CLINICAL PROFILE OF PARTIAL SEIZURES

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INTRODUCTION

Epileptic seizures have been recognized for millennia. As early as 400B.C, when Hippocrates wrote about epilepsy, till the present day when medical sciences have reached the height of sophistication and various investigations like Computed Tomography, Magnetic Resonance Imaging and Positron Emission Tomography(PET), Epilepsy a common neurological disorder remains an enigma.

Modern investigation of the etiology of epilepsy began with the work of Fritsch, Hitzig, Ferrier, and Caton in the 1870s. They recorded and evoked epileptic seizures in the cerebral cortex of animals. In 1929, Berger discovered that electrical brain signals could be recorded from the human head by using scalp electrodes; this discovery led to the use of electroencephalography (EEG) to study and classify epileptic seizures. Gibbs, Lennox, Penfield, and Jasper further advanced the understanding of epilepsy and developed the system of the 2 major classes of epileptic seizures currently used.

The Physician's approach to a patient with epilepsy necessitates that he or she should first define the nature or type of the patients seizures. Then if possible, determine the site in the brain from which they are arising and finally specify the nature of the underlying pathology or

pathophysiology. This idea which cannot always be achieved in full, is particularly applicable to patients with focal epilepsy.

In the past, a large number of cases were labeled as epilepsy of unknown origin. Advances in imaging have revolutionized the ability to visualize the lesions in the brain that cause neurologic dysfunction. The introduction of computed tomographic scan [CT scan] particularly, has really helped to sort out the causes of epilepsy. It easily picks up focal calcification, mass lesions, vascular lesions, abscesses, ring or disc enhancing lesions and other various innumerable abnormalities. Thus, the CT scan, one of the modern noninvasive investigation is a valuable tool in detecting intracranial abnormalities in patients with focal seizures.

AIM OF THE STUDY

1. To study the age and sex distribution of partial seizures,
2. To study the etiological factors responsible for partial seizures,
3. To study the value of neuroimaging of the brain in the diagnosis of the etiology of partial seizures.

REVIEW OF LITERATURE

The ability to identify the site of origin of a seizure on the basis of clinical manifestations began with pioneering efforts of John Huglings Jackson in the later 19th century which expanded with surgical studies conducted by Penfield and colleagues in the middle of the 20th century. It has further expanded in the last two decades with the increased availability of long term video electroencephalographic recording. Jackson, an eminent British neurologist, defined epilepsy in the year 1837 as an intermittent derangement of the various systems presumably due to a sudden, excessive, disorderly discharge of cerebral neurons. Modern electrophysiology offers no evidence to the contrary. Seizures are produced by an abnormal discharge from the cortical neurons resulting in stereotyped movements of the body, abnormal sensory perceptions or behaviour. Epilepsy is the condition of recurrent seizures caused by an inherent abnormality of the brain.

Lord Russell gave a more detailed definition. According to him, "An epileptic seizure is defined as an intermittent stereotyped disturbance of consciousness, behavior, emotion, motor function or sensation that on clinical grounds is believed to result from cortical neuronal discharge".

The basic physiological nature of epilepsy is expressed in Electroencephalographic terms [EEG]. The spikes and sharp waves are

the EEG hallmarks of inter-ictal recordings in patients with epilepsy. These are due to hypersynchronisation of electrical activity within an abnormal pool of neurons.

Epilepsy is a symptom of many diseases and a genetic predisposition may produce seizures in many individuals and not in others. Also lesions of brain in Rolandic area and the anterior temporal lobe appear to have lower threshold for seizures. It remains undecided whether all seizures share a common mechanism in which threshold vary from person to person and in the same person from time to time.

Modern view of epileptogenesis concerns changes in neurotransmitter level as well as complex interactions within neurons at receptor sites on cell membrane. The superior region of the hippocampus is one of the most seizure prone area of the brain.

Hippocampal studies demonstrate that generation of the epileptogenic discharge depends on interplay between three factors.

1. The inherent capacity of certain normal neurons to elaborate active responses, leading to sustained depolarization and paroxysmal bursting.
2. The breakdown of normal inhibitory mechanisms and the augmentation of excitatory synaptic mechanism, thereby

facilitating synchronous neuronal interaction. Post synaptic GABA mediated inhibition is suppressed and recurrent synaptic inhibition is sufficiently reduced to allow synchronization to develop.

3. The effects of modulation by neuro-transmitter substance helps to trigger and maintain epileptic discharge. Acetyl choline mediates neuronal excitability and promotes transmissions from an inter-ictal to an ictal state. Acetyl choline produces initial hyperpolarisation followed by prolonged depolarization. There is decreased conductance of sodium and calcium currents so that any excitation to the affected neuron is favoured.

In a chronic epileptogenic focus,

1. Manifestations of excitability are subtle.
2. There is high incidence of non-bursting cells.
3. The cellular voltages are low and shorter duration.
4. Cellular interaction shows less synchrony.
5. Cell bursts correlate with surface spikes 50% of them.
6. A large number of neurons are only partially or selectively affected; there are external inputs from the brainstem, thalamus etc, which are constantly charging.
7. There may be selective ions of GABAergic inter-neurons which leads to loss of effective post-synaptic inhibition.

8. Changes in neurological morphology could affect the density of different channels, disturbing the balance between excitatory and inhibitory mechanisms.
9. Some ill defined genetic factors may play a role.

Chronic foci show changes in neuronal morphology – loss of dendrites, simplification of the arborisation pattern, shrinking of the entire neuron, neuronal drop out and gliosis.

As epileptic discharges appear, either when inhibition is decreased or excitation is increased, recent research has focused in the role of inhibitory neuro-transmitters like GABA and glutamate receptors. This may explain the heterogeneity of seizures and the differences in their response to treatment.

A particular type of receptor for the excitatory amino acids has been studied intensively the N- methyl- D-asparatate (NMDA) receptors is expressed throughout the brain with the highest density in the hippocampus. The NMDA receptors mediate slow excitatory post synaptic potentials (EPSP) which rise in time of order of 20-25 milliseconds and decays of 200-300 msec. These receptors are permeable to calcium but can change the generation of inter-ictal epileptiform discharge. NMDA receptor antagonists have been shown

to have anticonvulsant properties and are being studied for use as antiepileptic drugs.

GABAergic neurons are preferentially lost at epileptic foci. While it appear certain that GABA plays an inhibitory role in epileptogenesis, its precise mode of action remains unclear.

Electrodes placed on the scalp may pick up high voltage, inter-ictal spike discharges; which are a characteristic feature of epilepsy. Intracellular micro-electrodes measures the synaptic potentials that occur synchronously in several cells. These corresponds to the inter-ictal spikes recorded in the scalp and are called Paroxysmal Depolarization Shifts (PDS) in resting membrane potential. The PDS are the basic building blocks from which the epileptogenic foci develop.

Neuronal membrane contains “voltage-gated channels”, primarily responsible to the electrical field changes at the membrane and “transmitter regulated channels” regulated by neurotransmitter receptors. Both voltage - regulated and receptor-regulated channels are involved in the production of PDS. GABA is the major inhibitory neuro-transmitter in the CNS and is present in 30% of all synapses. Most GABAergic neurons form local inhibitory loops.

NATURE OF THE DISCHARGING LESION

Seizure discharge can be initiated in an entirely normal cerebral cortex, as happens when the cortex is activated by administration of drugs; withdrawal of alcohol or other sedative or by repeated stimulation by sub convulsive pulses [kindling phenomenon]. The electrical properties of a cortical epileptogenic focus suggest that its neurons have an increased ionic permeability that renders them susceptible to activation by hypothermia, hypoxia, hypoglycemia, hypocalcaemia, hyponatraemia, repeated sensory stimulation and during some phases of sleep. Epileptic foci are characterized by spontaneous inter-ictal discharges during which neurons of the discharging focus exhibit large calcium mediated PDS followed by prolonged After Hyper Polarisation (AHP). The AHP are due to calcium dependent positive currents, better explained by enhanced synaptic inhibition. The PDS summate to produce surface recorded inter-ictal EEG spikes. The AHP corresponds to slow wave of EEG spike and wave complex.

The neurons surrounding the epileptogenic focus are hyperpolarized from the beginning and have GABAergic inhibitory neurons within the focus. Seizure spread depends on any factor or agent that activates the neurons within the focus or inhibits the surrounding neurons.

The level of extracellular potassium is elevated in glial scars near epileptic foci and a defect in voltage sensitive calcium channels has been postulated . Deficiency of neuro-transmitter GABA, increased glycine, decreased taurine, and increased glutamic acid etc, have been reported in human epileptogenic tissues.

Firing of epileptogenic neurons in cortical focus is reflected in EEG as a series of spike wave discharges that increase progressively in amplitude and frequency. Once the intensity of the seizure discharge exceeds certain limit it overcomes the inhibitory influences of the surrounding neurons and spreads to neighbouring cortical and subcortical synaptic connections.

If unchecked, the cortical impulse spreads to the adjacent cortex and to the lateral cortex via the inter-hemisphere pathways. The first clinical manifestation depends on that part of the brain from which the seizure originates. There is propagation to sub-cortical nuclei and spinal neurons via the corticospinal and recticulospinal pathways. The spread of excitation to the sub-cortical and brainstem centers correspond to the Tonic phase of the seizure, loss of consciousness and signs of autonomic overactivity. Soon after the spread of excitation, the diencephalon-cortical inhibition begins and intermittently interrupts the seizure discharge, changing it from tonic to clonic phase. The

intermittent clonic burst become less and less frequent and eventually ceases completely leaving in their wave exhausted neurons in the epileptogenic-focus and increased permeability of the blood brain barrier.

These changes form the basis of Todds post epileptic paralysis. There is 2-3 fold increase in the glucose utilization of neurons during seizure discharge and the paralysis could be due to neuronal depletion of glucose and accumulation of lactate.

The spike and wave complex which represents brief excitation followed by slow wave inhibition is the type of pattern that characterizes the clonic (inhibitory) phase of focal motor or Grandmal seizures. Temporal lobe seizures arise in the medial temporal lobe, amygdaloid nuclei and hippocampus. They may also arise in the convexity of the temporal lobe. Loss of memory for events of the episode is due to the paralytic effect of the neuronal discharge in the hippocampus. The seizure focus may establish a persistent secondary focus in the corresponding control area of the opposite hemisphere via cortical connections called mirror focus. The mirror form is the source of confusion in trying to identify the side of the primary lesion by EEG.

Severe seizures may cause systemic lactic acidosis, fall in arterial pH, reduction in arterial O_2 saturation and rise in pCO_2 which are secondary to

respiratory spasm and excessive muscular activity. Heart rate, blood pressure and CSF pressure rise during seizure.

NEUROIMAGING

A dramatic increase in the role of imaging in diagnosis of neurologic diseases occurred with the development of computed tomography (CT) in the early 1970s and of magnetic resonance imaging (MRI) in the 1980s. MRI has gradually replaced CT for many indications and has also reduced the indications for invasive neuroimaging techniques, such as myelography and angiography. In general, MRI is more sensitive than CT for the evaluation of most lesions affecting the central nervous system, particularly those in the spinal cord, cranial nerves, and posterior fossa. CT is more sensitive than MRI for visualizing fine osseous detail, such as temporal bone anatomy and fractures. Recent developments, such as helical CT, CT angiography (CTA), MR angiography (MRA), positron emission tomography (PET), Doppler ultrasound, and interventional angiography have continued to advance diagnosis and guide therapy.[97]

COMPUTED TOMOGRAPHY

Technique: The CT image is a computer-generated cross-sectional representation of anatomy created by analysis of the attenuation of xray beams passed through various points around a section of the body. As the x ray source, collimated to the desired slice thickness, rotates around the

patient, sensitive x-ray detectors aligned 180º from the source detect x-rays attenuated by the patients anatomy. A computer calculates a "back projection" image from the 360º x-ray attenuation profile. Greater x-ray attenuation, as caused by bone, results in areas of high "density," while soft tissue structures, which attenuate x-rays less, are lower in density. The resolution of an image depends on the radiation dose, the collimation (slice thickness), the field of view, and the matrix size of the display. A typical modern CT scanner is capable of obtaining sections 1 to 2, 5, and 10 mm thick at a speed of 1 to 3 s per section; complete studies of the brain can be completed in <2 to 3 minutes.[94]

Intravenous contrast is often administered prior to or during a CT study to identify vascular structures and to detect defects in the blood-brain barrier (BBB) associated with disorders such as tumors, infarcts, and infections. An intact BBB prevents contrast molecules, which are large, from exiting the intravascular compartment. In the central nervous system, only vessels and those structures not having a BBB enhance [95]. The use of contrast agents carries a risk of allergic reaction, increases the dose of radiation when both noncontrast and contrast CT scan are to be obtained, adds expense, and may mask hemorrhage; thus before contrast is administered, the indication for its use should always be considered carefully.

Helical CT is a new technique in which continuous three dimensional CT information is obtained. In the helical scan mode, the table moves continuously through the rotating x-ray beam, generating a “helix” of information that can be reformatted into various slice thicknesses. Advantages include shorter scan times, reduced patient and organ motion, and the ability to acquire images during the infusion of intravenous contrast.[96] The contrast images can be used to construct CT angiograms of vascular structures. CTA images require a workstation to threshold and segment CT images for display.

Magnetic Resonance Imaging [MRI] remains the imaging modality of choice when structural or anatomic abnormalities are suspected.[1,2] Sequences of particular importance include T2- weighted and fluid attenuated inversion recovery (FLAIR) images and gadolinium-enhanced T1-weighted images in cases, such as tumors, vascular abnormalities, infectious or inflammatory nidi, and cortical dysplasia.[1-4] Although 1.5T MRI is widely available, the use of 3.0T MRI produces images with improved signal-to-noise ratios[5] which can help localize structural abnormalities that may underlie epileptogenesis.

Although 3T MRI may only identify brain abnormalities 25% of previously normal 1.5 T scans, the addition of surface coils can increase this rate to 65%. The improved detection rate is critical, because detection of an

imaging abnormality dramatically increases post surgical freedom from seizures.[6,7] MRI has been shown to be particularly sensitive in identifying the structural abnormalities related to mesial temporal sclerosis(MTS).[8,9] In 80% to 90% of mesial temporal sclerosis cases, MRI allows the detection of T2 and FLAIR hyperintensity in the mesial temporal structures whereas coronal images can allow volumetric comparision of the 2 hippocampi and the demonstration of unilateral hippocampal atrophy.

Positron Emission Tomography (PET) is a neuroimaging modality that involves the metabolic use of a radio active substrate as an index of brain metabolic changes that may be coupled to seizures.[10,11] The most commonly used tracer is [^{18}F]fluorodeoxyglucose (FDG). FDG-PET has been particularly useful in case of temporal lobe epilepsy in which it has a sensitivity of approximately 90%.[12] Typically interictal PET identifies focal areas of hypo metabolism. In a recent study by Theodore et al, presence of glucose hypometabolism was found in the left temporal lobe of 70-80% of patients with temporal epilepsy, who have undergone surgery.

Single photon emission computed tomography (SPECT) represents a functional imaging modality believed to represent cerebral perfusion. The substrates that are imaged are usually ^{99}mTC labled molecules. They are rapidly taken up by brain tissue within less than 1 minute after intravenous

injection, and remain trapped within brain tissue for upto 4 hours. Because of such kinetics SPECT can be used to acquire an ictal profile.[13]

Subtraction ictal SPECT coregistered to MRI(SISCOM) refers to a combined imaging modality in which the interictal SPECT is subtracted from the ictal SPECT and the subtraction image is merged with an MRI to anatomically define the area with the perfusion abnormality.[14,15] SISCOM was shown to be superior to ictal and interictal SPECT in localizing epileptogenic foci and predicting the outcome of epilepsy surgery. In the case of temporal lobe epilepsy, SISCOM was shown to have a localization sensitivity of 97%.[16]

Even in affluent countries these investigative modalities are still used as research tools only. CT scan can be used as the primary imaging modality in any case of partial seizures. If diagnostic dilemma remains or if surgical treatment is anticipated, MRI and other imaging studies may be advised. CT scan has better resolution for calcified lesions than MRI. Cerebral calcification can be due to metabolic, neoplastic, vascular, congenital, infections or non-infectious inflammatory causes. Metabolic processes like hyperparathyroidism and other abnormalities of calcium and phosphorus metabolism can cause bilateral basal ganglia calcification, but these calcified foci do not themselves produce seizures.

Tumors that are found during surgery for chronic epilepsy often lack radiological characteristics like edema mass effect and contrast enhancement that are usually typical of tumors. Examples are low grade astrocytomas, oligodentrogliomas, gangliomas. Calcification can be a manifestation of any of these tumors, especially on CT scans, but on MRI they usually appear as heterogeneous masses rather than a uniform focus of calcification.

Vascular lesions that may give rise to epilepsy include infarcts, primary intracerebral haemorrhages and congenital vascular anomalies. The last category comprises arteriovenous malformations, venous angiomas, capillary telangiectasias and cavernous angiomas. Arteriovenous malformations consists of anomalous vessels that have the characteristics of both arteries and veins with intervening parenchymal tissue and that are visible on MRI or conventional angiography; MRI shows characteristic multiple flow voids.

Cysticercosis is the other cerebral infection resulting in cerebral calcification and recurrent seizures. Recent literature suggest that cysticercosis may be the most common cause of symptomatic epilepsy in the world.[17,18,19] Cerebral lesions usually evolve from active forms appear as thin walled fluid filled cysts with mural nodule (the line scolex); it causes no inflammatory reaction. The transitional form is a more proteinaceous encapsulated cyst with ring enhancement. The cyst becomes a

granulomatous, irregularly enhancing lesion as the organism dies. The inactive lesion contains the dead organism and is usually calcified with no enhancement.

At any of these stages there may be multiple lesions, most often the lesions are solitary. Seizures are the most common clinical manifestation at all stages of intraparenchymal infection, although focal headaches and focal symptoms are common during the active and transitional stages.

An international classification of epileptic syndromes was proposed in 1981. The classification of seizures is constantly being modified. In the latest version (epilepsia 30:389, 1989); an attempt has been made to incorporate all of the epilepsies, epileptic syndrome and related seizure disorders and to categorize them not only as partial or generalized; but also according to the age of onset, primary or secondary nature of the seizure and the clinical settings in which they occur (Adapted from commission on the terminology and classification of International league against epilepsy. Epilepsia: 26,268-275 / 1985)

Classification of seizures and epilepsy syndromes

1. Localization related epilepsies and syndromes
    a. Idiopathic with age related onset
        i. Benign childhood epilepsy with temporal spikes

ii. Childhood epilepsy with occipital paroxysms

b. Symptomatic

Related to the area of onset and clinical EEG features (This encompasses most partial seizures).

2. Generalised epilepsies and syndromes

a. Idiopathic with age related onset in order of age

i. Benign familial neonatal convulsions

ii. Benign neonatal convulsions

iii. Benign myoclonic epilepsy in childhood

iv. Childhood absence epilepsy

v. Juvenile absence

vi. Juvenile myoclonic epilepsy (impulsive petitmal)

vii. Epilepsy with grandmal Seizures (GTCS)

b. Idiopathic and/Symptomatic in order of age

i. West syndrome

ii. Lennox Gestaut syndrome

iii. Epilepsy with myoclonic seizure

iv. Epilepsy with myoclonic absences

c. Symptomatic epileptic seizures as part of a clinical picture

AV malformation or degenerative diseases with or without metabolic etiology that present with seizures as part of the clinical picture.

3. Epilepsies and syndromes undetermined as to whether they are localized or generalized.

a. Both generalized and focal seizures

i. Neonatal seizures

ii. Severe myoclonic epilepsy in infancy

iii. Epilepsy with continuous spike waves during slow wave sleep.

iv. Acquired epileptic aphasia (Landau-Kleffner Syndrome)

b. Without unequivocal generalized or focal features

GTCS in which a focal or generalized onset cannot be determined by clinical or EEG features.

Common seizure patterns and their Localization

CLINICAL TYPE	LOCALIZATION
Somatic motor	
Jacksonian (focal motor)	Prerolandic gyrus
Masticatory, salivation, speech arrest	Amygdaloid nuclei, opercular
Simple contraversive	Frontal
Head and eye turning associated with arm movement or athetoid dystonic postures	Supplementary motor cortex
Somatic and special sensory (auras)	
Somatosensory	Contralateral postrolandic
Unformed images, lights, Patterns	Occipital
Auditory	Heschl's gyri
Vertiginous	Superior temporal
Olfactory	Mesial temporal
Gustatory	Insula
Visceral: autonomic	Insular-orbital-frontal cortex
Complex partial seizures	
Formed hallucinations	Temporal neocortex or amygdaloid-hippocampal complex

Illusions	-
Dyscognitive experiences (de´ja` vu, dreamy states, depersonalization)	-
Affective states (fear, depression, or elation)	Temporal
Automatism (ictal and postictal)	Temporal and frontal
Absence	Frontal cortex, amygdaloid Hippocampal complex, reticular-cortical system

SOURCE: Modified by permission from Penfield and Jasper.

Classification of partial seizures

A. Simple partial seizures (consciousness not impaired)
    1. With Motor signs
        a. Focal motor without march
        b. Focal motor with march (Jacksonian)
        c. Versive
        d. Postural
        e. Phonatory (vocalization or arrest of speech)
    2. With somato sensory or special sensory signs simple hallucination
        a. Somatosensory

b. Visual

c. Auditory

d. Olfactory

e. Gustatory

f. Vertiginous

3. With autonomic symptoms or signs (including epigastric sensation, pallor, sweating, flushing, piloerection and pupillary dilation)
4. With psychic symptoms (disturbance of higher cerebral functions). These symptoms rarely occur without impairment of consciousness and are more commonly experienced as complex partial seizures.

   a. Dysphasic

   b. Dynamic(e.g. déjà vu)

   c. Cognitive (dreamy states, distortion of time sense)

   d. Affective (fear, anger etc)

   e. Illusions (e.g. macropsia)

   f. Structural hallucinations (e.g. music, scenes)

B. Complex partial seizures (with impairment of consciousness; may sometime begin with simple symptomatology).

1. Simple partial onset followed by impairment of consciousness.

   a. With simple partial seizures (A1- A4) followed by impaired consciousness.

b. With automatism

2. With impairment of consciousness at onset

a. With impairment of consciousness at onset

b. With automatism

C. Partial seizures evolving to secondarily generalized seizures (may be generalized tonic – clonic, tonic or clonic).

1. Simple partial seizures (A) evolving to generalized seizures
2. Complex partial seizures (B) evolving to generalized seizures
3. Simple partial seizures (A) evolving to complex partial seizures and then to generalized seizures.

Classification of generalized seizures

A. Absence Seizures

1. Typical absences

a. Impairment of consciousness only

b. With mild clonic movements

c. With atonic components

d. With tonic components

e. With automatisms

f. With autonomic components (b through f may be seen alone or in combination).

2. Atypical absences may have
    a. changes of tone that are more pronounced than in A1
    b. onset and/or cessation that are abrupt

B. Myoclonic seizures

C. Clonic seizures

D. Tonic seizures

E. Tonic clonic seizures

F. Atonic seizures (Astatic seizures)

Combinations of these may also occur

E.g. B & D, B & F, etc…

Classification of status epilepticus

1. Convulsive status
    A. Primary generalized major motor status
        i. Tonic – clonic status
        ii. Myoclonic status
    B. Partial or focal status
        i. Focal motor status (Jacksonian type)
        ii. Epilepsia partialis continua
    C. Generalised major motor status with partial onset
        i. Adversive (head, eyes or body onset)
        ii. Partial motor onset

2. Non – Convulsive status

    A. Petitmal status

    B. Psychomotor status or complex partial status

Risk Factors

Several studies have classified risk factors and prognosis for Generalized Tonic Clonic Seizures(GTCS), Absence and Complex partial seizures. History of maternal convulsions, fever etc. are risk factors for GTCS. History of febrile seizures was the risk factor for absence seizures. Past history of febrile seizures or neonatal convulsions, cerebral palsy, head trauma and viral encephalitis were significantly more in patients with complex partial seizures than in control subjects.

Risk factors that were significant included twin pregnancy, breech presentation and perinatal asphyxia for complex partial seizures. Epidemiological studies indicate that parents and sibling of probands have more epilepsy than predicted population rates.

While about 4% of the population may have seizure of some type by the age of 20 yrs, the rate for siblings and offspring of probands is mere 10%. The risk of recurrence of seizure is greatest in the first week after the initial seizure. 70% of the patients seen on the day of the first seizure have a second seizure by the second day; whereas for patients seen eight weeks

after their first seizure, the risk of recurrence within the next three years is 22%.

Ammon horn sclerosis or hippocampal sclerosis has been found in 50-60 % of chronic epileptic patients. This abnormality, consisting of severe neuronal loss and gliosis, tends to affect selective regions of the hippocampus. In these cases atrophy is most obvious to the naked eyes. These pathological changes are unilateral in 80% of the patients. When sclerosis involves the amygdala, the uncus and spreads laterally to the parahippocampal gyrus and other parts of the temporal lobe, it is called Mesial Temporal Sclerosis.

Mesial Temporal Sclerosis is found in nearly 50% of the patients who underwent temporal lobectomy for intractable complex partial seizures. Changes have been demonstrated in the human brain after status epilepticus. Repeated seizures lead to ischemic cell damage in the hippocampal neocortex and cerebellum. Pathological changes are evident when seizures last for more than 90minutes.

Tuberculoma

Tuberculomas of the brain account for 20-30% of intracranial tumors in India. In paediatric age group, 41% of intracranial space occupying lesion have been found to be tuberculous in nature. Tuberculomas develop in the

brain when the “Rich focus” does not rupture into the meninges but expands locally in the brain parenchyma. Tuberculoma may also originate from the meninges and superficial cortex. The meningeal form resembles a meningioma. For reasons that are unclear the majority are supratentorial in adults while infratentorial granulomata predominate in children. In most patients lesions are solitary but in 15 to 34% of cases multiple tuberculomata are found. (De Angelis 1981[20])

Tuberculoma has a creamish, white, gritty, caseating centre, surrounded by a firm to hard greyish rim of granulomatous tissue. Histologically, the central necrotic zone is surrounded by tuberculous granulation tissue containing epithelioid cells, lymphocytes and Langhans giant cells. Surrounding the tuberculoma there may be arteritis, neuronal damage and edema of varying degrees of severity. As a tuberculoma regresses there is increasing collagen formation, sometimes associated with the deposition of calcium salts.

Tubercle bacilli can be found in the majority of surgical or autopsy specimen, but in their absence the pathological diagnosis can be made on the histological appearance of the granuloma alone.[21] Intracranial tuberculomas occur mainly in adults in Western countries whilst in India, children are mostly affected (De Angelis, 1981).[20] Most intracranial tuberculomas present

with symptoms and signs of a space occupying lesion and only a minority develop tuberculous meningitis in the course of illness.

Compared to other intracranial mass lesions, the incidence of convulsions is particularly high and in some series they occur in as many as 85% of cases (Arseni 1958).[24] Fever and general ill health are usual findings but a past history of tuberculosis or evidence of active infection outside the nervous system occurs in about 50% of the patients. 60-70% of intracranial tuberculoma present with seizures. 56-93% have features of increased intracranial tension, 33-68% have focal neurological deficit. Gulati et al[22] found the commonest cause of focal seizures is tuberculoma. 63-73% of patients have single confluent large granuloma with necrotic centre. Tuberculomas may also be multiple.[23,98]

Tuberculomas are avascular when studied angiographically, its appearance on CT and MRI varies. Initial phase of the disease – edema, necrosis may appear as low attenuating areas on CT scan. Once the granuloma starts to organize there may be high attenuation, contrast enhancement and calcification as ring enhancement with variable degree of surrounding edema. The enhancement may be homogenous or there may be a central radioluscent area corresponding to the central zone of necrosis.

MRI is more sensitive than CT in detecting tuberculoma. Tuberculomas are isointense with gray matter on T1- weighted MR images.

On T2- weighted images lesions show central hyper intensity. In some cases hypo intense ring is present in the wall of a tuberculoma on T2- weighted images. A collar of high signal resulting from edema on T2- weighted images. In tuberculoma, a central speck of calcification, “target sign” has been considered pathagnomonic. Similar punctate calcification may also be seen in cysticercus granuloma.[25,26]

Presence of increased intracranial tension, focal neurological defecit, along with CT features of irregular margin and midline shift were suggestive of intracranial parenchymatous tuberculoma.[27] There will be evidence of pulmonary tuberculosis in 60% of cases of tuberculoma.[26]

Other causes for localized brain granulomatous lesions:

Brain abscess

Headache is the most common presenting symptom and is observed in 70% of the patients. The location of the brain abscess define the clinical presentation. CT scan reveal central cystic lesion within a well defined enhancing ring lesion with substantial amount of edema. Tuberculous abscess may be clinically and radiologically indistinguishable from a pyogenic abscess.

Syphylitic gumma:

Solitary lesion in brain but this lesion would be unusual without evidence of syphilis elsewhere.

Protozoal disease

Produce focal brain lesions especially amoebiasis, toxoplasmosis. Acquired toxoplasmosis is a disease of immuno- compromised.

Fungal Disease

Cryptococcal neoformans Candida albicans Mucormycosis Aspergillosis	Usually causes chronic meningitis and results in solitary granuloma, multiple parenchymal brain abscess or granuloma

Serological evidence of TB

Based on recognition of serum IgG antibody to mycobacterial antigen and the rise of ELISA. When the diagnosis, is doubtful serological evidence of TB may prove useful. A positive test by ELISA technique can be taken as a supportive evidence of intracranial tuberculoma.[28] Major limiting factor for the serological test is the cost.

TB elsewhere

If, facilities for serological studies are not available, a reliable diagnosis can be made if there is evidence of TB elsewhere.[29]

Newer Methods

1. Gene amplification by PCR to identify mycobacterial DNA.
2. If the diagnosis of tuberculoma is doubtful a trial of ATT may be instituted without histological confirmation.
3. Improvement in clinical and radiological features may provide a valuable evidence for diagnosis of lesions.
4. Response to ATT may not be rewarding due to the paradoxical enlargement of the lesion when started on ATT.

CEREBROVASCULAR DISEASE

In population studies, stroke is the most commonly identified cause of epilepsy in adult populations older than 35 years.[72] Seizures may be a more common accompaniment of hemorrhagic rather than ischemic stroke. Bladin et al[73] found the incidence of seizures to be 10.6% among 265 patients with intracerebral hemorrhage vs 8.6% among 1632 with ischemic stroke. In another prospective series[75] seizures occurred in 4.4% of 1000 patients, including 15.4% with lobar or extensive intracerebral hemorrhage, 8.5% with subarachnoid hemorrhage, 6.5% with cortical infarction, and 3.7% with hemispheric transient ischemic attacks.

Seizures after stroke are classified as early or late onset, according to their timing after brain ischemia. An arbitrary cut point of 2 weeks after the presenting stroke has been recognized to distinguish between early- and late-

onset poststroke seizures.[76-78] Early seizures are thought to result from acute biochemical disturbances and may result in part from the damaging effects of the excitatory neurotransmitter glutamate in response to ischemia. In contrast, late seizures are attributed to gliosis and cortical scarring with resulting selective neuronal loss and hyperexcitability of the surrounding tissue.

In patients with ischemic stroke, epilepsy developed in 35% of patients with early-onset seizures and in 90% of patients with late-onset seizures.[80] The risk for epilepsy was comparable in patients with hemorrhagic stroke; epilepsy developed in 29% of patients with early-onset seizures vs 93% with late-onset seizures.[76]

Cortical location is among the most reliable risk factors for poststroke seizures.[73] Poststroke seizures were more likely to develop in patients with larger lesions involving multiple lobes of the brain than in those with single lobar involvement.[82]

Analogous to cortical involvement in ischemic stroke, a lobar site is considered to be the most epileptogenic location in patients with intracerebral hemorrhage. In a series of 123 patients,[83] seizure incidence was highest with bleeding into lobar cortical structures (54%), low with basal ganglionic hemorrhage (19%), and absent with thalamic hemorrhage.

Hemorrhage due to cerebral venous thrombosis also commonly presents with seizures. Parenchymal, often cortical, hemorrhage resulting from local venous congestion is the likely cause of seizure activity. Seizures due to arteriovenous malformations and aneurysms typically occur when these lesions rupture, but these vascular lesions may cause seizures by directly irritating adjacent brain parenchyma.

Finally, seizures associated with vascular lesions occur in the setting of significant reperfusion after revascularization procedures, most commonly carotid endarterectomy

In a study of early-onset seizures in 90 patients,[81] simple partial seizures were the most common type (61%), followed by secondarily generalized seizures (28%). In other series,[74] [79] early-onset seizures were more likely to be partial, whereas late-onset seizures were more likely to generalize secondarily. Poststroke seizures usually respond well to a single antiepileptic drug.

NEUROCYSTICERCOSIS

Neurocysticercosis (NCC) is an infection of the brain and its coverings by the larval stage of the tapeworm Taenia-solium. It is the most common helminthic infestation of the central nervous system (CNS) and a leading cause of acquired epilepsy worldwide.[40] [41] [42] NCC results from ingestion of

the eggs of T. solium. The oncospheres hatch in the intestine, penetrate the intestinal wall and disseminate to several body tissues, showing strong tropism to the CNS. Seizures are the commonest manifestation, occurring in 50% to 80% of patients.

T. solium has a complex life cycle, requiring two hosts .Humans are the definitive host whereas pigs are the intermediate host.[43,44] Humans can also act as intermediate hosts after ingestion of Taenia eggs.

Clinical presentation

Differences in the clinical picture depend on the number, size, stage and localization of cysts and the patient's immune response. Patients commonly present with seizures or headaches.[42] Seizures are the commonest manifestation, occurring in 50% to 80% of patients.[43,44] NCC has been classified depending on the location of cysts, its clinical presentation, prognosis and cyst viability.[48-51]

Parenchymatous form

In the parenchymatous form, the parasite lodges in the brain parenchyma as single or multiple cysts forming clumps. Pericystic inflammation results in a granuloma formation, which is the cause of epilepsy in most patients. In India, the single small enhancing lesion (SSEL) is the commonest form of parenchymatous NCC.[45,46,47] Tuberculous

granuloma, microabscess, meningo-encephalitis, neoplasms & vascular lesions should be considered in differential diagnosis.[52,53] [27] Chandy et al. reported that 12 out of 15 patients with SSELs were shown to have cysticercosis.[54]

In disseminated or miliary NCC, there are multiple cysts in varying stages of development (i.e. living, dying and calcified cysts). There are four stages of development of a parenchymal larval cyst: vesicular, colloidal, nodular and calcified. The vesicular cyst is viable, where the scolex exists as an eccentric nodule within the cyst, and there is little or no enhancement due to a minimal host immune response. As the scolex dies after cysticidal treatment or an effective immune response, the transparent vesicular fluid is replaced by a viscous and turbid fluid, which is readily identifiable on MRI.[55] The fluid migrates from the degenerating cyst into the surrounding parenchyma and incites a strong immune response, characterized by strong enhancement on contrast CT scans or MRI. This is the colloidal cyst, which has contrast enhancement but lacks a well-defined scolex. As the cyst further degenerates, it develops into a nodular cyst, which still shows some contrast enhancement. Finally, the degenerated cyst calcifies and is recognized as a punctuate calcification on CT scan.

Meningeal form

The meningeal form commonly presents with raised intracranial pressure secondary to widespread subarachnoiditis, arachnoiditis and adhesions resulting in cerebrospinal fluid (CSF) obstruction and hydrocephalus.

Intraventricular and subarachnoid (cisternal) forms

The intraventricular and subarachnoid (cisternal) forms of NCC are seen in 15% to 54% of patients and present clinically in a more aggressive manner as compared to the parenchymatous form.[56-58] Patients commonly present with raised intracranial pressure caused by large cyst size or load, occlusion of CSF pathways, associated ependymitis and basal arachnoiditis.[57] Ventricular entrapment may occur secondary to ependymitis and cause double compartment syndrome.[56] The fourth ventricle is the commonest site (53%) followed by the third ventricle (27%), lateral ventricle (11%) and the aqueduct (9%).

Spinal form

The spinal form occurs in 1.6% to 13% of patients with NCC.

i. leptomeningeal (extramedullary)
ii. intramedullary

The leptomeningeal (extramedullary) form is 6 to 8 times more common than the intramedullary form. The parasite commonly lodges in the

thoracic spinal cord according to percentage distribution of blood flow to the spinal cord.[59,60] Del Brutto OH et al.[99] proposed diagnostic criteria for neurocysticercosis.

DIAGNOSTIC CRITERIA

ABSOLUTE CRITERIA

- Demonstration of cysticerci by biopsy
- Visualisation of the parasite in the eye by fundoscopy
- Imaging – cystic lesion showing scolex

MAJOR CRITERIA

- Neuroradiological lesions suggestive of NCC
- EITB – demonstration of antibodies in serum
- Resolution cystic lesion spontaneously or after cysticidal drugs

MINOR CRITERIA

- Lesions compatible with NCC detected by imaging
- Clinical manifestation suggestive of NCC
- Demonstration of antibodies in CSF- ELISA method
- Evidence of cysticerosis outside CNS- cigar shaped soft tissue calcification.

EPIDEMIOLOGIC CRITERIA

- Residence in endemic area
- Frequent travel to endemic area
- House hold contact with an individual infected with T-sodium.

DEGREE OF CERTAINITY

DEFINITIVE DIAGNOSIS

- 1 Absolute criteria
- 2 Major + 1 minor + 1 epidemiologic criteria

PROBABLE DIAGNOSIS

- 1 Major + 2 minor
- 1 Major + 1 minor + 1 epidemiologic criteria
- 3 Minor + 1 epidemiologic

The major drawback of these criteria is that they do not differentiate between NCC and tuberculoma, which is of utmost importance in endemic countries, such as India.[63,67] Moreover, access to enzyme-linked immunoelectrotransfer blot assay (EITB) is limited in India and consequently the usefulness of these criteria has been questioned.[64]

Radiological Investigations

Plain X-ray

Plain x-rays of muscles and the skull may show cigar shaped calcifications.

CT scan of the head

CT scan of the head have a sensitivity and specificity of over 95% in the diagnosis of NCC,[61,62] but it is much lower for diagnosing ventricular or cisternal forms of the disease.

On a CT scan, cysts may appear as single or multiple, rounded lesions of variable size and low density, with a small, hyperdense, eccentric mural nodule representing the scolex. This gives a “starry night” effect in the parenchyma. Ring enhancement occurs with an inflammatory reaction or a granuloma formation. Perilesional edema is seen around dying cysts.

SSELs are the commonest cause of partial seizures in children in India.[63,64,65] Most of these lesions represent solitary cerebral cysticercus granuloma(SCCG). SCCG is the granulonodular form of the parenchymal cyst and accounts for nearly 60% to 70% of all forms of NCC seen in patients in India.[65]

Rajshekhar et al [27] described CT scan criteria for differentiating NCC and tuberculoma. An enhancing ring lesion that is <20 mm in size, regular in outline and not producing a midline shift is likely to be NCC. These criteria

are not absolute and it may be difficult to differentiate a small tuberculoma from NCC on a CT scan alone. MRI has better sensitivity to differentiate tuberculomas from NCC. Preliminary experience with proton magnetic resonance spectroscopy has shown promise in differentiating tuberculoma from NCC.[66]

MRI of the brain

MRI is the most accurate technique to assess degree of infection, location and evolutionary stage of the parasite. The main disadvantages of MRI are its high cost and limited availability. It is possible to differentiate the various cyst stages on MRI.[67]

The intensity of fluid in live cysts is similar to CSF. The scolex appears as a mural nodule of high signal intensity on T1-weighted MRI and of low signal intensity on T2-weighted MRI (like a hole with a dot, or a pea in a pod). There is no perilesional edema at this stage.[34]

In a degenerated cyst, the fluid becomes turbid (colloid vesicular stage), appearing as high intensity on a T1-weighted MRI. In the granulonodular stage, ring enhancement occurs on gadolinium injection (isointense on T1-weighted and hypointense on T2-weighted MRI) and there is variable perilesional edema. However, calcified cysts are better delineated on CT scans.

The racemose type of cyst is a large lobulated cyst without a scolex whereas the cellulose type contains a scolex inside a vesicle. Intraventricular cysts are well delineated with a small metacystode inside the cyst.[35] Abnormal enhancement after gadolinium suggests ependymitis or ventricular entrapment.[36] MRI is useful in follow-up to assess reduction in the number or size of cysts with treatment.

Serology

The available serological tests are of little value in clinical practice. These have a sensitivity of 65% to 98% and a specificity of 67% to 100%, depending on the specific test used, and the cyst burden, location, and phase of infection.[37] [38] The most commonly used enzyme- linked immunosorbent assay is neither sensitive nor specific.[37] EITB is reported to have a sensitivity of 98% and specificity of 100%, and it is the test of choice as a major diagnostic criterion.[38] However, its sensitivity in a patient with a single enhancing or calcified lesion is much lower.[39]

Other tests

Biopsy of brain, skin or muscle for ocular, extraocular muscle or painful muscular or subcutaneous cysts. Dilated ophthalmologic examination is sensitive for the detection of ocular cysts.

Treatment

The treatment modalities available to patients with NCC include:

1. cysticidal agents – to kill larvae
2. corticosteroids – to decrease or prevent the inflammatory reaction
3. anti-epileptic drugs (AEDs) – to prevent or decrease the severity and number of seizures.
4. surgery – to remove the cyst or insert a shunt for hydrocephalus

Tumors

Intracranial tumors are yet another cause of focal seizures. Apart from seizures, they also may present with headache or focal neurologic symptoms reflecting the particular anatomic site of the tumor. Advances in neuroradiology have contributed greatly to the diagnosis and management of patients with neoplastic diseases of the central nervous system. Contrast enhanced CT imaging delineates intracranial masses as small as 0.5 cm in diameter. Tumors commonly appear as homogenous or ring enhancing masses surrounded by variable amounts of edema.

MRI using T2-weighted pulsing techniques appears to be even more sensitive than CT in the detection of focal structural abnormalities of the brain. Nevertheless, MRI has some disadvantages in comparison with CT for assessment of intracranial abnormalities. Cortical bone and intralesional calcifications emit no signal in MR images and thus areas of hyper-ostosis or bone destruction as well as tumor calcification are often not seen on MR images.

MATERIALS AND METHODS

60 patients with history of focal seizures admitted in the medicine, neurology wards, IMCU or attending the neurology op department of the Government Royapettah Hospital during the period of September 2009-2010 were included in the study. Patients with a history of recent head injury were not included.

The study was reviewed and approved by the Institutional Ethical Committee, Kilpauk Medical College and General Hospital, Chennai.

A detailed history (both from the patient and an intelligent attender) regarding the age of onset, number of episodes of seizures, site and side of the seizures and part of the body involved, was recorded in each case. Total duration of the seizure disorder and the average frequency of seizure episode was noted. The time of occurrence of the fits (sleep/awake) relationship with food and lunar cycle were also recorded.

Presence of aura, automatisms, precipitating factors, specific motor, sensory, psychic symptoms, post-ictal symptoms and level of consciousness, were also noted. Presence of amnesia, Jacksonian march and secondary generalization were also sought for important past history like birth hypoxia, developmental disorder, febrile seizures,

head injury, CNS infection, Diabetes Mellitus, Hypertension, Pulmonary Tuberculosis, Ischemic and Valvular Heart disease, CVA, Malignancy, exposure to STD were elicited and recorded. Dietary history was also taken. If non-vegetarian, whether pork eater or not were recorded.

The investigations carried out include complete haemogram, blood sugar, urea and creatinine, serum electrolytes and chest x-ray, and also Mantoux test, sputum AFB, CSF examination and HIV/VDRL in relevant cases.

CT scan of the brain, both plain and contrast enhancement were done in all cases and the results were analysed. In CT scans, structural abnormalities like calcifications, granulomatous lesions, tumors, vascular anomalies, infarcts, haemorrhage, and infections were carefully looked for and the pattern of contrast enhancement were studied. Follow up CT Scans were done in cases where initial scans showed curable lesions like tuberculoma or neurocysticercosis and the changes were noted.

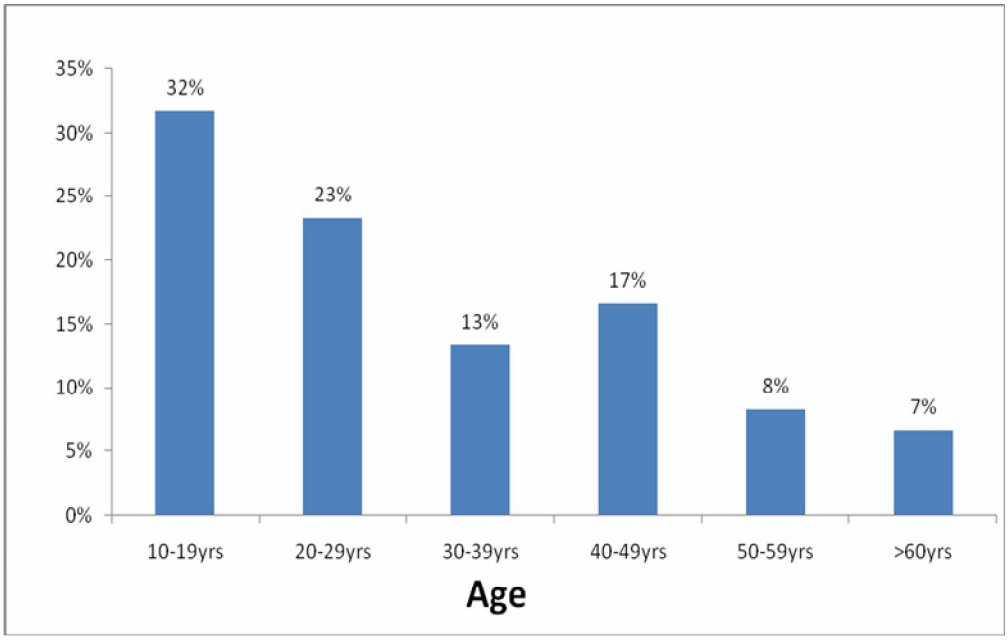
EEG were recorded in patients who had no CT abnormality or obvious metabolic abnormality to account for the seizures.

RESULTS AND OBSERVATION

Age distribution of patients with focal seizures

Table 1

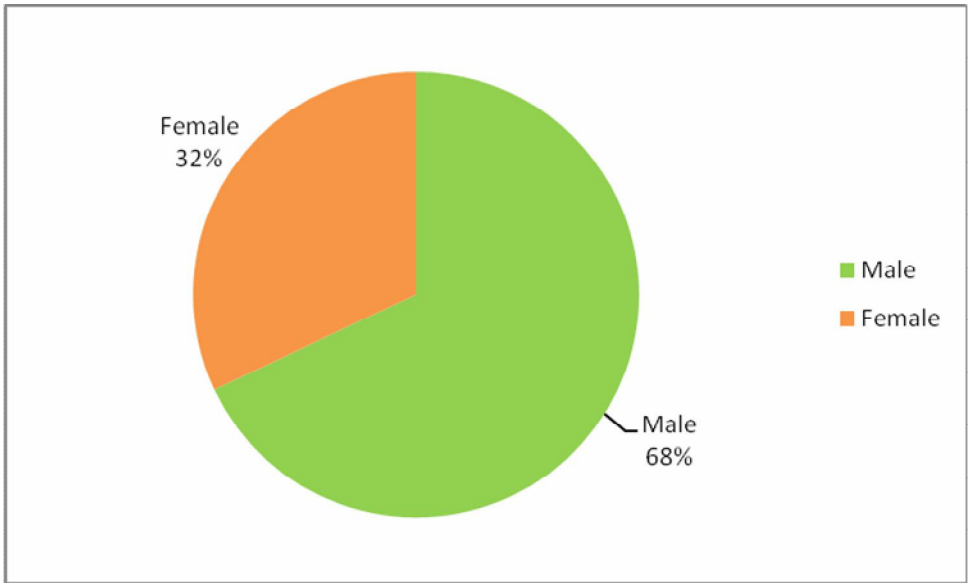
AGE	SPS	CPS	SGS	NO OF CASES	Percentage (%)
10-19yrs	4	8	7	19	32
20-29yrs	3	2	9	14	23
30-39yrs	1	6	1	8	13
40-49yrs	2	3	5	10	17
50-59yrs	0	2	3	5	8
>60yrs	1	1	2	4	7



Sex distribution of patients with focal seizures

Table 2

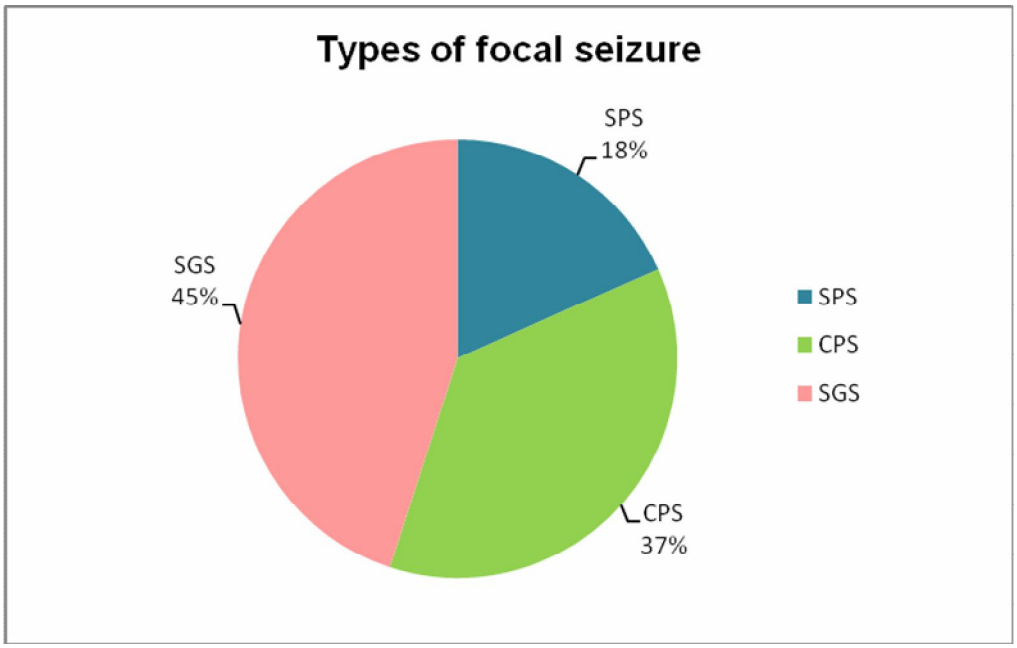
SEX	SPS	CPS	SGS	NO. OF CASES	Percentage (%)
Male	5	16	20	41	68
Female	6	6	7	19	32



Types of focal seizures

Table 3

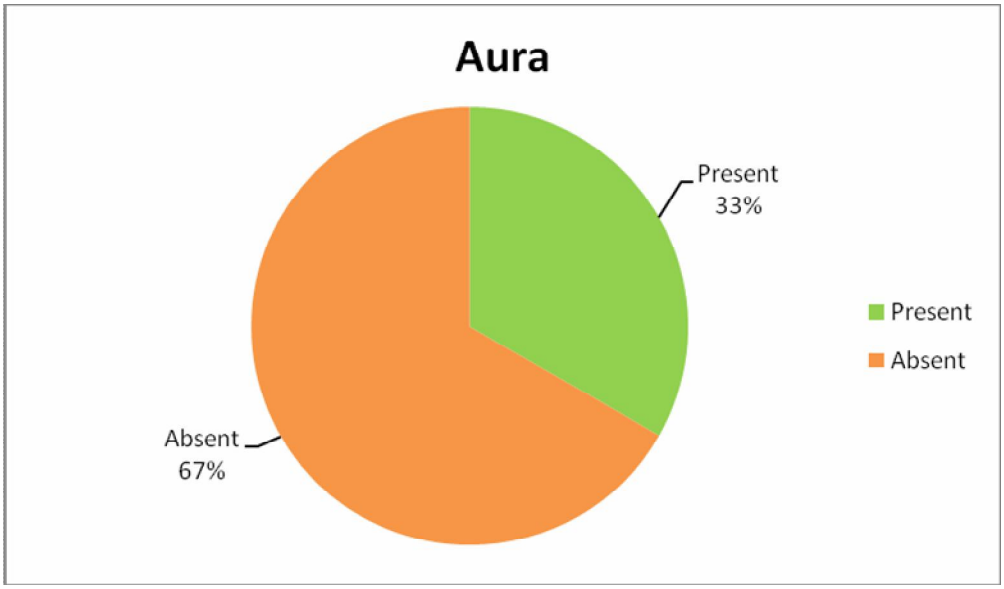
Types of focal seizure	No. of cases	Percentage (%)
SPS	11	18
CPS	22	37
SGS	27	45



Number of patients with Aura

Table 4

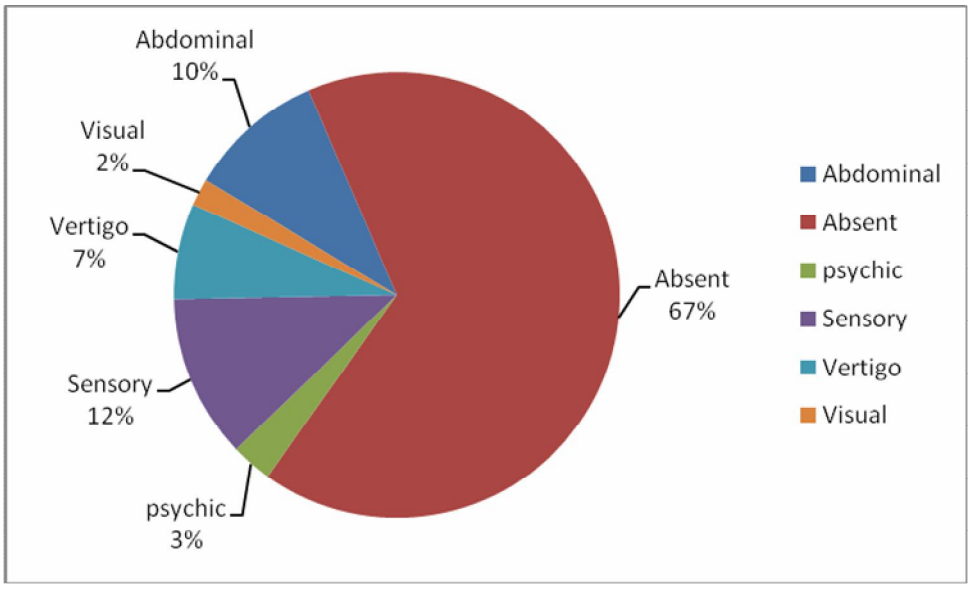
No of patients with aura	SPS	CPS	SGS	NO. OF CASES	Percentage (%)
Present	6	7	7	20	33
Absent	5	15	20	40	67



Types of Aura

Table 5

Aura	CPS	SGS	SPS	Total Cases	Percentage (%)
Abdominal	2	3	1	6	10
Absent	15	20	5	40	67
Psychic	1	0	1	2	3
Sensory	2	3	2	7	12
Vertigo	1	1	2	4	7
Visual	1	0	0	1	2
Olfactory	0	0	0	0	0
Gustatory	0	0	0	0	0
Total Cases	22	27	11	60	100



Illnesses in the past

Table 6

Illnesses in the past	SPS	CPS	SGS	Total no of cases	Percentage (%)
DM	0	4	5	9	15
SHT	2	3	4	9	15
CVA	0	3	2	5	8
PTB	0	1	2	3	5
IHD	0	1	2	3	5
OTHERS	0	3	0	3	5

Others include

Birth asphyxia – 1

Febrile seizures – 1

Post encephalitic sequelae – 1

Of the 60 patients, 52 patients had seizures while they were awake, 3 while they were asleep and 5 had both during sleep and awake period.

Only 6 patients had “precipitating factors”

Fever -2

Emotional Disturbance – 2

Chronic alcoholism -1

Anti convulsant withdrawal – 1

The incidence of focal neurological deficit among the patients was as follows

4 patients had hemiparesis for varying periods following the seizures

6 patients had pre-existing neurological deficit

The following abnormalities were found in the optic fundi

2 patients had papilloedema

Abnormal lab tests

Hyperglycaemia -3

Raised ESR – 4

Mantoux test -3

Sputum AFB -3

HIV -1

EEG was recorded in 13 patients with normal CT scan. The findings were as follows

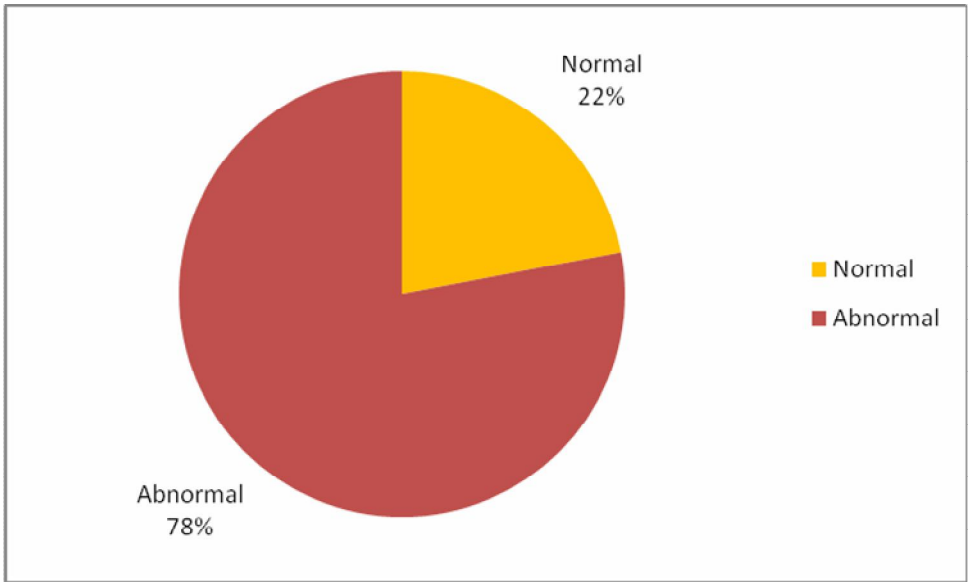
Normal record – 4

Abnormal record – 9

Results of CT scan

Table 7

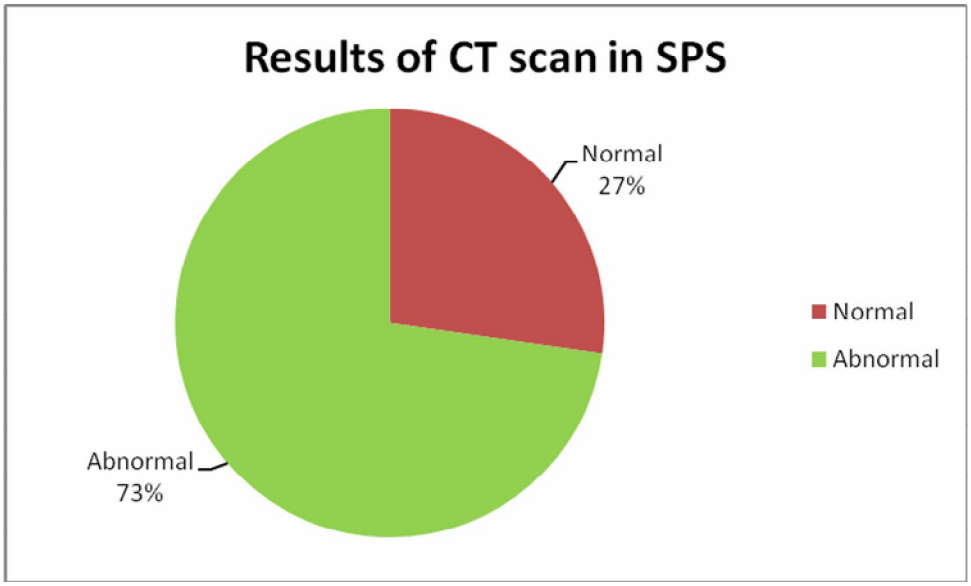
Results of CT scan	Number of cases	Percentage (%)
Normal	13	22
Abnormal	47	78



Results of CT scan in SPS

Table 8

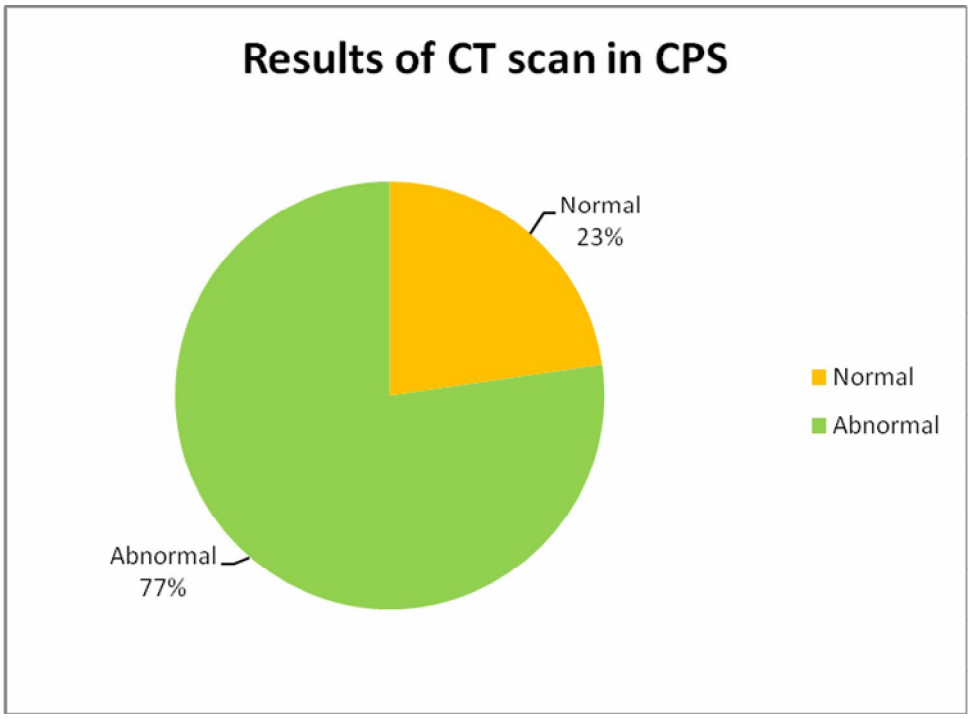
Results of CT scan In SPS	Number of cases	Percentage (%)
Normal	3	27
Abnormal	8	73



Results of CT scan in CPS

Table 9

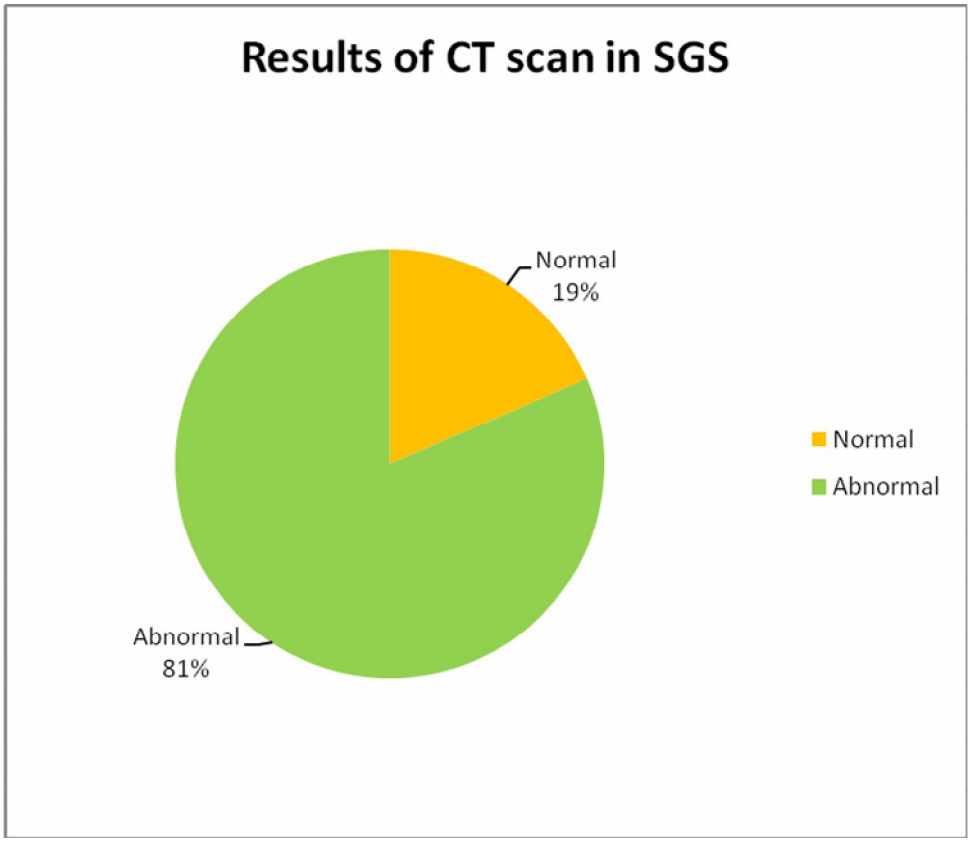
Results of CT scan In CPS	Number of cases	Percentage (%)
Normal	5	23
Abnormal	17	77



Results of CT scan in SGS

Table 10

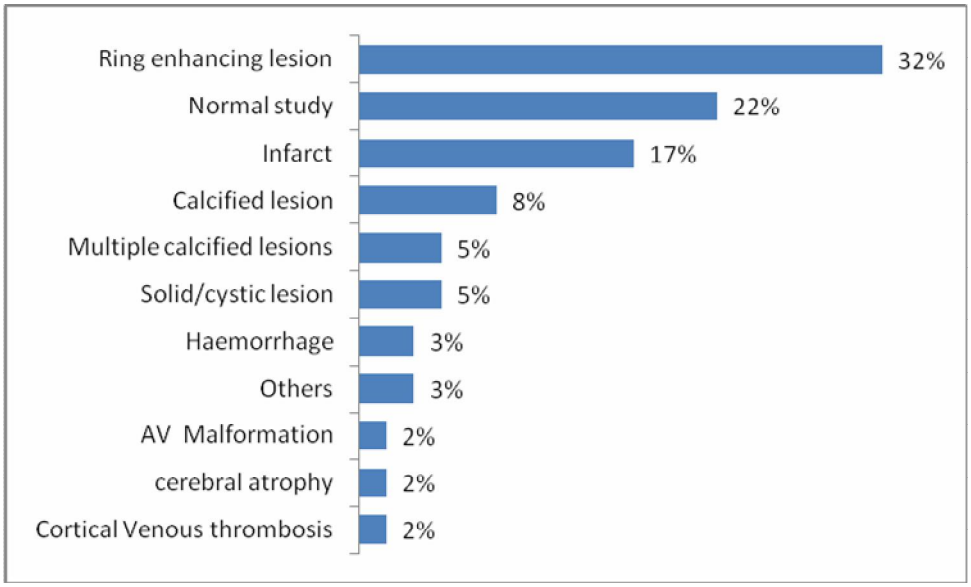
Results of CT scan In SGS	Number of cases	Percentage (%)
Normal	5	19
Abnormal	22	81



CT Scan finding

Table 11

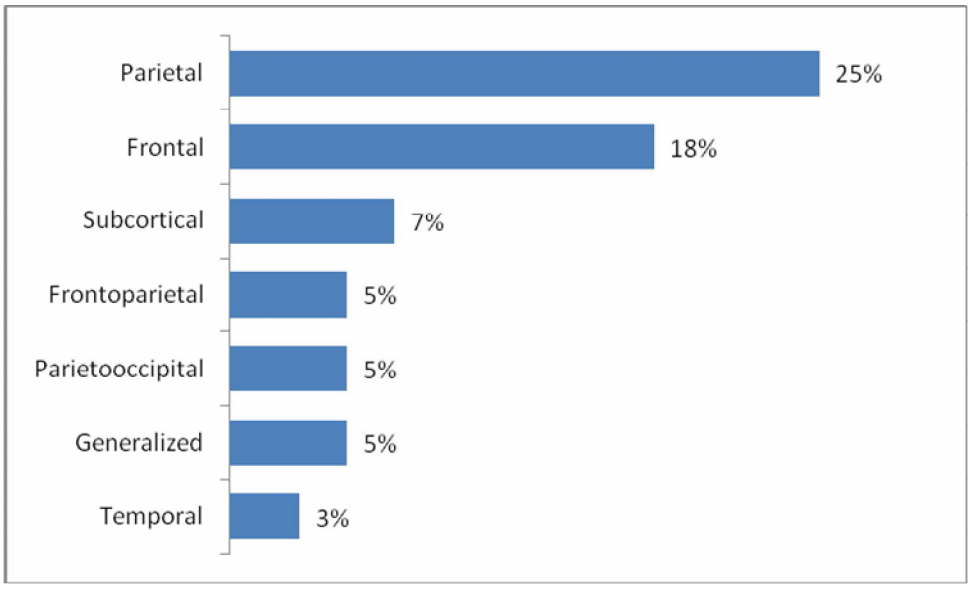
CT Scan finding	CPS	SGS	SPS	Total Cases	Percentage (%)
AV Malformation	1	0	0	1	2
Calcified lesion	2	2	1	5	8
cerebral atrophy	1	0	0	1	2
Cortical Venous thrombosis	1	0	0	1	2
Haemorrhage	0	2	0	2	3
Infarct	5	3	2	10	17
Multiple calcified lesions	0	2	1	3	5
Normal study	5	5	3	13	22
Others	1	0	1	2	3
Ring enhancing lesion	5	12	2	19	32
Solid/cystic lesion	1	1	1	3	5
Total Cases	22	27	11	60	



Location of the lesion in CT scan(brain)

Table 12

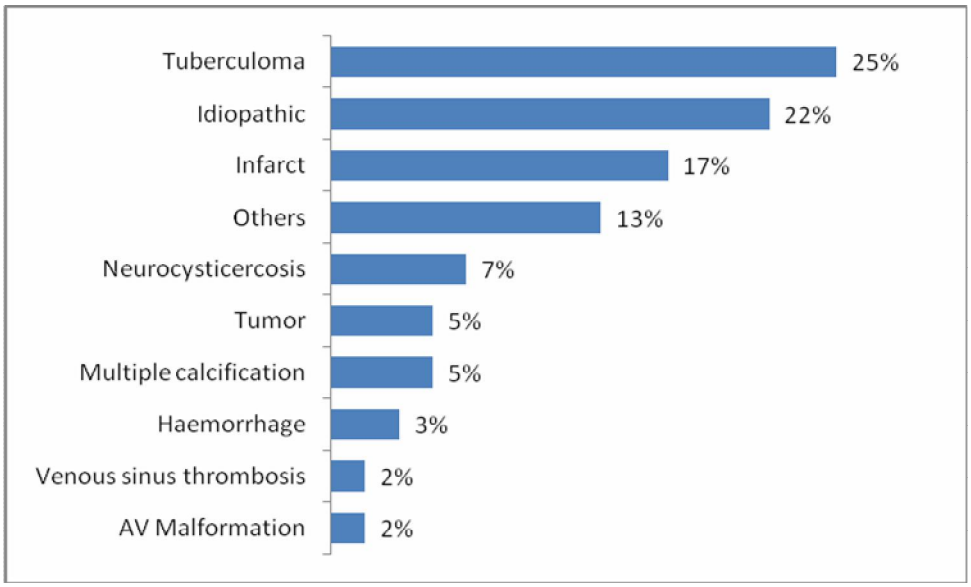
Site	No of patients	Percentage (%)
Parietal	15	25
Frontal	11	18
Temporal	2	3
Frontoparietal	3	5
Parietooccipital	3	5
Generalized	3	5
Subcortical	4	7



Aetiological diagnosis

Table 13

Aetiological diagnosis	No of cases	Percentage (%)
Tuberculoma	15	25
Idiopathic	13	22
Infarct	10	17
Others	8	13
Neurocysticercosis	4	7
Tumor	3	5
Multiple calcification	3	5
Haemorrhage	2	3
Venous sinus thrombosis	1	2
AV Malformation	1	2



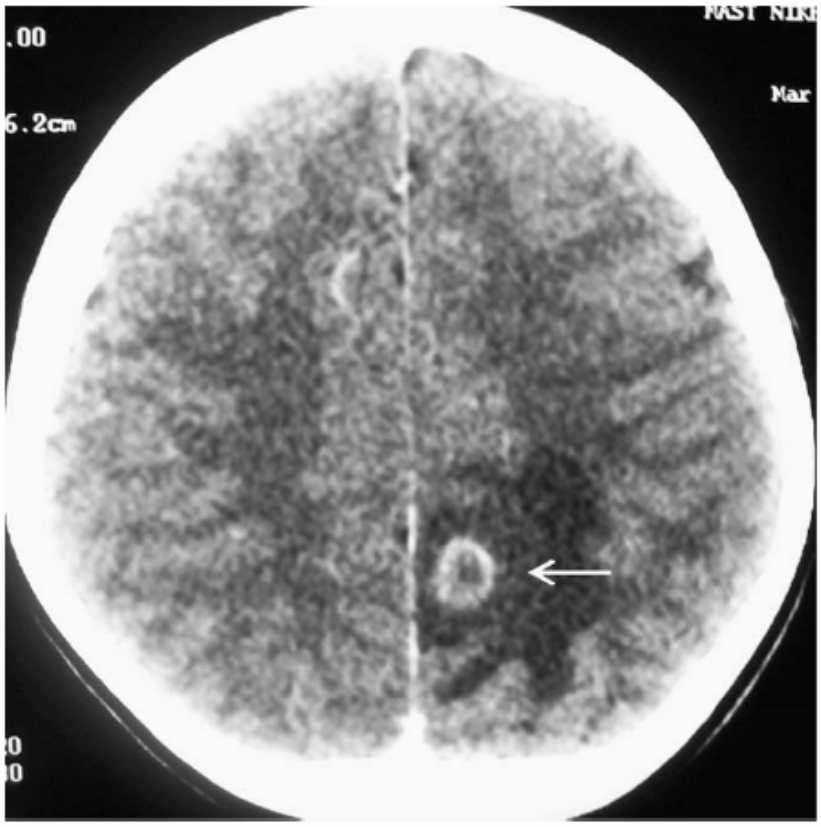
Others include,

old healead granuloma – 5

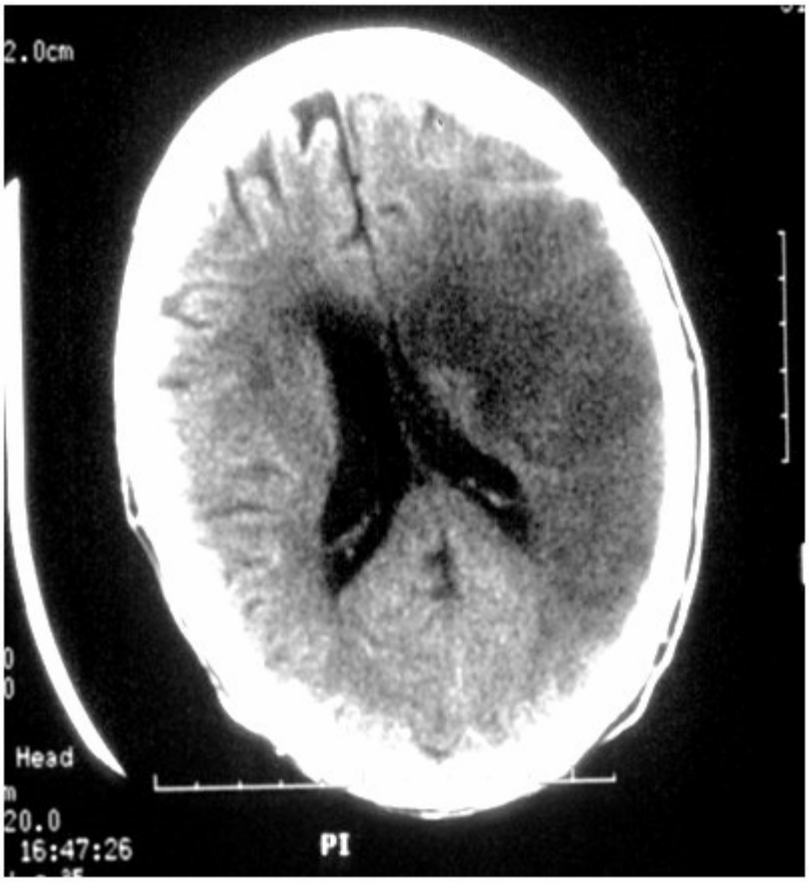
Cerebral atrophy -1

Post encephalitic sequelae -1

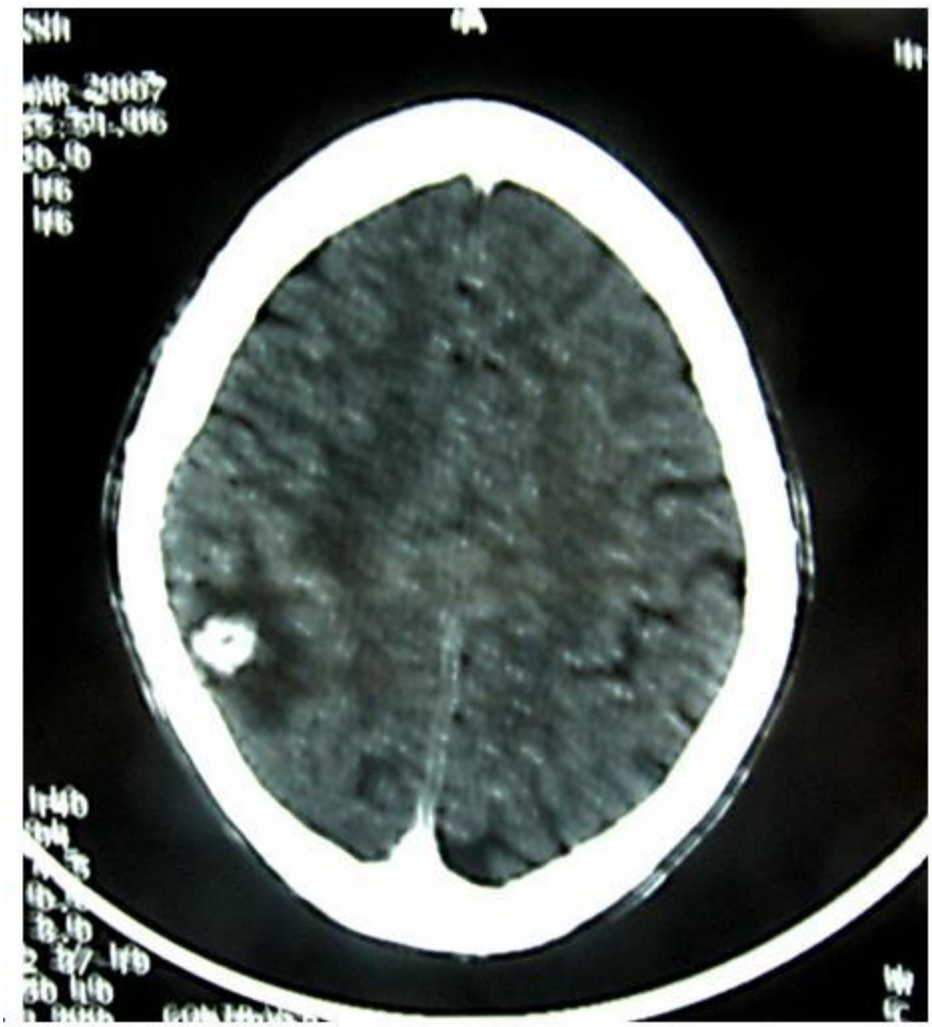
Gliosis - 1



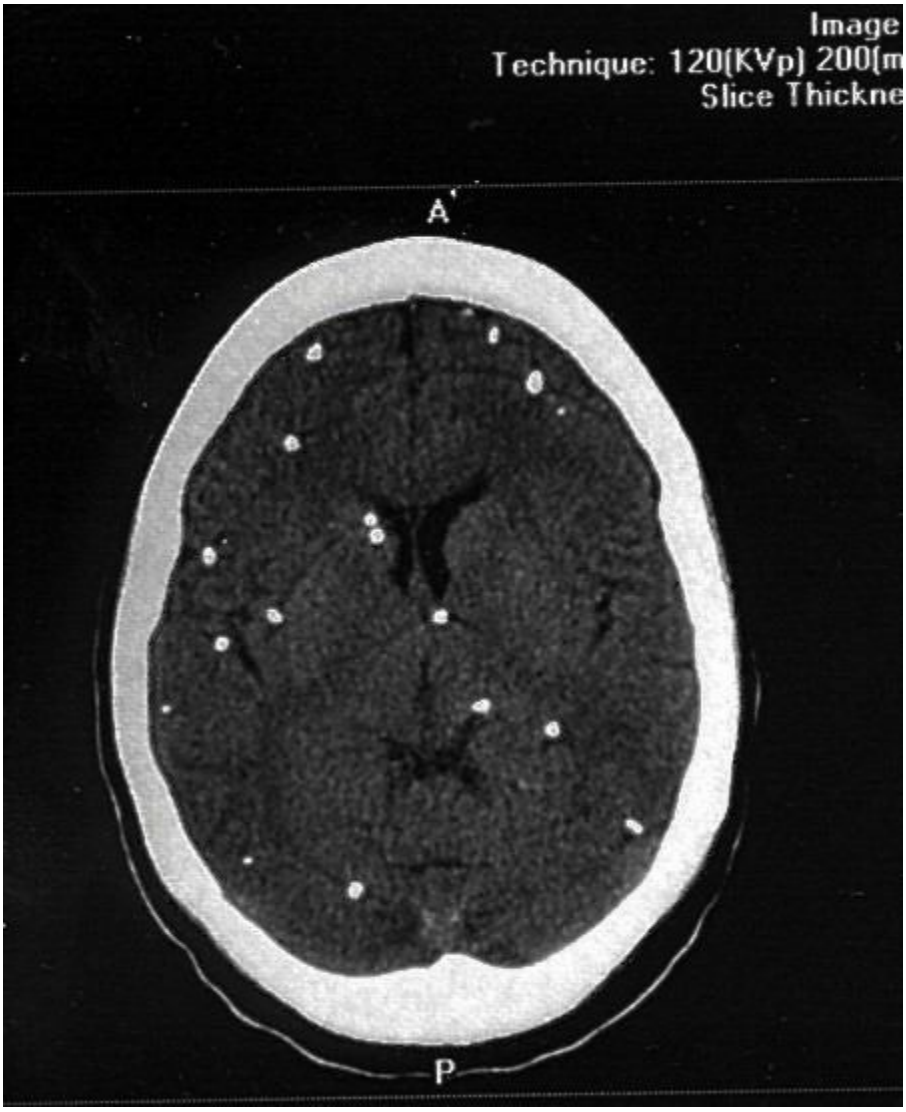
Contrast enhanced axial CT Scan of a patient with neurocysticercosis showing a single dying parenchymal cyst (solitary cerebral cysticercal granuloma) with a scolex of Taenia solium and surrounding perilesional edema



CT scan showing left middle cerebral artery territory infarction



CT Scan showing tuberculoma in right parietal region



Multiple calcified lesions on CT Scan of the brain

DISCUSSION

60 patients of focal seizures who were admitted in medicine, neurology wards, IMCU or attending the neurology OP department of Govt. Royapettah hospital were studied. On analysis it was found that the highest incidence of focal seizures was from 10-19 yrs. There was a male to female ratio of 2.1:1.

The commonest clinical presentation of focal seizures was secondary generalized seizures (45%). In verma[18] series, he reported that majorities of his cases had secondary generalized seizures. Forsgren et al[87,88] in a population based prospective study of epileptic seizures in adults aged > 17 years found that two thirds of the patients had partial seizures, in 80% of these, seizures became secondarily generalized. 16% had generalized seizures without known local onset.

Of the 60 patients registered, 47 patients (78%) had structural lesion and only 13 patients (22%) had normal CT scan. Daras et al[89] studied 155 patients from a city hospital population who developed seizures after age 20. CT was normal in 58 (37.4%) and abnormal in 98 patients (62.6%). The occurrence of abnormal CT scan was higher (73%) in patients with partial seizures. In the series of Ramirez – Lassepas et al.[90] CT scan identified

structural lesions in 37% of patients. In the group of patients with nonfocal findings 15% had structural lesions in CT scan.

Of the patients registered 19 patients (32%) revealed ring enhancing lesion. Wadia et al[91] reported that 26% of Indian patients with focal seizures have an enhancing ring or disc lesion in CT scan.Tandon and Bhargava[92] believed that such CT lesions were tuberculomas based on histopathologic findings, or were associated with tubercular meningitis.

Of the patients registered, 15 patients (25%) had lesions in the parietal lobe. The commonest site of lesion was the parietal lobe. The next common being frontal lobe lesion in 11 patients (18%). It correlates with the study of kumar et al.[19]

In my study Tuberculoma was found to be the commonest etiology of focal seizures (25%), which goes according to the study done by Gulati et al.[22] Of the 60 patients studied, 15 patients had evidence of tuberculoma, 12 patients had large confluent solitary ring enhancing lesion. A study done by Gee GT et al [23] also showed solitary lesions common in tuberculoma. Of the 15 patients with tuberculoma, 11 patients had the pathognomonic target lesion in CT scan brain. In my study 3 patients had x-ray evidence of pulmonary tuberculosis.

Mantoux test was positive in 3 among 15 cases. Study done by Vijayashekaran et al[86] showed tuberculin positivity to be 21% in case

of tuberculoma and 53% in case of lymph node tuberculosis. Mantoux positivity was low in CNS tuberculosis when compared to other forms of tuberculosis.

The diagnosis of tuberculoma was made in 15 cases using CT scan of brain, chest x- ray, sputum AFB, lymph node biopsy and Mantoux test. 12 patients had the pathognomonic target lesion on CT scan of the brain, 3 patients had the chest x-ray evidence of tuberculosis and their sputum being positive for AFB and 1 patient had TB lymphadenitis.

All the 15 patients were subjected to medical treatment with ATT and repeat CT scan brain were taken at the end of nine months. Also patients who had recurrent seizures in between were advised CT scan brain whenever warranted.

Of the 60 patients registered neurocysticercosis was diagnosed in 4 patients. Though it is reported common in pork eaters, in this study all 4 patients were non- pork eaters. Desai had reported a majority of the patients to be vegetarians in his study on neurocysticercosis, but in our study all the patients were non-vegetarians.

The diagnosis of neurocysticercosis were made in 2 patients who had the typical CT feature of a small cyst with hypodensity, well clear

defined edges and a hypodense nodule inside the cyst showing a scolex without perilesional edema or midline shift and also these patients did not have any focal neurological deficit.[84] 2 other patients had small multiple cystic hypodense lesions with regular margins without edema. These patients fulfilled Rajashekaran's et al criteria for diagnosis of neurocysticercosis. These 4 patients were started on albendazole and steroids. Patients did not develop recurrent seizures or worsening of symptoms.

10 (17%) of our patients had cerebral infarction which is the second most common cause of focal seizure. 2 (3%) of our patients had cerebral haemorrhage. 1 (2%) of our patients diagnosed to have venous sinus thrombosis. The single most common cause for seizures in adults is a scar from a previous CVA, or cerebrovascular accident. As explained in "Adams and Victor's Principles of Neurology," one quarter of patients who have had a stroke will later develop some form of seizure.

Of the 10 patients with cerebral infarction 8 were males and 2 were females. The age group was from 40 to 70 yrs. CT Scan revealed old infarct in 6 patients and acute infarct in 4 patients. The clinical presentation was complex partial seizure in 5 patients and secondary generalized seizure in 3 patients. Most of the patients had risk factors like SHT, DM, Smoking. Bladin et al[73] found the incidence of seizures to be 10.6% among 265

patients with intracerebral hemorrhage vs 8.6% among 1632 with ischemic stroke. In another prospective series[75] seizures occurred in 4.4% of 1000 patients, including 15.4% with lobar or extensive intracerebral hemorrhage, 8.5% with subarachnoid hemorrhage, 6.5% with cortical infarction, and 3.7% with hemispheric transient ischemic attacks.

In the present study 3 (5%) patients had brain tumor as a cause for their seizure. All the three were females, one patient was diagnosed to have pilocytic astrocytoma whose age is 16yrs and two others were diagnosed to have gliomas.

Although neoplasms of the brain account for only 1% of cases of epilepsy, seizures occur in approximately 50% of children with supratentorial tumors and 35% to 40% of adults with brain tumors.[68] Seizures occur more commonly with oligodendrogliomas[69] and also with meningiomas. Seizures occur more frequently in supratentorial (22% -68%) than infratentorial (6%) lesions.[70]

Superficial and cortical tumors are associated with higher incidence of epilepsy than are non cortical deep lesions. Both the chronicity of tumor growth and patient age affect the incidence of epilepsy. Slowly growing tumors such as gangliogliomas and dysembryoplastic neuroepithelial tumors are often epileptogenic. Seizure incidence of 70% is reported for astrocytoma

but only 37% for glioblastoma multiforme.[71] Young age is also correlated with the presence of epilepsy.

5 (8%) patients had focal calcified lesion in CT scan and 3 (5%) of patients had multiple calcification in CT scan for which the possible etiology could not be made out. These cases could be due to any healed neuroinfections like tuberculomas or neurocysticercosis etc.

13 (22%) patients in our study had normal CT scan. EEG was taken for all these 13 patients. 9 patients had abnormal record and the remaining 4 patients did not show any abnormality on EEG.

Patients with focal seizures with a normal CT and a normal EEG should be further evaluated by MRI scan. MRI scan is more sensitive and specific than CT scan in evaluation of epilepsy. Convers and collegues performed MRI in hundred patients with intractable partial seizures who had a normal CT and a normal EEG, 31 patients showed abnormalities on MRI of which 20 patients had temporal lobe epilepsy. MRI can detect hippocampal sclerosis which is the commonest cause of complex partial seizures. Chadwick and Smith[93] concluded that plausible arguments may be made for obtaining routine early CT scan and reserving MRI for patients with epilepsy whose seizures are not controlled by antiepileptic drugs.

However, MRI is not affordable by most of the patients in developing countries. The limited availability of MRI in Indian centers makes CT scan the initial investigation of choice of all patients with focal seizures.

SUMMARY AND CONCLUSION

- The highest incidence of partial seizures was in 10-19 yrs age group.
- Male preponderance was noted in this study (68%).
- 78% of patients with focal seizures showed structural lesions on CT scan.
- Tuberculoma of the brain is the commonest cause of partial seizures present in 25% of our patients.
- The most common site of involvement of tuberculoma are parietal and frontal lobes and no infratentorial lesions were observed.
- The next common cause was cerebral infarction in 17% of patients.
- Since neuroimaging alone will not help exactly delineating between tuberculoma and neurocysticercosis other supportive clinical evidence must be taken in to account while arriving at a conclusion.
- MRI is more specific and sensitive, given the socio economic setup of our country CT scan remains the initial investigation of choice in making the etiological diagnosis of partial seizures.

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PROFORMA

A. SL.NO. :

B. Name :

C. Age :

D. Sex :

E. Religion :

F. IP/OP No. :

G. Address :

H. History :

Seizures:

- Onset
- No. of seizures at the time of presentation
- Type of seizure
- Prodromal symptoms
- Aura
-Automatisms
- level of consciousness
- Tongue bite
- Bladder / bowel incontinence
- Post – ictal phase
- Status epilepticus

Headache:

Vomiting:

Fever:

Altered behaviour:

Weakness of limbs:

Blurring of vision:

Other Symptoms:

I.Precipitating factors

J. Past History :

- Last seizure
- Febrile seizures
- Birth hypoxia
- Developmental Disorder
- CNS infection / stroke / head injury
- DM
- Hypertension
- Tuberculosis
- IHD

K. Drug History :

- No. of drugs with dosage

L. Family History :

- Parents / siblings / off-springs

M. Personal History :

- Vegetarian/ Non- vegetarian:
- Pork eater
- Exposure

N. Social History :

- Education
- Marital status
- Socio economic class:

O. Examination :

General Examination

- Temperature / pulse / BP
- Pallor / icterus / cyanosis / clubbing / lymphadenopathy / pedal edema
- Signs of chronic liver disease or renal disease
- Neurocutaneous markers, subcutaneous nodules.
- Clinical evidence of anticonvulsant therapy

Systemic examination

CNS examination

- Higher Mental Functions
- Cranial nerves (incl. Fundus examination)
- Motor system
- Reflexes
- Sensory system
- Gait
- Coordination
- Cerebellar signs
- Signs of meningeal irritation
- Skull and spine

CVS examination

RS examination

PA examination

Investigations :

- Hb, CBC, ESR
- RBS
- BUN, Serum creatinine
- LFT
- HIV
- CXR:
- VDRL

- Mantoux test

- CSF

- EEG

- CT – Scan

Additional investigations:

Final Diagnosis

Remarks:

LIST OF ABBREVIATIONS USED

AED	-	Antiepileptic drug
ATT	-	Anti Tuberculous Treatment
BBB	-	Blood Brain Barrier
CNS	-	Central Nervous System
CPS	-	Complex partial seizures
CSF	-	Cerebrospinal fluid
CT Scan	-	Computerized Tomography
CTA	-	Computerized Tomography Angiography
CVA	-	Cerebrovascular accident
CxR	-	Chest X-ray
DM	-	Diabetes Mellitus
EEG	-	Electroencephalogram
EITB	-	Enzyme Immunotransfer Blot
ELISA	-	Enzyme Linked Immunosorbent assay
HIV	-	Human Immunodeficiency Virus
IHD	-	Ischaemic Heart Disease
MRA	-	Magnetic Resonance Angiography
MRI	-	Magnetic Resonance Imaging
NCC	-	Neurocysticercosis

PET	-	Positron Emission Tomography
PTB	-	Pulmonary Tuberculosis
SGS	-	Secondary Generalized Seizures
SCCG	-	Solitary Cerebral Cysticercus Granuloma
SHT-	-	Systemic Hypertension
SPECT	-	Single Photon Emission Computerized Tomography
SPS	-	Simple Partial Seizures
SSECT	-	Small Single Enhancing CT lesion
SSEL	-	Small solitary enhancing lesion

S.No	Name	Age/Sex	History	Aura	Type	Past H/o	Diet H/o	Cl.Examination		Investigations	Sputum AFB	EEG	CT Scan	Treatment	Etiological Diagnosis
								CNS	CVS RS P/A	CBC RFT FBS HIV	Mx CXR				
1	Kirubhakaran	15/m	Lt Focal Seizure	Sensory	SPS	Nil sig			N	N	N	Normal	Rt high parietal healed granuloma	AED	Calcified lesion
2	Kanaga Sabai	33/m	Rt Focal Seizure Motionless Stare +	Absent	CPS	Nil sig	Non Veg , Pork eater	NFND	N	N	Mx +ve AFB -ve CXR NAD		Lt frontal Ring Enhancing Lesion	ATT AED	Tuberculoma
3	Kalaiarasi	16/f	Rt Focal Seizure Lip Smacking +	Absent	CPS	Nil sig	Non Veg	NFND	N	N	N		Rt Parietal lobe lesion with central hypodense scolex	Albendazole AED steroids	Neurocysticercosis
4	Selvaraj	60/m	Rt Focal Seizure + Chewing + Lip Smacking +	Abdominal	CPS	DM+ SHT+	Non Veg	NFND	N	RBS 220mg	N		Lacunar infarct Lt	AED	Infarct
5	Venkatesan	65/m	Rt Focal Seizure Jacksonian March +	Absent	SPS	SHT +	Non Veg	NFND	N	N	N		Acute infarct Lt Corona Radiata	AED	Infarct
6	GnanaSekar	57/m	Lt Focal Seizure	Absent	SGS	IHD + Infantile hemiparesis Lt +	Non Veg	Lt hemiparesis	N	N	N		old infarct with gliosis Rt Basalganglia/Rt periventricular Region extending to Rt parietal lobe	AED	Infarct
7	Periasamy	65/m	Lt Focal Seizure	Absent	SGS	DM+ HT+	Non Veg	Fundus papilloedema	N	RBS 242mg	N		Rt parieto occipital hemorrhage	AED	Haemorrhage
8	Karikalcholan	46/m	Lt Focal Seizure	Absent	CPS	DM+ SHT+ IHD + old CVA Rt MCA infarct +	Non Veg	Lt hemiparesis	N	N	N		old Rt MCA cortical territory infarct .	AED	Infarct
9	Neelavathi	70/f	Lt Focal Seizure	Absent	SGS	DM+ SHT+ IHD + old CVA Lt hemiparesis CKD+		Fundus Hyperemic Lt hemiparesis	N	N	N		old infarct Rt parieto occipital region	AED	Infarct
10	Tamilarasi	40/f	Lt Focal Seizure +	Sensory	SPS	Nil sig	Non Veg	NFND	N	N	N		Well defined enhancing leison 6x6 cm Rt parietal lobe with perilesional edema S/o Glioma	AED	Glioma
11	Manoharan	13/m	Rt Focal Seizure	Absent	SGS	Nil sig		NFND	N	ESR ↑	Mx +ve CXR NAD AFB -ve		Sub cortical ring enhancing lesion with perilesional edima Lt parietal lobe S/o granuloma	ATT AED	Tuberculoma
12	Divya	16/f		Abdominal	SPS	Nil sig	Veg	NFND	N	N	N		?Edema ?gliosis in b/l parietal lobe suggested follow up	AED	gliosis
13	Kutty	40/m	Rt Focal Seizure Motionless Stare +	Absent	CPS	Cannabis Abuse +	Non Veg	NFND	N	N	N		Water shed infarct Lt fronto parietal region	AED	Infarct
14	Venkatesan	40/m	Lt Focal Seizure Picking movements of hands +	Absent	CPS	SHT + CVA Lt hemiparesis +	Non Veg	Lt hemiparesis	N	N	N		old infarct Rt MCA territory,Rt parietal cortex	AED	Infarct

15	jeromiyas	55/m	Rt Focal Seizure	Absent	SGS	SHT	Non Veg	BP 170/100 Lt hemiparesis + Lt Facial Lag +	N	N	N		Acute intra cerebral haemorrhage in Rt thalamus extending into lateral 3rd & 4th ventricles	AED	Haemorrhage
16	sonu	24/f	Lt focal Seizure Giddiness	Absent	SGS	Nil sig	Non Veg	NFND	N	N	N		Multiple small non ring enhancing cystic lesion parietal region	AED albendazole	neurocysticercosis
17	parveen	23/f	Lt Focal Seizure Chewing + lip smacking + Motionless stare+	Sensory	CPS	Nil sig		NFND	N	N	Mx -ve CXR NAD AFB -ve	Normal	Rt Parietal ring enhancing lesion	AED	Tuberculoma
18	Rajadurai	16/m	Rt Focal Seizure upward gaze + tonic posturing + onset 3 yrs	Absent	CPS	febrile seizures + AV malformation	Veg	NFND	N	N	N		Focal cerebral atrophy with cortical gliosis & calcification in Rt frontal lobe S/o chronic ischaemic changes with distrophic calcification	AED	AV malformation
19	Thiruvasagam	44/m	Lt Focal Seizure Motionless Stare+ onset 37 yrs	Absent	SGS	Nil sig	Non Veg	NFND	N	N	N		Calcified lesion in Rt parietal region intraventricular calcified deposits seen	AED	Calcified lesion
20	Babu	17/m	Rt focal Seizure	Sensory	SGS	Nil sig	Non Veg	NFND	N	Lymph Node Bx - TB ESR ↑	Mx +ve CXR NAD AFB -ve		Single ring enhancing parietal lobe lesion	AED ATT	Tuberculoma
21	Dhiraviam	34/m	Rt Focal Seizure	Absent	CPS	Nil sig	Non Veg	NFND	N	N	N		calcific lesion with out perilesional edema Lt parietal lobe S/o old healed granuloma	AED	Calcified lesion
22	Marimuthu	43/m	behavioural arrest +	Sensory	SGS	DM+ SHT+	Non Veg	NFND	BBS Rt Upper Zone	N	CXR - haziness Rt Upperzone AFB +ve		Solitary ring enhancing lesion with perilesional edema frontal region	ATT AED	Tuberculoma
23	Sangeetha	25/f	Starring look + onset 19 yrs	Vertigo	SPS	Nil sig	Veg	NFND	N	N	N	Normal	Normal study	AED	Idiopathic
24	Parameswari	48/f	Rt Focal Seizure	Abdominal	SGS	DM+	Non Veg	NFND	N	N	N	Normal	Normal study	AED	Idiopathic
25	Shanthi	20/f	Rt Focal Seizure	Vertigo	SGS	Nil sig	Non Veg	NFND	N	N	CXR - NAD		Multiple calcified lesions ?tuberculosis ?neurocysticercosis	AED, albendazole	Multiple calcification
26	Muthiyan	35/m	Lt Focal Seizure Deviation of head to Lt Intense fear +	psychic	CPS	Post encephalitic sequelae Lt hemi paresis	Veg	Lt hemiparesis	N	N	N		No significant abnormalities	AED	Post encephalitic sequelae
27	Velu	13/m	Lt Focal Seizure Lip smacking + Deviation of angle of mouth +	Absent	CPS	Nil sig	Non Veg	NFND	N	N	N	Abnormal record	Normal study	AED	Idiopathic
28	Karunakaran	19/m	Lt Focal Seizure	Absent	CPS	Nil sig	Non Veg	NFND	N	N	Mx -ve CXR NAD AFB -ve		Ring enhancing lesion Rt parietal lesion with perilesional edema	ATT AED	Tuberculoma

29	Narayanan	54/m	Lt Focal Seizure	Absent	SGS	old CVA - Lt hemiparesis	Non Veg	Lt hemiparesis	N	N	N		old infarct Rt MCA terrritory with gliosis		Infarct
30	Anjalai	19/f	Head nodding	Absent	SPS	Nil sig	Veg	NFND	N	N	N	Abnormal record	Normal study	AED	Idiopathic
31	Satish	17/m	Rt Focal Seizure	Sensory	SGS	Nil sig	Non Veg	NFND	N	N	N		Ring enhancing lesion Lt parietal & Rt occipital S/o neurocysticercosis	Albendazole AED steroids	Neurocysticercosis
32	Muthu	25/m	Rt Focal Seizure	Absent	SGS	PTB	Non Veg		Rt Ul BBS +	N	Mx -ve CXR apical fibrosis AFB -ve		Lt frontal ring enhancing lesion with perilesional edema	AED ATT	Tuberculoma
33	Bharathi	13/f	Lt Focal Seizure flashes of light spots +	Visual	CPS	Nil sig	Veg	NFND	N	N	N	Abnormal record		AED	Idiopathic
34	Kirijaramani	26/f	Rt Focal Seizure picking movements of hands +	Absent	SGS	Nil sig	Non Veg	NFND	N	N	N	Abnormal record	Normal study	AED	Idiopathic
35	Antony	40/m	Rt Focal Seizure	Absent	SGS	Seizure disorder 10 yrs duration discontinued trt 2yrs back	Non Veg	NFND	N	N	N		Lt parietal region calcified granuloma(healed)	AED	Calcified lesion
36	Bhaskar	24/m	Rt focal Seizure	Absent	SGS	Nil sig	Non Veg	NFND	N	N	Mx -ve CXR NAD AFB -ve	Normal	Frontal ring enhancement with perilesional edema , typical target lesion	AED ATT	Tuberculoma
37	Karunakaran	28/m	Rt focal seizure	Absent	SPS	Nil sig	Non Veg	NFND	N	N	N	Abnormal record	Normal study		Idiopathic
38	Ravi	35/m	Focal Movements	Absent	SGS	PTB	Non Veg	NFND	N	ESR ↑	CXR apical fibrosis Mx -+ve AFB +ve		Ring enhancing lesion Lt parietal with edema	AED	Tuberculoma
39	Tamilarasi	16/f	CPS	Abdominal	SGS	Nil sig	Veg	NFND	N	N	N		cystic mass lesion with calcification Rt temporal lobe ?pilocytic astrocytoma		pilocytic astrocytoma
40	Kannan	18/m	Aggressive behaviour	psychic	SPS	Nil sig	Non Veg	NFND	N	N	Mx - +ve AFB -ve CXR NAD	Normal	Ring enhancing lesion Rt frontal with perilesional edema	ATT albendazole AED	Tuberculoma
41	Sivakami	45/f		Vertigo	SPS	SHT	Veg	NFND	N	N	N	Normal		AED	Infarct
42	kasi	22/m	Focal becoming generalized	Absent	SGS	Nil sig	Non Veg	NFND	N	N	Mx -ve AFB -ve CXR NAD		Frontal lesion with constrast ring enhancement perilesional edema , typical target lesion	AED ATT	Tuberculoma
43	Rajendran	24/m	Rt Focal Seizure	Absent	SGS	Nil sig	Non Veg	NFND	N	N	N	Normal	Normal study	AED	Idiopathic
44	Vanisree	38/f		Abdominal	CPS	Nil sig	Non Veg	NFND	N	N	N		Calcification Lt temporal cortical region	AED	Calcified lesion
45	Rajaraman	44/m	Recurrent Focal Seizure	Absent	SGS	DM	Non Veg	NFND	Fibro cavity lesion	ESR ↑	CXR fibro cavity AFB +ve		Single ring enhancing parietal lobe lesion	AED ATT	Tuberculoma
46	Selvi	28/f	deviation of angle of mouth	Absent	SPS	Nil sig	Non Veg	NFND	N	N	N	Abnormal record	Multiple healed calcified lesions	AED	Multiple calcification
47	pasupathiram	60/m	Motionless Stare	Absent	CPS	Nil sig	Veg	NFND	N	N	N	Normal	cerebral atrophy	AED	cerebral atrophy
48	Baskaran	62/m	Lt focal seizure	Sensory	CPS	DM+ SHT+	Non Veg	Fundus papilloedema	N	RBS 288mg	CXR - Cardiomegaly		Hypodense lesion Rt parietal lobe ?infarct		Infarct
49	Kalpana	21/f	CPS --> SGS Lip smacking	Absent	SGS	Nil sig	Veg	NFND	N	N	N	Abnormal record	Normal study		Idiopathic

50	Sekar	28/m	Rt Focal Seizure Headache, vomiting ,slurring of speech	Vertigo	CPS	Nil sig	Non Veg	Rt hemi paresis Rt UMN facial palsy Rt hemisensory impairment	N	Homocysteine 31.69 mic mol/l Lupus anticoagulant weakly +ve	N		2.5*2cm lobar hemorrhage in high frontoparietal region Hyper dense sinus		Venous sinus thrombosis, Hyperhomocysteinemia . Lupas anti cogulant weakly +ve .
51	Kiran kumar	12/m	Behavioural arrest, eye deviation	Absent	CPS	Nil sig	Non Veg	NFND	N	N	N	Shows epileptiform activity	Normal study	AED	Idiopathic
52	Gomati	15/f	Focal Movements	Absent	CPS	Mental retardation +	Veg	NFND	N	N	N	Normal	Normal study	AED	Idiopathic
53	Ramesh	15/m	Behavioural arrest	Absent	CPS	Nil sig	Veg	NFND	N	N	N	Shows epileptiform activity	Normal study	AED	Idiopathic
54	Elangovan	28/m	Recurrent SPS with SGS	Absent	SGS	Nil sig	Non Veg	NFND	N	N	N		Generalised multiple calcified lesions	AED albendazole	Multiple calcification
55	Kali	33/m	Rt Focal Seizure	Absent	CPS	Nil sig	Non Veg	NFND	N	N	Mx -ve AFB -ve CXR NAD		Frontal lesion with constrast ring enhancement perilesional edema , typical target lesion	ATT AED	Tuberculoma
56	Giri	17/m	Rt Focal Seizure	Absent	SGS	Nil sig	Non Veg	NFND	N	N	N	Abnormal record	Normal study	AED	Idiopathic
57	Jagadesh	17/m	SPS with SGS	Absent	SGS	Nil sig	Non Veg	NFND	N	N	Mx -ve AFB -ve CXR NAD		Single ring enhancing parietal lobe lesion	ATT AED	Tuberculoma
58	Vasantha	35/f	Rt Focal Seizure	Absent	CPS	Nil sig	Non Veg	NFND	N	Bx oligodendroglioma (low grade)	N		SOL frontal lobe ?glioma	AED	oligodendroglioma
59	Kuppan	28/m	Rt Focal Seizure	Abdominal	SGS	PTB	Non Veg	NFND	N	N	Mx -ve AFB -ve CXR NAD		Lt frontal hypodense lesion with contrast enhancement with perilesional edema	ATT AED	Tuberculoma
60	Rathnam	32/m	Recurrent Focal Seizure	Absent	SPS	Nil sig	Non Veg	NFND	N	N	N		Frontal lobe lesion with central hypodense scolex	AED albendazole steroids	Neurocysticercosis