CUTANEOUS TUBERCULOSIS

Dr.Jeyakumari Jeevan, *Professor.*
Dr.Srinivasan G, *Professor & Head,*

Department of Dermatology, Shri Sathya Sai Medical College & Research Institute,
Sri Balaji Vidyapeeth, Nellikuppam, Kancheepuram Dist., Tamil Nadu – 603 108, India.

Introduction

Cutaneous tuberculosis has been part of human history since pre-historic period and it has been a global health problem. Due to the HIV epidemic the emergence of resistance strains of M.tuberculosis, the rise in immunosuppressive therapy, the ease of migration of people, decline in TB control efforts, super-imposed on the pre-existing factors of poverty and malnutrition there was rise in the incidence of mycobacterial infection in developing countries. Better living standard and improved treatment methods have led to a decline in the incidence of mycobacterial infections in developed countries.[1]

The discovery of M-tuberculosis by Robert Koch in 1882 and advance in descriptive pathology during 19th century helped to establish cutaneous tuberculosis as a part of this infectious diseases.

Classification[2]

Exogenous sources: Tb chancre, warty Tb and lupus vulgaris.

Endogenous sources: contiguous spread-scrofuloderma, Auto inoculation-orificial Tb and haematogenous consisting of miliary Tb, lupus vulgaris and Tb gumma

Tuberculides: micro popular, lichen scrofulosorum, popular, Papulo Necrotic Tuberculide and Nodular and Erythema nodosum consisting of Erythema induratum (BAZIN)

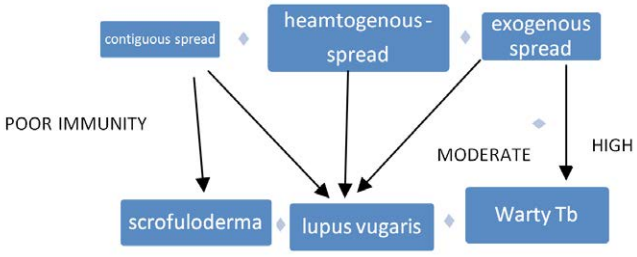

Fig 1: Spectrum Of Cutaneous Tuberculosis

Clinical Features[3]

Clinical variants dependent on the host's immunity and route of entry. The common presentations of cutaneous TB are lupus vulgaris, scrofulderma, TB verrucosa cutis and tuberculides

1 Lupus Vulgaris: (TB Luposa)

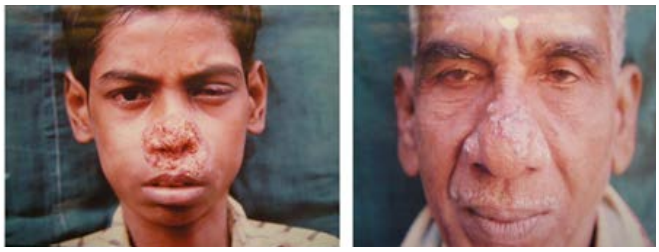

Fig 2: Lupus Vulgaris of the face

The incidence is 10-15%, 2-3 times more seen in females and may present as single or few lesions. well demarcated, annular or accurate plaques which slowly spread centrifugally. The periphery shows erythematous to brownish, deep seated nodules which on diascopy may stand out as apple jelly nodules. Centre becomes atrophic in course of time, de-pigmented and scarred. characteristically, new nodules appear within area of scarring.

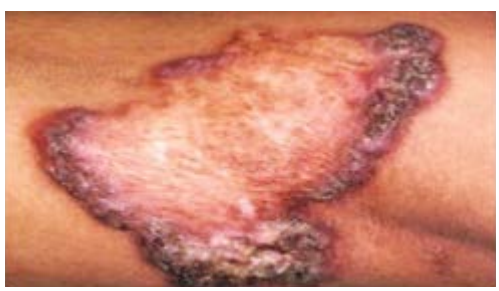

Fig 3: Lupus Vulgaris of the buttocks

Sites of predilection: Buttocks, upper extremities and face

Complications: Ulcerations, Hypertrophic lesions and squamous cell carcinoma, It needs to be differentiated from **Discoid Lupus Erythematous.**

2. Scrofuloderma: (TB cutis colliquativa)

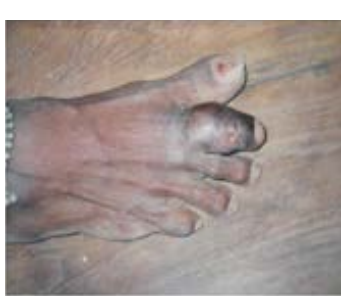

Fig 4: Scrofuloderma of the foot

Figure 5 shows cutaneous TB due to direct extension of the infection from an underlying TB present either in a lymph node (cervical less often, axillary and inguinal), and also seen in bone or a joint.It manifest as chronic sinuses with hyper pigmented undermined edges. Tuberculin test will be positive.

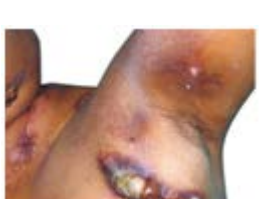

Fig 5: Cutaneous TB of the lymph node

3. Tuberculosis Verucosa Cutis:

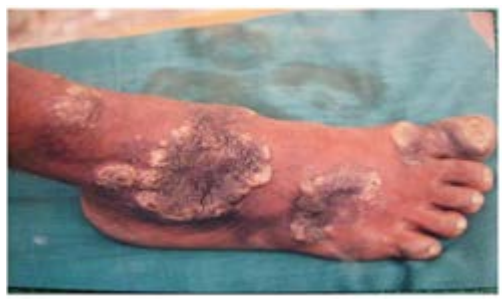

Fig 6: Tuberculosis Verucosa Cutis of the foot

Tuberculosis Verucosa Cutis is a common form of cutaneous TB, inoculated from outside into a skin of a individual with a high degree of immunity due to previous exposure. It is seen as a single indolent verrucous wart nodule with a serpiginous edge and an erythematous areola with indurate base centre may shows scarring. Often seen on trauma prone sites like hands and feet. Can be easily differentiated from verruca vulgaris

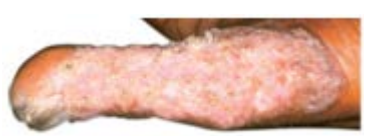

Fig 7: Tuberculosis Verucosa Cutis of the finger

4. Orificial TB

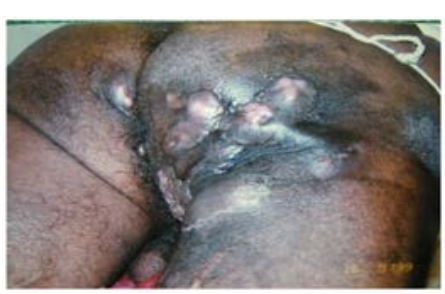

Fig 8 : Orificial TB

Orificial TB is a rare type. Auto inoculation of mucosa or skin adjacent to a natural orifice draining an active internal Tb infection.

5. Acute Millary TB: (TB Cutis Disseminata, TB Cutis Acuta Generalisata)

A rare, hematogenous dissemination from primary lung focus in patients with low immunity

6. TB Gumma: (Metastatic TB Ulcer)

TB Gumma is seen in children with low socio economic status and immune suppressive hosts. It is transmitted by acute heamtogenous dissemination from primary focus,

Investigations [4]

To confirm Diagnosis of TB we need to do Biopsy.

ISOLATION OF M.TUBERCULOSIS: AFB in pus and culture of TB may be possible, but only from some lesions<10%.We can also do PCR test

Treatment [5]

S.NO	PHASE	DURATION	DRUG	DOSAGE
1.	Intensive Phase (to achieve rapid bacterial killing)	2 months	Isoniazid Rifampicin Ethambutol Pyrazinamide	5mg/kg 10mg/kg 15mg/kg 30mg/kg
2.	Maintenance Phase (kill persistent bacteria)	4 months	Isoniazid Rifampicin	5mg/kg 10mg/kg

REFERENCES

1. Bravo FG, Gotuzzo E. Cutaneous tuberculosis. Clin Dermatol. 2007;25(2):173–180.
2. Fariña MC, Gegundez MI, Piqué E, et al. Cutaneous tuberculosis: a clinical, histopathologic, and bacteriologic study. J Am Acad Dermatol. 1995;33(3):433–440.
3. MacGregor RR. Cutaneous tuberculosis. Clin Dermatol. 1995;13(3):245–255.
4. Barbagallo J, Tager P, Ingleton R, et al. Cutaneous tuberculosis: diagnosis and treatment. Am J Clin Dermatol. 2002;3(5):319–328.
5. Dinnes J, Deeks J, Kunst H, et al. A systematic review of rapid diagnostic tests for the detection of tuberculosis infection. Health Technol Assess. 2007;11(3):1–196.