LIST OF ABBREVIATIONS USED

RAPD	-	Relative afferent pupillary defect
BCVA	-	Best Corrected Visual Acuity
CT SCAN	-	Computerized Tomographic scan
MRI SCAN	-	Magnetic resonance imaging
CF	-	Counting fingers
HMCF	-	Hand Movements Close to Face
OD	-	Oculus Dextra (right eye)
OS	-	Oculus Sinestra (left eye)
CP	-	Cerebello-pontine
BCNV	-	Best corrected near vision
IOP	-	Intraocular pressure
Fig.	-	Figure
h/o	-	history of
s/o	-	suggestive of
No.	-	Number
VA	-	Visual acuity
DOV	-	Diminution of vision
ICSOL	-	Intracranial space occupying lesion

ABSTRACT

Background and objectives

To evaluate ocular manifestations and visual field changes in patients with intra cranial space occupying lesions. A spectrum of visual manifestations has been reported with these tumors, ranging from the presence of any visual symptoms to severe visual field defects and loss of vision. The most common visual field defect is bitemporal hemianopia. However, other types of visual field defects may also be observed. The current study was undertaken to study the ocular manifestations in patients with intra cranial space occupying lesions at a single institute over a one-year period. Ocular manifestations include blurring of vision, visual field defects, loss of colour vision, optic disc atrophy or papilloedema. Knowledge of the more common tumors will assist the ophthalmologist in advising patients on plan of surgery, and deciding which patients should be followed and which require referral neurosurgeons or radiation oncologists.

Methodology

The present one year cross sectional study was conducted in the Department of Ophthalmology, KLES Dr. Prabhakar Kore hospital and Medical Research Centre, Belagavi on patients undergoing cataract surgery during the period of 1st January 2014-31st December 2014. The study was approved by the Ethical and Research Committee of Jawaharlal Nehru Medical College, Belagavi. Fifty patients with intra cranial space occupying lesions underwent a complete ophthalmic assessment and visual field analysis using the Humphrey Field Analyzer 30-2 program. Visual acuity,

duration of symptoms, optic nerve head changes, pattern of visual field defects were noted.

Results

In present study, 50 patients with intracranial space occupying lesions are evaluated. The patient with least age in the present study was of 15yrs and the patient with maximum age was 66 yrs. The mean age of patients is 45. 90% of cases constitutes from age between 20 to 60. Male is to female ratio is 3:2. Diminution of vision (90%) was most common ocular presenting symptom. Headache was the most common systemic presenting symptom (70%) followed by seizures (2%). Maximum number of patients presented with more than one symptom. 66% of patients had the vision from 6/6-6/12, 10% patients were between 6/18-6/36 most of which presented with diminution of vision, 4% patients had vision less than 1 meter and 6% had no perception of light due to optic atrophy. Optic atrophy was the most common sign in the present study (20%).

VIth cranial nerve palsy (10%) was most commonly found in the patients of present study followed by VIIIth cranial nerve palsy (04%) and III and IV cranial nerve palsy(04%). Papilloedema was found in 9 patients. Perimetry of 13 patients showed bitemporal hemianopia and 3 patient showed homonymous hemianopia. Constriction of peripheral field were present in 7 cases and 2 cases were found to be with arcuate scotoma. Most number of intracranial lesion were found peri-chismatic area (56%). 2nd most common site was found to be temporal lobe tumours (10%) followed by cerebello-pontine angle (6%). Automated perimetry of 6 cases did not show any visual field defects. Perimetry of 5 patients could not be done due to decreased visual acuity. In the present study the most commonly found type of

tumour were the meningiomas (38%). 2nd most common site was found to be Pituitary Macroadenomas (32%),which were followed by Craniopharyngiomas(16%).

Conclusion and interpretation

If symptoms of patients of intracranial space occupying lesions are evaluated properly a detailed examination and investigations are done properly, these conditions can be diagnosed earlier. Thus prognosis is much improved as the patients gets proper treatment at an early stage. Inspite of advances made in the field of diagnostic investigations for intracranial tumours, the ophthalmologist continues to play a essential role in early detection, diagnosis and prognosis of these tumours as is indicated by a great number of ocular manifestations in our study.

Key Words: Intracranial space occupying lesions; Suprasellar tumour; Visual field; Bitemporal hemianopia; Pituitary adenomas; Craniopharyngioma; Meningiomas.

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This is to certify that the dissertation entitled **"A ONE YEAR CROSS SECTIONAL STUDY OF OCULAR MANIFESTATIONS OF INTRACRANIAL SPACE OCCUPYING LESIONS IN PATIENTS AT KLE'S DR. PRABHAKAR KORE HOSPITAL AND MRC, BELAGAVI"** is a bonafide research work done by **REG.NO. BK0113004.**

Seal & Signature of the HOD

Dr.(Mrs.) Rekha B. K **DOMS, MS , PhD**
Professor & Head,
Department of Ophthalmology,
J. N. Medical College,
Belagavi – 590010.
Karnataka, India.

Date :

Place: Belagavi

Seal & Signature of the Principal

Dr.(Mrs.)N. S. Mahantashetti **MD (Paed)**
Principal,
J. N. Medical College,
Nehru Nagar,
Belagavi - 590010.
Karnataka, India.

Date :

Place: Belagavi

INTRODUCTION

Neuro-ophthalmology deals with ophthalmic disorders with neurological problems. This subspeciality in ophthalmology is connected with other super specialities like neurology, otorhinolaryngology, neurosurgery and radiology.[1]

This research work covers the assessment of ocular manifestations in intracranial space occupying lesions (ICSOL) and also correlates the ocular manifestations and visual field defects caused by space occupying lesions**.**

ICSOL occupy physical space within the cranial cavity which results in symptoms and signs due to

- localized damage of the nearby structures
- Pressure symptoms may cause rise in intracranial pressure
- Electrical disturbance like Seizures

Intracranial space occupying lesions include primary and secondary brain tumours, haematomas, granulomatous inflammations and parasitic cysts.[3]

The intracranal tumours can be classfied into supratentorial and infratentorial group which are separated from each other by tentorium cerebelli.[4] Supratentorial group of tumours consists of cerebral hemisphere with their frontal, temporal, parietal, occipital lobes, the pituitary gland and its surrounding area, the anterior and middle cranial fossa. The infratentorial group includes the tumour of cerebello- pontine angle, the cerebellar hemisphere, the pons and the medulla oblongata.

General effect of raised intra cranial pressure includes symptoms like headache, vomiting, seizures and systemic signs include altered sensorium, hypertonicity in limbs, changes in vitals such as bradycardia, hypertension and irregular respiration.[5]

Half of the patients with intracranial space occupying lesions usually present with ocular symptoms and signs because there is a close functional link between eye and brain.

Ocular symptoms of raised intracranial pressure are deviation of eye, diplopia due to pressure palsy of sixth cranial nerve, diminution of vision depend upon site of brain lesion, sluggish pupillary reflexes, unilateral mydriasis due to pressure on third nerve, commonly occurring field defects like bitemporal hemianopia, peripheral field constriction, homonymous hemianopia, bitemporal quadrantic defects.[6]

Ocular signs associated with raised intracranial pressure are papilloedema, abducent nerve palsy and other cranial nerve palsies, sluggish pupillary reflexes, unilateral mydriasis due to pressure on third nerve, commonly occurring field defects like bitemporal hemianopia and homonymous hemianopia, nystagmus and optic atrophy.[7] In patients with intracranial space occupying lesions , manifestation of ocular symptoms and signs usually occur earlier than general signs and symptoms. Gradual progression of these focal symptoms and signs is an important sign of intracranial tumour.[8]

Always remember the fact that among twelve cranial nerves, six cranial nerves have their nuclei associated with the eyes besides the vagus and the sympathetic so any compression caused by the tumour leads to palsy of cranial nerves.[9]

60 percent of these patients with intracranial tumours sometimes first present to an ophthalmologist.[2]

Focal signs of intraocular mass lesions depend upon the site of lesion such as prefrontal tumours, frontal tumours, temporal lobe tumours, parietal lobe tumours, occipital lobe tumours, mid brain tumours, tumours of pons, cerebellar tumours, chiasmal and pituitary tumours.

Visual field is a reliable indicator for early detection of site of lesion.[10]

Earlier, Goldmannn perimeter was first kinetic perimeter with standardized background illumination, the target size and the target intensity, it has Cupula with telescope for fixation monitoring, the target projection system and recording device. The cupula has radius of 33 cm and fixed background illumination of 31.5 asb. There is a telescope at centre of cupula projecting on examiner's side for monitoring patients fixation throughout the procedure. White fixation target is situated at the aperture of the telescope at the centre of cupula. Disadvantages of Goldmann over Humphrey's perimetry are more patient co-operation is required and less sensitive in comparison with Humphrey's.[11]

The study of patients of intracranial space occupying lesions present with ocular symptoms and signs gives the clue to site of brain involved and hence its early diagnosis will benefit the patients from treatment point of view by appropriate specialists.[12] Early recognition and localization of these signs and symptoms play a crucial role in early diagnosis and management of neuro-ophthalmic disorders associated with intracranial lesions.

AIMS AND OBJECTIVES

The study was undertaken with the following aims and objectives:

Primary objective:

To study the ocular manifestations of intracranial space occupying lesions.

Secondary objective:

To study the visual field defects caused by intracranial space occupying lesions.

REVIEW OF LITERATURE

A close co-relation between neurology and ophthalmology is demonstrable in intracranial space occupying lesion.[1] The task to localize the lesion and to determine its nature demands the highest knowledge of the anatomy and physiology of intracranial structures. Individuality of symptoms and signs in such cases need to be evaluated with every available new technique, clinical and extra clinical investigations and above all a frank and free discussion between the specialists on a common platform, giving full credit to each other's contribution to the case.[13]

How far the ophthalmologist's effort can be useful to the neurologist can be judged from the extensive distance the optic pathways cover in the brain from pole to pole, and from the fact that six of the twelve cranial nerves with their nuclei are associated with the eyes besides the vagus and the sympathetic pathway.[9] The opportunity for such collaboration is thus very extensive.

INTRACRANIAL SPACE OCCUPYING LESIONS

Intracranial tumours produce ocular symptoms and signs because of damage to specific part of the brain and general symptoms and signs because of raised intracranial pressure.

TYPES OF INTRACRANIAL TUMOURS

Ocular manifestations in pituitary adenomas

Pituitary adenoma is the most common tumour to affect pituitary gland situated in the sella tursica[14] accounting for 10 to 15 % of intracranial neoplasms.[15]

The clinical manifestations are varied depending on the cell type within the tumour, hypo or hypersecretion of hormones, direction of local spread and invasion of adjacent structures.[16] Both non visual and visual manifestations are of extreme importance in the diagnosis, management and prognosis of patients with pituitary adenomas.

The normal size of pituitary gland is 5.4±0.9 mm in height, and width. Enlargement of the pituitary <10 mm is called Microadenoma. An enlargement more than this is called Macroadenoma. Because of its anatomical relation with the chiasma, the pituitary tumors cause visual field defects by compressing the optic fibres. The visual field defects are the mirror images of the optic nerve defects i.e. since compression is from below, scotomas first appear in the temporal hemifield as the crossed fibres are most susceptible, especially the inferior ones.[17] Thus the visual field seems to progress from above – below i.e. clockwise in the right eye and anticlockwise in the left eye. These defects may be complete / incomplete/ partial/ total dense. Chiasma can be prefixed [10%] or postfixed [10%]. Visual field loss patterns are different in these cases. In prefixed chiasma, optic tract would get affected first, i.e. homonymous hemianopic pattern of visual field loss and in postfixed either one of the optic nerves may be affected more, with visual field defects of one eye more, and an altitudinal pattern.[18]

In the case series studied by Anderson et al (200 consecutive cases of pituitary tumours between 1971 to 1982) only 16 % patients had decreased visual acuity and 32 % had visual field defects.[7] .

Defect in visual fields are among the most important signs occurring typically when the tumour presses upon chiasma from below, which usually

causes peripheral depression. Field changes start in supero-temporal quadrant. Bitemporal hemianopia is the hallmark of chiasmal compression. Field defect is asymmetrical in two eyes. Nasal quadrants are affected only in late stages. A central or para central scotoma may occur. (Asbury 1965)[20] (Wilson et al 1968)[21] Clarke (1963)[22] reviewed 75 cases of pituitary adenomas of which 81% cases had visual field defects.

In a study of 50 patients with chromophobe by Wilson et al (1968)[21], visual deterioration due to optic pathway compression was the main symptom. The commonest field defect was bitemporal hemianopia and central scotomatous hemianopia 94.81%). Classical bitemporal hemianopia affecting peripheral field was seen in 36% cases.

Hollenhorst and younge (1977)[23] presented analysis of 1000 cases of pituitary adenomas. Headache was the most prominent symptom. Symptoms related to vision were found in 61.3% cases of which loss of vision was found only in 0.7% cases. In 29.9% cases no visual field defect was found. In 30% cases bitemporal hemianopia was found, superior temporal defect was detected in 10.1% cases and central scotoma was found in 2.7% cases.

Visual field defects were improved in 62% of patients after surgery and in 75% of patients after surgery along with radiation therapy . In 50% patients vision remained unchanged and in 10% it worsened after surgical removal (Dhasmana et al 2011)[15]. In 1978, Savino et al[24] presented 21 patients with optic tract syndrome , out of these three patients had pituitary adenomas.

Rarely patient may present with pituitary apoplexy[25]. It is caused by rapid expansion or swelling of pituitary tumour owing it to either infarction or haemorrhage into the tumour. Patients present with the rapid, usually bilateral visual loss, diplopia, severe headache, altered sensorium (Wenya Linda et al in 2015)[26]

Sometimes patients present with diplopia because of disturbance of 3rd 4th and 6th nerves (Symonds C. 1962)[26]. Uhthoff (1995)[28] found paresis of III nerve palsy in 11% of cases, VI nerve palsy in 2% and total ophthalmoplegia in 1.5%, similar observations were made in studies of Petrohelos (1951)[29].

Marcus et al (1991)[30] studied visual parameters in 82 patients of pituitary adenomas before and after trans-sphenoidal surgery. 19 patients had normal preoperative neuro ophthalmic examination, these patients maintained normal visual acuity postoperatively. The remaining 63 patients had tumour related diminution of vision and field defects with average period duration of symptoms as 6 months. Preoperative visual acuity was 6/12 of better eye. Both visual acuity and visual field improved postoperatively in majority of eyes. Visual fields took longer to stabilize than visual acuity. With preoperative optic atrophy had a poorer visual prognosis.

Meenakshi et al (2007) studied 66 eyes out of 114 [57patients] had normal visual acuity. Also only 16 patients (28.1 %) had normal visual fields in both eyes. Bitemporal hemianopia is commonest field defect, 4 patients with abnormal extraocular movements.[31]

In 2008 Yoshifumi Okamoto et al[9] presented a series of 169 cases of pituitary adenomas. Disturbance of trigeminal nerve was found in 14 cases, in 6 of these cases there were no signs of visual pathway affection. In 10 patients, occulomotor nerve

paresis was present, 6th nerve paresis present in 7 cases and 5th nerve paresis was present in 2 cases.

C. Lamirel et al.(2012)[32] studied binocular transient visual loss usually results from intracranial processes involving the chiasmal and retrochiasmal visual pathways

Ocular manifestations in Meningiomas

Meningiomas are tumors arising from the meningoepithelial cells of the arachnoid layer of the meninges. They represent approximately 4% of all intraorbital tumours and 20% of all intracranial tumors. Orbital lesions can be primary lesions (i.e. optic nerve) or secondary lesions (i.e. extension from adjacent structures).[17]

Suprasellar Meningiomas are very slow growing brain tumours.[32] The tumors may arise first, near a part of the chiasma or only one optic nerve, thus asymmetrical chiasmal syndrome would be found. The suprasellar meningioma usually arises from the dura of tuberculum sella or anterior clinoid process and hence tends to involve the intracranial segment of optic nerve producing unilateral visual loss and central scotoma. (Spencer W H 1972).[31]

Huber (1961)[33] had studied 19 patients of meningioma presenting with unilateral loss of vision. The tumours were very slow growing and involvement of the second eye was observed after 3-20years.

Knight et al (1972)[34] reported patients of meningioma presenting as an incipient compression of perichiasmal optic nerve. These patients had near normal acuity but poor colour perception along with relative afferent pupillary defect. These patients had normal appearance of optic disc. Because of modern neuro-radiologic

techniques these patients were diagnosed early. Savino et al (1978)[24] also reported patients presenting with optic tract syndrome.[35]

Gregorius FK et al[36] in 1975 documented the central visual acuity losses in group of 23 patients with surgically and histological verified suprasellar meningiomas. The pattern demonstrated was that of acute, gradual or fluctuating loss in one eye, followed by later loss of central visual acuity in the other eye. Both optic nerves and chiasma were invariably involved either by stretching or compression. Neither preoperative field abnormalities nor central acuity deficits could be correlated with the anatomical location of the tumour, nor could postoperative changes in vision be correlated with tumour size. Lengthy duration of visual acuity loss and severe visual deficit did not preclude postoperative recovery of vision. Improvement in sight most frequently occurred within the first several weeks after operation, and further return of vision was not noted after 1 year.

Kitthisak et al (2008)[8] focused on 69 patients with Suprasellar tumours among which 19 (28%) presented with Suprasellar meningiomas

Ocular manifestations in Craniopharyngiomas

Craniopharyngioma is a type of brain tumour derived from pituitary gland embryonic tissue, that occurs most commonly in children but also in men and women in their 50s and 60s. People may present with bitemporal inferior quadrantanopia leading to bitemporal hemianopia, as the tumour may compress the optic chiasma.[37]

It has a point prevalence of approximately 2/100,000. Craniopharyngiomas are also known as Rathke pouch tumors or hypophyseal duct tumours.[38]

Clinical presentation :

In children, headache was more common. This was associated with projectile vomiting. Ocular signs and symptoms of raised intracranial pressure dominate the clinical presentation in more than 50% of the children. Mental sluggishness, somnolence and memory loss may be also present.

Tumour invasion in third ventricle was common resulting in internal hydrocephalus. Papilloedema was common fundus sign sometimes followed by optic atrophy (Wegnar et al 1943).[39]

Extraocular muscle paralysis in craniopharyngioma are result of direct compression of respective nerves; occulomotor nerve palsy is the most common.[40]

See saw nystagmus is sometimes seen in patients of craniopharyngioma.[41]

Concomitant strabismus and paralytic strabismus because of palsy of III, IV nerve were reported by Hoff (1972)[42] and Kennedy (1975).[43] Savino et al (1978)[24] presented 8 patients presenting as optic tract syndrome, afferent pupillary defect, optic atrophy, incongruous homonymous hemianopia.

Symptoms of mental disturbances in patients of craniopharyngioma were also noted by Kennedy (1975).[43]

A patient of craniopharyngioma presenting as a strabismic amblyopia had been reported by T. Oshim et al (1982)[41]. Strabismus is not uncommon in children with craniopharyngioma. Concomitant squint, esotropia, or exotropia with marked visual loss in squinting eye may be present as demonstrated in a study by T.Oshim et al (1982)[45].

Visual field defect in craniopharyngioma

The typical field defect was seen as bitemporal hemianopia starting in lower quadrant progressing to blindness.

Bitemporal hemianopia was present in 27% cases. Other field defects observed were homonymous hemianopia and perichiasmal field defects (Kennedy et al 1975).[43]. (Savino et al 1978)[24]

In a case series study performed by Kennedy et al (1975)[43] visual field defect were observed in 50% of children and 94% of young adult. He also found that in 22% of patients distinct changes took place from one type of field defect to another type of field during the course of the tumor. An important observation made by Sofela et al in 2014[46] was the prechiasmal field defects, which were present in significant number of patient, that is almost 20%. The most common of pattern was central or paracentral scotoma in one eye with no defect or temporal depression in other eye.

Kitthisak et al(2008) focused on 69 patients with Suprasellar tumours among which 17 (25%) presented with craniopharyngioma.[8]

Tumours of Frontal lobe :

In tumours of frontal lobe ocular signs are rare because of its relatively distant position from visual pathway. General symptoms because of raised intracranial tension were more pronounced. Mental changes, psychotic disturbances are often found.

Slowly progressive mono ocular visual loss with central scotoma is significant clinical ocular manifestations, caused by pressure on underlying optic nerve.

Aphasia develops if the tumour occupies the speech area in the dominant hemisphere. Focal motor and jacksonian seizure in contralateral limbs may accompany with other signs and symptoms.[47]

Foster-Kennedy syndrome, an ipsilateral optic atrophy with contralateral papilloedema was generally considered as very characteristic of frontal lobe tumour, but very rarely it occurs in typical form.[48] Visual field defects were irregular, incongruous homonymous hemianopia may occur or paracentral scotoma on the side of optic atrophy may occur. (Huber 1961)[3] (Barcia D, Barcia JC 1964)[49]

Exophthalmos is occasionally demonstrated with frontal lobe tumours due to direct invasion of orbit or stasis in the cavernous sinus.

Pandit Y. (1965)[50] demonstrated that aphasia develops if the tumour occupies the speech area in the dominant hemisphere. Focal motor and jacksonian seizure in contralateral limbs may accompany with other signs and symptoms.

Dr. K.V.Raju et al (2009)[51] found that patients with frontal lobe tumours showed behavioral abnormalities, dementia, seizures, and urinary incontinence. In the study of 50 cases, gliomas were the most common tumour in frontal lobe, 9% of the patients showed homonymous hemianopia.

Tumours of Temporal lobes :

The most common tumours of temporal lobe are Gliomas. Subjective and objective signs in tumours of temporal lobe had localizing value. Seizures with aura, olfactory or gustatory hallucinations and visual hallucinations of formed objects, distinctive in nature are characteristic.

Most important objective sign is visual field changes – incongruous superior homonymous quadrantanopia or incongruous superior homonymous hemianopia are very pathognomic of temporal lobe lesions.

Smith et al (1962)[52] reviewed 100 cases of homonymous hemianopia. Out of these, 24 patients had temporal lobe lesions (14 proved and in 10 diagnosis was based on clinical findings only), out of these 24 cases tumours were found in 18 patients, vascular lesion in 2 , other causes in 4. Optokinetic nystagmus sign was negative in 19, positive in 3 and not recorded in 2. Vision was normal in 21 patients. In visual field changes, typical Meyer's loop superior quradrantinopia occurred in 18 cases, complete hemianopia in 6 patients and field defects were incongruous in 14 cases.

Harrington (1961)[53] also presented 4 patients of temporal lobe lesions in whom typical incongruous hemianopia was found.

In a study of intracranial space occupying lesions by Dr. K.V.Raju et al (2009)[51] 20% of cases with temporal lobe lesions had superior quadrantanopia and 25% of cases showed homonymous hemianopia.

Lesions of parietal lobe:

Lesions in anterior part of parietal lobe produces syndrome of post central gyrus, sensory jacksonian attack. Lesions in the non-dominant lobe produces hemiparesis, abnormal posturing of limbs and constructional apraxia. Lesions of dominant lobe produce alexia, agraphia and acalculia. Visual field defects are rare, usually complete homonymous hemianopia was found. Positive optokinetic nystagmus was present in many cases.

Huber (1961)[33] had 27 case of parietal lobe tumours .He found sensory disturbances and jacksonian attacks in 7 patients. In his series he found visual field changes were less frequent, occurring only if extensive down-growth was present. Inferior homonymous quadrantanopia, which is considered as a typical of parietal lobe lesion defect was less frequently found (only in 2 cases). While complete homonymous hemianopia was present in 7 cases.

Smith (1962)[52] in his review of 100 cases of homonymous hemianopia found parietal lobe lesions in 33% of cases which mostly occurred in older age groups. The ratio of tumour and vascular lesions was equal. Optokinetic nystagmus sign was positive in 28 of 33 cases. Splitting of macula was present in each of these cases.

Tumours of Occipital lobe

Vascular lesions of occipital lobe are more common than tumours. Except for ocular symptoms, it is otherwise neurologically silent zone. Visual hallucination of unformed, primitive nature can be seen in some cases. Typical field defect is congruous homonymous hemianopia with macular sparing.

Huber (1961)[33] reported 20 cases of occipital lobe tumour in his case series study, visual hallucinations of simple, primitive nature were present only in 3 cases. In all 20 patients, visual field changes were present. Complete homonymous hemianopia with sparing of macula was observed. In 4 patients other froms of field defects like inferior qudrantanopia field defect were present. In 17 patients, papilloedema was seen.

Harrington et al (1961)[52] reported 3 cases of lesion of occipital cortex presenting as congruous homonymous hemianopia. In his review of 100 cases of

homonymous hemianopia found occipital lobe lesions as commonest cause, representing 39% of cases. Average age of patients was 50 years. Optokinetic nystagmus sign was negative in 31 cases. In patients with vascular lesion optokinetic nystagmus sign was found to be negative and in patients with neoplastic lesion this sign was positive. Field defects were mostly congruous. Splitting of macula was commonly seen with the ratio 3:1 over sparing.

Roux et al (2001)[53] presented a review of 104 patients with isolated homonymous hemianopia presenting to ophthalmologists, cerebral infarction was diagnosed in 39% of patients. Most of the patients were in between the ages of 50 and 70 years. Majority of patients had history of some vascular disease . He presented 6 cases of occipital lobe tumour , in which optokinetic nystagmus was absent.

Cerebello-Pontine Angle Tumour:

Among these tumours acoustic neuroma is most common. These tumours have characteristic presentation along with cerebellar signs, ocular signs and symptoms caused by tumour are important.

VII and VIII nerve involvement is early. Diminished or abolished corneal reflex and hearing loss is present on ipsilateral side. There is horizontal nystagmus seen towards the side of lesion. Signs of raised intracranial tension are also present. Huber (1961)[3] reported such 31 cases, out of which 25 tensions were acoustic neuroma, 6 were other tumours like meningioma , astrocytoma and medulloblastoma. Ocular symptoms and hearing disturbances were early symptoms.

Baloh R.W (1995)[54] studied 12 patients of C-P angle tumour and evaluated vestibular tests. 9 to 12 patients had spontaneous nystagmus and 11 cases had

significantly abnormal caloric responses. In 5 patients optokinetic nystagmus (slow component) was abnormal. He also documented that lesions in cerebellum due to paraneoplastic syndrome begins with rapidly progressive ataxia of the trunk and extremities.

Even after the tumour is removed, the cerebellar deficit persists in most patients.

Dr. K.V. Raju et al (2009)[33] found 20% cases in his study were of cerebello-pontine tumours all of them had deafness, ataxia , impaired corneal sensation , papilloedema and VII and VIII nerve palsies. Other features also include gaze nystagmus.

Early diagnosis and role of ophthamologist

More than 60% patients with brain tumour present with ocular signs and symptoms (Huber 1961)[33]. Hence ophthalmologists play a vital role for early diagnosis. Importance of failing vision for early diagnosis of intracranial tumours was also stressed by Dony et al (2012)[55] and in their study of 28 and 50 cases of suprasellar meningioma. In these patients symptomatology was almost entirely confined to diminution of vision and disturbances of visual fields. Operative intervention was easy and prognosis was better in patients in whom diagnosis was made when tumour was of small size (Dony et al 2012)[59].

(Courtesy by: Dr. A.K. KHURANA)

Lesion of 1.Optic nerve 2. Proximal part of optic nerve 3. Central chiasma 4. Lateral chiasma 5. Optic tract 6. Geniculate body 7. Part of optic radiation in temporal lobe 8. Part of optic radiation in parietal lobe 9. Optic radiation 10. Visual cortex sparing the macula 11. Visual cortex only macula

Figure 1 . Visual field examination with Goldmann's perimetry *(Courtsey by Dr. A.K. Gupta, Visual fields)*

Earlier, Goldmannn perimeter was first kinetic perimeter with standardized background illumination, the target size and the target intensity, it has Cupula with telescope for fixation monitoring, the target projection system and recording device. Disadvantages of Goldmann over Humphreys perimetry are more patient co-operation is required and less sensitive in comparison with Humphreys.[11]

Review according to frequency of intracranial tumours

James Segal et al (1975)[14] reported a study of 38 patients of pituitary adenoma, 63.2% cases presented with diminution of vision. In 41.8% cases physician caused delayed diagnosis of more than one month after presentation was found. The reason for this delayed diagnosis were found to be inadequate history taking or failure to perform adequate testing including visual field testing, colour perception testing failure to provide follow up examinations.

The importance of all these tests were stressed in studies Knight (1972)[34] and Melen C (1987)[57] for early diagnosis and to improve prognosis.

Retrospective case-series review was conducted. Women who presented with visual deterioration either during pregnancy or in the early post partum period due to an intracranial tumor were included. Neurosurgical and obstetrical data were collected from the patients' hospital files and outpatient clinic records. Between 2005 and 2011, nine pregnant women with visual deterioration were diagnosed and treated.

In Huber's study (1976)[33] of 1166 patients of intracranial tumours frequency of each type is shown in the following table.

Types of Tumour	With papilloedema	Without papilloedema	Total
Glioblastomas	177	171	348
Astrocytomas	154	78	232
Meningioma	110	61	171
Metastatic lesions	68	43	111
Medulloblastoma	42	09	51
Haemangioma	21	18	39

Craniopharyngioma	11	02	13
Others	115	86	201
TOTAL	698	468	1166

Cranial nerve palsies in intracranial space occupying lesions :

Incidence;

Rush J.A et al (1992)[56] analysed a series of 1000 patients of acquired paralysis of III , IV, VI cranial nerves. They had given the distribution of nerves affected as ;

Number cases and cranial nerves affecetd :

Cranial nerve	Rush j.a (1992)[56]
III	290
IV	172
VI	419
Multiple	119
Total	1000

Vimla Menon et al (1987)[58] reviewed 197 cases of ophthalmoplegia and gave the distribution of nerves affected as :

Isolated III N. palsy -32.0%

Isolated IV N. Palsy -6.1%

Isolated VI N. Palsy -44.6%

Multiple Nerve palsy- 17.3%

In 1992 Kodsi S.R [59] reported reviews of 160 paediatric patients with acquired palsies of III, IV and VI cranial nerves. His reports were comparable with reports of adults (>18yrs) given by Rush in 1992.

Cranial nerve involved	Adults (>18 years) (Rush 1992)[56]	Paediatric (0-17 years) (Kodsi 1992)[59]
VI	44%	55%
III	24%	22%
IV	21%	12%
Multiple	11%	11%
TOTAL	100%	100%

Age group :

Kodsi S.R. (1992)[59] reviewed 160 paediatric patients with oculomotor palsies with age ranging from 0-17 years (mean age 9.4 years).

Kodsi S.R. (1992)[59] gave the incidence of various causes , comparing incidences of adult population , as follows:

CAUSE	ADULT (Rush1992)[56]	PAEDIATRIC PATIENTS (Kodsi 1992)[59]
Undetermined	26.9%	14.4%
Trauma	15.4%	42.5%

Neoplasm	15.2%	16.9%
Vascular	16.9%	--
Aneurysm	4.5%	1.3%
Other	21.1%	25.0%
TOTAL	100%	100%

OCULOMOTOR NERVE PALSY in intracranial occupying lesions

Incidence:

Incidence of III nerve palsy was given by Rush J.A (1992)[62] as 29.0% and Vimal Menon (1987)[64] had given 32.0%. Incidence in paediatric patient was found to be 22% (Kodsi S.R.1992)[59].

Age group affected :

As previously described, age range in paediatric was 0-17 years (Kodsi S.R.1992)[59].

Rush J.A. (1992)[56] studied III nerve palsies in patients with age range 2-71 years. But 90% were above the age of 19 years.

Causes :

Frequency of various causes as given by Rush J.A. (1992)[56] and Kodsi J.R (1992)[59], is given in following table :

CAUSE	Rush J.A (1992)[56]	Kodsi S.R (1992)[59]
Undetermined	23.1%	17.1%
Head trauma	16.2%	40.0%

Neoplasm	11.7%	14.3%
Vascular	13.8%	--
Aneurysm	13.8%	--
Other	14.5%	28.6%
TOTAL	100%	100%

In 2007. Kim SH et al[60] reviewed 12 patients with pituitary tumour and cranial nerve palsy. In this study occulomotor nerve was found to be most frequently affected.

ABDUCENT NERVE PALSY in intracranial space occupying lesions

Incidence :

Incidence of VI nerve palsy was given by Rush J.A. (1992)[56] found 70% of VI nerve palsies over the age of 50 years. 30% cases were seen in the 7th decade.

Causes :

Frequency of various causes as given by Rush J.A. (1992)[56], and Kodsi S.R. (1992)[59], each showed in following table:

CAUSE	Rush J.A (1992)[56]	KodsiS.R (1992)[59]
Undetermined	29.6%	14.8%
Head trauma	16.7%	42.0%
Neoplasm	14.6%	20.5%
Vascular	17.7%	--
Aneurysm	3.6%	--

Other	17.9%	22.7%
TOTAL	100%	100%

TROCHLEAR NERVE PALSY in intracranial space occupying lesions

Incidence :

Incidence of IV nerve palsy was given by Rush J.A. (1992)[56] as 17.2% , Vimla Menon et al (1987)[58] as 6.1% and by Kodsi S.R.(1992)[59] as 12%.

Age group :

In paediatric age group (<17 years) most common cause of IV nerve palsy was trauma. Mean age was 9.4years. age range was 19-76 years.

CAUSE	Rush J.A (1992)[56]	Kodsi S.R(1992)[59]
Undetermined	62	4
Head trauma	55	7
Neoplasm	7	1
Vascular	32	--
Aneurysm	3	--
Other	13	7
Total	172	19

James R Keane (2005)[61] did the consecutive study of the 979 cases of multiple cranial nerve palsies and found the following results.

Cause	**Overall cases of C N palsies**
1) Intracranial tumours	305
2) Vascular diseases	128
3) Trauma	128
4) Infection	102
5) Guillian barre syndrome	62
6) Fishers syndrome	29
7) Cavernous sinusitis	56
8) Surgical complications	54
9) Multiple sclerosis	54
10) Other	110
TOTAl	**1028**

CRANIAL NERVE	OVERALL CASES OF CRANIAL NERVE INVOLVED
II	155
III	339
IV	143
V	353
VI	565
VII	466

VIII	180
X	220
XI	98
XII	163

He concluded that the locations and causes of multiple cranial neuropathy are highly diverse, the fact that intracranial tumour composes more than one quarter of cases a premium on prompt diagnosis.

Onakpoya Oluwatoyin Helen (2009)[62] et al did a retrospective study on patients with brain tumours in the Neurosurgical Unit of Obafemi Awolowo University Teaching Hospitals Complex , lle –lf from January 2003 to December 2007.

Out of a total of 94 patients ,88 with complete information were reviewed. There were 53 [60.2%] males and 35 [39.8%] females; the mean age was 36.2±20 years . 14 [15.9%] patients were referred by Ophthalmologists. Meningiomas (36.4%) craniopharyngioma ((13.6%) and gliomas (9.1%) were the most common brain tumours encountered. Fifty nine (67.9%) had visual complaints at presentation; poor vision (46.6%) and double vision (12.5%) were the most common ocular symptoms while optic atrophy was the commonest ocular sign (23.9%), 46 (52%) were blind while 14(16%) had visual impairment.

In 2009, Dr.K.V.Raju et al[51] studied 50 cases of diagnosed cases of intracranial space occupying lesions. He found that female patients in the 40-50 yrs age group were commonly affected. Headache was the common symptom [63.3%] followed by diminution of vision . VII cranial nerve was most commonly

involved followed by VIth cranial nerve palsy. Papilloedema was the most common sign. Visual field defects correlated with the site of tumour, whereas, cerebellopontine angle tumours were most common according to the site of tumour.

In 2012, Dony et al[55] encountered that among all intracranial lesions most commonly ocular manifestations are seen with Sellar and Suprasellar tumours. Papilloedema was the most common sign followed by optic atrophy.

MATERIAL AND METHODS

The present study was conducted in the department of ophthalmology, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi. All patients attending Ophthalmology OPD and ophthalmology ward, neurosurgery OPD and neurosurgery ward with neuroimaging proved intracranial space occupying lesions at the Tertiary eye care centre.

STUDY DESIGN :

A Cross Sectional Study

STUDY PERIOD:

One year – 1st January 2014 to 1st December 2014

STUDY SETTING:

- Ophthalmology OPD and ophthalmology ward, neurosurgery OPD and neurosurgery ward at the Tertiary eye care centre.

SAMPLE SIZE:

Sample size for the study is calculated by following formula:

$n = \frac{4p}{d^2}$,

n = sample size

p = prevalence of the major risk factor

q = 100 – p

d = absolute error i.e. 10% of p

But the incidence of disease is less so according to KLES Hospital statistics:

From: January 2012 to December 2012 : 45

January 2011 to December 2011 : 46

January 2010 to December 2010 : 40

Sample size of my study will be 50

Selection criteria:

Inclusion criteria

- Patients with CT or MRI proved intracranial space occupying lesions.

Exclusion criteria

- Rapidly deteriorating general health conditions.
- Marked behavioral disorders.
- Uncooperative patients.
- Unwilling to participate.

  Approval from Institutional Ethic Committee was obtained in December 2013.

METHODOLOGY

All patients who satisfy inclusion criteria will form the subjects of the study, will be called to Ophthalmology OPD for further evaluation and written informed consent will be taken from them by investigator.

In each case clinical evaluation was done after obtaining a detailed history.

- History of blurring of vision.
- History of headache.
- History of giddiness.
- History of nausea and vomiting.
- History of proptosis.
- History of drowsiness
- History of ptosis.
- History of diplopia.
- History of seizures.
- History of loss of appetite.
- History of loss of hair
- History of gynaecomastia.
- History of any previous chemotherapy taken.
- Past history of DM, HTN, STROKE.
- Past histoy of any tumour

A complete examination of the central nervous system which included examination of the higher mental function, motor system, sensory system , cerebellar signs

Cranial nerves examination:

- Olfactory : Test smell sensation with soap, toothpaste etc.
- Trigeminal: motor function of masseter, pterygoid and temporalis. Sensations over the face , corneal reflex, jaw jerk.
- Facial : palpebral fissure, frowning, symmetry of blinking, eye closure, nasolabial folds, angle of mouth, blowing , whistling, showing the teeth, epiphora, dribbling of saliva.

  Power of individual facial muscle.

  Upper half of face escaped or not.

  Taste sensation of anterior 2/3 of tongue.
- Vestibulococchlear: hearing watch test, rinnes test, webers test. Positional nystagmus

This is followed by ocular examination that includes visual acuity testing for Distance vision (using Green's chart) Near vision (using New Roman chart)

Colour vision charting (using Ishihara charts).

Ophthalmologic assessment included routine ocular examination under torch light with special reference to ocular movements which include uniocular movements: adduction abduction elevation depression binocular movements: versions and vergence

Corneal sensation with a cotton wick

Pupillary abnormalities: direct light reflex, consensual light reflex,rapid afferent pupillary defect, nystagmus.

Intra ocular pressure will be measured with Non contact tonometer.

Detailed Ocular fundus evaluation with direct as well as indirect ophthalmoscope using 20 D lens.

Visual fields charted by Confrontation method, Bjerrums charting, Humphry field analyser.

Protocol for visual field analysis: The commonly performed test is full threshold program of Humphreys perimeter. Perimetrist should first enter the patients demographic data and clinical details. Test is performed at central 30 degree suprathreshold (30-2) is selected. Size III stimulus central 76 points are taken.

Patients best corrected vision is placed in trial lens holder. The eye not being tested is occluded. Adjust the patient's chin and forehead and make the patient comfortably seated at the perimeter bowl and explain when to press the response button. Patient's head is centrally placed so that eye to be tested is directly opposite the central fixation light. Room lights should be dimmed and trial holder is positioned in front of eyelashes. Instruct the patient that he/she has to fix the eye to central fixation point and should not look at other lights, and when the light is perceived they need to press the response button. For patients who are performing the test for the first time can undergo one minute demonstration test.

For measuring the foveal threshold patient is asked to look at the centre of small diamond which consists of four lights below the central fixation light. By doing this foveal sensitivity is measured .

Computer is programmed to present stimuli at four locations 9° from horizontal and vertical meridian also known as primary points. These are checked twice. 6 other points are used to calculate short term fluctuations like any point with + or - 5 decibel then expected similar age population

The advantage of this test is it takes just ¼ th of time for detection of neuro ophthalmic defect, without reducing the sensitivity.

Figure 2. An ophthalmologist examining visual fields by Humphrey's field analyser

Anterior segment examination by Slit lamp .

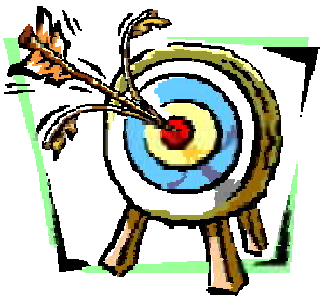
Ocular symptoms along with ocular manifestations in various types of lesion and prevalence of different type of field defects depending upon the site of lesion.

Correlation between CT scan and automated perimetry findings.

The study requires the following investigations to be performed on patients :

1. **CT or MRI**
2. **Visual field charting**
3. **Evaluation of Optic Disc**

Introduction



Objectives

Review of Literature

Methodology

Results

Discussion

Conclusion

Summary

Bibliography

Annexure-I

Annexure-II

Annexure-III

Annexure-IV

OBSERVATIONS AND RESULTS

Table I: Showing gender wise distribution of brain tumours in present study

Sex	Number of cases	Percentage
Male	30	60%
Female	20	40%
Total	50	100%

Male is to female ratio is 3:2

Fig. 1: The number of males more than females in present study.

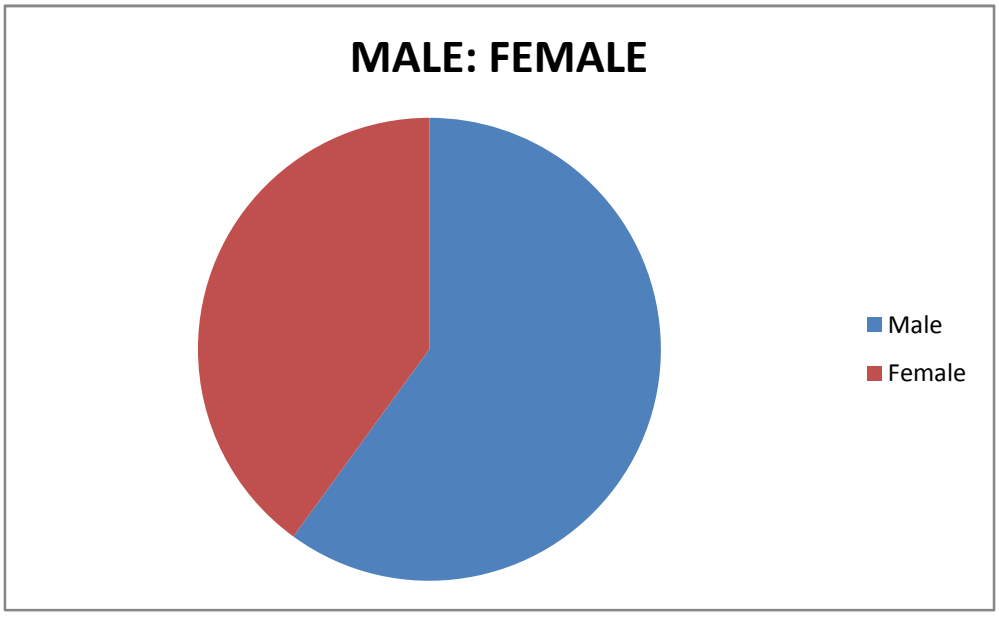
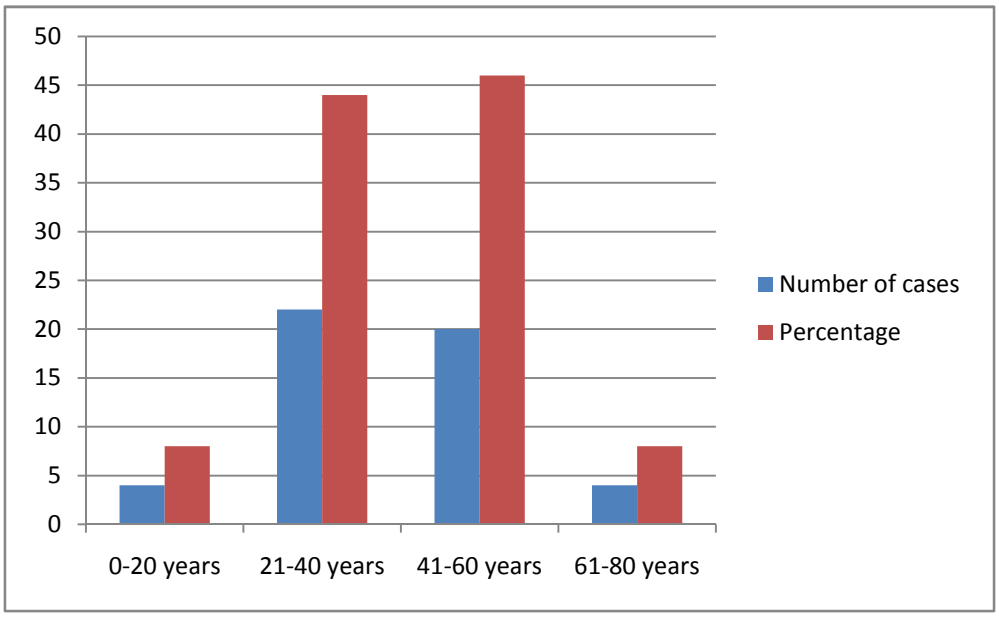


Table II : Showing age incidence in present study

Age Group	Number of cases	Percentage
0-20 years	4	8
21-40 years	22	44
41-60 years	20	46
61-80 years	4	8
Total	50	100

Fig. 2: Maximum number of patients belonged to age group 21-40 years.



The mean age of patients is 40.8 ± 15 yrs.

The patient with minimum age in the present study was of 15yrs and the patient with maximum age was 66 yrs.

90% of cases constitutes from age between 20 to 60.

Graph depicting the age wise distribution of various intracranial tumours

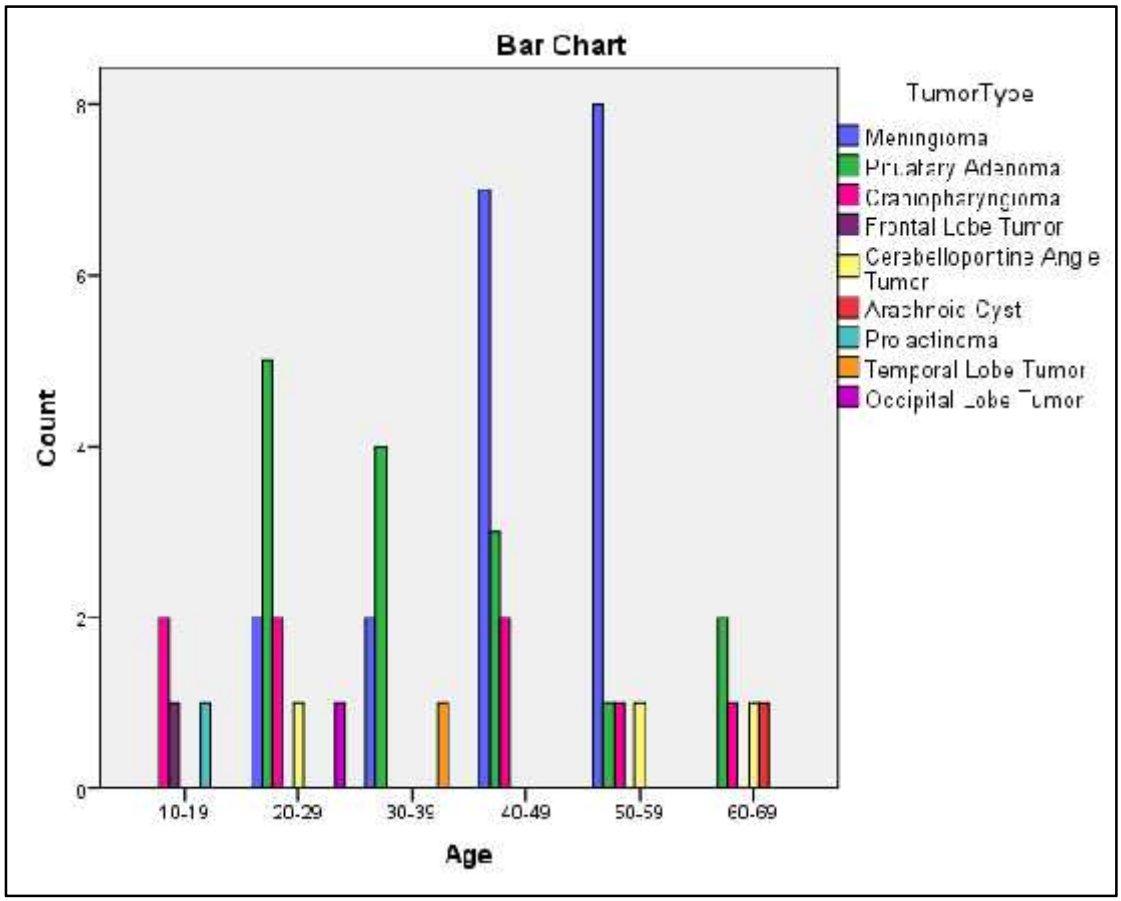


Table III : Showing distribution of cases according to presenting symptoms

Symptoms	Number of cases	Percentage
Headache	35	70%
Diminution of vision	45	90%
Diplopia	04	08%
Deviation of eye	03	06%
Deviation of angle of mouth	02	04%
Projectile vomiting	06	12%
Drooping of eyelids	01	02%
Seizures	01	02%
Hearing loss	02	04%
Protrusion of eyeball	03	06%
Scotoma in front of eye	02	04%
Slurring of speech	02	04%
Ataxia	01	02%
Generalized weakness	03	06%
Head injury	03	06%

Table shows the main presenting symptoms in the present study.

Diminution of vision (90%) was most common ocular presenting symptom

Headache was the most common systemic presenting symptom (70%) followed by seizures (2%).

Maximum number of patients presented with more than one symptom.

Fig. 3: Showing distribution of cases according to presenting symptoms

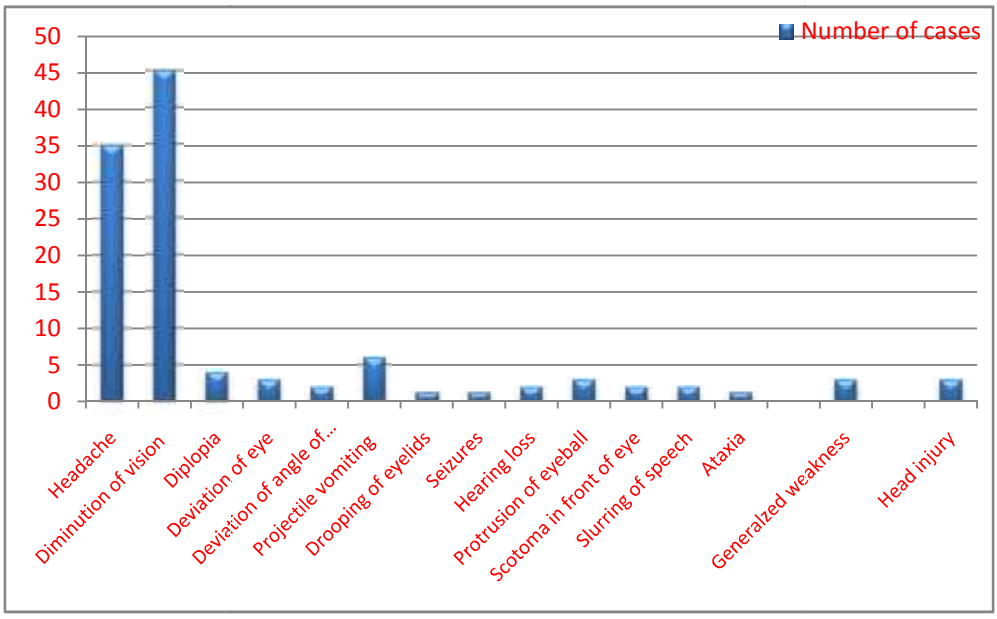
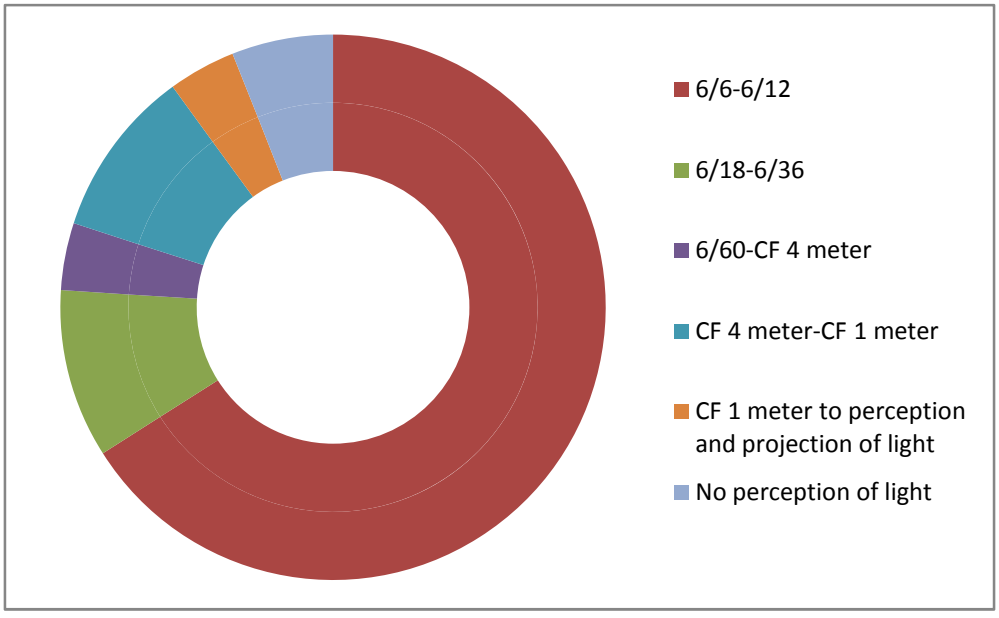


Table IV : Showing visual acuity of patients in the present study

Visual Acuity (Distant)	Number of Cases	Percentage
6/6-6/12	33	66%
6/18-6/36	05	10%
6/60-CF 4 meter	02	04%
CF 4 meter-CF 1 meter	05	10%
CF 1 meter to perception and projection of light	02	04%
No perception of light	03	06%
Total	50	100

Fig. 4: Shows the distribution of visual acutity in patients with intracranial tumours.



66% of patients had the vision from 6/6-6/12, 10% patients were between 6/18-6/36 most of which presented with diminution of vision, 4% patients had vision less than 1 meter and 6% had no perception of light due to optic atrophy.

Table V : Showing distribution of cases according to signs

Sign	Cases	Percentage
Papilloedema	09	18%
VI cranial nerve palsy	05	10%
VII cranial nerve palsy	02	04%
VIII cranial nerve palsy	02	04%
III cranial nerve palsy	02	04%
IV cranial nerve palsy	02	04%
Optic atrophy	10	20%
Abnormal pupillary reactions	18	36%
Proptosis	01	02%
Nystagmus	01	02%
Glaucomatous disc	02	04%

Optic atrophy was the most common sign in the present study (20%).

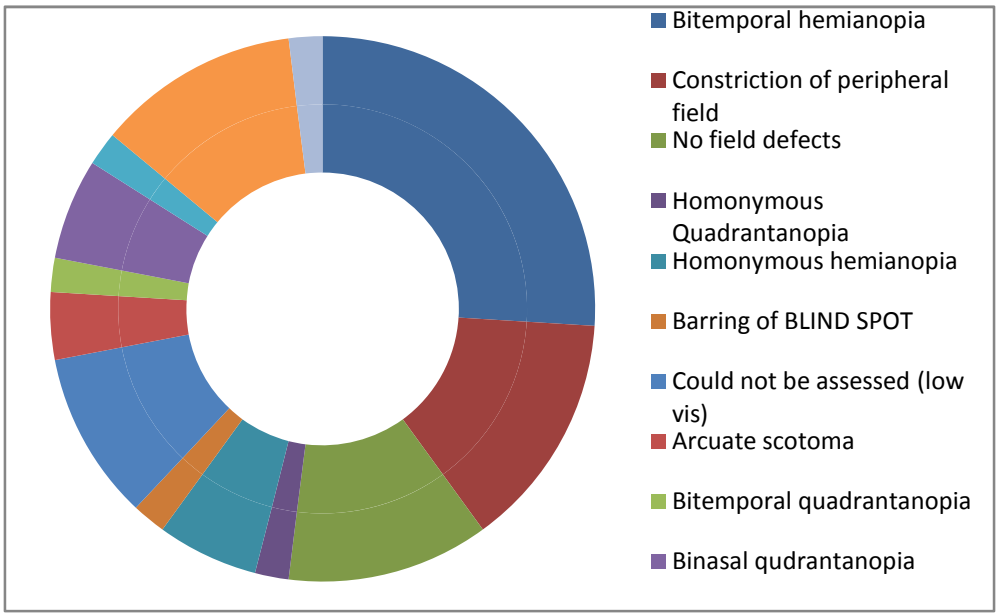
VI cranial nerve palsy (10%) was most commonly found in the patients of present study followed by VIII cranial nerve palsy (04%) and III and IV cranial nerve palsy(04%).

Papilloedema was found in 9 patients .

Table VI : Showing visual field defects in the present study

Nature of field defects	Number of cases	Percentage
Bitemporal hemianopia	13	26%
Constriction of peripheral field	07	14%
No field defects	06	12%
Homonymous quadrantanopia	01	2%
Homonymous hemianopia	03	06%
Barring of Blind spot	01	02%
Could not be assessed (low vision)	05	10%
Arcuate scotoma	02	04%
Bitemporal quadrantanopia	01	02%
Binasal qudrantanopia	03	06%
Tubular vision	01	02%
Unilateral temporal defects	06	12%
Binasal hemianopia	01	02%
Total	50	100%

Fig. 5: Showing visual field defects in the present study



Perimetry of 13 patients showed bitemporal hemianopia and 3 patient showed homonymous hemianopia.

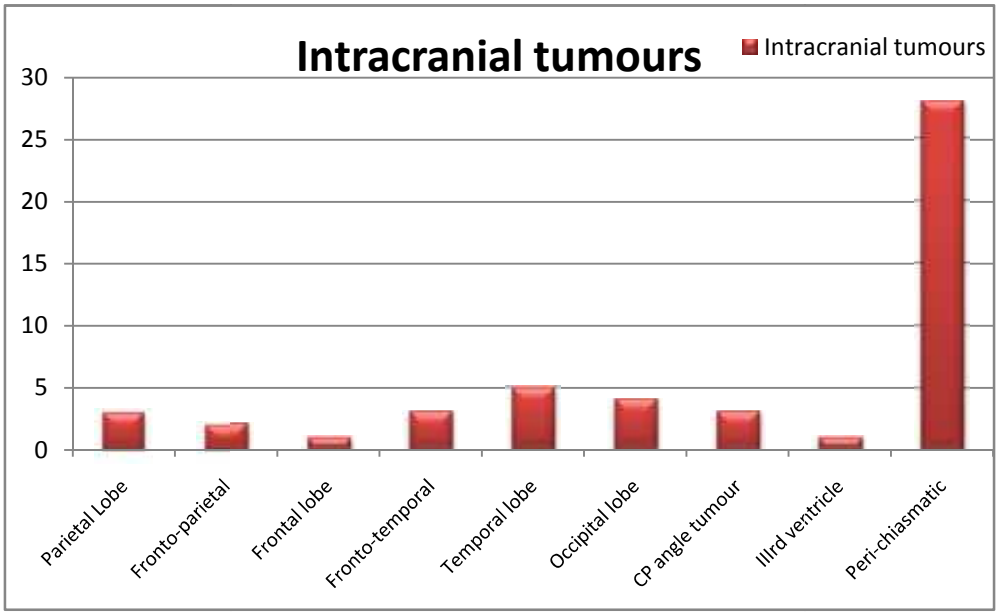
Constriction of peripheral fields were present in 7 cases and 2 cases were found to be with arcuate scotoma.

Automated perimetry of 6 cases did not show any visual field defects. Perimetry of 5 patients could not be done due to decreased visual acuity.

Table VII : Shows the distribution of intracranial lesion according to site in the present study

Site	Intracranial tumours
Parietal Lobe	03
Fronto-parietal	02
Frontal lobe	01
Fronto-temporal	03
Temporal lobe	05
Occipital lobe	04
CP angle tumour	03
III ventricle	01
Peri-chiasmatic	28
Total	50

Fig. 6: Shows the distribution of intracranial lesion according to site in the present study



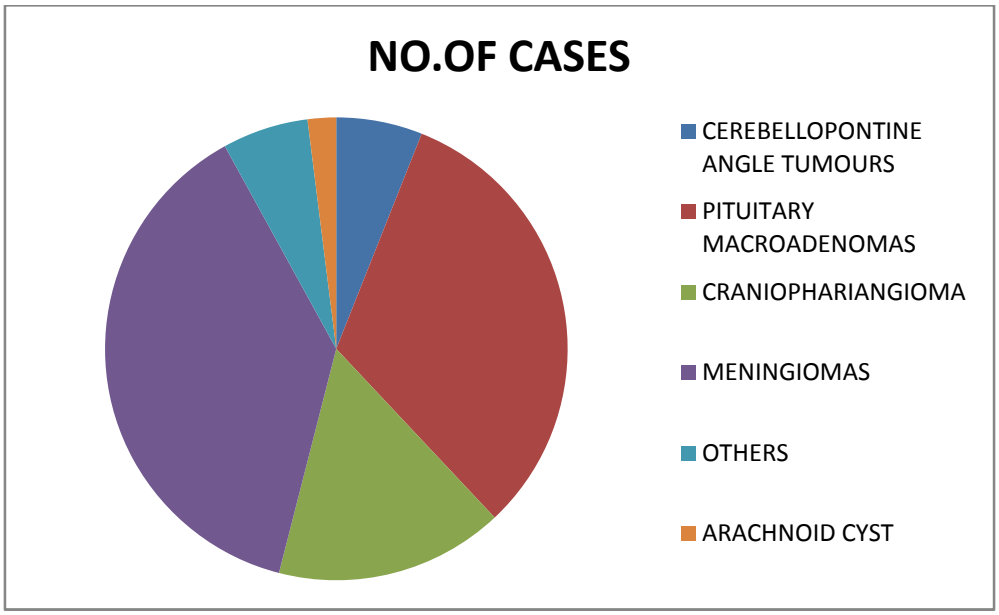
Most number of intracranial lesion were found in perichismatic area (56%).

2nd most common site was found to be temporal lobe tumours (10%), followed by cerebello pontine angle (6%)

Table VIII : Shows the type wise distribution of intracranial space occupying lesion in the present study

Type of intracranial tumour	No.of cases	Percentage
Cerebellopontine angle tumours	3	9%
Pituitary macroadenomas	16	32%
Craniopharyngioma	8	16%
Meningiomas	19	38%
Others	3	6%
Arachnoid cyst	1	2%
Total	50	100%

Fig. 7: Shows the type wise distribution of intracranial space occupying lesion in the present study



In the present study the most commonly found type of tumour were the meningiomas. (38%).

2nd most common site was found to be Pituitary Macroadenomas (32%), which were followed by Craniopharyngiomas (16%).

DISCUSSION

The presenting symptoms would vary according to the neuro -ophthalmic problem and so would be different in different studies. In our study, majority of patients presented with more than one symptom, either headache or diminution of vision , diplopia, drooping of eye lids and deviation of eyes etc.

Headache (70%) and diminution of vision (90%) were most frequent symptoms in present study. Projectile vomiting (12%) was the 3rd most common presenting symptom . Diplopia in 4 cases, deviation of eye was present in 3 cases. Drooping of eyelids (2%) , proptosis (6%) , and inability to close eye (4%) were other less common ocular presenting complaints.

Seizures (2%), deafness (4%) , ataxia (1%) , slurred speech (2%), memory loss (2%) and deviation of angle of mouth (2%) were also present in some cases.

Diminution of vision (90%) was most common ocular presenting symptom

Headache was the most common systemic presenting symptom (70%) followed by seizures (2%).

Maximum number of patients presented with more than one symptom (as per table III)

In Snyder's study (1993)[65], headache was the most common presenting symptom in patients of brain tumours.

Onakpoya Oluwatoyin Helen et al (2009)[62] reported 59 cases out of 88 cases had diminution of vision (46.6%). Diplopia was found in 11 cases (12.5%) ,

protrusion of eye in 2(2.3%) , drooping of eyelid 2 (2.3%) and eye deviation in 1(1.1%) cases.

In Huber's study (1961)[33] of 1166 cases intracranial tumours, headache was most frequent symptom.

Dr. K.V.Raju et al al (2009)[51] found in his 50 cases study that Headache was the most common presenting symptom (63.%) which usually occurred with other symptoms which was followed by diminution of vision (50%).

According to Wadud et al (2014)[1] common neuro-ophthalmological findings were blurring of vision (91.07%), visual field defect (71.42%), optic disc changes (50%), pupillary light reaction defect (48.21%) and colour vision defect (46.42%).

66% of patients had the good visual acuity from 6/6-6/12, 10% patients were between 6/18-6/36 most of which presented with diminution of vision, 4% patients had vision less than 1 meter and 6% had no perception of light due to optic atrophy (as per table IV)

James Segal et al (1987)[14] reported a study of 38 patients of pituitary adenoma, 63.2% cases presenting with decreased vision.

Melen C (1987)[11] also found that diminution of vision was the major presenting complaint in most of the patients.

In Hollenhorst's study (1973)[64] of 1000 cases of tumour, diminution of vision was present in 61.3% of cases.

Kennedy et al (1975)[45], reviewed 45 cases of brain tumours. In 60% patients visual acuity was reduced.

Dr. K.V Raju et al (2009)[51] performed a study on 50 proven cases of diagnosed intracranial tumours and found that about 50% of cases diminution of vision during the course of the disease.

Optic atrophy was the most common sign in the present study (20%) followed by papilloedema in 18% cases.

VI cranial nerve palsy (10%) was most commonly found in the patients of present study followed by VIII cranial nerve palsy (04%) and III and IV cranial nerve palsy were found in 8% cases. Papilloedema was found in 9 patients (as per tabe V)

About 18 cases had the pupillary defects at the time of presentation, proptosis was noticed in 1 patient and 1 patient presented with nystagmus.

This is in agreement with the study of Petrohelos (1951)[29] and where 40% cases had normal fundus and 60% cases had papilloedema.

Later studies of Huber A. (1976)[13] had shown 59% incidence of papilloedema in patients with brain tumours.

They attributed this decline in incidence to modern diagnostic methods, which had made early diagnosis possible, so fewer patients reached the stage of papilloedema. Incidence of papilloedema in infratentorial tumours was 70% and in supratentorial tumours 60% (Huber,1976)[13].

Huber (1976)[13] had given incidence of papilloedema in rapidly growing tumours as 50% , 65% in slowly growing tumours and 61% in cerebral metastasis .

Incidence of intracranial tumours is less in children and very old patiEnts , but papilloedema can observed at almost any age.

Neacsu et al (2008)[4] studied 446 patients of brain tumours and concluded that about 47% of patients were having papilloedema at the time of presentation in different stages.

Onakpoya Oluwatoyin Helen et al (2009)[62] found papilloedema in 18 cases (20.5%), pupillary defects in 12 cases (13.6%) of 88 cases.

Dr. K.V Raju et al (2009)[51] performed a study on 50 proven cases of diagnosed intracranial tumours and found that 56% patients with papilloedema at the time of presentation.

Wadud (2014)[1] had shown papilloedema in 80% of patients with brain tumours.

VI cranial nerve palsy (10%) was most commonly found in the patients of present study followed by VIII cranial nerve palsy (04%) and III and IV cranial nerve palsy was seen in 8 cases.

Rush J.A et al (1992)[56] analysed a series of 1000 patients of acquired paralysis of III, IV, VI cranial nerves. And found that abducent nerve was most commonly affected in any intracranial lesion.

The various etiologies of these cranial nerve palsies indicates that the intracranial space occupying lesions are the 3rd most common cause in the study.

Rush J.A . in (1992)[56] gave incidence of III cranial nerve as 29.0% and also mentioned that intracranial tumours stands as 3rd most common cause of oculomotor nerve palsy as per their study.

The incidence of oculomotor nerve palsy in paediatric patient which was found to be 22% (Kodsi S.R. 1992)[59].

Incidence of oculomotor nerve palsy in present study is found to be 4% and lateral rectus palsy is seen in 10 % cases.

Rush J.A. (1992)[56] found VI cranial nerve palsy in 41.9%.

Study by Kodsi S.R (1992)[59] found that 55% of cases presented with VI cranial nerve palsy of various intracranial etiologies which is compatible with present study.

James R. Keane (2005)[61] did the consecutive study of the 979 cases of multiple cranial nerve palsies and Dr. K.V. Raju et al (2009)[51] found in his 50 cases study that VII cranial nerve was the most commonly involved followed by VI cranial nerve.

Automated Perimetry of 13 patients showed bitemporal hemianopia and 3 patient showed homonymous hemianopia.

Constriction of peripheral field were present in 7 cases and 2 cases were found to be with arcuate scotoma (as per table VI)

Automated perimetry of 6 cases did not show any visual field defects. Perimetry of 5 patients could not be done due to decreased visual acuity.

1 case showed barring of blind spot.

Bitemporal hemianopia was the hallmark of chiasmal compression. Field defects were asymmetrical in two eyes.

Clarke (1963)[22] reviewed 75 cases of pituitary adenomas. Visual field defects were found in 81% cases.

In a study of 50 patients with pituitary adenomas by Wilson et al (1968)[19], visual deterioration due to optic pathway compression was the main symptom. The commonest field defect was bitemporal hemianopia and central scotomatous hemianopia (48.1%). Classical bitemporal hemianopia affecting peripheral field was seen in 36% cases. In 2001, Roux[55] reported a case which presented with hemianopic scotoma.

Hoyt (1989)[12] also described 2 such cases, Kearns and Rucker (1966)[66] also reported such cases.

Field loss in our study is varied depending upon the site of tumour and helped in localization. Result of present study are comparable with several above mentioned studies.

Dr. K.V. Raju et al (2009)[51] found the enlargement of blind spot as the most common finding followed by bitemporal hemianopia.

Most number of intracranial lesions were found in the perichiasmatic region, that is in sellar and suprasellar region (56%). 6% of tumours were found in cerebello-pontine angle region of which mostly are acoustic neuromas, that is in the posterior fossa of skull (as per table VII)

Compared with Hubers study of 1166 patients of intracranial tumours, number of cases in present study was small (100 cases) but the results are found to be in accordance with Hubers results.

Pituitary adenoma:

In present study, we had 16 patients of pituitary macroadenomas. All of them presented with slowly progressive diminution of vision, and 6 of those had primary type of optic atrophy in both eyes. These cases also presented with symptoms like headache and projectile vomiting along with diminution of vision. Visual field examination showed classical bitemporal hemianopia in 8 patients and 2 cases showed supra-temporal quadrantanopic field defect, 1 patient had generalized constriction of fields.

Pituitary adenomas are second most common manifestation seen in present study.

2 patients presented with III, IV and VI cranial nerve palsy and abnormal pupillary reactions were found in 8 patients. Diagnosis was confirmed on MRI-SCAN head with orbits.

Presentation of our cases was comparable with study of Clarke (1963)[22] and Hollenhorst et al (1973)[64].

In Hollenhorst's study of 1000 patients of pituitary adenoma, symptoms related to vision were found in 60% of cases, optic atrophy was seen in 40% of cases, in 70% of cases visual field defects were seen, in 30% of cases bitemporal hemianopic defect and in 10% cases supra-temporal defect was found[64].

Wadud et al (2014)[1] noted pituitary adenoma was the most common type of intracranial tumor (58.04%).

Craniopharyngioma:

In present study, 8 patients had craniopharyngioma. 5 of them presented with headache and 2 with deviation of eyes inwards. Field defect was classical bitemporal hemianopia in 5 patients, 1 with arcuate scotoma, 1 with homonymous hemianopia. 5 cases presented with gradual progressive diminution of vision, 3 of which had primary type of optic atrophy in either eye, 2 cases had RAPD and 1 with ill sustained reaction and MRI had confirmed the diagnosis.

The result of present study are comparable with the results of kennedy et al (1975)[35] in their series, 60% of cases had optic atrophy and 40% cases had papilloedema. Bitemporal hemianopia was present in 27% cases and peri-chiasmal defects in others.

In a study by Wadud et al (2014)[1] incidence of Craniopharyngioma is 20.53%.

Meningioma:

Meningioma is the most common intracranial manifestation seen in our study followed by Pituitary Adenomas.

19 patients of meningiomas were found in present study most of which presented with symptoms like headache and projectile vomiting and 5 patients presented with papilloedema in either of the eye. 3 of the cases were frontal lobe meningiomas which also presented with proptosis of eye. 4 were temporal lobe

meningioma which also had seizures.1 with malignant meningiomas and 6 presented with optic atrophy. On MRI scan final diagnosis was made.

Results are comparable with Huber's study, in his study all patients with meningiomas presented with papilloedema.[33]

But in study of Knight et al (1972)[34] one patient presented as slowly developing optic atrophy.

Tumours of third ventricle:

In present study we found 1 case of third ventricle tumour but there was no visual field defect noted.

It may cause chiasmal syndrome either by direct pressure from above or because of internal hydrocephalus. Visual field defects were irregular either asymmetrical bitemporal hemianopia or homonymous hemianopia (Huber 1976)[33].

Acoustic neuroma:

In this study 3 cases of Cerebello-pontine angle tumour, all were acoustic neuromas. Many of these had papilloedema and 1 patient had post-papilloedemic optic atrophy in both eyes. 1 patients had nystagmus . Most of these cases had V, VII and VIII nerve involvement. These results are comparable with Huber's study (1961)[33] of 31 cases of C-P angle tumour which had cerebellar signs, nystagmus and papilloedema in late stages.

Dr. K.V.Raju et al (2009)[51] found 20% cases in his study of cerebello-pontine tumours all of them had deafness, ataxia, impaired corneal sensation,

papilloedeoma and VIIth and VIII th nerve palsies. Other features also include gaze nystagmus.

Gliomas:

Gliomas are the most common intracranial tumours but in my study none of the cases of intracranial tumours presented with glioma

Dr. K.V.Raju et al (2009)[57] found gliomas most common tumour in frontal lobe in study of 50 cases. 9% of which had homonymous hemianopia.

The 3 cases of cerebello pontine angle tumours were found in the present study, all were acoustic neuromas.

In present study meningiomas were found to be the most common intra cranial space occupying lesion

Walsh documented gliomas in most number of cases in his study of brain tumours.

According to Dr.K.V. Raju et al (2009)[57] astrocytomas were the most common intracranial tumour in their study.

A study by Wadud et al (2014)[1] concluded that the pituitary adenoma is the most common tumor that impairs the visual pathway structures followed by craniopharyngioma, posterior fossa tumour & meningioma.

According to gender incidence, male to female ratio in our study in general was 3:2 (shown in Table I)

Mehrazin M et al (2006)[63] presented a study of 3,437 cases of brain tumors for over twenty five years and found that about 55% of cases were males and 45% were females between the age group 30-50 years.

Onakpoya Oluwatoyin Helen et al (2009)[62] showed out of 88 patients 53(60.2%) were males and 35(39.8%) were females.

Dhasmana et al (2011)[15] presented a study on 18 patients diagnosed with Pituitary Adenomas found 10 males and 8 females.

According to age incidence, as seen from table II. Patients in all age groups were affected.

Maximum number of patients belonged to age group 21-40 years.

The mean age of patients is 40.8 ± 15.4 yrs.

The patient with least age in the present study was of 15yrs and the patient with maximum age was 66 yrs.

84% of cases constitutes from age between 20 to 60.

According to Huber,[33] incidence of intracranial tumour is more after 40 years and in our study 52% cases of intracranial tumours were above 40 years.

Mehrazin et al (2006)[63] presented a study of 3,437 cases of brain tumours for over twenty five years and concluded that about 79% cases was between the age group 30-50 years.

According to Dr. K.V Raju et al (2009)[51] the maximum incidence of intracranial space occupying lesions were between 40-50(30%).

Dhasmana et al (2011)[15] presented in their study that mean age of patients was 35.1 yrs

CONCLUSION

If symptoms of patients of intracranial space occupying lesions are evaluated properly, a detailed examination and investigations are done properly, these conditions can be diagnosed earlier. Thus prognosis is much improved as the patients gets proper treatment at an early stage.

Inspite of advances made in the field of diagnostic investigations for intracranial tumours, the ophthalmologist continues to play an essential role in early detection, diagnosis and prognosis of these tumours as is indicated by a great number of ocular manifestations in our study.

SUMMARY

There are a large variety of ocular manifestations associated with intracranial tumours. In Sellar, Suprasellar and Parasellar tumours the ocular manifestations are an outstanding feature of the clinical picture. The ocular signs and symptoms are of equal significance with other neurological signs and symptoms in temporal lobe, parietal lobe and infratentorial tumours. Diseases of central nervous system like tumours, infections, circulatory disorders, vascular lesions, demyelinating diseases, certain hereditary and degenerative diseases may present in form of ocular manifestations. Hence picking up of these ocular manifestations not only helps in early diagnosis of intracranial tumours and referral of the patients but also helps in reducing morbidity and mortality of the patients. Hence, this study covers the assessment of incidence of ophthalmologic manifestations in intracranial space occupying lesions and correlate the ocular manifestations with the site of brain tumours. Also about the effect on visual fields due to intracranial tumours

Neuro-ophthalmology is subspeciality of ophthalmology and neurology which deals ocular ailments which arises due to diseases involving central nervous system.

A Cross sectional study of 50 cases of intracranial space occupying lesions presenting with ocular complaints in ophthalmic outpatient department, neurosurgery outpatient department and neurosurgery ward in a tertiary care centre were studied during the period of 1 year. (Jan 2014- Dec2015).

History of the patients was taken carefully from the patients as well as from the relatives. Various ocular symptoms and signs were noted. Necessary investigations which are helpful in exact diagnosis of lesion like Automated

Perimetry and Magnetic Resonance Imaging or Computerized Tomography Scan were done.

Those patients who were uncooperative , deteriorating general conditions and marked behavioural changes , patients with demylineting diseases, vascular lesions, degenerative and hereditary diseases were excluded from the study.

Maximum number of patients belonged to age group 20-40 years. Diminution of vision (90%) was the most common ocular presenting symptom followed by diplopia (8%). Headache was the most common systemic presenting symptom (70%). Optic atrophy is commonly seen sign in the present study (20%) followed by Papilloedema seen in 10% cases. VI cranial nerve palsy (10%) was most commonly found in the patients of present study followed by III(4%), IV(4%), VII(4%) and VIII cranial nerve palsy (4%). Automated perimetry of 13 patients showed bitemporal hemianopia and 3 patient showed homonymous hemianopia. Most number of intracranial lesions were found in peri-chiasmatic(56%) area comprising of pituitary macroadenomas, meningiomas and craniopharyngiomas. 2nd most common site was found to be temporal lobe tumours (18%), and cerebello pontine angle tumour was seen in 9% cases. In the present study the most commonly found type of tumour was the Meningioma (38%), followed by pituitary macroadenomas (32%). The ocular manifestations due to intracranial space occupying tumours correlated with the site in which they are present.

Hence the following conclusion was drawn from the present study performed.

1. Majority of patients of intracranial space occupying lesion are between 20-40 years of age.
2. The ocular manifestations due to intracranial space occupying tumours correlate with the site in which they are present.

3. Main symptom of patients of intracranial space occupying lesions are headache and diminution of vision, hence patients coming with these symptoms should be evaluated properly.
4. Diminution of vision is the commonest ocular symptom in present study
5. Headache is the commonest systemic symptom in present study.
6. Maximum patients have false localizing signs or signs of systemic neurological involvement at the time of presentation.
7. Common ocular signs of raised intracranial tension in cases of intracranial space occupying lesions are optic atrophy (20%) and cranial nerve palsies.
8. Among intracranial space occupying meningiomas are commonest (36%) followed by Pituitary Macroadenomas (32%).
9. Among the cranial nerve palsies, oclumotor nerve (10%), facial nerve palsies (4%), troclear nerve, abducent nerve and auditory nerve sometimes multiple nerve palsies are common.
10. Visual fields must be tested in every patient complaining of disturbances of vision, without any obvious ocular cause to account for it.
11. Bitemporal hemianopia is most commonly found visual field defects followed by peripheral constriction of fields.
12. Plain radiographs of skull and orbit are not much helpful for exact diagnosis of patients of intracranial space occupying lesions.
13. To arrive at the exact anatomical localization and diagnosis of lesion Magnetic Resonance Imaging scan is mandatory.

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ANNEXURE-I

CONSENT FOR PARTICIPATION IN RESEARCH STUDY

IP/0P No. _____

You are invited to participate in our research study titled **"**A one year cross sectional study of ocular manifestations of intracranial space occupying lesions at KLE'S DR. Prabhakar Kore Hospital and MRC, Belgaum.**"** conducted

Respected Sir/Ma'am we request you to enroll yourself in our study as you are eligible for participation. Your participation in research is voluntary. If you decide to participate you are free to withdraw at any time.

Need of the Study: The purpose of research is to determine the ocular manifestations like blurring of vision, diplopia and visual field changes which manifest in cases of intracranial space occupying lesions. Early diadnosis of ocular manifestations reduces ocular defects caused by the tumours.

Procedure Involved: If you agree to enroll yourself in this study, you will be asked your present, past and family history. Ocular examination will be done once you are diagnose with intracranial space occupying lesion on the basis of your CT and MRI report. You will be examined on direct and indirect ophthalmoscope which are non invasive procedures and a visual field charting will be done to record your visual field changes.

Risks and Benefits: There are no risk involved, however some complications may occur primarily due to the disease process itself. Your participation may benefit you

and others with the same condition in future, by helping us learn more about the disease process. No financial incentives are promised for being a part of the study.

Alternatives: If you are not willing to participate you will be treated according to the existing protocol & it will not affect your relationship with this hospital.

Costs for participating in this research: There will not be any extra cost incurred by you. You will, however, have to pay for the investigations which are part of the existing management protocol for the condition. There is no commitment for any reimbursement or any other compensation.

Privacy and Confidentiality: Your privacy is guaranteed. However, your medical records can be directly accessed and reviewed by authorized individuals or by the ethics committee. Records, which could reveal your identity, will be kept confidential. Personal data will remain anonymous if data is being published or written as a dissertation.

Authorization to Publish Results: When the results of the research are published or discussed, in a conference, no information will be displayed that would disclose your identity.

Compensation: In the event of injury related to the study, treatment will be made available through KLES Dr.Prabhakar Kore Hospital & MRC, Belgaum. There is no compensation or payment for such medical treatment by law. The doctors and the staff will provide facilities and medical attention to you.

Questions

If you have any questions about the research you may please contact:

1. Chairman, Dr. GANGA S. PILLI, Department of Pathology, JNMC, Belgaum.

CONSENT FOR PARTICIPATION IN RESEARCH TRIAL

I, Mr./Ms./Mrs _____ voluntarily agree for the participation as a subject of this study. By signing this consent form, I am not giving up any of my legal rights. I may withdraw from the study at anytime. I am signing the consent form after having read or been read for me in my own vernacular language, including the risks and the benefits and having all my questions answered.

Subject Name : _____

Signature or the Left Thumb Print of Subject : _____

Witness Name :_____

Signature of Witness:_____

Investigators Name: _____

Signature of Investigator : _____

Date: Place:

Name of Guide:_____

Signature of guide:_____

Name of Co Guide:_____

Signature of Co giuide_____:

ANNEXURE-II

DATA COLLECTION FORM

"A ONE YEAR CROSS SECTIONAL STUDY OF OCULAR MANIFESTATIONS OF INTRACRANIAL SPACE OCCUPYING LESIONS IN PATIENTS AT KLE'S DR. PRABHAKAR KORE HOSPITAL AND MRC, BELGAUM"

CASE NO. : DATE :

1. PATIENT OPD/IPD NO. ☐

2. NAME:_____

3.INFORMANT: SIGNATURE:

4. AGE: ☐ yrs

5.SEX: ☐ (1-male, 2-female)

6. OCCUPATION:_____

7. ADDRESS :

_____

8. TELEPHONE
No.(s)_____

9. IS THE PATIENT ELIGIBLE FOR STUDY: ☐ 1-YES 2-NO

10. HAS INFORMED CONSENT BEEN GIVEN? ☐ 1-YES 2-NO

11. SYMPTOMS**:**

	RIGHT EYE	LEFT EYE	DURATION
A. HEADACHE	☐		
1- YES			
2- NO	☐		
(If 1, then, 1=mild, 2=moderate, 3=severe)			
B. DIMINUTION OF VISION	☐	☐	
1- YES		☐	
2- NO	☐		
(If 1,then, 1.painless progressive gradual	☐	☐	
2. painful static sudden			
3. painful static gradual			
4. painless progressive sudden)			
C. DIPLOPIA	☐	☐	
1- YES			
2- NO	☐	☐	

D. PTOSIS

1- YES

2- NO

(If 1, then, 1=mild, 2=moderate,

3=severe)

E. PROPTOSIS

1- YES

2- NO

F. DEVIATION OF EYE

1- YES

2- NO

G. VOMITTING

1- YES

2- NO

H. ANY HEAD INJURY

1- YES

2- NO

(If 1, then, 1=Agricultural, 2=RTA

3=domestic, 4=others)

I. CONVULSIONS ☐

1- YES

2- NO

J. LOSS OF HAIR

1-YES

2-NO

K. LOSS OF APPETITE ☐

1-YES

2-NO

L. ANY OTHER COMPLAINTS ☐

_____

_____

12. DRUG HISTORY: (1- YES, 2- NO)

1. ANTIBIOTIC ☐ IF YES SPECIFY _____
2. ANTIBIOTIC + STEROID ☐ IF YES SPECIFY _____
3. ANTICANCER DRUG ☐ IF YES SPECIFY _____
4. ANY OTHER MEDICATIONS ☐ IF YES SPECIFY _____

13. PAST HISTORY (1- YES, 2- NO) **DURATION**

1. HYPERTENSION ☐
2. DIABETES MELLITUS ☐
3. SYPHILIS ☐
4. TUBERCULOSIS ☐
5. ANY OTHER TUMOUR ☐
6. PARASITIC INFESTATIONS ☐
7. HIV ☐
8. ANY SIMILAR COMPLAINTS IN PAST ☐

14. PERSONAL HISTORY:

PERSONAL HISTORY OF ☐ (1- SMOKING/ TOBACCO CHEWER, 2- ALCOHOLISM, 3-BOTH, 4- ALOPECIA, 5- GYNAECOMASTIA, 6-BOWEL BLADDER COMPLAINTS 7. NONE)

DIET ☐ (1- VEG, 2- NON VEG, 3- MIXED)

15. GENERAL PHYSICAL EXAMINATION

PALLOR ☐ (1- PRESENT, 2- ABSENT)

OEDEMA ☐ (1- PRESENT, 2- ABSENT)

LYMPHADENOPATHY ☐ (1- PRESENT, 2- ABSENT)

PULSE: _____/ MINUTE

BLOOD PRESSURE: _____mmHg

TEMPERATURE: _____(1- AFEBRILE, 2- FEBRILE)

16. SYSTEMIC EXAMINATION (1-Normal, 2-Abnormal, If Abnormal, specify : _____

CARDIO VASCULAR SYSTEM: ☐

RESPIRATORY SYSTEM: ☐

PER ABDOMEN: ☐

CENTRAL NERVOUS SYSTEM: ☐

HIGHER MENTAL FUNCTION

CONCIOUSNESS

BEHAVIOUR

EMOTIONAL STATE

ORIENTATION

MEMORY

INTELLIGENCE

SPEECH

CRANIAL NERVE EXAMINATION (1-Normal, 2-Abnormal, If Abnormal, specify : _____)

OLFACTORY ☐

OPTIC ☐

OCCLUMOTOR ☐

TROCHLEAR ☐

TRIGEMINAL ☐

ABDUCENT

FACIAL ☐

VESTIBULOCOCHLEAR ☐

GLOSSOPHARYNGEAL ☐

VAGUS ☐

ACESSORY ☐

HYPOGLOSSAL ☐

MOTOR FUNCTIONS

NUTRITION

TONE

POWER

COORDINATION

INVOLUNTARY MOVEMENTS

SENSORY FUNCTIONS

EXTROCEPTIVE

PAIN

TOUCH

TEMPERATURE

PROPRIOCEPTIVE (1- Present, 2- Absent)

VIBRATION SENSE ☐

MUSCLE SENSE ☐

PRESSURE SENSE ☐

JOINT SENSE ☐

POSITION SENSE ☐

CORTICAL

ONE POINT LOCATION

TWO POINT DISCRIMINATION

STEREOGNOSIS

GRAPHAESTHESIA

REFLEXES

GAIT

17. OCULAR EXAMAINATION:

HEAD POSTURE ☐ (1- ERECT, 2- HEAD TILT, 3- FACE TURN, 4- CHIN ELEVATION)

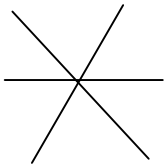
FACIAL SYMMETRY ☐ (1- SYMMETRICAL, 2- ASYMMETRICAL)

VISUAL AXIS ☐ (1- PARALLEL, 2- DEVIATED)

EXTRA OCULAR MOVEMENTS (1- NORMAL, 2- RESTRICTED, 3- PARTIALLY RESTRICTED,4- HyPERTROPIA, 5- HYPOTROPIA, 6- EXOTROPIA, 7- ESOTROPIA)

RIGHT LEFT

Uniocular 

Binocular 

- **Visual Acuity**

Vision

	RE	LE
Unaided		
Pin-hole		
Spectacles		

NEAR VISION

Unaided
spectacles

- Refraction

	RE				LE			
	Sphere	Cylinder	Axis	Vision	Sphere	Cylinder	Axis	Vision
Distance								
Near								

COLOUR VISION

(1-Normal; 2-Abnormal, if abnormal specify: _____)

- **Adnexa**

(1-Normal; 2- Ptosis; 3- Retraction of lids; 4- Proptosis)

- **Conjunctiva**

(1-Normal; 2- Conjunctival congestion; 3- Circumcorneal congestion

4- Chemosis; 5- Subconjunctival haemorrhage)

- **Cornea (** 1.clear, 2. Hazy, 3. Oedematous)

Corneal sensation (1. Presnt, 2. Absent)

- **Sclera**

(1- Normal; 2- Congested; 3- episcleritis, 4- Scleritis)

- **Anterior Chamber**

o Depth :

(1-Normal depth ; 2-Shallow, 3- Deep, 5- Irregular)

- **Iris**

(1=Normal colour and pattern; 2-Iritis; 3- Iridodonesis; 4- Synechiae)

- **Pupil**

(1-Round,regular,reacting; 2-Abnormal)

If 2, then specify3-RAPD)

- **Lens**

(1=Clear; 2=Cataractous; 3=details not made out

If 2, then, 1=immature; 2=mature; 3=hypermature; 4- Aphakia;

5- Pseudophakia; 6- Pseudophakia with PCO)

18. OCULAR INVESTIGATIONS: (1- NORMAL, 2- HYPERTONY, 3- HYPOTONY)

INTRA OCULAR PRESSURE (mmHg)

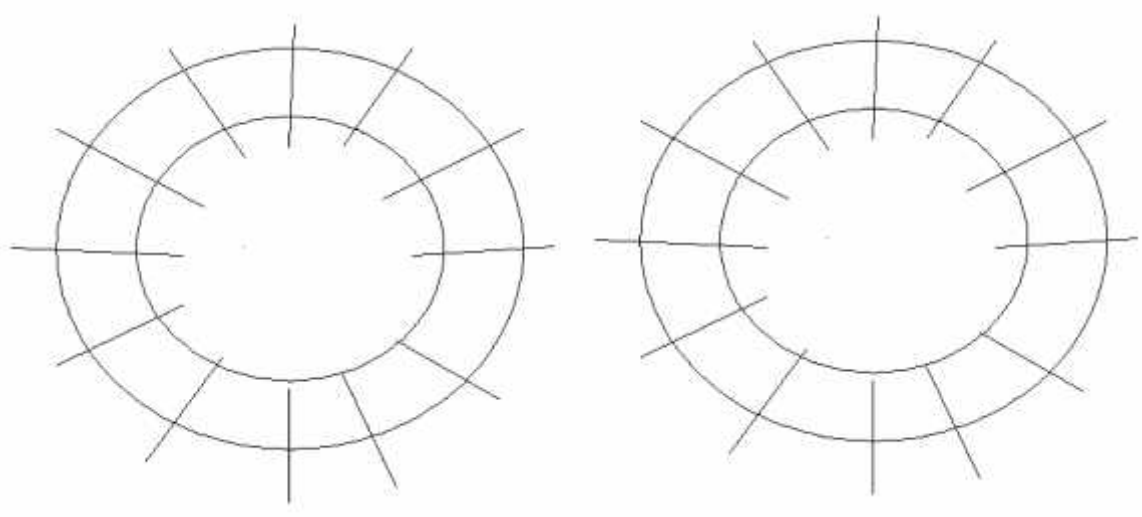
DIRECT OPHTHALMOSCOPY:

FUNDUS	RE	LE
GLOW		
MEDIA		
DISC		
C:D RATIO		
BLOODVESSELS		
BACKGROUND		
MACULA		

IMPRESSION;

INDIRECT OPHTHALMOSCOPY:

RIGHT LEFT



IMPRESSION:

BJERRUMS CHARTING:

AUTOMATED PERIMETRY :

19. INVESTIGATIONS: (1- Normal, 2- Abnormal if abnormal specify: _____)

- CT ☐
- MRI ☐

2O. DIAGNOSIS :

21. TREATMENT GIVEN :

ANNEXURE III – PHOTOGRAPHS

CASE -1

- A 54 year old elderly female came to eye opd with c/o of

  Diminution of vision since 4 months

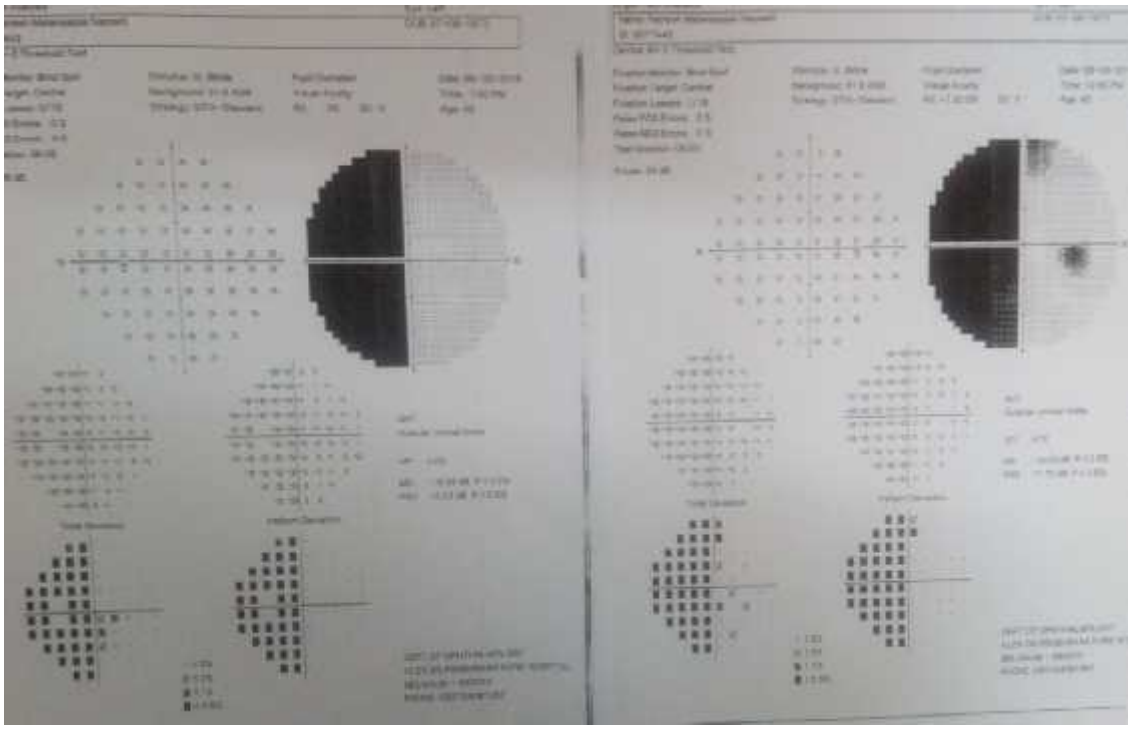
  Headache since 2 months

With best corrected visual acuity in RE 6/6 Near vision RE N6

LE 6/12 LE N8

- Perimetry showed the following findings.

Perimetry-Left eye Perimetry- Right eye



Photograph 1: Right side homonymous hemianopia

MRI SCAN

CORONAL VIEW **SAGITTAL VIEW**

Photograph 2: MRI s/o Right parieto temporal falcine meningiomas

CASE-2

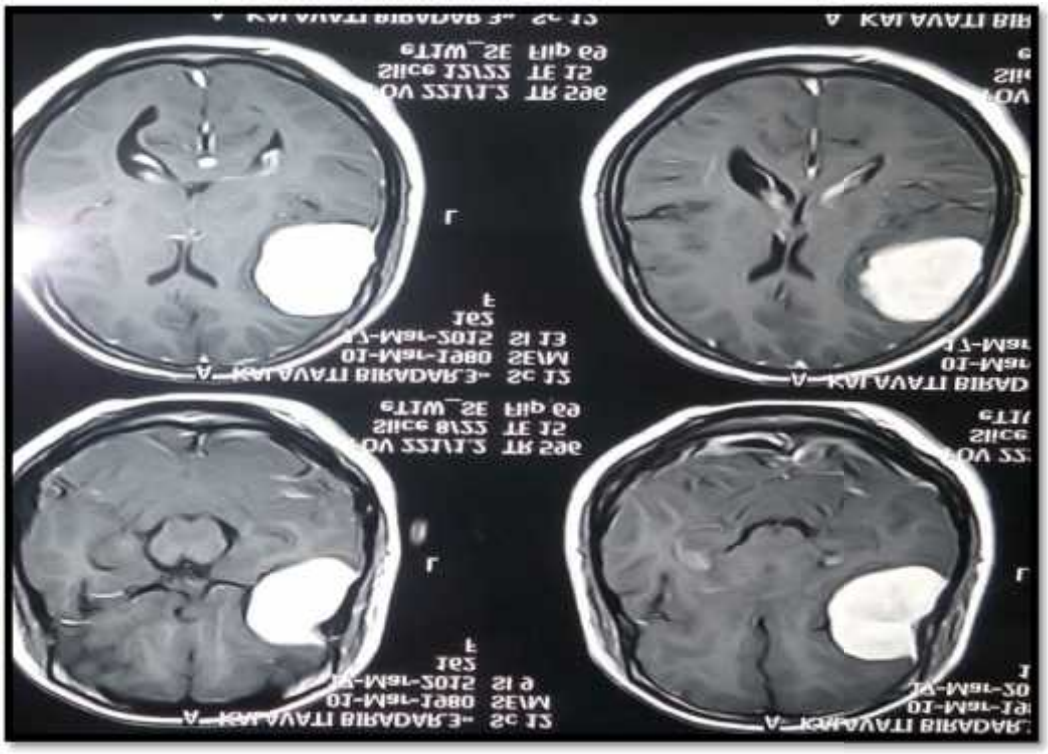
- 43 years old female.
- h/o severe headache with projectile vomiting since 6 months.
- h/o diminution of vision in both eyes since 25 days.

FUNDUS PHOTOGRAPH OF SAME PATIENT

Photograph 3: Papilloedema

MRI SCAN

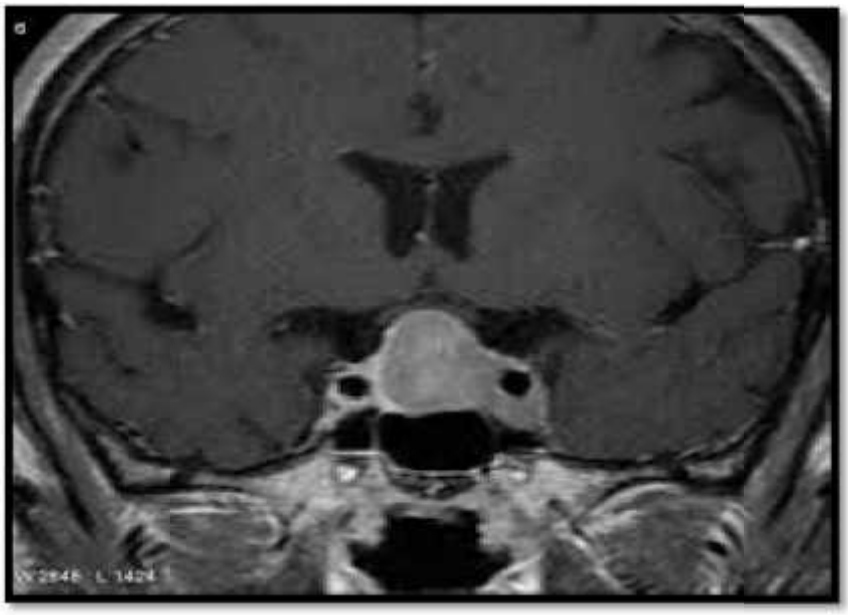
- Ill defined round to oval mildly enhancing lesion in right frontal region with perilesional edema measuring approximately 3.4x3.8x3.0 cm in transverse, anterio-posterior and cranio-caudal respectively.



Photograph 4: MRI s/o Right Fronto –Temporal Meningioma

CASE 3

- 33 years old male pt.
- h/o headache since 4 months
- h/o drooping of left eyelid since 20 day and
- h/o episodes of vomiting

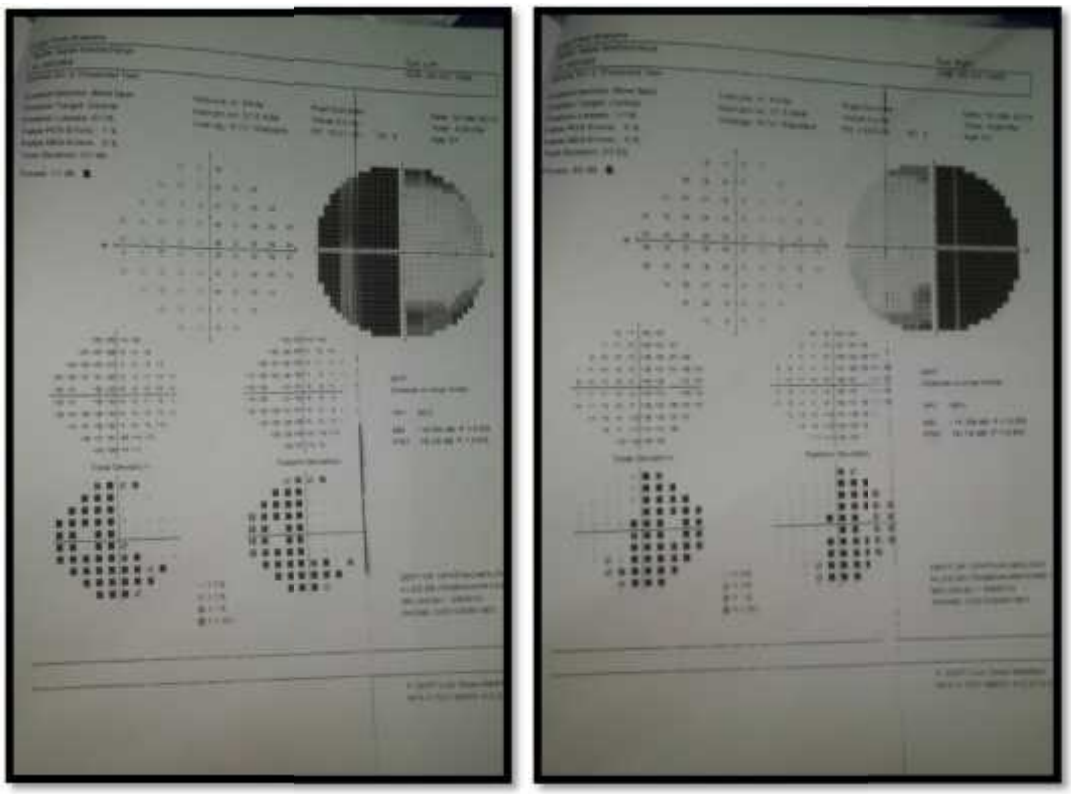


Photograph 5: MRI SCAN s/o Pituitary Macroadenoma

Photograph 6: RE Ptosis

Photograph 7: Third cranial nerve palsy

Perimetry Left eye Perimetry Right eye



Photograph 8: Bitemporal hemianopia

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By

DR. SANYA GARG

REG.NO. BK0113004

Dissertation

Submitted to the KLE University, Belagavi, Karnataka. In partial fulfilment of the requirements for the degree of

MASTER OF SURGERY

IN

OPHTHALMOLOGY

Under the Guidance of:

Dr. ARVIND L. TENAGI M.S.(Ophthalmology)

Professor

DEPARTMENT OF OPHTHALMOLOGY,

JAWAHARLAL NEHRU MEDICAL COLLEGE,

BELAGAVI, KARNATAKA.

APRIL 2016

KLE UNIVERSITY, BELAGAVI,
KARNATAKA.

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Date:

Place: Belagavi

Dr. SANYA GARG

REG NO-BK0113004

KLE UNIVERSITY, BELAGAVI,
KARNATAKA.

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Date:

Place: Belagavi

Dr. ARVIND L. TENAGI **M.S.(Ophthalmology)**
Professor, Department of Ophthalmology,
J. N. Medical College,
Belagavi – 590010.
Karnataka. India.

KLE UNIVERSITY, BELAGAVI, KARNATAKA.

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This is to certify that the dissertation entitled **"A ONE YEAR CROSS SECTIONAL STUDY OF OCULAR MANIFESTATIONS OF INTRACRANIAL SPACE OCCUPYING LESIONS IN PATIENTS AT KLE'S DR. PRABHAKAR KORE HOSPITAL AND MRC, BELAGAVI"** is a bonafide research work done by **Dr. SANYA GARG** in partial fulfilment of the requirement for the degree of **Master of Surgery in Ophthalmology.**

Date:

Place: Belagavi

Dr. RAJESH Y. SHENOY **M.Ch.(Neurosurgery)**
Associate Professor, Department of Neurosurgery,
J. N. Medical College,
Belagavi – 590010.
Karnataka. India.

KLE UNIVERSITY, BELAGAVI,

KARNATAKA.

Endorsement by the Head Of Department, Principal/ Head of the Institution

This is to certify that the dissertation entitled **"A ONE YEAR CROSS SECTIONAL STUDY OF OCULAR MANIFESTATIONS OF INTRACRANIAL SPACE OCCUPYING LESIONS IN PATIENTS AT KLE'S DR. PRABHAKAR KORE HOSPITAL AND MRC, BELAGAVI"** is a bonafide research work done by **DR. SANYA GARG** under the guidance of **Dr. ARVIND L. TENAGI** **M.S.(OPHTHO)** Professor, Department of Ophthalmology, J. N. Medical College, Belagavi.

Seal & Signature of the HOD

Dr.(Mrs.) Rekha B. K **DOMS, MS , PhD**
Professor & Head,
Department of Ophthalmology,
J. N. Medical College,
Belagavi – 590010.
Karnataka, India.

Date :

Place: Belagavi

Seal & Signature of the Principal

Dr.(Mrs.)N. S. Mahantashetti **MD (Paed)**
Principal,
J. N. Medical College,
Nehru Nagar,
Belagavi - 590010.
Karnataka, India.

Date :

Place: Belagavi

KLE UNIVERSITY, BELAGAVI,

KARNATAKA.

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Date:

Place: Belagavi

Dr. SANYA GARG
Department of Ophthalmology
J. N. Medical College,
Belagavi – 590010.
Karnataka, India