“CLINICAL OUTCOME OF STROKE IN RELATION TO ADMISSION DAY GLYCEMIC STATUS”

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CERTIFICATE

This is to certify that the dissertation titled **"CLINICAL OUTCOME OF STROKE IN RELATION TO ADMISSION DAY GLYCEMIC STATUS"** is the bona fide original work of **DR.G.VINOTH KHANNA** in partial fulfillment of the requirements for **M.D.Branch-I – (General Medicine)** Examination of the Tamilnadu DR. M.G.R Medical University to be held in APRIL 2013. The Period of study was from NOVEMBER 2011 to OCTOBER 2012.

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DECLARATION

I**, DR.G.VINOTH KHANNA** , solemnly declare that dissertation titled **"CLINICAL OUTCOME OF STROKE IN RELATION TO ADMISSION DAY GLYCEMIC STATUS"** is a bona fide work done by me at Govt. Tirunelveli Medical College and Hospital from November 2011 To October 2012 under the guidance and supervision of my unit chief **PROF.M.R.VAIRAMUTHURAJU,M.D**., Professor of Medicine.

This dissertation is submitted to Tamilnadu DR. M.G.R Medical University, towards partial fulfillment of requirement for the award of **M.D. Degree (Branch-I) in GENERAL MEDICINE.**

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CONTENTS

1. INTRODUCTION

Cerebrovascular disorders are most devastating disorders of mankind. They are supposed to be the third most common cause of death among developed nations. For India , numerous community surveys have shown a prevalence of 200 cases per 1,00,000 persons. It indicates that burden of stroke in INDIA constitutes 1.5 % of all urban hospital admissions and 4.5 % of all medical cases and about 20 % of all neurological cases.

Stroke is defined clinically and diagnosis is supported by investigations like imaging modalities. There is a wide variation in the clinical presentation of stroke. This is because of the fact that our brain has complex anatomy and vasculature. Age is the predominant risk factor for stroke, because increasing age is associated with increased occurrence of stroke.

Three major types of stroke are now recognized. These are

- Ischemic stroke
- Hemorrhagic stroke
- Lacunar stroke.

Ischemic variety is due to atherothrombosis or brain embolism to cerebral vessels.

Hemorrhagic stroke with bleeding within the nervous tissue occurs due to ruptured cerebral aneurysms in the young and hypertensive

intracerebral bleeding in the elderly. Lacunar strokes are due to small cerebral infarcts in the basal ganglia or deep white matter due to diseases of small vessels called penetrating vessels.

Numerous literature are replete with valuable information indicating increased prevalence of stroke in diabetic as well as non diagnosed diabetic and patients with impaired glucose tolerance. Many studies have demonstrated evidence for increased severity of stroke associated with admission day blood glucose more than 110 mg %[1]. Reason for this observation could be micro as well as macro vascular complications associated with hyperglycemia.

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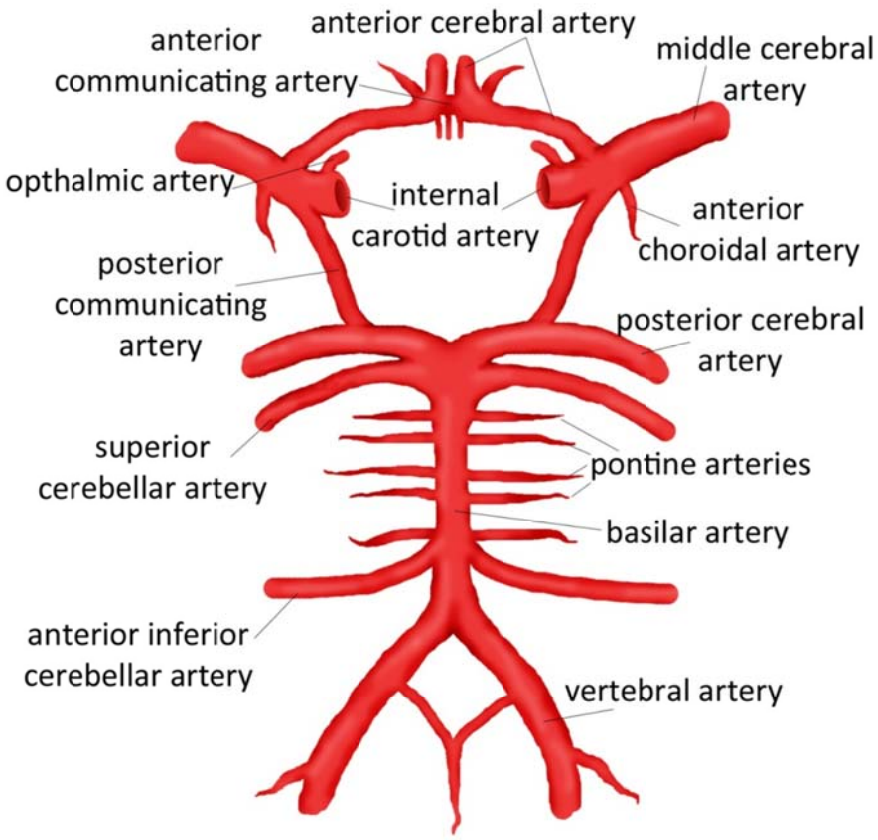


2.AIM OF STUDY

To measure the blood glucose levels within twenty four hours of the onset of stroke in both diabetics and in non diabetics and to evaluate the clinical outcome of stroke in relation to admission day glycemic status.

3.REVIEW OF LITERATURE

DEFINITION

Stroke

Defined as rapid onset of focal neurologic deficit resulting from diseases of the cerebral vasculature and its contents, the neurological signs and symptoms lasting for more than 24 hours[1].

Transient ischemic attack

Implies complete recovery of such a deficit within 24 hours

Reversible ischemic neurological deficit[2]

Implies neurological deficit resolves within 7 days

RISK FACTORS[2]

MAJOR

- Hypertension
- Smoking
- Diabetes mellitus
- Obesity
- Hyperlipidaemia
- Polycythemia
- High plasma fibrinogen
- Cardiac lesion
    - Atrial fibrillation

- Mitral valve prolapse
- Rheumatic heart disease
- Ischemic heart disease
- Infective endocarditis
- Dilated cardiomyopathy

- Thrombocythemia
- IncreasingAge
- TIA
- Angina
- Claudication
- Hypercoagulable disorders

MINOR

- High alcohol intake(Binge drinking)
- Positive family history of stroke
- Oral contraceptive pills
- Trauma

DIABETES MELLITUS

Diabetes mellitus increases the risk of cerebrovascular disease compared with the risk in people without diabetes. In addition, diabetes mellitus increases morbidity and mortality after stroke. Macro vascular disease and micro vascular disease are responsible for the complications among patients with diabetes mellitus[3]. The mechanisms of stroke

secondary to diabetes may be caused by atherosclerosis or embolism from cardiac source. The excess stroke risk in diabetes is not dependent on age and other factors like blood pressure levels.

HYPERGLYCEMIA AND STROKE

Numerous literatures show evidence for a detrimental association between blood glucose levels and grave outcome of stroke patients. Hyperglycemia aggravates cerebral edema because it affects the integrity of blood brain barrier[4]. It elevates the levels of neurotoxic substance like glutamate[4]. Numerous studies throw light about the neuroprotective role of insulin.[10] Furthermore chances for hemorrhage transformation are more with increased blood glucose levels.

MECHANISM OF INJURY BY HYPERGLYCEMIA

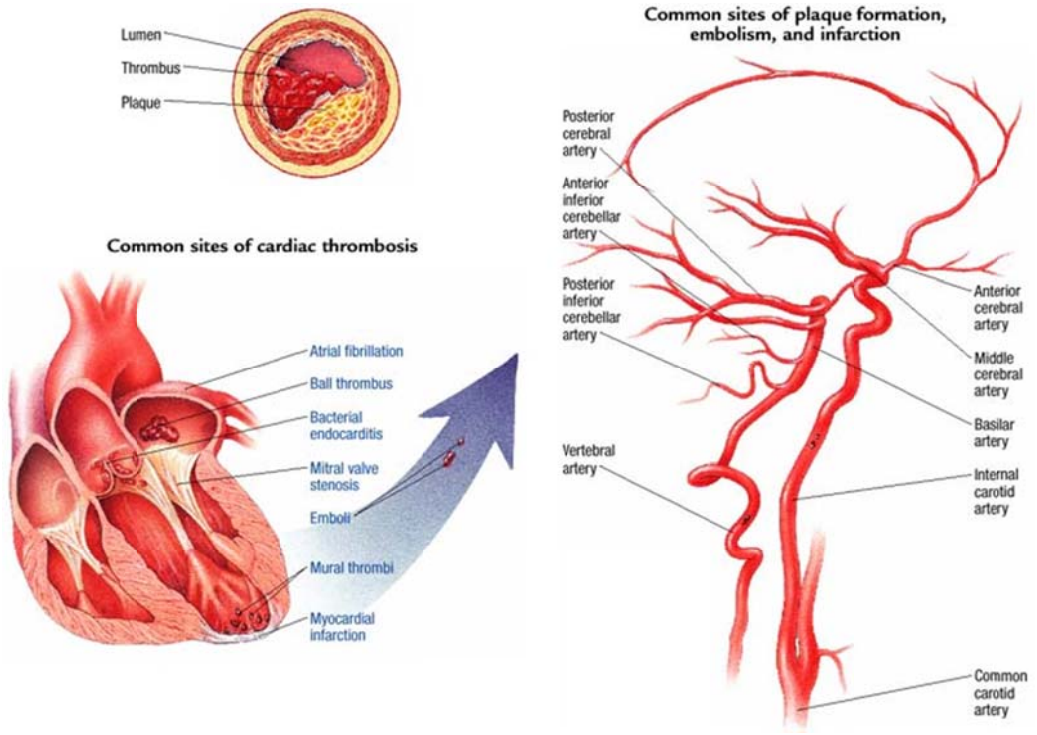
Hyperglycemia stimulates the production of lactic acid[5, 6, 7] by the mechanism of anaerobiosis. This leads to the severe damage of penumbra as evidenced by advanced MRI techniques. Hyperglycemia also affects mitochondrial function and also generates free radicals.[8]

PATHOPHYSIOLOGY OF STROKE

Cerebral infarction is usually attributed to partial or total occlusion of its regional microvasculature by thromboembolism. Cerebral atheroma is the most common underlying intimal vascular pathology whereas thrombosis with arteritis (tuberculosis, syphilis, rheumatic) is not an uncommon cause for stroke in the young in India. Studies indicate that

infarction is composed of dense central core, surrounded by a less dense zone of ischemia called as “penumbra”.[2,3] Neuronal death occurs in this central zone. On the other hand, cells in the zone of penumbra remain viable for three hours and can be salvaged by neuroprotective agents and reperfusion therapy. Due to brain hypoxia, there is energy depletion leading to loss of ATP. This causes delay in the resynthesis of macromolecular proteins essential for endothelial cell structure and function. Energy failure leads into proteolysis and lipolysis causing free radical generation , and a vicious cycle of further neuronal damage.

CARDIAC THROMBOSIS AND EMBOLISM



CAUSES OF STROKE

MAJOR CAUSES

- Obesity
- Hypertension
- Smoking
- Diabetes mellitus
- Atrial fibrillation
- Heart disease
- Dyslipidemia
- Hyperfibrinogenaemia
- Alcohol
- Coagulopathies
- Contraceptive pill

HYPERCOAGULABLE DISORDERS

- Polycythemia
- Sickle cell disease
- Thrombotic thrombocytopenic purpura
- Paroxysmal nocturnal hemoglobinuria
- Lupus anticoagulant
- Protein C deficiency
- Protein S deficiency
- Antithrombin III deficiency

- Homocysteinaemia
- Leukemia

CARDIAC CAUSES

- Rheumatic valve disease
- Atrial fibrillation
- Prosthetic heart valves
- Myocardial infarction/ventricular aneurysm
- Atrial aneurysm
- Mitral valve prolapse
- Calcific aortic valve
- Cardiomyopathy
- Bacterial endocarditis
- Patent foramen ovale
- Mitral annular calcification
- Cardiac operation

RARE CAUSES

- Marfan syndrome
- Marantic endocarditis
- Meningovascular syphilis
- Moyamoya disease
- Mitochondrial cytopathy[MELAS]
- Arterial dissection
- Ehlers-Danlos syndrome
- Fibromuscular dysplasia
- Pseudoxanthoma elasticum
- Fabry's disease
- Scleroderma
- Collagen vascular disease
- Hanging/strangulation
- Drug abuse
- Cervical irradiation

CLINICAL CLASSIFICATION OF STROKE[3]

1.COMPLETED STROKE

Rapid in onset with persistent neurological deficit which does not progress beyond 96 hours.

2.EVOLVING STROKE

Gradual step wise onset of neurological deficit

3.TRANSIENT ISCHEMIC ATTACK

4.REVERSIBLE ISCHAEMIC NEUROLOGICAL DEFICIT

5.STUTTERING HEMIPLEGIA

Internal carotid lesions show repeated episodes of TIA followed by fully evolved stroke.

YOUNG STROKE[2]

Stroke occurring in persons below 40 years of age.

CLINICAL FEATURES

GENERAL EXAMINATION

General examination may show the following.

- Obesity
- Feeble or absent peripheral arterial pulsations
- Vascular bruits
- Unequal or raised blood pressure
- Postural hypotension
- Retinopathy

The Stroke may announce itself abruptly as a major catastrophic event due to accomplished infarction or completed stroke. When the stroke evolves in a step – wise manner, appearing in each limb in succession, it is called ‘thrombosis in evolution’. This stuttering or

intermittent progression is classical of atherothrombosis. The other clinical manifestation depends on the site of arterial occlusion. The specific neurovascular syndromes are described below.

INTERNAL CAROTID SYNDROME[2]

The cervical portion of the internal carotid artery near the carotid sinus is a common site for Athero-stenosis. Most of the 60 % of all thrombotic lesions are located here. These lesions can be asymptomatic due to the presence of extensive collateral circulation. A pathognomonic feature of carotid artery syndrome is the occurrence of ipsilateral monocular blindness, accompanied by contralateral hemiplegia or sensory deficit. But this classical feature is seen in 15 % to 20 % of the patients.

Clinical features of acute carotid artery occlusion are almost indistinguishable from those of middle cerebral syndrome. Clinical features are as follows.

- Ipsilateral monocular transient blindness.
- Feeble internal carotid pulsations
- Feeble superficial temporal artery pulsation
- Dilated pupil
- Fundus- poorly pulsating vessels on the side of lesion.
- Ocular or cervical bruit on ipsilateral side.
- Investigations of choice are carotid duplex, Doppler sonography& Angiography, which show extent and degree of stenosis.

Asymptomatic cervical bruit[2,3]

- Asymptomatic patients around the age of 55- 80 years may have carotid bruit in the neck in about 5 % of cases.
- It is difficult to correlate the presence of bruit to subsequent TIA or stroke in that territory unless it is haemodynamically significant.
- Antiplatelet therapy is preferred in these patients.
- The role of prophylactic endarterectomy in preventing future stroke has not been established by clinical trials.

MIDDLE CEREBRAL SYNDROME

AREAS SUPPLIED BY MCA

Cortical branches

Supply lateral surface of the cerebral hemisphere.

Penetrating branches

Supply putamen , globus pallidus, genu and posterior limb of the internal capsule. Lenticulo –striate branches is other name for penetrating vessels.

Clinical picture is as follows,

- Contralateral hemiplegia
- Hemianaesthesia
- Homonymous hemianopia
- Aphasia (if dominant hemisphere involved)

BROCAS APHASIA[1,2]

- Brocas area is the motor speech area (area44) . It is located in the posterior most portion of inferior third frontal convolution of the dominant hemisphere.
- It is important for fluency, rhythm of speech and for the maintenance of grammar and syntax.
- This aphasia is due to the blockage of superior division, features are contralateral hemiparesis with sensory deficit and expressive aphasia.(motor aphasia).
- The patient will be non-fluent, agrammatic, dysprosodic and may be mute.

WERNICKES APHASIA[2,3]

- Wernicke area is situated in dominant tempero – parietal – occipital region at the posterior part of area 22.
- Its role is comprehension of the received speech and in the selection of words to express ideas.
- Patients present with features like fluent speech, incomprehension, no repetition, alexia, agraphia , paraphasia .
- Site of lesion is the inferior division of MCA.
- Speech contains paraphasia , neologism and jargons.
- When lesion in temporal region, there is disturbance in words heard.

- When lesion is in parieto-occipitalregion , there is disturbance of words seen.

CONDUCTION APHASIA

- Paraphasia , difficulty in repetition and in reading aloud and with normal comprehension are the common features.
- Site of lesion is posterior branch of MCA.
- Associated features include contralateral hemihypesthesia, homonymous hemianopia and optokinetic nystagmus.
- Patient has impaired repetition. (inability of the patient to repeat words spoken by the examiner)

GLOBAL APHASIA[1]

- There are marked elements of both anterior (broca) and posterior (Wernicke s aphasia).
- Minimal speech/nonfluent
- Comprehension for spoken and written –poor
- Lesion site is internal carotid and middle cerebral arteries.
- Associated features include hemiplegia, hemianopia and hemi sensory loss.

ANTERIOR CHOROIDAL SYNDROME[3]

Areas supplied ,

- Supplies posterior limb of internal capsule, which carries the corticospinal and sensory fibres for the contralateral limb.

- This syndrome is characterized by dense sensory-motor hemiplegic syndrome.
- This syndrome represents a true capsular hemiplegia (dense hemiplegia , hemianaesthesia and homonymous hemianopia)

ANTERIOR CEREBRAL SYNDROME

Areas supplied by ACA

- Cortical branches supply medial superior surface of the frontal lobe and the parietal lobe up to the Para central lobule.
- Penetrating branches supply anterior limb of the internal capsule and part of the head of caudate nucleus.

Occlusion of A1 segment is usually well tolerated due to the presence of extensive collateral circulation.

An anterior cerebral artery occlusion proximal to the anterior communicating artery in subjects with a symmetrical circle of Willis is frequently asymptomatic.

Occlusion distal to the anterior communicating artery manifests itself by a sensorimotor paralysis of the opposite lower extremity with mild weakness of the opposite shoulder.[2]

Occlusion of unpaired anterior cerebral artery (supplying both the hemispheres) results in a cortical type of paraplegia, sphincter incontinence and a mental state in which the patient is alert but mute. This state is called as akinetic mutism.

- Aphasia and hemianopia are never seen.
- Occlusion of the Heubner's artery is frequently associated with ataxic tremors of the contralateral limbs called as frontal ataxia. Apraxia,idiomotor dyspraxia of the limbs and gait are also present.
- Atretic anterior communicating artery presents with stroke.
- Reason for occlusion of the anterior cerebral artery is usually embolism , source of which could be cardiac or from major arteries.
- There is evidence of literature highlighting occlusion caused by surgical prodedure like aneurysmal clip.

SIGNS AND SYMPTOMS	**STRUCTURES INVOLVED**
Paralysis of opposite face, arm and leg	Somatic motor area
Sensory impairment of same area	Somatic sensory area
Motor speech disorder	Broca's area
Homonymous hemianopia	Optic radiation deep to second temporal convolution.
Central aphasia, word deafness , anomia,alexia, agraphia	Central language area
Paralysis of conjugate gaze to the opposite side.	Frontal contraversive field.
Pure motor hemiplegia	Superior half of the internal capsule.

OCCLUSION OF A2 SEGMENT[2]

SIGNS AND SYMPTOMS	STRUCTURES INVOLVED
Paralysis of opposite foot and leg.	Motor leg area
Paresis of opposie arm	Involvement of arm area of cortex.
Cortical sensory loss over toes , foot and leg.	Sensory area for foot and leg.
Urinary incontinence	Superior frontal gyrus
Contralateral grasp and sucking reflexes	Medial surface of the posterior frontal lobe.
Abulia ,motor inaction	Superomedial lesion near subcallosum.
Impairment of gait and stance	Infero- medial frontal striatal region.

POSTERIOR CEREBRAL ARTERY[2,3]

PCA occlusion can be either in the central territory or in the peripheral territory.

PERIPHERAL TERRITORY (cortical branches)

SIGNS AND SYMPTOMS	STRUCTURE INVOLVED
Homonymous hemianopia with macular sparing	Calcarine cortex
B/L homonymous hemianopia , cortical blindness	Bilateral occipital lobe
Dyslexia without agraphia	Dominant calcarine lesion
Memory defect	Inferomedial position of temporal lobe bilaterally
Topographic disorientation	Nondominant calcarine gyrus
Simultagnosia	Dominant visual cortex

PCA CENTRAL TERRITORY[2]

SIGNS AND SYMPTOMS	STRUCTURES INVOLVED
Thalamic syndrome-all modalities of sensory loss, spontaneous pain	Ventral postero lateral nucleus of thalamus
Thalamo perforate syndrome-crossed cerebellar ataxia with ipsilateral 3rd nerve palsy	Dentatothalamic tract
Ipsilateral 3RD nerve palsy and contralateral hemiplegia	Cerebral peduncle and 3rd nerve
Paralysis of vertical eye movement, skew deviation	Supranuclear fibres to 3rd nerve , high midbrain tegmentum

VERTEBRAL ARTERY SYNDROMES

They are the chief arteries of medulla, supplying the following structures

- Lower three-fourths of the pyramid
- Medial lemniscus
- Lateral medullary region
- Inferior cerebellar peduncle

LATERAL MEDULLARY SYNDROME[1,2]

Due to occlusion of any of the following vessels.

- Vertebral artery
- Posterior inferior cerebellar artery

- Superior/inferior/ middle lateral medullary arteries.

CLINICAL FEATURES

Clinical features indicate lateral medullary infarction.

ON THE SIDE OF LESION

1. Pain, numbness and impaired sensation over half the face (descending tract and nucleus of fifth nerve).
2. Ataxia of limbs , falling to the side of lesion (cerebellum , olivocerebellar fibres, restiform body)[1,2]
3. Vertigo , nausea , vomiting , nystagmus , diplopia , oscillopsia (vestibular nuclei involvement)
4. Horner's syndrome (miosis, ptosis, anhydrosis due to involvement of the descending sympathetic tract)
5. Loss of taste (nucleus and tractus solitarius)
6. Dysphagia, hoarseness, vocal cord paralysis , diminished gag reflex (ninth and tenth nerves)
7. Hiccup
8. Numbness of ipsilateral arm , trunk or leg (cuneate and gracile nuclei)

ON THE OPPOSITE SIDE[1,2]

Impaired pain and thermal sense over half of the body, sometimes face (due to involvement of spinothalamic tract).

MEDIAL MEDULLARY SYNDROME

This syndrome is due to occlusion of vertebral artery or branch of vertebral or lower basilar artery.

CLINICAL FEATURES

ON THE SIDE OF LESION

Paralysis and atrophy of half of the tongue.(twelfth nerve).

ON THE OPPOSIDE SIDE

- Paralysis of arm and leg sparing face (pyramidal tract)
- Impaired tactile and proprioceptive sense over half the body (medial lemniscus)

A combination of medial and lateral medullary syndrome can occur as a result of occlusion of vertebral artery.

BASILAR ARTERY SYNDROMES[3]

Complete basilar artery syndrome:

Features are bilateral long tract signs with variable cerebellar, cranial nerve and other segmental abnormalities of the brain stem. Occlusion of branches may result in various combinations of symptoms and signs. Features include somnolence,visual hallucinations , memory deficit ,disorders of eye movements , skew deviation of the eyes, confusional state and visual defects.

LOCKED IN SYNDROME[3]

- Quadriplegia with bilateral conjugate , lateral gaze palsy and mute state with fully preserved consciousness.
- Due to infarction of the basis pontis sparing the tegmentum from a mid basilar occlusion.

OCCLUSION OF THE SUPERIOR CEREBELLAR ARTERY

- Ipsilateral cerebellar ataxia
- Loss of pain and temperature over the opposite side of the body due to the involvement of the spinothalamic tract.
- Partial deafness
- Ipsilateral Horner's Syndrome
- Palatal myoclonus
- Nausea , vomiting , slurred speech, static tremor of the ipsilateral upper extremity

OCCLUSION OF THE ANTERIOR INFERIOR CEREBELLAR ARTERY

Features are vertigo , nausea , vomiting , nystagmus , tinnitus , deafness, facial weakness, ipsilateral cerebellar ataxia, ipsilateral Horner's syndrome , paresis of lateral conjugate gaze , contralateral loss of pain and temperature with or without hemiplegia.

AORTIC ARCH SYNDROME

It is characterized by diminution or absence of the arterial pulsations in the vessels of the arms and neck.

Etiology

- Atheromatosis
- Congenital anomalies
- Trauma with or without aneurysm
- Chronic dissecting aneurysm
- Mediastinal tumours
- Thrombophilia
- Syphilitic aortitis

TRANSIENT ISCHEMIC ATTACKS[2,3](TIA syndrome)

Approximately 80% of ischemic strokes occur in the carotid (or anterior) circulation, and 20% occur in the vertebrobasilar (or posterior) circulation.

During a follow up of nearly five years, 30 % of TIA patients develop a full fledged stroke.

Many theories are proposed for TIA , out of which embolic hypothesis and hemodynamic crisis are gaining support.

Repetitive (5- 10 attacks / day) short lived (15 min) typical spells of arm and hand weakness suggest proximal arterial narrowing with poor collateral circulation.

On the other hand, a single spell of speech difficulty with or without arm involvement lasting for about 12 hours suggests embolic ischemic attack with some degree of infarction.

The specific type of TIA points to the particular artery involved

TYPES OF TIA[2,3]

1.LOW FLOW TIA

Brief, recurrent and stereotyped. Due to atherosclerotic lesion at internal carotid artery or MCA stem or junction of vertebral and basilar artery.

2.EMBOLIC TIA

Discrete , usually single and more prolonged. If lasting for more than 24 hours, it indicates infarction has already occurred.

3.LACUNAR TIA

Occlusion of small vessels due to lipohyalinosis.

4. CRESCENDO TIA

TIA occurring in increased number and frequency and having a high likelihood of evolving into stroke.

BLOOD SUPPLY OF SPECIFIC BRAIN AREAS[2]

THALAMUS

Supplied by posterior communicating artery , basilar and posterior cerebral artery.

MIDBRAIN

Supplied by posterior cerebral, superior cerebellar and basilar artery.

MEDULLA OBLONGATA

Supplied by vertebral artery, anterior and posterior spinal artery , posterior inferior cerebellar and basilar artery.

CEREBELLUM

Supplied by superior cerebellar , anterior inferior cerebellar and posterior inferior cerebellar artery.

CORPUS STRIATUM

Supplied by medial and lateral central branches of MCA

BLOOD SUPPLY OF INTERNAL CAPSULE

Divided into anterior and posterior limb.

ANTERIOR LIMB

- Upper Half- MCA
- Lower Half-ACA

POSTERIOR LIMB

- Upper Half-MCA
- Lower Half-Anterior choroidal and posterior communicating artery.

EYE POSITION IN HEMIPLEGIA[3]

1. Hemisphere lesion-eye deviated towards side of lesion.
2. Pontine lesion-eyes deviated to paralysed side.
3. Putamen lesion-loss of conjugate lateral gaze.
4. Thalamic lesion-loss of upward gaze, skew deviation, unequal pupil, lateral gaze palsy.

LOCALISATION OF THE SITE OF LESION[3]

NO	SITE OF LESION	LOCALISING CLINICAL FEATURES
1.	Cortex	Aphasia ,bladder involvement ,cortical sensory loss, denial, epilepsy Flaccid mono or hemiplegia
2.	Internal capsule	Hemiplegia , hemianaesthesia, hemianopia, marked spasticity
3.	Thalamus	Fleeting hemiparesis in the side opposite to lesion. Impairment of Superficial and loss of deep sensation in the opposite side. Spontaneous pains and hyperpathia
4.	Midbrain	Weber's syndrome-3rd nerve palsy and contralateral hemiplegia Benedikt's syndrome-3rd nerve palsy, contralateral cerebellar or rubral tremor and contralateral hemiplegia.

5.	Pons	Millard-Gubler syndrome-Ipsilateral facial and gaze palsy, contralateral hemiplegia. Foville's syndrome-Ipsilateral6th ,7th nerve palsy and contralateral hemiplegia.
6.	Medulla	Medial medullary syndrome Lateral medullary syndrome
7.	Spinal cord	Same side hemiplegia

STROKE

CLINICAL FEATURES	EMBOLISM	THROMBOSIS	HAEMORRHAGE
Age	Younger	Middle or old	Middle or old
Mode of onset	Acute	Insidious	Acute
Time of onset	Often during day	Often during sleep	During waking hours
Convulsions	Present	Absent	Present
Hypertension	Absent	Absent	Present
Cardiac lesion	Present	Absent	Absent
Recovery	Rapid recovery	Gradual	delayed

DIFFERENTIAL DIAGNOSIS

1. Hypoglycemia
2. Post-epileptic hemiplegia
3. Subdural hematoma

4. Head injury
5. Hysteria
6. Posterior fossa tumours

DIAGNOSTIC EVALUATION OF STROKE[11,12]

IMAGING:

CT SCAN

- Computerised cranial tomography is the non-invasive investigation of choice to distinguish an ischemic infarction and cerebral hematoma from other conditions simulating stroke like subdural hematoma and tumour.
- It clearly shows the extent and location of supratentorial cerebral infarction
- It can detect small lacunar infarct of up to 0.5 cm.
- CT scan clearly highlights surrounding edema and haemorrhagic infarction.

DRAWBACKS OF CT

- Does not differentiate early ischemic lesion from normal tissue, meaning CT taken within 1 to 3 hours of stroke onset.
- Brainstem lacunae are difficult to detect by conventional CT scan.

MRI BRAIN[3]

- Magnetic Resonance Imaging { T2 weighted, diffusion and perfusion weighted studies ,MRA} of brain will define arterial and cerebral lesions even in hyper acute stage, which are not visualized on routine CT.
- MRI/MRA helps in taking therapeutic decisions like thrombolytic therapy.
- T1- haemorrhage appears hyperintense.
- T2-haemorrhage has mixed intensity.

OTHER INVESTIGATIONS

- Complete haemogram
- Routine baseline investigations, ECG, x-ray chest

CAROTID DOPPLER

Special investigations

- C-reactive protein
- Lupus anticoagulant
- Homocystein level
- VDRL
- HIV
- Anti-nuclear antibodies
- Anti-DS DNA

CT IN ISCHAEMIC STROKE[13]

STAGES OF INFARCT	CT FINDINGS
< 12 hours	Normal in 50 % Hyperdense artery Obscuration of lentiform nuclei
12 – 24 hours	Insular ribbon sign Sulcal effacement
1-7 days	Mass effect , wedge shaped low density area
1-8 weeks	Contrast enhancement Mass effect resolves
Months to years	Encephalomalacic change

MRI IN CEREBRAL INFARCTION[13]

STAGES OF INFARCTION	MRI FINDINGS
Immediate	Alteration of perfusion/diffusion coefficient
<12 hours	Sulcal effacement
12-24 hours	Leptomeningeal enhancement Mass effect
1 to 3 days	Early parenchymal contrast enhancement
4 to 7 days	Parenchymal enhancement,Haemorrhage (25%)
1 to 8 weeks	Mass effect resolves, haemorrhage signal evolves
Months to years	Encephalomalacic changes

HYPERGLYCEMIA AND STROKE OUTCOME

Mortality and morbidity from stroke is increased in patients admitted with hyperglycaemia. There are multiple mechanism by which hyperglycaemia influences stroke outcome.

Detrimental effects of hyperglycemia have been studied in multiple human and animal studies. Hyperglycemia occurs in stroke either as a manifestation of preexisting undiagnosed diabetes mellitus or as a result of sympathetic response of the body due to the release of cortisol and noradrenaline.

1. Vascular occlusion causes hypoxic atmosphere in the neurons. As a result, glucose is converted to lactic acid by anaerobic metabolism. Glucose is delivered more to the ischemic zone due to damage of blood brain barrier. Acidosis causes damage to neurons ,neuroglial tissue and vascular areas.[14,15,16]
2. Hyperglycemia stimulates the release of excitatory neurotransmitters like glutamate and aspartate. These neurotransmitters are neurotoxic in nature. They cause hyper stimulation of postsynaptic nerve terminals, resulting ultimately in neuronal death.[17,19]
3. Numerous studies have clearly indicated that hyperglycemia increases calcium levels in the neurons, causing cell injury and death.[20,21] We are already aware of the fact that nimodipine , a calcium channel blocker has definite role in reducing cerebral acidosis.

4. Hyperglycemia stimulates the formation of advanced glycation end products. These products interfere with the function of various enzymes. Also these products result in toxic damage to the endothelial cells. Thus vascular injury is aggravated.[22,23,24]
5. A negative impact on the stroke outcome by hyperglycemia is explained by the fact that poor reperfusion occurs due to the vascular injury.[25]
6. Another disastrous effect is the loss of vascular tone due to the oxidation of the nitric oxide dependent mechanisms.[26,27,28]
7. Martini and Kent state that even when a blocked vessel is recanalized promptly, effective reperfusion of ischemic brain tissue is not obtained.[29,30]
8. Function of mitochondria is severely impaired in the zone of ischemic penumbra. As a result , size of the infarct progresses.[31,32,33]
9. Another crucial factor is increased lipolysis due to insulin deficient state, resulting in free fatty acid production. As a result , ischemic brain tissue is further damaged.[34,35,36]

Numerous studies have shown that admission day glucose level directly correlates with the occurrence of cerebral edema and its known deadly complications causing clinical deterioration of the patient. Blood glucose level is one of the determinant of infarct progression.

TREATMENT OF ISCHEMIC STROKE[2,3,18]

GOAL OF THERAPY

1. Avoid the development of cerebral infarction.
2. If already developed, then the goal is to reduce its progression and recurrence.

MANAGEMENT OUTLINE

1. Medical management
2. Antiplatelet drugs
3. Anticoagulants
4. Thrombolytic therapy

- Intravenous
- Intra-arterial

5. Neuroprotective agents
6. Surgical management

Treatment is divided into three phases.

PHASE 1

Saving life and speedy recovery

PHASE 2

Rehabilitation to achieve adaptation to gainful employment.

PHASE 3

Prevention of recurrence of stroke.

PHASE 1

GENERAL MEASURES

- Maintenance of vital signs
- Airway patency
- Fluid and electrolyte balance
- Prevention of complications like pulmonary aspiration, bedsores.

BLOOD PRESSURE CONTROL[3]

Due to loss of cerebral auto regulation, perfusion in the ischemic zone is solely dependent on mean arterial BP. In the presence of severe hypertension (BP-220/120mmhg), intravenous agents like labetalol or enalapril which reduce the BP smoothly are preferred. Calcium channel blockers are avoided because they produce marked fall in BP.

FLUID/ELECTROLYTE AND BLOOD GLUCOSE

Hyperglycemia or hypoglycaemia should be treated appropriately. Hyperglycemia adversely influences stroke outcome. Hence according to American Diabetes Association, strict glycemic control with insulin therapy is preferred.

IV 5% dextrose is best avoided in early stages of infarction with edema.

Judicious restriction of fluid intake is advocated in first 48 hours.

REDUCTION OF INTRACRANIAL PRESSURE AND CEREBRAL EDEMA[1,2]

- Head elevation by 30 degree.
- If associated with increasing drowsiness, intubation and hyperventilation to maintain pCO_2 around 25-30 mmhg would be helpful.
- Hyperosmolar solution like intravenous mannitol will reduce vasogenic brain edema. Its contraindications are renal failure and cardiac failure.
- IV 10% glycerol has role in reducing edema in patients with incipient left ventricular failure.
- Restriction of fluids and furosemide induced diuresis is also effective in reducing edema.
- High dose of corticosteroids reduces cerebral edema but its role in the management of vasogenic edema is doubtful.
- Careful reduction of BP in situations of hypertensive crisis using parenteral antihypertensive is greatly helpful.

HAEMODILUTION THERAPY

Haematocrit is the chief determinant of whole blood viscosity.

Lowering of haematocrit to 30% - 33 % improves cerebral blood flow and oxygenation of infracted tissues. But recent trials showed no consistent benefit of haemodilution therapy.

SPECIFIC THERAPY

ANTIPLATELET DRUGS[4]

- ASPIRIN
- TICLOPIDINE
- CLOPIDOGREL
- GlycoproteinIIb/III a inhibitors

  Abciximab 0.25mg/kg IV bolus

Aspirin prevents platelet aggregation by blocking production of platelet derived thromboxane –A2. Its widely used in primary and secondary prevention of stroke.

Ticlopidine inhibits ADP-induced transformation of glycoprotein IIb/IIIa receptors on platelet membrane. Patients with raised creatinine levels benefit more with ticlopidine 250 mg bid. MATCH study revealed no real benefit in outcome of vascular end points with combination therapy of aspirin and clopidogrel.

ANTICOAGULANTS

INDICATIONS OF HEPARIN IN STROKE

- Cardio-embolic stroke
- Dissection of carotid and vertebral artery
- Hypercoagulable states like protein C deficiency / protein S deficiency.
- Cerebral venous thrombosis

- Recurrent TIA s
- Thrombosis in evolution

Dosage is 3000 to 5000 units given every 6 - 8thhourly.APTT is kept up to two times the control.

Among oral anti-coagulants, coumarin sodium 2-5 mg/day is generally well tolerated.

THROMBOLYTIC THERAPY

INDICATIONS

- When stroke is clinically suspected.
- Symptom duration < 3 hours
- Consent by patient
- CT SCAN revealing no haemorrhage or significant cerebral edema.
- Patient should have crossed the age of eighteen years.

DOSE OF rtPA-

Recombinant tissue plasminogen activator is administered as follows. Maximum dose is 90 mg. Total dose is 0.9 mg/kg IV. Total dose is calculated as per body weight. Dose for bolus injection is 10 % of the calculated dose. The remaining dose of 90 % is given intravenously as a continuous infusion for a estimated time of about one hour.

INTRA-ARTERIAL THROMBOLYTIC THERAPY

Latest advances using microcatheter techniques enables to inject thrombolytic agent directly over the clot surface, thereby giving more effective recanalization.

NEUROPROTECTIVE AGENTS

Clinical trials with multiple agents like calcium channel blocker nimodipine have not shown clear benefits in several vascular endpoints. Results of several trials awaited.

HOMOCYSTEINE[3]

Elevated levels of homocysteine affects endothelial cell function and also cause oxidative stress, ultimately leading to thrombosis.

Treatment with multivitamins like B6, B12 and folic acid helps in reducing homocysteine levels and thus reducing vascular complications.

STATINS

SPARCL study indicates that statins reduce the recurrence of cerebrovascular accident and the benefit obtained with statin is independent of its cholesterol lowering action.

SPARCL study-stroke prevention by aggressive reduction in cholesterol

SURGICAL MANAGEMENT-

Carotid artery endarterectomy has shown clear benefits in patients admitted with tight cervical stenosis defined as 70% to 99 %. In mild stenosis , the role of endarterectomy is controversial.

STENTING AND ANGIOPLASTY[2]

Stenting is associated with less frequent complications like hematoma in neck. So it is an effective alternative for patients not feasible for endarterectomy. Stenting along with embolic protection devices are accepted as alternative treatment.

STEM CELL THERAPY

Ethical issues prevent experimental trials in humans with stem cell therapy in ischemic stroke. Animal experiments need to be explored. Reliable reports are not available.

PHASE 2

NEUROREHABILITATION

Active physiotherapeutic measures should be commenced as soon as the patient shows signs of recovery. This is necessary to prevent the occurrence of joint contracture and also to promote recovery of strength and coordination.

PHASE 3

STROKE PREVENTION

Chances of recurrent stroke are high during the first few weeks and nearly 10% of cases occur during the first year. Prevention strategies are

- Control of smoking
- Stop smoking/tobacco use
- Antiplatelet agents.
- Anticoagulants
- Statins
- Glycemic control
- Correction of modifiable risk factors
- Regular physical exercise
- Maintenance of ideal body weight.

INTRACEREBRAL HAEMORRHAGE

It is the deadliest, most disabling and least treatable form of stroke. Following a haemorrhage, 35% to 52 % are dead within a month and less than 20% were living independently after 6 months. It constitutes 15% of all stroke in the west and 20% to 30% in western population. There is increasing literature revealing higher incidence of haemorrhagic stroke in north-eastern regions.

Most significant risk factors for haemorrhagic stroke are chronic uncontrolled hypertension and advancing age.

Haemorrhagic strokes occur in increased frequency in patients admitted with hyperglycemia irrespective of whether the patient is known diabetic or newly diagnosed diabetic or stress induced hyperglycemia.

CAUSES OF INTRACEREBRAL HAEMORRHAGE[4]

ABNORMAL BLOOD VESSELS

- Vascular malformations
- Arteriovenous malformations
- Saccular aneurysm
- Cavernous angioma
- Septic and mycotic aneurysm
- Lipohyalinosis
- Microaneurysm
- Amyloid angiopathy
- Cerebral tumour
- Cerebral venous thrombosis
- Vasculitis
- Moyamoya syndrome
- Haemorrhagic transformation

SYSTEMIC BLEEDING TENDENCY

- Haemophilia
- Leukaemia
- Thrombocytopenia

- Anticoagulants
- Antiplatelet agents
- Thrombolytic agents

ILLICIT DRUGS

- Amphetamines
- Cocaine

HYPERPERFUSION SYNDROME

TRAUMA

SITES

Most common sites of hypertensive haemorrhage are deep grey matter

- Putamen
- Globuspallidus
- thalamus

} above three constitute 65%

- Subcortical white matter-10 to 20 %
- Pons – 10 to 15 %
- Cerebellum- 8 to 10 %

CLINICAL FEATURES

- Signs and symptoms accompanying ICH reflect the location of haemorrhage.

- Lobar haemorrhages frequently produce contralateral weakness or sensory loss, language disturbance, hemianopia and parietal lobe signs.
- Majority of ICH cause complete stroke.
- Cerebellar haemorrhage are particularly important to identify clinically as they require surgical intervention which can be lifesaving.

INVESTIGATIONS

Plain CT scan of the brain is the mainstay of diagnosis of ICH. Blood appears hyper dense on noncontrast enhanced CT scans.

MRI Scan is also very sensitive for ICH. Following haemorrhage, a series of events occurs that can be detected using MRI. It is particularly useful to distinguish between previous cerebral infarction and haemorrhage at a time when the appearance on CT are identical.

TREATMENT

Control of blood pressure

- Head of bed elevation
- Osmotic therapy
- Hyperventilation
- Maintenance of euvolemia
- Prevention of seizures
- Management of body temperature.

- Strict glycemic control in the presence of hyperglycemia
- Surgery
- evacuation of hematoma, VP shunt

IMPACT OF HYPERGLYCEMIA ON HAEMORRHAGIC STROKE

- Hyperglycemia severely impairs blood brain barrier. As a result, it aggravates edema formation in surrounding area of haemorrhage.[9]
- Relative insulin deficiency further causes release of free fatty acids, which further aggravate neuronal damage.
- Multiple studies have demonstrated association between elevated blood glucose levels and hemorrhagic transformation of ischemic stroke.[37,38]
- But controversial results are also obtained in few studies. One such study is the Copenhagen stroke study. The result obtained was diabetics had lesser frequency of hemorrhagic stroke.[40]
- Demchuk et al stated that elevated blood glucose is a crucial predictor of intracerebral hemorrhage in a study conducted among 138 patients with ischemic stroke who received tissue plasminogen activator.[41]
- Hyperglycemia is a crucial determinant of outcome in supratentorial intracranial hemorrhage.

4. MATERIALS AND METHODS

Number of patients undertaken for my study are hundred and ten patients. All are admitted in the Department of Medicine, Government Tirunelveli medical college hospital, tirunelveli. Period of study was from November 2011 to October 2012. Both male and female are undertaken for study. Both ischemic and haemorrhagic stroke are studied. Criteria for selecting patients were formulated and are as follows.

CRITERIA FOR SELECTION

- Age> 40 years
- Admission to our hospital within 24 hours of onset of sroke
- Measurementof blood glucose levels within 24 hours of onset of cerebrovascular accident.
- First episode of stroke

CRITERIA FOR REJECTION

- Age< 40 years
- Admission after 24 hours of onset of cerebrovascular accident.
- Measurement of blood glucose levels after 24 hours of symptoms onset.
- Recurrent stroke
- Intravenous glucose administration before sample collection.

Among the hundred and ten patients, follow up of ten patients could not be done. So for my study remaining hundred patients were selected.

Detailed complete neurological history taken and from the presentation of manifestations, clinical diagnosis was made out.

Vital signs measurement done in all patients.

For all admitted patients, routine basic investigations done like complete blood count, blood glucose, renal function test,.

Xray chest and ECG done.

Stroke severity is assessed on each patient considering the clinical presentation into account and each finding is awarded specific points.

NIH STROKE SCALE

1a. Level of Consciousness:

0 = Alert

1 = drowsy

2 = stupurous

3 = comatose

1b. LOC Questions:

0 = Answers both correctly.

1 = Answers one correctly.

2 = Incorrect

1c. LOC Commands:

0 = Obeys both correctly.

1 = Obeys one correctly.

2 = Incorrect

2. Best Gaze:

0 = Normal.

1 = partial palsy

2= forced deviation

3. Visual:

0 = No loss of vision

1 = Partial hemianopia.

2 = Complete hemianopia.

3 = Bilateral hemianopia

4. Facial Palsy

0 = Normal symmetric

1 = Minor paralysis

2 =Partial paralysis

3 = Complete paralysis

5. Best Motor Arm/Leg(right/ left):

0 = No drift

1 = Drift

2 = some antigravity effect

3 = No antigravity effect

4 = No movement.

6. Limb Ataxia: =

0 = Absent.

1 = Present in one limb.

2 = Present in both limbs.

7.Sensory:

0 = no sensory loss.

1 = Mild-to-moderate sensory loss

2 = total sensory loss

8. Best Language:

0 = No aphasia

1 = Mild to moderate aphasia

2 = Severe aphasia

3 = Mute

9. Dysarthria:

0 = Normal.

1 = Mild to moderate

2 = Severe

11. Extinction and Inattention

0 = No abnormality.

1 = Visual/ tactile/ spatial/ personal inattention

2 = Profound hemi-inattention.

From the above scale, points are added for each patient, assessing the severity of involvement. Maximum points that can be awarded for each patient are thirty points.

PLAN OF STUDY

- Blood glucose is estimated within 24 hours of onset of stroke.
- All the patients are classified into **4 broad groups** based on the following three parameters.

- Admission Day Blood glucose
- HBA1C level
- History of diabetes mellitus

		CATEGORY
1.	Blood glucose < 110 mg/dl	EUGLYCEMIC
2.	History of Diabetes Mellitus	KNOWN DIABETIC
3.	Blood glucose > 110mg/dl No History of diabetes HBA1C > 6.5%	NEWLY DIAGNOSED DIABETIC
4.	Blood glucose > 110 mg/dl No History of diabetes HBA1C < 6.5 %	STRESS HYPERGLYCEMIC

HBA1C normal range is 3.8% to 6.4 %. Only for those patients, presenting with blood glucose levels more than 110 mg/dl and also with no history of diabetes mellitus , HBA1C level is measured.

Glucose attaches to haemoglobin in an irreversible fashion throughout its life span. At any given point of time, a sample represents a collection of new-born, middle age and senescent RBCs. Hence glycohaemoglobin level obtained represents a glucose level that is reflective of the glucose environment confronting red cells over the previous 3 months period.

HBA1=HBA1 (a,b,c). HBA1C is preferred because glycosylation is with glucose and not with other sugars.

HBA1C of 6% corresponds approximately to mean plasma glucose of 120 mg. For every 1 % rise in HBA1C, mean blood glucose rises by 30 mg. Haemolyticanemia , Haemoglobinopathies and Uraemia may interfere with the estimation of glycated haemoglobin.

CT brain is done in all patients. In addition to making a diagnosis of stroke, size of lesion can be made out.

SIZE OF LESION

SMALL - 5 mm

MEDIUM - 5-10 mm

LARGE - > 10 mm

FOLLOW UP

All the patients under study are followed up for thirty days, progression of disease assessed. 10 patients are dropped as routine follow up could not be done. Outcome of remaining 100 patients is classified into 4 categories.

OUTCOME CATEGORY

GOOD	Improvement in symptoms along with independency in carrying out day to day activities, Motor function and Aphasia with no persistent disability
POOR	Not able to carry out any form of work with persistent disability and dependent on others for day to day activity, Stable deficit with no evidence of recovery
Moderate	Patients not fitting into above two categories are classified as moderate.
DEATH	

5. OBSERVATIONS AND RESULTS

STATISTICAL TOOLS

The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using **Epidemiological Information Package (EPI 2010)** developed by Centre for Disease Control, Atlanta.

Using this software range, frequencies, percentages, means, standard deviations, chi square and 'p' values were calculated. Kruskul Wallis chi-square test was used to test the significance of difference between quantitative variables and Yate's chi square test for qualitative variables. A 'p' value less than 0.05 is taken to denote significant relationship.

RESULTS

PROFILE OF CASES STUDIED

Age distribution

Age group	Cases	
	No	%
40-49 years	18	18
50-59 years	23	23
60-69 years	39	39
70 & above	20	20
Total	100	100
Range Mean SD	40 - 88 years 60.1 years 10.5 years	

Sex distribution

Sex	Cases	
	No	%
Male	68	68
Female	32	32
Total	100	100

RISK FACTORS

Risk factors	Male	Female	Total
Hypertension	24	16	40
Diabetes	13	11	24
Dyslipidemia	15	14	29
Atrial fibrillation	1	1	2
Coronaryartery disease	23	3	26

Personal habits among males

Personal habits among males(68)	Cases	
	No	%
Smoking	4	5.9
Alcoholic	20	29.4
Smoker/ Alcoholic	16	23.5
Nil	28	41.2
Total	68	100

CT findings

CT findings	Cases	
	No	%
Hemorrhage	21	21
Infarct	79	79
Total	100	100

Admission day blood glucose

Admission day blood sugar	Cases	
	No	%
< 110 mg	31	31
110-125 mg	7	7
126-199 mg	49	49
> 199 mg	13	13
Total	100	100
Range	72 - 334	
Mean	147.4	
SD	53.5	

NIHSS / HbAIC

Parameter	NIHSS	HbAIC
Range	6-26	6-10.4
Mean	12.9	6.8
SD	6.0	1.2

PROGNOSIS

Prognosis	Group					
	Hemorrhage Group		Infarct group		Total case	
	No	%	No	%	No	%
Good	3	14.3	21	26.6	24	24
Moderate	4	19	14	17.7	18	18
Poor	7	33.3	28	35.4	35	35
Death	7	33.3	16	20.3	23	23
Total	21	100	79	100	100	100

GLYCEMIC STATUS

Glycemic status	Total
Euglycemia	31
Stress hyperglycemia	26
Known diabetes	24
Newly detected diabetes	19

GLYCEMIC STATUS AND STROKE SEVERITY

Glycemic status	NIHSS
Euglycemia	7.1
Stress hyperglycemia	17.2
Known diabetes	18.3
Newly detected diabetes	16.5

SIZE OF LESION IN STROKE

Glycemic status	Small	Medium	Large	Total
Euglycemia	19	6	6	31
Stress hyperglycemia	2	11	13	26
Known diabetes	1	7	16	24
Newly detected diabetes	1	6	12	19

X^2 =41.5 p=0.001

NIHSS and clinical outcome

Clinical outcome	NIHSS values					
	Hemorrhage Group		Infarct group		Total cases	
	Mean	S.D.	Mean	S.D.	Mean	S.D.
Good	7.0	-	6.9	0.5	6.9	0.4
Moderate	12.5	2.9	10.1	2.6	10.6	2.7
Poor	22.1	4.0	15.1	3.7	16.5	4.7
Death	22.4	5.6	12.5	5.7	15.5	7.2
'p'	0.0054 Significant		0.0001 Significant		0.0001 Significant	

HbAIC and clinical outcome

Clinical outcome	HbAIC values					
	Hemorrhage Group		Infarct group		Total cases	
	Mean	S.D.	Mean	S.D.	Mean	S.D.
Good	-	-	6.1	-	6.1	-
Moderate	8.2	3.1	6.7	1.6	7.1	1.9
Poor	6.9	1.3	6.6	0.6	6.7	0.8
Death	7.4	2.4	6.6	0.8	6.8	1.3
'p'	0.8204 Not significant		0.3857 Not significant		0.4589 Not significant	

ADMISSION DAY GLUCOSE LEVELS AND CLINICAL OUTCOME

Clinical outcome	Admission day glucose values					
	Hemorrhage Group		**Infarct group**		**Total cases**	
	Mean	**S.D.**	**Mean**	**S.D.**	**Mean**	**S.D.**
Good	88	6.1	94.0	20.6	93.3	19.4
Moderate	159.3	43.4	134.6	40.7	140.1	41.3
Poor	198.7	70.5	167.6	39.8	173.8	47.9
Death	173.9	47.2	167.6	56.1	169.5	52.5
'p'	**0.0403 Significant**		**0.0001 Significant**		**0.0001 Significant**	

GLYCEMIC STATUS AND CLINICAL OUTCOME IN HAEMORRHAGE GROUP

BLOOD SUGAR	Clinical outcome in Hemorrhage group				
	GOOD	MODERATE	POOR	DEATH	TOTAL
Euglycemia	3	1	-	1	5
Stress hyperglycemia	-	1	3	2	6
Known diabetes mellitus	-	1	2	2	5
Newly detected diabetes	-	1	2	2	5
Total	3	4	7	7	21

GLYCEMIC STATUS AND CLINICAL OUTCOME IN INFARCT GROUP

BLOOD SUGAR	Clinical outcome in Infarct group				
	GOOD	MODERATE	POOR	DEATH	TOTAL
Euglycemia	19	4	2	1	26
Stress hyperglycemia	1	5	8	6	20
Known diabetes mellitus	1	4	9	5	19
Newly detected diabetes	-	1	9	4	14
Total	21	14	28	16	79

GRADE OF BLOOD SUGAR AND CLINICAL OUTCOME IN HAEMORRHAGE GROUP

BLOOD SUGAR LEVEL	Clinical outcome in Hemorrhage group				
	GOOD	MODERATE	POOR	DEATH	TOTAL
< 110 mgs	3	1	-	1	5
110 – 125 mgs	-	-	-	-	-
126 – 199 mgs	-	3	5	4	12
>199 mgs	-	-	2	2	4
Total	3	4	7	7	21

GRADE OF BLOOD SUGAR AND CLINICAL OUTCOME IN INFARCT GROUP

BLOOD SUGAR	Clinical outcome in Infarct group				
	GOOD	MODERATE	POOR	DEATH	TOTAL
< 110 mgs	19	4	2	1	26
110 – 125 mgs	1	1	4	1	7
126 – 199 mgs	1	8	16	12	37
>199 mgs	-	1	6	2	9
Total	21	14	28	16	79

6. DISCUSSION

AGE, SEXAND RISK FACTORS

Among our study of 100 patients, majority of them belonged to male sex showing a male preponderance which is commonly seen in most studies. Majority of the patients, 39 were between the age group of 60 to 69 years. In our study of 100 patients, 40 patients had hypertension, 24 patients had diabetes mellitus, 29 had evidence of dyslipidemia and 2 had atrial fibrillation. Nearly half of patients had smoking as a risk factor and one third had history of alcohol intake.

GLYCEMIC STATUS

In our study of 100 patients, 69 patients had elevated admission day blood glucose levels and 31 patients had normal blood glucose values. Diabetes was noticed in 24 patients and stress hyperglycemianoted in 26 patients. Newly diagnosed diabetic patients contributed to a number of 19. In ischemic stroke, stress hyperglycemia contributed to one third of patients and in haemorrhagic stroke also, stress hyperglycemia contributed to one third of patients.

SEVERITY OF STROKE

Severity of stroke was assessed with stroke scale devised by national institute of health. Patients with elevated admission day blood glucose levels had a higher score when compared to euglycemic patients which was statistically significant with p value of 0.001. This clearly

shows that patients with elevated admission day blood glucose levels at the time of stroke had severe stroke.

SIZE OF STROKE

All patients were subjected to CT brain and size of the lesion was analyzed. Results are, most of the euglycemic patients had small sized infarcts and hemorrhagic lesions, whereas majority of the admission day hyperglycemic patients had large sized lesion with edema and midline shift. These data are statistically significant with p value of 0.001. As already discussed, hyperglycemia by means of multiple mechanisms like altered mitochondrial function, increased free radical production, causes toxic injury to brain and this is responsible for large sized infarcts and hemorrhages observed in patients admitted with elevated admission day blood glucose levels.

TYPE OF STROKE

In our study of 100 patients, seventy nine patients had ischemic stroke and 21 patients had hemorrhagic stroke. Among the euglycemic group, predominant patients had ischemic stroke and one fourth of patients had hemorrhagic stroke. Among patients with stress hyperglycemia, twenty patients had infarct whereas 6 patients had hemorrhage. On observing diabetes mellitus patients, nineteen patients had infarct and the remaining patients had hemorrhage. In patients with

newly diagnosed diabetes, five persons presented with hemorrhage and fourteen presented with infarct.

OUTCOME OF STROKE

- In this study of hundred acute stroke patients, euglycemic patients had a better outcome when compared to admission day hyperglycemic patients.
- Patients with normal blood glucose levels had a better recovery after acute stroke at the end of thirty day follow up. On the contrary, patients with admission day hyperglycemia are associated with either slower recovery or poorer recovery.
- Admission day blood glucose levels have a direct correlation with severity of stroke. Among patients with good outcome in hemorrhage group, the mean blood glucose level is 88 mg, likewise for infarct group with good outcome, mean value for glucose is 94 mg.
- For patients with poor outcome, mean blood sugar value in patients with hemorrhagic stroke was 198mg , whereas mean value in ischemic patients was 167 mg.
- Regarding mortality , death is associated with mean admission day blood glucose level of 173 mg in the hemorrhage group and the mean value for infarct group is 167 mg. These data were statistically significant with p value of 0.0403 for hemorrhagic stroke and p value for ischemic stroke is 0.0001 which is significant.

- This study of hundred acute stroke patients shows that admission day elevated blood glucose levels was associated with a high early mortality rate and an increased risk of poor functional recovery.
- 70 % of patients with euglycemia had good outcome with speedy recovery in both types of stroke. On the other side, only 3.8 % of stress hyperglycemia had good outcome. Among 31 patients of euglycemia, 6.45 % of patients showed worse outcome. On the contrary, 42 % of patients with stress hyperglycemia showed poor outcome.
- Among diabetes mellitus patients (both known diabetic and newly diagnosed diabetics) , only 2.32 % of patients had good outcome. But poor outcome was seen in as many as 51.16 % of patients with diabetes. Mortality rate is also high evidenced by death in 30.23 % of patients. These data are statistically significant with p value of 0.001.
- There is straight correlation between blood sugar levels and outcome of cerebrovascular accident. This is evidenced by the fact revealing that 60 % of patients with poor outcome had blood sugar values between 126- 199 mgs. On comparison, only 4 % of patients with good outcome had blood sugar value in the range of 126-199 mg%. 97 % of patients with good outcome had blood glucose levels in the range of < 110 mgs.

- Our study clearly shows a positive correlation between admission day sugar value and the outcome of cerebrovascular accident. Higher admission day elevated blood glucose level has increased mortality and high risk of poor functional recovery.

COMPARISON WITH OTHER STUDIES

- A study published on July 2012 by European Journal of Neurology stated that expansion of infarct volume is caused by admission day elevated blood glucose levels[44]. In our study, patients with hyperglycemia had larger infarct when compared to euglycemia.
- Another study by University of Glascow published by cardiovascular and medical sciences division conducted on 2011, indicates hyperglycemia in the per infarct time causes grave outcome in both diabetes and non-diabetes patients. Our study clearly demonstrated poor outcome in hyperglycemia patients.
- A study published by European journal of neurology, concluded that elevated glucose level after acute stroke is associated with higher severity than those with normal level. The mean stroke scale was 7.1 in patients with euglycemia and was 18.3 in patients with diabetes mellitus[45].
- Regarding insulin therapy and stroke outcome, a study was conducted in the year 2011 by neurology clinic, Poland stated that intensive insulin therapy maintaining strict glycemic control improves

cerebrovascular accidents outcome and concluded that insulin therapy is safe.

- In the study by clinical endocrinology journal, patients admitted with elevated blood glucose levels had a significant higher early mortality and a lower functional outcome than patients with normoglycemia[46]. Our study conducted in hundred patients signified same results.
- In our study, admission day hyperglycemia was associated with higher mortality and slower outcome or poor outcome in patients presented with both hemorrhagic stroke and ischemic stroke. Now many trials are under way to improve the outcome of stoke with human recombinant insulin for patients with hyperglycemia. Few studies have shown insulin administration improves functional recovery and vital activity of stroke. Our study has clearly demonstrated the detrimental effect of glucose on aggravating brain damage in stroke.

7. CONCLUSION

- The combined diabetes and stress hyperglycemic are found to have larger sized severe stroke and a poor functional outcome in the form of increased mortality. There is a linear correlation between admission day hyperglycemia and stroke in its severity, size and outcome.
- According to a study conducted at 2007 by Bangur institute of neurology, Calcutta medical college, admission day elevated glucose level was a significant predictor of mortality and poor functional outcome after acute stroke [47,48]. My study has concluded similar results.
- According to the **GLIAS STUDY** conducted in 2009, hyperglycemia in the range of >155 mg% in the first 48 hours is associated with poor outcome in ischemic stroke[49]. In my study , blood glucose in the range of 125-199 mg in the first 24 hours of onset of stroke is associated with poor outcome
- According to the Harrisons textbook of medicine, 18th edition, blood glucose should be maintained below <110 mg/dl using insulin infusion if necessary.
- Hence restoration of normoglycemia as soon as possible should be encouraged .In the interim, we should fare well with adhering to

good general stroke management, normalization of body temperature, fluid balance and hemodynamics.

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Antonio Dávalos, Antonio Gil Nuñez, José Egido a .Pertttu J. Lindsberg MD PhD RistoO .Roine MD hyperglycemia in acute stroke, stroke, 2004 , 35 ; 363

9. PROFORMA

NAME OF THE PATIENT :

AGE :

SEX :

IP NO :

ADDRESS :

OCCUPATION :

CHIEF COMPLAINTS :

HISTORY OF PRESENTING COMPLAINTS :

PAST HISTORY :

HT / DM / TIA / PT / BA / COPD / CRF / CAHD / DCM / RHD / AS / MVP / HEPATIC DISEASE / SEIZURES / PSYCHIATRIC DISORDERS

PERSONAL HISTORY :SMOKING, ALCOHOLISM, DIET

GENERAL EXAMINATION :

VITALS :BP, PULSE RATE, RESPIRATORY RATE AND PATTERN

CLINICAL EXAMINATION :

CENTRAL NERVOUS SYSTEM :

CARDIOVASCULAR SYSTEM :

RESPIRATORY SYSTEM :

ABDOMEN EXAMINATION :

SPINE AND CRANIUM :

CLINICAL DIAGNOSIS :

INVESTIGATIONS :

- Blood sugar , urea , Serum creatinine
- Serum electrolytes : sodium, Potassium
- Blood hemoglobin , TC , DC, ESR
- Urine albumin , sugar , deposits
- Total cholesterol
- Electrocardiogram
- Chest X ray
- CT scan brain
- Glycosylated Hemoglobin when required

FINAL DIAGNOSIS :

PROGNOSIS (30 DAY FOLLOW UP)

- Good / Moderate / Poor / Death

ABBREVIATIONS

HT	-	Hypertension
DM	-	Diabetes mellitus
CAHD	-	Coronary artery heart disease
DCM	-	Dilated cardiomyopathy
AF	-	Atrial fibrillation
HBA1C	-	Glycosylated hemoglobin
ACA	-	Anterior cerebral artery
MCA	-	Middle Cerebral Artery
PCA	-	Posterior cerebral artery
ICH	-	Intracerebral hemorrhage
TIA	-	Transient Ischemic Attack
COPD	-	Chronic Obstructive Pulmonary Disease
PT	-	Pulmonary Tuberculosis
BA	-	Bronchial Asthma
CRF	-	Chronic Renal Failure
MVP	-	Mitral Valve Prolapse
RHD	-	Rheumatic Heart Disease

KEY TO MASTER CHART

HT	-	Hypertension
DM	-	Diabetes mellitus
CAHD	-	Coronary artery heart disease
DCM	-	Dilated cardiomyopathy
AF	-	Atrial fibrillation
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BA	-	Bronchial Asthma
CRF	-	Chronic Renal Failure
MVP	-	Mitral Valve Prolapse
RHD	-	Rheumatic Heart Disease

NO.	IPNO.	AGE	SEX	PAST H/O	PERSONAL H/O	CT FINDINGS	ADMISSION DAY BLOOD-GLUCOSE	NIHSS	HBA1C	OUTCOME
1	16389	47	M	DM/ CAHD	Alcoholic/Smoker	Lt MCA &PCA infarct	160	12	-	Moderate
2	16299	65	M	-	Smoker/Alcoholic	Lt MCA infarct	140	10	6.2	Moderate
3	25711	41	F	HT	-	Lt MCA infarct	91	6	-	Good
4	14102	72	F	TIA/	-	RT MCA infarct	151	12	6	Moderate
5	25791	65	M	CAHD	Alcholic	Hemorrhage In Lt Thalamus region	131	23	6.2	Poor
6	35104	72	M	-	Alcholic	Infarct Lt MCA Area	81	7	-	Good
7	35809	67	M	HT	-	Infarct in RT MCA Area	91	6	-	Good
8.	35161	42	M	-	Alcoholic	Hemorrhage in Rt ventricle	161	25	6.2	Death
9.	33124	60	M	-	Smoke/Alcoholic	Infarct in parieto Occipital region	141	14	6.1	Moderate
10.	35187	76	M	HT	-	Left Frontal Hemorrhage	152	26	6	Death
11	24186	65	F	HT/TIA		Rt MCA infarct	163	16	6.2	Poor
12	65836	70	M	HT/	Alcoholic	Hemorrhage in Lt fronto Parietal Area	125	8	-	Good
13	67165	70	M	HT/ DM	Alcoholic/Smoker	Infarct LT MCA Area	165	18	-	Poor
14	60600	60	M	HT	Alcoholic/smoker	Hemorrhage in Rt Frontal Hemorrhage	166	25	9	Poor
15	30355	50	M		Alcoholic	Multiple infarct in Rt cerebral hemisphere	160	22	7	Poor
16	59550	45	M	-	Alcoholic	Multiple infarct in Rt Cerebral hemisphere	145	17	6.1	Death
17.	37705	59	M	HT	Alcoholic/Smoker	Hemorrhage in parietal area	91	7	-	Good
18	68466	80	F	HT/DM		Lt MCA infarct	222	16	-	Poor
19	38466	62	M	HT/ DM	Smoker	Rt MCA infarct	177	22	-	Poor
20	75836	63	M	HT	Alcoholic	Rt MCA infarct	167	18	8	Poor
21	66222	66	M	HT/ DM	Alcoholic/Smoker	Hemorrhage in fronto Parietal Area	248	24	-	Poor
22	213234	66	F	HT/ DM		Rt MCA Infarct	201	22	-	Poor
23	24442	48	M	-	Alcoholic	Hemorrhage in frontal area	81	7	-	Good

NO.	IPNO.	AGE	SEX	PAST H/O	PERSONAL H/O	CT FINDINGS	ADMISSION DAY BLOOD-GLUCOSE	NIHSS	HBA1C	OUTCOME
24	214232	42	F	CAD		Lt MCA Infarct	92	6	-	Good
25.	234543	45	F	-		Rt MCA infarct/midlins shift	145	8	6.1	Death
26	213423	48	M	-	Alcoholic	Rt MCA infarct/midline shift	198	10	8	Death
27	312021	50	M	-	-	Lt MCA infarct	199	16	6.1	Poor
28	213423	70	F	DM		Hemorrhage in Rt parieto occipital area	214	23	-	Death
29	312293	60	M	-		Hemorrhage in Rt frontal area	334	25	7	Poor
30	49015	73	M	HT		Lt MCA infarct	85	7	-	Good
31	25836	60	F	DM		RtMCA infarct/midline shift	300	10	-	Death
32	37165	77	M	DM		Lt MCAinfarct	232	16	-	Poor
33	58430	56	M	CAHD	Smoker/ Alcoholic	Lt MCA infarct	90	7	-	Good
34	55836	70	F	HT/DM		Lt MCA infarct	218	16	-	Poor
35	68430	82	M		Alcoholic	Lt MCA infarct /midline Shift	140	9	6	Death
36	35891	60	F	HT/DM		Rt MCA Infarct	189	16	-	Poor
37	58447	69	M	HT/DM		Lt MCA Infarct	199	16	-	Poor
38	42490	58	F	HT		RtMCA infarct	92	7	-	Good
39	43953	41	M		Smoker/ Alcoholic	Rt ICH with intraventricular extension	198	23	6	Death
40	43903	67	F	HT/DM		Parietal hemorrhage	240	10	-	Death
41	42653	60	F	HT		Lt MCA infarct	92	7	-	Good
42	41467	50	M			RtMCA infarct	89	7	-	Good
43	42708	70	M			Lt MCA infarct	144	16	6.2	Poor
44	42705	75	M		Alcoholic	Lt MCA infarct	124	10	6.5	Poor
45	42687	43	M		Smoker/ Alcoholic	Lt MCA& PCA infarct	149	10	6.5	Death
46	43997	65	F			Lt MCA infarct	87	7	-	Good
47	63162	88	F			Lt massive MCA infarct	144	10	7	Death

NO.	IPNO.	AGE	SEX	PAST H/O	PERSONAL H/O	CT FINDINGS	ADMISSION DAY BLOOD-GLUCOSE	NIHSS	HBA1C	OUTCOME
48	54361	75	F	HT/DM		Lt MCA infarct/midline shift	135	7	-	Death
49	41572	74	M			parietal infarct	92	6	-	Good
50	53521	56	M	CAHD		Rt MCA infarct	123	10	6.1	Poor
51	36431	67	M	CAHD		Lt MCA infarct	89	7	-	Poor
52	35413	58	M	CAHD		Lt MCA INFARCT	95	7	-	Poor
53	33514	61	M	CAHD	Smoking	Lt MCA infarct/ midline shift	147	9	6	Death
54	38156	73	M	CAHD	Smoking/Alcoholic	Lt massive MCA infarct	194	14	8	Death
55	32501	53	M	HT		Rt MCA infarct	142	10	6.1	Moderate
56	32506	48	M	HT/DM		Lt MCA infarct	160	10	-	Moderate
57	73721	55	M	CAHD/ DM		Rt MCA infarct/ midline shift	180	10	-	Death
58	41575	68	M	CAHD/	Alcoholic	Lt MCA infarct	140	10	6	Moderate
59	32568	61	M	HT		Rt MCA infarct	150	17	6.5	Poor
60	35512	62	M	HT	Alcoholic	Rt MCA infarct	88	7	-	Good
61	63125	61	F	HT/DM		Lt MCA infarct	178	15	-	Poor
62	64332	60	M	HT	Alcoholic	Lt MCA infarct	152	14	6.5	Poor
63	46122	62	M	CAHD/DM		Lt massive MCA infarct	164	10	-	Death
64	49586	65	M	CAHD		Frontal infarct	78	7	-	Good
65	43758	61	M	CAHD	Alcoholic	Cerebellar infarct	90	7	-	Good
66	51523	59	M	CAHD/ DM		Rt MCA infarct	112	10	-	Moderate
67	41532	72	M	CAHD/ DM	Alcoholic	Lt MCA infarct	241	14	-	Poor
68	61108	52	M	DCM/AF		Parietal infarct	88	7	-	Good
69	45324	61	F			Rt MCA infarct	173	7	6.1	Good
70	53254	55	F	CAHD		Hemorrhage in capsulo ganglion area	197	15	10.4	Moderate
71	26858	60	M	HT		Rt MCA infarct	121	14	6.1	Poor

NO.	IPNO.	AGE	SEX	PAST H/O	PERSONAL H/O	CT FINDINGS	ADMISSION DAY BLOOD-GLUCOSE	NIHSS	HBA1C	OUTCOME
72	21615	65	M	CAHD	Alcoholic	Lt MCA infarct	92	7	-	Good
73	35462	53	M	CAHD	Alcoholic	Temporal hemorrhage	92	7	-	Good
74	28017	59	M	DM/HT/ CAHD		Parieto temporal hemorrhage	187	10	-	Moderate
75	28481	53	M	CAHD	Smoking/ Alcoholic	Lt MCA infarct	123	17	6	Poor
76.	28292	59	M	CAHD		Parieto temporal hemorrhage	101	10	-	Moderate
78	65332	79	M		Smoking	Rt massive MCA infarct/ midline shift	145	9	6	Death
79	35421	76	M	CAHD	Smoking/ Alcoholic	Rt MCA with PCA, watershed zone infarct	121	25	6	Death
80	45231	53	M	CKD		Lt ICH	98	25	-	Death
81	87768	65	F	HT		Rt MCA infarct	88	7	-	Good
82	25480	55	M		Alcoholic	Lt MCA infarct/ midline shift	87	17	-	Death
83	25521	45	M		Alcoholic	Frontal infarct	72	7	-	Good
84	23539	47	M	HT	smoking	Rt MCA infarct	138	15	6.5	Poor
85	29195	63	M	HT		Lt MCA infarct	92	7	-	Moderate
86	45841	60	M	HT	Smoking/alcohol	Rt MCA infarct	152	15	6.5	Poor
87	58430	60	M	HT	Smoking/ Alcoholic	Rt MCA with PCA, watershed zone infarct	166	10	6.1	Poor
88	39105	64	M	HT/CKD/ DM		Hemorrhage in Rt parietal lobe	189	15	-	Poor
89	47615	45	M	HT	Smoking/ Alcohol	Rt ICH with dilated ventricle with midline shift	154	25	10.2	Death
90	39125	60	F			Rt MCA INFARCT	172	15	7	Poor
91	48625	47	F	HT		Rt ICH	210	16	8	Poor
92	37619	58	F	HT		Rt MCA infarct	99	7	-	Good
93	27915	65	F	DM/HT		Massive Rt MCA infarct	288	25	-	Death
94	17676	56	F			Lt MCA infarct	72	7	-	Moderate
95	17915	50	F	CAHD		Rt ACA infarct	194	10	10	Moderate

NO.	IPNO.	AGE	SEX	PAST H/O	PERSONAL H/O	CT FINDINGS	ADMISSION DAY BLOOD-GLUCOSE	NIHSS	HBA1C	OUTCOME
96		40	F	HT		ICH with intraventricular extension	178	18	6.1	Poor
97		66	F	DCM/AF		Parieto temporal hemorrhage	152	15	6	Moderate
98		62	F			Lt MCA infarct	85	7	-	Moderate
99		42	F			Rt MCA infarct	188	15	7	Poor
100		40	F	DM		Lt MCA infarct	208	15	-	Moderate

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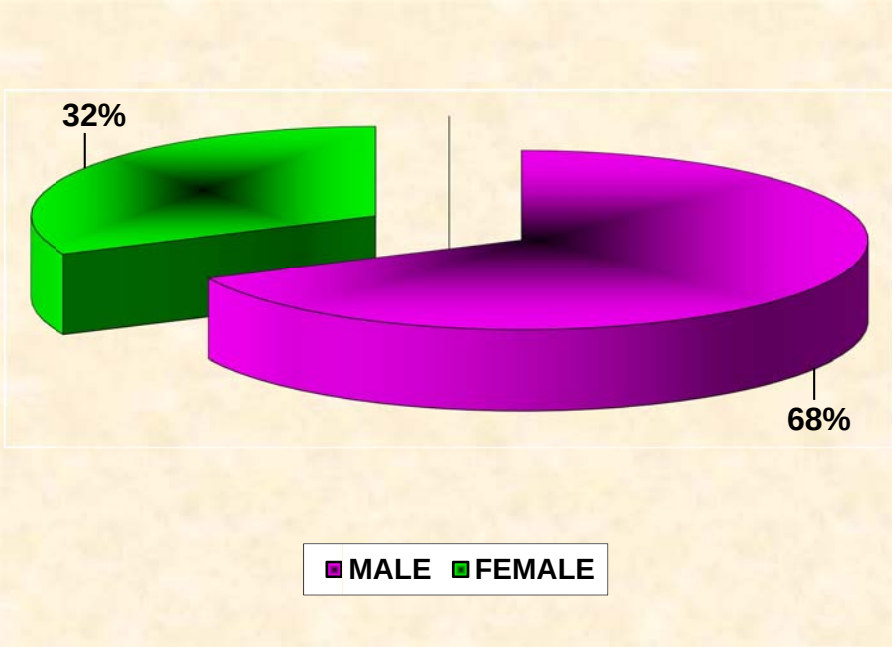

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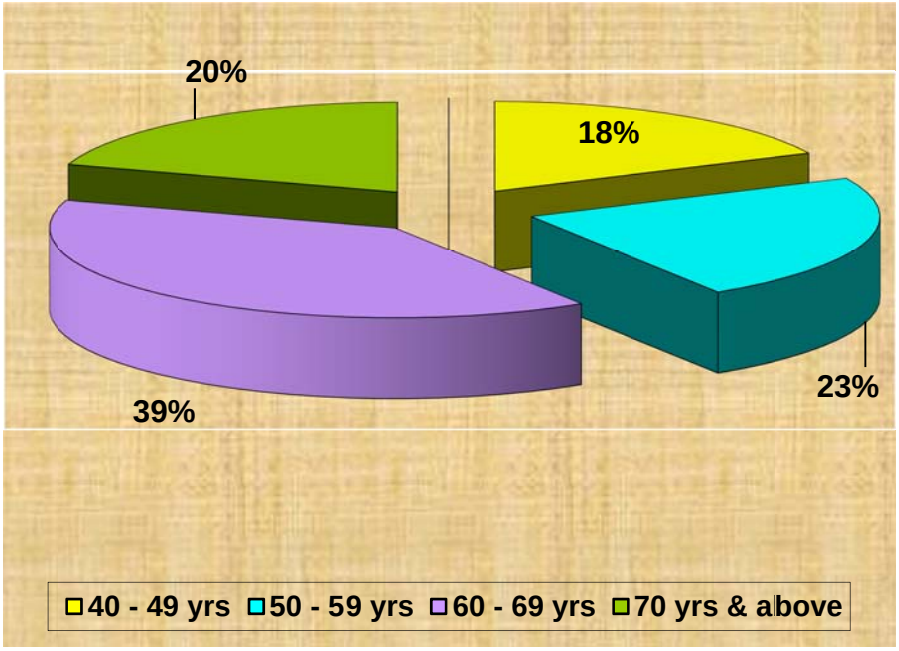

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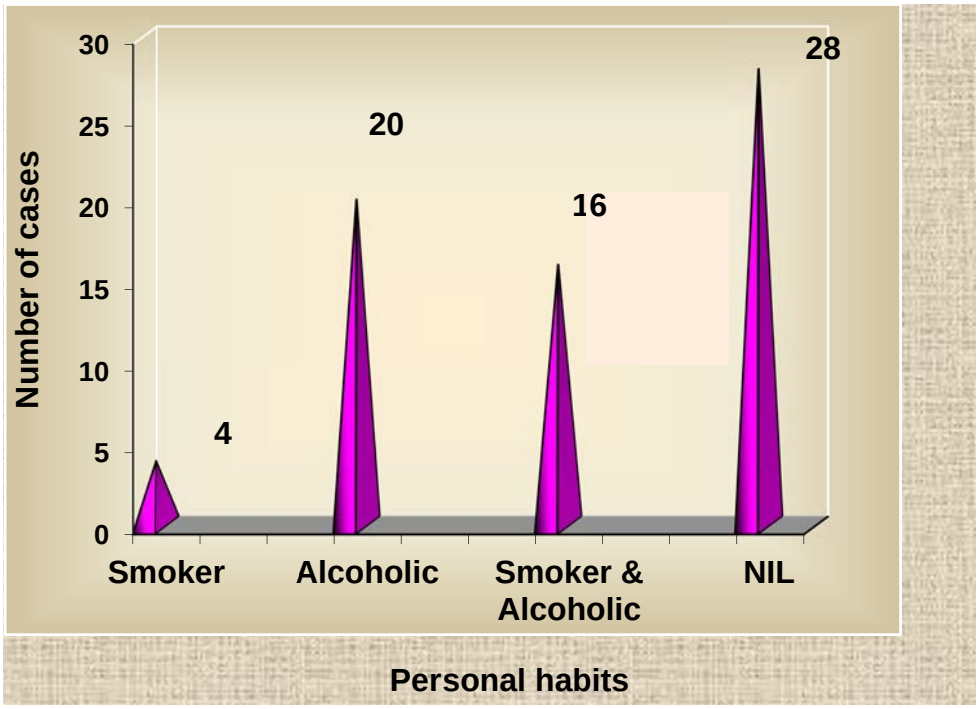

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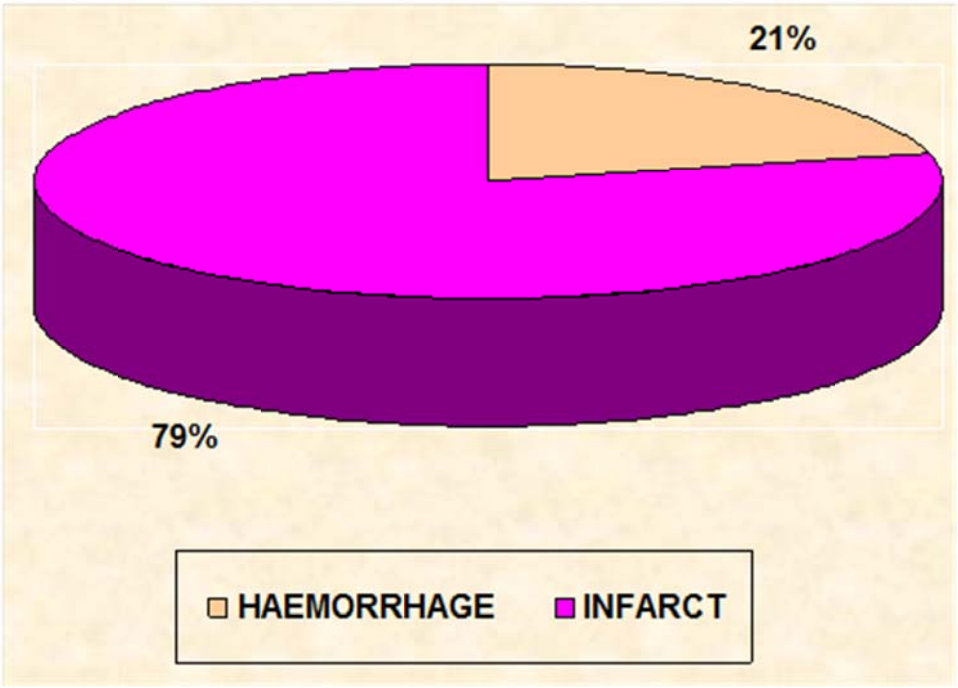

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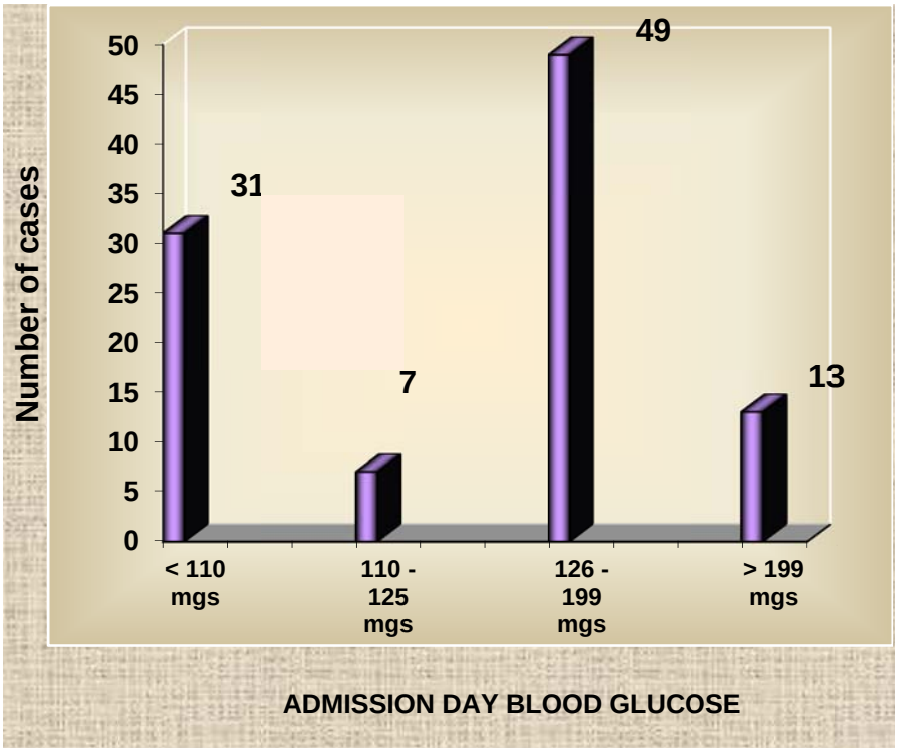
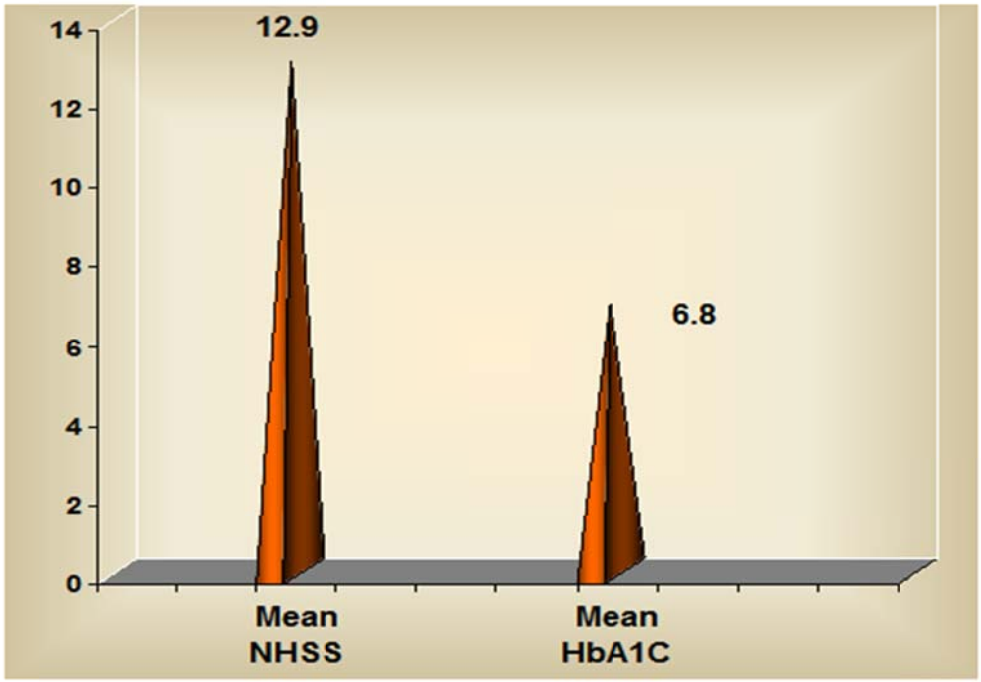


NIHSS AND HBA1C



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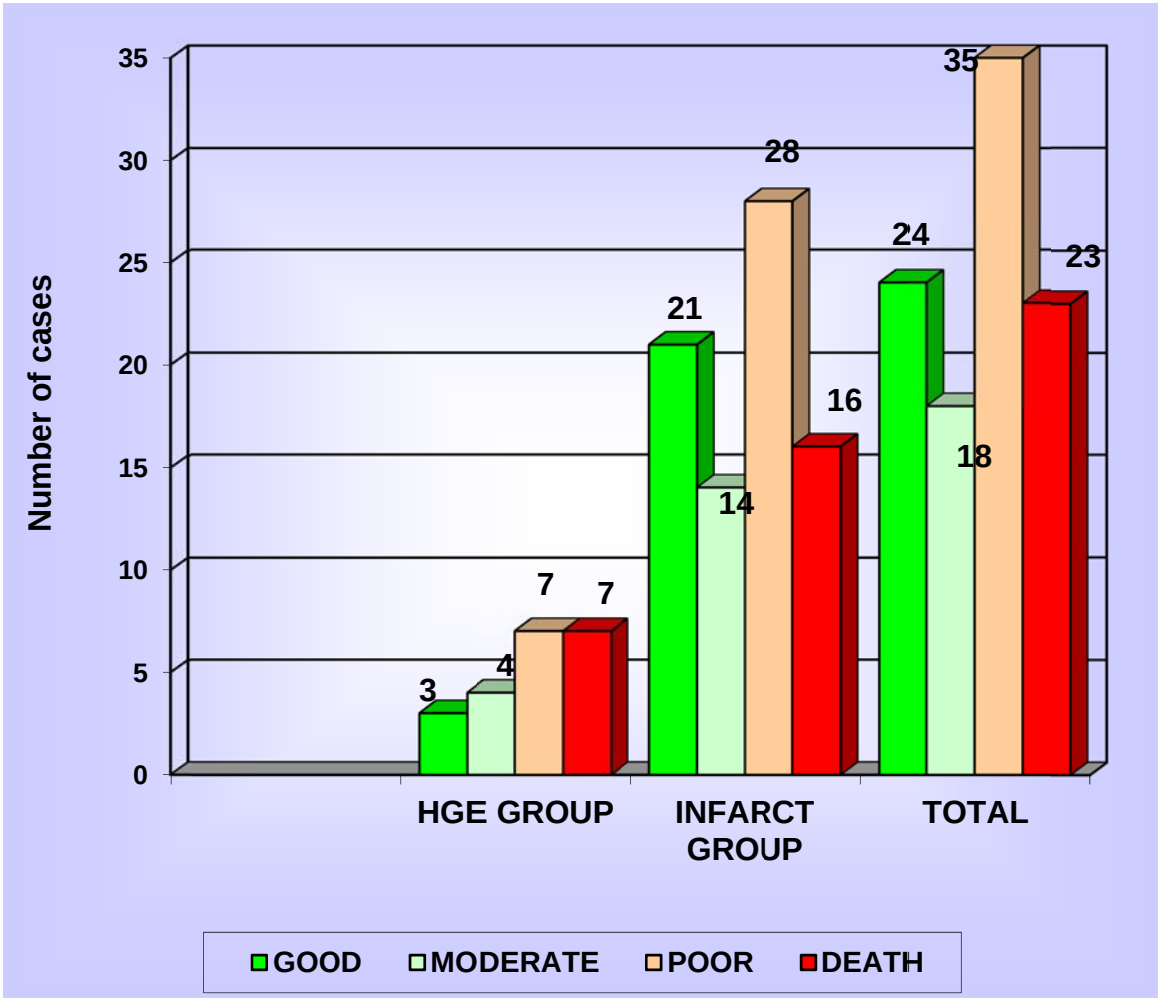
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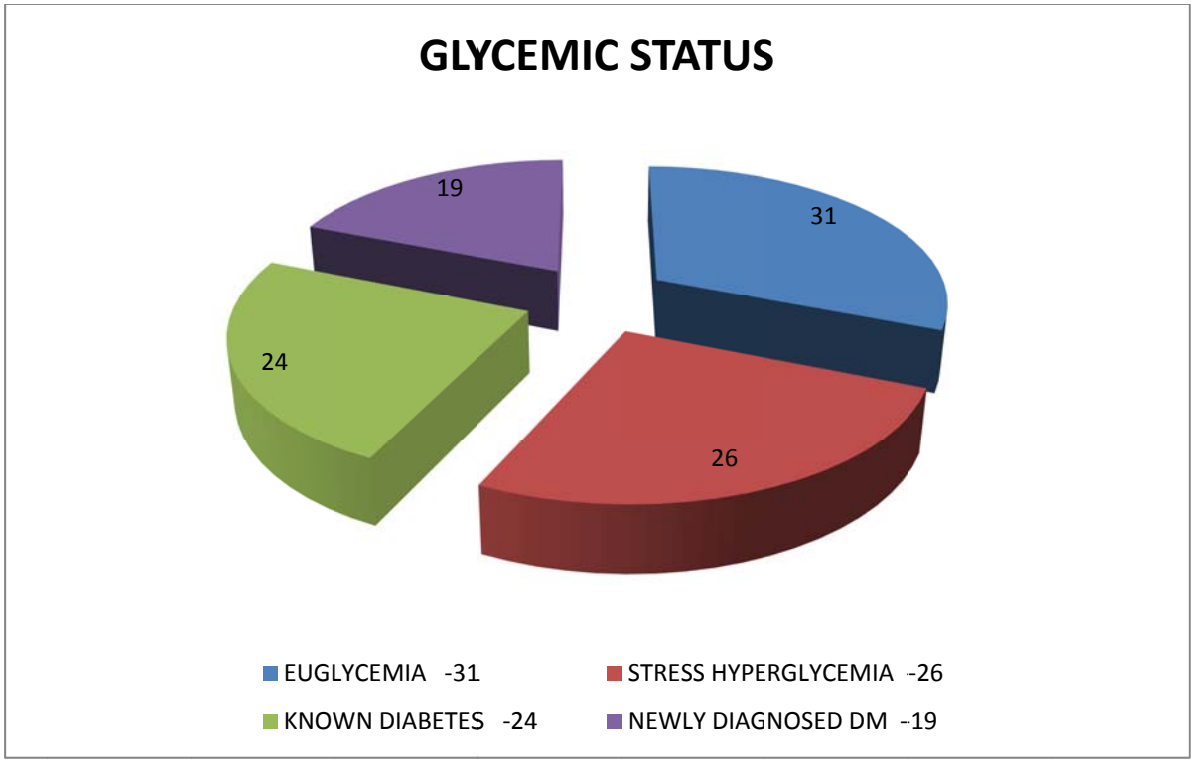
35
30
25
20
15
10
5
0
3
4
7
7
21
14
28
16
24
18
35
23
HGE GROUP
INFARCT GROUP
TOTAL
GOOD
MODERATE
POOR
DEATH



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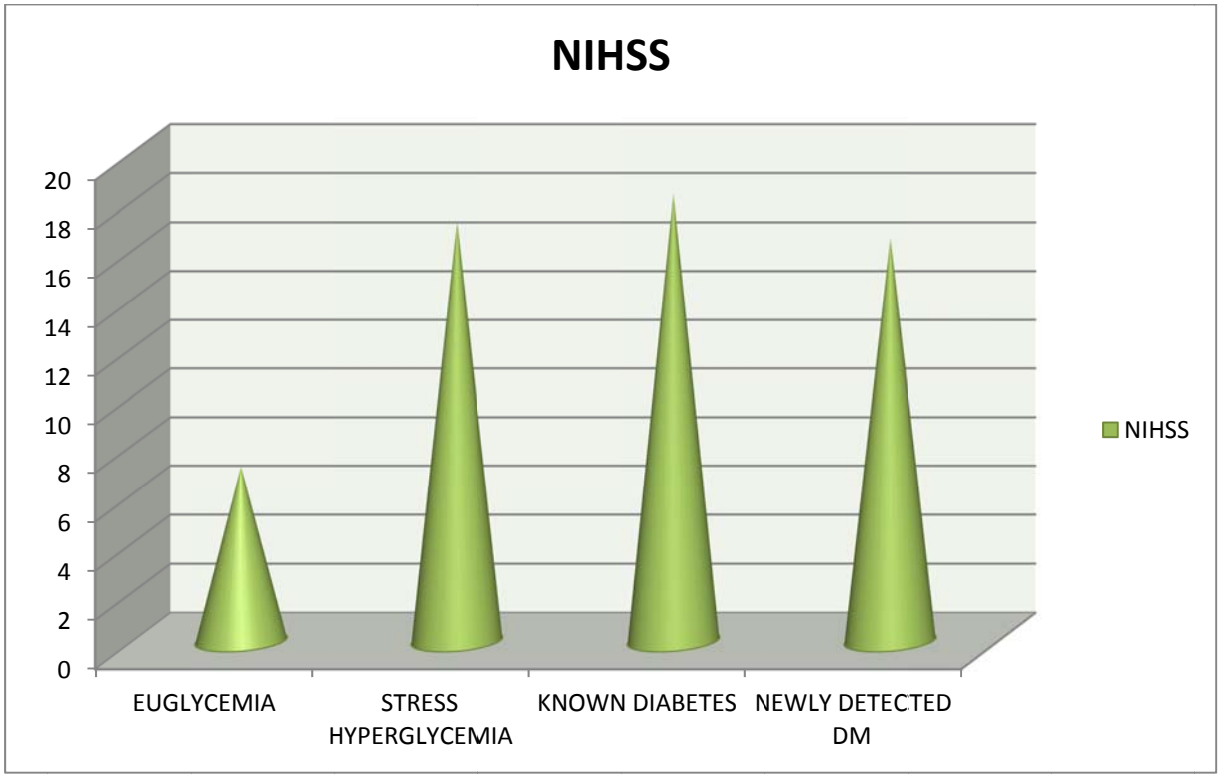
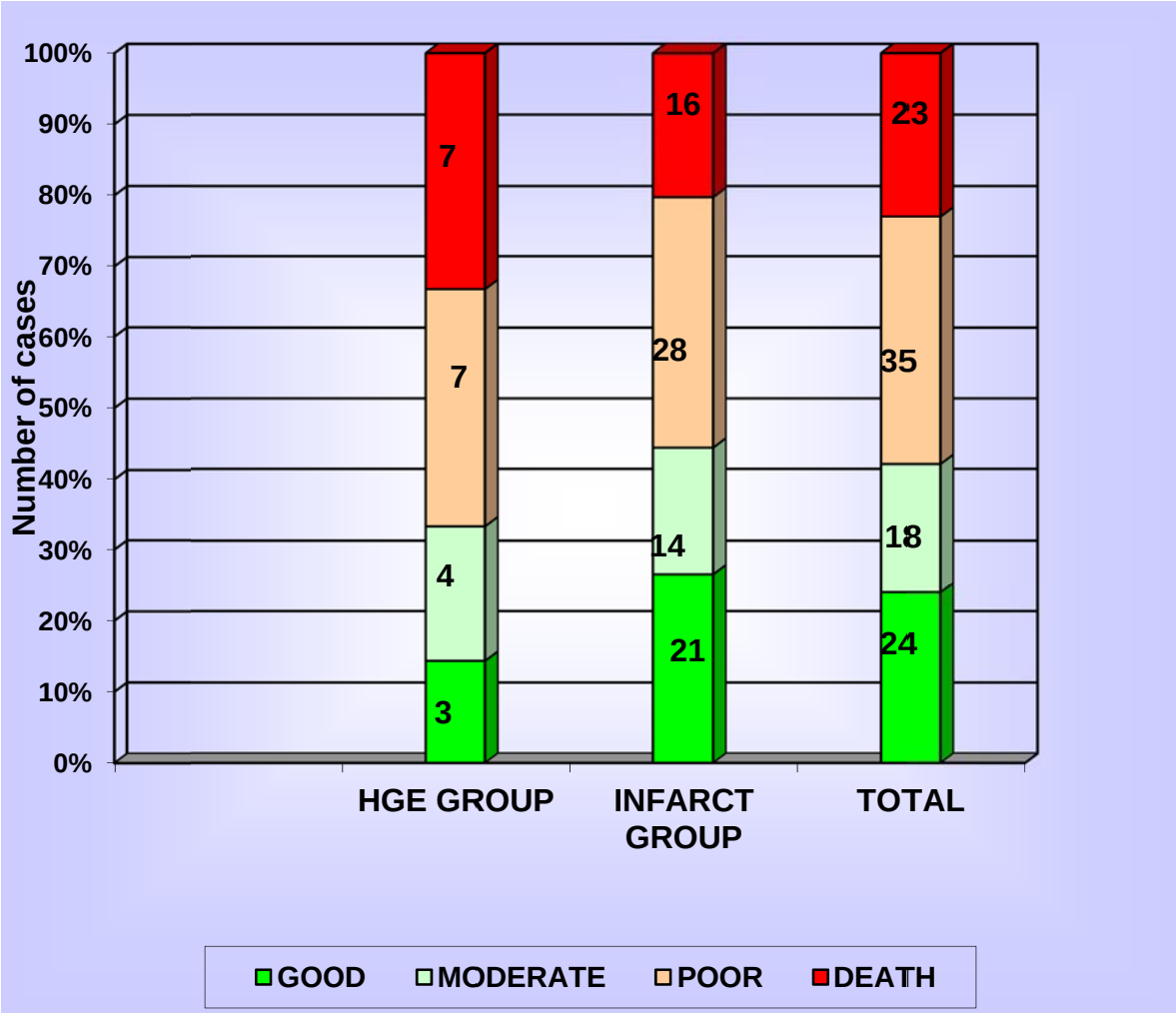


PROGNOSIS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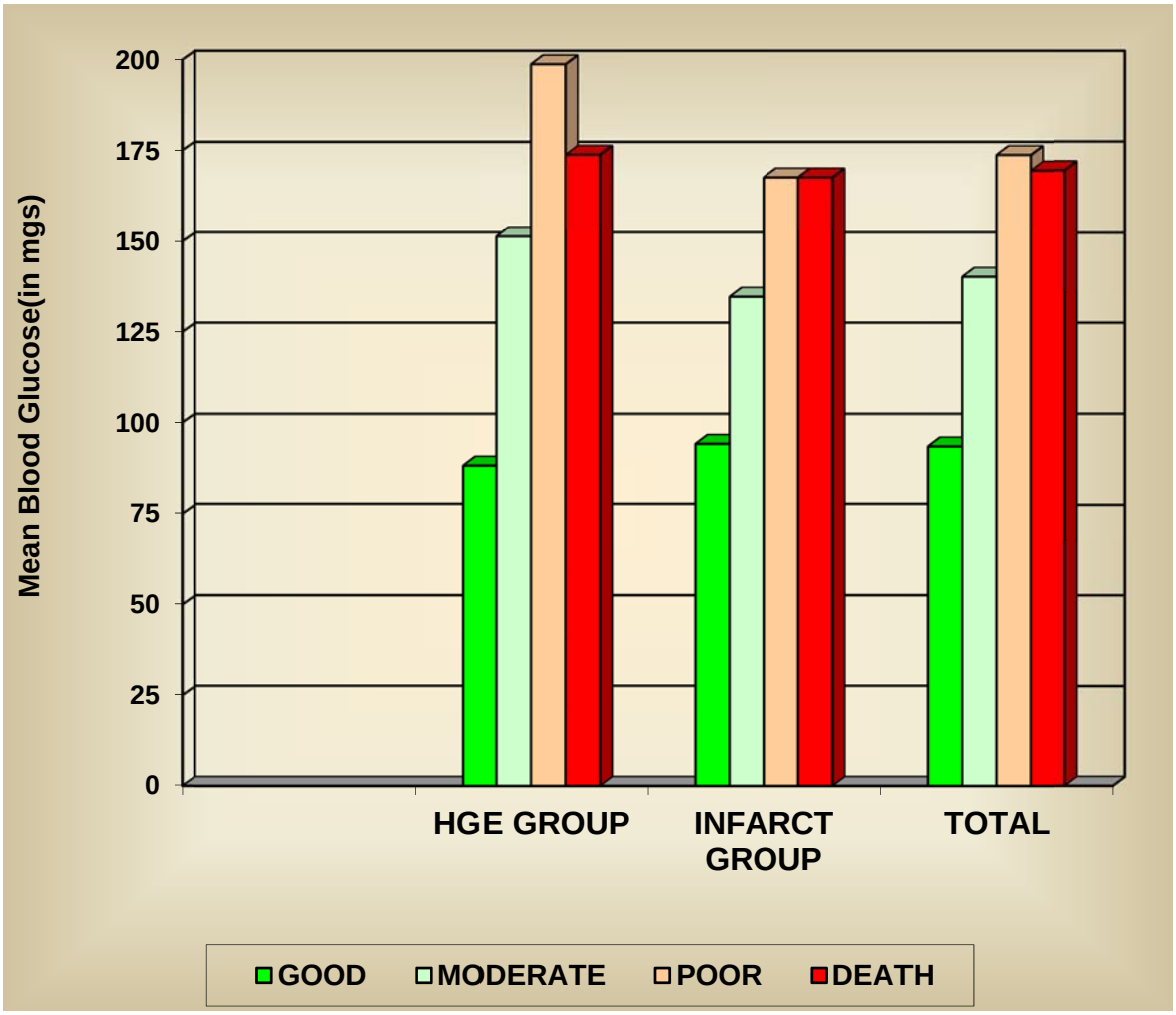

BLOOD SUGAR VALUES &OUTCOME IN HEMORRHAGE

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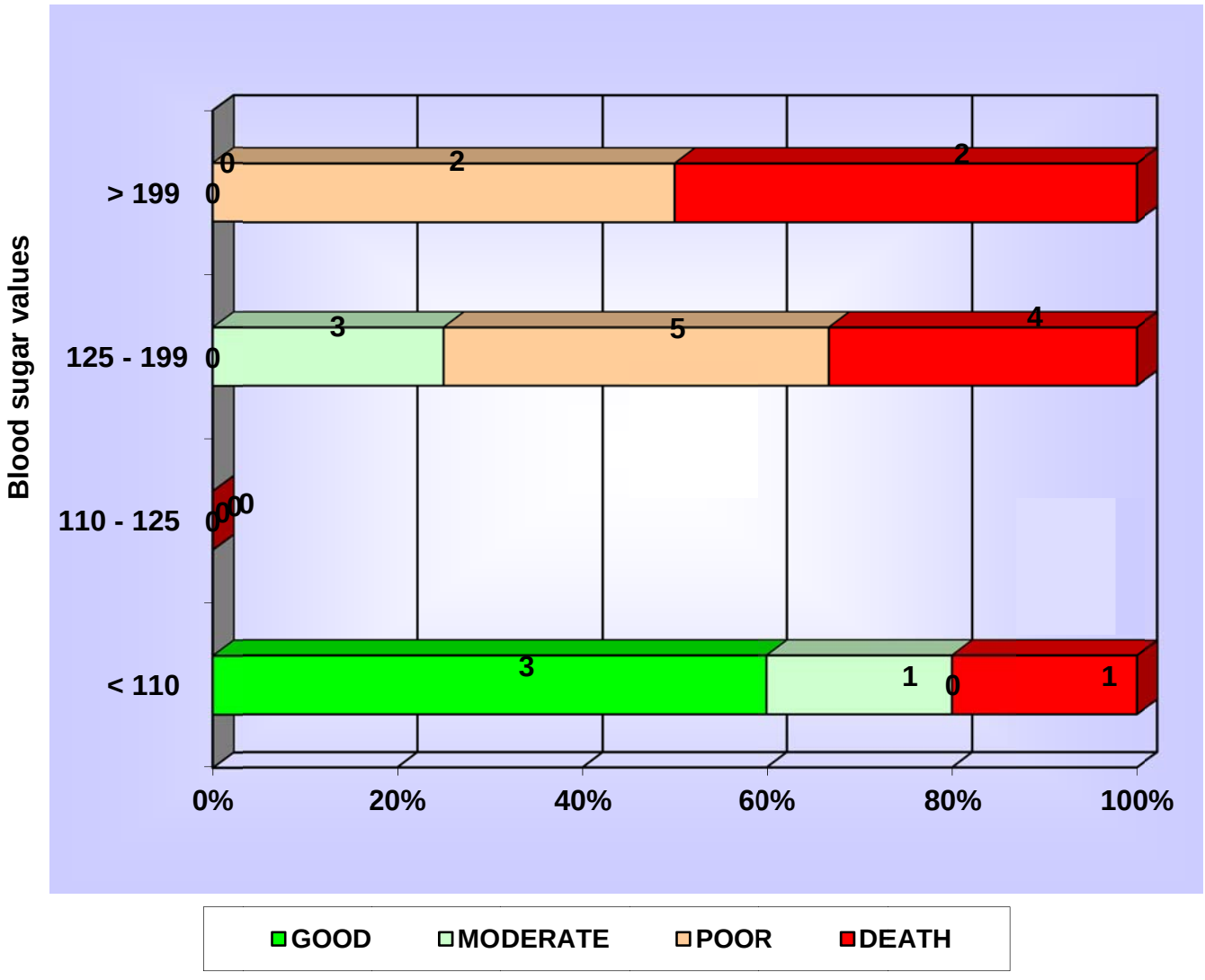
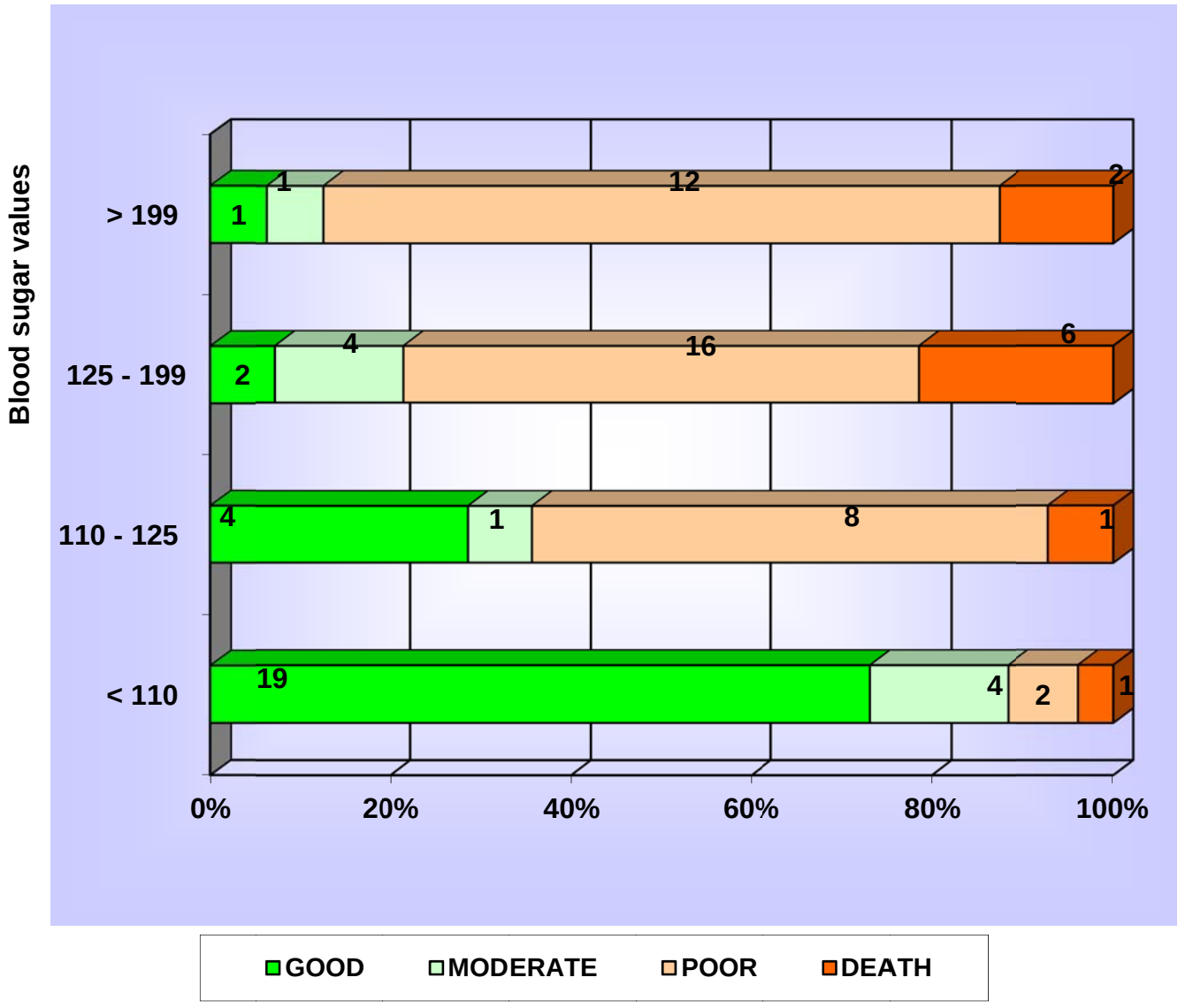


BLOOD SUGAR VALUES & OUTCOME IN INFARCT GROUP



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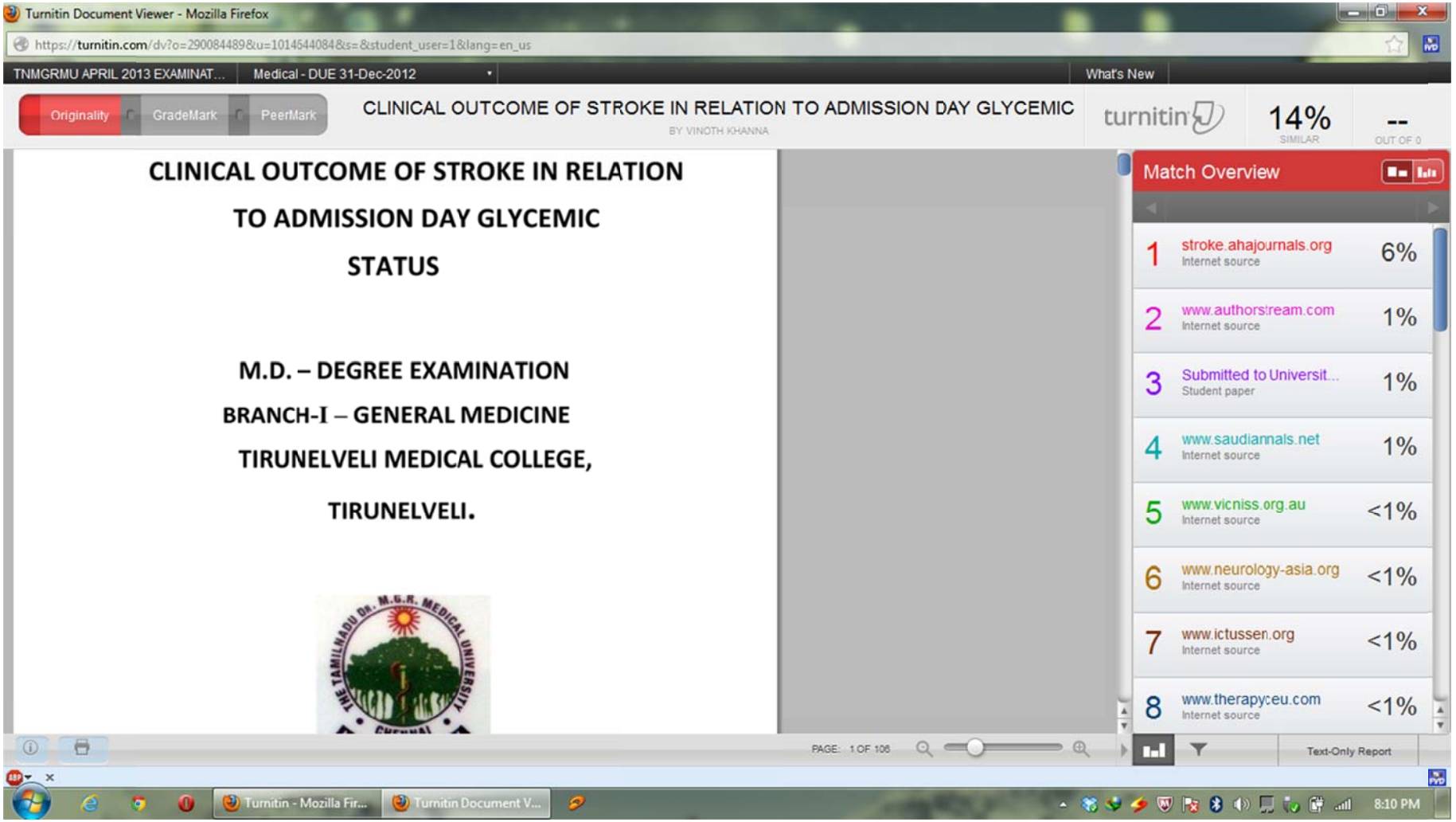
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