

Negative Myoclonus Induced by Ciprofloxacin

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Abstract

Background: Negative myoclonus is characterized by a brief sudden loss of muscle activity, and can be caused by a variety of acquired factors and epilepsy syndromes.

Phenomenology Shown: We show a clear video example of a patient with an extensive negative myoclonus that was induced by ciprofloxacin.

Educational Value: Several neurotoxic effects have been associated with the use of ciprofloxacin, but negative myoclonus has not been reported previously.

Keywords: Negative myoclonus, ciprofloxacin, adverse reaction

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Ethics Statement: All patients that appear on video have provided written informed consent; authorization for the videotaping and publication of the videotape was provided.

An 84-year-old patient with a history of Parkinson's disease was admitted to the cardiology ward because of chest pain. The day after admission, the patient complained of shaking limbs, for which the neurologist was consulted. Neurologic examination showed a pronounced negative myoclonus (Video 1). Laboratory examination including electrolytes, liver and renal function, thyroid function, glucose, and arterial blood gas was performed, showing no abnormalities. The patient had been using ciprofloxacin 500 mg twice a day for 5 days for a urinary tract infection. The ciprofloxacin was discontinued after which the negative myoclonus disappeared within 1 day.

Negative myoclonus is characterized by a brief sudden loss of muscle activity in agonist muscles followed by a compensatory jerk of the antagonistic muscles, appearing as involuntary jerky movements.¹ Negative myoclonus may be of cortical or subcortical origin. Cortical negative myoclonus may be caused by epilepsy syndromes and a variety of acquired factors, such as focal brain lesions. Subcortical myoclonus occurs mainly in toxic-metabolic encephalopathy, caused by electrolyte disturbances, liver and respiratory failure or several drugs.² To our knowledge, negative myoclonus has not been

previously described in association with the use of ciprofloxacin. However, several neurotoxic effects have been associated with the use of quinolones, including seizures, extrapyramidal manifestations, and positive myoclonus.³ The hypothesized mechanism of the neurotoxicity is inhibition of gamma-aminobutyric acid (GABA-A) receptors and activation of excitatory N-methyl-D-aspartate (NMDA) receptors, leading to a toxic encephalopathy. Patients with prior central nervous system disease, such as Parkinson's disease, impaired renal function, and advanced age may be particularly vulnerable.³

In patients with negative myoclonus, laboratory examination and medication evaluation should be performed to identify the underlying cause. The differential diagnosis of negative myoclonus includes positive myoclonus and tremor.¹ Electrophysiology can assist with determining this; however, this was not performed in our patient. The treatment includes correction of electrolyte disturbances or, like in our case, cessation of the provocative drug. If the negative myoclonus persists, neuro-imaging and/or emergent EEG may be useful to analyze other causes. If necessary, benzodiazepines such as clonazepam or anti-epileptic drugs can be administered to suppress the symptoms.¹



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Video 1. Demonstration of negative myoclonus in our patient. This video shows negative myoclonus of the patient's arms (mostly) and legs. First, the patient is asked to stretch his arms forward. Involuntary jerky movements are observed caused by a sudden, brief interruption of muscle activity. When the patient is asked to raise his arms higher, this can be seen more clearly. Secondly, the patient is asked to put his arms down, after which the involuntary jerky movements disappear. Next, the patient is asked to raise his left leg, in which the involuntary jerky movements can be seen. After putting his left leg down, it can no longer be observed. The same can be observed when raising the right leg. When the patient's arms and legs are at rest, no myoclonus is observed.