

Case report of idiopathic thrombocytopenic purpura and tuberculosis in a 47-year-old male

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ABSTRACT

Idiopathic Thrombocytopenic Purpura (ITP) is a condition of low platelet count of unknown cause where most of the causes of ITP are the result of antibodies binding to platelets. One of the extra pulmonary manifestations of TB occurs in the hematological system which can cause anemia, leukopenia, leukocytosis, thrombocytopenia and thrombocytosis and ITP due to Tuberculosis (TB) is a rare manifestation of TB. This case report aims to describe a 47 year old male patient with ITP accompanied by TB. A 47 year old male patient came with complaints of weakness since 7 days ago. Based on the history, physical and supporting examinations carried out, the patient was diagnosed with ITP due to pulmonary TB. Manifestations of ITP due to TB are similar to ITP in general. Generalized purpura, bruising after minor trauma, epistaxis, subconjunctival hemorrhage and hematuria are possible manifestations. Treatment for ITP is to treat the underlying disease that causes it and specific treatment for ITP can also be given if the patient's platelets are very low. The specific treatment for ITP that can be given is corticosteroids or IVIG. Idiopathic Thrombocytopenic Purpura is a rare hematological manifestation of TB. Correct diagnosis and treatment play an important role in reducing the worsening prognosis of ITP patients due to TB.

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INTRODUCTION

Idiopathic Thrombocytopenic Purpura (ITP) is a condition characterized by a low platelet count (thrombocytopenia) with no known cause (idiopathic) (Sandal et al., 2021; Thakre et al., 2023; Thaware et al., 2022). ITP, also called Idiopathic Thrombocytopenic Purpura, is an autoimmune disorder characterized by a low platelet count due to excessive platelet destruction (Mititelu et al., 2024; Sandal et al., 2021; Tärniceriu et al., 2022). Most cases of ITP result from antibodies binding to platelets, leading to the alternate designation of the disease as Immune Thrombocytopenic Purpura (Haider et al., 2022; Kayal et al., 2014; Yadav et al., 2021). In severe cases of thrombocytopenia, it

can potentially lead to death due to blood loss or bleeding in vital organs (Santoshi et al., 2022; Scharf, 2021). According to the American Society of Hematology, ITP is defined by a platelet count $<100,000/\mu\text{L}$ (Kashiwagi et al., 2020; Kuter, 2019), accompanied by normal leukocyte and hemoglobin levels, in the presence of a generalized purpura rash (Haider et al., 2022). Idiopathic thrombocytopenic purpura is classified into primary and secondary ITP. Primary ITP is characterized by isolated thrombocytopenia, while secondary ITP is associated with other disorders such as autoimmune conditions (systemic lupus erythematosus), viral infections (hepatitis C), lymphoproliferative neoplasms, and others (Onisâi et al., 2019).

The incidence of ITP varies depending on the source. According to Sherwin et al., the estimated incidence of ITP is 2 to 5 cases per 100,000 people in the general population (DeSouza & Angelini, 2021). Another study suggests that the incidence of ITP in adults is approximately 66 cases per 1,000,000 people per year, while in children, it is around 50 cases per 1,000,000 people per year (Gauer & Braun, 2012).

Tuberculosis (TB) is a disease caused by the germ *Mycobacterium tuberculosis* and can manifest in both the lung and extra-lung regions (Charifa et al., 2023). One of the extra-pulmonary manifestations of TB affects the hematological system, leading to conditions such as anemia, leukopenia, leukocytosis, thrombocytopenia, and thrombocytosis (Khan et al., 2021). The manifestation of immune thrombocytopenic purpura (ITP) caused by TB is a rare occurrence. Therefore, this case report aims to describe a 47-year-old male patient with ITP accompanied by pulmonary TB based on history, physical examination, and the support and management provided to the patient.

The importance of conducting research on idiopathic thrombocytopenic purpura (ITP) and tuberculosis (TB) stems from the infrequency and intricacy of the co-occurrence of these two conditions, especially in comprehending the pathophysiological mechanisms and optimal treatment approaches. ITP is an uncommon autoimmune disorder characterized by a decreased platelet count, whereas TB is a severe infectious illness caused by *Mycobacterium tuberculosis*. The development of ITP as a result of TB is an unusual occurrence, and there are no specific protocols for effectively addressing this situation. Given the potential life-threatening complications linked to severe thrombocytopenia and TB, such as bleeding and organ impairment, urgent research is imperative to enhance early detection, improve treatment outcomes, and establish comprehensive guidelines for managing ITP within the context of TB.

The anticipated publication of a case report detailing the coexistence of idiopathic thrombocytopenic purpura (ITP) and tuberculosis (TB) in a 47-year-old male patient holds great promise for advancing medical research. This case report sheds light on the infrequent presentation of ITP as a consequence of TB, thereby enhancing our comprehension of the various hematological manifestations associated with this infectious disease. By addressing existing knowledge gaps and clinical practices, further investigation in this domain can significantly influence patient care and outcomes, underscoring the urgent and focused need for research into this uncommon hematological manifestation of TB-induced ITP.

RESEARCH METHOD

A 47-year-old male patient presented to the Emergency Department (ED) of RSUD RAA Soewondo Pati with a complaint of weakness persisting for the past 7 days. The weakness was continuous, progressively worsening, especially during activities, and offered no relief with rest. The patient also reported a lack of appetite, particularly during periods of fever. Additionally, he described a cough that had been present for the past 7 days, accompanied by weight loss. The cough, worse at night and without phlegm, was associated with shortness of breath, particularly when lying down. However, this symptom improved when sitting or using a sufficiently elevated pillow. The patient denied other complaints such as nausea, vomiting, loss of consciousness, seizures, or bowel and bladder dysfunction. There was no family history of similar complaints, no known drug allergies,

and no history of degenerative diseases. The patient had a past medical history of pulmonary tuberculosis but had never undergone treatment.

Upon physical examination, vital signs revealed a blood pressure of 100/70 mmHg, pulse rate of 102 beats/min, temperature of 36.0°C, and respiratory rate of 18 breaths/min. Systemic examination indicated ronchi in both lung fields, decreased stem fremitus on the right chest, and petechiae on all four extremities. Other examinations yielded results within normal limits. Laboratory and radiology tests were conducted, revealing a decrease in erythrocytes (4.09 10⁶/μL), hemoglobin (11.0 g/dL), and hematocrit (33.5%). Platelet count was also reduced (2/μL), with elevated SGOT (171.4 U/L) and SGPT (77.2 U/L) levels, decreased sodium (130.3 mmol/dL), and reduced albumin (2.8 g/dL). Thoracic X-ray indicated active pulmonary TB and right pleural effusion.

Based on history, physical examination, and support, the patient was diagnosed with ITP with active pulmonary TB accompanied by pleural effusion and hyponatremia. The patient received initial management in the emergency room, including lactated Ringer's fluid infusion at 16 drops/minute, nasal cannula oxygen at 3 Lpm, Omeprazole injection at 1x40 mg, and Salbutamol at 3x2 mg. Following the initial treatment, the patient was admitted to the ward and received further management, including Ceftriaxone injection at 1x2 g, Methylprednisolone injection at 3x1, Tranexamic acid injection at 3x1, N-Acetylcysteine at 3x200 mg, and NaCl capsules at 3x1.

RESULTS AND DISCUSSIONS

Idiopathic Thrombocytopenic Purpura is an autoimmune disease characterized by a low platelet count, purpura, and hemorrhagic episodes caused by antiplatelet autoantibodies. It can arise from various factors, including infection, malignancy, immunodeficiency, and autoimmune diseases (AA & Gupta, 2019). Additionally, it can be associated with hematogenous manifestations of tuberculosis (TB). The occurrence of Idiopathic Thrombocytopenic Purpura due to TB is rare, with only a few reported cases (Mohamed et al., 2020). The pathophysiology of TB-induced ITP is not fully understood. It occurs through multiple mechanisms, such as immune-mediated platelet destruction facilitated by antiplatelet antibodies shared between Mycobacterium tuberculosis and platelets. Other contributing factors include platelet-associated immunoglobulins, tuberculosis-induced hemophagocytic syndrome, reduced platelet production due to bone marrow infiltration, hypersplenism, intravascular coagulation, and Anti-tuberculosis Drug (OAT)-induced thrombocytopenia (Alkhatib et al., 2021; Mohamed et al., 2020).

Table 1. Laboratory results (07/09/2022)

	Result	Normal Value
Total Leukocytes Count	12.7	3.8-10.6
Total Erythrocytes Count	4.8	4.7-6.1
Hemoglobin	12.5	13.2-17.3
Hematocrit	37.6	40-52
Mean Corpuscular Volume (MCV)	83.6	80-100
Mean Corpuscular Hemoglobin (MCH)	29.2	26-34
Mean Corpuscular Hemoglobin Concentration (MCHC)	34.9	32-36
Total Platelet Count	3	150-400
Neutrophil	49.90	50.0-70.0
Lymphocyte	8.00	25.0-40.0
Monocyte	10.20	2.0-8.0
Eosinophil	5.50	2-4
Basophil	0.10	0-1
Glucose	97	70-160
Urea	14.1	10-50

	Result	Normal Value
Creatinine	0.66	0.60-1.20
Blood Sodium	130.3	135-155
Blood Potassium	4.67	3.6-5.5
Blood Chloride	93.3	95-108

Table 2. Laboratory results (09/09/2022)

	Result	Normal Value
Total Leukocyte Count	14.7	3.8-10.6
Total Erythrocyte Count	4.47	4.7-6.1
Hemoglobin	12.1	13.2-17.3
Hematocrit	35.6	40-52
Mean Corpuscular Volume (MCV)	79.6	80-100
Mean Corpuscular Hemoglobin (MCH)	29.2	26-34
Mean Corpuscular Hemoglobin Concentration (MCHC)	34.9	32-36
Total Platelet Count	2	150-400
Neutrophils	49.90	50.0-70.0
Lymphocytes	8.00	25.0-40.0
Monocytes	10.20	2.0-8.0
Eosinophils	5.50	2-4
Basophils	0.10	0-1

Table 3. Laboratory results (10/09/2022)

	Result	Normal Value
Total Leukocyte Count	10.1	3.8-10.6
Total Erythrocyte Count	4.09	4.7-6.1
Hemoglobin	11.0	13.2-17.3
Hematocrit	33.5	40-52
Mean Corpuscular Volume (MCV)	83.0	80-100
Mean Corpuscular Hemoglobin (MCH)	29.2	26-34
Mean Corpuscular Hemoglobin Concentration (MCHC)	34.9	32-36
Total Platelet Count	2	150-400
Neutrophils	49.90	50.0-70.0
Lymphocytes	8.00	25.0-40.0
Monocytes	10.20	2.0-8.0
Eosinophils	5.50	2-4
Basophils	0.10	0-1

Table 4. Results of data analysis

Parameter	Laboratory Results
Platelet count	$2 \times 10^3/\mu\text{L}$
Leukocyte levels	$12.7 \times 10^3/\mu\text{L}$
Erythrocyte levels	$4.09 \times 10^6/\mu\text{L}$
Hemoglobin	11.0 g/dL
Hematocrit	33.5%
SGOT	171.4 U/L
SGPT	77.2 U/L
Thoracic X-ray	Active pulmonary TB and right pleural effusion

TB-induced Idiopathic Thrombocytopenic Purpura presents clinical manifestations similar to general ITP, which can manifest acutely or chronically. Acute manifestations include generalized purpura, bruising after minor trauma, epistaxis, subconjunctival bleeding, and hematuria. Chronic manifestations are typically silent or asymptomatic and may involve petechiae and bleeding that is challenging to stop (Park et al., 2022; Yadav et al., 2021). Supportive examinations can confirm the diagnosis. In ITP patients, the platelet count is usually $10-100 \times 10^9/L$, and hemoglobin concentration and white blood cell count are typically within the normal range. Blood tests reveal significantly reduced platelet counts, often in large numbers, with no morphologic abnormalities in

other cell lines (Robier, 2020; Zaninetti & Greinacher, 2020). Bone marrow examination shows normal or increased megakaryocyte counts. Sensitive tests can detect specific anti-glycoprotein GPIIb/IIIa or GPIb antibodies on the surface of platelets or in the serum of most (Porcelijn et al., 2020; Weber et al., 2024; Yadav et al., 2021).

This aligns with the typical manifestations of ITP in patients, where the primary complaints are general symptoms such as weakness, difficulty in daily activities, and weight loss. During the physical examination, purpura was observed on all four extremities of the patient. A supporting blood laboratory examination revealed decreased platelet levels ($2 \times 10^3/\mu\text{L}$) and increased leukocyte levels ($12.7 \times 10^3/\mu\text{L}$) due to TB infection.

The management of ITP involves addressing the underlying disease. In this case, the underlying cause of ITP is a TB infection, necessitating the administration of OAT to resolve the TB disease. Additionally, patients may receive ITP-specific therapy if their platelet levels are critically low (Alkhatib et al., 2021). First-line therapies include glucocorticoids and IVIG, both proven to be effective and well-tolerated by patients. Prednisone is a suitable glucocorticoid with an initial dose of 1 mg/KgBW/day in adults, tapered off after 10-14 days. Intravenous immunoglobulin therapy is administered at a dose of 400 mg/kg/day for 5 days or 1 g/kg/day for 2 days (Alkhatib et al., 2021).

In this case, the patient received causal therapy that did not align with the recommended treatment for the disease. Although the patient should have been given OAT, they declined the therapy. Consequently, the patient was administered Ceftriaxone injection (1x2 g), Omeprazole injection (1x40 mg), Tranexamic acid injection (3x1), N-Acetylcysteine (3x200 mg), and NaCl capsules (3x1). Specific therapy for ITP involved the administration of the acorticosteroid Methylprednisolone injection (3x1 ampoule).

Studies conducted by Mohamed et al. emphasize the crucial role of anti-tuberculosis treatment in managing ITP in TB patients (Mohamed et al., 2020). The absence of OAT in this case could have a significant impact on the course of the patient's disease. Consequently, it can be concluded that the prognosis for this patient is *dubia ad malam*. Potential complications in patients may include extensive lung damage, Horner's syndrome, Acute Respiratory Distress Syndrome, miliary spread or disseminated tuberculosis, and death.

CONCLUSION

Idiopathic Thrombocytopenic Purpura is a rare hematologic manifestation of TB. Early diagnosis and appropriate management are crucial in mitigating the worsening prognosis of patients with TB-induced ITP. Currently, there is no specific guideline for managing ITP due to TB. Treatment involves addressing both TB and ITP separately. Therefore, further research is essential to develop comprehensive guidelines for the effective management of this condition.

The study has certain limitations, such as its reliance on a single-case design, the absence of longitudinal data, limited follow-up, and the lack of comparative analysis. To overcome these limitations, future research endeavors can be directed towards conducting multi-center studies involving a larger population. Additionally, implementing longitudinal cohort studies to continuously monitor patients can provide valuable insights. Furthermore, performing comparative analysis with different patient subgroups can offer a more comprehensive understanding of the subject matter. Lastly, investigating the underlying mechanisms of the relationship between TB and ITP can contribute to a stronger foundation for the development of more effective treatment strategies. By addressing these aspects, a deeper understanding of TB-induced ITP can be achieved, leading to the advancement of more efficient treatment approaches in the future.

The research findings have significant implications and contributions across various domains. Firstly, from a clinical perspective, this study offers valuable insights into the hematological manifestations of TB and the potential complications associated with concurrent

autoimmune disorders. It aids in a more profound comprehension of the pathophysiological mechanisms underlying TB-induced ITP. Secondly, the study underscores the significance of early detection and appropriate management to mitigate adverse outcomes in patients with ITP linked to TB. It stresses the importance of a personalized treatment strategy that addresses TB and ITP separately, highlighting the necessity of tailored care for complex medical conditions. Thirdly, the research identifies the absence of specific guidelines for managing TB-induced ITP, indicating the requirement for further research to establish comprehensive guidelines for effective treatment. This could result in enhanced clinical practices, standardized treatment approaches, and improved outcomes for patients with these uncommon hematological conditions. Lastly, this case study sets the stage for future investigations focusing on large-scale studies, multi-center collaborations, longitudinal follow-ups, and comparative analyses to advance our knowledge of TB-induced ITP. By pinpointing gaps in current clinical understanding and practice, this research lays the groundwork for additional studies aimed at optimizing patient care and outcomes in similar scenarios.

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