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Long-Term Follow-Up of a Child with Congenital Toxoplasmosis: A Case Report

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ABSTRACT

Congenital toxoplasmosis is a parasitic infection caused by vertical transmission of Toxoplasma gondii from an infected pregnant woman to the fetus via the transplacental route. Without timely treatment, it can lead to significant long-term morbidity. We report a 12-month longitudinal follow-up (October 2024 – October 2025) of a 3-month-old male infant initially admitted with bronchopneumonia. Comprehensive evaluation revealed the classic triad of congenital toxoplasmosis: chorioretinitis, hydrocephalus, and multiple intracranial calcifications. The infant was born prematurely at 32–33 weeks of gestation with a history of maternal exposure to Toxoplasma gondii through contaminated water. Diagnosis was confirmed by persistently elevated IgG anti-Toxoplasma >300 IU/mL beyond 12 months of age and CT scan findings of hydrocephalus with multiple cerebral calcifications. Co-infection with cytomegalovirus (CMV) was detected by urine PCR. Cotrimoxazole (TMP-SMX) was used as an alternative first-line regimen given the unavailability of pyrimethamine and sulfadiazine. The child underwent ventriculoperitoneal (VP) shunt placement for hydrocephalus and extracapsular cataract extraction (ECCE) of the right eye. Over 12 months of follow-up, physical growth improved in accordance with age, but developmental delay persisted across all domains of the Denver II assessment. Valganciclovir therapy for CMV co-infection could not be initiated due to drug unavailability. This case highlights the critical importance of long-term multidisciplinary follow-up and active family engagement in managing congenital toxoplasmosis.

Keywords: *congenital toxoplasmosis; TORCH; cotrimoxazole; long-term follow-up; hydrocephalus; CMV co-infection; ventriculoperitoneal shunt*

INTRODUCTION

Congenital toxoplasmosis is a parasitic condition arising when the protozoan Toxoplasma gondii is vertically transmitted from an actively infected mother to the fetus through the transplacental route.[1] The incidence of congenital toxoplasmosis shows wide regional variability, with a global estimate of 1.5 cases per 1,000 live births.[2] In Indonesia, seroprevalence among pregnant women reaches 60%, with the highest regional variation in Java (51.15%) and the lowest in Nusa Tenggara (32.56%), while Sumatra registers approximately 41.9%.[3]

The pathognomonic clinical manifestations of congenital toxoplasmosis comprise chorioretinitis, hydrocephalus, and intracranial calcifications. Additional neurological features frequently encountered include micro- or macrocephaly, cerebral calcifications, epilepsy, nystagmus,

neurodevelopmental delay, and sensorineural hearing loss. [2,4] Retinal involvement is the dominant and most characteristic ophthalmological complication, and destructive changes in retinal tissue can become permanent, ultimately resulting in severe visual disability or blindness. [5,6]

Standard first-line treatment of congenital toxoplasmosis consists of a three-drug regimen: pyrimethamine, sulfadiazine, and leucovorin. Their synergistic, parasitstatic interaction administered for a full 12 months has demonstrated efficacy in reducing the risk of neurological deficits, developmental regression, and progressive retinal lesion-related visual impairment.[7] Given the ongoing challenges in diagnosis and management, and the potential impact on child development into adulthood, we present a longitudinal case report of a child with congenital toxoplasmosis.

CASE REPORT

A 3-month-old male infant, MHM, was brought to the Emergency Department, with progressively worsening respiratory distress for one day. The complaint was accompanied by low-grade fever and productive cough for one week. The infant was breastfed with good tolerance.

Birth history: delivered via caesarean section at 32–33 weeks gestation, birth weight 2,200 g, birth length 47 cm, cried immediately at birth. Neonatal jaundice was present from birth to 2 months of age. Maternal risk factor: the mother had unknowingly consumed water from a well in which a cat had died during the second trimester of pregnancy. Prior to this admission, the child had been seen by an ophthalmologist and diagnosed with congenital cataract of the right eye (OD), suspected chorioretinitis due to toxoplasmosis of the left eye (OS), and superotemporal retinal detachment OS. The child had never received any routine immunizations.

Physical Examination

The infant appeared severely ill but was alert and responsive. Vital signs: blood pressure 98/64 mmHg, heart rate 120–130 bpm (strong pulse), respiratory rate 60–70 breaths/min, temperature 37.8°C. Anthropometrics: weight 4.5 kg (WAZ 0–2 SD), length 56 cm (LAZ 0–3 SD), head circumference 40 cm (normocephalic), nutritional status adequate. Nasal flaring was present with minimal epigastric retractions. Auscultation revealed bronchovesicular breath sounds with bilateral fine crackles. The right eye showed a reduced light reflex. Cardiovascular, abdominal, and extremity examinations were unremarkable.

Investigations

Laboratory: haemoglobin 10.1 g/dL, white cell count 11,790/mm³, platelets 204,000/mm³. Arterial blood gas: pH 7.26, pCO_2 40, pO_2 71, HCO_3 17.9, BE −9.2, SaO_2 96%. Electrolytes and organ function were within normal limits. Chest X-ray: impression of bronchopneumonia.

Working Diagnoses

(1) Bronchopneumonia with respiratory distress; (2) Congenital cataract OD secondary to suspected TORCH infection; (3) Toxoplasma chorioretinitis OS; (4) Superotemporal retinal detachment OS; (5) Global developmental delay; (6) Incomplete immunization.

Initial Management

The infant was admitted to the PICU and supported with continuous positive airway pressure (CPAP). Parenteral nutrition was provided with KAEN1B. Intravenous ampicillin and gentamicin were administered, along with intravenous paracetamol for fever. Multidisciplinary consultations were arranged with the infectious diseases, respirology, neurodevelopmental-social paediatrics, and ophthalmology teams.

Semester I Follow-Up (Age 3–9 Months)

The infant was hospitalised for one week and discharged with clinical improvement. After discharge, head growth accelerated rapidly. Repeat physical examination showed head

circumference expanding from 40 cm to 47 cm within 3 months (macrocephaly), weight gain of 2.6 kg (to 6.6 kg), and length increase of 5 cm (to 60 cm).

Investigations during Semester I: complete blood count and metabolic panel were within normal limits. Hearing screening (OAE Pass/Pass, tympanometry B/B bilaterally). BERA: wave V detectable in the left ear at 40 dB. TORCH serology: IgG anti-Toxoplasma >300 IU/mL, IgM anti-Toxoplasma 0.15 IU/mL, IgG anti-CMV 22 IU/mL, IgM anti-CMV 1 IU/mL, urine CMV PCR positive. Head CT scan revealed hydrocephalus with multiple cerebral parenchymal calcifications, consistent with congenital toxoplasmosis. Neurosurgical consultation confirmed congenital hydrocephalus and ventriculoperitoneal (VP) shunt insertion was performed.

Toxoplasmosis therapy was initiated with cotrimoxazole 200/40 (2×2.5 mL orally) as an alternative first-line regimen, given the unavailability of pyrimethamine and sulfadiazine. Valganciclovir for CMV co-infection could not be started due to drug unavailability. Denver II developmental screening: all four domains (gross motor, fine motor, language, personal–social) were delayed — overall impression: abnormal. Routine immunizations were deferred as the child was consistently febrile at each scheduled visit.

Semester II Follow-Up (Age 10–16 Months)

During Semester II (ages 10–16 months), the child had a functioning VP shunt and was continuing cotrimoxazole therapy. He was clinically well and attending regular outpatient reviews with ophthalmology and ENT. Anthropometrics: weight 7.5 kg (+1.0 kg), length 71 cm (+8 cm), head circumference stable at 47 cm. Appetite was good throughout the observation period.

At 17 months of age, extracapsular cataract extraction (ECCE) with primary posterior capsulotomy of the right eye was performed by the ophthalmology team. The child received his first immunizations (DTP, hepatitis B, Hib, and oral polio), experiencing only mild post-vaccination fever without serious adverse events. Denver II repeated: all four domains remained delayed. Examination of the left eye under general anaesthesia had not yet been performed as the family had not provided consent. CMV therapy remained unavailable, and the family had not been willing to procure it independently. Repeat BERA was also pending.

Serial laboratory results are presented in Table 1. IgG anti-Toxoplasma declined gradually from >300 IU/mL (October 2024) to 273 IU/mL (September 2025), while IgM anti-Toxoplasma remained consistently negative throughout follow-up. CMV IgG increased from 22 IU/mL to 44 IU/mL by September 2025. Liver enzymes, renal function, and haematological indices remained within the reference range during cotrimoxazole therapy, indicating good tolerability.

Table 1. Serial Laboratory Results During 12-Month Follow-Up

Parameter	Oct 2024	Nov 2024	Jan 2025	May 2025	Sep 2025	Reference Range
Hb (g/dL)	10.1	11.7	13.2	12.3	10.9	10.4–16
WBC (/mm³)	11,790	5,960	13,110	11,250	11,780	6,000–18,000
IgG Toxo (IU/mL)	>300	>300	—	>300	273	—
IgM Toxo (IU/mL)	0.26	0.15	—	0.31	0.1	—
IgG CMV (IU/mL)	22	—	—	—	44	—
SGOT (U/L)	57	51	61	31	31	20–67
SGPT (U/L)	27	31	27	18	19	5–33

DISCUSSION

This case reports the clinical course of a 3-month-old male infant admitted with bronchopneumonia who was subsequently found to have congenital toxoplasmosis with the classic triad of hydrocephalus, intracranial calcifications, and chorioretinitis. Congenital toxoplasmosis belongs to the TORCH group of congenital infections and can result in significant long-term morbidity if not appropriately managed and monitored.

The presenting bronchopneumonia was consistent with the heightened susceptibility of premature infants to lower respiratory tract infections. Preterm neonates have lower transplacentally transferred maternal IgG and reduced lymphocyte counts during the first year of life, increasing vulnerability to bacterial and viral pathogens.[8] This infant was born at 32–33 weeks of gestation, placing him at elevated risk for infectious complications.

Congenital toxoplasmosis arises from vertical transmission of T. gondii from an infected mother to the fetus via the placenta.[1] Transmission risk increases with advancing gestational age, from less than 15% at 13 weeks to over 70% at 36 weeks. Routes of maternal infection include consumption of undercooked meat and ingestion of oocysts from cat faeces contaminating food, soil, or water.[2] In this case, infection was presumed to have occurred through ingestion of well water contaminated by a dead cat during the second trimester. The classic triad first described by Wolf in 1939 - chorioretinitis, hydrocephalus, and intracranial calcifications - was present in its entirety in this patient.[2,4]

Serology showed IgG anti-Toxoplasma >300 IU/mL with negative IgM. The postnatal gold standard for confirming congenital infection is persistence of IgG anti-Toxoplasma beyond 12 months of age, as passively transferred maternal IgG normally wanes within that period in uninfected infants.[9] In this patient, IgG remained >300 IU/mL through 9 months and declined gradually to 273 IU/mL at 12 months, confirming active congenital infection. Negative IgM does not exclude congenital toxoplasmosis, as it may be undetectable due to early elimination, assay sensitivity limitations, or timing of sampling.[10]

CMV co-infection was identified by urine PCR. Co-infection with multiple TORCH pathogens frequently worsens prognosis and amplifies the risk of neurodevelopmental delay.[11] However, the diffuse pattern of calcifications and the presence of chorioretinitis were more consistent with toxoplasma as the dominant pathogen. Regarding valganciclovir for CMV, evidence indicates that treatment initiated beyond one month of age does not improve sensorineural hearing loss, as the optimal therapeutic window is before 1–2 months of age.[12,13] Since CMV PCR was performed after 3 months, postnatal acquisition cannot be excluded, further limiting the benefit of antiviral therapy.

The standard treatment for congenital toxoplasmosis - pyrimethamine, sulfadiazine, and leucovorin for 12 months - was unavailable in this setting.[7] Cotrimoxazole (TMP-SMX) was used as the alternative regimen. Trimethoprim inhibits dihydrofolate reductase (DHFR) and sulfamethoxazole inhibits dihydropteroate synthase (DHPS), acting synergistically on the parasite's folate metabolism pathway - analogous to the mechanism of pyrimethamine–sulfadiazine.[14,15]

Multiple studies have demonstrated comparable efficacy of TMP-SMX with a more favourable safety profile and lower discontinuation rates due to toxicity.[16,17] Serial laboratory monitoring in this patient revealed no significant haematological adverse events throughout therapy.

Hydrocephalus, occurring in approximately 4.4% of congenital toxoplasmosis cases, requires VP shunt insertion.[18,19] Shunt placement in this patient successfully stabilised the previously rapidly enlarging head circumference. ECCE of the right eye was performed at 17 months to prevent stimulus-deprivation amblyopia from

congenital cataract, which arises from disrupted lens formation at the time of intrauterine infection.

Neurodevelopmental impairment occurs in 38.8–62% of children with congenital toxoplasmosis. [20,18] In this patient, developmental delay persisted across all Denver II domains, though incremental progress was noted — the child acquired the ability to roll independently and consistently responded to sound by the end of follow-up. If diagnosis is established early and antiparasitic therapy initiated promptly, the overall prognosis is favourable: 74% of treated children remain asymptomatic, and more than 72% of those with moderate-to-severe neurological findings at birth achieve normal neurocognitive development. [19,21] Non-medical challenges encountered in this case — including distance from tertiary care, limited access to certain therapies and diagnostic procedures, and parental hesitancy regarding some interventions — are common in resource-constrained settings. Integrated multidisciplinary one-stop-service clinics and telemedicine platforms are recommended strategies to optimise management in the face of such access barriers.

CONCLUSION

This case illustrates the management and 12-month longitudinal follow-up of a child with congenital toxoplasmosis and concurrent CMV co-infection. Cotrimoxazole proved to be an effective and safe alternative when the first-line regimen was unavailable, with serial laboratory monitoring confirming good tolerability. VP shunt placement and ECCE provided clear clinical benefit. Persistent neurodevelopmental delay underscores the need for early, intensive, and sustained rehabilitation. Long-term coordinated multidisciplinary follow-up — encompassing neurology, ophthalmology, otolaryngology, and developmental paediatrics — is essential to optimise functional outcomes in children with congenital toxoplasmosis.

Declaration by Authors

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