index of suspicion would render an early detection and recovery. Our case is one such incident.

Keywords Cutaneous tuberculosis · Rare presentation · Surgical debridement

Introduction

Tuberculosis (TB) caused by *Mycobacterium* is a worldwide prevalent communicable disease that has scorched the humans since their inception in this world. The disease was found in the vertebrae of the Neolithic humans in Europe and Egyptian mummies, but it was not until c that Robert Koch discovered the causative agent as *Mycobacterium tuberculosis*.

Most commonly TB is an airborne disease of the lungs. The skin manifestations present secondary to hematogenous spread or from the direct tissue extension from a latent or an active underlying focus of infection. The incidence of cutaneous TB (CTB) has fallen from 2 to 0.15% in India, whereas it is rare in developed countries [1] due to the availability of effective anti-TB drugs, elimination of dairy herds, and general improvement in the living standards [2].

However, its prevalence is increasing in the past few decades due to the rampant diseases of immunosuppression such as HIV, cancer, diabetes mellitus, end-stage renal disease, congenital immunodeficiency, and the high rates of immunosuppressive usage for cancer chemotherapy [3]. Many clinical variants of CTB have been extensively studied and reported, but clinical identification of the disease and microbiological confirmation, even with the most evolved and sophisticated laboratory methods, is still difficult in view of its nonspecific presentations. Hence, it is important to consider this infection when faced with a suggestive clinical picture [4].

Aim of this case report is to point out that in differential diagnosis of skin lesions taking longer duration to heal, it is necessary to take TB into consideration as well. We present a rare case of primary CTB that required surgical procedure.

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Case Report

A 46-year-old woman, with diabetes, presented to us with multiple pustules and brownish discoloration of the skin over the left thigh and fever which began 20 days earlier. She consulted many physicians and was prescribed antibiotics (amoxicillin/clavulanic acid, ampicillin/sulbactam), analgesics, and anti-inflammatory medicines/antipyretics each time with a diagnosis of erysipelas. Even after 5 days there was no improvement. In our hospital, she had multiple well-defined punched-out ulcerative lesions (Fig. 1), floor covered with unhealthy tissue and purulent, surrounding brawny edematous changes occupying the medial and the anterior parts of the left thigh. Multiple abscesses with pus points were also noted. The lesions were warm and tender with notable expression of purulent material through the multiple ulcers on light pressure. Mild continuous non-spiking fever was present since the lesions appeared. The patient however reported no other symptoms of cough, chills, fatigue, trauma, or any previous exposure or treatment for TB. On general physical examination, there was no evidence of poor nutrition, lymphadenopathy, or other skin lesions. She had been vaccinated in her childhood with BCG as evident with the scar in her left arm.

As the lesion had multiple abscess cavities, the pus was aspirated and sent for culture sensitivity, which showed *Streptococcus* and *Acinetobacter* growth, both sensitive to linezolid, and hence the same was started (600 mg OD). There was no evident healing noted and some of the abscesses burst open (day 3). Hence, an incision and drainage of the infected wound was performed over the most dependent

Fig. 2 Incision and drainage of the infected wound performed over the most dependent area

area (Fig. 2). Tissue biopsy from the wound edge was sent for histopathological examination and mycobacterial culture. Histopathology showed multiple neutrophilic microabscesses in the epidermis, mixed neutrophilic, and chronic granulomatous infiltrate in the dermis. However, no caseous necrotic areas were seen. Ziehl–Neelsen stain for acid-fast bacilli showed very few acid-fast bacilli. As it was suggestive of chronic inflammatory process, the patient was started on anti-TB therapy as per the guidelines of Revised National Tuberculosis Control Program (RNTCP) with isoniazid, rifampicin, pyrazinamide, and ethambutol. Regular dressings were done.

After 3 weeks, the culture of the tissue on the Lowenstein–Jensen medium grew *M. tuberculosis*. Sputum for acid fast bacillus (AFB) was negative. The X-ray of the chest showed

Fig. 1 Multiple well defined punched out ulcerative lesions, floor covered with unhealthy tissue and surrounded by brawny edema

no pulmonary lesions and the X-ray of the spine and left thigh showed no evidence of TB of the bone.

One month later there was a good resolution of the cutaneous lesions with healing granulation tissue (Fig. 3). A follow-up after 6 months showed complete healing.

Discussion and Review of Literature

CTB is usually caused by *M. tuberculosis*, *Mycobacterium bovis* and rarely by bacillus Calmette-Guerin [5]. Although human disease with *M. tuberculosis* and *M. bovis* is usually spread by droplets and the portal of entry is often the respiratory tract, skin can also be primarily involved.

Cutaneous involvement represents only 2% of extrapulmonary TB [6]. In the past, it was more prevalent in temperate countries with cold and humid climate with a few hours of daily sunlight, but now it is being encountered in tropical countries like India. Malnutrition and low socioeconomic conditions are predisposing factors for CTB. Two decades back a decline was observed in the incidence of CTB, but recently there is resurgence of CTB due to multidrug-resistant strains of *M. tuberculosis* [7]. Incidence of CTB appears low in areas such as India and China where TB prevalence is high. Cutaneous manifestations of TB have been found in less than 0.1% of individuals seen in dermatology clinics [8].

An AFB smear is useful if lesions have a high bacterial load as seen in lichen scrofulosorum (LS), miliary TB, and TB gumma. Currently, polymerase chain reaction (PCR) appears to be the most useful in multibacillary forms of CTB [9, 10]. In one report of AFB-negative specimens, the overall sensitivity of PCR was found to be 50–72% [11]. Another source reported AFB-positive respiratory specimens to have a sensitivity and specificity of 95 and 98%, respectively [12].

Fig. 3 Good resolution of the cutaneous lesions with healing granulation tissue after 1 month of AntiTubercularTherapy(ATT)

Nonetheless, a combination of diagnostic tests is needed when a case of CTB is suspected to confirm a diagnosis. In developing countries, PCR is not always readily available and therefore physicians must rely on a positive response to anti-TB drugs to confirm difficult cases [11, 13]. It is essential to search for extracutaneous foci of TB by urine, blood, and sputum samples; X-ray or CT scan of the chest; and bone scans. It is imperative that physicians have a high index of suspicion in high-risk patients and in atypical presentations.

There is a delay in the diagnosis because CTB is not always considered in the differential diagnosis of atypical or nonhealing skin lesions [14].

The three most common forms of CTB are scrofuloderma, lupus vulgaris, and tuberculosis verrucosa cutis (TBVC). Scrofuloderma forms the largest group followed by lupus vulgaris (23.52%) and TBVC (13.73%) [15]. Lupus vulgaris occurs mainly in patients with moderate or high degree of immunity. It occurs secondary to inoculation by exogenous source and by hematogenous spread. Scrofuloderma manifests after the breakdown of the skin overlying a tuberculous focus usually a lymph node but sometimes an infected bone or joint. A patient with moderate or high degree of immunity can develop TBVC if accidental superinfection from extraneous source and autoinoculation or post-traumatic inoculation with infected sputum occurs. Even though *M. tuberculosis* is the most common etiological agent of CTB in the morphological variants of scrofuloderma, lupus vulgaris, and TBVC, *M. scrofulaceum* and *M. avium* complex are also isolated [16]. In a study Banerjee noticed association of pulmonary TB in 45% cases and Wong noticed it in 10% indicating the variable nature of the association of the CTB [17, 18].

CTB can occur following any injury. Postinjection tubercular abscess is a well know, yet a very rare entity. There are two modalities of infection. First, a primary inoculation of the organisms can occur through a contaminated injection material or instrument, which is usually rare [19]. The second and common pathogenesis is seen during the incubation period and in those who have recently contacted primary infection. During this early stage of the disease, a number of bacilli reach the bloodstream, either directly from the initial focus or via regional the lymph node and thoracic duct [19, 20]. This sporadic dissemination, also termed as occult hematogenous TB, takes place for a very short time and is unlikely to continue after delayed hypersensitivity develops. During this period the main clinical manifestation is fever lasting for a few days. The bacilli are seeded at various organs and may heal completely, progress to active lesions, or remain quiescent, containing viable bacilli to become active again during periods of lowered immunity.

Microhematoma that occurs at the injection site or at the traumatic sites, in patients with primary infection, acts as an

area of lowered resistance resulting in seedling of tubercular bacilli that get fixed at these sites and later progress to abscess formation [21]. A hypothesis is that the high lactic acid content, absence of reticuloendothelial cells, and lymphatic tissue with very rich blood supply may help in localization of *Mycobacterium* in the muscles [22], but such an occurrence in skin is not yet defined. In a study by Patra et al. [23], BCG vaccination scar was found in 59.62% of cases of TB, which reflects the incapability of the vaccine to protect TB completely.

However, an isolated tuberculous infection and abscess is a rare phenomenon reported rarely [24]. A case of primary TB of the cheek skin has been reported in the endemic Balkan area requiring surgical treatment along with anti-TB therapy [25].

Thus, tubercular infection/abscess should be considered in the differential diagnosis of any chronic infection/local abscess that forms especially if there has been an interval of 2–3 weeks between injury and the development of abscess.

Conclusion

CTB is one of those myriad of presentations of TB especially in high-risk patients. It should be considered in the differential diagnosis of any chronic and atypical skin lesions suggestive of underlying infectious etiology. This can be achieved with high index of suspicion and appropriate diagnostic methods. Any lesion with high suspicion should always be investigated for TB, at least with the tissue culture and histopathology. Attempt should always be made at earliest diagnosis so that appropriate curative treatment can be initiated and the disease is cured with less morbidity.

References

1. Sehgal VN, Srivastava MD, Khurana VK et al (1987) An appraisal of epidemiologic, clinical, bacteriologic, histopathologic and immunologic parameters in cutaneous tuberculosis. *Int J Dermatol* 26:521–516
2. Kumar B, Kaur S (1986) Pattern of cutaneous tuberculosis in North India. *Ind J Dermatol Venereol Leprol* 52:203–207
3. Handog EB, Gabriel TG, Pineda RT (2008) Management of cutaneous tuberculosis. *Dermatol Ther* 21(3):154–161
4. Rai VM, Sheno SD, Gowrinath (2005) Tuberculous gluteal abscess coexisting with scrofuloderma and tubercular lymphadenitis. *Dermatol Online J* 11(3):14
5. Frankel A, Penrose C, Emer J et al (2009) Cutaneous tuberculosis: a practical case report and review for the dermatologist. *J Clin Aesthet Dermatol* 2(10):19–27
6. Yasmeen N, Kanjee A (2005) Cutaneous tuberculosis: a three-year prospective study. *J Pak Med Assoc* 55(1):10–12
7. Bazex J, Bauriaud R, Margeury MC (1996) Cutaneous mycobacteriosis. *Rev Pract* 46(13):1603–1610
8. Thomas Herenlippe, MP, chief editor, et al emedicine.medscape.com/article/230802 overview
9. Tan SH, Tan BH, Goh CL et al (1999) Detection of *Mycobacterium tuberculosis* DNA using polymerase chain reaction in cutaneous tuberculosis and tuberculids. *Int J Dermatol* 38(2):122–127
10. Pai M (2004) The accuracy and reliability of nucleic acid amplification tests in the diagnosis of tuberculosis. *Natl Med J India* 17(5):233–236
11. Hsiao PF, Tzen CY, Chen HC, Su HY (2003) Polymerase chain reaction based detection of *Mycobacterium tuberculosis* in tissues showing granulomatous inflammation without demonstrable acid-fast bacilli. *Int J Dermatol* 42(4):281–286
12. Diagnostic Standards and Classification of Tuberculosis in Adults and Children. This official statement of the American Thoracic Society and the Centers for Disease Control and Prevention was adopted by the ATS Board of Directors, July 1999. This statement was endorsed by the Council of the Infectious Disease Society of America, September 1999. *Am J Respir Crit Care Med*. 2000; 161:1376–1395
13. Ramam M, Mittal R, Ramesh V (2005) How soon does cutaneous tuberculosis respond to treatment? Implications for a therapeutic test of diagnosis. *Int J Dermatol* 44(2):121–124
14. Frankel A, Penrose C, Emer J (2009) Cutaneous tuberculosis: a practical case report and review for the dermatologist. *J Clin Aesthetic Dermatol* 2(10):19–27
15. Pandhi RK, Bedi TR, Kanwar AJ et al (1977) Cutaneous tuberculosis—clinical and investigative study. *Ind J Dermatol* 22:99–117
16. Gopinathan R, Pandit D, Joshi J, Jerajani H, Mathur M (2001) Clinical and morphological variants of cutaneous tuberculosis and its relation to *Mycobacterium* species. *Indian J Med Microbiol* 19:193–196
17. Banerjee RK, Bedi TR, Kanwar AJ, Bhutani LK (1977) A clinical and investigative study of cutaneous tuberculosis. *Indian J Dermatol* 22:63–66
18. Wong K, Lee KP, Chiu SF (1968) Tuberculosis of the skin in Hong Kong. A review of 160 cases. *Br J Dermatol* 80:424–429
19. Speert DP (1992) Tuberculosis. In: Krugman S, Katz SI, Gershon AA, Wilfort CM (eds) *Infectious diseases of children*, 9th edn. Mosby, Missouri, pp 551–552
20. Kovats F, Miskovits G, Hutas I (1955) Experimental studies on the formation of tuberculous abscesses following intramuscular injections. *Orv Hetil* 96:1126–1129
21. Jones VS, Philip C (2005) Isolated gluteal tuberculosis. *Indian Pediatr* 42:955
22. Peter CK (1937) Some thoughts on tuberculosis of fascia and muscle. *Lancet* 57:156–159
23. Patra AC, Gharami RC, Banerjee PK (2006) A profile of cutaneous tuberculosis. *Ind J Dermatol* 51(2):105–107
24. Abdelwahab IF, Kenan S, Hermann G, Klein MJ (1998) Tuberculous gluteal abscess without bone involvement. *Skeletal Radiol* 27:36–39
25. Knezevic P, Knezevic B, Skerlev M, Uglesic V (2009) Primary tuberculosis of the cheek skin: difficulties in diagnostic procedure. *MJMS* 2(3):235–238