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A CHRONIC *IMMUNE THROMBOCYTOPENIA PURPURA (ITP)* PATIENT WITH MULTIPLE ABSCESSES

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ABSTRACT

Chronic Immune Thrombocytopenia Purpura (ITP) is a medical condition characterized by a decrease in the number of platelets in the blood due to the body's immune system attacking the platelets. Chronic ITP sufferers can experience various complications, one of which is multiple abscesses. Multiple abscesses are a medical condition characterized by a collection of pus in the body tissue which can occur in various organs. The reported case is a patient with chronic ITP with multiple abscesses. Sufferers experience symptoms of fever, pain in the part of the body affected by the abscess, and a decrease in the number of platelets in the blood. The diagnosis is made through physical examination, blood tests and radiological examinations. Treatment given to sufferers includes giving antibiotics to treat infections that occur, giving drugs to increase the number of platelets in the blood, and drainage procedures to remove pus from the abscess. Sufferers also need to undergo long-term treatment to control their chronic ITP condition. This case shows that chronic ITP sufferers can experience complications in the form of multiple abscesses which require appropriate and immediate treatment. Prevention of infection and control of the chronic condition of ITP are very important in reducing the risk of complications in sufferers .

Keywords: Immune Thrombocytopenia Purpura, Multiple Abscesses, Infections

1. INTRODUCTION

Immune thrombocytopenia Purpura (ITP) is an autoimmune disease characterized by low platelet counts and an increased risk of bleeding. [1] Low platelet counts can be caused by the process of platelet destruction mediated by the antibody system along with impaired platelet production. [1] ITP is a common hematological disease and can affect patients of all ages, genders and races, previously known as "idiopathic thrombocytopenic purpura" but now as "Immune thrombocytopenia", this terminology is recommended by the International Working Group (IWG) because of the pathogenesis of ITP which involves the immune system, and not always patients who experience ITP have Purpura. [2] ITP is not a single disorder, but a syndrome in which thrombocytopenia can be primary or secondary based on an infectious or immune process. [3,4]

The overall prevalence of ITP in adults and children can be compared and is not significantly different through meta-analysis studies [5] . The incidence was found to be slightly higher in women aged 45-49 years. Studies in Scandinavia identified between 2.25 and 2.68 per 100,000 individuals/year, and found that women were more affected [6] . Other studies also stated that the prevalence of ITP in adults ranged from 4.0 to 23.6/100,000 people/year. [6]

Corticosteroids are the first line treatment for ITP, and are immunosuppressive and the concept is to inhibit the formation of platelet autoantibodies. Corticosteroids also have many side effects, especially long-term use, one example of which is infection [8] . ITP sufferers tend to experience bleeding and infection 5 to 8 times more than people in general, bleeding and infection are the 2

highest causes of mortality in ITP (Nørgaard et al, 2011). Although there is not enough data on the mortality of ITP sufferers, a study at Leiden University examined 152 ITP sufferers, in the study it was found that 7 people had infections, 2 of them died due to gram-negative sepsis, the clinical manifestations varied between the 7, reported pneumonia, sepsis , and abscesses in the abdominal cavity found in the study. [9]

2. METHOD

A female patient, Ms. H, 23 years old, Javanese, unmarried, came to the hemato and medical oncology clinic on February 23 2022 with complaints of excessive menstruation for 15 days. The patient has been diagnosed with Immune Thrombocytopenia (ITP) since 6 years ago (2016) and regularly takes medication.

The patient came with complaints of excessive menstruation for 15 days (the last time the patient experienced complaints like this was last year), the patient felt weak and tired easily recently. The patient also felt that his legs had been swollen for the past 4 days. Sometimes patients feel short of breath, but there is no cough or fever. The patient sleeps with 1 pillow, the patient never wakes up because of the shortness of breath. Patients also complain that their skin often experiences rashes. In this 1 year, the patient's weight (BB) has increased by 8 kg, previously the patient's weight was around 88 kg, but currently the patient's weight is 92 kg. There are no complaints about urinating and defecating. The patient has never experienced nosebleeds or bleeding gums.

Initially the patient experienced excessive menstruation in 2015, then the patient was checked at the Internal Medicine Clinic at Mojokerto Hospital. Then in 2016 the patient was first diagnosed with Immune Thrombocytopenia (ITP). The patient was taken for control at S wasta Hospital in Surabaya and did not recover. At that hospital the patient received Rebozet 2x1 therapy at that time but the patient did not take the medication regularly, and because the therapy was not successful, finally in 2018 the patient was referred to Dr. Soetomo Surabaya. At the regional hospital, patients have been given the drug MP (Methylprednisolone) 3x16 mg since 2018, according to the patient's statement, the dose sometimes goes up and down, but mostly it is 3x16 mg. The patient also takes the drug Sandimmun, rebozet from Poli in 2018, but the patient never uses it regularly and has stopped taking it.

In 2020 the patient was tested for Lupus with negative results. The patient also had a history of hospitalization in May 2021 with complaints of heavy and long menstruation.

Denied history of diabetes mellitus, hypertension, heart disease, liver disease or autoimmune disorders. The patient is a student at a private university in Surabaya.

On physical examination, it was found that the general condition was adequate, conscious composment, Glasgow Coma Scale (GCS) E4V5M6, blood pressure 150/90 mmHg, pulse rate 150 times per minute, respiratory rate 28 times per minute, peripheral saturation 97% free air, axillary temperature 36 .8°C. Height (TB) 155 cm and weight (BB) 90 kg.

Examination of the head and neck revealed anemia, and there was no jaundice, cyanosis or dyspnea. The patient's face appears round or what is usually called a moon face and both cheeks appear slightly reddish. In the patient's nape area there is a collection of fat that looks like a hump or what is usually called Buffalo Hump.

Thoracic examination revealed symmetrical movement, no retraction. On cardiac examination, a single S1 and S2 were found, there were no heart murmurs, gallop rhythms, or pericardial rubbing

sounds. On examination of the lungs, vesicular breath sounds were found, there were no rales or wheezing in both lung fields.

Abdominal examination revealed normal bowel sounds. The stomach appears slightly distended, there is no dilation of collateral veins or medusal heads, there is shifting dullness and there is minimal ascites. There is no tenderness in the abdomen, *the liver* and spleen are difficult to evaluate. In the patient's lower abdominal area it seems that there is fat hanging down or what is usually called a Pendulous Abdomen. In the patient's stomach area there are many striae which are reddish purple in color.

Examination of the extremities revealed warm, dry and pale acral. Edema of both legs and pitting edema on pressure, there is purpura on both legs. Laboratory examination Hb 5.8 g/dL, HCT 20.7%, MCV 90.4 fl, MCH 25.3 pg, MCHC 28.3 g/dL, leukocytes 12,390/ul, neutrophils 90%, lymphocytes 8.3% , platelets 18,000/ul, SGOT 82 u/L, SGPT 22 u/L, GDA 110 mg/dL, BUN 7 mg/dL, serum creatinine 0.29 mg/dL, Na 136 mmol/L, K 4.2 mmol /L, Cl 101 mmol/L, Ca 8.05 mmol/L, albumin 3.43 g/dL. HBsAg is non-reactive. Thoracic X-Ray Examination: Cast and Pulmo have no abnormalities. Lumbar MRI examination (2019): Biconcave vertebrae form a codfish image at Vth10-VL5 which can be visualized in blood disorders (history of ITP), incidental findings, left kidney hypoplasia.

The patient's working diagnosis is Chronic *Immune Thrombocytopenia Purpura (ITP)* accompanied by Cushing's Syndrome. Diagnosis plan, complete blood count after transfusion, serum cortisol (morning), kidney function, liver function, serum electrolytes, and fasting blood sugar. Initial management includes TKTP diet 1900 kcal/day, 0.9% NaCl infusion 500 ml/24 hours, PRC transfusion 2 kolf/24 hours up to Hb ³10 g/dL, TC transfusion 10 kolf/24 hours up to platelets ³ 20,000/ uL, MP 3x16 mg, imuran 2x50mg, omeprazole capsule 1x20 mg orally, folic acid tablet 3x1 mg orally, spironolactone tablet 1x 25 mg orally.

On the second treatment day (23/2/2022), the patient still felt weak, bleeding was still found through the menses. The patient also received a PRC transfusion of 1 kolf/24 hours, and a TC transfusion of 5 kolf/24 hours. There was no change in other therapies. The patient was still taking MP 3x16 tabs, Omeprazole 1x20mg tab, As Folate 3x1 .

On the third treatment day (24/2/2022), the patient felt that the bleeding was still there but reduced, compos mentis consciousness, GCS 456, with blood pressure 146/87, pulse 120x/min, temperature: 36.7, RR 22x/min, SpO2 98% Free Air, the patient still received PRC transfusion 1 kolf/24 hours, and TC transfusion 5 kolf/24 hours, with the MP tablet changed to MP injection 3x125mg. The patient was referred to the Endocrine division. The sixth day of treatment (27/2/2022), the patient had no complaints, GCS 456, with blood pressure 131/72, N> 98x/min, temperature: 36.7, RR 22x/min, SpO2 99% Free Air , and the results of the post-transfusion complete blood test were Hb 9.4 g/dL, HCT 28.8%, MCV 89.8 fl, MCH 26.2 pg, leukocytes 9,630/ul, neutrophils 88.9%, lymphocytes 3.7 %, platelets 50,000/ul. Results of anti-HCV examination non-reactive, HIV 3 methods non-reactive, ANA test negative, C3 136 mg/dL, C4 15.4 mg/dL, Cortisol 16.41 ng/dL, Ferritin 271.6 ng/dL, ACTH <10, Coomb test negative, Indirect comb test negative, IPF 22.10%, IPF 4,000/uL. Complete feces: no occult blood, amoeba, larvae, cysts, mucus, eggs, negative worms were found. Leukocytes and erythrocytes 0-1/lp. The patient went home on 28/2/2022 with 3x16 mg methylprednisolone tablet therapy, 1x50mg Rebozet, 3x1 As Folate, 1x25mg spironolactone, and planned control at the

HOM polyclinic for tapering off the methylprednisolone tablet dose.

Two weeks after KRS, the patient returned to the emergency room with complaints of pain in the wound on the skin in the buttock area, it felt hot. The patient did not go to the clinic because he felt he had no symptoms. Complaints about this wound were initially blistered and then became sores starting 4 days ago. Previously there was a lump in the waist area but it has not been painful since 4 days ago. Patients also complain that their skin often experiences rashes. Body feels weak (+). The patient also complained of slight shortness of breath for the last 4 days. On physical examination, it was found that his general condition was fair, conscious, Glasgow Coma Scale (GCS) E4V5M6, blood pressure 150/90 mmHg, pulse rate 110 times per minute, respiratory rate 24 times per minute, peripheral saturation 95% via Nasal Cannula 4 lpm, axillary temperature 36.6°, laboratory examination Hb 9.5 g/dL, HCT 32.5%, MCV 87.1 fl, leukocytes 18,700/ul, platelets 83,000/ul, SGOT 38 u/L, SGPT 42 u/L , GDA 391 mg/dL, BUN 10 mg/dL, serum creatinine 0.66 mg/dL, Na 132 mmol/L, K 4.0 mmol/L, Cl 92 mmol/L. Blood Gas Analysis Results pH 7.48; pO2 59; pCO2 33; HCo3 24.6; BE 1.1; SpO2 92; P/F ratio 281. Thoracic X-Ray examination: Pulmo has no abnormalities, aortosclerosis.

Pelvic Photo Examination: no abnormalities. The patient's working diagnosis is Sepsis + Gluteus region abscess + multiple decubitus ulcers + secondary infection + hyperglycemia ec stress hyperglycemia dd steroid induced + hypovolemic hypotonic hyponatremia + HT stage 1 INASH 2019 + ITP on treatment. Blood culture diagnosis plan, sputum and wound base, random blood sugar evaluation, fasting blood sugar, 2 hour blood sugar pp, Hba1c, Blood Gas Analysis Evaluation, complete blood count, kidney function, liver function, serum electrolytes, albumin, and procalcitonin. Initial management includes O2 via mask 6-8 lpm, TKTP diet 1900 kcal/day, 0.9% NaCl infusion 1500 ml/24 hours, 2x1g ceftriaxone injection, 3x500mg metronidazole injection, 3x1000mg metamizole injection, 3x500mg paracetamol tablets orally, MP 3x16 mg. The patient was also consulted for BTKV and anesthesia, with the suggestion of debridement in the operating room, but this was not done due to fear of worsening the condition and the consul's answer from Anesthesia was to consider intensive care, but at this time the patient refused to take action.

The third treatment day (11/3/2022) the patient felt hot since early morning, GCS 456, with blood pressure 127/80, N 145x/min, temperature: 38.3, RR 26x/min, SpO2 99% with mask on 6-8 lpm, Patient received novorapid therapy 3x4 iu SC The fifth day of treatment (13/3/2022) the patient felt increasingly tight and had pain in the stomach, but the patient refused the ventilator, compos mentis consciousness, GCS 456, with blood pressure 131/82, N 128x/min, temperature: 37.1, RR 26x/min, SpO2 97% with mask 6-8 lpm, complete blood test results Hb 10 g/dL, HCT 33.6%, MCV 87.7 fl, MCH 26.1 pg, leukocytes 13,747/ul, platelets 77,000/ul, Albumin 2.72g/dL, Na 136 mmol/L, K 4.3 mmol/L, Cl 96 mmol/L, procalcitonin 0.287 ng/ml, Blood Gas Analysis Results pH 7.41; pO2 46; pCO2 51; HCo3 32.3; BE 6.8; SpO2 95; P/F ratio 203.

Repeat chest x-rays were performed and infiltrates were found in both lung fields, additional therapy was levofloxacin 1x750 mg IV infusion and fluconazole 1x400 mg IV infusion, and an abscess was found on the abdominal wall. The seventh day of treatment (15/3/2022) the patient felt the tightness increased, the pain in the skin had decreased, GCS 456, with blood pressure 132/91, N 132x/min, temperature: 36.7, RR 26x/min, SpO2 97 % with a mask cap

of 8 lpm. In sputum culture ESBL was found and in pus culture *Staphylococcus xylosus and Enterobacter cloacae were found.* The patient received cefoperazone therapy for pneumonia. On the ninth day, the patient underwent another chest x-ray and it was found to be worse, and the BGA results showed respiratory failure. The patient was declared dead with CM Sepsis Shock and Respiratory Failure.

3. RESULTS

Immune thrombocytopenia Purpura (ITP) is an acquired autoimmune disease, a disorder that occurs characterized by a low platelet count of less than 100 (103/µl). Although the exact pathogenesis remains unknown, it is believed to result from *autoimmune destruction of platelets* due to the formation of *antiplatelet autoantibodies* and decreased platelet production arising from megakaryopoiesis defects. As per literature, both B- and T-cell immune response play an important role in the pathogenesis of ITP, ITP can be classified into two subtypes–primary ITP (when no cause is found) and secondary ITP when there is an underlying cause, the disorder can be associated with destruction *platelet autoimmunity* , and the latter includes *[Human immunodeficiency]* virus infection (HIV), Hepatitis C (HCV), and Helicobacter pylori], autoimmune diseases [such as systemic lupus erythematosus (SLE), rheumatoid arthritis (RA), antiphospholipid, antibody syndrome (APS), and lymphoproliferative disorders such as chronic lymphocytic leukemia (CLL). Most studies say that in developed countries 80% of cases of ITP are primary, and only 20% are secondary, in contrast to developing countries that secondary ITP is more common than primary ITP due to population heterogeneity and the high incidence of related infections in developing countries.10 ITP is divided into the following categories:

Table 1. ITP categories according to the International Working Group.2

ITP category	
Primary ITP	No other factors were found to cause thrombocytopenia
Secondary ITP	It can occur due to disease or other conditions that underlie thrombocytopenia
Newly diagnosed ITP	Refers to cases of ITP that have just been diagnosed within 3 months
Persistent ITP	Refers to ITP cases diagnosed 3-12 months previously
Chronic ITP	ITP cases that are more than 12 months old
Severe ITP	Bleeding symptoms appear that require therapy or new bleeding symptoms require additional intervention or an increase in the therapeutic dose

It is important to know the ITP category because it also influences the goals of the treatment. The table below states that newly diagnosed ITP is easier to remission than the other 2 phases, as well as the aim of treatment in each of the phases below, it is explained that in chronic ITP the patient should accept that thrombocytopenia will become chronic and the quality of life takes priority over the number of platelets.2

Table 2. Disease phases and goals and therapy.2

Phase	Definitions	Treatment goals
Newly diagnosed	Up to 3 months after diagnosis. Spontaneous remissions common	Prevention or termination of bleeding, cure. Because treatment might only take a short time period side effects are more acceptable.
Persistent	Between 3 and 12 months after diagnosis. Spontaneous remissions are less common	Prevention or termination of bleeding, cure. Since therapy now extends over a longer time period, the benefits and side effects must be weighed more strongly against each other.
Chronic	More than 12 months after diagnosis. Spontaneous remissions are uncommon	Prevention or termination of bleeding, cure. Patients should accept that thrombocytopenia will most likely be chronic. Quality of life and avoidance of side effects become more important than platelet count.

This patient is chronic ITP because it has been more than 12 months since the first diagnosis, and the target of therapy for this patient is to avoid bleeding, and educate that thrombocytopenia will be chronic, the importance of taking medication regularly , and avoiding side effects.

ITP is an autoimmune disease caused by the destruction of normal platelets due to antibodies and disruption of megakaryocyte production. 7 T cells are activated due to the recognition of platelet-specific antigens on APCs *(antigen presenting cells)* which then induce antigen-specific expansion on B cells. Then B cells produce specific autoantibodies against glycoproteins expressed on platelets and megakaryocytes. Circulating platelets are bound by platelet autoantibodies and then attach to the FC receptor of splenic macrophages which results in the destruction of platelets. Apart from that, anti-megakaryocyte autoantibodies are also formed which reduce the ability of megakaryocytes to produce platelets. There is production of autoantibodies (A) which increases the destruction of platelets by splenic macrophages (B) and decreased platelet production due to anti-megakaryocyte antibodies (C). 7 There is some literature that divides them into Platelet Autoantibodies, T *Lymphocytes* , and Impaired Thrombopoiesis, which are Platelets autoantibodies are divided into 4, antibody-coated platelets which attach to Fc receptors on macrophages and dendritic cells in the spleen and liver, which will be phagocytes and degraded by them, after binding to the Ashwell receptor in the liver degradation of the damaged platelets occurs, the process This will stimulate the formation of new thrombopoietin in the liver. [11]

Then there is direct damage of platelets by autoantibodies and platelet function defects, antibodies can directly damage platelets and trigger complement lysis, and there is also the process of autoantibodies attaching to the platelet receptor membrane which disrupts the adhesion and aggregation process, thus causing thrombocytopenia - increasing the tendency to bleed, then the second is T *Lymphocytes* and immune dysregulation, in this study it was found that a reduction in the number of regulatory T lymphocytes triggers immunodysregulation, and T lymphocytes can directly damage platelets. Lastly is insufficient thrombopoiesis, namely damage to megakaryocytes due to the GP membrane, where platelet autoantibodies directly damage

megakaryocytes in the bone marrow and disrupt the production of platelets. 12

Bleeding is the most frequently encountered clinical manifestation. Bleeding can take the form of petechiae in the leg area, rarely found on the back and hands, bleeding on mucous membranes such as the mouth and nose, urogenital bleeding and menstrual bleeding, bleeding or hematoma even from small trauma, bleeding can also occur in internal organs such as intracerebral bleeding but very rare. Petechiae are usually flat and cannot be palpated, palpable purpura is more likely to be purpuric vasculitis. Patients with ITP have a higher risk of infection due to immunosuppressive therapy or *splenectomy* . 14 Platelets also have a role as a host for the body's defense against infection [15] .

In line with studies which say that platelets under 30,000 tend to be 4 times more susceptible to infection and higher mortality than the general population. Iron deficiency anemia can also arise due to bleeding, therefore microcytic iron deficiency anemia can occur in patients with ITP, but not macrocytic anemia, apart from that patients with ITP often complain of feeling tired and tired easily, including several episodes of depression which have also been reported16.

The diagnosis of ITP is a diagnosis by excluding other diseases. ITP is established after other causes of thrombocytopenia can be excluded. Some infections need to be ruled out such as HIV, Hepatitis C, Helicobacter Pylori, and CMV. Suspicion of malignancy (can be ruled out through imaging) and the influence of drugs such as valproate, *heparin should* also be ruled out. Examination of antiphospholipid antibodies and lupus anticoagulant must be checked if the symptoms of ITP become persistent/chronic. 17 The BMA examination cannot prove ITP, the BMA examination only excludes other diagnoses. In ITP the patient must also be checked for coagulation function, and several recent publications state that there is a close link. between ITP and Helicobacter infection, especially in Asian areas, because ITP sufferers often receive corticosteroids and this causes a high risk of developing ulcers. [17]

This patient has been diagnosed with ITP since 2016, the patient came with complaints of heavy and prolonged menstrual bleeding, with low platelets of 11,000, the patient also complained of feeling tired, and there was purpura and petechiae on the body, the possibility of secondary ITP such as infection has been checked. HIV, hepatitis C, hepatitis B, autoimmune panel were not obtained, but the Urea Breath Test for Helicobacter pylori could not be checked because the patient was still taking omeprazole, and after that he was scheduled to be examined, but the patient experienced a worsening due to respiratory failure and septic shock.

According to *the American Society of Hematology 2019 guidelines for immune thrombocytopenia* , therapy for ITP is divided into several recommendations according to *the Grading of Recommendations Assessment, Development and Evaluation (GRADE),* namely Recommendations 1A and 1B, where patients receive *corticosteroid therapy* first if they meet the new ITP criteria. diagnosed and platelets below 30,000 (the patient is asymptomatic / there is mucocutaneous bleeding), while recommendation 1B if the patient has platelets more than 30,000 is observed without corticosteroids. 18 For steroid doses, follow recommendations 4 and 5, namely *corticosteroids* can be given prednisone (0.5-2.0 mg/ kg per day) or dexamethasone (40 mg daily for 4 days), dexamethasone is reported to be superior because healing can be seen within 7 days, and in newly diagnosed ITP, it is recommended to administer *corticosteroids* alone rather than the combination. [18]

Recommendation 3 according to ASH 2019 also states that newly diagnosed ITP cases should be given *corticosteroids* for <6 weeks including main therapy and tapering off. In a literacy study comparing the prolonged course and short course (<6 weeks), it was found that the results of the prolonged course (> 6 weeks) causes more side effects, although there is still not much literature that discusses this in more depth, but you need to be aware of the big side effects such as hypertension, hyperglycemia, sleep and *mood disorders, gastric* irritation or *ulcers* , *glaucoma, myopathy* , and *osteoporosis.* . [18] Other studies state that systemic and long-term use of corticosteroids can also cause several side effects, such as osteoporosis because it affects the trabecular bone 6-12 months after use, in the endocrine and metabolic fields such as increased blood sugar, Cushing's syndrome such as *buffalo hump, moon face,* abdominal obesity. Long-term *glucocorticoids will suppress the hypothalamic-pituitary-adrenal (HPA) axis,* which will cause *corticotropin-releasing hormone (CRH)* and adrenocorticotropic hormone (ACTH) to decrease. Long-term suppression of ACTH can cause the adrenal glands to atrophy. [7]

In this patient there were symptoms of Cushing's, such as moon face, buffalo hump, striae, pendulous abdomen, high blood sugar, in this patient also found decreased cortisol accompanied by kidney hypoplasia on MRI due to long-term use of corticosteroids .

According to the 2019 ASH Guideline, if it has been more than 3 months and is still steroid dependent or unresponsive, you can be given additional line 2, namely eltrombopag or romiplostim, where it is said that eltrombopag is superior to romiplostim. 18 If it is more than 1 year, *splenectomy can be considered* , *splenectomy* is recommended more than 1 year after diagnosis because the potential for remission in the first year is very high. 18 According to Matzdorff (2018) Eltrombopag and Romiplostim are effective in patients with or without *splenectomy* , both are also effective for young and old, with TPO-RA about half of patients can stop the steroids. [19]

In this patient, it is chronic ITP that is >12 months, the treatment options for this patient are *splenectomy* , TPO-RA or rituximab, but the patient refused surgery and received TPO-RA because it had a better *outcome , and this patient was already receiving Rebozet (eltrombopag)* , However, the patient had no control for approximately 2 years, and consumed MP himself by increasing and decreasing the dose independently. It is also known that patients have problems regarding costs related to TPO-RA therapy which is relatively expensive.

In patients with ITP for less than 12 months, it is best to choose TPO-RA or rituximab, however TPO-RA is recommended because of the better response, but is expensive, rituximab can be considered if you want to avoid long-term treatment or for those who cannot afford TPO-RA. Meanwhile, patients with ITP for more than 12 months can choose *splenectomy* , TPO-RA or Rituximab, patients who want to avoid long-term therapy can choose *splenectomy* or rituximab, although *splenectomy* has good *outcomes* and higher remission than rituximab, but ASH remains recommend rituximab because of the risks of surgery and the risk of infection and thrombosis. 18 Infections in ITP sufferers can include pneumonia, sepsis, and abscesses which were found in a study conducted at Leiden University. [9]

Abscesses often arise in many tissues and organs of the body, such as subcutaneous tissue, lymph nodes, soft tissue, adipose around the anus, and breasts in pregnant women. The most dangerous complication is the spread of the abscess to surrounding tissue which can sometimes cause death of the tissue (gangrene). The main causes are pathogenic bacteria, fungi or parasites, the most common cause is Staphylococcus

aureus. Staphylococcus aureus can also cause subdural abscesses and parasites cause abscesses especially in developing countries. Among hospitalized patients, the prevalence rate of abscesses ranges from approximately 7–10%. Abscess is the number 3 disease frequently encountered in emergency service centers after chest pain and asthma21. There are various risk factors that can cause skin abscesses, such as old age and sufferers who have immune deficiencies, such as human immunodeficiency virus and diseases of the liver, kidneys and vascular insufficiency (especially lymphatic or venous), predisposing factors associated with the risk of skin abscesses such as loss of skin surface such as in trauma (corrosion, penetrating wounds, pressure sores, leg vein wounds, insect bites, injection drug use), skin inflammation (such as eczema, psoriasis and radiation therapy), edema due to poor lymphatic drainage, edema due to venous insufficiency , obesity, diseases or medications that suppress immunity (such as diabetes or HIV infection) [21] .

Steroids can trigger the formation of abscesses, steroids cause peripheral neutrophilic leucocytosis, steroids in high doses can suppress the phagocytosis process and the bactericidal process of neutrophils, steroids suppress the accumulation of leukocytes at the site of inflammation. Long-term corticosteroids can also cause infections, there are studies that say the duration and dose are linear with the process of infection, especially bacterial, viral or fungal infections. The safe dose for using steroids is prednisone under 10 mg, which is said to have minimal side effects, apart from that, the use of *corticosteroids* can obscure the initial symptoms of infection by inhibiting the release of cytokines associated with inflammation and fever, especially in the early phase of infection. [7]

Other studies also say that when there is an open wound, bacteria can enter through the skin and form an abscess in response to inflammation which will cause blockage of the sweat glands or sebaceous glands or hair follicles or pockets of pre-existing abscesses. Staphylococcus aureus, E. coli, P. aeruginosa, and Streptococcus pyogenes are the most common types of bacteria that cause skin abscesses in areas of the body. Some bacteria from S.aureus produce a toxin called Panton-Valentine leukocidin (PVL), which kills white blood cells, and causes the body to produce more white blood cells to continue fighting infection. This strain of PVL bacteria can cause more serious conditions: such as septicemia which causes bacteria to multiply in the blood and pneumonia and even systemic sepsis. [22]

In this patient, abscesses were found in the gluteus region and abdomen due to long-term use of *corticosteroids* , ESBL was also found in sputum and urine, and at the base of the wound there was a staphylococcus xylosus infection which was obtained from culture results. The patient worsened on the 10th day of MRS, and experienced complications such as pneumonia and septic shock.

4. CONCLUSION

The case of a 23 year old female patient with ITP and its complications has been reported. The diagnosis is made based on the history, clinical signs and symptoms, as well as supporting examinations. However, the patient experienced complications from inappropriate and prolonged treatment, which caused systemic infection, the patient finally died during the second hospitalization due to shock mortis, sepsis and respiratory failure.

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