



Toxic or Septic Myocarditis in a Patient Suffering from Aplastic Anemia

**Karim Mounaouir ^{a*}, Mohammed Nachid ^a, Ali El Jazouli ^a,
Meryem Haboub ^a, Arous Salim ^a, Ghali Bennouna ^a,
Abdenasser Drighil ^a and Rachida Habbal ^a**

^a *Cardiology Division, Ibn Rochd University Hospital, Casablanca 20250, Morocco.*

Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

Article Information

Open Peer Review History:

This journal follows the Advanced Open Peer Review policy. Identity of the Reviewers, Editor(s) and additional Reviewers, peer review comments, different versions of the manuscript, comments of the editors, etc are available here: <https://www.sdiarticle5.com/review-history/100282>

Case Report

Received: 23/03/2023

Accepted: 25/05/2023

Published: 02/06/2023

ABSTRACT

The present case report highlights about Toxic or septic myocarditis in a patient suffering from aplastic anemia. A 21 years old female patient, without any cardiovascular risk factor, suffering from Aplastic Anemia, admitted initially for feverish neutropenia for which she received adequate antibiotherapy followed by Thymoglobulin® (anti-thymocyte globulin (rabbit)), which is an immunosuppressive agent used in treatment of Aplastic Anemia. Adverse cardiovascular reactions that have been reported in association with ATG include myocarditis, "cardiac irregularity," chest pain, hypertension, hypotension, tachycardia, and bradycardia.

Keywords: *Toxic myocarditis; septic myocarditis; aplastic anemia; myocarditis; chest pain; electrocardiogram; cardiac enzymes; cardiac magnetic resonance imaging; drug reactions.*

1. INTRODUCTION

"Myocarditis refers to the inflammation of the myocardium, resulting in tissue degeneration or

necrosis. Previously known as inflammatory myocarditis, it typically affects younger people. Myocarditis is classified as acute, fulminant, chronic active, or chronic persistent" [1]. Acute

*Corresponding author: Email: KARIM.MOUNAOUIR@gmail.com;

myocarditis constitutes most cases of myocarditis (65%) and is mostly caused by an infection.

The clinical presentation is variable and includes febrile illness, mild chest pain, arrhythmias, heart failure, cardiogenic shock, or death. The clinical diagnosis is often challenging, and the management is usually supportive.

2. CASE PRESENTATION

A 21 years old female patient, without any cardiovascular risk factor, suffering from Aplastic Anemia, admitted initially for feverish neutropenia for which she received adequate antibiotherapy followed by Thymoglobulin® (anti-thymocyte globulin (rabbit)), which is an immunosuppressive agent used in treatment of Aplastic Anemia. 3 days after after it's administration, the patient was complicated by septic shock. After 12 hours, she presented chest pain. Her pulse was 150 bpm, blood pressure 62/43, respiratory rate was 32c per minute, purulent sputum.

Physical examination found rhonchi and crackling sound when auscultating the lungs, there were signs of global heart failure such as: crackling sound when auscultating the lungs, oedema in legs, Jugular Vein Distention.

The electrocardiogram showed a sinus tachycardia of 150 bpm with negative T waves in inferior and anterior derivations (Fig. 1). A bedside echocardiography showed an impaired left and right ventricular function, LVFE was at 30%, slightly elevated left ventricular filling pressure, moderate mitral regurgitation (Fig. 2), mild tricuspid regurgitation.

On laboratory investigation, CRP was high 365, neutropenia at 50, Hb at 6.9, Platelets <10 000, at troponin at 648, normal renal function. The patient received 2mg of noradrenaline on continuous perfusion, triple antibiotherapy: vancomycin + Imipenem/cilastatin + amikacyn.

We suspected cardiotoxicity due to anti-thymocyte globulin (ATG) or a Myocarditis due to septic shock, so a cardiac MRI have been indicated.

The patient was transferred to intensive unit care in which she presented multivisceral failure even with the increased doses of noradrenaline and introduction of dobutamine. She passed away 7 days later.

3. DISCUSSION

The diagnosis of myocarditis is challenging, due to the variability of symptoms that are often unspecific, ranging from asymptomatic to life-threatening.

“ATG is a monomeric immunoglobulin G (IgG) obtained from the serum of horses or rabbits immunized with human thymus lymphocytes. It is a lymphocyte-selective immunosuppressant” [1]. “The most commonly reported side effects of treatment with ATG are fever, chills, leukopenia, thrombocytopenia, and dermatologic manifestations (rashes, urticaria, pruritis, wheal, and flare)” [1]. Adverse cardiovascular reactions that have been reported in association with ATG include myocarditis, “cardiac irregularity,” chest pain, hypertension, hypotension, tachycardia, and bradycardia.



Fig. 1. Patient ECG

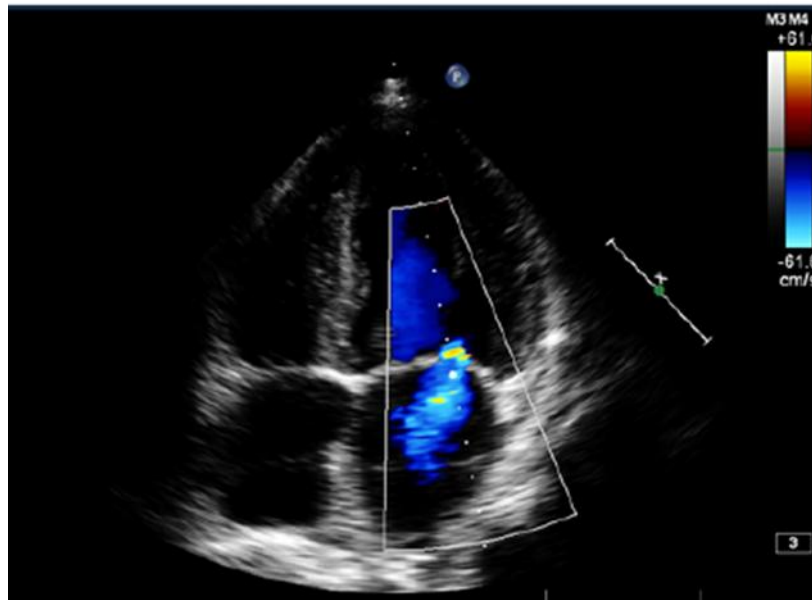


Fig. 2. TTE of patient showing moderate mitral regurgitation

Sepsis, defined by consensus conference as “the systemic inflammatory response syndrome (SIRS) that occurs during infection,” is generally viewed as a disease aggravated by the inappropriate immune response encountered in the affected individual [2]. “Myocardial dysfunction affects approximately half of patients with sepsis admitted to intensive care units (ICUs)” [3]. “Cardiac abnormalities can involve both the left and right ventricles and may include both systolic and diastolic dysfunctions” [4]. “Studies have shown that plasma concentrations of troponin, as biomarker of myocardial injury, correlate well with functional abnormalities seen on echocardiography” [5].

Toxic or septic myocarditis is a rare but serious condition characterized by inflammation of the heart muscle (myocardium) caused by toxins or infection. It can be associated with various underlying medical conditions, including aplastic anemia, a rare bone marrow disorder characterized by the failure of the bone marrow to produce an adequate number of blood cells [6].

Aplastic anemia is a hematological disorder characterized by pancytopenia, which refers to the simultaneous reduction in the number of red blood cells, white blood cells, and platelets in the peripheral blood. The pathophysiology of aplastic anemia involves immune-mediated destruction of hematopoietic stem cells and impaired production of blood cells. Patients with aplastic

anemia are prone to infections due to the compromised immune system, and the risk of developing severe complications, such as myocarditis, is increased [7].

It is important to note that toxic or septic myocarditis carries a high mortality rate, particularly in the presence of underlying medical conditions like aplastic anemia especially if we add the immunosuppressive therapy (like: anti-thymocyte globulin) and its side effects. Therefore, early recognition and intervention are crucial to improve patient outcomes. This case highlights the need for heightened clinical suspicion for myocarditis in patients with aplastic anemia presenting with cardiac symptoms [8].

Infectious Myocarditis is more likely to be the cause given the context of lymphopenia, septic shock, the high serum levels of CRP, purulent sputum and leg ulceration.

4. CONCLUSION

Myocarditis is a serious consequence of sepsis that has a detrimental impact on patient survival. Sepsis-induced myocarditis is present in half of all the patients with septic shock and is characterized by the significant impairment of the left ventricular (LV) systolic and diastolic function. Despite significant progress in understanding the pathophysiology and treatment of sepsis over the last two decades, much of the associated mortality and morbidity remains high.

CONSENT

As per international standard or university standard, patient(s) written consent has been collected and preserved by the author(s).

ETHICAL APPROVAL

As per international standard or university standard written ethical approval has been collected and preserved by the author(s).

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Pfizer Inc. Atgam (lymphocyte immune globulin, antithymocyte globulin [equine] sterile solution) 2011.
2. Bone RC, Balk RA, Cerra FB, Dellinger RP, Fein AM, Knaus WA, Schein RMH, Sibbald WJ. Definitions for sepsis and organ failure and guidelines for the use of innovative therapies in sepsis. Chest. 1992;101:1644–1655.
3. Zaky A, Deem S, Bendjelid K, Treggiari MM. Characterization of cardiac dysfunction in sepsis: An ongoing challenge. Shock. 2014;41:12–24. DOI: 10.1097/SHK.000000000000065
4. Pulido JN, Afessa B, Masaki M, Yuasa T, Gillespie S, et al. Clinical spectrum, frequency, and significance of myocardial dysfunction in severe sepsis and septic shock. Mayo Clin Proc. 2012;87:620–628. DOI: 10.1016/j.mayocp.2012.01.018
5. Landesberg G, Jaffe AS, Gilon D, Levin PD, Goodman S, Abu-Baih A, Beerli R, Weissman C, et al. Troponin elevation in severe sepsis and septic shock: The role of left ventricular diastolic dysfunction and right ventricular dilatation*. Crit Care Med. 2014;42:790–800. DOI: 10.1097/CCM.000000000000107
6. Raza F, Anwer F, et al. Myocarditis in aplastic anemia: A rare complication. Cureus. 2020;12(10):e10756.
7. Samanta S, Samanta S, Mishra TS, et al. Aplastic anemia presenting as fulminant myocarditis: a case report and review of the literature. J Med Case Rep. 2018;12(1):175.
8. Saini H, Kumar P, Singh S, et al. Septic myocarditis as a presenting manifestation of aplastic anemia. J Pediatr Hematol Oncol. 2019;41(4):e245-e248. DOI: 10.1097/MPH.0000000000001354

© 2023 Mounaouir et al.; This is an Open Access article distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Peer-review history:

The peer review history for this paper can be accessed here:
<https://www.sdiarticle5.com/review-history/100282>