INCIDENCE OF SEIZURES IN STROKE PATIENTS

M.D. – DEGREE EXAMINATION
BRANCH-I – GENERAL MEDICINE
STANLEY MEDICAL COLLEGE, CHENNAI.

Dissertation Submitted to
THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY, CHENNAI

SEPTEMBER 2006

CERTIFICATE

This is to certify that the dissertation titled **"incidence of seizures in stroke patients"** is the Bonafide original work of Dr. C. S. Gauthaman in partial fulfillment of the requirement for M.D. Branch –I (General Medicine) examination of The Tamil Nadu Dr. M. G. R. Medical university to be held in September 2006.The period of study is from June 2005 to January 2006.

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I **Dr. C.S. GAUTHAMAN**, solemnly declare that the dissertation titled, **"INCIDENCE OF SEIZURES IN STROKE PATIENTS"** was done by me at Govt. Stanley Medical College and Hospital during 2005 – 2006 under the guidance and supervision of my unit chief **Prof. Dr. A.K. GEETHA DEVI, M.D.**

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ACKNOWLEDGEMENT

I owe my thanks to the Dean, Govt. Stanley Medical College and Hospital **Dr. M.VASANTHA, M.D.** for allowing me to avail the facilities needed at her disposal for my dissertation work.

It gives me great pleasure to express my deep sense of gratitude to **Prof. Dr. S. NATARAJAN, M.D.** Professor and Head of the Department of Internal Medicine, Stanley Medical College and Hospital for permitting me to do this dissertation and indebted to him for his foresight, guidance and encouragement during the study.

I thank wholeheartedly **Prof. Dr. A. K GEETHA DEVI, M.D.,** my Unit Chief & Professor of Medicine, for being a source of inspiration & excellent guidance during the period of my study.

My sincere thanks to **Prof. M. DHANRAJ M.D., D.M,** and his team of Assistant Professors and Post Graduates for their valuable guidance, co-operation and help in every stage of study, which has made this dissertation work possible.

I am extremely thankful to my unit Assistant Professors **Dr. NALINI KUMARAVELU M.D.,** and **Dr. S. CHANDRASEKAR M.D** for their valuable guidance and encouragement.

Last but not the least, my sincere thanks to the patients who co-operated for this study, without whom the study could not have been completed, and all my colleagues who shared their knowledge about this study.

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INTRODUCTION

Stroke is one of the most common causes of seizures in elderly.

The relation between seizures and stroke was recognized more than a century ago by John Hughling Jackson.

The reported incidence of seizures after stroke varies from 4.1% - 12.5%. This is related to different study population and follow up times. The incidence of post stroke seizures in India is 13%. Despite the relatively low incidence of seizures after cerebral stroke, Post stroke seizures is one of the most common causes of seizures due to the high incidence of stroke.

A seizure may occur before, at the onset of, or weeks to months after a stroke. Hence the onset of seizures in adult or elderly population may be a warning sign for further stroke and warrants a study of patients cerebral circulation.

An important risk factor for development of seizures after stroke is the involvement of cerebral cortex. Hemorrhagic strokes result in seizures more frequently than do Ischemic strokes.

The presence of precipitating factors like hyperglycemia, hypoglycemia, hypernatremia, hypocalcaemia, hypomagnesaemia, renal failure and infections – increase the chance of seizures. Atrial fibrillation and diabetes are found to be associated with increased risk of early seizures.

There is a strong link between stroke severity and risk of seizures after stroke. The risk is very low in mild strokes.

AIM

Both early and late onset post stroke seizures left sided cortical infarcts, increased stroke severity and recurrent strokes are the risk factors for post stroke late epilepsy. The present study was conducted prospectively to define the clinical features, CT findings and EEG correlation of stroke patients with seizures.

STROKE

Stroke was defined according to WHO criteria that is, rapidly developed clinical signs of focal disturbance of cerebral function lasting more than 24 hours or leading to death with no apparent cause other than vascular origin.

Despite gradual decline in overall stroke death rates in industrialized countries, stroke remains the third leading cause of death. Stroke is also the leading cause of disability in adults. Stroke is of two major types - Ischemic and hemorrhagic.

ISCHEMIC STROKE:

Pathophysiology of cerebral Ischemia:

Cerebral Ischemia is caused by reduced blood supply to the brain microcirculation. Ischemia causes impairment of brain energy metabolism, loss of aerobic glycolysis, intracellular accumulation of sodium and calcium ions, release of excitotoxic neurotransmitters, elevation of lactate levels with local acidosis, free radical production, cell swelling, over activation of lipases and proteases and cell

death. Many neurons undergo Apoptosis. Ischemic brain injury is exaggerated by leucocyte infiltration and development of brain edema.

Pathology of Ischemic stroke:

The pathological characteristics of Ischemic stroke depend on

- the mechanism of stroke
- the size of the obstructed artery
- the availability of collateral blood flow

The changes do not occur immediately and may be delayed up to 6 hours after the infarction. There is neuronal swelling initially, which is followed by shrinkage, hyperchromasia and pyknosis. Chromatolysis appears and the nuclei become eccentric. Swelling and fragmentation of the astrocytes and endothelial swelling occur.

Neutrophils infiltrations appear as early as 4 hours and microglia proliferate within 48 hours. In large infarction there are three distinct zones.

1. An inner area of coagulative necrosis
2. A middle zone of vacuolated neutrophils, leucocyte infiltrates, swollen axons and thickened capillaries and
3. An outer marginal zone of hyperplastic astrocytes

Clinical Syndromes of Cerebral Ischemia:

1. Transient Ischemic attacks
2. Carotid artery system syndromes
3. Vertebro basilar system syndromes
4. Lacunar syndromes
5. Syndromes of thalamic infarction
6. Watershed Ischemic syndrome.

1. TRANSIENT ISCHEMIC ATTACKS (TIA):

A TIA is a temporary, focal and “nonmarching” neurological deficit of sudden onset, related to Ischemia of brain, retina or cochlea and lasting less than 24 hours.

TIAs may result from athero embolism that originates from ulcerated extra cranial arteries, emboli of cardiac origin, occlusion of small penetrating arteries that arise from the large surface arteries of the circle of Willis, altered local blood flow (perfusion failure) caused by severe arterial stenosis, non atherosclerotic vasculopathies or hyper coagulable states.

The following symptoms are considered typical of TIAs in the carotid circulation- ipsilateral amaurosis fugax, contralateral sensory or motor dysfunction limited to one side of body, aphasia, contralateral homonymous hemianopia or a combination thereof.

The following symptom is considered typical of vertebrobasilar system TIA - bilateral or shifting motor or sensory dysfunction, complete or partial loss of vision in the homonymous fields of both eyes or any combination of these symptoms.

2. CAROTID ARTERY SYSTEM SYNDROMES:

An MCA infarction is one of the most common manifestations of cerebrovasular disease. The clinical picture with an MCA infarction is varied and depends on whether the site of occlusion is in the stem, superior division, inferior division or lenticulostriate branches and whether there is good collateral blood flow.

When the stem of the MCA is occluded, there is usually a large infarction with contralateral hemiplegia, conjugate eye deviation towards the site of infarct, hemianesthesia and homonymous hemianopia. Associated global aphasia occurs if

the dominant hemisphere is involved and hemineglect with nondominant hemispheric lesions.

In upper division MCA infarction, the hemiparesis usually affects the face and arms more than the leg. A Broca type of aphasia is more common in upper division infarcts. With lower division MCA syndromes Wernick type aphasia is seen with dominant hemispheric infarction and behavioral disturbance are seen with non-dominant infarction.

A leuticulo striate branch occlusion may cause a lacunar infarction with involvement of the internal capsule producing a syndrome of pure motor hemiparesis. Alexia with agraphia may occur with left sided angular gyrus involvement. Gerstmann's syndrome is seen with dominant hemisphere parietal lesions.

Anterior cerebral artery (ACA) territory infarctions are uncommon. They occur in patients with vasospasm after Subarachnoid hemorrhages caused by anterior cerebral artery or anterior communicating artery aneurysm.

The characteristics of anterior cerebral artery infarction include

contralateral weakness involving primarily the lower extremity and to a lesser extent, the arm. Also include abulia, akinetic mutism (with bilateral mesio frontal damage), impaired memory or emotional disturbances, Trans cortical motor aphasia (with dominant hemisphere lesions), deviation of head and eye towards lesion, Paratonia (gegelhaten), discriminative and proprioceptive sensory loss and sphincter incontinence.

The anterior choroidal artery syndrome is often characterized by

hemiparesis caused by involvement of the posterior limb of internal capsule, hemi sensory loss caused by involvement of the posterolateral nucleus of the thalamus or thalomo cortical fibres and hemianopia secondary to involvement of the lateral geniculate body.

3. VERTEBRO BASILAR SYSTEM SYNDROMES:

PICA territory cerebellar infarctions:

1. If the medial branch is affected clinical findings include prominent vertigo, ataxia and nystagmus.

2. If the lateral cerebellar hemisphere is involved, Patients can have vertigo, gait ataxia, limb dysmetria and ataxia, nausea, vomiting, conjugate or dysconjugate gaze palsies, miosis and dysarthria.

Anterior Inferior cerebellar artery syndrome:

- Causes ventral cerebellar infarction
- The signs and symptoms include vertigo, nausea, vomiting and nystagmus – caused by involvement of vestibular nucleus. Involvement of the trigeminal spinal nucleus and tract may cause ipsilateral facial hypalgesia and thermo anesthesia
- Ipsilateral ataxia and asynergia caused by involvement of the cerebellar peduncle and cerebellum
- Ipsilateral Horner's and contra lateral trunk and extremity hypalagesia.

Superior cerebellar artery territory infarction:

- Produces a dorsal cerebellar syndrome.
- Vertigo, nystagmns, ipsilateral Horner's, ipsilateral ataxia, asynergia and gait ataxia may be present.
- Intention tremor and choreiform dyskinesias may be present ipsilaterally.

- Contra laterally there is hearing loss and extremity hyalgesia and thermo anesthesia.

Locked-in syndrome:

- Is the result of bilateral ventral pontine lesions that produce quadriplegia, aphonia and impairment of horizontal eye movements in some patients.
- Wakefulness and normal sleep-wake cycles are maintained because of sparing of the reticular formation.

Top of the basilar syndrome:

- Caused by occlusion to rostral basilar artery leading to infarction of the midbrain, thalamus, and portions of temporal and occipital lobes
- Behavioral abnormalities like somnolence, pendular hallucinosis, and memory disturbance or agitated delirium may occur.

Other syndromes include:

1. Weber's
2. Benedict's
3. Claude
4. Lateral inferior pontine syndrome
5. Lateral pontomedullary syndrome
6. Lateral medullary syndrome (Wallenberg syndrome.)
7. Medial medullary (Dejerrine) syndrome

4 **LACUNAR SYNDROME**:

Lacunar infarcts are small ischemic infarctions in the deep regions of the brain or brainstem that range in the diameter of 0.5 – 15.0 mm.

These infarctions result from occlusion of the penetrating arteries, chiefly the anterior choroidal, middle cerebral, and posterior cerebral and basilar arteries.

At least 20 lacunar syndromes have been described and the five best recognized syndromes are

1. Pure motor hemiparesis:

   Often caused by an internal capsule, basis pontis or corona radiata lacune and is characterized by contra lateral hemiparesis or hemiplegia and mild dysarthria at the onset of stroke.

2. Pure sensory stroke:

   Caused by lacune involving the ventroposterolateral nucleus of thalamus.

3. Sensory – motor stroke:

   Caused by lacune involving the internal capsule and thalamus or posterior limb of internal capsule.

4. Homolateral ataxia and crural paresis:

   Caused by lacune either in the contralateral posterior limb of the internal capsule or the contralateral basis pontis.

5. Dysarthria – Clumsy hand syndrome:

   Caused by a lacune involving the deep areas of the basis pontis and is characterized by supranuclear facial weakness, dysarthria, dysphagia, loss of fine motor control of hand and Babinski's sign

Syndromes of thalamic infarction:

The main thalamic blood supply comes from the posterior communicating arteries and the perimesencephalic segment of the PCA. Thalamic infarctions typically involve one of the four major vascular regions.

1. Postero lateral
2. Anterior
3. Para median and
4. Dorsal

Postero lateral thalamic infarctions include three clinical syndromes

1. Pure sensory stroke
2. Sensory motor stroke
3. Thalamic syndrome of Dejerrine roussy

Watershed Ischemic syndromes:

Watershed infarcts occur in the border zone between adjacent arterial perfusion beds.

Ischemia may occur in the watershed areas between the major circulations, during or after cardiac surgery or after an episode of sustained and severe arterial

hypotension after cardiac arrest, during prolonged hypoxemia or bilateral severe carotid artery disease.

Diagnosis and treatment of Ischemic stroke:

Ischemic stroke accounts for approximately 80% - 85% of all strokes.

Ischemic stroke may result from

1. Large artery atherosclerotic disease resulting in stenosis or occlusion.
2. Small vessel or penetrating artery disease (lacunar)
3. Cardiogenic or artery to artery embolism
4. Non atherosclerotic vasculopathies
5. Hyper coagulable disorders
6. Infarcts of undetermined causes

Essential investigations for stroke patients:

A basic workup is done in all stroke patients which include full blood cell count, ESR, prothrombin time, aPTT, plasma glucose level, blood urea nitrogen, serum creatinine, lipid and cholesterol analysis, urinalysis, chest roentgenography and ECG.

Non-enhanced cranial CT is also being done to detect hemorrhage, mass lesion or cerebral infarction. MRI and MRA (Intracranial and extra cranial) improve the ability to localize an acute stroke and provide powerful non-invasive mean to evaluate the Pathological changes that occur following acute Ischemic stroke.

Duplex Doppler ultrasonography are used to detect stenosis of blood vessels. Transcranial Doppler sonography assist in evaluation of blood flow velocities and patency of the main intracranial arteries and in the identification of high intensity transient micro embolic signals.

Cardiac investigations to determine whether emboli have a cardiac source are advised in selected circumstances. Serial two-dimensional echocardiography is used to detect left ventricular thrombus and left atrial thrombus.

Conventional angiography or intra-arterial digital substraction is the gold standard for establishing the extent of vascular disease. Cerebral angiography is indicated in several circumstances particularly when the diagnosis remains uncertain and when surgical treatment is planned. Angiography is also indicated in patients with very early evolving stroke symptoms and when distinctions affecting treatment are unclear.

Treatment:

The first Goal is to prevent or reverse brain injury. Treatments designed to reverse or lessen the amount of tissue infarction fall within five categories.

1. Medical support
2. Thrombolysis
3. Anticoagulation
4. Antiplatelets and
5. Neuroprotection

1. **Medical Support:**

The immediate Goal is to optimize cerebral perfusion in the surrounding ischemic penumbra.

Blood pressure should be lowered if there is malignant hypertension or concomitant myocardial Ischemia or if the blood pressure is >185/110 mmHg and when thrombolytic therapy is anticipated.

When faced with competing demands of myocardium and brain, lowering the heart rate with a β adrenergic blocker can be a first step to decrease cardiac work and maintain blood pressure.

Fever is detrimental and should be treated with antipyretics. Serum glucose should be monitored and kept at <11.1 mmol/l or 200mg/dl. Edema peaks on the second or third day but may cause mass effect for 10 days. Even small amount of cerebellar edema can actually increase intracranial pressure in the posterior fossa or directly compress the brainstem. Water restriction and intravenous mannitol may be used to raise the serum osmolarity, but hypotension should be avoided as this may contribute to hypo perfusion and worsening infarction.

Attention is also directed towards preventing common complications of bedridden patients – Infections (Pneumonia, urinary tract and skin) and deep vein thrombosis with pulmonary embolism.

2. Thrombolysis:

If patients meet appropriate criteria, thrombolytic therapy may be administered.

a) The national institute of neurological disorders and stroke (NINDS) recombinant tPA (rtPA) stroke study show a clear benefit for intravenous rtPA in selected stroke patients.

   Dose: 0.9mg/kg to 90 mg maximum

   10% as bolus and remaining over 1 hour

b) The Prolyse in Acute Cerebral Thromboembolism (PROACT) II trial found benefit for intra-arterial pro-urokinase for acute middle cerebral artery occlusion up to the sixth hour following onset of stroke.

3. Antiplatelet agents:

Aspirin is the only anti platelet agent that has been prospectively studied for the treatment of acute Ischemic stroke. The recent large trials, the International Stroke Trial (IST) and the Chinese Acute Stroke Trial (CAST) found that the use of aspirin within 48 hours of stroke occurrence reduce risk and mortality minimally.

Agents that act at the glycoprotein II b/ III a receptor are undergoing clinical trials in acute stroke treatment. Early results show that intravenous

Abciximab can be used safely within 6 hr of stroke onset and suggest that it may be effective.

4. Anticoagulation:

The role of anticoagulation in atherothrombotic cerebral ischemia is uncertain. The US trial of organon 10172 in acute stroke treatment (TOAST) an Investigational low molecular weight heparin, failed to show any benefit over aspirin.

5. Neuro protection:

Is the concept of providing a treatment that prolongs brain tolerance to ischemia.

INTRACEREBRAL HEMORRHAGE

Intracerebral hemorrhage (ICH) accounts for approximately 10% of strokes.

The main cause of ICH is hypertension. The actual vascular lesion produced by chronic hypertension that leads to arterial rupture and ICH is probably due to lipohyalinosis of small intra parenchymal arteries. The role of microaneursym of Charcot and Bouchard is uncertain.

The non-hypertensive causes of ICH are

1. Vascular malformations (Saccular or mycotic aneurysm, AVmalformations, cavernous, angiomas)
2. Intracranial tumors
3. Bleeding disorders, anticoagulant and fibrinolytic treatment
4. Cerebral amyloid angiopathy
5. Granulomatous angitis of the CNS and other vasculitides, such as polyarteritis nodosa
6. Sympathomimetic agents (including amphetamine and cocaine)
7. Hemorrhagic infarction
8. Trauma

Hemorrhagic infarction:

Hemorrhagic infarction is pathologically and pathogenically different from ICH in that it results from arterial or venous occlusion, rather than vascular rupture that causes ICH.

The pathological aspect is one of multifocal petechial hemorrhagic staining of an area of the brain primarily affected by Ischemic necrosis.

Hemorrhagic infarction characteristically occurs in the setting of cerebral embolism or, less frequently cerebral infarction secondary to venous occlusion. (e.g. superior sagittal sinus thrombosis).

Clinical features of intra cerebral hemorrhage:

The clinical presentation of ICH has two main elements

- Symptoms that reflect the effects of intracranial hypertension and
- Those that are specific for the location of the hematoma.

The general clinical manifestations of ICH related to increased intracranial pressure (headache, vomiting and depressed level of consciousness) are variable in their frequency at the onset of ICH.

A characteristic ICH at presentation is the frequent progression of the focal neurological deficits over period of hours. This early course reflects the progressive enlargement of hematoma.

Classification of ICH according to anatomic location:

1. Putaminal hemorrhage:
    - Most common variety represents approximately 35% of the cases.
2. Lobar hemorrhage:
    - Second to putaminal hemorrhage in frequency, accounting for approximately 25% of the cases.
3. Thalamic hemorrhage:
    - Represents 10 – 15% of the cases of ICH
4. Caudate hemorrhage
5. Cerebral hemorrhage
6. Pontine hemorrhage
7. Mesencephalic hemorrhage
8. Medullary hemorrhage and
9. Intranventricular hemorrhage

Laboratory and imaging evaluation:

The CT scan reliably detects focal hemorrhages in the supratentorial space. Small pontine hemorrhages may not be identified because of motion and bone induced artifact.

Images of flowing blood on MRI scan may identify AVMs as the cause of hemorrhage. MRI, CT angiography and conventional x-ray angiography are used when the cause of intra cranial hemorrhage is uncertain.

Laboratory test should include coagulation studies especially in ICH patients receiving anticoagulants or previously treated with thrombolytic agents

Treatment of intracerebral hemorrhage:

Patients with ICH need to be immediately evaluated for stabilization of vital signs and airway protection. If the patient has a depressed level of consciousness with a Glasgow coma scale of 8 or less, endotrached intubation should follow.

Because ICH is associated frequently with increased ICP, most of the therapies used in these settings are directed at lowering ICP or preventing its increase.

General measure includes control of hypertension. The antihypertensive agent of choice is the intravenous β and α blocking agent labetalol, often used in combination with loop diuretics. Nitroprusside and hydralazine are the most

appropriate choices when labetalol fails to control the blood pressure, although theoretically contraindicated because of their cerebral vasodilator properties.

Specific treatment for increased intra cranial pressure include hyperventilation, diuretic therapy and corticosteroids

Early tonic clonic seizures need immediate control because they may contribute to increased ICP.

SEIZURES

A seizure (from the LATIN ‘Sacire’, ‘to take possession of’) is a paroxysmal event due to abnormal, excessive, hypersyschronous discharges from an aggregate of central nervous system neuron.

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Classification of seizures:

Classification of international league against epilepsy published in 1981

1. Partial seizures
    a. Simple partial seizures (with motor, sensory, autonomic and Psychic signs)
    b. Complex partial seizures
    c. Partial seizure 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 seizures

Partial seizures:

Partial seizures are those in which the seizure activity is restricted to discrete areas of cerebral cortex and are usually associated with structural abnormalities of the brain

a. Simple partial seizures:

Simple partial seizures cause motor, sensory, autonomic or psychic symptoms without an obvious alteration in consciousness.

b. Complex partial seizures:

Are characterized by focal seizure activity accompanied by a transient impairment of patient's ability to maintain normal contact with environment. The behavioral arrest is usually accompanied by 'Automatism'.

c. Partial seizures with secondary generalization:

Partial seizures can spread to involve both cerebral hemispheres and produce 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.

Generalized seizures:

Generalized seizures may be practically defined as bilateral clinical and electrographic events without any detectable focal onset.

a. Absence seizures (Petit mal):

Absence seizures are characterized by sudden, brief lapses of consciousness without loss of postural control. The seizure typically lasts for only seconds, consciousness returns as suddenly as it was lost and there is no post ictal confusion.

b. Generalized Tonic – clonic seizures (grand mal):

Primarily generalized tonic – clonic are the main seizure type in 10% of all persons with epilepsy. The seizures usually begin abruptly

without warning, although some patients describe vague premonitory symptoms in the hours leading up to the seizure.

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. 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 post ictal phase is characterized by unresponsiveness, muscular flaccidity and excessive salivation that can cause stridorous breathing and partial airway obstruction. Bladder and bowel incontinence may occur.

c. Atonic seizures:

Are characterized by sudden loss of postural muscle tone lasting 1 to 2 sec. Consciousness is briefly impaired but there is usually no postictal confusion.

d. Myoclonic seizures:

Myoclonus is a sudden and brief muscle contraction that may involve one part of the body or the entire body.

Unclassified seizures:

Some seizures that occur in neonates and infants cannot be classified as partial or generalized. The distinctive phenotypes of seizures at their early ages likely result, in part, from differences in neuronal function and connectivity in the immature versus mature CNS.

Mechanism of seizure initiation and propagation:

The initiation phase is characterized by two concurrent events in an aggregate of neurons

1. High frequency burst of action potentials
2. Hyper synchronization

The bursting activity is caused by a relatively long lasting depolarization of the neuronal membrane due to influx of extra cellular calcium (Ca+), which leads to the opening of voltage dependent sodium (Na+) channels, influx of Na+ and generation of repetitive action potentials. This is followed by a hyperpolarizing after potential mediated by GABA receptors or potassium (K+) channels, depending on the cell type.

This bursting activity is prevented from spreading by intact hyper polarization and a region of surrounding inhibition created by inhibitory neurons. With sufficient activation there is recruitment of surrounding neurons, which leads to loss of surrounding inhibition and propagation of seizure activity.

Mechanism of epileptogenesis:

Epileptogenesis refers to the transformation of a normal neuronal network into that is chronically hyper excitable. There is often a delay of months to years between the initial CNS injury such as trauma, stroke or infection and the first seizure. The injury appears to initiate a process that gradually lowers the seizure threshold in the affected region until a spontaneous seizure occurs. There is also evidence that, in response to the loss of neurons, there is re organization or “sprouting” of surviving neurons in a way that affects the excitability of the network. The local hyper excitability leads to further structural damage that evolve over time until the focal lesions produce clinically evident seizures.

MECHANISM OF ACTION OF ANTIEPILEPTIC DRUGS

Antiepileptic drugs appear to act primarily by blocking the initiation or spread of seizures. This occurs through a variety of mechanisms that modify the activity of ion channels or neurotransmitters and in most cases the drugs have pleiotropic effects.

The mechanisms include;

1. Inhibition of Na+ dependent action potentials in a frequency dependent manner (e.g. phenytoin, carbamezepine, lamotrigine, topiramate, zonisamide)
2. Inhibition of voltage gated Ca 2+ channels (phenytoin)
3. Decrease of glutamate release (lamotrigine)
4. Potentiation of GABA receptor function (benzodiazepines and barbiturates) and
5. Increase in the availability of GABA (valproic acid, gabapentin, tiagabine)

The two most effective drugs for absence seizures ethosuximide and valproic acid, probably act by inhibiting T-type Ca 2+ channels in thalamic neurons.

LABORATORY STUDIES

Routine blood studies are indicated to identify the more common metabolic causes of seizures, such as abnormalities in electrolytes ,glucose, calcium or magnesium and hepatic or renal failure .

A screen for toxins in blood and urine should also be obtained from all patients in appropriate risk groups, especially when no clear precipitating factor is identified

A lumbar puncture is indicated if there is any suspicion of meningitis or encephalitis and is mandatory in all patients infected with HIV

ELECTROENCEPHALOGRAPHY

The EEG remains central to the diagnosis of epilepsy. When the EEG is Abnormal, it is useful to localize the epileptogenic region in patients with partial seizures or to distinguish seizure types. The EEG may also be helpful in the interictal period by showing certain abnormalities that are highly supportive of the diagnosis of epilepsy

NEUROIMAGING

Neuro imaging has become increasingly important in the diagnosis and management of epilepsy, especially in patients with intractable seizures who are being considered for surgery.

Computed tomography (CT) may be helpful in screening for tumors or other major structural changes that can cause seizures, but CT scan is normal in most patients with epilepsy.

Developments in magnetic resonance imaging (MRI), magnetic resonance spectroscopy (MRS), single photon emission computed tomography (SPECT), and positron emission tomography (PET) have opened up new opportunities for non-invasive brain surgery

SEIZURES IN STROKE

Stroke is an important cause of acute asymptomatic seizures and epilepsy in the elderly. The reported incidence of seizures after stroke varies from 4.1% to 12.5% .The incidence of post stroke epilepsy in India is 13%.

Post stroke seizures can be classified as early onset seizures, occurring within two weeks following stroke onset, and late onset seizures occurring after two weeks. It is important to differentiate between early onset and late onset post stroke seizures as it helps to determine the need and duration to treat these patients with antiepileptic drugs.

POST STROKE EARLY SEIZURES

In a prospective study of 1000 patients with stroke and TIA's, the incidence of early seizures was highest in patients with supratentorial lobar or extensive (lobar and deep) hemorrhages (15.4%) followed by subarachnoid hemorrhages (8.5%), carotid artery cortical infarction (6.5%) and hemispheric TIA's (3.7%). Arterio venous malformations a common cause of lobar hemorrhage is complicated by early seizures

Among the patients with cortical infarcts and early epilepsy the commonest site of infarction is in the middle cerebral artery territory. Predictably, sub cortical and brain stem infarcts, deep cerebral and infratentorial hemorrhages are not associated with increased risk of seizures. The incidence of seizures in lacunar infarcts is low (1%). In the acute stage almost 60% of seizures are partial seizures, 75% are simple partial motor while 25% become generalized.

The EEG may show mild non-specific changes or focal slowing including periodic lateralized epileptiform discharges (PLEDS)

PATHOPHYSIOLOGY OF EARLY POST STROKE SEIZURES:

Infarct consists of dead tissue in the core surrounded by nonfunctional but potentially viable tissue in the penumbra. In contrast to the dead neurons in the core zone of infarct, neurons in the penumbra are still alive and able to discharge. Enhanced release of excitotoxic glutamate, ionic imbalances, breakdown of membrane phospholipids and release of free fatty acids in the penumbra play an important role in epileptogenesis

Status epilepticus:

Early seizures presenting with status epilepticus (SE) were seen in 0.7% to 1.1% of patients with stroke. Status epilepticus represents 15.8% to 27% of patients with early seizures. It is common with lobar hemorrhages than cortical infarcts. Status epilepticus may occur as a presenting symptoms or within the first 14 days of stroke

POST STROKE LATE SEIZURES

Although post stroke late seizures occur after first two weeks of stroke, these may begin months to a year after stroke. The incidence of post stroke late seizures is about 15%. The interval between stroke and onset of seizures ranges widely. Nearly 24%of seizures occur within 2 wks and 93% in 2 yrs. The prevalence of late seizures is high in patients with permanent neurological deficits or those needing rehabilitation. A primary generalized seizure is common with late onset seizures (56%) compared to early onset seizures, which are generally simple partial in nature. Status epilepticus is more frequent in early onset than take onset seizure.

PATHOPHYSIOLOGY:

Late seizures occurring months to years after the stroke are probably due to structural brain abnormalities leading to the development of an epileptic focus when compared to early seizures, which result from acute local brain metabolic alteration in individuals, by cerebrovascular accident.

The incidence of status epilepticus (SE) in post stroke first time seizure patients is 9% at the end of 3.7% years. There is no relationship between the occurrence of SE and stroke risk factors, stroke type, stroke topography, cause and

cortical involvement, size of lesion and seizure type or EEG findings. Status epilepticus occurred more frequently among patients with higher disability rating.

Risk of epilepsy:

The incidence of recurrent seizures (epilepsy) after stroke in a large prospective multicenter study is 2.5%. The possibility of recurrence is greater in late onset (58%) compared with early onset seizures (12%). In a population based study from Rochester, Minnesota, the cumulative probability of developing initial late seizures was 3.0% by 1 year, 4.7% by 2 years, 7.4% by 5 years and 8.9% by 10 years.

Late onset of first seizures is an independent risk factor for epilepsy after Ischemic stroke but not after hemorrhagic stroke. However some studies has recorded high prevalence of late seizures in putaminal and lobar hemorrhage.

MATERIALS AND METHODS

This study was carried out in the Department of General medicine and the Department of Neuromedicine at the Govt Stanley hospital, Chennai, India from August 2005 to Jan 2006. The patients in the age group of 30 to 88 yrs with the following criteria were included in the study.

INCLUSION CRITERIA

1. Diagnosis of stroke
2. With or without seizures

EXCLUSION CRITERIA

1.Previous seizures

2. Previous brain surgery

3. Head trauma

4. Sub arachnoid hemorrhage

5. Aneurysm, tumor

6.AVM related bleed

7. Significant metabolic abnormality

8. Septicemia

All patients were interviewed using a structured proforma. This includes a detailed history regarding stroke, type of stroke, time and nature of onset, associated with seizures, level of consciousness etc.

A detailed past history regarding SHT, DM, RHD were recorded.

A detailed clinical examination was performed.

The biochemical investigations done in these patients include blood sugar, urea, creatinine, serum electrolytes and LFT.

Chest X ray and ECG were taken for all patients.

Computerized tomography scan of brain was done to all patients with special emphasize to look for infarct, hemorrhage and the site of lesion.

EEG was taken for nearly 50% of patients who presented without seizures and eight out of nine patients who presented with seizures.

RESULTS AND DATA ANALYSIS

A total of eighty-one patients who satisfied the inclusion criteria between the ages of 30 and 88 years were included in the study.

17(21%) of the eighty-one patients were females and 64(79%) were males. The patients were grouped based on the CT scan findings.

GROUP	CT FINDINGS
Group I	Infarct in cerebral hemisphere
Group II	Hemorrhage in cerebral hemisphere
Group III	Normal study

There were sixty patients (74%) in group I, seven (8.6%) in group II and fourteen (17.3%) patients in group III

Characteristics of study groups:

The characteristic of study groups is illustrated in the table.

GROUP	I	II	III
Numbers	60	7	14
Male: female	47:13	5:2	12:2
Age Range	40 – 80 yrs	58 – 75 yrs	30 – 86yrs

The distributions of side of involvement of brain in various groups were individually studied and it is shown in the table.

GROUP	I	II	III
Right hemispherical brain lesion	33	3	2
Left hemispherical brain lesion	22	4	12
Bilateral lesion	5	-	-

ANALYSIS OF STROKE PATIENTS WITH SEIZURES

Nine patients (6 males and 3 females) in age range of 47 yrs to 75 yrs (mean age 61 yrs) from 81 patients of stroke who fulfilled the selection criteria had seizures. The incidence of seizures is 11.1%.

5 (55.6%) of the 9 patients who had seizures had infarct in brain, 3(33.3%) patients had hemorrhage in the brain and 1(11.1%) patient showed normal study in CT scan.

None of the 5 patients who showed infarct in CT scan brain had an evidence for embolic stroke (vascular disease, atrial fibrillation and myocardial infarction).

Of the nine patients who had seizures 5 patients showed left sided cortical involvement 3 pts showed right-sided cortical involvement one showed bilateral cortical involvement

All the 5 patients who had an infarct in the brain showed involvement of cortical areas with or without sub cortical region involvement, but no patient showed pure sub cortical lesion on cranial CT.

2 of the hematoma were in the cerebral cortex and 1 was primarly in the capsulo ganglionic region. None of the hematoma showed an evidence of intraventricular extension.

6 patients (67%) had early immediate seizures (i.e. within 24 hrs of onset of stroke and 3 (33%) had late onset seizures. In patients with early immediate seizures 2(33%) of them presented with focal seizures, 2(33%) presented with GTCS, 1 with focal becoming secondary generalized (17%) and one (17%) patient presented with status epilepticus.

Of the 3 patients who presented with late onset seizures 2(67%) had GTCS and 1(33%) had focal seizure, none of 3 patients had history of recurrent seizures.

EEG recordings were normal in 6 of them, 2 of them showed diffuse slowing, and in one patient EEG cannot be recorded. None of the patients showed focal slowing or epileptiform discharges. No specific EEG pattern was seen with early versus late seizures.

DISCUSSION

Until the 1950s, studies of seizures after stroke were largely confined to necropsy studies. The advent of computed tomography (CT) in the 1970s and MRI a decade later markedly increased the accuracy of clinical diagnosis of stroke mechanisms, particularly with respect to distinguishing between ischemic and hemorrhagic infarcts. Nevertheless, comparison among different studies has been difficult because of different inclusion and exclusion criteria.

Fortunately, there have been several recent prospective studies of early, or both early and late, seizures that has followed large populations (more than 500 patients) for an adequate period of time to draw valid conclusions and has attempted to separately analyze clinically important subgroups. The smaller prospective or case-control studies, although less valid as sources of incidence data, still provide useful information about risk factors and clinical characteristics.

Prospective analyses of combined groups have found lower overall seizure rates after ischemic stroke than hemorrhagic stroke. Bladin and colleagues, for example, found that 8.6% of 1,632 patients with ischemic stroke had seizures, versus 10.6% of 265 patients with hemorrhagic stroke. Because of higher mortality among patients with hemorrhagic stroke, survival analysis revealed an almost twofold increased risk of seizures in that group.

Among the patients in this study who had ischemic stroke, 5% had seizures within 24 hours (“onset”). For patients with hemorrhagic stroke, 42% had early seizures. Late seizures occurred with 5% of ischemic strokes, a finding verifying nearly all-earlier studies. In multivariate analysis, cortical location was the only risk factor for seizures after either type of stroke. Increased disability predicted seizures among the ischemic group. Hemorrhagic transformation conferred greater risk on univariate analysis, but a high likelihood of embolism did not, a finding confirmed by a large cohort in the National Institute of Neurological Disorders and Stroke’s Stroke Registry, despite conflicting results in earlier studies and in one large retrospective study.

Lacunar infarction was associated with seizures in 2.6% of cases, although further analysis questioned this relationship. However, risk factors for lacunar disease, including hypertension, serum cholesterol, and left ventricular hypertrophy, have been associated with the development of seizures or epilepsy, even in those without overt stroke.

Kilpatrick and coworkers found early seizures in 24 (4%) of 604 patients with ischemic stroke (or 4.4%, excluding brain stem and cerebellar strokes, as did Bladin et al.). All 24 patients with early seizures had cortical infarcts of the anterior circulation, although again embolism was not a risk factor. So and colleagues found 4.7% onset seizures and 6.2% early (1 week) seizures among those with infarction; initial late seizures occurred in an additional 5% of patients,

and epilepsy in 3.3%. Early seizures were a significant risk factor for late seizures and epilepsy. The cumulative risk for initial late seizures was 7.4% by 5 years and 8.9% by 10 years.

A British prospective study of 675 patients with a first stroke, 545 of whom had infarction, found a 5-year actuarial risk of seizure of 11.5%. Such population-based studies have found seizure risk relative to the general population to be elevated by factors of 20 to 40. Early (2 weeks) seizures in a Danish cohort of 1,197 patients with ischemic infarct occurred in 4.2% of patients, 2.8% within 24 hours. Stroke severity was the only risk factor in a multivariate analysis.

Seizures have been attributed to transient ischemic attack (TIA) in 1–4% of patients. In some earlier studies, the possibility of seizures heralding TIA or stroke was advanced, although this relationship is tenuous.

Smaller studies examining occurrence of late seizures or epilepsy confirm the importance of large cortical infarcts and suggest a role for apparently preserved cerebral tissue within the infarcted area. Extensive white matter disease in combination with cortical infarction is also important.

No specific incidence figures have been published for seizures following border zone infarcts. One would expect that when these infarcts extend to the cortex, there would be risk similar to that of thrombotic lesions. However, the common electroencephalography (EEG) occurrence of periodic lateralizing

epileptiform discharges (PLEDs) after border zone infarcts argues that the frequency of seizures may be higher and that epilepsia partialis continua or other forms of partial status epilepticus might occur.

There are no data concerning seizures with less common stroke mechanisms, such as vasospasm as a result of migraine or vasculitis—only limited evidence linking seizures to specific cortical locations. An excess of seizures with strokes involving the anterior rather than posterior circulation may relate to a higher likelihood of cortical involvement. Within a given vascular territory, the probability of seizures or epilepsy may also relate to the intrinsic epileptogenicity of specific cortical regions. Limited evidence parallels that for brain tumors or unselected patients and suggests the highest probability associated with perirolandic cortex, followed by the temporal lobe, then prefrontal, parietal, and occipital regions.

Specific studies of intracerebral hemorrhages confirm that early seizures are common, occurring in 4.6–17.0%. All studies show lobar hemorrhages to have the highest rates, 15–24% or more. Most of these patients had hypertension. Among those with deep hemorrhages, the rate of early seizures ranges from 0% to 11%. The caudate and perhaps putamen are most often involved, and the thalamus least involved. As in infarctions, the majority of early seizures occur within the first day or two. About half of these patients had one or more seizures as the first symptom. Late seizures and epilepsy are much less common, even in those with early

seizures. Between 2 and 5 years, only 6.5% of survivors had any seizures, in contrast to an observed cumulative incidence of 32%. Furthermore, life-table analysis in the same study suggested a cumulative prevalence of seizures to be 50% had all patients survived to 5 years.

Many patients with lobar hemorrhage in early studies may have had cerebral amyloid angiography, although specific data for seizure risk in this syndrome are not available. Nor are figures available specifically for hemorrhage caused by coagulopathies, but location probably plays an important role here also. In a retrospective study to evaluate the incidence of seizures in chronic subdural hematomas (some of which could result from coagulopathies), the rate of occurrence was less than 2%. After hemorrhage from dural arteriovenous fistulas, 5% of patients presented with generalized seizures.

In general, the incidence of epilepsy as a late sequela of stroke has been estimated at 3% to 10%, with those who have a late-onset seizure at higher risk (a second unprovoked, late-onset seizure by definition constituting epilepsy). Late seizures may occur earlier after hemorrhage than after infarction. The timing of the initial late seizure (after 1–2 weeks) does not predict later recurrence.

Status epilepticus can occur, involving 31% of stroke patients with seizures in one large study. Status epilepticus was the initial seizure type in more than half of these patients.

With respect to hemorrhagic infarcts due to vascular malformations, AVMs commonly (17– 40%) present with seizures. Presentation with a seizure alone is more common in younger patients. The cumulative risk of epilepsy in untreated patients is estimated at 1% per year, relative to a 2–4% risk of hemorrhage. Cavernous angiomas are commonly undiagnosed until a seizure occurs, with 40–70% presenting with seizures, usually in midlife; epilepsy is typical if the lesion is untreated. Venous angiomas rarely bleed and are usually incidental findings, but when hemorrhage occurs, seizures are common.

Subarachnoid hemorrhage presents with a seizure in a sizable minority (6.3–18.0%) of patients. In one study, thickness of the cisternal clot was the only predictive factor. Another 7% may have had presenting seizures, but loss of consciousness at the time of aneurysm rupture more commonly reflects temporary cessation of cerebral perfusion secondary to an acute rise in intracranial pressure. Acute symptomatic seizures may occur in 24–26%, and epilepsy can later develop in 25%, with a higher risk if there are neurologic sequelae and if acute seizures occurred. In a small retrospectively studied cohort with unruptured intracranial aneurysms, the overall risk of postoperative seizures in initially seizure-free patients was 15.7%.

With cerebral venous thrombosis, seizures have been described in 40% of cases, but subsequent epilepsy appears rare, despite the high incidence of hemorrhage.

Acute symptomatic seizures are one of the hallmarks of hypertensive encephalopathy and related conditions (e.g., eclampsia, hyperperfusion syndrome), occurring in the majority. Later epilepsy is rare, however, paralleling the resolution of the often-posterior white matter and cortical lesions typically seen on MRI.

Migraine occurs in 18% of women and 6% of men. The prevalence of migraine with aura (which includes hemiplegic migraine) is lower, around 4%, and only a tiny fraction of patients ever experience the neuroimaging changes or persistence of symptoms for more than 1 week that defines migrainous infarction. No data are available on the frequency of seizures or epilepsy as a complication of this cause of stroke.

CONCLUSION

1. Post stroke early onset seizures occur within two weeks of stroke onset, while late onset seizures occur after two weeks.

2. The incidence of seizures in this study is 11.1%

3. The incidence of early onset seizures is 7.4%

4. The incidence of late onset seizures is 3.7%

5. The incidence of focal seizures in early onset post stroke seizures is 33%

6. The incidence of GTCS (including status epilepticus) in early onset post stroke seizures is 50%

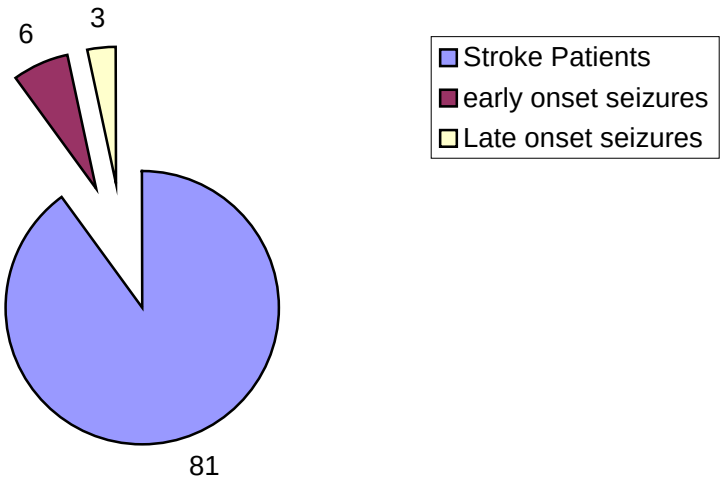
7. EEG recordings were normal in 78% of patients while 22% showed diffuse slowing.

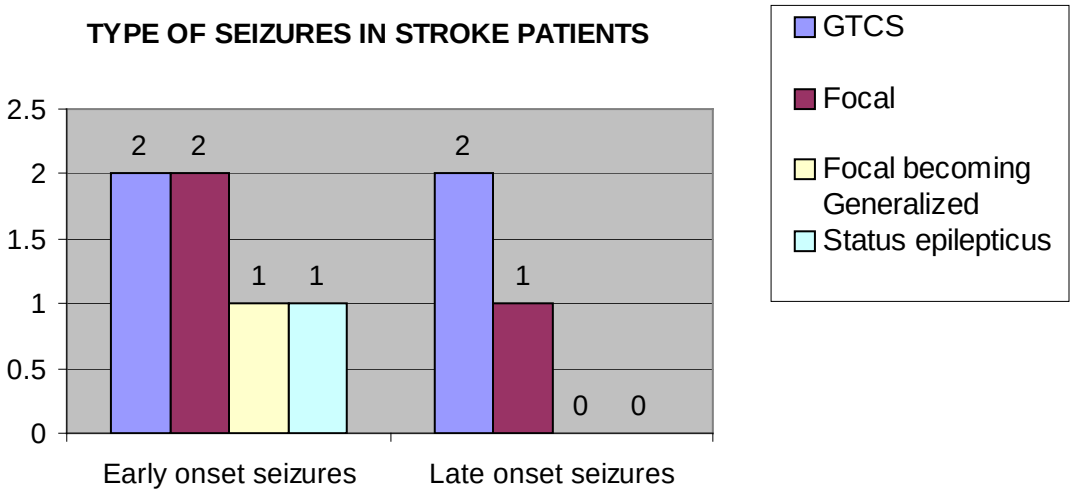
8. The involvement of cerebral cortex has been emphasized in the pathogenesis of epilepsy caused by stroke. In the present study 100% of infarctions leading to seizure involved cerebral cortex with or without involvement of subcortical region.

9. 67% of hematomas were localized exclusively to the cortical region.

10. 55% of patients showed left side cortical involvement while 33.3% showed right side cortical involvement, and 11.1% showed bilateral involvement.

INCIDENCE OF SEIZURES





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MASTER CHART INCIDENCE OF SEIZURES IN STROKE

S.no	Name	Age	Sex	Stroke side	Seizure	EEG	CT Brain	RFT S/ U/ Cr
1	Prema	45	F	LT Hemiplega	Nil	Normal	RT MCA infarct (Embolic)	116/ 28/ 1.0
2	Logambal	78	F	LT Hemiplegia	Nil	Normal	RT MCA infarct	122/32/1.2
3	Ganesan	55	M	LT Hemiplegia	Nil	Normal	RT Parietal infarct	136/26/1.0
4	Abdul Khadir	50	M	RT Hemiparesis	Nil		LT Parietal infarct	92/32/0.8
5	Subramani	70	M	RT Facio brachio Monoplegia	Nil		Normal study	140/28/1.0
6	Jagadesan	62	M	RT Hemiparesis	RT Focal seizures	Normal	LT Parietal infarct	102/26/1.08
7	Anvar Bashah	55	M	RT Hemiplegia	Nil		LT MCA infarct	112/32/0.9
8	Durai	70	M	LT Hemiparesis	Nil		RT Parietal infarct	66/28/0.9
9	Chinnaponnu	40	F	LT Hemiplegia	Nil		RT Corona radiata infarcts	140/35/1.0
10	Jana	48	M	RT ataxic hemiparesis with VII, IX, X, N. palsy	Nil		Atrophic changes	110/24/0.8
11	Ramanujam	64	M	LT Hemiplegia	Nil		RT MCA infarct	124/33/1.1

S.no	Name	Age	Sex	Stroke side	Seizure	EEG	CT Brain	RFT S/ U/ Cr
12	Ganesan	47	M	RT Facio brachial Monoplegia	RT Focal seizures	Normal	Normal	120/22/0.8
13	Rajendran	50	M	LT Hemiplegia with LT III N.palsy	Nil	Normal	Multiple infarcts	132/38/0.9
14	Rosemary	60	F	LT Hemiparesis	Nil	Normal	RT temporoparietal infarct	161/25/0.6
15	Abdul Azeez	60	M	RT Hemiparesis with RT UMN facial palsy	Nil		Normal	116/35/0.9
16	John	40	M	RT Hemiparesis	Nil		LT MCA infarct	80/29/0.8
17	Anandaraj	50	M	RT Hemiparesis with RT UMN facial palsy	Nil	Diffuse slowing of discharges	Normal	75/22/0.8
18	Chellaiah	65	M	LT Hemiplegia	Nil		RT MCA infarct	172/18/0.8
19	Angammal	60	F	LT side Hemiparesis	Nil	Normal	RT parietal infarcts	100/88/0.4
20	Srinivasan	55	M	RT Hemiparesis	Nil	Normal	Normal	122/28/0.9
21	Ramalingam	44	M	RT Hemiparesis	Nil	Normal	LT MCA infarct	102/32/0.8
22	Balfour	72	M	LT Hemiplegia	Nil		ICH – RT thalamus with extn. into lat.vent.	82/24/1.0
23	Arumaikannu	47	M	RT Hemiplegia with RT UMN facial palsy	Nil	Normal	LT MCA infarct	132/32/0.9
24	Jayaraman	55	M	RT Hemiparesis	Nil	Normal	LT MCA infarct	110/40/1.0

S.no	Name	Age	Sex	Stroke side	Seizure	EEG	CT Brain	RFT S/ U/ Cr
25	Kannadasan	53	M	RT Hemiplegia with RT UMN facial palsy	Nil	Normal	LT MCA infarct	122/32/0.9
26	Ranganathan	60	M	LT Hemiplegia	Nil		RT Corona radiata infarct	110/33/1.0
27	Munusamy	46	M	LT Hemiplegia	Nil	Normal	RT MCA infarct	122/22/1.0
28	Munusamy	58	M	LT Hemiparesis	Nil		RT MCA infarct	88/36/1.2
29	Nandagopal	47	M	LT Hemiparesis	Nil	Normal	RT MCA infarct	156/32/1.1
30	Devika	45	F`	LT Hemiparesis	Nil	Normal	RT MCA infarct	138/28/0.7
31	Lakshmi	60	F	LT Hemiparesis	Nil		Normal study	92/40/0.9
32	Sammuelraj	51	M	LT Hemiparesis	Nil	Normal	RT MCA infarct	188/30/1.0
33	Rathinam	61	M	LT Hemiparesis	Nil		RT MCA infarct	162/52/1.3
34	Rajendran	55	M	RT Hemiplegia	Nil	Normal	LT MCA infarct	111/42/1.1
35	Subramani	60	M	RT Hemiparesis	Nil		Normal study	162/38/1.0
36	Narayanan	70	M	LT Hemiparesis	Nil		RT parietal infarct	116/28/1.0
37	Dayalan	57	M	LT Hemiplegia	GTCS	Diffuse slowing of discharges	Infarct in LT MCA territory Infarct in RT lentiform N Infarct in parieto occipital (old)	128/32/1.1
38	Subramani	48	M	LT Hemiplegia with LT UMN facial palsy	Nil		RT occipital infarct	78/38/1.2
39	Mandurajan	60	M	LT Hemiplegia	Nil	Normal	Large ICH with Intravent.extn.	129/19/1.0

S.no	Name	Age	Sex	Stroke side	Seizure	EEG	CT Brain	RFT S/ U/ Cr
40	Ramasamy	75	M	LT Hemiplegia	Nil		Lacunar infarct RT ext capsule Infarct RT corona radiate and thalamus	112/29/1.0
41	Pattabi	55	M	RT Hemiparesis with aphasia	Nil	Normal	LT MCA infarct	106/36/0.9
42	Mahendran	43	M	LT Hemiplegia with aphasia	Nil	Normal	MRI- PICA, s.cerebrellar territory infarct (Embolic)	112/36/1.1
43	Krishnan	60	M	RT Hemiparesis	Nil		Normal study	116/49/1.2
44	Helen	60	F	RT Hemiplegia	GTCS (15 mts) at onset	Normal	ICH, LT Parieto occipital region	129/300/1.0
45	Dhakshnamoorty	61	M	LT Hemiplegia with LT UMN VII N.palsy	Nil		Multiple infarct	90/22/0.8
46	Gopinath	60	M	LT Hemiparesis	Nil	Normal	RT MCA infarct	76/41/1.0
47	Sheikh Ibrahim	62	M	RT Hemiparesis	RT focal with sec. generalisation	Diffuse slowing of discharges	RT Parieto occipital infarct	159/26/1.0
48	Angamma	70	F	RT Hemiplegia and RT UMN palsy	Nil		LT Parietal infarct	96/26/0.9

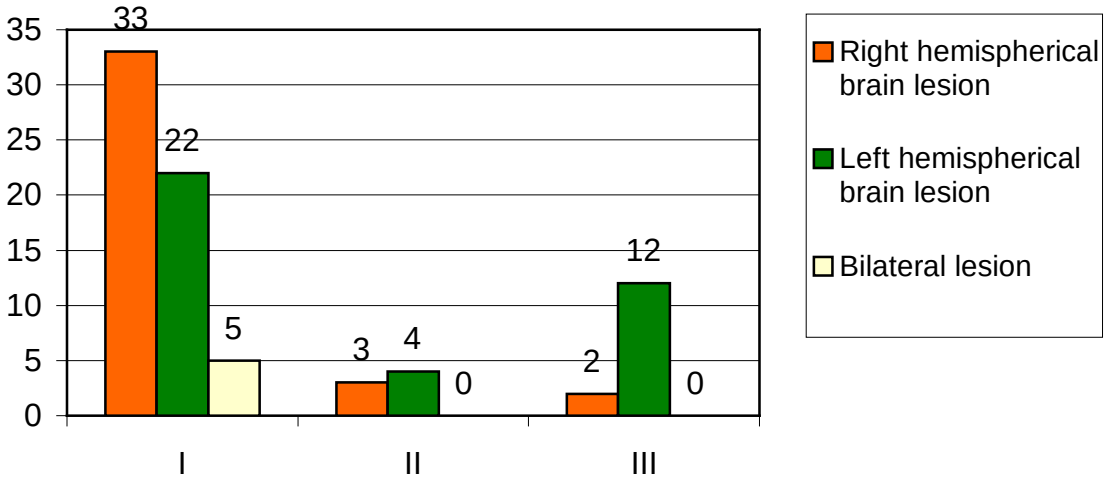
S.no	Name	Age	Sex	Stroke side	Seizure	EEG	CT Brain	RFT S/ U/ Cr
49	Ponnammal	58	F	RT Hemiplegia	Nil		LT Cerebellar hge	108/28/0.8
50	Chakkubai	62	F	Old LT Hemiparesis	GTCS	Normal	Old infarct with atrophic changes	121/37/1.2
51	Shakila	55	F	RT Hemiparesis	Nil	Normal	RT Parietal infarct	151/41/1.1
52	Mani	45	M	RT Hemiplegia	Nil	Normal	LT MCA infarct with perilesional edema	97/22/0.8
53	Raman	80	M	RT Hemiparesis	Nil		Normal study	102/22/0.8
54	Natarajan	65	M	RT Hemiparesis with aphasia	Nil	Normal	LT Parietal infarct	140/25/0.8
55	Venkatesan	30	M	RT facio brachial monoparesis	Nil	Normal	Normal study	128/22/0.8
56	Vardhan	53	M	LT Hemiparesis	Nil	Normal	RT Capsulo ganglionic infarct	82/19/0.8
57	Sukumar	50	M	RT Hemiparesis with loss of vision	Nil	Normal	B/L occipital infarct	170/32/0.9
58	David	52	M	RT Hemiparesis	Nil	Normal	LT MCA infarct	102/32/0.8
59	Annamalai	65	M	LT Hemiparesis	Nil		RT MCA infarct	168/23/0.9
60	Chinnaiya	65	M	LT Hemiparesis with UMN facial palsy	GTCS	Normal	RT Capsulo ganglionic hemorrhage	82/19/0.8
61	Krishnan	70	M	LT Hemiparesis	Nil	Normal	RT MCA infarct	152/42/1.2
62	Munusamy	48	M	RT Hemiparesis	Nil		LT MCA infarct	110/30/0.8
63	Krishnaveni	65	M	LT Hemiparesis	Nil		Normal study	72/22/0.8

S.no	Name	Age	Sex	Stroke side	Seizure	EEG	CT Brain	RFT S/ U/ Cr
				with VII N				
64	Ramaiah	80	M	LT Hemiparesis	Nil		RT MCA infarct	142/38/0.4
65	Murugesan	70	M	RT Hemiplegia with aphasia	Nil		LT MCA infarct	180/28/1.0
66	Chithiram	58	M	RT Hemiparesis	Nil	Normal	LT MCA infarct	96/40/1.0
67	Karim Bashah	70	M	RT Hemiplegia	Nil	Diffuse slowing of discharges	ICH LT cerebral hemisphere	92/41/0.8
68	Govindammal	60	F	LT Hemiplegia	Nil		RT MCA infarct	86/39/1.0
69	Jeslina	65	F	LT Hemiparesis	Nil		RT MCA infarct	102/42/1.1
70	Ruckmani	57	F	LT Hemiparesis with LT UMN facial weakness	Nil	Normal	Cortical atrophy	132/19/1.0
71	Thirunavukarasu	60	M	RT Hemiplegia	Nil		LT MCA infarct	65/48/0.8
72	Kannan	58	M	RT Hemiparesis with RT UMN facial palsy	Nil	Normal	LT MCA infarct	116/39/1.1
73	Anandhan	50	M	LT Hemiparesis with LT UMN facial palsy	Nil	Normal	Infarct RT centrum semi ovale Corona radiata Capsulo ganglionac reg.	88/32/1.1
74	Arumugam	75	M	RT Hemi paresis RT UMN facial palsy	Status epileptus		LT ICH	162/42/1.1

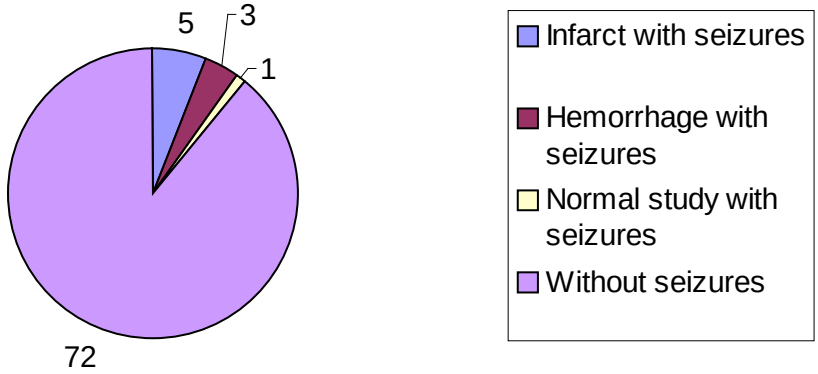
S.no	Name	Age	Sex	Stroke side	Seizure	EEG	CT Brain	RFT S/ U/ Cr
75	Alagammal	59	F	RT Hemi paresis old	RT focal seizure	Normal	Old LT MCA infarct New RT MCA infarct	118/28/1.1
76	Jayaraman	58	M	RT Hemi paresis	Nil	Normal	Lacunar infarct LT side	83/37/0.8
77	Sivasamy	50	M	LT Hemiparesis with LT UMN palsy	Nil	Normal	RT MCA infarct	128/38/0.9
78	Chandrasekar	52	M	LT Hemiplegia	Nil	Normal	RT MCA infarct	175/26/1.1
79	Rajendran	52	M	RT Hemi paresis	Nil		LT MCA infarct	108/28/0.9
80	Duraisamy	50	M	RT side Hemi paresis Old LT Hemi paresis	Nil	Normal	Atrophic changes LT MCA infarct	142/32/0.8
81	Ramu	56	M	RT Hemiplegia	Nil		LT occ. infarct	116/35/1.1

S-Blood Sugar, U-Blood urea, Cr-Serum Creatinine

INVOLVEMENT OF BRAIN IN THE STUDY GROUP



CT SCAN FINDINGS IN POST STROKE SEIZURE PATIENTS



APPENDIX
PROFORMA

NAME
AGE/SEX
D.O.A
WARD

Clinical findings:

Stroke –Onset
Progression
Side of weakness
Associated findings

Seizures –Onset
Type of seizure
-Focal
-Generalized
-Status epilepticus

General examination

Consciousness
Temperature
Anemia
Icterus
Cyanosis
Pedal edema

PULSE
BLOOD PRESSURE
RESPIRATORY RATE

CNS examination

Higher functions
Cranial N. examination
Sensory system examination
Motor system examination
Cerebellar signs

CVS examination

Apical impulse
Heart sounds
Murmur

Resp sys examination

Tracheal position
Chest movements /Deformity
Breath sounds
Added sounds

Abdominal examination

INVESTIGATION

Blood SUGAR
Blood urea
Serum creatinine
Serum electrolytes

Liver function test

Chest x ray

Electrocardiogram

Electroencephalogram

Computerized tomography scan- Brain