



Sudan University of Science & Technology

College of Graduate Studies



**Role of Computed Tomography (CT) in Diagnosis Causes of
Headache in Emergency Department**

دور الأشعة المقطعية في تشخيص اسباب الصداع في اقسام الطوارئ

*Thesis Submitted For Partial Fulfillment For Requirement of M. Sc.
Degree in Diagnostic Radiologic Technology.*

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Dedication

To my parents

To my haspan and my children

My brothers

My sisters

TO everyone whom

Gave me a bit of

Wise and advice.

ACKNOWLEDGMENT

I am heartily thankful to my supervisor, D r. Ikhals Who Encouragement, guidance and support from the beginning to final level enabled me to an understanding of subject.

I would like to thank all people who have helped me and contributed to this Research.

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Abbreviations

A Com	Anterior communicating
APTT	Activated partial Thromboplastin Time
AVM	Arterio venous malformation
BP	Blood pressure
CCF	Congestive cardiac failure
CSF	Cerebrospinal fluid
CT	Computer tomography
CTV	CT Venogram
CVA	Cerebrovascular accident
ECA	External carotid artery
ECG	Electrocardiogram
EDH	Extradural hemorrhage
ETA	Estimated time of arrival
ETT	Endotracheal tube
GCS	Glasgow Coma Scale
HR	Heart rate
HU	Hounsfield Unit
I .m.	intramuscular
I. v .	Intravenous
ICA	Internal carotid artery
LP	Lumbar puncture
M:F	Male: female
MCA	Middle cerebral artery
NICE	National Institute of Clinical Excellence
P Com	Posterior communicating
RIND	Reversible Ischaemic neurological deficit
RR	Respiratory rate

SAH	Subarachnoid Hemorrhage
SDH	Subdural Hematoma
SLE	Systemic lupus Erythematosus
SSS	Superior sagittal sinus
TIA	Transient Ischaemic attack
WCC	White cell count

Abstract

The Study aim to determine the role of CT in diagnosis under line causes of headache in emergency departments in Khartoum Stats .The study takes 100 patients from emergency department complines of headache (traumatic non traumatic) emergency conditions. The sample comprised of 100 individuals with majority of them males 76 and24 females, have different ages. They did CT scan in Khartoum Advanced Center and Al Rebat University Hospital Department .The major of the requests for emergency scan Based on the clinical diagnosis the distribution was such intracranial pathologies predominated, head injuries, VA, and ICSOL.

The main finding of this study was CT diagnosis was the same as that clinically suspected in major cases and it was Best modality in diagnosis for headache causes and it available every time.

The study concluded that CT is most valued tools in the diagnosis of traumatic and non-traumatic patients have headache condition in emergency departments.

مستخلص البحث

اجريت دراستي بغرض معرفة دور الاشعة المقطعية في تشخيص اسباب الصداع في اقسام الطوارئ بولاية الخرطوم

قامت الدراسة باخذ 100 مريض في اقسام الطوارئ يعانون من صداع ناتج من حوادث او غيرها . كانت العينة معظمهم من الذكور 76 و24 من الاناث من مختلف الاعمار. خضعوا لفحص الاشعة المقطعية في اقسام الطوارئ بمستشفى الرباط الجامعي ومركز الخرطوم المتطور.

طلب التصوير للاشعة المقطعية يعتمد علي التشخيص السريري ويتوقع ان يكون امراض داخل الراس السائدة او اصابات الراس او اصابات الاوعية الدموية او اورام داخل الراس.

الدراسة وجدت ان تشخيص الاشعة المقطعية مطابق للتشخيص السريري في معظم الحالات كما وجدت ان هي الافضل في تشخيص اسباب الصداع في اقسام الطوارئ وهي متوفرة في اي وقت.

توصلت الدراسة ان الاشعة المقطعية هي اكثر الادوات قيمة لتشخيص حالات المرضى الذين يعانون من صداع ناتج من اصابة او غيرها في اقسام الطوارئ.

Chapter One

Introduction

Chapter One

1.1 Introduction:

Computer tomography (CT) is now widely available and is being used more and more. CT is often the initial imaging modality of choice not only for diagnosis but also to guide treatment. The most common request for CT out hours is brain imaging. CT is a vital tool in the assessment of patients with serious head injury; it remains the investigation of choice for the assessment of acute hemorrhage and bone injury. Consequently, patient management has been transformed since its inception, as rapid imaging and diagnosis of intra cranial pathology can facilitate emergency intervention. Computed tomography (CT) originally known as tomography (CT) and body section Roentgen nongraphic is a medical imaging method employing tomography where digital geometry processing used to generate a three dimensional image of the internals of object from a large series of two dimensional X-ray images taken around a single axis of rotation. The word (tomography) is derived from the Greek tomes (slice) and graphic (describing). CT produces a series of axial images which can be manipulated through a process known as windowing in order to recreate the image in different plane (Interpretation of Emergency Head CT Erskine J.et.al 2008).

1.2 Objectives:

1.2.1 General Objective

To determine the role of computed tomography (CT) in Diagnosis headache in emergencies departments

1.2.2 Specific Objective

-Evaluation the prevalence of headache disorders for traumatic and non-traumatic cases in emergency departments.

-Assessment the accuracy of CT in diagnosing headache in emergency departments.

1.3 Problem of Study:

Due to increase headache condition between the population, increase number of trauma daily due to increase cares in country, and more people's death without care and best investigation or diagnosis.

1.4 Importance of the study:

- The research tried to give some recommendation about the suitable modality for headache patients in emergency departments.
- To mention the most suitable stable protocol by Ct in emergence department.

1.5 Overview of the study

My study consists of five chapters: chapter one which is: an Introduction, deal with theoretical frame of the study, it presents the statement of study objectives, problems, importance of the study, methodology). Chapter two is Divide into Two sections, section one deal with Theoretical back ground: Anatomy of the head, pathology of the head, Fundamental of CT imaging) and section two which deal with previous studies. Chapter three discusses the Material and Methods: The material (Subject, Machine used). The Methods (CT protocol used, Images interpretation, Data collection, Analysis of data).chapter Four includes: Results presentation. Finally chapter five will Includes: Discussion, conclusion, Recommendations, references and appendix.

Chapter Two

**Literature review and previous
studies**

Chapter Two

Literature review and previous studies

2.1 Theoretical background:

2.1.1 Anatomy of the head

The brain is supported by the skull base and enclosed within the skull Vault. Within, the cranial cavity is divided into the anterior, middle And posterior fossae. The anterior and middle cranial fossae contain The two cerebral hemispheres. The posterior fossa contains the brainstem, consisting of the midbrain, pons and, most inferiorly, the medulla, and the cerebellum. Twelve paired cranial nerves arise from the brainstem, exit the skull base through a number of foramina, and innervate a variety of structures in the head proper. The largest of these foramina is the foramen magnum, through which the brainstem And spinal cord are in continuity. The brain is invested by the Meninges and bathed in cerebrospinal fluid (CSF), circulating in the subarachnoid space. Part of the meninges, the Dura, forms an incomplete partition between the cerebral hemispheres, known as the flax and roofs the posterior fossa as the tentorium cerebellum. There is A gap in the tentorium, called the hiatus, through which the midbrain joins the hemispheres. Within the brain are a number of cavities, the lateral, third and fourth cerebral ventricles, which contain CSF produced by the choroid plexuses within the ventricles. CSF flows from the ventricles into the subarachnoid spaces over the cerebral surface and around the spinal cord. Blood reaches the brain by the carotid and vertebral arteries and is drained by cerebral veins into a series of sinuses within the Dura into the internal jugular veins (*Applied Radiological Anatomy*, Adam et,al 2007).

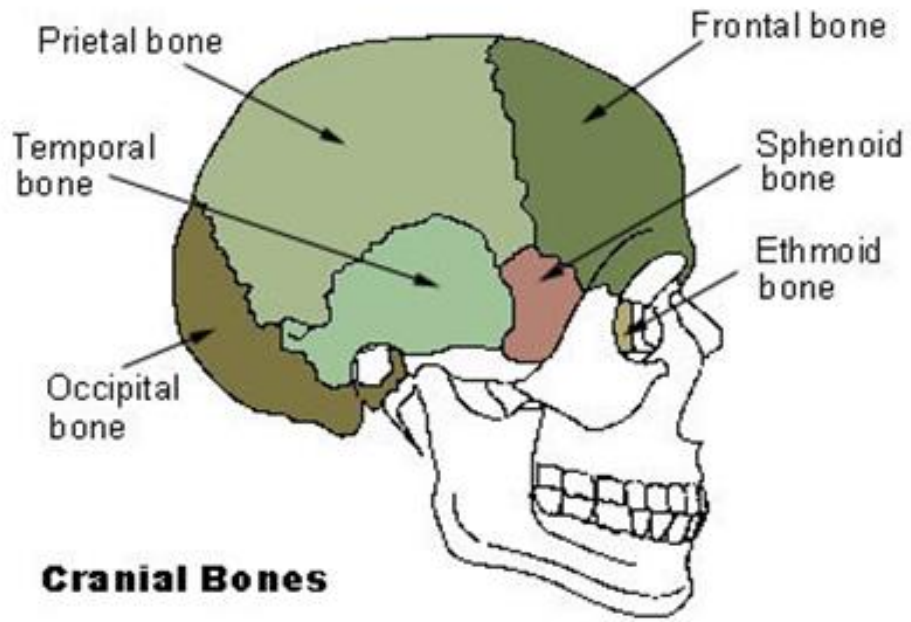
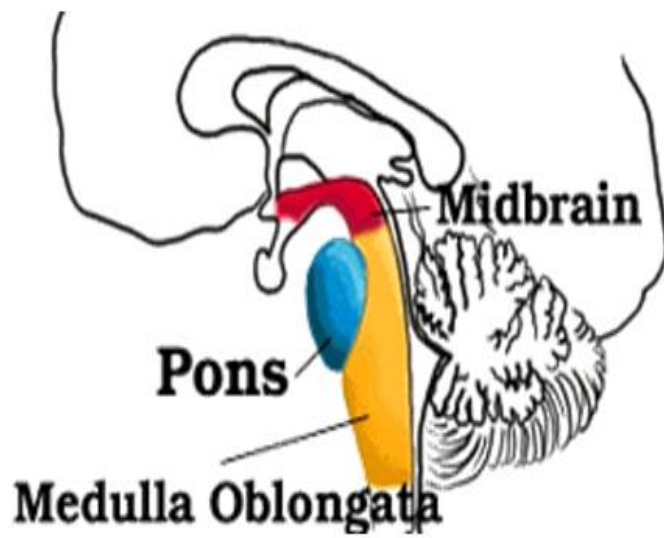


Fig. (2.1): Shows Cranial Bones of the Skull (<http://www.ctisus.org/>)



The Brain stem

Fig.(2.2)Show The brain Stem. (<http://www.halls.md/ct/ct.htm>)

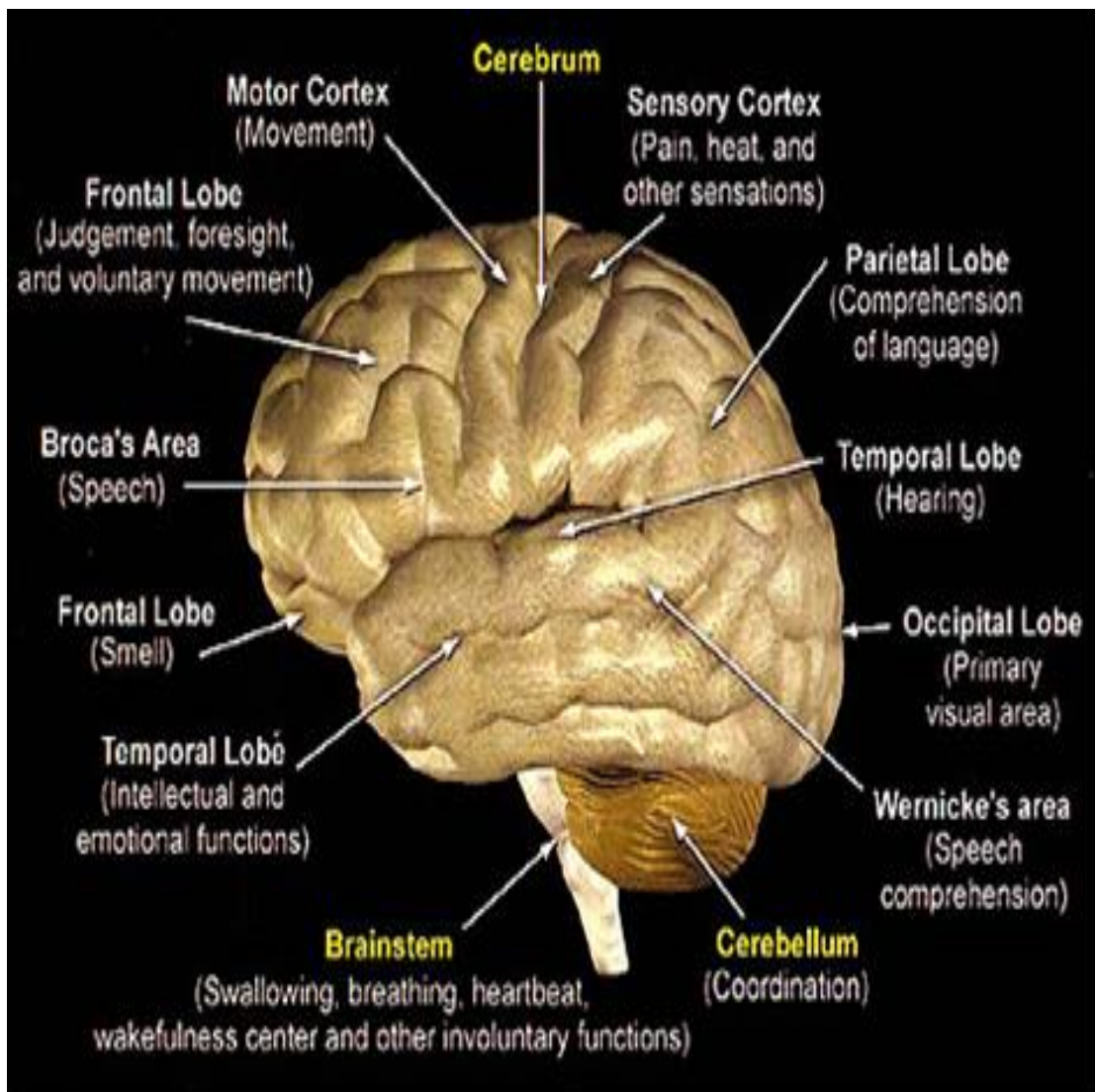


Fig.(2.3)Show Lobes and sensory areas Of Brain.

(<http://www.slaney.org/pct/>)

2.1.2 pathology of the head

2.1.2.1 Acute stroke

2.1.2.1.1 Ischemic stroke

2.1.2.1.1.1 Characteristics

Stroke is the third most common cause of death, and the leading cause of disability. 80% of strokes are ischemic large vessel occlusive athermanous disease (50%) Small vessel disease of penetrating arteries (25%) ¼ lacunar infarct Cardiogenic emboli (20%) Non-athermanous causes (5%) Ischemic infarction of the brain may be secondary to thrombosis or embolic disease. Transient ischemic attacks (TIAs) precede a quarter of ischemic strokes, and over 40% of these are in the 7 days before the stroke. The risk is highest in those patients with carotid stenosis or atrial fibrillation. The incidence of stroke increases with age, although one in four people who experience a stroke are Under 65 years Risk factors include hypertension, smoking, diabetes, hyperlipidemia, atherosclerosis, atrial fibrillation, the oral contraceptive pill and obesity. Temporal classification . _ TIA = transient ischemic attack. The clinical syndrome lasts less than 24 hours, although in a proportion there may be infarction on cerebral imaging. Progressing stroke = stepwise or gradually progressing accumulative neurological deficit evolving over hours or days.

_ Completed stroke = persistent stable neurological deficit – cerebral infarction as end stage of prolonged ischemia. Contraceptive pill and obesity. (Interpretation of Emergency Head CT Erskine J. et. al 2008)

2.1.2.1.1.2 Clinical features

Spectrum of presentation from mild symptoms and signs, in a well patient, to a moribund comatose patient. Commonly presents with unilateral weakness And /or sensory loss, visual field defect, dysphasia, and inattention/neglect.

Lacunar infarcts typically present with a purely motor and/or sensory deficit. Features of cortical involvement (visual field defect, dysphasia or inattention/neglect) are absent.

Posterior circulation infarcts commonly present with vertigo, ataxia, diplopia, dysarthria, dysphasia or bilateral limb signs. The neurological deficit can be sudden, often occurring during sleep this makes the time of onset difficult to ascertain. .(Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.2.1.1.3 Radiological features

CT features

Hyper acute infarct (< 12 hours): Non-contrast CT may appear normal in up to 60%. However, contrary to general opinion, the CT may be abnormal in up to 75% of patients with MCA infarction, imaged within the first 3 hours (Interpretation of Emergency Head CT Erskine J. et.al2008). ‘Hyper dense MCA’ sign represents acute Intraluminal thrombus, and is seen in 25–50% of acute MCA occlusions. It is recognized as focal or linear white density within the MCA in the Sylvain fissure. Although not sensitive, it is a relatively specific sign. .(Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.2.1.2 Hemorrhagic stroke

2.1.2.1.2 .1 Characteristics

Hemorrhagic strokes account for only 10–15% of CVAs. is associated with a high mortality rate, Small intra cerebral arteries, often damaged by chronic hypertension, rupture and blood leaks directly into the parenchyma.

Hematoma, with resulting edema, leads to mass effect and further compromise to blood supply. .(Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.2.1.2.2 Risk factors:

Hyper tension, underlying brain pathology, bleeding diatheses, anticoagulation treatment, thrombolysis therapy and cocaine abuse.

Clinical features

Hemorrhagic and ischemic strokes are difficult to distinguish clinically.

Patients with hemorrhagic strokes are generally sicker, with abrupt onset and rapid deterioration. Common symptoms are headache, decreased conscious level, seizures, nausea and vomiting. Hypertension is characteristic. (Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.2.1.2.3 Radiological features

Non-contrast head CT is the investigation of choice. Acute hemorrhage is hyper dense. Surrounding edema will result in loss of the grey/white matter differentiation. Mass effect will result in compression of overlying sulci, ventricular compression, midline shift and reduction in the size of the basal cisterns (Interpretation of Emergency Head CT Erskine J.et.al 2008).

2.1.2.2 Subdural hematoma (SDH)

2.1.2.2.1 Characteristics

Subdural hemorrhage arises between the inner layer of Dura and arachnoid membrane of the brain. Bleeding results from torn bridging veins that cross the potential space between the cerebral cortex and Dural venous sinuses.

May be acute, sub-acute or chronic and are bilateral. Acute SDH carries a high mortality and morbidity. Direct pressure results in ischemia on the adjacent brain. Re bleeding secondary to osmotic expansion, or further trauma, leads to acute on chronic hemorrhage.

The etiology of chronic SDH is often unclear. Subdural hematomas are more common in elderly and alcoholic patients, where the subdural spaces are larger due to age related involution. Subdural hemorrhage in the newborn is usually due to obstetric trauma. In neonates, non-accidental injury needs to be considered. (Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.2.2.2 Clinical features

2.1.2.2.2.1 Acute SDH

Patients often present following severe head trauma.

Associated with underlying brain injury with a worse long-term prognosis than extradural hematoma. Patients generally have a decreased level of consciousness with focal neurological defects or seizures. Patients with a primary or secondary coagulopathy (e.g. alcoholics) may develop an acute SDH after only minor head trauma. A small acute SDH may be asymptomatic. (Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.2.2.2 Chronic SDH

Chronic SDH is the result of: Resolving phase of medically managed acute subdural hematoma. And repeated episodes of subclinical hemorrhage until becoming symptomatic. Chronic SDH often presents in the elderly with vague Symptoms of, gradual depression, personality changes, fluctuations of consciousness unexplained headaches or evolving hemiplegia

Predisposing factors: alcoholism, increased age, epilepsy, coagulopathy And prior placement of ventricular shunt. (Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.2.2.3 Radiological features

Location: Blood is seen over the cerebral convexity, often extending into the Inter hemispheric fissure, along the Tentoria margins, and beneath the temporal and occipital lobes do not cross the midline.

2.1.2.2.4 CT features

2.1.2.2.4.1 Acute SDH

Peripheral high density crescent fluid collection between the skull and cerebral hemisphere usually with: A concave inner margin. A small hematoma may only minimally press into brain substance. Convex outer margin following normal contour of cranial vault. Occasionally with a blood–fluid level Signs of mass effect with compression. Of overlying sulci, ventricular Compression , midline shift and reduction in the size of the basal cisterns.

2.1.2.2.4.2 Sub acute SDH

After approximately 1 to 2 weeks the subdural collection becomes isodense to grey matter; therefore detection may be challenging and only be recognized due to persistent mass effect: Effacement of cortical sulci, Deviation of lateral ventricle, Midline shift, white matter buckling and Displacement of grey white matter interfaces. Contrast enhancement will often define cortical–subdural interface. .(Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.2.2.4.3 Chronic SDH

After approximately 2 weeks, chronic SDH's are often hypodense crescent collections, with or without mass effect. Acute-on-chronic SDHs can further complicate the images, with hyperdense fresh hemorrhage intermixed, or layering posteriorly, within the chronic collection. Complex septated collections, and in rare cases calcification, may develop. .(Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.2.3 Extradural hematoma

2.1.2.3.1 Characteristics

An extradural hemorrhage arises within the potential space between the skull and Dura. The young are more frequently affected as the Dura is more easily stripped away from the skull. The Dura becomes more adherent with age.

Associated with a skull fracture in most cases. Most commonly bleeding is from a lacerated (middle) meningeal artery/vein, adjacent to the inner table, from a fracture of the adjacent calvarium. Early diagnosis is imperative, as prognosis is good with early intervention. Conversely, a delay may result in cerebral herniation and brainstem compression.

The types : Acute extradural hematoma from arterial bleeding, Sub acute hematoma and Chronic hematoma from venous bleeding. .(Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.2.3.2 Clinical features

Patients often present with a history of head trauma. Associated with a variable level of consciousness. Neurological examination may reveal Lateralizing signs with a unilateral up-going plantar response.

A sensitive sign in the conscious patient is pronator drift of the upper limb, when asked to hold both arms outstretched with the palms upwards. (Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.2.3.3 Radiological features

Location: Temporoparietal (most often from laceration of middle meningeal artery). Frontal pole, parietal-occipital region, between occipital lobes and posterior fossa (most often from laceration of the Dural sinuses from A fracture).

2.1.2.3.4 CT features

Biconvex hyper dense elliptical collection with a sharply defined edge.

Mixed density suggests active bleeding. Hematoma does not cross suture lines unless a diastolic suture fracture is present. May separate the venous sinuses and flax from the skull; this is the only type of intracranial hemorrhage to do this.

Mass effect depends on the size of the hemorrhage and associated edema.

Venous bleeding is more variable in shape. Associated fracture line may be seen. (Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.2.4 Subarachnoid hemorrhage

2.1.2.4.1 Characteristics

Subarachnoid hemorrhage accounts of CVAs. The Causes of it: Spontaneous, ruptured aneurysm, AVM, hypertensive hemorrhage.

And Trauma. Blood enters the subarachnoid space onto the surface of the brain, between the Pie and arachnoid, and may lead to raised intracranial pressure by obstructing the ventricular outflow of CSF. Incidence increases with age and peaks at age 50 years. Morbidity can be severe and is increased by re bleeding, which often occurs in the first few days, and cerebral vasospasm 7 to14 days

after the initial event. Berry aneurysms are associated with hypertension, polycystic kidney disease, SLE, connective tissue disorders, use In non-traumatic SAH's, no lesion is found at post-mortem. (Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.2.4.2 Clinical features

SAH classically presents with a sudden onset of a severe 'thunderclap' occipital headache, often described as the 'worst headache in their life'.

Associated with physical or emotional stress, coitus or head trauma.

A leading cause of maternal mortality, accounting for maternal deaths during pregnancy. Meningeal irritation generates symptoms of neck Stiffness, photophobia and low back pain, with a positive Kerning's sign.

Focal neurological signs include third nerve palsy from compression by An Expanding berry aneurysm of the posterior communicating artery of the Circle of Willis. Consider SAH in the comatose or fitting patient. (Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.2.4.3 Radiological features

2.1.2.4.3.1 Location of aneurysm rupture

The most common sites of rupture are as follows:

The internal carotid artery, including the posterior communicating(P Com) junction ,The anterior communicating (A Com) , artery/anterior cerebral Artery, The middle cerebral artery (MCA) , The vertebrae solar and other arteries

2.1.2.4.4 CT features

CT scan without contrast. CT scan findings are positive in patients who Have SAH. Sensitivity decreases with time from onset of ictus.

May be falsely negative in patients with small hemorrhages and in those with severe anemia. The location of blood within the subarachnoid space correlates directly with the location of the aneurysm rupture .Blood localized to the basal cisterns, the Sylvain or intra hemispheric fissures suggests rupture of a secular aneurysm.

Blood found lying over the cerebral convexities or within the superficial brain parenchyma suggests rupture of an AVM or mycotic aneurysm.

Anterior communicating artery aneurysms are often associated with inter hemispheric and intraventricular hemorrhages. Middle cerebral artery and posterior communicating artery aneurysms are associated with intraparenchymal hemorrhages. Serial CT allows for surveillance of evolving mass effect and hydrocephalus; in the first 2 weeks post-ictus. (Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.2.5 Cerebral venous sinus thrombosis

2.1.2.5.1 Characteristics

Rare cause of stroke, affecting both sexes equally.

Risk factors

Septic causes esp. in childhood: (Mastoiditis, facial cellulitis, meningitis, encephalitis, brain abscess, intracranial empyema). Aseptic causes: Hypercoagulable states: (polycythemia rubra Vera, idiopathic thrombocytosis, thrombocytopenia, pregnancy oral contraceptive pill). Low-flow state: (CCF, shock). (Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.2.5.2 Clinical features

Classically presents with sudden, severe headache, worsened by coughing and associated with vomiting. Focal neurological deficit may be seen if venous infarction occurs. Seizures may occur. Sigmoid sinus thrombosis causes cerebellar signs and lower cranial nerve palsies. Periorbital edema and chemosis are seen with cavernous sinus thrombosis.

2.1.2.5.3 Radiological features

2.1.2.5.3.1 CT features

CT may be normal.

Non-contrast CT: Hyperdense material within a vessel representing thrombosis/blood. Not reliable as also seen with slow flowing blood.

Contrast CT: Look for the 'delta'/'empty triangle' sign. This is a filling defect within the straight/superior sagittal sinus and represents flow around a central

non-enhanced clot. Goral enhancement peripheral to an infarct. Co-existing signs of infection or inflammation (e.g. sinusitis/ mastoiditis) should raise suspicion. .(Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.2.6 Contusions

2.1.2.6.1 Characteristics

Traumatic injury to cortical surface of brain. Commonest form of traumatic cerebral injury: head injuries. Children: adults =2:1. Usually the result of linear acceleration/ deceleration forces or penetrating injuries.

Often described as ‘coup’/‘contra-coup’ injuries: Coup injury – site of the direct impact on the stationary brain. Contra-coup – site of impact of the moving brain upon the stationary. Inner table, opposite to the site of the coup injury. Cerebral contusions are also produced secondary to depressed skull fractures and are associated with injuries other intracranial. .(Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.2.6.2 Clinical features

Patients often present with a history of head trauma or external signs of injury. Usually associated with a brief loss of consciousness. And altered GCS may be prolonged. Headache with vomiting in the conscious patient. Focal neurological deficit may occur if contusions arise near the sensorimotor cortex. Most patients make an uneventful recovery, but a few develop raised intracranial pressure, post-traumatic seizures and persisting focal neurological deficits. The elderly, alcoholics and those taking anticoagulants that are at increased risk of hemorrhage. .(Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.2.6.3 Radiological features

CT features

Location; Often multiple bilateral lesions at the interface between grey and white matter . Commonly along anterior, lateral and inferior surfaces of frontal and temporal lobes. Less frequently seen in parietal and occipital lobes and

the posterior fossa. CT sensitive for hemorrhage in the acute post-traumatic period. The site of scalp swelling often indicates the site of the coup injury.

Focal/multiple areas of low attenuation, representing edema, are intermixed with tiny areas of increased density, representing petechial hemorrhage.

In children, a common appearance is of diffuse cerebral swelling without hemorrhage in the acute post-traumatic period. True extent becomes apparent over time with progression of cell necrosis, edema and mass effect.

.(Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.2.7 Skull fractures

2.1.2.7.1 Characteristics

Result from trauma to the head. Classified as linear, depressed or base of skull fractures. Linear fractures are often uncomplicated and do not require treatment. However temporal bone fractures may result in an extradural hematoma.

Depressed skull fractures may require surgery to elevate the bone fragments to prevent brain injury. Increased significance if the fracture is open, or Communicates with an adjacent sinus, due to increased risk of infection. In basal skull fractures prophylactic antibiotics were once routinely prescribed to reduce the risk of meningitis, but their effectiveness is not validated and use is now restricted. .(Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.2.7.2 Clinical features

Open fractures underlie scalp lacerations and are often diagnosed during evaluation of the wound for closure. Depressed skull fractures are often palpable or visible during examination but may be masked by swelling around the area. Clinical signs of base of skull fracture:

CSF rhinorrhea, Haemotympanum, Bleeding from the external auditory meatus, ‘Raccoon’ eyes, Sub conjunctiva hemorrhage (with no posterior limit), Battle’s sign (bruising over the mastoid area), Cranial nerve deficits. .(Interpretation of Emergency Head CT Erskine J.et.al 2008).

2.1.2.7.3 Radiological features

CT features

Look closely at the initial scout image as this may demonstrate a fracture.

Soft tissue swelling, or an underlying brain abnormality, may be associated

With a fracture. Fractures may be missed if appropriate ‘window’ parameters are not chosen. Always assess for fractures on bony windows. Fractures appear as sharply defined lines and should not be mistaken for a suture or vascular groove; a vascular groove often branches and both have typical sites.

The presence of intracranial air may be secondary to an open fracture or connection with an air-containing sinus. (Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.2.8 Meningitis

2.1.2.8.1 Characteristics

Defined as inflammation of the meninges. Anatomically divided into:

Inflammation of the Dura, sometimes referred to as pachymeningitis(less common). Inflammation of the arachnoid membrane and subarachnoid space, referred to as leptomeningitis (more common). Meningoencephalitis also involves the parenchyma. Overcrowded closed communities (e.g. schools, day centres) predispose patients to meningitis, especially if immunocompromised. Concurrent illnesses such as pneumonia or other sites of sepsis (e.g. sinusitis, mastoiditis, otitis media) may contribute. (Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.2.8.2 Clinical features

Fever, neck stiffness, photophobia, unremitting headache, mental status changes, with CSF findings, are essentials for diagnosis.

Kerning’s sign – pain and resistance on passive knee extension with hips fully flexed. Brzezinski’s sign – hips flex on bending head forward.

Seizures and cranial nerve palsies are common. Patients may present with signs of raised intracranial pressure.

Children may present with poor feeding, irritability, lethargy and vomiting. The elderly may only have a low grade fever and delirium. CSF sampling reveals raised WCC, predominantly neutrophils, with low glucose and high protein, in cases of bacterial meningitis (Interpretation of Emergency Head CT Erskine J. Holmes, Anna C. Forest-Hay and Rakish R. Misran 2008).

2.1.2.8.3 Radiological features

CT features

Non-contrast CT is often normal. Contrast enhanced CT

Enhancement of the meningeal surfaces is a non-specific and often an inconsistent finding in patients with meningitis.

When present, enhancement is usually seen over the cerebral convexities and in the inter hemispheric and Sylvian fissures. In tense contrast enhancement, with associated meningeal thickening, is suggestive of granulomatous meningitis (such as TB and sarcoidosis).

Associated obliteration of the basal cisterns, Sylvian fissures and supra seller cistern. The cerebral sulci may be effaced, with associated flattening of the Ventricles, due to cerebral edema.

The cerebral sulci may be effaced, with associated flattening of the ventricles, due to cerebral edema. Cerebral infarction is not uncommon. (Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.2.9 Raised intracranial pressure

2.1.2.9.1 Characteristics

The skull defines a fixed volume. Increasing the volume of contents, or brain swelling from any cause, rapidly increases intracranial pressure.

Causes of raised intracranial pressure include:

Hemorrhage (subdural, extradural, subarachnoid, intra cerebral, intra ventricular), Brain abscess, Eningo encephalitis, Primary or metastatic tumors,

Hydrocephalus, Cerebral edema (vasogenic , cytotoxic or interstitial).
(Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.2.9.2 Clinical features

Patients often present with a vague history of listlessness, irritability, drowsiness, early morning headaches, nausea and vomiting.

The presentation may be acute with sudden neurological deterioration.

Classic progression of symptoms:

Brady cardia, Rising blood pressure, Respiratory depression (Cushings response), Pupillary constriction and then dilation, Third nerve palsy – dilated ipsilateral pupil and ophthalmoplegia, Develop as intracranial pressure increases, Papilledema is an unreliable sign. Look for absence of venous pulsation. (Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.2.9.3 Radiological features

CT features

CSF spaces are reduced in size with effacement of sulci and the basal cisterns.

Herniation of brain parenchyma (representing shift of the normal brain, through or across regions, to another site due to mass effect) occurs late.

Types of cerebral herniation:

Subalpine herniation (most common form of herniation and occurs as the brain extends under the flax in the supra tentoria cerebrum).

Transt entorial herniation(occurs when the brain traverses across the tentorium at the level of the tentorial incisura). Can be divided into ascending and descending transtentorial herniation:

Descending transtentorial herniation is caused by mass effect in the cerebrum which pushes the supratentorial brain through the incisura into the posterior fossa. Ascending transtentorial herniation is caused by mass effect in the posterior fossa which pushes the infra tentorial brain through the incisura in an upward direction. Cerebellar tonsillar herniation (cerebellar tonsils are forced

through the foramen magnum). .(Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.2.10 Hydrocephalus

2.1.2.10.1 Characteristics

Hydrocephalus results from an excess of CSF, due to an imbalance between CSF production and absorption, resulting in increased intra ventricular pressure. Classified as communicating and non-communicating hydrocephalus:

2.1.2.10.1.1 Communicating hydrocephalus:

Elevated intra ventricular pressure secondary to obstruction of CSF flow beyond the outlet of 4th Ventricle. This may be due to impeded CSF flow over the cerebral convexities and/or impeded reabsorption of CSF by the arachnoid villi. Causes Subarachnoid hemorrhage, meningeal metastases and granulomatous meningitis. A less common cause of communicating hydrocephalus results from rapid CSF production, e.g. choroid plexus papilloma.

2.1.2.10.1.2 Non-communicating hydrocephalus:

Blockage of CSF flow within the ventricular system, with dilatation proximal to the obstruction.

Often referred to as obstructive hydrocephalus.

Location of obstruction / causes:

Lateral ventricles, e.g. ependymal, meningioma, Foramen of Monroe, e.g. third ventricular colloid cyst, Third ventricle, e.g. large pituitary adenoma, craniopharyngioma, Aqueduct of Sylvius, e.g. congenital aqueduct stenosis, post intra ventricular hemorrhage, Fourth ventricle/foramina of Lustra and Magnesia, e.g. congenital, Obstruction, intra ventricular tumor, extrinsic compression. .(Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.2.10.2 Clinical features

Neonates/infancy: Enlarged cranium, bulging fontanel, widely separated sleepiness and irritability. Older children and adults: headaches, nausea, vomiting, papilledema, diplopia, problems with balance and coordination,

Gait disturbance, urinary incontinence, and changes in cognition including memory loss. .(Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.2.10.3 Radiological features

2.1.2.10.3.1 Non-communicating hydrocephalus

Ventricular dilatation proximal to the level of an obstructing lesion.

Dilatation of the occipital horns precedes dilatation of the frontal horns.

Commensurate dilatation of the temporal horns with lateral ventricles.

Progressive enlargement of the ventricular system, disproportionate to narrowed and effaced cortical sulci. Periventricular low attenuation is seen with acute onset of hydrocephalus; this represents interstitial edema from Trans ependymal flow of CSF. The obstructing lesion may be evident.

2.1.2.10.3.2 Communicating hydrocephalus

Symmetrical enlargement of the lateral, third, and fourth ventricles.

Normal / effaced cerebral sulci. Dilatation of subarachnoid cisterns.

Periventricular low attenuation, secondary to trans ependymal CSF flow, may be seen with acute onset hydrocephalus.(Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.2.11 Abscesses

2.1.2.11.1 Characteristics

Localized purulent bacterial infection often developing in an area of celebrities.

2.1.2.11.2 Causes:

Generalised septicaemia:

Extension from adjacent sin nasal infection, mastoiditis, otitis media.

Respiratory causes: bronchiectasis, lung abscesses, empyema and pneumonia.

Cardiac causes: right to left shunt, AVM and endocarditis. Osteomyelitis.

Penetrating trauma or surgery. Predisposing factors Diabetes mellitus.

Steroids /immunosuppressive therapy. Immune deficiency. .(Interpretation of Emergency Head CT Erskine J.et.al 2008)

Causative organisms Anaerobic streptococcus (most common), Staphylococcus, Bactericides, Multiple organisms in 20%, Mycobacterium/salmonella more commonly in developing countries, Toxoplasmosis in AIDS patients. .(Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.2.11.3 Clinical features

Patients may present with headaches, vomiting, seizures, and altered mental state, in association with spiking pyrexia. Cranial nerve palsies or localized peripheral neurological deficits may be present. Signs of raised intracranial pressure. Source of sepsis may be clearly identifiable, or the patient may present with pyrexia of unknown origin Diagnosis and treatment is difficult in those who are immunosuppressed. Significant long-term morbidity. Complications include cavernous sinus thrombosis, venous infarction and coning. .(Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.2.11.4 Radiological features

CT features

Location

Supratentorial : infratentorial = 2:1 . Typically at the corticomedullary junction in the frontal and temporal lobes.

Non-contrast CT

Low density lesion with associated mass effect. Gas within lesion due to gas-forming organisms.

Contrast-enhanced CT

Ring enhancement, with central necrosis, and surrounding oedema.

Homogeneous enhancement in lesions <5 mm. Lesions may be multi-loculated and adjacent daughter abscesses may develop. .(Interpretation of Emergency Head CT Erskine J.et.al 2008).

2.1.2.12 Arteriovenous malformation

2.1.2.12.1 Characteristics

Congenital abnormality consisting of abnormally dilated tortuous arteries and veins, with closely packed abnormal pathological vessels which shunt blood between the two. Most common intra cerebral vascular lesion. 80% occur by the age of 40; 20% present under 20 years of age. May be part of a congenital syndrome, e.g. Sturge–Weber. Venous malformations are less common, e.g. medullary venous malformation, cavernous malformation. Arterial-venous fistulae are usually post-traumatic. (Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.2.12.2 Clinical features

Often a symptomatic. are diagnosed incidentally. May present with headaches, seizures, acute intracranial hemorrhage or progressive neurological deficit.

2.1.2.12.3 Radiological features

2.1.2.12.3.1 Location

Supra tentorial (90%): parietal>frontal>temporal>occipital lobe.

Infratentorial (10%).

Vascular supply Pial branches of ICA in of supratentorial lesions and of posterior fossa lesions. Dural branches of ECA in with infratentorial lesions.

2.1.2.12.3.2 CT features

Non-contrast CT Irregular lesion with large feeding arteries and draining veins. Mixed density lesion, composed of large dense vessels, hemorrhage and calcification. Is Odense lesion, which may only be recognizable by associated mass effect. Low-density lesion due to atrophy secondary to associated local cerebral ischemia. Are not visualized on unenhanced CT.

Contrast-enhanced CT Dense serpiginous enhancement in, representing tortuous dilated vessels. Lack of mass effect and edema unless thrombosis or secondary hemorrhage. No enhancement in thrombosis AVM. Adjacent brain

atrophy due to local cerebral ischemia .(Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.2.13 Solitary space-occupying lesions

Is usually tumors. 30% are secondary tumors from breast, lung or melanoma primary lesions. Metastases tend to Solitary lesions.

2.1.2.13.1 Characteristics

Is most commonly found in the supratentorial compartment with the exception of those from renal cell carcinoma that tend to be in the posterior fossa.

Primary tumors (e.g. astrocytoma, glioblastoma multiform, oligodendrogliomas, ependymomas) have a <50% 5-year survival.

Frontal lobe masses often present late. Other solitary lesions include cerebral abscess, aneurysm, tubercular, granuloma or cyst. .(Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.2.13.2 Clinical features

May present with signs of raised intracranial pressure. Seizures, with or without a localising aura. Focal neurology may evolve There may be false localizing signs. Solitary mass lesions can cause local effects, e.g. proptosis or epistaxis.

Patients may present with odd behavior, headache or vomiting.

Clinical presentation may help localize the site of the lesion:

Temporal lobe – complex partial seizures, hallucinations, taste, smell, dysphasia, field defects, fugue, functional psychosis and hyper sexuality.

Frontal lobe– hemiparesis, seizures, personality change, grasp Reflex(unilateral is significant), expressive

Dysphasia (Broca's area) and anosmia.

Parietal lobe – hemi sensory loss, decreased stereo gnosis, sensory inattention, dysphasia and Gerstmann's syndrome (finger agnosia, left/right disorientation, dysgraphia, calculi).

Occipital lobe – contralateral visual field defects.

Cerebellum – past pointing, intention tremor, nystagmus, dysdiadochokinesis and truncal ataxia (worse if eyes open).

Cerebello-pontine angle – nystagmus, reduced corneal reflex, fifth and seventh cranial nerve palsies, ipsilateral cerebellar signs and ipsilateral deafness.

Mid-brain – unequal pupils, confabulation, somnolence and an inability to direct the eyes up or down. (Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.2.13.3 Radiological features

Cerebral masses encompass a spectrum of appearances. Lesions may be hypodense, isodense or hyperdense. May be seen due to asymmetry or the presence of edema and mass effect. Calcification may be present. Appearance post-contrast is often helpful.

2.1.2.14 Multiple lesions

2.1.2.14.1 Characteristics

Neoplastic causes: Brain metastases are the most common neoplastic intracerebral lesion. They are found in patients that die from cancer, and represent brain tumors in adults.

Infective causes: For example, cerebral abscesses, granulomata.

Vascular causes: Multiple lesions of varying age are seen in multi-infarct dementia.

Inflammatory causes: Demyelinating plaques can be seen as multiple low density lesions on CT, predominantly in the periventricular deep white matter.

Traumatic causes: Contusions are frequently multiple after head trauma. (Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.2.14.2 Clinical features

Depends on the underlying pathology. See solitary lesions.

2.1.2.14.3 Radiological features

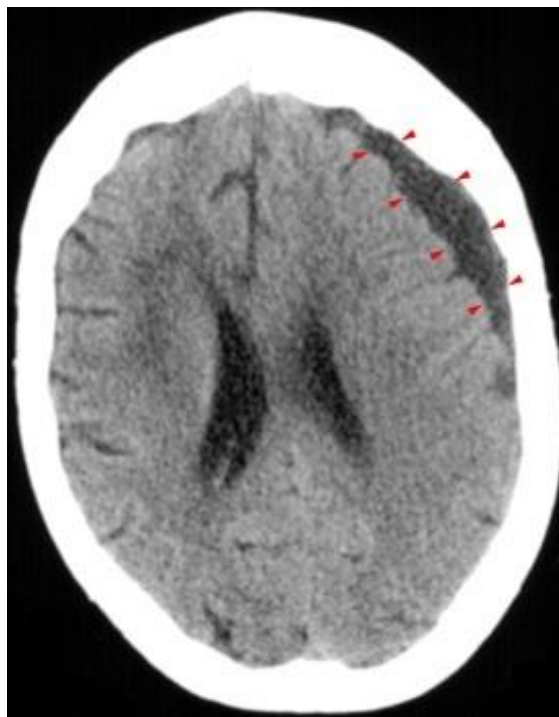
Contrast is taken up in tumors, inflammatory granulation tissue or areas of damage to the blood–brain barrier. Melanoma and adenocarcinoma metastases may appear hyper dense prior to contrast. Calcification in malignant tumors is uncommon but, if present, suggests granulomatous infection in not uncommon. Hemorrhage into metastases occurs infrequently, and when present suggests hyper vascular tumors such as melanoma or hyper nephroma. A follow-up CT performed two weeks after a traumatic event makes multiple contusions more conspicuous(Interpretation of Emergency Head CT Erskine J.et.al 2008).



Fig.(2.4) Acute Intra cerebral hematoma (Interpretation of Emergency Head CT Erskine J. et.al 2008).



Fig.(2.5) Subdural hematoma (Interpretation of Emergency Head CT Erskine J. et.al 2008).



Fig(2.6) Chronic Subdural Hematoma (Interpretation of Emergency Head CT Erskine J. et.al 2008)

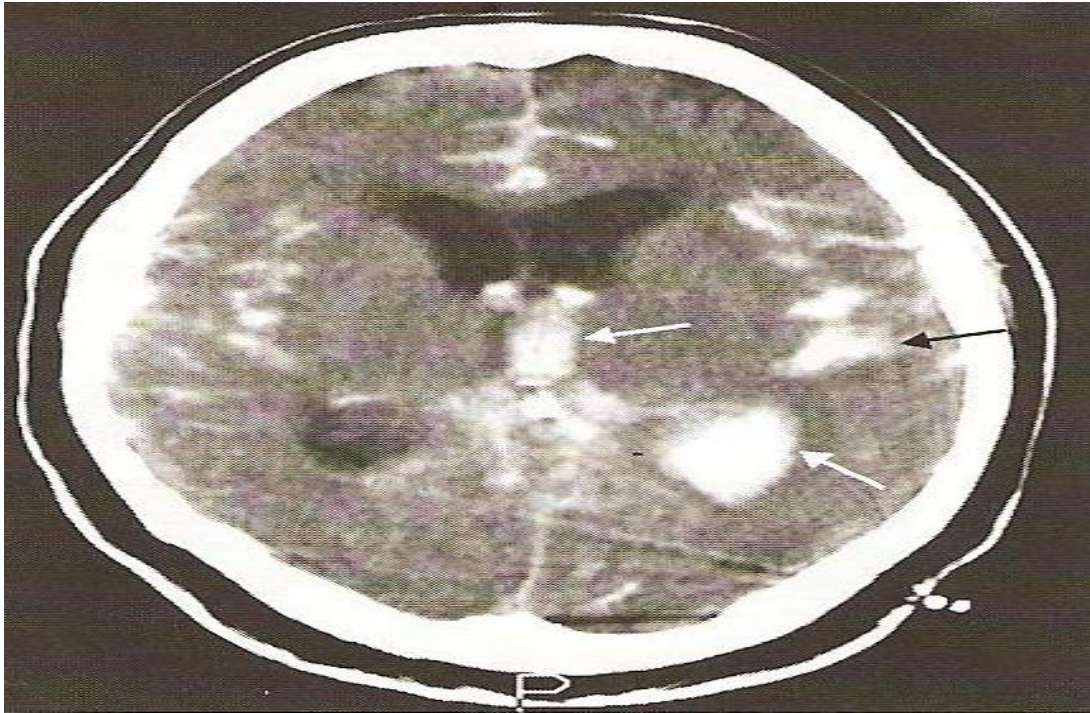


Fig.(2.7) Subarachnoid Hemorrhage(Interpretation of Emergency Head CT Erskine J. et.al 2008).



Fig.(2.8) Epidural Hemorrhage(Interpretation of Emergency Head CT Erskine J. et.al2008).



Fig.(2.9) Hydrocephalus (Interpretation of Emergency Head CT Erskine J. et.al 2008).



Fig.(2.10) Brain infarct (Interpretation of Emergency Head CT Erskine J. et.al 2008).

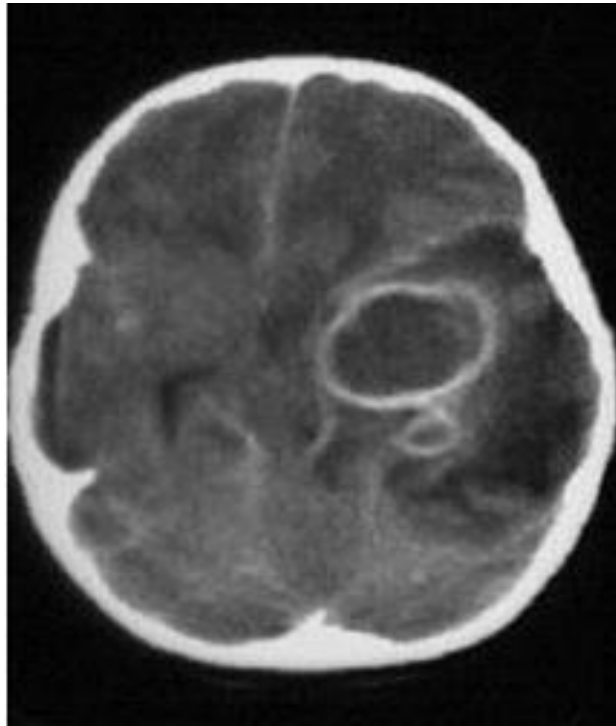


Fig.(2.11) Brain cyst with contrast (Interpretation of Emergency Head CT Erskine J.et.al 2008).

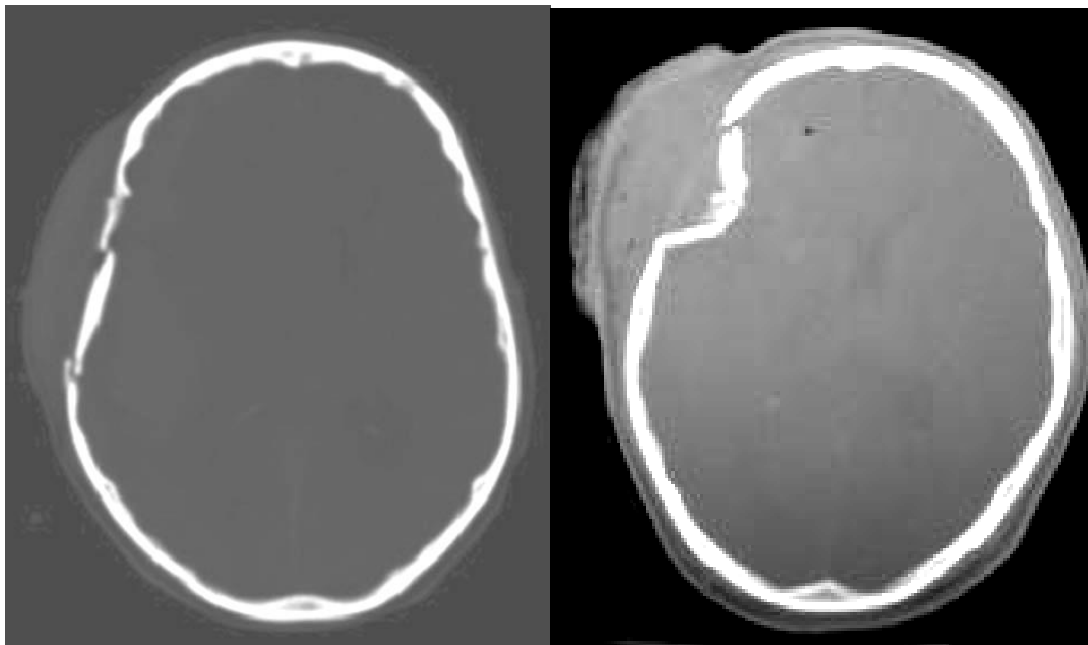


Fig.(2.12) Bone window showing depressed skull fracture (Interpretation of Emergency Head CT Erskine J.et.al 2008).



Fig.(2.13) Venous hemorrhage in the left front parietal cortex due to sagittal sinus thrombosis. (Interpretation of Emergency Head CT Erskine J. et.al 2008).

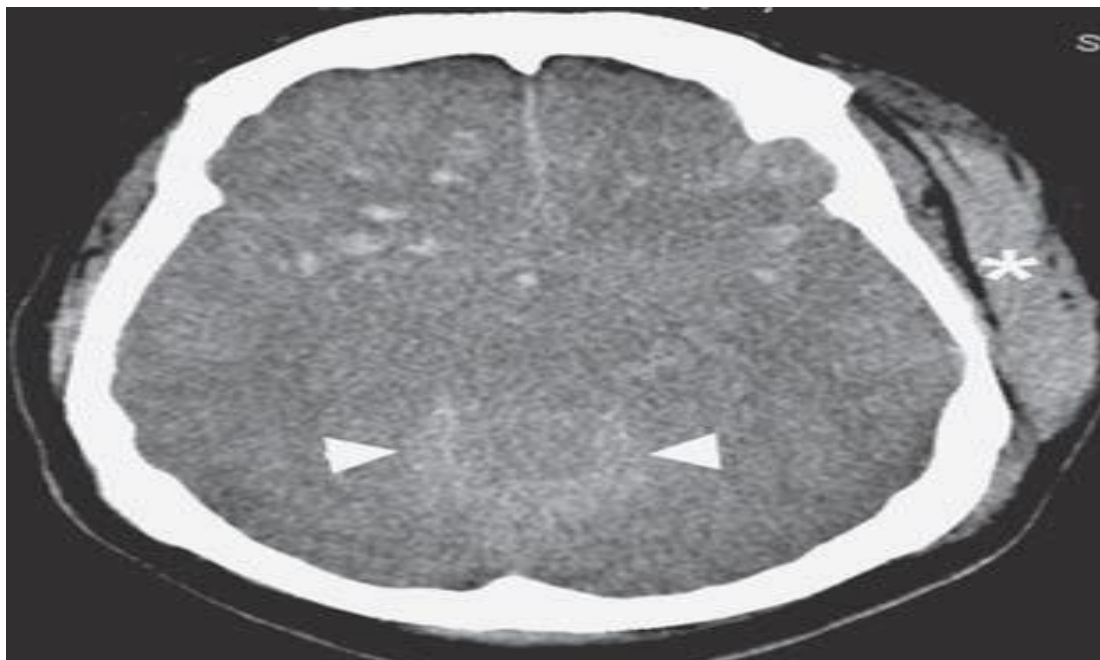


Fig.(2.14) Multi-focal contusions within both frontal lobes, with additional acute Subarachnoid hemorrhage on the tentorium (arrowheads). Marked left fronto-parietal soft tissue swelling(asterisk). (Interpretation of Emergency Head CT Erskine J. et.al 2008).

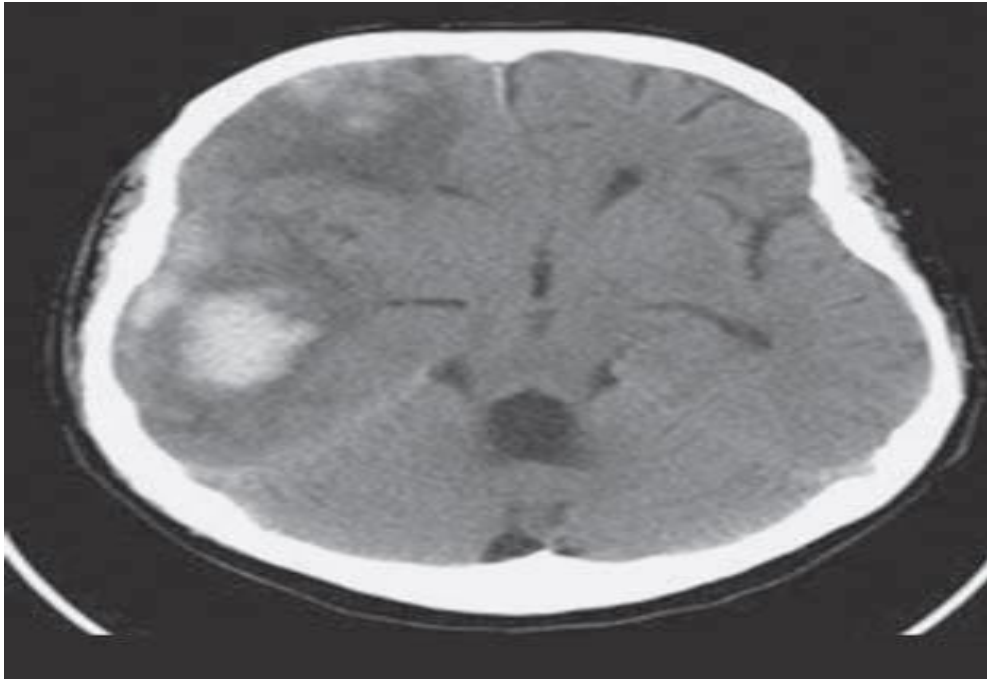


Fig.(2.15) Large contusions in the right frontal and temporal lobes
(Interpretation of Emergency Head CT Erskine J. et.al 2008).

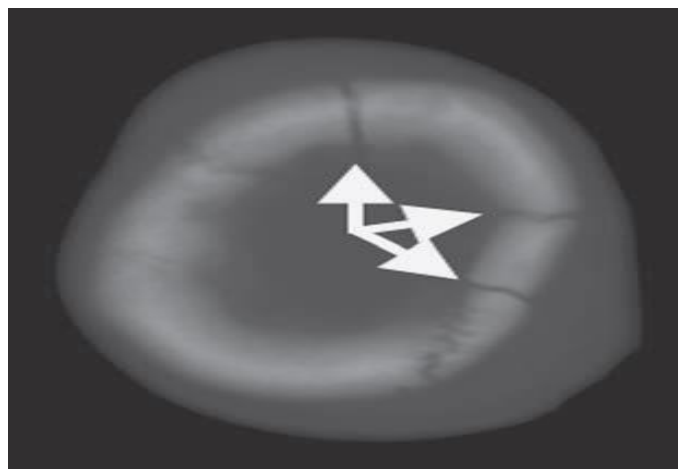


Fig.(2.16) Complex vault fracture (arrows). (Interpretation of
Emergency Head CT Erskine J.et.al 2008).

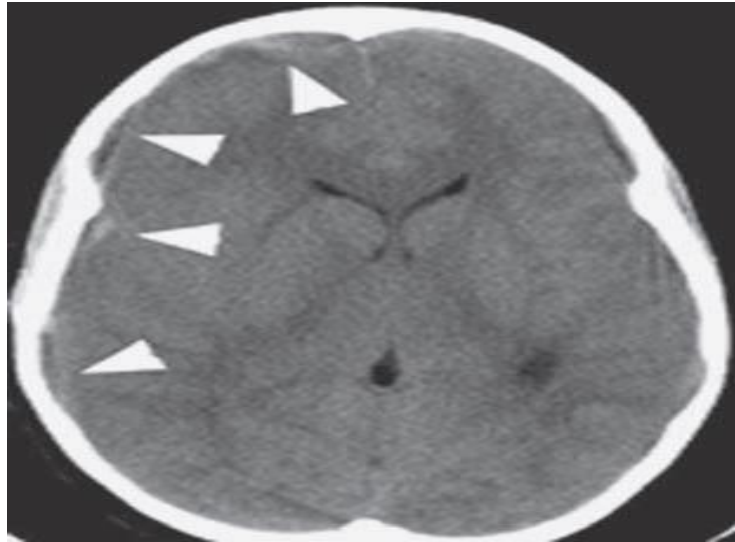


Fig.(2.17) Leptomeningitis: dense haemorrhagic leptomeningeal collection over the right cerebral hemisphere (arrowheads), in a young patient with sinusitis (Interpretation of Emergency Head CT Erskine J. et.al 2008).



Fig.(2.18) Acute hydrocephalus: periventricular low attenuation is seen (arrows) representing interstitial oedema from transependymal flow of CSF (Interpretation of Emergency Head CT Erskine J.et.al 2008).



Fig.(2.19) Cerebra I abscess. Scan. Right superior parietal ring Enhancing lesion (arrows), with surrounding vasogenic edema (Interpretation of Emergency Head CT Erskine J.et.al 2008).

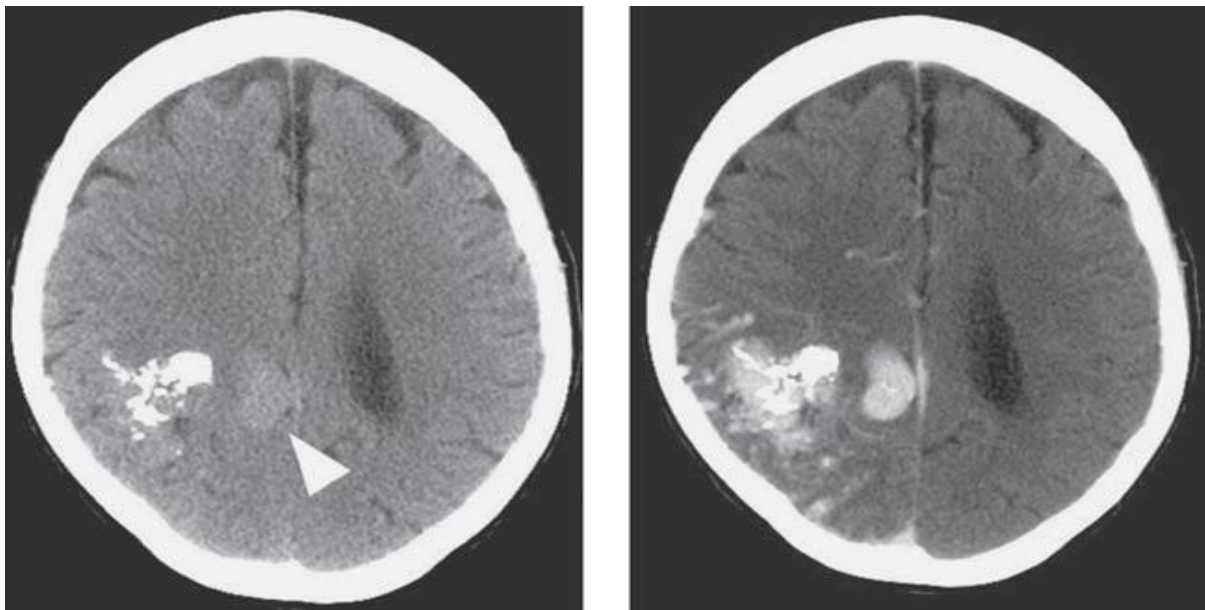


Fig.(2.20) Arterio venous malformation: mixed density lesion composed of coarse Calcification, and faintly hyper dense vessels (arrowhead). Marked enhancement post-contrast (Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.3 Fundamentals of CT imaging

2.1.3.1 History

In the early 1970s Sir Godfrey Hounsfield's research produced the first clinically useful CT scans. Original scanners took approximately 6 minutes to perform a rotation(one slice) and 20 minutes to reconstruct. Despite many technological advances since then, the principles remain the same. On early scanners the tube rotated around a stationary patient with the table then moved to enable a further acquisition. The machine rotated clockwise and counter-clockwise as power was supplied via a cable. Modern-day helical or spiral scanners obtain power via slip ring technology, thus allowing continuous tube rotation as the patient moves through the scanner automatically. This allows a volume of data to be acquired in a single rotation with the benefits of faster scanning, faster patient throughput and less re-imaging as patient movement Artifact is reduced. New multi-slice scanners use existing helical scanning technology but have multiple rows of detectors to acquire multiple slices per. In turn, advanced computer processing power allows reconstructive techniques, such as three-dimensional and multi planar reformats , to be more easily accessible. Consequently, scans are now performed routinely at a reporting workstation where the image can be viewed dynamically. (Interpretation of Emergency Head CT Erskine J.et.al 2008).

2.1.3.2 Windowing and grey scale

Modern CT scanners are able to differentiate in excess of 2000 CT numbers; however, the human eye can differentiate only around30 shades of grey. To maximize the perception of medically important features, images can be digitally processed to meet a variety of clinical requirements. The grey scale Values assigned to processed CT numbers on a display monitor, can be adjusted to suit special application requirements. Contrast can be enhanced by assigning just a narrow interval of CT numbers to the entire grey scale on the display

monitor. This is called window technique. The range of CT numbers displayed on the whole grey scale being called the window width and the average value the window level. Changes in window width alter contrast, and changes in window level select the structures in the image to be displayed on the grey scale, i.e. from black to white. Narrowing the window compresses the grey scale to enable better differentiation of tissues within the chosen window. . For example, in assessment of CT of the head, a narrow window of approximately 80 HU is allowing, used thus the eye to discriminate tissues only 2–3 HU apart. In practical terms, if we center the window at 30 HU, then CT numbers above 70 will appear white and those below –10 will appear black. This allows subtle Differences in tissue densities to be identified.

Conversely, if the window is widened to 1500 HU, then each detectable shade of grey would cover 50 HU and soft tissue differentiation would be lost however bone/soft tissue interfaces would be apparent.

In practical terms the window width and level are preset on the workstation and can be adjusted by choosing the appropriate setting, i.e. a window setting

For brain, posterior fossa, bone, etc. .(Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.3.3 Tissue characteristics

Unlike conventional radiography, CT has relatively good contrast resolution and can therefore differentiate resolution and can therefore differentiate resolution and can therefore differentiate between tissues which vary only slightly in density. This is extremely valuable when assessing the brain, as grey and white matter vary only slightly in density.

Artifacts aside, the densest structure in the head is bone, appearing white on CT. This is followed by acute hematoma, which is denser than flowing blood due to clot retraction and loss of water.

Blood is thought to be hyper dense due to the relative density of the hemoglobin, molecule. Brain can be differentiated into grey and white matter due to the difference in fatty myelin content between the two.

Typically white matter (higher fatty myelin content – HU_30) is darker than the adjacent grey matter (HU_40). Fat and air have low attenuation values and can be readily identified. CSF has a similar attenuation value to water appearing black, Pathological processes may become apparent due to edema within, or adjacent to, an abnormality Edema is less dense than normal brain.

Occasionally the use of a contrast medium will reveal an abnormality either due to the inherent vascular nature of a lesion or due to alteration in the normal blood brain barrier. Tumors may be very variable in their appearance, but may be hyper dense due to a high nuclear/cytoplasmic ratio or tumor calcification. (Interpretation of Emergency Head CT Erskine J.et.al 2008).

2.1.3.4 Reviewing a CT scan

Suggested systematic approach to interpretation

Check patient information and review scan protocol (e.g. non-contrast/ contrast enhanced), Check the scout image. May reveal a fracture or gross abnormality not obvious on the axial images, Review alignment of upper cervical vertebrae and A quick ‘first pass’ is recommend, noting gross pathology, followed by a more detailed analysis of the images. Use the mnemonic ‘ABBACS’ to remember important structures.

2.1.3.4.1 ABBACS

- (A) Asymmetry – Assess all slices comparing one side with another, remembering to allow for head tilt and to account for various forms of artefact.
- (B) Blood – Acute haemorrhage appears hyperdense in relation to brain, due to clot retraction and water loss.

Haemorrhage typically has a CT number in the range of 50–100 HU.

Assess the ventricles and CSF spaces for the presence or layering of blood.

Review the sulci and fissures for subtle evidence of a SAH.

Remember slow-flowing blood within a vessel can mimic clot.

2.1.3.4.2 Conversely clot within a vessel is an important diagnosis:

(A) Venous sinus thrombosis Dense MCA sign in acute CVA

(B) Brain:

Abnormal density: Hyper density – acute blood (free and within vessels), tumour, bone, contrast and artefact/foreign body and Hypo density – oedema/infarct, air and tumour.

Displacement: Look for midline shift.

Examine midline structures such as the falx cerebri, pituitary and pineal glands. Look for asymmetry of CSF spaces such as effacement of an anterior horn of the lateral ventricles or loss of sulcal pattern suggesting oedema

Assess for effacement of the basal cisterns and tonsillar herniation at the foramen magnum, as an indicator of raised intracranial pressure. (Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.3.4.3 Grey/white matter differentiation

Normal grey/white matter differentiation should be readily apparent; white matter is of slightly reduced attenuation in comparison to grey matter due to increased fatty myelin content.

In an early infarct, oedema leads to loss of the normal grey/white matter differentiation. This can be subtle and again only apparent when comparing both sides; identify normal structures such as internal capsule thalamus, lentiform and caudate nuclei.

(A) CSF spaces – Cisterns, sulci and ventricles

Assess the sizes of the ventricles and sulci, in proportion to each other and the brain parenchyma.

Identify normal cisterns (quadrigeminal plate, supra sellar and the mid brain region) and fissures (interhemispheric and Sylvian).

2.1.3.4.4 The ventricles often hold the key to analysing the image:

Pathology may be primary, within a ventricle, or may result from secondary compression from adjacent brain pathology.

If a ventricle is enlarged, consider whether it is due to an obstructive/ non-communicating or non-obstructive cause.

The former depends on site and the latter usually involves pathology in the subarachnoid space.

Dilatation ex vacuo is caused by loss/atrophy of brain tissue, often resulting in abnormal secondary enlargement of the adjacent ventricle. Small ventricles can be normal in children (increases in size with age).

Diffuse brain swelling can result in ventricular compression and reduced conspicuity of the normal sulcal/ gyral pattern. Causes include metabolic/anoxic injury, infection, trauma and superior sagittal sinus thrombosis.

(S) Skull and scalp – Assess the scalp for soft tissue injury. Can be useful in patients where a full history is absent. Can help to localise coup and contracoup injuries. Carefully assess the bony vault underlying a soft tissue injury for evidence of a fracture. Assess the bony vault for shape, symmetry and Mineralisation (focal sclerotic or lytic lesions).

Remember to adjust windowing to optimize bony detail. (Interpretation of Emergency Head CT Erskine J.et.al 2008)

2.1.3.5 Head CT

The basic positioning of skull which used for skull radiography are used for all cranial CT. it is very important to ensure that there is no rotation or tilt of head in order to demonstrate any bilateral asymmetry due to pathologic processes. Patient Supine Head first, Arms along the sides of the body ,Head immobilized in the head holder,Support is placed under the head

2.1.3.6 Brain Standard

Scout : Lateral

Landmark : OML

Gantry Tilt : 0° to 10°.from OM.

Slice Plane : Axial

Breath Hold : None

I.V. Contrast : As required (40-50ml)

Start Location : Foramen Magnum

End Location : Vertex

Slice Thickness : 5 mm -10 mm

2.1.3.7 Filming

Soft tissue(WW140-1000 WL 30-100)

Bone (WW 1500-3000 WL 200-400) (Protocols for Multislice Helical Computed Tomography, The fundamentals. by Peter Dawson).

2.2 Previous studies

-Raja Ismaeel Rukabi(2007) who studied the role of CT in emergency department his sample size was 5000 patients for different part of the body but may study at head only.

-The role of unenhanced CT alone for the management of headache in an emergency department. A feasibility study by Rizk B¹, Platon A, Tasu JP, Becker CD, Mendes Pereira V, Perneger T, Shanmuganathan K, Lövblad KO, Poletti PA. Whom studied suggests that a normal unenhanced CT might be sufficient to exclude the cause of headache in the initial screening of a selected group of patients admitted with cephalalgia. It compels researchers to perform further prospective studies to confirm the current data on a larger amount of patients..

-The role of unenhanced CT alone for the management of headache in An emergency department. A feasibility study by Benoît Rizk^a, Alexandra Platon^b, Jean-Pierre Tasu^a, Christoph D. Becker^b, Vitor Mendes Pereira^b, Kathirkamanathan Shanmuganathan Thomas Perneger^c, Pierre-Alexandre Poletti^b. This feasibility study suggests that a normal unenhanced CT might be sufficient to exclude the cause of headache in the initial screening of a selected group of patients admitted with cephalalgia. It compels researchers to perform further prospective studies to confirm the current data on a larger amount of patients.

-These studies agree with my study , the CT might be sufficient to exclude the cause of headache in the initial screening of a selected group of patients.

Chapter Three

Materials and Methods

Chapter three

Materials and Methods

3.1 Materials

3.1.1 Subjects:

100 patients who underwent CT examination in Khartoum Advanced Diagnostic Center (KADC) department and ALariat University department.

3.1.2 Machine used

In AL Rebate university Hospital department the Type of machine Siemence16slices and KADC department Toshiba 4slices.

3.2 Methods

3.2.1CT Protocol used

In both department used protocol The basic positioning of skull which used for skull radiography are used for all cranial CT Patient Supine Head first ,Arms along the sides of the body,Head immobilized in the head holder,Support is placed under the head ,slice plan Axial, slice thickness 5mm and printed two windows Soft-tissue and bone.

3.2.2Image interpretation

By Radiologist of department and Technologist of CT.

3.2.3 Data collection: Used Data collection Sheet

3.2.4 Analysis of data:

The use of descriptive analytical method using the SPSS statistical program based cross charts and graphs to demonstrate the possibility of the diagnosis. The test was used for Chi-square test study hypothesis which states can use the use of CT in the diagnosis. Analysis was done using SPSS. Chi square was used to determine association between the variables for each traumatic and non-traumatic emergency condition .best protocol will used in CT department for emergency cases. The collimation, table speed(pitch) and image reconstruction intervals should be clearly defined .the routine cranial CT protocol should include continuous sections,5-10mm thick, from the skull base through the

vertex displayed at window /level setting as follows. Bone:W 3000,L800
,Brain:W-90,L-40,Subdural or intermediate.

Chapter four

Results

Chapter four

Results

Table (4.1) Demonstrates Gender Distribution:

Gender	Number	%
Male	76	76%
Female	24	24%

Table (4.2) Headache Duration:

Time	Number	%
Hour	13	13%
Day	26	26%
Month	34	34%
Year	27	27%

Table (4.3) Diagnosis of Cases:

Diagnosis	Number	%
Normal	32	32%
Abnormal	68	68%

Table (4.4) Specification of abnormal Cases:

Pathology	Number	%
Hydrocephalus	10	15%
Haemorrhage	4	6%
Infarct	14	21%
Storke	6	9%
Calcification	1	1.4%
Haematoma	5	7%
Atrophy	2	3%
Meningitis	4	6%
Sol	1	1.4%
Shunt	1	1.4%
Tumor	5	7%
Skull fracture	9	13%
Oedema	2	3%
Abscess	2	3%
Cystic lesion	1	1.4%
Contusions	1	1.4%

The data was presented by the following figures

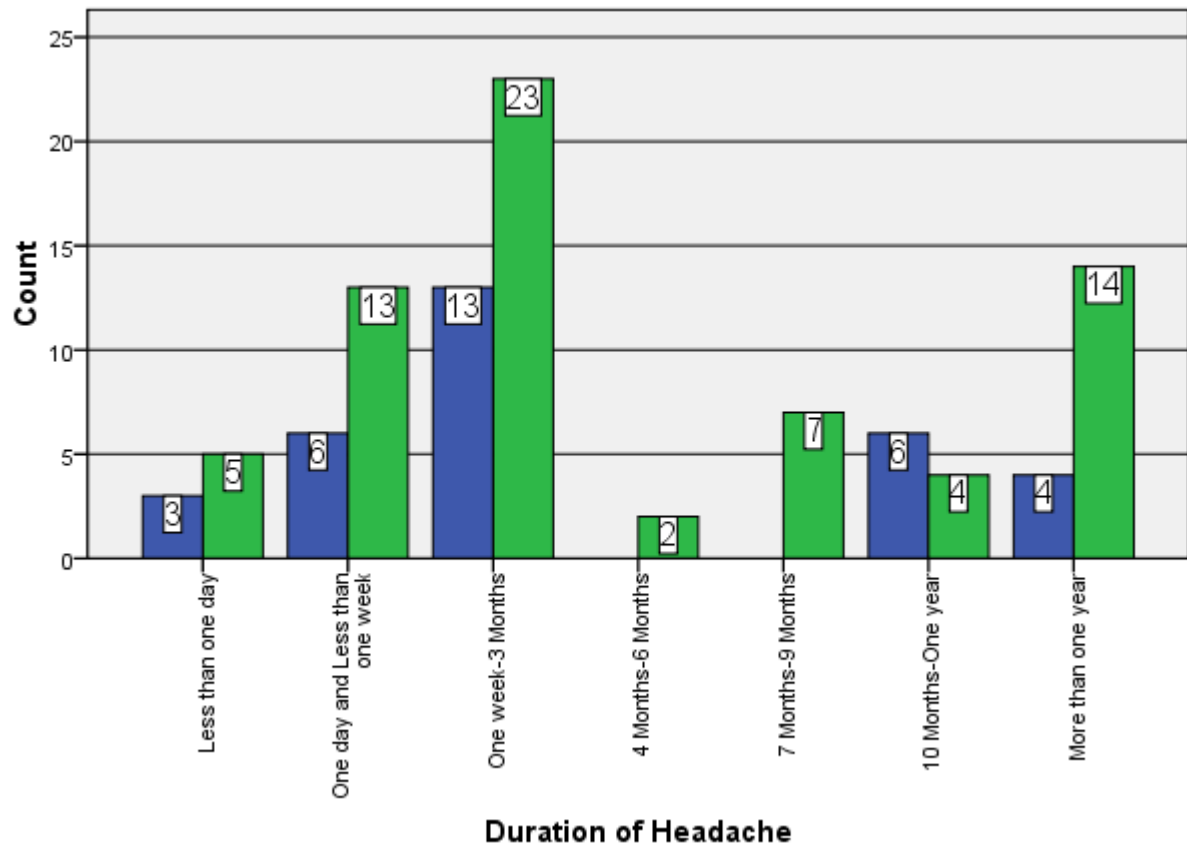


Figure (4.1) shows the frequency distribution of the result of the diagnosis by the length of time for headaches:

Chapter five

Discussion, conclusion and Recommendations

Chapter five

Discussion, conclusion and Recommendations

5.1 Discussion:

The current study is important in that it is the first large scale study that look at the profile of CT scans carried out of at a multi –specialty hospital. The advent of this useful radiological tool has resulted in enhancement of diagnostic process and also management aspect in almost all field of medicine.

The study conducted in Khartoum Status at CT departments for emergency cases in AL Rebat University hospital department and Advanced Khartoum center. It is interesting to note that CT diagnosis was the same as that suspected clinically in majority of the scans.

Therefore the slightly increased initial expense (to do a CT) is made up by getting greater information of clinical diagnosis. Patients were categorized based on their clinical diagnoses. The CT scans of the head constituted the majority in department due to increased trauma in the contrary. These included intracranial hemorrhage; head Injuries, CVA, ICSOL, Hydrocephalous ,AVM, Haemorrhage,Infarct,Storke,Calcification,Haematoma,Atrophy,Meningitis, Shunt, Tumor, Skull fracture, Edema , Abscess, Cystic lesion, Contusions.

The advantages are two folds: this would allow the clinician to choose cases for imaging more judiciously and also offers the radiologist a better vantage point to interpret the radiological findings.

The major strength of the study is the large Number of cases and variety of departments from which patients were referred. Since the analysis was carried out by an independent person the possibility of bias was eliminated. The limitations of the study include difficulty in follow up. Follow up was

possible only if the patient was re-attending the center for A repeat CT. Because of this the clinical perspective may not have been evident and the study was pre dominantly from a Radiological view point.

5.2 Conclusion

The study concluded the CT scan is best modality in diagnosis for headache causes in emergency departments .

Computed Tomography (CT) has revolutionized the imaging work-up of patient in the emergency department.

CT now is considered to be one of the most valued tools in the diagnostic work –up of trauma patient and patients with non-traumatic emergency conditions.

Today, most emergency centers are equipped with CT scanners that are available for the evaluation of emergency patients, 24hours a day, and 7days a week. During the past 20 years, improvements in scanner hardware and soft were have provided increased scanning speed and faster, data acquisition, as well as improved spatial resolution and increased low contrast detectability.

As a consequence, emergency patients now benefit from faster and more accurate CT examination. Helical (or spiral) CT, the most important CT development of the current decade, permits simultaneous patient translation and data acquisition through the use of continuously rotating x-ray source and detector array combined with the use of a high –heat-capacity x-ray tube.

Helical CT offers a number of imaging advantages for the emergency patient. Helical technology permits a head CT scene within 20-30 minutes of total scanner room time. Newer multi detector scanners may decrease this time to 10-15 minutes.

5.3 Recommendations

- Clear justification of examination is highly recommended avoid repetition of examination (CT examinations should not be repeated without clinical justification).
- The use of current modulation and dose reduction techniques.
- Requests of CT scanning must be generated only by qualified medical Practitioners and justified by both the referring doctor and the radiologist.
- Design a proper requesting form for CT examination and rule out the responsibility of technologists, physicians, and radiologists for reducing of radiation dose and image quality.

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Appendix

Appendix (1): The sheet of data collection:

No	Age	sex	Clinical Indication	Duration of Headache	Diagnosis		CT PRTOCAL		
					Normal	Abnormal	Patient position	Slice plane	Slice thick nees
1	60y	F	Headache	2month	Normal		Supine	axial	5mm
2	4y	F	- headache	3 years	Abnormal	Hydrocephalus	Supine	axial	5mm
3	18y	M	Headache	1day	Normal		Supine	axial	5mm
4	8y	F	Headache	15day	Normal		Supine	axial	5mm
5	45y	M	Headache	9month	Abnormal	Haemorrhage	Supine	axial	5mm
6	39y	M	Headache	7day	Normal		Supine	axial	5mm
7	35Y	F	Headache	6 day	Normal		Supine	axial	5mm
8	20 Y	M	Headache	3day	Abnormal	Hydrocephalus	Supine	axial	5mm
9	53Y	M	Headache	3day	Normal		Supine	axial	5mm
10	60Y	M	Headache	3day	Abnormal	Infarct	Supine	axial	5mm
11	40y	M	Headache	2day	Abnormal	Haemorrhage	Supine	axial	5mm
12	50y	M	Headache	3day	Abnormal	Storke	Supine	axial	5mm
13	2day	F	Headache	2day	Abnormal	Hydrocephalus	Supine	axial	5mm
14	8y	F	Headache	3horus	Abnormal	Skull fracture	Supine	axial	5mm
15	5y	F	Headache	3month	Abnormal	Hydrocephalus	Supine	axial	5mm
16	17	M	Headache	1year	Normal		Supine	axial	5mm
17	24	M	Headache	3year	Abnormal	Calcification	Supine	axial	5mm

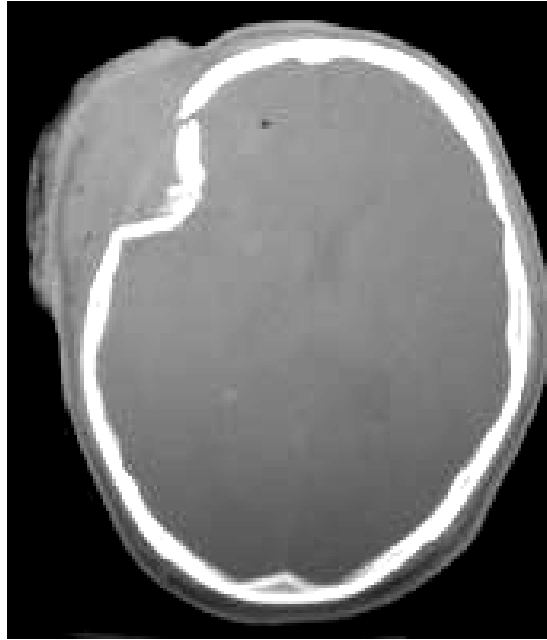
18	20y	M	Headache	1year	Normal		Supine	axial	5mm
19	17y	M	Headache	7day	Normal		Supine	axial	5mm
20	40	M	Headache	1day	Abnormal	Haematoma	Supine	axial	5mm
21	18y	M	Headache	6day	Abnormal	Atrophy	Supine	axial	5mm
22	57y	F	Headache	7month	Abnormal	Hydrocephalus	Supine	axial	5mm
23	85y	M	Headache	1month	Abnormal	Infarct	Supine	axial	5mm
24	70y	M	Headache	3day	Abnormal	Infarct	Supine	axial	5mm
25	35y	M	Headache	7day	Abnormal	Infarct	Supine	axial	5mm
26	54d	M	Headache	5day	Abnormal	Meningitis	Supine	axial	5mm
27	75y	F	Headache	3year	Abnormal	Infarct	Supine	axial	5mm
28	35y	M	Headache	2hours	Abnormal	Haematoma	Supine	axial	5mm
29	37y	M	Headache	2hours	Abnormal	Headfracture	Supine	axial	5mm
30	65y	M	Headache	2year	Abnormal	Haematoma	Supine	axial	5mm
31	67y	M	Headache	7hours	Normal		Supine	axial	5mm
32	43y	F	Headache	1month	Abnormal	Sol	Supine	axial	5mm
33	55y	F	Headache	1year	Normal		Supine	axial	5mm
34	85y	F	Headache	2month	Abnormal	Stroke	Supine	axial	5mm
35	23y	M	Headache	2year	Normal		Supine	axial	5mm
36	65y	F	Headache	1day	Abnormal	Stroke	Supine	axial	5mm
37	70y	M	Headache	2month	Normal		Supine	axial	5mm
38	20y	M	Headache	20 day	Abnormal	Shunt	Supine	axial	5mm
39	45y	M	Headache	2year	abnormal	Meningioma	Supine	axial	5mm

40	81y	M	Headache	day7	Abnormal	Meningioma	Supine	axial	5mm
41	85y	M	Headache	7day	Abnormal	Interaction	Supine	axial	5mm
42	12y	M	Headache	1hours	Normal		Supine	axial	5mm
43	47y	F	Headache	4month	Abnormal	Skull fracture	Supine	axial	5mm
44	22y	M	Headache	1year	Normal		Supine	axial	5mm
45	75y	M	Headache	8month	Abnormal	Skull fracture	Supine	axial	5mm
46	29y	F	Headache	9month	Abnormal	Tumor	Supine	axial	5mm
47	37y	M	Headache	1month	Abnormal	Tumor	Supine	axial	5mm
48	32y	M	Headache	2year	Normal		Supine	axial	5mm
49	51y	F	Headache	7month	Abnormal	Tumor	Supine	axial	5mm
50	17y	M	Headache	1hours	Abnormal	skull fracture	Supine	axial	5mm
51	65y	M	Headache	1year	Abnormal	Haemorrhage	Supine	axial	5mm
52	85y	M	Headache	1year	Normal		Supine	axial	5mm
53	20y	M	Headache	2year	Abnormal	Medulloblastom a	Supine	axial	5mm
54	17y	M	Headache	2day	Abnormal	Oedema	Supine	axial	5mm
55	46y	F	Headache	3month	Normal		Supine	axial	5mm
56	18y	M	Headache	1month	Abnormal	Skull fracture	Supine	axial	5mm
57	62y	M	Headache	2year	Abnormal	Infarction	Supine	axial	5mm
58	23y	F	Headache	1month	Normal		Supine	axial	5mm
59	58y	M	Headache	8month	Abnormal	Tumor	Supine	axial	5mm
60	50y	M	Headache	2year	Normal		Supine	axial	5mm
61	3y	M	Headache	2year	Abnormal	Hydrocephalus	Supine	axial	5mm
62	10y	M	Headache	1year	Abnormal	Haematoma	Supine	axial	5mm
63	31y	M	Headache	24hours	Normal		Supine	axial	5mm
64	45y	M	Headache	72hours	Abnormal	Abscess	Supine	axial	5mm

65	80y	F	Headache	2month	Abnormal	Infarct	Supine	axial	5mm
66	12y	M	Headache	1month	Abnormal	Contusions	Supine	axial	5mm
67	52y	M	Headache	2year	Abnormal	Abscess	Supine	axial	5mm
68	45y	M	Headache	5month	Abnormal	Meningioma	Supine	axial	5mm
69	48y	M	Headache	24hours	Normal		Supine	axial	5mm
70	33y	M	Headache	7day	Abnormal	Skull fracture	Supine	axial	5mm
71	26y	M	Headache	3year	Normal		Supine	axial	5mm
72	55y	M	Headache	1month	Abnormal	Infarct	Supine	axial	5mm
73	38y	M	Headache	2month	Normal		Supine	axial	5mm
74	44y	M	Headache	3month	Abnormal	Infarct	Supine	axial	5mm
75	28y	M	Headache	2day	Abnormal	Oedema	Supine	axial	5mm
76	12y	M	Headache	2month	Normal		Supine	axial	5mm
77	10y	M	Headache	2month	Abnormal	Cystic lesion	Supine	axial	5mm
78	55y	M	Headache	3month	Abnormal	Infarct	Supine	axial	5mm
79	15y	M	Headache	2year	Abnormal	Hydrocephalus	Supine	axial	5mm
80	7y	M	Headache	1year	Abnormal	Atrophy	Supine	axial	5mm
81	70y	M	Headache	2year	Abnormal	Infarct	Supine	axial	5mm
82	55y	M	Headache	2month	Abnormal	Stroke	Supine	axial	5mm
83	75y	M	Headache	1year	Normal		Supine	axial	5mm
84	18y	M	Headache	24hours	Abnormal	Skull fracture	Supine	axial	5mm
85	26y	F	Headache	1hours	Normal		Supine	axial	5mm
86	65y	M	Headache	2year	Abnormal	Stroke	Supine	axial	5mm
87	27y	F	Headache	24hours	Normal		Supine	axial	5mm
88	41y	M	Headache	2month	Normal		Supine	axial	5mm
89	56y	M	Headache	27day	Normal		Supine	axial	5mm
90	9y	F	Headache	2hours	Abnormal	Hydrocephalus	Supine	axial	5mm

91	70y	F	Headache	15day	Abnormal	Infarct	Supine	axial	5mm
92	26y	M	Headache	1month	Normal		Supine	axial	5mm
93	80y	M	Headache	2month	Abnormal	Infarct	Supine	axial	5mm
94	38y	M	Headache	1month	Normal		Supine	axial	5mm
95	2y	M	Headache	7month	Abnormal	Hydrocephalus	Supine	axial	5mm
96	65y	M	Headache	5year	Abnormal	Haemorrhage	Supine	axial	5mm
97	11y	M	Headache	1year	Abnormal	Hydrocephalus	Supine	axial	5mm
98	35y	F	Headache	3month	Abnormal	Fracture	Supine	axial	5mm
99	53y	M	Headache	3month	Abnormal	Stroke	Supine	axial	5mm
100	49y	M	Headache	3year	Abnormal	Tumor	Supine	axial	5mm

Appendix (2): depressed skull fracture .



Patient from KAC department ,have 23year old ,male, traumatic patient.