RESEARCH PAPER

When Typhoid Fever Unmasks the Immune Storm: Secondary Hemophagocytic Lymphohistiocytosis with Junctional Bradycardia, Cranial Nerve Palsy, and Bilateral Lower Limb Weakness - A Case Report from Southern India

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ABSTRACT

Background: Secondary hemophagocytic lymphohistiocytosis (HLH) triggered by Salmonella Typhi is exceedingly rare, with fewer than thirty well-characterised adult cases globally. Unlike other bacterial triggers, enteric fever presents a specific diagnostic trap - its features of sustained fever, hepatosplenomegaly, and cytopaenia are indistinguishable from early HLH, and the immune storm it unleashes can be fatal if attributed solely to infection.

Case Summary: A 24-year-old man was admitted with five days of fever, loose stools, bilateral lower limb weakness, left ptosis, and a maculopapular rash. Tri-lineage cytopaenia worsened despite empirical antibiotics. Dengue, HIV, HBsAg, and leptospira were excluded. Widal serology confirmed Salmonella Typhi (O and H 1:160). Serum ferritin exceeded 1,650 ng/mL, triglycerides were 268 mg/dL, AST 426 U/L, and LDH 950 IU/L. Splenomegaly was confirmed on USG. Five of seven HLH-2024 criteria were fulfilled and the HScore was approximately 187, corresponding to greater than 93% HLH probability. Echocardiography showed EF 62% with no pericardial effusion; cardiac biomarkers were negative. On Day 6, junctional bradycardia at 55 bpm prompted emergency pacing referral. Management included Inj. Ceftriaxone, Inj. Metronidazole, PRBC and platelet transfusions, and Inj. Dexamethasone 8 mg TDS from Day 3.

Conclusion: This case underlines that in any typhoid patient who is worsening rather than improving, secondary HLH must be the leading differential. The HScore offers immediate probabilistic guidance from standard ward tests, cardiac monitoring is non-negotiable, and corticosteroids must accompany antibiotics once HLH is established.

Keywords: hemophagocytic lymphohistiocytosis; secondary HLH; Salmonella Typhi; enteric fever; HScore; HLH-2024; junctional bradycardia; pancytopenia; hyperferritinaemia; cranial nerve palsy; tropical infection

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1. Introduction

Hemophagocytic lymphohistiocytosis is a syndrome of unbridled immune activation in which the body's own macrophages and cytotoxic lymphocytes, driven beyond the bounds of regulation by an external trigger, turn against the haematopoietic compartment. [1] Secondary HLH complicating enteric fever is rare - fewer than thirty well-documented adult cases exist in the English-language literature - with the most recent reports published in the Oxford Medical Case Reports in December 2024 [3] and in Cureus in January 2026. [4] What makes this particular trigger so treacherous is that severe typhoid fever and early secondary HLH are clinically nearly identical: both present with sustained fever, hepatosplenomegaly, relative leucopaenia, and systemic inflammation. The HLH-2004 criteria [5] and the 2024 revision by the Histiocyte Society, [6] alongside the HScore of Fardet and colleagues, [7] exist precisely because validated scoring tools are needed to distinguish between the two before bone marrow results or specialist assays are available.

2. Case Report

2.1 History and Presentation

A 24-year-old construction worker from Jharkhand, presented to our Emergency Department at SSSMCRI on 1 January 2026 (IP/26/000027) with five days of high-grade fever, five to six episodes of loose stools daily, progressive bilateral lower limb weakness for two days, drooping of the left eyelid, and blurring of vision in the left eye. His family

had noticed a rash over his chest. He had eaten food from a roadside stall approximately twelve days before admission. He had no comorbidities and no family history of blood disorders or immunodeficiency. He occasionally consumed alcohol.

2.2 Examination

On arrival: temperature 103°F, heart rate 136 bpm, blood pressure normal, SpO_2 98% on room air. A maculopapular rash was visible over the anterior chest - the rose spots of typhoid. Neurological examination revealed left upper eyelid ptosis, restricted left extraocular movements consistent with a partial third nerve palsy, and bilateral lower limb power MRC 4+/5 with intact deep tendon reflexes. Upper limb power and all other cranial nerves were intact; sensation was normal. A palpable spleen was confirmed on abdominal examination; chest auscultation was clear.

2.3 Investigations

Haematology (Day 1): haemoglobin 11.0 g/dL, WBC 1,410/mm³, platelets 0.61 Lakh/mm³ - tri-lineage cytopaenia at presentation. Peripheral smear: thrombocytopaenia and leucopaenia with atypical lymphocytes and giant platelets. By Day 2: haemoglobin 10.3, WBC 1,960/mm³, platelets 0.34 Lakh/mm³ - all three lineages worsening on antimicrobials. Day 4 (post-transfusion): haemoglobin 12.5, WBC 3,580/mm³, platelets 0.52 Lakh/mm³; PCV 34%, RBC 4.5 million/mm³, MCV 83, MCH 28, MCHC 34.

Serology and microbiology (Day 1): Widal - Salmonella Typhi O 1:160, H 1:160 (positive); Para Typhi A and B - negative. Dengue NS1, IgM, IgG - all negative. HIV - non-reactive. HBsAg - non-reactive. Leptospira IgG and IgM - both negative. Malaria parasite and microfilariae - negative. Blood, urine, and stool cultures - no growth. CRP positive 1:32 (>96 mg/L). Stool occult blood - positive on Day 2.

Biochemistry (Day 3): AST 426, ALT 269, ALP 321 U/L; total bilirubin 1.7 mg/dL (direct 0.6 mg/dL); albumin 3.3 g/dL; total protein 5.8 g/dL (A:G 1.3); uric acid 2.9 mg/dL; phosphorus 2.7 mg/dL. LDH 950 IU/L (reference 132–248 U/L). Urea 31, creatinine 0.9 mg/dL. Electrolytes: Na 142, K 3.5, Cl 105, Ca 8.6 mEq/L. Serum magnesium 3.5 mg/dL - normal. ESR 16 mm/hr.

Lipid profile (Day 3, NABL-accredited SSSMCRI laboratory, Request BY/26/000237): triglycerides 263 mg/dL (reference <150 mg/dL) - fulfilling the HLH hypertriglyceridaemia criterion. [1] Total cholesterol 155 mg/dL, HDL 54 mg/dL, LDL 48 mg/dL, VLDL 53 mg/dL. Thyroid function tests (Request BY/26/000238): Free T3 0.46 pg/mL (low; reference 0.7– 4.5), Free T4 1.65 ng/dL (normal), TSH 0.83 µIU/mL (normal) - euthyroid sick syndrome; no primary thyroid disease. [9]

ABG (Day 1): pH 7.44, pCO_2 20 mmHg, pO_2 95 mmHg, HCO_3^- 13.6 mmol/L, lactate 1.5 mmol/L, BE(ecf) −10.6, SO_2c 98% - compensated metabolic acidosis with respiratory alkalosis. ABG (Day 3): pH 7.45, pCO_2 24 mmHg, HCO_3^- 20 mmol/L, lactate 1.6 mmol/L - stable. Coagulation (Day 1): PT 11.8 s, APTT 38 s, INR 0.78 - normal.

Serum ferritin (Day 5, VRR Diagnostics, SID 31001364): greater than 1,650 ng/mL - the upper detection limit of the assay; the true value was certainly higher. [10]

Echocardiogram (02/01/2026, screening echo by Ms. Vishalini, Echo Technologist): HR 136 bpm; EF 62%; no regional wall motion abnormality; normal cardiac chambers and valves; mild mitral regurgitation; trivial tricuspid regurgitation; TRPG 19 mmHg, no PAH; no clot or pericardial effusion; IVC 1.2 cm - normal and collapsing; TAPSE 23 mm (normal RV function). Preserved biventricular function and absence of effusion excluded significant myocarditis and pericarditis as contributors to the haemodynamic picture.

Cardiac biomarkers (Day 5): CK-MB 12 U/L; Troponin I - negative. These confirmed that the junctional bradycardia subsequently observed was a conduction system event, not a myocardial injury event.

USG abdomen: splenomegaly (13.7 cm, normal echotexture, no focal lesions); liver 14.0 cm (normal); portal vein not dilated. CT brain (02/01/2026): no haemorrhage, no neuro-parenchymal abnormality - structural cause for neurological features excluded.

Day 6: on routine cardiac monitoring, the patient developed a junctional escape rhythm at 55 bpm. The ECG was flagged as a significant acute arrhythmia. No metabolic explanation was found: potassium 3.5 mEq/L, magnesium 3.5 mg/dL (confirmed on laboratory report BY/26/000236), no QT-prolonging agents in use. Cardiology was urgently consulted and the patient was referred for temporary transvenous pacing consideration.

Table 1. Laboratory Data and HLH-2024 Diagnostic Relevance

Investigation	Day 1	Day 2–3	Day 5–6	Diagnostic Relevance
Haemoglobin (g/dL)	11.0	10.3	12.5*	Cytopenia - HLH-2024
WBC (/mm³)	1,410	1,960	3,580*	Leucopaenia - HLH-2024
Platelets (Lakh/mm³)	0.61	0.34	0.52*	Thrombocytopaenia - HLH-2024
Ferritin (ng/mL)	-	-	**>1,650 (assay limit)**	≥500 µg/L - HLH-2024 criterion
Triglycerides (mg/dL)	-	263 ↑ (ref <150)	-	Hypertriglyceridaemia - HLH-2024
LDH (IU/L)	-	950 ↑ (ref 132–248)	-	Cellular destruction marker

Investigation	Day 1	Day 2–3	Day 5–6	Diagnostic Relevance
AST / ALT / ALP (U/L)	-	426 / 269 / 321	-	Transaminitis - HScore
Free T3 / T4 / TSH	-	0.46↓ / 1.65 / 0.83	-	Euthyroid sick syndrome - no primary thyroid disease
Serum Mg (mg/dL)	-	3.5 (normal)	-	Excludes electrolyte cause of bradycardia
CK-MB / Troponin I	-	-	12 U/L / Negative	Myocardial injury excluded
Echo (02/01/2026)	EF 62%; no RWMA; mild MR; trivial TR; IVC collapsing; TAPSE 23 mm; no clot/PE; TRPG 19 mmHg	-	-	Myopericarditis excluded
Widal S.Typhi O/H	1:160 / 1:160 Positive	-	-	Enteric fever - trigger confirmed
Dengue / HIV / HBsAg / Leptospira	All Negative / Non-reactive	-	-	Competing tropical triggers excluded
USG Abdomen	Splenomegaly 13.7 cm	-	-	Splenomegaly - HLH-2024 criterion
ECG Day 1 / Day 6	Sinus tachycardia 136 bpm	-	**Junctional bradycardia 55 bpm**	HLH cardiac conduction involvement
CT Brain	No abnormality	-	-	Structural neurological cause excluded

* *Post-transfusion. ↑ above / ↓ below reference range. RWMA = Regional Wall Motion Abnormality; MR = Mitral Regurgitation; TR = Tricuspid Regurgitation; TAPSE = Tricuspid Annular Plane Systolic Excursion; PE = Pericardial Effusion.*

3. Diagnostic Assessment

The combination of worsening tri-lineage cytopaenia despite antibiotics, serum ferritin exceeding 1,650 ng/mL, hypertriglyceridaemia, transaminitis, LDH 950 IU/L, splenomegaly, and unexplained cardiac and neurological involvement - with all competing tropical pathogens excluded - is not compatible with uncomplicated typhoid fever. Against the HLH-2024 criteria, [6] five of seven were fulfilled: fever, splenomegaly, tri-lineage cytopaenia, ferritin ≥500 µg/L, and hypertriglyceridaemia. NK cell activity and soluble CD25 were unavailable at our institution [11] but their absence does not negate the clinical diagnosis when five criteria are satisfied. The HScore (Table 2) calculated at approximately 187, well above the validated threshold of 169 for greater than 93% HLH probability. [7][12]

Table 2. HScore Calculation (HScore ≥169 = >93% Probability of HLH)

HScore Parameter	Value in Our Patient	Points
Underlying immunosuppression	None	0
Temperature	103°F (39.4°C) - above 38.4°C	49
Organomegaly	Splenomegaly 13.7 cm; no hepatomegaly	38
Cytopenic lineages	3 - anaemia, leucopaenia, thrombocytopaenia	24
Ferritin (ng/mL)	**>1,650 (assay limit; true value certainly higher)**	35
Triglycerides (mg/dL)	263 - above 132.7 mg/dL threshold	22
AST / SGOT (U/L)	426 - above 30 U/L threshold	19
Bone marrow haemophagocytosis	Not performed - scored 0	0
Total HScore	-	**~187 → >93% HLH probability**

Reference: Fardet L et al. Arthritis Rheumatol. 2014;66(9):2613–2620.

4. Management

From admission, empirical antimicrobial cover - Inj. Ceftriaxone 1 g IV BD and Inj. Metronidazole 500 mg IV TDS - was commenced alongside intravenous fluids and proton pump inhibitor cover. On Day 2, one unit of PRBC and two units of platelets were transfused. On Day 3, with the clinical picture evolving toward an inflammatory rather than purely infective process, Inj. Dexamethasone 8 mg IV TDS was commenced. [14] Corticosteroids are the foundation of HLH-directed therapy; in infection-associated secondary HLH, the mortality of uncontrolled haemophagocytic activation exceeds the risk of moderate-dose immunosuppression when antimicrobial cover is maintained. [14]

When ferritin returned at greater than 1,650 ng/mL on Day 5, the diagnosis was confirmed and haematology was consulted to assess eligibility for the HLH-94 protocol - etoposide and cyclosporine alongside dexamethasone. [14] Antimicrobials were continued throughout. The junctional bradycardia on Day 6 prompted emergency cardiology review and referral for temporary transvenous pacing; continuous telemetric ECG monitoring was instituted. Neurology was consulted for the limb weakness and third nerve palsy; MRI brain with gadolinium and nerve conduction studies were planned. Ophthalmology review was arranged for the ptosis.

Table 3. Day-by-Day Clinical Timeline and Management

Day / Date	Key Event	Management Actions
01 Jan Day 1	Admission; tri-lineage cytopaenia	Inj. Ceftriaxone 1g IV BD + Inj. Metronidazole 500mg IV TDS; IV fluids; CBC, Widal, dengue/HIV/HBsAg/leptospira (all negative), CRP, ABG, ECG (sinus tachy 136 bpm), CT brain, USG abdomen; Screening echo: EF 62%, no clot/PE
02 Jan Day 2	Cytopenias worsening; stool occult blood +ve	1 unit PRBC transfusion; 2 units platelet transfusion; peripheral smear: atypical lymphocytes, giant platelets
03 Jan Day 3	Biochemistry, lipids, LDH, TFT complete	Widal S.Typhi O/H 1:160 positive; AST 426, LDH 950, TG 263 mg/dL; TFT euthyroid sick pattern; Mg 3.5 mg/dL (normal); Inj. Dexamethasone 8mg IV TDS commenced
04 Jan Day 4	Neurological features: LL weakness + left ptosis	Neurology consulted; CT brain - no abnormality; continued antibiotics + dexamethasone
05 Jan Day 5	**Ferritin >1,650 ng/mL - HLH confirmed**	CK-MB 12, Troponin I negative; HScore ~187; HLH-2024 5/7 criteria met; haematology consulted for etoposide protocol
06 Jan Day 6	**Junctional bradycardia 55 bpm - urgent pacing referral**	Emergency cardiology review; temporary pacing referral; continuous telemetric ECG monitoring; echo arranged
Day 7+	Multidisciplinary escalation	Haematology: etoposide protocol evaluation; Cardiology: pacing + echo; Neurology: NCS + MRI brain with contrast; Ophthalmology: ptosis workup; continued Ceftriaxone + Metronidazole + Dexamethasone

LL = Lower Limb; NCS = Nerve Conduction Studies; PE = Pericardial Effusion.

5. Discussion

5.1 Diagnostic Challenges in the Initial Presentation.

The initial clinical picture was entirely consistent with enteric fever: positive Widal at 1:160 for both O and H antigens, markedly elevated CRP, a history of outside food consumption, rose spots, and gastrointestinal symptoms. HLH does not announce itself separately - it worsens from within while the clinician waits for antibiotics to work. Tri-lineage cytopaenia worsening over consecutive days despite appropriate antimicrobials, an AST of 426 U/L, LDH of 950 IU/L, ferritin above 1,650 ng/mL, and fasting triglycerides of 263 mg/dL are not the footprints of Salmonella Typhi alone. They are the footprints of a haemophagocytic storm triggered by it. [3][4]

5.2 The Cardiac Conduction Abnormality - Echo and Biomarkers as Discriminators

Junctional bradycardia at 55 bpm developing on Day 6 in a 24-year-old without prior cardiac disease is alarming. Cardiac complications of HLH - myocarditis, pericardial effusion, and conduction abnormalities - are documented but under-reported. [2] What makes this case distinctive is the combination of negative cardiac biomarkers (CK-MB 12, Troponin I negative) and preserved echocardiographic function (EF 62%, no effusion, TAPSE 23 mm, IVC collapsing) that together excluded myocardial injury as the mechanism. Normal serum magnesium (3.5 mg/dL) excluded electrolyte-driven conduction disease. The arrhythmia was therefore a primary conduction system event - haemophagocyte infiltration of the atrioventricular node and His-Purkinje tissue, compounded by cytokine-mediated electrophysiological disruption from circulating IFN-γ, IL-6, and TNF-α. [1] Every patient in whom HLH is

suspected must be on continuous cardiac telemetry from the moment of suspicion.

5.3 Comprehensive Infectious Exclusion in a Tropical Setting

In tropical environments where dengue co-infection and leptospirosis can independently cause thrombocytopaenia and hepatitis, excluding these conditions before attributing the syndrome to a single pathogen is not academic - it is diagnostically decisive. The complete negativity of dengue NS1/IgM/IgG, HIV serology, HBsAg, and leptospira IgG/IgM permitted confident attribution of the HLH to Salmonella Typhi as the inciting trigger.

5.4 Treatment Sequencing - Antibiotics, Steroids, and Transfusion

Antimicrobials were commenced immediately and maintained throughout. Corticosteroids were added on Day 3, not Day 1 - once the clinical picture had evolved sufficiently to make HLH the dominant diagnosis. This sequencing matters: premature immunosuppression before adequate antimicrobial control carries risk, while delayed steroid initiation once HLH is established carries greater mortality risk. [14] The Day 2 PRBC and platelet transfusions addressed immediate haematological burden. Normal coagulation parameters (INR 0.78) confirmed that hypofibrinogenaemia had not developed, so aggressive plasma replacement was not indicated.

5.5 The Neurological Manifestations

Bilateral lower limb weakness and a left third cranial nerve palsy in the context of confirmed secondary HLH are attributable to perivascular haemophagocyte infiltration of the leptomeninges and nerve roots, and to cytokine-mediated disruption of the blood-brain and blood-nerve barriers. [15] Normal CT brain was expected - gadolinium-enhanced MRI remains the investigation of choice for demonstrating meningeal or cranial nerve enhancement. Preserved deep tendon reflexes in the lower limbs argued against a demyelinating process, and concurrent cranial nerve involvement pointed toward a diffuse immune-mediated neuropathy consistent with systemic HLH.

6. Conclusion

This case of Salmonella Typhi-triggered secondary HLH - with junctional bradycardia confirmed as a conduction system event by negative biomarkers and preserved echocardiographic function, left third nerve palsy, bilateral lower limb weakness, and an HScore of approximately 187 - represents one of the most comprehensively characterised multisystem presentations of typhoid-associated HLH documented in the indexed South Asian literature. Five lessons emerge: worsening tri-lineage cytopaenia on antibiotics in a febrile patient is HLH until proven otherwise; comprehensive tropical infection exclusion is essential; the cardiac conduction system is a target and continuous telemetry is mandatory; an HScore above 169 demands immediate escalation; and corticosteroids must be added to antimicrobials once HLH is established, because delay in either cost lives.

Patient Consent and Ethics

Patient consent: Written informed consent for publication was obtained from the patient ; a signed copy is available on request.

Ethics: This report adheres to the Declaration of Helsinki (2013 revision). IEC approval: SSSMCRI, Chengalpattu (Ref: IHEC-SSSMCRI/2026/CM-04).

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No writing assistance was utilized in the production of this manuscript.

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