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Rabbit Syndrome – A Rare Complication of Long-Term Neuroleptic Medication

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A case of rabbit syndrome, a complication of long-term neuroleptic medication, is reported. It is important to differentiate it from tardive dyskinesia and continuous therapy with an antiparkinsonian agent may be required for control of symptoms of rabbit syndrome.

The term 'rabbit syndrome' was first coined by Villeneuve (Sovner & Dimascio, 1977). It is an uncommon, tongue-sparing, orofacial movement disturbance, which is rapid and regular and associated with prolonged use of neuroleptics (Todd *et al*, 1983).

It is important to diagnose tardive dyskinesia differentially from rabbit syndrome by noting the sparing of tongue movements and the response to antiparkinsonian agents in the latter.

Case report

Mr K, a 52-year-old male patient, diagnosed as suffering from schizophrenia since 1969, had been admitted to hospital repeatedly for acute exacerbations of psychotic features. He had been receiving neuroleptics almost regularly for 20 years, on an out-patient basis. On one such out-patient visit, he was found to have orofacial and jaw movements which were rapid, involuntary and rhythmic and were occurring at a rate of 50 cycles/min. There were no involuntary movements of the tongue or of any other part of the body. These movements reduced on voluntary activity. No other signs of extra-pyramidal dysfunction were found.

A diagnosis of rabbit syndrome was made and oral trihexyphenidyl (2 mg t.i.d.) was started and oral trifluoperazine, which he had been receiving, was discontinued. By the third day of starting trihexyphenidyl, his orofacial and jaw movements reduced, disappearing completely by the fifth day. The patient, however, stopped his medication of his own accord and his abnormal orofacial and jaw movements reappeared after four to five days. He was restarted on oral trihexyphenidyl (2 mg t.i.d.) and again his abnormal movements disappeared completely within four to five days. Since then he has been on oral trifluoperazine (5 mg t.i.d.) and trihexyphenidyl (2 mg t.i.d.) and is followed up regularly.

Discussion

Our case illustrates the successful use of oral trihexyphenidyl in controlling the symptoms of rabbit syndrome. The disappearance and reappearance of the symptoms of rabbit syndrome after a period of starting and stopping the antiparkinsonian agent suggests that the syndrome does respond to these drugs, and continuous treatment may be required to control the symptoms. These observations also help to confirm the diagnosis of rabbit syndrome. A favourable response to an antiparkinsonian agent indicates that the pathophysiology of rabbit syndrome may be similar to that of acute forms of drug-induced parkinsonism (Jus *et al*, 1972).

Both rabbit syndrome and tardive dyskinesia are usually seen after a prolonged exposure to neuroleptic agents, in middle-aged or elderly patients. The high frequency of the perioral movements, and the absence of lingual movements are the main features that distinguish rabbit syndrome from tardive dyskinesia since the latter is characterised by less frequent, more irregular movements (Todd *et al*, 1983). The use of intravenous physostigmine has also been described to distinguish between rabbit syndrome and tardive dyskinesia. Physostigmine may worsen the symptoms of rabbit syndrome but reduce those of tardive dyskinesia (Weiss *et al*, 1980). However, we did not use this test.

Recognising rabbit syndrome and differentiating it from tardive dyskinesia is thus important in the proper treatment of orofacial disorder following long-term neuroleptic medication.

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