Case Report

Myoclonic Jerks, Exposure to Many Cats, and Neurotoxoplasmosis in an Immunocompetent Male

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Abstract

Background: Myoclonic jerks are due to sudden, brief, involuntary muscle contractions, positive myoclonus, or brief cessation of ongoing muscular activity, negative myoclonus, and may be difficult to recognize.

Case Report: We describe an immunocompetent, adult, male patient with sleep-related, multifocal, myoclonic jerks and neurotoxoplasmosis with abnormal cerebrospinal fluid but normal brain imaging. There was complete resolution of the myoclonus with antitoxoplasmosis therapy after 1 week, and no relapse after 1 year.

Discussion: Neurotoxoplasmosis may be subtle in presentation, difficult to diagnose, and more common than realized, and it is being increasingly implicated in epileptogenesis in humans.

Keywords: Myoclonus, movement disorder, toxoplasmosis, epilepsy

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Introduction

Neurotoxoplasmosis can induce a wide variety of movement disorders including myoclonic jerks especially in immunodeficient individuals.1 We report a case illustrating sleep-related, multifocal, myoclonic jerks in neurotoxoplasmosis in an immunocompetent adult that responded adequately to treatment.

Toxoplasmosis is also a ubiquitous infectious illness, which has been considered a global threat. Neurotoxoplasmosis is additionally being increasingly recognized as a cause of cryptogenic epilepsy and this case provides further credence to this hypothesis.1

By this report, we highlight myoclonus as a manifestation of neurotoxoplasmosis even in the apparently immunocompetent patient, thereby promoting earlier and widespread recognition and consequently appropriate treatment.

Case Report

We describe a case of multifocal, myoclonic jerking during sleep [Video 1] in encephalitis as a result of neurotoxoplasmosis in a 39-year-old immunocompetent adult male who had daily contact with 20 resident cats for 2 years. The patient presented to hospital with a flu-like syndrome for 3 days and with multifocal, sleep-related, myoclonic jerks for 1 day. These movements consisted of abnormal, sudden, isolated, brief, small-amplitude, multifocal muscle jerks involving various body areas, in particular the patient’s head, right upper limb, fingers, and legs. The phenomenon was observed only during sleep without causing incontinence or arousal and its occurrence was not noted during wakefulness. The myoclonus would start in the first hour of sleep and remained unchanged throughout sleep during the day or at night. We found a pattern of three to five sequences of muscle contractions...
per minute, each lasting 2–3 minutes followed by a period of non-observable phenomena of 20–30 minutes' duration. These movements occurred four to six times daily for 7 days. The patient was always unaware of those events that subsided spontaneously without benzodiazepines.

There was no history of previous illness, surgery, exposure to chemicals, use of recreational drugs, consumption of alcohol, or recent travel abroad. He was febrile (38.8°C), tachycardic (110 beats/minute) with blood pressure of 145/82 mmHg and a respiratory rate of 20 breaths per minute, and he had 100% oxygen saturation on room air. Two, non-tender, soft lymph nodes, 1.2 cm in diameter, were palpable in the cervical region.

There was leukocytosis (13.8 × 10⁹/L) with eosinophilia (14.1%) and elevated C-reactive protein at 31.1 mg/dL. Immunoglobulin (Ig) G antibodies to *Toxoplasma gondii* were detected in the serum and cerebrospinal fluid (CSF) with an elevated titer of 198 and 20 IU/mL respectively by using an electro-chemiluminescence immunoassay. Serum and CSF IgM antibodies to *T. gondii* were also positive. The CSF contained 47 cells/mm³ and 0.8 g/L protein, both being elevated values. An enzyme-linked immunosorbent assay for human immunodeficiency virus infection (HIV) was non-reactive in serum and polymerase chain reaction testing was unavailable. Magnetic resonance imaging of the brain and spinal cord and scalp electroencephalography (EEG) were normal. The patient had an excellent outcome after specific treatment for toxoplasmosis (trimethoprim–sulfamethoxazole) and the myoclonic jerking disappeared completely in 7 days. The investigations and treatment of the patient are illustrated in Tables 1 and 2. He remained healthy at the 1-year follow-up.

**Discussion**

Central nervous system (CNS) toxoplasmosis can cause multiple movement disorders and seizures including multifocal myoclonic jerks, even with normal EEG and magnetic resonance imaging/magnetic resonance angiography scans of the brain.¹⁻³ Myoclonic jerks because of neurotoxoplasmosis in immunocompetent individuals are rare and we found no other similar reports in the literature. Recent studies have also implicated chronic or latent toxoplasmosis as a possible cause of cryptogenic seizures and/or epilepsy in the immunocompetent individual, which gives added relevance to this case.¹ We suggest that mild clinical expression of disease and normal imaging studies in this case were due to immunocompetence in the patient.

Atypical toxoplasmosis encephalitis with limited clinical expression and with normal conventional imaging may also demonstrate multifocal myoclonic jerking in an immunocompetent host and be due to atypical genotypes of *T. gondii*. Reinfection with toxoplasmosis has been reported among immunocompetent human hosts. Reinfection with toxoplasmosis can occur among people chronically and with heavy exposure to *T. gondii* such as in our patient. Owing to the severity of encephalitis caused by these atypical genotypes that can cause latent or subclinical infections worldwide, suspicion, early diagnosis, and appropriate treatment are essential even in settings with limited resources.¹⁻³

Multifocal myoclonic jerks are characterized by sudden, isolated, arrhythmic, asynchronous and asymmetric involuntary brief twitches, and jerks of muscles or muscle fibers involving various body areas,
### Table 1. Medical Investigations

| Tests Performed on Admission                  | Result                  | Reference Range            |
|-----------------------------------------------|-------------------------|----------------------------|
| **Blood test**                                |                         |                            |
| WBCs                                          | $13.8 \times 10^9$/L    | $4.5–11.0 \times 10^9$/L   |
| Eosinophils                                   | 14.1%                   | 0.0–0.6%                   |
| Hemoglobin                                    | 15.2 g/dL               | 14.0–17.5 g/dL             |
| Mean corpuscular volume                       | 83.2 fl/L/red cell      | 80–96 fl/L/red cell        |
| Platelet count                                | $350 \times 10^3$/µL    | 156–373 $\times 10^3$/µL   |
| Serum potassium                               | 4.1 mmol/L              | 3.5–5.1 mmol/L             |
| Serum sodium                                  | 138 mmol/L              | 135–145 mmol/L             |
| Serum creatinine                              | 0.8 mg/dL               | 0.5–1.2 mg/dL              |
| BUN                                           | 11 mg/dL                | 3–20 mg/dL                 |
| Uric acid                                     | 4.5 mg/dL               | 2.5–8 mg/dL                |
| Alanine aminotransferase                      | 60 IU/L                 | 20–60 IU/L                 |
| Aspartase aminotransferase                    | 40 IU/L                 | 5–40 IU/L                  |
| Gamma glutamyl transpeptidase                 | 60 U/L                  | 8–61 IU/L                  |
| Lactate dehydrogenase                         | 330 IU/L                | 105–333 IU/L               |
| Alkaline phosphatase                          | 129 U/L                 | 40–129 IU/L                |
| Albumin                                       | 4.9 g/dL                | 3.5–5.5 g/dL               |
| Albumin-corrected calcium                     | 9.6 mg/dL               | 9.6–11.2 mg/dL             |
| CRP                                           | 31.1 mg/dL              | 0.0–1.0 mg/dL              |
| Fasting blood sugar                           | 80 mg/dL                | 60–120 mg/dL               |
| VDRL test                                     | Non-reactive            | Non-reactive or reactive   |
| FTA-ABS                                       | Negative                | Positive or negative       |
| Elisa for HIV                                  | Non-reactive            | Non-reactive or reactive   |
| Antistreptolysin O titer                      | 90 IU/mL                | 0–200 IU/mL                |
| *Toxoplasma gondii* IgG antibodies            | 198 IU/mL               | Positive: greater than 1.09 IU/mL |
| *Toxoplasma gondii* IgM antibodies            | Positive                | Positive or negative       |
| *Toxoplasma gondii* specific IgG avidity      | High avidity (AI > 50%) | Low avidity (AI ≤ 50%)     |
| *High avidity (AI > 50%)*                     |                         | High avidity (AI > 50%)    |
| Herpes virus 1 IgG antibodies                 | Less than 0.9           | Index negative: Less than 0.9 |
| Herpes virus 1 IgM antibodies                 | Less than 0.9           | Index negative: less than 0.9 |
| Herpes virus 2 IgG antibodies                 | Less than 0.9           | Index negative: Less than 0.9 |
**Table 1.** Continued

| Tests Performed on Admission                              | Result     | Reference Range                          |
|-----------------------------------------------------------|------------|------------------------------------------|
| Herpes virus 2 IgM antibodies                             | Less than 0.9 | Index negative: less than 0.9           |
| CMV IgG antibodies                                        | 0.800 UA/mL | Negative: less than 1.5 UA/mL           |
| CMV IgM antibodies                                        | 0.778 UA/mL | Negative: less than 1.1 UA/mL           |
| EBV IgG antibodies                                        | 3.3        | Positive: Greater than 22               |
| EBV IgM antibodies                                        | 0.1        | Negative: less than 0.8                 |
| Hepatitis BsAG                                            | Negative   | Positive or negative                    |
| Hepatitis C IgG antibodies                                | Negative   | Positive or negative                    |
| Hepatitis C IgM antibodies                                | Negative   | Positive or negative                    |
| *Echinococcus granulosus* IgG antibody                    | Negative   | Positive or negative                    |
| Anti-double stranded DNA                                  | Negative   | Positive or negative                    |
| Antinuclear antibody                                      | Negative   | Positive or negative                    |
| Perinuclear antineutrophil cytoplasmic antibodies         | 5.42 U/mL  | Negative: less than 10.0 U/mL           |
| Cytoplasmic antineutrophil cytoplasmic antibodies         | 3.73 U/mL  | Negative: Less than 10.0 U/mL           |
| PCR for viral infections or toxoplasmosis                 | Tests not obtained | Negative or positive |

**Other investigations**

| Test                                      | Result                     | Reference Range                      |
|-------------------------------------------|----------------------------|--------------------------------------|
| Mantoux test                              | Negative                   | Positive or negative                 |
| Electrocardiogram                         | Sinus tachycardia          | Normal or abnormal                   |
| Chest X-ray                               | Normal                     | Normal or abnormal                   |
| Echocardiogram                            | Normal ejection fraction 75% | Normal or abnormal                   |
| CT scan of the brain with contrast        | Normal                     | Normal or abnormal                   |
| MRI/MRA scans of the brain               | Normal                     | Normal or abnormal                   |
| CSF analysis                              | CSF opening pressure was 14 cm of H₂O. CSF contained 47 cells/mm³, 0.8 g/L of proteins, and the glucose concentration was 60 mg/dL. CSF culture showed no bacterial growth and cytology was negative for neoplastic cells. VDRL was non-reactive and India ink test for *Cryptococcus neoformans* was negative |  |
| *Toxoplasma gondii* IgG antibodies in CSF | 20 IU/mL                   | Positive: greater than 1.09 IU/mL    |
| *Toxoplasma gondii* IgM antibodies in CSF | Positive                  | Positive or negative                 |
in particular the corners of the mouth, fingers, toes, limbs, several limbs, or a combination of limbs plus face, palate, head, jaw, neck, tongue, eyes, or trunk. Myoclonic jerks may have their origins at different levels of the nervous system and have many causes, and specialized tests such as video EEG, electromyography, and polysomnography are often necessary to ascertain its precise origin and accurate classification.3

Myoclonus may be cortical, subcortical, spinal, or peripheral in origin. Myoclonic jerking that is fleeting at the onset of sleep, or hypnic myoclonus, is considered physiological. It can be multifocal but seldom

### Table 1. **Continued**

| Tests Performed on Admission | Result | Reference Range |
|------------------------------|--------|----------------|
| **Tests performed in the follow-up blood test** | | |
| *Toxoplasma gondii* IgG antibodies | 20 IU/mL | Positive: greater than 1.09 IU/mL |
| *Toxoplasma gondii* IgM antibodies | Positive | Positive or negative |
| PCR for viral infections or toxoplasmosis | Tests not obtained | Negative or positive |
| **Other investigations** | | |
| Scalp EEG | Normal | Normal or abnormal |
| EMG and nerve conduction studies | Normal | Normal or abnormal |
| PCR for viral infections or toxoplasmosis in CSF | Tests not obtained | Negative or positive |
| Video-EEG, polysomnography and jerk locked backed averaging studies | Tests not obtained | Normal or abnormal |

Abbreviations: BUN, Blood Urea Nitrogen; BsAG, B surface antigen; CMV, Cytomegalovirus; CRP, C-reactive Protein; CSF, Cerebrospinal Fluid; CT, Computed Tomography; DNA, Deoxyribonucleic Acid; EBV, Epstein–Barr Virus; EEG, Electroencephalogram; ELISA, Enzyme-linked Immunoassay; EMG, Electromyography; FT-ABS, Fluorescent Treponema Pallidum Antibody Absorption; HIV, Human Immunodeficiency Virus; Ig, Immunoglobulin; MRI/MRA, Magnetic Resonance Imaging/Magnetic Resonance Angiography; PCR, Polymerase Chain Reaction; VDRL, Venereal Disease Research Laboratory; WBC, White Blood Cell.

### Table 2. **Medical Treatment**

| Dosage | Period of Treatment |
|--------|---------------------|
| **Intravenous therapy initiated on admission day** | | |
| Trimethoprim–sulfamethoxazole 160 mg/800 mg 3 times daily | 2 weeks |
| Normal saline isotonic solution 1 L daily | 1 week |
| Pantoprazole 40 mg twice daily | 3 days |
| **Oral drugs initiated on admission day** | | |
| Carbamazepine 200 mg 2 times daily | 6 days |
| Paracetamol 1 g three times daily | 7 days |
| **Other oral drugs** | | |
| Trimethoprim–sulfamethoxazole 80 mg/400 mg per tablet 2 tablets 2 times daily | 4 weeks initiated on day 15 |
| Pantoprazole 40 mg once daily | 14 days initiated on day 4 |
Benign myoclonus can occur in healthy individuals and is most commonly caused by muscle contractions or during the induction of general anesthesia with intravenous etomidate and propofol or in benign fasciculation syndrome. 3 Epilepsy syndromes and a variety of acquired factors, such as focal brain lesions, may cause cortical myoclonus. Subcortical myoclonus occurs mainly in toxic–metabolic encephalopathy, electrolyte disturbances, liver and respiratory failure, or as a reaction to several drugs. In cortical myoclonus, the electroencephalogram may be normal as seen in our patient, and this point was recently emphasized in a description of myoclonus in Wilson’s disease. 2 Also, typical classification of physiological, hypnic, or hypnagogic jerks describes generalized jerks noted on falling asleep. The jerks observed in our patient presented within the first hour after sleep and they were multifocal. 3

The differential diagnosis of myoclonus includes negative myoclonus, tremor, opsoclonus myoclonus syndrome, Creutzfeldt–Jacob disease, 1–3 Clinical, video-imaging, laboratory and serological examination, radiological studies, and electrophysiological analysis should be performed to identify the underlying cause; however, electrophysiology was not performed in our patient. Analysis of the differential diagnoses showed no other disease that could explain our patient’s reversible encephalopathy (Table 3).

We implemented Bayesian inference, response to treatment, and long-term follow-up for accuracy in diagnosis and here raise awareness of this rare movement disorder associated with serological evidence of a ubiquitous pathogen, whose role in epileptogenesis is being interrogated.

The treatment of myoclonus includes correction of the underlying cause such as electrolyte disturbances or, as in our case, specific antitoxoplasmosis therapy. However, if necessary, benzodiazepines such as clonazepam or antiepileptic drugs can be administered to suppress the symptoms in some patients. 3

The hypothesized mechanism of the neurotoxicity in the pathogenesis of myoclonus is inhibition of gamma-aminobutyric acid receptors and activation of excitatory N-methyl-D-aspartate receptors, leading to a toxic encephalopathy. 1–5

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