# PREVALENCE AND PREDICTORS OF SLEEP DYSFUNCTION AFTER ISCHEMIC STROKE AND ITS IMPACT ON STROKE RECOVERY

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**DM NEUROLOGY THESIS**

**(2021-2023)**

**DEPARTMENT OF NEUROLOGY**

**SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES AND TECHNOLOGY, THIRUVANANTHAPURAM**

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A THESIS SUBMITTED BY

**DR. AVINASH T KULKARNI**

TO

**DEPARTMENT OF NEUROLOGY**

**SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES AND TECHNOLOGY, THIRUVANANTHAPURAM**

**INDIA**

IN PARTIAL FULFILMENT OF THE REQUIREMENTS

FOR THE AWARD OF

**DM NEUROLOGY**

**(2021-2023)**

# DECLARATION BY THE STUDENT

I hereby declare that this thesis titled "Prevalence & predictors of sleep dysfunction after ischemic stroke & its impact on stroke recovery" has been prepared by me under the supervision and guidance of Dr.Sapna Erat Sreedharan (Professor) & Dr. Sylaja P N (Professor & HOD), Department of Neurology, Sree Chitra Tirunal Institute for Medical Sciences and Technology, Trivandrum.

Date: 24/8/2023 (Dr. Avinash T Kulkarni)

Place: Thiruvananthapuram

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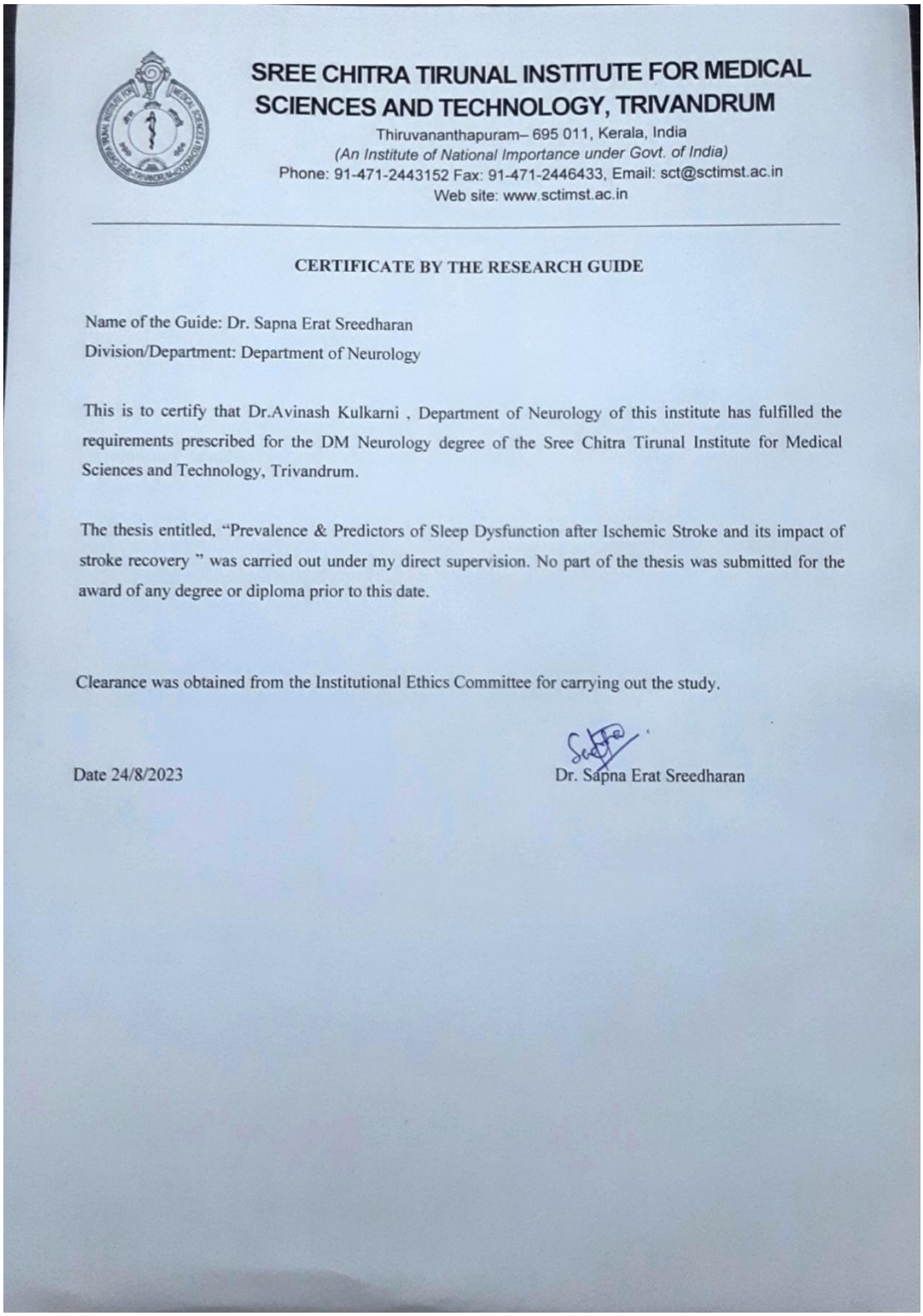

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The thesis entitled, "Prevalence & Predictors of Sleep Dysfunction after Ischemic Stroke and its impact of stroke recovery " was carried out under my direct supervision. No part of the thesis was submitted for the award of any degree or diploma prior to this date.

Clearance was obtained from the Institutional Ethics Committee for carrying out the study.

Date 24/8/2023

Dr. Sapna Erat Sreedharan

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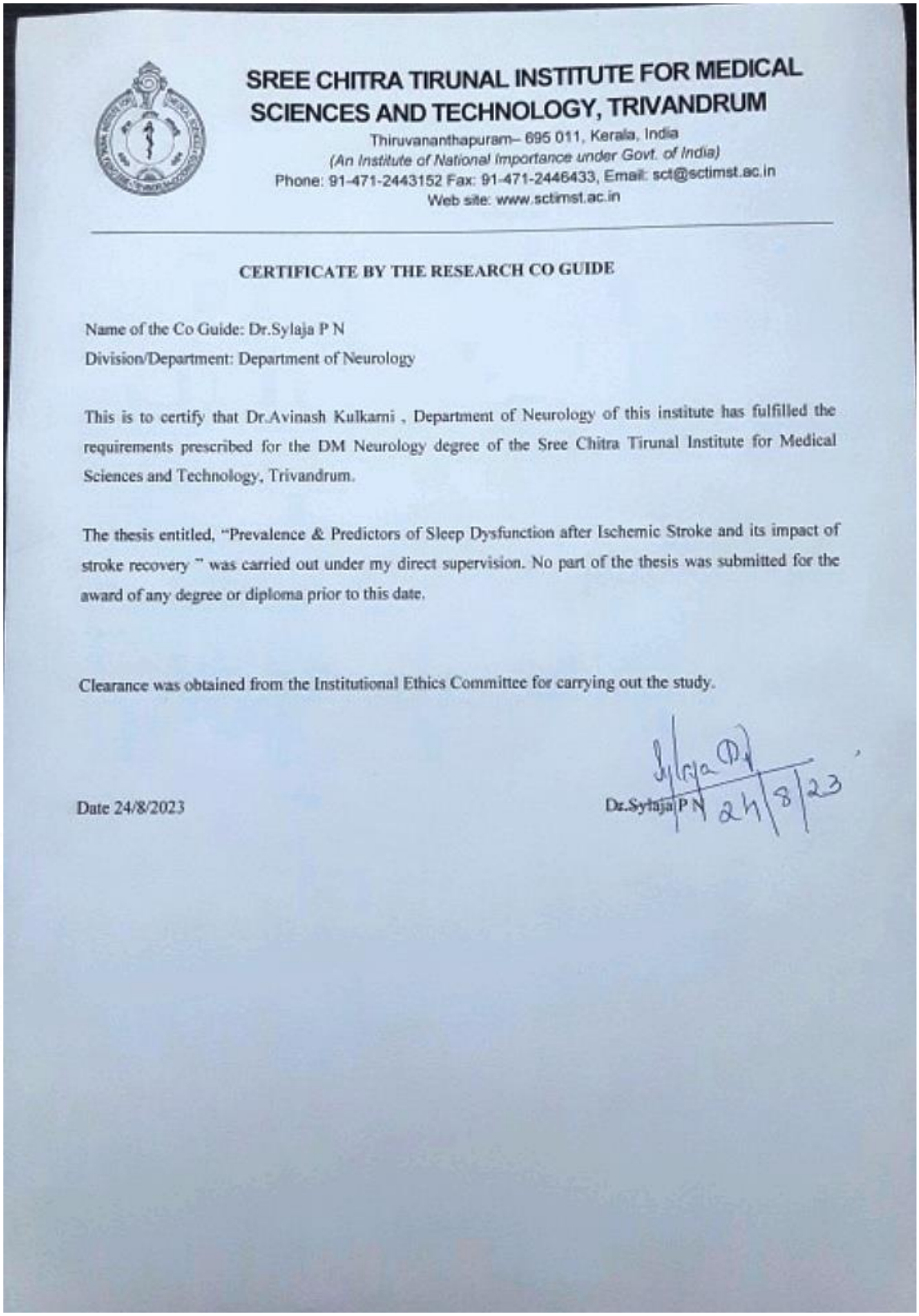

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## APPROVAL OF THESIS

The thesis entitled

"Prevalence & predictors of sleep dysfunction after ischemic stroke & its impact on stroke recovery"

Submitted by

Dr Avinash T Kulkarni

for the degree of

**DM NEUROLOGY**

Of

SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES AND TECHNOLOGY, TRIVANDRUM

is evaluated and approved by

Dr Sapna Erat Sreedharan

(Guide) (External Examiner)

# ACKNOWLEDGEMENT

I am deeply indebted to my teachers & guides especially Dr. Sapna Erat Sreedharan (Professor) & Dr. Sylaja P N (Professor & HOD) for their constant unwavering support, insightful criticism, expert supervision and immense patience throughout this study.

I am profoundly grateful Miss Chandralekha S C, for all of her support and effort in data collection & patient follow up.

I would especially like to acknowledge my gratitude to my past and present colleagues, nursing staff & the technologists of the Department Neurology for their valuable assistance at all times during this study.

I also would like to thank Mr Rajeev & Mr Deepak from R & D Healthcare for performing ambulatory PSG at a reduced rate.

I would also like to extend my heartfelt gratitude to my family for being immensely supportive through my endeavours. I could not have achieved what I have without their prayers, love, and support.

Last but not least I am eternally grateful to all my patients & their relatives who have been very understanding and generous with their cooperation all through the study.

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# ABBREVIATIONS

NIHSS : National Institute of Health Stroke Scale

mRS : Modified Rankin Scale

HTN : Hypertension

DM : Diabetes Mellitus

SDB : Sleep disordered breathing

OSA : Obstructive sleep apnea

CAD : Coronary artery disease

POVD : Peripheral occlusive vascular disease

REM : Rapid Eye Movement

NREM : Non Rapid eye movement

PSG : Polysomnography

AHI : Apnea hypoapnea index

RLS : Restless leg syndrome

PLMS : Periodic limb movement syndrome

TST : Total sleep time

SE : Sleep efficiency

WASO : Wake up after sleep onset

PRO : Patient reported outcome

MRI : Magnetic resonance imaging

ASPECTS: Alberta stroke programme early CT score

ACA : Anterior cerebral artery

MCA : Middle cerebral artery

PCA : Posterior cerebral artery

CSF : Cerebrospinal fluid

PET : Positron emission tomography

EEG : Electroencephalogram

ESS : Epworth sleepiness scale

ISI : Insomnia severity index

PSQI : Pittsburgh sleep quality index

CBS : Care giver burden scale

PHQ 9 : Patient health questionnaire 9

# SYNOPSIS

## PREVALENCE AND PREDICTORS OF SLEEP DYSFUNCTION AFTER ISCHEMIC STROKE AND ITS IMPACT ON STROKE RECOVERY

**Background and Aim**: Sleep dysfunction is often reported post stroke ,but its impact on short term outcome and caregiver burden remains less studied. Here, we looked at the prevalence and predictors of sleep dysfunction and its relationship with short term functional outcome and caregiver burden 3 months after stroke.

**Methods:** Ours was a prospective observational study where consecutive patients with acute ischemic stroke at 3 months follow-up visit were recruited after informed consent. Clinical and imaging data were collected and 3 and 6 month functional outcome was measured using modified Rankin score with scores 0-2 taken as good outcome. All the patients were administered 5 questionnaires (Epworth Sleepiness Scale, Insomnia severity index and Pittsburgh sleep quality index for sleep, Patient health quality 9 for depression and Zarit's care giver burden scale). 20% patients underwent overnight ambulatory polysomnography. Clinical and sleep characteristics were correlated with functional outcome and caregiver burden scores.

**Results**: Of 100 patients, 70% were men with mean age 62.2 ± 11.2 years, 67% had moderate to severe strokes at admission with mean NIHSS 8.3 ± 6.24. 46% had hypersomnolence,35% had insomnia and 40% had poor sleep quality at 3 months after stroke.45% reported depression and 22% care givers experienced significant burden. NIHSS at onset, smoking, recurrent strokes and anterior circulation strokes had strongest correlation with sleep dysfunction. Frequency of sleep dysfunction was not affected by the presence of sleep apnea. Care giver burden had strong correlation with patient reported hypersomnolence and poor sleep quality. Sleep dysfunction had significant association with poor short-term outcome at 3 and 6 months post stroke.

**Conclusion:** Sleep dysfunction is present in a significant number of ischemic stroke survivors and can contribute to poor outcome and caregiver stress. The impact of early

recognition and timely treatment of sleep dysfunction to improve stroke outcome needs to be studied in larger populations.

# INTRODUCTION

A variety of sleep disorders are reported in stroke survivors during acute and chronic phase, well studied being sleep disordered breathing. There is ample literature suggesting that SDB can be a risk factor for stroke and have an adverse impact on neurological outcome [(1)]. However, the effect of stroke on sleep wake cycle is less studied, with some authors reporting hypersomnolence post stroke while some patients develop severe insomnia [(2)]. Experimental studies in animal stroke models have shown that sleep deprivation can reduce neuronal plasticity and impair functional recovery at short term [(3)]. Sleep wake cycle disturbances after stroke has been reported to be associated with increased risk of depression and suboptimal functional outcome [(4)]. The association of sleep and stroke extends beyond SDB to disorders of the sleep-wake cycle, including long and short sleep duration, circadian rhythm disorders and insomnia. Approximately half of stroke survivors have insomnia. Insomnia has also been associated with higher rates of incident stroke and worse post-stroke outcome.

Predictors of sleep dysfunction at short term after stroke is poorly understood. Also the impact of sleep dysfunction in stroke survivors on recovery, functional outcome and the care giver burden remains less known. Despite estimates of greater than 50% prevalence of sleep disorders after stroke, only about 6% of stroke survivors are offered formal sleep testing and an estimated 2% complete such testing in the 3-month post-stroke period [(5)]. The reasons for the low rate of screening are at least partly related to the lack of awareness regarding sleep disorders among stroke providers.

In India stroke occurs a decade earlier, leaves over half of survivors unable to resume their jobs & results in significant caregiver burden. Sapna et al reported significant loss of occupation (62% pre stroke to 20% post stroke), decline in social function among stroke survivors and a significant caregivers burden [(94)]. There is a paucity of data on factors associated with sleep dysfunction, other than sleep apnea after ischemic stroke, especially from low-middle income countries like India and its impact on stroke recovery and caregiver burden.

In this study, we have investigated the prevalence and predictors of sleep dysfunction after ischemic stroke and its impact on short term recovery and caregiver burden.

# AIMS & OBJECTIVES

# AIMS & OBJECTIVES

1. To study the prevalence and predictors of sleep dysfunction (Hypersomnolence, Insomnia, Poor sleep quality) at 3 months post stroke.

2. To study the prevalence and predictors of depression & care giver burden at 3 months post stroke.

3. To study effect of sleep dysfunctions on short term functional outcome at 6 months after stroke.

# REVIEW OF LITERATURE

# REVIEW OF LITERATURE

The suspension of consciousness is the behavioural definition of sleep. Scientists have been baffled by the purpose of sleep up until recently. On average, people spend a third of their lives in this appearance of passivity. Long-term sleep deprivation in rodent and human models has been proven lethal [8,9]. It is now understood that sleep does not just come from a reduction in brain activity. For instance, the brain's activity during rapid eye movement (REM) sleep is similar to that of awakening [10]. Recent experimental evidence suggests that sleep may also play a critical neuroprotective and restorative role within the central nervous system [11]. Brain ionic currents and neuronal voltage variations are used to electro physiologically quantify sleep. Utilising a battery of concurrent sensors, polysomnography is the gold standard and de facto method for evaluating sleep in both clinical and laboratory settings [12]. Alternative methods that are less intrusive include objective measures such as actigraphy or consumer-grade accelerometers, and subjective measures such as sleep diaries and self-report questionnaires. Recent solid evidence indicates that neurological diseases and sleep disturbances are significantly correlated [13]. Sleep-wake disorder may be directly attributed to neurological conditions that affect the thalamocortical tracts and ascending reticular activating system, though it has also been demonstrated that areas outside of these pathways control sleep-wake function [14]. Similar to this, sleep issues may have a role in the aetiology of neurodegenerative disorders [15]. The link between sleep disorders and cerebrovascular diseases like stroke, the most typical neurological cause of long-term disability in adults, has, however, received less attention [16].

Stroke patients commonly experience sleep-related difficulties, with reviews describing evidence dating back to the 19th Century [17]. A wide range of sleep disorders are associated with stroke, including sleep-disordered breathing, insomnia, parasomnia, circadian rhythm disorder, sleep-related movement disorder, hypersomnia and excessive daytime sleepiness [18,19] . Sleep problems can arise as a consequence of the stroke itself, in which brain areas involved in sleep regulation (including the hypothalamus, brainstem and thalamus) are impaired [20] although the poor correlation between lesion site and

sleep disturbance has been noted [21] . More commonly, sleep disturbances arise due to factors associated with stroke, such as sleep-disordered breathing, medication use, inactivity, environment, depression, stress and premorbid health problems[18].

However, the impact of such factors is complex, as highlighted by Taylor et al [22] who reported not only a higher prevalence of insomnia in those with medical problems than those without, but also a higher prevalence of medical problems in those with insomnia than those without.

Additionally, sleep disruption can increase the risk of ischemic stroke [19] making it a risk factor for stroke.[23]

**Neural Circuitry Involved in Sleep-Wake Functioning**

The first time sleep and wakefulness-related brain areas were identified was in 1949 when cholinergic neurons near the pons-midbrain junction were triggered. Electroencephalographic (EEG) alterations indicating awake and arousal were produced when this area was activated, specifically, low-amplitude (30 uV), high frequency (15-60 Hz), activity [24]. Findings from Magoun

and Moruzzi were among the first to discover that wakefulness is not simply the result of heightened sensory input, rather it arises through systematic activation of specialised brain regions responsible for arousal and waking states [24]. This central arousal system in the brain is now known as the ascending reticular activating system and extends from the medulla and pons onto fibre tracts projecting toward thalamic nuclei and forebrain cholinergic systems (Figure 1).

Slow-wave sleep has been demonstrated to be induced by reticular activation system inhibition and low-frequency electrical stimulation of the thalamus [10,25,26]. Slow-wave sleep is characterised by low frequency (0.5–4 Hz), high amplitude (100–150 uV), synchronous brain activity. The thalamus plays a role in sleep-wake functioning by producing sleep spindles, K complexes, and neocortical high-amplitude, low frequency slow waves that are signs of restorative non-rapid eye movement (NREM) sleep [14]. Studies into the neural circuitry involved in rapid eye movement (REM) sleep [27] have offered additional proof of how sleep interacts with the thalamus and cortical brain areas. Electroencephalographic (EEG) recordings of REM sleep, as opposed to NREM sleep, is

similar to those of awake people, they exhibit low-amplitude, mixed-frequency, and disorganised activity. Near-total muscular atonia caused by the activation of locus coeruleus neurons distinguishes REM sleep from other types of sleep. Endogenously controlled signals from the pontine reticular formation that are sent to the superior colliculus' motor region cause the ballistic-like eye movements that are a hallmark of REM sleep (Figure 1) [28] . These collicular neuronal signals are projected onto the paramedial pontine reticular formation and the rostral interstitial nucleus.

The low voltage, high frequency EEG changes that typify REM sleep are known as ponto-geniculo-occipital (PGO) waves and originate in the pontine reticular formation[27]. These signals are then propagated onto the lateral geniculate nucleus and the occipital lobe. The PGO waves representative of REM sleep provide yet another neural network by which brainstem nuclei impact activation of neocortical regions (Fig 2) [26]

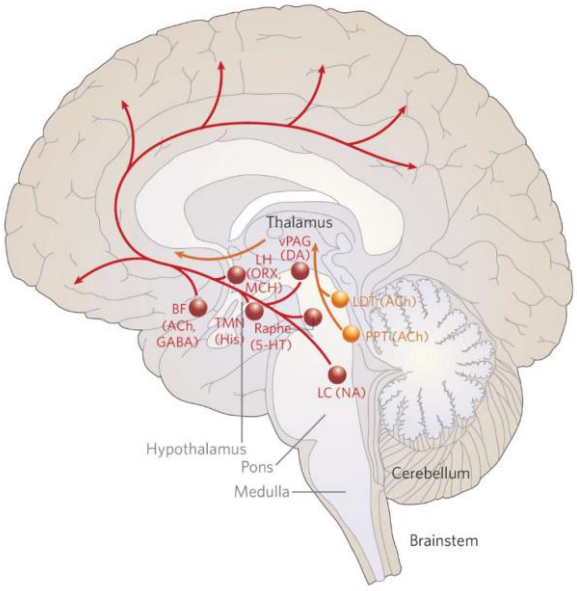


**Figure 1**. [30] **The ascending reticular activating system.** Input to thalamic nuclei (orange) project from pontine cholinergic (ACh) cell groups, the pedunculopontine (PPT) and laterodorsal tegmental nuclei (LDT). A second pathway (red) activates the cerebral cortex to facilitate the processing of inputs from the thalamus through monoaminergic cell groups, including the tuberomammillary nucleus (TMN) containing histamine (His), dopamine (DA), the dorsal and median raphe nuclei containing serotonin (5-HT), and the locus coeruleus (LC) containing noradrenaline (NA). This pathway also receives projections from peptidergic neurons in the lateral hypothalamus (LHA) containing orexin (ORX) or melanin-concentrating hormone (MCH), and from basal forebrain (BF) neurons that contain GABA or ACh. By Saper, Scamelli, & Lu (2005) [30].

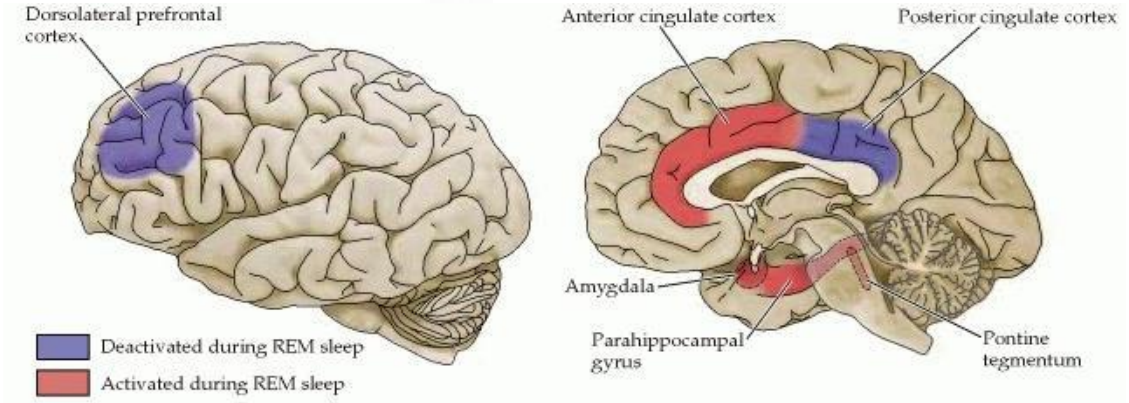


**Figure 2** [31] Diagram showing cortical regions where activity is increased or decreased during REM sleep. Purves et al. (2010) [31]

Key brain areas that are engaged during REM sleep have been identified by researchers using functional magnetic resonance imaging (fMRI) and positron emission tomography (PET) in combination. The anterior cingulate cortex, the posterior parietal cortex, and the subcortical regions of the reduced activity is most noticeable in the dorsolateral prefrontal cortex and the posterior cingulate cortex, as well as the amygdala, the parahippocampal gyrus, and the pontine tegmentum (Figure 2) [31]. The frequently absurd but intensely emotional nature of dreams occurring mostly in REM sleep may be explained by increased limbic system activity and decreased neocortical activity.

Debilitating sleep disorders may result from damage to the brain areas that activate REM. Patients with brainstem infarcts, for instance, had a considerably increased likelihood of developing REM behaviour disorder, a type of parasomnia that leads to reduced muscular atonia and dream "acting out" [32] . However, it is unknown how REM behavioural disorder would affect functional recovery and outcomes after a stroke.

The cholinergic nuclei found in the pons-midbrain junction of the brainstem play a critical role in the reticular activating system's control of sleep-wake mechanisms. These cholinergic cells are components of a large, interconnected network, rather than a singular REM sleep “hub”. Stimulating cholinergic nuclei within the pontine-midbrain junction causes a shift of EEG activity from high amplitude, synchronised slow-waves to lower amplitude, high-frequency desynchronization similar to that of awakening and REM [33]. These findings suggest that cholinergic projections within the reticular

activating system are the primary source of wakefulness and REM sleep, and deactivation to these net works is essential for initiation of slow-wave (NREM 3) and other NREM sleep stages. However, the neuronal basis for wakefulness and NREM sleep extends beyond these cholinergic neurons [25].

A spectrum of sleep-wake functioning, including deep sleep (such as slow-wave sleep) and high degrees of alertness (such as sympathetic nervous system activation), is also attributed to monoaminergic network activity (Table 1).

| Brainstem nuclei responsible | Neurotransmitter involved | Activity state of the relevant brainstem neurons |
|---|---|---|
| **Wakefulness** | | |
| Cholinergic nuclei of pons-midbrain junction | Acetylcholine | Active |
| Locus coeruleus | Norepinephrine | Active |
| Raphe nuclei | Serotonin | Active |
| **NREM Sleep** | | |
| Cholinergic nuclei of pons-midbrain junction | Acetylcholine | Decreased |
| Locus coeruleus | Norepinephrine | Decreased |
| Raphe nuclei | Serotonin | Decreased |
| **REM Sleep Activation** | | |
| Cholinergic nuclei of pons-midbrain junction | Acetylcholine | Active (PGO waves) |
| Raphe nuclei | Serotonin | Inactive |
| **REM Sleep Inhibition** | | |
| Locus coeruleus | Norepinephrine | Active |

**Tabe 1.** [10,34] Summary of the cellular mechanisms that govern sleep and wakefulness. Purves et al. (2010)

## SLEEP DYSFUNCTION AFTER STROKE

After ischaemic stroke, a cascade of physiological responses begin due to restriction of blood flow that affects the recovery of cerebral tissue, particularly of the ischaemic penumbra and distal networks. Excitotoxicity, a pathological process by which several of these processes occur, lead to overexpression of excitatory neurotransmitters like glutamate leading to neuronal death [35-37]. According to preclinical research, sleep deprivation (forced wakefulness) reduces the expression and function of neurotransmitter receptors as well as synaptic and membrane excitability in hippocampus neurons [38] . Lack of sleep has been demonstrated to increase the levels of glutamate receptors and extracellular glutamate, which furthers the excitotoxicity caused by ischaemia. Similarly, long-term sleep deprivation may decrease antioxidative stress markers such as glutathione peroxidase and superoxide dismutase which may also intensify neuronal damage via free radical generation [39]. Moreover, following a stroke prolonged sleep deprivation aggravates proinflammatory responses [40]. Sleep appears to have a neuroprotective role in the acute stages of stroke, according to newly revealed mechanisms underlying its function and the effects of sleep deprivation on neuronal injury, such as stroke [41, 42] . Study by Gao et al. (2010) [43] on effects of sleep deprivation on size of infarcts in rats showed sleep deprivation after ischaemia impairs neuroplasticity and is associated with increased lesion volumes. Inversely, sleep deprivation prior to ischaemia initiates compensatory sleep rebounding effects which may be neuroprotective [44]. Novel work by Xie et al. (2013) has shown that sleep serves a vital role maintaining brain metabolic homeostasis through activation of the glymphatic system[45] . Unlike the peripheral lymphatic system which utilizes lymph vessels to recirculate excess interstitial proteins into the liver for degradation, the glymphatic system of the central nervous system contains perivascular channels formed by astroglial cells to facilitate elimination of soluble proteins and metabolites [46-48]. During slow-wave-sleep, a 60% increase in interstitial space causes CSF to recirculate and flood through the brain parenchyma to perivenous drainage pathways . In turn, convective exchanges of CSF with interstitial fluid dramatically increase the removal rate of potentially toxic interstitial metabolic proteins.

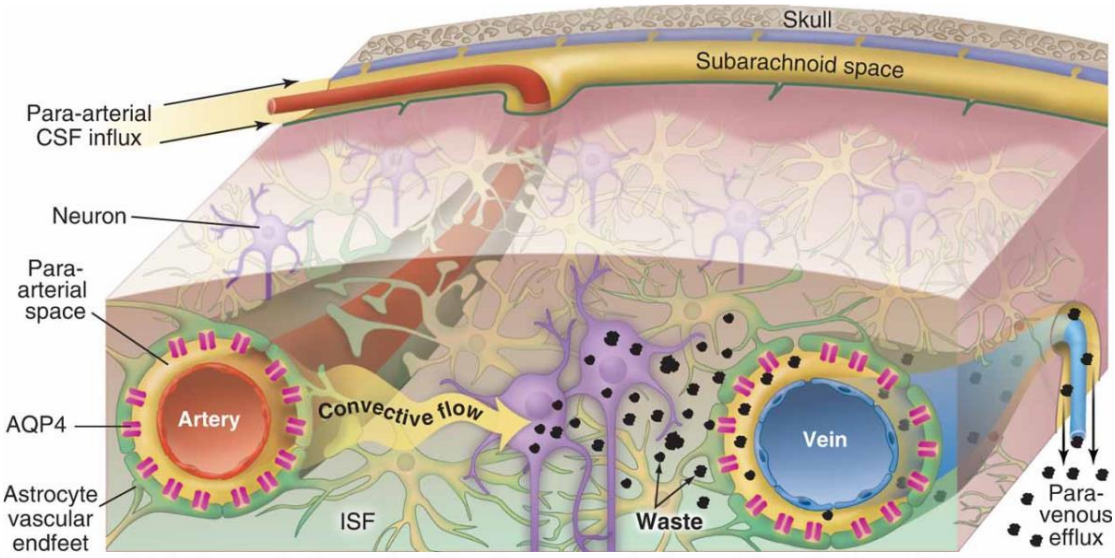


**Figure 3.** [47] **Schematic outline of the glymphatic system.** Convective glymphatic fluxes of cerebrospinal fluid (CSF) and interstitial fluid (ISF) propel the waste products of neuron metabolism into the paravenous space, from which they are directed into lymphatic vessels and ultimately return to the general circulation for clearance by the kidney and liver [47].

Experimental deletion of AQP4 channels responsible for glymphatic perfusion reduces clearance of exogenous Aβ by 65%, suggesting that convective movement of interstitial fluid is a substantial contributor to the removal of interstitial waste products and other products of cellular activity [46]. Therefore, chronic sleep deprivation may be a factor in the aggregation of A Beta collection, especially in late adulthood, as demonstrated by recent experimental results showing 80–90% reduction in glymphatic flow in ageing mice [49].

Ischaemic stroke causes severe acute impairment of glymphatic perfusion 24 hours after ischaemia [50]. Glymphatic disruption may promote excitotoxicity of surrounding brain penumbra and prevent acute clearance of excitatory neurotransmitters. As previously described, a surge of excess glutamate is secreted after stroke (a process known as excitotoxicity) and elicits a myriad of signalling cascades that work synergistically to induce neuronal death [51]. According to Gaberel et al. (2014), the mechanisms responsible for glymphatic blockading after ischaemia may include a reduction of arterial pulsation as a result of vessel occlusion and compression by intravascular thrombus [50]

## Clinical sleep-wake disturbances and disorders in stroke

Sleep-wake disorders are proposed to be both a risk factor and a consequence of stroke that may affects stroke recovery [52].

## SDB & Stroke

Most well studied sleep dysfunction after stroke being sleep disordered breathing. There is ample literature suggesting that SDB can be a risk factor for stroke and has an adverse impact on neurological outcome [1]. In a meta-analysis by Johnson KG, included total 29 studies consisted of 2,300 patients of ischaemic stroke, haemorrhagic stroke, or TIA, 72% had SDB defined by an apnoea-hypopnea index of >5/hr [43]. More than 50 % of these patients had AHI of > 10 / hr at 4 weeks post stroke. Although OSA has long been recognised as the most prevalent kind of SDB, stroke patients recently were discovered to have combination of OSA & central types of SDB, including central sleep apnea and Cheyne-Stokes breathing, as well as combinations of both [53]. In addition, meta-analyses of prospective clinical and population-based research have found that SDB is an independent predictor of stroke, with stroke risk escalating with AHI [54]. Studies investigating the factors that increase the risk of stroke in people with SDB have revealed that persistent hypoxia in OSA alters intrathoracic pressure, activates the sympathetic nervous system, and increases blood pressure surges that may put people at risk for drug-resistant arterial hypertension, atherosclerosis, cardiac arrhythmia, hypercoagulation, heart failure, and paradoxical embolisms via oxidative stress and brain inflammation. [52,55, 56]

## Hypersomnia and Insomnia in ischaemic stroke

Hypersomnia is usually seen after pontomesencephalic stroke. It is characterised by excessive daytime sleepiness as measured by the Epworth Sleepiness Scale (score >8) or more than 10 hrs sleep per day. Significant fatigue as determined by the Fatigue Severity Scale, which was reported by nearly half of participants with a score of >4.0 [53]. Study on fatigue after stroke [57] showed constant fatigue many years after stroke.

Systematic review & metanalysis on sleep duration & cerebrovascular outcomes in 2011 [58] showed longer sleep duration is an independent predictor of incident stroke, after adjustments for age, sex, vascular risk, and attributing comorbidities. Long sleep durations (more than nine hours) have also been associated with subcortical white matter hyperintensities in adults, suggesting that excessive sleep may be caused by cerebral small vessel disease and reflect subclinical atherosclerosis [59].

Inversely, insomnia is also common in the months following stroke and is found in 30-50% of sufferers [52]. Post-stroke insomnia is associated with poor life satisfaction, depression, and stroke severity [53,60,61].

Few researchers have looked into sleep architecture in patients with insomnia after stroke. It has been noted that a stroke affecting the paramedian thalamic nuclei leads to near complete absence of sleep spindles [62]. Neuroplasticity and stroke recovery have been demonstrated to be better when drugs promoting NREM & REM sleep are used [63]. However similar data from human studies is lacking. Similar to results from hypersomnia literature, authors of recent meta-analyses have found that short sleep, characteristic of insomnia (defined by < 5-6 hours of sleep/night) is also an independent predictor of incident stroke after adjustment for age, sex, vascular risk factors, and comorbidities. These findings show that there may be a "U" based relationship between sleep duration and incident stroke, with increased risk resulting from insufficient (i.e., < 5-6 hours) or excessive (i.e., > 9 hours) sleep [64,65].

However qualitative sleep parameters (e.g., sleep latency, circadian stability, nighttime awakenings, day-time naps, post-awakening latency, and sleep architecture etc) are not assessed in these prospective studies.

A recent systematic review on bidirectional impact of sleep & circadian rhythm dysfunction in human ischemic stroke revealed long sleep duration and sleep disorders increase the risk of developing ischemic stroke . Inversely, after IS, sleep and endogenous rhythm disruption is common and may be associated with IS severity and outcome [66].

## STROKE & PARASOMNIA

Parasomnias are complex movements and behaviors during sleep which include REM sleep behavior disorder (RBD), nightmares, sleep paralysis, and other disorders of arousal including sleepwalking and sleep terrors. RBD is characterised by dream-enacting behaviours and vivid or unpleasant nightmares, and it is typically observed in neurodegenerative diseases like Parkinson's disease and multiple systems atrophy [95]. Studies have established that individuals with RBD have a higher likelihood of also having concomitant stroke risk factors including diabetes mellitus and dyslipidemia. One study demonstrated that adults with RBD were 1.5 times more likely to develop stroke independent of other demographic variables including age, gender, hypertension, and tobacco use.[96] It is possible that sleep fragmentation, which causes an increase in sympathetic tone and alterations in heart rate variability and blood pressure surges, is the mechanism through which RBD and stroke occur.

Pontine tegmental strokes have been reported in conjunction with REM sleep behavioural abnormalities (RBD)[97,98]. Visual hallucinations may be brought on by lesions in the pontine tegmentum, midbrain, or paramedial thalamus, particularly in the evening or when sleep first begins [99] . Increased dreaming, nightmares, and/or a dream-reality confusion may result after thalamic, temporal, parietal, and occipital lobe strokes[73] Clonazepam (0.5–2 mg) can be used to treat RBD one hour before the patient goes to bed [99]

### Stroke and periodic limb movement disorder during sleep

RLS is a condition of the nervous system that causes frequent urges to move the legs. This usually causes jerking movements of the legs and arms during sleep, known as Periodic Limb Movements in Sleep (PLMS). The presence of PLMS can be painful and disrupt the normal patterns of sleep leading to increased wakefulness during the night Periodic limb movement (PLM) during sleep may increase or decrease after a unilateral ictus and persist after a spinal cord stroke [100, 101].

In a cohort of 137 stroke survivors recruited while they were in the hospital, Lee et al. (2009) discovered that 12.4% of them had stroke-related RLS, which was categorised as a first-time diagnosis of RLS at one month after the stroke. Similar to this, 12.5% of a longitudinal stroke group had RLS. (Medeiros et al., 2010) of 96 patients. None of these patients had previously received a medical diagnosis of RLS prior to the stroke and all RLS patients had worse sleep quality and lower improvement on the functional recovery scores at 3 and 12 months post-stroke suggesting a potential relationship between poor sleep and functional recovery.

A 2015 study also found a similar prevalence of RLS with 15% of their 149 patients having the syndrome after stroke/ TIA in comparison to 3% of controls (Schlesinger, Erikh, Nassar & Sprecher, 2015).

The majority of recorded cases of RLS include the pontine, thalamic, basal ganglia, and lesions of the corona radiata. Two-thirds of RLS patients with a stroke report bilateral symptoms, while a third report symptoms on the contralateral side of the stroke.

Dopaminergic agonists may be used to treat stroke-related RLS and PLM. It's crucial to keep in mind that medication for depression may include neuroleptics, methoclopramide, and lithium may worsen PLM and RLS[(99)]

## CARE GIVER BURDEN POST STROKE

Caregiver burden can be defined as the strain that is experienced by a person who cares for a chronically ill, disabled, or older family member [(107)].With increasing life expectancy of the population and high prevalence of life style diseases, low and middle income countries are facing great social and financial challenges in coping with disabled stroke survivors [(106)]. In developing countries like India, hospital and community based rehabilitation facilities are limited. This puts the burden of caring stroke survivors mainly upon the family leading to high levels of caregiver burden. With spouse being the major caregiver, their employment and social function also suffers on the long run. Due to the abrupt onset of disability and the chronic nature of stroke recovery, caring for a stroke survivor has been found to have a negative impact on the physical, mental, and psychological health of caregivers [(108, 109)]. Primary caregivers of stroke patients tend to report more somatic and depressive symptoms, sleep disorders, stress and social isolation

than general population [110]. Various factors found to influence care giver burden from previous studies include degree of dependence of the patients for the daily living activities [75], gender (women expressing higher burden) [94], older patients [74,94], post stroke seizures [94]. A recent study assessing role of sleep deprivation among care givers of stroke patients found sleep duration of less than 5 hrs per day lead to higher care giver burden & psycological stress [111] . Sleep disordered breathing (SDB) & insomnia after stroke are found to affect stroke recovery which leads to continued burden on care givers.

**ASSESING THE SLEEP DYSFUNCTION AFTER STROKE**

Although a polysomnography (PSG) is the gold standard for diagnosis of or differentiating sleep disorders, PSGs cannot be performed on all stroke patients in practice because of the high price and limitation of accessibility; additional tools that can screen for sleep disorders, such as valid sleep questionnaires, are needed. Sleep questionnaires are very commonly used in sleep literature, they provide a useful insight into the perception of sleep quality and are commonly used in screening for sleep dysfunction . Many studies investigating sleep and stroke have used questionnaire data as a measure of sleep quality, including a number of studies that have found that patients report their sleep quality as worse than controls (Chen et al., 2015; Jiang et al., 2013; Wu et al., 2016).

1. Epworth Sleepiness Scale (ESS) [67] - Dr Murray Johns first developed the ESS for adults in 1990 and subsequently modified it slightly in 1997. He developed it so he could assess the ‘daytime sleepiness’ of the patients in his own private practice of Sleep Medicine. He named the questionnaire after Epworth Hospital in Melbourne, where he established the Epworth Sleep Centre in 1988. The ESS asks the respondent to rate on a 4-point scale (0-3) their usual chances of having dozed off or fallen asleep while engaged in eight different activities that differ widely in their somnificity. Respondents to the ESS rate their chances of having dozed off or fallen asleep in particular situations ‘in recent times’. It was intended to be long enough for the respondent to have experienced at least most of the activities, so they could estimate in retrospect their chances of dozing in each.

Because ESS item-scores are based on subjective reports, they can be influenced by the same sources of bias and inaccuracy as any other such reports. The ESS does not distinguish which factors, or which sleep disorders, have caused any particular level of ASP. The ESS is not a diagnostic tool by itself. The ESS is not suitable for use among people with serious cognitive impairment. Thus, 'in recent times' was intended to mean a few weeks to a few months, not a few hours or days. Epworth Sleepiness Scale (ESS) use in the South Indian population was validated by previous study as a tool for measuring excessive day time sleepiness [(102)] . In addition it was found that EDS assessed by ESS positively correlated with the severity of OSA. In narcolepsy, the Epworth Sleepiness Scale has both a high specificity (100%) and sensitivity (93.5%)[(103)] . Previous study validating the use of ESS in stroke patients found that it's a robust scale for detecting cases of pathological sleepiness in stroke [(104)].

**2.** Insomnia severity index : [(68)]

The Insomnia Severity Index (ISI), developed by Morin, is currently one of the most used PRO insomnia questionnaires. The ISI is a brief seven item self-rated instrument, increasingly used to assess insomnia based on criteria from the International Classification of Sleep Disorders. The ISI has been translated into multiple languages, validated in 12 countries and as a web-based measurement . In comparison to other PRO sleep measures, the ISI has diagnostic properties [(69)], and can be completed in a few minutes , diminishing the response burden. Of the few insomnia-specific PRO instruments available, the ISI is designed to capture patient-perceived insomnia severity and impact on daytime functioning. Patients are asked answer questions as per their symptoms in the last 2 weeks. With cutoff score of 10, the scale has a 86.1% sensitivity and 87.7% specificity for detecting insomnia cases in the community sample [(105)]. Many studies investigating insomnia & stroke use ISI questionnaire found that, insomnia is common in the months following stroke and is found in 30-50% of sufferers [(52)]. Post-stroke insomnia is associated with poor life satisfaction, depression, and stroke severity [(53,60,61)].

**3.** Pittsburgh Sleep quality index(PSQI) (70)

The PSQI was developed by Daniel J. Buysse and collaborators in 1988 to measure quality of sleep and to help discriminate between individuals who experience poor sleep versus individuals who sleep well. The scale has several domains, which include subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medication, and daytime dysfunction. The scale has two parts: self-rated questions, utilized to rate the scale, and five questions rated by a bed partner. The scale can also be given by a clinician or research assistant. Most of the items are organized in multiple choice questions and are brief and easy to understand and answer.

The PSQI questions are rated from 0 = no difficulty to 3 = severe difficulty, generating scores that correspond to the domains of the scale. The scores range from 0 to 21 and the authors suggest that a score >5 be considered as a significant sleep disturbance. Time to complete PSQI scale: 5–10 min.

The reliability of the scale is considered good with Cronbach's alpha of 0.83 for the total score. Test–retest reliability is also considered good. The validity of PSQI has been described by the authors as good with a sensitivity of 89.6% and a specificity of 86.5% of patients versus control subject. Studies using PSQI to asses sleep quality after stroke[73, 2, 87, 88] have reported prevalence of 32 to 71 %.

4. Patient health questionnaire 9(PHQ-9) [71]

The PHQ-9 is a brief tool used to diagnose and measure severity of depression. The PHQ-9 was developed by Dr Robert L. Spitzer, Janet W.B. Williams and Kurt Kroenke in 1999. The PHQ-9 is shorter than many of the other depression screening instruments and can be self-administered. Adapted from the Diagnostic and Statistical Manual of Mental Disorders, 4th edition (DSM-IV), the PHQ-9 includes all 9 diagnostic symptom criteria used in the DSM-IV, including the two cardinal signs of depression: anhedonia and depressed mood. The PHQ-9 is widely used by clinicians including in patients with stroke. Previous studies assessing post stroke depression [74, 2] have used

PHQ 9 with score cut off ≥ 10 to define depression with estimated prevalence of 30-35 %.

5. Zarit's care giver burden [72]

   The Zarit Burden Interview, a popular caregiver self-report measure used by many aging agencies, originated as a 29-item questionnaire (Zarit, Reever & Bach-Peterson, 1980). The 29-item instrument is included in Zarit et al., 1980. The revised version contains 22 items. Translations of the Zarit Burden Inventory have been studied as well, including versions in Chinese, French, Japanese, German, Hebrew, Spanish, Korean, Hindi, and Portuguese. Each item on the interview is a statement which the caregiver is asked to endorse using a 5-point scale. Response options range from 0 (Never) to 4 (Nearly Always).

**POLYSOMNOGRAPHY**

PSG studies allow for very detailed observations of the state of the brain and cardiorespiratory functions during periods of sleep. By using Electroencephalography (EEG) to monitor the brain, it is possible to measure not only the duration of sleep, but also the time spend in each sleep stages. Level 1 sleep testing, or polysomnography, requires an overnight stay in a sleep laboratory with a technician in attendance. It captures 7 or more channels of data (but typically ≥ 16), including respiratory, cardiovascular and neurologic parameters, to produce a comprehensive picture of sleep architecture. Level 1 is considered the reference standard for diagnosing all types of sleep-disordered breathing and sleep disorders [115,116,117] . However, limited facilities and the growing demand for sleep studies have resulted in long wait times[118] . Level 2 sleep testing uses level 1 equipment, but is performed without a technician in attendance.

Level 3 testing uses portable monitors that allow sleep studies to be done at the patient's home or elsewhere. This option was introduced as a more accessible and less expensive alternative to in-laboratory polysomnography. Level 3 devices record at least 3 channels of data (e.g., oximetry, airflow, respiratory effort). Unlike level 1, level 3 testing cannot

measure the duration of sleep, the number of arousals or sleep stages, nor can it detect non respiratory sleep disorders. Level 4 devices are also portable, but they capture less data — usually only 1 or 2 channels.

A systematic review and meta-analysis of PSG papers investigating sleep after stroke found that sleep quality is worse for stroke survivors in a number of measures [(112)]. Total Sleep Time (TST), Sleep Efficiency (SE) (the proportion of time in bed, spent asleep) and Wake After Sleep Onset (WASO) were all found to be reduced in stroke groups in comparison to controls. When looking specifically at sleep stages the meta analyses found the duration of stage 2 sleep, SWS and REM sleep to be significantly reduced in stroke patients when compared to controls (Stage 2 = 36% vs 45%, SWS = 10% vs 12%). Although the differences in TST, SE and WASO between groups was found regardless of the participants' sleeping environment, the difference in SWS and REM duration was only found when comparing hospitalised stroke patients with hospitalised controls. As the controls assessed in those studies were assessed in sleep labs, it would suggest that the reduction in deep sleep and REM could be due to hospitals being a specifically detrimental environment to normal sleeping as opposed to just being purely novel environments. Interestingly, when comparing the duration of Stage 1 sleep, stroke patients show a
significantly larger percentage in relation to their whole night's sleep than controls (13% vs 10%) [(112)]. This suggests that stroke could affect specific parts of sleep. architecture, reducing the amount of deep and REM sleep in exchange for larger periods of light sleep. This could be particularly detrimental to stroke patients' rehabilitation and recovery given that stage 2 sleep, SWS and REM have all been shown to be connected to memory consolidation leading to improved skill performance [(113)]. Specifically, levels of stage 2 sleep correlate with improved performance on a motor task and levels of SWS and REM sleep correlate with improved performance on a visual discrimination task.

Van Someren (2007) [(114)] investigated the reliability of actigraphy recording in insomnia over multiple nights. They concluded that multiple nights recordings are needed when investigating insomnia due to the high variability in sleep and sleep-wake rhythms within this cohort. Therefore, PSG studies recording for only one night such as Terzoudi et

al.(2009), although richer in detail than actigraphy, may not be collecting representative data on the sleeping patterns of stroke survivors with sleep problems. Meaning that further research with longer periods of sleep recordings is needed to reliably measure sleep problems.

Systematic review and meta-analysis done to compare the diagnostic accuracy of the widely used level 3 portable monitors to in-laboratory polysomnography showed no significant difference in the clinical management parameters between patients who underwent either test to receive their diagnosis [119].

# MATERIALS AND METHODS

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## INTRODUCTION

This was a prospective observational study conducted after obtaining the institutional ethics committee approval (IEC number : SCT/IEC/1805/JANUARY/2022). Informed written consent was obtained from all the participants who were part of this study. The study period was from January 2022 to March 2023,including a 3 month telephonic follow-up.

After screening for inclusion/exclusion criteria,subjects were selected for the study from stroke outpatient clinic, during their 3 month follow-up review after stroke. Demographics, risk factors and clinical characteristics were collected as per proforma appended. The severity of stroke was recorded using National Institute of Health Stroke scale score (NIHSS). All the patients/caregivers of those who had communication difficulties were administered 5 questionnaires - Epworth sleepiness scale for assessing excessive day time sleepiness, Insomnia severity index to assess the severity of insomnia and Pittsburgh Sleep quality index(PSQI) for overall sleep health for assessing their sleep post stroke in the last 1 month before interview. In addition, stroke functional and psychological outcome were assessed using Modified Rankin Scale (mRS) and Patient health questionnaire 9(PHQ-9) respectively. Caregiver burden scale(Zarit's)(CBS) was used to assess the impact of stroke on care givers. 20 % of participants (20 patients) underwent overnight ambulatory polysomnography . Scores from screening tools about sleep quality, functional recovery, depression, care giver burden post stroke were compared with patient parameters to find predictors of sleep dysfunction . All the patients

were followed up at 3 months after recruitment, i.e 6 post stroke by telephone, to assess functional recovery assessed using Modified Rankin Scale (mRS).

**INCLUSION CRITERIA**

1. Acute ischemic stroke patients aged 18 or above
2. Coming for follow-up at 3 months post stroke
3. Functional status by mRS below 5
4. Consenting to participate in the study

**EXCLUSION CRITERIA**

1. Intracerebral hemorrhage
2. Severe organ dysfunction-like Cardiac failure/Cor pulmonale/liver or renal failure or cancer which by itself can impact sleep
3. Prior history of depression/psychiatric illness/primary sleep disorders requiring treatment before stroke/had been on psychiatric medications before stroke.

**STUDY DESIGN**

Ours was a prospective observational study.

With around 400 admissions annually in stroke unit and 80% being ischemic strokes, sample size of 130 was calculated assuming prevalence as 30% & setting alpha error at 5% & absolute precision of ± 5% . However we could collect 100 patients in the study period. The recruitment of subjects for the prospective study was done by the principal investigator from consecutive patients who reported to Stroke Clinic, SCTIMST at 3 months post stroke.

The study period was from January 2022 to March 2023.

Consent for inclusion in the study was obtained from appropriate persons or guardians. No inclusion of person incompetent to give informed consent, normal/healthy volunteer, Prisoner, student/staff of the institute was done. Clinical information was obtained through patient interviews and supplemented with a chart review.

**FLOW CHART OF STUDY DESIGN** (Fig 4)

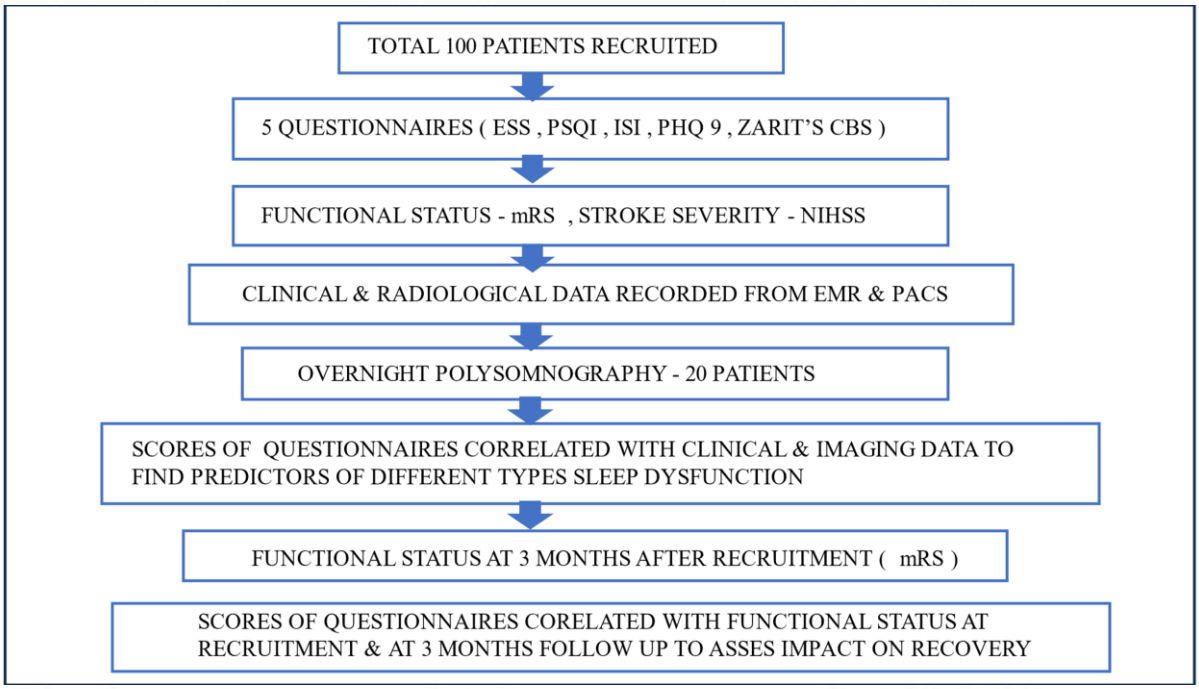



**Fig. 4: Flow chart of study design**

## STUDY PROTOCOLS

Patients satisfying the inclusion criteria were selected for the study. The severity of stroke was recorded using National Institute of Health Stroke scale score (NIHSS). All the patients were administered 5 questionnaires

1. Epworth sleepiness scale (ESS)
2. Insomnia severity index (ISI)
3. Pittsburgh Sleep quality index(PSQI)
4. Patient health questionnaire 9(PHQ-9)
5. Caregiver burden scale(Zarit's)(CBS)

In addition 20 % of participants (20 patients) underwent overnight ambulatory polysomnography (level 3),to rule out sleep disordered breathing.

## QUESTIONAIRES:

**1.** Epworth Sleepiness Scale (ESS) [67]

Scoring :

Divided into 2 categories

1) Less than 8 – no excessive daytime sleepiness
2) 8 or more - excessive daytime sleepiness

**2.** Insomnia severity index : [68]

Scoring :

Divided into 2 categories

1) Less than 8 – no excessive daytime sleepiness
2) 8 or more - excessive daytime sleepiness

**3.** Pittsburgh Sleep quality index(PSQI) [70]

Scoring :

Divided into 2 categories

1) Less than 5 – good sleep quality
2) 5 or more – significant sleep disturbance

4. Patient health questionnaire 9(PHQ-9) [71]

Scoring :

Divided into 2 categories

1) Less than 10 – no significant depression
2) 10 or more – significant depression

5. Zarit's care giver burden [72]

Scoring :

Divided into 2 categories

1) Less than 21 – no significant caregiver burden
2) 21 or more – significant caregiver burden

Scores from sleep questionnaires were compared with various clinical & laboratory patient parameters to find predictors of sleep dysfunction . All the patients were followed up at 3 months after recruitment, to assess its impact on stroke recovery assessed using Modified Rankin Scale (mRS).

**STATISTICAL ANALYSIS**

Statistical analysis was done using SPSS 24 analysis software. In addition to descriptive statistical tools for basic frequencies of patient characteristics and demographics, a chi-square test & student's t test were used to asses risk factors for sleep dysfunction . p-value < 0.05 was defined as statistically significant. Binary logistic regression model was used for multivariate analysis.

# RESULTS

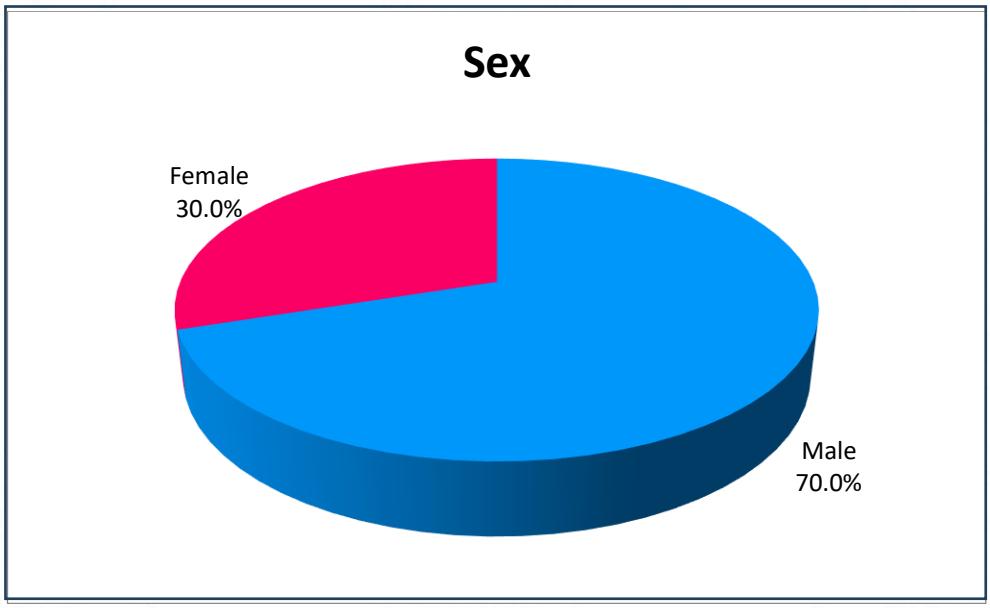
# RESULTS

As part of the study, a total of 100 study subjects were recruited. All patients completed 3 months telephonic follow up after recruitment.

## COHORT CHARACTERISTICS

70 % of the subjects were male. The age of the patients ranged from 28-86 years with median age 62.2 ± 11.2 years. The minimum age was 28 years and the oldest patient was 86 years in the study population.

FIG 5 : SEX OF PATIENTS




Risk factor profile :
Among the subjects, 77 % had hypertension, 64 % had diabetes, 72% had dyslipidaemia, 33 % had coronary artery disease, 30 % had prior stroke, 14 % had prior TIA, 3 % had PVOD, 15 % had renal dysfunction, 26 % had alcoholism & smoking history

TABLE 2 : RISK FACTORS & COMORBIDITIES

| | Frequency | Percent |
|---|---|---|
| HTN | 77 | 77 |
| DM | 64 | 64 |
| CAD | 33 | 33 |
| Prior Stroke | 30 | 30 |
| Prior TIA | 14 | 14 |
| Dyslipidemia | 72 | 72 |
| POVD | 3 | 3 |
| Renal Dysfunction | 15 | 15 |
| Valvular heart disease | 7 | 7 |
| Smoking | 26 | 26 |
| Alcoholism | 24 | 24 |

FIG 6 EDUCATION

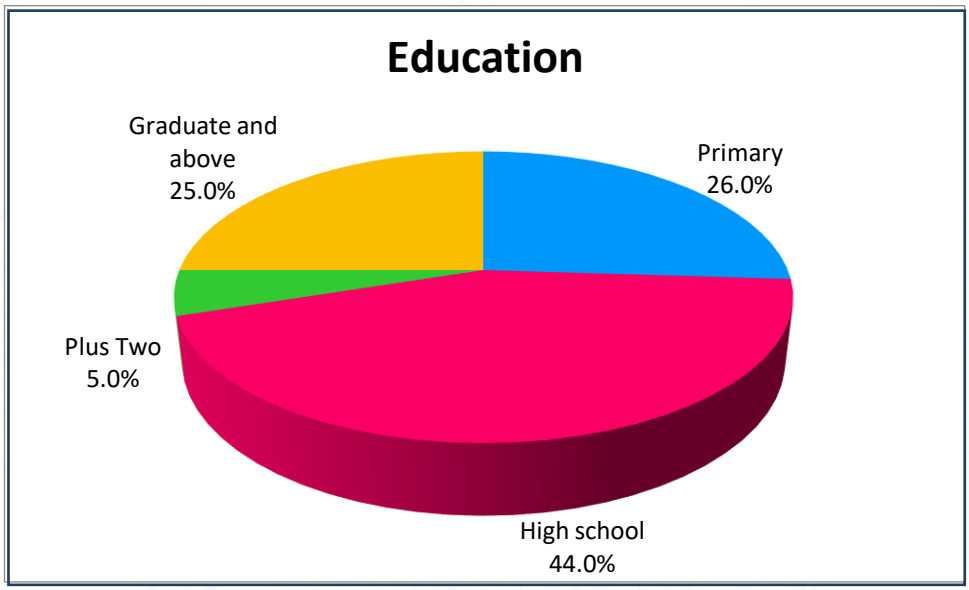


TABLE 3 ETIOLOGY OF STROKE

| Etiology | Frequency | Percent |
|---|---|---|
| Lacunar | 26 | 26 |
| LVAD | 45 | 45 |
| Cardioembolism | 12 | 12 |
| Undetermined | 17 | 17 |
| Total | 100 | 100 |

Etiology of stroke : Large artery atherosclerosis was the most common etiology (45 %) followed by lacunar strokes (26 %)

TABLE 4 STROKE SEVERITY (NIHSS)

| NIHSS | Frequency | Percent |
|---|---|---|
| Mild (1-4) | 33 | 33 |
| Moderate (5-15) | 50 | 50 |
| Severe (>15) | 17 | 17 |
| Total | 100 | 100 |

TABLE 5 NIHSS & mRS scores

| | N | mean ± sd | Range | Median | IQR |
|---|---|---|---|---|---|
| Age | 100 | 62.2 ± 11.2 | 28 - 86 | 62 | 56 - 69 |
| mRS at admission | 100 | 3.35 ± 1 | 0 - 4 | 4 | 3 - 4 |
| mRS at 3 months | 100 | 1.83 ± 1.54 | 0 - 4 | 2 | 0 - 3 |
| NIHSS at admission | 100 | 8.3 ± 6.24 | 0 - 24 | 7.5 | 3 - 12 |
| NIHSS at discharge | 100 | 4.45 ± 4.28 | 0 - 17 | 3 | 1 - 7 |
| NIHSS at 3 months | 100 | 2.61 ± 3.25 | 0 - 15 | 1 | 0 - 5 |

Admission NIHSS was ranging from 0-24 with a mean of 8.3 ± 6.24. Based on NIHSS, minor strokes with NIHSS less than 5 were 33 %, moderate strokes of NIHSS 5-15 was seen in 50 %, and severe strokes with NIHSS >15 were seen in 17 % (17) cases only.

TABLE 6 : FUNCTIONAL STATUS (mRS)

| MRS | At Admission | | At 3 months | | At 6 months | |
|---|---|---|---|---|---|---|
| | n | % | n | % | n | % |
| ≤2 | 21 | 21 | 59 | 59 | 66 | 66 |
| >2 | 79 | 79 | 41 | 41 | 34 | 34 |
| Total | 100 | 100 | 100 | 100 | 100 | 100 |

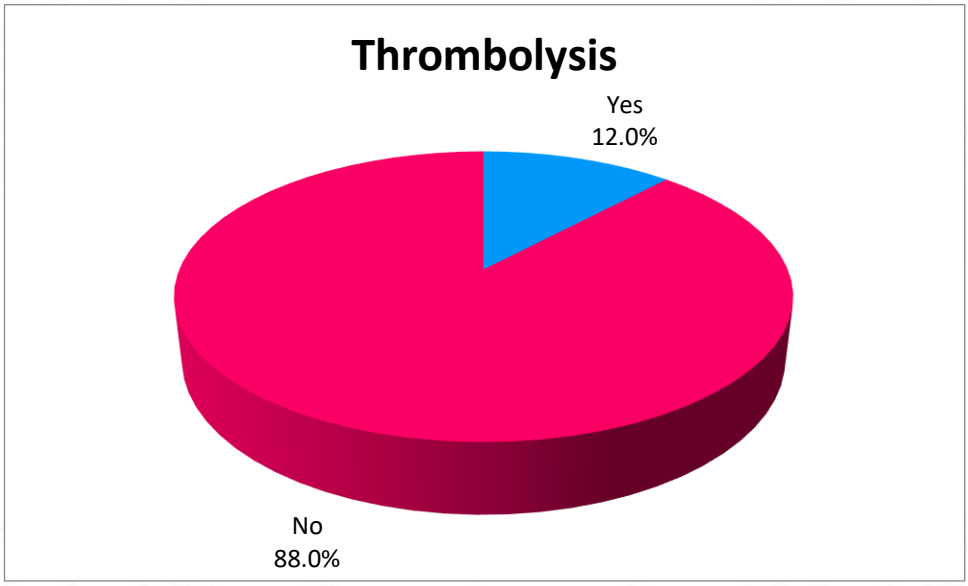
mRS at admission ranged from 0-4, mean of 3.35 ± 1.
mRS at 3 months ranged from 0-4, mean of 1.83 ± 1.54
mRS at 6 months ranged from 0-3, mean of 1.3 ± 0.6

TABLE 7 : IMAGING DATA

| **Territory** | Frequency | Percent |
|---|---|---|
| MCA | 64 | 64 |
| ACA | 4 | 4 |
| Posterior circulation | 27 | 27 |
| MCA + ACA | 1 | 1 |
| MCA + Posterior circulation | 4 | 4 |
| Total | 100 | 100 |
| **Circulation affected** | | |
| Right Hemispheric | 39 | 39 |
| Left Hemispheric | 30 | 30 |
| Posterior Circulation | 27 | 27 |
| Bihemispheric | 4 | 4 |
| Total | 100 | 100 |
| **Haemorrhagic Transformation** | | |
| HI 1 | 12 | 12 |
| HI 2 | 10 | 10 |
| PH 1 | 1 | 1 |
| PH 2 | 0 | 0 |
| No HT | 73 | 73 |
| Total | 100 | 100 |
| **White matter hyperintensities (Fazeka)** | | |
| Grade 0 | 20 | 20 |
| Grade 1 | 44 | 44 |
| Grade 2 | 32 | 32 |
| Grade 3 | 4 | 4 |
| Total | 100 | 100 |

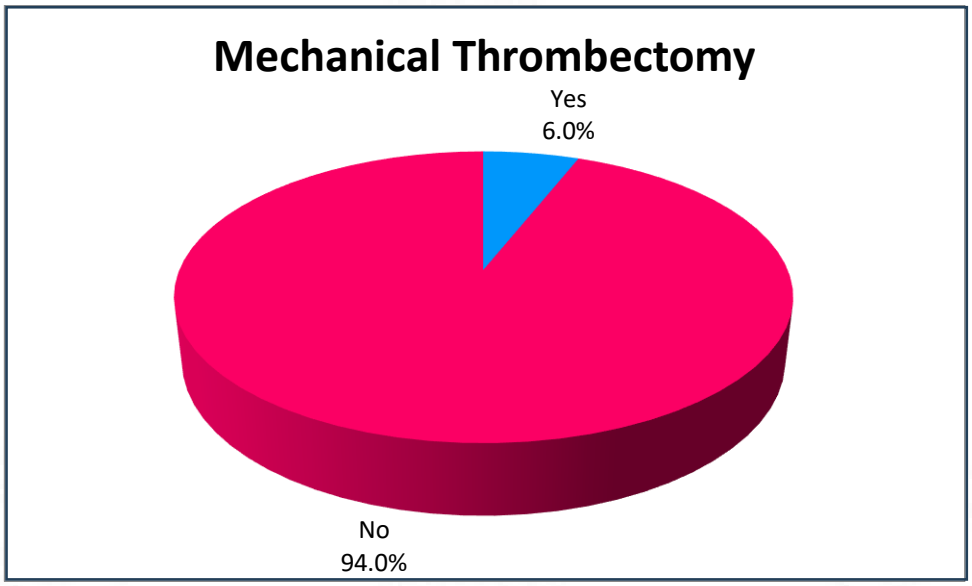
Territory of stroke: 64 % were MCA territory, 27 % posterior circulation, 4 % ACA territory. Circulation affected: 39 % strokes were right hemispheric & 30 % were left hemispheric Haemorrhagic Transformation: 73 % patients didn't had haemorrhagic transformation, 12 % had HI 1 , 10 % had HI 2 & 1 patient had PH 1 haemorrhagic transformation Small vessel ischemic changes (Fazeka) : 44 % had grade 1 Fazeka changes.

FIG 7 : IV THROMBOLYSIS



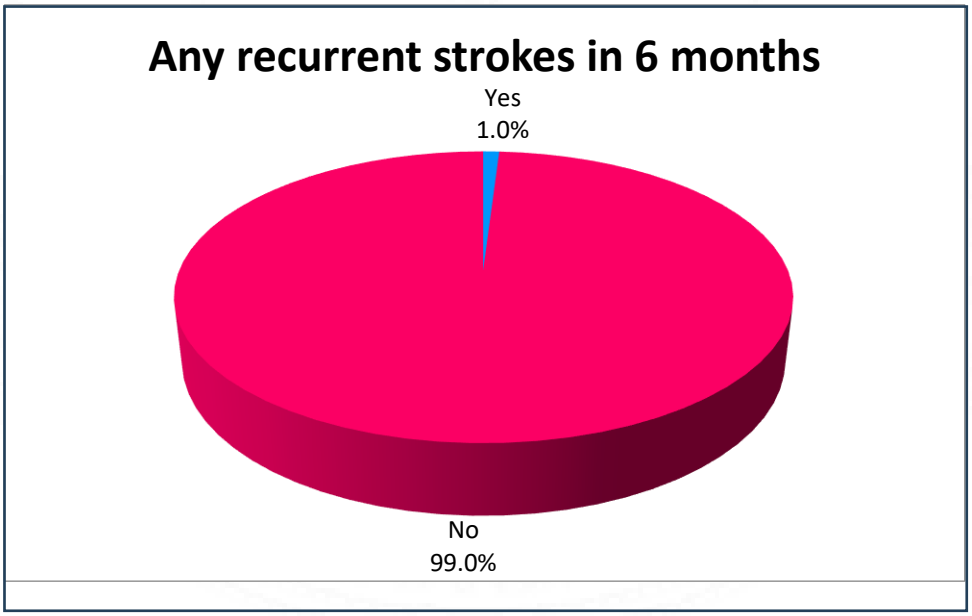

12 % patients received Iv Thrombolysis

FIG 8 : MECHANICAL THROMBECTOMY




6 % patients underwent mechanical thrombectomy

FIG 9 : RECURRENT STROKES




1 % (1) patient had recurrent stroke in 3 month follow up

**Sleep Questionnaire Scores :**

TABLE 8 : HYPERSOMNOLENCE AFTER STROKE AS MEASURED BY ESS

| ESS | Frequency | Percent |
|---|---|---|
| <8 | 54 | 54 |
| 8-9 | 26 | 26 |
| 10-15 | 20 | 20 |
| Total | 100 | 100 |

46 % patients had hypersomnolence

TABLE 9 : INSOMNIA (ISI)

| Insomnia | Frequency | Percent |
|---|---|---|
| <8 | 65 | 65 |
| 8-14 | 23 | 23 |
| 15-21 | 12 | 12 |
| Total | 100 | 100 |

35 % patients reported insomnia

TABLE 10 : SLEEP QUALITY (PSQI)

| PSQI | Frequency | Percent |
|---|---|---|
| <5 | 60 | 60 |
| 5-21 | 40 | 40 |
| Total | 100 | 100 |

40 % patients had poor sleep quality

TABLE 11: SELF REPORTED DEPRESSION (PHQ 9)

| PHQ9 | Frequency | Percent |
|---|---|---|
| <10 | 55 | 55 |
| 10-14 | 40 | 40 |
| 15-19 | 4 | 4 |
| 20-27 | 1 | 1 |
| Total | 100 | 100 |

45 % patients reported significant depression

TABLE 12: CARE GIVER BURDEN SCALE (Zarit’s CBS)

| Zarit's CBS | Frequency | Percent |
|---|---|---|
| <20 | 78 | 78 |
| 21-40 | 20 | 20 |
| 41-60 | 2 | 2 |
| Total | 100 | 100 |

22 % of care givers expressed burden

TABLE 13: FACTORS PREDICTING HYPERSOMNOLENCE

| | ESS | | | | Total | | p |
|---|---|---|---|---|---|---|---|
| | <8 | | ≥8 | | | | |
| | N | % | N | % | N | % | |
| **Age** | | | | | | | |
| ≤60 | 26 | 48.1 | 19 | 41.3 | 45 | 45 | |
| >60 | 28 | 51.9 | 27 | 58.7 | 55 | 55 | 0.493 |
| **Sex** | | | | | | | |
| Male | 38 | 70.4 | 32 | 69.6 | 70 | 70 | |
| Female | 16 | 29.6 | 14 | 30.4 | 30 | 30 | 0.930 |
| **Education** | | | | | | | |
| Primary | 15 | 27.8 | 11 | 23.9 | 26 | 26 | |
| High school | 23 | 42.6 | 21 | 45.7 | 44 | 44 | |
| Plus Two | 0 | 0 | 5 | 10.9 | 5 | 5 | |
| Graduate and above | 16 | 29.6 | 9 | 19.6 | 25 | 25 | 0.070 |
| **Smoking** | | | | | | | |
| Yes | 9 | 16.7 | 17 | 37 | 26 | 26 | |
| No | 45 | 83.3 | 29 | 63 | 74 | 74 | 0.021 |
| **Alcoholism** | | | | | | | |
| Yes | 11 | 20.4 | 13 | 28.3 | 24 | 24 | |
| No | 43 | 79.6 | 33 | 71.7 | 76 | 76 | 0.357 |
| **HTN** | | | | | | | |
| Yes | 39 | 72.2 | 38 | 82.6 | 77 | 77 | |
| No | 15 | 27.8 | 8 | 17.4 | 23 | 23 | 0.219 |
| **DM** | | | | | | | |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Yes | 35 | 64.8 | 29 | 63 | 64 | 64 | |
| No | 19 | 35.2 | 17 | 37 | 36 | 36 | 0.854 |
| **CAD** | | | | | | | |
| Yes | 16 | 29.6 | 17 | 37 | 33 | 33 | |
| No | 38 | 70.4 | 29 | 63 | 67 | 67 | 0.437 |
| **Prior Stroke** | | | | | | | |
| Yes | 13 | 24.1 | 17 | 37 | 30 | 30 | |
| No | 41 | 75.9 | 29 | 63 | 70 | 70 | 0.161 |
| **Prior TIA** | | | | | | | |
| Yes | 3 | 5.6 | 11 | 23.9 | 14 | 14 | |
| No | 51 | 94.4 | 35 | 76.1 | 86 | 86 | 0.008 |
| **Dyslipidemia** | | | | | | | |
| Yes | 36 | 66.7 | 36 | 78.3 | 72 | 72 | |
| No | 18 | 33.3 | 10 | 21.7 | 28 | 28 | 0.198 |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| **Etiology** | | | | | | | |
| Lacunar | 17 | 31.5 | 9 | 19.6 | 26 | 26 | |
| LVAD | 22 | 40.7 | 23 | 50 | 45 | 45 | |
| Cardioembolism | 5 | 9.3 | 7 | 15.2 | 12 | 12 | |
| Undetermined | 10 | 18.5 | 7 | 15.2 | 17 | 17 | 0.436 |
| **Territory** | | | | | | | |
| Right Hemispheric | 17 | 31.5 | 22 | 47.8 | 39 | 39 | |
| Left Hemispheric | 17 | 31.5 | 15 | 32.6 | 30 | 32 | |
| Posterior Circulation | 19 | 35.2 | 8 | 13 | 27 | 25 | |
| Bihemispheric | 1 | 0 | 3 | 4.3 | 4 | 4 | 0.052 |
| **POVD** | | | | | | | |
| Yes | 0 | 0 | 3 | 6.5 | 3 | 3 | |
| No | 54 | 100 | 43 | 93.5 | 97 | 97 | 0.057 |
| **Renal dysfunction** | | | | | | | |
| Yes | 8 | 14.8 | 7 | 15.2 | 15 | 15 | |
| No | 46 | 85.2 | 39 | 84.8 | 85 | 85 | 0.955 |
| **Valvular heart disease** | | | | | | | |
| Yes | 4 | 7.4 | 3 | 6.5 | 7 | 7 | |
| No | 50 | 92.6 | 43 | 93.5 | 93 | 93 | 0.863 |
| **Fazeka** | | | | | | | |
| Grade 0 | 15 | 27.8 | 5 | 10.9 | 20 | 20 | |
| Grade 1 | 20 | 37 | 24 | 52.2 | 44 | 44 | |
| Grade 2 | 15 | 27.8 | 17 | 37 | 32 | 32 | |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Grade 3 | 4 | 7.4 | 0 | 0 | 4 | 4 | 0.031 |
| **Mechanical Thrombectomy** | | | | | | | |
| Yes | 3 | 5.6 | 3 | 6.5 | 6 | 6 | |
| No | 51 | 94.4 | 43 | 93.5 | 94 | 94 | 0.839 |
| **Thrombolysis** | | | | | | | |
| Yes | 11 | 20.4 | 1 | 2.2 | 12 | 12 | |
| No | 43 | 79.6 | 45 | 97.8 | 88 | 88 | 0.086 |
| **Grade of HT** | | | | | | | |
| Grade 1 | 2 | 3.7 | 10 | 21.7 | 12 | 12 | |
| Grade 2 | 52 | 96.3 | 35 | 76.1 | 87 | 87 | |
| Grade 3 | 0 | 0 | 1 | 2.2 | 1 | 1 | 0.011 |
| | | | | | | | |
| **Antiplatelets** | | | | | | | |
| Yes | 50 | 92.6 | 45 | 97.8 | 95 | 95 | |
| No | 4 | 7.4 | 1 | 2.2 | 5 | 5 | 0.231 |
| **Anticoagulation** | | | | | | | |
| Yes | 5 | 9.3 | 6 | 13 | 11 | 11 | |
| No | 49 | 90.7 | 40 | 87 | 89 | 89 | 0.547 |
| **Statin use** | | | | | | | |
| Yes | 53 | 98.1 | 45 | 97.8 | 98 | 98 | |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| No | 1 | 1.9 | 1 | 2.2 | 2 | 2 | 0.909 |
| **Any recurrent strokes in 6 months** | | | | | | | |
| Yes | 0 | 0 | 1 | 2.2 | 1 | 1 | |
| No | 54 | 100 | 45 | 97.8 | 99 | 99 | 0.276 |
| **Posterior Circulation** | | | | | | | |
| Yes | 21 | 38.9 | 6 | 17.4 | 27 | 29 | |
| No | 35 | 61.1 | 38 | 82.6 | 73 | 71 | 0.058 |
| **Hemorrhagic transformation** | | | | | | | |
| Yes | 7 | 13 | 14 | 30.4 | 21 | 21 | |
| No | 47 | 87 | 32 | 69.6 | 79 | 79 | 0.033 |
| **LVAD** | | | | | | | |
| No | 32 | 59.3 | 23 | 50 | 55 | 55 | |
| Yes | 22 | 40.7 | 23 | 50 | 45 | 45 | 0.354 |
| **mRS @ admission** | | | | | | | |
| ≤2 | 28 | 43.1 | 8 | 22.9 | 36 | 36 | |
| >2 | 37 | 56.9 | 27 | 77.1 | 64 | 64 | 0.018 |
| **NIHSS** | | | | | | | |
| ≤5 | 31 | 57.4 | 10 | 21.7 | 41 | 41 | |
| >5 | 23 | 42.6 | 36 | 78.3 | 59 | 59 | <0.001 |
| **ASPECTS** | | | | | | | |
| <7 | 12 | 22.2 | 21 | 45.7 | 33 | 33 | |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| ≥7 | 42 | 77.8 | 25 | 54.3 | 67 | 67 | 0.013 |

History of smoking, prior TIA, small vessel ischemic changes (fazeka), haemorrhagic transformation, mRS at stroke onset, NIHSS, ASPECTS predicted presence of Hypersomnolence.

Table 14: Binary logistic regression model for ESS

| | B | S.E. | Wald | df | p | OR | 95% C.I.for OR | |
|---|---|---|---|---|---|---|---|---|
| | | | | | | | Lower | Upper |
| Smoking | 0.629 | 0.566 | 1.235 | 1 | 0.266 | 1.877 | 0.618 | 5.696 |
| Prior Stroke | 0.378 | 0.513 | 0.543 | 1 | 0.461 | 1.459 | 0.534 | 3.983 |
| Haemorrhagic transformation | 0.166 | 0.637 | 0.068 | 1 | 0.795 | 1.18 | 0.338 | 4.115 |
| MRS | 1.345 | 0.657 | 4.196 | 1 | 0.041 | 3.84 | 1.06 | 13.914 |
| NIHS | 0.515 | 0.695 | 0.55 | 1 | 0.458 | 1.674 | 0.429 | 6.537 |
| ASPECTS | -0.632 | 0.537 | 1.387 | 1 | 0.239 | 0.531 | 0.186 | 1.522 |
| Constant | -1.407 | 0.785 | 3.214 | 1 | 0.073 | 0.245 | | |

mRS at 6 months was independent risk factor that predicted Hypersomnolence ; p 0.041 (CI 1.06 to 13.914)

Table 15: Effect of hypersomnolence on care giver burden

| Zarit's CBS | ESS | | | | | | Total | |
|---|---|---|---|---|---|---|---|---|
| | 0-7 | | 8-9 | | 10-15 | | | |
| | n | % | n | % | n | % | n | % |
| <20 | 41 | 97.6 | 12 | 38.1 | 7 | 41.2 | 60 | 60 |
| 21-40 | 4 | 2.4 | 18 | 57.1 | 16 | 52.9 | 38 | 38 |
| 41-60 | 0 | 0 | 1 | 4.8 | 1 | 5.9 | 2 | 2 |
| Total | 45 | 100 | 31 | 100 | 24 | 100 | 100 | 100 |

| | $\chi^2$ | df | p |
|---|---|---|---|
| Chi-square test | 25.436 | 4 | <0.001 |

Caregivers of patients with hypersomnolence expressed higher care giver burden which was statistically significant ($p < 0.001$)

Table 16: Effect of hypersomnolence on self-reported depression

| PHQ 9 | EPSS | | | | | | Total | |
|---|---|---|---|---|---|---|---|---|
| | 0-7 | | 8-9 | | 10-15 | | | |
| | n | % | n | % | n | % | n | % |
| <10 | 32 | 76.2 | 30 | 42.9 | 3 | 23.5 | 65 | 65 |
| 10-14 | 4 | 21.4 | 8 | 42.9 | 18 | 70.6 | 30 | 30 |
| 15-19 | 1 | 2.4 | 2 | 9.5 | 1 | 5.9 | 4 | 4 |
| 20-27 | 0 | 0 | 1 | 4.8 | 0 | 0 | 1 | 1 |
| Total | 37 | 100 | 41 | 100 | 22 | 100 | 100 | 100 |

| | $\chi^2$ | df | p |
|---|---|---|---|
| Chi-square test | 19.163 | 6 | 0.004 |

Patients with hypersomnolence had higher self-reported depression which was statistically significant (p 0.004)

Table 17: Effect of Hypersomnolence on short term functional outcome

| | ESS | | | | Total | | p |
|---|---|---|---|---|---|---|---|
| | <8 | | ≥8 | | | | |
| | N | % | N | % | N | % | |
| mRS @ 6 months | | | | | | | |
| ≤2 | 47 | 87 | 19 | 41.3 | 66 | 66 | |
| >2 | 7 | 13 | 27 | 58.7 | 34 | 34 | <0.001 |

Patients with hypersomnolence had poor functional outcome (mRS >2) 6 months post stroke which was statistically significant ($p < 0.001$)

TABLE 18 : FACTORS PREDICTING INSOMNIA

| | Insomnia | | | | Total | | p |
|---|---|---|---|---|---|---|---|
| | <8 | | ≥8 | | | | |
| | N | % | N | % | N | % | |
| **Age** | | | | | | | |
| ≤60 | 31 | 47.7 | 14 | 40 | 45 | 45 | |
| >60 | 34 | 52.3 | 21 | 60 | 55 | 55 | 0.461 |
| **Sex** | | | | | | | |
| Male | 45 | 69.2 | 25 | 71.4 | 70 | 70 | |
| Female | 20 | 30.8 | 10 | 28.6 | 30 | 30 | 0.819 |
| **Education** | | | | | | | |
| Primary | 15 | 23.1 | 11 | 31.4 | 26 | 26 | |
| High school | 28 | 43.1 | 16 | 45.7 | 44 | 44 | |
| Plus Two | 3 | 4.6 | 2 | 5.7 | 5 | 5 | |
| Graduate and above | 19 | 29.2 | 6 | 17.1 | 25 | 25 | 0.566 |
| **Smoking** | | | | | | | |
| Yes | 14 | 21.5 | 12 | 34.3 | 26 | 26 | |
| No | 51 | 78.5 | 23 | 65.7 | 74 | 74 | 0.166 |
| **Alcoholism** | | | | | | | |
| Yes | 14 | 21.5 | 10 | 28.6 | 24 | 24 | |
| No | 51 | 78.5 | 25 | 71.4 | 76 | 76 | 0.432 |
| **HTN** | | | | | | | |
| Yes | 48 | 73.8 | 29 | 82.9 | 77 | 77 | |
| No | 17 | 26.2 | 6 | 17.1 | 23 | 23 | 0.307 |
| **DM** | | | | | | | |
| Yes | 42 | 64.6 | 22 | 62.9 | 64 | 64 | |
| No | 23 | 35.4 | 13 | 37.1 | 36 | 36 | 0.861 |
| **CAD** | | | | | | | |
| Yes | 19 | 29.2 | 14 | 40 | 33 | 33 | |
| No | 46 | 70.8 | 21 | 60 | 67 | 67 | 0.275 |
| **Prior Stroke** | | | | | | | |
| Yes | 17 | 26.2 | 13 | 37.1 | 30 | 30 | |
| No | 48 | 73.8 | 22 | 62.9 | 70 | 70 | 0.253 |
| **Prior TIA** | | | | | | | |
| Yes | 8 | 12.3 | 6 | 17.1 | 14 | 14 | |
| No | 57 | 87.7 | 29 | 82.9 | 86 | 86 | 0.506 |
| **Dyslipidemia** | | | | | | | |
| Yes | 46 | 70.8 | 26 | 74.3 | 72 | 72 | |
| No | 19 | 29.2 | 9 | 25.7 | 28 | 28 | 0.709 |

| | Insomnia | | | | Total | | p |
|---|---|---|---|---|---|---|---|
| | <8 | | ≥8 | | | | |
| | N | % | N | % | N | % | |
| **Etiology** | | | | | | | |
| Lacunar | 22 | 33.8 | 4 | 11.4 | 26 | 26 | |
| LVAD | 24 | 36.9 | 21 | 60 | 45 | 45 | |
| Cardioembolism | 7 | 10.8 | 5 | 14.3 | 12 | 12 | |
| Undetermined | 12 | 18.5 | 5 | 14.3 | 17 | 17 | 0.056 |
| **Territory** | | | | | | | |
| Right Hemispheric | 17 | 31.5 | 22 | 47.8 | 39 | 39 | |
| Left Hemispheric | 17 | 31.5 | 15 | 32.6 | 30 | 32 | |
| Posterior Circulation | 19 | 35.2 | 8 | 13 | 27 | 25 | |
| Bihemispheric | 1 | 0 | 3 | 4.3 | 4 | 4 | 0.052 |
| **POVD** | | | | | | | |
| Yes | 2 | 3.1 | 1 | 2.9 | 3 | 3 | |
| No | 63 | 96.9 | 34 | 97.1 | 97 | 97 | 0.951 |
| **Renal dysfunction** | | | | | | | |
| Yes | 9 | 13.8 | 6 | 17.1 | 15 | 15 | |
| No | 56 | 86.2 | 29 | 82.9 | 85 | 85 | 0.660 |
| **Valvular heart disease** | | | | | | | |
| Yes | 4 | 6.2 | 3 | 8.6 | 7 | 7 | |
| No | 61 | 93.8 | 32 | 91.4 | 93 | 93 | 0.651 |

| | Insomnia | | | | Total | | p |
|---|---|---|---|---|---|---|---|
| | <8 | | ≥8 | | | | |
| | N | % | N | % | N | % | |
| **Fazeka** | | | | | | | |
| Grade 0 | 16 | 24.6 | 4 | 11.4 | 20 | 20 | |
| Grade 1 | 26 | 40 | 18 | 51.4 | 44 | 44 | |
| Grade 2 | 19 | 29.2 | 13 | 37.1 | 32 | 32 | |
| Grade 3 | 4 | 6.2 | 0 | 0 | 4 | 4 | 0.154 |
| **Mechanical Thrombectomy** | | | | | | | |
| Yes | 4 | 6.2 | 2 | 5.7 | 6 | 6 | |
| No | 61 | 93.8 | 33 | 94.3 | 94 | 94 | 0.930 |
| **Thrombolysis** | | | | | | | |
| Yes | 9 | 13.8 | 3 | 8.6 | 12 | 12 | |
| No | 56 | 86.2 | 32 | 91.4 | 88 | 88 | 0.439 |
| **Grade of HT** | | | | | | | |
| Grade 1 | 3 | 4.6 | 9 | 25.7 | 12 | 12 | |
| Grade 2 | 61 | 93.8 | 26 | 74.3 | 87 | 87 | |
| Grade 3 | 1 | 1.5 | 0 | 0 | 1 | 1 | 0.007 |
| **Antiplatelets** | | | | | | | |
| Yes | 63 | 96.9 | 32 | 91.4 | 95 | 95 | |
| No | 2 | 3.1 | 3 | 8.6 | 5 | 5 | 0.229 |
| **Anticoagulation** | | | | | | | |
| Yes | 5 | 7.7 | 6 | 17.1 | 11 | 11 | |
| No | 60 | 92.3 | 29 | 82.9 | 89 | 89 | 0.150 |
| **Statin use** | | | | | | | |
| Yes | 64 | 98.5 | 34 | 97.1 | 98 | 98 | |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| No | 1 | 1.5 | 1 | 2.9 | 2 | 2 | 0.653 |

| | Insomnia | | | | Total | | p |
|---|---|---|---|---|---|---|---|
| | <8 | | ≥8 | | | | |
| | N | % | N | % | N | % | |
| **Any recurrent strokes in 6 months** | | | | | | | |
| Yes | 0 | 0 | 1 | 2.9 | 1 | 1 | |
| No | 65 | 100 | 34 | 97.1 | 99 | 99 | 0.171 |
| **ACA** | | | | | | | |
| Yes | 6 | 61.5 | 31 | 88.6 | 71 | 71 | |
| No | 59 | 38.5 | 4 | 11.4 | 29 | 29 | 0.004 |
| **Posterior Circulation** | | | | | | | |
| Yes | 24 | 36.9 | 5 | 14.3 | 29 | 29 | |
| No | 41 | 63.1 | 30 | 85.7 | 71 | 71 | 0.017 |
| **MCA** | | | | | | | |
| Yes | 40 | 61.5 | 31 | 88.6 | 71 | 71 | |
| No | 25 | 38.5 | 4 | 11.4 | 29 | 29 | 0.004 |
| **LVAD** | | | | | | | |
| No | 41 | 63.1 | 14 | 40 | 55 | 55 | |
| Yes | 24 | 36.9 | 21 | 60 | 45 | 45 | 0.027 |
| **mRS @ admission** | | | | | | | |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| ≤2 | 28 | 43.1 | 8 | 22.9 | 36 | 36 | |
| >2 | 37 | 56.9 | 27 | 77.1 | 64 | 64 | 0.045 |
| **NIHSS** | | | | | | | |
| ≤5 | 33 | 50.8 | 8 | 22.9 | 41 | 41 | |
| >5 | 32 | 49.2 | 27 | 77.1 | 59 | 59 | 0.007 |
| **ASPECTS** | | | | | | | |
| <7 | 21 | 32.3 | 12 | 34.3 | 33 | 33 | |
| ≥7 | 44 | 67.7 | 23 | 65.7 | 67 | 67 | 0.841 |

Hemorraghic transformation, Mrs at stroke onset, NIHSS , large artery atherosclerosis, ACA & MCA territory strokes predicted presence of Insomnia.

Table 19 : Binary logistic regression model for Insomnia

| | B | S.E. | Wald | df | p | OR | 95% C.I.for OR | |
|---|---|---|---|---|---|---|---|---|
| | | | | | | | Lower | Upper |
| Posterior Circulation | -0.293 | 0.941 | 0.097 | 1 | 0.756 | 0.746 | 0.118 | 4.723 |
| MCA | 0.922 | 1.059 | 0.758 | 1 | 0.384 | 2.516 | 0.315 | 20.057 |
| LVAD | 0.622 | 0.479 | 1.687 | 1 | 0.194 | 1.863 | 0.729 | 4.762 |
| MRS | 0.469 | 0.761 | 0.38 | 1 | 0.538 | 1.598 | 0.359 | 7.108 |
| NIHS | -0.014 | 0.84 | 0 | 1 | 0.987 | 0.986 | 0.19 | 5.12 |
| Constant | -2.392 | 1.649 | 2.105 | 1 | 0.147 | 0.091 | | |

Binary logistic regression didn’t show any independent predictors of insomnia

Table 20 : Effect of Insomnia on care giver burden

| Zarit's CBS | Insomnia | | | | | | Total | |
|---|---|---|---|---|---|---|---|---|
| | 0-7 | | 8-14 | | 15-21 | | | |
| | n | % | n | % | n | % | n | % |
| <20 | 43 | 86 | 24 | 70 | 3 | 30 | 70 | 70 |
| 21-40 | 6 | 12 | 7 | 25 | 15 | 70 | 28 | 28 |
| 41-60 | 1 | 2 | 1 | 5 | 0 | 0 | 2 | 2 |
| Total | 50 | 100 | 32 | 100 | 18 | 100 | 100 | 100 |

| | $\chi^2$ | df | p |
|---|---|---|---|
| Chi-square test | 16.907 | 4 | 0.002 |

Caregivers of patients with Insomnia expressed higher care giver burden which was statistically significant (p 0.002)

Table 21 : Effect of Insomnia on self reported depression

| PHQ 9 | Insomnia | | | | | | Total | |
|---|---|---|---|---|---|---|---|---|
| | 0-7 | | 8-14 | | 15-21 | | | |
| | n | % | n | % | n | % | n | % |
| <10 | 37 | 74 | 10 | 25 | 8 | 30 | 55 | 56.3 |
| 10-14 | 12 | 24 | 20 | 65 | 8 | 50 | 40 | 37.5 |
| 15-19 | 1 | 2 | 1 | 5 | 2 | 20 | 4 | 5 |
| 20-27 | 0 | 0 | 1 | 5 | 0 | 0 | 1 | 1.3 |
| Total | 50 | 100 | 32 | 100 | 18 | 100 | 100 | 100 |

| | $\chi^2$ | df | p |
|---|---|---|---|
| Chi-square test | 22.778 | 6 | 0.001 |

Patients with insomnia had higher self reported depression which was statistically significant (p 0.001)

Table 22 : Effect of Insomnia on short term functional outcome

| | Insomnia | | | | Total | | p |
|---|---|---|---|---|---|---|---|
| | <8 | | ≥8 | | | | |
| | N | % | N | % | N | % | |
| mRS @ 6 months | | | | | | | |
| ≤2 | 52 | 80 | 14 | 40 | 66 | 66 | |
| >2 | 13 | 20 | 21 | 60 | 34 | 34 | <0.001 |

Patients with insomnia had bad functional outcome (mrs >2) 6 months post stroke which was statistically significant ($p < 0.001$)

Table 23 : FACTORS PREDICTING POOR SLEEP QUALITY (PSQI)

| | PSQI | | | | Total | | p |
|---|---|---|---|---|---|---|---|
| | <5 | | ≥5 | | | | |
| | N | % | N | % | N | % | |
| **Age** | | | | | | | |
| ≤60 | 29 | 48.3 | 16 | 40 | 45 | 45 | |
| >60 | 31 | 51.7 | 24 | 60 | 55 | 55 | 0.412 |
| **Sex** | | | | | | | |
| Male | 40 | 66.7 | 30 | 75 | 70 | 70 | |
| Female | 20 | 33.3 | 10 | 25 | 30 | 30 | 0.373 |
| **Education** | | | | | | | |
| Primary | 15 | 25 | 11 | 27.5 | 26 | 26 | |
| High school | 30 | 50 | 14 | 35 | 44 | 44 | |
| Plus Two | 2 | 3.3 | 3 | 7.5 | 5 | 5 | |
| Graduate and above | 13 | 21.7 | 12 | 30 | 25 | 25 | 0.426 |
| **Smoking** | | | | | | | |
| Yes | 10 | 16.7 | 16 | 40 | 26 | 26 | |
| No | 50 | 83.3 | 24 | 60 | 74 | 74 | 0.009 |
| **Alcoholism** | | | | | | | |
| Yes | 15 | 25 | 9 | 22.5 | 24 | 24 | |
| No | 45 | 75 | 31 | 77.5 | 76 | 76 | 0.774 |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| **HTN** | | | | | | | |
| Yes | 46 | 76.7 | 31 | 77.5 | 77 | 77 | |
| No | 14 | 23.3 | 9 | 22.5 | 23 | 23 | 0.923 |
| **DM** | | | | | | | |
| Yes | 34 | 56.7 | 30 | 75 | 64 | 64 | |
| No | 26 | 43.3 | 10 | 25 | 36 | 36 | 0.061 |
| **CAD** | | | | | | | |
| Yes | 14 | 23.3 | 19 | 47.5 | 33 | 33 | |
| No | 46 | 76.7 | 21 | 52.5 | 67 | 67 | 0.012 |
| **Prior Stroke** | | | | | | | |
| Yes | 13 | 21.7 | 17 | 42.5 | 30 | 30 | |
| No | 47 | 78.3 | 23 | 57.5 | 70 | 70 | 0.026 |
| **Prior TIA** | | | | | | | |
| Yes | 6 | 10 | 8 | 20 | 14 | 14 | |
| No | 54 | 90 | 32 | 80 | 86 | 86 | 0.158 |
| **Dyslipidemia** | | | | | | | |
| Yes | 43 | 71.7 | 29 | 72.5 | 72 | 72 | |
| No | 17 | 28.3 | 11 | 27.5 | 28 | 28 | 0.928 |

| | PSQI | | | | Total | | p |
|---|---|---|---|---|---|---|---|
| | <5 | | ≥5 | | | | |
| | N | % | N | % | N | % | |
| **Etiology** | | | | | | | |
| Lacunar | 22 | 36.7 | 4 | 10 | 26 | 26 | |
| LVAD | 16 | 26.7 | 29 | 72.5 | 45 | 45 | |
| Cardioembolism | 8 | 13.3 | 4 | 10 | 12 | 12 | |
| Undetermined | 14 | 23.3 | 3 | 7.5 | 17 | 17 | <0.001 |
| **Circulation** | | | | | | | |
| Right Hemispheric | 20 | 33.3 | 19 | 47.5 | 39 | 39 | |
| Left Hemispheric | 19 | 31.7 | 13 | 32.5 | 32 | 32 | |
| Posterior Circulation | 19 | 31.7 | 6 | 15 | 25 | 25 | |
| Bihemispheric | 2 | 3.3 | 0 | 0 | 2 | 2 | 0.437 |
| **POVD** | | | | | | | |
| Yes | 1 | 1.7 | 2 | 5 | 3 | 3 | |
| No | 59 | 98.3 | 38 | 95 | 97 | 97 | 0.338 |
| **Renal dysfunction** | | | | | | | |
| Yes | 8 | 13.3 | 7 | 17.5 | 15 | 15 | |
| No | 52 | 86.7 | 33 | 82.5 | 85 | 85 | 0.568 |
| **Valvular heart disease** | | | | | | | |
| Yes | 3 | 5 | 4 | 10 | 7 | 7 | |
| No | 57 | 95 | 36 | 90 | 93 | 93 | 0.337 |
| **ACA** | | | | | | | |
| Yes | 6 | 10 | 1 | 2.5 | 7 | 7 | |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| No | 54 | 90 | 39 | 97.5 | 93 | 93 | 0.150 |
| | PSQI | | | | Total | | p |
| | <5 | | ≥5 | | | | |
| | N | % | N | % | N | % | |
| **Fazeka** | | | | | | | |
| Grade 0 | 16 | 26.7 | 4 | 10 | 20 | 20 | |
| Grade 1 | 24 | 40 | 20 | 50 | 44 | 44 | |
| Grade 2 | 18 | 30 | 14 | 35 | 32 | 32 | |
| Grade 3 | 2 | 3.3 | 2 | 5 | 4 | 4 | 0.237 |
| **Mechanical Thrombectomy** | | | | | | | |
| Yes | 3 | 5 | 3 | 7.5 | 6 | 6 | |
| No | 57 | 95 | 37 | 92.5 | 94 | 94 | 0.606 |
| **Thrombolysis** | | | | | | | |
| Yes | 9 | 15 | 3 | 7.5 | 12 | 12 | |
| No | 51 | 85 | 37 | 92.5 | 88 | 88 | 0.258 |
| **Grade of HT** | | | | | | | |
| Grade 1 | 1 | 1.7 | 11 | 27.5 | 12 | 12 | |
| Grade 2 | 59 | 98.3 | 28 | 70 | 87 | 87 | |
| Grade 3 | 0 | 0 | 1 | 2.5 | 1 | 1 | <0.001 |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| **Antiplatelets** | | | | | | | |
| Yes | 56 | 93.3 | 39 | 97.5 | 95 | 95 | |
| No | 4 | 6.7 | 1 | 2.5 | 5 | 5 | 0.349 |
| **Anticoagulation** | | | | | | | |
| Yes | 5 | 8.3 | 6 | 15 | 11 | 11 | |
| No | 55 | 91.7 | 34 | 85 | 89 | 89 | 0.297 |
| **Statin use** | | | | | | | |
| Yes | 59 | 98.3 | 39 | 97.5 | 98 | 98 | |
| No | 1 | 1.7 | 1 | 2.5 | 2 | 2 | 0.771 |

| | PSQI | | | | Total | | p |
|---|---|---|---|---|---|---|---|
| | <5 | | ≥5 | | | | |
| | N | % | N | % | N | % | |
| **LVAD** | | | | | | | |
| No | 44 | 73.3 | 11 | 27.5 | 55 | 55 | |
| Yes | 16 | 26.7 | 29 | 72.5 | 45 | 45 | <0.001 |
| **Any recurrent strokes in 6 months** | | | | | | | |
| Yes | 0 | 0 | 1 | 2.5 | 1 | 1 | |
| No | 60 | 100 | 39 | 97.5 | 99 | 99 | 0.218 |
| **Posterior Circulation** | | | | | | | |
| Yes | 21 | 35 | 8 | 20 | 29 | 29 | |
| No | 39 | 65 | 32 | 80 | 71 | 71 | 0.105 |
| **MCA** | | | | | | | |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Yes | 37 | 61.7 | 34 | 85 | 71 | 71 | |
| No | 23 | 38.3 | 6 | 15 | 29 | 29 | 0.012 |
| **ACA** | | | | | | | |
| Yes | 6 | 10 | 1 | 2.5 | 7 | 7 | |
| No | 54 | 90 | 39 | 97.5 | 93 | 93 | 0.150 |
| **Hemorrhagic transformation** | | | | | | | |
| Yes | 5 | 8.3 | 16 | 40 | 21 | 21 | |
| No | 55 | 91.7 | 24 | 60 | 79 | 79 | <0.001 |
| **mRS @ admission** | | | | | | | |
| ≤2 | 25 | 41.7 | 11 | 27.5 | 36 | 36 | |
| >2 | 35 | 58.3 | 29 | 72.5 | 64 | 64 | 0.148 |
| **NIHSS** | | | | | | | |
| ≤5 | 31 | 51.7 | 10 | 25 | 41 | 41 | |
| >5 | 29 | 48.3 | 30 | 75 | 59 | 59 | 0.008 |
| **ASPECTS** | | | | | | | |
| <7 | 15 | 25 | 18 | 45 | 33 | 33 | |
| ≥7 | 45 | 75 | 22 | 55 | 67 | 67 | 0.037 |

Smoking, CAD, prior stroke, MCA territory strokes, haemorrhagic transformation, NIHSS, large artery atherosclerosis predicted poor sleep quality assessed by PSQI

Table 24 : Binary logistic regression model for PSQI

| | B | S.E. | Wald | df | p | OR | 95% C.I.for OR | |
|---|---|---|---|---|---|---|---|---|
| | | | | | | | Lower | Upper |
| Smoking | 0.65 | 0.663 | 0.959 | 1 | 0.327 | 1.915 | 0.522 | 7.028 |
| CAD | 0.651 | 0.609 | 1.141 | 1 | 0.285 | 1.917 | 0.581 | 6.33 |
| Prior Stroke | 1.176 | 0.602 | 3.817 | 1 | 0.051 | 3.242 | 0.996 | 10.552 |
| MCA | 0.659 | 0.709 | 0.864 | 1 | 0.353 | 1.933 | 0.482 | 7.754 |
| Hemorrhagic transformation | 1.02 | 0.776 | 1.728 | 1 | 0.189 | 2.774 | 0.606 | 12.706 |
| LVAD | 1.889 | 0.651 | 8.414 | 1 | 0.004 | 6.614 | 1.845 | 23.703 |
| NIHSS | -0.061 | 0.713 | 0.007 | 1 | 0.932 | 0.941 | 0.233 | 3.802 |
| ASPECTS | 0.03 | 0.706 | 0.002 | 1 | 0.966 | 1.031 | 0.259 | 4.11 |
| Constant | -3.171 | 1.843 | 2.959 | 1 | 0.085 | 0.042 | | |

Large artery atherosclerosis was independent risk factor that predicted poor sleep quality ; p 0.004 (CI 1.845 to 23.703)

Table 25 : Effect of poor sleep quality on care giver burden

| Zarit's CBS | PSQI | | | | | | Total | |
|---|---|---|---|---|---|---|---|---|
| | <5 | | 5-14 | | 15-21 | | | |
| | n | % | n | % | n | % | n | % |
| <20 | 41 | 89.1 | 29 | 61.3 | 0 | 0 | 70 | 75 |
| 21-40 | 4 | 8.7 | 22 | 38.7 | 2 | 66.7 | 28 | 22.5 |
| 41-60 | 1 | 2.2 | 0 | 0 | 1 | 33.3 | 2 | 2.5 |
| Total | 46 | 100 | 51 | 100 | 3 | 100 | 100 | 100 |

| | $\chi^2$ | df | p |
|---|---|---|---|
| Chi-square test | 26.571 | 4 | <0.001 |

Caregivers of patients with poor sleep quality expressed higher care giver burden which was statistically significant ($p < 0.001$)

Table 26 : Effect of poor sleep quality on self reported depression

| PHQ 9 | PSQI | | | | | | Total | |
|---|---|---|---|---|---|---|---|---|
| | <5 | | 5-14 | | 15-21 | | | |
| | n | % | n | % | n | % | n | % |
| <10 | 32 | 69.6 | 23 | 41.9 | 0 | 0 | 55 | 56.3 |
| 10-14 | 14 | 30.4 | 24 | 45.2 | 2 | 66.7 | 40 | 37.5 |
| 15-19 | 0 | 0 | 3 | 9.7 | 1 | 33.3 | 4 | 5 |
| 20-27 | 0 | 0 | 1 | 3.2 | 0 | 0 | 1 | 1.3 |
| Total | 46 | 100 | 51 | 100 | 3 | 100 | 100 | 100 |

| | $\chi^2$ | df | p |
|---|---|---|---|
| Chi-square test | 16.098 | 6 | 0.013 |

Patients with poor sleep quality had higher self reported depression which was statistically significant (p 0.013)

Table 27 : Effect of poor sleep quality on short term functional outcome

| | PSQI | | | | Total | | p |
|---|---|---|---|---|---|---|---|
| | <5 | | ≥5 | | | | |
| | N | % | N | % | N | % | |
| mRS @ 6 months | | | | | | | |
| ≤2 | 48 | 80 | 18 | 45 | 66 | 66 | |
| >2 | 12 | 20 | 22 | 55 | 34 | 34 | <0.001 |

Patients with poor sleep quality had poor functional outcome (mRS >2) 6 months post stroke which was statistically significant ($p < 0.001$)

Table 28 : FACTORS PREDICTING DEPRESSION

| | PHQ-9 | | | | Total | | p |
|---|---|---|---|---|---|---|---|
| | <10 | | ≥10 | | | | |
| | N | % | N | % | N | % | |
| **Age** | | | | | | | |
| ≤60 | 27 | 49.1 | 18 | 40 | 45 | 45 | |
| >60 | 28 | 50.9 | 27 | 60 | 55 | 55 | 0.363 |
| **Sex** | | | | | | | |
| Male | 41 | 74.5 | 29 | 64.4 | 70 | 70 | |
| Female | 14 | 25.5 | 16 | 35.6 | 30 | 30 | 0.273 |
| **Education** | | | | | | | |
| Primary | 12 | 21.8 | 14 | 31.1 | 26 | 26 | |
| High school | 26 | 47.3 | 18 | 40 | 44 | 44 | |
| Plus Two | 2 | 3.6 | 3 | 6.7 | 5 | 5 | |
| Graduate and above | 15 | 27.3 | 10 | 22.2 | 25 | 25 | 0.609 |
| **Smoking** | | | | | | | |
| Yes | 12 | 21.8 | 14 | 31.1 | 26 | 26 | |
| No | 43 | 78.2 | 31 | 68.9 | 74 | 74 | 0.292 |
| **Alcoholism** | | | | | | | |
| Yes | 11 | 20 | 13 | 28.9 | 24 | 24 | |
| No | 44 | 80 | 32 | 71.1 | 76 | 76 | 0.300 |
| **HTN** | | | | | | | |
| Yes | 39 | 70.9 | 38 | 84.4 | 77 | 77 | |
| No | 16 | 29.1 | 7 | 15.6 | 23 | 23 | 0.110 |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| **DM** | | | | | | | |
| Yes | 35 | 63.6 | 29 | 64.4 | 64 | 64 | |
| No | 20 | 36.4 | 16 | 35.6 | 36 | 36 | 0.933 |
| **CAD** | | | | | | | |
| Yes | 17 | 30.9 | 16 | 35.6 | 33 | 33 | |
| No | 38 | 69.1 | 29 | 64.4 | 67 | 67 | 0.623 |
| **Prior Stroke** | | | | | | | |
| Yes | 13 | 23.6 | 17 | 37.8 | 30 | 30 | |
| No | 42 | 76.4 | 28 | 62.2 | 70 | 70 | 0.125 |
| **Prior TIA** | | | | | | | |
| Yes | 4 | 7.3 | 10 | 22.2 | 14 | 14 | |
| No | 51 | 92.7 | 35 | 77.8 | 86 | 86 | 0.032 |
| **Dyslipidemia** | | | | | | | |
| Yes | 37 | 67.3 | 35 | 77.8 | 72 | 72 | |
| No | 18 | 32.7 | 10 | 22.2 | 28 | 28 | 0.244 |

| | PHQ-9 | | | | Total | | p |
|---|---|---|---|---|---|---|---|
| | <10 | | ≥10 | | | | |
| | N | % | N | % | N | % | |
| **Etiology** | | | | | | | |
| Lacunar | 18 | 32.7 | 8 | 17.8 | 26 | 26 | |
| LVAD | 24 | 43.6 | 21 | 46.7 | 45 | 45 | |
| Cardioembolism | 4 | 7.3 | 8 | 17.8 | 12 | 12 | |
| Undetermined | 9 | 16.4 | 8 | 17.8 | 17 | 17 | 0.214 |
| **Circulation** | | | | | | | |
| Right Hemispheric | 20 | 36.4 | 19 | 42.2 | 39 | 39 | |
| Left Hemispheric | 16 | 29.1 | 16 | 35.6 | 32 | 32 | |
| Posterior Circulation | 18 | 32.7 | 7 | 15.6 | 25 | 25 | |
| Bihemispheric | 0 | 0 | 2 | 4.4 | 2 | 2 | 0.214 |
| **POVD** | | | | | | | |
| Yes | 0 | 0 | 3 | 6.7 | 3 | 3 | |
| No | 55 | 100 | 42 | 93.3 | 97 | 97 | 0.052 |
| **Renal dysfunction** | | | | | | | |
| Yes | 9 | 16.4 | 6 | 13.3 | 15 | 15 | |
| No | 46 | 83.6 | 39 | 86.7 | 85 | 85 | 0.673 |
| **Valvular heart disease** | | | | | | | |
| Yes | 4 | 7.3 | 3 | 6.7 | 7 | 7 | |
| No | 51 | 92.7 | 42 | 93.3 | 93 | 93 | 0.906 |
| **ACA** | | | | | | | |
| Yes | 4 | 7.3 | 3 | 6.7 | 7 | 7 | |
| No | 51 | 92.7 | 42 | 93.3 | 93 | 93 | 0.906 |

| | PHQ-9 | | | | Total | | p |
|---|---|---|---|---|---|---|---|
| | <10 | | ≥10 | | | | |
| | N | % | N | % | N | % | |
| **Fazeka** | | | | | | | |
| Grade 0 | 14 | 25.5 | 6 | 13.3 | 20 | 20 | |
| Grade 1 | 20 | 36.4 | 24 | 53.3 | 44 | 44 | |
| Grade 2 | 17 | 30.9 | 15 | 33.3 | 32 | 32 | |
| Grade 3 | 4 | 7.3 | 0 | 0 | 4 | 4 | 0.080 |
| **Mechanical Thrombectomy** | | | | | | | |
| Yes | 2 | 3.6 | 4 | 8.9 | 6 | 6 | |
| No | 53 | 96.4 | 41 | 91.1 | 94 | 94 | 0.271 |
| **Thrombolysis** | | | | | | | |
| Yes | 10 | 18.2 | 2 | 4.4 | 12 | 12 | |
| No | 45 | 81.8 | 43 | 95.6 | 88 | 88 | 0.035 |
| **Grade of HT** | | | | | | | |
| Grade 1 | 3 | 5.5 | 9 | 20 | 12 | 12 | |
| Grade 2 | 52 | 94.5 | 35 | 77.8 | 87 | 87 | |
| Grade 3 | 0 | 0 | 1 | 2.2 | 1 | 1 | 0.041 |
| **Antiplatelets** | | | | | | | |
| Yes | 54 | 98.2 | 41 | 91.1 | 95 | 95 | |
| No | 1 | 1.8 | 4 | 8.9 | 5 | 5 | 0.107 |
| **Anticoagulation** | | | | | | | |
| Yes | 5 | 9.1 | 6 | 13.3 | 11 | 11 | |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| No | 50 | 90.9 | 39 | 86.7 | 89 | 89 | 0.500 |
| **Statin use** | | | | | | | |
| Yes | 53 | 96.4 | 45 | 100 | 98 | 98 | |
| No | 2 | 3.6 | 0 | 0 | 2 | 2 | 0.196 |

| | PHQ-9 | | | | Total | | p |
|---|---|---|---|---|---|---|---|
| | <10 | | ≥10 | | | | |
| | N | % | N | % | N | % | |
| **Any recurrent strokes in 6 months** | | | | | | | |
| Yes | 1 | 1.8 | 0 | 0 | 1 | 1 | |
| No | 54 | 98.2 | 45 | 100 | 99 | 99 | 0.363 |
| **Posterior Circulation** | | | | | | | |
| Yes | 21 | 38.2 | 8 | 17.8 | 29 | 29 | |
| No | 34 | 61.8 | 37 | 82.2 | 71 | 71 | 0.025 |
| **MCA** | | | | | | | |
| Yes | 35 | 63.6 | 36 | 80 | 71 | 71 | |
| No | 20 | 36.4 | 9 | 20 | 29 | 29 | 0.073 |
| **LVAD** | | | | | | | |
| No | 31 | 56.4 | 24 | 53.3 | 55 | 55 | |
| Yes | 24 | 43.6 | 21 | 46.7 | 45 | 45 | 0.762 |
| **mRS @ admission** | | | | | | | |
| ≤2 | 21 | 38.2 | 15 | 33.3 | 36 | 36 | |
| >2 | 34 | 61.8 | 30 | 66.7 | 64 | 64 | 0.615 |
| **NIHSS** | | | | | | | |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| ≤5 | 26 | 47.3 | 15 | 33.3 | 41 | 41 | |
| >5 | 29 | 52.7 | 30 | 66.7 | 59 | 59 | 0.159 |
| **ASPECTS** | | | | | | | |
| <7 | 16 | 29.1 | 17 | 37.8 | 33 | 33 | |
| ≥7 | 39 | 70.9 | 28 | 62.2 | 67 | 67 | 0.358 |

Prior TIA , posterior circulation stroke, haemorrhagic transformation predicted self reported depression assessed by PHQ 9

Table 29 : Binary logistic regression model for PHQ-9

| | B | S.E. | Wald | df | p | OR | 95% C.I.for OR | |
|---|---|---|---|---|---|---|---|---|
| | | | | | | | Lower | Upper |
| Prior TIA | 0.846 | 0.685 | 1.528 | 1 | 0.216 | 2.331 | 0.609 | 8.919 |
| Thrombolysis | -1.776 | 0.839 | 4.478 | 1 | 0.034 | 0.169 | 0.033 | 0.877 |
| Posterior Circulation | -1.217 | 0.506 | 5.786 | 1 | 0.016 | 0.296 | 0.11 | 0.798 |
| Constant | 0.717 | 0.427 | 2.821 | 1 | 0.093 | 2.048 | | |

Binary logistic regression didn't show any independent predictors of self reported depression

Table 30 : Effect of depression on care giver burden

| PHQ 9 | Zarit's CBS | | | | | | Total | |
|---|---|---|---|---|---|---|---|---|
| | <20 | | 21-40 | | 41-60 | | | |
| | n | % | n | % | n | % | n | % |
| <10 | 51 | 68.3 | 4 | 22.2 | 0 | 0 | 55 | 56.3 |
| 10-14 | 28 | 30 | 10 | 55.6 | 2 | 100 | 40 | 37.5 |
| 15-19 | 1 | 1.7 | 3 | 16.7 | 0 | 0 | 4 | 5 |
| 20-27 | 0 | 0 | 1 | 5.6 | 0 | 0 | 1 | 1.3 |
| Total | 81 | 100 | 18 | 100 | 2 | 100 | 100 | 100 |

| | $\chi^2$ | df | p |
|---|---|---|---|
| Chi-square test | 20.714 | 6 | 0.002 |

Caregivers of patients with poor sleep quality expressed higher care giver burden which was statistically significant (p 0.002)

Table 31 : Effect of depression on short term functional outcome

| | PHQ-9 | | | | Total | | p |
|---|---|---|---|---|---|---|---|
| | <10 | | ≥10 | | | | |
| | N | % | N | % | N | % | |
| mRS @ 6 months | | | | | | | |
| ≤2 | 47 | 85.5 | 19 | 42.2 | 66 | 66 | |
| >2 | 8 | 14.5 | 26 | 57.8 | 34 | 34 | <0.001 |

Patients with depression had poor functional outcome (mRS >2) 6 months post stroke which was statistically significant (p < 0.001)

TABLE 32 : PREDCICTORS OF CARE GIVER BURDEN

| | Zarit's CBS | | | | Total | | p |
|---|---|---|---|---|---|---|---|
| | <21 | | ≥21 | | | | |
| | N | % | N | % | N | % | |
| **Age** | | | | | | | |
| ≤60 | 37 | 47.4 | 8 | 36.4 | 45 | 45 | |
| >60 | 41 | 52.6 | 14 | 63.6 | 55 | 55 | 0.357 |
| **Sex** | | | | | | | |
| Male | 54 | 69.2 | 16 | 72.7 | 70 | 70 | |
| Female | 24 | 30.8 | 6 | 27.3 | 30 | 30 | 0.752 |
| **Education** | | | | | | | |
| Primary | 18 | 23.1 | 8 | 36.4 | 26 | 26 | |
| High school | 35 | 44.9 | 9 | 40.9 | 44 | 44 | |
| Plus Two | 3 | 3.8 | 2 | 9.1 | 5 | 5 | |
| Graduate and above | 22 | 28.2 | 3 | 13.6 | 25 | 25 | 0.305 |
| **Smoking** | | | | | | | |
| Yes | 16 | 20.5 | 10 | 45.5 | 26 | 26 | |
| No | 62 | 79.5 | 12 | 54.5 | 74 | 74 | 0.018 |
| **Alcoholism** | | | | | | | |
| Yes | 17 | 21.8 | 7 | 31.8 | 24 | 24 | |
| No | 61 | 78.2 | 15 | 68.2 | 76 | 76 | 0.331 |
| **HTN** | | | | | | | |
| Yes | 60 | 76.9 | 17 | 77.3 | 77 | 77 | |
| No | 18 | 23.1 | 5 | 22.7 | 23 | 23 | 0.973 |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| **DM** | | | | | | | |
| Yes | 49 | 62.8 | 15 | 68.2 | 64 | 64 | |
| No | 29 | 37.2 | 7 | 31.8 | 36 | 36 | 0.644 |
| **CAD** | | | | | | | |
| Yes | 22 | 28.2 | 11 | 50 | 33 | 33 | |
| No | 56 | 71.8 | 11 | 50 | 67 | 67 | 0.055 |
| **Prior Stroke** | | | | | | | |
| Yes | 19 | 24.4 | 11 | 50 | 30 | 30 | |
| No | 59 | 75.6 | 11 | 50 | 70 | 70 | 0.020 |
| **Prior TIA** | | | | | | | |
| Yes | 7 | 9 | 7 | 31.8 | 14 | 14 | |
| No | 71 | 91 | 15 | 68.2 | 86 | 86 | 0.006 |
| **Dyslipidemia** | | | | | | | |
| Yes | 54 | 69.2 | 18 | 81.8 | 72 | 72 | |
| No | 24 | 30.8 | 4 | 18.2 | 28 | 28 | 0.246 |

| | Zarit's CBS | | | | Total | | p |
|---|---|---|---|---|---|---|---|
| | <21 | | ≥21 | | | | |
| | N | % | N | % | N | % | |
| **Etiology** | | | | | | | |
| Lacunar | 24 | 30.8 | 2 | 9.1 | 26 | 26 | |
| LVAD | 33 | 42.3 | 12 | 54.5 | 45 | 45 | |
| Cardioembolism | 8 | 10.3 | 4 | 18.2 | 12 | 12 | |
| Undetermined | 13 | 16.7 | 4 | 18.2 | 17 | 17 | 0.204 |
| **Circulation** | | | | | | | |
| Right Hemispheric | 29 | 37.2 | 10 | 45.5 | 39 | 39 | |
| Left Hemispheric | 23 | 29.5 | 9 | 40.9 | 32 | 32 | |
| Posterior Circulation | 23 | 29.5 | 2 | 9.1 | 25 | 25 | |
| BiHemispheric | 2 | 2.6 | 0 | 0 | 2 | 2 | 0.143 |
| **POVD** | | | | | | | |
| Yes | 1 | 1.3 | 2 | 9.1 | 3 | 3 | |
| No | 77 | 98.7 | 20 | 90.9 | 97 | 97 | 0.058 |
| **Renal dysfunction** | | | | | | | |
| Yes | 11 | 14.1 | 4 | 18.2 | 15 | 15 | |
| No | 67 | 85.9 | 18 | 81.8 | 85 | 85 | 0.636 |
| **Valvular heart disease** | | | | | | | |
| Yes | 5 | 6.4 | 2 | 9.1 | 7 | 7 | |
| No | 73 | 93.6 | 20 | 90.9 | 93 | 93 | 0.663 |
| **ACA** | | | | | | | |
| Yes | 6 | 7.7 | 1 | 4.5 | 7 | 7 | |
| No | 72 | 92.3 | 21 | 95.5 | 93 | 93 | 0.609 |

| | Zarit's CBS | | | | Total | | p |
|---|---|---|---|---|---|---|---|
| | <21 | | ≥21 | | | | |
| | N | % | N | % | N | % | |
| **Fazeka** | | | | | | | |
| Grade 0 | 16 | 20.5 | 4 | 18.2 | 20 | 20 | |
| Grade 1 | 32 | 41 | 12 | 54.5 | 44 | 44 | |
| Grade 2 | 26 | 33.3 | 6 | 27.3 | 32 | 32 | |
| Grade 3 | 4 | 5.1 | 0 | 0 | 4 | 4 | 0.555 |
| **Mechanical Thrombectomy** | | | | | | | |
| Yes | 3 | 3.8 | 3 | 13.6 | 6 | 6 | |
| No | 75 | 96.2 | 19 | 86.4 | 94 | 94 | 0.088 |
| **Thrombolysis** | | | | | | | |
| Yes | 12 | 15.4 | 0 | 0 | 12 | 12 | |
| No | 66 | 84.6 | 22 | 100 | 88 | 88 | 0.050 |
| **Grade of HT** | | | | | | | |
| Grade 1 | 7 | 9 | 5 | 22.7 | 12 | 12 | |
| Grade 2 | 71 | 91 | 16 | 72.7 | 87 | 87 | |
| Grade 3 | 0 | 0 | 1 | 4.5 | 1 | 1 | 0.032 |
| **Antiplatelets** | | | | | | | |
| Yes | 74 | 94.9 | 21 | 95.5 | 95 | 95 | |
| No | 4 | 5.1 | 1 | 4.5 | 5 | 5 | 0.912 |
| **Anticoagulation** | | | | | | | |
| Yes | 6 | 7.7 | 5 | 22.7 | 11 | 11 | |
| No | 72 | 92.3 | 17 | 77.3 | 89 | 89 | 0.057 |
| **Statin use** | | | | | | | |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Yes | 76 | 97.4 | 22 | 100 | 98 | 98 | |
| No | 2 | 2.6 | 0 | 0 | 2 | 2 | 0.448 |

| | Zarit's CBS | | | | Total | | p |
|---|---|---|---|---|---|---|---|
| | <21 | | ≥21 | | | | |
| | N | % | N | % | N | % | |
| **Any recurrent strokes in 6 months** | | | | | | | |
| Yes | 0 | 0 | 1 | 4.5 | 1 | 1 | |
| No | 78 | 100 | 21 | 95.5 | 99 | 99 | 0.058 |
| **Posterior Circulation** | | | | | | | |
| Yes | 26 | 33.3 | 3 | 13.6 | 29 | 29 | |
| No | 52 | 66.7 | 19 | 86.4 | 71 | 71 | 0.072 |
| **MCA** | | | | | | | |
| Yes | 50 | 64.1 | 21 | 95.5 | 71 | 71 | |
| No | 28 | 35.9 | 1 | 4.5 | 29 | 29 | 0.004 |
| **Hemorrhagic transformation** | | | | | | | |
| Yes | 12 | 15.4 | 9 | 40.9 | 21 | 21 | |
| No | 66 | 84.6 | 13 | 59.1 | 79 | 79 | 0.009 |
| **LVAD** | | | | | | | |
| No | 45 | 57.7 | 10 | 45.5 | 55 | 55 | |
| Yes | 33 | 42.3 | 12 | 54.5 | 45 | 45 | 0.308 |
| **mRS @ admission** | | | | | | | |
| ≤2 | 34 | 43.6 | 2 | 9.1 | 36 | 36 | |
| >2 | 44 | 56.4 | 20 | 90.9 | 64 | 64 | 0.003 |
| **NIHSS** | | | | | | | |
| ≤5 | 39 | 50 | 2 | 9.1 | 41 | 41 | |
| >5 | 39 | 50 | 20 | 90.9 | 59 | 59 | 0.001 |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| **ASPECTS** | | | | | | | |
| <7 | 17 | 21.8 | 16 | 72.7 | 33 | 33 | |
| ≥7 | 61 | 78.2 | 6 | 27.3 | 67 | 67 | <0.001 |

Smoking, prior stroke & TIA, MCA territory strokes, haemorrhagic transformation, NIHSS, mRS at admission predicted care giver burden.

Table 33 : Binary logistic regression model for Zarit's CBS

| | B | S.E. | Wald | df | p | OR | 95% C.I.for OR | |
|---|---|---|---|---|---|---|---|---|
| | | | | | | | Lower | Upper |
| Prior Stroke | 1.529 | 0.716 | 4.556 | 1 | 0.033 | 4.612 | 1.133 | 18.773 |
| Prior TIA | 0.538 | 0.985 | 0.299 | 1 | 0.585 | 1.712 | 0.249 | 11.794 |
| MCA | 3.29 | 1.554 | 4.481 | 1 | 0.034 | 26.842 | 1.276 | 564.582 |
| Hemorrhagic transformation | -0.417 | 0.909 | 0.21 | 1 | 0.646 | 0.659 | 0.111 | 3.915 |
| MRS | 1.727 | 1.39 | 1.544 | 1 | 0.214 | 5.623 | 0.369 | 85.667 |
| NIHSS | -0.871 | 1.519 | 0.328 | 1 | 0.567 | 0.419 | 0.021 | 8.223 |
| ASPECTS | -2.502 | 0.775 | 10.422 | 1 | 0.001 | 0.082 | 0.018 | 0.374 |
| Constant | -3.616 | 1.615 | 5.015 | 1 | 0.025 | 0.027 | | |

Prior stroke, MCA territory stroke & ASPECTS were independent risk factor that predicted care giver burden with p values 0.033 , 0.034 & 0.001 respectively.

TABLE 34 : POLYSOMNOGRAPHY

| AHI | FREQUENCY | PERCENTAGE |
|---|---|---|
| < 5 | 9 | 45 % |
| ≥ 5 | 11 | 55 % |

20 patients underwent overnight polysomnography of which 11 (55 %) had sleep disordered breathing.

TABLE 35 : SDB & HYPERSOMNOLENCE

| | EPSS | | | | Total | | p |
|---|---|---|---|---|---|---|---|
| PSG | <8 | | ≥8 | | | | |
| | N | % | N | % | N | % | |
| Normal | 6 | 46.2 | 3 | 42.9 | 9 | 45.0 | |
| Abnormal | 7 | 53.8 | 4 | 57.1 | 11 | 55.0 | 0.888 |
| Total | 13 | 100.0 | 7 | 100.0 | 20 | 100.0 | |

There was no difference in hypersomnolence in patients with and without sleep disordered breathing

TABLE 36 : SDB & INSOMNIA

| | Insomnia | | | | Total | | p |
|---|---|---|---|---|---|---|---|
| PSG | <8 | | ≥8 | | | | |
| | N | % | N | % | N | % | |
| Normal | 6 | 40.0 | 3 | 60.0 | 9 | 45.0 | |
| Abnormal | 9 | 60.0 | 2 | 40.0 | 11 | 55.0 | 0.436 |
| Total | 15 | 100.0 | 5 | 100.0 | 20 | 100.0 | |

There was no difference in insomnia in patients with and without sleep disordered breathing

TABLE 37 : SDB & POOR SLEEP QUALITY

| | PSQI | | | | Total | | p |
|---|---|---|---|---|---|---|---|
| PSG | <5 | | ≥5 | | | | |
| | N | % | N | % | N | % | |
| Normal | 6 | 42.9 | 3 | 50.0 | 9 | 45.0 | |
| Abnormal | 8 | 57.1 | 3 | 50.0 | 11 | 55.0 | 0.769 |
| Total | 14 | 100.0 | 6 | 100.0 | 20 | 100.0 | |

There was no difference in sleep quality in patients with and without sleep disordered breathing

TABLE 38 : SDB & SELF REPORTED DEPRESSION

| | PHQ-9 | | | | Total | | p |
|---|---|---|---|---|---|---|---|
| PSG | <5 | | ≥5 | | | | |
| | N | % | N | % | N | % | |
| Normal | 5 | 41.7 | 4 | 50.0 | 9 | 45.0 | |
| Abnormal | 7 | 58.3 | 4 | 50.0 | 11 | 55.0 | 0.714 |
| Total | 12 | 100.0 | 8 | 100.0 | 20 | 100.0 | |

There was no difference in self reported depression in patients with and without sleep disordered breathing

TABLE 39 : SDB & CARE GIVER BURDEN

| | Zarit's CBS | | | | Total | | p |
|---|---|---|---|---|---|---|---|
| PSG | ≤20 | | >20 | | | | |
| | N | % | N | % | N | % | |
| Normal | 8 | 44.4 | 1 | 50.0 | 9 | 45.0 | |
| Abnormal | 10 | 55.6 | 1 | 50.0 | 11 | 55.0 | 0.881 |
| Total | 18 | 100.0 | 2 | 100.0 | 20 | 100.0 | |

There was no difference in care giver burden in patients with and without sleep disordered breathing

# DISCUSSION

# DISCUSSION

**PREVALENCE:**

Of the 100 patients recruited, 46 % patients reported hypersomnolence, 45 % had reported depression, 40 % had poor sleep quality , 35 % had insomnia & 22 % of care givers expressed burden. Previous studies have shown hypersomnolence in 20 % [73] to 48 % [87], insomnia in 14 [73] to 50 % [2], depression in 35 % [74], poor sleep quality in 32 to 71% [73, 87, 88] & care giver burden varying from 56 to 72 % [75, 94,102,120, 121]. Care giver burden in our study (22 %) was lower than previous studies [75, 94,102,120, 121]. Previous studies on Indian population by Sapna et al [102] & Das et al [121] have reported higher incidence of care giver burden . However care giver burden is a complex parameter influenced by various factors like attitudes, financial status, physical health status of care giver & various social factors in addition patient related factors. This could explain the lower care giver burden in current study.

**CLINICAL FEATURES :**

Our study had a total of 100 patients and mean age was 62.2 ± 11.2 years . We found total 8 studies [2,73,68,76,77,83,87,88] that studied different types of sleep dysfunction after stroke. Metanalysis by Fulk et all [76] that included 33 studies was the largest **.** Mean age of the patients in the previous studies were varies from 42-72 years .

In the previous studies, the prevalence of risk factors- hypertension ranged from 61-74% [76,77, 88] and Diabetes was 10-38% [76,77, 87, 88] . Our cohort had a high prevalence of vascular risk factors and coronary disease also. This finding has been noticed in most of the studies on risk factor profile and etiology of AIS from India. As per the study from Calcutta by Das et al, hypertension rate was higher in the community including unrecognised hypertension [78]. In Trivandrum stroke registry study by Sapna et al, it was found that around 90% had risk factors [79]. Among our subjects, hypertension (77 %) and diabetes (64 %) were the predominant risk factors.

26 % had history of smoking in our study. This is similar to the study conducted by Sapna et al which stated that 26.8% had history of smoking [79].

In our study, predominantly strokes were anterior circulation - hemispheric strokes (right more than left) while 4 % were bihemispheric strokes. This is comparison to the study conducted by Alia H. Mansour et al [73], where 47% were left hemisphere, 30% right hemisphere, and 23 % vertebrobasilar stroke.

In previous studies majority patients were of mild to moderate strokes [76,73] . In study by Alia H Mansur et all 61 % were moderate strokes [73] & 38 % were minor strokes. Study by Fulk et al also had similar distribution with majority being moderate strokes. This was comparable with the 50 % moderate & 33 % mild strokes in our study. Mean NIHSS of our study was 8.3 ± 6.24 at admission and 4.45 ± 4.28 at the time of discharge. Mean NIHSS score at admission from Zhang et al [77] was 1.117 (1.069–1.167) which constituted minor strokes only & 3.26 ± 3.64 in study by Keun Tae Kim et al [88]. NIHSS scores are not available from rest of previous studies for comparison.

In our study large artery atherosclerosis was the most common etiology (45 %) followed by lacunar strokes (26 %), undetermined (17 %) & cardio embolism (12 %). This was comparable to results from meta analysis of 50 years of Indian data from S K Das et al (2016) [80] where most common etiology of stroke was large artery atherosclerosis (41 %) followed by lacunar strokes (18 %) . This was comparable to study by Keun Tae Kim et al [88] studying sleep dysfunction after stroke where majority strokes were due to large artery atherosclerosis (63 %).

**PREDICTORS OF SLEEP DYSFUNCTION**

**HYPERSOMNOLENCE**

Most of the studies assessing hypersomnia after stroke have used ESS [73, 2, 66, 76, 77,87, 88]. However the cut off value to consider significant hypersomnolence varied across them. Alia H et al [73] used cut of more than 16 while Zhang et al [77], Hermann D M et al [2] &

Keun Tae Kim et al [88] used cut of score ≥ 10 . These studies have reported lower prevalence of 20-28 % . Study by Sreedharan et al [102] used cut off value of > 11 in sleep clinic attendees & found that severity of OSA correlated well with ESS scores in Indian populations.

Others [66,2,76] have used sleep duration of ≥ 9-10 hrs per day to define hypersomnia. Jinil Kim et al [87] used ≥ 8 as cut off and found prevalence of hypersomnia to be 48.8 %. In this study we have used similar cut of ≥ 8 & 46 % of our study population had hypersomnia.We selected a lower cut-off in our study as higher cut-offs over 11 had more specificity in diagnosing primary and secondary disorders of hypersomnolence,with low sensitivity.

Univariate analysis showed history of smoking, prior TIA, small vessel ischemic changes on imaging scored by fazeka's grade, haemorrhagic transformation, mRS at stroke onset, NIHSS,ASPECTS predicted presence of Hypersomnolence. Except for history of smoking & prior TIA other factors were directly / indirectly were related to stroke severity.

In bivariate analysis mRS (> 2) at 6 months was an independent risk factor that predicted Hypersomnolence (p 0.041) (CI 1.06 to 13.914). Previous studies have reported hypersomnolence [2, 73] with posterior circulation strokes which was not seen in our study. In our study posterior circulation strokes represented a minor set of patients only (27 %) which could explain the disparity. Patients with hypersomnolence had higher self reported depression & higher care giver burden as shown by previous studies [2, 73].

**INSOMNIA**

Most of previous studies on insomnia after stroke [2,73, 81] have used sleep duration of ≤ 5 to 6 hrs per day to define insomnia & reported insomnia in upto 50% patients. Studies using ISI scale in stroke patients by Jinil Kim et al [87] used ≥ 8 as cut off and found prevalence of insomnia to be 53.3 % & Keun Tae Kim1 et al [88] used cut off of ≥ 15.5 & found prevalence of 12%. In this study we have used similar cut of ≥ 8 & 46 % of our study population had insomnia. Most of the other studies using Insomnia Severity

Index (ISI) are conducted in non-stroke patients using cut of values of ≥ 8 to define insomnia [82].

Univariate analysis haemorrhagic transformation, mRS at stroke onset, NIHSS, large artery atherosclerosis, ACA & MCA territory strokes predicted presence of insomnia. Except for large artery atherosclerosis, ACA & MCA territory strokes other factors were directly / indirectly were related to stroke severity. However bivariate analysis didn't show any independent factor that predicted post stroke insomnia.

This is contrary to previous studies that reported insomnia with severe strokes & right hemispheric strokes [2,73,76]. Patients with insomnia had higher self reported depression & higher care giver burden as shown by previous studies[2,73,74].

**POOR SLEEP QUALITY**

Previous studies using PSQI to assess sleep quality after stroke [73,2,87,88] used score of ≥ 5 with estimated prevalence of 32 to 71 % . We have used similar cut of score of ≥ 5 & prevalence (40 %) was comparable to previous studies.
Univariate analysis showed history of smoking, CAD, prior stroke, MCA territory strokes, haemorrhagic transformation, NIHSS, large artery atherosclerosis predicted poor sleep quality assessed by PSQI
. Except for history of smoking, prior stroke, CAD other factors were directly / indirectly were related to stroke severity.
In bivariate analysis large artery atherosclerosis was independent risk factor that predicted poor sleep quality ; p 0.004 (CI 1.845 to 23.703) . This was a novel finding which could be due to higher prevalence other comorbidities in patients with large artery atherosclerosis like obesity, CAD, renal dysfunction which are known to affect sleep [22]

## SELF REPORTED DEPRESSION

Previous studies assessing post stroke depression [74,2] have used PHQ 9 with score cut off ≥ 10 to define depression with estimated prevalence of 30-35 %. Few studies have used HADS (Hospital anxiety & depression scale for depression) & Beck Depression Inventory reporting depression in 38-36 % patients [88]. In our study we used same cut off score of ≥ 10. Prevalence of self reported depression (45 %) was comparable to previous studies.

Univariate analysis showed Prior TIA , posterior circulation stroke, haemorrhagic transformation predicted self reported depression assessed by PHQ 9 . However bivariate analysis didn't show any independent factor that predicted post stroke depression. This is in concordance with the majority of earlier

Studies on predictors of post stroke depression(PSD) have yielded mixed results.While some authors found that post stroke depression was not showing association with variables like age, hemisphere, etiology of stroke and post-stroke duration [89–91],which was similar to our finding as well.But some have reported that site of the lesion [26]and extremes of age[92,93] as being among the important risk factors for PSD .

## CARE GIVER BURDEN

Care giver burden in our study (22 %) was lower than previous studies, including one from our Institute itself,done a decade earlier [75, 94,120,121]. Care giver burden is a complex parameter influenced by various factors like attitudes, financial status, physical health status of care giver & various social factors in addition patient related factors and reporting bias also.

Univariate analysis showed history of smoking, prior strokes, MCA territory strokes, haemorrhagic transformation, NIHSS, mRS at admission predicted higher care giver burden. Except for history of smoking, prior stroke, MCA territory strokes other factors were directly / indirectly were related to stroke severity. In bivariate analysis prior stroke, MCA territory strokes & ASPECTS were independent risk factor that predicted care giver burden with p values 0.033, 0.034 & 0.001 respectively. This is comparable

to previous studies that showed recurrent strokes, dense hemiplegia & severe strokes had higher care giver burden [2, 73, 75, 76] mostly related to the physical disability of the stroke survivor adding to the burden.

## SLEEP DISORDERED BREATHING

Of the 20 % patients that underwent overnight PSG of which 11 patients (55%) had sleep disordered breathing. However it did not show an association with other sleep disorders captured using questionnaires (hypersomnia, insomnia, poor sleep quality, depression). This is contrary to existing data that reports higher levels of self-reported sleep dysfunction {hypersomnia [84], insomnia [83], poor sleep quality [85], depression [86] } in patients with sleep disordered breathing.

In the current study only a small proportion,ie 20 % of study population underwent PSG which may have resulted in underestimation of effect of SDB on sleep dysfunction in our cohort.Also we could not sub-categorize subjects with OSA based on their severity and analyse each groups with self reported sleep complaints,which would have influenced the results.

## EFFECT OF SLEEP DYSFUNCTION ON SHORT TERM FUNCTIONAL OUTCOME

In the current study, hypersomnolence, insomnia, poor sleep quality & self reported depression were all associated with poor functional outcome (mRS > 2) at 6 months post stroke ($p < 0.001$).

Recent study by Keun Tae (2017) reported hypersomnolence lead to poorer outcome & affects rehabilitation in stroke patients [88] . Also, excessive daytime sleepiness can be regarded as a symptom of SDB or a compensatory mechanism of the stroke itself. These findings were in-line with the previous studies suggesting the importance of SDB for stroke prognoses [2,73, 66,76,77,87]. Systematic review & metanalysis on sleep duration & cerebrovascular outcomes in 2011 [58] showed longer sleep duration is an independent predictor of incident stroke, after adjustments for age, sex, vascular risk, and attributing

comorbidities. Long sleep durations (more than nine hours) have also been associated with subcortical white matter hyperintensities in adults, suggesting that excessive sleep may be caused by cerebral small vessel disease and reflect subclinical atherosclerosis [59]. This makes a strong case for early recognition of SDB post stroke & to treat it.

Inversely, insomnia is also common in the months following stroke and is found in 30-50% of sufferers [52]. In the current study 46 % patients had insomnia & it lead to poor short term functional outcome. Post-stroke insomnia is associated with poor life satisfaction, depression, and stroke severity [53,60,61].

Few researchers have looked into sleep architecture in patients with insomnia after stroke. It has been noted that a stroke affecting the paramedian thalamic nuclei leads to near complete absence of sleep spindles [62]. Neuroplasticity and stroke recovery have been demonstrated to be better when drugs promoting NREM & REM sleep are used [63]. Thus insomnia by affecting the neuroplasticity post stroke leads to poorer functional outcome.

Patients who reported overall poor sleep quality had worse functional scores at 6 months implying the restorative role of sleep in stroke recovery. . Ischaemic stroke causes severe acute impairment of glymphatic perfusion 24 hours after ischaemia[50]. Glymphatic disruption may promote excitotoxicity of surrounding brain penumbra and prevent acute clearance of excitatory neurotransmitters. As previously described, a surge of excess glutamate is secreted after stroke (a process known as excitotoxicity) and elicits a myriad of signalling cascades that work synergistically to induce neuronal death [51]. This excitoxicity, disrupted glymphatic circulation when coupled with sleep deprivation / poor sleep quality is proposed to be involved in impairing the post stroke functional recovery.

Post-stroke depression (PSD) is one of the common emotional disorders afflicting stroke survivors. In our study population depression was reported in 45 % of patients. Previous studies have reported prevalence rates that have ranged from 18% to 61%, depending upon patient selection and criteria used [122,123,74]. Diagnosis of PSD is challenging; therefore, it often remains unrecognized and/or undertreated PSD is associated with cognitive impairment, increased mortality and risk of falls, increased

disability, and worse rehabilitation outcome [124] . Depression is considered as the strongest predictor of poor quality of life among stroke survivors [125] . Improvement of depressive symptoms has been associated with better functional recovery [126]. Thus screening for depression should be become part of standard stroke care for better outcome.

Severe stokes are associated with higher care giver burden as reported by previous studies [2, 73, 75, 76]. In addition this study found burden was higher among care givers of patients with sleep dysfunction. As discussed above different sleep dysfunctions (insomnia, hypersomnolence, SDB, poor sleep quality) are all associated with poor functional outcome which in turn increases care giver burden. Interestingly enough, sleep dysfunction (sleep deprivation) is noted to be significantly increased in care givers of stroke patients making them at risk for various neurological disorders including stroke [111]. Current study reported significantly lower care giver burden as compared to other studies from west. This can be explained by better family support systems still prevailing in our part of the country as compared to the West. Also Care giver burden is a complex parameter influenced by various factors like attitudes, financial status, physical health status of care giver & various social factors in addition patient related factors.

**LIMITATIONS**

Though all patients were taken for the study during their 3 month follow-up post stroke and the interview and completion of scales were performed by the same person, the patient population were not uniform in their disability levels, with mRS at 3 months ranging from 0-4, with mean of 1.83 ± 1.54 . We did not include subjects with maximum disability (mRS 5) in our study. Though our target sample size was 130, we could only recruit 100 subjects after screening for exclusion criteria, which reduced the power of the study. The low sample numbers precluded analysis of correlations between sleep dysfunction and subpopulations of our stroke survivors.

We could perform overnight ambulatory level 3 polysomnography in 20% of our patients, which could only serve to screen for sleep apnea .Because of lack of funds, we could not perform level 1 PSG in our subjects which would have given us information on other polysomnographic parameters like sleep efficiency, arousals, duration of slow wave sleep and REM sleep and periodic leg movements. We used standard questionnaires for capturing information on sleep quality and other abnormalities, but there is always a recall bias in such studies.

However, we believe that our observations are important in understanding this less often reported problem in post stroke survivors. Ours is the first systematically done study on predictors of post stroke sleep dysfunction at short term from South India. We used standard scales for insomnia, excessive day time sleepiness and sleep quality along with scales for depression and caregiver burden at 3 months after stroke and assessed its relation with 6 month outcome. We could perform ambulatory PSG in a proportion of subjects, which identified sleep apnea in around half of stroke survivors, but did not show a correlation with parameters of sleep dysfunction.

# CONCLUSION

# CONCLUSION

- We found a high prevalence of sleep dysfunction at 3 months after stroke (hypersomnolence in 46 %, poor sleep quality in 40 %, insomnia in 35 %).
- Our cohort was a male predominant one, with mean age of 62.2 ± 11.2 years and a high prevalence of vascular risk factors.
- Majority of our cohort consisted of mild (33 %) to moderate (50%) strokes
- 45 % of patients reported of depression & 22% care givers expressed burden
- There was significant corelation between severity of stroke & different parameters of sleep dysfunction studied
- Hypersomnolence showed a positive correlation with poor functional status at 6 months .
- Stroke due to large artery atherosclerosis independently predicted poor sleep quality
- Recurrent strokes, anterior circulation strokes and severe strokes independently predicted higher care giver burden.
- Sleep dysfunction lead to poor short term functional outcome (mRS > 2 at 6 months) and higher caregiver burden scores.

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# ANNEXURES

# *CURRICULUM VITAE*

| | | |
|---|---|---|
| Last Name KULKARNI | First Name AVINASH | Middle Name T |
| Date of Birth (dd/mm/yy) 10/07/1991 | | Sex Male |
| Affiliation : Department of Neurology , SCTIMST | | |
| Professional Mailing Address | | Study Site Address |
| DEPT OF NEUROLOGY,<br>SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES AND TECHNOLOGY<br>THIRUVANANTHAPURAM<br>KERALA 695011 | | DEPT OF NEUROLOGY,<br>SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES AND TECHNOLOGY<br>THIRUVANANTHAPURAM<br>KERALA 695011 |
| Telephone (Office) : | | Mobile Number: 8792466031 |
| Telephone (Residence) : | | Email- kavinash1677@gmail.com |

| Academic Qualifications (Most recent qualification first) | | |
|---|---|---|
| Degree/Certificate | Year | Institution, Country |
| MD General Medicine | 2019 | RNT Medical College , Udaipur , Rajasthan , India , 313001 |
| MBBS | 2015 | Raichur Institute Of Medical Sciences , Raichur , Karnataka , India , 584102 |

Details of professional registration : (MCI/State Registration/Bar Council/DCI/etc including Registration Number and Year of Registration

TRAVANCORE COCHIN MEDICAL COUNCIL
Registration Number : 80172
Year of Registration : 2021

| Current and previous positions (most recent position first) | | |
|---|---|---|
| Month and Year | Title | Institution/Company, Country |
| January 2021 till present | Senior Resident , Dept of Neurology | SCTIMST, INDIA |
| May 2016 till May2019 | Junior Resident , General Medicine | RNT Medical College , Udaipur , Rajasthan. |

Brief summary of relevant research experience:

1 ) Poster presentation at ICTRIMS Delhi 2023 : Patterns of clinical presentation and disease modifying therapy use in multiple sclerosis over two decades

2 ) Poster presentation at Rajasthan endocrine & diabetes update 2017, Udaipur : Serum creatine kinase (CPK) levels in subclinical & overt hypothyroidism.

3 ) Research article publication- IJCR 2017 : Prevalence of low vitamin d levels in newly diagnosed HIV patients

4 ) Rare case publication in JAPI , April 2020 : Case of sine scleroderma

5 ) MD Thesis : serum sodium levels as prognostic marker in patients of cerebral malaria in a tertiary care hospital in southern Rajasthan

Current project/s at hand:
NONE

| Signature:  | Date:<br>24/8/2023<br>Place:<br>TRIVANDRUM |
|---|---|

# INSTITUTIONAL ETHICS COMMITTEE CLEARANCE

श्री चित्रा तिरुनाल आयुर्विज्ञान और प्रौद्योगिकी संस्थान, त्रिवेन्द्रम
तिरुवनन्तपुरम - ६९५०११, केरल, इंडिया
SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES AND TECHNOLOGY, TRIVANDRUM
Thiruvananthapuram - 695 011, Kerala, India
*(An Institute of National Importance under Govt. of India)*
Grams : Chitramet, Phone : +91-471-2443152, Fax : +91-471-2550728 / 2446433, E-mail : sct@sctimst.ac.in, Website : www.sctimst.ac.in

DUPLICATE

## Institutional Ethics Committee

**(IEC Regn No. ECR/189/Inst/KL/2013/RR-21)**

SCT/IEC/1805/JANUARY/2022

21.02.2022

**Dr. Avinash T Kulkarni**
Senior Resident
Department of Neurology
SCTIMST, Thiruvananthapuram

Dear Dr. Avinash Kulkarni,

The Institutional Ethics Committee held on 29th January, 2022, reviewed and discussed your application to conduct the study titled "**PREDICTORS OF SLEEP DYSFUNCTION AFTER ISCHEMIC STROKE AND ITS IMPACT ON STROKE RECOVERY**" (IEC/1805).

**The following members of the Ethics Sub-committee were present at the meeting held on 29th January, 2022.**

| SL. No. | Member Name | Highest Degree | Gender | Scientific /Non Scientific | Affiliation with Institution(s) |
|---|---|---|---|---|---|
| 1. | Dr. Kala Kesavan P | MBBS,MD | Female | Basic Medical Scientist | No |
| 2. | Adv. N Anand | BAL, L.LB | Male | Legal Expert | No |
| 3. | Dr. Harikrishna Varma P. R | Ph.D (Materials Sciences) | Male | Medical Technology | Yes |
| 4. | Dr. Manikandan.S | MBBS,MD,PDCC | Male | Clinician | Yes |
| 5. | Dr. Ashalatha R | MBBS, MD,DM | Female | Clinician | Yes |
| 6. | Dr. Biju Soman | MBBS,MD, DPH, MSc, DLSHTM | Male | Basic Medical Scientist | Yes |
| 7. | Dr. Srinivas G | PhD | Male | Basic Medical Scientist (Member Secretary) | Yes |

Page 1 of 2

**SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES AND TECHNOLOGY, THIRUVANANTHAPURAM, KERALA - 695011**

**COMPREHENSIVE STROKE CARE PROGRAM**

**PATIENT INFORMATION SHEET**

**Title of the study:**
PREDICTORS OF SLEEP DYSFUNCTION AFTER ISCHEMIC STROKE AND ITS IMPACT ON STROKE RECOVERY

**Principal Investigator:**
Dr. Avinash T Kulkarni, Senior Resident, Department of Neurology, SCTIMST

**Co-Principal Investigator:**
Dr. Sapna Erat Sreedharan, Additional Professor, Department of Neurology, SCTIMST
Dr. Sylaja P N, Professor & HOD, Department of Neurology, SCTIMST

**Sir/ Madam,**
We invite you to take part in our study titled *"Predictors of sleep dysfunction after ischemic stroke and its impact on stroke recovery "* a prospective study. Before you agree to participate in this research study, it is important that you read and understand this information sheet which will provide you with all the information needed for participation in this study so that you can make a well informed and considered decision about participation. In addition, should you have any questions, the investigator and his team members will be happy to answer them and explain to you more about this research study, the procedure involved and the related issues. You may ask them any questions you may have regarding the study, or ask them to explain any word or information that you don't clearly understand.

**Study Overview**
You are invited to take part in this study as you have acute ischemic stroke diagnosed 3 months ago & now coming for follow up. As part of this study we will be collecting information from you regarding the sleep dysfunction that you have & try to analyse the same to find the predictors for it. We will also be following you up after 3 months to understand the effect of this sleep dysfunction on your recovery from stroke. All patients diagnosed with acute ischemic stroke coming for follow-up at 3 months post stroke with functional status by mRS below 5 at Comprehensive stroke care centre, Department of Neurology, SCTIMST fulfilling the inclusion and exclusion criteria will be selected.

**Purpose of this study**

The purpose of this study is to find the predictors of sleep dysfunction after ischemic stroke & its impact on stroke recovery.

**Study Procedures**

If you are willing to participate, you will be interviewed and examined by neurologist and the clinical findings will be noted. This shall be planned when you will be coming for 3 month follow up post stroke . It will be done free of cost as a part of the study. The investigators will share the details with you.

**Risks and Discomfort**

This study involves only a structured interview by neurologist which will be completed in 30 to 40 minutes time. There is no specific risks associated with the study.

**Benefits**

Taking part in this research study may not benefit you immediately in any manner. However, we do hope that this study will shed light on the high prevalence of sleep dysfunction & its effect on short term recovery post stroke thus helping stroke care providers to keep a low threshold to detect sleep dysfunction & treat it promptly at the earliest in future .

**Confidentiality**

Your privacy is very important to us and the results of the tests performed on you will be treated as highly confidential, and nobody other than the investigators listed above will be knowing the test results. Your name or any other identifiable details will not be published in any research paper or scientific presentation arising out of the study.

**Rights**

Your participation in the trial is voluntary. You do not have to take part in this study if you are unwilling and you will not be losing any of your rights as a patient if you choose not to participate. You will also be at the liberty to withdraw from the study at any stage (even after signing this consent form) of the study in case you want to withdraw.

**Contact Information**

- When you read this information, your treating doctor will be available to discuss and answer any questions you may have. If you have any queries please contact:

  **Dr Avinash T Kulkarni**

Senior Resident, Department of Neurology,
Sree Chitra Tirunal Institute for Medical Sciences and Technology
Tel: +91 8792466031, Email: avinashkulkarni@sctimst.ac.in

- If you have any questions, concerns or complaints about the research please contact:

**Dr. Srinivas G**
Member Secretary, Institutional Ethics Committee,
Sree Chitra Tirunal Institute for Medical Sciences and Technology
Tel: 0471- 2524689, Email: iec.mem.sec@sctimst.ac.in

ശ്രീ ചിത്ര തിരുനാൾ ഇൻസ്റ്റിറ്റ്യൂട്ട് ഫോർ മെഡിക്കൽ സയൻസസ് ആന്റ് ടെക്നോളജി,
തിരുവനന്തപുരം, കേരളം – 695011
മസ്തിഷ്കാഘാത പരിചരണത്തിനായുള്ള സമഗ്ര പരിപാടി

## രോഗിക്കുള്ള കാര്യവിവരണപത്രം

പഠനശീർഷകം:
ഇന്ത്യയിലെ മസ്തിഷ്കാഘാത രോഗമുള്ള ഉറക്കരോഹിതുത്തിന്റെ സൂചകങ്ങളും മസ്തിഷ്കാഘാതം മൂലപ്പെടുന്നതിൽ അതിന്റെ പ്രഭാവവും.

പ്രധാന ഗവേഷകൻ

ഡോ. അഖിനാഷ് ടി കുൽക്കർണി, സീനിയർ മെഡിക്കൽ, സ്ട്രോക്ക് ഡിപ്പാർട്ട്മെന്റ്, SCTIMST

സഹ പ്രധാന ഗവേഷക

ഡോ. സംഗീത ഏറാട്ട് ശ്രീധരൻ, പ്രൊഫസർ, സ്ട്രോക്ക് ഡിപ്പാർട്ട്മെന്റ്, SCTIMST

ശ്രീ/ശ്രീമതി
ഇന്ത്യയിലെ മസ്തിഷ്കാഘാത രോഗമുള്ള ഉറക്കരോഹിതുത്തിന്റെ സൂചകങ്ങളും മസ്തിഷ്കാഘാതം മൂലപ്പെടുന്നതിൽ അതിന്റെ പ്രഭാവവും എന്ന പഠനത്തിൽ പങ്കെടുക്കാൻ താങ്കളെ ഞങ്ങൾ ക്ഷണിക്കുന്നു.
ഈ പഠനത്തിൽ പങ്കെടുക്കുവാൻ താങ്കൾ സമ്മതിക്കുന്നതിനുമുമ്പ് ഈ പഠന സംബന്ധമായി അറിയേണ്ടുന്ന പ്രസക്തമായ എല്ലാ വിവരങ്ങളും നൽകുന്ന ഈ കാര്യവിവരണപത്രം വായിക്കുകയും മനസ്സിലാക്കുകയും ചെയ്യേണ്ടത് കാര്യബോധത്തോടെ സമ്മതം നൽകാൻ തീരുമാനമെടുക്കുന്നതിൽ പ്രധാനമാണ്. അതിനെപ്പറ്റിയോ താങ്കൾക്കെന്തെങ്കിലും ചോദ്യങ്ങളുണ്ടെങ്കിൽ അവ വിശദീകരിച്ചുതരുവാൻ ഗവേഷകനും സംഘാംഗങ്ങളും സന്തോഷത്തോടെ തയ്യാറാകുകയും ഗവേഷണ പഠനം, ഉൾപ്പെട്ട നടപടികൾ സംബന്ധപ്പെട്ട വിഷയങ്ങൾ എന്നിവയെപ്പറ്റി കൂടുതൽ വിശദീകരണം നൽകുകയും ചെയ്യും. പഠനത്തെപ്പറ്റി താങ്കൾക്കെന്തെങ്കിലും ചോദ്യങ്ങളോ വ്യക്തമായി മനസ്സിലാകാത്ത ഏതെങ്കിലും വാക്കുകളോ വിവരങ്ങളോ ഉണ്ടെങ്കിൽ പഠനം നടത്തുന്നവരോട് ചോദിക്കുക.

### പഠന അവലോകനം

താങ്കൾക്ക് ഇന്ത്യയിലെ മസ്തിഷ്കാഘാതം 3 മാസം മുമ്പ് നിർണ്ണയിക്കപ്പെടുകയും ഇപ്പോൾ തുടർചികിത്സയ്ക്കായി വരുന്നതിനാലുമാണ് ഈ പഠനത്തിൽ പങ്കെടുക്കാൻ താങ്കളോട് ആവശ്യപ്പെടുന്നത്. പഠനത്തിന്റെ ഭാഗമായി ഉറക്കത്തകരാറിനെപ്പറ്റിയുള്ള വിവരങ്ങൾ താങ്കളിൽനിന്നും ശേഖരിക്കാനും അവലോകനം ചെയ്ത് സൂചകങ്ങൾ കണ്ടെത്താനും ഈ പഠനത്തിന്റെ ഭാഗമായി ഞങ്ങൾ ശ്രമിക്കുന്നു. താങ്കളുടെ മസ്തിഷ്കാഘാതത്തിൽ നിന്നുള്ള രോഗവിമുക്തിയെ ഇത് എങ്ങനെ ബാധിക്കുന്നു എന്നു മനസ്സിലാക്കാനായി 3 മാസത്തിനുശേഷവും താങ്കളെ പിന്തുടരും. ഗുരുതരമായ ഇന്ത്യയിലെ മസ്തിഷ്കാഘാതമുണ്ടായ ഏതൊരാൾക്കും പ്രകാരം പ്രവർത്തന നിലവാരം 3ൽ താഴെയുള്ള, മസ്തിഷ്കാഘാത സമഗ്ര പരിചരണത്തിനായുള്ള കേന്ദ്രം, സ്ട്രോക്ക് ഡിപ്പാർട്ട്മെന്റ്, SCTIMST യിൽ, 3-00 മാസം തുടർചികിത്സയ്ക്കായി വരുന്ന രോഗികളെ ഉൾപ്പെടുത്തുന്നതിന്റെയും ഉൾപ്പെടുത്താത്തതിന്റെയും മാനദണ്ഡപ്രകാരം തിരഞ്ഞെടുക്കും.

### പഠനത്തിന്റെ ഉദ്ദേശം

ഇന്ത്യയിലെ മസ്തിഷ്കാഘാത രോഗമുള്ള ഉറക്കരോഹിതുത്തിന്റെ സൂചകങ്ങളും മസ്തിഷ്കാഘാതം മൂലപ്പെടുന്നതിൽ അതിന്റെ പ്രഭാവവും കണ്ടെത്തുക എന്നതാണ് ഈ പഠനത്തിന്റെ ഉദ്ദേശം

### പഠനത്തിന്റെ നടപടികൾ

താങ്കൾ പങ്കെടുക്കുവാൻ സമ്മതിച്ചാൽ സ്ട്രോക്കിസ്റ്റുമായി ഒരു അഭിമുഖത്തിനും ശാരീരിക പരിശോധനയ്ക്കും വിധേയമാകുകയും ക്ലിനിക്കൽ കണ്ടെത്തലുകൾ രേഖപ്പെടുത്തുകയും

ചെയ്യും. താങ്കൾ മസ്തിഷ്കാഘാതരോഗിയുടെ തുടർചികിത്സയ്ക്കായി വരുന്ന സമയത്തെക്കുറിച്ച് ഇത് ആസൂത്രണം ചെയ്യും. ഇത് പഠനത്തിന്റെ ഭാഗമായി സമയബന്ധിതമായി ചെയ്യും. ഗവേഷകർ വിശദാംശങ്ങൾ താങ്കളുമായി പങ്കുവയ്ക്കും.

അപായങ്ങളും അസൗകര്യങ്ങളും
ഈ പഠനത്തിൽ സ്കൂറോളജിസ്റ്റ് നടത്തുന്ന സൂഷ്മപരിശോധനയായ 30 മുതൽ 40 മിനിട്ടിൽ പൂർത്തിയാകുന്ന നീളുന്ന ഒരു അഭിമുഖം മാത്രമേ ഉള്ളൂ. ഈ പഠനവുമായി ബന്ധപ്പെട്ട് പ്രത്യേകിച്ച് അപായങ്ങളൊന്നുമില്ല.

നേട്ടങ്ങൾ
ഈ ഗവേഷണപഠനത്തിൽ പങ്കെടുക്കുന്നതുകൊണ്ട് ഉടനെ താങ്കൾക്ക് നേട്ടമൊന്നും ഉണ്ടാകില്ല. എന്നിരുന്നാലും മസ്തിഷ്കാഘാതരോഗിയുടെ ഹൃസ്വകാല ഭാഗധേയത്തിൽ ഉറക്കമോഹിതുത്തിന്റെ ഉയർന്ന വ്യാപ്തിയിലും അതിന്റെ സ്വാധീനത്തിലും ഈ പഠനം വെളിച്ചം വീശുമെന്നതിനാൽ മസ്തിഷ്കാകാഘാത പരിചരണം നടത്തുന്നവർക്ക് ഉറക്കമോഹിതുകത്തെ പരമാവധി നേരത്തെ കണ്ടെത്തുന്നതിനും അതിന്റെ ചികിത്സയ്ക്കും ഭാവിയിൽ സഹായകമാകും.

രഹസ്യാത്മകത
താങ്കളുടെ സമ്മതപത്രം ഞങ്ങൾക്ക് പ്രധാനമാകയാൽ താങ്കളിൽ നടത്തിയ പരിശോധനകളുടെ ഫലങ്ങൾ വളരെ രഹസ്യമായിരിക്കും, മുകളിൽ പറഞ്ഞ ഗവേഷകർ മാത്രമേ മറ്റാർക്കും പരിശോധനാഫലങ്ങൾ അറിയുകയില്ല. ഈ പഠനഫലമായി ഉണ്ടാകുന്ന ഗവേഷണ പ്രസിദ്ധീകരണത്തിലോ പ്രദർശനത്തിലോ താങ്കളുടെ പേരോ തിരിച്ചറിയാനിടയാക്കുന്ന മറ്റേതെങ്കിലും വിശദാംശങ്ങളോ പ്രസിദ്ധീകരിക്കുകയുമില്ല.

അവകാശങ്ങൾ
ഈ പരീക്ഷണത്തിലെ താങ്കളുടെ പങ്കാളിത്തം സ്വമേധയായാണ്. താങ്കൾക്ക് സമ്മതമില്ലെങ്കിൽ ഈ പഠനത്തിൽ പങ്കെടുക്കേണ്ടതില്ല. പങ്കെടുക്കുന്നില്ലെന്ന് തീരുമാനിച്ചാലും മറ്റേതെന്ന നിലയിലുള്ള താങ്കളുടെ അവകാശങ്ങളൊന്നും നഷ്ടപ്പെടില്ല. പഠനത്തിന്റെ ഏത് ഘട്ടത്തിലും (സമ്മതപത്രം ഒപ്പിട്ടശേഷവും) താങ്കൾക്ക് പഠനത്തിൽ നിന്നും പിൻമാറാവുന്നതാണ്.

ബന്ധപ്പെടാനുള്ള വിവരങ്ങൾ

- താങ്കൾ ഈ വിവരങ്ങൾ വായിക്കുമ്പോൾ ചർച്ചചെയ്യാനും താങ്കൾക്കുണ്ടായേക്കാവുന്ന ചോദ്യങ്ങൾക്ക് ഉത്തരങ്ങൾ നൽകാനും താങ്കളെ ചികിത്സിക്കുന്ന ഡോക്ടർ ഉണ്ടാവും. താങ്കൾക്കെന്തെങ്കിലും ചോദ്യങ്ങളുണ്ടെങ്കിൽ ഒരുവായി ബന്ധപ്പെടുക

ഡോ. അവിനാശ് കുൽക്കർണി,
സീനിയർ കൺസൾട്ടന്റ്, സ്കൂറോളജി ഡിപ്പാർട്ട്മെന്റ്,
ശ്രീ ചിത്ര തിരുനാൾ ഇൻസ്റ്റിറ്റ്യൂട്ട് ഫോർ മെഡിക്കൽ സയൻസസ് ആന്റ് ടെക്നോളജി
ഫോൺ 91 8792466031, ഇമെയിൽ: avinashkulkarni@sctimst.ac.in

ഗവേഷണത്തെപ്പറ്റി താങ്കൾക്ക് ചോദ്യങ്ങൾ, ഉൽകണ്ഠം അല്ലെങ്കിൽ പരാതി എന്നിവയുണ്ടെങ്കിൽ ഒരുവായി ബന്ധപ്പെടുക:
ഡോ. ശ്രീനിവാസ് ജി
മെമ്പർ സെക്രട്ടറി, ഇൻസ്റ്റിറ്റ്യൂഷണൽ എത്തിക്സ് കമ്മിറ്റി
ശ്രീ ചിത്ര തിരുനാൾ ഇൻസ്റ്റിറ്റ്യൂട്ട് ഫോർ മെഡിക്കൽ സയൻസസ് ആന്റ് ടെക്നോളജി
ഫോൺ 0471- 2524689, ഇമെയിൽ: iec.mem.sec@sctimst.ac.in

**SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES AND TECHNOLOGY, THIRUVANANTHAPURAM, KERALA - 695011**

**COMPREHENSIVE STROKE CARE PROGRAM**

**INFORMED CONSENT FORM**

**Title of Study:**
PREDICTORS OF SLEEP DYSFUNCTION AFTER ISCHEMIC STROKE AND ITS IMPACT ON STROKE RECOVERY

**Principal Investigator:**
Dr. Avinash T Kulkarni, Senior Resident, Department of Neurology, SCTIMST

**Co-Principal Investigator:**
Dr.Sapna Erat Sreedharan, Additional Professor, Department of Neurology, SCTIMST
Dr.Sylaja P N, Professor & HOD, Department of Neurology, SCTIMST

Please tick the following points:

| | |
|---|---|
| I agree to participate as a participant in the study described in the Participant Information Sheet attached to this form. | [ ] |
| I acknowledge that I have read the Participant Information Sheet, which explains why I have been selected, the aims of the study and the nature and the possible risks of the investigation, and the information sheet has been explained to me to my satisfaction. | [ ] |
| Before signing this consent form, I have been given the opportunity of asking any questions relating to any possible physical and mental harm I might suffer as a result of my participation and I have received satisfactory answers. | [ ] |
| I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected. | [ ] |
| I agree that research data gathered from the results of the study may be published, provided that I cannot be identified. | [ ] |

| | |
|---|---|
| I understand that if I have any questions relating to my participation in this research, I may contact my doctor, who will be happy to answer them. | [ ] |
| I acknowledge receipt of a copy of this Consent Form and the Participant Information Sheet attached to this form | [ ] |

_____
Name of Participant

_____ _____
_____
Signature of Participant
Date Time

_____
Name of Caretaker or Next of Kin
*(If patient not directly consented)*

_____
Relationship with the patient

_____ _____
_____
Signature of Caretaker or Next of Kin
Date Time

_____
Name of Witness

_____ _____
_____
Signature of Witness

Date Time

_____

Name of Person conducting Informed Consent discussion

_____ _____ _____

Signature of Person conducting Informed Consent discussion

Date Time

ശ്രീ ചിത്ര തിരുനാൾ ഇൻസ്റ്റിറ്റ്യൂട്ട് ഫോർ മെഡിക്കൽ സയൻസസ് ആന്റ് ടെക്നോളജി,
തിരുവനന്തപുരം, കേരളം – 695011
മസ്തിഷ്കാഘാത പരിചരണത്തിനായുള്ള സമഗ്ര പരിപാടി

രോഗിക്കുള്ള കാര്യവിവരണപത്രം

പഠനശീർഷകം:
ഈഷ്മിക് മസ്തിഷ്കാഘാത ശേഷമുള്ള ഉറക്കരോഹിത്യത്തിന്റെ സൂചകങ്ങളും മസ്തിഷ്കാഘാതം ഭേദപ്പെടുന്നതിൽ അതിന്റെ പ്രഭാവവും.

പ്രധാന ഗവേഷകൻ

ഡോ. അവിനാഷ് റ്റി കുൽക്കർണി, സീനിയർ റെസിഡന്റ്, ന്യൂറോളജി ഡിപ്പാർട്ട്മെന്റ്, SCTIMST

സഹ പ്രധാന ഗവേഷക

ഡോ. സപ്ന ഏറാട്ട് ശ്രീധരൻ, അപ്രാഫസർ, ന്യൂറോളജി ഡിപ്പാർട്ട്മെന്റ്, SCTIMST
(താങ്കളുടെയുന്നവയിൽ ദയവായി ടിക് ചെയ്യുക)

| | |
|---|---|
| ഈ പത്രികയോടൊപ്പമുള്ള, പങ്കെടുക്കുന്നവർക്കുള്ള കാര്യവിവരണപത്രത്തിൽ വിശദീകരിക്കുന്ന പഠനത്തിൽ പങ്കെടുക്കാൻ ഞാൻ സമ്മതിക്കുന്നു. | [ ] |
| എന്നെ എന്തുകൊണ്ട് തിരഞ്ഞെടുത്തു, പഠനത്തിന്റെ ഉദ്ദേശം, സ്വഭാവം, പരിശോധനയിൽ ഉണ്ടാവാനിടയുള്ള അപായങ്ങൾ എന്നിവ വിവരിക്കുന്ന പങ്കെടുക്കുന്നവർക്കുള്ള കാര്യവിവരണപത്രം വായിച്ചതായും എന്റെ തൃപ്തിയ്ക്കനുസരിച്ച് വിശദീകരിച്ചുതന്നതായും ഞാൻ സമ്മതിക്കുന്നു. | [ ] |
| സമ്മതപത്രത്തിൽ ഒപ്പു വയ്ക്കുന്നതിനുമുമ്പ്, ഈ പഠനത്തിൽ പങ്കെടുക്കുന്നതുകൊണ്ട് ശാരീരികവും മാനസികവുമായ എന്തെങ്കിലും ഹാനി എനിക്ക് ഉണ്ടാകാൻ സാദ്ധ്യതയുണ്ടോ എന്നതുമായി ബന്ധപ്പെട്ട ചോദ്യങ്ങൾ ചോദിക്കാൻ എനിക്ക് അവസരം ഉണ്ടാവുകയും തൃപ്തികരമായ മറുപടി ലഭിക്കുകയും ചെയ്തു | [ ] |
| എന്റെ പങ്കാളിത്തം സ്വമേധയായുള്ളതാണെന്നും, കാരണമൊന്നും നൽകാതെയും എന്റെ തുടർചികിത്സയെ ബാധിക്കാതെയും ഏതു സമയത്തും എനിക്ക് പിൻമാറാൻ സ്വാതന്ത്ര്യമുണ്ടെന്നും മനസ്സിലാക്കുന്നു. | [ ] |
| പഠനഫലമായി ശേഖരിച്ച വിവരങ്ങൾ പ്രസിദ്ധീകരിക്കുമ്പോൾ എന്നെ തിരിച്ചറിയാനിടയാകുന്നതൊന്നും വെളിപ്പെടുത്തുകയില്ലെന്ന് ഞാൻ മനസ്സിലാക്കുന്നു. | [ ] |
| ഗവേഷണത്തിൽ പങ്കെടുക്കുന്നതുമായി ബന്ധപ്പെട്ട് എനിക്ക് ചോദ്യങ്ങളുണ്ടെങ്കിൽ എനിക്ക് ഡോക്ടറെ ബന്ധപ്പെടാമെന്നും ഉത്തരം തരുന്നതിൽ അദ്ദേഹത്തിന് സന്തോഷമേയുള്ളൂവെന്നും ഞാൻ മനസ്സിലാക്കുന്നു. | [ ] |
| ഈ പത്രികയോടൊപ്പം നൽകിയിട്ടുള്ള പങ്കാളികൾക്കുള്ള വിവരണപത്രവും സമ്മതപത്രവും കിട്ടിയതായി ഞാൻ അറിയിക്കുന്നു. | [ ] |

_____
പങ്കെടുക്കുന്നയാളുടെ പേര്

_____ _____ _____

## QUESTIONNAIRES USED IN THE STUDY

### 1. EPWORTH SLEEPINESS SCALE

### The Epworth Sleepiness Scale

The Epworth Sleepiness Scale is widely used in the field of sleep medicine as a subjective measure of a patient's sleepiness. The test is a list of eight situations in which you rate your tendency to become sleepy on a scale of 0, no chance of dozing, to 3, high chance of dozing. When you finish the test, add up the values of your responses. Your total score is based on a scale of 0 to 24. The scale estimates whether you are experiencing excessive sleepiness that possibly requires medical attention.

**How Sleepy Are You?**
How likely are you to doze off or fall asleep in the following situations? You should rate your chances of dozing off, not just feeling tired. Even if you have not done some of these things recently try to determine how they would have affected you. For each situation, decide whether or not you would have:

- No chance of dozing =0
- Slight chance of dozing =1
- Moderate chance of dozing =2
- High chance of dozing =3

Write down the number corresponding to your choice in the right hand column. Total your score below.

| Situation | Chance of Dozing |
|---|---|
| Sitting and reading | • |
| Watching TV | • |
| Sitting inactive in a public place (e.g., a theater or a meeting) | • |
| As a passenger in a car for an hour without a break | • |
| Lying down to rest in the afternoon when circumstances permit | • |
| Sitting and talking to someone | • |
| Sitting quietly after a lunch without alcohol | • |
| In a car, while stopped for a few minutes in traffic | • |

Total Score = _____

**Analyze Your Score**

**Interpretation:**

**0-7:** It is unlikely that you are abnormally sleepy.
**8-9:** You have an average amount of daytime sleepiness.
**10-15:** You may be excessively sleepy depending on the situation. You may want to consider seeking medical attention.
**16-24:** You are excessively sleepy and should consider seeking medical attention.

Reference: Johns MW. A new method for measuring daytime sleepiness: The Epworth Sleepiness Scale. *Sleep* 1991; 14(6):540-5.

## 2. INSOMNIA SEVERITY INDEX (ISI)

**Insomnia Severity Index**

The Insomnia Severity Index has seven questions. The seven answers are added up to get a total score. When you have your total score, look at the 'Guidelines for Scoring/Interpretation' below to see where your sleep difficulty fits.

For each question, please CIRCLE the number that best describes your answer.

*Please rate the CURRENT (i.e. LAST 2 WEEKS) SEVERITY of your insomnia problem(s).*

| Insomnia Problem | None | Mild | Moderate | Severe | Very Severe |
|---|---|---|---|---|---|
| 1. Difficulty falling asleep | 0 | 1 | 2 | 3 | 4 |
| 2. Difficulty staying asleep | 0 | 1 | 2 | 3 | 4 |
| 3. Problems waking up too early | 0 | 1 | 2 | 3 | 4 |

4. How SATISFIED/DISSATISFIED are you with your CURRENT sleep pattern?

| Very Satisfied | Satisfied | Moderately Satisfied | Dissatisfied | Very Dissatisfied |
|---|---|---|---|---|
| 0 | 1 | 2 | 3 | 4 |

5. How NOTICEABLE to others do you think your sleep problem is in terms of impairing the quality of your life?

| Not at all Noticeable | A Little | Somewhat | Much | Very Much Noticeable |
|---|---|---|---|---|
| 0 | 1 | 2 | 3 | 4 |

6. How WORRIED/DISTRESSED are you about your current sleep problem?

| Not at all Worried | A Little | Somewhat | Much | Very Much Worried |
|---|---|---|---|---|
| 0 | 1 | 2 | 3 | 4 |

7. To what extent do you consider your sleep problem to INTERFERE with your daily functioning (e.g. daytime fatigue, mood, ability to function at work/daily chores, concentration, memory, mood, etc.) CURRENTLY?

| Not at all Interfering | A Little | Somewhat | Much | Very Much Interfering |
|---|---|---|---|---|
| 0 | 1 | 2 | 3 | 4 |

**Guidelines for Scoring/Interpretation:**

Add the scores for all seven items (questions 1 + 2 + 3 + 4 + 5 +6 + 7) = _____ your total score

Total score categories:
0–7 = No clinically significant insomnia
8–14 = Subthreshold insomnia
15–21 = Clinical insomnia (moderate severity)
22–28 = Clinical insomnia (severe)

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## 3. PITTSBURGH SLEEP QUALITY INDEX (PSQI)

### PITTSBURGH SLEEP QUALITY INDEX (PSQI)

**INSTRUCTIONS:** The following questions relate to your usual sleep habits during the past month only. Your answers should indicate the most accurate reply for the majority of days and nights in the past month. Please answer all questions.

1. During the past month, when have you usually gone to bed at night?

   USUAL BED TIME _____

2. During the past month, how long (in minutes) has it usually take you to fall asleep each night?

   NUMBER OF MINUTES _____

3. During the past month, when have you usually gotten up in the morning?

   USUAL GETTING UP TIME _____

4. During the past month, how many hours of actual sleep did you get at night? (This may be different than the number of hours you spend in bed.)

   HOURS OF SLEEP PER NIGHT _____

**INSTRUCTIONS:** For each of the remaining questions, check the one best response. Please answer all questions.

5. During the past month, how often have you had trouble sleeping because you...

| | Not during the past month | Less than once a week | Once or twice a week | Three or more times a week |
|---|---|---|---|---|
| (a) ...cannot get to sleep within 30 minutes | ☐ | ☐ | ☐ | ☐ |
| (b) ...wake up in the middle of the night or early morning | ☐ | ☐ | ☐ | ☐ |
| (c) ...have to get up to use the bathroom | ☐ | ☐ | ☐ | ☐ |
| (d ...cannot breathe comfortably | ☐ | ☐ | ☐ | ☐ |
| (e) ...cough or snore loudly | ☐ | ☐ | ☐ | ☐ |
| (f) ...feel too cold | ☐ | ☐ | ☐ | ☐ |
| (g) ...feel too hot | ☐ | ☐ | ☐ | ☐ |
| (h) ...had bad dreams | ☐ | ☐ | ☐ | ☐ |
| (i) ...have pain | ☐ | ☐ | ☐ | ☐ |
| (j) Other reason(s), please describe _____ _____ | | | | |
| How often during the past month have you had trouble sleeping because of this? | ☐ | ☐ | ☐ | ☐ |

***PSQI Page 1***

| | Very good | Fairly good | Fairly bad | very bad |
|---|---|---|---|---|
| 6. During the past month, how would you rate your sleep quality overall? | ☐ | ☐ | ☐ | ☐ |

| | Not during the past month | Less than once a week | Once or twice a week | Three or more times a week |
|---|---|---|---|---|
| 7. During the past month, how often have you taken medicine (prescribed or "over the counter") to help you sleep? | ☐ | ☐ | ☐ | ☐ |
| 8. During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity? | ☐ | ☐ | ☐ | ☐ |

| | No problem at all | Only a very slight problem | Somewhat of a problem | A very big problem |
|---|---|---|---|---|
| 9. During the past month, how much of a problem has it been for you to keep up enough enthusiasm to get things done? | ☐ | ☐ | ☐ | ☐ |

| | No bed partner or roommate | Partner/ roommate in other room | Partner in same room, but not same bed | Partner in same bed |
|---|---|---|---|---|
| 10. During the past month, how much of a problem has it been for you to keep up enough enthusiasm to get things done? | ☐ | ☐ | ☐ | ☐ |

If you have a roommate or bed partner, ask him/her how often in the past month you have had...

| | Not during the past month | Less than once a week | Once or twice a week | Three or more times a week |
|---|---|---|---|---|
| (a) ...loud snoring | ☐ | ☐ | ☐ | ☐ |
| (b) ...long pauses between breaths while asleep | ☐ | ☐ | ☐ | ☐ |
| (c) ...legs twitching or jerking while you sleep | ☐ | ☐ | ☐ | ☐ |
| (d) ...episodes of disorientation or confusion during sleep | ☐ | ☐ | ☐ | ☐ |
| (e) Other restlessness while you sleep; please describe | ☐ | ☐ | ☐ | ☐ |

_____

_____

***PSQI Page 2***

**SCORING INSTRUCTIONS FOR THE PITTSBURGH SLEEP QUALITY INDEX:**

The Pittsburgh Sleep Quality Index (PSQI) contains 19 self-rated questions and 5 questions rated by the bed partner or roommate (if one is available). Only self-rated questions are included in the scoring. The 19 self-rated items are combined to form seven "component" scores, each of which has a range of 0-3 points. In all cases, a score of "0" indicates no difficulty, while a score of "3" indicates severe difficulty. The seven component scores are then added to yield one "global" score, with a range of 0-21 points, "0" indicating no difficulty and "21 " indicating severe difficulties in all areas.

Scoring proceeds as follows:

## Component 1: Subjective sleep quality

Examine question #6, and assign scores as follows:

| Response | Component 1 score |
|---|---|
| "Very good" | 0 |
| "Fairly good" | 1 |
| "Fairly bad" | 2 |
| "Very bad" | 3 |

***Component 1 score:*** _____

## Component 2: Sleep latency

1. Examine question #2, and assign scores as follows:

| Respo\nse | Score |
|---|---|
| ≤15 minutes | 0 |
| 16-30 minutes | 1 |
| 31-60 minutes | 2 |
| > 60 minutes | 3 |
| *Question #2 score:* | _____ |

2. Examine question #5a, and assign scores as follows:

| Response | Score |
|---|---|
| Not during the past month | 0 |
| Less than once a week | 1 |
| Once or twice a week | 2 |
| Three or more times a week | 3 |
| *Question #5a score:* | _____ |

3. Add #2 score and #5a score

*Sum of #2 and #5a:* _____

4. Assign component 2 score as follows:

| Sum of #2 and #5a | Component 2 score |
|---|---|
| 0 | 0 |
| 1-2 | 1 |
| 3-4 | 2 |
| 5-6 | 3 |

***Component 2 score:*** _____

### Component 3: Sleep duration

Examine question #4, and assign scores as follows:

| Response | Component 3 score |
|---|---|
| > 7 hours | 0 |
| 6-7 hours | 1 |
| 5-6 hours | 2 |
| < 5 hours | 3 |

***Component 3 score:*** _____

---

### Component 4: Habitual sleep efficiency

1. Write the number of hours slept (question #4) here: _____
2. Calculate the number of hours spent in bed:

   Getting up time (question #3): _____

   Bedtime (question #1): _____

   _____

   *Number of hours spent in bed:* _____

3. Calculate habitual sleep efficiency as follows:

   (Number of hours slept/Number of hours spent in bed) X 100 = Habitual sleep efficiency (%)

   (_____ / _____ ) X 100 = %

4. Assign component 4 score as follows:

| Habitual sleep efficiency % | Component 4 score |
|---|---|
| > 85% | 0 |
| 75-84% | 1 |
| 65-74% | 2 |
| < 65% | 3 |

***Component 4 score:*** _____

## Component 5: Step disturbances

1. Examine questions #5b-5j, and assign scores for each question as follows:

| Response | Score |
|---|---|
| Not during the past month | 0 |
| Less than once a week | 1 |
| Once or twice a week | 2 |
| Three or more times a week | 3 |

*5b score:* _____
*5c score:* _____
*5d score:* _____
*5e score:* _____
*5f score:* _____
*5g score:* _____
*5h score:* _____
*5i score:* _____
*5j score:* _____

2. Add the scores for questions #5b-5j:

*Sum of #5b-5j:* _____

3. Assign component 5 score as follows:

| Sum of #5b-5j | Component 5 score |
|---|---|
| 0 | 0 |
| 1-9 | 1 |
| 10-18-4 | 2 |
| 19-27 | 3 |

***Component 5 score:*** _____

## Component 6: Use of sleeping medication

Examine question #7 and assign scores as follows:

| Response | Component 6 score |
|---|---|
| Not during the past month | 0 |
| Less than once a week | 1 |
| Once or twice a week | 2 |
| Three or more times a week | 3 |

***Component 6 score:*** _____

## Component 7: Daytime dysfunction

1. Examine question #8, and assign scores as follows:

| Response | Score |
|---|---|
| Never | 0 |
| Once or twice | 1 |
| Once or twice each week | 2 |
| Three or more times each week | 3 |

*Question#8 score:* _____

2. Examine question #9, and assign scores as follows:

| Response | Score |
|---|---|
| No problem at all | 0 |
| Only a very slight problem | 1 |
| Somewhat of a problem | 2 |
| A very big problem | 3 |

*Question #9 score:* _____

3. Add the scores for question #8 and #9:

*Sum of #8 and #9:* _____

4. Assign component 7 score as follows:

| Sum of #8 and #9 | Component 7 score |
|---|---|
| 0 | 0 |
| 1-2 | 1 |
| 3-4 | 2 |
| 5-6 | 3 |

***Component 7 score:*** _____

## Global PSQI Score

Add the seven component scores together:

***Global PSQI Score:*** _____

## 4. PATIENT HEALTH QUESTIONNAIRE 9 (PHQ 9)

**PATIENT HEALTH QUESTIONNAIRE (PHQ-9)**

**ID #:** _____ **DATE:** _____

Over the last 2 *weeks*, how often have you been bothered by any of the following problems?
*(use "✓" to indicate your answer)*

| | Not at all | Several days | More than half the days | Nearly every day |
|---|---|---|---|---|
| **1.** Little interest or pleasure in doing things | 0 | 1 | 2 | 3 |
| **2.** Feeling down, depressed, or hopeless | 0 | 1 | 2 | 3 |
| **3.** Trouble falling or staying asleep, or sleeping too much | 0 | 1 | 2 | 3 |
| **4.** Feeling tired or having little energy | 0 | 1 | 2 | 3 |
| **5.** Poor appetite or overeating | 0 | 1 | 2 | 3 |
| **6.** Feeling bad about yourself—or that you are a failure or have let yourself or your family down | 0 | 1 | 2 | 3 |
| **7.** Trouble concentrating on things, such as reading the newspaper or watching television | 0 | 1 | 2 | 3 |
| **8.** Moving or speaking so slowly that other people could have noticed. Or the opposite—being so figety or restless that you have been moving around a lot more than usual | 0 | 1 | 2 | 3 |
| **9.** Thoughts that you would be better off dead, or of hurting yourself | 0 | 1 | 2 | 3 |
| add columns | | + | + | |
| *(Healthcare professional: For interpretation of TOTAL, please refer to accompanying scoring card).* | TOTAL: | | | |

**10.** If you checked off *any problems*, how *difficult* have these problems made it for you to do your work, take care of things at home, or get along with other people?

- Not difficult at all _____
- Somewhat difficult _____
- Very difficult _____
- Extremely difficult _____



A2663B 10-04-2005

## PHQ-9 Patient Depression Questionnaire

**For initial diagnosis:**

1. Patient completes PHQ-9 Quick Depression Assessment.
2. If there are at least 4 ✓'s in the shaded section (including Questions #1 and #2), consider a depressive disorder. Add score to determine severity.

***Consider Major Depressive Disorder***

- if there are at least 5 ✓'s in the shaded section (one of which corresponds to Question #1 or #2)

***Consider Other Depressive Disorder***

- if there are 2-4 ✓'s in the shaded section (one of which corresponds to Question #1 or #2)

**Note:** Since the questionnaire relies on patient self-report, all responses should be verified by the clinician, and a definitive diagnosis is made on clinical grounds taking into account how well the patient understood the questionnaire, as well as other relevant information from the patient.
Diagnoses of Major Depressive Disorder or Other Depressive Disorder also require impairment of social, occupational, or other important areas of functioning (Question #10) and ruling out normal bereavement, a history of a Manic Episode (Bipolar Disorder), and a physical disorder, medication, or other drug as the biological cause of the depressive symptoms.

**To monitor severity over time for newly diagnosed patients or patients in current treatment for depression:**

1. Patients may complete questionnaires at baseline and at regular intervals (eg, every 2 weeks) at home and bring them in at their next appointment for scoring or they may complete the questionnaire during each scheduled appointment.
2. Add up ✓'s by column. For every ✓: Several days = 1 More than half the days = 2 Nearly every day = 3
3. Add together column scores to get a TOTAL score.
4. Refer to the accompanying **PHQ-9 Scoring Box** to interpret the TOTAL score.
5. Results may be included in patient files to assist you in setting up a treatment goal, determining degree of response, as well as guiding treatment intervention.

**Scoring: add up all checked boxes on PHQ-9**

**For every** ✓ Not at all = 0; Several days = 1;
More than half the days = 2; Nearly every day = 3

**Interpretation of Total Score**

| Total Score | Depression Severity |
|---|---|
| 1-4 | Minimal depression |
| 5-9 | Mild depression |
| 10-14 | Moderate depression |
| 15-19 | Moderately severe depression |
| 20-27 | Severe depression |



A2662B 10-04-2005

## 5. ZARIT BURDEN INTERVIEW

# The Zarit Burden Interview

0: NEVER
1: RARELY
2: SOMETIMES
3: QUITE FREQUENTLY
4: NEARLY ALWAYS

Please circle the response the best describes how you feel.

| Question | Score |
|---|---|
| 1 Do you feel that your relative asks for more help than he/she needs? | 0 1 2 3 4 |
| 2 Do you feel that because of the time you spend with your relative that you don't have enough time for yourself? | 0 1 2 3 4 |
| 3 Do you feel stressed between caring for your relative and trying to meet other responsibilities for your family or work? | 0 1 2 3 4 |
| 4 Do you feel embarrassed over your relative's behaviour? | 0 1 2 3 4 |
| 5 Do you feel angry when you are around your relative? | 0 1 2 3 4 |
| 6 Do you feel that your relative currently affects our relationships with other family members or friends in a negative way? | 0 1 2 3 4 |
| 7 Are you afraid what the future holds for your relative? | 0 1 2 3 4 |
| 8 Do you feel your relative is dependent on you? | 0 1 2 3 4 |
| 9 Do you feel strained when you are around your relative? | 0 1 2 3 4 |
| 10 Do you feel your health has suffered because of your involvement with your relative? | 0 1 2 3 4 |
| 11 Do you feel that you don't have as much privacy as you would like because of your relative? | 0 1 2 3 4 |
| 12 Do you feel that your social life has suffered because you are caring for your relative? | 0 1 2 3 4 |

| Question | Score |
|---|---|
| 13 Do you feel uncomfortable about having friends over because of your relative? | 0 1 2 3 4 |
| 14 Do you feel that your relative seems to expect you to take care of him/her as if you were the only one he/she could depend on? | 0 1 2 3 4 |
| 15 Do you feel that you don't have enough money to take care of your relative in addition to the rest of your expenses? | 0 1 2 3 4 |
| 16 Do you feel that you will be unable to take care of your relative much longer? | 0 1 2 3 4 |
| 17 Do you feel you have lost control of your life since your relative's illness? | 0 1 2 3 4 |
| 18 Do you wish you could leave the care of your relative to someone else? | 0 1 2 3 4 |
| 19 Do you feel uncertain about what to do about your relative? | 0 1 2 3 4 |
| 20 Do you feel you should be doing more for your relative? | 0 1 2 3 4 |
| 21 Do you feel you could do a better job in caring for your relative? | 0 1 2 3 4 |
| 22 Overall, how burdened do you feel in caring for your relative? | 0 1 2 3 4 |

Interpretation of Score:

0 - 21 little or no burden

21 - 40 mild to moderate burden

41 - 60 moderate to severe burden

61 - 88 severe burden

PROFORMA

**Sree Chitra Tirunal Institute for Medical Sciences and Technology**
**Thiruvananthapuram, Kerala-695011**

TITLE OF STUDY:

**PREDICTORS OF SLEEP DYSFUNCTION AFTER ISCHEMIC STROKE AND ITS IMPACT ON STROKE RECOVERY**

## 1. Personal Data:

1.1. Age ---------- years
1.2 Sex ---------- 1.Male 2.female
1.3. Hospital number
1.4. Patient study number : ----------
1.5. Patient education - 1. Elementary (completed $5^{th}$), 2. Secondary school (completed $10^{th}$), 3.Higher secondary (10+2), 4.Graduate , 5. Postgraduate

## DETAILS ABOUT THE ISCHEMIC STROKE

## 2. Risk factors:(1=Yes, 2=No)

2.1. Hypertension----------
2.2. Diabetes mellitus--------
2.3a. Smoking – Y/N
2.3b. Alcoholism----------
2.4. Coronary artery disease--------
2.5. Peripheral vascular disease--------
2.6. Hyperlipidemia----------
2.7. History of prior stroke --------
2.8. History of prior TIA----------
2.9. Patients on treatment ----------

2.9a. If yes, Type of treatment ---------- 1.Aspirin 2.Clopidogrel 3.Statins 4. Warfarin 5. NOAC
2.10. Renal dysfunction ----------
2.11.History of psychiatric illness ----------
2.12.History of sleep dysfunction before stroke ---------
2.12.History of psychiatric medications intake before onset of stroke ----------

**3. Clinical Examination:**
3.1. NIHSS at admission ----------
3.2. NIHSS at discharge……
3.3. mRS at admission ----------
3.4. mRS at discharge----------
3.5. GCS at admission
3.6. GCS at discharge

**4. Other Investigations during the time of acute stroke:**
4.1. Hb A1C----------
4.2. Blood Urea----
4.3. Creatinine---

**5. Diagnostic imaging**
**5.1 CT scan** ----------1. Normal.2. New infarct 3. Old infarct 4. Small vessel Ischemic changes 5.Not done
5.1a. Infarct pattern----------1. Perforator 2. Territorial 3. Border zone pattern 4. Mixed
If mixed specify the combination……..
5.1b. Arterial Territory ----------1.ACA 2.MCA-complete 3.MCA-Inferior division 4.MCA superior division 5.MCA subcortical 6. Posterior circulation
5.1c. CT ASPECTS at admission ----------

**5.2 CT angiogram**
5.2a. CT angiogram neck -----1. Normal 2.abnormal 3.not done.

5.2b. If abnormal Specify ----------, Percentage of stenosis --------- (Exact percentage)only if more than 50%
5.2c. CTA Intracranial --------1. Normal 2.abnormal 3.not done .
5.2d. If abnormal Specify ---------- 1.Intracranial ICA 2.MCA-proximal/mid/distal

**5.3 MRI scan**
5.3a. Territory ----------1.right hemispheric 2 . left hemispheric 3.Posterior circulation 4. Bihemispheric 5. Bihemispheric + posterior circulation
5.3c. If small vessel ischemic changes grade ----------
5.3d. Stroke subtype----------1.LVAD 2.Cardioembolic.3 Other Specific causes.4.Undetermined 5.lacunar

**5.4 MRA neck**----------1.normal 2.abnormal 3.Not done**. S**pecify----------
**5.5 MRA intracranial**----------1.normal 2. Stenotic 4. Occluded 5. Irregular and occluded
**5.7 DSA**---------- 1.Normal 2.Abnormal 3.Not done
Data entry same as CTA

**6. Thrombolysis**
6.1. If thrombolysed----------1.Yes 2.No
6.2. If yes ----------1.intravenous 2.Mechanical 3.Bridging

**7. Treatment at discharge:(1=Yes, 2= No)**
7.1. Aspirin----------
7.2. Clopidogrel----------
7.3. Aggrenox(Aspirin + Dipyridamole)----------
7.4. Anticoagulation-VKA/NOAC
7.5. Statins ----------

**8.STROKE ETIOLOGY BY TOAST CLASSIFICATION**

Lacunar, Large artery atherosclerotic disease, Cardioembolism, Other determined etiology, Undetermined

**9. 3 MONTH DATA**

9.1Any recurrent strokes in 3 months -if yes,specify(TIA/ischemic stroke/ICH)

9.2.Any cardiac events in 3 months

9.3.Any other complications in the 3 months-if yes-specify(infections/DVT/Metabolic/seizures)

9.4.Any hospitalization in 3 months-if yes-admitting diagnosis

9.5. 3 month NIHSS

9.6. 3 month MRS

9.7. If died-cause of death

10. **Questionnaires (scores at 3 months)**

10.1. Epworth sleepiness scale – score ---

10.2. Insomnia severity index --- score---

10.3. Pittsburgh Sleep quality index(PSQI)--- score ----

10.4. Patient health questionnaire 9(PHQ-9)—score –

10.5. Caregiver burden scale (CBS) (Zarit's)—score—

**11. Outcome at 6 months**

11.1. Any recurrent strokes in 3 months -if yes,specify(TIA/ischemic stroke/ICH

11.2.Any cardiac events in 3 months

11.3.Any other complications in the 3 months-if yes-specify(infections/DVT/Metabolic/seizures)

11.4.Any hospitalization in 3 months-if yes-admitting diagnosis

11.5.mRS at 6 months----------

11.6. If died, cause of death---------- 1.Vascular 2.Non vascular. Specify----------

# PLAGIARISM CHECK REPORT

PLAGIARISM SCAN REPORT

**Date** July 19, 2023

**Exclude URL:** NO

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