

# A Very Rare Case of Complete Visual Recovery After a Week of Complete Cortical Blindness with No Light Perception in Both Eyes Following Burns

**Henrietta Olubusayo Osoba\*, Ojuroye Bolajoko and Akintade Yetunde**

*Consultant Ophthalmologist, General Hospital Gbagada, Lagos, Nigeria*

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**\*Corresponding author:** Henrietta Olubusayo Osoba, Consultant Ophthalmologist, General Hospital Gbagada, Lagos, Nigeria

## Abstract

A 23-year-old female on admissions for burns injury who developed sudden inability to see in both eyes on waking up after ten weeks on admission. On examination, patient was found to have No Light Perception (NPL) in both eyes, with a negative test for malingering. Ocular examination revealed normal anterior and posterior segment with round and reactive pupils bilaterally. Due to a confirmation of blindness, a normal ophthalmological examination, and a positive history of prior use of oxygen, a suspicion of cerebral ischemia secondary to the burns with resulting cortical blindness was made. An MRI was requested which came back negative. Absolute platelet count requested after the negative MRI result was however elevated. Patient was immediately commenced on bright light stimulation and a trial therapy after 96 hours when the result of the platelet count was received. A week after commencement of therapy, patient suddenly started seeing, with complete visual recovery after two weeks.

**Keywords:** Cortical Blindness; Cerebral Ischemia; Burns Injury; No Light Perception (NPL); Visual Recovery

**Abbreviations:** MRI: Magnetic Resonance Imaging; ESR: Erythrocyte Segmentation Rate; NPL: No Perception of Light; CF: Counting Fingers; TMS: Transcranial Magnetic Stimulation

## Introduction

Cortical blindness is a neurological condition that occurs due to a disruption in the geniculocalcarine pathway in the occipital lobe causing bilateral loss of vision, [1] and characterized by normal funduscopy, ocular motility and pupillary function [1]. It is an important cause of blindness from occipital cortical damage [2] from diverse aetiologies. Cortical blindness differs in terms of presence/absence of sparing of visual functions, severity, awareness of visual deficit as well as the magnitude of recovery of functions across different patients [3]. Although as a consequence of cerebral anoxia and ischemia, cortical blindness might occur secondary to burns, this is however rarely reported. The mechanism of burns causing this might be by causing a disruption to the blood flow to the brain, resulting in cerebral hypoperfusion which potentially might cause cortical blindness from involvement of the visual cortical area. Although cortical blindness might be common in brain surgeries, head trauma [4], stroke amongst others, it is very rare in burns patients.

While the management of cortical blindness involves addressing the underlying cause and focusing on visual

rehabilitation to maximize the remaining vision, there is no cure currently known for the pathology. The three main visual rehabilitation strategies include replacement, compensation and recovery techniques [5]. Furthermore, there is also no generally agreed management option for those with absolutely no vision in other to regain vision. Cortical blindness following stroke commonly presents as a homonymous or contralateral visual field loss more often unilaterally but might be bilateral [6]. Although it is a wide assumption that after a short period of spontaneous improvement the visual loss becomes permanent, visual training has been found to help recover some visual loss [6].

The thalamoperforating vessels from the posterior cerebral artery and the anterior choroidal artery supplies the optic tract, while the occipital lobe and optic radiation are supplied primarily by the posterior cerebral artery and the middle cerebral artery respectively. Additionally, the middle cerebral artery also supplies the occipital cortex. Insults that cause distorts this vascular supply either due to ischemia, emboli amongst others can cut off the functionality of the visual cortical area [4].

## Description of Case Report

We present a case of a 23-year-old woman who developed sudden inability to see anything (No Perception of Light) in both eyes while being on admission for burns. She was being managed for 38% residual full thickness burns involving both lower limbs secondary to gas explosion. She had been on admission for ten weeks and had been given oxygen in the course of the illness before developing sudden blindness on waking up. Patient is not a known hypertensive or diabetic, and had no known preexisting medical condition.

Consult was sent to the Ophthalmologist on account of sudden loss of vision in both eyes. On examination by the Ophthalmologist, the patient was found to have No Perception of Light (NPL) in both eyes, the confrontational visual field did not elicit any response in any of the four quadrants of the visual field in both eyes. Riddoch's phenomenon, visual hallucination and blind sight were all absent in this case. There was no associated headache but patient appeared slightly drowsy with mildly slurred speech. An assessment of the anterior segment revealed normal conjunctiva and cornea, normal depth and clear anterior chamber, round and reactive pupils. On fundoscopy, pink disc CD 0.35 bilaterally, normal vessels and macula and flat retina. Tests to rule out malingering including menace test, eye contact test, and surprise test were done and all negative. Colour vision test could not be done due to complete absence of vision. An assessment of cortical blindness secondary to cerebral ischemia was made. The patient was requested to do an urgent Magnetic Resonance Imaging (MRI) of the brain and orbit. She was told to commence bright light stimulation at least 10 times daily and was requested to be reviewed by the neurologist.

Twenty-four hours later, MRI came out as normal. Magnetic resonance angiography could not be done due to financial constraint. An absolute platelet count and Erythrocyte Segmentation Rate (ESR) was thereafter requested. ESR came back as normal, however, the platelet count was elevated. As no generally agreed guideline on this was available, patient was then commenced on a trial of Clopidogrel 375mg stat and low dose aspirin 75mg for six weeks. Bright light stimulation at least 10x daily was continued. Patient was already on tab slow k 1 bd, tab vitamin C 500mg bd, tab vitamin E 1 daily, tab zinc 50mg daily and tab tavanic 500mg daily while on admission before developing the visual loss, these medications were continued.

One week after commencement of treatment plan, patient suddenly started seeing with a visual acuity of counting fingers (CF) at 3 meters, with slight impairment of depth perception. Vision improved within another six days to a visual acuity of 6/6 and N5 for near vision, with Sloan's letter chart. Colour vision was normal, and confrontational visual field test was full. The depth perception however remained slightly impaired for up to three weeks after, but thereafter normalized. Patient was requested to

continue therapy for six weeks and for review by neurologist. The patient was requested to come to the ophthalmology clinic for follow up but however did not. Follow up on the ward four weeks and eight weeks after shows the visual restoration was sustained. Patient was subsequently discharged from the burns ward after five months on admission. The complete vision loss in this patient before visual recovery was for a period of one week.

To our knowledge, this demonstrates a very rare occurrence of complete visual recovery following complete blindness in both eyes, particularly because the trial intervention was not commenced within the stipulated 48 hours window due to the rarity of the diagnosis (Cortical blindness secondary to cerebral ischemia from burns).

## Discussion

Although rare, cases of burns can seldom result in cortical blindness. However, in this case, the incidence unusually happened ten weeks after admission. It is important to note that a lot of changes happen in the cortex following burns injury. In a case series by Garside et al [7] to investigate post burn intracortical inhibition and to demonstrate the usefulness of transcranial magnetic stimulation (TMS), a strong suggestion was made that burns injury causes cortical inhibition and found that TMS is a sensitive and useful investigative method for monitoring cortical inhibition changes in burns

Visual stimulation has been found to improve visual functions in patients with cortical blindness [5]. Although literature recognizes the role of visual training and rehabilitation in the recovery of lost visual fields in cortical blindness, there is no clearly defined management modality, nor standardized vision restoration treatment for cortical blindness [6] and more so if there is no vision at all.

In a study by Aldrich et al to review cases of cortical blindness between 1974 and 1984, the commonest underlying pathology was spontaneous ischemic stroke, cardiac surgery and cerebral angiography [8]. Regarding prognosis in this study, significantly improved outcomes were noted in those younger than 40 years, with no history of diabetes, hypertension. This is similar to the case in our study in terms of the age and absence of risk factors. This might suggest that younger patients might benefit more from the effect of neuroplasticity and have better visual recovery from cortical blindness. This is also similar to findings in literature [9,10]. Although this study by Aldrich et al suggests that reliable statements on visual prognosis can be made regarding prognosis 24 to 48 hours after blindness sets in in vascular related aetiology, with poor outcome in all cases with spontaneous stroke. It also noted that complete visual loss with no light perception is uncommon but when present, said to portend a poor prognostic sign [8], this is very much in contrast to findings from our study wherein patient had complete visual recovery in spite of initial visual acuity of no light perception.

Unlike the case report in the study by Rumbiak et al [11] which showed a gradual onset, although bilateral occipital lobe infarctions were demonstrable by CT scan, our study showed a sudden onset, with no obvious infarctions on imaging. This reveals that absence of changes on imaging does not rule out cerebral ischemic damage in cortical blindness.

Carbon monoxide poisoning in burns might also lead to blindness, as presented in a case report by Karishma et al in which a 32-year-old suffered blindness after exposure to smoke in a fire incidence. This patient, similar to the patient in our study was diagnosed with cortical visual impairment. A diagnosis of cerebral hypoperfusion was however made on magnetic resonance imaging unlike our own study which showed a normal imaging finding. Unlike this study however, wherein patient had partial recovery of vision following oxygen therapy, our case developed blindness in spite of receiving oxygen therapy earlier. Additionally, the case in the study by karishma et al [9] showed the blindness in an acute burn setting, unlike in our study in which the incidence occurred after two months on admission for burns. This might be because the hemodynamic changes continued into the chronic burns stage in our study as evidenced by the elevated platelet count. As a result of this, there could be microthrombi deposition in visual cortical vasculature which might not be detectable on routine Magnetic Resonance Imaging.

Despite the notable findings from our study, the absence of a magnetic resonance angiography in this case might pose a limitation as it makes it more difficult to detect the exact vessels involved in this pathological process.

### Conclusion

This case report highlights the importance of meticulous reasoning in the detection of rare and unusual neuroophthalmic pathologies even in the absence of obvious risk factors. It also demonstrates the need for further studies on the neuroplasticity of cortical neuronal regeneration in the context of cortical blindness, as well as the timing involved. It has been revealed by the study

that visual cortical neurons might still be reactivated to function fully even after over 48 hours of injury from cerebral ishaemia, and that no light perception for even up to a week in the context of cortical blindness might still be fully reversible.

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