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route for intubation is generally not recommended in the presence of a coagulopathy and if not performed with due care can result in bleeding which could complicate the intubation procedure as well as further compromise the patient's airway. Also, this management option would not be viable in the presence of active oral bleeding. Conservative management with oxygen therapy is another option, bearing in mind that if deterioration occurred rapidly, the option of a controlled awake fiberoptic intubation may be lost. Finally, tracheostomy. In a stable, partially obstructed patient, one would try to avoid a tracheostomy, in view of the transient nature of the obstruction. However, this was felt to be the only option for this patient who was hypoxic due to an upper airway obstruction, unconscious with intraoral

bleeding and unable to protect the airway. Airway patency can be temporarily achieved using a nasopharyngeal airway. Despite the fact that insertion of the nasopharyngeal airway is likely to cause haemorrhage, provision of a life saving patent airway is the only consideration in a totally obstructed patient. Subsequently, a tracheostomy can be done urgently to secure the airway as well as to protect it from aspiration of blood and gastric contents.

General anaesthesia is administered with great caution, as is sedation, in any upper airway obstruction, as this can lead to inability of the patient to maintain his airway and convert a partial obstruction to a complete one.

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Neural Leprosy – A Case Report

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Summary

Neural leprosy is rare. This is a report of a 63-year-old Indian man who had long standing multiple peripheral neuropathy. The slit skin smear for acid-fast bacilli of *Mycobacterium leprae* was positive. The skin and nerve biopsies were normal. He was treated with rifampicin, dapsone and clofazimine.

Key Words: Neural leprosy, Slit skin smear, Nerve biopsy

Introduction

The varying clinical features in leprosy provides a challenge in its diagnosis. This is usually straightforward when both cutaneous lesions and neurological damage are present together in the same patient. In the absence of clinically apparent skin lesions, neural leprosy is not only uncommon but difficult to diagnose.

The following is such a case presenting with multiple peripheral neuropathy and the diagnosis was confirmed with the finding of acid-fast bacilli in the slit skin smears.

Case History

A 63-year-old Indian man was seen in September 1996. He had numbness of his hands and feet for 2 to 3 years. At the same time there was progressive and painless loss of his toes each time following trauma. His right leg was weak and he had been unable to walk for the last 6 months. There was no history of any skin rash and there was no contact with persons with leprosy. The patient is non-diabetic. He has had a long history of alcoholic ingestion but stopped for 6 months prior to examination as he could not walk to the 'toddy' shop.

On examination, he was dishevelled and had bed sores on his back and at the side of his right knee. There was mild pallor. He was afebrile. There were bilateral claw hands with wasting of intrinsic muscles, loss of all toes of the right foot and the second toe of the left foot. There was severe wasting of muscles of the right calf and there was a right foot drop. Loss of sensation in a glove and stocking distribution was present up to the levels of both elbows and knees. The right ulnar and right posterior tibial nerves were enlarged but not tender. The deep tendon reflexes were present except for the right ankle jerk. Inspection of his skin did not reveal any lesion suggestive of leprosy. The rest of the examination was normal.

Investigations revealed hypochromic, microcytic anemia. Iron deficiency was confirmed with a low serum iron level and elevated total iron binding capacity. Blood sugar, liver and renal function tests were normal. The VDRL test was non-reactive. His chest X-ray was normal.

The slit skin smear for acid-fast bacilli (AFB) was positive. The bacteriological index (B.I.) was 0.3 and the morphological index (M.I.) was 0. The positive smears were from both ear lobes. The results of our slit skin smear were confirmed in Sungai Buloh Hospital where fragmented AFB were present in both ear lobes and from the right elbow.

The skin biopsy from the right forearm did not reveal any granulomata and the Fite stain did not reveal any AFB. The nerve biopsy taken from the right saphenous nerve was normal. This patient was diagnosed as borderline neural leprosy. He was started on a 3-year course of rifampicin, dapsone and clofazimine. His treatment was supplemented with haematinics, vitamin B complex, physiotherapy and education on the care of anaesthetic hands and feet.

Discussion

Neural leprosy is rare in Malaysia. A case was reported by Ganesapillai in 1988 where the patient was a young boy with bilateral ulnar nerves involvement¹. Neural leprosy is not uncommon in India where it amounted to one sixth of all cases².

Neural leprosy is difficult to diagnose because of the absence of skin lesions. In this patient, the absence of skin lesions maybe due to previous indeterminate lesions which could have gone unnoticed and which had self-healed.

Enlargement of peripheral nerves in neural leprosy is not consistently present. Nerve abscess has also been described. In this patient, neural leprosy was diagnosed based on the presence of multiple peripheral neuropathy and on finding AFB in the slit skin smear. The skin and nerve biopsies were normal. Other causes of peripheral neuropathy were also considered here especially alcoholic neuropathy. However this causes a symmetrical and distal neuropathy without any nerve enlargement and no AFB in the skin smears.

Biopsy of a peripheral sensory nerve is extremely helpful in establishing leprosy as the cause of a peripheral neuropathy if granulomata or AFB are found. However, a normal result does not exclude its diagnosis. In a review of 77 patients with neural

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leprosy, only 50% had histologic evidence of leprosy. Of the remainder, 60% showed no abnormalities, 30% showed demyelination, 5% showed vasculitis and 5% showed neural hyalination³.

In general the skin smears for AFB of *M. leprae* tend to be negative in neural leprosy. Furthermore the absence of any skin lesion makes it difficult to choose the sample site. In this patient, the skin smear had fortuitously been extremely helpful. Slit skin smear is a simple test widely available in our skin clinics. This is in contrast to nerve biopsy which is technically more difficult to perform.

The spectrum of neural leprosy tends to reflect better cell mediated immunity ie. the tuberculoid and borderline forms. This patient was classified as borderline neural leprosy to reflect the multiple nerves involvement and the low B.I. In accordance with smear

positive cases, he was started on a 3-year course of rifampicin, dapsone and clofazimine. Although corticosteroids will be useful in reducing the neuritis, it was not given to him because, on balance, the risks of steroid side effects appeared to outweigh any little benefit in reversing the long standing neurological damage in this patient.

In conclusion, leprosy is endemic in our country and should be considered in our patients presenting with peripheral neuropathy. The slit skin smear is easily available in our skin clinics and may prove invaluable in its diagnosis.

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