

Abstract Form

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Abstract

Introduction

Cardiotoxicity is a potential adverse reaction of chemotherapy that can vary in its presentation from subclinical to overt cardiogenic shock. Additionally, there have been increasing reports of chemotherapy-associated posterior reversible encephalopathy syndrome (PRES), a rare diagnosis that shares some similar pathophysiology with stress-induced cardiomyopathy. Studies have demonstrated that early detection and management of these diagnoses promote favorable clinical outcomes. We report a patient presenting with PRES and new-onset cardiomyopathy after treatment with vincristine, doxorubicin, cyclophosphamide, ifosfamide, etoposide (VDC/IE) and a novel dual BET-CBP/p300 inhibitor.

Case Report

A 31-year-old male with metastatic NUT carcinoma of the sinuses presented to the hospital with new-onset seizure. Prior treatment included 7 cycles of multiagent chemotherapy with vincristine, doxorubicin, and cytoxan alternating with ifosfamide and etoposide (VDC/IE) completed five months prior to admission, and followed by a novel dual BET-CBP/p300 inhibitor. His initial hospital course was complicated by elevated blood pressures >180/120 and another episode of recurrent seizure. Neurology was consulted, and he was started on IV Keppra 1000mg twice daily which was subsequently increased to 1500mg twice daily. Initial MR brain without contrast showed patchy generalized areas of FLAIR hyperintensity involving the gray matter and subcortical white matter representing post ictal changes. MR brain with contrast showed multifocal areas of T2/FLAIR signal abnormality within the subcortical and to a lesser degree the cortex of the bilateral frontal lobes, parietal lobes, occipital lobes, and temporal lobes consistent with PRES. CSF studies were negative for malignant cells. The patient continued with anti-epileptics and started on anti-hypertensive medications without further seizure activity. Echocardiogram noted newly reduced left ventricular ejection fraction (LVEF) of 35-40%, global hypokinesis of his left ventricle, and grade I diastolic dysfunction, decreased from pre-chemotherapy LVEF of 55-60%. He was gradually started on guideline directed medical therapy (GDMT) with a beta-blocker, mineralocorticoid antagonist, sodium-glucose cotransporter-2 (SGLT2) inhibitor, and angiotensin receptor blocker prior to discharge. GDMT titration was limited by hypotension. A month later, a repeat echocardiogram showed improvement in the LVEF to 50-55%, suggestive of a component of reversible stress induced cardiomyopathy, likely in a background of anthracycline-induced cardiomyopathy. Given improvement in LVEF to >50%, patient was deemed stable from a cardio-oncology standpoint to resume anthracycline therapy with dexrazoxane, ongoing GDMT and a statin for cardioprotection along with frequent cardiac monitoring. He is also scheduled for repeat brain imaging to assess for resolution of PRES.

Discussion

This case report demonstrates the risk of cardiotoxicity from multiple possible pathophysiologic mechanisms in advanced cancer patients. This underscores the importance of close cardiac monitoring in patients receiving chemotherapy, especially given its broad spectrum of clinical presentation and in light of the need for early GDMT in the management of such patients.