REACTIONS IN LEPROSY

Muhammad Zubair Khan, Sabina Saeed

Department of Dermatology,
Postgraduate Medical Institute, Lady Reading Hospital Peshawar.

ABSTRACT

Objective: To assess the frequency of reactions during leprosy treatment and their complications.

Material and Methods: Patients inducted in this study were referred from all over the province for hospitalisation in Leprosy Unit Lady Reading Hospital Peshawar from 1st June 2001 till 1st June 2004. All patients with clinical diagnosis of reaction in leprosy and confirmed by SLIT SKIN SMEAR for Acid Fast Bacilli (AFB), Voluntary Muscle Testing (VMT), Insensitive Points Testing (IP). Examination of skin lesions was recorded in detail.

Results: Out of 452 patients admitted in leprosy unit LRH during a study period of three years, 49 patients with leprosy reactions were included in the study. Type 1 upgrading reaction was found in 10 patients (20.41%) while type II Erythema Nodosum Leprosum (ENL) reaction was found in 39 patients (79.59%). Reactions were severe in 18 (36.7%)cases and moderate in 26 (53.1%) cases. All patients being treated for Leprosy were given chemotherapy for it.

Conclusion: It is concluded that different types of reactions do occur during treatment of leprosy and if not properly handled, can result in debilitating complications.

Key words: Reactions in leprosy, Complications, Erythema Nodosum Leprosum reaction.

INTRODUCTION

Leprosy is a chronic infectious disease of human beings caused by Mycobacterium Leprae, which affects skin, nerves and mucous membranes. The incubation period is not known with certainty. It ranges from 3 months to 40 years, the average being about 5 (+/-2) years.[1] For direct transmission a prolonged and close contact is usually considered. Leprosy bacilli are rarely found on the intact skin of patients with multi bacillary leprosy.[2] Leprosy bacilli may remain alive outside the human body in droplets of nasal secretion for several days.[3] Indirect transmission does not play an important role in the spread of leprosy. The peak age of onset is between 10-20 years. Males and females ratio is 2:1.[4]

During the chronic course of leprosy acute exacerbations (reactions) may occur in any type of leprosy, except early indeterminate form. These acute episodes are immunologically mediated and are associated with remarkable persistence of mycobacteria in tissue after bacillary death. All such immunological reactions fall into one or other of two etiological types.[5] Usually it occurs due to some predisposing factor, e.g. infections, mental or physical stress, puberty, pregnancy, parturition or surgical interventions.

Type II reaction is restricted to lepromatous end i.e., pure lepromatous leprosy (LL) & borderline lepromatous leprosy (BL) and is called erythema nodosum leprosum (ENL). It is due to humoral hypersensitivity.[6] It is not due to alteration in cell mediated immunity (CMI). It occurs later (e.g., 1 or 2 years) during the course of treatment, when skin lesion appears quiescent and most of the bacilli in the skin are granular. However, a patient may be in Type I and II reaction, when first seen. During reaction, inflamed skin lesions and nerves may be extremely painful and tender. Acute neuritis may cripple patients with borderline leprosy over night. In patients with borderline leprosy both type I and II reactions may occur simultaneously.

Type II E.N.L is graded as severe if ulcerated or if the nerves become painful or develops loss of nerve function. The leprosy skin lesions become more erythematous, oedematous and infiltrated with systemic illness.

The second type of reaction, which is particularly common in borderline lepromatous

AGE WISE DISTRIBUTION

Age in years	Frequency n = 49	%age
0-12	1	2.05%
13-20	5	10.20
20-30	7	14.28
30-40	10	20.40
40-50	11	22.44
50-60	10	20.40
Above 60	5	10.20

Table 1

(BL), pure borderline (BB) and borderline tuberculoid (BT) has many names. The W.H.O Expert Committee on leprosy in its 16th report used the term "*Reversal Reaction*". Many workers suggested *type I* or *upgrading reaction* in which only skin or nerves are involved.[7] Type I reaction can be normally diagnosed without difficulty on the clinical signs and symptoms, supported if necessary by histopathology and natural history of episodes, the underlying immunological mechanisms are being increasingly identified.[8]

Reactions if not treated can result in complications like scarring, facial paralysis, wrist drop, muscle weakness, anaesthesia, paresthesia etc.[9]

Our study was conducted to assess the frequency of reactions occurring patients under treatment for leprosy as well as the different complications encountered in non-treated patients with reactions.

MATERIAL AND METHODS

Patients inducted in this study were referred from all over the province for hospitalisation in Leprosy Unit Lady Reading Hospital Peshawar.

DISTRICT WISE DISTRIBUTION OF PATIENTS

DIR	15
BAJAUR	09
NOWSHERA	07
SWAT	06
SWABI	02
BUNER	01
CHITRAL	01
KOHAT	01
KHYBER AGENCY	01
MALAKAND AGENCY	01
KURRAM AGENCY	02
AFGHAN REFUGEES	01
MOHMAND AGENCY	02

Table 2

DISTRIBUTION ACCORDING TO SEVERITY OF REACTIONS

Severity of reaction	No.	%age
Severe	18	36.7
Moderate	26	53.1
Mild	05	10.2

Table 3

A detailed history of the patients experiencing any previous reactions (type, severity, frequency, and duration) was taken at the time of enrolment. A standard reaction Proforma was filled for each patient for each reaction episode during a fixed time frame (1st June 2001 to 1st June 2004). This proforma included history of fever, joint pains, epistaxis, inflammation of lesion, appearance of new lesions, pedal oedema, lymphadenopathy, epididymo-orchitis and neuritis, voluntary muscle testing (VMT) and insensitive point testing (IP).

Examinations of skin for inflamed lesions, ENL, nodules, desquamation, ulceration of nodules was looked for. Pain, tenderness of peripheral nerves, recent development of sensory, motor deficit/deformities was elicited. Slit skin smear for Acid Fast Bacilli (AFB) was taken in all cases. Grading of reaction was labelled as "mild" "moderate" and "severe". Treatment/management details during reactional episodes were recorded separately on the proforma. Mild reaction cases were managed with rest and NSAIDs. For moderate and severe cases along with the above oral prednisolone in a dose of 40-60 mg daily reducing by 5 mg every 2-4 weeks was given.

RESULTS

There are 5952 registered leprosy patients in NWFP and 32 leprosy clinics provide domiciliary treatment in the whole province. Average annual detection rate is 105 per year. Patients with specific complication are referred for

TYPES OF LEPROSY (CLASSIFICATION WAS DONE ACCORDING TO RIDLEY-JOPLING'S SYSTEM)

Type of Leprosy	Frequency	%age
NE	1	2.0
BT	8	16.3
BB	7	14.3
BL	11	22.5
LB	2	4.1
LL	20	40.8

Table 4

PRESENTING SYSMPTOMS (TYPE 1) REACTION

Presenting Symptom	Frequency (n = 10)	%age
Neuritis ulnar nerve only	01	10.0
Neuritis more nerves	03	30.0
Infiltration/inflammation of previous lesions	04	40.0
Appearance of new skin lesions	02	20.0

Table 5

PRESENTING SYMPTOMS (TYPE 11 REACTION)

Presenting Symptom	Frequency (n = 39)	%age
Nodular eruption with systemic illness	13	33.3
Appearance of new skin lesions	04	10.3
Nodular eruption with out systemic illness	05	12.8
Arthralgia (joint pains)	03	7.7
Epidedemo-orehitis	02	5.1
Uveitis	02	5.1
Peripheral neuritis with marked neural deficit	04	10.3
Peripheral neuritis with out marked neural deficit	06	15.4

Table 6

hospitalisation to leprosy unit LRH. From 1st June 2001 to 1st June 2004, total 452 patients were admitted in leprosy unit LRH. Out of these, 49 patients (10.8%), with reaction were included in study.

Out of these 49 cases, 43(87.76%) were male, 6(12.24%) females. Mean age was 40 years. Majority of patients (n=31/49) were ranging in age from 30-60 years while there was only one child 2.05%) below 14 years (Table 1). Seven (14.29%) cases were diagnosed as new leprosy cases with reaction, while 42(85.71%) cases got reaction during treatment. Majority of patients (n=43) were referred for hospitalisation and six cases self-reported. Area distribution is showed in table 2.

Out of 49 cases 10 (20.40%) cases were upgrading reaction (type 1) and 39(79.60%) were type II reaction (ENL). Severe disability occurred in 2(20%) out of 10 cases of upgrading reaction (table 3).

LL was the most common type of leprosy (40.8%) followed by BL (22.55) according to Ridley Jopling's system of classification (table 4).

Bacterial index (BI) was positive in 42(85.71%) cases while in 7(14.29%) cases was negative.

Presenting symptoms of type 1 and type11 reaction are given in tables 5 & 6 respectively.

Two cases had severe disabilities in form of wrist drop and facial paralysis (one case each). Neural deficit showed that enlargement of peripheral nerves was present in 14 (63.6%)cases and tenderness of peripheral nerves in 08 (36.4%)cases. Sensory testing showed increased area of insensitivity in 06 cases. Grading is showed in table 7.

Voluntary muscle test (VMT) of 40(81.63%) patients was normal (Grade-5) while 9(18.36%) patients showed decreased VMT (table 8).

GRADING: (42 CASES)

Grade	No.	%age
+ ve 1+	01	2.4
+ ve 2+	04	9.5
+ ve 3+	10	23.8
+ ve 4+	08	19.1
+ ve 5+	12	28.5
+ ve 6+	07	16.7

Table 7

DISTRIBUTION ACCORDING TO VOLUNTARY MUSCLE TEST (VMT)

Grade	Frequency (n = 49)	%age
Grade 0	02	4.1
Grade 1	00	0
Grade 2	00	0
Grade 3	05	10.2
Grade 4	02	4.1
Grade 5 (Normal)	40	81.6

Table 8

DISCUSSION

Leprosy also called Hansen's disease is a chronic infectious disease, dependent in large upon the individual's immunologic response to mycobacterium leprae.[1] The spectrum of illness ranges from tuberculoid (or paucibacillary) disease characterized by relatively few (no more than 5) skin lesions and a marked cell mediated immune response to M. Leprae, to lepromatous (or multibacillary) disease, which exhibits multiple skin lesions and little cell mediated immunity to M. laprae.[3] Patients can also present with acute inflammatory responses to M. Leprae, called Leprosy reactions.[4] They can result in marked nerve damage, facial paralysis, eye complications, muscle weakness and subsequent limb disability and deformity.

The two common types of Leprosy reactions are type I or reversal reaction, mediated by an upgrade in cellular immune responses to the bacterium, and type II or erythema nodosum leprosum (ENL), which corresponds to a systemic response to immune complex deposition caused by dead or dying M. laprae.[2,5] Type I reaction occurs in borderline disease and are characterized by acute neuritis and/or acutely inflamed skin lesions.[5] Existing skin lesions become erythematous or oedematous or may desquamate or ulcerate. Occasionally edema of the face, hands or feet is the presenting symptom.[6] Although type I reaction can start spontaneously, the commonest time is after starting treatment and during the puerperium.[8]

A type I reaction implies a change in cell mediated immunity and often a corresponding shift of borderline leprosy towards the tuberculoid pole.[9] These reverse reactions typically occur after initiation of leprosy treatment but may occur spontaneously before therapy. Reversal reactions may be dangerous, in that the nerve damage can be asymptomatic and may progress "silently" for prolonged periods. Painless nerve damage has been glorified as silent neuropathy. It is very likely that those nerves have whispered before destruction, which patients were not able to hear due to faintness of sound or their preoccupation with other things.[9,11] In addition to treatment other precipitants of type I reactions include puberty, pregnancy and parturition.

Type II (ENL) reactions occur in patients with multibacillary (LL and BL) disease. They can occur spontaneously (Roseolar Leprosy) or whilst on treatment. Attacks may be acute at first, but may be prolonged or recurrent over several years and eventually quiet but insidious, especially in eye.[10] ENL characterized by humoral hypersensitivity, occurs in patients with lepromatous leprosy and often presents with crops of tender, subcutaneous nodules, fever, arthralgia, neuralgia and occasionally vasculitis, adenopathy, orchitis and dactylitis.[11] Acute lesions crop and desquamate fading over several days. Peripheral nerve neuritis and uveitis with complications can occur. ENL can be triggered by treatment vaccination, tuberculin skin testing and other stimulants of immune system.[12]

These two reaction can lead to impaired sensory, autonomic and motor nerves. Anaesthetic limbs are subject to repeated trauma, pressure necrosis and secondary infection, all of which culminate in the loss of digits and limb deformity, that are classically associated with leprosy. Autonomic disruption leads to dry skin that easily fissures and ulcerates, and loss of protective corneal reflex.. Common motor findings in the late disease include clawing of hand; wrist drop and foot drop due to destruction of ulnar, radial, cutaneous and common peroneal nerves respectively.[13]

So the reactions should be viewed as medical emergencies. Although few physicians have expertise in leprosy, prompt recognition and treatment are essential to limit morbidity and loss of quality of life.

CONCLUSION

It is concluded that during the treatment of leprosy reactions do occur. These reactions if not identified in time can result in debilitating complications like permanent nerve damage, resulting in wrist drop, facial paralysis, uveitis, arthralgias etc. if they are detected earlier, all these complications can be avoided

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Address for Correspondence:

M. Zubair Khan,
Department of Dermatology
Postgraduate Medical Institute,
Lady Reading Hospital Peshawar