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CASE REPORT

Co-infections and Acute Disseminated Encephalomyelitis: A Case Report of a Child in Batna, Algeria

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ABSTRACT

Acute disseminated encephalomyelitis (ADEM) is an inflammatory, autoimmune demyelinating disorder of the central nervous system (CNS) that predominantly affects the white matter. It typically occurs following a viral infection or vaccination, although idiopathic cases are also described. ADEM is uncommon but not rare in children. Between April 1, 2012, and August 31, 2015, we conducted a prospective, longitudinal, exhaustive cohort study including patients older than 28 days who presented with clinical features suggestive of HIV-negative encephalitis or meningoencephalitis, met the SPILF inclusion and exclusion criteria, and were admitted to three departments (intensive care, infectious diseases, and pediatrics) of a tertiary care hospital in Batna, Algeria. Among the 141 cases identified, 38 were children (27%). From this cohort, we report a particularly severe pediatric case of monophasic ADEM in a 12-year-old boy with documented exposure to four infectious agents. The patient presented with a 7-day history of febrile meningoencephalitis, altered consciousness, and seizures. Brain MRI showed bilateral frontoparietal demyelinating lesions with supra- and infratentorial as well as medullary involvement. Blood PCR testing was positive for adenovirus and parvovirus B19; a pharyngeal swab detected a coronavirus; and serum IgM was positive for *Borrelia burgdorferi*. Cerebrospinal fluid analysis was acellular and sterile. Despite empirical broad-spectrum anti-infective therapy and high-dose corticosteroids, the patient developed a deep coma, progressed to quadriplegia and severe respiratory distress, required prolonged intensive care, and ultimately died 7 years and 8 months later from chronic respiratory complications unrelated to a new demyelinating episode. This case highlights the diagnostic and therapeutic challenges of severe pediatric ADEM, particularly in the context of multiple potential infectious triggers. It underscores the importance of distinguishing causative pathogens from incidental or sequential infections and illustrates the difficulty of establishing causality in complex co-infection scenarios.

Keywords: ADEM, encephalitis, co-infections, demyelination, adenovirus, parvovirus B19, Lyme disease, coronavirus.

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1. INTRODUCTION

Acute disseminated encephalomyelitis (ADEM) is a monophasic inflammatory demyelinating disease of the CNS, predominantly involving the white matter of the brain and spinal cord, particularly in perivenous regions. It often occurs following a viral infection or vaccination, though idiopathic cases are recognized. ADEM is thought to result from a transient autoimmune reaction, potentially mediated by molecular mimicry, where host immune responses to foreign antigens cross-react with myelin components such as

proteolipid protein (PLP), myelin basic protein (MBP), and myelin oligodendrocyte glycoprotein (MOG). Experimental data support this hypothesis: T lymphocytes from children with ADEM show heightened reactivity to MBP compared to children with uncomplicated viral infections. Other proposed mechanisms include viral integration into host membranes, epitope spreading, and bystander activation during systemic infections. The annual incidence of ADEM in children is estimated at 0.4 per 100,000(5), with a higher incidence in winter and spring(5,6). Infections or vaccinations are the most common triggering factors, reported in about 75% of pediatric cases (7,8) and 45–50% of adult cases (8,9).

Clinically, ADEM presents as acute encephalopathy with rapidly progressive multifocal neurological signs and symptoms, usually occurring 1 to 4 weeks after the triggering event (infection or vaccination). CSF protein electrophoresis may reveal patterns compatible with demyelinating disease. MRI typically shows multifocal white matter abnormalities. The course is usually monophasic. High-dose intravenous corticosteroids, often combined with intravenous immunoglobulins, are commonly used and appear beneficial (10). Prognosis is generally favorable with early treatment, but neurological sequelae are possible, and severe or fatal forms are described.

This report aims to present a clinically and biologically documented case of ADEM with multiple co-detected pathogens, derived from a broader prospective cohort, and to discuss its pathophysiological, diagnostic, and therapeutic implications. Patient anonymity was strictly preserved, and informed consent for publication was obtained from the patient's family.

2. CASE REPORT

This case report originates from a prospective, longitudinal cohort study conducted at the University Hospital of Batna, Algeria, between April 1, 2012, and August 31, 2015. The study included patients older than 28 days with clinical signs suggestive of encephalitis or meningoencephalitis, who were HIV-negative and fulfilled inclusion/exclusion criteria as defined by the SPILF consensus. Participants were recruited from three clinical departments: intensive care, infectious diseases, and pediatrics. Among the 141 patients included, 38 (27%) were children.

We present here a single case of a 12-year-old boy selected from this cohort due to the exceptional severity of his illness and the detection of four potential infectious agents.

Background and Epidemiological Context

The patient, B.I., was a 12-year-old boy from Batna with no known immunodeficiency. He had a prior history of post-traumatic extradural hematoma, surgically managed without neurological sequelae. His vaccination status was complete for age, including BCG, measles, and rubella. Twenty days prior to admission, an outbreak of eruptive febrile illness occurred at his school. He reported a transient febrile illness during this period but without rash or medical consultation.

Initial Presentation (Day 0 to Day 7)

Seven days prior to his admission, the patient developed progressive symptoms consisting of fever, headaches, vomiting, marked fatigue, and progressive deterioration of general condition. Neurological symptoms then appeared, with altered consciousness, drowsiness, apathy, and generalized seizures. The evolution was subacute over one week, prompting referral and admission in April 2013 for suspected meningoencephalitis.

On admission, he presented with encephalopathy and fluctuating consciousness. Focal neurological deficits were difficult to assess because of the altered mental status and seizures. There was no clinical evidence of meningeal irritation. Vital signs and a detailed neurological examination were recorded but are not all available retrospectively.

Diagnostic Work-up

Neuroimaging: Cerebrospinal MRI revealed bilateral fronto-parietal involvement with multiple demyelinating lesions both above and below the tentorium, as well as medullary lesions. The lesions were consistent with multifocal inflammatory demyelination typical of ADEM. No abscess, space-occupying lesion, or vascular malformation was identified.

EEG: Normal background activity; no epileptiform discharges.

CSF: Clear, 2 lymphocytes/mm^3, normal glucose and protein, sterile on direct exam and culture. CSF PCR for common neurotropic viruses (including HSV, VZV, enteroviruses) was negative.

Blood and Serology: Blood PCR: adenovirus and parvovirus B19 positive. Pharyngeal swab PCR: seasonal coronavirus positive. Serum Lyme IgM (Borrelia burgdorferi): reactive. Serologies for toxoplasmosis, rubella, and measles: negative. CRP, ESR, blood counts, and electrolytes: normal. No dominant acute infection emerged; rather, multiple microbial markers of recent exposure were identified.

Treatment Strategy

In light of the clinical severity and pending etiologic confirmation, empirical treatment with cefotaxime, vancomycin, acyclovir, and ofloxacin was initiated. This regimen aimed to cover bacterial meningitis, herpesviruses, and atypical pathogens like Mycoplasma pneumoniae, consistent with epidemiological context. High-dose intravenous corticosteroids were administered due to MRI findings suggestive of ADEM and non-purulent CSF. Supportive care included mechanical ventilation, seizure control, fluid management, and hemodynamic monitoring.

Short-term Evolution

Despite treatment, the patient deteriorated rapidly. He entered deep coma (GCS < 8), developed quadriplegia, and experienced severe respiratory distress, requiring ICU admission and prolonged mechanical ventilation. Recurrent aspiration events and ventilator-associated infections were documented. Virological and serological results confirming co-detection of four pathogens became available later from a reference laboratory in France. The clinical course was monophasic without subsequent demyelinating events.

Long-term Outcome

The patient survived the acute illness but remained severely disabled, with persistent quadriplegia, chronic respiratory insufficiency, and total dependence for care. Over nearly 8 years, he experienced repeated aspirations and infections. He died at home from respiratory failure at age 19. No evidence of recurrent demyelination was found prior to death.

Timeline of Clinical Course : Day –20: Exposure to eruptive fever outbreak at school . Day –7 to 0: Onset of febrile meningoencephalitis symptoms. Day 0: Admission and diagnostic evaluation. Days 1–3: Empirical therapy and MRI. Days 3–10: Coma, quadriplegia, ICU care. Months 1–6: Recurrent respiratory infections. Years 1–7: Chronic phase with severe disability. Year 7.7: Death from respiratory complications.

3. DISCUSSION

ADEM is a rare disease, with an estimated 3 to 6 cases per year reported in large medical centers in the UK, USA, and Australia (11) It is a monophasic inflammatory demyelinating disorder more frequently observed in children than adults. It is typically preceded by infections or vaccinations. Diagnosis requires a combination of acute encephalopathy, multifocal neurologic deficits, and compatible MRI findings. Several pediatric cohorts have shown a male predominance, with a sex ratio between 1.25 and 1.66 (6,7). No specific ethnic predisposition has been identified, but the median age of onset is generally between 4.5 and 7.5 years (13). The interval between the triggering event (infection or vaccination) and clinical onset ranges from 2 to 30 days (1), and a seasonal distribution with a peak in winter and spring has been reported (5,6). In our case, a 12-year-old boy developed a post-infectious encephalomyelitis approximately 21 days after exposure to an eruptive fever epidemic in his school, in early spring (April). This timing and context are highly compatible with a post-infectious immune-mediated process.

Multiple infectious triggers and correlation with ADEM

A wide range of pathogens have been implicated as potential triggers of post-infectious encephalitis and ADEM, including Epstein–Barr virus, cytomegalovirus, herpes simplex virus, varicella-zoster virus, HIV, β-hemolytic Streptococcus, Mycoplasma pneumoniae, Chlamydia pneumoniae, and others (14). In our patient, we identified four micro-organisms: Borrelia burgdorferi (positive serum IgM), adenovirus (blood PCR), parvovirus B19 (blood PCR), and a coronavirus (pharyngeal swab PCR). Each of these agents has been individually associated in the literature with ADEM or post-infectious demyelinating events, including neuroborreliosis presenting as ADEM (15), adenovirus-associated ADEM (16), and coronavirus-associated ADEM (17). The patient had no known immunodeficiency, and routine laboratory tests (including inflammatory markers) were normal. The co-detection of several pathogens in this context therefore does not simply reflect severe immunosuppression. Instead, it likely reflects intense and repeated exposure to multiple respiratory and systemic pathogens in a school setting, followed by a dysregulated immune response in a genetically susceptible host.

Importantly, co-detection of multiple infectious agents is not rare in encephalitis cohorts, particularly in children. Mixed infections have been reported in 2% of encephalitis cases in a large UK study (21) and up to approximately 40% in a Thai cohort (22), illustrating the complexity of attributing causality when several micro-organisms are identified in the same patient. In our case, the temporal relationship between the eruptive fever epidemic and the onset of neurologic symptoms (~3 weeks), combined with the MRI pattern of multifocal supra- and infratentorial demyelination with medullary involvement, is typical of post-infectious ADEM. We cannot definitively determine which of the identified pathogens was the primary trigger of the immune-mediated demyelinating process. However, the coexistence of several plausible infectious agents—each individually known to be capable of inducing or preceding

ADEM—strongly supports the hypothesis of a converging, multi-pathogen "hit" leading to a single, monophasic but extremely severe demyelinating episode.

Rather than attributing causality to a single pathogen, we interpret these findings as evidence that, in a child with no prior neurological disease or immunodeficiency, dense exposure to multiple infectious agents within a short time frame may collectively prime and amplify an aberrant autoimmune response against myelin. In this sense, the detection of several micro-organisms serves as a marker of intense infectious pressure and supports the concept of a multifactorial infectious trigger in ADEM, while acknowledging that causality remains presumed and not demonstrated for each individual micro-organism.

Biological plausibility of multiple pathogens in one host

Children, especially those in school environments, are frequently exposed to multiple respiratory and enteric pathogens over short periods, as illustrated by the eruptive fever epidemic affecting the patient's school 20 days before admission. Several mechanisms can explain the presence of multiple infectious markers in a single host: *Sequential infections within weeks* that leave overlapping virological footprints (PCR positivity, IgM responses), even after clinical resolution of the initial infection ; *Polymicrobial colonization of the respiratory tract* with asymptomatic or pauci-symptomatic carriage of organisms such as adenovirus or seasonal coronaviruses ; *Priming and boosting of the immune response*, whereby an initial infection primes the immune system, and subsequent exposures amplify a dysregulated response against self-antigens (e.g., myelin), through mechanisms such as molecular mimicry and bystander activation.

In this context, it is biologically plausible that our patient's ADEM resulted from complex, multi-pathogen immune stimulation, rather than from a single, dominant etiologic agent. This case also reflects the epidemiologic reality of many low- and middle-income settings, in which children may experience dense and recurrent exposures to a variety of pathogens in overcrowded environments. When advanced virological testing is available and performed, it may reveal this complexity, highlighting that "multiple hits" are more the rule than the exception in some contexts.

Clinical, CSF, and MRI findings

The clinical manifestations in our case—altered consciousness, drowsiness, apathy, seizures, and fever—are consistent with published series. Altered consciousness has been reported in 19–69% of ADEM cases (8,18), seizures in 13–35% (18,19), and fever in 43–52% (7,8,19). There are no pathognomonic biological markers for ADEM. Inflammatory markers are often normal or only mildly elevated. Blood count abnormalities such as lymphopenia or neutrophilic leukocytosis, which may be observed but are not specific. In our patient, routine laboratory tests were normal, which is in line with a Moroccan series of nine pediatric ADEM cases in which inflammatory markers were normal in most patients, except one with an associated respiratory infection (20).

CSF analysis plays a central role in ruling out infectious meningoencephalitis requiring specific antimicrobial therapy. CSF in ADEM may show mild lymphocytic pleocytosis and elevated protein, but normal CSF is also possible. In our case, CSF was entirely normal, a finding that is consistent with data from the Mohammed V Military Instruction Hospital in Morocco, where 72% of ADEM cases had normal CSF (23).

Neuroimaging is crucial for diagnosis. The multiple supra- and infratentorial demyelinating lesions and medullary involvement seen on MRI in our patient are in keeping with reported series, in which approximately 35% of ADEM cases show combined supra- and infratentorial involvement (8) and up to 66% show infra- and supratentorial lesions in some cohorts (23). The bilateral fronto-parietal involvement in our case also prompted empirical antiviral therapy with acyclovir as part of the initial differential that included viral encephalitis.

Treatment, prognosis, and long-term outcome

Empirical broad-spectrum anti-infective therapy remains standard in severe encephalitis while awaiting etiologic clarification. In our setting, this included acyclovir, cefotaxime, vancomycin, and ofloxacin, combined with high-dose intravenous corticosteroid bolus therapy. The retrospective identification of multiple pathogens underscores the difficulty of tailoring therapy in real time when diagnostic results are delayed and multiple potential etiologies coexist.

Currently, the mortality rate of ADEM is generally below 5% (24). Poor prognostic factors include deep coma at admission, refractory seizures, brainstem involvement, and the need for prolonged intensive care. In most series, clinical improvement occurs within days or weeks after initiation of immunotherapy (25). In contrast, our patient experienced an exceptionally severe course, with early deep coma, brainstem and medullary involvement, quadriplegia, and prolonged intensive care stay. Although he survived the acute phase, he remained in a state of profound neurological disability for 7 years and 8 months and ultimately died from long-term complications (recurrent aspiration, chronic respiratory insufficiency, and severe infections).

This long interval between the initial demyelinating event and death may appear surprising if not clearly explained. It is crucial to emphasize that the fatal outcome was not due to a new demyelinating episode or a multiphasic ADEM, but rather to the chronic consequences of a single, extremely severe monophasic attack that left the patient in a highly vulnerable state.

Limitations

This report has limitations. First, we were unable to include representative MRI images due to technical constraints. This constitutes a significant limitation in visually demonstrating the demyelinating lesions. Second, we lacked longitudinal imaging to assess lesion evolution. Third, although multiple pathogens were detected, their temporal relationships and individual contributions to pathogenesis remain speculative. CSF was sterile, limiting confirmation of direct CNS invasion.

4. CONCLUSION

The management of acute disseminated encephalomyelitis (ADEM) must be early, multidisciplinary, and adapted to local epidemiological and resource constraints. This case illustrates an extremely severe, monophasic ADEM episode in a child, associated with multiple plausible infectious triggers (Borrelia burgdorferi, adenovirus, parvovirus B19, and a coronavirus) and resulting in profound, lifelong neurological sequelae and ultimate death from chronic respiratory complications (table 1). The detection of several pathogens in this setting underscores both the intensity of infectious exposure in children living in low- and middle-income countries and the complexity of attributing causality when advanced virological testing is performed. Our observations highlight the need for heightened awareness of ADEM in pediatric encephalitis, prompt neuroimaging and immunotherapy, and long-term multidisciplinary follow-up of survivors, with particular attention to respiratory management and prevention of secondary complications.

Table 1. Summary of Detected Pathogens and ADEM Associations.

Pathogen	Detection Method	Known ADEM Association	Reference
Adenovirus	Blood PCR	Yes (rare pediatric cases)	(16)
Parvovirus B19	Blood PCR	Yes (immune-mediated cases)	(1)
Coronavirus (seasonal)	Pharyngeal PCR	Yes (case reports)	(17)
Borrelia burgdorferi	Serum IgM	Yes (neuroborreliosis)	(15)

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