A STUDY ON "INCIDENCE OF STROKE IN PATIENTS PRESENTING WITH ACUTE DIZZINESS, VERTIGO AND IMBALANCE IN EMERGENCY DEPARTMENT"

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Dept. of .....GENERAL.....MEDICINE..... **Thanjavur Medical College, Thanjavur**

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DR. C. BABU ANAND MD,
GUIDE, PROFESSOR OF MEDICINE
DEPARTMENT OF GENERAL MEDICINE
THANJAVUR MEDICAL COLLEGE HOSPITAL,

PROF. DR. D. NEHRU, M.D.,
PROFESSOR AND H.O.D,
DEPARTMENT OF GENERAL MEDICINE,
THANJAVUR MEDICAL COLLEGE HOSPITAL,

PROF. DR. S. JEYAKUMAR M.S, MCH, DNB, FRCS(EDIN)
THE DEAN
THANJAVUR MEDICAL COLLEGE HOSPITAL,
THANJAVUR

DECLARATION

I solemnly declare that the dissertation titled "***A STUDY ON INCIDENCE OF STROKE IN PATIENTS PRESENTING WITH ACUTE DIZZINESS, VERTIGO AND IMBALANCE IN EMERGENCY DEPARTMENT***" was done by me from MARCH 2017 TO AUGUST 2017 under the guidance and supervision of **PROF. DR. C. BABU ANAND MD.**

This dissertation is submitted to **THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY** towards the partial fulfilment of the requirement for the award of MD Degree in General Medicine (Branch I).

Place: Thanjavur **Dr. AMITH VISWANATH**

Date:

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Date: *Dr. Amith Viswanath*

CERTIFICATE – II

This is to certify that this dissertation work titled "***A STUDY ON INCIDENCE OF STROKE IN PATIENTS PRESENTING WITH ACUTE DIZZINESS, VERTIGO AND IMBALANCE IN EMERGENCY DEPARTMENT*** " of the candidate DR. AMITH VISWANATH with registration Number 201511201 for the award of M.D in the branch of General Medicine. I personally verified the urkund.com website for the purpose of plagiarism Check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows 2% of plagiarism in the dissertation.

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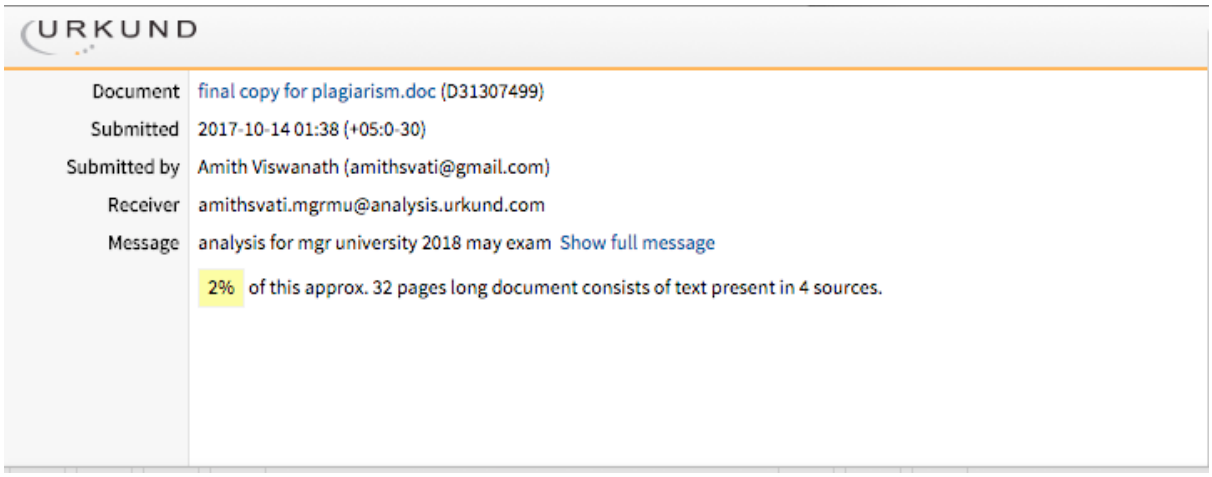

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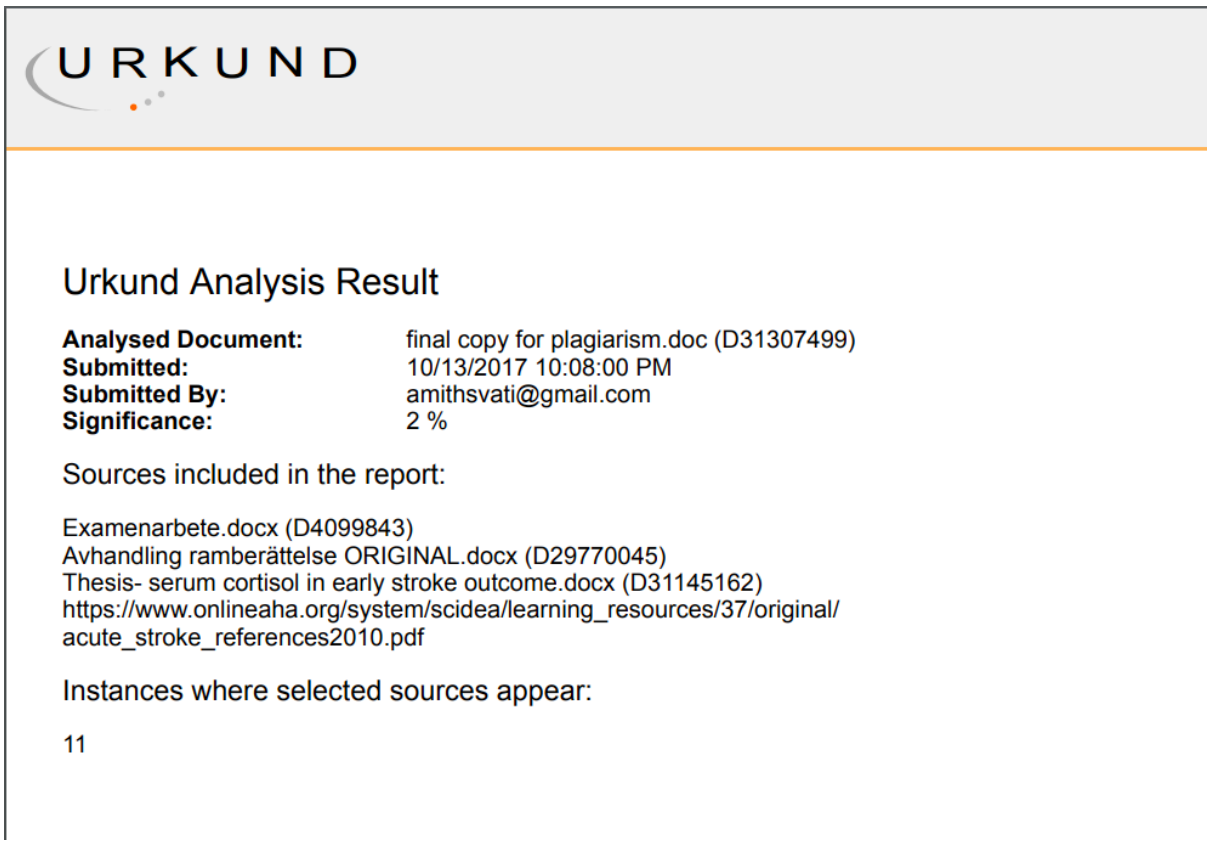

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LIST OF ABBREVIATIONS USED

ACA – ANTERIOR CEREBRAL ARTERY

ACI – ANTERIOR CIRCULATION INFARCTION

ADC – APPARENT DIFFUSION COEFFICIENT

ASPECTS – ALBERTA STROKE PROGRAM EARLY CT SCORE

BPM – BEATS PER MINUTE

CI – CONFIDENCE INTERVAL

CN – CRANIAL NERVE

CNS – CENTRAL NERVOUS SYSTEM

CT – COMPUTED TOMOGRAPHY

CTA – CT ANGIOGRAPHY

CTA-SI – CTA-SOURCE IMAGES

CTP – CT PERFUSION IMAGING

CTP-SI – CTP – SOURCE IMAGES

DWI – DIFFUSION WEIGHTED IMAGING

ECG – ELECTROCARDIOGRAM

ECVA- EXTRACRANIAL VERTEBRAL ARTERY

HINTS – HEAD-IMPULSE-NYSTAGMUS-TEST OF SKEW

ICVA – INTRACRANIAL VERTEBRAL ARTERY

IV – INTRAVENOUS

LOC – LOSS OF CONSCIOUSNESS

MCA – MIDDLE CEREBRAL ARTERY

MRI – MAGNETIC RESONANCE IMAGING

NCCT – NON-CONTRAST CT

NS – NOT SIGNIFICANT

OR – ODDS RATIO

PCI – POSTERIOR CIRCULATION INFARCTION

PICA – POSTERIOR INFERIOR CEREBELLAR ARTERY

PPV – POSITIVE PREDICTIVE VALUE

PWI – PERFUSION WEIGHTED IMAGING

RHD – RHEUMATIC HEART DISEASE

SD – STANDARD DEVIATION

TIA – TRANSIENT ISCHEMIC ATTACK

UMN – UPPER MOTOR NEURON

CONTENTS

INTRODUCTION

Dizziness is one of the most common presenting complaint in patients admitted in emergency department constituting more than 23 lakh admissions (1.3% of the total)[1] and more than $1.6 billion in health care expenditures for dizziness evaluations in the United States each year.[2] No such similar study has been conducted in India.

The term "dizziness" is very nonspecific but may refer to vertigo, light headedness, pre-syncope, anxiety, or just not feeling well. [3-7] Although some doctors consider imbalance to be a more serious symptom of neurologic disease, it is also considered by many authors to be a type of dizziness.[3–7] Some patients have difficulty describing specific type of dizziness and often doctors do not attempt to differentiate among these types.[8] The most common causes of dizziness are usually benign,[3-7] but the differential diagnosis includes potentially life-threatening stroke.

Vertigo is classified as either peripheral (vestibular) or central vertigo etiologically. Peripheral vertigo is commonly benign, whereas central vertigo could be related to life-threatening situations such as posterior fossa bleeding and infarction. In the acute setting, cerebellar or brain stem infarction must be a leading concern because hydrocephalus could follow which is a cause of immediate mortality.[9] Infarctions which are undiagnosed or undetected in

Emergency Departments (EDs) have a mortality rate of 40%.[10] Dizziness may not be always be accompanied with focal neurological signs in stroke and sometimes dizziness may be an isolated symptom in stroke as suggested by some case studies and small series of studies. [11-20]

Dizziness is evaluated in the emergency department with a quick medical history and brief physical examination. In some clinical situations such as unclear complaints, uncooperative patients, overcrowded Emergency Departments (EDs) especially in a country with high patient turnover like India and the fear of misdiagnosing the patient encourage doctors to utilize neurological screening techniques.

Since the widespread use following the availability and low cost of Cranial Computerized Tomography (CCT), it is widely used to determine posterior fossa bleeding and the central causes of vertigo. [21-22] It is a known fact that Cranial Computerized Tomography is inferior to Magnetic Resonance Imaging (MRI) especially for evaluating posterior fossa infarction and bleeding because of the bony artefacts in Cranial CT. [23-26]

MRI is not reasonable and practical for every patient presenting with dizziness because of the following issues:

1. High Cost
2. Lack of availability at smaller centres and lack of availability of round the clock support even in advanced centres.

3. Not ideal for patients presenting acutely with giddiness because of claustrophobia attached with the longer duration of examination compared to Cranial CT.
4. Impractical because of the huge number of patients presenting with giddiness and lack of sufficient resources.
5. Cannot be used in most cases with metal or stent in the body.

Cardiac arrhythmia is another common cause of dizziness. ECGs can sometimes miss transient arrhythmias. Sensitive tests like cardiac telemetry is not practical in all patients presenting with dizziness.[27]

The idea for this study came when nearly all patients admitted with giddiness or dizziness were evaluated in the emergency department to rule out posterior circulation stroke with CT Brain as the primary modality. The aim was to identify the patients who really need CT Brain by analyzing the different risk factors and clinical presentation to conserve resources and for appropriate management.

OBJECTIVES

1) Incidence of stroke as diagnosed by abnormal CT Brain finding in patients presenting with acute dizziness ≤ 2 weeks in the emergency ward.
2) Study the variation in clinical presentation of acute dizziness in emergency ward.
3) Risk stratification of patient population
    a) Correlation between abnormal CT brain and patient factors like age, sex,
    b) Correlation between abnormal CT brain and factors in history relevant to dizziness.
    c) Correlation between alcoholism and smoking to abnormal CT Brain.
    d) Correlation between modifiable risk factors like systemic hypertension, diabetes mellitus, dyslipidemia with abnormal CT Brain.
4) Understanding the correlation between abnormal CT Brain in patients presenting with dizziness and different examination findings
    a) Vitals like blood pressure and pulse
    b) CNS examination
    c) Other system examination

5) Understanding the correlation between abnormal CT brain and biochemical parameters like
    a) Random blood sugar
    b) Kidney function tests like S. Creatinine and Blood Urea
6) Abnormal CT Brain and relation with abnormal ECG on admission
7) Creating a risk profile for patients presenting to emergency department with dizziness for the indication of CT Brain.
8) Study the various abnormalities detected in CT Brain in patients admitted with acute dizziness.

REVIEW OF LITERATURE

"Dizziness" is a nonspecific term often used by patients to describe symptoms. The most common disorders lumped under this term include vertigo, nonspecific "dizziness," disequilibrium, and presyncope.

The reported proportion of patients with different etiologies of dizziness in community surveys[28], primary care setting[29,30], the emergency department [31-36], and the specialized dizzy clinic [37-41] are similar:

- approximately 40 percent of dizzy patients have peripheral vestibular dysfunction;
- 10 percent have a central brainstem vestibular lesion;
- 15 percent have a psychiatric disorder; and
- 25 percent have other problems, such as presyncope and disequilibrium.
- The diagnosis remains uncertain in approximately 10 percent.

The distribution of causes varies with age of the individuals. The elderly have a higher incidence of central causes of vertigo (approaching 20 percent). The most common cause in them is due to stroke. Careful history taking is critical for classifying the etiology of dizziness.

In one series, the history was most sensitive for identifying vertigo (87 percent), presyncope (74 percent), psychiatric disorders (55 percent), and disequilibrium (33 percent) [29]. The physical examination generally confirmed but did not make the diagnosis. Positional changes in symptoms, orthostatic blood pressure and pulse changes, observation of gait, and detection of nystagmus were most helpful on physical examination [29]. Most psychiatric disorders were not detected prior to standardized psychological testing using the diagnostic interview schedule (DIS).

The spinning quality of vertiginous sensations is notoriously unreliable [42]. Lack of spinning cannot be used to exclude vestibular disease, given the difficulty many patients have in putting their dizzy experience into words. On the other hand, some patients with presyncope from vasovagal or cardiac disease can interpret their sensation of dizziness as a spinning sensation [43].

The presence of additional neurologic signs strongly suggests the presence of a central vestibular lesion. Symptoms such as staggering or ataxic gait, vomiting, headache, double vision, visual loss, slurred speech, numbness of the face or body, weakness, clumsiness, or incoordination should be reviewed with the patient.

A careful neurologic examination should be performed for cranial nerve abnormalities, Horner syndrome, motor or sensory changes, dysmetria, or abnormal reflexes. However, the absence of other neurologic findings does not entirely exclude a central process.

Psychiatric disorders may be the primary cause of nonspecific dizziness in some cases. One-quarter of such individuals had major depression, one-quarter had generalized anxiety or panic disorder, and the remainder had somatization disorder, alcohol dependence, and/or personality disorder in one series [29]. Other series report higher rates of panic disorder [44]. Ill-defined disorders such as fibromyalgia have also been associated with dizziness and vertigo [45]. Patients who have a chief cause of dizziness that is not psychiatric may also have a psychiatric disorder as a contributing factor. Psychotherapy may help manage this type of dizziness. A meta-analysis of three randomized trials that used cognitive behavioural therapy in combination with relaxation techniques or vestibular rehabilitation found that therapy was helpful in managing dizziness in the short term, although not associated anxiety and depression[46].

Dizziness in the older adult deserves specific mention because of its high prevalence, up to 38 percent in some series, and its attendant risk of falls, functional disability, institutionalization, and even death [47,48]. Assessment of dizziness in older patients is challenging because it is frequently attributable to multiple problems, including vertigo, cerebrovascular disease, neck disorders, physical deconditioning, and medications [49]. Visual impairment from cataracts and other conditions is common in older adults and likely exacerbates the disability that is associated with dizziness [38]. One study found that 44 percent

of patients aged 65 to 95 years had more than one condition causing dizziness [50]. Some call this entity multiple-sensory defect dizziness.

In a population-based study of 1087 community living individuals 72 years of age or older, 261 (24 percent) reported having an episode of dizziness during the two months prior to study onset and that the dizziness (whether persistent or intermittent) had been present for at least one month [47].

The investigators found seven characteristics that were independently associated with dizziness on multivariate analysis:

- Anxiety trait
- Depressive symptoms
- Impaired balance
- Past myocardial infarction
- Postural hypotension (mean decrease in blood pressure ≥20 percent)
- Five or more medications
- Impaired hearing

Only 10 percent of study participants with none of these seven characteristics reported dizziness. The prevalence of dizziness in those who had one, two, three, four, and five or more of these characteristics was 18, 27, 33, 50, and 68 percent, respectively. The authors concluded that while dizziness in some older individuals may primarily be due to one problem, a number of older patients likely have a multifactorial etiology.

One study of 417 patients aged 65 to 95 years found that most, 69 percent, of patients had presyncope-type dizziness [50]. Underlying cardiovascular disease was the most common contributing factor in 57 percent, followed by peripheral vestibulopathy (14 percent), and psychiatric conditions (10 percent).

IMAGING IN CEREBROVASCULAR DISEASE

Cerebrovascular disease, with its complex anatomy, and ischemia in different parts of the brain can produce the same neurological deficits. Because important decisions, such as revascularization, are often based on the association between symptoms and stenosis in a specific vascular territory, the clinician must be confident in the localization.

Traditionally, the Oxfordshire Community Stroke Project classification used a simple clinical scheme with a high correspondence to radiological findings to distinguish posterior circulation infarction (PCI) from anterior circulation infarction (ACI).[47-50]

TABLE 1: APPEARANCE OF THE INFARCT ON THE BRAIN SCAN AND THE CLINICAL SYNDROMES CONSIDERED APPROPRIATE TO THAT APPEARANCE

CT or MRI appearance	Clinical syndrome
Large cortical MCA infarct (whole of the cortex supplied by the MCA plus adjacent white matter and part or all of the ipsilateral basal ganglia) or more than half of the MCA territory plus ACA or PCA territory	Total anterior circulation infarction
Medium sized cortical infarct (about half the MCA territory)	Partial or total anterior circulation infarction
Small cortical infarct (less than a quarter of the MCA territory) or any of the ACA territory	Partial anterior circulation infarction
Border zone cortical infarct between ACA and MCA or PCA and MCA territories	Partial anterior circulation infarction
Large (>1.5 cm) subcortical infarct (striatocapsular)	Total or partial anterior circulation infarction
Small (<1.5 cm) subcortical infarct (lacunar) (including centrum semiovale infarcts)	Lacunar infarction
Cortical infarct in PCA territory	Posterior circulation infarction
Brainstem or cerebellar infarct (including small infarcts in the pons)	Posterior circulation infarction

The presence of classic brain stem and cerebellar symptoms was used to diagnose PCI. However, this approach may not accurately localize all ischemic strokes. A substantial proportion of PCIs may not be accurately classified only by symptoms/signs, because they lack typical clinical features. Misdiagnosis usually occurs in the initial phase of patient evaluation, which may lead to erroneous clinical decision-making.

In some studies, PCI simulated involvement of the anterior circulation.[51–54] Recent evidence from magnetic resonance imaging (MRI)-based studies demonstrated that unilateral limb weakness, unilateral limb numbness, nausea/vomiting, and headache are the major clinical neurological deficits in PCI rather than crossed paralysis, crossed sensory deficits, visual field disturbance, isolated vertigo, and dysphagia.[55-57] This suggests that the clinical manifestations of PCI and ACI are more alike than dissimilar.

In a study published in stroke in 2012 [58], Compared with ACI, patients with PCI were more often men, had a lower mean baseline National Institutes of Health Stroke Scale score, and a higher mean Glasgow Coma Scale. PCI patients had a greater frequency of diabetes (20.5% versus 13.8%; P0.005) and smoking (39.7% versus 31.5%; P0.009), but a lower frequency of atrial fibrillation (3.0% versus 8.7%; P0.001) and valvular heart disease (2.0% versus 6.4%; P0.003).

The neurological deficits with the highest predictive values favouring the diagnosis of PCI were in the less common subgroup, including Horner's syndrome (PPV100.0%; OR4.00), crossed sensory deficits (PPV100.0%; OR3.98), quadrantanopia (PPV100.0%; OR3.93), oculomotor nerve palsy (PPV100.0%; OR4.00), and crossed motor deficits (PPV 92.3%; OR36.04). However, all of them occurred with an extremely low sensitivity ranging from 1.3% to 4.0% of PCI patients, which means that the probability of detecting neurological deficits in patients with PCI was very low. [58]

Neurological deficits with a relatively good predictive value such as ataxia (PPV66.9%; OR8.06), nausea/vomiting (PPV52.8%; OR4.38), nystagmus (PPV83.7%; OR16.72), vertigo (PPV79.2%; OR13.29), and diplopia (PPV78.6%; OR11.34), also displayed low sensitivities that decreased their diagnostic value. [58]

The cause of dizziness is misdiagnosed in many patients and the physicians are eager for diagnostic guidelines. [59,60]

COMPUTED TOMOGRAPHY

The main advantages of CT are widespread access and speed of acquisition. In the hyperacute phase, a noncontrast CT (NCCT) scan is usually ordered to exclude or confirm hemorrhage; it is highly sensitive for this indication. A NCCT scan should be obtained as soon as the patient is medically stable. The presence of hemorrhage leads to very different

management and concerns than a normal scan or one that shows infarction. Immediate CT scanning of all patients with suspected stroke is also the most cost-effective strategy when compared with alternate strategies such as scanning selected patients or delayed rather than immediate imaging [61].

The utility of CT for acute stroke has been enhanced by the advent of additional CT techniques including CT perfusion imaging (CTP) and CT angiography (CTA). Multimodal CT evaluation that employs the three techniques (NCCT, CTA, and CTP) combined shows improved detection of acute infarction when compared with NCCT evaluation alone [62-65]. In addition, multimodal evaluation that includes CTA and CTP may permit assessment of the site of vascular occlusion, infarct core, salvageable brain tissue and degree of collateral circulation [66,67].

EARLY SIGNS OF INFARCTION ON NON-CONTRAST CT

The sensitivity of standard non-contrast CT for brain ischemia increases after 24 hours. However, in a systematic review involving 15 studies where CT scans were performed within six hours of stroke onset, the prevalence of early CT signs of brain infarction was 61 percent (standard deviation +/- 21 percent) [68].

Early signs of infarction include the following [68-72]:

●Hypoattenuation involving one-third or more of the middle cerebral artery (MCA) territory

- Obscuration of the lentiform nucleus
- Cortical sulcal effacement
- Focal parenchymal hypoattenuation
- Loss of the insular ribbon or obscuration of the Sylvian fissure
- Hyperattenuation of large vessel (eg, "hyperdense MCA sign")
- Loss of gray-white matter differentiation in the basal ganglia

The presence of early CT signs of infarction implies a worse prognosis. In the systematic review, the presence of these signs was associated with an increased risk of poor functional outcome (odds ratio 3.11, 95% CI 2.77-3.49) [68].

While early CT signs of infarction are associated with a worse outcome, it remains unclear whether early infarction signs should be considered when deciding whether to use intravenous (IV) thrombolytic treatment for acute ischemic stroke [68].

Studies that have examined the ability of neurologists, neuroradiologists, and general practitioners have found that early infarction can be very difficult to recognize on CT[73]. However, the importance of a truly normal head CT in acute stroke should not be underestimated; it excludes major ischemic damage with high specificity [74].

Standardized methods such as ASPECTS have been developed to aid recognition of early ischemia because of the known difficulty in detecting such changes. In addition, accentuating the contrast between normal and edematous (ischemic) brain tissue by variable window width and center level settings may improve detection of early ischemic change on noncontrast CT [75].

UTILITY OF CT CONTRAST DYE

Spiral (helical) CT and new generation multidetector CT scanners increase scan speed and allow CTA of both extracranial and intracranial cerebral arteries. The speed of these CT units also offers CTP capabilities. These scans can be performed immediately after conventional CT scanning, requiring only 5 to 10 minutes of additional time. In practice, one can perform both CTA and CTP during the same examination, with separate contrast boluses [76].

CT ANGIOGRAPHY

CTA is performed by administering a rapid bolus of standard intravenous CT contrast through a large bore intravenous line in the antecubital fossa. The helical CT scan is timed to capture the arrival of dye into the brain. Dye can be seen in the great vessels on the raw CT images. Clot causes a filling defect in the vessel on CTA, which often can be seen on the raw images.

Advantages of these fast CT scans include the ability to rapidly identify patients with occlusion of the major vessels within the circle of Willis or extracranial cerebral arteries, as well as the ability to evaluate the perfusion status of the brain parenchyma.

Additional information about brain perfusion can be obtained by post imaging analysis of the raw data (or source images) of CTA and CTP studies.

CTA SOURCE IMAGES

CTA source images can provide an estimate of perfusion by taking advantage of the contrast enhancement in the brain vasculature that occurs during a CTA [77], potentially obviating the need for a separate CT perfusion study and a second contrast bolus. CTA source images typically cover the entire brain, in contrast to CT perfusion source images that are limited to a few brain slices.

During a CTA, contrast dye fills the brain microvasculature in the normal perfused tissue that is accessible to the blood pool and appears as increased signal intensity on the CTA source images. In distinction, contrast dye does not fill the microvasculature in ischemic brain regions that are less accessible to the blood pool and have poor collateral flow. These ischemic areas are easily seen as regions of hypoattenuation (low density or dark) on CTA source images [78,79].

CTA source images are more sensitive than noncontrast CT scans for the detection of early brain infarction [80,81]. Hypoattenuation on CTA source images correlates with ischemic edema [79], and with the abnormality on diffusion-weighted MRI [82]. In this sense, CTA source images (or raw images of CT perfusion studies) can be considered as a surrogate for DWI.

CT PERFUSION IMAGING

Using an intravenous bolus of CT dye, a whole brain "perfused blood volume map" can be obtained by timing the scan to the passage of the contrast dye through the brain [83]. This can be obtained by continuing to scan the brain during a CT angiogram or by using a new bolus of contrast following the CTA. However, CTP requires repeatedly scanning the same portion of the brain parenchyma over the time required for the bolus to pass through the vasculature.

Similar to CTA-SI, the source images of the CTP (CTP-SI) are available for analysis. As with CTA-SI, areas of hypoattenuation on CTP-SI should correlate with ischemic brain regions.

MAGNETIC RESONANCE IMAGING

Advanced MRI imaging techniques have the potential for further defining stroke subgroup populations that may benefit from intravenous thrombolysis or interventional vascular treatments [84]. In addition, MRI

sequences using high susceptibility methods, such as gradient echo (GRE) pulse sequences, are equivalent to CT for the detection of acute intracerebral hemorrhage (ICH) and better than CT for the detection of chronic hemorrhage [85-87]. ICH can be diagnosed by MRI with up to 100 percent sensitivity and accuracy by experienced readers [86].

Brain MRI protocols that combine conventional T1 and T2 sequences with diffusion-weighted imaging (DWI), perfusion-weighted imaging (PWI), and GRE can reliably diagnose both acute ischemic stroke and acute hemorrhagic stroke in emergency settings. These MRI techniques may obviate the need for emergent CT in centers where brain MRI is readily available.

Newer ultrafast MRI imaging protocols can reduce acquisition times from the 15 to 20 minutes required by conventional MRI to five minutes or less.

DIFFUSION-WEIGHTED IMAGING

DWI is based upon the capacity of fast MRI to detect a signal related to the movement of water molecules between two closely spaced radiofrequency pulses. This technique can detect abnormalities due to ischemia within 3 to 30 minutes of onset, [88-90] when conventional MRI and CT images would still appear normal.

In a study comparing CT, DWI, and standard MRI, abnormal DWI was a sensitive and specific indicator of ischemic stroke in patients presenting within six hours of symptom onset [91].

CLINICAL UTILITY OF DWI

A systematic review published in 2010 from the American Academy of Neurology (AAN) concluded that DWI is superior to noncontrast CT for the diagnosis of acute ischemic stroke in patients presenting within 12 hours of symptom onset [92].

In the evaluation of acute ischemic stroke or TIA, the presence of multiple DWI lesions on the baseline MRI scan is associated with an increased risk of early lesion recurrence [93-95].

Furthermore, the presence of multiple DWI lesions of varying ages, as determined by the ADC value, is an independent predictor of future ischemic events [96].

PERFUSION-WEIGHTED IMAGING

Diffusion-weighted imaging reveals evidence of ischemic injury, not ischemia itself. In contrast, perfusion-weighted imaging (PWI) uses fast MRI techniques to quantify the amount of MR contrast agent reaching the brain tissue after a fast intravenous bolus. Integration of the amount of gadolinium entering the brain on first pass allows construction of maps of cerebral blood

volume. Analysis that also includes the time course of arrival and washout permits the construction of maps of relative cerebral blood flow and mean transit time. The latter sensitively identifies the ischemic zone.

PWI can be performed with standard MRI and MR angiography, requiring a total imaging time of less than 15 minutes.

POSTERIOR CIRCULATION CEREBROVASCULAR SYNDROMES

●About one-third of posterior circulation strokes are caused by occlusive disease of the vertebral arteries in the neck and the intracranial vertebral, basilar, and posterior cerebral arteries [97-100].

●The proximal portion of the vertebral artery in the neck is the most common location of atherosclerotic occlusive disease within the posterior circulation [97-101].

●Dissection of the extracranial and intracranial vertebral arteries is another common cause of ischemia within the posterior circulation.

● Most infarcts in the posterior cerebral artery territory are due to embolism from the heart, aorta, or vertebral arteries.

SUBCLAVIAN ARTERY

In the subclavian steal syndrome, obstruction of the proximal subclavian artery produces symptoms related to the ipsilateral arm and hand. Coolness, weakness, and pain on use of the arm are common. Dizziness is by far the most common neurologic symptom of the subclavian steal syndrome.

EXTRACRANIAL VERTEBRAL ARTERIES

Among a series of 100 patients with angiographically documented vertebral artery lesions, 92 percent were atherosclerotic in origin [102].

The most common location of atherosclerotic occlusive disease within the posterior circulation is the proximal portion of the vertebral artery in the neck [97-101].

Another common cause of posterior circulation stroke is arterial dissection.

Dizziness is the most common and often the only neurologic symptom. Sometimes it is accompanied by other signs of hindbrain ischemia. Diplopia, oscillopsia, weakness of both legs, hemiparesis, and numbness.

ARTERY TO ARTERY EMBOLISM AND LOW FLOW

Embolization of white platelet-fibrin and red erythrocyte-fibrin thrombi from atherostenotic occlusive lesions is the most common presentation of ECVA origin disease [97-101,103]. The intraarterial emboli travel from the ECVA origin to reach the ipsilateral intracranial vertebral artery (ICVA), and sometimes travel on to block the rostral basilar artery and/or its branches. In support of this observation, patients presenting with ischemia in the distribution of the ICVA (the medulla and posterior inferior cerebellum) or the distal basilar artery (superior cerebellum, occipital and temporal lobes in the territory of the posterior cerebral arteries, or the thalamus or midbrain) show a high frequency of recent ECVA occlusions [97,98-101].

In patients with proximal ECVA stenosis, intraarterial (artery to artery) embolism is a much more frequent cause of ischemia to the intracranial posterior circulation arteries than hemodynamic insufficiency (ie, low flow). This is illustrated by results from the New England Medical Center Posterior Circulation Registry, which evaluated a series of 407 patients who had

posterior circulation TIAs or strokes within the prior six months and included 80 patients with severe stenosis or occlusion of the proximal ECVA [97].

DISSECTION AND OTHER CAUSES

Dissection of the ECVA usually involves the distal portion of the ECVA as it winds around the upper cervical vertebrae [104]. Sometimes dissections involve the proximal ECVA between the origin of the artery and its entry into the vertebral column, usually at C5 or C6. Pain in the neck and/or occiput and TIAs or strokes involving the lateral medulla and cerebellum are the most common findings.

Ischemic symptoms due to ECVA dissection are most often vestibulocerebellar and include dizziness, vertigo, veering to one side, and loss of balance.

Rotational vertebral artery occlusion is an uncommon cause of transient posterior circulation ischemic symptoms, mainly paroxysmal vertigo or nonspecific dizziness, which may be accompanied by nystagmus, tinnitus, syncope, blurred vision, nausea, or vomiting [105,106]. The nystagmus typically has a prominent downbeat component, but may also include torsional and horizontal components [107]. The symptoms are due to dynamic compression of one (dominant) vertebral artery by bony elements of the cervical spine, triggered by head turning to one side, or less often by head turning to both sides or head tilting [105,106]. The symptoms are relieved by returning the head to the neutral position.

INTRACRANIAL VERTEBRAL ARTERIES

Atherostenotic disease can involve any portion of the intracranial vertebral arteries (ICVA). The most common location of ICVA stenosis is the distal portion of the artery at or near the vertebral-basilar artery junction. Dissection of the ICVA also occurs, and ischemic symptoms are usually accompanied by prominent headache [108].

Occlusive ICVA disease presents in a variety of different ways [100,109,110]:

- Asymptomatic occlusion
- Transient ischemic attacks (TIAs)
- Lateral medullary infarcts
- Medial medullary infarction
- Infarction of one-half of the medulla
- Cerebellar infarction in PICA territory
- Embolization of the ICVA thrombus to the distal basilar artery
- Propagation of the ICVA thrombus into the basilar artery

LATERAL MEDULLARY INFARCTION

Lateral medullary infarction (Wallenberg syndrome) is the most common and important syndrome related to intracranial vertebral artery occlusion.

VESTIBULOCEREBELLAR SYMPTOMS AND SIGNS

Vestibulocerebellar symptoms and signs are nearly always present in patients with lateral medullary infarcts [109]. These are related to involvement of the vestibular nuclei and their connections, and to involvement of the inferior cerebellar peduncle (restiform body).

Common symptoms and signs are as follows:

- Feeling dizzy or off-balance, which may take a number of forms:
- Turning, rotating, whirling, or moving in relation to the environment
- Being pulled or falling towards one side, most often ipsilateral to the lesion
- Swaying or rolling as if moving from side to side
- Tilting or leaning
- Difficulty sitting upright without support.
- Hypotonia of the ipsilateral arm.
- Blurred vision or diplopia.
- Nystagmus

- Ocular torsion. The eye and ear ipsilateral to the lateral medullary infarct may rest in a down position below the contralateral eye and ear [111].
- Limb ataxia ipsilateral to the lateral medullary infarct.

SENSORY SYMPTOMS AND SIGNS

- Pain or unpleasant feelings in the face are sometimes the earliest and most prominent feature of the lateral medullary syndrome and are diagnostic of a lateral tegmental brainstem localization.
- Loss of pain and temperature sensation in the contralateral trunk and limbs is related to lesions of the lateral medullary spinothalamic tract.
- The most common pattern of sensory abnormality with lateral medullary infarcts is loss of pain and temperature sensation in the ipsilateral face and the contralateral trunk and limbs.
- Examination usually shows ipsilateral decreased pain and temperature sensation in the face.

BULBAR MUSCLE WEAKNESS

Weakness of bulbar muscles innervated by the lower cranial nerves is a very prominent feature when lateral medullary infarcts extend medially.

RESPIRATORY DYSFUNCTION

Respiratory dysfunction is an important feature of lateral medullary ischemia.

The most common abnormality described in patients with lateral tegmental caudal brainstem lesions is failure of automatic respirations, a phenomenon especially apparent during sleep. This failure to initiate respiration has been referred to as Ondine's curse.

AUTONOMIC DYSFUNCTION

- The ipsilateral eye often shows features of Horner's syndrome due to lesions of the descending sympathetic nervous system.
- Cardiovascular abnormalities include tachycardia, orthostatic hypotension without cardiac rate acceleration, and intermittent bradycardia [100].

MEDIAL MEDULLARY INFARCTION

The most consistent finding in patients with medial medullary ischemia is a contralateral hemiparesis [100,112].

In approximately one-half of patients, the face is also involved. Sensory symptoms are related to ischemia of the medial lemniscus. Some patients report paresthesias or, less commonly, dysesthesias in the contralateral lower limb and trunk.

Ipsilateral tongue paralysis is the least common but most topographically localizing sign of medial medullary infarction, and is due to involvement of the hypoglossal nucleus. Tongue paresis causes slurring of speech, especially of lingual consonants.

HEMIMEDULLARY INFARCTION

Occasional patients have infarction that involves both the lateral and medial medullary territories on one side (hemimedullary infarcts). Symptoms are identical to those found in patients with lateral medullary ischemia with the addition of a hemiparesis contralateral to the lesion. The hemiparesis may develop concurrently with lateral medullary symptoms and signs or can occur later.

CEREBELLAR INFARCTION

Cerebellar infarction in PICA distribution can involve just the vermis, or the lateral surface, or the full PICA territory. Full PICA territory infarcts are often accompanied by edema formation and mass effect (so-called pseudotumoral cerebellar infarcts).

Approximately one-fifth of PICA territory cerebellar infarcts are accompanied by infarction in the dorsal or dorsolateral medulla [99,100]. The combination of lateral medullary and PICA cerebellar infarction occurs when the ICVA is occluded and blocks the orifice of both PICA and the lateral medullary penetrators.

Infarcts limited to the medial vermis in medial PICA territory usually cause a vertiginous labyrinthian syndrome that closely mimics a peripheral vestibulopathy. Severe vertigo and prominent nystagmus are the major findings.

Lateral cerebellar hemisphere PICA territory infarcts are usually characterized by minor degrees of dizziness and gait incoordination with veering to the side of the lesion. Minor limb hypotonia and incoordination are found. A common syndrome is acute unsteadiness with ataxia but without vertigo or dysarthria. Body sway towards the side of the lesion, ipsilateral limb ataxia, and abnormal rapid alternating movements are also common.

When the full PICA cerebellar territory is involved, headache is usually present in the occiput or high neck on the ipsilateral side. The head may also be tilted with the occiput tending to tilt toward the ipsilateral side.

Vomiting, gait ataxia, truncal lateropulsion, and limb incoordination are other common findings. The truncal dysfunction is similar to that found in the lateral medullary syndrome; the body is often tilted or pulled ipsilaterally upon sitting or standing. The limb incoordination consists mostly of hypotonia rather than a rhythmic intention tremor.

PSEUDOTUMORAL CEREBELLAR INFARCTION

The syndrome of pseudotumoral cerebellar infarction, with edema formation and mass effect, is most often associated with large full PICA territory infarcts. After the first day or so, patients with this form of cerebellar infarction typically develop increased headache, vomiting, and decreased consciousness, with drowsiness followed by stupor. Bilateral Babinski signs are an early sign of cerebellar mass effect.

Characteristic oculomotor abnormalities of large cerebellar space-taking infarcts can develop and include the following features:

- Most common are a conjugate gaze paresis to the side of the lesion or a paresis of abduction limited to the ipsilateral eye
- Bilateral sixth nerve paresis may occur

- Later bilateral horizontal gaze palsies may develop, often accompanied by ocular bobbing

These signs are due to compression of the pontine tegmentum by the swollen cerebellar infarct. Stupor is followed by deep coma when the oculomotor abnormalities become bilateral.

DOLICHOECTASIA

Dolichoectasia (dilatative arteriopathy) is a term that describes arterial elongation, widening, and tortuosity [113-118]. The intracranial vertebral and basilar arteries are most often affected. The most important clinical presentations of dilatative arteriopathy are as follows:

●Acute brain ischemia

●A progressive course related to compression of cranial nerves, the brainstem, or the third ventricle

●A catastrophic outcome caused by vascular rupture

Flow in dilated arteries may become to-and-fro, causing reduced antegrade flow and thrombus formation. Elongation and angulation of arteries can stretch and distort the orifices of arterial branches leading to decreased blood flow, especially in penetrating branches. Dilated intracranial vertebral arteries can compress the medulla leading to the gradual onset of hemiparesis [116].

DISTINGUISHING VERTIGO OF BRAINSTEM AND CEREBELLAR ISCHEMIA FROM PERIPHERAL CAUSES

A composite three-part test entitled HINTS (Head-Impulse-Nystagmus-Test of Skew) is useful for distinguishing brainstem and cerebellar ischemia from vestibular neuritis or other peripheral causes of vertigo [119-122]. The test is most helpful in patients who have had continuous feelings of vertigo or dizziness. It is not useful in patients with momentary position-related transient vertigo (often benign positional vertigo) or those with TIAs who are not dizzy when examined.

HEAD IMPULSE

For the head impulse test, also called head thrust test, the patient is instructed to fix their gaze on a distant target while wearing his or her usual prescription eyeglasses. The head is then turned quickly and unpredictably by the examiner, about 15º; the starting position should be about 10º from straight ahead. The normal response is that the eyes remain on the target .The abnormal response is that the eyes are dragged off of the target by the head turn (in one direction), followed by a saccade back to the target after the head turn; this response indicates a deficient vestibulo-ocular on the side of the head turn, implying a peripheral vestibular lesion (ie, the inner ear or

vestibular nerve) on that side. This reflex is preserved in central lesions, except when cranial nerve (CN) VIII fascicles are affected in the lateral pons.

NYSTAGMUS

Peripheral vestibular lesions often are accompanied by nystagmus that is always in the same direction, while brainstem and cerebellar lesions are typically associated with nystagmus that changes direction with different positions of gaze.

TEST FOR SKEW

The test involves covering one eye and seeing if there is a vertical shift in the eye when uncovered. Brainstem and cerebellar lesions sometimes cause a slight skew deviation.

Any of the following, whether present or untestable, suggest a brainstem or cerebellar lesion [119-122]:

●Normal head impulse test on both sides

●Direction-changing nystagmus

●Skew deviation

The presence of all of the following suggests a peripheral lesion :

●An abnormal head impulse test on one side

●Unidirectional, horizontal, torsional nystagmus that increases in intensity with gaze toward the fast phase

●Absent skew

The importance of these oculomotor tests is that brain imaging with either CT or MRI may be normal during the acute phase of ischemic symptoms. In this regard, the HINTS test appears to be more sensitive for the diagnosis of acute stroke than even brain MRI within the first two days after symptom onset [122].

One caveat is that there are rare examples of inner ear infarction (usually due to occlusion of the internal auditory artery, an anterior inferior cerebellar artery branch) causing an acute vestibular syndrome. These are typically associated with new hearing loss on the side of the vestibular lesion, which may be the only clue that the mechanism is stroke [121,122]. In such cases, the eye movements elicited on HINTS are indistinguishable from peripheral vestibular causes.

METHODOLOGY

1) This study is a prospective, observational study of patients admitted in our hospital with acute dizziness in the emergency ward.
2) The study was done in the emergency ward of Thanjavur Medical College which is manned by residents and professors from the department of Internal Medicine. There is no separate emergency medicine program here.
3) CT scanning is available 24 hours a day for emergency cases.
4) 43 patients admitted in emergency ward with symptoms of acute dizziness, vertigo and imbalance were included.
5) The period of study was for 6 months (March 2017 to August 2017)
6) Informed Consent of the patient was taken after approval from the ethical committee of the institute.
7) Clinical variables were identified based on the past literature and clinical experience of the investigators.
8) All patients in the study underwent the following:
    a) Detailed history of
        i) dizziness, giddiness and imbalance – dizziness is very nonspecific and can refer to vertigo, lightheadedness, imbalance, presyncope, anxiety or just not feeling well. We didn't try to differentiate the types and all were included under the same. The duration of the same was noted.

ii) history of loss of consciousness – present or not present was considered irrelevant of the duration.

iii) history of fall – present or absent was considered irrelevant of the number of falls.

iv) history of seizure – present or absent was considered irrelevant of the number of seizures, focal or generalized.

v) history of headache, vomiting, nausea – presence or absence of headache, vomiting or nausea was considered irrelevant of the duration or severity.

vi) history of altered mental status – history of altered behavior as explained by the attender was considered as presence. Irrelevant of the duration.

vii) history suggestive of motor deficit – history of weakness of a limb or both limbs or facial deviation was considered in motor deficit as presence or absence.

viii) history suggestive of sensory deficit – history of hemi sensory loss was considered as present. Irrelevant of the duration. Glove and stock sensation, pins and prick sensation were not considered.

ix) history of speech disturbance – history of speech disturbance in the form of aphasia or dysphasia or dysarthria were included as present. Duration was not considered.

x) history of visual changes – history of visual changes including double vision, sudden blurring of vision and sudden vision loss were considered. Duration >2 weeks was excluded.

xi) history of tinnitus/ear ache/discharge – history of ringing sensation in the ear, ear ache and discharge of acute onset ≤ 2 weeks was considered as present or absent.

xii) history of palpitation, chest discomfort – history of palpitation, chest discomfort in the form of chest pain, heavy sensation on the chest was included. Duration ≤ 2 weeks was included and considered as present or absent.

xiii) history of neck pain – absence or presence was included. Duration was not considered.

xiv) Past history of hypertension, diabetes, coronary artery disease, dyslipidemia – Past history of hypertension, diabetes, coronary artery disease and dyslipidemia as given by history or included in the old investigation reports or discharge summaries were considered as present or absent. Duration was not noted.

xv) History of previous TIA – History of previous TIA in the form of hemiparesis or facial deviation with or without aphasia recovered within a day was asked for and considered as present or absent.

xvi) Personal habits of smoker/alcohol consumption – Current smokers were considered. Never smoked and Former smokers were not

considered as smokers. Heavy alcohol consumption with weekly consumption of more than 300 units of alcohol was considered as alcoholic risk factor present. One unit is considered as equivalent to standard measure of spirit or one standard glass of wine or half pint of beer. On average, a standard unit contains 8.5g of alcohol.

xvii) History of RHD/atrial fibrillation – History of RHD or Atrial fibrillation as mentioned in previous medical records were considered as present. Irrelevant of the duration.

xviii) History of use of anticoagulants – History of use of anticoagulants for any indication was considered as present. Irrelevant of the duration.

(b) Detailed examination was done which included

a. Vitals monitoring of Blood Pressure, Pulse Rate – Blood pressure measured in right upper limb in supine position and measured in mmHg within an hour of admission in the emergency ward was taken. Pulse rate was measured per minute in right radial artery within an hour of admission in the emergency ward.

b. Detailed CNS examination with focus on cerebellar signs – detailed CNS examination included Higher mental functions, cranial nerves, motor system, sensory system, cerebellar system including nystagmus, gait ataxia. CNS examination was considered abnormal if anything was found abnormal in any of the subsystems.

Subsystems of higher mental functions, cranial nerves, motor system, sensory, cerebellum were also considered and were noted as abnormal or normal. Examination was done in the emergency ward within an hour of admission. Detailed examination was not done. UMN signs refer to pyramidal tract signs in the study.

c. Examination of other systems – Screening examination of other systems – cardiovascular, respiratory, gastrointestinal were done and noted as normal or abnormal in the emergency ward.

(c) Investigations done included:

a. Hemoglobin from venous blood collected at the emergency ward within one hour of admission.

b. Blood Urea/Serum Creatinine from venous blood collected at the emergency ward within one hour of admission.

c. Admission Blood glucose from venous blood collected at the emergency ward within one hour of admission.

d. 12 lead Electrocardiography was done in the emergency ward within one hour of admission. ECG was considered abnormal in the presence of Ischemic changes (ST-T changes), Bradycardia and complex arrhythmias.

e. CT Brain – Non-Contrast CT Brain was taken within 12 hours of admission in the Emergency Ward. Ischemic changes including acute or subacute infarct, Hemorrhagic changes including acute

intracerebral hemorrhage, cerebellar hemorrhage and subarachnoid hemorrhage were considered as abnormal CT Brain. Traumatic causes of subdural hemorrhage, extradural hemorrhage was not considered. Normal Non-Contrast CT Brain was reported as normal.

9) Inclusion Criteria:
   a) Patients presenting in emergency ward with symptoms of acute dizziness, vertigo and imbalance ≤ 2 weeks.
   b) Patients initially diagnosed in the emergency ward as "to rule out posterior circulation stroke" or "Posterior circulation stroke"
   c) Patients with age more than 25 years.
   d) Both males and females.

10) Exclusion Criteria
   a) Patients below age of 25 years
   b) Patients with history of stroke
   c) Patients with history of seizure disorder
   d) Patients with diagnosed cervical spondylosis
   e) Pregnancy
   f) Patients who underwent diagnostic imaging – CT or MRI at some other center and referred here
   g) Patients with history of recent head trauma ≤ 2 weeks.

DATA MANAGEMENT AND ANALYSIS

Data was entered into Microsoft Excel. Statistical Analysis was done using software GraphPad Prism version 5 and SPSS v24. Numerical values were reported using mean and standard deviation or median. Categorical values are reported using number and percentages. Probability value (p) value less than 0.05 was considered a statistically significant.

RESULTS

1. AGE

Table 2: Comparison of Age in years between the patients with normal CT and abnormal CT

S. No	Parameter	CT Normal Group (N=28)		CT Abnormal Group (N=15)		P Value	Statistical Test
		Mean	SD	Mean	SD		
1	Age in years	55.1	13.9	61.6	11.5	0.12 (NS)	Unpaired 't' test

Data are expressed as mean with standard deviation. * indicates $p<0.05$ and considered statistically significant.

Table 3: Age Distribution of patients with normal and abnormal CT

Parameter		Abnormal CT group (n=15)		Normal CT group (n=28)		P value
		n	%	n	%	
Age range (years)	<40	0	0	5	100	0.2 (NS)
	40-60	7	38.9	11	61.1	
	>60	8	40	12	60	

Data are expressed as absolute numbers with proportions. * indicates $p<0.05$ and considered statistically significant.

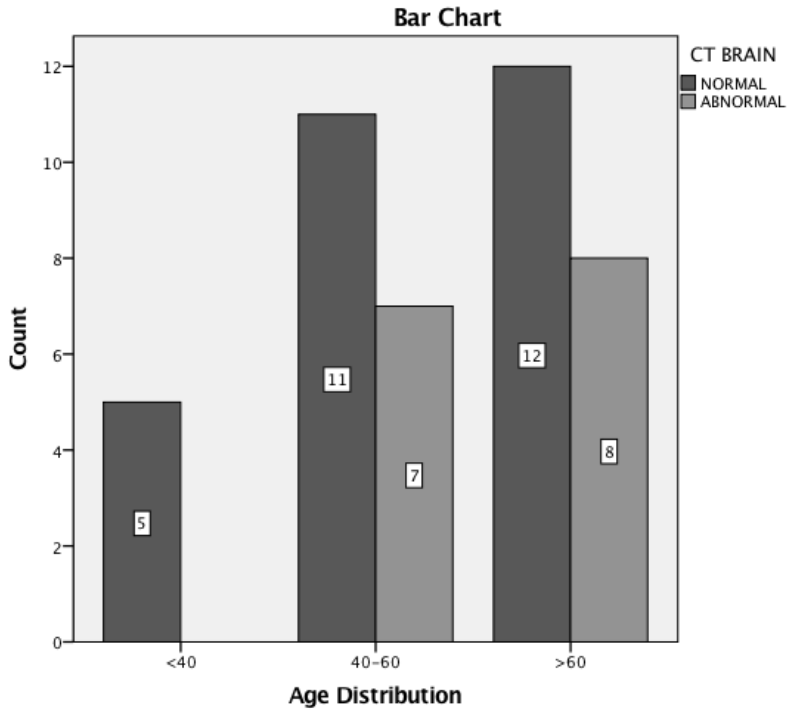


Figure 1: Bar chart representing the age distribution of the study population in the x axis against the number of patients with normal or abnormal CT Brain in the y axis.

2. SEX DISTRIBUTION

Table 4: Comparison of proportions of gender between the patients with normal and abnormal CT findings.

Parameter		**Abnormal CT group (n=15)**		**Normal CT group (n=28)**		**P value**
		n	**%**	**n**	**%**	
Sex	Male	8	53.3	12	42.9	0.54 (NS)
	Female	7	46.7	16	57.1	

Data are expressed as absolute numbers with proportions. Fisher's exact test was used to test the difference between the proportions. * indicates $p<0.05$ and considered statistically significant.

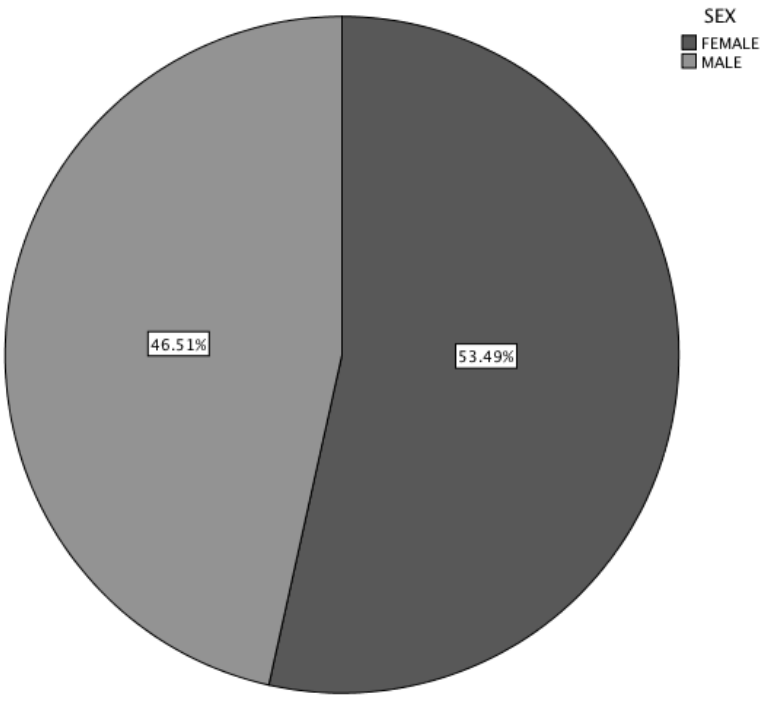


Figure 2: Pie Chart showing distribution of Sex in the study population.

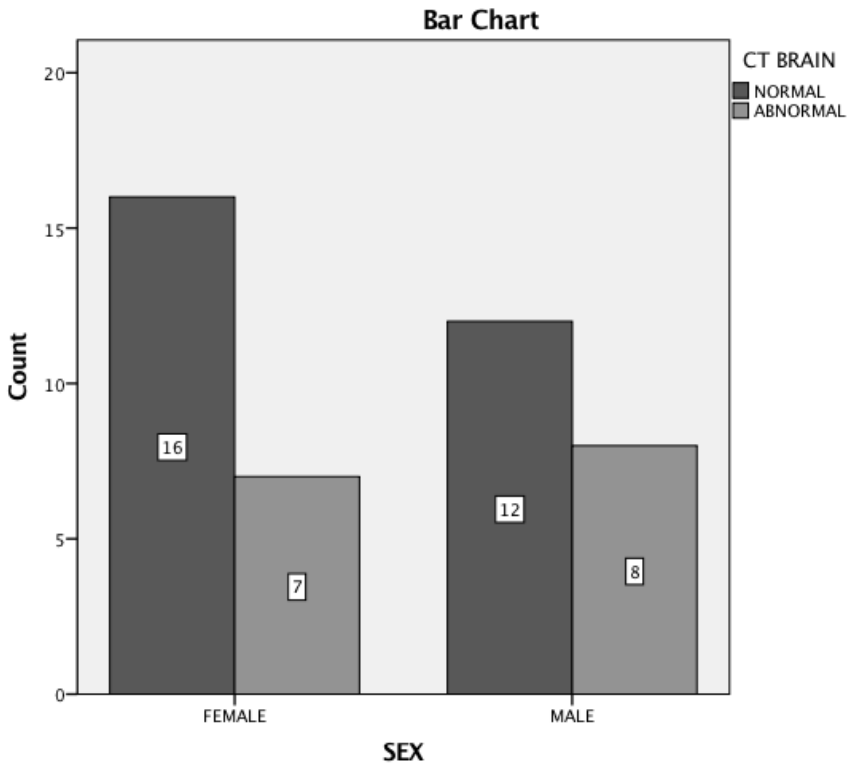


Figure 3: Bar chart representing the gender of the study population in the x axis against the number of patients with normal or abnormal CT Brain in the y axis.

3. DURATION OF DIZZINESS

Table 5: Comparison of duration of dizziness between the patients with normal CT and abnormal CT

Parameter	**CT Normal group (n=28)**		**CT abnormal group (n=15)**		**P value**	**Statistical test**
	Mean	**SD**	**Mean**	**SD**		
Duration of dizziness (in days)	3.02	3.80	0.97	0.35	0.009 *	Unpaired 't' test

Data are expressed as mean with standard deviation. * indicates $p<0.05$ and considered statistically significant.

Table 6: Dizziness Duration

Parameter		**Abnormal CT group (n=15)**		**Normal CT group (n=28)**		**P value**
		n	**%**	**n**	**%**	
Duration of Dizziness	<1 Day	14	46.7	16	53.3	0.042*
	1-3 Days	1	14.3	6	85.7	
	>3 Days	0	0	6	100	

Data are expressed as absolute numbers with proportions. * indicates $p<0.05$ and considered statistically significant.

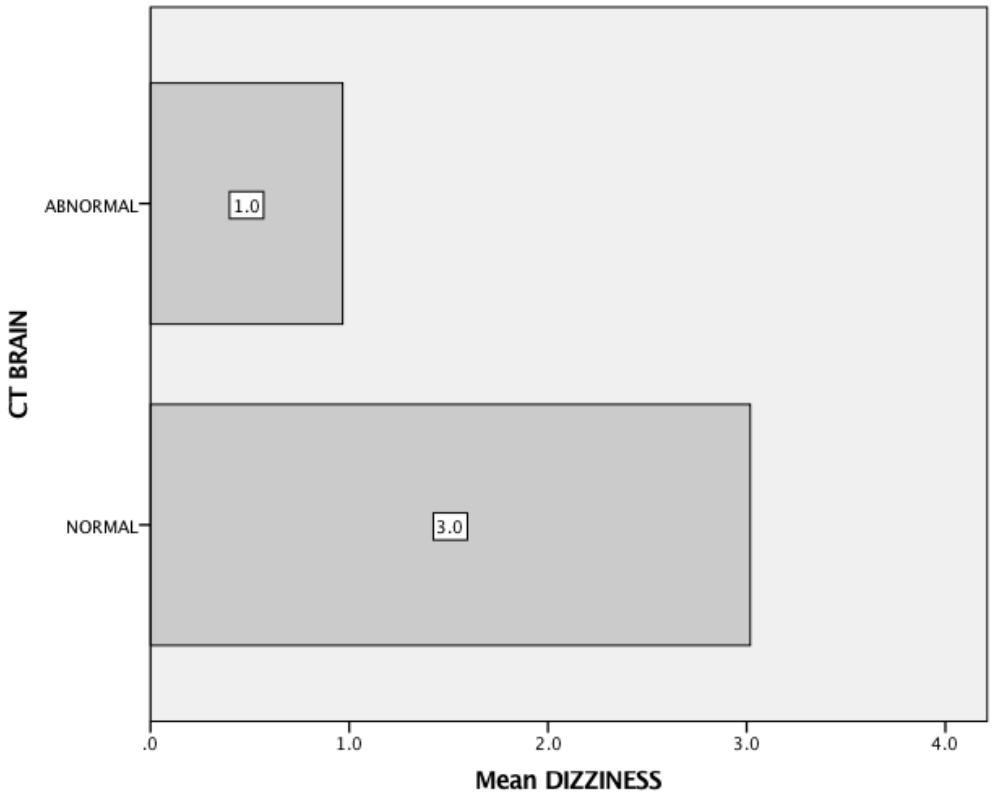


Figure 4: Bar chart showing the mean of dizziness duration in the study population in the X axis against the patients with normal and abnormal CT brain in the Y axis.

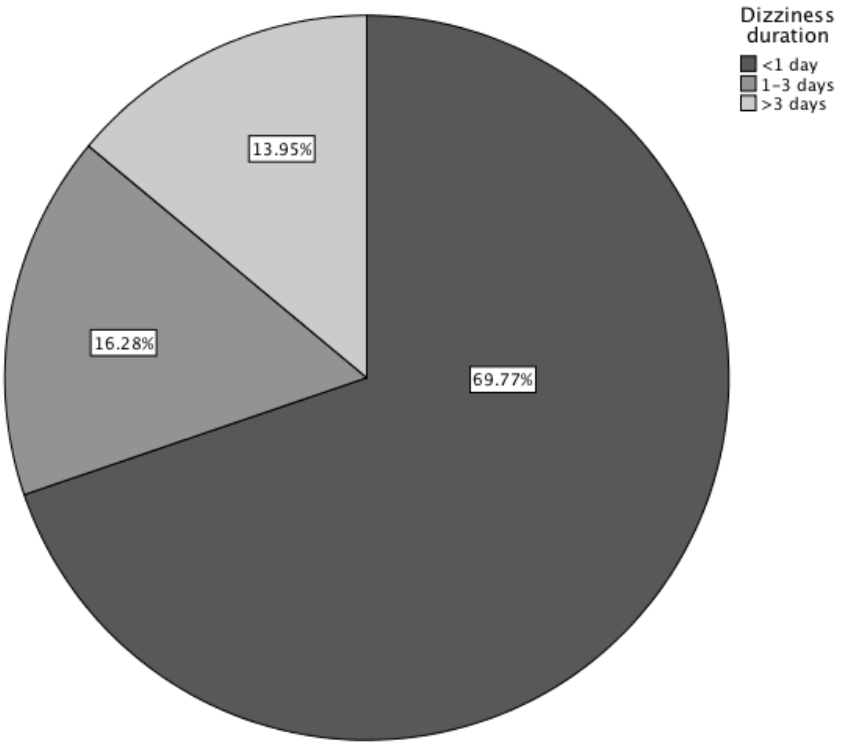


Figure 5: Pie Chart showing different duration of giddiness in the study population.

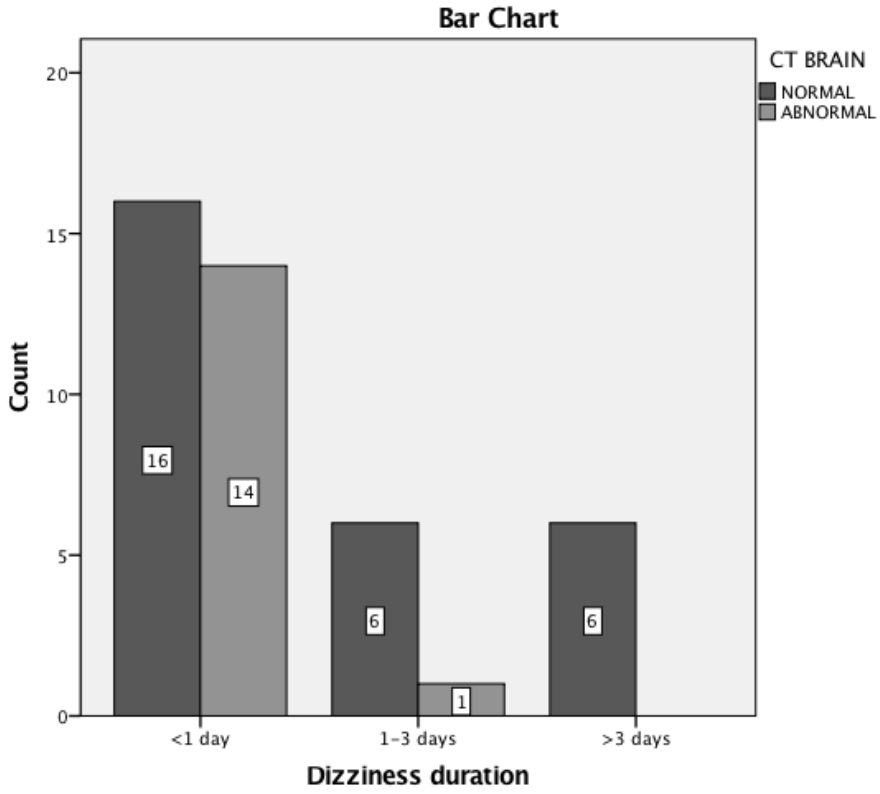


Figure 6: Bar chart representing the duration of giddiness of the study population in the x axis against the number of patients with normal or abnormal CT Brain in the y axis.

4. OTHER HISTORIES

Table 7: Comparison of proportions of various histories between the patients with normal and abnormal CT findings.

S. No	Parameter		Abnormal CT group (n=15)		Normal CT group (n=28)		P value	Odds ratio (95% CI)
			n	%	n	%		
1	History of LOC	Present	4	26.7	5	17.9	0.69 (NS)	---
		Absent	11	73.3	23	82.1		
2	History of fall	Present	0	0	2	7.1	0.53 (NS)	---
		Absent	15	100	26	92.9		
3	History of seizure	Present	1	6.7	0	0	0.34 (NS0	---
		Absent	14	93.3	28	100		
4	History of headache	Present	4	26.7	3	10.7	0.21 (NS)	---
		Absent	11	73.3	25	89.3		
5	History of vomiting	Present	7	46.7	10	35.7	0.52 (NS)	---
		Absent	8	53.3	18	64.3		
6	History of altered mental status	Present	2	13.3	0	0	0.11 (NS)	---
		Absent	13	86.7	28	100		
7	History of motor disturbance	Present	4	26.7	0	0	0.011*	22.3 (1.1 to 448.9)
		Absent	11	73.3	28	100		
8	History of sensory disturbances	Present	0	0	1	3.6	0.99 (NS)	---
		Absent	15	100	27	96.4		
9	History of speech disturbances	Present	2	13.3	3	10.7	0.99 (NS)	---
		Absent	13	86.7	25	89.3		
10	History of CN abnormality	Present	1	6.7	1	3.6	0.99 (NS)	---
		Absent	14	93.3	27	96.4		

11	History of visual disturbances	Present	1	6.7	4	14.3	0.64 (NS)	---
		Absent	14	93.3	24	85.7		
12	History of Tremors	Present	0	0	1	3.6	0.99 (NS)	---
		Absent	15	100	27	96.4		
13	History of tinnitus	Present	1	6.7	2	7.1	0.99 (NS)	---
		Absent	14	93.3	26	92.9		
14	History of neck pain	Present	0	0	4	14.3	0.28 (NS)	---
		Absent	15	100	24	85.7		
15	History of palpitation	Present	0	0	1	3.6	0.99 (NS)	---
		Absent	15	100	27	96.4		
16	History of chest discomfort	Present	0	0	1	3.6	0.99 (NS)	---
		Absent	15	100	27	96.4		

Data are expressed as absolute numbers with proportions. Fisher's exact test was used to test the difference between the proportions. * indicates $p<0.05$ and considered statistically significant.

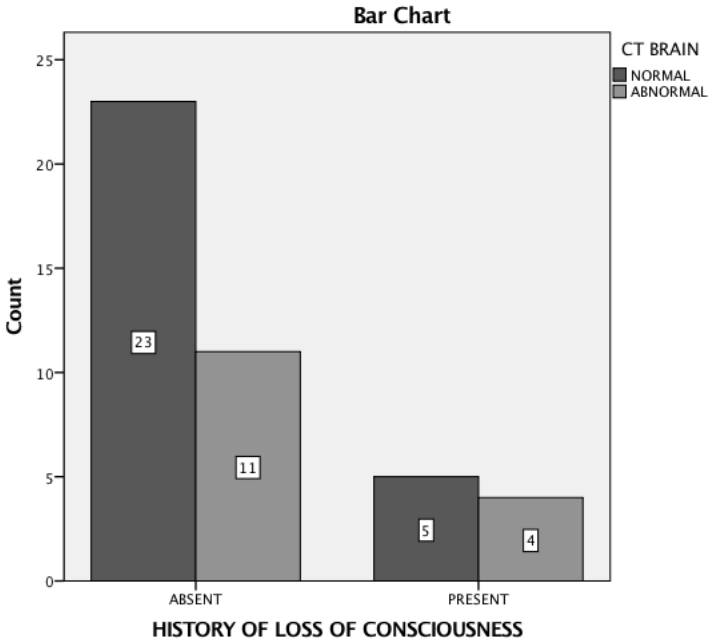


Figure 7: Bar chart representing the history of loss of consciousness in the study population in the x axis against the number of patients with normal or abnormal CT Brain in the y axis.

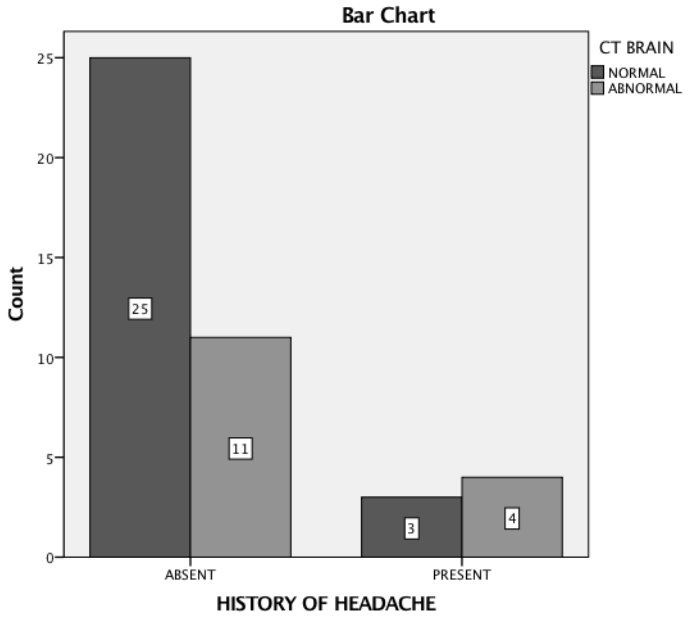


Figure 8: Bar chart representing the history of headache in the study population in the x axis against the number of patients with normal or abnormal CT Brain in the y axis.

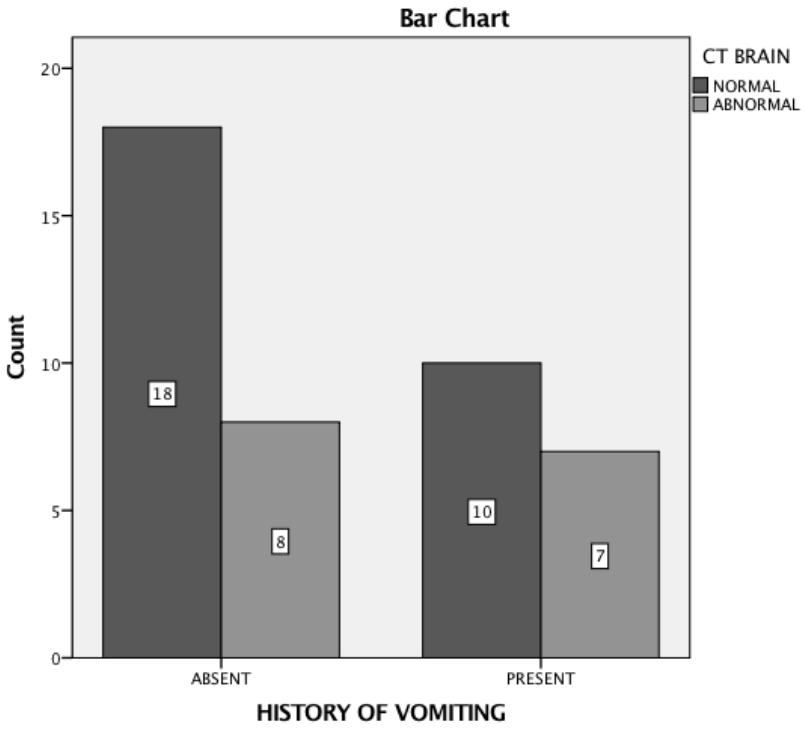


Figure 9: Bar chart representing the history of vomiting in the study population in the x axis against the number of patients with normal or abnormal CT Brain in the y axis.

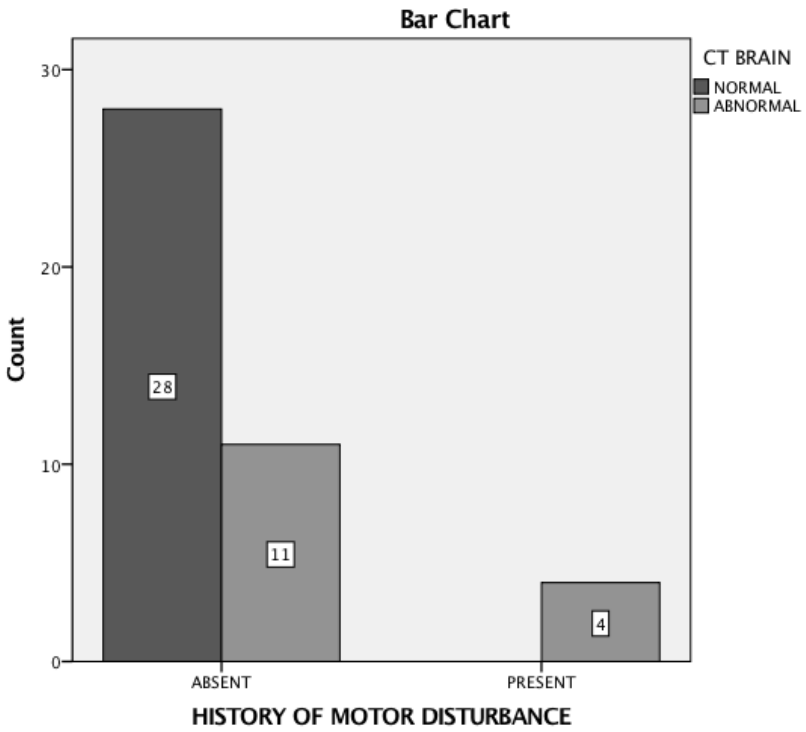


Figure 10: Bar chart representing the history of motor disturbance in the study population in the x axis against the number of patients with normal or abnormal CT Brain in the y axis.

5. RISK FACTORS

Table 8: Comparison of proportions of various risk factors between the patients with normal and abnormal CT findings.

S. No	Parameter		Abnormal CT group (n=15)		Normal CT group (n=28)		P value	Odds ratio (95% CI)
			n	%	n	%		
1	Hypertension	Present	10	66.7	9	32.1	0.049*	4.2 (1.1 to16.05)
		Absent	5	33.3	19	67.9		
2	Diabetes mellitus	Present	8	53.3	7	25	0.09 (NS)	---
		Absent	7	46.7	21	75		
3	Coronary artery disease	Present	2	13.3	1	3.6	0.27 (NS)	---
		Absent	13	86.7	27	96.4		
4	Heavy alcohol consumption	Present	8	53.3	6	21.4	0.045*	4.1 (1.07 to 16.3)
		Absent	7	46.7	22	78.6		
5	Current smoker	Present	3	20	2	7.1	0.32 (NS)	---
		Absent	12	80	26	92.9		

Data are expressed as absolute numbers with proportions. Fisher's exact test was used to test the difference between the proportions. * indicates $p<0.05$ and considered statistically significant.

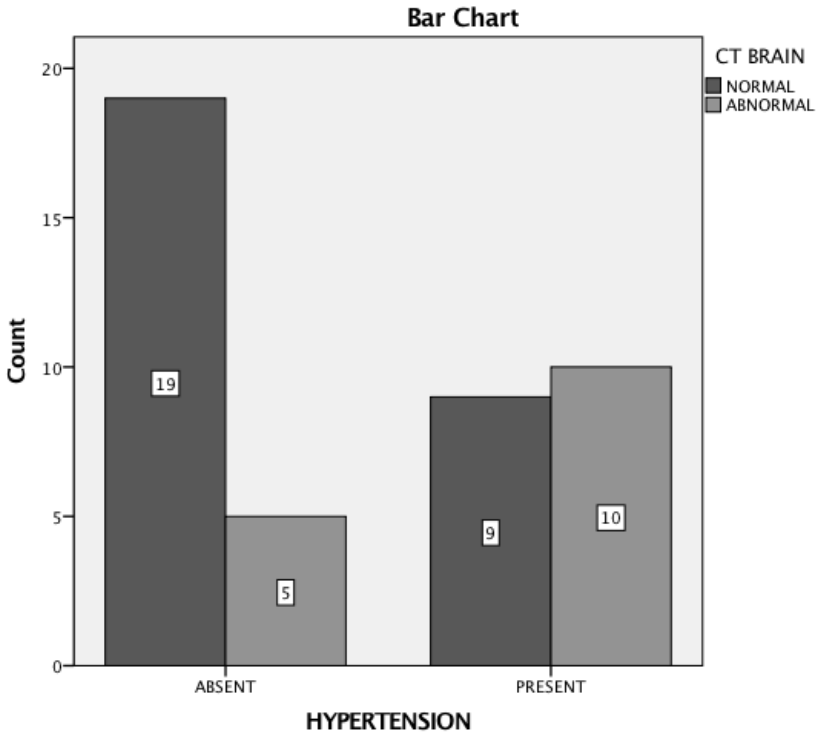


Figure 11: Bar chart representing the history of hypertension in the study population in the x axis against the number of patients with normal or abnormal CT Brain in the y axis.

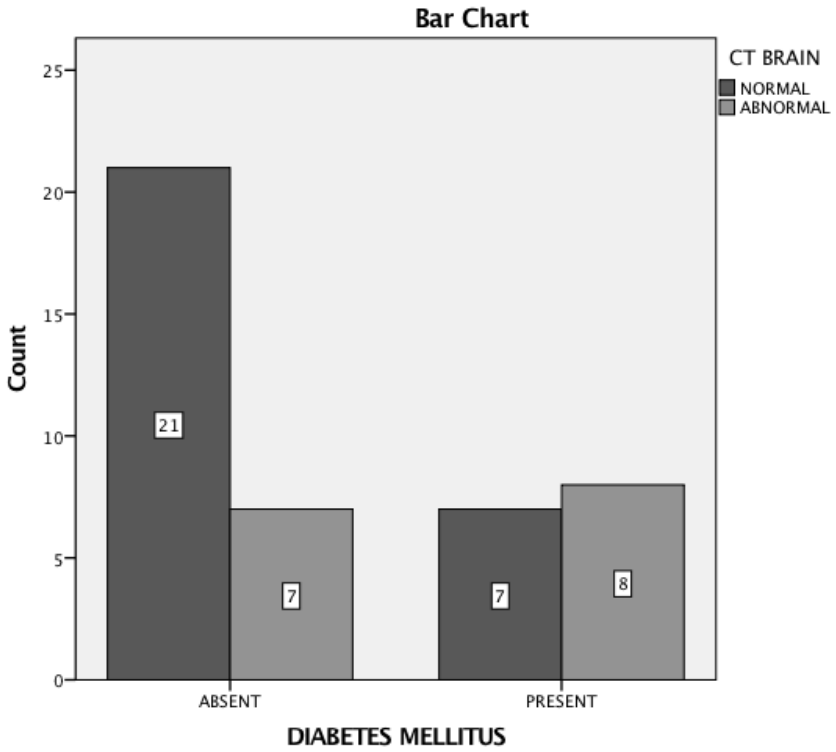


Figure 12: Bar chart representing the history of diabetes mellitus in the study population in the x axis against the number of patients with normal or abnormal CT Brain in the y axis.

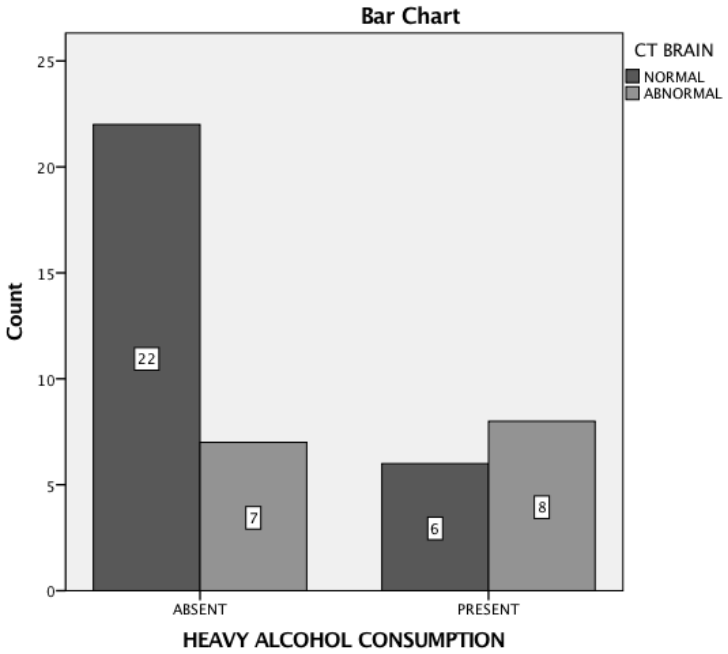


Figure 13: Bar chart representing the history of heavy alcohol consumption in the study population in the x axis against the number of patients with normal or abnormal CT Brain in the y axis.

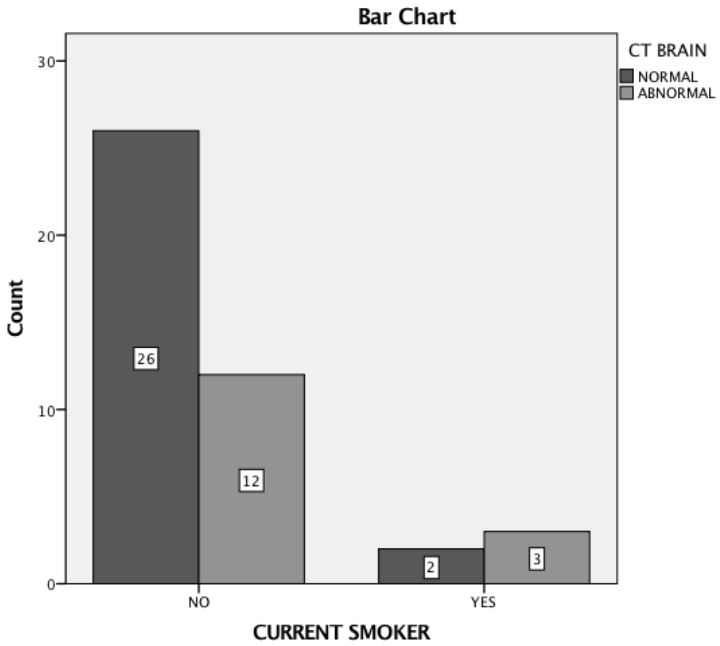


Figure 14: Bar chart representing the current smoker in the study population in the x axis against the number of patients with normal or abnormal CT Brain in the y axis.

6. VITAL SIGNS

Table 9: Comparison of proportions of various vitals between the patients with normal and abnormal CT findings.

S. No	**Parameter**	**CT Normal group (n=28)**		**CT abnormal group (n=15)**		**P value**	**Statistical test**
		Mean	**SD**	**Mean**	**SD**		
1	Systolic blood pressure (mm of Hg)	133	28.4	150	32.9	0.08 (NS)	Unpaired 't' test
2	Diastolic blood pressure (mm of Hg)	79.6	11.04	90	16.9	0.019*	Unpaired 't' test
3	Pulse rate (bpm)	79	11.1	78	15.2	0.8 (NS)	Unpaired 't' test

Data are expressed as mean with standard deviation. * indicates $p<0.05$ and considered statistically significant.

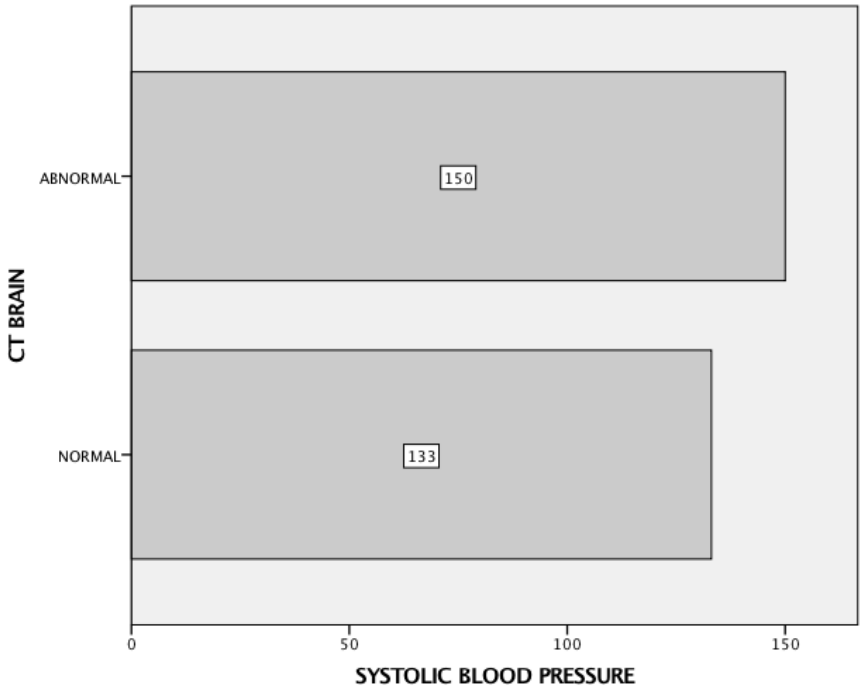


Figure 15: Bar chart showing the mean of systolic blood pressure in the study population in the X axis against the patients with normal and abnormal CT brain in the Y axis.

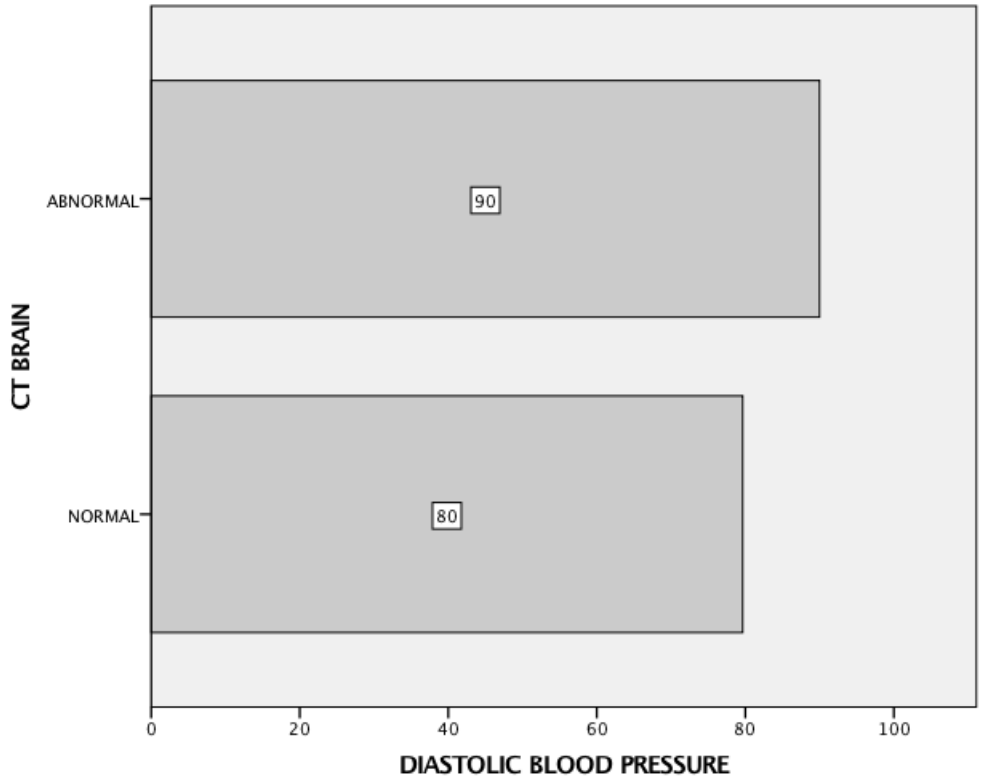


Figure 16: Bar chart showing the mean of diastolic blood pressure in the study population in the X axis against the patients with normal and abnormal CT brain in the Y axis.

7. CNS EXAMINATION

Table 10: Comparison of proportions of various histories between the patients with normal and abnormal CT findings.

S. No	Parameter		Abnormal CT group (n=15)		Normal CT group (n=28)		P value	Odds ratio (95% CI)
			n	%	n	%		
1	General CNS examination	Abnormal	13	86.7	13	46.4	0.02*	7.5 (1.4 to 39.6)
		Normal	2	13.3	15	53.6		
2	Defects in higher mental functions	Present	2	13.3	2	7.1	0.6 (NS)	---
		Absent	13	86.7	26	92.9		
3	Nystagmus	Present	2	13.3	2	7.1	0.6 (NS)	---
		Absent	13	86.7	26	92.9		
4	Ataxia	Present	3	20	9	32.1	0.49 (NS)	---
		Absent	12	18	19	67.9		
5	UMN signs	Present	7	46.7	2	7.1	0.004*	11.37 (1.95 to 66.1)
		Absent	8	53.3	26	92.9		

Data are expressed as absolute numbers with proportions. Fisher's exact test was used to test the difference between the proportions. * indicates $p<0.05$ and considered statistically significant.

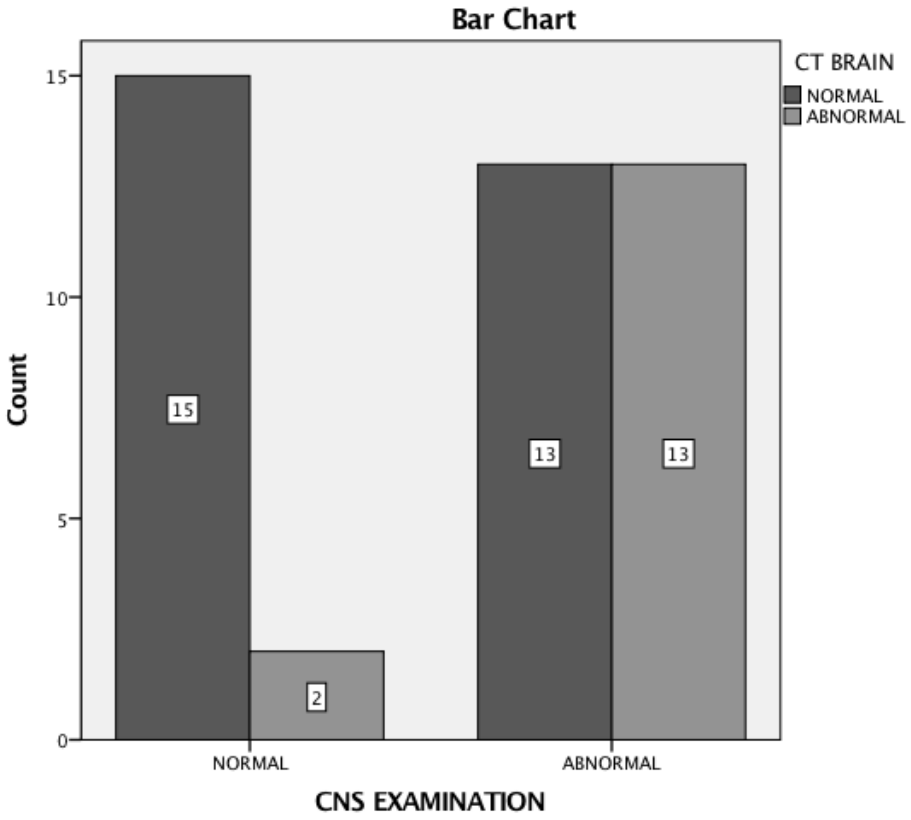


Figure 17: Bar chart representing the CNS examination in the study population in the x axis against the number of patients with normal or abnormal CT Brain in the y axis.

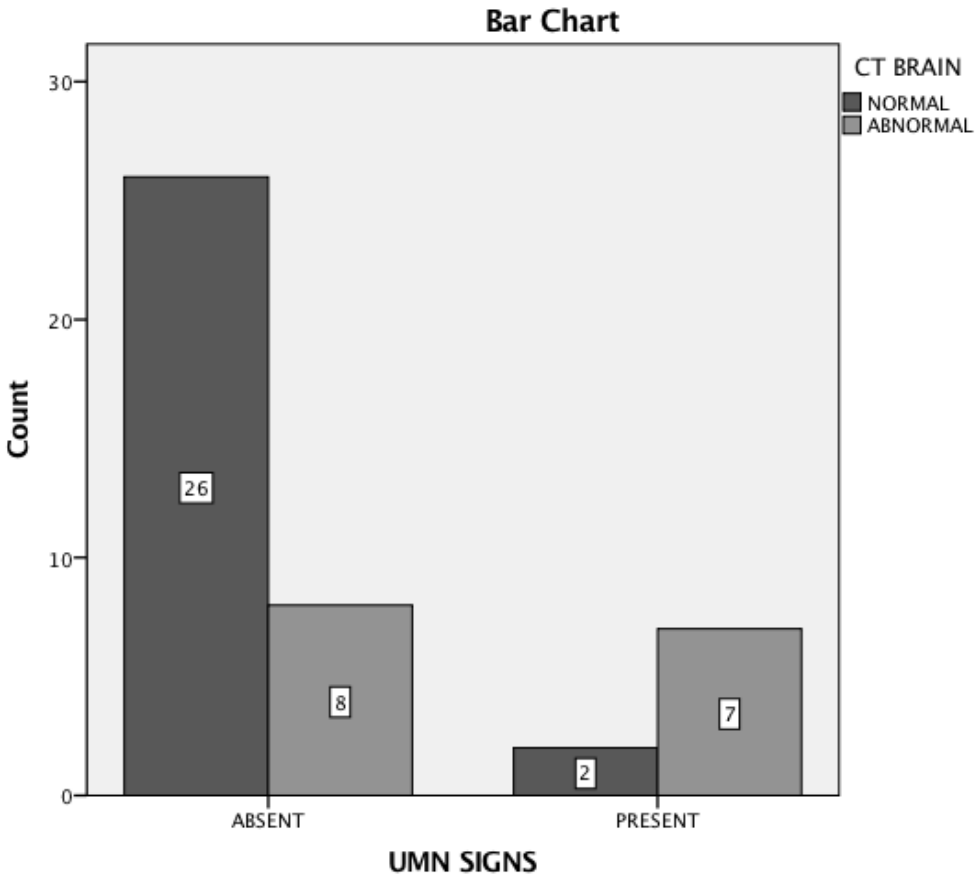


Figure 18: Bar chart representing the demonstration of Upper Motor Neuron signs in the study population in the x axis against the number of patients with normal or abnormal CT Brain in the y axis.

8. LABORATORY INVESTIGATIONS

Table 11: Comparison of proportions of various blood investigations between the patients with normal and abnormal CT findings.

S. No	Parameter	CT Normal group (n=28)		CT abnormal group (n=15)		P value	Statistical test
		Mean	SD	Mean	SD		
1	Hemoglobin (g/dl)	10.01	1.66	9.03	1.05	0.04*	Unpaired 't' test
2	Blood Urea (mg/dl)	36.5	11.8	46.67	27.3	0.18 (NS)	Mann Whitney U test
3	Serum creatinine (mg/dl)	0.92	0.29	1.2	0.66	0.059 (NS)	Unpaired 't' test
4	Random Blood sugar (mg/dl)	197	162	151	113	0.33 (NS)	Mann Whitney U Test

Data are expressed as mean with standard deviation. * indicates $p<0.05$ and considered statistically significant.

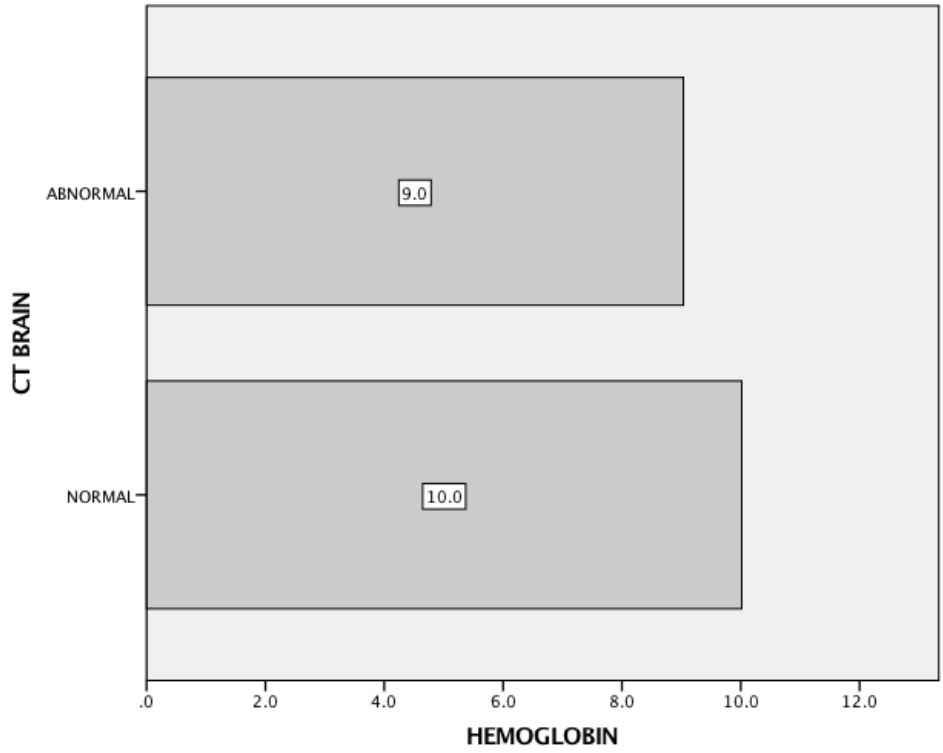


Figure 19: Bar chart showing the mean of Hemoglobin values (in g/dl) in the study population in the X axis against the patients with normal and abnormal CT brain in the Y axis.

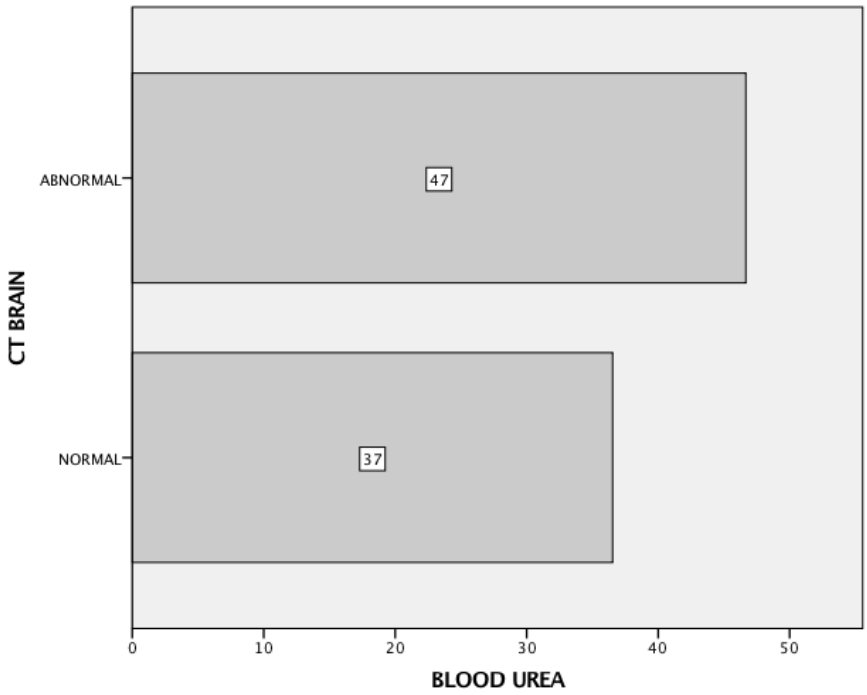


Figure 20: Bar chart showing the mean of Blood Urea values (in mg/dl) in the study population in the X axis against the patients with normal and abnormal CT brain in the Y axis.

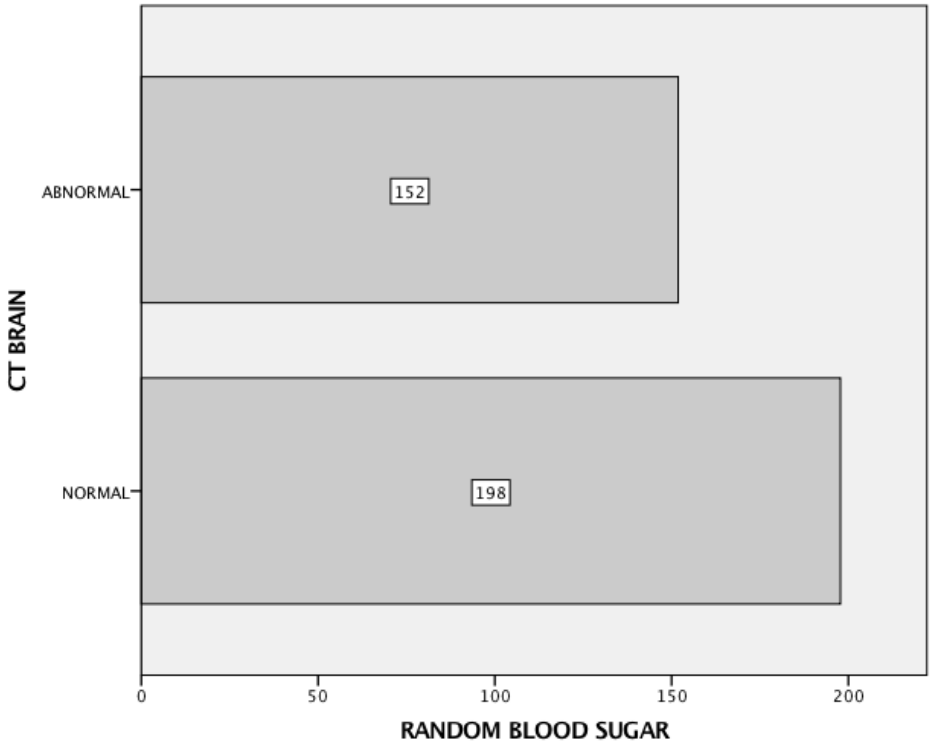


Figure 21: Bar chart showing the mean of Random blood sugar (mg/dl) in the study population in the X axis against the patients with normal and abnormal CT brain in the Y axis.

9. ECG CHANGES

Table 12: Comparison of ECG changes between the patients with normal and abnormal CT findings.

Parameter		**Abnormal CT group (n=15)**		**Normal CT group (n=28)**		**P value**
		n	**%**	**n**	**%**	
ECG changes	Present	5	33.3	9	32.1	0.99 (NS)
	Absent	10	66.7	19	67.9	

Data are expressed as absolute numbers with proportions. Fisher's exact test was used to test the difference between the proportions. * indicates $p<0.05$ and considered statistically significant.

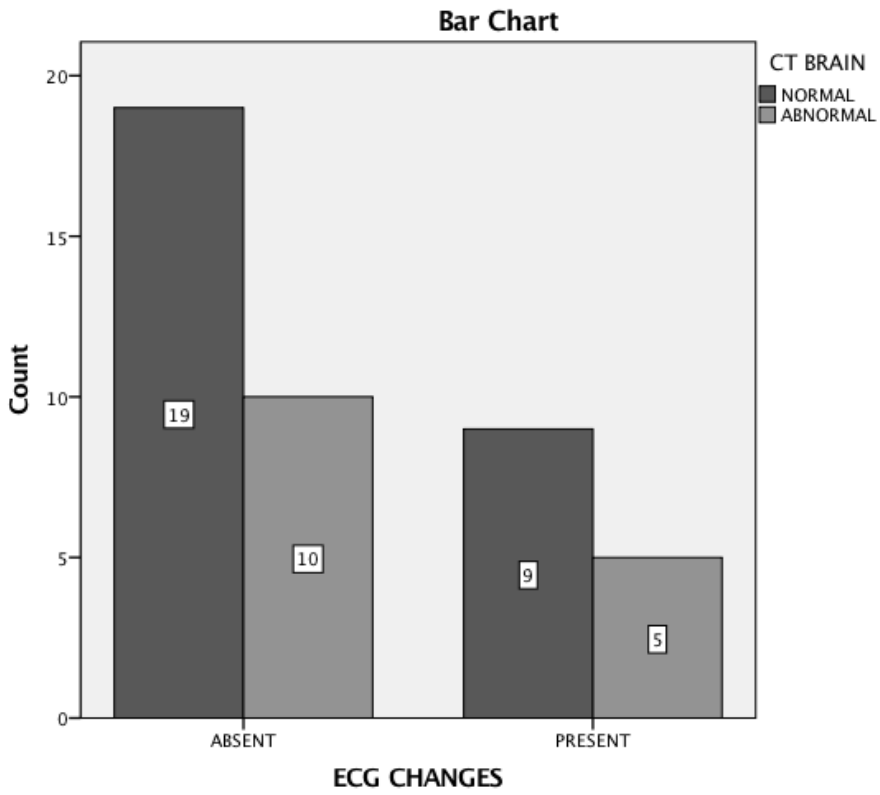


Figure 22: Bar chart representing the Ecg changes in the study population in the x axis against the number of patients with normal or abnormal CT Brain in the y axis.

10. ADMISSION RANDOM BLOOD SUGAR

RBS with hypoglycemia defined as <81 mg/dl, normoglycemia as 81-200mg/dl and hyperglycemia as >200mg/dl. 10 patients with dizziness had hypoglycemia and even among them 4 had abnormal CT Brain (40%).

Table 13: Comparison of Admission RBS between the patients with normal and abnormal CT findings.

Parameter		**Abnormal CT group (n=15)**		**Normal CT group (n=28)**		**P value**
		n	**%**	**n**	**%**	
RBS (mg/dl)	<81	4	40	6	60	0.8 (NS)
	81-200	8	36.4	14	63.6	
	>200	3	27.3	8	72.7	

Data are expressed as absolute numbers with proportions. * indicates $p<0.05$ and considered statistically significant.

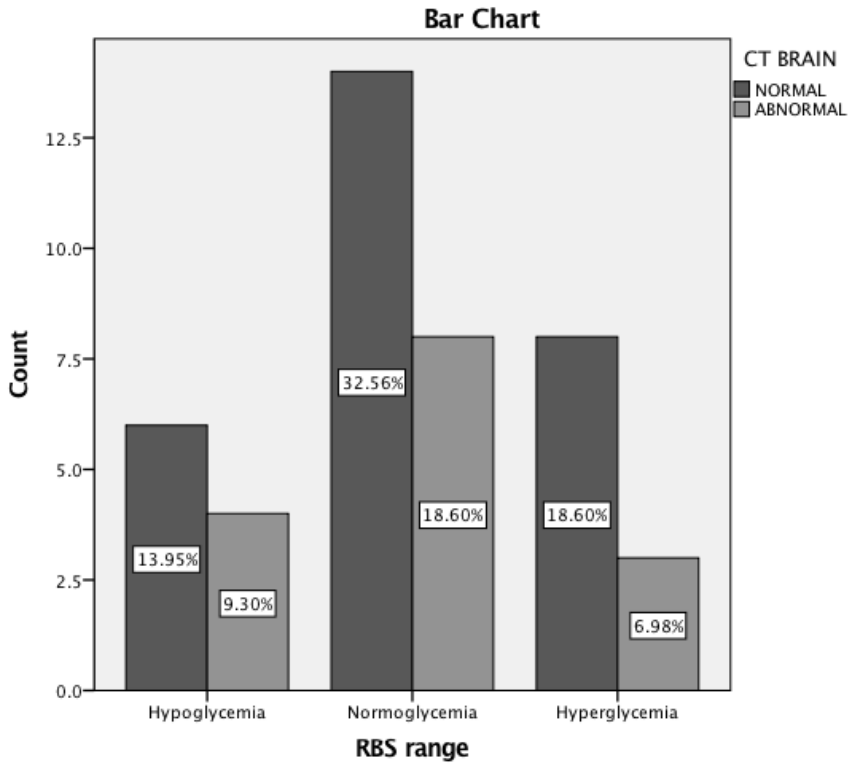


Figure 23: Bar chart representing the RBS range in the study population in the x axis against the number of patients with normal or abnormal CT Brain in the y axis.

From the observed patients (n=43) presenting with giddiness, history of hypertension, vomiting, diabetes, alcohol consumption and Loss of consciousness formed the majority.

11. CT BRAIN

15 patients had abnormal CT Brain (34.88%) and 28 patients had normal CT Brain (65.12%).

5 patients had imaging suggestive of Posterior circulation stroke. (11.6%)

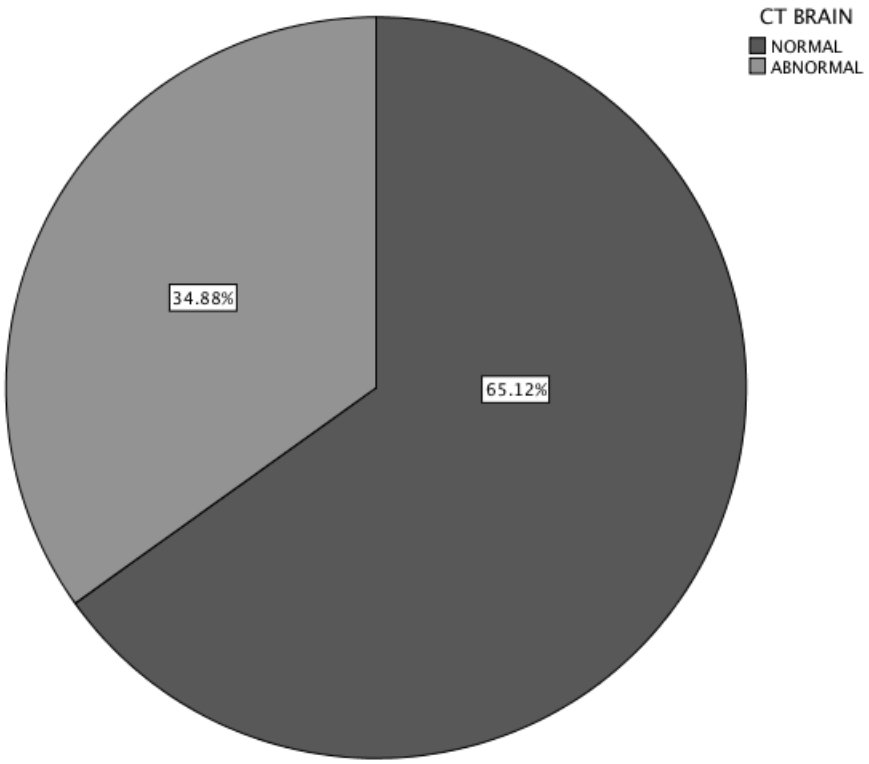


Figure 24: Pie Chart showing CT Brain finding in the study population.

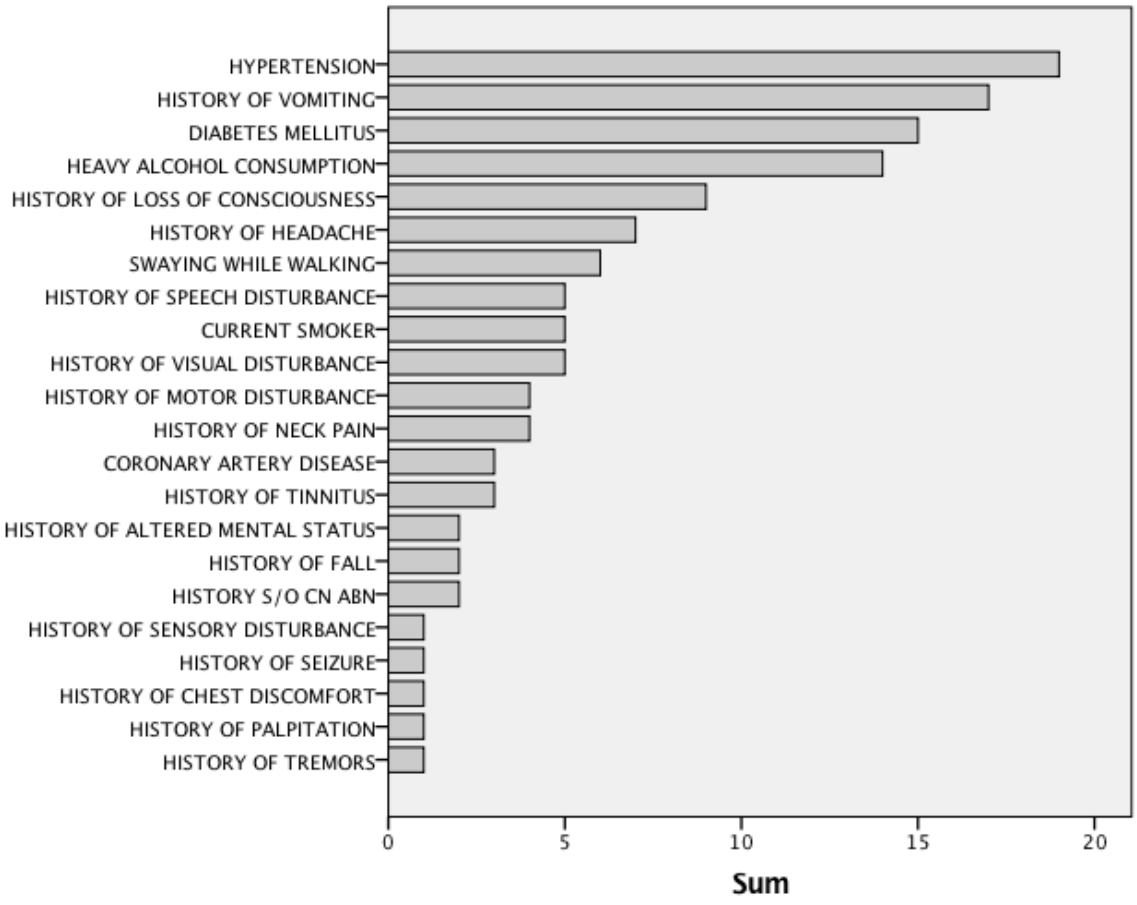
TABLE 14: CT BRAIN VS CNS EXAMINATION IN STUDY POPULATION				
N	SEX	AGE	CNS EXAMINATION	CT BRAIN
1.	M	29	NORMAL	NORMAL
2.	F	55	NORMAL	NORMAL
3.	F	41	ABNORMAL, NYSTAGMUS PRESENT	HYPODENSE LESION LEFT FRONTAL CORTEX
4.	F	65	ABNORMAL, ATAXIA PRESENT	NORMAL
5.	M	42	ABNORMAL, RIGHT III, VI PALSY	NORMAL
6.	M	51	ABNORMAL, DROWSY, BILATERAL EXTENSOR PLANTAR	LEFT PARIETAL LACUNAR INFARCT
7.	M	60	ABNORMAL, GAIT ATAXIA, NYSTAGMUS TO RIGHT	HYPODENSITY RIGHT CEREBELLAR REGION
8.	M	55	SLURRING SPEECH	NORMAL
9.	M	45	NORMAL	NORMAL
10.	M	55	NORMAL	NORMAL
11.	M	63	MOTOR APHASIA	ACUTE INFARCT LEFT PERIVENTRICULAR REGION

12.	M	72	NORMAL	NORMAL
13.	F	84	NORMAL	BILATERAL PERIVENTRICULAR HYPODENSITY
14.	F	70	DROWSY, RIGHT PLANTAR EXTENSOR	BRAINSTEM HEMORRHAGE WITH INTRAVENTRICULAR EXTENSION
15.	M	42	NORMAL	NORMAL
16.	M	65	DROWSY, LEFT HEMIPARESIS	NORMAL
17.	M	62	UNCONSCIOUS, ANISOCORIA, BILATERAL PLANTAR EXTENSOR	BILATERAL DIFFUSE SAH WITH IVH, BASAL CISTERN BLEED
18.	F	70	DROWSY, BILATERAL PLANTAR NO RESPONSE	NORMAL
19.	F	45	NORMAL	NORMAL
20.	M	63	NORMAL	NORMAL
21.	M	67	NORMAL	HYPODENSITY LEFT CEREBELLAR REGION
22.	F	56	ATAXIA TO LEFT	NORMAL
23.	F	37	ATAXIA, NYSTAGMUS	NORMAL

24.	F	80	STANCE ATAXIA	NORMAL
25.	F	70	NORMAL	NORMAL
26.	F	50	NORMAL	NORMAL
27.	F	60	LEFT HEMIPLEGIA, LEFT UMN FACIAL PALSY	SUBACUTE RIGHT MCA TERRITORY INFARCT
28.	F	42	ATAXIA TO LEFT	NORMAL
29.	F	40	NORMAL	NORMAL
30.	F	65	NYSTAGMUS, RHOMBERGS, ATAXIA	NORMAL
31.	M	50	STANCE ATAXIA, GAIT ATAXIA	LEFT CEREBELLAR HYPODENSITY
32.	F	50	LEFT HEMIPARESIS	RIGHT THALAMOCAPSULAR INFARCT
33.	F	42	STANCE ATAXIA, GAIT ATAXIA	NORMAL
34.	F	70	STANCE ATAXIA	NORMAL
35.	F	67	NORMAL	NORMAL
36.	F	40	NORMAL	NORMAL

37.	F	65	STANCE ATAXIA, NECK STIFFNESS	SAH WIT IVH
38.	F	52	LEFT HEMIPARESIS	RIGHT PARIETAL INFARCT
39.	M	75	BILATERAL DECREASED VISUAL ACUITY	MULTIPLE INFARCTS
40.	M	39	GAIT ATAXIA, RHOMBERGS +	NORMAL
41.	M	75	RIGHT HEMIPARESIS	LEFT CEREBELLAR HYPODENSITY
42.	M	72	NORMAL	NORMAL
43.	M	70	NORMAL	NORMAL

12. Bar diagram representation of various histories and risk factors in the study population: Figure 25



13. Table 15: Diagnostic test values of covariates that predicts the normal/abnormal findings in the CT scan.

S. No	Variables (if positive)	Sensitivity (%)	Specificity (%)	Positive predictive value	Negative predictive value
1	Hypertension	66.7	67.8	52.6	79.1
2	Heavy alcohol consumption	53.3	78.5	57.1	75.8
3	History of motor disturbance	26.6	100	100	71.8
4	CNS defects on examination	86.7	53.5	50	88.2
5	UMN signs	46.7	92.8	77.8	76.5

14. Table 16: Factors selected for multivariate analysis based on p value obtained in univariate logistic regression and p values in table to.

S.No	Variables (Selected based on p values in table 1 to 3)	Type	P value (Wald test)	P value (LR test)	Selected for multivariate regression analysis
1	Dizziness Duration	Numerical	0.09(NS)	0.003	NO
2	Hypertension	Categorical	0.034	0.02	YES
3	Heavy alcohol consumption	Categorical	0.039	0.035	YES
4	History of motor disturbance	Categorical	0.999 (NS)	0.002	NO
5	CNS defects on examination	Categorical	0.018	0.007	YES
6	UMN signs	Categorical	0.007	0.003	YES
7	Hemoglobin	Numerical	0.053 (NS)	0.03	NO
8	Diastolic blood pressure	Numerical	0.03	0.01	YES

15. Table 17: Multivariate logistic regression model of predictors of stroke associated with dizziness, vertigo or imbalance

S. No	Variables (Selected based on p values in table 1 to 3)	Crude (unadjusted) Odds ratio Exp (B)	95% confidence interval of crude odds ratio	Adjusted Odds ratio	95% Confidence interval
1	UMN signs	11.37	1.95 to 66.13	4.91	0.69 to 34.7
2	CNS defects on examination	7.5	1.4 to 39.6	5.08	0.7 to 33.4
3	History of Hypertension	4.2	1.1 to 16.05	4.17	0.87 to 19.9

Five covariates namely UMN signs, CNS defects on examination, hypertension, diastolic blood pressure and heavy alcohol consumption were included for multivariate logistic regression analysis. By forward and backward LR method, with p values based Walds test and LR test, heavy alcohol and diastolic blood pressure lost the significance. Hence both these factors were eliminated in the final analysis. The crude (unadjusted) odds ratio of remaining three covariates was considered for the interaction and the adjusted odds ratio was calculated.

DISCUSSION

1. Among the 43 patients included in this study, 35% had abnormal CT Brain (15 patients) and 65% had normal CT Brain (28 patients)
2. Among 15 patients with abnormal CT Brain, 5 had posterior circulation stroke. (11.6%)
3. Among the various risk factors and histories studied, the most common ones in descending order are hypertension, history of vomiting, diabetes mellitus and history of alcohol consumption.
4. Age
    a. The mean age group of the abnormal CT group was 61.6 as compared to 55.5 in the normal CT group but it was statistically insignificant. (p - 0.12)
    b. When the age was divided into different age groups of <40 years, 40-60 years and >60 years, patients with abnormal CT brain was maximum in the >60 years group with 40% of the total. None in the age group <40 years had abnormal CT Brain.
5. Sex
    a. Males constituted 53% of cases in the abnormal CT group whereas females constituted 47%.
    b. In the normal CT group females constituted 57%.
    c. P value was 0.54 and not significant for sex distribution.

6. Duration of dizziness
    a. The mean duration of dizziness was 3 days in normal CT group
    b. The mean duration of dizziness was 1 day in abnormal CT group and it was statistically significant using unpaired t test with a p value of 0.009.
    c. When the duration of giddiness was divided into <1 day, 1-3 days and > 3 days, the p value was 0.042 and was statistically significant. 93.3% of patients with abnormal CT brain had duration of <1 day.
    d. 69.7% of the study population had dizziness duration of less than 1 day.
7. Other histories
    a. History of loss of consciousness was present in 27% of the patients with abnormal CT brain. It was absent in 82% of patients with normal CT brain. It was statistically not significant. (p value 0.69)
    b. History of fall was present in none of the patients with abnormal CT brain. It was absent in 93% of patients with normal CT brain. It was statistically not significant. (p value 0.53)
    c. History of seizure was present in 7% of cases with abnormal CT brain. It was absent in 100% of patients with normal CT brain. It was statistically not significant. (p value 0.34)

d. History of headache was present in 27% of patients with abnormal CT Brain. It was absent in 89% of patients with normal CT Brain. It was statistically not significant. (p value 0.21)
e. History of vomiting was present in 47% of patients with abnormal CT Brain. It was absent in 64% of patients with normal CT Brain. It was statistically not significant. (p value 0.52)
f. History of altered mental status was present in 13% of patients with abnormal CT Brain. It was absent in 100% of patients with normal CT Brain. It was statistically not significant. (p value 0.11)
g. History of motor disturbance was present in 27% of patients with abnormal CT Brain. It was absent in 100% of patients with normal CT Brain. It was statistically SIGNIFICANT. (p value 0.011)
h. History of sensory disturbance was not present in patients with abnormal CT Brain. It was absent in 96% of patients with normal CT Brain. It was statistically not significant. (p value 0.99)
i. History of speech disturbance was present in 13% of patients with abnormal CT Brain. It was absent in 89% of patients with normal CT Brain. It was statistically not significant. (p value 0.99)
j. History of Cranial Nerve abnormality was present in 7% of patients with abnormal CT Brain. It was absent in 96% of patients with normal CT Brain. It was statistically not significant. (p value 0.99)

k. History of visual disturbance was present in 7% of patients with abnormal CT Brain. It was absent in 86% of patients with normal CT Brain. It was statistically not significant. (p value 0.64)

l. History of Tremors was not present in patients with abnormal CT Brain. It was absent in 96% of patients with normal CT Brain. It was statistically not significant. (p value 0.99)

m. History of tinnitus was present in 7% of patients with abnormal CT Brain. It was absent in 93% of patients with normal CT Brain. It was statistically not significant. (p value 0.99)

n. History of neck pain was not present in patients with abnormal CT Brain. It was absent in 86% of patients with normal CT Brain. It was statistically not significant. (p value 0.28)

o. History of palpitation was not present in patients with abnormal CT Brain. It was absent in 96% of patients with normal CT Brain. It was statistically not significant. (p value 0.99)

p. History of chest discomfort was not present in patients with abnormal CT Brain. It was absent in 96% of patients with normal CT Brain. It was statistically not significant. (p value 0.99)

8. Risk factors

a. Hypertension was present in 66.7% of patients with abnormal CT and was absent in 68% of patients with normal CT. It was

statistically SIGNIFICANT with a p value of 0.049. The Odds ratio calculated with 95% confidence interval is 4.2.

b. Diabetes Mellitus was present in 54% of patients with abnormal CT and absent in 75% of patients with normal CT. It was statistically not significant with a p value of 0.09.

c. Heavy alcohol consumption was present in 53% of patients with abnormal CT and absent in 79% of patients with normal CT. It was statistically SIGNIFICANT with a p value of 0.045. The Odds ratio calculated with 95% confidence interval is 4.1.

d. 20% of patients with abnormal CT were current smokers and 93% non-smokers had normal CT. It was statistically not significant with a p value of 0.32.

9. Vital signs

    a. The mean systolic blood pressure in patients with abnormal CT brain was 150 mmHg and in patients with normal CT brain was 133mmHg. It was found to be statistically not significant using Unpaired t test with a p value of 0.08.

    b. The mean diastolic blood pressure in patients with abnormal CT brain was 90mmHg and in patients with normal CT brain was 80mmHg. It was found to be statistically SIGNIFICANT using Unpaired t test with a p value of 0.019.

c. The mean pulse rate in patients with abnormal CT brain was 78 per minute and in patients with normal CT brain was 79 per minute. It was found to be statistically not significant using Unpaired t test with a p value of 0.8.

10. CNS Examination

a. General Complete CNS examination was abnormal in 87% of patients with abnormal CT Brain and normal in 54% of patients with normal CT brain. It was found to be statistically SIGNIFICANT with a p value of 0.02. The Odds ratio calculated using 95% confidence interval was 7.5.

b. Defects in higher mental function were present in 13.3% of patients with abnormal CT brain and was absent in 93% of patients with normal CT Brain. It was found to be statistically not significant with a p value of 0.6.

c. Nystagmus were present in 13% of patients with abnormal CT brain and was absent in 93% of patients with normal CT Brain. It was found to be statistically not significant with a p value of 0.6.

d. Ataxia were present in 20% of patients with abnormal CT brain and was absent in 68% of patients with normal CT Brain. It was found to be statistically not significant with a p value of 0.49.

e. Upper motor neuron signs were present in 47% of patients with abnormal CT Brain and not present in 93% of patients with normal CT brain. It was found to be statistically SIGNIFICANT with a p value of 0.004. The Odds ratio calculated using 95% confidence interval was 11.37.

11. Laboratory Investigations

a. The mean hemoglobin in patients with abnormal CT brain was 9g/dl and in patients with normal CT brain was 10 g/dl. It was found to be statistically SIGNIFICANT using Unpaired t test with a p value of 0.04.

b. The mean blood urea in patients with abnormal CT brain was 46.7mg/dl and in patients with normal CT brain was 36.5mg/dl. It was found to be statistically not significant using Mann Whitney U test with a p value of 0.18.

c. The mean serum creatinine in patients with abnormal CT brain was 1.2mg/dl and in patients with normal CT brain was 0.9mg/dl. It was found to be statistically not significant using Unpaired t test with a p value of 0.059.

d. The mean random blood sugar in patients with abnormal CT brain was 151mg/dl and in patients with normal CT brain was 197mg/dl. It was found to be statistically not significant using Mann Whitney U test with a p value of 0.33.

e. Random blood sugar was divided into 3 categories of hypoglycemia (<81mg/dl), normoglycemia (81-200mg/dl) and hyperglycemia (>200mg/dl). It was found that 10 patients with hypoglycemia had abnormal CT brain (40%).

12. Significant ECG Changes were present in 33% of patients with abnormal CT brain and 68% of patients with normal CT Brain. It was found to be statistically not significant with a p value of 0.99.
13. Univariate logistic regression was done for different variates which showed significance earlier with unpaired t test or Mann Whitney U test or fischer exact test:
    a. Dizziness duration was found to be not significant with a p value of 0.09 using Wald test and was not selected for multivariate regression analysis.
    b. History of Hypertension was found to be SIGNIFICANT with a p value of 0.034 using Wald test and was selected for multivariate regression analysis.
    c. Heavy alcohol consumption was found to be SIGNIFICANT with a p value of 0.039 using Wald test and was selected for multivariate regression analysis.
    d. History of motor disturbance was found to be not significant with a p value of 0.999 using Wald test and was not selected for multivariate regression analysis.

e. CNS defects on examination was found to be SIGNIFICANT with a p value of 0.018 using Wald test and was selected for multivariate regression analysis.
f. UMN signs was found to be SIGNIFICANT with a p value of 0.007 using Wald test and was selected for multivariate regression analysis.
g. Hemoglobin was found to be not significant with a p value of 0.053 using Wald test and was not selected for multivariate regression analysis.
h. Diastolic blood pressure was found to be SIGNIFICANT with a p value of 0.03 using Wald test and was selected for multivariate regression analysis.

14. Multivariate logistic regression was done for five covariates selected. By forward and backward LR method, with p values based Walds test and LR test, heavy alcohol and diastolic blood pressure lost the significance.
    a. UMN signs showed an adjusted odds ratio of 4.91 using 95% confidence interval.
    b. CNS defects on examination showed an adjusted odds ratio of 5.08 using 95% confidence interval.
    c. History of hypertension showed an adjusted odds ratio of 4.17 using 95% confidence interval.

SUMMARY

1. CT Brain is still a useful investigation in the era of MRI and advanced imaging and a significant number of patients presenting with dizziness – 35% had abnormal CT brain imaging suggestive of posterior circulation stroke.
2. 12% of patients presenting with dizziness had features of posterior circulation stroke on CT Brain imaging.
3. Most patients (70%) admitted with dizziness had duration of dizziness of less than 1 day.
4. 10 patients with hypoglycemia had abnormal CT Brain. So, hypoglycemia cannot be considered as an exclusion for not considering CT brain.
5. Patients with history of hypertension had a 4 times risk of having an abnormal CT brain as compared to patients without hypertension.
6. Patients with UMN signs or pyramidal signs on examination had 5 times odds of having an abnormal CT brain as compared to patients without any UMN signs.
7. Patients with abnormality in CNS examination had 5 times odds of having an abnormal CT brain as compared to patients with a normal CNS examination.

CONCLUSION

Acute posterior circulation stroke can present with nonspecific clinical findings. It can also present with a normal CT Brain. Despite limitations of our study we were able to identify that history of hypertension as a significant risk factor in our population associated with abnormal CT Brain finding. We were also able to hypothesize that abnormal CNS examination and Upper Motor Neuron signs or Pyramidal signs demonstrated on examination correlate with abnormal CT Brain. These findings suggest that a thorough history and CNS examination are essential for patients presenting with dizziness, vertigo and imbalance. Increased education and focus on these features will allow us to do appropriate diagnostic workup including emergency CT Brain and the need for MRI brain. More focused large-scale studies are needed for the same to identify other risk factors to conserve health care resources by doing appropriate diagnostic workup.

LIMITATIONS

- Small study population
- Small study period of 6 months
- Most patients are not examined by a neurologist in the emergency department.
- Subtle neurological findings may have been missed in some patients[123]
- Study is not designed to collect specific features sought by neuro-otology specialists.
- Brain MRI was not ordered in most patients because of limitations
- Brain MRI may identify small brainstem or cerebellar strokes resembling peripheral vestibular disorders[124-126]
- Patient population predominantly included people from Thanjavur district and this might not be generalized to all populations.

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127.

ANNEXURE 1

PROFORMA

A STUDY ON INCIDENCE OF STROKE IN PATIENTS PRESENTING WITH ACUTE DIZZINESS, VERTIGO AND IMBALANCE IN EMERGENCY DEPARTMENT

DEMOGRAPHIC DATA:

NAME: AGE/SEX:

ADDRESS: OCCUPATION:

PHONE NO: IP NO.:

PROVISIONAL DIAGNOSIS:

HISTORY: **DESCRIPTION & DURATION**

	ISOLATED DIZZINESS SYMPTOMS	
	LOSS OF CONSCIOUSNESS	
	FALL	
	SEIZURE	
	HEADACHE/VOMITING/NAUSEA	
	ALTERED MENTAL STATUS	
	MOTOR DEFICIT	
	SENSORY DEFICIT	
	LANGUAGE DISTURBANCE	
	VISUAL CHANGES	
	TINNITUS/EAR ACHE/DISCHARGE	

	PALPITATION/CHEST DISCOMFORT	
	NECK PAIN	
	HYPERTENSION/DIABETES/CORONARY HEART DISEASE/ DYSLIPIDEMIA	
	PREVIOUS TIA/STROKE	
	CURRENT SMOKER	
	HEAVY ALCOHOL CONSUMPTION	
	RHD/ATRIAL FIBRILLATION	
	USE OF ANTICOAGULANTS	

PHYSICAL EXAMINATION:

BP: PR:

CNS:

OTHER SYSTEMS:

INVESTIGATIONS:

HEMOGLOBIN:

BLOOD UREA/SERUM CREATININE:

ADMISSION BLOOD GLUCOSE:

LIPID PROFILE:

ECG:

CT BRAIN:

MRI BRAIN:

COURSE IN THE HOSPITAL:

ANNEXURE -2

CONSENT FORM

Yourself Mr./Mrs./Ms…….. are being asked to be a participant in the research study titled "**A Study On "Incidence Of Stroke In Patients Presenting With Acute Dizziness, Vertigo And Imbalance In Emergency Department**"in Thanjavur Medical College, conducted by Dr. Amith Viswanath M.D., Post Graduate Student, Department of General Medicine, Thanjavur Medical College. You are eligible after looking into the inclusion criteria. You can ask any question you may have before agreeing to participate.

Research Being Done

A study on the incidence of stroke in patients presenting with acute dizziness, vertigo and imbalance in the emergency department by comparing various variables like different histories, risk factors, vital signs, basic investigations, ecg with the ct brain finding.

Purpose of Research

1. To determine the prevalence of stroke in patients presenting with acute dizziness, vertigo and imbalance in the emergency department as diagnosed by CT Brain.
2. To identify the importance of emergency CT Brain.

Decline from Participation

You have the option to decline from participation in the study existing protocol for your condition.

Privacy and Confidentiality

Privacy of individuals will be respected and any information about you or provided by you during the study will be kept strictly confidential.

Authorization to publish Results

Results of the study may be published for scientific purposes and/or presented to scientific groups, however you will not be identified.

Statement of Consent

I volunteer and consent to participate in this study. I have read the consent or it has been read to me. The study has been fully explained to me, and I may ask questions at any time.

---------- ----------

Signature /Left thumb impression Date

(volunteer)

---------- ----------

Signature of witness Date

ANNEXURE 3

MASTER CHART

KEY TO MASTER CHART

1. NAME
2. SEX – M – MALE, F - FEMALE
3. AGE IN YEARS
4. IP NUMBER – IN PATIENT NUMBER
5. ADDRESS
6. DIZZINESS – DURATION IN DAYS
7. LOC – HISTORY OF LOSS OF CONSCIOUSNESS – 1 – PRESENT, 0 – ABSENT
8. FALL – HISTORY OF FALL – 1 – PRESENT, 0 – ABSENT
9. SEIZURE – HISTORY OF SEIZURE – 1 – PRESENT, 0 – ABSENT
10. HEADACHE – HISTORY OF HEADACHE – 1 – PRESENT, 0 – ABSENT
11. VOMITING – HISTORY OF VOMITING – 1 – PRESENT, 0 – ABSENT
12. ALTERED MENTAL STATUS – HISTORY OF ALTERED MENTAL BEHAVIOUR – 1 – PRESENT, 0 – ABSENT
13. MOTOR DEFICIT – HISTORY SUGGESTIVE OF MOTOR DEFICIT – 1 – PRESENT, 0 – ABSENT
14. SENSORY DEFICIT – HISTORY SUGGESTIVE OF SENSORY DEFICIT – 1 – PRESENT, 0 – ABSENT

15. LANGUAGE DISTURBANCE – HISTORY SUGGESTIVE OF SPEECH DISTURBANCE – 1 – PRESENT, 0 – ABSENT
16. CN – HISTORY SUGGESTIVE OF CRANIAL NERVE ABNORMALITY – 1 – PRESENT, 0 – ABSENT
17. VISUAL DISTURBANCE – HISTORY SUGGESTIVE OF VISUAL DISTURBANCE– 1 – PRESENT, 0 – ABSENT
18. TREMORS – HISTORY OF TREMORS– 1 – PRESENT, 0 – ABSENT
19. TINNITUS – HISTORY OF TINNITUS– 1 – PRESENT, 0 – ABSENT
20. NECK PAIN- HISTORY OF NECK PAIN– 1 – PRESENT, 0 – ABSENT
21. PALPITATION – HISTORY OF PALPITATION – 1 – PRESENT, 0 – ABSENT
22. CHEST DISCOMFORT – HISTORY OF CHEST PAIN OR DISCOMFORT – 1 – PRESENT, 0 – ABSENT
23. HTN – KNOWN CASE OF SYSTEMIC HYPERTENSION – 1 – PRESENT, 0 – ABSENT
24. DM – KNOWN CASE OF TYPE 2 DIABETES MELLITUS – 1 – PRESENT, 0 – ABSENT
25. CAD – KNOWN CASE OF CORONARY ARTERY DISEASE – 1 – PRESENT, 0 – ABSENT
26. ALCOHOL – HISTORY OF HEAVY ALCOHOL CONSUMPTION – 1 – PRESENT, 0 – ABSENT
27. SMOKER – CURRENT SMOKER – 1 – YES, 0 – NO
28. SBP – SYSTOLIC BLOOD PRESSURE IN MMHG

29. DBP – DIASTOLIC BLOOD PRESSURE IN MMHG
30. PR – PULSE RATE PER MINUTE
31. CNS – ABNORMAL COMPLETE CNS EXAMINATION –
    1 – ABNORMAL 0 – NORMAL
32. HMF – HIGHER MENTAL FUNCTIONS – 1 – ABNORMAL
    0 – NORMAL
33. NYSTAGMUS - 1 – PRESENT, 0 – ABSENT
34. ATAXIA – PRESENT, 0 – ABSENT
35. UMN – UPPER MOTOR SIGNS ON EXAMINATION 1 – PRESENT, 0 –
    ABSENT
36. HB – HEMOGLOBIN IN G/DL
37. BLOOD UREA – IN MG/DL
38. S.CR – SERUM CREATININE IN MG/DL
39. RBS – RANDOM BLOOD SUGAR IN MG/DL
40. ECG – 1 – ABNORMAL 0 – NORMAL
41. CT BRAIN – 1 – ABNORMAL 0 – NORMAL

S.NO	NAME	SEX	AGE	IPNUMBER	ADDRESS	DIZZINESS	LOC	FALL	SEIZURE	HEADACHE	VOMITING	ALTEREDMENTALSTATUS	MOTORDEFICIT	SENSORYDEFICIT	LANGUAGE DISTURBANCE	CN	VISUALDISTURBANCE	TREMORS	TINNITUS	NECKPAIN
1	ILANGESHWARAN	M	29	38000	PATTUKOTAI	6.0	0	0	0	0	0	0	0	0	0	0	0	0	0	1
2	PERIYANAYAKI	F	55	36998	PUDUKOTTAI	1.0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
3	MAHESHWARI	F	41	40700	THANJAVUR	1.0	0	0	0	1	0	0	0	0	0	0	0	0	1	0
4	MARIKANNU	F	65	44471	PUDUKOTTAI	14.0	0	0	0	0	1	0	0	0	0	0	0	0	0	0
5	YAGAPPAN	M	42	44398	PUDUKOTTAI	3.0	0	0	0	0	0	0	0	0	0	1	1	0	0	0
6	DURAIKANNU	M	51	44606	THIRUVARUR	1.0	0	0	0	0	1	0	0	0	0	0	0	0	0	0
7	SUNDARAJAN	M	60	43680	THIRUVARUR	1.0	0	0	0	1	0	0	0	0	0	0	0	0	0	0
8	RAJENDRAN	M	55	44770	ARIYALUR	14.0	0	0	0	0	0	0	0	0	1	0	0	0	0	0
9	SUBRAMANIYAN	M	45	44737	NAGAPATTINAM	1.0	1	0	0	0	0	0	0	0	1	0	0	0	0	0
10	KARUPPAIYAN	M	55	44841	THANJAVUR	10.0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
11	RAMALINGAM	M	63	44857	THANJAVUR	1.0	0	0	0	0	0	0	0	0	1	1	0	0	0	0
12	MAHALAKSHMI	M	72	44645	THANJAVUR	1.0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
13	SUSAIMARY	F	84	43523	BOOTHALUR	0.5	0	0	0	0	0	0	0	0	0	0	0	0	0	0
14	KUTTACHI	F	70	42736	ORATHANADU	0.5	0	0	0	0	1	0	0	0	0	0	0	0	0	0
15	SEKAR	M	42	43328	THANJAVUR	1.0	0	0	0	0	0	0	0	0	0	0	1	1	0	0
16	CHINNAPILLAI	M	65	43329	ARIYALUR	1.0	1	0	0	0	0	0	0	0	0	0	0	0	0	0
17	NATARAJAN	M	62	43812	PAPANASAM	1.0	1	0	1	1	1	0	0	0	0	0	0	0	0	0
18	KALIYATHAL	F	70	42716	BOOTHALUR	0.5	0	0	0	0	1	0	0	0	0	0	0	0	1	0

S.NO	PALPITATION	CHESTDISCOMFORT	HTN	DM	CAD	ALCOHOL	SMOKER	SBP	DBP	PR	CNS	HMF	NYSTAGMUS	ATAX	UMN	HB	BLOODUREA	S.CR	CTBRAIN
1	0	0	0	0	0	1	0	130	90	90	0	0	0	0	0	11.0	23	0.7	0
2	0	0	0	0	0	0	0	126	80	84	0	0	0	0	0	9.4	24	0.7	0
3	0	0	0	0	0	0	0	110	70	86	1	0	1	0	0	9.0	26	0.9	1
4	0	1	1	0	1	0	0	180	80	86	1	0	0	1	0	10.6	53	1.5	0
5	0	0	0	0	0	1	1	100	70	86	1	0	0	0	0	12.0	40	0.9	0
6	0	0	1	0	1	1	0	110	80	106	1	0	0	0	1	8.8	110	3.1	1
7	0	0	1	0	1	1	0	170	100	80	1	0	1	1	0	11.0	30	0.8	1
8	0	0	0	0	0	1	0	110	70	88	1	0	0	0	0	12.8	34	1.0	0
9	0	0	1	1	0	0	0	180	90	86	0	0	0	0	0	10.6	30	1.0	0
10	0	0	0	0	0	0	0	100	60	100	0	0	0	0	0	9.0	44	1.6	0
11	0	0	1	1	0	1	1	150	90	86	1	0	0	0	0	7.7	57	1.2	1
12	0	0	1	1	0	0	0	190	80	88	0	0	0	0	0	11.8	28	0.7	0
13	0	0	1	0	0	0	0	120	70	80	0	0	0	0	0	8.0	45	1.0	1
14	0	0	0	0	0	1	0	170	100	70	1	1	0	0	1	8.0	29	1.0	1
15	0	0	0	0	0	1	0	110	80	86	0	0	0	0	0	12.0	39	0.8	0
16	0	0	1	1	0	1	1	140	90	82	1	1	0	0	1	8.8	54	1.0	0
17	0	0	1	0	0	1	1	180	90	70	1	1	0	0	1	9.0	32	1.0	1
18	0	0	0	1	0	0	0	140	70	70	1	1	0	0	1	8.0	50	1.0	0

S.NO	NAME	SEX	AGE	IPNUMBER	ADDRESS	DIZZINESS	LOC	FALL	SEIZURE	HEADACHE	VOMITING	ALTEREDMENTALSTATUS	MOTORDEFICIT	SENSORYDEFICIT	LANGUAGE DISTURBANCE	CN	VISUALDISTURBANCE	TREMORS	TINNITUS	NECKPAIN
19	MALAR	F	45	40445	BOOTHALUR	3.0	0	0	0	0	0	0	0	1	0	0	1	0	0	0
20	GANESAN	M	63	40633	THANJAVUR	1.0	0	0	0	0	1	0	0	0	0	0	0	0	0	0
21	NATARAJAN	M	67	39978	ARIYALUR	1.0	0	0	0	0	1	0	0	0	0	0	0	0	0	0
22	MUTHAMMAL	F	56	29708	PAPANASAM	1.0	0	0	0	0	1	0	0	0	0	0	1	0	1	1
23	JAYANTHI	F	37	32584	THANJAVUR	1.0	0	0	0	0	1	0	0	0	0	0	0	0	0	0
24	JAYAM	F	80	39624	THANJAVUR	1.0	0	0	0	0	0	0	0	0	0	0	0	0	0	1
25	DHAVAMANI	F	70	39893	ARIYALUR	2.0	0	0	0	0	0	0	0	0	1	0	0	0	0	0
26	RAJAMMAL	F	50	40658	PAPANASAM	4.0	0	1	0	0	1	0	0	0	0	0	0	0	0	0
27	PALANIYAMMAL	F	60	29741	ORATHANADU	2.0	0	0	0	0	1	0	1	0	0	0	0	0	0	0
28	JACK	F	42	30008	PAPANASAM	2.0	0	0	0	1	1	0	0	0	0	0	0	0	0	1
29	KAVERY	F	40	32264	THANJAVUR	0.5	1	0	0	1	0	0	0	0	0	0	0	0	0	0
30	SAMIYAMMAL	F	65	32416	PUDUKOTTAI	1.0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
31	GOKILAVASAN	M	50	37210	PUDUKOTTAI	1.0	0	0	0	0	1	0	0	0	0	0	0	0	0	0
32	VIJAYA	F	50	33586	THANJAVUR	1.0	0	0	0	0	0	0	1	0	0	0	0	0	0	0
33	MANGALAM	F	42	37723	ARIYALUR	2.0	0	0	0	0	1	0	0	0	0	0	0	0	0	0
34	SANKARAMMAL	F	70	37961	THANJAVUR	1.0	1	1	0	0	1	0	0	0	0	0	0	0	0	0
35	RENGANAYAKI	F	67	37032	THANJAVUR	3.0	0	0	0	1	0	0	0	0	0	0	0	0	0	0
36	JAMUNADEVI	F	40	36011	ORATHANADU	7.0	0	0	0	0	1	0	0	0	0	0	0	0	0	0
37	MARIYAMMAL	F	65	45245	THANJAVUR	1.0	1	0	0	1	0	1	0	0	1	0	0	0	0	0

S.NO	PALPITATION	CHESTDISCOMFORT	HTN	DM	CAD	ALCOHOL	SMOKER	SBP	DBP	PR	CNS	HMF	NYSTAGMUS	ATAX	UMN	HB	BLOODUREA	S.CR	RBS	PALPITATION	CHESTDISCOMFORT
19	1	0	0	1	0	0	0	100	70	84	0	0	0	0	0	7.6	64	1.3	497	1	0
20	0	0	0	1	0	0	0	110	70	88	0	0	0	0	0	11.2	52	1.5	590	0	0
21	0	0	1	1	0	1	0	150	90	80	0	0	0	0	0	9.8	39	1.0	250	0	0
22	0	0	1	0	0	0	0	180	100	82	1	0	0	1	0	8.8	57	0.9	88	0	0
23	0	0	0	0	0	0	0	100	60	84	1	0	1	1	0	8.3	26	0.7	117	0	0
24	0	0	0	0	0	0	0	130	80	60	1	0	0	1	0	10.4	30	0.6	96	0	0
25	0	0	1	0	0	0	0	170	90	64	0	0	0	0	0	8.0	36	1.0	72	0	0
26	0	0	0	0	0	0	0	100	70	62	0	0	0	0	0	11.8	31	0.9	73	0	0
27	0	0	1	1	0	0	0	100	70	106	1	0	0	0	1	10.4	112	2.5	62	0	0
28	0	0	0	0	0	0	0	120	80	70	1	0	0	1	0	8.5	25	0.6	193	0	0
29	0	0	0	0	0	0	0	140	90	86	0	0	0	0	0	8.6	23	0.8	69	0	0
30	0	0	0	0	0	0	0	100	70	72	1	0	1	1	0	8.6	24	0.6	447	0	0
31	0	0	0	0	0	1	0	110	70	74	1	0	0	1	0	7.0	44	0.8	96	0	0
32	0	0	1	1	0	0	0	180	100	78	1	0	0	0	1	9.2	29	1.0	440	0	0
33	0	0	0	0	0	0	0	100	70	76	1	0	0	1	0	12.1	25	0.7	58	0	0
34	0	0	1	1	0	0	0	140	90	76	1	0	0	1	0	12.0	26	0.7	56	0	0
35	0	0	1	0	0	0	0	150	90	48	0	0	0	0	0	7.0	48	1.5	123	0	0
36	0	0	0	0	0	0	0	140	70	88	0	0	0	0	0	8.6	33	0.7	94	0	0
37	0	0	0	1	0	0	0	140	80	46	1	0	0	1	0	10.0	39	1.0	79	0	0

S.NO	NAME	SEX	AGE	IPNUMBER	ADDRESS	DIZZINESS	LOC	FALL	SEIZURE	HEADACHE	VOMITING	ALTEREDMENTALSTATUS	MOTORDEFICIT	SENSORYDEFICIT	LANGUAGE DISTURBANCE	CN	VISUALDISTURBANCE	TREMORS	TINNITUS	NECKPAIN
38	CHITHRA	F	52	45284	BOOTHALUR	1.0	0	0	0	0	0	0	1	0	0	0	0	0	0	0
39	DHARMARAJ	M	75	44970	ORATHANADU	1.0	1	0	0	0	1	1	0	0	0	0	1	0	0	0
40	DHARMARAJ	M	39	29769	THANJAVUR	1.0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
41	DHARMALINGAM	M	75	37777	ARIYALUR	0.5	1	0	0	0	0	0	1	0	0	0	0	0	0	0
42	KALIYAPERUMAL	M	72	53596	ARIYALUR	0.5	1	0	0	0	0	0	0	0	0	0	0	0	0	0
43	TAMILAN	M	70	52108	PUDUKOTTAI	1.0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

S.NO	PALPITATION	CHESTDISCOMFORT	HTN	DM	CAD	ALCOHOL	SMOKER	SBP	DBP	PR	CNS	HMF	NYSTAGMUS	ATAX	UMN	HB	BLOODUREA	S.CR	RBS	ECG	CTBRAIN
38	0	0	0	1	0	0	0	180	120	60	1	0	0	0	1	9.6	40	0.9	194	1	1
39	0	0	1	1	0	0	1	200	120	80	1	0	0	0	0	9.0	28	0.9	116	1	1
40	0	0	0	0	0	1	0	140	90	86	1	0	0	1	0	11.0	40	0.9	120	0	0
41	0	0	1	1	0	1	0	180	100	70	1	0	0	0	1	9.0	40	1.0	112	0	1
42	0	0	1	0	0	0	0	150	100	86	0	0	0	0	0	11.0	32	0.9	112	0	0
43	0	0	0	0	0	0	0	150	80	76	0	0	0	0	0	11.0	32	0.6	140	0	0