BRAIN MRI & CT FINDINGS IN PATIENTS PRESENTING WITH SEIZURES

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To my brother Abdel Azeem who had helped me a lot, my parents, my family, and to my teachers.

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ABBREVIATIONS

EEG  Electro Encephalo Gram
CT    Computed Tomography
MRI   Magnetic Resonance Imaging
ABSTRACT

It has been estimated that about 7% to 8% of the population experiences at least one epileptic seizures during a life time. This study was planned with the flowing Objectives:

1/To identify the cause of convulsions(if any) in the sample selected as shown on imaging.

2/To correlate the imaging findings to clinical findings.

3/To try to identify the clinical features which help in choosing cases needing imaging in the future.

Patients ,material and methods: 142 consecutive outpatients presenting with more than one seizure and who had cross-sectional brain imaging were selected. Their images reviewed and results analysed.

Results: 114 patients had generalized seizures , and 28 had focal seizures. 89 patients had computed (CT) scan only, 48 had magnetic resonance imaging (MRI) , and 5 had both CT and MRI scans . 70 of 142 scans (49.7%) were abnormal. Abnormalities were infarction (n=30), tumours (n=10), malformations of cortical development (n=6),non-specific lesions
(n=6), vascular abnormalities (n=4), infection (n=5), hippocampal sclerosis (n=2). The percentage of detected abnormalities was 68% in focal seizures, 37% in generalised seizure patients.

**Conclusions:**

1/ Neuroimaging is important for patients presenting with seizures to identify underlying abnormalities.

2/ Patients with focal seizures had a higher incidence of imaging abnormalities.

3/ CT should be done first and MRI reserved for patients who had normal CT or a CT that was inconclusive.
ملخص الظروحة

لم يتضح في النص الذي تم إخراجه، ولكن يمكن أن يشير إلى أن بعض الأشخاص قد يكونون مرضى، حيث ذُكرت أن هناك أربعة وثمانية وعشرة حالة لم تتجاوزها، وتعرف على أن هناك تراجعاً في حالة بعض الأشخاص. كما ذُكر أن هناك تحسناً في حالة البعض الآخر. في النهاية، يمكن أن يكون هناك تحسناً في حالة البعض الآخر.
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1. INTRODUCTION AND LITERATURE REVIEW

1.1 Seizures:

1.1.1 Historical background

In 1873, Jackson presented his classic definition of epileptic seizures, that is, "occasional sudden, excessive, rapid and local discharges of gray matter,"\textsuperscript{1} which is generally held to be the origination of the modern view of epilepsy. Subsequent developments led to the view that localization of the site of seizure origin was possible based on clinical symptoms. Using these concepts, in 1879 the neurosurgeon Macewen correctly localized and removed a frontal meningioma; subsequently, the patient became seizure free. This central principle of seizure surgery remains largely unchanged that is, if the seizure focus is accurately localized, surgical removal of the focus will alleviate the occurrence of seizures.

Major technologic developments, including the advent of electroencephalography (EEG) and neuroimaging techniques, (computed tomography (CT) and magnetic resonance (MR) ), have revolutionized the localization of seizure foci and continue to develop. The technique of EEG, first published by Berger,\textsuperscript{2} provided a means
of localization of seizure foci originating in less eloquent areas of the cortex that did not present with characteristic and localizing symptoms. In 1951, Bailey and Gibbs reported on a series of patients who underwent temporal lobe resection based only on EEG criteria, thus helping to establish EEG as the primary method of seizure localization. Magnetic resonance imaging (MRI) techniques were first applied to human studies in the late 1970s, providing structural information with greater sensitivity than that offered by computed tomography (CT), as well as functional and biochemical assessment capabilities, all of which continue to evolve.
1.1.2 Epileptogenesis

Epilepsy was once thought to be a result of possession by evil spirits, with treatment involving religion and the occult. It is now believed that the basic process of epileptogenesis is the abnormal discharge of neurons from normal brain tissue characteristics that may have been modified, exaggerated, or released from normal physiologic controls. Seizures are generally a symptom of a focal or generalized brain abnormality primarily involving the gray matter. In intractable cases, the clinical problem requires a precise definition of normal and abnormal brain structure and function if surgical treatment of the disorder is to be considered. The goals of imaging are to determine whether a structural abnormality of the brain exists, to define the location and extent of the region (or regions) responsible for the seizure generation, and to identify how functional events relate to the underlying structural abnormality, as well as to identify important functional areas of cortex (speech, memory, movement). Allot these must be considered in neurosurgical decision-making.

1.1.3 Classifications

The classification of epileptic seizures by the International League Against Epilepsy was last revised in 1989 (Table 1). The
classification of seizure disorders is important because causative
diagnosis, appropriate treatment, and accurate prognostication of
seizure disorders are dependent on the correct identification of
seizures and epilepsy. There are two main seizure types: partial
seizures and generalized seizures. Partial (formerly referred to as
focal) seizures show either clinical or EEG evidence of onset from a
localized area within the cerebral hemisphere. The nature of the signs
and symptoms in most cases of partial seizures indicates the region
of the brain involved by the epileptic process. Partial seizures are
designated as simple or complex. Complex partial seizures are
associated with loss of consciousness. In simple seizures, the
epileptic process is usually confined to neocortical structures with
sparing of the limbic system and brain stem. Most simple seizures are
less disabling than those associated with loss of consciousness. The
signs and symptoms in most cases of partial seizures indicate the
region of the brain involved by the epileptic process. Partial seizures
can progress from simple partial to complex partial, indicating
propagation into the limbic system and resulting in impairment of
consciousness. Partial seizures can spread and develop into
secondarily generalized seizures. Primary generalized seizures
originate simultaneously from both cerebral hemispheres, and clinical manifestations involve both sides of the body. Primary generalized seizures first occur during childhood, are more likely to be associated with a family history of seizure disorders, and are less likely to be associated with focal cerebral lesions. A few seizure types remain unclassified because the underlying mechanism of their origin or propagation is unknown.\textsuperscript{5}


table 1 outline of the international classification of epileptic seizures

I. Partial Seizures (Seizures with Focal Onset)

Simple partial seizure (consciousness not impaired)

With motor signs

With somatosensory or special-sensory symptoms

With autonomic symptoms or signs

With psychic symptoms (disturbance of higher cerebral functions).

Complex partial seizure (consciousness impaired)
1. Starting as simple partial seizures
   Without automatisms
   With automatisms

2. With impairment of consciousness at onset
   Without automatisms (impairment of consciousness only)
   With automatisms Partial seizures evolving into secondarily generalized seizures

II. Generalized Seizures

   Absence seizures and atypical absence seizures (may have the following components: mild clonic, atonic, tonic, or autonomic activities, or automatic behavior)
   
   Myoclonic seizures
   Clonic seizures
   Tonic seizures
   Tonic-clonic seizures
   Atonic seizures

III. Unclassified Epileptic Seizures

   Seizures may also be classified according to the degree of association with underlying factors. Idiopathic (or primary) epilepsies
and syndromes have no underlying cause other than a possible hereditary predisposition. When underlying causes are present, epilepsies and syndromes are designated symptomatic (secondary). Cryptogenic epilepsies and syndromes are presumed to be symptomatic, but the suspected underlying cause is not identified. Seizures are considered to be provoked or acute symptomatic when one or more precipitating factors are present. Provoking factors can be any temporary or permanent, acute disturbance that disrupts the normal electrical activity of the brain, such as stroke, traumatic head injury, intracranial infection, and medication. Seizures that occur more than 1 to 2 weeks after the provoking factor are considered remote symptomatic unprovoked seizures. The provoking factors in this instance are referred to as remote symptomatic or predisposing factors. When no provoking or predisposing factors are identified, seizures are designated idiopathic unprovoked seizures. Remote symptomatic unprovoked seizures have a higher risk of recurrence than idiopathic unprovoked seizures.

Epilepsy is defined as a chronic condition with a genetic or acquired predisposition to recurrent epileptic seizures. An epileptic syndrome is an "epileptic disorder characterized by a cluster of signs
and symptoms customarily occurring together. The signs and symptoms that characterize an epileptic syndrome are derived from the clinical history, the seizure-type classification, the physical examination, and the EEG and imaging findings. Epileptic syndromes have distinctive profiles that facilitate their diagnosis or are age-specific or age-related.

1.1.4 Epidemiology

Epilepsy is a common disorder, affecting 0.5% to 1.0% of the U.S. population at any given time, with a prevalence of 5 to 8 per 1000, with an increased prevalence from childhood to adolescence and a slight increase after age 70 years. The incidence of epileptic seizures is approximately 30 to 50 per 100,000 person-years. The rates are high during the first year of life, declining until middle age, and then rising sharply in later years. It has been estimated that about 7% to 8% of the population experiences at least one epileptic seizure during a lifetime.

Partial (localized, focal) epilepsy is the most frequent type, with an incidence of 20 per 100,000 until age 65 years, and 80 per 100,000 thereafter. Partial seizures account for 40% to 60% of all newly diagnosed cases. Between 60% and 70% of patients with
newly diagnosed partial complex epilepsy can be expected to achieve complete seizure control, and most may be able to be managed without antiepileptic medication. Approximately 5% to 10% will not have improved seizure control or will worsen. Onset at less than 2 years of age, a large number of seizures, the presence of secondarily generalized seizures, and a history of status epilepticus or febrile seizures are associated with a poor outcome. Intractable epilepsy is very disabling with formidable psychosocial consequences as well as an increased incidence of early death, sudden death, and significant body injuries.\textsuperscript{9}

1.1.5 Imaging

Certain types of seizure disorders are likely to be associated with structural brain lesions, including tumors, infection, infarction, traumatic brain injury, vascular malformations, developmental abnormalities, and seizure-associated brain pathology,\textsuperscript{10} whereas others are not (Table 2). Hence, knowledge of seizure types helps to determine whether neuroimaging is clinically indicated and what type of study is appropriate.

Although the imaging evaluation of epilepsy was greatly advanced by the clinical introduction of CT in the early 1970s,
because of MRI’s superior soft tissue contrast, multiplanar imaging capability, and lack of beam-hardening artifacts, virtually all the substrates of epilepsy are visualized with greater sensitivity and accuracy by MRI. As a result, MRI has become the modality of choice for imaging in epilepsy.

MRI plays several important roles in the clinical management of patients with epilepsy, the most important being to localize and identify the structural substrate of partial seizures. Although patients' seizures are usually classified on the basis of clinical and EEG findings, precise classification may at times be uncertain. At times, secondary generalized partial-onset seizures may be misinterpreted as primarily generalized. The identification of an epileptogenic lesion on MRI in cases such as these could clarify seizure classification as focal in onset.

TABLE II Outline of the International Classification of Epilepsies and Epileptic Syndromes

1. Localization-Related (Focal, Local, Partial) Epilepsies and Syndromes

1.1 Idiopathic (with age-related onset)
Benign childhood epilepsy with centrotemporal spike
Childhood epilepsy with occipital paroxysms
Primary reading epilepsy

1.2 Symptomatic

Chronic progressive epilepsia partialis continua of childhood (Kojewnikows syndrome)

Syndromes characterized by seizures with specific modes of precipitation (e.g., reflex epilepsy)

Temporal lobe epilepsies
Frontal lobe epilepsies
Parietal lobe epilepsies
Occipital lobe epilepsies

1.3 Cryptogenic

2. Generalized Epilepsies and Syndromes

2.1 Idiopathic (with age-related onset)

Benign neonatal familial convulsions
Benign neonatal convulsions
Benign myoclonic epilepsy in infancy
Childhood absence epilepsy (pyknolepsy)
Juvenile absence epilepsy
Juvenile myoclonic epilepsy (impulsive petit mal)

Epilepsy with grand mal seizures on awakening

Other generalized idiopathic epilepsies not defined above

Epilepsies with seizures precipitated by specific modes of activation

2.2 Cryptogenic or symptomatic

West syndrome (infantile spasms, Blitz-Nick-Salaam Krampe)

Lennox-Gastaut syndrome

Epilepsy with myoclonic-estatic seizures

Epilepsy with myoclonic absences

2.3 Symptomatic

2.3.1 Nonspecific etiology

Early myoclonic encephalopathy

Early infantile epileptic encephalopathy with suppression-burst

Other symptomatic generalized epilepsies not defined above

2.3.2 Specific syndromes

Epileptic seizures complicating disease states

3. Epilepsies and Syndromes Undetermined Whether Focal or Generalized

3.1 With both generalized and focal seizures
Neonatal seizures

Severe myoclonic epilepsy in infancy

Epilepsy with continuous spike-waves during slow wave sleep

Acquired epileptic aphasia (Laundau-Kleffner syndrome)

Other undetermined epilepsies not defined above

3.2 Without unequivocal generalized or focal features

4. Special Syndromes

4.1 Situation-related seizures

   Febrile convulsions

   Isolated seizures or isolated status epilepticus

   Seizures occurring only with acute metabolic or toxic event

As the classification of epilepsies shifts from electroclinical phenomena toward an emphasis on the structural substrates that produce the observed electroclinical abnormalities, the role of MRI has become more central in conceptually defining the epilepsies. MRI is also an essential tool in surgical planning and is used to define the volume of tissue targeted for resection, identify the surgical approach,
and assess the relationship of the lesion to functionally eloquent areas of the brain.

The sensitivity of MRI techniques to the structural substrates responsible for epilepsy, based on the recent imaging literature, is estimated at 100% for neoplasm, 90% to 100% for neuronal migration disorder, 100% for vascular malformation, 90% to 95% for mesial temporal sclerosis, and 80% to 90% for brain injury due to trauma, infarction, or infection. Wherever routine evaluation techniques on the range of scanner field strengths may be sufficient for determination of mass lesions, optimized protocols for scans obtained on high-field (1.5-Tesla) scanners may be necessary for evaluation of partial complex epilepsy, requiring scrutiny of the hippocampus and temporal lobe for atrophy and subtle signal alteration, as well as detection of certain structural abnormalities such as cortical dysplasias, hamartomas, and other developmental abnormalities.

With the impending widespread clinical availability of high-performance MRI systems, a comprehensive MR examination may be of even greater value in epilepsy, with functional techniques providing additional information, adding corroborative information, and improving overall accuracy.
1.1.6 Pathology

The spectrum of pathologic substrates associated with seizures include:

1. Developmental disorders
   • Neuronal migration disorders
   • Hamartomas
   • Vascular malformations

2. Neoplasms
   • Gliomas
   • Mixed neuronoglial tumors
   • Dysembryoplastic neuroepithelial tumor
   • Others

3. Mesial temporal sclerosis

4. Brain injury
   • Infections (bacterial, viral, fungal, parasitic diseases)
   • Immune-mediated disorders (Rasmussens syndrome)
   • Cerebrovascular diseases, including stroke, porencephaly, and ulegyria
     • Trauma

Developmental Disorders
Neuronal migration disorders include the following:

1. Tuberous sclerosis
2. Focal cortical dysplasias
3. Polymicrogyria
4. Schizencephaly
5. Heterotopias
6. Lissencephaly (agyria-pachygyria)
7. Hemimegalencephaly
8. Microdysgenesis

Congenital disorders of brain development characterized by disturbances of neuronal migration and organization are commonly associated with epilepsy.\(^{13}\)

1.1.7 Tuberous Sclerosis

Tuberous sclerosis is an autosomal dominant inherited disease, most commonly seen in children. The most common clinical presentation is seizure, occurring in more than 80% of cases. The classic clinical presentation includes mental retardation and sebaceous adenomas of the face. Brain lesions in this disorder include hamartomas (tubers) with or without calcification involving the cortical and subcortical white matter, the deep white matter, and the
subependymal region; and giant cell astrocytoma. Cortical tubers have been classified as both tumors and disorders of neuronal migration by various authors and would seem most appropriately placed in an intermediate category. The cortical tuber is most likely responsible for seizures, as resection of a cortical tuber sometime eliminates chronic seizures.\textsuperscript{12} The cortical hamartoms are predominantly seen in the frontal and parietal lobes and are typically isointense or hypointense to gray matter on MR T1-weighted images and hyperintense on T2 weighted images. The tubers are located immediately beneath the cortical gray mantle, and typically, the involved gyrus is expanded. The lesions may be most conspicuous on fluid attenuated inversion recovery (FLAIR techniques). A raylike projection of increased signal from the base of the tuber toward the ventricular surface is frequently observed. Subependymal hamartomas, most commonly within the head of the caudate at the foramen of Monro, may degenerate into giant cell astrocytomas in less than 10% of patients.\textsuperscript{14}

1.1.8 Focal Cortical Dysplasias

Focal cortical dysplasia is a migrational disorder encountered in partial complex seizures, with the seizure usually occurring in the first
or second decade life. This condition is characterized by lesions consist of large anomalous neurons in the cortex. Histologically, this dysplastic abnormality is similar to the cortical abnormality described in tuberous sclerosis. These lesions are manifest grossly and, on imaging by thickened cortex (>4 mm) and blurring of the interface between gray and white matter. These lesions share a similar histology with the cortical lesions of tuberous sclerosis, and differentiation of tuberous sclerosis from this condition is difficult histologically, clinically, or by imaging criteria.15

1.1.9 Polymicrogyria

Polymicrogyria refers to an area of brain with small abnormal gyri. It is characterized by abnormal, cobblestone-like secondary gyral formation with small indentations over the gyral crowns, lacking intervening actual leptomeningeal spaces or true sulci. Macroscopically, the microgyric nature of the abnormality may be evident, or the outer molecular layers of adjacent microgyri may be fused, creating an erroneous impression of a single large gyrus (ie., macrogyria or pachygyria). The most common location of involvement is the perisylvian region. Polymicrogyria may present as deep infolding, or cleft58. Patients have a seizure disorder and some
degree of developmental delay. The severity of clinical symptoms is related to the amount and location of brain involvement. A syndrome consisting of bilateral perisylvian polymicrogyria, pseudobulbar palsy, secondarily generalized epilepsy, and mental retardation has been described. 16

**1.1.10 Schizencephaly**

Schizencephaly denotes abnormal gray matter-lined clefts that extend through the entire cerebral hemisphere from the lateral ventricle (ependymal surface to the cerebral cortex (pial surface). The clefts are typically lined by dysplastic polymicrogyric cortex. The clefts can be unilateral or bilateral and are most common in the sylvian or perisylvian region. The clefts have been divided into (1) type I in which the lips of the cleft are in contact with each other and (2) type II in which the defect is open. The ventricular extension of a closed-lip cleft may be demarcated by a dimple along the ventricular surface. Symptoms relate to the degree of brain involvement and vary from severe impairment to essential normal development and intellectual ability. Patients with bilateral clefts are always severely retarded and present with seizures at very early age. 17
1.1.11 Heterotopias

Along with focal cortical dysplasia, nodular heterotopia is the most common developmental disorder found in epilepsy. Heterotopia is the name given to focal collections of gray matter located in an abnormal position. Nodular heterotopias can be subependymal and subcortical and present as masses with MR signal characteristics identical to those of gray matter. Subependymal heterotopias appear as nodular rests of gray matter that project into the ventricle and may present as one or a few isolated nodules or as a conglomerate mass of gray matter lining the ventricular surface. These lesions are most commonly bilateral. In unilateral involvement, the relationship of subependymal heterotopias to seizure production is questionable, and patients are often developmentally and intellectually normal. Nodular subcortical heterotopias are conglomerate masses of gray matter that may be purely subcortical in location or may fan outward from the ependymal surface into the white matter. The overlying cortex is abnormal with polymicrogyria or pachygyria, or both. A raylike projection of increased T2 signal may be identified projecting from the underlying ependymal surface to the base of the subcortical heterotopia. In addition to seizures, many patients have neurologic
deficits, developmental delay, and intellectual impairment. Features of heterotopias useful in distinguishing them from neoplasm include signal intensity always identical to gray matter, no enhancement, lack of mass effect, and absence of edema.

Laminar heterotopia is a relatively uncommon developmental abnormality that presents as a thick, circumferential laminar zone of heterotopic gray matter throughout both hemispheres situated between the subcortical white matter and the deep periventricular white matter. The term band heterotopia or double cortex syndrome has been used to describe this disorder. The overlying cortical mantle is abnormal, commonly thicker than normal, and usually associated with areas of pachygyria. This abnormality is usually fatal in the developing male, and therefore, the anomaly is found nearly exclusively in women. Patients almost always present with seizure disorders. The severity of disability is proportional to the degree of brain involvement, ranging from normal development and intellectual ability to severe developmental delay and mental retardation.

1.1.12 Lissencephaly (Agyria-Pachygyria)

These conditions are the most severe of the neuronal migration disorders and represent a spectrum of gyral abnormalities. At the
severe end of the spectrum are smooth brains completely devoid of sulci and gyri, also known as agyria. The term pachgyria refers to broad, flat gyri. Patients with this malformation are microcephalic and severely retarded, with developmental delay and hypotonia.\textsuperscript{19} Medically refractory seizures begin within the first few months of life. The clinical course is uniformly poor, with most patients dying within the first 2 years of life.

Lissencephaly is a family of disorders of cortical migration that may have different genetic bases. Five distinct subcategories have been described,\textsuperscript{20} types I and II are the most well recognized. Type I lissencephaly is also referred to as classic lissencephaly. Many of these patients have abnormalities of chromosome 17pl3. The category of type I lissencephaly contains subsets of patients with distinct clinical syndromes, the most well known being the Miller-Dieker syndrome. The abnormal cortex is composed of the following four layers, from the outside in:

(1) a molecular layer, (2) an outer cellular layer (true cortex), (3) a cell-sparse zone, and (4) a thick inner layer of neurons whose outward migration was arrested prematurely. On imaging studies, type I lissencephaly shows a markedly thickened cortex with broad,
flat gyri and a few shallow sulci, diminished white matter, and shallow, vertically oriented sylvian fissures that produce a figure-of-eight appearance on axial images\textsuperscript{21}.

Type II lissencephaly consists of a spectrum of abnormalities. The most common syndrome in type II lissencephaly is the Walker-Warburg syndrome. Similar to those with type I, type II patients are severely impaired with developmental delay, severe retardation, hypotonia, and seizures. As with type I, the malformation is due to a global arrest of radial neuronal migration. Type II lissencephaly has a characteristic appearance with finger-like projections from the inner margin of the thickened cortex into the underlying white matter.

Lissencephaly, pachygyria, and laminar heterotopia may share a common pathologic mechanism of global arrest of radial neuronal migration. Therefore, a spectrum of severity exists, with lissencephaly at the severe end and laminar heterotopia at the mild end.\textsuperscript{22}

1.1.13 Unilateral Hemimegalencephaly

In hemimegalencephaly, there is hamartomatous overgrowth of all or part of the cerebral hemisphere. The lateral ventricle is enlarged, the volume of white matter is increased, and a variety of associated migrational abnormalities are usually present, including
pachygyria, polymicrogyria, and heterotopias. White matter gliosis may be present. The affected area of the brain is not functional. The extent of involvement varies from a single lobe to the entire hemisphere. Patients present with intractable seizures that typically begin within the first few months of life, as well as hemiplegia and developmental delay. Ipsilateral somatic hypertrophy may also be present.

1.1.14 Microdysgenesis

Microdysgenesis describes subtle, microscopic alterations in cortical architecture. These have been described in patients with primary generalized epilepsy, particularly Lennox-Gastaut syndrome and Wests syndrome, and in patients with focal-onset seizures. The causal relationship between microdysgenesis and epilepsy is controversial because the same histologic features are seen in pathologic specimens from nonepileptic brains. Microdysgenesis has not yet been described by MRI.

1.1.15 Vascular Malformations

The spectrum of cerebral vascular malformations includes cavernous hemangioma, capillary telangiectasia, venous angioma, and arteriovenous malformation (AVM). Seizure is the principal
clinical presentation in vascular malformations, seen in 24% to 69% of AVMs and 34% to 51% of cavernous malformations. The majority of venous angiomas and capillary telangiectasias are clinically silent.\textsuperscript{25}

The hallmark of an AVM is the presence of direct arteriovenous shunts without an intervening capillary network. The most common presenting symptom is hemorrhage, although seizures are common. Surgical decision-making is primarily based on minimizing the risk of hemorrhage.

Occult vascular malformations are those not visualized on conventional arteriography. These consist of cavernous hemangioma, capillary telangiectasia, and thrombosed AVM, all of which have a similar appearance on MRI and CT and cannot be mutually distinguished. MRI is extremely sensitive to the presence of occult malformations.\textsuperscript{26} Cavernous hemangiomas are composed of large abnormal vascular spaces. The central nidus has a heterogeneous appearance with increased signal on T1- and T2-weighted images. A gliotic hemosiderin-laden rim surrounds the nidus, creating a hypointense lesion periphery on MRI.

The chief pathologic distinction between cavernous hemangioma and capillary telangiectasia is the absence of normal neuronal tissue
interspersed in the nidus of the cavernous hemangioma. Normal neuronal tissue is present between the abnormal vascular channels in capillary telangiectasia. When symptomatic, the most common clinical presentation of a cavernous hemangioma is chronic seizures. Cavernous malformations are the vascular malformations most commonly considered for surgical resection in intractable seizures.

Venous angiomas consist of a "caput" of dilated draining veins that converge on a large draining vein. Typically, the nidus is located along an ependymal surface. Venous angiomas are usually considered to represent normal variants and are typically not associated with seizures.

1.1.16 Sturge-Weber Disease

Sturge-Weber disease, a congenital phakomatosis syndrome, also known as encephalotrigeminal angiomatosis, typically consists of a port wine nevus involving the skin of the upper portion of the face, ocular and leptomeningeal angiomatosis, mental retardation, epilepsy, and neurologic deficits. Seizures are the most common manifestation, occurring in up to 90% of patients. The intracranial hallmark is a pial angioma that is believed to represent persistent embryonic vasculature. Seizures are believed to occur because of the
effects of this vascular malformation on the underlying brain. MRI features include enhancement of the pial angioma and underlying cortical atrophy. The pial malformation is usually unilateral and located in the parieto-occipital region. Characteristic "tram track" calcification of the cortex produces a low-signal-intensity ribbon on MRI.

1.1.17 Neoplasms

Imaging features of tumors responsible for intractable epilepsy tend to be small, are well-circumscribed, have little or no surrounding edema, are usually cortical in location, and when cortical, remodel the calvarial inner table. The lesions enhance variably, lack necrosis, and are supratentorial and extraventricular with a predilection for the frontal and temporal lobes.

1.1.18 Astrocytomas

Gliomas account for approximately 72% to 88% of tumors associated with epilepsy. Most lesions are low grade, and outstanding seizure control is expected after resection. Of gliomas presenting with intractable epilepsy, astrocytomas are predominant, accounting for 50% to 70% of gliomas, followed by oligodendrogliomas and oligoastrocytomas.
On MRI, low-grade astrocytomas are well circumscribed, produce little or no mass effect or edema, and do not enhance with contrast. Pilocytic astrocytomas are nearly as common as low-grade diffuse astrocytomas in epilepsy series. These lesions may form cysts and calcify, and they typically have a component that enhances intensely. Pilocytic astrocytomas commonly occur in the diencephalon, optic nerve or chiasm, and cerebellar hemisphere. Seizures are a common presenting symptom when these lesions occur in the cerebral hemisphere.\textsuperscript{12} Pleomorphic xanthastrocytoma is a rare variant with a predilection for the temporal lobe. It is most commonly found in children or young adults with a long history of seizures. The lesions are low grade, and cyst formation is common.

Overall, oligodendrogliomas account for about 5\% of all brain tumors. In epilepsy series, however, oligodendrogliomas are only slightly less common than astrocytomas. The lesions tend to infiltrate cortex and enhance variably. Dense calcification and cyst formation are common. Among gliomas, oligodendrogliomas show the highest potential to develop seizures (71\%), followed by astrocytomas (59\%) and glioblastomas (29\%).\textsuperscript{29}
Low-grade tumors with mixed astrocytic and oligodendroglial elements are common in epilepsy series. The MRI characteristics are indistinguishable from those of low-grade diffuse astrocytomas.

### 1.1.19 Mixed Neuronoglial Tumors

Gangliogliomas and dysembryoplastic neuroepithelial tumors are the two mixed neuronoglial tumors that are most commonly associated with seizures.

Seizures are experienced in 42% to 100% of patients with gangliogliomas. The incidence in intractable epilepsy ranges from about 10% to about 40%. These tumors exist along a spectrum and are termed gangliogliomas if the glial component predominates and gangliocytoma if the neural element predominates. Gangliogliomas are differentiated from gliomas histologically by the presence of malformed neurons in the former. Gangliogliomas are seen most commonly within the temporal lobe. On imaging, the lesions enhance, are frequently cystic with mural nodules, and may calcify.

Dysembryoplastic neuroepithelial tumor occurs almost exclusively in the first 2 decades of life, typically presenting with long-standing epilepsy with onset in early childhood. The lesions consist of all three cell lines (neuronal, oligodendroglial, and astrocytic
elements) with little cellular atypia. The incidence in intractable epilepsy ranges from 1.7% to 7.6%.32 Dysembryoplastic neuroepithelial tumors are cortical lesions that occur almost exclusively in the supratentorial region, particularly in the temporal lobe. On MRI, the lesions are usually well circumscribed, with multinodular irregular cortical thickening and variable signal intensity changes on T1- and T2-weighted sequences. Erosion of the inner table of the calvarium is a common feature. The lesions often have a multicystic appearance, and they may enhance and calcify. The lesion demonstrates remarkably benign biologic behavior, and therapeutic indications are based on considerations of epilepsy control. Some authors classify dysembryoplastic neuroepithelial tumor as a disorder of neuronal migration and proliferation. These tumors are sometimes found in conjunction with an area of focal cortical dysplasia. These lesions may be most appropriately classified somewhere between the categories of migration disorder and tumor.

1.1.20 Other Neoplasms

Other tumors, such as meningioma and metastatic disease, are rare in young patients typically considered for surgical resection; however, they are commonly encountered in late-onset epilepsy. In
patients who present with seizures after age 60 years, the most frequent brain neoplasms are metastatic tumors.\textsuperscript{12}

1.1.21 Mesial Temporal Sclerosis (Hippocampal Sclerosis)

Temporal lobe epilepsy (TLE) is one of the most common medically intractable seizure disorders. Mesial temporal sclerosis is the most common abnormality found in the temporal lobes of these patients. It is generally considered to be a highly epileptogenic lesion and is presumed to be of intractable partial complex epilepsy.\textsuperscript{33} Seventy percent to 90\% of these patients with intractable TLE are seizure-free or suffer only occasional seizures after surgery.\textsuperscript{34}

Hippocampal sclerosis describes neuronal loss and gliosis with atrophy and sclerosis of the cornu ammonis (CA) 1 to CA 4 areas of the hippocampus, the dentate gyrus, and the subiculum. In Ammon's horn sclerosis, the abnormality is confined to areas CA 1 to CA 4. Mesial temporal sclerosis is another term that encompasses changes in the hippocampus as well as in the amygdala and adjacent entorhinal cortex.

Hippocampal sclerosis has been divided into several subgroups.\textsuperscript{35} The most common form is classic Ammon's horn sclerosis with primary nerve cell loss in the CA 1 and CA 4 sections.
of the hippocampus, with less damage to the CA 3 and CA 2 regions. Less commonly, there is widespread cell loss throughout the entire hippocampus, labeled total Ammon’s horn sclerosis. The least common form is end-folium sclerosis with cell loss confined to the CA 4 region.

Autopsy studies have shown that hippocampal sclerosis is bilateral in 47% to 86% of epilepsy cases. Involvement is typically asymmetric, with one side more severely affected. The side of greatest involvement is believed to be the site of seizure origin. A study of the distribution of involvement of hippocampal sclerosis with MRI demonstrated atrophy of the hippocampal body in 88%, the tail in 61%, and the head in 51%. A history of febrile seizures has been associated with hippocampal neuronal loss. Hippocampal sclerosis was seen in 90.4% of 150 epilepsy patients with a febrile seizure history, compared with 46.8% with no history of febrile seizures.

Hippocampal sclerosis is seen in 30% of the general epilepsy population and can be seen in the normal population. There is an inverse relationship between the frequency of hippocampal sclerosis and the age at seizure onset. Although the older the age of seizure onset was, the less likely the patient was to have hippocampal
sclerosis, a young age of onset was consistently associated with hippocampal sclerosis.

Sclerosis of the amygdala was seen in 22% to 27% of postmortem epilepsy cases. Amygdala sclerosis was noted only in cases with hippocampal disease. In 9%, the amygdala was involved bilaterally. A review of MRI studies revealed involvement of the amygdala in 12% of 57 cases of mesial temporal sclerosis.38

The MRI manifestation of neuronal loss and astrogliosis is atrophy and signal alteration.39 Hippocampal volume loss is best detected on heavily T1-weighted oblique coronal images perpendicular to the long axis of the hippocampus. Increased cell water, presumably related to gliosis, is associated with increased signal on T2-weighted images and with decreased signal on T1-weighted images. Ancillary findings seen in hippocampal sclerosis include (1) loss of internal architecture of the involved hippocampus, (2) unilateral atrophy of the mammillary body, (3) unilateral atrophy of the columns of the fornix, (4) unilateral atrophy of the amygdala, and (5) unilateral atrophy of the white matter bundle in the parahippocampal gyrus. Dilatation of the temporal horn is an
unreliable indicator of hippocampal volume loss because it is a common normal variant.

Quantitative assessment of the main findings in hippocampal sclerosis is widely used at major epilepsy centers. A technique for volumetric assessment of the hippocampus has been developed and correlates well with the degree of cell loss measured in subsequent histologic specimens. It has been shown that when EEG evidence of unilateral temporal lobe seizure onset is concordant with predominantly unilateral hippocampal atrophy, the probability of an excellent surgical outcome exceeds 90%. Conversely, in a patient with nonlesional TLE and EEG evidence of unilateral temporal lobe seizure onset, but without volumetric evidence of predominantly unilateral hippocampal atrophy, the probability of an excellent surgical outcome drops below 50%. T2 relaxometry has also been employed to quantify the signal change found in mesial temporal sclerosis.

Quantitative assessment is a beneficial technique, essential for correlative clinical research. Studies of the accuracy of visual perception of hippocampal volume loss have demonstrated an accuracy of 90% by experienced readers. Thus, for clinical purposes,
visual inspection of the hippocampus is sufficient for the identification of predominantly unilateral hippocampal sclerosis.\textsuperscript{12}

1.1.22 Brain Injury

Sclerosis is the final common pathway of many brain insults, any of which may produce an area of brain necrosis, in which death of all cell lines has occurred. Necrosis is typically surrounded by sclerosis. Sclerosis, regardless of cause, has a characteristic MRI appearance, volume loss and signal alteration consistent with increased tissue-free water. The precise cause of seizures in patients with post-brain injury sclerosis is unknown. Response to resective surgery is not as uniformly favorable for neocortical sclerosis as for mesial temporal sclerosis.\textsuperscript{12}

1.1.23 Trauma

The first cortical resection for intractable epilepsy was performed in 1886 with removal of a posttraumatic cortical scar in a patient with a traumatic brain injury.\textsuperscript{42} Trauma is the cause of seizures in 13\% to 17\% of epilepsy cases. Five percent of unselected patients with head injuries develop at least one seizure during the first week after trauma, and 5\% of patients with head injury may develop late epilepsy. The presence of an acute hematoma, a depressed skull
fracture, or a history of early epilepsy increases the risk of late epilepsy to 31%. The frontal and temporal lobes are the most common sites of injury, particularly the orbital surface of the frontal lobes, the ventral surface of the temporal lobes, and the frontal and temporal poles.

1.1.24 Cerebrovascular Diseases

Stroke

Stroke is one of the most frequent causes of seizure in adulthood. After age 50 years, stroke is the most common cause of seizures. Infarction is the most common cause of stroke-related seizures, most likely owing to much higher incidence than hemorrhagic conditions, such as subarachnoid hemorrhage and intracerebral hemorrhage, which have a higher epileptogenicity. Seizures are presumed to arise from gliotic tissue at the periphery of the infarcted zone. The presence of early seizures is not an adverse prognostic factor for patients with strokes. Seizures that occur more than 2 weeks after a stroke have about a 50% chance of causing chronic epilepsy. Early stroke-related seizures are associated with a much better prognosis.

1.1.25 Porencephaly
Porencephaly is one of the most common prenatal brain lesions, related to regions of circumscribed necrosis occurring in utero or before the hemisphere is formed. The underlying mechanism in the formation of these lesions is usually ischemia or intracerebral haemorrhage, related to a major cerebral vascular occlusion. Resorbed neonatal hemorrhages may give rise to these lesions. The developmental origin of these lesions is evident in the lack of perilesional gliosis and the adjacent cortical developmental abnormalities that are commonly associated.47

Porencephaly is commonly bilateral, typically occurring in the territory of the middle cerebral artery, but it may be unilateral. The cysts are not lined by ependyma, and their outer surface is covered by the arachnoid membrane. Porencephalic cysts may communicate with the ventricular system or the subarachnoid space, or both. The cyst may be separated from the ventricle by a thin layer of tissue.

1.1.26 Ulegyria

Ulegyria describes gyral atrophy and sclerosis that are generally more pronounced near the depth of the sulci. The affected gyri become somewhat mushroom-shaped. The lesions are usually unilateral, are most commonly observed in the border zone between
the anterior and the middle cerebral arteries, and are believed to be a result of a perinatal anoxic or ischemic insult. \(^\text{10}\)

1.1.27 Infections

Seizures are often associated with central nervous system infections and may occur in acute, subacute, and chronic meningitis; in encephalitis; and in patients with infectious space-occupying lesions. Many of these conditions present with seizures during the acute phase of the illness, but patients may not develop chronic seizures.\(^\text{48}\) Nineteen percent of patients experience seizures during the acute phase of infection. Early seizures are an important predisposing factor for late unprovoked seizures. The 20-year risk of developing unprovoked seizures in central nervous system infections is 6.8%. Approximately 5% of epilepsy cases are related to previous central nervous system infections.\(^\text{49}\)

1.1.27.1 Viral

Most viral central nervous system infections produce an aseptic meningitis or a mild clinical syndrome of meningoencephalitis. The incidence of viral leptomenigitis in the United States is estimated at 60,000 cases. The most common cause is herpes simplex.
Seizures are common in patients with herpes simplex encephalitis, occurring in approximately 40% of patients.\textsuperscript{50}

1.1.27.2 Bacterial

Approximately 25,000 cases of bacterial meningitis occur each year in the United States. About 70% occur in children less than 5 years of age.\textsuperscript{1} Seizures occur in up to 40% of children with acute bacterial meningitis. Bacterial infections may result in cerebritis and abscess formation. Seizure is a common symptom in brain abscess, seen in 29% of cases, and is the presenting symptom in 25% of these patients.\textsuperscript{51}

1.1.27.3 Tuberculosis and Fungal Diseases

Central nervous system tuberculosis is fairly commonly associated with seizures. In the acute phase, tuberculous meningitis may present with seizures in up to 20% of children and 15% of adults. Seizures may correlate with the presence of a focal lesion, such as a granuloma or tuberculoma. Seizures are frequent in mycotic central nervous system infections, seen in 41% of central nervous system aspergillosis and in 15% of central nervous system cryptococcal infections. Fungal diseases are a rare cause of chronic epilepsy.\textsuperscript{52}

1.1.27.3 Helminthic Infections
Of the helminthic infections that are frequently seizure-associated, seizures are extremely common in cysticercosis, seen in 56% to 70% of cases with cerebral involvement. Cysticercosis is distributed worldwide, but it is particularly common in Central and South America, Africa, India, and some parts of Asia. A drastic increase has occurred within the United States because of an increased number of Latin American immigrants. Other helminthic infections, such as echinococcosis, are less common causes of chronic epilepsy.

1.1.28 Immune-Mediated Disorders

One of the most devastating unilateral epileptic conditions is chronic Rasmussen's encephalitis. This syndrome is characterized by slowly progressive neurologic deterioration and severe focal seizures. The disorder is rare, observed in 1% to 2% of epilepsy cases and in 7.4% of pediatric patients who undergo surgery for intractable epilepsy. An autoimmune process involving glutamate receptors has been shown to be the underlying mechanism. The clinical features consist of early-onset partial seizures, developing in 85% before the age of 10 years, as well as hemiparesis and mental deterioration. Up to 50% of patients have a history of an infectious or inflammatory
episode before the syndrome onset, usually an upper respiratory tract infection. The most common associated seizure types are partial motor seizures, seen in 75% of patients. Epilepsia partialis continua, a condition with frequent seizure attacks, is reported in 60% of patients. Imaging studies demonstrate a hallmark slowly progressive, unilateral hemispheric atrophy, which begins in the temporal insular region. Gliosis may produce increased signal on T2-weighted MRI. Functional isolation of the affected hemisphere by surgery is the treatment of choice.54

1.1.29 Imaging Techniques

Although standard screening protocols may suffice for the evaluation of patients presenting with new-onset seizures, MRI of patients with intractable epilepsy requires an approach tailored to the unique requirements of this population. Specialized sequences employed must be sufficient to detect subtle structural abnormalities (mesial temporal sclerosis, cortical dysgeneses), as well as be sensitive to lesions that may be hemorrhagic or calcified (cavernous malformation, tuberous sclerosis, infection). Optimization must extend to the efficient application of these techniques. In addition, scan techniques must evolve continuously, taking advantage of
technologic advances as they occur, such as computer and surface coil advances that enable higher-resolution imaging matrices, gradient enhancements allowing thinner slices, and new pulse sequence designs that may improve sensitivity to subtle structural and functional abnormalities.

In addition to routine T1- and T2-weighted screening sequences, the MR scanning protocol in epilepsy must include an acquisition with sufficient spatial resolution to detect the most subtle alterations in cortical architecture found in patients with epilepsy, including subtle hippocampal volume loss and cortical thickening. The currently most widely accepted approach employs a thin-section (1.5 to 2.5 mm), three-dimensional, volumetric gradient-echo T1-weighted acquisition in the coronal or, ideally, coronal oblique plane. The data should be corrected for any side-to-side rotation and, if necessary, for perpendicularity with the long axis of the hippocampus before interpretation. This series can be used for three-dimensional reconstruction of the brain surface if necessary.

In suspected TLE, scanning should also include evaluation of the hippocampus with long TR imaging. The contrast properties of the recently available FLAIR fast spin echo (FSE) pulse sequence are
ideal for detection of subtle cortical abnormalities. FLAIR, in which the inversion time is set to null the signal of cerebrospinal fluid, with an echo time long enough to maintain T2-weighting, has been shown to be superior to standard dual-echo T2 pulse sequences in epilepsy.

The MRI seizure evaluation protocol currently in place of the study is as follows:

Axial T1W and T2W, Sagittal T1W, coronal T2W and GE sequence images.

The utility of CT in the evaluation of patients with epileptic seizures became evident soon after the widespread introduction of this technique in the early 1970s. It is readily available. Particularly after the administration of intravenous contrast, CT scanning is useful in the diagnosis of malignant cerebral tumors, either primary CNS neoplasms or metastatic lesions. It is also sensitive in detecting medium- and large-sized cerebral scars resulting from infarctions, infectious diseases, or traumatic brain injuries, and is sensitive in demonstrating calcified lesions, acute intracranial hemorrhages, and arteriovenous malformations (AVMs).
In tertiary medical centers, MRI has largely replaced CT scanning in the evaluation of patients with epileptic seizures. CT is insensitive for lesions such as low-grade neoplasms, cavernous angiomas, hippocampal sclerosis, and cortical dysplasias, which in aggregate are the most common abnormalities in individuals with epilepsy. CT is useful as an adjunct to MRI in the evaluation of calcified lesions. In developed countries where MRI is readily available, routine performance of CT scanning is not cost-effective and is largely unwarranted in the evaluation of epilepsy. It is still widely utilized (perhaps overutilized) in emergency departments for patients presenting with a single seizure, either in the setting of established epilepsy or as a first seizure. In clinical settings where MRI is not available, CT scanning with fourth- or fifth-generation instruments is still a valuable part of the evaluation of patients with partial epilepsy.
OBJECTIVES

This study was planned with the following objectives:-

1/To identify the cause of epilepsy (if any) in the sample selected as shown on imaging.

2/To correlate the imaging findings to clinical findings.

3/To try to identify the clinical features which help in choosing cases needing imaging in the future.
2. Patients, material and methods

2.1 Patients:
In this retrospective study 142 consecutive patients who presented with more than two attacks of seizures and had cross sectional brain imaging at Yastabshiroon Centre were selected.

2.2 Material and methods
The imaging reports were reviewed. The imaging modality was identified, that is, CT, MRI or both. The neuroradiological findings were classified as normal and abnormal. The abnormal finding were classified to consistent with (infarction, haemorrhage, tumours, trauma, malformations of cortical development, vascular malformation, infection, hippocampal sclerosis and non-specific lesions). CT imaging was performed on a spiral Philips CT scanner. Axial images were obtained. Standard MRI was performed on a Philips 0.5 Tesla scanner. Sagittal T1 weighted images and axial T2 weighted and coronal T2 weighted images were obtained.

Data were filled in special data sheet. Imaging findings were correlated to the clinical findings (types of seizure either focal or generalized, findings of EEG (if any).
3. RESULTS

Clinical data

142 patients were identified. Eighty five patients are males and 57 are females (Fig 1). The mean age was 35.2 years (median 35, minimum 0.13, maximum 85 years, standard deviation 23 years). Age distribution was illustrated in (Fig 2). One hundred fourteen patients (80.3%) had generalized seizures and 28 (19.7%) patients had focal seizures (Fig 3). Only 3 patients had EEG done and it was abnormal.

Neuroimaging data:

Eighty nine patients had CT only, 48 patients had MRI only. Five patients only had both CT and MRI scans (Fig 4). 70 of 142 scans (49.3%) were abnormal. 54% of the CT scans were normal and 46% were abnormal, while 52% of the MRI scans were abnormal (Fig 5). Abnormalities were infarction (n=30), tumours (n=10), malformations of cortical development (n=6), non-specific lesions (n=6), vascular abnormalities (n=4), infection (n=5), hippocampal
sclerosis(n=2) (Fig 6). The percentage of detected abnormalities was 68% in focal seizures, 37% in generalised seizure patients.

**Scan results:**

70 of the 142 reviewed scan results (49.3%) were abnormal.

**Focal seizures:**

Abnormalities were most frequently detected in this group (68% abnormalities). In this group 15 patients had CT scans only, 12 patients had MRI only and only 1 patient had both CT and MRI scans.

**Generalised seizure:** Only 37% of patients in this group showed abnormalities. In this group 74% patients had CT scans only, 36 patients had MRI only and only 4 patient had both CT and MRI scans.
Fig. 1: Gender distribution
Fig. 2: Age distribution of the study population
Fig.3: Types of seizures in the study population
Fig. 4 Type of imaging and Findings
Fig. 5: Imaging Findings and age distribution

No of Patients

0 100 200 300

0-20 21-40 41-60 >60 Total Age

- Total
- Abnormal
- Normal
Fig.6: Findings of imaging
4. DISCUSSION
Computed Tomography was introduced in Sudan in 1990 in the Armed Forces Hospital and MRI was introduced in 1998 in Soba University Hospital and to our knowledge no study was conducted to show the findings of these imaging and correlate it with the clinical finding in patients presented with seizures. The study included all patients presented with more than two attacks of seizures and had cross-sectional brain imaging, during the period January to September 2003. Abnormalities were detected in (49.3%) of all patients who presented with seizures irrespective of generalized or focal seizures. Most of the patients had only CT and this may be due to the low cost of CT compared with the cost of MRI. The true prevalence of structural abnormalities may be have been higher because the study included only patients presented with seizures. The results of research studies are not directly applicable to clinical routine because such studies differ from clinical routine in various ways. Research studies may suffer from selection bias, favoring patients with potential cerebral abnormalities. On the other hand, the imaging modalities applied in research studies are likely to be optimal for the detection of abnormalities and the scans are likely to be reviewed by specialists with an interest in epilepsy. This may not
necessarily apply for clinical routine work. A striking finding of our study was that a high proportion of patients had no EEG. The rationale for imaging the brain of patients developing convulsions is first to identify underlying abnormalities such as vascular lesions and tumours that require specific treatment and second to assist the formulation of syndromic and aetiological diagnosis.\textsuperscript{58} Our own data showed that in patients with focal convulsions who were scanned, the prevalence of structural abnormalities was high. More than half of all scans were abnormal.

In patients with generalised seizures the yield of neuroimaging as used in our study was lower than in focal seizures.

The other main finding was the frequent use of CT than MRI in the clinical setting. About two third (89 patients) of patients had only CT scan, one third(48 patients) had a standard MRI with 5 mm thick slices and only 5 patients had both CT and MRI scans . Since the initial application in 1984,\textsuperscript{61} the superiority of MRI over CT in terms of sensitivity and specificity for identifying the aetiology of epilepsy has become firmly established.\textsuperscript{62} Epileptogenic lesions may be missed in patients who had only CT or standard MRI. Hippocampal sclerosis,
small low grade gliomas or small arterio-venous malformations and malformations of cortical development are all difficult to detect on CT. In addition a CT examination of the head involves a radiation dose of 2.3 mSV, which equals more than 100 chest radiographs or the background radiation of about one year.

CT may retain a role as the initial investigation if MRI is not possible because of claustrophobia, acute disorders such as status epilepticus, contraindication for MRI, or if MRI is not available within reasonable time. CT also retains a role, supplementary to MRI, in the recognition of intracranial calcifications that may not be easily detected on MR images. Calcified tubers in tuberous sclerosis, which is commonly associated with epilepsy, are more conspicuous on CT images than MRI. CT is also important in patients with subarachnoid haemorrhage. Calcifications also commonly occur in neurocysticercosis, worldwide main cause of epilepsy. New MRI techniques such as magnetisation transfer imaging may increase the yield of MRI in neurocysticercosis and possible replace CT. In Fig.6, it is realized that for abnormalities the yield of MRI is higher than for CT.
MRI is a versatile diagnostic tool and it is important to choose the appropriate imaging parameters to get optimal results in epilepsy. Important abnormalities such as hippocampal sclerosis, subtle focal cortical dysplasias, and band heterotopia may be difficult to detect on standard MRI and high resolution MRI (slice thickness 1.5 mm) is the method of choice.\textsuperscript{67} The sensitivity of MRI for hippocampal sclerosis can be further improved by the use of FLAIR.\textsuperscript{68} The standard MRI scan of a patient with hippocampal sclerosis on postmortem examination was initially reported as normal. It is recommended to use MRI with thin slices (and preferably including a FLAIR sequence). These MRI sequences are readily available on MRI scanners and add only a few minutes to the examination. Magnetic resonance spectroscopy are developing techniques. Other magnetic resonance techniques such as diffusion imaging,\textsuperscript{69} functional MRI,\textsuperscript{70} quantitative measurements of volume of structures and T2 relaxation time,\textsuperscript{71} serial MRI,\textsuperscript{72} or spectroscopy and multi-modal imaging\textsuperscript{73} have all been used in epilepsy research and may gain a clinical role in selected epilepsy patients in the future.
CONCLUSION

1/Neuroimaging is important for patients presenting with seizures to identify underlying abnormalities.

2/Patients with focal seizures had a higher incidence of imaging abnormalities.

3/CT should be done first and MRI reserved for patients who had normal CT or a CT that was inconclusive.
RECOMMENDATIONS

1/ Imaging of patients presented with seizures is highly recommended.
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