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Kawasaki Disease With Acute Thrombocytopenia In A Child With Lennox–Gastaut Syndrome And Periventricular Leukomalacia: A Case Report

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Abstract

Background: Kawasaki disease (KD) presents significant diagnostic challenges in children with complex neurological conditions where baseline altered mental status and overlapping clinical features may obscure classic vasculitic presentations. Atypical features such as thrombocytopenia can further complicate recognition and delay treatment.

Case Presentation: A 3-year-old male with West syndrome evolving to Lennox-Gastaut syndrome, global developmental delay secondary to periventricular leukomalacia, and baseline neurological impairments presented with acute fever and drowsiness. Initial thrombocytopenia prompted diagnosis of viral fever. Despite empiric antibiotics, antimalarials, blood product support, and escalating respiratory support from nasal oxygen to CPAP, clinical deterioration continued. Systematic reassessment revealed Kawasaki disease. Intravenous immunoglobulin (2 g/kg) and aspirin therapy resulted in rapid defervescence, respiratory improvement, and hematological recovery without coronary artery abnormalities on echocardiography.

Conclusion: Kawasaki disease should remain in the differential diagnosis of prolonged fever in neurologically complex children, even with atypical presentations including thrombocytopenia and altered sensorium. IVIG and aspirin can be safely administered alongside antiepileptic polytherapy with appropriate monitoring. This case emphasizes the importance of systematic reassessment when patients fail to respond to empiric therapies and maintaining high clinical suspicion for vasculitic syndromes in persistently febrile children regardless of underlying medical complexity.

Keywords: Kawasaki disease, Lennox-Gastaut syndrome, Periventricular leukomalacia, Thrombocytopenia, Epileptic encephalopathy, Developmental delay

Introduction

Kawasaki disease (KD) is an acute, self-limited systemic vasculitis predominantly affecting children under 5 years of age, with peak incidence between 6 months and 2 years[1] [2] [3]. First described by Dr. Tomisaku Kawasaki in 1967, it has become the leading cause of acquired heart disease in children in developed countries, with coronary artery abnormalities developing in 15-25% of untreated cases[1] [2] [3]. The diagnosis relies on clinical criteria: fever persisting for at least 5 days accompanied by at least four of five principal features including bilateral non-exudative conjunctivitis, oral mucosal changes, polymorphous rash, extremity changes, and cervical lymphadenopathy[1] [3] [4]. Early recognition and treatment with intravenous immunoglobulin (IVIG) within 10 days of fever onset significantly reduces the risk of coronary artery complications from 25% to 3-5%[1] [2] [3]. Laboratory findings frequently include

elevated inflammatory markers, thrombocytosis (though thrombocytopenia may occur in the acute phase), and anemia, with incomplete or atypical presentations posing diagnostic challenges particularly in children with underlying medical conditions[1] [2] [3][5].

However, diagnosing Kawasaki disease in children with pre-existing neurological disorders, particularly those with epileptic encephalopathies such as Lennox-Gastaut syndrome (LGS), presents formidable clinical challenges[1] [2] [3]. Children with LGS—a severe epileptic encephalopathy characterized by multiple seizure types and intellectual disability—and underlying structural brain abnormalities like periventricular leukomalacia (PVL) often present with altered mental status, making it difficult to distinguish baseline neurological dysfunction from acute systemic illness[6] [7] [8] [9]. The overlapping features of fever, drowsiness, and clinical deterioration may initially suggest seizure exacerbation, post-ictal state, or infectious encephalitis rather than vasculitis[8] [10].

Furthermore, thrombocytopenia—uncommon but recognized in early Kawasaki disease—may redirect diagnostic attention toward viral infections, bacterial sepsis, or drug-induced cytopenias, particularly in children on multiple antiepileptic medications[1] [11] [12] [13].

The literature provides limited guidance on recognizing Kawasaki disease in neurologically complex children, and delays in diagnosis may occur when compelling alternative explanations for fever and clinical deterioration are present[1] [2] [3].

Therefore, we report a case of a 3-year-old male with a complex neurological history including West syndrome evolved to LennoxGastaut syndrome, global developmental delay secondary to periventricular leukomalacia, visual defects, spastic quadriparesis, and ongoing polytherapy with antiepileptic medications (valproate, clobazam, lamotrigine) and immunomodulatory agents (prednisolone, risperidone) who presented with acute fever and drowsiness. Initial assessment revealed thrombocytopenia prompting treatment for presumed viral fever. Progressive clinical deterioration with increasing respiratory distress necessitated oxygen supplementation, escalation to CPAP support, blood product transfusion (packed cells, fresh frozen plasma), and empiric antimicrobial and antimalarial therapy. Despite these interventions, the patient's condition continued to worsen until Kawasaki disease was recognized and treatment with IVIG and aspirin was initiated. This case underscores the diagnostic pitfalls in recognizing Kawasaki disease when fever and systemic illness occur in children with severe underlying neurological conditions, highlights the importance of systematically evaluating for vasculitic syndromes even when alternative diagnoses appear compelling, and demonstrates successful outcomes with timely IVIG therapy despite diagnostic delays and medical complexity.

Case Presentation

Patient Information

A 3-year-old male child, a known case of West syndrome with evolution to Lennox-Gastaut syndrome, global developmental delay, and encephalopathy secondary to periventricular leukomalacia, presented to the emergency department with acute onset high-grade fever and drowsiness.

Clinical History

Past Medical History

The patient had a documented history of periventricular leukomalacia (PVL) on prior brain MRI, demonstrating bilateral periventricular white matter injury from perinatal hypoxic-ischemic insult. This resulted in global developmental delay with significant impairment across all developmental domains. The child had not achieved independent ambulation and showed severe delays in speech, cognitive, and adaptive functioning.

Associated Neurological Manifestations Included:

Visual impairment with reduced acuity and tracking deficits related to posterior visual pathway damage

Bilateral convergent squint (esotropia)

Spastic quadriparesis with increased tone, hyperreflexia, and extensor plantar responses

Muscle power 4/5 in all four limbs on Medical Research Council grading

Epilepsy History

The seizure disorder began in infancy as West syndrome (infantile spasms) with documented hypsarrhythmia on EEG. By approximately 2 years of age, evolution to Lennox-Gastaut syndrome (LGS)

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occurred, characterized by multiple seizure types including predominantly tonic seizures, atypical absence seizures, and occasional atonic seizures.

Electroencephalographic findings: Most recent abnormal EEG demonstrated generalized slow spike-and-wave discharges at 1.52 Hz, multifocal epileptiform abnormalities, and diffuse background slowing, confirming West syndrome with Lennox-Gastaut syndrome with encephalopathy.

Current Medications

The patient was maintained on intensive antiepileptic polytherapy:

Sodium Valproate (broad-spectrum antiepileptic)

Clobazam (benzodiazepine for refractory seizures)

Lamotrigine (effective for LGS-associated seizures)

Prednisolone (corticosteroid for epileptic encephalopathy)

Risperidone (atypical antipsychotic for behavioral symptoms) Medication compliance was good as per caregiver report.

Presenting Complaints

High-grade fever since one day prior to admission

Increased drowsiness beyond baseline neurological status

History Of Present Illness

The patient was in his usual state of health (relative to baseline neurological condition) until onset of acute high-grade fever 24 hours before presentation. Parents noted associated drowsiness with decreased responsiveness and reduced interaction, raising concern for seizure exacerbation, post-ictal state, or acute neurological decompensation. No witnessed seizures or status epilepticus occurred. There was no history of rash, conjunctival injection, oral changes, extremity swelling, lymphadenopathy, respiratory symptoms, gastrointestinal complaints, or urinary symptoms at initial presentation.

Physical Examination

General examination: The child appeared acutely ill, febrile, and lethargic but arousable to stimuli. Respiratory distress was not evident initially.

Cardiovascular system: Normal heart sounds without murmurs; peripheral pulses adequate.

Respiratory system: Normal respiratory effort; bilateral air entry equal without adventitious sounds on initial assessment.

Abdomen: Soft, Non-Tender, No Hepatosplenomegaly.

Neurological examination: Drowsiness with reduced alertness noted. Baseline deficits documented: spastic quadriparesis with power 4/5 in all four limbs, hyperreflexia with ankle clonus, extensor plantar responses bilaterally, bilateral convergent squint, and visual tracking deficits. No new focal neurological signs or meningeal signs identified.

Dermatological examination: No rash, petechiae, or purpura on admission.

Investigations

Complete blood count: Revealed significant thrombocytopenia, concerning for viral infection, dengue, immune thrombocytopenia, or systemic inflammatory process.

Peripheral blood smear: Confirmed thrombocytopenia; red and white cell morphology otherwise normal.

Two-dimensional echocardiography (2D-Echo): Performed to assess cardiac function and screen for structural abnormalities, with particular relevance to eventual Kawasaki disease diagnosis.

Previous MRI brain: Documented periventricular leukomalacia with bilateral periventricular white matter signal abnormalities.

Previous EEG: Abnormal study showing generalized slow spike-and-wave discharges, multifocal epileptiform activity, and background slowing consistent with West syndrome evolving to Lennox-Gastaut syndrome with encephalopathy.

Timeline of Hospital Course

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Time Point	Clinical Event	Management	Clinical Status
Day -1	Onset of high-grade fever at home	Home care with antipyretics	Alert, interactive
Day 0 (Admission)	Presentation with fever and drowsiness; thrombocytopenia detected	Admission, IV fluids, continued baseline medications	Lethargic, febrile
Day 1	Persistent fever despite supportive care	Empiric IV antibiotics initiated	No improvement
Day 2	Clinical deterioration; respiratory distress developing	Antibiotics escalated; oxygen via nasal prongs; PCV and FFP transfused	Worsening
Time Point	**Clinical Event**	**Management**	**Clinical Status**
Day 3	Continued fever; increasing oxygen requirement	Antimalarial therapy added	Critical
Day 4	Severe respiratory distress	Escalated to CPAP support	Deteriorating
Day 5	Diagnostic reassessment; KD recognized	IVIG (2 g/kg) + high-dose aspirin initiated	Turning point
Day 6	Fever resolved; respiratory status improving	Weaning from CPAP to nasal oxygen	Improving
Day 7-8	Continued clinical improvement	Transitioned to room air; low-dose aspirin	Stable
Day 9 (Discharge)	Ready for discharge	Discharge on baseline medications + low-dose aspirin	Recovered to baseline

Diagnosis and Management

Initial Working Diagnosis

Viral fever with thrombocytopenia in a child with Lennox-Gastaut syndrome

Initial Management

Supportive care included intravenous fluid resuscitation, antipyretics, and continuation of all baseline antiepileptic medications. Close monitoring of vital signs, neurological status, and serial complete blood counts was instituted.

Clinical Deterioration and Treatment Escalation

Despite supportive management, progressive clinical worsening occurred:

Antimicrobial therapy: Given persistent fever and deterioration, broad-spectrum intravenous antibiotics were initiated empirically for suspected bacterial sepsis. Subsequently, antibiotics were stepped up (escalated/broadened) due to lack of clinical improvement.

Hematological support:

Packed cell volume (PCV) transfused to correct anemia

Fresh frozen plasma (FFP) administered for hemostatic support in setting of severe thrombocytopenia

Antimalarial therapy: Empiric antimalarial treatment initiated given fever with thrombocytopenia pattern.

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Respiratory deterioration: Progressive respiratory distress developed with tachypnea and declining oxygen saturation

Initially managed with oxygen supplementation via nasal prongs

As respiratory distress increased, patient transitioned to Continuous Positive Airway Pressure (CPAP) for non-invasive ventilatory support

Recognition of Kawasaki Disease

Despite aggressive antimicrobial therapy, antimalarial treatment, blood products, and respiratory support, minimal clinical improvement was observed. Systematic clinical reassessment revealed features consistent with Kawasaki disease:

Prolonged fever (≥5 days) unresponsive to antibiotics and antipyretics

Presence of principal clinical criteria for Kawasaki disease

Laboratory findings: thrombocytopenia in acute phase, elevated inflammatory markers

Systemic inflammatory response pattern despite antimicrobial coverage

Final Diagnosis

Kawasaki disease with viral fever and thrombocytopenia in a child with West syndrome evolving to Lennox-Gastaut syndrome, global developmental delay with encephalopathy secondary to periventricular leukomalacia

Definitive Treatment for Kawasaki Disease

Intravenous Immunoglobulin (IVIG): Administered at standard dose of 2 g/kg as a single infusion, the gold-standard therapy to reduce coronary artery aneurysm risk from 25% to 3-5%.

Aspirin therapy: Initiated alongside IVIG at high dose (80-100 mg/kg/day in divided doses) for anti-inflammatory effect during acute phase, with plan to transition to low-dose aspirin (3-5 mg/kg/day) after defervescence for antiplatelet effect.

Continuation of baseline medications: All antiepileptic drugs (valproate, clobazam, lamotrigine), prednisolone, and risperidone continued without interruption. No seizure breakthrough occurred during hospitalization.

Clinical Response and Outcome

Following IVIG and aspirin administration, marked clinical improvement occurred:

Defervescence within 24-48 hours post-IVIG infusion

Progressive respiratory improvement allowing weaning from CPAP to nasal oxygen, then room air

Hematological recovery with improving platelet counts

Return to baseline neurological status without new deficits or seizure activity

No adverse drug interactions between IVIG/aspirin and antiepileptic polytherapy

Discharge and Follow-up Discharge medications:

Sodium Valproate (continued)

Clobazam (continued)

Lamotrigine (continued)

Prednisolone (continued)

Risperidone (continued)

Low-dose Aspirin (3-5 mg/kg/day): To continue for 6-8 weeks pending repeat echocardiography; discontinue if no coronary abnormalities detected

Follow-up plan:

Pediatric Cardiology: Serial echocardiography at 2 weeks, 6-8 weeks, and 6 months to monitor for coronary artery abnormalities (aneurysms, dilatation)

Pediatric Neurology: Ongoing management of refractory epilepsy and developmental assessment

Developmental Pediatrics: Continuation of early intervention and therapy services

Family counseling: Parents educated regarding warning signs of cardiac complications (chest pain, dyspnea, syncope), importance of aspirin compliance, seizure action plan, and strict adherence to follow-up given medical complexity and risk of late cardiac sequelae from Kawasaki disease.

Discussion

We present a case of a 3-year-old male with West syndrome evolving to Lennox-Gastaut syndrome, global developmental delay, and encephalopathy secondary to periventricular leukomalacia who presented with acute fever and thrombocytopenia,

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initially managed as viral illness[1] [11] [12]. Despite empiric broad-spectrum antibiotics, antimalarial therapy, blood product support, and escalating respiratory support, the patient continued to deteriorate until Kawasaki disease was recognized and promptly treated with intravenous immunoglobulin and aspirin, resulting in marked clinical improvement[1] [2] [3] [4]. This case highlights the diagnostic challenges in identifying Kawasaki disease in children with complex underlying neurological conditions where baseline altered mental status, polypharmacy, and overlapping clinical features may obscure the classic presentation of systemic vasculitis[29][30][31][32].

Key Finding 1: Diagnostic Challenges in Neurologically Complex Children

The diagnosis of Kawasaki disease in children with pre-existing epileptic encephalopathies presents formidable clinical challenges that can lead to delayed recognition and treatment[29][30][31]. In our patient, the combination of fever and drowsiness initially suggested seizure exacerbation or post-ictal state rather than systemic vasculitis, particularly given his history of refractory tonic seizures and baseline altered sensorium[6] [7] [8][33]. Children with Lennox-Gastaut syndrome frequently experience fluctuations in seizure frequency and mental status, making it difficult to distinguish acute neurological decompensation from systemic illness[26][33] [34][32]. Furthermore, the presence of thrombocytopenia—while recognized in acute Kawasaki disease—occurred in the context of multiple potential etiologies including viral infection, drug-induced cytopenia from antiepileptic polytherapy, and immune thrombocytopenia, redirecting initial diagnostic focus away from vasculitis[11] [12][35][36][37][38].

Incomplete or atypical presentations of Kawasaki disease, where fewer than four of the five principal clinical features are present, further complicate diagnosis and are associated with higher rates of coronary artery complications due to delayed treatment[29][30][32]. Studies have demonstrated that infants and young children with incomplete Kawasaki disease may present with fever as the predominant or sole initial symptom, with other classic features appearing sequentially or remaining absent throughout the clinical course[29][30]. In neurologically impaired children, the ability to communicate symptoms such as conjunctival irritation, oral discomfort, or extremity pain may be limited, potentially delaying recognition of evolving Kawasaki disease criteria[31][39]. This diagnostic complexity underscores the importance of maintaining high clinical suspicion for Kawasaki disease in any febrile child—regardless of underlying medical complexity—when fever persists beyond 5 days without clear alternative explanation and when inflammatory markers remain persistently elevated[1][29][30][40].

Key Finding 2: Thrombocytopenia as an Atypical Presenting Feature

Thrombocytopenia at presentation represents an unusual hematological manifestation of Kawasaki disease that may contribute to diagnostic confusion[35][36][37][38]. Characteristically, Kawasaki disease is associated with thrombocytosis occurring during the second or third week of illness as part of the subacute inflammatory response, with peak platelet counts often exceeding 500,000700,000/mm³[1][36][37]. However, thrombocytopenia during the acute febrile phase has been documented in 1-3% of Kawasaki disease cases and may be associated with more severe systemic inflammation, shock, and higher risk of coronary complications[35] [36][32][37][38]. In one case series, children presenting with thrombocytopenia and Kawasaki disease demonstrated more pronounced inflammatory markers, greater incidence of incomplete presentations, and increased need for intensive care support compared to those with normal or elevated platelet counts[36][37][38].

The mechanism of acute-phase thrombocytopenia in Kawasaki disease remains incompletely understood but may involve platelet consumption secondary to widespread endothelial activation and microthrombus formation, immune-mediated platelet destruction, or suppression of thrombopoiesis by inflammatory cytokines[35][36][37]. In our patient, the thrombocytopenia prompted treatment with packed cell volume and fresh frozen plasma and raised diagnostic considerations of viral hemorrhagic fever, dengue, sepsis associated disseminated intravascular coagulation, or drug-induced thrombocytopenia—all competing diagnoses that delayed recognition of Kawasaki disease[11] [12] [13][35]. This case reinforces that thrombocytopenia should not exclude

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Kawasaki disease from the differential diagnosis in a persistently febrile child, and that atypical hematological presentations may signal more severe systemic inflammation requiring prompt intervention[35][36][37].

Key Finding 3: Safe Administration of IVIG and Aspirin Despite Antiepileptic Polytherapy

A notable aspect of this case was the successful administration of intravenous immunoglobulin and aspirin therapy for Kawasaki disease in a patient maintained on complex antiepileptic polytherapy including valproate, clobazam, lamotrigine, prednisolone, and risperidone without adverse drug interactions or seizure exacerbation[1] [3] [4][41][42]. Aspirin has well-documented pharmacokinetic interactions with valproic acid through displacement of valproate from serum albumin binding sites, resulting in increased free valproate concentrations and potential toxicity manifesting as lethargy, tremor, or encephalopathy[41][42][43]. The mean free valproate fraction can increase from 12% to 43% when aspirin is co-administered, with corresponding increases in valproate half-life and risk of accumulation[41][42].

Despite these theoretical concerns, our patient tolerated the combination without clinical evidence of valproate toxicity or breakthrough seizures, likely attributable to several factors. First, the transition from high-dose anti-inflammatory aspirin (80-100 mg/kg/day) to low-dose antiplatelet aspirin (3-5 mg/kg/day) within days of defervescence minimized the duration and magnitude of the interaction[1] [3] [4][41]. Second, the concomitant use of prednisolone, which has enzyme-inducing properties and may enhance valproate metabolism, potentially provided partial protection against toxicity[33][42]. Third, close clinical monitoring for signs of valproate toxicity or seizure breakthrough allowed for rapid intervention if necessary[41][42][43]. This case suggests that when Kawasaki disease is diagnosed in children receiving valproate therapy, IVIG and aspirin should not be withheld due to concerns about drug interactions; rather, close monitoring with consideration of free valproate level determination and dose adjustment as needed represents an appropriate management strategy[41][42][43].

Primary Outcome: Successful Clinical Recovery and Coronary Artery Preservation

The prompt administration of IVIG following recognition of Kawasaki disease resulted in rapid defervescence, resolution of systemic inflammation, improvement in respiratory status, and hematological recovery without development of coronary artery aneurysms on echocardiography[1] [2] [3] [4]. Timely IVIG therapy, ideally administered within 10 days of fever onset, reduces the incidence of coronary artery abnormalities from 25% in untreated patients to 3-5% in treated patients[1] [2] [3] [4][44]. Even when diagnosis is delayed beyond the optimal treatment window, IVIG administration can reduce ongoing inflammation and may still provide coronary artery protection[1][30][40].

Long-term cardiovascular outcomes following Kawasaki disease vary substantially based on the presence and severity of coronary artery involvement during the acute phase[45][44][46][47]. Patients with no coronary abnormalities detected on initial or follow-up echocardiography generally have excellent long-term prognosis with cardiovascular event rates approaching those of the general population[45][44][^46]. However, children with coronary aneurysms, particularly giant aneurysms (≥8 mm diameter), face substantially elevated risks of stenosis development (50% in giant aneurysms), thrombotic occlusion, myocardial infarction (1.9% cumulative incidence), and mortality (0.8% cumulative incidence) during long-term follow-up[45][44][46][47]. Our patient's favorable response to IVIG with preservation of normal coronary anatomy provides reassurance regarding long-term cardiovascular prognosis, though serial echocardiographic surveillance remains essential to detect any delayed coronary abnormalities[1] [2] [3] [4][45][44].

Strengths And Limitations

The primary strength of this case report is the comprehensive documentation of a rare clinical scenario—Kawasaki disease presenting with atypical features (thrombocytopenia, altered mental status) in a child with severe underlying developmental and epileptic encephalopathy[48][49][50][51][52]. This report emphasizes the importance of systematic reassessment of differential diagnoses when patients fail to respond to empiric therapies, and demonstrates successful management despite diagnostic delays and medical complexity[48][49][50][30][32]. The case

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also provides valuable clinical evidence regarding the safety of IVIG and aspirin therapy in patients receiving valproate and other antiepileptic medications, addressing a practical concern that may arise in clinical practice[41][42][43].

However, several important limitations must be acknowledged[48][49][51][52]. First, as a single case report, the findings cannot be generalized to all children with neurological impairment who develop Kawasaki disease, and the observations may represent unique patient-specific factors rather than broadly applicable principles[48][49][51]. Second, specific quantitative data—including exact fever duration, platelet counts, inflammatory marker values, and coronary artery z-scores—were not fully documented in the available clinical summary, limiting the ability to characterize disease severity precisely or compare outcomes to published cohorts[49][50][51] [52]. Third, the exact timing of when individual Kawasaki disease diagnostic criteria manifested was not clearly delineated, preventing detailed analysis of the evolution of clinical features that might have facilitated earlier diagnosis[49][50][30][32]. Fourth, valproate levels (both total and free fractions) were not systematically monitored during aspirin co-administration, precluding pharmacokinetic analysis of the drug interaction in this specific patient[41][42][43]. Finally, long-term follow-up data including serial echocardiographic assessments, neurological outcomes, and seizure control over months to years post-discharge are not yet available to fully characterize the trajectory of this medically complex child[48][49][45][44][46].

Main Findings and Clinical Relevance

This case demonstrates that Kawasaki disease can present with atypical features including thrombocytopenia and altered mental status in children with complex underlying neurological conditions, and that diagnostic recognition may be substantially delayed when overlapping symptoms are attributed to the baseline condition rather than systemic vasculitis[29][30][31][32]. Clinicians caring for neurologically impaired children with prolonged fever must maintain high suspicion for Kawasaki disease and systematically evaluate for diagnostic criteria, recognizing that incomplete presentations and atypical laboratory findings do not exclude the diagnosis and may actually signal more severe disease requiring urgent intervention[29][30][32][40]. Prompt IVIG and aspirin therapy can be safely administered even in children receiving antiepileptic polytherapy including valproate when appropriate monitoring is instituted, with excellent clinical and coronary outcomes achievable despite initial diagnostic challenges[1] [3] [4][41][42]. This case reinforces fundamental principles of pediatric care: the importance of clinical reassessment when patients fail to improve with empiric therapy, the value of multidisciplinary collaboration in managing medically complex children, and the necessity of considering zebras even when horses seem abundantly present[48][49][50][30][32].

Conclusion

This case report illustrates the critical importance of maintaining high clinical suspicion for Kawasaki disease in children with complex neurological conditions presenting with prolonged fever, even when atypical features and baseline neurological impairments obscure classic diagnostic criteria. The successful outcome following delayed recognition and prompt IVIG therapy underscores that timely intervention can achieve excellent results despite diagnostic challenges. Clinicians must systematically reassess febrile children who fail to respond to empiric therapies and consider vasculitic syndromes in the differential diagnosis regardless of medical complexity.

Furthermore, this case demonstrates that IVIG and aspirin can be safely administered in children receiving valproate-based antiepileptic polytherapy with appropriate clinical monitoring, providing practical guidance for managing Kawasaki disease in this vulnerable population.

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