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CASE REPORT

Acute Hypertensive Episode Induced by Sulpiride – A Case Report

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A case study is described of a hypertensive episode associated with sulpiride which is a selective antagonist of cerebral dopamine receptors. The association with sulpiride was confirmed by withdrawal and rechallenge with the drug.

KEY WORDS–Sulpiride, neuroleptic, hypertension.

INTRODUCTION

Sulpiride is a selective antagonist of cerebral dopamine (DA_2) receptors, with no significant cardiovascular activity. Hypertension associated with its use has not previously been reported in this country. We describe a case in which an acute rise in blood pressure (BP) in a previously normotensive patient was seen with sulpiride and confirmed by withdrawal and rechallenge with the drug.

CASE REPORT

A 76 year old caucasian woman had received treatment for a severe depressive illness with imipramine 50 mg tds for 6 weeks. There was a substantial but incomplete response and a persistence of paranoid symptoms. Sulpiride 400 mg tds was commenced after discontinuation of the imipramine.

Following four doses (1600 mg) of sulpiride, the patient complained of feeling unwell, with dizziness, headache and palpitations, and a BP of 210/110 mm/Hg was recorded. The medication was stopped and the BP returned to pre-treatment levels (140/90 mm/Hg) within 48 hours. The patient was later rechallenged with a single dose of 400 mg sulpiride. An asymptomatic rise in BP to 170/105 mm/Hg was recorded, which returned to pre-treatment levels within 48 hours. Prior to admission, the patient had been fit with no past medical history of note. Physical examination and routine blood screen were normal. CXR showed slight cardiac enlargement, but ECG was within normal limits. Twenty four hour urinary Vanillyl Mandelic Acid (VMA's) were 40 umol/24 hours.

DISCUSSION

Sulpiride has been registered in the UK since 1983 for treatment of schizophrenia. During this period the manufacturers have received no reports of hypertensive episodes (Squibb-personal communication). The CSM have received three reports of hypertension associated with its use (CSM – personal communication).

In a retrospective review of 65 publications covering 2851 patients treated with sulpiride (Alberts *et al.*, 1985), there were three reports of hypertension. A single case report of a hypertensive crisis was attributed to sulpiride but without rechallenge (Licata, 1981). Corvol *et al.* (1974) found that sulpiride at a dose of 100 mg caused a significant further rise in blood pressure in 6 out of 26 hypertensive patients. In four of these there was a rise in urinary VMA, three of whom were subsequently shown to have phaeochromocytoma. Sulpiride is contraindicated in the presence of this condition. In the case of our patient, she was previously normotensive and there was no abnormality in urinary VMA excretion. Rechallenge with sulpiride indicates that the hypertensive effect is unlikely to be due to an interaction with imipramine with which she had previously been treated.

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The mechanism by which sulpiride may cause hypertension is unknown. It inhibits the hypotensive effect of dopaminergic agonists by blockade of the peripheral DA_2 receptor (Lombardi *et al.*, 1987). Neuroleptics, however, characteristically produce orthostatic hypotension by peripheral blockade of the postsynaptic adrenergic alpha1 receptor. Sulpiride, a substituted benzamide is chemically related to the antiemetic metoclopramide. Both of these drugs block alpha2 adrenoreceptors but have no effect on alpha1 receptors and have caused hypertensive crises in the presence of phaeochromocytoma (Plouin *et al.*, 1976; Agabiti-Rosei *et al.*, 1977). It has been suggested (Davies, 1987) that (in the presence of phaeochromocytoma) since the activation of presynaptic alpha2 receptors reduces the release of noradrenaline from nerve terminals, blockade by sulpiride might cause hypertension in the presence of excessive stores in the nerve endings.

Hypertension induced by sulpiride is uncommon and hypertensive crisis rare. Nevertheless, the possibility of an acute hypertensive episode should be borne in mind when instituting treatment.

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