CASE REPORT

Coinfection of leprosy and tuberculosis

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SUMMARY

Leprosy and tuberculosis (TB) are endemic to India, however, their coinfection is not frequently encountered in clinical practice. Here, we report a 32-year-old female patient who presented with a history of high-grade intermittent fever, cough and painless skin lesions since a month, along with bilateral claw hand (on examination). The haematological profile was suggestive of anaemia of chronic disease, chest radiograph showed consolidation, sputum smears were positive for *Mycobacterium tuberculosis*, and skin slit smear confirmed leprosy. The patient was prescribed WHO recommended multidrug therapy for multibacillary leprosy with three drugs. Additionally, prednisolone was added to her regimen for 2 weeks to treat the type 2 lepra reaction. For treatment of TB, she was placed on the standard 6-month short course chemotherapy. She was lost to follow-up, and attempts were made to contact her. Later, it came to our notice that she had discontinued medications and passed away 3 months after diagnosis.

BACKGROUND

Leprosy and tuberculosis (TB) are both endemic to India. Taking into account the population and size of the country, the burden of both these diseases is very high in India. The prevalence of leprosy in India is 0.68/10 000 and the prevalence of TB is estimated to be 211/100 000 population for the year 2013.^{1,2} India alone accounted for 6 burden in 2015³ and contributes 23% to the global incident TB cases accounting to 21% of global TB-related mortality.²

TB is known to occur throughout the spectrum of leprosy^{4,5} with some studies, in fact, suggesting an increased susceptibility to TB in patients with leprosy and a higher morbidity and mortality. Conversely, some studies suggest cross-immunity, wherein TB protects against leprosy.^{5,6}

TB may occur in patients with leprosy with predisposing pre-existing conditions such as malnutrition, diabetes or other immunosuppressed states and also in patients being treated with corticosteroids for the complications of leprosy. We report one case of leprosy and pulmonary TB in a treatment-naïve patient with no comorbidities, both conditions diagnosed at the same time.

CASE PRESENTATION

A 32-year-old woman presented with a history of high-grade, intermittent fever and cough with minimal sputum production for the duration of 1 month. She also complained of painless nodular

skin lesions (which started out as one skin lesion a month ago and then progressed to multiple lesions on her extremities). Some of these skin lesions resolved spontaneously, some persisted and new lesions continued developing. Patient also reported she had anorexia and weight loss. Her family history, medical history and personal history were unremarkable. A history of (bacille Calmette-Guerin)BCG vaccination could not be ascertained as the patient was unaware of her vaccination status. None of her family members had suffered from TB or leprosy. On further questioning, the patient also described some skin lesions, which she had for a longer duration. The patient was unable to give the exact duration of onset of these skin lesions, however, in view of her claw hand, we assume that the leprosy infection possibly predated pulmonary TB.

On examination, she was moderately built and nourished with a body mass index of 20. She was febrile and pale. There was no significant lymphadenopathy. Cutaneous examination revealed multiple, non-tender and anaesthetic skin nodules on her arms and legs, and multiple, erythematous, indurated plaques on the thighs (figure 1). The patient also had thickened right and left ulnar nerves and clawing of fourth and fifth digits in both hands (figure 2). However, our patient did not ascribe any importance to the bilateral claw hand and was unable to tell us the duration of the deformity. There was no loss of sensation noted in the hands or feet. Chest examination revealed signs of a left lower lobe pneumonia, which was confirmed by her chest X-ray (figure 3).

Figure 1 Cutaneous examination showing multiple non-tender and anaesthetic skin nodules in the arms and legs, multiple, erythematous, indurated plaques in the thighs.

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Figure 2 Showing thickened right and left ulnar nerve and clawing of fourth and fifth digits in both hands.

INVESTIGATIONS

Initial blood investigations revealed: Hb: 8 gm%; white cell count: $9^9/L$; erythrocyte sedimentation rate: 92 mm/hour. Patient's blood sugars, glycosylated haemoglobin, renal function tests and liver function tests were normal. Peripheral smear showed normocytic normochromic red blood cells and was suggestive of anaemia of chronic disease. Serum iron profile was normal, and HIV ELISA was negative. Chest X-ray and ultrasound chest showed left lower lobe consolidation with minimal pleural effusion (figure 3), which confirmed pneumonia.

With a provisional diagnosis of community-acquired pneumonia, sputum Gram's stain and culture were performed. Also, in view of the prolonged history of a cough and fever of more than 3 weeks' duration, pulmonary TB was suspected. While

Figure 3 Chest X-ray showing left lower lobe consolidation with minimal pleural effusion.

Figure 4 Skin biopsy showing $\times 40$ nodular infiltrate of macrophages and neutrophil infiltration.

two samples of sputum were acid-fast bacilli (AFB) negative, the third sample was 3+ AFB. Sputum PCR was positive for *Mycobacterium tuberculosis*; sputum BACTEC culture for AFB was positive for *M. tuberculosis*.

A dermatologist opinion was obtained for the multiple, anaesthetic skin lesions which numbered more than five, associated with thickened nerves which were highly suggestive of Hansen's disease. Slit skin smear stained with Ziehl-Nielsen stain was positive for AFB (3+Bacteriologic Index and 1+Morphologic Index) and confirmed the diagnosis of multi-bacillary (MB) leprosy.

A full dermal thickness skin biopsy from the active edge of a nodule was undertaken. It was stained with H&E and Fite-Faraco stains and showed skin with the normal epidermis. The dermis showed aggregates of foamy macrophages, few lymphocytes and plasma cells. The subcutaneous fat showed dense infiltrates of neutrophils, surrounded by histiocytes, large collections of foamy macrophages and fibroblasts. No AFB were detected (figures 4 and 5). These histological findings along with the clinical history of the recent increase in skin lesions were suggestive of erythema nodosum leprosum (ENL).

Figure 5 Skin biopsy showing high magnification $\times 150$ from the edge of the nodular infiltration.

DIFFERENTIAL DIAGNOSIS

Since leprosy and TB are both endemic to India, a provisional diagnosis of the coinfection was made, and investigations were undertaken as per protocol. Owing to the typical anaesthetic skin lesions, numbering more than five, along with bilateral ulnar nerve thickening, a clinical diagnosis of MB leprosy was made. In consultation with the dermatologist, a slit skin smear and a skin biopsy from the active edge of a lesion were sent for histopathology. The final diagnosis of sputum-positive pulmonary TB with Hansen's disease (borderline lepromatous to lepromatous leprosy) with ENL and ulnar claw hand was made.

In non-endemic countries, however, leprosy can remain undiagnosed, as it is not encountered in practice nor curriculum. The differential diagnosis for leprosy is elucidated below⁷:

1. non-infectious, non-granulomatous dermatitic conditions such as non-scarring alopecias, vitiligo, contact dermatitis, nummular eczema, psoriasis, Reiter's syndrome, lichenoid dermatitides (lichen planus, drug eruptions) and gyrate erythema;
2. Superficial non-granulomatous skin infections such as tinea corporis ('ringworm'), tinea versicolor, impetigo circinatum and erythema migrans (*Lyme borreliosis*);
3. Non-infectious plaque and nodular diseases (with or without systemic involvement) such as sarcoidosis, palisading, histiocytic diseases, mastocytosis, granuloma faciale, appendageal hamartomas, Kaposi's sarcoma, drug reactions, neurofibromatosis 1 and lymphoproliferative disorders;
4. Skin lesions such as dermatofibromas or nodular-like keloids, TB, 'lepromatoid' atypical mycobacterial infection (eg, *Mycobacterium chelonae*), leishmaniasis skin infections;
5. Systemic disorders with diffuse cutaneous infiltration such as systemic sclerosis, scleromyxedema, amyloidosis and mycosis fungoides.

TREATMENT

For TB

The patient was put on the standard 6 months short course chemotherapy for sputum-positive pulmonary TB. This regimen included a 2-month intensive phase of four drugs daily, that is, isoniazid, rifampicin, pyrazinamide and ethambutol to be followed by 4 months of continuation phase with two drugs, that is, isoniazid and rifampicin. This was in accordance with the WHO and Revised National Tuberculosis Control Programme guidelines for India.⁸

For leprosy

The WHO prescribed 1-year multidrug therapy (MDT) for multibacillary leprosy⁹ with three drugs (dapsone, clofazimine and rifampicin) was initiated for this patient. Dapsone (10 mg/kg body weight) and clofazimine (1 mg/kg daily plus 6 mg/kg once a month) were initiated, whereas monthly rifampicin was skipped since it was already a part of the antitubercular regimen the patient was prescribed. For the type 2 lepra reaction, she was treated with steroids, that is, prednisolone (1 mg/kg body weight) for 2 weeks, with a plan to taper on review.

For ulnar claw

The prednisolone prescribed¹⁰ was expected to prevent further degenerative changes and deterioration of the ulnar claw. Physical and occupational therapy was advised. Since the patient did not have any specific complaints due to the ulnar claw, no other treatment modalities were considered.

OUTCOME AND FOLLOW-UP

The patient was advised to come for a follow-up visit after 2 weeks but she did not return and was unfortunately lost to follow-up. Our efforts to contact her family yielded information that she had discontinued the prescribed medications, for no apparent reason, within a few weeks of being diagnosed. The patient is deceased and passed away around 3 months after her diagnosis and discharge.

DISCUSSION

Leprosy, also known as Hansen's disease, is not a very common disease in most parts of the world. Only 14 countries in the world actively report leprosy: Bangladesh, Brazil, Democratic Republic of Congo, Tanzania, Ethiopia, India, Indonesia, Madagascar, Mozambique, Myanmar, Nepal, Nigeria, Philippines, Sri Lanka and Tanzania.³ Because of this, clinicians in other parts of the world are often not up-to-date with the diagnosis and management of the disease.

Leprosy presents in a spectrum of clinical manifestations that have been classified by Ridley and Jopling into five categories¹¹:

1. Polar tuberculoid leprosy (in which there is a high degree of cell-mediated *Mycobacterium leprae*-specific TH1-type immunity);
2. Borderline tuberculoid;
3. Borderline borderline;
4. Borderline lepromatous (BL);
5. Polar lepromatous leprosy (LL; in which there is little or no cell-mediated TH1-type immune response).

Progression from the tuberculoid end to the lepromatous end of the spectrum is associated with an increasing bacillary load, more frequent skin and nerve lesions and progressive reduction in *M. leprae*-specific cell-mediated immunity.

For clinical operational purposes, WHO on the other hand, classifies leprosy¹² as paucibacillary leprosy (PB) and MB. This is done by the number of skin lesions and bacilli seen on skin smear. PB is characterised by five or fewer skin lesions and no organisms on skin smear, whereas the multibacillary form shows six or more lesions and/or visualisation of bacilli on skin smear. It must be remembered that patients with leprosy seek medical attention mainly for the dermatological manifestations, and careful skin examination is the key to a successful and timely diagnosis.¹³

WHO recommends that any one of the following cardinal signs is diagnostic of leprosy¹⁴:

1. Hypopigmented or reddish patches with definite sensory loss;
2. Thickened peripheral nerves;
3. Positive skin smears or biopsy material for acid-fast bacilli.

Our patient had multiple anaesthetic skin lesions numbering more than five and her slit skin smear was positive for AFB with BI=3+ suggesting she had MB leprosy.

The immune response in an individual to *M. leprae* can fluctuate leading to changes in the clinical state which are termed as leprosy reactions. These reactions may be medical emergencies and are characterised by inflammatory episodes of the skin, nerves and other organs and can result in irreversible nerve damage. They can occur anytime during the course of leprosy but are found to occur most often after starting treatment. There are two main types of reactions: type 1 or reversal reaction and type 2 or ENL. Type 1 leprosy reactions occur in a third of patients with borderline forms of disease and are caused by a spontaneous increase in cell-mediated immunity to *M. leprae*. Starting MDT is considered the most important risk factor. Type

Table 1 Comparison of published reports of TB and leprosy coinfection

Author and year	No. of cases	Age/Sex of patient	Symptoms	First infection	Gap between both infections	Type of leprosy (Jopling's)	Type of TB	Predisposing factors	Outcome
Gajwani <i>et al</i> ¹ 1968	3	60 M	Fever, cough, haemoptysis	TB	6 months, diagnosed simultaneously	BT	Pulmonary	Malnutrition	Unknown
		30 M	Fever, cough	Leprosy	2 years, diagnosed simultaneously	TT			
		60 M	Cough, expectoration	TB	2 years, diagnosed simultaneously	BT			
Gupta and Prasad ² 1971	2	50 M	Asymptomatic	Leprosy	1 year, diagnosed simultaneously	TT	Pulmonary	Diabetes, CAD	Lepra reaction, Better
		25 F	Fever, cough, expectoration	Leprosy	6 months, diagnosed simultaneously			Euthyroid, nodular goitre	Better
Agnihotri <i>et al</i> ³ 1973	3	65 M	Fever, emaciation	TB	1 year, diagnosed simultaneously	TT	Pulmonary	Malnutrition	Better
		18 M	Cough, expectoration, haemoptysis	TB	4 years, relapse simultaneously diagnosed			None	
		30 F	Cough	Leprosy	1 month				
Bhargava and Mathur ⁴ 1976	4	39 M	Fever, cough	Leprosy	3 years	LL	Pulmonary	None	Unknown
		50 M	Cough, weakness		1 year				
		45 M	Cough, expectoration		4 years			Sputum smear	
		35 M	Cough, expectoration		15 years			None	
Premnath and Ramu ⁵ 1976	40 cases in 2 years	21 to 64 years (median age: 27)	Cough, expectoration (87.5%), fever (57.5%), and weight loss (35%)	Leprosy	1 to 25 years (individual data unavailable)	LL (72.5%); BL (27.5%)	Pulmonary	Malnutrition	Died (30%), left against medical advice (20%), improved (50%)
Ganapathi <i>et al</i> ⁶ 1976	1	30 M	Unknown	Leprosy	Unknown	LL	Cutaneous (lupus vulgaris)	None	Unknown
Vachharajani <i>et al</i> ⁷ 1977	4	50 M	Hypopigmented anaesthetic patches	TB	4 months	TT	Pulmonary	None	Better
		26 M	Single hypopigmented anaesthetic patch		4 months				
		30 M	Macular rash		2 months	LL			
		29 M	Multiple patches		1.5 months	TT			
Nigam <i>et al</i> ⁸ 1979	20	16 to 58 years (mean age: 28.4) (15 M; 5 F)	Cough, expectoration (100%), fever (80%), weight loss (60%), haemoptysis (25%)	Leprosy	10 to 15 years	LL (15); BL (3); TT (2)	Pulmonary, pleural effusion (2)	Malnutrition	Died (4), left against medical advice (5), Better (11)
Kaur <i>et al</i> ⁹ 1979	2	Unknown	Unknown	Leprosy	4 years	LL	Pulmonary	Malnutrition	Unknown
Gatner <i>et al</i> ¹⁰ 1980	15 active; 8 healed	Unknown	Unknown	Leprosy	Unknown	LL (4); BL (3); BB (1); BT (7)	Pulmonary	Malnutrition	Improved (10)
Kumar <i>et al</i> ¹¹ 1982	9	Unknown	Unknown	Leprosy	Unknown	LL (4); BL (3); TT (2)	Pulmonary	Unknown	Unknown
Singh <i>et al</i> ¹² 1987	25	Unknown	Unknown	Leprosy	Unknown	Individual data unavailable	Pulmonary	Unknown	Unknown
Saha and Rao ¹³ 1989	18	15 to 65 years (15 M; 3 F)	Unknown	Leprosy	Unknown	Individual data unavailable	Pulmonary	Malnutrition	Unknown
Palki <i>et al</i> ¹⁴ 1990	1	35 F	Swelling	Leprosy	5 years	BL	Multicentric lupus vulgaris	None	Better
Pinto <i>et al</i> ¹⁵ 1991	1	36 M	Warty lesion	Simultaneous occurrence		BT	Cutaneous	None	Jaundice
Inamdar and Sampagavi ¹⁶ 1994	1	23 M	Patch, ulcer with discharge	Simultaneous occurrence		TT	Cutaneous, and Pulmonary	None	Type 1 reaction, better
Arora and Johr ¹⁷ 1994	1	40 M	Patch, sinus	Simultaneous reoccurrence due to HIV		BL	Lymph nodal	HIV	Better
Agarwal <i>et al</i> ¹⁸ 2000	1	40 M	Fever, cough, anaesthetic patch	Simultaneous occurrence		LL	Pulmonary	CKD, transplantation, immunosuppression	Reaction, resolved
Srilakshmi <i>et al</i> ¹⁹ 2003	1	32 M	Fever, cough	Leprosy	10 years	LL	Pulmonary	None	Dead
Lee <i>et al</i> ²⁰ 2003	1	62 M	Cough, expectoration	TB	6 months	BL	Pulmonary	None	Type 1 reversal reaction, better
Agarwal and Sharma ²¹ 2007	1	36 F	Fever, weight loss	Simultaneous occurrence		BL	Pulmonary	Rheumatoid arthritis, methotrexate, steroids leflunomide	ENL, better
Sreeramareddy <i>et al</i> ²² 2007	2	65 M	Cough, expectoration, chest pain	Leprosy	3 months	BL	Pulmonary, Pleural effusion	Steroid therapy	Better
		65 M	Fever, cough		2 years	LL	Pulmonary	Prednisolone, thalidomide	
Prasad <i>et al</i> ²³ 2010	1	34 M	Breathlessness, loss of appetite, cough, expectoration, skin lesions over face and arms	Leprosy	11 months	BL	Pulmonary	Steroid therapy	Better

Continued

Table 1 Continued

Author and year	No. of cases	Age/Sex of patient	Symptoms	First infection	Gap between both infections	Type of leprosy (Jopling's)	Type of TB	Predisposing factors	Outcome
Rajagopal <i>et al</i> ⁵ 2012	1	55 M	Swelling and purulent discharge from foot	Simultaneous occurrence		TT	Extrapulmonary	Diabetes mellitus	Cured
Trindade <i>et al</i> ²⁵ 2013	2	31 M	Paraesthetic skin lesions	TB	6 months	BB-BT	Pleural	None	Better
		46 F	Red macules on face and feet	Leprosy	1 month	BT-BB	Pulmonary	Steroid therapy	

BL, borderline lepromatous; BT, borderline tuberculoid; CAD, coronary artery disease; CKD, chronic kidney disease; ENL, erythema nodosum leprosum; LL, lepromatous leprosy; TB, tuberculosis; TT, polar tuberculoid.

2 reactions may occur among patients with MB leprosy (BL and LL) and the higher the bacterial load the greater the risk of type 2 reaction.^{11 14}

Our patient had a recent increase in the number of skin lesions and the skin biopsy was suggestive ENL at the time of initial diagnosis of leprosy and prior to starting therapy. The prevalence rates of ENL are highly variable with average incidences in field-based populations for all leprosy cases (1.2%) and for MB leprosy cases (4.5%) and 37% in a hospital samples of patients with MB leprosy.¹⁵ Literature shows that ENL may occur before, during or after MDT and even several years later. However, one systematic review of ENL cases noted that the incidence of ENL is at least twice as high during MDT than at the time of the initial diagnosis, with the highest incidence in the first year of MDT.¹⁵ One study¹⁶ has noted leprosy reactions with concurrent TB infection; however, precipitating factors could not be elicited.

The exact relationship between TB and leprosy still remains unresolved. Both are chronic granulomatous diseases caused by mycobacteria which are AFB, and the mode of transmission for both is mainly by the aerosol route. However, leprosy caused by *M. leprae*, principally affects the skin and peripheral nervous system, whereas TB caused by *M. tuberculosis* can be pulmonary or extrapulmonary, affecting varied organs.

Conflicting theories have been proposed for the rare reports of leprosy and TB coinfection, with some researchers proposing that the two diseases are antagonistic with a relative protection against dual infection by cross-immunity.¹⁷⁻¹⁹ This argument is buttressed by evidence that BCG vaccine gives some protection against leprosy.^{20 21} However, the cross-immunity hypothesis has been countered by a coinfection hypothesis,⁶ suggesting that dual mycobacterial infection is not infrequent but in fact, persons with leprosy, especially MB leprosy are more susceptible to TB due to a reduced immune system response, which leads to increased mortality from the disease. Retrospective studies and archaeological evidence indicate a high incidence of the dual infection in the past.

A high mortality with dual infection was confirmed in a recent case review of 156 patients which noted a mortality of 37.2%.⁵ This study also observed that most of the cases of TB were associated with LL (52.5%) followed by BL leprosy (20.5%), while the association of tuberculoid form of leprosy with tuberculosis was uncommon. The time interval between the development of leprosy and TB varies between 2 months and 15 years in one case review²² and between 1 month and 25 years in another.⁵ Our patient had pulmonary TB which is the most common presentation of TB in leprosy, but cases of extrapulmonary TB in the form of cutaneous, lymph nodal, laryngeal have also been reported.^{5 23}

Pre-existing comorbid conditions like malnutrition, diabetes mellitus, chronic kidney disease or treatment with corticosteroids have been noted in leprosy patients developing tuberculosis;⁵ however, our patient had no premorbid illnesses and was not on any immunosuppressant therapy.

As literature states^{23 24} that the sputum of patients of leprosy (mainly lepromatous leprosy) may stain positive for AFB giving diagnostic dilemma with sputum-positive pulmonary TB, we confirmed our diagnosis with sputum culture for *M. tuberculosis* and TB PCR, both of which came positive. Table 1 compares the other cases of dual infections reported in the literature.^{4 5 22 25}

Our patient was treated simultaneously for both TB and leprosy. In the Indian scenario, the treatment of leprosy is according to guidelines from WHO and India's National Leprosy Elimination Programme.^{9 26} As in TB, use of MDT is advocated in leprosy for greater efficacy and to prevent development of drug resistance.²⁷ The MDT regimens for leprosy have undergone several changes especially with regard to the duration of treatment. Currently, two finite duration MDT regimens using dapsone, clofazimine and rifampicin are commonly followed, that is, the WHO regimen⁹ followed almost worldwide and the Public Health Service regimen followed in the USA.²⁸ These are described in table 2.

The WHO advocates a shorter duration of therapy, with only monthly doses of rifampicin to maintain cost-effectiveness and better implementation in the developing world, with a primary aim of interrupting transmission of disease.²⁹ The advantages of such a shortened duration of treatment, however, need to be balanced against the risk of relapse. A higher relapse rate among MB cases and the demonstration of persistent active lesions in patients with PB many years after completion of therapy have caused some authorities to advocate more intensive therapy in affordable patients.²⁹

Rifampicin, one of the frontline antitubercular drugs is also used in the treatment of leprosy. Rifampicin is a highly effective bactericidal agent against both *M. leprae* and *M. tuberculosis*. Recognition of TB in leprosy is important especially in the developed world where leprosy treatment regimens include daily rifampicin doses to prevent monotherapy with rifampicin and the emergence of rifampicin-resistant TB.

Table 2 Leprosy treatment regimens currently used worldwide

The WHO MDT regimen ⁹	PB: rifampicin 600 mg monthly plus dapsone 100 mg daily; six cycles in 9 months MB: rifampicin 600 mg plus clofazimine 300 mg monthly and dapsone 100 mg plus clofazimine 50 mg daily; 12 cycles in 18 months
US National Hansen's Disease Program treatment ²⁸	PB: dapsone 100 mg daily plus rifampicin 600 mg daily for 1 year. Follow-up every 6 months for 5 years MB: dapsone 100 mg daily plus rifampicin 600 mg daily plus clofazimine 50 mg daily for 2 years. Follow-up every 6 months for 10 years
Intensive regimens ²⁸	PB: dapsone 100 mg daily for 5 years MB: rifampicin 600 mg daily for 3 years plus dapsone (100 mg/day) indefinitely

MB, multibacillary leprosy; MDT, multidrug therapy; PB, paucibacillary leprosy.

Learning points

- Dual mycobacterial infections of leprosy and tuberculosis (TB) are not uncommon though infrequently reported. Delayed recognition of such cases of dual infections may result in poor outcomes if correct and timely treatment for both infections is not instituted.
- Sputum of patients with leprosy (mainly lepromatous leprosy) may stain positive for acid-fast bacilli creating a diagnostic dilemma with sputum-positive pulmonary TB.
- It is important that TB should not be overlooked in patients with leprosy to prevent inadvertent monotherapy of TB with rifampicin and emergence of rifampicin-resistant TB.

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