

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

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ABSTRACT

Introduction: Cisplatin is one of the most extensively used chemotherapeutic agents for treating many malignancies. Cisplatin chemotherapy, on the other hand, is linked to cardiotoxicity, which may vary from silent arrhythmias to heart failure to sudden cardiac death. In this article, we describe a case of supraventricular tachycardia induced by cisplatin in a breast cancer patient.

Case Presentation: Our patient had no history of heart disease and had SVT during cisplatin administration. This condition resolved when the infusion was stopped and amiodarone was administered intravenously. The electrolyte levels were within the usual range. No abnormalities were detected on her echocardiography imaging. Primary cardiotoxicity from cisplatin was subsequently determined to be this patient's root cause of SVT. The patient also had an excellent response to the subsequent cycles of treatment.

Conclusions: It is important to note that cisplatin therapy is associated with cardiac toxicity. Arrhythmias such as SVT have been associated with chemotherapy drugs. Hence the ECG has to be closely monitored during cisplatin administration. In addition, An ECG and echocardiogram should be done regularly to rule out the possibility of a secondary form of chemotherapy-induced arrhythmia.

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.

CASE PRESENTATION

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 hasn'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**).

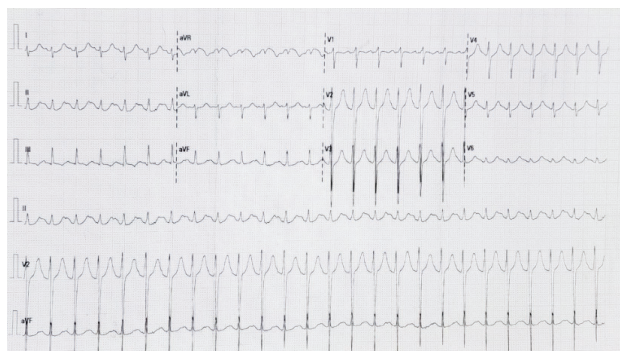


Figure 1. Electrocardiogram (ECG) showed a supraventricular tachycardia

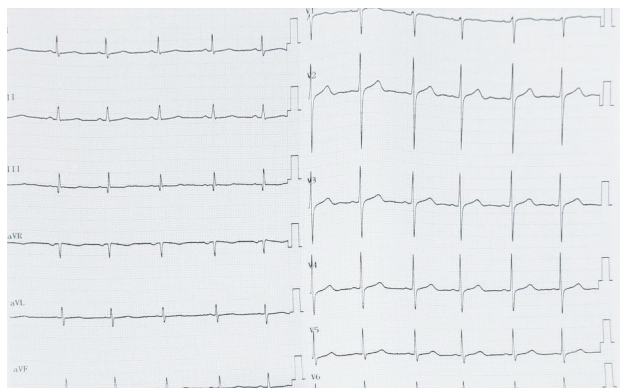


Figure 3. ECG showed a conversion to sinus rhythm

The patient's cisplatin infusion was discontinued, and 150 mg of amiodarone was administered intravenously as a bolus injection. In the meantime, blood samples were taken to check serum sodium, potassium, calcium, and magnesium levels, which were found to be normal. The echocardiography studies were performed subsequently, and the results are within normal limits, with an ejection fraction (EF) of 69.7 percent, with the rest parameter of interventricular septal end diastole (IVSd) and Interventricular septal end systole (IVSs), Left ventricular internal diameter end diastole (LVIDd) and Left ventricular internal diameter end systole (LVIDs), Left ventricular posterior wall end diastole (LVPWd) and Left ventricular posterior wall end systole (LVPWs), end diastolic volume (EDV), ejection fraction (EF), End systolic volume (ESV), Fractional shortening (FS), Interventricular septum (IVS), LV mass are all within the normal range (Figure 2).

Interventricular septal end diastole (IVSd) and Interventricular septal end systole (IVSs), Left ventricular internal diameter end diastole (LVIDd), and Left ventricular internal diameter end systole (LVIDs), Left ventricular posterior wall end diastole (LVPWd) and Left ventricular posterior wall end systole (LVPWs), end diastolic volume (EDV), ejection fraction (EF), End systolic volume (ESV), Fractional shortening (FS), Interventricular septum (IVS), LV mas

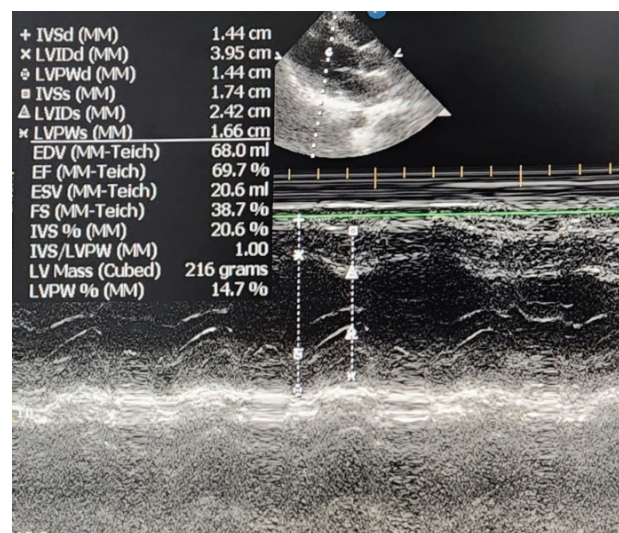


Figure 2. Echocardiography result

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 medication.

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

Acknowledgment

Not applicable.

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