Case Report

Acute Myocardial Infarction Following Carboplatin Therapy in a Patient with Nasopharyngeal Carcinoma: A Case Report

Mohd Nadzir bin Mohd Noor[1,2], Andey bin Rahman[1,2]

[1] Department of Emergency Medicine, School of Medical Sciences, Universiti Sains Malaysia, Health Campus, 16150 Kubang Kerian, Kelantan, Malaysia
[2] Hospital Pakar Universiti Sains Malaysia, Health Campus, 16150 Kubang Kerian, Kelantan, Malaysia

Abstract

Platinum-based chemotherapy agents such as carboplatin have been associated with rare but severe adverse cardiac events, including myocardial infarction (MI). Nasopharyngeal carcinoma (NPC), which responds well to chemoradiotherapy, presents unique cardiovascular challenges due to cancer-induced inflammation and thrombosis. We present the case of a 65-year-old woman with stage III NPC who developed acute inferolateral MI during concurrent carboplatin-based chemoradiotherapy. She presented with severe chest pain, ST elevation on ECG, and elevated troponin levels. Coronary angiography revealed multiple vesels disease, and prompt percutaneous coronary intervention (PCI) was performed. This case highlights the need for vigilance during carboplatin therapy, especially in high-risk patients. Cardiovascular screening and monitoring should be integrated into clinical care. Carboplatin-induced MI through multiple mechanisms, including direct myocardial injury, vascular disturbances, and electrolyte imbalances.

Keywords: *carboplatin-induced, myocardial infarction, carcinoma, adverse event*

INTRODUCTION

Chemotherapeutic agents have been implicated in cardiotoxicity, including the induction of myocardial infarction (MI). Chronic inflammation in cancer also predisposes patients to arterial and venous thromboembolism, including MI.[1] Nasopharyngeal carcinoma (NPC) is the most prevalent otorhinolaryngology malignancy in Southeast Asia and is associated with chronic proinflammatory tumors that promote a hypercoagulable state and increase the risk of thrombotic events. It is highly sensitive to both radiotherapy and chemotherapy.[2]

Carboplatin is a second-generation platinum-based chemotherapy that is advocated for use in the treatment of patients with NPC and ovarian cancer, and it has also shown promise in the treatment of small cell lung cancer.[3] Commonly documented adverse reactions include nausea, bone marrow suppression, nephrotoxicity, and neurotoxicity.[4] Cardiac complications from carboplatin are listed as rare or very rare in the drug's product summary. Kim et al. demonstrated that the incidence of arterial thrombosis was greater in a group of lung cancer patients treated with carboplatin than in a group treated with cisplatin.[4]

This case report aims to share a rare but important adverse event of acute MI during carboplatin-based therapy. Highlighting this case can help raise awareness about the need to monitor heart health in cancer patients, especially those with underlying risk factors, and add to the growing understanding of chemotherapy-related heart issues.

CASE PRESENTATION

A 65-year-old Chinese woman with a history of hypertension and hyperlipidaemia was diagnosed with stage III NPC in July 2022. She received induction chemotherapy with cisplatin and gemcitabine, which she completed by November. She then continued with a combination of carboplatin and radiotherapy, finishing her treatment plan in March 2023.

In January 2023, while undergoing chemoradiotherapy, she came to the emergency department (ED) with left-sided chest pain that felt tight and radiated to her neck and shoulders. She also

experienced nausea, vomiting, and palpitations. Her blood pressure was 170/96 mmHg, but her pulse (68/min) and oxygen saturation (98% on room air) were within normal limits. Her lungs were clear, and no abnormal heart sounds were noted. An electrocardiogram (ECG) was performed and revealed ST elevation in leads II, III, AVF, I, V5, and V6 (Figure 1).

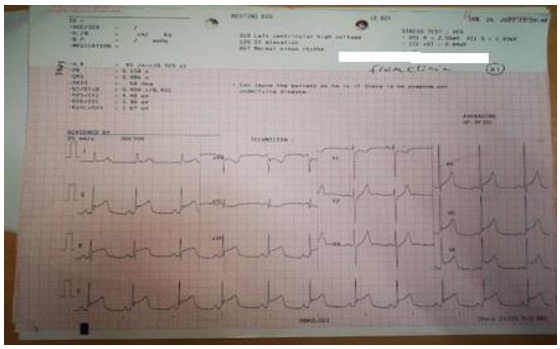

Figure 1: ECG at admission showing ST elevation in leads II, III, AVF, I, V5–V6.

The patient was given 0.5 mg sublingual glyceryl trinitrate, 300 mg aspirin and 300 mg clopidogrel at the ED. Her pain improved, and serial ECG revealed resolution of ST elevation (Figure 2). Her troponin T level was significantly elevated (1009 ng/mL), indicating myocardial injury.

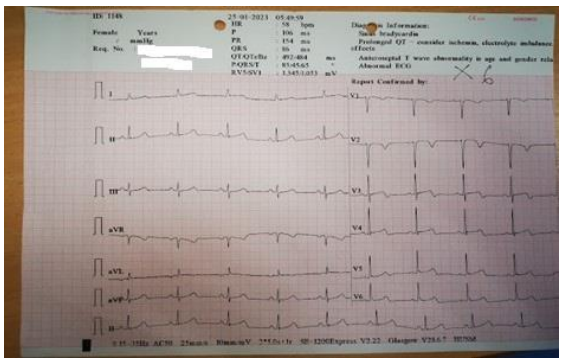

Figure 2: ECG was repeated after initial treatment, which revealed partial resolution of the ST elevation.

She was admitted for close monitoring and later underwent coronary angiography. Adhoc PCI was performed on the pLAD, mLAD and distal RCA (Table 1). The patient was diagnosed with inferolateral myocardial infarction with two-vessel disease. The patient was discharged home well.

DISCUSSION

Carboplatin-induced MI is rare but is a clinically significant complication that warrants careful monitoring due to its potential severity. Several case reports have documented cardiac toxicity associated with carboplatin-based regimens. Yano and Shimada described a case of angina occurring five hours postinfusion of carboplatin and etoposide in a patient with small cell lung cancer, notably in the absence of preexisting cardiovascular disease. Similarly, Cornillet et al. reported heart failure in an elderly patient with metastatic disease following six cycles of carboplatin combined with gemcitabine, highlighting the potential cumulative cardiotoxic effects of combination chemotherapy.

Table 1: Coronary angiography revealed critical stenoses in the LAD and RCA arteries.

LMS	Normal
LAD	pLAD 80-90% stenosis, mid 90-99%, D1 proximal 90% small vessel
LCX	Small OM1 Normal
RCA	Dominant, distal 90-99%
RPDA	Tortuous, Proximal 60-70%
IMP:	2 Vessel Disease Adhoc PCI to pLAD and mLAD Adhoc PCI to distal RCA

Naito and colleagues reported a patient who suffered from acute MI during carboplatin and gemcitabine therapy for non-small cell lung cancer. This case highlights a patient with NPC and preexisting cardiovascular risk factors who developed acute inferolateral MI during concurrent chemoradiotherapy with carboplatin. Several mechanisms, such as the proinflammatory and procoagulant states present in cancer patients, may contribute to these scenarios. Oncological therapy can also predispose patients to acute thrombosis, accelerated atherosclerosis and coronary spasm. Platinum base agents such as carboplatin may induce a prothrombotic state through several potential mechanisms, including reduced levels of endogenous anticoagulants, induction of TF (tissue factor) procoagulant activity, activation of the endothelium, endothelial cytotoxicity, and activation of platelets.[8]

Alterations in oxidative metabolism play important roles in the pathophysiological mechanisms underlying cancer and heart disease, especially in elderly patients. The concentrations of antioxidant enzymes are reduced in elderly subjects and may explain the reduced cardiac tolerance to oxidative stress, thus favouring the development of cardiovascular alterations.[1] The prompt initiation of antiplatelet therapy, glyceryl trinitrate, and subsequent percutaneous coronary intervention (PCI) is critical in stabilizing patients. Given the patient's preexisting hypertension and hyperlipidaemia, this case emphasizes the importance of baseline cardiovascular assessment before initiating carboplatin therapy. Clinicians should maintain a high

index of suspicion for cardiotoxicity and consider early diagnostic testing, including ECG and cardiac biomarkers, in symptomatic patients. Preventive strategies, such as cardiovascular risk assessment and optimization of modifiable risk factors, are critical for improving patient outcomes. This case adds to the growing recognition of the potential cardiovascular effects of carboplatin, emphasizing the need for further research and heightened clinical awareness.

CONCLUSION

Carboplatin-induced myocardial infarction can occur particularly in high-risk individuals, underscoring the need for proactive cardiovascular risk assessment and integrated monitoring strategies. The incorporation of diagnostic modalities such as echocardiography into routine clinical practice is essential for the early detection and management of acute cardiac events. Prospective studies are warranted to better define the incidence, mechanisms, and predictive factors of platinum-based chemotherapy–associated cardiotoxicity.

CORRESPONDENCE

Dr. Andey bin Rahman
MD (USM), MMED Emerg (USM)
Department of Emergency Medicine,
School of Medical Sciences,
Universiti Sains Malaysia,
16150 Kubang Kerian,
Kelantan, Malaysia
Email: andey@usm.my

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