Infection and Drug Resistance

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CASE REPORT

Clinically Suspected Acute Post-Measles Encephalitis Complicated by Paracetamol-Induced Hepatotoxicity in an Unvaccinated Child: A Case Report from a Resource-Limited Setting

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Introduction: Measles remains a major cause of childhood morbidity and mortality in settings with low vaccination coverage. Although most cases are self-limited, severe neurological complications can occur, including post-measles encephalitis, which may be life-threatening and diagnostically challenging in resource-limited settings.

Case Presentation: We report an unvaccinated young child who developed a clinical syndrome most consistent with acute post-measles encephalitis after a typical measles illness characterized by fever, maculopapular rash, conjunctivitis, cough, and household exposure. The child later presented with seizures and altered consciousness. Laboratory evaluation showed anemia, elevated inflammatory markers, and markedly elevated liver enzymes in the setting of supratherapeutic paracetamol exposure. Because cerebrospinal fluid analysis and magnetic resonance imaging were not available, the diagnosis was made clinically based on the history, timing of neurological deterioration, and exclusion of major structural intracranial pathology on brain computed tomography. The patient was treated with supportive intensive care, empirical antimicrobial and antiviral therapy, vitamin A, intracranial pressure management, and intravenous N-acetylcysteine, with complete neurological recovery.

Conclusion: Acute post-measles encephalitis should be considered in unvaccinated children who develop new neurological symptoms after recent measles illness. This case highlights the importance of early clinical recognition, routine measles immunization, and safe fever management, including careful weight-based paracetamol dosing and caregiver counseling in resource-limited settings.

Keywords: measles, post-measles encephalitis, acute encephalitis, unvaccinated child, neurological complications

Introduction

Measles is a highly contagious viral illness caused by a member of the Paramyxoviridae family, which is transmitted through respiratory droplets. Clinically, it presents with fever, cough, coryza, conjunctivitis, Koplik spots, and morbilliform rash. Although most patients recover uneventfully, measles remains a major cause of childhood morbidity and mortality worldwide, particularly in regions with poor vaccination coverage.[1,2]

Global vaccination programs have reduced measles deaths by 88% between 2000 and 2024, thereby preventing approximately 59 million deaths worldwide. However, measles continues to cause significant disease burden, with 11 million infections and 95,000 deaths reported in 2024, mostly among children under five years.[3,4] The African region remains disproportionately affected, accounting for over half of global measles deaths, with 194,147 cases reported in 2024 despite progress in vaccination campaigns.[4,5] In resource-limited settings, the management of persistent fever in children may also be

https://doi.org/10.2147/IDR.S602243
Received: 9 February 2026
Accepted: 29 April 2026
Published: 4 May 2026

complicated by medication dosing errors, particularly when caregivers rely on repeated unsupervised administration of commonly available antipyretics. Although paracetamol is generally safe at recommended doses, supratherapeutic use can result in hepatotoxicity and may further complicate the clinical course of already vulnerable pediatric patients.

This persistent burden is particularly concerning because measles is not only a cause of widespread childhood mortality, but also carries the risk of severe neurological complications. Neurological complications are among the most severe outcomes, occurring in approximately one per 1000 cases. Four distinct forms have been described: primary measles encephalitis, acute post-infectious measles encephalomyelitis, measles inclusion body encephalitis, and subacute sclerosing panencephalitis.[2,6] Case reports have illustrated variability in these complications, ranging from full recovery to fatal progression.[1,6]

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Case Presentation

A 37-month-old female child was admitted to our hospital with altered consciousness following a prolonged febrile illness. Fifteen days prior to admission, she developed high-grade fever associated with oral thrush for two days, followed by a maculopapular rash that started behind the ears and subsequently spread to the trunk and limbs. This was accompanied by conjunctivitis and a mild cough. There was a clear history of measles exposure, as her older sister had recently been diagnosed with the infection. The child and her siblings did not receive routine childhood vaccinations, including measles vaccination.

Initially, the child was treated at a local facility with ceftriaxone and paracetamol, which temporarily reduced the fever. However, on day 14 of illness, she remained highly febrile and experienced a sudden generalized tonic–clonic seizure that progressed in frequency and duration, ultimately leading to loss of consciousness. At the regional hospital, she received ampicillin, gentamicin, and rectal diazepam, however, her condition did not improve. Given her deteriorating neurological status, the patient was urgently referred to our facility for further management.

On arrival, the patient had an unconscious GCS score of 10/15, febrile (38.9°C), tachycardia (110 bpm), blood pressure of 96/59 mmHg, and oxygen saturation of 96% on room air. The patient's blood sugar level was 90 mg/dL. Chest examination revealed mild bilateral crackles, that were more pronounced on the right side. Neurological examination revealed generalized hypotonia with a normal muscle bulk. The upper limbs demonstrated minimal spontaneous movement, whereas the lower limbs showed withdrawal movements in response to deep pain stimuli. The deep tendon reflexes were exaggerated, and bilateral extensor plantar responses were observed. Cranial nerve examination revealed intact facial nerve function. The neck was supple without stiffness, and the abdomen was soft and nontender. No meningeal irritation was observed.

Laboratory investigations showed a white blood cell count of 7.34 $\times 10^9$/L, hemoglobin 8.4 g/dL, platelet count 287 $\times 10^9$/L, ALT 1123.2 U/L, AST 96 U/L, and CRP 27 mg/L. Renal function and serum electrolytes level were within normal limits. The coagulation profile revealed a PT of 19 seconds and activated partial thromboplastin APTT of 30.8 seconds. Arterial blood gas analysis revealed a pH of 7.43, pCO of 40 mmHg, and pO of 114 mmHg (Table 1). A computed tomography (CT) scan of the brain showed normal findings (Figure 1). The child was diagnosed with acute post-measles encephalitis and admitted to the pediatric intensive care unit (ICU). She was started on empirical treatment for encephalitis, including a 10-day course of acyclovir

Table 1 Laboratory Investigation During Hospitalization

Tests	Reference Range	On ICU Admission	On Ward	On Discharge
White cell count (WBC, $*10^9$/L)	4.00–10.00	7.34	20	10
Hemoglobin (HB, g/dl)	12.0–16.0	8.4	10.2	11
Platelet (PLT, $*10^9$/L)	100–300	287	400	340
Neutrophil %	0.500–0.700	0.521	0.661	0.550
Lymphocyte %	0.200–0.400	0.09	0.06	0.05
Eosinophil %	0.005–0.050	0.002	0.002	0.050
C-reactive protein (CRP, mg/L)	2.5–10	27	250	20
Aspartate transaminase (AST-U/L)	6–38	86	68.2	24
Alanine transaminase (ALT, U/L)	6–40	1123.2	119.9	25.2
Creatinine (Creatinine, mg/dl)	0.4–1.4	0.5	0.7	1.00
Blood urea (Blood urea, mg/dl)	10–50	33.6	27.3	23.4
Sodium (Na+, mmol/l)	135.0–145.0	140.3	142.2	139.6
Potassium (K+, mmol/l)	3.5–5.5	4.3	3.6	3.9
Calcium (Ca+, mmol/l)	2.12–2.62	2.01	2.26	2.24

Notes: Serial laboratory findings on ICU admission, on the ward, and at discharge showing improvement in liver enzymes, inflammatory markers, and hematologic parameters during treatment and recovery. Marked ALT elevation on admission was clinically attributed to supratherapeutic paracetamol exposure. *Marked ALT elevation on ICU admission was clinically attributed to supratherapeutic paracetamol exposure. Units: $\times10^9$/L = $\times10^9$ per liter.

Abbreviations: WBC, white blood cell count; HB, hemoglobin; PLT, platelet count; CRP, C-reactive protein; AST, aspartate aminotransferase; ALT, alanine aminotransferase; Na+, sodium; K+, potassium; Ca+, calcium; ICU, intensive care unit.

and cefotaxime, along with supportive care. Empirical antimicrobial therapy was initiated because the child presented with fever, seizures, and altered consciousness, and bacterial central nervous system infection or sepsis could not be safely excluded at initial presentation in our setting.

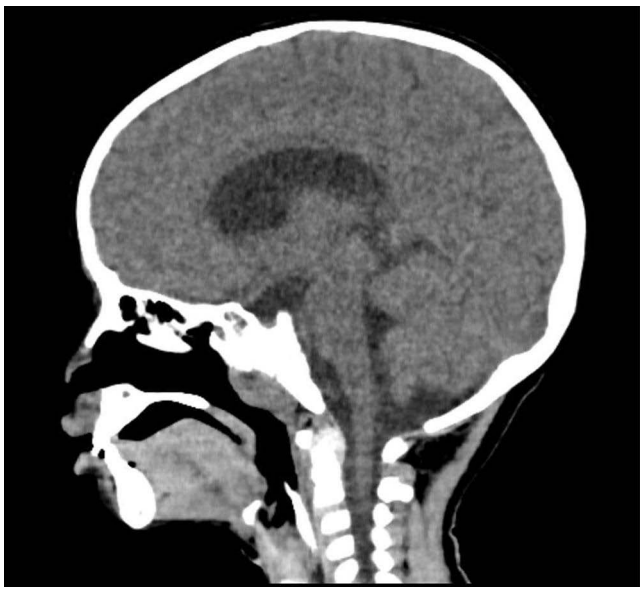

Figure 1 Brain computed tomography on admission. Non-contrast brain CT showed no major structural intracranial abnormality. This unremarkable scan supported exclusion of gross structural pathology while the diagnosis remained based on clinical and epidemiologic features.

https://doi.org/10.2147/IDR.S602243

An ophthalmology consultation was obtained, and supportive eye care with artificial tears was provided. Lumbar puncture was not performed because of the clinical suspicion of raised intracranial pressure, supported by fundoscopic findings of blurred disc margins and vascular obscuration. Given the markedly elevated liver enzyme levels, a thorough medication history was obtained, revealing supratherapeutic paracetamol administration. Based on the child's weight (13 kg) and syrup concentration (250 mg/5 mL), the recommended 130–195 mg (2.6–3.9 mL) per dose. However, approximately 300 mg (6 mL) per dose was administered three times daily for persistent fever, which substantially exceeded the recommended pediatric dosing. In this context, paracetamol-induced hepatotoxicity was strongly suspected, and intravenous N-acetyl cysteine was promptly initiated.

On the first day in the ICU, the patient remained unconscious, and supportive care and close monitoring were continued. By the second day, she had become fully conscious, hemodynamically stable, and tolerated oral feeding, allowing discontinuation of intravenous fluids. No neurological deficits were observed, and the patient was subsequently transferred to the ward for continued observation. On the third day after admission, the laboratory parameters showed significant improvement, (Table 1). Clinically, she remained stable without recurrence of seizures or other complications. The patient showed gradual improvement with an intensive rehabilitation program. On day 10 of hospitalization (the sixth day post-ward admission), oral iron therapy (ferrosinol syrup) was initiated at a dose of 20 mg (4 mL) twice daily to address anemia and support hematological recovery during convalescence.

On day 10 after admission, the patient was discharged in stable condition. She was prescribed oral antibiotics and scheduled for follow-up one week later. At discharge, the patient demonstrated full neurological recovery, was feeding normally, and exhibited no residual deficits. This case highlights the critical role of timely ICU management, empiric antimicrobial and antiviral therapy, vitamin A supplementation, and careful monitoring of medication dosing in the successful treatment of post-measles encephalitis in unvaccinated children.

Discussion

This case report describes a rare presentation of measles-associated acute encephalitis in an 37-month-old infant, emphasizing the unpredictable and potentially life-threatening nature of this complication. The delayed onset of altered consciousness following the resolution of classical measles manifestations highlights the diagnostic challenge of post-infectious neurological complications. Importantly, timely supportive management resulted in complete neurological recovery, underscoring the critical role of early recognition and vigilant care in improving outcomes.

Measles remains an important cause of preventable childhood morbidity and can rarely be complicated by severe neurologic disease, including post-infectious encephalitic syndromes. Reported CNS complications include primary measles encephalitis, post-infectious encephalitis/encephalomyelitis, measles inclusion body encephalitis, and subacute sclerosing panencephalitis. Published cases show variable outcomes, ranging from full recovery to persistent neurologic sequelae or death. In the present case, the delayed onset of seizures and encephalopathy after the measles illness made a post-infectious process clinically more likely than acute measles-related CNS involvement.[7,8]

During the acute phase, measles typically presents with fever, cough, coryza, conjunctivitis, and nasal congestion, followed by a characteristic morbilliform rash. Koplik spots on the buccal mucosa are observed in approximately 70% of cases.[9,10] Beyond these manifestations, measles can cause serious complications, including diarrhea, otitis media, pneumonia, central nervous system (CNS) infections and sequelae, blindness, and hearing loss.[10,11] Morbidity and mortality remain disproportionately high in developing countries due to undernutrition, overcrowding, limited access to healthcare, and inadequate vaccination coverage.[12]

The present discussion situates this case within the broader spectrum of measles-related CNS complications. The measles virus may affect the CNS during active infection or after viral clearance. Recognized neurological complications include primary measles encephalitis (PME), acute post-infectious measles encephalomyelitis (APME), measles inclusion body encephalitis (MIBE), and subacute sclerosing panencephalitis (SSPE)[6] (Table 2). While these entities differ in pathogenesis, immune status, and clinical presentation, all involve complex interactions between the virus, host immunity, and neural tissue, leading to significant morbidity and mortality.[6]

Although global measles incidence declined markedly following widespread immunization, recent years have seen a concerning resurgence. Between 2000 and 2017, measles cases declined by approximately 83%; however, in 2017

Table 2 Types of Measles-Induced Encephalitis

Type	Timing and Clinical Features	Mortality
Primary measles encephalitis	Occurs during the acute phase of measles infection; associated with active rash; caused by direct viral invasion of neuronal tissue; measles virus RNA may be detected in cerebrospinal fluid (CSF).	10–15%
Acute post-measles encephalitis	Develops 2–3 weeks after measles infection; immune-mediated inflammatory process; presents with seizures, altered consciousness, and encephalopathy following apparent recovery from measles.	~5% in children; up to 25% in adults
Measles inclusion body encephalitis	Occurs months to one year after infection; primarily affects immunocompromised patients; progressive neurological deterioration; measles viral RNA detected in brain tissue.	High
Subacute sclerosing panencephalitis (SSPE)	Develops 6–15 years after measles infection; more common in children infected at a younger age; progressive cognitive decline leading to coma and death; very high measles antibody titers in CSF.	Nearly 100% (death within 1–3 years)

Notes: Summary of the major forms of measles-associated encephalitis, including timing, principal clinical features, and reported mortality.
Abbreviations: CSF, cerebrospinal fluid; RNA, ribonucleic acid.

alone, an estimated 173,330 cases and 110,000 deaths were reported worldwide, predominantly in Asia and Africa.[13,14] Over the past several years, both developing and industrialized countries have experienced increased outbreaks. Notably, a 300% rise in measles cases was reported in countries such as the United States and France in 2019, largely attributed to declining vaccination coverage and vaccine hesitancy.[14–16]

Published literature demonstrates wide variability in neurological outcomes. Reports from Oman describe a child with acute post-measles encephalitis who achieved full recovery following corticosteroids and vitamin A supplementation.[17] Conversely, Ukrainian data document both benign post-infectious encephalitis and fatal SSPE occurring years after initial infection.[18] More recent reports describe atypical cases, including MIBE in immunocompromised patients and diagnostic challenges due to overlapping clinical features.[19] Encephalitis remains the most frequent neurological complication of measles, occurring in approximately 1 per 1000 infected children, with risks of seizures and long-term neurological sequelae.[20,21]

Although acute post-infectious measles encephalitis may overlap conceptually with ADEM-spectrum disorders, formal application of IPMSSG criteria was not possible in this case because MRI and cerebrospinal fluid data were unavailable; accordingly, we describe the presentation as clinically most consistent with acute post-measles encephalitis rather than a definitively classified ADEM-spectrum disorder.

The clinical presentation in this case was most consistent with acute post-measles encephalitis. Neurological symptoms developed more than one week after the initial measles illness, after the rash had already evolved, which favored a post-infectious process rather than uncomplicated acute febrile measles. The diagnosis was made clinically on the basis of the characteristic measles prodrome, clear household exposure, delayed onset of encephalopathy and seizures, and the absence of meningeal irritation on examination. Cerebrospinal fluid analysis and magnetic resonance imaging were not available because lumbar puncture was deferred due to concern for raised intracranial pressure and advanced neuroimaging was not accessible. Brain computed tomography was unremarkable and helped exclude major structural intracranial pathology.[22–24] Bacterial meningitis was considered less likely because there was no neck stiffness or other overt meningeal signs, while primary herpes simplex encephalitis could not be definitively excluded at presentation and therefore justified empiric acyclovir therapy. Primary measles encephalitis was also considered less likely because the neurological deterioration occurred after the initial measles phase rather than during the peak of active infection. In view of these findings, the presentation was considered clinically most compatible with acute post-measles encephalitis, although definitive confirmation was not possible.[19,25,26]

Management in this case was primarily supportive and guided by the child's acute presentation. Because she presented with fever, seizures, and encephalopathy, empiric acyclovir and antibacterial therapy were started at admission in accordance with standard initial management for suspected encephalitis while alternative infectious causes remained under consideration. Once the clinical picture became more compatible with post-measles encephalitis, management focused on supportive care, control of suspected raised intracranial pressure, vitamin A supplementation, and close neurologic monitoring.[27] Corticosteroids have been

described in some reports of post-infectious measles-related encephalitic illness, but the evidence remains limited and largely based on case reports and small series. We therefore revised the discussion to avoid overstating the role of escalation therapies and to emphasize that treatment decisions should be individualized according to clinical severity, available investigations, and local resources.[28]

Additional antimicrobial therapies may be considered based on clinical suspicion.[29] Once measles is established as the etiology, treatment focuses on immunomodulation and supportive care.[2,30] Vitamin A supplementation is recommended for all pediatric measles cases to reduce morbidity and mortality.[26,30] Case reports from Oman and Ukraine highlight the role of steroids and supportive therapy in achieving full recovery, whereas delayed diagnosis or immunocompromised status may lead to poor outcomes.[1,6]

An additional important lesson from this case is the risk of paracetamol hepatotoxicity during home-based fever management. In many low-resource settings, caregivers may administer antipyretics repeatedly without clear guidance regarding weight-based dosing, dosing intervals, or formulation strength. Confusion between syrup concentration and volume, together with ongoing concern about persistent fever, may unintentionally lead to supratherapeutic dosing. In this patient, the markedly elevated liver enzymes and medication history were strongly suggestive of paracetamol-induced hepatotoxicity, prompting timely initiation of intravenous N-acetylcysteine. This aspect of the case highlights the need for caregiver education, clear prescribing instructions, and careful medication counseling whenever antipyretics are recommended for children.[1,6]

This case has several important limitations that should be acknowledged. Cerebrospinal fluid analysis was not performed because lumbar puncture was deferred due to clinical concern for raised intracranial pressure, supported by fundoscopic findings. In addition, magnetic resonance imaging and confirmatory measles virologic testing were not available because of resource and clinical constraints. Accordingly, the diagnosis was made on a clinical and syndromic basis rather than through full laboratory or radiologic confirmation. To minimize diagnostic uncertainty, we relied on the characteristic measles prodrome and rash, clear household exposure history, the temporal relationship between measles illness and subsequent neurological deterioration, brain CT to exclude major structural intracranial pathology, fundoscopy, close ICU monitoring, empiric management for acute encephalitis, and serial clinical reassessment during hospitalization. Although these limitations prevented definitive confirmation, the overall presentation and favorable clinical course were considered most consistent with acute post-measles encephalitis. These constraints should be considered when interpreting the case and its implications.

This case highlights the unpredictable course of measles-associated encephalitis and reinforces the importance of early clinical recognition, even in resource-limited settings. Prompt supportive and immunomodulatory treatment can significantly reduce mortality and prevent long-term neurological sequelae.

Conclusion

Acute post-measles encephalitis should be considered in unvaccinated children who develop seizures or encephalopathy after a recent measles-like illness, particularly in resource-limited settings. In this case, the diagnosis was clinical and considered most compatible with acute post-measles encephalitis on the basis of the characteristic prodrome, epidemiological exposure, delayed neurological deterioration, and overall clinical course, although definitive confirmation was not possible because key investigations were unavailable. This case illustrates the potential value of early recognition, careful supportive management, and appropriate empiric treatment when alternative infectious causes cannot initially be excluded. Most importantly, it underscores two practical implications: strengthening routine measles immunization to prevent measles and its neurological complications, and ensuring safe fever management through careful weight-based paracetamol dosing, clear caregiver counseling, and vigilance for medication-related toxicity.

Ethics and Consent

Written informed consent for publication of this case report and the accompanying clinical details and images was obtained from the patient's parent because the patient was a minor. In accordance with institutional policy, formal ethical approval was not required for a single case report. The report was prepared in accordance with the ethical principles of the Declaration of Helsinki. This case report was prepared in accordance with the CARE reporting guideline.

Acknowledgments

This research was supported by SIMAD University, Mogadishu, Somalia. We gratefully acknowledge the Center for Research and Development at SIMAD University for their valuable guidance and constructive recommendations throughout this work.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Disclosure

The authors declare no conflicts of interest in this study.

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