



Case Series

Secondary Hemophagocytic Lymphohistiocytosis Triggered by Salmonella Typhi: A Pediatric Case Series

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Abstract

Background: Secondary hemophagocytic lymphohistiocytosis (HLH) is a rare, life-threatening hyperinflammatory syndrome that may complicate severe infections. Although viral pathogens are the most common triggers, *Salmonella enterica* serovar Typhi is an uncommon cause, particularly in children. Early recognition is challenging because clinical manifestations overlap with severe typhoid fever.

Case Presentation: We describe five pediatric patients (aged 3-13 years) with culture-confirmed *Salmonella Typhi* infection who presented after recent travel to endemic regions. All had prolonged fever and gastrointestinal symptoms, with hepatosplenomegaly and hyperferritinemia as common findings. Two patients fulfilled the HLH-2004 diagnostic criteria for secondary HLH, while the remaining three demonstrated incomplete or evolving hyperinflammatory features. Three infections were caused by extended-spectrum β -lactamase (ESBL)-producing *S. Typhi*, requiring treatment with intravenous meropenem. Management consisted of targeted antimicrobial therapy, supportive care, and corticosteroids with or without intravenous immunoglobulin in selected patients with suspected HLH. All patients recovered without mortality or major complications.

Conclusion: *Salmonella Typhi* -associated HLH represents a rare but important complication of pediatric typhoid fever and may present as a spectrum ranging from evolving hyperinflammation to definite HLH. Persistent fever, hepatosplenomegaly, cytopenias, and marked hyperferritinemia should prompt consideration of HLH in children with typhoid fever. Early diagnosis, appropriate antimicrobial therapy, and timely immunomodulatory treatment in selected patients may improve clinical outcomes.

Keywords: Hemophagocytic lymphohistiocytosis; Salmonella Typhi; Typhoid fever; Pediatrics; Hyperferritinemia; ESBL; Case series

Background

Hemophagocytic lymphohistiocytosis (HLH) is a rare yet potentially fatal hyperinflammatory syndrome characterized by uncontrolled immune activation, leading to excessive cytokine release, tissue damage, and multiorgan failure if untreated [1]. It is broadly classified into primary (familial) and secondary (acquired) forms. Primary HLH typically presents in early childhood and is associated with genetic mutations, including those seen in syndromes such as Chediak–Higashi, Griscelli syndrome, X-linked lymphoproliferative disorder, and interleukin-2–inducible T-cell kinase deficiency [2-5].

Secondary HLH occurs due to dysregulated activation of cytotoxic T lymphocytes and macrophages, resulting in a hyperinflammatory state characterized by persistent fever, cytopenias, hepatosplenomegaly, coagulopathy, and hyperferritinemia [3,5]. Diagnosis is commonly based on the HLH-2004 criteria, which require at least five of the following: fever, splenomegaly, cytopenias, hypertriglyceridemia and/or hypofibrinogenemia, hemophagocytosis, reduced natural killer cell activity, elevated ferritin levels, or increased soluble CD25 [6,7]. Early recognition is critical, as delayed diagnosis is associated with high mortality [8,9].

Infections are the most frequent triggers of secondary HLH, with viral etiologies (particularly Epstein Barr virus) being the most commonly reported [10]. In contrast, bacterial associated HLH is uncommon and often underrecognized due to overlapping clinical features with severe sepsis. Among bacterial pathogens, *Salmonella Typhi*, the causative organism of typhoid fever, has rarely been described as a trigger of HLH [11,12]. Typhoid fever remains endemic in many regions and can present with prolonged fever, hepatosplenomegaly, and hematologic abnormalities, which may mimic or obscure early manifestations of HLH [12].

Given the rarity of this association and the diagnostic challenges it presents, there is limited literature describing HLH secondary to *Salmonella Typhi*, particularly in pediatric populations. This study reports a series of five pediatric patients with *Salmonella Typhi* infection complicated by secondary HLH at a tertiary care

center in Bahrain. We aim to highlight the spectrum of clinical presentation, diagnostic challenges, and treatment outcomes, and to emphasize the importance of early recognition and individualized management of this rare but serious complication.

Case Presentation

Five pediatric patients presented to a tertiary care center in the Kingdom of Bahrain with *Salmonella Typhi* infection complicated by features of secondary hemophagocytic lymphohistiocytosis (HLH). All patients had recent travel history to endemic regions, including Iraq, Pakistan, and Bangladesh. The clinical spectrum ranged from uncomplicated typhoid fever to fulminant hyperinflammatory syndromes fulfilling HLH-2004 diagnostic criteria.

Clinical presentation and investigations

Case 1

A 9-year-old boy with glucose-6-phosphate dehydrogenase deficiency presented with a 3-day history of high-grade fever, vomiting, diarrhea, abdominal pain, and poor oral intake after returning from Iraq 10 days earlier. Examination revealed hepatosplenomegaly and persistent fever (39°C). Laboratory investigations demonstrated progressive bicytopenia (hemoglobin 8.6 g/dL, platelets $45 \times 10^9/L$), markedly elevated inflammatory markers (CRP 240 mg/L, procalcitonin 45.1 ng/mL), hyperferritinemia ($>16,500$ ng/mL), hypofibrinogenemia (185 mg/dL), elevated LDH (1,479 U/L), and cholestatic liver dysfunction. Blood cultures grew extended-spectrum β -lactamase (ESBL)-producing *Salmonella Typhi*, susceptible to meropenem and azithromycin. Bone marrow aspirate demonstrated hemophagocytosis.

The patient fulfilled six HLH-2004 diagnostic criteria (fever, splenomegaly, cytopenias, hypofibrinogenemia, hyperferritinemia, and hemophagocytosis), confirming secondary HLH. He was treated with intravenous meropenem, dexamethasone according to the HLH-2004 protocol, and intravenous immunoglobulin, together with supportive care. The patient showed progressive clinical improvement with resolution of fever and hepatosplenomegaly, normalization of cytopenias and liver function tests, and was discharged in stable condition with outpatient follow-up.

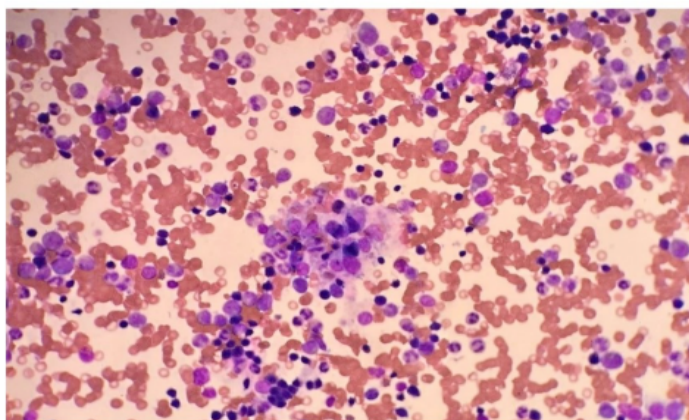


Figure 1: Bone marrow aspirate smear from Case 1 showing activated histiocytes with hemophagocytosis, characterized by engulfment of erythrocytes and other hematopoietic elements, consistent with hemophagocytic lymphohistiocytosis (HLH). (Wright–Giemsa stain).

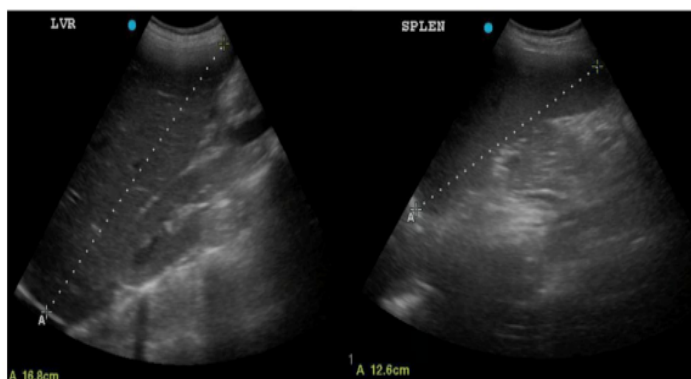


Figure 2: Abdominal ultrasound from case 1 demonstrating hepatosplenomegaly. The liver measures 16.8 cm and the spleen measures 12.8 cm, both enlarged for age. No focal lesions or ascites were identified.

Case 2

A 13-year-old girl with glucose-6-phosphate dehydrogenase deficiency presented with a 4-day history of high-grade fever, vomiting, and bloody diarrhea one week after returning from Iraq. On examination, she was febrile (39°C), mildly dehydrated, hypotensive (95/55 mmHg), and had splenomegaly on abdominal ultrasound (14.3 cm). Laboratory investigations demonstrated pancytopenia (hemoglobin 8-9 g/dL, white blood cell count $2-2.75 \times 10^9/L$, platelets $80-92 \times 10^9/L$), hyperferritinemia (6,620 ng/mL), hypertriglyceridemia (2.7 mmol/L), elevated inflammatory markers, cholestatic liver dysfunction, and hypoalbuminemia.

Blood and stool cultures grew extended-spectrum β -lactamase (ESBL)-producing *Salmonella Typhi*, susceptible to meropenem and azithromycin.

The patient fulfilled five HLH-2004 diagnostic criteria (fever, splenomegaly, cytopenias, hypertriglyceridemia, and hyperferritinemia), confirming secondary HLH; therefore, bone marrow examination was not performed. She was treated with intravenous meropenem, supportive care, and close clinical monitoring. Although dexamethasone was initiated early, full HLH-directed therapy was not required because of rapid clinical and laboratory improvement. She completed a 14-day course of antimicrobial therapy and was discharged in stable condition.



Figure 3: Abdominal ultrasound from Case 2 demonstrating hepatosplenomegaly. The liver measures 13.93 cm and the spleen measures 14.3 cm. No focal lesions were identified.

Case 3

A 13-year-old previously healthy girl presented with a 6-day history of fever, recurrent vomiting, and epigastric abdominal pain 10 days after returning from Bangladesh. Examination demonstrated mild epigastric tenderness and splenomegaly on ultrasound (13.3 cm), while the remainder of the examination was unremarkable. Blood cultures grew *Salmonella Typhi* resistant to ceftriaxone, ampicillin, and trimethoprim-sulfamethoxazole but susceptible to meropenem and azithromycin. Laboratory investigations revealed leukopenia ($3.74 \times 10^9/L$), hyperferritinemia (2,479 ng/mL), mild hypertriglyceridemia (2.2 mmol/L), elevated transaminases, and increased LDH.

The patient fulfilled three HLH-2004 criteria (fever, splenomegaly, and hyperferritinemia) but did not meet the diagnostic threshold for HLH. Given her stable clinical condition and absence of progressive hyperinflammation, HLH-directed therapy was not initiated. She was treated with a 14-day course of intravenous meropenem and supportive care, with complete clinical recovery and normalization of laboratory abnormalities prior to discharge.



Figure 4: Abdominal ultrasound from case 3 demonstrating hepatosplenomegaly. The liver measures 13.9 cm and the spleen measures 12.75 cm. No focal lesions or additional abnormalities were identified.

Case 4

A previously healthy 3-year-old boy presented with a 10-day history of high-grade fever, profuse vomiting, diarrhea, abdominal pain, and poor oral intake after returning from Pakistan. On admission, he was hypotensive (79/43 mmHg), tachycardic, tachypneic, and clinically dehydrated. Examination demonstrated hepatosplenomegaly. Blood cultures grew extended spectrum β -lactamase (ESBL)-producing *Salmonella* Typhi, susceptible to meropenem, azithromycin, ciprofloxacin, and trimethoprim-sulfamethoxazole. Laboratory investigations revealed severe anemia (hemoglobin 6.6 g/dL), hyperferritinemia (3,947 ng/mL), hypofibrinogenemia (78.3 mg/dL), elevated triglycerides, markedly elevated inflammatory markers, and deranged liver function tests. Echocardiography demonstrated mild noncardiogenic pulmonary edema with small pleural effusions.

Although the patient did not fulfill the HLH-2004 diagnostic criteria, he exhibited several features suggestive of evolving HLH, including persistent fever, hepatosplenomegaly, hyperferritinemia, and combined hypofibrinogenemia and hypertriglyceridemia. He was treated with intravenous meropenem, supportive care, dexamethasone, and intravenous immunoglobulin because of concern for progressive hyperinflammation. The patient improved clinically, with normalization of inflammatory markers and fibrinogen, resolution of hepatosplenomegaly, and was discharged in stable condition.



Figure 5: Abdominal ultrasound from case 4 demonstrating hepatosplenomegaly. The liver measures 14.36 cm and the spleen measures 10.5 cm. No focal lesions or additional abnormalities were identified.

Case 5

A 10-year-old boy presented with a 10-day history of periumbilical abdominal pain, lowgrade fever, diarrhea, and vomiting after returning from Bangladesh. Examination demonstrated hepatomegaly, while abdominal ultrasound confirmed hepatosplenomegaly. Blood and stool cultures grew *Salmonella* Typhi resistant to ampicillin and ceftriaxone but susceptible to meropenem, azithromycin, ciprofloxacin, and trimethoprim-sulfamethoxazole. Laboratory investigations revealed leukopenia ($3.7 \times 10^9/L$), thrombocytopenia ($65 \times 10^9/L$), hyperferritinemia (6,500 ng/mL), hypertriglyceridemia (4.9 mmol/L), elevated inflammatory markers, hypoalbuminemia, and cholestatic liver dysfunction.

The patient fulfilled four HLH-2004 diagnostic criteria (fever, splenomegaly, hyperferritinemia, and hypertriglyceridemia), suggesting evolving secondary HLH despite not meeting the diagnostic threshold. Given the severity of his inflammatory response, treatment was initiated with intravenous meropenem, dexamethasone, intravenous immunoglobulin, and supportive care. The patient demonstrated progressive clinical and biochemical improvement, with resolution of inflammatory markers and hepatosplenomegaly, and was discharged in stable condition on a tapering course of oral dexamethasone.

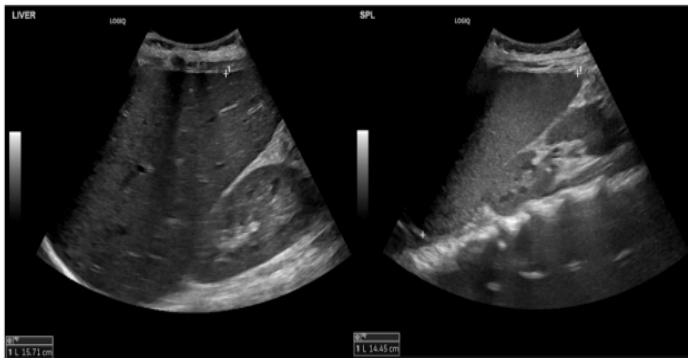


Figure 6: Abdominal ultrasound from Case 5 demonstrating hepatosplenomegaly. The liver measures 15.7 cm and the spleen measures 14.5 cm. No focal lesions or additional abnormalities were identified.

Summary

We report five pediatric cases of Salmonella Typhi infection following travel to endemic regions, spanning uncomplicated enteric fever to secondary hemophagocytic lymphohistiocytosis (HLH). Clinical severity correlated with progressive systemic inflammation characterized by persistent fever, cytopenias, hyperferritinemia, and hepatosplenomegaly. Three patients fulfilled HLH-2004 diagnostic criteria, while two exhibited incomplete or evolving hyperinflammatory features. A high proportion of isolates were multidrug-resistant ESBL-producing *S. Typhi*, necessitating escalation to carbapenem therapy. Early recognition of hyperinflammatory progression and timely initiation of antimicrobial and, when indicated, immunomodulatory therapy were associated with favorable outcomes across all cases (Table 1).

HLH-2004 criterion	Case 1	Case 2	Case 3	Case 4	Case 5
Fever	✓	✓	✓	✓	✓
Splenomegaly	✓	✓	✓	✓	✓
Cytopenia (≥2 cell lines)	✓	✓	x	x	✓
Hypertriglyceridemia and/or hypofibrinogenemia	✓	✓	x	✓	✓
Hyperferritinemia	✓	✓	✓	✓	✓
Hemophagocytosis	✓	NA	NA	NA	NA
Low NK-cell activity	NA	NA	NA	NA	NA
Elevated soluble CD25	NA	NA	NA	NA	NA
Total criteria fulfilled	06-Aug	05-Aug	03-Aug	04-Aug	04-Aug
HLH diagnosis	Definite	Definite	No	Evolving HLH	Evolving HLH
HLH, hemophagocytic lymphohistiocytosis; NK, natural killer; ND, not determined.					

Table 1: Fulfillment of the HLH-2004 diagnostic criteria among five pediatric patients with Salmonella Typhi –associated secondary hemophagocytic lymphohistiocytosis (HLH). A diagnosis of HLH was established by fulfillment of ≥5 of the 8 HLH-2004 diagnostic criteria.

Discussion

This case series describes a spectrum of *Salmonella enterica* serovar Typhi-associated hyperinflammation in five pediatric patients, ranging from uncomplicated typhoid fever with inflammatory features to definite secondary hemophagocytic lymphohistiocytosis (HLH). Two patients fulfilled the HLH-2004 diagnostic criteria, including one with bone marrow-confirmed hemophagocytosis, two demonstrated evolving HLH despite not meeting the full diagnostic threshold, and one exhibited only limited hyperinflammatory features. Three patients had extended-spectrum β -lactamase (ESBL)-producing *S. Typhi*, necessitating escalation to meropenem. Hyperferritinemia and hepatosplenomegaly were the most consistent findings, while cytopenias and hypofibrinogenemia varied in severity. All patients survived with appropriate antimicrobial therapy, and selected patients required adjunctive immunomodulatory treatment.

HLH is a rare but life-threatening hyperinflammatory syndrome characterized by impaired cytotoxic function of natural killer cells and CD8+ T lymphocytes, leading to persistent macrophage activation, excessive cytokine release, and multiorgan dysfunction [1,3,5,10]. Although viral infections, particularly Epstein-Barr virus, remain the most common infectious triggers, bacterial-associated HLH is increasingly recognized but remains underdiagnosed because its clinical manifestations frequently overlap with severe sepsis [9,10,12]. Among bacterial pathogens, *Salmonella Typhi* is a rare trigger of secondary HLH, with only isolated pediatric cases previously reported [7,13-18].

Published pediatric cases describe a similar constellation of persistent fever, hepatosplenomegaly, cytopenias, hyperferritinemia, and elevated inflammatory markers, with most patients receiving corticosteroids with or without etoposide in addition to antimicrobial therapy [14-18]. Compared with these reports, our cohort demonstrates several important differences. First, three of five isolates were ESBL-producing *S. Typhi*, reflecting the growing challenge of antimicrobial resistance and the increasing need for carbapenem therapy in severe typhoid fever. Second, our patients demonstrated a broader clinical spectrum, ranging from evolving hyperinflammation to definite HLH, rather than uniformly fulminant disease. Finally, several patients improved without etoposide, suggesting that prompt antimicrobial therapy together with careful clinical assessment and selective immunomodulation may be sufficient in appropriately selected patients.

A key finding of our series is the continuum of hyperinflammatory disease associated with *Salmonella Typhi* infection. While the HLH-2004 criteria provide a valuable diagnostic framework, they may not identify patients with early or evolving HLH who have not yet fulfilled the required diagnostic threshold [3,6,10].

Several patients in our cohort exhibited progressive biochemical abnormalities suggestive of evolving HLH despite fulfilling fewer than five diagnostic criteria. Because typhoid fever itself commonly presents with prolonged fever, hepatosplenomegaly, cytopenias, and hepatic dysfunction, distinguishing severe infection from early HLH remains a significant diagnostic challenge [12,14]. Consequently, serial clinical assessment and laboratory monitoring are essential, rather than relying solely on a single assessment of the HLH-2004 criteria. Marked hyperferritinemia was the most consistent laboratory abnormality across our cohort, supporting previous evidence that ferritin is an important marker of disease severity and should prompt consideration of HLH in children with severe typhoid fever [11].

Management should be individualized according to disease severity. All patients received targeted antimicrobial therapy, with meropenem administered for ESBL-producing isolates. Patients with confirmed or evolving HLH received corticosteroids and intravenous immunoglobulin in addition to antimicrobial therapy, whereas clinically stable patients improved with supportive care and antibiotics alone. Importantly, none of our patients required etoposide-based therapy, and all demonstrated complete clinical recovery with resolution of fever, organomegaly, inflammatory markers, and hematologic abnormalities. These findings suggest that early recognition and treatment of the underlying infection, together with selective immunomodulatory therapy, may prevent progression to fulminant HLH in selected cases [8,12].

The pathogenesis of *Salmonella Typhi*-associated HLH is thought to involve endotoxin-mediated macrophage activation, particularly through lipopolysaccharide-induced cytokine release, resulting in sustained immune activation and hemophagocytosis [3,5,13]. Persistent impairment of cytotoxic lymphocyte function further amplifies macrophage activation and contributes to the hyperinflammatory state characteristic of HLH.

This study is limited by its retrospective single-center design and small sample size. Bone marrow examination and advanced immunological investigations, including soluble CD25 and natural killer cell activity, were not available in all patients, and long-term follow-up was limited. Nevertheless, this series represents one of the larger pediatric cohorts of *Salmonella Typhi*-associated HLH and expands the limited literature by demonstrating the wide clinical spectrum of disease, the growing importance of ESBL-producing isolates, and the favorable outcomes achieved with early recognition and individualized management.

Conclusion

Secondary HLH associated with *Salmonella Typhi* infection is a rare but clinically significant hyperinflammatory complication that may present across a spectrum ranging from severe typhoid

fever with evolving hyperinflammation to definite HLH. Early recognition of persistent fever, hepatosplenomegaly, cytopenias, and marked hyperferritinemia is essential for timely diagnosis and appropriate management. Our findings highlight that strict reliance on the HLH-2004 criteria alone may delay recognition of evolving disease and that clinical judgment remains crucial when initiating antimicrobial and, where appropriate, immunomodulatory therapy. Larger multicenter studies are needed to better define predictors of disease progression, optimize the timing of immunomodulatory treatment, and improve the management of infection-associated HLH.

Abbreviations

ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; BM: Bone marrow; BMC: Blood and Medical Culture (if intended)/(journal format: not used as clinical abbreviation unless specified); CD25: Cluster of differentiation 25 (soluble IL-2 receptor alpha); CRP: C-reactive protein; EBV: Epstein-Barr virus; ESBL: Extended-spectrum beta-lactamase; G6PD: Glucose-6-phosphate dehydrogenase; GGT: Gamma-glutamyl transferase; Hb: Hemoglobin; HLH: Hemophagocytic lymphohistiocytosis; HLH-2004: Hemophagocytic Lymphohistiocytosis 2004 diagnostic criteria; HIV: Human immunodeficiency virus; ICU: Intensive care unit; INR: International normalized ratio; IVIG: Intravenous immunoglobulin; LDH: Lactate dehydrogenase; MAS: Macrophage activation syndrome; NK: Natural killer (cells); PT: Prothrombin time; RBC: Red blood cells; S. Typhi: *Salmonella enterica* serovar Typhi; TG: Triglycerides; WBC: White blood cells.

Ethics approval and consent to participate

Approved by Salmaniya Medical Complex Ethics Committee, Bahrain (Approval No. 121-31125). Conducted in accordance with the Declaration of Helsinki.

Consent for publication

Written informed consent was obtained from parents/legal guardians for publication.

Availability of data and materials

Data is included within the article. Additional data available from corresponding author on reasonable request.

Competing interests

The authors declare no competing interests.

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