

TRANSIENT VISUAL LOSS FOLLOWING FORAMEN MAGNUM DECOMPRESSION - SURGERY IN PRONE POSITION: A CASE REPORT

Nayak Vittal I¹, Anantha Kishan², Balasubrahmanyam A³, Patil S. R⁴, Savitha H. N⁵

¹Professor & HOD, Department of Ophthalmology, Vydehi Institute of Medical Sciences & Research Centre, Nallurahalli, Whitefield, Bengaluru.

²Professor & HOD, Department of Neurosurgery, Vydehi Institute of Medical Sciences & Research Centre, Nallurahalli, Whitefield, Bengaluru.

³Professor, Department of Ophthalmology, Vydehi Institute of Medical Sciences & Research Centre, Nallurahalli, Whitefield, Bengaluru.

⁴Assistant Professor, Department of Neurosurgery, Vydehi Institute of Medical Sciences & Research Centre, Nallurahalli, Whitefield, Bengaluru.

⁵Assistant Professor, Department of Ophthalmology, Vydehi Institute of Medical Sciences & Research Centre, Nallurahalli, Whitefield, Bengaluru.

ABSTRACT

Perioperative visual loss is an uncommon complication of surgery in the prone position, spinal surgery, and is also reported after cardiac surgery. It is important that the nursing faculty, attending on patients recovering from general anaesthesia for these procedures, check the vision immediately upon recovery. Prompt referral is essential if the patient has a deficit that was not noticed earlier. Visual deficit can be due to ischaemic optic neuropathy (Anterior or posterior), retinal artery occlusion (Branch or central), retinal vein occlusion (Branch or central), commotio retinae, pituitary apoplexy and occipital lobe infarction. Each condition has its clinical picture with fundus findings but in posterior ischaemic optic neuropathy (PION), pituitary apoplexy and occipital lobe infarction, the fundus is normal. Fundus finding will be disc pallor (Primary Optic Atrophy), in PION, pituitary apoplexy and occipital lobe infarction and take few weeks or months to appear. The case described outlines how early detection and referral can reduce visual morbidity, salvage vision, improving the quality of life of the patient.

KEYWORDS

Perioperative Visual Loss, Surgery in Prone Position.

HOW TO CITE THIS ARTICLE: Nayak VI, Kishan A, Balasubrahmanyam A, et al. Transient visual loss following foramen magnum decompression - surgery in prone position: A case report. J. Evid. Based Med. Healthc. 2016; 3(44), 2227-2231.

DOI: 10.18410/jebmh/2016/493

INTRODUCTION: It is important for clinicians to anticipate and avoid complications in all fields of medicine; however, it is impossible to totally avert these. Hence the need for being alert in every stage of a procedure, preoperative, intraoperative and postoperative. This especially on account of the fact that retinal ischaemia has to be treated within few hours of occurrence, to be of any benefit to the patient. In this case report and short review of perioperative visual loss, the value of early recognition of visual loss, diagnosis and management is brought out.

CASE REPORT: History: A 32-year-old male patient presented with blurred vision, right eye, few hours after recovery from Foramen Magnum decompression surgery for Chiari Malformation-1 with syrinx. In this procedure, the patient is made to lie prone, flexing the neck and supporting the head using a horseshoe shaped headrest. This facilitates adequate exposure of the craniovertebral junction. A motorised system was used to decompress & enlarge the foramen magnum.

On examination, examination was performed on the day of surgery by the resident on call. Visual acuity distance counting fingers close to face on the right side and counting fingers at 6 meters on the left side at the bedside. Lid oedema upper and lower on both sides, anterior segment examination of the eyes revealed a round pupil of 6 mm diameter reacting sluggishly to light (Direct and Consensual) on the right side, normal pupil on the left side, otherwise unremarkable. Fundus examination revealed in the right eye an area of retinal whitening in the posterior pole. A provisional diagnosis of Right eye retinal artery occlusion was made and the patient was administered intravenous twenty percent mannitol 350 mL slow drip over 45 minutes. The patient was re-examined in the outpatient department on day 1 after surgery, distance visual acuity was counting fingers at 1 meter in right eye, 6/9 Snellen in left eye, Near Vision N10 right eye, N6 left eye. Lids on the right side showed mild lid oedema. Anterior segment right eye (RE) pupil 5 mm dilated and sluggishly reacting to light, otherwise unremarkable. Left eye unremarkable fundus examination.

*Financial or Other, Competing Interest: None.
Submission 08-04-2016, Peer Review 20-04-2016,
Acceptance 27-04-2016, Published 02-06-2016.*

Corresponding Author:

*Dr. Nayak Vittal I,
#1656, 10th Main, H. A. L. 3rd Stage,
Bengaluru-560075, Karnataka.
E-mail: vnayakirvathur@gmail.com
DOI: 10.18410/jebmh/2016/493*

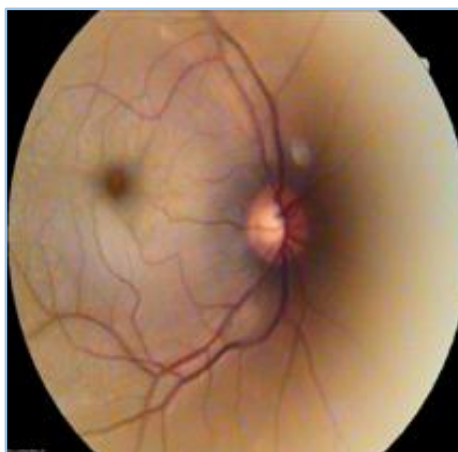


Fig. 1: Area of Retinal Oedema Following Decompression of Foramen Magnum

Right Eye: A localised area of oedema Figure 1.

Left Eye: Unremarkable fundus.

Both Eyes: Intraocular pressure was normal.

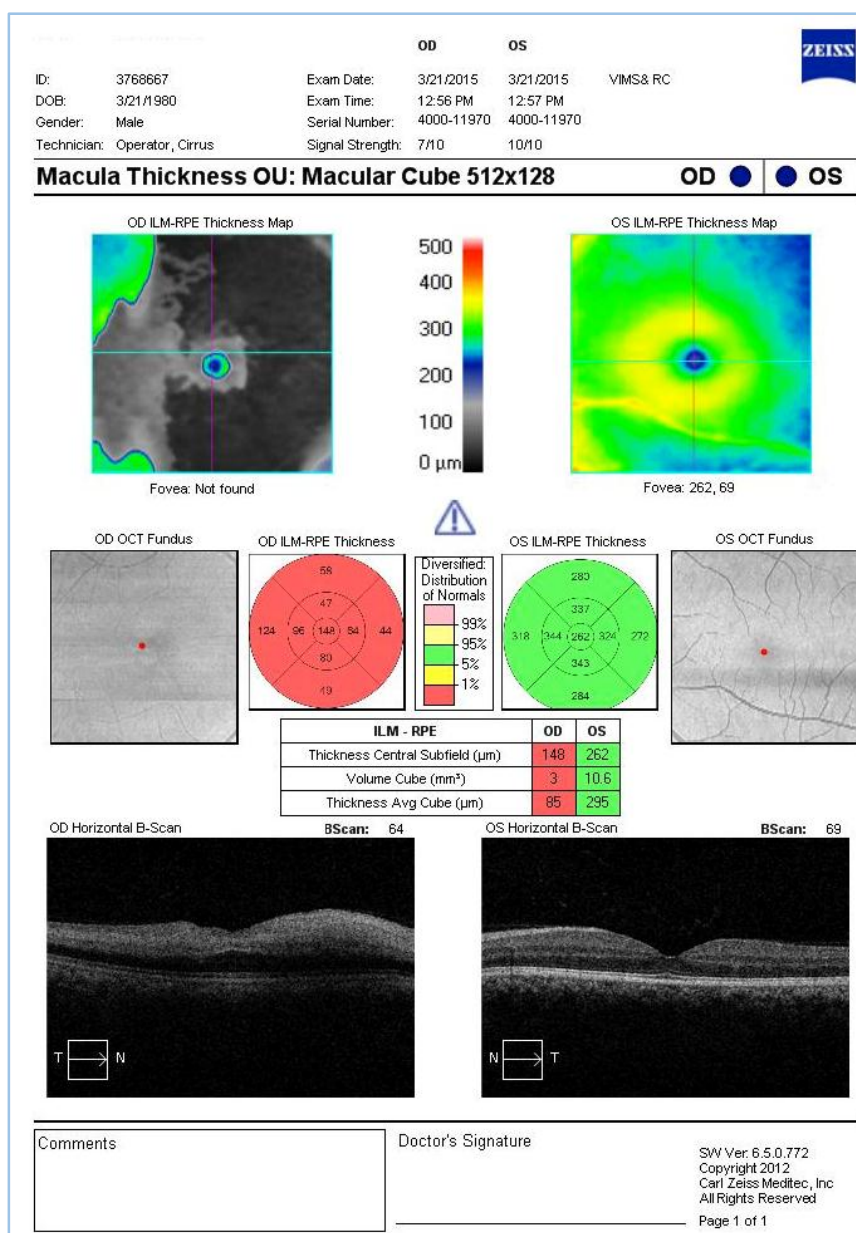


Fig. 2: Right Eye Disruption of the Outer Retinal Layers

Optical coherence Tomography (OCT) showed abnormal findings-(RE) decreased macular volume, disruption of the outer retinal layers and 3 D visualisation showed elevated folds around fovea. Treated on day one, topical nonsteroidal anti-inflammatory drugs, topical steroids, intravenous 20% mannitol, for two days after presentation. On Day 2, patients vision improved in RE 6/18 Snellen, LE 6/9, right pupillary reaction improved to normal. Macular oedema reduced over next few days and the cherry red spot disappeared.

Differential Diagnosis: Cherry red spot noted about twelve hours after neurosurgical procedure is most likely due to transient retinal arterial occlusion which recovered spontaneously. However, if the cherry red spot was due to retinal arterial occlusion, the visual recovery would not have taken place. (The longest period of retinal arterial ischaemia recovering completely is two hours).¹ The duration of this procedure was more than four hours.

Berlin's oedema following prolonged pressure on the eye is the other differential diagnosis. It occurs due to prone positioning to allow adequate exposure of the dorsal aspect of the neck and easy access to the surgical areas. In this case, as there was mild lid oedema, a differential diagnosis of acute central retinal artery occlusion or commotio retinae was made. The management of Central Retinal Artery (CRA) occlusion, and commotio retinae did include intravenous mannitol, this is what probably improved the condition.

DISCUSSION: Perioperative visual loss following non-ocular surgery can be due to ischaemic optic neuropathy (anterior or posterior), central retinal artery or vein (CRA/CRV) occlusion, cortical blindness and pituitary apoplexy.^{2,3} When no cause is detected, MRI scans of the brain and orbits are indicated. There is no proven effective treatment for perioperative ischaemic optic neuropathy.³ Fundus features of various conditions described are Acute Anterior Ischaemic Optic neuropathy-disc oedema, Acute Posterior ischaemic optic neuropathy- normal fundus, later the disc may show pallor due to primary optic atrophy, Acute Central Retinal Artery occlusion-cherry red spot, Central retinal vein occlusion- venous dilatation and tortuosity, superficial haemorrhages, cotton wool spots. Cortical blindness, pituitary apoplexy may not show any fundus findings.

While spinal surgery performed in prone position causes ischaemic optic neuropathy, coronary artery bypass grafting, valvular surgeries cause pituitary apoplexy and cortical blindness.³ Commotio retinae or Berlin's oedema is not described commonly and is associated with facial oedema or puffy eyelids due to prone positioning. The American Society of Anesthesiologists (ASA) – constituted a task force to formulate a Perioperative Visual Loss Practice Advisory. The ASA Task Force members consisted of anaesthesiologists, neuro-ophthalmologists, spine surgeons (Orthopedic and neurosurgical) and methodologists.^(3,4)

A "Practice Advisory" is a systematically developed report to assist decision making in areas of patient care, but it is not supported by the scientific literature to the same

degree as a "Standard" or "Guideline" because of the lack of sufficient numbers of adequately controlled studies. Hence, a practice advisory is not intended to dictate a standard of care. The ASA Task Force decided to focus on the perioperative management of patients undergoing spine procedures in the prone position under general anaesthesia, (The group at highest risk). The ASA Task Force came to the following consensus conclusions regarding this group of patients:

1. Some patients who undergo spine procedures while positioned prone and receiving general anaesthesia may have an increased risk of perioperative visual loss. This includes patients who are anticipated preoperatively to undergo procedures that are prolonged, have substantial blood loss, or both ("High-Risk Patients").
2. Consider informing high-risk patients that there is a small, unpredictable risk of perioperative visual loss.
3. The use of deliberate hypotensive techniques during spine surgery has not been shown to be associated with the development of perioperative visual loss.
4. Colloids and crystalloids should be used to maintain intravascular volume in patients who have substantial blood loss.
5. At this time, there is no apparent transfusion threshold that would eliminate the risk of perioperative visual loss related to anaemia. American Society of Anesthesiologists in general has recommended that transfusion is rarely indicated when the intraoperative haemoglobin is greater than 10 g/dL, and almost always indicated when the haemoglobin is less than 6 g/dL, but they caution that this should be individualised for each patient.
6. High-risk patients should be positioned so that their heads are level with or higher than the heart when possible. In addition, their heads should be maintained in a neutral forward position (i.e. without significant neck flexion, extension, lateral flexion, or rotation) when possible.
7. Consider the use of staged spine procedures in high-risk patients.
8. Most theories assume that the disorder is vascular in aetiology, and ischaemia can result from poor oxygen content within the circulating blood, reduced arterial perfusion pressure, or increase resistance to blood flow.

The ASA Practice Advisory.^(4,5) suggests that in the high-risk spine surgery patient, vision should be assessed as soon the patient is alert (e.g., in the recovery room, intensive care unit, or nursing floor). If there is visual loss, an urgent ophthalmologic consultation should be obtained to determine its cause. If an obvious ocular cause is not apparent, then urgent neuroimaging should be obtained, preferably magnetic resonance imaging, MRI (with gadolinium and stroke protocol techniques) is advised. When imaging is unrevealing, the likely cause of visual loss is

ischaemic optic neuropathy. Additional management may include optimising haemoglobin levels, haemodynamic status, and arterial oxygenation. Comotio retinae is a retinal injury after non-penetrating blunt trauma damage to the outer retinal layers caused by pressure on the eye. On fundus evaluation –Diffuse retinal whitening appears some hours after injury. Most commonly seen in posterior pole when it can be mistaken for central retinal artery (CRA) occlusion.

Mechanisms for the retinal opacification proposed axonal synapse disruption, photoreceptor outer segment disruption, extracellular oedema, glial oedema. Retinal blood vessels unaffected, but intraretinal haemorrhages may accompany the retinal pallor.^{5,6,7} This loss of transparency is associated with no damage to the retinal cells and minimal damage to the choriocapillaries.^{7,8,9,10}

Light and electronic microscopic studies of Berlin's oedema have shown that the outer retinal whitening is caused by fragmentation of the photoreceptor outer segments and acute damage to the receptor cell.^{6,7,8,9,11} Retinal pigment epithelial (RPE) hyperplasia occurs in response to disruption of the photoreceptor outer segment resulting in a corpuscular intraretinal pigmentary pattern similar to that seen in retinitis pigmentosa.⁸ Macular cube scans of Spectral Domain–optical coherence tomography (OCT) revealed no significant differences of the foveal thickness and total macular volume between injured and uninjured eyes in all 9 cases.¹² This result is consistent with previous reports demonstrating no extracellular oedema histologically.^{4,5} OCT shows revealed loss of definition of alternate layers, increased reflectivity of the inner and outer segments of photoreceptor cells, in the area of the inner segment/outer segment junction and RPE complex, and mild disruption of outer photoreceptor layer OCT.^{12,13,14,15} OCT picture of acute arterial occlusion through the area of retinal ischaemia (Area of whitening) shows hyperreflectivity of the nerve fibre layer.¹⁶

Multifocal electroretinogram, shows depression of the amplitude in the affected area which recovers if the outer segment regenerate or remain permanently affected if the receptors do not recover.¹⁷ Fortunately, the prognosis for visual recovery is good in most cases, as the condition clears in 3–4 weeks, but in some cases altered hyper/hypopigmentation or macular holes may occur and the damage to the photoreceptors and retinal pigment epithelium is extensive enough to result in permanent vision loss.⁵ The visual prognosis depends on the location and the extent of the photoreceptor outer segment regeneration. A normal fundus fluorescein angiogram will indicate a good visual prognosis, and spontaneous recovery is expected. In this report, if the cherry red spot was due to retinal arterial occlusion, the visual recovery should not have taken place. (The longest period of retinal arterial ischaemia recovering completely is two hours.).¹ The duration of this procedure was more than four hours; however, the authors attribute the cherry red spot to intermittent pressure and temporary branch artery occlusion which did not last the entire duration of the procedure.

CONCLUSION: It is imperative to check visual acuity, pupils and fundus of every patient who has undergone a neurosurgical procedure on the spine, or posterior cranial fossa soon after recovery from anaesthesia. These procedures necessitate prone position, with flexion of the neck and head support with cushioned horseshoe. Prolonged pressure on the eyes and face can result in branch or central retinal vein or artery occlusion, ischaemic optic neuropathy or rarely commotio retinae. However, intermittent changes in position could have resulted in transient ischaemia for few hours or minutes explaining the cherry red spot. In this patient, administration of intravenous mannitol resulted in improvement of the transient retinal ischaemia or commotio retinae.

Neurosurgeons have devised skull clamps in such cases to avoid the prolonged prone position and pressure on the face; however, in the absence of such a facility the need for early evaluation in the postoperative period is suggested. Early detection and management of retinal vein/artery occlusion, commotio retinae will reduce the chances of visual loss and morbidity, saving the patient's vision. Thus, the neurosurgeon can not only save the life but also salvage the vision improving the quality of life of the patient.

REFERENCES

1. Hayreh SS, Zimmerman MB, Kimura A, et al. Central retinal artery occlusion, retinal survival time exp. Eye Research 2004;78(3):723-736.
2. Newman JN. Perioperative visual loss after nonocular surgeries. Am J Ophthalmol 2008;145(4):604-610.
3. Berg KT, Harrison AR, Lee SM. Perioperative visual loss. Clinical Ophthalmology 2010;4:531-536.
4. Warner A Mark, Arens F James, Connis T. Richard et al., Practice advisory for perioperative visual loss associated with spine surgery. A report by the American Society of Anesthesiologists Task Force on Perioperative Blindness. Anesthesiology 2006;104: 1319–1328.
5. Dugel U Pravin, Win H Peter, Ober R Richard. Posterior segment manifestations of closed globe contusion injury. In Ryan SJ Retina. St. Louis: Mosby 2006;4th edn:2366.
6. Sipperley JO, Quigley HA, Gass DM. Traumatic retinopathy in primates: the explanation of commotio retinae. Arch Ophthalmol 1978;96(12):2267-2273.
7. Kohno T, Ishibashi T, Inomata H, et al. Experimental macular oedema of commotion retinae: preliminary report. Jpn J Ophthalmol 1983;27(1):149-156.
8. Mansour AM, Green WR, Hogge C. Histopathology of commotio retinae. Retina 1992;12(1):24-28.
9. Agarwal Anita. Gass atlas of macular diseases. Elsevier Saunders 2005;19(5th edn):219-221.
10. Bunt-Milam AH, Black RA, Bensinger RE. Breakdown of the outer blood-retinal barrier in experimental commotio retinae. Exp Eye Res 1986;43(3):397-412.

11. Blight R, Hart JCD. Structural changes in the outer retinal layer following blunt mechanical non perforating trauma to the globe: an experimental study. *Br J Ophthalmol* 1977;61(9):573-587.
12. Joo Youn Park, Woo Ho Nam, Seung Hoon Kim, et al. Evaluation of the central macula in commotio retinae not associated with other types of traumatic retinopathy. *Korean J Ophthalmol* 2011;25(4):262-267. Doi: 10.3341/kjo.2011.25.4.262.
13. Kitchens John W, Rubsamen Patrick E. Posterior Segment Ocular Trauma. In: Yanoff M, Duker JS, *Ophthalmology*. Elsevier Saunders 2014;4th edn:671.
14. Gupta V, Gupta A, Dogra MR. Atlas optical coherence tomography of macular disease and glaucoma. *Jaypee Highlights* 2012;4th edn:550.
15. Puliafito A Carmen, Hee R Michael, Schuman S Joel, et al. Optical coherence tomography of ocular disease. *SLACK Incorporated* 2004;2nd edn:121-122.
16. Saxena Sandeep, Saxena RC. *Retina atlas- a global perspective*. Jaypee Brothers Medical Publishers Pvt Ltd 2008;1st edn:167,174.
17. Lai TY, Yip WW, Wong VW, et al. Multifocal electroretinogram and optical coherence tomography of commotio retinae and traumatic macular hole. *Eye (Lond)* 2005;19(2):219-221.