Correspondence

EDITED BY LOUISE HOWARD

Risperidone treatment of amphetamine psychosis

Sir: Risperidone is an atypical antipsychotic drug indicated for psychoses in which both positive and negative symptoms are prominent. Misra & Kofoed (1997) have reported for the first time a case of methamphetamine-associated psychosis responding to risperidone. In that report risperidone was prescribed after the discontinuation of methamphetamine. We report another case of amphetamine psychosis responding to risperidone while still on amphetamine.

Mrs P., a 76-year-old married Caucasian woman, has been on dexamphetamine for narcolepsy since the age of 28. She remained well until September 1996 when she developed acute schizophrenia-like psychosis. She experienced auditory hallucinations – voices told her that her husband was having an affair, and she believed people were following her and commenting on her activities. She also believed an implant had been placed in her skull and voices were transmitted through a satellite. She attempted to remove the implant with a needle. There were no signs of an affective disorder. She required formal admission and responded well to sulpiride while still taking dexamphetamine.

She discontinued sulpiride after five months and again developed paranoid delusions and auditory hallucinations. She believed she would be burgled and killed, and stopped eating and drinking, believing her food was poisoned. She was admitted formally and treated with risperidone 3 mg and dexamphetamine 15 mg daily. Routine investigations were normal. She had recovered fully by discharge four weeks later. She was followed-up regularly and has remained well on risperidone and dexamphetamine for the past 18 months.

To our knowledge this is the first report of a successful outcome of risperidone treatment of amphetamine psychosis while on risperidone and dexamphetamine concomitantly. It has been a well-tolerated and effective treatment and may be useful for other narcolepsy sufferers.

Misra, L. & Kofoed, L. (1997) Risperidone treatment of methamphetamine psychosis (letter). *American Journal of Psychiatry*, **154**, 1170.

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Amiodarone-induced depression

Sir: Amiodarone hydrochloride is a class III antiarrhythmic agent used for the management of ventricular and supraventricular arrhythmias. The adverse effects of amiodarone are well documented and include cardiovascular effects (i.e. severe bradycardia and sinus arrest), thyroid malfunction, severe pulmonary toxicity (including pulmonary fibrosis and interstitial pneumonitis), liver toxicity, peripheral neuropathy, myopathy, ataxia and tremors (British Medical Association & Royal Pharmaceutical Society of Great Britain, 1998). However, amiodarone's effect on mental state does not appear to have been reported in the currently available literature, with the exception of an amiodarone-induced delirium that occurred 17 days after starting therapy in a 66-year-old man (Trohman, 1988).

We report this case in which amiodarone may have played a significant role in the timing, as well as in the clinical presentation, of a depressive episode.

A 65-year-old school dinner lady presented for admission in a severe retarded depressive state with obsessive–compulsive features. She appeared physically unwell and somewhat older than her age. The presenting clinical features included psychomotor retardation, fatigue, social withdrawal, and morbid preoccupation with health. The depressive symptoms had had a gradual onset, beginning nearly eight months before presentation. At that time she had suffered a myocardial infarction which was further complicated by ventricular tachycardia for which she was treated with amiodarone. At the time of presentation, she was already on a selective serotonin reuptake inhibitor, prescribed a few months earlier, but with little or no effect. Past psychiatric history revealed one major depressive episode that occurred postnatally and was treated with electroconvulsive therapy 37 years previously. She recovered fully and remained well until the onset of the presenting complaints. The patient has longstanding obsessive–compulsive disorder but with no real evidence of any previous neurotic or social decompensation at any time prior to the present episode.

The rather atypical presentation of her depressive episode in its mode of onset, course, duration, limited response to antidepressants and a predominant somatic component, favoured the possibility of an underlying aetiology other than just functional.

Thyroid function tests were within normal limits, with slightly elevated free thyroxine levels but normal levels of thyroid-stimulating hormone. There was no clinical evidence to suggest a hyperactive thyroid.

After consultation with the medical team, amiodarone was discontinued. Within one week and without any change in psychotropics, a rapid and dramatic improvement was observed in the mental state as well as her somatic symptoms. She became clinically asymptomatic except for her obsessional constitutional traits and longstanding obsessive–compulsive disorder.

The clinical improvement which coincided with the amiodarone withdrawal appears to suggest that amiodarone may be implicated, directly or indirectly, in triggering psychiatric symptoms in hitherto predisposed patients. In this case, we have no reason to assume that the patient's depression may have been caused by any amiodarone-induced thyroid disorder.

Although no firm conclusion can be made based on a single case, we recommend that amiodarone may be relevant in elderly psychiatric patients before assuming functional aetiology for a gradually developing depressive state.

British Medical Association & Royal Pharmaceutical Society of Great Britain (1998)

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