Stridor and parkinsonism

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Summary

A patient is described with idiopathic Parkinson’s disease and severe laryngeal stridor. Other than urinary frequency and urgency, not uncommon in this condition, and postoperative laevodopa-sensitive postural hypotension, there were no features of generalized autonomic failure. The laryngeal stridor responded to laevodopa therapy and we are not aware that this has been reported previously.

KEY WORDS: Parkinson’s disease, stridor, laevodopa.

Introduction

Stridor is a very unusual feature of parkinsonism, only two cases having been reported, one of these having atypical features including extensor plantar responses (Vas, Parsonage and Lord, 1965). We describe a third case which responded to laevodopa.

Case report

A previously healthy 72-year-old woman presented with a 6-month history of hoarseness, stridor, clumsiness of the left hand, salivation, lethargy, constipation, weight loss and occasional nocturia. There was marked inspiratory and expiratory stridor, worse on exercise. Rest tremor affected both hands and there was severe bradykinesia with mild cogwheel rigidity, mainly left-sided. The blood pressure was 140/80 mmHg without postural fall.

Haematological and biochemical profiles, thyroid function tests, chest and skull radiographs, technetium brain scan, barium swallow and meal examinations were all normal. The Wasserman reaction was negative. Laryngeal tomography and indirect laryngoscopy showed bilateral vocal cord palsy with the cords fixed in the paramedian position.

A diagnosis of parkinsonism was made and laevodopa 250 mg and carbidopa 25 mg (Sinemet) 3 times a day prescribed. After 3 months, there was marked improvement in the stridor but she developed urinary frequency and urgency. One month later, she was readmitted with worsening symptoms although it was subsequently discovered that she had halved her treatment dosage. There was no postural hypotension and the other physical signs were unchanged. The cerebro-spinal fluid was normal and electromyography of the laryngeal muscles did not show any lower motor neurone pathology.

Withdrawal of laevodopa led to an immediate exacerbation of the parkinsonism and an alarming increase in the stridor, now present at rest and during sleep. Reintroduction of laevodopa produced a dramatic improvement in the parkinsonism and also in the stridor which could now only be detected during exercise. Despite this improvement, prophylactic tracheostomy was thought desirable. Postoperatively, the urge incontinence increased and severe symptomatic postural hypotension developed, only partially improved by withdrawing laevodopa.

During the next 21 months, the parkinsonism worsened despite anti-cholinergic drugs. No evidence of generalized autonomic failure as judged by simple clinical tests (Campbell et al., 1976) and sweating ability was obtained but there was no improvement in the postural hypotension, although it was now asymptomatic. Increasing disability led to admission to the geriatric unit where 9a-fludrocortisone and elastic stockings produced a normal blood pressure and allowed the use of small doses of laevodopa which improved the parkinsonian symptoms. Thirty-three months after the onset, she was found dead in bed at home. No autopsy was obtained.

Conclusion

The remarkable response of the stridor to laevodopa strongly suggests that it was part of the parkinsonism. Laryngeal spasm in post-encephalitic parkinsonism and oesophageal spasm in the idiopathic form have been reported. Bendall (1976) described parkinsonian bovine cough and a man with typical parkinsonian features ‘whose breathing is of a
half-sobbing character’ was reported by Buzzard (1881).

Urge incontinence and postural hypotension do occur in some patients with idiopathic parkinsonism and our patient had no other features of olivo-ponto-cerebellar degeneration or the Shy-Drager syndrome in both of which stridor occurs (Williams, Hanson and Calne, 1979). Of particular interest, however, are the laryngeal electromyographic studies of Guindi et al. (1981) on 5 patients with multiple system atrophy and a further 9 patients with Parkinson’s disease, 2 of whom had autonomic failure in addition. Abnormalities were found in all 5 patients with multiple system atrophy but, like the patient described here, both their patients with Parkinson’s disease and autonomic failure were electromyographically normal. To our knowledge, the stridor of the Shy-Drager syndrome has not been recorded as responding to laevodopa; it would appear therefore that although rare, marked laryngeal stridor may occur in classical Parkinson’s disease and we have been unable to find any previous report of this particular symptom responding to laevodopa.

Acknowledgments

We are grateful to Dr Geoffrey Rushworth for performing the electromyographic studies.

References


(Accepted 7 December 1982)
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Postgrad Med J 1983 59: 520-521
doi: 10.1136/pgmj.59.694.520

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