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(CASE REPORT)

When immune thrombocytopenia purpura unveils congenital CMV infection: A case report

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Abstract

Immune thrombocytopenic purpura (ITP) is an acquired autoimmune disorder characterized by immune-mediated platelet destruction and impaired thrombopoiesis. In infants, the occurrence of ITP requires a systematic search for secondary causes, particularly infectious etiologies. Cytomegalovirus (CMV), the most frequent cause of congenital viral infection worldwide, is known to induce profound immune dysregulation and hematopoietic abnormalities. We report the case of a three-month-old infant presenting with severe and relapsing ITP revealing a congenital CMV infection. Platelet recovery was achieved only after initiation of antiviral therapy. This case highlights the importance of an integrated immunological and biochemical diagnostic approach in infantile ITP and underscores the pathogenic role of CMV in immune-mediated thrombocytopenia.

Keywords: Immune thrombocytopenia; Congenital cytomegalovirus infection; Infancy; Immune dysregulation; Megakaryopoiesis; Antiviral therapy

1. Introduction

Immune thrombocytopenic purpura (ITP) is defined as an isolated platelet count below 100×10^9/L resulting from an imbalance between increased peripheral platelet destruction and inadequate bone marrow platelet production [1]. It represents the most common benign hematological disorder in childhood, with an estimated annual incidence of 2–6 cases per 100,000 children [2].

The pathophysiology of ITP involves the production of autoantibodies directed against platelet surface glycoproteins, mainly GPIIb/IIIa and GPIb/IX, leading to Fc receptor–mediated phagocytosis by splenic macrophages. In addition, cellular immune mechanisms play a crucial role, including cytotoxic T lymphocyte–mediated platelet destruction, reduced regulatory T-cell (Treg) activity, and dysregulated cytokine production [3].

ITP can be classified as primary or secondary to underlying conditions such as autoimmune diseases, immunodeficiencies, or infections. In pediatric populations, post-infectious ITP accounts for nearly two-thirds of cases [1]. Among infectious agents, cytomegalovirus (CMV) is of particular interest due to its hematopoietic tropism and its capacity to induce sustained immune dysregulation [4].

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We report a case of severe infantile ITP revealing a congenital CMV infection, illustrating the complex interactions between viral infection, immune dysfunction, and thrombopoiesis.

2. Case presentation

A three-month-old infant was referred for generalized petechial lesions involving the four limbs, trunk, face, and eyelids, incidentally discovered during a routine vaccination visit. The clinical presentation occurred in an afebrile context with preserved general condition.

Maternal history was notable for gestational diabetes managed with dietary measures, threatened preterm labor at 35 weeks of gestation, and isolated thrombocytopenia during the third trimester of pregnancy.

On physical examination, the infant was alert, hemodynamically and respiratorily stable. Cutaneous examination revealed diffuse non-blanching petechiae, small ecchymoses on the lower limbs, and petechiae on the soft palate, without gingival bleeding or hemorrhagic bullae (*Figure 1*). No hepatosplenomegaly or lymphadenopathy was observed.

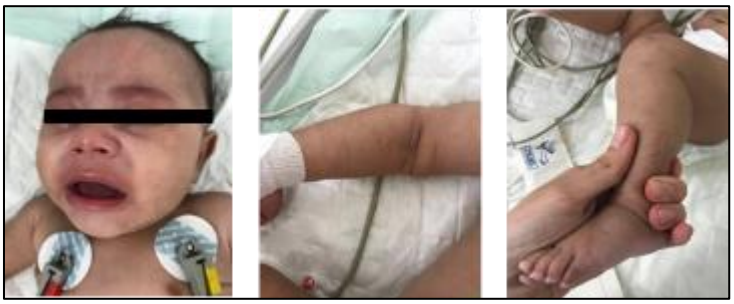

Figure 1 Petechiae on the four limbs, trunk, and eyelids

Laboratory investigations demonstrated severe thrombocytopenia (26×10^9/L), associated with normocytic normochromic non-regenerative anemia (hemoglobin 10 g/dL), mild leukocytosis, and relative lymphopenia affecting CD4+ T lymphocytes and NK cells. Peripheral blood smear showed no morphological abnormalities.

Bone marrow aspiration revealed a normocellular marrow with marked megakaryocytic hyperplasia at various stages of maturation, without dysplasia or blast excess, consistent with peripheral platelet destruction. Immunological screening including antinuclear antibodies and anti–double-stranded DNA antibodies was negative.

The diagnosis of ITP was established. Initial management included intravenous immunoglobulin (IVIg) at 1 g/kg/day for two consecutive days, systemic corticosteroids, and platelet transfusion, resulting in a transient normalization of platelet count. However, a severe relapse occurred one week later, suggesting secondary or refractory ITP.

Infectious workup revealed recent CMV infection, with positive CMV-specific IgM and IgG antibodies in both the infant and the mother. CMV infection was confirmed by real-time PCR, demonstrating active viral replication. Serologies for HIV, hepatitis C virus, Epstein–Barr virus, and parvovirus B19 were negative or indicative of past infection.

Antiviral therapy with ganciclovir (5 mg/kg every 12 hours for 15 days) was initiated, leading to sustained normalization of platelet count and complete suppression of CMV viral load.

3. Discussion

3.1. Viral infections and immune thrombocytopenia purpura

Viral infections represent a major cause of secondary ITP in children. Several immunological mechanisms have been implicated, including molecular mimicry between viral antigens and platelet glycoproteins, polyclonal B-cell activation, impaired Treg function, and excessive production of pro-inflammatory cytokines such as IL-6, IFN-γ, and TNF-α [3,5,6].

CMV is distinguished by its ability to establish lifelong persistence and to manipulate both innate and adaptive immune responses. CMV infection induces oligoclonal expansion of virus-specific CD8+ T cells, alters NK-cell receptor

expression, and modulates major histocompatibility complex (MHC) molecules, leading to prolonged immune imbalance [4,7].

3.2. Direct impact of CMV on thrombopoiesis

Unlike many other viruses, CMV exhibits direct tropism for hematopoietic progenitor cells and megakaryocytes. Experimental studies have shown that CMV infection of megakaryocytes inhibits their differentiation, induces apoptosis, and ultimately reduces platelet production [8].

From a biochemical perspective, CMV interferes with thrombopoietin (TPO) signaling and its receptor c-Mpl, disrupting key intracellular pathways involved in megakaryocyte maturation and platelet release [9]. These mechanisms provide a plausible explanation for the poor response to conventional ITP therapies, which primarily target peripheral platelet destruction rather than impaired production.

3.3. Diagnostic contribution of immunological and molecular tools

The diagnosis of CMV infection relies on a combined approach using serological and molecular assays. In infants, the presence of CMV-specific IgM antibodies together with detectable viral load by real-time PCR strongly supports active infection [10]. Quantitative PCR also allows monitoring of viral kinetics and assessment of therapeutic response.

Recent international recommendations emphasize the importance of systematically screening for CMV infection in cases of relapsing or refractory ITP, particularly in infants and immunologically immature children [11].

3.4. Therapeutic implications

Accumulating evidence indicates that antiviral therapy with ganciclovir or valganciclovir is essential for achieving sustained remission in CMV-associated ITP, whereas immunosuppressive therapy alone may be insufficient or ineffective [12]. Early identification and treatment of the underlying viral infection can prevent unnecessary therapeutic escalation and reduce morbidity.

4. Conclusion

Infantile ITP requires a thorough etiological investigation to identify potential secondary causes. Congenital CMV infection, often underdiagnosed, may present as severe and relapsing immune thrombocytopenia. Understanding the immunological and biochemical mechanisms involved is crucial for optimizing diagnosis and management. This case highlights the pivotal role of integrated clinical, immunological, and molecular approaches in improving outcomes for CMV-associated ITP.

Compliance with ethical standards

Acknowledgments

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Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of ethical approval

The study was conducted in accordance with ethical standards. Approval was obtained from the appropriate ethics committee.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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