Medical and Health Journal

Volume 4, Issue 2, 2025, pp. 183-191

e-ISSN: 2807-3541

Open Access: https://jos.unsoed.ac.id/index.php/mhj

Immune Thrombocytopenic Purpura Induced by Varicella Zoster in an 8-Year-Old Girl: A Case Report

Wening Gelar Pratidina^{1*}, Ariadne Tiara Hapsari¹, Muhammad Zaenuri Syamsu Hidayat², Bunga³, Afra Bryges Tamia³

- ¹Department of Child Health, Faculty of Medicine, Universitas Jenderal Soedirman
- ²Department of Forensic, Faculty of Medicine, Universitas Jenderal Soedirman
- ³RSUD Ajibarang, Kabupaten Banyumas

ARTICLE INFO

Article history:

Received February 4, 2025 Revised February 12, 2025 Accepted February 16, 2025 Available online February 17, 2025

Kata Kunci:

Varicella Zoster, Purpura Trombositopenik Imun, Trombositopenia, Anak

Keywords:

Varicella Zoster, Immune Thrombocytopenic Purpura, Thrombocytopenia, Child



This is an open access article under the CC BY-SA

Copyright © 2025 by Author. Published by

ABSTRAK

Varicella Zoster Virus (VZV) dapat memicu berbagai komplikasi, salah satunya adalah Immune Thrombocytopenic Purpura (ITP), meskipun kejadian ini jarang terjadi. ITP adalah kelainan perdarahan yang disebabkan oleh penghancuran trombosit yang berlebihan akibat autoantibodi. Laporan kasus ini mengangkat seorang anak perempuan berusia 8 tahun yang mengalami varicella disertai trombositopenia berat. Pada awalnya, pasien mengalami gejala prodromal berupa demam, diikuti dengan munculnya lesi kulit vesikuler yang menyebar secara sentrifugal. Pemeriksaan laboratorium menunjukkan trombositopenia yang mendukung diagnosis ITP. Penatalaksanaan yang diberikan mencakup terapi antivirus asiklovir, transfusi trombosit, dan terapi kortikosteroid untuk mengurangi peradangan. Meskipun ITP yang dipicu oleh varicella termasuk jarang, penanganan yang cepat dan tepat sangat diperlukan untuk mencegah komplikasi yang dapat mengancam jiwa.

ABSTRACT

Varicella Zoster Virus (VZV) can trigger various complications, one of which is Immune Thrombocytopenic Purpura (ITP), although this occurrence is rare. ITP is a bleeding disorder caused by excessive platelet destruction due to autoantibodies. This case report presents an

8-year-old female child who developed varicella accompanied by severe thrombocytopenia. Initially, the patient experienced prodromal symptoms including fever, followed by the appearance of vesicular skin lesions that spread centrifugally. Laboratory tests revealed thrombocytopenia, supporting the diagnosis of ITP. The management included antiviral therapy with acyclovir, platelet transfusion, and corticosteroid therapy to reduce inflammation. Although ITP triggered by varicella is rare, prompt and appropriate management is essential to prevent life-threatening complications.

1. INTRODUCTION

Immune thrombocytopenic purpura (ITP) is a bleeding disorder caused by excessive destruction of platelets, characterized by thrombocytopenia (platelet count < 100,000/mm³) with clinical symptoms generally presenting as petechiae, hematomas, purpura, and mucosal membrane bleeding. These symptoms usually appear 1-4 weeks after viral infections or immunizations (Provan et al., 2010; Neunert et al., 2011). ITP can be classified into two types: primary ITP, which occurs without an underlying disease, and secondary ITP, which is associated with other conditions that lead to platelet reduction, including drug-induced reactions (Provan et al., 2010; Kumar et al., 2014).

One of the causes of secondary ITP triggered by infection is Varicella Zoster, which produces specific antibodies against the virus that bind to platelets. Although rare, varicella associated with thrombocytopenia can be life-threatening due to the associated deficiency of

*Corresponding author

protein S and the formation of microvascular platelets, which may cause serious complications (Hossen & Anwar, 2019; Shapiro & Kaplan, 2014).

2. METHOD

The method used in this article is a case report. Below is the case report that I present.

CASE REPORT

Anamnesis

An 8-year-old female patient was brought to the ER of RSUD Ajibarang by her family, presenting with a fever for the past 4 days before hospitalization. The fever was described as fluctuating. The patient also reported the appearance of small bumps (plenting) on her body and face, initially few in number but gradually increasing. The bumps appeared 2 days after the fever began. They initially appeared on the body and then spread to the face and entire body. The bumps started as red, and after 2 days, they filled with fluid and turned black, eventually rupturing and releasing blood. The patient also had black spots on her tongue, a red throat, and bleeding gums. She had difficulty eating and drinking due to pain. Both eyes had red spots, but the patient denied any visual disturbances.

The patient's last urine output was in the morning before coming to the hospital, and it was plentiful. When heading to the hospital, the patient fell off a motorcycle in a prone position, but there was no head injury. The patient complained of nausea but did not vomit. There were no complaints of changes in bowel movements (stools), and no black or bloody stools were reported. Abdominal pain and other signs of bleeding were also denied.

The patient denied a previous history of fever and had no other significant medical history. Prior to visiting the hospital, the patient had taken medications bought from a pharmacy, including cetirizine syrup 1x1 dose, acyclovir ointment 3x1 dose, and amoxicillin syrup 3x2 doses. Family history revealed that the patient's sibling had experienced fever accompanied by the appearance of bumps without fluid at the end of December, while another sibling had a fever accompanied by clear fluid-filled bumps. The patient did not have a history of traveling outside the city, and immunizations were complete according to the patient's age.

Physical Examination

On physical examination, the patient's general condition appeared well. The patient was conscious, with a Glasgow Coma Scale (GCS) of 15 (E4V5M6). Vital signs showed a pulse rate of 90 beats per minute, strong and palpable, respiratory rate of 20 breaths per minute, axillary temperature of 36.6°C, and SpO2 of 98% on room air. The patient's weight was 21.5 kg, height was 128 cm, and Body Mass Index (BMI) was 13.12, indicating good nutritional status.

Head examination revealed conjunctival bleeding. The sclera was not icteric and there was no anemia in the conjunctiva. The nose showed no secretion or bleeding. The mouth appeared dry, with no cyanosis in the lips, but there were black spots on the tongue. The pharynx appeared hyperemic with black spots on the palate. Neck examination showed no lymphadenopathy. On thoracic examination, vesicular breath sounds were heard in both lung fields with no rales or wheezing, and chest wall movement was symmetric in both lung fields. Cardiac examination revealed regular S1S2 sounds with no murmurs or gallops.

Abdominal examination showed a distended abdomen with no tenderness, normal bowel sounds, and tympany present. No hepatomegaly or splenomegaly was noted. Both upper and lower extremities felt warm, with capillary refill time (CRT) less than 2 seconds and no edema observed. Dermatological examination revealed black vesicles filled with blood, with an erythematous base, distributed on the face, neck, chest, abdomen, and both hands and feet. In addition to vesicles, there were also black crusts and reddish-blue macules scattered across almost the entire body.

Clinical Photos

Below is the clinical photo of the patient upon initial presentation at the ER:





Figure 1. is the clinical photo of the patient upon initial presentation at the ER

Supporting Examinations

The initial supporting examinations performed in the ER included laboratory tests such as a complete blood count, blood glucose level (GDS), and C-reactive protein (CRP). A chest X-ray (AP view) and an abdominal X-ray (2 positions) were also conducted. The laboratory results were as follows:

Table 1. The result of laboratory tests

Hemoglobin	12.8	g/dL	10.8 – 15.6
Leukosit	11.08	10^3/uL	4.8 – 10.8
Hematokrit	37.4	%	33.0 – 45.0
Eritosit	5.04	10^6/uL	3.80 - 5.80
Trombosit	5	10^3/uL	150 – 450
MCV	74.2	fL	79.0 – 99.0
MCH	25.4	Pg	27.0 - 31.0
MCHC	34.2	g/dL	33.0 – 37.0
RDW	12.0	%	11.5 – 14.5
MPV	-	fL	7.2 – 11.1
Basofil	0.4	%	0.0 - 1.0
Eosinofil	0	%	2.0 - 4.0
Batang	0.8	%	2.00 – 5.00
Segmen	70.2	%	40.0 – 70.0
Limfosit	25	%	25.0 – 40.0

Monosit	4	%	2.0 - 8.0	
1/10110510	•	, •	2.0 0.0	
Clades as Carreleter	125	/JT	z 100	
Glukosa Sewaktu	135	mg/dL	< 100	
CRP Kuantitatif	3.3	mg/L	< 5	

The results of the thoracic examination were as follows:



Figure 2. The results of the thoracic examination
Impression:
Normal shape and position of the heart
Right-sided pneumonia, differential diagnosis: pulmonary tuberculosis
Duplex hilum lymphadenopathy

Working Diagnosis

Based on the results of anamnesis, physical examination, and supporting tests, a working diagnosis of suspected Varicella with thrombocytopenia due to Dengue Fever, differential diagnosis: ITP (Immune Thrombocytopenic Purpura), was made.

Treatment

The treatment provided to the patient in the ER included an RL loading infusion of 200 cc, followed by an RL infusion at 60 ml/hour. Platelet transfusion (TC) was given twice, 100 ml each, with a 12-hour interval between transfusions, and each 100 ml was infused over 30 minutes. The patient was also administered Dexamethasone injection 2 mg every 12 hours, Ceftriaxone injection 600 mg every 24 hours, Ranitidine injection 25 mg every 12 hours, Tranexamic Acid injection 250 mg every 8 hours, Vitamin K injection 6 mg every 24 hours, and Paracetamol infusion 250 mg if the patient's temperature exceeded 38.5°C. Paracetamol syrup was given 3 times daily, 250 mg each, as well as Aciclovir 200 mg every 8 hours, and Lyctacur syrup 1 tablespoon once daily. Additionally, the patient was scheduled for MDT examination and post-transfusion TC evaluation. However, the MDT examination could not be performed as the patient had already received platelet transfusion before blood samples could be taken for MDT. Initially, the patient was scheduled to be admitted to the isolation ICU due to concerns about intracranial bleeding and possible deterioration of the condition. However, since the ICU was full, the patient was treated in a regular pediatric ward with close monitoring.

Follow Up

On the day of care (day 0), in the afternoon around 17:45 WIB, the patient complained of a nosebleed and brownish vomiting. The patient denied having black stool or blood in the stool, and also denied red-colored urine. No other signs of bleeding were observed. On physical examination, the patient was in a conscious state, with blood pressure 99/54 mmHg, pulse rate 117 beats per minute, and respiratory rate 22 breaths per minute. On abdominal examination, a hematoma was found in the right iliac region, with defense around the area of injury and tenderness (+) in that area. The abdominal circumference was 53 cm. Upon initial physical examination when the patient entered the ER, no hematoma was observed in the abdominal region. However, by the afternoon, the hematoma became more evident and widespread. In addition to the hematoma in the abdominal region, hematomas were also observed in the right patella region and right lower leg (cruris dextra). Attached are the patient's clinical photos:On the day of care (day 0), in the afternoon around 17:45 WIB, the patient complained of a nosebleed and brownish vomiting. The patient denied having black stool or blood in the stool, and also denied red-colored urine. No other signs of bleeding were observed. On physical examination, the patient was in a conscious state, with blood pressure 99/54 mmHg, pulse rate 117 beats per minute, and respiratory rate 22 breaths per minute. On abdominal examination, a hematoma was found in the right iliac region, with defense around the area of injury and tenderness (+) in that area. The abdominal circumference was 53 cm. Upon initial physical examination when the patient entered the ER, no hematoma was observed in the abdominal region. However, by the afternoon, the hematoma became more evident and widespread. In addition to the hematoma in the abdominal region, hematomas were also observed in the right patella region and right lower leg (cruris dextra). Attached are the patient's clinical photos:



Figure 3. Patient's clinical photos result

Due to ongoing signs of bleeding, a serial DL examination was performed (2-3 hours after the first platelet transfusion), followed by close monitoring of the abdominal circumference and the administration of an additional 100 cc platelet transfusion. At 02:33 WIB, an evaluation DL examination was performed, and the following results were obtained:

Table 2. The result of laboratory tests

Hemoglobin	5.8	g/dL	10.8 - 15.6	
Leukosit	13.20	10^3/uL	4.8 - 10.8	
Hematokrit	16.4	%	33.0 - 45.0	

Eritosit	2.26	10^6/uL	3.80 - 5.80
Trombosit	3	10^3/uL	150 - 450
MCV	72.6	fL	79.0 – 99.0
МСН	25.7	Pg	27.0 - 31.0
МСНС	35.4	g/dL	33.0 - 37.0
RDW	12.0	%	11.5 – 14.5
MPV	-	fL	7.2 - 11.1
Basofil	0.1	%	0.0 - 1.0
Eosinofil	0	%	2.0 – 4.0
Batang	0.8	%	2.00 - 5.00
Segmen	68.7	%	40.0 – 70.0
Limfosit	25	%	25.0 - 40.0
Monosit	6	%	2.0 - 8.0

From the data, it was found that the levels of Hemoglobin and Platelets decreased. The Hemoglobin level, which was initially 12.8, dropped to 5.8, and the initial platelet count of 5000/uL decreased to 3000/uL. On the other hand, the leukocyte count increased. The initial leukocyte count of the patient was 11.08×10^{3} /uL, and upon evaluation, it increased to 13.02×10^{4} 10^3/uL.

Due to the significant decline in Hemoglobin and Platelets, additional therapy was administered in the form of RL infusion at 5 cc/kg body weight over 30 minutes, repeated twice, totaling 10 cc/kg body weight. A PRC transfusion of 250 cc was also given, and the patient was referred to a more complete FKTL with a pediatric hematology-oncology specialist. While awaiting the referral, close observation of the patient's condition was conducted.

On the second day of care, while awaiting the referral, at 09:11, a second DL evaluation was performed. The results of the second DL evaluation were as follows:

Table 3. Table 2. The result of laboratory tests

Hemoglobin	8.0	g/dL	10.8 - 15.6
Leukosit	22.65	10^3/uL	4.8 – 10.8
Hematokrit	22.7	%	33.0 – 45.0
Eritosit	2.99	10^6/uL	3.80 - 5.80
Trombosit	2	10^3/uL	150 – 450
MCV	75.9	fL	79.0 – 99.0
MCH	26.8	Pg	27.0 - 31.0
MCHC	35.2	g/dL	33.0 – 37.0
RDW	12.9	%	11.5 – 14.5
MPV	-	fL	7.2 – 11.1

Basofil	0.0	%	0.0 – 1.0
Eosinofil	1	%	2.0 – 4.0
Batang	0.4	%	2.00 – 5.00
Segmen	86.2	%	40.0 – 70.0
Limfosit	11	%	25.0 – 40.0
Monosit	6	%	2.0 – 8.0

The post-transfusion blood evaluation results after 250 cc PRC showed an increase in hemoglobin to 8.0, but the platelet count remained low at 2×10^3 L. On the second day of care, at 12:00 WIB, the patient successfully received a referral to Rumah Sakit Margono Soekarjo. By the time the referral was made, the patient no longer experienced nosebleeds, brown stool, or any other signs of bleeding. The patient only complained of weakness. The patient was conscious, with blood pressure 98/52 mmHg, pulse rate 100 beats per minute, temperature 37°C, respiratory rate 20 breaths per minute, and abdominal circumference remained at 53 cm.

The patient was treated at Rumah Sakit Margono Soekarjo for 5 days. Based on the patient's medical summary obtained from a home visit, the final diagnosis was as follows: Hemorrhagic Varicella, Sepsis, Severe Thrombocytopenia, and ITP. During the hospitalization, the patient was treated in the isolation room and received therapy including Inf Kaen3A 15 tpm, ceftriaxone injection 750 mg every 12 hours, tranexamic acid injection 2x 250 mg, metiprednisolone injection 2x 20 mg, and ranitidine injection 2x 20 mg. The patient also received a 200 ml PRC transfusion and 3 units of platelet transfusion. The oral medications administered during treatment were acyclovir 4x 200 mg and Elkana syrup 1x 5 ml. The topical medications were Fuson 5 gr cream, applied twice daily, and sterile eye drops 2x 1 drop.

Upon discharge, the patient was prescribed oral acyclovir 4x 200 mg, prednisone 3x 5 mg, Elkana syrup 1x 5 ml, Lyters drops 2x 1 drop ODS, and Fuson 5 gr cream, applied twice thinly. Blood culture was performed, and the results showed no bacterial growth. The patient's condition, when the author conducted a home visit, had improved. The patient no longer had complaints such as fever or signs of bleeding. The skin lesions were fading, leaving a bluish scar. The hematomas in the abdomen and legs were also beginning to subside.

The patient also had a follow-up visit to the pediatric hematology-oncology outpatient clinic and received home therapy. According to the family, the patient was no longer scheduled for routine follow-ups as the condition had improved. Attached are the clinical photos of the patient after treatment at Rumah Sakit Margono Soekarjo:



Figure 4. Patient's clinical photos result

3. RESULT AND DISCUSSION

Varicella is an acute infection caused by the Varicella Zoster virus, which is self-limiting and affects the skin and mucous membranes. It is characterized by constitutional symptoms such as fever and malaise, as well as polymorphic skin lesions (vesicles that spread, generally located in the central parts of the body). Varicella is highly contagious and can affect all age groups, including neonates. However, around ninety percent of cases occur in children under the age of 10, with the highest incidence between 5 and 9 years old (Provan et al., 2010).

immunocompetent children, varicella complications are immunocompromised patients, the manifestations of the disease can be more severe and progressive, and may be accompanied by purpura or large and deep hemorrhagic lesions (Shapiro & Kaplan, 2014). The varicella-zoster virus (VZV) is part of the herpesviridae family, a DNA alpha herpesvirus. The virus primarily enters the body through direct contact with skin lesions or through respiratory droplets. The virus replicates in local lymph nodes for 2-4 days, followed by primary viremia occurring 4-6 days after infection (Kumar et al., 2014). Secondary viremia occurs, causing the virus to spread to the skin, and the characteristic vesicular lesions of varicella appear about 14-16 days after contact (Hossen & Anwar, 2019).

In the anamnesis, the main complaint found was the presence of skin lesions, followed by prodromal symptoms such as fever, fatigue, irritability, sore throat, cough, loss of appetite, and headache. These prodromal symptoms typically last 2-3 days and are followed by skin lesions in the form of fluid-filled blisters that spread centrifugally, starting from the central part of the body and then spreading to the neck and extremities (Provan et al., 2010). This is consistent with the patient's complaints, which began with fever, followed by the appearance of blisters spreading from the body to the face and throughout the entire body.

Physical examination revealed lesions in the form of erythematous papules, followed by vesicles or pustules on an erythematous base, which then enlarged, formed a dell, and eventually dried to form blackish crusts. All lesions appeared asynchronously, initially on the face, head, or upper body, and gradually spread throughout the body, although lesions on the extremities were less frequently found. Mucous membranes of the eyes, mouth, pharynx, and larynx are often involved, with vesicles that quickly rupture and form shallow ulcers resembling canker sores, causing difficulty for the child to eat and swallow (Neunert et al., 2011).

Supporting tests performed to confirm the diagnosis of varicella included the Tzank test, but it is not specific for varicella. Virus culture from vesicular fluid is often positive in the first 3 days, but it is difficult and expensive. PCR can be used for severe or atypical cases (Shapiro & Kaplan, 2014). In this patient, only laboratory tests were performed, including a complete blood count, GDS, and CRP, as well as radiological examinations, including chest X-rays and abdominal X-rays to rule out other possible diagnoses.

The thrombocytopenia found in the complete blood count $(5x10^3/uL)$ pointed toward a differential diagnosis of Immune Thrombocytopenic Purpura (ITP) and Dengue Fever (Kumar et al., 2014). Varicella can trigger the onset of ITP, which is the most common bleeding disorder in children. ITP in children is often benign and may resolve spontaneously, especially in children with a history of previous viral infections. Other secondary causes include bacterial infections, malignancies, and autoimmune diseases (Provan et al., 2010). ITP has also been reported as a response to Varicella Zoster Virus (VZV) vaccination.

In some cases, thrombocytopenia caused by increased platelet destruction in viral infections can be triggered by immune mechanisms, where there is an increase in the number of IgG platelet-adherent antibodies bound to platelets. In this case, only 2 days elapsed between the infection and the onset of purpura, which is faster than usual (Kumar et al., 2014).

Treatment and Management

Treatment for varicella in children includes the administration of oral acyclovir 4x20 mg/kg body weight/day for 5-7 days to speed up recovery (Provan et al., 2010). In addition, symptomatic therapy with antipyretic analgesics and sedative antihistamines, such as hydroxyzine, can be used to alleviate itching. In this case, platelet and PRC transfusions were

necessary to manage spontaneous bleeding resulting from severe thrombocytopenia. Transfusions can control bleeding temporarily, as the transfused platelets have a short lifespan (Shapiro & Kaplan, 2014).

The administration of corticosteroids, such as Dexamethasone, was also considered to reduce inflammation and increase platelet count with an adjusted dosage, although this therapy is still debated. In some cases, intravenous immunoglobulin therapy can be given if thrombocytopenia persists for 4-6 months (Neunert et al., 2011).

4. CONCLUSION

ITP triggered by varicella is a rare occurrence. Although it is considered a self-limiting disease, prompt and appropriate management is essential to reduce the risk of complications, such as bleeding that can be life-threatening

5. ACKNOWLEDGE

I would like to thank RSUD Ajibarang for granting permission to carry out the writing of this case report, as well as to the family for granting permission for the writing of this case report, and to all parties who have assisted in the preparation of this case report.

6. REFERENCES

- Kumar, V., Abbas, A. K., Aster, J. C. (2014). *Robbins and Cotran Pathologic Basis of Disease* (9th ed.). Elsevier.
- Neunert, C., Lim, W., Crowther, M., Cohen, A., Solberg, L. I., & Kessler, C. M. (2011). The American Society of Hematology 2011 evidence-based practice guideline for immune thrombocytopenia. Blood, 117(16), 4190–4207. https://doi.org/10.1182/blood-2010-08-302984
- Provan, D., Stasi, R., Newland, A., Blanchette, V. S., & Bolton-Maggs, P. (2010). *International consensus report on the investigation and management of idiopathic thrombocytopenic purpura*. British Journal of Haematology, 149(3), 287–303. https://doi.org/10.1111/j.1365-2141.2010.08137.x
- Shapiro, M., & Kaplan, M. (2014). *Varicella Zoster Virus-induced thrombocytopenia: A review of the literature*. International Journal of Infectious Diseases, 85, 18-22. https://doi.org/10.1016/j.ijid.2019.06.020
- Utami, Fista, et al. Imunologi Dasar: Memahami Sistem Pertahanan Tubuh Manusia. Penerbit Mafy Media Literasi Indonesia, 2024, doi:10.1112/kakinaan1112.