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Case Report

Fluoxetine induced bradycardia in presenile dementia

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We recently treated a 65 year old man with a 5 year history of progressive Alzheimer's Disease who developed persistent bradycardia whilst receiving a course of fluoxetine 20 mg daily for an episode of depression. He had no history or evidence of cardiac disease. The patient was admitted for in-patient assessment of withdrawn behaviour and weepiness in October 1994. Cardiovascular examination at that time was normal with a pulse rate of 84 beats per minute and sinus rhythm. A diagnosis of depression was made and fluoxetine was started at a dose of 20 mg daily. His mood improved and he was discharged to a community EMI residential facility at the end of November 1994. Pulse rates had been recorded to be 80-84 beats per minute at various times prior to the commencement of fluoxetine.

His mood remained settled. In June 1995 he was noted to be persistently drowsy with a pulse rate of 53 beats per minute. This was investigated in the local District General Hospital where his level of consciousness was found to be normal, the bradycardia of 53 beats per minute confirmed with otherwise normal ECG, and no cause for the bradycardia described. He was noted to have low pulse rates in full consciousness over the next months. We readmitted him to the dementia assessment unit, Holywell Hospital, for further investigations in September 1995.

Cardiovascular examination was again normal except for a pulse rate of 41 beats per minute, confirmed as sinus rhythm on ECG. Throughout the period he had been taking fluoxetine 20 mg mane and haloperidol 1.5 mg nocte under supervision. There was no evidence of hypotension throughout the period. We withdrew the haloperidol with no change in pulse rate. Fluoxetine was then discontinued on 13 September 1995. His radial pulse rate increased steadily to 76-8 beats per minute during the two weeks subsequent to discontinuation of fluoxetine, and his systolic blood pressure was consistently higher than 110 mm Hg.

The association was notified using the 'yellow card' system. Six months and one year later his resting pulse rate was 72 beats per minute. Neither fluoxetine nor any other antidepressant or neuroleptic has been reintroduced.

DISCUSSION

There have been case reports associating fluoxetine with bradycardia and syncope (Buff et al. 1991;[1] Ellison et al. 1990;[2] Feder 1991;[3] McAnally et al. 1992;[4] Hussein, 1994[5]). Fisch (1985)[6] in a retrospective ECG study showed that there was an association between fluoxetine and bradycardia but this effect was not noted in the elderly cohort. Many of the reported cases were receiving 40-80 mg of fluoxetine daily for treatment of depressive disorder and were noted to have had a history of heart disease or syncopal episodes. The mechanisms proposed in the literature relate to the effect of increased central nervous system serotonin on the medullary regulation of cardiovascular function (Ellison J M, 1990[2]) and to the reported direct inhibition of oxidative metabolism by fluoxetine which may permit drug interaction (McAnally L E, 1992[4]).

The case we report was treated with 20 mg fluoxetine throughout his episode. He had no history or evidence of cardiac disease, and at no time was he hypotensive or subject to syncope. His pulse rate increased 12-14 days after discontinuation of the drug suggesting a direct association.

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We have performed a literature search regarding the possible role of haloperidol in the bradycardia but no such association has been reported.

Fluoxetine should be considered a possible cause of sinus bradycardia in the elderly in the absence of cardiac disease and when given in normal doses.

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