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Diagnostic Challenges of Pediatric Tuberculosis in a Severely Malnourished Child from a Rural Area: A Case Report

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Abstract
Introduction: *Tuberculosis (TB) remains a major global health problem affecting both adults and children, particularly in TB-endemic countries. Children are vulnerable to TB infection, especially those with malnutrition, which significantly impairs immune function and increases disease severity, mortality, and diagnostic challenges. Malnutrition may obscure typical clinical manifestations and reduce the sensitivity of standard diagnostic tests, leading to delayed diagnosis and treatment.* ***Case description:*** *a four-year-old girl with severe wasting, multiple cervical lymphadenopathy, hypoalbuminemia, and severe dehydration. The patient had a history of incomplete immunization and close contact with a TB case. Initial investigations revealed a negative tuberculin skin test and chest radiography suggestive of bilateral bronchopneumonia. Despite intravenous ceftriaxone and supportive therapy, no clinical improvement was observed. Further evaluation using a rapid molecular test from a gastric aspirate confirmed TB.* ***Discussion:*** *Severe malnutrition likely contributed to immune anergy, resulting in false-negative initial diagnostic findings and delayed TB diagnosis. This case highlights the limitations of conventional diagnostic methods in malnourished children and underscores the importance of molecular testing in high-risk pediatric patients.* ***Conclusion:*** *TB and malnutrition have a bidirectional relationship that worsens clinical outcomes. Early TB screening in malnourished children and routine nutritional assessment in TB patients are essential to improve diagnosis, treatment outcomes, and survival.*

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Introduction

Tuberculosis (TB) remains a major global health problem affecting both adults and children, particularly in low- and middle-income countries. According to the World Health Organization, children account for a substantial proportion of undiagnosed TB cases due to non-specific clinical manifestations and diagnostic difficulties (World Health Organization) (World Health Organization, 2023). In Indonesia, despite the national target to end tuberculosis, pediatric TB continues to pose significant diagnostic and management challenges.

Malnutrition is one of the most important risk factors for tuberculosis in children. It impairs both innate and adaptive immune responses, increases susceptibility to infection, and worsens disease severity and treatment outcomes (Seddon et al., 2021). Children with severe malnutrition often present with atypical or subtle symptoms, which may obscure classical signs of TB and reduce the sensitivity of commonly used diagnostic tests such as the tuberculin skin test (Munthali et al., 2021; Snow et al., 2020).

In resource-limited and rural settings, these challenges are further compounded by delayed access to diagnostic facilities and reliance on clinical judgment. This case report describes the diagnostic challenges of bacteriologically confirmed tuberculosis lymphadenitis in a severely malnourished child and highlights the importance of early molecular testing and integrated management of TB and malnutrition.

Case Description

A four-year-and-six-month-old girl from Dompu, West Nusa Tenggara, was admitted to the pediatric ward with complaints of multiple neck and postauricular masses that had gradually increased in size over the past year. Three months prior to admission, an additional mass appeared in the right inguinal region. The masses were painless, mobile, and progressively enlarging. The child also had intermittent low-grade fever, progressive weight loss, abdominal distension, and poor appetite.

The patient had a history of incomplete immunization and lived in a rural environment. Both parents worked as farmers with low household income. The patient lived with extended family members, and a neighbor within a 500-meter radius was undergoing treatment for tuberculosis. There was no history of previous TB treatment.

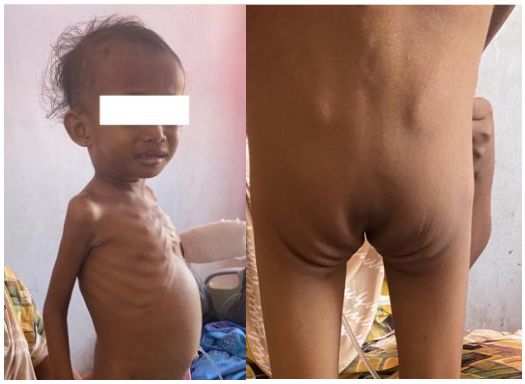
On physical examination, the patient appeared severely ill with marked wasting and lethargy. Vital signs revealed fever (38°C), tachycardia, and tachypnea. Anthropometric measurements showed severe acute malnutrition with a body weight of 10 kg, height of 93 cm, mid-upper arm circumference of 10 cm, and weight-for-height Z-score below −3 SD. Clinical features included pallor, sparse hair, “old-looking” facial appearance, prominent ribs, abdominal distension with shifting dullness, and baggy pants sign. Multiple cervical and submandibular lymph nodes measuring up to 2 × 3 cm were palpated, along with a solitary right inguinal lymph node.

Laboratory investigations revealed severe microcytic hypochromic anemia consistent with anemia of chronic disease and hypoalbuminemia. The tuberculin skin test was negative. Chest radiography demonstrated bilateral infiltrates consistent with bronchopneumonia and right hilar lymphadenopathy. Abdominal radiography revealed ascites and fecal impaction. The pediatric TB scoring system yielded a score of 7, suggesting clinical tuberculosis.

Despite empirical intravenous ceftriaxone and supportive therapy, the patient showed no clinical improvement. A gastric aspirate sample was obtained through a nasogastric tube and tested using a rapid molecular assay, which confirmed

Mycobacterium tuberculosis. The patient was diagnosed with bacteriologically confirmed tuberculosis lymphadenitis with severe acute malnutrition (marasmus–kwashiorkor).

Treatment included anti-tuberculosis fixed-dose combination therapy, nutritional rehabilitation using F-75 formula during the stabilization phase, micronutrient supplementation, blood transfusion for severe anemia, and supportive care. Gradual clinical improvement was observed, including resolution of fever, improved appetite, and stabilization of nutritional status.



Picture 1. Clinical symtomps of the patient

Tabel 1
Hematologic test results

Hematology	03/05/2024	10/05/2024	12/05/2024	Reference
Hb	**7.1 ↓**	**6.3 ↓**	**9 ↓**	12-15 g/dL
Hematocrit	**25.3 ↓**	**22.9 ↓**	**31 ↓**	35-46%
RBC	**4.08 ↓**	**3.7 ↓**	4.56	4-5.2 million/uL
MCV	**62 ↓**	**62 ↓**	**68 ↓**	80-100 fL
MCH	**17.4 ↓**	**17 ↓**	**19.7 ↓**	26-34 pg
MCHC	**28.1 ↓**	**27.5 ↓**	**29 ↓**	32-36 g/dL
RDW-CV	20.80	19.60	22.30	11.5-14.5%
WBC	8.10	6.00	5.90	4.500-13.500 /uL
Blood count				
Lymph	1.700	900	1.20	1000-7.200
MID	900	800	0.70	0-1.600
GRA	5.500	4.300	4.00	1.700-7.800
LYM%	20.9	15.00	19.9	13.1-52.9%
MID%	11.2	14.1	11.8	0-11%
GRA%	67.9	70.9	68.3	24-54%
Platelet	398	347	301	150.000-450.000 /uL

Table 2
Chemical lab test results

Name of lab test	Value	Reference
SGOT (AST)	26	<52 U/L
SGPT (ALT)	7	<39 U/L
BUN	14.95	6-20 mg/dl
Kreatinin	0.33	0.5-0.9 mg/dl
GDS	79	80-139 mg/dl
Albumin	**3.09 ↓**	3.97-4.94g/dL

1. Discussion

The clinical presentation of this patient is characteristic of chronic pediatric tuberculosis complicated by severe acute malnutrition. Prolonged painless lymphadenopathy involving cervical, submandibular, and inguinal regions is a hallmark of tuberculous lymphadenitis, the most common extrapulmonary manifestation of tuberculosis in children. The gradual enlargement, absence of tenderness, and long disease duration strongly suggest a granulomatous ethology rather than acute bacterial infection (Kementerian Kesehatan Republik Indonesia, 2023; Martinez et al., 2021).

Systemic manifestations such as intermittent low-grade fever, progressive weight loss, and failure to thrive reflect the chronic inflammatory state induced by *Mycobacterium tuberculosis*. Persistent immune activation leads to increased production of pro-inflammatory cytokines including tumours necrosis factor-alpha (TNF-α), interleukin-1 (IL-1), and interferon-gamma (IFN-γ), which promote anorexia, muscle catabolism, and impaired growth in children (Ma et al., 2022; Seddon et al., 2021).

Severe acute malnutrition in this patient showed features of overlapping marasmus–kwashiorkor, evidenced by extreme wasting, edema, ascites, hypoalbuminemia, and classical physical signs such as baggy pants and an aged facial appearance. Protein-energy malnutrition causes thymic atrophy, reduced CD4+ T-cell count, impaired macrophage activation, and decreased cytokine production, resulting in profound suppression of cell-mediated immunity, which is crucial for controlling tuberculosis infection (Dipasquale et al., 2020; Soeters et al., 2019).

The negative tuberculin skin test observed in this patient can be explained by immune anergy associated with severe malnutrition. Tuberculin skin testing relies on intact delayed-type hypersensitivity mediated by T lymphocytes. In malnourished children, impaired T-cell responses frequently lead to false-negative results, and therefore a negative test does not exclude active tuberculosis (Bunker & Pandey, 2021; Kementerian Kesehatan Republik Indonesia, 2023).

Radiological findings in paediatric tuberculosis are often non-specific, particularly in younger children. The presence of bilateral infiltrates and right hilar lymphadenopathy in this patient reflects lymphatic dissemination from a primary pulmonary focus, known as the primary TB complex. Such findings are frequently misinterpreted as bronchopneumonia, leading to inappropriate empirical antibiotic therapy and diagnostic delay (Marais et al., 2019; World Health Organization, 2022).

The absence of clinical improvement following broad-spectrum antibiotic therapy represents a critical diagnostic clue. In TB-endemic settings, failure to respond to appropriate antibiotics should prompt re-evaluation for tuberculosis, especially in children with risk factors such as malnutrition, household exposure, and prolonged symptoms (Marais et al., 2019; World Health Organization, 2023).

Bacteriological confirmation using rapid molecular testing was essential in establishing the diagnosis in this case. Molecular assays such as Xpert MTB/RIF detect M. tuberculosis DNA with higher sensitivity than smear microscopy and are particularly valuable in paediatric and extrapulmonary TB, where bacterial load is low. Current guidelines recommend molecular testing as the primary diagnostic tool in suspected paediatric TB cases with severe disease or diagnostic uncertainty (Cuevas et al., 2020; World Health Organization, 2022).

Haematological abnormalities in this patient included severe microcytic hypochromic anaemia, consistent with anaemia of chronic disease compounded by nutritional deficiencies. Chronic inflammation in tuberculosis increases hepcidin production, which impairs iron absorption and mobilization, while malnutrition further disrupts erythropoiesis through deficiencies in iron, folate, and other micronutrients (Gao et al., 2023; Kurniaji et al., 2023).

Hypoalbuminemia and ascites observed in this patient result from both protein deficiency and systemic inflammation. Inflammatory cytokines suppress hepatic albumin synthesis, while inadequate dietary protein intake limits substrate availability. Reduced plasma oncotic pressure leads to oedema and fluid accumulation, which are classical features of kwashiorkor and severe inflammatory states (Dipasquale et al., 2020; Soeters et al., 2019).

Management of this patient required an integrated approach addressing both tuberculosis and severe malnutrition. Anti-tuberculosis therapy using paediatric fixed-dose combinations was initiated according to national guidelines. Nutritional rehabilitation followed the stabilization phase protocol using F-75 formula to correct metabolic derangements while minimizing the risk of refeeding syndrome (Kementerian Kesehatan Republik Indonesia, 2020; World Health Organization, 2011).

Adjunctive therapies, including vitamin B6, folic acid, zinc supplementation, and blood transfusion, were administered based on clinical indications. Vitamin B6 supplementation is essential during isoniazid therapy, particularly in malnourished children, to prevent neurotoxicity. Zinc supplementation has been shown to improve serum albumin levels and reduce inflammatory stress during tuberculosis treatment (Zolfaghari et al., 2021; Wahidiyat & Adnani, 2016).

Children with concurrent tuberculosis and severe malnutrition are at high risk for complications such as treatment failure, drug toxicity, secondary infections, and increased mortality. Malnutrition alters drug pharmacokinetics and immune recovery, necessitating close monitoring and prolonged nutritional support. Early diagnosis and comprehensive management are critical to improving prognosis and survival in this vulnerable population (Ma et al., 2022; Slogrove et al., 2020).

Conclusion

Severe malnutrition can significantly alter the clinical presentation and diagnostic accuracy of pediatric tuberculosis, leading to delayed diagnosis and treatment. Negative tuberculin skin tests and non-specific radiological findings should not exclude TB in high-risk children. Early molecular diagnostic testing and integrated nutritional rehabilitation are crucial to improving outcomes in malnourished children with suspected tuberculosis.

Limitation

This case report is limited by its single-patient design, which restricts generalizability to broader pediatric populations. The lack of comparative data and limited follow-up also preclude assessment of long-term outcomes and the relative impact of severe malnutrition on diagnostic delay.

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