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Case Report

Could Diabetes Increase The Likelihood of Cystic Macular Edema in Patients Treated with Paclitaxel?: A case report

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Abstract

A case of cystoid macular edema (CME) induced by paclitaxel in a diabetic patient. A 65-year-old female diagnosed with breast cancer presented with decreased vision in both eyes one week after cessation of paclitaxel. Ophthalmological examination revealed decreased visual acuity OU and asymmetrical CME with no signs of diabetic retinopathy. Optical coherence tomography and Fundus Fluorescein Angiography findings were consistent with Taxane-associated CME. Paclitaxel use in Diabetic patients might lead to increased susceptibility to Taxane-induced CME. Oncologists and Ophthalmologists should work closely to ensure early diagnosis and effective patient care.

Keywords: Taxane, Diabetes, Cystic macular edema, Oncology, Toxicology

INTRODUCTION

Taxane-based chemotherapy is widely used for breast cancer and can significantly improve the progression-free as well as overall survival of patients (1). Taxane-induced CME is an extremely rare complication, and while there are numerous case reports, the incidence has not been determined (2). Diabetes is also a common cause of CME, but the pathogenesis is believed to be different. FFA findings can be used to distinguish between the two. We report a case of Taxane-induced CME with concomitant diabetes and discuss whether our approach to these patients should differ.

CASE

A 65-year-old female patient with breast cancer presented to the ophthalmology clinic with decreased vision. Her past medical history was significant for diabetes and ischemic coronary artery disease. When the patient's tests were examined, it was seen that her hemoglobin A1c value was 7%. The patient was using oral antidiabetic and anticoagulant medications.

She was diagnosed with breast cancer six months ago, and her pathology report was consistent with luminal B type T1N1 invasive ductal carcinoma. Adjuvant 80 mg/m2 paclitaxel for 12 weeks was started 13 weeks before applying to the ophthalmology clinic. Best-corrected visual acuity was 20/200 OD and 20/40 OS, her anterior segment examination was normal, and dilated fundoscopy revealed no signs of diabetic retinopathy or any other retinal pathology. Optical coherence tomography (OCT) revealed asymmetric CME, and fluorescein angiography showed no capillary leakage and absent late hyper

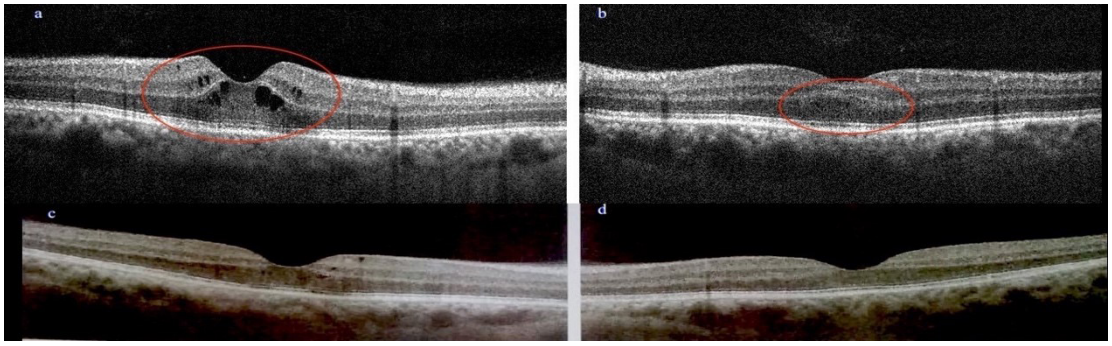

Figure 1. Optic coherence tomography of right and left eye showing cystic macula oedema at presentation (red circles indicate cystic changes) (fig a,b) ; Decrease in cystic oedema in both eyes at follow-up visit (fig. c,d).

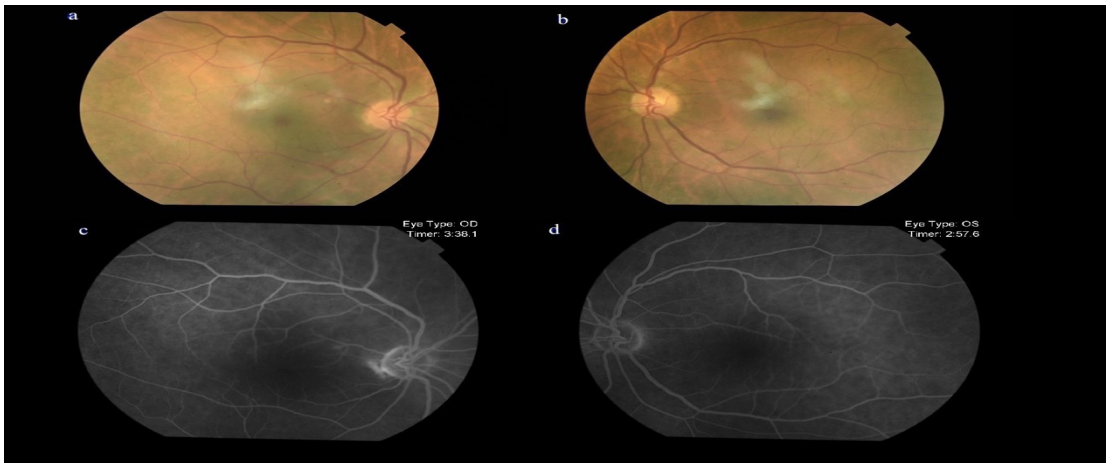

Figure 2. Fundus photo of the right and left eye of the patient (fig. a,b); Shows late-stage Fundus Fluorescein Angiography with no leakage (fig. c,d)

fluorescence. OCT and FFA findings were typical for Taxane-induced CME (**Figure 1a, b**) (Figure 2) As the patient had already completed her chemotherapy she was followed up. Two weeks later at the follow up visit the patient's vision had increased to 20/25 and 20/20 with improvement of the oedema (**Figure 1c, d**) Informed consent was signed by the patient.

DISCUSSION

Several studies have been reported examining the OCT and angiographic features of taxane-induced CME, its possible pathogenesis, and treatment options (3,4). Therapeutic options that have been proven effective suggest a noninflammatory etiology, and vascular endothelial growth factor (VEGF) has also been shown to be ineffective, clearly distinguishing this CME from diabetes-induced CME. The chemotherapeutic effect of taxane is mediated through disruption of microtubule function, which is believed to cause retinal pigment epithelium (RPE) dysfunction, possible Müller cell dysfunction, and aquaporin dysfunction at the intermediate and deep capillary plexus (DCP) levels of the retina, resulting in leaky CME (5).

Regarding the relationship between taxane-associated CME and diabetes we could find no previous report describing such a patient. Paclitaxel has been shown to increase the severity and duration of diabetic neuropathy (6). Diabetic neuropathy, diabetic retinal neuropathy and diabetic retinopathy are strongly linked (7). Even before signs of retinopathy appear, we know that diabetes affects the retina (8).

OCTA imaging of patients with diabetes without retinopathy showed impairment in all three layers of the capillary plexus (9). As taxane-induced CME is also believed to affect the intermediate and DCP, theoretically we would expect to see an increased likelihood of taxane-induced CME in Diabetic patients (10).

CONCLUSION

The effect of taxane in the retina of diabetic patients can possibly be seen at lower dosages and earlier in the treatment cycle. In our study the asymmetric presentation and relatively rapid recovery could be due to increased susceptibility to Taxane in diabetics i.e., as the retina is more susceptible even in the contralateral eye a mild form of oedema was present, and as the retina is more sensitive to the drug, withdrawal leads to rapid decrease in the oedema. Oncologists and ophthalmologists should be aware of a possible association between diabetes and Taxane-associated CME. Further studies examining this association and dosage effects on macular thickening could shed further light on this matter.

DECLERATIONS

Ethics Committee Aproval: Not available
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