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CLINICAL CORRELATION WITH CT SCAN BRAIN FINDINGS

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CERTIFICATE

This is to certify that this dissertation entitled **"STUDY ON CVA - CLINICAL CORRELATION WITH CT SCAN BRAIN FINDINGS"** is the bonafide record work done by **Dr. R. BALAMURUGAN,** submitted as partial fulfillment for the requirements of M.D. Degree Examinations, General Medicine (Branch I) to be held in September 2006.

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CONTENTS

	Page No.
1. INTRODUCTION	7
2. AIM OF THE STUDY	9
3. REVIEW OF LITERATURE	11
4. MATERIALS AND METHODS	42
5. RESULTS AND OBSERVATIONS	50
6. DISCUSSION	69
7. CONCLUSION	8

BIBLIOGRAPHY

PROFORMA

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INTRODUCTION

Stroke / CVA [16] is defined as abrubt onset of neurological deficit due to a focal vascular cause. The incidence of stroke increases with age and they cause about 200000 deaths [29] each year and was a major cause of disability.

Early diagnosis and treatment is necessary to prevent mortality and morbidity.[19] Strokes mimics are to be differentiated from stroke to avoid inappropriate treatment. Though there are many investigations, CT and MRI [17] play major role in the diagnosis of CVA.

Though MRI is superior to CT scan, the higher cost of the MRI, [17] and the easy availability of CT scan brain makes CT the commonest investigation in diagnosis and treatment of stroke.

CT [23] plays a major role in CVA to assess the site, size and nature of the lesion. This study is done in Thanjavur Medical College, during the period of 2004 to 2006, in inpatients admitted for CVA and, tried to correlate the clinical features with CT findings.

AIM OF THE STUDY

1. To study the role of CT Scan Brain in the Diagnosis of CVA.

2. To correlate the nature of lesion on clinical grounds with CT Scan findings.

3. To correlate the site of lesion on clinical grounds with CT scan findings.

4. To correlate the severity of lesion on clinical grounds with CT scan findings.

REVIEW OF LITERATURE

The diagnosis of cerebrovascular disease is historically more ancient than that of heart disease. Apoplexy was a recognized clinical syndrome and cause of death before the time of Hippocrates. Morgagni (1682-1771) studied CVD extensively in the mid 18th Century and by the beginning of the 20th Century cerebral haemorrhage, thrombosis and embolism were recognized diagnoses.

The tight revision of the International statistical classification of disease (WHO:1967) came into use in 1968. Many writers emphasized (Marshall Shaw 1960: Riishide 1967, 1968, leading Article 1968) that clinical signs and symptoms alone cannot establish an exact diagnosis, nor even separate infarction from hemorrhage. [31] Jane-way and Toole (1968) believed that a thorough examination which includes readily available lab tests permit the correct diagnosis of 90% of cases, while Resch (1966) stressed the need for more complex diagnostic procedures for the remaining 10%. Further revision of classification was made by the WHO in 1977 (9th revision).

The visualization of cerebral vessels by the radio contrast medium method was introduced into clinical neurology by the Protugeese neurologist Egas Moniz in 1927. This led to the better understanding of the pathological aspects of the vascular disorder. In 1934, Moniz went into great detail in a discussion of the four angiographic phases-the arterial, capillary the first and second venous phases.

The vascular supply of arteriovenous malformations was visualized in a masterful fashion by Tonnis. This was the beginning of the intensive and fruitful work in the field of cerebral angiography.

In the following years, neuro radiology was developed and perfected with far reaching effect at the clinic of Olivecrona by Lindgren Hemmingson and Wickborn and at the Clinic of Trokildesn by Engeset and Kristiansen.

In 1937, Castellanos et al first described the concept of eliminating unwanted background from angiographic images-the Digital substraction Angiography (DSA). This was clinically applied to a large number of patients in 1938 by Robb and Steinberg.

In 1981, Weinstern studied the results in 10 Digital angiographic examinations of the carotid arteries. He found that the major limitations of IV DSA (intra venous DSA) were artifacts produced by swallowing and subtraction misregistration.

Ackerman R.H (1979) described non-invasive diagnosis of carotid disease – Doppler imaging with carotid ultrasound examination.

CT scan (Computed tomography) since its inception in 1972 (Hounds field) has become vital for proper current management of many neurologic problems including cerebrovascular insufficiency. Four generations of CT scanners have evolved since then based on the scan time per tomographic slice. CT and angiographic studies have demonstrated that the arterial distribution of infarct revealed by CT may not correlate with the angiographic level of lesion, thus showing that CT scan and angiography are complementary. [23]

CT Scan changes in infarction can be divided into three Stages:

(i) Acute,

(ii) Subacute, and

(iii) Chronic.

Acute Stage – increased water content is the hall mark, being a the low-density lesion the CT Scan correlate in most instances. With acute infarcts, no contrast media need to be given.

After the acute stage, contrast enhancement will be helpful in demonstrating a small number of infarcts otherwise undetectable – probably attributable to extravasation of the iodinated compound through new capillaries with immature “tight-junctions”. However, patterns of enhancement are not pathognomonic and can be mistaken for other lesions.

Subacute Stage-CT Scan reveals a lesion of lower density with less mass effect and with more clearly defined margins that during the acute stage.

The chronic stage is reached when the resorption of necrotic tissue is complete.

The location of cerebral infarction detected by CT is most commonly in the middle cerebral territory. In one large series, the arterial areas were as follows: 74% MCA, 17% PCA, 3% ACA, 1.5% Cerebellar and 3% unclassified. [23]

Hemorrhagic infarction is identified by areas of confluent hemorrhage. This can be distinguished from intracerebral hematomas by their dense and heterogenous scan pattern indicative of bleeding within the brain substance. Development of hemorrhagic infarction depends upon incomplete or transient disruption of the blood supply to the infarcted area as well as ischemic damage to the endothelial cells allowing diapedesis of erythrocytes.

Neurological examination together with vascular examination forms the initial step in the evaluation of stroke. It has been emphasized that the clinical presentation alone cannot establish a diagnosis nor even differentiate infarction from hemorrhage presented with a sudden neurologic deficit. Headache was complained of at the onset, in 100% of cerebral haemorrhages while vomiting in only 40%.

Heyman et al [39] (1980) state that the carotid bruit may be more of an indicator of generalized atherosclerosis than a direct inciting cause of impending stroke.

Recent advances in the evaluation of stroke

A recent advancement in computed Tomography is the Incremental Dynamic Scanning which permits the making of certain specific observations of blood flow and vascular morphology together.

Nuclear Magnetic Resonance Imaging [27] (Lauterbur, 1973) a non-invasive diagnostic technique is the focus of rapid technical research and development.

With the introduction of the varied diagnostic techniques in the neurosurgical procedures-Carotid endarterectomy and Extra-carnial-Intracranial anastomosis, the fact that many did not benefit from surgery, led to the study on Cerebral hemodynamics and metabolism utilizing conventional nuclear medicine detection and imaging devices-Positron Emission Tomography.

Discriminating among Infarct subtype of clinical grounds alone is difficult. To determine some of these distinguishing clinical features in Stroke Data Bank, investigators Compared demographic, stroke risk, clinical and radiologic features for 246 cardioembolic cerebral infarcts.

Noninvasive transcranial Doppler ultrasonography, Incremental Dynamic Computed Tomogrphy and Magnetic Resonance Imaging are fast advancing among the Diagnostics.

The intracranial vessels may be examined by the Doppler technique using a 2-MHz probe that provides bone penetration. Lawrence R. Wechsler et al conducted a preliminary trial in 32 normal persons and 11 patients with cerebrovascular disorders. Clinical and angiographic evidence of MCA vasospasm or stenosis was associated with increased peak velocity and spectral broadening of the Doppler signal.

Intracranial stenoses of the ICA and MCA were diagnosed by way of transtemporal and trans orbital ultrasonic recording with angiographic control in 16 patients by E.B. Ringelsterm et al.

It is clearly evident that transcranial Doppler sonography is a valuable tool in clinical neuro angiology.

Incremental Dynamic CT is done after injection of 50 – 80 ml of 76% urografin. 12 – 16 axial scans and paraxial images are reformatted alone the axis of the internal and external carotid arteries at intervals of 0.5 – 1.5 mm. The incremental study is done with increments of 3 mm, a scan duration of 2 seconds and interscan intervals of 2.3 seconds.

CT of the carotid bifurcation holds promise for non invasive assessment.

When MRI becomes more widely available, it may become the imaging procedure of choice for the diagnostic evaluation of ischemic stroke. With improved computer MRI softer, there is potential to demonstrate within six hours, [17]

(1) Accumulation of tissue water,

(2) Vascular transit time,

(3) Tissue perfusion and metabolism and

(4) Measurements of intracellular and extracellular water and electrolytes both in the core of the infarction and in the surrounding penumbra.

(5) Because of its cost and easy repeatability CT scan remains still the investigation of choice.

Promising Therapeutic Advances

During cerebral angiography interventional neuroradiologists are now able to deliver thrombolytic therapy. Vascular stenoses have been successfully dilated using intravascular inflatable balloons.

Treatment of acute stroke can change dramatically by the end of the decade. This means emergency diagnostic evaluation and treatment should be available to all stroke victims so as to reduce the morbidity and cost factors in caring for survivors.

PHYSIOPATHOLOGY:

The cerebral arterial circulation differs from the peripheral circulation in the following manners:

a) Cerebral blood supply is not under autonomic control and it has sufficient capacity of autoregulation that enables the blood flow to adjust the metabolic demands despite marked alterations in the systolic pressure-range of Mean arterial blood varies from 70 mm of Hg to more than 150 mm of Hg.

b) The cerebral circulation is a higher-pressure system at small vessel level than the peripheral circulation in the limbs.

c) The main cerebral vessels are short and directly supply the brain enroute and no other areas drain this supply.

d) There are built in anastomoses or alternative pathways at different levels, circle of willis, proximally pre willisian anastomoses and distal post willisian anastomoses and also at pia level.

Understanding of recent concepts of 'Luxury perfusion' [65] and 'Intra cerebral steal' [35] ('Obrien, 1972), provides rationalization of clinical symptoms, presentation and possible therapeutic approach in cases with interfered cerebral circulation.

In the normal arterial tree, a single arterial input divides into two, supplies two adjacent cones (segments) of tissues. Assuming an equal pressure in both limbs of the artery, the interface between the supply of the two vessels will be half-way, however, if the pressure falls in one artery but not in the other, the interface will move towards the side of the reduced pressure. There is a limit to which this would occur before the pressure drops across the overlapping zone was sufficient to reduce the perfusion to below that required for life; tissue distal to this would then be totally ischaemic.

Following an acute infarction causing a completed stroke, there is a central ischaemic area surrounded by zone of reactive hyperaemia. This area of reactive hyperaemia has caused considerable interest recently because the vessels become fully dilated as a result of ischaemia and the local release of metabolites and they have therefore partly lost their autoregulatory ability, in that further dilatation is not possible and in this area blood flow is pressure dependent.

If the perfusion pressure is adequate, the blood flow is high and exceeds the metabolic demands of the tissue, a situation which has been called ‘the luxury perfusion syndrome’ by Lassen [65] (1966) and which accounts for the red vein phenomenon often seen by neurosurgeons as well as the early filling veins sometimes seen on angiography. This phenomenon is acute and lasts for a short while as long as autoregulation is disrupted and is not seen in chronic stages.

The perfusion pressure modifies the flow through the autoregulated zone both in the acute and chronic stages of an ischaemic episode. During acute phase a redistribution of blood takes place, there is increase of flow in the adjacent normal region of the brain due to vaso dilatation and this bringe reduction of flow in the ‘disauto-regulated zone’. This situation has been called the ‘intra-cerebral steal syndrome’[65] perfusion pressure at this stage is thus crucial for the final outcome and high systemic pressure may be optimized in disautoregulated areas for sustaining its adequate blood supply. Hypotension may thus be ominous while mild hypertension would be beneficial.

PATHOGENESIS:

Quite contrary to the earlier belief, the underlying vascular disease in a majority of cases of (cerebral thrombosis) even in young subjects, is atherosclerosis, rather than arteritis.

Atherosclerosis is a non-inflammatory degenerative disease affecting large and medium sized arteries. In arteritis (syphilitic, tuberculous, collagen disease etc.) the thrombi form on the surface of the diseased intima. Hypertension appears to aggravate the process.

The location and size of the cerebral infarction will reflect upon the clinical manifestations. For example, sylvian region is mostly involved in middle cerebral artery occlusion (Mid-field infarct), [16] whereas in the carotid artery lesions the cerebral infarctions are mostly located in distal or watershed territory ('end-field infarct'). [16] A mid field infarct produced by occlusion of a small penetrating vessel, with poor collateral circulation in the neighbourhood, and if located in the territory of major anatomical pathways, may prove more catastrophic, whereas an end-field infarct resulting from occlusion of a major vessel in the neck (e.g. carotid artery block) with good collateral circulation, may be entirely asymptomatic.

One must differentiate the 'stroke-syndrome' [16] resulting from vascular disease from other non-vascular clinical disorders mimicking as stroke. Decision must be taken about the site and extent of the cerebral damage and whether it is ischaemic vascular disease or haemorrhagic and if possible the underlying vascular disease. Detailed analysis of various symptoms signs and precipitating factors, meticulous physical examination and intelligent serial observation will help a great deal in arriving at a diagnosis and prognosis.

Un-doubtedly, the most characteristic feature of a stroke is it's temporal profile. The speed of evolution of the clinical features varies from a few seconds, minutes or rarely hours, to atmost a few days.

Sudden onset characterizes strokes from embolism and ruptured aneurysm, the deficit reaching its maximum almost immediately.

Thrombotic strokes are also usually sudden in onset, but may develop over a period of several days usually progressing in a stepwise fashion i.e. in a series of sudden changes.

In Parenchymatous Haemorrhage, the deficit evolves smoothly over minutes or hours. Rapid reversal of the deficit may occur from ischaemic cause but never in haemorrhage.

Later, in the course of a non-fatal stroke, stabilisation occur, and is followed by some degree of improvement, especially in cases with deficits in language function. The improvement is gradual, taking place over weeks and months.

Neurological deficit in a stroke reflects both the location and size of the infarct or haemorrhage in the brain. Hemiplegia is the classic sign of vascular disease and occurs with strokes involving the cerebral hemisphere or the brain stem. However depending on its location, a stroke may also give rise to many other manifestations accompanying or independent of hemiplegia, including numbness, sensory deficit, dysphasia, blindness, diplopia, dizziness and dysarthria and sometimes minor infarcts called lacunar infarcts.

Strokes are often preceded by transient ischaemic attacks in many patients irrespective of the underlying pathological process at one time or the other. It may be transient symptoms or minor spells either in a single episode or in the form of multiple attacks. These episodes may have been trivial or bothersome. By and large the neurological deficit, if any, clears up virtually completely within 24 hrs. and may give the patient a false sense of security. With timely investigations after identification of these cases and appropriate treatment, it might be possible to avert a major catastrophic event in a great many.

In the carotid territory, it may manifest in the form of transient impairment of intellectual function with varying degree of speech difficulties. Visual difficulties i.e. transient unclear sight, misty or foggy vision or frank mono ocular blindness may be the presenting symptoms in some instances. Transient sensory disturbances like tingling-parasthesia on one half of the body, may be accompanied by focal paresis, and this may bring the patient for medical opinion. Often these early symptoms are taken to indicate a minor indisposition and in a known hypertensive or a diabetic attributed to irregularity in medication as prescribed.

In the vertebro-basilar territory, the episodes are often preceded by vertigo, characterized by a sense of rotation or swaying (subjective or objective), and often it is accompanied by tinnitus or transient deafness with a feeling of nausea or vomiting. Vertigo accompanied by visual, speech or sensory alterations calls for immediate medical attention. Such episodes may remain trivial and recurrent or become major and serious with objective neurological deficit.

Though the exact mechanism for these transient episodes is ill-understood during the episodes of monocular blindness, Opaque white intravascular masses (fibrino-platelet plugs) have been documented, and it has been presumed that temporary arrest of carotid or vertebro, basilar circulation by such platelet-fibrin embolic plugs may have been responsible for the above episodes. Haemodynamic crises, disturbances in homeostasis, failure of collateral circulation and other modifying factors have been considered as alternative possibilities. The search for the source of embolisation-heart, atheromatous plaques in the aorta or vertebral or carotid artery should be diligently made.

STROKE IN EVOLUTION:

This refers to the speed with which the symptoms and the neurological deficit evolve. For example, the patient may wake up with numbness and clumsiness in one hand with no significant deterioration or progression on that day, the next day, however, he may notice that his right hand is much weaker and he may experience some difficulty with speech, and on the third day, he may wake up with a complete right sided hemiplegia with aphasia. Thus, the neurological deficit evolved is slow, and in a step wise fashion, and hence the term ‘stroke in evolution’ [16] or thrombosis in evolution.

Hence it is presumed that the cerebral blood flow is being reduced in a graded fashion as a result of a building thrombus leading to nearly complete occlusion of the blood vessel and probably from inadequate collateral circulation of this region.

Clinical syndromes are mainly in the carotid, middle cerebral territory or in the vertebro basilar territory. Specific neurovascular syndromes resulting from occlusion of anterior cerebral artery, posterior cerebral artery and lateral medullary artery (Wallenberg's syndrome) [20] are not uncommon.

In cerebral infarction, prodromal warning symptoms, relative sudden onset of a focal neurological deficits and intermittent progression of signs (temporal profile), a constellation of symptoms and signs nearly diagnostic of a well defined neurovascular syndrome, relative preservation of consciousness, clear cerebrospinal fluid, some degree of recovery and vascular disease elsewhere in the body (absent carotid pulsations or bruits over major vessels, history of angina pectoria / myocardial ischaemia or a previous stroke, intermittent claudication in the legs with feeble peripheral pulsations etc.) are the principal hallmarks or cerebral infarction from thromobo-embolism. In distinguishing cerebral haemorrhage from cerebral infarction, the following collective criteria though not always diagnostic, may prove helpful in suspecting an intracerebral bleed. [65] These are:

1. Moderately severe to intense headache (throbbing, pulsating or pounding) in a known hypertensive subject, accompanied by nausea and vomiting.
2. Altered state of consciousness (drowsiness progressing to deepening coma) with irregular respirations.
3. Neck stiffness, accompanied by dissociated eye movements, or forced gaze deviation.
4. Blurred disc margins or pre-retinal haemorrhage with changes suggestive of hypertensive retinopathy.
5. Hemiparesis on one side with shivering movements or even frank convulsions on the non paralysed side, or quadriparesis (paucity of withdrawal movements to pinch stimuli in each limb) with an extensor planter on both sides etc. Of course, the presence of hypertension, and blood in the C.S.F. confirm the diagnosis. Sometimes blood may be absent in C.S.F. in those cases CT scan 4 vessel angiography will be useful.

Alternatively, intra-hemispheric hematomas and the cerebral oedema, caused by massive hemispheric infarctions acting as mass lesion producing trans-tentorial herniation and secondary vascular damage to the diencephalons and brain stem. Whatever the cause, patients in mortal danger in the first few days after a stroke can be recognized by few cardinal signs. The prognostic significance of these signs is enhanced when they appear together (Marquardsen [18] 1969, Oxbury et al 1975).

Clinical features useful in predicting early death after an acute stroke: [22]

Depressed level of consciousness (on admission)

Pupillary abnormalities (loss of light reflex)

Paralysis of conjugate ocular movements (lateral eye deviation)

Bilateral extensor plantar

Respiratory abnormalities (cheyne stokes breathing)

syndrome of acute stroke: [16]

The presenting syndrome seen in most patients with an acute stroke can be described by observing the presence or absence of five basic neurological deficits which are defined in the following table:

Motor Hemideficit	Hemiperesis, Hemiplegia, or unilateral central facial weakness.
Simple sensory deficit	Loss or diminution of pin prick and or light tough sensation
Motor Hemideficit with cortical deficits	Homonymous visual field defect in confrontation.
Higher cerebral Dysfunction	Aphasia and / or parietal deficit as defined by sensory or visual in attention, visuospatial neglect or joint position sense loss.
Brain stem deficit	Ataxia, vertigo dysarthria (without aphasia) and oculomotor palsy (except conjugate deviation) with or without motor or simple sensory hemideficit but without higher cerebral dysfunction.

Patients of middle and younger age group have a fair prognosis. Associated lesions like myocardial ischaemia and renal failure may lead to poor outcome.

This forecast is not only useful to ascertain the immediate mortality and functional recovery in the survivors, but also very much useful in the rehabilitation programmes of these stroke victims.

Diaschisis and Stroke: [13]

Diaschisis refers to functional deactivation of morphologically intact brain regions remote from but connected to an area of primary structural damage. The term coined by von Monakow [4] in 1914 is derived from the Greek schizein meaning *"to spilt"*. [6] The three general types of diaschisis that have been recognized are

1) Contralateral effects on the opposite hemisphere in the mirror regions to the injury (interhemispheric, transhemispheric or transcallosal diaschisis);
2) Ipsilateral effects on the injured cerebral hemisphere but separate from the injury; and
3) Effects on the cerebellum contralateral to the affected hemisphere (crossed cerebellar diaschisis [CCD]

I Interhemispheric Diaschisis

Depression of rCBF, metabolism in the contraalateral hemisphere, or both after cerebral infarction has been recognized in numerous studies. [5] It has been attributed to transhemispheric diaschisis, and the mechanism is thought to be transcallosal neuronal depression. Transhemispheric diaschisis usually occurs in association with large cerebral infarcts, and the extent of rCBF or metabolic reduction is proportional to but less than that occurring in the infarct region.

II Subcortical-Cortical Diaschisis

In patients with lesions of subcortical white matter, basal ganglia, or thalamus, depression of rCBF and metabolism has been found in the ipsilateral overlying cortex. The depression is thought to be due to interruption of neural connections between subcortical structures and cortex and, therefore, to represent diaschisis. This form of diaschisis has been found to correlate with the presence of cortical deficits such as aphasia, neglect, visual field defects, visuospatial abnormalities, and other neuropsychological deficits associated with subcortical lesions. [2] One specific and well-recognsized example is the aphasia seen with thalamic lesions that is associated with thalamocortical diaschisis.

III Cortical-Subcortical Diaschisis

The reverse process of the preceding condition has also been reported. In patients with hemispheric cortical infarction, remote effects have been observed in the structurally intact ipsilateral thalamus. [7] Although this finding might be due to decreased excitation of thalamic neurons secondary to loss of afferent input from the region of cortical infarction, other explanations must be considered. They include direct ischaemia due to involvement of small penetrating arteries and compression of the thalamic circulation by postinfarction oedema of perithalamic tissue.

IV Intrahemispheric Diaschisis

Depression of rCBF and regional glucose metabolism have been observed in the frontal and parietal cortices adjacent to hemispheric lesions. [3] Although this depression might be related to interruption of afferent input to these regions as a result of undercutting of associative fibers, several other factors might also play a role. They include direct extension of the ischemic processes including tissue edema, raised intracranial pressure, diffusion of toxic waste products from the necrotic core, and selective neuronal cell death without necrosis.

V Crossed Cerebellar Diaschisis [8.10.12]

Crossed cerebellar diaschisis [8] (CCD) is a matched depression of blood flow and metabolism in the cerebellar hemisphere contralateral to a focal, supratentorial lesion, and is a well-recognized phenomenon after cerebral infarction.

CCD has been observed in association with isolated lesions of the frontal cortex, parietal cortex, multilobar lesions, has also been observed in association with deep MCA or capsular infarctions, nonlacunar subcortical strokes, and basal ganglia and putaminal lesion.

Mechanism

The most likely mechanism underlying CCD is thought to be interruption of the corticopontocerebellar connections by the lesion causing deafferentation and transferred metabolic depression of the contralateral cerebellar hemisphere.

MCA migratory embolism:

The MCA MIGRATORY [25] EMBOLISM is the syndrome of fading hemiparesis with Wernicke's aphasia. The embolus lodges initially at the stem of the MCA, occluding the penetrating lenticulostriate branches long enough to produce-scattered foci of infarction through the Basalganglia and internal capsule, the involvement of the latter producing the hemiparesis distal migration of embolus then occurs, finally occluding the lower division of the MCA at the superior temporal plane and beyond. This infarct yields Wernicke's Aphasia. Two separate foci of infarction occur, but they result from the same embolic event.

Crossed Aphasia:[11]

It has been reported that patients with non dominant hemispherical lesions had Aphasia. (Crossed Aphasia)

Crossed Aphasia (or) Aphasia in a patient with Non dominant hemispherical lesion may be due to

i) Activation or silent lesion in left hemisphere by a sudden lesion in Right hemisphere, (or)

ii) Ipsilateral control of dominant hand, (or)

iii) Dual representation of language function, (or)

iv) Arrest in the development of lateralisation of language function, (or)

v) Disorders of neuronal migration during early stages of foetal development due to perinatal (or) developmental trauma.

Pure Motor Stroke [30]

The clinical presentation in such patients consisted of a mild and transient pure motor syndrome affecting the face and limbs and lesions was found predominantly in the posterior angle of the putamen, with impingement of the posterior limb of the internal capsule. At times, a small capsular lesion has manifested as pure motor stroke and dysarthria, although the clinical syndrome has been more properly that of a “pure sensory-motor” stroke, related to a component of lateral thalamic compression accompanying the capsular lesion.

Pure Sensory Stroke

The syndrome of pure sensory stroke, related to thalamic lacunar infarction. These patients had posteriorly located putaminal lesions that were adjacent to the posterior limb of the internal capsule and the adjacent thalamus. The clinical syndrome was a contralateral hemisensory syndrome involving both superficial and deep sensory modalities, with more severe involvement of the leg than of the arm and face. The imaging studies demonstrated involvement of the dorsolateral thalamus and the ascending thalamocortical projections located in the posterior ("retrolenticular") portion of the posterior limb of the internal capsule.

Putaminal lesions

They clinically presented with hemiplegia, hemianaesthesia, conjugate ocular deviation with having more prominent aphasia or parietal lobe findings and impaired consciousness.

The clinical-CT correlations in putaminal lesions [32] allowed to characterize a number of clinically useful patterns, as follows:

a. Intraventricular hemorrhage was seen with large hematomas, and both features were associated with high mortality,
b. All patients had combined motor and sensory deficits,
c. Delayed neurologic deterioration occurred only in patients with lesions that extended into the cerebral hemisphere or the thalamus.

Cortical lobar lesions [38]

They occur in all cerebral lobes but have a predilection for the parietal, temporal, and occipital lobes. This predilection for the posterior half of brain is due to the predilection of intracerebral microaneurysms for the parietooccipital area. [38]

The neurologic deficits seen with lobar lesions [14]

The neurologic deficits seen with lobar lesions [14] in the study depend on the location and size of the lesions. They include

- **a.** Sudden hemiparesis, worse in the arm, with retained ability to walk, in frontal lobe lesions,
- **b.** Combined sensory and motor deficits, the former predominating, and visual field defects in parietal lobe lesions,
- **c.** Fluent paraphasic speech with poor comprehension and relative sparing of repetition in left temporal lobe lesions; and
- **d.** Homonymous hemianopia, occasionally accompanied by mild sensory changes (extinction to double simultaneous stimulation), in occipital lobe lesions.

16 patients had lobar lesions in CT Scan in this study and found to correlate well clinically.

Cerebellar lesions

Neurologic findings in cerebellar lesions [40] for Noncomatose Patients were Appendicular ataxia, Truncal ataxia, Gait ataxia, Dysarthria, Gaze palsy, Cranial nerve findings like Peripheral facial palsy, Nystagms, Miosis, Decreased corneal reflex, Abducent palsy, Loss of gag reflex, Skew deviation, Trochlear palsy, Hemiparesis, Extensor plantar response, Respiratory irregularity, Nuchal rigidity and Subhyaloid hemorrhage.

It may be possible to make accurate predictions from the combined analysis of clinical and CT data [38] at the time of onset. Especially important is careful monitoring of the status of the patient. The development of obtundation and bilateral extensor plantar responses is ominous and is virtually always followed by a fatal outcome unless surgery is performed.

Mid brain lesions

These patients presented with isolated fourth and third nerve palsies as well as various combination of a dorsal midbrain syndrome. These cases were remarkable for the absence or paucity of signs of long-tract involvement, stressing the fact that small midbrain lesions can present with isolated ophthalmoplegia. [56,59]

Pontine lesions

These patients with presented with pinpoint pupil, decerebrate rigidity impairment of horizontal ocular movements, hyperpnoea, hypertension, Hyperhidrosis and deep coma.

Medullary lesions

The characteristic profile was one of sudden onset of headache, vertigo, dysphagia, dysphonia or dysarthria, and limb incoordination. Common findings on examination were palatal weakness (88%), nystagmus, cerebellar ataxia, or both (75%) limb weakness (68%), and hypoglossal nerve palsy (56%). Less common signs were facial palsy and Horner's syndrome.

Lacunar infarctions [51]

Lacunar infarctions are understood to reflect arterial disease of the vessels penetrating the brain to supply the capsule, basal ganglia, thalamus, and paramedian regions of the brain-stem. The most common lesion is a tiny focus of microatheroma or lipohyalinosis stenosing one of the deep penetrating arteries.

The term lacunar hypothesis [51,28] is used to refer to the clinicopathophysiologic correlation of the condition. The hypothesis consists of two parts:

a. Symptomatic lacunes usually present with a small number of distinct lacunar syndromes and
b. Lacunes are caused by a characteristic disease of the penetrating artery. After satisfying both parts of the hypothesis, the stroke can be classified as a lacunar infarction.

Lacunar syndromes

The characteristic features of all these syndromes are their relative purity and their failure to involve higher cerebral functions [58] such as language, praxis, behaviour controlled by the nondominant hemisphere, memory, and vision. The classic lacunar syndromes include pure motor, pure sensory, and sensorimotor syndromes; ataxic hemiparesis; clumsy hand dysarthria; and hemichorea/hemiballismus. Out of these pure motor, pure sensory, ataxic, syndromes were reported in this study.

There are clearly examples in the study of thrombotic or embolic infarcts manifesting as pure motor hemiparesis or sensorimotor stroke, and, conversely, large lacunar infarcts in the caudate nucleus or thalamus that may initially manifest as impairment of higher cerebral functions. The latter syndromes are probably due to a reversible functional disconnection [13] between the subcortical infarcted areas and their cortical projections. In the acute setting, therefore, the reliance on clinical grounds alone for the identification of lacunar infarcts can be misleading for both prognostic estimates and therapeutic choices.

Lacunar infarct CT correlation [54]

CT scanning [58] is positive only for roughly half of the cases of even the most common form of lacune, pure motor stroke. [9] Visualizing lacunes depends on their location, and MRI is clearly superior to CT in evaluating lesions, especially in the posterior fossa.

a. In cases of pure motor hemiparesis, lacunes can be most commonly found in the internal capsule and corona radiata, but they have also been imaged in the basal ganglia, pons, and thalamus. In this study 4 cases showed lacunar infarct in capsular region.

b. Pure sensory stroke from low densities in the centrum semi-ovale are also probably lacunar was also reported.

c. Brain scans performed once only within a few hours of the onset of an ischemic stroke have a similarly low yield. Brain scans performed no matter how often may remain negative in some cases of small lacunar infarction. If the lesion is below the limits of resolution of the scan technique.

d. In this study 16 cases showed normal CT Scan. Most of these cases were pure motor hemiparesis and it was clear that CT might have failed to pick up these lesions which needs MRI

In most circumstances the clinical features and CT or MRI findings suffice to differentiate acute intracerebral hemorrhage from infarction within the first hours after stroke onset decreased consciousness, headache, and nausea and vomiting are not only predictors of hemorrhage, but also reported in infarcts also.

Small deep, or lobar hematomas can manifest as circumscribed focal deficits and can easily lead those relying on the clinical syndrome alone to diagnose infarction mistakenly, these scores can never be used to make a definitive diagnosis. The advent of CT and MRI has led to the correction of these potential misdiagnoses, resulting in a greater proportion of hemorrhages in stroke series.

MATERIALS AND METHODS

MATERIAL AND METHODS

Selection criteria:

Any patient admitted with

1. Acute onset of focal neurological deficit pertaining to vascular territory and on clinical examination suggestive of CVA was included in this study.
2. Stroke developing in already hospitalized patients.
3. History of trauma preceding the time of onset of deficit were excluded from this study.
4. All the patients clinically diagnosed as CVA irrespective of their age were included in this study.

Total number of patients in the study:

Total number of patients in the study is 102.

Period of Study:

All the patients admitted as inpatients in Thanjavur Medical College Hospital, during the period of 2004 – 2006 were included in this study.

Geographic distribution

Geographic distribution of the patients were predominantly from rural areas of Thanjavur, Nagapatinam, Perambalur and Trichy Districts.

Limitation of the Study:

1. Angiogram could not be done to assess the vascular territory involved and severity of ischaemia.
2. MRI could not be done in all patients because of its non availability in the institution and its cost factor.
3. Follow up study could not be done for these patients.

Selection and study of this patients were done as mentioned in the proforma.

All the patients underwent a thorough and detailed general and neurological examination. All other system examination was done in all the patients.

Neurological examination was done with reference to motor, sensory, autonomic and higher functional disturbances.

Power is graded according to MRC grading system from 0 to 5.

Sensory system examinations were done for somatic, special and cortical sensation.

Higher functions testing were done with minimental scale.

Lobar functions were done in all these patients.

Frontal lobe functions [42,62]

Frontal lobe functions is evidenced by the following clinical signs.

1. Alternating rapid motor sequences by Fist Ring Test. [62]
2. Presence of release reflexes like palmomental, glabellar tap, snout reflexes.
3. Presence of urinary incontinence
4. Emotional incontinence.
5. Broca's aphasia.

Parietal lobe functions: [42,62]

Parietal lobe functions were assessed with following clinical signs.

Homonymous hemianopia.

Sensory neglect.

Cortical sensory loss.

Agraphia, acalculia, Finger agnosia & Lt to Rt disorientation.

Anosognosia.

Visuospatial disorientation.

Dressing apraxia and construction apraxia.

Optokinetic nystagmus.

Temporal Lobe functions [42,62]

Temporal lobe functions were assessed by the presence of Wernicke's aphasia, Visual, Auditory, Olfactory, Hallucinations.

Occipital Lobe functions: [42,62]

Occipital lobe functions were assessed by the presence of Optokinetic nystagmus, Homonymous hemianopia and cortical blindness.

Brain Stem Deficits [16]

Brain stem deficits were studied by the presence of ataxia, vertigo, dysarthia, oculomotor palsy, with or without sensory / motor hemiplegia (crossed) but without higher cerebral dysfunction and crossed hemiplegia.

Clinical assessment of nature of lesion

Haemorrhagic lesions in CT Scan brain was suspected clinically by the presence of intense throbbing headache, projectile vomiting, seizures, altered sensorium, Neck stiffness, dissociated eye movements, Papilloedema in a known hypertensive patients.

Absence of these clinical findings with pure motor hemiplegia, intermittent progression of signs, relative preservation of consciousness and some degree of recovery were suspected to be **Infarcts in CT Scan brain**.

Autonomic functions for sympathetic and parasympathetic system were done for all patients.

After careful clinical examination of the patients all the patients were submitted to the following investigations.

I Basic lab investigations:

a) Urine analysis.

b) Blood-Sugar, Urea, Creatinine.

c) Serum electrolytes.

d) Lipid profile.

II ECG

12 lead ECG was taken atleast once in all those patients. The ECG was taken soon after admission and subsequently thereafter if needed.

Rhythms strips were also taken when required.

Rate, Rhythm, PR interval, QRS axis, QTC interval, ST segment, U waves, Ischaemic Changes, chamber hypertrophy were analysed and recorded.

III ECHO

The ECHO was done in 18 patients who showed ischaemic and other changes in ECG. Special attention was given for any valvular dysfunction, reduced LVEF, and any regional wall motion abnormalities.

IV CT Scan Brain

CT Scan Brain was done in all patients included in this study within 24 – 48 hrs of admission. It was repeated after 48 hrs when the initial CT Scan was negative and also repeated when the patient suddenly detoriated or if there is no expected recovery.

The Imagining of brain was done to

a. To diagnose CVA.

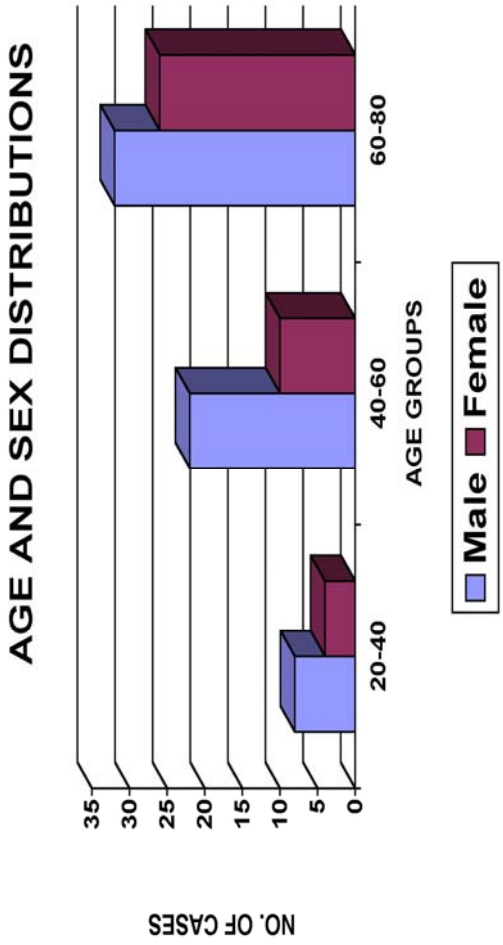
b. To document the presence of haemorrhage or infarct.

c. To locate the site of lesion, size of lesion, extent of brain damage and surrounding edema.

d. To asses evidence of impending herniation.

e. To identify and exclude Neoplasm, abscesses and other conditions masquerading as stroke.

RESULTS AND OBSERVATIONS


AGE AND SEX DISTRIBUTIONS
NO. OF CASES
35
30
25
20
15
10
5
0
20-40
40-60
60-80
AGE GROUPS
Male
Female

RESULTS AND OBSERVATIONS

I AGE AND SEX DISTRIBUTIONS

The total number of patients included in the study was 102 during the period of 2004 – 2006 in Thanjavur Medical College and Hospital. Out of which 62 were male and 40 were female patients.

The Age, Sex distributions were compiled in tabular columns as follows:

Sex	20-40 years	40-60 years	60-80 years
Male	8	22	32
Female	4	10	26

The total number of male patients included in the study was 62. Out of which 8 patients belong to the age group 20-40 years. The youngest of them was 22 years. 22 patients belong to the age group 40-60 years and 32 patients belong to the age group 60-80 years, the oldest of them was 80 years.

The total number of female patients included in the study was 40. Out of which 4 patients belong to the age group 20-40 years. The youngest of them was 26 years. 10 patients belong to the age group 40-60 years and 26 patients belong to the age group 60-80 years, the oldest of them was 78 years.

II NATURE OF LESION IN CT SCAN

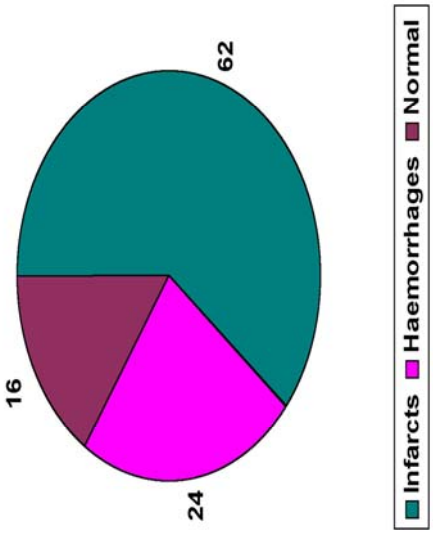
Out of the 102 cases, 62 cases showed infarct in CT Scan and 24 showed intracerebral haemorrhage in CT. In 16 patients CT Scan was normal.

Out of the 62 patients who showed Infarct in CT Scan, 6 were massive infarcts with midline shift and 4 were lacunar infarcts.

The total number of haemorrhages in CT Scan in the study is 24 and out of which 4 patients showed massive ICH with mid line shift.

6 patients out of 102 patients diagnosed clinically as CVA due to ICH showed massive Infarct with midline shift in CT Scan Brain.

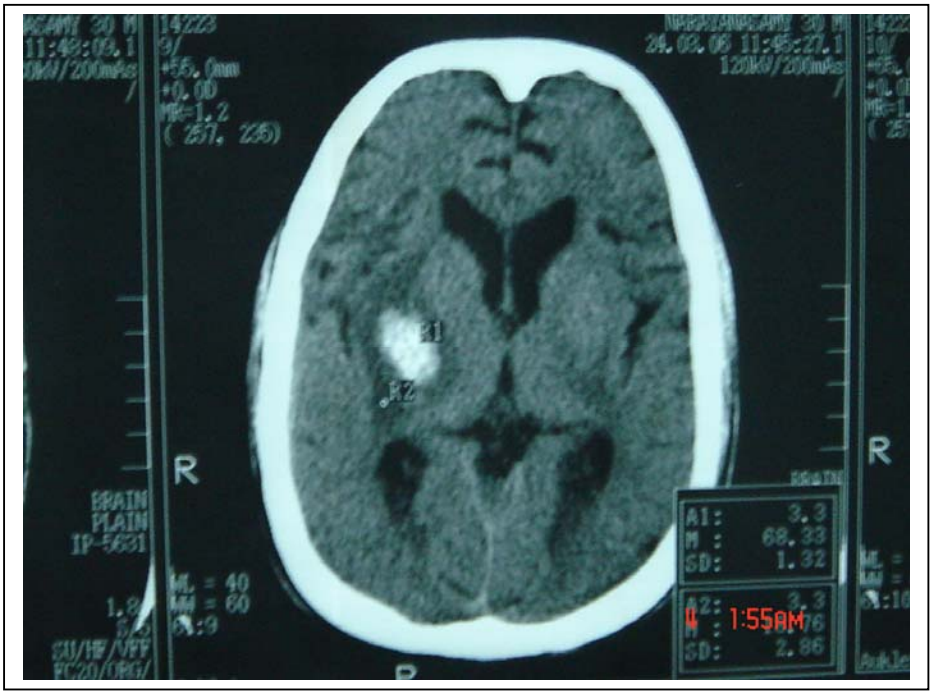
5 patients who presented clinically as pure motor hemiplegia, in the absence of sings of ICH. (headache, seizure, vomiting, altered sensorium, papilloedema) showed small haemorrhagic lesion in CT Scan Brain.


62
16
24
Infarcts
Haemorrhages
Normal

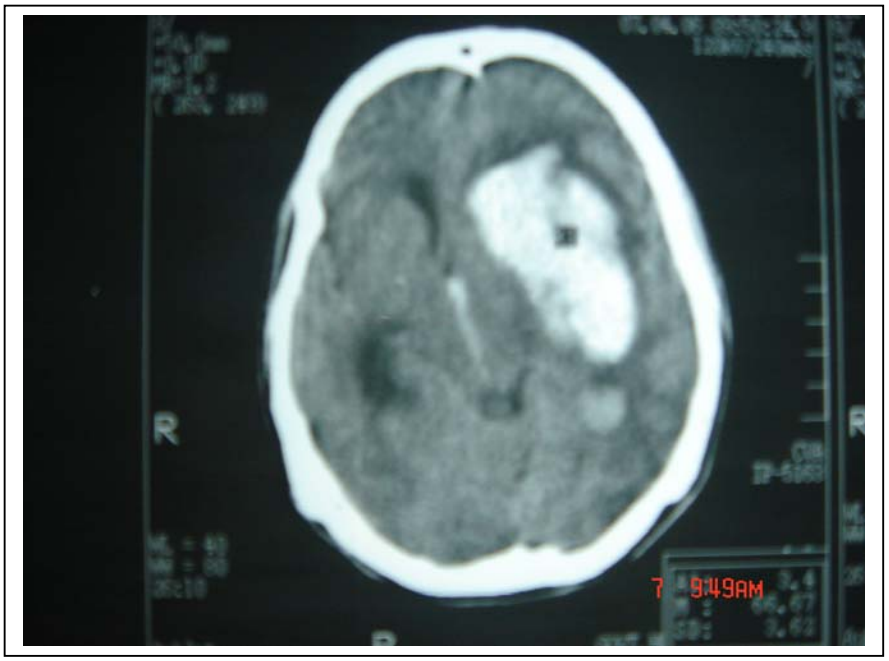
From this it was clear that massive infarct in CT scan brain mimicked haemorrhagic lesion clinically and small haemorrhagic lesion in CT Scan Brain mimicked ischaemic stroke clinically.

S. No.	Infarcts		Haemorrhages		Normal CT	
i)	Massive Infarct with midline shift	6	Massive haemorrhage with midline shift	4		
ii)	Infarct without midline shift	52	Haemorrhages without midline shift	20		
iii)	Lacunar infarcts	4				
	Total	**62**		**24**		**16**

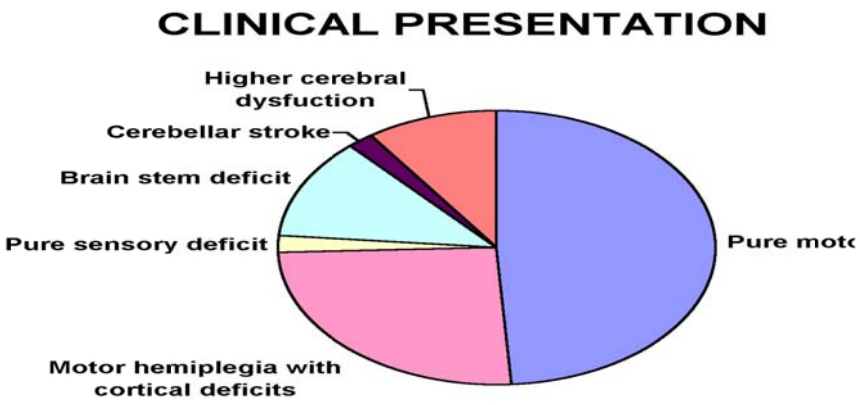
Out of these patients 62 patients had right sided hemiplegia and 40 patients had left sided hemiplegia clinically.



CT Scan Brain showing Right Capsuloganglionic ICH with surrounding edema



Left Massive ICH with Intraventricular extension and midline shift of ventricles



Predominant site of lesion in CT scan:

- Capsuloganglionic region (subcortical region)
- Capsuloganglionic region (subcortical region)
- Normal CT
- Normal CT
- Cerebellar hemispheric lesion and fronto parietal region
- Capsuloganglionic and Fronto parietal region

III ANALYSIS OF THE SITE OF LESION:

Clinical aspects versus CT Scan findings

S. No.	Clinical presentation	No. of cases	Predominant site of lesion in CT scan	Percentage of predominant site of lesion in CT Scan
A	Pure motor hemiplegia	50	Capsuloganglionic region (subcortical region)	72%
B	Motor hemiplegia with cortical deficts	26	Capsuloganglionic region (subcortical region)	73%
C	Pure sensory deficit	2	Normal CT	0%
D	Brain stem deficit	12	Normal CT	67%
E	Cerebellar stroke	2	One ® cerebellar hemispheric lesion	50%
			One fronto parietal lesion (crossed cerellar diaschisis)	50%
F	**Motor Hemiplegia with higher cerebral dysfunction**			
	i) Frontal lobe features	4	Capsuloganglionic	75%
	ii) Parietal lobe features	4	Non-dominant high parietal region	100%
	iii) Temporal lobe features	0	-	
	iv) Occipital lobe features	2	1 – occipital region	50%
			Other – capsuloganglionic region	50%

A) Pure Motor Hemiplegia:

Out of the 102 patients 50 patients presented with pure motor hemiplegia alone without any cortical lobar dysfunction or brain stem or cerebellar signs. Most of these patients, about 36 revealed recent lesion in capsuloganglionic region in CT Scan.

SITE OF LESION IN 50 PATIENTS WITH PURE MOTOR HEMIPARESIS

Capsuloganglionic region (subcortical region)	Brain stem	Cerebellum	Cortical lobar areas	Normal CT Scan
36	6	0	5 (Fronto parietal region)	3

Out of the 50 patients who presented with pure motor hemiplegia, one patient's CT Scan revealed infarct in the SAME SIDE capsuloganglionic region. We know that this lesion was not responsible for the hemiplegia and probably CT might have not picked up the lesion responsible for the FND. MRI may be more useful to pick up the lesions not picked by routine CT Scan.

From this we know well mostly dense hemiplegias were due to lesion in the capsuloganglionic region.

When CT shows a non correlating lesion, MRI may be useful to pickup the lesions missed by conventional CT Scan.

B) Motor hemiplegia with cortical deficits

Out of the total 102 patients 26 patients showed clinically motor hemiplegia with cortical deficits like aphasia, neglect, visual field defects, visuospatial abnormalities, apraxia, release reflexes etc.,

Most of these patients about 19 showed lesion in capsuloganglonic region (subcortical lesion).

Site of lesion in 26 patients with motor hemiplegia with cortical deficits:

Capsuloganglonic region (subcortical)	Brain stem	Cerebellum	Cortical lobar areas	Normal CT Scan
19	-	-	4 (fronto parietal region)	3

C) Pure sensory deficits:

Out of the 102 patients only two patients presented clinically with pure sensory deficits. Their CT Scans were found to be normal.

D) Brain stem deficits:

Out of the 102 patients 12 patients showed brain stem deficits clinically. Of which most of the patients about 8, showed normal CT Scan.

Site of lesion in 12 patients with brain stem deficits

Capsuloganglionic	Brain stem	Cerebellum	Lobar areas	Normal
3	1	-	-	8

E) Cerebellar stroke:

Only two patients showed clinically cerebellar signs. Out of which one showed lesion in right cerebellar hemisphere and the other in frontoparietal region explaining the phenomenon of crossed cerebellar diaschisis. [8]

F) Motor Hemiplegia with higher cerebral dysfunction

Totally only 10 patients showed motor hemiplegia with higher cerebral dysfunction in the study. Out of which 4 patients who presented clinically with frontal lobe dysfunctions, as evidenced by failure to do 'FIST RING' test three patients showed lesion in the subcortical areas (capsuloganglionic area) in CT scan and the rest in fronto parietal region which correlated well. 4 patients presented clinically with parietal lobe

dysfunctions showed lesion in the non dominant parietal lobe which correlated well with clinical features.

Out of the 2 patients who showed occipital lobar sign clinically, one showed lesion exactly in occipital cortex and the other in capsuloganglionic area.

Site of lesion in higher cerebral dysfunctions:

Capsuloganglionic	Brain stem	Cerebellum	Cortical lobar areas	Normal CT
4	-	-	Frontal 1 Parietal 4 Occipital 1	-

From these results it was clear that most of the cases showed lesion in the subcortical white matter. i.e., capsuloganglionic area (63%) in the study.

It is also found that the Hypertensive and Diabetic patients also showed lesion predominantly in subcortical white matter (capsuloganglionic area).

Since **capsuloganglionic** area is the mostly involved site, it is clear that **'MCA territory'** is the artery most commonly involved in hemiplegia patients in the study.

Also clinical features correlated well with the site of lesion in CT scan except few cases (16 patients – 16%) whose CT was Negative.

These patients may be investigated by MRI scans for better correlations and occult lesions missed by CT scan.

IV ANALYSIS OF SEVERITY OF LESION:

7 patients out of 102 patients presented clinically with pure dense hemiplegia of power 0/5 and were disoriented and drowsy on examination. These patients who were expected to have large lesions in CT Scan showed only tiny lesions (<3mm) in CT Scan brain.

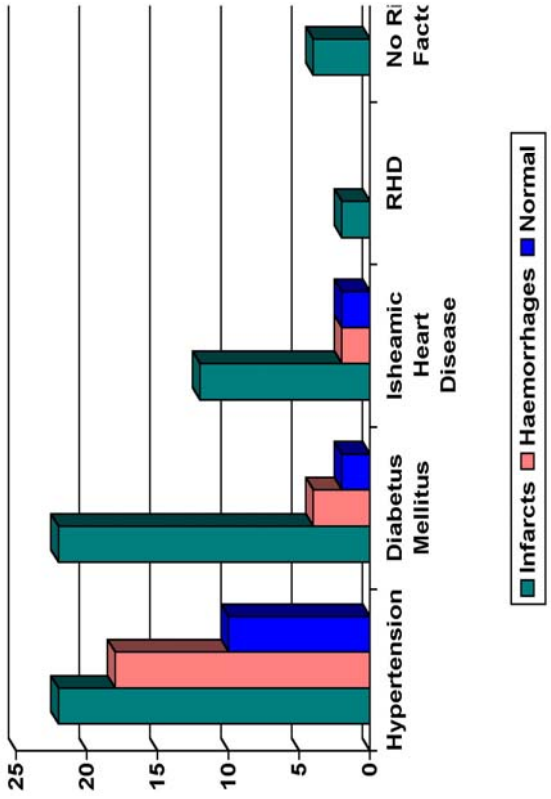
From this it was clear that severity of lesion clinically does not correlate with size and extent of lesion in CT Scan.

V RISK FACTOR – ANALYSIS

Out of the 102 patients included in the study 50 patients were hypertensives on regular antihypertensive drugs. 30 patients were hypertensives for more than 4 years and the rest were newly detected Hypertensives only 6 months prior to admission.

Out of the 50 Hypertensive patients 22 showed infarct, 18 showed haemorrhage and in 10 patients CT Scan was normal. Most of the patients about 30 (60%) showed lesion in the capsuloganglionic region.

The total number of Diabetic patients in the study was 28. All of them were type 2 Diabetes mellitus patients on OHA except one patient who was type 1 DM on insulin.


25
20
15
10
5
0
Hypertension
Diabetus Mellitus
Isheamic Heart Disease
RHD
No Ri Factc
Infarcts
Haemorrhages
Normal

Risk factor	Total no. of patients	Nature of lesion in CT Scan			Most common site of lesion in CT Scan
		Infarcts	Haemorrhages	Normal	
Hypertension	50	22	18	10	Capsuloganglionic region (60%) (30 patients)
Diabetes mellitus	28	22	4	2	Capsuloganglionic region (68%) (19 patients)
Ischaemic heart disease	16	12	2	2	Capsuloganglionic region (50%) (8 patients)
RHD	2	2	-	-	Capsuloganglionic region (100%) (2 patients)
No Risk Factors	6	4	-	2	Capsuloganglionic region (50%) (3 patients)

Out of the 28 Diabetic patients 22 patients showed infarct in CT Scan, 4 showed haemorrhages and two-showed Normal CT Scan.

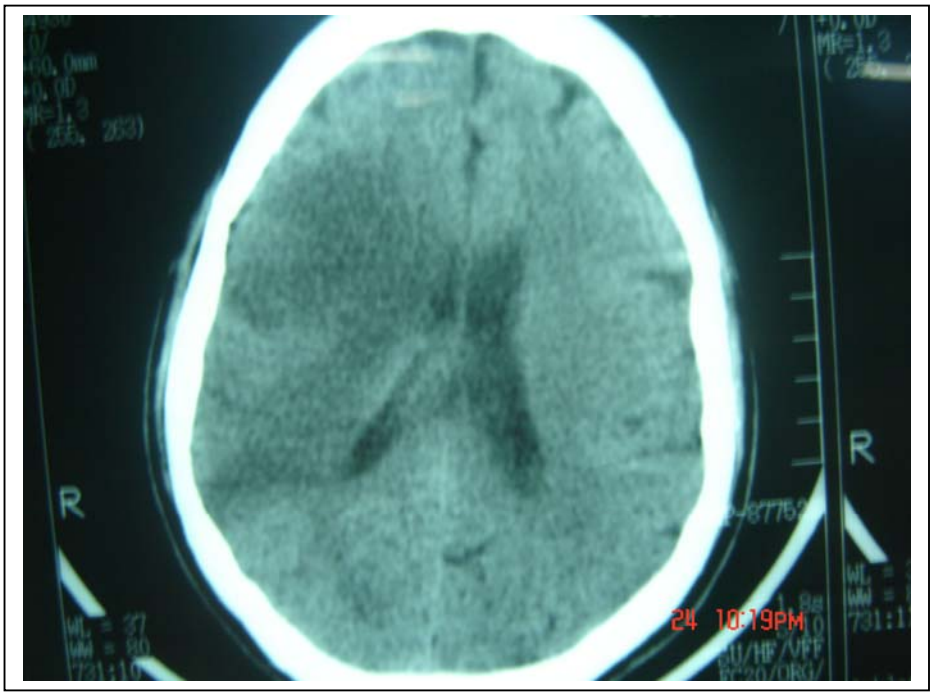
Most of the Diabetic patients about 19 patients (68%) showed lesion in the capsuloganglionic region.

Out of the 16 patients who were known ischaemic heart disease patients on drugs, 12 showed infarct, 2 showed haemorrhage and 2 showed normal CT Scan.

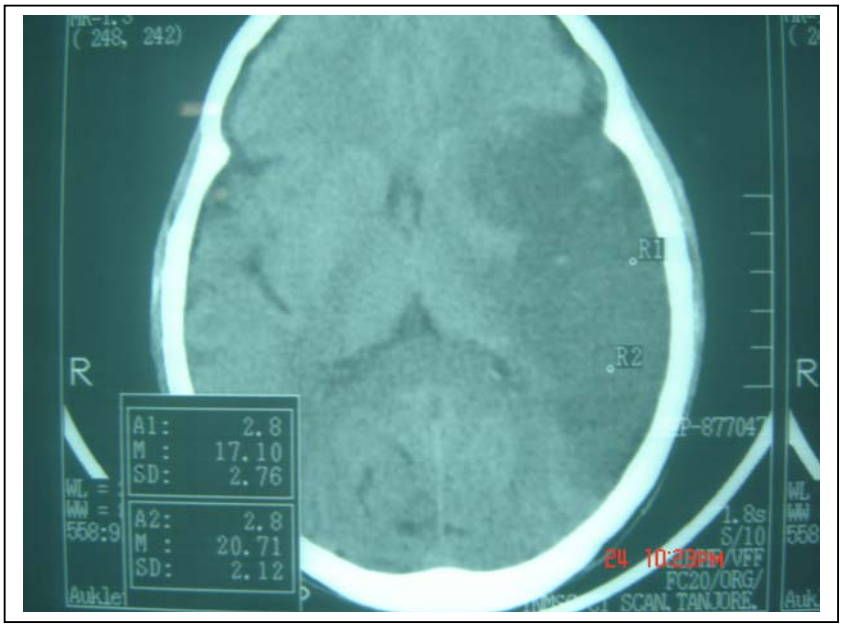
Among the 16 patients, 8 patients (>50%) showed lesion in capsuloganglionic region.

Among the 102 patients, 2 patients were patients (RHD) who showed infarct in the capsulogarglionic region.

Among the 102 patients, 6 patients had no risk factors. They were not a known Diabetic, hypertensive, CAHD, RHD patients. Out of these 6 patients 3 showed infarct in capsuloganglionic region, one in Frontoparietal region and the rest 2 were normal.



CT Scan Brain showing massive right parietal infarct (MCA territory)



CT Scan Brain showing Left massive tempero parietal infarct (MCA territory)

DISCUSSION

DISCUSSION

All the 102 patients selected for this study were thoroughly examined and the diagnosis of CVA was made clinically and CT Scan was done in all the patients.

It has been established well in this study that there are some variations in the clinical and CT Scan findings and they do not correlate exactly in all.

I DIAGNOSIS OF CVA: CLINICAL VERSUS CT

Out of the 102 patients clinically diagnosed as CVA included in this study, 84 patients showed lesion (POSITIVE CT) in the CT scan either infarct or hemorrhage and in 16 patients CT scan was normal (NEGATIVE CT).

Out of the 16 patients who showed normal CT, 8 patients belong to posterior circulation stroke (Brain stem stroke) clinically (50% of normal CT) [73]

4 patients clinically presented with pure motor hemiplegia and 2 patients presented clinically as pure sensory stroke, also showed normal CT. These 6 cases were suspected to have small lacunar infarct which were occult and CT might have failed to pick up these lesions which needs further evaluation with MRI. It is clear that 38% of the normal CT in this study were suspected to be lacunar infarcts.[72]

In 2 patients (no. 43 and 53) CT scan was initially negative when taken very early with in 24 hours and the repeat CT scan revealed capsuloganglionic infarcts. This shows that 12% of normal CT in this study were due to early CT scans taken with in 24 hours. [45]

II NATURE OF LESION: CLINICAL VERSUS CT

In distinguishing cerebral hemorrhage from cerebral infarction the following criteria though not always diagnostic may prove helpful in suspecting an intracerebral bleed [65]. They are

1. FND preceded by intensive throbbing headache, projectile vomiting, seizures in a known hypertensive patient.
2. Altered sensorium (drowsiness to coma).
3. Neck stiffness, dissociated eye movements.
4. Papilloedema.

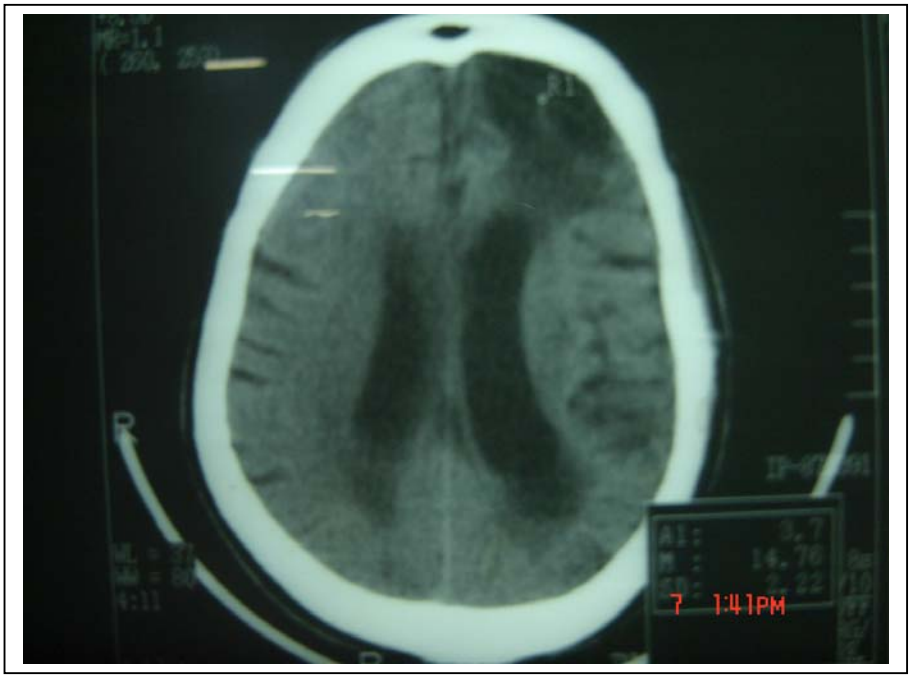
Absence of these clinical findings with pure motor hemiplegia, intermittent progression of sings, relative preservation of consciousness and some degree of recovery were suspected to be Infarcts in CT scan Brain.

6 (6%) out of 102 patients diagnosed clinically as CVA probably due to hemorrhage (ICH) showed massive infarct with midline shift in the CT scan brain.

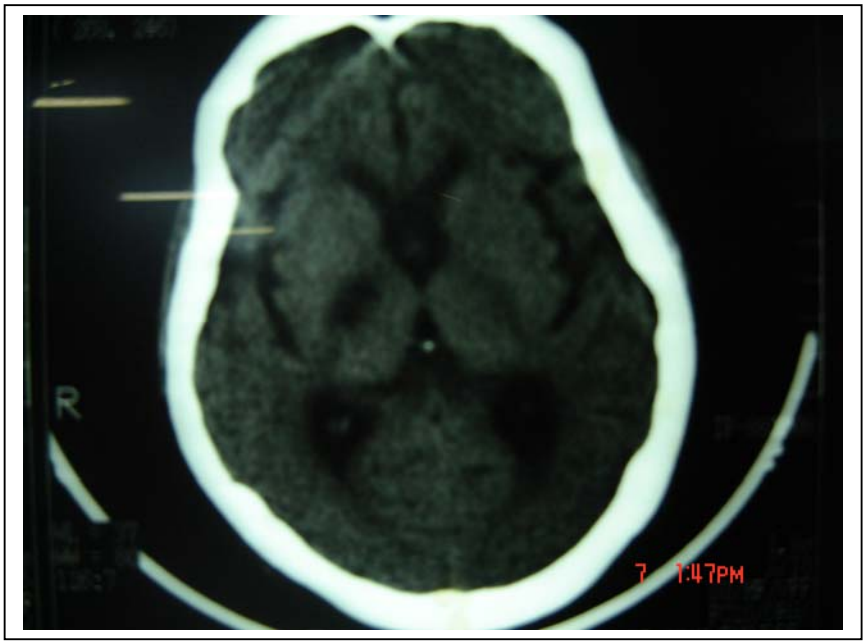
5 patients (5%) who presented clinically as pure motor hemiplegia, in the absence of signs of ICH (headache, seizures, vomiting, altered sensorium, papilloedema) showed small hemorrhagic lesion in capsuloganglionic region in CT scan brain.

Since small hemorrhagic lesion in CT mimicked ischemic stroke clinically it is unwise to start anti-platelets in patients with CVA before taking CT Scan bran.

Thus it was clear that clinical signs and symptoms alone cannot establish an exact diagnosis of nature of lesion, like hemorrhage or infarction and hence CT is mandatory. [1]



CT Scan Brain showing left frontal infarct



CT Scan Brain Showing right capsuloganglionic infarct

III SITE OF LESION: CLINICAL VERSUS CT

A) Predominant site of lesion in pure motor hemiplegia

Out of 50 patients presented clinically with pure motor hemiplegia alone without any cortical lobar dysfunction or brain stem or cerebellar signs revealed lesion in capsuloganglionic area in CT Scan.

It was clear that the site of lesion correlated well in patients with pure motor hemiplegia (72%) in this study.

The rest of the lesions were in fronto parietal and brain stem regions. This was explained by the **"Phenomenon of cortical subcortical diaschisis"[13].** This refers to functional deactivation of morphologically intact subcortical brain region remote from but connected to an area of cortical primary structural damage and also due to interruption of neural connections between cortical and subcortical structures according to Von Monakow study in 1914.

B) Site of lesion in motor hemiplegia with cortical deficits

26 patients out of 102 presented clinically as motor hemiplegia with cortical deficits like aphasia, neglect, visual field deficts, visuo spatial abnormalities, apraxia, release reflexes. Most of these patients about 19 showed lesion in capsuloganglionic area, this was explained by **"Phenomenon of sub-cortical cortical diaschisis" [2].**

According to “Demeunsse G, Capon A, Verhas M, et al; pathogenesis of Aphasia, in Deep seeted lesions, likely role of cortical diaschisis. Eur Neural 30; 67 1990 lesions in subcortical white matter (eg.) capsuloganglionic area showed cortical deficts clinically such as aphasia, neglect, apraxia, visuospatial abnormalities due to interruption of neural connections between subcortical structures and cortex, therefore to represent diaschisis.

C) Site of lesion in pure sensory deficit

2 cases presented clinically with pure sensory stroke showed normal CT which needs further evaluation with MRI to detect the occult lacunar infarcts. [72]

D) Site of lesion in Brain stem deficits

Most of the patients (about 8) with brain stem stroke (posterior circulation stroke) showed normal CT probably due to bony artifact and lack of sensitivity of CT in detecting posterior fossa lesions. [73]

3 patients (3%) presented clinically as Brain Stem stroke clinically with features of depressed consciousness papillary abnormalities, paralysis of conjugate ocular movements were found to have massive lesions with midline shift in the CT.

E) Site of lesion in cerebellar stroke

Out of the 2 patients who presented clinically with cerebellar signs 1 showed lesion exactly in ipsilateral cerebellar hemisphere which correlated well.

The other patient showed lesion in contralateral frontal lobe. This was explained by "Phenomenon of crossed cerebellar diaschisis" [8]

According to this CCD is a matched depression of blood flow and metabolism in the morphologically intact contralateral cerebellar hemisphere.

F) Site of lesion in motor hemiplegia with higher cerebral dysfunction

4 patients who presented clinically with parietal lobe dysfunctions like sensory neglect, hemianopia, anosognosia, dressing and construction apraxia, visuospatial disorientation showed lesion exactly in non-dominant parietal lobe which correlated well.(100%)

Out of the 2 patients who presented, clinically with occipital lobe features like hemianopia, cortical blindness, one showed lesion exactly in occipital lobe which correlated well (50%).

Patients presented with frontal lobe features showed lesion in capsuloganglionic area predominantly which doesn't correlate which could be explained by the phenomenon of "subcortical cortical diaschisis".

Diaschisis and Stroke: [13]

Diaschisis refers to functional deactivation of morphologically intact brain regions remote from but connected to an area of primary structural damage. The term coined by von Monakow [4] in 1914 is derived from the Greek schizein meaning *"to spilt"*. [6] The three general types of diaschisis that have been recognized are

1. Contralateral effects on the opposite hemisphere in the mirror regions to the injury (interhemispheric, transhemispheric or transcallosal diaschisis);

2. Ipsilateral effects on the injured cerebral hemisphere but separate from the injury; and

3. Effects on the cerebellum contralateral to the affected hemisphere (crossed cerebellar diaschisis [CCD]

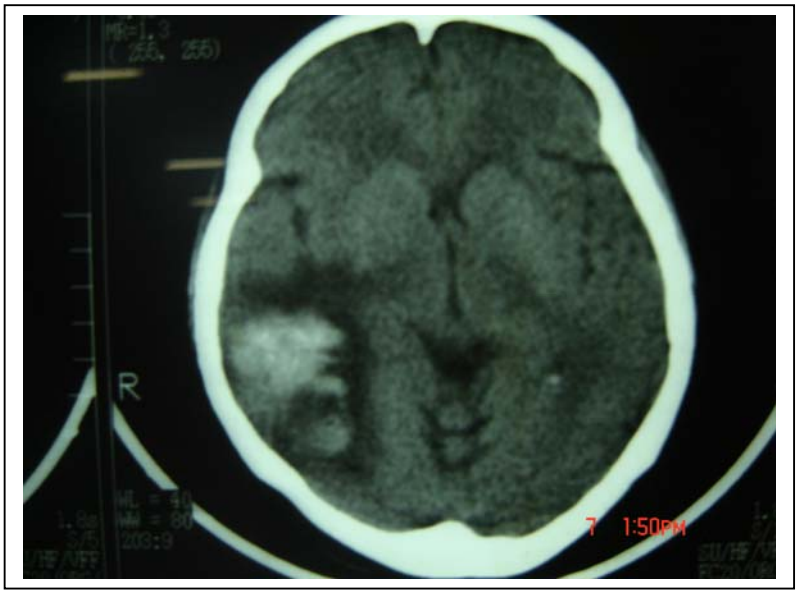
IV SEVERITY OF LESION : Clinical Versus CT

Seven patients presented clinically with dense pure motor hemiplegia of power 0/5. These patients were also drowsy and were also disoriented on clinical examination. These patients were suspected to have larger lesions in CT scan brain. But they showed only tiny lesions (<3mm), but on vulnerable regions i.e., Internal capsule.

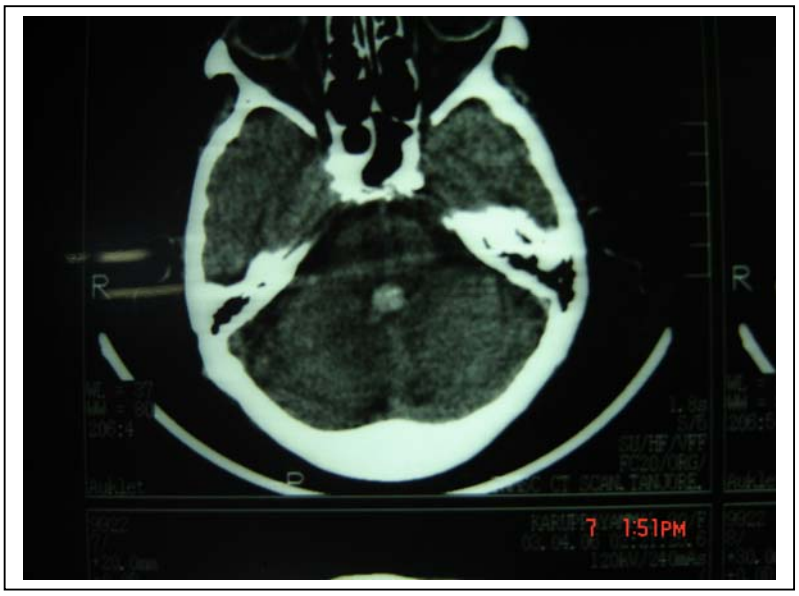
Out of the 7 patients, 4 patients showed small lacunar Infarcts and three showed tiny Intra cerebral haemorrhage (ICH < 3 mm).

From this it was clear that severity of lesion clinically does not correlate with the size and extent of lesion in CT scan. [65]

Depending upon the location of occlusion vascular lesions extracranial or intracranial, the availability of the collateral flow to that area, the presence or absence of anatomical variations in the cerebral vasculature-kinks, bends, asymmetry etc., associated diseases (e.g. hypertension, cardiopulmonary diseases, diabetes mellitus etc.), viscosity of the blood, and other ischaemia modifying factors, the size of the cerebral infarction resulting from the thromboembolism may be minimal to massive and the resulting neurovascular deficit may be trivial to catastrophic.

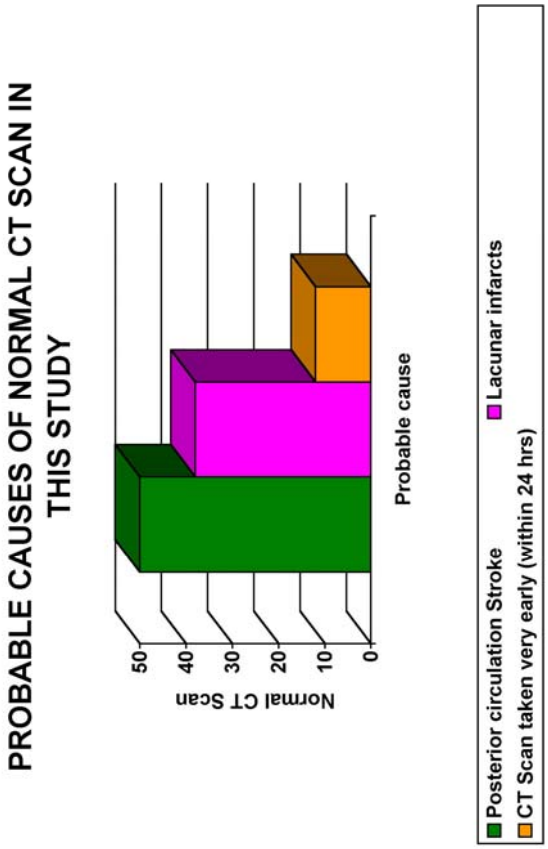


CT Scan Brain Showing right parieto occipital ICH with surrounding edema



CT Scan Brain Showing pontine haemorrhage

CONCLUSION


PROBABLE CAUSES OF NORMAL CT SCAN IN THIS STUDY
Normal CT Scan
0
10
20
30
40
50
Probable cause
Posterior circulation Stroke
Lacunar infarcts
CT Scan taken very early (within 24 hrs)

CONCLUSION

1) Regarding the role of CT Scan Brain in the diagnosis of CVA it has been found that in this study, CT Scan was positive in 84% of the clinically diagnosed CVA and Negative (i.e. Normal) in 16% of the patients.

Normal CT was found in the following clinically diagnosed CVA cases

- **a.** Posterior circulation stroke (Brain Stem Strokes).
- **b.** Small occult lacunar infarcts in cortical, subcortical areas which were not picked up by the CT.
- **c.** When taken very early (within 24 hours). In these cases repeat CT Scan was positive.

2) Regarding the nature of lesion in CVA it has been found that in some cases Massive infarcts in CT Scan mimicked Haemorrhagic stroke clinically and small haemorrhagic lesions mimicked ischaemic stroke clinically. Also massive lesions with midline shifts resembled brain stem stroke clinically.

3) a. Regarding the site of lesion in CVA Clinical localization correlated well in majority (70%) of cases with CT Scan brain and only in 30% of cases if does not correlate which was explained by phenomenon of 'Diaschisis' (Reversible functional disconnection between subcortical infracted areas and their cortical projections which has been discussed).

b. It has been found that, the most common site of lesion in CT Scan was found to be capsuloganglionic region (63%) indicating MCA territory involvement.

4) Size and extent of lesion in CT scan does not correlate with severity of lesion clinically.

5) Finally to conclude over all, the various clinical aspects in CVA do not correlate exactly with CT Scan findings in all cases. Hence both thorough clinical examination and CT Scan Brain are mandatory.

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STUDY ON CVA

CLINICAL CORRELATION WITH CT SCAN BRAIN FINDINGS

Patient Name: **Age:** **Sex:** **Occupation:** **IP. No.:**

Handedness:

CLINICAL HISTORY:

Complaints: **Duration**

PAST HISTORY: **DM/HT/CAHD/RHD/TIA/Relevant Previous illness**

GENERAL EXAMINATION: **Pulse:** **BP:** **Both Carotids:**

EXAMINATION OF CNS:

HIGHER MENTAL FUNCTIONS:

Level of Sensorium: Alert / Lethargic / Stuporous / Comatous

Appearance and behaviour: Emotionally stable / Elated / Depressed / Confused

Language: Normal / Broca's aphasia / Wernicke's aphasia / Global Aphasia

	Affected	**Unaffected**	**Could not be tested**
Orientation (Time / Place / Person):			
Memory:			
Intelligence:			
Attention and concentration:			

Description if affected:

Lobar Functions:

FRONTAL LOBE	**Present**	**Absent**	**Could not be tested**
Common: Adversive seizures Fist ring test: Urinary incontinence: Released reflexes: Akinetic mutism:			

Dominant: Broca's aphasia			
Non-Dominant: Elevation of Mood			
PARIETAL LOBE:			
Common: Optokinetic nystagmus: Homonymous hemianopia: Sensory neglect: Cortical sensory loss:			
Dominant: Gerstmann's syndrome: (Agraphia, Acalculia, Finger agnosia & Lt-Rt disorient) Apraxia:			
Non-Dominant: Anosognosia: Visuospatial disorientation: Dressing apraxia: Construction apraxia:			
TEMPORAL LOBE:			
Common: Korsak off amnesia: Hallucinations: (Visual, Olfactory, Auditory) Unicinate fits: (CPS)			
Dominant: Wernicke's aphasia: Amusia: Verbal memory: Upper quadrantanopia:			
Non-Dominant: Non verbal memory:			
OCCIPITAL LOBE:			
Common: Cortical blindness: Simultagnosia: Hemianopia: Anton's syndrome: Balint's syndrome:			
Dominant: Visual agnosia: Colour anomia:			
Non-Dominant: Visual hallucinations:			

Cranial Nerves involved:

Examination of Fundus:

Examination of Motor system: Right Left

Upper limb and Lower limb: Bulk, Tone, Power, Reflexes, Coordination, Gait

Sensory system:

Touch, Pain and Temp:

Sense of Position and vibration sense:

Autonomic Nervous system:

Sympathetic: Horner's syndrome / Postural hypotension / Cardiac arrhythmias/ skin Changes

Parasympathetic: III, VII, IX, X and Bladder.

Other Systems: CVS: RS: Abdomen:

Clinical localization of the lesion:

Clinical assessment of the Nature of lesion:

Investigation:

CT Scan Brain:

Diagnosis of CVA

Nature of lesion:

Site of lesion:

Midline shift:

Size of the lesion:

MASTER CHART

S. No.	IP. No.	Name	Age	Sex	Major Risk Factor	BP mm/Hg	Clinical Presentation	Nature of lesion assessed clinically	Nature of Lesion in CT Scan	Site of Lesion in CT Scan	ECG	ECHO	Blood Sugar (R)	Blood Urea	Serum Crea tinine	Serum		Lipid profile
																Na	K+	
1	875951	ARUMUGAM	70	M	HT	150/90	Brain Stem Deficit	Infarct	Infarct	® Capsuloganglionic region	N	-	120	42	1.2	138	4.2	TGL ↑ Choles ↑
2	877712	SIVAPRAKASAM	54	M	DM	100/70	Pure Motor and cortical deficits	Infarct	Infarct	® Capsuloganglionic region	N	-	180	40	1.2	140	4.2	-
3	877793	GOVINDAN	60	M	CAHD	110/70	Pure Sensory Deficit	Infarct	Normal	® Normal	ST↓ V5 V6	Hypokinesia of lat wall	100	32	1	138	5	LDL ↑
4	872823	PERIYASAMY	54	M	-	130/80	Pure motor hemi deficit	Infarct	Infarct	(L) Capsuloganglionic (Same side of Hemiplegia)	N	-	120	34	1.2	136	4	-
5	876664	ANDAL	65	F	CAHD	120/80	Pure motor hemi deficit	Infarct	Infarct	(L) Capsuloganglionic (Same side of Hemiplegia)	ST↓T↓ V1 - V6	Hypokinesia of Ant wall	90	40	0.8	140	3.6	LDL ↑
6	876403	RASATHI	26	F	DM	130/80	Opthalmoplegia with crossed Hemiparesis (weber)	Infarct	Infarct	Brain stem -Midbrain	N	-	96	40	1	138	3.6	-
7	878620	RAJENDRAN	54	M	HT/RF	180/110	Pure motor hemiplegia	Infarct	Infarct	® Capsuloganglionic	LVH Strain	-	110	100	3.2	140	5.2	-
8	878609	SEKAR	45	M	-	100/70	Pure motor hemiplegia	Infarct	Infarct	® Capsuloganglionic	N	-	104	42	1.2	138	4	-
9	875771	IRUDHAYA SAMY	80	M	DM	100/70	Pure motor hemiplegia	Infarct	Normal	Normal	N	-	200	38	1	136	4.2	-
10	879525	ANGALAGAN	48	M	CAHD	110/70	Pure motor hemiplegia	Haemorrhage with midline shift	Haemorrhage with midline shift	(L) Fronto parietal	ST ↓lavl V5 V6	Hypokinesia Ant Lat wall	100	32	1.4	140	4	LDL ↑
11	887433	PITCHAIAMMAL	65	F	HT	140/90	Iron dominant parietal lobe dysfunction	Infarct	Infarct	® High parietal	N	-	110	36	1.2	138	4.2	TGL ↑
12		CHINNAPPONNU	80	F	DM	120/80	Pure motor hemi deficit	Infarct	Infarct	(L) Capsuloganglionic	N	-	200	40	1.4	140	4	-
13	879610	MOOKAIYAN	58	M	HT	160/100	Motor + cortical deficits	Infarct	Infarct	(L) Capsuloganglionic (lacunar)	LVH Strain	-	120	42	1	138	5.2	-
14	880330	NAMAS	45	M	HT	150/100	Pure Motor Deficit	Haemorrhage	Haemorrhage	® Capsuloganglionic	LVH Strain	-	120	40	1	136	4.2	-
15	880353	DHARMAPUTHRAN	45	M	-	130/80	Motor + cortical	Infarct	Normal	Normal	N	-	110	40	1	138	4.6	-
16	880345	CHINNAVEERAN	75	M	HT	160/110	Pure Motor	Haemorrhage	Haemorrhage	® Capsuloganglionic	LVH Strain	-	100	42	0.8	140	3.2	-
17	880312	VELLAIYAN	55	M	DM	130/80	Motor + Cortical	Haemorrhage	Haemorrhage	(L) Capsuloganglionic	N	-	240	44	0.8	138	3.4	-
18	878423	PAKKIAMMAL	70	F	HT	170/100	Motor + Cortical	Haemorrhage	Haemorrhage	(L) Capsuloganglionic	N	-	140	30	1	140	3.8	-

19	877301	LAKSHMI	58	F	HT	160/110	Brain Stem Deficit	Infarct	Normal	Normal	ST↓T↓ V1 - V4	-	130	28	1	138	4	↑TGL ↑LDL
20	874883	KAMACHIAMMAL	80	F	DM	110/90	Occipital lobar dysfunction	Infarct	Infarct	® Capsulogan glionic	N	-	220	20	1.2	136	4.2	-
21	875683	SEENIAMMAL	50	F	HT	180/110	Motor + Cortical	Haemorrhage	Haemorrhage	(L) Capsulogan glionic	LVH Strain	-	110	20	1.1	140	4.4	-
22	87575	SANGEETHA	62	F	DM	100/70	Pure Motor	Infarct	Infarct	® Capsulogan glionic	N	-	300	30	1	140	4.8	-
23	873859	CHINNAIYAN	50	M	HT/RF	160/100	Pure Motor	Infarct	Infarct	(L) Capsulogan glionic (lacunar)	N	-	90	100	7.2	138	6	-
24	876636	ACHIAMMAL	45	F	HT	200/120	Pure Motor	Haemorrhage	Infarct	(L) Fronto parietal with midline shift	LVH Strain	-	100	32	1	130	5	-
25	875318	GOVINDAN	75	M	DM	110/70	Non dominent Parietial Lobe dysfunction	Haemorrhage	Infarct	Non dominal parietal with midline shift	N	-	300	34	1	128	4.2	-
26	876449	NATARAJAN	60	M	CAHD	120/80	Motor + Cortical sensory	Infarct	Infarct	® Capsulogan glionic	ST↓II III avf	Hypokinesia of Inj wall	110	40	1.2	140	4.2	-
27	878199	KALIAMOORTHY	59	M	HT	244/170	Brain Stem Deficit B/L9,10 CN palsy	Haemorrhage	Haemorrhage	Brain stem (Pons)	LVH Strain	-	120	32	1.4	138	4	-
28	879108	THEVENTHRAN	30	M	HT/RF	110/70	Pure Motor	Infarct	Infarct	® Capsulogan glionic region	N	-	140	80	4.2	136	5.4	↑TGL ↑Choles
29	855365	DURAIMANICKAM	42	M	HT	160/110	Occipital lobar dysfunction	Haemorrhage	Haemorrhage	® Occipital lobar	N	-	100	34	1	138	4	↑TGL
30	853924	VEERASAMY	50	M	CAHD	130/80	Motor + Sensory hemi deficit	Infarct	Infarct	® Capsulogan glionic	ST↓T↓ II III avf V5 V6	Hypokinesia of Inj wall	120	40	1	140	5	↑LDL
31	879604	KANAKAMBAL	65	F	CAHD	110/70	Motor + Cortical	Haemorrhage	Haemorrhage	® Fronto parietal region	N	-	110	42	0.8	136	5	-
32	865017	SANTHA	70	F	HT	170/100	Pure Motor hemi deficit	Infarct	Infarct	(L) Capsulogan glionic	N	-	130	40	1	138	4	-
33	866606	KAVERIAMMAL	56	F	HT	180/110	Lateral medullary syndrome	Haemorrhage	Haemorrhage	Brain stem (L) Medullary haemotoma	LVH Strain	-	110	68	7	140	6.2	↑sr Cholesters
34	880905	BALAN	35	M	DM	160/90	Brain stem deficits	Haemorrhage	Infarct	(L) Capsulogan glionic region with midline shift	N	-	240	40	1.2	128	3.6	-
35	855409	ETHIRAS	60	M	HT	140/100	Parietal lobar dysfunction	Infarct	Infarct	Non dominal parietal lobe	N	-	120	30	1	130	4	-
36	867257	RAJAMMA	55	F	HT	170/100	Motor + cortical deficits	Infarct	Haemorrhage	® Capsulogan glionic	LVH Strain	-	130	20	1	132	4.2	↑LDL
37	872987	JAMAL BEEVI	61	F	HT	180/110	Parietal lobar dysfunction	Haemorrhage	Haemorrhage	Non dominal parietal with midline shift	LVH Strain	-	110	30	1.2	140	3.6	-
38	856662	SUNDARAMOORTHY	65	M	CAHD	120/80	Frontal Lobal features	Infarct	Infarct	® Frontal Lobe	ST↓ V1 - V6	Hypokinesia of Ant wall with clot	100	20	1.2	134	4	-

39	857118	THANGARASU	62	M	HT	160/110	Pure motor hemiplegia	Infarct	Infarct	Brain Stem Medulla	LVH Strain	-	110	40	1	136	4.2	-
40	874664	SHANTHI	38	F	DM	130/80	Motor + Cortical	Haemorrhage	Infarct	® Capsuloganglionic	N	-	230	36	1	138	4.2	-
41	875343	KANAGAMBAL	75	F	DM	120/80	Cerebellar signs	Infarct	Infarct	® Fronto Parietal with midline shift	N	-	260	26	1	130	3.6	-
42	857129	RAJENDRAN	65	M	DM	110/70	Motor + cortical	Infarct	Infarct	® Capsuloganglionic	ST↓ V1 - V3	-	240	40	1.2	138	3.6	-
43	878257	DIVYA	34	F	CAHD	120/70	Brain stem deficit	Infarct	Normal CT	Normal CT (L) Capsuloganglionic region in repeat CT	N	N	110	42	1.1	140	3.8	↑Cholesters ↑LDL
44	878294	AMIRTHAM	35	F	HT	170/110	Pure motor hemiplegia	Infarct	Infarct	® Capsuloganglionic Lacunar infarct	LVH Strain	-	100	50	1.2	142	4	-
45	857403	PANDIYAN	33	M	RHD	130/80	Pure motor hemiplegia	Infarct	Infarct	(L) Capsuloganglionic	AF/RVH	MS/PHT AF	120	40	1.2	144	4	-
46	857404	MUTHUPANDIYAN	43	M	DM	110/70	Pure motor hemiplegia	Haemorrhage	Infarct	(L) Fronto parietal with midline shift	N	-	260	20	1.1	132	4.2	-
47	875248	LAKSHMI	45	F	RHD	120/90	Motor + cortical deficits	Infarct	Infarct	® Capsuloganglionic region	AF/RVH	MS/PHT AF	130	30	1.2	140	4.2	-
48	876346	SEETHAIYAMMAL	45	F	HT	140/90	Frontal Lobel features	Infarct	Infarct	(L) Capsuloganglionic region	N	-	110	20	1.1	140	4.4	-
49	857476	KULANDAISAMY	55	M	HT	150/90	Frontal Lobel features	Infarct	Haemorrhage	(L) Capsuloganglionic region	N	-	120	20	1.2	134	4	-
50	859002	MUTHU	40	M	CAHD	130/100	Mortor + Cortical	Infarct	Infarct	® Fronto Parietal	ST↓II III avf	Hypokinesia of Inj wall	140	20	1.1	136	4	↑LDL ↑Cholesters
51	876375	SARASU	42	F	-	120/90	Pure motor hemiplegia	Infarct	Infarct	® Fronto Parietal	N	-	100	26	1.1	140	3.4	-
52	864578	DHANAM	60	F	CAHD	110/70	Pure motor hemiplegia	Infarct	Infarct	(L) Capsuloganglionic	ST↓ V5 - V6	Hypokinesia of lat wall	110	36	1.2	138	3.4	-
53	858241	ANNAMALAI	73	M	HT	160/100	Brain Stem deficits	Infarct	Normal CT	Normal CT (L) Capsuloganglionic region in repeat CT	N	-	120	30	1	130	3	-
54	860790	RAJENDRAN	61	M	HT	160/100	Brain Stem deficits	Infarct	Haemorrhage	(L) Capsuloganglionic	LVH Strain	-	100	30	1	134	3.2	-
55	860721	MUTHUSAMY	77	M	DM	120/70	Motor + Cortical	Infarct	Infarct	(L) Capsuloganglionic	N	-	280	20	1	140	4	-
56	864562	KADAMPATTI	65	F	HT	180/100	Pure Motor	Infarct	Infarct	(L) Capsuloganglionic	LVH Strain	-	90	40	1	138	4	-
57	867588	SUSEELA	50	F	-	130/80	Pure Motor	Infarct	Normal CT	Normal CT	N	-	80	40	1.2	140	5.2	-
58	860733	GANESH	32	M	CAHD	110/70	Pure Motor	Infarct	Infarct	® Capsuloganglionic	N	N	60	40	1.1	140	4	↑LDL
59	875873	VIRUTHAMMAL	80	F	HT	160/80	Pure Motor	Infarct	Infarct	(L) Capsuloganglionic lacunar infarct	LVH Strain	-	100	20	1.2	138	4.2	-
60	875836	THAIYAL NAYAGI	60	F	DM	120/70	Pure Motor	Infarct	Haemorrhage	® Capsuloganglionic	N	-	210	20	1.1	132	4	-
61	861595	RENGASAMY	60	M	HT	150/80	Brain Stem Deficit	Infarct	Normal CT	Normal CT	N	-	110	30	1.2	140	5	-

62	861629	JEYAPAUL	35	M	HT	140/100	Pure motor hemiplegia	Infarct	Normal CT	Ⓑ Capsuloganglionic	N	-	70	36	1.4	138	4.2	-
63	864312	UTHAMA RAJAN	60	M	HT	160/100	Pure motor hemiplegia	Infarct	Haemorrhage	Ⓑ Capsuloganglionic	N	-	130	20	1	140	5	-
64	865111	GOPAL	46	M	CAHD	170/90	Pure motor hemiplegia	Infarct	Infarct	Ⓑ Fronto parietal region	N	N	140	40	1	138	4.2	↑TGL ↑LDL
65	865130	RAMAIYAN	65	M	HT	170/110	Brain stem deficits	Infarct	Normal CT	Normal CT	LVH Strain	-	120	42	1	136	4.2	-
66	868932	ARUMUGAM	60	M	HT	160/100		Infarct	Normal CT	Normal CT	LVH Strain	-	130	36	1.2	136	4.6	-
67	869928	MAHALINGAM	54	M	HT	150/90	Motor + Cortical sensory deficits	Infarct	Normal CT	Normal CT	N	-	120	38	1	140	5.2	↑LDL
68	870636	PITCHAI	65	M	HT	150/90	Brain stem deficits	Infarct	Normal CT	Normal CT	N	-	100	38	1	140	4	-
69	870593	RADHAKRISHNAN	40	M	DM	120/60	Motor + cortical deficits	Infarct	Infarct	Ⓑ Capsuloganglionic	N	-	220	36	1	138	4	-
70	877660	ELIZAPATH	76	F	CAHD	130/80	Pure motor hemiplegia	Infarct	Infarct	(L) Capsuloganglionic	ST↓ V1 - V6	Hypokinesia of Ant wall	120	42	1.2	136	5.2	↑LDL ↑Chol
71	876574	SATHYA	77	F	DM	160/100	Motor + Cortical	Haemorrhage	Infarct	Ⓑ Fronto Parietal with midline shift (L)	LVH Strain	LV Diastolic Dysfunction	200	26	1	130	4.8	-
72	863879	ALAGAMMAL	63	F	HT	140/90	Pure motor hemiplegia	Haemorrhage	Haemorrhage	Capsuloganglionic (L)	N	-	140	32	1	140	4.2	-
73	832470	NATESAN	70	M	HT	150/90	Pure motor hemiplegia	Infarct	Infarct	Capsuloganglionic (L)	N	-	100	42	1.2	138	4.8	-
74	865382	KATHAYEE	70	F	DM	140/80	Brain stem deficits	Infarct	Normal CT	Normal CT	N	-	210	40	1	140	5	-
75	833178	PALANIVEL	42	M	CAHD	100/70	Motor + cortical deficits	Infarct	Infarct	Ⓑ Capsuloganglionic	N	N	130	38	1	136	5.2	-
76	869177	DHARMAMBAL	78	F	HT	160/110	Pure motor hemiplegia	Infarct	Infarct	Ⓑ Capsuloganglionic	LVH Strain	-	120	30	1	140	4	-
77	870079	ANNAPATTU	60	F	-	110/70	Pure motor hemiplegia	Infarct	Infarct	(L) Capsuloganglionic	N	-	100	32	1	138	4.2	-
78	871707	BALAMBAL	60	F	HT	150/90	Pure motor hemiplegia	Haemorrhage	Haemorrhage	(L) Capsuloganglionic	N	-	110	30	1.4	140	4.4	-
79	833204	RAJARAM	70	M	DM	120/70	Pure motor hemiplegia	Infarct	Infarct	(L) Capsuloganglionic	N	-	320	34	1.2	136	4.6	-
80	872395	VAIRAKANNU	60	F	HT	160/110	Cerebellar signs	Haemorrhage	Haemorrhage	Ⓑ Cerebellar hemisphere	LVH Strain	-	120	36	1	140	4.2	-
81	872525	MANGALAM	75	F	HT	170/100	Pure motor hemiplegia	Infarct	Infarct	Ⓑ Capsuloganglionic	N	-	110	40	1	138	4.4	↑LDL
82	873378	CHINNAMANI	52	F	HT	170/110	Pure motor hemiplegia	Haemorrhage	Haemorrhage	Brain stem pons	LVH Strain	-	100	42	1.2	140	4.6	-
83	834041	THIRUNAVUKARASU	72	M	HT	160/100	Pure motor hemiplegia	Haemorrhage	Haemorrhage	Ⓑ Capsuloganglionic	N	-	130	38	0.8	142	4.4	-
84	874197	NEELAMBAL	65	F	HT	170/100	Pure motor hemiplegia	Infarct	Infarct	Ⓑ Capsuloganglionic	N	-	120	30	1	136	4.2	-

85	876865	DHARMAVALLI	75	F	DM	130/80	Opthalmoplegia with crossed Hemiparesis (weber)	Haemorrhage	Haemorrhage	Brain Stem midbrain with midline shift	N	-	240	36	0.8	130	4.4	-
86	83489	GOVINDAN	70	M	CAHD	120/80	Pure motor hemiplegia	Infarct	Infarct	® Capsuloganglionic	T↓ V1 - V6	Hypokinesia of Ant wall	110	38	0.8	140	4.2	↑LDL
87	889611	SARASWATHY	40	F	DM	100/60	Motor + cortical deficits	Haemorrhage	Haemorrhage	(L) Capsuloganglionic	N	-	220	40	0.8	138	4	-
88	835687	SUNDARAMOORTHY	70	M	HT	160/100	Pure motor hemiplegia	Infarct	Infarct	® Capsuloganglionic	N	-	110	40	0.8	136	4.2	-
89	837242	MARIMUTHU	70	M	DM	110/70	Motor + Cortical	Infarct	Infarct	® Capsuloganglionic	N	-	200	40	1.4	140	4.8	-
90	836696	KALIYAN	35	M	HT	170/100	Pure motor hemiplegia	Haemorrhage	Haemorrhage	® Capsuloganglionic	N	-	100	38	1.2	136	5	-
91	842235	PAKIRISAMY	70	M	HT	160/110	Frontal Lobe dysfunction	Infarct	Infarct	(L) Capsuloganglionic	LVH Strain	-	110	40	1.2	138	5.2	-
92	843335	SUBRAMANI	72	M	HT	160/110	Pure Sensory Deficit	Infarct	Normal CT	Normal CT	N	-	120	42	1	140	5.4	-
93	844110	DHATHNAMOORTHY	80	M	DM	120/80	Motor + Cortical	Infarct	Infarct	® Capsuloganglionic	ST↓ I AVL V1 - V4	-	240	44	1	138	4.2	-
94	845045	JOHN SUNDAR	65	M	CAHD	130/80	Pure motor hemiplegia	Infarct	Infarct	Brain stem pons	ST↓ V1 - V6	Hypokinesia of Ant wall	110	40	1.2	140	4.4	↑TGL ↑LDL
95	845476	MARUTHANAYAGAM	80	M	DM	130/70	Pure motor hemiplegia	Infarct	Infarct	Capsuloganglionic	N	-	180	42	1	138	4.8	-
96	848951	VASUDEVAN	60	M	DM	140/70	Pure motor hemiplegia	Infarct	Infarct	® Capsuloganglionic	N	-	160	44	1	140	5	-
97	850694	ETHIRAJ	65	M	HT	170/110	Motor + Cortical	Infarct	Infarct	® Capsuloganglionic	LVH Strain	-	140	40	1	138	4.2	-
98	852993	CHANDRAN	40	M	DM	130/80	Pure motor hemiplegia	Infarct	Infarct	(L) Capsuloganglionic	N	-	170	42	1	140	4.4	-
99	857107	PERIYASAMY	55	M	HT	170/110	Motor + Cortical	Infarct	Infarct	(L) Capsuloganglionic	N	-	100	44	1.2	140	4.1	-
100	882424	VELLACHAMY	38	M	DM (Type 1)	140/90	Motor + Cortical	Infarct	Infarct	(L) Capsuloganglionic	N	-	110	46	1.4	140	4.2	↑LDL
101	853244	VADIVEL	22	M	HT	170/100	Brain Stem deficits	Infarct	Normal CT	Normal CT	N	-	120	30	1	142	4.2	-
102	855085	SUBARMANIYAN	70	M	DM	100/70	Pure motor hemiplegia	Infarct	Infarct	(L) Capsuloganglionic	N	-	200	32	1.2	130	4.4	-