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CASE STUDY

PHOTIC RETINOPATHY CAUSED BY EXCESSIVE USE OF SMARTPHONE: **REVIEW AND CASES SERIES REPORT**

Essam Yahiya Al-Shamahi¹, Emad Hassan Al-Shamahi¹, Nawal Mohammed Al-Hababi¹, Nashwan Yahya A Al-Shamahi² , Hassan Abdulwahab Al-Shamahy^{2,3}

¹Opthalmology Department, Faculty of Medicine and Health Sciences, Sana'a University, Republic of Yemen. Medicine Department, Faculty of Medicine and Health Sciences, Sana'a University, Republic of Yemen. ³Department of Basic Sciences, Faculty of Dentistry, Sana'a University, Republic of Yemen.

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Abstract



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*Address for Correspondence:

Dr. Hassan A. Al-Shamahy, Medicine Department, Faculty of Medicine and Health Sciences, Sana'a University, Republic of Yemen. Tel: +967-1-239551: E-mail: shmahe@yemen.net.ye

Damage to the retina, especially the macula, brought on by extended exposure to sunlight or other strong light sources like lasers or arc welding is known as photo retinopathy. The phrase is interchangeable with retinal phototoxicity and includes solar, laser, and welding retinopathy. Staring at the sun, observing a solar eclipse, or exposing oneself to UV light, Illuminant D65, or other strong light are the usual causes. Overuse of smartphones has recently been linked to incidences of blue light-induced photoretinopathy. Reversible vision loss from photo retinopathy usually lasts anywhere from a month to over a year. Fundus changes, which are variable and often bilateral. Although there is currently no proven treatment for photo retinopathy, it usually resolves on its own over time. A method that is sometimes tried but gives ambiguous results is the use of corticosteroids to treat early macular edema. A study of six patients aged 26-35 years visited the eye consultants Center in Sana'a city, Yemen, including 4 males and 2 females, found that they experienced binocular blurred vision for 5-7 days. All patients underwent corticosteroid treatment and reduced smartphone usage. Five cases achieved reversible recovery within few days to 2 months, while one case persisted over a year. All patients had normal anterior and posterior segments, no altered macular reflex, and no macula capillary network abnormalities with hypopigmentation dot at the centre of fovea. High-definition optical coherence tomography (OCT) discovered foveolar harm, which be similar to some cases of solar photic retinopathy. In conclusion this study suggests that prolonged exposure to phone screen light by excessive use increases the risk of foveal injury.

Keywords: Blue light, excessive use, photic retinopathy, smartphone, Yemen.

INTRODUCTION

Light can induce photomechanical and/or photothermal reactions that are harmful to the eye. Photo injury occurs when the retina and choroid are damaged as a result of the consequences of light absorption or diffraction, which include heat generation, production of oxygen free radicals, and resulting tissue inflammation^{1,2}. Both visible light (400-700 nm) and shorter-wavelength ultraviolet (UV) radiation (UV-C, 100-280 nm; UV-B, 280-320 nm; UV-A, 320-400 nm) are considered forms of light radiation. Below 300 nm, the cornea shields the retina from UV rays. Most UV-B and UV-A rays are blocked by crystalline lenses, but only a tiny quantity of potentially dangerous UV-B radiation in the 320 nm range is transmitted by crystalline lenses in individuals under 30^{3,4}. Brow tinting, corneal refraction of light not incident perpendicular to its surface (Fresnel's laws), papillary reactions, aversion, strabismus, and blinking are further ocular defensive mechanisms against UV and strong visible light^{5,6}. The extent of photochemical retinal injury depends on individual defense mechanisms, the location and area of the retina exposed to light, and the time, strength, and spectrum of light exposure^{7,8}. Photochemical effects also depend on circadian rhythms⁹, oxygen tension¹⁰, and body temperature¹¹. The extent of photochemical retinal injury depends on individual defense mechanisms, the location and area of the retina exposed, and the duration, intensity, and spectrum of light exposure^{7,8}. Optical coherence tomography (OCT) provides clinical insights into the effects of damage. The most common signs of acute photomechanical retinal trauma are focal retinal pigment tears (RPE) and chorioretinal hemorrhages. OCT of photomechanical injuries typically shows focal

tears in the outer retinal hyperreflective zones, which increase with the severity of the damage⁷. Macular holes may be present initially or develop after the injury, closing spontaneously or requiring surgery^{12,13}. Epiretinal membranes^{12,14}, choroidal neovascularization¹⁴, and subretinal internal limiting membrane (ILM) hemorrhages^{12,15} may regress after the injury^{12,14}, and choroidal neovascularization¹⁴, and subretinal internal limiting membrane (ILM) hemorrhages^{12,15} may require treatment. Retinal pigment damage and hyperplasia can regress or worsen after photomechanical trauma¹².

Accidental retinal damage (phototoxicity) is known as photoretinitis or retinal phototoxicity. These injuries result from prolonged exposure to intense light, which is usually tolerable if the patient is exposed only momentarily^{7,16}. Retinal phototoxicity occurs when chorioretinal temperature rises are too low to cause photothermal damage, but requires light levels much higher than normal environmental levels and exposure times ranging from several seconds to minutes. Short wavelength light radiation can produce reactive oxygen species (ROS) in the retina (such as hydroxyl radicals, superoxide anions, and lipid hydroperoxides), which damage cell membranes, proteins, carbohydrates, and nucleic acids. The light-absorbing molecules that produce these unstable oxygen free radicals are known as photosensitizers. The photochemical damage leads to retinal cell dysfunction and death^{7,16}.

The risk of photosensitizer induced retinal phototoxicity (UV-blue, Category 2, HAM, or blue light hazard) enhances rapidly with reduce wavelength^{7,16}, comparable to the absorption spectrum of lipofuscin in the retinal pigment, its initial mediator. Consequently, UV light is significantly more hazardous than visible light, and violet light is more hazardous than blue light¹⁶. This spectrum of action has been repeatedly validated and forms the basis of the internationally agreed-upon lens phototoxicity index (PFI) used to estimate the risk of acute retinal

phototoxicity^{1,17}. Damage occurs in the retinal pigment and/or photoreceptor layers¹⁸. Photoretinopathy only occurs if the acute cellular damage is so excessive that it overwhelms retinal repair mechanisms. Photoretinopathy can be divided into photopigment-mediated and photosensitizer mediated phototoxicity^{7,16}.

Each class has its own characteristic action spectrum that describes how effective different wavelengths are at inducing photochemical damage¹⁹. The severity of retinal phototoxicity mediated by photopigments (bluegreen, class 1, Noel type, or white light) peaks at approximately 500 nm (blue-green), similar to the light sensitivity of night vision because the photopigment rhodopsin mediates both processes. Damage occurs in the photoreceptor layer or in both the photoreceptors and the retinal epithelium layer²⁰. The severity of retinal phototoxicity mediated by photosensitizers (ultraviolet-blue, class 2, Ham type, or blue light hazard) increases rapidly with decreasing wavelength, similar to the absorption spectrum of lipofuscin in the retinal epithelium, the primary mediator²¹. Thus, ultraviolet light is more dangerous than visible light, and violet light is more dangerous than blue light¹⁷.

The following six cases of photoretinitis are selected from a large cohort that we treat at our center in Sana'a and will provide an overview of the high incidence of this condition in Yemen, the lack of awareness in the community about the threat posed by light from mobile phone screens to the eyes, and the increasing number of people addicted to mobile phones or using them for programming and educational purposes.

CASE SERIES REPORT

Two females and four males, aged 26 to 35 years were integrated in the study. Four of the patients worked in jobs related to smartphones, such as computer programmers, sales representatives, and telecommunications equipment salespeople.

Table 1: General characteristics and symptoms of photo-retinopathy in 6 patients diagnosed with smartphone-
induced photo-retinopathy.

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S. N.	Sex	Age	Occupation	Time exposure per days	Symptoms	Eye side	Follow up	
1	F	26	Student preparing graduation program	3 continuous days	Blurring vision, visual acuity reduced (0.6), reduced visual contrast sensitivity	Both	Residual effect lasting after one month 0.9	
2	F	28	House wife watching TV series	More than 8 hours daily for 3 days	Blurring vision, reduced visual contrast sensitivity	Both	Reversible lasting for one month, 1.0	
3	М	29	Sales representative	14-16 hours more than one week	Blurring vision, visual acuity 0.9, reduced visual contrast sensitivity	Both	Reversible lasting for 2 months, 1.0	
4	М	35	Construction worker	6-8 hours daily for more than one month	Blurring vision, reduced visual contrast sensitivity	Both	Reversible lasting for 3 months, 1.0 p	
5	М	29	Employee addicted Watching TV series	6-8 hours daily for more than 3 months	Blurring vision, visual acuity reduced (0.7), reduced visual contrast sensitivity and reduced color saturation	Both	Irreversible lasting for more than 1 year, 0.9 Reduce color saturation and contrast sensitivity	
6	М	26	Works in selling communications equipments	6-8 hours daily for more than one month	Blurring vision, reduced visual contrast sensitivity	Both	Reversible lasting for 2 months	



Case I: A 26-year-old woman, who had been exposed to Smartphone light for three consecutive days, complained of drop in VA with blurred vision in both eyes. Severe macular edema (foveal) is shown in both OCT and central foveal yellow to white spot is shown in fundoscopic pictures in both eyes.



Case II: A 28-year-old housewife, addicted to watching TV series on Phone for 6-8 hours a day for two months, complained of blurred vision in both eyes. Figures (B) and (C) shows central foveal yellow spot similar to the lesion of solar retinopathy in right and left eye. Figures (A) and (D) Shows interruption of EZ and IZ lines in fovea.

Case 1: She was exposed to screen light for three consecutive days (she hadn't slept for three days due to psychological reasons, so she spent most of her time on her smartphone). Her average exposure to screen light in Case 3 was 12 to 16 hours per day. The other two patients were housewives and employees addicted to watching TV series on their smartphones. Their exposure was 6 to 8 hours per day. All patients visited the Eye Consultant Center in Sana'a, Yemen, between March 1, 2024, and the end of March 2025, due to binocular blurred vision and reduced visual contrast sensitivity for 5 to 7 days, claiming they were unable to function properly without eye pain or headache. They experienced no other eye discomfort, and their visual acuity was unaffected in four cases, although visual acuity was reduced in two cases. All patients underwent corticosteroid treatment and reducing time of using smartphone; and five cases achieved reversible recovery within 2 weeks to 2 months, but one (case 5) was non-reversible and the symptoms persisted to date (over 1 year). In all cases, slit lamp biomicroscopy revealed normal. There were no cells in the vitreous. Fundus examination showed hypopigmentation of the fovea, resembling the typical lesion of solar phototoxicity. Optical coherence tomography (OCT) was then performed, revealing disturbances in the over

fovea, particularly in the ellipsoid zone (EZ). Furthermore, the capillary network in the macular appeared normal.

DISCUSSION

Given the challenge of diagnosis and the first exclusion of common macular lesions, we chose to begin with a history survey. It was discovered that the patients had no record of systemic illness, alcohol and tobacco misuse, systemic or ocular medicine, or eye disease in the family. A further history survey revealed no prior experience with electro welding or sun gazing. However, a thorough background check showed that they had a three-year smartphone addiction. In addition to using their phones normally during the day, they were used to using them for six to fourteen hours every day. They frequently used their phones while in bed at night with the lights out, without turning on "Night Shift" with protective mode, even in the dark. The patients were instructed to put on "Night Shift" with protective mode, go to bed early, and restrict the amount of time they spent staring at their phones outside of regular social interactions.



Case III: A 29-years old sales representative 14-16 hours exposure per day, complained of blurred vision in left eye due to phone photo toxicity and, decreased visual acuity in Right eye due to previous solar retinopathy. Figure A shows typical lesion of solar retinopathy in right eye, figure C shows yellow to white lesion at the fovea of left eye, figure B shows retinal interruption of EZ and IZ in the fovea of Right eye.



Case IV: A 35-years male, 6-8 hours exposure per day complained of blurred vision in both eyes. Figures A and B (Right eye fundus) showing yellow to white dot at the centre of fovea, Figures C, D representing left and Right eye OCT respectively both shows interruption at ellipsoidal zone above the RPE at fovea.

Following our advice strictly, and treatment with corticosteroid they showed improvement of their eyesight after about 1-3 weeks except for one case. Our findings in these six instances are consistent with the fact that light-induced retinal damage, particularly from blue light, is a known phenomena in both lab and real-world settings²²⁻²⁹. It is generally accepted that the actual spectrally weighted irradiance is lower than the natural exposures when compared to the reasonably

anticipated exposure to optical radiation from mobile phones²⁵. The worries have been satisfactorily addressed by the International Commission on Nonionizing Radiation Protection Guide, yet the spectral peaks of cell phones are strikingly similar to shortwavelength blue visible light²³. Although the danger is considered modest in the majority of assessments, it is unknown if or when a harm threshold is achieved.



Case V: A 29-years male, 6-8 hours exposure per day employee addicted Watching TV series, complained of blurred vision and decreased contrast sensitivity in both eyes. Figure A shows yellow to white spot at the centre of fovea, figure B retinal interruption at the fovea.

On the contrary, millions of people rely on their mobile phones more and more these days. In addition to reading on a phone, which draws people's eyes very near to the screen, people may spend more time staring at their phones these days than they do outside. We gave the patients a cautious and well-considered diagnosis because there aren't many case reports of light-induced retinopathy brought on by smartphone addiction in Yemen and worldwide. Following our six patients many months of follow-up and the elimination of recognized macular disorders, we think our initial diagnosis was plausible.

First, during our follow-up visits over a few months, we ruled out all known macular disorders. Second, a thorough history was taken, which showed that they had used smartphones extensively during the previous three years. Finally, in accordance with people's viewing patterns, a simultaneous binocular lesion arises in the fovea maculae. The pathologic alterations of chronic light damage align with the outer segment alterations seen by OCT²⁶. As demonstrated by Huang *et al.*, who found that following a 6-month behavioral intervention, patients' visual acuity considerably improved to normal, and this was corroborated with updated OCT pictures, in thier patients' and diagnosis may be confirmed by follow-up for six months as they suggested²².

To our knowledge, these are rare cases of light-induced retinopathy due to smartphone addiction in Yemen. Studies currently limited to the effects of smartphones on human visual acuity or temporary blindness resulting from prolonged smartphone use. Aside from causing diagnostic confusion, this may not be a cause for concern, as the symptoms of our condition are associated with repeated and prolonged exposure to smartphone screens. However, it will certainly have a profound impact on both manufacturers and consumers, especially as we enter the era of virtual reality and its increasing user base.

Limitation of the study

In Yemen, several studies have been conducted on ophthalmic diseases, including posterior scleritis³⁰,

patterns of uveitis³¹, bacterial conjunctivitis in adults³², epidemiology and etiological diagnosis of corneal ulceration³³, prevalence of risk factors for trachoma among primary school children³⁴, bacterial causes and antimicrobial susceptibility pattern of external ocular infections³⁵, prevalence of S. aureus in external ocular infections and incidence of methicillin resistant S. aureus in isolates³⁶, comparison of peripheral, combined peripheral and superficial approaches using vitrectomy for the removal of congenital cataracts with primary intraocular lens implantation³⁷, prevalence of risk factors for trachoma among primary school children³⁸, and neonatal bacterial conjunctivitis³⁹, but there is no single report or study on light-induced retinopathy, and this is the first study on this topic in Yemen. A limitation of this study is its reliance on short-term follow-up. A case study is needed at all eye centers in Yemen to assess this problem in the country and build a better understanding of it, as well as its association with other causes of light-induced retinopathy.

CONCLUSIONS

A large portion of the world's population is exposed to blue light for a few minutes to several hours during odd times of the day (night). To reduce your risk of blue light exposure, examine the spectral output of a light source, as light has a cumulative effect and many properties such as wavelength, duration, intensity, and time of day. This study suggests that prolonged exposure to blue light increases the risk of eye disease, but this can be extrapolated to longer exposures. Consequently, this study suggests more research be done on the effects of prolonged exposure to even little levels of blue light on the eyes.

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AUTHOR'S CONTRIBUTIONS

Al-Shamahi EY: case's diagnosis, investigation. Al-Shamahi EH: case's diagnosis. Al-Hababi NM: data evaluation. Al-Shamahi NYA: investigation. Al-Shamahy HA: writing original draft, review. Final manuscript was checked and approved by all authors.

DATA AVAILABILITY

The accompanying author can provide the empirical data that were utilized to support the study's conclusions upon request.

CONFLICT OF INTEREST

There is no conflict of interest around this work.

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