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Case Report

Beyond the Glow: Life-Threatening Diarrhea and Hypovolemic Shock Following Repeated Intensive Phototherapy in a Neonate with ABO Hemolytic Jaundice

Mohamed Sadik, MD[1]; Ahmed Elmelhat, MD[1*]

[1]Department of Pediatrics/Neonatology, HMS Mirdif Hospital, Dubai, UAE.

***Corresponding Author:** Ahmed Elmelhat, Department of Pediatrics/Neonatology, HMS Mirdif Hospital, Dubai, UAE.

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Abstract

Phototherapy is the mainstay of treatment for neonatal hyperbilirubinemia and is generally considered safe, with loose stools regarded as a trivial side effect. We describe a late-preterm female infant with ABO hemolytic jaundice who required three separate courses of intensive phototherapy and subsequently developed severe watery diarrhea with hypovolemic shock (pH 7.0, bicarbonate 8 mmol/L, potassium 6.1 mmol/L, sodium 150 mmol/L) at 18 days of life. An exhaustive workup including a comprehensive stool viral PCR panel, plasma amino acids, urine organic acids and amino acids, repeat acylcarnitine profile, abdominal and transcranial ultrasound, echocardiography, and chest radiograph excluded infection, inborn errors of metabolism, and structural disease. She recovered fully after introduction of an amino-acid–based (elemental, lactose-free) elimination formula, which was begun to exclude any possibility of milk protein intolerance or allergy. We propose phototherapy-associated enteropathy, manifesting as secretory diarrhea with transient carbohydrate malabsorption, as the most plausible explanation, supported by a focused literature review. Clinicians should recognize that phototherapy-associated diarrhea, though usually benign, can rarely progress to life-threatening dehydration and shock particularly after repeated or intensive courses.

Keywords: *Phototherapy, Neonatal Diarrhea, Dehydration, Hypovolemic Shock, Abo Incompatibility, Hyperbilirubinemia, Secretory Diarrhea, Transient Lactose Intolerance*

Introduction

Few things in newborn medicine feel as reassuring as the soft blue glow of a phototherapy unit—a therapy that has prevented countless cases of kernicterus and remains the mainstay of treatment for neonatal jaundice.[1] It is, by and large, safe and wonderfully effective. Yet, like any powerful tool, it is not without a shadow side. Clinicians know that phototherapy often produces loose, frequent, sometimes green stools [2,4] this is usually shrugged off as a harmless byproduct of moving bilirubin through the gut. But what happens when that "harmless" side effect tips into something dangerous? We encountered an infant in whom repeated, intensive phototherapy for ABO hemolytic jaundice was followed by catastrophic watery diarrhea and hypovolemic shock—and in whom nothing else could explain it. Here we tell her story and revisit the under-appreciated literature linking phototherapy to a genuine, secretory enteropathy.

Case Presentation

Birth and initial course. A female infant was born at 36+6 weeks of gestation to a 21-year-old mother (gravida 3, para 2) by normal vaginal delivery. Birth weight was 3.02 kg (appropriate for gestational age), length 49 cm, and head circumference 34.5 cm. Apgar scores were 8, 9, and 10 at 1, 5, and 10 minutes. The mother was blood group O positive; the infant was blood group A positive, with a positive direct Coombs test—classic ABO isoimmunization. Her first few days were spent under phototherapy for newborn jaundice, and she went home on vitamin D drops with plans for outpatient bilirubin checks.

First and second NICU admissions (jaundice). At 5 days of life she returned with a total serum bilirubin of 29.6 mg/dL—in the exchange-transfusion range—again attributable to ABO incompatibility. She received 48 hours of intensive phototherapy and went home once bilirubin fell to 12.4 mg/dL. She was back at 9 days, deeply jaundiced once more (bilirubin 18.2 mg/dL), with persistent ABO incompatibility; expanded newborn screening had flagged an abnormal acylcarnitine profile, prompting a broader metabolic workup. Another course of intensive phototherapy and intravenous fluids followed, and she was discharged at 13 days (bilirubin 9.5 mg/dL).

Third admission: severe diarrhea and shock. Eighteen days into life, she came in with a stark chief complaint—profuse watery diarrhea (more than eight episodes in the prior day at home, five more since arrival) and inconsolable crying. She looked cachectic and pale, with the unmistakable facies of severe dehydration: dry lips and tongue, sunken eyes, a depressed fontanelle, and skin that stayed tented after a pinch. Capillary refill was delayed at 3 seconds. Oxygen saturation sat at 90% on room air, climbing above 96% with a little nasal cannula support. Her blood pressure was unrecordable in the emergency room and only recovered after fluids. Remarkably, she remained neurologically intact, crying strongly throughout. The full clinical course is illustrated in Figure 1.

Her admission labs were alarming (Table 1). Hematology showed hemoglobin 10.1 g/dL (mild anemia), platelets 728 × 10^3/μL (reactive thrombocytosis), and C-reactive protein 29 mg/L. Arterial blood gas and electrolytes revealed pH 7.0, bicarbonate 8 mmol/L (profound metabolic acidosis), potassium 6.1 mmol/L (critical hyperkalemia), sodium 150 mmol/L (hypernatremia signaling hemoconcentration), and chloride 128 mmol/L. Liver and kidney studies showed gamma-glutamyl transferase strikingly elevated at 240 U/L and persistently so since birth, total bilirubin 7.5 mg/dL with direct 0.9 mg/dL, but normal transaminases, total protein, and albumin—effectively ruling out fulminant liver failure. Urea 20 mg/dL and creatinine 0.54 mg/dL were stable. Stool was soft, watery, yellow, pH 6.0, with only trace reducing substances and no blood, mucus, leukocytes, or parasites.

Imaging and microbiology were unrevealing. Echocardiography showed a small secundum atrial septal defect with normal ventricular function. Abdominal ultrasound revealed gallbladder sludge and mild gaseous distension with otherwise normal liver, spleen, kidneys, and pancreas; transcranial ultrasound and chest radiograph were normal. A multiplex stool viral PCR panel was negative for rotavirus, norovirus, sapovirus, adenovirus, astrovirus, *Salmonella*, *Campylobacter*, *Shigella*/EIEC, EPEC, ETEC, STEC, EAEC, *Yersinia*, *Vibrio* spp., *C. difficile* toxins, *Giardia*, *Cryptosporidium*, *Entamoeba*, *Cyclospora*, and *Plesiomonas*; blood CMV PCR was not detected.

Management and course. In the emergency room she received three fluid boluses (two 10 mL/kg normal saline, one 10 mL/kg Ringer's lactate) and perfusion improved. She was then placed on continuous IV fluids engineered to correct her hypernatremia gradually over 48 hours—important to avoid cerebral edema—with diarrheal losses replaced 1:1 with Ringer's lactate. For hyperkalemia we nebulized albuterol to push potassium intracellularly, kept IV calcium gluconate at the bedside as insurance against arrhythmias, and rechecked electrolytes and gases every few hours until potassium settled and bicarbonate began to climb. Empirical meropenem and vancomycin covered for sepsis while cultures were pending, and all remained sterile. The gut was rested (nothing by mouth) so we could confirm the diarrhea was secretory—it persisted despite fasting.

By day 22, sodium (137 mmol/L) and potassium (4.4 mmol/L) had normalized, though bicarbonate was still recovering at 15 mmol/L, albumin was a little low at 3.0 g/dL, and phosphate was 4.8 mg/dL; C-reactive protein had dropped below 1 mg/L. Repeat plasma and urine amino-acid profiles came back normal, and the repeat acylcarnitine study was not diagnostic of any fatty-acid oxidation or organic acid disorder. We then introduced an amino-acid–based (elemental, lactose-free) elimination formula—chosen empirically to exclude any possibility of milk protein intolerance or allergy while providing complete nutrition. She did beautifully—and went home in stable condition, remaining well on follow-up with the diarrhea gone.

Table 1. Key investigations during the third admission (severe diarrhea and hypovolemic shock).

Parameter	Admission	Follow-up	Reference range
Hemoglobin (g/dL)	10.1	—	12.5–20.5
Platelets (×10³/μL)	728	477	150–450
C-reactive protein (mg/L)	29	<1	<5
pH	7.0	—	7.35–7.45
Bicarbonate (mmol/L)	8	15	22–30
Potassium (mmol/L)	6.1	4.4	3.5–5.1
Sodium (mmol/L)	150	137	134–146
Chloride (mmol/L)	128	113	98–113
Gamma-glutamyl transferase (U/L)	240	—	—
Total bilirubin (mg/dL)	7.5	—	0–10
Direct bilirubin (mg/dL)	0.9	—	0.1–0.5
AST/ALT, albumin	normal	—	—
Urea (mg/dL)	20	26	18–45
Creatinine (mg/dL)	0.54	0.31	0.40–0.90
Stool pH / reducing substances	6.0 / trace	—	—

All microbiological, metabolic, and imaging studies were negative or normal except where noted above.

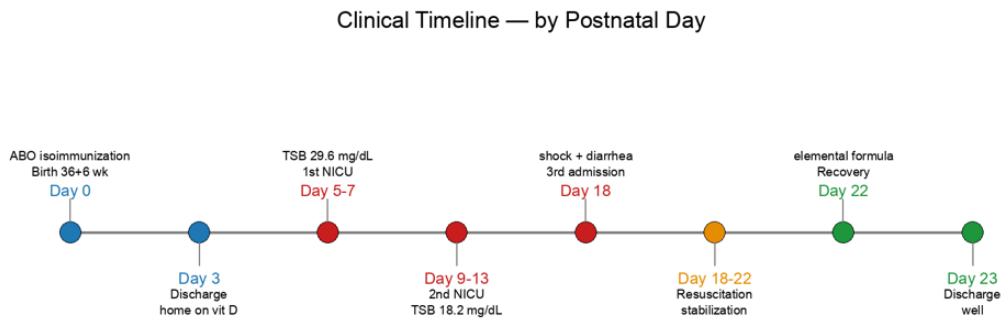

Figure 1. Clinical timeline by postnatal day.

Discussion

Diarrhea as a complication of phototherapy—more than we thought. Loose, frequent, often green stools accompany phototherapy so commonly that we barely notice them, but the physiology underneath is more interesting—and more worrisome—than "the gut is moving faster." De Curtis and colleagues studied 30 jaundiced newborns with diarrhea during phototherapy and showed, using rectal dialysis, that the diarrhea was truly secretory: colonic absorption of water, sodium, and chloride was markedly impaired, and—crucially—this impairment vanished once phototherapy stopped.[3] The same group had earlier flagged this secretory pattern in phototherapy-treated infants.5 Both hyperbilirubinemia *and* light exposure were needed for the effect. Berant and co-workers measured fecal bile salts in 14 jaundiced infants on phototherapy and found them rise significantly within 12 hours of "lights on," with six infants developing watery, high-sodium stools; levels returned to baseline a day after the lights went off.6 High colonic bile-salt concentration is a textbook stimulus for secretory diarrhea.

Bakken first described temporary intestinal lactase deficiency in light-treated jaundiced infants,[7] and several guidelines list "diarrhea from intestinal hypermotility" and, tellingly, "temporary lactose intolerance" among phototherapy's adverse effects.[4] The prevailing view is a functional, reversible brush-border disturbance rather than a true enzymatic absence. Contemporary reviews note that phototherapy drives bilirubin-breakdown products into the intestine, irritating the wall and altering the epithelial transmembrane potential, thereby causing diarrhea with real losses of water, sodium, and potassium,[2] with dehydration expressly called out as a recognized acute adverse reaction—especially in preterm and intensively treated babies.[2] Phototherapy also accelerates gut transit8 and unconjugated bilirubin itself can inhibit intestinal water and electrolyte absorption,[9] while its cumulative duration correlates with declining serum sodium and potassium.[10]

Why this infant fits the picture. Our patient did not have one standard phototherapy course she had three separate, intensive courses in her first two weeks. Each is independently tied to loose stools and fluid loss; stacked together, the enteric insult is plausibly cumulative and pronounced. The clinical signature torrential watery diarrhea, hypovolemic shock, hypernatremia with hemoconcentration, metabolic acidosis, and hyperkalemia is exactly what you would expect from massive gastrointestinal water and electrolyte loss. The secretory nature was suggested by persistence during fasting (despite gut rest) and by a stool pH of 6.0 with only trace reducing substances, which argues against a purely osmotic, fermentable-carbohydrate process at the time of testing (early-phase samples were not captured).

The formula response: exclusion, not confirmation, of milk allergy. The introduction of an amino-acid–based, lactose-free elemental formula led to prompt and complete resolution. Importantly, this therapeutic response helps *exclude* rather than confirm an underlying cow's milk protein allergy (CMPA) or primary lactose intolerance. A favorable response to an elimination formula is supportive but not diagnostic of CMPA; here, no corroborating criteria were met there was no blood or mucus in the stools, no eczema, no IgE sensitization, and no confirmed relapse on milk reintroduction. CMPA therefore remains only one item in the differential, not an established diagnosis. The formula was started principally to eliminate milk protein intolerance or allergy from consideration while the infant recovered, not as proof of either entity.

What we ruled out. This is, importantly, a diagnosis of exclusion built carefully. Infection was excluded by the comprehensive stool viral PCR panel, negative blood CMV, and empirical antibiotics with uniformly sterile cultures. Inborn errors of metabolism were effectively excluded: although screening flagged an abnormal acylcarnitine profile, repeat acylcarnitine, plasma amino acids, urine amino acids, and urine organic acids were all normal. Structural or anatomic disease was excluded by normal abdominal and transcranial ultrasound, echocardiography, and chest radiograph apart from gallbladder sludge and mild gaseous distension; no obstruction or congenital diarrheal disorder was demonstrated, and the brisk, sustained response to a formula change argues against an intractable congenital enteropathy such as microvillus inclusion disease or tufting enteropathy. Hepatobiliary disease was considered GGT was persistently elevated since birth with mildly raised direct bilirubin but transaminases, albumin, and hepatic architecture were normal, and the shock-level diarrheal illness was out of proportion to, and temporally separate from, any liver dysfunction.

Honest limitations. Two caveats deserve a place at the table. First, the diarrhea began roughly five days *after* the last phototherapy course ended, whereas phototherapy-associated diarrhea is classically described *during* treatment. We suggest that in a baby subjected to repeated, intensive courses, the enteric disturbance may have been cumulative and only became clinically—and catastrophically—apparent once losses at home outstripped intake.

Second, an unrelated, transient feed-protein or lactose intolerance (for example, food-protein–induced enterocolitis or allergic colitis) cannot be fully excluded; however, the clustering after multiple phototherapy exposures, together with the literature linking phototherapy to temporary lactose intolerance,[4,7] makes a shared, phototherapy-related mechanism the more parsimonious explanation. The persistently elevated GGT warrants its own hepatology and metabolic follow-up, which was already arranged.

What this means at the bedside. This case is a reminder that phototherapy-associated diarrhea, though usually trivial, can on rare occasions spiral into severe dehydration and shock—particularly after repeated or intensive courses. Stool output, weight, hydration, and electrolytes deserve attention during *and* after phototherapy, and clinicians should keep a phototherapy-related enteropathy in mind when a recently jaundiced neonate presents with unexplained severe diarrhea. Early, aggressive fluid and electrolyte resuscitation is lifesaving; an empirical trial of a lactose-free or elemental formula may both treat and help exclude milk protein intolerance or allergy.

Differential Diagnosis

Severe, watery, dehydrating diarrhea in a neonate carries a broad differential. Each entity was systematically considered; with the exception of phototherapy-associated enteropathy, all were effectively excluded:

- **Infectious gastroenteritis (viral, bacterial, parasitic).** A comprehensive multiplex stool PCR was negative for rotavirus, norovirus (GI/GII), sapovirus, adenovirus F, astrovirus, *Salmonella*, *Campylobacter*, *Shigella*/EIEC, EPEC, ETEC, STEC, EAEC, *Yersinia*, *Vibrio* spp., *C. difficile* toxins A/B, *Giardia*, *Cryptosporidium*, *Entamoeba*, *Cyclospora*, and *Plesiomonas*; blood CMV PCR was negative; and all bacterial cultures remained sterile despite empirical meropenem and vancomycin. An infectious etiology is therefore excluded.

- **Necrotizing enterocolitis (NEC).** Excluded by the absence of bilious emesis, abdominal distension, blood in stools, or pneumatosis/portal venous gas; abdominal ultrasound and radiograph were unremarkable apart from mild gaseous distension and gallbladder sludge, and the course was rapidly reversible.

- **Hirschsprung-associated enterocolitis.** Excluded by the absence of constipation, failure to pass meconium, or intestinal obstruction; no radiographic or ultrasonographic signs of aganglionosis, and the diarrhea resolved completely on a formula change rather than requiring decompression.

- **Congenital diarrheal disorders** (microvillus inclusion disease, tufting enteropathy, congenital chloride/sodium diarrhea, glucose–galactose malabsorption). These typically present in the first days of life with intractable diarrhea unresponsive to dietary modification. Our patient's diarrhea was monophasic, appeared after phototherapy courses, and resolved promptly on an elemental formula—discordant with a primary congenital enteropathy (no mucosal biopsy was performed).

- **Cow's milk protein allergy / food-protein–induced enterocolitis (CMPA/FPIES).** Considered but unsupported: stools had no blood or mucus, there was no eczema, no IgE sensitization, and no confirmed relapse on milk reintroduction. The amino-acid formula was introduced to *exclude* milk protein intolerance or allergy, not to confirm it; CMPA therefore remains only a differential.

- **Lactose intolerance (primary or secondary).** Primary congenital lactase deficiency is exceedingly rare and would not follow the phototherapy timeline; secondary malabsorption after enteric infection is unlikely given the negative stool PCR. A phototherapy-associated *transient* lactose intolerance is itself described in the literature and may overlap with our hypothesis.

- **Inborn errors of metabolism** (aminoacidopathies, organic acidemias, fatty-acid oxidation defects, carbohydrate disorders). Excluded by normal plasma amino acids, urine organic acids, urine amino acids, and a repeat acylcarnitine profile; the initial abnormal newborn-screen acylcarnitine was a false positive.

- **Adrenal insufficiency / salt-wasting** (congenital adrenal hyperplasia, pseudohypoaldosteronism). These present with *hyponatremia* and hyperkalemia, whereas our infant was hypernatremic with hemoconcentration and stable renal function; electrolytes normalized with hydration alone, excluding a mineralocorticoid disorder.

- **Intestinal obstruction / malrotation–volvulus.** Excluded by the absence of bilious vomiting, normal abdominal ultrasound (no malrotation or midgut volvulus), and a benign abdominal examination.

- **Cholestatic / hepatobiliary disease.** Although GGT was persistently elevated with mildly raised direct bilirubin, transaminases, albumin, and hepatic architecture were normal and imaging showed no biliary obstruction; liver disease alone cannot explain the acute secretory diarrhea and shock.

- **Osmotic / medication-induced diarrhea.** No enteral osmotically active agents or laxatives were administered; intravenous antibiotics are an unlikely sole cause of this profile, and diarrhea persisted despite gut rest.

Having excluded these alternatives, phototherapy-associated enteropathy emerges as the most probable diagnosis: repeated, intensive phototherapy for ABO hemolytic jaundice precipitated a secretory, bile-salt– and bilirubin-driven intestinal injury with transient carbohydrate malabsorption, culminating in severe dehydration and hypovolemic shock.

Conclusion

We describe a neonate who developed severe dehydrating, secretory diarrhea and hypovolemic shock after repeated intensive phototherapy for ABO hemolytic jaundice. Infection, inborn errors of metabolism, structural disease, and other enteric disorders were comprehensively excluded by an exhaustive workup, and the differential diagnosis is summarized above. The clinical course and the dramatic response to an amino-acid–based (lactose-free) elimination formula introduced to exclude milk protein intolerance or allergy together with the temporal relation to three courses of intensive phototherapy, lead us to conclude that the diarrhea and shock were most probably related to phototherapy, representing a rare, severe form of phototherapy-associated enteropathy encompassing secretory diarrhea and transient carbohydrate malabsorption. Greater awareness of this potentially fatal but preventable complication is warranted.

Patient Consent

Written informed consent was obtained from the infant's legal guardian (mother) for publication of this case report and any accompanying data.

Author Contributions

A.E. and M.S. conceived the report, provided clinical care, and drafted the manuscript. [Co-author] contributed to the literature review and critical revision. All authors approved the final version.

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Conflict of Interest

The authors declare no conflicts of interest.

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