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.

Diabetic Ketoacidosis Following Steroid Therapy in a Rural Leprosy Hospital

TO THE EDITOR:

The diagnosis of ketoacidosis in a known diabetic is not difficult. However, its appearance in a patient not previously known to have diabetes will require differentiation from other common causes of metabolic acidosis, such as lactic acidosis, uremia, alcoholic ketoacidosis, etc. Its appearance as a result of drug therapy in leprosy is reported here.

A case of borderline tuberculoid leprosy was admitted to our hospital for management of foot drop. The patient was a 27-year-old male who presented with a large anesthetic patch on his left leg and a left-sided foot drop. There were also a few patches on the trunk and arms. His bacterial index (BI) (read from routine sites) was negative for acid-fast bacilli (AFB). Routine blood counts and urine sugar were normal or negative at admission. The patient was hospitalized at the time of induction of multidrug therapy (MDT) because of the short duration of the left foot drop which required steroid therapy. He was started on the World Health Organization (WHO) recommended multibacillary (MB) MDT. The patient was also put on 60 mg of prednisolone once daily. (This is the starting dose we adopt in adult males who are inpatients and have presented with severe paralysis of short duration. Major side effects have been none so far in our 4-year experience here.) This was steadily tapered by 5 mg every 2 weeks.

After approximately 12 weeks of steroid therapy, his foot drop condition was showing gradual improvement in muscle power (from 0 to 3 in tibialis anterior). At about this time the patient complained of increased frequency of micturition and polydipsia. His urine sugar done at this time was reported as 4+ by the dipstick method. Fasting and postprandial blood sugars were done and reported as 219 mg/dl and 298 mg/dl. The diagnosis of diabetes mellitus was obvious. The possibility of steroid-induced diabetes was considered. The only other drugs the patient received were the MDT noted above.

To avoid the side effects of abrupt steroid stoppage, tapering was hastened and the patient given an oral antidiabetic drug along with diet control. The patient apparently improved and obtained a 1-hr postprandial sugar reading of 111 mg/dl within a week. Two days later the patient complained of generalized weakness, anorexia and giddiness. There was nausea but no icterus. He was passing urine normally. On examination the patient appeared drowsy but fully conscious. Higher functions were normal. The pulse was 110/min, blood pressure 120/80 mm Hg, and respiratory rate was normal but deep. The smell of ketones in his breath raised the suspicion of ketoacidosis.

A spot blood sugar test showed 273 mg/dl and a urine test for ketone bodies was positive. A chest X-ray was normal and an electrocardiogram did not show peaking of

T waves or the presence of U waves. Other conditions, such as starvation ketosis, lactic and alcoholic ketoacidoses, were ruled out only by history and clinical features. Prednisolone was stopped, and therapy for diabetic ketoacidosis was initiated with fluid replacement and intravenous insulin. His blood sugar and urine acetone were monitored frequently. No other blood chemistry analysis could be done. All vital signs including intake-output were monitored and recorded round the clock. After 48 hr of therapy the urine was rendered negative for ketone bodies, and blood sugar levels reached normal on insulin.

The patient was kept on insulin for a week, but was changed to an oral antidiabetic agent at the patient's request. The patient also was discharged on request. Muscle power with regard to his foot drop after the initial improvement was static at the time of discharge.

From this report a few points may be pertinent for leprosy workers:

1) Peripherally placed leprosy hospitals normally do not or ideally should not manage a condition like ketoacidosis. Present-day management of this condition will require, in addition to what we could do, monitoring of serum electrolytes, bicarbonate values, pH and osmolarity, among others. Key parameters followed to assess therapeutic progress are the pH and the anion gap ([2]). Yet physicians dealing with poor leprosy patients in countries such as India are known to face situations where a patient is simply unable to afford transport or funds for treatment in specialist centers which are generally few and far between. This case shows that prior knowledge of the possibility of such a condition occurring, early detection, and appropriate although simple management can salvage the situation.

2) It has been noted that prolonged exposure to large doses of glucocorticoids leads to a diabetic-like state ([3]). The initial dose that our patient received may have been a causative factor and the ketoacidosis may have been triggered by the continuation of the prednisolone, although in smaller doses. Leprosy hospitals using similar or higher doses of prednisolone may do well to check blood or urine sugar frequently. Also, interaction with the other drugs that the patient was receiving was considered but unlikely. Rifampin, due to its property of enzyme induction, will only reduce the half-life of prednisolone ([4]).

3) Steroids are often advocated in leprosy patients with neuritis for preventing deformity in patients on MDT. Field programs dispense prednisolone to be taken on a domiciliary basis for patients with neuritis or muscle weakness ([1]). In our case, it is still not clear whether the steroid therapy unmasked latent diabetes or whether the diabetes was *de novo* and purely steroid induced. Unfortunately, the patient has yet to return for his first follow up.

This case should serve as a reminder that diabetes can arise in patients on prednisolone and can progress to a life-threatening condition such as ketoacidosis if therapy is continued. The policy of dispensing steroids on a domiciliary basis must be reviewed. At least intermittent checks on blood or urine sugars of patients on steroids must be considered. Clinicians managing medical problems in leprosy need to be aware of the possibility that diabetes or its complications may arise in patients on relatively short-term steroid therapy.

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REFERENCES

1. Becx-Bleumink, M., Berhe, D. and T'Mannetje, W. The management of nerve damage in the leprosy control services. Lepr. Rev. **61** (1990) 1–11.
2. Foster, D. W. Diabetes mellitus. In: *Harrison's Principles of Internal Medicine.* 12th edn. Wilson, J. D., Braunwald, E., Isselbacher, K. J., *et al.*, eds. New York: McGraw Hill, 1991.
3. Haynes, R. C., Jr. Adrenocorticotropic hormone; adrenocortical steroids and their synthetic analogs; inhibitors of the synthesis and actions of adrenocortical hormones. In: *Goodman & Gilman's The Pharmacological Basis of Therapeutics.* 8th edn. Gilman, A. G., Rall, T. W., Nies, A. S. and Taylor, P., eds. New York: Pergamon Press, 1990, pp. 1431–1463.

4. MANDELL, G. L. and SANDE, M. A. Antimocrobial agents (continued); Drugs used in the chemotherapy of tuberculosis and leprosy. In: *Goodman & Gilman's The Pharmacological Basis of Therapeutics.* 8th edn. Gilman, A. G., Rall, T. W., Nies, A. S. and Taylor, P., eds. New York: Pergamon Press, 1990, pp. 1146–1165.

Infected Trophic Ulcers and Tenderness of Posterior Tibial Nerve in Cured Leprosy Patients

TO THE EDITOR:

This is with reference to the letter by Dr. Anil H. Patki on the above subject in the September 1993 issue [Vol. 61 (3) pp. 473–474].

1. Very often patients with acutely inflamed plantar ulcers complain of pain (which can be quite severe) in the foot, and careful questioning reveals the site of pain as behind the tibial malleolus. The posterior tibial nerve shows exquisite tenderness in these cases. The inguinal lymph nodes are often, but not always, enlarged and tender. The nerve tenderness improves as the ulcer improves, rather slowly but steadily. Mild tenderness may persist for a while even after complete healing of the ulcer.

2. In my opinion this is not merely due to lymphangitis although that, perhaps, contributes to the clinical picture because: a) there is rarely, if ever, any associated inflammation of the superficial lymphatics in most of these cases, and b) the tenderness persists, although at a lower level, for some time even after control of the acute infection. It appears quite likely that there is a real neuritis of the posterior tibial nerve in these cases only it is not related to leprosy, as such, but to acute infection in the ulcer. The fact that the incidence of tetanus in leprosy patients with chronic plantar ulcers appears to be very much less than one would expect has been explained on the basis of intraneural and perineural fibrosis which would also block the lymphatics ([1]).

3. In the old dapsone monotherapy days, this clinical phenomenon was not very much stressed and, also, it did not matter. However, the situation today is different and, as Dr. Patki says, somebody examining such a patient (who has already been treated and discharged as cured) may easily diagnose disease activity/acute neuritis/reaction/relapse just because the posterior tibial nerve is very tender. I congratulate Dr. Patki for bringing this matter to our attention and I endorse his cautionary statement.

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REFERENCES

1. SRINIVASAN, H. and DHARMENDRA. Neuropathic ulceration. In: *Leprosy, Vol. I,* Dharmendra, ed. Bombay: Kothari Medical Publishing House, 1978, p. 232.

Mast Cells in Lepromatous Leprosy

TO THE EDITOR:

Leprosy is a chronic disease, caused by *Mycobacterium leprae,* which is characterized histologically by granuloma formation. The type of granuloma depends upon the cellular-immune response of the host to the organism ([8]). Apart from the usual cells which participate in the granuloma, such as epithelioid cells, macrophages and lymphocytes, mast cells also have been observed in the lesion. It has been noted that the numbers of mast cells in lesions of lepromatous leprosy are more compared to those of tuberculoid leprosy ([1]). The purpose of this study is to study the role of mast cells in treated and untreated cases of lepromatous leprosy.

Skin biopsies from 21 patients with lepromatous leprosy were reviewed. Classifi-