

Trend of Improvement in Visual Field Deficits after Transsphenoidal Endoscopic Surgery in Pituitary Tumour Patients: A Case Series

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Abstract

Introduction: Pituitary tumours account for approximately 15% of all brain tumours, and the growing tumours press against the optic chiasm, resulting in impairment of visual function manifested as visual field defects, decreased visual acuity, and decreased color vision. Compression of the optic chiasm by pituitary tumours generally results in selective loss of the temporal VFs, or bitemporal hemianopia, implying that the nasal retinal fibers are preferentially damaged. The reason for this preferential damage is not fully understood. Transsphenoidal surgery has been reported to safely reduce the pressure on the anterior visual pathway in most patients. Improvement in visual function may occur after transsphenoidal decompression of the chiasm. Because improvement in visual function may occur from a variety of proposed biologic processes.

Case Series : The number of patients according to gender was 71% male (10 people) while 29% female (4 people). The age distribution was found mostly at the age of 40-50 years 36% (5 people). The most common clinical symptoms were field disturbances 85% (12 people). Patients complained of visual field disturbances for 1-2 years as many as 58% (7 people). Vision before surgery is 1/6 as much as 45% (4 people). Improvements in vision were found for 1 month postoperatively as much as 22% (2 people).

Discussion : Compression of the optic chiasm by pituitary tumours generally results in selective loss of the temporal VFs, or bitemporal hemianopia, implying that the nasal retinal fibers are preferentially damaged. The minimally invasive transsphenoidal approach can be used effectively for 95% of pituitary tumours. Exceptions are those large tumours with significant temporal or anterior cranial fossa extension. In such circumstances, transcranial approaches are often more appropriate. Occasionally, combined transsphenoidal and transcranial approaches are used. Nevertheless, some surgeons extend the basic transsphenoidal exposure in order to remove some of these tumours and avoid a craniotomy. Potential mechanisms of axonal injury from a compressive lesion include direct disruption of conduction along the axon, impaired axoplasmic flow, demyelination with impaired signal conduction, and ischemia from compression or stretching of the blood supply of the chiasm by the tumour. An early fast phase of improvement is consistent with restoration of signal conduction along retinal ganglion cell axons after removal of a compressive lesion. In some individuals, we observed the rapid restoration of normal vision, which would be consistent with this hypothesis. In these individuals, a physiologic conduction block is presumably the main mechanism of injury.

Conclusion: The pattern of improvement of visual function after decompression of the anterior visual pathways suggests three phases of improvement. Improvement in visual function may occur after transsphenoidal decompression of the chiasm. Because improvement in visual function may occur from a variety of proposed biologic processes, we sought to better define this potential for improvement.

Keyword: Pituitary tumour, transsphenoid, neurosurgery

Introduction

Pituitary tumours are common in the sellar area. The prevalence of clinically apparent pituitary lesions is estimated to comprise approximately 10% of all intracranial lesions, while incidental pituitary tumours are detected in approximately 11% of

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individuals at autopsy. Pituitary tumours are virtually always benign adenomas, however pituitary carcinomas are reported to comprise about 0.5% of pituitary tumours. Microadenomas are tumours measuring less than 10 mm in diameter and those of more than 10 mm are termed macro-adenomas. Diffuse adenomas are ones that lead to sellar expansion, often compressing the residual gland into a thin membrane. Massive adenomas often replace the sellar floor; displace surrounding structures and undergo suprasellar extension. Endocrinologically functional tumours are often small; whereas silent or non-functioning tumours are large, detected only as a result of mass effects. [1]

Typically, nonfunctioning adenomas present as macroadenomas that cause neurological symptoms due to intracranial mass effects since hormonal inactivity leads to a delay in diagnosis compared with functioning pituitary adenomas. It has been reported that 96.5% of nonfunctioning adenomas present as macroadenomas and that 67.8% of patients with these tumours experience visual defects. For nonfunctioning pituitary adenomas, neurosurgery is the treatment of choice [2]

Functioning pituitary adenomas that secrete prolactin account for 40 to 60% of pituitary adenomas. Treatment of these tumours can be begun with a dopamine-agonist such as bromocriptine. Surgical resection, usually with the transphenoidal approach, should be considered for pituitary adenomas that secrete prolactin and show rapid deterioration in visual function as well as adenomas that secrete adrenocorticotrophic hormone, growth hormone, or thyroid stimulating hormone. When medical and surgical treatments are unsuccessful, radiotherapy may be used postoperatively.[3]

The visual deficits associated with pituitary adenoma depend on the size, location, and hormonal activity of the tumour as well as the position of the chiasm as it relates to the sella turcica . According to a recent study, the tumour volume also affects the severity of the visual field (VF) defect.[1,3]

Compression of the optic chiasm by pituitary tumours generally results in selective loss of the temporal VFs, or bitemporal hemianopia , implying that the nasal retinal fibers are preferentially damaged. The reason for this preferential damage is not fully understood. One explanation is the “anatomical theory,” in which nasal fibers are selectively vulnerable because of their anatomical location in the centre of the chiasm. Various authors have suggested that the basis for this anatomical theory is that the centre of the chiasm, containing the nasal fibers, is subject to the greatest pressure and/or stretch as a result of extrinsic compression. It also has been proposed that nasal fibers in the centre of the chiasm may be more susceptible to compression-induced ischemia due to the anatomy of the blood supply. Any or all of these factors may contribute to the

increased susceptibility of nasal fibers to chiasmal compression. However, they should also affect the temporal fibers and hence the nasal VFs, albeit to a lesser extent. Thus, a purely anatomical theory does not, by itself, account for the highly selective damage to nasal fibers that manifests as bitemporal hemianopia often with complete sparing of the nasal fields.[4-6]

An alternative explanation that could account for the selective damage to nasal fibers is the crossing theory. McIlwaine et al hypothesized that nasal fibers are selectively vulnerable simply because they cross each other: crossing fibers have a smaller area of contact than fibers that run parallel to each other. Therefore, any compressive force applied to the chiasm will result in greater stress on fibers which cross, compared to those that do not. If this crossing theory is correct, nasal fibers should be particularly vulnerable to compression independent of the anatomical location of chiasmal compression. Specifically, wherever the chiasm is damaged, there should be proportionately greater involvement of the temporal fields. The crossing theory has previously been investigated using finite element modelling, but the model remains limited by a lack of precise anatomical information.[7]

Neurologic signs and symptoms develop as adenomas grow beyond the confines of the sella turcica and exert pressure upon adjacent brain structures. As tumours enlarge, they compress the optic nerves and optic chiasm, and patients experience visual deficits and diminished visual acuity. Classically this causes a bitemporal hemianopia, i.e. visual loss in the temporal fields of each eye. Tumour growth may also affect other nerves (such as the 3rd, 4th, 5th, or 6th cranial nerves) and cause facial pain and/or double vision or drooping of the eyelid. Headache, although a non-specific complaint, can occur when a tumour stretches the dural sac that surrounds the pituitary gland. Headache from pituitary lesions is usually frontal or retro-orbital – it may be bitemporal or radiate to the occipito-cervical region. Many patients will have been previously diagnosed with “migraine”, or “tension-headache”[8]

Although some incidentally discovered microadenomas that do not cause symptoms may be followed clinically and with repeated MRI, patients with macroadenomas generally need medical or surgical intervention. Therapeutic goals are improved quality of life and survival ; elimination of mass effect and reversal of related signs and symptoms, normalization of hormonal hypersecretion; preservation or recovery of normal pituitary function, and prevention of recurrence of the pituitary tumour.[9,10]

The minimally invasive transsphenoidal approach can be used effectively for 95% of pituitary tumours. Exceptions are those large tumours with significant temporal or anterior cranial fossa extension. In such circumstances, transcranial approaches are often

more appropriate. Occasionally, combined transsphenoidal and transcranial approaches are used. Nevertheless, some surgeons extend the basic transsphenoidal exposure in order to remove some of these tumours and avoid a craniotomy.[11,12]

The transsphenoidal approach is a versatile method for treating pituitary tumours. Endoscopic approaches may be used in isolation or as an adjunct to the other transsphenoidal approaches. Computer guided neuronavigational techniques are nearly ubiquitous at major pituitary centres in lieu of traditional fluoroscopic guidance. The role of neuronavigation is most pertinent in recurrent adenomas in which the midline anatomy has been distorted by previous transsphenoidal surgery. Intraoperative MRI is increasingly available and appears to be most applicable for large tumours. There are three basic variations of the transsphenoidal approach.[13,14]

Transsphenoidal surgery has been reported to safely reduce the pressure on the anterior visual pathway in most patients. However, it is important to predict an accurate postoperative prognosis because effective decompression by transsphenoidal surgery does not necessarily restore optic nerve function. Recently, efforts have been made to evaluate the correlation between postoperative prognosis and various clinical symptoms. Previous studies have reported age, tumour size, preoperative degree of visual field defect and visual acuity, preoperative period of symptoms, and thickness of the layer of the optic nerve fiber as factors related to the prognosis of postoperative visual field, but results vary and are at times conflicting.

Case Report

The number of patients according to gender was 71% male (10 people) while 29% female (4 people). The age distribution was found mostly at the age of 40-50 years 36% (5 people). The most common clinical symptoms were field disturbances 85% (12 people). The most preoperative vision was /6 as much as 45% (4 people). Hormonal disorders were found in 40% of urinary disorders (2 people), 40% of libido disorders (2 people), and 20% of menstrual disorders (1 person). The most visual field improvements found in 1 month postoperatively were 41% (5 people). Improvements in vision were found for 1 month postoperatively as much as 22% (2 people). Hormonal symptoms were found to improve at 1 month postoperatively as much as 60% (3 people). For complaints of headaches reduced on 3 days postoperative as much as 57% (4 people).

Table 1. Clinical symptom of patients

Time	Improvement			
	Visual fields	Visus	Hormonal	Headache
3 days	2			4
7 days	3			2
14 days	2			1
1month	5	2	3	

Table 2. Demographic of patients

	n	Percentage
Gender		
Boys	10	71%
Girls	4	29 %
Age		
20-30 years	2	16 %
30-40 years	4	28 %
40-50 years	5	36 %
50-60 years	2	16%
> 60 years	1	7%
Clinical manifestation		
Visual field deficit	12	85 %
Visual defect	9	64 %
Hormonal Problem	5	35 %
Headache	7	50 %

Discussion

Pituitary tumours account for approximately 15% of all brain tumours, and the growing tumours press against the optic chiasm, resulting in impairment of visual function manifested as visual field defects, decreased visual acuity, and decreased colour vision. As the tumours continue to grow, they impair visual function by pressing on the anterior visual pathway. In addition to impairing visual function, neurological symptoms such as headache, vomiting, dizziness, and diplopia, may occur, because the cavernous sinus, internal carotid artery, and cranial nerves III, IV, and VI anatomically surround the pituitary gland. However, among these neurological symptoms, visual symptoms, such as decreased visual acuity or visual field defect, have been found to occur most frequently. Pituitary adenoma is the most common benign tumour of the central nervous system that

can affect the optic chiasm, and the frequency of accompanying visual field defects has been reported at approximately 10% to 30% . These accompanying visual field defects are recognized as one of the primary indications for surgery on pituitary tumours.[15,16]

The optic chiasm contains nerve fibers from the optic nerves that partially decussate before forming the optic tracts. The nasal retinal fibers from each eye decussate at the chiasm to join the uncrossed temporal retinal fibers from the other eye in the contralateral optic tract. Topographic representation of the visual fields (VFs) is very precisely preserved in the chiasm.[17]

Compression of the optic chiasm by pituitary tumours generally results in selective loss of the temporal VFs, or bitemporal hemianopia, implying that the nasal retinal fibers are preferentially damaged. The reason for this preferential damage is not fully understood. One explanation is the “anatomical theory,” in which nasal fibers are selectively vulnerable because of their anatomical location in the centre of the chiasm. Various authors have suggested that the basis for this anatomical theory is that the centre of the chiasm, containing the nasal fibers, is subject to the greatest pressure and/or stretch as a result of extrinsic compression. It also has been proposed that nasal fibers in the centre of the chiasm may be more susceptible to compression induced ischemia due to the anatomy of the blood supply. Any or all of these factors may contribute to the increased susceptibility of nasal fibers to chiasmal compression. However, they should also affect the temporal fibers and hence the nasal VFs, albeit to a lesser extent. Thus, a purely anatomical theory does not, by itself, account for the highly selective damage to nasal fibers that manifests as bitemporal hemianopia often with complete sparing of the nasal fields.[18]

An alternative explanation that could account for the selective damage to nasal fibers is the crossing theory. McIlwaine et al hypothesized that nasal fibers are selectively vulnerable simply because they cross each other: crossing fibers have a smaller area of contact than fibers that run parallel to each other. Therefore, any compressive force applied to the chiasm will result in greater stress on fibers which cross, compared to those that do not. If this crossing theory is correct, nasal fibers should be particularly vulnerable to compression independent of the anatomical location of chiasmal compression. Specifically, wherever the chiasm is damaged, there should be proportionately greater involvement of the temporal fields. The crossing theory has previously been investigated using finite element modeling , but the model remains limited by a lack of precise anatomical information.[19]

Although the transnasal resection of sellar tumours is regarded a safe and efficient treatment option, complications may occur ranging from minor headaches to severe

carotid artery hemorrhage and even death. Factors influencing the postoperative outcome as well as the complication rate include age, body mass index (BMI), number of surgeries, and the surgical approach used as well as tumour size and sinus suprasellar growth. Typical vision changes and field defects include the bitemporal hemianopsia, leading to binocular vision difficulties. The visual postoperative outcome and recovery are known to be favorable, although influencing factors are currently discussed such as preoperative deficits, tumour size and tumour location, age, duration of symptoms, and tumour recurrence.[20]

Improvement in visual function has been postulated to occur in three stages: rapid recovery within minutes to a couple of days, delayed recovery over weeks to months, and late recovery over months to years. Improvement in vision may take place immediately after decompression, and visual evoked potentials have been documented to improve within 10 minutes of decompression.[21] This initial rapid recovery within the first week after surgery is well established.[1,5,8,13,15] It has been postulated that the initial improvement is the result of the removal of a physiologic conduction block.^{1,8,20} Further improvement during a stage of delayed recovery is thought to be the result of remyelination of the decompressed optic pathways.[8,10,20] Finally, late recovery of visual field over months to years has not been well studied.[13,20]

In fact, a distinction between delayed recovery and late recovery is not established. Most studies of improvement of visual function after treatment of pituitary tumours compressing the anterior visual pathways have compared pre operative visual function with visual function at a single postoperative visit.[6,12-14,16-19] Preoperative factors associated with a better visual outcome include younger age, shorter duration of symptoms, visual acuity better than 20/100, and absence of disk pallor.[13,15] Some studies have reported improvement in visual function using kinetic perimetry between the first week of surgery and a later visit. Many of these patients were also treated with radiation therapy. The follow-up of these patients varied from 1 month to 12 years in one study and was not stated in another. Thus, the course of improvement cannot be determined.[5,15]

Improvement in visual function may occur after transsphenoidal decompression of the chiasm. Because improvement in visual function may occur from a variety of proposed biologic processes, we sought to better define this potential for improvement. Potential mechanisms of axonal injury from a compressive lesion include direct disruption of conduction along the axon, impaired axoplasmic flow, demyelination with impaired signal conduction, and ischemia from compression or stretching of the blood

supply of the chiasm by the tumour. An early fast phase of improvement is consistent with restoration of signal conduction along retinal ganglion cell axons after removal of a compressive lesion. In some individuals, we observed the rapid restoration of normal vision, which would be consistent with this hypothesis. In these individuals, a physiologic conduction block is presumably the main mechanism of injury.[22]

The pattern of improvement of visual function after decompression of the anterior visual pathways suggests three phases of improvement. The early fast phase (surgery to 1 week) of improvement may lead to normalization of visual fields in some individuals. The early slow phase (1 month to 4 months) is the period of most notable improvement. A late phase (6 months to 3 years) of mild improvement does not appear significant overall but may be marked in some individuals. Each of these phases may have one or more biologic mechanisms underlying the observed improvement.[22]

Conclusion

The pattern of improvement of visual function after decompression of the anterior visual pathways suggests three phases of improvement. Improvement in visual function may occur after transsphenoidal decompression of the chiasm. Because improvement in visual function may occur from a variety of proposed biologic processes, we sought to better define this potential for improvement

References

- [1]. Schiefer U, Isbert M, Mikolaschek E, et al. Distribution of scotoma pattern related to chiasmal lesions with special reference to anterior junction syndrome. *Graefes Arch Clin Exp Ophthalmol* 2004; 242:468–477
- [2]. Hennessey JV, Jackson IM. Clinical features and differential diagnosis of pituitary tumours with emphasis on acromegaly. *Baillieres Clin Endocrinol Metab* 1995;9:271-314.
- [3]. Kerrison JB, Lynn MJ, Baer CA, et al. Stages of improvement in visual fields after pituitary tumor resection. *Am J Ophthalmol* 2000;130:813-20.
- [4]. Agarwal A, Kedar S (2015) Prognosis and treatment of visual field defects. *Semin Neurol* 35:549–556.
- [5]. Barker FG, Klibanski A, Swearingen B (2003) Transsphenoidal surgery for pituitary tumors in the United States, 1996-2000: mortality, morbidity, and the effects of hospital and surgeon volume. *J Clin Endocrinol Metab* 88:4709–4719.
- [6]. Jahangiri A, Wagner J, Han SW, Zygorakis CC, Han SJ, Tran MT, Miller LM, Tom MW, Kunwar S, Blevins LS, Aghi MK (2014) Morbidity of repeat transsphenoidal surgery assessed in more than 1000 operations. *J Neurosurg* 121:67–74.
- [7]. Juraschka K, Khan OH, Godoy BL, Monsalves E, Kilian A, Krischek B, Ghare A, Vescan A, Gentili F, Zadeh G (2014) Endoscopic endonasal transsphenoidal approach to large and giant pituitary adenomas: institutional experience and predictors of extent of resection. *J Neurosurg* 121:75–83.
- [8]. Symon L, Jakubowski J. Transcranial management of pituitary tumour with suprasellar extension. *J Neurol Neurosurg Psychiatry* 1979;42:97382.
- [9]. Goel A, Nadkarni T, Muzumdar D, Desai K, Phalke U, Sharma P : Giant pituitary tumors : a study based on surgical treatment of 118 cases. *Surg Neurol* 61 : 436-445; discussion 445-446, 2004
- [10]. Hennessey JV, Jackson IM : Clinical features and differential diagnosis of pituitary tumours with emphasis on acromegaly. *Baillieres Clin Endocrinol Metab* 9 : 271-314, 1995

- [11]. Hollenhorst RW, Younge BR : Ocular manifestations produced by adenomas of the pituitary gland : analysis of 1000 cases in Kohler PO, Ross GT (eds) : *Diagnosis and Treatment of Pituitary Tumors*. Amsterdam : Excerpta Medica, 1973, pp53-63
- [12]. Ikeda H, Yoshimoto T : Visual disturbances in patients with pituitary adenoma. *Acta Neurol Scand* 92 : 157-160, 1995
- [13]. Jaeger W, Thomann H : [German Ophthalmological Association. Recommendations for evaluation of reduced earning capacity caused by damage to vision. September 1981]. *Klin Monbl Augenheilkd* 180 : 242-244, 1982
- [14]. Bowers, C. A., Altay, T. & Couldwell, W. T. Surgical decision-making strategies in tuberculum sellae meningioma resection. *Neurosurgical focus* 30, E1, <https://doi.org/10.3171/2011.2.focus1115> (2011).
- [15]. Moher, D., Liberati, A., Tetzlaff, J., Altman, D. G. & Group, P. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. *BMJ* 339, b2535, <https://doi.org/10.1136/bmj.b2535> (2009).
- [16]. Stang, A. Critical evaluation of the Newcastle-Ottawa scale for the assessment of the quality of nonrandomized studies in meta-analyses. *Eur J Epidemiol* 25, 603–605, <https://doi.org/10.1007/s10654-010-9491-z> (2010).
- [17]. Apkarian P, Bour LJ: See-saw nystagmus and congenital nystagmus identified in the non-decussating retinal-fugal fiber syndrome. *Strabismus*2001, 9:143–163.
- [18]. Beck AD, Newman NJ, Grossniklaus HE, et al.: Optic nerve enlargement and chronic visual loss. *Surv Ophthalmol* 1994, 38:555–566.
- [19]. Miele DL, Odel JG, Behrens MM, et al.: Functional bitemporal quadrantanopia and the multifocal visual evoked potential. *J Neuroophthalmol* 2000,20:159– 162.
- [20]. Cushing H, Walker CB. Distortion of the visual fields in cases of brain tumor. IV. Chiasmal lesions with special reference to bitemporal hemianopsia. *Brain* 1915;37:341–400.
- [21]. Mackey DA. Three subgroups of patients from the United Kingdom with Leber hereditary optic neuropathy. *Eye*1994; 8:431– 436.
- [22]. Jacobson SG, Eames RA, McDonald WI. Optic nerve fiber lesions in adult cats: pattern of recovery of spatial vision. *Exp Brain Res* 1979;36:491–508.