Scholars Journal of Medical Case Reports
Abbreviated Key Title: Sch J Med Case Rep
ISSN 2347-9507 (Print) | ISSN 2347-6559 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

Anesthesia

Acute Disseminated Encephalomyelitis Complicated by Acute Kidney Injury: A Case Report

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DOI: https://doi.org/10.36347/sjmcr.2024.v12i12.024 | **Received:** 11.11.2024 | **Accepted:** 14.12.2024 | **Published:** 16.12.2024

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Abstract **Case Report**

Acute disseminated encephalomyelitis (ADEM) is a rare inflammatory demyelinating disease of the central nervous system, usually triggered by an infection or, less commonly, vaccination. Although ADEM is primarily a neurological condition, systemic complications such as acute kidney injury (AKI) are exceptionally rare and insufficiently documented in the literature. We report the case of a young adult with ADEM complicated by AKI, an unusual association that posed diagnostic and therapeutic challenges in a resource-limited setting. The aim of this study is to highlight the rare association between ADEM and AKI while describing the clinical features, management challenges, and therapeutic implications.

Keywords: Acute Disseminated Encephalomyelitis (ADEM), Acute Kidney Injury (AKI), Rare Association, Clinical Features, Therapeutic Implications.

INTRODUCTION

Acute disseminated encephalomyelitis (ADEM) is a rare monophasic inflammatory disease primarily affecting the brain and spinal cord. It is characterized by multifocal demyelination of the central nervous system (CNS), often triggered by an infection or, less commonly, vaccination [1].

Although ADEM is predominantly a neurological disorder, systemic complications such as acute kidney injury (AKI) are extremely rare and poorly described in the literature [2]. The standard treatment involves high-dose corticosteroids, with plasma exchange or intravenous immunoglobulins (IVIG) reserved for refractory cases [3].

CASE PRESENTATION

A 28-year-old male with no significant medical history presented with initial symptoms of fever and rhinopharyngitis, which began three days before admission. Treatment with paracetamol, vitamin C, and amoxicillin was prescribed, but his clinical condition worsened, progressing to confusion and increased agitation.

Upon admission, the patient was febrile (38.2°C), confused, disoriented, with a Glasgow Coma Scale (GCS) score of 13/15, and tachycardic (120 bpm) with blood pressure at 135/65 mmHg. He exhibited mild tachypnea with good oxygen saturation. Neurological examination revealed no focal deficits. The patient also experienced vomiting, and the abdominal examination showed epigastric guarding.

Biological investigations revealed marked leukocytosis (22,700/mm³) with elevated C-reactive protein (CRP) levels (109 mg/L), along with acute kidney injury (serum creatinine at 18.4 mg/L and urea at 0.48 g/L). Serum sodium was normal, while mild hypokalemia (3.3 mmol/L) was noted. A computed tomography (CT) scan of the brain performed on admission was normal, whereas an abdominal CT scan suggested peritonitis secondary to a perforated ulcer, prompting exploratory laparotomy, which revealed no abnormalities.

In the following days, the patient's neurological status rapidly deteriorated, with persistent confusion and a progressive decline in consciousness. A lumbar puncture revealed clear cerebrospinal fluid (CSF) with elevated protein (1.17 g/L) and normal glucose levels. These findings initially raised the suspicion of aseptic

meningitis, leading to empirical treatment with acyclovir and ceftriaxone. CSF culture was sterile.

Despite this treatment, the patient's neurological condition worsened, necessitating intubation for neurological distress. Magnetic resonance imaging (MRI) of the brain revealed multifocal hyperintense lesions in the white matter, consistent with ADEM (Figure 1, 2, 3, 4, 5). An immunological workup, including tests for antinuclear antibodies and other autoimmune markers, was performed and found to be unremarkable.

Simultaneously, the patient developed worsening renal function with the onset of anuria, requiring hemodialysis. Four dialysis sessions were conducted between days 6 and 15 to stabilize his metabolic condition.

Treatment was adjusted with the initiation of high-dose intravenous methylprednisolone (1 g/day for three days), followed by oral corticosteroids.

The patient showed transient clinical improvement, marked by the return of diuresis and successful extubation on day 15. However, a neurological relapse on day 19 necessitated reintubation. Due to the unavailability of second-line therapies such as plasmapheresis or IVIG, the patient was transferred to a tertiary care center for specialized management.

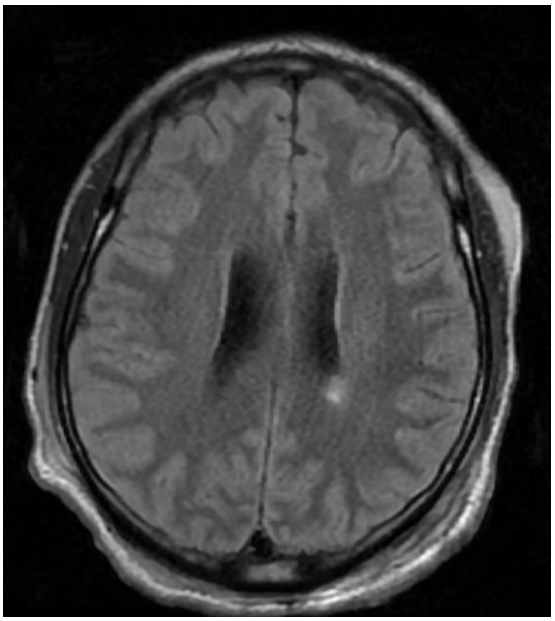

Figure 1: Axial T2 FLAIR: Nodular demyelinating lesions with T2 FLAIR hyperintensity in the juxtaventricular white matter of the bilateral parieto-occipital region

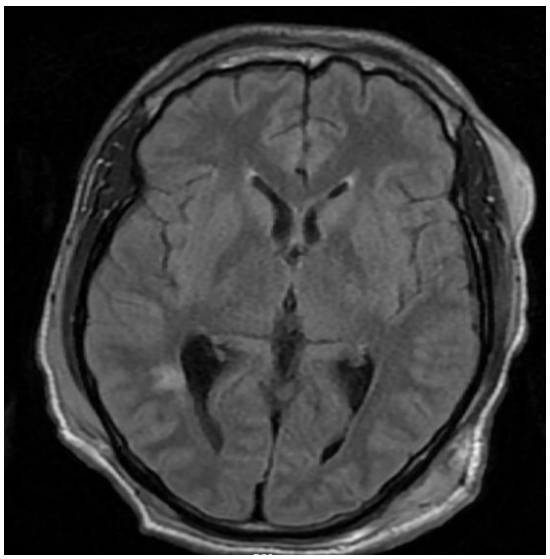

Figure 2: Axial T2 FLAIR: Nodular demyelinating lesions with T2 FLAIR hyperintensity in the juxtaventricular white matter of the bilateral parieto-occipital region

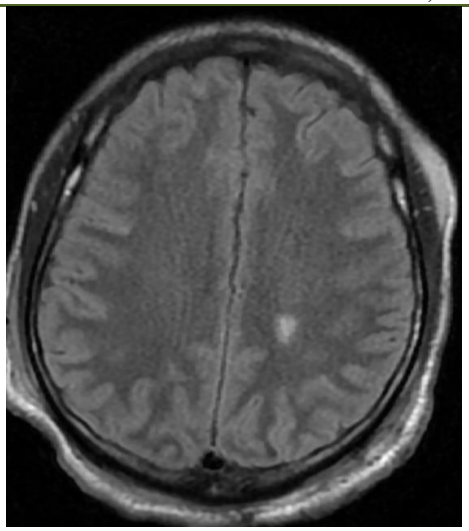

Figure 3: Axial T2 FLAIR: Nodular demyelinating lesions with T2 FLAIR hyperintensity in the juxtaventricular white matter of the bilateral parieto-occipital region

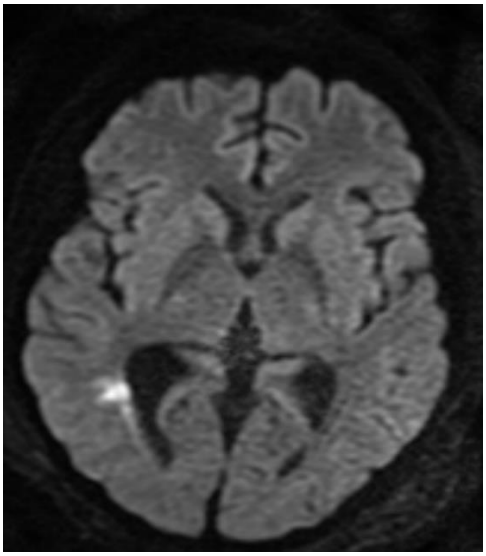

Figure 4: Diffusion sequence B 1000: Some demyelinating lesions are active with diffusion hyperintensity

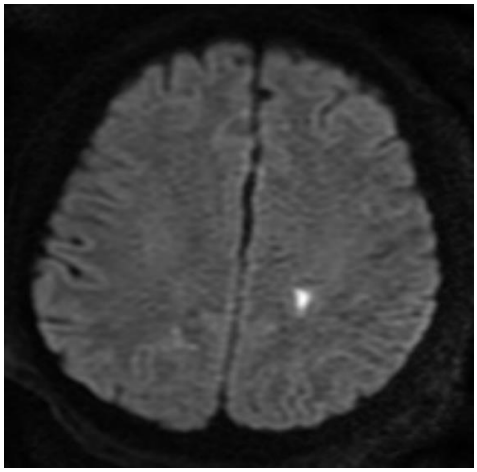

Figure 5: Diffusion sequence B 1000: Some demyelinating lesions are active with diffusion hyperintensity

DISCUSSION

Acute disseminated encephalomyelitis (ADEM) is a rare inflammatory demyelinating disease that most commonly occurs following a viral or bacterial

infection or, less frequently, vaccination. This post-infectious context is well-documented in the literature, with pathogens such as measles, varicella, and respiratory viruses implicated in its onset [1]. In our patient's case, ADEM occurred after an episode of febrile rhinopharyngitis. This association highlights the CNS's susceptibility to a dysregulated immune response triggered by molecular mimicry between pathogen antigens and myelin proteins [2].

ADEM is characterized by significant clinical polymorphism, making its diagnosis challenging, particularly in the early stages. Patients may present with motor, sensory, and cognitive deficits, as well as altered consciousness [3]. In our case, atypical symptoms such as vomiting and abdominal guarding initially suggested a perforated ulcer, while lumbar puncture findings pointed toward meningitis. Such diagnostic errors are common, as systemic manifestations can obscure neurological symptoms [4].

Lumbar puncture is a fundamental diagnostic tool in differentiating CNS pathologies. It helps exclude infectious causes and reveals moderately elevated protein levels with normal glucose in ADEM, as observed in our patient [5]. Immunological testing can be useful in ruling out associated autoimmune diseases, although it was unremarkable in our case. MRI, however, remains the key diagnostic modality, as it demonstrates multifocal hyperintense lesions in the white matter on T2-weighted and FLAIR sequences, characteristic of ADEM. In our patient, MRI confirmed the diagnosis [6].

The association between ADEM and acute kidney injury (AKI) is extremely rare, with few cases reported in the literature. Proposed mechanisms include severe dehydration, drug-induced nephrotoxicity, or autoimmune-mediated renal involvement [7]. In our patient, the progression to anuria requiring dialysis underscores the severity of this association. This complication highlights the importance of close metabolic monitoring and early organ support.

The treatment of ADEM primarily relies on high-dose corticosteroids, which reduce inflammation and limit the progression of demyelinating lesions [8]. In our patient, the administration of methylprednisolone (1 g/day for three days), followed by oral corticosteroids, led to transient clinical improvement. In cases of resistance or relapse, second-line therapies such as plasmapheresis or IVIG are recommended in the literature, although these were unavailable in our setting [9].

CONCLUSION

Acute disseminated encephalomyelitis (ADEM) is a rare but potentially severe inflammatory demyelinating disease of the central nervous system, often presenting with a wide range of clinical features that make its diagnosis challenging. This case highlights the unusual association of ADEM with acute kidney injury (AKI), a complication that remains exceptionally rare and poorly understood. The co-occurrence of these conditions underscores the importance of a multidisciplinary approach in diagnosis and management, especially in resource-limited settings.

Conflicts of Interest: The authors declare no conflicts of interest.

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