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Radio Diagnosis

TO EVALUATE THE DIAGNOSTIC ACCURACY OF COMPUTED TOMOGRAPHY OVER MAGNETIC RESONANCE IMAGING IN THE EVALUATION OF ACUTE AND SUBACUTE ISCHEMIC STOKE

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ABSTRACT

AIM &OBJECTIVES:

To calculate sensitivityof Computed tomography with diffusion weighted magnetic resonance imaging as the gold standard in acute ischaemic stroke in adult patients admitted with clinically diagnosed acute CVA .

To find out the positive predictive values of Computed tomography in acute/subacute ischemic stroke.

METHODS

210 patients clinically diagnosed as acute/subacute ischemic stroke, referred to department of Radio Diagnosis were included in the study and have undergone Computed tomography and magnetic resonance imaging.

Initial collection of patient details using prepared proforma and clinical history assessment to be done to know the onset of symptoms.Further Computed tomography imaging and MR imaging is done.

RESULTS AND CONCLUSION

Total of 210patients with suspected stroke were taken for the study. Out of the 171 patients were true positive cases according to DWMR images. CT and MRI performed without any delay after onset of ischemic stroke resulted in significant differences in diagnostic accuracy. We got 89% sensitivity of CT over DWMRI in hyperacute phase, 83% in acute and 76% in subacute phases. Positive predictive value was low in hyperacute phase of ischemic stroke, where as it was 83% & 80% in acute and subacute phases respectively.

KEYWORDS : Magnetic Resonance Imaging, Computed Tomography, Diffusion Weighted Images, Acute Ischemic Stroke,

INTRODUCTION

Through the long journey of ages, understanding the human brain and its function was the fundamental desire for all the philosophers. As one of the leading worldwide cause to death and disability, the burden of stroke is felt physically, mentally, socially, economically and emotionally on patients as well as their families and health care services (1).

Stroke is a clinical designation which refers to the sudden neurological deficit which results from a cerebrovascular event.

It is divided into two broad categories: (2).

1.Ischemic stroke (80%)

2.Hemorrhagic stroke (15-20%)

Ischemic strokes can be furtherclassified as:

a)early hyperacute, a stroke that is 0–6 hours old;

b)late hyperacute, a stroke that is 6–24 hours old;

c)acute, 24 hours to 7 days;

d)subacute, 1–3 weeks;

e)chronic, more than 3 weeks old (2,3)

Today,ischemic stroke is more often disabling rather than fatal but remains the most common life threatening neurological disorder(4). Around 15% of the patients die within 3 months and rest have neurological deficits within 6 months of period(3,4).

The remaining 20% of stroke cases are due hemorrhage, especially due to intracerebral or subarachnoid hemorrhage which are also potentially devastating (5).

The imaging algorithm for evaluation of a patient who arrives with sudden onset of neurological symptoms of acute stroke begins with a non-contrast CT (NCCT) which is fast, inexpensive and readily available(6,7).

CT is the initial investigation done in a primary care delivery system to diagnose acute stroke and to exclude as because most hospitals have 24-hour access to CT, it has remained that way despite evidence showing MRI is more sensitive at picking up acute ischemic changes(8,9).

DWI appears to be sensitive to an early pathophysiological process in cerebral infarction, the loss of ATP, which causes a sudden shift of water from the extracellular space to the intracellular space. It has been shown to have a high sensitivity and specificity(10-12).

.To avoid any bias against CT resulting from delay after symptom onset, we prospectively randomized our patients to both imaging modalities , initially by NECT followed by MRI images.

We retrospectively studied 210 patients whowere referred from other departments with clinical suspicion of stroke to our department of radio diagnosis in DRSMCSI medical college for imaging. Out of which 19 cases were non ischemic cases like hemorrhagic stroke, tumors.

171 patients were diagnosed to have ischemia by DWMR imaging which is the gold standard for diagnosing ischemiacases. Out of 171 DW positive cases, 105 showed atleast one sign of ischemia on NECT.

We evaluated the sensitivity and accuracy of CT over MRI in various phases of stroke and to see the accuracy of early signs of infarction detectable by NECT ,ie 1) loss of insular ribbon sign,2)obliteration of lentiform nucleus, 3)loss of grey white matter differentiation,4) hyperdense MCA sign and 5) hypodensity to DWI hyperintense signal (representing the restricted diffusion of water) using standardized CT and MR scanning protocols.

Although MRI is the standard tool for evaluating a stroke patient, our study aims to evolve an accurate method of evaluating a suspected stroke case in a primary care setting and to evolve a standard protocol according to stages of infarct and territorial involvement. This thesis has the potential to give a valid evidence regarding the acute ischemic protocol in a primary care delivery system.

Despite the inherent challenges and past failures, stroke imaging is rapidly evolving with enormous on-going research and global public health impact. In this study we reviewed recent cumulative evidence including evolving expert opinions and recommendation to assess the accuracy of MRI over CT in a standard setting in diagnosing acute/subacute ischemic stroke and to evaluated for better information for the clinician with the vital information which needs to proceed to the appropriate diagnosis and treatment on time.

AIMS AND OBJECTIVES:

To calculate sensitivity of Computed tomography with diffusion weighted magnetic resonance imaging as the gold standard in acute ischaemic stroke in adult patients admitted with clinically diagnosed acute CVA.

To find out the positive predictivevalue of Computed tomography imaging an acute/subacute ischemic stroke.

To evolve an ideal imaging protocol in stroke patient in a primary health care delivery system.

CONTENTS IN REVIEW OF LITERATURE
-INTRODUCTION
-PREDISPOSING FACTORS
-EPIDEMIOLOGY AND DEMOGRAPHICS
-VASCULAR ANATOMY
a)ANTERIOR CIRCULATION
b)POSTERIOR CIRCULATION
c)NORMAL VARIENTS
d)WATERSHED AREAS
-ETIOLOGY
a)EVOLUTION OF CHANGES IN ISCHEMIC STROKE.
-IMAGING MODALITIES.
a)CT IMAGING
1)CT PROTOCOL
2)CT SIGNS IN ISCHEMIC STROKE
b)MRI IMAGING
1)MRI PROTOCOL
2)IMAGING FINDINGS IN DIFFERENT MRI SEQUENCES.
c)ROLE OF DWI IN ACUTE /SUBACUTE ISCHEMIC STROKE
-TREATMENT AND PROGNOSIS

REVIEW OF LITERATURE:
Patients presenting with a sudden focal neurological deficit do not always suffer an ischemic stroke.

Approximately 5% of these patients will harbor other diseases like tumors, postictal paresis, multiple sclerosis, hemiplegic migraine, transient ischemic attacks, malingering, hypoglycemia or liver disease, where MRI is usually far more informative than CT(13-14). Another 20% of these patients will have intracerebral hemorrhage that can easily be detected either by CT which is the primary and easily available modality in day to day life.

The role of imaging is to get few answers for the questions related to imaging:
1. Evidence of Hemorrhage?
2. Arterial and territory involvement?
3. Ischemic Penumbra?
4. Core area involved? (16,17).

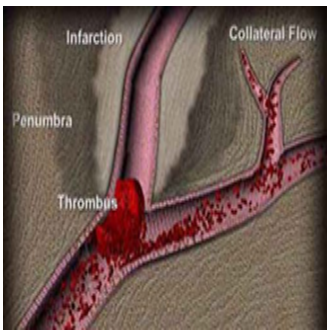


Image No 1: Schematic Diagram Showing Infarction Due To Thrombus

Computed tomography is the most common imaging modality used to assess patients with suspected stroke as it is widely available, fast, easy, and less expensive than Magnetic resonance imaging(17).

MR Imaging has now become an indispensable tool in the evaluation of ischemic strokes, especially diffusion weighted MR (DWMR) imaging is now the gold standard for evaluation of ischemic stroke patients (22).

In this study the informations which can be provided both from CT and MRI regarding stroke patients will be analyzed, and evaluated for better information for the clinician with the vital information which needs to proceed to the appropriate diagnosis and treatment in a basic setting.

PREDISPOSING FACTORS AND GENETICS
Ischemic stoke is a multifactorial disease. Some of the common predisposing factors are hypertension, diabetes, smoking, metabolic syndromes and elevated triglycerides.

Few gene disorders such as CADASIL or FABRY disease is seen associated, however no consistency have been identified till now(23).

EPIDEMIOLOGY AND DEMOGRAPHICS
Third leading cause of death and most common cause for disability in majority of the countries world wide. Incidence is 180 per 100000 per year.

In includes all the age groupsfrom new born to old age.
Children are effected mainly by cardio vascular anomalies and blood discrasias.

Patients usually present with facial droop, slurred speech, paresis, decreased consciousness or weakness in most of the cases.

For public awareness, national stroke association & stroke awareness foundation have laid an easy way to identify the symptoms of ischemic stroke- “ACT FAST” (24)

a)F- FACE: ask he patient to smile. does one side of the face droop.
b)A-ARMS: ask the patient to raise both arms. does one arm drift.
c)S-SPEECH: ask the patient to repeat a simple phrase. Is the slurring of speech?
d)T-TIME: if we see any of the above findings, should be immediately taken for treatment.

Prognosis of the patient depends upon the contributing factors such as which vessel is occluded, collateral flow, presence of ischemic penumbra.

Nearly half of the ischemic strokes have poor collateral flow and most patients with major vessel occlusion have very poor outcome unless it is intervened at the right time.

Uncontrolled or non- treated major vessel occlusion such as MCA occlusion can cause severe brain edema and herniation which leads to brain death, so called MALIGNANT MCA infarction (22).

VASCULAR ANATOMY OF BRAIN
It is very important to understand the cerebral vascular anatomy especially in stroke as it is associated with treatment and its prognosis.

Endovascular therapies require understanding of the vascular anatomy in the target area of the intervention as well as the appropriate and safe route.

The pathology and natural history of the disease and the structure of the arteries or veins supplying the ischemic area predicate the technique and intervention that must be employed during therapy.

A large proportion of strokes worldwide are caused by atherosclerosis affecting the arteries that supply blood to the brain.

All traditional vascular risk factors such as hypertension, diabetes, hypercholesterolemia and smoking are associated with atherosclerosis.

However, geometric patterns of blood flow and the differential shear stress in the arterial wall are important determinants of the localization of atherosclerosis.

The brain receives arterial blood from the bilateral internal carotid artery (ICA) and VA(25).

The ICA carries blood into the anterior cerebral circulation including the middle and anterior cerebral arteries, that is the VA carries blood into the posterior circulation including the basilar artery (BA) and posterior cerebral arteries (PCAs).

The anterior and posterior circulations communicate through the Circle of Willis via anterior and posterior communicating arteries. This circle theoretically allows cerebral perfusion to continue via collaterals in the event of trauma or disease to one or more arteries supplying that territory(25).

Broadly cerebral circulation can be divided into two:-
1)Anterior circulation.

2)Posterior circulations.

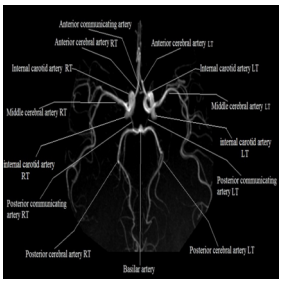


Image No 2: Image Showing Vascular Anatomy Of Brain

Anterior Circulation Consist Of:
-Anterior cerebral artery-: consist of three segments.

1)Segment A1: It originates from ACA and gives rise to medial lenticulostriate arteries which supplies the inferior parts of head of caudate nucleus and anterior limb of internal capsule.
2)Segment A2: Originates from ACA to bifurcation into pericallosal and callosomarginal artery.
3)Segment A3: Supplies the medial parts of frontal lobes, superior parts of parietal lobe and also anterior part of the corpus callosum(25).

Main Branches Are:-
a)Anterior choroidal artery.
b)Middle cerebral artery- The cortical branches of the supplies the lateral surface of the hemisphere, except for the medial part of the frontal and the parietal lobe(25).

Posterior Circulation Consist Of:
1)Posterior cerebral artery (PCA) :-
Posterior thalamoperforating arteries branch off the P1 segment to supply blood to the midbrain and thalamus.
Cortical branches of the PCA supply the inferomedial part of the temporal lobe, occipital pole, visual cortex, and splenium of the corpus callosum.

The artery also supply of hippocampus usually arise from PCA.
2)Basilar artery- Arises from the confluence of left and right vertebral arteries at the base of the pons.
3)Superior cerebellar artery- Arises from the distal basilar artery, just below the posterior cerebellar artery.
4)Anterior inferior cerebellar artery- Mostly 99% arises from the basilar artery.
5)Posterior inferior cerebellar artery- About 20% arises extracranially, inferior to foramen magnum, 10% from the basilar and occasionally loops around the tonsil (25,26).

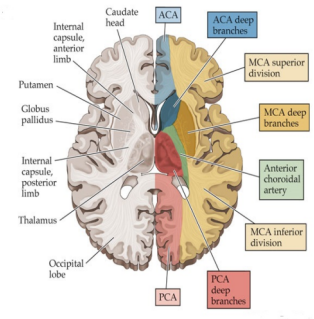


Image No 3: Image Showing Vascular Territory Of Brain

Normal Variants Of Cerebral Circulation:-
A duplication is defined as two distinct arteries with separate origins and no distal arterial convergence (27) whereas fenestration is defined as a division of the arterial lumen into distinctly separate channels, each with its own endothelial and muscularis layers, while the adventitia may be shared between the two (28).

Intracranial arterial fenestration is more common in the vertebrobasilar arteries than in the arteries of the anterior circulation (the middle cerebral, anterior cerebral and internal carotid arteries (27).

ANTERIOR CIRCULATION VARIANTS
a)Anterior Communicating Artery- Duplication of the anterior communicating artery has a prevalence of about 18%, whereas fenestration of the anterior communicating artery is present in 12%–21% of the population (29). Clinical relevance is however no significant findings associated with stroke.

b)Anterior Cerebral Artery- Fenestration of the anterior cerebral artery is a rare finding. The prevalence of fenestration of the A1 segment is between 0% and 4% in anatomic imaging studies (30).

c)Middle Cerebral Artery (MCA)duplication- Middle cerebral artery duplication occurs when a MCA branch arises above the bifurcation of the internal carotid artery. The duplicate vessel parallels the main middle cerebral artery and supplies the anterior temporal lobe (31). Most fenestrations of the middle cerebral artery are located in the proximal portion of the M1 segment.

VARIANTS OF CIRCLE OF WILLIS:-
a)Azygos Anterior Cerebral Artery- The anomaly is clinically very relevant also because in the event of anterior cerebral artery occlusion secondary to thromboembolic disease, the resultant ischemia affects both cerebral hemispheres (33,34).

b)Anterior Cerebral Artery Trifurcation-Trifurcation of the anterior cerebral artery is defined as the occurrence of three A2 segments. However no clinical relevance to secondary ischemic changes till the recent studies.

c)Bihemispheric Anterior Cerebral Artery-This anomaly is characterized by hypoplasia of one A2 segment, with the contralateral A2 segment providing the major arterial supply bilaterally to the anterior cerebral artery territory.. Occlusion of the dominant A2 segment results in ischemia of both hemispheres (34).

d)A1 Segment Absence or Hypoplasia-This is clinically relevant as in the event of thromboembolic disease, these conditions result in a diminished collateral supply and therefore an increased risk of infarction (35).

e)Absent Anterior Communicating Artery-It is a rare occurrence, however no clinical relevance.

f)Accessory Middle Cerebral Artery-An accessory middle cerebral artery is an artery that arises from the anterior cerebral artery and courses parallel to the M1 segment of the middle cerebral artery, supplying the anterior-inferior region of the frontal lobe (31).The accessory middle cerebral artery may provide collateral blood supply to the distal middle cerebral artery territory in the presence of middle cerebral artery occlusion (31).

g)Early Branching of the Middle Cerebral Artery- Early division of the M1 segment close to its origin at the internal carotid artery is a common finding and may be unilateral or bilateral (33).

h)Hyperplastic Anterior Choroidal Artery:-The prevalence of hyperplastic anterior choroidal arteries is reported to be 1.1%–2.3% (36).However its clinical relevance also very less.

POSTERIOR COMMUNICATING ARTERY ANOMALIES &NORMAL VARIANTS:-
a)Basilar and Vertebral Arteries- Basilar artery fenestration has been found in 0.6% of angiographic examinations (32). They are most commonly located in the proximal basilar trunk, close to the vertebrobasilar junction (27). The reported frequency of aneurysm formation in cases of basilar artery fenestration is 7%.

b)Posterior Cerebral Artery Fenestration ,an extremely rare occurrence, has been found in both the P1 and the P2 segments.

c)Posterior Communicating Artery &Internal Carotid Artery fenestrations are very rare compared to others. However there is increased risk of aneurysmal dilatation and its complications.

d)Fetal Origin of the Posterior Cerebral Artery- It occurs when the embryonic posterior cerebral artery fails to regress (37). Its clinical relevance in ischemic stroke is less.

e)Posterior Communicating Artery Infundibulum- It is a funnel shaped dilatation at the origin of the posterior communicating artery from the internal carotid artery.

f)Common Posterior Cerebral and Superior Cerebellar Artery Trunk- The prevalence of a common trunk of the posterior cerebral and superior cerebellar arteries is 2%–22% (34). This anomaly does not have any reported clinical significance.

g)Persistent Carotid- Basilar Artery Anastomoses-There are seven transversely oriented arteries in the cervical region. The first of these is the proatlantal intersegmental artery (38). Failure of these vessels to regress during embryonic development results in various persistent carotid-vertebrobasilar anastomoses. Occlusion of these branches may be responsible for ischemia and neuralgias (39,40)

Watershed Areas Or Zones
Watershed infarcts occur at junctional zones between major cerebral arterial territories because of hypo perfusion.

Broadly two patterns are:-
a)Cortical border zone infarct: ischemic infarctions of the cortex and adjacent subcortical white matter located at the border zone of ACA/MCA and MCA/PCA.

b)Internal border zone infarcts: These are the infarctions of the deep white matter of the centrum semiovale and corona radiata at the border zone between lenticulostriate perforators and the deep penetrating cortical branches of the MCA or at the border zone of deep white matter branches of the MCA and the ACA(41).

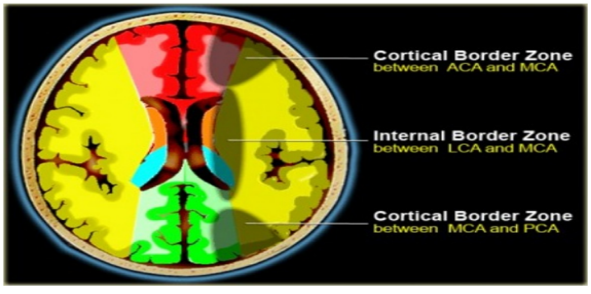


Image No 4: Image Describing Watershed Areas

ETIOGY:
Stroke etiology may been classified into 5 main groups using the TOAST classification or subsequent variations on TOAST(42,43):
- Large artery atherosclerosis
- Cardiac embolus
- Small vessel occlusion
- Stroke of other determined etiology
- Stroke of undetermined etiology.

Most common site of ischemic infarction area involves the MCA territory, followed by PCA and vertebra basilar locations.

ACA is the less commonly effected area compared to other sites.
Almost two million neurons are lost when a major vessel is occluded suddenly. The center of the ischemic area is known as the ischemic core.

Typically the CBF will be very less (<6-8 cm^2/100g/min) in the core area compared to other sites. Oxygen rapidly depletes, cellular energy production fails and thus ion hemostasis lost (43,44).

Neuronal infarctions are irreversible in the core of the stroke. Part of ischemic area around the core, which is at high risk of infarction is called penumbra, which is salvageable if re-perfused on time (44).

The penumbra shows moderate decrease in cerebral blood flow time, increased mean transit time and normal or reduced cerebral blood volume. These areas are at high risk if not intervened on right time.

EVOLUTION OF CHANGES IN ACUTE/SUBACUTE ISCHEMIC STROKE
Cerebral ischemic changes occurs when the cerebral blood flow threshold goes below and thus results in disruption of energy metabolism with subsequent failure of ion pumps and anoxic cell membrane depolarization(45,46). The evolution takes place in a systematic step, which are described as follows:-
·**Sodium-Potassium(Na+-K+)pump interruption:**
Usually in a normal brain cell, 3 Na+ ions are pumped out of the brain cell and 2 K+ ions are pumped into maintain the cells potential(46).

The activity of the ion pumps (Na-K ATPase pump) is ATP dependent.

As local CBF drops below a critical threshold, the energy supplied to the brain cells becomes inadequate for its action.

Ischemic stroke causes passive diffusion of Na+ ions (Sodium influx) into the cells which leads to a rapid accumulation of Sodium in the intracellular space, therefore disrupting the equilibrium between fluid concentrations in and out of the cells(46,47).

Thus the activity of this pump fails and the cells begin to swell. The membrane permeability increases and water shifts from the extracellular space to the intracellular space.

This resulting in cytotoxic edema without causing a net increase in brain volume or brain fluid .Thereafter brain tissue swells up and volume increases(48,49).

Calcium (Ca2+) pump interruption:
In the normal brain tissue, when neurons are at rest, they are polarized (negative inside and positive outside).

When enough positive Ca2+ ions cross the cell membrane, a change in the negative/positive membrane charge takes place called depolarization takes place. In the ischemic tissue, calcium channels can stay open for a long time which increases Ca2+ ions influx into the cells causing slow intracellular walls damage and ultimately causing cell necrosis and apoptosis(50,51).

Blood brain barrier (BBB) breakdown:
Ischemic damage of the capillary endothelial cells that form the BBB allows water to come out of the vessels and accumulate in the extracellular space.

This also contributes to the appearance of the vasogenic edema which causes an absolute increase in the net volume of water in the ischemic tissue. This also causes edema in the brain tissues(45-51).

IMAGING MODALITIES
The initial step in approaching the imaging is to summarize the targeted clinical goals for patients with suspected acute ischemic stroke in the acute care setting.

The fundamental objective of treatment is to enable rapid reperfusion for maximal tissue salvation. There is substantial evidence to suggest efficacy of intravenous thrombolytic therapy in the first 4.5 hours from onset of symptoms as well as increased risk of hemorrhagic complications and lower efficacy outside the therapeutic window(65).

The European Cooperative Acute Stroke Study (ECASS) investigators demonstrated the efficacy of treatment instituted within the first 4.5hours(65).This was confirmed on a systematic review with pooled data from 11 randomized controlled trials evaluating intravenous thrombolysis (IVT) and 3 randomized controlled trials evaluating intra-arterial thrombolysis(IAT). This review concluded efficacy of IVT within 4.5 hours of onset of symptoms, beyond which the risk of treatment outweighed the benefit(65,66).

Ischemic stroke imaging can be done using either computed tomography (CT) or magnetic resonance imaging (MRI). The choice is based mostly on the available infrastructure. CT is the imaging workhorse available in most hospitals on a round-the-clock basis. Use of CT imaging can readily exclude the presence of an acute cerebral hemorrhage which is very important for treatment purpose. The recent technical advancements in CT and its speed confer additional advantages to this technique(77).

However, there is limitation of NECT in core characterization due to multitude of factors including inherently low sensitivity to early

ischemic changes. However MR with DWI is the imaging gold standard for an acute setting. In an ideal world with no cost, availability or individual applicability issues, MR protocol for acute stroke imaging would be more preferred from the view point of obtaining accurate and consistent tissue specific information with minimum susceptibility to post processing variability (68).

If MRI is not available and decision for endovascular therapy has to been taken based on the initial NECT, then immediate CTA (to assess vessel occlusion and collateral status) and CTP (infarct core assessment on CBF map) should be considered for perfect management (69).

CT IMAGING FINDINGS IN ACUTE/SUBACUTE ISCHEMIC STROKE:-

CT is the mostly used imaging modality in the present era as it is widely available, fast & no IV contrast is needed for acute emergency purposes in a primary care setting.

The most widely used to confirm diagnosis of ischemic stroke and to exclude other mimics such as intracerebral haemorrhage, subdural haematoma and tumour.

CT is very sensitive for detection of haemorrhage which alters the treatment as it is ccontraindication to thrombolytic therapy. Can identify early stage acute ischemiadue to increased water content in infarcted area (66).

Hypodense foci on CT early after symptom onset continue to appear the same on follow up CT scans, emphasizing the specificity of hypodensity for infarct core(66,67).

While CT-based hypodensity is highly specific for the infarct core, CT has reduced sensitivity for detecting early ischemia.

ACUTE ISCHEMIA CT PROTOCOL

For early detection of stroke, neuroradiology section, in a consensus symposium led by GilGonzalez, developed the following acute stroke imaging algorithm delineating the imaging evaluation that we consider to be essential and sufficient for determining stroke treatment eligibility(68).

A)Unenhanced head CT to exclude hemorrhage
B)Head and neck CTA, performed immediately following head CT (while the IV-TPA is being mixed, so as not to slow thrombolysis administration)
a) Axial, coronal, and sagittal thick-slab maximum intensity projections reviewed in real-time at the scanner console.
b) If MR imaging is contraindicated and endovascular therapy will not immediately to be performed, CT perfusion imaging should be considered (58,69).
C)Next imaging of choice is MR DW images-
a)If large-vessel occlusion is present and infarct core is <70–100 mL, proceed immediately to endovascular treatment.
b) If the patient is not an endovascular candidate, MR perfusion imaging should be considered(69).

Focal swelling (seen as loss of normal cortical sulcal pattern without hypodensity) is considered to reflect the early ischemic changes which are not irreversible and might have increased blood volume suggesting an auto regulation to the same site, consistent with penumbra (69).

Detection of focal swelling and hypodensity may not be possible due to low sensitivity for CT to demonstrate the changes, although hypodensity and focal swelling have high specificity for infarct core and penumbra respectively when its been identified .

Alberta stroke program early CT score (ASPECTS) is a ten point quantitative topographic CT scan score which is seen in patients with MCA stroke. Segmental assessment of the MCA territory is made by deducting one point each from the score ten for every region effected with ischemic stroke .ie- caudate, putamen, internal capsule, insular cortex and M1 to M6 branches. An ASPECT score less than or equal to 7 predicts a worse outcome at three months as well as a symptomatic hemorrhage. According to studies done by R.I AVIV et al, patients with score less than 8 treated with thrombolysis dint have a good outcome (70).

Direct Signs In Acute Ischemic Stroke Are:

1)Insular ribbon sign:
The insular cortex is particularly vulnerable to a proximal middle cerebral artery (MCA) occlusion because it is the region most distal from the potential anterior and posterior collateral circulation, and therefore it is a watershed arterial zone.

When ischemic, the insular region shows loss of definition of the gray-white interface, or loss of the insular ribbon is seen. [71-74].

2)Obscuration of the lentiform nucleus:
Due to its blood supply via end-arteries, the basal ganglia are also particularly vulnerable to early infarction [75].
When ischemic takes place, an obscured outline or partial disappearance of the lentiform nucleus can be seen on NECT.

3)Presence of hyperdense vessels due to clot:
This is an indirect sign may help confirm the diagnosis in the absence of hypodensity and swelling, although the sensitivity for this sign is very low (30%)(59). Specificity is generally high, however false positive hyperdense vessels can occur, but to differentiate between these two is a real challenge.

Comparison to the normal cerebral hemisphere and measurement of HU intensity can be used to confirm if the vessel is with suspicious Hyperdensity (75,76).

Hyperdense vessels have been associated with poor outcomes after stroke, but their presence only suggests an occlusion but not the severity(59,60).

Disappearance of the sign at follow up CT is considered a good sign of improvement as it a marker for recanalization(77).

Hyperdensity in cortical M2 / M3 segments in sylvian fissure is called as Sylvian dot sign. It has low sensitivity and high. Proximal clot termed the hyperintense carotid artery sign (78-80).

The presence of hyperdense thrombus on NECT can be visualised more readily using thin slice acquisitions where clot contrasts surrounding structures more clearly due to reduced volume averaging effects than thicker slice CT(81,82).

Thrombus assessment with thin slice NECT is important in assessing response to thrombolysis and could help select patients for intra-arterial therapy(84).

However clot composition may affect the likelihood of a hyperdense vessel sign being visualized, with low amounts of fibrin content which shows a reduced clot density appearance(85).

High hematocrit level which may be associated hyperdense vessels in the absence of thrombus might be seen, however that appearances should be bilateral if due to elevated hematocrit alone (85,86).

MRI EVALUATION IN ACUTE/SUBACUTE ISCHEMIC STROKE:-

MRI is more sensitive and specific than CT in the first few hours after stroke.

Newer and latest sequences are playing a vital role in diagnosis and thereby treatment.

The method was introduced into clinical practice in the middle 1990s, but because of its demanding MR engineering requirements—primarily high-performance magnetic field gradients—it has only recently undergone widespread dissemination.

The primary application of DW MR imaging has been in brain imaging, mainly because of its high sensitivity to ischemic stroke, a common condition that appears in the differential diagnosis in virtually all patients who present with a neurologic complaint.

It is very much sensitive and specific in detection of acute/subacute ischemic stroke and also for differentiation of acute ischemic stroke from other processes that manifest sudden onset of neurologic deficits(69).

MR Imaging Protocol For Stroke: (70)
1)T1 weighted images
a)plane: sagittal (or volumetric 3D)
b)sequence: fast-spin echo (T1 FSE) or gradient(T1 MPRAGE)
c)The main purpose is for anatomical evaluation of brain. Cortical laminar necrosis or pseudo laminar necrosis may be seen as a ribbon of intrinsic high T1 signal, usually after 2 weeks, but can be seenmuch earlier also.

2) T2 weighted
A) plane: axial.
B)sequence: T2 FSE
C)The main purpose is - loss of normal signal void in large arteries may be visible immediately after the vent occurred.
D) 6-12 hours after the infarction, tissue becomes high signal. sulcal effacement and mass effect develop and become maximal in the first few days of infarction.

3) FLAIR
a) plane: axial
b)sequence: FLAIR
c) purpose: After 6-12 hours infarcted tissue becomes high signal in the ischemic sites. Sulcal effacement and mass effect develop and become maximal in the first few days of infarction.

4) Diffusion-weighted imaging (DWI)
a)plane: axial
b)sequence: DWI: B=0, B=1000 and ADC
c)purpose: early identification of ischemic stroke: diffusion restriction may be seen within minutes following the onset of ischemia, especially in the brain stem where it is usually missed on NECT. It correlates well with infarct corediffferentiation of acute from chronic stroke

5) Susceptibility weighted imaging (SWI)
a)plane: axial
b)sequence: susceptibility weighted imaging or T2*
c)purpose: highly sensitive in the detection of hemorrhage.

6) MRA
a)MR Angiography is an alternative to conventional angiography and CT angiography
b)Purpose :It can be utilized to assess vascular structures of almost any part of the body (70).

MRI Findings In Acute/subacute Cerebral Ischemia Are:
T2 and Fluid Attenuated Inversion Recovery (FLAIR) imaging—
On T2-weighted and FLAIR images, ischemic infarction appears hyperintense lesion usually seen within the first 3–8 hours after stroke onset [71,72).

MRI done within 6 hours of onset of symptoms, patients without a visible hyperintense lesion on FLAIR images mostly have greater probability of being imaged within the first 3 hours of symptom onset in DWMR(72).

Thus, a mismatch between positive DWMR and negative FLAIR images appears to be useful in the identification of patients who are likely to benefit from thrombolysis [71].

FLAIR images are very highly sensitive to subarachnoid hemorrhage [73] as well as acute cerebral venous sinus thrombosis.
In hyperacute stroke, T2-weighted images can be useful to detect the loss of the arterial signal flow void in occluded vessels within minutes of the onset of acute stroke [72,73].

Diffusion-Weighted Imaging (DWI) and Apparent Diffusion Coefficient (ADC)
Diffusion magnetic resonance imaging provides image contrast that is dependent on the molecular motion of water [75,76].

As mentioned, cerebral ischemia leads to energy metabolism disruption with the failure of the Na+/K+ and other ionic pumps. This induces a loss of ionic gradients and a net transfer of water from the extracellular to the intracellular compartment causing a cytotoxic edema [29,30].

Excessive intracellular water accumulation leads to a reduced extracellular volume, where water mobility is relatively facilitated. Therefore occurs a reduction of water diffusion in the extracellular space [77,78].

This phenomenon is detected with DWMR within minutes of vessel occlusion [79] and can be measured quantitatively with the ADC.

Patients with a diffusion images taken within 3 hours of symptom onset may still help in finding salvageable tissue at risk and might benefit from thrombolysis(79).

Gradient-recalled echo (T2*) weighted imaging
Hyperacute stroke imaging demands the differentiation between ischemic stroke and hemorrhagic stroke, which is impossible by clinical methordsonly(80).

Although NECT is sensitive and the standard method for the diagnosis of ICH, hyperacute ICH can be identified on MR (mainly FLAIR and gradient-recalled echo imaging) with excellent accuracy [81].

However microbleeds (small hemosiderin deposits) wont be much evident on CT, but can be detected by T2*-weighted images [75].

These chronic lesions are associated with an increased risk of spontaneous ICH and may also be a risk factor for thrombolysis-related hemorrhage [84].

In a suspected acute stroke, T2*-weighted images can detect an intraluminal thrombus as a linear low signal region (signal void) of magnetic susceptibility [84].

ROLE OF DWI MR IN ACUTE/SUBACUTE ISCHEMIC STROKE
DWI MR sequences are highly sensitive in detecting early cerebral ischemic changes in acute stroke. They are fast to acquire and do not necessitate injection of a contrast agent.

The investigation of diffusion weighted imaging in neurological disorders was introduced in 1986 [Le Bihan 1986].

The MR acquisition of the DW signal is similar to that of conventional T1-w and T2-w MR images,where protons in water tissue will be excited using a large magnet and aligned with the direction of the applied magnetic field(85,86).

Their relaxation times T1 and T2 during which they come back to a state of equilibrium encode the MR intensity signal of every voxel.

However it is made sensitive to water motion in scanned tissue by applying a non-homogeneous magnetic field: two gradient pulses with same magnitude but in opposite directions–one to dephase the proton spins and the second one is to rephrase them(87).

If water molecules are in some areas where they are freely mobile between the two pulses then rephasing process will not bring them back to the exact initial state. So therefore it results in a decrease of the acquired DWI signal as the relaxation takes longer(88).

This idea of double-gradient proton excitation process was introduced by Stejskal and Tanner in [Stejskal 1965]

Conventional MR sequences and CT images do not show early changes of ischemic stroke until 6-12 h.

However, diffusion weighted MR imaging (DWI) can detect an infarct within 1-2 h because ischemic tissue exhibits reduced diffusion, which is observed within about half an hour of ischemia, which is an important factor in treatment and prognosis(89,90).

On DW MRI, a proton of water molecule experiences slightly different field strength because of local inhomogeneity in the magnetic field, dependent on its spatial location.

Diffusion-weighted images are obtained by adding a series of two gradient pulses, which are applied symmetrically with respect to 180 radio frequency pulse.

The first gradient pulse is applied between the 90 pulse and the 180 radio frequency (RF) pulse.

Even small microscopic motion after this pulse causes molecules to acquire phase shift relative to their transverse magnetization. Both 180 pulse and the second gradient pulse rephase stationary spins(89-92).

he technique most commonly used to acquire the DWI is an ultrafast one, echo-planar imaging (EPI); this technique decreases scanning time significantly and eliminates movement artifacts and imaging time ranges from a few seconds to two minutes.

High signal intensity on DWMR and hypointensity on apparent diffusion co-efficient (ADC) images, which are definitive features of acute cerebral infarction, however there are diverse conditions as hemorrhage, abscess, tumor and even in Wernicke's encephalopathy in which we are able to see such features, but differentiating between these are crucial for appropriate treatment(90-93).

Decrease in ADC of the ischemic brain tissue has been shown to coincide with the onset of this cytotoxic edema and this area is seen as area of hyperintensity in the DWI and hypointensity in ADC images.

As the time progresses, ischemic process progresses, cells lysis and the macrophage activity increases leading to an evolution towards vasogenic edema, observable more in the T2 weighted images(94).

During the transmission of hyperacute to chronic stage of ischemic stroke, there are dramatic changes occurring in the ADC values.

Following reduction in the acute phase the ADC values re-normalize several days (approximately 7-10 days) after stroke onset and then increase there after. Thereby these values make a dramatic change in the field of diagnosis and there by treatment of ischemic stroke(96,97).

Early reperfusion may cause renormalization of the reduced ADC at a much earlier time point as early as 1 to 2 days in human who have received the tissue plasminogen activator (RTPA) administered within 3 hours of stroke onset(96).

At times we get false positive results in conditions such as edema,mets ,partial volume overloading,that we often misinterpret as ischemic stroke in Computed tomography,diffusion weighted magnetic resonance imaging gives clear cut image of these results and turn out to be negative for ischemic strokes.

Magnetic resonance imaging also remains non-ionizing, which is of prime importance if one considers patients that require frequent follow-ups.

This is why, even in patients who have had an initial Computed tomography, magnetic resonance imaging is to be preferred for follow-up(97,98).

False positive DWI lesions with restricted diffusion can be seen in cerebral abscess (due to high viscosity) and in tumor (due to dense cellularity).

Differentiation can be made by reviewing other sequences and post contrast images.

DWI can help in differentiation of stroke sub-types (Lacunar vs Embolic) which may be necessary in deciding the patient management.

Lacunar strokes remain a relative contraindication to thrombolytic therapy because risk of intracranial hemorrhage with rTPA use (99,100).

TREATMENT

Stroke treatment is still one of the most challenging processes since it needs to be determined within the first few hours after onset(101).
The ultimate goal of any stroke treatment is to rapidly restore the cerebral blood flow as early as possible in the affected brain cells. Although thrombus is dissolved by the body's natural fibrinolysis systems but it rarely occurs to prevent tissue damage(102).

The most important factor in a successful intervention is selection of patient, with the two important considerations-
1) Time from the symptoms of onset,
2) Imaging findings(103).

This can be achieved in many ways- 1) thrombectomy or 2) by usingdrugs –thrombolysis, 3) Intracranial Angioplasty and Stenting. (103,104)

Thrombectomy is the removal by interventional neuroradiological methods of blood clots in the cerebral arteries. There are several methods for doing this but none have yet proved to be more effective than intravenous thrombolysis in randomized trials(88).

Thrombolysis rapidly recanalizes the occluded artery and improves the chances for a good neurological outcome in acute ischemic stroke using recombinant tissue plasminogen activator (rTPA).

When rTPA is given within the first 4.5, possibly up to six hours after stroke, this improves functional outcome. However, thrombolysis suffers from the hazard of increasing serious brain hemorrhage in few of the cases.

Ischemic stroke treatment is an very essential as it make very drastic change in the outcome if intervened earlier(103).

Intravenous tissue plasminogen activator (rTPA) is used in patients in “golden hours”, that is less than three hours from ictus.

Intra arterial thrombolysis is typically not done in less than six hours except in basilar artery thrombosis and patients with more than six hours.

Intra arterial thrombolysis is done in cases involving less than one third of the MCA territory involvement.

Intra-arterial rTPA and other clot dissolving drugs such as desmoteplase have a good outcome recently in selected cases.

Endovascular mechanical thrombectomy is an alternative and potentially synergistic method for thrombolysis(104).

Intracranial Angioplasty and Stenting

Maximal medical therapy is the treatment of choice in patients with substantial cerebrovascular atherosclerotic lesions, as evidenced by the Stenting and Aggressive Medical Management for Preventing Recurrent Stroke in Intracranial Stenosis (SAMMPRIS) trial(104)

In patients with more than 70% symptomatic atherosclerotic lesions in whom optimal medical therapy is ineffective, revascularization with angioplasty or stenting is a feasible option .

Emergent stenting is becoming increasingly popular; for this use, only one system (Wingspan Stent System with Gateway PTA Balloon Catheter is been in use(105).

MATERIALS AND METHODS

This study is a prospective one with a sample size of 210 patients. All Patients referred with clinical diagnosis of CVA in the department of Radio Diagnosis.

Initially CT was taken followed by MRI stroke protocol for all the 210 patients using Siemens 1.5 Tesla MAGNETOM AVANTO Tim (32x8), (after getting scientific and ethical clearance from the institutional research board).

CT TECHNIQUE USED

Plain CT
Axial images :KVP- 130,MAS- 270, axial thickness- 4.8mm & 1.2mm.,scan time-1.5sec,no of images- 36, radiation dose- 36.4ms.
We used the following criteria for diagnosing acute and subacute ischemic stroke
e)Obliteration of lentiform nucleus
f)Loss of grey white matter differentiation
g)Dense MCA sign.
h)Insular ribbon sign

i)hypodensity

MR TECHNIQUE USED

The sequences used were:
Axial T2W:TR 3600ms, TE 91ms, FOV-230mm, slice thickness 5mm, no of slices 20
Axial T1W:TR 400ms, TE 87ms, FOV-230mm, slice thickness 5mm, no of slices 20
Coronal T2W:TR 3460ms, TE 93ms, FOV- 230mm, slice thickness- 5mm ,slices - 20
FLAIR AXIAL :TR 9000, TE 90, FOV-230,Slice thickness – 5mm, slices -20
DWI AXIAL :TR 3400, TE 102, FOV-230,Slice thickness – 5mm, slices -19
GRE AXIAL: TR 780,TE 26,FOV- 230, Slice thickness- 5mm, slices- 20
SAG T2: TR 3460,TE 93,FOV- 230, Slice thickness- 5mm, slices- 20

The MRI images were reviewed under the guide and the findings were documented in the proforma. The presence or absence of each direct signs of acute/subacute ischemic stroke was noted for all patients examined. Based on this a final MRI diagnosis of acute/subacute ischemic stroke was assigned for each patient.The results were analyzed using various statistical tests.

INCLUSION CRITERIA:

All adult patients with clinical diagnosis of acute and subacute onset of ischemic stroke who have undergone Computed tomography andmagnetic resonance imaging in the Department of Radio diagnosis.

EXCLUSION CRITERIA

I.Patients with MR incompatible devices or implants.
ii.Patients with claustrophobia
iii.Patients on life support systems

SAMPLE SIZE

210 patients referred from clinical departments, with clinical diagnosis of stroke, undergoing CT &MRI evaluation were included in the study. And 171 patients were diagnosed to have acute/subacute ischemic stroke with our imaging techniques.

ANALYSIS PLAN

Data collected was entered into MS excel spreadsheet. Analysis was conducted using statistical package, SPSS (Statistical Package for Social Sciences). Tests of diagnostic accuracy and percentage & proportions were applied to assess the outcome of the study.

This study strictly confines to ethics and was done after receiving full consent and co-operation from the patients.

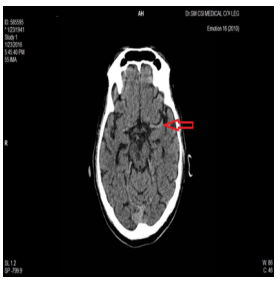


Image No5: Axial section NECT of brain showing dense MCA vessel on the left

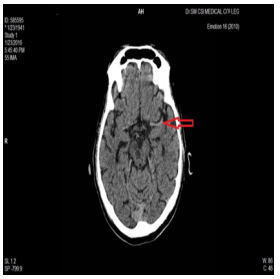
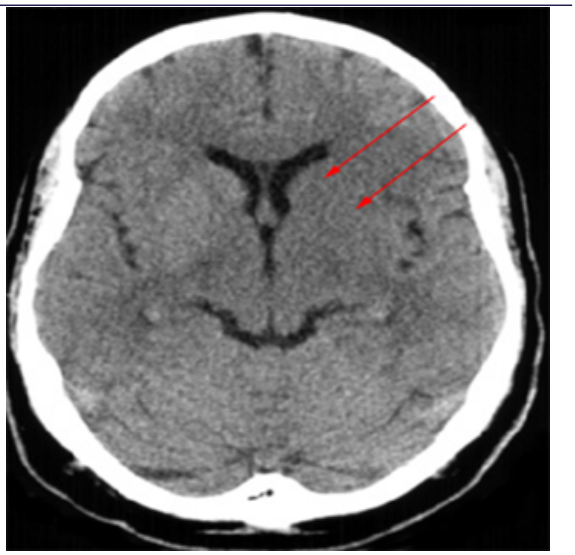
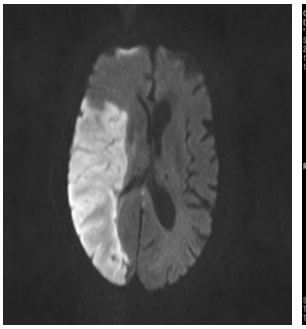


Image No 6: Axial section NECT of brain showing loss of insular ribbon sign



ImageNo 7: Axial section NECT of brain showing obliteration of lentiform nucleus



ImageNo 8a: Axial section MRDW image of brain showing restricted diffusion in the right MCA territory

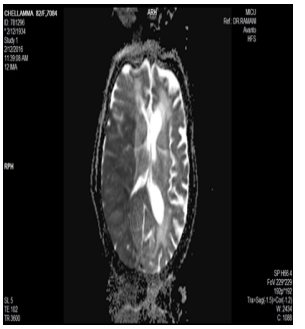


Image 8b: Corresponding ADC image showing hypointensity suggestive of acute ischemia

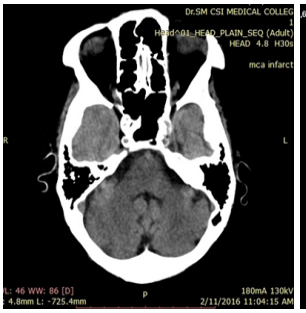
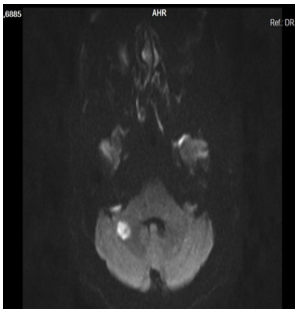


Image No9A: Axial section NECT image showing a normal cerebellum



ImageNo9B: Currespoding axial DWMR image showing an area of restricted diffusion suggestive of acute infarct

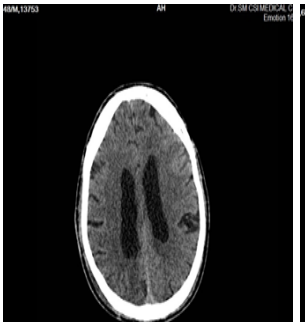


Image No10A: Axial section NECT image at the level of corona radiata showing a normal parenchyma

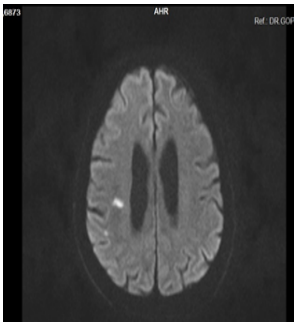


Image No10B: Corresponding axial DWMR image showing an area of restricted diffusion suggestive of acute infarct.

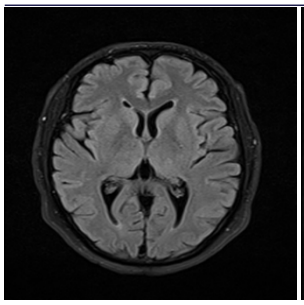


Image No11A: Axial section FLAIR image showing a normal parenchyma

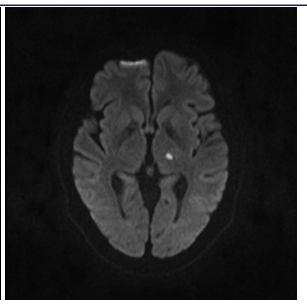


Image No 11B: Corresponding axial DWMR image showing an area of restricted diffusion in the right thalamus suggestive of acute infarct.

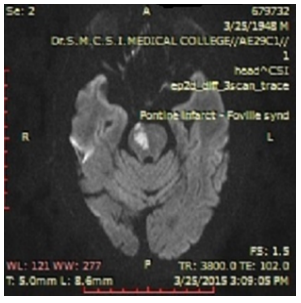


Image No 12a:DWMR image showing an area of restricted diffusion in the infero-medial pontine (Foville) syndrome.

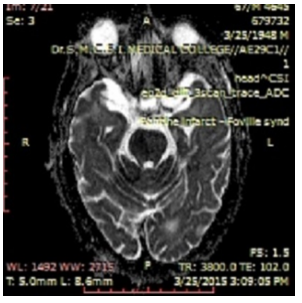


Image No 12B: Corresponding A D C i m a g e s h o w i n g hypointensitysuggestive of acute ischemia.

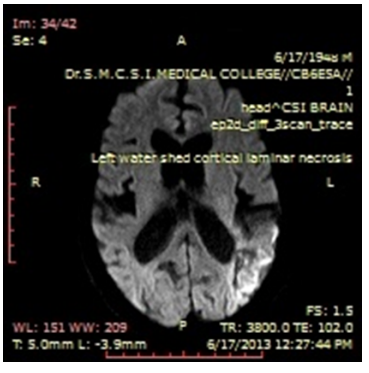
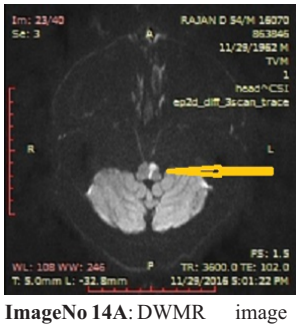


Image13 : Axial section of MRI DW images showing left cortical watershed area infarct.



ImageNo 14A: DWMR image showing an area of restricted diffusion in the medial medulla-(A case of medial medullary syndrome).

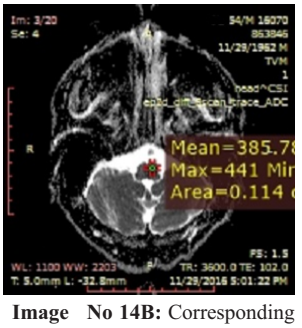


Image No 14B: Corresponding image shows low ADC image suggestive of acute ischemia.

RESULTS

Table No:1- Percentage Of Distribution According To Gender In Clinically Suspected Cva

GENDER	NUMBER	PERCENTAGE
MALE	119	56
FEMALE	91	44
TOTAL	210	100

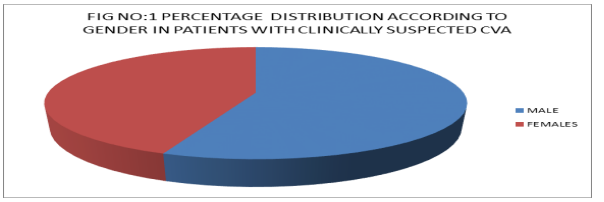
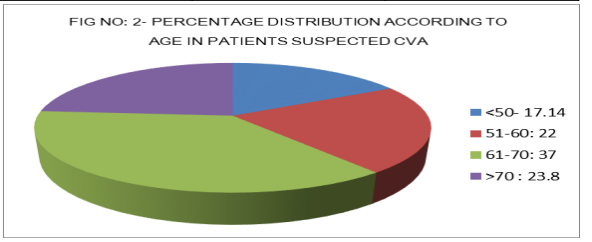


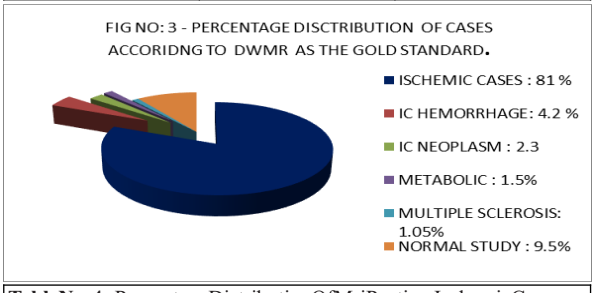
Table No: 2-Percentage Distribution According To Age In Patients Suspected Cva

AGEINYEARS	NUMBEROFCASES	PERCENTAGE
<50	36	17.14
51-60	46	22
61-70	78	37
>70	50	23.8
TOTAL	210	100



TableNo:3-Percentage Distribution Of Cases Accoridng To Dwmr As The Gold Standard.

DWMR DIAGNOSIS	NUMBER	PERCENTAGE
ISCHEMICSTROKE	171	81
INTRACRANIAL BLEED	9	4.2
INTRACRANIAL TUMOR	5	2.3
METABOLIC CAUSE	3	1.5
MULTIPLE SCLEROSIS	2	1.05
NORMALSTUDY	20	9.5
TOTAL	210	100



TableNo:4- PercentageDistributionOfMriPostive IschemicCases AccordingToAge

Age	NUMBER	Percent
<=50	36	21.1
51-60	40	23.4
61-70	60	35.1
>70	35	20.5
Mean±SD	61±12.5	

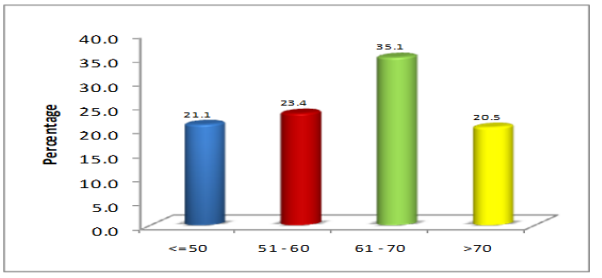


Fig No:4- Percentage Distribution Of Postive Ischemic Cases According To Age

Table No 5: Percentage Distribution Of Mri Postive Ischemic Cases According To Gender

gender	number	Percent
Male	100	58.5
Female	71	41.5
Total	171	100

Fig.5 Percentage Distribution Of Postive Ischemic Cases According To Gender

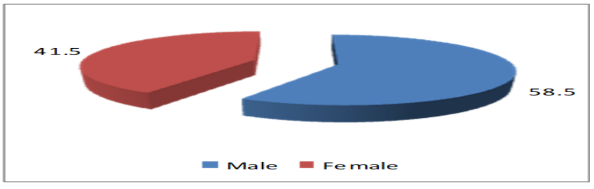


Table No 6: Distribution Of Cases According To Dwmri

DIAGNOSIS	NUMBER	PERCENTAGE
DWMRI POSITIVE	171	89
DWMRI NEGATIVE	20	11
TOTAL	191	100

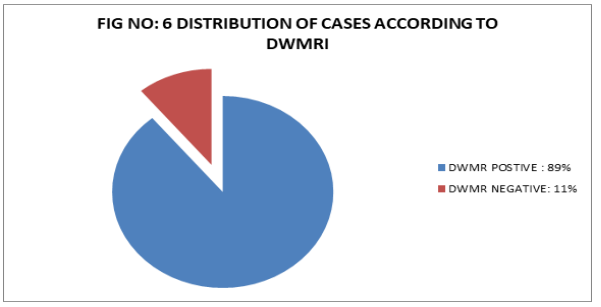


Table No 7: Distribution Of Ischemic Criteria In A False Positive Nect Cases

ISCHMIC CRITERIA	ACUTE		SUBACUTE	
	NUMBER	PERCENTAGE	NUMBER	PERCENTAGE
HYPERDENSE MCA	2	10%	4	20%
OBLITERATION OF LENTIFORM NUCLEUS	5	25%	3	15%
INSULAR RIBBON SIGN	5	25%	3	15%
LOSS OF GREY WHITE MATTER DIFFERENTIATION	6	30%	1	5%
HYPODENSITY	5	25%	13	65%
MULTIPLE SIGNS/ CRITERIA OF ISCHEMIC CASES	8	40%	15	75%

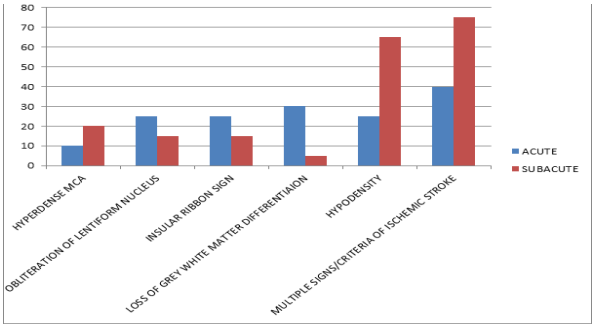


Fig No 7: Distribution Of Ischemic Criteria In A False Positive Nect Case

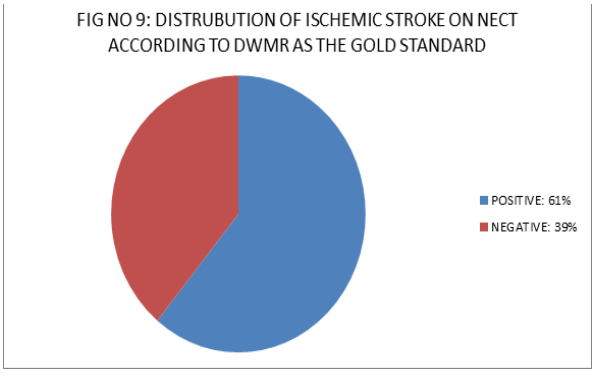


Table No 8 : Distribution Of False Postive Ischemic Nect (atlest One Criteria) According To Dwmr

TERITORY	NUMBEROFCASES	PERCENTAGE
ANTERIOR CIRCULATION	10	50
WATERSHED	3	15
POSTERIOR CIRCULATION	7	35
TOTAL	20	100

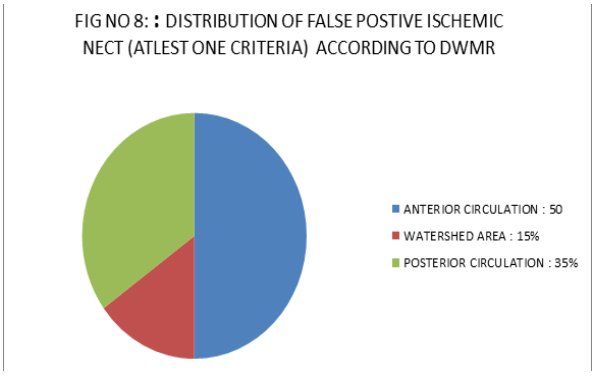


Table No 9 : Distrubution Of Ischemic Strokeon Nect According To Dwmr As The Gold Standard

CTDIAGNOSIS	NUMBER	PERCENTAGE
POSTIVE	105	61
NEGATIVE	66	39
TOTAL	171	100

Table No: 10 Percentage Distribution Of True Posiitve And False Positive Early Ischemic Cases With Dwmr As The Gold Standard

	NUMBEROFCASES	PERCENTAGE
TRUEPOSITIVE	105	84
FALSEPOSITIVE	20	16
TOTAL	125	100

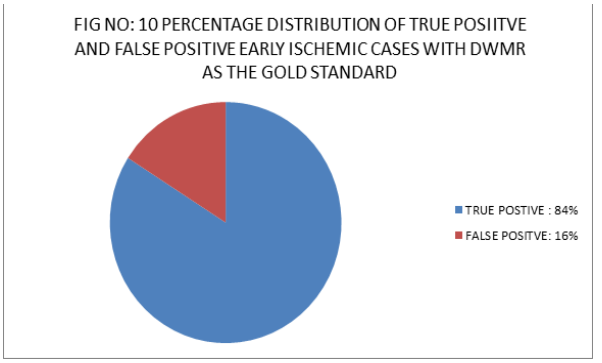
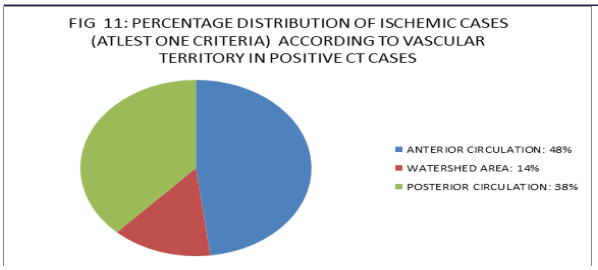


Table 11 Percentage Distribution Of Ischemic Cases (atlest One Criteria)According To Vascular Territory In Positive Ct Cases

VASCULAR TERRITORY	NUMBEROFCASES	PERCENTAGE
ANTERIOR CIRCULATION	50	48
WATERSHEDAREA	15	14
POSTERIOR CIRCULATION	40	38
TOTALCASES	105	100



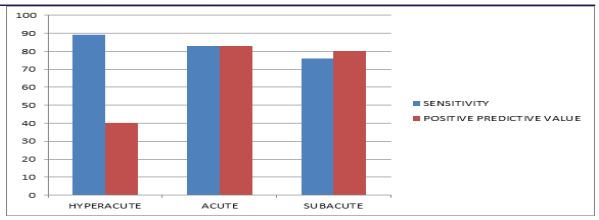


Fig No 12: Sensitivity And Positive Predoctive Value Of Positive Ct Cases With Positive Dwmr According To Time Of Onset

TableNo12: Sensitivity And Positive Predoctive Value Of Positive Ct Cases With Positive Dwmr According To Time Of Onset

TIME DISTRIBUTION	POSITIVE CT CASES	POSITIVE DWMR CASES	SENSITIVITY	POSITIVE PREDICTIVE VALUE
HYPERACUTE	35	86	89 %	40%
ACUTE	50	60	83%	83%
SUBACUTE	20	25	76%	80%
OVERALL CASES	**105**	**171**	**84%**	**61%**

TableNo:13 Percentage Distribution Of Hyperacute/acute/subacute Ischemic Criteria In Ct Compared To Dwmr

ISCHEMIC SIGNS	HYPERACUTE		ACUTE		SUBACUTE	
	NUM	%	NUM	%	NUM	%
HYPERDENSE MCA SIGN	10	31%	12	37%	10	31%
LOSS OF GREY WHITE MATTER DIFFERENTIATION	40	46%	25	39	10	15%
OLITERATION OF LENTIFORM NUCLEUS	25	65%	10	26%	3	8%
INSULAR RIBBON SIGN	25	65%	9	24%	4	10.5
HYPODENSITY	5	6%	25	28%	60	66%

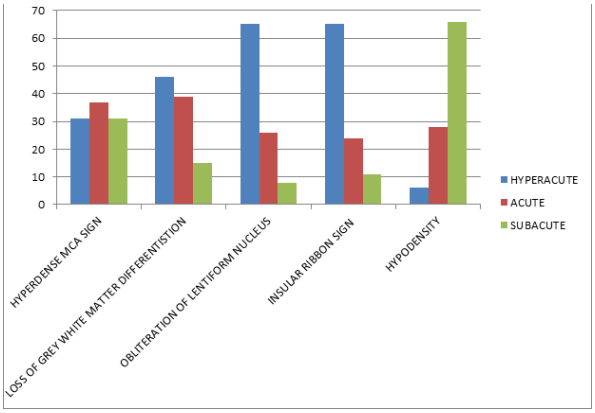


Fig No 13 Percentage Distribution Of Hyperacute/acute/subacute Ischemic Criteria In Ct Compared To Dwm

Table: 14 Percentage Distribution Of Blooming On Gre In Dwmr Positive Cases

GRE	NUMBER	Percent
POSITIVE	6	3.5
NEGATIVE	165	96.5
TOTAL	171	100

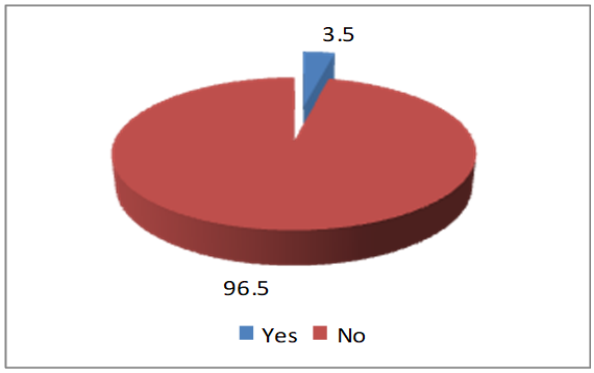


Fig.14 Percentage Distribution Of Blooming On Gre In Dwmr Positive Cases

Table.15 Percentage Distribution O f Dw Positive Cases Comparing With Other Mr Sequence (t1,t2,flair)

Other MR sequence	NUMBER	Percent
POSITIVE	63	36.8
NEGATIVE	108	63.2

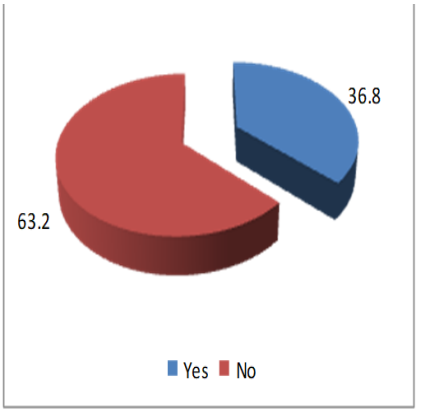


Fig.15 Percentage Distribution Of Dw Positive Cases Comparing With Other Mr Sequence (t1,t2,flair)

TableNo16: Percentage Distribution Of Other Stroke Mr Standard Sequence (t1,t2& Flair) Positivity According Time Of Onset Of Ischemic Stroke

SEQUENCES	HYPER ACUTE		ACUTE		SUBACUTE		TOTAL
	NUM	%	NUM	%	NUM	%	
T1	7	18	11	29	20	52.6	38
T2	18	37.5	18	37.5	12	25	48
FLAIR	25	43	20	34.5	13	22.4	58

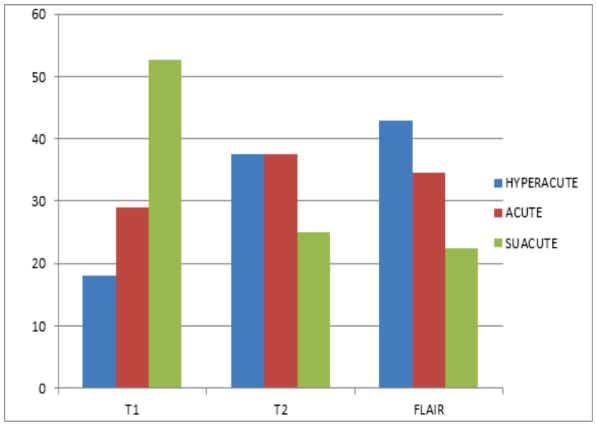
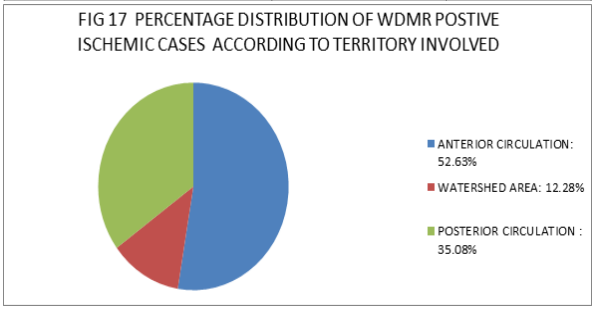


Fig No16: Percentage Distribution Of Other Stroke Mr Standard Sequence (t1,t2& Flair) Positivity According Time Of Onset Of Ischemic Stroke

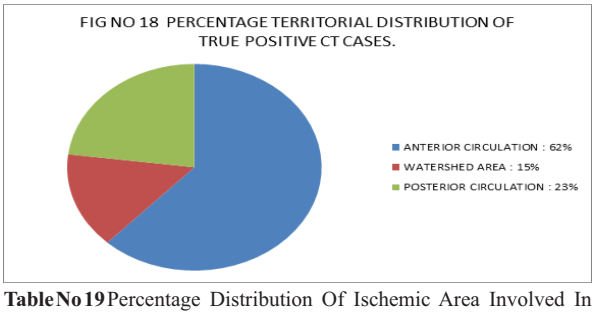
Table No17: Percentage Distribution Of Dwmr Ischemic Cases According To Territory Involved

TERRITORY	NUMBEROF CASES	PERCENTAGE
ANTERIORCIRCULATION	90	52.63
WATERSHED AREA	21	12.28
POSTERIORCIRCULATION	60	35.08
TOTAL	171	100



TableNo18 Percentage Territorial Distributions Of True Positive Ct Cases.

TERRITORY INVOLVED	NUMBER OF POSITIVECTCASES	PERCENTAGE
ANTERIOR CIRCULATION	65	62
WATERSHED AREA	15	15
POSTERIOR CIRCULATION	25	23
TOTAL	105	100



TableNo19 Percentage Distribution Of Ischemic Area Involved In Anterior Circulation According To Dwi

ARTERIAL TERITORY	NUMBEROFCASES	PERCENTAGE
ANTERIOR CHOROIDAL ARTERY	5	5.5
ANTERIOR CEREBRAL ARTERY	32	35.5
MIDDLE CEREBRAL ARETERY	53	58.88
TOTAL	90	100

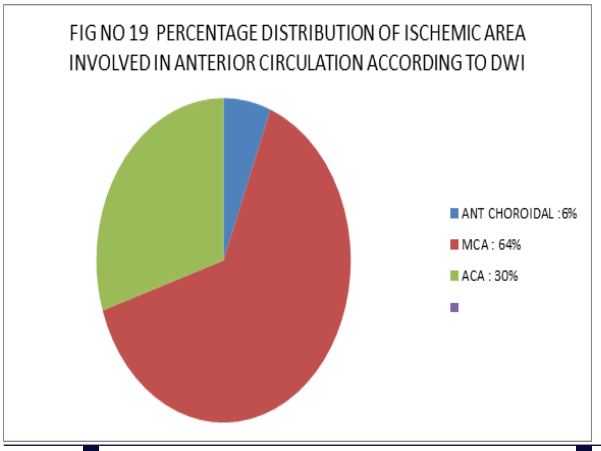


Table No : 20 Percentage Distribution Of True Positive Ct Cases In Anterior Circulation

REGIONINVOLVED	NUMBEROFCASES	PERCENTAGE
ACA	22	35
MCA	40	61
ANTEIOR CHOROIDAL	3	4
TOTAL	65	100

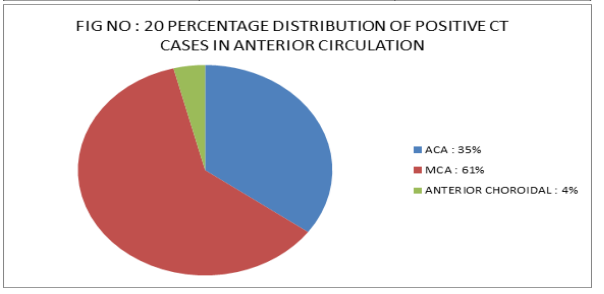


Table 21: Percentage Distribution Of Dwi Positive Ischemic Cases In Watershed Area

REGIONINVOLVED INWATERSHED AREA	NUMBEROFCASES	PERCENTAGE DISTRIBUTION
CORTICAL BORDERZONE INFARCT	10	47.6
INTERNAL BORDERZONE INFARCT	11	52.4
TOTAL	21	100

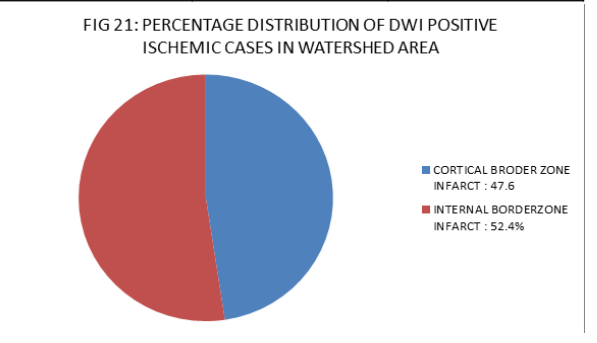


Table 22: Percentage Distribution Of Positive Ct Cases In Watershed Area

AREA INVOLVED	NUMBEROFCASES	PERCENTAGE DISTRIBUTION
CORTICAL BORDERZONE	9	60
INTERNAL BORDERZONE	6	40
TOTAL	15	100

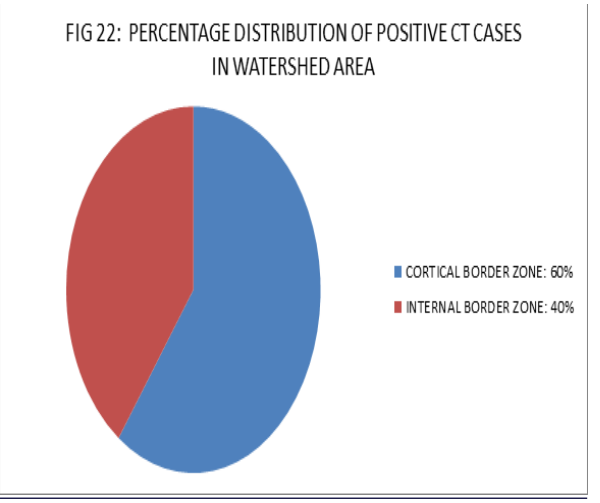


Table 23: Percentage Distribution Of Ischemic Cases In Posterior Circulation Acccording To Dwi

AREAINVOLVED	NUMBEROFCASES	PERCENTAGEOF DISTRIBUTION
PCA	38	63.33
BA	10	16.6
SCA	6	10
AICA	2	3.3
PICA	4	6.6
TOTAL	60	100

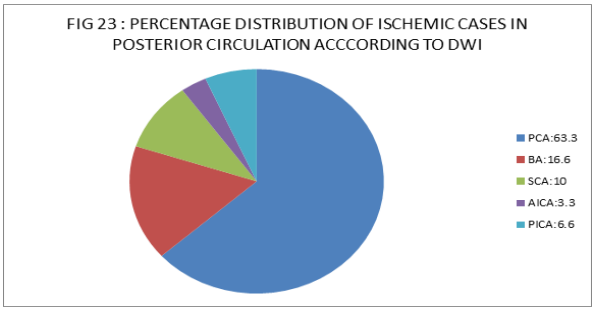


Table 24: Percentage Distribution Of True Positive Ct Cases In Posterior Circulation

AREAINVOLVED	NUMBEROFCASES	PERCENAGE DISTRIBUTION
PCA	11	44
BASILARARTERY	5	20
SCA	3	12
PICA	3	12
AICA	3	12
TOTAL	25	100

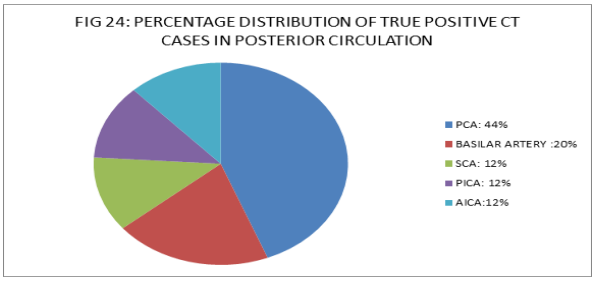
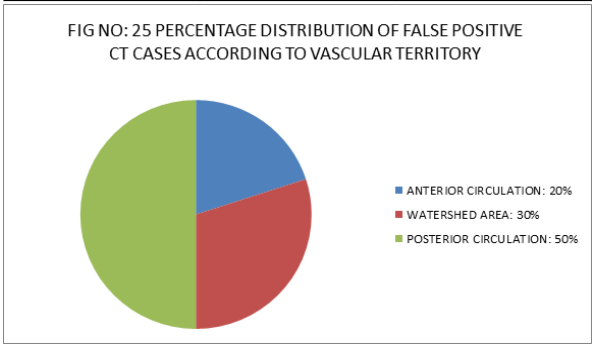


Table 25: Percentage Distribution Of False Positive Ct Cases According To Vascular Territory

Areainvolved	number	percentage
Anterior circulation	4	20
Watershed area	6	30
Posterior circulation	10	50
Totalcases-CTFALSE POSITVECASES	20	100



DISCUSSION:

This study was done to evaluate the diagnostic accuracy of CT over MRI in acute/subacute ischemic stroke. Therefore, we randomized eligible patients suffering from clinically suspected which was send from various other clinical departments to our department of radio-diagnosis for sequence of the imaging modalities.

The study was done on 210 patients who were clinically suspected stroke. Out of that 19 patients were diagnosed to have hemorrhage, intracranial neoplasms, multiple sclerosis and metabolic causes.

In the rest 191 patients, 171 patients showed hyperintense signal intensity and corresponding ADC showed hypointense area, ie restricted diffusion which are diagnosed to have acute/subacute ischemic stroke.Rest of the 20 NECT cases showed atleast one positive evidence of early acute/subacute ischemic stroke, however DWI images were normal.

Study shows male predominance in ischemic cases.100 patients out of 171 patients were males which was 58.5% with 71 females which accounts 41.5% of the total cases.

According to age distribution also, ischemic stroke cases were more in patients above 60 year of age and with associated comorbidities.

According to the onset of symptoms, 50% cases were found to have ischemic symptoms before 24 hours of onset(hyperacute), 35% were acute (1-7 days)and rest 14.6% were subacute(1-3 weeks).

Out of 171 positive in DW MR cases and we found that 105 cases were true positive on CT with atleast one positive evidence of acute/subacute ischemic stroke on NECT. Sensitivity of detecting hyperacute ischemic stroke was 89% and positive predictive value was 83% each in our study. In subacute ischemic stroke sensitivity is 76% and positive predictive value 80%. This interprets that the positive predictive value of hyperacute cases are low compared to the other phases of ischemic infarct.

Saur et al considered all of the early ischemic signs on CT assessment of early ischemic changes, with a resultant sensitivity of 73% (105). Comparing with this study, we were able to get only 84% overall sensitivity. This might be due to the large number of cases we took in our study.

DETECTION OF EARLY SIGNS ON NECT:

Out of 171 DWMR positive cases, 105 cases showed atleast one criteria of acute/subacute ischemic infarct, ie 61% of the cases and the rest 39% cases were false negative(dint show any signs of acute/subacute infarct).

Gonzáles et al.cohort study of 31 patients ,ischemic criteria were found in 45% of cases .In our setting we got 61% of the cases. This might be due to the increased number of cases in our study(106).

Atleast one early ischemic signs were found in these 105 cases. In that signs like obliteration of lentiform nucleus(65%), loss of grey white matter differentiation (46%) and insular ribbon signs(65%) were found more during hyperacute and in the acute phases of ischemia. Other ischemic signs like hyperdense MCA (31%) and hypodensity (66%) were more during later phases.

Comparing studies with Nakano S, Iseda T Kavano H et al also shows high sensitivity in detecting signs like obliteration of lentiform nucleus, loss of grey white matter differentiation, insular ribbon sign are more in hyperacute and early acute phases of ischemic infarct(107).

Pressman BD, Tourje EJ studies shows sensitivity of detecting the late acute and subacute ischemic signs like hypodensity are very high. In our study also, we were able to get high percentage distribution of detecting hypodensity in late acute/subacute phases(108).

Distribution According To Vascular Territory

Territorial distribution of true positive CT cases assessed and out of 105 cases, 62% cases were in the anterior circulation, 23 % in the posterior and 15 % in the watershed area .

According to 171 DW MR positive case, 62% of total cases were found to be anterior circulation, followed by 23% , 15% in posterior circulation and in watershed area respectively.

Fiebach J, Jansen O, Schellinger P et al studies states the involvement of anterior circulatory ischemic stroke is high compared posterior and watershed areas. In our study also we were able to get more ischemic areas in the anterior circulation(109).

In anterior circulation of true positive 105 NECT cases, 61% were seen in the MCA territory which is the most common site in ischemic infarction followed by anterior cerebral artery (35%) & least in the anterior choroidal artery (4%).

15positive NECT cases were seen in the watershed areas, in which 60% of the cases were seen in cortical border zone and rest 40% in the internal border zone.

In case of posterior circulation in a true positive CT case, 60% were in the PCA territory, 20 % in the basilar artery and rest in the other three regions.

In DWMR 171 cases, 90 cases (52%) were seen in the anterior circulation. Out of that more than 58 % cases are detected in the middle cerebral artery region which is a common site of ischemic infarction and the rest were seen mostly in the anterior cerebral artery and very few in the anterior choroidal region.

In watershed area, the distribution is almost equal in both cortical border zone and in the internal border zone regions.

DWMR positive cases in posterior circulation, 60 cases (35% of total) were detected in the posterior circulation. In that 63% cases were positive in posterior cerebral artery regions. While CT in the same area showed only 26% sensitivity in the posterior circulation..

Nouh A, Remke J, Ruland S et al &Merwick Á, Werring D studies shows that CT, which is the main brain imaging modality in hyperacute stroke, unfortunately, has a known limited sensitivity to assess strokes involving the posterior circulation, especially in the posterior fossa structures(110).

Comparison Between Dwmr To Other Standard Sequences.

Comparing the DWI imaging with other standard MR sequences (T1,T2& FLAIR), the sensitivity of other sequences are less with that of DWMR imaging. Out of 171 DWI positive cases, hyperintense signal intensity on T2/FLAIR and T1 hypointensity were seen in only 36% (ie 63cases)of the cases. In this most cases showed T2/FLAIR hyperintense signal on hyperacute and subacute ischemic cases where as T1 hypointesnties are more visible in late acute and subacute phases.

In a study by Thomalla et al (111) of 120 consecutive patients with stroke, it was reported that when restricted diffusion was present and T2/FLAIR imaging findings were negative, specificity (93%) and positive predictive value (94%) were high that the stroke was less than 3 hours old. However as the ischemic time progresses, the hyperintense signal develops, especially on T2.

Hemorrhagic transformation demonstrates a spectrum of findings ranging from small petechial areas of micro bleeding to large parenchymal hematoma.

Grossman RI, Yousem (112) have reported the hemorrhagic transformations in infarct cases, ranging from small petechial areas of micro bleeding to large parenchymal hematoma. However in our study it was found to have micro bleeds, iein 6 cases (3.5 %) were found to have blooming on GRE in subacute infraction. This is highly sensitive for detection of micro hemorrhages than in a standard ischemic CT protocol.

CONCLUSION:

In our study, criteria for diagnosis of acute/subacute ischemicstroke according to time of onset have got a vital role in diagnosis.We got 89% sensitivity of CT over DWMRI in hyperacute phase, 83% in acute and 76% in subacute phases. Positive predictive value was low in hyperacute phase of ischemic stroke, where as it was 83% & 80% in acute and subacute phases respectively.

While reviewing the ischemic criteria in positive CT cases, percentage distribution of ischemic criteria like hypodensity, dense MCA signs were more in the later phases of ischemia, iein the subacute phase, whereas other criteria like obliteration of lentiform nucleus, loss of grey white matter differentiations are more in the hyperacute and acute phases.

In case of DWI with other sequences, more findings of ischemic stroke were seen in subacute phases compared to hyperacute and acute phases. Other than DWI, FLAIR and T2 images shows a high sensitivity of detecting ischemic findings following DWI images.Our data indicate that in many patients with restricted diffusion and no change on FLAIR images, it is more likely than was initially thought that the stroke is less than 6 hours old. This states the importance of diffusion weighted images when compared to other MR imaging techniques.

In NECT, it is found difficult to diagnose small infarcts in brain stem and in the cerebellum mainly due to skull base artifacts, DWMR can provide even very small ischemic infarcts as early within an hour of onset. In our studies we are able to prove this theory with only few cases were positive brainstem regions on CT compared to DWMR.

In a primary care center the main role of imaging is to exclude an intracranial hemorrhage, define the ischemic region, to distinguish between infarct core and penumbra and to depict the vessel status. CT and MRI are modalities that can be used with confidence, both having their strengths and weaknesses.

MR imaging may help determine the age of an ischemic stroke, particularly in elderly patients. Findings on ADC maps and diffusion-weighted, FLAIR, and T1- and T2-weighted gradient-echo may help classify strokes as early hyperacute, late hyperacute, acute, subacute, or chronic and provide useful information for the medical team.

In a primary care center our recommendation is ,if NECT is negative in an acute onset with a strong suspicious symptoms, patients should undergo an DWMR sequence, where as the positive predictive value in NECT is more in later phases of ischemic stroke. Due to the high sensitivity and accuracy of DW MRI in the acute/subacute brain infarcts, a thrombolytic treatment can be tailored more accurately accordingly especially within and beyond the standard therapeutic window period of time.

The debate regarding the superiority of either CT or MRI for acute stroke imaging should not obscure the ultimate goal; that is, to increase the availability and improve the efficiency of thrombolytic therapy. From this standpoint, CT and MRI must be considered equivalent tools, and whichever technique available at each individual institution should be used in the best interest and benefit of the acute stroke patient.

List Of Abbreviations

MRI	MAGNETIC RESONANCE IMAGING
DWMR	DIFFUSION WEIGHTED MAGNETIC RESONANCE
T2W	T2 WEIGHTED
T1W	T1 WEIGHTED
TE	ECHO TIME
TR	REPITITION TIME
FOV	FIELD OF VIEW
FLAIR	FLUID ATTENUATION INVERSION RECOVERY
GRE	GRADIENT ECHO
ADC	APPARENT DIFFUSION COEFFICIENT
NECT	NON ENHANCED COMPUTED TOMOGRAPHY
ACA	ANTERIOR CEREBRAL ARTERY
PCA	POSTERIOR CEREBRAL ARTERY
MCA	MIDDLE CEREBRAL ARTERY
AICA	ANTERIOR INFERIOR CEREBELLAR ARTERY
PICA	POSTERIOR INFERIOR CEREBELLAR ARTERY
CBF	CEREBRAL BLOOD FLOW
MRA	MAGNETIC RESONANCE ANGIOGRAPHY
SWI	SUSCEPTIBILITY WEIGHTED IMAGES
ICH	INTRACRANIAL HEMORRHAGE
RTPA	TISSUE PLASMINOGEN ACTIVATOR

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