Pk Heart J 2025:58(02)

ISSN:0048-2706 E-ISSN:2227-9199

Recurrent Supraventricular Tachycardia Following Carboplatin-Docetaxel Chemotherapy: A Hidden Threat in High-Risk Cancer Survivors

Ni Made Elva Mayasari, MD[1], Maria Lerista, MD[2], Nanda Kristania Abelfa, MD[1]

[1]Faculty of Medicine, Universitas Muhammadiyah Palembang (UMPalembang), Indonesia.
[2]Research Assistant (General Practitioner), Cardiology and Vascular Department, Mohammad Hoesin General Hospital, Palembang, Indonesia

Corresponding Author: Ni Made Elva Mayasari, MD

Abstract

A 57-year-old male with hypertension, diabetes, prior stroke, and nasopharyngeal carcinoma treated with carboplatin-docetaxel presented with chest pain and palpitations. Electrocardiography revealed unstable supraventricular tachycardia (SVT) requiring multiple electrical cardioversions. Echocardiography showed preserved ejection fraction but abnormal global longitudinal strain and prolonged QTc, indicating subclinical cardiotoxicity. He was stabilized with antiarrhythmic and supportive therapy and discharged after five days. This case highlights that recurrent SVT is a rare complication of carboplatin-docetaxel chemotherapy in high-risk patients. Timely recognition and aggressive management are crucial. Further studies are needed to elucidate the mechanisms and preventive strategies for chemotherapy-induced arrhythmias.

Keywords: Cardiotoxicity; Carboplatin; Docetaxel; Supraventricular Tachycardia.

Introduction

Cancer and cardiovascular disease are the leading causes of mortality in the world.[1] Cardiotoxicity is a significant complication due to chemotherapy. Cardiovascular events may consist in blood pressure changes, thrombosis, electrocardiographic changes, arrhythmias, myocarditis, pericarditis, myocardial infarction, cardiomyopathy, cardiac failure (left ventricular failure) and congestive heart failure. [2,3] Cancer treatment–induced arrhythmia (CTIA) arises from complex interactions between chemotherapeutic agents and cardiac tissue, involving direct effects on molecular pathways as well as structural injury from ischemia, inflammation, or radiation.[4]

Cardiotoxicity is classically associated with anthracyclines,[5,6] evidence linking platinum-based compounds remains limited. A MEDLINE database revealed four case reports describing cardiac toxicity attributable to carboplatin and more than 10 cases associated with cisplatin. [7] Cisplatin has been reported in a few cases to induce atrial fibrillation (AF) and supraventricular tachycardias (SVT). In contrast, carboplatin is rarely associated with arrhythmogenic effects, with only isolated case reports describing sinus arrhytmia in a patient with non-small cell lung cancer, atrial flutter in a patient with small cell lung cancer (SCLC) and third-degree heart block in a patient with asymptomatic ventricular ectopy.[4,8]

These rare instances highlight the importance of heightened clinical vigilance, particularly in patients with pre-existing cardiovascular disease or concurrent cardiac stressors undergoing chemotherapy. In this context, we present a case of reccurence SVT induced by chemotherapy carboplatin and docetaxel in a patient with nasopharyngeal carcinoma.

Case Illustration

ISSN:0048-2706 E-ISSN:2227-9199

A 57-year-old male presented to the emergency department in August 2024 with intermittent, non-radiating, left-sided chest pain that had worsened over the preceding 24 hours. The pain was not relieved by rest or positional changes. Associated symptoms included palpitations, nausea, and tingling, but there was no vomiting, syncope, or loss of consciousness.

His past medical history was significant for untreated hypertension (self-managed with herbal medicine), type 2 diabetes mellitus (treated with metformin 500 mg twice daily), and a prior ischemic stroke in 2018 that resulted in left-sided hemiparesis and central-type facial and hypoglossal nerve paresis. He was a smoker, and both parents had histories of hypertension and type 2 diabetes mellitus. He had no history of myocardial infarction. The patient was also a cancer survivor, with a diagnosis of nasopharyngeal carcinoma previously treated with six cycles of chemotherapy (carboplatin 494.15 mg and docetaxel 112.95 mg) completed in 2022, followed by surgical resection in 2023.

On arrival, his vital signs were as follows: blood pressure, 99/52 mmHg; heart rate, 191 bpm (regular); respiratory rate, 20 breaths per minute; SpO_2, 95%; temperature, 36.8°C; and random blood glucose, 116 mg/dL. Cardiac examination revealed an enlarged cardiac silhouette with normal first and second heart sounds, and no murmurs or gallops. Laboratory studies, including complete blood count, serum electrolytes, CK-MB, and CPK, were within normal limits. A chest X-ray confirmed cardiomegaly.

A strain echocardiogram performed earlier at a cardio-oncology clinic (August 2024) showed a preserved LVEF of 69%, but with a prolonged QTc interval of 480 ms and a ΔGLS of 23.8% (decreased <15%), indicating high-risk HFA–ICOS cardiotoxicity with evidence of subclinical myocardial injury.

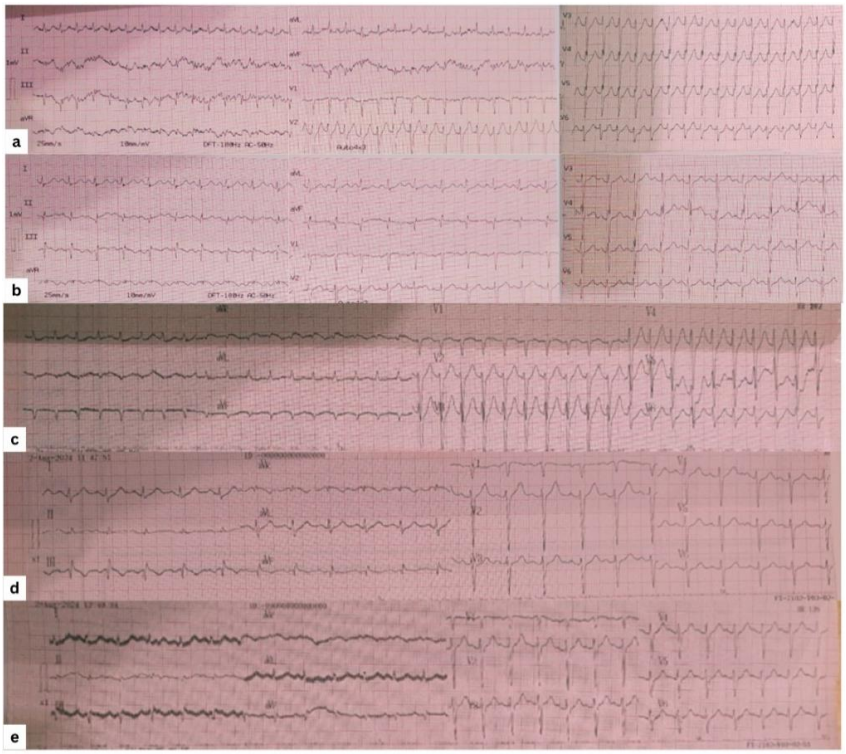

Figure 1. Collective ECG of patient

(a) Pre-cardioversion 1 in the emergency unit; (b) Post-cardioversion 1 in the emergency unit; (c) Pre-cardioversion 2 in the ICU; (d) Post-cardioversion 2 in the ICU; (e) Post-cardioversion 3 in the ICU.

ISSN:0048-2706 E-ISSN:2227-9199

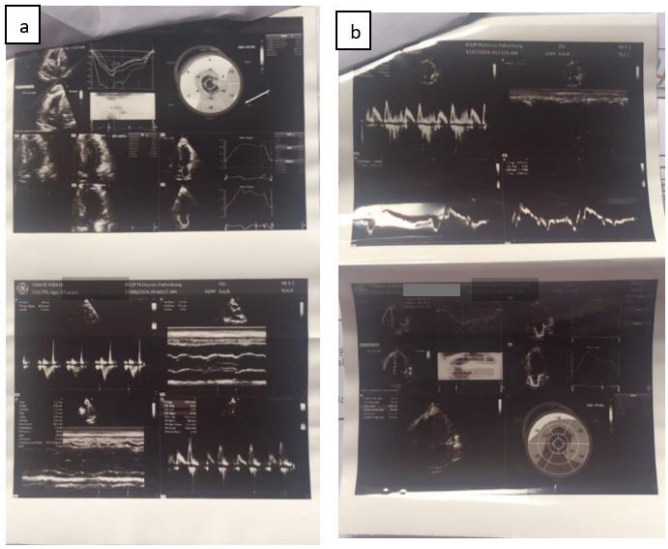

Figure 2. Patient's echocardiogram results
(a) Pre-chemotherapy; (b) Post-chemoterapy.

Electrocardiography on admission revealed an unstable SVT, for which the patient underwent immediate electrical cardioversion to sinus tachycardia, with ST-segment depression in leads V3–V4. Two hours after transfer to the intensive care unit, the patient lost consciousness and developed a recurrent episode of SVT, requiring a second cardioversion. A third episode of SVT occurred one hour later and was managed in the same way, with successful cardioversion.

The patient was started on supportive therapy, including a dobutamine infusion (1 ampoule in 50 mL normal saline at 5 cc/hour as needed), bisoprolol 5 mg once daily, clopidogrel 75 mg once daily, aspirin 70 mg once daily, and amiodarone 20 cc/hour for 6 hours followed by 10 cc/hour for 18 hours, in addition to his regular metformin 500 mg twice daily. The patient remained hemodynamically stable and was discharged after five days of inpatient care.

After one month, the patient underwent coronary angiography, which revealed a subtotal occlusion of the proximal left anterior descending artery and 30% stenosis of the distal left circumflex artery. The condition was managed successfully with percutaneous coronary intervention of the left anterior descending.

Discussion

The prevalence of cardiovascular risk factors and coronary artery disease in cancer patients is higher and is associated with increased mortality. Several mechanisms, such as the proinflammatory and procoagulant states present in cancer patients are contributing mechanisms. Furthermore, oncological therapy can predispose patients to acute thrombosis, accelerated atherosclerosis and coronary spasm. [1] Chemotherapy has been increasingly linked to cardiotoxic effects, with documented cases of myocardial infarction and arrhythmia triggered by several chemotherapeutic agents.[9] In this case, the patient had left chest pain followed by palpitations. These risk factors of hypertension, type 2 diabetes mellitus, smoking, stroke are stratified as high risk according to HFA-ICOS risk score. The patient also had a history of NPC that had gone through 6 cycles of carboplatin and docetaxel chemotherapy. These risk factors and chemotherapy experienced by the patient increase the risk of atherosclerosis and thromboembolism.

Carboplatin, a chemotherapeutic agent from the platinum-based class, has been shown to exert direct toxic effects on cardiac muscle, contributing to both acute and long-term cardiac complications. This effect caused by its interaction with cellular DNA, which may lead to significant genetic damage. When this damage is not adequately repaired, it can trigger apoptosis in myocardial tissue. Experimental studies, both *in vitro* and *in*

vivo, suggest that carboplatin-related cardiotoxicity is associated with mitochondrial dysfunction and an increase in reactive oxygen species (ROS), leading to considerable injury to heart muscle. Moreover, early vascular toxicity can develop either during or following chemotherapy, likely due to changes in protein kinase C isoforms, elevated TRPC1 channel expression, and activation of NF-κB. These molecular changes contribute to increased vascular permeability and albumin leakage, which have been linked to the severity of the cardiotoxic response. [10] In retrospective cohort study comparing cisplatin and carboplatin, 15.2% thromboembolic events from carboplatin group which included pulmonary embolism, cerebrovascular accidents, and myocardial infarction.[11]

A taxoid antineoplastic drug, docetaxel is frequently used to treat breast, nasopharyngeal, and prostate cancer. Docetaxel and Paclitaxel can induce cardiotoxicity in 2.3% to 8% of patients. Docetaxel works by converting tubulin into permanent microtubules and preventing their degradation. The quantity of free tubulin will be significantly reduced. This mechanism is responsible for the anticancer activity of docetaxel but can also alter the cardiovascular system. Apart from its direct toxicity to cardiomyocytes, docetaxel can also cause endothelial dysfunction and oxidative stress by increasing cell apoptosis.[12] Patients with existing cardiovascular risk factors are particularly vulnerable to myocardial ischemia. Additionally, arrhythmias such as SVT, AF, ventricular tachycardia, and various forms of bradyarrhythmia are frequently reported and can pose serious clinical concerns.[10]

Cancer treatment–induced arrhythmia (CTIA) is a multifactorial condition arising from a complex interplay of mechanisms. It is generally categorized into primary CTIA, caused by chemotherapeutic agents that disrupt specific molecular pathways essential for cardiac electrical conduction, and secondary CTIA, which results from structural injury to the endocardium, myocardium, or pericardium due to ischemia, inflammation, or radiation therapy. [13] Chemotherapy also can alter calcium homeostasis, as indicated by mechanisms such as altered calcium oscillations and calcium influx regulation, which can lead to automaticity disturbances and re-entry phenomena. This dysregulation is driven in part by oxidative stress. While the exact pathways through which these drugs trigger arrhythmias are still not clear.[14]

Treatment of arrhythmias is a consideration for clinical management of heart failure, myocardial infarction and cardiomyopathy.[14] In the treatment of SVT, acute therapy must consider hemodynamic conditions. In this case, hypotension indicated unstable hemodynamics, and synchronized cardioversion was performed. By synchronizing with the QRS complex, the shock is timed to avoid the T wave, reducing the risk of ventricular fibrillation caused by the R-on-T effect. Premature atrial or ventricular beats sometimes recur after conversion, which may trigger SVT. The use of anti-arrhythmic drugs may be considered to prevent re-initiation of tachyarrhythmias. Some studies suggest that first-line administration of calcium antagonists and beta blockers is effective and has minimal side effects. If unsuccessful, intravenous amiodarone may be given to restore sinus rhythm or slow the ventricular response in hemodynamically stable tachyarrhythmic patients. [12] Permanent treatment, such as ablation therapy, should be considered for arrhythmias with high success or cure rates, such as atrial flutter, AVNRT, AVRT, and atrial tachycardia. [11] In these cases, catheter ablation therapy is the best option for patients with medically refractory SVT, sustained SVT, or patients complicated by tachycardia-induced cardiomyopathy.

The limitation of this case report is that our patient had multiple other cardiovascular risk factors, which is a compounding factor. The absence of both an electrophysiology study and a coronary angiography study represent additional weaknesses. If recurrent episodes of SVT occur, it may be necessary to do further electrophysiology studies to evaluate the role of each conduction system component, pinpoint the cause of arrhythmia, assess the patient's level of risk, and establish the optimal course of therapy.

Conclusion

Combination of chemotherapy carboplatin and docetaxel as predictor myocardial infarction is a rare but fatal complication, especially in patient with previous risk factor for coronary artery disease. Physicians should be more aware of this side effect for prompt diagnosis and treatment, to prevent significant morbidity and mortality. More research is needed to determine the causes of arrhythmias brough on by different chemotherapy treatments and find ways to both prevent and treat them.

References

1. Costa IBS da S, Andrade FT de A, Carter D, Seleme VB, Costa MS, Campos CM, et al. Challenges and Management of Acute Coronary Syndrome in Cancer Patients. Front Cardiovasc Med. 2021;8.
2. Herrmann J, Lenihan D, Armenian S, Barac A, Blaes A, Cardinale D, et al. Defining cardiovascular toxicities of cancer therapies: an International Cardio-Oncology Society (IC-OS) consensus statement. Eur Heart J. 2022 Jan 21;43(4):280–99.
3. Reshadmanesh T, Mohebi R, Behnoush AH, Reshadmanesh A, Khalaji A, Norouzi M, et al. The effects of sodium-glucose cotransporter-2 inhibitors in chemotherapy-induced cardiotoxicity and mortality in patients with cancer: a systematic review and meta-analysis. Cardio-Oncology. 2025;11.
4. Laksono S, Kusharsamita H. Supraventricular Tachycardia Induced by Cisplatin in a Patient with Breast Cancer: A Case Report. Indones J Cancer. 2024 Jun 27;18(2):208–10.
5. Lyon AR, López-Fernández T, Lancellotti P, Thuny F, Abdelhamid M, Aboyans V, et al. 2022 ESC Guidelines on cardio-oncology developed in collaboration with the European Hematology Association (EHA), the European Society for Therapeutic Radiology and Oncology (ESTRO) and the International Cardio-Oncology Society (IC-OS). Eur Heart J Cardiovasc Imaging. 2022 Oct 1;23(10):E333–465.
6. Idrees I, Ahmad R, Nadeem M, Khan A, Gilani A, Khattak R. Antracycline Induced Early Onset Chronic Cardiotoxicity in Cancer Patients. Pak Armed Forces Med J. 2024 Dec 31;74(6):1492–5.
7. Khan S, Chen CL, Brady MS, Parameswaran R, Moore R, Hassoun H, et al. Unstable angina associated with cisplatin and carboplatin in a patient with advanced melanoma. J Clin Oncol. 2012 Jun 20;30(18).
8. Zakaria S, Lu KY, Nussenblatt V, Browner I. Atrial flutter associated with carboplatin administration. Ann Pharmacother. 2011;45(11).
9. Fradley MG, Beckie TM, Brown SA, Cheng RK, Dent SF, Nohria A, et al. Recognition, Prevention, and Management of Arrhythmias and Autonomic Disorders in Cardio-Oncology: A Scientific Statement from the American Heart Association. Circulation. 2021;144:E41–55.
10. Rachma B, Savitri M, Sutanto H. Cardiotoxicity in platinum-based chemotherapy: Mechanisms, manifestations, and management. Cancer Pathog Ther. 2024.
11. Kim GE, Ibrahim AR, Shalatouni D, Abouzeid NH, Othman F. Paclitaxel-induced acute myocardial infarction: a case report and literature review. BMC Cardiovasc Disord. 2024 Dec 1;24(1).
12. Bendahou H, Ettagmouti Y, Abouriche A, Maaroufi A, Haboub M, Arous S, et al. Cardiotoxicity Due to Docetaxel Rare But it Exists: About a Case and Literature Review. J Case Rep Med Hist. 2023 Jan 16;
13. Li W, Cheng X, Zhu G, Hu Y, Wang Y, Niu Y, et al. A review of chemotherapeutic drugs-induced arrhythmia and potential intervention with traditional Chinese medicines. Front Pharmacol. 2024;15.
14. Dai M, Chen Y, Qin J. Atrioventricular re-entrant tachycardia and atrioventricular node re-entrant tachycardia in a patient with cancer under chemotherapy: a case report and literature review. Front Cardiovasc Med. 2024;11.
15. Budhi Raharjo S, Yuniadi Y, Yansen I, Andina Munawar D, Yugo Hermanto D, Korespondensi dr Sunu Budhi Raharjo A, et al. Pedoman Tatalaksana Takiaritmia Supraventrikular (TaSuV). Indones J Cardiol. 2017;38(2):109–50.