Scientific Research Publishing

Case Reports in Clinical Medicine, 2025, 14(7), 341-346
https://www.scirp.org/journal/crcm
ISSN Online: 2325-7083
ISSN Print: 2325-7075

Temporary Corrective Effect of Influenza A (H1N1) in a Patient with Acute Immune Thrombocytopenia: A Case Report

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How to cite this paper: Tuncer, A.M. and Pekuz, O. (2025) Temporary Corrective Effect of Influenza A (H1N1) in a Patient with Acute Immune Thrombocytopenia: A Case Report. *Case Reports in Clinical Medicine*, **14**, 341-346.
https://doi.org/10.4236/crcm.2025.147045

Received: June 27, 2025
Accepted: July 13, 2025
Published: July 16, 2025

Abstract

The effects of viral infection on ITP (acute immune thrombocytopenia) were discussed in a patient who had been followed with a diagnosis of acute ITP and had not received any treatment for ITP during the period of influenza A (H1N1) infection. It is known that a decrease in platelet counts can be seen more frequently after viral infections. However, transient increases in viral infections, especially in ITP, are uncommon. A case report was published to report this rare condition.

Keywords

ITP, Immune Thrombocytopenia, Influenza A, H1N1, Thrombocytopenia, Viral Infection, Temporary Remission

1. Introduction

Immune thrombocytopenic purpura (ITP) is an autoimmune disease characterized by a low platelet count, purpura, and hemorrhagic episodes caused by antiplatelet autoantibodies. The diagnosis is typically made by excluding the known causes of thrombocytopenia. IgG autoantibodies sensitize the circulating platelets [1].

Most children have spontaneous remission within a few weeks or months, and splenectomy is rarely needed. However, young adults rarely have spontaneous remissions necessitating splenectomy within the first few months after diagnosis [2].

The International Working Group on ITP defines ITP according to the following clinical phases [2]. These are as follows [3].

Newly diagnosed ITP occurs in the first three months after diagnosis. Acute ITP is the most common form of ITP and is most commonly seen in children. Symp-

toms onset suddenly, usually last fewer than six months, and do not return. According to the National Organization for Rare Disorders, about 80 percent of kids who are newly diagnosed with ITP will have a case that resolves within 12 months (https://rarediseases.org/rare-diseases/immune-thrombocytopenia).

Persistent ITP is for 3 - 12 months.

Chronic ITP is for >12 months. More common in adults, but it can also affect adolescents. Symptoms can last anywhere from six months to several years. It can be recurring.

Refractory ITP is the failure of splenectomy.

Thrombocytopenia frequently follows viral infections, and viruses use various strategies to decrease circulating platelet levels.

Viruses can also directly interact with platelets and megakaryocytes and modulate their function. Furthermore, platelets can be activated by viral antigen—antibody complexes, and in response to some viruses, B-lymphocytes also generate antiplatelet antibodies. It is well known that platelets and their released products have been reported to directly and indirectly suppress infection and support virus persistence.

Transfusion may be withheld if there is no significant bleeding and the platelet count is >30,000/dL [3]. In a recent analysis, severe bleeding was seen in about 20% of children and about 10% of adults. Bleeding usually does not occur until the platelet count decreases to below 30,000/dL. Cutaneous and mucosal bleeding may occur below 20,000/dL, and life-threatening hemorrhage (e.g. intracranial) typically occurs with counts below 10,000/dL. It is preferred to give one apheresis unit; 4 to 6 pooled platelet units may also be given [3].

2. Case Report

A.A.C., a 2-year-7-month-old boy (date of birth October 11, 2022), applied to AnkaPedia Child Health Center with a complaint of bruises on his body for the last four weeks. It was learned from the patient's history that there was no family history of bleeding diathesis, that there was no prolonged umbilical bleeding at birth, that he was circumcised, and that no bleeding was observed during his circumcision.

Ecchymoses of 2 cm in diameter were found, especially on his legs. Physical examination was routine in the patient; there were no adenopathies or organomegaly. Petechiae were not found. His CBC was within normal limits except for thrombocytopenia (14,000/mm^3). His blood smear distribution was normal, but his platelets were single and large in size. No platelet morphological abnormality was detected. Other CBC findings were as follows: WBC: 12.700/mm^3, %50.1 pmnl, HB: 14 gr/dl, MCV: 77 fL.

The family was informed that the diagnosis was ITP, that bone marrow aspiration and analysis were required for definitive diagnosis, and that blood tests were required to rule out certain diseases. The disease was likely to resolve spontaneously, and information was provided on treatment options.

The family's questions about the drawbacks of monitoring without examination were answered. Since the patient's family preferred to be monitored without any intervention, the patient was called for weekly check-ups and monitored.

There was no profound clinical change in the patient's follow-up. No petechiae were observed. The platelet count follow-up was as follows; on admission day platelet number was 14,000/mm^3, on second day was 30,000, on day 15, platelets increased to 61,000, on day 45 he infected by İnfluenza A (Jan. 20, 2025), and platelet was dropped to 30,000, but one week after influenza A it was 135,000. On day 50, it was still higher than 100,000. On day 70, bruised and epistaxis were observed, and the platelet number was 22,000 at that time. There were no any signs related with infection. On day 150, platelets increased by more than 60,000 and the number was stabilized. Even on day 240, the platelet number was 140,000.

Since the platelet count was 61,000 in the second week of follow-up and there was no active bleeding in the clinic, it was decided to continue monitoring without treatment and further examination. Young, large platelets were seen in the peripheral smear during this period.

When he was seen with upper respiratory tract infection symptoms with fever in the sixth week of follow-up, his platelets had decreased to 31,000 in the CBC examination. However, there were no clinical signs of bleeding. He had no petechiae or ecchymosis. His smear showed single and large platelets. Influenza A (H1N1) was diagnosed with the tests performed.

Due to its effect on platelets, no antiviral agent was started, and he was monitored with antipyretics (paracetamol). In his control examination 2 days later, his platelet count was measured at 135,000/mm^3, despite his continued fever. His platelets were also sufficient in his smear. On the 50th day of ITP diagnosis and the fifth day of influenza diagnosis, when the infection was observed to decrease, his platelet count was measured as 130,000.

Approximately 1 month later, when he applied with bruising on his body, physical examination revealed bruising, especially on his legs and where he had hit his forehead. His platelet count was 22,000. He had no petechiae.

The patient was being monitored in the third and fourth months. His platelet count was about 30,000, and he had no clinical complaints.

At days 180 and 240, the control physical examination and growth were normal, and platelet counts were higher than 130,000/mm^3.

Our patient is still being monitored without symptoms.

3. Discussion and Conclusions

For typical cases of childhood ITP, 80% of patients will have platelet counts return to normal ≤ 2 months of presentation, with or without therapy, another 10% will recover normal platelet levels in the next few months, ~10% will go on to have chronic thrombocytopenia (>12 months' duration), ~25% of children will have a relapse after initial treatment., 10% have chronic ITP, 10% have recurrence but resolution ≤ 6 months, and 5% have ITP recurrences and remissions throughout

their lives [2].

It has been demonstrated that partial correction of thrombocytopenia in ITP can occur during viral [4]-[6] infections, bacterial [7] infections, and Mycoplasma [8] infections, including pneumonia [9]. The mechanisms underlying these changes are unknown. One plausible explanation may be a saturation of Fc receptor sites of splenic macrophages by antigen-antibody complexes following antigenic challenge [7]. Another valid explanation in the case of viral infection is transient immunosuppression secondary to decreased helper T cells and to suppression of IL-2 and y-interferon production [8].

Influenza virus infection is associated with a severity-dependent thrombocytopenia. Pediatric outpatients with confirmed influenza A or B infection showed slightly, though significantly, lower mean platelet counts compared to asymptomatic controls. Whole-blood transcriptome studies have found gene expression signatures in patients during H1N1 infection associated with a poor response to antiplatelet agents. Conversely, patients undergoing coronary catheterization who had a gene expression signature associated with viral infection were more likely to have a confirmed myocardial infarction compared to those who did not express this signature. Pathogenic H3N2 and H1N1 strains can infect pulmonary vascular endothelial cells, increasing platelet adhesion to infected and nearby uninfected cells through interaction between endothelial fibronectin and platelet integrins [10].

Various influenza A strains cause thrombocytopenia in experimentally infected ferrets, with highly pathogenic strains (H5N1) showing a more substantial decrease compared to moderate (H1N1) or mildly pathogenic (H3N2) strains. In addition, these viruses can directly infect platelets *in vivo* by binding sialic acids on glycans on their cell surface. EM imaging has demonstrated the ability of platelets to phagocytose influenza virus particles. This infection of platelets results in their activation, aggregation, and subsequent clearance from the circulation. Interestingly, desialylation of platelet glycans by viral neuraminidase is hypothesized to reduce the lifespan of affected platelets through increased hepatic clearance [11]. Influenza virus can also interact with platelets through TLR7, which leads to platelet-neutrophil aggregates forming through complement (C3) secreted by the platelets [10]. Immune complexes of antibodies against the influenza virus can also activate platelets by interacting with the Fc-*Y*IIA receptor on the platelet surface, leading to thrombocytopenia in a humanized mouse model. Combined with reported influenza vaccine-induced ITP, these findings indicate a link between influenza virus-specific adaptive immunity and thrombocytopenia [12].

It is known that a decrease in platelet counts can be seen more frequently due to increased phagocytic activity after viral infections. However, transient increases in viral infections, especially in ITP, are uncommon. This may be due to suppressing the phagocytic system [4].

In appropriate clinical cases, it may be appropriate to monitor childhood ITP without clinical urgency and laboratory critical levels without further invasive in-

terventions.

Clinical follow-up and platelet monitoring are important.

Some infections may provide temporary improvements in platelet counts.

Pediatric ITP is mostly a self-limiting disease. Except in some instances, such as severe clinical bleeding, severe thrombocytopenia (<20,000/mm^3), situations requiring mandatory interventions, a conservative approach will be less distressing for the patient and family [13].

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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