Case Report

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# Seizures Induced Pleocytosis

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## Abstract

Cerebrospinal fluid pleocytosis has rarely been described as a possible transient finding following epileptic activity. We report a 68-year-old woman who presented with acute confusion and Cerebrospinal fluid pleocytosis. No evidence of an inflammatory, infectious or neoplastic cause explaining the pleocytosis was found. We presume that the pleocytosis in this patient was a result of epileptic convulsions. The mechanism for this finding is not fully understood.

**Keywords:** Seizure; Pleocytosis; Epilepsy

## Introduction

Cerebrospinal fluid (CSF) is normally acellular and up to 5 white blood cells (WBCs) are considered normal in adults when obtained by lumbar puncture (LP). An increase in the total number of CSF cells, referred to as pleocytosis, is mostly associated with infectious disorders of the CNS, neoplasms, or intracranial hemorrhage.

CSF pleocytosis may be a transient finding following epileptic activity, however to date only scarce reports of CSF pleocytosis associated with seizures alone have been reported [1-3]. Considering these reports it may be difficult to interpret CSF abnormalities following epileptic activity and determine whether they are due to the seizure or another underlying etiology. We present a 68-year-old woman with a history of schizophrenia and epilepsy, who presented to our emergency room due to acute confusion. Lumbar puncture tests revealed pleocytosis with predominant mononuclear cells.

## Case Report

A 68-year-old woman was admitted to our department due to an acute confusional state.

Earlier that day, she was found at her apartment naked and confused with difficulty to complete sentences. Her colleague reported that during the days prior to her admission she arrived at work as usual, showing no apparent symptoms.

The patient had a medical history of schizophrenia, epilepsy, diabetes mellitus type II, hypo-thyroidism, dyslipidemia, a prior event of thrombotic thrombocytopenic purpura and cerebral vein thrombosis. She lived alone and was able to perform daily activities independently. Her medications included Lamotrigine, Simvastatin, Bisoprolol, Aspirin, Paroxetine and Olanzapine. Approximately one month before her admission, her neurologist had increased the dosage of Lamotrigine (from 125 mg/day to 150 mg/day) due to sub-therapeutic serum levels.

On examination the patient reported feeling confused but was oriented to person, place, and time. Her temperature was 37.2°C, pulse was 109 beats/minute and blood pressure was 109/56 mm Hg. She had no obvious recollection of the last two days, with no clear memory of the events which brought her to hospital. She was not aware of any seizures during the days before admission and her last reported seizure was two years prior to her admission. The neurological examination was normal, with no signs of meningitis or focal deficits. Cardiac, respiratory and abdominal examination was normal as well.

Laboratory tests revealed leukocytosis and highly elevated creatine kinase level (CK) as shown in detail in **Table 1**.

**Table 1** Blood test result.

| Variable | Reference range | On admission | Day after admission |
|---|---|---|---|
| WBCs (K/microL) | 4-10.8 | 14.4 | 11 |
| Hemoglobin (g/dl) | 11.7-15.7 | 13.72 | 12.8 |
| Creatinine (mg/dl) | 0.51-0.95 | 1.52 | 0.8 |
| Urea (mg/dl) | 15-45 | 72 | 38 |
| Glucose (mg/dl) | 70-110 | 118 | 129 |
| Calcium (mg/dl) | 8.1-10.4 | 10.4 | 9.1 |
| Sodium (meq/l) | 136-148 | 138 | 147 |
| Potassium (meq/l) | 3.5-5.2 | 4 | 3.8 |
| Albumin (g/dl) | 3.6-5.5 | 4.7 | 4.1 |
| LDH (IU/l) | 100-260 | 480 | 379 |
| CK total (IU/l) | 0-170 | 8978 | 3997 |
| CRP (mg/l) | <0.08-5 | 23 | 26 |

| | | | |
|---|---|---|---|
| Lamotrigine serum (micg/ml) | 2-20 | 1.5 | - |
| Alcohol serum (mg/dl) | - | negative | - |
| WBC: White Blood Cell; LDH: Lactic Dehydrogenase; CK: Creatine Kinase; CRP- C re-active protein. | | | |

A computed tomography (CT) of the brain did not reveal any acute changes, apparent hemorrhage or space occupying lesions. Following the CT a lumbar puncture (LP) was performed obtaining non-purulent CSF with pleocytosis; the glucose and protein levels were 76 mg/dl and 34 mg/dl (normal 15-45 mg/dl) respectively. The cell count showed 42 cells mostly mononuclear cells.

Specimens of blood, urine and CSF were obtained for culture (**Table 2**) and empirical antibiotic treatment and Acyclovir was initiated.

**Table 2** Microbiology evaluation.

| CSF specimen | Results |
|---|---|
| Gram stain | Negative |
| Aerobic culture | Negative |
| Mycobacterium tuberculosis culture | Negative |
| Entero Virus PCR | Negative |
| HSV1, HSV2, VZV PCR | Negative |
| Cryptococcal antigen | Negative |
| Blood culture | Negative |
| Urine culture | Negative |
| PCR- polymerase chain reaction; HSV-Herpes simplex virus; VZV- varicella zoster virus. | |

An Electroencephalography performed the morning after admission demonstrated low amplitude beta activity over the frontal regions with no evidence of epileptic activity.

During her hospitalization she remained in generally good condition and her neurologic examination remained unchanged. Repeat laboratory tests showed normalization of kidney function and decline in CK levels. The treatment with antibiotics and acyclovir was discontinued following the negative results of the CSF and the patient was discharged on the second day of her hospitalization.

The day following her discharge, laboratory results of Lamotrigine serum levels obtained during first day of hospitalization showed sub-therapeutic levels of 1.5 micg/ml.

## Discussion

Our clinical diagnosis was that the confusional state presented by the patient was due to a seizure with postictal phase. Sub-therapeutic Lamotrigine blood levels revealed an obvious cause for recurrent seizures. In addition, the elevated CK levels seen on admission support the diagnosis of recent epileptic activity. As the patient was first evaluated in the emergency room, the exact ictal phenomena and the patient's activities during the preceding days were not well known.

Our clinical, radiographic and laboratory investigations, found no obvious infectious, neoplastic, inflammatory or traumatic cause to explain the presence of pleocytosis.

CSF pleocytosis produced by epileptic activity has been rarely described. Most of the evidence for postictal CSF abnormalities was derived from anecdotal reports or from a few case series of adult populations [1-3]. The reported frequency of seizure-induced CSF abnormalities varies among different studies but has been estimated to be as high as 30% [1]. Increased CSF leukocyte counts were found after single simple, complex partial, or generalized tonic-clonic seizures, while some of the studies have emphasized that pleocytosis is more common after repetitive generalized tonic-clonic seizures [2,3].

The mechanism inducing postictal pleocytosis is not fully understood. Petito et al. [4] described a transient disruption of the blood brain barrier (BBB) or release of chemotactic substances during experimental convulsions in animals.

It should be noted that in our patient another plausible cause for CSF pleocytosis was her psychiatric disease as we have found few reports suggesting CSF alterations due to neuropsychiatric disorders [5]. However these reports described pleocytosis mainly during episodes of acute psychosis or prior to antipsychotic treatment, while our patient's was well balanced under medical treatment.

In conclusion, although infectious causes of CSF pleocytosis always need to be excluded, it appears that seizures alone may also explain this phenomenon and this diagnosis should be considered when others have been ruled out.

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