Dear Editor

Antidepressants that alter brain 5-hydroxytryptamine (5-HT) levels are known to be associated with the potentially fatal serotonin syndrome (Sternbach, 1991). A case of probable serotonin syndrome is described following the use of trazodone alone.

Case report

A 73-year-old married man was referred to the old age psychiatry service following a transurethral resection of the prostate (TURP). Following the prescription of single doses of temazepam 20 mg and chlorpromazine 25 mg, he was readmitted to hospital following frequent falls, where no psychotropic medication was administered. A psychiatric assessment was carried out 2 weeks after the TURP, when a diagnosis of mixed anxiety/depressive disorder with coexisting obsessive-compulsive disorder (OCD) was made. A neurological examination revealed a rigid/stooped gait and poverty of facial expression, but no upper limb cogwheel rigidity, tremor or bradykinesia. Trazodone was started at a dose of 50 mg and increased to 75 mg a week later.

Three weeks after initial psychiatric assessment, he was admitted to a medical ward following sudden collapse with muscular rigidity. He was conscious but mute, pyrexial, rigid, hyperreflexic with myoclonic jerking, shivering, and had a pronounced resting tremor. A routine blood screen, electrocardiogram and chest X-ray were all within normal limits. A CT scan showed mild cortical atrophy and no evidence of infarct or space-occupying lesion.

A neurological opinion was sought and a diagnosis of serotonin syndrome made. Trazodone was stopped on admission, leading to a resolution of tremor and rigidity but the persistence of a Parkinsonian gait. He also began to exhibit frequent falls, an increasingly festinating gait, fluctuating disorientation and visual hallucinations. Further investigations failed to clarify the causation of this clinical picture.

Unfortunately, he died suddenly after a month of hospitalization, following the aspiration of vomitus; a postmortem revealed small bowel obstruction secondary to ileal adhesions. No histopathological studies were performed on the brain at postmortem.

Two particular aspects of the case are noteworthy: firstly, the presence of OCD may have played a part in the evolution of drug toxicity, in the brain 5-HT function is known to be altered in this condition (Lucey, 1994); secondly, the abnormalities of gait/facial expression (predating the administration of trazodone), excessive neuroleptic sensitivity and the development of fluctuating disorientation, visual hallucinations and frequent falls following the discontinuation of trazodone raise the possibility of Lewy body dementia. This remains tentative in the absence of neuropathological confirmation, but raises the possibility of other central neurotransmitter dysfunction.

Drugs commonly implicated in serotonin syndrome have been combinations of monoamine oxidase inhibitors (MAOIs) and L-tryptophan, although there have also been single case reports of its occurrence with trazodone in combination with buspirone (Goldberg and Huk, 1992), paroxetine (Reeves and Bullen, 1995) and isocarboxazid (Bodner et al., 1995). There appear to be no previous reports of serotonin syndrome associated with the use of trazodone alone; this may call for increased vigilance when using this drug in older people.

Rahul Rao
Heathside Community Team for Older People, Maidstone, Kent

Address for correspondence: Dr R. Rao, Ward ALI, Maudsley Hospital, Denmark Hill, London SE9 2HF, UK.

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REFERENCES


