



(CASE REPORT)



## ACUTE BILATERAL OPTIC NEURITIS ASSOCIATED WITH ENTEROVIRAL MENINGOENCEPHALITIS IN A SEVEN-YEAR-OLD CHILD: A CASE REPORT

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World Journal of Advanced Research and Reviews, 2026, 31(03), 1828–1833

Publication history: Received on 18 August 2026; revised on 24 September 2026; accepted on 26 September 2026

Article DOI: <https://doi.org/10.30574/wjarr.2026.31.3.2468>

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### ABSTRACT

Enteroviral central nervous system infections are common in children, but severe encephalitic presentations associated with optic nerve involvement are rare. We report a previously healthy seven-year-old girl admitted with a generalized seizure, severe impairment of consciousness, and acute complete bilateral visual loss. Cerebrospinal fluid contained fewer than 3 white blood cells/mm<sup>3</sup>, while multiplex polymerase chain reaction was positive for enterovirus. Brain magnetic resonance imaging supported an encephalitic process, and ophthalmological examination confirmed bilateral optic neuritis. The course was complicated by recurrent seizures, transient right hemiplegia, and severe hyponatremia with a nadir of 116 mmol/L. Urine output exceeded 2 L/24 h (>4.17 mL/kg/h at an initial weight of 20 kg), with marked thirst, hypernatruresis, hyperosmolar urine, and a 10% weight loss. Because fluid intake was not quantified and overt clinical hypovolemia was absent, cerebral salt wasting remained probable but unconfirmed, and differentiation from the syndrome of inappropriate antidiuresis was limited. Treatment included antiepileptic therapy, intravenous corticosteroids for three days followed by an oral taper, intravenous immunoglobulin at 2 g/kg over two days, tailored hydration, and sodium supplementation. Visual function recovered completely within approximately 12 days, and serum sodium reached 134 mmol/L at discharge. This case emphasizes that an acellular cerebrospinal fluid profile does not exclude enteroviral meningoencephalitis and illustrates a rare temporal association with bilateral optic neuritis.

**Keywords:** Pediatric Optic Neuritis; Enteroviral Meningoencephalitis; Bilateral Optic Neuritis; Severe Hyponatremia; Cerebral Salt Wasting; Pediatric Neuroinfection

### 1 INTRODUCTION

Meningoencephalitis involves inflammation of the meninges and brain parenchyma and is an important cause of neurological morbidity in children [1]. Enteroviruses are among the leading causes of viral meningitis in this population [2]. Although most enteroviral central nervous system infections are self-limited, severe manifestations may include encephalopathy, seizures, and other neurological complications [3]. Molecular testing is particularly valuable when routine cerebrospinal fluid findings are nondiagnostic.

Optic neuritis is an uncommon complication of central nervous system infection [4,5]. In children, optic nerve inflammation may occur during infection or as a para-infectious or post-infectious immune-mediated event. Myelin oligodendrocyte glycoprotein antibody-associated disease is an important cause of pediatric optic neuritis and may be preceded by infection [4,6-8]. We describe acute bilateral optic neuritis occurring in temporal association with enteroviral meningoencephalitis and discuss the diagnostic uncertainty created by concomitant severe hyponatremia.

## 2 CASE PRESENTATION

A previously healthy seven-year-old girl with normal psychomotor development was admitted on Day 0 because of an acute generalized seizure associated with complete bilateral visual loss. Her vaccination status was reported to be current according to the national immunization program. There was no history of recent head trauma, medication or toxic exposure, herpes labialis, tuberculosis contact, or significant personal or family neurological disease.

The pre-admission course and the principal clinical events are summarized in Table 1.

**Table 1: Relative clinical timeline anchored to admission as Day 0**

| Relative time   | Clinical event                                                                                                                           |
|-----------------|------------------------------------------------------------------------------------------------------------------------------------------|
| Day -14         | Febrile diffuse abdominal pain; improvement after symptomatic treatment                                                                  |
| Day -1          | Recurrent abdominal pain, diarrhea, fever, generalized seizure, and prolonged postictal impairment                                       |
| Day 0           | Admission with complete bilateral visual loss and GCS score of 6/15; cerebrospinal fluid obtained                                        |
| Day +1          | Brain MRI showing bilateral asymmetric frontoparietal cortical and subcortical abnormalities                                             |
| Day +6          | Computed tomography angiography and venography negative for arterial stroke and cerebral venous thrombosis                               |
| Hospital course | Recurrent seizures, convulsive status epilepticus, transient right hemiplegia, sodium nadir of 116 mmol/L, polyuria, and 10% weight loss |
| Day +12         | Complete visual recovery documented                                                                                                      |
| Discharge       | Neurologically stable; serum sodium 134 mmol/L; follow-up planned                                                                        |

**Table 2: Admission vital signs and principal laboratory findings**

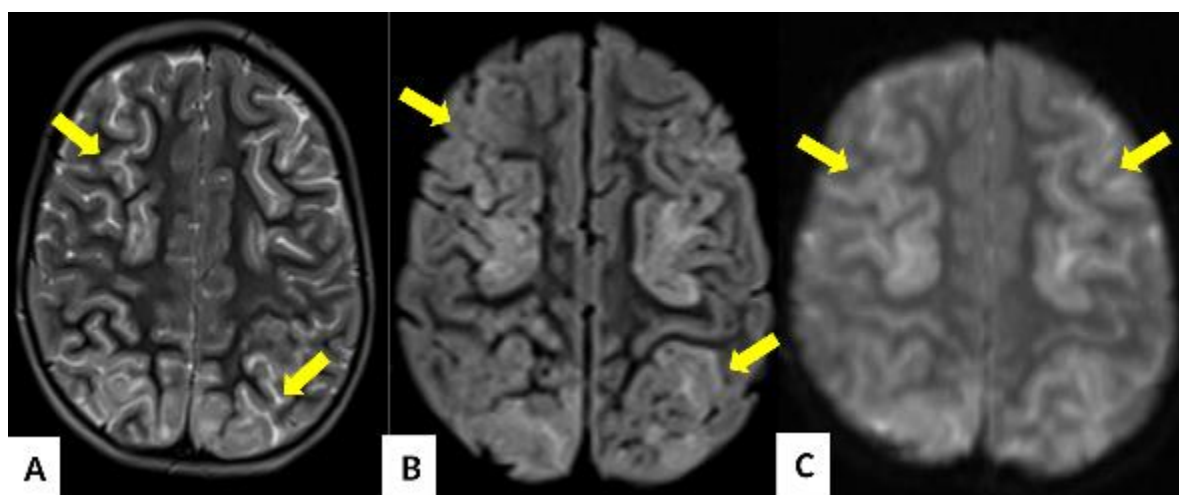
| Parameter                                  | Patient result   | Reference range  | Unit                  |
|--------------------------------------------|------------------|------------------|-----------------------|
| Glasgow Coma Scale score                   | 6/15             | 15               | points                |
| Temperature                                | 38               | 36.5-37.5        | degrees C             |
| Heart rate                                 | 130              | 70-110           | beats/min             |
| Blood pressure                             | 120/70           | 90-120/50-80     | mmHg                  |
| Peripheral white blood cell count          | 21.3             | 4.5-13.5         | $\times 10^9/L$       |
| Absolute neutrophil count                  | 19.2             | 1.5-8.0          | $\times 10^9/L$       |
| C-reactive protein                         | 67               | <5               | mg/L                  |
| Cerebrospinal fluid white blood cell count | <3               | 0-5              | cells/mm <sup>3</sup> |
| Cerebrospinal fluid direct microscopy      | No organism seen | No organism seen | Not applicable        |
| Cerebrospinal fluid enterovirus PCR        | Positive         | Negative         | Not applicable        |
| Serum sodium, nadir                        | 116              | 135-145          | mmol/L                |
| Serum sodium, discharge                    | 134              | 135-145          | mmol/L                |

PCR: polymerase chain reaction. Reference intervals represent commonly used age-appropriate pediatric clinical and laboratory ranges and may vary by institution.

On Day 0, the Glasgow Coma Scale (GCS) score was 6/15 [9]. Neurological examination showed severe impairment of consciousness and bilateral miosis; the remainder of the systemic examination was unremarkable. Admission vital signs and the principal laboratory findings are summarized in Table 2.

Lumbar puncture on Day 0 yielded clear cerebrospinal fluid with no microorganism on direct examination. Cerebrospinal fluid multiplex polymerase chain reaction was positive for enterovirus. Enteroviral central nervous system infection was therefore diagnosed despite the absence of pleocytosis (Table 2).

Brain magnetic resonance imaging performed on Day +1 showed bilateral asymmetric frontoparietal gyriform cortical and subcortical signal abnormalities, interpreted as compatible with an encephalitic process (Figure 1). Subsequent brain computed tomography showed no acute structural abnormality. Ophthalmological examination identified hyperemic optic discs and supported the diagnosis of bilateral optic neuritis in the context of acute visual loss.



**Figure 1: Selected axial brain magnetic resonance images obtained during the acute illness. Panel A is a T2-weighted image, panel B is a fluid-attenuated inversion recovery image, and panel C is a diffusion-weighted image. Yellow arrows indicate representative bilateral asymmetric cortical and subcortical regions of signal abnormality, predominantly in the frontoparietal areas. These brain images are not dedicated orbital sequences and do not demonstrate optic-nerve enhancement.**

The subsequent course included transient right-sided hemiplegia and another episode of convulsive status epilepticus lasting approximately one hour, requiring midazolam and phenobarbital. Computed tomography angiography and venography performed on Day +6 showed no evidence of arterial stroke or cerebral venous thrombosis.

During the same period, serum sodium fell to a nadir of 116 mmol/L. Urine output exceeded 2 L/24 h; at an initial weight of 20 kg, this corresponded to more than 4.17 mL/kg/h. The patient also had marked thirst, persistent urinary sodium loss, hyperosmolar urine, and a 2-kg weight loss from 20 to 18 kg, equivalent to 10% of initial body weight. These findings favored renal salt and water loss. However, no overt clinical signs of hypovolemia were documented, and oral and intravenous fluid intake had not been quantified. A net fluid balance could therefore not be calculated. Cerebral salt wasting was considered probable but could not be definitively distinguished from the syndrome of inappropriate antidiuresis.

HIV serology was negative. The overall immunological assessment, including lymphocyte immunophenotyping, did not identify a significant immunodeficiency.

Intravenous corticosteroids were administered for bilateral optic neuritis for three days, followed by oral corticosteroids tapered to discontinuation. Intravenous immunoglobulin was given at a total dose of 2 g/kg over two days because of the severity and recurrence of the neurological manifestations. Antiepileptic treatment was initiated for recurrent seizures. Hyponatremia was treated with sodium supplementation and tailored hydration. Serum sodium progressively increased to 134 mmol/L at discharge.

Neurological status improved progressively. Complete recovery of visual function was documented on Day +12. The patient became afebrile and regained normal consciousness, without persistent focal neurological deficit or recurrent seizures during the later hospital course. She was discharged on antiepileptic treatment and a tapering oral corticosteroid regimen, with planned neurological and ophthalmological follow-up and repeat brain magnetic resonance imaging.

Myelin oligodendrocyte glycoprotein immunoglobulin G testing was requested to investigate a possible post-infectious demyelinating disorder but could not be performed because of financial constraints. The patient subsequently did not attend the planned reassessment, limiting evaluation of long-term outcome and recurrence.

### 3 DISCUSSION

This case describes a severe enteroviral central nervous system infection associated with status epilepticus, transient focal neurological deficit, profound hyponatremia, and acute bilateral optic neuritis. The positive cerebrospinal fluid multiplex polymerase chain reaction established enteroviral infection in the appropriate clinical context, but it does not by itself prove that optic nerve injury resulted from direct viral invasion. The temporal relationship and rapid response to immunotherapy are compatible with a para-infectious immune-mediated mechanism, although causality cannot be established from a single observation.

The absence of cerebrospinal fluid pleocytosis did not exclude infection. Yun et al. reported that a proportion of children with enteroviral meningitis had no pleocytosis and found an association with early lumbar puncture [2]. This observation supports the use of molecular testing in children with otherwise unexplained encephalopathy or seizures, even when the initial cerebrospinal fluid cell count is normal. Enterovirus identification and characterization generally rely on appropriate molecular diagnostic methods [3,10].

Bilateral optic neuritis in a child raises a broad differential diagnosis that includes infectious, para-infectious, and antibody-associated demyelinating disorders. Myelin oligodendrocyte glycoprotein antibody-associated disease is particularly relevant in pediatric bilateral optic neuritis [4,6,7]. Aseptic meningitis and leptomenigeal inflammation have also been described in association with myelin oligodendrocyte glycoprotein antibodies [8]. In this patient, antibody testing was not available; consequently, myelin oligodendrocyte glycoprotein antibody-associated disease cannot be either confirmed or excluded. The case should therefore be interpreted as an association between enteroviral meningoencephalitis and bilateral optic neuritis rather than proof of enterovirus-triggered demyelinating disease.

Published reports suggest that inflammatory demyelination may overlap with enteroviral encephalitis. Pillai et al. described four children with confirmed enterovirus encephalitis and steroid-responsive acute disseminated encephalomyelitis-like disease [11]. Aljarallah et al. subsequently reported myelin oligodendrocyte glycoprotein antibody-associated optic neuritis after enterovirus meningitis, with visual recovery after intravenous methylprednisolone [12]. These observations support clinical consideration of an immune-mediated complication but are insufficient to define a standardized treatment strategy.

The hyponatremia posed an additional diagnostic challenge. Both the syndrome of inappropriate antidiuresis and cerebral salt wasting may occur during acute central nervous system disease and may present with hyponatremia, concentrated urine, and elevated urinary sodium [13-15]. The principal distinction is volume physiology: inappropriate antidiuresis is generally associated with relative euvolemia and water retention, whereas cerebral salt wasting involves renal sodium loss and extracellular volume contraction. In this patient, polyuria greater than 2 L/24 h, hypernatremia, hyperosmolar urine, marked thirst, and 10% weight loss favored salt and water loss. Nevertheless, absent quantified intake, serial net balance, and objective confirmation of hypovolemia, the available data do not establish cerebral salt wasting. The improvement with hydration and sodium supplementation provides only supportive, nonspecific evidence.

Treatment of most non-polio enteroviral meningoencephalitis remains supportive [3,10]. Corticosteroids and intravenous immunoglobulin were used here because bilateral optic neuritis and recurrent severe neurological manifestations raised concern for an inflammatory complication. Recovery occurred after treatment, but the concurrent therapies, natural viral clearance, and lack of a comparator prevent attribution of benefit to either corticosteroids or intravenous immunoglobulin. The favorable temporal course should therefore not be interpreted as evidence of efficacy.

This report is limited by the absence of myelin oligodendrocyte glycoprotein antibody testing, incomplete quantification of fluid intake, lack of a calculated net fluid balance, and loss to long-term follow-up. In addition, some quantitative urinary indices required for formal differentiation between inappropriate antidiuresis and cerebral salt wasting were not available. Its clinical value lies in highlighting a rare neuro-ophthalmological association, the possibility of enteroviral infection despite acellular cerebrospinal fluid, and the need for cautious interpretation of hyponatremia during central nervous system disease. Longer follow-up is important because pediatric antibody-associated demyelinating disorders may recur [4,16].

#### 4 CONCLUSION

Enteroviral meningoencephalitis can present with severe neurological manifestations despite an acellular initial cerebrospinal fluid profile, making molecular testing important when clinical suspicion remains high. Acute bilateral optic neuritis may occur in temporal association with enteroviral infection, but direct viral injury cannot be distinguished from a para-infectious immune-mediated process without specific testing. Profound hyponatremia with polyuria, hypernatruresis, thirst, and weight loss may suggest cerebral salt wasting; however, volume assessment and quantified fluid balance are necessary before making a definitive distinction from inappropriate antidiuresis. This case supports prompt multidisciplinary neurological, ophthalmological, and electrolyte evaluation while underscoring that treatment response in a single patient cannot establish therapeutic efficacy.

#### STATEMENTS ON COMPLIANCE WITH ETHICAL STANDARDS

##### *Disclosure of conflict of interest*

The authors declare that they have no conflicts of interest.

##### *Statement of informed consent*

Written informed consent for publication, including publication of anonymized clinical images, was obtained from the patient's parent or legal guardian.

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