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CORRESPONDENCE

This department is for the publication of informal communications that are of interest because they are informative and stimulating, and for the discussion of controversial matters. The mandate of this JOURNAL is to disseminate information relating to leprosy in particular and also other mycobacterial diseases. Dissident comment or interpretation on published research is of course valid, but personality attacks on individuals would seem unnecessary. Political comments, valid or not, also are unwelcome. They might result in interference with the distribution of the JOURNAL and thus interfere with its prime purpose.

Peptic Ulcer and Corticosteroid Therapy

TO THE EDITOR:

It is usually believed that corticosteroid therapy is contraindicated in peptic ulcer. Some even believe that steroids cause peptic ulcer, even though others call this a myth and claim that the addition of nonsteroidal antiinflammatory agents (NSAIDs) to corticosteroid therapy contributes to the causation of peptic ulcer ([1–4]). Whatever be the ultimate truth, the problem of corticosteroid therapy and the possible precipitation of acid peptic disease is very relevant in leprosy since most of the leprosy reactions (both type 1 and type 2) are treated by corticosteroids.

We present here a patient who developed such a serious problem during corticosteroid therapy and how we approached the problem.

A 35-year-old male patient [borderline lepromatous leprosy, skin-smear positive, bacterial index (BI) = 4+] came to our hospital in February of 1997. He had been a chronic smoker for more than 10 years, and he had had episodes of abdominal pain relieved by food for 1 year. He was started on multibacillary multidrug therapy (MB/MDT) and sent home. After 1 month in March of 1997 he developed type 1 reaction with weakness of the left ulnar supplied muscles. MDT was continued and oral prednisolone 30 mg/day was added along with oral antacids. One month later (April 1997) he developed severe abdominal pain and was rushed to a nearby hospital where a perforated peptic ulcer was found at laparotomy. During the postoperative period after closure of the perforation and convalescence, prednisolone was discontinued. He once again developed a very severe type 1 reaction with multiple paralyses in May of 1997 and returned to our hospital. He felt grossly handicapped in the following ways: a) walking was seriously compromised due to bilateral foot drop; b) dressing and undressing and even eating became big problems (South Indians eat by scooping up the food, usually rice, with the right hand) because of bilateral ulnar median paralysis and, of course, gainful employment was totally out of the question with these multiple paralyses.

We decided to start corticosteroid therapy under cover of anti-ulcer therapy (oral ranitidine and oral antacids). The patient was started on oral prednisolone 40 mg/day, ranitidine 150 mg was given twice a day, and antacid liquids three times a day. When the dose of prednisolone was reduced to 30 mg/day, ranitidine 150 mg was given once daily. Later, only oral antacid tablets were supplemented. After 4 months of continuous corticosteroid therapy the edema of the skin lesions has completely subsided and the paralyses have greatly improved (The Table). He does not presently have any visible deformity due to leprosy nerve damage. He has no problem with personal care and has resumed his farm work. He still has

THE TABLE. *Improvement of disabilities in response to steroid therapy.*

Right					Left			
Nov.	Sept.	July	May		May	July	Sept.	Nov.
				Voluntary muscle testing (VMT) (MRC grading 0–5)				
				Ulnar				
4	4	3	0	Abductor digiti minimi	0	2	2	2
4	4	3	0	First dorsal interosseus	0	3	3	4
				Median				
5	5	5	4	Abductor pollicis brevis	2	4	5	5
5	5	5	4	Opponens pollicis	2	4	5	5
				Common peroneal				
5	5	5	2	Tibialis anterior	2	5	5	5
5	5	5	3	Peronei	3	5	5	5
				Graded sensory testing—palms[a]				
				Ulnar				
5	4	0	0	Little finger pulp	0	0	4	3
4	4	4	0	Hypothenar eminence	0	1	0	2
				Median				
5	5	5	0	Thumb pulp	0	5	5	5
5	5	5	0	Index finger pulp	0	5	5	5
5	5	5	0	Thenar eminence	0	5	5	5
				Sensory testing—soles (posterior tibial)[b]				
1	0	0	0	Heel	0	0	0	0
0	0	0	0	Great toe pulp	0	0	0	0
0	0	0	0	Little toe pulp	0	0	0	0

[a] Sensory testing done by Semmes-Weinsten monofilaments:

Hands

Score	Filament force felt
5	200 mg
4	2.0 gm
3	4.0 gm
2	10.0 gm
1	300 gm
0	No filament felt

[b] Sensory testing done by Semmes-Weinsten monofilaments:

Feet

Score	Filament force felt
3	4.0 gm
2	10.0 gm
1	300 gm
0	No filament felt

Because of thickening of sole skin, thin filaments not used.

bilateral plantar anesthesia and we are continuing oral prednisolone 20 mg/day, hoping that he will regain at least plantar protective sensation.

As far as we know this kind of problem and its management has not been reported in the leprosy literature. Feedback from the readers of their experiences in such problem areas will greatly help to view these difficult situations with the proper perspective and will be of benefit to both patients and physicians.

—S. Arunthathi, M.D., D.D.

Head, Branch of Medicine

—D. Samuel Thomson Sugumaran, M.B.B.S., D.P.M.

Specialist, Branch of Medicine

—Sundar Raju Gorla, M.B.B.S.
Sunitha Abraham, M.B.B.S.

Medical Officers
Schieffelin Leprosy Research & Training Center
Karigiri 632 106
N.A.A. District, Tamil Nadu
South India

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Leprosy and HIV Infection in Bahia, Brazil

To the Editor:

Human immunodeficiency virus (HIV) infection progression is characterized by a gradual decrease of CD4+ T cells associated with the loss of host defenses against several pathogens and development of opportunistic infections. Since the protection against leprosy correlates with the expression of cell-mediated immunity, one could expect an increased number of leprosy cases among HIV-infected individuals. In Brazil, the number of registered cases of AIDS is continuously increasing. This is also a problem in Bahia, where leprosy is an endemic disease (prevalence rate per 10,000 was 3.91 in 1995). The aim of this study is to determine the possible association between new leprosy cases and HIV infection.

Newly diagnosed cases of leprosy who had attended the outpatient leprosy service of the Hospital Universitário Prof. Edgard Santos in Salvador, Bahia, Brazil, between March 1993 and May 1995 were enrolled in the present study. Additionally, sera obtained from leprosy patients and controls living at Irecê, an endemic leprosy rural area in the center of Bahia, were also included. A case of leprosy was defined as an individual with newly diagnosed and previously untreated leprosy, aged between 15–60 years, and a resident of Bahia. Up to two controls living in the same area and/or healthy contacts were chosen for each patient. A standardized questionnaire was used to register gender, age, residence and high-risk behavior to HIV infection in the two groups, including sexual activity, intravenous drug use and blood transfusion.

Blood was obtained from consenting patients or controls and the serum was separated and kept at –20°C until the realization of the assays to determine anti-HIV antibodies. All sera were tested with an enzyme linked immunosorbent assay (ELISA) for antibodies to HIV (HIV-1/HIV-2 ELISA test kit; Cambridge Biotech). The positive samples were further analyzed with a second ELISA (Enzygnost anti-HIV 1/2 plus; Behring), or with a solid-phase enzyme immunoassay (Immunocomb Bi-spot HIV1 and HIV2). The samples which gave negative results in the second ELISA and/or Immunocomb were considered negative for HIV infection. The samples that remained positive were tested by Immunofluorescence assay (IFA) or Western blot (WB) (Genelabs diagnostic HIV1 1.3, Cellular products) in order to confirm HIV seropositivity.

RESULTS AND DISCUSSION

Sera from 234 patients and 468 controls were evaluated. Distribution by gender and age are shown in Table 1. Most of the patients (79.5%) and controls (86%) lived in Salvador city and the remainder in rural areas.

Case distribution according to leprosy classification and the results of HIV tests are shown in Table 2. Although 13 out of 234 patients and 1 out of 468 controls had at least one positive ELISA test, no positiv-