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Case Report

A case of malignant syndrome induced by triiodothyronine (T3) in a patient with refractory depression

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Introduction

Neuroleptic malignant syndrome (NMS) is an idiosyncratic, potentially fetal reaction to neuroleptics with an estimated incidence of 1.4% (Pope et al. 1986). It usually develops while the patient is on neuroleptics, however, recent reports indicated that it is associated with the use of variety of psychotropic medication but neuroleptics. This report describes a case of NMS induced by triiodothyronine (T3) which was prescribed as an augmentation therapy for a patient with refractory depression.

Case Report

Miss A is a Japanese 52 year-old married woman who had her second depressive episode. She was diagnosed as major depression according to DSM-IV (APA). She was treated with amoxapine 150mg daily, trazodone 75mg daily, and lithium carbonate 900mg daily, but her symptoms did not improve. She was admitted to a psychiatric ward at August 16. At the time of admission, she showed severe depressive mood and psychomotor retardation, however, intensive physical and neurological examination revealed no abnormal findings. Thyroid function was normal.

She was started on amoxapine 150mg, lithium carbonate 400mg, and T3 25μg. On August 21, four days later, she showed disturbance of consciousness and developed a temperature of 40.5 C with a elevated creatine kinase (CPK) (1167 IU/l) and BUN (31.2mg/l). Physical examination was normal. A brain CT scan and cerebrospinal fluid examination revealed no abnormal findings, while an electroencephalogram showed a generalized slowing of background activity. NMS was suspected, and all the oral drugs was immediately discontinued and an intravenous drip injection was administered. On day 24 she showed muscular rigidity of the limbs with mild cogwheel phenomenon. The deep tendon reflexes were present bilaterally and symmetrical, no pathological reflexes being detectable. A peak CPK of 123,221 IU/l was reached on August 25. Bromocriptine (7.5–15mg) was given and dantrolene sodium (40mg) was added. Her level of consciousness and muscular rigidity improved gradually, however, the level of CPK kept elevated for approximately a week.

Discussion

The clinical features of this patient would fulfill the criteria for NMS as set out by Caroff (1991). At the time of occurrence of NMA, the patient was receiving T3 in addition of amoxapine and lithium carbonate. Since T3 was started only four days before the onset, this drug might be responsible in this case. It is established that T3 has an effect to potentate tricyclic antidepressants. To our knowledge, there has been no report that examined the association of NMS and T3. There have been some reports that found association between NMS and lithium or amoxapine therapy. Susman and Addonizio (1987) reported a case of reinduction of NMS with lithium. There have also been reports of a possible association of lithium to NMS in patients with

Parkinson's disease (Pfeiffer and Suscha 1985). Tricyclic antidepressants can rarely cause NMS. Fava and Galizia (1995) reported a case of NMS that occurred in a patient on amitriptyline and lithium carbonate. Although the association between amoxanpine and/or lithium carbonate and NMS cannot be fully denied, T3 seemed to be most responsible for the occurrence of NMS in this case.

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