Clinical Profile of Type II Reaction in Leprosy A Cross Sectional Study

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Leprosy is a chronic granulomatous disease that primarily affects the peripheral nervous system. Cutaneous nerves are severely affected in lepra reaction and this leads to morbidity. The objective of this study was to analyse the clinical profile of Type-II reactions in leprosy. The present cross-sectional study was undertaken in 21 leprosy patients with Type-II reactions attending in and out-patient department of Dermatology & Venereology, B.R.D. Medical College, Gorakhpur, Uttar Pradesh, India from July 2005 to October 2006. Type-II reaction was more common in male. Erythema nodosum leprosum (ENL) was the presenting feature associated with high grade fever. Initiation of multidrug therapy was main precipitating factor for development of Type-II reaction in leprosy which was generally seen within first six month. Our study carried out during MDT era shows that profile of symptomatology as well as triggers for initiating reactions does not seem to have changed. As there could be variation in the course of these reactions due to changes in bacillary load of cases diagnosed and Clofazimine being part of regular Multibacillary regimen after MDT, periodic analysis of statistically significant number of such cases could be meaningful from therapeutic angle.

 $\textbf{Key Words:} \ Leprosy, Erythema\ Nodosum\ Leprosum, Neuritis, MDT\ erack and the sum of the sum$

Introduction

Leprosy is one of the oldest diseases of mankind. The first authentic description of leprosy is given in Sushruta Samhita written in India in 600 B.C. The chronic and placid course of leprosy is punctuated by episodes termed as "reactions" (Dharmendra 1978). The clinical diagnosis and treatment of these reactions are of immense importance as these determine the final functional outcome, especially with reference to the

nerves (Dharmendra 1978, Pfaltzgraff and Bryceson 1979, Sehgal 1979, Job 1989). Introduction of highly bactericidal multi-drug treatment (MDT), changing spectrum of disease with different bacterial load and Clofazimine being part of regular Multibacillary regimen after MDT are among the factors that can have effect on course and profile of such reactions. This study has been carried out to clinically evaluate the pattern of Type-II reaction in leprosy patients

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192 Gupta et al

treated with MDT at a Tertiary care Centre of Medical College in eastern Uttar Pradesh.

Materials and Methods

The present study was undertaken on 21 Leprosy patients. All the patients who reported with type II reaction to the Department of Dermatology & Venereology, B.R.D. Medical College, Gorakhpur from July 2005 to October 2006 were included. Electrophysiological studies were conducted in these cases.

Clinical History - A detailed history of all patients was taken with special reference to age, sex, marital status, occupation and place of residence. A complete clinical history for the diagnosis of leprosy and reactions was taken and recorded in proforma emphasizing the age of onset of leprosy, duration of disease, site of first skin lesion, progress of disease, symptoms suggestive of reactions like exacerbations of existing lesions, appearance of new lesions, appearance of evanescent 'rose-spot' nodules, nerve pain, fever and malaise and precipitating factors like concurrent infection, change of seasons, pregnancy and childbirth, stress, age at onset of reaction, duration of reaction and history of specific treatment being taken. Past history of reactions and specific treatment for leprosy and reactions were also recorded. Relevant history regarding tuberculosis, diabetes mellitus, and concurrent infections was obtained. Personal history including dietary habits, smoking and alcohol intake was recorded. A detailed history suggestive of leprosy in family members was taken.

Clinical Examination - A thorough clinical examination was done in good day light, giving special attention to nerves and cutaneous examination. Skin lesions were examined for characteristics of Hansen's disease as number, shape, size, surface, color and sites. Peripheral sensations as well as sensations over skin lesions were checked for temperature, pin-prick and touch recorded in the proforma. All superficial peripheral nerves were examined for thickening, tenderness and nerve abscess. Any muscle weakness, deformity, or presence of ulcer was noted. Diagnosis of leprosy was confirmed by slit skin smear examination and skin biopsy in suspected cases.

Results

In present study, more than half of patients (61.69%) were between the age group of 21-40 years. Below 21 years age group 4 (19.05%) patients were recorded (Table 1).

Table 1 shows the age group and sex distribution of Type-II reaction cases of leprosy included in this

Age (years)	Male	Female		Total
			No.	%
0-10				
11-20	04		04	19.05
21-30	05	01	06	28.58
31-40	06	01	07	33.33
41-50		01	01	04.76
51-60		01	01	04.76
61-70	02		02	09.52
Total	17	04	21	100.00

Table 1: Age and sex distribution

study. It was observed that more than half of the cases 13 (61.91%) were affected between the age group of 21-40 years. We found 4 (19.05%) cases were below the age group of 21 years. Minimum age recorded was 12 years (1 case) and maximum 67 years (only 1 case). Male: female ratio was 17:4 (4.25:1) in these cases.

Erythema Nodosum Leprosum were present in all patients (100%), exacerbation of existing

lesions was observed in 11 (52.38%) patients, appearance of new lesions in 7 (33.3%) patients and neuritis in 5 (23.80%) patients respectively (Table 2).

As shown in Table 3 constitutional symptoms such as fever were present in all (100%) cases followed by swelling and pain in joints in 18 (85.71%) cases, neuralgia in 16 (76.19%) cases, body ache in 12 (57.14%) cases, edema of hands and feet in 11

Table 2 : Clinical manifestation suggestive of Type-II reaction in leprosy

Lesions	No. of patients	Percentage (%)
Exacerbation of existing lesions	11	52.38
Appearance of new lesions	07	33.33
ENL	21	100.0
Neuritis	05	23.80

Table 3: Constitutional symptoms present in Type-II reaction in the leprosy cases studied

Constitutional symptoms	No. of patients	Percentage (%)
Fever	21	100.00
Swelling and pain in joints	18	85.71
Neuralgia	16	76.19
Body ache	12	57.14
Edema of hands and feet	11	52.38
Lymphadenopathy	09	42.85
Eye involvement	08	38.09
Epididymo-orchitis	05	23.80
Epistaxis	03	14.28
Gvnaecomastia	02	09.52

Table 4: Precipitating factors for Type-II reaction in leprosy

Precipitating factors	No. of patients	Percentage (%)
Anti-leprosy drugs	20	95.23
Concurrent infection		
Change of seasons		
Pregnancy and childbirth		
Devitalizing disease		
Stress and Strain		
Unknown	01	04.70
Total	21	100.0

194 Gupta et al

Table 5 : Time interval from initial symptoms of Leprosy seen by doctor and occurrence of Type-II reaction

Time interval (month)	No. of patients	Percentage (%)
0-1	02	04.53
1-6	13	61.90
7-12	06	28.57
Total	21	100.0

(52.38%) cases, lymphadenopathy in 9 (42.85%) cases, eye involvement in 8 (38.09%) cases, epididymo-orchitis in 5 (23.80%) cases, epistaxis in 3 (14.28%) cases and gynaecomastia in 2 (9.52%) cases.

In our study 20 patients (95.23%) received anti-leprosy drugs (standard MB MDT) before developing the Type-II reactions which might have acted as precipitating factor (Table 4).

Ten patients (47.63%) had 3 recurrent episodes of Type-II reaction and 3 (14.29%) had 4 recurrent episodes.

As may be seen (Table 5), 13/21 (61.9%) patients reported to the doctor with 1 to 6th months, where as 2/21 (4.5%) patients reported to doctor within one month of starting treatment.

Discussion

Leprosy is a chronic infectious communicable disease caused by *Mycobacterium leprae* and still continues to be serious public health problems (Dharmendra 1978). *Mycobacterium leprae* primarily affects and damages the peripheral nerves that ultimately result in anaesthesia, paralysis, autonomic disturbances, ulcerations and deformities (Jopling and McDougall 1992). The indolent course of the disease is interrupted by acute outburst termed as reactions, during which there is acute neuritis that is a most painful complication of lepra reaction (Bedi and Bhutani 1975).

When we compared our study with published literature we found that Nigam et al (1975) and

Sehgal et al (1977) also reported the age group 20-40 years to be common, being 59.5% and 63.1% respectively and 7.1% and 6.8% patients to develop reactions below the age of 21 years respectively. Lockwood et al (1993) observed the mean age 35 years in a study of reversal reactions. Male predominance has been observed 3:1 by Nigam et al (1975), 1.7:1 by Sehgal et al (1977) and 1.3:1 by Debi and Mohanti (1977). Rea and Levan (1975) observed ENL lesions in 100% cases of their series. Ramu and Ramanujam (1964) observed ENL lesions to be present only in 28.2% of cases, exacerbations of existing lesions in 6.7% cases in 135 patients of lepromatous leprosy. Debi and Mohanti (1977) reported ENL lesions in 54.5%, exacerbation of existing lesions in 90.9% and new lesions in 81.8% cases in 55 lepromatous leprosy patients. Ramu and Ramanujam (1964) reported joint pain in 62.2%, lymphadenitis in 46.7%, peripheral edema in 22.2%, epididymoorchitis in 5.9%, gynaecomastia in 2.2% cases. Nigam et al (1975) in a study of lepromatous leprosy in reactions, observed fever in 96.7%, epistaxis in 25.8%, epididymo-orchitis in 12.9% and gynaecomastia in 6.4%. Rea and Levan (1975) noticed fever in 56%, joint swelling and tenderness in 1.6% and epididymo-orchitis in 12%. Sehgal et al (1977) reported fever in 100%, joint pain in 87.5%, and peripheral edema in 40.6%. Debi and Mohanti (1977) observed fever in 90.9%, lymphadenitis in 98.2%, peripheral edema in 72.7%, neuralgia in 58.2%, joint pain in 54.5% and eye involvement in 50.9%. Nigam et al (1975)

and Sehgal et al (1977) have reported that 64.4% and 22.3% patients got precipitation of reactions after dapsone therapy. Debi and Mohanti (1977) reported sulphone as precipitating factors in 2.53% patients. On the other hand Dhople and Mager (1963) stated that lepra reactions were not related to the Dapsone concentration in blood. Barnetson et al (1976) observed that Dapsone may provide protective effect in controlling the reactions. Lockwood et al (1993) noticed reversal reactions in 50% patients within one month after starting anti-leprosy treatment, out of 494 cases of leprosy. Sehgal et al (1977) observed 15.6% of lepromatous leprosy developed more than 4 episodes of reaction and in borderline group, 25.7% developed 2 episodes of reaction. Jopling and McDougall (1992) stated that most likely time to develop upgrading reaction is during first 6 months but longer interval have been recorded in borderline lepromatous cases. Lockwood et al (1993) observed period of 12 to 24 months after first symptoms as peak time to develop reactions.

Our study carried out during MDT era shows that profile of symptomatology as well as triggers for initiating type II reactions do not seem have not changed. As number is small, such study should include statistically significant numbers. However, there could be variation in the course of these reactions due to changes in bacillary load of cases diagnosed and Clofazimine being part of regular Multibacillary regimen after MDT. There is need to carry out such periodic assessment in cases being treated in different parts of country.

References

- Barnetson R Stc, Pearson JMH, Rees RJW (1976). Evidence for the prevention of borderline leprosy reactions by dapsone. *Lancet.* 2: 1171-1172.
- 2. Bedi TR, Bhutani LK (1975). Reaction in leprosy-1

- (Nomenclature, pathogenesis, pathology and clinical features). *Indian J Dermatol Venerol.* **41**: 176-180.
- Debi B, Mohanti HC (1977). Reactional state of leprosy - A clinical assessment. Lepr India. 49: 229-233.
- 4. Dharmendra (1978). Acute exacerbations (reactions) in leprosy. In: Leprosy. 1st vol. 1st ed. Editor Dharmendra: Kothari Medical Publishing House, Bombay. pp.108-139.
- 5. Dhople AM, Mager NG (1963). Absorption and excretion of DDS in leprosy. *Int J Lepr.* **31**: 68.
- 6. Job CK (1989). Nerve damage in leprosy-XIII Leprosy Congress State of the art Lectures. *Int J Lepr.* **57**: 532-539.
- Jopling WH, McDougall AC (1992). Leprosy reactions (Reactional states). In: Handbook of Leprosy 4thedn. Editors. Jopling WH and Mc-Dougall AC; Heinemann Professional Publishing Oxford. pp. 83-92.
- Lockwood DNJ, Solomonm V, John NA et al (1993).
 Clinical features and outcome of reversal (Type-I) reactions in Hyderabad, India. Int J Lepr. 61: 8-15.
- Nigam P, Mukhija RD, Goyal BM et al (1975). Leprosy - A study of reactions among 398 patients of leprosy. *Indian J Dermatol Venerol*. 41: 183-186.
- 10. Pfaltzgraff RE, Bryceson A (1979). Management of Reactions: In Medicine in Tropics, Leprosy. 2nd ed. pp.72-76.
- 11. Ramu G, Ramanujam (1964). Reactive state in lepromatous leprosy study of clinical and bacteriological aspects. *Lepr India*. **36**: 3-19.
- 12. Rea TH, Levan NE (1975). Erythema nodosum leprosum in a general hospital. *Arch Dermatol*. **111**: 1575-1580.
- 13. Sehgal VN (1979). Reactions in leprosy, In: Clinical Leprosy. Vikas Publishing House Pvt. Ltd. Ghaziabad, UP, India. XIII: pp.52-58.
- 14. Sehgal VN, Rege VL, Mascarenhas MF (1977). Pattern of reactions in leprosy. A clinical appraisal. *Lepr India*. **49**: 221-228.

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