Supraventricular Tachycardia Induced by Cisplatin in a Patient with Breast Cancer: A Case Report

Sidhi Laksono1,2*, Hillary Kusharsamita3
1 Department of Child’s Health, Wangaya General Hospital, Denpasar, Bali, Indonesia
2 Department of Clinical Pathology, Wangaya General Hospital, Denpasar, Bali, Indonesia
3 Pertamina Central Hospital, South Jakarta, Indonesia

*Corresponding author:
Sidhi Laksono
Faculty of Medicine, Universitas Muhammadiyah Prof Dr Hamka, Tangerang, Indonesia
sidhilaksono@uhamka.ac.id

INTRODUCTION

Cisplatin is very effective and frequently used to treat many human cancers. Cisplatin is a robust anticancer substance that is used in combination chemotherapy regimens. Despite its efficacy, cisplatin treatment is restricted due to renal and cardiac effects. Although there have been numerous evaluations on the renal toxicity of cisplatin, there have been very few studies on its cardiac toxicity effects [1].

Cardiotoxicity caused by cisplatin is uncommon, and its incidence is unknown. According to a study, cardiotoxicity was seen in 6% of patients receiving cisplatin combined with 5-fluorouracil (5-FU) and 1.6% of patients getting 5-FU alone [2].

Earlier research has shown that cisplatin treatment is linked to cardiotoxicity. Electrocardiographic alterations, arrhythmias, myocarditis, cardiomyopathy, and congestive heart failure are some of the cardiac events that have been recorded in several case reports [3]. Here we provide a case of a patient with no known cardiac history who had supraventricular tachycardia after receiving cisplatin as her breast cancer treatment.

A 38-year-old woman who presented to our hospital had been diagnosed as having left breast carcinoma, Stage IV, triple-negative luminal type. The patient underwent a biopsy which showed an invasive left breast carcinoma of no special type. The patient had normal blood pressure and no history of cardiovascular disease. The baseline electrocardiogram (ECG) shows normal sinus rhythm. She was scheduled to undergo chemotherapy. The chemotherapy protocol consisted of hydration with 500 cc of sodium chloride 0.9% for 6 hours, followed by premedication of dexamethasone, ranitidine, ondansetron, and diphenhydramine. Then, 260 mg of paclitaxel was given in 350 cc of sodium chloride 0.9% for 90 minutes, followed by 250 cc of sodium chloride 0.9% for 30 minutes. At this point, the patient hadn’t complained about anything or had any palpitations. The patient was then given 120 mg of cisplatin in 500 cc of sodium chloride 0.9% in 1 hour. The patient had palpitations during the cisplatin infusion cycle. Supraventricular tachycardia (SVT) was detected on an ECG (Figure 1).
Eventually, the patient’s palpitations subsided, and subsequent ECG showed a conversion to sinus rhythm (Figure 3). The patient was then able to complete the first treatment cycle without complication. The patient was given 5 mg of tab bisoprolol before each cycle, and since she had no arrhythmias during subsequent cycles, her chemotherapy protocol was not altered.

DISCUSSION

One of the most frequent malignancies in women is breast cancer. Cisplatin has had extensive clinical usage in the treatment of this disease. However, cisplatin has a wide range of adverse effects that prevent it from being used routinely, including nephrotoxicity, neurotoxicity, gastrointestinal side effects, and ototoxicity. Cisplatin cardiotoxicity includes electrocardiographic abnormalities, arrhythmias, myocarditis, cardiomyopathy, and congestive heart failure. Some arrhythmias observed during or shortly after cisplatin administration may be clinically significant and even life-threatening. These arrhythmias include supraventricular tachycardia, bradycardia, and block of any degree. However, despite its adverse effects, cisplatin is still given to patients with recurrent or metastatic cancer as part of their initial clinical therapy [4].

The mechanism of cisplatin-induced toxicity is intricate and incompletely understood. Research suggests that cisplatin’s cardiotoxicity can lead to LV dysfunction, slowed myocardial contractions, mitochondrial dysfunction, enhanced endoplasmic reticular stress, cell apoptosis, reactive oxygen species production, and inflammation [5]. Arrhythmias produced by cancer treatments may be classified as either primary (generated by a medicine interrupting certain molecular processes important for developing a specific arrhythmia) or...
secondary. Cisplatin’s toxicity to the heart might result from the drug’s direct toxic effect on cardiac myocytes. It can also result from the drug’s creation of reactive oxygen species (ROS), which induces oxidative stress and causes the heart to switch to a prothrombotic state. One study showed that sixty-seven percent of patients showed evidence of cisplatin-related acute arrhythmia with no reported symptoms. Researchers concluded that cisplatin’s direct effect on cardiac sodium channels leading to an increase in QT dispersion might cause inhomogeneity of ventricular recovery appears to be independent of changes in the blood electrolyte levels. However, endocardium, myocardium, and pericardium damage from ischemia, inflammation, or radiation therapy during cancer treatment can lead to arrhythmia as a secondary phenomenon. Secondary cancer treatment-induced arrhythmia is substantially more prevalent [6–8].

Because of the complexity of the disease and the lack of complete understanding of the processes by which many medications work, it is difficult to draw clear lines between arrhythmias that occur in primary or secondary to cancer therapy. In most studies, cardiac monitoring does not begin until after chemotherapy has started, making it impossible to distinguish between arrhythmias caused by the treatment and those that existed before it [8].

The SVT that occurred during cisplatin infusion in our patient, who had no prior history of cardiac disease, was stopped and amiodarone was administered intravenously. The levels of electrolytes were normal. His echocardiogram and electrophysiology tests showed no abnormalities. This patient’s SVT was ultimately attributed to the primary cardiotoxicity caused by cisplatin. The successive cycles of chemotherapy were also well tolerated by the patient.

The limitation of this case report is the absence of an electrophysiology (EP) study. If recurrent episodes of SVT occur, it may be necessary to do further EP 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.

CONCLUSIONS

Significant adverse effects of cisplatin treatment include cardiac toxicities. Chemotherapy drugs have been linked to arrhythmias like SVT; hence the ECG has to be monitored carefully. To rule out a secondary type of chemotherapy-induced arrhythmia, it is necessary to monitor the patient’s heart with an ECG and echocardiogram during chemotherapy. More research is needed to determine the causes of arrhythmias brought on by different chemotherapy treatments and find ways to both prevent and treat them. To assist in averting severe cardiac morbidity and death in these individuals, future cancer drug development paths should include tools to analyze the cardiac characteristics of these medications.

DECLARATIONS

Ethics approval and consent to participate
The authors declare that research ethics approval was not required for this study. Informed consent for the publication of patient information in a case report was obtained.

Competing interest
The authors declare no competing interest in this study

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REFERENCES