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Ct findings of ischemic stroke and hemorrhagic stroke

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الإهداء

وصلت رحلتي الجامعية إلي نهايتها بعد تعب ومشقة..

وها أنا ذا أختم بحث تخرجي بكل همة ونشاط،

وأمتن لكل من كان له فضل في مسيرتي،

وساعدني ولو باليسير،

الأبوين، والأهل، الأصدقاء، الأساتذة الكرام..

أهديكم بحث تخرجي.....

Objective:

To analyze CT findings in hemorrhagic and ischemic stroke patients correlation with clinical outcome and assess the interobserver agreement of stroke identification on CT imaging.

Material and Method:

CT imaging features of 100 cases and clinical data were verified and collected at AL-Nasiriyah Hospital from November 2021 to April 2022 and retrospectively analyzed for type, location, Risk factors (modifiable and non-modifiable) . The percentages, predictive values and compare the results with other countries results.

Results:

From both types of stroke ischemic stroke (64 %) is more common than Haemorrhagic stroke (36%) ,From all types of hemorragic stroke , intracerebral hemorrhage remains a common and devastating clinical problem (23%) , and There were (6%) of intraventricular hemorrhage , (4%) subdural Haemorrhage and (3%) of subarachnoid haemorrhage .

Also we analysed the most important risk factors of stroke which can classified according to whether they are a) non-modifiable such as age , gender hereditary or family history , b) well-documented and modifiable

Such as Cardiovascular disease , Hypertension , Diabetes mellitus , Dyslipidaemia , social life-style factors ,Cigarette smoking ,Obesity, physical inactivity Dietary factors ,or c) Less-well documented or potentially modifiable risk factors such as metabolic syndrome , Sleep apnoea.

Two variables on CT imaging, identified as significant as early mortality predictors, were hematoma volume more than 60 cm3, and presence of intraventricular hemorrhage extension ($p < 0.05$). The mass effect defined as midline and/or enlargement of contralateral ventricle was not significant ($p = 0.067$). The present study found concordance between CT brain interpretation by two neuroradiologists for the type of hemorrhagic stroke was very good otherwise ischemic stroke be very obvious by CT after few hours to days

Conclusion:

CT imaging is an imaging instrument for early identification of stroke patients and .providing imaging evidence of high mortality risk

Introduction:-

Stroke :- is a clinical syndrome , it's fourth leading cause of death in the United States, and the most common medical cause of disability.. Acute stroke is a medical emergency in which the outcome is highly dependent on prompt recognition and treatment. (1)Several states have adopted legislative Stroke Acts, which require emergency medical personnel to transport stroke victims to the nearest certified stroke center.. With the advent of specialized stroke centers, the inclusion of guideline-driven acute stroke care, including early rehabilitation, has become an important component in the total management of the stroke patient. (2) .Acute care issues address prevention of common post-stroke complications such as deep venous thrombosis; emphasize early mobilization, assessment, and management of dysphagia and nutritional status, cognitive and communication deficits, incontinence, and preventative skin care; and initiate interactive rehabilitation education including the family and caregivers. (3). Multiple rehabilitation modalities must be initiated during the acute care period, and continuity of care plans established to optimize long-term functional and health outcomes for the stroke survivor. (4).

Arterial Supply to the Brain :-

The central nervous system, like any system of the body, requires constant oxygenation and nourishment. The brain has a particularly high oxygen demand – at rest it represents one fifth of the body's total oxygen consumption. It is also very sensitive to oxygen deprivation, with ischemic cell death resulting within minutes.

There are two paired , arteries which are responsible for the blood supply to the brain; the vertebral arteries, and the internal carotid arteries. These arteries arise in the neck, and ascend to the cranium. Within the cranial vault, the terminal branches of these arteries form an anastomotic circle, called the Circle of Willis. From this circle, branches arise which supply the majority of the cerebrum. Other parts of the CNS, such as the pons and spinal cord, are supplied by smaller branches from the vertebral arteries. We shall now look at these individual components in more detail.

Internal Carotid Arteries :- The internal carotid arteries (ICA) originate at the bifurcation of the left and right common carotid arteries, at the level of the fourth cervical vertebrae (C4).

They move superiorly within the carotid sheath, and enter the brain via the carotid canal of the temporal bone. They do not supply any branches to the face or neck.

Once in the cranial cavity, the internal carotids pass anteriorly through the cavernous sinus. Distal to the cavernous sinus, each ICA gives rise to:

Ophthalmic artery – supplies the structures of the orbit.

Posterior communicating artery – acts as an anastomotic 'connecting vessel' in the Circle of Willis (see 'Circle of Willis' below).
Anterior choroidal artery – supplies structures in the brain important for motor control and vision.
Anterior cerebral artery – supplies part of the cerebrum.
The internal carotids then continue as the middle cerebral artery, which supplies the lateral portions of the cerebrum.

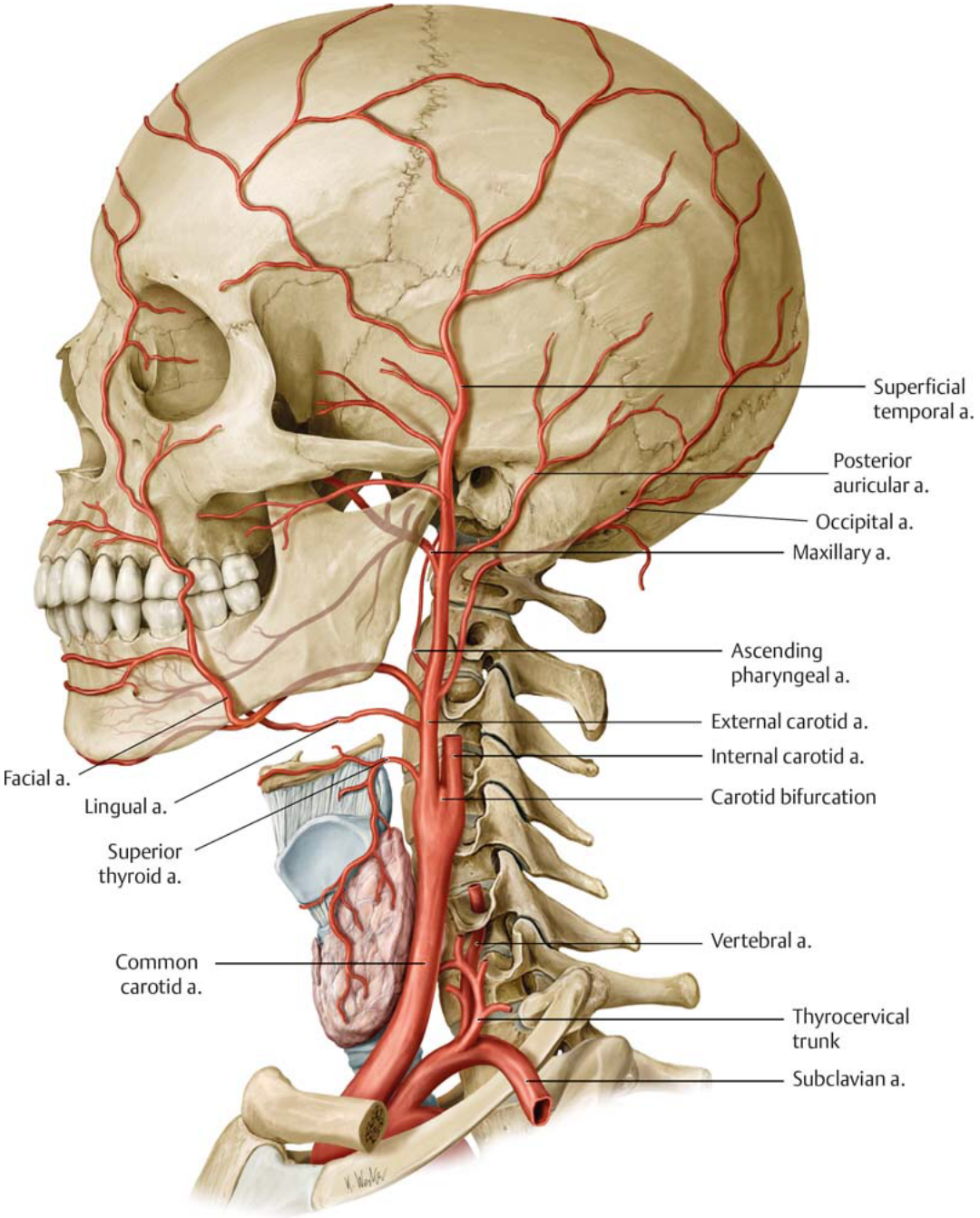


Fig. 1. Lateral view at the carotid bifurcation. The common carotid artery is divided into the external and internal carotid arteries.

Arterial Circle of Willis :-

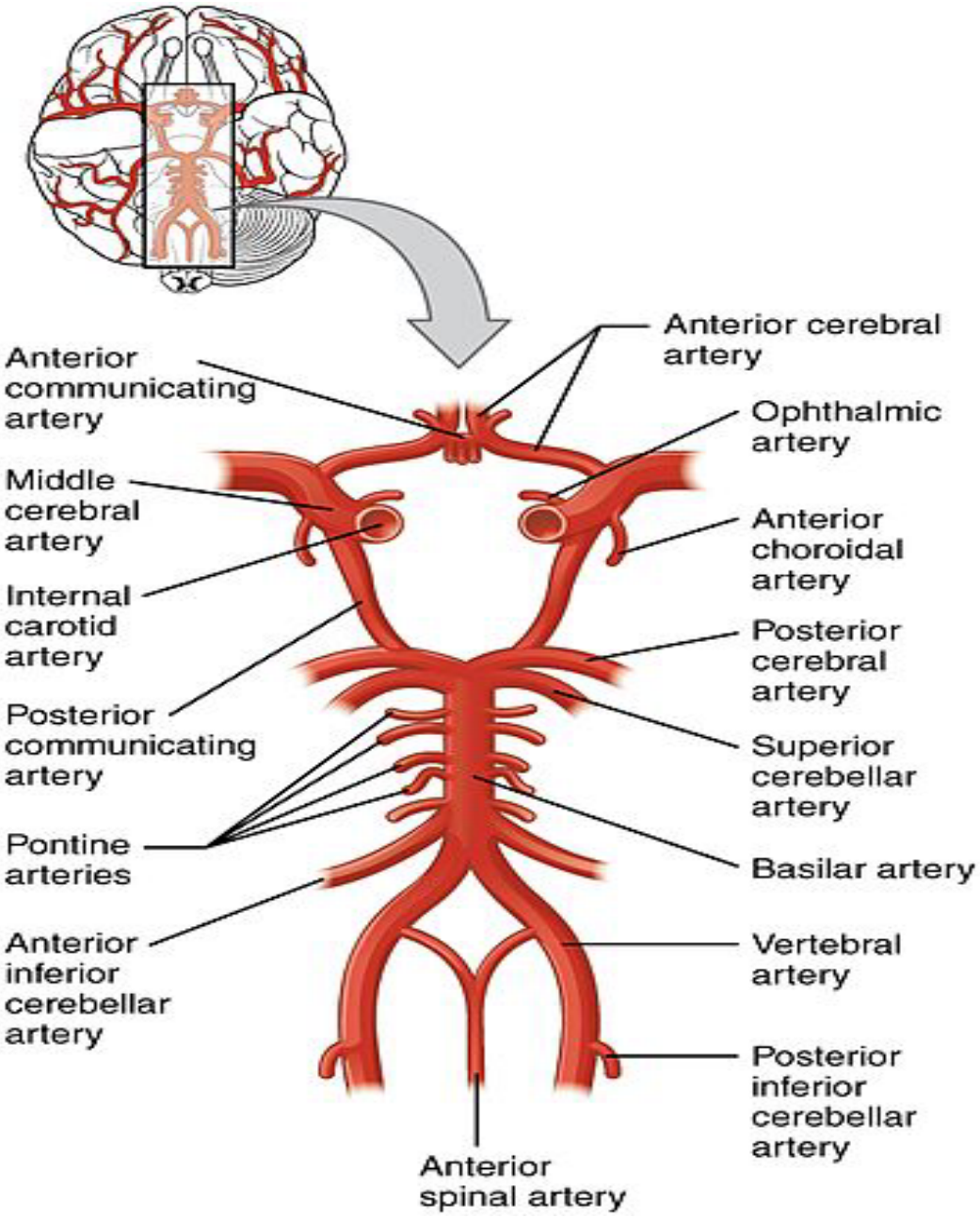


Fig .2. Schematic of the blood supply to the brain. Circle of Willis , by teachMeSeries (2022).

The terminal branches of the vertebral and internal carotid arteries all anastomose to form a circular blood vessel, called the Circle of Willis. There are three main (paired) constituents of the Circle of Willis:
Anterior cerebral arteries – terminal branches of the internal carotid arteries.

Internal carotid arteries – located immediately proximal to the origin of the middle cerebral arteries.
Posterior cerebral arteries – terminal branches of the basilar artery.
*To complete the circle, two 'connecting vessels' are also present:
Anterior communicating artery – connects the two anterior cerebral arteries.
Posterior communicating artery – branch of the internal carotid, this artery connects the ICA to the posterior cerebral artery.

Regional Blood Supply to the Cerebrum :-

There are three cerebral arteries; anterior, middle and posterior. They each supply a different portion of the cerebrum. The anterior cerebral arteries supply the anteromedial portion of the cerebrum. The middle cerebral arteries are situated laterally, supplying the majority of the lateral part of the brain. The posterior cerebral arteries supply both the medial and lateral parts of the posterior cerebrum.

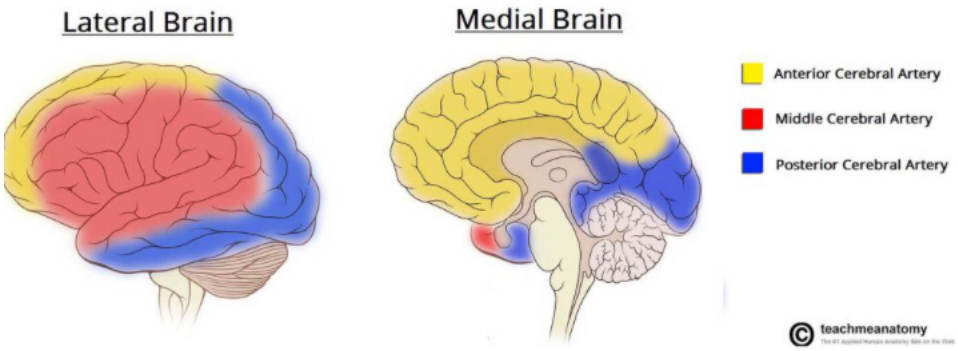


Fig .3. Overview of the blood supply to the cerebrum, by Frank Gaillard and Patrick J. Lynch [CC-BY-SA-3.0], via Wikim edia Commons. teachMeSeries (2022). (5).

STROKE PATHOPHYSIOLOGY:-

The brain is particularly sensitive to oxygen starvation. A stroke is an acute development of a neurological deficit, due to a disturbance in the blood supply of the brain.There are four main causes of a cerebrovascular accident:
Thrombosis – obstruction of a blood vessel by a locally forming clot.
Embolism – obstruction of a blood vessel by an embolus formed elsewhere.
Hypoperfusion – lack of blood supply to the brain, due to systemically low blood pressure (e.g shock).
Haemorrhage – an accumulation of blood within the cranial cavity.
Out of these four, the most common cause is embolism. In many patients, the atherosclerotic embolus arises from the vessels of the neck.

How does Haemorrhagic stroke occur ?

Haemorrhagic stroke:- caused by damage to cerebral blood vessels , leading to bleeding in the brain or surrounding space puts pressure on brain cells

and damages them. The potential etiologies are hypertensive , Amyloid angiogiopathy , AVM , DVA , SAH , medication and traumatic.

How does ischemic stroke occur?

Ischemic stroke:- caused by blockage of a blood vessel in the brain. Acute Ischemic infarction at left frontoparietal region.

Intracerebral Aneurysms:- An aneurysm is a dilation of an artery, which is greater than 50% of the normal diameter. They are most likely to occur to occur in the vessels contributing to the Circle of Willis. They are particularly dangerous – producing few symptoms until they rupture. Upon rupture, blood typically accumulates in the subarachnoid space – with a subsequent increase in intracranial pressure.Once the artery wall has ruptured, it is a medical emergency, and the patient is likely to die unless treated swiftly. Treatment of an intracerebral aneurysm is surgical.

STROKE SYNDROMES :- Knowledge of the vascular anatomy of the brain and the effects of specific arterial occlusions is important in determin- ing the location and size of the infarct. Chapters 5 provides detailed clinical and vascular-neuroanatomic descriptions of anterior and posterior circulation stroke syndromes. Most stroke syndromes can be divided into (a) anterior circula- tion syndromes—middle cerebral artery (MCA) and anterior cerebral artery (ACA) syndromes, (b) posterior circulation syndromes, (c) lacunar syndromes, and (d) borderzone (or watershed) infarct syndromes. A brief description of each of these syndromes is given in the following text.

Middle Cerebral Syndromes:- Occlusion of the MCA will present with contralateral weak- ness, face and arm more than leg associated with sensory loss, visual field cut, and aphasia when the dominant hemi- sphere is affected and hemineglect when the nondominant hemisphere is involved.

ACA Syndromes:- Occlusion of the ACA will present with weakness, leg more than arm and face, behavioral disturbances such as abulia, mut- ism, anterograde amnesia, grasping and sphincter dysfunction.

Posterior Circulation Syndromes:- Strokes in the posterior circulation can present with dizziness, anisocoria, diplopia, dysphagia, ataxia, hemiplegia, quadri- plegia, and coma depending on the location of the occlusion. The major posterior circulation syndromes include posterior inferior cerebellar artery (PICA, also known as "Wallenberg") syndrome, anterior inferior cerebellar artery (AICA) syn- drome, and posterior cerebral artery (PCA) syndrome. Wal- lenberg syndrome presents with ipsilateral facial sensory loss, contralateral body sensory loss, dysphagia, dysphonia, ipsi- lateral ataxia, Horner's syndrome, and nystagmus. Infarction of the AICA territory usually presents with sudden hearing loss, vertigo, vomiting, ipsilateral facial palsy, ataxia, and con- tralateral sensory loss. Infarction of the PCA territory can pres- ent with multiple symptoms depending on the area affected. The most common symptom is visual field loss, and when there is involvement of the P2 branches, patients present with impaired cognition, amnesia, and changes in personality.

Lacunar Syndromes:- Most lacunar infarcts are secondary to occlusion of small penetrating arteries; there are five "classic" lacunar syn- dromes: pure motor hemiparesis, pure sensory stroke, sen- sorimotor stroke, dysarthria–clumsy hand syndrome, and ataxic hemiparesis. The most common presentation is pure motor and pure sensory stroke.

Watershed Infarct Syndromes:- Watershed infarcts are secondary to severely reduced flow in one or multiple vascular territories that leads to infarction of distal areas lying between two vascular territories. The most common are infarcts in the ACA/MCA watershed region, classically described as the man-in-a-barrel syndrome because of the especially proximal upper extremity weakness, although most of the time patients present with heteroge- neous symptoms.

STROKE RECOGNITION :-

Rapid recognition of the signs and symptoms of stroke, and timely access to stroke centers, are crucial to optimizing acute care for stroke. Too often, patients develop signs of stroke and wait hours before seeking care, believing that the deficits will go away if they wait long enough. A study by Feldman in 1993 showed that the median time from onset of symptoms to pre- sentation to emergency departments was 13 hours . Only 42% of patients presented within 24 hours. During the course of the National Institutes of Health rt-PA Pilot Study, pub- lic education and awareness campaigns were conducted to encourage early hospital arrival. Following this campaign, the mean time from symptom onset to hospital arrival declined significantly (3.2 h vs. 1.5 h). The use of 911 increased from 39% in the first quartile of the study to 60% in the fourth quar- tile. Community forums have also been successful in creating and extending awareness of the need for immediate action. The five most common symptoms of stroke include:

1. Sudden numbness or weakness of face, arm, or leg, especially on one side of the body
2. Sudden confusion, trouble speaking or understanding 3. Sudden trouble seeing in one or both eyes
4. Sudden trouble walking, dizziness, loss of balance, or coordination
5. Sudden severe headache with no known cause

Public education as to the significance of these symptoms and the importance of early evaluation is the goal of any comprehensive stroke program.

Emergency medicine system protocols are also critical in the early treatment of stroke. Proper training of paramedics allows these frontline personnel to obtain crucial information from family or bystanders. This includes obtaining history regarding time of onset and medications the patient might be taking. This historical information, as well as physical findings such as aphasia, motor deficit, and vital signs, can be called to the hospital emergency department so that a stroke alert protocol can be activated, saving significant time in treatment.(6).

Risk factors for stroke :-

risk factors for stroke have been derived from analyses of
data collected during prospective and case control studies of men and women. Reported predisposing factors are :-

Non-modifiable risk factors :- Age Race Sex Family history of genetic diseases , e.g Moya-moya disease, CADASIL , COL4A1 , , hereditary cavernous malformations.
Well-documented and modifiable risk factors :- Cardiovascular disease Coronary artery disease Non-valvular atrial fibrillation Cerebral large artery disease Cerebral small artery disease Peripheral artery disease Hypertension (>130/80mmHg) Diabetes mellitus Dyslipidaemia High total cholesterol High low-density lipoprotein cholesterol (>190mg/dL) Environmental and social life-style factors Cigarette smoking Obesity, physical inactivity Dietary factors Sodium intake >2.300mg Potassium intake <4.700mg Arterial dissection Arteritis/vasculitis Sickle cell disease Fabre disease
Less-well documented or potentially modifiable risk factors :- Metabolic syndrome Alcohol use (>5 drinks/day or more than moderate intake) Hyperhomocysteinemia Patent foramen ovale (PFO) Drug abuse Hypercoagulability Oral contraceptive use Acute/chronic inflammation (poor dental health care) Migraine including genetic forms High lipoprotein (a), high triglycerides, HDL cholesterol High lipoprotein-associated phospholipase A2 Sleep apnoea

Risk factors for stroke may be classified according to whether they are a) non-modifiable, b) well-documented and modifiable or c) less-well documented or potentially-modifiable (Table.1). OXFORD NEUROLOGY LIBRARY ,Stroke ,Michael G. Hennerici ,Rolf Kern ,Kristina Szabo ,Johannes Binder ,Department of Neurology, University of Heidelberg, Universitätsklinikum Mannheim, Mannheim, Germany , 2012.

Management of acute stroke :-

EMERGENCY DEPARTMENT MANAGEMENT OF STROKE :- Because of the importance of rapid intervention in treating patients with ischemic stroke, it is important that hospitals develop protocols and order sets to be used when a patient with symptoms of stroke arrives. If the patient is transported by emergency medical services that communicate with the emergency department, the hospital can institute the pro- tocol immediately on arrival, but preferably before. Several criteria should be **established in**

the emergency department regarding minimizing delays, including activation of the stroke team, timely interpretation of studies, and adminis- tration of fibrinolytic therapy.

Patient History and Evaluation :- The history should focus on determining whether the symptoms are a result of stroke or other medical conditions that can mimic stroke symptoms. The latter include migraine, seizure, syncope, and hypoglycemia. To determine whether the patient may be a candidate for fibrinolytic therapy, it is essential to establish the time of symptom onset, or, if this is unavailable, the time that the patient was last known to be free of stroke symptoms.As with other critically ill patients, initial evaluation and management should address the ABCs: airway, breathing, and circulation. Patients with hypoxia should be given supplemental oxygen to maintain oxygen saturation >94%. In the emergency care setting, a rapid but thorough neurolog- ical assessment is needed. The National Institutes of Health Stroke Scale (NIHSS), shown in Table 2, is an 11-item scale.

Ia. Level of Consciousness (LOC)	Alert——>0 Not alert but arousable by minor stimulation to obey, answer, or respond—>1 Not alert; requires repeated stimulation to attend, or is obtunded and requires strong or painful stimulation to make movements.——>2 Response only with reflex motor or autonomic effects or totally unresponsive, flaccid, and are flexic. ——>3
Ib. LOC Questions	Answers both correctly ——> 0 Answers one correctly——> 1 Answers neither correctly——>2
Ic. LOC Commands	Performs both tasks correctly——0 Performs one task correctly——>1 Performs neither task correctly——>2
II. Best Gaze	Normal ——> 0 Partial gaze palsy——>1 Forced deviation or total gaze paresis——>2
III. Visual	No visual loss——> 0 Partial hemianopia——>1 Complete hemianopia——>2 Bilateral hemianopia (blind including cortical blindness)——>3

IV. Facial Palsy	Normal symmetrical movements——> 0 Minor paralysis——>1 Partial paralysis——>2 Complete paralysis of one or both sides——>3
V. Motor Arm Right arm Left arm	No drift; limbs holds 90 (or 45) degrees for full 10 seconds——>0 Drift; limb drift down before full 10 seconds, does not hit bed or other support——>1 Some effort against gravity——>2 No effort against gravity——>3 No movement——>4
VI. Motor Leg	No drift; limbs holds 90 (or 45) degrees for full 5 seconds——>0 Drift; limb drift down before full 5 seconds, does not hit bed or other support—>1 Some effort against gravity——>2 No effort against gravity——>3 No movement——>4
VII. Limb Ataxia	Absent——>0 Present in one limb——>1 Present in two limbs——>2
VIII. Sensory	Normal——>0 Mild-to-moderate sensory loss——>1 Severe to total sensory loss——>2
IX. Best Language	No aphasia; normal——>0 Mild-to-moderate aphasia——>1 Severe aphasia——>2 Mute, global aphasia——>3
X. Dysarthria	Normal——>0 Mild-to-moderate dysarthria——>1 Severe dysarthria——>2
XI. Extinction or Inattention	No abnormality——> 0 Visual, tactile, auditory, spatial, or personal inattention in one of the sensory modalities——> 1 Profound hemi-inattention or extinction to more than one modality——>2

Table. 2. The NIH Stroke Scale.

that can rapidly quantify the neurological defi cits of a stroke patient. The use of standardized assessment tools has proved useful when discussing the

patient's condition with the treating primary medical team, and for continuity of rehabilitation care. Training for this, as well as certifi cation for performing the evaluation, can be obtained through several mechanisms such as the American Stroke Association website.

Tissue Plasminogen Activator: :- Recombinant tissue plasminogen activator (rt-PA) is a serine protease that converts plasminogen to plasmin, a fi brinolytic enzyme (Figure 4) Upon administration, rt-PA increases plasmin enzymatic activity, resulting in fi brinolysis. It is used to treat the stroke in the acute stage in an attempt to restore fl ow to the ischemic area, and should be administered as quickly as feasible, by protocol standards within a 3- to 4.5-hour time period to restore blood supply and optimize recovery. rt-PA was approved by the FDA in 1996 for the treatment of acute stroke based on fi ndings of the NINDS stroke trial in 1995 (7). This double-blind placebo-controlled trial demonstrated that patients treated with rt-PA within 3 hours of symptom onset had a 30% greater likelihood of having minimal to no disability 90 days following treatment, compared to a placebo-treated group. There was a 6.4% risk of symptomatic intracerebral hemorrhage in the rt-PA treated group, compared to 0.6% in the placebo group. However, even considering the risk of bleeding, the mortality at 90 days was 21% in the placebo group, and only 17% in the rt-PA group. Subsequent analyses have shown that these f i ndings with rt-PA in the NINDS trial hold up for improved outcomes at the one-year time point (8). The benefi ts of early thrombolytic therapy are corroborated by the results of two European Cooperative Stroke Studies, as well as clinical experience, substantiating the effectiveness of rt-PA when used according to the guidelines of the clinical trials (9). More recently, the third European Cooperative Stroke Study showed benefi t for patients who were treated between 3 and 4.5 hours from symptom onset. Patients treated within this time window were more likely to have a favorable

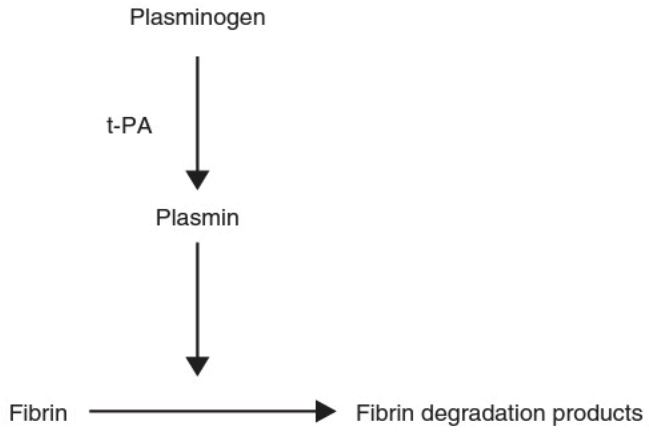


FIG .4. Diagram showing the site of action of recombinant t-PA to activate plasmin, which mediates fi brinolysis.

outcome (52.4% vs. 45.2%). However, there was a higher incidence of intracranial hemorrhage in the treatment group. The Food and Drug Administration considered this study to be insuffi cient to approve the use of rt-PA beyond the three hour window (10). Current American Heart Association/ American Stroke Association guidelines recommend treatment of patients in the 3- to 4.5-hour time period, excluding those who are more than 80 years old, those who are taking oral anticoagulants independent of international normalized ratio (INR), and those with NIHSS > 25, ischemia involving more than one-third of the MCA territory, and those with history of both stroke and diabetes mellitus (11). If it is determined that a patient's symptoms have been present less than 3 hours, there are other criteria that must be considered to determine if a patient is to be considered for rt-PA administration. These criteria are listed in Figure 3.3. If the patient is brought in between 3 and 4.5 hours after symptom onset, treatment can be considered using the same criteria listed in Figure 3.3 with additional exclusions: age >80 years old, taking anticoagulants, NIHSS > 25, involvement of >1/3 of the MCA territory affected, and history of both stroke and diabetes mellitus. The consideration of rapidly improving symptoms as they relate to thrombolytic therapy decision making has been somewhat problematic. Improvement over the baseline NIH score is not considered rapid improvement if the patient continues to have a significant deficit. A good rule of thumb has been to assume that the patient is not going to show further improvement in his or her condition. Is the deficit mild enough that the patient can continue to function at a high level? Even mild weakness might be devastating to an individual whose occupation depends on fine motor movements; hence, rt-PA could be a consideration in such patients, even if they have a low NIHSS value. Barber and colleagues noted that one-third of patients deemed to have mild stroke symptoms that excluded them from rt-PA treatment either died or were left in a dependent state (12). A majority of acute stroke patients have elevated blood pressure at the time of admission and across the initial days after stroke, which must be carefully managed. After rt-PA is administered, the patient should be monitored for at least 24 hours in an intensive care facility. The present recommendations are to keep systolic blood pressure below 185 mmHg and diastolic below 110 mmHg. Labetalol or nicardipine are the recommended agents to lower blood pressure. The lower limits for blood pressure should be a diastolic of 60 mmHg. Serial neurologic examinations are requisite, with appropriate clinical pathways for emergent management of complications such as symptomatic intracranial hemorrhage.

Imaging and Laboratory Studies :- Stroke outcome is highly dependent on time; therefore, all eligible patients should be treated with rt-PA within 60 minutes of hospital arrival. Hospitals should determine the delaying steps in their process and work with the different departments to improve the flow.

If any of the following are answered YES, Patient may NOT receive rt-PA :- Stroke symptom onset more than 3 hours (last time patient was known to be

without stroke symptoms, Age 18 or younger ,Comatose or unresponsive , Stroke symptoms clearing spontaneously. Stroke symptoms minor and isolated , Intracranial/Subarachnoid hemorrhage (SAH). Clinincal history suggestive of SAH even if CT negative , Active internal bleeding or acute trauma (fracture) on examination , INR greater than 1.7 , Platelet count less than 100,000 , Glucose less than 50 , HTN uncontrolled despite medication with systolic BP greater than 185 or diastolic BP greater than 110.

History of :- Active malignancy , Recent MI or pericarditis within the past 3 months , Recent arterial puncture at noncompressible site within previous 7 days (such as subclavian) , Lumbar puncture within 3 days , History of GI or urinary hemorrhage within 21 days , Pregnancy, lactation, or childbirth within 30 days , History of intracranial hemorrhage , Major surgery or serious trauma within in past 14 days , Seizure with postictal residual neurologic impairment , Major ischemic stroke or head trauma within the past 3 months , Heparin within 48 hours with PTT greater than upper limits of normal , Known AV malformation or aneurysm , Known bleeding disorder. A CT scan should be performed as soon as possible to exclude a hemorrhagic stroke. The CT scan may also demonstrate subtle early signs of infarction. Although the presence of these signs is associated with a poor outcome, this does not preclude the use of rt-PA unless there is evidence of hemorrhage. Only a noncontrast head CT is necessary to treat patients; obtaining advanced imaging is usually associated with delays in treatment. Further considerations relevant to management in the acute stroke setting include evaluation of glucose, systemic antithrombotic status, and blood pressure. In the acute stroke setting, glucose is important to determine, as hypoglycemia can be associated with focal neurologic deficits, while patients with hyperglycemia have a less favorable prognosis. Partial thromboplastin time (PTT), INR for prothrombin time, and platelet count should be obtained to prevent the use of thrombolytic therapy in patients with coagulation defects. However, for patients without known coagulopathy who are not taking anticoagulants, current guidelines recommend waiting only for finger stick glucose before treatment with rt-PA to avoid delays in treatment (11).

INPATIENT CARE OF ACUTE ISCHEMIC STROKE : -

Numerous studies have shown that patients admitted to specialized stroke units with multidisciplinary teams have reduced lengths of stay, reduced mortality, and better functional outcomes findings that have been translated into clinical practice with similar positive results . Among the issues that are central to inpatient care are management of blood pressure, blood glucose, fluid balance, close neurologic monitoring to detect any signs or symptoms of clinical deterioration, prevention of common post-stroke complications, and initiation of appropriate secondary stroke prevention measures. Early rehabilitation includes assessment for therapy needs with initiation of early mobilization, and comprehensive treatment plans, which should involve the family and caregivers and plans for continuity of care. (12).

Blood Pressure, Fluid, and Glucose Management:-

Blood pressure management in the acute stroke patient is not the same as for the general population. Normally, cerebral auto-regulation results in a constant CBF for mean arterial pressures between 60 and 160 mmHg. However, auto-regulation may be lost in the acute stroke setting, and as a result, decreasing blood pressure decreases CBF in the area of ischemia. Extreme hypertension should also be avoided, as it may cause hemorrhagic transformation of the infarct, encephalopathy, and result in systemic complications. The optimal range of blood pressure in the acute stroke setting is not well established. For patients who are not candidates for rt-PA, unless there is a cardiac, renal, or other medical reason for which the pressure must be lowered, the current recommendation is to treat the blood pressure only when it is above 220/120 mmHg. Agents such as

sublingual nifedipine that lower the blood pressure quickly should be avoided. A reasonable decrease in blood pressure would be 15% over 24 hours. For patients who have pre-existing hypertension and are taking antihypertensive medications, it is generally agreed that antihypertensive medications should be temporarily held or reduced, but can be restarted at 24 hours if the patients are neurologically stable, unless a specific contraindication to restarting treatment is known. Hypotonic and glucose-containing intravenous fluid are not recommended in the acute setting of cerebral infarction. Cytotoxic edema resulting from cellular membrane disruption with resulting swelling of the cell body develops with infarct. The use of these solutions can increase the cellular damage with influx of water into the cell. Normal saline is, therefore, generally utilized in these patients. Hypoglycemia can mimic stroke symptoms and, if severe, can result in neuronal injury. Blood glucose should be checked immediately in patients presenting with stroke symptoms, with rapid correction of hypoglycemia. Numerous studies have shown that, in addition to hypoglycemia, sustained glucose greater than 140 predicts less favorable stroke outcomes than lower glucose values. Hyperglycemia after acute ischemic stroke has been shown to predict higher mortality and worse 90-day clinical outcomes for individuals with and without preexisting history of type 2 diabetes mellitus , and appears to blunt the beneficial effect of early recanalization that accompanies rt-PA therapy . Glucose levels should be monitored, and if greater than 140 to 180, treatment with insulin is similar to management in other medical intensive care conditions, with close monitoring to prevent hypoglycemia. (13).

Evaluating and Managing Neurologic Deterioration :-

Between 15% and 30% of individuals with acute ischemic stroke experience neurologic deterioration during the acute hospitalization period, and this portends a much poorer prognosis . Factors linked to early neurological deterioration tend to be neurovascular, including stroke in progression, recurrent stroke, brain swelling, and hemorrhagic transformation.

Neurologic factors beyond recurrent or progressing stroke that can mediate clinical deterioration in the acute hospital setting include brain swelling with mass effect, herniation syndromes, hemorrhagic transformation of ischemic stroke, and seizures, including subclinical variants that are difficult to diagnose without electroencephalography and can greatly compromise stroke outcomes. A number of other potentially modifiable systemic and medical factors must be considered. They include evaluation of cardiopulmonary and fluid status, glucose and electrolyte status, assessment for infection, and metabolic and toxic abnormalities, as well as consideration of medication side effects. Any neurologic deterioration or signs of fluctuating mental status should trigger rapid assessment for possible etiologies of worsening (14).

Management of Malignant Cerebral Edema:-

The management of malignant cerebral edema in large infarctions has historically proved problematic. Clinical signs of brain edema from large supratentorial cerebral infarctions typically begin with a decreased level of consciousness, followed by upper brainstem signs and involvement of anterior and posterior cerebral arterial territories. Malignant cerebral edema typically presents within the first 5 days, including one-third of cases within less than 24 hours, and portends a poor prognosis, with fatality approaching 80% regardless of medical management . Pooled analyses from three European randomized clinical trials showed that decompressive surgery performed within 48 hours of stroke onset reduces mortality (78% vs. 29%) and increases the likelihood of achieving a favorable 1-year outcome, defined as Modified Rankin Score of 4 or less, when compared to usual medical management (75% vs. 24%). To place this in perspective, a modified Rankin of 4 indicates moderately severe deficits: inability to walk or attend to activities of daily living (ADLs) without assistance. Early decompressive surgery also led to doubling of chances to recover to a Modified Rankin Score of 3 or less by 1 year; a score of 3 indicates moderate disability, ability to walk without assistance. Notably, chances of surviving with severe disability (Score 5, bedridden, incontinent, requiring constant nursing) were not different for decompressive surgery versus usual medical care (4% vs. 5%). Note that these favorable outcomes for decompressive surgery come from studies that employ strict eligibility and exclusion requirements, including age 60 years or less, NIHSS greater than 15 in the setting of more than 1/2 MCA territory infarction, and with no space-occupying hemorrhagic lesions, fixed dilated pupils, or other major illnesses that could affect outcomes. These factors must be taken into consideration when making decisions regarding early decompressive surgery for individual patients. (15).

Seizures :-

Any change in mental status, particularly episodic, should trigger evaluation for seizures. Incidence of seizures is reported at 9% for ischemic stroke. By contrast, seizures are reported in one-third of intracranial hemorrhage cases,

and clinical studies suggest that more than half of these are electrographic only, and are not accompanied by clinical signs or symptoms of seizure). Specifically, continuous EEG recording has revealed seizures in up to 36% of lobar intracranial hemorrhage; contrary to conventional thinking, convulsive or nonconvulsive seizures are reported in 21% of subcortical intracranial hemorrhage cases, and they are linked to increased hemispheric mass effect and poorer outcomes. Seizures increase cerebral metabolic demands and intracranial pressure; generalized seizures can increase body temperature. All these factors can potentially worsen neurologic status and extend brain infarction in individuals with ischemic or hemorrhagic stroke, conditions in which cerebral auto-regulation is already impaired. Hence, seizures must be treated urgently in the setting of stroke. (16). Likewise, suspicion of subclinical seizures warrants bedside electroencephalography and monitoring, along with rapid and aggressive anticonvulsant therapy, if seizures are diagnosed.

Infection:- is known to be a prothrombotic trigger mechanism in as many as 25% to 33% of ischemic strokes, and has long been recognized as an etiology for clinical worsening in the setting of acute stroke. Therefore, survey for infection is recommended at the time of initial stroke presentation, and comprehensive infection evaluation should be conducted if clinical deterioration occurs. Aspiration pneumonia and urinary tract infections are the most prevalent and must be treated aggressively. Because acute infection is linked to a prothrombotic state, and elevated temperature can accelerate neuroexcitotoxicity, both fever and infection must be aggressively treated in the acute stroke setting to protect the brain from further ischemic damage.

Dysphagia, Aspiration Risk, and Nutritional Management :- One of the major dangers following stroke is aspiration, which may be silent, resulting in aspiration pneumonia. Dysphagia occurs in nearly half of hospitalized stroke patients and strongly predisposes to risk for aspiration pneumonia. Notably, the presence of a gag reflex is not indicative of safety in swallowing. Therefore, patients should be kept NPO until a bedside evaluation can be performed. Speech and language therapists play a significant role in this evaluation, and a videofluoroscopy swallowing study is recommended if bedside screening reveals abnormalities. If the patient is deemed to be at risk of aspiration, a dysphagia therapy program should be provided, optimally in consultation with a speech/language professional, as this has been shown to reduce pneumonia in the acute phase of stroke. Individuals at high risk for aspiration pneumonia may require nasogastric feeding tubes or percutaneous endoscopic gastrostomy (PEG). There is controversy regarding which is safer and more effective. While prior reviews provided some initial suggestions that PEG might be more efficacious, emerging evidence based on meta-analyses of 15 prospective studies suggests that nasogastric tubes are not associated with higher death rates, as previously believed. Hence, best clinical judgment must be used until the results of further clinical research are available. Dysphagia also identifies individuals who are inherently at greater risk for

developing malnutrition, which is reported in 15% of stroke cases at the time of initial presentation, and doubles to 30% across the first week of hospitalization .Because malnutrition is linked to poorer clinical outcomes, ongoing monitoring of nutritional as well as hydration and electrolyte status becomes an important component of clinical pathways for stroke, particularly in individuals with dysphagia and compromised oral intake (17).

Anti thrombotic and Anticoagulant Therapy :-

Aspirin is the anti-platelet agent that has been most extensively studied in the management of acute stroke. The combined results of two large acute stroke trials demonstrated a modest benefit in mortality and disability when aspirin therapy was initiated within 48 hours of stroke . This led to a recommendation of instituting aspirin at a dose of 325 mg within the first 48 hours after stroke. For patients who have received rt-PA, aspirin is not recommended within 24 hours of rt-PA administration. The efficacy of other anti-platelet agents as mono-therapy or in combination with aspirin is not well established for acute stroke. A 2007 study compared clopidogrel plus aspirin to aspirin alone given within 24 hours of onset of minor stroke (NIHSS < 4) or transient ischemic attack (TIA) . There was a 7% recurrent stroke incidence in the combined group, compared to 11% in the aspirin group. However, this did not reach statistical significance. The recent CHANCE trial demonstrated that the combination of aspirin and clopidogrel initiated within 24 hours of stroke or TIA was superior to aspirin alone in preventing recurrent stroke, without an increase in hemorrhagic complications, in a Chinese population. A similar study (POINT) comparing the combination of aspirin and clopidogrel to aspirin alone is currently underway in North America. Combinations of antithrombotic agents are generally not recommended for long-term secondary stroke prevention, as they are linked to increased bleeding risks. Anticoagulants are generally avoided in the treatment of acute stroke. Numerous studies have shown that although the use of heparin or heparinoids in the management of acute stroke results in a decrease in the risk of early recurrence of stroke, there is an increased risk of hemorrhagic complications, including symptomatic intracerebral hemorrhage, with the use of anticoagulants .An example of this is seen in Figure 5 . Historically, heparin had often been used acutely in patients with suspected cardioembolic stroke . (18) .

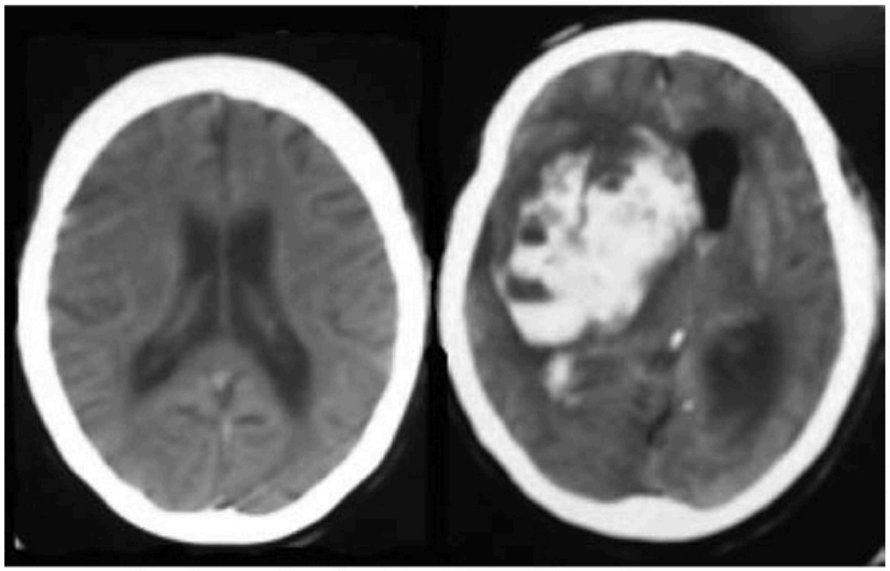


FIGURE .5. Patient presents at 10 a.m. with left hemiparesis, and the scan on the left is obtained. Heparin was started. At 4 p.m., the patient becomes obtunded, and the scan on the right is obtained.

Early Rehabilitation Care and Mobilization :-

Consensus recommendations support early mobilization in appropriate medically stable subjects . While medical instability can limit the scope of rehabilitation early on, there are numerous physiological reasons for early care emphasizing mobility, including prevention of deep venous thrombosis, pressure ulcers, autonomic deconditioning, skin and lung infections, contracture formation, and muscular wasting. Muscular wasting can be rapid and devastating for functional recovery, particularly in frail elderly subjects. For those patients incapable of volitional muscle activation because of altered consciousness or severe motor deficits, early range-of-motion exercises and/or appropriate splinting as indicated to reduce contracture development, along with change of body positioning and other strategies to minimize skin pressure and friction, are recommended to minimize common post-stroke complications of contractors and pressure ulcers, respectively. For individuals with cardiopulmonary stability with higher neurologic function, resumption with appropriate supervision and training in basic mobility, self-care, and socialization skills is fundamental to early comprehensive rehabilitation care .Involvement of family and caregivers, including provision of structured written materials mapping rehabilitation plans and issues, is strongly recommended.

Prevention of Deep Venous Thrombosis :- Patients with stroke often have deficits that impair their ability to ambulate safely, or even cause them to be conf i ned to bed. This immobilization, along with elements of the prothrombotic state that are associated with acute ischemic stroke, increase the risk for deep venous thrombosis and associated pulmonary embolism. Therefore, measures should be taken to prevent this complication. Consensus

recommendations strongly support the use of subcutaneous low-dose unfractionated heparin or low molecular weight heparin or heparinoids .Some evidence exists that the latter may have greater efficacy. For example, a study by Sherman and Alpers indicated a 43% improvement in venous thromboembolism in patients treated with the heparinoid enoxaparin given 40 mg subcutaneously daily, compared to subcutaneous unfractionated heparin given twice a day.This is consistent with meta-analyses concludingthat both unfractionated heparin and low molecular weight heparin/heparinoids are partially effective in reducing deep venous thrombosis, but some evidence suggests that low molecular weight heparinoids may be more effective .As previously stated, heparin should not be used during the initial 24 hours of postthrombolytic therapy. Intermittent pneumatic compression devices (or elastic stockings) can also be used to help prevent deep vein thrombosis in patients with contraindications to anticoagulants, and for individuals presenting with acute intracranial hemorrhage; stable patients with intracranial hemorrhage may be switched over to low-dose subcutaneous heparin as early as the second day postevent. Aspirin may provide some mild benefit, and is safe to use in combination with low-dose heparin. Regardless of the medical coverage, early mobilization (preferably in collaboration with physical therapy consultation) and walking are important and can significantly reduce the risk for venous thrombosis (19).

Skin Care and Prevention of Pressure Ulcers:-Pressure ulcers occur in approximately one-tenth of hospitalized stroke patients and one-fourth of those in nursing homes. Individuals at particular risk are those with mobility deficits of greater severity and medical conditions that compromise skin vascular integrity (including diabetes and peripheral arterial occlusive disease), those with urinary incontinence, and frail elderly patients with low body mass. Nursing pathways for stroke employ daily monitoring of skin integrity, and scheduled care including turning, proper positioning, and other appropriate methodologies to reduce the pressure and friction that propagate pressure ulcer formation . This includes the use of pressure-relief ankle-foot orthoses as a means to prevent contractures and pressure sore development. Consistent with clinical rehabilitation practice guidelines, early physical therapy assessment and care to optimize recovery of mobilization are also recommended to reduce the longitudinal risk profile for skin breakdown and pressure ulcer development.

Incontinence :- Urinary incontinence is a prevalent early problem after stroke, occurring in about half of hospitalized cases, and decreasing in prevalence to 20% in the chronic poststroke recovery period. Factors increasing the predisposition to urinary incontinence are similar to those for pressure ulcers: greater stroke severity, diabetes, and advanced age. Because of the extremely high prevalence of urinary incontinence (and often fecal incontinence) in individuals with moderate to severe stroke, indwelling catheters are often used during the acute stage. This can facilitate fluid

management and reduce the risk for skin breakdown. However, continued indwelling catheter usage for more than 48 hours predisposes to infection. During acute stroke hospitalization, assessment for urinary retention should be conducted via catheterization or bladder scan, urinary volume and control assessed, and dysuria documented. Some evidence exists that silver alloy-coated catheters may have fewer complications. Regardless, it is optimal to discontinue indwelling catheter use after 48 hours, and to employ an individualized bladder training program with prompted voiding training for appropriate cases . Similarly, bowel incontinence is common after stroke and can be associated with an increased risk for skin breakdown and infection complications. Patients should be carefully assessed for presence, pattern, and etiology of fecal incontinence, including consideration of mental status and neuromotor control of sphincter function, diarrhea, or constipation with diarrhea around a hardened stool mass, medication, and potential infectious complications that can increase the risk for fecal incontinence. Physical therapy to optimize mobility recovery can be useful, as physical activity can influence gastrointestinal transit time. Maintaining skin cleanliness and integrity of the perineal area is crucial, along with attention to dietary fiber content, and implementation of a time-structured regular bowel program and associated medications as clinically indicated. (20).

Cognitive Function and Communication :-

Global alterations in mental status, as well as a spectrum of specific neuropsychologic syndromes, are highly prevalent during the acute phase of stroke. Approximately one- third of stroke patients have a globally altered mental status during their acute hospitalization that can influence all other cognitive and communication assessments and limit early rehabilitation participation. The first item of the NIHSS categorically documents consciousness level. Those with stupor (or coma), or who fluctuate between drowsiness and stupor, regardless of cardiopulmonary stability, are best managed in a more intensive care setting, with frequent examinations documenting the specific stimuli needed to produce arousal. This enables a more rapid detection of fluctuating or progressing strokes and triggers urgent evaluation pathways. All patients with adequate alertness require evaluation for visual, motor, and sensory hemineglect syndromes, aphasia and apraxia, memory deficits, and impaired executive function—all factors that influence acute and longitudinal rehabilitation care pathways. Some symptoms, such as denial of deficit and hemineglect syndromes, particularly in visual and motor domains, also lead to challenges for the therapist and safety concerns, as the patient may not realize there is a dysfunction. These and other prevalent post-stroke neuropsychologic syndromes should optimally be identified during the acute hospitalization period and their significance explained to caregivers and future rehabilitation providers to optimize continuity of care. Assessment of communication skills, including speech, comprehension, repetition, reading, and writing, with speech/ language therapist consultation is a standard of care.

Early initiation of speech therapy, including visual communication aids, may be useful to facilitate patients' interaction with staff for routine care, and for socialization with family. Other elements of neurocognitive health that are often overlooked during the acute hospitalization stage are mood-and sleep integrity. Approximately one-third of stroke survivors develop some form of depression, which in many cases can be identified even during acute care hospitalization. Early recognition and management of depression is requisite to optimizing long-term rehabilitation outcomes. Sleep disordered breathing is also highly prevalent after stroke, particularly during the acute and subacute stroke recovery period, where fragmented sleep architecture and/ or apnea have been reported in more than half of patients (21). A particular concern is obstructive sleep apnea that is linked to increased stroke risk and prothrombotic state, and may be associated with or exacerbate other neuropsychologic issues such as fatigue, depression, and memory impairment: factors that can complicate rehabilitation and recovery . Individuals fitting the profile for sleep disordered breathing may be screened using nocturnal pulse oxymetry, or further evaluated by polysomnography as clinically indicated. Many acute stroke patients have a disturbed sleep–wake cycle, particularly when in intensive care units. Approaches to improving sleep hygiene include transferring the patient from the intensive care unit as soon as possible, and providing a quiet environment with dark during the night and sunlight during the day to facilitate return of more normal circadian patterns. Selected medications properly timed may be used to facilitate sleep (e.g., trazadone, chloral hydrate), to try to avoid regular use of major sedative-hypnotics and antipsychotics, which can contribute to confused states, particularly in the elderly, and may further alter sleep architecture. Because abnormal sleep architecture is common in acute stroke, and given increasing evidence suggesting that sleep is critical to memory consolidation and, hence, may facilitate sensorimotor recovery in the rehabilitation setting, careful attention to sleep hygiene should be addressed early on .In summary, a diversity of neurocognitive and communication deficits, as well as sleep disturbances, can complicate early stroke management and ongoing rehabilitation care. A summary of all cognitive and communication deficits and neuropsychologic syndromes, including depression and sleep disorders, that are diagnosed during acute hospitalization should be discussed with the family and outlined to subsequent care providers to optimize continuity of stroke care. (22).

Screening for Risk Factors :-

Risk factors for stroke include hypertension, which increases both small and large vessel arterial atherothrombotic risk, atrial fibrillation, and selected other cardiac arrhythmias, extracranial carotid and intracranial large vessel stenosis, cardiomyopathy, hyperlipidemia, vasculitis, cigarette smoking, hypercoagulable states, diabetes, syphilis, elevated C-reactive protein, and elevated homocysteine levels. A timely evaluation of these risk factors is

recommended in patients with acute stroke and TIA. Here we present a brief overview of a few of these risk factors and secondary stroke prevention measures, which are covered in more detail in later chapters.

Carotid artery stenosis is an important cause of ischemic stroke. Carotid ultrasound may be used for screening patients, as it has a sensitivity of approximately 85%, compared to digital arteriography. In combination with MR angiography, the sensitivity of detecting carotid stenosis improves to close to 100%. CT angiography is also helpful in assessing carotid lesions, with a sensitivity of 88% to 98% depending on the study. If a question remains regarding the lesion, catheter arteriography may be necessary. These studies are also needed to evaluate for less common conditions such as arterial dissection. In patients with carotid stenosis greater than 70%, the North American Symptomatic Carotid Endarterectomy Trial indicated that carotid endarterectomy reduced the risk of ipsilateral stroke from 26% to 9%, compared to medical management at 2 years .There was no significant change between the groups for less severe stenosis. The surgical/ arteriographic risk for these procedures was less than 3%. For patients who are at higher risk, the benefits of endarterectomy compared to medical therapy would be less, perhaps indicating that medical management would be preferable. The greatest benefit occurred when surgery was performed within two weeks of symptom onset. Carotid artery stenting is an alternative, less invasive method of revascularization. The CREST trial compared stenting to endarterectomy in patients with asymptomatic and symptomatic carotid stenosis, and found no difference in the composite endpoint of stroke, myocardial infarction, or death .There was a higher risk of stroke in the stenting group, and a higher risk of myocardial infarction in the endarterectomy group. Notably, younger patients had fewer events with stenting, whereas older patients did better with endarterectomy. Carotid artery stenting is also suitable for patients who have contraindications to surgery, such as prior radiation treatment to the neck or lesions that cannot be approached surgically. Hyperlipidemia is a risk for cardiovascular disease and, to a lesser degree, cerebrovascular disease. Current guidelines recommend treatment with HMG-CoA reductase inhibitors (statins) for patients with clinical atherosclerotic cardiovascular disease, including stroke or TIA. High-dose atorvastatin has been shown to decrease the risk of recurrent stroke .The benefit of statins after stroke may not be due solely to cholesterol reduction, as they also have effects on CBF, endothelial function, and anti-inflammatory properties. The use of statins for neuroprotection is currently being investigated in humans. Cardiac monitoring is essential for stroke patients for detection of atrial fibrillation and other cardiac arrhythmias, at least for the first 24 hours after stroke. The risk of atrial fibrillation increases with age and comorbid conditions such as congestive heart failure, hypertension, and diabetes. More prolonged monitoring on an outpatient basis increases the chances of detecting paroxysmal atrial fibrillation. The use of anticoagulants significantly decreases the risk of thromboembolism in patients with atrial fibrillation. Warfarin (23).

decreases the risk of thromboembolic events by two-thirds ,and the newer oral anticoagulants, such as dabigatran, rivaroxaban, and apixaban, have similar, or reduced, rates of thromboembolic events compared to warfarin . Aspirin decreases the risk slightly, but is significantly less effective than warfarin. Cigarette smoking is another major modifiable risk factor. All smokers should receive counseling and education regarding the importance of smoking cessation. Several agents and techniques are available to help patients with thisendeavor. They include nicotine patch and gum, hypnosis, and pharmacological agents such as varenicline and bupropion.

DISPOSITION AND DISCHARGE PLANNING:-

Decisions regarding the level of care for ongoing rehabilitation, particularly whether intensive inpatient rehabilitation is needed, should be made by the primary medical or neurologic team in consultation with rehabilitation providers. Three criteria influence the triage decision: 1. The premorbid and current functional statuses of the stroke survivor 2. The psychosocial and financial systems to support the stroke survivor in the community 3. The conditions of third-party reimbursement The first criterion is heavily weighted by the recommendations of the physical, occupational, and speech/language therapists. Consideration must be given to whether the candidate has adequate physical and neurocognitive capacity to perform basic ADL functions, including mobility with safety using the appropriate assistive device and/or orthosis. For individuals with mobility deficits and elevated fall risk, a home assessment may be recommended to optimize safety and facilitate ADL functionality. Some individuals may require the capacity to maintain their own instrumental ADL functions, such as banking, shopping, and cooking, for independent functioning. This involves higher levels of communicative and cognitive skills for home management, community living, health management, and the ability to react safely and correctly to emergency situations. Hence, instrumental ADL status must be ascertained prior to discharge, and a follow-up plan for repeated assessment made in the event of discharge to the community. Psychosocial and financial systems are essential to support the stroke survivor if he or she wishes to return to the community. If the caregivers are ready, willing, and able to assist or supervise the stroke survivor in the community, the stroke survivor may be admitted to an inpatient rehabilitation facility. If the stroke survivor has inadequate support systems to return to the community, it may be prudent to transfer him or her to a less intensive environment, to give the individual more time for spontaneous improvement. If the stroke survivor remains at an assisted functional level that cannot be supported in the community, he or she will ultimately be transferred to a long-term care facility. The final component of the rehabilitation triage decision rests, ultimately, upon the type of third-party payer policy under which the stroke survivor is covered. Although most insurance policies carry contingencies for different levels of rehabilitation care, some may limit the amount of inpatient and/or outpatient coverage per

diagnosis. Some may not have provisions for specific levels of rehabilitation. Some may force the stroke survivor to pay for a portion of his or her rehabilitation hospital bills. Some may limit their networks to specific inpatient rehabilitation facilities. In any case, it is imperative for the stroke survivor and his or her caregiver to review the insurance policy to ensure proper coverage in the event of a catastrophic event such as stroke. It is equally essential for the case manager to review the policy and confirm benefits before transferring a stroke survivor to an inpatient rehabilitation facility, to ensure that the third-party payer will pay for the rehabilitation stay and to minimize the financial liability for the stroke survivor and caregiver. A comprehensive rehabilitation follow-up plan should be set in motion before any community discharge. Attention should be given to therapeutic modalities and prevention of post-stroke complications. Other factors such as return to work, driving, sexual function, adaptive equipment, social adjustments, and planning free-living physical activity and health-promoting exercise are generally managed in the outpatient environment and are dealt with in other chapters. (24).

TRANSIENT CEREBRAL ISCHEMIA OR MILD STROKE SYMPTOMS :-

TIAs were previously defined as stroke symptoms that subsided within 24 hours. With the advent of rt-PA treatment, which requires treatment within 3 hours, that definition has been modified. Indeed, MRI studies with diffusion weighted imaging have indicated that more than half of the patients whose symptoms lasted more than 60 minutes actually have areas of infarction despite resolution of symptoms. Although the clinical symptoms in TIA may have subsided, it is important that the patient be thoroughly evaluated. Emerging guidelines for evaluation and acute medical management of TIA are now moving toward paralleling those of acute ischemic stroke .This approach toward the rapid management of TIA is being driven by an increasing recognition that many clinical events formerly classified as TIAs do, indeed, result in structural damage to the brain, and because the cerebrovascular event rate following TIA is dangerously high . Hence, TIA should be considered a medical emergency, with an imperative to optimize comprehensive secondary prevention strategies immediately. Meta-analyses of 11 prospective studies suggest that, following a TIA, the risk for recurrent stroke is 3.5%, 8%, and 9.2% at 2, 30, and 90 days post-TIA, respectively (53). Twenty-one percent of these strokes are fatal, with another 64% resulting in disability. Rapid evaluation and management of patients with TIA, therefore, offer an opportunity to intervene and prevent a significant number of strokes. Indeed, studies from Paris and Oxfordshire, United Kingdom, have indicated that early evaluation may decrease the risk of stroke in the 90-day period by as much as 80% (54). Notably, the mean time to comprehensive clinical evaluation in the EXPRESS prospective study of stroke prevention following TIA was less than one day, which underscores the importance of rapid care (54). The development of dedicated clinics that see the patient immediately and institute the evaluation of the TIA patient has been possible in some communities. This

approach also allows for the timely evaluation and institution of appropriate treatment, but is not widely available. (25).

In this research we will talk about Radiology images of stroke so ,' ' In the investigation of stroke and transient ischaemic attack (TIA) imaging is used to differentiate :-

- vascular from non-vascular lesions, such as tumours or infections
- ischaemic from haemorrhagic stroke
- arterial from venous infarction
- and to distinguish anterior and posterior circulation strokes to determine whether a tight carotid stenosis is symptomatic or not.
- In the future imaging may be used to show the extent of salvageable tissue in acute stroke before treatment. Imaging should be used to direct management. Investigation should be organised to resolve specific, preferably articulated, management dilemmas. This may include imaging to clarify and guide prognosis. (26)

COMPUTED TOMOGRAPHY :-

Computed tomography (CT) will differentiate infarct from haemorrhage up to at least five days after stroke. Recent haemorrhages are high density (white) and usually rounded and space occupying. Infarcts are usually low density (dark) and occupying a vascular territory with some swelling .In a patient with a stroke a normal scan excludes a haemorrhage and, in the absence of an alternative, infarction is assumed. (27). Intravenous contrast is not normally required and may cause confusion.There is some uncertainty as to how quickly small haemorrhages lose their characteristic whiteness and become isodense and then hypodense compared with normal brain, and so indistinguishable from an infarct. Certainly by 10 days, small haemorrhages will be indistinguishable from infarcts, and we have seen small haemorrhages disappear by seven days. Large haemorrhages remain visible as such for 2–3 weeks.

There is no "optimal" time to image stroke patients with CT and expect to show a definite infarct. Many infarcts do not become visibly hypodense until hours or even a day after the stroke, if ever.[3] Small infarcts are less likely to be visible than large ones—about 90% of patients with symptoms of a large cortical infarct (total anterior circulation infarct—TACI) have a visible infarct by 48 hours after stroke compared with about 40% of patients with lacunar (LACI) or small cortical infarcts (partial anterior circulation infarct—PACI).[28] Many larger infarcts are visible within six hours though the appearance is subtle and depends on how closely the scan is examined. Observer reliability for specific signs of early infarction (even among experts) is poor. Furthermore, between 10 days and three weeks after stroke, infarcts loose their hypodensity and become isodense with normal brain for several days to two weeks.[29]As the swelling has also gone by this stage, they may be completely invisible or their true extent impossible to determine. This phase

is termed as "fogging". By 2–3 months the infarct has usually become shrunken and is of cerebrospinal fluid density, and so is more readily visible. The "best time" to image stroke routinely with CT is as soon as possible; nothing is gained by waiting, and much may be lost. In practice the timing of scanning is influenced by what treatments are being considered and available resources.(30) In patients considered candidates for recombinant tissue plasminogen activator (rt-PA), CT scanning is mandatory to exclude intracranial haemorrhage or sizeable infarction before the thrombolytic drug is given.[31] Currently, in view of the potential hazards, there are good arguments for only considering the use of thrombolysis in a highly organised clinical environment, with appropriate dedicated care pathways established and expert CT scan review available immediately. In view of the relative lack of harm arising from a few doses of aspirin in patients who turned out to have a haemorrhage in the IST/CAST trials (International stroke trial/Chinese acute stroke trial), clinicians should consider starting aspirin pending the scan if they consider that haemorrhage is *unlikely* on clinical grounds, and obtain a CT scan the next day. Aspirin may then be stopped if the CT scan shows a haemorrhage. Clinicians and radiologists need to have established guidelines on scanning which reflect the available local resources (see below).

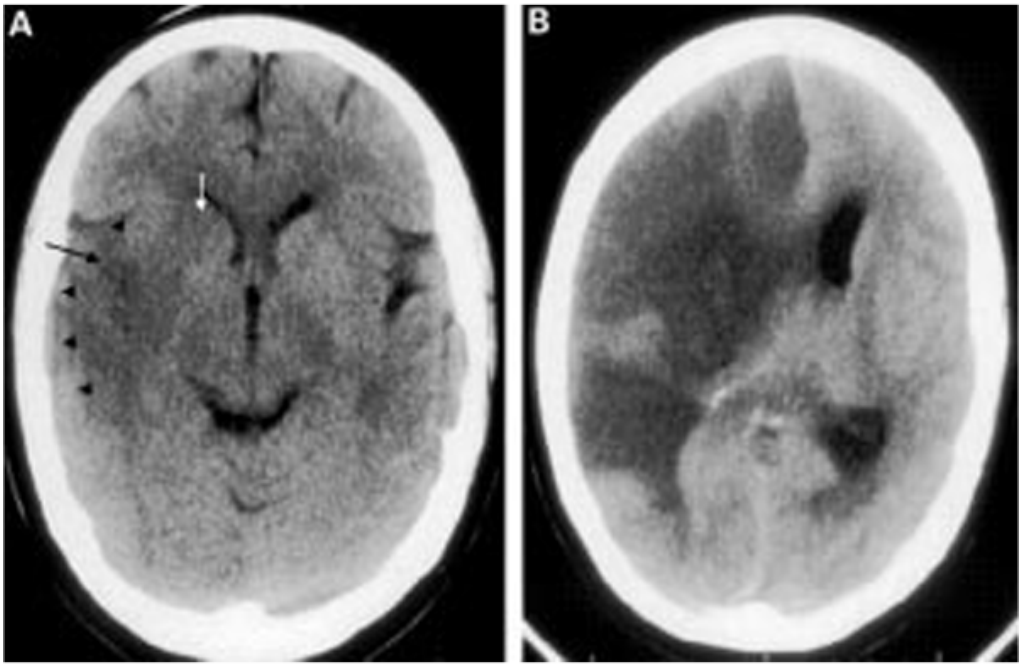


Figure .6. CT brain scan showing a right hemisphere total anterior circulation infarct (A) at four hours, and (B) at five days after symptom onset. Note on (A) the subtle signs of early infarction: loss of the basal ganglia on the right (white arrowÐcompare with the left where the caudate and lentiform nuclei are clearly visible), loss of the grey/white matter cortical differentiation (black arrowheads), a little swelling with sulcal effacement (black arrow and compare left side). On day 5 there is obvious hypodensity and massive infarct swelling with midline shift and obstruction of the left lateral ventricle.

Uses of CT in stroke :-

To differentiate vascular from non-vascular disorders .To differentiate infarct from haemorrhage .If in doubt, repeat scan a few weeks later without contrast .Contrast can be misleading and should only be used in special circumstances

Caveats on CT in stroke :- .

Identifies all parenchymal haemorrhage with near 100% accuracy only within 5±7 days of strokeÐthereafter small haemorrhages are indistinguishable from infarcts .Only about 50% of infarcts ever become visible .There is no ªoptimalº time for ªseeingº an infarct .ªSeeingº the infarct is not necessary to diagnose ischaemic stroke . (32).

Routine magnetic resonance imaging:-

The magnetic resonance (MR) appearance of acute haemorrhage is complex, but with the appropriate sequence, acute haemorrhage may be identified correctly even within the first six hours of stroke.[2] Routine (spin echo) MR sequences remain specific for haemorrhage indefinitely in 90% of patients.[33] In the remaining 10% who have had a definite intracerebral haemorrhage, the diagnostic features (that is, low signal caused by haemosiderin) are not visible on spin echo T2 MR, although cerebromalacea may be visible. Other MR sequences vary in their sensitivity to the presence of haemosiderin. In particular, the now frequently used fast spin echo T2 and proton density sequences are relatively insensitive whereas gradient echo sequences are the most sensitive (fig 7). If the identification of former haemorrhage is important, it is imperative that the radiologist is told otherwise an inappropriate sequence may be used. With the right sequence, MR will make this diagnosis in the majority of cases. The same principles of infarct evolution apply to routine MR imaging (T2, proton density and T1) as described for CT, including the problems of "fogging" and that a proportion of infarcts will never be visible on routine.

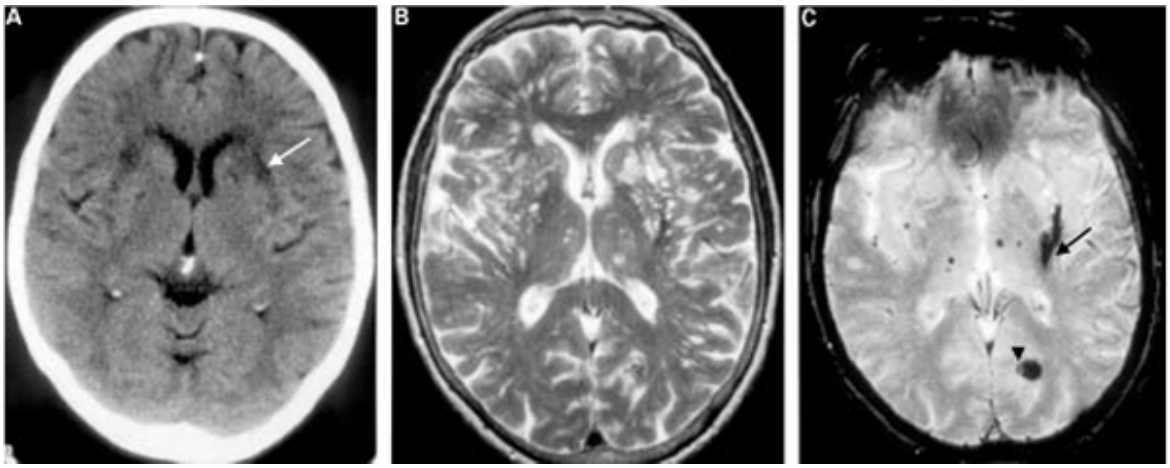


Figure .7. Brain imaging from a 75 year old woman presenting six weeks after a left hemisphere stroke. (A) CT scan. (B) Fast spin echo T2 MR scan. (C) Gradient echo MR scan. Note on the CT scan (A) the lucent areas consistent

with small vessel disease. The lucent area in the left hemisphere would be consistent with the symptoms and looks like an infarct. The MR (B, C) obtained on the same day shows not only more small vessel ischaemic changes (white spots) but also haemorrhage (dark area arrowed) in the left lentiform nucleus. The haemorrhage is more readily identified on the gradient echo MR (C) than on the fast spin echo T2 (B). On the latter, old haemorrhage may easily be overlooked. There are also several old microhaemorrhages visible on the gradient echo MR (black dots) and an incidental small calcified lesion in the occipital lobe (arrowhead).

[a]Advanced[o] magnetic resonance techniques :-

Infarcts may be demonstrated on MR with greater sensitivity than CT using certain sequences such as fluid attenuated inversion recovery (FLAIR) or diVusion weighted imaging (DWI). Unfortunately FLAIR also shows many more additional incidental white matter lesions that may simply add to confusion rather than help to identify the new lesion.Of all techniques diVusion imaging has the greatest sensitivity, but is not specific for infarction.[34] Encephalitis, multiple sclerosis plaques, and tumours may all appear of increased signal and be mistaken for an infarct by the unwary. On DWI, larger infarcts are visible more often than small ones, and a proportion of patients with a definite stroke never have a visible infarct; some patients who turn out to have a TIA have a relevant visible lesion on DWI when imaged within 24 hours of symptom onset.[35] Although there is considerable interest in the use of DWI and perfusion MR imaging to identify patients with salvageable tissue, this is still experimental. MR takes longer than CT (so may expose the patient to greater risk of aspiration while supine in the scanner), and may delay the start of treatment as most centres do not have the immediate MR availability required. DWI may be most useful clinically to identify positively the lesion site in patients with minor cortical or lacunar strokes, or to determine whether a patient with a previous infarct and worsening signs has developed a new infarct or not; DWI is likely to be positive up to a week at least after the stroke.[36]Perfusion imaging with MR is not in routine use. The most readily available method provides only relative, not absolute, perfusion measurements. Further work is required to improve the interpretation of this technique. MR spectroscopy provides information on metabolites, notably lactate, choline creatine, and *N* acetyl aspartate (NAA—found in normal functioning neurones). Improved spectroscopic imaging and faster image acquisition times may bring it into wider use, but currently its main application is in stroke research. The exceptions where it may occasionally be useful clinically are to distinguish tumours from infarcts where other imaging is ambiguous (a high choline content is found in tumours, whereas infarcts typically have reduced NAA and normal or reduced choline); or in rare metabolic conditions associated with stroke like MELAS (mitochondrial encephalomyopathy with lactic acidosis and stroke).

Uses of MR in stroke :-

To distinguish haemorrhage from infarct in patients presenting late after strokeÐbut only if the appropriate sequences are used . More often shows an ischaemic lesion than CT so may be more useful in ªdifficultº young patients with suspected stroke . In ªdifficultº strokes such as suspected venous infarction or carotid or vertebral dissections, as it may show the vascular anatomy also

Special clinical circumstances :-

infarcts are probably underdiagnosed as a cause of stroke. Increasing awareness leads to better recognition.[37]Venous infarcts become hypodense and swollen much more rapidly than arterial infarcts and more often contain central areas of haemorrhage. Additional features such as thrombosed venous sinuses (hyperdense sinus pre-contrast, or filling defect in the sinus post-contrast), or opacified paranasal sinuses or mastoids indicating probable infection as the cause of thrombosis, should be sought. MR shows these features more clearly, though they may be visible on CT. Distinguishing tumours from infarcts on CT (or MR) is not usually a problem, but occasionally slow growing tumours such as gliomas can mimic a small cortical infarct by appearing wedge shaped, involving cortex and adjacent white matter, being slightly hypodense, and not enhancing with contrast. Occasionally tumours may also present as a haemorrhage and the bleeding may be extensive enough to obliterate temporarily the underlying neoplasm on the scan. Time is a useful diagnostic tool; repeating the imaging will clarify diagnosis, as infarcts and haemorrhages generally get smaller whereas tumours stay the same or get bigger. Furthermore, patients who initially present with what seems like a straightforward stroke, but who do not behave subsequently as a typical stroke, should have a repeat scan to identify the occasional tumour or other non-vascular lesion. Encephalitis can occasionally mimic stroke, particularly in patients found unwell with diminished consciousness, focal neurology, and no available history of the onset. Imaging, either CT, MR or advanced MRwith diminished consciousness, focal neurology, and no available history of the onset. Imaging, either CT, MR or advanced MR techniques, does not always reliably distinguish between these clinical entities. Diagnosis depends on other assessment. Dissection of the carotid or vertebral artery should be suspected in patients with neck pain and a stroke. MR is best as it may show the vascular and the parenchymal lesion. A typical feature is narrowing of the flow void in the symptomatic carotid or vertebral artery due to a ring or cu$\mathcal{V}$ of high signal caused by fresh haemorrhage in the artery wall. Unfortunately this appearance can also be mimicked (more commonly in the carotid than the vertebral artery) by slow flow in the artery above a tight (atheromatous) stenosis, or proximal to a major intracranial arterial occlusion, so some caution is required not to overdiagnose dissection. In view of possible therapeutic implications, intra-arterial angiography should be considered if there is any doubt as to the diagnosis of dissection. CADASIL (cerebral autosomal dominant arteriopathy

with subcortical infarcts and leucoencephalopathy) causes prominent subcortical white matter abnormalities which may mimic multiple lacunar infarcts and atrophy, often in a relatively young patient. Imaging is supportive of the diagnosis. MR shows more detail than CT. MELAS (mitochondrial encephalopathy, lactic acidosis, and stroke) presents with stroke in younger patients. On CT or MR cortical infarct-like lesions are visible usually in the posterior temporal or occipito-temporal regions, often bilaterally and not strictly occupying atypical vascular territory.

Who should read the scan?

MR is complicated and should be left to neuroradiologists or occasionally general radiologists who have a particular interest in neuroimaging. Neurologists and stroke physicians are more used to reading CT scans as the technology has been available for longer, and they should be able to differentiate an infarct from a haemorrhage and to know when a lesion looks non-vascular. Gaining this experience takes time and requires looking *closely* at as many films as possible. Clinicians should be aware, however, that the signs of early infarction and small haemorrhages can be subtle and overlooked, and calcification may be mistaken for haemorrhage. Therefore clinicians may not be confident to base management decisions on their interpretation of the scan where the identification of early infarction or of subtle haemorrhage is particularly important (for example, before thrombolysis). This is particularly true in the case of trainees in neurology and stroke medicine who simply may not have had the opportunity to see enough scans to gain the requisite explanation.

What about the neck arteries?

Carotid Doppler ultrasound is the simplest, safest, quickest, and best way of assessing the carotid and vertebral arteries to identify atheromatous stenosis or dissection. However, although it looks deceptively easy in experienced hands and when the patient is normal, it is very operator dependent and there are numerous pitfalls for the unwary. Thus it should only be done by properly trained operators with a reasonable throughput of patients to maintain their expertise and where efforts are made to audit the results. Patients with lacunar strokes are less likely than those with cortical infarcts to have a stenosis; about 8% of lacunar stroke patients will have a tight stenosis in the symptomatic artery.[39] In some centres ultrasound is the only imaging of the neck done before endarterectomy, while others still rely on intra-arterial angiography for the definitive measurement of stenosis, or use MR or CT angiography. Intra-arterial angiography is risky in patients with symptomatic ischaemic cerebrovascular disease (4% permanent stroke, 1% death) and delays endarterectomy, and MR and CT angiography are no better than Doppler ultrasound in competent hands (though they may be alternatives where ultrasound expertise is lacking). Whatever is used, it is important that the imaging is reported by radiologists with an interest in carotid disease.

Use of guidelines and organising local investigations :-

Stroke is such a common condition that an average general hospital will admit one or two cases per day. Thus the investigation of stroke must be well organised, otherwise a backlog develops resulting in delayed, inefficient, and suboptimal patient management. Imaging resources (either money or scanner time) are not infinite. Inappropriate resource use also displaces patients with other (possibly more needy) diagnoses, and creates conflict and friction between different clinical departments and radiological service providers. These difficulties may be particularly true in hospitals where there is a large mixed general body scanning workload. Imaging resources in most parts of the country are still well below those required by the local populations. Various bodies have issued guidelines for the investigation of stroke.[40] Individual hospitals will need to determine how best to apply these guidelines in light of their own resources. Good communication between clinical and radiological departments is absolutely fundamental to this process.

Material and Method .:-

The patients were identified from The International Classification of Diseases, 10th Revision (ICD-10), codes with the diagnosis of nontraumatic intracranial hemorrhage (code I-60 subarachnoid hemorrhage, I-61 Intracerebral hemorrhage and I-62 other non-traumatic intracranial hemorrhage).

Criteria for inclusions into the study were: (1) All patients that were available in assessment of both CT imaging of the brain in PACS workstation and medical record data for review. (2) Patients who had undergone cranial surgery, the preoperative CT imaging was used for review. (3) Patients who had more than one CT scan, only the first CT scan was used for analysis.

Plain axial study of CT scan of the brain was performed with multidetector CT scanner (Light speed 16; General Electric Medical System, Milwaukee, Wisconsin, USA). Technique: 120 kV, 250 mA, and 5-mm section thickness. Coverage was from the skull base to the vertex by obtaining contiguous axial sections. The CT scan were searched from databases at a PACS workstation in Al-Nasiriyah Hospital and reviewed by two neuroradiologists.

The CT scan of patients who had non-traumatic intracranial hemorrhage (code ICD-10 = I 60, I 61, I62) and underwent CT scan the brain at *AL-Nasiriyah Hospital from November 2021 to April 2022* , Retrospective review was performed at a PACS workstation by two neuroradiologists in the interpretation of stroke CT scans and blinded to the clinical history and lateralization of symptoms. CT scan was described for abnormalities as the precise extent of intracranial hemorrhage, the size and location of the hemorrhage, ,, intraventricular hemorrhage, subarachnoid hemorrhage and subdural hemorrhage. Discrepancies in interpretation between two observers were resolved by consensus.

All CT examination was reviewed for the following findings:

The hyperdense lesion represented a hematoma but the hypodense represented infarction (Fig. 1). Hemorrhage were categorized as ICH, SAH, SDH and IVH (Fig. 2, 3).Classification of lesion location as either lobar (frontal, parietal, temporal or occipital), Thalamic-ganglionic hematoma (caudate, putaminal and thalamic), brainstem and cerebellum (Fig. 4-6). The size of the lesion using multiple sections, A useful method of measurement of hematoma volume on CT scans is to use the formula ABC/2, where A is the largest diameter, B is the diameter 90° to A and C is the approximate number of slices that show hemorrhage multiplied by the thickness of each slice, the authors divided the size of hemorrhages to small (less than 30 cm3), moderate (between 30-60 cm3), and large (more than 60 cm3) size. The prognosis of patients with hematoma depends heavily on the size of the lesion (Fig. 7). Blocking of CSF pathways causing hydrocephalus, obstruction of the foramen of Monro, the sylvian aqueduct or the outflow foramina of the fourth ventricle is common in patients with hematoma. (Fig. 8).

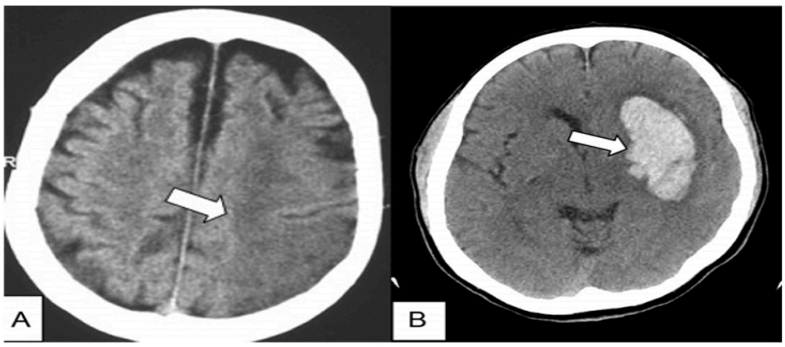


Fig. 1 A) CT brain scan of patient shows parietal infarction (arrow), B) Left basal ganglia hemorrhage (arrow).

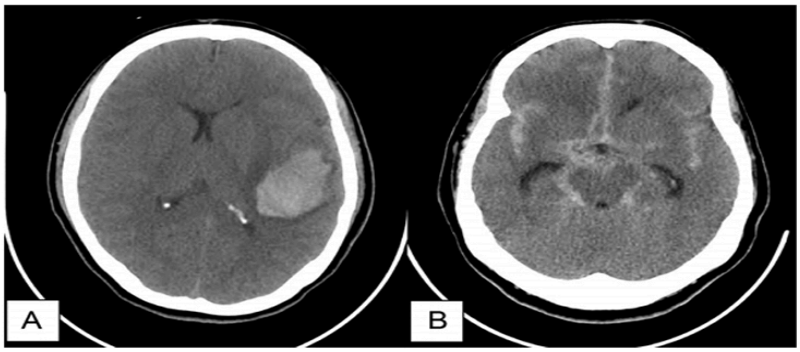


Fig. 2 A) CT scan revealed left temporal lobe intracerebral hemorrhage, B) An extensive of subarachnoid hemorrhage. This is seen as hyperdensity involving the basal cisterns with extension into the ventricular system.

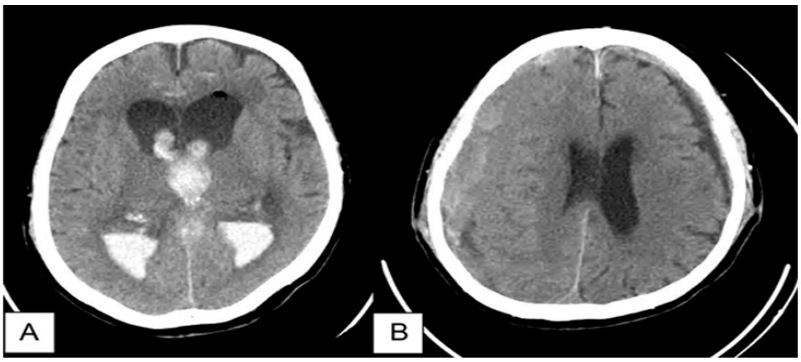


Fig. 3 CT brain scan of patient shows: A) Intraventricular hemorrhage bleed in to 3rd ventricle, B) Demonstrates isodensity of subacute right subdural hematoma and chronic hypodense of left subdural hematoma

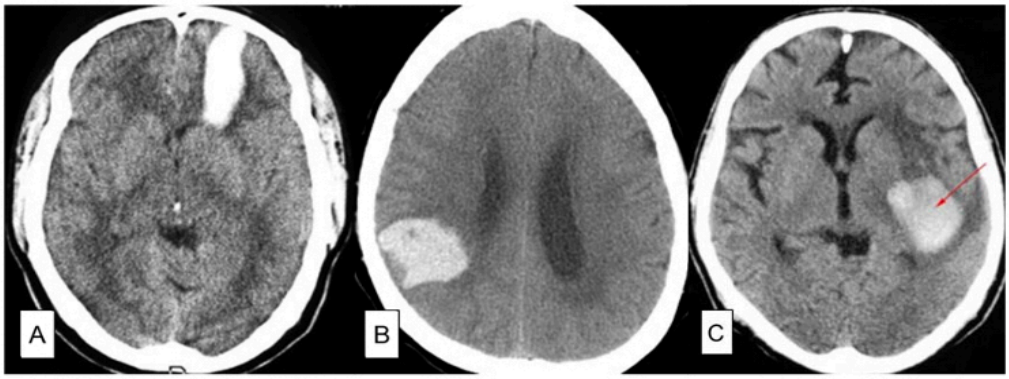


Fig. 4 CT image of Lobar hemorrhage: A) Frontal lobe intracerebral hemorrhage, B) Parietal lobe intracerebral hemorrhage, C) Temporo-occipital lobe intracerebral hemorrhage

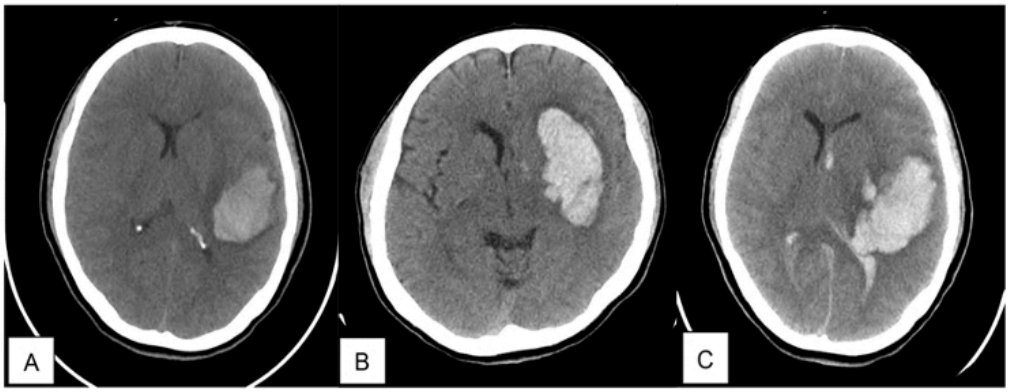


Fig. 5 Hypertensive hemorrhage in the basal ganglia. Hypertensive hemorrhage has a predilection for deep structures including the thalamus and basal ganglia, particularly the putamen and external capsule. Thus, it often appears as a high-density hemorrhage in the region of the basal ganglia: A) Lobar hemorrhage, B) Putamen hemorrhage, C) External capsule hemorrhage

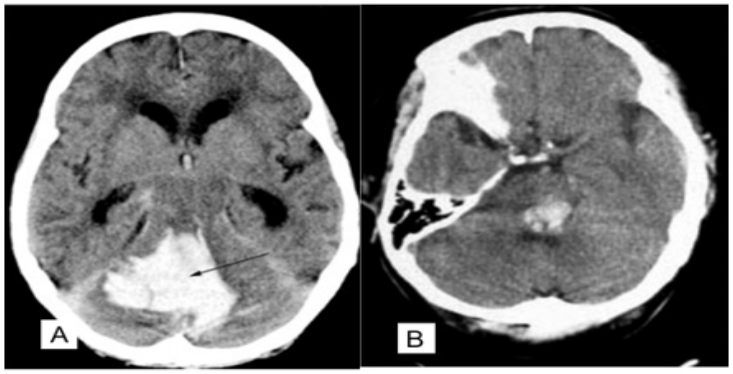


Fig. 6 CT scan shown: A) Cerebellar hemorrhage, B) Brain stem hemorrhage.

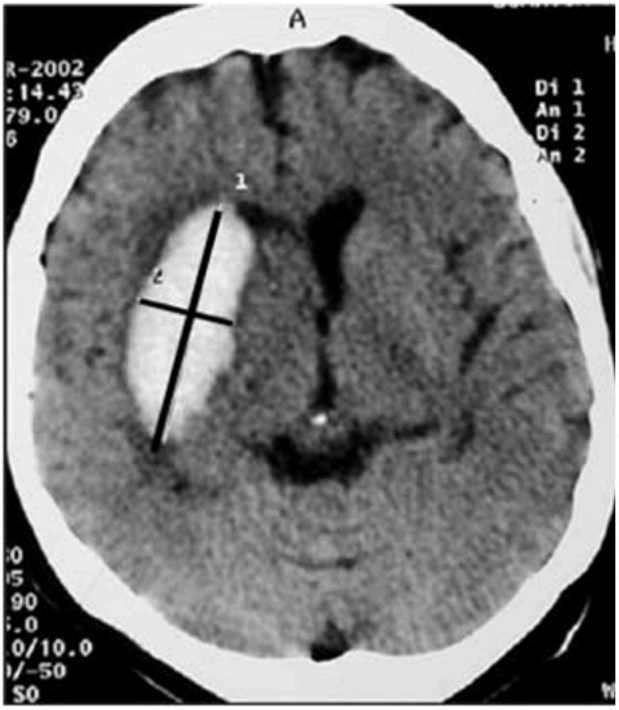


Fig. 7 The volume of the hemorrhage was calculated by using the “bedside formula” of Kothari et al: hemorrhage volume (in ml) = (A x B x C)/2, where: A = greatest hemorrhage diameter (in mm at CT), B = diameter 90° to A (in mm at CT), and C = approximate number of CT sections with hemorrhage x section thickness

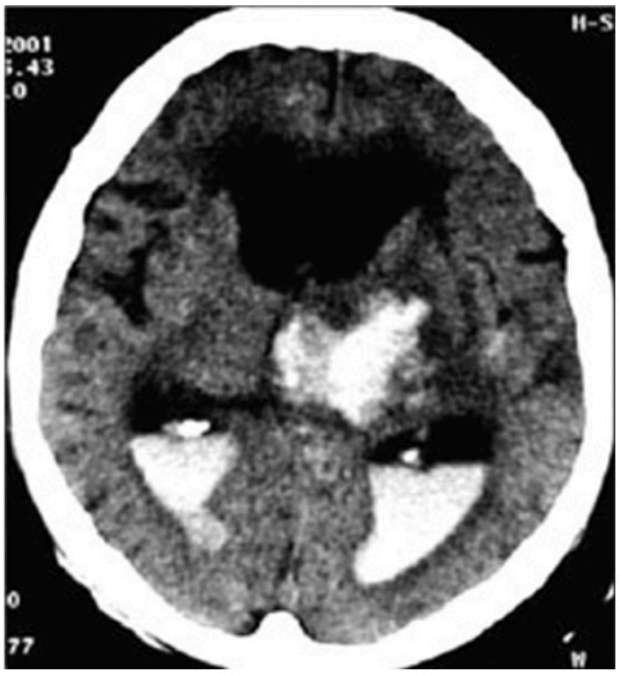


Fig. 8 CT brain presented blocking of CSF pathways causing hydrocephalus

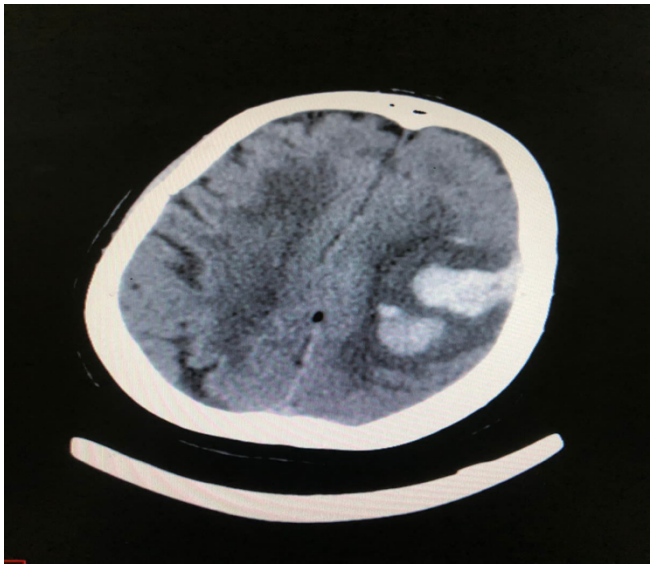
Statistical analysis :-

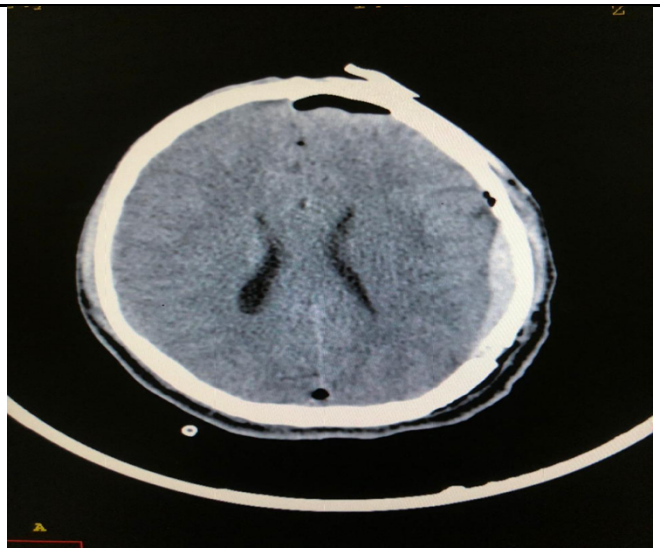
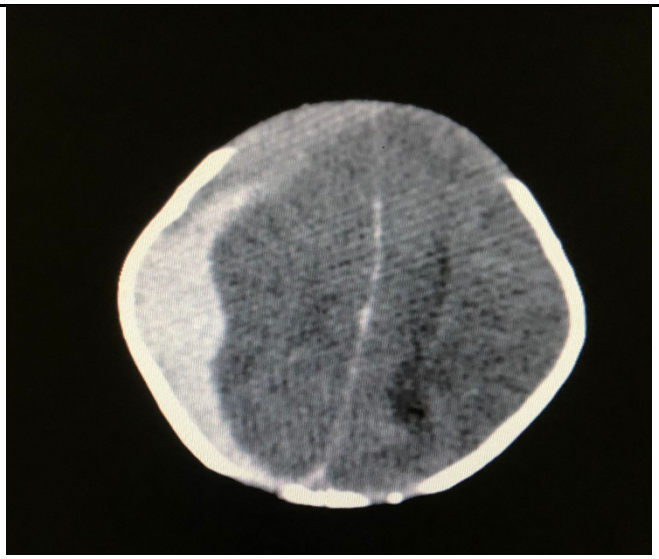
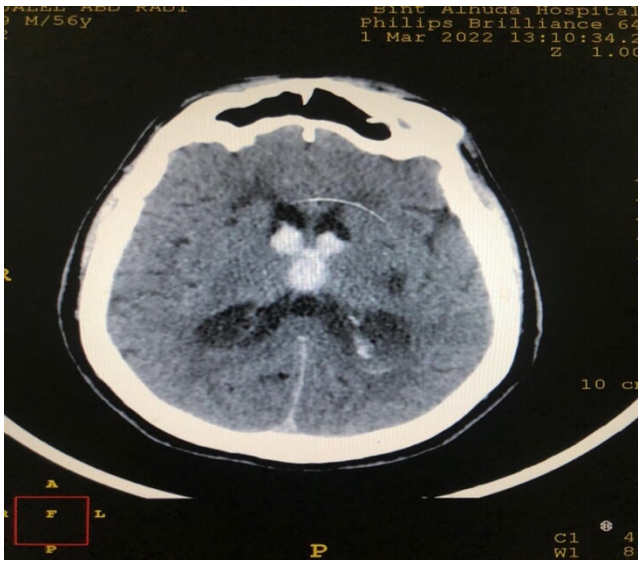
1. Baseline characteristics of the present study patients were summarized in terms of frequencies and percentages for categorical variables and continuous data was presented as mean + standard deviation (SD).

2. Agreement between interpretation of the two neuroradiologists were calculated with the Kappa test (K) of concordance. Concordance between observers was considered as: Poor agreement = Less than 0.20 Fair agreement = 0.20 to 0.40 Moderate agreement = 0.40 to 0.60 Good agreement = 0.60 to 0.80 Very good agreement = 0.80 to 1.00 3. Association between clinical data, CT finding data and mortality rate was calculated with Pearson's chi square. A value of $p < 0.05$ was considered statistically significant.

Results :-

One hundred patients (61 male and 39 female) ranging in age from 25 to 95 years (mean + Standard Deviation, 76 + 21.6 years) fulfilled the inclusion criteria. All 100 patients with non-traumatic hemorrhagic and ischemic stroke were classified into many types. There were comprised of 64 of cases (ischemic stroke) , 36 of cases (Hemorrhagic stroke) " 23 of them intracerebral hemorrhages, 3 of them subarachnoid hemorrhages, 4 of them subdural hemorrhages and 6 of them intraventricular hemorrhages". The relation between age group and stroke as summarized in Figure .12 .

Subacute left parietal lobe intracerebral hematoma	

Acute epidural hematoma in the left parietal convexity	
Acute subdural hematoma at right cerebral convexity	
Intraventricular hemorrhage	

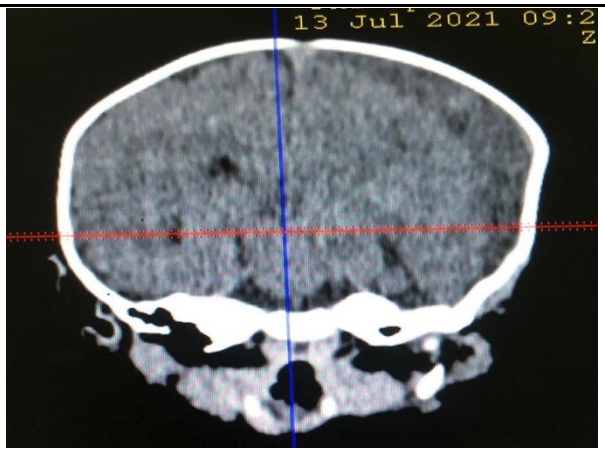
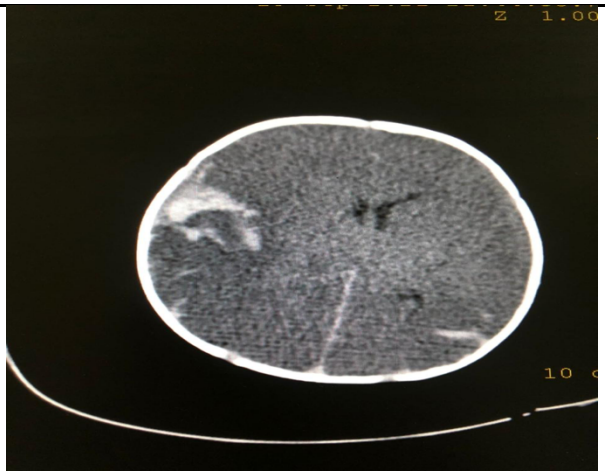
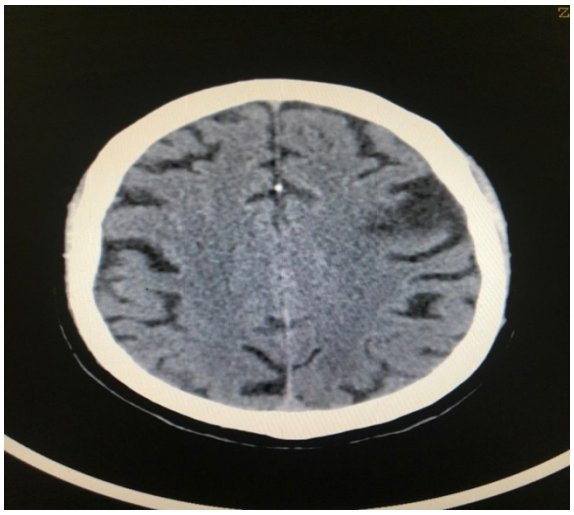
Chronic subdural hematoma at left cerebral convexity	
Subarachnoid hemorrhage	

Fig .9. CT- scan of Haemorrhagic stroke types , which taked from Radiology department of Al- Nasiriyah Hospital.

Acute Ischemic infarction at left frontoparietal region	

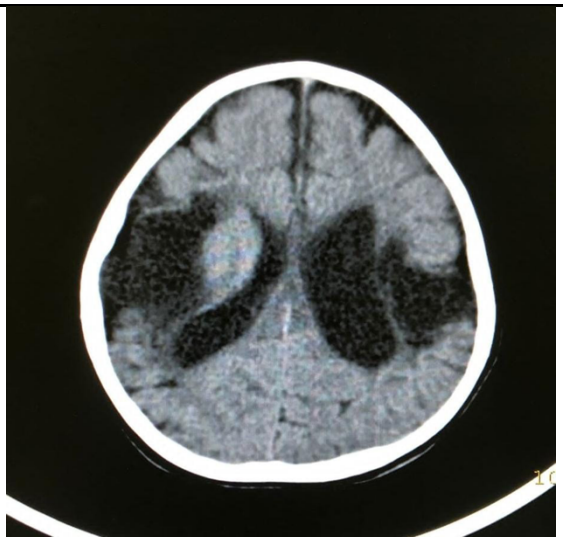
Bilateral parieto-temporal old ischemic infarction	

Fig .10. CT- scan of ischemic stroke (acute and chronic), which taked from Radiology department of Al- Nasiriyah Hospital.

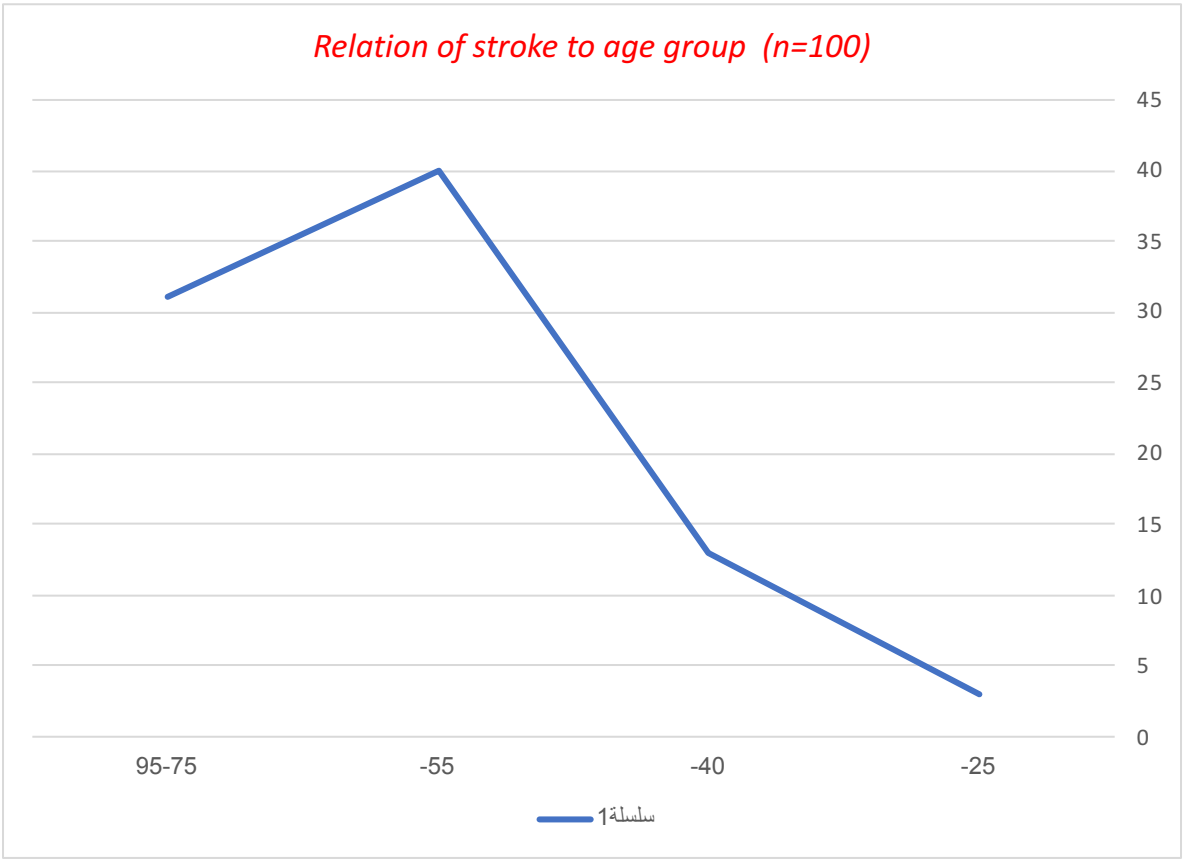


Figure .11 .relation between age group and stroke as summarized.

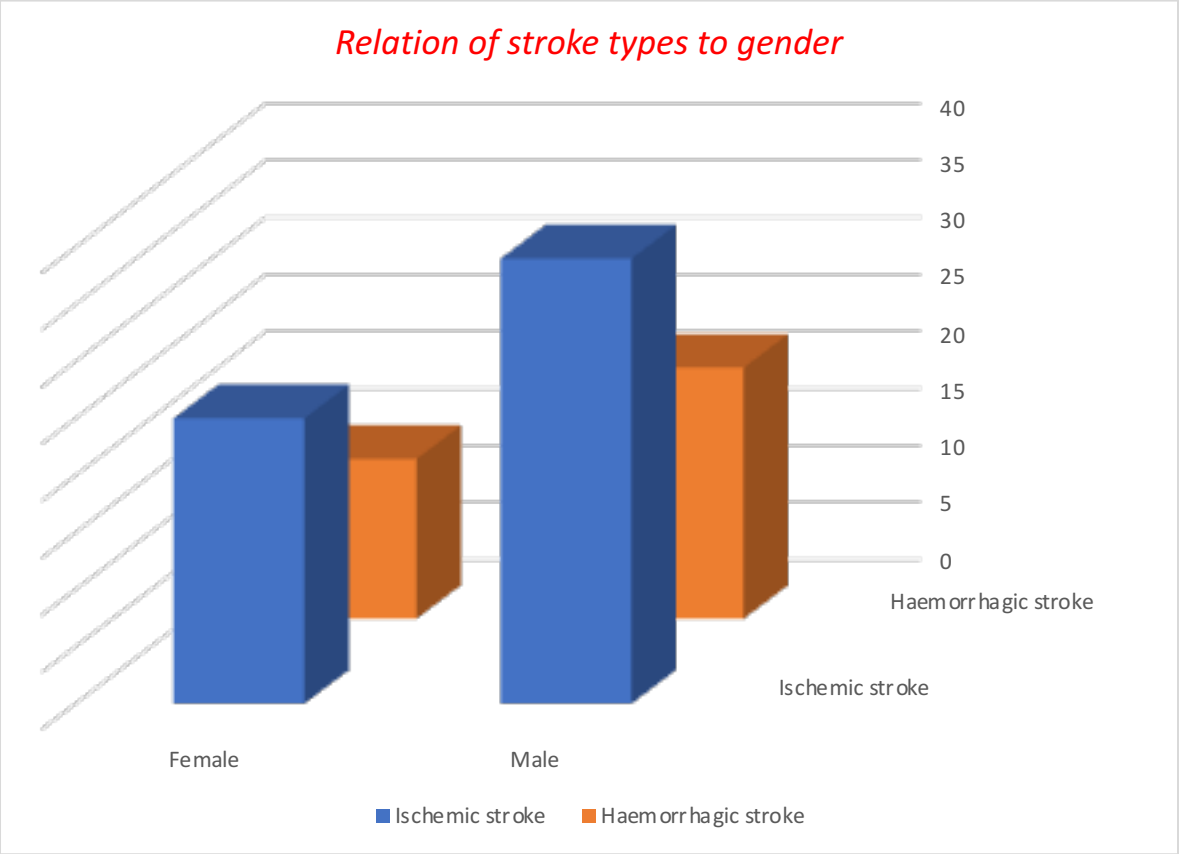


Fig . 12. Association between stroke and gender.

Risk factors relation to stroke were as follows: (86%) cases with hypertension, (61 %) cases with heredity and family history of chronic disease , (53%) cases with diabetes mellitus , (46%) cases with hyperlipedemia , (41%) cases with heart disease , (39%) cases with obesity , (38%) cases who were smokers, (26%) cases with sleep apnea , (24%) cases with physical inactivity, (17%) cases with metabolic syndrome, (9%) cases with poor diet.

A clinical presentation of the patients, each might have one or more signs or symptoms. (38 %) presented weakness and numbness, (21%) with severe headache, (11%) with vomiting, (10%) with confusion, (4 %) with seizure, and (13 %) were unconscious.

Regarding the duration time of onset, most cases presented in less than 24 hours period. (83 %) cases less than 24 hours, (17%) cases between 1-7 days and (2%) cases more than 7 days.

The Glasgow coma score (GCS) on admission, (34%) cases were scored between 3 to 8, (23 %) cases between 9 to 12 and (44%) cases between 13 to 15.

Outcome, (27 %) of patients died and (73 %) survived.

Types of hemorrhagic stroke	Percentage (n= 100)
intracerebral hemorrhages	23 %
intraventricular hemorrhages	6 %
subdural hemorrhages	4 %
Subarachnoid haemorrhage	3 %

Table . 3 . Types of hemorrhagic stroke and it's percentage from all 100 case.

Risk Factors	Ischemic stroke 64%	intracerebral hemorrhages 23%	intraventricular hemorrhages 6%	subdural hemorrhages 4%	Subarachnoid haemorrhage 3%
HT 86%	52 %	24 %	6 %	2%	3 %
Heart Disease 41%	19%	16%	2%	1%	3%
Poor Diet 9%	8%	1%	.....	.....	.....
Sleep Apnea 26%	13%	6%	3	1%	2
Obesity 39%	23%	9%	5%	2%	1
Physical In-Activity	14%	8%	2%	.....	.....
Smok-Ing 38%	17%	12%	5%	3%	1%

DM 53%	29%	18%	3%	1%	2
Hyper-Lipidemia 46%	31%	9%	3%	1%	2%
Age Group					
(25-) yrs	2%	1%	……	……	…….
(40-) yrs	7%	3%	1%	……	……
(55-)yrs	35%	14%	2%	3%	3%
(75-95) yrs	21%	5%	3%	1%	…..
Gender					
Male 61%	37%	19%	5%	……	……
Female 39%	25%	11%	3%		
Family History Or Heredity	34%	19%	6%	2%	2%

Table .4. Risk factors percentage of stroke subtype:-

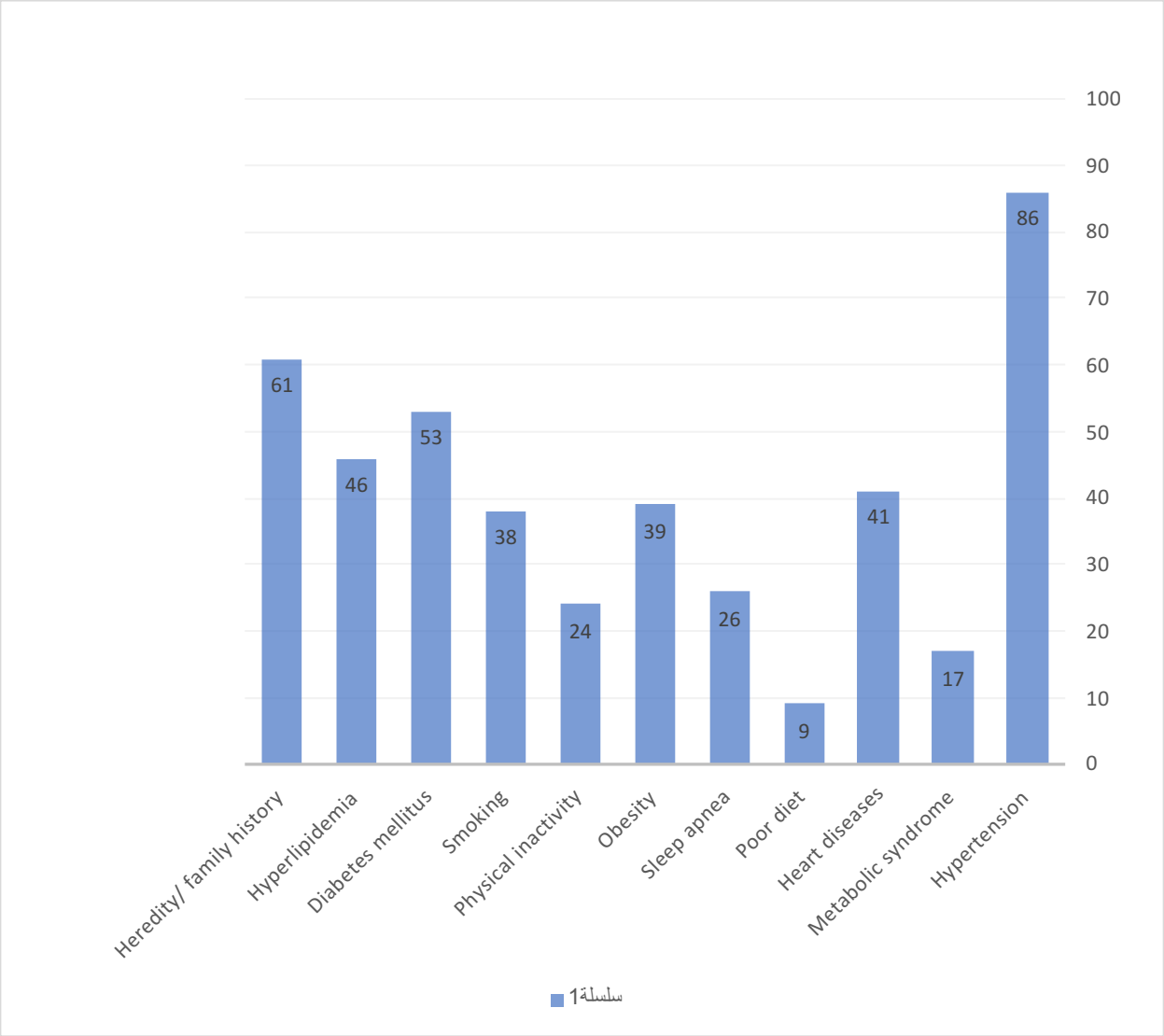


Figure.13. Risk factors percentage per 100 cases of stroke.

Interobserver agreement The CT findings, interpretation by two neuroradiologists, were calculated with Kappa test (K) of concordance. Concordance for the type of hemorrhagic stroke interpretation between observers was very good (K = 0.861). Result of interpretation by neuroradiologist 1 showed intracerebral hemorrhage 23 cases, intracerebral hemorrhage with intraventricular extension 2 cases, intraventricular hemorrhage 4 cases, subarachnoid hemorrhage 3 cases , subdural hemorrhage 4 cases. The neuroradiologist 2 shown intracerebral hemorrhage 24 cases, intracerebral hemorrhage with intraventricular extension 4 cases, intraventricular hemorrhage 3 cases, subarachnoid hemorrhage 3 cases, and subdural hemorrhage 4 cases . Agreement for location of hematoma in intracerebral hemorrhage interpretation between observers was very good (K = 0.866). Result of interpretation by

Correlation of GCS score and outcome shown score 3 to 8 had associated with death and score 13 to 15 associated with survival (p = 0.000). Hematoma size divided into three categories, in survived patients (77%) were small, (15%) medium and five (8%) large size. In dead patients, (46%) were small, medium and large was resembled (27%). Large size of hematoma association with death (p = 0.013). Intraventricular hemorrhage extension in

survival patients present (44%) cases, (56%) cases absent and in dead patients (72%) cases present Intraventricular hemorrhage extension, (28%) absent. Survived patients associated with absent Intraventricular hemorrhage extension and death associated with presence of Intraventricular hemorrhage ($p = 0.003$). (14%) cases with midline shift in survived patients, (18%) cases died, (15%) cases with ventricle dilated survived and (24%) cases died. (23%) cases were combined with midline shift and ventricle dilated in survived patients and (38%) died. No association between mass effect and survival or dead patients.

Discussion :-

Stroke is an important public heath problem in older age and can cause permanent neurological damage or even death if not promptly diagnosed and treated. According to World Health Organization data(41) estimates that in 2001 there were over 20.5 million strokes worldwide and up to 5.5million were fetal.

By these figures, stroke is an important disease for the Public heath system in Iraq . Neuro-imaging plays a crucial role in the evaluation of patients presenting with acute stroke symptoms. While patient symptoms and clinical examinations may suggest the diagnosis, the brain imaging study can confirm the diagnosis and differentiate hemorrhage from ischemia with high accuracy. Imaging of the brain using computed tomography is a helpful imaging modality to determine the type and severity of stroke particularly in hemorrhagic stroke). The aim of the present study was to evaluate the CT findings in non-traumatic hemorrhagic stroke patients and association of clinical data, CT finding data and clinical outcome. The present study in 100 cases of hemorrhagic stroke, the incidence of intracerebral hemorrhage, subarachnoid hemorrhage, intraventricular hemorrhage and subdural hemorrhage were 23 ,6 ,4 , and 3 cases, respectively. The present study shows that the mean age of patients who have nontraumatic hemorrhagic stroke is 6^{th}-7^{th} decade of life. Incidence of hypertension risk factor was found57% in ischemic stroke , 19% in intracerebral hemorrhage, 3% in subarachnoid hemorrhage, 2%in subdural hemorrhage and 6% in intraventricular hemorrhage, Hypertension is present in approximately 86% of stroke cases. The risk of stroke rises in proportion to blood pressure, for males as well as for females). However, the present study shows that hypertension is an associated risk factor in 86 patients out of 100 patients. In the present study weakness and numbness was the major presenting symptom of the patients in ICH group). Headache at onset was the most important clinical feature in subarachnoid hemorrhage followed by stiff neck: 50% of subarachnoid haemorrhage commonly caused by ruptured aneurysm. Ruptured aneurysm is suggestive when CT scan shows subarachnoid hemorrhage with hematoma located near the major branches of arteries of the circle of Willis. Subdural hemorrhage present headache). followed by weakness and vomiting in the same percent (18%), headache and vomiting most presented in intraventricular hemorrhage. There is no limitation in evaluation of attenuation of acute hematoma in the presented patients because of the estimated time from stroke onset to first medical contact less than 24 hours in all types of hemorrhage. This is also an important factor for CT scan in evaluation of hemorrhagic stroke due to 93% sensitivity if the scan time is within 48 hrs compared to 17%-58% if CT scan performed beyond 48 hrs(42).

The level of consciousness on admission was evaluated through the Glasglow Coma Scale (GCS). Level of consciousness is an important determinant of outcome in patients with hemorrhage as observed in the present study found a high percent in GCS score 13 to 15, 44% in intracerebral hemorrhage, 60% in subarachnoid hemorrhage, 54% in subdural hemorrhage and 50% in intraventricular hemorrhage. Patients were already in coma when admitted (GCS score less than or equal to 8) shown in 33% in intracerebral hemorrhage, 27% in subarachnoid hemorrhage, 15% in subdural hemorrhage and 25% in intraventricular hemorrhage. Low scores of GCS on admission were associated with a greater global and stroke-related mortality ($p = 0.000$). There is a choice of treatment, one is operative treated and the other is conservative treated. In the present study operative treated more than conservative treated (52% in intracerebral hemorrhage, 67% in subarachnoid hemorrhage, 85% in subdural hemorrhage and 50% in intraventricular hemorrhage. During the present hospitalization 39 patients died (29%). The highest incidence of death was found in intracerebral hemorrhage (35%) but the incident in other type was 23% in subarachnoid hemorrhage, 15% in subdural hemorrhage. None of intraventricular hemorrhage had died. the mean total hospital stay of stroke at the stroke unit in Al-Naseriah Hospital was 15.64 days, ranging from 1-120 days(43). The use of CT has greatly facilitated the diagnosis of hemorrhagic stroke and the differentiation of intracerebral hemorrhage, subarachnoid hemorrhage, subdural hemorrhage and intraventricular hemorrhage. Thus, CT is for the most part was responsible for the high levels of interrater agreement in these stroke subtypes (in the present study, $k = 0.861$ for type of hemorrhage and $k = 0.866$ for location of hemorrhage). In Wermer et al(44), studied the interobserver variation between three radiologists in classifying, fifty large hematoma on CT as deep or lobar. The kappa values were almost perfect, ranging from 0.88 to 0.96. The CT finding of non-traumatic intracranial hemorrhage appears to be distinctive. This has allowed definitive diagnosis and precise anatomical delineation of the lesions. Thus, CT is for the most part responsible for the high levels of inter-rater agreement in these stroke subtypes. In the present study the authors found concordance between CT brain interpretation by two neuroradiologist for the type of hemorrhagic stroke was very good, $k = 0.861$ and for location was $k = 0.866$. In Klaus et al(45) reported that Interobserver agreement is high ($k = 0.96$ for ICH and $k = 0.82$ for SAH) for major stroke types as well as for categories of hemorrhagic stroke on the basis of review of medical records and results of imaging data.

The finding of risk factors association to stroke in Al-nasriah hospital is similar to Risk factors, clinical presentations and predictors of stroke among adult patients admitted to stroke unit of Jimma university medical center, south west Ethiopia: prospective observational study , A total of 116 eligible stroke patients were recruited during the study period. The mean age of the patients was 55.1±14.0years and males comprised 62.9%. According to world health organization (WHO) criteria of stroke diagnosis, 51.7% of patients had ischemic while 48.3% had hemorrhagic stroke. The most common risk factor identified was hypertension (75.9%) followed by family history (33.6%), alcohol intake (22.4%), smoking (17.2%) and heart failure (17.2%). The most common clinical

presentation was headache complained by 75.0% of the patients followed by aphasia 60.3% and hemiparesis 53.4%. Atrial fibrillation was the independent predictor of hemorrhagic stroke.

The finding of hematoma formation at different sites is similar to Weisbergs' series. They found ganglionic-thalamic hemorrhage most frequently involved (77% from cases of Haemorrhagic stroke) followed by lobar hematoma (15%). Cerebellar, brainstem and pure intraventricular hemorrhage are less common. Multiple intracerebral hemorrhaging occurred in 5 cases in the present series. In the present study found the basal ganglia and thalamus are the most frequent sites of intracranial bleeding. 53 of 84 intracerebral hemorrhages (40%) affected this region. The most frequent cause of bleeding is vascular rupture, most often associated with hypertension. The relative proportions of hematoma in lobar (14.5%), brainstem (3.8%), cerebellum (3.8%) and pure intraventricular hemorrhage (2.3%) are similar to those previously reported. The intracerebral hemorrhage (ICH) and subarachnoid hemorrhage (SAH) often have poor outcomes. Indeed, the most common hemorrhagic strokes were ICH. ICH has the highest mortality and morbidity rates of any stroke subtype. The comparison of patients who died with those who survived revealed five variables which were significant. In the statistical analyses, the authors used clinical data on the patients, including hospital length of stay, initial level of consciousness (GCS score) and CT findings data, including hematoma size, intraventricular extension and mass effect. From the analyses, it was obvious that the variables hospital length of stay, lower level of consciousness, large hematoma size and presence of intraventricular hemorrhage extension were significant importance, while the presence of mass effect (midline shift and ventricle dilated (hydrocephalus) on CT scan had no prognostic influence on outcome (survival or death).

Conclusion :-

Computed tomography (CT) scan is the most widely used imaging procedure to evaluate stroke patients. CT is by no means the only neuroradiologic examination that has to be performed in patients with stroke, but it is safe, non-invasive creates a series of cross-sectional images of the brain and can show evidence of bleeding into the brain almost immediately after stroke symptoms appear. CT provides a substantial amount of information, including the size and location of the hemorrhage and the presence of intraventricular, subarachnoid, or subdural blood. The present study demonstrates that from all type of hemorrhagic stroke, intracerebral hemorrhage there remains a common and devastating clinical problem. A most risk factor were hypertension, but AVM, blood dyscrasia and unknown etiologies were the cause of non-hypertensive intracerebral hemorrhage, while ruptured aneurysm resulted in bleeding subarachnoid hemorrhage. CT findings by themselves can help predict which patients are at high risk of short-term mortality and may require intensive treatment. Three variables on CT imaging were identified as early mortality predictors in patients where hematoma volume was more than 60 cm3 ($p = 0.013$), the presence of intraventricular hemorrhage extension ($p = 0.003$) and mass effect were defined as the presence of ventricular shift across the midline

and/or enlargement of contralateral ventricle. In the present study, there was no mass effect association with clinical outcome ($p = 0.067$). Although the majority of hemorrhagic strokes are visible on the computed tomography scan, it necessitates finding a competent reader to appreciate the finding. The present study is not epidemiological in nature and results cannot be used for incidence calculations. Some limitations in the present study included retrospectives method and uncompleted medical records. This observational survey from computed tomography diagnosed and localized hemorrhaging in hemorrhagic stroke patients. A prospective study with a larger sample size is suggested.

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