

Conditions Associated with an Increased BB Band of CPK Isoenzymes in Serum

Condition	Reference
Small nonhemorrhagic stroke	This paper
Massive cerebral infarction	3
Cerebral hematoma, myocardial infarction	3
Malignant hyperthermia	6
Uremia	5
Brain anoxia after cardiac arrest	3
Necrosis of large intestine	3
Reye syndrome	5

stroke in the face of a positive BB fraction of the CPK isoenzymes.

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Hydroxyzine-Associated Tardive Dyskinesia

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Tardive dyskinesia from antihistamines is uncommon and typically follows years of use [1, 5]. We report the appearance of tardive dyskinesia after 7½ months of hydroxyzine therapy.

In June, 1981, a 74-year-old woman was hospitalized for

evaluation of her continual head rolling and lip licking. Athetoid tongue movements had first appeared in May while she was receiving doxepin, 100 mg at bedtime, and hydroxyzine, 50 mg twice daily. Her most recent medication change had been the addition of hydroxyzine the previous September. Her medical history included arthritis, lymphatic lymphoma, depression, arteriosclerotic cerebrovascular disease, and peptic ulcer disease. She had no history of dementia. Routine laboratory studies were normal except for a microcytic anemia resulting from her lymphoma.

She had used phenothiazine derivatives intermittently for several years to control nausea; however, she had not taken any phenothiazines for 12 months. Doxepin withdrawal five days prior to hospitalization did not affect her dyskinesias. Hydroxyzine was discontinued upon admission. Six months later her dyskinesias persisted but were diminished by chlorazepate, 15 mg at bedtime.

In all reports of antidepressant-induced dyskinesias, recent or concomitant neuroleptic therapy has been a contributing factor [2, 3]. Antidepressant-induced dyskinesias disappear within one to two weeks after the challenge is removed. Concomitant neuroleptic therapy has not been a consistent finding in antihistamine-associated dyskinesias, which typically appear after years of chronic use and persist despite drug withdrawal [1, 5]. Kobayashi implied, by reference to animal studies, that patients with prior neuroleptic use may later develop dyskinesias when exposed to drugs from other chemical classes [4]. In animals, this period of dopaminergic hypersensitivity is variable.

The persistence of dyskinesias in our patient led us to believe that the ingestion of hydroxyzine provoked dyskinesias after her prior sensitization from phenothiazine exposure. We wish to alert clinicians to possible tardive dyskinesia from short-term hydroxyzine, especially in patients with known phenothiazine use.

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