Anterior Extensive STEMI in a 26-year-old patient, can Takayasu Arteritis be the Culprit: A Case Report

A. F. Theno[a], A. Regina[a], P. K. Lituhayu[a], A. D. Lamara[b], Y. H. Oktaviono[b*]

[a] Audrey.florencia.theno-2020@fk.unair.ac.id
[a]Faculty of Medicine, Airlangga University, Surabaya 60132, Indonesia,
[b]Departement of Cardiology and Vascular Medicine, Dr. Soetomo General Hospital, Surabaya 60132, Indonesia

Abstract

Background: Takayasu arteritis (TA), also known as pulseless disease, is a chronic idiopathic rare granulomatous vasculitis of large arteries and its branches. TA can be classified into 2 phases: 1) systemic phase; and 2) occlusive phase. The systemic phase commonly presents with constitutional symptoms of inflammation, followed by symptoms caused by narrowed vessels in the systemic phase. Clinical manifestations of TA vary along the clinical course, ranging from fever, chest pain, limb claudication, decreased or absence of arterial pulsation to ischemic symptoms. Although, the main entity to the diagnosis is imaging to determine stenosis of major vessels.

Case Summary: A 26-year-old male was referred to our center with typical chest pain 11 hours prior to admission, without a previous history of myocardial infarction. The patient also had intermittent claudication, with a history of amputation in the right toe 1 year prior. Currently, the patient consumes alcohol, in addition to being an active smoker for 10 years. ECG study revealed a vast anterior STEMI. Systolic blood pressure between arms showed a difference of more than 10 mmHg. The patient had a high ESR (34 mm/hour), elevated transaminase enzymes (AST 694 IU/L, ALT 109 IU/L), high inflammatory markers (CRP 12.6 mg/L, LED 34mm/hr), and elevated troponin I (32.04 ng/mL). Chest X-Ray displays enlarged heart size with 55% CTR. Echocardiography was performed and showed an HFrEF (EF by Teich 38%), LV dilatation, and severe hypokinetic of anterior to lateral wall of LV. Doppler Ultrasound (DUS) found no colour coded on the right popliteal artery, right dorsalis pedis artery and no flow from left deep femoral artery, left common femoral artery, left anterior tibial artery, and left posterior tibial artery, suggesting a peripheral artery disease (PAD) of both lower extremities. DUS also revealed a small abdominal aorta diameter (1.4 cm). Following findings highly suggest the probability of TA. Besides PPCI stent in proximal-mod LAD, this patient also received loop diuretic, anticoagulant, dual antiplatelet, antihypertension, statin, and beta-blocker. Antiinflammation was not yet given because the real confirmation of TA diagnosis needed to be done.

Discussion: Takayasu arteritis is a rare form of vasculitis. Delays of TA diagnosis may result in significant morbidity, hence proper assessment is pivotal. From Ishikawa criteria, this case consists of one major

criteria, that is more than one month duration of clinical signs and symptoms, following two minor criterias, which are high ESR and coronary artery lesion, suggesting high probability of TA. From American College of Rheumatology (ACR) criteria, this case is including presence of claudication of extremities, systolic blood pressure difference of >10 mmHg, and narrowing of aorta as arteriogram abnormality, highly suggesting TA diagnosis. Both criteria include age <40 years old as one of the criteria. Besides therapy for the cardiovascular symptoms, antiinflammation is also needed to treat the underlying cause.

Keywords: Takayasu Arteritis; STEMI; Peripheral Artery Disease; Heart Failure; Vascular Inflammation

1. Introduction

When discussing cardiovascular medicine, the relationship between anterior extensive ST-segment elevation myocardial infarction (STEMI) and the development of Takayasu arteritis presents a compelling avenue for investigation. Anterior extensive STEMI, characterized by ischemia affecting a large portion of the anterior wall of the heart, triggers a cascade of inflammatory responses and immune reactions. This may serve as a potential trigger for the development or exacerbation of autoimmune diseases, including Takayasu arteritis, an uncommon but serious form of large vessel vasculitis that primarily affects the aorta and its major branches. While the precise etiological mechanisms linking these two conditions remain to be fully elucidated, exploring the correlation between anterior extensive STEMI and Takayasu arteritis could offer valuable insights into the complex interplay between cardiovascular events and autoimmune phenomena.

In this case report, we present a detailed examination of a patient who experienced anterior extensive STEMI and subsequently developed Takayasu arteritis. By dissecting the clinical course, laboratory findings, and imaging studies, we aim to shed light on potential causative factors and pathophysiological connections between these seemingly disparate conditions. This investigation may contribute not only to our understanding of the intricate relationship between cardiovascular events and autoimmune responses but also to the development of targeted therapeutic strategies for individuals who face the challenge of managing these coexisting medical complexities.

2. Case

A 62-year-old female was referred to our center with progressive loss of consciousness. CT scan examination was immediately done for the patient to strike out other possible differential diagnosis. The CT scan report (Figure 1) showed no evidence of new cerebrovascular stroke, leading to other underlying possibilities. Following radiologic examination, cardiomegaly and ischaemic cardiomyopathy were present in x-ray as shown in Figure 2.

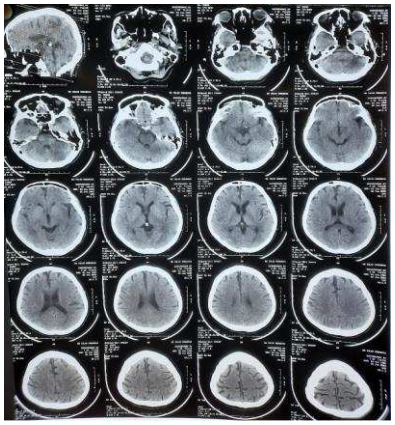

Figure 1. CT Scan Examination on Patient during Hospital Admission

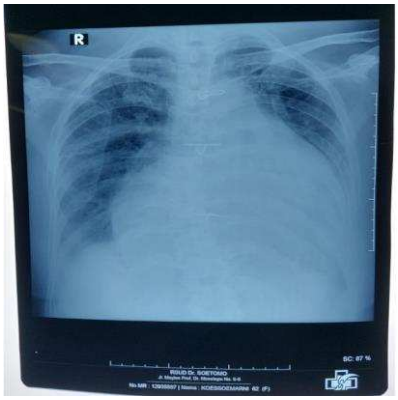

Figure 2. X-Ray of Patient with Cardiomegaly and Ischaemic Cardiomyopathy

Further cardiologic examination was done with ECG. ECG examination results as shown in Figure 3 and 4 indicate sinus arrest with junctional escape rhythm 30 bpm, multiple PVC, ST depression with T inversion in V3-V6 appeared to be Salvador Dali sign as a classical sign of digoxin toxicity, sometimes turning into slow atrial fibrillation. Echocardiography was also done in the patient showing dilation in all chambers of the heart whereas eccentric LVH (LVDMI 194 g/m^2; RWT 0.42), decreased systolic function of the left ventricle (EF by TEICH 39%), and akinetic in anteroseptal M anterior M-A with other segments showing hypokinetics.

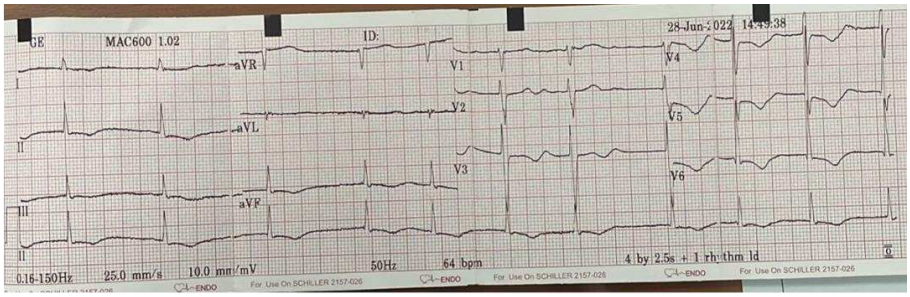

Figure 3. Electrocardiogram with Prominent 'Salvador Dali' Sign

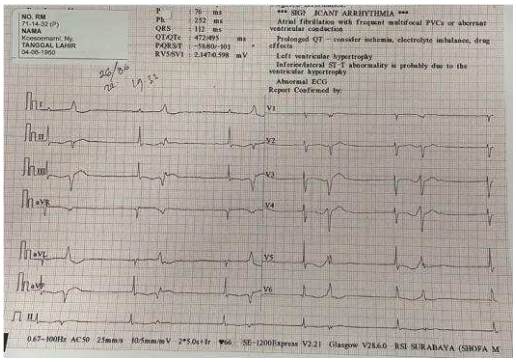

Figure 4. Electrocardiogram Result of Patient

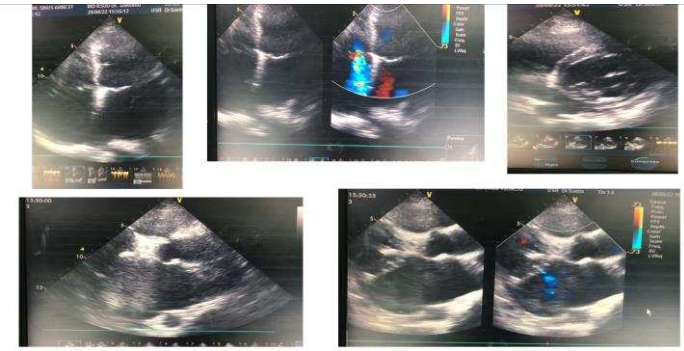

Figure 5. Echocardiogram Result of Patient

Two years prior, this patient routinely consumed digoxin 0.25 mg daily for heart failure. The patient had a reduced potassium level (3.3 mEq/L), increased creatinine serum level (1.3 µmol/L), and increased digoxin serum (8 ng/mL). Hence, the patient was diagnosed with digoxin intoxication. The patient underwent Ventricular Tachycardia and successfully cardioverted with 200 Joule. Unfortunately, DS-Fab was not present. Thus, we proceeded haemodynamic stabilization and prevented Ventricular Tachycardia using Temporary Pacemaker. The treatment plan was initiated with KCL infusion in PZ with a total input of 1250 ml fluid within 24 hours, followed by nitroglycerin pump (20 mcg/min) and bolus furosemide followed by furosemide pump of 5 mg per hour to increase urine production. Oral medications include candesartan, clopidogrel, spironolactone, atorvastatin, and warfarin. After 5 days of intensive treatment and diuretics, the digoxin intoxication was finally reversed (0.2 ng/mL).

3. Discussion

Loss of consciousness may be presented as symptoms to varying etiologies. The initial diagnosis was based on the main symptom during hospital admission, which was progressive loss of consciousness without any complaint of neither chest pain nor shortness of breath. The patient was initially assessed based on the medical record from the previous hospital prior to referral. CT scan was also done as the patient was referred for neurologic consultation.

The patient has a previous history of cardiovascular disease, arrhythmia problems, and heart surgery for ASD closure at 20 years of age. The patient's medication history was also taken into consideration during the initial assessment. Digoxin was one of the medications being consumed daily by the patient over the past 2 years due to the heart failure condition. Assessing from the ECG examination, prominent Salvador Dali sign was found to be evident in the patient, substantiated with an elevated digoxin serum level[5]. Hence, digoxin intoxication was assumed to be the underlying cause.

Due to its narrow therapeutic index, digoxin intoxication is very common to occur following prolonged use of digoxin. The likelihood for digoxin intoxication significantly increases in settings of hypokalemia, renal insufficiency, advanced age, hypercalcemia, alkalosis, acidosis, and myocardial infarction[6,7]. Digoxin intoxication may be lethal if not treated in the right manner, as it may slow down the conduction and increase refractory period of the heart through direct vagal tone increase, hence resulting in any types arrhythmia in particular should be taken into consideration when ventricular arrhythmias occur[8], as found in the patient which underwent ventricular fibrillation[6,9]. The patient was also presented with hypokalemia, which theoretically increases the risk of digoxin intoxication as the affinity of digoxin towards the Na^+/K^+ ATPase pump increases[10].

The approach to cases of digoxin intoxication relies on digoxin-specific antibody fragments in which the dose is given based on the serum concentration of digoxin as proven in several studies to be associated with lower mortality rate[11,12].

In a limited setting where DS-Fab is not present, intensive care for 3-5 days with adequate haemodynamic support could be an alternative while waiting for the digoxin naturally cleared by renal excretion. This is supported as digoxin excretion primarily occurs through renal[13]. In addition to that, although digoxin reduces hospital admission among heart failure patients, digoxin administration requires close monitoring for creatinine and potassium levels to minimize the risk for toxicity[14,15].

4. Conclusion

In conclusion, appropriate and adequate hemodynamic support may serve as a potential alternative approach in cases of digoxin intoxication where no DS-Fab is available.

5. Acknowledgments

None to declare

References

1. Dec GW. Digoxin remains useful in the management of chronic heart failure. The Medical Clinics of North America [Internet]. 2003 Mar 1 [cited 2022 Mar 6];87(2):317–37. Available from: https://pubmed.ncbi.nlm.nih.gov/12693728/
2. Parikh RR, Patel KR, Pergolizzi JV, Breve F, Magnusson P. Effects of Digoxin in Heart Failure (HF) With Reduced Ejection Fraction (EF). Cureus. 2022 Mar 2;
3. Chan BSH, Buckley NA. Digoxin-specific antibody fragments in the treatment of digoxin toxicity. Clinical Toxicology. 2014 Aug 4;52(8):824–36.
4. Pincus M. Management of digoxin toxicity. Australian Prescriber [Internet]. 2016 Feb 1;39(1):18–20. Available from: https://www.nps.org.au/australian-prescriber/articles/management-of-digoxin-toxicity
5. Vyas A, Bachani N, Thakur H, Lokhandwala Y. Digitalis toxicity: ECG vignette. Indian Heart Journal. 2016 Sep;68:S223–5.
6. Rameez Rehman, Hai O. Digitalis Toxicity [Internet]. Nih.gov. StatPearls Publishing; 2018. Available from: https://www.ncbi.nlm.nih.gov/books/NBK459165/
7. Marik PE, Fromm L. A case series of hospitalized patients with elevated digoxin levels. The American Journal of Medicine. 1998 Aug;105(2):110–5.
8. Gheorghiade M, Adams KF, Colucci WS. Digoxin in the Management of Cardiovascular Disorders. Circulation. 2004 Jun 22;109(24):2959–64.
9. S. Serge Barold. Alternans during fascicular ventricular tachycardia due to digitalis toxicity. Journal of Electrocardiology. 2018 May 1;51(3):450–1.
10. BrJCardiol. Digoxin: current clinical uses and management of toxicity [Internet]. bjcardio.co.uk. 2023. Available from: https://bjcardio.co.uk/2023/06/digoxin-current-clinical-uses-and-management-of-toxicity/
11. Chhabra N, Valento M, Bryant SM, Aks SE. Digoxin-Specific Antibody Fragment Dosing: A Case Series. American Journal of Therapeutics. 2016 Nov;23(6):e1597–601.
12. Frédéric Lapostolle, Borron SW, Verdier C, P. Taboulet, Guerrier G, Frédéric Adnet, et al. Digoxin-specific Fab fragments as single first-line therapy in digitalis poisoning. 2008 Nov 1;36(11):3014–8.
13. Cummings ED, Swoboda HD. Digoxin Toxicity [Internet]. PubMed. Treasure Island (FL): StatPearls Publishing; 2023. Available from: https://www.ncbi.nlm.nih.gov/books/NBK470568/#:~:text=Treatment%20%2F%20Management
14. Ziff OJ, Lane DA, Samra M, Griffith M, Kirchhof P, Lip GYH, et al. Safety and efficacy of digoxin: systematic review and meta-analysis of observational and controlled trial data. BMJ. 2015 Aug 30;h4451.
15. Konstantinou DM, Karvounis H, Giannakoulas G. Digoxin in Heart Failure with a Reduced Ejection Fraction: A Risk Factor or a Risk Marker? Cardiology. 2016;134(3):311–9.