Clinical Pattern and Imaging Findings of Intracranial Space Occupying Lesions in Children

By

Dr. Gawahir Mohamed Ahmed Mukhtar

M.B.B.S (U of K) 1999

A Thesis submitted in partial fulfillment for the requirements of clinical MD in

Paediatrics and Child Health

March 2007

Supervisor

Prof. Zein A. Karrar

FRCP(Lond)FRCPCH(UK),MRCP(UK)

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بسم الله الرحمن الرحيم

{ ولولا فضل الله عليك ورحمة لهتمت طائفة منهم أن يضلوك وما يضلون إلا أنفسهم وما يضرونك من شيء وأنزل الله عليك الكتاب والحكمة وعلمه ما لم تكن تعلم وكان فضل الله عليك عظيمًا }

صدق الله العظيم
سورة النساء
ال아ية (113)
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To my father, mother, sisters and brothers

To my friends everywhere…

for their love, care and encouragement
My great thanks to Prof. Zein A. Karrar for his meticulous supervision, guidance and patience. My gratitude to Dr. Arbab my co supervisor for his help and support.

I am thankful my colleagues in neurosurgical department for their great help and support.

My thanks extend to Mr. Hassan Ali for his statistical analysis. I do appreciate the effort made by Mrs. Fadia and Miss. Amona in the computer work.

My thanks to all other colleagues not mentioned individually here.
ABSTRACT

A cross sectional and prospective hospital based study in Elshaab
Teaching Hospital (National Center For Neurological Science),
Khartoum Teaching Hospital and Dr Gafaar Ibn Auf Children's
hospital. The duration of the study was from 1st of September 2005
to the end of February 2007.

The objectives were to study the clinical presentations, causes
and the imaging findings associated with the intracranial space
occupying lesions.

The study included 103 children; male to female ratio was
1.7:1. One third of children were below 5 years of age, 31%
between 5-10 years and 35% were above 10 years. The study tools
included questionnaire, clinical examination and investigations
including brain C.T and MRI studies.

The commonest intracranial SOL was astrocytoma (35.0%),
followed by abscess (19.4%) and medulloblastoma in (10.7%).

Headache was found to be the main presenting symptom
occurring in (59.2%). Cranial nerves involvement was the
commonest clinical sign detected in (43.7%) followed by gait disturbance detected in (42.7%) respectively. The commonest fundal change was papilloedema (18.5%). Most of lesions detected were supratentorial (72.8%) and they were found to be commonly benign in nature (54.7%), malignant lesions were confined mainly to infratentorial area (78.6%).

Malignant lesions affected males more than females (1.6:1). Ventricular dilatation was the common imaging finding detected in (46.7%). Malignant lesions (65.6%) needed V.P shunt operation more than benign lesions (35.0%).

Congenital heart disease (4.8%) was a risk factor for brain abscess.

Tuberculoma was detected less than expected (1.9%).

The main recommendations are: To consider headache and vomiting early warning signs, proper evaluation of children and use of CT and MRI to detect lesions early. Decentralization of services and training of general doctors and pediatricians.
لا يوجد نص يمكن قراءته بشكل طبيعي من الصورة المقدمة.
The heart is in the imagination of the afflicted patients. We find 4.8% of them are more frequent and are more expected, which is less than the normal level of the study. This is a result of the patient's health and is very important.

Regarding the treatment and surgery, we use the general treatment and surgery. We apply the general treatment and surgery in the patients with previous treatment and surgery.

Some abbreviations are used.
<table>
<thead>
<tr>
<th>ADH</th>
<th>Antiduretic hormone</th>
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<tbody>
<tr>
<td>AIDS</td>
<td>Acquired immunodeficiency syndrome</td>
</tr>
<tr>
<td>ALL</td>
<td>Acute lymphocytic leukaemia</td>
</tr>
<tr>
<td>AVM</td>
<td>Arteriovenous malformation</td>
</tr>
<tr>
<td>CNS</td>
<td>Central nervous system</td>
</tr>
<tr>
<td>CPA</td>
<td>Cerebellopontine angle</td>
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<tr>
<td>CT</td>
<td>Computed tomography</td>
</tr>
<tr>
<td>HC</td>
<td>Hydatid cyst</td>
</tr>
<tr>
<td>MRA</td>
<td>Magnetic resonance angiogram</td>
</tr>
<tr>
<td>MRI</td>
<td>Magnetic resonance imaging</td>
</tr>
<tr>
<td>NS</td>
<td>Neuro-schistosomiasis</td>
</tr>
<tr>
<td>PNET</td>
<td>Primitive neuroectodermal tumour</td>
</tr>
<tr>
<td>SOL</td>
<td>Space occupying lesion</td>
</tr>
<tr>
<td>TB</td>
<td>Tuberculosis</td>
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<tr>
<td>TBM</td>
<td>Tuberculous meningitis</td>
</tr>
<tr>
<td>VDRL</td>
<td>Veneral disease research labrotory</td>
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<td>V.P</td>
<td>Ventriculoperitoneal</td>
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Chapter One

1. INTRODUCTION AND LITERATURE REVIEW

1.1. Definition:

The term intracranial space occupying lesion is generally used to identify any lesion, whether vascular or neoplastic or inflammatory in origin which increases the volume of intracranial contents and leads to a rise in the intracranial pressure \(^{(1)}\).

1.2. Classification of intracranial space occupying lesions:

I. Congenital: Dermoid, Epidermoid, Teratoma.

II. Traumatic: Subdural and Extradural haematoma

III. Inflammatory: Abscess, Tuberculoma, Syphilitic gumma, Fungal granulomas.

IV. Parasitic: Cysticercosis, Hydratid cyst, Amebic abscess, Schistosoma japonicum.

V. Neoplasms;

a) Tumors arising from neural structures: Gliomas – astrocytoma, ependymoma, oligodendroglioma, germinoma, medulloblastoma.

b) Tumors arising from appendages: Meningioma, schwannoma,
chondroma, osteoma.

c) Pituitary lesions: Pituitary adenoma, Craniopharyngioma.
d) Vascular lesions: Angioma, Hemangioblastoma, Papilloma of choroid plexus.
e) Secondary neoplasms.

1.3. Brain Tumours:

The term "brain tumours" refers to a mixed group of neoplasms originating from intracranial tissues and the meninges with degrees of malignancy ranging from benign to aggressive. Each type of tumour has its own biology, treatment, and prognosis and each is likely to be caused by different risk factors. Even "benign" tumours can be lethal due to their site in the brain, their ability to infiltrate locally, and their propensity to transform to malignancy. This makes the classification of brain tumours difficult and creates problems in describing the epidemiology of these conditions(2).

1.3.1. Incidence and Distribution:

Malignant tumours of the brain are a rare occurrence accounting for approximately 2% of all cancers in adults. Approximately 4400 people are newly diagnosed with a brain tumour each year in the UK compared to over 40000 women with breast cancer and approximately 25000 men with
prostate cancer. The overall annual incidence rate of all brain tumours is 7 per 100,000 population\textsuperscript{(2)}.

Brain tumours are second only to leukaemia as the most prevalent malignancy in childhood, and they account for the most common solid tumours at this age group, comprising 15–25% of all paediatric malignancies\textsuperscript{(1)}. Different proportions of histological subtypes are present in children compared to adults, with gliomas (approximately 40%) and medulloblastomas (approximately 25%) mainly arising infratentorially, with the remainder, germ cell tumours and craniopharyngiomas, occurring in the midline. There is a small peak in incidence in early childhood accounted for by medulloblastomas. Studies in the USA, Sweden, and the UK have reported what appear to be true rises in incidence over the last three decades which are unexplained by changes in diagnostic practice, treatment\textsuperscript{(2)}. Metastatic brain tumours are common in adults but relatively rare in children\textsuperscript{(3)}. A recent study by the National Cancer Institute shows that a significant increase in childhood brain tumours brings them the dubious distinction as the most common paediatric tumour.

In 1974, brain tumours occurred at an annual rate of 2.35 per 100,000 children younger than 15 years, which increased to a corresponding value of 3.45 in 1994. Similar changes in incidence were noted in ALL, which
showed a corresponding change in annual incidence from 2.74 to 3.33 per 100,000 children younger than 15 years. This rise in brain tumour incidence among children is attributed to improved diagnostic methods and more awareness of brain tumours among physicians. However, environmental factors cannot be completely ruled out\(^4\).

The intracranial tumours of childhood differs from the adult forms in term of distribution within the brain, histological characteristics and prognosis\(^5\). They can present in many ways depending on the location, type and rate of growth of tumour and the age of the child\(^5\).

In the study conducted by Abu Salih and Abdul Rahman the incidence of brain tumours in the Sudan in the period between 1971-1981 was 7% (123 patients) of all neurosurgically admitted patients (1757 patients). They concluded that their series showed a high incidence of meningioma and very low incidence of acoustic neuroma compared to regional and international series\(^6\). Incidence of brain tumours in the Sudan as reported by Abu Salih and Abdul Rahman was as follows\(^6\): Menengiomas 45.5%, Gliomas 39.8%, Pituitary adenomas 10.6%, Craniopharyngiomas 5.7%, Acoustic neuromas 0.8% and metastatic tumours 6.5%.

Brain tumours were found to be more common in males (63.4%) and most commonly affect those in the third decade, followed by those in the
Bella Ahmed Elsherif, studied the pattern of malignant disease in children presenting to Khartoum hospital during the period July 1982-May 1983 in one hundred children and he found that the age distribution was similar to other works for all forms of cancer, apart from the peak incidence of leukemia, which was above the age of 4 years, while in Western countries, it was reported to be between 2-4 years. The male preponderance in all forms of cancer. Lymphoma (25% of all forms of cancer), was the leading malignant disease. Leukemia was the fourth in rank order (13%). Wilms tumour ranked the second in his study (14%). The percentage of retinoblastoma in the study (11%) contrasted significantly with the paucity of this form of cancer elsewhere. Carcinoma nasopharynx (10%) was another good example for such a discrepancy. Brain tumours ranked the seventh in his study (7%). Interestingly, some rare forms of cancer such as skin, liver and ovarian cancer, in Africa as well as Western series, have been found relatively prominent (2%) \(^{(7)}\).

In Saudi Arabia, malignant astrocytoma accounts for 16% of all intracranial space-occupying lesions \(^{(8)}\) and although patients harboring this tumour are being managed in most neurosurgical units all over the country published data from Saudi Arabia relating to the survival of patients with
malignant astrocytoma has been limited to one report which focused on malignant astrocytoma in children\(^{(8)}\).

In Nairobi, Wanyoike reported that thirty seven children were treated for posterior fossa tumours between 1998 and 2003, twenty four were females while thirteen were males giving a male: female ratio of 1:1.8. The age varied between 2-16 years. Cerebellar symptoms were the most common mode of presentation (30\%) followed by headaches and vomiting. Twenty percent of patients were blind at presentation probably due to chronic effects of raised intracranial pressure. Out of 11 patients with histological diagnosis of medulloblastomas, over 99\%, were females and only one was a male. Astrocytomias were evenly distributed at five males and six females. Posterior fossa tumours in this study are more common in females than in males, M: F ratio of 1:1.8. Over 90\% of medulloblastomas are found in female children making it a predominantly female tumour as opposed to available literature\(^{(9)}\).

Mwang studied the frequency, mode of presentation and outcome following treatment of gliomas in patients treated at the Kenyatta National Hospital in Nairobi. Two hundred and fourteen histologically confirmed intracranial tumours were included. Ninety seven (45.8\%) of these were gliomas of which eighty one were astrocytomias, ten ependymomas and six
oligodendrogliomas. Meningiomas were the next common tumours (34.4%). Gliomas affected the young age group most, with the peak in the first decade of life. Males were most affected with a male to female ratio of 1.4:1. Features of increased intracranial pressure were the commonest mode of clinical presentation. The parietal region was the commonest site of intracranial gliomas (37.5%) \(^{(10)}\).

In India, Desai reported that 102 patients under the age of 12 years with cerebellar astrocytomas were retrospectively analyzed. The clinical features were predominantly related to increase intracranial pressure and the location of the tumour. Twenty-six tumors were located in the vermis and 76 in the cerebellar hemisphere. The brain stem was involved in 20 patients. All 102 patients had a preoperative contrast-enhanced CT scan. Midline vermian tumors were predominantly solid and enhancing, whilst the hemispheric tumors were cystic and nonenhancing \(^{(11)}\).

1.3.2. Risk factors:

Brain tumours develop as a consequence of accumulated genetic alterations that permit cells to evade normal regulatory mechanisms and destruction by the immune system. These alterations may be in part or wholly inherited but any agents—chemical, physical or biological—that damage DNA are possible neurocarcinogens\(^{(2)}\).
1.3.3. Genetics:

Genetic predisposition to developing brain tumours is associated with certain inherited syndromes such as tuberous sclerosis, neurofibromatosis types 1 and 2, nevoid basal cell carcinoma syndrome, and syndromes involving adenomatous polyps. These syndromes account for 1–2% of all tumours. The Li-Fraumeni cancer family syndrome is also associated with a predisposition to brain tumours and specifically with mutations in the TP53 gene. Mutations in constitutional (that is, non-tumour tissue) TP53 have been linked to patients with gliomas\(^{(2)}\).

Familial aggregations of brain tumours occurring in different generations and sibships occur very rarely and the patterns of inheritance are inconsistent. In these situations common environmental exposures cannot be excluded as an explanation. Overall, it appears that only a very small proportion of brain tumours can be due to the effect of inherited predisposition\(^{(11)}\).

1.3.4. Immune factors: Viruses, Allergies & Infections:

In experimental animal models brain tumours can be induced by a number of viruses, including retroviruses, papovaviruses, and adenoviruses but there is little epidemiological support for this occurring in humans. At
one time it was thought that live polio vaccines contaminated with SV40 might increase the risk of brain tumours, but this was not supported by more detailed powerful studies. Direct examination of brain tumour tissue for evidence of a viral cause has shown the presence of different viral DNA sequences in some cases within separate pathological series. However, the mechanisms of how a virus might initiate malignant transformation remain unknown\(^{(11)}\).

Atopic diseases such as asthma, eczema, and allergies can be markers of immune dysfunction. In a number of independent studies from different countries atopic conditions have been shown to be "protective", particularly in the development of gliomas. Patients with gliomas report fewer symptoms of atopy compared to control subjects \(^{(12)}\).

In utero infections with influenza and chicken pox (varicella) have been cited as a risk factor but the case for this is not strong. Some recent epidemiological work on a series of children from the north west of England diagnosed with brain tumours has shown geographical distributions which are suggestive of an infectious aetiology for some of the tumour types \(^{(12)}\).

1.3.5. Chemicals:

N-nitroso compounds are found in the environment but the most
common source of human exposure is through foods, with vegetables and cured meats being major sources. Alkylating agents, such as methyl nitrosurea, are known transplacental carcinogens, particularly for brain tumours in rats (11).

1.3.6. Head trauma and injury:

Patients with brain tumours inevitably recall occurrences of trauma or injury to the head, and studies of patients’ reports are therefore subject to "recall bias". Some epidemiological investigations of the relation between head trauma/injury and the subsequent development of a tumour have attempted to overcome this by examining medical records, but these mainly fail to demonstrate any relation (12).

1.3.7. Tumour types:

Molecular cytogenetic techniques have helped to understand that brain tumours arise from genetic disruptions in cells, causing the cells to become neoplastic, but the causes of genetic disruption remain unclear. Brain tumours in the same family members are extremely rare. Furthermore, brain tumours can occur anywhere in the intracranial space (13). The tumour is named according to the cellular origin and the microscopic appearance. Most childhood brain tumours arise in the supporting cells of the brain (glia) and
are called gliomas. The most common is the astrocytoma, derived from astrocytes, which are major supportive cells. Astrocytes constitute nearly 40% of the total CNS cell population and are widely spread throughout the central nervous system including the optic nerves (13). The tumours are classified histologically from grade I through grade IV. Grade I and II are histologically benign, but grade III and grade IV are malignant, hence glioblastoma. Other tumours are ependymomas, gangliogliomas, choroid plexus papillomas, and oligodendrogliomas. Other common brain tumours in childhood arise in the primitive nerve cells, and are much more common in children than in adults (14). When they occur in the cerebrum, they are called “primitive neuroectodermal tumour (PNET). In the infratentorial location they are called medulloblastomas, while those in the pineal gland are called pineoblastomas. They are malignant, grow rapidly, and tend to spread through the CSF (14).

A third type of childhood brain tumour arises in the non-neuronal embryonal cells. They are germ cell tumours, craniopharyngiomas, or dermoids. Tumours arising in the meninges, nerve sheaths, or pituitary gland have an expansible nature with little or no infiltration to the brain or spinal cord. They are meningiomas, neurinomas, and pituitary adenomas respectively. They usually occur in adults, but can appear in children (15).
1.3.8. Brain tumours’ warning signs:

Space-occupying lesions such as brain tumours increase the intracranial pressure. This causes the brain structures to shift within the intracranial space, which may be life-threatening. Many childhood tumours are found in the skull midline, which often produces hydrocephalus. In such cases, hydrocephalus is the primary cause of symptoms rather than the tumour itself\(^4\).

Initial symptoms are early morning intermittent headaches and nausea, lasting for several months or years. Then, the headaches become more frequent and intense with emesis accompanied by dizziness, weakness, unsteadiness, and double vision. Increasing sleepiness, gait disturbance, ocular changes (abducens palsy in particular), visual disturbance, seizures, tremors or weakness of the extremities are more definitive signs for childhood brain tumour. The presentation depends on age group, the tumour’s location and histology. Infants and younger children typically present with irritability and rapidly expanding head size. They may also present with failure to thrive or precocious puberty\(^4\).

Papilloedema is another sign of hydrocephalus or a mass-induced rise in intracranial pressure. If papilloedema is persistent and untreated blindness
may result. Shifts of brain contents through the tentorial opening or the foramen magnum may occur, causing twisting and compression of the brain stem, leading to bradycardia, hypertension, and irregular respirations. Due to compression of the third nerve, the ipsilateral pupil is enlarged and does not react to the light, the patient exhibits changes in body and limb tone and posture (decortication and decerebration), and stops breathing\textsuperscript{(15)}.

1.3.9. Diagnosis:

CT images show skull, blood clots, and the calcified mass which appears white, while the brain is gray, and the CSF, fat and air appear black. Contrast dye injected intravenously enhances visualization of the blood vessels and most pathological conditions such as tumours. Thus, CT scan is capable of disclosing not only a tumour mass, its location and extension but also any associated pathological changes such as brain oedema around the tumour, hydrocephalus, hemorrhage, cystic formation, calcification, etc.

The risks are the requirement of sedation for the young child, possible allergic reaction to the intravenous contrast dye, and radiation exposure. Published reports warn that more than 20 rads may cause later cataracts\textsuperscript{(11)}.

Magnetic resonance imaging (MRI), which involves a high-powered
magnet, became available in the mid 1980’s. MRI images allowing a more
detailed examination than is possible with CT.

Due to the risks involved in standard angiography, MRA (magnetic
resonance angiography) has replaced angiography in most situations\(^{(11)}\).

1.4. Intracranial abscesses:

Intracranial abscesses are uncommon, serious, life-threatening
infections. They include brain abscess and subdural or extradural empyema.
A high number of brain abscesses are polymicrobial\(^{(16)}\).

1.4.1. Epidemiology:

Brain abscesses are rare in developed countries but are a significant
problem in the developing world; they occur more frequently in males and in
those younger than 40 years old. A decrease in meningitis due to the
Haemophilus influenzae vaccine has reduced the prevalence in young
children. The prevalence of brain abscess is higher in patients with HIV
infection\(^{(16)}\).

1.4.2. Pathology:

It begins with vascular seeding of the brain, producing early cerebritis
During the first 1-3 days. Inflammatory infiltrates of polymorphonuclear cells, lymphocytes and plasma cells follow within 24 hours. By 3 days the surrounding area shows a marked increase in perivascular inflammation. The late cerebritis phase develops approximately 4 to 9 days after infection during which time the centre becomes necrotic, containing a mixture of debris and inflammatory cells. Neovascularity is maximal at this time. Early reactive astrocytes surround the zone of infection and proceed to early capsule formation between approximately 10 to 13 days. At this time, the necrotic centre shrinks slightly and a well developed peripheral fibroblast layer evolves. The late capsule stage continues to evolve between 14 days and 5 weeks with continual shrinking of the necrotic centre and relative decrease in the inflammatory cells. The capsule thickens as reactive astrocytes proliferate.\(^{(17)}\)

Causative organisms include: Bacteria: common bacterial causes include Staphylococcus aureus, Streptococci, Bacteroides species and Listeria.

Fungi: Aspergillus, Candida, Cryptococcus, Coccidioides, Histoplasma, Blastomyces.

Protozoa: e.g. Toxoplasma gondii, Entamoeba histolytica, Trypanosoma
cruzi, Schistosoma.

Helminths, e.g. Taenia solium.

The frequency of fungal brain abscess has increased because of the frequent administration of broad-spectrum antimicrobials, immunosuppressive agents, and corticosteroids. They can originate from infection of adjacent structures, e.g. otitis media, dental infection, mastoiditis, sinusitis. Abscess formation can also develop following blood-borne spread from a remote site, e.g. in patients with cyanotic congenital heart disease, endocarditis, dental caries (18). In at least 20% of cases, no source can be identified.

1.4.3. Presentation:

Symptoms of onset may be sudden or subacute over several weeks.

Common presenting symptoms include fever, headache, changes in mental state (drowsiness, confusion), focal neurological deficits, seizures, nausea and vomiting, neck stiffness.

A suddenly worsening headache, followed by emerging signs of meningism, is often associated with rupture of the abscess (15).

The main signs include: fever, focal motor or sensory deficits, raised
blood pressure and bradycardia associated with raised intracranial pressure
papilloedema, ataxia, confusion, drowsiness, bulging fontanelle in infants.

1.4.4. **Differential Diagnosis includes:**

Meningitis, encephalitis, brain tumour or other intracranial space occupying
lesion.

1.4.5. **Investigations:**

Full blood count: marked leucocytosis. Raised ESR and CRP.

Renal function and electrolytes: serum sodium levels may be lowered as a
result of inappropriate antidiuretic hormone production.

At least two blood cultures should be taken and preferably before
antibiotics are started.

Serological tests are available for some pathogens. Lumbar puncture
and CSF analysis is rarely helpful (unless required to rule out meningitis)
and is contraindicated if increased intracranial pressure is present\(^{15}\).

CT scanning is the investigation of choice\(^{15}\), although MRI scan provides
greater contrast between cerebral oedema and the brain and early detection
of satellite lesions. MRI is especially useful for posterior fossa
In addition, MRI with intravenous gadolinium contrast is superior in demonstrating cerebritis surrounding oedema, the extent of the mass effect, or associated venous thrombosis. MRI with or without gadolinium is preferable to CT scan for demonstrating multiple lesions. The evolution of the abscess can be followed radiologically. In the early cerebritis stage, CT images reveal a low density lesion with ring enhancement. In the late cerebritis and early capsule stage, well-formed, ring enhancement is typically thin walled and uniform or nodular enhancement should raise the possibility of an alternative cause. Delayed contrast scans show diffusion of the contrast. Other ring enhancing lesions that may mimic the image of the brain abscess include primary and metastatic tumour, a resolving infarct or haematoma and rarely demyelinating disease (17).

Other investigations include, aspiration of abscess for culture and biopsy of cerebral lesion.

1.4.6. Management:

Early treatment with antimicrobial therapy, anticonvulsant therapy and measures to control increasing intracranial pressure is essential.

Initial antimicrobial therapy should be started immediately and then
modified according to the results of cultures. Initial therapy choices include high doses of penicillins, metronidazole, either gentamicin or chloramphenicol, vancomycin, meropenem and cephalosporins (e.g. cefotaxime). If fungal cause is suspected then amphotericin, flucytosine fluconazole or voriconazole are indicated. The treatment of choice for toxoplasmosis is a combination of pyrimethamine and sulfadiazine. Therapy should be given intravenously for at least the first week. Corticosteroids: intravenous dexamethasone is used if massive cerebral oedema is seen on the CT scan (17).

Once an abscess has formed, surgical excision or drainage through a burr hole, combined with prolonged antibiotics (usually 4-8 weeks), remains the treatment of choice.

Aspiration is the most common procedure and is often performed using a stereotactic procedure with the guidance of CT scanning or MRI. Craniotomy is generally performed in patients with larger, multiloculated abscesses and for those whose conditions failed to resolve.

Management of subdural or epidural empyema requires prompt surgical evacuation of the infected site and antimicrobial therapy (17).

Mawang in Nairobi studied the aetiology, mode of presentation and
outcome following treatment of brain abscesses. Sixty five patients with brain abscesses were seen at Kenyatta National Hospital. There were more male patients than females (ratio 2.4:1). Thirty eight per cent of the patients were children below the age of ten years. Trauma was the commonest cause of brain abscess. The aetiology was unknown in 24% of the cases. Sixty eight per cent of the patients had seizures. All the patients were diagnosed by computerised tomography (CT) scanning (18).

1.5. Tuberculosis:

1.5.1. Epidemiology:

About 2000 million people in the world today are infected with tuberculosis,(19) but only 10% develop clinical disease. In 2002 approximately 15,000 new cases of TB disease were diagnosed in the United States. Of these, 6% were among children aged <15 years (20). Although the number of cases in this age group has been decreasing since 1992, the number co infected with HIV is uncertain because only a limited number of U.S. children who have TB have been tested for HIV infection. Close correlation exists between the observed incidence of TBM in children aged 0-4 years, and the population's annual average risk of infection with M
tuberculosis. The incidence of TBM has been calculated to represent 1% of the annual risk of infection \(^{(21)}\). The total number of tuberculosis cases in the world is increasing \(^{(22)}\). It is estimated that most of these new cases will be in south east Asia \(^{(23)}\) fuelled by the rapid spread of HIV. It has been predicted that without intervention 200 million people alive today will develop TB \(^{(24)}\).

An estimated Annual Risk of infection (ARI) of 1.8% which gives an incidence of 90/100,000 smear positive cases puts Sudan among the high prevalence countries for TB in the East Mediterranean Region \(^{(25)}\).

The Sudan National Tuberculosis Programme (NTP) declared DOTS all over in January 2002. The years 2004, 2005 (of 70% case detection and 85% cure rate) \(^{(26)}\). Percentage of admission with TB from all inpatients was 1.0% in 2000, and 0.6% in 2004. In addition, the percentage of death due to the disease of admission: 11.2% in 2000, and 10.5% in 2004 \(^{(27)}\).

Mustafa Sid Ahmed studied the clinical pattern of intracranial space occupying lesion in adults during the period 1998-1999 in 118 patients in Sudan, and he found that tuberculoma represented (8.5%) \(^{(28)}\).
1.5.2. **Causative agent:**

Tuberculous meningitis was first described as a distinct pathological entity in 1836 and Robert Koch demonstrated that tuberculosis was caused by *Mycobacterium tuberculosis* in 1882\(^{(29)}\). *M tuberculosis* is an aerobic gram positive rod that stains poorly due to its thick cell wall containing lipids, peptidoglycans, and arabinomannans. The Ziehl-Neelsen stain uses the properties of the cell wall to form a complex that prevents decolourisation by acid or alcohol \(^{(30)}\).

1.5.3. **Pathogenesis:**

The development of TBM is a two step process \(^{(31)}\); *M tuberculosis* bacilli enter the host by droplet inhalation, the initial point of infection being the alveolar macrophage. Escalating localized infection within the lung with dissemination to the regional lymph nodes produces the primary complex. During this stage there is a short but significant bacteraemia that can seed tubercle bacilli to other organs in the body. In those who develop TBM bacilli seed to the meninges or brain parenchyma, forming small subpial or subependymal foci. These are called Rich foci, after the original pathological studies of Rich and McCordick\(^{(31)}\). In about 10% of cases, particularly in children, the primary complex does not heal but progresses. Tuberculous
pneumonia develops with heavier and more prolonged tuberculous bacteraemia. Dissemination to the CNS is more likely, particularly if miliary TB develops. The second step in the development of TBM is rupture of a Rich focus into the subarachnoid space. This heralds the onset of meningitis which if left untreated, will result in severe and irreversible neurological pathology. In 75% of children the onset of TBM is less than 12 months after the primary infection (32). A rare complication of TBM is tuberculous encephalopathy. Usually occurring in a young child with progressive primary TB, the presentation is of reducing conscious level with few focal signs and minimal meningism. Diffuse oedema and white matter pallor with demyelination are found pathologically. The pathogenesis is uncertain, but is presumed to be immune mediated (33).

1.5.4. Clinical Features:

Recent contact with tuberculosis should be elucidated: several studies have shown that between 70% and 90% of children have had recent contact with TB (34). The prodrome is usually non-specific with no one symptom predominating: 28% report headache, 25% were vomiting, and 13% had fever (35). Only 2% reported meningitic symptoms.

In a review of 205 children only 38% had fever at presentation with 9%
reporting photophobia \(^{35}\). 14% remained free from meningism throughout the illness. Recent reviews confirm the wide variety of presentations seen with TBM \(^{36}\).

An Australian series of 58 patients found that on the day of admission TBM was considered a diagnosis in 36% of cases, with 6% receiving immediate treatment \(^{34}\). The duration of presenting symptoms varied from 1 day to 9 months, although 55% presented with less than two weeks of symptoms.

Adhesions can result in cranial nerve palsies (particularly II, III, IV, VI, VII, and VIII), constriction of the internal carotid resulting in stroke, and obstruction of CSF flow leading to raised intracranial pressure, reduced conscious level, and hydrocephalus. Infarcts occur in about 30% of cases \(^{37}\) commonly in the internal capsule and basal ganglia, causing a range of disorders from hemiparesis to movement disorders. Seizures are common, especially in children and elderly people. Hydrocephalus, tuberculoma, oedema, and hyponatraemia due to inappropriate ADH secretion can all cause seizures \(^{38}\).

Over the past 10 years there have been studies documenting the relation between HIV and TBM. Although HIV infected patients with TB are at
increased risk of TBM, the clinical features and outcomes of the disease do not seem to be altered. Those with TBM and HIV often have concomitant extrameningeal disease. In one report 65% had clinical or radiographic evidence of extrameningeal TB on admission. In another series 77% of those with HIV had clinical evidence of extrameningeal TB compared with 9% in those without HIV. In more than half there may also be a CNS tuberculoma. These distinguishing characteristics may facilitate the diagnosis of TBM in those with HIV.

1.5.5. Diagnosis:

Newer methods such as those involving the amplification of bacterial DNA by the polymerase chain reaction (PCR) and comparable systems are incompletely assessed, and are not suitable for widespread use in the developing world. The careful and repeated search for acid fast bacilli with Ziehl-Neelsen staining is still one of the most effective rapid diagnostic tests. A history of recent TB contact is helpful as is the presence of extrameningeal TB. Tuberculin testing is of limited value. Early studies found 22% of those with TBM were negative to 100 units PPD. A recent study demonstrated cumulative reactivity with 10-100 units PPD to be 75%. Some studies suggest that tuberculin testing may be more useful in
children, with 86% having greater than 15 mm of induration with 5 units purified protein derivative (PPD) \(^{(36)}\). Abnormalities in the CSF depend on a tuberculin reaction within the subarachnoid space. Those with depressed cell mediated immunity may have atypical findings in the CSF. Acellular CSF in elderly and HIV positive patients have been reported\(^{(38)}\). Lymphocytosis of between 100 and 1000 cells/mm\(^3\) is more usual, although in the first 10 days polymorphonuclear leucocytes may predominate\(^{(44)}\). A raised CSF protein occurs in most, and CSF glucose will be reduced in 70\% \(^{(39)}\).

Both CT and MRI of the brain will disclose hydrocephalus, basilar meningeal thickening, infarcts, oedema, and tuberculomas. In a CT study of 60 cases of TBM in adults and children only three had normal brain scans\(^{(45)}\). Hydrocephalus was reported in 87\% of children and 12\% of adults. The incidence of hydrocephalus is greater in the young, and increases with duration of the illness. In children hydrocephalus is almost always present after 6 weeks of illness \(^{(46)}\). Infarcts are seen on CT in 28\%, with 83\% occurring in the middle cerebral artery territory. The basal ganglia are the most commonly affected region.

Both CT and MRI are sensitive to the changes of TBM, particularly hydrocephalus and basal meningeal exudates, but they lack specificity \(^{(46)}\).
The challenge facing new diagnostic strategies in TBM is that they must improve on the sensitivity of conventional Ziehl-Neelsen staining and culture, but maintain the specificity.

Adenosine deaminase is produced by lymphocytes and monocytes. Its detection in CSF has been reported with variable success, with sensitivities and specificities as high as 99% being suggested (47).

Serological techniques that detect the intrathecal synthesis of antimycobacterial antibodies have been studied (47).

The advent of DNA amplification techniques such as PCR has turned attention away from serological techniques.

1.6. Schistosomiasis:

Schistosomiasis associated with Schistosoma mansoni infection is endemic in the Caribbean islands, the Middle East, South America, and Africa and may be imported to any other area in the world via immigration and travel to foreign lands. It is estimated that between 200 and 300 million people are infected by S. mansoni worldwide (48). In Brazil, it is estimated that between 10 and 12 million people have schistosomiasis mansoni (49). Spread of the disease from rural to peri-urban regions has been recently
 described \(^{(49)}\). The infection is localized in the digestive system, but the nervous system is the second most common involved site \(^{(50)}\). Pittella\& Lana-Peixoto recorded that ova were found in the brain of 26\% of patients who have hepatosplenic S. mansoni infection. A study in Tanzania indicated that 6\% of non-traumatic myelopathies in endemic areas may be attributable to schistosomiasis \(^{(51)}\).

In Sudan: A study was conducted in Southern Sudan to determine the prevalence of intestinal parasites among school children. A total of 275 stool sample were examined. Hookworm with a prevalence of (13.1\%) was the predominant nematode followed by S. Mansoni (2.2\%) \(^{(52)}\).

Amir Eltayeb studied the prevalence of intestinal protozoa and parasites infestations among under five children in Jabel Aweleia Governorate in 390 patient in the period September 2003 to September 2004 and he found that schistosoma mansoni was found in 1 (0.3\%) from the rural area \(^{(53)}\).

A parasitological survey of refugees based in Juba, involving 241 faecal samples, revealed that S. mansoni is the most prevalence(52\%) among older teenagers, S. stercoralis shows (44\%) in the five to nine-year old group \(^{(54)}\).

1.6.1. Pathogenesis:

Neuroschistosomiasis (NS) occurs when ova and or adult worms reach the central nervous system (CNS). Two mechanisms have been postulated
for this process: the ova are carried to the CNS through arterial or retrograde venous blood flow via the valveless perivertebral plexus of Batson, being deposited anywhere along the path of the blood flow, or the ova are deposited in situ after the anomalous migration of adult worms\(^{(55)}\).

NS can be present in the early stages of the infection, while the patient is still asymptomatic, during the slow and gradual evolution of the disease to its chronic forms, or concomitantly with the chronic intestinal hepatointestinal or hepatosplenic forms \(^{(56)}\). In the early post infective stage especially in non-immune subjects, neurological symptoms may occasionally appear in the Katayama syndrome which is attributed to an immunological reaction to cercariae or schistosomula or ova \(^{(57)}\).

The granulomatous reaction of the host to the presence of the ova is the major immune response to the antigens released from the ova and is at a maximum intensity in the early stages of infection, leading to the formation of necrotic-exudative granulomas and the immune response declines over the course of the infection \(^{(58)}\).

1.6.2. Clinical syndromes:

The asymptomatic form of NS is more common than the symptomatic ones \(^{(59)}\).

Clinical manifestations of cerebral NS include seizures associated
with an increase in intra-cranial pressure and focal CNS signs, depending on the site of the cerebral lesion, caused by the masses produced by the granulomas\(^{60}\). Headache, papilloedema, visual abnormalities, speech disturbances, nystagmus and ataxia are common manifestations\(^{59}\). Duration of the symptoms varies from a few weeks to more than one year\(^ {61}\). Different form of presentation: acute encephalitis or encephalomyelitis together with or immediately after the systemic manifestations of the acute phase, which include fever, headache, malaise, anorexia, coughing, skin rash, diarrhoea and abdominal pain (Katayama syndrome)\(^ {59}\), patients may become confused, develop focal or generalized seizures or become stupors and visual impairment and papilloedema may occur; signs of encephalopathy such as hemiplegia and opisthotonus with extensor plantar responses or evidence of myelopathy such as ataxia, weakness of the legs, paraesthesiae, sensory loss and sphincter disturbances may also occur\(^ {62}\).

1.6.3. Diagnosis:

CSF in NS, which includes lymphomononuclear hypercellularity associated with the presence of eosinophils, an increase in protein concentration, and the presence of antibodies to S. mansoni. Habeebulla&Ross have recently reported a case of NS presenting with eosinophilic meningitis\(^ {63}\).
Serologic techniques have been used but none have yet achieved sufficient levels of sensitivity and specificity to justify their consideration as gold standard techniques (64).

CT/MRI findings of cerebral schistosomiasis have been described as single or multiple hyperdense lesions with variable enhancement surrounded by low-density oedema with an associated mass effect (65), that is a punctuate enhancement and a heterogeneous internal structure which correlates with a moderately large granuloma due to the presence of a collection of eggs (66). Definitive diagnosis of NS is based on the demonstration of eggs and or adult worms in the CNS tissue (67).

1.7. Arachnoid cysts:

Arachnoid cysts are non-tumorous intra-arachnoid fluid collections that account for about 1% of all intracranial space-occupying lesions (68). They may develop throughout the cerebrospinal axis, with a predominance in the sylvian region (69). Because of their benign nature and slow expansion arachnoid cysts may remain a symptomatic or produce only subtle symptoms and signs, sometimes give rise to focal neurological deficits, raised intracranial pressure, and/or epileptic seizures.

The arachnoid cyst wall is histologically indistinguishable from normal
arachnoid membrane, moderate thickening of the arachnoid and increase in connective tissue is common⁷⁰.

In about 15% of middle fossa arachnoid cysts, an asymptomatic lesion may become symptomatic as a result of bleeding in association of the cyst and raised intracranial pressure. Treatment is by cystoperitoneal shunt after evacuation⁷¹.

1.8. Dermoid and epidermoid cysts:

Dermoid and epidermoid cysts are rare space-occupying lesions of the central nervous system. Although characterized by a slow growth rate, they are often associated with serious complications. Surgery is the only effective treatment, and radical resection of the entire cyst, whenever possible generally succeeds in achieving a cure⁷². In dermoid cysts occurring during adulthood, symptoms and signs more clearly indicate a dysfunction of the posterior fossa. It is particularly important to establish the presence and type of communication of cysts with the CSF pathways. Although infratentorial cysts often communicate, they can be space-occupying masses because of increasing CSF retention, which may be due to a ball-valve mechanism or to inadequate communication. The frequently associated hydrocephalus, can be due to mechanical factors⁷³.
1.9. Toxoplasmosis:

Toxoplasma gondii is a protozoon that commonly affects mammals and birds throughout the world. Toxoplasma gondii infection in humans is usually asymptomatic.

1.9.1. Epidemiology:

The major mode of transmission of Toxoplasma gondii infection among infants and young children is congenital. The incidence of congenital toxoplasmosis in the United States is an estimated one case per 1,000--12,000 live-born infants \(^{(74)}\) and is believed to have decreased substantially during the preceding 20 years. Older children, adolescents, and adults typically acquire Toxoplasma infection by eating poorly cooked meat that contains parasitic cysts or by ingesting sporulated oocysts in soil or contaminated food or water.

1.9.2. Clinical Manifestations:

In studies of non immunocompromised infants with congenital toxoplasmosis, the majority of infants (70%-90%) are asymptomatic at birth; however, the majority of asymptomatic children develop late sequelae (e.g., retinitis, visual impairment, and intellectual or neurologic impairment) with the interval until the onset of their symptoms ranging from several months to years. Symptoms can include maculopapular rash, generalized
lymphadenopathy, hepatosplenomegaly, jaundice, hematologic abnormalities including anemia, thrombocytopenia and neutropenia, and substantial CNS disease, including hydrocephalus, intracerebral calcification, microcephaly, chorioretinitis, and seizures\textsuperscript{(75)}.

1.9.3. Diagnosis:

Serologic testing is the major method of diagnosis, isolation of organisms, and PCR.

A presumptive diagnosis of CNS toxoplasmosis is based on clinical symptoms, serologic evidence of infection, and the presence of a space-occupying lesion on imaging studies of the brain. Cases of Toxoplasma encephalitis have been reported in persons without Toxoplasma-specific IgG antibodies; therefore, negative serology does not exclude that diagnosis. Computerized tomography of the brain might indicate multiple, bilateral ring-enhancing lesions in CNS toxoplasmosis, especially in the basal ganglia and cerebral corticomedullary junction \textsuperscript{(76)}. Magnetic resonance imaging is more sensitive and will confirm basal ganglia lesions in the majority of patients. F-fluoro-2-deoxyglucose-positive emission tomography can be helpful in distinguishing toxoplasma abscesses from primary CNS lymphoma, but the accuracy is not high and this test is not widely available\textsuperscript{(77)}. 

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1.10. **Hydatid disease:**

Hydatid disease is caused by larvae of cestodes of the genus *Echinococcus*, the adult of which is found in carnivores, which are the definitive hosts. The intermediate hosts are infected by swallowing eggs passed in the faeces of the definitive host. Two main forms of hydatid disease occur in man (78).

1.10.1. **Geographical distribution:**

The most extensive and endemic areas of human infection are found in the sheep raising countries; South Australia, New Zealand, Tasmania, parts of North, South and East Africa the southern half of the south America. In addition human infection is frequently found in south-west states of the USA, Southern and Eastern Europe, Iraq, Syria, Lebanon, Turkey, Mongolia, Turkistan, North China, Southern Japan and North Vietnam (79).

1.10.2. **Incidence:**

Amongst 1802 patient in Australian hydatid register, hydatid disease of the brain was found in 1% (80). According to another source it occurs in 2% of patients with hydatid disease (12). In reported series of hydatidosis, the chance of brain hydatid cyst has been between 1 to 3% (81), and it comprises about 0.02-2% of space occupying lesions of the brain (82). The human brain
can be involved primarily via the haematogenous route or by metastatic spread when a cyst ruptures in the heart or lung. The majority of the hydatid cysts in the brain are single (83). About 50-75% of intracranial hydatid cysts are seen in children (84). This high incidence in children is probably related to patent ductus arteriosus (85). Most cysts are supratentorial (87). Infratentorial HC is very rare (86). The other less common sites reported are skull cavernous sinus eyeball, pons, extradural, cerebellum and ventricles (87).

The hydatid cyst has a wall composed of two layers: an inner layer of germinal epithelium (endocyst), and an outer layer of laminated hyaline membrane (ectocyst), the ectocyst is striated and nonnucleated, and the endocyst is granular, nucleated, and friable. In most parts of the body, the host reacts to the presence of this alien organism by enveloping it in a fibroblastic capsule (adventitial membrane), but in the brain this membrane hardly develops (88). The fluid in the cyst is colourless and has a low specific gravity (1005-1015). The albumin content is 2 to 2.5 g/L, the glucose content is 0.30 to 0.50 g/L, and the chloride content is 6.49 g/L. Some lymphocytes, scolices, and hooks are also present in each milliliter of the fluid (89).
1.10.3. Clinical Features:

Generally are those of space occupying lesion and hydatid involvement of the brain is marked by slow mass effect, hydrocephalus and often seizures, occasionally metastatic lesions in the brain are the first to cause symptoms by local inflammation or mass effect\(^{(90)}\).

1.10.4. Diagnosis:

Thirty percent may show eosinophilia. Confirmatory evidence of infection may be obtained by serology.

The appearances of hydatid cysts in CT scan are cystic, spherical with a sharp border, a central absorptive value similar to CSF, and no perifocal oedema and usually with significant ventricular distortion and a shift of midline structures. There is lack of enhancement and of the perifocal oedema seen in cystic tumours\(^{(90)}\).

1.11. Mycetoma:

Mycetoma is a chronic, localized subcutaneous infection characterized by draining sinus tracts, that containing granule. The disease most often affects the lower extremities with the majority of cases involving the foot. The organisms are usually introduced by a thorn. Rare sites such as
eye lids, testis, mandible, paranasal sinus, head and neck and spine has also been described. Neurological deficits are quite rare and less well described\(^{(16)}\).

In the study conducted by Arbab, Idris, Sokrab et al, in nine cases comprising seven males and two females, they mentioned that the commonest causative organism was streptomyces somaliensis(66.7%). Males were affected more often than females(22.2%) and the source of infection was unknown in the majority of cases and only known in (33.3%)of cases. The most common mode of presentation was headache and scalp swelling (88.9%) the next common presentation was epilepsy (55.6%). Other focal neurological disorders such as hemiplegia and cranial nerve affection were also found. CT findings of the cranium showed osteosclerotic rather than osteolytic changes. They concluded that mycetoma of the cranium may present with various neurological disorders and may stimulate cerebral neoplasm or pseudotumour cerebri\(^{(91)}\).

1.12. Aneurysms and arteriovenous malformations:

Aneurysm: Is widening of a vessel involving the stretching of fibrous tissue within the media of the vessel \(^{(16)}\). These can be classified
morphologically into saccular, fusiform or mycotic. The pathogenesis of saccular aneurysms reflects a combination of congenital, acquired and hereditary factors. A modest increase in incidence of familial saccular aneurysms as well as their association with polycystic kidney disease, Ehler Danlos Syndrome and other connective tissue disorders implicate hereditary factors\(^{(16)}\).

Fusiform aneurysms: spindle shape dilatation and elongation that occur in large arteries, most frequently in the basilar artery, which can compress cranial nerves v, vii, viii causing facial pain hemifacial spasm and hearing loss with vertigo respectively. Fusiform aneurysms may imitate the features of CPA angle tumours, or they mimic pituitary and suprasellar mass lesions.

Mycotic Aneurysms: Caused by septic degeneration of arterial wall muscle and elastic tissue. They form in distal cerebral arteries at the point where small septic cardiogenic emboli lodge. They are frequently multiple and can be found in anterior or posterior cerebral circulation

1.12.1. **Clinical Features:**

A part from those related to subarachnoid haemorrhage due to rupture of aneurysm, it may present with features of space occupying lesion.

Bien SM, Thorn K and Hassler W, mentioned that CT diagnosis of
cerebral aneurysm is possible. In nonthrombosed giant aneurysm, CT shows a homogenous, primarily hyperdense space occupying lesion with enhancement. The partially thrombosed giant aneurysms appear hyperdense with hypodense or isodense portion in the plain CT scan. CT scan diagnosis is possible in every case of partially or non thrombosed aneurysm, but cerebral angiography remains the definitive study to detect the lesion.  

1.12.2. Vascular malformation:  

Venous angioma: The most common type, usually lie close to the surface of the brain, seldom produce seizures or headache.

Acerebral varix: is a single dilated vein and very rarely causes clinical symptoms.

Telangetasia: Are common, they are usually located deep in the brain and rarely produce symptoms. Because of their location, haemorrhage from small vessels can occasionally be fetal.

Cavernous angiomas: Are large sinusoidal channels which can be thrombosed. They are readily detected by CT scan and rarely bleed, but they may cause headache and seizures.  

Arteriovenous malformation: The most common symptomatic vascular anomaly. Familial causes are rare, indicating that the problem reflects sporadic abnormalities in embryologic development.
1.12.3. Diagnosis:

By CT scan and MRI. Angiography remains the definitive test to identify the AVM and delineate its feeding arteries and draining veins\textsuperscript{(16)}. 
JUSTIFICATIONS

• Intracranial SOLs is a serious problem in any children.

• Early detection of symptoms and signs of intracranial SOLs can give better chance for treatment.

• No similar study was done in children in Sudan.
The study aims to study the:

1. clinical presentations of intracranial SOLs.
2. causes of intracranial SOLs in children.
3. imaging findings associated with intracranial SOLs.
2. MATERIALS AND METHODS

2.1. Study design:

It is a combined cross sectional and prospective hospital based study.

2.2. Study Area:

The study was conducted in Elshaab Teaching Hospital(National Center For Neurological Sciences), Khartoum Teaching Hospital and Dr Gafaar Ibn Auf Children's Hospital.

2.3. Study Duration:

The study was done during the period from 1st September 2005 to end of February 2007.

2.4. Study Population and Sampling Technique:

2.4.1. Study Population:

The study population included 103 children less than 18 years of age, 63 males and 40 were females.

2.4.2. Sampling technique:

The sample is an inclusive sample with no selection. All children who diagnosed as having intracranial SOLs after confirmation by brain CT scan or MRI admitted or seen in referral clinics in Elshaab Teaching Hospital
(National Center For Neurological Sciences) in Mondays and Thursdays, or admitted in the selected hospitals during the study period were included.

2.5. Inclusion criteria:

All children presenting with intracranial SOLs below 18 years old and new cases in the selected hospitals.

2.6. Exclusion criteria:

- Refusal to give consent for inclusion in the study by the parents or caretakers.
- Patients with intracranial haemorrhage and or subdural haematoma were not included (because most of these cases are due to trauma).

2.7. Methods

2.7.1. Study Tools and Technique:

a: Questionnaire:

A standardized questionnaire was used to obtain information concerning personal history of the child, detailed history about the presenting complaint, symptoms of raised intracranial pressure, mental symptoms such as coma, sensory and motor symptoms, symptoms related to the cranial nerves. Past medical history of chronic cough, trauma to the head, schistosomaisis and epilepsy, family history and socioeconomic information.
b: Clinical examination:

Every child was subjected to a thorough clinical examination including a general check up for pallor, cyanosis, jaundice; chest was examined for evidence of congenital heart disease, also abdominal examination for organomegaly, generalized lymphadenopathy and proper C.N.S examination including fundoscopy, some difficult cases were referred to the ophthalmologist.

All children had anthropometric measurements including skull circumference, height and weight.

2.7.2. Investigations:

Routine investigations:

CBC, ESR, urea and electrolytes, x-ray of the skull, x-rays of other parts of the body are ordered if secondaries were suspected.

Investigations essential for diagnosis of intracranial SOL:

All patients had an MRI or brain CT scan to confirm the presence of SOL.

Specific investigations:

Mantoux test, sputum for AAFB, chest x-ray, abdominal U/S and
pituitary hormones were done in selected patients.

**Other investigations:**

Biopsy- histological confirmation of the lesion was obtained in those who had access to surgery.

**2.8. Data Analysis:**

The questionnaire was pre-coded, and a master sheet was constructed to arrange the raw data. Tables were drawn and descriptive statistics were measured.

Data was entered into SPSS (statistical package for social science) computerized program for analysis, and Chi-square test was then used to 5% confidence level (P value $\leq 0.05$).

**2.9. Ethical Issues:**

- Permission was obtain from the relevant hospitals administrations.
- Verbal consent was taken from the child, his parents or caretakers.

**2.10. Research Team:**

- The author, who performed the clinical examination, fulfilled the questionnaire and helped some patients to perform unaffordable investigations like brain CTscan and MRI.
• Ophthalmologist who performed fundus examination for the difficult cases.
• Lab technician
3. RESULTS

3.1. Demographic characteristic of children in the study population:

3.1.1. Sex Distribution:

The study included 103 patients; Males were 63 and females were 40 patients. Male to female ratio was 1.7: 1. (Figure 1)

3.1.2. Age Distribution:

Thirty five patients (34%) were less than 5 years of age, 32 (31%) patients were 5-10 years and 36 (35%) patients were more than 10 years. (Figure 2)

3.2. Pattern of presentation:

Figure 3 shows that the commonest presenting symptoms were headache in 61 (59.2%) patients, seizures in 51 (49.5%) patients, vomiting in 48 (46.6%) patients and motor weakness in 40 patients (38.8%). The least presenting symptoms were paraesthesias in 2 (1.9%) patients, sensory loss in 1 (1.0%) patient and dysphagia in 1 (1.0%) patient.
Figure 1: The distribution of gender for the study population

n=103

Female 39%

Male 61%
Figure 2: The distribution of age for the study population

n=103

>10yr 35%

<5yr 34%

5-10 yr 31%
Figure 3: The presenting symptoms in the study population

n=103

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>headache</td>
<td>52.9%</td>
</tr>
<tr>
<td>seizures</td>
<td>5.8%</td>
</tr>
<tr>
<td>vomiting</td>
<td>4.9%</td>
</tr>
<tr>
<td>motor weakness</td>
<td>4.8%</td>
</tr>
<tr>
<td>visual impairment</td>
<td>3.9%</td>
</tr>
<tr>
<td>increase size of head</td>
<td>3.8%</td>
</tr>
<tr>
<td>gait disturbance</td>
<td>2.1%</td>
</tr>
<tr>
<td>loss of consciousness</td>
<td>1.8%</td>
</tr>
<tr>
<td>incontinence</td>
<td>1.8%</td>
</tr>
<tr>
<td>speech disturbance</td>
<td>1.2%</td>
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<tr>
<td>deviation of mouth</td>
<td>0.8%</td>
</tr>
<tr>
<td>mental symptoms</td>
<td>0.8%</td>
</tr>
<tr>
<td>deafness</td>
<td>0.7%</td>
</tr>
<tr>
<td>paresthesias</td>
<td>0.5%</td>
</tr>
<tr>
<td>sensor/case</td>
<td>0.5%</td>
</tr>
<tr>
<td>dysphasia</td>
<td>0.5%</td>
</tr>
</tbody>
</table>
3.3. Clinical examination:

*Figure 4* shows that abnormal gait was detected in 44 (42.7%) patients.

Cranial nerves involvement in 45 (43.7%) patients. The commonest affected cranial nerve was the optic cranial nerve; this was occurred in 18 (17.5%) patients mainly with astrocytoma in 11 (61.1%) patients. *(Table 1)*

Fundus examination was abnormal in 32 (31.1%) patients; optic atrophy in 9 (8.7%) patients, papilloedema in 19 (18.5%) patients and both optic atrophy and papilloedema in 4 (3.9%) patients and difficult to assess in 10 (9.7%) patient. *(Table 2)*

3.4. Sites of lesions:

All patients had brain MRI or brain CT scan, the supratentorial lesions were found in 75 (72.8%) patients and infratentorial lesions were found in 28 (27.2%) patients. *(Figure 5)*

3.4.1. Sites of lesions in relation to sex:

*Table 3* shows that supratentorial lesions occurred in 50 (66.7%) males compared to 25 (33.3%) females. The rest of patients lesions were in infratentorial area.
Figure 4: The clinical signs in patients in the study population
n=103

- Abnormal gait: 42.7%
- Cranial nerves lesions: 43.7%
- Abnormal fundus: 31.1%
- Pallor: 23.3%
- Wasting: 26.2%
- Hyperreflexia: 27.2%
- Hypotonia: 16.5%
- Hypertonia: 13.7%
Table 1: Individual cranial nerves involvement in children with intracranial SOLs in the study population

<table>
<thead>
<tr>
<th>Lesion</th>
<th>1&lt;sup&gt;st&lt;/sup&gt;</th>
<th>2&lt;sup&gt;nd&lt;/sup&gt;</th>
<th>3&lt;sup&gt;rd&lt;/sup&gt;</th>
<th>4&lt;sup&gt;th&lt;/sup&gt;</th>
<th>5&lt;sup&gt;th&lt;/sup&gt;</th>
<th>6&lt;sup&gt;th&lt;/sup&gt;</th>
<th>7&lt;sup&gt;th&lt;/sup&gt;</th>
<th>8&lt;sup&gt;th&lt;/sup&gt;</th>
<th>9&lt;sup&gt;th&lt;/sup&gt;</th>
<th>10&lt;sup&gt;th&lt;/sup&gt;</th>
<th>11&lt;sup&gt;th&lt;/sup&gt;</th>
<th>12&lt;sup&gt;th&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asrtocytoma</td>
<td>11</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abscess</td>
<td>2</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>6</td>
</tr>
<tr>
<td>Medulloblastoma</td>
<td>1</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Others</td>
<td>1</td>
<td>4</td>
<td>4</td>
<td>1</td>
<td>3</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>1</td>
<td>18</td>
<td>8</td>
<td>1</td>
<td>10</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 2: Fundal changes in children with intracranial SOLs in the study population

<table>
<thead>
<tr>
<th>Fundal change</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Papilloedema</td>
<td>19</td>
<td>18.5</td>
</tr>
<tr>
<td>Optic atrophy</td>
<td>9</td>
<td>8.7</td>
</tr>
<tr>
<td>Optic &amp; papilloedema</td>
<td>4</td>
<td>3.9</td>
</tr>
<tr>
<td>Normal</td>
<td>61</td>
<td>59.2</td>
</tr>
<tr>
<td>Difficult to assess</td>
<td>10</td>
<td>9.7</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>103</td>
<td>100.0</td>
</tr>
</tbody>
</table>
Figure 5: Supratentorial and infratentorial classification for the lesions in the study population

n=103

- Supratentorial: 73%
- Infratentorial: 27%
<table>
<thead>
<tr>
<th>Gender</th>
<th>Supratentorial</th>
<th>Infratentorial</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No</td>
<td>%</td>
<td>No</td>
</tr>
<tr>
<td>Male</td>
<td>50</td>
<td>66.7</td>
<td>13</td>
</tr>
<tr>
<td>Female</td>
<td>25</td>
<td>33.3</td>
<td>15</td>
</tr>
<tr>
<td>Total</td>
<td>75</td>
<td>100.0</td>
<td>28</td>
</tr>
</tbody>
</table>

P=0.06
The commonest site of the lesion was the whole cerebellum in 11 (10.7%) patients, the right cerebellum in 6 (5.8%) patients and in the left cerebellum is one (1.0%) patient. The lesion was detected in the occipital lobe in 9 (8.7%) patients. The least sites affected were occipito temporal lobes in one (1%) patient and occipito cerebellar area in one patient (1.0%). (Figure 6)

3.4.2. Sites of lesions in relation to age group:

The commonest site for all age group was the supratentorial area. <5 years 28 (37.3%) patients, 5-10 years 23 (30.7%) patients and > 10 years in 24 (32.0%) patients, this result was statistically insignificant (p=0.4). (Table 4)

3.4. Sites of lesions in relation to the commonest symptoms and signs:

3.4.3.1. Headache:

_Table (5)_ shows that headache was a common symptom with the infratentorial lesions detected in 21 (75.0%) patients compared to 40 (53.3%) patients with supratentorial lesion, this result was statistically significant (p=0.04).
Figure 6: Sites for lesions in the study population
n=103
### Table 4: Sites of lesions (supratentorial and infratentorial) in relation to age in the study population

<table>
<thead>
<tr>
<th>Gender</th>
<th>Supratentorial</th>
<th>Infratentorial</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No</td>
<td>%</td>
<td>No</td>
</tr>
<tr>
<td>&lt;5 yr</td>
<td>28</td>
<td>37.3</td>
<td>7</td>
</tr>
<tr>
<td>5-10 yr</td>
<td>23</td>
<td>30.7</td>
<td>9</td>
</tr>
<tr>
<td>&gt;10 yr</td>
<td>24</td>
<td>32.0</td>
<td>12</td>
</tr>
<tr>
<td>Total</td>
<td>75</td>
<td>100.0</td>
<td>28</td>
</tr>
</tbody>
</table>

P=0.4

### Table 5: Headache in relation to sites of lesions in the study population

<table>
<thead>
<tr>
<th>Headache</th>
<th>Supratentorial</th>
<th>Infratentorial</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No</td>
<td>%</td>
<td>No</td>
</tr>
<tr>
<td>Present</td>
<td>40</td>
<td>53.3</td>
<td>21</td>
</tr>
<tr>
<td>Absent</td>
<td>35</td>
<td>46.7</td>
<td>7</td>
</tr>
<tr>
<td>Total</td>
<td>75</td>
<td>100.0</td>
<td>28</td>
</tr>
</tbody>
</table>

P=0.04
3.4.3.2. Vomiting:

No significant difference in the symptom of vomiting in relation to the site (p=0.6). *(Table 6)*

3.4.3.3. Seizures:

Forty four (58.7%) patients with supratentorial lesions and 7 (25.0%) patients with infratentorial lesions presented with seizures, this relation with the sites was statistically significant (p=0.002). *(Table 7)*

3.4.3.4. Motor weakness:

Twenty six (34.7%) patients from the supratentorial area and 14 (50.0%) patients from infratentorial area, presented with motor weakness, the result was statistically significant (p=0.00). *(Table 8)*

3.4.3.5. Gait disturbance:

Gait disturbance was found to be more common with the infratentorial lesions than supratentorial lesions, 10 (35.7%) patients compared to 9 (12.0%) patients presented with gait disturbance, this relation was statistically significant (p=0.006). *(Table 9)*

3.4.3.6. Fundal changes:

Papilloedema was the commonest lesion, found mainly in the supratentorial lesions in 12 (63.2%) patients compared to 7 (36.8%) patients
Table 6: Vomiting in relation to sites of lesions in the study population

<table>
<thead>
<tr>
<th>Vomiting</th>
<th>Supratentoria</th>
<th>Infratentorial</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No</td>
<td>%</td>
<td>No</td>
</tr>
<tr>
<td>Present</td>
<td>36</td>
<td>48.0</td>
<td>12</td>
</tr>
<tr>
<td>Absent</td>
<td>39</td>
<td>52.0</td>
<td>16</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>75</td>
<td>100.0</td>
<td>28</td>
</tr>
</tbody>
</table>

P=0.6

Table 7: Seizures in relation to sites of lesions in the study population

<table>
<thead>
<tr>
<th>Seizures</th>
<th>Supratentoria</th>
<th>Infratentorial</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No</td>
<td>%</td>
<td>No</td>
</tr>
<tr>
<td>Present</td>
<td>44</td>
<td>58.7</td>
<td>7</td>
</tr>
<tr>
<td>Absent</td>
<td>31</td>
<td>41.3</td>
<td>21</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>75</td>
<td>100.0</td>
<td>28</td>
</tr>
</tbody>
</table>

P=0.002
### Table 8: Motor weakness in relation to sites of lesions in the study population

<table>
<thead>
<tr>
<th>Motor weakness</th>
<th>Supratentorial</th>
<th>Infratentorial</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No</td>
<td>%</td>
<td>No</td>
</tr>
<tr>
<td>Present</td>
<td>26</td>
<td>34.7</td>
<td>14</td>
</tr>
<tr>
<td>Absent</td>
<td>49</td>
<td>65.3</td>
<td>14</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>75</td>
<td>100.0</td>
<td>28</td>
</tr>
</tbody>
</table>

*P=0.00*

### Table 9: Gait disturbance in relation to sites of lesions in the study population

<table>
<thead>
<tr>
<th>Gait</th>
<th>Supratentorial</th>
<th>Infratentorial</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No</td>
<td>%</td>
<td>No</td>
</tr>
<tr>
<td>Normal</td>
<td>66</td>
<td>88.0</td>
<td>18</td>
</tr>
<tr>
<td>abnormal</td>
<td>9</td>
<td>12.0</td>
<td>10</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>75</td>
<td>100.0</td>
<td>28</td>
</tr>
</tbody>
</table>

*P=0.006*
with infratentorial lesions, this result was statistically insignificant (p=0.7). *(Table 10)*

### 3.5. Nature of lesions:

The nature of lesions was found to be neoplastic in 65 (63.1%) patients and non neoplastic in 38(36.9%) patients. *(Figure 7)*

Malignant lesions (which were the malignant tumours) were 56 (54.4%) lesions and benign lesions (which were tuberculoma, cysts and benign tumours) were 47(45.6%) lesions. *(Figure 8)*

The commonest histological lesions were astrocytoma in 36 (35.0%) patients, followed by brain abscess in 20 (19.4%) patients and Medulloblastoma was detected in 11 (10.7%) patients.

Tuberculoma was detected, in 2 (1.9%) patients only. The least lesions were found are Adenoma in 1 (1.0%) patient, germ cell papilloma in one (1.0%) patient and epidermoid cyst in 1 (1.0%) patient. *(Figure 9)*

### 3.5.1. Types of lesions (benign and malignant) in relation to Sex:

The malignant lesions commonly affected males 34 (60.7%) patients compared to 22 (39.3%) females, the result was statistically insignificant (p=0.9). *(Table 11)*
Table 10: Fundal changes in relation to sites of lesions in the study population

<table>
<thead>
<tr>
<th>Fundal finding</th>
<th>Supratentorial</th>
<th>Infratentorial</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No</td>
<td>%</td>
<td>No</td>
</tr>
<tr>
<td>Normal</td>
<td>47</td>
<td>77.0</td>
<td>14</td>
</tr>
<tr>
<td>Atrophy</td>
<td>6</td>
<td>66.7</td>
<td>3</td>
</tr>
<tr>
<td>Papilloedema</td>
<td>12</td>
<td>63.2</td>
<td>7</td>
</tr>
<tr>
<td>Atrophy &amp; papilloedema</td>
<td>3</td>
<td>75.0</td>
<td>1</td>
</tr>
<tr>
<td>Difficult to assess</td>
<td>7</td>
<td>70.0</td>
<td>3</td>
</tr>
<tr>
<td>Total</td>
<td>75</td>
<td>72.8</td>
<td>28</td>
</tr>
</tbody>
</table>

P=0.7
Figure 7: Natures of the lesions in the study population

n=103

neoplastic 63%

non neoplastic 37%
Figure 8: Classification of the lesions into benign and malignant in the study population

n=103

benign 46%
malignant 54%
Figure 9: Histological types of lesions in the study population
n=103

Histological type

Astrocytoma
Glioblastoma
Anaplastic astrocytoma
Medulloblastoma
Cranial Neurinoma
Germ cell tumor
Ependymoma
Liposarcoma
Hydatid
Glioma
Papilloma
Adenoma
Germ cell tumor
Epidermoid

Percentage
Table 11: Types of lesions in relation to sex in the study population

<table>
<thead>
<tr>
<th>Type</th>
<th>Benign</th>
<th>Malignant</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No</td>
<td>%</td>
<td>No</td>
</tr>
<tr>
<td>Male</td>
<td>29</td>
<td>61.7</td>
<td>34</td>
</tr>
<tr>
<td>Female</td>
<td>18</td>
<td>38.3</td>
<td>22</td>
</tr>
<tr>
<td>Total</td>
<td>47</td>
<td>100.0</td>
<td>56</td>
</tr>
</tbody>
</table>

$P=0.9$
3.5.2. Types of lesions (benign and malignant) in relation to age

Group:

Table 12 shows that the benign lesions were common among children < 5 years: 24 patients (51.1%), the malignant conditions were more common among children >10 years (42.9%), this was statistically significant (p=0.004).

3.5.3. Types of lesions in relation to sites:

Table 13 shows that supratentorial lesions were benign in 41 (54.7%) patients while infratentorial lesions were malignant in 22 (78.6%) patients, this relation was statistically significant (p=0.003).

3.5.4. Types of lesions in relation to presenting symptoms:

3.5.4.1. Headache:

Table 14 shows that headache was detected in 42 (75.0%) patients with malignant lesions compared to 19 patients (40.0%) with benign lesions, this relation was statistically significant (p=0.00).

3.5.4.2. Vomiting:

Vomiting was a presenting symptom in 23 (48.9%) patients with benign lesions compared to 25 (44.6%) patients with malignant lesions; this result was not significant statistically. (Table 15)
Table 12: Types of lesions (benign and malignant) in relation to age in the study population

<table>
<thead>
<tr>
<th>Age group</th>
<th>Supratentorial</th>
<th></th>
<th>Infratentorial</th>
<th></th>
<th>Total</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No</td>
<td>%</td>
<td>No</td>
<td>%</td>
<td>No</td>
<td>%</td>
</tr>
<tr>
<td>&lt;5 yr</td>
<td>24</td>
<td>51.1</td>
<td>11</td>
<td>14.6</td>
<td>35</td>
<td>34.0</td>
</tr>
<tr>
<td>5-10 yr</td>
<td>11</td>
<td>23.4</td>
<td>21</td>
<td>37.5</td>
<td>32</td>
<td>31.0</td>
</tr>
<tr>
<td>&gt;10 yr</td>
<td>12</td>
<td>25.5</td>
<td>24</td>
<td>42.9</td>
<td>36</td>
<td>35.0</td>
</tr>
<tr>
<td>Total</td>
<td>47</td>
<td>100.0</td>
<td>56</td>
<td>100.0</td>
<td>103</td>
<td>100.0</td>
</tr>
</tbody>
</table>

P=0.004
### Table 13: Types of lesions in relation to sites in the study population

<table>
<thead>
<tr>
<th>Type of lesion</th>
<th>Supratentorial</th>
<th>Infratentorial</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No</td>
<td>%</td>
<td>No</td>
</tr>
<tr>
<td>Benign</td>
<td>41</td>
<td>54.7</td>
<td>6</td>
</tr>
<tr>
<td>Malignant</td>
<td>34</td>
<td>45.3</td>
<td>22</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>75</td>
<td>100.0</td>
<td>28</td>
</tr>
</tbody>
</table>

P=0.003

### Table 14: Headache in relation to types of lesions in the study population

<table>
<thead>
<tr>
<th>Headache</th>
<th>Benign</th>
<th>Malignant</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No</td>
<td>%</td>
<td>No</td>
</tr>
<tr>
<td>Present</td>
<td>19</td>
<td>40.4</td>
<td>42</td>
</tr>
<tr>
<td>Absent</td>
<td>28</td>
<td>59.6</td>
<td>14</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>47</td>
<td>100.0</td>
<td>56</td>
</tr>
</tbody>
</table>

P=0.00
Table 15: Vomiting in relation to types of lesions in the study population

<table>
<thead>
<tr>
<th>Vomiting</th>
<th>Benign</th>
<th></th>
<th>Malignant</th>
<th></th>
<th>Total</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No</td>
<td>%</td>
<td>No</td>
<td>%</td>
<td>No</td>
<td>%</td>
</tr>
<tr>
<td>Present</td>
<td>23</td>
<td>48.9</td>
<td>25</td>
<td>44.6</td>
<td>48</td>
<td>46.6</td>
</tr>
<tr>
<td>Absent</td>
<td>24</td>
<td>51.1</td>
<td>31</td>
<td>55.4</td>
<td>55</td>
<td>53.4</td>
</tr>
<tr>
<td>Total</td>
<td>47</td>
<td>100.0</td>
<td>56</td>
<td>100.0</td>
<td>103</td>
<td>100.0</td>
</tr>
</tbody>
</table>

P=0.6
3.5.4.3. Seizures:

Seizures were detected in 31 (66.0%) patients with benign lesions and in 20 (35.7%) patients with malignant lesions, this was statistically significant (p=0.002). (*Table16*)

3.5.4.4. Motor weakness:

*Table 17* shows that 25 (44.6%) patients with malignant lesions presented with motor weakness compared to 15 (31.9%) patients with benign lesions presented with motor weakness, the result was statistically insignificant (p=0.18).

3.5.4.5. Gait disturbance:

*Table 18* shows that 7 (14.9%) patients with benign lesions presented with abnormal gait compared to 12 (21.4%) patients with malignant lesions, this was insignificant statistically (p=0.4).

3.5.4.6. Fundal changes:

Papilloedema was the commonest finding in fundus examination, found mainly in the malignant lesions in 11 (57.9%) patients compared to 8 (42.1%) patients with benign lesions, this was statistically insignificant (p=0.06). (*Table19*)
Table 16: Seizures in relation to types of lesions in the study population

<table>
<thead>
<tr>
<th>Seizures</th>
<th>Benign</th>
<th>Malignant</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No %</td>
<td>No %</td>
<td>No %</td>
</tr>
<tr>
<td>Present</td>
<td>31 66.0</td>
<td>20 35.7</td>
<td>51 49.5</td>
</tr>
<tr>
<td>Absent</td>
<td>16 34.0</td>
<td>36 64.3</td>
<td>52 50.5</td>
</tr>
<tr>
<td>Total</td>
<td>47 100.0</td>
<td>56 100.0</td>
<td>103 100.0</td>
</tr>
</tbody>
</table>

P=0.002

Table 17: Motor weakness in relation to types of lesions in the study population

<table>
<thead>
<tr>
<th>Motor weakness</th>
<th>Benign</th>
<th>Malignant</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No %</td>
<td>No %</td>
<td>No %</td>
</tr>
<tr>
<td>Present</td>
<td>15 31.9</td>
<td>25 44.6</td>
<td>40 38.8</td>
</tr>
<tr>
<td>Absent</td>
<td>32 68.1</td>
<td>31 55.4</td>
<td>63 61.2</td>
</tr>
<tr>
<td>Total</td>
<td>47 100.0</td>
<td>56 100.0</td>
<td>103 100.0</td>
</tr>
</tbody>
</table>

P=0.18
Table 18: Gait disturbance in relation to types of lesions in the study population

<table>
<thead>
<tr>
<th>Gait</th>
<th>Benign</th>
<th></th>
<th>Malignant</th>
<th></th>
<th>Total</th>
<th></th>
</tr>
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<tbody>
<tr>
<td></td>
<td>No</td>
<td>%</td>
<td>No</td>
<td>%</td>
<td>No</td>
<td>%</td>
</tr>
<tr>
<td>Normal</td>
<td>40</td>
<td>85.1</td>
<td>44</td>
<td>78.6</td>
<td>84</td>
<td>81.6</td>
</tr>
<tr>
<td>Abnormal</td>
<td>7</td>
<td>14.9</td>
<td>12</td>
<td>21.4</td>
<td>19</td>
<td>18.4</td>
</tr>
<tr>
<td>Total</td>
<td>47</td>
<td>100.0</td>
<td>56</td>
<td>100.0</td>
<td>103</td>
<td>100.0</td>
</tr>
</tbody>
</table>

P=0.4
Table 19: Fundal changes in relation to types of lesions in the study population

<table>
<thead>
<tr>
<th>Fundal finding</th>
<th>Benign</th>
<th>Malignant</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No</td>
<td>%</td>
<td>No</td>
</tr>
<tr>
<td>Normal</td>
<td>28</td>
<td>45.9</td>
<td>33</td>
</tr>
<tr>
<td>Atrophy</td>
<td>3</td>
<td>33.3</td>
<td>6</td>
</tr>
<tr>
<td>papilloedema</td>
<td>8</td>
<td>42.1</td>
<td>11</td>
</tr>
<tr>
<td>Atrophy and papilloedema</td>
<td>0</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>Difficult to assess</td>
<td>8</td>
<td>80.0</td>
<td>2</td>
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<tr>
<td><strong>Total</strong></td>
<td>47</td>
<td>45.6</td>
<td>56</td>
</tr>
</tbody>
</table>

P=0.06
3.6. Imaging findings: *(Figure 10)*:

The commonest imaging finding were ventricular dilatation in 45 (43.7%) patients, shifting of the midline in 28 (26.0%) patients and oedema in 26 (25.2%) patients, ring enhancement in 19 (18.4%) patients. Calcification was found in only 4 (3.9%) patients.

3.6.1. The commonest imaging findings in relations to sites of lesions :*(Table 20)*

- **Ventricular dilatation:**

  Ventricular dilatation was detected in 35(46.7%) patients with supratentorial lesions compared to 13 (46.4%) patients with infratentorial lesions, this was statistically insignificant (p=0.9).

- **Shifting of the midline:**

  shows that midline shift was detected in 22 (29.3%) patients with supratentorial lesions compared to 4 (14.3%) patients with infratentorial lesions, this result was statistically insignificant (p=0.1).

- **Oedema:**

  Oedema was detected in 19 (25.3%) patients with supratenorial lesions and in 7 (25.0%) patients with infratentorial lesions, the result was insignificant statistically (p=0.9).
Figure 10: Imaging findings of patients in the study population
n=103
Table 20: The commonest imaging findings in relation to sites of lesions in the study population

<table>
<thead>
<tr>
<th>Imaging findings</th>
<th>Supratentorial</th>
<th></th>
<th>Infratentorial</th>
<th></th>
<th>Total of the study</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Present</td>
<td>Absent</td>
<td>Present</td>
<td>Absent</td>
<td>Present</td>
<td>Absent</td>
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<tr>
<td>----------------------</td>
<td>---------</td>
<td>--------</td>
<td>---------</td>
<td>--------</td>
<td>---------</td>
<td>--------</td>
</tr>
<tr>
<td>Ventricular dilatation</td>
<td>35</td>
<td>46.7</td>
<td>40</td>
<td>53.3</td>
<td>13</td>
<td>46.4</td>
</tr>
<tr>
<td>Midline shift</td>
<td>22</td>
<td>29.3</td>
<td>53</td>
<td>70.7</td>
<td>4</td>
<td>14.3</td>
</tr>
<tr>
<td>Oedema</td>
<td>19</td>
<td>25.3</td>
<td>56</td>
<td>74.7</td>
<td>7</td>
<td>25.0</td>
</tr>
</tbody>
</table>
3.6.2. The commonest imaging findings in relation to types of lesions: *(Table 21)*

- **Ventricular dilatation:**
  Ventricular dilatation was detected in 19 (40.4%) patients with benign lesions compared to 26 (46.4%) patients with malignant lesions, this result was statistically insignificant (p=0.5).

- **Shifting of the midline:**
  Midline shift was detected in 16 patients (34.0%) with benign lesions compared to 10 patients (14.3%) with malignant lesions, this result was statistically insignificant (p=0.06).

- **Oedema:**
  Oedema was detected in 13 patients (27.7) with benign lesions compared to 13 patients (23.2%) with malignant lesions, this result was statistically insignificant (p=0.6).

3.7. **Types lesions in relation to the operative treatment:**

  Seventeen (85.0%) patients with benign lesions were operated while only 11 (36.7%) patients with malignant lesions were operated, this result was statistically insignificant (p=0.6).
Table 21: The commonest imaging findings in relation to types of lesions (benign and malignant) in the study population

<table>
<thead>
<tr>
<th>Imaging findings</th>
<th>Supratentorial</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Present</td>
<td>Absent</td>
<td>Present</td>
<td>Absent</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>%</td>
<td>No</td>
<td>%</td>
<td>No</td>
<td>%</td>
<td>No</td>
<td>%</td>
</tr>
<tr>
<td>Ventricular dilatation</td>
<td>19</td>
<td>40.4</td>
<td>28</td>
<td>59.6</td>
<td>26</td>
<td>46.4</td>
<td>30</td>
<td>53.6</td>
</tr>
<tr>
<td>Midline shift</td>
<td>16</td>
<td>34.0</td>
<td>31</td>
<td>66.0</td>
<td>10</td>
<td>17.9</td>
<td>46</td>
<td>82.1</td>
</tr>
<tr>
<td>Oedema</td>
<td>13</td>
<td>27.7</td>
<td>34</td>
<td>72.3</td>
<td>13</td>
<td>23.2</td>
<td>43</td>
<td>76.8</td>
</tr>
</tbody>
</table>

n= 103

Total of the study | Total of present | Total of absent | Total | No | % | No | % | No | % |
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Present</td>
<td>45</td>
<td>43.7</td>
<td>58</td>
<td>56.3</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Absent</td>
<td>26</td>
<td>25.2</td>
<td>77</td>
<td>74.8</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>103</td>
<td>100</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
3.8. Types of lesions in relation to the Shunt operation:

Table 22 shows that 21 (65.6%) patients with malignant lesions had ventriculoperitoneal shunt, compared to only 7 (35.0%) patients with benign lesions had the shunt operation, this difference was statistically significant (p=0.03).

3.9. Commonest Detected Lesions:

3.9.1. Astrocytoma:

Astrocytoma was the commonest malignant condition detected in 36 (35.0%) children.

3.9.1.1. Age and sex distribution:

There were 5 children (13.9%) <5 years, 10 children were 5-10 years (27.8%) and 21 children (58.3%) were > 10 years.

Males were 19 patients (52.8%) and females were 17 patients (47.2%).

3.9.1.2. Clinical presentation: (Figure 11)

The commonest presentation were headache in 27 (75.0%) patients, visual impairment in 19 (52.8%) patients and unsteadiness was found in 18 (50.0%) patients.
Table 22: The relation between types of lesions and the V.P shunt operation in the study population

<table>
<thead>
<tr>
<th>Shunt operation</th>
<th>Benign</th>
<th></th>
<th>Malignant</th>
<th></th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No</td>
<td>%</td>
<td>No</td>
<td>%</td>
<td>No</td>
</tr>
<tr>
<td>present</td>
<td>7</td>
<td>35.0</td>
<td>21</td>
<td>65.6</td>
<td>28</td>
</tr>
<tr>
<td>Absent</td>
<td>13</td>
<td>65.0</td>
<td>11</td>
<td>34.4</td>
<td>24</td>
</tr>
<tr>
<td>Total</td>
<td>20</td>
<td>100.0</td>
<td>32</td>
<td>100.0</td>
<td>52</td>
</tr>
</tbody>
</table>

P=0.03
Figure 11: The presenting symptoms of patients with Astrocytoma

n=36
3.9.1.3. **Clinical examination:** *(Figure 12)*

The commonest clinical finding was hyporeflexia in 13 (36.1%) patients, abnormal gait (mainly ataxia) in 12 (33.3%) patients and cranial nerve involvement was detected in 12 (33.3%) patients.

Fundus examination revealed that 8 (22.2%) patients had optic atrophy, 8 (22.2%) patients had papilloedema.

3.9.1.4. **Imaging findings:**

The lesions were detected in supratentorial and infratentorial equally, 18 (50.0%) patients. The commonest site for astrocytoma was the cerebellum in 17 (47.2%) patients. The commonest lesion found in the cerebellum was astrocytoma compared to other types of lesions, this result was statistically significant (*p*=0.001). The lesion was parietal in 5 (13.9%) patients, temproparietal in 5 (13.9%) patients. *(Figure 13)*

Shifting of the midline was detected in 8 (22.2%) patients, oedema was found in 10 (27.8%) patients. In precontrast study there were 6 (16.7%) lesions showed hypodense, 5 (13.9%) lesions showed hyperdense and 9 (25.0%) lesions showed mixed nature. Five (13.9%) lesions showed ring of enhancement. Ventricular dilatation was detected in 16 (44.4%) patients. In contrast study 13 (36.1%) patients showed mixed feature and 11 (30.6%) patients showed hypodense lesions.
Figure 12: The clinical signs in patients with Astrocytoma
n=36

- Hyperreflexia: 36.1%
- Abnormal gait: 33.3%
- Cranial N. lesions: 33.3%
- Optic atrophy: 22.2%
- Papilledema: 22.2%
- Hypotonia: 13.9%
- Unable to walk: 8.4%
- Hypertonia: 8.3%
Figure 13: Sites of Astrocytoma in the study population
n=36
3.9.2 Abscess:

Abscess was found in 20 patients (19.4%).

3.9.2.1. Age and sex distribution:

There were 9 (45.0%) patients below the age of 5 years, 5 (25.5%) patients between 5-10 years and 6 (30.0%) patient were above 10 years of age. They were 13 (65.0%) male (65.0%) and 7 (35.0%) female.

3.9.2.2. Clinical presentation:

The commonest presenting symptoms were seizures in 16 (80.0%) patients, vomiting in 11 (55.0%) patients, fever in 11 (55.0%) patients and headache in 10 patients. (Figure 14)

3.9.2.3. Clinical examination:

Cranial nerves involvements were detected in 5 (25.0%) patients. Fundal examination detected 6 (30.0%) patients with papilloedema and 2 (10.0%) patients with optic atrophy. (Figure 15)

3.9.2.4. Imaging findings:

The lesions were detected in supratentorial in 18 (90.0%) patients. The commonest sites were parietal lobes in 9 (45.0%) patients, frontal lobes in 5 (25.0%) patients and temporal lobes in 3 (15.0%) patients. (Figure 16)
Figure 14: The presenting symptoms for patients with Abscess in the study population

n=20
Figure 15: Clinical signs in patients with Abcess in the study population

n=20
Figure 16: Sites of Abscess in the study population
n=20

Sites

- Parietal
- Frontal
- Temporal
- Cerebellar
- Lateral ventricle
- Pons

Percentage
Midline shift was detected in 6 (30.0%) patients, oedema was detected in 14 (70.0%) patients, this relation was statistically significant (p=0.00).

Ring of enhancement was found in 10 (50.0%) patients, the result was statistically significant (p=0.00). Ventricular dilatation was detected in 9 (45.0%) patients. Contrast enhancement showed hypodense lesions in 6 (50.0%) patients.

### 3.9.3. Medulloblastoma:

Medulloblastoma was detected in 11 (10.7%) patients.

#### 3.9.3.1. Age and sex:

There were 3 (27.3%) children below age of 5 years, 6 (54.5%) children between 5-10 years and 2 (18.2%) children their age above 10 years.

The males were 9 (81.8%) patients and the females were 2 (18.2%) patients.

#### 3.9.3.2. Clinical presentation:

The commonest presenting symptoms were vomiting which was detected in 9 (81.8%) patients, headache in 8 (72.0%) patients and motor weakness in 7 (63.6%) patients. (*Figure 17*)
Figure 17: The presenting symptoms of patients with Medulloblastoma in the study population

n=11
3.9.3.3. Clinical examination:

Cranial nerves involvements were found in 4 (36.4%) patients. Papilloedema was detected in 2 (18.2%) patients. (Figure 18)

3.9.3.4. Imaging findings:

The lesion was in the cerebellar vermis in 7 (63.6%) patients occipital in 2 (18.2%) and in the 4th ventricle in 2 (18.2%) patients. (Figure 19)

Midline shift and ring of enhancement were not detected in this lesion while odema was detected in only 1 (9.1%) patient. Ventricular dilatation was found in 2 (18.2%) patients and contrast enhancement showed mixed lesions in 4 (66.6%) patients.

3.9.4. Other intracranial space occupying lesions: Table (23)

The commonest presenting symptom in the cystic lesions (arachnoid cyst and dermoid cyst) was increased the size of the head, in (57.1%) of arachnoid cyst patients and in (60%) of dermoid cyst patients. This relation was statistically significant (p=0.04). Cranial nerves lesions were the commonest findings in most of the lesions. Tuberculomas were detected in only 2 (1.9%) patients.
Figure 18: The clinical signs of patients with Medulloblastoma in the study population

n=11
Figure 19: The commonest sites for Medulloblastoma in the study population

n=11

Percentage

Sites

cerebellar vermis 63.6%
occipital 18.2%
fourth ventricle 18.2%
Table 23: Other histological types of lesions in intracranial SOLs in the study population

<table>
<thead>
<tr>
<th>Lesion</th>
<th>Age</th>
<th>Symptom</th>
<th>sign</th>
<th>Number</th>
<th>percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arachnoid cyst</td>
<td>&lt;5 yr</td>
<td>Increase head</td>
<td>Cranial</td>
<td>7</td>
<td>6.8</td>
</tr>
<tr>
<td></td>
<td></td>
<td>size</td>
<td>nerve lesion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dermoid cyst</td>
<td>&lt;5 yr</td>
<td>Increase head</td>
<td>Cranial</td>
<td>5</td>
<td>4.9</td>
</tr>
<tr>
<td></td>
<td></td>
<td>size</td>
<td>nerve lesion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Meningioma</td>
<td>All age</td>
<td>Seizures</td>
<td>Cranial</td>
<td>3</td>
<td>2.9</td>
</tr>
<tr>
<td></td>
<td></td>
<td>nerves lesion</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Epindymoma</td>
<td>&lt; 5yr</td>
<td>Headache</td>
<td>Cranial</td>
<td>4</td>
<td>3.9</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Vomiting</td>
<td>nerves</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>seizures</td>
<td>lesions</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cranipharyngioma</td>
<td>5-10</td>
<td>Headache</td>
<td>Cranial</td>
<td>5</td>
<td>4.9</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Vomiting</td>
<td>nerves</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Seizures</td>
<td>lesions</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tuberculoma</td>
<td></td>
<td>Headache</td>
<td>Motor</td>
<td>2</td>
<td>1.9</td>
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<td></td>
<td></td>
<td></td>
<td>weakness</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hydatid cyst</td>
<td></td>
<td>Seizures</td>
<td>Motor</td>
<td>2</td>
<td>1.9</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>weakness</td>
<td></td>
<td></td>
</tr>
<tr>
<td>papilloma</td>
<td></td>
<td>Vomiting</td>
<td>Cranial</td>
<td>2</td>
<td>1.9</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>nerve lesion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oligodendrogloma</td>
<td></td>
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<td></td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Epidermoid cyst</td>
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<td></td>
<td></td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Adenoma</td>
<td></td>
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<td>1</td>
</tr>
<tr>
<td>Germ cell</td>
<td></td>
<td></td>
<td></td>
<td>1</td>
<td>1</td>
</tr>
</tbody>
</table>
3.9.5. Other lesions detected:

Congenital heart disease was detected in 5 (4.8%) patients, 2 (1.9%) patients had central precocious puberty, one (0.9%) patient had dextrocardia, one (0.9%) patient had mitral valve disease, one (0.9%) patient had Dandy-Walker disease, and one (0.9%) patient had sickle cell disease. One (0.9%) patient had meningomyelocele. (Table 24)
Table 24: Other lesions detected with intracranial cranial SOLs in the study population

<table>
<thead>
<tr>
<th>Lesion</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Central precious puberty</td>
<td>2</td>
<td>1.9</td>
</tr>
<tr>
<td>Congenital H.D</td>
<td>5</td>
<td>4.8</td>
</tr>
<tr>
<td>Dandy walker</td>
<td>1</td>
<td>0.9</td>
</tr>
<tr>
<td>Dextrocardia</td>
<td>1</td>
<td>0.9</td>
</tr>
<tr>
<td>Mitral valve lesion</td>
<td>1</td>
<td>0.9</td>
</tr>
<tr>
<td>Meningiomyelocele</td>
<td>1</td>
<td>0.9</td>
</tr>
<tr>
<td>Sicke cell disease</td>
<td>1</td>
<td>0.9</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>12</strong></td>
<td><strong>11.2</strong></td>
</tr>
</tbody>
</table>
Chapter Four

DISCUSSION

The study included 103 children with predominance of males, male to females ratio was 1.7:1. This is comparable to that reported in literature \(^{(1)}\) and this result is comparable also to the study by Ifran in Karachi who reported that male : female ratio was 1.6:1\(^{(93)}\). This can be explained by the fact that most of SOLs are predominating in males. Although the age groups were divided into three groups generally no apparent differences in the distributions were found, (less than 5 years were 34%, between 5-10 years were 31% and > 10 years were 35%). So all age groups were affected, this result may be explained by the fact that many pathologies were included in the SOLs.

The most common presentations were that of increased intracranial pressure, this was in agreement with Desai result in India, mentioning that the commonest presentations were symptoms of increased intracranial pressure \(^{(11)}\). Headache was the commonest presenting symptom (59.2%) but did not occur in all patients and this goes with literature mentioning that headache is common but not invariable \(^{(16)}\), this is more than Sanchez result.
which reported that only 3 (3.2%) children out of 94 children presented with headache necessitated hospital admission had intracranial space occupying lesions (94).

The next common presenting symptom was seizures in 51 (49.5%) patients and it seems to be higher in our study compared to Mustafa result (29) who found that vomiting was found in (46.6%) and seems to be similar to that reported in literature (1), however Sanchez in his study mentioned that vomiting was accompanying symptom in 38.2% (94). Motor weakness was found in 40 (38.8%) cases, which also was found to be significant. The presence of fever in 26.2% of patients can be explained by the fact that abscesses constituting a significant proportion of SOLs in this study 19.4% could be responsible for the fever.

Increased in the size of the head was found in 21% can be explained by the fact that the study included patients below 2 years of age which were 19 (18.5%) patients, this increase in the size due to opened sutures in those children.

Abnormal gait was present in (42.7%) and more common with infratentorial lesions, a possible cause of this result is that the cerebellar involvement is common in this study so many patients presented with abnormal gait mainly ataxia. Cranial nerves involvement occurred in
(39.8%) and the commonest nerve to be affected is the 2nd followed by the
3rd then 6th table(2) , Wanyoike reported that 20% of his cases were blind at
the time of presentation (6). The commonest finding in the optic fundi was
papilloedema (16.6%), followed by optic atrophy which was present in
(7.8%), compared to Mustafa , who studied intracranial SOLs in adults in
118 patients he found the same order of the lesions(29).

Supratentorial lesions were more common than infratentorial lesions
this can be explained by the fact that this study included all lesions not only
tumours which are more common infratentorial in children (13). The
cerebellum was the commonest site for tumours, mainly astrocytoma that in
keeping with literature (3), however Desai mentioned that the commonest site
in the cerebellum was the vermis (11). The next common site is the occipital
region (8.7%). These findings are not comparable to that reported by
Mustafa , that the commonest location was tempoparietal(29), however his
study confined to adults, also Abu Salih and Abdul Rahman reported the
commonest site was frontal (6), their study was confined to patients with
brain tumours.

Supratentorial lesions were found to be commonly benign in
41(54.7%) children compared to infratentorial lesions which were
commonly malignant in 22 children (78.6%), this may be due to that fact
that most of tumours confined to infratentorial area while most of abscess and cysts confined to supratentorial.

The benign conditions were found to be commonly below 5 years, this because of increased incidence of the congenital cysts. The malignant conditions increased with age and common in male more than female, this in keeping with Hoffman study mentioned that there is increasing incidence of primary brain tumors over the past few decades. Data for the years 1985 through 1999 were used to determine incidence trends in the broad age groups 0-19, 20-64, and ≥65 years, he concluded that incidence increased modestly. When brain lymphomas were excluded, this increase remained statistically significant, increases that were not specific to any population subgroup were seen for oligodendrogliomas, ependymomas, meningiomas, and nerve sheath tumors \(^{(95)}\).

Astrocytoma was found to be the commonest intracranial SOL 35% and it was the most common primary brain tumour. These findings are comparable to those in literature \(^{(3)}\). Ifran in Karachi mentioned that the commonest lesion in his study was astrocytoma that comprised 32.1% of the total cases (386 patients) followed by meningioma (13.7%), however Abu Salih and Abdul Rahman found that meningioma was the commonest brain tumour \(^{(6)}\). The age group >10 years affected more common (58.3%)
followed by age group 5-10 years (27.8%) then the age group <5 years (13.9%), this result can be explained by the fact that there is increase incidence of brain tumours (96). Males were affected more commonly than females (52.8%) this is in keep with literature also Mustafa concluded the same result (29) but in consistent with Wanyoike result mentioning that female affected more than male (9).

The commonest symptom was headache (75%), in view of different reports in literature about occurrence of headache in astrocytoma and gliomas in general, headache in this study seems to be higher than that reported by Abu Salih and Abdul Rahman (6), but less than that reported by Mustafa (29). The next commonest symptoms were vomiting and seizures in (30.6%), the result of vomiting was comparable to Mustafa result (32.2%) but seizures seems to be less than that reported by Mustafa SA (51.6%) (29) this can be explained by the fact that most of astrocytoma in our study was confined to the cerebellum not to lobes where there are increase risk of irritation and so to seizures, this result was not similar to Wanyoike reported that cerebellar symptoms were the most common mode of presentation (30%) followed by headaches and vomiting and no seizures this because the lesions were in the posterior fossa (9). The relationship between head trauma and astrocytoma statistically insignificant, however many patients related
their headache to a history of head trauma. Cranial nerves involvement was evident in (33.3%) which is less than that reported by Mustafa (41.9)\(^{(29)}\), it was mentioned in literature that Posterior fossa lesions carry a high risk of obstructive hydrocephalus, cranial nerves palsy and brain stem compression pituitary and chiasmatic tumors a risk of blindness\(^{(97)}\), the commonest affected cranial nerve in this study was the 2\(^{nd}\) Mustafa reported that the commonest affected cranial nerve was the 7\(^{th}\)\(^{(29)}\). Papilloedema and optic atrophy were equally detected (22.2%). Ataxia was detected in (33.3%) this may be due to the fact that most of astrocytoma in our study was in the cerebellum (30.6%), these comparable with literature\(^{(3)}\) reported that ataxia is often associated with posterior fossa tumours although sometimes large tumours can cause no abnormalities in movement, this result is higher than that reported by Mustafa (12.9%).

Shifting of the midline in astrocytoma in imaging was detected in (22.2%), while edema was found in 27.8%. Most of astrocytomas were mixed lesions in this study (36.1%), some hypodense (30.6%) and this finding is different from literature mentioned that astrocytoma more common hypodense lesions\(^{(98)}\), Mustafa reported that the commonest attenuation was hypodense lesions\(^{(29)}\), Vaghi mentioned that forty surgically proved gliomas have been studied by MRI: 21 were low-grade gliomas and
19 were anaplastic astrocytomas and glioblastomas. MRI findings were similar in low-grade and anaplastic astrocytomas, but quite different from imaging of glioblastomas. Tumoral cyst and areas of necrosis were recognized on MRI studies and confirmed by surgical findings. Differentiation between tumour and oedema was difficult (99).

Brain abscess constituted (19.4%) of all intracranial SOLs. It was the second common SOL. It affected mostly those in the age group <5 years (45.0%) which can be explained by increase incidence of congenital heart disease, sickle cell disease and otitis media at these age, followed by those in age group >10 years (30.0%) then (25.5%) in age group 5-10 years, this incomparable with literature in which abscess more common in age group 5-10 years (3). Males were predominantly affected (65.0%), this in keeping with literature reporting that abscess is 2 to 3 times common in males (13), this also in consistent with Mawag result mentioning that males are more affected but he mentioned that the common cause for brain abscess in his study was trauma (10).

The commonest symptom were seizures (80.0%) followed by vomiting and fever in (55.0%), headache was found in (50%), this is not comparable to literature stating that headache and fever are the commonest presenting symptoms (3), this can be explained by the fact that most of our patients < 5
years (45.0%) had increased incidence of congenital heart disease and otitis media which commonly affect the temporal lobe so cause cortical irritation which lead to seizures, another explanation is that seizures are more detectable in young children by mothers than fever. Motor weakness was found in (40%), unsteadiness in (25%). Cranial nerves involvement were detected in (25%), this is less than that reported by Mustafa where cranial nerves involvement in (50%) of his cases (29), the most affected cranial nerve was the 6th. Papilloedema was detected in (30%) and optic atrophy in (10%), in Mustafa SA study papilloedema was detected in (33.3%) (29).

Commonly the abscess was located in parietal in (45.0%), frontal in (25.0%) and temporal in (15.0%), this is comparable with literature which mentioned that (80.0%) of abscesses were divided between the frontal, parietal and temporal areas (3).

Hypodense lesion and ring enhancement were found in (50.0%). Mustafa reported that all abscesses in his study showed hypodense lesion and all had ring of enhancement (100.0%) (29).

Oedema was detected in (60.0%) of cases which statistically significant (p=0.00).

Congenital heart diseases was a risk factor in (4.8%) which may added to the risk factors for brain abscess, Mehnaz A in Pakistan in his study found
that cyanotic congenital heart disease was a predisposing factor in 11 children (37%) out of 30 children \(^{(100)}\).

Medulloblastoma accounts for (10.7%) of all cases and the second common brain tumour in the study, it was mentioned that astrocytoma and medulloblastoma are more common in children than the other types of tumours \(^{(3)}\). The commonest age group between 5-10 years (54.5%) this result comparable with literature reported that medulloblastoma is the most prevalent brain tumours in children less than 7 years \(^{(3)}\), followed by <5 years (27.3%) then >10 years (18.2%), it was mentioned in literature that medulloblastoma uncommon in adults, i.e 20 years of age \(^{(101)}\).

Males were commonly affected (81.8%), and females were (18.2%), this result comparable with the literature mentioned that males more commonly affected than females \(^{(101)}\), however Wanyoike reported that Medulloblastoma in his study a predominantly female tumour as opposed to available literature \(^{(9)}\).

Clinical presentation was dominated by raised ICP, thus vomiting was the commonest presenting symptoms (81.8%), then headache in 72.0% it was mentioned that no specific symptoms and signs for medulloblastoma and headache and vomiting was due the hydrocephalus result rather than the mass it self \(^{(101)}\).
Cranial nerves involvement was found in (27.3%), the affected cranial nerves were the 2nd, 3rd and 6th cranial nerve. Papilloedema was detected in (18.2%).

Medulloblastoma was found to be more commonly infratentorial which was comparable which literature (101) mentioning that medulloblastoma is a typically posterior fossa tumour but can metastasize by CSF. The commonest site was cerebellar vermis in (63.6%), 4th ventricle is the second common place in (18.2%), literature mentioned that medulloblastoma can originate from the roof of the 4th ventricle and grows rapidly to fill the 4th ventricle (3). Contrast enhancement was mixed in (66.6%) this result was uncomparable with literature mentioning that the common appearance is the hyperdense a feature that can differentiate it from astrocytoma (101) however Mueller mentioned that medulloblastoma can have a typical features included cyst formation in (77%) of his cases who were 13 patients and mixed enhancement in (14%) (95).

Arachnoid cyst accounted for (6.8%) of all cases, this was higher than that reported in literature which reported only (1%) of all SOLs (70) and higher than Mustafa result where reported (2.5%) of all intracranial SOLs. The commonest age group was below 5 years (85.7%), this was comparable with literature and this due to the fact that it is a congenital lesion. Males
were affected more (57.1%). The commonest presenting symptom was increase the size of the head (57.1%) which comparable with literature (70). Seizures were detected higher in our study (57.1%) than that mentioned by Gomez in his study (25.7%) (70). Cranial nerves lesion was detected in (28.6%).

The commonest site for the lesion was temporal in (49.2%), this result was comparable with literature (101) then frontal in (28.6%). Midline shift and ventricular dilatation were detected in (42.8%).

Dermoid cyst was detected in (4.9%) of all cases, All the cases below 5 years. Male were commonly affected (60%). The commonest presenting symptoms are those of increase intra cranial pressure (increase size of the head and vomiting) in (60.0%), in literature it is reported that dermoid cysts can be space-occupying masses because of increasing CSF retention, which may be due to a ball-valve mechanism or to inadequate communication, the frequently associated hydrocephalus, can be due to mechanical factors (77).

Meningioma was detected in (2.9%) of all cases, Abu Salih and Abdul Rahman found that meningioma was the commonest brain tumour in their study; this was true for adult population (6).

Age distribution was equal in all age group (33.3%) and males were affected more (66.7%) this not in keep with literature that mentioned females
are common affected than males \(^{(13)}\), but this may be due to the small size of the affects only 3 patients.

Ependymoma accounts for \((3.9\%)\) for all cases of SOLs. The commonest age group was below 5 years \((75.0\%)\), this result goes with literature mentioning that it is more common in young children \(^{(101)}\). Males and females were equally affected. The commonest presenting symptoms were headache, vomiting and seizures \((50\%)\). Cranial nerves lesion was detected in \((50.0\%)\). The commonest site was occipital in \((50.0\%)\) which was incomparable with literature which mentioned the 4\(^{th}\) ventricle as the common site \(^{(101)}\), this difference may be due to the small size of patients.

Cranioharyngioma was detected in \((4.9\%)\) of all cases. The commonest age group was between 5-10 years \((60.0\%)\), which was comparable with literature \(^{(101)}\). Headache and vomiting were the commonest presenting symptoms in \((80.0\%)\), this result was different from the study mentioned that visual deficits and endocrine dysfunctions are the most frequent presenting symptoms \(^{(101)}\), however children frequently presented with symptoms of increased intracranial pressure.

The commonest sites for the lesion were occipital and suprasellar in \((40.0\%)\) and occupied both sellar and supra sellar regions in \((20.0\%)\), this was comparable with literature which mentioned sellar and suprasellar as
commonest sites$^{(3)}$. Calcification was detected in (80.0%) of cases and it was found to be significant as mentioned in literature$^{(3)}$.

Although tuberculosis was a common endemic disease in our country, it seems to be that brain tuberculoma is underdiagnosed (1.9%), Ramamurthi mentioned that tuberculomas of the brain in children constitute 5% to 8% of intracranial space occupying lesions in developing countries, the image morphology of tuberculoma could simulate other lesions like gliomas$^{(96)}$.

Two patients (1.9%) had hydatid cysts, this result comparable with literature mentioned that the incidence of hydatid is (2%)$^{(12)}$ and (1%)$^{(83)}$ in another source, however Mustafa reported higher result (2.5%). In the 2 patients no evidence of extracranial hydatid cyst was detected.
CONCLUSION

- Different types of intracranial SOLs were identified. Neoplastic conditions accounts for the majority of all intracranial SOLs.
- The commonest intracranial SOL was astrocytoma followed by abscess and medulloblastoma.
- Intracranial SOLs are more common in males and all age groups are affected.
- The commonest clinical presentations were that of raised intracranial pressure, headache and vomiting seem to be important signs for serious problems.
- Intracranial SOLs commonly located supratentorial, while tumours commonly located infratentorially mainly the cerebellum.
- The benign conditions were more common in age group <5 years and the malignant conditions were more common in age group <10 years.
- Malignant lesions commonly affected males.
- Benign conditions more operable than the malignant conditions and malignant lesions more needed for ventriculoperitoneal shunts.
- Incidence of tuberculoma was found to be less than expected therefore a high index of clinical suspicion in addition to proper investigations for tuberculosis should be considered.
- Some associated diseases were detected which may need further studies.
- Imaging studies were helpful in detecting the lesions and their secondary effects. In some lesions the finding has a high validity.
RECOMMENDATIONS

- Simple complaint like headache and vomiting must be considered and appropriately evaluated.
- Proper history taking and proper examination should be executed to detect the symptoms and signs of the intracranial space occupying lesions early enough to offer better opportunity for better outcome.
- Imaging techniques should be utilized early in the presentation; MRI might offer better chance for detection of lesions at an early stage.
- Early interactions to relief the increased I.C.P by placement of V.P shunts.
- There is a need for performing more biopsies for reaching the final diagnosis in accessible tumours.
- Emphasis on diagnosis of SOL in undergraduate curricular and CPD courses for general practitioners stressing the importance of early referral.
- Decentralization of services by establishment of regional diagnostic centers and specialized team.
- Training more paediatrics neurologists, neurosurgeons and radiologists to deal with cases in the district hospitals.
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Questionnaire

CLINICAL PATTERN AND IMAGING FINDINGS OF INTRACRANIAL SPACE OCCUPYING LESIONS IN CHILDREN

Name ……………    Age ……………    Sex ……………
Residence……………..    Tribe……………

General Symptoms:
- Headache    yes □    no □
- Vomiting    yes □    no □
- Nausea    yes □    no □
- Seizures    yes □    no □
- Fever    yes □    no □
- Loss of consciousness    yes □    no □
- Neck stiffness    yes □    no □
- Mental symptom(specific)    yes □    no □
- Increase size of the head    yes □    no □

Sensory symptoms:
- Loss    yes □    no □ site...
- Paraesthesias    yes □    no □ site…

Motor symptoms:
- Unsteadiness    yes □    no □ site…
• Tremors yes □ no □ site..
• Stiffness yes □ no □ site..
• Motor weakness yes □ no □ site...

Symptom due to cranial nerve involvement:

• Anosmia yes □ no □
• Visual impairment yes □ (specify) no □
• Diplopia yes □ no □
• Squint yes □ no □
• Deviation of the mouth yes □ no □
• Deafness yes □ no □
• Dysphagia yes □ no □

Other symptoms:

• Speech disturbance yes □ no □
• Behavioral changes yes □ (specify) no □
• Incontinence yes □ no □
• Gait disturbance yes □ (specify) no □

Past medical history:

• TB yes □ no □
• Trauma yes □ no □
• Epilepsy yes □ no □
• Otitis media yes □ no □
• Sinusitis
  yes □ no □

• Schistosomiasis
  yes □ no □

Family history:
  Similar disease
  yes □ no □
  Who………
  Other disease
  yes □ no □
  What………
  who…………..

Drug history:

Physical examination:-
  Wt………………….  Ht………….  Hc…….

  ➢ Fever
    yes □ no □
  ➢ Pallor
    yes □ no □
  ➢ Jaundice
    yes □ no □
  ➢ Lymph node
    yes □ no □

  i.  CVS
      normal □ abnormal □
  ii. RS
      normal □ abnormal □
  iii. Abdomen
       normal □ abnormal □
iv. Skin condition
   - normal □
   - abnormal □

v. Nervous system:
   - Oriented    yes □
   - yes □
   - no □
   - Dementia    yes □
   - yes □
   - no □
   - Confused    yes □
   - yes □
   - no □
   - Motor dysphasia yes □
   - yes □
   - no □
   - Comatosed   yes □
   - yes □
   - no □
   - GCS…………………

   - Cranial nerve lesions:
      R:
      L:

      Fundoscopy:
      - R: normal □
      - abnormal □
      - specify………..
      - L: normal □
      - abnormal □
      - specify………..

   - Neck:
      - Neck stiffness yes □
      - yes □
      - no □
      - Weakness of flexor yes □
      - yes □
      - no □
      - Weakness of extensor yes □
      - yes □
      - no □
Limbs:

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Note: increased=3  normal=2  reduced=1

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Abdominal reflexes

Planter reflex........( R)...........(L)...............  R=L R<L R>L

Coordination (R) .......(L)...........R=L R<L R>L

Gait normal box abnormal box specify........

The back normal box abnormal box specify........

Skull normal box abnormal box specify........
Investigation:

CBC……………………………………
………………………………………………………………………………
…………………………………………………………………………………..
RFT…………………………………………………………
………………………………………………………………………………
LFT…………………………………………………………
………………………………………………………………………………
Skull X-ray……………………………………………………………..

MRI:………………………………………………………………………………
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Brain CT scan………………………………………………………
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Abdominal U/S……………………………
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Histopathology………………………………………………………………
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Other investigations:………………………………………………………………
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Treatment:

a) Medical (specify)……………………………………
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b) Surgical (specify)……………………………………
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