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CASE REPORT PEER REVIEWED | OPEN ACCESS

Multimodal imaging in a case of acute erdafitinib-induced maculopathy

Nicholas Hallman, Joby J Tsai, Drew Bawcombe, Heather B Seith, Arash Maleki, Shailesh Gupta

ABSTRACT

Introduction: To analyze multimodal imaging findings in a patient with erdafitinib-induced maculopathy.

Case Report: An 80-year-old male presented to our retina clinic with decreased vision in his left eye that began two weeks prior to his most recent visit. His past ocular history included epiretinal membranes in both eyes. He had been diagnosed with bladder cancer and had been undergoing aggressive chemotherapy, which was discontinued two months prior to this visit due to serious adverse effects. One month before the most recent visit, his oncologist had initiated treatment with erdafitinib. His best corrected visual acuity was 20/50 in the right eye and 20/100 in the left eye. Fundus examination revealed macular pucker, mild diabetic retinopathy, and mild hypertensive retinopathy in both eyes. Optical coherence tomography imaging of the macula showed new onset subretinal fluid in both eyes. Fluorescein angiography and indocyanine green angiography of the retina and choroid did not show any abnormalities explaining the subretinal fluid.

Conclusion: Erdafitinib can cause toxic maculopathy with subretinal fluid on optical coherence tomography of the retina with no abnormalities on fluorescein angiography and indocyanine green angiography.

Keywords: Erdafitinib, Fibroblast growth factor receptors, Subretinal fluid, Toxic maculopathy

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INTRODUCTION

Immunological therapies have become increasingly effective and widely used in treating various types of cancer in recent years. Dysregulation of fibroblast growth factor receptors (FGFRs), a type of receptor tyrosine kinase signaling pathway, has been observed in various cancer cell types. Fibroblast growth factor receptor inhibitors (FGFRIs) are a class of targeted immunotherapies designed to block this receptor and its associated pathway [1].

Erdafitinib, a FGFRI, can cause serious eye problems. These include dry eye, inflammation of cornea, and retinal problems. Becker et al. reported a case of erdafitinib-induced mitogen-activated protein kinase inhibitor-associated retinopathy [2].

In this case report, we analyzed multimodal imaging findings in a patient with erdafitinib-induced maculopathy.

CASE REPORT

An 80-year-old male, an established patient, presented to our retina clinic with decreased vision in his left eye (OS) that began two weeks prior to his most recent visit. His past ocular history included epiretinal membranes in both eyes (Figure 1). His past medical history was significant for hypertension, diabetes, and bladder cancer. He had been on long-term treatment with losartan and metformin for hypertension and diabetes, respectively, both of which were well controlled. Additionally, He had been undergoing aggressive chemotherapy for bladder cancer for six months, which was discontinued two months prior to this visit due to serious adverse effects. One month before the most recent visit, his oncologist had initiated treatment with erdafitinib, an immunogenic biologic agent, for his bladder cancer. At this visit, patient's best corrected visual acuity (BCVA) had decreased to 20/50 in the right eye (OD) from 20/25, and to 20/100 in the left eye (OS) from 20/40, compared to the penultimate visit. Intraocular pressure at this visit was 12 mmHg in both eyes (OU). Pupils were round and reactive with a negative relative afferent pupillary defect. Slit-lamp examination revealed moderate nuclear cataract OU. Fundus examination revealed macular pucker OU, few dot and blot hemorrhages and microaneurysms (mild nonproliferative diabetic retinopathy) OU, and mild arterial narrowing (grade 1 hypertensive retinopathy) OU (Figure 2A and B). Optical coherence tomography (OCT) imaging of the macula showed new onset subretinal fluid (SRF) OU (Figure 2C and D). Fluorescein angiography (FA) (Figure 2E and F) and indocyanine green (ICG) angiography (Figure 2G and H) of the retina and choroid revealed no abnormalities related to the current condition in either eye.

Based on multimodal imaging findings and his medication history, we suspected toxic maculopathy secondary to erdafitinib treatment. After discussing the case with his oncologist, the medication was discontinued. Unfortunately, this was the patient's final visit, as he passed away two weeks thereafter due to his metastatic bladder cancer complications.

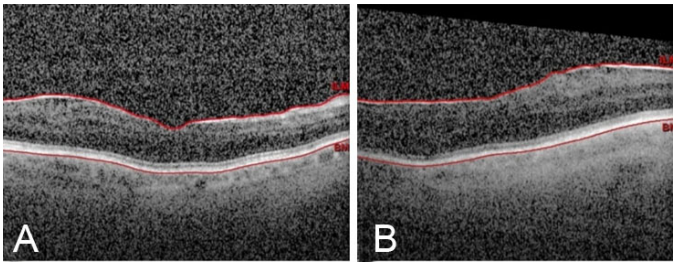

Figure 1: Optical coherence tomography (OCT) imaging of the right (A) and left (B) eyes three months before his current visit. Mild epiretinal membrane (ERM) with mild intraretinal structural changes are noted bilaterally.

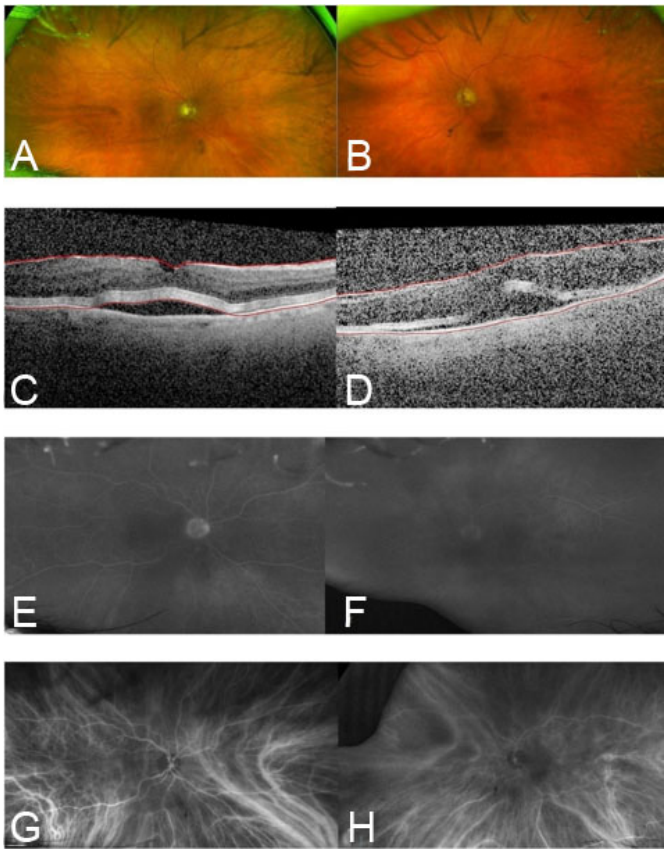

Figure 2: Multimodal imaging of the most recent visit. (A, B) Ultra Widefield Fundus photos of the right (A) and left (B) eyes which reveal mild Epiretinal membrane, few dot and blot hemorrhages and microaneurysms in both eyes (mild non-proliferative diabetic retinopathy), and mild arterial narrowing in both eyes (grade 1 hypertensive retinopathy). (C, D) Optical coherence tomography (OCT) imaging of the right (C) and left (D) eyes which depict a new onset subretinal fluid in the center of the macula bilaterally. (E, F) Late images of fluorescein angiography of the right (E) and left (F) eyes. There is no leakage, pooling, or staining in the macula bilaterally. (G, H) Indocyanine green (ICG) angiography of the right (G) and left (H) eyes which show a normal choroidal vasculature pattern in the macula.

DISCUSSION

Fibroblast growth factor receptors (FGFRs) are transmembrane receptor tyrosine kinases activated by fibroblast growth factors (FGFs). They play key roles in regulating tissue and metabolic homeostasis, endocrine functions, and healing.

Fibroblast growth factor signaling often activates the Mitogen-Activated Protein Kinase–Extracellular Signal-Regulated Kinase (MAPK–ERK) pathway downstream [3]. In the eye, the MAPK pathway, activated by the fibroblast growth factor receptor (FGFR), plays a key role in the maintenance, survival, and repair of the retinal pigment epithelium (RPE) [4]. Basic fibroblast growth factor (bFGF) is a neurotrophic factor distributed throughout the retina, with its highest expression localized in the nuclei of macroglial cells and the RPE [4].

Its active presence is essential for the proper functioning of the RPE. It is also crucial for promoting DNA synthesis and growth in young RPE cells, while also preventing apoptosis in mature RPE cells [5].

The above-mentioned hypotheses are supported by evidence from several animal models. For instance, bFGF has been shown to preserve photoreceptors in the retinas of rats, which were genetically predisposed to retinal degeneration due to a defect in the RPE. Interestingly, in these rats, both bFGF and the expression of FGFRs were downregulated, suggesting that diminished FGF signaling may be a primary cause of retinal dystrophy in these animals [6]. Additionally, in cat and rabbit models of retinal detachment (RD), FGF, and components of the MAPK pathway have been shown to play an important role in retinal repair [7].

Another significant clinical finding reveals that bFGF levels are elevated in the SRF of patients with retinal detachment, suggesting that FGF may play similar protective and survival roles in humans [8]. Furthermore, in another study, it was shown that the protective effect of VEGF inhibition on the retinal epithelium may outweigh the potential harmful effects of FGFR inhibition [9].

Fasalino et al. [10] analyzed flow-void OCT angiography, macular OCT, and FA in a patient with retinopathy associated with pan-FGFR inhibitor therapy. However, the patient was concurrently receiving multiple chemotherapeutic agents, making it challenging to isolate the retinal effects specifically attributable to erdafitinib. While no abnormalities were initially observed in their imaging studies by them, after a closer examination of the images, they reported distinct alterations in the choriocapillaris and larger choroidal vessels. Based on this case report and other above-mentioned experimental findings, we opted to assess our patient using multimodal imaging. In our patient, FA and OCT macula showed angiography silent SRF which, in our opinion, is similar to angiographic silent cystoid macular edema (CME). However, CME is characterized by the accumulation of fluid in cystic spaces within the inner nuclear and outer plexiform layers, with or without associated SRF. In the absence of intraretinal structural changes, we believe that the observed fluid is more consistent with toxic maculopathy rather than CME.

In contrast to the findings reported by Fasalino et al. [10], ICG in our patient demonstrated a normal choroidal vascular pattern in both the macula and peripheral retina. These findings suggest that neither the choriocapillaris nor the larger choroidal vessels are likely to be the underlying pathology.

We believe that, in FGFR inhibitors retinopathy, similar to CME, fluid accumulation in subretinal space can happen as a result of fluid accumulation in the extracellular spaces and/or inside the cells because of cytotoxic cell swelling [11]. Extracellular fluid accumulates when fluid passes from blood vessels into the tissue due to the breakdown of the blood-retina barrier and intracellular fluid forms by swollen muller and glial cells [11]. Although these medications impact the entire eye, we hypothesize that the higher density of photoreceptors, taller RPE cells, and thicker choroid in the posterior pole make the macula more susceptible to their adverse effects, which are expected to occur either focally or multifocally. This hypothesis is more consistent with intracellular fluid accumulation and toxic maculopathy. Furthermore, angiographically silent SRF does not indicate the absence of leakage; rather, the leakage is too minimal to be detected using current protocols up to 30 minutes after dye injection. We assume that prolonged image capturing may reveal leakage and pooling of fluorescein dye in the subretinal space. This hypothesis, as well as the optimal duration of FA required for confirmation, warrants further investigation to determine whether SRF accumulation is extracellular or intracellular. However, based on our findings—specifically, the presence of SRF on macular OCT in conjunction with normal fluorescein angiography and indocyanine green angiography—there appears to be a trend toward intracellular fluid accumulation.

Considering the temporal relationship and principles of causality, we believe that other chemotherapy agents and concomitant routine medications were unlikely to be responsible for this recent finding for several reasons. First, four months prior to the most recent visit—while the patient was receiving all other chemotherapy agents and routine medications—macular OCT imaging (Figure 1) did not demonstrate the changes now observed. Second, all other chemotherapy agents were discontinued two months before the most recent visit, and the patient was not receiving any treatment for bladder cancer other than erdafitinib, which was initiated one month before the last visit and coincided with the onset of visual decline two weeks prior to his last visit.

Based on these hypotheses and the multimodal imaging findings in our case, we believe that toxic *maculopathy* is a more accurate term to describe the retinal changes induced by erdafitinib.

Becker et al. [2]. found acetazolamide ineffective in treating their case, in contrast to its success in managing angiographically silent CME in some retinitis pigmentosa patients; however, the spontaneous resolution of SRF following the discontinuation of FGFR inhibitors is anticipated, based on cause-and-effect theory. We are unable to comment on this, as the patient passed away two weeks after his last visit.

CONCLUSION

Erdafitinib can cause toxic maculopathy with subretinal fluid on macula OCT with no abnormalities on FA and ICG. The natural course and treatment options should be further investigated in more comprehensive studies.

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Author Contributions

Nicholas Hallman – Conception of the work, Design of the work, Acquisition of data, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Joby J Tsai – Conception of the work, Design of the work, Acquisition of data, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Drew Bawcombe – Interpretation of data, Drafting the work, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Heather B Seith – Interpretation of data, Drafting the work, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Arash Maleki – Conception of the work, Design of the work, Analysis of data, Interpretation of data, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Shailesh Gupta – Conception of the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

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Conflict of Interest
Authors declare no conflict of interest.

Data Availability
All relevant data are within the paper and its Supporting Information files.