"A STUDY OF COMPUTED TOMOGRAPHIC FINDINGS IN FOCAL SEIZURES"

By

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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
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Under the Guidance of

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

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation entitled **"A STUDY OF COMPUTED TOMOGRAPHIC FINDINGS IN FOCAL SEIZURES"** is a bonafide and genuine research work carried out by me under the guidance of **Dr. PURNIMA HEGDE**, Professor & Head, Department of Radiodiagnosis, Sri Devaraj Urs Medical College, Kolar, in partial fulfilment of University regulation for the award "**M.D. DEGREE IN RADIODIAGNOSIS**", the examination to be held in May 2017 by SDUAHER. This has not been submitted by me previously for the award of any degree or diploma from the university or any other university.

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"A STUDY OF COMPUTED TOMOGRAPHIC FINDINGS IN FOCAL SEIZURES"

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Dr. BHARATH REDDY M

LIST OF ABBREVIATIONS

CT	:	*Computed Tomography*
MRI	:	*Magnetic Resonance Imaging*
CSF	:	*Cerebrospinal Fluid*
CNS	:	*Central Nervous System*
CTA	:	*Computed Tomographic Angiography*
HU	:	*Hounsfield Unit*
EBCT	:	*Electron Beam Computed Tomography*
EEG	:	*Electroencephalography*
ILAE	:	*International League against Epilepsy.*
FMRI	:	*Functional MRI*
MRS	:	*Magnetic resonance spectroscopy*
NAA	:	*N-acetylaspartate*
SPECT	:	*Single photon emission computed tomography*
PET	:	*Positron emission tomography*
HMPAO	:	*Hexamethylpropyleneamine oxime*
18 FDG	:	*18 F-deoxyglucose*
NMDA	:	*N-Methyl-D-aspartate receptor*
NCC	:	*Neurocysticercosis*
T. solium	:	*Taenia solium*
ELISA	:	*Enzyme Linked Immuno Sorbent Assay*
IgG	:	*Immunoglobulin G*
CNST	:	*central nervous system tuberculoma*

TBM	:	*Tuberculous meningitis*
EPTB	:	*Extrapulmonary tuberculosis*
ICSOLs	:	*Intracranial space occupying lesions*
TORCH	**:**	*Toxoplasmosis, Other (syphilis, varicella-zoster, parvovirus B19), Rubella, Cytomegalovirus (CMV), and Herpes infections*
PNET	:	*Primitive neuroectodermal tumor*
DNET	:	*Dysembryonic neuroectodermal tumor*
SCTEL	:	*Single CT enhancing lesion.*
SSCCCTL	:	*Small single cerebral calcific CT lesion.*
FLAIR	:	*Fluid Attenuated Inversion Recovery*
DWI	:	*Diffusion weighted imaging*
MRA	:	*Magnetic resonance angiogram*
AVM	:	*Arteriovenous malformations*
DVA	:	*Developmental venous anomalies.*
IT	**:**	*Infra Tentorial*
kVp	**:**	*Kilovolts peak*
mAs	**:**	*Milliampere Second*
MDCT	**:**	*Multi Detector Computed Tomography*
MLS	:	*Midline Shift*
NCCT	**:**	*Non Contrast Computed Tomography*
ST	:	*Supra Tentorial*
SI	:	*Signal Intensity*
TB	:	*Tuberculosis*

ABSTRACT

Background: Seizures is one of the most common health care problems worldwide and there is evidence that it is increasing in prevalence. Computed tomography (CT) brain (plain ± contrast) study remains key investigation for accurate diagnosis and treatment.

Aims and Objectives: To evaluate morphological changes on CT brain study in patients who present with focal seizures and to study their etiological factors.

Methodology: The prospective study was conducted in 151 patients who were clinically diagnosed with focal seizures and referred for CT brain to department of Radiology. Of these, 86 patients had no abnormality on CT scan and they were given symptomatic treatment. 65 patients had abnormalities on the CT scan requiring further evaluation and they were included in our study.

Results: More than 45% of patients (n = 30; 46.1%) were in the age group of 0-20 years. There was slight male preponderance (n = 37; 56.9%). Simple seizures (n = 47; 72.3%) were more common compared to complex seizures (n = 12; 18.4%). Frontal lobe was the commonest site involved (n = 37; 43%) followed by parietal lobe (n = 32; 37.5%). 23 patients had lesions in more than one lobe.

Solitary lesions (n = 41; 63%) were more common than multiple lesions (n = 24; 37%). Supratentorial location (93.8%) of lesions was more common compared with infratentorial location.

Neuroinfections were the commonest cause for focal seizures (n = 36; 55.7%), which included NCC in 24 patients (36.9%) followed by tuberculoma (n = 11; 17%) and abscess (n = 1; 1.5%). Neuroinfections accounted for more than half of our cases. This is in agreement with a number of studies carried out in various parts of India. However, neuroinfections accounted for miniscule of cases in western studies.

Other etiologies included parenchymal calcifications and neoplasm (n = 9 each; 13.8%). Three patients had infarcts and cystic lesions (arachnoid epidermoid and colloid cyst). Post trauma complications (chronic SDH, gliosis) and vascular malformations were seen in two patients each. One patient had hydrocephalus.

Our study confirms the importance of CT in evaluation of focal seizures. CT is able to identify findings such as site of lesion, density of lesion, surrounding edema, hemorrhage, infarction, calcifications, mass effect and pattern of contrast enhancement of the lesion.

Among 151 patients with focal seizures who had CT scan, only 10 patients required further imaging with MRI. The remaining 141 patients were adequately diagnosed by CT.

CT brain (plain ± contrast) study remains the key investigation for accurate diagnosis in patients with focal seizures and helps in early management. We conclude that CT should be carried out in every patient with focal seizure to rule out or confirm any organic lesion.

Conclusion: NCC and tuberculoma were the commonest cause for focal seizures. CT helped in identifying the various stages of NCC. CT also helped in differentiating NCC from tuberculoma. Other etiological factors included parenchymal calcifications, metastasis, tumors, vascular malformations, abscess and infarcts.

CECT helps in screening patients with focal seizures in order to identify patients with structural abnormalities. Majority of patients have classical findings on CT and undergo prompt treatment. A minority of patients may require further evaluation such as MRI, and CSF analysis.

Given its easy availability and affordability, CT is the primary investigation in evaluation of focal seizures.

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INTRODUCTION

Seizure disorder is the commonest neurological disorder in India[1]. Epilepsy is characterized by recurrent unprovoked seizures. The incidence of epilepsy is 24–53 per 1, 00,000 people in developed countries. There are few studies in developing countries, none of which are prospective: they show rates from 49.3 to 190 per 1,00,000 populations. The etiology of seizures is different in India and other developing countries as compared to the developed world[2]. Although, generalized tonic–clonic seizures occur uniformly throughout the life, partial seizures are common in very young and elderly people[1].

Computed tomography (CT) scan plays a very important role in radiological assessment of patients presenting with seizures[3]. CT helps in detecting structural abnormalities of the brain (calcifications, hemorrhage, cyst, and tumors), ventricular pathologies, infections, infarctions and lesions with underlying calcification[4]. The sensitivity of CT scan for detecting intracranial structural disease in epileptics is approximately 30-40%. There is probably an increased sensitivity in focal seizures[5]. CT of brain in children shows abnormal findings in up to 58%, which include infective lesions, focal and vascular lesions[6]. Inspite of availability of Magnetic Resonance Imaging (MRI), CT scan is still crucial in emergency situations because of its relative easy availability[7].

AIMS AND OBJECTIVES

The objectives of this study are:

1. To describe the CT scan findings in patients with focal seizures.
2. To correlate the CT scan findings with probable etiology in these patients.

REVIEW OF LITERATURE

Anatomy

The brain is supported by the skull base and enclosed within the skull vault. The cranial cavity is divided into the anterior, middle and posterior fossae (Figure 1). The anterior and middle cranial fossae contain the two cerebral hemispheres. The posterior fossa contains the brainstem, consisting of the midbrain, pons and, most inferiorly, the medulla, and the cerebellum[8].

Twelve paired cranial nerves arise from the brainstem, exit the skull base through a number of foramina, and innervate a variety of structures in the head proper. The largest of these foramina is the foramen magnum, through which the brainstem and spinal cord are in continuity[8].

The brain is invested by the meninges and bathed in cerebrospinal fluid (CSF), circulating in the subarachnoid space. Part of the meninges, the dura, forms an incomplete partition between the cerebral hemispheres, known as the falx and roofs the posterior fossa as the tentorium cerebelli. There is a gap in the tentorium, called the hiatus, through which the midbrain joins the hemispheres. Within the brain are a number of cavities, the lateral, third and fourth cerebral ventricles, which contain CSF produced by the choroid plexuses within the ventricles. CSF flows from the ventricles into the subarachnoid spaces over the cerebral surface and around the spinal cord [8].

Blood reaches the brain by the carotid and vertebral arteries and is drained by cerebral veins into a series of sinuses within the dura into the internal jugular veins[8].

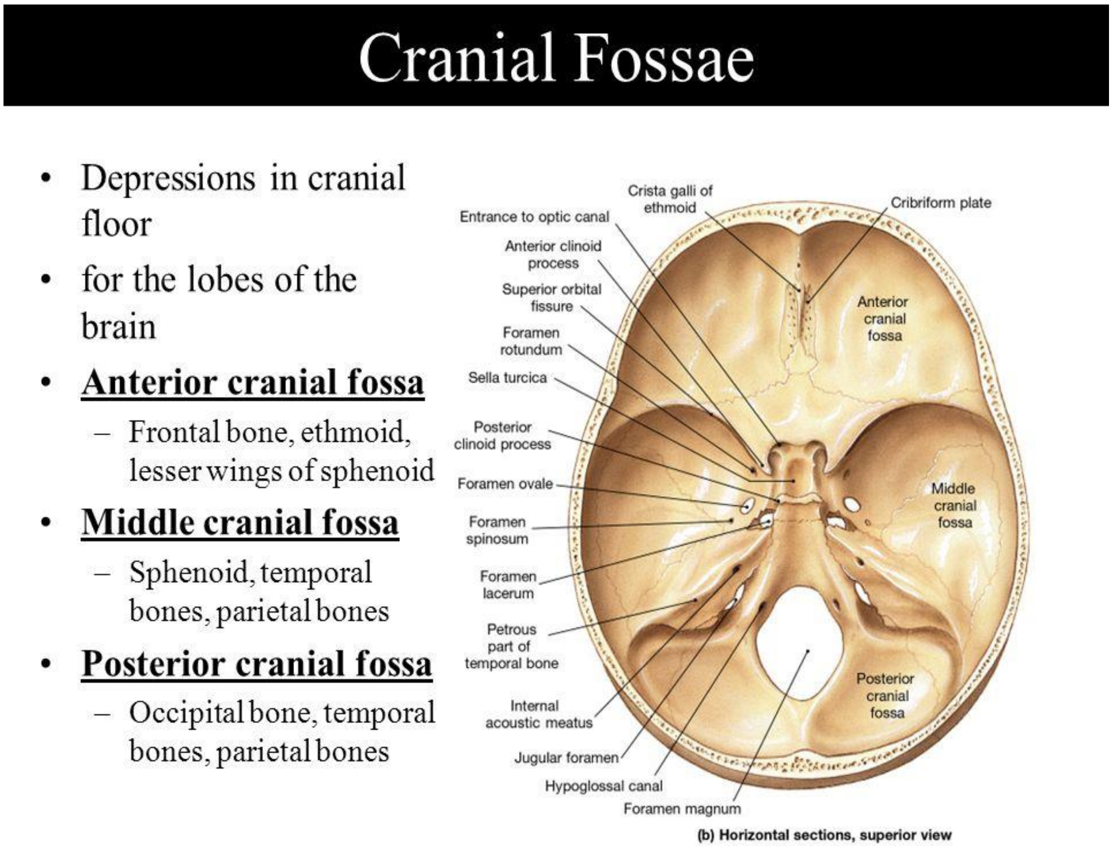


Figure1. Structures of cranial fossa

The cerebral hemispheres

The cerebral hemispheres lie above the tentorium and are divided by fissures and sulci into frontal, parietal, temporal, and occipital lobes (Figure2). The limbic system is also considered to be a lobe. The hemispheres are linked by the corpus callosum, the largest of the commissural tracts, which interconnect paired structures across the midline. Other examples of commissural tracts are the anterior, posterior, and habenular commissures. The anterior and posterior commissures are landmarks used in image-guided neurosurgical procedures.

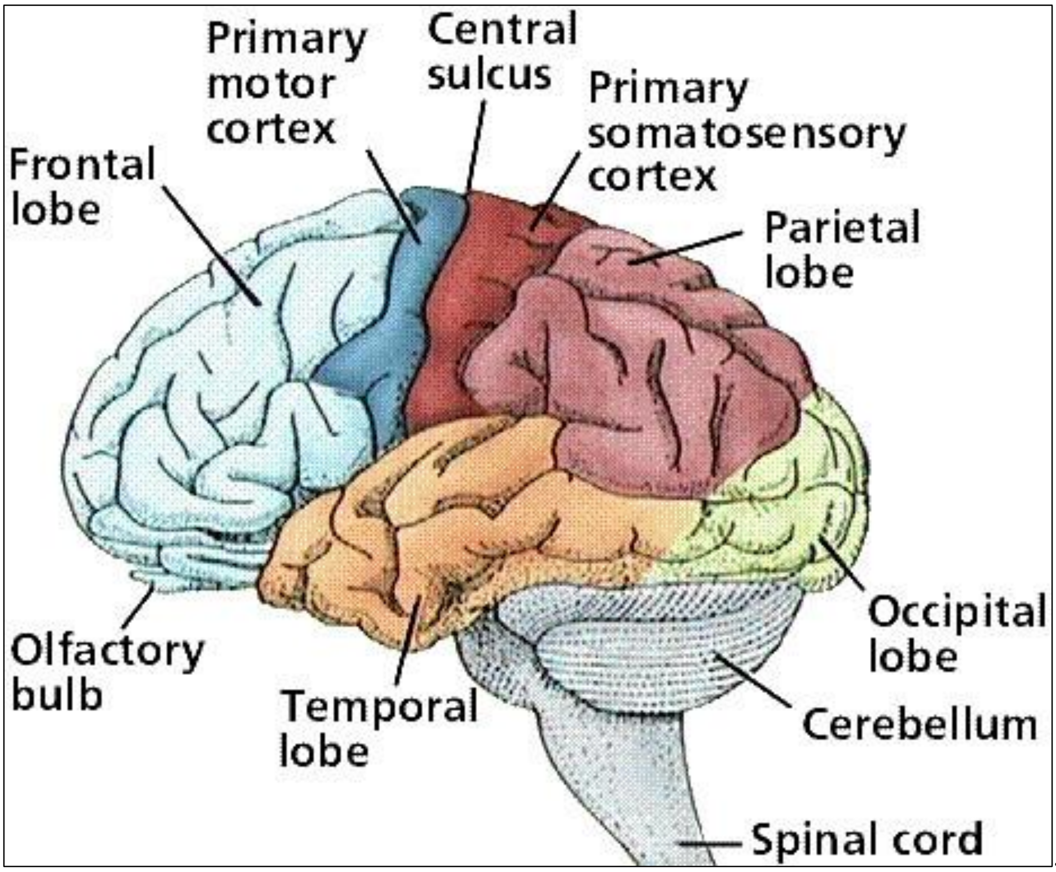


Figure 2. Image illustrating lobes of brain.

The corpus callosum is a myelinated tract and appears curved in sagittal images. The anterior rostrum blends with the anterior commissure inferiorly. The curved genu (knee) leads posteriorly to the body, hence the largest and most posterior part is the splenium. Fibers from the corpus callosum sweep anteriorly into the frontal white matter as the forceps minor and posteriorly into the occipital white matter as the forceps major[8].

There is considerable individual variation in gyral anatomy. It is also important to appreciate that the relationship of function to structure may be variable and that of speech, for example, it may be represented over a number of gyri with intervening white matter. Equally, it may be difficult to identify the central sulcus and adjacent motor strip accurately[8].

The anatomical boundaries of the individual lobes may be indistinct, depending on the aspect. The frontal lobe is the largest of the anatomical lobes occupying the anterior cranial fossa and extending posteriorly to the central sulcus. In common with the temporal lobe, the frontal lobe has three major gyri, superior, middle, and inferior, which are oriented horizontally. The temporal lobe occupies the middle cranial fossa The anterior limit of the parietal lobe is the central sulcus, which, running in the coronal plane, separates the precentral (motor) gyrus of the frontal lobe from the postcentral (sensory) gyrus. The boundary between the parietal and temporal lobes laterally is indistinct but the parieto-occipital incisure medially defines the two lobes. The main cortical supply of the occipital lobe relates to vision[8].

The calcarine (visual) cortex can be seen to indent the posterior (occipital) horns of the lateral ventricles. The cortex here is deeply in folded with little intervening white matter. Inferiorly and laterally the temporo-occipital fissure marks the division between the two lobes [8].

The Sylvian or lateral fissure separates the superior surface of the temporal lobe from the inferior frontal lobe and the anterior parietal lobe. During development, the cortex overlying the basal ganglia is invaginated to form the insula. The cortex in front of, above, and below this depression expands to form covering folds termed the operculum. The Sylvian fissure is formed between these folds. On axial imaging it runs in the coronal plane on the lower cuts and in the sagittal plane on the higher slices. On coronal MRI, it resembles the shape of a T lying on its side[8].

The cerebellum

The cerebellum consists of two hemispheres joined by a central vermis. The cortical mantle of the cerebellum overlies the white matter core as in the cerebral hemispheres but the cerebellar cortical ridges, known as the folia, and the intervening sulci are approximately parallel to one another and are linked to the brainstem by the paired cerebellar peduncles. They are named logically. The inferior cerebellar peduncles join the medulla to cerebellum; the middle cerebellar peduncles (the largest), pons to cerebellum; the superior cerebellar peduncles, midbrain to cerebellum[8].

The brainstem

The brainstem consists of medulla, pons, and midbrain. The medulla, pons, and cerebellum together constitute the hindbrain. The medulla commences at the foramen magnum as a continuation of the spinal cord. Initially it is “closed,” possessing a central canal like the spinal cord. More superiorly, it becomes “open” as the central canal leads into the fourth ventricle[8].

In the brainstem, the motor tracts are generally anterior to the sensory, hence the clinical usage of “anterior” columns meaning motor and “posterior” column, sensory. A number of decussations occur within the brainstem where both motor and sensory fibers cross the midline in accordance with the general principle that functional control of one-half of the body is largely exercised by the contralateral cerebral hemisphere. The sensory decussation is craniad to the motor, but both occur in the closed portion of the medulla. The medulla leads superiorly into the pons, which has an anterior “belly” and a posterior tegmentum[8].

The midbrain has two cerebral peduncles transmitting the motor tracts. Its posterior portion is pierced by the cerebral aqueduct (of Sylvius), to connect the third and fourth cerebral ventricles. The posterior portion is known as the tectum or tectal plate. It consists of four colliculi or quadrigeminal bodies concerned with auditory and visual reflexes[8].

The cerebral ventricular system cerebrospinal fluid spaces

The cerebral ventricular system consists of the paired lateral and single third and fourth ventricles. Cerebrospinal fluid (CSF) is produced in the choroid plexuses, and most of it is in the lateral ventricles, entering medially through the choroidal fissures. It flows from the lateral ventricles to the third ventricle through the foramen of Monro, in the anterior portion of the roof of the third and from the third to fourth via the cerebral aqueduct of the midbrain. From the fourth ventricle, the CSF enters the subarachnoid spaces, leaving through the paired foramina of Luschka, laterally and the midline, single foramen of Magendie. (Figure-3) These foramina provide a potential route of spread for intraventricular tumors[8].

At the base of the brain, there are relatively large CSF spaces, the basal CSF cisterns, which are important both anatomically and in CT or MRI diagnosis. Although named individually, according to adjacent structures, they interconnect freely with each other and with the CSF spaces generally[8].

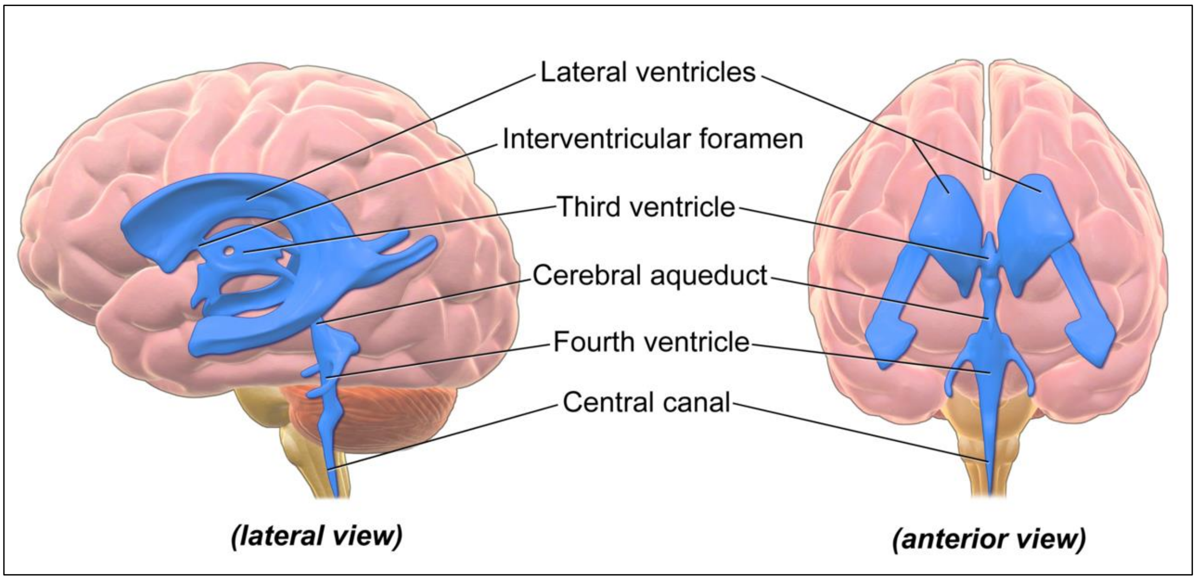


Figure 3. Cerebral ventricular system.

The cerebral blood circulation

Cerebral arteries

The brain is supplied with oxygenated blood by the paired internal carotid and vertebral arteries. The common carotid artery in the neck divides at the approximate level of the upper border of the thyroid cartilage (C4) into its internal and external branches, the latter supplying the various craniofacial structures[8]. The internal carotid artery is the larger of the two branches, receiving 70% of the common carotid blood flow. It lies posterolateral to the external carotid near to the bifurcation and, neither common nor internal carotid arteries have cervical branches. The internal carotid artery enters the skull through the carotid canal and courses (Figure-4, 5, 6) anteromedially and horizontally (the petrous segment) before turning superiorly into the cavernous sinus. In this position, the artery forms the shape of a siphon. Emerging from the cavernous sinus, the artery enters the subarachnoid space and divides into its terminal branches, the anterior and middle cerebral arteries[8].

The circle of Willis is situated in the suprasellar cistern and links the internal carotid arteries with each other and with the vertebrobasilar system, via the single anterior and paired posterior communicating arteries. It affords some protection in the event of occlusion of major arteries by facilitating "cross-flow." Arising from the proximal anterior and middle cerebral arteries, a leash of small, perforating arteries (the lenticulostriates) supplies a variety of structures including the basal ganglia and internal capsule[8].

The vertebral arteries are the first branches of the subclavian arteries. They ascend vertically within the foramina transversaria of the 6th to the 2nd cervical vertebrae and posterolaterally through the foramen transversarium of the atlas, (first cervical vertebra). The arteries then travel superomedially to pass into the skull through the foramen magnum, piercing the dura and entering the subarachnoid space. At the level of the pontomedullary junction, the two arteries join to form the midline basilar artery, which runs anterior to the brainstem. The cerebellum is supplied by the posterior inferior cerebellar arteries, arising from the vertebral arteries just before the confluence, and the anterior inferior- and superior cerebellar arteries, arising from the basilar artery[8].

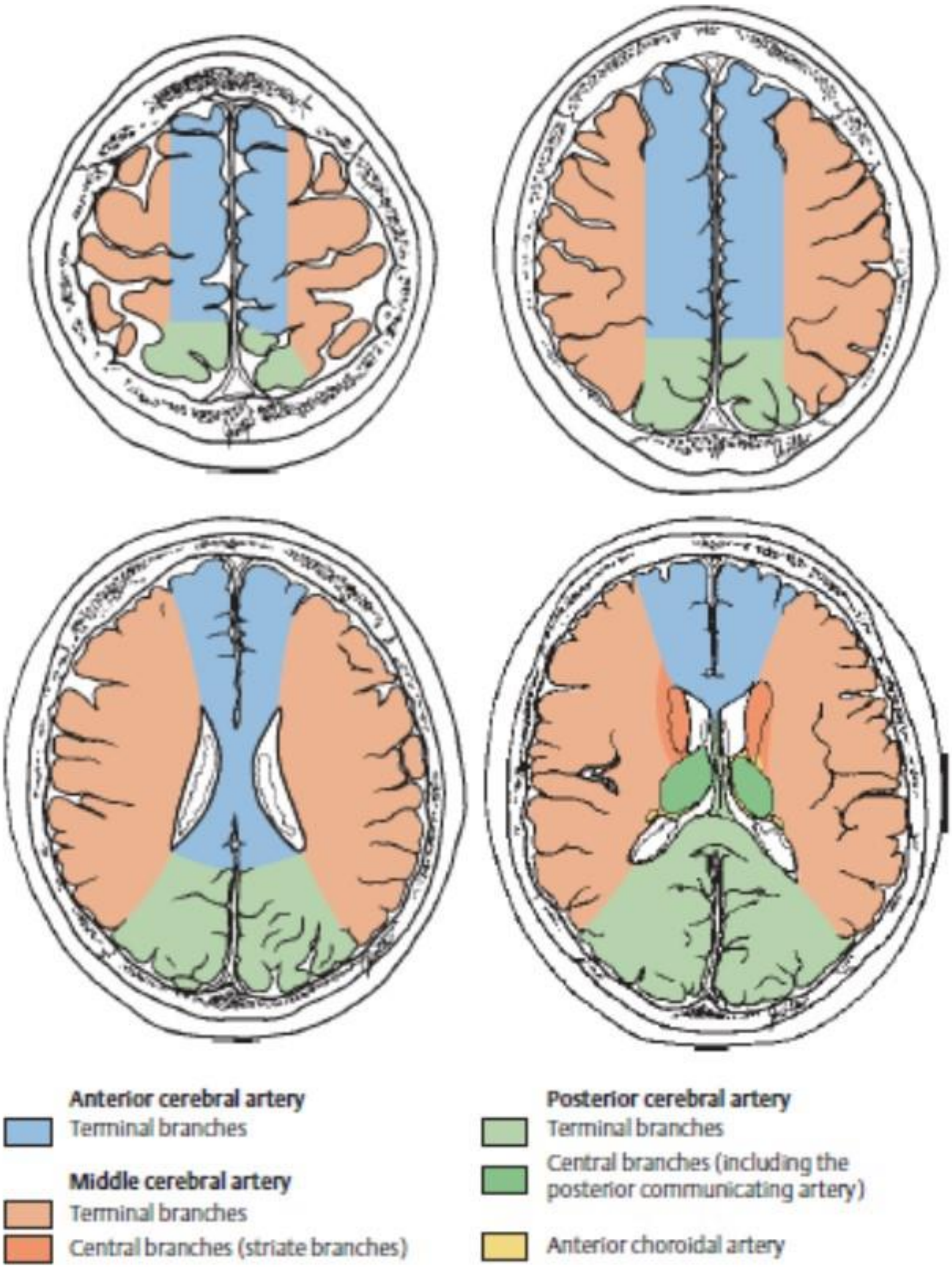


Figure 4. Vascular supply of brain at the level of basal ganglia and above[9].

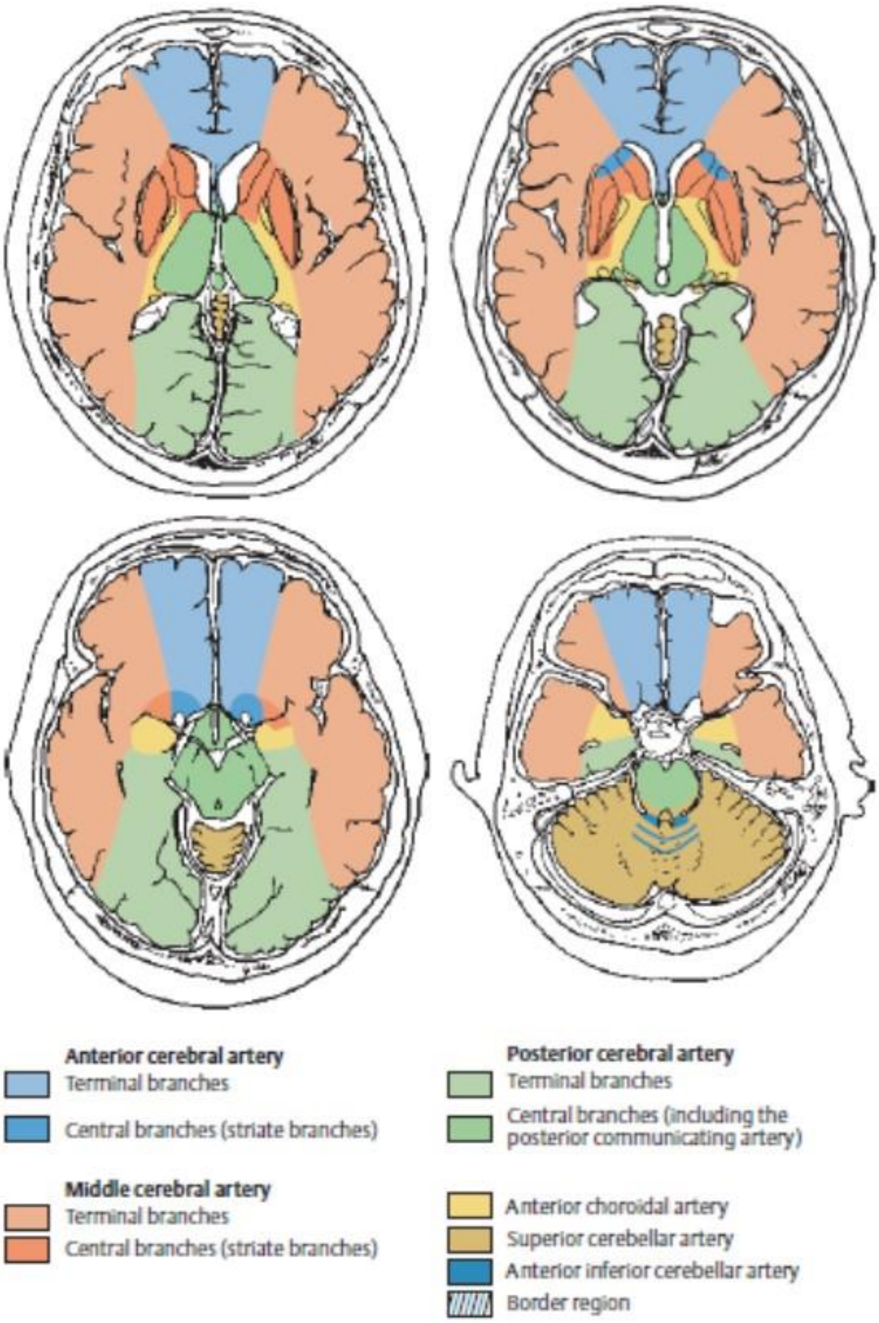


Figure 5. Vascular supply of brain at the level of basal ganglia and below[9].

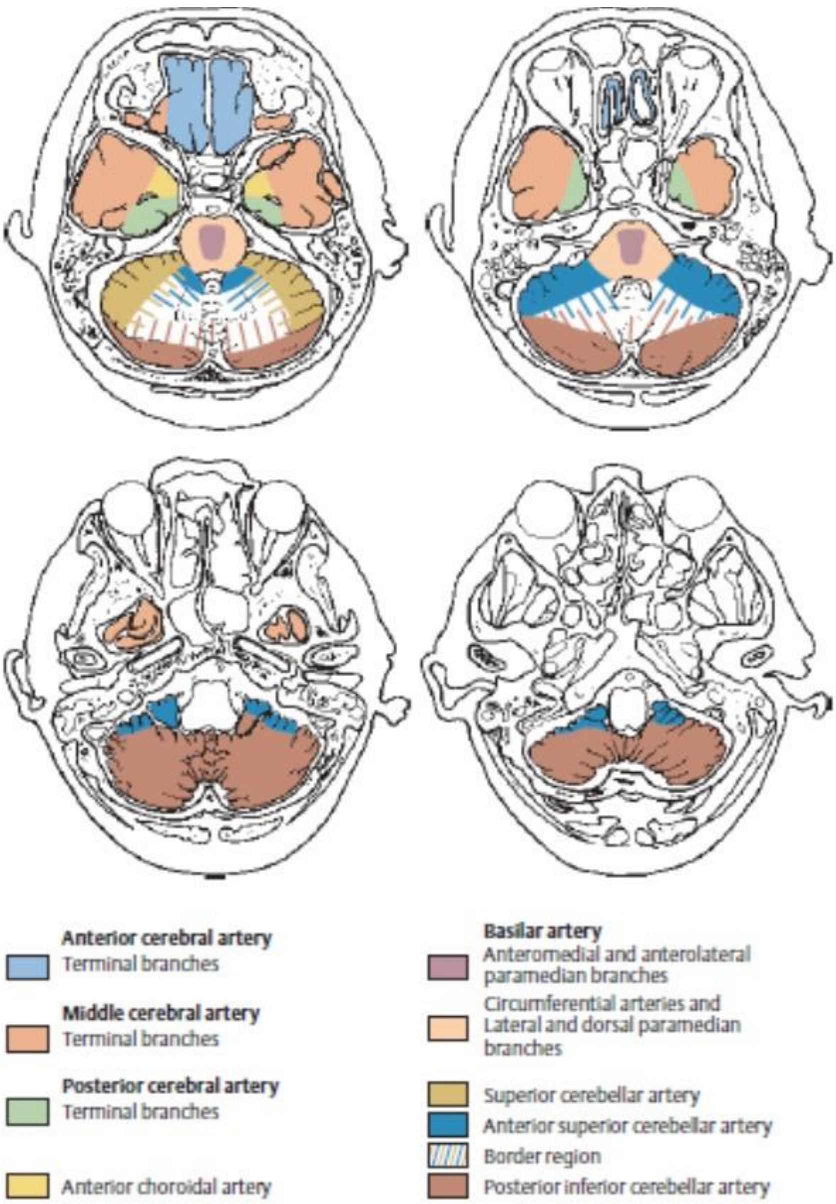
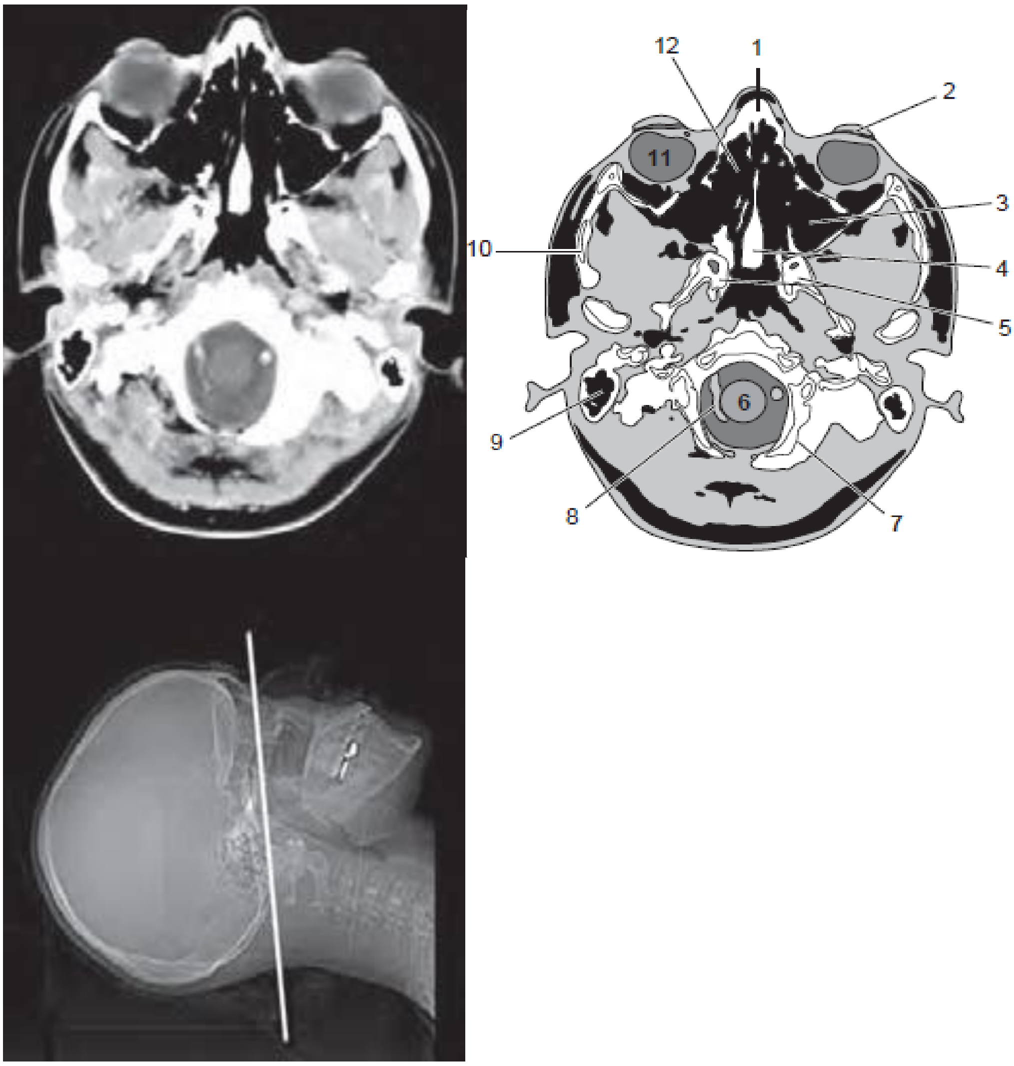
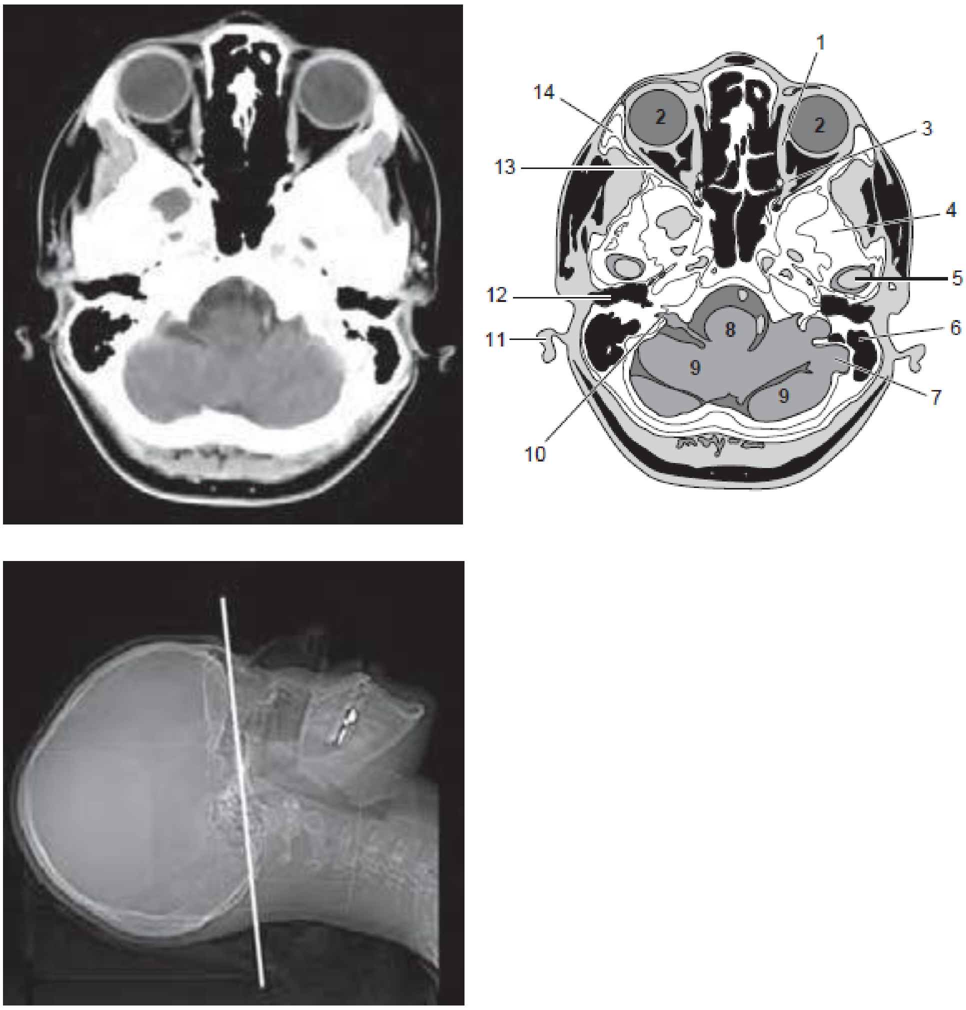


Figure 6. Vascular supply of brain at the level of temporal lobes, mid and hindbrain[9].



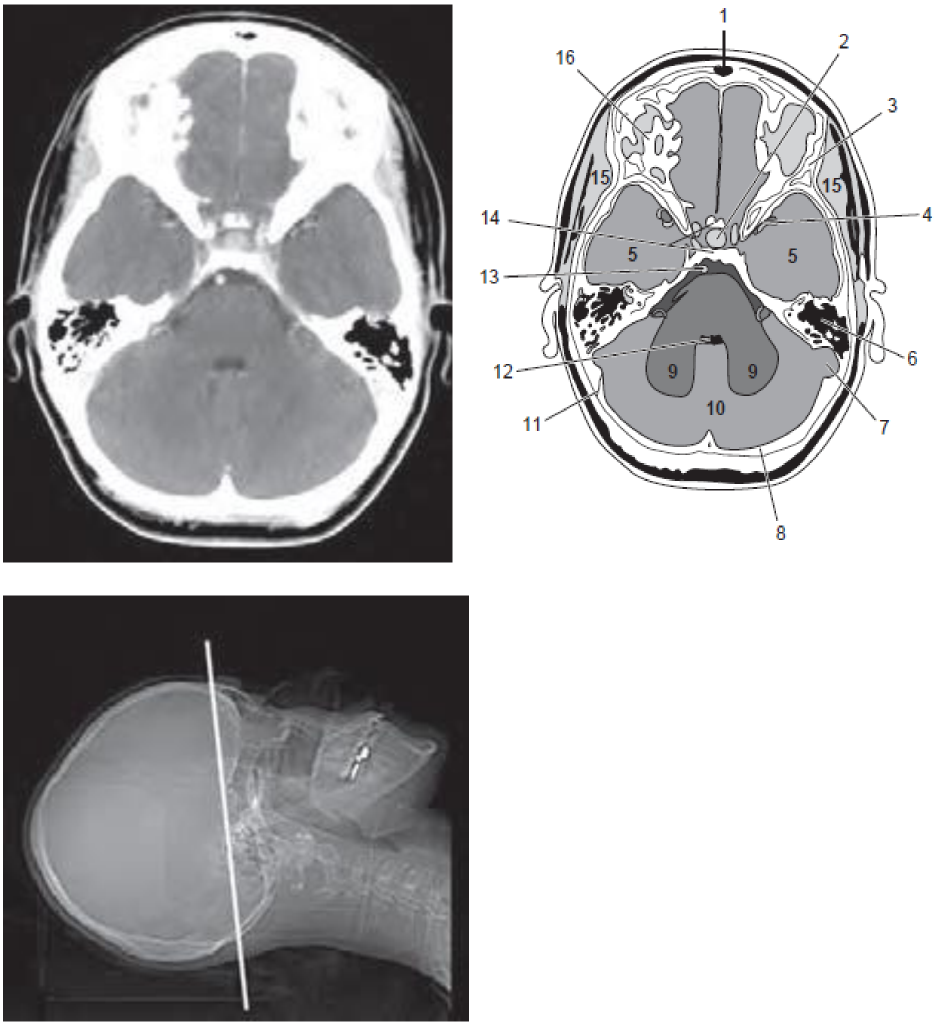
1. Nasal bones 7. Occipital bone
2. Eye, lens 8. Vertebral artery
3. Maxillary sinus 9. Mastoid air cells
4. Vomer 10. Zygoma
5. Sphenoid bone 11. Eye, globe.
6. Medulla oblongata 12. Ethmoid sinus

Figure 7. Cross Sectional Anatomy of CT Brain at the level of medulla[10].



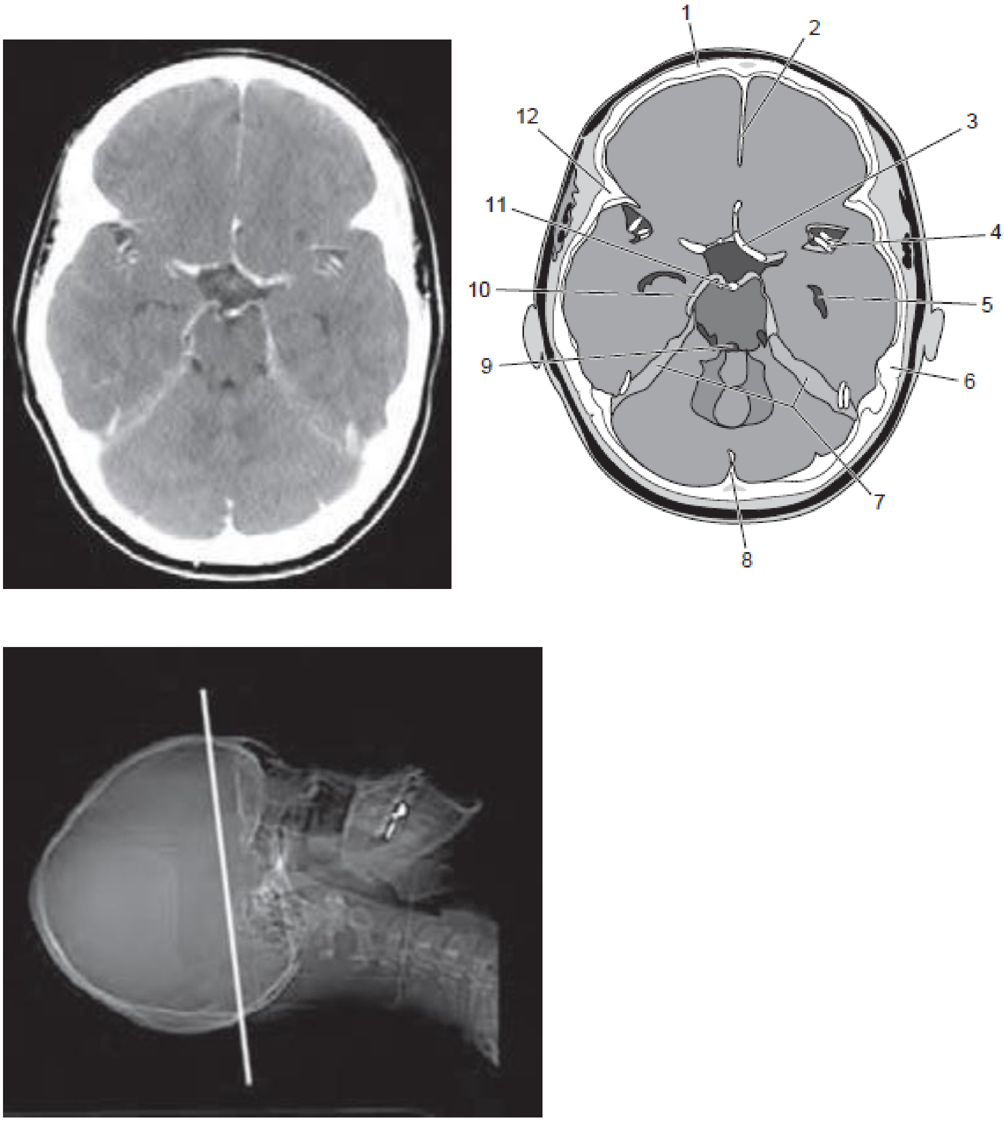
1. Medial rectus m.
2. Globe of eye
3. Optic n.
4. Greater Wing of Sphenoid bone
5. Mandibular condyle
6. Mastoid air cells in left temporal bone
7. Sigmoid sinus
8. Pons
9. Cerebellum
10. Internal auditory canal
11. Auricle
12. External auditory meatus
13. Lateral rectus m.
14. Zygoma

Figure 8. Cross Sectional Anatomy of CT Brain at the level of lower pons and cerebellum[10].



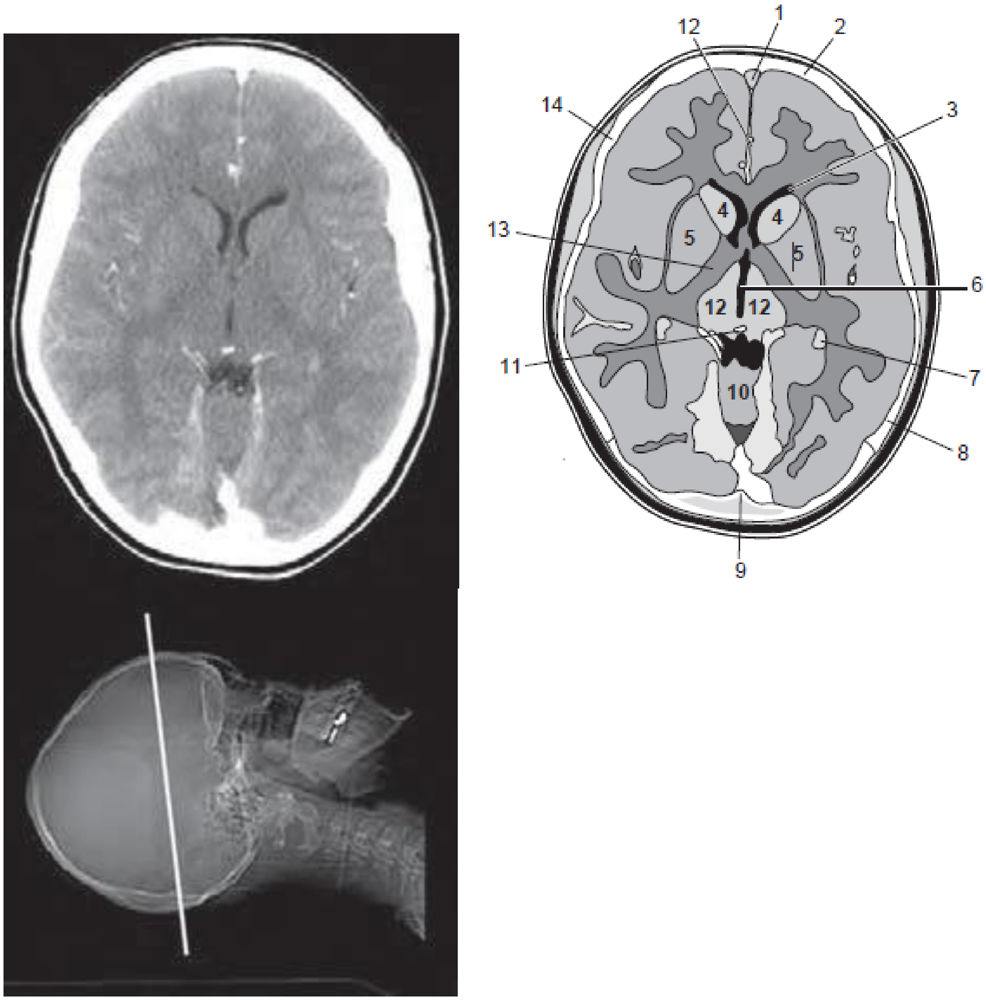
1. Frontal sinus
2. Pituitary
3. Sphenoid bone
4. Middle cerebral a.
5. Temporal lobe
6. Mastoid air cells in left temporal bone
7. Sigmoid sinus
8. Occipital bone.
9. Cerebellar peduncles
10. Cerebellum
11. Right lambdoid suture
12. Fourth ventricle
13. Basilar a.
14. Sella tursica
15. Temporalis m.
16. Frontal bone, orbital roof

Figure 9. Cross Sectional Anatomy of CT Brain at the level of Cerebellum [10].



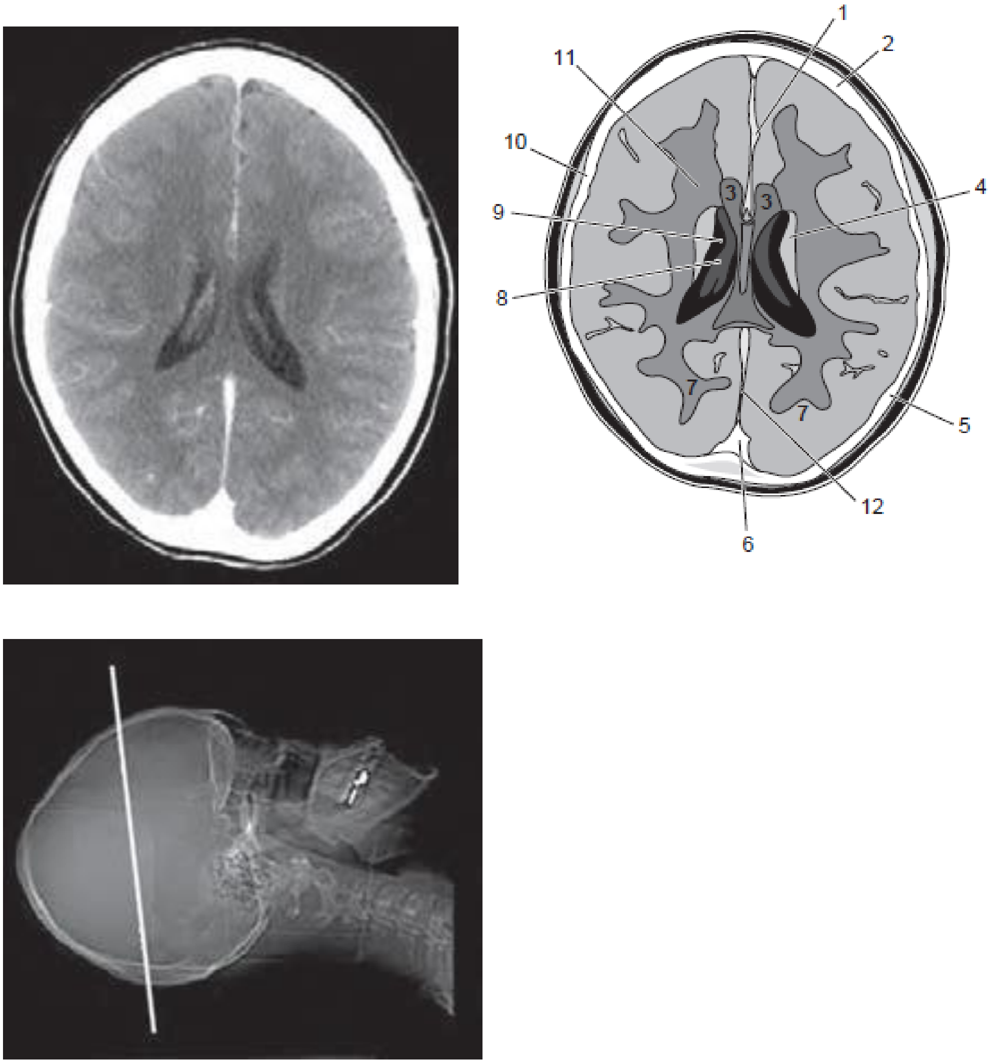
1. Frontal bone
2. Falx cerebri
3. Anterior cerebral a.
4. Middle cerebral a.
5. Lateral ventricle, temporal horn
6. Parietal bone
7. Cerebellum, tentorium
8. Internal occipital protuberance
9. Fourth ventricle
10. Posterior cerebral a.
11. Basilar a.
12. Temporal bone.

Figure 10. Cross Sectional Anatomy of CT Brain at the level of fourth ventricle[10].



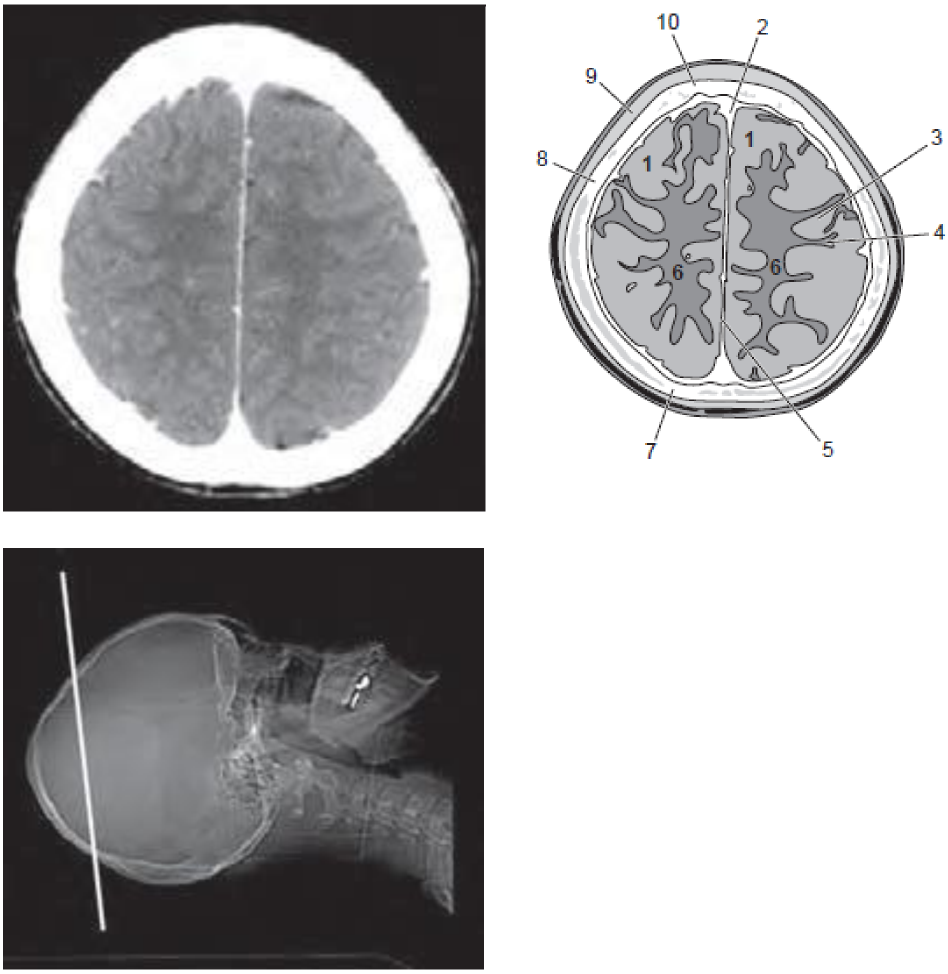
1. Superior sagittal sinus
2. Frontal bone
3. Lateral ventricle,
anterior horn
4. Caudate nucleus, head
5. Putamen/Globus pallidus
6. Third ventricle
7. Choroid plexus
8. Parietal bone
9. Internal occipital
protuberence
10. Cerebellar vermis
11. Pineal body
12. Thalamus
13. Internal capsule
14. Temporal bone.

Figure 11. Cross Sectional Anatomy of CT Brain at the level of third ventricle[10].



1. Falx cerebri	7. Occipital lobe
2. Frontal bone	8. Choroid plexus
3. Corpous callosum	9. Lateral ventricle, body
4. Caudate nucleus, body	10. Temporal bone
5. Parietal bone	11. Corona radiate
6. Confluence of sinuses (torcula)	12. Straight sinus

Figure 12. Cross Sectional Anatomy of CT Brain at the level of corona radiate[10].



1. Frontal lobe
2. Superior sagittal sinus
3. Precentral gyrus
4. Central sulcus
5. Falx cerebri
6. Parietal lobe
7. Parietal bone
8. Temporal bone
9. Scalp
10. Frontal bone.

Figure 13. Cross Sectional Anatomy of CT Brain at the level of centrum semiovale[10].

HISTORICAL BACKGROUND

CT IMAGING:

Medical imaging has experienced significant changes in both the technologic and clinical areas since the discovery of X-ray in 1895 by Wilhelm Conrad Roentgen, a German Physicist. Innovations have become common in the Radiology Department, and today the introduction of new ideas and methods and refinements in existing techniques are apparent. One such evolution is the invention of computed tomography (CT). The first clinical results were presented in 1967 and it became available as practical tool in 1971[11].

Sir Godfrey Hounsfield, an electronic engineer working at the Central Research Laboratories of EMI in England commenced work on image reconstruction in 1968. His original apparatus consisted of a collimated isotope source mounted on a lathe bed. The objects examined were phantoms contained within a ten-inch water. The scan took 9 days to complete because of the low intensity of the X-ray radiation source, and a further 2 1/2 hours to process the reading through a computer. The resulting image though of poor quality proved that the system worked. To provide sufficient intensity the equipment was modified by replacing the isotope with an industrial X-ray tube[11].

A prototype scanner was then developed and installed in Atkinson Morley Hospital in Wimbledon, England on 1st October 1971. The first patient scanned is 41 year old female with suspected frontal lobe tumor. The tumor was clearly demonstrated on the scan [11].

Hounsfield and Ambrose presented their paper on CT to the annual congress of the British Institute of Radiology on 20th April 1972 to great acclaim. The first CT papers, by these authors appeared in BJR in 1973. The invention of this technique resulted in the award of 1979 Nobel Prize in physiology and medicine to Sir G. N. Hounsfield, Central Research Lab., England (EMI), A. N. Cormack of Physics Department, Tufts University, Massachusetts, U.S.A. Advanced Technological Developments. Over the last ten years four different generations of CT scan equipment were produced. The most important improvements have been in the reduction in the single image generation time from five minutes to 2.5 seconds in the third and fourth generations scanners and an increase in spatial resolution and contrast. The introduction of second generation CT scanners further reduced the scan time from about six minutes to about two minutes. Late second generation CT scanners with ≥ 20 detectors further reduced scanning time to about ≤ 20 seconds. This dramatically improved quality of body scans, which could not be performed previously within a breath hold. The third generation scanners further reduced the scan time to 5 seconds or less, which has now further improved to about 0.33 seconds[11].

In the mid-1980s, another high speed CT scanner was introduced, which is referred as the Electron Beam CT (EBCT) scanner used for imaging cardiovascular system. In 1989, Dr. Willi Kalender introduced volume scanning by using Spiral /

Helical CT scanners. In Spiral/Helical CT Scanners, a thin X-ray beam traces a path around the patient and scans a volume of the tissue. Recently, dual slice spiral /helical CT scanner and multislice CT scanners were introduced which mainly increased the speed and volume of scan. Volume CT scanning has resulted in a wide range of applications such as CT Fluoroscopy, CT Angiography (CTA), Three Dimensional Imaging and Virtual Reality Imaging[11].

Slip Ring Scanners

There was no significant improvement in CT technology following 4th generation CT scanners in late 1980's. The only limitation at that time was interscan delays. Following one 360^0 rotation, the cables connecting rotating components (x-ray tube and detectors) to the rest of the gantry required rotation to be stopped and reversed for next slice, all of which added time of scan. All this changed with application of low-voltage slip rings. Slip rings provide electricity to the rotating components without fixed connections. Slip rings made it possible for continuous rotation, thereby reducing scan time. This technology also paved the way for introduction of spiral/helical CT scans[11].

Other imaging modalities in evaluation of seizures

Ultrasound examination of the cranium would help in detecting hydrocephalus (up to the age of 18 months only). Radiological investigations should begin with a chest radiograph to look for a pulmonary focus and skull roentgenograph including views of the sinuses and mastoids should be taken.

MRI may identify an epileptogenic lesion in 12–14%, but up to 80% of the patients with recurrent seizures have structural abnormalities evident on MRI. Functional MRI (fMRI) offers a tool for visualizing regional brain activity. It helps in localization and lateralization of cognitive functions of epilepsy patients who are evaluated for surgery in order to minimize the risk of causing a fixed deficit. Magnetic resonance spectroscopy (MRS) provides measurements of specific brain metabolites include N-acetyl aspartate (NAA), choline, creatine, lactate, c - aminobutyric acid (GABA), and glutamate. There is evidence that NAA is located primarily within neurons and precursor cells. Creatine and choline are found in both neurons and glia[11].

Single photon emission computed tomography (SPECT) is a nuclear medicine imaging method that allows measurements of regional cerebral blood flow changes in the areas affected by epileptic activity. Seventy five per cent of 99m Tc-HMPAO is extracted across the blood–brain barrier, reaching peak concentrations within one minute after injection. The images can then be acquired up to six hours after tracer injection. Both ictal and postictal SPECT studies should be performed during simultaneous video-EEG monitoring to determine the relation between seizure onset and tracer injection[11].

Positron emission tomography (PET) maps cerebral glucose metabolism using 18 F-deoxyglucose (18 FDG) and cerebral blood flow using15 O-labelled water. Interictally PET shows areas of reduced glucose metabolism and blood flow that usually include the seizure focus but are more extensive[11].

SEIZURES

CLINICAL ASPECTS

A seizure is a result of excessive nerve-cell discharges in the brain. It is seen as a sudden abnormal function of the body, often with loss of consciousness, an excess of muscular activity, or sometimes a loss of it, or an abnormal sensation.

The excessive nerve-cell discharges or excitation may remain in a small area of the brain (a localized lesion or focus) giving rise to partial (focal) seizures, or start immediately in the whole brain or spread from the small area (focus) to the whole brain and spinal cord giving rise to generalized seizures[12].

International classification of epileptic seizures

The classification of epileptic seizures divides into three categories

1) Partial focal seizures beginning locally

- Simple (consciousness not impaired)
- Complex (with impairment of consciousness)
- Partial seizures becoming secondarily generalized.

2) Generalized seizures (bilaterally symmetrical and without local onset)

A. 1. Absence seizures

2. Atypical absence seizures

B. Myoclonic seizures

C. Clonic seizures

D. Tonic seizures

F. Atonic seizures

3) Unclassified epileptic seizures[13].

Focal (partial) seizures:-

In focal seizures, epileptic activity starts in just part of the person's brain. Different areas of the brain (lobes) are responsible for controlling all of our movements, body functions, feelings or reactions. So, focal seizures can cause many different symptoms.

Seizures can start in any of these lobes. Each person will have their own experiences and symptoms during a focal seizure[14].

Focal seizures that act as a warning of a generalised seizure

The epileptic activity that causes a focal seizure can sometimes spread through the brain and develop into a generalized seizure. Once the epileptic activity spreads to both halves of your brain, you quickly have a generalized seizure, usually a tonic-clonic, tonic or atonic seizures[14].

Pathophysiology

Seizures are caused by paroxysmal discharges from groups of neurons, which arise as a result of excessive excitation or loss of inhibition. About one third of seizures are caused by genetic abnormalities, mostly involving ion channels. A quarter or so are caused by structural lesions. Structural lesions are frequently detected in focal seizures. The most common such lesions are cerebral changes resulting from perinatal brain damage, malformations, cerebral infarcts, trauma, brain tumours, and infections. These lesions involve the cerebral cortex and are characterized by neuronal loss and gliosis. Others, including brain malformations, genetic tumor syndromes, and metabolic disorders are genetic or have a strong genetic component. In about half of seizure disorders, no genetic or structural abnormality is evident[15].

Basic Mechanisms of Focal Seizure initiation and Propagation

The hypersynchronous discharges that occur during a seizure may begin in a very discrete region of cortex and then spread to neighboring regions.

Seizure initiation is characterized by two concurrent events:

High-frequency bursts of action potentials, and

Hypersynchronization of a neuronal population.

The synchronized bursts from a sufficient number of neurons result in a so-called spike discharge on the EEG. At the level of single neurons, epileptiform activity consists of sustained neuronal depolarization resulting in a burst of action potentials, a plateau-like depolarization associated with completion of the action potential burst, and then a rapid repolarization followed by hyperpolarization. This sequence is called the paroxysmal depolarizing shift. The bursting activity resulting from the relatively prolonged depolarization of the neuronal membrane is due to influx of extracellular Ca^{++}, which leads to the opening of voltage-dependent Na^{+} channels, influx of Na^{+}, and generation of repetitive action potentials. The subsequent hyperpolarizing after potential is mediated by GABA receptors and Cl^{-} influx, or by K^{+} efflux, depending on the cell type[16].

Seizure propagation, the process by which a partial seizure spreads within the brain, occurs when there is sufficient activation to recruit surrounding neurons. This leads to a loss of surrounding inhibition and spread of seizure activity into contiguous areas via local cortical connections, and to more distant areas via long association pathways such as the corpus callosum[16].

Repetitive discharges lead to: 1) an increase in extracellular K+, which blunts the extent of hyperpolarizing outward K+ currents, tending to depolarize neighboring neurons; 2) accumulation of Ca++ in presynaptic terminals, leading to enhanced neurotransmitter release; and 3) depolarization-induced activation of the NMDA subtype of the excitatory amino acid receptor, which causes more Ca++ influx and neuronal activation. Seizures typically end, usually after seconds or minutes, and what underlies the failure of this spontaneous seizure termination in the life-threatening condition known as status epilepticus[16].

Causes:

1. Neonates(<1 month) :-

1.1. Perinatal hypoxia and ischemia

1.2. Intracranial hemorrhage and trauma

1.3. Acute CNS infection

1.4. Metabolic disturbances (hypoglycaemia, hypocalcaemia, hypomagnesemia, pyridoxine deficiency)

1.5. Drug withdrawal

1.6. Developmental disorders.

2. Infants and children (>1 month and <12 years):-

2.2. Febrile seizures
2.3. CNS infection
2.4. Developmental disorders
2.5. Trauma
2.6. Genetic disorders (metabolic, degenerative, primary epilepsy
2.7. syndromes)
2.8. Idiopathic.

3. Adolescents (12–18 years):-

3.2. Trauma
3.3. Infection
3.4. Brain tumor
3.5. Illicit drug use
3.6. Idiopathic

4. Young adults (18–35 years):-

4.2. Trauma
4.3. Alcohol withdrawal
4.4. Illicit drug use
4.5. Brain tumor
4.6. Idiopathic

5. Older adults (>35 years):-

5.2. Cerebrovascular disease
5.3. Brain tumor
5.4. Alcohol withdrawal
5.5. Metabolic disorders
5.6. Alzheimer's disease and other degenerative CNS diseases
5.7. Idiopathic [17].

Management &Treatment:

1. Management during a seizure

1.1. Move patient away from fire, traffic or water

1.2. Take away any objects that could harm the patient

1.3. Loosen tight clothes, remove glasses

1.4. Put something soft under the head

1.5. Turn patient on his or her side, so that saliva and mucus can run out of the mouth

1.6. Remain with the patient until he or she regains consciousness

1.7. Let the patient rest and then resume whatever activity he was doing, if he feels like it

2. Some Dont's

2.1. Do NOT try to put anything into the mouth

2.2. Do NOT give anything to drink

2.3. Do NOT try to stop the jerking, or restrain the movements[18].

Guiding principles to start anticonvulsant drug treatment in previously untreated patients

1. Carefully establish diagnosis.

2. Start drug treatment with one drug.

3. Start drug treatment with a small dose.

4. Gradually increase dosage until complete seizure control. This is the minimum maintenance dose.

5. The aim of treatment is to achieve the lowest maintenance dose which provides complete seizure control.

6. A gradual introduction of an anticonvulsant can produce therapeutic effects just as fast as rapid initiation with large doses, but with fewer side-effects.

7. Severe “intoxication” side-effects appearing at the beginning of the treatment can indicate too rapid or too large dose increases. Side-effects to anticipate include fatigue, excess sleep need, dizziness, or difficulty walking (ataxia).

8. If the initial drug of choice is not well tolerated, e.g., if side-effects occur or if the maximum tolerated dose does not produce seizure control, substitute the initial drug with another first line anticonvulsant.

9. A second anticonvulsant should be added gradually and the first then slowly withdrawn.

10. In case of acute withdrawal symptoms, e.g., recurrence of seizures, use diazepam as a control drug[19].

Neurocysticercosis:-

Neurocysticercosis (NCC) is a common cause of seizures and neurologic disease[20]. NCC is the more frequent parasitic disease of the CNS in immunocompetent patients associated with infection due to its larval form of *Taenia solium* (Figure-14)[21].

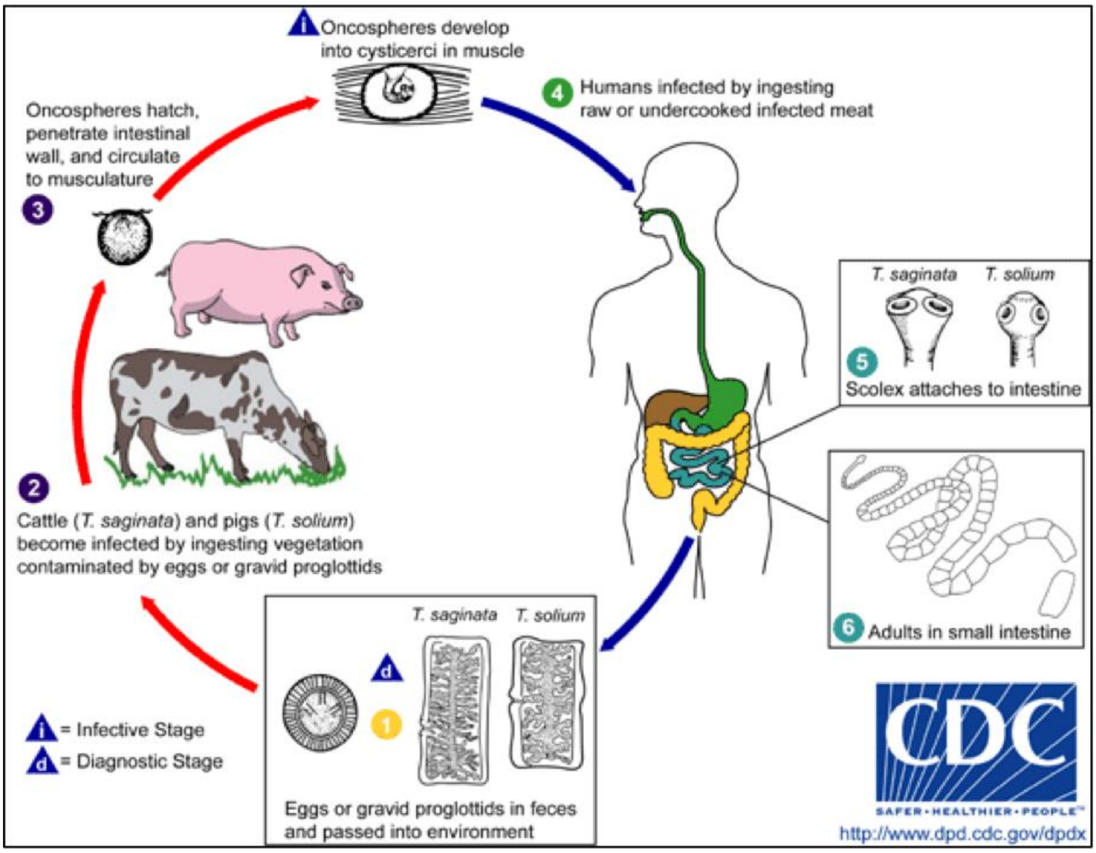


Figure 14. Life cycle of *Taenia solium* cysticercosis.

Incidence

It is believed that approximately 50 million people are infected with the taeniasis-cysticercosis complex around the world. In America, 350,000 patients suffer from this disease and a high percentage of them are affected neurologically. It is seen mainly in developing countries where there are worse hygiene conditions and lower socioeconomic and cultural factors which contribute to its transmission[21].

Clinical Presentation of NCC

Most children (> 80%) present with seizures particularly partial seizures. About a third of cases have headache and vomiting[20]. According to its presentation, laboratory test and imaging studies, NCC can be divided in active and inactive forms[21].

Staging

NCC is classified according to imaging studies in five stages

Non Cystic - It is asymptomatic and cannot be identified in imaging studies.

Vesicular-presence of multiple cysts of different sizes, smaller than 20 mm. Scolexes can be demonstrated in up to a 50% of the cases, located eccentrically at the walls of the cystic lesions. They appear as slightly small hyperdense nodules in CT and hyperintense nodules in Fluid Attenuated Inversion Recovery (FLAIR) MRI sequences. They are non-enhancing or mildly enhancing and are not surrounded by edema.

Colloidal Vesicular -There is evidence of fibrous capsule which enhances after contrast injection associated with perilesional edema on CT which is more evident on T2 and FLAIR sequences.

Granular Nodular- At this stage, there is cystic retraction and collapse that leads to the appearance of nodular structures in CT and MRI and they show enhancement after contrast injection and perilesional edema due to the inflammatory response from the host to the parasite.

Calcified Nodular-It is the inactive stage of parasitosis, where there is a total involution of the non-vital cyst with calcium deposits. There is neither perilesional edema nor enhancement; it is best observed in CT studies. In MRI, the Gradient Weighted sequences in T2 help improve the sensibility for the detection of calcium lesions at this stage[20].

Diagnosis

The clinical suspicion in patients of endemic areas associated with diagnostic imaging studies, laboratory and cerebrospinal fluid (CSF) tests searching for specific Immunoglobulin G (IgG) through Enzyme Linked Immuno Sorbent Assay (ELISA), contributes to reaching the NCC diagnosis[21].

However, CT has better performance in neurocysticercosis cases in their calcified stage due to its greater sensibility to detect calcium in the lesions than MRI.

Single enhancing lesion with visualization of a scolex confirms the diagnosis. For usual cases of suspected NCC, getting a CT scan is enough ; however MRI is more sensitive for detecting scolex and extra parenchymal NCC [20] .The eccentric DWI hyperintensity presumably representing the scolex has an important diagnostic implication because the detection of the scolex is pathognomonic of NCC and considered an absolute diagnostic criterion of the disease. In vesicular and colloidal vesicular stages, the scolex can present as a DWI hyperintense dot or comma-shaped structure[22].

Complications and Associated Findings

Arachnoiditis can be focal or diffuse, generating leptomeningeal fibrosis and neuropathy because of cranial nerves compression. It can be identified in imaging studies by the enhancement of the basal cisterns after contrast injection.

Vasculitis (cysticercus angiitis) is another radiological manifestation related to NCC. There is arterial focal narrowing which can compromise the bloodstream and develop brain infarcts generally in relation to the subarachnoid forms of the disease[21].

Differential Diagnosis

NCC lesions in its different stages should be differentiated from other cystic lesions such as tuberculoma, abscesses, arachnoid cysts, cystic neoplasms, including metastasis and other parasitosis. It is important to take into account the presence of calcifications, scolexes, cysts location and form and the coexistence of lesions in different stages[21].

Tuberculoma is the commonest differential diagnosis in developing countries. Certain criteria such as the presence of raised ICP, size of CT lesion> 20 mm, lobulated/ irregular shape and marked edema causing midline shift favour the diagnosis of tuberculoma. Tuberculomas are most often seen in the posterior fossa or base of the brain whereas NCC lesions are seen near the gray white matter junction of the cortex. Mantoux test, X-ray chest, CSF analysis and other tests for exclusion of tuberculosis should be done in all cases of enhancing lesions where the scolex is not seen[20].

In the year 2015, Singh A et al showed that out of 446 cases with partial seizures 239 cases were abnormal and 207 had normal CT scan findings. They stated that in India, tuberculoma and neurocysticercosis have highest prevalence in partial seizures[23].

In 2015, Ojha R et al in a study, out of 131 seizure patients, found 21 patients with NCC (male: 15, female: 6) and neuroimaging showed multiple NCC lesions in 8 and a single NCC lesion in 13 patients [24].

In a study done in 2006, Kotokey RK et al reported that maximum incidence of neurocysticercosis was found in the age group between 21 and 30 years and seizures being their commonest clinical presentation (100%). Of eleven patients (21.56%) had ring enhancing lesions with central scolex and fourty patients (78.44%) showed only ring enhancing lesions[25].

Kumar A et al in a study including 103 patients observed that children in the 11–20 years age group were most affected and solitary lesions were more common (66.7%) than multiple lesions (33.3%) on radiological diagnosis by CT scan. Among those 32% patients were positive for *Taenia solium* IgG antibodies in sera[26].

In a study of 119 cases, Kuzniecky et al revealed that CT scan can accurately detect structural abnormalities like disc or ring enhancing lesion, calcified or nodular lesion and small cystic lesion and can be used for evaluation depending on circumstances of seizures[7].

Management:-

Del Brutto et al stated that "Risk for seizure recurrence was lowered after cysticidal treatment in patients with enhancing lesions". Although both praziquantel and albendazole have been found to be effective in NCC, albendazole is better than praziquantel, less expensive and better tolerated. Phenytoin and carbamezepine are generally used as first line anticonvulsants in patients with seizures due to NCC[21].

Tuberculoma

Intracranial tuberculoma are usually solitary lesions, although 15- 34% are multiple[24]. Tuberculosis is a highly widespread disease in developing countries. Meningitis is by far the most frequent manifestation of tuberculosis in the central nervous system[27]. The diagnosis and management of central nervous system tuberculoma (CNST) is an important public health problem in both developing and industrialized nations[27].

Chest imaging is being carried out since earlier times for pulmonary tuberculosis in many patients with CNSTs. It is helpful when they are shown by chest imaging studies. A high index of clinical suspicion is needed to diagnose CNSTs[27].

Tuberculous meningitis (TBM) is the most important and dangerous form of extra pulmonary tuberculosis (EPTB). The most common form of intracranial tuberculoma is tubercular meningitis and affects mainly children and young adults[28].

Diagnosis

In CSF analysis, elevated protein levels were the most common abnormality[29]. Computed Tomography (CT) & Magnetic Resonance Imaging (MRI) are the most frequent imaging techniques utilized in characterization, localization of CNSTs and provide diagnostic information at presentation[27].

The radiological features of tuberculoma on CT scan vary according to its stage. Mature tuberculomas appear as a well-delineated round or oval ring enhancing mass with occasionally a target sign. The immature tuberculomas are iso- to hyperdense on the plain CT scan and show ring or nodular contrast enhancement following contrast administration[27].

Basal meningeal enhancement, hydrocephalus and basal ganglia infarctions form the common triad of neuroradiological findings and tuberculomas are assessed as the most characteristic lesions of TBM[28].

The MRI manifestations of CNSTs rely on whether it is caseating with a solid center, caseating with a liquid centre or noncaseating. The solid caseating granuloma gives isointense on T1 and is isointense or hypointense on T2-weighted images. The non-caseating granuloma usually appears hypointense on T1 and hyperintense on T2-weighted images, with homogeneous contrast enhancement[27].

In proton MR spectroscopy, lipids at 0.9 ppm, 1.3 ppm, 2.0 ppm and 2.8 ppm have been shown in tuberculomas that appear hypointense on T2 weighted images. Recently, choline (3.22 ppm) has been observed along with lipids at 1.3 ppm and 0.9 ppm in a histologically cellular tuberculomas that showed mixed intensity on T2 weighted image[27].

Vengsarkar US et al opined that the advent of CT has greatly influenced the diagnosis and management of intracranial tuberculomas[30]. CT appearance of 14 cases of tuberculoma by Welchman JM depicted that the tuberculoma appears as mass lesion which is isodense with the brain substance surrounding by intense unbroken ring on contrast enhancement[31].

Tandon PN et al a study series of 50 patients with intracranial tuberculomas treated with antituberculosis chemotherapy, Most of the small and medium sized lesions resolved completely. It was proved that tuberculomas of the brain account for 20 to 30 per cent of intracranial tumors in India and 41% of intracranial space occupying lesions (ICSOLs) are tuberculous in nature in pediatric age group[32].

Differential Diagnosis

In many cases, solitary tuberculoma may be indistinguishable from abscess (pyogenic, fungal), tumor, cysticercus granuloma and other ring enhancing lesions on CT morphology alone. In these cases clinical, history, laboratory investigation, MRI and MR spectroscopy is helpful for differentiating these similar lesions[29].

Treatment

The treatment of Central nervous system tuberculoma is becoming more conservative. It consisted of four antituberculous antibiotics (Rifampicin 10-20 mg kg; Oral 9-12 months, Isoniazid 10-15 mg kg; 9-12 months, Ethambutol 15-20 mg kg; Oral 2 months, Pyrazinamide 15-30 mg kg; Oral 2 months). The addition of fluoroquinolones may be of interest. Anticonvulsant treatment is mandatory for seizure control. In addition, surgical treatment should be considered for patients with life-threatening neurological involvement and for patients with lesions that fail medical treatment[27].

Intracranial abscess:

A brain abscess is an intra parenchymal collection of pus. The incidence of brain abscesses is approximately 8% of intra-cranial masses in developing countries and 12% in the western countries. They begin as localized areas of cerebritis in the parenchyma and evolve into collections of pus enclosed by a well vascularized capsule. It is still a life-threatening disease and remains a potentially fatal entity. It may lead to serious disability, or even death if misdiagnosed or managed improperly[33].

Incidence:-

The male to female ratio varied from 1.3:1 to 3:1. Most brain abscesses occurred in the first two decades of life[34].

Etiology and clinical features:-

Seizure is common as an initial symptom, occurring in 34% of patients. Majority, about 90% result from pericranial infection (sinusitis, mastoiditis, otitis media) and many who are hematogenous-borne (those from bacterial endocarditis) are multifocal, especially from cyanotic congenital heart disease.

Stages:-

Development of brain abscess can be divided into four stages:

1. Early Cerebritis: 1-3 days

A perivascular infiltration of inflammatory cells around a central core of coagulation necrosis.

2. Late Cerebritis: 4-9 days

Pus formation in necrotic center which is surrounded by inflammatory cells and fibroblasts.

3. Early Capsule Formation: 10-13 days

A capsule that is better developed on cortical side than on ventricle side of lesion

4. Late Capsule Formation: beyond 14 days

A well-defined necrotic center surrounded by a dense collagenous capsule[27].

Diagnosis:-

CT facilitates early detection, exact localization, and accurate characterization, determination of number, size and staging of the abscess. In the earlier phases, a non-contrast CT may show only low-attenuation abnormalities with mass effect. In later phases, a complete peripheral ring may be seen. The CT features of human brain abscess can be used to predict the degree of encapsulation. On contrast CT, uniform ring enhancement is virtually always present in later phases. On early phases the capsule will be difficult to visualize via conventional techniques, and double contrast CT often is helpful in defining encapsulation of abscess. Diffusion of contrast material into the central portion of the abscess was characteristic of the early cerebritis Stage. This central diffusion became less prominent in the late cerebritis stage and ceased with the appearance of encapsulation[34].

MRI findings also depend on the stage of the infection. In the early phase, MRI can have low T1-weighted (T1WI) signal and high T2-weighted (T2WI) signal with patchy enhancement. In later phases, the low T1WI signal becomes better demarcated, with high T2WI signal both in the cavity and surrounding parenchyma. The abscess cavity shows a hyperintense rim on non-contrast T1- weighted images and a hypointense rim on T2WI. Diffusion-weighted imaging (DWI) has sensitivity and specificity of over 90% for distinguishing abscess (low ADC) from necrotic tumor (high ADC).DWI usually shows restricted diffusion (bright signal) that helps to differentiate abscesses from necrotic neoplasms, which are not usually restricted.

MRS has been shown to be specifically beneficial in differentiating between brain abscesses and other cystic lesions[34].

Staphylococcus aureus was the most commonly found causative agent. In patients with abscesses originating from sinus, dental or otogenic infections, anaerobic bacteria dominated and most patients had multiple bacterial isolates. A majority of patients with diagnosed abscesses were treated with both surgical drainage and systemic antibiotics[35].

Nair KP et al showed that CT scan of brain in epileptic children had abnormal findings such as infective lesions which commonly included tuberculoma, neurocysticercosis and brain abscess) [6]. In another study by Rodriguez et all observed that incidence of brain abscess in patients with seizure was 3.24%[36].

Berlit P et al in a study managed 67 pts with brain abscess over a span of 19 years and found that the most frequent presenting signs and symptoms were neurological deficits and seizures. With advent of CT, burr hole aspiration of the abscess was possible in 30 cases[37].

Differential diagnosis

Radiological features alone are inadequate to differentiate pyogenic brain abscess from fungal, nocardial or tuberculous abscess, inflammatory granuloma (tuberculoma), neurocysticercosis, toxoplasmosis, metastasis, glioma, resolving haematoma, infarct, hydatid cyst, lymphoma and radionecrosis. However, fever, meningism, raised ESR, multilocularity, leptomeningeal or ependymal enhancement, reduction of ring enhancement in delayed scan and finding of gas within the lesion favor a diagnosis of abscess [33].

Treatment

Combination of high dose parenteral antibiotics with or without neurosurgical drainage. Abscess diameter more than 2 cm need surgical intervention. Prophylactic anticonvulsant should be continued[33].

Parenchymal calcification:

CT exam is the gold standard in detecting intracranial calcification[38].

Causes

1. Age-related and physiologic:-

Pineal gland, habenula, choroid plexus, falx cerebri, tentorium cerebelli, dura mater, petroclinoid ligament and sagittal sinus.

2. Congenital:-

Sturge-Weber syndrome, tuberous sclerosis, neurofibromatosis, lipoma, Cockayne syndrome, Gorlin syndrome.

3. Infectious

TORCH diseases, granulomatous infections, chronic viral encephalitis.

Cysticercosis:- Typical appearance is that of a small calcified cyst with eccentric calcified nodule, representing the dead scolex. The most frequent calcification locations are in the brain parenchyma, especially the gray-white matter junction and subarachnoid spaces in the convexities, ventricles, and basal cisterns.

Cryptococcosis: - affects immunocompromised patients. Calcifications can be present in both the brain parenchyma and the leptomeninges.

Tuberculosis: - Calcified parenchymal tuberculoma can occur in intracranial tuberculosis. The "target sign" formed by the calcified central nidus with peripheral ring enhancement signifies tuberculoma.

4. Endocrine and metabolic:-

Fahr disease, hypothyroidism, hypoparathyroidism, hyperparathyroidism, pseudohypoparathyroidism, post-thyroidectomy.

5. Vascular:-

Primary atherosclerosis, cavernous malformation, arteriovenous malformation, aneurysms and chronic vasculitis.

6. Neoplastic

Oligodendroglioma, craniopharyngioma, germ cell neoplasms, neurocytoma, primitive neuroectodermal tumor (PNET), ependymoma, dysembryonic neuroectodermal tumor (DNET), meningioma, choroid plexus papilloma, medulloblastoma, low grade astrocytoma, pilocytic astrocytoma, pinealoblastoma, schwannoma, dermoid, Epidermoid and calcified metastases (osteogenic sarcoma, mucinous adenocarcinoma)[38].

7. Metastasis-

Intracranial calcification may occur from lung, breast, colon cancer and osteogenic sarcoma. The presence of intracranial calcifications, their distribution and appearance in association with the clinic and biological data and in particular cases the follow up of the patient, help to make an accurate diagnosis[39].

Bajaj S et al studied 170 patients with CT, found that CT was normal in 64 patients and the commonest abnormality was the focal ring on disc enhancing lesion in 66 (62.2%) followed by calcification in 18 patients (16.9%)[40].

Murthy JM, in a study of 991 patients found that infections of CNS including single CT enhancing lesion (SCTEL) accounted for 77% of patients with epilepsy. In whom neurocysticercosis, SCTEL and small single cerebral calcific CT lesion (SSCCCTL) together accounted for 40% of etiological factors and neurotuberculosis for 10%[41].

Wadia et al and Young et al reported calcification in 3.75% and 3.8% of cases respectively[42]. According to Garg RK et al, in 77 patients with uncontrolled partial seizures, single cerebral calcification was the most common imaging abnormality[43].

Metastasis:

Metastatic brain tumors are the most common brain tumor in adults. Brain metastasis can present as a single tumor or multiple tumors. Research indicates that approximately 10–20% of metastatic brain tumors arise as a single tumor and 80% as multiple tumors within the brain[44]. Headache and seizures are the two most common symptoms[45].

Lung, breast, melanoma (skin cancer), colon and kidney cancers commonly spread to the brain. If the site of the primary cancer is not found, this is called an “unknown” primary site. The metastatic brain tumor usually contains the same type of cancer cells found at the primary site. However, recent research is suggesting that some of the tumors develop or acquire new genetic alterations in the primary tumor when they spread to the brain[45].

Diagnosis

MRI or CT is the most commonly available imaging tool and use of contrast dye makes the tumors easier to see. Magnetic resonance spectrometry (MRS) is used to measure chemical content in the brain. Full body PET scans can be helpful in identifying the primary cancer site when brain metastases are found first[45].

On CT- A peripheral location, spherical shape, ring enhancement with prominent peritumoural edema, and multiple lesions all suggest metastatic disease. Diffusion - weighted (DW) MR imaging may be useful for the differential diagnosis of ring – enhancing cerebral lesions (restricted diffusion in abscesses compared to unrestricted diffusion in cystic or necrotic glioblastomas or metastases) [45].

Lavizzari SG et al in a study of 193 cases, found 19 patients (9.8 %) with metastases and Pathan SA et al studied 439 patients with focal seizure, of whom brain metastasis was seen in 15 patients (3.4 %) [47].

Management

The three main categories of treatments include surgery, radiation and medical therapy (chemotherapy, targeted therapy or immune-based therapy). More than one type of treatment might be suggested. Antiepileptic drugs such as or phenytoin or lacosamide are commonly used to control seizures[44].

Tumor:

Tumors arising from the neuroepithelium encompass a subgroup of neoplasms collectively referred to as “Gliomas”. Of this subgroup; astrocytomas are by far the most common. Astrocytomas are further subdivided by a four grading system based on The World Health Organization (WHO) classification[48].

However, the most frequent presentation is new onset seizure activity, which occurs in 50–90% of patients. Taking into consideration clinical presentation, age, sex and typical CT features, it is possible to predict the nature of an intracranial tumor with a high sensitivity and specificity[49].

Localized Gliomas:

Pilocytic astrocytomas can be seen either sporadically or as a manifestation of NF-1 or NF-2 while subependymal giant cell astrocytomas are found in the setting of tuberous sclerosis. Calcifications are rare and have been reported in 11% of pilocytic astrocytomas examined by CT scan (Lee et al. 1989). As for the subependymal giant cell astrocytoma, calcification is a common finding seen on CT scan. T1-weighted MRI scans, the pilocytic astrocytoma classically appears as a well-circumscribed isointense cystic tumor with a mural nodule, which is enhanced following the administration of contrast agents. While on T2-weighted imaging, the solid component may in some cases be hypointense to the gray matter similar to that of the cerebrospinal fluid (CSF). This can help distinguish radiographically a pilocytic astrocytoma from a medulloblastoma (Arai et al. 2006)[48].

The most frequently used treatment regimen for malignant primary brain tumors include surgery for debulking or biopsy followed by postoperative radiation that is often combined with chemotherapy followed by 6 months of adjuvant chemotherapy[48].

Diffuse Gliomas:

The most commonly encountered WHO grade II primary brain tumors are the diffuse gliomas consisting of the well-differentiated astrocytoma, oligodendroglioma, and oligoastrocytoma. Collectively, these grade II tumors are well-differentiated, hypercellular neoplasms with minimal cellular pleomorphism and nuclear atypia. Mitotic figures are rare with microvascular proliferation and absent necrosis. They have indistinct borders, infiltrating deep into the gray matter[48].

On imaging, these tumors are hypointense to the brain on T1-weighted imaging and do not demonstrate enhancement following the administration of gadolinium. They are best visualized on the Fluid Attenuated Inversion Recovery (FLAIR) sequences where they appear as a hyperintense space-occupying mass which is the most sensitive imaging technique for all grade II diffuse gliomas[48].

Medulloblastoma:

On CT, they appear as well defined hyperdense mass lesion in the posterior fossa which shows uniform homogenous enhancement on contrast administration associated with hydrocephalus, peritumoral edema and punctate calcification [49].

Cystic lesions:

Colloid cyst:

Well defined rounded hypodense mass lesion located within the 3rd ventricle obstructing the Foramen of Monro causing the obstructive hydrocephalus. They may or may not show any enhancement on post contrast study[49].

Epidermoid cyst: Well defined homogenously hypodense mass lesions of CSF density with lobulated margins located in the cerebellopontine angle on pre contrast study. No enhancement was seen in either case on post-contrast study. No evidence of edema, calcification or ventricular dilatation was noted[49].

In a study, 150 children with seizures Patel PJ et al revealed 80% percent of the children had normal CT scan; 11.3% had specific abnormality such as infection and cysts[50].

Cerebral infarction:

Computed tomography (CT) will differentiate infarct from haemorrhage up to at least five days after stroke. Many larger infarcts are visible within six hours though the appearance is subtle and depends on how closely the scan is examined. CT scanning is mandatory to exclude intracranial haemorrhage or sizeable infarction before the thrombolytic drug is given[51].

Signs on CT:

a. Parenchymal hypodensity
b. Loss of grey/white differentiation
c. Effacement of cortical sulci
d. Local mass effect
e. Loss of the insular ribbon
f. Obscuration of the lentiform nucleus
g. Hyperdense middle or other cerebral artery

Advantages:

a. Widely available and less expensive than MRI
b. Excellent sensitivity for detecting acute haemorrhage

Disadvantages:

a. Less sensitive than DWI for the detection of acute ischemia.

Hyperacute: <12 hours

a. Normal in 50-60%
b. Hyperdense artery (dense MCA sign)
c. Obscuration of the lenticular nucleus
d. Loss of grey-white interfaces (insular ribbon sign).

Acute: 12-24 hrs

a. Low density basal ganglia
b. Sulcal effacement.

Subacute: 1 to 3 Days

a. Increasing mass effect
b. Wedge-shaped low density area involving gray and white matter
c. Possible hemorrhagic transformation

Chronic stage:

a. Well defined lucent zone in both cortex and white matter.
b. Gliosis with widening of adjacent sulci and ipsilateral ventricular dilation[52].

Uses of MRI in stroke

To distinguish haemorrhage from infarct in patients presenting late after stroke—but only if the appropriate sequences are used. More often shows an ischemic lesion than CT so may be more useful in "difficult" young patients with suspected stroke. In "difficult" strokes such as suspected venous infarction or carotid or vertebral dissections, as it may show the vascular anatomy also[51].The use of MRA in combination with MRI + DWI for determining the vascular territory of ischemic stroke has a sensitivity of 89%-100%, and positive predictive value of 95%-100%[53].

In 2015 Amaravati KS et al in a prospective study of 50 cases with new onset focal seizures, enumerated the etiological factors are Post stroke epilepsy (38%), Calcified granuloma (14%), Neurocysticercosis (14%), Tuberculoma (10%), Idiopathic (10%), Brain tumor (6%), Brain abscess (4%) and Meningitis(4%) and concluded that cerebrovascular disease was the most common etiology after 50 years of age[54].

Vascular lesions

Vascular malformations involving the brain are divided into subgroups, including arteriovenous malformations (AVM), developmental venous anomalies (DVA), Cavernous malformations and capillary telangiectasias[55].

Arteriovenous Malformation

The classic AVM also known as (pial AVM) results from an abnormal connection between the arteries that normally supply the brain parenchyma and the veins that would normally drain this region[55]. The peak age for presentation is in the 20 to 40 year old age group. AVM of the scalp are rare vascular lesions present as a pulsatile mass with a propensity to hemorrhage[56]. Imaging and pathology reveal an enlarged feeding artery, a nidus consisting of numerous arteriovenous shunts and dysplastic vessels, and an enlarged draining vein[55].

Imaging findings of AVMs vary depending on the size of the lesion, and presence of hemorrhage or calcification. CT and MR angiography can be utilized to demonstrate the feeding arteries and draining veins. Conventional angiography is still considered the "gold standard" for demonstrating the internal angioarchitecture. On CT, calcification may be seen in up to 30% of cases. On MRI, flow voids are seen within the lesion, giving the classic "bag of worms" appearance.T2* GRE may reveal hypointense blooming if hemorrhage or calcification is present[55]. AVMS can be treated with surgery, endovascular embolization, radiosurgery, or a combination of these methods[56].

Developmental Venous Anomaly

They are thought to represent anatomic variants that arise from maldevelopment of fetal cortical venous drainage, most likely resulting from recruitment of parenchymal veins to compensate for loss or absence of a portion of the cerebral venous system. DVAs are comprised of enlarged medullary veins that drain into a venous trunk that flows into a dural sinus or deep ependymal vein, resulting in a “palm tree” or caput medusa appearance on imaging[55].

The classic imaging finding of the caput medusa allows for ease of diagnosis on both contrast enhanced CT or MRI. On non-contrast CT, the draining vein will typically appear isoattenuating to slightly hyperattenuating to the cortex, but if acutely thrombosed, then a markedly hyperattenuating vein may be seen. MR imaging may reveal flow voids in the region of the medullary veins and draining vein depending on size. DVAs are “leave alone” lesions in that resection will result in venous infarction of the area drained by the DVA[55].

Minford AM et al, in a study of 82 patients with focal seizures, found that two patients (2.4%) had AVM. They concluded that a computed tomogram is indicated in every child with focal seizures[57].Seollo lavizzari balmer C carried out CT in 112 patients with partial seizures out of 38 abnormal CT finding, vascular abnormality was found in 1 case[58].In a study of 80 cases Chee MW et al, revealed focal CT abnormalities were seen in 26 of the 80 patients (32.5%) of which arteriovenous malformation (AVM) was seen in three patients[59].

MATERIALS AND METHODS

Source of data:

The study was conducted at R. L. Jalappa Hospital and Research Centre, attached to Sri Devaraj Urs medical college, Tamaka, Kolar. The study was conducted over a period of 18 months from January 2015 to June 2016. An informed consent was taken from individuals for their willingness to participate in the study.

Inclusion Criteria:

All patients with clinically diagnosed focal seizures who had abnormalities on CT scan of brain.

Exclusion Criteria:

Altered renal function test (Serum creatinine > 1.4 mg/dL).

Pregnancy

Method of collection of data:

This study was approved by the institutional review board and informed consent was taken from all the individuals prior to inclusion in the study. All the patients underwent CT brain (plain ± contrast study) with SIEMENS® SOMATOM Emotion® CT 16-slice scanner. Contrast enhancement was obtained using nonionic contrast agent Iopromide 300, at a dose of 50 ml in adults and 1.5 ml/kg body weight in children.

Examination technique:-

CT of brain was performed from orbito-meatal line upto vertex at 5mm interval in axial planes parallel to orbito-metal line. Post study reconstruction in axial plane was done at 1.2 mm section. Multiplanar sagittal and coronal reconstruction using standard algorithm was done as and when required.

CT parameters used were:-

- Standard brain (axial scan):

Adults:

- Slice thickness - 5 mm
- kV - 130
- mAs - 270
- Effective dose [mSv] - 3.84

Baseline data were collected from patients along with pertinent clinical history. Any previous studies, if available was requested and reviewed.

CT diagnosis was confirmed by one or more of the following:

- Response to treatment on follow up CT scan.
- CSF analysis.

- Surgery and histopathological findings.
- MRI / MR spectroscopy.
- Typical findings such as metastases in patients known to have primary malignancy elsewhere in the body.

Note: patients with 1. Parenchymal calcifications with no enhancement / edema, 2. Post traumatic complications 3. Vascular malformations and 4. Hydrocephalus did not require further evaluation.

CT findings, clinical diagnosis and final diagnosis were recorded in the proforma. Results were analyzed to evaluate causes for focal seizures.

A total of 151 patients were clinically diagnosed with focal seizures and referred for CT brain to department of Radiology. Of these, 86 patients had no abnormality on CT scan and they were given symptomatic treatment. 65 patients had abnormalities on the CT scan requiring further evaluation – they were included in our study.

Figure 15. SIEMENS® SOMATOM EMOTION 16® CT scanner used in the study.

RESULTS

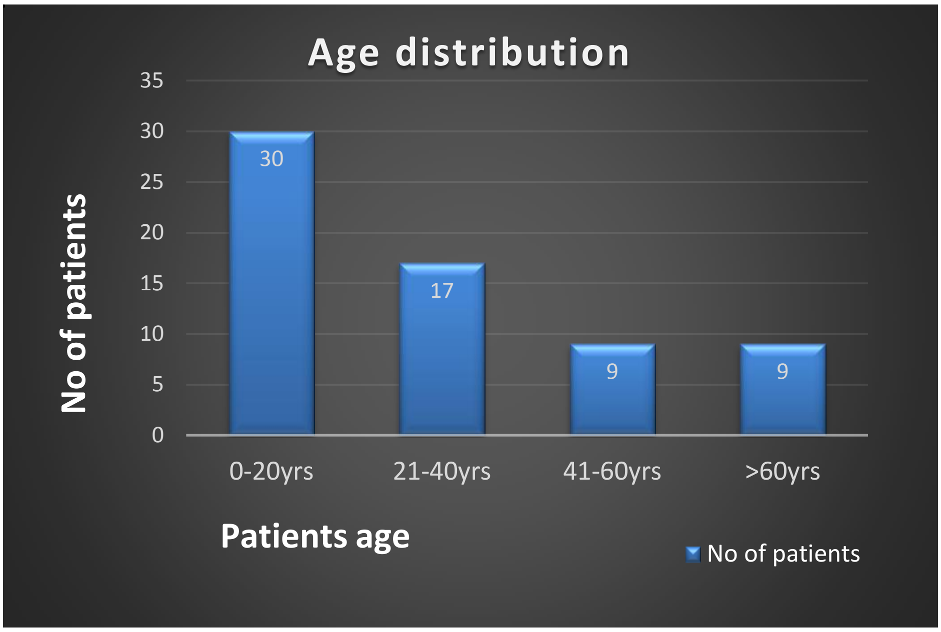


Figure -16: Age distribution of patients.

In our study of 65 patients, 30 patients were in the age group 0-20 years (46.1%). This was followed by 17 patients in the age group 21-40 years (26.1%), nine patients in the age groups 40-60 years and 9 patients were > 60 years (13.8%) (Figure 15; table 1).

Table1. Age distribution of Patients

Age group	Number of patients(%)
0-20	30(46.1%)
21-40	17(26.1%)
41-60	9(13.8%)
>60	9(13.8%)
Total	65 (100%)

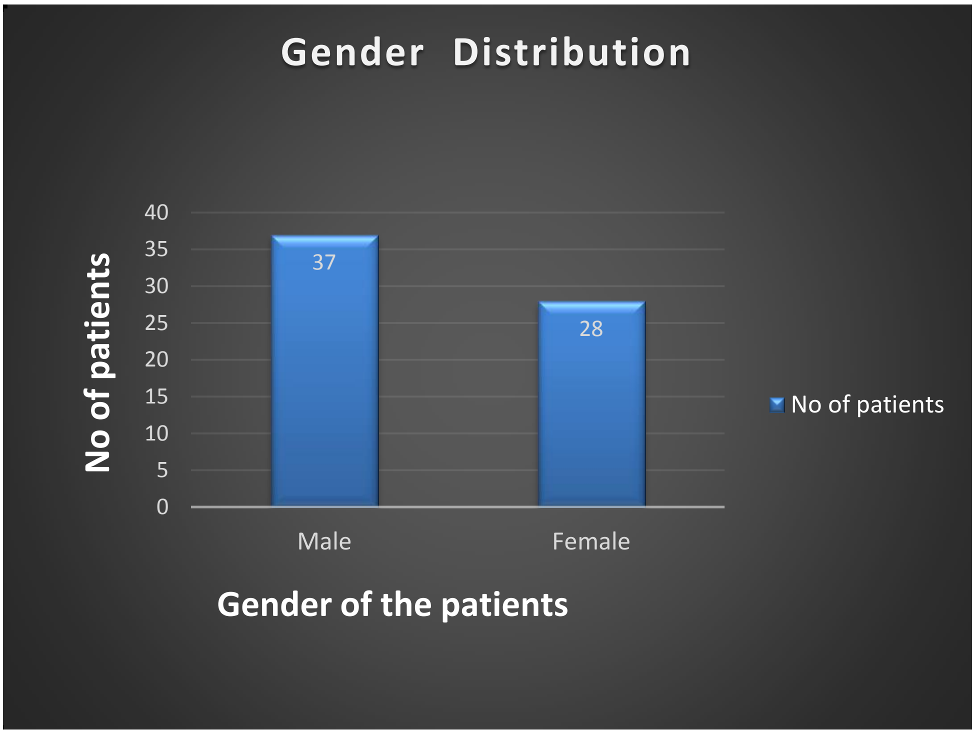


Figure 17. Gender distribution of patients.

There was slight male predominance 56.9% (n = 37) (Figure16; Table 2).

Table 2.Gender distribution of Patients

Gender	No of patients (%)
Male	37(56.9%)
Female	28 (43.0%)
Total	65 (100%)

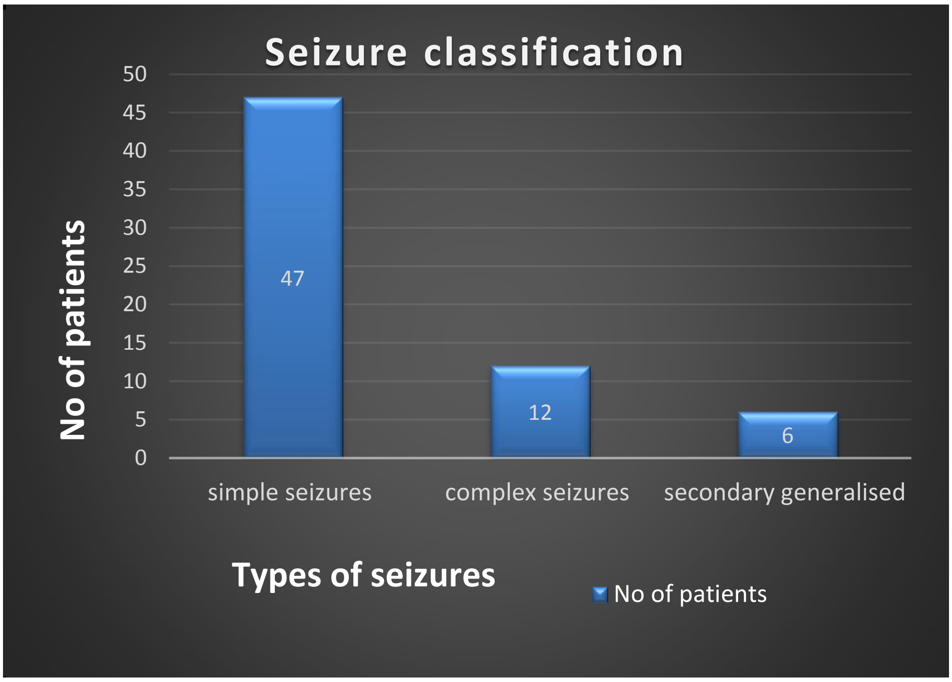


Figure 18. Types of seizures

Focal seizures were classified as simple seizures, complex seizures and secondary generalized seizures[13].In our study, more number of patients presented with simple seizures (n = 47; 72.3%) followed by complex seizures (n = 12; 18.4%) and secondary generalized seizures (n = 6; 9.2%) (Figure17; Table 3).

Table 3.Types of seizures

Clinical diagnosis	**No. of Patients**
Simple seizures	47 (72.3%)
Complex seizures	12(18.5%)
Secondary generalized seizures	6(9.2%)
Total	65(100%)

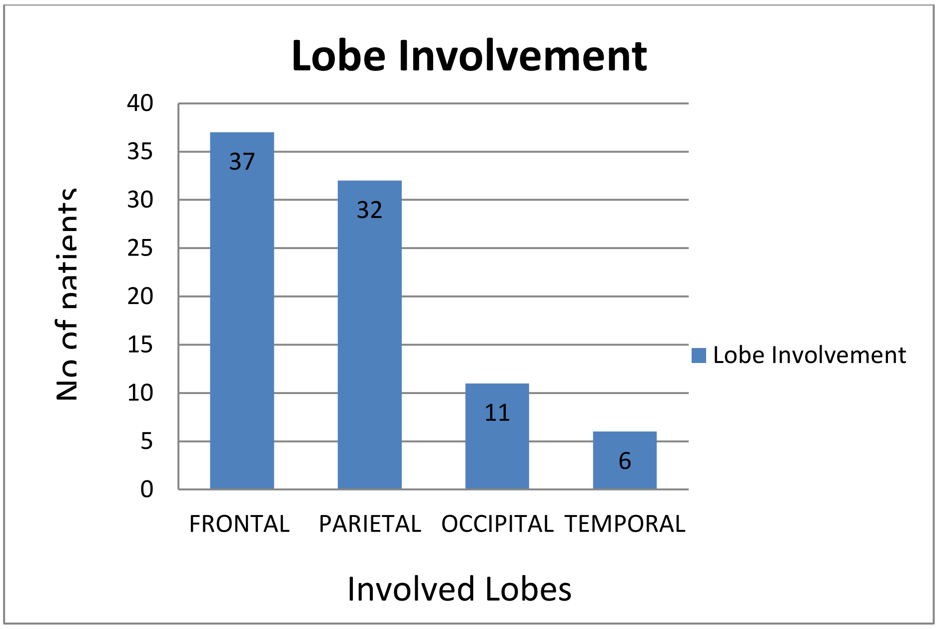


Figure 19. Lobe involvement

Frontal lobe was the commonest site involved (n = 37; 43%) followed by parietal lobe (n = 32; 37.2%), occipital lobe (n = 11; 12.7%) and temporal lobe (n = 6; 6.9%).

Note: A total of 21 patients had lesions in more than one lobe (Figure 8; Table 4).

Table 4. Lobe involvement

Lobe Involvement	**Number**
Frontal	37(43.0%)
Parietal	32(37.2%)
Occipital	11(12.7%)
Temporal	6(6.9%)
Total	86

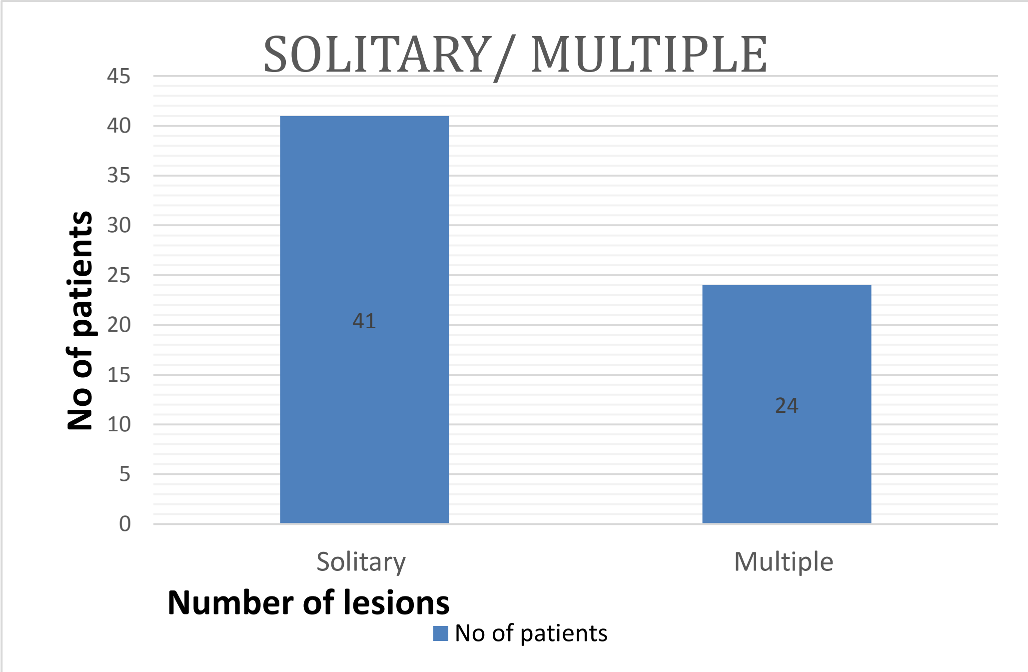


Figure 20.Number of lesions

In our study, solitary lesions were seen in 63% of patients (n = 41) and multiple lesions were seen in 37% of patients (n = 24) (Figure 19; Table-5).

Table 5. Number of lesions

Number of lesions	Number (%)
Solitary	41(63%)
Multiple	24(37 %)
Total	65 (100%)

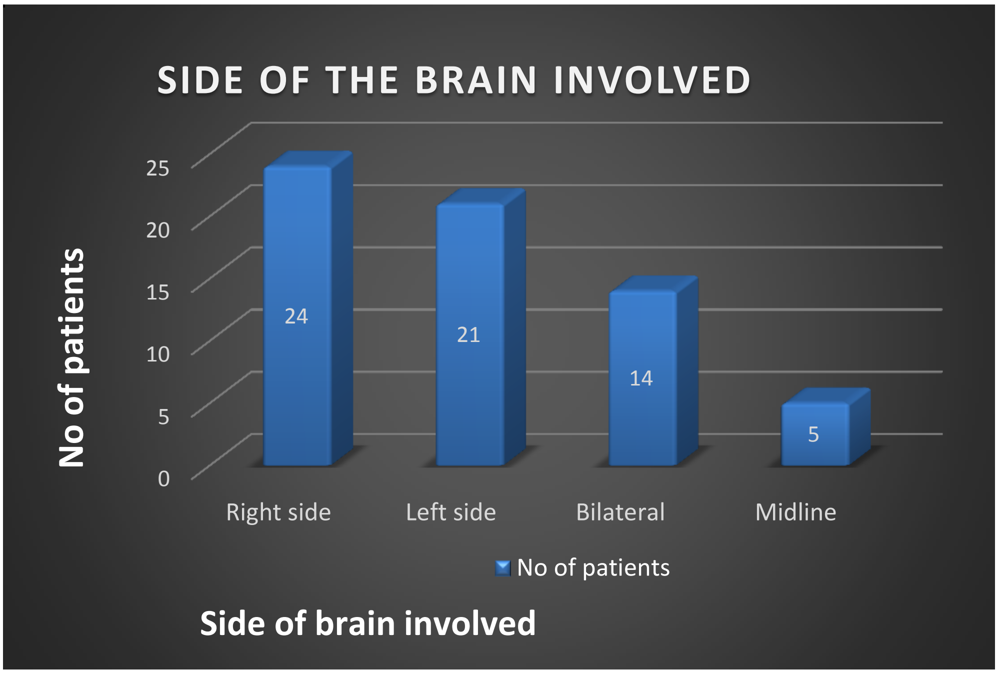


Figure 21.Side of brain involved

In our study, there were 24 patients with lesions in right cerebral hemisphere and 21 patients with lesions in left cerebral hemisphere (37.5% and32.8%) respectively. 14 patients had lesions in bilateral cerebral hemispheres (n = 14;21.8%) (Figure 20).There were five lesions (7.8%) in midline, which included two cases of pilocytic astrocytoma and one case each of medulloblastoma, colloid cyst, and arachnoid cyst. One patient had dilatation of all the ventricles.

Table 6.Side of brain involved

Side of brain involved	**No of patients (%)**
Right side	24(37.5%)
Left	21(32.8%)
Bilateral	14 (21.8%)
Midline	5(7.8%)
Total	64(100%)

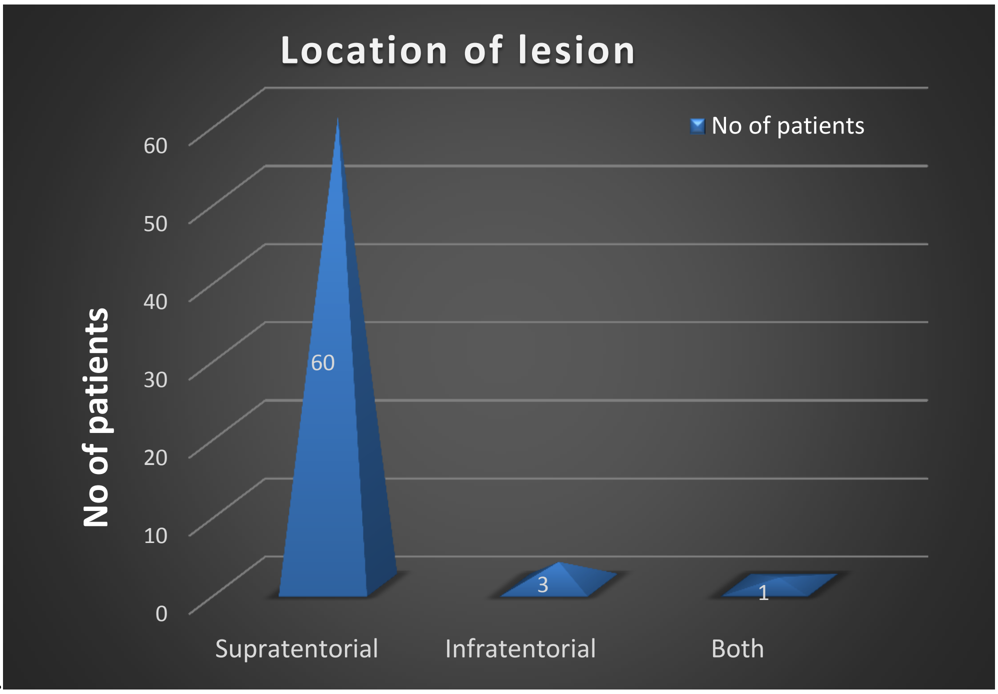
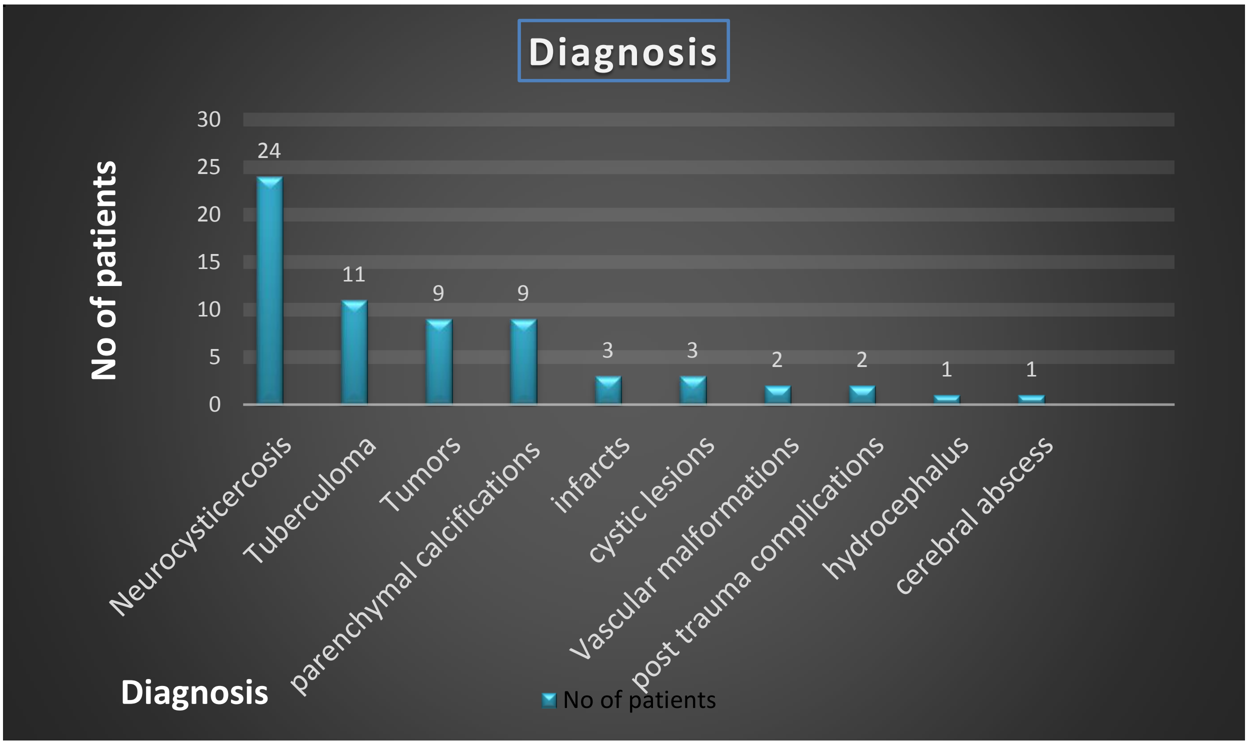


Figure 22. Location of lesion

In our study, more than 90% of patients had lesions that were supratentorial in location (n = 60; 93.6%). (Figure 21; Table7). Infratentorial lesions include medulloblastoma, pilocytic astrocytoma, metastasis and arachnoid cyst. In one patient with carcinoma of breast there was both supratentorial and infratentorial metastatic deposits. One patient had dilatation of all ventricles.

Table 7. Location of lesion

Location	**No. of Patients**
Supratentorial	60 (93.8%).
Infratentorial	3 (4.7%)
Both	1 (1.5)
Total	64 (100%)



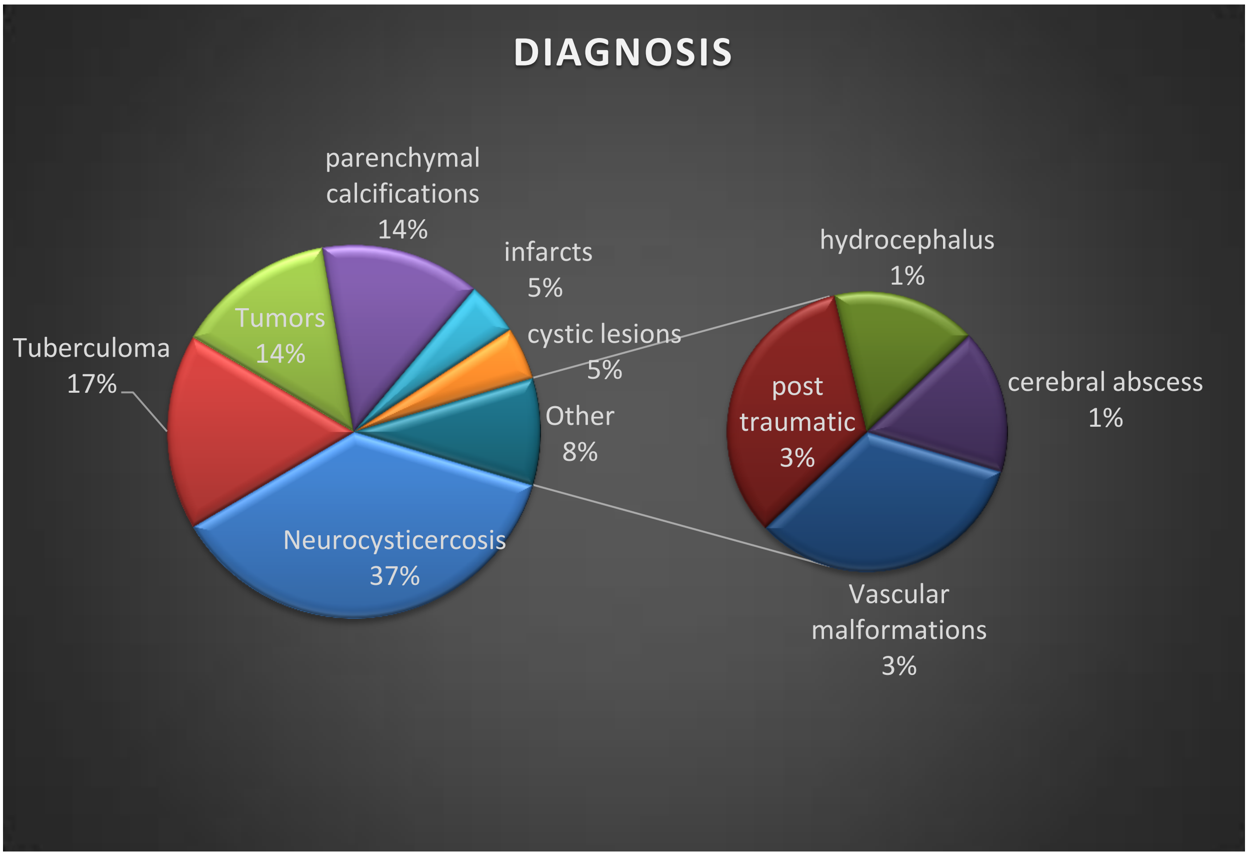


Figure 23. Diagnosis

Neurocysticercosis (NCC) was most common (n = 24; 36.9%) followed by tuberculoma (n = 11; 17%) These two conditions accounted for more than half of all cases for focal seizures. Other causes for focal seizures were parenchymal calcifications, tumors (n = 9 each), infarcts, cystic lesions (n = 3), post trauma complications, vascular malformations and hydrocephalus (n = 2 each) and cerebral abscess (n=1).

Table 8:- Diagnosis

Diagnosis	**No. of Patients**	**Percentage**
Neurocysticercosis	24	36.9%
Tuberculoma	11	17.3%
Tumors	9	13.8%
Parenchymal Calcifications	9	13.8%
Infarcts	3	4.6%
Cystic lesions (Epidermoid, colloid, arachnoid)	3	4.6%
Post traumatic (chronic SDH and gliosis)	2	3.0%
Vascular malformations	2	3.0%
Hydrocephalus	1	1.5%
Cerebral abscess	1	1.5%
Total	**65**	**100%**
SDH = subdural hematoma		

CT diagnosis was confirmed by one or more of the following: response to treatment on follow-up CT scan (n = 36); CSF analysis (n = 9), surgery and histopathological findings (n = 5) and MRI/MR spectroscopy (n = 10) (Figure 24).

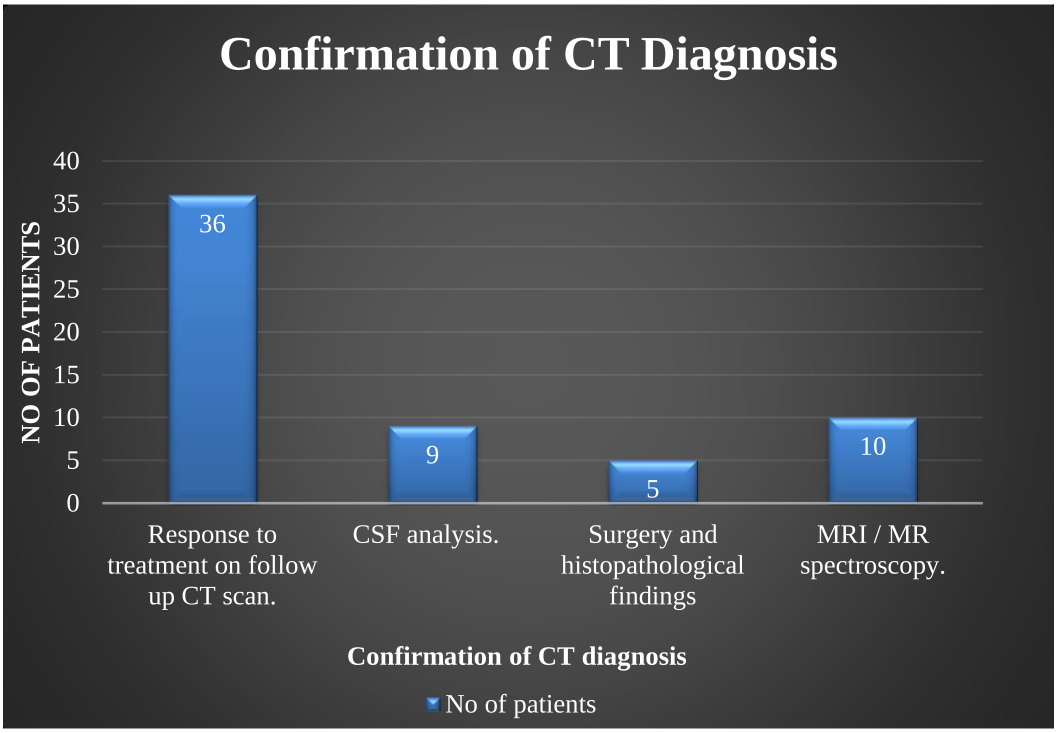


Figure 24. Confirmation of CT Diagnosis

IMAGES

Figure 25:- Nonenhanced CT scan of the brain, demonstrates the multiple cystic lesions with eccentric hyperattenuating scolex. No surrounding edema- vesicular stage of NCC.

Figure 26: Contrast enhanced CT scan of the brain showing a cyst with a enhancing wall and an eccentric hyperattenuating scolex– Vesicular stage of NCC.

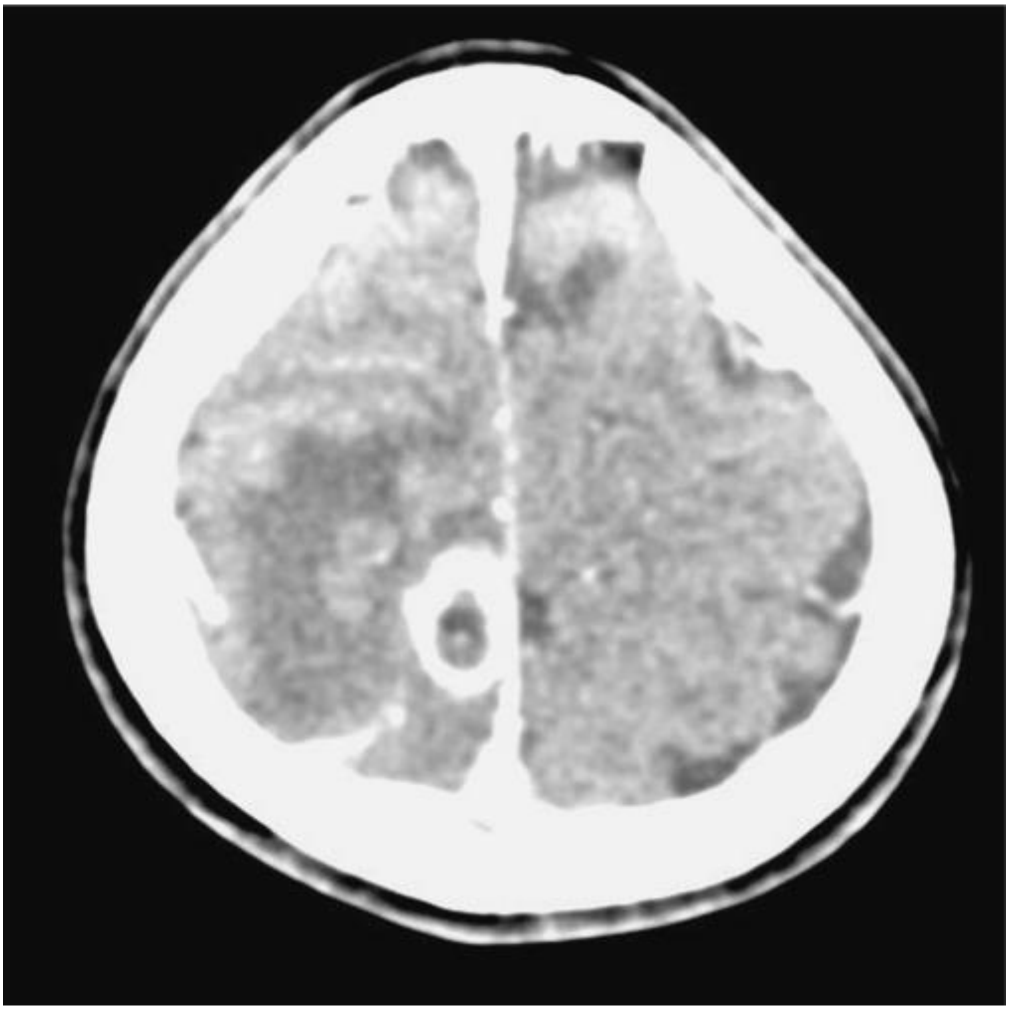


Figure 27:- CECT brain study showing well circumscribed ring enhancing lesion involving parietal lobe cortex with perilesional edema. The lesion show calcified scolex – Colloid-vesicular stage of NCC.

Figure 28:- Calcified granulomatous lesion with perilesional edema in right parietal lobe - suggestive of calcified stage of NCC.

Figure 29:- CECT showing well circumscribed ring enhancing lesion measuring 20 mm in left parietal lobe with moderate perilesional edema. No scolex. CSF analysis confirmed – Tuberculoma.

Figure 30:- Parenchymal calcification in right frontal lobe. No perilesional edema.

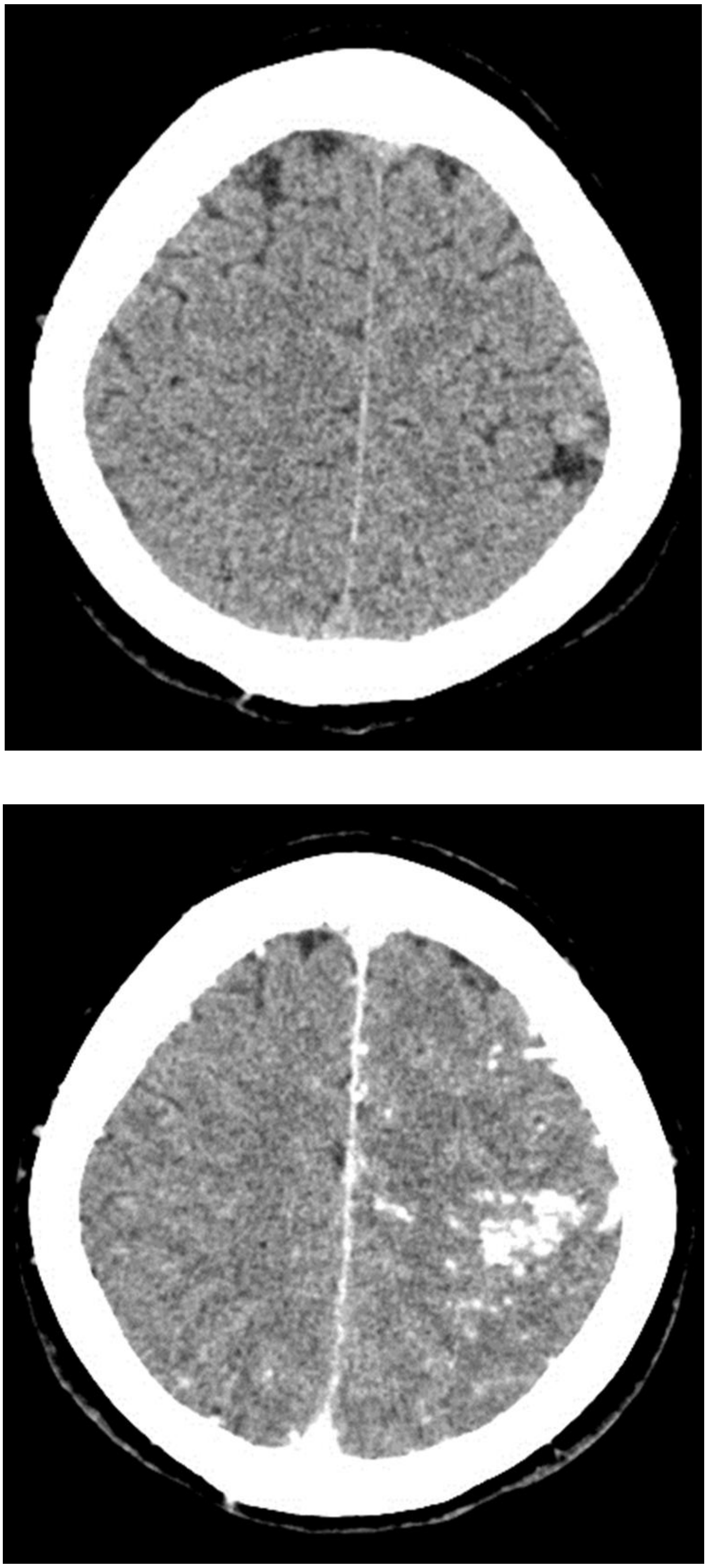


Figure 31:- Nodular serpiginous enhancing mass like lesion in left parietal lobe with dilated MCA branches and enlarged dural veins - AVM.

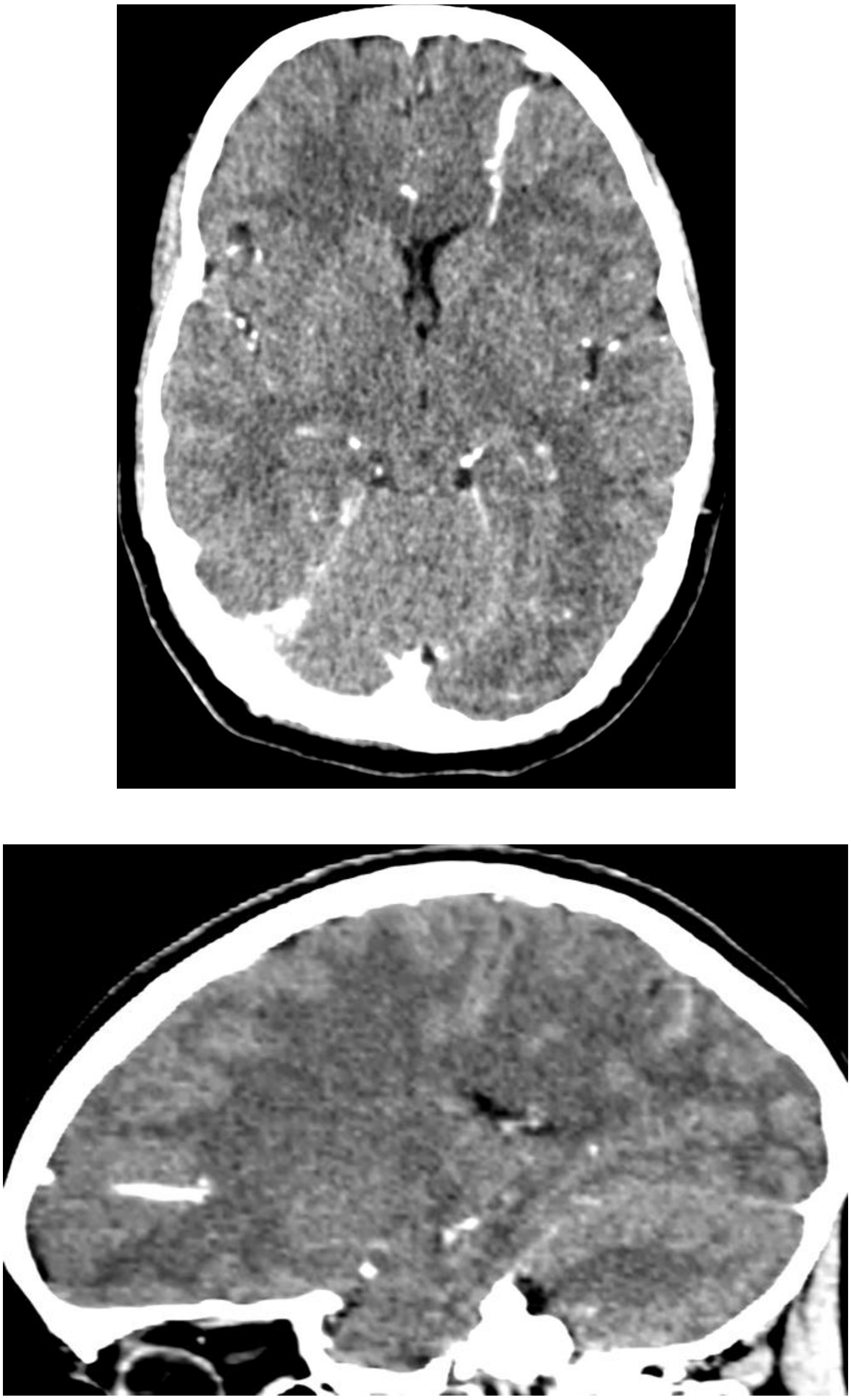


Figure 32:- Prominent contrast opacifying transcortical draining vein (2 mm in diameter) –developmental venous anomaly.

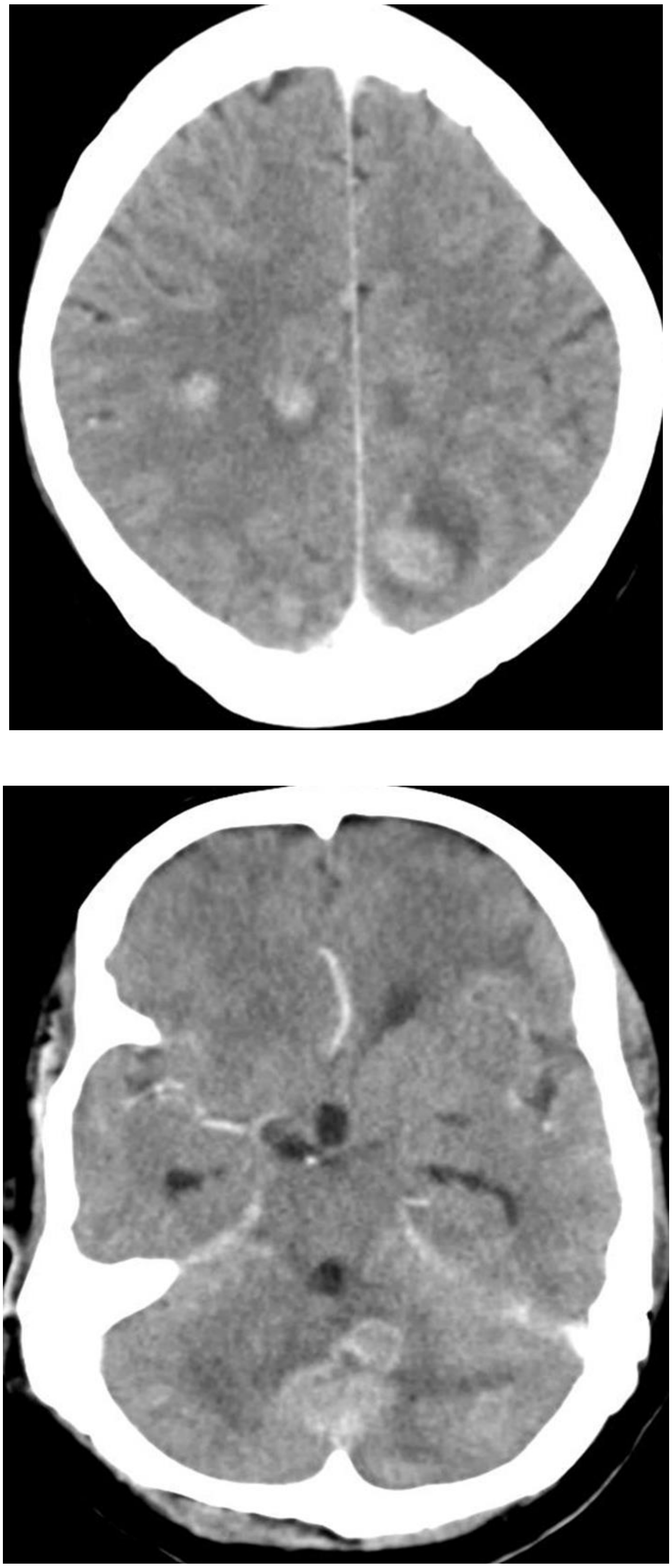


Figure 33:- Multiple rounded enhancing lesions with perilesional edema - metastasis from Carcinoma of breast.

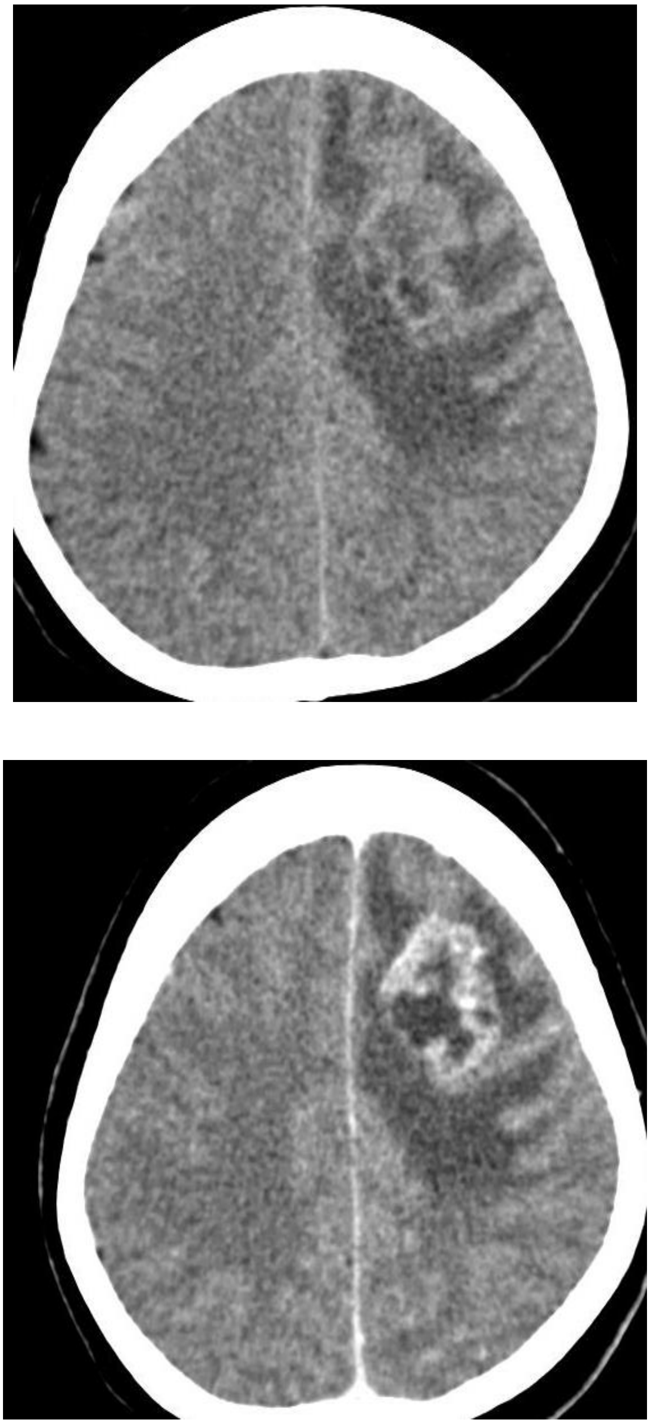


Figure 34:- Solitary lesion with necrotic core in left frontal lobe with perilesional edema, in a patient with carcinoma of lung. MRI confirmed metastatic lesion.

Figure 35:- A fairly well defined CSF density cyst lesion in posterior fossa – Arachnoid cyst.

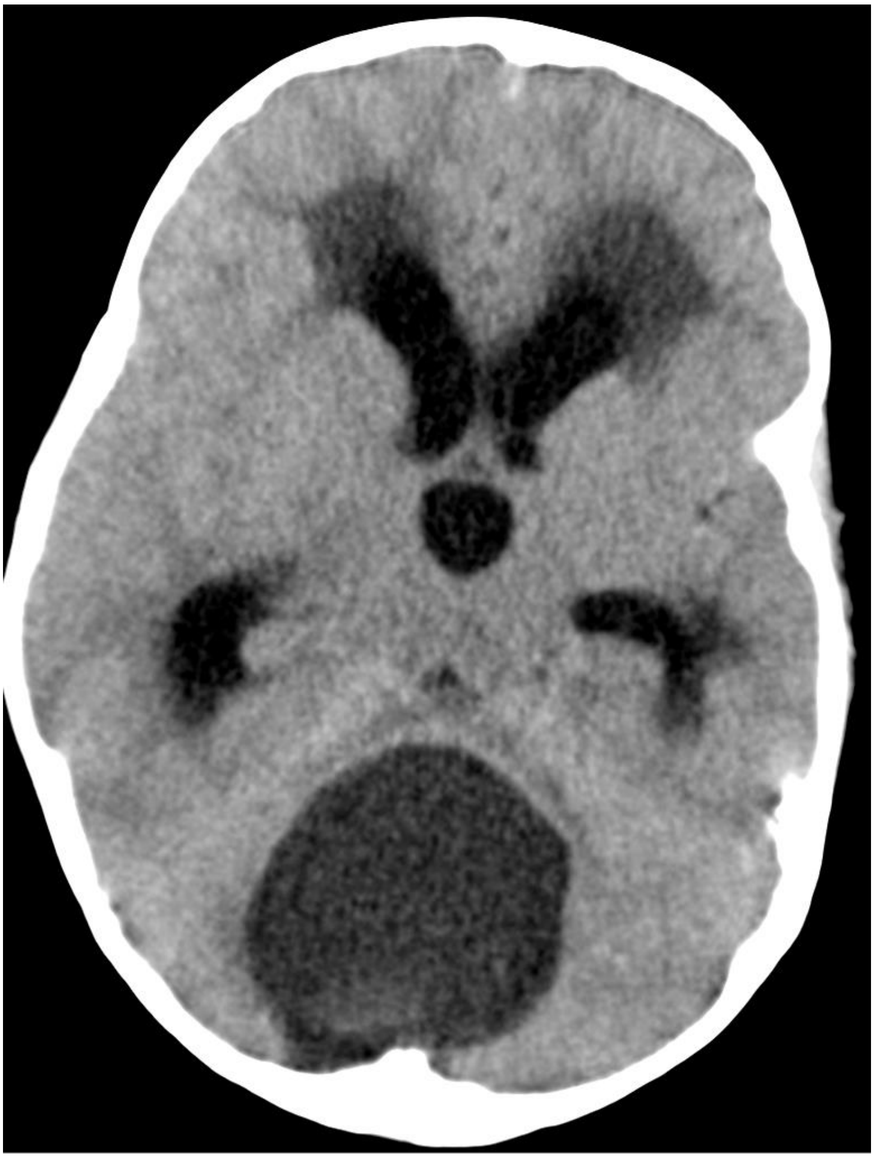


Figure 36:- Fairly well-defined rounded cystic mass lesion in midline posterior fossa. There is solid component seen posteriorly. Surgery + histopathology confirmed Pilocytic astrocytoma.

Figure 37.a) Fairly well-defined predominantly cystic mass epicentred in suprasellar region with enhancing mural nodular component. b) MRI FS T1WI + contrast showed enhancement of the wall and mural nodular component. Surgery + histopathology confirmed Pilocytic astrocytoma.

Figure 38. Venous infarct with hemorrhagic transformation

DISCUSSION

Patients presenting with focal seizures can have wide range of CT abnormalities depending upon the etiology. CT can reliably identify and localize the abnormalities so that further management can be planned accordingly. The aim of the study is to document the various abnormalities on CT scan and to study the causes of focal seizures.

151 patients who were clinically diagnosed with focal seizures were referred for CT brain to Department of Radiology. Of these 151 patients, 86 patients having no abnormality on CT did not require further imaging and they were treated symptomatically. A total of 65 (43.3%) patients with abnormalities on the CT scan were evaluated.

Our results are in agreement with data reported from other studies. In a study evaluating CT abnormality in 84 patients with focal seizures, abnormal findings were observed in 40 (47.65%) patients[60]. Another similar study in 26 patients reported abnormal finding in 13 (50 %) patients[1]. There is however, wide variability in incidence of abnormal CT findings in patients with focal seizures, with few studies reporting a lower incidence of about 35%[47, 61], while other studies reported a higher incidence of abnormal findings of upto 64%[62, 63].

In our study, the incidence of focal seizures was maximum in age group between 1 to 20 years (n = 30cases; 46.1%). We also observed that incidence of focal seizures decreases with advancing age. Our results are in agreement with Singh A et al**,** who in their study of 446 patients with focal seizures found that about 47 % (n = 210)were in age group of 1 to 20 years. Additionally, they also reported a reduction in incidence of focal seizures with advancing age[23].

In our study, incidence of focal seizures was higher among males (n = 37; 56.9%). Our results are in agreement with findings reported by Kafle DR et al who in their study of 70 patients with focal seizures also found a similar male predominance (n = 40; 57.1 %)[64].

In our study, we observed that solitary lesions (n = 41; 63 %) are more common than multiple lesions. However in a study done by Garg R K et al in 101 patients, showed that nearly half of the patients had solitary lesions (n = 49; 48.5 %)[65].

Of 65 patients in our study, frontal lobe was the commonest location (n = 37; 43%) for the lesions. Similar finding was seen in study done by Pandey J et al, who observed that more than 31% were seen in the frontal lobe[4].

In our study, supratentorial lesions (n = 60; 93.7 %) were seen in more patients. Our results are in agreement with Jindal N et al. who in a study of 80 patients found 61(76.2%) patients with supratentorial lesions[66].

In our study, diagnosis was confirmed by response to treatment / follow up CT scan in 36 patients, MRI in 10 patients, CSF analysis in 9 patients and histopathology in 5 patients.

In our study, we observed neurocysticercosis (NCC) to be the most common (n = 24; 36.9%) cause for focal seizures, followed by tuberculoma (n = 11; 17.3%). These two conditions accounted for more than half of all the cases. Other causes were parenchymal calcifications (n = 9), tumors (n = 9), infarct (n = 3), cystic lesions (n = 3), post trauma complications (chronic SDH and gliosis) (n = 2), vascular malformations (n = 2), hydrocephalus (n = 1) and cerebral abscess (n=1). Our results are in agreement with Yashodhara P et al who in a study of 40 cases found 24 (60%) had NCC, followed by tuberculoma 8(20%), neoplasms 5 (12.5%), and calcified granuloma, arachnoid cyst and brain abscess were found in one patient each (2.5%) [67].

Neurocysticercosis (NCC)

Among patients with NCC, 16 had ring enhancing lesions with central scolex and moderate perilesional edema (colloidal vesicular stage), some patients with multiple lesions showed NCC of various stages (from vesicular stage to calcified stage). Del Brutto et al stated that ring enhancing lesion with scolex is the absolute diagnostic criterion for (NCC)[68]. This was seen in most of our cases (Fig-24-27).

In our study, NCC was the primary cause for focal seizures in 24 patients (36.9 %).Our results are in agreement with Chandy M J et al who in a study of 30 patients and Garg R K et al who in a study of 151 patients, found NCC to be the most common etiology[69,65].

In our current study of 24 patients with NCC, 14 (58.4%) showed single lesion and 10(41.6%) had multiple lesions. This was seen in other studies. In a study of 109 patients by Gauchan E et al[70], 89 (81.6%) patients had single lesion and in a study of 51 patients by Bhattacharjee S et al 37 (71.1%) showed a single lesion[71].

Tuberculoma

In our study, eleven patients (17%) were diagnosed as tuberculoma. The lesions were ill defined isodense or hypodense areas, showing irregular ring enhancement with surrounding edema. Welchman JM described CT appearance of 14 cases of tuberculoma as mass lesion which is isodense with the brain substance surrounded by intense ring on contrast enhancement[31]. **(**Fig -28-29**)**

Diagnosis of tuberculoma was based on CT appearances along with one or more supporting evidences such as meningeal enhancement, past history of TB, CSF analysis, chest X-ray and MRI.

Out of eleven patients, four patients with neck stiffness showed leptomeningeal enhancement, four patients had past history of TB, five patients had elevated protein in CSF analysis and 2 patients were diagnosed by MRI (hypointense signal intensity on T2 weighted images and MRS showed increased lipid peak).

Of 11 patients with tuberculoma, 4 patients showed leptomeningeal enhancement. Zhang SR et al in a study which included 14 cases of intracerebral tuberculoma, 9 cases had leptomeningeal enhancement [72].

In our study of 11 tuberculoma cases, 7 had solitary lesion and 4 had more than one lesions. In 8 of the patients the lesion / largest lesion measured 20 mm or more. All lesions showed irregular enhancement with extensive perilesional edema. Rajshekar et al showed that out of 31 ring enhancing lesions, 25 were cysticerci, and 6 tuberculomas All cysticercus granulomas were less than 20 mm in size in comparison to tuberculomas which were greater than 20 mm. Moreover, 5 out of 6 tuberculomas were irregular in outline [73].

In our study, all patients with tubercular lesions resolved following ATT. Our findings are similar to findings reported by Tandon PN et al who in a study of 50 patients with intracranial tuberculomas treated with ATT, found that most of the small and medium sized lesions had resolved completely[32].

Zhang SR et al reported that majority of tuberculomas could decrease in size or get completely resolved with adequate antituberculous treatment[72]. Vengsarkar US et al opined that the advent of CT has greatly influenced the diagnosis and management of intracranial tuberculomas[30].

Parenchymal calcifications

In our study, nine patients (13.8 %) had parenchymal calcifications. The lesions were hyperdense with no surrounding edema or contrast enhancement. **(**Fig-30**)**

Our results are in agreement with Bajaj S et al who in a study of 170 patients, found calcification in 18 (16.9%) patients[40].

Primary tumors

There were three patients (4.6%) with tumors. All patients underwent subsequent MRI. All patients underwent surgery and the final diagnosis was pilocytic astrocytoma in two patients and medulloblastoma in the third. Of the two patients with pilocytic astrocytoma, one was located in midline in the posterior fossa and the other was suprasellar in location. Both patients had cystic masses with enhancing mural nodule. (Fig 36 & 37)

The patient with medulloblastoma had a large, well defined, mass lesion in midline posterior fossa. The mass was heteogenous with multiple small cystic components. The lesion showed heterogeneous enhancement.

Minford AM et al, in a study of 82 patients with focal seizures, found that two patients (2.4%) had primary tumours. They concluded that a computed tomogram is indicated in every child with focal seizures[57].

Metastasis

There were six patients (9.2%) with metastatic lesions. Our results are in agreement with Lavizzari SG et al who in a study of 193 cases, found 19 patients (9.8 %) with metastases[46]. (Fig- 33 & 34)

Age of the patients in our study ranged from 40 to 65 years. Primary lesion was carcinoma breast in three patients, carcinoma lung in two and renal cell carcinoma in one. The lesions were hypodense with extensive vasogenic edema causing mass effect on surrounding structures. The lesions showed homogenous, heterogeneous or irregular ring enhancement.

Infarcts

In our study, three (4.6%) patients had infarcts. Of these, two patients had MCA territory infarcts. Third patient had venous infract in left fronto-parietal region with hemorrhagic transformation. Singh A et al in a study of 446 cases with focal seizures, found 29 (6.5%) cases with infarcts[23] and Singh P et al in a study of 44 cases with focal seizures, found 6 (13.4 %) cases with infarcts[74]. (Fig- 38)

Vascular malformations

Vascular malformations were seen in two (3.0 %) patients - one patient had arterio-venous malformation in left parietal lobe and the other had developmental venous anomaly in left frontal lobe. Seollo-lavizzaribalmer C in a study carried out on 112 patients, found vascular abnormality in one patient (1.12%) [58] and Chee MW et al in a study of 80 patients found arteriovenous malformation (AVM) in three patients (3.75%) [59]. (Fig-31 & 32)

Abscess

Cerebral abscess was seen in one patient. The lesion appeared as a large hypodense lesion and showed peripheral ring enhancement with surrounding edema. On MRI the lesion was hypointense on T1and hyperintense on T2WI with restricted diffusion. MRS showed increased lactate peak. In a study Rodriguez et al observed that incidence of brain abscess in patients with seizure was 3.24%[36].

Post traumatic focal seizures

Two patients (3.0 %) had post traumatic focal seizures due to subdural hematoma and gliotic changes in one patient each. Hirani M in a study of 50 patients found 4 patients (4 %) had post traumatic seizures[75].Vidwan S in a study of 23 patients with focal seizures found gliotic changes in one patient.

Cystic lesions

Three patients (4.6%) had cystic lesions which included epidermoid cyst, arachnoid and colloid cyst. Patel P J in a study of 115 patients found 11.3 % having specific abnormality, such as infarction and cystic lesions (arachnoid and porencephalic cysts) [50]. (Fig- 35)

Hydrocephalus

One patient (1.5 %) had hydrocephalus. All ventricles (bilateral lateral, third and fourth) were dilated. No mass lesion was detected. Our results are in agreement with Kramer et al, Baheti et al and Singh P et al who in their study observed hydrocephalus in 1 (2.1%)[76],1 (2.1%) [1] and 2 (4.54 %)[74]patients of focal seizures respectively.

Causes of focal seizures in developed vs developing countries

In our study, NCC has higher prevalence than tuberculoma (37% vs 17%). Similar findings have been reported elsewhere in India. Singh S et al in their evaluation of 44 cases with focal seizures, observed NCC in 7 patients (15.91%) patients and tuberculoma in 4 patients (9.1%) [77]. Trentin AP et al in their study of 330 cases of focal seizures from brazil found NCC to be commonest abnormality (37.4%)[78]. Few studies however have reported equal incidence for NCC and tuberculoma (13% vs 12%) [13].

Our results are different compared with few other studies. A study conducted in Western Rajasthan by Bahethi R et al reported cerebral atrophy as a cause for focal seizures in 23% of patients, followed by intraparenchymal calcifications (11%). They had a significantly lower proportion of patients with infectious causes such as NCC or tuberculoma[1]. Possible reasons for this difference could be cultural and socioeconomic differences between these regions, including eating habits.

In a study conducted in U.S. among seizure patients, alcohol withdrawal /drug abuse and head trauma accounted for 19 % and 7.8% respectively whereas infectious diseases like NCC and meningitis accounted only for miniscule 3.4% (2.2% and 1.3%)[76], highlighting the social and cultural differences when compared with our study[76].

In a retrospective study performed in Pennsylvania, USA in 397 patients with focal seizures, 180 patients (46%) had abnormal CT scans. The most common lesion was a slowly growing tumor in 46 patients (11.6%) followed by generalized and focal atrophy, infarctions, infections, arteriovenous malformations, cysts, tuberous sclerosis and leukodystrophies. Sixteen percent of the lesions demonstrated by CT were amenable to corrective surgery[79].

An association between NCC and epilepsy is found in various studies in Africa and Latin America with prevalence of 30%.The incidence in Asia varies significantly – 50 to 80%[76].

Role of CT in focal seizures

Few studies have reported the importance of CT and MRI in focal seizures. International League against Epilepsy guidelines for neuroimaging studies recommend CT in patients with seizures, whenever magnetic resonance imaging is not available. It also recommends that patients who have intractable seizures have an MRI study if a CT is normal[7]. This suggests the role of CT as an initial investigation for evaluation of focal seizures.

Few studies have shown no significant difference in incidence of abnormal CT and MRI findings in patients with focal seizures (27.8 % vs 30.5 %). The authors concluded that sensitivity of CT was similar to MRI[80.]

In our study of 151 patients with focal seizures, CT scan was normal in 86 patients – they didn't required any further imaging and were offered symptomology treatment.

65 patients had abnormalities on CT scan. Only 10 patients out of a total of 151 patients with focal seizures needed further imaging with MRI. This shows that CT scan is a reliable modality in evaluation of focal seizures.

CONCLUSION

NCC and tuberculoma were the commonest cause for focal seizures. CT helped in identifying the various stages of NCC. CT also helped in differentiating NCC from tuberculoma. Other etiological factors included parenchymal calcifications, metastasis, tumors, vascular malformations, abscess and infarcts.

CECT helps in screening patients with focal seizures in order to identify patients with structural abnormalities. Majority of patients have classical findings on CT and undergo prompt treatment. A minority of patients may require further evaluation such as MRI, and CSF analysis.

Given its easy availability and affordability, CT is the primary investigation in evaluation of focal seizures.

SUMMARY

Focal seizures are one of the common health problems in developing countries like India. CT is the primary modality of choice for evaluation of focal seizures because of its easy availability, affordability and short scan time compared to MRI.

The aim of the study is to describe the CT scan findings in patients with focal seizures and to correlate the CT scan findings with probable etiology in these patients.

151 patients who were clinically diagnosed with focal seizures were referred for CT brain to Department of Radiology. Of these 151 patients, 86 patients having no abnormality on CT did not require further imaging and they were treated symptomatically. A total of 65 (43.3%) patients with abnormalities on the CT scan were evaluated.

More than 45% of patients (n = 30; 46.1%) were in the age group of 0-20 years. There was slight male preponderance (n = 37; 56.9%). Simple seizures (n = 47; 72.3%) were more common compared to complex seizures (n = 12; 18.4%).Frontal lobe was the commonest site involved (n = 37; 43%) followed by parietal lobe (n = 32; 37.5%). 23 patients had lesions in more than one lobe. Solitary lesions (n = 41; 63%) were more common than multiple lesions (n = 24; 37 %). Supratentorial location of lesions was more common compared with infratentorial location.

Neuroinfections were the commonest cause for focal seizures (n = 36; 55.7%), which included NCC in 24 patients (36.9%) followed by tuberculoma (n = 11; 17%) and abscess (n = 1; 1.5%). Neuroinfections accounted for more than half of our cases this is in agreement with a number of studies carried out in various parts of India. However, neuroinfections were accounted for miniscule of cases in western studies.

Other etiologies included parenchymal calcifications and neoplasm (n = 9 each; 13.8%). Three patients had infarcts and cystic lesions. Post trauma complications (chronic SDH, gliosis) and vascular malformations were seen in two patients each. One patient had hydrocephalus.

Most of the malignant lesions were metastasis (n = 6; 9.2%) and seen in patients aged from 40 to 65 years. Remaining three patients had glioma (n = 2) and Medulloblastoma (n = 1).

Our study confirms the importance of CT in evaluation of focal seizures. CT is able to identify findings such as site of lesion, density of lesion, surrounding edema, hemorrhage, infarction, calcifications, mass effect and pattern of contrast enhancement of the lesion.

Among 151 patients with focal seizures who had CT scan, only 10 patients required further imaging with MRI. The remaining 141 patients were adequately diagnosed by CT.

CT brain (plain ± contrast) study remains the key investigation for accurate diagnosis in patients with focal seizures and helps in early management. We conclude that CT should be carried out in every patient with focal seizure to rule out or confirm any organic lesion.

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ANNEXURE - I

PROFORMA

Demographic details:

Name:

Age:

Sex: ☐**Male** ☐**Female**

Hospital No:

Chief complaints:

History of present illness:

Type of seizure:

Duration of seizure:

Fever:

Headache:

Trauma:

Others:

Associated with other complaints:

Investigations:

TLC:

ESR:

Chest X-ray:

CSF:

Blood urea:

Serum creatinine:

MRI:

Clinical Diagnosis:

CT Findings: any lesion present:

1. Site and location: Frontal ☐ parietal ☐ temporal ☐ occipital ☐

2. Shape &: well defined ☐ ill-defined ☐

   Margins: Regular ☐ Irregular ☐

3. Location: Right☐ left ☐

4. Density: Hemorrhage ☐ Fat☐ Air ☐ Fluid ☐ Bone ☐

5. Calcification: Yes ☐ No☐

6. Surrounding edema Yes ☐ No☐

7. Hemorrhage Yes ☐ No☐

8. Hydrocephalus Yes ☐ No☐

9. Atrophy Yes ☐ No☐

10. Infarction Yes ☐ No☐

11. Any contrast enhancement Yes ☐ No☐

12. Type of contrast enhancement. Homogenous☐ Heterogenous ☐

CT diagnosis:

Final diagnosis:

Treatment:

Follow up CT scan:

ANNEXURE-II

Name of the subject:

Age :

Gender :

a. I have been informed in my own language that this study involves use of contrast material as part of procedure. I have also been explained the possible risk of radiation with CT scan. I thoroughly understand 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 ___________________ (chief researcher/ 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

Signature of the witness: Date:

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:

ANNEXURE-III

Patient Information Sheet

Title: **THE ROLE OF CT SCAN IN FOCAL SEIZURES**

Name of the principal investigator: **Dr. BHARATH REDDY M**

Name of the organization: **SRI DEVRAJ URS MEDUCAL COLLEGE, TAMAK, KOLAR**

Co-investigators: **Dr. PURNIMA HEGDE, Dr. ANIL KUMAR SAKALECHA**

I, **Dr. BHARATH REDDY M**, a postgraduate student in the department of Radio-diagnosis would like to involve you as a part of my study because your presentation is unusual. This study will help others know one of the unusual presentations and will help in management of similar patients in the future.

You will undergo routine investigations.

Confidentiality will be maintained with respect to your name, hospital number and contact details and only the findings and reports will be used to present in a conference or publish in a journal.

Your participation is voluntary and you are free to withdraw from the study at any point of time. Any query regarding investigations and management will be answered.

Person to contact in case of any queries: Dr. BHARATH REDDY M

POSTGRADUATE,

DEPARTMENT OF RADIODIAGNOSIS,

SDUMC, KOLAR.

ANNEXURE-IV

KEY TO MASTER CHART

AE	Antiepileptic
ATT	Anti-tuberculosis treatment
ALB	Albendazole
AVM	Arteriovenous malformation
B/L	Bilateral
CPS	Complex partial seizures
DVA	Developmental venous anomaly
F	Female
F/U	Follow up with CT scan
K/C/O	Known case of
LT	Left
LM	leptomeningeal enhancement
LT MCA I	Left middle cerebral artery infarct
M	Male
METS	Metastasis
MU	Multiple

MCA	Middle cerebral artery
MRS	Magnetic resonance spectroscopy
N	No
NCC	Neurocysticercosis
PA	Pilocytic astrocytoma
PC	Parenchymal calcifications
PS with SG	Partial seizures with secondary generalization
RT	Right
SO	Solitary
SDH	Subdural hematoma
SPS	Simple partial seizures
SURG	Surgery
TB	Tuberculosis
TUB	Tuberculoma
VI with HT	Venous infarct with hemorrhagic transformation
Y	Yes

Sl. No	ID	Age	Sex	Type of Seizures	No of Lesions	Side Of Brain Involved		Lobe Involved				Largest Lesion (in mm)	CT findings	NCC Staging				Treatment	Confirmation of CT diagnosis
				SPS/CPS	SO/MU	Right lobe	left lobe	Frontal	Parietal	Temporal	Occipital			I	II	III	IV		
1	1111	4	M	SPS	MU		Y	Y	Y			7 mm	NCC		Y			AE +ALB	F/U CT
2	2222	60	F	CPS	SO		Y		Y				AVM					Endovascular Embolization	SURG
3	3333	18	M	SPS	MU	Y		Y	Y			9 mm	NCC		Y	Y		AE + ALB	F/U CT
4	4444	11	F	CPS	SO		Y	Y				(6 mm)	NCC	Y		Y		AE + ALB	F/U CT
5	5555	24	M	SPS	SO	Y		Y				9 mm	NCC				Y	AE + ALB	F/U CT
6	6666	16	M	SPS	SO	Y			Y			10mm	NCC		Y			AE + ALB	F/U CT
7	7777	14	F	CPS	MU		Y	Y	Y			13mm	NCC		Y			AE + ALB	F/U CT
8	8888	18	M	SPS	MU	Y	Y	Y	Y			19-20mm	TUB					AE +ATT	LM + k/c/o TB + F/U CT
9	9999	40	F	SPS	SO	Y					Y	9 mm	NCC	Y				AE + ALB	F/U CT
10	1010	22	F	SPS	SO		Y	Y				7 mm	NCC				Y	AE + ALB	F/U CT
11	1212	60	M	CPS	MU	Y		Y	Y	Y			VI with HT					AE	
12	1313	33	F	SPS	MU	Y		Y			Y	7x4 mm	PC					AE	
13	1414	27	M	SPS	MU	Y	Y	Y	Y		Y	11mm	NCC		Y	Y		AE + ALB	F/U CT
14	1515	30	F	SPS	SO		Y		Y			3mm	PC					AE	
15	1616	50	F	SPS	MU	Y	Y	Y					Gliosis post traumatic					AE	

16	1717	35	F	SPS	MU	Y			Y		Y	2mm	NCC					AE	
17	1818	17	M	SPS	SO		Y	Y				11mm	NCC		Y			AE + ALB	F/U CT
18	1919	5	F	PS with SG	SO							49 x 44 mm	PA					Surgery	Histopathalogy + MRI
19	2020	13	M	SPS	SO	Y		Y				14-15mm	TUB					AE +ATT	MRS + F/U CT
20	2121	8	M	PS with SG	MU	Y	Y	Y	Y	Y		6 mm	NCC				Y	AE + ALB	F/U CT
21	2323	40	F	CPS	MU	Y	Y	Y	Y			42 x 40 mm	METS.					Radiation therapy	k/c/o ca of right breast
22	2424	15	M	SPS	SO		Y		Y			9 mm	NCC		Y	Y		AE + ALB	F/U CT
23	2525	17	M	SPS	SO	Y		Y				10x4 mm	PC					AE	
24	2626	12	F	SPS	MU		Y	Y	Y			8x4mm	PC					AE	
25	2727	1	F	SPS	SO								Hydrocephalus					AE	
26	2828	5	M	SPS	SO	Y			Y			8mm	NCC		Y	Y		AE + ALB	F/U CT
27	2929	16	F	SPS	SO		Y	Y				11mm	NCC	Y			Y	AE + ALB	Follow up CT scan
28	3030	50	M	CPS	MU	Y	Y		Y		Y	31x25 mm	METS					Radiation therapy	k/c/o ca of left kidney
29	3131	5	F	SPS	MU		Y	Y	Y			22mm	TUB					AE +ATT	CSF + F/U CT
30	3232	35	M	CPS	SO	Y	Y	Y				19 x 17mm	Abscess					AE + Antibotics	MRS + F/U CT
31	3434	7	F	SPS	SO	Y			Y			24 mm	TUB					AE +ATT	CSF + F/U CT
32	3535	60	M	SPS	MU		Y	Y	Y	Y			LT MCA I					AE	MRI (DWI)
33	3636	35	M	SPS	SO	Y						11x6 mm	PC					AE	

34	3737	62	F	SPS	SO		Y	Y				21mm	TUB					AE +ATT	MRS + (k/c/o TB) + F/U CT
35	3838	16	F	SPS	SO	Y		Y					RT MCA I with Meningioma					AE + Follow up	
36	3939	13	M	SPS	MU	Y	Y	Y				8 mm	NCC		Y	Y		AE + ALB	F/U CT
37	4040	70	M	CPS	MU	Y	Y		Y			25.5mm	TUB.					AE +ATT	(k/c/o TB) + LM + F/U CT
38	4141	60	F	CPS	SO		Y	Y				43x37 mm	METS					Radiation therapy	k/c/o ca of breast
39	4242	8	F	SPS	SO	Y			Y			21 x 17mm	TUB					AE +ATT	CSF + F/U CT
40	4343	33	M	SPS	MU	Y	Y	Y	Y			23x18mm	TUB.					AE +ATT	MRI + CSF + F/U CT
41	4545	20	M	SPS	SO	Y		Y				10mm	NCC		Y			AE + ALB	F/U CT
42	4646	65	M	SPS	MU	Y			Y		Y	9 mm	NCC		Y			AE + ALB	F/U CT
43	4747	4	M	SPS	SO	Y			Y			14 mm	TUB					AE +ATT	(k/c/o TB) + F/U CT
44	4848	60	M	PS with SG	SO		Y					25 x 15 mm	METS					Radiation therapy	MRI + k/c/o ca of left kidney
45	4949	23	M	SPS	SO		Y	Y				6 mm	NCC					AE + ALB	F/U CT
46	5050	65	F	CPS	MU	Y	Y	Y	Y	Y	Y	23 x 21mm	METS					Radiation therapy	F/U CT
47	5151	62	M	SPS	MU	Y	Y	Y	Y		Y	11x6 mm	NCC	Y	Y	Y	Y	AE + ALB	F/U CT
48	5252	13	M	SPS	SO		Y	Y				12 mm	NCC		Y	Y		AE + ALB	F/U CT
49	5353	15	F	SPS	SO		Y	Y					DVA					surgery	F/U CT
50	5454	9	M	SPS	SO	Y				Y		8mm	NCC		Y	Y	Y	AE + ALB	F/U CT

51	5656	72	F	PS with SG	SO		Y		Y		Y	63 x 50 mm	Epidermoid cyst with parafalcine mengioma					Surgery	Histopathalogy + MRI
52	5757	3	F	SPS	SO	Y					Y	12 mm	NCC		Y			AE + ALB	F/U CT
53	5858	8	F	SPS	SO	Y			Y			24x16mm	TUB					AE +ATT	LM + F/U CT
54	5959	17	M	SPS	SO	Y		Y				13x4 mm	PC					AE	
55	6060	2	M	PS with SG	0							4.8 x 3.8	Medullobla stoma					Surgery	Histopathalogy + MRI
56	6161	77	M	SPS	SO		Y		Y			15x8 mm	PC					AE	
57	6262	70	M	CPS	SO							24x19mm	arachnoid cyst.					AE	MRI
58	6363	40	F	SPS	SO	Y		Y					chronic SDH post traumatic					AE	
59	6464	65	M	SPS	SO	Y			Y			14x9mm	TUB					AE + ATT	LM + F/U CT
60	6565	55	F	PS with SG	SO							5.4 x 4.4	PA					Surgery	Histopathalogy + MRI
61	6767	22	M	SPS	SO							4 mm	Colloid cyst					AE	
62	6868	50	M	SPS	MU		Y		Y	Y		41 x 31 mm	METS					Radiation therapy	k/c/o ca of lung
63	6969	21	M	SPS	MU	Y	Y	Y	Y			24 x 14mm	NCC	Y	Y	Y	Y	AE + ALB	F/U CT
64	7070	28	F	SPS	SO	Y		Y				15 x 8 mm	PC					AE	
65	7171	32	M	SPS	MU	Y	Y	Y			Y	11 mm	NCC				Y	AE + ALB	F/U CT