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SCIENCE DIRECT®
European Psychiatry 19 (2004) 237–240
EUROPEAN PSYCHIATRY

Letters to the Editor

Mianserin for the rapid improvement of chronic akathisia in a schizophrenia patient

Dear Sir,

Akathisia is a frequent and common adverse effect of treatment with antipsychotic medication. Chronic akathisia, a subtype of akathisia, has been defined as a mixed category including persistent acute akathisia and tardive akathisia in patients receiving long-term antipsychotic medication [1]. While management of neuroleptic-induced acute akathisia (NIA) usually responds to treatment [2], the treatment of neuroleptic-induced chronic akathisia (NICA) remains incomplete. We describe a patient whose NICA was rapidly resolved with mianserin treatment. A 55-year-old male outpatient with chronic schizophrenia had been treated with fluphenazine depot injections (12.5 mg) every 2 weeks and biperiden (2 mg) twice a day for the year prior to reassessment for movement disorder. He presented himself with signs of akathisia, which began soon after the initiation of fluphenazine injections and which was not ameliorated by biperiden co-administration. On first evaluation with the Barnes akathisia rating scale (BARS) he scored 4 (marked akathisia) on the global clinical assessment of akathisia subscale (GCA). He refused any change of his antipsychotic regimen. Diazepam was administered (10 mg/d) with no improvement of akathisia after 7 d of treatment. Diazepam was withdrawn and propranolol at a dose of 30 mg/d was initiated. His pulse decreased to 65 beats per minute with no beneficial effect on akathisia after 1 week of treatment. Due to decreased heart rate with only 30 mg/d propranolol, it was gradually discontinued and trazodone was administered (300 mg/d) before bedtime, however, he still scored 3 on GCA (moderate akathisia). He was withdrawn from trazodone and suffered full recurrence of akathisia at the same level of severity as initial presentation. Mianserin at a dose of 15 mg/d before bedtime was added to his regimen and when assessed after 1 week he experienced full remission of akathisia. According to the patient he felt complete relief the following day after initiation of mianserin treatment. To the best of our knowledge this is the first report of a serotonin antagonist-demonstrating efficacy in NICA [1]. In a double blind study mianserin has been demonstrated to be effective in the management of NIA [2] and trazodone in an open trial [3]. Apart from clozapine [4] to the best of our knowledge there are no reports of other medications that have demonstrated efficacy in the treatment of NICA. We suggest that treatment with serotonin antagonists may be effective for the treatment of chronic akathisia, as reported previously in the treatment of acute NIA [2]. The reasons for the improved efficacy of mianserin over trazodone (both serotonin 2A/2C antagonists) as noted in this case are unclear. However, while speculative, it is possible that the trazodone metabolite *m*-chlorophenylpiperazine [5], which has an anxiogenic effect mediated by its agonistic activity at the 5-HT2C receptor [6], may inhibit dopamine release [7] thus leading to attenuation of the beneficial effect of trazodone's 5-HT2A antagonistic activity on akathisia. In view of our clinical observation of the immediate and potent beneficial effect of mianserin, it may be possible that serotonin antagonist treatment may be efficacious, safe, and well tolerated for this chronic and difficult to treat condition.

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https://doi.org/10.1016/j.eurpsy.2003.12.008 Published online by Cambridge University Press

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Received 4 May 2003; received in revised form 5 December 2003; accepted 19 December 2003

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doi:10.1016/j.eurpsy.2003.12.008

Naso-gastric administration of risperidone to treat delusions of poisoning

Dear sir,

M was 15 years old when he was referred to the Child and Adolescent Service in a condition thought to be life-threatening. This case is interesting in its administration of antipsychotic medication and because there is evidence that both cannabis abuse [1] and rapid weight loss [2] are associated with mental disorders.

M had been a regular cannabis user for 2 years. His behaviour changed a year ago when he became socially withdrawn. He stopped going to school, believing that his classmates were making fun of him and because he had difficulty concentrating. He had mentioned suicide, and neglected himself. He slept little and would lie on the floor, screaming and crying in strange voices. M had lost weight; he had consumed almost nothing over the past three weeks and, afraid of being poisoned, would only sip water if his brother first drank from the cup.

M moved to England at the age of 10 and settled without major problems. His parents divorced 9 years ago. His mother suffered from depression; his father had previously experienced a psychotic episode and depression, and been addicted to cannabis and heroin; and his brother had used cannabis.

On presentation, M was unkempt, pale and thin; weakness made walking difficult. He spoke few words in a whisper, and there was evidence of thought interference as he found it difficult to talk and unable to elaborate. M was diagnosed with unspecified non-organic psychosis (ICD 10; F29).

Due to his fragility, M was admitted to a general medical ward for immediate rehydration. He was prescribed olanzapine 2.5 mg increasing to 5 mg the next day, but he refused to swallow tablets, and was given an injection of haloperidol 5 mg. The day after, M showed severe rigidity. Haloperidol was discontinued, and olanzapine 5 mg daily (Velotab®) and sertraline 50 mg daily were started. Non-compliance with medication and psychosis persisted so, when naso-gastric feeding was begun on his fifth day of hospital stay, all medication was stopped and risperidone liquid 1.5 mg daily was administered via the naso-gastric tube.

Improvement was rapid. Over the next couple of days he started to eat, drink, talk and walk. He was transferred to a psychiatric ward and discharged home on risperidone liquid 2 mg daily after 2 weeks. He remained stable until he used cannabis when drinking beer. This caused some deterioration in mental state, so risperidone was increased to 3 mg daily and he was cautioned against cannabis use. Six months on, M is stable; he eats and drinks normally, attends to his personal hygiene and participates in family activities.

There is evidence to suggest an association between psychosis and cannabis abuse [3]. Indeed, there is increasing evidence that the cannabinoid system may be involved in psychotic disorders [4,5]. Our patient supports others [1] in that the use of cannabis (i) precipitates psychotic disorders in vulnerable individuals (i.e. patients at high risk, e.g. family history), and (ii) exacerbates symptoms of psychosis in those that continue to use cannabis.

We believe this to be the first report of risperidone administration through a naso-gastric tube. It was appropriate in this patient who refused to swallow, afraid of being poisoned. Psychiatric disturbance has also been associated with considerable and rapid weight loss though diet, and return to a normal diet and weight has resulted in an improvement of condition [2]. Although some improvement may have been due to eating and subsequent weight gain, it was risperidone treatment that was considered to play an important role in the rehabilitation of this patient.

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