ISSN PRINT 2319 1775 Online 2320 7876

Research paper UGC CARE Listed (Group -I) Journal Volume 11, S Iss 3, Dec 2022

Magnetic Resonance Imaging Of Space Occupying Lesions In Brain

Dr. Prakash Patil 1 Dr.Deshmukh Amit Hindurao 2 Assist. Prof.
Department of Radio-diagnosis Krishna Institute of Medical Sciences, Krishna Institute of Medical Sciences Deemed to be University ,Karad
Email : drprakash24@gmail.com

Abstract
"With the introduction of CT & MRI scanning, imaging of intracranial space occupying lesion has acquired a new dimension whereby excellent anatomical detail in axial, sagittal & coronal planes as well as tumor tissue characterization has become possible. The advent of MR Angiography has helped to create a virtual 3-D vascular map of tumor blood supplynoninvasively which have helped in the early diagnosis & localization of the SOL & in complement to advanced neurosurgical techniques, have enhanced the prognosis of mass lesions." [2]

"Combined with MR angiography, these modalities give the neurosurgeon a virtual roadmap by which the feasibility and approach to surgery can be decided. [3] On the other hand, CT being less expensive modality is readily available. Also it is better for acute bleed, calcification and bone destruction". [1]

Introduction

"Advantages of MRI over CT are: to know the exact characteristic of the lesion, whether diffuse or focal, residual tumor or recurrence, localization of multiple lesions, relation and extent to the spine, and exactly differentiate doubtful CT tumors. MRI is also useful by virtue of multi-planar imaging &MR angiography".[4] "Intracranial space occupying lesions comprise of a diverse group of lesions. The higher morbidity and mortality that is associated with Intracranial Space Occupying Lesions necessitates their early diagnosis to plan the intervention that is required to prevent them at the earliest". [1]

"The term space occupying lesions of brain is conveniently applied to localized intracranial lesions whether of neoplastic, vascular or chronic/ acute inflammatory origin, which due to its space occupying nature within the skull leads to raised intracranial tension. As skull has capacity to accommodate a fixed amount of tissue, anything which is extra within it will produce symptoms of raised intra cranial tension". [5]

ISSN PRINT 2319 1775 Online 2320 7876

Research paper UGC CARE Listed (Group -I) Journal Volume 11, S Iss 3, Dec 2022

As there are numerous conditions which contribute to intracranial space occupying lesions it is important to assess the patient clinically as well as differentiate between neoplastic & non-neoplastic nature of the lesion as detected on neuro-imaging e.g. CT/MRI, etc.

"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". [10]
Classification of intra cranial 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 ofchoroid plexus.
   e. "Secondary neoplasms."

Table 1: Classification of intra cranial space occupying lesions :

1. Congenital	"Dermoid, Epidermoid, Teratoma."	
2. Traumatic	"Subarachnoid hemorrhage, Subdural and Extradural hemorrhage, Hemorrhagic contusion"	
3. Inflammatory	"Multiple sclerosis and its variants ,Neuromyeltis Optica spectrum, Acute disseminated Encephalomyelitis, Acute Hemorrhagic Leucoencephalitis, Neurosarcoidosis"	
4. Infective	"Abscess, Tuberculoma, Syphilitic Gumma, Fungalgranulomas"	
5. Parasitic	"Cysticercosis, Hydratid cyst, Amebic abscess, Schistosoma japonicum."	
6. Neoplasms	Neuronal Tumors	Gliomas – astrocytoma, ependymoma, oligodendroglioma, germinoma, medulloblastoma.
	"Tumors arising from appendages"	"Meningioma, schwannoma, chondroma, osteoma."
	"Pituitary lesions"	"Pituitary adenoma, Craniopharyngioma."
	"Vascular lesions"	"Angioma, Hemangioblastoma, Papilloma of choroid plexus."
	"Secondary Neoplasms"	"Metastasis"

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 sand creates problems in describing the epidemiology of these conditions." [11]

ISSN PRINT 2319 1775 Online 2320 7876

Research paper UGC CARE Listed (Group -I) Journal Volume 11, S Iss 3, Dec 2022

In the present study ,"those cases were included which were either clinically suspected cases brain space occupying lesions or already diagnosed cases of brain space occupying lesions on cross sectional imaging of MRI"

OBJECTIVES

1. To study the number and distribution of various intracranial space occupying lesions (SOL) and its impact on surrounding structures.
2. To study MRI features of different intracranial space occupying lesions.

Review of Literature

The term "intracranial space occupying lesions" is defined as any neoplasm, benign or malignant, primary or secondary, as well as any inflammatory or parasitic mass lying within the cranial cavity. It also includes haematomas, different types of cysts, & vascular malformations. [6] Different authors have reported that majority of patients of ICSOL had neoplasms followed by infective & traumatic etiology. [7,8] Gliomas are more common followed by meningiomas, abscesses, pituitary tumors &tuberculoma. [9]

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. [10]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".[11]

"Metastatic brain tumours are common in adults but they are relatively rare in children. [12] A study by the National Cancer Institute shows that a significant increase in childhood brain tumours brings them the dubious distinction as themost common paediatric tumour."

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." [19]

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". [11]

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 predispositionto brain tumours and specifically with mutations in the TP53 gene. Mutations in constitutional (that is, non-tumour tissue) TP53 have been linked to patientswith gliomas." [11]

"Familial aggregations of brain tumours occurring in different generations and sibships occur very

ISSN PRINT 2319 1775 Online 2320 7876

Research paper UGC CARE Listed (Group -I) Journal Volume 11, S Iss 3, Dec 2022

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". [19]

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." [19]

"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 patients 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". [20]

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 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, and radiation exposure. Published reports warn that more than 20 rads may cause cataracts later". [19]

"Magnetic resonance imaging (MRI), which involves a high-powered magnet, became available in the mid 1980's. MRI images allow 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". [19]

Intracranial abscesses:
"Intracranial abscesses are rare, severe and potentially lethal infections. They include brain abscess and subdural or extradural empyema. A large numberof brain abscesses are polymicrobials". [25]

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." [51]

Epidemiology:
"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. About 50-75% of intracranial hydatid cysts are seen in patients. This high incidence in patients is probably related to patent ductus arteriosus. Most cysts are supratentorial. Infratentorial hydatid cyst

ISSN PRINT 2319 1775 Online 2320 7876

Research paper UGC CARE Listed (Group -I) Journal Volume 11, S Iss 3, Dec 2022

is very rare. The other less common sites reported are skull, cavernous sinus eyeball, pons, extradural, cerebellum and ventricles". [52, 53, 54]

"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. 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 contentis 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". [53]

ISSN PRINT 2319 1775 Online 2320 7876

Research paper UGC CARE Listed (Group -I) Journal Volume 11, S Iss 3, Dec 2022

Intracranial Tumors[74]

"As a general rule, brain tumors increase in frequency with age, with individual exceptions (e.g. pilocytic astrocytoma, the vast majority of which are found in young patients), and a number of uncommon tumors found in infancy . There are few gender differences, except that as a general rule, gliomas are more frequent in men and meningiomas are more frequent in women."

Epidemiology

- "Incidence increases with age"
- "Equivocal gender distribution"
- "Risk factors"
- "Malignancy elsewhere"

Presentation

- "Headache"
- "Features of raised intracranial pressure"
- "Nausea & vomiting worse in the morning or positional"
- "Altered mental state"
- "Focal neurology may occur as the tumor grows"
- "Adult-onset seizures"
- "Incidental finding"
- "Patients may be imaged for another reason, e.g. Trauma"

Investigation

- "CT is often the first test performed to assess presenting symptoms"
- "MRI is the investigation of choice to characterize the tumor"
- "MRI may be used with symptoms of headaches or seizures"

Treatment

- "Parenchymal brain tumors generally have a poor prognosis"
- "Treatment should be in specialist centers"
- "Anti-epileptic agents may help for those with seizures"
- "Steroids may alleviate symptoms caused by edema"
- "A biopsy may be performed neurosurgically"
- "Some tumors may be removed, e.g. Pituitary tumors
- "Stereotactic radiotherapy can be used for small lesions"

Role of imaging

- "Confirm intracranial abnormality and prioritise MRI"
- "Tumor characterization"
- "Help to determine the grade, and make a decision about biopsy"
- "Follow up **Radiographic features**CT"
- "Often the first line test"
- "Variety of appearances depending on the tumor"
- "Contrast may make lesions more conspicuous"
- "Ct is especially helpful for determining bony involvementMRI"
- "Investigation of choice"
- "Fantastic contrast and spatial resolution"
- "Origin of tumors can be determined"

ISSN PRINT 2319 1775 Online 2320 7876

Research paper UGC CARE Listed (Group -I) Journal Volume 11, S Iss 3, Dec 2022

- "Different sequences are used to determine the likely diagnosis"
- "Specialized sequences can be useful to look at tumor metabolites"

"A definitive diagnosis and the characterization of intracranial space occupying lesions based on the structural MRI alone is difficult. In those cases, the proton magnetic resonance spectroscopy (1H-MRS) along with some other noninvasive techniques represent an advance in the specificity of brain intracranial space occupying lesion diagnosis. 1H-MRS gives completely different information related to the cell membrane proliferation, energy metabolism, neuronal damage, and necrotic transformation of brain or the tumor tissues." [78]
1H-MRS is superior to MRI in the detection of tumor growth in morphologicallynormal tissue and in the differential diagnosis of untreated intracranial SOLs.

"MRS provides a detailed biochemical analysis (metabolites) of the tissue, allowing direct insight into in-vivo human brain metabolism. The metabolites reliably mapped using 1H-MRS include choline [Cho, 3.20 parts per million (ppm)], creatine (Cr, 3.02 ppm), N-acetyl-l-aspartate (NAA, 2.02 ppm), lactate (1.33 ppm), and lipids (1.28–1.33 ppm). Alanine (1.5 ppm) and acetate (1.92 ppm) were also reported." [79]

"MRS provides information on the metabolic state of brain tissue. Thus, it is useful to arrive at a more definitive diagnosis in doubtful intracranial SOLs with similar morphological imaging patterns." [80]

"In the literature on the validation of stereotactic biopsy, great efforts have been made in creating new systems to define accuracy. Currently, aside from meta-analyses conducted by other study groups, our investigation comprises the largest number of biopsies and patients". [86]

"Researchers have reported in the past that even in a community hospital, the invasive diagnosis by stereotactic biopsy had improved the medical management as compared with empirical diagnosis from a computed tomographic or magnetic resonance imaging scan". [87]This picture is nowchanging with the more advanced scans available today.

MATERIALS AND METHODS
The study was conducted in the MRI SECTION OF Department of Radio- diagnosis at Krishna Hospital after obtaining ethical committee clearance. Thepatients included in the study were referred from the various clinical departments of Krishna Hospital**.**

STUDY DESIGN- Observational Study.

SAMPLE SIZE- 90 patients

SAMPLING TECHNIQUE-Convenience Sampling **STUDY DURATION** – November 2017 to November 2019**INCLUSION CRITERIA:**

- Patients of adult age group (age>18years) and both sexes willing to give consent for examination.
- Clinically suspected cases of intracranial SOL which were confirmed onCT.

EXCLUSION CRITERIA:

- Patients with contraindications to MR Imaging: patients with ferromagnetic implants, cochlear implants, etc.

ISSN PRINT 2319 1775 Online 2320 7876

Research paper UGC CARE Listed (Group -I) Journal Volume 11, S Iss 3, Dec 2022

- Critically ill patients who are on life support.
- Patients with claustrophobia.
- Patients with known allergy to contrast agent.

METHODOLOGY

Informed consent was taken from the patient/attendant/legally acceptable representative for inclusion in the study as per the proforma attached.

Computed Tomography:

CT was performed on 16 slice Multi-detector CT Siemens Somatom Emotion machine in our institute or done outside with high suspicion of intracranial space occupying lesion and were found positive.

Those cases were sent to MRI section of department of Radio-diagnosis.

Clinical evaluation:

A detailed history was taken with complete physical and systemic examinationof thepatient. Relevant biochemical investigations were done wherever required.

Magnetic Resonance Imaging:

MRI was performed on **MAGNETOM AVANTO 1.5 –TA Tim+Dot MR System (SIEMENS).** Patients were positioned in the gantry with all precautionary measures. Scout images in axial, sagittal and coronal planes were taken. Basic sequences for brain,e.g.,T1W,T2W,FLAIR,DWI,ADC& HEMO were taken in different planes. Additional sequences,e.g., SWI,Mag,PHASE,T1FS pre and post contrast images, CISS and MRS were taken as per the requirement of the study . The contrast used was Gadolinium based (i.e. gadobenetedimeglumine 0.5M solution-10ml vial).The dose given was 0.2mmol/kg.Dynamic contrast imaging was also done wherever needed.
Acquired images were analysed and then entered in the masterchart.

OBSERVATIONS AND RESULTS

The present study was carried out in the MRI section of Department of Radio- diagnosis, Krishna Hospital, Karad, among a total of 90 cases suspected of having ICSOL on the basis of history and/or suspected on CT.

DEMOGRAPHIC CHARACTERISTICS

In our study, we observed cases with suspected ICSOL belonged to different adult age group and both genders. Hence, in order to study their age and gender wise distribution, we assessed their demographic characteristics.

Age distribution:

The mean age observed in our study was 48.37 ± 14.64 years. Most common age group decade was 41-50 years with 27 patients (30%), followed by 51 —
60 years with 16 patients (17.78%) and 41-50 years with 16 patients(17.78%).

Table 2: Distribution of patients according to their age group

Age	Number	%
18-20	4	4.44
21-30	8	8.88
31-40	16	17.78
41-50	27	30.00
51-60	16	17.78
61-70	14	15.56

ISSN PRINT 2319 1775 Online 2320 7876

Research paper UGC CARE Listed (Group -I) Journal Volume 11, S Iss 3, Dec 2022

71-80	5	5.56
Total	90	100

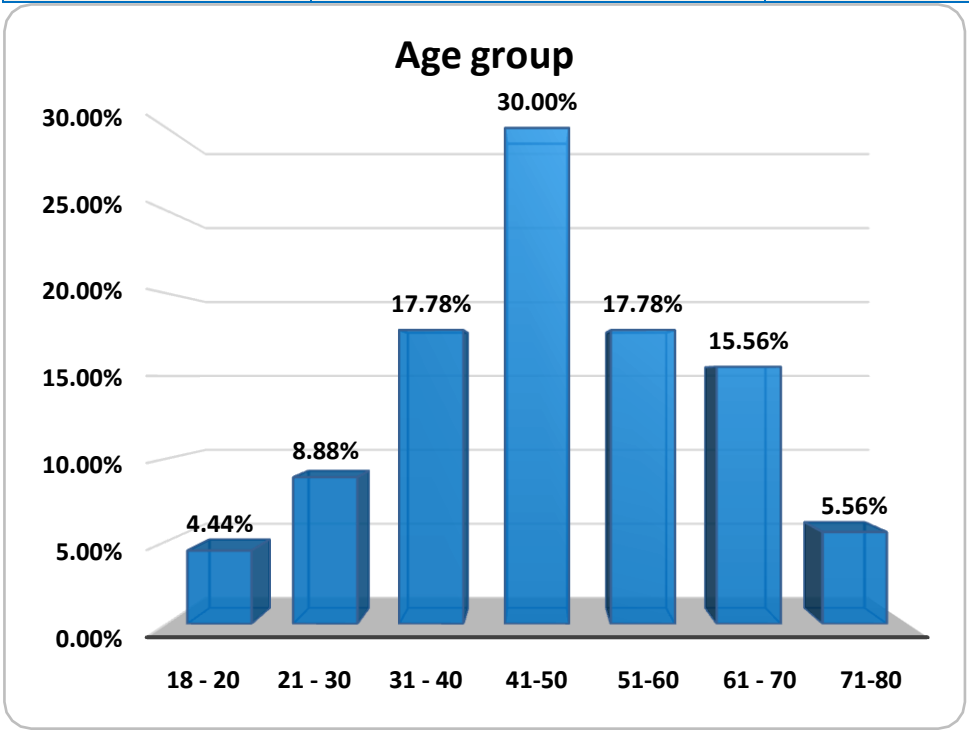
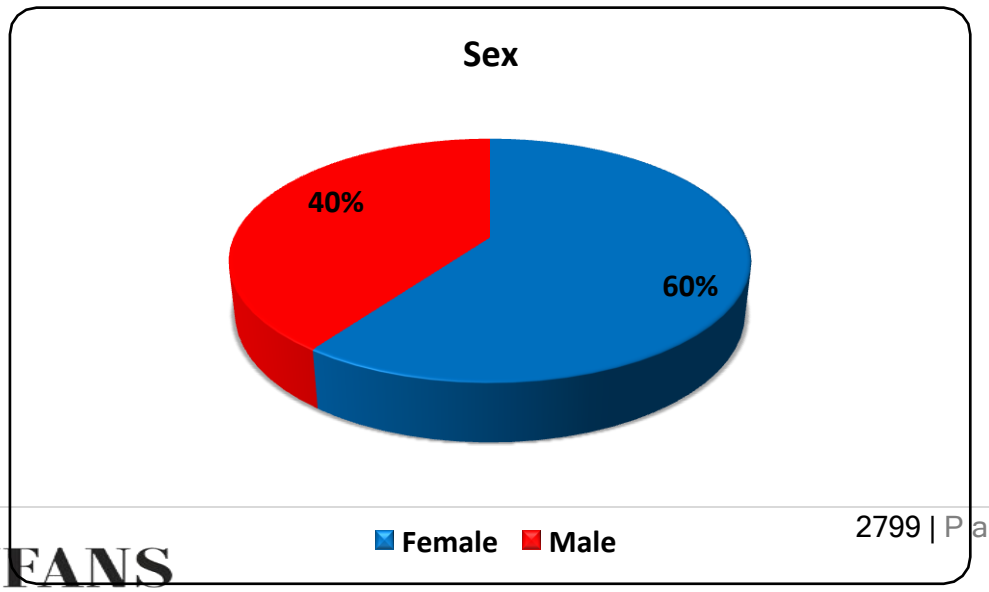


Fig.1: Distribution of patients according to their age group

Sex:
There were 54 females (60%) and 36 males (40%) in our study. Male tofemale ratio was 0.67: 1.

Table 3: Gender wise distribution of patients

Gender	Frequency	%
FEMALE	54	60
MALE	36	40
TOTAL	90	100



ISSN PRINT 2319 1775 Online 2320 7876

Research paper UGC CARE Listed (Group -I) Journal Volume 11, S Iss 3, Dec 2022

Fig. 2: Gender wise distribution of patients

Symptoms:
Most common symptom was seizures seen in 44 patients (48.88%), followed by headaches in 32 patients (35.56%), motor neuro-deficit in 20 patients (22.22%), giddiness in 11 patients (12.22%), vomiting in 5 patients (5.56%), fever in 4 patients (4.44%) & h/o TB in one patient (1.11%).

Table 4: Distribution of patients according to their symptoms

	NUMBER	%
SEIZURES	44	48.88
HEADACHE	32	35.56
MOTOR NEURODEFICIT	20	22.22
GIDDINESS	11	12.22
VOMITING	5	5.56
FEVER	4	4.44
TB	1	1.11
OTHERS	11	12.22

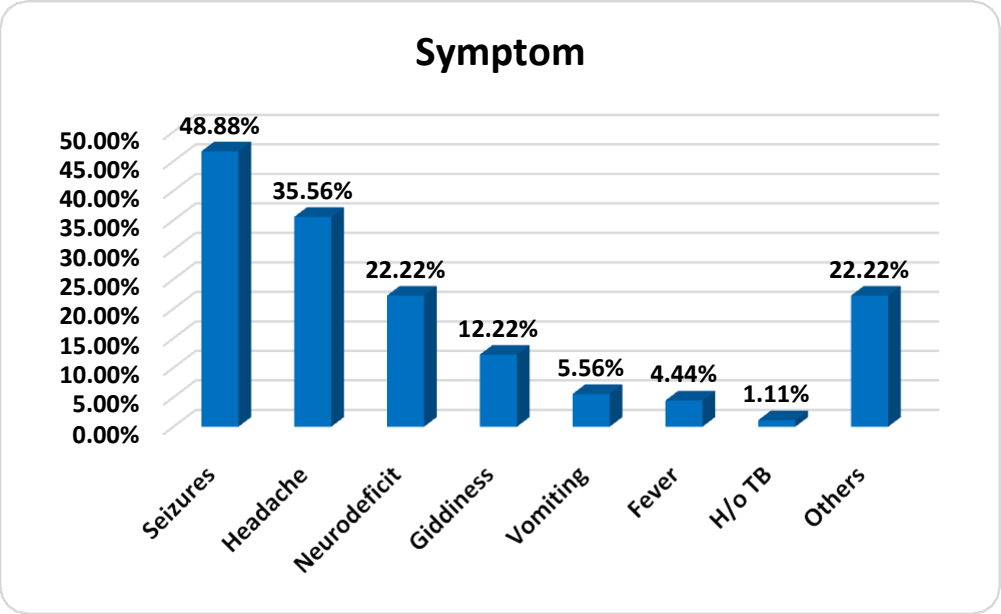


Fig. 3: Distribution of patients according to their symptoms

DISCUSSION
The mean age observed in our study was 48.37 ± 14.64 years. Most common age group decade was 41-50 years with 27 patients (30%), followed by 51 — 60 years with 16 patients (17.78%) and 41-50years with 16 patients (17.78%).

Most common symptom was seizures seen in 42 patients (46.67%), followed by headaches in 32 patients (35.56%), Neurodeficit in 20 patients (22.22%), giddiness in 11 patients (12.22%), vomiting in 5 patients (5.56%), fever in 4 patients (4.44%) & H/O TB in one patient (1.11%).

Midline tumours were seen in 94 (24.3%) patients, out of which 55 patients (58.5%) had supratentorial and rest 39 patients (4 1.5%) had posterior fossa masses. Glioma was the commonest tumour seen in their study, which was followed by infective lesions, meningiomas, pituitary tumours,

ISSN PRINT 2319 1775 Online 2320 7876

Research paper UGC CARE Listed (Group -I) Journal Volume 11, S Iss 3, Dec 2022

accoustic neuroma and others.

Majority were intra axial lesions seen in 51 out of 90 patients (56.67%), rest39 patients (43.33%) had extra axial lesions.

B Karpagam et al.[88] also found similar results with majority of the patients having intraaxial lesions (68%) as compared to extra axial lesions (32%).

Kaki RR et al. [89]found that out of 50 patients under their study, there were 31intra axial (62%) and 19 extra axial (38%) lesions. Majority of the lesions were solid seen in 64 patients (71.11%), some were solid- cystic in 14 patients (15.56%) and few were cystic in 12 patients (13.33%). Calcification was seen in 18 patients (20%).

Majority of the patients, 57 out of 90 (63.33%) were negative for HMG /Necrosis, 21 patients were positive for HMG (23.33%), 7 patients were positive for HMG /Necrosis (7.78%) and 5 patients had necrosis (5.56%).

Mass effect was present in 61 patients (67.78%), while it was absent in the rest 29 patients (32.22%).

Edema was present in 65 patients (72.22%) and absent in 25 patients (27.78%).

On T1W, most commonly patients had heterogeneously hypointense imagesin 60 out of 90 patients (66.67%), some patients (26 patients) had isointense images (28.89%), while few patients, 4 had hyperintense images (4.44%).

On T2W images, major patients were seen heterogenously hyperintense seenin 74 out of 90 (82.22%), 10 patients showed Isointense images (11.11%) & 6 patients showed hypointense (6.67%).

Diffusion restriction was seen in 21 patients (23.33%), while it was absent in69 patients (76.67%).

Post contrast enhancement was seen in 61 patients (67.78%), peripheralenhancement was seen in 17 patients (18.89%), it was absent in 9 patients (10%) & ring enhancement seen in 3 patients (3.33%).

The most common category of lesion was primary neoplasm seen in 45patients (50.00%) followed by secondary neoplasm in 22 patients (24.44%). Rest 23 patients had other diagnosis (25.56%) which included conditions like parasitic infections, inflammatory and benign lesions each seen in 5 patients (5.56%). some less common lesions were vascular seen in 2 patients (2.22%) and demyelinating seen in 1 patient (1.11%).

Associations

There was no any significant association between the different categories of lesions and sex, age, presence of symptoms like fever, vomiting and neurodefecit (all $p > 0.05$).

Significant association is seen between the lesions being single or multiple & type of lesion. Primary neoplasms are found more to be single (44 of 45 — 97.78%) as compared to secondary neoplasms (5 of 22 – 22.73%) and other lesions. (13 of 23 – 56.52%).

Side of lesion and type of lesion showed significant association, where secondary neoplasms (17 out of 22 — 77.27%) and other lesions (14 out of 23-60.87%) are seen more on the midline or Bilaterally as compared to primary lesions which were more on the right (44.44%) and left side (24.44%)of the brain than midline (31.11%). There was significant association seen between the side of the lesions & type of lesion ($p < 0.0001$).

ISSN PRINT 2319 1775 Online 2320 7876

Research paper UGC CARE Listed (Group -I) Journal Volume 11, S Iss 3, Dec 2022

All the lesions were more solid in nature, but primary neoplastic lesions were also showing some lesions of solid- cystic lesions (10 out of 45 — 22.225) which were more compared to seen in secondary neoplasms (2 of 22 — 9.09%) and others (1 of 23 — 4.35%). This difference in the solid /cystic characteristic of lesions was significant (p = 0.021).

Calcification was seen in only primary neoplasms and other lesions, it was absent in secondary neoplasms. Primary neoplasms showed more calcification (34 out of 45 — 75.56%) as compared to other lesions (16 out of 23 — 69.57%). There was significant difference between the different type of lesions and calcification (p = 0.022).

No significant difference was seen between the lesion types and HMG /Necrosis. (p = 0.084)

Mass effect was seen more in primary neoplasms (38 out of 45 – 84.44%) as compared to secondary neoplasms (13 out of 22 – 59.09%) or others (10 out of 23 – 43.48%). There was significant difference seen between the presence or absence of mass effect and the type of lesion (p = 0.002).

There was significant difference between the T1W appearance of the images seen on MRI of different lesions, all type of lesions were seen more Hypo while primary neoplasms were also having to be seen as iso images (20 out of 45 — 44.44%) as compare to other types of lesions. (p = 0.002)

All lesions were seen mostly as hyper images on T2W MRI as compared to primary neoplasms, some of which were also seen as iso images (8 out of 45

- 17.78%) being significantly more than other lesion types (p = 0.009).

No Significant difference was seen in Diffusion Restriction amongst different lesion types.
Significant association was seen between the post contrast results of the different lesion types (p < 0.0001). It was seen more absent in other lesions (8 out of 23 — 3478%) as compared to primary neoplasms (2.22%) and not seen in secondary (0%). It was present in 5 other type of lesions (21.74%), 39 of primary lesions (86.67%) and 15 of the secondary neoplasms (68.18%).

- sms (68.18%).

CONCLUSION

"MRI has helped in the early and quick diagnosis and localisation of the ICSOL and in complement with advanced neurosurgical techniques have brightened the prognosis of mass lesions and their treatment."

"For diagnosing and evaluation of intracranial space occupying lesion with a reasonable degree of the diagnostic accuracy, magnetic resonance imaging still remains the first line investigation along with the advancement of newer modifications of MRI such as MR spectroscopy and the newer techniques like MR perfusion."

ISSN PRINT 2319 1775 Online 2320 7876

Research paper UGC CARE Listed (Group -I) Journal Volume 11, S Iss 3, Dec 2022

IMAGES

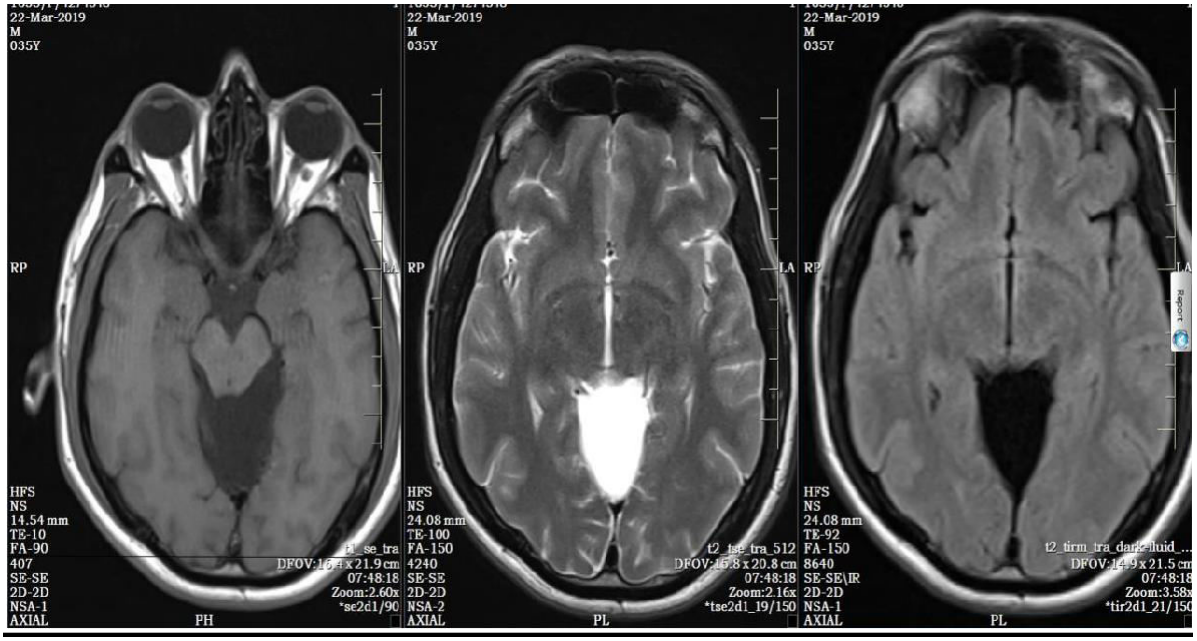


Illustration 1: A 35 years old male with complaints of giddiness.
AxialT1W, T2W and FLAIR images show a well defined cystic lesion of CSF signal intensity in infratentorial region involving superior cerebellar cistern and quadrigeminal plate cistern causing mass effect on the left side of mid brain posteriorly suggestive of **Superior cerebellar arachnoid cyst.**

ISSN PRINT 2319 1775 Online 2320 7876

Research paper UGC CARE Listed (Group -I) Journal Volume 11, S Iss 3, Dec 2022

BIBLIOGRAPHY

1. Anne G. Osborn- Diagnostic Neuroradiology, copyright 1994 by Mosby. 127-130
2. Bourgouin PM, Tampieri D, Grohovac SZ et al. : CT and MR imagingfindings in adults with cerebellar medulloblastoma. Comparision with findings in children. AJR 1992;609-612.
3. Lee SH MD, Krishna CVG Rao MD: Cranial MRI and CT, Ed. 3,1992.
4. John Haga, CT & MRI evaluation of whole body, Third edition, 1994 and Fourth edition.
5. Goyani BR, Ukani BV, Naik P, Vadel HB, Sheth R. A study on role of magnetic resonance imaging (MRI) in intracranial space occupying lesions. Headache. 2015 Jan;36:51-42.
6. Butt M.E, Khan S.A, Chaudhary NA, Qureshi GR. Intracranial space occupying lesions a morphological analysis. E:/biomedica 2005;21:31-5. Available from: 888http://www.thebiomedicapk.com/ articles/31.pdf.
7. Rathod V, Bhole A, Chauhan M, Ramteke H, Wani B: Study ofclinico-radiological & clinico-pathological correlation of intracranialspace occupying lesion at rural center. The Internet Journal of Neurosurgery. 2010;7(1). doi: 10.5580/ba1.
8. Mahmoud MZ. Intra Cranial Space Occupying Lesions In Saudi Patients Using Computed Tomography. Asian J Med Radiol Res 2013 ;1 (1): 25-8. Available from: http://www.researchgate.net/ publication / 236869508.
9. Irfan A, Qureshi A. Intracranial space occupying lesions-review of 386 cases. JOURNAL-PAKISTAN MEDICAL ASSOCIATION. 1995 Dec;45:319-.
10. Mark Wiles. Brain tumours. In: Sir John Walton, editor. Brain Diseases of the Nervous System, 4th ed. Oxford: Oxford Medical Publications; 1985.p. 143-172.

ISSN PRINT 2319 1775 Online 2320 7876

Research paper UGC CARE Listed (Group -I) Journal Volume 11, S Iss 3, Dec 2022

11. McKinney PA. Brain tumours: incidence, survival, and aetiology. J Neurol Neurosurg Psychiatr 2004;75: ii12.
12. Robert HA. Brain tumours in children. In: Richard E, Bhrman S, Robert M, Kliegman Z, Jenson HB, editors. Nelson Text Book of Pediatrics, 16th ed. Philadelphia: WB. Saunders; 2000.p1862.1859
13. Teddy PJ. Intracranial tumours. In: Weatherall DJ, Ledingham JGG, Warrell DA, editors. Oxford Textbook of Medicine, 3rd ed. Oxford: Medical Publication; 1996.p.4029-4040.
14. Abu-Salih HS, Abdul-Rahaman AM. Tumors of the brain in the Sudan. Surg Neurology 1988; 29(3):199-96.
15. Bella ME. Elsherif. The Pattern of Malignant disease in children presenting to Khartoum hospitals. MD Thesis; University of Khartoum, Sudan. 1982-1983.p132.
16. Jamjoom ZAB. Pattern of intracranial space-occupying lesions: the experience at King Khalid University Hospital. Ann Saudi Med 1989;9: 3-10.
17. Wanyoike PK. Posterior cranial fossa tumours in children at Kenyatta National Hospital, Nairobi. East Afr Med J 2004;81(5):258- 60.
18. Mwang'ombe NJ, Ombachi RB. Brain tumours at the Kenyatta National Hospital, Nairobi. East Afr Med J 2000; 77(8):444-7.
19. Desai KI, Nadkarni TD, Muzumdar DP. Prognostic factors for cerebellar astrocytomas in children: a study of 102 cases. Pediatr Neurosurg 2001; 35(6):311-7.
20. Coleman MP, Babb P, Damiecki D. Cancer survival trends in England and Wales, 1971–1995: deprivation and NHS region. Studies in medical and population subjects No. 61. London: Stationery Office; 1999.p713-714.
21. Wrensch M, Minn Y, Chew T. Epidemiology of primary brain tumours: current concepts and review of the literature. Neurooncology 2002; 4: 278–99.
22. Davis FG, McCarthy BJ. Current epidemiological trends and surveillance issues in brain tumours. Expert Rev Anticancer Ther 2001; 1: 395–401.
23. Al-Mefty O, Al-Rodhan NRF, Phillips RL. Factors affecting survival of children with malignant gliomas. Neurosurgery 1987;20: 416-20.
24. Tomita T. Pediatric brain tumours, overall review. Chicago, Illinois. J Child Neurol 1995; 20(1): 283-88.
25. Nicholas AV. Intracranial tumours and state of altered intracranial pressure. In: Clude JB, Plum F, editors. Cecil Text Book of Medicine,20th ed. Philadelphia: WB Saunders; 1996.p. 2125-2132.
26. Schliamser SE. Intracranial abscesses in adults: an analysis 54 consecutive cases. Scand J Infect Dis 1988; 20(1):1-9.
27. Yechoor VK, Shandera WX, Rodriguez P. Tuberculous meningitis among adults with and without HIV infection. Arch Int Med 1996; 156: 1710-716.
28. Green PH. Tubercular meningitis. Lancet 1836; ii: 232-35.
29. Rich AR, McCordick HA. The pathogenesis of tuberculous meningitis. Bulletin 1933; 52: 5-37.
30. Jenkins PA. The microbiology of tuberculosis. In: PDO Davies, editor. Clinical tuberculosis. London: Chapman and Hall Medical;1994.p.743.
31. Lincoln EM, Sordillo SUR, Davies PA. Tuberculous meningitis inchildren. J Paediatr 1960; 57: 807-23
32. Donald, JF Schoeman, Van Zyl LE. Intensive short course chemotherapy in the management of tuberculous meningitis. Int J Tuberclung Dis 1998; 2: 704-11.
33. Illingworth RS. Miliary and meningeal tuberculosis. Lancet 1996;ii:646-49.
34. Leonard J, Des Prez RM. Tuberculous meningitis. Infect Dis Clin North Am 1990; 4:769-87.
35. Dube MP, Holtom PD, Larsen RA. Tuberculous meningitis in patients with and without human immunodeficiency virus infection. Am J Med 1992; 93:520-24.

ISSN PRINT 2319 1775 Online 2320 7876

Research paper UGC CARE Listed (Group -I) Journal Volume 11, S Iss 3, Dec 2022

36. Smith HV, Vollum RL. The diagnosis of tuberculous meningitis. Br Med Bull 1954; 10:140-44.
37. Jeren T, Beus I. Characteristics of cerebrospinal fluid in tuberculous meningitis. Acta Cytol 1982; 26:678-80.
38. Ribera E, Martinez-Vazquez JM, Ocana I. Activity of adenosine deaminase in cerebrospinal fluid for the diagnosis and follow-up of tuberculous meningitis in adults. J Infect Dis 1987; 155:603-7.
39. Andrade ZA. The situation of hepatosplenic schistosomiasis in Brazil today. Mem Inst Oswaldo Cruz 1998; 93 (suppl. 1): 313-16,
40. Pittella JE, Lana-peixoto MA. Brain involvement in hepatoesplenic schistosomiasis mansoni. Brain 1981; 104: 621-32.
41. Mahmoud AAF. Trematodes (schistosomiasis) and other flukes. In: Mandell GL, Bennett JE, Dolin R, editors. Mandel, Douglas, and Bennett's Principles and Practice of Infectious Diseases, 5th ed.New York: Churchill Livingstone; 2000. p. 2950-953.
42. Ferrari TC. Involvement of central nervous system in the schistosomiasis. Mem Inst Oswaldo Cruz 2004; 99 (suppl 1): 59- 62.ttella JE. Neuroschistosomiasis. Brain Pathol 1997;7:649-62.
43. Lesprit, P, Adle-Biasette H, Liance M. Photo quiz: cerebral s. mansoni infection. Clin infect Dis 2001; 32: 1347-371.
44. Urban CD, Piovesan EJ, Almeida SM. Acute schistosomiasis with the brain involvement: case report. Arq Neuropsiquiatr 1996; 54(4): 677- 82.
45. Nascimento-Carvalho CM, Moreno-Carvalho OA. Clinical and cerebrospinal fluid findings in patients less than 20 years old with a presumptive diagnosis of neuroschistosomiasis. J Trop Paediatr 2004; 50: 98-100
46. Braga BP, Costa-Júnior LB, Lambertucci JR. Magnetic resonance imaging of cerebellar schistosomiasis mansoni. Rev Soc Bras Med Trop 2003; 36: 635-36.
47. Galassi E, Piazza G, Giast G, Frank F. Arachnoid cysts of the middle fossa: a clinical and radiological study of 25 cases treated surgically. Surg Neurol 1980; 14: 211-19.
48. Baurer BL, Hellweek P, Henry H S, William HS. Arachnoid cyst. In: Behrman S, Richard E, Hal B. Arachnoid Cyst in Operative Neurosurgical Techniques, 3rd. New York: WB Saunders; 1995:586- 89.
49. Guerina NG, Hsu HW, Meissner HC, Maguire JH. Neonatal serologic screening and early treatment for congenital Toxoplasma gondii infection. N Engl J Med 1994; 330:1858-863.
50. Jara M, Hsu HW, Eaton RB, Demaria A Jr. Epidemiology of congenital toxoplasmosis identified by population-based newborn screening in Massachusetts. Pediatr Infect Dis J 2001; 20:1132-135
51. Altinors N, Bavbek M, and Caner HH, Erdogan B. Central nervous system hydatidosis in Turkey: a cooperative study and literaturesurvey analysis of 458 cases. J Neurosurg 2000; 113: 1-8.
52. Beggs I. The radiology of hydatid disease. Am J Roentgenol 1985; 145(3): 639-48.
53. Lunardi P, Missori P, Di Lorenzo N, Fortuna A. Cerebral hydatidosis in childhood: a retrospective survey with emphasis on long termfollow up . Neurosurgery 1991; 29:515-18.
54. Krajewski R, Stelmasiak Z. Cerebral Hydatid Cyst in Children.Childs Nerv Syst1991 Jun; 7(3):154-5.
55. Robinson RG. Hydatid disease affecting the nervous system. Ann R Coll Surg Eng 1960; 26:146-56.
56. Arbab MA, Idris MN, Sokrab TE, Saeed ES, Ali GM, Tariq MY, et al. Clinical presentation and CAT scan findings in mycetoma of thehead. East Afr Med J 1998; 75(4):243-45.
57. Bein-Schumacher M, Thorn-Klein HJ, Hassler W. Neuroradiological diagnosis and clinical aspects of giant intracranial aneurysms. Radiology 1986; 26(1):515-19.
58. Leach J, Kerr R. Elective neurosurgery,in, bailey & love's (ed), Short practice of surgery, 25 (U.K: Edward Arnold, 2008) 623-44.

ISSN PRINT 2319 1775 Online 2320 7876

Research paper UGC CARE Listed (Group -I) Journal Volume 11, S Iss 3, Dec 2022

59. Miabi Z, Mashrabi O. Pediatric brain tumor. Res J Biol Sci 2009;4(6):647-50. doi: 10.3923/rjbsci.2009.647.650.
60. Kieffer SA, Brace JR. Intracranial Neoplasms, in, Hagga JR (ed). CT & MRI of the whole body, 5 (Philadelphia: Mosby 2009) 49-144.
61. Tilgner J, Herr M, Ostertag C, Volk B. Validation of intraoperative diagnoses using smear preparations from stereotactic brain biopsies: intraoperative versus fi nal diagnosis – infl uence of clinical factors. Neurosurgery 2005; 56: 257–265.
62. Raichle ME. A paradigm shift in functional brain imaging. Journal of Neuroscience. 2009 Oct 14;29(41):12729-34.
63. Bandettini PA, Wong EC, Hinks RS, Tikofsky RS, Hyde JS (1992) Time course EPI of human brain function during task activation.Magn Reson Med 25:390 –397.
64. Frahm J, Bruhn H, Merboldt KD, Ha¨nickeW (1992) Dynamic MR imaging of human brain oxygenation during rest and photic stimulation. J Magn Reson Imaging 2:501–505
65. Kwong KK, Belliveau JW, Chesler DA, Goldberg IE, Weisskoff RM, Poncelet BP, Kennedy DN, Hoppel BE, Cohen MS, Turner R (1992) Dynamic magnetic resonance imaging of human brain activity during primary sensory stimulation. Proc Natl Acad Sci U S A 89:5675– 5679.
66. Ogawa S, Tank DW, Menon R, Ellermann JM, Kim SG, Merkle H, Ugurbil K (1992) Intrinsic signal changes accompanying sensory stimulation: functional brain mapping with magnetic resonance imaging. Proc Natl Acad Sci U S A 89:5951–5955
67. Marder E, Goaillard JM (2006) Variability, compensation and homeostasis in neuron and network function. Nat Rev Neurosci 7:563–574.
68. Kuhn TS (1996) The Structure of scientific revolutions, Ed 3. Chicago: University of Chicago. p 86 – 92.
69. Al-Okaili RN, Krejza J, Wang S, Woo JH, Melhem ER. Advanced MR imaging techniques in the diagnosis of intraaxial brain tumors in adults. Radiographics. 2006 Oct;26(suppl_1):S173-89.
70. Heckmann JG, Bockhorn J, Stolte M, Druschky A, Neundörfer B. An instructive false diagnosis: steroid-induced complete remission of a CNS tumor--probably lymphoma. Neurosurg Rev 1998; 21: 48-51]
71. Bromberg JE, Siemers MD, Taphoorn MJ. Is a ―vanishing tumor‖ always a lymphoma? Neurology 2002; 59: 762-764
72. Krogh-Jensen M, D'Amore F, Jensen MK, Christensen BE, Thorling K, Pedersen M, Johansen P, Boesen AM, Andersen E.Clinicopathological features, survival and prognostic factors of primary central nervous system lymphomas: trends in incidence of primary central nervous system lymphomas and primary malignant brain tumors in a well-defined geographical area. Population-based data from the Danish Lymphoma Registry, LYFO, and the Danish Cancer Registry. Leuk Lymphoma 1995; 19: 223-233
73. Okita Y, Narita Y, Miyakita Y, Ohno M, Fukushima S, Maeshima A, Kayama T, Shibui S. Long-term follow-up of vanishing tumors in the brain: how should a lesion mimicking primary CNS lymphoma be managed? Clin Neurol Neurosurg 2012; 114: 1217-1221
74. Packer RJ, Cohen BH, Cooney K et-al. Intracranial germ cell tumors. Oncologist. 2000;5 (4): 312-20.
75. Kaye AH. Essential neurosurgery. Wiley-Blackwell. (2005) ISBN:1405116412, p – 45-48.
76. Kornienko VN, Pronin IN. Diagnostic Neuroradiology. Springer Verlag. (2009) ISBN:3540756523.
77. Govindaraju V, Young K, Maudsley AA. Proton NMR chemical shifts and coupling constants for brain metabolites. NMR Biomed 2000;13: 129-153
78. Aasly J, Silfvenius H, Aas TC, Sonnewald U, Olivecrona M, Juul R, White LR. Proton magnetic resonance spectroscopy of brain biopsies from patients with intractable epilepsy. Epilepsy

ISSN PRINT 2319 1775 Online 2320 7876

Research paper UGC CARE Listed (Group -I) Journal Volume 11, S Iss 3, Dec 2022

Res 1999; 35:211–217.
79. Abeloff MD, Armitage JO, Lichter AS. Clinical oncology. 3rd ed.Edinburgh, UK: Churchill Livingston; 2004. 20. 72–77.
80. Angelie E, Bonmartin A, Boudraa A, Gonnaud PM, Mallet JJ, Sappey-Marinier D. Regional differences and metabolic changes in normal aging of the human brain: proton MR spectroscopic imaging study. Am J Neuroradiol 2001; 22:119–127.
81. Tilgner J, Herr M, Ostertag C, Volk B. Validation of intraoperative diagnoses using smear preparations from stereotactic brain biopsies: intraoperative versus final diagnosis—influence of clinical factors. Neurosurgery. 2005 Feb 1;56(2):257-65.
82. Perez-Cruet MJ, Adelman L, Anderson M, Roth PA, Ritter AM, Saris SC. CT-guided stereotactic biopsy of nonenhancing brain lesions. Stereotactic and functional neurosurgery. 1993;61(3):105-17.
83. Firlik KS, Martinez AJ, Lunsford LD. Use of cytological preparations for the intraoperative diagnosis of stereotactically obtained brain biopsies: a 19-year experience and survey of neuropathologists. Journal of neurosurgery. 1999 Sep 1;91(3):454-8.
84. Ostertag CB, Mennel HD, Kiessling M: Stereotactic biopsy of brain tumors. Surg Neurol 14:275–283, 1980.
85. Ryken TC, Hitchon PW, Roach RM, Vincent TC: Infratentorial stereotactic biopsy: A review of 11 cases. Stereotact Funct Neurosurg 59:111–114, 1992.
86. Benabid A, Blond S, Chazal J, Cohadon F, Daumas-Duport D, Delisle M, Farnarier P, Franck F, Gherardi M, Gontier M. Stereotaxic
biopsies (SB) of intracranial neoplasms. Considerations apropos of 3,052 cases. Neuro-Chirurgie. 1985;31(4):295-301.
87. Plunkett R, Allison R, Grand W. Stereotactic neurosurgical biopsy is an underutilized modality. Neurosurgical review. 1999 Oct 1;22(2- 3):117-20.
88. Karpagam B, Vadanika V. Brain Space Occupying Lesions by Magnetic Resonance Imaging: A Prospective Study. INTERNATIONAL JOURNAL OF SCIENTIFIC STUDY. 2015 Nov 1;3(8):123-7.
89. Kaki RR, Anuradha B, Rani BS, Rao KS, Kusumalatha P, Satyasuneetha K. IMAGING OF INTRACRANIAL SPACE OCCUPYING LESIONS-A PROSPECTIVE STUDY IN A TERTIARY CARE CENTRE-GGH, KAKINADA, AP.
90. Mahal GB, Neki NS. Role of MRI in the Evaluation of Space Occupying Lesions of the Brain. Int. J. Curr. Res. Med. Sci. 2018;4(1):143-57