

Chapter (1)

General Introduction

1-1Introduction: -

The story of MRI is along one of courtship between physics and medicine. In 1952 , Dr Bloch from Stanford university and Dr Purcell from Harvard university were awarded the Nobel prize for their work on what was then known as nuclear magnetic resonance (NMR) . However, the turning point came after 20 years with the advent of computers in medical imaging. By this time, the word nuclear is substituted and it is now known as Magnetic Resonance Imaging (MRI).

MRI is non-invasive procedures that produce images in three planes and give suitable diagnosis in case of cerebral infarction than other radiological modalities.

Magnetic Resonance Angiography (MRA) is highly accurate for detection of stenosis greater than 50% or occlusion, Furthermore, as expected, gadolinium enhanced MRA yielded consistently better results. While MRA is less invasive than traditional angiography, and typically less expensive and detailed images of blood vessels and blood flow are obtained without having to insert a catheter directly into the area of interest, so that there is no risk of damaging an artery. No exposure to ionizing radiation during MRI study. With magnetic resonance imaging any desired plane can be detected. In clinical practice however particular planes are preferred, such as

coronal, sagittal or axial planes. This procedure of representing the human in several series of parallel slices is known, as multi planner representation. A comparison between these different sectioning planes of the head can provide information or the complex 3- dimensional structure of the anatomical and neurofunctional system. The modern imaging technique clarify the indication for any treatment and enable one to assess better balance between risk and benefit .For example in case of intracranial hemorrhage or brain abscesses. The time and the advisability of an operation are better identified. Tumors can be more accurately located for subsequent operation and radiotherapy, in order to protect threatened neurofunctional system in the best possible way. A recurrence can be detected at much earlier stage. In modern medical practice evaluation of result and failures of conversation and surgical treatment can be verified much sooner and more accurately than in the days before these new imaging modalities and techniques (MRI/MRA).

1.2 Proposal: -

1.2.1 Problem of the study: -

MRA is accurate techniques that demonstrate blood vessels (blood flow in circle of Willis) in case of cerebral infarction.

1.2.2 Objectives of the study: -

- To determine the role of MRA in cerebral infarction.
- To measure the sensitivity of MRA in diagnosis of cerebral infarction.
- To explain the suitable technique that used to demonstrate cerebral infarction.
- To show different appearance of abnormality causes cerebral infarction.

1.2.3 Importance of the study: -

Due to previous problem in other modalities MRA is very important because MRA is the best technique, which used to determine the degree of infarction and the causes of lack blood flow.

1.2.4 Hypotheses: -

MRA is an accurate technique that detect and diagnosis cerebral infarction.

1.2.5 Methodology: -

30 patients are selected with different ages and sex, and diagnosed with MRI but fail to show the site of obstruction. These patients underwent MRA examination to detect the level and degree of infarction.

1.2.6 Data analysis:-

The data analyzed statistically by percentage on graphs.

1.2.7 Area of the study and duration:-

The study was taken place in Khartoum Advanced Diagnostic Center (KADC). And duration is about six months.

1.2.8 The scope of the study: -

The study contains five chapters:

- Chapter one:

Introduction and proposal of research.

- Chapter two:

Is the literature review which consist of the following:

- Anatomy of the brain.
- Physiology of the brain.
- Pathology of the brain.
- MRI equipments.
- MRI techniques.
- MRA appearance.
- MRI/MRA advantages and disadvantages.

- Chapter three:

Material, method and film evaluation.

- Chapter four:

Results and data analysis.

- Chapter five:

Discussion, conclusion and recommendations.

Appendixes.

References.

Chapter (2)

Literature Review

2-1 Anatomy of CNS: -

The skull, the skeleton of the head, is the most complex bony structure in the body because it encloses the brain, which is irregular in shape, houses the organs of special senses for seeing, hearing, testing and smelling.

2-1-1 Sulci and Fissures of the cerebral cortex:

These elongated depressions are distinctive landmarks that are used to subdivide the cerebral hemispheres in to smaller areas for descriptive purpose.

There are six main Sulci and Fissures:

2-1-1-1 The longitudinal cerebral Fissures:-

This long, deep, median cleft incompletely separates the two cerebral hemispheres.

2-1-1-2 The transverse cerebral fissure: -

This cleft separates the cerebral hemispheres superiorly from the cerebellum, midbrain and diencephalons inferiorly. The tentorium cerebelli lies in the posterior part of this fissure.

2-1-1-3 The lateral sulcus: -

This deep cleft begins inferiorly on the inferior surface of the cerebral hemisphere as a deep tissue, separating the frontal

and temporal lobes. Posteriorly, it separates part of the parietal and temporal lobes.

2-1-1-4 The central sulcus: -

This prominent groove runs inferoanteriorl from about the middle of the superior margin of the cerebral hemisphere, just short of the lateral sulcus. The central sulcus is an important landmark of the cerebral cortex because the motor area (pre-central gyrus) lies anterior to it and the general sensory cortex (post- central gyrus) posterior to it.

2-1-1-5 The parieto-occipital sulcus:

This deep fissure, as it name indicates, separates the parietal and occipital lopes of the brain on the medial aspect of the brain. It extends from the calcarine sulcus to the superior border of the cerebral hemispheres and continues for a short distance on the dorsolateral surface.

2-1-1-6 The calcarine sulcus: -

This groove on the medial surface of the cerebral hemisphere commences inferior to the posterior end of the corpus callosum and follows an arched course to the occipital pole.

2-1-2 Gyri of cerebral cortex: -

Agyrus or convolution is an area of cerebral cortex that lies between two Sulci or fissures. Most of the cortex of a gyrus is not visible on the surface of the brain.

The important gyri are: The pre- central gyrus, between the central and pre- central fissures, the post- central gyrus, between the central and post central fissures, the supra marginal gyrus, around the end of the lateral sulcus, the angular gyrus, around the end of the superior temporal fissures, the superior temporal fissure, and the cingulated gyrus on the medial aspect of the hemisphere, between cingulated gurus and the corpus callosum.

2-1-3 The main lobes of the cerebral hemispheres:-

There are four main lobes:

Frontal

Parietal

Temporal

Occipital

2-1-3-1 Frontal lobes: -

These are the largest of all the lobes, and they form the anterior part of the cerebral hemispheres. They are located anterior to the central Sulci and superior to the lateral Sulci, their lateral and superior surfaces extent posterior to the coronal suture of the skull. The basal surfaces of the frontal lobes rest on the orbital parts of the frontal bone in the anterior cranial fossa.

2-1-3-2 Parietal lobes: -

These lobes are related to the internal aspects of the posterior and superior parts of the parietal bones. Each lobe is bounded anteriorly by the central sulcus and posteriorly by the

superior part of the line joining the parieto-occipital sulcus and preoccipital notch.

The inferior boundary of each lobe is indicated by an imaginary line extending from the posterior ramus of the lateral sulcus to the inferior and of the posterior boundary of the lobe.

2-1-3-3 Temporal lobes: -

These lobes lie inferior to the lateral Sulci. Their convex anterior ends, called temporal poles, fit into the anterior and lateral parts of the middle cranial fossa. Their posterior parts lie against the middle one-third of the inferior part of the parietal bone.

2-1-3-4 Occipital lobes: -

These lobes are relatively small and are located posterior to the parieto-occipital Sulci. They rest on the tentorium cerebelli, superior to the posterior cranial fossa. Although they are small, these lobes are very important because they contain the visual area.

2-1-4 The main parts of the brain: -

2-1-4-1 The cerebral hemispheres: -

It is form the largest part of the brain. They occupy the anterior and middle cranial fossa and extent posteriorly over the tentorium cerebelli and the cerebellum to the internal occipital protuberance. They comprise the cerebral cortex, the basal ganglia their fiber connections, and the lateral ventricles. The

cavity in each cerebral hemispheres, known as a lateral ventricle, is part of the ventricular system of the brain. (Fig 2-1)

2-1-4-2 The Diencephalons: -

This part of the brain is composed of the thalamus, hypothalamus, epithalamus and sub thalamus. It surrounds the third ventricle of the brain and forms the central core of the brain. It is surrounded by the cerebral hemispheres. The two thalami make up four-fifths of the diencephalons. The cavity of the diencephalons is the narrow third ventricle lying between the right and left thalami.

2-1-4-3 The midbrain: -

This is the smallest part of the brain. It is located at the junction of the middle and posterior cranial fossae, lying partly in each. The cavity of the mid brain is represented by an arrow canal, called the cerebral aqueduct. It conducts CSF from the lateral and third ventricles to the fourth ventricle.

2-1-4-4 The pons: -

It lies in the anterior part of the posterior cranial fossa, posterior to the superior part of the clivus and the posterior surface of the dorsum sellae. The Latin term pons means a bridge, which is an appropriate name for this part of the brain because the part of it that is visible from the inferior surface is a wide bridge like transverse band of nerve fibers, although it appears to be bridging between the cerebellar hemispheres. The pontine fibers connect one cerebral hemisphere with the

opposite cerebellar hemisphere. The cavity in the pons forms part of the fourth ventricle.

2-1-4-5 The medulla oblongata: -

This is the most caudal part of the brain stem, composed of the midbrain, pons and medulla. It is located in the posterior cranial fossa with its ventral aspect facing the clivus. The medulla is continuous with the spinal cord at the foramen magnum, but the transition is gradual. The distinctive characteristics of its ventral surface are the elongated pyramids, which contain the cortico-spinal tracts from the cerebral cortex. The medulla contains the cardiovascular and respiratory centers for the automatic control of heartbeats and respiration. The cavity of the medulla forms the inferior part of the fourth ventricle.

2-1-4-6 The Cerebellum: -

This little brain over lies the posterior aspects of the pons and medulla and extends laterally beneath the tentorium cerebelli. It occupies most of the posterior cranial fossa. The cerebellum consists of a midline portion; the vermis and the lateral lobes or hemispheres. These hemispheres lie posterior to the petrous part of the temporal bones and rest in the concavities of the posterior cranial fossae. The cerebellum is mainly concerned with motor functions that regulate posture, muscle tone and muscular coordination.

2-1-5 Ventricular system and CSF: -

The ventricular system in the brain consists mainly of four cavities called ventricles. The first and second ventricles, called lateral ventricles are the largest components of the system. They occupy a considerable part of cerebral hemispheres. Each lateral ventricle opens in the third ventricle through an interventricular foramen. The third ventricle is a narrow, slit like cavity between the two thalami. It is continuous posteriorly with the cerebral aqueduct in the midbrain, which connects the third and fourth ventricles.

The fourth ventricle is diamond-shaped when viewed superiorly and tent-shaped when observed laterally. It is located in the pons, anterior to the cerebellum, and extends posteriorly in to the central canal of the spinal cord.

2-1-6 Blood supply of the brain: -

The brain is supplied through an extensive system of branches from two pair of vessels, the internal carotid arteries and the vertebral arteries.

2-1-6-1 The internal carotid arteries: -

Each artery arises in the neck from the common carotid artery opposite the superior border of the thyroid cartilage. The cervical part of the artery ascends almost vertically to the base of the skull, where it turns and enters the carotid canal in the petrous part of the temporal bone. The petrous part of the artery enters the middle cranial fossa through the superior part of the

foramen lacerum and then runs anteriorly in the cavernous sinus and the cavernous part of the internal carotid artery is covered by the endothelium of this sinus. At the anterior end of the cavernous sinus, the internal carotid artery makes hairpin turn and leaves the sinus to enter the subarachnoid space.

The cerebral part of the internal carotid artery immediately gives off the important ophthalmic artery, which supplies the eye. The internal carotid artery then passes inferior to the optic nerve. Finally it turns obliquely superiorly, lateral to the optic chiasma for a variable distance, before branching into the anterior and middle cerebral arteries at the medial end of the lateral sulcus. The sinus course taken by the cavernous and cerebral parts of the internal carotid artery forms a U-shaped bend, often called the carotid siphon. Within the cranial cavity, the internal carotid artery and its branches supply the hypophysis cerebri (pituitary gland), the orbit and much of the supratentorial part of the brain.

2-1-6-2 The vertebral arteries: -

Each artery begins in the root of the neck as a branch of the first part of the subclavian artery. It ascends vertically through the transverse foramina of the cervical vertebrae and then inclines laterally in the transverse foramen of C2 vertebra. Superior to this foramen, the vertebral artery ascends vertically into the transverse foramen of C1. It then bends posteriorly at

right angles and winds around the superior part of the lateral mass of the atlas.

The vertebral artery pierces the posterior atlanto-occipital membrane and the dura, and the arachnid. It enters the subarachnoid space of the cerebellomedullary cistern at the level of the foramen magnum. The vertebral artery runs anteriorly on the anterolateral surface of the medulla and unites with its fellow of the opposite side at the caudal border of the pons to form the basilar artery.

2-1-6-3 The basilar artery: -

The union of the two vertebral arteries forms this artery. It runs through the pontine cistern to the superior border of the pons, where it ends by dividing into the two posterior cerebral arteries.

2-1-6-4 The cerebral arterial circle (circle of Willis): -

This circle is important anastomoses between the four arteries that supply brain (the two vertebral and the two internal carotid arteries). The posterior cerebral, posterior communicating, internal carotid, and anterior cerebral and anterior communicating arteries form it. The circle is located at the base of the brain; principally in the interpeduncular fossa .It extends from the superior border of the pons to the longitudinal fissure between the cerebral hemispheres. The cerebral arterial circle encircles the optic chiasma, the infundibulum, and the

mamillary bodies. Two types of branches, central and cortical, arise from the cerebral arterial circle and the main cerebral arteries. Central arteries penetrate the substance of the brain and supply deep structures. Cortical branches pass in the pia and supply the more superficial parts of the brain. In general each of the cerebral arteries, (anterior, middle and posterior) supplies a surface and a pole of the brain.

The anterior cerebral artery supplies most of the medial and superior surface and frontal pole. The middle cerebral artery supplies the lateral surface and the temporal pole. The posterior cerebral artery supplies the inferior surface and the occipital pole.

2-1-7 The venous system: -

The venous drainage of the brain ultimately reaches the venous sinuses of the dura mater. The superior cerebral veins drain on to the superior sagittal sinus. The inferior cerebral veins drain in to the transverse sinus. The superficial middle cerebral vein drains into the cavernous sinus, and the deep middle cerebral vein and anterior cerebral vein drain in to the basal vein.

2-1-7-1 The basal vein of (Rosenthal): -

The anterior cerebral, deep middle cerebral and striate veins form the basal vein. The thalamostriate, septal and choroids veins form the internal cerebral vein. The union of the

two internal cerebral veins forms the great cerebral vein. It receives the basal veins before it joins the inferior sagittal sinus.

2-2 Physiology: -

2-2-1 Introduction: -

The vast complexity of the body functions controlled by the nervous system is unique. These functions range from the somatic motor and sensory functions to highly integrated functions, such as perception, memory, learning and emotions.

The central nervous system (CNS) consists of the brain and spinal cord. The peripheral nervous system, on the other hand, consists of 43 pairs of nerves, which enter and leave the CNS. Afferent (sensory) fibers carry nerve impulses from the periphery to the CNS and efferent (motor) fibers carry impulses from CNS to effector organs, i.e. muscles and glands.

The nervous system responds to changes in the external and internal environments. Receptors sensitive to various stimuli (e.g. light, sound, touch, blood pressure, muscle tension, etc.) are excited. Nerve impulses carried along afferent fibers to the CNS are processed and integrated before appearing in the efferent fibers to produce appropriate responses in the effector organs (e.g. muscle contraction, glandular secretion). The pathway the impulses follow from receptor to effector is called the reflex arcs vary in complexity. They are simple at the level of the spinal cord, but get more complex at the level of the brain stem. The most complex reflex arcs are in the cerebral cortex. (Fig 2-2).

2-2-2 Cells of the nervous system: -

2-2-2-1 Neurons: -

The neuron is the functional unit of the nervous system and the human brain has 10^{11} neurons. These vary in size and shape, but they all basically consist of the cell body (soma), one or more dendrites and only one axon, with synaptic terminals. The soma contains the nucleus and the cytoplasm as contains granules, which stain with basic, dyes, known as Nissl substance. These are the source of the neuron's protein. Dendrites are tapered branches from the cell body that make contact with other neurons. Both soma and dendrites receive information from synaptic connections with other neurons. They are thus usually considered to be the sensory end of the neuron.

The signal axon varies in length (from a few hundred micrometers to more than a meter) and also diameter. It arises from the part of the soma known as the axon hillock, which is the most excitable part of the neuron. Axons are usually covered by myelin sheaths and their ends branch to form terminals, which make contact with other neurons at synapses (Fig 2-3).

A synapse consists of:

- A presynaptic terminal, which contains the synaptic vesicles enclosing a neurotransmitter.
- A synaptic cleft, which is a small space (20-30 nm wide) between neurons that the transmitter has to cross to arrive at the postsynaptic neurons.

- The postsynaptic membrane, which contains the receptors specific to a particular neurotransmitter.

It is important to note that, by about the 6th month of age, the maximum number of neurons attained, because mature neurons do not multiply. Therefore new ones will not replace dead neurons. In fact, there is progressive loss of neurons from birth onwards, due to the normal ageing process.

2.2.2.2 Neuroglia: -

Neuralgia (or supporting cells): is the matrix of the CNS and maintain its structure. There are three types of neuralgia.

2-2-2-2-1 Astrocytes:

Are large in size and its function is also to maintain a constant k^+ environment around neurons. This is necessary for the normal functioning of the nerve cell.

2-2-2-2-2 Oligodendroglia:

They are responsible of formation and maintenance of myelin sheaths around axons, similar to the Schwann cells in the periphery.

2-2-2-2-3 Microglia:

Are the macrophages of the CNS which phagocytes tissue debris, thus helping the process of tissue repair.

2-2-3 Functional anatomy of the CNS: -

2-2-3-1 Spinal cord: -

The spinal cord consists of segment, each of which has a pair of nerve root, on each side. The dorsal roots carry impulses from peripheral receptors into spinal cord, while the ventral roots carry impulses to the periphery (i.e. muscles). Grey matter forms the core of the spinal cord and appears like the letter 'H' in cross-section. It contains the cell bodies of neurons. White matter surrounds the grey matter. It is made up of ascending (sensory) and descending (motor) tracts. (Fig 2-4)

2-2-3-2 The spinal cord functions include:

Transmission of sensory serving as a centre for some reflexes.

2-2-3-3 Brain: -

The brain can be divided into four subdivisions:

Brain stem.

Diencephalons.

Cerebellum.

Cerebrum.

2-2-3-3-1 Brain stem:

The brain stem consists of the medulla, pons and midbrain. The medulla forms the upper extension of the spinal cord. It contains motor and sensory nuclei of the throat, mouth and neck, and nuclei for respiratory and cardiovascular control centers.

The medulla also contains the nuclei of cranial nerves; it also contains the nuclei of cranial nerves IX, X, XI, and XII.

The pons is continuous with the medulla and also contains control centers for the respiratory and cardiovascular systems. It also contains nuclei of some sensory and motor nerve (i.e. nuclei of cranial nerve V, VI, VII and VIII).

The midbrain is continuous with the pons below and the diencephalons above. It contains nuclei of cranial nerves III and IV, which mediate papillary reflexes and eye movement. The reticular formation of the brain stem extends from the medulla through the pons to the midbrain. It is composed mainly of ascending and descending tracts and some nuclei. The reticular formation plays an important role in the control of muscle tone and in arousal or alerting mechanisms.

2-2-3-3-2 Diencephalons: -

The diencephalons is composed of the two thalami laterally and the hypothalamus ventrally. Thalamic nuclei are functionally divided in to several groups. The most important of these are as follows. One group relays all types of sensation to the sensory cortex except olfaction. Another group relays signals from the cerebellum and basal ganglia to the motor cortex. The third group controls the general level of activity of the whole cerebral cortex and is therefore responsible for the level of consciousness. The hypothalamus is the higher autonomic centre (e.g. control of blood pressure, heart rate and body temperature).

It also secretes hormones that control the release of other hormones from the pituitary gland. Being part of the limbic system, the hypothalamus plays a role in the generation of emotions. There is also centers play control of appetite and water intake.

2-2-3-3-3 Cerebellum: -

The cerebellum lies in the posterior aspect of the brain stem and is connected to it by three thick bundles of white matter, which carry impulses in and out of the cerebellum. The cerebellum is concerned with:

Control of rate, range and direction of movement.

Control of muscle tone.

Control of equilibrium and posture.

2-2-3-3-4 Cerebrum: -

The cerebrum consists of the right and left cerebral hemispheres, connected in the midline by the corpus callosum. The superficial layer of each hemisphere is composed of grey matter, the cerebral cortex. This encloses a large area of white matter, inside which lie a number of nuclei, known as basal ganglia. These play a very important role in the planning and control of movement.

The white matter of the cerebral hemisphere constitutes three types of fibers:

association fibers:

These connect gyri in the same hemisphere

projection fibers:

These form the ascending and descending tracts, which carry impulses to and from the hemispheres.

commissural fiber:

These connect gyri of one hemisphere with corresponding gyri of the other hemisphere.

The most important commissural is the corpus callosum.

2-2-4 The sensory system: -

The sensory system provides us with information about our environment-both the external and the internal environment. All information comes to us through our sense organs, which contain structures called receptors. Receptors are therefore detectors and are also transducers.

Anatomically, receptors are specialized structures present at the peripheral terminations of afferent. From the receptors to the brain are pathways made up of chains of neurons. These pathways, e.g. visual pathway, pain pathway (Fig 2-5).

2-2-5 The motor system: -

Motor functions of the spinal cord and spinal reflexes: sensory information is integrated at all levels of the nervous system and causes different types of motor reflexes.

- Sensory information integrated in the spinal cord leads to simple spinal reflexes.
- Sensory information integrated in the brain stem lead to more complicated brain stem reflexes.

- Sensory information integrated in the responses and also leads to the perception of sensations.

The basic unit of integrated neural activity is the reflex arc, which consist of:

- A sense organ.
- An afferent neuron.
- One or more synapses.
- An efferent neuron.
- An effectors.

2-2-6 The afferent neuron:

Afferent impulses entering the spinal cord via the posterior root are conducted to two separate destinations. Some end on cells in the grey matter of the spinal cord of the same segment or near by segments. Afferent neurons carry impulses from receptors to the CNS. They can undergo divergence thus helping to spread a single stimulus to a wide area of the CNS, at the level of the spinal cord, afferent impulses will lead to facilitator effects.

2-2-7 Interneurons:

Interneurons are small, highly excitable cells, which may be excitatory or inhibitory. In the spinal cord they lie in all areas of the cords grey matter, making connections with one another, and many of them directly synapse with anterior motor neurons.

Interneurons are also arranged to allow convergence and divergence to occur. Their organization also allows the

important process of after-discharge. In this process an impulse in an afferent neuron is not merely relayed but causes prolonged output discharge even after the incoming impulse is over. After-discharge can last from a few milliseconds to as long as many minutes to hours.

2-2-8 The efferent neuron: -

The efferent neurons are the motor neurons present in the anterior horn of the spinal cord. Their axons leave the spinal cord through the anterior roots to innervate skeletal muscles.

Anterior motor neurons are of two types: alpha and gamma motor neurons. The alpha motor neurons are the larger cell, and they give rise to large myelinated type Alpha and gamma fibers.

Each fiber excites from three to several hundred skeletal muscle fibers. The fiber and the muscle fibers, its supplies are collectively called a motor unit.

In addition to motor neurons, the anterior horn contains large numbers of a special interneuron, Located in close association with motor neurons. This called the Renshaw cell.

2-2-9 Types of reflexes: -

There are three types of reflexes; deep reflexes, superficial reflexes and visceral reflexes.

2-2-10 The cerebral hemispheres: - Secondary and association areas: -

Close to each primary area of the cerebral cortex is a secondary area. Each secondary sensory area is concerned with analysis and interpretation of information received from the nearby primary sensory area.

On the motor side, the secondary motor area works in the collaboration with the primary motor area to integrate patterns of movements.

The secondary area consists of:

- The secondary somatosensory area.
- The secondary visual area.
- The secondary auditory area.
- The secondary motor area (i.e. premotor and supplementary motor area).

2-2-11 Motor functions of the basal ganglia and cerebellum: -

The power and precision of muscle activity is the product of normal function not only of the cerebral cortical area, but also of two other essential brain structures, namely the basal ganglia and the cerebellum. These work in close association with cerebral cortex and corticospinal tracts.

2-2-11-1 The basal ganglia: -

Anatomically the motor portions of the basal ganglia are composed of the caudate nucleus and the putamen, collectively known as the neostriatum, and of the globus pallidus. The substantia nigra, the sub thalamus, part of the thalamus and the reticular formation are functionally related to the basal ganglia.

2-2-11-2 Functions of the basal ganglia:-

The most important functions of the basal ganglia include the following:

- They help the corticospinal system in executing slow sustained movements.'
- The caudate nucleus, in association with cortical association area, plays an important role in the cognitive control of motor activity. The caudate circuit is also responsible for modifying the timing and spatial dimensions of the slow sustained movements executed by the putamen circuit.
- The basal ganglia are inhibitory to muscle tone throughout the body.
- The basal ganglia are also responsible for the initiation and regulation of the gross, subconscious, associated movements of the body.
- The globus pallidus is responsible for the posture taken by the body to perform a particular voluntary movement.

2-2-11-3 The Cerebellum: -

Lies below the occipital lobes of the cerebral hemispheres, it is connected to the brain stem by three cerebellar peduncles: superior, middle and inferior.

2-3 Pathology: -

2-3-1 Cerebral infarction: -

This results from local arrest or reduction of cerebral blood flow and consists of an area of tissue within which all the cellular elements have undergone necrosis. An infarct may range from a small discrete lesion to necrosis of a large part of the brain. It may occur in any part of the brain, but the commonest site is in distribution of the middle cerebral artery. The entire arterial territory may be affected or only part of it. The structural changes in a cerebral infarct depend upon the size of the lesion and the survival time.

A cerebral infarct may be hemorrhagic or pale. An intensely hemorrhagic infarct may resemble a haematoma, but the distinctive feature is the preservation of the intrinsic architecture of the tissue. A pale infarct less than 24 hours old may be difficult to identify macroscopically, but thereafter, the dead tissue becomes soft and swollen and there is a loss of the normal sharp definition between the grey and white matter. At this stage histological examination will show ischaemic necrosis of neurons, pallor of myelin staining and sometimes, polymorphs around the necrotic wall vessels. If the infarction is large, swelling of the necrotic tissue and edema of the surrounding brain may lead to its action as an acute expanding intra – cranial pressure. Within a few days the infarct becomes distinctly soft and the dead tissue disintegrative. Histological

examination at this stage will show macrophages filled with globules of lipid produced by the breakdown of myelin and around the dead tissue, enlarged astrocyte and early capillary proliferation, during the following weeks the dead tissue is removed and there is gliosis. The lesion ultimately becomes shrunken and cystic, and the cyst is often traversed by small blood vessels and glial fibrils. If the infarct has been hemorrhagic, some of the macrophages will contain haemosiderin and the cyst wall will appear brown, Shrinkage of an infarcted area in the cerebral hemispheres is usually accompanied by enlargement of the adjacent lateral ventricle.

A consequence of infarction is wallerian degeneration of the nerve fibers that have been interrupted.

Thus, if the infarct involves the internal capsule, there is progressive degeneration and shrinkage of the corresponding pyramidal tract in the brain stem and spinal cord.

2-3-1-1 Pathogenesis of cerebral infarction: -

Embolism, cerebral emboli arise from vegetation of infective endocarditis, from mural thrombus in patient with arrhythmias a myocardial infarct, and from non-bacterial thrombotic endocarditis in association with cachexia of advanced chronic disease. Brain damage due to embolism may also complicate open-heart surgery and more recently coronary artery surgery using cardiopulmonary by pass.

Thrombus formation from ulcerated atheromatous lesions in the aorta and in the neck arteries is also another source of embolism.

2-3-1-2 Thrombus and atheroma:

In addition to the intracranial arteries, the internal carotid and vertebral arteries in the neck are often affected. Atheroma of the cerebral arteries is usually associated with atheromatous in the other parts of the body including the arteries to the limbs.

Atheroma causing stenosis does not necessarily lead to cerebral infarction because at normal blood pressure the internal cross-sectional area of an artery must be reduced by up to 90% before blood flow is impaired. In many cases, however, cerebral infarction results from a combination of systemic circulatory insufficiency and stenosis of the cervical and/or cerebral arteries by atheroma. Infarction may also result from occlusion of arteries within the skull or in the neck. The commonest intracranial site of thrombotic occlusion is the middle cerebral artery. Atheromatous narrowing or occlusion may occur in any part of the carotid or vertebral arteries, but the infarction results only if the collateral circulation impairs or becomes inadequate. In some cases, thrombosis extends along the internal carotid artery, in the middle and anterior cerebral arteries to produce infarction of a large part of the cerebral hemisphere. When the vertebral arteries are more severely atheromatous or are

occluded ischaemic damage occurs, characteristically in the brain stem, The cerebellum and the occipital lobes.

2-3-1-3 Miscellaneous causes of cerebral infraction: -

Types of vasculitis that cause infarction include arteritis due to micro- organisms and of collagen diseases such as polyarteritis nodosa systemic lupus erythematosus and giant cell arteritis. Cerebrovascular accidents may complicate various disorders including polycythasemia rubra vera and sickle cell disease. Also may occur in pregnancy, puerperium in women taking oral contraceptives and in decreases of drug addiction due to heroin and lysergic acid diethylamide (L.A.D)

2-3-1-4 Brain damage due to cardiac arrest: -

Many patients who suffer severe diffuse brain damage as a result of a cardiac arrest die within a few days. The brain damage is usually restricted to selective neuronal necrosis rather than frank infarction. Provided the patient has survived for more than 12 hours, microscopic examination will disclose widespread and severe neuronal necrosis, because of the selective vulnerability of groups of neurons to hypoxia, the necrosis is most prominent in the amon's horn (hippocampus), in the third fifth and sixth layers of cerebral cortex (particularly within the Sulci of the posterior halves of the cerebral hemispheres), in certain basal nuclei and the purkinje cells of the cerebellum. After a few days the dead neurons disappear and

reactive changes in astrocyte, microglia and capillaries become intense. An essentially similar pattern of damage occurs in carbon monoxide, intoxication, states epilepticus and severe hypoglycemia.

2-3-1-5 Brain damage due to hypotension: -

These types of damage are concentrated in the boundary zones (water shades) between the main cerebral and cerebellar arterial territories. The infarct tends to be largest in the parietooccipital regions where the territories of the anterior, middle and posterior cerebral arteries meet.

There is variable involvement of the basal nuclei, particularly the head of the caudate nucleus and the upper third of the putamen. The hippocampi, despite their extreme vulnerability to cardiac arrest, are usually not involved.

This type of brain damage appears to be caused by a major and abrupt episode of hypotension followed by a rapid return to normal arterial pressure. Because of the precipitous, decrease in arterial pressure, auto regulation fails and the most remote from the parent arteries

i.e. The boundary zones are subjected to the greatest reduction in cerebral blood flow. Many examples of this pattern of brain damage have been described in an association with major operations under general anesthesia, myocardial infarcts, or severe hemorrhage.

Most cerebral infarctions are caused by a local vascular obstruction of some sort and either thrombotic or embolic. However, infarction also occurs when cerebral arteries are compressed against unyielding dural folds during cerebral herniations, and occasionally it occurs without obstruction when there is a severe reduction in cerebral perfusion and marked narrowing of one or more vessels.

Both embolic and compressive arterial occlusion are often only temporary and, either by relief of obstruction or the establishment of collateral flow, reflow of blood may occur into the infarcted area. When this happens, blood enters vessels rendered abnormally permeable by ischemia and, not surprisingly, leaks out converting a previously anemic infarct into a hemorrhagic one.

On gross examination, anemic infarction is not detectable with any certainty until 6 to 12 hours after their occurrence. The earliest visible change is a slight discoloration and softening of the affected area, so that the gray matter structure becomes blurred and the white matter loses normal fine-grained appearance. Within 48 to 72 hours, necrosis is well established and there is softening and disintegration of the ischemic area with pronounced circumferential swelling that may, in lesions of sufficient size, produce brain herniations. As resolution proceeds there is liquefaction, resulting in cyst formation with lesions of sufficient size. Their cysts are traversed by trabeculations of

blood vessels and surrounded by firmglial tissue. The leptomenings, when involved become thickened and opaque and may form the outer wall for the cyst.

The first histological change, seen after 6 to 12 hours, is a diffuse reduction in the staining intensity of the tissue, in chromatic stains; the first discrete change is in the nerve cell bodies, with disarrangement and disorganization of the cytoplasm and nuclear chromatin. In addition, but probably following the initial swelling of the cells, there are large numbers of red neurons suffering from ischemic damage. Special stains reveal fragmentation of axons and early disintegration of myelin sheaths, there is also loss of oligodendrocytes and astrocytes.

At about 48 hours, the blood vessels stand out prominently, and some neutrophils begin to pass through the vessel walls and into the tissue. Occasionally, this response is so intense as to simulate aseptic infarct, but usually it is replaced at 72 to 96 hours by the aggregation of macrophages around blood vessels. At this stage the macrophages are the dominant reactive cells, attaining their maximal number at about two weeks. After this they gradually disappear, but even years later they may still be found in the interslices of old infarcts. Astrocytosis becomes prominent during the second week and eventually, as the final stage in the resolution of an infarct, results in fibrillary gliosis, which encloses or replaces the necrotic region. The time

required for an infarct to resolve ranges from weeks to many months, depending on its size.

2-3-6-1 Hemorrhagic Infarcts: -

As already indicated, are usually seen in embolic or compressive infarcts. Grossly, the hemorrhage in these infarcts is confined to the cortex, the infarct white matter does not become hemorrhagic. This difference probably reflects the different caliber of vessel in the cortex and white matter. Hemorrhage does not seem to affect the processes of infarct resolution, except that, histologically, many of macrophages contain haemosiderin.

2-3-1-7 Lacunes: -

(Little lakes) are small infarcts ranging from a few millimeters to 15 mm in diameter, most commonly found in the deep portions of the brain, especially the putamen, thalamus, internal capsule, basis pontis, and hemispheric white matter. Their occurrence is particularly associated with systemic arterial hypertension and they are thought to result from the occlusion of deep arterioles, either by emboli or by hypertensive hyalinization of these arterioles. Pigmented macrophages can be found in some Lacunes. Suggesting that a hemorrhagic component may have been present or that may have been minute hemorrhages. The symptoms and signs produced by cerebral infarction depend on the location of the infarcted area and its size. Good descriptions of the many syndromes of infarction can

be found in neurology texts, but a few generalizations and some examples are helpful to understanding of the variety of their pathophysiology.

Stupor and/or coma accompanying cerebral infarction are usually a consequence of either extensive bilateral hemispheric injury or damage to the ascending reticular formation in the mid brain and pons. With hemispheric lesions, any depression in the level of consciousness implies a large infarct with associated cerebral swelling. In contrast, damage to the reticular formation can be produced by small infarctions. The distinction between these two situations is made by the other localization signs and symptoms that accompany the loss of consciousness

Convulsions at the onset of an infarct are uncommon. When they occur they are most frequently associated with embolic infarcts and cerebral hemorrhages. However, they are relatively common as a late consequence of infarct and can usually correlated with scarring of cortex.

Of all the arterial occlusion syndromes, these associated with obstruction of the internal carotid artery produce the most varied symptomatology, ranging from being symptom less to the production of acatastrophic hemiplegia. The reason for this variability lays in the ability of the anatomic connections of the circle of Willis to compensate for the occlusion. As might be expected, gradual occlusion is more likely to be symptom less than is rapid obstruction. Conversely, a sudden obstruction may

produce infarction in the territory of the middle, anterior and even sometimes-posterior cerebral artery on the affected site. Infarcts of this size are particularly likely to produce depression in the level of consciousness secondary to herniation. Atherosclerotic stenosis of the internal carotid artery without complete obstruction may also be symptomatic. The stenosis usually occurs at the bifurcation of the common carotid artery. Most of the symptoms take the form of transient ischemic attacks (TIAS). There is still controversy about the best treatment for the symptomatic carotid stenosis, surgical or medical.

Obstruction of the major intracranial arteries product stereotyped syndromes, specific to each artery.

Most of these occlusions are embolic. Involvement of the middle cerebral artery and its branches is the most frequent and provides good examples of the type of syndromes to be expected. Total infarction of the territory supplied by the middle cerebral artery will involve much of the caudate, all of the lentiform nucleus, and the intervening internal capsule, all supplied by the lenticulostriate branches. Also, the cortex of the insula and the superior and inferior lips of the sylvian fissure will be infarcted, thereby involving parts of the frontal, parietal and temporal lobes. Clinically, because of the infarction of the internal capsule, there will be a dense hemiplegia involving contralateral limbs and the face, a cortical hemisensory deficit,

and usually a visual field quadrantanopia. If the left hemisphere is infarcted, there will also be severe aphasia in 95% of cases.

The middle cerebral artery usually has two major branches, a superior division that supplies the frontal and parietal cortex, making up the upper lip of the sylvian fissure, and an inferior division that supplies the temporal lobe component of the middle cerebral territory. Obstruction of the left superior division produces a hemiparesis, worse in the face and arm (because only the cortical and not the internal capsule motor fibers are affected), a cortical hemi sensory deficit, and a confluent (Broca) type of aphasia. With a lower division infarction there will be affluent (Wernicke) type of aphasia and usually a visual field cut, but no hemiparesis or sensory defect since no motor or sensory cortex is infarcted. Infarction in smaller branches of the artery produces correspondingly smaller deficits. Infarction in other vascular territories produce equally distinctive clinical syndromes that, when decided by history and examination, usually permit identification of the affected arterial territory.

Mention must also be made of the infarctions that follow venous obstruction. Because of the great collateralization of the cerebral venous drainage, infarction only follows either occlusion of a large sinus or widespread smaller vein obstruction. It is most commonly seen in occlusion of the superior sagittal sinus, which is itself most often the consequence of the predisposition to thrombosis associated with cancer. The infarction is characteristically bilateral,

parasagittal, multiple hemorrhagic with hemorrhage in both gray and white matter.

Clinically, this infarction is often distinguished by the occurrence of particularly intractable seizures.

2-4 MRA appearance: -

MRA showed brain arteries as high signal when applied post-processing procedure MIP.



Fig (2-6) MIP

MRI brain showed cerebral infarction as high signal in T2w and low signal in T1w FLAIR.

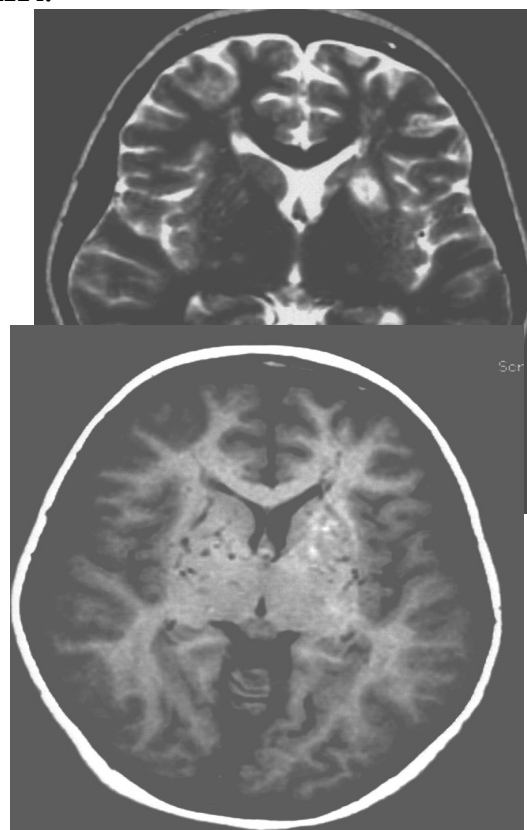


Fig (2-8) T₁w

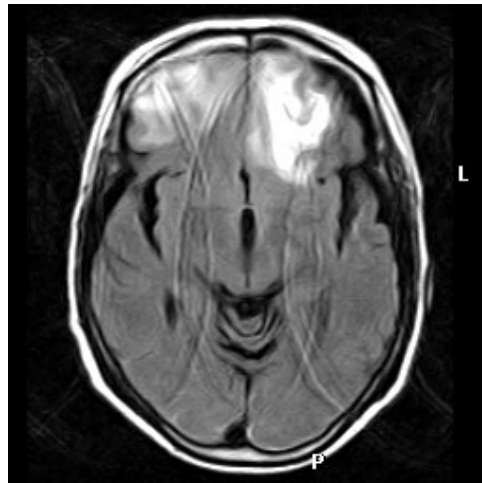


Fig (2-9) FLAIR

2-5 Equipment: -

2-5-1 MRI components: -

MRI requires magnet, which magnet the major components of the MRI system in the scanning room. This magnet must be large enough to surround the patient, antennas that are required for radio-wave transmission and reception, a computer necessary for analyzing the MRI signal, and an operators console that used to control the computer. Fig ().

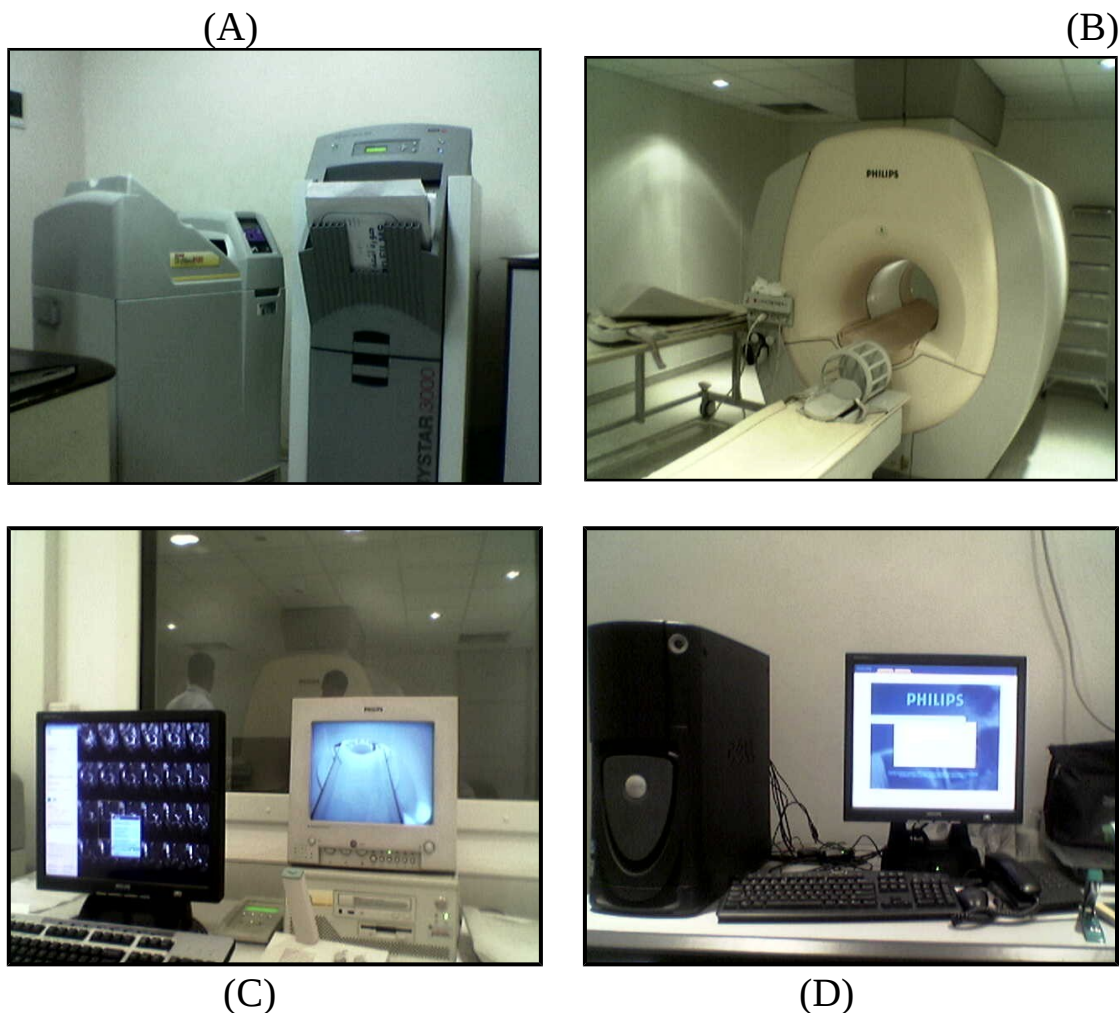


Fig (2-10): (A): printer, (B): magnet room, (C) console, (D) work station

2-5-2 Advantages and disadvantages of MRI:

-

2-5-2-1 Advantages of MRI:

- Non ionizing radiation.
- No known biological hazard.
- Multi-planar imaging: images can obtained in all planes, namely coronal, sagittal, axial or any oblique plane.
- High soft tissue resolution.
- Blood flow imaging.
- Non-invasive imaging technique.

2-5-2-2 Disadvantages of MRI:-

- High cost.
- Claustrophobia.
- Longer imaging time.
- Cortical bone and calcific lesions are poorly visualized.
- Patients with pacemakers, surgical clips, autologic and ocular implants are unable to undergo an MRI examination.

2-5-2-3 Advantages and disadvantages of MRA: -

Advantages of MRA:

- MRA technique combine with MRI brain, sot it show if there are any brain abnormalities, such as cyst, tumours, heamorrahe, multiple sclerosis (MS) and brain atrophy.
- In MRA can rotate the picture in different direction to show any abnormality clearly.
- No need for patient preparation.

- No need for contrast media.
- It takes short time compared with conventional angiography.

Disadvantages of MRA: -

- Non-available in any place and required high strength field (1-1.5Tesla).
- Sensitive to patient movement.
- Need qualified operator and co-operative patient.

Chapter (3)

Data Presentation

3-1 Patients: -

Random sample of 30 patients who were clinically has cerebral infarction. Undergo the MR examination to detect the infarction. Their age ranging between (45- 62) years. (11) Patients are females and (19) are males.

3-2 Machine used: -

Philips (Intera) with the following specification: field strength (1.5tesla) the frequency is approximately (63) MHz. Type of magnet used is super conductive magnet; type of coil used is head coil.

3-3 Technique: -

3-3-1 General brain protocols:

- Survey (Scout): -

T2TSE axial.

T1 TSE axial.

T2 Flair axial.

T2 TSE coronal.

T1 TSE sagittal.

SEQ	Plane	TR	TE	Flip angle	Field of view	Slice thickness	Pulse sequence	Scan time
-----	-------	----	----	------------	---------------	-----------------	----------------	-----------

						and inter slice gap		
1	Sagittal T1W	594 ms	15 ms	68 ⁰	235 mm	5/1mm	Fats spin	3.44 sec
2	Axial T1W	594 ms	15 ms	68 ⁰	230 mm	5/1 mm	Fats spin	3.44 sec
3	Axial T2W	4481 ms	100 ms	90 ⁰	230 mm	5/1 mm	Fats spin	4.56 sec
4	Axial FLAIR	11000 ms	140 ms	90 ⁰	230 mm	5/1 mm	Fats spin	5.08 sec
5	Coronal T2W	4481 ms	100 ms	90 ⁰	230 mm	5/1 mm	Fats spin	4.56 sec

3-3-2 MRA protocols: -

3-3-2-1 Survey (Scout): -

Show the brain anatomy with three localizers axial, coronal and sagittal, so as adjusted the area.

3-3-2-2 TOF (inflow): -

Inflow MRA is based on enhancement of flowing blood and suppression of stationary tissue.

Image contrast depends on:

TR.

Flip angle.

Blood flow velocity.

Slice thickness.

- 2D inflow: -

Large Flip angle.

Short TR → shorter scan time.

Low through – plane resolution.

- 3 D inflow: -

Small flip angle.

Large TR to prevent.

Saturation → longer scan time → more motion sensitive.

Higher through – plane resolution — ~~better~~ MIP image quality.

3-3-2-3 PC (Phase Contrast):-

Phase contrast angiography is based on the phase of the MR signal.

Contrast between vessels and background is obtained by the difference of the phase between flowing and static spins.

There is a relation between flow velocity and signal intensity, if flow velocity = v_{enc} → highest signal intensity.

2scans are made; a flow sensitive scan and a flow compensated scan, and are automatically subtracted resulting in the PCA – phase image.

Fov	150	Slices	100
Rfov	100 %	Thk	0.5 /-0.2
Res	136/256	TR	5.5
Flip angle	10	NSA	1

3-3-2-3-1 Advantages and disadvantages of PC:

Advantages: -

- Complete background suppression, no signal from short T1 tissues like fat and methaemoglobin.
- Visualize fast and slow flow.
- In-plane flow can be measured.
- Flow sensitivity in 3 directions.
- FFE/M and PCA/M images allow good visualization of the vessels in relation to surrounding soft tissue.

Disadvantages: -

- Long acquisition time, each for flow direction two measurements → motion sensitive.
- V enc (Velocity encoding) dependant.
- Sensitive to disturbed flow.

MIP: -

Post processing: after MRA procedure is done the operator can be made MIP, which is subtracted all the tissues of the brain except the pixel with high signal (arteries).

3-4 Films evaluation: -

- All scan film by one radiologist and one technologist to explain the pathological changes and role of MRA incase cerebral infarction.
- MRA compare with MRI brain and CT scan if find to showing the arteries of circle of willis.
- In MRA can be seen all the arteries of circle of willis, therefore determine the affected area and site of infarction.

Chapter (4)

Result and data analysis

4-1 Results: -

CNS consists mainly of peripheral nervous system and central nervous system. The visualization of these structures and their pathological changes obtain by MR technique.

MRA examination for 30 patients (male and female) was done, evaluated and analyzed as follow:

- Table (4-1): shows the range of patient's age.
- Table (4-2): shows the sex of patients.
- Table (4-3): pathological appearance: shows obstructed in cerebral arteries with 9 patients appeared as complete obstruction, 13 patients appeared as partial obstruction and 8 patients appeared as normal.

MRA is significantly accurate in diagnosis and detecting level of infarction, cerebral hemorrhage, aneurism, evaluation of potency of the blood vessels and detection of A-V malformation.

Table (4.1)

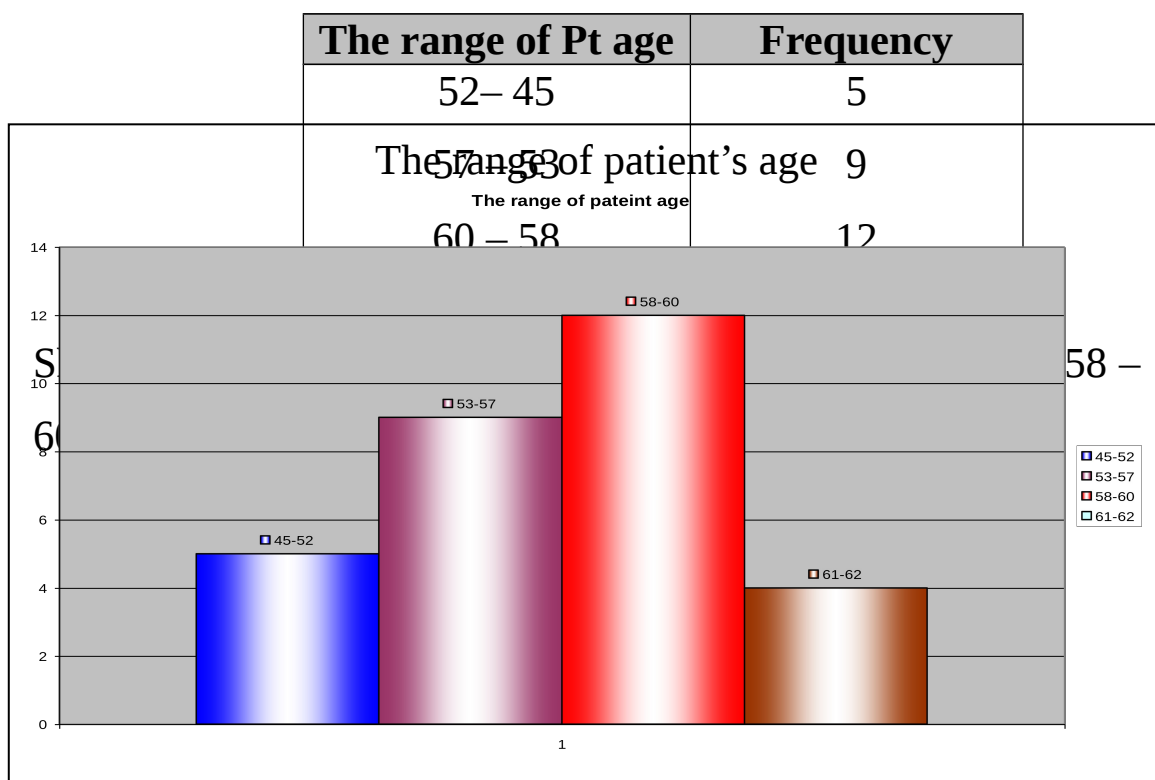


Fig () presentation of data in table (4-1)

Table (4.2)

Total cases	No of males	Percentage	No of Female	Percentage
30	19	% 63	11	% 37

Shows the percentage of males to females, males are more than females.

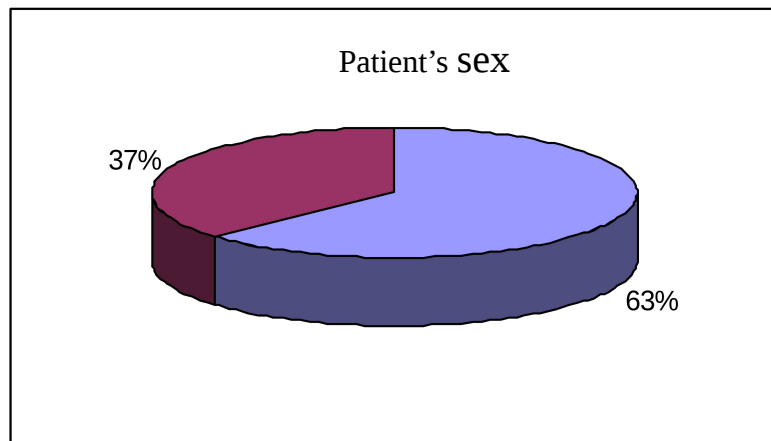


Fig () presentation of data in table (4-2)

Table (4-3)

Pathological	Presence	Percentage	Absence	Percentage
Cerebral infarction	22	% 73	8	% 27

Shows the cerebral infarction in 22 patients, absence in 8 patients.

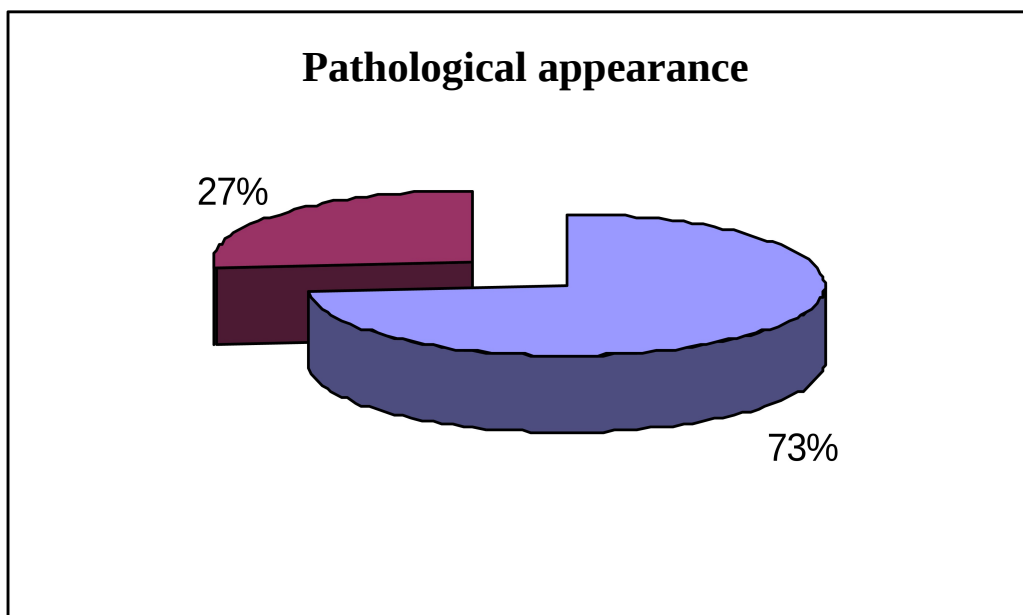


Fig () presentation of data in table (4-3)

Chapter (5)

5-1 Discussion: -

The main objective of this study is to determine the role of MR to detect the cerebral infarction, measure the sensitivity of MR in diagnosis of cerebral infarction to explain the suitable technique that used to demonstrate the cerebral infarction. And to show different appearance of abnormality causes cerebral infarction.

The study investigate experimentally 30 patients with suggestive cerebral infarction in the period from April to October 2005, a random sample is used.

The data analyzed through statistical method that include frequency table and percentage. The main finding of the study can be sum marginal in the following:

1. 22 patients (73 %) out of 30 show cerebral infarction clearly .
2. 8 patients (27 %) show normal brain .

In this study obtained that (MRA) is very perfect technique to detect cerebral infarction.

According to hypothesis of the study the results we gained is agree.

Finally, can be said that it is possible to introduce other studies in the future.

5-2 Conclusion: -

MRA technique improves the detection of cerebral infarction. Its application improves MRI diagnostic efficiency and become a method of choice for examination of cerebral infarction.

This modern technique (MRA) has more benefit in diagnosing the blood vessel potency and result is a high quality image with high technical proprieties.

Applying MRA technique will improve efficiency of cerebral blood flow diagnostic and provides excellent details regarding the anatomy, anatomical variants, pathology and determine accurate the level of infarction, which is important factor of disease management.

Lastly, we can say that the MRA technique is very accurate due to ability of demonstrating the level of infarction and other pathological changes.

5-3 Recommendations: -

- MRA is recommended for small cerebral infarct (less than 1 cm).
- For normal brain CT for those whom suspect to be affected by cerebral infarction it is better to re-examined them by MR.
- The radiologists and technologists should be well trained about this modern technique (MRA) so as to show the importance and efficiency of it.
- Further studies could be made to compare between MRA technique and other radiological modalities to explain which is better.
- Effective Co-operation between technologist, radiologist, and neurologist to make full use of available MRA facilities, give early detection of cerebral infarction and quick management.