"A CLINICAL AND ETIOLOGICAL STUDY OF LATE ONSET SEIZURES – A CROSS SECTIONAL STUDY AT KLES DR. PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE"

By

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Dissertation submitted to the
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M. D. (GENERAL MEDICINE)

Under the Guidance of

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I hereby declare that this dissertation entitled **"A CLINICAL AND ETIOLOGICAL STUDY OF LATE ONSET SEIZURES – A CROSS SECTIONAL STUDY AT KLES DR PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE"** is a bonafide and genuine research work carried out by me under the guidance of **Dr. VIJAYAKUMAR. G. SOMANNAVAR** MD Professor, Department of Medicine, Jawaharlal Nehru Medical College, Nehru Nagar, Belgaum-590010.

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LIST OF ABBREVIATIONS USED

AVM	-	Arteriovenous malformation
BP	-	Blood pressure
CBC	-	Complete blood count
CNS	-	Central nervous system
CSF	-	Cerebrospinal fluid
CT	-	Computed tomography
CVT	-	Cortical venous thrombosis
ECG	-	Electrocardiography
EEG	-	Electroencephalography
EITB	-	Enzyme linked immuno electro transfer blot
ELISA	-	Enzyme linked immuno sorbent assay
FLAIR	-	Fluid attenuated inversion recovery
GTCS	-	Generalized tonic clonic seizures
HIV	-	Human immunodeficiency virus
ICT	-	Intracranial tension
ILAE	-	International League Against Epilepsy
LP	-	Lumbar puncture
MRI	-	Magnetic resonance imaging
PCR	-	Polymerase chain reaction
SSS	-	Superior sagittal sinus
TB	-	Tuberculosis

ABSTRACT

Background and Objectives

Patients who have experienced a seizure or have been suffering from epilepsy form a big chunk of neuromedical practice. Seizure activity can have various manifestations ranging from dramatic convulsive activity to experimental phenomenon not readily discernable by an observer. Detailed work up is generally required for total evaluation of such cases and treatment. The objective of the present study was to determine various clinical presentations and etiological factors of late onset seizures.

Methods

The present cross-sectional study was conducted in Department of Medicine, at KLES Dr Prabhakar Kore Hospital and Medical Research Centre, Belgaum during January 2007 to December 2007 on 40 patients above 35 years presenting with seizures for the first time. Detailed history, clinical examination, blood investigations, EEG and cranial imaging were performed. Other investigations like serological tests, LP CSF study, ECG, Chest X-Ray and Echocardiography as necessary were performed

Results

Majority of patients presenting with seizures who had focal neurodeficits had organic lesion in the brain. Abnormal EEG helped to confirm the diagnosis of seizures and when it showed focal abnormalities gave a clue to underlying structural abnormality. Overall cranial imaging was abnormal in 72.5% of patients, 62.5% of generalized seizure group and 87.5% of partial seizure group

patients. The commonest etiology of seizure in the age group between 35 to 54 years was idiopathic and after the age of 55 years commonest etiology was cerebral infarct.

Conclusion and interpretation

Majority of patients presenting with seizures who had focal neurodeficits had organic lesion in the brain. EEG was not very useful in evaluation of seizures. However abnormal EEG helped to confirm the diagnosis of seizures. Proportion of patients with idiopathic etiology was higher in generalized seizure group as compared to partial seizure group and proportion of patients with structural brain lesion was higher in partial seizure group.

Keywords

Seizures; Epilepsy; Cranial imaging; Electroencephalography; Partial seizures; Primary generalized seizures;

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INTRODUCTION

Patients who have experienced a seizure or have been suffering from epilepsy form a big chunk of neuromedical practice. Seizures being a complex symptom of underlying disease, a detailed work up is generally required for the total evaluation of such cases and treatment. No fixed guidelines exist to evaluate the cases of seizures. Epilepsy is a group of conditions and not a single homogeneous disorder and seizure may be a symptom of both diverse brain disorders or an otherwise normal nervous system.[1] It is neither possible nor desirable to develop inflexible guidelines for what constitutes a standard or minimal set of diagnostic tests.

In the past a large number of cases used to be labelled as seizure of unknown origin. The introduction of electroencephalography (EEG), magnetic resonance imaging (MRI) and computed tomography (CT) scan has helped to sort out the causes of seizures. The evaluation of cases of seizures includes a detailed history, clinical examination, EEG, advanced neuroimaging and functional neuroimaging. Various physicians use various combinations of these methodologies considering their cost factor and yield of information.

Seizure activity can have various manifestations ranging from dramatic convulsive activity to experimental phenomenon not readily discernable by an observer.[1] Study of its various clinical manifestations helps to identify specific etiology which guides in selecting appropriate therapy and provides potentially vital information regarding prognosis.

Localization abnormalities in cases of partial seizures are from 28% to 80% as observed in different studies.[2,3] Studies done on patients with generalized seizures also show similar abnormalities.[4]

In view of the above facts, present study was conducted to determine the various clinical manifestations and etiological factors of late onset seizures.

OBJECTIVES

The objective of the study is to determine the various clinical presentations and etiological factors of late onset seizures.

REVIEW OF LITERATURE

HISTORY

It is reasonable to assume that epilepsy is as old as mankind. The irregular and intermittent advance toward understanding epilepsy began with the first known book on epilepsy, On the Sacred Disease about 2,400 years ago. The author of On the Sacred Disease is not known but is referenced as Hippocrates.[5,6] He rejected both the then belief that individual Greek gods cause epilepsy and the superstitions and magic that were in use to avoid and cure epilepsy.

The Hippocratic writings were known to Galen, the influential Greek physician of the second century A.D. who dissected the brain and speculated that epilepsy resulted from an accumulation within the cerebral ventricles of two of the four Greek "humors" phlegm and bile. After this till fifteenth century diseases such as epilepsy were generally attributed to supernatural control.

At the turn of the seventeenth century William Gilbert abruptly accelerated a change in approach from mystical and supernatural to scientific particularly for magnetic and electric phenomena. In 1667, Thomas Willis, the London physician and anatomist who originated the term neurology and became immortalized by describing the circle of Willis, reaffirmed Descarte's ideas that the source of both seizures and their auras was in the brain.

Richard Caton[7] established himself as the first person in the world to observe the continuous spontaneous electrical activity of the brain. He described the existence of electrical currents of the grey matter and noted that feeble

currents of varying direction pass through the multiplier (amplifier) when the electrodes are placed on two points of the external surface, or one electrode on the grey matter, and one on the surface of the skull.

In 1929, Dr. Hans Berger,[8] a professor of psychiatry and chair of the psychiatric clinic at the university of Jena in Germany, published his discovery that spontaneous brain electrical activity in humans could be recorded from the scalp. By 1931 he reported that interictal EEG changes were common in epilepsy and later that year he recorded human spike and wave activity.

In a research EEG laboratory set up at Boston City Hospital in 1934, Frederick Gibbs, Hallowell Davis and William G. Lennox[9] in 1935 first demonstrated spike and wave complexes interictally and during clinical absences. Gibbs, Lennox and Gibbs and Jasper[10] demonstrated focal spikes in localization related epilepsy in 1936. In 1937 the first clinical department in US to formally perform and charge for EEG services[11] was opened at Massachusetts General Hospital by Robert Schwab and others quickly followed.

However, the EEG years ago lost its place as a frontline noninvasive method for recognizing structural lesions. Structural neuroimaging prior to the introduction of CT and MRI were largely limited to X-ray skull, pneumoencephalography and conventional radoactive isotope imaging.[12,13]

The development of CT scan in the 1970's represented the beginning of contemporary structural neuroimaging. A large number of workers have worked on the role of CT in the evaluation of patients with epilepsy and to this the credit must certainly go to Hounsfield's[14] description of the CT scan and clinical studies

reported by Ambrose[15] who have shown CT to be a sensitive and accurate diagnostic modality with no risk or mortality what so ever.

DEFINITIONS OF SOME TERMS

Seizure

A seizure (From the Latin *sacire* "to take possession of") is a paroxysmal event due to abnormal, excessive, hypersynchronous discharges from an aggregate of central nervous system (CNS) neurons. Depending on the distribution of discharges, this abnormal CNS activity can have various manifestations, ranging from dramatic convulsive activity to experimental phenomena not readily discernible by an observer.[1]

Epilepsy

Epilepsy describes a condition in which a person has recurrent seizures due to a chronic, underlying process.[1] The definition implies that a person with single seizure or recurrent seizures due to correctable or avoidable circumstances does not necessarily have epilepsy.

Incidence and Prevalence

- In most developed countries incidence rates range from 40 to 70 per 100,000 but in developing countries the rates may be as high as 100 to 190 per 100,000.[16]

- Similarly the prevalence of active epilepsy defined as persons who take anticonvulsants or who have had a seizure in the past five years ranges

from 4 to 10 per 10,000 in developed countries and upto 57 per 10,000 in developing countries.[16]

- Studies have estimated that 1.5% to five percent of any population will have a seizure at some time.[16]

- The estimated number of persons with epilepsy in India is approximately 5.5 million. [17]

- Based on a study[18] which reported an incidence of 49.3 per 100,000 population, the number of new persons with epilepsy in India each year would be close to half million.

Classification of seizures

The classification to be followed here was first proposed by Gastaut in 1970 and was then refined repeatedly by commission on classification and terminology of the International League Against Epilepsy (ILAE). In 1981, ILAE published a modified version of the international classification of Epileptic Seizures[1] which is presented below.

This system is based on the clinical features of seizures and associated electroencephalographic findings. Other potentially distinctive features such as etiology or cellular substrate are not considered in this classification system.

Classifications of seizures[1]

1. Partial seizures
    a) Simple partial seizures (with motor, sensory, autonomic or psychic signs)
    b) Complex partial seizures.
    c) Partial seizures with secondary generalization.
2. Primarily generalized seizures
    a) Absence (petit mal)
    b) Tonic clonic (Grand mal)
    c) Tonic
    d) Atonic
    e) Myoclonic
3. Unclassified seizures
    a) Neonatal seizures
    b) Infantile spasms

A fundamental principle is that seizures may be either partial (synonymous with focal) or generalized. Partial seizures are those in which the seizure activity is restricted to discrete areas of the cerebral cortex. Generalized seizures involve diffuse regions of the brain simultaneously.

Partial seizures

Partial seizures occur within discrete regions of the brain. If consciousness is fully preserved during the seizure, the clinical manifestations are considered relatively simple and the seizure is termed a simple partial seizure. If

consciousness is impaired, the symptomatology is more complex and the seizure is termed a complex partial seizure. An important additional subgroup comprises those seizures that begin as partial seizures and then spread diffusely throughout the cortex, i.e. partial seizures with secondary generalization.

Simple Partial Seizures

Simple partial seizures cause motor, sensory, autonomic or psychic symptoms without an obvious alteration in consciousness. The EEG recorded with scalp electrodes during the seizure (ictal EEG) may show abnormal discharges in a very limited region over the appropriate area of cerebral cortex if the seizure focus involves the cerebral convexity. Seizure activity occurring within deeper brain structures is often not recorded by the standard EEG, however, and may require intracranial electrodes for its detection.

Simple partial seizures may also manifest as changes in somatic sensation (for example paresthesias), vision (flashing lights or formed hallucinations), equilibrium (sensation of falling or vertigo), or autonomic function (flushing, sweating, piloerection). Simple partial seizures arising from the temporal or frontal cortex may also cause alterations in hearing, olfaction or higher cortical function (psychic symptoms).[1]

Complex Partial Seizures

Complex partial seizures are characterized by focal seizure activity accompanied by a transient impairment of the patient's ability to maintain normal contact with the environment. The patient is unable to respond appropriately to visual or verbal commands during the seizure and has impaired recollection or

awareness of the ictal phase. The start of the ictal phase is often a sudden behavioral arrest or motionless stare, which marks the onset of the period of amnesia. The behavioral arrest is usually accompanied by automatisms, which are involuntary, automatic behaviours that have a wide range of manifestations. Automatisms may consist of very basic behaviors such as chewing, lip smacking, swallowing or "picking" movement of the hands or more elaborate behaviors such as a display of emotion or running. Examination immediately following the seizure may show an anterograde amnesia or in cases involving the dominant hemisphere, a post-ictal aphasia.

The routine, interictal (that is between seizures) EEG in patients with complex partial seizures is often normal or may show brief discharges termed epilepiform spikes, or sharp waves.

The range of potential clinical behaviours linked to complex partial seizures is so broad that extreme caution is advised before concluding that stereoptypic episodes of bizarre or atypical behavior are not due to seizure activity. In such cases additional, detailed EEG studies may be helpful.

Partial Seizures with Secondary Generalization

Partial seizures can spread to involve both cerebral hemispheres and produce a generalized seizure, usually of the tonic-clonic variety. Secondary generalization is observed frequently following simple partial seizures, especially those with a focus in the frontal lobe, but may also be associated with partial seizures occurring elsewhere in the brain. A partial seizure with secondary generalization is often difficult to distinguish from a primarily generalized tonic-

clonic seizure, since bystanders tend to emphasize the more dramatic, generalized convulsive phase of the seizure and overlook the more subtle, focal symptoms present at onset. In some cases, the focal onset of the seizure becomes apparent only when a careful history identifies a preceding aura (i.e. simple partial seizure). Often, however, the focal onset is not clinically evident and may be established only through careful EEG analysis.

Generalized seizures

By definition, generalized seizures arise form both cerebral hemispheres simultaneously. However, it is currently impossible to exclude entirely the existence of a focal region of abnormal activity that initiates the seizure prior to rapid secondary generalization. For this reason, generalized seizures may be practically defined as bilateral clinical and electrographic events without any detectable focal onset.

Absence Seizures (Petit Mal) are characterized by sudden, brief lapses of consciousness without loss of postural control. The seizure typically lasts for only few seconds, consciousness returns as suddenly as it was lost, and there is no postictal confusion.

Absence seizures usually begin in childhood (ages four to eight) or early adolescence and are the main seizure type in 15 to 20% of children with epilepsy.

The electro physiologic hallmark of typical absence seizures is a generalized, symmetric, three Hz spike and wave discharge that begins and ends suddenly, superimposed on a normal EEG background.

Generalized, Tonic-Clonic Seizures (Grand Mal)

Primarily generalized tonic-clonic seizures are the main seizure type in ~10% of all persons with epilepsy. They are also the most common seizure type resulting form metabolic derangement and are therefore frequently encountered in many different clinical settings.

The initial phase of the seizure is usually tonic contraction of muscles throughout the body, accounting for a number of the classic features of the event. Tonic contraction of the muscles of expiration and the larynx at the onset will produce a loud moan or “ictal cry.” Respirations are impaired, secretions pool in the oropharynx, and cyanosis develops. Contraction of the jaw muscles may cause biting of the tongue. A marked enhancement of sympathetic tone leads to increases in heart rate, blood pressure, and papillary size. After 10 to 20s, the tonic phase of the seizure typically evolves into the clonic phase, produced by the superimposition of periods of muscle relaxation on the tonic muscle contraction. The periods of relaxation progressively increase until the end of the ictal phase, which usually lasts not more than one minute. The postictal phase is characterized by unresponsiveness, muscular flaccidity and excessive salivation that can cause stridorous breathing and partial airway obstruction. Bladder or bowel incontinence may occur at this point. Patients gradually regain consciousness over minutes to hours, and during this transition there is typically a period of postictal confusion.

Atonic Seizures

Atonic seizures are characterized by sudden loss of postural muscle tone lasting one to two seconds. Consciousness is briefly impaired, but there is usually no postictal confusion.

Myoclonic Seizures

Myoclonus is a sudden and brief muscle contraction that may involve one part of the body or the entire body. Pathologic myoclonus is most commonly seen in association with metabolic disorders, degenerative CNS diseases or anoxic brain injury.[1]

The causes of seizures and epilepsy

Seizures are a result of a shift in the normal balance of excitation and inhibition within the CNS. Given the numerous properties that control neuronal excitability, there are many different ways to perturb this normal balance, and therefore many different causes of both seizures and epilepsy.[19]

The normal brain is capable of having a seizure under the appropriate circumstances, and there are differences between individuals in the susceptibility or threshold for seizures. This implies there are various underlying, endogenous factors that influence the threshold for having a seizure.

There are a variety of conditions that have an extremely high likelihood of resulting in a chronic seizure disorder. The high propensity for severe traumatic brain injury to lead to epilepsy suggests that the injury results in a long-lasting, pathologic change in the CNS that transforms a presumably normal neural

network into one that is abnormally hyperexcitable. This process is known as epileptogenesis, and the specific changes that result in a lowered seizure threshold can be considered epileptogenic factors.[19] Other processes associated with epileptogenesis include stroke, infections and abnormalities of CNS development.

Seizures are episodic. This implies there are important provocative or precipitating factors that induce seizures in patients with epilepsy. Precipitants include those due to intrinsic physiologic processes, such as psychological or physical stress, sleep deprivation or hormonal changes associated with the menstrual cycle. They also include exogenous factors such as exposure to toxic substances and certain medications.

These observations emphasize the concept that the many causes of seizures and epilepsy result form a dynamic interplay between endogenous factors, epileptogenic factors and precipitating factors.[1]

Etiological factors of seizures

The literature contains a considerable number of studies devoted to analyzing etiological factors of seizures in adults.

In a Denmark study[20] (1985) involving 221 patients who had their first seizure after the age of 25 years, all patients underwent clinical evaluation, EEG and brain CT scan. The major etiological group was the one where no cause could be detected (38%). Alcohol abuse as the etiology defined as cases with a history of long standing alcohol overuse, concomitant signs of alcohol intoxication and spontaneous recurrent epileptic seizures made up a group of one

fourth (25%) of all the patients with late onset epilepsy. Brain tumor was the cause in 16% and cerebrovascular infarction in 14%.

In another retrospective study,[21] 250 patients with late onset seizures presenting in the age group between 22 to 88 years were evaluated clinically with EEG and CT scan. No cause could be identified in 49 patients (19.5%). Among the rest of the cases the most frequent etiology were chronic alcoholism 62 (25%), brain tumors 41 (16%), cerebrovascular disease 33 (13%) and post traumatic epilepsy 28 (11%).

In a retrospective study[22] of three years duration involving 247 adult patients presenting with first seizure, CT scan was performed in 247 patients and EEG in 209. Etiologies were found to be (1) unknown (2) alcohol abuse (3) stroke (4) tumor.

Another study[23] involving 119 adult patients presenting to casualty with generalized seizure reported the etiologies as withdrawal from alcohol, drug or drug intoxication in 48 (40%), cerebral infraction in 14 (11.7%), intracranial haemorrhage in six (5%), primary cerebral neoplasm 10 (8.4%), metastases seven (5.8%), idiopathic 17 (14.2%), remote trauma and human immunodeficiency virus (HIV) encephalopathy in four each of patients, two each form Alzheimer's disease and toxoplasmosis, one each from viral encephalitis, bacterial meningitis, arteriovenous malformation and arachnoid cyst.

A study[24] was conducted on 100 consecutive patients with seizures that started after the age of 25 years in Mexico. All patients underwent CT, EEG and

additionally cerebrospinal fluid (CSF) analysis was performed in 82 of them. Neurocysticercosis or its sequelae were diagnosed in 50 patients.

In a south Indian study,[4] the putative etiologies in 991 patients with symptomatic localization related epilepsies were studied. Cerebrovascular diseases were the risk factors in 48% of patients with remote symptomatic epilepsy. Neurocysticercosis, single CT enhancing lesion (SCTEL), and small single cerebral calcific CT lesions (SSCCCTL) together accounted for 40% of etiological factors and neurotuberculosis for 10%.

In another prospective study[25] of 130 patients with adult onset epilepsy, all patients had CT scan brain and when necessary brain magnetic resonance imaging. Structural brain lesion was found in 51% of patients. The most frequent causes of seizures were Neurocysticercosis in 28% followed by cerebral infarct (11%) and brain atrophy (11%).

In a epilepsy survey study[26] involving rural population of Honduras, among 6473 residents surveyed, 151 persons with epilepsy were identified. 100 of whom had active epilepsy. These patients underwent video EEG, CT brain and serum enzyme linked immunoelectro transfer blot (EITB) for cysticercosis. Symptomatic epilepsy was primarily due to neurocysticercosis (37%), perinatal brain damage (8%), post traumatic (3%) and past stroke (2%). Eight percent were idiopathic and 30% were cryptogenic.

In another epilepsy survey study[27] in rural Europe in 2005, among 2,548 residents, patients with epilepsy as well as age and sex matched controls underwent a CT head and scalp EEG. Blood samples were also collected for

determination of anticysticercal antibodies. Neurocysticercosis was associated with one third cases of epilepsy.

Another Indian study[28] involving 450 patients with single small enhancing CT lesion (SSECT) opined that solitary cerebral cysticercous granuloma was one of the commonest causes of seizures in Indian patients.

Electroencephalography (EEG)

The EEG provides a dynamic record of electrical potentials of brain. This is recorded by the EEG acting like a powerful and complex amplifier which has the ability to amplify these potentials. These amplified potentials further cause deflections of ink writing pens strategically placed. The pens then produce a wave like pattern on a fast moving strip of paper. The EEG may be helpful in the interictal period by showing certain abnormalities that are highly supportive of the diagnosis of epilepsy.

The EEG is recorded from metal electrodes placed on the scalp. The placement of recording electrodes is generally based on the international 10 to 20 system.[29]

International 10 to 20 system

(i) The midline of the head is divided into portions of 10%, 20%, 20%, 20% 20% and 10% beginning at the nasion and ending at the inion.

(ii) The transverse line of the head is divided into portions of 10% 20% 20%, 20%, 20% and 10% beginning at the right preauricular point and ending at the left preauricular point.

(iii) Numerals or anatomical nomenclature are given to the points as shown in the Figure No. 1

(iv) Electrode pairs are interconnected in different arrangements called Montages to permit a comprehensive survey of the brain electrical activity. Typically montages are designed to compare symmetrical areas of the two hemispheres as well as anterior versus posterior regions or parasagittal versus temporal area in the same hemisphere. The EEG can be reconstructed after digital recording.

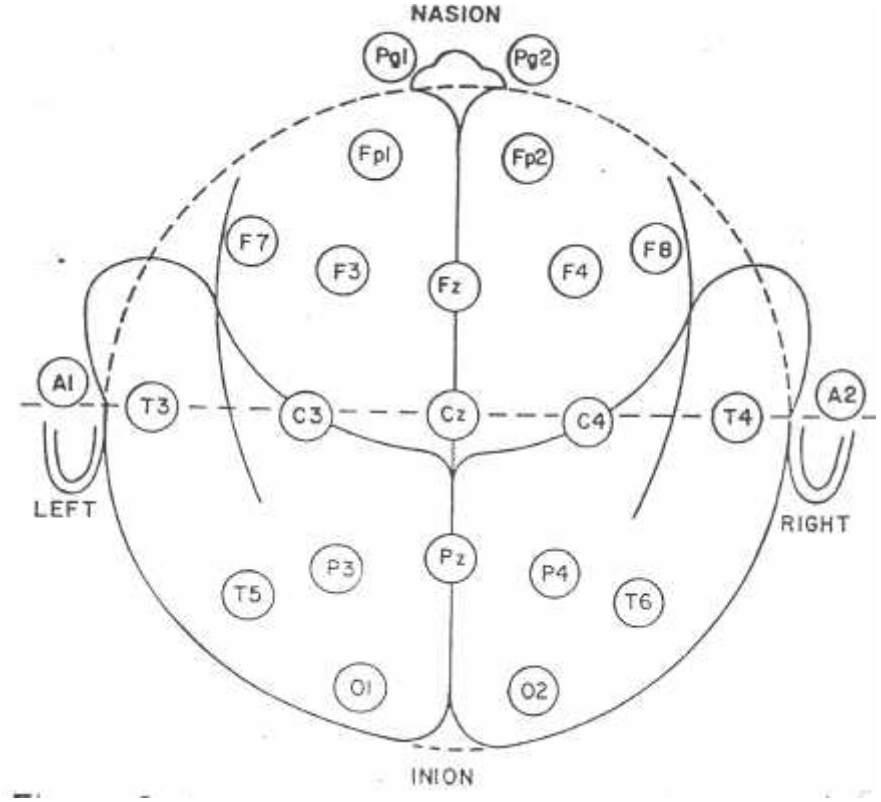


Figure No. 1: The International 10 to 20 system of electrode placement.

Earlobe (A), Central (C), Frontal (F), Frontal Polar (Fp), Parietal (P), Nasopharyngeal (Pg), Temporal (T), Occipital (O).

Right sided placements are indicated by even numbers, left sided placement by odd numbers and Midline placements by z.

Table No. 1: Designations of the electrode positions

Electrode Number		International Symbol		Name
Left	**Right**	**Left**	**Right**	
1	2	FP1	FP2	Frontal Pole
3	4	F3	F4	Frontal
5	6	C3	C4	Central
7	8	P3	P4	Parietal
9	10	O1	O2	Occipital
13	14	F7	F8	Anterior Temporal
15	16	T3	T4	Middle Temporal
17	18	T5	T6	Posterior Temporal
19		Fz		Midline Frontal
24		Cz		Midline Central
20		Pz		Midline Parietal
11	12	A1	A2	Auricular

Various rhythms recorded in the EEG are

Rhythm	Frequency
(i) Alpha	8 to 12/second
(ii) Beta	>12/second
(iii) Theta	4 to 7/second
(iv) Delta	< 4 /second

Normal EEG activity

In most normal adults, the waking pattern of EEG activity consists mainly of sinusoidal oscillations occurring at 8 to 12 Hz (alpha rhythm). The alpha rhythm has the additional characteristic of being attenuated by eye opening, mental activity and drowsiness.

Activity faster than 12 Hz (Beta rhythm) is recorded from frontal region symmetrically and may be especially prominent in patients receiving barbiturates or benzodiazepine group of drugs.

A small amount of theta (4 to 7 Hz) activity may normally be present over the temporal regions, somewhat more so in persons over 60 years of age.

Delta (1 to 3 Hz) activity is not present in normal waking adult.

Abnormal recording in epileptic seizures

The main types of epileptiform discharges are spikes, sharp waves and spike and wave discharges.

- Spikes are brief potentials having a steep ascending and descending limb with a duration of < 70 msec.
- Sharp waves are broader potentials with pointed peaks, having a duration that usually ranges between 70 and 200 msec.
- Spike and wave discharge consists of a spike followed by slow wave

Epileptic discharges may be focal or generalized.

EEG abnormalities indicating focal dysfunction

- Focal delta activity is the classic EEG sign of local disturbance in cerebral function. A structural lesion is most strongly suggested if the delta activity is continuously present, shows variability in waveform, amplitude, duration and morphology (so called "polymorphic" or "arrhythmic" activity) and persists during changes in physiological state.
- Periodic lateralized epileptiform discharges (Pled's).
- Voltage attenuation.
- Intermittent rhythmic slow waves. When bursts of rhythmic slow waves occur (theta or delta) focally or are lateralized to one hemisphere usually indicate a structural abnormality.

NEUROIMAGING

Neuroimaging is one of the most important advances made in the past decade in the management of seizure disorders. Magnetic resonance imaging has increased substantially the ability to detect causes of seizure disorders, to plan therapy, and to prognosticate the outcome of disorders and therapy. However, MRI must be performed with techniques that will maximize the detection of potentially epileptogenic lesions. Functional imaging is relied on when results from standard diagnostic methods, such as clinical information, electroencephalography, and MRI, are insufficient to localize the seizure focus. Despite the availability of multimodality imaging, the epileptogenic zone is not determined solely by a single imaging modality. Evidence and experience have shown that concordance of results from clinical, electrophysiologic, and

neuroimaging studies is needed to identify the epileptogenic zone and etiology of seizure accurately.[30]

One of the primary steps in evaluating new-onset seizure disorders is to determine whether there is an underlying brain lesion. Common causes of acute seizures are stroke, head trauma, and brain tumor.[31]

The sensitivity of MRI in detecting structural lesions in the brain is unparalleled. In addition to its ability to detect with high sensitivity nearly all types of lesions in patients with epilepsy, it correctly distinguishes between tumors and vascular malformations in 95% of patients.[32] The superiority of MRI to CT was recognized soon after MRI was developed. Studies with early-generation MRI machines showed clinically relevant lesions in nearly 10% of adults who had new-onset epilepsy and in 5% to 20% of patients with chronic focal epilepsy and normal CT scans.[33] Moreover, MRI accurately locates the brain lesion and surrounding structures. Unlike standard CT, MRI displays normal and pathological structures in three dimensions, thus allowing the construction of three dimensional images.

Computed tomography is valuable for the early detection of blood densities, as in subarachnoid hemorrhage or hemorrhagic stroke lesions. Therefore, it is generally the initial brain-imaging procedure performed in neurologic emergencies, especially when artifacts (for example, catheters or ventilation devices) associated with critically ill patients produce less interference than with MRI.[34] Also, CT is sensitive in detecting calcified lesions, which appear as signal void on MRI. Although overall CT is inferior to MRI in

detecting structural lesions, it is an alternative to MRI for patients who cannot undergo MRI because of cardiac pacemakers, large body size, severe claustrophobia, or ferromagnetic objects in the head or neck (for example, aneurysm clips).

Patients who present with first-time seizures should have emergent neuroimaging if (1) a serious structural lesion is suspected, (2) the patient presents with focal seizures, or (3) the patient is older than 40 years.[35] For other situations, neuroimaging may still be needed urgently if the patient cannot be monitored appropriately after seizure occurrence and imaging is essential for planning care. In all such situations, the clinical history and neurologic examination are essential for assessing the probability of an intracranial lesion that requires emergent attention.

The International League Against Epilepsy recommends that MRI be performed in nonemergent situations on all epilepsy patients except for those with idiopathic epilepsy.[34] The League further recommends that MRI be performed if the patient has (1) historical or EEG evidence of a focal onset of seizures at any age, (2) onset of unclassified or apparently generalized seizures in the early years of life or adulthood, (3) evidence of a focal fixed deficit on neurologic or neuropsychological examination, (4) difficulty controlling seizures with first-line antiepileptic drugs, or (5) loss of control of seizures with antiepileptic drugs or a change in pattern of seizures, which may imply a progressive underlying lesion.

COMPUTED TOMOGRAPHY SCAN (CT SCAN)

The introduction of CT scan in 1970s has changed the diagnosis of cerebral lesion in a big way in patients suffering from seizures. A large number of workers have worked on the role of CT scan in evaluation of patients with epilepsy.

In a Canadian study,[36] CT scan was done in a consecutive series of 196 adult epileptics. In the series, overall incidence of abnormal scans was 16% with the highest yield (44%) found in patients with partial seizures. In 25 of 51 cases with abnormal scans a specific lesion amenable to therapy was detected, including 16 neoplasms and five arteriovenous malformations. Other lesions included generalized or focal atrophy, infarcts, calcified lesion of tuberous sclerosis, unexplained calcifications and focal low density or enhancing lesions.

In another European study[37] incidence and CT abnormalities and their correlates with clinical and EEG features were evaluated in a consecutive series of 202 adult patients with newly diagnosed epileptic seizures. Abnormal CT findings were found in 36% of the patients. The abnormalities consisted of brain tumors (17%), atrophic lesions (11%) and other finding (8%) such as arteriovenous malformations.

In a review[38] of the CT findings on 387 patients with new onset seizures after the age of 50 years showed that, cerebral atrophy was seen in 29.2% cases, ischemic lesions in 19.4%, cerebral neoplasm in 5.1% and no abnormality in 45.7% cases.

In an Indian study,[39] results of CT findings in 170 patients who developed seizures showed that the commonest abnormality was a focal ring or disc enhancing lesion (62.3%) followed by calcification (16.9%), cerebral atrophy (8.5%), vascular lesions (6.6%), tumors (3.8%) and congenital hydrocephalus (1.5%).

In a north Indian study[40] conducted on 150 consecutive cases of solitary seizures who reported between 1995 and August 1997, CT scan head was done in 119 patients. Out of the total of 119 CT scans, 91 cases (76.4%) were normal and 28 (23.6%) were abnormal. The abnormalities were disc or ring enhancing lesion in eight (6.7%), calcified or nodular lesion in nine (7.5%) and small cystic lesion in two (1.6%) cases.

Single enhancing lesions visualized on CT scanning are the most common radiological abnormality in Indian patients with new onset seizures.[41]

SINGLE ENHANCING COMPUTERIZED TOMOGRAPHY LESIONS

Neurocysticercosis and tuberculoma both can present with seizure and CT scan picture of single enhancing lesion. Rajshekar V, Chandy MJ[42] based on their experience proposed diagnostic criteria for cysticercus granuloma.

Clinical Criteria

1. Seizures (partial or generalized) as initial symptom.
2. Absent persistent raised intracranial tension (ICT).
3. No progressive neurological deficit.
4. No active systemic disease

Computed tomography criteria

1. Solitary contrast enhancing lesion.
2. Lesion less than or equal to 20 mm in diameter.
3. Absence of severe cerebral edema (no midline shift)

However, none of these features are specific enough, for the diagnosis of neurocysticercosis.

Revised diagnostic criteria of neurocysticercosis (Del Brutto et al)[43]

Absolute

1. Histological demonstration of parasite.
2. CT or MRI showing cystic lesions with scolex.
3. Fundoscopic visualization of parasite.

Major

1. Lesions suggestive of neurocysticercosis on CT or MRI
2. Positive serum EITB.
3. Resolution of cyst after therapy.
4. Spontaneous resolution of single enhancing lesions.

Minor

1. Lesions compatible with neurocysticercosis on CT or MRI.
2. Suggestive clinical features.
3. Positive CSF enzyme linked immunosorbent assay (ELISA).
4. Cysticercosis outside CNS.

Epidemiologic

1. Household contact with Taenia solium infection.
2. Immigration from or living in an endemic area.
3. Travel to an endemic area.

Definite: One absolute; or two major + one minor + one epidemiologic.

Probable: One major + two minor; one major + one minor + one epidemiologic; three minor + one epidemiologic.

METHODOLOGY

The present study was conducted in the Department of Medicine, at KLES Dr Prabhakar Kore Hospital and Medical Research Centre, Belgaum during the period of January 2007 to December 2007.

Study design

This cross-sectional study was conducted on patients above 35 years presenting with seizures for the first time.

Study Period

Over period of one year from 1st Jan 2007 to 31th Dec 2007.

Source of Data

Adult patients aged above 35 years presenting with seizures for the first time to the Department of Medicine or Neurology at KLES Dr Prabhakar Kore Hospital and Medical Research Centre, Belgaum.

Method of collection of data

Sample size

40 adult patients aged above 35 years.

Sampling procedure

80% of average number of similar cases admitted to KLES Dr. Prabhakar Kore Hospital Belgaum over a period of last three years were considered to calculate the sample size.

Selection Criteria

Inclusion Criteria

All the patients presenting with seizures for first time after 35 years of age.

Exclusion Criteria

- Patients with seizures who present before the age of 35 years.
- Patients with known past history of seizures.

Procedure

All cases were evaluated by detailed medical history and physical examination. The study was approved by the Ethical and Research Committee of J. N. Medical College, Belgaum.

All patients aged above 35 years presenting with seizures for the first time to the Department of Medicine or Neuromedicine at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre were screened for the eligibility into the study. After finding the suitability as per inclusion and exclusion criteria they were selected for the study and briefed about the nature of the study and the procedures used and written informed consent was obtained (Annexure–I). The consented patients were enrolled in the present study. Further, descriptive data of the participants like name, age, sex, detailed history, were obtained by interviewing the participants and recorded on predesigned and pretested proforma (Annexure-II). Detailed history, clinical examination, blood investigations, EEG

and cranial imaging were performed. Finally data obtained was systematically tabulated and data analysis was done.

Investigations

- Complete blood count
- Routine urine examination
- Blood sugar
- Blood urea
- Serum creatinine
- Serum calcium and magnesium

Electroencephalography

Electroencephalography was done in all the patients using 32 channel digital EEG machine of Nihon Kohden. Resting awake with hyperventilation and photic stimulation records were taken. EEG was reported as.

Cranial imaging studies

Either MRI or CT scan of brain or both were performed in every patient based on clinical indications.

Magnetic resonance imaging of brain

Magnetic resonance imaging was done in patients using Siemen's Symphony 1.5 T MRI machine. Brain plain imaging with or without contrast was done. Diffusion weighted imaging, T1, T2, FLAIR sequences were taken.

Computed tomography scan

- The patients were scanned using Siemen's single slice spiral CT machine.
- Brain plain with or without contrast scan was done.

Other investigations

Other investigations as necessary were performed.

- Serological tests
    - For syphilis.
    - For HIV detection.
    - Serology for toxoplasma.
    - Demonstration of antibodies to cysticerci in serum.
- LP CSF study
    - Demonstration of antibodies to cysticerci in CSF.
    - CSF PCR for TB.
    - CSF TB IgM antibodies by ELISA.
- ECG, Chest X-Ray, Echocardiography.

Statistical analysis

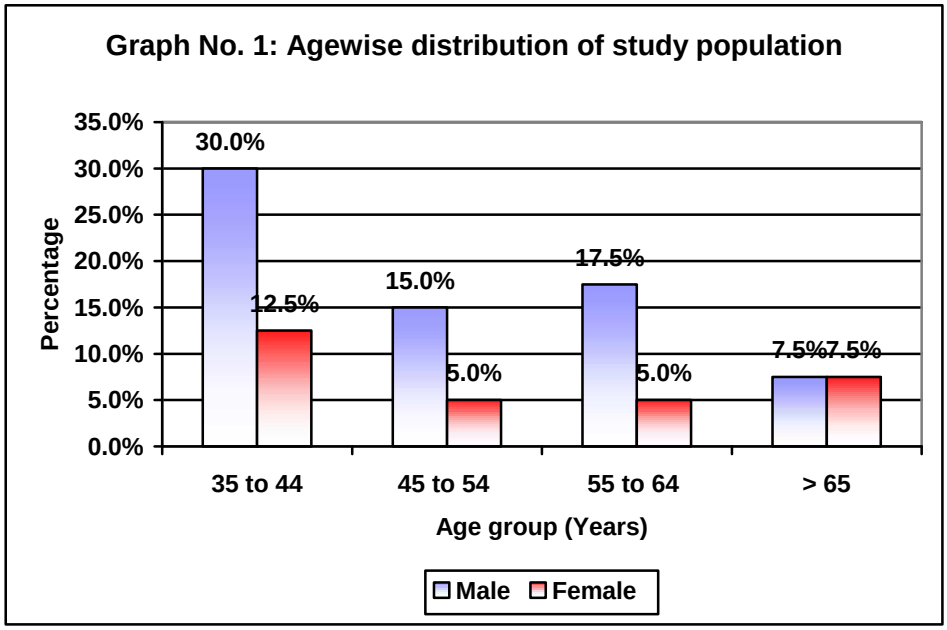
The results were tabulated and the data was analysed using rates, ratios and percentages of different clinical manifestations, EEG, cranial imaging findings, etiologies and diagnosis.

RESULTS

The present study was conducted on 40 patients presenting with seizures for the first time after 35 years of age to the Department of Medicine or Neuromedicine, KLES Dr. Prabhakar Kore Hospital and Medical Research centre, Belgaum. The results obtained are tabulated as below.

Table No. 2: Agewise distribution of study population

Age (years)	Male		Female		Total	
	No.	%	No.	%	No.	%
35 to 44	12	30.00%	05	12.50%	17	42.50%
45 to 54	06	15.00%	02	5.0%	08	20.00%
55 to 64	07	17.50%	02	5.0%	09	22.50%
≥ 65	03	7.50%	03	7.50%	06	15.00%
Total	**28**	**70.00%**	**12**	**30.00%**	**40**	**100.00%**

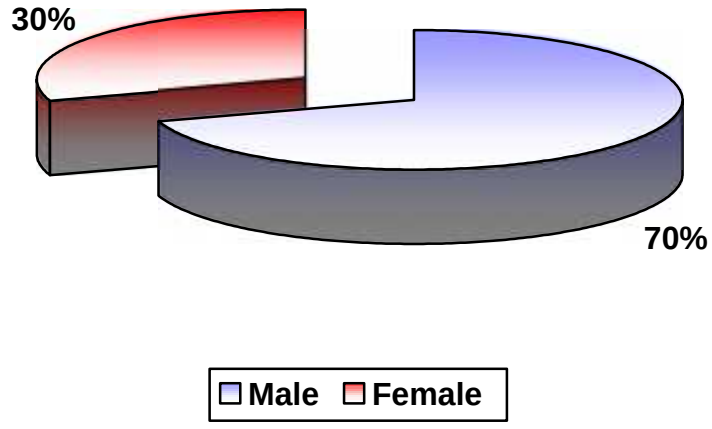


The maximum number of patients were in the age group of 35 to 44 years (42.50%). Number of male patients were more than female patients. The youngest patient was 36 years old and the eldest was 89 years old.

Table No. 3: Sex Distribution of study population

Gender	Number of patients	Percentage
Male	28	70.00%
Female	12	30.00%
Total	**40**	**100.00%**

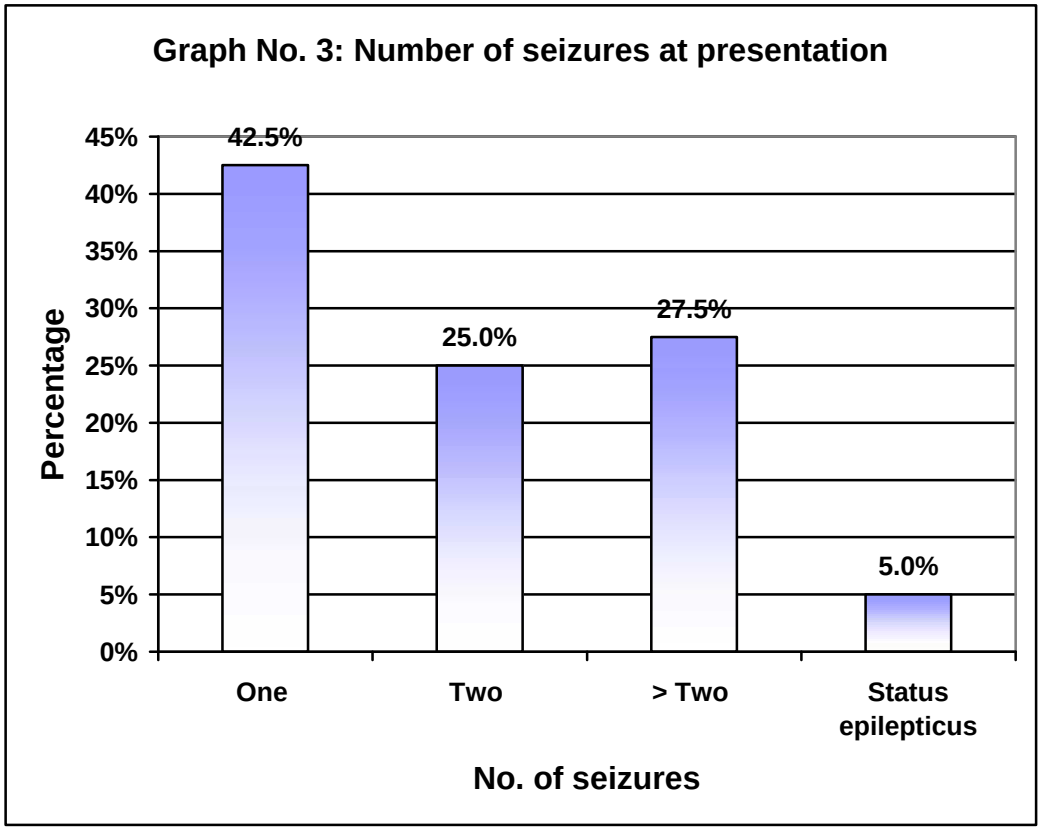
Graph No. 2: Sex Distribution of study Population



In the present study population, out of 40 cases, 28 (70.00%) were males and 12 (30.00%) were females with Male to Female ratio of 2.33 : 1.

Table No 4: Number of seizures at presentation

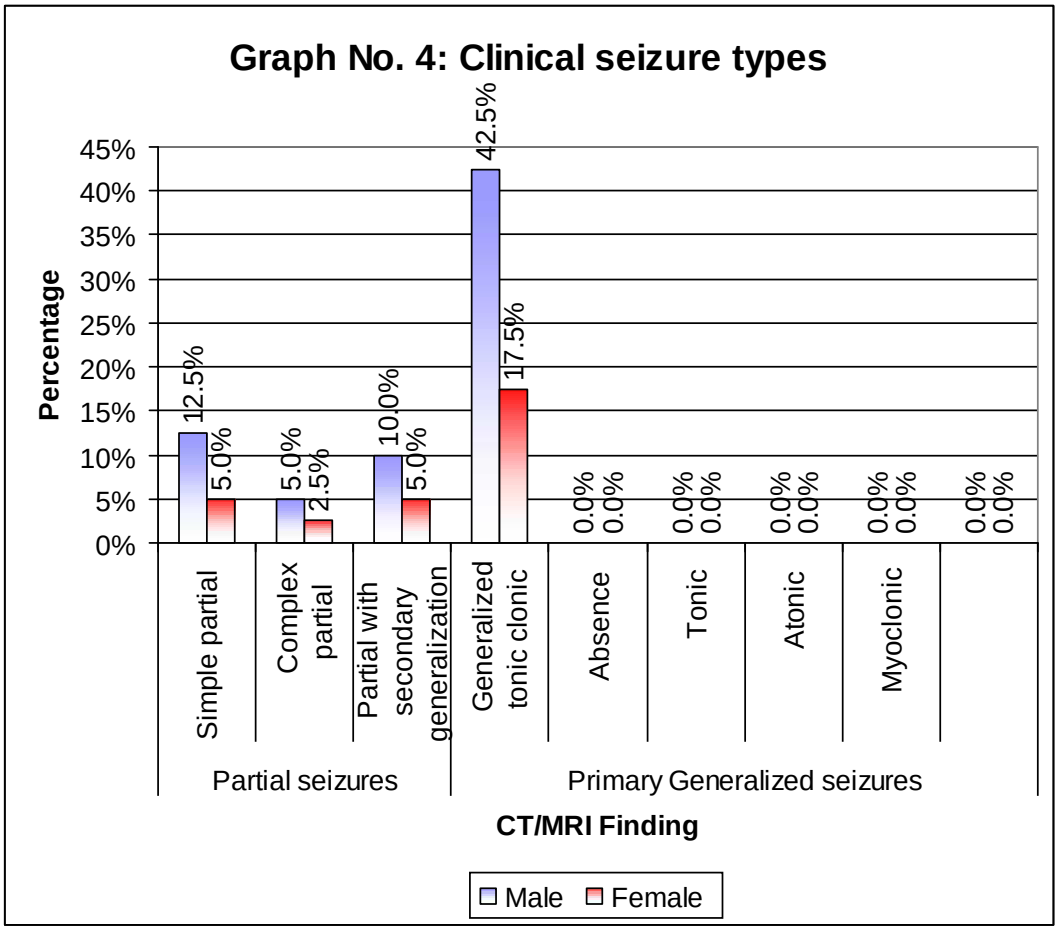
No. of seizures	Number of patients	Percentage
One	17	42.50%
Two	10	25.00%
> Two	11	27.50%
Status epilepticus	02	5.00%



In the present study 17 (42.5%) patients had one seizure, 10 (25.0%) had two seizures, 11 (27.5%) had more than two seizures and in only two (five percent) patients status epilepticus was observed.

Table No. 5: Clinical seizure types (n=40)

Clinical seizure types		Male		Female		Total	
		No.	**%**	**No.**	**%**	**No.**	**%**
Partial Seizures	**Simple partial**	05	12.5%	02	5.0%	07	17.5%
	Complex partial	02	5.0%	01	2.5%	03	7.5%
	Partial with secondary generalization	04	10.0%	02	5.0%	06	15.0%
	Total	**11**	**27.4%**	**05**	**12.5%**	**16**	**40.0%**
Primary generalized seizures	**Tonic clonic (Grandmal)**	17	42.5%	07	17.5%	24	60.0%
	Absence (Petit mal)	00	00.0%	00	00.0%	00	00.0%
	Tonic	00	00.0%	00	00.0%	00	00.0%
	Atonic	00	00.0%	00	00.0%	00	00.0%
	Myoclonic	00	00.0%	00	00.0%	00	00.0%
	Total	**17**	**42.5%**	**07**	**17.5%**	**24**	**60.0%**



Among the 40 patients, 24 (60.0%) had generalized tonic clonic seizures, seven (17.5%) had simple partial seizures, six (15.0%) had partial seizure with secondary generalization and three (7.5%) had complex partial seizures. Among the seizure types generalized tonic clonic seizure was the most common type followed by simple partial seizures.

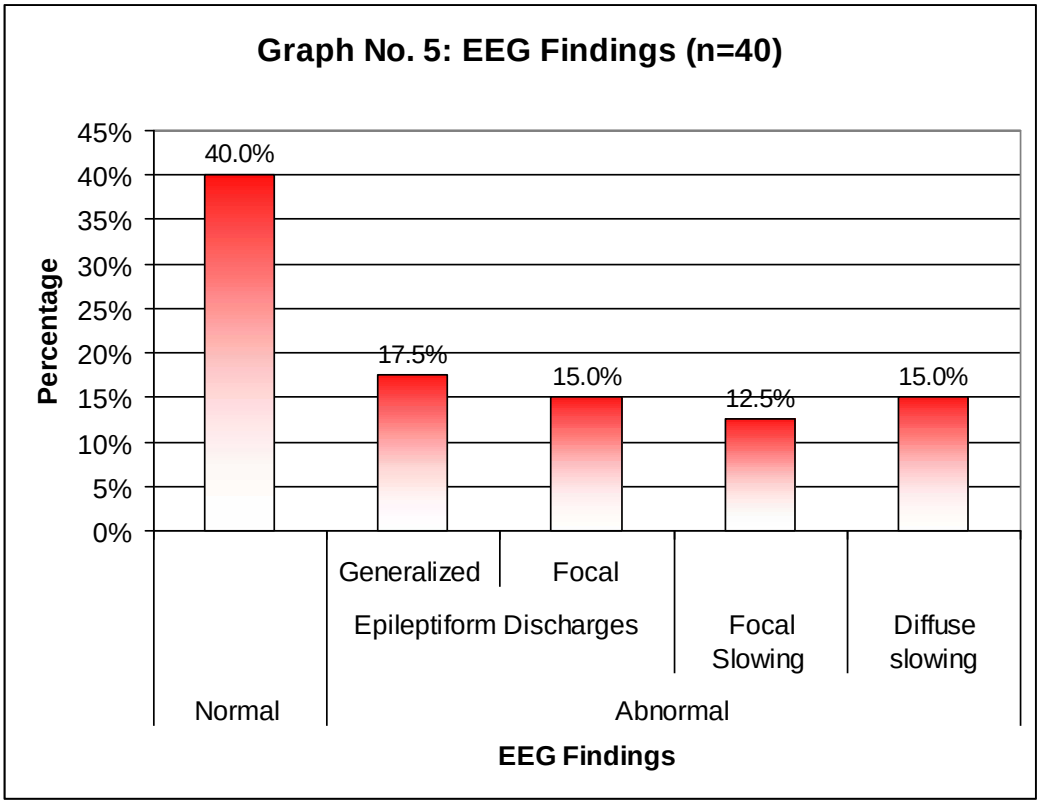
Table No. 6: Clinical features observed in 40 patients

Clinical features	Number of patients	Percentage
Focal neurodeficits	27	67.5%
Altered sensorium	18	45.0%
Feature of raised ICT	11	27.5%
Urinary incontinence during seizure	10	25.0%
Hypertension	08	20.0%
Type 2 Diabetes mellitus	07	17.5%
Ictal cry	06	15.0%
Tongue bite	05	12.5%
Head trauma	02	5.0%
Cognitive dysfunction	02	5.0%
Features of neuroinfection	02	5.0%

In majority of patients (67.5%) focal neurodeficits were found, followed by altered sensorium (45.0%), features of raised intracranial tension (27.5%) and urinary incontinence during seizure (25.0%). Other clinical features observed were as depicted in the table.

Table No 7: EEG findings

EEG (n=40)			Number of patients	Percentage
Normal			**16**	**40.0%**
Abnormal	**Epileptiform Discharges**	**Generalized**	07	17.5%
		Focal	06	15.0%
		Total	**13**	**32.5%**
	Focal slowing		05	12.5%
	Diffuse slowing		06	15.0%
	Total		**24**	**60.0%**
Total			**40**	**100.0%**



Overall EEG was abnormal in 24 (60.0%) patients. It showed epileptiform discharges which were generalized in seven (17.5%) and focal in six (15.0%) patients. It showed focal slowing in five (12.5%) patients and diffuse slowing in six (15.0%) patients. Overall generalized epileptiform discharges were the most common EEG abnormality.

Table No 8: EEG in generalized seizures

EEG (n=24)			**Cases**	**Percentage**
Normal			10	41.66%
Abnormal	**Epileptiform Discharges**	**Generalized**	08	33.33%
		Focal	04	16.66%
		Total	12	50.0%
	Focal slowing		01	4.16%
	Diffuse slowing		01	4.16%
	Total		14	38.33%
Total			24	100.00%

Out of 24 patients in generalized seizure group, EEG was abnormal in 14 (58.34%) patients and normal in 10 (41.66%) patients. It showed epileptiform discharges which were generalized in eight (33.33%) patients and focal in four (16.66%) patients. It showed focal slowing in one (4.16%) patient and diffuses slowing in one (4.16%) patient. Generalized epileptiform discharges were the most common EEG abnormality.

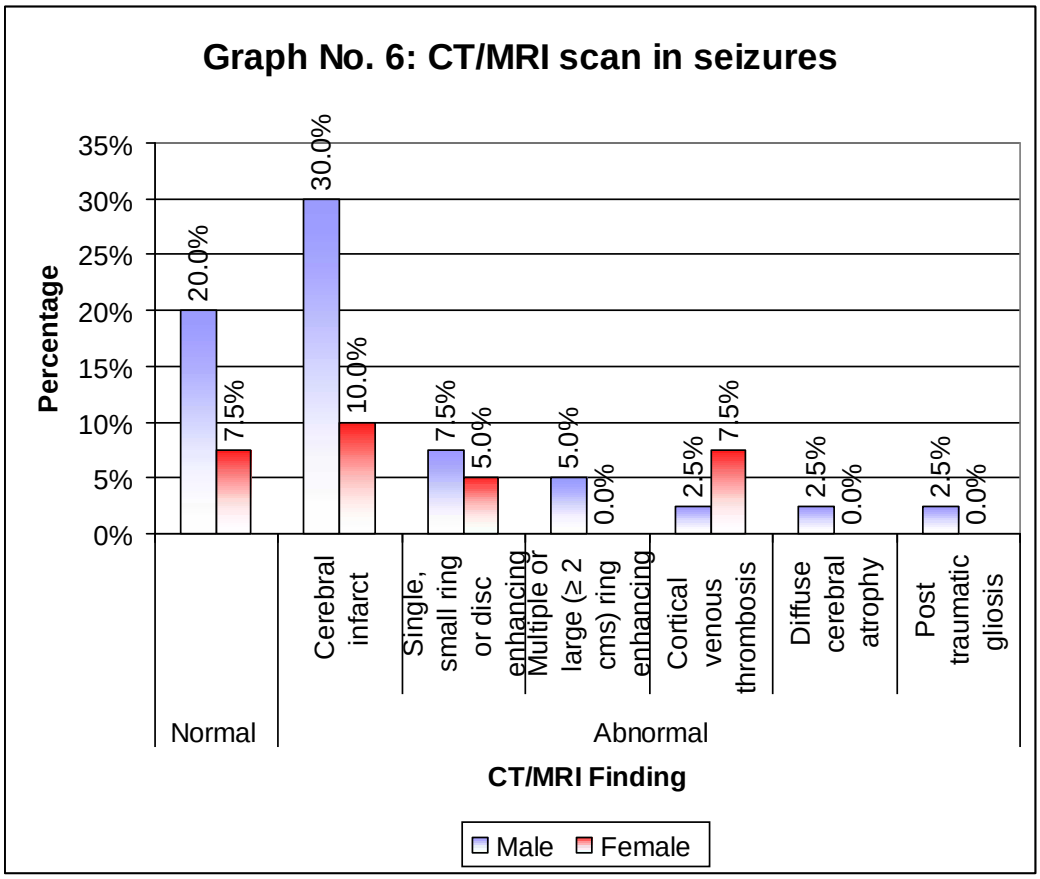
Table No. 9: EEG in partial seizures

EEG (n=16)			Cases	Percentage
Normal			**06**	**37.50%**
Abnormal	**Epileptiform Discharges**	**Generalized**	03	18.75%
		Focal	05	31.25%
		Total	**08**	**50.00%**
	Focal slowing		01	6.25%
	Diffuse slowing		01	6.25%
	Total		**10**	**62.50%**
Total			**16**	**100.00%**

Out of 16 patients with partial seizures, EEG was abnormal in 10 (62.50%) patients and normal in six (37.50%) patients. It showed focal epileptiform discharges in five (31.25%) patients, generalized epileptiform discharges in three (18.75%) patients, focal slowing in one (6.25%) patients and diffuse slowing in one (6.25%) pateint. Overall epileptiform discharges were the most common EEG abnormality.

Table No. 10: CT/MRI scan in seizures

CT/MRI Scan		Male		Female		Total	
		No.	**%**	**No.**	**%**	**No.**	**%**
Normal		**08**	**20.00%**	**03**	**7.50%**	**11**	**27.50%**
Abnormal	**Cerebral infarct**	12	30.00%	04	10.00%	16	40.00%
	Single, small ring or disc enhancing lesion (≤ 2 cms)	03	7.50%	02	5.00%	05	12.50%
	Multiple or large (≥ 2 cms) ring enhancing lesions	02	5.00%	00	0.00%	02	5.00%
	Cortical venous thrombosis	01	2.50%	03	7.50%	04	10.00%
	Diffuse cerebral atrophy	01	2.50%	00	0.00%	01	2.50%
	Post traumatic gliosis	01	2.50%	00	0.00%	01	2.50%
	Total	**20**	**50.00%**	**09**	**22.50%**	**29**	**72.50%**
Total		**28**	**70.00%**	**12**	**30.00%**	**40**	**100.00%**



CT/MRI scan was abnormal in 29 (72.5%) patients. It showed cerebral infarct in 16 (40.0%) patients, single, small ring or disc enhancing lesions (less than or equal to two cm) in five (12.5%) patients, multiple or large ring enhancing lesions (more than 2 cm) in two (5.00%), cortical venous thrombosis in four (10.0%) and diffuse cerebral atrophy in one (2.50%) patient and post traumatic gliosis in one (2.5%) patient. Overall cerebral infarct was the most common abnormality identified.

Table No. 11: CT/MRI Scan in generalized seizures

CT/MRI Scan (n=16)		Total	
		Number	**Percentage**
Normal		09	37.50%
Total		**09**	**37.50%**
Abnormal	**Cerebral infarct**	09	37.50%
	Single, small ring or disc enhancing lesion (≤ 2 cms)	02	8.33%
	Multiple and large (> 2 cms) ring enhancing region	00	0.00%
	Cortical venous thrombosis	02	8.33%
	Diffuse cerebral atrophy	01	4.16%
	Post traumatic gliosis	01	4.16%
	Total	**15**	**62.50%**
Total		**24**	**100.00%**

Cranial imaging was abnormal in 15 (62.5%) and normal in nine (37.5%) patients of generalized seizures. The abnormalities were cerebral infarct in nine (37.5%) patients, single small ring or disc enhancing lesion (≤ 2 cms) in two (8.33%) patients, cortical venous thrombosis in two (8.33%) patients, diffuse cerebral atrophy in one (4.16%) patient and post traumatic gliosis in one (4.16%) patient.

Table No. 12: CT/MRI Scan in partial seizures

CT/MRI Scan (n=16)		Total	
		Number	**Percentage**
Normal		**02**	**12.50%**
Abnormal	**Cerebral infarction**	07	43.75%
	Single, small ring or disc enhancing lesion (≤ 2 cms)	03	18.75%
	Multiple or large (≥ 2 cms) ring enhancing lesion	02	12.50%
	Cortical venous thrombosis	02	12.50%
	Diffuse cerebral atrophy	00	0.00%
	Post traumatic gliosis	00	0.00%
	Total	**14**	**87.50%**
Total		**16**	**100.00%**

Out of 16 patients in partial seizures group, cranial imaging was abnormal in 14 (7.5%) and normal two (12.5%) patients. The abnormalities were cerebral infarct (43.75%), single small ring and disc enhancing lesion less than or equal to two cm (18.75%), multiple and large more than two cm ring enhacing lesion (12.5%) and cortical venous thrombosis (12.5%).

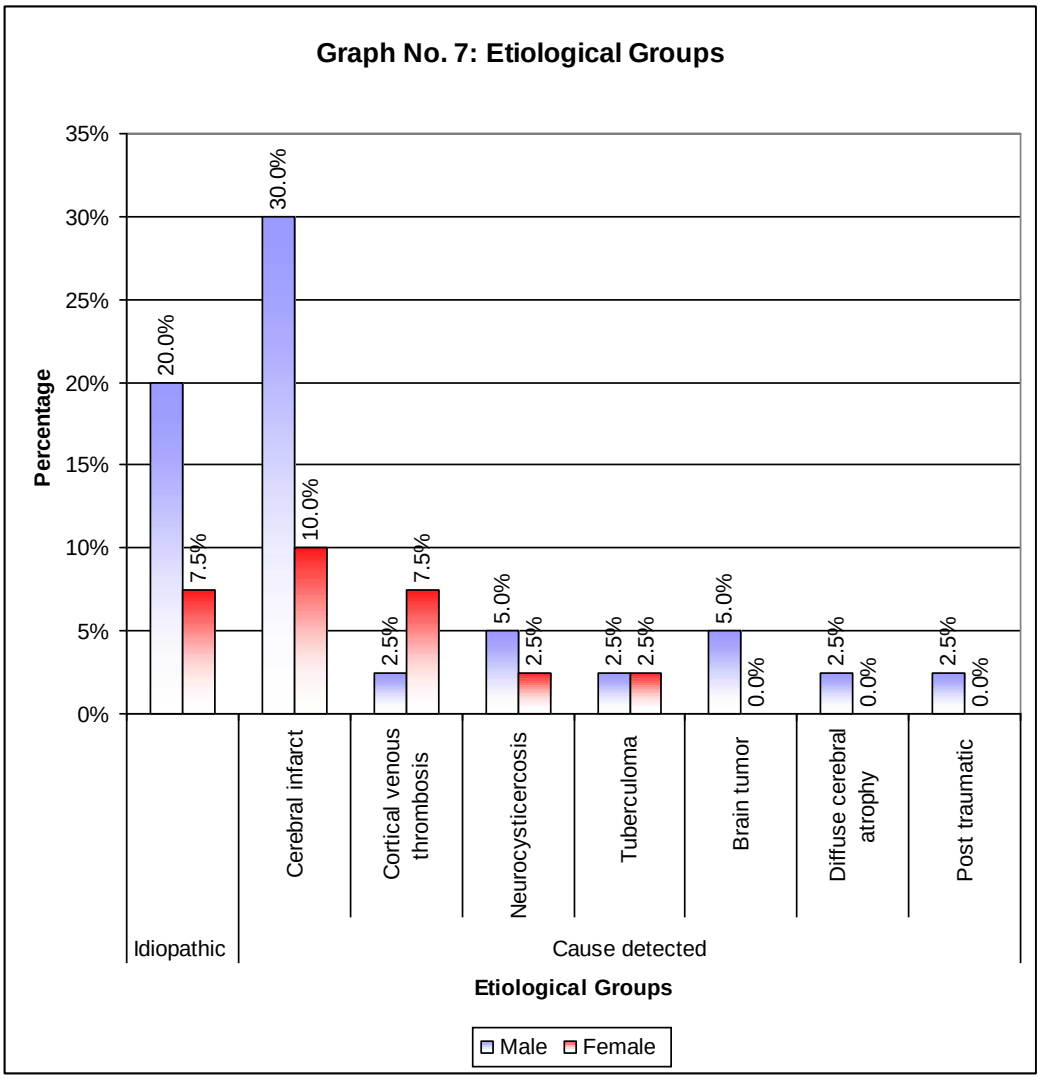
Table No. 13: EEG and imaging (CT/MRI) correlation in seizures

Scan	Normal EEG		Abnormal EEG		Total	
	No.	%	No	%	No	%
Normal CT/MRI Scan	07	17.50%	04	10.00%	11	27.50%
Abnormal CT/MRI Scan	09	22.50%	20	50.00%	29	72.50%
Total	16	40.00%	24	60.00%	40	100.00%

Majority of patients (72.5%) had abnormal CT/MRI brain findings. Out of these, 50.0% had abnormal EEG and 22.5% had normal EEG. Out of 11 (27.5%) patients with normal CT/MRI brain findings, seven (17.5%) had normal EEG and four (10.0%) had abnormal EEG.

Table No. 14: Etiological groups

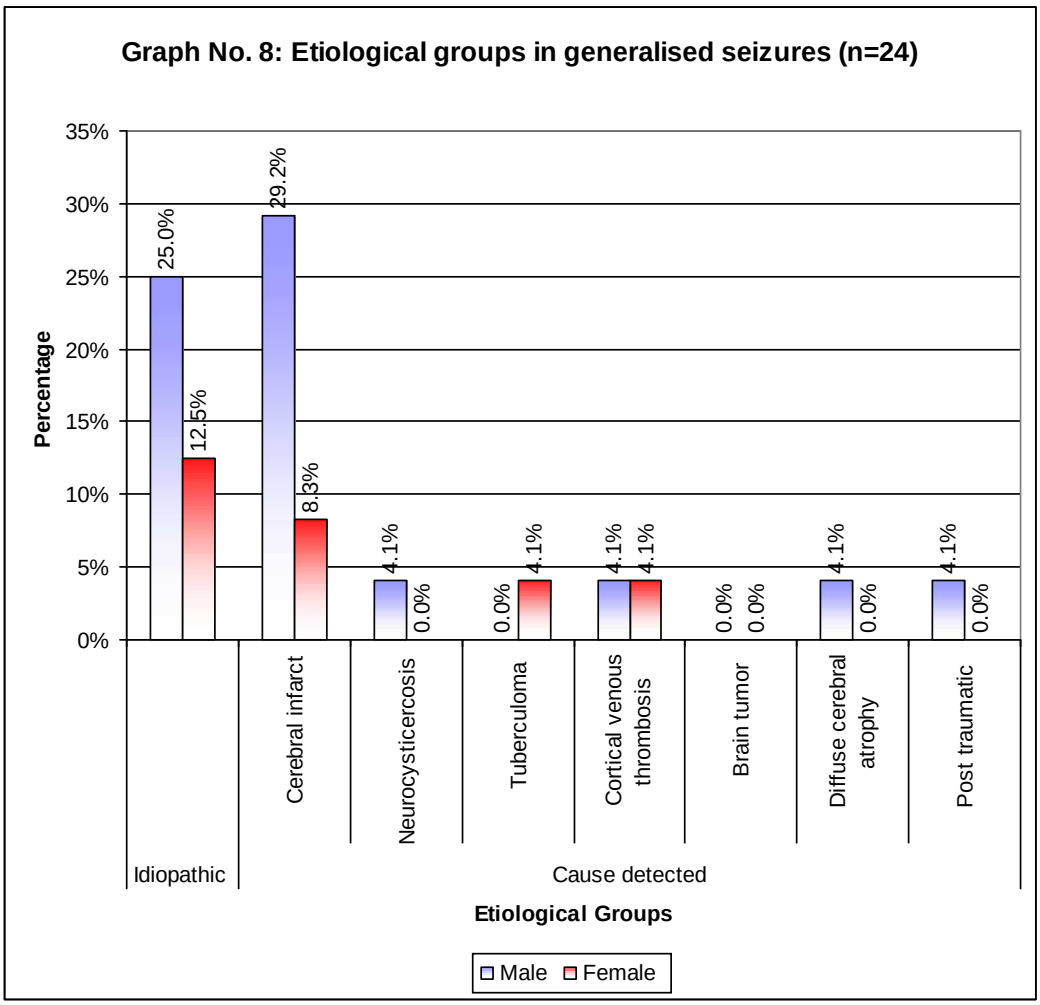
Etiological group (n=24)		Males		Females		Total	
		No.	%	No	%	No	%
Idiopathic		08	20.00%	03	7.50%	11	27.50%
Cause detected	**Cerebral infarct**	12	30.00%	04	10.00%	16	40.00%
	Cortical venous thrombosis	01	2.50%	03	7.50%	04	10.0%
	Neurocysticercosis	02	5.00%	01	2.50%	03	7.50%
	Tuberculoma	01	2.50%	00	2.50%	02	5.00%
	Brain tumor	02	5.00%	00	0.00%	02	5.00%
	Diffuse cerebral atrophy	01	2.50%	00	0.00%	01	2.50%
	Post traumatic	01	2.50%	00	0.00%	01	2.50%
	Total	20	50.00%	09	22.50%	29	72.50%
Total		28	70.00%	12	30.00%	40	100.00%



In majority of patients 29 (72.5%), the etiology for seizures was identified. However in 11 (27.5%) patients cause could not be found. Among 29 (72.5%) patients, 16 (40.0%) had cerebral infarct, four (10.0%) had CVT, three (7.5%) had neurocysticercosis, two (5.0%) had tuberculoma, one (2.5%) each had brain tumor, diffuse cerebral atrophy and post traumatic gliosis.

Table No. 15: Etiological groups in generalized seizures

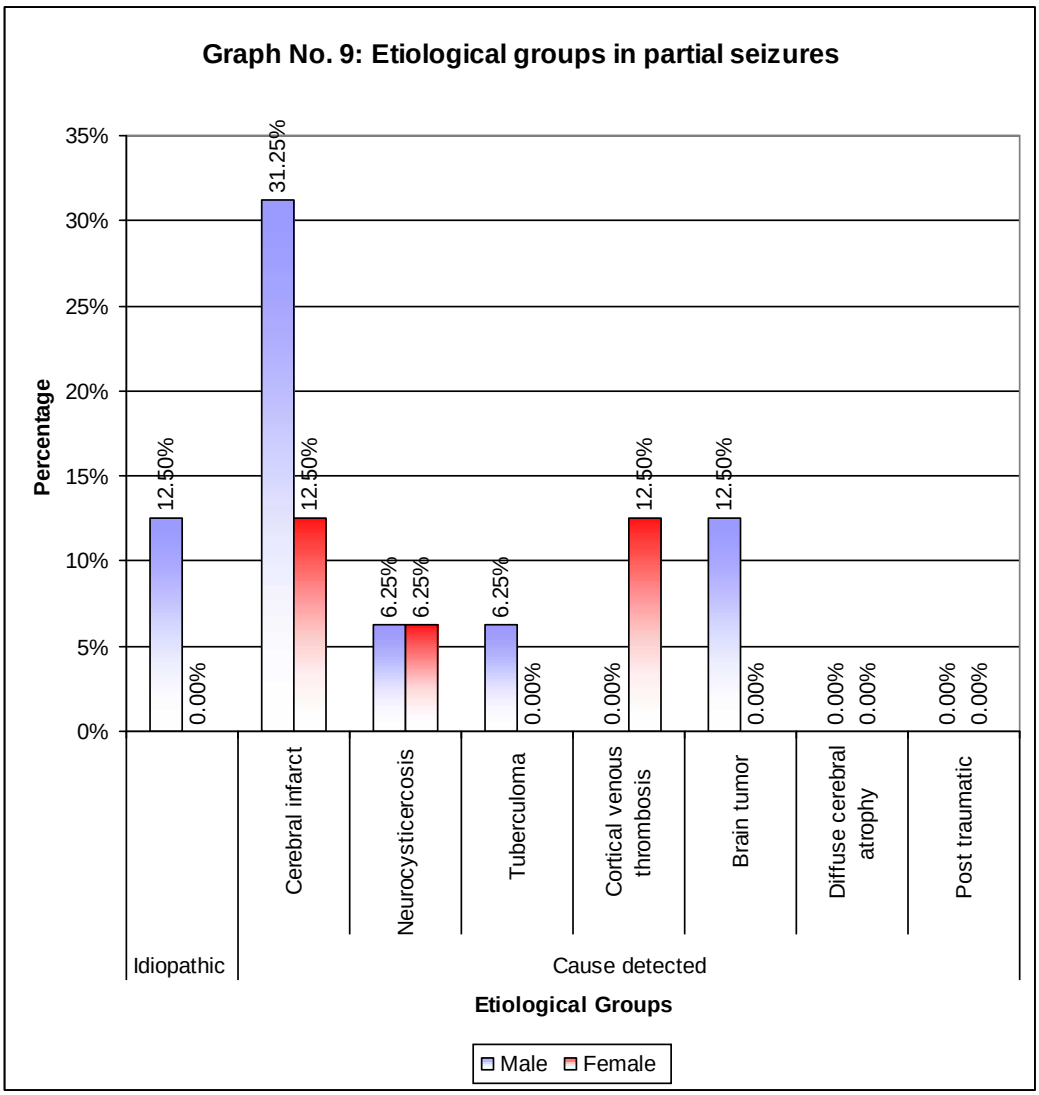
Etiological group (n=24)		Males		Females		Total	
		No.	%	No	%	No	%
Idiopathic		06	25.00%	03	12.50%	09	37.50%
Cause detected	**Cerebral infarct**	07	29.16%	02	8.30%	09	37.50%
	Cortical venous thrombosis	01	4.10%	01	4.10%	02	8.30%
	Neurocysticercosis	01	4.10%	00	0.00%	01	4.16%
	Tuberculoma	00	0.00%	01	4.10%	01	4.10%
	Brain tumor	00	0.00%	00	0.00%	00	0.00%
	Diffuse cerebral atrophy	01	4.10%	00	0.00%	01	4.10%
	Post traumatic	01	4.10%	00	0.00%	01	4.16%
	Total	10	41.66%	05	20.84%	15	62.50%
Total		16	66.66%	08	33.34%	24	100.00%



Out of 24 patients in generalized seizure group, cause was not ascertained in nine (37.5%) patients. In the remaining 15 (62.5%) patients, the causes were cerebral infarct in nine (37.5%) patients, cortical venous thrombosis in two (8.3%) patients, neurocysticercosis, tuberculoma, diffuse cerebral atrophy and post traumatic gliosis in one (4.1%) each of patients.

Table No. 16: Etiological groups in partial seizures

Etiological group (n=16)		Males		Females		Total	
		No.	%	No	%	No	%
Idiopathic		02	12.50%	00	0.00%	02	12.50%
Cause detected	**Cerebral infarct**	05	31.25%	02	12.50%	09	56.20%
	Cortical venous thrombosis	00	0.00%	02	12.50%	02	12.50%
	Neurocysticercosis	01	6.25%	01	6.25%	02	12.50%
	Tuberculoma	01	6.25%	00	0.00%	01	6.25%
	Brain tumor	02	12.50%	00	0.00%	02	12.50%
	Diffuse cerebral atrophy	00	0.00%	00	0.00%	00	0.00%
	Post traumatic	00	0.00%	00	0.00%	00	0.00%
	Total	09	56.25%	05	31.25%	14	87.50%
Total		11	68.75%	05	31.25%	16	100.00%



In 16 patients of partial seizures, the cause was found in 14 (87.50%) patients. Of which, majority were cerebral infarcts (43.75%) followed by neurocysticercosis (12.50%), cortical venous thrombosis (12.50%), brain tumor (12.50%) and tuberculoma (6.25%). In two (12.50%) patients the cause was not found.

Table No. 17: Type of seizure in various etiological factors

Clinical seizure types		Number	Percentage
Simple partial seizure	**Infarct**	04	10.0%
	Cortical venous thrombosis	02	5.0%
	Tumor	01	2.5%
	Tuberculoma	01	2.5%
Complex partial seizure	**Infarct**	02	5.0%
	Brain tumor	01	2.5%
Partial seizure with secondary generalization	**Cerebral infarct**	03	7.5%
	Neurocysticercosis	02	5.0%
	Idiopathic	01	2.5%
Generalised tonic clonic seizure	**Idiopathic**	10	25.0%
	Cerebral infarct	07	17.5%
	Cortical venous thrombosis	02	5.0%
	Tuberculoma	01	2.5%
	Neurocysticercosis	01	2.5%
	Diffuse cerebral atrophy	01	2.5%
	Post traumatic	01	2.5%^

Among patients with simple partial seizure, cerebral infarct (10.0%) was the commonest etiology followed by cortical venous thrombosis (5.0%).

In complex partial seizure group cerebral infarct (5.0%) was commonest etiology followed by brain tumor (2.5%). In partial seizure with secondary

generalization, cerebral infarct (7.5%) was the commonest etiology followed by neurocysticercosis (5.0%).

In generalized tonic clonic seizure group, idiopathic (25.0%) etiology was the commonest followed by cerebral infarct (17.5%).

Table No. 18: Etiological factors of seizures in various age groups

Etiological group		35-44 Years		45-54 Years		55-64 Years		≥ 65 Years		Total	
		No.	%	No	No	%	No	%	%	No	%
Idiopathic		05	12.5	04	10.0	01	2.5	01	2.5	11	27.5
Causes detected	**Cerebral infarct**	03	7.5	02	5.0	07	17.5	04	10.0	16	40.0
	Cortical venous thrombosis	04	10.0	00	0.0	00	0.0	00	0.0	04	10.0
	Neurocysti cercocis	02	5.0	01	2.5	00	0.0	00	0.0	03	7.5
	Tuberculoma	02	5.0	00	0.0	00	0.0	00	0.0	02	5.0
	Brain tumor	01	2.5	00	0.0	01	2.5	00	0.0	02	5.0
	Diffuse cerebral atrophy	00	0.0	00	0.0	00	0.0	01	2.5	01	2.5
	Post traumatic	00	0.0	01	2.5	00	0.0	00	0.0	01	2.5
	Total	12	30.0	04	10.0	08	20.0	05	12.5	29	72.5
Total		17	42.5	08	20.0	09	22.5	06	15.0	40	100.0

The most common etiology in the age group 35 to 54 years was idiopathic (12.5%). Above 55 years cerebral infarct was the commonest etiology.

Table No. 19: Clinical features according to etiologies

Clinical features		Number	Percentage
No neurodeficits	**Idiopathic**	10	25.0%
	Cerebral infarct	08	20.0%
	Neurocysticercosis	02	5.0%
	Tuberculoma	01	2.5%
	Brain tumor	01	2.5%
Raised ICT	**Cerebral infarct**	07	17.5%
	CVT	01	2.5%
	Tuberculoma	01	2.5%
	Neurocysticercosis	01	2.5%
	Idiopathic	01	2.5%
Hemiparesis	**Cerebral infarct**	06	15.0%
	CVT	02	5.0%
Facial nerve palsy	**Cerebral infarct**	05	12.5%
	CVT	02	5.0%
Visual field defects	**Cerebral infarct**	02	5.0%
	CVT	02	5.0%
Brocas aphasia	**Cerebral infarct**	04	10.0%
Monoparesis	**Cerebral infarct**	01	2.5%
	Neurocysticercosis	01	2.5%
	Brain tumor	01	2.5%
Cognitive dysfunction	**Cerebral infarct**	01	2.5%
	Diffuse cerebral atrophy	01	2.5%
Quadriparesis	**CVT**	01	2.5%

Among the study population 22 (55%) patients had no neurodeficits. Features of raised ICT was present in 11 (27.5%) patients, facial nerve palsy in seven (17.5%) patients, visual field defects and Brocas aplasia in four (10.0%) each of patients, monoparesis in three (7.5%) patients, cognitive dysfunction in two (5.0%) patients and quadriparesis in one (2.5%) patient.

DISCUSSION

In the present study of 40 patients it was observed that unprovoked seizures were more common in males (70%) as compared to females (30%). This is in consistence with international studies all of which report a male preponderance. Age-standardized rates after correction for heterogenecity due to inter study variation, in a meta analysis in India, have revealed prevalence rate per 1000 as follows: overall 5.59, males 6.05, females 5.18 which also shows male preponderance.[17]

In the present study, 60% of patients had generalized seizures whereas 40% had partial seizures, among which 17.5% had simple partial, 7.5% had complex partial and 15% had partial seizure with secondary generalization. Above the age of 65 years, 62% of patients had partial seizure. In a study[17] of new cases of epilepsy presenting before the age of 40 years, 50% have seizures of partial origin and 50% of generalized origin. After 40 years the proportion of partial seizures increases and reaches upto 75% by the age of 75 years. In another study in South India[44] involving 154 patients, 55% had generalized seizures and 45% had partial seizures. These figures are consistent with the present study.

In present study, 22 (55%) patients had no neurodeficits, 11(27.5%) had features of raised ICT, eight (20%) had hemiparesis, seven (17.5%) had facial nerve palsy, four (10%) had visual field defects, four (10%) had Brocas aphasia, three (7.5%) had monoparesis, two (5%) had cognitive dysfunction and one (2.5%) had quadriparesis. In a south Indian study[45] involving 23 patients with late onset seizure, 9(39.1%) had no neurodeficits at the time of presentation, six

(29.1%) had hemiparesis, four (17.4%) had monoparesis, four (17.4%) had facial nerve palsy and one (4.3%) had aphasia. Higher incidence of neurodeficits in this study as compared to our study was due to older study population with greater proportion of stroke patients.

In the present study EEG was abnormal in 24 (60%) patients. It was abnormal in 58.33% of patients with generalized seizures. In this group, epileptiform discharges were generalized in 33.33% of patients and focal in 16.66% of patients. 4.16% of patients had focal slowing where it correlated with underlying structural lesion in the form of brain tumor. 4.16% of patients had diffuse slowing where in there was diffuse cerebral atrophy.

EEG was abnormal in 62.5% of patients with partial seizures. In this group, 31.25% had focal and 18.75% had generalized epileptiform activity. 6.25% patients had focal slowing and another 6.25% had diffuse slowing which correlated with underlying structural lesion.

In a study[46] involving 91 patients with late onset seizures, EEG was taken within 48 hours of the seizures. Abnormal EEGs were obtained in 69% of patients. Epileptiform activity was present in 21% of patients (10% focal, nine percent generalized, two percent focal and generalized), slowing in 59% (21% focal, 31% generalized, seven percent focal and generalized) and both epileptiform activity and slowing in 10%.

In another study,[47] 103 patients with newly diagnosed seizures were studied with EEG. Epileptiform activity was recorded in 18% of the patients and was more common in partial than generalized seizures.

In a study in India[40] involving 150 patients of late onset seizures, EEG was done in all patients and was abnormal in 22% of patients.

Another Indian study[48] involving 52 patients found an abnormal EEG in 73% and 76% of patients with partial and generalized seizures respectively.

A study[49] conducted on 89 patients with epileptic seizure disorder showed that EEG was abnormal in 89% of patients.

An European study[50] involving 130 patients with late onset seizures found EEG to be abnormal in 84.6% patients with 49.2% having generalized epileptiform discharges and 24.6% having focal discharges.

As compared to our study there is a subtle variation in the EEG findings observed in various other studies quoted. This is because of smaller sample size (40 patients) in the present study.

In the present study cranial imaging was found to be abnormal in 72.5% of patients. It was abnormal in 62.5% of generalized seizures and 87.5% of partial seizures. The most common etiology in age group 35 to 55 years was idiopathic (12.5%). Above 55 years cerebral infarct was the commonest etiology. Other cranial imaging findings were; ring or disc enhancing lesions in seven (7.5%) patients, cortical venous sinus thrombosis in four (10.0%), diffuse cerebral atrophy in one (2.5%) and post traumatic gliosis in one (2.5%) patients.

In a European study,[37] incidence and CT abnormalities and their correlates with clinical and EEG features were evaluated in a consecutive series of 202 adult patients with newly diagnosed epileptic seizures. Abnormal CT

findings were found in 36% of patients. The abnormalities consisted of brain tumors (17%) atrophic lesions (11%) and other findings (8%) such as arteriovenous malformations.

Another study in Europe[50] in 2005 involving 130 patients showed cerebrovascular disease (50.8%) as commonest cause of seizure followed by idiopathic (22.3%), head trauma (13.1%) and brain tumor (10.7%).

In another study in North India[39] involving 170 patients the commonest abnormality in cranial imaging was a focal ring or disc enhancing lesion in 66 (62.3%) patients followed by calcification in 18 (16.9%), cerebral atrophy in nine (8.5%), vascular lesions in seven (6.6%) and brain tumor in four (3.8%) patients.

In another study in India[40] involving 150 patients of late onset seizures, CT scan brain was done in 119 patients. Out of that 91 (76.4%) were normal and 28 (23.6%) were abnormal. The abnormalities were disc or ring enhancing lesions in eight (6.7%), calcified and nodular lesions in nine (7.5%) and small cystic lesion in two (1.7%) patients.

In an European study[51] involving 99 patients of late onset seizures, abnormal CT / MRI scan of brain was observed in 62.5% of patients with partial seizures and 34% of patients with generalized seizures.

In the present study the higher incidence of structural abnormality in partial seizures (87.5%) correlated with other studies.[49,50] Higher percentage of idiopathic etiology in generalized seizures (37.5%) as compared to partial seizures (12.5%) also correlated with other studies.[49,50]

Cerebral infarct was the most common etiology of seizures in patients above 55 years which correlated with other studies.[50,52]

Higher incidence of cerebral infarct in the present study was not consistent with other Indian studies,[53,54,55] which was due to younger age of inclusion of study population in their studies with lesser incidence of stroke.

Other variations in etiological factors could be explained by smaller sample size in the present study as compared to other studies.

In the present study the type of seizure and number of seizures did not correlate with any particular etiological factor which was in accordance with other studies.[52,56]

In the present study it was observed that the commonest etiology for seizure in the age group between 35 to 54 years was idiopathic. After the age of 55 years the commonest etiology was cerebral infarct. This observation was comparable to other studies.[50,57]

The present study made an attempt of determining various neurodeficits in different etiological factors and found following observation. No neurodeficits were found in most patients (55%) and most of these cases were idiopathic seizures (25%) followed by cerebral infarct (20%), neurocysticercosis (5%), brain tumor (2.5%) and tuberculoma (2.5%).

The absence of neurodeficits in cases with underlying structural lesions suggest the lesion was either smaller in size or located in non localizing area of the brain.

CONCLUSION

Among the patients presenting with seizures 60% had generalized seizures and 40% had partial seizures.

Majority of patients presenting with seizures who had focal neurodeficits had organic lesion in the brain.

EEG was not very useful in the evaluation of seizures. However abnormal EEG helped to confirm the diagnosis of seizures and when it showed focal abnormalities gave a clue to underlying structural abnormality.

Overall cranial imaging was abnormal in 72.5% of patients, 62.5% of generalized seizure group and 87.5% of partial seizure group patients. Cranial imaging was reliable in revealing structural abnormalities and was helpful in establishing the diagnosis in majority of cases.

Proportion of patients with idiopathic etiology was higher in generalized seizure group as compared to partial seizure group and proportion of patients with structural brain lesion was higher in partial seizure group.

The commonest etiology of seizure in the age group between 35 to 54 years was idiopathic and after the age of 55 years the commonest etiology was cerebral infarct.

Number and type of seizures was not useful in predicting the underlying etiology.

In patients of seizures with organic brain lesion presenting without neurodeficits, the lesion was most likely to be small or located in non localizing area of brain.

SUMMARY

The present study was conducted to know the various clinical manifestations and etiological factors of late onset seizures. This study was conducted on 40 patients presenting with seizures for the first time after the age of 35 years to the Department of Medicine and Neuromedicine, KLES Dr Prabhakar Kore Hospital and Medical Research Centre, Belgaum during the period of January 2007 to December 2007.

Results of the present study showed that, among patients presenting with unprovoked seizures, generalized seizures were seen in 60% of cases and partial seizures in 40% of cases. EEG was not very useful in evaluation of seizures. However abnormal EEG helped to confirm the diagnosis of seizures and when it showed focal abnormality gave a clue to underlying structural abnormality. Cranial imaging was reliable in detecting structural brain abnormality. It was abnormal in majority of patients with both partial and generalized seizures. Cerebral infarct was the most common structural abnormality identified in both partial and generalized seizure group and equal number of patients in generalized seizure group had normal cranial imaging. Patients with partial seizure had a higher incidence of structural brain lesion as compared to those with generalized seizures. In patients with structural lesion on neuroimaging, who had no neurodeficits, the lesion was most likely to be small or located in non localizing area of the brain.

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ANNEXURE I – CONSENT FORM

Mr./Mrs._____ we are requesting you to enroll yourself in study titled **"A CLINICAL AND ETIOLOGICAL STUDY OF LATE ONSET SEIZURES- A CROSS SECTIONAL STUDY AT KLES HOSPITAL AND MRC "** conducted by **DR MANJUNATH R DESAI**, postgraduate student in MD GENERAL MEDICINE under the guidance of **DR VIJAYAKUMAR G. SOMANNAVAR MD** at J. N. Medical College, Belgaum.

You have been requested to participate in research because your profile matches with the study group. Adults (>35 years) who present for the first time with seizures (fits) are included in the study group. During the study you will be asked some questions and you are supposed to answer to the best of your knowledge.

Your participation in the research is absolutely voluntary. Your decision to participate in the study or otherwise will not affect your relationship with J.N.M.C. If you decide not participate you are free to withdraw at any time.

The purpose of research is to find the clinical presentation and various causative factors for late adult onset seizures (fits).

PROCEDURE INVOLVED:

Electroencephalography and CT scan/MRI of brain, which are not invasive procedures.

RISKS AND BENEFITS:

There are no risks involved and benefits are many. The study helps to identify various clinical features of the disease and identify different causative

factors, avoidance of which prevents future recurrence of fits. The results deduced at the end of study will help all similar patients admitted in the hospital.

ALTERNATIVES:

Even if you decline to participate, there will not be any change in the line of your management or the relationship with your doctor. You will be told about all the new information that may affect your decision to participate in the study.

PRIVACY AND CONFIDENTIALITY:

The only people to know that you are a research subject are the members of research team. No information about you or provided by you during the research will be disclosed to others without your written permission except:

1. In emergency to protect your rights and welfare.
2. If required by law.

FINANCIAL INCENTIVES FOR PARTICIPATION:

You will not be paid any monetary benefits or free gifts for participation in the research. You will not be reimbursed for expenses.

AUTHORISATION TO PUBLISH RESULTS:

When the results of the research are published or discussed in a conference, no information will be displayed that would disclose your identity. Any information that is obtained in connection with this study and that can be identified with you will remain confidential.

CONSENT STATEMENT:

I, the undersigned, have been explained in my own vernacular language about the study and my participation in the study is voluntary. If I want I can

withdraw at any time. Also I have been given enough time to clear my doubts about the study and my rights as a study participant.

In case you have any questions related to the study you can contact Dr Manjunath R. Desai (Phone no. 99644 03657).

In case you have any questions about your rights as a study participant you can contact Dr V. D. Patil (0831-2471350).

Signature or the left thumb impression of the participant or legally authorized representative.

Participant's name_____ Signature_____

Witness name _____ Signature_____

Experimenter's name _____ Signature_____

Place _____

Date _____

ANNEXURE II – PROFORMA

A clinical and etiological study of late onset seizures - A cross sectional study at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre

Name: IP No.:

Age: Sex:

Address: Religion:

D.O.A: D.O.D:

Occupation:

A. PRESENT HISTORY

Seizure pattern

1. Date and time of onset:
2. Number of attacks
3. Duration
4. Interval between attacks
5. Type of seizure
    a. Partial
        i. Simple
        ii. Complex
        iii. Partial seizure with secondary generalization
    b. Generalised
        i. Tonic- Clonic
        ii. Tonic
        iii. Atonic
        iv. Myoclonic
        v. Absence

c. Others

6. Prodromes:
7. Aura:
8. Detailed description of the seizure:

ASSOCIATED NEUROLOGICAL SYMPTOMS:

1. Symptoms of raised intracranial tension:

a. Headache

b. Blurred vision, Diplopia

c. Projectile vomiting

d. Altered consciousness

2. Focal neurological deficits:
3. Symptoms of neuroinfection

a. Fever

b. Neck stiffness

c. Focal deficits

4. Trauma
5. Features of degenerative neurological disease
6. Any other

ASSOCIATED NON-NEUROLOGICAL PROBLEMS

1. Infection

a. Fever

b. Ear discharge

c. Nasal discharge

d. Osteomyelitis

e. Productive cough

2. Cardiovascular disease:
    a. Chest pain
    b. Dyspnea
    c. Palpitations
3. Collagen vascular disease:
    a. Rash
    b. Joint pains
4. Malignant disease:
    a. Anorexia
    b. Weight loss
5. Any other:

B.PAST HISTORY

1. Mental retardation
2. Febrile convulsions
3. Childhood seizures
4. Infections (Encephalitis, Meningitis, Ear discharge)
5. Diabetes
6. Hypertension
7. Tuberculosis
8. Stroke, TIA, trauma
9. IHD/RHD
10. History of any drug intake
11. Any other:

C. FAMILY HISTORY

1. Seizures

2. Neurological disease
3. Diabetes, hypertension, tuberculosis

D. PERSONAL HISTORY

1. Diet
2. Appetite
3. Sleep
4. Bowel and Bladder
5. Habits
   - Alcohol
   - Tobacco
   - Stimulant drugs (Amphetamine/cocaine)

PHYSICAL EXAMINATATION

I. General

1. Pallor
2. Jaundice
3. Cyanosis
4. Clubbing
5. Lymphadenopathy
6. Pedal oedema
7. Cutaneous markers of neuroepidermal syndromes :
   a. Café-au-lait spots
   b. Malar rash
   c. Facial port wine spot
   d. Depigmented spots on trunk

II. Vitals

- Pulse
- B.P.
- Respiratory Rate
- Temperature

III. Systemic examination

A) Nervous system

a. Higher mental function

b. Cranial Nerves

c. Motor system

d. Sensory system

e. Signs of meningeal irritation

f. Cerebellar signs

g. Skull and spine

B) Cardiovascular system

C) Respiratory system

D) Abdominal system

INVESTIGATIONS

- Complete blood count
- Routine urine examination
- Blood sugar
- Blood urea
- Serum creatinine
- Serum calcium, magnesium
- Electroencephalography

- Normal
- Abnormal
    a. Focal –.
    b. Diffuse
    c. Epileptiform discharges
        i. Generalized
        ii. Focal
    d. Provocative technique

Cranial Imaging Study:

1. MRI Brain
2. CT scan Brain
3. Others
    a. Normal
    b. Abnormal
        i. Site of lesion
        ii. Nature of lesion
        iii. Diagnosis

Other investigations (As necessary)

1. Serological tests
    - For syphilis
    - For HIV detection
    - Demonstrations of antibodies to Cysticerci in serum or CSF
    - Serology for Toxoplasma infection
2. LP CSF study
3. ECG, Chest X- Ray, Echocardiography

Final Diagnosis

ANNEXURE III – FIGURES

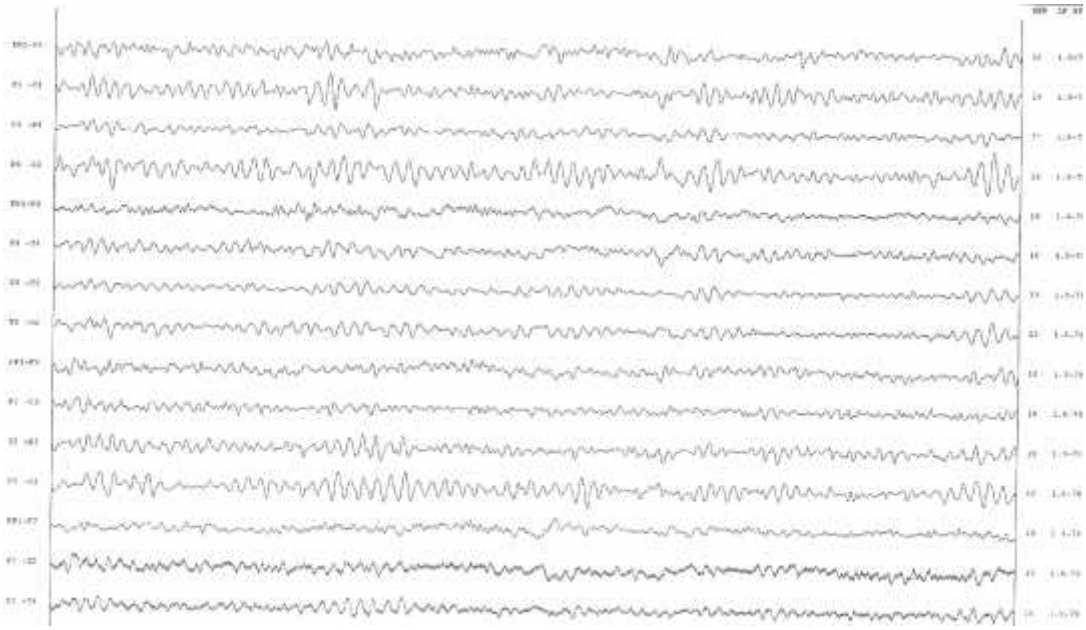

Figure No. 2: EEG showing generalized sharp waves

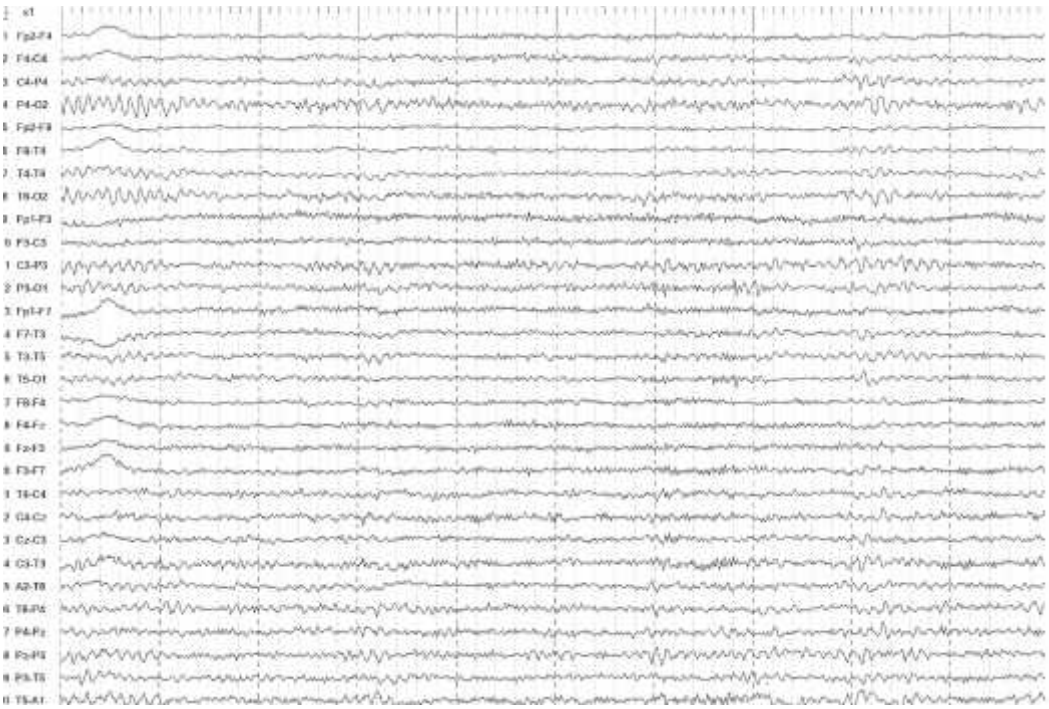

Figure No. 3: EEG showing biparietal sharp waves

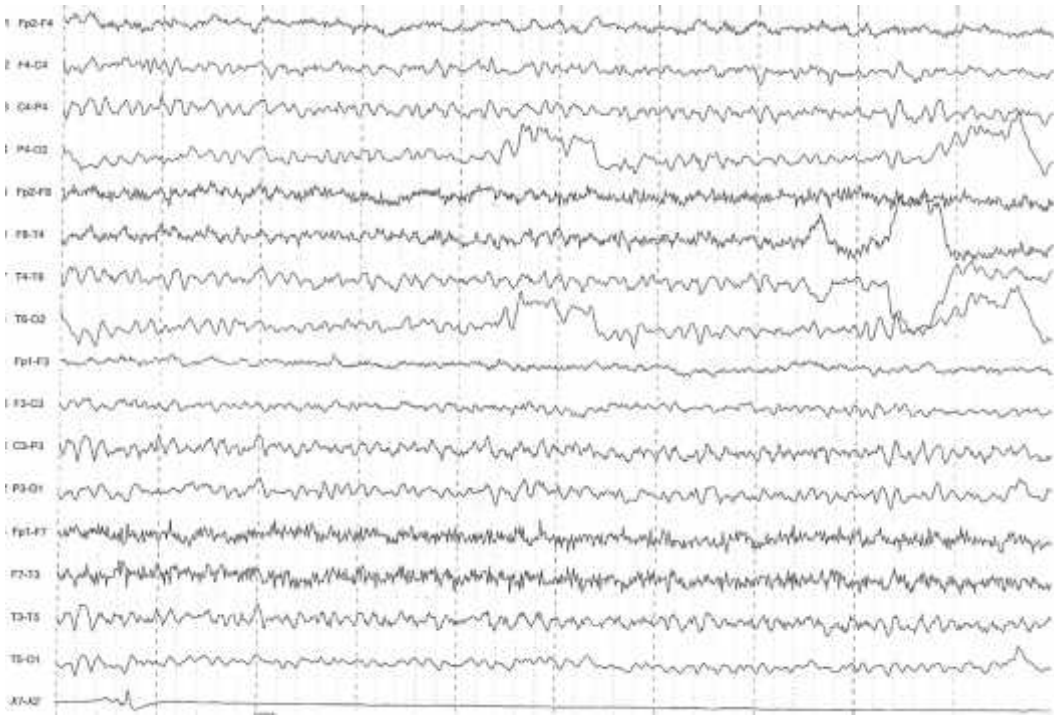

Figure No. 4: EEG showing diffuse slowing

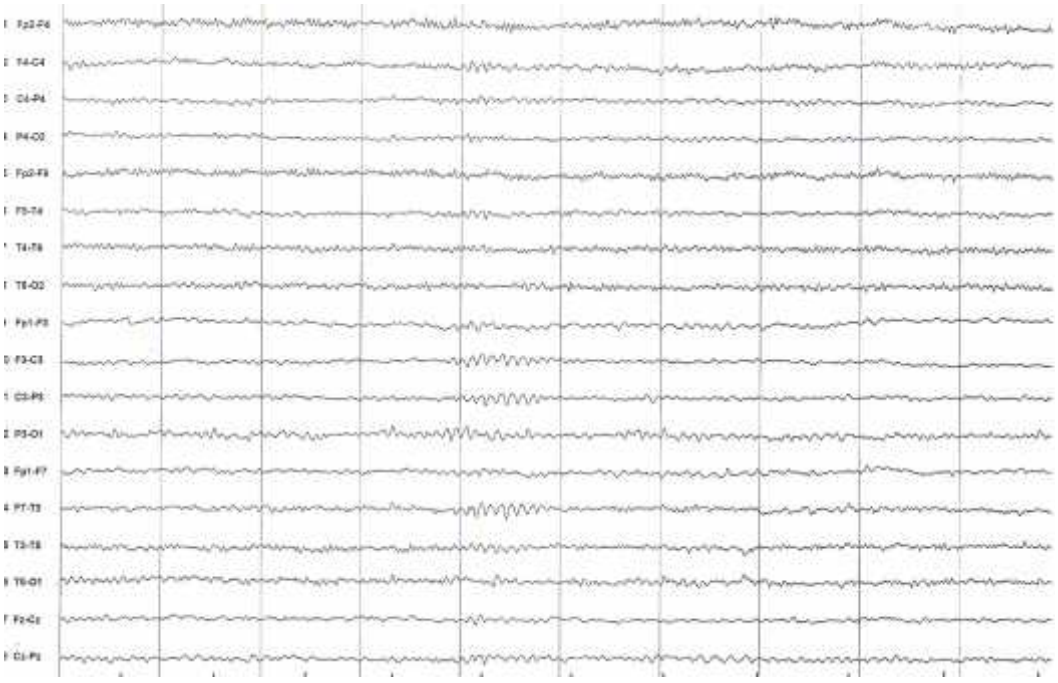

Figure No. 5: EEG showing left parietal slowing

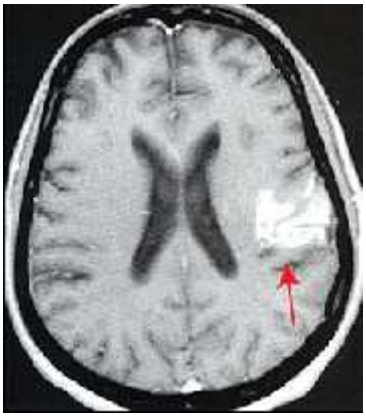

Figure No. 6: MRI of a patient with acute ischaemic stroke in left parietal area of brain

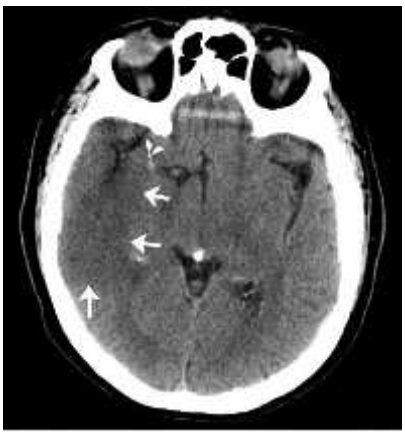

Figure No. 7: Axial CT scan showing acute infarct in right middle cerebral artery territory.

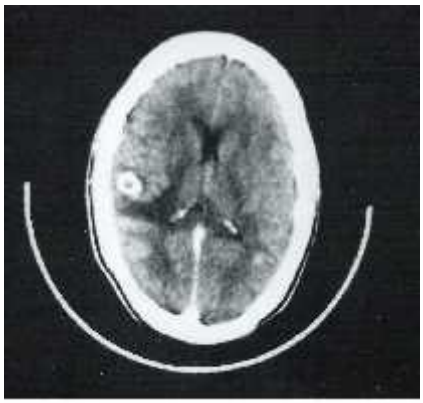

Figure No. 8: Ring enhancing lesion of neurocysticercosis

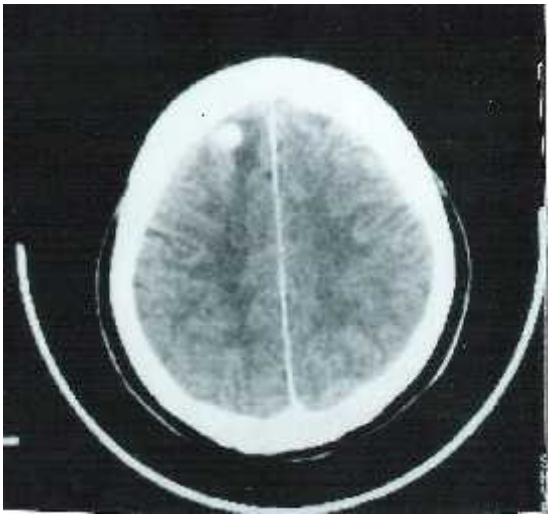

Figure No. 9: Disc enhancing lesion of neurocysticercosis

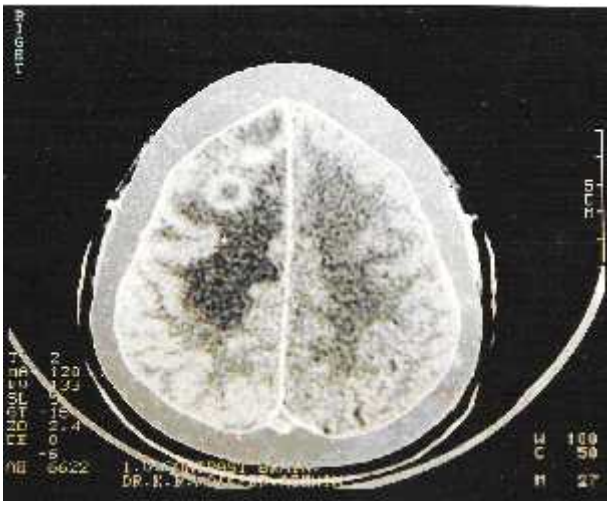

Figure No. 10: Ring and disc enhancing lesions of tuberculoma

MASTER CHART

Sl. No.	I.P.No.	Age (Years)	Sex	Type of Seizure	No. of seizures at presentation	Associated neurological problem	Associated nonneurological problem	Past History	Family Hisotry	Personal Hisotry	GPE	Neurological examination	Other systems	EEG	Cranial Imagig	Other investigations
1	244510	62	F	Right focal motor	2	RH	-	T2 DM	-	-	P	BA, RH	-	Left hemispheric sharp waves	Left posterior frontal infarct	-
2	236412	44	M	GTCS	4	QP, Alt S	-	-	-	AL	-	Alt S, QP	-	Right frontal slow waves	SSS thrombosis with right frontal haemorrhagic infarct	-
3	235891	48	M	GTCS	1	Fever, Vomiting	ED	-	-	-	P	NR	-	Intermittent bifrontal slowing	Right mastoid hyperintensity	-
4	237798	44	M	Left focal motor	6	-	-	T2 DM, HTN	-	-	-	-	-	N	Right parietal REL (2-5 cms)	-
5	242990	60	M	GTCS	2	Raised ICT, RH	-	DM, HTN	-	-	-	BA, RUFP RH	-	N	Old infarct in left parieto occipital area	-
6	238507	59	M	GTCS	4	LOC	-	-	-	-	-	-	-	N	Left posterior parietal infarct	-
7	236512	39	F	CPS	6	Alt S	-	-	-	-	P	-	-	Left temporal sharp waves	Left temporal infarct	-
8	216238	63	F	GTCS	4	Alt S	-	DM	-	-	-	Alt S	-	N	Left frontal infarct	-
9	228002	42	F	GTCS	2	-	-	-	-	-	TBt	-	-	Right frontal spike and sharp waves	N	-
10	236412	72	F	GTCS	1	Right ULW	-	DM HTN	-	-	P, TBt	Alt S, Right ULW	-	N	Old infarct in bilateral fronto parietal lobes	-
11	235126	37	M	GTCS	2	Alt S, RUFP	-	-	-	-	P	RUFP	-	Generalized sharp waves	Infarct in left temporo parietal region	-
12	224162	36	M	GTCS	1	Fever, Vomiting	-	-	-	-	P	LLRP, NR	-	N	N	-
13	238507	89	M	GTCS	1	Alt S, C Dys	-	C Dys	-	-	P	Recent memory impaired	-	Left frontal sharp waves	Infarct in left high parietal area	-
14	224815	36	F	Right focal motor	1	LH	-	-	-	-	P	Alt S, LUFP, LH	-	Generalized sharp waves	SSS thrombosis with bilateral parietal venous infarct	-

MASTER CHART

Sl. No.	I.P.No.	Age (Years)	Sex	Type of Seizure	No. of seizures at presentation	Associated neurological problem	Associated nonneurological problem	Past History	Family Hisotry	Personal Hisotry	GPE	Neurological examination	Other systems	EEG	Cranial Imagig	Other investigations
15	225223	37	M	GTCS	2	Alt S	-	-	-	A	P	Tremors	-	N	N	-
16	224342	38	M	Right focal sensory	4	-	-	HTN	-	-	-	-	-	Left fronto parietal slow waves	Left parietal SSREL ? Tuberculoma	CSF, IgM TB ELISA +, CSF ACA -
17	229672	70	M	GTCS	1	Alt S	-	HTN, Old RH	-	-	P, TBt	Alt S, RH	-	N	Old left pontine infarct	-
18	228330	36	M	GTCS	1	Alt S	-	-	-	A	P, TBt	Alt S	-	N	Old infarct in right MCA territory	-
19	233155	70	F	Left focal motor with SG	1	-	-	-	-	-	-	-	-	Mild Background slowing +	Old infarct in right basal ganglia	-
20	234316	54	F	CPS	1	-	-	-	-	-	-	-	-	Left temporo parietal sharp waves	Periventricular white matter hyperintensity	-
21	226391	38	M	GTCS	15-20	Alt S	-	-	-	-	P	Alt S	-	Bifrontal intermittent sharp waves	N	-
22	259246	42	F	GTCS	3	Fever, Vomiting	-	-	-	-	P, TBt	NR	-	Generalized sharp waves	Right parietal SSREL	CSF TB PCR + HIV +
23	258513	54	M	GTCS	1	-	-	-	-	A	-	-	-	N	N	-
24	258320	37	M	GTCS	3	-	-	-	-	A	-	-	-	Generalized sharp waves	Left temporal SSREL	Serum, CSF ACA +
25	245913	62	M	Left focal motor with SG	3	LH	-	Old LH	-	A	-	BA, LUFP, LH	-	Diffuse spike and sharp waves	Old infarct in right temporoparietal area	-
26	232450	58	M	Left focal motor	2	ULW	Cough 1 year	-	-	S	P	Left ULW	Right lung cavity +	N	Multiple REL in right parietal area	CT thorax right lower lobe cavity
27	245867	65	F	GTCS	5	Alt S, C Dys	-	-	-	-	P	Alt S	-	Diffuse slowing of background	Diffuse cerebral atrophy	-
28	229637	39	F	Left focal sensory with SG	3	Left ULW	-	-	-	-	P	Left ULW	-	Generalized sharp waves	Right parietal SSREL	CSF ACA +
29	256751	52	M	GTCS	2	Alt S	-	Head Injury	-	S, A	TBt	Alt S	-	N	Right frontal gliosis ? Post traumatic	-

MASTER CHART

Sl. No.	I.P.No.	Age (Years)	Sex	Type of Seizure	No. of seizures at presentation	Associated neurological problem	Associated nonneurological problem	Past History	Family Hisotry	Personal Hisotry	GPE	Neurological examination	Other systems	EEG	Cranial Imagig	Other investigations
30	258671	47	M	Right focal sensory, SG	3	Alt S	-	-	-	-	-	-	-	Left fronto parietal sharp waves	Left parietal SSREL	CSF ACA +
31	271756	42	M	GTCS	4	Alt S	-	-	-	A	-	Left UFP, LH	-	Bilateral frontotemporal spike waves	SSS Thrombosis with right parietal venous infarct	-
32	271205	43	M	GTCS	3	Alt S	-	-	-	A	P	-	-	Generalized sharp waves	N	-
33	271375	51	M	Left focal motor	1	LH	-	T2 DM, Head Injury	-	-	P	-	-	Right fronto parietal sharp waves	Old infarct in right temporal area	-
34	270568	56	M	GTCS	2	Diplopia, Alt S, RH	-	DM HTN	-	S, A	-	BA, Right UFP, RH	-	Generalized sharp waves	Infarct in left parietal area	-
35	272302	57	M	GTCS	1	-	-	-	-	-	-	-	-	Left temporal spike waves	N	-
36	271345	43	M	CPS	1	-	-	RHD	-	A	-	-	-	N	Old infarct in left parietal area	-
37	270258	72	M	Left focal motor with SG	3	LH	-	HTN	-	-	P	Left UFP LH	-	N	Old infarct in right parietal region	-
38	270131	48	M	Right focal motor with SG	1	-	-	-	-	-	TBt	-	-	N	N	-
39	269823	64	M	Right focal sensory	2	Left ULW	-	-	-	A	-	Ataxia	-	N	Right parietal lobe infarct	-
40	270136	50	F	GTCS	1	-	-	-	-	-	-	-	-	N	N	-

ANNEXURE IV – KEY TO MASTER CHART

A	-	Chronic alcoholic
ACA	-	Anti cysticercal antibody
Alt S	-	Altered sensorium
BA	-	Brocas Aphasia
C DYS	-	Cognitive dysfunction
GTCS	-	Generalized tonic clonic seizure
HTN	-	Hypertension
LLRP	-	Left lateral rectus palsy
LH	-	Left hemiparesis
LUFP	-	Left upper motor neuron type facial palsy
NR	-	Neck rigidity
P	-	Pallor
PCA	-	Polymerase chain reaction
QP	-	Quadriparesis
REL	-	Ring enhancing lesion
RH	-	Right hemiparesis
RUFP	-	Right upper motor neuron type facial palsy
S	-	Chronic smoker
SG	-	Secondary generalization
SSREL	-	Single small ring enhancing lesion
SSS	-	Superior sagittal sinus
T2 DM	-	Type 2 diabetes mellitus
TBt	-	Tongue bite
ULW	-	Upper limb weakness