"COMPARISION OF T1 FAT SUPPRESSED (FS) POSTCONTRAST AND T2 FLUID ATTENUATED INVERSION RECOVERY (FLAIR) POSTCONTRAST SEQUENCES IN EVALUATION OF INTRACRANIAL LESIONS"

By

Dr. SAHANA N. GOWDA

DISSERTATION SUBMITTED TO SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, KOLAR, KARNATAKA

In partial fulfilment of the requirements for the degree of

DOCTOR OF MEDICINE
IN
RADIODIAGNOSIS

Under the Guidance of

Dr. N. RACHEGOWDA, M.D., DMRD.

PROFESSOR & HOD,

DEPARTMENT OF RADIODIAGNOSIS

DEPARTMENT OF RADIODIAGNOSIS,

SRI DEVARAJ URS MEDICAL COLLEGE,

TAMAKA, KOLAR – 563 101.

2021

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Dr. SAHANA N. GOWDA
Postgraduate in Radiodiagnosis,
Sri Devaraj Urs Medical College,
Tamaka, Kolar.

Date:

Place: Kolar.

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR, KARNATAKA

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Dr. N. RACHEGOWDA, M.D., DMRD.

Professor & HOD,
Department of Radiodiagnosis,
Sri Devaraj Urs Medical College,
Tamaka, Kolar.

Date:

Place: Kolar.

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH TAMAKA, KOLAR, KARNATAKA

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Dr. N. RACHEGOWDA, M.D., DMRD.

Professor & HOD,

Department of Radiodiagnosis,

Sri Devaraj Urs Medical College,

Tamaka, Kolar.

Date:

Place: Kolar.

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION

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Dr. N. RACHEGOWDA,
Professor & HOD,
Department Of Radiodiagnosis,
Sri Devaraj Urs Medical College,
Tamaka, Kolar.

Date:

Place: Kolar.

Dr. P. N. SREERAMULU,
Principal,
Sri Devaraj Urs Medical College,
Tamaka, Kolar.

Date:

Place: Kolar.

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AND RESEARCH, TAMAKA, KOLAR, KARNATAKA

ETHICAL COMMITTEE CERTIFICATE

This is to certify that the Ethical committee of Sri Devaraj Urs Medical College, Tamaka, Kolar has unanimously approved

Dr. SAHANA N. GOWDA

Post-Graduate student in the subject of Radiodiagnosis

at Sri Devaraj Urs Medical College, Kolar

to take up the Dissertation work entitled

"COMPARISION OF T1 FAT SUPPRESSED (FS) POSTCONTRAST AND T2 FLUID ATTENUATED INVERSION RECOVERY (FLAIR) POSTCONTRAST SEQUENCES IN EVALUATION OF INTRACRANIAL LESIONS"

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Postgraduate in Radiodiagnosis,

Sri Devaraj Urs Medical College,

Tamaka, Kolar.

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Dr. SAHANA N. GOWDA

Postgraduate in Radiodiagnosis,
Sri Devaraj Urs Medical College,
Tamaka, Kolar.

LIST OF ABBREVIATIONS

ACA – Anterior cerebral artery

ADC – Apparent diffusion co-efficient

AG – Angiocentric glioma

AICA – Anterior inferior cerebellar artery

CE – Contrast enhanced

CN – Cranial nerve

CNS – Central nervous system

CPC – Choroid plexus carcinoma

CPP – Choroid plexus papilloma

CSF – Cerebrospinal fluid

CT – Computed tomography

DWI – Diffusion weighted imaging

EVN – Extra ventricular neurocytoma

FLAIR – Fluid attenuated inversion recovery

FS – Fat suppressed

GCT – Granular cell tumor

Hypo – Hypointense

ICSOL – Intracranial space occupying lesion

Iso – Isointense

JXG – Juvenile xanthogranuloma

LCH – Langerhans cell histiocytosis

MCA – Middle cerebral artery

MR – Magnetic resonance

MRI – Magnetic Resonance Image

MRS – Magnetic resonance spectroscopy

NAA – N-acetylaspartate

NCC – Neurocyticercosis

NF – Neurofibroma

OPG – Optic pathway glioma

PA – Pilocytic astrocytoma

PC – Postcontrast

PCA – Posterior cerebral artery

PICA – Posterior inferior cerebellar artery

PTPR – Papillary tumor of pineal region

PXA – Pleomorphic xanthoastrocytoma

SD – Standard deviation

SFT – Solitary fibrous tumor

SWI – Susceptibility weighted imaging

TI – Time to inversion

WHO – World health organisation

WI – Weighted imaging

ABSTRACT

Background: T2 FLAIR postcontrast (PC) sequence can provide additional information with regards to evaluation of pyogenic abscesses, perilesional edema & intracranial tumors. T2 FLAIR PC is also better at delineating meningeal and cranial nerve diseases[1]. T2 FLAIR PC sequence provides several advantages over T1 FS PC imaging, which includes suppression of the cerebrospinal fluid (CSF) signal, no or minimal enhancement of blood vessels, reduction of phase-shift artifacts derived from enhanced blood vessels or dural sinuses, and better detection of peritumoral edema making lesions more conspicuous[1]. Therefore, T2 FLAIR PC sequence could be considered as an adjunct over T1 FS PC sequence in evaluation of various intracranial pathologies[2].

Purpose: The objectives of the study were to determine if T2 FLAIR postcontrast sequence is superior to T1 FS postcontrast sequence in evaluation of intracranial lesions and to compare the utility of T2 FLAIR postcontrast sequence when combined with T1 FS postcontrast sequence in evaluation of intracranial lesions.

Material and Methods: This prospective observational study was conducted over a period of eighteen months from January 2019 to June 2020 on 64 patients who underwent plain and gadolinium enhanced MRI brain. Baseline data and MRI sequences including T1 and T2 axial, T1 sagittal, T2 coronal, FLAIR axial, diffusion weighted imaging (DWI), and susceptibility weighted imaging (SWI) were accuqired. Postcontrast sequences obtained were T1 FS

(axial, coronal and sagittal), T2 FLAIR, and VIBE. Final diagnosis will be obtained by comparing T1 FS postcontrast & T2 FLAIR postcontrast sequences.

Results: There were total of 64 cases with predominantly male patients. Out of 64 cases, intra-axial pathology outweighed extra-axial and meningeal pathologies. In this study, T1 FS PC sequence showed better characterization of intra-axial lesions in 11 cases, extra-axial lesions in 6 cases and meningeal enhancement in 3 cases when compared to T2 FLAIR PC sequence. T1 FS PC sequence showed equal enhancement of intra-axial lesion in 1 case, extra-axial lesion in 2 cases and meningeal enhancement in 1 case when compared to T2 FLAIR PC sequence. T2 FLAIR PC sequence showed better characterization of intra-axial lesions in 28 cases, extra-axial lesions in 7 cases and meningeal enhancement in 17 cases when compared to T1 FS PC sequence.

Conclusion: In our study, we concluded that T2 FLAIR PC sequence acts as an adjuvant to T1 FS PC sequence and plays a major role in characterization of the lesion. Diagnostic accuracy has increased significantly with addition of T2 FLAIR PC sequence to routine MRI protocol. Our study showed that there is significant contribution of T2 FLAIR PC sequence in assessing subtle lesions, extent of perilesional edema, solid component enhancement and in diagnosing meningeal pathology with few limitations.

Keywords: MRI, Gadolinium, T2 FLAIR postcontrast, intracranial lesion, intra-axial lesions, extra-axial lesions, meningeal enhancement, T1 FS postcontrast, parenchymal metastatic lesion.

TABLE OF CONTENTS

Serial No.	TOPIC	Page No.
1	INTRODUCTION	1
2	AIMS AND OBJECTIVES	3
3	REVIEW OF LITERATURE	4
4	MATERIALS AND METHODS	46
5	CLINICAL STUDIES	49
6	RESULTS	51
7	IMAGES	58
8	DISCUSSION	69
9	CONCLUSION	73
10	SUMMARY	75
11	BIBLIOGRAPHY	77
12	ANNEXURES	84

LIST OF TABLES

Table No.	TABLES	Page No.
1	Graphical representation of neural tube derivatives.	7
2	Age distribution.	51
3	Gender distribution.	53
4	Distribution of Intracranial space occupying lesions.	54
5	Degree of enhancement in Intra-axial lesions.	55
6	Degree of enhancement in Extra-axial lesions.	56
7	Degree of Meningeal enhancement.	57

LIST OF FIGURES

Figure No.	FIGURES	Page No.
1	Pictorial representation of derivatives of neural tube.	6
2	Pictorial representation of development of external form of brain.	6
3	Pictorial representation of forebrain, midbrain & hindbrain.	7
4	Pictorial representation of sulci, gyri & lobules – Dorsal aspect.	9
5	Pictorial representation of sulci, gyri & lobules – Ventral aspect.	9
6	Pictorial representation of depicting relation of cranial meninges to brain.	12
7	Pictorial representation of cranial nerves.	12
8	Pictorial representation of cerebral arteries.	13
9	Pictorial representation of penetrating arterial territories.	14
10	Pictorial representation of Circle of Wills.	14
11	Pictorial representation of veins & venous sinuses.	15
12	1.5 Tesla, 16 channel, MR Scanner (Magnetom, Avanto®, Siemens, Erlangen, Germany).	48
13	Graphical representation of Age distribution.	52
14	Graphical representation of Gender distribution.	53

15	Graphical representation of distribution of Intracranial space occupying lesions.	54
16	Graphical representation of degree in enhancement of Intra-axial lesions.	55
17	Graphical representation of degree in enhancement of Extra-axial lesions.	56
18	Graphical representation of degree Meningeal enhancement.	57
19	MRI Brain images of a case of Neurocysticercosis.	58
20	MRI Brain images of a case of Glioblastoma.	59
21	MRI Brain images of a case of Glioblastoma.	60
22	MRI Brain images of a case of Meningioma.	61
23	MRI Brain images of a case of Cerebellopontine angle meningioma.	62
24	MRI Brain images of a case of Neurocysticercosis.	63
25	MRI Brain images of a case of Choroid plexus xanthogranuloma.	64
26	MRI Brain images of a case of Vestibular schwannoma.	65
27	MRI Brain images of a case of Meningoencephalitis.	66
28	MRI Brain images of a case of Epidural abscess.	67
29	MRI Brain images of a case of Disseminated Tuberculosis.	68

INTRODUCTION

INTRODUCTION

Intracranial tumors are a significant health problem. The annual incidence of primary and secondary central nervous system neoplasms ranges from 10 to 17 per 100,000 persons[2]. Brain lesions are areas of abnormal tissue that have been damaged due to injury or disease, which can range from being relatively harmless to life threatening[3].

Imaging plays an integral role in intracranial tumor management. Magnetic resonance (MR) imaging in particular has emerged as the imaging modality most frequently used to evaluate intracranial tumors, and it continues to have an ever-expanding, multifaceted role. In general, the role of MR imaging in the workup of tumors can be broadly divided into tumor diagnosis & classification, treatment planning and post treatment surveillance[2].

T2 Fluid Attenuated Inversion Recovery (FLAIR) image sequence is an inversion recovery sequence that suppresses CSF signal allowing better detection and delineation of extent of intracranial lesions. FLAIR sequence shows mild T1 effect owing to its long TI (time to inversion) and therefore can delineate lesions on postcontrast study. T2 FLAIR post contrast study is highly sensitive sequence in detecting meningeal inflammation, infections and carcinomatosis[1.]

Gadolinium-based intravenous contrast agents are used to improve intracranial lesion detection and characterization on magnetic resonance imaging (MRI) of brain[1,4]. Routine post-gadolinium based contrast administration studies are performed with T1 weighted imaging (WI). Gadolinium causes T1 shortening effect, which makes it stand out on T1WI[1].

AIMS & OBJECTIVES

AIMS AND OBJECTIVES

The aims and objectives of the study were:

1. To determine when T2 FLAIR postcontrast sequence is superior to T1 FS postcontrast sequence in evaluation of intracranial lesions.
2. To compare the utility of T2 FLAIR postcontrast sequence when combined with T1 FS postcontrast sequence in evaluation of intracranial lesions.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

EMBRYOLOGY

At the beginning of 3rd week, the central nervous system (CNS) develops as a slipper-shaped plate of thickened ectoderm, the neural plate. The neural plate is located in the middorsal region in front of the primitive pit. Its lateral edges get elevated and forms neural folds. These neural folds further become more elevated to meet each other at the midline & fuse to form the neural tube.

By the 4th week of gestation, three vesicular dilatations develop at the rostral portion of the neural tube, thereby defining the prosencephalon (forebrain), mesencephalon (midbrain) and rhombencephalon (hindbrain).

By the 5th week of gestation, the developing forebrain divides into a telencephalon (cephalic) and a diencephalon (caudal). The developing hindbrain subdivides into a cephalic metencephalon (cephalic) and a myelencephalon (caudal). Later, the metencephalon becomes the pons and cerebellum while the myelencephalon forms the medulla oblongata.

From the telencephalic end of the neural tube, bilateral diverticula form the cerebral hemispheres. These cerebral hemispheres undergo complex expansion and folding with formation of permanent primitive fissures by the 4th month. The

developing brain divides into cerebrum, cerebellum and spinal cord by three major flexures i.e, the midbrain, pontine and cervical flexures.

Early in development, the cerebral hemispheres are lissencephalic (smooth-surfaced).The germinal matrix forms at about seven weeks of gestational age and involutes at about 28 to 30 weeks, although it persists in the form of focal cell clusters till 36 to 39 weeks. Each lateral ventricle is surrounded by germinal matrix of primitive cells. These cells proliferate, migrate outward to the cortex in an "inside out" sequence and mature as neural and glial cells. During the 6^{th} and 7^{th} fetal months, the cerebral surfaces convolute to form primitive gyri and sulci; therefore, the adult pattern can already be recognized towards the end of gestation. The formation of fiber tracts is concomitant with cortical development, including the commissures between the two cerebral hemispheres[5].

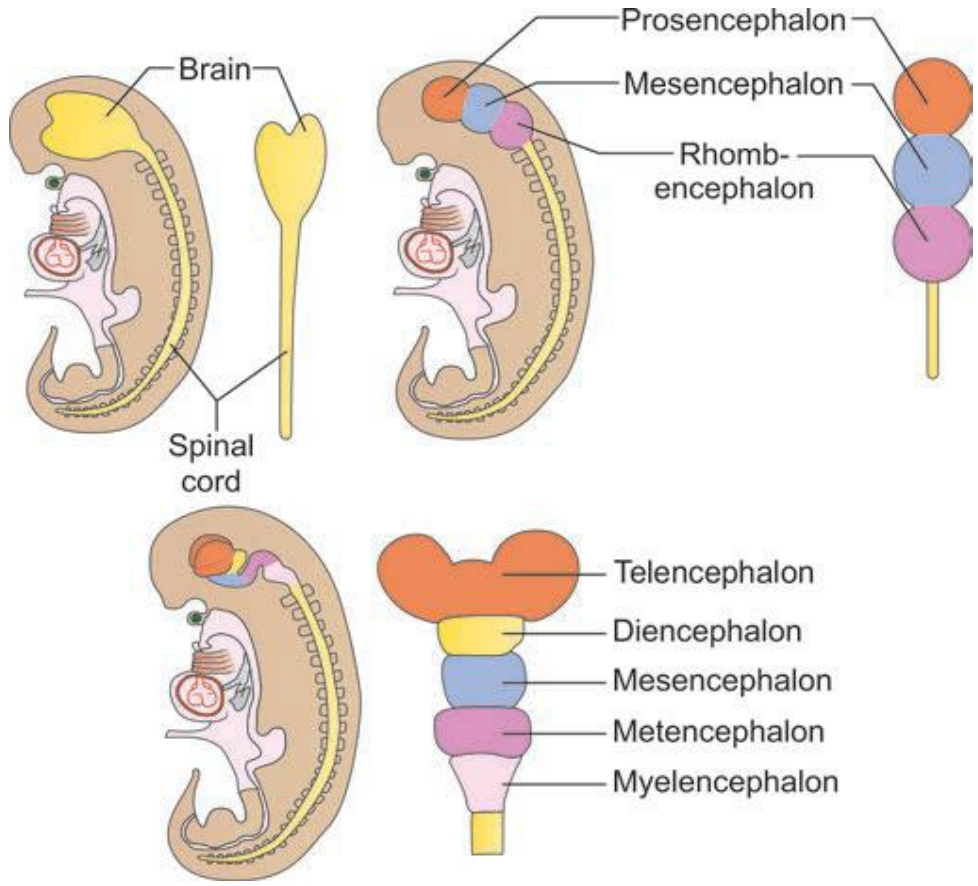

Figure 1: Pictorial representation of derivatives of neural tube.

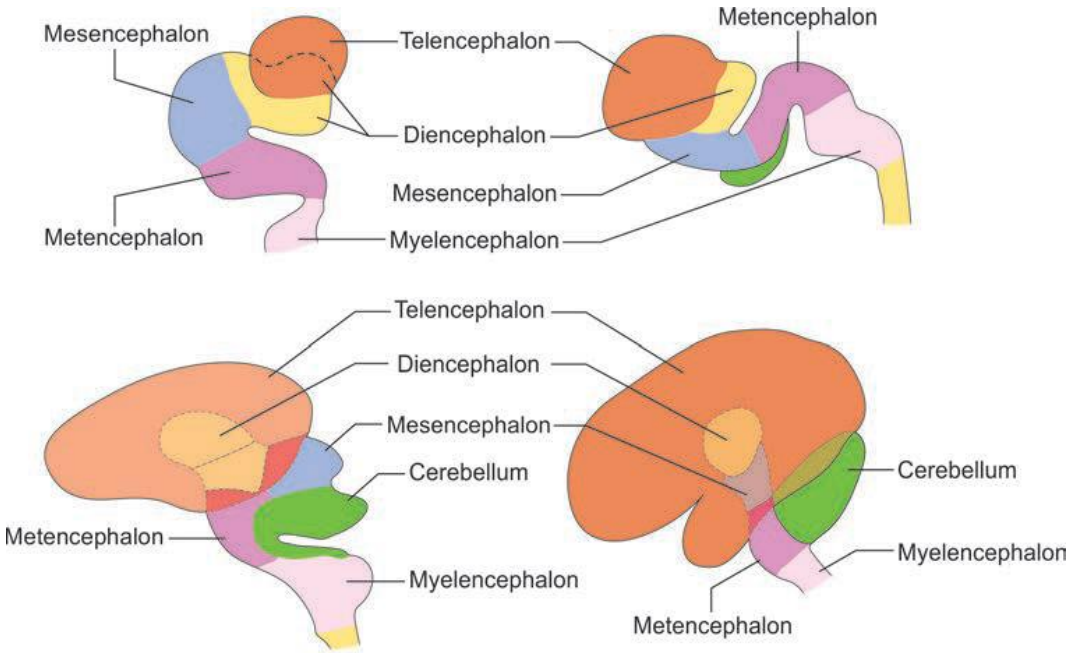

Figure 2: Pictorial representation of development of external form of brain.

Table 1: Neural tube derivatives.

Neural tube subdivisions	Primary brain vesicles	Secondary brain vesicles	Parts of adult brain	Cavities
Brain	Prosencephalon (Forebrain)	Telencephalon	Cerebral hemispheres a. Cerebral cortex b. Corpus striatum i. Caudate nucleus ii. Lentiform nucleus	Lateral ventricles
		Diencephalon	a. Thalamus b. Hypothalamus c. Epithalamus	Third ventricle
	Mesencephalon (Midbrain)	Mesencephalon	Midbrain	Cerebral aqueduct
	Rhombencephalon (Hindbrain)	Metencephalon	a. Pons b. cerebellum	Fourth ventricle
		Myelencephalon	Medulla oblongata	
Spinal cord	Spinal cord	Spinal cord	Spinal cord	Central canal

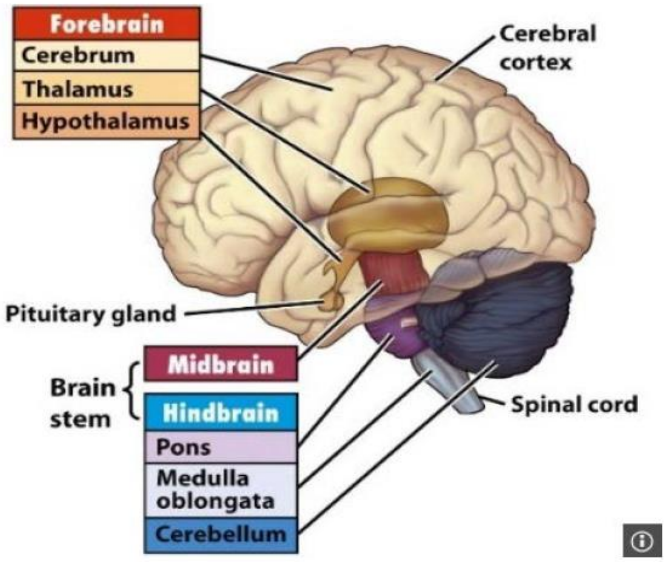

Figure 3: Pictorial representation of forebrain, midbrain and hindbrain.

NORMAL ANATOMY

The contents of the cranial cavity are brain parenchyma, cranial nerves, meninges and CSF spaces. The anatomy of the head is studied in the form of cross-sectional images (axial, sagittal and coronal planes).

Cerebral Hemispheres

The two cerebral hemispheres are separated by the falx cerebri and interhemispheric fissure. Each cerebral hemisphere consists of inner white matter and outer gray matter (cerebral cortex). Sulci and gyri are found on the surface of the cerebral hemispheres. Each hemisphere is divided into five lobes – frontal, parietal, temporal, occipital and insula (central lobe). The central fissure (Rolandic fissure) starts from the superior mid hemisphere and extends antero-inferiorly to separate the frontal lobe from the parietal lobe. On the lateral surface, the sylvian fissure begins anteriorly and extends inferiorly, separating the frontal lobe from the temporal lobe.

Corpus callosum is a band of central white matter that connects two cerebral hemispheres. Genu, body, splenium and rostrum are four parts of the corpus callosum. Basal ganglia represent the central gray matter which consists of caudate nucleus, lentiform nucleus (Globus pallidus and putamen), claustrum and amygdala. The internal capsule is a boomerang shaped thick white matter, consisting of anterior limb, genu and posterior limb. It is bounded laterally by the lentiform nucleus, anteromedially by caudate nucleus and postero-medially bythalamus. The centrum semiovale constitutes the white matter of the cerebral hemispheres.

The diencephalon is made up of thalami, geniculate bodies (medial and lateral), epithalamus, subthalamus and hypothalamus.

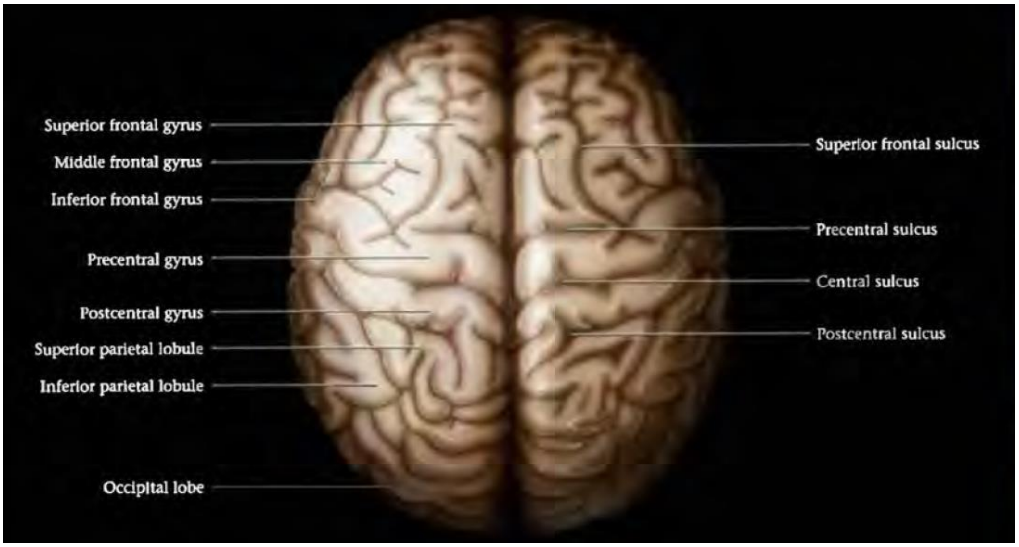

Figure 4: Graphical representation of sulci, gyri and lobules – dorsal aspect.

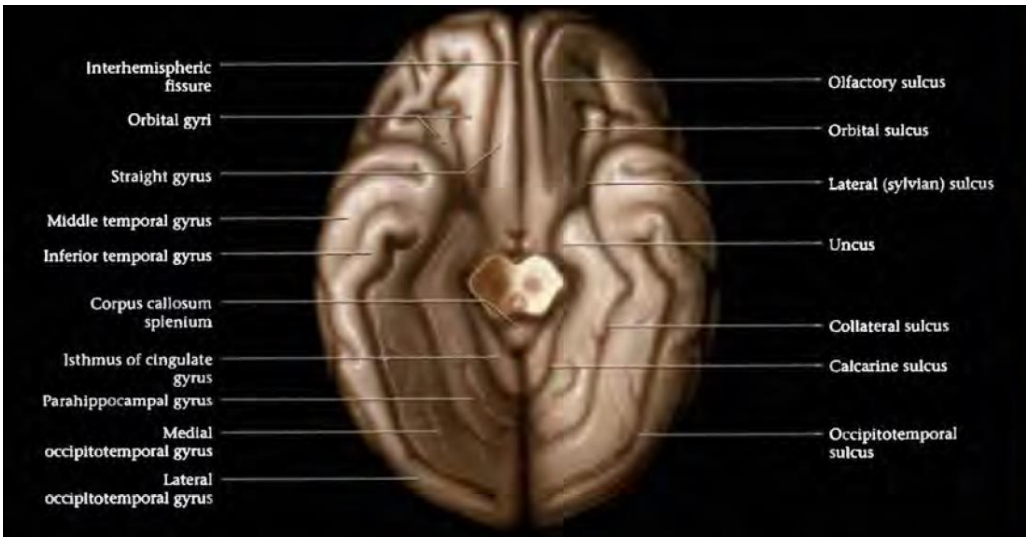

Figure 5: Graphical representation of sulci, gyri and lobules – ventral aspect.

Cerebellum

Cerebellum occupies the greater part of posterior cranial fossa. It is situated posterolateral to the pons and medulla and is separated from these two structures by the fourth ventricle. The cerebellum consists of two hemispheres, which are connected in the midline by vermis and three peduncles, namely superior, middle and inferior, which are connected to midbrain, pons and medulla respectively.

Brain Stem

Brain stem is formed by the midbrain, pons and medulla oblongata. Midbrain is the superior part of the brain stem, which forms the connecting link between the forebrain and hind brain. It consists of the larger ventral portion called cerebellar peduncle and a smaller dorsal portion called tectum.

The triangular space between the two cerebral peduncles forms the interpeduncular fossa or cistern. The tectum is composed of four rounded projections called colliculi or corpora quadrigemina. The pons connects with the midbrain above and medulla below. It has an anterior bulge, which is caused by the middle cerebellar peduncle and pontocerebellar connections. The pons is separated from the medulla by the inferior pontine sulcus, and from the cerebral peduncles by superior pontine sulcus. The medulla is the caudal most portion of the brain stem and continues as cervical cord. At the level of foramen magnum it contains anterior median fissure and posterior median sulcus.

CSF spaces

The ventricular system within the brain parenchyma and subarachnoid cisterns and sulci surrounding the brain constitutes the CSF spaces. The CSF is mainly produced by the choroid plexus situated in the lateral ventricles. The ventricles of the brain are lateral ventricles, third ventricle and fourth ventricle. The two lateral ventricles communicate through the foramen of Munro with the third ventricle. The third ventricle is connected to the fourth ventricle by cerebral aqueduct (Aqueduct of Sylvius). The fourth ventricle in turn is continuous with the narrow central canal of spinal cord and through foramen of Magendie and Luschka into the subarachnoid cisterns.

Meninges:

Brain encased by three meninges:

1. Dura – It is a dense fibrocollagenous sheet consisting of two layers: periosteum of inner calvarium which forms the outer layer and the inner layer folds inwards forming the falx cerebri, tentorium cerebelli, etc. and receives blood supply from numerous dural vessels with extensive extra and intracranial anastomoses.
2. Arachnoid – It is a nearly transparent thin layer with outer surface loosely adherent to dura and can be easily separated. It does not invaginate into the sulci. It is normally not seen.
3. Pia – It is the innermost layer covering the brain and it invaginates into the sulci. It follows the penetrating cortical arteries into the brain forming the perivascular spaces[6].

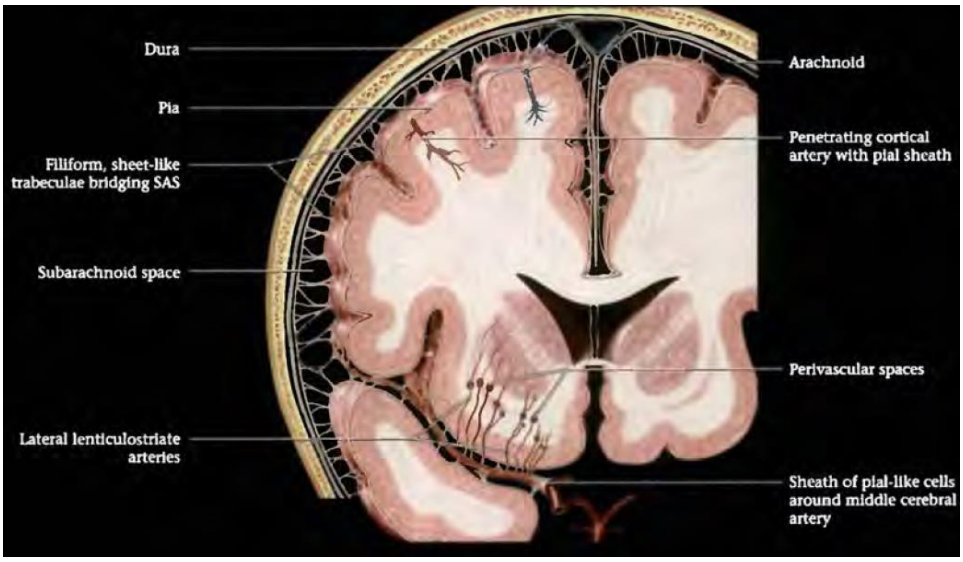

Figure 6: Pictorial representation of depicting the relationship of cranial meninges to brain &subarachnoid space.

Cranial Nerves

There are twelve pairs of cranial nerves. Cranial nerves I and II are derived from the forebrain, while all others are derived from the brain stem, including nerves V through VIII that originate from the pons. With MRI, it is possible to identify the cisternal segment of the cranial nerves.

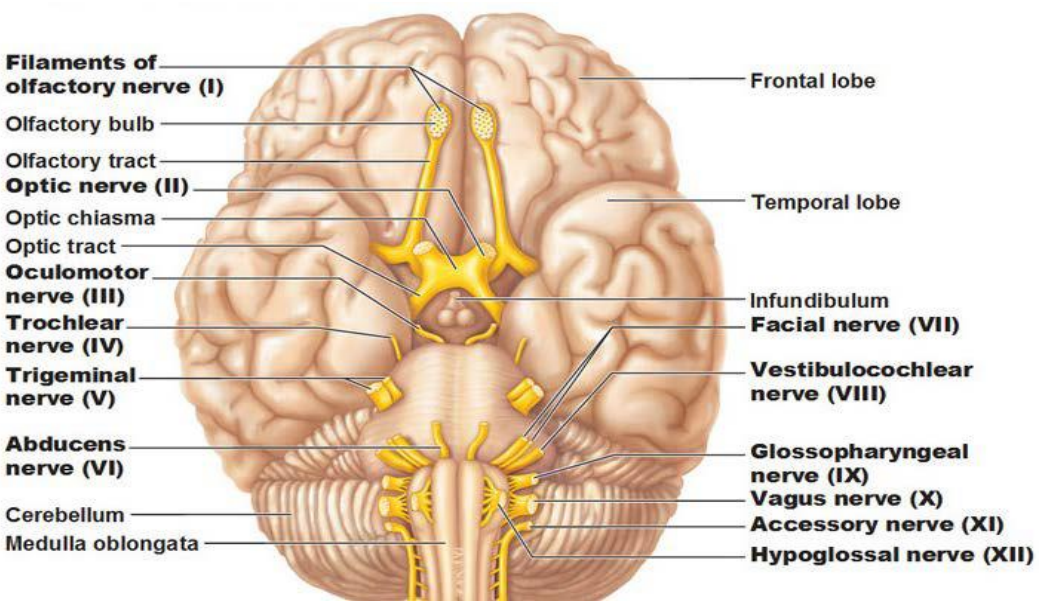

Figure 7: Pictorial representation of cranial nerves.

Vascular Systems

The two internal carotid and two vertebral arteries supply brain. These four arteries lie within the subarachnoid space and their branches anastomose on the inferior surface of the brain to form circle of Wills. The anterior communicating, anterior cerebral, internal carotid, posterior communicating, posterior cerebral and basilar arteries contribute to the formation of circle of Wills.

The circle of Wills allows the blood that enters by either internal carotid or vertebral arteries to be distributed to any part of both cerebral hemispheres. The cerebral venous system is composed of dural venous sinuses, superficial cortical and deep (medullary and subependymal) veins.

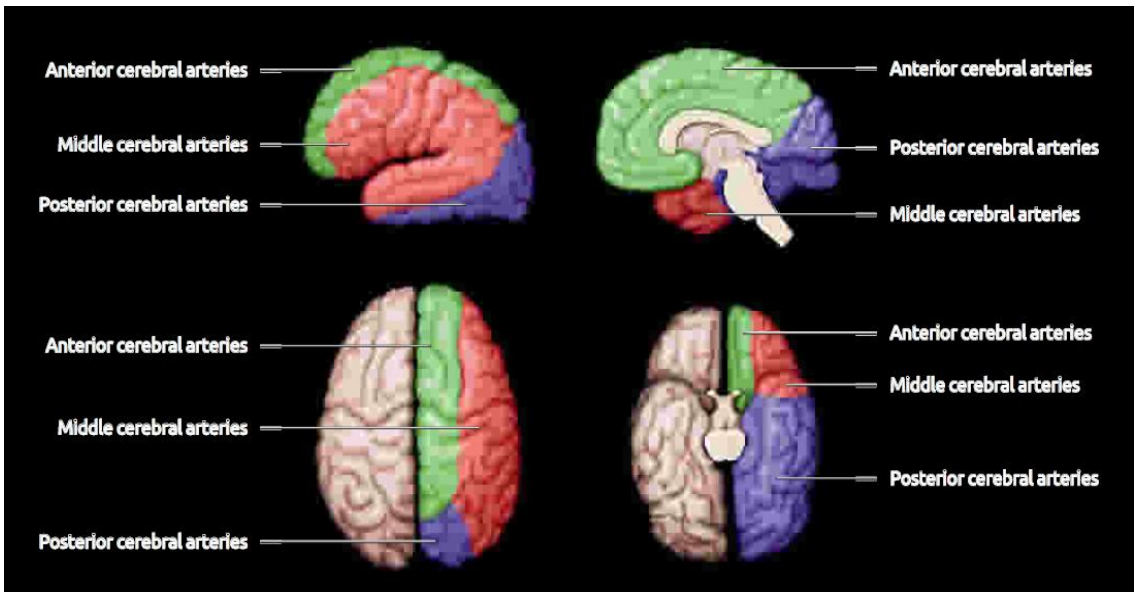

Figure8: The 3 major cerebral arteries supply the hemispheres. ACA, MCA & PCA are shown in green, red and purple. The junction of territories are called cortical watershed zone.

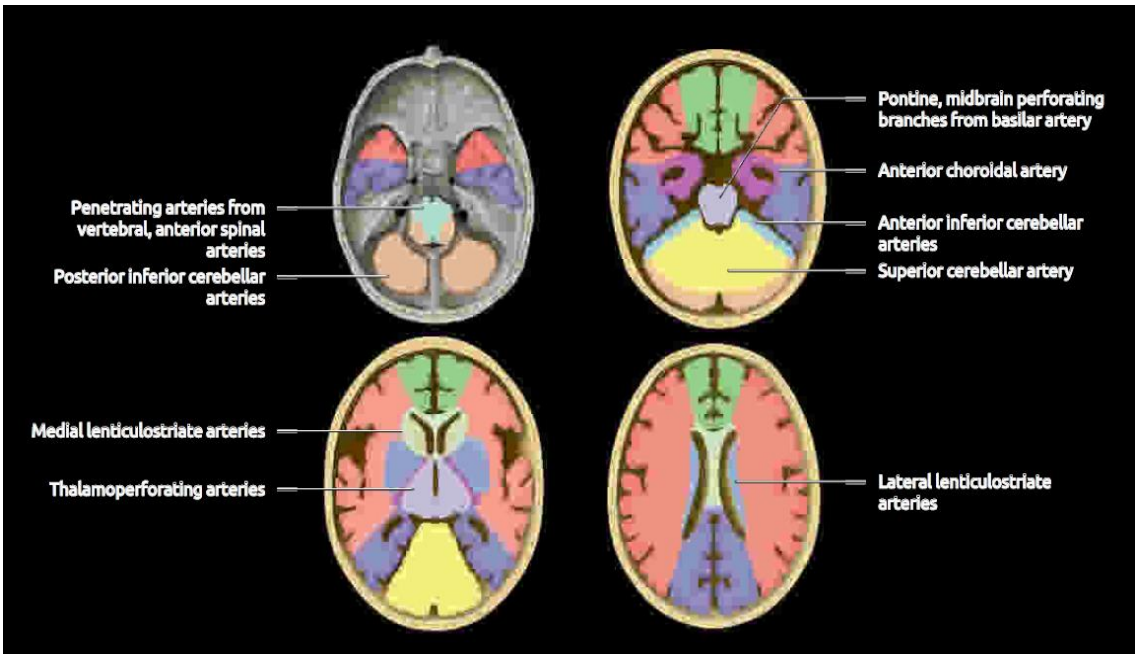

Figure 9: Axial sections showing penetrating artery territories. PICA is shown as tan color, supplying inferior cerebellum and lateral medulla. AICA is shown as light blue color. Superior cerebellar artery is depicted in yellow. Medullary (aqua), pontine, and thalamic perforating arteries.

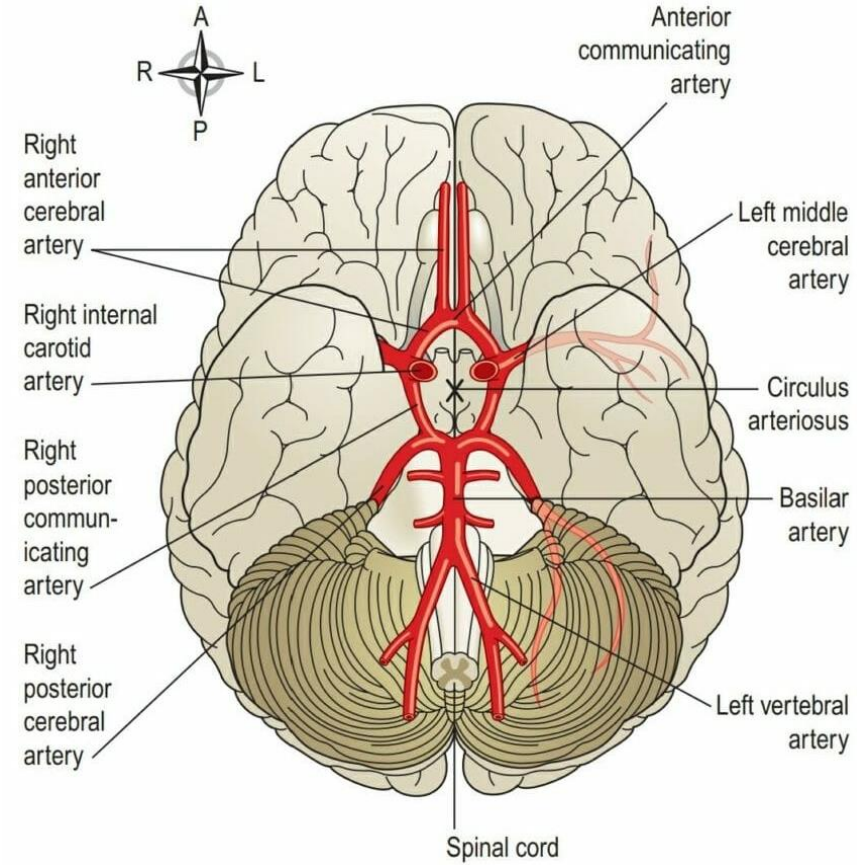

Figure 10: Circle of Wills

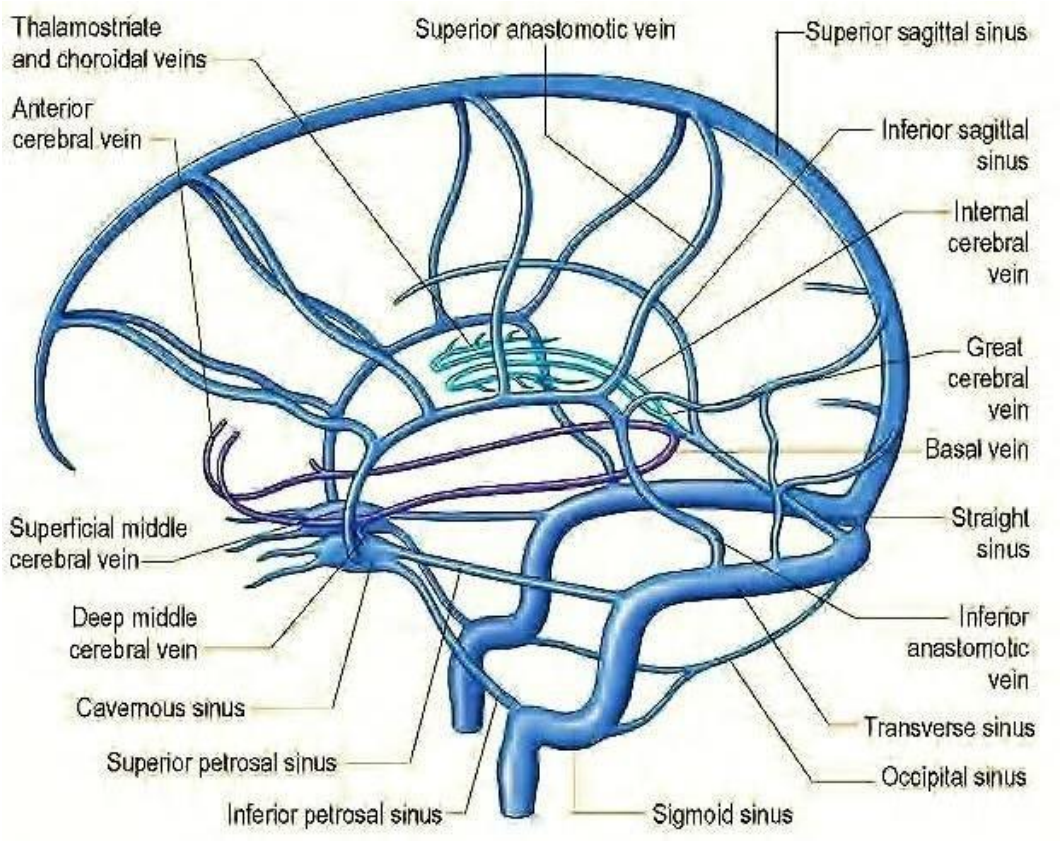

Figure 11: Veins and venous sinuses of brain.

Dura and Dural Structures

The meninges - piamater, arachnoid membrane and duramater cover the brain. The piamater follows all the gyri and is separated from arachnoid membrane by CSF. The duramater is separated from the arachnoid membrane by a potential subdural space. The outer layer of dura is attached to the periosteum of bony calvarium. The inner layer of dura forms folds within the cranial vault. The two major folds are falx cerebri and tentorium cerebelli. The falx cerebri separates the two cerebral hemispheres and tentorium cerebelli seperates the superior surface of cerebellum from the inferior surface of cerebral hemispheres.

MECHANISMS OF CONTRAST MATERIAL ENHANCEMENT:

Contrast material enhancement in CNS is the amalgamation of two primary processes: intravascular (vascular) enhancement and (extravascular) interstitial enhancement. Intravascular enhancement basically refers to vasodilatation, neovascularity or hyperemia, and shunting or shortened transit time. Endothelial cell specialization, with the help of adjacent foot processes of the perivascular astrocytes in the brain and spinal cord form the blood brain barrier which helps the brain, spinal cord and nerves to create a selectively pervious capillary membrane to protect themselves from inflammatory mediators and plasma proteins. The neural capillaries have a continuous basement membrane, narrow intercellular gaps, junctional complexes, and a paucity of pinocytotic vesicles. The semipervious blood-brain barrier allows lipophilic compounds (quantified by octanol/water partition fraction) and a few chemical substances to be transported actively with ease. Certain specific cells that possess the required surface marker proteins may pass uninterruptedly through the blood-brain barrier, whereas most other cells are precluded. Lipophobic compounds are blocked by the blood brain barrier causing the appropriate homeostatic environment for the neural tissues. A gradient is created across the capillary endothelial membrane when a bolus injection of contrast material in injected into a large peripheral vein. Contrast agent leaks across the vessel wall and accumulates in the perivascular interstitial fluid in areas with relatively free capillary permeability.

Leakage of contrast material in the brain, spinal cord, and proximal cranial and spinal nerves is prevented by the intact blood-brain barrier. Extravascular enhancement is related to remodeling in the permeability of the blood-brain-barrier,

whereas intravascular enhancement is proportional to increase in blood flow or blood volume. At intermediate times, or with a continuous drip infusion of contrast material, enhancement is a combined variable mixture of both intravascular and extravascular compartments.

Observation of contrast material enhancement is altered by several features of the MR imaging protocols. Most pulse sequences are subject to "flow void phenomena," whereby rapidly flowing fluids have low signal intensity. As a result which vascular shunt lesions appear dark on MR images. In addition, interstitial enhancement on MR images requires both free water protons and gadolinium. If a tissue is "dry" (ie, without water or free water), gadolinium enhancement will not be observed on routine T1-weighted MR images. Normal dura mater, which is extra-axial non-neural connective tissue, does not have a blood-brain barrier, but it lacks sufficient water to show T1 shortening required for enhancement on MRI.

Abnormal contrast enhancement is produced by various physiologic and pathologic conditions (which may either be unrelated or secondary to the primary lesions under investigation) and are also associated with alterations in permeability of the blood-brain barrier, new blood vessels (angiogenesis), active inflammation (infectious and noninfectious), cerebral ischemia, and pressure overload (ecclampsia and hypertension). Shortened mean transit time is seen in reactive hyperemia and neovascularity due to increased blood volume and blood flow (in comparison with normal brain tissue). Abnormal contrast enhancement on static gadolinium-enhanced MR images is seen due to abnormally increased capillary permeability and altered blood volume and flow[7].

INTRACRANIAL LESIONS

The brain can be the victim of numerous pathologies, including malignant tumors, strokes, infection, head injuries, disease, and dystonia[8]. We will be discussing mainly about the pathologies pertaining to this study, as well as few other lesions. In this study, intracranial pathologies are segregated as meningeal infection and intracranial space occupying lesions.

Intracranial tumors are a significant health problem. The annual incidence of primary and secondary central nervous system neoplasms ranges from 10 to 17 per 100,000 persons. Imaging plays an integral role in intracranial tumor management. MRI in particular has emerged as the most frequently used imaging modality to evaluate intracranial tumors, and it continues to have an ever expanding, multifaceted role. In general, the role of MRI in the workup of intracranial tumors can be broadly divided into diagnosis, classification, treatment planning, and post-treatment surveillance. Discrimination of extra-axial and intra-axial brain tumors is relatively easy with only anatomic imaging; however, the major diagnostic challenge is to reliably, non-invasively, and promptly differentiate intra-axial tumors to avoid biopsy and follow-up imaging studies. Integration of diagnostic information from advanced MR imaging techniques can further improve the classification accuracy of conventional anatomic imaging[2].

Contrast material enhancement for cross-sectional imaging has been used since the mid-1980s for magnetic resonance imaging. Knowledge of the patterns and mechanisms of contrast enhancement facilitate radiologic differential diagnosis.

I. Intracranial space occupying lesions

Intracranial space occupying lesions is further divided as infectious etiology and intracranial tumors.

A. Infectious conditions:

1.Intracranial abscess:

Brain abscesses are uncommon but potentially life-threatening manifestation of intracranial infection[9]. Four stages of brain abscess development: early cerebritis, late cerebritis, early capsulation, and late capsulation[27]. Classic MRI finding of abscess is T1 isointense to hyperintense relative to white matter and T2 hypointense surrounding a necrotic center with peripheral rim enhancement on postcontrast images. Characteristic peripheral T1 and T2 signal shortening is due to collagen, hemorrhage, or free radicals. Bacterial and mycobacterial abscesses classically have a smooth or lobulated capsular margin, compared with fungal disease, where intracavitary projections are more common[9].

2.Tuberculoma:

Tuberculoma is the most common parenchymal form of tuberculosis. It typically manifests as a lesion with well-defined hypointensity and solid or ring like enhancement at T2WI. These lesions may be single or multiple. Central non-enhancing high signal intensity at T2WI depends on extent of liquefaction or caseation. A small focal area of calcification or enhancement in the center of a ring-enhancing mass referred as “Target sign,”is pathognomonic for tuberculous granuloma but nonspecific. A tuberculous granuloma has less surrounding vasogenicedema than tuberculous abscess. Histopathologic examination is more

definitive in distinguishing a caseating tuberculoma from tuberculous abscess than imaging[8].

3.Neurocysticercosis:

Neurocysticercosis (NCC) is caused by infection due to Taenia solium after faecal-oral contamination from a tapeworm carrier of infective embryos. Patients with neurocysticercosis are frequently asymptomatic during early stages of the disease but later develop seizures (50%–70%), headaches (43%), and findings related to hydrocephalus (30%) when scolices begin to die or from cysts in the ventricular system.

There are four stages of parenchymal neurocysticercosis. In vesicular stage, parasite resides quiescently in brain parenchyma as a small non-enhancing cyst protected by a cyst wall rich in glycoproteins that provides an effective barrier from surrounding tissue, without surrounding edema. MRI findings of cysts in this stage are consistent with fluid. The presence of scolex in such a cyst may create a “target” or “dot in a hole” appearance because slight soft-tissue attenuation and signal intensity in the larva help distinguish it from surrounding fluid. The vesicular colloidal (also referred to as colloidal vesicular or colloidal) stage is characterized by death of the scolex from natural processes or from effects of therapy with associated disruption of the cyst wall. As a consequence, an intense inflammatory reaction to the then unprotected and decaying parasite results in altered signal intensity, compared with CSF. At T2 FLAIR MRI sequence, this reaction is particularly conspicuous related to the developing proteinaceous and gelatinous debris and typically substantial surrounding edema. A ring like pattern of enhancement is often evident at postcontrast T1FS and T2 FLAIR images. The

granular nodular stage begins with cyst retraction and formation of a granulomatous nodule with surrounding gliosis. Calcification of this nodule demarcates the calcified nodular stage as last (and non-active) stage of neurocysticercosis. Any remaining edema or enhancement at neuroimaging resolves during this stage. Multiple lesions corresponding to varying stages of the disease are typical[8].

4.Toxoplasmosis:

It is caused by Toxoplasma gondii, an obligate intracellular protozoan parasite, is the most common opportunistic infection affecting CNS in patients with AIDS.

Neuroimaging studies in patients with toxoplasmosis commonly show multifocal abscesses with a predilection for the basal ganglia. However, solitary lesions have been noted in about one-third of patients. Most lesions show enhancement, often in a ring like pattern. Although it occurs in less than 30% of cases, the eccentric target sign, which is related to a small enhancing nodule along lesion margin, has been reported to be highly suggestive of a diagnosis of toxoplasmosis. In distinction to the well-defined enhancing wall seen in bacterial abscess formation, toxoplasmosis abscesses may have poorly defined peripheral enhancement in immunocompromised patients; this finding is believed to reflect a poor host response to the infection. Diffuse cerebral volume loss is seen in approximately 30% of cases and likely reflects changes related to superimposed HIV infection.

Imaging appearance of toxoplasmosis overlaps that of CNS lymphoma and other neoplasms. Subcortical location, eccentric target sign, absence of corpus

callosal or leptomeningeal involvement, and marked edema are imaging findings that favour toxoplasmosis, whereas hyperattenuation, hypointensity on T2WI, restricted diffusion, and periventricular location favour CNS lymphoma[8].

B. Intracranial tumors:

A brain tumor is defined as a mass which is formed by overgrowth of abnormal cells. Tumors originating in the brain, brain's coverings, or its nerves are considered primary brain tumors, which are the most common type occurring in children. Metastatic or secondary brain tumors are most common in adults which are caused by cancer that has spread to the brain from other parts of the body. Nearly one in four people with cancer will get a secondary brain tumor.

Brain tumors are classified as benign or malignant. Benign tumors are composed of non-cancerous cells that do not invade the brain or other tissues. Benign brain tumors can grow large enough to put pressure on the sensitive areas of the brain, impair function, lead to coma and death.

Malignant brain tumors may contain benign-appearing cells which invade normal tissue, or contain cancerous cells either from the brain or other body cancers. Brain tumors are most commonly found in adults when they are between the age of forty and seventy, with only two to three percent of new cancer cases attributed to primary brain tumors.

The most common primary brain tumors in adults include pituitary tumors & meningiomas (both of which are typically benign) and glioblastomas. Brain tumors in children are typically found between the age of three and twelve which accounts for ~25 % of all pediatric cancers. The most common pediatric primary brain tumors are medulloblastoma, astrocytoma, ependymoma and brain stem glioma.

Diffuse astrocytic and oligodendroglial tumors:

a. Diffuse astrocytoma

Low-grade diffuse astrocytomas demonstrate as an infiltrative growth pattern. These are characterized by a high degree of cellular differentiation, slow growth and absence of microvascular proliferation and necrosis. They are classified as WHO grade II tumoursand include fibrillary, protoplasmic and gemistocytic variants.

Fibrillary astrocytomas are most common variant. It accounts for 65-80%. It is usually supratentorially in origin. On MRI, the lesion presents as well-defined T1 hypointense-isointense to white matter and T2 homogeneous hyperintense with no postcontrast enhancement and no restricted diffusion on DWI. On MR spectroscopy: increased choline, decreased N-acetylaspartate (NAA) and no lactate peak is seen.

Protoplasmic astrocytomas represent approximately 5% of low grade diffuse astrocytomas. On MRI, the lesion appears as T2 homogeneous hyperintense lesion with marked suppression of portions of tumour on FLAIR. On postcontrast sequences, the lesion demonstrates mild enhancement with a complete or incomplete ring of reduced apparent diffusion co-efficient (ADC). MR spectroscopy: elevated choline to creatine ratio, reduced NAA with increased choline to NAA ratio.

Gemistocytic astrocytoma accounts for 10% of low-grade diffuse astrocytomas, are even less typical exhibiting features usually associated with

higher grade tumours. This subtype has a greater tendency to dedifferentiate into anaplastic astrocytoma and glioblastoma. On imaging, heterogeneously high T2 signal due to cyst formation that suppresses on FLAIR with peripheral enhancement on postcontrast study with variable DWI/ADC. MR spectroscopy demonstrates increased choline to NAA ratio[10].

b.Glioblastoma

It is the most malignant brain tumor which is categorized as WHO grade IV. It accounts for ~ 12–15% of all intracranial tumors and 60–75% of astrocytic tumors[11]. It occurs commonly between 45 and 70 years of age with mean age of 55 years. It frequently affects cerebral hemisphere in adults and brainstem in children. On MRI, the lesion demonstrates a large, heterogeneous mass in cerebral hemisphere exhibiting necrosis, hemorrhage and enhancement on postcontrast sequence[12].

c. Oligodendroglioma

They are glial tumours, together with mixed oligoastrocytoma. It accounts for 5–20% of all gliomas. They frequently occur predominantly in adults (peak between 40 &60 years of age). These tumors are frequently located supratentorially (85%), with majority of frontal lobe involvement (50–65%) involving both cortex and subcortical white matter. Oligodendroglioma is generally round to oval appearing as a relatively well-circumscribed mass of variable size. Presence of calcification and cortical-subcortical location of the lesion is the hallmark features of oligodendroglioma. On MRI, the lesion is hypointense to grey matter on T1WI and heterogeneously hyperintense on T2WI. Calcification contributes to the

heterogeneity of tumor appearance. On postcontrast sequences, usually do not enhance; however in 50% of cases demonstarte minimal to moderate patchy, multifocal enhancement with a dot-like or lacy pattern can be seen. Bone adjacent to lesion shows changes in the form of focal thinning, remodelling or even erosion of overlying skull[13].

d.Oligoastrocytoma

Oligoastrocytomas are part of glial cell continuum that includes both astrocytic and oligodendrocytic components. These tumors are graded, based on WHO classification, as either low grade (WHO grade II) or anaplastic (WHO grade III). These tumors typically occur in young adults (mean age: 35–45 years) and manifest with partial or generalized seizures. Although less common, patients may present with headaches or may have no symptoms at all. Most oligoastrocytomas occurred in the frontal or temporal lobes. Initial radiologic evaluation of patients with a suspected intracranial mass is usually performed with unenhanced CT of the head. MR imaging typically reveals a lesion that is hypointense on T1WI, hyperintense on T2WI and no contrast enhancement is seen[14].

1.Other astrocytic tumors

a.Pilocytic astrocytoma

Pilocytic astrocytoma (PA) is a rare, slow-growing glioma, classified as grade I by WHO occurring typically in children and young adults with peak incidence btween the age of 5 and 13 years of age. The most common glial neoplasm in children is PA. Cerebellum is the most common location in children whereas supratentorial is the most common location in adults. On MRI, there are four

predominant patterns. They are a non-enhancing cyst with intensely enhancing mural nodule; a mass with an enhancing cyst wall & mural nodule; a necrotic mass with a central non-enhancing zone and a predominantly solid mass with minimal to no cyst like component. The solid component of PAs can be homogeneous or heterogeneous with T1 hypo- to isointensity and T2 hyperintensity compared to grey matter with variable enhancement pattern of solid component[15]. Intratumoral hemorrhages are rare in PAs[16].

b.Pleomorphic xanthoastrocytoma

Pleomorphic xanthoastrocytoma (PXA) is a rare, usually low-grade (WHO grade 2), astrocytic tumor. It presents below the age of 20 years with no gender predilection. They are superficially located supratentorial glioma commonly involving the temporal location showing extensive involvement of leptomeninges[17]. PXA was included in 1993 in the WHO classification of tumors of CNS. On MR, these lesions present as cortical based cystic masses. The solid component of the lesion that are T1 isointense to gray matter and T2 mildly hyperintense with heterogenous enhancement on postcontrast study. Cystic component of the lesion appears T1 hypointense and T2 hyperintense with homogenous enhancement. There is also leptomeningeal enhancement demonstrated on postcontrast study in majority of cases with dural tail mimicking meningioma; however absence of signs of extra-axial lesion helps to include PXA in differential diagnosis[18]. Minimal surrounding edema can be seen[19,20].

2.Ependymal tumors

a. Subependymoma:

Subependymoma arises from subependymal glial layer surrounding cerebral ventricles. It is usually less than 2 cms in size and commonly located within 4th and lateral ventricles; however can also present as brain parenchyma, cerebellopontine angle, and spinal cord lesions[21,22,23]. Males are more frequently affected. On MRI, these tumors are T1 hypointense and T2 hyperintense compared with white matter with heterogeneity due to cyst like areas interspersed within the mass. On postcontrast sequences, variable enhancement is demonstrated, i.e, the lesion may not enhance, minimally enhance, or show avid heterogeneous enhancement. Extension of a subependymoma beyond the ventricular margins helps in distinguishing subependymomas from ependymomas[24].

b.Ependymoma:

Ependymoma arises from ependymal cells lining cavities and passage ways of the brain (ventricles) and spinal cord. They account for ~ 6% of all CNS tumors with ~ 85% of them are considered as low-grade. In children, it is the 3rd most common brain tumor. They are commonly seen between 1 and 5 years of age. On MRI, both infra- and supratentorial ependymomas present as T1 hypointense and T2 hyperintense lesions with avid postcontrast enhancement of soft tissue components intermixed with poorly enhancing or non-enhancing areas[25,26]. Calcifications are seen in ~ 50% of cases[25].

3.Other gliomas

a. Chordoidglioma of third ventricle

A chordoidglioma is a rare, low-grade, slow growing tumor arising from ependymal cells of lamina terminalis of anterior wall or roof of third ventricle[27].It is mostly seen in adult female population. On MRI, these lesion appear are well defined, round or oval in shape, iso- to hyperintense third-ventricle masses on T2WI with homogenous avid postcontrast enhancement. Occasionally, cystic changes can be seen within the periphery of these tumors[28].

b. Angiocentric glioma

Angiocentric glioma (AG) is seen predominantly in children and young adults. These lesions are predominantly supratentorial, are often seen superficially, and have predominant cortical involvement. Involvement of the fronto-parietal cortex, temporal lobe, and hippocampal region has been documented. Involvement of the thalamus can also be seen[29].

On MRI images, AGs are T2 hyperintense, T1 hypointense, and show no significant postcontrast enhancement. They are predominantly cortical based and well delineated, and often show a stalk like extension to the subjacent ventricle. Calcifications within the lesion is unusual finding. Calvarial remodeling can also be seen[30,31].

c. Astroblastoma

Astroblastoma is a rare glial tumor accounting for 0.45–2.8% of primary brain gliomas. These tumors have radiating arrangement of spindle-shaped tumor cells

forming perivascular pseudo-rosettes with broad tapering cellular processes extending to central vessels, perivascular hyalinization, and lack of fibrillarity. On MR images, classic appearance of astroblastoma is large, well-circumscribed, lobulated peripheral mass in supratentorial brain. The mass typically consists of a mixture of cystic and solid components exhibiting heterogeneous bubbly appearance on T2WI. The solid portions of masses are relatively hypointense to gray matter on T1WI and isointense to gray matter on T2WI. A relative lack of peritumoral T2 hyperintensity exists, representing vasogenic edema or tumor infiltration or both in surrounding brain parenchyma. Enhancement is heterogeneous, with rim enhancement of cystic portion and heterogeneous enhancement of solid portion[32].

d. Brain stem gliomas:

Brain stem glioma accounts for 10 – 15% of pediatric brain tumors and ~ 2% of adult brain tumors. It is seen usually below 2nd decade of life. These are seen commonly in pons portion of brain stem and are called as pontine gliomas, which are aggressive in nature. On imaging, these lesions present as ill-defined, infiltrative masses with variable enhancement patterns.

e. Tectal plate glioma

These are low-grade gliomas. On MRI, Tectal plate gliomas present as T2 hyperintense and T1 iso- to hypointense lesions with no or minimal postcontrast enhancement[33].

4.Choroid plexus tumors

Choroid plexus tumors are rare intraventricular papillary neoplasms derived from choroid plexus epithelium, which account for approximately 2% to 4% of intracranial tumors in children and 0.5% in adults. Choroid plexus papillomas (CPPs) are more common than choroid plexus carcinomas (CPCs)[34].

a. Choroid plexus papilloma

Choroid plexus papiloma (CPP) is a rare benign intracranial neoplasm comprising 1% of all brain tumors, but more common in young children comprising 3% of all brain tumors. They arise from ventricles with origin being more common from lateral ventricles in children and fourth ventricle in adults. The frequency of involvement of ventricles in children- 80 %-lateral, 16% fourth, 4%-third ventricle[35,36].

b. Choroid plexus carcinoma

On MRI, the lesion is usually T1 iso-hypointense with areas of increased signal intensity due to haemorrhage. T2WI shows heterogeneously hyperintense mass lesion relative to normal brain parenchyma. On postcontrast sequence, tumor shows intense enhancement. Associated hydrocephalus may be noted. MR is more sensitive for detection of spread of tumor, particularly to spinal subarachnoid space & meninges[35].

5.Neuronal and mixed neuronal glial tumors

a. Gangliocytoma

Gangliocytoma denotes a spectrum of rare tumors in which neuronal cell

lineages are the sole neoplastic constituents along with neuronal population, there is a highly variable normocellular network of non-neoplastic glial cells. This distinguishes gangliocytomas from gangliogliomas, which also contain anaplastic glial cells; however clear-cut separation of gangliocytomas from gangliogliomas is difficult. They represent opposite ends of a spectrum of differentiated ganglion cell tumors. The most common sites are cerebral hemispheres and cervicothoracic spinal cord. Within cerebral hemispheres, temporal lobe, either alone or in combination with frontal or parietal lobes are favored site. Other intracranial locations include cerebellum, hypothalamus adjacent to floor of the 3rd ventricle, pineal region, and pituitary. Patients typically present with seizures or focal neurologic signs according to location and resultant mass effect. MR imaging of gangliocytomas reveals masses of low signal intensity on T1WI, high signal intensity on T2WI, and frequent enhancement on postcontrast study. However, calcification and cyst formation are frequently revealed on MR images[37].

b.Ganglioglioma

Gangliogliomas are the most common of the neuronal-glial neoplasms arising within central nervous system. Clear-cut separation between ganglioglioma and gangliocytoma is difficult because there are varying degrees of neoplastic evolution of the glial component. Classically, ganglioglioma is described as a cystic mass with a mural nodule in approximately 40% of diagnosed cases. Most gangliogliomas occur in children and young adults. Most tumors develop in temporal lobe.

On MR imaging, a well-defined cystic mass with a solid mural nodule is

typically seen. Solid mass shows non-specific low to intermediate signal intensity on T1WI and high signal intensity on T2WI. Enhancement of solid portion is highly variable, ranging from non-enhancing to ring like to intense homogeneity. Calcification has been noted in about 30% of cases. There is usually little associated mass effect or surrounding vasogenic edema. Peripherally located gangliogliomas may cause scalloped pressure erosion of the overlying calvaria due to their slow-growing nature[37].

c. Central neurocytoma

It was termed an intraventricular oligodendroglioma or ependymoma of foramen of Monro, before neuronal immunohistochemical and ultrastructural characteristics of central neurocytoma were recognized. This tumor affects mainly young adults and has a favorable prognosis. It is located in lateral ventricles near foramen of Monro with a characteristic attachment to the septum pellucidum. Its clinical history is short, and majority of patients present with symptoms of increased intracranial pressure secondary to obstructive hydrocephalus.

On MR imaging, central neurocytoma is inhomogeneously isointense on T1WI. Signal intensity on T2WI is variable but is mostly isointense to hyperintense with the cerebral cortex. Areas of low signal intensity or absent signal on both T1 and T2WI can represent calcification, cyst, hemorrhage, or tumor vessels.

d. Extraventricular neurocytoma

Extraventricular neurocytoma (EVNs) is a rare entity. It frequently occurrs in brain parenchyma outside the ventricular system.The solid parts of tumors are

primarily isointense on T1WI and show heterogeneous enhancment on postcontrast sequence. Cerebral EVNs can present a wide spectrum of appearances on MR. The imaging patterns appear to vary according to anatomic location and cellularity. Lesions in frontal or parietal lobes often present as well-demarcated large masses with cystic degeneration, hemorrhage, mild-to-moderate edema and inhomogeneous enhancement[38].

e. Paraganglioma

Paragangliomas are slowly growing hypervascular tumors arising from neural crest cell derivatives throughout the body. MR imaging provides a unique tool to assess the patient with suspected paragangliomas. MRI detects the vascular nature of these lesions which is highly characteristic for diagnosis of paraganglioma. Multiple areas of low signal on both T1 and T2WI resulting from high velocity signal loss is characteristic of rapid arterial and venous blood flow present in the matrix of these tumors. Calcification can also produce signal void on MR images. The advantages of MR include the ability to characterize the vascularity of a lesion without the use of intravenous contrast material as well as demonstration of tumor extension into the posterior fossa without the high spatial-frequency artifact from the petrous bone that frequently diminishes the quality of CT examination. Additional information regarding the relation of the tumor to the surrounding important vascular structures such as the carotid artery and jugular vein was also more easily evaluated by MR than CT. T1WI give better spatial resolution and better show the highly vascular internal matrix of tumor. T2WI give better tissue contrast, making smaller lesions more conspicuous. In larger lesions, the "salt-and-pepper" appearance on T2WI was characteristic of paragangliomas. On post

contrast sequences, heterogeneous enhancement is demonstrated[39].

6.Tumors of pineal region

a. Pineocytoma

Pineocytoma is a slow-growing WHO grade I lesion accounting for 14%–60% of pineal parenchymal neoplasms. They occur throughout life but predominantly manifest in adults (mean age, 38 years)[40]. No gender predilection. On MR imaging, pineocytomas are well-circumscribed lesions that are hypo- to isointense on T1WI and hyperintense on T2WI. On postcontrast images, they demonstrate avid, homogeneous enhancement. Cystic or partially cystic changes may occur, occasionally making differentiation from a pineal cyst difficult. However, at immediate postcontrast imaging, cystic-appearing pineocytomas demonstrate internal or nodular wall enhancement. Hemorrhage into the lesion (pineal apoplexy) occurs rarely[41].

b. Pineoblastoma

Pineoblastomas are highly malignant WHO grade IV lesions representing the most primitive form of pineal parenchymal tumors and account for 40% of pineal parenchymal tumors. They most commonly occur in first 2 decades but can occur at any age, and there is no gender predilection. CSF dissemination commonly occurs and is the most common cause of death. The 5-year survival is 58%[40]. On MR imaging, pineoblastomas are heterogeneous in appearance, with solid portion appearing hypo- to isointense on T1WI and iso- to mildly hyperintense to the cortex on T2WI. Pineoblastomas demonstrate heterogeneous enhancement on postcontrast images. Necrotic regions and hemorrhage may be present[41].

c. Papillary tumor of pineal region

Papillary tumor of pineal region (PTPR) is a recently recognized neoplasm in the WHO 2007 classification. It is a rare neuroepithelial neoplasm that occurs in both children and adults, with a reported age range of 5–66 years (mean age, 31.5 years)[42]. PTPRs are thought to arise from specialized ependymocytes in the subcommissural organ, which is located in pineal region. PTPRs are well-circumscribed lesions with variable signal intensity on T1WI, high signal intensity on T2WI, and heterogeneous enhancement on postcontrast images. Cystic areas are commonly present. Hyperintensity on T1WI has been described, which is hypothesized to be related to secretory inclusions containing protein or glycoprotein[41].

7.Embryonal tumors

a. Medulloblastoma

It is the most common pediatric CNS malignancy. It is also the most common primary tumor of the posterior fossa in children. It occurs more common in males and seen usually in less than 10 years of age; however, less frequently, the disease is seen in adults, usually in 3rd and 4th decades of life. The tumor has characteristic hyperattenuation on unenhanced CT scans reflecting the high nuclear-cytoplasmic ratio on histologic analysis. Typically, the tumor appears heterogeneous on images, due to cyst formation, hemorrhage, and calcification. Evidence of leptomeningeal metastatic spread is present at the time of diagnosis which is well evaluated with contrast enhanced (CE)-MRI brain and the spine. On MR imaging, the lesion is T1 iso- to hypointense relative to white matter and T2 variable signal intensity relative to white matter. On postcontrast study, the lesion demonstrates heterogeneous

enhancement. MR spectroscopy typically shows elevated choline peaks, reduced N-acetyl aspartate & creatine peaks and occasionally elevated lipid & lactic acid peaks. This MRS findings are characteristic spectrographic signature for a neuroectodermal tumor but not specific for medulloblastoma.

Foraminal extension from the fourth ventricle to involve the cerebellopontine angle, cisterna magna, and other cisternal compartments may be seen. Imaging of desmoplastic medulloblastoma demonstrates a cerebellar mass that extends to the overlying meninges leading to abnormal leptomeningeal enhancement which in-turn mimics imaging appearance of a meningioma[43].

b.CNS neuroblastoma

Primary CNS neuroblastoma was often categorized under primitive neuroectodermal tumors, although this was controversial.

No pathognomonic appearances are seen on CT or MRI scans. Cystic changes or calcification can be observed.The exact data about the lesion is not available in the literature because of the rarity of the disease. It is a differential diagnosis in pediatric age group for intraventricular or periventricular masses. On plain CT scan, tumor was homogenously hyperdense with postcontrast enhancement. On MRI, the lesion is uniformly hypointense on T1WI and predominantly hyperintense with few areas of cystic changes within the lesion on T2WI[44,45].

8.Tumors of cranial and paraspinal nerves

a. Schwannoma

A schwannoma is a benign nerve sheath tumor composed of spindle cells.

Intracranial schwannomas are most often associated to a cranial nerve (CN). It has a characteristic appearance on MRI. On MRI, the lesion appear T2 heterogeneously hyperintense and T1 hypo to isointense. The "target sign" appearance of peripheral hyperintensity with central hypointensity in peripheral nerve schwannomas on T2WI is highly specific but insensitive; however, it is rarely seen at imaging of CN schwannoma. On postcontrast sequences, the lesion demonstrates avid enhancement with or without non-enhancing cystic spaces. Larger lesions demonstrates heterogeneous enhancement due to cystic spaces, and foci of hemosiderin due to internal hemorrhage[46].

b.Neurofibroma

Intracranial CNS manifestations include characteristic Neurofibroma (NF1) "spots" and low-grade neoplasms. The NF1 "spots" are regions of signal abnormality representing regions of myelin vacuolization. The lesion is commonly seen in 1st decade of life.On MRI, the lesion is T2 hyperintense and typically iso- to mildly hyperintense on T1WI. On postcontrast sequences, usually no enhancement is seen; however, if present then it suggests development of a low-grade glioma.

Low-grade optic pathway glioma (OPG) is the most common CNS neoplasm associated with NF1. It is considered pathognomonic for NF1 if bilateral OPG is present. On MRI, these tumors are T1 hypointense and T2 hyperintense with variable postcontrast enhancement[47].

9.Meningiomas

Meningiomas are the most common mass dural lesion. It accounts for 38% of intracranial tumours in women and 20% in men. It is commonly seen beyond 50

years of age. WHO classifies them as benign (grade I - 80%), atypical (grade II - 18%) and anaplastic/ malignant (grade III - 2%) based on their histological characteristics and recurrence risk. The majority of meningiomas arise spontaneously. Most often the etiology is unknown. Risk factors for meningioma are radiation exposure, genetic disorders such as NF type 2 and post head injury. The parasagittal aspect of the cerebral convexity, the lateral hemisphere convexity, the sphenoid wing, middle cranial fossa and the olfactory groove are the most common locations. The modality of choice for the investigation of meningiomas is MRI, as it provides superior contrast differentiation and the ability to differentiate between intra- and extra-axial lesions.

Meningiomas are extra axial lobular masses with well-circumscribed margins, broad-based dural attachment and buckling of cortical grey matter. On MRI, the lesion is T1 isointense to slight hypointense and T2 isointense to slight hyperintense relative to grey matter. On postcontrast sequences, meningiomas demonstrate avid, homogeneous enhancement with non-enhancing areas of central necrosis or calcification. Postcontrast sequences help delineating enplaque meningiomas which are typically seen as asymmetric thickened sheets of enhancing dura. Enhancement of the dura infiltrating away from the lesion known as dural tail and presence of a CSF cleft between the tumor and the underlying brain helps to distinguish meningiomas from other potential etiologies. Bony changes associated with the lesion can present as osteolysis or hyperostosis[48].

10. Mesenchymal & non-meningothelial tumors

a. Solitary fibrous tumor

Primary Solitary fibrous tumor (SFT) is a rare tumor occurring in the

meninges. It was first reported in 1996. On MRI, the lesion usually appears to be in contact with meninges. The lesion is T1 isointense and T2 heterogeneous hypointense. The areas of low and high signal intensities on T2-WI are referred to as patch or "ying-yang" appearance which is characteristic for the SFT. On postcontrast sequences, the solid portion of the lesion demonstrates avid heterogeneous enhancement[49].

b.Hemangioblastoma

It is a benign tumor of the CNS accounting for 1%-2.5% of all intracranial neoplasms. It is commonly seen between 3rd & 5th decade of life with slight male predilection. These tumors may be predominantly solid or have both solid and cystic areas. It is commonly seen in the cerebellum (83%-86%) and it comprise 7%-12% of primary posterior fossa tumors. On MRI, the lesion is T1 hypo- to isointense and T2 hyperintense. Occasionally, the lesion may be heterogeneous on T1WI hyperintense foci within solid portion of the tumor representing lipid within stromal cells or methemoglobin from hemorrhage within the tumor.The cyst fluid is T1 hyperintense compared to CSF and much more hyperintense on T2WI which are attributable to its high protein content; leading to better visualization of tumor nidus on postcontrast sequences. If the cyst is lined by neoplasm, the wall will enhance on postcontrast sequence. Traditionally the patterns of hemangioblastoma are described as purely solid or solid mural nodule with an adjacent non-enhancing surrounding cyst or completely cystic[40].

c.Hemangioma

Cavernous hemangiomas are defined as vascular malformations consisting of abnormal, dilated vessels within intervening neural tissue. On MRI, the lesion is

well-defined with T1 iso to hypointense and T2 markedly hyperintense signal intensity. On postcontrast sequences, the lesion demonstrates homogeneous enhancement. The pattern of contrast enhancement is very helpful to distinguish cavernous hemangioma from other intracavernous masses such as meningiomas and schwannomas which show heterogeneous enhancement[50].

d. Lipoma

Intracranial lipoma is a rare congenital lesion accounting for less than 0.1% of all intracranial tumors. It is characterized by presence of fatty tissue deposits in CNS. It is more commonly seen in pericallosal zone. On radiograph, pericallosal lipomas appear as increased lucency area with surrounding curvilinear calcifications. On CT scan, the lesion presents as lobulated extra-axial mass (density: –50 to –100 HU) with calcification. On MRI, the lesions are T1 homogenously hyperintense that decreases on T2 and gets vanished on fat suppression sequences[51].

11. Melanocytic tumors

a. Meningeal melanocytosis

Primary leptomeningeal melanomatosis is a rare aggressive neoplasm of the CNS arising from melanocytes within the leptomeninges with poor prognosis. It occurs frequently in adults with peak incidence at 4th decadeof life. On MRI, the melanocytic regions are T1 iso to hyperintense and T2 hyperintense with diffuse thickening of the leptomeninges, demonstrating abnormal avid postcontrast enhancement[52].

b.Meningeal melanocytoma

Meningeal melanocytomas are rare, slow-growing pigmented benign neoplasms with an annual incidence of 1 in 10 million population with female predilection. It more commonly presents in 5th decade of life. The lesion arises from the normal melanocytic cells of the leptomeninges. It is commonly seen in posterior cranial fossa, Meckle's cave, or cervical & thoracic spinal canal. On MRI, the lesions appear T1 isointense to hyperintense and T2 iso to hypointense with homogeneous postcontrast enhancement[52].

c.Meningeal melanoma

Primary CNS malignant melanoma is a rare. It accounts for ~ 1% of all melanoma cases which present in adults with mean age of 50 years and male predilection. It arises from neural crest origin of both melanocytes and meningothelial cells within the leptomeninges. Patients with primary CNS malignant melanoma have better prognosis than that of those with metastatic melanoma & with the presence of leptomeningeal spread. On MRI, these lesions demonstrate T1 & T2 shortening with postcontrast homogenous enhancement; however inhomogeneous, peripheral, or nodular enhancement pattern can also be seen[52].

12. Lymphomas

Primary CNS lymphoma is of non-Hodgkin's B-cell type. It usually presents in patients of ~50 years of age with male predilection. The lesion are solitary usually supratentorially in location in the white matter of frontal or parietal lobes or insubependymal regions. On MRI, the lesions appear as T1 isointense to

hypointense and T2 hypointense with homogenous postcontrast enhancement. Necrosis, cyst formation, calcification, and hemorrhage are rarely seen. A classic butterfly pattern is seen when the lesion crosses corpus callosum[53].

13. Histiocytic tumors

a. Langerhans cell histiocytosis– LCH

Incidence of CNS involvement in LCH cases is ~16%. On MRI, there is loss of normal posterior pituitary bright spot on T1WI and the lesion appears hyperintense on T2WI with non-specific enhancement pattern and thickening of pituitary stalk on postcontrast sequences[54].

b. Juvenile xanthogranuloma

Intracranial Juvenile xanthogranuloma (JXG) is an uncommon benign lesion often secondary systemic disease and to infiltration of Non-Langerhans cell histiocytes; however primary CNS lesions are rare. It is frequently seen below the age of 10 years[55]. Cerebral parenchyma, ventricular-cortex and dura are three most common locations. Imaging features of intracranial JXG are variable and complex. On MRI, the lesion is T1 hyperintense and T2 hypointense with postcontrast homogenous enhancement. T1 hyperintensity of the lesion is attributable to the presence of lipid within these lesions[56].

14. Germ cell tumors

a. Germinoma

Germ cell tumor accounts for 0.15 % of lesions in major transsphenoidal series. They usually arise in suprasellar or hypothalamic region with extension

along the floor of the third ventricle. It is seen commonly in children and adolescents (mean age of 18 years). They frequently present in children and adolescents, with a mean age of 18 years. These lesions initially present as thickening or enhancement of the infundibulum later leading to development of a focal suprasellar mass.

On MRI, the lesion appear as isointense area to brain on T1WI & T2WI with avid postcontrast enhancement[57].

15. Tumors of sellar origin

a. Craniopharyngioma

Craniopharyngiomas are slow-growing benign tumors originating from squamous epithelial rests of Rathke's pouch. These are primarily tumors of children and young adults but may also have delayed presentation in middle age or older. On MRI, cystic lesions with high cholesterol content or containing methemoglobin demonstrated T1 & T2 hyperintensity; however T1 & T2 hypointense signal was seen in a cystic lesion containing keratin and extensive bone trabeculae. The presence of calcification is often diagnostic of Craniopharyngioma which is shown better on CT compare to MR. In evaluation of tumor extent for presurgical & radiation planning and in detecting tumor recurrence; MRI is the modality of choice[57].

b. Granular Cell Tumor (GCT)

It was first described Granular Cell Tumor (GCT) as a distinct neurohypophyseal tumor in 1893. It usually presents in middle-aged or older adults. GCTs are suprasellar inorigin. On CT, GCT appears as hyper attenuated lesion

when compared with brain parenchyma, demonstrating homogeneous postcontrast enhancement. On MRI, the lesion demonstrates T1 and T2 isointensity with homogenous or heterogeneous postcontrast enhancement[58].

II. Meningeal infection:

Meningitis is an acute or chronic inflammatory infiltrate of meninges and CSF. Pachymeningitis involves the dura-arachnoid and leptomeningitis affects the pia and subarachnoid spaces. Geographic spread from paranasal sinus, mastoid, or dental infection through valveless emissary veins, hematogenous spread in the context of extracranial infection, and direct inoculation through trauma or surgery may play a role in the development of meningitis. Neisseria meningitidis, Streptococcus pneumoniae, and Hemophilus influenzae are still the three main causes of acute pyogenic meningitis in the 21st century.

Meningitis is a clinical and laboratory diagnosis. Less than half of patients with acute bacterial meningitis present with classic triad of fever, neck stiffness, and altered mental status. Cross-sectional imaging is neither sensitive nor specific for detection of meningitis.

Abnormal enhancement of pia and subarachnoid space (leptomeningeal) is caused by inflammatory breakdown of blood-brain barrier. Thin linear enhancement in cerebral sulci is typical pattern seen in both acute pyogenic (bacterial) and lymphocytic (viral) meningitis, whereas thick nodular enhancement in the basal cisterns is more typical of granulomatous or carcinomatous meningitis. Fungal meningitis produces thicker, lumpy, or nodular enhancement in the subarachnoid

space[7]. Additional MR imaging sequences that may be used for increased sensitivity for leptomeningeal disease include postcontrast T2 FLAIR and delayed postcontrast T1 sequences[8].

MATERIALS AND METHODS

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Source of data

This study was conducted over a period of eighteen months from January 2019 to June 2020 on 64 patients with intracranial lesions who underwent gadolinium contrast enhanced MRI brain in the Department of Radio-diagnosis at R. L. Jalappa Hospital and Research Center attached to Sri Devaraj Urs Medical College, Kolar. Prior informed consent was taken from the patients for their willingness to participate in the study.

Study design: Hospital based prospective observational study.

Sample size:

Sample size could not be estimated as the study was qualitative and done by a single observer. This is also a time bound study.

A total of 64 patients with intracranial lesions were included in the final analysis. The patients were included in the study if they fulfilled the inclusion/exclusion criteria listed below:

Inclusion criteria: All patients with intracranial lesions detected in routine MRI brain study.

Exclusion criteria:

1. History of head injury.
2. Altered renal function test.
3. Allergy to contrast.

Method of collection of data:

This study was approved by the institutional review board and ethical committee. Informed consent was taken from all the patients prior to inclusion in the study. Patients diagnosed with intracranial lesions on CE MRI brain were included in the study.

MRI technique:

Magnetic resonance imaging was performed using 1.5 Tesla, 16 channel, MR Scanner (Magnetom, Avanto®, Siemens, Erlangen, Germany) with the standard head coil. Standard MRI sequences including T1 and T2 WI axial, T1 sagittal, T2 coronal, FLAIR, diffusion weighted imaging (DWI), and susceptibility weighted imaging (SWI) were accquired. Postcontrast sequences obtained were T1 FS (axial, coronal and sagittal), T2 FLAIR, and VIBE.

The study was performed with the patient in supine position with quiet respiration. Base line data and other previous imaging studies & investigations were reviewed in available cases.

Imaging studies was de-identified and reviewed by experienced radiologist. Reviewer was blinded to each subject's diagnosis, clinical characteristics and results of other imaging examination but was aware of age and ethnicity. MR findings and diagnosis was recorded in the proforma. Final diagnosis was obtained by comparing the T1 FS postcontrast & T2 FLAIR postcontrast sequence.

Figure 12: 1.5 Tesla, 16 channel, MR Scanner (Magnetom, Avanto®, Siemens, Erlangen, Germany).

Data analysis

The data were entered in Microsoft excel sheet. The degree of enhancement of each lesion on T1 FS postcontrast sequence and T2 FLAIR postcontrast sequence were tabulated. The results were obtained after comparision of degree of enhancement of the lesion on T1 FS postcontrast sequence and T2 FLAIR postcontrast sequence and were further analysed and concluded.

CLINICAL STUDIES

Bhargava R et al, in his study concluded that detection of lesions depends on various factors such as the size of the lesion, image contrast and location which can be ameliorated by injecting high doses of contrast material that increases the signal of the lesion. The authors were also of the opinion that T1WI are primarily used for post contrast brain MR imaging in the detecting and characterizing intracranial lesions and FLAIR images are equivalent to T2WI except for dark CSF which is due to T1 effect present because of the long T1, there is T1 shortening which is seen as hyperintensity on FLAIR images after administration of gadolinium therefore, lesions showing enhancement on postcontrast T1WI will also enhance on postcontrast FLAIR[4].

William G. Bradley Jr. stated that since T2 prolongation and T1 shortening are synergistic, contrast enhanced FLAIR is more sensitive for subtle abnormalities than either flair alone or postcontrast T1 weighted imaging alone. He also stated that contrast enhanced FLAIR was more advantageous than post contrast T1-weighted imaging alone as it could detect subtle cotrtical abnormalities like leptomeningeal carcinomatosis[60].

Waneerat et al., in his study, concluded that contrast enhanced T1 weighted MR imaging with FS was superior to contrast enhanced FLAIR imaging in majority of the cases where abnormal meningeal enhancement and subarachnoid space lesions in patients with intracranial meningeal disease[61].

Lee et al in his study stated that contrast enhanced FLAIR imaging has many advantages for intracranial disease manifestations, it can be used as a primary or adjunctive sequence to contrast enhanced T1WI in equivocal cases to increase the diagnostic confidence and improve patient care. Contrast enhanced FLAIR imaging is more effective than CE-T1WI because it does not demonstrate enhancement in the normal vascular structures or normal meninges that can be confused with abnormal meningeal enhancement on CE-T1WI[62].

In a study by Vaswani et al., postcontrast FLAIR sequence was more sensitive than postcontrast T1WI for diagnosis of meningitis. The result showed superiority of contrast enhanced FLAIR images in comparison with contrast enhanced spin echo T1WI in detecting abnormal meningeal enhancement[63].

A study conducting by Ercan N et al., concluded that postcontrast FLAIR imaging is a valuable adjunct to post contrast T1WI. Precontrast and postcontrast FLAIR imaging effectively delineates parenchymal metastases particularly leptomeningeal – cisternal and cranial nerve metastases[59.]

RESULTS

RESULTS

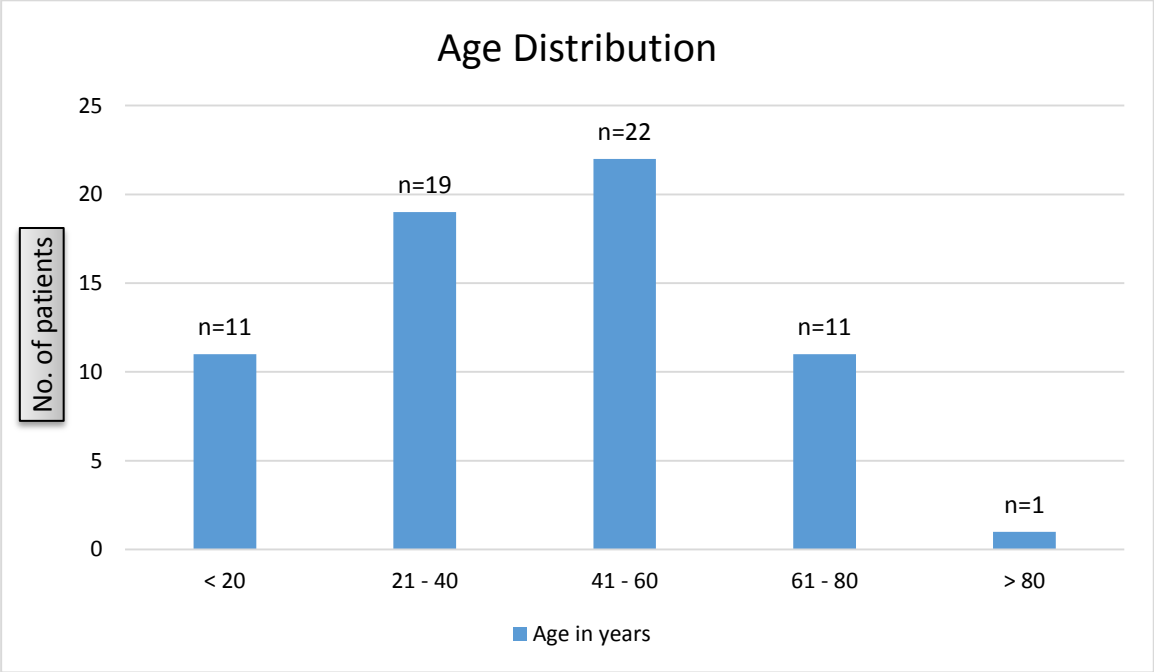
In the study, 64 cases were included in which 29 (45.3 %) were females and 35 (54.7 %) were males (Figure 14; Table 2).

Age distribution:

Commonest age group in our study was 41-60 years (n = 22; 34.4%), followed by 21-40 years (n = 19; 29.7%). Cases with age group of 61-80 years were 17.2% (n= 11), age group of 20 years & below constituted 17.2% (n =11) and cases aged more than 80 years constituted 1.5% (n=1) (Figure 14; Table 2). The mean age of the cases was 43 ± 20 years (mean ± SD) with range of 1 to 90 years.

Table 2: Age distribution

Age group	Number of cases	%
0-20 years	11	17.2
21-40 years	19	29.7
41-60 years	22	34.4
61-80 years	11	17.2
81 years	1	1.5
Total	64	100



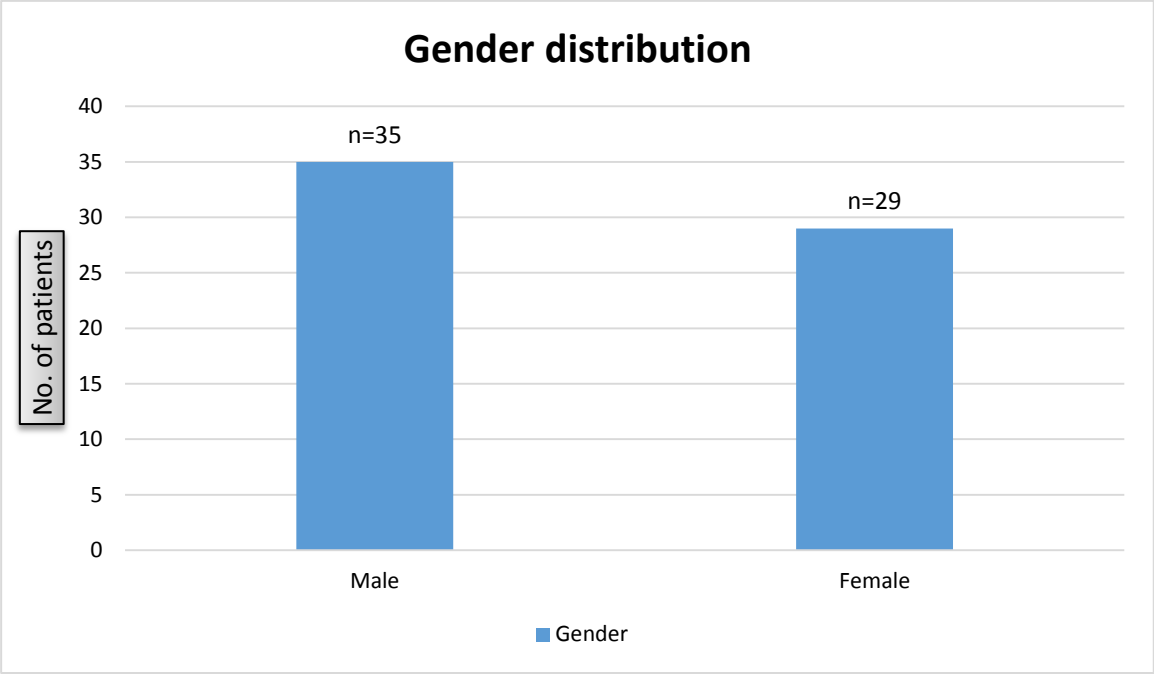
(Figure13 - Age group distribution)

Gender distribution:

Out of 64 cases, there were total 29 females and 35 males (Table 3 & Figure 14).

Table 3: Gender distribution

Gender distribution	Number of cases	%
Male	35	54.7
Female	29	45.3
Total	64	100



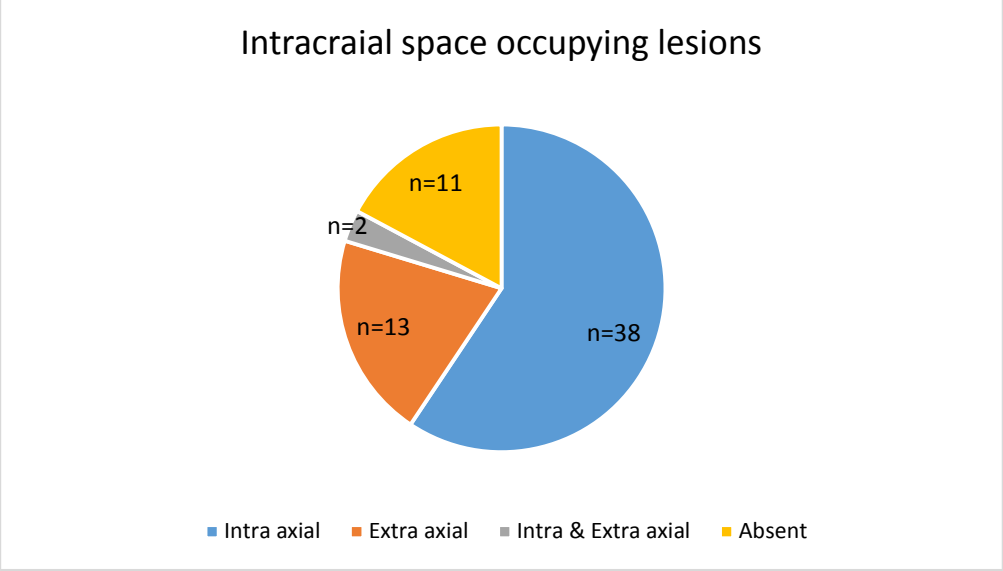
(Figure 14- Gender distribution)

Intracranial space occupying lesions (ICSOL):

In our study, out of 64 cases; 38 cases had intra axial lesions, 13 cases had extra axial lesions, 2 cases had both intra & extra axial lesions and 11 cases had no intracranial space occupying lesions (Table 4 & Figure 15).

Table 4: Intracranial space occupying lesions.

ICSOL	Number of cases	%
Intra axial	38	59.4
Extra axial	13	20.3
Intra and extra axial	2	3.1
Absent	11	17.2
Total	64	100



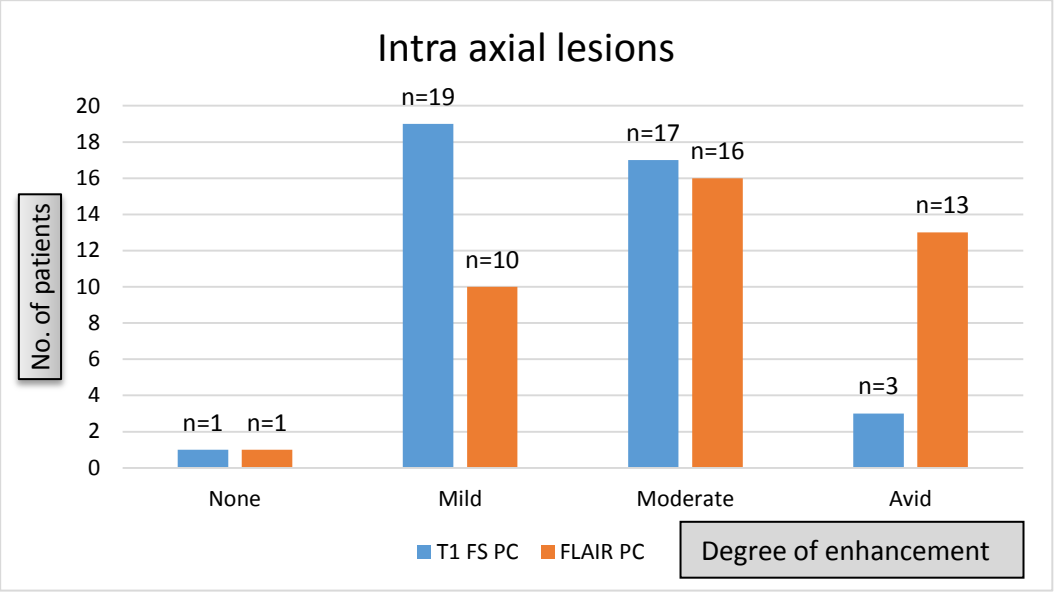
(Figure 15 – Intracranial space occupying lesions)

1. Intra-axial lesions:

On final diagnosis out of 64 cases, 40 had intra-axial lesions. On T1 FS PC sequence, 19 lesions demonstrated mild enhancement, 17 lesions demonstrated moderate enhancement, 3 lesions demonstrated avid enhancement and 1 lesion showed no enhancement. On FLAIR PC sequences, 10 lesions demonstrated mild enhancement, 16 lesions demonstrated moderate enhancement, 13 lesions demonstrated avid enhancement and 1 lesion showed no enhancement. (Table 5; Figure16)

Table 5: Degree of enhancement of intra-axial lesions

Degree of enhancement of intra-axial lesions	Postcontrast enhancement (No of lesions; %)	
	T1 FS PC	T2 FLAIR PC
None	1 (2.5%)	1 (2.5%)
Mild	19 (47.5%)	10 (25%)
Moderate	17 (42.5%)	16 (40%)
Avid	3 (7.5%)	13 (32.5%)
Total	40 (100%)	40 (100%)



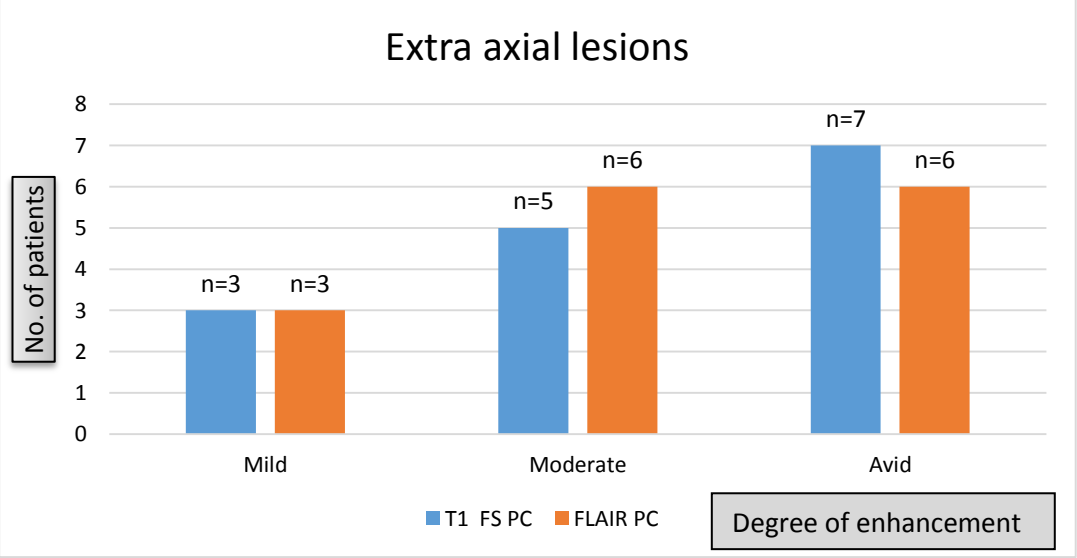
(Figure 16- Degree of enhancement in Intra-axial lesions)

2. Extra-axial lesions:

On final diagnosis out of 64 cases, 15 cases had extra-axial lesions. On T1 FS PC sequence, 3 lesions demonstrated mild enhancement, 5 lesions demonstrated moderate enhancement and 7 lesions demonstrated avid enhancement. On FLAIR PC sequences, 3 lesions demonstrated mild enhancement, 6 lesions demonstrated moderate enhancement and 6 lesions demonstrated avid enhancement. (Table 6; Figure 17)

Table 6: Degree of enhancement of extra-axial lesions

Degree of enhancement of extra-axial lesions	Postcontrast enhancement (No of lesions; %)	
	T1 FS PC	T2 FLAIR PC
Mild	3 (20%)	3 (20%)
Moderate	5 (33.3%)	6 (40%)
Avid	7 (46.7)	6 (40%)
Total	15 (100%)	15 (100%)



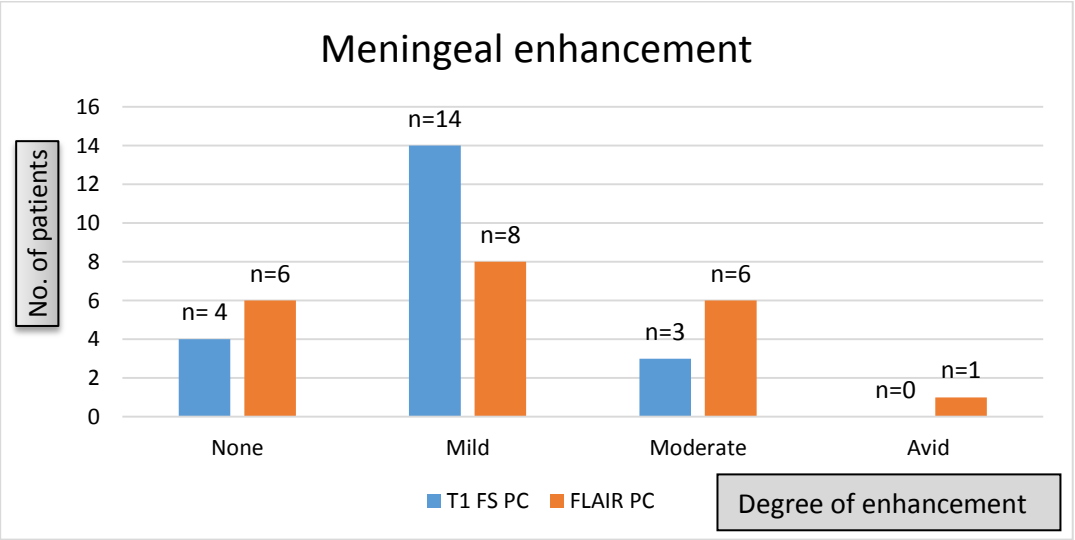
(Figure 17- Degree of enhancement in Extra-axial lesions)

3. Meningeal involvement:

On final diagnosis out of 64 cases, 21 had meningeal enhancement. On T1 FS PC sequence - 4 lesions showed no enhancement, 14 lesions demonstrated mild enhancement, 3 lesions demonstrated moderate enhancement and no lesions demonstrated avid enhancement. On FLAIR PC sequences - 1 lesion showed no enhancement, 6 lesions demonstrated mild enhancement, 8 lesions demonstrated moderate enhancement and 6 lesions demonstrated avid enhancement. (Table 7; Figure 18)

Table 7: Degree of meningeal enhancement

Degree of meningeal enhancement	Postcontrast enhancement (No of lesions; %)	
	T1 FS PC	T2 FLAIR PC
None	4 (19%)	1 (4.7%)
Mild	14 (66.7%)	6 (28.6%)
Moderate	3 (14.3%)	8 (38.1%)
Avid	0 (0)	6 (28.6%)
Total	21 (100%)	21(100%)



(Figure 18: Degree of meningeal enhancement)

IMAGES

IMAGES

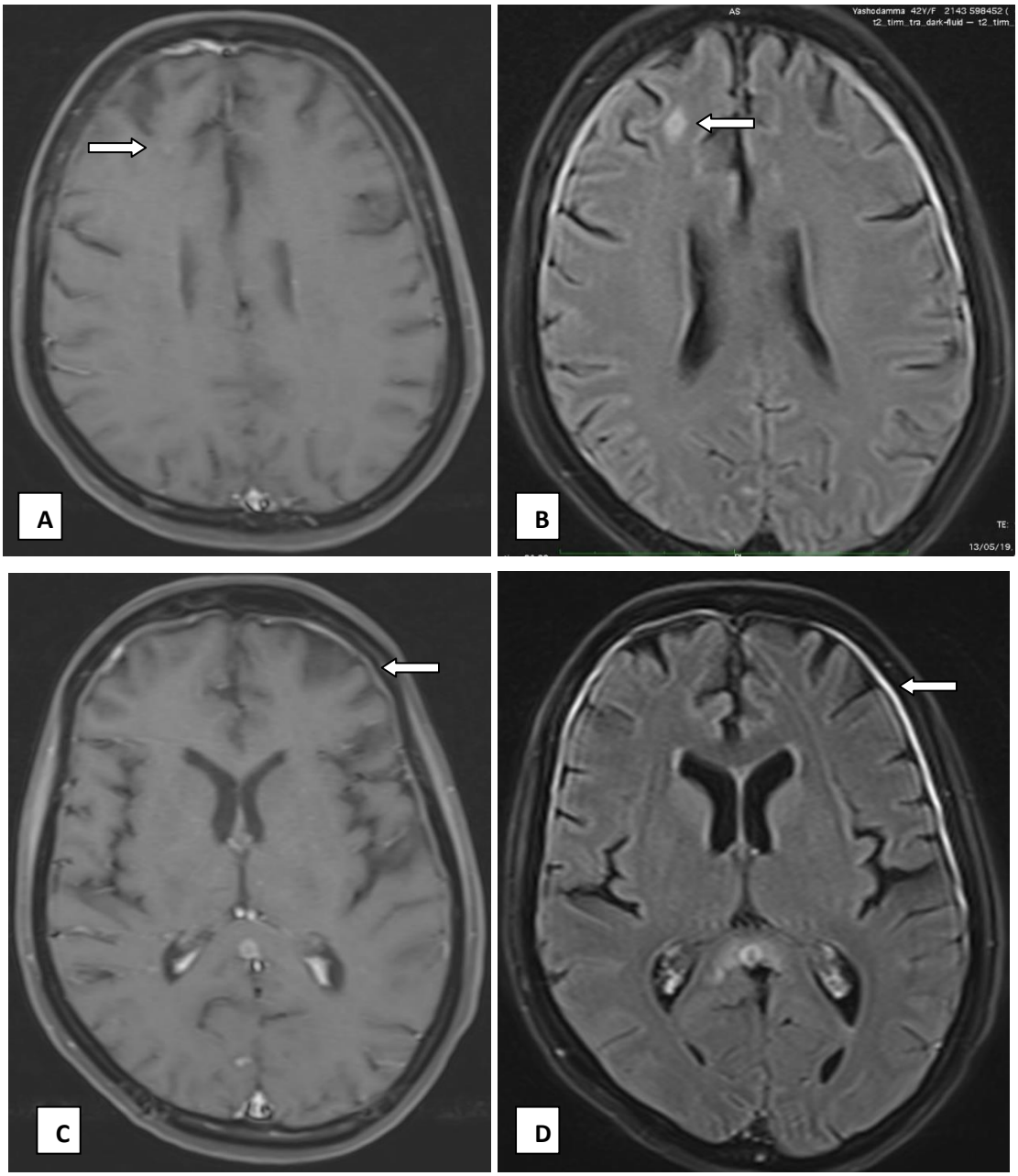


Figure19: (A) T1 FS PC sequence shows a subcentimetric lesion with faint ring enhancement in right frontal lobe & same lesion on (B) T2 FLAIR PC sequence shows mild ring enhancement. (C) T1 FS PC sequence shows mild meningeal enhancement along the bilateral frontal convexities whereas on (D) T2 FLAIR PC sequence avid enhancement of meninges is seen. Therefore, in this case, T2 FLAIR PC sequence proved to be superior to T1 FS PC sequence.

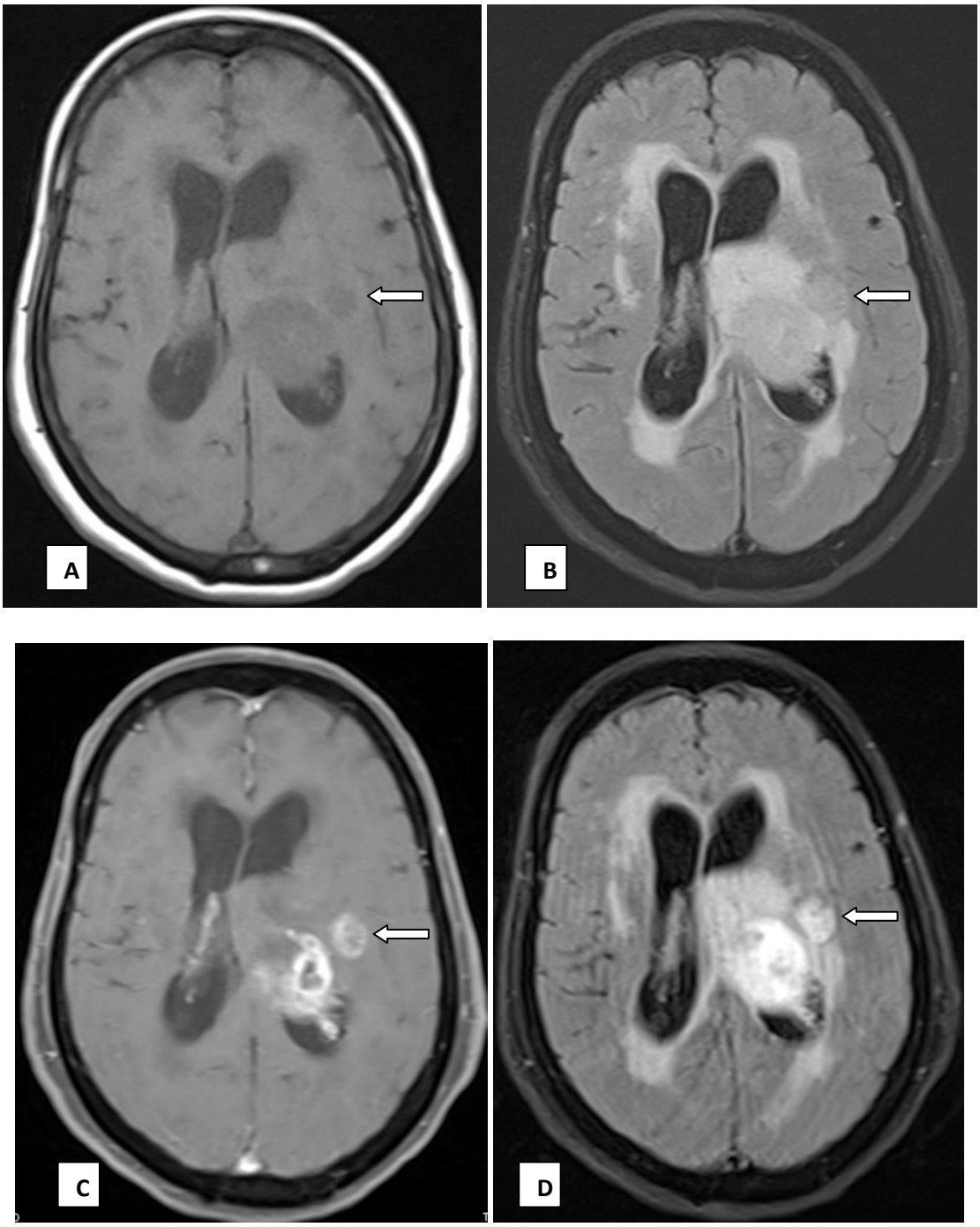


Figure 20: MRI Brain axial sections of 79-year-old female diagnosed with glioblastoma showing a fairly well-defined, T1 isointense (A) & (B) T2 FLAIR heterogeneously hyperintense lesion demonstrating mild heterogenous enhancement on (C) T1 FS PC & moderate heterogenous enhancement on (D) T2 FLAIR PC images.

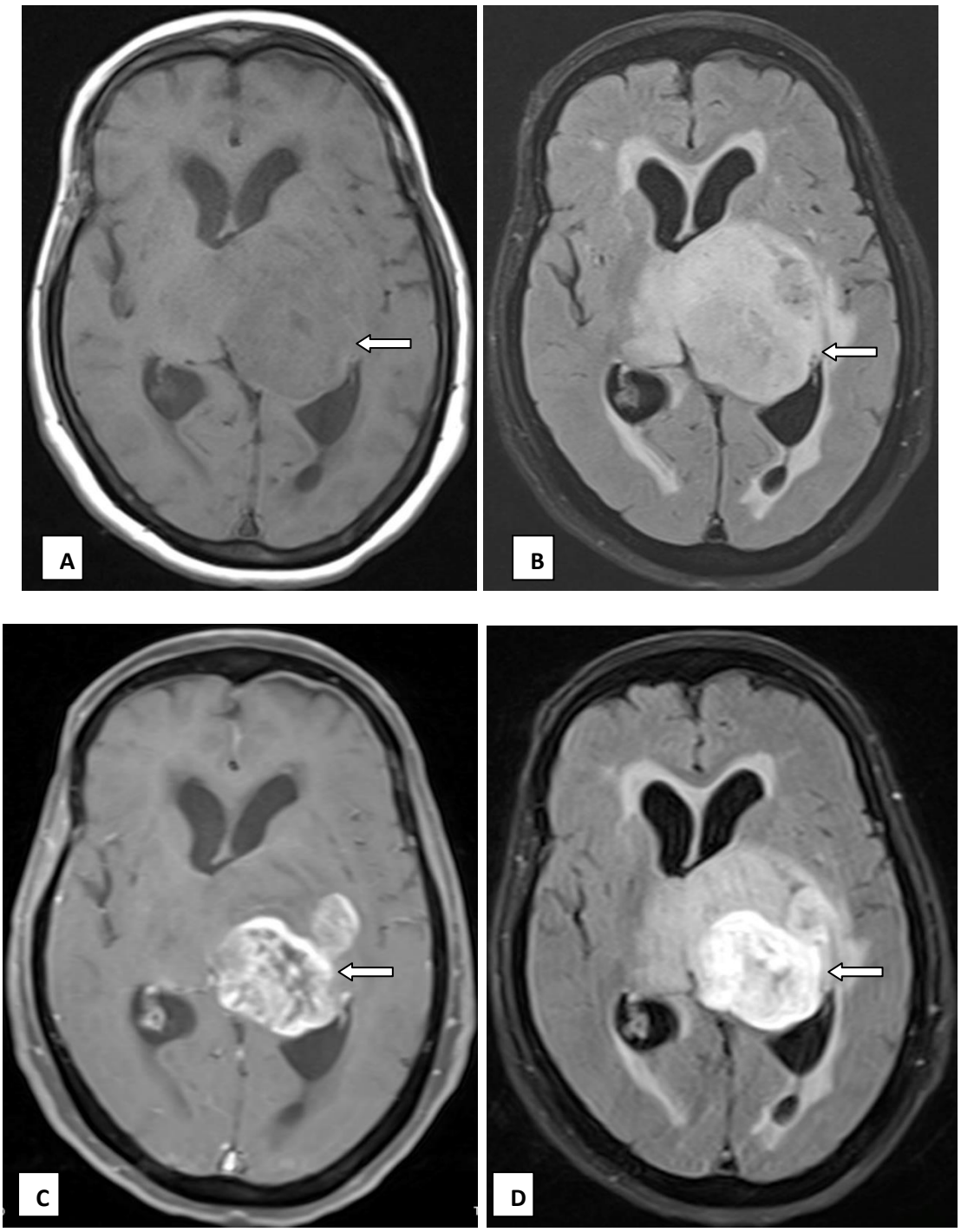


Figure 21: MRI Brain axial sections of 79-year-old female diagnosed with glioblastoma showing a well-defined, T1 isointense (A) & (B) T2 FLAIR heterogeneously hyperintense lesion demonstrating moderate heterogenous enhancement on (C) T1 FS PC & avid heterogenous enhancement on (D) T2 FLAIR PC images,with peri lesional edema better depicted in the latter image.

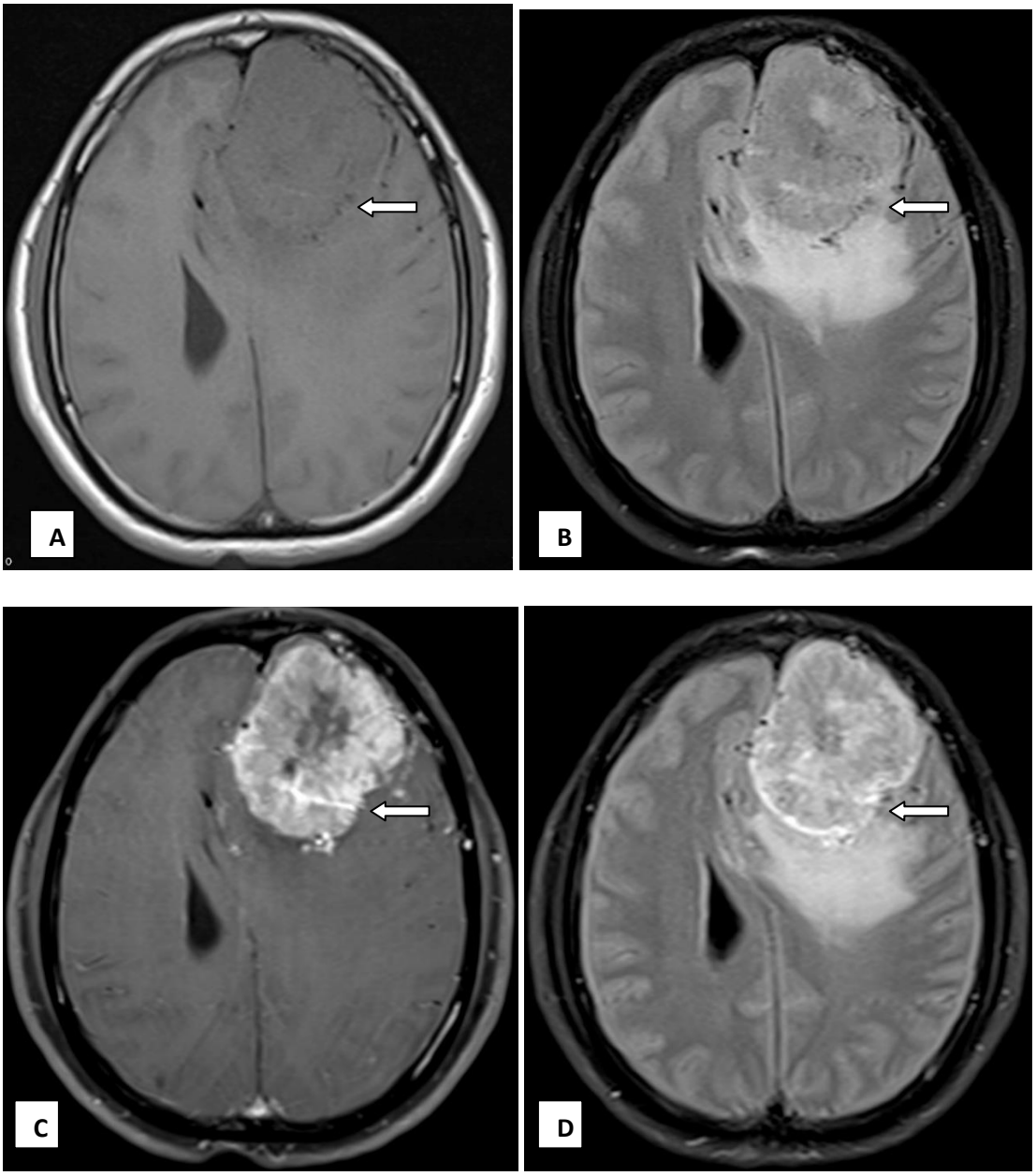


Figure 22: MRI Brain axial sections of 42 year old male diagnosed with meningioma in right frontal region showing a well-defined, T1 isointense (A) & (B) T2 FLAIR heterogeneously hyperintense lesion demonstrating heterogenous enhancement of lesion which is better depicted on (C) T1 FS PC than (D) T2 FLAIR PC images. The central necrotic component is also better demonstrated in the T1 FS PC images.

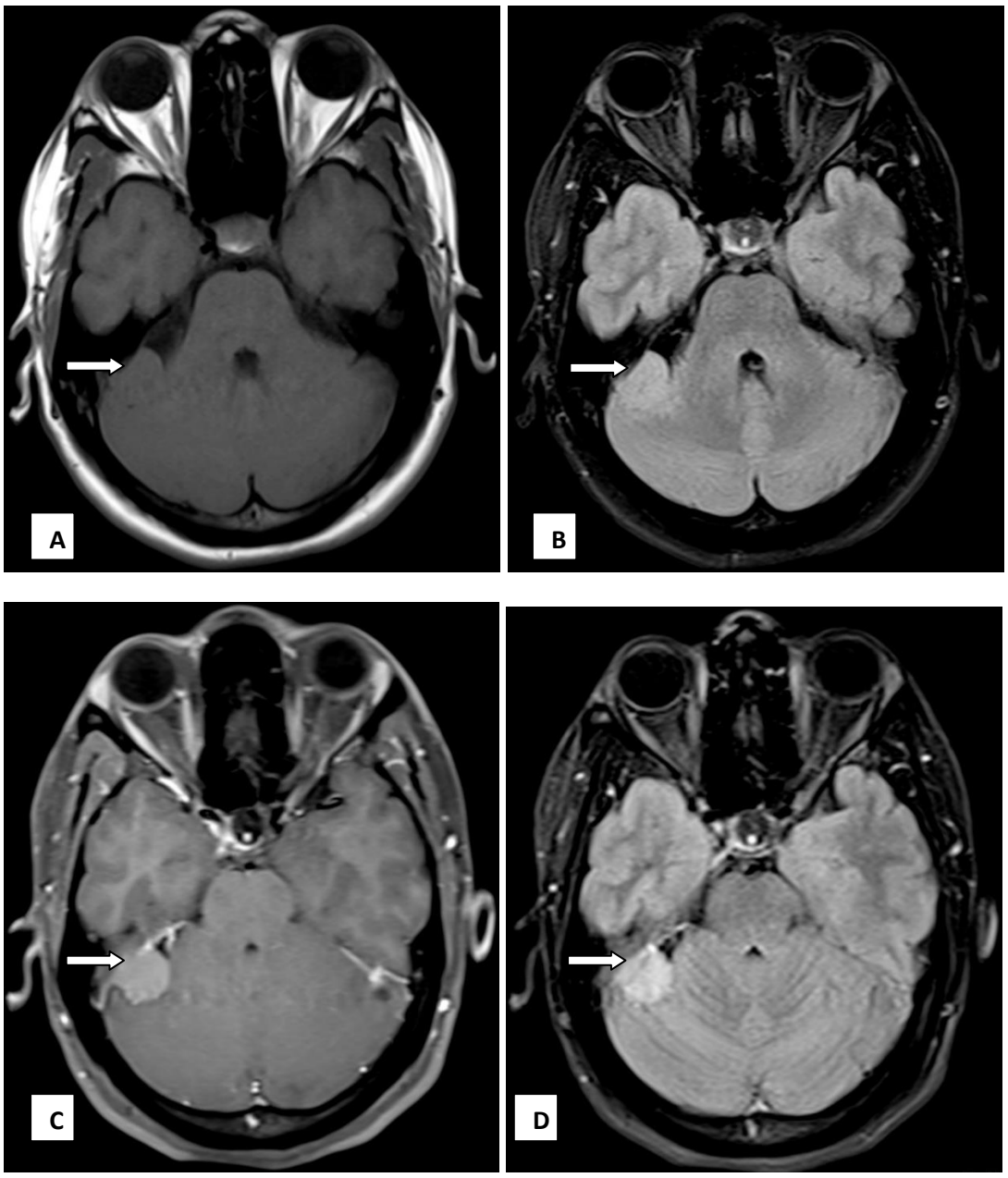


Figure 23: MRI Brain axial sections of 29 year old female diagnosed with right cerebellopontine angle meningioma showing a well-defined , T1 isointense (A) & (B) T2 FLAIR heterogeneously hyperintense lesion demonstrating mild enhancement on (C) T1 FS PC & moderate enhancement on (D) T2 FLAIR PC images.

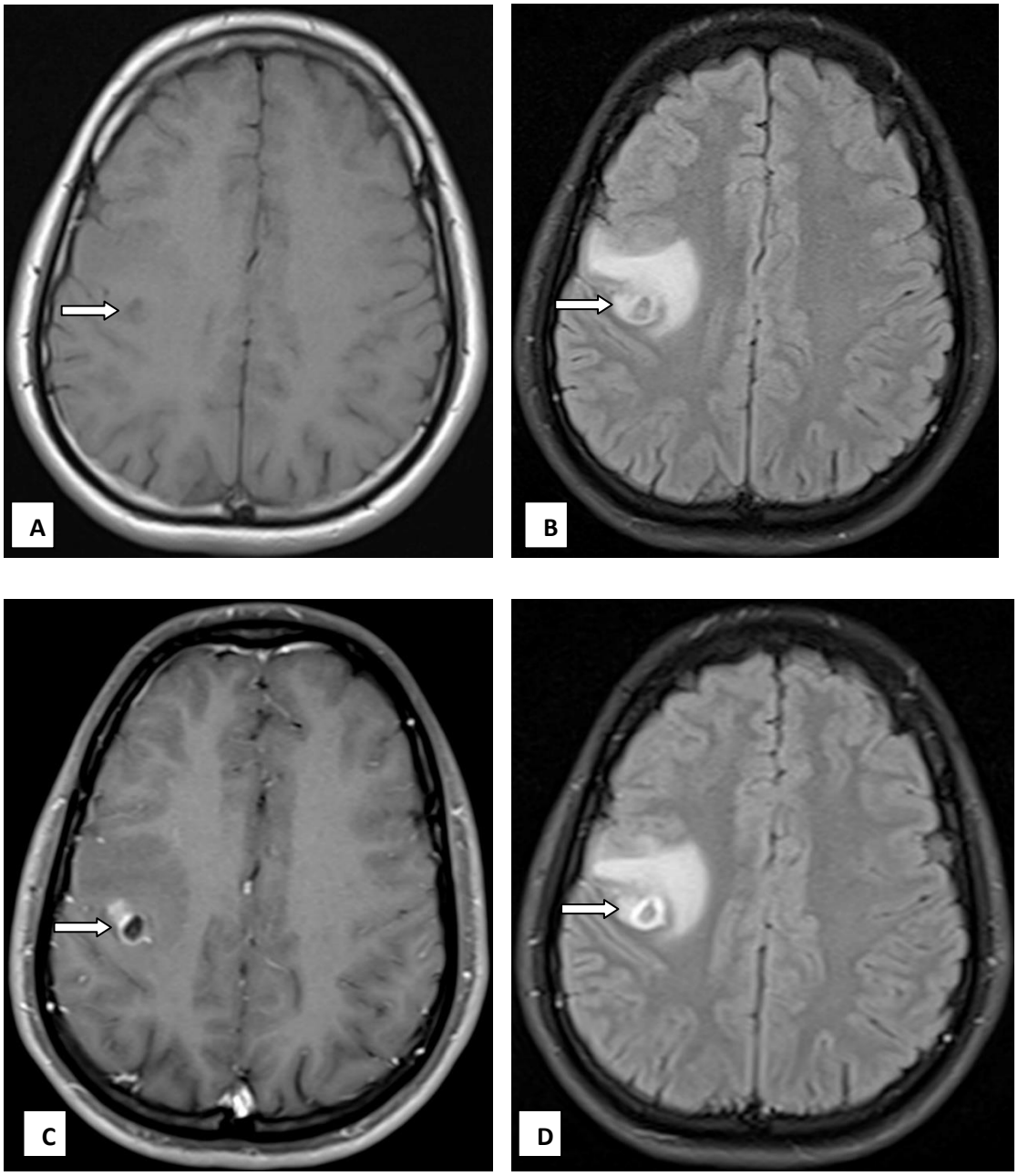


Figure 24: MRI Brain axial sections of 21-year-old male diagnosed with neurocysticercosis shows hypointense lesion on (A) T1WI. On (B) T2 FLAIR image a hypointense lesion with hyperintense rim and perilesional edema is noted. The lesion demonstrates moderate ring enhancement on (C) T1 FS PC and avid enhancement on (D) T2 FLAIR PC image. The extent of perilesional edema is better appreciated on T2 FLAIR PC image than T1 FS PC image.

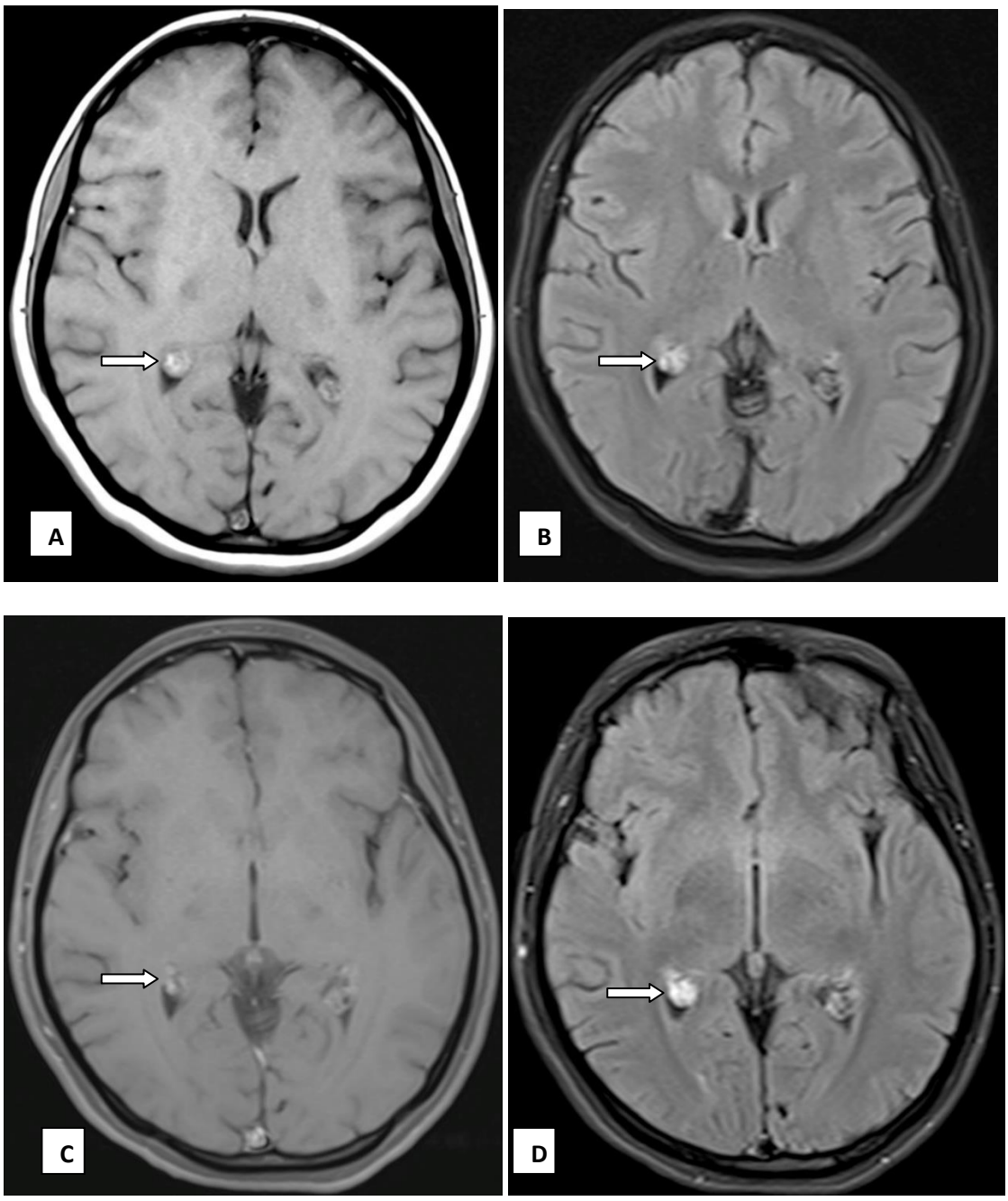


Figure 25: A 20-year-old female diagnosed with choroid plexus xanthogranuloma. MRI Brain axial sections (A) T1WI & (B) T2 FLAIR shows heterogeneously hyperintense lesion in occipital horn of right lateral ventricle. This lesion demonstrates mild enhancement on (C) T1 FS PC image & avid enhancement on (D) T2 FLAIR PC image.

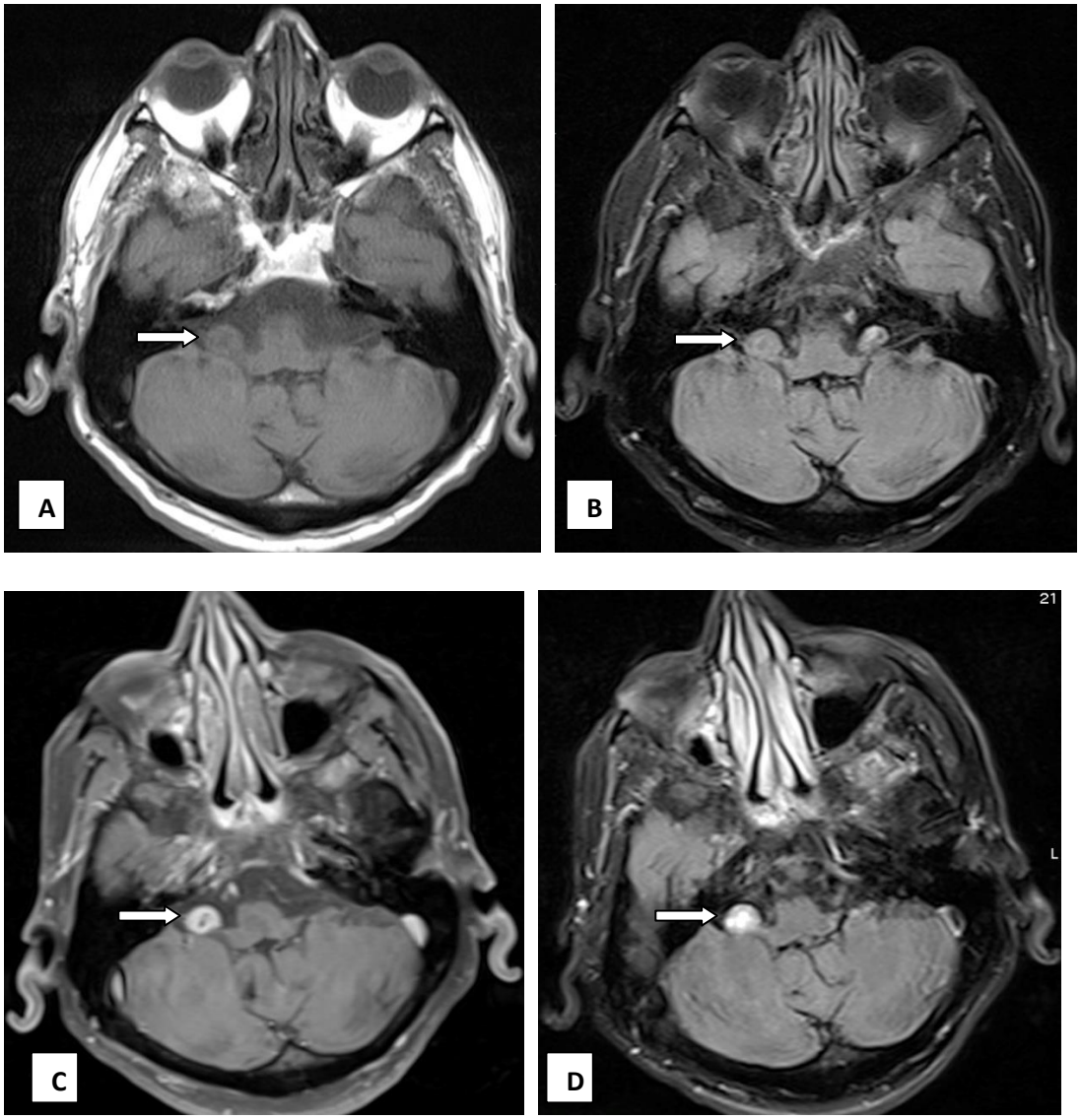


Figure 26: MRI brain axial sections of a 74-year-old male presented with tinnitus and post imaging was diagnosed with vestibular schwannoma on right side. (A) T1WI & T2 FLAIR image showed a well circumscribed, heterogeneously hypointense lesion in right cerebello-pontine angle. The lesion demonstrates better enhancement on (D) T2 FLAIR PC image than (C) T1 FS PC. The limitation of the study seen in this case: Cystic component in the lesion shows hyperintensity which makes it difficult to differentiate from enhancement.

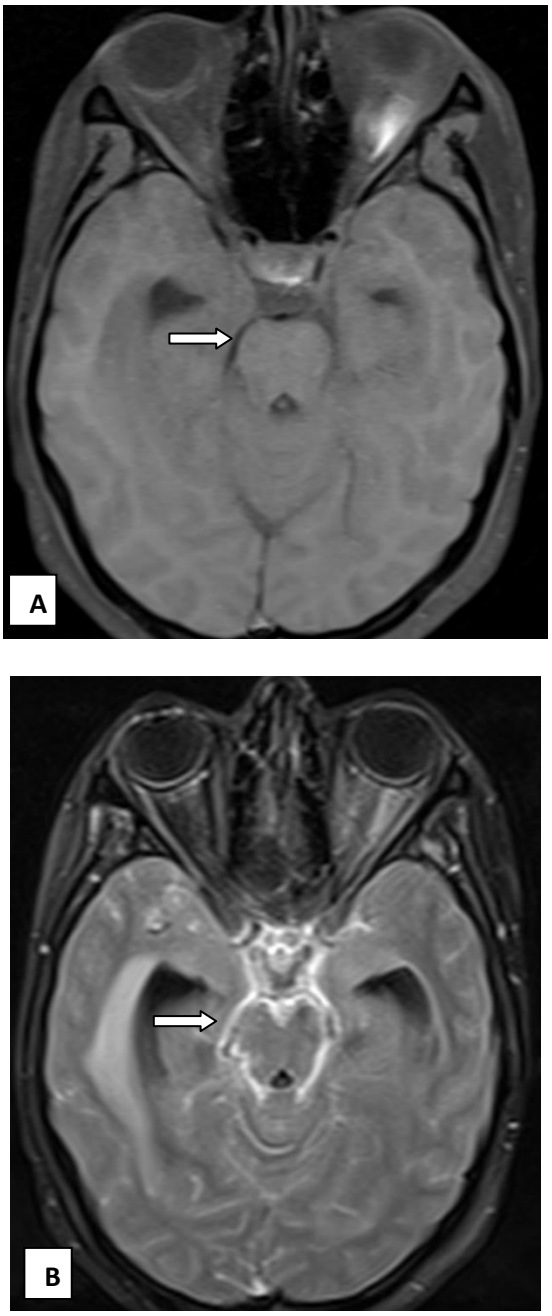


Figure 27: MRI Brain axial sections of 22 years old female with meningoencephalitis. (A) T1 FS PC shows no meningeal enhancement but avid meningeal enhancement is noted on (B) T2 FLAIR PC sequence giving "sugar coated" appearance.

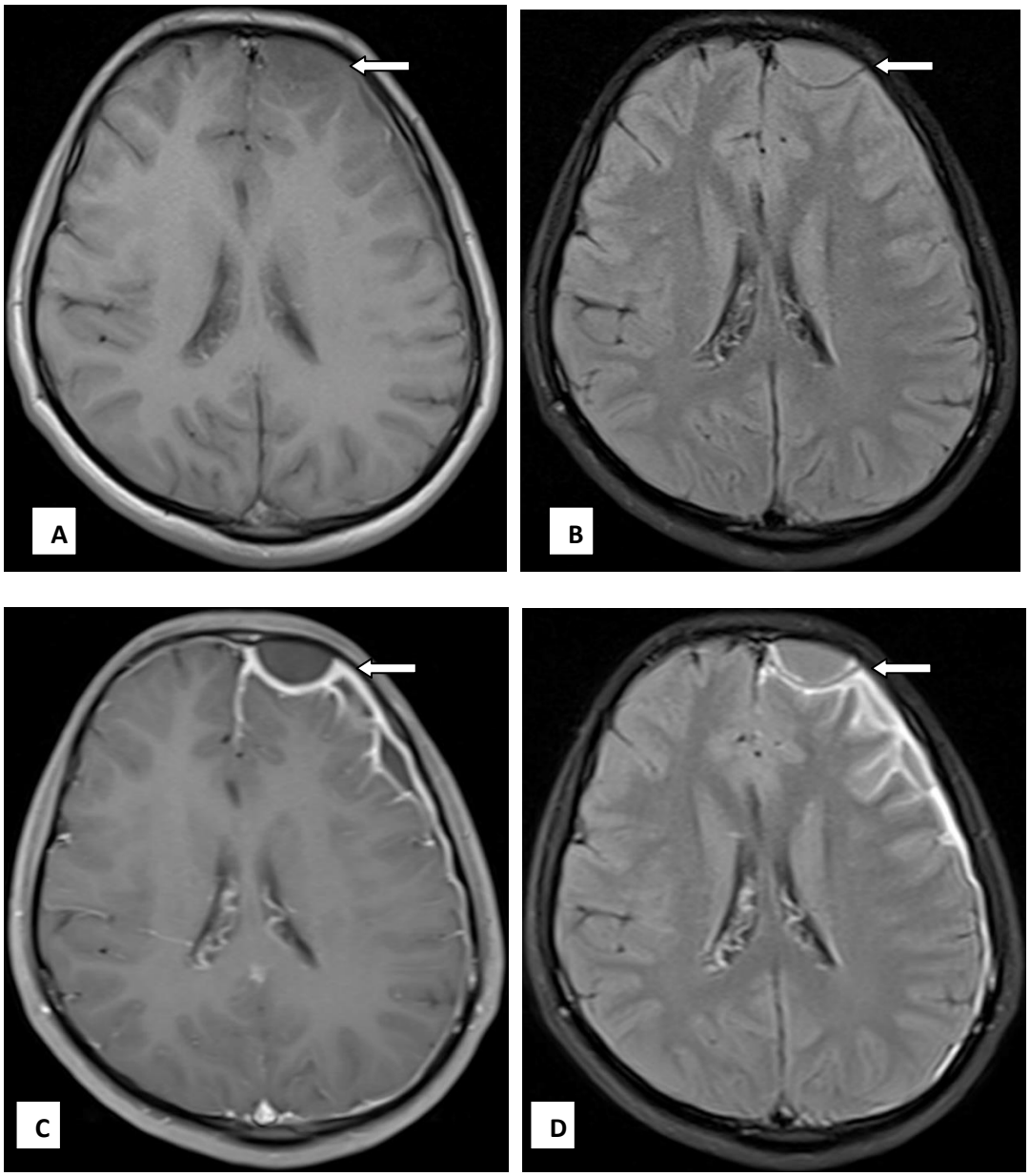


Figure 28: An 18-year-old male patient presented with headache and was diagnosed with epidural abscess & meningitis. MRI axial sections (A) T1WI & (B) T2 FLAIR sequences demonstrated hypo-isointense lesion with faint peripheral hyperintense rim in left frontal region. (C) T1 FS PC & (D) T2 FLAIR PC sequences showed hypoenhancing lesion with peripheral enhancement along with pachymeningeal & leptomeningeal enhancement. The margins of the lesion better depicted on T2 FLAIR PC sequence compared to that of T1 FS PC sequence. Enhancement of meninges is superior on T2 FLAIR PC than T1 FS PC sequences.

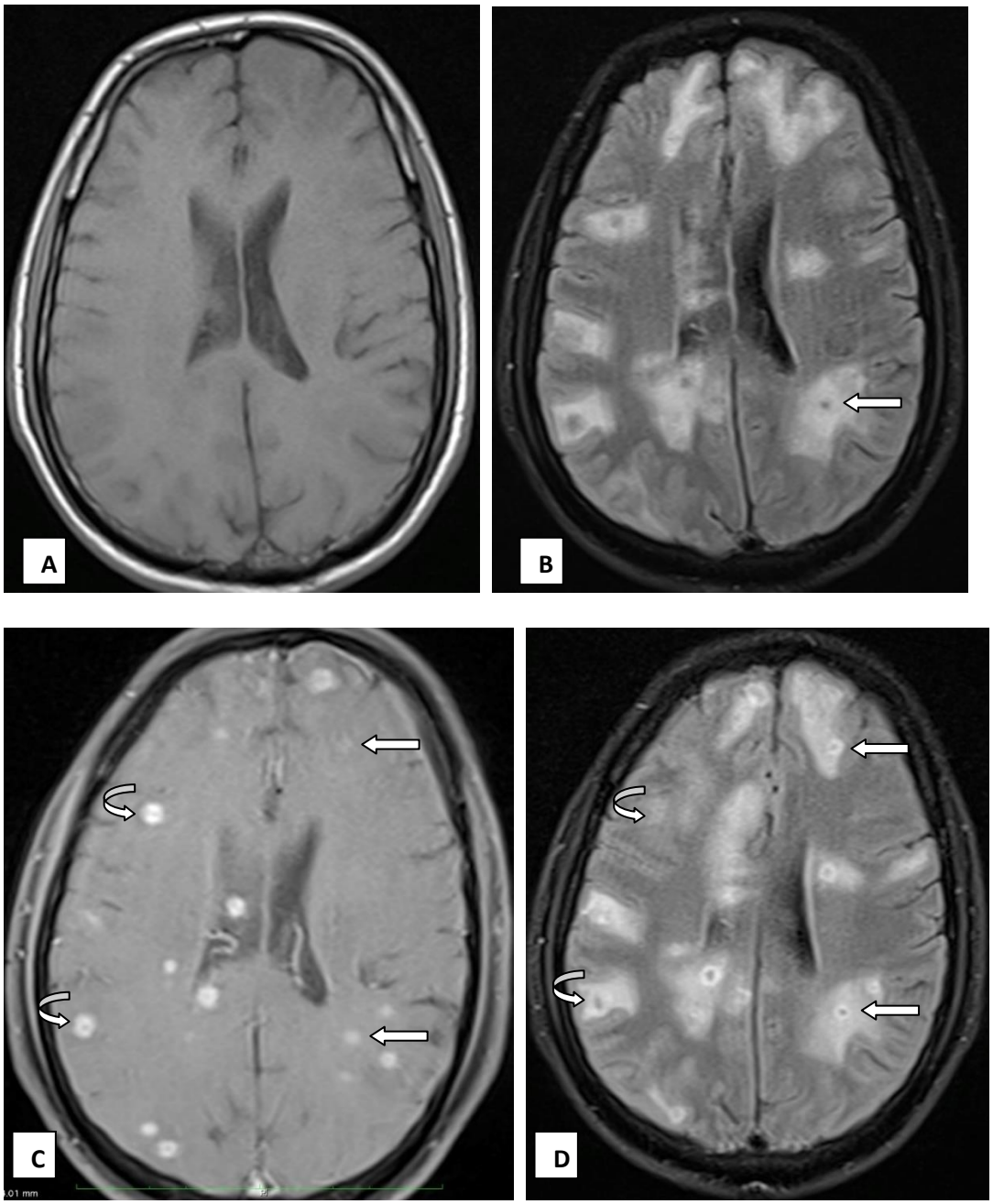


Figure 29: A case of 54-year-old male with disseminated tuberculoma. MRI axial images: (A) T1WI reveals no abnormality. (B) T2 FLAIR revealed multiple, well defined, subcentimetric hypointense lesions with perilesional edema. (C) T1 FS PC & (D) T2 FLAIR PC images demonstrated multiple, ring enhancing lesions with perilesional edema. In comparison with T1 FS PC sequence, T2 FLAIR PC sequence shows better contrast enhancement of few lesions and perilesional edema (Arrow mark). In comparison with T2 FLAIR PC sequence, T1 FS PC sequence shows better contrast enhancement of few lesions (Curved arrow).

DISCUSSION

DISCUSSION

Diagnosing an intracranial lesion as benign or malignant is clinically important because it can guide in management of the patient. Addition of T2 FLAIR postcontrast sequence in routine MRI protocol helps in better characterization of a lesion.

In our study we reviewed 64 cases. The most common age group in our study was 41-60 years (34.4%), followed by 21 to 40 years (29.7%) with a mean age of 43 ± 20 years (mean ± SD) with a range 1 to 90 years. Our results were similar to a study by Bhargava R. et al who reported the commonest age with intracranial lesions to be 24-81 years of age[4].

The incidence of intracranial lesion was more in males which were in correlation with a study conducted by Bhargava R. et al which showed incidence of intracranial lesions to be more in males compared to females[4].

In our study, more than half the intracranial lesions were intra-axial (59.4%), 20.3% were extra-axial, 2% had both intra and extra-axial lesions. A similar observation was also made by Bhargava R. et al also who reported that more than half of intracranial lesions were intra-axial (71.4%)[4].

In a case of neurocysticercosis, T2 FLAIR PC sequence proved its higher ability than T1 FS PC sequence in detecting the number of lesions. In a case of metastasis with a lesion size less than one centimetre, the lesion was better appreciated on T2 FLAIR PC sequence which was not visualized on T1 FS PC

sequence. This observation was comparable with a study done by Ercan et al.,that stated T2 FLAIR PC sequence is better in comparison to T1 FS PC sequence in detecting the number of lesions may be due to delayed enhancement[59].

Intra-axial lesions showed better enhancement on T2 FLAIR postcontrast sequence in comparison with T1 FS postcontrast sequence in 28 cases (70%), equal enhancement on T1 FS postcontrast sequence and T2 FLAIR postcontrast sequence in 1 case (2.5%) and better enhancement on T1 FS postcontrast sequence in comparison with the T2 FLAIR postcontrast sequence in 11 cases (27.5%). The majority of lesions that depicted better enhancement on T1 FS PC sequence than T2 FLAIR PC sequence were lesion of more than one centimetre size.

A study conducted by Bhargava R. et al. found ring enhancement in neurocysticercosis and tuberculoma. It was similar to the imaging findings in our study where neurocysticercosis and tuberculoma cases demonstrated ring enhancement. In our study, a case of NCC demonstrated equal enhancement on T1 FS PC and T2 FLAIR PC sequences; however 3 cases demonstrated better contrast enhancement on T2 FLAIR PC sequence. This was correlating with findings of a study conducted by Bhargava R. et al where almost all lesions demonstrated almost equal signal intensity; however seven lesions showed better T2 FLAIR contrast enhancement than surrounding perilesional edema[4].

Bhargava et al also stated that extent of edema was better seen on FLAIR which was correlating with our study[4].

Extra-axial lesions showed better enhancement on T2 FLAIR postcontrast sequence in comparison with the T1 FS postcontrast sequence in 7 cases (46.67%), equal enhancement on T1 FS postcontrast sequence and T2 FLAIR postcontrast sequence in 2 cases (13.33%) and better enhancement on T1 FS postcontrast sequence in comparison with T2 FLAIR postcontrast sequence in 6 cases (40%). According to Bhargava R. et al study, extra-axial lesions showed better postcontrast characterisation on T1 FS PC compared to T2 FLAIR PC. It was contrary to the findings of our study where T2 FLAIR PC sequence was mildly superior to T1 FS PC in demonstrating the characteristics of extra-axial lesions[4].

Few lesions in our study depicted that, solid component of the lesions showed better enhancement on T2 FLAIR postcontrast sequence. This was a similar finding in a study conducted by Bhargava R. et al which also stated that solid component enhancement was better on T2 FLAIR PC than T1 FS PC sequences[4].

Bhargava R. concluded that parenchymal metastatic lesions showed similar enhancement on both T1 FS PC and T2 FLAIR PC sequences[4]. This finding was contrary with the findings in our study i.e, one metastatic lesion with edema showed better postcontrast characterisation on T1 FS sequence than T2 FLAIR sequence, another necrotic metastatic lesion demonstrated better enhancement on T1 FS PC sequence than T2 FLAIR PC sequence and a small sized metastasis showed better enhancement on T2 FLAIR PC sequence than T1 FS PC sequence. However, in other 4 cases of metastases in our study demonstrated better postcontrast characterisation of lesion on T2 FLAIR sequence compared to T1 FS sequence.

William G. et al., stated that T2 FLAIR PC sequence was better than T1 PC sequence in detection of subtle cortical lesion which was also observed in our study[60]. A case of cerebral toxoplasmosis showed no enhancement on T1 FS PC sequence but showed moderate enhancement on T2 FLAIR PC sequence.

In our study, it was also observed that in a case of Glioblastoma with post-radiation therapy, there was superior characterisation of lesion on T2 FLAIR PC sequence than T1 FS PC sequence.

A study conducted by Lee et al., was also comparable with our study which opined regarding the necessity of precontrast and postcontrast T2 FLAIR sequences for hyperintense intracranial lesions. Hence, due to the lesion hyperintensity, the postcontrast enhancement assessment of the lesion is suboptimal. Therefore, this is one of the limitations of this study[62].

In our study, meningeal abnormalities were better delineated on T2 FLAIR postcontrast sequence in comparison with T1 FS postcontrast sequence in 17 cases (80.95%), equal on T1 FS postcontrast & T2 FLAIR postcontrast sequence in 1 case (4.76%) and better delineated on T1 FS postcontrast sequence in comparison with T2 FLAIR postcontrast sequence in 3 cases (14.29%). A study done in Korea by Lee E. J. et al., concluded that T2 FLAIR PC sequence was very sensitive in detecting the meningeal enhancement than T1 FS PC sequence. In our study, this conclusion was proved to be correct as majority of cases showed better meningeal enhancement on T2 FLAIR PC sequence than T1 FS PC sequence[62].

CONCLUSION

CONCLUSION

In our study, we concluded that T2 FLAIR PC sequence acts as an adjuvant to T1 FS PC sequence and plays a major role in characterization of the lesion. Diagnostic accuracy has increased significantly with addition of T2 FLAIR PC sequence to routine MRI protocol. Our study showed that, there is significant contribution of T2 FLAIR PC sequence in detecting subtle cortical & subcentimetric lesions, in assessing the extent of perilesional edema and in diagnosing meningeal pathology.

Our study also found that, T2 FLAIR has significant role in characterizing a lesion. By performing T2 FLAIR PC sequence, it added additional information like perilesional edema, solid component enhancement, delayed enhancement of lesions etc., which inturn helped in narrowing down to the differential diagnosis with high diagnostic accuracy and planning the management of the patient.

As per our study, a new MRI protocol including postcontrast T2 FLAIR sequence is recommended in evaluation of intracranial lesions, especially in cases involving meninges and cases with subtle cortical abnormalities. However, T2 FLAIR PC sequence cannot replace T1 FS PC sequence but can be added as adjunct along with T1 FS PC sequence in detection and describing the characteristics of lesion due to limitations of the T2 FLAIR PC sequence like timing of the sequence done after contrast injection which sometime results in suboptimal assessment of enhancement of lesion due to contrast wash out, in cases with hyperintense lesions where it is difficult to differentiate between the increased signal intensity of lesion & degree of

contrast enhancement and perilesional edema which will obscure the wall enhancement of the lesion. T2 FLAIR PC sequence should be performed in evaluation of intracranial lesions, whenever the facility is available.

SUMMARY

SUMMARY

Intracranial lesions can be of varied aetiology like infectious, neoplastic and inflammatory. Establishing an accurate neoplastic aetiology is essential for timely diagnosis and neurosurgical intervention. Most of the patients with neoplasms have a fairly characteristic presentation. However, many patients with intracranial masses present a greater diagnostic challenge because of atypical presentation secondary to intratumoural haemorrhage, arterial occlusion and cerebral infarction or tumour involvement of silent areas. In such cases it is important to utilize modern neuro-radiological imaging techniques like CT scan and MRI in order to detect the lesion to localize it. The present study attempts to provide preliminary data on characterization of the lesion on postcontrast sequences.

In this study 64 patients with intracranial lesions on MRI were evaluated, which was conducted over a period of 18 months from January 2019 to June 2020 of various age groups (1-90 years). The study subjects were predominantly males with peak age incidence in 41-60 years. T2 FLAIR postcontrast sequence was added to our routine MRI protocol.

This study included various infective etiologies like neurocysticercosis, tuberculoma, cerebral toxoplasmosis, meningitis, epidural abscess and neoplastic etiologies like meningiomas, glioblastoma, low grade astrocytoma, hemorrhagic metastases, parenchymal metastases, clivalchordoma, vestibular schwannoma, hemangioblastoma, ependymoma etc. CE-MRI Brain was performed and characteristic enhancement of lesions on T1 FS postcontrast and T2 FLAIR postcontrast sequences were assessed and documented. Comparison of lesion

enhancement on T1 FS and T2 FLAIR postcontrast sequences were done. The advantages and the limitations of T2 FLAIR postcontrast sequence were derived. After obtaining the results, it was concluded that T2 FLAIR postcontrast sequence cannot be used as a replacement for T1 FS postcontrast sequence but instead it can be used as an adjunct to T1 FS postcontrast sequence.

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ANNEXURES

STUDY TITLE: COMPARISION OF T1 FAT SATURATED (FS) POSTCONTRAST AND T2 FLUID ATTENUATED INVERSION RECOVERY (FLAIR) POSTCONTRAST SEQUENCES IN EVALUATION OF INTRACRANIAL LESIONS.

INFORMED CONSENT FORM

PG guide's name: Dr. N. RACHEGOWDA

PROFESSOR & HEAD,

DEPT. OF RADIO-DIAGNOSIS

Principal investigator: Dr. SAHANA N. GOWDA

Name of the subject:

Age :

Gender :

a. I have been informed in my own language that this study involves CE-MRI and use of contrast material as part of procedure. I have been explained thoroughly and understood its complication and possible side effects.
b. I understand that the medical information produced by this study will become part of institutional record and will be kept confidential by the said institute.
c. I understand that my participation is voluntary and may refuse to participate or may withdraw my consent and discontinue participation at any time without prejudice to my present or future care at this institution.
d. I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s).

e. I confirm that _____ (name of PG guide) has explained to me the purpose of research and the study procedure that I will undergo and the possible risks and discomforts that I may experience, in my own language. I hereby agree to give valid consent to participate as a subject in this research project.

Participant's signature/thumb impression

Signature of the witness: Date:

1)

2)

I have explained to _____ (subject) the purpose of the research, the possible risk and benefits to the best of my ability.

Chief Researcher/ Guide signature Date:

COMPARISION OF T1 FAT SUPPRESED (FS) POSTCONTRAST AND T2 FLUID ATTENUATED INVERSION RECOVERY (FLAIR) POSTCONTRAST SEQUENCES IN EVALUATION OF INTRACRANIAL LESIONS.

PATIENT INFORMATION SHEET

Principal Investigator: Dr. SAHANA N. GOWDA / Dr. N. RACHEGOWDA

I, Dr. Sahana N. Gowda, post-graduate student in Department of Radio-Diagnosis at Sri Devaraj Urs Medical College. I will be conducting a study titled **"COMPARISION OF T1 FAT SUPPRESED (FS) POSTCONTRAST AND T2 FLUID ATTENUATED INVERSION RECOVERY (FLAIR) POSTCONTRAST SEQUENCES IN EVALUATION OF INTRACRANIAL LESIONS."** for my dissertation under the guidance of Dr. N. RACHEGOWDA, Professor & Head, Department of Radio-Diagnosis. In this study, we will assess the accuracy/ ability of T2 FLAIR postcontrast in evaluation of intracranial lesions. You will not be paid any financial compensation for participating in this research project.

All of your personal data will be kept confidential and will be used only for research purpose by this institution. You are free to participate in the study. You can also withdraw from the study at any point of time without giving any reasons whatsoever. Your refusal to participate will not prejudice you to any present or future care at this institution

Name and Signature of the Principal Investigator

Date:

ಸಮ್ಮತಿ ಪತ್ರ:

ಈ ಕೆಳಗೆ ಸಹಿ ಮಾಡಿರುವ ---------- ಆದ ನಾನು ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವ ಸಲುವಾಗಿ ವೈದ್ಯಕೀಯ ಪ್ರಯೋಗ ಪರೀಕ್ಷೆಗೆ ಒಳಪಡಲು ನನ್ನ ವೈಯ್ಯಕ್ತಿಕ ವಿವರಗಳನ್ನು ನೀಡಲು ಸಮ್ಮತಿಸಿರುತ್ತೇನೆ.

ಈ ಅಧ್ಯಯನದ ಉದ್ದೇಶ, ಅಧ್ಯಯನದ ಸಂದರ್ಭದಲ್ಲಿ ನೀಡುವ ಮತ್ತು ಸಂಗ್ರಹಿಸುವ ಮಾಹಿತಿಯ ಗೌಪ್ಯತೆಯ ಬಗ್ಗೆ ನನಗೆ ನನ್ನ ಸ್ಥಳೀಯ ಭಾಷೆಯಲ್ಲಿ ಓದಿ ಹೇಳಲಾಗಿದೆ/ವಿವರಿಸಲಾಗಿದೆ ಮತ್ತು ನಾನು ಇದನ್ನು ಅರ್ಥ ಮಾಡಿಕೊಂಡಿರುತೇನೆ. ಈ ಅಧ್ಯಯನದ ವಿವಿಧ ಅಂಶಗಳ ಬಗ್ಗೆ ಪ್ರಶ್ನೆಗಳನ್ನು ಕೇಳುವ ಅವಕಾಶವನ್ನು ನನಗೆ ನೀಡಲಾಗಿದೆ ಮತ್ತು ನನ್ನ ಪ್ರಶ್ನೆಗಳಿಗೆ ತೃಪ್ತಿಕರವಾದ ಉತ್ತರಗಳು ದೊರೆತಿರುತ್ತೆವೆ. ಈ ಅಧ್ಯಯನದ ಮೂಲಕ ಸಂಗ್ರಹಿಸಿರುವ ಮಾಹಿತಿಯನ್ನು ಸಂಶೋಧನೆಯ ಉದ್ದೇಶಕ್ಕೆ ಮಾತ್ರ ಬಳಸತಕ್ಕದ್ದು.

ಈ ಅಧ್ಯಯನದಿಂದ ಯಾವುದೇ ಸಂದರ್ಭದಲ್ಲಿ ಹಿಂದೆ ಸರಿಯುವ ಸ್ವಾತಂತ್ರ್ಯ ನನಗಿದೆ ಎಂಬುದನ್ನು, ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವುದರಿಂದ ನನಗೆ ಯಾವುದೇ ಹೆಚ್ಚುವರಿ ವೆಚ್ಚ ತಗಲುವುದಿಲ್ಲವೆಂಬುದನ್ನು ತಿಳಿಸಿರುತ್ತೇನೆ.

ಪರೀಕ್ಷಾರ್ಥಿಯ ಹೆಸರು ಮತ್ತು ಸಹಿ/ಹೆಬ್ಬೆಟ್ಟಿನ ಗುರುತು

ಸಾಕ್ಷಿಗಳ ಹೆಸರು ಮತ್ತು ಸಹಿ

1. ದಿನಾಂಕ:

2. ದಿನಾಂಕ:

ಸಂದರ್ಶಕರ ಹೆಸರು ಮತ್ತು ಸಹಿ

ದಿನಾಂಕ:

ಪ್ರಧಾನ ಪರೀಕ್ಷಕರ ಹೆಸರು ಮತ್ತು ಸಹಿ

ದಿನಾಂಕ:

ಸಂಪರ್ಕ ವಿವರಗಳು:

ಡಾ ಸಹನ ಎನ್. ಗೌಡ ದೂರವಾಣಿ – 8880746470

ಡಾ ರಾಚೇಗೌಡ ಎನ್. ದೂರವಾಣಿ - 9945327418

ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆ

ಮುಖ್ಯ ಸಂಶೋಧಕರು: ಡಾ. ಸಹನ ಎನ್. ಗೌಡ/ ಡಾ. ರಾಚೇಗೌಡ ಎನ್.

ನಾನು ಡಾ. ಸಹನ ಎನ್. ಗೌಡ, ಶ್ರೀ ದೇವರಾಜ್ ಅರಸ್ ಮೆಡಿಕಲ್ ಕಾಲೇಜಿನ ರೇಡಿಯೋ ರೋಗನಿರ್ಣಯ ಇಲಾಖೆಯಲ್ಲಿನ ಸ್ನಾತಕೋತ್ತರ ವಿಧ್ಯಾರ್ಥಿನಿ. " ಮೆದುಳಿನ ಗೆಡ್ಡೆಗಳಲ್ಲಿ ಟಿ೧ ಕೊಬ್ಬು ಸುಧಾರಿತ ಪೋಸ್ಟ್ ಕಾಂಟ್ರಾಸ್ಟ್ ಮತ್ತು ಟಿ೨ ದ್ರವ ಕ್ಷೀಣಿಸಿ ವಿಪರ್ಯಯ ಚೇತರಿಕೆ ಪೋಸ್ಟ್ ಕಾಂಟ್ರಾಸ್ಟ್ " ಎಂಬ ನನ್ನ ಮಹಾಪ್ರಬಂಧಕ್ಕಾಗಿ ಡಾ. ರಾಚೇಗೌಡ ಎನ್, ಪ್ರಾಧ್ಯಾಪಕರು ಮತ್ತು ಮುಖ್ಯಸ್ಥರು, ರೇಡಿಯಾಲಜಿ ವಿಭಾಗ ಮಾರ್ಗದರ್ಶನದಲ್ಲಿ ಮಾಡುತ್ತೇನೆ.

ಮೆದುಳಿನ ಗೆಡ್ಡೆಗಳ ಸಮಸ್ಯೆ ಅನೇಕ ರೋಗಗಳಿಂದ ಹುಟ್ಟಿಕೊಳ್ಳಬಹುದು. ಹೀಗಾಗಿ, ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಅಂತಹ ಸಂದರ್ಭಗಳಲ್ಲಿ ನಿರ್ವಹಣೆಯಲ್ಲಿ ಪರ್ಯಾಯ ರೋಗನಿರ್ಣಯದ ಸಾಧನವಾಗಿ ಮತ್ತು ಸಹಾಯ ವರ್ತಿಸುತ್ತೇವೆ.

ನೀವು ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಒಪ್ಪಿದಲ್ಲಿ ನಾವು ನಿಮ್ಮ ಆಸ್ಪತ್ರೆಯ ದಾಖಲೆಗಳಿಂದ ನಿಮ್ಮ ಬಗ್ಗೆ ಸಂಬಂಧಿಸಿದ ವಿವರಗಳನ್ನು ಸಂಗ್ರಹಿಸುತ್ತೇವೆ. ಸಂಗ್ರಹಿಸಿದ ಮಾಹಿತಿಯನ್ನು ಮಾತ್ರ ಸಂಶೋಧನೆ ಉದ್ದೇಶಕ್ಕಾಗಿ ಬಳಸಲಾಗುತ್ತದೆ. ಈ ಅಧ್ಯಯನವು ಸ್ಥಳೀಯ ನೈತಿಕ ಬೋರ್ಡ್ ವಿಮರ್ಶೆ ಮಾಡುತ್ತದೆ ಮತ್ತು ಕೇವಲ ಅವರ ಔಪಚಾರಿಕ ಅನುಮೋದನೆ ನಂತರ ಪ್ರಾರಂಭಿಸಲಾಗುವುದು. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನೀವು ಯಾವುದೇ ವೆಚ್ಚದಲ್ಲಿ ಒಳಗೊಳ್ಳುವುದಿಲ್ಲ. ನೀವು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಒಪ್ಪುತ್ತೀರಿ ಎಂದು ತಿಳಿಸಿ, ನಿಮ್ಮ ಹೆಬ್ಬೆಟ್ಟಿನ ಗುರುತು ಅಥವಾ ಸಹಿ ಅಗತ್ಯವಿದೆ.

ಸಂಪರ್ಕ ವಿವರಗಳು:

ಡಾ ಸಹನ ಎನ್. ಗೌಡ ದೂರವಾಣಿ – 8880746470

ಡಾ ರಾಚೇಗೌಡ ಎನ್. ದೂರವಾಣಿ - 9945327418

Comparison of T1 Fat suppressed (FS) postcontrast and fluid attenuated inversion recovery (FLAIR) postcontrast sequences in evaluation of intracranial lesions.

PROFORMA:

Patient ID -

MRI no -

Demographic details:

Name -

Age -

Sex - ☐ Male ☐ Female

History of allergy to contrast: Yes/ No

Study details:

Location of lesion		T1 FS Plain	T2 FLAIR Plain	T1 FS PC	T2 FLAIR PC
Meningeal enhancement	Present				
	Absent				
Intra axial lesion					
Extra axial lesion					
FS – Fat suppressed, PC – Post contrast					

MRI findings/ diagnosis:

Concomitant findings (if any):

Radiologist:

Sl.no	Name	Age	Sex	Meningeal echancement		Intracranial lesions				Diagnosis
						IA		EA		
				T1 FS PC	FLAIR PC	T1 FS PC	FLAIR PC	T1 FS PC	FLAIR PC	
1	Akkalamma	72	F			Moderate	Mild			Tuberculoma
2	ANASUYAMMA	65	F			Mild	Moderate			Parietal lobe glioma
3	Anitha	36	F	Mild	None	Avid	Mild			Meningioma
4	Anjanappa	55	M			Avid	Mild	Avid	Mild	Tuberculoma & Meningioma
5	Anjanappa	52	M			Mild	Moderate			Metastasis (Subcentimetric)
6	Anjanappa	52	M			Moderate	Mild			Necrotic metastasis
7	Anjanappa	52	M			Moderate	Mild			Metastasis with perilesional edema
8	ARCHANA	19	F			Mild	Moderate			Tubercular Abscess
9	ASHWATH GOWDA	45	M					Moderate	Avid	Trigeminal schwanomma
10	B/O RADHAMMA	1	M	Mild	Moderate					Leptomeningitis
11	Bhagamathi	21	F	None	Mild					Meningitis
12	Bhagavathi - 2	19	F	None	Mild	Mild	Moderate			Tuberculoma with meningitis
13	Bhagyalakshmi	48	F			Mild	mild			Neurocysticercosis
14	Bhavana shree	19	F	Mild	Mild					Meningitis
15	Chandra	40	F					Avid	Mild	Meningioma
16	Chandrashekar Bharathi	59	M			Mild	Moderate			Glioblastoma
17	CHANDRU SHEKAR BHARATHI	59	M			Mild	Avid			Glioblastoma multiforme
18	Changalarayappa	30	M			Moderate	Avid			Neurocysticercosis
19	CHOWDAPPA	39	M					Mild	Mild	Pitutary Macroadenoma
20	Deepak	25	M			Moderate	Mild			Neurocysticercosis
21	Dr. Aruna	49	F					Moderate	Avid	Recurrent chordoma
22	Dr. Aruna	49	F					Moderate	Avid	Recurrent chordoma
23	DR. RAMAIAH	90	M			Moderate	Avid			Multicentric Glioblastoma
24	Felix	67	M			Mild	Moderate	Mild	Moderate	Hemorrhagic metastasis
25	Hemavathi	25	F			Moderate	Mild			Neurocysticercosis
26	HIDAYATHULLA	43	M			Moderate	Avid			High grade glioma
27	John kennedy	58	M			Moderate	Avid			Dissseminated intracranial & spinal cord tuberculoma.
28	Jyothi	34	F	Mild	Avid	Moderate	Mild			Tuberculoma with meningitis
29	Lakshmidevamma	71	F	Mild	Avid					Meningitis

Sl.no	Name	Age	Sex	Meningeal echancement		Intracranial lesions				Diagnosis
						IA		EA		
				T1 FS PC	FLAIR PC	T1 FS PC	FLAIR PC	T1 FS PC	FLAIR PC	
30	Mahesh	44	M			Mild	Moderate			Low grade glioma
31	MANJUNATH G.	34	M			None	Moderate			Cerebral Toxoplasmosis
32	Mumtaz begum	54	F			Mild	Moderate			Neurocysticercosis
33	MUNAB SAHEB	58	M			Moderate	Mild			Tuberculoma
34	Muniyappa	60	M	Mild	Moderate	Mild	Moderate			Primary CNS lymphoma with meningeal metastasis
35	Nagarajappa	65	M			Moderate	Avid			Metastasis
36	NAGESH	27	M	Mild	Moderate					Meningitis
37	Nandini	23	F	None	Mild					Leptomeningitis
38	Naramma	70	F					Avid	Moderate	Meningioma
39	Narayana swamy	60	M	Mild	Moderate					Meningitis
40	NARAYANASWAMY	37	M	Mild	Moderate					Meningitis
41	Nithin	18	M	Moderate	Avid			Moderate	Avid	Epidural abscess with meningitis
42	PRUTHVI RAJ	20	M			Mild	Avid			Ependymoma
43	Ragava	18	M	Mild	Moderate					Leptomeningitis
44	Ramachandrappa(MRI NO: 537)	65	M			Moderate	Avid			Hemorrhagic metastasis
45	Ramesh	35	M	None	Mild					Leptomeningitis
46	Sakkamma	50	F					Avid	Moderate	Meningioma
47	Shamma Sulthana	22	F	Mild	Avid	Mild	None			Meningoencephalitis - Koch's
48	SHASHIKALA	54	F			Mild	Moderate			Low grade oligodendroglioma
49	Sheela	47	F	Moderate	Avid			Avid	Avid	Meningioma with meningitis
50	Shravani	11	F			Moderate	Avid			Neurocysticercosis
51	SONNEGOWDA	74	M					Moderate	Avid	Vestibular Schwannoma
52	Soundharya	20	F					Mild	Moderate	Choroid plexus xanthogranuloma
53	SREENATH	21	M			Mild	Avid			Neurocysticercosis
54	Srinivasulu	65	M	Mild	Moderate					Leptomeningitis
55	Sriram reddy	72	M	Moderate	Mild			Avid	Moderate	Atypical Meningioma with meningitis.
56	Suma	20	F			Mild	Moderate			Tuberculoma
57	Sunil kumar	20	M			Avid	Moderate			Neurocysticercosis
58	Sunitha	35	F	Mild	Moderate	Mild	Moderate			Tuberculoma with meningitis

Sl.no	Name	Age	Sex	Meningeal echancement		Intracranial lesions				Diagnosis
						IA		EA		
				T1 FS PC	FLAIR PC	T1 FS PC	FLAIR PC	T1 FS PC	FLAIR PC	
59	Swapna	29	F			Moderate	Avid			Meningioma
60	VANEJA	40	F			Mild	Moderate			Grade III Glioma
61	VENKATESHAPPA	42	M					Avid	Moderate	Meningioma
62	VENKATESHWARI	79	F			Moderate	Avid			Glioblastoma
63	Yashodamma	42	F	Mild	Avid	Mild	Moderate			Tuberculoma with meningitis
64	Sameer Khan	33	M			Moderate	Avid			Hemagioblastoma