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Ocular Neurotoxicity Caused by Paclitaxel: A Case Report (Experience of the Oncology-Radiotherapy Department of the Mohammed VI University Hospital in Marrakech)

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Abstract **Case Report**

Paclitaxel (PTX) and/or cisplatin (CDDP), as important cytotoxic anticancer agents, are widely used to treat various solid tumors. Both may cause moderate or severe neurotoxicity, but ocular neurotoxicity is also occasionally reported. We report a case of a 78-year-old female patient being treated for metastatic right breast cancer involving the bone and lung. She received second-line chemotherapy consisting of weekly paclitaxel. After the fourth cycle, the patient reported blindness in the left eye and blurred vision in the right eye. Brain MRI did not reveal any lesions in the cerebral parenchyma other than bony lesions of the skull vault. An ophthalmological examination was performed and was normal. The patient was treated with glucocorticoids (methylprednisolone), calcium channel blockers (flunarizine), vasodilators, and neurotrophic agents over the following 2 weeks, but her vision did not improve in the 6 months following chemotherapy, and she had a very poor quality of life.

Keywords: Paclitaxel, neurotoxicity, ocular, chemotherapy.

INTRODUCTION

Paclitaxel is an antineoplastic agent belonging to the taxane family, widely used in the treatment of many solid tumors, particularly breast cancer, including metastatic forms. Its mechanism of action is based on the stabilization of microtubules, leading to the inhibition of mitosis and tumor cell apoptosis [1].

Despite its proven efficacy, paclitaxel administration is associated with several adverse effects, including peripheral neurotoxicity, which is one of the most common and dose-limiting complications [2].In contrast, taxane-related neuro-ophthalmological manifestations are rare but potentially severe, including optic neuropathies, maculopathies, visual field defects, and decreased visual acuity that can progress to irreversible vision loss [3,4].

The pathophysiology of this ocular toxicity remains poorly understood. Several mechanisms have been proposed, including microvascular damage leading to ischemia of the optic nerve or retina, as well as a direct neurotoxic effect on retinal neuronal cells [5].

Given the rarity of this complication and its potential impact on the quality of life of patients with metastatic breast cancer, early recognition of visual symptoms and its potential impact on the quality of life of patients with metastatic breast cancer. The literature reports a limited number of clinical observations, highlighting the importance of documenting each new case to improve understanding of the pathophysiological mechanisms and optimize therapeutic management.

We report here the case of a patient being treated for metastatic breast cancer who developed ocular neurotoxicity after four cycles of paclitaxel, illustrating the need for increased clinical vigilance in the presence of any visual symptoms occurring during taxane therapy.

CASE REPORT

We report the case of a 78-year-old Moroccan woman, a widow and mother of three children, who had been under follow-up since 2017 for an invasive ductal carcinoma of the right breast, initially classified as T2N1M0, treated with mastectomy, chemotherapy consisting of 3 cycles of AC60 and 3 cycles of docetaxel, followed by adjuvant radiation therapy.

The patient remained under observation with a good clinical and biological response. In June 2025, a follow-up CT scan revealed right pulmonary nodules with secondary lesions on bone scintigraphy.

She was then started on palbociclib at a dose of 125 mg daily for 21 days, followed by a 7-day break. Three months later, the patient's lung disease progressed while on palbociclib.

Following this progression, she was switched to palliative chemotherapy consisting of weekly paclitaxel at a dose of 80 mg/m² administered intravenously.

After the fourth cycle, the patient reported sudden total blindness in her left eye, along with blurred vision in her right eye.

A brain MRI was performed, which revealed no evidence of damage to the brain parenchyma other than bone lesions in the skull vault.

Several ophthalmic examinations were performed after the patient became blind; the best-corrected visual acuities were 0.3 on the right and only slight light perception on the left. The intraocular pressures were normal bilaterally at 11 mmHg on the left and 13 mmHg on the right. The pattern visual-evoked potentials (VEPs) and transient, flash electroretinograms (ERGs) were abnormal.

Non-specific waves were found in his left eye, but the VEP and ERG were normal in his right eye. The direct ophthalmoscopic examination of the fundus, retina, and vitreous humor was normal bilaterally, the left eye visual field could not be assessed due to its loss of light perception. A temporal hemianopsia, and a nasal peripheral visual field defect were found in the right eye. The retinal angiography of the left eye was normal (Figure 1)

Numerous therapies were administered, including glucocorticoids (methylprednisolone), calcium antagonists (flunarizine), vasodilators, and neuro-nutrition drugs over the following 2 weeks, but his sight did not improve in the 4 months following adjuvant chemotherapy, and he had a very poor quality of life.

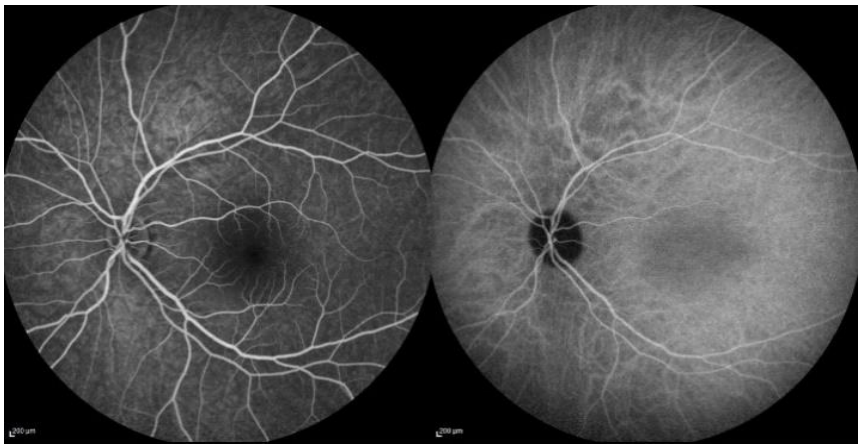

Figure 1 : Retinal Angiography

DISCUSSION

Ocular toxicity by paclitaxel is little known by both ophthalmologists and oncologists. It is a chemotherapeutic agent that inhibits the formation of intracellular microtubules, and whose ophthalmological side effects often include low visual acuity, scintillating scotomas and changes in evoked visual potential. Some articles have suggested the association of the drug to the onset of cystoid macular edema [6,7].

Chemotherapeutic agent-induced ocular neurotoxicity is commonly associated with interferon [8]. Ocular neurotoxicity generally presents as 1) blepharitis and conjunctivitis; 2) hemianopia, homonymous or bi-temporal visual field defects; 3) periorbital edema with firm eyelid swelling and ocular pain; 4) retinal toxicity with maculopathy characterized by pigmentary changes resulting from localized retinal pigment disturbances, altered color perception attributable to cone dysfunction, or mild retinal ischemic changes such as cotton-wool spots and posterior pole intraretinal hemorrhage; and 5) some other rare ocular toxicities [7,9,10].

Paclitaxel-induced neurotoxicity has been reported previously [3]. The patients typically complained of a transient and scintillating scotoma,

visual impairment, or bilateral cystoid macular edema. The majority of these symptoms occurred within the first 30 minutes of drug administration and completely resolved within 3 hours. These symptoms occurred more frequently in patients who received doses greater than 250 mg/m^2 [11,12]. Tan *et al.,* [13] described two patients with breast and ovarian cancer who suffered ocular toxicity after the administration of PTX and carboplatin. Though the underlying causes remained unknown, the authors surmised that PTX was the primary agent. Bakbak et al[14] assessed CDDP- and PTX-associated toxicities in the optic nerve by measuring the retinal nerve fiber layer (RNFL) thickness and visual field changes in patients with lung cancer who received systemic CDDP and PTX. They found the peripapillary RNFL thicknesses and visual field indices changed based on frequency-doubling technology (FDT) perimetry.

The mechanism of ocular neurotoxicity remains unknown, and neither the ischemic nor electrophysiological hypotheses could fully explain the pathogenesis of Paclitaxel (PTX) in the previous study [15]. A study by Scaioli et al [16] suggested that the visual symptoms and electrophysiological changes following intravenous PTX administration were likely caused by retinal vascular dysregulation or optic nerve ischemia. Because the cystoid macular edema occurred after treatment of PTX, one theory is that Müller cell toxicity results subsequent to intracellular fluid accumulation and subclinical extracellular fluid leakage. In patients with reversible scotoma, the abnormal visual-evoked potential was comparable to those with changes observed in ischemic neuropathies, which suggested that the target of anti-cancer drugs such as PTX was within the optic nerve [3].

Oncologists should take all potential severe toxicities into consideration prior to initial treatment and be familiar with their pathogenesis and management. Despite the lack of consensus outlining routine ophthalmologic monitoring and ocular toxicity management at present, for some drugs with potential ocular toxicity a baseline ophthalmologic examination and regular monitoring is strongly recommended before treatment.

The monitoring examinations could include visual acuity, tonometry, fundoscopy, color vision test, automated perimetry, retinal photography, and others [13]. Potential treatment options including warm compress, eyelid hygiene, corticosteroids, topical anti-inflammatory medications and lubricants, and avoiding light exposure may prove useful, but further clinical study is needed. Hofstra et al [6] reported that flunarizine, a selective calcium channel antagonist, has been used successfully to treat one patient diagnosed with visual-evoked potential abnormalities. Kwan et al [9] reported a case of bilateral panretinal laser photocoagulation, but the therapy was ineffective because the best-corrected visual acuity remained unchanged 6 months later. Based on previous reports, nearly all PIONs (Paclitaxel Induces Ocular Neurotoxicity) were irreversible, and the best-corrected visual acuity failed to improve significantly. Currently, there is no biomarker available to effectively predict these severe PIONs.

Moreover, also the measurement of TIN (Taxane Induces Neuropathy) has been different in the various trials. In the majority of phase III trials conducted on metastatic and adjuvant breast cancer patients, TIN was quantified using the National Cancer Institute's Common Terminology Criteria for Adverse Events (NCICTCAE) [17], [18].

Other measures, like the EORTC (European Organization for Research and Treatment of Cancer) CIPN-20 have been widely utilized [19]. Recently, a new tool to quantify signs and symptoms of TIN can be used: the Total Neuropathy Score (TNS) [36,37] (Table 1). Another way to assess the grade of neuropathy is the short 11-item FACT/GOG-Ntx subscale [20]. Evaluating neurotoxicity with quality-of-life tests (such as FACT-Taxane) can cause inevitable bias due to the other side effects of taxane therapy. It is difficult to objectively discriminate the grade of severity of TIN, and in few reports, patients have undergone electrophysiological evaluation.

CONCLUSION

Paclitaxel induced ocular toxicity is extremely rare and usually irreversible. The clinical manifestations mainly include abnormal vision, hemianopia, photopsia, visual disturbance, blindness, ocular pain, cystoid macular edema, and other similar symptoms. The possible mechanisms of PION might be related to the ischemic and electrophysiological changes within ocular neural structures. There are no effective strategies for the early detection, treatment, and prediction of PION, which is warranted for further investigation. Clinical oncologists should consider the risk of severe PION prior to initiating anti-cancer treatment.

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