

# VENLAFAXINE INDUCED-MYOCLONUS IN A PATIENT WITH MIXED DEMENTIA

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Myoclonus is a clinical sign defined as sudden, brief, shock like, involuntary movements caused by muscular contractions or inhibitions<sup>1</sup>. Certain commonly prescribed drugs cause movements disorders as myoclonic jerks and polymyoclonus in the absence of other signs of toxicity<sup>2</sup>. Venlafaxine is a serotonin-norepinephrine reuptake inhibitor used for the treatment of depression, post traumatic stress disorder, obsessive compulsive disorders, chronic pain and menopausal symptoms<sup>3,4</sup>.

We report a case of myoclonus induced by venlafaxine in a patient with mixed dementia.

## CASE

A seventy-five-year-old female presented to neurologic consultation at emergency department complaining of tremors and abnormal movements that started 4 days before the admission. It started in the four limbs and face at the same time, and according to her daughter, they were shock like, abrupt and nonrhythmic. The patient denied any similar events before. The movements could not be suppressed. Her medical history showed 3 minor strokes, but no sequelae, hypertension and a mixed dementia which was diagnosed 3 years before. She needed some help for common activities such as bathing and taking medications due memory problems. For the treatment of dementia she was receiving galantamine 8 mg twice a day and risperidone 1mg per day for almost 10 months. She also received enalapril 20 mg and aspirin 100 mg per day. Twelve days before the admission, her physician prescribed venlafaxine 75 mg per day for depression. She denied other medications, fever or cough.

Physical examination showed an arterial pressure of 130 per 80 mmHg and regular cardiac rhythm. She was alert and cooperative. There was no face rubor. Myoclonus was present in inferior limbs, superior limbs and face. There were no signs of parkinsonism. Motor strength, sensory functions and language were preserved but she presented snout and palmomentonian reflexes.

Her Mini Mental was 13. Blood count, blood chemistry including glucose, sodium, potassium and calcium levels was normal. Urinalysis excluded infection. We also performed a CT scan which did not show evidence of stroke.

We decided to stop venlafaxine and after 3 days the myoclonus ceased.

## DISCUSSION

It is well described in the current literature the association of myoclonus with anticonvulsants, antidepressants, antipsychotic, cephalosporin and carbapenem antibiotics<sup>2,4,5</sup>. Although our patient was receiving risperidone, we believe that myoclonus was induced by venlafaxine, once there was a complete remission after discontinuation of the drug. Venlafaxine was associated with other movements disorders as blepharospasms<sup>2</sup> and myoclonus in the setting of serotonin syndrome, which is a potentially fatal complication of the combined use of agents that enhance serotonin activity<sup>3</sup>, but we couldn't find any report of myoclonus and venlafaxine without serotonin syndrome.

Degenerative diseases might present with myoclonus, but the myoclonus seen in some of these disorders has been primarily limited to small-amplitude jerks of the distal limbs (predominantly fingers), termed minipolymyoclonus. Our patient presented a more generalized and prominent myoclonus, that should raise the possibility of drug-induced myoclonus<sup>6</sup>.

We believe that myoclonus was induced by the serotonergic effects of venlafaxine. The relation between serotonin and myoclonus was first documented by Lhermitte<sup>7</sup>. There are several reports of myoclonus and other movements disorders induced by selective inhibitors of serotonin<sup>2-4,8-10</sup>. In one of them, Lauterbach reported myoclonus induced by fluoxetine in a patient with Pick disease<sup>8</sup> and postulated that might be an underlying hypersensitivity of 5HT<sub>1A</sub> receptors in Pick's disease. Recently upregulation of 5HT<sub>1A</sub> receptors in hippocampus in mild cognitive impairment was described, as well as reduction in the number of receptors 5HT<sub>1A</sub> in the hippocampus of patients with Alzheimer disease<sup>9,11,12</sup>. Thus, it is possible that a dysfunction in the serotonin pathway exists in degenerative diseases, which predisposes to serotonin inhibitors-

## MIOCLONIAS INDUZIDAS POR VENLAFAXINA EM UMA PACIENTE COM DEMÊNCIA MISTA

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induced movement disorders. This is in accordance with a review of movement disorders and serotonin reuptake inhibitors conducted by Gerber and Lynd, who concluded that patients at higher risk may be those with preexisting neurologic disorders and the elderly. Also, the combined use of neuroleptic and serotonin reuptake inhibitors increases the susceptibility of movement disorders<sup>10</sup>.

We conclude that older patients, especially those presenting degenerative diseases are at risk for movement disorders when serotonin reuptake inhibitors are prescribed. One should carefully evaluate patients with degenerative disease in use of venlafaxine and other serotonin reuptake inhibitors.

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