

Visual function outcome post intracranial surgery in sellar region brain tumor



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ABSTRACT

Background: The proximity of sellar tumor to the optic nerve and optic chiasm can cause compressive optic neuropathy. Manifestation of optic pathway dysfunction varies from papilledema to optic nerve atrophy and affects visual function. This case report aims to present two patients with sellar region tumor treated with intracranial surgery.

Case Presentation: Two cases of sellar region tumor patients had complaints decreased visual function treated with intracranial surgery.

Case 1. An 8 years old female, visual acuity pre and post-surgery right eye (RE) 2.2;1.3, left eye (LE) 0.70;0.5. Color vision pre and post-surgery RE 0/38;0/38, LE 0/38;1/38 plate. Contrast sensitivity RE pre and post-surgery can't be evaluated and left eye 10%;2.5%. The visual field pre and post-surgery was hemianopsia bitemporal. The evaluation had been taken 1-week post-surgery. Case 2. A 24 years old female, visual acuity pre and post-surgery RE 1.5;1.0, LE 1.8;1.8. Color vision pre and post-surgery RE 0/38;1/38, LE 0/38;0/38 plate. Contrast sensitivity RE pre-surgery can't be evaluated and 25% post-surgery and LE pre and post-surgery can't be assessed. The visual field pre and post-surgery was hemianopsia bitemporal. The evaluation had been taken 1-week post-surgery.

Conclusion: All patients with sellar region tumor who underwent intracranial surgery had visual function improvement although progress differed between individuals. It represents a particular challenge for the physicians treating it.

Keywords: sellar, tumour, chiasm, optic.

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INTRODUCTION

Sellar region tumors constitute 10-15% of all brain tumors. The common lesions in the sella turcica are pituitary adenomas, and craniopharyngioma. Sella turcica is located in the base of skull, in early-stage often patients showed no intracranial hypertension and other signs of neural system. The mortality rate of sellar tumors is low, the morbidity in early-stage often showed decreased visual function and could lead to permanent visual loss, ophthalmoplegia, and other neurological complications in advanced stage that could impair the quality of life.^{1,2}

The proximity of sellar tumor to the optic nerve and optic chiasm, this condition can cause compressive optic neuropathy. Optic pathway dysfunction varies from papilledema to optic nerve atrophy and affects visual function. Slow and progressive decrease of visual function such as visual acuity, color vision, and contrast sensitivity. Visual field defect

also can happen related to anatomical distortions these lesions cause on the optic nerve and optic chiasm.^{2,3,4}

There have been several studies evaluation visual acuity on sellar region tumor, significant visual decrease after diagnosis in about 30% of the patients and visual acuity can improve after intracranial surgery (decompression), but still few studies talk about color vision and contrast sensitivity evaluation. This case report aims to present visual function outcomes in two patients with sellar region tumor treated with intracranial surgery.^{2,3,5}

CASE PRESENTATION

Case 1

A 8 years old female, was initially admitted to our hospital with headache, blurred vision on both eyes since 2 months and slowly getting worst. Visual acuity using Snellen Chart and convert to logMar was 2.2 right eye and 0.7 left eyes. Color vision test using Ishihara 38 Plates was 0/38 plate

on both eyes. Contrast sensitivity using Low Contrast Chart LEA Numbers can't be evaluated on right eye and 10% on left eye. The visual field with confrontation test was hemianopsia bitemporal. Segment anterior was expected, and segment posterior with direct funduscopy using slit lamp and color funduscopy was papil nervus opticus edema on both eyes (Figure 1). The brain and orbital Magnetic Resonance Imaging (MRI) with contrast displayed solid mass accompanied by cystic and calcified sections in the suprasellar region extending to the superior posterior aspect (size ± AP 4.1 x LL 2.9 x CC 3.2 cm) appears to press the third ventricle, mesencephalon posteriorly, compress chiasma opticum inferiorly, and cerebral anterior artery right left to anterior and attached to the internal carotid artery segment C6 right-left, posterior cerebral artery segment P1 right-left, and conclusion with craniopharyngioma. (Figure 2). There are signs of increased intracranial pressure. The intracranial surgery for



Figure 1. Color funduscopy in case 1: (a) Pre-surgery, papil nervus opticus edema both eyes. (b) Post-surgery optic atrophy both eyes.



Figure 2. Brain and orbital MRI with contrast in case 1; tumor in sellar region and compress the optic chiasm.

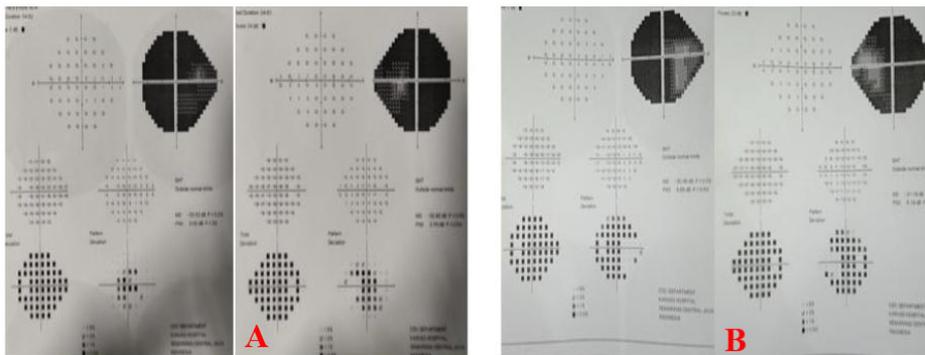


Figure 3. HVFA in case 2. Hemianopsia bitemporal (a) Pre-surgery and; (b) Post-surgery.

decompression, tumor was removed with a craniotomy surgery. Evaluation neuro-ophthalmological assessment 1 week after surgery showed some improvement visual function. Visual acuity was 1.3 right eye and 0.5 left eyes. Color vision test was 0/38 plate on right eye and 1/38 plate on left eye. Contrast sensitivity was right eye

couldn't be evaluated, and 2.5% on left eye (Table 1). Funduscopy was optic atrophy both eyes (Figure 1). The visual field was hemianopsia bitemporal.

Case 2

A 24 years old female, was initially admitted to our hospital with headache, blurred

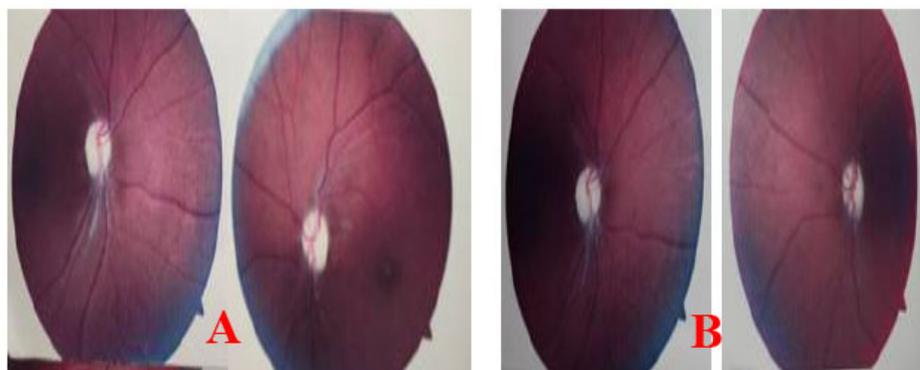
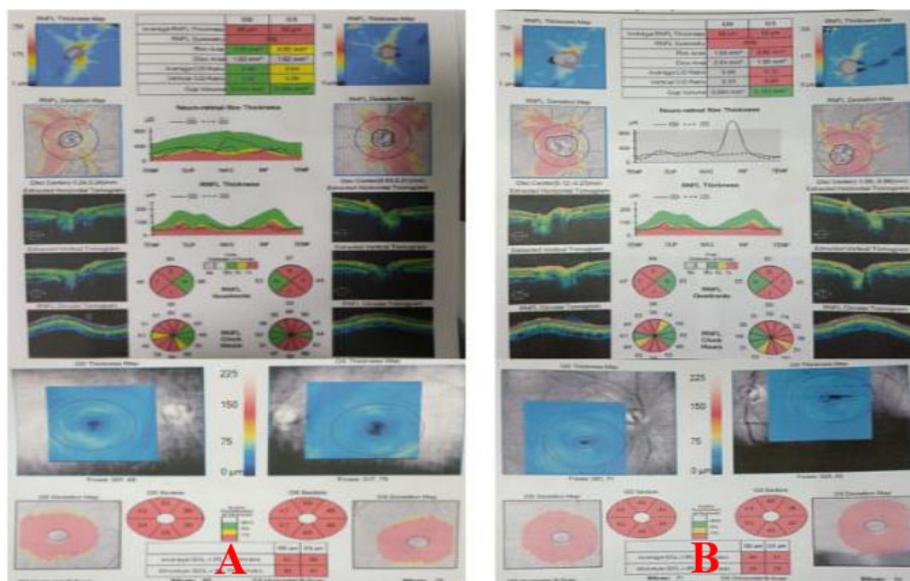
vision on both eyes since 4 months and slowly getting worst. The patient also has hypothyroid and had been surgery. Visual acuity using Snellen Chart and convert to logMar was 1.5 right eye and 1.8 left eyes. Color vision test using Ishihara 38 Plates was 0/38 plate on both eyes. Contrast sensitivity using Low Contrast Chart LEA Numbers can't be evaluated on both eyes. The visual field using Humphrey Visual Field Analyser (HVFA) was hemianopsia bitemporal (Figure 3). Segmen anterior was normal, and segment posterior with direct funduscopy using slit lamp and color funduscopy was optic atrophy on both eyes (Figure 4)

Optical Coherence Tomography (OCT) showed a thinning on macular ganglion cell layer (GCL) and inner plexiform layer (IPL) of the right and left eye with average thickness 52 µm and 58 µm. Average RNFL thickness was 56 µm right eye and 52 µm (Figure 5).

The brain and orbital MRI with contrast displayed intra-sella heterogeneous (semisolid-cystic) mass (AP size 2.86 x LL 3.32 x CC 4.54 cm) leading to sella turcica dilation. The lesion appears to extend to the right and left para sellar region, pushing the mesencephalon superiorly, attaching and pushing against the optic chiasma, pars intracranialis optic nerve right left and attaching to the left internal carotid artery, and conclusion with craniopharyngioma DD/ adenoma pituitary (Figure 6). There were no signs of intracranial elevation. In the intracranial surgery for decompression, tumor was removed with transsphenoidal surgery. Evaluation neuro-ophthalmological assessment 1 week after surgery showed some improvement visual function. Visual acuity was 1.0 right eye and 1.8 left eyes. Color vision test was 1/38 plate on right eye and 0/38 plate on left eye. Contrast sensitivity was 25 % on right eye and left eye can't be evaluated (Table 2). Funduscopy was optic atrophy both eyes (Figure 4). OCT still showed a thinning on ganglion cell layer (GCL) and inner plexiform layer (IPL) of the right and left eye with average thickness 46 µm and 41 µm. Average RNFL thickness was 55 µm right eye and 53 µm (Figure 5). The visual field was hemianopsia bitemporal become slightly recover (Figure 3)

Table 1. Visual function pre and post intracranial surgery in case 1

| Visual Function | Right Eye | | Left Eye | |
|------------------------------|--------------------|--------------------|-------------|--------------|
| | Pre Surgery | Post Surgery | Pre Surgery | Post Surgery |
| Visual acuity (LogMar) | 2.2 | 1.3 | 0.7 | 0.5 |
| Color Vision (Ishihara test) | 0/38 | 0/38 | 0/38 | 1/38 |
| Contrast Sensitivity | can't be evaluated | can't be evaluated | 10 % | 2.5 % |

**Figure 4.** Color funduscopy in case 2. Optic atrophy on both eyes. (a) pre-surgery and; (b) post-surgery.**Figure 5.** Optical coherence tomography thinning on RNFL,GCL and IPL both eyes (a) pre-surgery and (b) post-surgery.

DISCUSSION

The optic nerve is part of the central nervous system (CNS). CNS lacks the ability for regeneration and axonal growth. When exposed to axonal damage, glial scars are formed that limits the

diffusion of growth factors. Inhibitory myelin proteins like Nogo and myelin-associated glycoprotein, low expression of growth factors, and lack of laminin are also some factors that hinder the ability for re-growth. The more proximal the damage is to the eye, the quicker the apoptosis of

retinal ganglion cells will be. Apoptosis will lead to a cascade of p53 that will result in further cell death. Compressive optic neuropathy can occur by compressing the vascular supply and causing ischemia to the nerve or directly causing mass effect upon the axons, thereby impairing axonal transport and signal transmission. The optic disc may be swollen or atrophy in chronic condition.^{4,6,7}

Compression of the visual pathways causes a disturbance of conduction and consequent depression of visual functions such as slowly progressive reduced visual acuity, visual field, contrast sensitivity and dyschromatopsia. The most common sign is a slow progressive visual loss, sometimes associated with headaches. Bilateral visual loss can result from midline lesions. In this case report, visual acuity decreased in both eyes due to compression of the optic chiasm by a tumor in the sellar region as seen in the visual field defect is hemianopsia bitemporal and lesions on MRI.^{3,5,7}

The contrast sensitivity in this report also decreased, in case 2 couldn't be evaluated while in case 1 only the left eye could read the contrast chart 10%. Contrast sensitivity is the ability to detect grayness and background or distinguish between similar shades of light and dark. There are a variety of testing charts for contrast sensitivity. Contrast sensitivity is usually reduced in patients with compressive optic neuropathy. Abnormal contrast sensitivity is another sign of optic nerve dysfunction and maybe the most valuable test in terms of long-term follow-up and in clinical trials. Some patients with optic neuropathy have good acuity but may have reduced contrast sensitivity thresholds, it can be used to document visual recovery in compressive optic neuropathy, which is almost always associated with a reduction

in contrast sensitivity.^{8,9}

Another visual function affected is color vision. In both cases, the patient was unable to read the Ishihara Plate. In optic nerve damage the color vision disturbances are classified as acquired blue-yellow type III defects or acquired red-green type II defects, the type II red-green defects being the most typical. In cases of an acquired type II red-green defect the cause in most if not all patients is a lesion in the conduction system. A type II defect points to optic nerve disease but tells us nothing about the etiology (retrobulbar neuritis, toxic optic neuropathy or compression). Color vision studies in the chiasmal syndrome revealed type II acquired red-green defects. In advanced cases, color vision disturbance is so severe that it is no longer possible to classify the defect.^{10,11}

Color vision disturbance in eyes with visual field defects caused by meningioma, carotid aneurysm, pituitary adenoma and craniopharyngioma. These differences are probably attributable to differences in the location of these tumors in relation to the optic pathway and its vasculature. Sellar tumors can cause a bitemporal

hemianopia by chiasmal compression without compression of the optic nerves or non-decussating chiasmal fibers.^{10,11}

Management of compressive optic neuropathy is treating the underlying condition. Direct compression of the optic pathway by lesions or bone fragments or hematoma is usually treated surgically. In both cases, the underlying condition is lesion, sellar region tumor, so intracranial surgery is performed which is a decompression procedure. In case 1 was performed craniotomy surgery while in case 2 was performed transsphenoidal surgery.^{1,4,7}

In case 1 there was a better visual function improvement in the left eye than in the right eye. The right eye was blurred earlier than the left eye and the preoperative funduscopy optic nerve papilla was more edematous than the left eye. In case 2, the right eye's visual function was better than in the left eye. The left eye was blurred earlier than the right eye, and in preoperative funduscopy the optic nerve papilla in the left eye was more pallor than the right eye. In case 2 the decrease in visual function was heavier

than in case 1 due to the more prolonged onset of complaints in case 2 than in case 1. There was atrophic optic nerve papilla in case 2 while pre-surgery funduscopic in case 1 there was edema of the optic nerve papilla.

This case report was comparable with some sellar region tumor studies said that the visual function was can improved after decompression surgery, but in other studies the visual function was not found to improve. The prognosis of compressive optic neuropathy depends on the pathophysiology (vascular insufficiency or axonal damage) and the time between presentation and treatment.^{2,3,5,7}

The recovery of visual function is usually correlated with the length of time to decompress. The consensus in Fukiyama et al., that the earlier the decompression, the better the outcome. Complete visual function recovery has been seen as early as one week. Slow progressive vision improvements have also been reported in Bulters et al. study. Early decompression is recommended, but good results can still be obtained even if decompression is performed delayed. Better visual improvement in eyes with good preoperative vision rather than preoperative blindness, preoperative optic disc atrophy has a worse prognosis of visual function improvement.^{1,7,12,13}

The limitation of this study is the short term evaluation. The evaluation that is carried out is more extended, so it can better compare or know the progression of the visual function, whether it is improving or decreasing.⁷

CONCLUSION

All patients with sellar region tumor who underwent intracranial surgery had visual function improvement although progress



Figure 6. Brain and orbital MRI with contrast in case 2; tumor in sellar region and compress the optic chiasm.

Table 2. Visual function pre and post intracranial surgery on case 2

| Visual Function | Right Eye | | Left Eye | |
|------------------------------|--------------------|--------------|--------------------|--------------------|
| | Pre Surgery | Post Surgery | Pre Surgery | Post Surgery |
| Visual acuity (LogMar) | 1.5 | 1.0 | 1.8 | 1.8 |
| Color Vision (Ishihara test) | 0/38 | 1/38 | 0/38 | 1/38 |
| Contrast Sensitivity | can't be evaluated | 25 % | can't be evaluated | can't be evaluated |

differed between individuals. It represents a special challenge for the physicians treating it and early decompression is recommended.

ETHICAL CONSIDERATION

The patient had received signed informed consent regarding publication of their respective medical data in medical journal.

CONFLICTS OF INTEREST

The authors declare no conflict of interest.

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AUTHOR CONTRIBUTIONS

Supervision, methodology, and, writing the original draft: Yuriz Bakhtiar, Riski Prihatningtias.

Writing the original draft: Dea Prita Caesarita.

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