



Review Article

Congenital Tuberculosis: A Comprehensive Review of a Rare but Critical Neonatal Infection

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Abstract

Congenital tuberculosis (TB) remains a rare but serious infection with significant morbidity and mortality when diagnosis is delayed. Transmitted vertically from infected mothers to their fetuses via the placenta or amniotic fluid, this condition poses unique diagnostic challenges due to its nonspecific clinical presentation in newborns. This review synthesizes current knowledge on the epidemiology, pathogenesis, clinical manifestations, diagnostic approaches, and management of congenital tuberculosis. With fewer than 300 cases reported worldwide, maintaining a high index of suspicion—particularly in infants born in TB-endemic regions or to mothers with risk factors—remains essential for timely intervention and improved outcomes

Introduction

Tuberculosis remains a major global health concern, with an estimated 10 million new cases annually worldwide. While pulmonary TB in adults is common, congenital tuberculosis—infection transmitted from mother to fetus—represents a rare but clinically significant manifestation of this ancient disease. Historically considered almost nonexistent, congenital TB has gained renewed attention as changes in TB epidemiology and increased awareness have led to more frequent recognition.

The diagnosis of congenital tuberculosis requires distinction from postnatally acquired infection. The classic criteria established by Bietykl in 1935 remain relevant today and require three elements: (1)

confirmation of tuberculous lesions; (2) presence of a primary hepatic complex, indicating hematogenous transmission via the umbilical vein; and (3) onset of disease at birth or within days after delivery, effectively excluding postnatal infection.

Epidemiology and Risk Factors

Congenital tuberculosis is a disease of mothers, not just infants. The infection occurs when a pregnant woman has active tuberculosis, particularly with hematogenous dissemination or involvement of the placenta, endometrium, or cervix. The risk of vertical transmission correlates with the timing and severity of maternal infection. Women with miliary or disseminated TB, those co-infected with HIV, and those diagnosed late in pregnancy or postpartum appear at highest risk of transmitting infection to their offspring

Pathogenesis and Transmission Routes

The pathogenesis of congenital tuberculosis involves several potential mechanisms of vertical transmission. The most widely accepted route is hematogenous spread through the umbilical vein, with bacilli reaching the fetal liver and establishing a primary hepatic complex. From this site, organisms may disseminate widely, explaining the multisystem involvement characteristic of congenital infection

Alternative transmission routes include aspiration or ingestion of infected amniotic fluid, which may occur when placental or endometrial lesions rupture into the amniotic cavity. This mechanism can produce primary

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pulmonary or gastrointestinal complexes, respectively, and explains cases where hepatic involvement is absent.

Histopathological examination of placental tissue in cases of congenital TB typically reveals characteristic caseating granulomas with abundant acid-fast bacilli. The fetal response to infection is notably poor, with lesions showing extensive caseous necrosis, abundant organisms, but minimal lymphocytic infiltration or granuloma formation—reflecting the relative immunological immaturity of the fetus and neonate

Severe and Disseminated Disease

Without treatment, congenital tuberculosis progresses to disseminated disease. Miliary patterns on chest radiography are characteristic, with bilateral diffuse micronodular infiltrates reflecting hematogenous spread. Meningitis occurs in a substantial proportion of cases, manifesting as irritability, seizures, bulging fontanelle, or altered consciousness. Abdominal involvement may produce distension, ascites, and hepatic dysfunction. The mortality of untreated congenital tuberculosis approaches 50%, and even with appropriate therapy, delays in diagnosis increase the risk of poor outcomes, including chronic pulmonary sequelae, neurodevelopmental impairment, and death

Treatment

Treatment of congenital tuberculosis follows the same principles as treatment of pediatric TB, with adjustments for age and disease severity. All regimens should be initiated promptly when the diagnosis is suspected, without waiting for microbiological confirmation, given the high mortality of untreated disease.

First-Line Regimens

The standard intensive phase consists of four drugs: isoniazid (10-15 mg/kg daily), rifampin (10-20 mg/kg daily), pyrazinamide (30-40 mg/kg daily), and ethambutol (15-25 mg/kg daily) or an aminoglycoside such as amikacin. This four-drug regimen is continued for two months, followed by a continuation phase of isoniazid and rifampin for an additional four to ten months, depending on disease severity and extent

Prognosis and Outcomes

The prognosis of congenital tuberculosis depends primarily on early diagnosis and prompt initiation of appropriate therapy. With timely treatment, most infants achieve full recovery, although long-term follow-up is essential to monitor for complications such as bronchiectasis, obstructive lung disease, and neurodevelopmental sequelae.

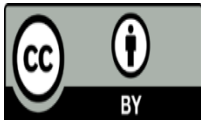
Conclusion

Congenital tuberculosis, though rare, represents a critical diagnostic and therapeutic challenge in neonatal medicine. Its nonspecific presentation, rapid progression, and high mortality demand a high index of suspicion, particularly in infants born to mothers from TB-endemic regions or with risk factors for infection. The key to improved outcomes lies in considering the diagnosis early, evaluating both mother and infant thoroughly, and initiating treatment promptly when clinical suspicion is strong. As global migration patterns and TB epidemiology continue to evolve, awareness of this condition remains essential for clinicians caring for newborns worldwide

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