"A STUDY OF THE EFFECT OF DEHYDRATION ON EARLY NEUROLOGICAL DETERIORATION IN ACUTE ISCHEMIC STROKE"

By

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IN

GENERAL MEDICINE

Under the Guidance of

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ABSTRACT

A STUDY OF THE EFFECT OF DEHYDRATION ON EARLY NEUROLOGICAL DETERIORATION IN ACUTE ISCHEMIC STROKE

BACKGROUND: Stroke is a major cause of long-term disability among patients and has enormous emotional and socio-economic consequences. In 20-40 per cent of patients with acute ischemic stroke, neurological symptoms progress during the initial hours. Early Neurological Deterioration (END) has potentially serious consequences on the short term (morbidity and death) and long term (recovery from stroke) outcomes for the patients of acute ischemic stroke. Therefore, attempts to predict and prevent END should be made promptly and aggressively. The role of dehydration status of patient at the time of presentation as a risk factor for END is not yet proved and studies on the effect of dehydration on stroke outcome are limited. So the present study was designed to evaluate the effect of dehydration on early neurological deterioration in patients of acute ischemic stroke.

OBJECTIVES:

1. To assess the hydration status of acute ischemic stroke patients at the time of presentation by calculating Blood Urea Nitrogen to serum creatinine ratio.
2. To diagnose the Early Neurologic Deterioration among the patients with acute ischemic stroke by assessing NIHSS score on day 1 and day 3 of admission.
3. To correlate the hydration status at the time of presentation and development of early neurologic deterioration among the patients of acute ischemic stroke.

MATERIALS AND METHODS : All patients with Acute ischemic stroke enrolling to General Medicine OPD and Emergency department at R L Jalappa hospital, Kolar satisfying the inclusion criteria were taken up for study.

Study Design:

The study was conducted among acute ischemic stroke patients presented to the department of General medicine, RLJH. 125 patients with first episode of acute ischemic stroke presenting within first 24 hours after onset of symptoms were

enrolled in the study. The neurological status of the patients and the severity of stroke were assessed by applying the NIHSS (National Institute of Health Stroke Scale) score on day 1 and day 3 on all the patients. Early Neurological Deterioration was diagnosed if there was an increase in the NIHSS score by 3 or more than 3 points from day 1 to day 3 of admission.

Hydration status of the patients was assessed by calculating BUN/ serum creatinine ratio. BUN/ serum creatinine ratio more than 15 was be considered as marker of dehydration. The hydration status was correlated with Early neurologic deterioration.

RESULTS:

The prevalence of dehydration is more among patients who developed Early neurologic deterioration than who did not develop Early neurologic deterioration. On comparing hydration status in groups of people with and without END, 64.1% of patients with END were dehydrated whereas in group without END, 29.06% were dehydrated.($p < 0.001$).

CONCLUSION: Results of our study suggests that dehydration can be a risk factor for developing early neurologic deterioration in acute ischemic stroke patients.

KEY WORDS: Acute ischemic stroke, Early Neurologic Deterioration Dehydration

ABBREVATIONS

BUN	**BLOOD UREA NITROGEN**
CVD	**CEREBROVASCULAR DISEASE**
END	**EARLY NEUROLOGICAL DETERIORATION**
BUN/CR	**BLOOD UREA NITROGEN/ SEUM CREATININE**
CT	**COMPUTED TOMOGRAPHY**
CVT	**CORTICAL VEIN THROMBOSIS**
IHD	**ISCHAEMIC HEART DISEASE**
RHD	**RHEUMATIC HEART DISEASE.**
AF	**ATRIAL FIBRILLATION.**
CAF	**CHRONIC ATRIAL FIBRILLATION.**
CHF	**CONGESTIVE HEART FAILURE.**
TIA	**TRANSIENT ISCHAEMIC STROKE.**
ICA	**INTERNAL CAROTID ARTERY.**
NIHSS	**NATIONAL INSTITUTE OF HEALTH STROKE SCALE**
TPA	**TISSUE PLASMINOGEN ACTIVATOR.**
WHO	**WORLD HEALTH ORGANIZATION**

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INTRODUCTION

Stroke strikes fast. So should you.

\- Anonymous quote

Stroke is the third most leading cause of death worldwide after coronary heart disease and cancer, especially ischemic stroke[1]. It is more often disabling than fatal and is a major cause of long-term disability among patients and has enormous emotional and socio-economic consequences. In 20-40 per cent of patients with acute ischemic stroke, neurological symptoms progress during the initial hours[2]. Early Neurological Deterioration (END) has potentially serious consequences on the short term (morbidity and death) and long term (recovery from stroke) outcomes for the patients of acute ischemic stroke. Therefore, attempts to predict and prevent END should be made promptly and aggressively.

Various studies have reported the following factors to be predictors of early neurological deterioration (END) which include clinical parameters like stroke severity at presentation[3], medical history of diabetes mellitus[3], hypertension[3,4], body temperature[3], and laboratory tests like elevated markers of coagulation[3], markers of inflammation and serum glucose at admission[3,4].But most of these factors are either not reversible or difficult to be assessed.

Patients with stroke are often at increased risk of dehydration as they have a reduced level of consciousness, are physically dependent, unable to communicate, have difficulties in swallowing and decreased oral intake. Dehydration is a factor that can be readily assessed in an Emergency department (ED) setting and can be easily corrected to prevent worsening of neurological status.

But the role of dehydration status of patient at the time of presentation as a risk factor for END is not yet proved and studies on the effect of dehydration on stroke outcome are limited. So the present study was designed to evaluate the effect of dehydration on early neurological deterioration in patients of acute ischemic stroke. If the association is proved positive, volume resuscitation in patients to correct dehydration could be a safe, inexpensive and globally available technique to prevent Early Neurological Deterioration in acute stroke patients.

OBJECTIVES OF THE STUDY

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REVIEW OF LITERATURE

Acute ischemic stroke is a global problem that is associated with significant mortality and morbidity. It is the fifth most common cause of death worldwide[1]. More often disabling than fatal, stroke is not only the leading cause of long term disability worldwide, it is also the leading cause of preventable disability[1]. Moreover, stroke causes major social and economic burden to society.[2]

EPIDEMIOLOGYOF STROKE

The incidence of first onset stroke is 17 million per year worldwide[3].The lifetime risk of stroke after 55 years of age is 1 in 5 for women and 1 in 6 for men[3]. 1 in 8 strokes are fatal within the first 30 days. And 1 in 4 strokes are fatal within a year[3]. Studies indicate that both the worldwide incidence and the associated mortality of stroke have plateaued over the last few decades[4].

But in contrast, the stroke incidence in India and other developing countries has been rising[5]. More than four-fifth of all strokes are occuring in developing countries[5].The average annual incidence rate of stroke in India currently is 145 per 100,000 population[6], which is higher than the western nations. Rapid socio-economic changes have led to changes in people's lifestyle, work related stress, altered food habits and the risk of developing hypertension, diabetes and hyperlipidaemia. This coupled with increased lifespan has resulted in increase in the incidence of stroke.

DEFINITION OF STROKE

World health organization defines stroke as “rapidly developing clinical signs or symptoms of focal (at times global) disturbance of cerebral function with

symptoms lasting more than 24 hours or leading to death with no apparent cause other than that of vascular origin [7].

TYPES OF STROKE

Stroke is of two categories – Ischaemic and Haemorrhagic. Ischaemic infarction is again classified into thrombotic and embolic. Ischemic stroke constitutes about 80% of the total stoke cases[7].

ETIOLOGY OF ISCHEMIC STROKE :

I.THROMBOSIS:

- Atherosclerosis
- Vasculitis — Collagen Vascular diseases, Syphilis. Meningitis etc.
- Arterial Dissection
- Hematological disorders -- Polycythemia, thrombocytosis, TTP, DIC etc.
- Miscellaneous — Binswanger's disease,MoyaMoya disease ,fibromuscular dysplasia.

II.Embolism

- Cardiac sources
- Atherothrombotic arterial sources
- Unknown sources

III. Vasoconstriction

- Vasospasm
- Reversible cerebral vasoconstriction

IV. Venous

- Dehydration

- Postpartum and post- op states,
- Systemic cancer etc.

PATHOPHYSIOLOGY OF ISCHAEMIC STROKE

Cerebral ischemia is caused by a reduction in blood flow that lasts longer than several second. Neurologic symptoms manifests within seconds of ischemia because neurons lack glycogen, so energy failure is rapid .If the cessation of flow lasts for more than a few minutes, infarction of brain tissue results[8].

ACUNAR INFARCTS

They are small infarcts in the deep white matter of the cerebral hemisphere or brainstem. They are usually due to hypertension induced lipohyalinosis or arteriosclerosis of small penetrating arteries, rather than to large artery arteriosclerosis or cardio embolism [8].

- Patients with acute ischemic stroke present with a neurological deficit that is maximum at onset of stroke.
- 10-20% of thrombotic strokes may be associated with one or more transient ischeamic attacks(TIAs)[8].

ANATOMY OF CIRCULATION OF BRAIN:

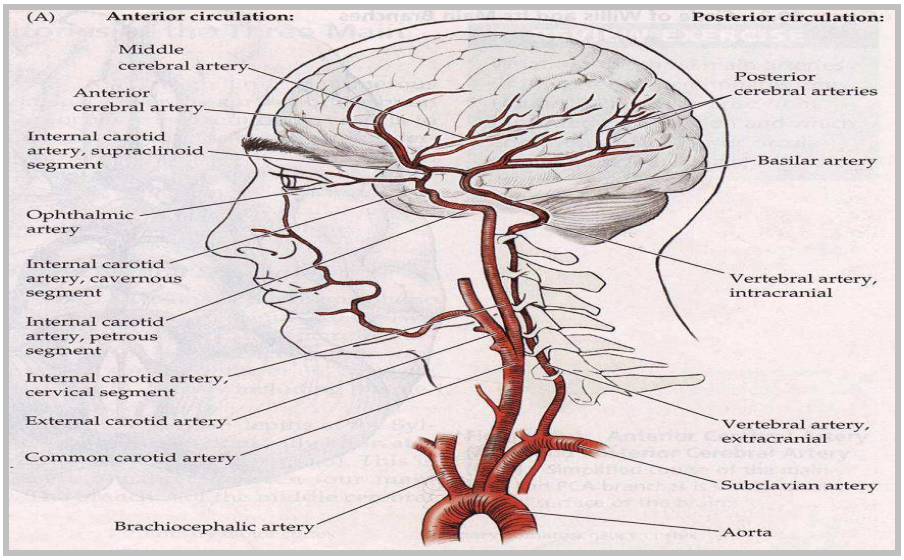


Fig no.1 Anatomy of Blood supply of brain

- **At rest the cardiac output is about 5 litres, of which 1 liter is retained by the brain[9] .**

Three types of vessels supply the brain[9]:-

- **Paramedian arteries : These vessels penetrate the brain on either side of the midline and supply the central nuclear areas near the midline.**
- **Short circumferential arteries : These travel for some distance before supplying the brain.**
- **Long circumferential arteries : These travel on the surface of the brain for some distance and then anastomose with branches of other circumferential vessels.**

The brain is supplied by two internal carotids and two vertebral arteries . The carotid circulation is designated as anterior circulation and vertebra basilar circulation as posterior circulation.

The internal carotid artery (ICA) begins at the bifurcation of the common carotid artery. It ascends the neck and perforates the base of the skull by passing through the carotid canal of the temporal bone. It enters the subarachnoid matter and turns posteriorly to region of anterior perforated substance of brain at medial end of lateral cerebral sulcus . Here it divides into anterior perforated substance of the brain at the medial end of lateral cerebral sulcus. Here it divides into anterior and middle cerebral arteries.[10]

ANTERIOR CIRCULATION

A) ANTERIOR CEREBRAL ARTERY

It is smaller terminal branch of the ICA. It runs forward and medially superior to the optic nerve and enters the longitudinal fissure of cerebrum. Here it is joined by ACA of opposite side by anterior communicating artery .It curves backwards over corpus callosum and finally anastomoses with posterior cerebral artery.(PCA)[10]

The cortical branches supply all of medial surface of cerebral cortex. They also supply a strip of cortex an inch wide on adjoining lateral surface. The ACA supplies the leg area of pre central gyrus[10].

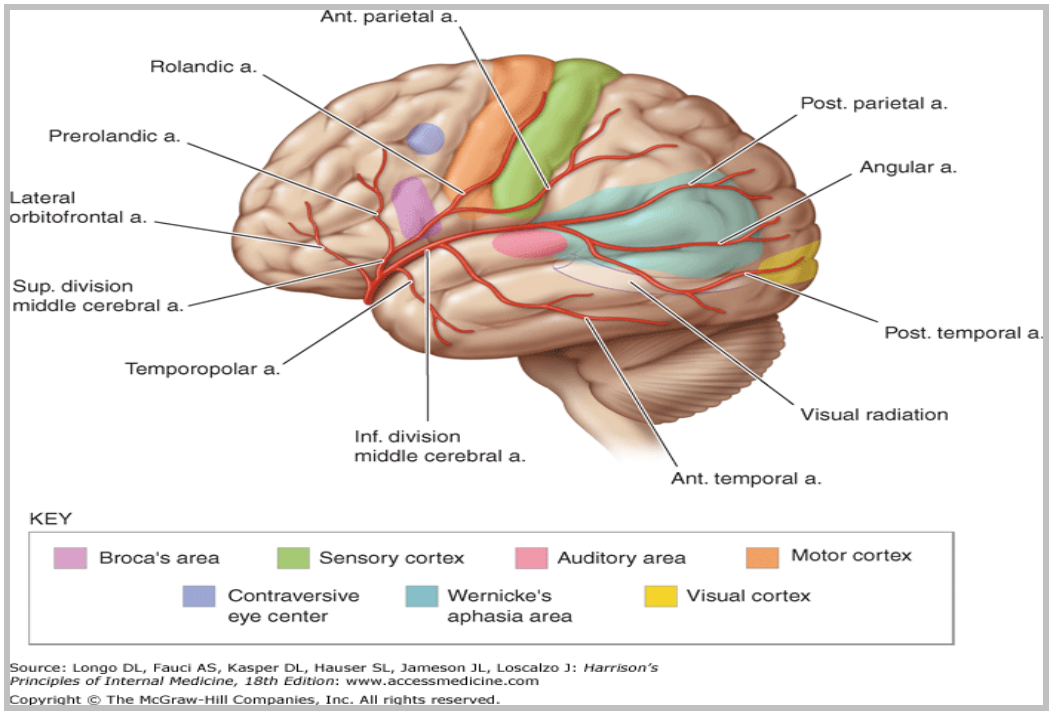


FIG 2: Diagram of a cerebral hemisphere , lateral aspect , showing the branches and distribution of the middle cerebral artery and the principal regions of cerebral localization.Note the bifurcation of middle cerebral artery in to suoerior and inferior division

B) MIDDLE CEREBRAL ARTERY

It is the largest branch of the internal carotid which runs laterally in the lateral cerebral sulcus[10].

Cortical branches supply the entire lateral surface of the hemisphere except for the narrow strip supplied by the ACA, the occipital pole and the inferolateral surface of the hemisphere supplied by the Posterior Cerebral Arteries (PCA). The artery thus, supplies all the motor area except the leg area [10]

Cerebral branches enter the anterior perforated substance and supply the lentiform and caudate nuclei and the internal capsule[10].

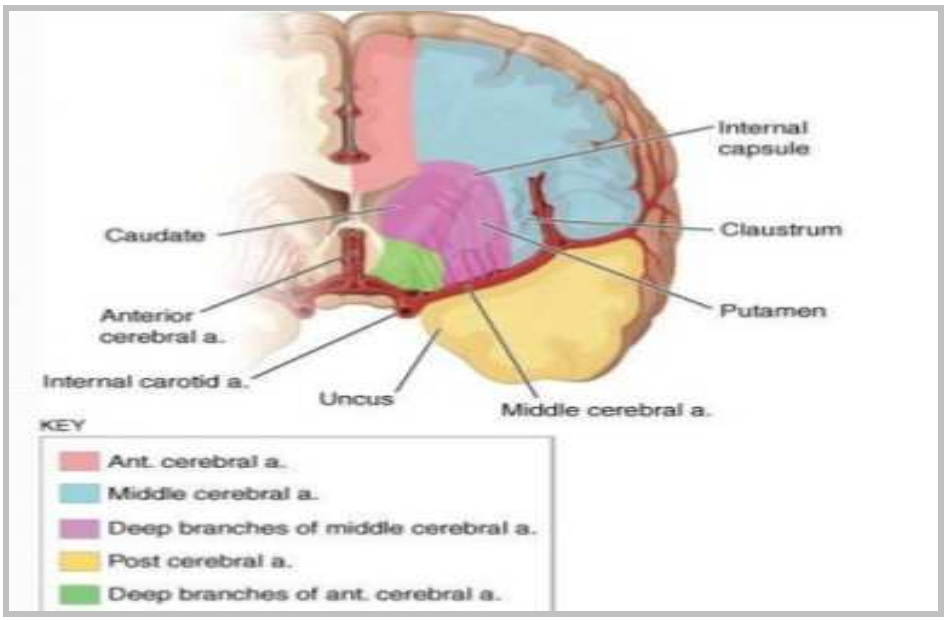


Fig 3 : Diagram of a cerebral hemisphere in coronal section showing the territories of the major cerebral vessels that branch from the internal carotid arteries.

POSTERIOR CIRCULATION

A) VERTEBRAL ARTERY

It is the branch of the first part of the subclavian artery which ascends the neck by passing through the foramina in the transverse processes of the upper six cervical vertebra. It enters the skull through foramen magnum and pierces the Jura and arachnoid mater to enter the sub arachnoid space. It then passes upward, forward and medially on the medulla. At the lower border of the pons it joins the vessel on the opposite side to form the basilar artery [10].

Branches of the cranial portion of the vertebral artery

- Meningeal branches : They supply the bone and the dura in the posterior cranial fossa.
- Posterior spinal artery : This vessel arises from the vertebral artery or the Posterior Inferior Cerebellar Artery (PICA). It descends as two branches, one anterior and one posterior, to the posterior roots of the spinal nerves. The branches are reinforced by radicular arteries that enter the vertebral canal through the intervertebral foramina
- Anterior spinal artery : This is formed from a contributory branch from each vertebral artery near its termination. The spinal artery descends on the anterior surface of the medulla and spinal cord and is embedded in the piamater along the anterior median fissure.
- Posterior Inferior Cerebellar Artery(PICA) : This is the largest branch of the vertebral artery which passes on an irregular course between the medulla and the cerebellum.It supplies the anterior surface of the vermis, Central nuclei of the cerebellum, the under surface of the cerebral hemisphere. It also supplies the medulla oblongata and the choroid plexus of the fourth ventricle.
- The medullary arteries: They are very small branches that are distributed to the medulla.

B) BASILAR ARTERY

It is formed by the union of two vertebral arteries. It ascends in a groove on the anterior surface of the pons. At the upper border of the pons it divides into two Posterior Cerebral Arteries (PCA)[10].

BRANCHES

- Pontine arteries
- Labryinthine artery- : This supplies the inner ear.
- Anterior Inferior Cerebellar Artery (AICA) : This Supplies the anterior and inferior parts of the cerebellum.
- Superior Cerebellar Artery (SCA) : This vessel arises close to the termination of the basillar artery, Supplies the superior surface of the cerebellum, pons, pineal gland and superior medullary velum.

PCA curves laterally and backward around the mid brain and is joined by the posterior communicating branch of the ICA.

Cortical branches supply the inferolateral and medial surfaces of the temporal lobes and the lateral and medial surfaces of the occipital lobe. Thus PCA supplies the visual cortex. Cortical branches pierce the brain substance and supplies parts of the thalamus. The lentiform nucleus, the mid brain, the pineal gland and the medial geniculate bodies. Choroidal branches supplies the choroid plexus.

THE CIRCULUS ARTERIOSIS (CIRCLE OF WILLIS)

This lies in the interpeduncular fossa at the base of the brain. It is formed by the anastamoses between the two ICAs and two vertebral arteries, Cortical and central branches arise from the circle and supply the brain substance.

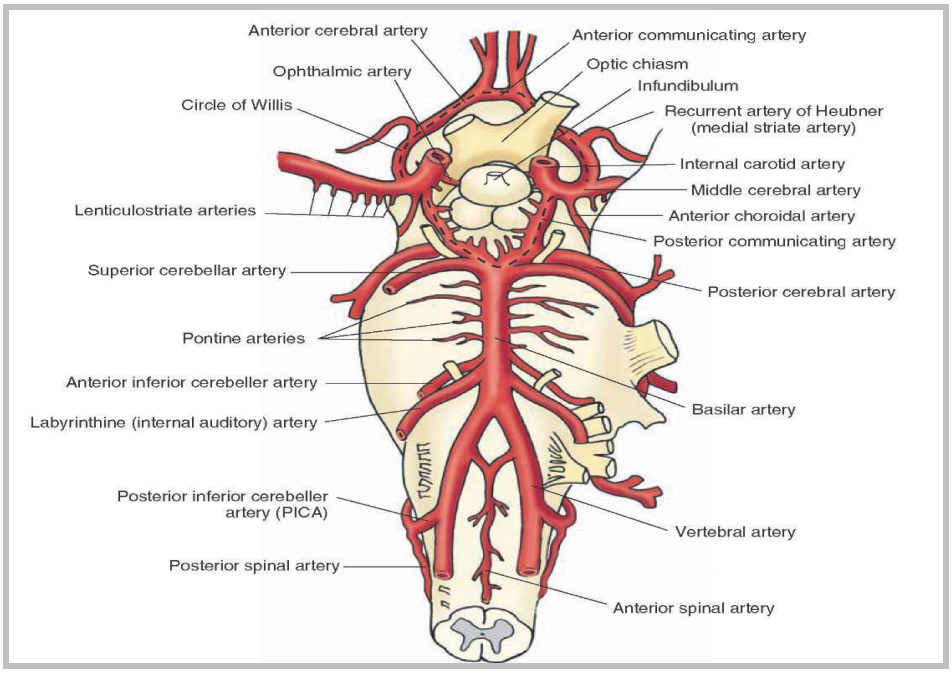


FIGURE NO. 4 CIRCLE OF WILLIS

RISK FACTORS IN CEREBROVASCULAR DISEASES

Risk factors were briefly classified as –

i. **Non modifiable**

ii. **Modifiable**

NON MODIFIABLE RISK FACTORS INCLUDE:

Age – advanced age

Sex- male sex

Race/ ethnicity

Family history of stroke

Genetics

MODIFIABLE RISK FACTORS INCLUDE:

Hypertension (Diastolic, Systolic)

Diabetes mellitus

Heart Diseases

Transient Ischaemic Attacks

Obesity

Dyslipidemias

Alcoholism

Smoking

Hyperuricemia

Infections

Hematocrit (Increased, decreased)

Migraine

Usage of Oral contraceptives etc

Age:

People of age group between 5th to 8th decade are more prone for stroke.[11,12]. A study done by Nagaraja et al[13] found that the peak incidence of stroke was in the 6th decade. The incidence of stroke continues to increase with advancing age. This is due to decreased blood flow which occurs normally with age. Studies from UK have shown that the risk of stroke in people aged between 75-84 years is 14.3/1000 / year i.e.25 times the risk in people aged between 45-54 years which is 0.57/1000/year[14] . The joint committee for stroke facility estimated that the death rate was 1/1000 at age 45-54 years and 9/1000 at age 65- 74 years [15].

Sex:

This change is more striking in women than in men especially after the age of 64 years[16]. In Nagaraja et al's study[13] men out numbered women in both fatal and non fatal stroke in the ratio of 1.3:1. In the Framingham cohort, 8% of all deaths in women and 5 % in men were due to stroke. This study also showed that the incidence of stroke in the age group 65-74 years was 84/10,000 /year for men and 86/10,000 /year for women [17].

In Kamel Abdelaziz mohamed study in 2013, male patients were 61% and female patients were 39%.[18]

In Abdu Hameed AI Kassir study in 2012, male patients were 67.6% and female patients were 32.4% [19]

In Hala El Kawas study in 2006, male patients were 56.6% and female patients were 43.3%. [20]

ARTERIAL HYPERTENSION

Hypertension is a major risk factor for both ischemic and haemorrhagic stroke [21]. Hypertension increases the stroke risk by increasing the extent and severity of atheroma[22] and the prevalence of microvascular disease in the small penetrating arteries within the brain which are end arteries[23].

60 % of strokes occurred in men with systolic BP > 160 mm Hg ,in a study done by A.G. Sharper et al Britain . They also found that patients with systolic BP between 160 and 180 mm Hg had a 4 times higher risk of stroke than in men with BP < 160 mm Hg. With the systolic > 180 mm Hg, this risk increases to six fold. They found a weaker relation of diastolic BP with stroke which was lost on 4 regression analysis [24].

Individuals who have other clinical manifestations of hypertension such as LVH, proteinuria or retinopathy,[21] the risk of stroke will be more. Sharper and colleagues found LVH as a contributory factor in patients with Ischaemic Heart Disease (IHD) [24].

Stephen McMohan et al in their study on hypertension as a risk factor found that diseases rates were lowest among those individuals whose baseline diastolic BP was 65 mm Hg and whose usual diastolic BP was probably 73 mm Hg[25].

In Indian studies, it was found that the incidence of hypertension in stroke patients varied from 16-55 % to 23-47.4% . Nagaraja and Pratap Chand[13] found the incidence of hypertension to be 24 % in fatal and 16 % in non fatal cases 17. The relative risk for hypertension is ischaemic stroke was 3.6 in their study.

Isolated systolic hypertension was more frequent in fatal cases while isolated diastolic hypertension was seen mainly in the non fatal cases[26].

DIABETES MELLITUS (DM)

Diabetes mellitus is quoted as an important risk factor for CVD in the developed world by WHO [27].

According to WHO stroke report, DM forms a risk factor in ischaemic strokes in large vessel diseases but is of questionable impact in small vessel diseases. The role of its risk in hemorrhage stroke is yet to be clarified[27].

Control of hyperglycemia can diminish the severity of cerebral damage during the acute stroke period but there is no evidence that controlling diabetes decreases stroke incidence [28].

Some authors like Kier et al suggested that diabetics as well as patients with stress hyperglycemia have severe stroke and these patients are associated with poor prognosis. [29]

Nagaraja and Pratap[13] from NIMHANS, Bangalore have found that DM was twice as common in fatal as compared to the non -fatal group, possibly the mechanism suggested by Jorgensen.

CARDIOVASCULAR DISEASES

Rheumatic heart disease (RHD), coronary artery diseases with MI, cardiac arrhythmias ,cardiac emboli are the most common risk factor for ischaemic stroke [30].

Atrial fibrillation (AF) is the most important and frequent cardiac source of embolism to the brain . Peterson P et al in their study showed a distinct clustering of emboli at the time of onset of paroxysmal atrial fibrillation [30]. It is well established that chronic atrial fibrillation (CAF) carries an increased risk of strokes. In the Framingham study dealing only with CAF, an increased risk of stroke was found. AF in the absence of RHD was associated with more than a five fold increase in stroke incidence, while AF with RHD had a 17 fold increase compared to the controls without AF[31] .

Prevalence of AF was 17% for all strokes types and 18 % for infarction in the community stroke project in Oxfordshire. AF was not associated with a definite excess risk of recurrent strokes, either within 30 days or within the first few years[32].

In Indian studies, cerebral emboli from cardiac sources is a major detectable cause of stroke in the young. Sridharan noted that 36.5 % of ischaemic stroke patients had heart diseases, the relative risk being 2.250, Besides RHD with AF and ASD with paradoxical embolus. MVP was also associated with increased.[33]

Patients with coronary heart disease were found to have three fold increase risk of stroke. Those with CHF had almost five fold increased risk. LVH in ECG is associated with five fold increased risk of stroke.[32] Cardiac impairment which have been found to contribute independently to stroke include LVH on ECG, cardiomegaly on CXR, coronary heart diseases, congestive heart failure and AF[24,25].

TIAS AND COMPLETED STROKES

TIAs and previous completed strokes are important risk factors for all strokes more so for ischaemic strokes. Previous strokes is a greater risk factor for subsequent stroke than TIA alone [30].

From Indian studies, Agarwal et al[33] noted an incidence of 19.8 % past history of TIAs in lschemic stroke and Sridharan noted 15 % TIAs in Ischaemic stroke [34].

In Oxfordshire community stroke project, the risk of stroke in the first year after TIAs was 12 % and approximately 6 % per year over the first 5 years. They found that patients who suffered a TIA had a 13 fold excess risk of stroke during the first year[35].

THROMBOSIS OF EXTRACRANIAL VESSELS

Atherosclerosis in extra cranial cerebral vessel is a risk factor for thrombotic stroke. The manifestations may be in the form of carotid bruit, occluded carotids and peripheral vascular diseases [27].

Sridharan noted carotid bruit in 6.8 % of lschemic stroke patients[34]. Carotid and supraclavicular bruit is a risk factor for subsequent stroke[36]. Atherothrombotic

disease of the large extracranial arteries including the carotids accounts for 34 % of strokes[37].

DYSLIPEDMIA

Elevated levels of LDL is an important risk factor for atherosclerosis per se[38]. Various lipid abnormalities have been studied and it has been proposed by Bansal et al that hyperlipidemia contributes to a large majority of non embolic thrombotic strokes even in the young[39].

Agarwal et al had found elevated free fatty acids as significant in women with thrombotic strokes [33]. Reed DM et al found that elevated blood lipid levels is associated with extra and intracranial atheroma[22].

ALCOHOLISM

Heavy drinking may be an independent risk factor ,moderate drinking can be protective[77 63] . the Hisagama study and the Honolulu studies have shown increased risks of hemorrhagic stroke in alcoholics [64]. There is evidence that an acute alcoholic episode or chronic alcoholism are each important risk factors for all strokes and for ischeamic stroke[65].

ORAL CONTRACEPTIVE PILLS

It is estimated that there is ten fold increased risk of stroke in women taking OC pills when compared to women not taking them. In a study conducted among young women consuming OC pills, it was showed the use of OC pills triples the risk of stroke in young women [34].

OBESITY

Whether obesity is an independent risk factor for stroke is not known[30]. The risk factor status of obesity in Indian studies is also not established[40]

INFECTIONS

Infections commonly associated are tuberculosis, helminthic infestations, malaria, syphilis and leptospirosis. Clinicians report that systemic viral and bacterial infection is a risk factor for stroke but the data is inconclusive[30].

HEMATOCRIT

Even though pathologically elevated hematocrit has long been recognised as a predisposing condition for stroke, the Framingham study showed that this was true even within the normal range of hematocrit[41].

Cerebral blood flow has been found to be significantly lower in patients with hemotocrit values between 36- 46 %[42].

In a case control study of ischaemic stroke in the young, Dalal found a low hematocrit to be a significant risk factor[43]. Chopra et al found a low hematocrit in 8% patients with thrombotic stroke and in 61 % of patients with puerperal intravenous occlusion[39]. The mechanism by which a low hematocrit predisposes to cerebral Ischemia is uncertain.

In the ICMR stroke study, low normal haemoglobin % has been reported as an important risk factor for stroke in young and elderly subjects[44].

On the other hand, the EC / IC bypass study group concluded that severity of strokes was not different in subjects with high hemoglobin concentration as against those with lower values[45]

SMOKING

Cigarette smoking is an important risk factor for all strokes. There is also ample evidence that cessation of cigarette smoking will eliminate it as a risk factor [15,30]. Nicotine transiently elevates blood pressure and could enhance the risk of stroke this way. It may also enhance platelet aggregation [21].

In earlier Indian studies cigarette smoking was not found to be a significant risk factor for stroke[39,46]. But the ICMR study confirms the relationship between cigarette smoking and stroke[44].

Sridharan in his study noted 33.5% smoking in ischaemic stroke patients with a relative risk of 1.7 [34].

FACTORS EFFECTING OUTCOME OF STROKE:

Many factors effect the outcome of ischemic stroke. Unlike the risk factors of the stroke, much is not known about the factors effecting poor prognosis of stroke. Studies done suggest a diversity of factors are associated with an unfavorable outcome after acute IS.

Clinical characteristics such as –

1.Initial severity of stroke[47,48],

2. Older age at stroke onset[47,49]and

3. Existence of comorbid conditions like Type 2 diabetes and hypertension[50,51]

4.High blood sugar level on presentation[51] are well established factors.

EARLY NEUROLOGICAL DETERIORATION (END)

Clinical deterioration of patients with acute ischemic stroke within the first few hours or days is a serious complication and is associated with increased rates of mortality and morbidity. The occurrence of deteriorating stroke varies from 13 to 37% among various published studies[52-59] . The reasons for such wide variability in incidence may be due to differences in study population, difference in terminology and in the concept of progressive stroke.

The terms 'stroke-in-evolution','progressive stroke', 'worsening stroke' and 'deteriorating stroke' are used interchangeably regardless of whether the deterioration is caused by extension of the infarction or various other reasons[60] . The terms 'stroke in- evolution' or progressive stroke' are used when the stroke progresses in a stepwise manner or smoothly over several hours. The term 'deteriorating stroke' includes not only 'stroke-in-evolution' but also other strokes that deteriorate as a result of either cerebral or systemic causes during the 1st week[61].Whereas 'progressive stroke' is used in those conditions in which neurological worsening parallels the progression of ischemia.[62]

Early neurological deterioration (END) is defined as the clinical worsening or recurrence during the first 72 h after ischaemic stroke[63]. The consequences of END can be serious, with a poor short-term prognosis

Mechanisms of END include failure of development of collateral circulation in patients with critical stenosis or occlusion of a large vessel, either intra- or extra-cranial[64]; progression of thrombosis leading to increase in the ischaemic area[65]; early recurrence especially in atherothrombotic strokes [66]; the development of cerebral oedema[66] in patients with large strokes and finally haemorrhagic transformation in patients treated with fibrinolytic drugs[67].

Recent studies have shown that END is an independent predictor of poor outcomes in the setting of AIS. More specifically, the investigators of SORCan (Stroke Outcomes Research Canada) registry have reported that END (defined as 1-point decrease in CNS) was an independent predictor of 7-day, 30-day and 1-year case fatality rate in a cohort of 3631 patients [68] Similarly, END was associated with higher rates of death during hospitalization, longer duration of hospitalization and lower rates of functional independence in an Australian study [69]

MECHANISMS OF END

Several mechanisms have been proposed to explain END in acute ischaemic stroke. Advances in the brain and vascular imaging techniques have provided great insight into their role in END in acute stroke.

Failure of collaterals

Occlusion of major cerebral vessels is one of the most important independent predictors of END. The occlusion of vessel leads to compromise of perfusion distal to it.

Unless effective collateral circulation develops, the affected region is not salvaged from infarction. Development of collaterals appears to be the mechanism underlying transient ischaemic attacks[70]

Diabetic microangiopathy and chronic hypertension impair microvascular function and reduce the potential for collateral development. [71] This leads to reduced oxygen delivery and regional metabolic disturbances, which may aggravate cellular damage by enhancing brain oedema and free radical injury.[71,72] Failure of development of collaterals appear to be the most common mechanism for END[73].

Clot progression

In the past, END in acute ischaemic stroke had been attributed to clot progression,[74] though this concept is not proved. Recent studies of early MRI in acute stroke have shown large vessel occlusion and failure of collaterals rather than clot progression as the main mechanism of END.[73,75,76] Hypoperfusion due to occluded vessels may impair washout of distal emboli. This two mechanisms can act together to cause END.[70]

Recurrent stroke

Patients with acute ischaemic stroke are at a high risk of recurrent stroke in the first week.[77,78] However, most of the recurrent strokes detected on diffusion weighted MRI scans do not produce clinical deficit.[79]

Transcranial Doppler can detect microembolic signals and may be useful for identifying patients at risk of early recurrent stroke.[80]

Cerebral oedema

Raised intracranial pressure accounts for ,19% of cases of early deterioration in ischaemic stroke [81] The overall risk of cerebral odema with anterior circulation stroke is low and is estimated to be 10–20%.[82]

Clinical features such as a deteriorating level of consciousness, bilateral ptosis and involvement of the nondominant hemisphere may suggest a high risk of deterioration.

Lesion volume 145 ml on diffusion weighted imaging scan predict evolution to fatal cerebral oedema.[83]Cerebral oedema in ischaemic stroke tends to be cytotoxic and does not respond to osmotic diuretics.

Haemorrhagic transformation

Haemorrhagic transformation in ischaemic stroke is common and ranges from small asymptomatic petechiae to a large haematoma with pressure effects. Symptomatic transformation occurs only in 0.6% of patients treated with supportive care, whereas the incidence is higher in those treated with intravenous recombinant tissue plasminogen activator (rt-PA) (6%), [84-86] Only parenchymal haematoma type 2 (large haematoma .30% of ischaemic lesion volume) are considered to be associated with adverse outcome.[87]

Seizures

Seizures are common in large cortical ischaemic infarcts and may account for END in ,5% of patients with ischaemic strokes.[88] Seizures often cause only temporary worsening, though prolonged partial seizures can lead to persistent worsening.[89]

High serum glucose values

History of diabetes have been associated with END.[81] In a case–controlled study, previous history of diabetes along with elevated admission systolic blood pressure predicted END.[90] Persistent hyperglycaemia during the first 24 h after stroke independently predicted expansion of the volume of ischaemic infarct and poor neurological outcomes.[91]

END has potentially serious consequences on the short term (morbidity and death) and long term (recovery from stroke) outcomes for the patient. Therefore, attempts to prevent and treat END should be made promptly and aggressively. Recognition of the

predictors of early worsening may help in selecting patients for admission to the high dependency units equipped with intensive monitoring and treatment of these ill patients and prompt initiation of appropriate therapy.

DEHYDRATION AS A PREDICTOR OF END

Dehydration is a common phenomenon after stroke. Patients with stroke are often at increased risk of dehydration due to decreased oral intake of water as they have a reduced level of consciousness, are physically dependent, unable to communicate and have difficulties in swallowing[91].

In a large study, among acute ischemic stroke patients, 36 per cent of patients were dehydrated on the day of admission and 62 per cent were dehydrated at some point during their admission[92]

Some studies show an association between dehydration on presentation to hospital and END[93,94]. But some studies deny any such association[95]

The clinical assessment of dehydration is not always accurate especially in geriatric patients. Hence, biochemical parameters like plasma osmolality, BUN/Serum creatinine ratio and urine specific gravity have been used by various investigators for assessment of hydration status.There is no gold standard diagnostic test for measuring hydration status. But these markers have been suggested in the critical care and stroke literature as potential markers of volume contraction.[96,97] Additionally, these measures are standardized lab measures that are readily available and routinely obtained in the hospital setting.

The Blood Urea Nitrogen/ Serum Creatinine ratio has been used as an surrogate indicator of dehydration in many previous studies. [98-102] Patients with

BUN/Cr ratio ≥ 15 are considered as dehydrated and those patients with a BUN/Cr ratio < 15 were considered in the non-dehydrated group.

The samples used to determine the Bun/Creatinine ratio can be collected at the time of admission; therefore, dehydration status can assessed within 3 h of presentation to hospital.

It remains unclear whether initial hydration status influences mortality or functional recovery. To fill this gap in knowledge, this study was conducted to determine if dehydration is a risk factor for END in acute ischemic stroke patients.

SCALES USED TO ASSESS STROKE SEVERITY AND OUTCOME

A variety of stroke scales are available to assess various aspects related to stroke. The ideal scale would be easy and quick to administer, acceptable to patients and researchers, valid for its chosen purpose, reliable, and responsive to meaningful clinical change. There is no ideal stroke measure that fulfills all these criteria.

Various scales[103] available are:

Scales used in pre hospital assessment:

1.cincinnati stroke scale

2.los angeles pre hospital stroke screen (LAPSS)

Scales used for acute assessment of neurological impairment in EMD:

1. Canadian Neurological Scale (CNS)
2. European Stroke scale
3. Glasgow Coma Scale
4. National Institute of Health Stroke Scale (NIHSS)
5. Scandinavian Stroke Scale (SSS)

Scales used to assess global disability and functional outcome:

1. Modified rankin scale (mRS)
2. Stroke impact scale
3. Stroke specific quality of life scale (SS-QOL)

Scales used to assess outcome in stroke patients:

1. Barthel Index(BI)

2. American Heart association stroke outcome classification (AHA SOC)

3. Functional Independence Measurement

Among these, three scales namely, NIHSS, mRS, BI are most commonly used stroke scales worldwide[104,105].

As we are assessing stroke severity on day 1 and day 3 if our study to detect early neurological deterioration, NIHSS was chosen in our study.

National Institutes of Health Stroke Scale

The NIHSS is a 15-item scale that incorporates assessment of language, motor function, sensory loss, consciousness, visual fields, extraocular movements, coordination, neglect, and speech.

It quantifies the neurological impairment, paying particular attention to those aspects most pertinent to stroke.

It assigns numerical values to various aspects of neurological function. It is scored from 0 (no impairment) to a maximum of 42.

Scores of 21 or greater are usually described as “severe.” It is a validated scale with proven utility and suited to differing assessment scenarios.

The NIHSS has many advantages as a stroke outcome-assessment tool. It is relatively straightforward and takes around 6 minutes to perform, with no need for additional equipment.

NIHSS scores are reliable across observers, and this has been demonstrated both in cohorts of neurology-trained and non-neurologist raters[106]. The availability of a reliable method for neurological exam that is suitable for nonspecialists is a particular strength of the NIHSS.

METHODOLOGY

1. Source of data:

All the patients with Acute ischemic stroke enrolled to General Medicine OPD and Emergency department at R L Jalappa hospital, Kolar satisfying the inclusion criteria were enrolled for study.

Study Design:

Observational prospective study

Sample size calculation:

Sample size was estimated by using the proportion of patients with END among Acute Ischemic Stroke patients as 21.9% from a previous study[107] using the formula :

$$\text{Sample size} = Z_{1-\alpha/2}^2 p(1-p)/d^2$$

Here

Z = Standard normal variate

[at 5% type 1 error ($p<0.05$), it is 1.96 and at 1% type 1 error ($p<0.01$), it is 2.58].

As in majority of studies, p values are considered significant below 0.05, hence Z =1.96 is used in the formula.

p = Expected proportion in population based on previous studies or pilot studies.

Here p = 21.9 or 0.219 and q,(1-p) = 78.1 or 0.781.

d = Absolute error or precision which is decided by researcher.

d = 10% or 0.1

Using the above values at 99% Confidence level, a sample size of 114 subjects with acute ischemic stroke should be included in the study. Considering 10% nonresponse, a sample size of 114 + 11.4 ≈ 125 subjects were included in the study.

Inclusion criteria:

1. All the patients of Acute ischemic stroke who are more than 18 years of age.
2. Patients with first episode of acute ischemic stroke presenting within first 24 hours after onset of symptoms.

Exclusion criteria:

1. Patients with evidence of haemorrhagic stroke.
2. Patients with transient ischemic attack.
3. Patients with co-morbid conditions like congestive cardiac failure (CCF), renal failure and decompensated cirrhosis of liver.

Method of study:

The study was conducted among acute ischemic stroke patients presenting to department of General medicine, RLJH satisfying the inclusion criteria. A written informed consent was obtained from the patients or their relatives.

A detailed history was taken and a thorough general physical and systemic examination was performed. The following details were noted: age; sex; presenting complaints; history of any comorbidities and signs on examination.

Blood urea and serum creatinine of all the patients were estimated from the blood sample collected at the time of presentation. Blood Urea Nitrogen(BUN) were derived from blood urea level using the formula, BUN = Blood urea / 2.14. Hydration

status of the patients was assessed by calculating BUN/ serum creatinine ratio. BUN/ serum creatinine ratio more than 15 was considered as marker of dehydration.

The neurological status of the patients and the severity of stroke was assessed by using the NIHSS scoring system NIHSS (National Institute of Health Stroke Scale). NIHSS score was calculated immediately at the time of admission, then subsequently after 24 after onset of symptoms and on day 3 of admission. Patients for whom the NIHSS score returned to zero within the initial 24 h will be classified as having a transient ischemic attack (TIA) and were excluded from the study. Early Neurological Deterioration was diagnosed if there was an increase in the NIHSS score by 3 or more than 3 points from day 1 to day 3 of admission.

At the end of the study, the study population were divided into two groups based on their neurologic outcome. One group included acute ischemic stroke patients who developed END and second one included patients without END. In each group, proportion of patients with dehydration at the time of presentation was estimated. The correlation between dehydration at the time of presentation and development of END was assessed.

Severity of stroke based on NIHSS score:

score	**severity**
0	No stroke
1-15	Mild stroke
16-20	Moderate stroke
21-42	Severe stroke

STATISTICAL ANALYSIS

Descriptive analysis was carried out by mean and standard deviation for quantitative variables, frequency and proportion for categorical variables. Data was also represented using appropriate diagrams like bar diagram, pie diagram and cluster bar.

The association between categorical explanatory variables and quantitative outcome was assessed by comparing the mean values. The mean differences along with their 95% CI were presented. Independent sample t-test was used to assess statistical significance. The association between explanatory variables and categorical outcomes was assessed by cross tabulation and comparison of percentages. Univariate logistic regression was done to assess the factors associated with occurrence of END. Unadjusted odds ratios along with their 95% CI were presented. Factors showing statistical significance in univariate analysis were included in the multivariate analysis. Adjusted odds ratios along with 95% CI and p-values were presented.

P value < 0.05 was considered statistically significant. IBM SPSS version 22 was used for statistical analysis.[108]

OBSERVATIONS AND RESULTS

The present study was carried out over a month of 15 months from March 2016 to May 2017 in the Department of General Medicine, Sri Devraj Urs Medical College, Kolar, Karnataka. A total of 125 subjects were included in the study.

DEMOGRAPHIC PROFILE:

AGE DISTRIBUTION:

The mean age of the study population was 62.54 ± 5.55 years with the range 46 to 75 years. (Table 1)

Table 1: Descriptive analysis for age in study population (N= 125)

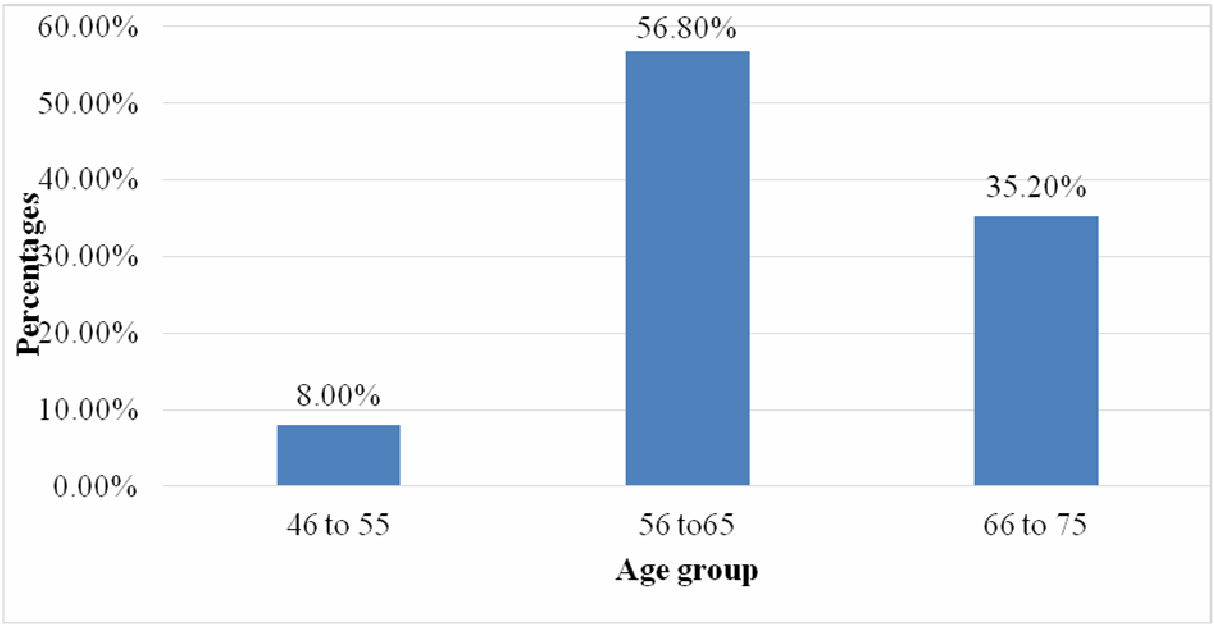
Parameter	Mean ±STD	Median	Min	Max	95% C.I. for EXP(B)	
					Lower	Upper
Age	62.54 ± 5.55	63.00	46.00	75.00	61.56	63.53

Among the study population, 10 (8%) were in the age group of 46 to 55 years, 71(56.80%) were in the age group of 56 to 65 and 44 (35.20%) were in the age group of 66 to 75years. (Table 2&figure5)

Table 2: Descriptive analysis of age group in study population (N=125)

Age group	Frequency	Percentages
46 to 55	10	8.00%
56 to65	71	56.80%
66 to 75	44	35.20%

Figure 5: Bar diagram showing study population in various age groups (N=125)



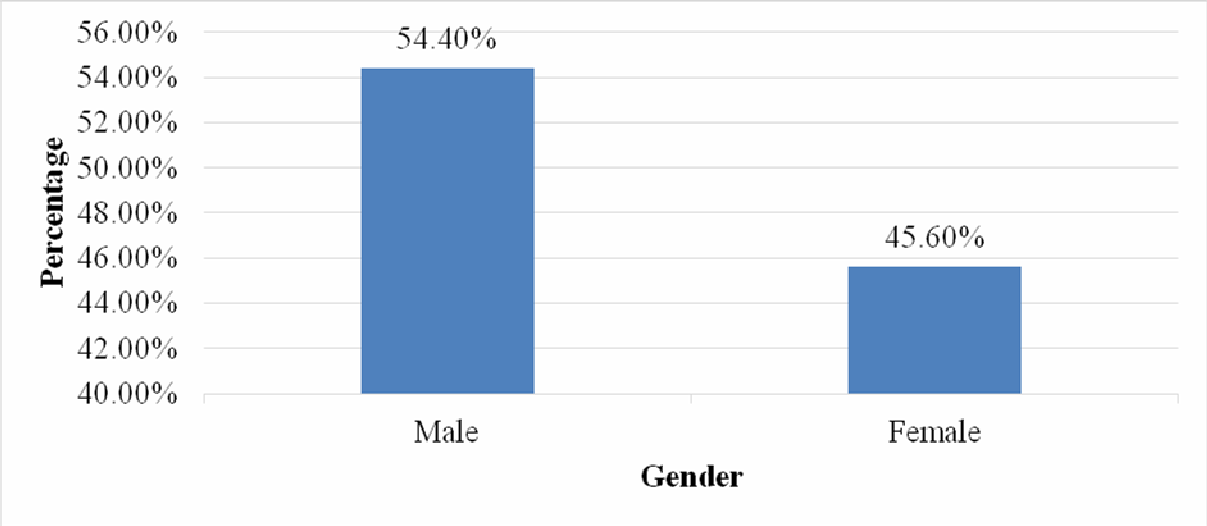
GENDER DISTIBUTION:

Table 3: Descriptive analysis of gender in study population (N=125)

Gender	Frequency	Percentage
Male	68	54.40%
Female	57	45.60%

Among the study population, 68(54.40%) were males and 57(45.60%) constitute females. (Table 3& figure 6)

Figure 6 : Bar chart of gender in study population (N=125)



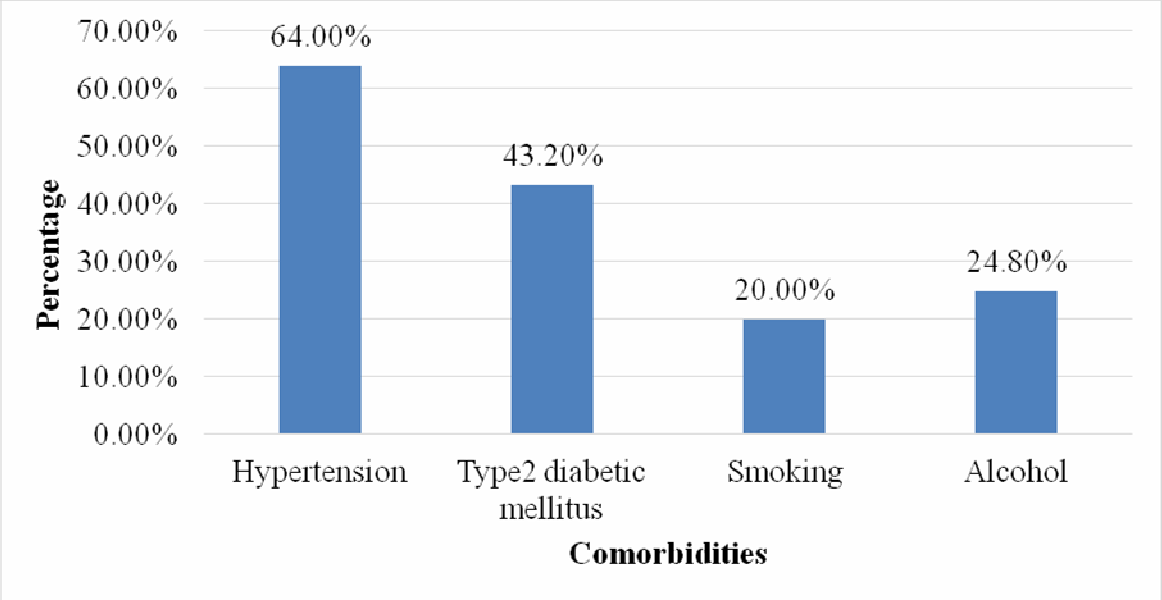
COMORBIDITIES AMONG STUDY POPULATION

Table 4: Descriptive analysis of comorbidities in study population (N=125)

Comorbidities	Frequency	Percentage
Hypertension	80	64.00%
Type2 diabetes mellitus	54	43.20%
Smoking	25	20.00%
Alcohol	31	24.80%

Among the study population, 80(64%) had hypertension, 54 (43.2%) have type 2 diabetes mellitus. Smoking and alcohol consumption was present in 25(20%) and 31(24.80%) respectively. (Table 3%& figure 7)

Figure 7 : Bar chart of comorbidities in study population (N=125)



BLOOD PRESSURE VALUES AMONG STUDY POPULATION:

Table 5: Descriptive analysis for vital signs in study population (N=125)

BLOOD PRESSURE (mm Hg)	**Mean ±STD**	**Median**	**Min**	**Max**	**95% C.I. for EXP(B)**	
					Lower	**Upper**
Systolic blood pressure	141.42 ± 14.4	142.00	110.00	170.00	138.86	143.98
Diastolic blood pressure	81.86 ± 8.82	80.00	62.00	100.00	80.29	83.43

The mean systolic and Diastolic blood pressures among study population was 141.42± 14.4 **mm Hg** and 81.86 ± 8.82 **mm Hg.**

Table 6: Descriptive analysis for Random blood sugar in study population (N=125)

Random Blood Sugar (mg/dl)	**Mean ±STD**	**Median**	**Min**	**Max**	**95% C.I. for EXP(B)**	
					Lower	**Upper**
Random blood sugar	135.86 ± 36.99	126.00	90.00	383.00	129.31	142.40

The mean RBS value among study population was135.86 ± 36.99 **mg/dl.**

RENAL FUNCTION TESTS AND HYDRATION STATUS:

Table 7: Descriptive analysis for renal_function testst in study population (N=125)

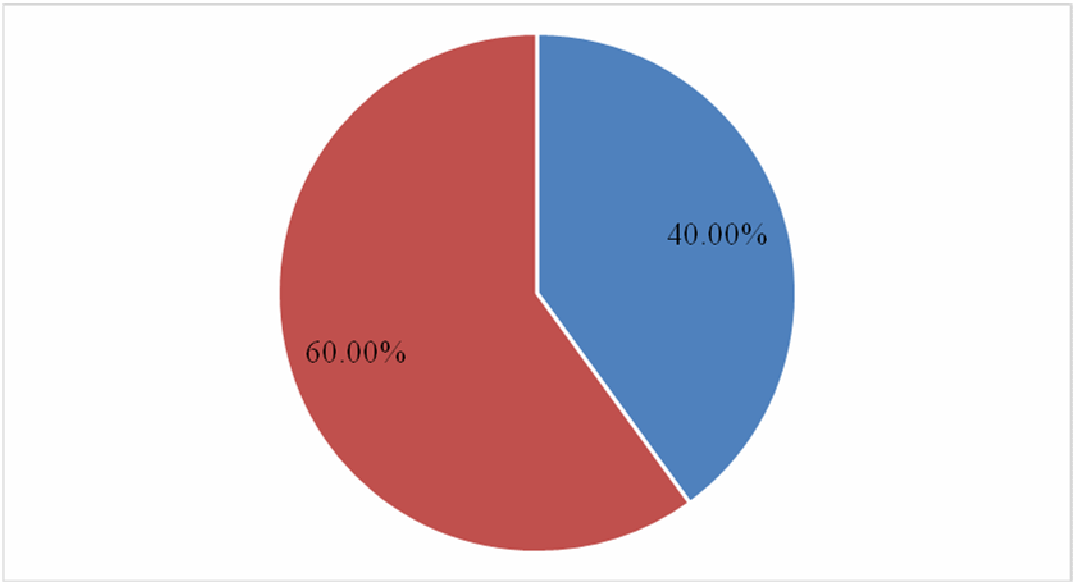
Renal_function	Mean ±STD	Median	Min	Max	95% C.I. for EXP(B)	
					Lower	Upper
Blood urea (mg/dl)	28.88 ± 11.04	27.00	11.00	72.00	26.93	30.84
Blood urea nitrogen (mg/dl)	13.5 ± 5.19	12.80	5.30	34.00	12.58	14.42
S. creatinine (mg/dl)	0.86 ± 0.23	0.90	0.50	1.30	0.82	0.90
BUN/ S. creatinine ratio	15.63 ± 4.13	14.00	10.00	26.00	14.90	16.36

The mean blood urea nitrogen was13.5 ± 5.19 mg/dl, mean Serum creatinine was 0.86 ± 0.23 mg/dl and mean BUN/S. creatinine was 15.63 ± 4.13 among study population.

Table 8: Descriptive analysis of hydration status in study population (N=125)

Hydration status	Frequency	Percentage
No. of patients dehydrated on presentation	50	40.00%
No. of patients not dehydrated on presentation	75	60.00%

Figure 8: Pie chart of hydration status in study population (N=125)



40% of study population i.e., 50 patients were in dehydrated state on presentation and 60%, 75 patients are not dehydrated on presentation to hospital.

Table 9: Descriptive analysis for NIHSS Score on day 1 and 3 in study population (N=125):

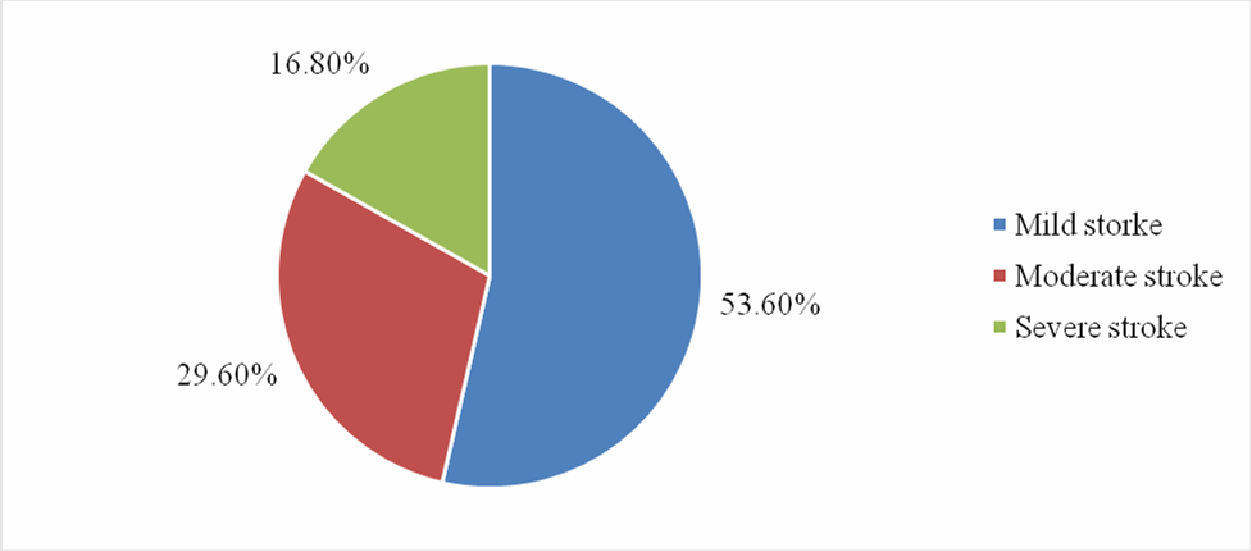
Parameter	Mean ±STD	Median	Min	Max	95% C.I. for EXP(B)	
					Lower	Upper
NIH Stroke score day1	16.52 ± 5.58	15.00	8.00	40.00	15.53	17.51
NIH Stroke score day 3	18.42 ± 6.35	17.00	8.00	40.00	17.29	19.54

The mean NIHSS score on day 1 was 16.52± 5.58 and on day 3 was 18.42 ± 6.35.

Table 10: Descriptive analysis of severity of stroke in study population (N=125) based on NIHSS score on day 1 :

NIH stroke score group	Frequency	Percentages
Mild stroke	67	53.60%
Moderate – severe stroke	37	29.60%
Severe stroke	21	16.80%

Figure 9 : Pie chart of stroke severity in study population (N=125)

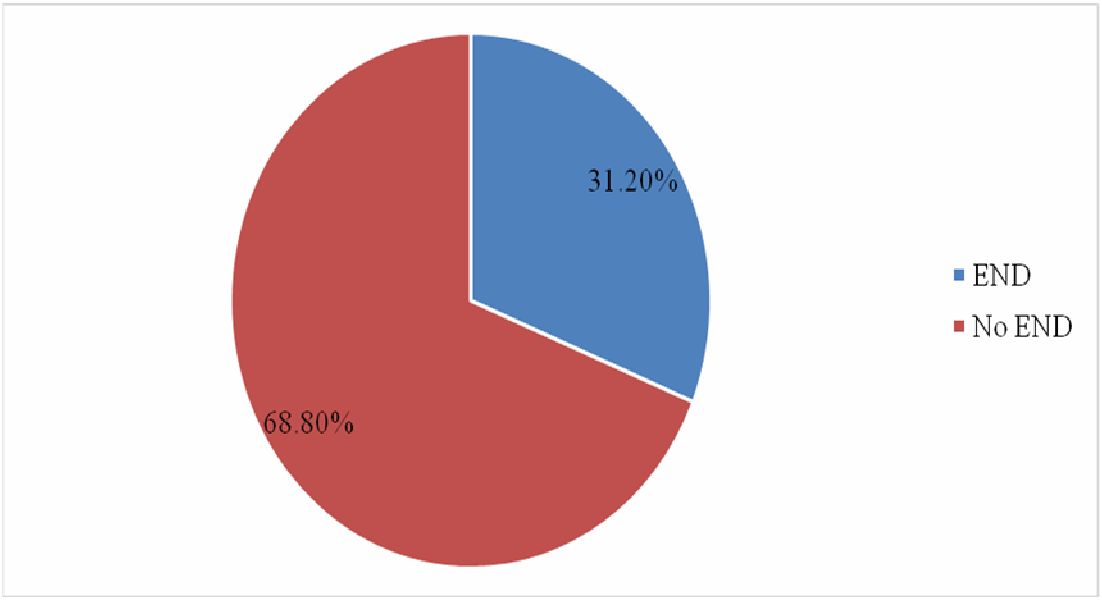


Based on NIHSS score on day 1 of admission, majority of the study population (53.6%) had mild stroke, 29.6% had moderate to severe stroke and 16.8 % had severe stroke.

Table 11: Descriptive analysis of early neurologic deterioration in study population (N=125)

Early Neurologic Deterioration (END)	Frequency	Percentage
Patients who developed END	39	31.2%
Patients who didn't develop END	86	68.8%

Figure 10: Pie chart of END in study population (N=125)



Among the total study population, early neurologic deterioration was developed in 39(31.2%) patients and 86 (68.8%) patients did not develop arly neurologic deterioration.

Table 12: Comparison of mean age between 2 groups (with and without END)

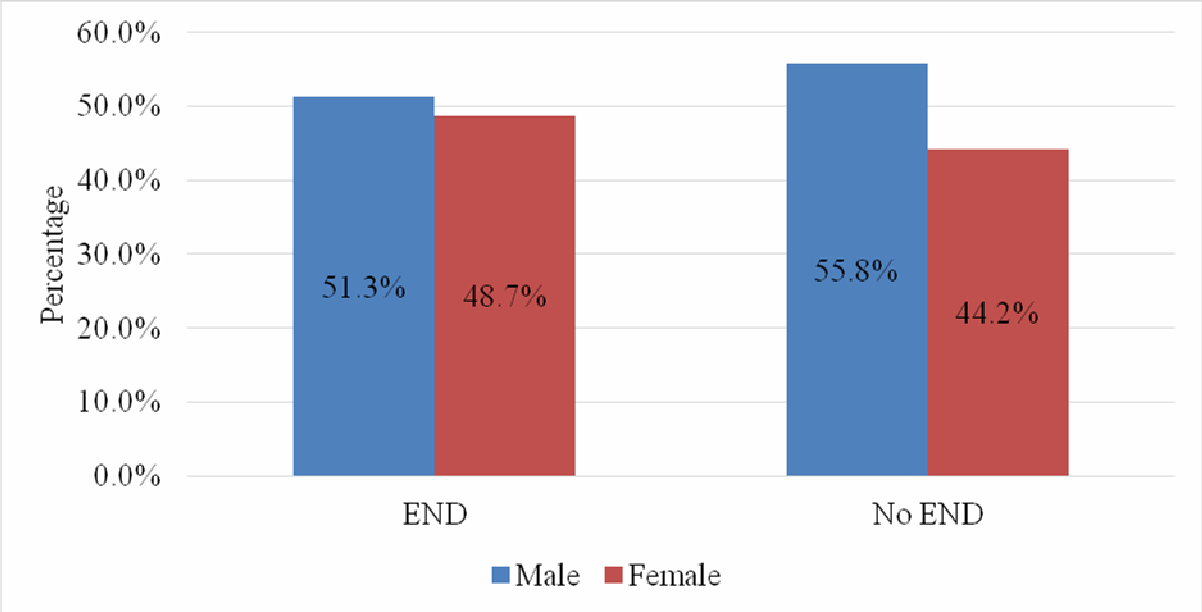
Early neurologic deterioration	Age Mean± STD	Mean difference	95% CI		P value
			Lower	Upper	
END	63 ± 5.87	0.66	1.46	2.79	.539
No END	62.34 ± 5.43				

The mean age of group with END was 63 ± 5.87 years and that of group with out END was 62.34 ± 5.43 years with p value of 0.539 which is not significant.

Table 13: Comparison of early neurologic deterioration with gender of study population (N=125)

Gender	Early Neurologic Deterioration		Chi square	P-value
	END(N=39)	No END(N=86)		
Male	20 (51.28%)	48 (55.81%)	0.222	0.64
Female	19 (48.71%)	38 (44.18%)		

Figure 11: Bar chart of early neurologic deterioration with gender in study population (N=125)

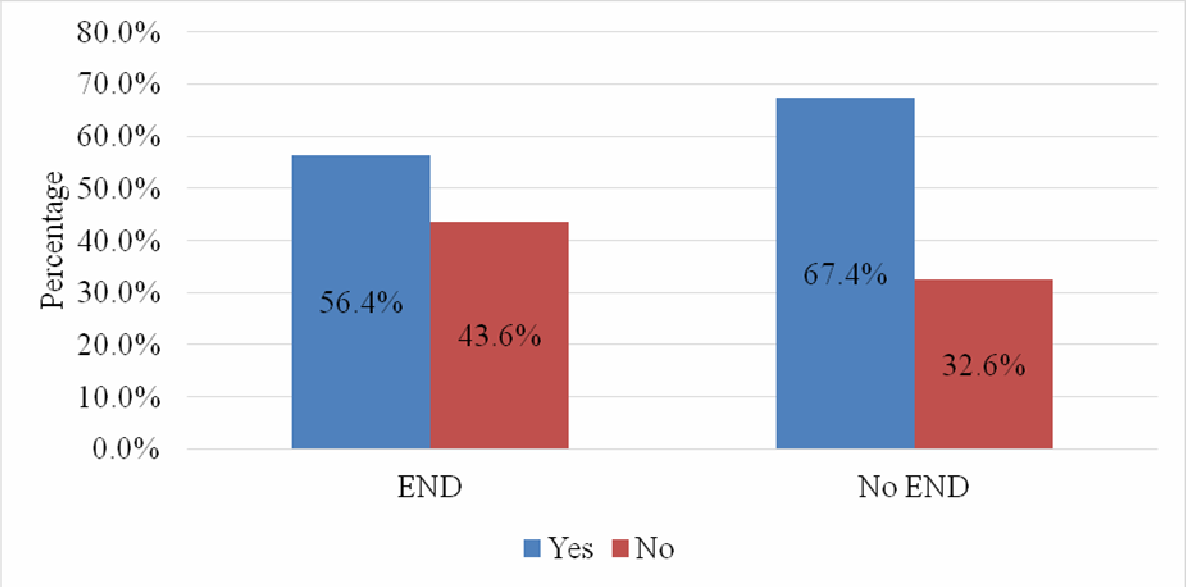


The percentages of males and females in group with END were 51.28% and 48.71% with p value of 0.64, which was not significant.

Table 14: Comparison of early neurologic deterioration with hypertension of study population (N=125)

Hypertension	Early Neurologic Deterioration		Chi square	P-value
	END(N=39)	No END(N=86)		
Yes	22 (56.41%)	58 (67.44%)	1.417	0.23
No	17 (43.58%)	28 (32.55%)		

Figure 12: Bar chart of early neurologic deterioration with hypertension in study population (N=125)

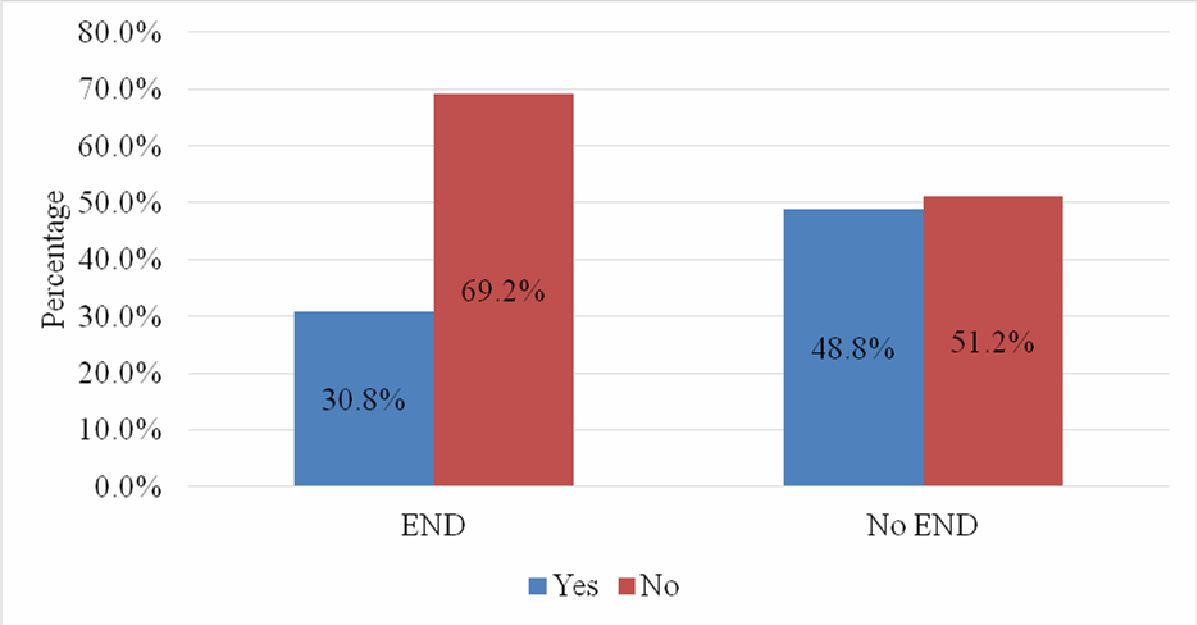


The percentages of hypertensives is 56.41% and 67.44% among patients with and without END respectively with p value of 0.23, which was not significant.

Table 15: Comparison of early neurologic deterioration with type 2 diabetes mellitus of study population (N=125)

Type 2 diabetes mellitus	Early Neurologic Deterioration		Chi square	P-value
	END(N=39)	No END(N=86)		
Yes	12 (30.76%)	42 (48.83%)	3.570	0.06
No	27 (69.23%)	44 (51.16%)		

Figure 13: Bar chart of early neurologic deterioration with type 2 diabetes mellitus in study population (N=125)

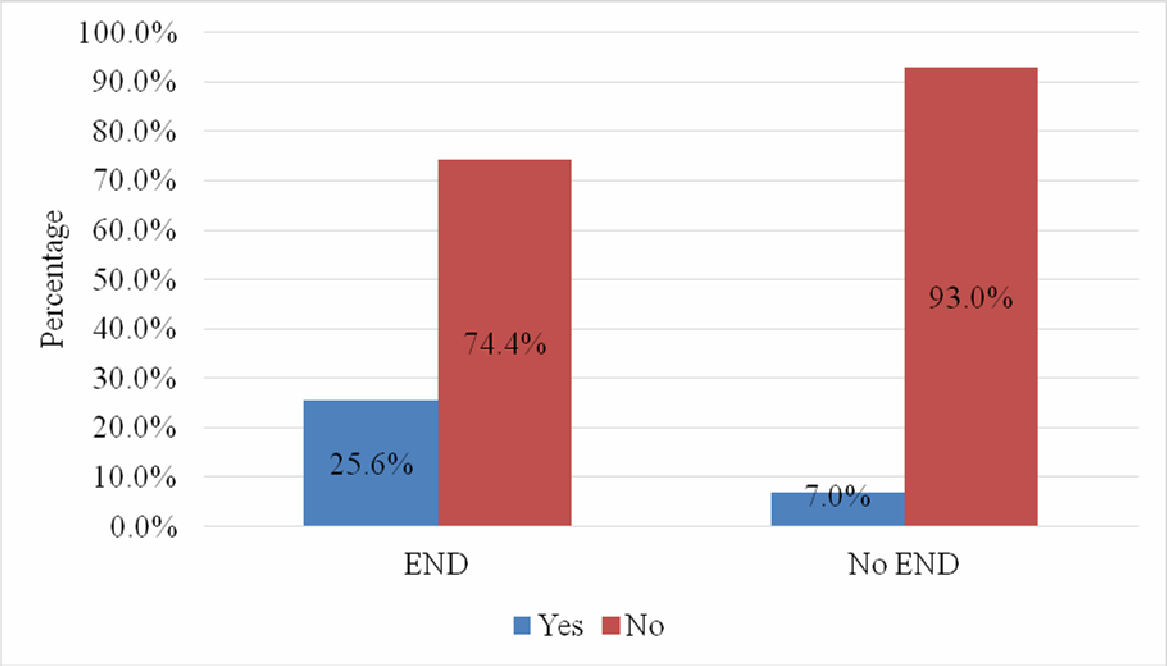


The percentages of diabetic patients was 30.76% and 48.83% among patients with and without END respectively with p value of 0.06, which was not significant.

Table 16: Comparison of early neurologic deterioration with smoking of study population (N=125)

Smoking	Early Neurologic Deterioration		Chi square	P-value
	END(N=39)	No END(N=86)		
Yes	10 (25.64%)	6 (6.976%)	8.374	***0.004***
No	29 (74.35%)	80 (93.02%)		

Figure 14: Bar chart of early neurologic deterioration with smoking in study population (N=125)



The percentages of patients with smoking history was 25.6 % and 7% among patients with and without END respectively with p value of 0.004, which was statistically significant.

Table 17: Comparison of early neurologic deterioration with alcohol of study population (N=125)

Alcohol	Early Neurologic Deterioration		Chi square	P-value
	END(N=39)	No END(N=86)		
Yes	15 (38.46%)	26 (28.60%)	5.673	***0.23***
No	24 (61.53%)	60 (71.39%)		

The percentages of patients with history of alcohol consumption was 38.46% and 28.6% among patients with and without END respectively with p value of 0.23, which was not statistically significant.

Figure 15: Bar chart of early neurologic deterioration with alcohol in study population (N=125)

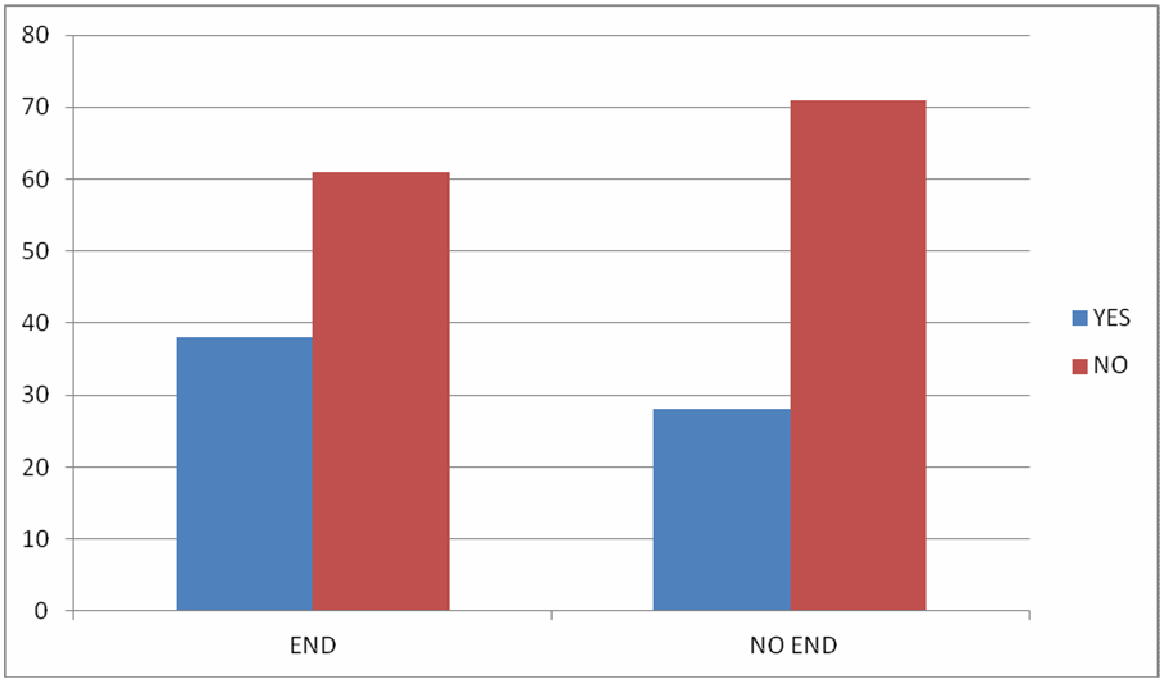


Table 18: Comparison of mean systolic blood pressure between study groups (N=125)

Early neurologic deterioration	systolic blood pressure Mean± STD	Mean difference	95% CI		P value
			Lower	Upper	
END	140.77 ±16.13	1.00	4.51	6.50	.720
No END	141.77 ±13.56				

Table 19: Comparison of mean diastolic blood pressure between study groups (N=124)

Early neurologic deterioration	Diastolic blood pressure Mean± STD	Mean difference	95% CI		P value
			Lower	Upper	
END	79.95 ± 8.02	2.79	0.56	6.15	0.102
No END	82.74 ± 9.08				

Table 20: Comparison of mean random blood sugar between study groups (N=125)

Early neurologic deterioration	Random blood sugar Mean± STD	Mean difference	95% CI		P value
			Lower	Upper	
END	150.72 ±49.77	21.60	7.94	35.26	***0.002***
No END	129.12 ±27.22				

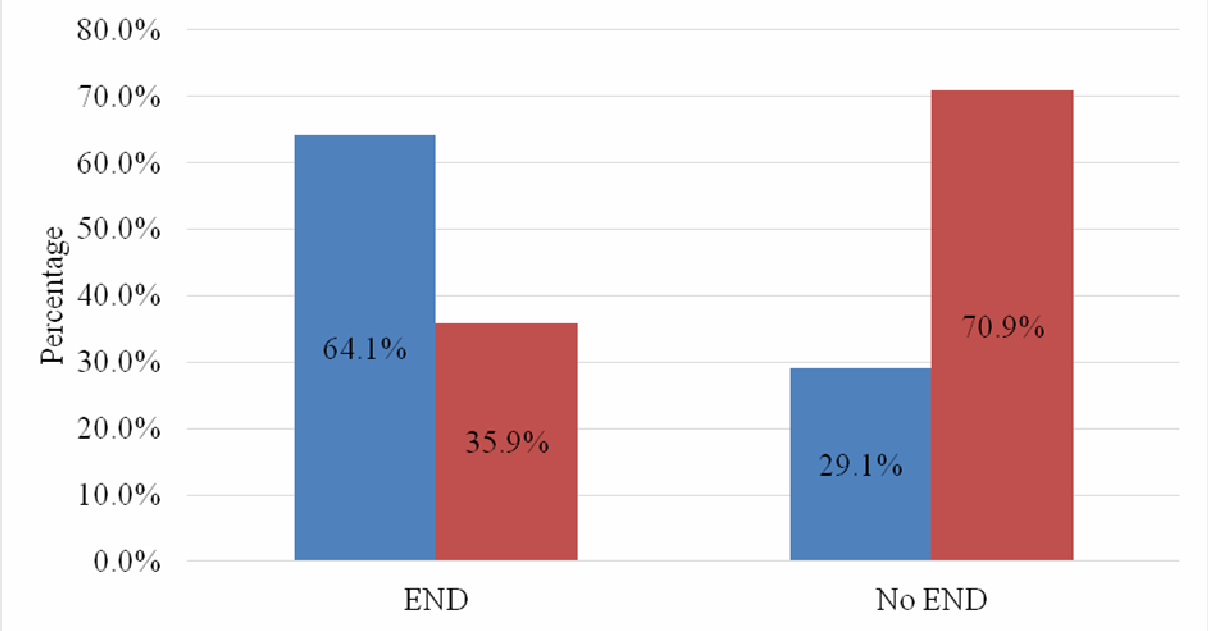
The difference in systolic and diastolic blood pressure was not significant in patients with and without END (p vlue:0.72, p value:0.102).

But there was statistically significant difference between blood sugar levels on presentation between 2 groups with p value of 0.002.

Table 21: Comparison of early neurologic deterioration with hydration status of study population (N=125)

Hydration status	Early Neurologic Deterioration		Chi square	P-value
	END(N=39)	No END(N=86)		
Dehydration present	25 (64.10%)	25 (29.06%)	13.72	*<0.001*
No dehydration	14 (35.89%)	61 (70.93%)		

Figure 16: Bar chart of early neurologic deterioration with hydration status in study population (N=125)

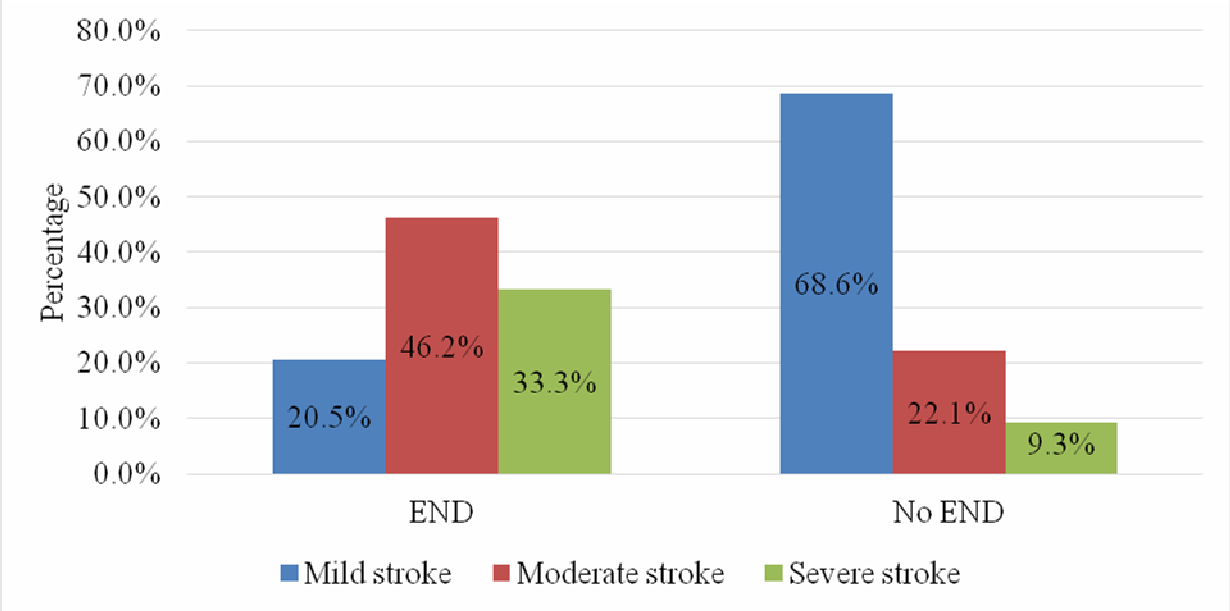


Among patients without END, 29.06% i.e, 25 had dehydration on presentation whereas among patients who developed END, out of 39, 25 were dehydrated on presentation who constitute 64.1 %. P value for dehydration was <0.001which shows the difference as statistically significant.

Table 22: Comparison of early neurologic deterioration with NIH stroke score group of study population (N=125)

NIH stroke score group	Early Neurologic Deterioration		Chi square	P-value
	END(N=39)	No END(N=86)		
Mild stroke	8 (20.51%)	59 (68.60%)	26.04	***<0.001***
Moderate stroke	18 (46.15%)	19 (22.09%)		
Severe stroke	13 (33.33%)	8 (9.302%)		

Figure 17: Bar chart of early neurologic deterioration with NIH stroke score in study population (N=125)



Percentage of patients with moderate and severe stroke is more among patients with END whereas percentage of patients with mild stroke are more in patients without END with p value of <0.001, which was statistically significant**.**

Univariate logistic regression analysis was applied to age, gender, smoking history, Random blood sugar levels, dehydration status and stroke severity.

Table23: Univariate logistic regression analysis of factors associated with END in study population(N=125)

Factors	Unadjusted Odds ratio	95%CI		P value
		Lower	Upper	
Age	1.022	0.954	1.096	0. 535
Gender(baseline =male)	1.200	0.562	2.562	0.638
Smoking(base line=No)	4.598	1.534	13.780	0.006
Random blood sugar	1.018	1.005	1.030	0.007
Dehydration (base line=No)	4.357	1.952	9.725	<0.001
Stroke severity (baseline= mild)				
Moderate	6.987	2.622	18.621	<0.001
severe	11.984	3.797	37.824	<0.001

The factors which have shown statistically significant association with END in univariate analysis were presence of smoking history, random blood sugar, dehydration status and stroke severity on presentation. Age and gender had no statistically significant association with END in the study.(Table 23)

Table24: Multivariate logistic regression analysis of factors associated with occurrence of END in study population(N=125)

Factors	Adjusted Odds ratio	95%CI		P value
		Lower	Upper	
Age	1.467	1.201	1.793	<0.001
Gender(baseline =male)	2.590	0.585	11.459	0.210
Smoking(base line=No)	42.629	4.185	434.252	0.002
Alcohol(baseline=No)	3.956	0.806	19.406	0.090
Red blood sugar	1.024	1.001	1.047	0.036
Hydration status(base line=No)	9.077	2.116	38.928	0.003
NIH stroke score (bassline=mild)				
Moderate	152.230	17.351	1335.586	<0.001
severe	278.823	20.286	3832.258	<0.001

After controlling for all the other factors in the multivariate analysis, with each 1-year increase in age the odds of END have increased 1.467 times (95% CI 1.201 to 1.793 p value<0.001). The odds of END were 42.629 times more in people with presence of smoking (odds ratio 42.629 95% CI 4.185 to 434.252) which was statistically significant. The odds of END are 1.024 times more with each 1unit increase in red blood sugar (odds ratio 1.024,95% CI 1.001 to 1.047), which was statistically significant. The odds of END were 9.077 times more in people with presence of hydration status (odds ratio 9.077 (95% CI 2.116 to 38.928), which was statistically significant. (Table 24)

DISCUSSION

This study was aimed at evaluating the association of dehydration with early neurological deterioration in acute ischemic stroke patients. BUN/Creatinine ratio >15 was taken as surrogate marker for dehydration. Stroke severity was determined based on clinical examination (NIHSS score).The primary outcome of interest was increase in NIHSS score by 3 or more than 3 from day 1 to day 3 of admission. .

In the study, the mean age of the study population was 62.54 ± 5.55 years with the range 46 to 75 years. Among the study population, 10 (8%) were in the age group of 46 to 55 years, 71(56.80%) were in the age group of 56 to 65 and 44 (35.20%) were in the age group of 66 to 75 years. The maximum people are in the age group of 46 to 55 years in our study.

Among the study population, both males and females are almost equal in number. 68 (54.40%) were males and 57(45.60%) constitute females.

Among the study population, 80(64%) had hypertension, 54 (43.2%) have type 2 diabetes mellitus. Smoking and alcohol consumption was present in 25(20%) and 31(24.80%) respectively. A study done by Basu et al from India assessed various risk factors for stroke in 40 patients and found that 87.5% were hypertensives and 35% had diabetes[109]. Study by Wu et al from China assessed various risk factors for stroke and found 66.3% were hypertensive, 31.5% were diabetic and 30.5% were smokers[110].

The mean Systolic and Diastolic blood pressures among study population was 141.42± 14.4 mm Hg and 81.86 ± 8.82 mm Hg respectively. The mean blood sugar value among study population was135.86 ± 36.99 mg/dl.

The mean blood urea nitrogen was13.5 ± 5.19 mg/dl, mean Serum creatinine was 0.86 ± 0.23 mg/dl and mean BUN/S. creatinine was 15.63 ± 4.13 among study population. 40% of study population i.e., 50 patients were in dehydrated state on presentation and 60%, 75 patients are not dehydrated on presentation to hospital.

The mean NIHSS score on day 1 was 16.52± 5.58 and on day 3 was 18.42 ± 6.35.

Based on NIHSS score on day 1 of admission, majority of the study population (53.6%) had mild stroke, 29.6% had moderate to severe stroke and 16.8 % had severe stroke.

Among the total study population, early neurologic deterioration was developed in 39(31.2%) patients and 86 (68.8%) patients did not develop early neurologic deterioration. This finding is similar to that of previously published studies[111-113]

In the study done by Kunal Bhatia, out of the 114 stroke patients enrolled in the study, END was observed in 25 (21.9%) patients[107].

The mean age of group with END was 63 ± 5.87 years and that of group without END was 62.34 ± 5.43 years with p value of 0.539 which is not significant.

The percentages of males and females in group with END were 51.28% and 48.71% with p value of 0.64, which was not significant.

The percentages of hypertensives is 56.41% and 67.44% among patients with and without END respectively with p value of 0.23, which was not significant.

The percentages of diabetic patients was 30.76% and 48.83% among patients with and without END respectively with p value of 0.06, which was not significant.

The percentages of patients with smoking history was 25.6 % and 7% among patients with and without END respectively with p value of 0.004, which was statistically significant.

The percentages of patients with history of alcohol consumption was 38.46% and 28.6% among patients with and without END respectively with p value of 0.23, which was not statistically significant.

The difference in systolic and diastolic blood pressure was not significant in patients with and without END (p vlue:0.72, p value:0.102).

This findings are similar to study done by Jorgensen HS et al. Their study showed statistically significant difference was present between blood sugar levels on presentation between group with and without END but the difference in systolic and diastolic blood pressure was not significant in patients with and without END [114].

The detrimental effects of hyperglycaemia have been attributed to tissue acidosis secondary to anaerobic glycolysis, lactic acidosis, free radical production, disruption of the blood–brain barrier, the development of brain oedema, and increased risk of hemorrhagic transformation.

The independent role of hypertension as a predictor of END has not been established.

The current stroke guidelines, therefore, do not advise treatment of hypertension in acute stroke, except when thrombolysis is contemplated or in the presence of extremely severe hypertension.

Percentage of patients with moderate and severe stroke is more among patients with END whereas percentage of patients with mild stroke are more in patients without END with p value of <0.001, which was statistically significant. This finding is similar to other studies done on predictors of END[115]

Among patients without END, 29.06% i.e, 25 had dehydration on presentation whereas among patients who developed END, out of 39, 25 were dehydrated on presentation who constitute 64.1 %. p value for dehydration was <0.001which shows the difference as statistically significant. A study done by Bahouth et al also showed that dehydration was associated with deterioration of hemispatial neglect in the ischemic stroke patients[116]. A study done by Schrock et al concluded that an[117]

elevated BUN/Cr ratio in patients with AIS is associated with poor outcome at 30 days. Further study is needed to see if acutely addressing hydration status in ED patients with AIS can alter outcome. Study by Liu CH et al showed that admission dehydration is associated with worse discharge outcomes and higher admission costs in acute ischaemic stroke but not in hemorrhagic stroke.

Univariate logistic regression analysis was applied to age, gender, smoking history, Random blood sugar levels, dehydration status and stroke severity.

The factors which have shown statistically significant association with END in univariate analysis were presence of smoking history, random blood sugar, dehydration status and stroke severity on presentation. Age and gender had no statistically significant association with END in the study.

After controlling for all the other factors in the multivariate analysis, with each 1-year increase in age the odds of END have increased 1.467 times (95% CI 1.201 to 1.793 p value<0.001). The odds of END were 42.629 times more in people with presence of smoking (odds ratio 42.629 95% CI 4.185 to 434.252) which was statistically significant. The odds of END are 1.024 times more with each 1unit increase in red blood sugar (odds ratio 1.024,95% CI 1.001 to 1.047), which was statistically significant. The odds of END were 9.077 times more in people with presence of hydration status (odds ratio 9.077 (95% CI 2.116 to 38.928), which was statistically significant.

LIMITATIONS AND MERITS OF THE STUDY:

One of the limitations of the study was a smaller sample size of 125 patients. Another limitation of this study is the lack of “gold standard” measure of dehydration. Although the BUN/Cr ratio is commonly used to assess hydration status, it is not a specific measure.

The merit of this study is the clinical relevance of its results. BUN/creatinine ratio is easy and inexpensive to measure and can be performed in any emergency department to assess the hydration status of the patient.

CONCLUSION

In this study early neurological deterioration has occurred around one third of acute ischemic stroke patients presented to the hospital. Among patients who developed END, 64.1% were dehydrated. There was significant difference in the hydration status between patients with and with out END. The patients who are dehydrated are 9.07 times at risk of developing early neurologic deterioration compared tpyo who are not dehydrated.

Dehydration being a treatable condition, the use of BUN/creatinine >15 as a marker of relative dehydration, can be helpful in detecting patients with dehydration early and thus may play a role in preventing neurological worsening.

SUMMARY

- Stroke is a major cause of long-term disability among patients and has enormous emotional and socio-economic consequences.
- Early Neurological Deterioration (END) has potentially serious consequences on the short term (morbidity and death) and long term (recovery from stroke) outcomes for the patients of acute ischemic stroke.
- Therefore, attempts to predict and prevent END should be made promptly and aggressively.
- The role of dehydration status of patient at the time of presentation as a risk factor for END is not yet proved and studies on the effect of dehydration on stroke outcome are limited.
- So the present study was designed to evaluate the effect of dehydration on early neurological deterioration in patients of acute ischemic stroke.
- 125 patients with Acute ischemic stroke presenting to General Medicine OPD and Emergency department at R L Jalappa hospital, Kolar were enrolled in the study.
- The neurological status of the patients and the severity of stroke were assessed by applying the NIHSS (National Institute of Health Stroke Scale) score on day 1 and day 3 on all the patients.
- Early Neurological Deterioration was diagnosed if there was an increase in the NIHSS score by 3 or more than 3 points from day 1 to day 3 of admission.
- Hydration status of the patients was assessed by calculating BUN/ serum creatinine ratio. BUN/ serum creatinine ratio more than 15 was be considered as marker of dehydration.
- The hydration status was correlated with Early neurologic deterioration.

- In the study, the mean age of the study population was 62.54 ± 5.55 years with the range 46 to 75 years. Among the study population, 10 (8%) were in the age group of 46 to 55 years, 71(56.80%) were in the age group of 56 to 65 and 44 (35.20%) were in the age group of 66 to 75 years. 68 (54.40%) were males and 57(45.60%) constitute females.
- Among the study population, 80(64%) had hypertension, 54 (43.2%) have type 2 diabetes mellitus. Smoking and alcohol consumption was present in 25(20%) and 31(24.80%) respectively.
- The mean Systolic and Diastolic blood pressures among study population was 141.42± 14.4 mm Hg and 81.86 ± 8.82 mm Hg respectively.The mean blood sugar value among study population was135.86 ± 36.99 mg/dl.
- The mean blood urea nitrogen was13.5 ± 5.19 mg/dl, mean Serum creatinine was 0.86 ± 0.23 mg/dl and mean BUN/S. creatinine was 15.63 ± 4.13 among study population.
- 40% of study population i.e., 50 patients were in dehydrated state on presentation and 60%, 75 patients are not dehydrated on presentation to hospital.
- The mean NIHSS score on day 1 was 16.52± 5.58 and on day 3 was 18.42 ± 6.35.
- Based on NIHSS score on day 1 of admission, majority of the study population (53.6%) had mild stroke, 29.6% had moderate to severe stroke and 16.8 % had severe stroke.
- Among the total study population, early neurologic deterioration was developed in 39(31.2%) patients and 86 (68.8%) patients did not develop early neurologic deterioration.
- The mean age of group with END was 63 ± 5.87 years and that of group without END was 62.34 ± 5.43 years with p value of 0.539 which is not significant.

- The percentages of males and females in group with END were 51.28% and 48.71% with p value of 0.64, which was not significant.
- The percentages of hypertensives is 56.41% and 67.44% among patients with and without END respectively with p value of 0.23, which was not significant.
- The percentages of diabetic patients was 30.76% and 48.83% among patients with and without END respectively with p value of 0.06, which was not significant.
- The percentages of patients with smoking history was 25.6 % and 7% among patients with and without END respectively with p value of 0.004, which was statistically significant.
- The percentages of patients with history of alcohol consumption was 38.46% and 28.6% among patients with and without END respectively with p value of 0.23, which was not statistically significant.
- The difference in systolic and diastolic blood pressure was not significant in patients with and without END (p vlue:0.72, p value:0.102).
- Among patients without END, 29.06% i.e, 25 had dehydration on presentation whereas among patients who developed END, out of 39, 25 were dehydrated on presentation who constitute 64.1 %. p value for dehydration was <0.001which shows the difference as statistically significant.
- After controlling for all the other factors in the multivariate analysis, with each 1-year increase in age the odds of END have increased 1.467 times (95% CI 1.201 to 1.793 p value<0.001).
- The odds of END were 42.629 times more in people with presence of smoking (odds ratio 42.629 95% CI 4.185 to 434.252) which was statistically significant.
- The odds of END are 1.024 times more with each 1unit increase in red blood sugar (odds ratio 1.024,95% CI 1.001 to 1.047), which was statistically significant.

- The odds of END were 9.077 times more in people with presence of hydration status (odds ratio 9.077 (95% CI 2.116 to 38.928), which was statistically significant.
- Our study suggests that dehydration can be a risk factor for developing early neurologic deterioration in acute ischemic stroke patients.

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PROFORMA

Sl. No: **Date:**

OP/ IP No:

Name:

Age:

Occupation:

Address:

CHIEF COMPLAINTS:

HISTORY OF PRESENTING ILLNESS:

PAST MEDICAL HISTORY:

FAMILY HISTORY :

PERSONAL HISTORY :

DRUG HISTORY :

PHYSICAL EXAMINATION :

GENERAL PHYSICAL EXAMINATION:

Any signs of dehydration :

1. **Mucous membranes: normal/ dry/parched**
2. **Skin turgor : normal/slow/tenting**
3. **Capillary refill : <2 sec/2 sec/ 2-4sec/>4 sec (cool limbs)**

VITAL DATA :

SYSTEMIC EXAMINATION:

- **CENTRAL NERVOUS SYSTEM :**

Level of consciousness:

Cranial Nerves examination:

Motor system examination:

Sensory system examination:

NIHSS SCORE:

On day 1:

After 24 hrs of onset of Symptoms:

On day 3:

Is there decrease in NIHSS score from day 1 to day 3 ?

Yes No

If yes, by how much the score decreased?

- CARDIOVASCULAR SYSTEM:
- RESPIRATORY SYSTEM:
- GASTROINTESTINAL SYSTEM:

INVESTIGATIONS:

1. NCCT Brain Report :
2. Blood urea :
3. Serum creatinine :

Blood Urea Nitrogen (blood urea / 2.14) :

BUN / Serum Creatine ratio :

PROVISIONAL DIAGNOSIS:

CONSENT FORM

Study title: **A STUDY OF THE EFFECT OF DEHYDRATION ON EARLY NEUROLOGICAL DETERIORATION IN ACUTE ISCHEMIC STROKE**

PG guide's name: **Dr Srinivasa S.V.**
Principal investigator: **Dr Modugula S Naga Swetha**

Name of the subject:
Age :
Address :

a. I have been informed in my own vernacular language the purpose of the study, the necessity of relevant investigations to be carried out.
b. I understand that the medical information produced by this study will become part of institutional record and will be kept confidential by the said institute.
c. I understand that my participation is voluntary and may refuse to participate or may withdraw my consent and discontinue participation at any time without prejudice to my present or future care at this institution.
d. I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s).
e. I confirm that **Dr Modugula S Naga Swetha** has explained to me the purpose of research and the study procedure that I will undergo and the possible risks and discomforts that I may experience, in my own language. I hereby agree to give valid consent to participate as a subject in this research project.

Participant's signature
Signature of the witness: **Date:**

I have explained to _____ (subject) the purpose of the research, the possible risk and benefits to the best of my ability.

Guide signature **Date:**

NIH STROKE SCALE

Patient Identification. ___ ___-___ ___ ___-___ ___ ___

Pt. Date of Birth ___ ___/___ ___/___ ___

Hospital _____(___ ___-___ ___)

Date of Exam ___ ___/___ ___/___ ___

Interval: [ ] Baseline [ ] 2 hours post treatment [ ] 24 hours post onset of symptoms ±20 minutes [ ] 7-10 days
[ ] 3 months [ ] Other _____(___ ___)

Time: ___ ___:___ ___ [ ]am [ ]pm

Person Administering Scale _____

Administer stroke scale items in the order listed. Record performance in each category after each subscale exam. Do not go back and change scores. Follow directions provided for each exam technique. Scores should reflect what the patient does, not what the clinician thinks the patient can do. The clinician should record answers while administering the exam and work quickly. Except where indicated, the patient should not be coached (i.e., repeated requests to patient to make a special effort).

Instructions	Scale Definition	Score
1a. Level of Consciousness: The investigator must choose a response if a full evaluation is prevented by such obstacles as an endotracheal tube, language barrier, orotracheal trauma/bandages. A 3 is scored only if the patient makes no movement (other than reflexive posturing) in response to noxious stimulation.	0 = **Alert;** keenly responsive. 1 = **Not alert**; but arousable by minor stimulation to obey, answer, or respond. 2 = **Not alert**; requires repeated stimulation to attend, or is obtunded and requires strong or painful stimulation to make movements (not stereotyped). 3 = Responds only with reflex motor or autonomic effects or totally unresponsive, flaccid, and areflexic.	_____
1b. LOC Questions: The patient is asked the month and his/her age. The answer must be correct - there is no partial credit for being close. Aphasic and stuporous patients who do not comprehend the questions will score 2. Patients unable to speak because of endotracheal intubation, orotracheal trauma, severe dysarthria from any cause, language barrier, or any other problem not secondary to aphasia are given a 1. It is important that only the initial answer be graded and that the examiner not "help" the patient with verbal or non-verbal cues.	0 = **Answers** both questions correctly. 1 = **Answers** one question correctly. 2 = **Answers** neither question correctly.	_____
1c. LOC Commands: The patient is asked to open and close the eyes and then to grip and release the non-paretic hand. Substitute another one step command if the hands cannot be used. Credit is given if an unequivocal attempt is made but not completed due to weakness. If the patient does not respond to command, the task should be demonstrated to him or her (pantomime), and the result scored (i.e., follows none, one or two commands). Patients with trauma, amputation, or other physical impediments should be given suitable one-step commands. Only the first attempt is scored.	0 = **Performs** both tasks correctly. 1 = **Performs** one task correctly. 2 = **Performs** neither task correctly.	_____
2. Best Gaze: Only horizontal eye movements will be tested. Voluntary or reflexive (oculocephalic) eye movements will be scored, but caloric testing is not done. If the patient has a conjugate deviation of the eyes that can be overcome by voluntary or reflexive activity, the score will be 1. If a patient has an isolated peripheral nerve paresis (CN III, IV or VI), score a 1. Gaze is testable in all aphasic patients. Patients with ocular trauma, bandages, pre-existing blindness, or other disorder of visual acuity or fields should be tested with reflexive movements, and a choice made by the investigator. Establishing eye contact and then moving about the patient from side to side will occasionally clarify the presence of a partial gaze palsy.	0 = **Normal.** 1 = **Partial gaze palsy;** gaze is abnormal in one or both eyes, but forced deviation or total gaze paresis is not present. 2 = **Forced deviation,** or total gaze paresis not overcome by the oculocephalic maneuver.	_____

NIH STROKE SCALE

Patient Identification. ___ ___-___ ___ ___-___ ___ ___

Pt. Date of Birth ___ ___/___ ___/___ ___

Hospital _____(___ ___-___ ___)

Date of Exam ___ ___/___ ___/___ ___

Interval: [ ] Baseline [ ] 2 hours post treatment [ ] 24 hours post onset of symptoms ±20 minutes [ ] 7-10 days
[ ] 3 months [ ] Other _____(___ ___)

3. Visual: Visual fields (upper and lower quadrants) are tested by confrontation, using finger counting or visual threat, as appropriate. Patients may be encouraged, but if they look at the side of the moving fingers appropriately, this can be scored as normal. If there is unilateral blindness or enucleation, visual fields in the remaining eye are scored. Score 1 only if a clear-cut asymmetry, including quadrantanopia, is found. If patient is blind from any cause, score 3. Double simultaneous stimulation is performed at this point. If there is extinction, patient receives a 1, and the results are used to respond to item 11.	0 = **No visual loss.** 1 = **Partial hemianopia.** 2 = **Complete hemianopia.** 3 = **Bilateral hemianopia** (blind including cortical blindness).	_____
4. Facial Palsy: Ask – or use pantomime to encourage – the patient to show teeth or raise eyebrows and close eyes. Score symmetry of grimace in response to noxious stimuli in the poorly responsive or non-comprehending patient. If facial trauma/bandages, orotracheal tube, tape or other physical barriers obscure the face, these should be removed to the extent possible.	0 = **Normal** symmetrical movements. 1 = **Minor paralysis** (flattened nasolabial fold, asymmetry on smiling). 2 = **Partial paralysis** (total or near-total paralysis of lower face). 3 = **Complete paralysis** of one or both sides (absence of facial movement in the upper and lower face).	_____
5. Motor Arm: The limb is placed in the appropriate position: extend the arms (palms down) 90 degrees (if sitting) or 45 degrees (if supine). Drift is scored if the arm falls before 10 seconds. The aphasic patient is encouraged using urgency in the voice and pantomime, but not noxious stimulation. Each limb is tested in turn, beginning with the non-paretic arm. Only in the case of amputation or joint fusion at the shoulder, the examiner should record the score as untestable (UN), and clearly write the explanation for this choice.	0 = **No drift;** limb holds 90 (or 45) degrees for full 10 seconds. 1 = **Drift;** limb holds 90 (or 45) degrees, but drifts down before full 10 seconds; does not hit bed or other support. 2 = **Some effort against gravity;** limb cannot get to or maintain (if cued) 90 (or 45) degrees, drifts down to bed, but has some effort against gravity. 3 = **No effort against gravity;** limb falls. 4 = **No movement.** UN = **Amputation** or joint fusion, explain: _____ **5a. Left Arm** **5b. Right Arm**	_____ _____
6. Motor Leg: The limb is placed in the appropriate position: hold the leg at 30 degrees (always tested supine). Drift is scored if the leg falls before 5 seconds. The aphasic patient is encouraged using urgency in the voice and pantomime, but not noxious stimulation. Each limb is tested in turn, beginning with the non-paretic leg. Only in the case of amputation or joint fusion at the hip, the examiner should record the score as untestable (UN), and clearly write the explanation for this choice.	0 = **No drift;** leg holds 30-degree position for full 5 seconds. 1 = **Drift;** leg falls by the end of the 5-second period but does not hit bed. 2 = **Some effort against gravity;** leg falls to bed by 5 seconds, but has some effort against gravity. 3 = **No effort against gravity;** leg falls to bed immediately. 4 = **No movement.** UN = **Amputation** or joint fusion, explain: _____ **6a. Left Leg** **6b. Right Leg**	_____

NIH STROKE SCALE

Patient Identification. ___ ___-___ ___ ___-___ ___ ___

Pt. Date of Birth ___ ___/___ ___/___ ___

Hospital _____(___ ___-___ ___)

Date of Exam ___ ___/___ ___/___ ___

Interval: [ ] Baseline [ ] 2 hours post treatment [ ] 24 hours post onset of symptoms ±20 minutes [ ] 7-10 days
[ ] 3 months [ ] Other _____(___ ___)

7. Limb Ataxia: This item is aimed at finding evidence of a unilateral cerebellar lesion. Test with eyes open. In case of visual defect, ensure testing is done in intact visual field. The finger-nose-finger and heel-shin tests are performed on both sides, and ataxia is scored only if present out of proportion to weakness. Ataxia is absent in the patient who cannot understand or is paralyzed. Only in the case of amputation or joint fusion, the examiner should record the score as untestable (UN), and clearly write the explanation for this choice. In case of blindness, test by having the patient touch nose from extended arm position.	0 = **Absent.** 1 = **Present in one limb.** 2 = **Present in two limbs.** UN = **Amputation** or joint fusion, explain: _____	_____
8. Sensory: Sensation or grimace to pinprick when tested, or withdrawal from noxious stimulus in the obtunded or aphasic patient. Only sensory loss attributed to stroke is scored as abnormal and the examiner should test as many body areas (arms [not hands], legs, trunk, face) as needed to accurately check for hemisensory loss. A score of 2, "severe or total sensory loss," should only be given when a severe or total loss of sensation can be clearly demonstrated. Stuporous and aphasic patients will, therefore, probably score 1 or 0. The patient with brainstem stroke who has bilateral loss of sensation is scored 2. If the patient does not respond and is quadriplegic, score 2. Patients in a coma (item 1a=3) are automatically given a 2 on this item.	0 = **Normal;** no sensory loss. 1 = **Mild-to-moderate sensory loss;** patient feels pinprick is less sharp or is dull on the affected side; or there is a loss of superficial pain with pinprick, but patient is aware of being touched. 2 = **Severe to total sensory loss;** patient is not aware of being touched in the face, arm, and leg.	_____
9. Best Language: A great deal of information about comprehension will be obtained during the preceding sections of the examination. For this scale item, the patient is asked to describe what is happening in the attached picture, to name the items on the attached naming sheet and to read from the attached list of sentences. Comprehension is judged from responses here, as well as to all of the commands in the preceding general neurological exam. If visual loss interferes with the tests, ask the patient to identify objects placed in the hand, repeat, and produce speech. The intubated patient should be asked to write. The patient in a coma (item 1a=3) will automatically score 3 on this item. The examiner must choose a score for the patient with stupor or limited cooperation, but a score of 3 should be used only if the patient is mute and follows no one-step commands.	0 = **No aphasia;** normal. 1 = **Mild-to-moderate aphasia;** some obvious loss of fluency or facility of comprehension, without significant limitation on ideas expressed or form of expression. Reduction of speech and/or comprehension, however, makes conversation about provided materials difficult or impossible. For example, in conversation about provided materials, examiner can identify picture or naming card content from patient's response. 2 = **Severe aphasia;** all communication is through fragmentary expression; great need for inference, questioning, and guessing by the listener. Range of information that can be exchanged is limited; listener carries burden of communication. Examiner cannot identify materials provided from patient response. 3 = **Mute, global aphasia;** no usable speech or auditory comprehension.	_____
10. Dysarthria: If patient is thought to be normal, an adequate sample of speech must be obtained by asking patient to read or repeat words from the attached list. If the patient has severe aphasia, the clarity of articulation of spontaneous speech can be rated. Only if the patient is intubated or has other physical barriers to producing speech, the examiner should record the score as untestable (UN), and clearly write an explanation for this choice. Do not tell the patient why he or she is being tested.	0 = **Normal.** 1 = **Mild-to-moderate dysarthria;** patient slurs at least some words and, at worst, can be understood with some difficulty. 2 = **Severe dysarthria;** patient's speech is so slurred as to be unintelligible in the absence of or out of proportion to any dysphasia, or is mute/anarthric. UN = **Intubated** or other physical barrier, explain:_____	_____

NIH STROKE SCALE

Patient Identification. ___ ___-___ ___ ___-___ ___ ___

Pt. Date of Birth ___ ___/___ ___/___ ___

Hospital _____(___ ___-___ ___)

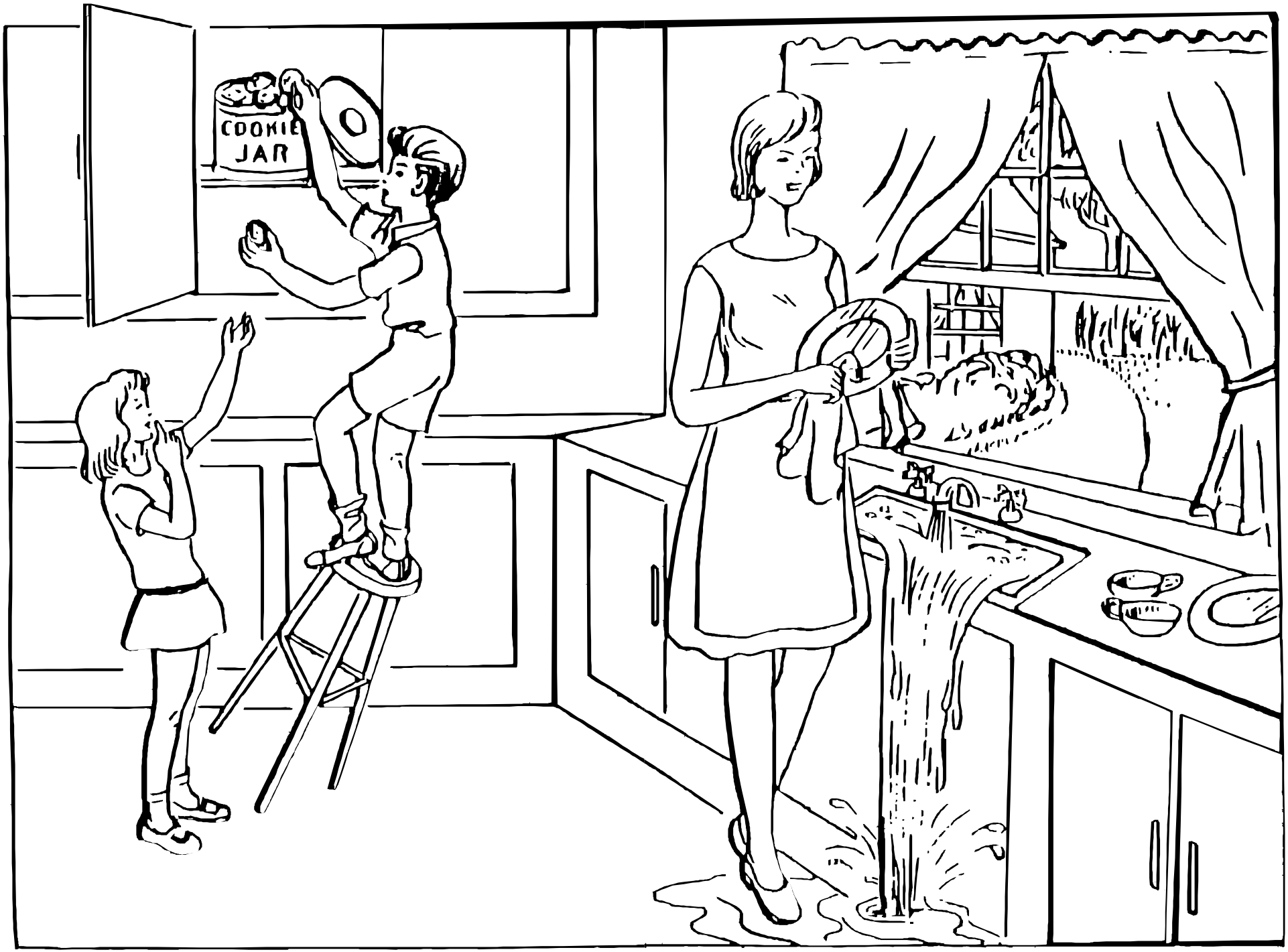
Date of Exam ___ ___/___ ___/___ ___

Interval: [ ] Baseline [ ] 2 hours post treatment [ ] 24 hours post onset of symptoms ±20 minutes [ ] 7-10 days
[ ] 3 months [ ] Other _____(___ ___)

11. Extinction and Inattention (formerly Neglect): Sufficient information to identify neglect may be obtained during the prior testing. If the patient has a severe visual loss preventing visual double simultaneous stimulation, and the cutaneous stimuli are normal, the score is normal. If the patient has aphasia but does appear to attend to both sides, the score is normal. The presence of visual spatial neglect or anosagnosia may also be taken as evidence of abnormality. Since the abnormality is scored only if present, the item is never untestable.	0 = **No abnormality.** 1 = **Visual, tactile, auditory, spatial, or personal inattention** or extinction to bilateral simultaneous stimulation in one of the sensory modalities. 2 = **Profound hemi-inattention or extinction to more than one modality;** does not recognize own hand or orients to only one side of space.	_____

_____

_____


COOKIE
JAR

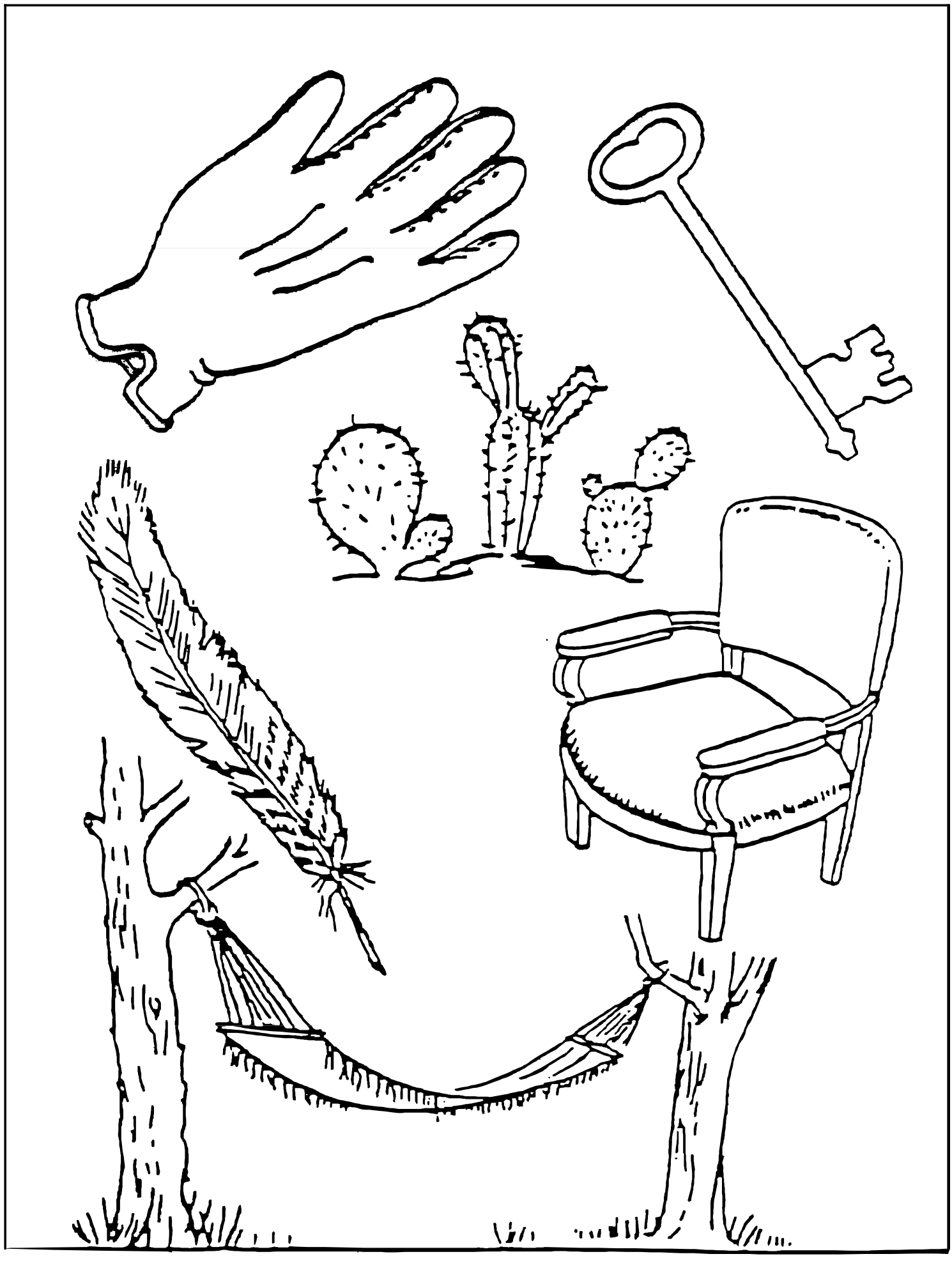
You know how.

Down to earth.

I got home from work.

Near the table in the dining room.

They heard him speak on the radio last night.



MAMA

TIP – TOP

FIFTY – FIFTY

THANKS

HUCKLEBERRY

BASEBALL PLAYER

KEY TO MASTER CHART

IP. NO.	Inpatient number
HTN	Hypertension
T2DM	Type 2 Diabetes mellitus
SBP	Systolic Blood Pressure
DBP	Diastolic Blood Pressure
RBS	Random Blood Sugar
BUN	Blood Urea Nitrogen
S.Creat	Serum Creatinine
NIHSS	National Institute of Health Stroke Severity

MASTER CHART

Serial no.	IP no.	Sex	Age	HTN	T2DM	SMOKING	ALCOHOL	SBP	DBP	RBS	Blood urea	BLOOD UREA/	S Creat	BUN/ s creat	hydration	NIHSS 1	NIHSS 3	END
1	243885	M	46	Y	N	Y	N	154	92	200	26	12.15	0.9	13.5	ND	18	22	P
2	245458	M	57	Y	Y	N	N	160	96	113	43	20.16	1.2	16.8	D	20	20	A
3	253108	M	72	N	N	N	N	148	84	132	30	14.04	0.9	15.6	D	19	20	A
4	253904	M	52	Y	Y	N	N	138	76	117	36	16.8	1	16.8	D	18	19	A
5	246228	M	56	Y	N	Y	N	144	86	123	23	10.8	0.9	12	ND	8	8	A
6	254716	M	53	N	N	N	N	146	68	132	34	16	0.8	20.2	D	18	19	A
7	267349	F	53	Y	Y	N	N	148	74	128	64	30	1.2	25	D	16	21	P
8	259420	M	66	N	N	Y	N	130	80	187	25	12	1	12	ND	16	15	A
9	259887	M	66	N	N	N	N	142	68	109	40	18.5	1.1	16.8	D	17	17	A
10	262809	M	65	Y	Y	N	N	156	78	112	30	13.8	0.8	17.3	D	10	10	A
11	226419	M	57	Y	N	Y	Y	170	86	136	55	25.6	1	25.6	D	18	23	P
12	269845	M	66	Y	Y	N	N	128	90	109	19	9	0.7	13	ND	12	12	A
13	269759	F	66	N	N	N	N	134	86	112	35	16.5	0.9	18.3	D	19	19	A
14	144332	F	67	Y	Y	N	N	146	74	112	30	14	1	14	ND	13	14	A
15	272029	F	70	N	N	N	N	126	82	118	20	8.3	0.6	13.8	ND	26	26	A
16	271163	M	64	Y	Y	N	Y	138	90	120	27	12.6	0.7	18	D	20	22	A
17	267352	M	75	Y	N	Y	Y	144	80	142	25	11.5	0.9	12.8	ND	36	40	P
18	271201	M	57	Y	Y	Y	N	148	78	250	28	13.3	0.6	22.2	D	30	35	P
19	276589	M	46	Y	Y	N	N	142	90	112	21	9.7	0.8	12.2	ND	20	21	A
20	272292	F	57	N	N	N	N	150	90	123	25	11.6	1	11.6	ND	19	19	A
21	272419	F	56	N	N	N	N	156	80	202	24	11.5	0.9	12.8	ND	28	32	P
22	273150	F	57	Y	Y	N	N	164	80	106	30	14	1.1	12.8	ND	33	33	A
23	272404	F	71	N	N	N	N	158	68	140	35	16.2	0.9	18	D	18	19	A
24	278112	M	57	Y	N	N	N	128	78	128	19	9	0.9	10	ND	40	40	A
25	263604	F	57	Y	Y	N	N	158	86	120	32	15.1	1.1	13.8	ND	17	17	A
26	280109	M	70	Y	Y	Y	Y	128	62	206	45	21	1	21	D	24	28	P
27	277044	F	58	N	N	N	N	136	78	118	51	24	1	24	D	26	25	A
28	280966	M	69	Y	N	N	Y	148	76	112	30	14	0.9	15.6	D	29	29	A
29	286579	M	57	N	N	Y	Y	162	80	128	23	11	0.5	21.8	D	16	20	P
30	289072	M	52	Y	Y	N	N	134	68	109	29	13.5	1	13.5	ND	18	19	A

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31	289352	M	56	Y	N	Y	Y	120	80	98	41	19.4	1	19.4	D	20	24	P
32	291448	M	56	N	N	N	N	128	80	104	31	14.7	1	14.7	ND	18	19	A
33	279687	F	56	N	N	N	N	134	78	152	17	8	0.5	16	D	20	26	P
34	281470	F	56	Y	N	N	N	156	64	103	23	11	0.8	13.6	ND	22	22	A
35	281030	F	57	Y	Y	N	Y	166	90	96	35	16.2	0.9	18	D	18	19	A
36	294212	M	59	N	N	Y	Y	160	86	106	37	17.4	1.1	15.8	D	18	24	P
37	283209	F	69	N	N	N	N	154	74	200	26	12.4	1	12.4	ND	19	19	A
38	295413	M	59	N	N	Y	Y	158	82	140	30	14	1	14	ND	24	27	P
39	283194	F	59	N	N	N	N	128	90	142	27	12.8	0.9	14.2	ND	26	29	P
40	283751	F	68	N	N	N	N	158	80	202	25	11.7	0.9	13	ND	20	20	A
41	285772	F	68	Y	N	N	N	128	78	142	26	12	0.6	20	D	19	23	P
42	240437	F	60	Y	Y	N	N	158	90	204	23	11	0.8	14	ND	22	23	A
43	298445	M	58	N	N	N	N	128	70	124	36	17	1	17	D	19	19	A
44	291222	F	69	Y	N	N	Y	116	70	123	27	12.9	0.9	14.4	ND	21	23	A
45	298817	M	59	Y	Y	Y	Y	148	90	156	30	14.3	1.1	13	ND	10	10	A
46	294270	F	61	N	N	N	N	162	92	173	13	5.9	0.5	11.7	ND	15	15	A
47	299684	M	58	Y	N	N	N	134	80	138	28	13.2	0.6	22	D	20	24	P
48	303627	M	61	N	N	N	N	148	78	102	42	19.8	1.1	18	D	11	11	A
49	307333	M	69	Y	Y	Y	Y	142	88	123	25	12	1	12	ND	14	16	A
50	392414	M	59	Y	Y	Y	Y	150	80	160	47	22	1	22	D	21	25	P
51	392505	M	62	Y	N	N	N	156	78	102	30	14	1	14	ND	10	11	A
52	296067	F	62	N	N	N	Y	134	74	103	24	11.2	0.8	14	ND	15	15	A
53	297417	F	69	Y	Y	N	N	158	100	165	14	6.6	0.6	11	ND	12	12	A
54	393311	M	62	N	N	N	Y	128	88	122	23	11	0.5	22	D	14	16	A
55	302246	F	62	Y	Y	N	N	158	78	126	21	10	0.5	20	D	16	20	P
56	145609	F	58	Y	Y	N	N	128	90	100	23	10.6	0.9	11.8	ND	11	13	A
57	391433	M	61	N	N	Y	Y	116	68	125	19	9	0.5	18	D	18	22	P
58	391462	M	58	Y	N	N	Y	148	98	120	22	10.2	0.8	12.8	ND	10	10	A
59	323308	M	59	N	N	N	Y	142	70	120	34	16	1	16	D	14	16	A
60	304557	F	61	Y	Y	N	N	132	84	134	18	8.4	0.7	12	ND	11	12	A
61	307491	F	59	Y	Y	N	N	126	94	121	15	7.2	0.6	12	ND	19	24	P
62	308896	F	69	Y	Y	N	N	128	92	116	24	11	0.8	14	ND	12	13	A

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63	313977	F	62	N	N	N	Y	126	68	118	28	13	1.3	10	ND	15	16	A
64	380834	M	70	N	N	Y	Y	136	86	110	46	21.6	1.2	18	D	20	24	P
65	322741	F	61	Y	N	N	N	158	88	167	24	11.2	0.7	16	D	15	15	A
66	313972	M	64	N	N	N	N	160	100	142	20	9.6	0.8	12	ND	11	11	A
67	323057	F	62	N	N	N	N	110	80	137	42.8	20	0.8	25	D	21	26	P
68	314495	M	64	Y	Y	N	Y	124	98	193	33	15.6	1.2	13	ND	10	10	A
69	315083	M	54	Y	Y	N	N	154	70	132	28	13.2	1.3	10.2	ND	12	14	A
70	317063	M	64	Y	Y	N	Y	152	84	176	23	11	0.5	22	D	12	14	A
71	317152	M	64	N	N	N	Y	140	94	194	12	5.7	0.5	11.5	ND	12	12	A
72	319167	M	65	Y	Y	Y	Y	140	92	383	25	11.5	0.8	14.4	ND	22	25	P
73	306581	F	63	N	N	N	N	146		123	43	20	1	20	D	14	16	A
74	308894	F	65	Y	N	N	N	158	82	106	36	16.8	0.7	24	D	23	26	P
75	324034	M	73	N	N	N	N	128	90	132	21	10	0.8	12.6	ND	11	11	A
76	331903	F	65	Y	Y	N	N	158	92	110	35	16.5	1.2	13.8	ND	13	15	A
77	324444	M	65	Y	Y	Y	N	128	80	140	72	34	1.3	26	D	24	30	P
78	303964	M	65	Y	Y	N	N	158	78	106	11	5.3	0.5	10.6	ND	15	16	A
79	328533	M	73	Y	Y	N	N	128	88	104	15	7	0.5	14	ND	14	15	A
80	335292	F	66	N	N	N	N	116	80	122	37	17.6	0.8	22	D	14	14	A
81	328700	M	65	N	N	Y	Y	148	82	136	34	11	1	11	ND	25	31	P
82	335296	M	66	Y	N	N	N	158	84	108	28	13.4	1.1	12.2	ND	10	11	A
83	339040	M	58	Y	N	N	N	128	68	132	14	6.3	0.5	12.7	ND	15	17	A
84	339505	M	66	N	N	N	N	158	94	132	23	11	0.8	13.8	ND	12	14	A
85	310356	M	66	Y	Y	Y	N	128	92	106	30	14	1	14	ND	14	14	A
86	360907	F	65	N	N	N	N	116	68	123	25	11.9	0.7	17	D	23	26	P
87	364864	F	54	Y	Y	N	N	148	86	126	19	9	0.6	15	D	15	20	P
88	392040	F	66	Y	Y	N	N	162	88	134	15	6.9	0.6	11.5	ND	11	12	A
89	390545	F	58	N	N	N	N	158	100	168	44	20.8	1.3	16	D	18	22	P
90	389026	F	66	Y	Y	N	N	128	80	122	14	6.5	0.5	13	ND	15	17	A
91	344814	F	49	Y	Y	N	N	116	98	128	15	7	0.5	14	ND	11	13	A
92	340076	M	66	Y	N	Y	Y	148	70	155	29	13.6	0.8	17	D	14	18	P
93	349814	F	60	Y	N	N	N	150	76	115	30	14	1	14	ND	10	12	A
94	340004	M	67	Y	Y	Y	Y	142	80	118	16	7.4	0.7	10.6	ND	14	14	A

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95	349742	F	67	Y	N	N	N	124	68	132	23	10.8	0.6	18	D	15	16	A
96	341660	M	64	Y	N	N	N	160	80	102	22	10.1	0.8	12.6	ND	14	15	A
97	176613	M	65	Y	Y	Y	N	126	80	185	36	16.9	1.3	13	ND	13	18	P
98	346150	M	55	Y	N	N	N	148	78	170	27	12.5	1.2	10.4	ND	14	15	A
99	350491	M	67	Y	Y	N	N	128	64	160	28	13.3	0.7	19	D	12	12	A
100	351460	F	63	Y	Y	N	N	128	90	132	25	11.6	0.8	14.5	ND	12	14	A
101	298870	F	67	N	N	N	N	150	86	142	35	16	0.8	20	D	11	13	A
102	381295	M	67	Y	Y	N	Y	142	74	112	36	16.8	1.2	14	ND	13	15	A
103	358227	F	68	N	N	N	N	126	82	100	36	16.9	1.3	13	ND	10	12	A
104	380834	M	68	Y	Y	N	N	146	90	106	13	6	0.5	12	ND	15	15	A
105	359921	F	68	N	N	N	N	144	80	122	14	6.5	0.5	11	ND	20	27	P
106	368916	F	63	Y	Y	N	N	132	80	137	36	16.8	0.8	21	D	12	13	A
107	379942	M	68	N	N	N	N	128	89	122	30	14.2	1	14.2	ND	16	18	A
108	369056	F	64	Y	Y	N	N	150	92	163	19.6	9.2	0.7	13.2	ND	15	16	A
109	391327	F	63	Y	Y	N	N	142	84	158	24	11.2	0.8	14	ND	14	14	A
110	388389	F	67	Y	Y	N	Y	124	72	124	56	26.4	1.2	22	D	12	17	P
111	379896	M	63	Y	N	N	N	160	98	132	18	8.4	0.6	14	ND	13	13	A
112	363462	F	65	Y	N	N	N	126	76	142	28	13.2	1.1	12	ND	12	12	A
113	300426	F	63	Y	Y	N	N	148	80	126	44	20.7	0.9	23	D	11	11	A
114	371485	F	60	Y	N	N	N	128	68	128	22	10.4	0.9	11.6	ND	18	21	P
115	379332	M	59	Y	Y	N	N	128	80	108	22	10.4	0.8	13	ND	10	12	A
116	373337	F	60	N	N	N	N	150	80	129	62	28.8	1.2	24	D	14	18	P
117	368189	M	68	Y	Y	N	N	142	78	104	17	7.8	0.6	13	ND	10	10	A
118	367467	M	68	Y	Y	Y	N	132	64	132	34	15.8	1.1	14.4	ND	18	23	P
119	363457	M	60	Y	Y	N	N	114	90	106	21	9.9	0.9	11	ND	15	16	A
120	389026	F	68	Y	N	N	N	170	86	122	13	6.1	0.5	12.2	ND	13	19	A
121	361873	M	63	N	N	Y	Y	158	74	138	48	22.5	0.9	25	D	13	16	P
122	361875	M	67	Y	Y	N	N	132	82	190	24	11.1	0.8	13.8	ND	12	12	A
123	451305	M	64	Y	Y	N	N	148	90	90	36	16.8	1.2	14	ND	11	11	A
124	390545	F	59	N	N	N	N	118	80	189	33	15.6	0.6	26	D	15	19	P
125	392040	F	70	N	N	N	N	128	82	183	30	14	1	14	ND	16	22	P