Scrofuloderma with psoriasis - A case report

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ABSTRACT

Scrofuloderma is caused by tuberculous involvement of the skin by direct extension, usually from underlying tuberculous lymphadenitis, which is rarely seen in psoriatic patients. We report a case of psoriasis with scrofuloderma, where, the patient presented with bilateral groin swellings for last two months which ruptured few days back along with purulent discharge. Smear was made from this purulent discharge and acid fast bacilli were demonstrated in it. There were erythematous scaly lesions all over the body with silvery white scales, pain and deformity in small joints of hands.

Key words: tuberculous, psoriasis, scrofuloderma, acid fast bacilli

INTRODUCTION

Psoriasis is a chronic, relapsing and remitting inflammatory skin and joint disease that has prevalence of 2-3% of world population.[1] Men and women are equally affected and exhibit a bimodal distribution with a peak between 15-30 years of age and another between 50-60 years of age.[1] It results from the interaction between genetic and environmental factors and can cause significant impairment of physical, emotional and psycho-social well being of patients.[2,3] Now-a-days many biological agents such as tumor necrosis factor- Alfa (TNF-α) inhibitors are used for psoriasis treatment along with the traditional therapies.[4,5] All the TNF-α inhibitors are associated with an increased risk of developing active disease in patients with latent tuberculosis infection. Psoriasis per se could represent an independent risk factor for tuberculosis since, interestingly, an unexpected high prevalence was found in patients affected by latent mycobacterium tuberculosis infection (LTBI=18%).[6] There are only scattered reports of pulmonary TB in psoriatic patients, but none of scrofuloderma.[7] This prompted us to report this interesting case.

CASE REPORT

58 years old man, married and shopkeeper by occupation came to the outpatient department with complains of bilateral groin swellings, ulcers, skin lesions and painful small joints of hands. Two months back, he had bilateral groin swellings, 2-3 inches in size initially, which gradually increased in size and ruptured. Now he was having bilateral inguinal ulcers with undermined edges and purulent discharge. He gave history of erythematous plaque with silvery white scales over the limbs for last 20-25 years. He had been treated by general practitioners with pain killers, steroids and a variety of topical agents. The disease started from extensor aspect of the extremities and was progressive in nature involving whole of upper and lower limbs and few lesions on the back and chest. History of seasonal variation with winter exacerbation was also observed. In the beginning there were periods of remission, later on the disease progressed and involved the small joint of both hands without remission. He consulted a dermatologist and got skin biopsy done and was diagnosed as a case of psoriasis. He was treated with pain killers, cetrizine, methotrexate, cyclosporine and topical steroids. He got some relief but not complete cure. He kept on changing doctors. Patient was non alcoholic and non smoker, with no family history of such disease.

On clinical examination he was afebrile with normal pulse, BP and respiration. Local examination revealed erythematous scaly lesions scattered all over the body. On scratching the lesions scales became prominent and on further scratching there was pinpoint bleeding. Hands showed flexion

deformities at interphalangial joints bilaterally. In the groins, there were firm lymphadenopathy and ulcers of 2.0X3.0 inches in size with undermined edges. These ulcers were covered with necrotic slough and purulent discharge. On removal of slough, unhealthy granulation tissue was seen. On glans penis there was a patch of erythema (circinate balanitis).

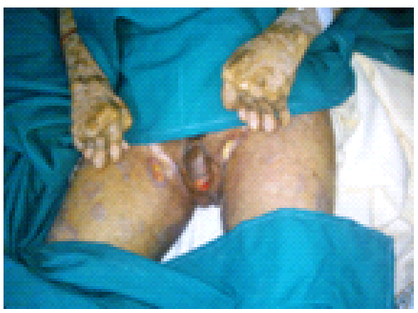

Figure 1. Flexion deformities and psoriatic lesions in both hands; multiple ulcers with undermined edges and purulent discharge in the groin area, a patch of erythema over glans penis (circinate balanitis)

Investigations showed hemoglobin 10gm%, leucocytosis (12500/cmm) with lemphocytosis (65%) and raised ESR (62mm/hr). Liver function tests (LFT) were slightly raised i.e. SGOT/PT (85/80). Smear was made from the groin ulcer and stained for AFB; it showed the presence of acid fast bacilli. Polymerase chain reaction (PCR) test was positive for mycobacterium tuberculosis. The history, clinical examination and investigations led us to arrive at a diagnosis of scrofuloderma in a patient of psoriatic arthritis.

DISCUSSION

Scrofuloderma, also called 'tuberculosis colliquativa cutis' is a common form of cutaneous tuberculosis affecting children and young adults in which there is breakdown of skin overlying a tuberculous focus in the lymph node, bone or joint.[8] Initially, there are firm painless, subcutaneous nodules that gradually enlarge and suppurate.[8] These lead to ulcers and sinus tracts with undermined edges and ultimately puckered scars.[8] Diagnosis is usually performed by needle aspiration cytology or excisional biopsy and the microbiological demonstration of stainable acid fast bacteria.[9] PCR has a low sensitivity but high specificity.[10] Differential diagnosis include mycobacterium scrofulaceum, M. avium-intracellulare, actinomycosis, sporotrichosis, botryomycosis, nocardiosis, syphilitic gumma and hidradenitis suppurativa.[7,11] In our case, swellings with draining sinuses, presence of acid fast bacilli on gram staining and positive result on PCR testing favoured the diagnosis of scrofuloderma.

Psoriasis could represent an independent risk factor for TB.[7,12] Up 70% of psoriatic patients require traditional systemic treatments, such as retinoids, methotrexate, and cyclosporine. Many of them import long-term toxicity, treatment resistance, and potential drug interactions, so only 25% of psoriatic patients are completely satisfied with their treatment.[13] Advanced drugs such as TNF-α inhibitors are associated with an increased risk of developing active disease in patients with latent tuberculosis infection (LTBI), because TNF-α is a key cytokine in protective host defense against Mycobacterium tuberculosis.[4,5] Our case also gave the history of treatment outside with some costly medication. This could be a TNF-α inhibitor, which might be responsible for flare up of latent tuberculosis. For this reason, exclusion of active TB and treatment of LTBI are, therefore, clinical imperatives prior to starting anti-TNF-α therapy and active surveillance for a history of untreated or partially treated TB or LTBI has already been shown to be effective in reducing the number of incident TB cases in psoriatic patients.

CONCLUSION

We conclude that severe psoriasis may be associated with an increased risk of developing

latent TB and patient taking systemic corticosteroids, non-steroidal anti-inflammatory drugs and newer drugs in the form of biological agents may be associated with scrofuloderma.

AUTHOR NOTE

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REFERENCES

1. Perera GK, Di Meglio P, Nestle FO. Psoriasis. *Annual Review of Pathology*. 2012;7:85-422.
2. Christophers E. Psoriasis—epidemiology and clinical spectrum. *Clinical and Experimental Dermatology.* 2001;26(4):314–20.
3. Vena GA, Altomare G, Ayala F, et al. Incidence of psoriasis and association with comorbidities in Italy: a 5-year observational study from a national primary care database. *European Journal of Dermatology.* 2010;20(5):593–8.
4. Lebwohl M, Bagel J, Gelfand JM, et al. From the Medical Board of the National Psoriasis Foundation: monitoring and vaccinations in patients treated with biologics for psoriasis. *Journal of the American Academy of Dermatology.* 2008; 59(2):209–17.
5. Kimball AB, Gladman D, Gelfand JM, et al. National Psoriasis Foundation clinical consensus on psoriasis comorbidities and recommendations for screening. *Journal of the American Academy of Dermatology.* 2008; 58(6):1031–42.
6. Bordignon V, Bultrini S, Prignano G, et al. High prevalence of latent tuberculosis infection in autoimmune disorders such as psoriasis and in chronic respiratory diseases, including lung cancer. *Journal of Biological Regulators and Homeostatic Agents.* 2011; 25(2):213–20.
7. Sánchez-Regaña M, Dilmé E, Puig L, et al. Adverse reactions during biological therapy for psoriasis: results of a survey of the Spanish Psoriasis Group. *Actas Dermo-Sifiliograficas.* 2010;101(2):156-163.
8. Tappeiner G. Tuberculosis and Infections with Atypical Mycobacteria. In: Wolff K, Goldsmith LA, Katz SI, et al., eds. Fitzpatrick's Dermatology in General Medicine, 7th edn. New York: McGraw-Hill; 2008:1768-78.
9. Meltzer SM, Nacy CA. Cutaneous Tuberculosis. Jan 28, 2009. Available from http://emedicine.medscape.com/artical/1105 317-overview.
10. Rajakumar D, Rosenberg AM. Mycobacterium tuberculosis monoarthritis in a child. *Pediatr Rheumatol.* 2008;6:2-10
11. Karabudak O, Dogan B, Harmanyeri Y. Long-lasting scrofuloderma of hands and foot. *Acta Derm Venereol.* 2007;87:91-2.
12. Bassukas ID, Kosmidou M, Gaitanis G, Tsiouri G, Tsianos AE. Patients with psoriasis are more likely to be treated for latent tuberculosis infection prior to biologics than patients with inflammatory bowel disease. *Acta Dermato-Venereologica.* 2011;91(4):444-6.
13. Stern RS, Nijsten T, Feldman SR, Margolis DJ, Rolstad T. Psoriasis is common, carries a substantial burden even when not extensive, and is associated with widespread treatment dissatisfaction. *Journal of Investigative Dermatology Symposium Proceedings.* 2004; 9(2):136-9.