International Journal of Advances in Medicine
Tanu M et al. Int J Adv Med. 2023 Feb;10(2):171-173
http://www.ijmedicine.com
pISSN 2349-3925 | eISSN 2349-3933
DOI: https://dx.doi.org/10.18203/2349-3933.ijam20230071

Case Report

Non-dengue related febrile thrombocytopenia in HIV patient: a case report

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Received: 10 December 2022
Accepted: 06 January 2023

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ABSTRACT

In tropical areas, febrile thrombocytopenia is often caused by infectious diseases, especially dengue. Thrombocytopenia is common in Human Immunedeficiency Virus (HIV) patients at any stage or as a complication of the illness. Here we presented a case report of a 38-year-old female initially diagnosed with dengue fever but turn out to be thrombocytopenia due to HIV infection with pneumocystis carini pneumonia (PCP). Febrile thrombocytopenia in people living with HIV should be observed carefully. It may be caused by a new opportunistic infection or as the natural progression of HIV disease.

Keywords: Dengue, Human immunodeficiency virus, Thrombocytopenia, Pneumocystis carini pneumonia

INTRODUCTION

In tropical countries, numerous infectious diseases coexist. It is predominated by infection and usually associated with fever. Acute fever with thrombocytopenia (febrile thrombocytopenia) is a common clinical problem in medical wards. The most frequent etiology especially in the rainy season between July until October is dengue fever.[1] Dengue infection is characterized with acute high fever/breakbone fever accompanied with thrombocytopenia and leukopenia.[2] Indonesia is one of endemic country for dengue infection and at the same time prevalent for Human Immunedeficiency Virus (HIV) infection. People living with HIV usually have thrombocytopenia at any stage or as a complication of the disease.[1]

Here we present a case report of a 38-year-old woman with non-dengue related febrile thrombocytopenia but turn out to be thrombocytopenia due to HIV infection with pneumocystis carini pneumonia (PCP).

CASE REPORT

A 38-year-old female presented with fever and progressive dyspnea two days before admission. Fever was high on the first day, reached 38.5^{0}C (on 26 August), and then relieved at next day. She also complains of dry cough for about three weeks, chills, headache, nausea, and vomiting. There were no signs of active bleeding (epistaxis, gum bleeding, and petechiae) and the tourniquet test was negative. She was diagnosed with dengue fever by the emergency department. The patient was diagnosed with HIV infection 5 years ago and routinely takes Tenofovir – Lamivudine – Efavirenz (TLE) once daily at night for Anti-Retroviral Therapy (ART). She was allergic with analgesic non-steroidal anti-inflammatory drug (NSAID), and paracetamol.

Physical examination revealed blood pressure of 120/89 mmHg, regular heart rate of 90 bpm, respiratory rate of 24 times/minute, no chest retraction, temperature of 38.3^{0}C, and oxygen saturation of 97% at room air. Body weight was 45 kg and height was 157 cm. Body mass index (BMI)

18.2 (underweight). There was no anemic conjunctiva. She had oral thrush on her tongue. There was no persistent generalized lymphadenopathy palpable. Cardiac examination within normal limits. Crackles were discovered in both lung areas during a chest examination. The abdominal examination was normal. The capillary refill time was less than 2 seconds, and there were no petechiae.

Table 1: Routine complete blood count result.

Day of illness	HB (g/dl)	HT (%)	Leukocyte (µl)	Platelet (µl)	Temperature (^{0}C)
Day 3 (28 August 22)	13.8	40.4	12.200	27	38.3
Day 4	13.9	40.9	10.900	18	36.9
(29 August 22)	13.3	39.3	9.400	35	36.8
Day 5	13.5	40.7	9.800	20	36.5
(30 August 22)	13.6	40.5	8.240	70	36.2
Day 6	13.6	41.4	8.520	73	36.4
(31 August 22)	13.5	41.6	8.600	50	36.6
Day 7	13.1	40.2	8.400	51	36.4
(1 Sept 22)	13.0	38.9	8.530	90	36.7
Day 8	13.4	40.1	8.350	117	36.8
(2 Sept 22)					

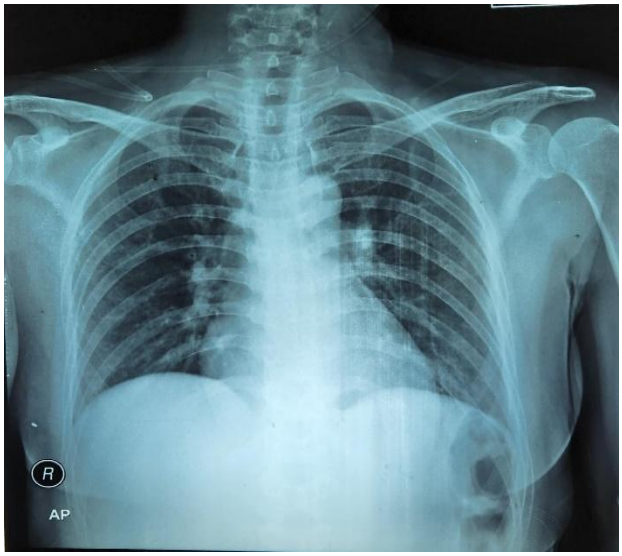


Figure 1: Chest X-Ray suggesting pneumonia.

On the first day of admission, a routine complete blood count showed hemoglobin 13.8 g/dl, hematocrit 40.4%, WBC 12.200/ µl, and thrombocyte 27.000/µl. Aspartate aminotransferase (AST) 63 U/L, alanine aminotransferase (ALT) 56 U/L. Neither the NS1 (fourth day) nor the IgM Dengue (eighth day) tests were negative. Urinalysis was normal. Chest X-ray revealed paracardial and perihilar infiltrates on both sides, indicating pneumonia. This patient was scheduled for 12 hourly blood tests. The results of her blood test are showed in Table 1.

The patient was managed with intravenous NaCl 0.9% solution, Esomeprazole 40 mg QD, Cotrimoxazole 960 mg QID, Levofloxacin 750 mg QD, methylprednisolone 62.5 mg BID, Nystatin drops 4 ml QID, and continue her ARV. She was discharged after four days of admission.

DISCUSSION

This patient was diagnosed with PCP and thrombocytopenia due to HIV infection. Although the symptoms may be similar to dengue fever, we believe it was not DF / DHF since dengue usually has an acute onset, with high and continuous temperature (may reach 39-40^{0}C), lasting 2-7 days before falling to a normal / subnormal level. A biphasic fever pattern may be observed.[2] Studies by Pradipa et al. revealed there is a triple peak of high-temperature pattern that may be an adjunct for diagnosis dengue fever.[3] The patient only had a fever of 38.5^{0}C on the first day, fever-free for the next day, rise again, then afebrile until she was discharged from the hospital. This pattern does not correlate to the typical dengue fever.

Thrombocytopenia is the most common hematologic manifestation during the acute phase of Dengue fever. Platelet count in DHF/DF shows a significant decrease on the 4th until the 7th day of illness and reaches a normal level on the 8th or 9th day. Dengue is also characterized by leucopenia.[4] In this patient, there is only thrombocytopenia in the absence of leukopenia. Her thrombocytes rise and fall on days 4 and 5 and keep rising after that. Neither the NS1 (fourth day) nor the IgM Dengue (eighth day) tests were negative. Three major factors have been proposed as the mechanism of thrombocytopenia in Dengue patients (1) direct lesion of progenitor cells by DENV; (2) infected stromal cells; (3) changes in bone marrow regulation.[4] Thrombocytopenia is a frequent hematologic disorder in patients infected with

HIV. It can occur independently of other cytopenia and at any stage of HIV infection, occurring at a rate of 4.1-40%.[5,6] Thrombocytopenia ranges from mild to moderate to severe (<50.000/ µl).[5] The underlying mechanisms of thrombocytopenia are still poorly understood. The possible mechanisms are increasing immune-mediated peripheral platelet destruction by antibodies, impaired megakaryocytes, or direct infection of megakaryocytes which leads to low production of platelets from those precursor cells. Besides that, thrombocytopenia can occur as a result of hypersplenism, opportunistic infections, malignancy, and toxic and myelosuppressive effects of HIV medication (Zidovudine).[5,6] This was assumed to be the possible pathogenic explanation of how this patient got thrombocytopenia. ARV can have a positive or negative effect on hematological disorder. Thrombocytopenia and anemia decreased after initiation of HAART.[6] She had no history of blood transfusions while using that ARV regimen. Therefore, her HIV infection and a new opportunistic infection (PCP and candidiasis oral) might be the root causes of her thrombocytopenia.

PCP is a fungal infection that can be life-threatening in immunocompromised patients.[7] In high HIV prevalence settings, the diagnosis of PCP is frequently made presumptively using case definitions that include clinical features and chest X-ray in an immunosuppressed individual with a CD4 < 200 cells/µL.[8] This patient has progressive dyspnea, fever, non-productive cough, oral thrush, and crackles on both sides of the lungs. These are often clinical symptoms that appear in a patient with PCP, especially if she is immunocompromised.

Cotrimoxazole, namely Trimethoprim-Sulfamethoxazole (TMP-SMX) is used for prophylaxis and the therapy of PCP for 21 days. While corticosteroids contribute as an additional anti-inflammation therapy to prevent lung damage, blunt inflammatory responses, oxygen deterioration, and the incidence of respiratory failure. Ideally, it should begin when PCP therapy is initiated and within the first 72 hours of treatment.[7] Nystatin drops is a safer therapy for this patient because she had a slight rise in AST level.

CONCLUSION

Febrile thrombocytopenia in people living with HIV should be observed carefully. It may be caused by a new opportunistic infection or as the natural progression of HIV disease.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Tanu M, Suryana K. Non-dengue related febrile thrombocytopenia in HIV patient: a case report. Int J Adv Med 2023;10:171-3.