OCULAR MANIFESTATIONS IN ADULTS WITH INTRACRANIAL NEOPLASMS ATTENDING THE NEUROSURGICAL UNIT IN KENYATTA NATIONAL HOSPITAL

A dissertation submitted in part fulfillment of the degree of Masters of Medicine (Ophthalmology) in the University of Nairobi

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DECLARATION

This dissertation is my original work, and has not been presented for a degree in any other university.

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DEDICATION

To my beloved family and friends for their support and encouragement.
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ABBREVIATIONS

BCVA........................................Best Corrected Visual Acuity

CNS........................................Central Nervous System

CI Scan.....................................Computer Tomography Scan

ICP........................................Intracranial pressure

KNH.........................................Kenyatta National Hospital

LE............................................Left Eye

LGB..........................................Lateral Geniculate Body

MRI..........................................Magnetic Resonance Imaging

RE............................................Right Eye

RAPD........................................Relative Afferent Pupillary Defect

SOL..........................................Space Occupying Lesion

TAPD........................................Total Afferent Pupillary Defect

VP.............................................Ventriculoperitoneal

WHO.........................................World Health Organisation
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SUMMARY

INTRODUCTION: Patients with intracranial neoplasm are routinely seen in Kenyatta National Hospital at the Neurosurgical Unit. Most patients however do not have a complete ophthalmologic assessment and are therefore not fully informed about their visual prognosis especially when the visual pathway is affected.

AIM: The aim of this study was to determine the prevalence and pattern of ocular findings within this population.

METHODOLOGY: It was a cross sectional hospital based study carried out at the Neurosurgical Clinic and Ward at the Kenyatta National Hospital. 60 adult patients diagnosed to be having intracranial neoplasm were recruited over a period of three months (November 2005 - January 2006). An ophthalmic examination consisting of Best Corrected Visual Acuity, colour vision, pupillary reactions to light, extra ocular motility, proptosis, diploplia and nystagmus was done. The anterior segment was examined using a Ilaag Streit Slit Lamp and the posterior by indirect ophthalmoscopy using a 90 Dioptrre loupe. Visual field assessment was done using the Goldman Perimeter.

RESULTS: 28 males and 32 females were recruited. The mean age was 37 years, the median 36 years and a range of 15 to 69 years. 44 (73%) patients complained of ocular symptoms, and 29 (48%) had had a previous ophthalmic assessment. The most
common neoplasm was the suprasellar tumour with a total of 17 (28%) patients. 20 (33%) patients had some degree of visual impairment, 11 (18%) of these were blind. 8(13%) patients had monocular blindness. Abnormal colour vision was recorded in 34 (28%) eyes. Pupillary reaction was abnormal in 30 (50%) patients. 13 (21%) patients had strabismus, 10(17%) had nystagmus, proptosis was noted in 6 (10%) patients and diplopia in 4 (7%). 11 (18%) patients had abducens nerve palsy. Papilloedema was a finding in 20 (33%) patients, and bilateral disc atrophy in 16 (27%) patients. Generalized constriction was the most common visual field defect seen in 23 (18%) eyes followed by homonymous hemianopia in 16 (27%) eyes. 45 (75%) patients were referred for routine follow-up in the eye clinic, 4 (7%) patients with severe visual impairment were referred for Low Visual Assessment. All the 11 (18%) blind patients were referred for rehabilitation.

CONCLUSION. There is a higher prevalence of ocular manifestations in patients with intracranial tumours in our set up compared to centres in developing countries. The number of patients who had ophthalmic assessment before the study was low. Tumours located in close proximity with the anterior visual pathway are most likely to cause optic nerve compression and subsequent blindness. The most common encountered visual field defect is the generalised constriction which is non-specific for localising the site of the tumour. Patients who are blind as a result of intracranial neoplasms are not properly rehabilitated. There is no protocol for the referral of these patients to other specialised institutions offering Low Vision Assessment and rehabilitation services.
**RECOMMENDATION:** Neurosurgeons should work closely with ophthalmologists to ensure complete assessment of patients with intracranial tumours both pre operative and post operative. Referral for Low Visual Assessment and Rehabilitation needs to be formalised. There should be in-patient rehabilitation services within Kenyatta National Hospital.
I. INTRODUCTION AND LITERATURE REVIEW

Morphological and functional disturbances of the visual system are frequently observed in patients with intracranial space occupying lesions (SOLs). At present there is not enough existing data in KNH giving recommendations for ophthalmic follow-up of these cases as patients are not always routinely sent for complete ophthalmic assessment at the time of diagnosis, before and after any surgical, radiotherapeutical or chemotherapeutical intervention.

The aim of treatment in patients with intracranial neoplasms should be a wholesome approach. In addition to neurosurgical intervention, an accurate ophthalmologic assessment together with basic counseling on visual prognosis is important. For patients with irreversible visual loss, use of Low Vision Devices or early referral to The Kenya Society for the Blind, may very well be what they need to boost their confidence, and expedite their integration into an independent social and professional life.

1.1 BACKGROUND

SOLs mainly consist of primary brain tumors which account for less than 2% of all cancers in adults and about 20% of all malignant tumours in children. Gliomas are the most common primary intracranial neoplasm, accounting for 40-67% of adult tumours; meningiomas account for approximately 9-27% and pituitary adenomas about 15%.
1.2.1 Headache

Up to 75% of patients with intracranial tumours complain of headaches.¹ The headache is of extreme variety, worse in the morning and is associated with nausea and vomiting. Vasalva maneuver causes exacerbations. They are described as "boring" or "bursting" and may be associated with transient focal neurological symptoms and signs.⁴

1.2.2 Abducens Nerve Paresis

This can be unilateral or bilateral and is a non-localizing sign of an intracranial tumour. With increased ICP, one or both of the abducens nerves becomes compressed between the pons and the basilar artery⁶ or are stretched along the sharp edge of the petrous bone.⁷ It is rarely a complete paresis and tends to resolve rapidly (within days to weeks) once the ICP is lowered.

1.2.3 Papilledema

Occurs due to hold up of axoplasmic flow (orthograde transport of cytoplasmic organelles from cell body to synapse) at the level of lamina cribrosa. This is due to compressive intracranial lesions and may precede development of optic atrophy.

Sood et al (2002) in a study of supratentorial tumours, papilloedema was detected in 8% of the patients.⁹ This figure is much lower than previous studies done by Huber who found a prevalence of 59% out of 1166 patients with brain tumours.¹⁰ Infratentorial tumours are more likely to produce papilloedema than supratentorial ones. Petrohelos and Henderson found papilloedema in 75.2% of patients with infratentorial tumours, but in only 53.4% with supratentorial tumours.⁹ Infratentorial
tumours produce papilloedema by obstruction of the aqueduct thus raising the ICP. Papilledema is usually bilateral, though it may be quite asymmetric or purely unilateral.

1.2.4 Foster Kennedy Syndrome

This was described by Robert Foster Kennedy, a British-American neurologist (1884-1952). It is a rare syndrome due to a tumour which causes direct compression of the optic nerve on the ipsilateral side leading to atrophy, and increased intracranial pressure causing contralateral papilloedema. This is classically due to meningioma of the olfactory groove, or more commonly a meningioma of the sphenoid wing, and in rare cases a tumour in the frontal lobe. The syndrome is associated with anosmia or hyposmia. In a review of 36 previously reported cases by Warnick, it was revealed that 12 cases (33%) were probably also caused by bilateral optic nerve compression, while only 8 (22%) satisfied the original hypothesis. 12

1.3 VISUAL PATHWAY

Each eyeball acts as a camera: it perceives the images and relays the sensations to the brain (Visual cortex) via the visual pathway, which comprises Optic nerve, Optic chiasma, Optic tracts, Lateral Geniculate Body (LGB) and Optic radiation.

1.3.1 Optic Nerve

This is the second cranial nerve and is a backward continuation of the Nerve Fibre Layer of the retina, which consists of axons originating from ganglion cells. It extends
to the optic chiasma where the two nerves meet. Optic disc changes in intracranial tumours are mainly papilloedema and disc atrophy.

1.3.1.1 Disc atrophy

Profound to complete visual loss has been described in patients with intracranial tumours who present late after optic atrophy has already occurred. In a study by F. Kagondu in Kenyatta National Hospital on non-glaucomatous optic atrophy, 7% of the cases were due to intracranial tumours and it was concluded that brain tumours were the commonest cause of bilateral optic atrophy. In a study by J. M Muthuuri on sellar and juxtasellar tumours in KNH, fundoscopy revealed bilateral optic atrophy in 97% of the patients. This is a rather high number as it would mean that most of the patients had very poor vision by the time they presented to the hospital. In a study of suprasellar meningioma and blindness in Saudi Arabia, the incidence of blindness in one or both eyes at presentation was high (42.2%). 12.8% were totally blind on admission due to optic atrophy.

i) Primary Optic Atrophy: This occurs without previous papilloedema. Lesions affecting the visual pathway from the retrolaminar portion of the optic nerve to the LGB may cause it. Lesions anterior to the optic chiasma result in unilateral atrophy which is diffuse on fundoscopy. Those involving the chiasma and optic tract result in bilateral optic atrophy with involving nasal and temporal portions of the disc, but sparing the superior and inferior areas (bow-tie atrophy).
ii) *Secondary optic atrophy:* This is preceded by longstanding papilloedema.

Also referred to as atrophic papilloedema.

Tumours that compress the intracranial portion of the optic nerve produce variable visual loss and can be associated with almost every type of visual field defect, including central scotoma, centrocecal scotoma, arcuate scotoma, peripheral constriction and hemianopic field loss."

### 1.3.2 OPTIC CHIASMA

This is a flattened structure which overlies the diaphragm sellae. The sella turcica (Turkish saddle) is a saddle shaped depression on the superior surface of the body of the sphenoid in which the pituitary gland lies. The roof is formed by the diaphragm sella. Posteriorly the chiasma continues as the optic tract. Anatomically, the chiasma can be central (80%), pre-fixed (10%), or post-fixed (10%) and these variations are important in the pattern of visual fields produced by different chiasmal lesions.

Intracranial tumours found at the sella turcica which commonly affect the chiasma are pituitary adenomas, meningiomas and craniopharyngiomas. In a study in China, it was found that pituitary adenomas were the majority of the 407 patients seen (72%), craniopharyngiomas (12%), meningiomas (10%) and other tumours <1%. Another study in Germany quoted an almost similar distribution. Other rare tumours found were gliomas, chordomas, dysgerminomas, nasopharyngeal tumours, infra- and supraclinoidal aneurysms, Rathke pouch cysts, fibrous dysplasia, sphenoid sinus mucocele, arachnoid cyst and secondary metastases.
Hemianoptic visual field defects and preferential involvement of the temporal visual field are the earliest and most common visual deficits. Progression of the lesion may cause compression of adjacent structures including the optic nerve, cavernous sinus and may result in a more profound visual loss, ocular motor deficits and hypopituitarism. Recent findings have shown that perimetry remains the most effective means of detecting and following the progression of the visual deficit and that MRI is the best mode of neuroimaging for most chiasmal lesions.

In the presence of the atypical or incipient bitemporal visual field defects, as they occur also in early stages of tumours of chiasmal region, one has to first of all exclude other causes for such fields defects such as refraction scotomas, tobacco-alcohol amblyopia, dominant hereditary optic atrophies, unilateral/bilateral optic neuritis, the intoxications of the optic nerve and distant effect of a hydrocephalus of the 3rd ventricle on the chiasma.

1.3.2.1 Pituitary Adenomas

The chromophobe adenoma is the most common primary intracranial tumour to produce neuro-ophthalmic features. Although usually detected by endocrinologists, non-secreting tumours may first present to the ophthalmologist. Tumours <10mm (microadenomas) often remain intrasellar while those >10mm (macroadenomas) tend to manifest extrasellar extension. Lestak et al. pointed out a relatively long period between the first compressive symptoms and the operation, especially in non-secreting pituitary adenomas (2.1 years). He concluded that an early diagnosis and appropriate therapy of the patients suffering from compressive lesions of the chiasmal region decreases the risk of permanent vision defects.
**Clinical ophthalmological features:**

- **Visual field defects:** If the chiasma is central, bitemporal hemianopia is common. Fang et al showed 87% of patients with pituitary macroadenomas had bitemporal hemianopia and showed or tended to have a medial vertical limit.\(^{22}\)

- **Reduced visual acuity, optic atrophy, hypopsia, colour desaturation.**

- **Pituitary apoplexy:** This is a clinical syndrome resulting from pituitary infarction, haemorrhage or both. It occurs most commonly in the setting of a previously unrecognized pituitary adenoma. It prevalence is 0.6-10% of patients with pituitary adenomas, twice as frequently in men than women. Signs and symptoms as recorded by Bill et al. are severe headache (95%), ophthalmoplegia (78%), vomiting (69%) abnormalities of visual fields (64%) and reduced visual acuity (52%)\(^*\)

### 1.3.2.2 Meningiomas

Intracranial meningiomas produce variable neuro-ophthalmological signs and symptoms depending on their location. Anderson et al studied 80 patients with meningiomas and found a third of them with ophthalmological findings. Me concluded that meningioma of the sphenoid bone, parasellar area and occiput most often produced visual deficits.\(^*\)

Frequently, it is difficult to differentiate the chiasmal meningioma from other suprasellar tumours, especially pituitary adenoma and craniopharyngioma. A few differences can be seen in that meningiomas affects females over 40 years more, the
duration of symptoms is 2-3 years, visual acuity and visual field disturbances are initially unilateral in most cases, then extend to the other side when the disturbance of one eye becomes severe. The visual field defect tends to be irregular bitemporal hemianopia with frequent involvement of the peripheral nasal field.²⁵

Meningiomas affecting the optic chiasma are those originating from the tuberculum sellae, sphenoid ridge, and olfactory groove. Other visual signs are hypopsia, optic atrophy, diplopia.²⁴ Rosenberg et al noted that after microsurgical removal of meningiomas involving the anterior visual system, postoperatively the visual acuity either improved or remained normal in 68.75%, and worsened in 25% of the eyes examined. The visual field changes paralleled the visual acuity changes. He concluded that visual results were mostly related to duration of visual symptoms and not to either tumour size or preoperative visual findings." ²⁴

1.3.2.3 Craniopharyngioma

The craniopharyngioma is a slow growing tumour originating from vestigial remains of Rathke pouch along the pituitary stalk. Patients generally present late and visual symptoms are often preceded by a long history of systemic symptoms. Children are more likely to present with systemic symptoms than adults. ⁷ Visual signs and symptoms are reduced visual acuity, visual field defects, optic atrophy, disc oedema, nystagmus, and oculomotor nerve paresis. ⁷ ²⁷ ²⁸ ²⁹ in the visual fields, bitemporal hemianopia and homonymous hemianopia are relatively common. Visual field pleomorphism, which is a distinct change from one type of field defect to another with progression of the disease, is said to be a characteristic feature of the tumour. ²⁵ ²⁸ ³⁰
1.3.3 OPTIC TRACT

Lesions in the suprasellar region and lesions deep to the temporal lobe may damage the optic tract. Complete loss of optic tract function causes:

i) *Homonymous hemianopia*, which is usually incongruous. This is because the uncrossed nerve fibres from the ipsilateral retina are not closely aligned.

ii) *Wernicke hemianopic pupil*. This is a relative afferent pupillary defect (RAPD) without loss of visual acuity or colour vision in the eye ipsilateral to the hemianopia.

iii) *Optic atrophy*. The fibres of the optic tract are axons of retinal ganglion cells. Therefore the ipsilateral disc manifests atrophy of superior and inferior aspects of the neuroretinal rim (fibres from temporal retina) while contralateral disc manifests a bow-tie pattern of atrophy (nasal retinal fibres)."

The combination of a complete homonymous hemianopia and ipsilateral RAPD without evidence of optic neuropathy is thus pathognomonic of optic tract damage even when it occurs in patients without any other neurological symptoms or signs."

1.3.4. LATERAL GENICULATE BODY (LGB)

Lesions of the LGB produce striking visual field defects that can be wedge shaped, horizontal homonymous sectoranopias that may be congruous or incongruous/~ The defects are related to the lamellar structure of the lateral geniculate nucleus and dual blood supply to the nucleus provided by the anterior choroidal and lateral choroidal arteries. The lesions also produce homonymous hemianopic atrophy in the ocular fundi. The most common tumours that damage the LGB are astrocytomas that originate in the deep temporal lobe and metastatic tumours.
1.3.5 OPTIC RADIATIONS

The optic radiations are third order neurones which extend from the LGB to the striate cortex. Visual field defects tend to be more congruous as the fibres from the corresponding retinal elements lie progressively together. The ocular defects are contralateral homonymous superior quadrantanopia (pie in the sky) because of the Meyer's loop. Lesions in the anterior parietal radiations produce an inferior quadrantanopia. Associated features are agnosias, visual hallucinations, visual perception difficulties and acalculia.

1.3.6. VISUAL CORTEX

Tumours of the occipital lobe are relatively infrequent. They represent only 3.5% of all intracranial tumours encountered by Walsh and Hoyt over a 10 year period from 1959 to 1969 and only 6% of tumours reported by Huber. They mostly occur in adults, with glioblastomas and meningiomas commonly, and the astrocytomas, oligodendromas and secondary metastases occurring less frequently.

The visual signs and symptoms may be caused by direct occipital lobe involvement or by direct or indirect damage to the neighbouring parietal and temporal lobes, cerebellum, or the brainstem. Visual field defects are observed in 95% of all patients with tumours that damage the occipital lobe. These are almost invariably contralateral to the side of the lesion and include homonymous hemianopia with or without macular sparing, incomplete homonymous hemianopia (and quadrantanopias) and hemianopic scotomas that are congruous, and selective loss or sparing of the temporal crescent.
Occasionally, extension of a left occipital lobe tumour into the splenium of the corpus callosum produces a syndrome of alexia without agraphia (retention of writing ability but loss of ability to read what is written).\textsuperscript{37}

Visual hallucinations are usually of the uniform variety may occur in the blind hemifield contralateral to the tumour. This occurs in up to 25\% of patients with occipital tumours.\textsuperscript{*} Other ocular feature are visual agnosia,\textsuperscript{5} denial of blindness (Anton syndrome) and Riddoch phenomenon characterized by the ability to perceive kinetic, but not static targets. Patients also get papilloedema, oculomotor nerve paresis, conjugate gaze palsies and nystagmus.

1.4 INTRAORBITAL EXTENSION

Occasionally, intracranial tumours may extend into the ipsilateral orbit producing ocular signs. These are rare manifestations and can occur with frontal lobe tumours, sphenoidal tumours, and temporal lobe tumours.\textsuperscript{40} These patients present with symptoms of orbital disease such as:

- Proptosis
- Disc oedema progressing to optic atrophy
- Optociliary shunts
- Choroidal folds
- Restricted extra-ocular motility
- Lid oedema
- Conjuctival chemosis
1.5 METASTATIC TUMOURS

Malignant tumours that arise in various parts of the body can spread to the CNS. Most metastases result from haematogenous spread of the tumour. The tumour may be disseminated to the bones of the skull, from which it may spread to the subarachnoid space, brain or both. Alternatively, it may metastasize directly to the brain parenchyma or to the subarachnoid space.

1.5.1 Bone Metastases:

The close application of the dura matter to the cranial bones facilitates direct spread of the malignancies. This manifest as flat, irregular plaques of metastatic tumour on the inner surface of this membrane, adjacent to an area of bone invasion by the tumour.

1.5.2 Parenchymal Metastases:

Brain parenchyma is affected more frequently by haematogenous spread of tumours, with a frequency of about 20%. The most common tumours are carcinomas of the lung, breast, kidney, stomach, prostate and thyroid. Metastases may be multiple or single, and they may produce a variety of neuro-ophthalmic manifestations, including loss of visual acuity or visual field and disorders of ocular motility and alignment. Meyer et al. reviewed 216 cases of secondary neoplasms to the CNS and found 16.2% were only a single metastases which was usually associated with other extracranial metastases. In rare cases, a solitary intracranial metastases may not only be the only evidence of metastases of the primary tumour, but may be the first evidence of the tumour itself. In some cases, neuroimaging cannot always distinguish between a
pituitary adenoma and intrasellar metastasis with suprasellar extension. Rapid onset and progression, age greater than 50 years, cranial nerve palsies and diabetes insipidus are all features that suggest a metastatic lesion rather than a benign pituitary adenoma.
2. RATIONALE

1. Currently there is limited existing data in KNH giving recommendations for ophthalmic follow-up of patients with intracranial SOLs prior to any intervention and after treatment. These patients may not benefit from objective visual assessment and subsequent referral for rehabilitation if necessary. Baseline ophthalmic assessment is necessary for all patients as in some cases, there is reported improvement or deterioration of vision after surgical, chemotherapeutical or radiotherapeutical intervention.

2. Review of literature has shown that most of the studies have been carried out in developed countries. Results available from those studies may not be applicable to our set up because of the different socio-economic status and limited access to health facilities in the rural area.

3. To date, no such study has been carried out at the Kenyatta National Hospital.
3. STUDY OBJECTIVES

3.1 Main Objective

To determine the prevalence and pattern of ocular manifestations in adult patients with intracranial neoplasms.

3.2 Specific Objectives

1. Determine the prevalence of ocular manifestations in patients with intracranial neoplasms.

2. Determine the pattern of ocular manifestations in patients with intracranial neoplasms.

3. Correlate ocular findings with radiological diagnosis.
4. MATERIALS AND METHODS

4.1 Study Area

The Kenyatta National Hospital, a national teaching and referral hospital in Kenya. The study was conducted at the Neurosurgical Clinic and Ward. The Clinic is number 24. and runs only on Monday and Tuesday afternoons every week. The Neurosurgical Ward is 4C where patients are admitted, awaiting surgery.

4.2 Study Design

This is a cross sectional hospital based study.

4.3 Reference Population

All adult patients with a known diagnosis of an intracranial neoplasm in Kenya.

4.4 Study population

Patients diagnosed with an intracranial neoplasm, attending the neurosurgical clinic or admitted to the neurosurgical ward in Kenyatta National Hospital during the period of study.

4.5 Sample size

A minimum sample size of 58 was calculated using the following formula:

\[ N = \frac{(Z-(t/2) \times p(1-p))}{d^2}, \text{ Where:} \]

\[ N = \text{required sample size} \]
P = estimated prevalence from other studies (30)°

a = Level of significance = 5%. D = absolute precision = 0.1

\[ Z - \frac{a}{2} = 1.96^2 \] if sample size is estimated at 95% confidence level

\[ N = 1.96^2 \times 0.30 \times 0.5/0.1^2 = 58. \] A total of 60 patients were recruited.

4.6 Case Definition

Any patient diagnosed with an intracranial neoplasm, attending the neurosurgical clinic or admitted to the neurosurgical ward in Kenyatta National Hospital.

4.7 Selection Of Cases

Patients will be recruited consecutively from the Neurosurgical Clinic and Neurosurgical Ward in the hospital by the principal investigator.

4.8 Inclusion Criteria

1. All adults (WHO definition is 15 years and over) attending the neurosurgical clinic or admitted to the ward with an intracranial neoplasm.

2. Radiological diagnosis confirmed by CT Scan or MRI.

3. Informed consent from the patient/guardian.

4.9 Exclusion Criteria

1. Other causes of brain compression e.g intracranial haemorrhages, abscesses etc

2. Patients without a radiological diagnosis of the intracranial neoplasm
4.10 Materials

- Questionnaire (See Appendix I)
- Torch and batteries
- Snellens Chart. Near Vision Chart
- Refraction Set and trial frames
- Colour vision chart
- Haag Streit Slit lamp
- 20 and 90 Dioptre Loupes
- Direct and Indirect Ophthalmoscope
- Goldman Perimetry Machine
- Hertels exophthalmometer
- Dilating drops (Tropicamide 1 %)
- Topical anaesthesia (Tetracaine 0.5%)
- Flourescein strips
- Surgical spirit and swabs
4.11 STUDY PROCEDURE

The subjects were recruited in the neurosurgical ward and clinic and consent for participation was obtained. Demographic data was obtained from the patient. This included details of their name, age, sex, whether in the ward or clinic. Current neurological signs and symptoms together with their duration were recorded. History of previous ophthalmic assessment by an eye specialist (ophthalmic nurse, ophthalmic clinical officer or ophthalmologist) was recorded. A brief ophthalmic history, together with duration of signs and symptoms was recorded. The radiological diagnosis was recorded and any current treatment or surgical intervention done to date was noted.

An ophthalmic assessment was done whereby the Visual Acuity was taken with a Snellens Chart and the Best Corrected Visual Acuity was recorded according to WHO categories of visual impairment (Appendix II). The colour vision test was done using Ishihara Colour Vision Charts. Pupillary reactions to light was tested, and alignment of the eyes was assessed and recorded as Hirschbergs test. Ocular movements was assessed and evaluation for proptosis using Hertel's Exophthalmometer was done where indicated. Any diplopia or nystagmus was assessed and recorded. Visual Field assessment was done using the Goldman Perimeter.

The examination of the anterior segment was performed using a Haag Streit Slit lamp. Pupillary dilation using Tropicamide and evaluation of fundus using indirect ophthalmoscopy was done using a 90 Dioptré loupe.
4.12 DATA ANALYSIS

Data was collected in the form of questionnaires and then entered into the computer awaiting analysis. Data validation was done before the analysis. Analysis was done using Statistical Package for Social Scientists (SPSS) version 11.5. Results were presented in tables, graphs and pie charts.

4.13 ETHICAL CONSIDERATIONS

1. Informed consent from the patient/guardian was taken (Appendix III)

2. Confidentiality of the patient was maintained. Each patient's detail was recorded using a numerical code.

3. Counseling was done to the patients not previously advised about their visual status.

4. All patients who needed additional treatment were referred to the appropriate clinician for management.

5. All ophthalmic medications used in the study were registered in Kenya and used as recommended.
5. RESULTS

5.1 DEMOGRAPHIC DATA

FIGURE 1: Distribution of patients by sex (n=60 patients)

60 patients were studied. 32 (53%) females, 28 (47%) males. F:M ratio 1.1:1

TABLE 1: Distribution of patients by location and sex (n=60 patients)

<table>
<thead>
<tr>
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<th>MALE</th>
<th>FEMALE</th>
<th>TOTAL</th>
<th>PERCENTAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>CLINIC</td>
<td>19</td>
<td>17</td>
<td>36</td>
<td>60</td>
</tr>
<tr>
<td>WARD</td>
<td>9</td>
<td>15</td>
<td>24</td>
<td>40</td>
</tr>
<tr>
<td>TOTAL</td>
<td>28</td>
<td>32</td>
<td>60</td>
<td>100</td>
</tr>
</tbody>
</table>

p=0.245

36 (60%) patients were recruited from the neurosurgical clinic and 24 (40%) from the neurosurgical ward. There was no statistically significant difference between the patients recruited from the clinic and the ward (p=0.245).
The mean age was 37 years and the median 36 years. The age bracket of 36 - 45 years had the largest number of participants with 24 (40%) patients, the majority being female 14 (23%). There was no statistically significant difference between the males and the females in any of the age groups ($p=0.261$). The 60 patients ranged from 15 to 69 years.
5.2 **HISTORY OF NEUROLOGIC & OPHTHALMIC SYMPTOMS**

*FIGURE III: Summary of Neurological & Ophthalmic History*

52 (85%) patients complained of headache and this was the most common symptom followed by ocular disturbances in 44 (73%) patients.

*FIGURE IV: Previous Ophthalmic Assessment (n=60 patients)*

Out of 60 patients, 29 (48%) had had a previous ophthalmic assessment by an eye specialist.
### 5.3 Radiological Diagnosis

**TABLE II: Radiological diagnosis (n=60)**

<table>
<thead>
<tr>
<th>Radiological Diagnosis</th>
<th>Total</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Suprasellar tumours</td>
<td>17</td>
<td>28</td>
</tr>
<tr>
<td>Frontal lobe tumour</td>
<td>7</td>
<td>12</td>
</tr>
<tr>
<td>Temporal lobe tumour</td>
<td>4</td>
<td>7</td>
</tr>
<tr>
<td>Parietal lobe tumour</td>
<td>10</td>
<td>17</td>
</tr>
<tr>
<td>Occipital lobe tumour</td>
<td>5</td>
<td>8</td>
</tr>
<tr>
<td>Sphenoidal ridge meningioma</td>
<td>7</td>
<td>11</td>
</tr>
<tr>
<td>Olfactory groove meningioma</td>
<td>3</td>
<td>5</td>
</tr>
<tr>
<td>Others</td>
<td>7</td>
<td>12</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>60</strong></td>
<td><strong>100</strong></td>
</tr>
</tbody>
</table>

The most common diagnosis was the suprasellar tumour with 17 (28%) patients. The least common was the olfactory groove meningioma of which 3 (5%) patients were recruited.
### TABLE 111: Distribution of radiological diagnosis according to age groups (n=60 patients)

<table>
<thead>
<tr>
<th>Radiological diagnosis</th>
<th>AGE (iROU PS)</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>15-25</td>
<td>26 - 35</td>
</tr>
<tr>
<td>Suprasellar tumours</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Frontal lobe tumour</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Temporal lobe tumour</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Parietal lobe tumour</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Occipital lobe tumour</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Sphenoidal ridge meningioma</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Olfactory groove meningioma</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Others</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>Total</td>
<td>13</td>
<td>11</td>
</tr>
<tr>
<td>Percentage</td>
<td>22</td>
<td>18</td>
</tr>
</tbody>
</table>

p=0.128

Majority of the patients between 36 - 45 years had suprasellar tumours, 8 (13%), followed by those with parietal lobe tumours with 5 (8%) patients in the same age category. Olfactory groove meningioma was least common with 3 (5%) patients, 2 of which were in the age group of >55 years. There was no statistically significant correlation between the type of tumour and the age group (p=0.128)
30 (50%) patients were not on any treatment and had not undergone surgery. 16 (27%) patients had undergone surgical excision of their tumours. 2 of whom had VP shunts, and 2 had completed radiotherapy treatment. 1 (2%) patients had a VP shunt inserted without excision of the tumour. The patients on medication only were 13 (21%) and this consisted of systemic dexamethasone and an anticonvulsant (either epanutin or carbamazepine).
5.4 EXAMINATION FINDINGS

FIGURE VI: Distribution of the BCVA According WHO classification (n =60 patients)

40 (67%) respondents had normal vision. 10 of whom had undergone surgical excision of the tumour. 5 (8%) patients presented with visual impairment, while 4 (7%) had severe visual impairment. 11 (18%) patients were blind, 5 (8%) of them had already undergone surgical excision of the tumours.


**TABLE IV: Distribution of radiological diagnosis according to BCVA (n=60 patients)**

<table>
<thead>
<tr>
<th>Radiological Diagnosis</th>
<th>Normal Vision</th>
<th>Visual Impairment</th>
<th>Severe Visual Impairment</th>
<th>Blind</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Suprasellar tumours</td>
<td>6</td>
<td>4</td>
<td>1</td>
<td>6</td>
<td>17</td>
</tr>
<tr>
<td>Frontal lobe tumour</td>
<td>4</td>
<td>0</td>
<td>0</td>
<td>3</td>
<td>7</td>
</tr>
<tr>
<td>Temporal lobe tumour</td>
<td>2</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>Parietal lobe tumour</td>
<td>9</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>10</td>
</tr>
<tr>
<td>Occipital lobe tumour</td>
<td>3</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>Sphenoidal ridge meningioma</td>
<td>7</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>7</td>
</tr>
<tr>
<td>Olfactory groove meningioma</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>Others</td>
<td>7</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>7</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>40</strong></td>
<td><strong>5</strong></td>
<td><strong>4</strong></td>
<td><strong>11</strong></td>
<td><strong>60</strong></td>
</tr>
</tbody>
</table>

\( p=0.039 \)

Patients with suprasellar tumours had the highest number of blind patients 6 (10%). followed by frontal lobe tumours with 3 (5%). There was a significant correlation between tumours affecting the anterior visual pathway (suprasellar and frontal) and blindness \( (p=0.039) \).
67 (56%) eyes had normal vision. 11 (9%) had visual impairment. 5 (4%) eyes had severe visual impairment, while 37 (31%) were blind.

**Monocular blindness**

Monocular blindness was defined as a patient with one eye with normal vision and a blind fellow. 8 (13%) patients satisfied this criteria and their distribution according the radiological diagnosis is shown below.

**TABLE V: Causes of monocular blindness (n=8 patients)**

<table>
<thead>
<tr>
<th>Tumour</th>
<th>Frequency with monocular blindness</th>
<th>Total without monocular blindness</th>
<th>Total Number of Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sphenoidal ridge meningioma</td>
<td>4 (57%)</td>
<td>3 (43%)</td>
<td>7 (100%)</td>
</tr>
<tr>
<td>Suprasellar tumours</td>
<td>3 (18%)</td>
<td>14 (82%)</td>
<td>17 (100%)</td>
</tr>
<tr>
<td>Parietal lobe tumour</td>
<td>1 (10%)</td>
<td>9 (90%)</td>
<td>10 (100%)</td>
</tr>
</tbody>
</table>
4 (57%) patients with sphenoidal ridge meningioma had monocular blindness and this consisted of more than half of the patients with that tumour. Only 1 (10%) patient with parietal lobe tumour presented with monocular blindness.

*FIGURE VIII: Colour Vision (n=120 eyes)*

30 (25%) eyes were unable to perform the colour vision test. Abnormal colour vision was observed in 34 (28%) eyes.

*FIGURE IX: Pupillary Reaction (n=60 patients)*

30 (50%) patients out of the total population had normal pupillary reactions, only 2 (3%) patients demonstrated Wernicke Hemianopic pupil.
### TABLE VI: Ocular examination findings

<table>
<thead>
<tr>
<th></th>
<th>Frequency</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Strabismus</strong></td>
<td>13 (21%)</td>
<td>60 (100%)</td>
</tr>
<tr>
<td><strong>Nystagmus</strong></td>
<td>10 (17%)</td>
<td>60 (100%)</td>
</tr>
<tr>
<td><strong>Proptosis</strong></td>
<td>6 (10%)</td>
<td>60 (100%)</td>
</tr>
<tr>
<td><strong>Diplopia</strong></td>
<td>4 (7%)</td>
<td>60 (100%)</td>
</tr>
</tbody>
</table>

Strabismus 13 (21%) was the most common finding, while diplopia least common, was demonstrated in 4 (7%) patients.

### TABLE VII: Causes of defective extra ocular motility (n=16 patients)

<table>
<thead>
<tr>
<th>Cause of defective extra ocular motility</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abducens nerve palsy</td>
<td>11</td>
</tr>
<tr>
<td>Intraorbital extension of tumour (causing restriction in all gazes)</td>
<td>4</td>
</tr>
<tr>
<td>Oculomotor nerve palsy</td>
<td>1</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td><strong>16</strong></td>
</tr>
</tbody>
</table>

16 patients had defective of extra ocular motility. Abducens nerve was affected in 11 patients. 1 patient had involvement of the oculomotor nerve while 4 patients had all gazes of the extra-ocular muscles affected due to intraorbital extension of the tumour.
42 (35%) eyes had disc oedema. Atrophic discs were observed in 41 (34%) eyes, while 37 (31%) eyes had normal discs.

**TABLE VIII: Laterality of disc appearance (n=60 patients)**

<table>
<thead>
<tr>
<th>DISC APPEARANCE</th>
<th>FREQUENCY</th>
<th>PERCENTAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Disc Oedema</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unilateral</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Bilateral</td>
<td>20</td>
<td>33</td>
</tr>
<tr>
<td>Atrophy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unilateral</td>
<td>9</td>
<td>15</td>
</tr>
<tr>
<td>Bilateral</td>
<td>16</td>
<td>27</td>
</tr>
<tr>
<td>Normal discs (Both Eyes)</td>
<td>13</td>
<td>22</td>
</tr>
<tr>
<td>TOTAL</td>
<td>60</td>
<td>100</td>
</tr>
</tbody>
</table>
13 (22%) had normal discs in both eyes. 20 (42%) patients had bilateral disc oedema (papilledema) and they were the majority of the Unilateral disc atrophy was found in 9 (19%) patients while bilateral atrophy in 16 (34%). No case of Foster Kennedy Syndrome was seen (unilateral disc oedema with contralateral disc atrophy).

**TABLE IX: Relationship between Disc appearance & BCVA (n=120 eyes)**

<table>
<thead>
<tr>
<th>BCVA</th>
<th>Normal</th>
<th>Oedema</th>
<th>Atrophy</th>
<th>Total</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>6/6 - 6/18</td>
<td>33</td>
<td>34</td>
<td>0</td>
<td>67</td>
<td>56</td>
</tr>
<tr>
<td>&lt;6/18-6/60</td>
<td>4</td>
<td>4</td>
<td>3</td>
<td>11</td>
<td>9</td>
</tr>
<tr>
<td>&lt;6/60 - 3/60</td>
<td>0</td>
<td>1</td>
<td>4</td>
<td>5</td>
<td>4</td>
</tr>
<tr>
<td>&lt;3/60-1/60</td>
<td>0</td>
<td>3</td>
<td>14</td>
<td>17</td>
<td>15</td>
</tr>
<tr>
<td>&lt;1/60 - PL</td>
<td>0</td>
<td>0</td>
<td>4</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td>NPL</td>
<td>0</td>
<td>0</td>
<td>16</td>
<td>16</td>
<td>13</td>
</tr>
<tr>
<td>TOTAL</td>
<td>37</td>
<td>42</td>
<td>41</td>
<td>120</td>
<td>100%</td>
</tr>
</tbody>
</table>

p=0.0001

There was a statistically significant correlation between eyes with optic atrophy and reduced visual acuity (p=0.0001). 8 eyes with disc oedema had reduced visual acuity.
5.5 PERIMETRY RESULTS

FIGURE XI: Distribution of Visual fields (n=121 eyes)

84 (70%) eyes were able to perform the Goldman Visual Fields test, 34 (28%) had normal fields. 36 (30%) eyes were unable to perform the test. 23 (19%) eyes had generalized constriction and this was the most common visual field defect.
TABLE X: Distribution of Radiological diagnosis according to Visual Fields (n=H4 eyes)

<table>
<thead>
<tr>
<th>Radiological diagnosis</th>
<th>Normal</th>
<th>Homonymous Hemianopia</th>
<th>Temporal Hemianopia</th>
<th>Generalised Constriction</th>
<th>Quadrant Nopia</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Suprasellar tumours</td>
<td>2</td>
<td>5</td>
<td>6</td>
<td>7</td>
<td>1</td>
<td>21</td>
</tr>
<tr>
<td>Frontal lobe tumour</td>
<td>7</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>8</td>
</tr>
<tr>
<td>Temporal lobe tumour</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>Parietal lobe tumour</td>
<td>7</td>
<td>3</td>
<td>0</td>
<td>4</td>
<td>0</td>
<td>14</td>
</tr>
<tr>
<td>Occipital lobe tumour</td>
<td>3</td>
<td>2</td>
<td>0</td>
<td>3</td>
<td>0</td>
<td>8</td>
</tr>
<tr>
<td>Sphenoidal ridge meningioma</td>
<td>5</td>
<td>2</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>9</td>
</tr>
<tr>
<td>Olfactory groove meningioma</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>3</td>
<td>0</td>
<td>6</td>
</tr>
<tr>
<td>Others</td>
<td>5</td>
<td>3</td>
<td>0</td>
<td>2</td>
<td>3</td>
<td>13</td>
</tr>
<tr>
<td>Total</td>
<td>34</td>
<td>16</td>
<td>6</td>
<td>23</td>
<td>5</td>
<td>84</td>
</tr>
<tr>
<td>Percentage</td>
<td>41%</td>
<td>19%</td>
<td>7%</td>
<td>27%</td>
<td>6%</td>
<td>100%</td>
</tr>
</tbody>
</table>

The classic temporal hemianopia was seen in 6 (7%) eyes all with suprasellar tumours.
The patients referred for routine follow-up were 45 (75%), constituting mainly of those who had normal vision, visual impairment, monocular blindness, diplopia and proptosis. Those with severe visual impairment were referred for Low Visual Assessment, while all blind patients were referred for rehabilitation.
6. DISCUSSION

A total of 60 patients were studied. 32 (52%) females, 28 (48%) males, with a F:M ratio of 1.1:1 (Figure I). There was an approximately equal distribution between the two sexes. 36 (60%) patients were recruited from the clinic, while 24 (40%) from the ward (Table I). The clinic is an out-patient neurosurgical unit and therefore more patients are seen. The ward has the in-patients who have a prolonged hospital stay especially during the post-operative period. The turnover of patients is therefore not as high as in the out-patient clinic.

The 60 patients ranged from 15 to 69 years. The mean age was 37 years and the median 36 years. The age bracket of 36 - 45 years had the largest number of participants with 24 (39%) patients, consisting of 14 (23%) females and 10 males (17%) as shown in Figure II. There was no statistically significant difference between the males and the females in any of the age groups (p=0.261). Sood et al. 9 in his study of supratentorial tumours found most patients were between 21-40 years, showing a similar pattern as this study.

52 (85%) patients gave a history of headache (Figure III). This was the most common symptom. Sood et al found 68% with headache6, while Honig PI et al had 75%.3 The headache is a non-localizing sign of raised intracranial pressure and was described in association with nausea or vomiting in the morning.

History of ocular signs and symptoms were reported in 44 (72%) of patients. These symptoms were either visual disturbance, proptosis, diplopia or hemi field loss. This is a
higher number than Huber et al. findings of 50%.
Anderson et al got 30% 2 while Herold et al had 62%. 4
The higher number of ocular symptoms in this study may be
explained by the fact that most of the patients interviewed were from rural health facilities
all over the country. They may have delayed in coming to KNH due to inability to access
of Primary Health Care Services or financial constraints. By the time they are finally seen
in KNH, their symptoms will have deteriorated.

Despite having 43 (72%) patients with ocular symptoms, only 29 (48%) had previously
been examined by a trained eye specialist defined in this study as an ophthalmic nurse,
ophthalmic clinical officer or ophthalmologist (Figure IV). Those who had not been
previously examined may have had limited access to the few eye units in the periphery.
Primary health care workers may have also delayed in referring these patients to tertiary
hospitals e.g. by prescribing pain killers for chronic headaches.

Suprasellar tumours were the most common radiological findings with 17 (28%) of the
cases (Table II). Amongst the suprasellar tumours, 10 were pituitary adenomas, 2 were
meningiomas. 2 craniopharyngiomas, 1 glioma and the remaining 2 were unclassified.
Parietal lobe tumours were the second in frequency with 10 (17%) cases. The least
common tumour was the olfactory groove meningioma with only 3 (5%) cases. The
tumours under the category of "others" consisted of lesions in the cerebellum, thalamas,
choroidal plexus and corpus callosum. The tumours were mainly found in the 36-45 age
group and the suprasellar tumours were the majority in this category with 8 (13%) patients
(Table III). There was no statistically significant correlation difference between the type
of the tumour and age group (p=0.128).
30 (50%) patients were not on any treatment as at the time of the study and 13 (22%) were on medication (Figure V). The neurosurgeons prescribe dexamethasone for reduction of brain oedema, and epanutin or carbamazepine as an anti-convulsant. 16 (27%) patients had had surgical excision of their tumours. Two of these had also completed radiotherapy sessions after their surgeries. Another two had VP shunts inserted at the time of surgical excision. Only one patient had a VP shunt without surgical excision of the tumour.

It was difficult to know what the pre-operative ocular findings were for the patients who had undergone surgery. None of their files had any ophthalmic assessment prior to the surgery and yet 5 out of the 16 post-surgical patients were blind and 1 had severe visual impairment. The patient with VP shunting done was also blind as at the time of the study. These findings should influence both neurosurgeons and ophthalmologists to take a keen interest in ensuring appropriate ophthalmic examinations are carried out both pre and post surgical excision. The patient should be well aware of his/her visual status and adequately informed on the prognosis after the surgery. Most of the blind patients interviewed were hopeful that the surgery would improve their visual status, and were not forewarned of the possibility of deterioration or no change in their vision. Without any ophthalmic assessment, the patient is literally left in the dark.

40 (67%) respondents had normal vision. 10 of whom had had their tumours surgically excised (Figure VI). 9 (15%) patients with parietal lobe tumours were the majority amongst those with normal vision, followed by 7 (12%) patients with sphenoidal ridge meningiomas (Table IV). This can be explained by the fact that parietal lobe tumours are located some distance from the visual pathway thus their presence is unlikely to cause any
visual symptoms unless the tumour is quite large in size. The sphenoidal ridge tumours are usually unilateral and are most like to cause monocular blindness.

Of the 5 (8%) patients with visual impairment, 4 had suprasellar tumours, while 1 had an olfactory groove meningioma. 4 (7%) patients had severe visual impairment. 11 (18%) patients were blind. 6 of whom had suprasellar tumours while 3 had frontal lobe tumours. The p value of 0.039 shows there is a statistically significance correlation between tumours compressing the anterior pathway (frontal, suprasellar) and visual impairment. However, this study did not factor in the size of the tumour whereby the bigger the tumour the more likely it is to compress the visual pathway at any level and cause a significant reduction in vision.

The 11 (18%) patients with blindness found in this study is much higher prevalence than in a study by Jallu A. et al who had a prevalence of 12.8% and Sood et with 8%. The higher prevalence from this study can be explained by the fact that the patients may have had prolonged symptoms and were unable to access medical facilities in the periphery. Also it may have been expensive for the patient to pay for the radiological investigations, transport to and from their homes and hospital consultation. All these confounding factors can be liked to delay in coming to referral settings and subsequently deterioration of vision. Lestak et al. concluded that an early diagnosis and appropriate therapy of patients with intracranial tumours decreases the risk of permanent vision defects. 5 (8%) out of the total 11 blind patients were post-operation. Unfortunately, none of them had a pre-operative ophthalmic examination.
Monocular blindness was defined as a patient with one eye with normal vision and a blind fellow eye (visual acuity of < 3/60). 8 (13%) patients satisfied this criteria out of the total 60 (100%) patients (Table V). Half of these patients (4 cases) had sphenoidal ridge meningiomas, suprasellar tumour accounted for 3 cases, while parietal tumours one case. Out of a total of 7 (100%) patients with sphenoidal ridge meningioma, 4 (57%) had monocular blindness. This finding is because of the fact that the tumour is usually unilateral and compresses or infiltrates the ipsilateral optic nerve. Out of the 17 (100%) patients with suprasellar tumours, 3 (18%) had unilateral blindness. This may be due to the tumour growing anterior to the optic chiasma at the suprasellar region with compression of the ipsilateral optic nerve. Only 1 (10%) patient with a parietal lobe tumour presented with unilateral blindness. This tumour could have been large enough in size to cause direct pressure on the ipsilateral optic nerve.

Colour vision was found to be abnormal in 34 (28%) eyes and normal in 46 (47%) eyes (Figure VIII). These findings were much lower than Repta et al. who got an abnormal colour vision of 71%.46 Out of the 34 (28%) eyes with abnormal colour vision, 12 had a normal visual acuity, indicating that they had a mild degree of optic nerve damage, while the remaining 22 eyes had vision worse than 6/18. All the eyes that were unable to perform the colour vision test (30 eyes) were blind.

30 (50%) patients had a normal pupillary reaction for both eyes. RAPD was demonstrated in 17 (28%) patients (Figure IX). These patients had partial optic nerve involvement in the affected eye. TAPD was seen in 11 (18%) patients, and this was in the eyes that were NPL. 2 (3%) patients demonstrated Wernicke Hemianopic pupil and they had corresponding hemianopic visual field defects.
Defective extraocular motility was elicited in 16 (26%) patients (Table VII). 11 patients had abducens nerve palsies and 4 of them who complained of diplopia. Eye patches or frosted lens were prescribed for these patients. Oculomotor nerve palsy was seen in 1 patient, it was partial and the patient had mild ptosis. He had a frontal lobe tumour that may have spread to the cavernous sinus or superior orbital fissure compressing the oculomotor nerve. Restriction of extraocular motility in all gazes was noted in 4 patients with proptosis. They had intraorbital extension of the tumour, thus causing restriction of ocular movements.

Proptosis was seen in 6 (10%) patients (Table VI). 3 of whom had frontal lobe tumours while the other 3 had sphenoidal ridge meningiomas. This is consistent with the fact that these anterior tumours have the ability to have intraorbital extension. One patient with a massive frontal lobe tumour had bilateral proptosis, bilateral blindness and early signs of exposure keratopathy. Tear substitutes were prescribed for this patient and was referred for rehabilitation. All the proptosed eyes had very poor vision ranging from severe impairment to NPL. On fundoscopy, 3 eyes with proptosis had tortuous vessels, and 1 eye had choroidal folds.

Nystagmus was observed in 10 (17%) patients. 4 of these patients had occipital lobe tumours. A study by Tsekov et al showed a lower prevalence of nystagmus of 8%\(^{24}\) though he studied tumours in the middle cranial fossa.

37 (31%) eyes had a normal disc appearance (Figure X). 33 of these eyes had normal vision, while 4 eyes had visual impairment (Table IX). These 4 eyes may have had early
optic atrophy not clinical detectable, hence the appearance of a normal disc. It is therefore important to do other test to exclude optic atrophy in the presence of a "normal disc" appearance e.g colour vision, pupillary reaction, visual fields.

Disc oedema was observed in 42 (35%) eyes. 34 eyes with disc oedema had normal vision, 4 had visual impairment, and 3 were blind. These findings show that an eye with disc oedema does not always have normal vision. There could be a partial optic atrophy that is obscured by the oedematous optic nerve head. It is always important to have an objective visual acuity assessment, pupillary reactions, colour vision followed by a fundoscopy in all patients with disc oedema. Bilateral disc oedema (papilloedema) was seen in 20 (33%) patients (Table VIII). Papilloedema is a non-specific sign of raised intracranial pressure. Sood et al detected papilloedema in 8% of patients with supratentorial tumours. This is much lower than Huber who found a prevalence of 59%.

In this study, all the tumours causing papilloedema were supratentorial with the majority being parietal tumours (4 patients), followed by suprasellar tumours (3 patients). Unilateral disc oedema was present in only 2 (3%) patients both with suprasellar tumours. This may have been due to unequal compression of the optic nerve in the ipsilateral side.

Disc atrophy was seen in 41 (34%) eyes, 3 had visual impairment, 4 had severe visual impairment and the remaining 34 were blind. This demonstrates that partial optic atrophy can cause varying degrees of reduction in vision ranging from visual impairment to NPL. Unilateral disc atrophy was observed in 9 (15%) patients, 4 of whom had sphenoidal wing meningiomas. These tumours are located on one side of the brain and cause an ipsilateral compression or infiltration of the optic nerve. 16 (27%) patients had bilateral optic atrophy, 9 of whom had suprasellar tumours and 3 had frontal lobe tumours. These
tumours are anteriorly located and can cause direct compression to both optic nerves. In a study by J. M Muthuuri on sellar and juxtasellar tumours in KNH, fundoscopy revealed bilateral optic atrophy in 97% of the patients. This figure is quite high as he only studied suprasellar tumours that are adjacent to the optic nerve. In a similar study of suprasellar meningioma and blindness in Saudi Arabia, the incidence of blindness in one or both eyes at presentation was high (42.2%) with 12.8% being blind at admission due to optic atrophy. This concludes that the more anterior the tumour is, the more likely to have bilateral disc atrophy, therefore increasing the likelihood of blindness.

In 84 (70%) eyes it was possible to perform the Goldman Visual Fields test, while in 36 (30%) it was not because either the eye was blind, or the patient was too sick to follow instructions (Figure XI). 34 (41%) eyes had normal visual fields, this constituted mainly tumours in the frontal and parietal lobe (14 eyes). This can be explained by the fact that these tumours are not in close proximity with the visual pathway unless they are large enough to cause compressive symptoms. Bilateral normal fields were observed in 11 patients, 6 of them had either frontal lobe (3 patients) or parietal lobe (3 patients) tumours.

Homonymous hemianopia was observed in 16 (19%) eyes, 5 of which had suprasellar tumours. 3 had parietal tumours (Table X). This can be explained by the direct pressure effect on the optic tract by these tumours. Temporal hemianopia was observed in 6 eyes all of which had suprasellar tumours that directly compress the optic chiasma. Generalized constriction of the visual field was the most common defect observed in 23 (27%) eyes. It signifies marked compression by the tumours along the visual pathway and is not a useful sign in locating the site of the lesion.
45 (75%) patients were advised to come for follow up routinely at the KNH Eye Clinic (Figure XII). This group consisted of all the patients with normal vision and visual impairment. 10 patients who already had surgical excision of their tumours were warned that any gradual reduction in vision could be associated with tumour recurrence therefore the need for routine comprehensive eye check-ups. Ocular lubricants were prescribed for the 6 patients with proptosis, 4 of them already had signs of exposure keratopathy. None of them required a tarsorrhaphy.

All the 8 patients with monocular blindness were to advised to attend the eye clinic to get prescription of protective plastic spectacles. These are helpful in giving added protection from eye injuries for the seeing eye. The problems associated with a reduced visual field were explained to the patient.

The 4 patients with diplopia were either given patches or had their spectacles frosted. They were then referred to the Orthoptic clinic for monitoring the improvement of the abducens nerve palsy and subsequent regression of the diplopia.

4 (7%) patients who were had severe visual impairment were advised on Low Vision Assessment and subsequent prescription of Low Vision Devices. All had not yet had surgical removal of their tumours. Skrzypezak J et al reported an improvement in visual acuity after surgical intervention in 41% of patients with hypophyseal tumours. 59% had regression of the scotomas. With the possibility of improvement in visual acuity after surgery, the 4 patients were advised to come for assessment of their visual status regularly from 6 weeks after their operations. If there would be no improvement, referral for Low Vision Assessment would be done. All the II (18%) blind patients were referred for
rehabilitation. 5 of them were post-operation patients and none had any form of psychological counselling or training on basic skills to enable them to lead an independent life. 4 patients had complete blindness with their vision being NPL, while another 7 had BCVA of 3/60-1/60. This group of patients had residual navigation vision.

Rehabilitation of the blind is a component of tertiary intervention and involves the following 3 phases:

**Phase I: Assessment and counselling**

a) Medical and psychological assessment and counselling  
b) Training of basic skills  
c) Socio-economic assessment  
d) Education and career assessment  
e) Career training assessment.

**Phase II: Career Training**

**Phase III: Occupational/Job placement and follow-up support service**

For patients with irreversible visual loss, early referral to The Kenya Society for the Blind and Kikuyu Hospital Low Vision Unit, may very well be what they need to boost their confidence, and expedite their integration into an independent social and professional life. Currently there are no trained personnel for rehabilitation nor specialized rehabilitation services in KNH to cater for these patients with.
7. CONCLUSION

1. Ocular symptoms were present in 44 (73%) of patients with intracranial neoplasms. There is a higher prevalence of ocular manifestations in patients with intracranial tumours in our set up compared to centres in developing countries.

2. In patients with intracranial neoplasms, the prevalence of bilateral blindness is 18% and monocular blindness is 13%.

3. The number of patients with intracranial tumours who had ophthalmic assessment before the study was low.

4. Tumours located in close proximity with the anterior visual pathway are most likely to compress the optic nerve and chiasma causing optic atrophy and subsequent blindness.

5. The most common encountered visual field defect is the generalised constriction which is non-specific for localising the site of the tumour.

6. Patients who are blind as a result of intracranial neoplasms are not properly rehabilitated. There is no protocol for the referral of these patients to other specialised institutions offering Low Vision Assessment and rehabilitation services.
8. RECOMMENDATIONS

1. Neurosurgeons should work closely with ophthalmologists to ensure complete assessment of patients with intracranial tumours both pre operative and post operative.

2. Drop in the visual status may be an early sign of recurrence of the tumour after surgical excision. This may be accompanied by other symptoms of raised intracranial pressure e.g headaches, vomiting. All patients should be forewarned about this possibility.

3. Apart from reduction in vision acuity, patients with cranial nerve palsies, diploplia and proptosis need additional care and follow up in the eye unit.

4. Referral for Low Visual Assessment and Rehabilitation needs to be formalised. There should be in-patient rehabilitation services within Kenyatta National Hospital.
APPENDIX I

QUESTIONNAIRE

CODE NO: _______________________________ DATE: _______________________________

LOCATION (Clinic - I, Ward = 2) □

NAME ___________________________ I/P NUMBER ____________________________

AGE ___________________________ SEX (Male = 1, Female = 2) □

CURRENT NEUROLOGICAL SIGNS/SYMPTOMS

Symptoms if present (Yes=1, No=0)

Headache □ 1

Hemiplegia/hemiparesis □ 1

Affected mental status □ 1

Seizures □

Others (specify)

OPHTHALMIC HISTORY (Yes=1, No=0)

Ocular Symptom □

Previous ophthalmic assessment □

EXAMINATION FINDINGS

<Refer to WHO categories of visual impairment Appendix II for 14.15>

Vision

BCVA

Colour Vision

Pupillary reaction

Hirshberg Test Extra ocular motility

Cranial Nerve(s) affected

Diploplia

Nystagmus

Proptosis

Lids and peri-ocular changes
Anterior Segment Examination

Conjuctiva
Cornea
Anterior chamber
Lens

Posterior Segment Examination

Disc
Macula
Vessels

Peripheral Fundus

Perimetry Findings

Radiological diagnosis

Treatment or intervention to date

Proposed Ophthalmic Management

1= Refer for Rehabilitation
2= Low Visual Assessment
3= Routine follow-up
4= Medication
APPENDIX II

WHO categories of visual impairment

<table>
<thead>
<tr>
<th>Category</th>
<th>Visual Acuity- with BCVA in the better eye</th>
<th>Degree of visual Impairment</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>6/6 - 6/18</td>
<td>Normal Vision</td>
</tr>
<tr>
<td>1</td>
<td>&lt;6/18 - 6/60</td>
<td>Visual Impairment</td>
</tr>
<tr>
<td>2</td>
<td>&lt;6/60 - 3/60</td>
<td>Severe Visual Impairment</td>
</tr>
<tr>
<td>3</td>
<td>&lt;3/60 - 1/60</td>
<td>Blind</td>
</tr>
<tr>
<td>4</td>
<td>&lt;1/60 - Light Perception</td>
<td>Blind</td>
</tr>
<tr>
<td>5</td>
<td>No Light Perception</td>
<td>Blind</td>
</tr>
<tr>
<td>6</td>
<td>Undetermined of unspecified</td>
<td></td>
</tr>
</tbody>
</table>

If the extent of visual field is taken into account, patients with a field not greater than 10° but greater than 5° around central fixation should be placed in category 3 and patients with a field no greater than 5° around central fixation should be placed in category 4, even if visual acuity is not impaired.
APPENDIX III

CONSENT EXPLANATION

Title of project: "Ocular manifestations in adults with intracranial neoplasms attending the neurosurgical unit in KNH"

I, Dr. Sheila Marco would like to give you information on the study titled "Ocular manifestations in adults with space occupying lesions attending the neurosurgical unit in KNH"

Intracranial Tumours and their effect on vision:

Visual disturbances are frequently observed in patients with intracranial space occupying lesions. The signs and symptoms can be due to direct pressure of brain structures by the tumour or to mass effect producing vascular, inflammatory or degenerative lesions. A complete visual assessment is necessary, as it will enable you to know your visual status and will be used as a guideline for any improvement or reduction in eye symptoms after any intervention.

Eye Examination

I will take a short history from you regarding your condition. I will then examine your visual acuity, colour vision, pupillary reactions to light and eye movements. I will later examine your eyes with a Slit Lamp which is a machine with a magnifier. After that I will do a Visual Field assessment using the Goldman Perimeter Machine. For a fundoscopy, I will dilate your pupils with eye drops. I will later inform you of your visual status and advise you accordingly.
Confidentiality'

All personal information gathered from you as my patient in this study will be kept confidential. Numeric codes will be used for data entry and all information will be used solely for the purpose of demonstrating the objectives of the study.

Informed Consent

For you to participate in this study, a signed consent is required. The eye examination is free and any intervention necessary will be communicated to you.

CONSENT FORM

| __________| of P.O Box

Confirm that I have agreed to participate in a study being carried out on "Ocular manifestations in adults with intracranial neoplasms attending the neurosurgical unit in KNH". I confirm that Dr Sheila Marco of University Of Nairobi has explained to me and I understand, the purpose of the study.

Signature/ Thumb print (patient/guardian) ..................... Date

Signature Dr. Sheila Marco ........................................ Date

Signature/ Thumb print (Witness) .......................... Date
REFERENCES


12. Warnick R L, Trobe J D. Bilateral optic nerve compression as a mechanism for the FKS. Ophthalmology. 1989 Dec; 96(12): 1793-8


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