

Case Report

Fatal *Pneumocystis jirovecii* Pneumonia in a Severely Malnourished Infant with Undiagnosed HIV: A Case Report

Tanı Almamış HIV Enfeksiyonlu, Ağır Malnütrisyonlu bir Bebekte Ölümcül Pneumocystis jirovecii Pnömonisi: Bir Olgu Sunumu

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ABSTRACT

This report describes a 5-month-old infant who presented with failure to thrive, severe respiratory distress and oral candidiasis. The clinical course was complicated by severe acute malnutrition, electrolyte imbalances characteristic of refeeding syndrome and progressive respiratory failure. A reactive serologic test for Human Immunodeficiency Virus (HIV) obtained during blood transfusion cross-matching prompted further investigation, with posthumous confirmatory results establishing HIV-1 infection, underscoring a delay in diagnosis. The pneumonia was attributed to *Pneumocystis jirovecii* (PCP). Although the infant met clinical criteria for severe PCP, adjunctive corticosteroids, which are recommended in such cases, were not initiated, representing a missed therapeutic opportunity. The illness evolved into a hyperinflammatory state resembling Hemophagocytic Lymphohistiocytosis (HLH), with multiorgan dysfunction, Acute Respiratory Distress Syndrome (ARDS) and pulmonary hemorrhage. Despite broad-spectrum antimicrobials and intensive supportive care, the outcome was fatal. This case highlights the importance of early HIV screening, timely initiation of PCP prophylaxis with adjunctive corticosteroids, and careful management of refeeding syndrome in severely malnourished infants.

Keywords: *hiv infection, pneumocystis jirovecii pneumonia, infant, malnutrition, refeeding syndrome*

ÖZET

Bu raporda, gelişme geriliği, ağır solunum sıkıntısı ve oral kandidiyazis ile başvuran 5 aylık bir bebek olgusu sunulmaktadır. Klinik seyir, ağır akut malnütrisyon, yeniden beslenme sendromuna özgü elektrolit dengesizlikleri ve ilerleyici solunum yetmezliği ile komplike olmuştur. Kan transfüzyonu çapraz karşılaştırması sırasında elde edilen reaktif İnsan İmmün Yetmezlik Virüsü (HIV) serolojik testi ileri araştırmayı tetiklemiş, ölüm sonrası doğrulayıcı sonuçlar HIV-1 enfeksiyonunu kanıtlayarak tanıda gecikmeye dikkat çekmiştir. Pnömoni, *Pneumocystis jirovecii*'ye (PCP) bağlanmıştır. Bebek ağır PCP için klinik kriterleri karşılamasına rağmen, bu tür olgularda önerilen adjuvan kortikosteroidler başlanmamış olup bu durum kaçırılmış bir tedavi fırsatını temsil etmektedir. Hastalık, çoklu organ disfonksiyonu, Akut Solunum Sıkıntısı Sendromu (ARDS) ve pulmoner hemoraji ile seyreden, Hemofagositik Lenfohistiyoitoz (HLH) benzeri hiperinflamatuvar bir duruma evrilmiştir. Geniş spektrumlu antimikrobiyaller ve yoğun destekleyici bakıma rağmen sonuç ölümcül olmuştur. Bu olgu, erken HIV taramasının, adjuvan kortikosteroidlerle PCP profilaksisinin zamanında başlatılmasının ve ağır malnütrisyonlu bebeklerde yeniden beslenme sendromunun dikkatli yönetiminin önemini vurgulamaktadır.

Keywords: *hiv enfeksiyonu, pneumocystis jirovecii pnömonisi, bebek, malnütrisyon, yeniden beslenme sendromu*

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INTRODUCTION

Perinatal transmission of Human Immunodeficiency Virus (HIV) remains a significant global health issue (1). Infants with undiagnosed HIV are at high risk of severe opportunistic infections, with *Pneumocystis jirovecii* pneumonia (PCP) being a leading cause of mortality (2). The diagnosis of PCP in children is often delayed due to nonspecific symptoms, such as cough, tachypnea and hypoxia, which overlap with those of other common childhood pneumonias (2). Management is further complicated when HIV coexists with severe acute malnutrition, which impairs both immune and metabolic function, worsening infection outcomes (3). Malnourished infants are also at risk of **refeeding syndrome**, a potentially fatal metabolic derangement that occurs when nutritional support is reintroduced after prolonged undernutrition (4).

In Malaysia, despite established prevention of mother-to-child transmission programs, missed antenatal HIV screening remains a concern, particularly among non-citizen or unscreened mothers (5). This case illustrates the tragic convergence of delayed HIV diagnosis, untreated PCP, and refeeding-related metabolic collapse in an infant, highlighting several missed opportunities for prevention and intervention.

CASE REPORT

A 5-month, 25-day-old term male infant was admitted with a one-week history of dry cough and progressive respiratory distress. He had a two-month history of failure to thrive, with weight declining from 3.6 kg at birth to 2.9 kg (weight-for-age < -3 SD, consistent with severe acute malnutrition). The mother reported poor feeding, lethargy, and oral thrush for one month. There was no known perinatal illness, and the mother had not undergone antenatal HIV screening. Both parents were foreign nationals.

On admission, the infant was cachectic and tachypneic, with subcostal and intercostal recessions. Oxygen saturation was maintained at 96% on nasal prong oxygen at 2 L/min. Chest auscultation revealed diffuse crackles, and a chest radiograph showed bilateral pneumonic infiltrates. Initial investigations revealed severe anemia (hemoglobin 7.5 g/dL) and hypoalbuminemia (25 g/L), while serum electrolytes were within normal limits (Table 1). He was started on

intravenous Benzylpenicillin (50,000 units/kg four times daily) for empiric treatment of community-acquired pneumonia and oral Nystatin (100,000 units four times daily) for oral candidiasis. Nutritional supplementation with multivitamins (1ml once daily) and folate (0.1mg once daily) was also initiated to address possible nutritional deficiencies contributing to anemia and poor growth.

Within 24 hours, the infant's respiratory distress worsened, requiring escalation to high-flow nasal cannula (HFNC). Oral Azithromycin (15 mg/kg on Day 1, followed by 7.5 mg/kg once daily from Day 2 to Day 5) was added to broaden coverage for atypical pathogens. By Day 4, oxygen saturation had fallen to 88% despite HFNC (40 L/min, FiO₂ 0.6), and the platelet count declined from $229 \times 10^9/L$ to $70 \times 10^9/L$. The infant was intubated for severe respiratory failure and transferred to the Neonatal Intensive Care Unit (NICU). He developed severe and refractory electrolyte derangements, including hypocalcemia, hypomagnesemia, and hypophosphatemia, which persisted despite repeated intravenous corrections, consistent with refeeding syndrome. During cross-matching for a packed cell transfusion, a routine HIV serology screen was reactive, prompting a confirmatory HIV-1 RNA PCR test. Intravenous Trimethoprim-Sulfamethoxazole (15mg/kg/day, based on the trimethoprim component) was initiated for suspected PCP.

In the NICU, the patient required high-frequency oscillatory ventilation (HFOV) at FiO₂ up to 100%, indicating severe acute respiratory distress syndrome (ARDS). Antibiotic coverage was broadened to intravenous Ceftriaxone 50 mg/kg/day, then escalated to Meropenem 40 mg/kg every 8 hours when abdominal distension and an abdominal X-ray revealing thickened bowel loops suggested septic ileus. He received multiple transfusions of packed red cells, platelets, fresh frozen plasma (FFP) and cryoprecipitate to correct anemia, thrombocytopenia, and coagulopathy. Laboratory findings showed markedly elevated ferritin (6010 µg/L) and prolonged PT/APTT with low fibrinogen (1.026 g/L), suggesting a hyperinflammatory state consistent with secondary hemophagocytic lymphohistiocytosis (HLH). On Day 10, the infant suffered from a massive pulmonary hemorrhage, for which intravenous vitamin K 0.3 mg/kg once daily was administered for 3 days.

Total parenteral nutrition (TPN) was initiated on Day 13 with cautious caloric advancement, achieving approximately 55% of estimated energy requirements. Despite this, metabolic instability persisted, and phosphate and magnesium levels remained critically

low. Thiamine was not given prior to TPN initiation. An extensive infectious workup, including bacterial, viral, and fungal cultures, as well as serologies for CMV, EBV, hepatitis B and C, and parvovirus B19, was negative. The Inborn Error of Metabolism (IEM) screen was non-diagnostic (Table 2).

Table 1. Summary of Key Laboratory Parameters During Hospitalization

Parameter	Pediatric Reference Range	Admission Value	Nadir / Peak	Trend / Comment
WBC (x 10 ⁹ /L)	6 - 17.5	17.95	25.17	Initial leukocytosis, with a later downtrend suggestive of bone marrow exhaustion
Hemoglobin (g/dL)	10.5 - 13.5	7.5	6.6	Improved transiently post-transfusion
Platelet (x 10 ⁹ /L)	150 - 400	229	8	Progressive decline, thrombocytopenia
CRP (mg/L)	< 5	< 1	24.3	Initially low, rising with worsening sepsis
ALT (U/L)	< 45	64	619	Consistent with multiorgan failure / HLH
AST (U/L)	< 80	173	2657	Marked transaminitis indicating severe liver injury
Serum albumin (g/L)	35 - 50	25	18	Persistent hypoalbuminemia
Calcium (mmol/L)	2.1 - 2.6	1.95	1.51	Persistent hypocalcemia
Magnesium (mmol/L)	0.65 - 1.05	1.18	0.64	Refractory hypomagnesemia
Phosphate (mmol/L)	1.3 - 2.3	1.61	0.57	Refractory hypophosphatemia
Ferritin (µg/L)	50 - 200	-	6010	Markedly elevated, HLH-like inflammation
PT (seconds)	11 - 14	-	16.5	Prolonged
APTT (seconds)	25 - 40	-	51.7	Prolonged
Fibrinogen (g/L)	2.19 - 3.94	-	1.026	Low, indicating consumptive coagulopathy / DIC

Note: Reference ranges adapted from Zajac J, Lee G, eds. *The Harriet Lane Handbook*. 23rd ed. Philadelphia, PA: Elsevier; 2024 (6). Abbreviations: WBC - white blood cell; CRP - C-reactive protein; ALT - alanine aminotransferase; AST - aspartate aminotransferase; HLH - Hemophagocytic Lymphohistiocytosis; DIC - disseminated intravascular coagulation.

Despite maximal ventilatory and supportive management, including broad-spectrum antimicrobials, electrolyte correction, transfusions, and nutritional therapy, the patient deteriorated and died on Day 16. The confirmatory HIV-1 RNA PCR, sent on Day 4, was received posthumously and returned positive, confirming vertically transmitted HIV infection.

DISCUSSION

This case highlights the complex interplay of delayed HIV diagnosis, severe malnutrition and opportunistic infection in an infant, culminating in a fatal outcome. It underscores critical lessons in early HIV detection, evidence-based management of PCP, prevention of refeeding syndrome and rational use of supportive care.

The infant's presentation with severe hypoxemic respiratory failure was typical of moderate-to-severe PCP in the context of an underlying immunodeficiency.

The posthumous confirmation of HIV infection emphasized a missed opportunity for early maternal screening, possibly related to the parents' foreign status.

According to the Centers for Disease Control and Prevention (CDC), adjunctive corticosteroids should be initiated within 72 hours of PCP diagnosis in cases of significant hypoxemia (PaO_2 below 70 mm Hg or alveolar-arterial gradient above 35 mm Hg) (7). Although this recommendation originates from adult

studies, pediatric data have demonstrated similar benefits. Newberry et al. (8) reported that adjunctive prednisone reduced in-hospital and six-month mortality among HIV-exposed infants with PCP, while Terblanche et al. (9) found improved survival when corticosteroids were introduced during clinical deterioration. In this case, the infant met criteria for severe PCP but did not receive adjunctive corticosteroid. This was likely a missed therapeutic opportunity that might have altered the outcome.

Table 2. Summary of Diagnostic Investigations Performed to Exclude Other Pathologies

Investigation Category	Specific Tests Performed	Result	Clinical Significance
Microbiology			
Bacterial Culture	Blood (multiple), Tracheal Aspirate, CSF	No growth	Rules out common bacterial sepsis or pneumonia
Mycobacterial Culture	Tracheal Aspirate for Acid-Fast Bacilli (smear and culture)	Negative	No evidence of pulmonary tuberculosis
Fungal Culture	Blood , CSF	No growth	Rules out invasive fungal infection
Virology			
Respiratory Pathogens	Multiplex PCR for common respiratory viruses	All negative	No evidence of typical viral pneumonia
Congenital Infections	Serology for CMV (IgM) and EBV (VCA IgM)	Non-reactive	No evidence of acute CMV or EBV infection
Other Viral Pathogens	Serology for Parvovirus B19 (IgM), and Hepatitis B and C	Non-reactive	Rules out viral causes of anemia or hepatitis
HIV Confirmation			
Initial Screen	HIV Antigen / Antibody Combo Assay	Reactive	First indication of potential HIV exposure
Confirmatory Test	HIV-1 RNA PCR	Positive (posthumous)	Confirms active HIV-1 infection
Metabolic Screening			
Inborn Error of Metabolism	Acylcarnitine and Amino Acid Panel (dried blood spot)	Non-diagnostic	Abnormalities attributed to severe malnutrition
	Urine Organic and Orotic Acids	Non-specific changes	Findings secondary to liver failure and ketosis
Plasma Ammonia	Ammonia level	46.5 $\mu\text{mol/L}$ (Normal)	Rules out hyperammonemia

Abbreviations: CSF - cerebrospinal fluid; PCR - polymerase chain reaction; CMV - cytomegalovirus; EBV - Epstein-Barr virus; HIV - Human Immunodeficiency Virus

The concurrent findings of cytopenias, markedly elevated ferritin, and coagulopathy suggested a hyperinflammatory response resembling secondary HLH, a recognized complication of advanced pediatric HIV and severe infection. Such immune dysregulation likely contributed to multiorgan failure and rapid clinical deterioration.

Severe acute malnutrition (weight-for-age < -3 SD) placed the infant at high risk for refeeding syndrome, a potentially fatal metabolic disturbance that can develop when nutrition is restarted after prolonged undernutrition (10). When feeding is reintroduced, the body shifts from using fat to carbohydrates as its primary energy source. This triggers insulin release that drives phosphate, potassium, and magnesium into cells and causes a rapid fall in the plasma levels (11).

In this case, the infant developed severe and persistent hypophosphatemia and hypomagnesemia despite repeated intravenous replacement, consistent with refeeding syndrome. Thiamine, an essential cofactor for carbohydrate metabolism, was not given before TPN initiation, likely worsening the metabolic imbalance. This case highlights that even cautious feeding can precipitate refeeding syndrome in severely malnourished infants. It underscores the need for thiamine supplementation and correction of electrolytes before initiating nutrition support in all high-risk children (10,11).

During admission, the infant received several infusions of human albumin to correct hypoalbuminemia. However, recent international guidelines do not recommend the routine use of intravenous albumin in pediatric patients with infection or hypoperfusion, as it has not been shown to reduce mortality (12). Hypoalbuminemia should instead be interpreted as a marker of disease severity rather than a therapeutic target. Following evidence-based fluid management principles and using crystalloids as first-line therapy help avoid unnecessary interventions and minimize the risk of fluid overload.

Preventative strategies are crucial. Routine maternal HIV screening during antenatal visits and early infant prophylaxis with oral cotrimoxazole at six weeks of age, as outlined in the Paediatric Protocols for Malaysian Hospitals, 5th Edition (13), could have facilitated early identification of HIV exposure and prevented the onset of severe PCP. Strengthening prevention-of-mother-to-child transmission programs,

particularly for non-citizen or unscreened mothers, remains vital to reducing pediatric HIV-related mortality.

CONCLUSION

The fatal outcome in this infant was the result of a delayed HIV diagnosis, severe malnutrition and refeeding-related metabolic complications, compounded by missed therapeutic opportunities in managing PCP. Early maternal HIV screening, timely infant prophylaxis, and adherence to pediatric treatment guidelines for PCP management could have altered the outcome. Clinicians should remain alert to the risks of refeeding syndrome and hyperinflammatory states such as HLH in similar patients. Strengthening maternal HIV screening and prevention programs remains essential to avert such preventable tragedies.

Patient Consent Form / Hasta Onam Formu

The parents' of this patient consent was obtained for this study.

Conflict of Interest / Çıkar Çatışması

The author declared no conflict of interest with respect to authorship and/or publication of the article.

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Author Contributions / Yazar Katkıları

The author contributed to the conceptualization of the case report, collection and curation of clinical data, analysis and interpretation of the clinical findings, literature review, and preparation of the manuscript. The author critically revised the manuscript, approved the final version for publication, and accepts responsibility for the accuracy and integrity of the work.

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