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Review Article

Serum ferritin and cardiogenic shock in severe pediatric dengue: pathophysiology, evidence and clinical correlation - a narrative review

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ABSTRACT

Severe dengue infection in children can lead to life-threatening shock classically attributed to plasma leakage. However, emerging evidence links hyperinflammation – often reflected by extreme elevations in serum ferritin – with myocardial dysfunction and cardiogenic shock in dengue. Ferritin, an acute-phase reactant, is markedly elevated in hyperinflammatory syndromes such as hemophagocytic lymphohistiocytosis (HLH), which is increasingly recognized as a complication of severe dengue. This narrative review critically examines the pathophysiology connecting dengue virus infection, hyperferritinemia, and cardiac dysfunction. We summarize key studies demonstrating that serum ferritin levels correlate with dengue severity and outcomes, including recent pediatric cohorts where ferritin >10,000 ng/ml portended higher mortality. Myocardial involvement in dengue – ranging from transient functional impairment to fulminant myocarditis – is discussed in light of cytokine-mediated injury and HLH-like immune activation. We also outline clinical implications, recommending vigilance for "dengue-HLH" in severe cases with unexplained shock and very high ferritin, and considering adjunctive immunomodulatory therapy in addition to standard supportive care. State-of-the-art evidence is presented to guide clinicians in early identification of hyperinflammatory dengue cases, prompt cardiac support (inotropes, fluid management, and extracorporeal life support when needed), and potential interventions to improve outcomes. This review provides a comprehensive, evidence-based update on the role of serum ferritin as both a biomarker and a clue to pathogenic processes (including HLH) that can culminate in cardiogenic shock in pediatric dengue.

Keywords: Severe dengue infection, Hemophagocytic lymphohistiocytosis, Hyperferritinemia

INTRODUCTION

Dengue fever is a major mosquito-borne viral illness with a wide spectrum of presentations, from mild febrile illness to severe, life-threatening disease.[1] Only a small proportion of dengue infections progress to severe manifestations characterized by plasma leakage, hemorrhage, and organ impairment.[2] In pediatric patients, severe dengue is often marked by dengue hemorrhagic fever and dengue shock syndrome due to increased capillary permeability leading to hypovolemic shock.[1,2] Shock in classic dengue results primarily from intravascular volume loss; however, there is growing recognition that myocardial dysfunction can also occur in dengue and exacerbate hemodynamic compromise.[2] Dengue can directly or indirectly affect the heart, with reported cardiac manifestations including arrhythmias, cardiac muscle impairment, and even fulminant myocarditis in rare cases.[2,3] Such cardiac involvement may contribute to refractory shock beyond what would be expected from plasma leakage alone.

Parallel to these developments, attention has turned to serum ferritin as a potential marker of severe dengue infection. Ferritin is an iron storage protein as well as an acute-phase reactant that rises in inflammatory states.[4] Strikingly high ferritin levels are a hallmark of hyperinflammatory syndromes such as hemophagocytic lymphohistiocytosis (HLH) and macrophage activation syndrome.[5] In the context of dengue, numerous studies

have found that higher ferritin levels correlate with greater disease severity.[4,5] This has led to the hypothesis that a subset of children with severe dengue may experience an "HLH-like" cytokine storm, sometimes termed dengue-associated HLH, which can drive both multi-organ failure and myocardial depression.

This review explores the pathophysiologic links between dengue infection, hyperferritinemia, and cardiogenic shock in children. We critically appraise the evidence base for ferritin as a prognostic biomarker in dengue, examine how hyperferritinemia might reflect and contribute to cardiac dysfunction, and discuss clinical correlations including recognition and management of dengue-induced hyperinflammatory syndromes. By synthesizing current evidence, we aim to provide clinicians with a state-of-the-art understanding of how serum ferritin can inform risk stratification and therapeutic decisions in severe pediatric dengue.

PATHOPHYSIOLOGY: DENGUE, HYPERFERRITINEMIA AND SHOCK

Severe dengue's hallmark is increased capillary permeability caused by endothelial dysfunction, leading to plasma leakage, hemoconcentration, and shock.[1] This vascular leak is thought to result from a complex interplay of viral factors and host immune responses. High levels of pro-inflammatory cytokines (often referred to as a "cytokine storm") have been documented in severe dengue, including tumor necrosis factor (TNF-α), interleukins (IL-1β, IL-6, IL-18), and others that contribute to endothelial activation and coagulopathy.[2,5]

The intense immune activation not only causes plasma leakage but can also depress myocardial function through circulating myocardial depressant factors and subclinical myocarditis.[2] Autopsy studies and clinical reports indicate dengue virus may infect cardiac myocytes in rare instances, and cardiac tissue can exhibit edema and inflammatory cell infiltration during severe infection.[2] Thus, the hemodynamic collapse in dengue may be multifactorial – primarily hypovolemic (distributive) shock from plasma leakage, compounded in some cases by cardiogenic components due to myocardial involvement.

Ferritin plays a notable role in this immunopathology. Under normal conditions, ferritin safely sequesters iron; during inflammation, ferritin synthesis is upregulated as part of the acute-phase response.[4] Cytokines such as IL-1, IL-6, and TNF-α stimulate ferritin production, and high ferritin levels in turn may contribute to immune dysregulation by modulating iron availability and directly suppressing hematopoiesis.[4,6]

In dengue infection, studies have consistently found hyperferritinemia (often defined as ferritin ≥500 ng/ml) in patients with more severe disease.[5] Van de Weg et al reported that dengue patients had significantly higher ferritin levels than those with other febrile illnesses, and ferritin elevations were strongly associated with markers of immune activation (e.g. elevated soluble IL-2 receptor) and coagulopathy.[5] This suggests that ferritin is not merely a bystander, but an indicator of the magnitude of the inflammatory response in dengue. Hyperferritinemia in dengue also correlated with viral load (viremia) and occurred more frequently in patients with warning signs or severe dengue compared to uncomplicated cases.[5]

Extreme ferritin elevations raise the consideration of HLH, a hyperinflammatory syndrome that can be triggered by severe infections. Secondary HLH (acquired HLH) is characterized by unregulated immune activation with fever, cytopenias, organomegaly, and very high ferritin, among other criteria. Notably, dengue viral infection is a recognized trigger of HLH.[5] Ferritin levels in the tens of thousands are typical in HLH; one landmark study in pediatric patients found that a ferritin >10,000 ng/ml was ~90% sensitive and 96% specific for diagnosing HLH.[7] In adults, ferritin is less specific, but in children such extreme hyperferritinemia should prompt an evaluation for HLH or HLH-like phenomena.[6]

In the setting of dengue, "dengue-HLH" refers to cases meeting HLH criteria secondary to dengue infection. These patients experience an overwhelming cytokine storm (with interferon-γ, IL-10, IL-18 often elevated) that can lead to hemophagocytosis in bone marrow and multi-organ failure.

Myocardial dysfunction in HLH is well-documented in critical care settings, stemming from cytokine-induced stunning of the heart and occasionally macrophage infiltration of organs. Therefore, it is biologically plausible that in some dengue cases, hyperferritinemia signifies an HLH-like state wherein unchecked immune activation is a direct contributor to cardiac depression and shock.

SERUM FERRITIN IN SEVERE DENGUE: EVIDENCE AND CORRELATIONS

A growing body of clinical research underscores the value of serum ferritin as a marker of dengue severity. Multiple studies across different settings have shown that higher ferritin levels are associated with more severe disease courses, including development of shock, organ failure, and death.

Early studies

Pioneering investigations such as Soundravally et al in 2015 found that serum ferritin levels were significantly elevated in patients with severe dengue compared to those with non-severe dengue.

Ferritin levels >1000 ng/ml were strongly predictive of progression to severe dengue.[8] This early insight suggested ferritin could serve as an early warning biomarker for clinicians.

Prospective cohorts

Murmu et al in 2021 conducted a prospective study in a tertiary care center in India to evaluate ferritin as a prognostic tool. They noted a clear gradation of ferritin levels: patients with dengue fever without warning signs had median ferritin ~500 ng/ml, those with warning signs ~1000 ng/ml, and those with severe dengue ~2350 ng/ml by day 3 of illness.[4] By day 7, ferritin remained highest in the severe dengue group (median ~2950 ng/ml). Importantly, they identified ferritin cut-off values for risk stratification. For instance, a serum ferritin of 1247 ng/ml on day 3 of fever had ~96% sensitivity and 91% specificity for predicting severe dengue.[4] Similarly, a day-7 ferritin of ~1050 ng/ml had ~98% sensitivity and 93% specificity for severe disease.[4] These high sensitivities and specificities highlight ferritin's potential as an early predictor of which patients might deteriorate.

Critical care studies

In children requiring intensive care for dengue, ferritin has also emerged as a key marker. A recent single-center pediatric ICU cohort by Jha et al in 2025 measured peak ferritin levels during hospitalization for severe dengue infection (SDI). Among 144 children in their study, the median peak serum ferritin was 6732 ng/ml (range ~2,800 to ~17,890).[9] Strikingly, >90% of these critically ill children had hyperferritinemia above 500 ng/ml, and a subset (35% of patients) exceeded 10,000 ng/ml.[9]

The outcomes in this cohort showed that children with extraordinarily high ferritin were at significantly greater risk: a peak ferritin >10,000 ng/ml was associated with a higher incidence of dengue-related complications and had a strong statistical association with mortality ($p<0.05$).[9] Using receiver-operating characteristic (ROC) analysis, the authors found that ferritin was a good predictor of fatal outcome – for example, a cut-off ~15,691 ng/ml had an area under the curve of 0.826 for predicting mortality.[9] Their conclusion was that hyperferritinemia is common in severe pediatric dengue, and extreme elevations (above 10,000 ng/ml) portend a markedly higher risk of adverse outcomes including death.[9]

Other studies

Investigations in other countries and age groups echo these findings. For instance, a study in Vietnam and Aruba by van de Weg et al in 2014 noted that dengue patients consistently had higher ferritin than those with other febrile illnesses, and those with severe dengue (according to World Health Organization 2009 criteria) had the highest ferritin levels.[5]

Ferritin levels were not only a marker of severity but also part of the discriminatory profile that could help distinguish dengue infection from other infections in endemic settings.[5] Similarly, researchers in Thailand have reported that elevated ferritin levels accompany severe dengue and may correlate with liver enzyme elevations and coagulopathy, reflecting the broader inflammatory impact of the virus.

These studies are summarized in Table 1. Collectively, the evidence strongly supports serum ferritin as a readily available laboratory marker that correlates with the severity of dengue illness. High ferritin likely signifies an intense immune response which, in turn, relates to greater capillary leakage, organ impairment, or even secondary HLH. It is important to note that while ferritin can aid in prognostication, it should be interpreted in the clinical context – e.g. trending ferritin levels over the course of illness and alongside other markers like liver enzymes, complete blood counts, and inflammatory markers.

Beyond serving as a biomarker, ferritin in severe dengue may also have direct pathophysiologic implications. Very high ferritin levels can induce a pro-inflammatory feedback loop (sometimes referred to as part of the "hyperferritinemic syndrome") wherein ferritin itself promotes further cytokine release and immune dysregulation. This has been described in conditions like HLH, adult-onset Still's disease, and severe sepsis.[5] In dengue patients, particularly those who fulfill criteria for HLH, ferritin may thus be more than a marker – it could be contributing to the ongoing inflammation and organ damage, including to the heart.

CARDIOGENIC SHOCK IN SEVERE DENGUE

Cardiac involvement in dengue has transitioned from a fringe consideration to an area of active research. While most dengue shock is due to hypovolemia, clinicians should be aware of the evidence that dengue can cause myocardial dysfunction which, in extreme cases, leads to cardiogenic shock or a mixed shock state. Several lines of evidence support this.

Clinical and echocardiographic studies

A prospective study by Kirawittaya et al in Thai children used serial echocardiography to assess cardiac function in dengue. It demonstrated that during the critical phase of dengue (around defervescence when plasma leakage peaks), patients had transiently decreased left ventricular function.[1] Children with dengue hemorrhagic fever (DHF) had lower cardiac index and impaired ventricular relaxation compared to those with dengue fever, suggesting that cardiac function is indeed affected at the height of plasma leak.[1]

The myocardial impairment observed was generally reversible and did not typically require intervention, but it provides proof that dengue causes a degree of myocardial depression in many patients.[1] Notably, this study found elevated cardiac troponin levels in a subset of severe cases, indicating myocardial injury.[1] The authors concluded that although fulminant myocarditis is uncommon, subtle

cardiac dysfunction is common in severe dengue and correlates with severity of plasma leakage.[1]

Case reports of myocarditis

There are growing reports of acute myocarditis caused by dengue virus, often in adolescents or children. Bagde (2016) reported a case of a 16-year-old girl with dengue who developed fulminant myocarditis and cardiogenic shock requiring veno-arterial extracorporeal membrane oxygenation (ECMO) support.[10] Despite aggressive management with fluids, inotropes (dopamine, dobutamine, epinephrine), and mechanical ventilation, the patient remained in refractory shock with depressed cardiac output (left ventricular ejection fraction fell to 45% and then much lower). The timely initiation of ECMO provided cardiac and respiratory support, allowing the myocardium to recover. The patient was successfully weaned off ECMO after 6 days and eventually recovered with normal cardiac function. This case underscores that fulminant dengue myocarditis, while rare, can be life-threatening and may necessitate advanced cardiac life support techniques. Other case reports and series have described complete heart block, arrhythmias, and acute heart failure in dengue patients as well, highlighting that dengue can directly cause severe cardiac events.[10,11]

Table 1: Select studies examining serum ferritin levels in dengue infection.

Study (year)	Population	Ferritin findings	Clinical outcome correlation
Soundravally et al (2015)[8]	Adults and children with dengue (n=150)	Ferritin >1000 ng/ml on admission was predictive of severe dengue	Higher ferritin in severe versus non-severe cases; proposed as early severity biomarker
Murmu et al (2021)[4]	Pediatric dengue cases, prospectively followed	Median ferritin (day 3): ~500 ng/ml (DF), 1000 ng/ml (DHF), 2352 ng/ml (severe); day-3 cutoff ~1247 ng/ml (96% sens, 91% spec) for severe dengue	Ferritin levels tracked with disease severity; early ferritin elevation strongly predicted progression to severe dengue
Jha et al (2025)[9]	PICU patients with severe dengue (n=144)	Median peak ferritin ~6732 ng/ml; 35% had ferritin >10,000 ng/ml; ferritin >10,000 ng/ml significantly associated with complications and mortality	Hyperferritinemia very common in critically ill dengue children. Peak ferritin >10k was a strong predictor of higher mortality risk (AUC~0.83 for death)

DF: dengue fever (no warning signs); DHF: dengue hemorrhagic fever (with warning signs or plasma leakage); PICU: pediatric intensive care unit

Epidemiological signals

In large cohorts, the contribution of myocardial dysfunction to dengue mortality has been difficult to quantify, but autopsy series and critical care observations suggest it plays a role. For example, retrospective analyses of dengue deaths have noted myocardial inflammation on histology in some cases, and clinically, a proportion of dengue shock syndrome patients require vasoactive support beyond fluid resuscitation, implying a component of cardiac impairment. Yacoub et al in a pointed out that myocardial involvement in dengue is “occasionally” severe (fulminant myocarditis) but functional myocardial impairment and ECG abnormalities are observed in many hospitalized dengue patients. They postulate that mechanisms include a possible direct viral effect on the myocardium, edema within the heart muscle, and circulating depressant factors such as cytokines. Dengue’s effect on the heart is therefore usually subclinical or mild, but can tip the balance in a critically ill patient.[2]

Given the above, cardiogenic shock in dengue should be suspected when a patient’s hemodynamics do not improve with appropriate fluid resuscitation, or when there are signs of cardiac involvement such as gallop rhythm, marked tachycardia disproportionate to fever, muffled heart sounds, or elevated biomarkers (troponin, BNP). Invasive hemodynamic monitoring or bedside echocardiography can be invaluable in such cases to distinguish purely hypovolemic shock from mixed shock. It’s worth noting that in children, distinguishing these can be challenging without an echocardiogram; thus, a low threshold for cardiac imaging in severe dengue with refractory shock is warranted.

Link to hyperferritinemia

How does the concept of hyperferritinemia tie into cardiogenic shock? The connection is primarily via the hyperinflammatory state. When ferritin is extremely elevated (e.g. in dengue-associated HLH), the levels of inflammatory cytokines like interferon-γ, IL-6, IL-1, and TNF-α are also expected to be very high.[12] These cytokines have known negative inotropic effects and can cause vasodilation and capillary leak. IL-6, for example, has been implicated in myocardial depression in sepsis; TNF-α can induce nitric oxide production leading to myocardial suppression. In HLH, the overactive immune cells (lymphocytes and macrophages) not only produce these cytokines but may also infiltrate organs. Dengue-triggered HLH can therefore present as a septic shock mimic, with poor cardiac output and systemic inflammatory response despite lack of ongoing infection.[12] Clinicians in dengue-endemic areas have observed that some children with dengue shock fail to improve with standard therapy because they are essentially in a hyperinflammatory shock

state rather than a pure volume-depleted state.[12] These children often have ferritin levels in the tens of thousands along with prolonged fever and cytopenias, pointing toward an HLH-like picture.[12] As Dr. Patil, a pediatric intensivist, noted in Pune (India), "a disproportionately high ferritin level and persistent drop in platelet count should prompt further investigations for HLH". He reported seeing pediatric dengue shock cases with ferritin as high as 50,000 ng/l who developed significant hyper-inflammatory responses involving cardiac dysfunction and coagulopathy, worsening the shock state.[12] This real-world observation aligns with the notion that beyond a certain point, the dengue illness behaves like an HLH cytokine storm – and managing it requires more than fluids and supportive care.

CLINICAL IMPLICATIONS AND MANAGEMENT

The recognition that hyperferritinemia and hyperinflammation contribute to some cases of severe dengue has practical clinical implications.

Monitoring ferritin and inflammatory markers

In children hospitalized with severe dengue or dengue shock, it is advisable to check serum ferritin levels, especially if the clinical course is not following the typical trajectory (for instance, if fever persists beyond the defervescence period or the child's condition is deteriorating despite adequate fluid management). Ferritin is an accessible test and can often be resulted within hours. A very high ferritin (e.g. above 5,000-10,000 ng/ml) in a dengue patient should raise concern for an overwhelming inflammatory syndrome such as HLH.[12,7]

Additional tests to consider include triglyceride levels, fibrinogen, liver function tests, complete blood counts (to look for cytopenias), and markers of organ dysfunction. These help in applying HLH diagnostic criteria (five of eight classic criteria, which include ferritin ≥500 ng/ml, fever, cytopenias, hypertriglyceridemia, hypofibrinogenemia, hemophagocytosis on marrow/spleen/lymph node biopsy, low NK cell activity, high soluble IL-2 receptor).

Early identification of dengue-associated HLH

Given that dengue can trigger HLH, a high index of suspicion is needed. Persistent or recurrent fever beyond the typical defervescent phase (usually dengue fever lasts 5–7 days) is a red flag, as HLH often presents with ongoing fever.[13] Unexplained cytopenias (especially if the hematocrit is not rising despite plasma leakage resolving, or if white cell count drops significantly) also suggest HLH. If dengue-associated HLH is suspected (for example, dengue patient with ferritin in the thousands plus organomegaly and cytopenias), many experts advocate early intervention rather than waiting for all criteria to be met.[12] In practical terms, this might mean consulting a pediatric hematologist or intensivist and starting therapies used for HLH.

Immunomodulatory therapy

The mainstay of dengue treatment is supportive care, as there are no specific antivirals. However, in the subset of patients where a hyperinflammatory state is driving disease (evidenced by hyperferritinemia and possible HLH), immunomodulatory treatment can be lifesaving. Corticosteroids are often the first-line immunosuppressive agents used in secondary HLH associated with infections. Indeed, there are case reports of dengue-HLH patients who responded well to high-dose corticosteroids (dexamethasone or methylprednisolone) when started early.[12] IV immunoglobulin (IVIG) is another therapy used in HLH (especially when suspicion for underlying macrophage activation is high); it has been used in some dengue-HLH cases by extrapolation from other viral HLH scenarios. The threshold for initiating such treatment in dengue is not firmly established, but many clinicians would treat if a patient fulfills HLH-2004 criteria or is rapidly worsening with evidence of cytokine storm. The potential benefit of immunomodulators must be balanced against risks (steroids can exacerbate hyperglycemia, infections; IVIG is expensive), but given the high mortality of unmitigated HLH, early therapy is justified when clinical signs point in that direction.[12] An "HLH-like" approach (steroids/IVIG) in severe dengue is an evolving area – no randomized trials yet – but case series suggest improved outcomes with timely intervention.[12]

Cardiac support

For patients with suspected myocardial involvement, management aligns with standard heart failure and shock protocols. This includes judicious fluid management (to avoid fluid overload in the setting of potential myocardial dysfunction), use of inotropes and vasopressors as needed, and mechanical ventilation if respiratory failure or significant pulmonary edema occurs. Echocardiography can guide inotrope choice (e.g., if poor contractility, an inotrope like milrinone or dobutamine might be used; if vascular tone is low, vasopressors like norepinephrine are added). In extreme cases, as illustrated by the ECMO-survived myocarditis case, advanced therapies such as ECMO should be considered.[10]

Pediatric centers with ECMO capabilities can support patients through the acute phase of myocarditis when conventional therapy fails. Thus, a child with dengue who develops refractory shock despite fluids and high-dose vasopressors should be urgently evaluated for transfer to a center with ECMO, if available.[10] The decision should be made early, as prolonged severe shock can lead to irreversible multiorgan damage. The recovery of the 16-year-old on ECMO in the case report demonstrates that even fulminant dengue myocarditis can be survivable with timely advanced support.[10]

Follow-up and monitoring

Children who suffer severe dengue with hyperferritinemia and cardiac involvement should have follow-up after recovery. Some may have lingering effects on heart function or other organ systems. Most, however, if they survive the acute illness, recover fully without long-term sequelae to the heart, as documented in the ECMO case (normal cardiac function on follow-up).[10]

Monitoring ferritin trends to ensure they normalize (to rule out an underlying primary HLH or other ongoing inflammation) can be considered.

CLINICAL RECOMMENDATION: RECOGNITION AND MANAGEMENT OF HYPERINFLAMMATORY DENGUE

Assess ferritin in severe dengue

Check serum ferritin in any child with severe dengue, especially if shock is disproportionate or not responding rapidly to fluids. Marked hyperferritinemia (e.g. >5,000 ng/ml) should prompt consideration of dengue-associated HLH.[7,12]

Suspect HLH in unusual course

If the child has persistent fever >7 days, cytopenias, organomegaly, or unexplained worsening after defervescence, apply HLH criteria. High ferritin plus these features = high suspicion.[5,12] Consider bone marrow examination for hemophagocytosis if feasible.

Early immunosuppression

Don't hesitate to initiate immunomodulatory therapy (e.g. dexamethasone 10 mg/m²/day or IVIG) in a probable dengue-HLH case before laboratory confirmation of every criterion.[12] Early treatment can be lifesaving by dampening the cytokine storm.

Optimize cardiac support

In a dengue patient with shock, use bedside ultrasound to evaluate cardiac function. If poor contractility is noted or if shock persists despite adequate volume, start inotropes as needed (dobutamine/milrinone for cardiac dysfunction; add vasopressor like norepinephrine if vascular tone is low). Avoid fluid overload to prevent pulmonary edema.[1]

Escalate care for myocarditis

For refractory shock with suspected myocarditis, arrange urgent transfer to an intensive care unit with capability for invasive monitoring and ECMO.[10] ECMO or advanced life support should be considered early in fulminant dengue myocarditis – it can stabilize the patient and allow time for myocardial recovery.[10]

CONCLUSION

Severe pediatric dengue is a complex multisystem disease. While plasma leakage and hypovolemic shock have long been the central focus, it is now evident that an immune-mediated hyperinflammatory process contributes significantly to morbidity and mortality in a subset of patients. Serum ferritin has emerged as a key biomarker linking these phenomena – acting as a surrogate for immune activation severity and helping unmask cases of dengue-associated HLH. Patients with extraordinarily high ferritin levels form a distinct high-risk group where conventional supportive care may not suffice without adjunctive immunosuppressive treatment. At the same time, dengue's impact on the heart – from transient dysfunction to cardiogenic shock – must be recognized, as timely cardiac support (including inotropes or ECMO) can make the difference between life and death in fulminant cases.

In this review, we have compiled evidence that serum ferritin not only correlates with severe dengue outcomes but also correlates with the presence of a hyperinflammatory state that can precipitate cardiac dysfunction. Ferritin thus serves as a bridge between the pathophysiology (e.g. cytokine storm, HLH) and clinical practice (risk stratification and management decisions). Maintaining a critical, evidence-based approach, clinicians are advised to integrate ferritin measurements into the care of severe dengue, remain vigilant for signs of HLH or myocarditis, and treat accordingly. Future research is needed to establish optimal ferritin cut-offs for interventions and to evaluate therapies (such as steroids or other anti-inflammatory agents) in dengue-related hyperinflammation. Until then, an individualized approach guided by diligent monitoring and supported by insights from current evidence will offer the best chance of improving outcomes for children with this dangerous manifestation of dengue.

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