INTRODUCTION

Stroke is the leading cause of morbidity . Cerebrovascular disease includes ischemic and haemorrhagic stroke. They are the major cause of disability.

Risk factors include smoking, diet , alcohol consumption and associated non communicable disease.

Stroke -sudden neurological deficit due to a vascular Cause.

Diagnosis of stroke involves clinical and laboratory study to support diagnosis.

Neuroimaging techniques such as MRI Brain and CT Brain remain the most reliable tool for evaluating and diagnosing stroke.

There are several other causes such as seizure, intracranial tumours, migraine, metabolic and septic encephalopathy that mimic stroke.

It is essential to differentiate between these conditions and to diagnose stroke in order to start acute treatment such as thrombolysis or thrombectomy.

Rapid diagnosis of stroke is needed for use of treatments such as thrombolysis or thrombectomy.

However ,most of the patients with stroke do not seek medical attention on their own, since most of the patients lose their consciousness.

It is often the bystander or family members, who bring the patient to hospital.

Hence they should be counseled to call emergency medical service , if they witness any person with weakness of limbs, loss of consciousness, headache..etc.

Hence in order to make a rapid diagnosis of stroke, based on history and clinical findings, Siriraj stroke score system can be used.

This study is been conducted to validate the Siriraj stroke score system to differentiate infarct from haemorrhagic stroke.

Siriraj stroke score system can be used in rural areas, since this score is purely based on history and clinical findings.

This scoring system can be used in areas where brain imaging study is not possible.

Using an interviewed structured questionnaire, relevant data were collected.

Based on Siriraj stroke score system, if the patient is suspected to have ischemic stroke, administration of recombinant tissue plasminogen activator can be done and may be beneficial in restoring cerebral perfusion.

If the patient is suspected to have haemorrhagic stroke, anti-oedema measures can be started.

AIMS AND OBJECTIVES

1.To ascertain the clinical profile of ischemic and haemorrhagic stroke.

2. To determine the validity of Siriraj stroke score system in differentiating ischemic and haemorrhagic stroke.

Secondary objectives:

1.To analyse the risk factors associated with ischemic and haemorrhagic stroke.

2.To assess the incidence of stroke in each age group and sex distribution.

3. To assess the common symptoms associated with stroke.

4. To evaluate the incidence of stroke and its severity associated with risk factors.

REVIEW OF LITERATURE

Stroke is defined as an abrupt onset of neurological deficit due to a focal neurological deficit. Definition of stroke is based on clinical and brain imaging.

TRANSIENT ISCHEMIC ATTACK :

Transient ischemic attack (TIA) is defined by the condition in which all neurological signs and symptoms usually resolve within 24 hrs of onset without evidence of brain infarction in brain imaging.

STROKE :

If neurological signs and symptoms lasts for more than 24 hrs or if brain infarction is demonstrated on brain imaging, then the condition is termed as stroke.

Stroke is a leading cause of morbidity and mortality worldwide [12, 13].

The World Health Organization defines stroke as a rapidly developing clinical syndrome of focal disturbance of cerebral function lasting longer than 24 hours which leads to death with no cause other than vascular origin.[11]

The disturbance of cerebral function due to vascular cause could be caused by three morphological abnormalities. i.e. (a) stenosis , (b) occlusion, or (c) rupture of arteries [23].

Cerebral infarction accounts for approximately 80 % of the stroke as opposed to 9-15% due to intracerebral bleed [24].

Stroke incidence in Nigeria and other Sub-Saharan African countries is on the increase [14].

RISK FACTORS:

Factors driving this transition include changes in diet, cigarette smoking, alcohol consumption , inadequate exercise and increase in the prevalence of obesity and also increase in other non communicable disease such as hypertension and diabetes mellitus [15].

Neuroimaging techniques, especially the computerized tomography (CT) scan or magnetic resonance imaging, remain the most reliable tools for diagnosing stroke types [16-18].

However these imaging modalities are not readily available in Nigeria, especially in rural communities, and when available, they are not easily affordable due to the prevailing poverty and high cost [12, 19].

Angina pectoris, diabetes mellitus, intermittent claudication and history of transient ischemic attack were taken as atheroma markers [20].

Patients with systolic blood pressure 140 mmHg and above or diastolic blood pressure 90mmHg were categorized as hypertensive [22].

SIRIRAJ STROKE SCORE SYSTEM :

The Siriraj stroke score system :

(2.5 * level of consciousness) + (2* headache)+ (2 *[vomiting]+ [0.1 * diastolic blood pressure] - 12 - [3 * atheroma marker]).

A score < -1 = infarction , a score > +1 = haemorrhage , while a score of -1 to +1 = indeterminate [21].

In this regard , based on clinical presentation SSS may be a good bedside tool to differentiate cerebral haemorrhage from infarction [23, 27].

Head CT scan allows the accurate diagnosis between the above two main groups but may miss small lesions especially of posterior fossa or if attempted early (less than 12 hours) of stroke [28].

Bedside scoring system for easy and correct evaluation of stroke. Guy's Hospital score based on eight variables but requiring cumbersome

calculations showed that 89 % of cerebral infarction and 55 % of cerebral haemorrhage could be correctly diagnosed at bed side [25].

Von Arbin's study showed 69 % correct bed side diagnosis [26].

WATERSHED INFARCT :

Low cerebral blood flow for a prolonged duration results in watershed infarct, that is infarction in border zones between major cerebral artery distribution.

Intracranial haemorrhage is due to bleeding into brain or around the brain, that causes neurological deficit by mass effect on neurological structures, or by raising intracranial tension, or by toxic effects of blood itself.

Stroke is a commonest problem among inpatients at Siriraj hospital .[1]

Doctor in charge of acute stroke have therefore to resort to the usual routine procedures , which shows to be unreliable. [2-5]

Ischemic stroke have multitude of possible causes, characterized b : 25% cardioembolic , 25% lacunar (small- vessel disease) and 25% due to other causes, with variation in proportions by population [31]. In contrast acute coronary syndromes are overwhelmingly due to rupture or erosion

of an atheromatous plaque, with formation of thrombus on the plaque, which causes arterial obstruction [32].

There is recognition that acute coronary syndromes involve arteroembolic mechanisms of occluding distal coronary arteries, similar to ischemic stroke caused by embolism from the carotid artery[33,34].

In case of small- vessel strokes, pathologic examination suggested that microatheroma with rupture is the most common cause of occlusion of vessels followed by microembolism [35, 36].

For many cases, stroke cause remain unidentified . Many of the cases of strokes have imaging that supports an embolic mechanism; hence, labeled as embolic stroke of undetermined source [37].

Head CT can rule out diagnosis of major stroke in most of the cases in which ischemic changes are evident, but it is less sensitive in diagnosing minor stroke [38-40].

For all cases of acute stroke syndrome, CT angiography should be taken followed by plain head CT [41,42]. Identifying occluded intracranial vessel and of the extracranial carotid and vertebral, aortic arch and great vessels is needed for the management of transient ischemic attack or minor stroke and major ischemic stroke [43].

In cases of hemorrhagic stroke, intracranial CT angiography will identify intracranial aneurysm as the cause of subarachnoid hemorrhage

or show the source of bleeding in intracerebral hemorrhage as a “spot sign”[44].

STROKE SYNDROMES :

Stroke divided into

1) anterior circulation stroke ,
2) small vessel stroke.
3) Posterior circulation stroke.

History and clinical examination can localize the region of brain dysfunction.

ANTERIOR CIRCULATION STROKE :

Anterior circulation is formed by internal carotid artery .

These vessels could be occluded either by atherosclerosis, dissection or by embolic occlusion.

Occlusion of any of the intracranial vessel has specific clinical manifestations.

Middle cerebral artery :

Cortical branches of MCA supplies the entire lateral surface of cerebral hemisphere, except frontal pole, strip along the superomedial border of frontal and parietal lobes, lower temporal and occipital pole.

Occlusion of proximal MCA is mostly caused by embolus rather than intracranial atherothrombosis.

Proximal MCA [M1 segment] gives rise to lenticulostriate branches that supply posterior limb of internal capsule, adjacent corona radiata , globus pallidum, putamen and caudate nucleus.

In the sylvian fissure, MCA [M2 segment] divides into superior and inferior divisions.

Superior division supplies frontal and superior parietal cortex.

Inferior division supplies inferior parietal and temporal cortex.

CORTICOSPINAL TRACT (Pyramidal) SYSTEM:

Upper motor neuron fibres which arise from premotor cortex, Betz cells in the cerebrum. These fibres are responsible for contralateral motor supply of upper limb, lower limb and trunk. These fibres descend to form corona radiate and pass through middle third of posterior limb of internal capsule.

It descends and pass through middle $3/5^{th}$ of cerebral peduncle and enter into midbrain. It is divided into bundles by transverse pontine fibres. It crosses in the lower part of medulla to the contralateral side.

A small portion of fibres remain uncrossed and descend in the anterior column. It does not synapse in the anterior horn cells directly. It synapse in the posterior horn cells and from there through internuncial neurons, synapse in to anterior horn cells.

In the cerebral hemisphere, upper limb, trunk are represented on the lateral surface of the hemisphere, whereas leg and foot are represented medially.

Any lesion in the pyramidal tract will lead to contralateral hemiparesis. It causes hypertonia in the antigravity muscles on the

contralateral upper limb and lower limb. And hence flexors of upper limb and extensors of lower limb are affected the most.

Whereas weakness is more in extensors of upper limb and flexors of lower limb. Tone distribution is opposite to that of weakness.

Abdominal reflex will be absent. Babinski's sign will be positive.

Distal movements are first affected and last to recover. Muscle wasting occur due to disuse atrophy of limbs.

Deep tendon reflexes will be exaggerated, whereas superficial abdominal reflex will be absent. Deep abdominal reflex will be exaggerated. This dissociation of superficial and deep abdominal reflex is characteristic feature of corticospinal tract involvement.

LOWER MOTOR NEURON:

Lower motor neuron arise from anterior horn cells and brainstem nuclei. It supplies the motor end plate. Any lesion in lower motor neuron, leads to wasting, paralysis of muscles, absent deep tendon reflexes. Plantar reflex may be absent or flexion.

Pseudo- Babinski sign :

Plantar extensor occur in the absence of pyramidal tract lesion. Causes are deep sleep, coma , deep anaesthesia, narcotics, infants, paralysis of flexors of toes, Cheyne- Stokes respiration, during the period of apnea.

MCA Occlusion:

If MCA is occluded at its origin, then the clinical manifestations are contralateral hemiplegia, homonymous hemianopia, and gaze preference to ipsilateral side, hemianaesthesia.

Because of facial weakness, dysarthria is more common.

When dominant hemisphere is involved , global aphasia occurs.

When non dominant hemisphere is involved , constructional apraxia, anosognosia , and neglect occur.

Complete MCA syndromes occurs, when an embolus occludes the stem of the artery.

Partial MCA syndromes occurs , in case of occlusion of the distal branches of the artery.

Partial syndromes include brachial syndrome [arm and hand weakness] alone ,

Facial weakness and nonfluent aphasia with or without arm weakness [frontal opercular syndrome].

Occlusion of proximal superior division leads to infarct in frontal and parietal cortices. It results in a combination of sensory disturbance, motor weakness, and broca's aphasia.

Occlusion of inferior division of MCA supplying dominant hemisphere results in infarct in temporal cortex resulting in Wernicke's aphasia. Contralateral homonymous superior quadrantanopia , jargon speech, loss of comprehension of written and spoken language occurs.

Occlusion of inferior division of MCA supplying non dominant hemisphere leads to hemineglect or spatial agnosia without weakness.

Lacunar stroke occurs when there is occlusion of lenticulostriate vessels.

It leads to pure motor stroke or sensory-motor stroke, occurring contralateral to the lesion.

Infarct in genu of internal capsule leads to facial weakness followed by upper limb weakness and lower limb weakness, as the infarct progresses posteriorly within the internal capsule.

Clumsy hand, dysarthria lacunar syndrome in which contralateral hand become ataxic and there will be prominent dysarthria.

Lacunar infarction involving globus pallidus and putamen leads to parkinsonism and hemiballismus.

ANTERIOR CEREBRAL ARTERY:

ACA branches- precommunal [A1] and postcommunal [A2].

The precommunal stem connects the internal carotid artery to anterior communicating artery.

Postcommunal segment lies distal to the anterior communicating artery.

Precommunal segment [A1] give rise to penetrating branches which supply anterior hypothalamus, anterior limb of internal capsule, anterior perforator substance , amygdala and inferior part of head of caudate nucleus.

Occlusion of proximal ACA is less severe because of collaterals through MCA and PCA and collaterals through anterior communicating artery.

Occlusion of single A2 segment results in involvement of

-Motor leg area : paralysis of contralateral leg and foot.

-Sensory area for foot and leg : cortical sensory loss over leg, foot and toes.

-Sensorimotor area in paracentral lobule : urinary incontinence.

-Arm area of cortex or fibres descending to corona radiate : lesser degree of paralysis of contralateral arm.

-Medial surface of posterior frontal lobe : contralateral sucking reflex, grasp reflex , gegenhalten paratonic rigidity.

-Frontal cortex near motor leg area : gait apraxia [impairment of stance and gait].

-Corpus callosum : tactile aphasia in left limbs and dyspraxia of left limbs.

-Medial inferior portion of frontal, parietal and temporal lobes and cingulate gyrus : abulia [akinetic mutism] , lack of spontaneity, delay, slowness, interrupted interruption , whispering and reflex distraction to sounds and sights.

In conditions such as contralateral A1 segment atresia, both A2 segments arise from single anterior cerebral stem. In such cases, occlusion may affect both hemispheres. It results in

- bilateral pyramidal signs and symptoms with paraparesis or quadriparesis,

-abulia [delay in verbal and motor response].

-urinary incontinence.

ANTERIOR CHOROIDAL ARTERY :

It arises from internal carotid artery. It supplies posterior limb of internal capsule and geniculocalcarine fibres and white matter posterolateral to posterior limb of internal capsule.

Occlusion of anterior choroidal artery leads to

-contralateral hemiplegia,

-hemianesthesia,

- homonymous hemianopia.

Only minimal deficit occur and patient recover substantially, since this territory is also supplied by posterior communicating artery, posterior choroidal artery, and by penetrating vessels of proximal MCA.

Causes of occlusion of anterior choroidal artery :

1)insitu thrombosis of vessel.

2)iatrogenic occlusion [during clipping of aneurysm arising from ICA].

INTERNAL CAROTID ARTERY:

Clinical features of occlusion of internal carotid artery depends on the etiology, whether the cause for infarct is due to embolus , thrombus or low flow.

MCA territory is most commonly affected. Due to extensive anastomosis of circle of Willis, occlusion may not produce clinical features.

If occlusion occurs at the top of the carotid artery , at the origin of ACA and MCA, results in hemiplegia , hemianaesthesia, abulia or stupor, aphasia or anosognosia .

When PCA arise from internal carotid artery, such condition is termed as fetal PCA. In such cases, it results in peripheral territory symptoms such as,

-Calcarine cortex or nearby optic radiation : upper quadrantic homonymous hemianopia.

-Bilateral occipital lobe with parietal lobe involvement : Cortical blindness, achromatopia, bilateral homonymous hemianopia , awareness or denial of blindness , tactile naming , failure to see to and fro movements, inability to count or enumerate objects.

-Posterior part of corpus callosum and dominant calcarine lesion : colour anomia , verbal alexia without agraphia.

-Bilateral hippocampal lesion or lesion on dominant lobe alone :memory defect.

-Lesion of non dominant , calcarine and lingual gyrus : topographic disorientation and prosopagnosia.

-Calcarine cortex [dominant hemisphere] :teleopsia , metamorphopsia , palinopsia , unformed visual hallucination , peduncular hallucinosis , central photophobia.

-Nondominant hemisphere : complex hallucinations.

CENTRAL TERRITORY, THALAMIC SYNDROME :

-Loss of all modalities of sensation , choreoathetosis , dysesthesias and spontaneous pain , intention tremor , mild hemiparesis, spasm of hand : posteroventral nucleus of thalamus; involvement of subthalamus or its afferent tracts.

-Thalamoperforate syndrome : Claude's syndrome - ipsilateral 3rd nerve palsy with crossed cerebellar ataxia : dentatothalamic tract and 3rd nerve.

-Weber's syndrome : third nerve palsy with contralateral hemiplegia : third nerve and cerebral peduncle.

-Skew deviation of eyes, paralysis of vertical eye movements, sluggish pupillary response to light, ptosis and mild miosis [associated retraction nystagmus and tucking of eyelids may be present : supranuclear fibres to 3rd nerve, interstitial nucleus of Cajal , posterior commissure, nucleus of Darkschewitsch.

-Dentatothalamic tract: contralateral ataxic action tremor ; rhythmic postural tremor or holding tremor [rubral tremor].

Internal carotid artery give rise to ophthalmic artery , that supplies optic nerve and retina. Hence ICA occlusion may lead to amaurosis fugax - recurrent transient monocular blindness.

Patients often complaints of blurring of vision .

Tightly stenotic lesion leads to high pitched prolonged carotid bruit . As the stenosis increases, bruit becomes fainter and in case of imminent occlusion , bruit disappear.

COMMON CAROTID ARTERY :

Occlusion may result in clinical features similar to ICA occlusion. Low flow in external carotid artery may lead to jaw claudication. Takayasu's arteritis may lead bilateral common carotid artery occlusion at their origin.

POSTERIOR CIRCULATION STROKE :

Posterior circulation is formed by paired PCA , basilar artery and paired vertebral artery. Paired vertebral artery joins together at pontomedullary junction to form basilar artery. Basilar artery give rise to paired PCA at interpeduncular fossa. Occlusion of each artery give rise to distinct clinical features.

POSTERIOR CEREBRAL ARTERY :

Occlusion of PCA leads to P1 syndrome and P2 syndrome.

P1 syndrome: occlusion of thalamogeniculate, posterior choroidal , and percheron arteries leads to midbrain, thalamic and subthalamic signs.

P2 syndrome : occlusion of PCA distal to the junction of PCA with the posterior communicating artery leads to cortical temporal and occipital lobe signs.

P1 syndrome : Claude's syndrome- ipsilateral 3rd nerve palsy with contralateral ataxia.

Weber's syndrome- ipsilateral 3rd nerve palsy with contralateral hemiplegia.

Hemiplegia is due to cerebral peduncle involvement. Ataxia is due to involvement of red nucleus or dentatorubrothalamic tract.

Subthalamic nucleus involvement leads to contralateral hemiballismus.

Occlusion of artery of Percheron leads to drowsiness, upward gaze paresis, abulia.

Bilateral PCA [proximal] occlusion leads to extensive infarction in midbrain and subthalamus, presenting as coma, bilateral pyramidal signs, unreactive pupil and decerebrate rigidity.

Thalamic Dejerine- Roussy syndrome : contralateral hemisensory loss followed by burning , agonizing pain in the affected area. Such pain is persistent and has poor response to analgesics. Anticonvulsants [gabapentin or carbamazepine] or tricyclic antidepressants may be given.

Occlusion of P1 segment leads to infarction in ipsilateral medial thalamus and subthalamus , cerebral peduncle and midbrain.

Medial midbrain syndrome : [occlusion of paramedian branches of proximal posterior cerebral arteries and upper basilar artery]

-involvement of 3^{rd} nerve fibers : eye - down and out , due to unopposed action of 4^{th} and 6^{th} cranial nerves. Dilated , unreactive pupils.[ipsilateral side]

- [contralateral side] paralysis of face, arm and legs- involvement of corticospinal and corticobulbar tracts descending in crus cerebri.

Lateral midbrain syndrome :[occlusion of penetrating arteries arising from PCA]

-[ipsilateral] involvement of 3rd nerve nucleus or third nerve fibers:

Eyes down and out with dilated, unreactive pupuil due to unopposed action of fourth and sixth cranial nerves.

-[contralateral] hyperkinesias, tremor and hemiataxia : involvement of dentatorubrothalamic pathway and red nucleus.

P2 syndromes : Occlusion of distal PCA leads to infarction in medial temporal and occipital lobe.

Usual manifestation of distal PCA occlusion is contralateral homonymous hemianopia with macula involvement. Whereas MCA occlusion will cause hemianopia with macula sparing as calcarine cortex is supplied by P2 segment of MCA.

If only calcarine cortex is involved and visual association areas are spared, patient will be aware of visual defects.

If lesion involves dominant hemisphere medial temporal lobe and hippocampus, patient will experience acute disturbance in memory. But soon, patient will recover as the memory has bilateral representation.

If the infarct extends and involve splenium of corpus callosum, then patient will be having alexia without agraphia.

Visual agnosia for faces, colours, objects, mathematical symbols and amnestic aphasia [anomia with paraphasic errors] can occur without callosal involvement.

Peduncular hallucinosis [visual hallucination of brightly coloured objects and scenes] occur with occlusion of PCA.

Anton's syndrome :

Bilateral infarction of distal PCA results in cortical blindness. [blindness with intact papillary light reaction]. Patient often denies blindness. Islands of vision may persist, and patient may only complaint of fluctuation of vision , as images are captured in the preserved portion.

Rarely central vision is preserved and peripheral vision is lost resulting in 'gun barrel' vision.

Balint's syndrome:

Watershed infarct in territory between distal PCA and MCA will lead to infarct in visual association areas bilaterally, which often occurs after cardiac arrest due to low blood flow.

It is a disorder of visual scanning of objects in environment.

Palinopsia- persistence of visual image for few minutes, inspite of gazing at another object.

Asimultanagnosia – difficulty in synthesizing the whole of an image.

Embolic occlusion at top of basilar artery will lead to acute onset of somnolence, bilateral signs such as ptosis, absent papillary light reaction, papillary asymmetry, myoclonic jerks and posturing that simulate seizure.

Diagnosis is made by noncontrast CT scan which shows hyperdense **'basilar artery sign'** or CT angiography which establishes the diagnosis.

It can produce any central or peripheral territory symptoms. Hence any patient presenting with cranial nerve deficits and new onset seizure, differential diagnosis of embolic occlusion of top of basilar artery should be kept, as it is one of the potentially treatable condition.

VERTEBRAL ARTERY AND POSTERIOR INFERIOR CEREBELLAR ARTERIES:

Vertebral artery arises from subclavian artery on left and from innominate artery on right side .

Vertebral artery is divided into four segments.

Segment 1 : from origin to its entry into 5^{th} or 6^{th} transverse vertebral foramen.

Segment 2 : portion of vertebral artery which traverse the vertebral foramen from C6 to C2.

Segment 3 : part of vertebral artery which exits from foramen and arches around atlas and pierces the dura at foramen magnum.

Segment 4 : part of vertebral artery which ascends upward to join the other vertebral artery to form basilar artery.

Only fourth segment give rise to branches that supplies cerebellum and brainstem.

Proximal portion of posterior inferior cerebellar arteries supply lateral medulla and its distal branches supply inferior surface of cerebellum.

First and fourth segment of vertebral artery are more prone for atherothrombotic lesion.

Atherothrombotic lesion in first segment of vertebral artery may produce posterior circulation stroke due to emboli.

When one vertebral artery is diseased, collateral circulation from contralateral vertebral artery, occipital artery, thyrocervical, ascending cervical will prevent low flow TIA.

When one vertebral artery is atretic and other vertebral artery is diseased at its origin , resulting in low flow TIA leads to vertigo , syncope and alternating hemiplegia.

SUBCLAVIAN STEAL SYNDROME :

Occlusion of subclavian artery , results in subclavian steal syndrome and the occlusion lies proximal to origin of vertebral artery will lead to reversal of flow in vertebral artery. On exercising the ipsilateral upper limb , there will be increased reversal of flow in vertebral artery , resulting in posterior circulation TIA. Such condition is termed as " subclavian steal syndrome" .

Second and third segment of vertebral artery are more prone to get affected by osteophytic spurs, dissection and fibromuscular dysplasia.

LATERAL MEDULLARY SYNDROME:

Thrombosis or embolic occlusion of 4th segment of vertebral artery leads to infarction of lateral medulla and the condition is termed as " Lateral medullary syndrome" or "Wallenberg's syndrome" .

Occlusion of any of the following vessels may lead to lateral medullary syndrome.

-vertebral artery .

-posterior inferior cerebellar artery .

-superior lateral medullary arteries.

-middle lateral medullary arteries .

-inferior lateral medullary arteries.

Signs and symptoms :

On the side of lesion ,

-descending tract and nucleus of fifth nerve involved –numbness, pain and impaired sensation over one half of face.

-vestibular nucleus – nausea, vomiting, vertigo, nystagmus, diplopia, oscillopsia.

-nucleus and tractus solitaries- loss of taste.

-nucleus cuneatus and nucleus gracilis- numbness of ipsilateral lower limb, trunk and upper limb.

-descending sympathetic tract involvement leading to Horner's syndrome.

-ninth and tenth nerve fibers leading to dysphagia, paralysis of palate, reduced gag reflex, hoarseness of voice, vocal cord paralysis.

-genuflected UMN fibers to facial nucleus –weakness of lower face.

-restiform body, cerebellar fibers, cerebellar hemisphere – ataxia and falling to the same side of lesion.

Contralateral side: due to involvement of spinothalamic tract, loss of temperature and pain sensation over half of the body, sometimes involving face.

MEDIAL MEDULLARY SYNDROME :

It occurs due to occlusion of branches of lower basilar artery [or] vertebral artery [or] branches of vertebral artery.

Ipsilateral 12^{th} nerve involvement : paralysis and atrophy of one half of tongue.

Involvement of contralateral medial lemniscus and pyramidal tract : paralysis of upper limb and lower limb, sparing face. Loss of proprioception and tactile sensation over one half of the body.

Occlusion of vertebral artery results in total unilateral medullary syndrome [combination of lateral and medial medullary syndromes].

Occlusion of vertebral artery results in lateral pontomedullary syndrome [combination of lateral inferior pontine syndrome and lateral medullary syndrome].

BASILAR ARTERY SYNDROME [similar to lone vertebral artery syndrome]:

Combination of any brainstem syndromes and syndrome of posterior circulation stroke.

Bilateral involvement of corticospinal tract and corticobulbar tracts:

Quadriparesis and involvement of all bulbar musculature.

Infarction of cerebellum leads to respiratory arrest due to increased intracranial tension , hydrocephalus, obstruction of aqueduct of sylvius, central herniation.

It leads to drowsiness, nausea, vomiting, headache, gait ataxia, which are early manifestation of cerebellar infarction. Neurosurgical decompression may be required, to prevent complications.

Differential diagnosis : viral labyrinthitis.

Unilateral dysmetria , headache and neck stiffness suggest stroke.

Basilar artery : It supplies base of the pons and superior cerebellum.

Its branches are divided into three categories ;

-long circumferential branches, that supply cerebellum.

-short circumferential branches, that supply lateral portion of pons, superior and middle cerebellar peduncles.

-paramedian branches, that supply a portion of pons on either side of midline.

Atheromatous lesion often involves distal portion of vertebral artery and proximal portion of basilar artery. Rarely dissection of vertebral artery may involve basilar artery , may result in multiple penetrating artery strokes depending on the location of true and false lumen.

Most common cause of occlusion of distal portion of basilar artery are emboli from heart or from proximal vertebral artery. It may also arise from atherothrombosis of basilar artery, resulting in “top of the basilar” syndromes.

It is essential to identify whether complete basilar artery occlusion or one of its branches has been occluded, because of therapeutic significance.

It is important to identify impending basilar artery occlusion, before the occurrence of infarction. History of multiple episodes of TIA and fluctuating stroke are important , as it indicates impending occlusion of proximal basilar artery or distal vertebral artery.

Complete occlusion of basilar artery will produce,

-multiple cranial nerve deficits,

-both motor and sensory bilateral long tract signs,

-cerebellar dysfunction,

- posturing movements [myoclonic], that simulate seizure activity.

-complete infarction of lower midbrain and pons , leading to "locked-in" syndrome, which is a state of quadriplegia with preserved consciousness, with multiple cranial nerve deficits.

TIAs of branches of basilar artery leads to clinical features that are unilaterally restricted, whereas TIAs of basilar artery leads to bilateral features. Usually TIAs of basilar artery are short lived, and they indicate impending basilar artery occlusion.

Proximal basilar artery TIAs produce diplopia, vertigo, dysarthria, circumoral numbness and facial numbness. Initial symptom of occlusion of basilar artery Treatment of such TIAs are antiplatelet agents or intravenous heparin or endovascular intervention.

Basilar artery occlusion may produce bilateral brain stem infarction resulting in internuclear ophthalmoplegia or gaze paresis with ipsilateral hemiparesis.

Occlusion of branches of basilar artery cause ipsilateral symptoms. If symptoms remain unilateral for a prolonged time, then occlusion of basilar artery is less likely, as it produce bilateral motor, sensory and cranial nerve deficits.

Occlusion of superior cerebellar artery leads to ipsilateral cerebellar ataxia , dysarthria , vomiting , nausea, ipsilateral upper extremity ataxic tremor , palatal myoclonus , deafness, Horner's syndrome and contralateral loss of pain and temperature over trunk , upper and lower limb and face , due to the involvement of spinothalamic and trigeminothalamic tract.

Occlusion of anterior inferior cerebellar artery produces,

-ipsilateral hearing loss, nausea , vomiting, vertigo , tinnitus , facial weakness , Horner's syndrome , cerebellar ataxia , conjugate lateral gaze paralysis.

-contralateral loss of temperature and pain.

SMALL VESSEL STROKE :

Occlusion of small penetrating arteries lead to small vessel stroke / lacunar infarction. Size of the infarct ranges from 3mm to 2cm. Major risk factors are hypertension and elderly age group.

Types of small vessel stroke :

1)Pure motor stroke due to infarct in pons or posterior limb of internal capsule.

2)Pure sensory stroke due to infarct in thalamus [ventral].

3)infarct in pons or internal capsule, results in ataxic hemiparesis.

4)infarct in genu of internal capsule or pons leads to clumsy hand and dysarthria.

Prognosis in small vessel stroke is better than large vessel stroke. However in some, residual disability may persist.

If recombinant tissue plasminogen activator (alteplase) is given within 4.5hrs of ischemic stroke, it has been shown in randomized controlled trials to reduce functional disability, and has an absolute risk reduction of 7%- 13% in comparison to placebo; thus it is the leading specific treatment for the management of infarct type of stroke[46- 49]. Treatment efficacy wanes rapidly, and risk of harm increases with time

elapsed from symptom onset; hence the need for diagnosis and treatment[50].

Most of the patients with ischemic stroke arrive more than 4.5 hrs after stroke and hence are not eligible for thrombolysis[56]. Many centres have shown that in most of the cases , delay in initiating treatment is caused by hesitation to contact hospital after the onset of symptom [57,58].

The Endovascular treatment for the treatment for small core and anterior circulation proximal occlusion with emphasis on minimizing CT to recanalization times (ESCAPE) trial, has shown that endovascular thrombectomy using devices will reduces both mortality and morbidity[51].

The ESCAPE findings shows results from the Netherlands[52] and study from Australia[53]. Many studies, one from United States and from Spain, shows further confirmatory findings[54,55].

Differentiating between transient ischemic attack and minor stroke has been found to be not particularly useful in improving prognosis since both have association with early and 90-day risks of causing major ischemic stroke, and immediate investigation and treatment are therefore necessary for TIA[43,70,71].

Cohort studies have shown that upto one-third of inpatients with a clinical diagnosis of transient ischemic attack (no residual deficits on

examination) have evidence of ischemia on MRI[72]. Almost all patients with minor stroke have small infarcts that can be visualized by MRI[73].

In this way, transient ischemic attack and minor stroke are conceptually similar to non-ST-segment elevation MI [74]. The risk of disabling stroke after transient ischemic attack or minor stroke is substantial and is heavily "front- end loaded" [75]. Recognition and early (i.e., same- day) evaluation and treatment may substantively reduce this risk [76-78]. Dual antiplatelet regimens for the short -term are warranted and continue to be investigated[45,59].

Carotid artery revascularization and anticoagulation for atrial fibrillation are proven effective treatments that prevent stroke [60,61]. For patients without these conditions, transient ischemic attack present a golden opportunity to prevent future stroke by intervening on lifestyle and vascular risk factors [62].

Specialized coronary care units are prevalent worldwide, but the same is not true for stroke units [63]. Randomized controlled trials have shown conclusively that patients treated in dedicated stroke units fare better than those treated in general wards without the same ready access to personnel trained and experienced in managing stroke [64-67].

In a trial, for stroke shows significant reduction in- hospital mortality (odds ratio or 0.50, 95%), case fatality rate (OR 0.45,95% CI

0.28-0.71, one-year mortality (95% CI 0.42-0.84) and discharge to home (95% CI 0.38-0.98) [68].

Patients with stroke who receive organized inpatient care in a stroke unit are more likely to be alive, independent .[65,68,69].

INTRACEREBRAL HAEMORRHAGE :

Cerebral haemorrhage is found in 5- 10% of patients with stroke in developed countries [6] , in developing countries, the lesion is commoner, because of hypertension. Globally stroke is the second leading cause of death[29]. Stroke affects both sexes equally and causes major economic burden to society. [30].

At Siriraj hospital, cerebral haemorrrage accounted for around 50 % of cases of stroke. [7]. Guy's hospital score was developed clinical for diagnosis of intracranial haemorrhage [8], and was validated by others. [9].

The prevalence of stroke differs between European and Asian population [10] , and predictive value of diagnostic score depends on prevalence of disease and hence Siriraj stroke score can not be applied in all areas.

It contributes around 10% of stroke cases. Causes of ICH include cerebral amyloid angiopathy, hypertension , bleeding disorders , coagulopathy and sympathomimetic drugs.

HYPERTENSIVE ICH :

It occurs due to spontaneous rupture of deep penetrating vessels in brain. Most common sites are putamen, basal ganglia, thalamus and cerebellum. If ICH occurs at other sites, then other causes such as coagulopathy, tumour, cerebral amyloid angiopathy should be excluded.

Clinical features :

Headache, nausea , vomiting, seizure, contralateral hemiparesis. If brainstem compression occurs, then it results in irregular, deep breathing, anisocoria, decerebrate rigidity.

Thalamic haemorrhage may lead to contralateral hemiparesis, loss of all modalities of sensation. Bleed into dominant thalamus may lead to aphasia with preserved verbal repetition. Haemorrhage into nondominant thalamus may lead to constructional apraxia and mutism.

Ocular disturbance in thalamic haemorrhage due to the extension into upper midbrain occur. Homonymous visual field defect, eyeball deviation medially and downwards, skew deviation, absent pupillary

response, vertical gaze paralysis, difficulty in convergence , retraction nystagmus and ipsilateral Horner's syndrome.

Some patients may develop chronic, contralateral pain syndrome termed as Dejerine-Roussy syndrome.

Pontine haemorrhage may lead to sudden loss of consciousness, quadriplegia, pinpoint pupil that react to light, impaired Doll's eye movement, hypertension, hyperhidrosis and hyperpnea are common.

CEREBELLAR HAEMORRHAGE :

It leads to occipital headache, vomiting, gait ataxia, dysarthria, dysphagia, conjugate gaze paresis towards the side of the lesion, ocular bobbing, skew deviation , blepharospasm and later leads to hydrocephalus, brainstem compression, and coma ensues.

Immediate surgical evacuation and decompression by external ventricular drainage are life saving procedures. If deep cerebellar nuclei are not involved, prognosis is good.

LOBAR HAEMORRHAGE :

Focal headache, vomiting, seizures, aphasia if dominat temporal lobe is involved, hemianopsia if occipital lobe is involved, arm weakness if frontal lobe haemorrhage occurs, and hemisensory loss if parietal lobe is involved.

May lead to coma if brainstem compression occurs.

OTHER CAUSES OF ICH :

-Cerebral amyloid angiopathy in which arteriolar degeneration followed by amyloid deposition occurs. It is most common among elderly and causes recurrent lobar haemorrhage.

Definitive diagnosis is by demonstration of congo red staining in cerebral vessels. Lumbar puncture may show inflammatory changes [CAA induced inflammation]. In such condition, steroids may be beneficial.

For noninflammatory CAA, there is no specific treatment. PET scan using specific antibody labeling may be useful in diagnosing CAA.

Drug induced ICH:

Sympathomimetics such as cocaine , methamphetamine causes ICH, most commonly among young age people. They cause acute raise in blood pressure, thereby causing lobar haemorrhage.

Anticoagulants may cause ICH if not properly monitored.

-head injury.

- bleeding disorders, coagulopathy, hematologic malignancies such as leukemia may cause bleeding at any site.

-tumours such as glioblastoma multiforme, medulloblastoma, renal cell carcinoma , malignant melanoma, bronchogenic carcinoma and choriocarcinoma causes ICH and may be the first manifestation of tumour.

-sepsis can cause multiple petechial haemorrhages throughout white matter.

-primary intraventricular haemorrhage may be the first manifestation of vascular anomaly.

-Moyamoya disease may sometimes cause ICH, vasculitis such as polyarteritis nodosa, systemic lupus erythematosus are the other causes of ICH.

INVESTIGATIONS :

Complete blood count, peripheral smear, renal function test, liver function test, coagulation profile, PT/aPTT/ INR.

CT Brain, most of the time accurately diagnose supratentorial haemorrhage. MRI brain and CT angiography may be necessary in young patients in whom cause of ICH is not known.

As ICH causes raised intracranial tension, lumbar puncture is contraindicated as it may lead to herniation.

TREATMENT :

In patients with ICH , due to vit K antagonist, rapid reversal of coagulopathy with either prothrombin complex concentrates , vit K or fresh frozen plasma may be helpful. Prothrombin complex concentrates can partially reverse factor Xa inhibitors effect.

For cerebellar haemorrhages of size < 1cm with preserved consciousness and no focal brainstem signs, surgical evacuation is not indicated. Haemorrhage of size 1-3cm, only observation is done to look for signs of increase intracranial tension and impending brainstem compression.

For cerebellar haemorrhage of size > 3cm, surgical evacuation is indicated.

Tissues surrounding haematoma are compressed but not necessarily infracted. As haematoma is mostly reabsorbed, survivors of ICH have minimal deficit.

Aim of ICH treatment should me maintaining airway, maintaining cerebral perfusion pressure between 50- 70mmHg, osmotic agents in case of raised intracranial pressure.

MATERIALS AND METHODS

Study centre :

Institute of Internal Medicine, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai.

Study design : Observational study.

Duration of study : 6 months from February 2019 till July 2019.

Sample size : 200 cases.

Inclusion criteria for cases :

1)Age more than 40 yrs.

2)Time period : Within 6 hrs of onset of typical signs and symptoms of stroke.

3)First episode of stroke.

Exclusion criteria for cases:

1)Patients with stroke presenting after 6 hrs.

2)Age less than or equal to 40 yrs.

3)Recurrent stroke.

4)Hemiplegia due to nonvascular causes.

5)Patient with conditions such as pacemaker implantation , cochlear implant, claustrophobia in whom MRI is contraindicated.

Materials and methods :

All patients are subjected to thorough history taking and clinical examination.

All patients are subjected to neuroimaging by noncontrast CT brain and MRI brain.

After getting informed consent from patient / patient's attenders, and after removing metal objects from patients body, patient is made to lie on CT examination table, lying flat on his/ her back, straps and pillows are used to maintain the correct position.

CTbrain (2msv) will be taken.

After getting informed consent , MRI brain (3T) will be taken.

Data collection and methods:

Using pretested proforma, patients details, history, and clinical examination and investigation details are recorded and subjected to statistical analysis.

DISCUSSION

A Hospital – based observational study was conducted at our Institute of Internal Medicine, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai for a period of 6 months from February 2019 till July 2019

A total of 200 patients with stroke were selected for this study based on inclusion and exclusion criteria, as mentioned above.

AGE DISTRIBUTION:

Percentage of people with age group 60-65yrs were highest (26%) among stroke patients followed by 56-60 yrs which contribute 22.5% [table 1]. Patients in age group 45-50 yrs contribute the least , around 8.5% [fig 11].

SEX DISTRIBUTION :

Male (60.5) are more commonly affected than females (39.5 %) [table 2].

LEVEL OF CONSCIOUSNESS:

Most of the patients presented in a condition of stupor (34.5%), followed by drowsy state (28.5) [fig 13]. Most common symptom associated with stroke was vomiting (24%) followed by headache (21.5%).

Around 32.1% patients with haemorrhage were associated with vomiting[table 17].

Around 8.7% patients with infarct were associated with vomiting [fig 27].

RISK FACTORS:

-Around 22.5 % people were associated with type 2 Diabetes mellitus [table 6]. -Around 14 % people had H/o angina [table 7].

- Around 10 % people were associated with claudication [fig 18].

-Around 29 % patients with infarct had associated type 2 diabetes mellitus[table 21].

-Around 19.1 % patients with haemorrhage had associated type 2 diabetes mellitus. H/o diabetes mellitus was most commonly associated with infarct (29 %) [fig 31].

Significance of headache in stroke:

-Around 27.5 % patients with haemorrhage were associated with headache [table 19].

- Around 10.1 % patients with infarct were associated with headache [fig 29].

BASED ON SIRIRAJ STROKE SCORE SYSTEM :

- Around 65 % patients found to have haemorrhage and around 10 % patients had score suggestive of infarct and around 25% patients had equivocal results [fig 19].

-Patients in age group 60-65yrs are most frequently affected by haemorrhage [34 patients]. 14 patients had equivocal results and 4 had score suggestive of infarct [table 12].

- Patients in age group 56-60 yrs are the second most frequently affected by haemorrhage [27 patients]. 2 patients had score suggestive of infarct. 16 patients had equivocal results [table 12].

- Patients in age group 66- 70 yrs are the third most frequently affected by haemorrhage [24 patients]. 6 patients had equivocal results. 3 patients had score suggestive of infarct.[table 12].

- Among patients in age group above 70 yrs, 19 patients had score suggestive of haemorrhage, 9 patients had equivocal results and 2 patients had score suggestive of infarct. [table 12].

- Among patients in age group 51-55 yrs, 18 patients had score suggestive of haemorrhage, 5 patients had score suggestive of infarct. None of them had equivocal results.[table 12].

- Patients in age group 45-50 yrs, 8 patients had score in favour of haemorrhage, 4 patients had score in favour of infarct and 5 patients had equivocal score.[table 12].

-Among 200 patients , 130 patients had score suggestive of haemorrhage, 20 patients had score suggestive of infarct, 50 patients had equivocal results. [fig 22].

Similar to MRI brain findings, both males and females were most commonly affected by haemorrhage than infarct.

- Most of the patients with haemorrhage presented in a state of coma [36.9%].[fig 26].

- Most of the patients with infarct presented in a state of stupor [70%]. [fig 26].

- Among patients in coma , most of the patients had haemorrhage [36.9 %].[table 16].

BASED ON MRI BRAIN :

-Around 65.5 % patients had haemorrhage and 34.5 % had infarct.[fig 20].

-Patients in age group 60-65 yrs are the most frequently affected by haemorrhage and contributes around 24.4 % , followed by 66-70 yrs age group who contribute 19.1 %.[table 11].

- Patients in age group 60-65 yrs are the most frequently affected by infarct and contributes around 29 %, followed by 56-60 yrs age group who contribute 26.1%.[table 11].

-Among 56- 60 yrs , around 18 patients had infarct and 27 patients had haemorrhage. [table 11].

- Among 66-70 yrs , out of 33 patients, 8 had infarct and 25 had haemorrhage.[table 11].

- Among >70 yrs, 22 patients had haemorrhage and 8 had infarct. Thus most of the patients are affected by haemorrhage than infarct.[table 11].

-among 60-65 yr , who are most commonly affected by stroke [52 patients], most patients [32 patients] were affected by haemorrhage than infarct [20 patients].[table 11].

- Among 200 patients, 131 were affected by haemorrhage and 69 had infarct. [table 11].

SEX DISTRIBUTION:

Both males and females were most commonly affected by haemorrhage than infarct.[fig 23].

Among them , males are most commonly affected by haemorrhage than females and contribute around 59.5% , whereas females contribute around 40.5% [table 13].

LEVEL OF CONSCIOUSNESS :

-Most of the patients with haemorrhage presented in a state of coma [35.9 %]. [fig 25].

- Most of the patients with infarct presented in a state of stupor [44.9 %]. Among patients presented with coma, most of the patients had haemorrhage [35.9 %]. [table 15].

VALIDATION OF SIRIRAJ STROKE SCORE SYSTEM:

Cross tabulation between Siriraj stroke score and MRI brain finding [table 27] has been made and Pearson Chi-square calculated . p value < 0.001 which showed a significant correlation between Siriraj stroke score and MRI brain report.

Sensitivity of Siriraj stroke score - 88.1 % [table 28].

Specificity of Siriraj stroke score - 95.5 % . [table 28].

CONCLUSION

-As Siriraj stroke score has significant correlation with MRI brain finding, it can be used as a bed side test for evaluating patient with stroke and to differentiate between haemorrhage and infarct.

-It can be used in situations where CT brain / MRI brain are not feasible.

-Since type 2 Diabetes mellitus and systemic hypertension has strong correlation with both types of stroke, early diagnosis and adequate treatment of DM and SHT will significantly reduce the incidence of stroke.

LIMITATIONS OF STUDY

It cannot be used in patients < 40 yrs [young stroke].

-This scoring system is not validated to be used in recurrent stroke.

-It cannot be applied to patients with cochlear implant , pacemaker implant, claustrophobia in whom MRI brain is contraindicated .

-It does not take into other features such as coagulation profile, drug induced, vasculitis which plays a major role in causing stroke.

-Sample size is comparatively small. Hence it would have been better if sample is collected from various centres.

BIBLIOGRAPHY

1. Viriyavejakul A , Viruchakul M, Utsaha V, Sukondhabhant S. The incidence of neurological diseases in Siriraj Hospital, Siriraj Hospital Gazette 1976;28:1648-55.

2. Van Arbin M, De Faire U, Helmers C, et al. Accuracy of bedside diagnosis in stroke. Stroke 1981;12:288-93.

3. Allan CMC. Clinical diagnosis of the acute stroke syndrome. Q J Med 1983;42:515-23.

4. Harrison MJG. Clinical distinction of cerebral haemorrhage and infarction.pougad MedJ 1980;56:629-32.

5. Schaafsma S.On the differential diagnosis between cerebral haemorrhage and infarction. J Neurol Sci 1968;7:83-95.

6. Kurtzke JF,Epidemiology of cerebrovascular disease. In:McDowell F,Caplan I.R,eds.Cerebrovascular survey report for the National Institute of Neurological and Communicative Disorders and Stroke. Maryland: National Institute of Health, Public Health Service, 1985:1-34.

7. Viriyavejakul A, Poungvarin N, Internal medicine patient: an analysis of incidence, mortality rate of 27325 admissions. Siriraj Hospital Gazette 1982;34:501-10.

8. Allan CMC. The accurate diagnosis and prognosis of acyte stroke. Cambridge: University of Cambridge, 1984.(MD thesis).

9. Sandercock PAG, Allan CMC, Corston RN, et al.Clinical diagnosis of intracranial haemorrhage using Guy's Hospital score. BMJ 1985;291:1675-7. (Correction 1986;292:173).

10. Viriyavejakul A. Stroke in Asia: an epidemiological consideration. Clin Neuropharmacol 1990;13(suppl 3): S26-33.

11. Sacco RL, Kasner SE, Broderick JP, et al. An updated definition of stroke for the 21st century. A statement for healthcare professionals from the American Heart Association / American Stroke Association. Stroke. 2013;44:2064-2089.

12. Sherin A, Khan A, Rehman S, Shah NH, Shabbier G, Zarif M. Comparability and validity of Siriraj stroke score and Allen stroke score in differentiation of acute ischemic and haemorrhagic stroke. JPMI. 2011;25:206-216.

13. Imarhiagbe FA, Ogbeide E. Clinical-imaging dissociation in strokes in a southern Nigerian tertiary hospital: review of 123 cases. Niger J Hosp Pract.2011;8:3-7.

14. Myles DC, Girish M, Charles PW.Accuracy of the Siriraj and Guy's hospital stroke scores in urban South Africans. Stroke. 2007;38:62-68.

15. Chukwuonye II, Chuku A, John C, et al. Prevalence of overweight and obesity among adult Nigerians- a systematic review. Diabetes Metab Syndr Obes.2013;6:43-47.

16. Eze CU, Okaro AO, Ohagwu CC. Pattern of computed tomography findings in cerebrovascular accident patients in south eastern Nigeria- a retrospective study of 480 patients.Eur J Sci Res.2009;34:104-109.

17. Khan J, Rehman A. Comparison of clinical diagnosis and computerized tomography in ascertaining type of stroke. J Ayub Med Coll Abbottabad. 2005;17:65-67.

18. Sandercock PA, Allen CM, Corston RN, Harrison MJ, Warlow CP. Clinical diagnosis of intracranial haemorrhage using Guy's hospital score. BMJ. 1985;291:1675-1677.

19. Kolapo KO, Ogun SA, Danesi MA, Osalusi BS, Odusote KA. Validation study of the Siriraj stroke score in African Nigerians and evaluation of the discriminant values of its parameters: a preliminary prospective CT scan study. Stroke. 2006;37:1997-2000.

20. Poungavarin N, Viriyavejakul A, Kpomontric C.Siriraj-stroke and validation study to distinguish supratentorial intracerebral haemorrhage from infarction .BMJ.1991;302:1565-1567.

21. Besson G, Robert C, Hommel M, Perret J. Is it clinically possible to distinguish nonhemorrhagic infarct from hemorrhagic stroke? Stroke. 1995;26:1205-1209.

22. Chukwuonye II, Chuku A, Onyeonoro UU, Okpechi IG, Madukwe OO, Ogah OS. Prevalence of abdominal obesity in Abia state, Nigeria : results of a population – based house-to-house survey. Diabetes Metab Syndr Obes. 2013;6:285-291.

23. Park K. Epidemiology of chronic non-communicable diseases and condition. In: Park's Textbook of Preventive and social medicine, 14th ed. Jabalpur: M/s Banarsi Dass Bhanot, 1994; 258-9.

24. Seisjo BJ. Pathophysiology and treatment of focal cerebral ischaemia- part II. Mechanism of damage and treatment. J Neuro Surg 1992;77: 337-54.

25. Allen CMC. Clinical diagnosis of acute stroke from department of Neurology, Guy's hospital London. Quarterly Journal of Medicine. New series L- II, 1983;208:515-23.

26. Von Arbin M, Briton M, DeFaire U, Helmers C, Miah K, Murray V. Accuracy of side diagnosis of stroke. Stroke 1981;12:288-93.

27. Poungvarin N, Viriyavejakul A,Komontri C. Siriraj stroke score-bedside and validation study to distinguish supratentorial

intracerebral haemorrhage from infarction. Br Med J 1991;203: 1565-7.

28. Shroff MM, Shetty PG, Joshi P. Imaging in stroke. In : Medicine Update.Bhopal :Dristi Offset, APICON 1996;6:II:2-8.

29. Go As, Mozaffarian D, Roger VL, et al . American Heart Association Statistics Committee and Stroke Statistics Subcommittee.Heart disease and stroke statistics-2014 update: a report from the American Heart Association.Circulation 2014;129:e28-e292.

30. Tracking heart disease and stroke in Canada. Ottawa: Public Health Agency of Canada; 2009.

31. Ay H, Furie KI, Singhal A, et al. An evidence- based causative classification system for acute ischemic stroke. Ann Neurol 2005;58:688-97.

32. DeWood MA, Spores J, Notske R, et al. Prevalence of total coronary occlusion during the early hours of transmural myocardial infarction. N Engl J Med 1980;303:897-902.

33. Farb A, Burke AP, Tang AL, et al. Coronary plaque erosion without rupture into a lipid core. A frequent cause of coronary thrombosis in sudden coronary death. Circulation 1996;93 :1354-63.

34. Falk E. Unstable angina with fatal outcome:dynamic coronary thrombosis leading to infarction and/or sudden death. Autopsy

evidence of recurrent mural thrombosis with peripheral embolization culminating in total vascular occlusion. Circulation 1985;71:699-708.

35. Fisher CM.The arterial lesions underlying lacunes.Acta Neuropathol 1968;12:1-15.

36. Fisher CM. Lacunar strokes and infarcts: a review.Neurology 1982;32:871-6.

37. Hart RG, Diener HC, Coutts SB, et al. Embolic strokes of undetermined sorce: the case for a new clinical construct. Lancet Neurol 2014;13:429-38.

38. Barber PA, Hill MD, Eliasziw M, et al. Imaging of the brain in acute ischaemic stroke: comparison of computed tomography and magnetic resonance diffusion weighted imaging.J Neurol Neurosurg Psychiatry 2005;76:1528-33.

39. Kidwell CS, Alger JR, Di Salle F, et al. Diffusion MRI in patients with transient ischemic attacks. Stroke 1999;30:1174-80.

40. Kidwell CS,Chalela JA, Saver JL,et al. Comparison of MRI and CT for detection of acute intracerebral haemorrhage. JAMA 2004;292:1823-30.

41. Casaubon LK, Boulanger JM, Blacquiere D, et al. Heart and stroke foundation of Canada Canadian Stroke best practice

recommendations: hyperacute stroke care guidelines, update 2015.Int J stroke.2015.July 6.

42. Coutts SB, Wein TH, Lindsay MP, et al. Canadian best practice recommendations: secondary prevention of stroke guidelines, update 2014.Int J stroke 2015;10:282-91.

43. Coutts SB, Modi J, Patel SK, et al.CT/CT angiography and MRI findings predict recurrent stroke after transient ischemic attack and minor stroke:results of the prospective CATCH study. Stroke 2012;43:1013-7.

44. Demchuk AM, Dowlatshahi D, Rodriguez- Luna D, et al.Prediction of haematoma growth and outcome in patients with intracerebral haemorrhage using the CT angiography spot sign[PREDICT]. A prospective observational study. Lancet Neurol 2012; 11:307-14.

45. Kennedy J, Hill MD, Ryckborst KJ, et al. Fast assessment of stroke and transient ischemic attack to prevent early recurrence[FASTER]: a randomized controlled pilot trial. Lancet Neurol 2007;6:961-9.

46. Hacke W, Kaste M, Fieschi C, et al. Randomised double blind placebo controlled trial of thrombolytic therapy with intravenous alteplase in acute ischemic stroke [ECASS II].Second European-Australasian acute stroke study investigators. Lancet 1998;352:1245-51.

47. Hacke W , Kaste M,Bluhmki E, et al. Thrombolysis with alteplase 3 to 4.5 hrs after acute ischemic stroke.N Engl J Med 2008;359:1317-29.

48. Lees KR,Bluhmki E, von Kummer R, et al. Time to treatment with intravenous alteplase and outcome in stroke: an updated pooled analysis of ECASS, ATLANTIS, NINDS, and EPITHET trials. Lancet 2010;375:1695-703.

49. Tissue plasminogen activator for acute ischemic stroke. The National institute of neurological disorders and stroke rt-PA stroke study group. N Engl J Med 1995;333:1581-7.

50. Emberson J, Lees KR, Lyden P, et al. Effect of treatment delay , age and stroke severity on the effects of intravenous thrombolysis with alteplase for acute ischemic stroke: a meta-analysis of individual patient data from randomized trials. Lancet 2014;384:1929-35.

51. Goyal M, Demchuk AM, MenonBK, et al. ESCAPE Trial investigators. Randomized assessment of rapid endovascular treatment of ischemic stroke. N Engl J Med 2015;372:1019-30.

52. Berkhemer OA,Fransen PS, Beumer D, et al. A randomized trial of intraarterial treatment for acute ischemic stroke.N Engl J Med 2015;372:11-20.

53. Campbell BC,Mitchell PJ, Kleinig TJ, et al. EXTEND IA investigators. Endovascular therapy for ischemic stroke with perfusion-imaging selection.N Engl J Med 2015;372:1009-18.

54. Saver JL, Goyal M, Bonafe A, et al. SWIFT PRIME Investigators.stent-retriever thrombectomy after intravenous t-PA vs t-PA alone in stroke. N Engl J Med 2015;372:2285-95.

55. Jovin TG, Chamorro A, Cobo E, et al. REVASCAT Trial investigators. Thrombectomy within 8 hours after symptom onset in ischemic stroke. N Engl J Med 2015;372:2296-306.

56. Kleindorfer D, Kiessela B, Schneider A ,et al. Neuroscience institute. Eligibility for recombinant tissue plasminogen activator in acute ischemic stroke. A population based study,. Stroke 2004; 35:e27-9.

57. Barber PA, Zhang J, Demchuk AM,et al.why are stroke patients excluded from TPA therapy?an analysis of patient eligibility.Neurology 2001;56:1015-20.

58. Tong D, Reeves MJ, Hernandez AF, et al. Times from symptom onset to hospital arrival in the get with the guidelines stroke program 2002-2009: temporal trends and implications. Stroke 2012;43:1912-7.

59. Wang Y, Wang Y, Zhao X, et al. clopidogrel with aspirin in acute minor stroke or transient ischemic attack. N Engl J Med 2013;369:11-9.

60. Barnett HJ, Taylor DW, Eliasziw M, et al. benefit of carotid endarterectomy in patients with symptomatic moderate or severe stenosis. North American symptomatic endarterectomy trial collaborators. N Engl Med 1998;339:1415-25.

61. Hart RG, Halperin JL, Pearce LA, et al. lessons from the stroke prevention in atrial fibrillation trials. Ann Inter Med 2003;138:831-8.

62. Johnston DC , Hill MD.The patient with transient cerebral ischemia: a golden opportunity for stroke prevention. CMAJ 2004;170: 1134-7.

63. Hill MD. Stroke units in Canada . CMAJ 2002;167:649-50.

64. Indredavik B, Bakke F, Solberg R, et al. Benefit of a stroke unit: a randomized controlled trial. Stroke 1991;22:1026-31.

65. Collaborative systematic review of the randomized trials of organised inpatient [stroke unit] care after stroke. Stroke unit trialists' collaboration. BMJ 1997;314:1151-9.

66. Zhu HF, Newcommon NN, Cooper ME, et al. impact of a stroke unit on length of hospital stay and in-hospital fatality. Stroke 2009; 40:18-23.

67. Jorgensen HS, Nakayama H, Raaschou HO, et al.the effect of a stroke unit: reductions in mortality, discharge rate to nursing home,

length of hospital stay and cost. A community-based study. Stroke 1995;26:1178-82.

68. Candelise L, Gattinoni M, Bersano A, et al. stroke unit care for acute stroke patients: an observational follow-up study. Lancet 2007;369:299-305.

69. Seenan P, Long M, Langhorne P. Stroke units in their natural habitat: systematic review of observational studies. Stroke 2007;38:1886-92.

70. Johnston SC, Gress DR, Browner WS, et al. short term prognosis after emergency department diagnosis of TIA. JAMA 2000;284:2901-6.

71. Merwick A, Albers GW, Amarenco P, et al. Addition of brain and carotid imaging to the ABCD[2] score to identify patients at early risk of stroke after transient ischaemic attack: a multicentre observational study. Lancet Neurol 2010;9:1060-9.

72. Coutts SB, Eliasziw M, Hill MD, et al. Hyperacute MR imaging and clinical predictor risk score predict dependent outcome after minor stroke and TIA. Stroke 2007;38:492.

73. Albers GW, Caplan LR, Easton JD, et al. transient ischemic attack-proposal for a new definition. N Engl J Med 2002;347:1713-6.

74. Amsterdam EA, Wenger NK, Brindis RG, et al.2014 AHA/ACC guideline for the management of patients with non-ST elevation acute coronary syndromes: a report of the American College of

Cardiology/ American Heart Association task force on practice guidelines. Circulation 2014;130:e344-426.

75. Wu CM, McLaughlin K, Lorenzetti DL, et al. Early risk of stroke after transient ischemic attack: a systematic review and meta-analysis. Arch Intern Med 2007;167:2417-22.

76. Wu CM, Manns BJ, Hill MD, et al. rapid evaluation after high risk TIA is associated with lower stroke risk. Can J Neurol Sci 2009;36:450-5.

77. Lavallee PC, Meseguer E, Abbound H, et al. A transient ischemic attack clinic with round the clock access[SOS-TIA]: feasibility and effects. Lancet Neurol 2007;6:953-60.

78. Rothwell PM, Giles MF, Chandratheva A, et al. effect of urgent treatment of transient ischemic attack and minor stroke on early recurrent stroke [EXPRESS study]: a prospective population- based sequential comparison. Lancet 2007;370:1432-42.

S.NO	AGE	SEX	LEVEL OF CONSCIOUSNESS	VOMITING	HEADACHE	DIASTOLIC BPmmHg	H/O DM	ANGINA	CLAUDICATION	SIRIRAJ STROKE SCORE		MRI BRAIN
1	62 yrs	M	stupor	no	yes	110	yes	no	no	0.5	equivocal	infarct
2	45 yrs	F	alert	no	no	100	no	no	no	-2	infarct	infarct
3	72 yrs	M	drowsy	yes	no	120	no	no	no	4.5	haemorrhage	haemorrhage
4	64 yrs	M	coma	yes	yes	90	yes	no	yes	3	haemorrhage	haemorrhage
5	47 yrs	F	stupor	no	no	100	yes	yes	no	-2.5	infarct	infarct
6	58 yrs	F	coma	no	no	130	no	no	no	6	haemorrhage	haemorrhage
7	63 yrs	M	drowsy	yes	yes	120	yes	no	no	3.5	haemorrhage	haemorrhage
8	58 yrs	F	semicoma	no	no	110	no	yes	no	1	equivocal	infarct
9	53 yrs	M	stupor	yes	yes	90	no	no	no	3.5	haemorrhage	haemorrhage
10	47 yrs	F	coma	no	yes	120	yes	no	no	4	haemorrhage	haemorrhage
11	59 yrs	M	drowsy	yes	no	130	no	no	yes	2.5	haemorrhage	haemorrhage
12	73 yrs	M	stupor	yes	yes	100	no	no	no	4.5	haemorrhage	haemorrhage
13	64 yrs	F	coma	no	no	90	yes	yes	no	-1	equivocal	infarct
14	48 yrs	M	alert	no	yes	110	no	no	no	1	equivocal	haemorrhage
15	56 yrs	F	stupor	yes	no	110	no	no	no	3.5	haemorrhage	haemorrhage
16	61 yrs	M	semicoma	no	yes	100	yes	yes	no	2	haemorrhage	haemorrhage
17	60 yrs	M	coma	yes	no	120	yes	no	no	4	haemorrhage	haemorrhage
18	46 yrs	F	alert	no	yes	90	no	no	no	-1	equivocal	infarct
19	72 yrs	M	coma	yes	no	110	no	no	no	6	haemorrhage	haemorrhage
20	51 yrs	M	semicoma	no	yes	120	no	no	yes	4	haemorrhage	haemorrhage
21	54 yrs	M	coma	no	yes	100	yes	no	no	2	haemorrhage	haemorrhage
22	61yrs	F	drowsy	yes	no	110	no	no	no	3.5	haemorrhage	haemorrhage
23	68 yrs	M	stupor	yes	no	100	no	no	no	2.5	haemorrhage	haemorrhage
24	62 yrs	M	stupor	no	no	110	yes	yes	no	-1.5	infarct	haemorrhage

S.NO	AGE	SEX	LEVEL OF CONSCIOUSNESS	VOMITING	HEADACHE	DIASTOLIC BPmmHg	H/O DM	ANGINA	CLAUDICATION	SIRIRAJ STROKE SCORE		MRI BRAIN
25	52 yrs	M	stupor	no	no	110	yes	no	no	-1.5	infarct	infarct
26	48 yrs	M	drowsy	yes	no	120	no	no	no	4.5	haemorrhage	haemorrhage
27	76 yrs	F	coma	no	no	100	no	no	no	3	haemorrhage	haemorrhage
28	59 yrs	M	coma	no	yes	130	no	yes	no	5	haemorrhage	haemorrhage
29	64 yrs	F	stupor	no	no	120	yes	no	no	-0.5	equivocal	infarct
30	53 yrs	M	stupor	no	no	110	no	no	no	1.5	haemorrhage	infarct
31	66 yrs	F	drowsy	yes	yes	120	yes	no	yes	3.5	haemorrhage	haemorrhage
32	59 yrs	M	coma	no	no	100	no	no	no	3	haemorrhage	haemorrhage
33	68 yrs	M	drowsy	no	no	90	no	no	no	-0.5	equivocal	haemorrhage
34	70 yrs	F	stupor	no	yes	110	no	no	no	3.5	haemorrhage	haemorrhage
35	63 yrs	M	drowsy	yes	no	120	yes	yes	no	1.5	haemorrhage	haemorrhage
36	54 yrs	M	coma	no	yes	100	no	no	no	5	haemorrhage	haemorrhage
37	62 yrs	F	stupor	no	no	110	no	no	no	1.5	haemorrhage	haemorrhage
38	70 yrs	F	stupor	yes	no	90	yes	no	no	-1.5	infarct	haemorrhage
39	65 yrs	M	coma	no	yes	80	no	no	no	3	haemorrhage	haemorrhage
40	64 yrs	M	drowsy	no	yes	120	no	no	no	4.5	haemorrhage	haemorrhage
41	72 yrs	F	drowsy	no	no	90	no	no	no	-0.5	equivocal	infarct
42	59 yrs	M	coma	no	no	100	no	no	no	3	haemorrhage	infarct
43	58 yrs	M	stupor	no	no	110	no	no	no	1.5	haemorrhage	infarct
44	71 yrs	F	coma	no	no	120	no	no	no	5	haemorrhage	haemorrhage
45	68 yrs	F	coma	yes	no	100	no	no	no	5	haemorrhage	haemorrhage
46	49 yrs	M	drowsy	no	no	90	no	no	no	-0.5	equivocal	infarct
47	60 yrs	M	coma	no	yes	110	yes	no	no	3	haemorrhage	haemorrhage
48	69 yrs	M	stupor	no	no	100	no	yes	no	-2.5	infarct	infarct

S.NO	AGE	SEX	LEVEL OF CONSCIOUSNESS	VOMITING	HEADACHE	DIASTOLIC BPmmHg	H/O DM	ANGINA	CLAUDICATION	SIRIRAJ STROKE SCORE		MRI BRAIN
49	74 yrs	M	drowsy	yes	yes	110	no	no	no	5.5	haemorrhage	haemorrhage
50	64 yrs	M	stupor	no	no	100	no	yes	no	-2.5	infarct	infarct
51	49 yrs	F	coma	yes	no	120	no	no	no	7	haemorrhage	haemorrhage
52	69 yrs	M	semicoma	no	yes	120	yes	no	no	4	haemorrhage	haemorrhage
53	59 yrs	M	stupor	no	no	90	no	no	no	-0.5	equivocal	infarct
54	51 yrs	F	drowsy	no	no	110	no	no	no	1.5	haemorrhage	haemorrhage
55	72 yrs	M	stupor	yes	no	80	no	no	no	0.5	equivocal	haemorrhage
56	66 yrs	F	coma	no	yes	100	yes	no	yes	2	haemorrhage	haemorrhage
57	70 yrs	M	semicoma	no	no	110	no	no	no	4	haemorrhage	haemorrhage
58	52 yrs	F	stupor	no	no	110	no	no	no	1.5	haemorrhage	infarct
59	76 yrs	M	stupor	yes	no	100	no	no	no	2.5	haemorrhage	haemorrhage
60	61 yrs	F	coma	no	yes	120	no	no	no	7	haemorrhage	haemorrhage
61	63 yrs	M	drowsy	no	no	80	yes	yes	no	-4.5	infarct	infarct
62	60 yrs	F	semicoma	yes	no	110	no	no	no	6	haemorrhage	haemorrhage
63	58 yrs	M	stupor	no	no	100	no	no	no	0.5	equivocal	haemorrhage
64	67 yrs	M	drowsy	no	no	120	no	no	no	2.5	haemorrhage	haemorrhage
65	51 yrs	F	coma	no	no	100	no	no	no	3	haemorrhage	haemorrhage
66	59 yrs	M	drowsy	no	yes	130	yes	no	yes	2.5	haemorrhage	haemorrhage
67	50 yrs	M	stupor	no	no	110	no	no	no	1.5	haemorrhage	haemorrhage
68	62 yrs	M	coma	yes	no	90	no	yes	no	1	equivocal	haemorrhage
69	48 yrs	F	drowsy	no	no	100	no	no	no	0.5	equivocal	haemorrhage
70	57 yrs	F	stupor	no	no	110	no	no	no	1.5	haemorrhage	infarct
71	64 yrs	M	coma	no	yes	120	no	no	no	7	haemorrhage	haemorrhage
72	65 yrs	F	drowsy	yes	no	100	yes	yes	no	-0.5	equivocal	infarct

S.NO	AGE	SEX	LEVEL OF CONSCIOUSNESS	VOMITING	HEADACHE	DIASTOLIC BPmmHg	H/O DM	ANGINA	CLAUDICATION	SIRIRAJ STROKE SCORE		MRI BRAIN
73	56 yrs	M	stupor	no	no	110	yes	no	no	-1.5	infarct	infarct
74	66 yrs	F	coma	no	no	100	no	no	no	3	haemorrhage	haemorrhage
75	61 yrs	F	drowsy	no	yes	120	no	no	no	1.5	haemorrhage	infarct
76	70 yrs	M	stupor	yes	no	110	no	no	no	3.5	haemorrhage	haemorrhage
77	54 yrs	F	semicoma	no	no	90	no	no	no	2	haemorrhage	haemorrhage
78	60 yrs	F	stupor	no	no	100	yes	yes	no	-2.5	infarct	infarct
79	57 yrs	M	drowsy	no	yes	110	no	no	no	3.5	haemorrhage	haemorrhage
80	62 yrs	F	drowsy	no	no	120	no	no	no	2.5	haemorrhage	haemorrhage
81	67 yrs	M	stupor	yes	no	110	no	no	no	3.5	haemorrhage	haemorrhage
82	71 yrs	M	coma	no	no	120	yes	no	yes	2	haemorrhage	haemorrhage
83	77 yrs	F	stupor	no	no	90	no	no	no	-0.5	equivocal	infarct
84	58 yrs	M	stupor	no	no	100	no	no	no	0.5	equivocal	haemorrhage
85	63 yrs	F	semicoma	no	yes	110	no	no	no	6	haemorrhage	haemorrhage
86	72 yrs	M	coma	yes	no	120	no	no	no	7	haemorrhage	haemorrhage
87	52 yrs	M	drowsy	no	no	100	yes	no	yes	-2.5	infarct	infarct
88	59 yrs	F	semicoma	no	no	110	no	yes	no	1	equivocal	infarct
89	64 yrs	F	stupor	no	no	110	no	no	no	1.5	haemorrhage	haemorrhage
90	68 yrs	M	semicoma	no	no	90	no	no	no	2	haemorrhage	haemorrhage
91	73 yrs	M	coma	no	no	100	no	no	no	3	haemorrhage	haemorrhage
92	53 yrs	F	drowsy	yes	no	120	yes	no	no	1.5	haemorrhage	infarct
93	60 yrs	F	stupor	no	yes	110	no	no	no	3.5	haemorrhage	haemorrhage
94	65 yrs	M	drowsy	no	no	100	no	no	no	0.5	equivocal	infarct
95	69 yrs	M	coma	no	no	120	no	yes	no	2	haemorrhage	haemorrhage
96	74 yrs	M	stupor	yes	yes	110	yes	no	no	2.5	haemorrhage	haemorrhage

S.NO	AGE	SEX	LEVEL OF CONSCIOUSNESS	VOMITING	HEADACHE	DIASTOLIC BPmmHg	H/O DM	ANGINA	CLAUDICATION	SIRIRAJ STROKE SCORE		MRI BRAIN
97	60 yrs	M	coma	no	no	110	no	no	no	4	haemorrhage	haemorrhage
98	58 yrs	M	stupor	yes	no	100	no	no	no	2.5	haemorrhage	haemorrhage
99	71 yrs	F	drowsy	no	no	120	yes	no	yes	-0.5	equivocal	infarct
100	49 yrs	M	semicoma	no	no	90	no	no	no	2	haemorrhage	haemorrhage
101	63 yrs	M	stupor	no	no	110	no	no	no	1.5	haemorrhage	infarct
102	61 yrs	F	coma	yes	yes	120	no	no	no	9	haemorrhage	haemorrhage
103	59 yrs	M	drowsy	no	no	90	no	no	no	-0.5	equivocal	infarct
104	72 yrs	M	drowsy	no	no	100	no	no	no	0.5	equivocal	infarct
105	50 yrs	M	stupor	no	no	130	yes	yes	no	0.5	equivocal	infarct
106	64 yrs	F	stupor	no	no	120	no	no	no	2.5	haemorrhage	haemorrhage
107	62 yrs	M	drowsy	no	no	110	no	no	no	1.5	haemorrhage	infarct
108	57 yrs	M	coma	yes	no	90	no	no	no	4	haemorrhage	haemorrhage
109	73 yrs	F	drowsy	no	no	110	no	no	no	1.5	haemorrhage	haemorrhage
110	51 yrs	F	semicoma	no	yes	100	no	yes	no	2	haemorrhage	haemorrhage
111	65 yrs	M	stupor	no	no	120	no	no	no	2.5	haemorrhage	haemorrhage
112	63 yrs	F	drowsy	no	no	110	no	no	no	1.5	haemorrhage	haemorrhage
113	55 yrs	M	stupor	no	no	80	yes	no	yes	-4.5	infarct	infarct
114	74 yrs	F	semicoma	yes	no	110	no	no	no	6	haemorrhage	haemorrhage
115	52 yrs	F	coma	no	no	100	no	no	no	3	haemorrhage	haemorrhage
116	66 yrs	M	stupor	no	no	120	no	no	no	2.5	haemorrhage	haemorrhage
117	62 yrs	F	stupor	no	yes	110	no	no	no	3.5	haemorrhage	haemorrhage
118	64 yrs	M	drowsy	no	no	100	no	no	no	0.5	equivocal	infarct
119	53 yrs	M	stupor	yes	no	120	yes	no	no	1.5	haemorrhage	infarct
120	70 yrs	M	coma	no	no	110	no	no	yes	1	equivocal	infarct

S.NO	AGE	SEX	LEVEL OF CONSCIOUSNESS	VOMITING	HEADACHE	DIASTOLIC BPmmHg	H/O DM	ANGINA	CLAUDICATION	SIRIRAJ STROKE SCORE		MRI BRAIN
121	64 yrs	M	stupor	no	yes	90	yes	no	no	-1.5	infarct	infarct
122	70 yrs	M	drowsy	yes	no	110	no	yes	no	0.5	equivocal	infarct
123	55 yrs	M	coma	no	no	100	no	no	no	3	haemorrhage	haemorrhage
124	61 yrs	F	stupor	no	no	120	no	no	no	2.5	haemorrhage	haemorrhage
125	65 yrs	M	coma	no	no	100	no	no	no	3	haemorrhage	haemorrhage
126	71 yrs	F	drowsy	yes	no	110	no	no	no	3.5	haemorrhage	haemorrhage
127	56 yrs	M	stupor	no	no	120	no	no	no	2.5	haemorrhage	haemorrhage
128	62 yrs	F	coma	no	no	100	no	no	no	3	haemorrhage	haemorrhage
129	60 yrs	M	drowsy	no	no	120	yes	no	yes	-0.5	equivocal	haemorrhage
130	66 yrs	M	coma	no	yes	110	no	yes	no	3	haemorrhage	haemorrhage
131	72 yrs	F	drowsy	no	no	100	no	no	no	0.5	equivocal	haemorrhage
132	57 yrs	M	stupor	yes	no	110	no	no	no	2.5	haemorrhage	haemorrhage
133	63 yrs	F	coma	no	no	120	no	no	no	5	haemorrhage	haemorrhage
134	67 yrs	M	drowsy	no	no	90	no	no	no	-0.5	equivocal	infarct
135	73 yrs	M	coma	no	no	120	no	no	no	5	haemorrhage	haemorrhage
136	58 yrs	F	stupor	no	no	110	no	no	no	1.5	haemorrhage	haemorrhage
137	64 yrs	M	coma	no	no	100	no	no	no	3	haemorrhage	haemorrhage
138	68 yrs	M	drowsy	yes	no	80	yes	no	yes	-2.5	infarct	infarct
139	74 yrs	F	semicoma	no	no	110	no	no	no	4	haemorrhage	haemorrhage
140	59 yrs	F	drowsy	no	no	90	no	no	no	-0.5	equivocal	haemorrhage
141	65 yrs	M	stupor	no	no	120	no	no	no	2.5	haemorrhage	haemorrhage
142	69 yrs	F	drowsy	no	yes	110	no	no	no	3.5	haemorrhage	haemorrhage
143	75 yrs	M	drowsy	no	no	100	no	yes	no	-2.5	infarct	infarct
144	60 yrs	M	stupor	yes	no	100	yes	no	no	-0.5	equivocal	infarct
145	58 yrs	F	drowsy	no	no	110	no	no	no	1.5	haemorrhage	infarct
146	52 yrs	M	coma	yes	no	120	no	no	no	7	haemorrhage	haemorrhage

S.NO	AGE	SEX	LEVEL OF CONSCIOUSNESS	VOMITING	HEADACHE	DIASTOLIC BPmmHg	H/O DM	ANGINA	CLAUDICATION	SIRIRAJ STROKE SCORE		MRI BRAIN
147	54 yrs	M	stupor	no	no	110	yes	no	no	-1.5	infarct	infarct
148	60 yrs	M	drowsy	yes	no	100	no	no	no	2.5	haemorrhage	haemorrhage
149	66 yrs	F	coma	no	yes	120	yes	no	no	4	haemorrhage	haemorrhage
150	72 yrs	M	stupor	no	no	100	no	no	no	0.5	equivocal	haemorrhage
151	57 yrs	F	drowsy	no	no	90	no	yes	no	3.5	haemorrhage	haemorrhage
152	55 yrs	F	coma	no	no	130	no	no	no	6	haemorrhage	haemorrhage
153	61 yrs	M	stupor	no	no	120	no	no	yes	-0.5	equivocal	infarct
154	67 yrs	F	drowsy	no	no	110	no	no	no	1.5	haemorrhage	haemorrhage
155	56 yrs	M	stupor	yes	no	90	no	no	no	1.5	haemorrhage	haemorrhage
156	62 yrs	M	semicoma	no	no	100	no	no	no	3	haemorrhage	haemorrhage
157	68 yrs	F	drowsy	no	no	110	yes	no	yes	1.5	haemorrhage	haemorrhage
158	73 yrs	M	coma	no	yes	130	no	no	no	8	haemorrhage	haemorrhage
159	47 yrs	M	stupor	no	no	110	no	no	no	1.5	haemorrhage	haemorrhage
160	57 yrs	F	stupor	no	no	100	no	no	no	0.5	equivocal	infarct
161	63 yrs	F	coma	yes	no	120	no	no	no	7	haemorrhage	haemorrhage
162	69 yrs	M	drowsy	no	no	100	no	no	no	0.5	equivocal	infarct
163	74 yrs	M	coma	no	no	100	no	yes	no	0	equivocal	infarct
164	58 yrs	M	stupor	no	no	120	no	no	yes	-0.5	equivocal	infarct
165	64 yrs	F	semicoma	no	no	110	no	no	no	4	haemorrhage	haemorrhage
166	70 yrs	M	drowsy	no	no	130	no	no	no	3.5	haemorrhage	haemorrhage
167	75 yrs	M	coma	yes	no	120	yes	no	no	4	haemorrhage	haemorrhage
168	59 yrs	M	stupor	no	no	90	no	no	no	-0.5	equivocal	haemorrhage
169	65 yrs	F	drowsy	no	yes	100	no	no	yes	-0.5	equivocal	infarct
170	71 yrs	F	stupor	no	no	110	no	yes	no	-1.5	infarct	infarct

S.NO	AGE	SEX	LEVEL OF CONSCIOUSNESS	VOMITING	HEADACHE	DIASTOLIC BPmmHg	H/O DM	ANGINA	CLAUDICATION	SIRIRAJ STROKE SCORE		MRI BRAIN
171	47 yrs	M	coma	yes	yes	110	yes	yes	no	5	haemorrhage	haemorrhage
172	56 yrs	M	stupor	no	no	90	no	no	no	-0.5	equivocal	infarct
173	61 yrs	F	drowsy	no	no	120	no	no	no	2.5	haemorrhage	infarct
174	65 yrs	F	semicoma	no	no	100	no	no	no	0	equivocal	infarct
175	70 yrs	M	stupor	no	no	110	no	no	no	1.5	haemorrhage	infarct
176	62 yrs	M	coma	yes	no	130	no	no	no	8	haemorrhage	haemorrhage
177	48 yrs	F	stupor	no	no	110	yes	no	yes	-1.5	infarct	haemorrhage
178	57 yrs	M	drowsy	no	no	120	no	no	no	2.5	haemorrhage	haemorrhage
179	62 yrs	F	drowsy	no	no	100	no	no	no	0.5	equivocal	infarct
180	66 yrs	M	stupor	no	no	110	no	no	no	1.5	haemorrhage	haemorrhage
181	71 yrs	F	coma	no	yes	90	no	yes	no	1	equivocal	infarct
182	58 yrs	M	stupor	no	no	100	no	no	no	0.5	equivocal	infarct
183	64 yrs	M	drowsy	no	no	130	yes	no	no	0.5	equivocal	infarct
184	49 yrs	M	stupor	yes	no	110	no	no	no	3.5	haemorrhage	haemorrhage
185	58 yrs	F	drowsy	no	no	120	no	no	no	2.5	haemorrhage	haemorrhage
186	63 yrs	F	stupor	no	no	100	no	no	no	0.5	equivocal	infarct
187	67 yrs	M	coma	no	no	90	no	no	no	2	haemorrhage	infarct
188	72 yrs	M	semicoma	no	no	110	no	no	no	4	haemorrhage	haemorrhage
189	55 yrs	F	drowsy	no	no	100	no	yes	no	-2.5	infarct	infarct
190	68 yrs	M	stupor	no	yes	120	no	no	yes	1.5	haemorrhage	haemorrhage
191	54 yrs	F	stupor	yes	no	90	no	no	no	1.5	haemorrhage	haemorrha
192	60 yrs	M	drowsy	no	no	110	no	no	no	1.5	haemorrhage	infarct
193	64 yrs	M	stupor	no	no	100	no	no	no	0.5	equivocal	infarct
194	55 yrs	M	coma	no	no	120	yes	no	no	2	haemorrhage	haemorrhage

S.NO	AGE	SEX	LEVEL OF CONSCIOUSNESS	VOMITING	HEADACHE	DIASTOLIC BPmmHg	H/O DM	ANGINA	CLAUDICATION	SIRIRAJ STROKE SCORE		MRI BRAIN
195	66 yrs	M	stupor	no	yes	110	yes	no	yes	0.5	equivocal	haemorrhage
196	60 yrs	M	drowsy	no	no	90	no	no	no	-0.5	equivocal	infarct
197	62 yrs	F	coma	yes	no	110	no	no	no	6	haemorrhage	haemorrhage
198	49 yrs	M	stupor	no	no	100	no	yes	no	-2.5	infarct	infarct
199	64 yrs	F	coma	no	no	120	yes	no	no	2	haemorrhage	haemorrhage
200	56 yrs	M	drowsy	no	yes	100	no	yes	no	-0.5	equivocal	infarct

S.NO	AGE	SEX	LEVEL OF CONSCIOUSNESS	VOMITING	HEADACHE	DIASTOLIC BPmmHg	H/O DM	ANGINA	CLAUDICATION	SIRIRAJ STROKE SCORE		MRI BRAIN
25	52 yrs	M	stupor	no	no	110	yes	no	no	-1.5	infarct	infarct
26	48 yrs	M	drowsy	yes	no	120	no	no	no	4.5	haemorrhage	haemorrhage
27	76 yrs	F	coma	no	no	100	no	no	no	3	haemorrhage	haemorrhage
28	59 yrs	M	coma	no	yes	130	no	yes	no	5	haemorrhage	haemorrhage
29	64 yrs	F	stupor	no	no	120	yes	no	no	-0.5	equivocal	infarct
30	53 yrs	M	stupor	no	no	110	no	no	no	1.5	haemorrhage	infarct
31	66 yrs	F	drowsy	yes	yes	120	yes	no	yes	3.5	haemorrhage	haemorrhage
32	59 yrs	M	coma	no	no	100	no	no	no	3	haemorrhage	haemorrhage
33	68 yrs	M	drowsy	no	no	90	no	no	no	-0.5	equivocal	haemorrhage
34	70 yrs	F	stupor	no	yes	110	no	no	no	3.5	haemorrhage	haemorrhage
35	63 yrs	M	drowsy	yes	no	120	yes	yes	no	1.5	haemorrhage	haemorrhage
36	54 yrs	M	coma	no	yes	100	no	no	no	5	haemorrhage	haemorrhage
37	62 yrs	F	stupor	no	no	110	no	no	no	1.5	haemorrhage	haemorrhage
38	70 yrs	F	stupor	yes	no	90	yes	no	no	-1.5	infarct	haemorrhage
39	65 yrs	M	coma	no	yes	80	no	no	no	3	haemorrhage	haemorrhage
40	64 yrs	M	drowsy	no	yes	120	no	no	no	4.5	haemorrhage	haemorrhage
41	72 yrs	F	drowsy	no	no	90	no	no	no	-0.5	equivocal	infarct
42	59 yrs	M	coma	no	no	100	no	no	no	3	haemorrhage	infarct
43	58 yrs	M	stupor	no	no	110	no	no	no	1.5	haemorrhage	infarct
44	71 yrs	F	coma	no	no	120	no	no	no	5	haemorrhage	haemorrhage
45	68 yrs	F	coma	yes	no	100	no	no	no	5	haemorrhage	haemorrhage
46	49 yrs	M	drowsy	no	no	90	no	no	no	-0.5	equivocal	infarct
47	60 yrs	M	coma	no	yes	110	yes	no	no	3	haemorrhage	haemorrhage
48	69 yrs	M	stupor	no	no	100	no	yes	no	-2.5	infarct	infarct

S.NO	AGE	SEX	LEVEL OF CONSCIOUSNESS	VOMITING	HEADACHE	DIASTOLIC BPmmHg	H/O DM	ANGINA	CLAUDICATION	SIRIRAJ STROKE SCORE		MRI BRAIN
49	74 yrs	M	drowsy	yes	yes	110	no	no	no	5.5	haemorrhage	haemorrhage
50	64 yrs	M	stupor	no	no	100	no	yes	no	-2.5	infarct	infarct
51	49 yrs	F	coma	yes	no	120	no	no	no	7	haemorrhage	haemorrhage
52	69 yrs	M	semicoma	no	yes	120	yes	no	no	4	haemorrhage	haemorrhage
53	59 yrs	M	stupor	no	no	90	no	no	no	-0.5	equivocal	infarct
54	51 yrs	F	drowsy	no	no	110	no	no	no	1.5	haemorrhage	haemorrhage
55	72 yrs	M	stupor	yes	no	80	no	no	no	0.5	equivocal	haemorrhage
56	66 yrs	F	coma	no	yes	100	yes	no	yes	2	haemorrhage	haemorrhage
57	70 yrs	M	semicoma	no	no	110	no	no	no	4	haemorrhage	haemorrhage
58	52 yrs	F	stupor	no	no	110	no	no	no	1.5	haemorrhage	infarct
59	76 yrs	M	stupor	yes	no	100	no	no	no	2.5	haemorrhage	haemorrhage
60	61 yrs	F	coma	no	yes	120	no	no	no	7	haemorrhage	haemorrhage
61	63 yrs	M	drowsy	no	no	80	yes	yes	no	-4.5	infarct	infarct
62	60 yrs	F	semicoma	yes	no	110	no	no	no	6	haemorrhage	haemorrhage
63	58 yrs	M	stupor	no	no	100	no	no	no	0.5	equivocal	haemorrhage
64	67 yrs	M	drowsy	no	no	120	no	no	no	2.5	haemorrhage	haemorrhage
65	51 yrs	F	coma	no	no	100	no	no	no	3	haemorrhage	haemorrhage
66	59 yrs	M	drowsy	no	yes	130	yes	no	yes	2.5	haemorrhage	haemorrhage
67	50 yrs	M	stupor	no	no	110	no	no	no	1.5	haemorrhage	haemorrhage
68	62 yrs	M	coma	yes	no	90	no	yes	no	1	equivocal	haemorrhage
69	48 yrs	F	drowsy	no	no	100	no	no	no	0.5	equivocal	haemorrhage
70	57 yrs	F	stupor	no	no	110	no	no	no	1.5	haemorrhage	infarct
71	64 yrs	M	coma	no	yes	120	no	no	no	7	haemorrhage	haemorrhage
72	65 yrs	F	drowsy	yes	no	100	yes	yes	no	-0.5	equivocal	infarct

S.NO	AGE	SEX	LEVEL OF CONSCIOUSNESS	VOMITING	HEADACHE	DIASTOLIC BPmmHg	H/O DM	ANGINA	CLAUDICATION	SIRIRAJ STROKE SCORE		MRI BRAIN
73	56 yrs	M	stupor	no	no	110	yes	no	no	-1.5	infarct	infarct
74	66 yrs	F	coma	no	no	100	no	no	no	3	haemorrhage	haemorrhage
75	61 yrs	F	drowsy	no	yes	120	no	no	no	1.5	haemorrhage	infarct
76	70 yrs	M	stupor	yes	no	110	no	no	no	3.5	haemorrhage	haemorrhage
77	54 yrs	F	semicoma	no	no	90	no	no	no	2	haemorrhage	haemorrhage
78	60 yrs	F	stupor	no	no	100	yes	yes	no	-2.5	infarct	infarct
79	57 yrs	M	drowsy	no	yes	110	no	no	no	3.5	haemorrhage	haemorrhage
80	62 yrs	F	drowsy	no	no	120	no	no	no	2.5	haemorrhage	haemorrhage
81	67 yrs	M	stupor	yes	no	110	no	no	no	3.5	haemorrhage	haemorrhage
82	71 yrs	M	coma	no	no	120	yes	no	yes	2	haemorrhage	haemorrhage
83	77 yrs	F	stupor	no	no	90	no	no	no	-0.5	equivocal	infarct
84	58 yrs	M	stupor	no	no	100	no	no	no	0.5	equivocal	haemorrhage
85	63 yrs	F	semicoma	no	yes	110	no	no	no	6	haemorrhage	haemorrhage
86	72 yrs	M	coma	yes	no	120	no	no	no	7	haemorrhage	haemorrhage
87	52 yrs	M	drowsy	no	no	100	yes	no	yes	-2.5	infarct	infarct
88	59 yrs	F	semicoma	no	no	110	no	yes	no	1	equivocal	infarct
89	64 yrs	F	stupor	no	no	110	no	no	no	1.5	haemorrhage	haemorrhage
90	68 yrs	M	semicoma	no	no	90	no	no	no	2	haemorrhage	haemorrhage
91	73 yrs	M	coma	no	no	100	no	no	no	3	haemorrhage	haemorrhage
92	53 yrs	F	drowsy	yes	no	120	yes	no	no	1.5	haemorrhage	infarct
93	60 yrs	F	stupor	no	yes	110	no	no	no	3.5	haemorrhage	haemorrhage
94	65 yrs	M	drowsy	no	no	100	no	no	no	0.5	equivocal	infarct
95	69 yrs	M	coma	no	no	120	no	yes	no	2	haemorrhage	haemorrhage
96	74 yrs	M	stupor	yes	yes	110	yes	no	no	2.5	haemorrhage	haemorrhage

S.NO	AGE	SEX	LEVEL OF CONSCIOUSNESS	VOMITING	HEADACHE	DIASTOLIC BPmmHg	H/O DM	ANGINA	CLAUDICATION	SIRIRAJ STROKE SCORE		MRI BRAIN
97	60 yrs	M	coma	no	no	110	no	no	no	4	haemorrhage	haemorrhage
98	58 yrs	M	stupor	yes	no	100	no	no	no	2.5	haemorrhage	haemorrhage
99	71 yrs	F	drowsy	no	no	120	yes	no	yes	-0.5	equivocal	infarct
100	49 yrs	M	semicoma	no	no	90	no	no	no	2	haemorrhage	haemorrhage
101	63 yrs	M	stupor	no	no	110	no	no	no	1.5	haemorrhage	infarct
102	61 yrs	F	coma	yes	yes	120	no	no	no	9	haemorrhage	haemorrhage
103	59 yrs	M	drowsy	no	no	90	no	no	no	-0.5	equivocal	infarct
104	72 yrs	M	drowsy	no	no	100	no	no	no	0.5	equivocal	infarct
105	50 yrs	M	stupor	no	no	130	yes	yes	no	0.5	equivocal	infarct
106	64 yrs	F	stupor	no	no	120	no	no	no	2.5	haemorrhage	haemorrhage
107	62 yrs	M	drowsy	no	no	110	no	no	no	1.5	haemorrhage	infarct
108	57 yrs	M	coma	yes	no	90	no	no	no	4	haemorrhage	haemorrhage
109	73 yrs	F	drowsy	no	no	110	no	no	no	1.5	haemorrhage	haemorrhage
110	51 yrs	F	semicoma	no	yes	100	no	yes	no	2	haemorrhage	haemorrhage
111	65 yrs	M	stupor	no	no	120	no	no	no	2.5	haemorrhage	haemorrhage
112	63 yrs	F	drowsy	no	no	110	no	no	no	1.5	haemorrhage	haemorrhage
113	55 yrs	M	stupor	no	no	80	yes	no	yes	-4.5	infarct	infarct
114	74 yrs	F	semicoma	yes	no	110	no	no	no	6	haemorrhage	haemorrhage
115	52 yrs	F	coma	no	no	100	no	no	no	3	haemorrhage	haemorrhage
116	66 yrs	M	stupor	no	no	120	no	no	no	2.5	haemorrhage	haemorrhage
117	62 yrs	F	stupor	no	yes	110	no	no	no	3.5	haemorrhage	haemorrhage
118	64 yrs	M	drowsy	no	no	100	no	no	no	0.5	equivocal	infarct
119	53 yrs	M	stupor	yes	no	120	yes	no	no	1.5	haemorrhage	infarct
120	70 yrs	M	coma	no	no	110	no	no	yes	1	equivocal	infarct

S.NO	AGE	SEX	LEVEL OF CONSCIOUSNESS	VOMITING	HEADACHE	DIASTOLIC BPmmHg	H/O DM	ANGINA	CLAUDICATION	SIRIRAJ STROKE SCORE		MRI BRAIN
121	64 yrs	M	stupor	no	yes	90	yes	no	no	-1.5	infarct	infarct
122	70 yrs	M	drowsy	yes	no	110	no	yes	no	0.5	equivocal	infarct
123	55 yrs	M	coma	no	no	100	no	no	no	3	haemorrhage	haemorrhage
124	61 yrs	F	stupor	no	no	120	no	no	no	2.5	haemorrhage	haemorrhage
125	65 yrs	M	coma	no	no	100	no	no	no	3	haemorrhage	haemorrhage
126	71 yrs	F	drowsy	yes	no	110	no	no	no	3.5	haemorrhage	haemorrhage
127	56 yrs	M	stupor	no	no	120	no	no	no	2.5	haemorrhage	haemorrhage
128	62 yrs	F	coma	no	no	100	no	no	no	3	haemorrhage	haemorrhage
129	60 yrs	M	drowsy	no	no	120	yes	no	yes	-0.5	equivocal	haemorrhage
130	66 yrs	M	coma	no	yes	110	no	yes	no	3	haemorrhage	haemorrhage
131	72 yrs	F	drowsy	no	no	100	no	no	no	0.5	equivocal	haemorrhage
132	57 yrs	M	stupor	yes	no	110	no	no	no	2.5	haemorrhage	haemorrhage
133	63 yrs	F	coma	no	no	120	no	no	no	5	haemorrhage	haemorrhage
134	67 yrs	M	drowsy	no	no	90	no	no	no	-0.5	equivocal	infarct
135	73 yrs	M	coma	no	no	120	no	no	no	5	haemorrhage	haemorrhage
136	58 yrs	F	stupor	no	no	110	no	no	no	1.5	haemorrhage	haemorrhage
137	64 yrs	M	coma	no	no	100	no	no	no	3	haemorrhage	haemorrhage
138	68 yrs	M	drowsy	yes	no	80	yes	no	yes	-2.5	infarct	infarct
139	74 yrs	F	semicoma	no	no	110	no	no	no	4	haemorrhage	haemorrhage
140	59 yrs	F	drowsy	no	no	90	no	no	no	-0.5	equivocal	haemorrhage
141	65 yrs	M	stupor	no	no	120	no	no	no	2.5	haemorrhage	haemorrhage
142	69 yrs	F	drowsy	no	yes	110	no	no	no	3.5	haemorrhage	haemorrhage
143	75 yrs	M	drowsy	no	no	100	no	yes	no	-2.5	infarct	infarct
144	60 yrs	M	stupor	yes	no	100	yes	no	no	-0.5	equivocal	infarct
145	58 yrs	F	drowsy	no	no	110	no	no	no	1.5	haemorrhage	infarct
146	52 yrs	M	coma	yes	no	120	no	no	no	7	haemorrhage	haemorrhage

S.NO	AGE	SEX	LEVEL OF CONSCIOUSNESS	VOMITING	HEADACHE	DIASTOLIC BPmmHg	H/O DM	ANGINA	CLAUDICATION	SIRIRAJ STROKE SCORE		MRI BRAIN
147	54 yrs	M	stupor	no	no	110	yes	no	no	-1.5	infarct	infarct
148	60 yrs	M	drowsy	yes	no	100	no	no	no	2.5	haemorrhage	haemorrhage
149	66 yrs	F	coma	no	yes	120	yes	no	no	4	haemorrhage	haemorrhage
150	72 yrs	M	stupor	no	no	100	no	no	no	0.5	equivocal	haemorrhage
151	57 yrs	F	drowsy	no	no	90	no	yes	no	3.5	haemorrhage	haemorrhage
152	55 yrs	F	coma	no	no	130	no	no	no	6	haemorrhage	haemorrhage
153	61 yrs	M	stupor	no	no	120	no	no	yes	-0.5	equivocal	infarct
154	67 yrs	F	drowsy	no	no	110	no	no	no	1.5	haemorrhage	haemorrhage
155	56 yrs	M	stupor	yes	no	90	no	no	no	1.5	haemorrhage	haemorrhage
156	62 yrs	M	semicoma	no	no	100	no	no	no	3	haemorrhage	haemorrhage
157	68 yrs	F	drowsy	no	no	110	yes	no	yes	1.5	haemorrhage	haemorrhage
158	73 yrs	M	coma	no	yes	130	no	no	no	8	haemorrhage	haemorrhage
159	47 yrs	M	stupor	no	no	110	no	no	no	1.5	haemorrhage	haemorrhage
160	57 yrs	F	stupor	no	no	100	no	no	no	0.5	equivocal	infarct
161	63 yrs	F	coma	yes	no	120	no	no	no	7	haemorrhage	haemorrhage
162	69 yrs	M	drowsy	no	no	100	no	no	no	0.5	equivocal	infarct
163	74 yrs	M	coma	no	no	100	no	yes	no	0	equivocal	infarct
164	58 yrs	M	stupor	no	no	120	no	no	yes	-0.5	equivocal	infarct
165	64 yrs	F	semicoma	no	no	110	no	no	no	4	haemorrhage	haemorrhage
166	70 yrs	M	drowsy	no	no	130	no	no	no	3.5	haemorrhage	haemorrhage
167	75 yrs	M	coma	yes	no	120	yes	no	no	4	haemorrhage	haemorrhage
168	59 yrs	M	stupor	no	no	90	no	no	no	-0.5	equivocal	haemorrhage
169	65 yrs	F	drowsy	no	yes	100	no	no	yes	-0.5	equivocal	infarct
170	71 yrs	F	stupor	no	no	110	no	yes	no	-1.5	infarct	infarct

S.NO	AGE	SEX	LEVEL OF CONSCIOUSNESS	VOMITING	HEADACHE	DIASTOLIC BPmmHg	H/O DM	ANGINA	CLAUDICATION	SIRIRAJ STROKE SCORE		MRI BRAIN
171	47 yrs	M	coma	yes	yes	110	yes	yes	no	5	haemorrhage	haemorrhage
172	56 yrs	M	stupor	no	no	90	no	no	no	-0.5	equivocal	infarct
173	61 yrs	F	drowsy	no	no	120	no	no	no	2.5	haemorrhage	infarct
174	65 yrs	F	semicoma	no	no	100	no	no	no	0	equivocal	infarct
175	70 yrs	M	stupor	no	no	110	no	no	no	1.5	haemorrhage	infarct
176	62 yrs	M	coma	yes	no	130	no	no	no	8	haemorrhage	haemorrhage
177	48 yrs	F	stupor	no	no	110	yes	no	yes	-1.5	infarct	haemorrhage
178	57 yrs	M	drowsy	no	no	120	no	no	no	2.5	haemorrhage	haemorrhage
179	62 yrs	F	drowsy	no	no	100	no	no	no	0.5	equivocal	infarct
180	66 yrs	M	stupor	no	no	110	no	no	no	1.5	haemorrhage	haemorrhage
181	71 yrs	F	coma	no	yes	90	no	yes	no	1	equivocal	infarct
182	58 yrs	M	stupor	no	no	100	no	no	no	0.5	equivocal	infarct
183	64 yrs	M	drowsy	no	no	130	yes	no	no	0.5	equivocal	infarct
184	49 yrs	M	stupor	yes	no	110	no	no	no	3.5	haemorrhage	haemorrhage
185	58 yrs	F	drowsy	no	no	120	no	no	no	2.5	haemorrhage	haemorrhage
186	63 yrs	F	stupor	no	no	100	no	no	no	0.5	equivocal	infarct
187	67 yrs	M	coma	no	no	90	no	no	no	2	haemorrhage	infarct
188	72 yrs	M	semicoma	no	no	110	no	no	no	4	haemorrhage	haemorrhage
189	55 yrs	F	drowsy	no	no	100	no	yes	no	-2.5	infarct	infarct
190	68 yrs	M	stupor	no	yes	120	no	no	yes	1.5	haemorrhage	haemorrhage
191	54 yrs	F	stupor	yes	no	90	no	no	no	1.5	haemorrhage	haemorrha
192	60 yrs	M	drowsy	no	no	110	no	no	no	1.5	haemorrhage	infarct
193	64 yrs	M	stupor	no	no	100	no	no	no	0.5	equivocal	infarct
194	55 yrs	M	coma	no	no	120	yes	no	no	2	haemorrhage	haemorrhage

S.NO	AGE	SEX	LEVEL OF CONSCIOUSNESS	VOMITING	HEADACHE	DIASTOLIC BPmmHg	H/O DM	ANGINA	CLAUDICATION	SIRIRAJ STROKE SCORE		MRI BRAIN
195	66 yrs	M	stupor	no	yes	110	yes	no	yes	0.5	equivocal	haemorrhage
196	60 yrs	M	drowsy	no	no	90	no	no	no	-0.5	equivocal	infarct
197	62 yrs	F	coma	yes	no	110	no	no	no	6	haemorrhage	haemorrhage
198	49 yrs	M	stupor	no	no	100	no	yes	no	-2.5	infarct	infarct
199	64 yrs	F	coma	no	no	120	yes	no	no	2	haemorrhage	haemorrhage
200	56 yrs	M	drowsy	no	yes	100	no	yes	no	-0.5	equivocal	infarct