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Occam Versus Hickam: Irreversible Vision Loss From Paclitaxel-Induced Cystoid Macular Edema

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Abstract

Purpose: To describe a case of cystoid macular edema (CME) caused by paclitaxel, a first-line chemotherapeutic for invasive breast cancer. **Methods:** A single case was presented. **Results:** CME developed shortly after initiation of paclitaxel; 7 months after discontinuation, improvement was seen. However, the patient's visual acuity never recovered. Changes consistent with macular telangiectasia, which may have been potentiated by paclitaxel toxicity, were seen on multimodal imaging. **Conclusions:** This is the first reported case of irreversible vision loss after paclitaxel treatment without evidence of phototoxicity. Paclitaxel may exacerbate underlying macular telangiectasia, resulting in ellipsoid zone loss after recovery from CME.

Keywords

paclitaxel, cystoid macular edema, macular telangiectasia, multimodal imaging, drug toxicity

Case Report

A 61-year-old woman presented with progressive vision loss of 6 months duration that eventually limited her ability to drive. Her medical history included stage 4 breast cancer notable for new metastases on surveillance positron emission tomography, which led to the initiation of nab-paclitaxel 2 months before the onset of blurry vision. She had no previous ocular history or ocular imaging. At initial presentation, the patient had received 13 total cycles of paclitaxel. Her visual acuity (VA) was 20/70 OD and 20/100 OS, with intraocular pressures of 17 mm Hg in both eyes. A fundus examination showed trace macular pigmentary changes in both eyes, and bilateral subfoveal cystoid cavitations, ellipsoid zone (EZ) disruption, and retinal pigment epithelium (RPE) irregularity were seen on optical coherence tomography (OCT) (Figure 1, A–B).

Included in the patient's differential diagnoses were paclitaxel-induced CME, type 2 macular telangiectasia, bilateral retinal vein occlusions, cancer-associated retinopathy (CAR), and late presentation of an inherited retinal disease. Ancillary testing was performed, including fundus autofluorescence (FAF), fluorescein angiography (FA), OCT angiography (OCTA), and electrophysiology. FAF showed hyperautofluorescence of the macula in both eyes, while FA showed no significant leakage in the right eye and temporal petaloid leakage of the macula in the left eye (Figure 1, C–D). OCTA showed a normal superficial plexus, but trace capillary dropout was seen in the deep plexus of the right eye, and trace capillary dilation was seen temporally in the left eye (Figure 1E). Full-field electroretinography (ERG) was normal, and a mild reduction in foveal amplitude was seen on multifocal ERG that was limited by eccentric gaze artifact (Figure 1F). Collectively, these findings did not support an inherited retinal disease, CAR, or previous bilateral retinal vein occlusion. In addition, OCTA and FA did not show venous dilation, telangiectatic vessels, or right-angle branching suggestive of overt type 2 macular telangiectasia.[1,2]

CME was thought most likely to be caused by paclitaxel. The medication was therefore discontinued and switched by her oncologist to capecitabine, with close follow-up. Over the next 7 months, serial OCT imaging showed a decrease in the number and size of cystoid cavitations but persistent disruption of the EZ that correlated with a continued decline of VA to 20/200 OD and 20/400 OS (Figure 2). The patient denied interest in further genetic testing.

Conclusions

This case is among the first to show irreversible severe and rapid vision loss and structural damage after stopping paclitaxel. Only 1 other case report has documented irreversible vision loss after paclitaxel-induced CME, thought to be due to photosensitivity that led to retinal phototoxicity from light exposure during pars plana vitrectomy.[3] Evidence supports drug-mediated CME, including angiographically silent CME in

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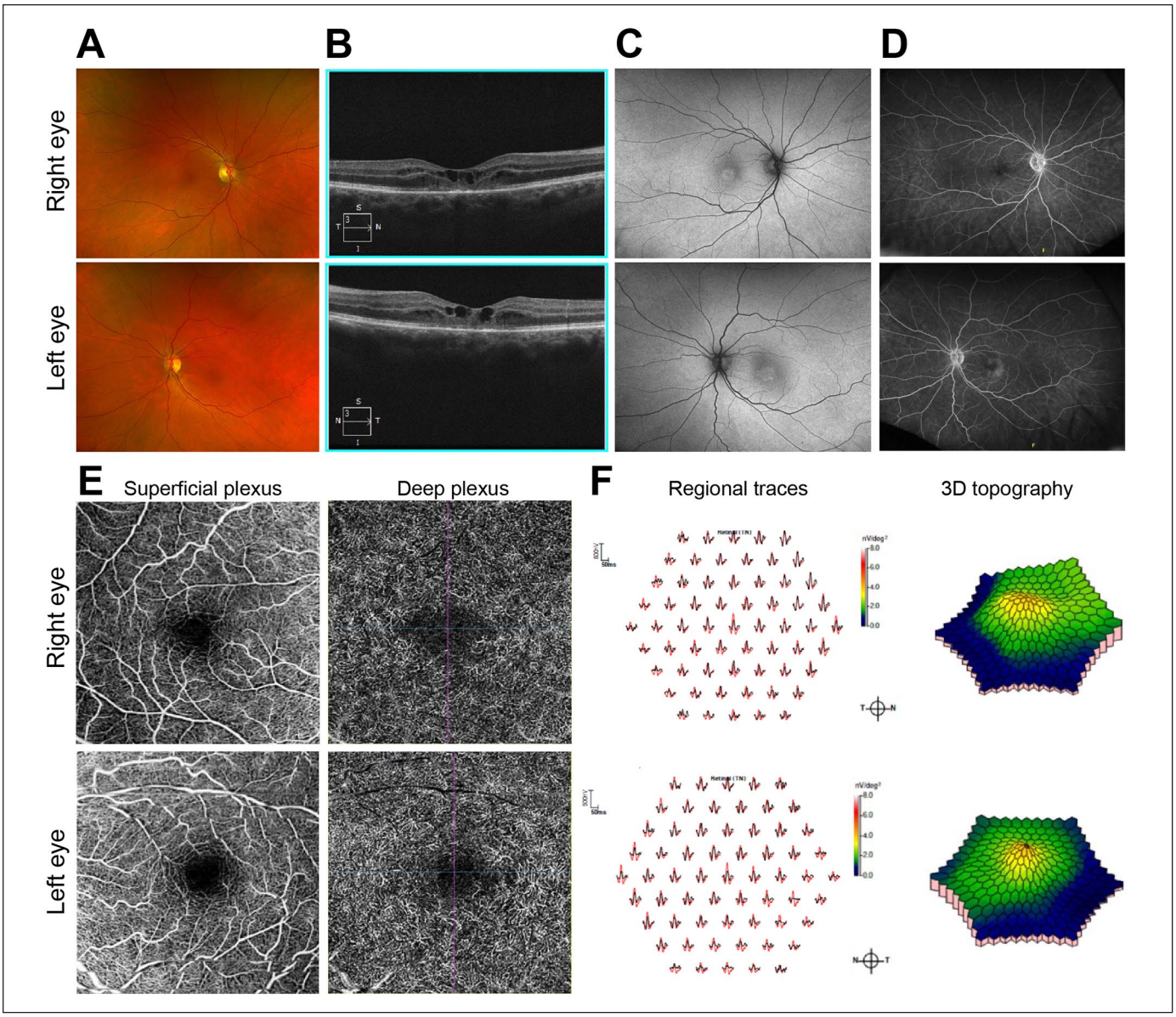

Figure 1. Multimodal imaging for evaluation of potential drug-induced cystoid macular edema. (A) Fundus photographs from the patient's initial visit. (B) Optical coherence tomography (OCT) of the macula. (C) Fundus autofluorescence. (D) Late recirculation phase of fluorescein angiography. (E) OCT angiography of the superficial and deep plexus. (F) Multifocal electroretinography shows regional traces and 3-dimensional topography of (top) the right eye and (bottom) the left eye.

the right eye, a classic imaging finding in paclitaxel-induced CME.[4,5] Furthermore, the cystoid cavitations seen on initial OCT imaging decreased in size and number after paclitaxel was stopped, a finding not expected in macular telangiectasia. However, the persistent EZ disruption and reduced vision is not typical of paclitaxel-induced CME.

An alternative diagnosis includes type 2 macular telangiectasia, which is supported by temporal petaloid leakage in the left eye, capillary abnormalities on OCTA, and bilateral macular hyperautofluorescence.[2,6,7] However, the relatively rapid and severe vision loss seen in this patient is atypical, because macular telangiectasia normally has a slow progression. One study found only 17% of patients progressed more than 2 lines over 5 years of follow-up.[8] Such severe progression in vision loss would be surprising given the lack of late-stage findings of macular telangiectasia, such as subretinal neovascularization or RPE migration.

One potential explanation that combines these findings is a 2-hit hypothesis. Macular telangiectasia is a neurodegenerative disease characterized by loss of Müller glia, to which paclitaxel is thought to be toxic.[9,10] Therefore, it is possible that paclitaxel exacerbated this dysfunction in a patient with underlying macular telangiectasia, causing rapid and progressive vision loss despite only early signs on imaging. Another consideration is that our patient could have an unrecognized or early stage inherited retinal disease that was worsened by paclitaxel. Genetic testing was denied by our patient, and no previous imaging was done to rule out preexisting pathology.

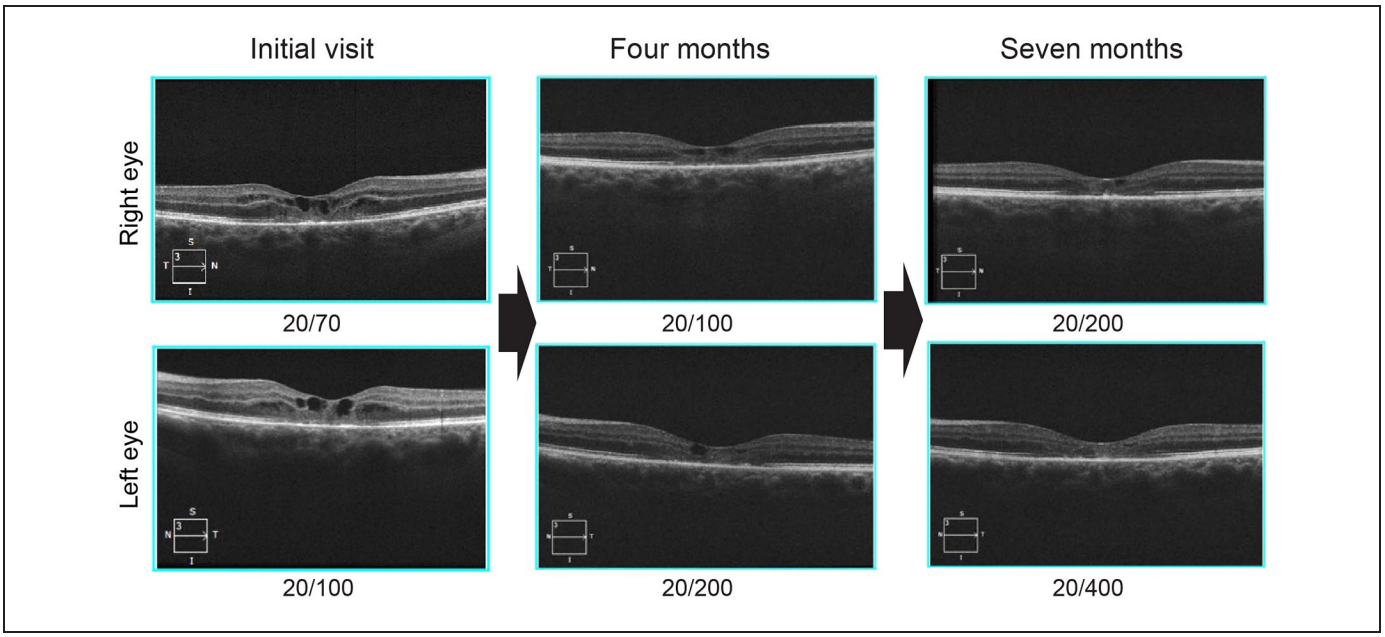

Figure 2. Serial macular optical coherence tomography (OCT) images from the patient's initial visit and at 4 months and 7 months after stopping paclitaxel. The visual acuity from each visit is shown below the OCT image.

This case highlights the potential severe and irreversible complications of paclitaxel-induced CME and emphasizes the importance of early recognition and discontinuation of the drug. Further research into retinal cell-specific toxicities is needed before initiating medication to guide patient counseling in addition to potential screening for underlying ocular disorders, such as macular telangiectasia.

Ethical Approval

Ethical approval was not sought because this paper is a case report.

Statement of Informed Consent

The patient verbally approved being included in this study, which was documented.

Declaration of Conflicting Interests

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of the article.

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