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Neuroradiology *Pictorial Essay*

A myriad spectrum of seizures on magnetic resonance imaging – A pictorial essay

Rahul Karthik Lingutla[1], Ajit Mahale[1], Akshatha R. Bhat[1], Sonali Ullal[1]

[1]Department of Radiodiagnosis, Kasturba Medical College, Mangalore, Manipal Academy of Higher Education, Mangalore, Karnataka, India.

***Corresponding author:**
Rahul Karthik Lingutla,
Department of Radiodiagnosis,
Kasturba Medical College,
Mangalore, Manipal Academy
of Higher Education,
Mangalore, Karnataka, India.

rahulkarthik127@gmail.com

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ABSTRACT

Patients with seizures represent a challenging clinical population both in pediatrics and adults. Accurate diagnosis of the cause of a seizure is important in choosing an effective treatment modality, surgical planning, predicting a prognosis, and follow-up. Magnetic resonance (MR) imaging using a dedicated epilepsy protocol plays a key role in the workup of these patients. Additional MR techniques such as T2 relaxometry and MR spectroscopy show a promising role to arrive at a final diagnosis. The spectrum of epileptogenic causes is broad. Radiologists and physicians need to be updated and require a patterned approach in light of clinical history and electroencephalogram findings to arrive at a reasonable differential diagnosis. This pictorial essay aims to review a few of the common and uncommon causes of seizures and their imaging features.

Keywords: Seizures, Magnetic resonance imaging, Dedicated magnetic resonance imaging protocol, Spectrum of causes, T2 Relaxometry, Mesial temporal sclerosis

INTRODUCTION

Epilepsy is one of the most common diagnoses encountered by neurologists. The challenge in seizure evaluation is to identify the most likely pathology in each case taking age, clinical history, and type of seizure activity into consideration, to best direct the imaging resources and protocols. Accurate diagnosis of the cause of a seizure is important in choosing an effective treatment modality, surgical planning, predict prognosis, and follow-up.[1]

Magnetic resonance imaging (MRI) with its excellent soft-tissue contrast resolution and multiplanar imaging capability is highly sensitive and specific in identifying the underlying structural pathology and demonstrating the precise anatomy with high detail.[2] Additional MR techniques such as contrast-enhanced MR, T2 relaxometry, MR spectroscopy (MRS), and diffusion tensor imaging not only add value to the final diagnosis but also play an important part in pre-surgical evaluation and follow-up. A dedicated epilepsy protocol includes high-resolution fluid-attenuated inversion recovery (FLAIR) coronal oblique sequences obtained perpendicular to the long axis of the hippocampus [Figure 1], coronal oblique T1 inversion recovery sequence and T2 relaxometry in addition to the conventional sequences.[3-5] MRI has a maximum yield in the evaluation of patients with focal onset of seizures on history, clinical examination, or electroencephalogram (EEG), and refractory seizures.[6]

This pictorial essay aims to illustrate the common and uncommon causes of seizures and their imaging features on MRI illustrated in Table 1[7,8] and a brief overview of the utility of T2 relaxometry in seizure evaluation.

DISORDERS OF CORTICAL MALFORMATION

Table 1: Common and uncommon MRI spectrum of abnormalities encountered in epilepsy imaging.

Disorders of cortical malformation	Gray matter heterotopia Schizencephaly Focal cortical dysplasia Hemimegalencephaly Polymicrogyria
Mesial temporal sclerosis	
Periventricular leukomalacia	
Phacomatosis	Tuberous sclerosis Sturge-Weber syndrome
CNS tumors	Glioma, dysembryoplastic neuroepithelial tumor, ganglioglioma, pleomorphic xanthoastrocytoma, craniopharyngioma, dermoid cyst, and meningioma.
Infections	Tuberculoma Leptomeningitis Neurocysticercosis
Vascular malformations	Cerebral arteriovenous malformation Cerebral slow flow venous malformation
Encephalomalacia and gliosis	
Miscellaneous uncommon causes	Dyke-Davidoff-Masson syndrome Rasmussen encephalitis Fahr disease Posterior reversible encephalopathy syndrome Arachnoid cyst causing significant mass effect Skull base meningoencephalocele Megalencephalic leukoencephalopathy with subcortical cysts/Van der Knaap disease

MRI: Magnetic resonance imaging

GRAY MATTER HETEROTOPIA

It occurs due to arrest/disruption of normal neuronal migration anywhere between the periventricular germinal zone and cortex during fetal life. They can be subependymal/periventricular, subcortical, or band heterotopia. The heterotopic nodules show gray matter signal intensity on all sequences with no enhancement post-contrast [Figure 2]. Heterotopia can be unilateral or bilateral and can vary from a single small nodule to a coalescent clump of gray matter nodules.[9] The major differential diagnosis for periventricular heterotopia is subependymal nodules of tuberous sclerosis which can be differentiated by the presence of calcification in the latter.

SCHIZENCEPHALY

These are transmantle clefts in the brain parenchyma lined by dysplastic gray matter extending from the cortex to the ependymal lining of the lateral ventricle and occur due to abnormal post-migrational development.[10] The two types include open and closed-lip schizencephaly. They are often associated with other forms of cortical migration abnormalities. A close differential to be wary, of is a porencephalic cyst which is lined by gliotic white matter.

FOCAL CORTICAL DYSPLASIA (FCD)

FCD is a common cause of medically refractory seizures in a pediatric population. On MR evaluation, the common findings include a focal area of cortical thickening/thinning with the blurring of gray-white matter margin [Figure 3] and

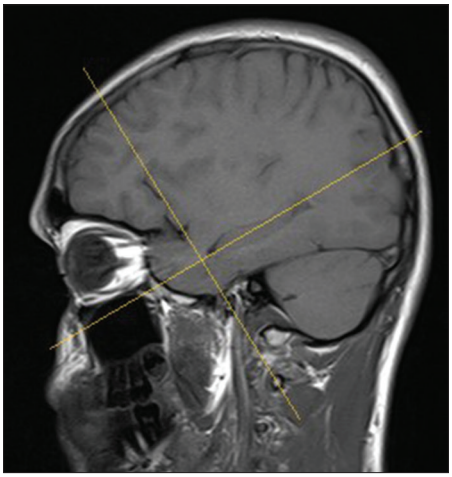

Figure 1: Magnetic resonance imaging planning of coronal oblique sequences obtained perpendicular to the long axis of hippocampus on a sagittal T1 localizer image.

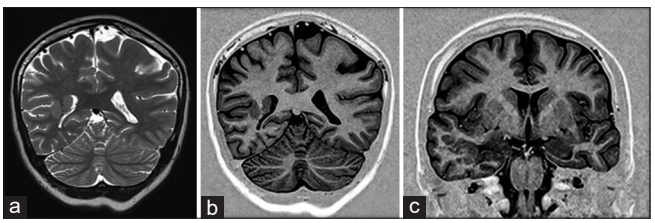

Figure 2: Subcortical and periventricular nodular heterotopia in a 12-year-old female patient with complex partial seizures. Magnetic resonance imaging brain (a) coronal oblique T2W and (b and c) coronal oblique T1 inversion recovery sequence demonstrate multiple nodules of gray matter signal intensity in subcortical and periventricular regions on the right posterior temporal lobe. The gray-white matter differentiation is better delineated in T1 IR sequence.

adjacent T2W/FLAIR hyperintensity extending in a tapered fashion up to the ventricular margin called the transmantle sign, which is better appreciated in FCD Type IIb.[11] FCD can occur together with mesial temporal sclerosis (MTS) and other epilepsy-associated tumors such as dysembryoplastic neuroepithelial tumor (DNET) and ganglioglioma.

The gray-white matter junction and blurring of margins are often appreciated better on T1 inversion recovery sequences than on conventional T1W sequences.[5] 3T MRI is proposed to have higher sensitivity in the detection of subtle FCDs which may be missed on 1.5 T MRI.[12-14] The main differential for FCD is cortical-based tumors like low-grade gliomas. Cortical thickening with the blurring of gray-white matter junction favors FCD while cortical expansion with mass effect on adjacent sulci favors tumor.[15] Decrease in NAA and increase in Choline peak help differentiate low-grade gliomas from FCD on MRS.[16]

MTS

MTS is a common structural abnormality seen in association with temporal lobe epilepsy. High-resolution coronal oblique T2W/FLAIR sequences obtained perpendicular to the long axis of the hippocampus are required for optimal evaluation of the hippocampus [Figure 1].

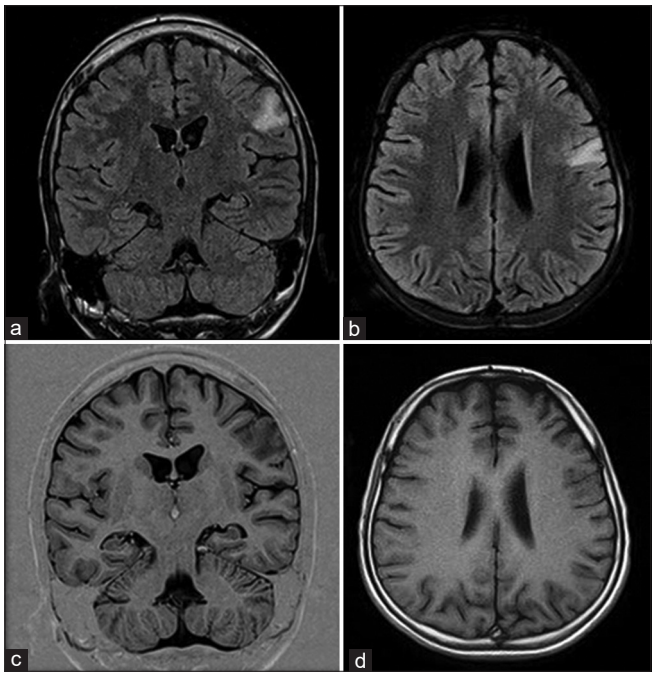

Figure 3: Focal cortical dysplasia in a 14-year-old male patient presenting with complex partial seizures. Magnetic resonance imaging brain (a and b) coronal oblique and axial fluid-attenuated inversion recovery images show focal hyperintensity involving cortex and subcortical white matter of the left frontal lobe with blurring of gray-white matter interface. (c and d) Coronal oblique T1 inversion sequence and routine axial T1W images show that the thickened dysplastic cortex was better appreciated on T1 IR sequence than routine T1W sequence.

Classical features of MTS include increased signal intensity on the T2W/FLAIR sequence with hippocampal volume loss and loss of normal internal architecture and hippocampal indentations [Figure 4a]. The findings are presumed to be secondary to gliosis.

Secondary signs associated with hippocampal sclerosis are atrophy of the ipsilateral mammillary body, fornix and amygdala, dilatation of ipsilateral temporal horn and adjacent choroid fissure. A subtle indistinct gray-white interface in the adjacent anterior temporal lobe is also seen less frequently.[17,18]

MTS can be bilateral and often one side is more severely affected than the other. A closer look at the adjacent cortex is warranted as these lesions can also be associated with cortical dysplasia.[17]

MR volumetric analysis of the hippocampus allows quantitative measurement of hippocampal volume and is obtained by tracing the entire length of the hippocampus. It can be used to detect the presence of hippocampal atrophy and enhance the standard visual analysis with subtle/borderline cases of MTS.[19]

T2 relaxometry allows quantification of T2 relaxation times of the hippocampus which is markedly elevated secondary to neuronal loss [Figure 4b]. T2 relaxometry has the potential to detect early MTS, where subtle volume loss often escapes detection by volumetric or visual examination. T2 relaxometry was proved more efficient in non-invasive lateralization of epileptogenic focus and more sensitive than MR volumetry in detecting early MR-negative lesions.[20] Abnormal relaxation time values are considered more than 2 standard deviations outside the mean value of control hippocampal T2 relaxation times.[21] Placement of region of interest (ROI) for calculating the hippocampal relaxation time is of great importance as abnormal high values are obtained if the ROI includes CSF from the adjacent temporal horn.

Metabolically healthy neurons are marked by the presence of N-acetyl aspartic acid (NAA) on MRS. In hippocampal sclerosis, there is a loss of NAA resulting in decreased NAA/

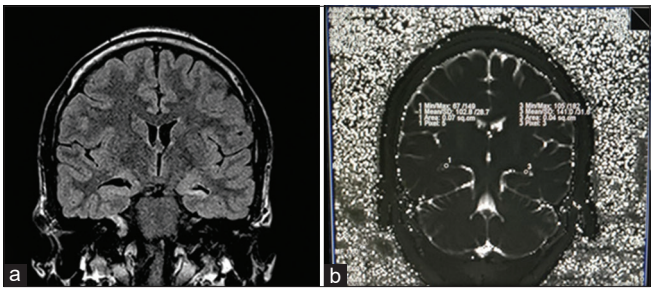

Figure 4: Mesial temporal sclerosis in a 23-year-old male patient with a known history of seizures. Magnetic resonance imaging brain (a) coronal fluid-attenuated inversion recovery image shows increased signal intensity with volume loss, involving the left hippocampus with associated enlargement of ipsilateral temporal sulcus. (b) T2 relaxometry sequence showing increased relaxation time (~141 ms) with ROI placed in the head region of the left hippocampus.

choline+creatinine ratio <0.71.[22] MRS was found to have higher sensitivity and specificity than conventional MRI in the detection of MTS.[23]

PERIVENTRICULAR LEUKOMALACIA (PVL)

PVL occurs as sequelae to perinatal hypoxic-ischemic encephalopathy and is characterized by the presence of patchy areas of gliosis with periventricular white matter volume loss. The posterior aspect of bilateral lateral ventricles is often dilated with irregular margins suggestive of colpocephaly [Figure 5].[24,25]

NEUROCUTANEOUS SYNDROMES

Tuberous sclerosis complex

Tuberous sclerosis complex is a neurocutaneous syndrome affecting multiple organ systems. Its CNS manifestations include multiple cortical tubers, radial migration lines and subependymal nodules with calcification [Figure 6]. Frequent follow-up is suggested for the development of subependymal giant cell astrocytoma in the region of foramen of Munro.[26]

These patients should undergo further investigations to look for associated renal, cardiac, thoracic, musculoskeletal, and cutaneous abnormalities, thus, ensuring appropriate clinical management of patients and counseling of parents.

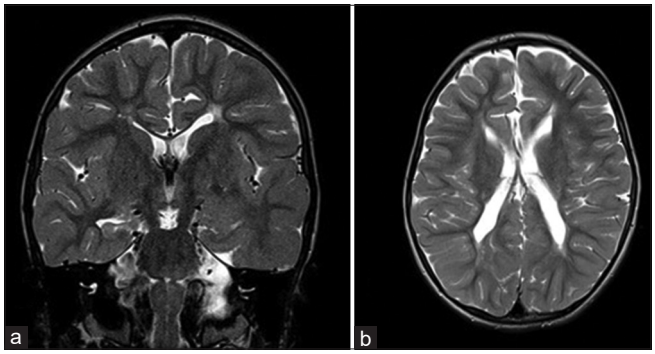

Figure 5: Periventricular leukomalacia with mesial temporal sclerosis in a 4-year-old female child with seizures and developmental delay. Magnetic resonance imaging brain (a) coronal oblique T2W image demonstrates hyperintensity with volume loss involving the right hippocampus. (b) Axial T2W image shows hyperintensities involving periventricular white matter with paucity of white matter suggested by the cortical sulci reaching up to the ventricular margin and colpocephaly.

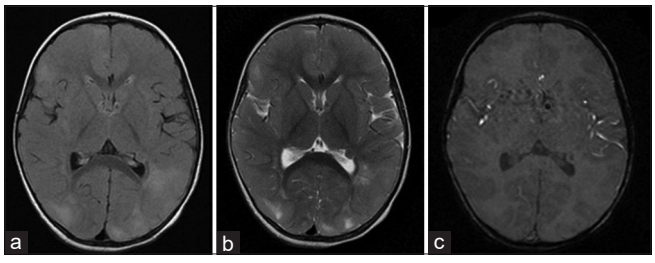

Figure 6: A 2-year-old male child with tuberous sclerosis complex presented with GTCS and global developmental delay. Magnetic resonance imaging brain (a and b) axial fluid-attenuated inversion recovery and T2W images show multiple nodular cortical-based hyperintensities in bilateral cerebral hemispheres suggesting cortical tubers. Hyperintense radial bands extending from the cortical tubers to the ventricular surface. Isointense subependymal nodules along bilateral lateral ventricles. (c) Susceptibility-weighted imaging reveals blooming foci at the subependymal nodules suggestive of calcification.

STURGE-WEBER SYNDROME

Sturge-Weber syndrome is a rare sporadic vascular neurocutaneous syndrome characterized by a dermal capillary venular malformation in the sensory distribution of the trigeminal nerve, retinal choroidal angioma, and a cerebral capillary venous leptomeningeal angioma. On MRI, there is hemicerebral atrophy with gyriform cortical/subcortical T2 hypointensity that blooms on SWI suggesting calcifications. FLAIR scans show serpentine sulcal

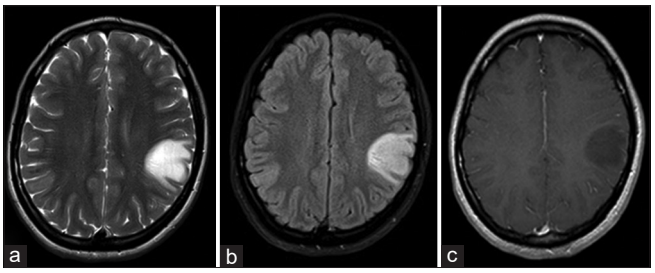

Figure 7: Dysembryoplastic neuroepithelial tumor in a 20-year-old female patient with complex partial seizures. Magnetic resonance imaging brain (a) axial T2W image showing bubbly cortical-based lesion in the left parietal lobe with no surrounding edema. (b) Axial fluid-attenuated inversion recovery image shows a hyperintense rim around the lesion. (c) Axial post-contrast T1W image with no enhancement.

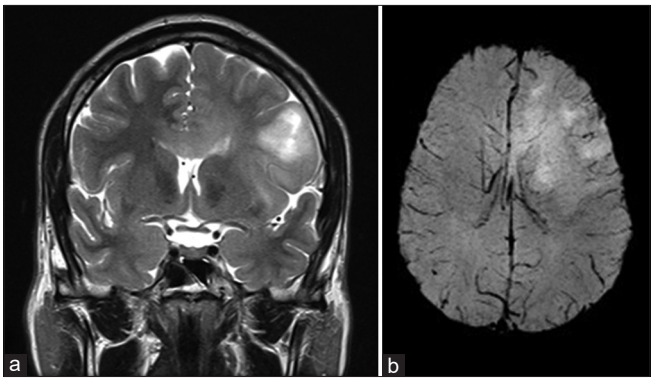

Figure 8: Oligodendroglioma in a 28-year-old male patient presenting with seizures. Magnetic resonance imaging brain (a) coronal T2W image shows a heterogeneous hyperintense cortical-based lesion in the left frontal lobe with surrounding edema. (b) Susceptibility-weighted image shows few small foci of blooming suggesting calcifications.

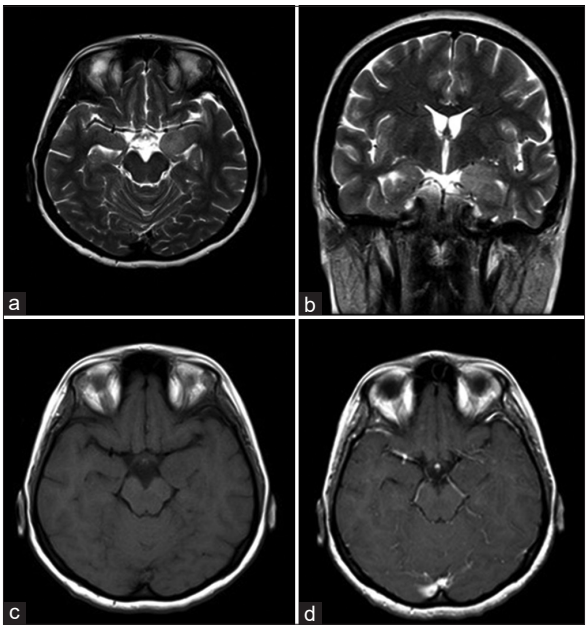

Figure 9: Low-grade glioma in a 20-year-old male patient with complex partial seizures. Magnetic resonance imaging brain (a and b) axial and coronal T2W images showing intra-axial hyperintense lesion in the left medial temporal lobe. (c and d) Pre- and post-contrast T1W image, lesion is hypointense with no enhancement post-contrast.

hyperintensity called the ivy sign. Post-contrast images show enhancing pial angioma with enlarged ipsilateral choroid plexus and enlarged perimedullary veins.[27]

CNS TUMORS

The common CNS tumors associated with seizures include DNET [Figure 7], ganglioglioma, oligodendroglioma [Figure 8], pleomorphic xanthoastrocytoma, and low-grade glioma[8] [Figure 9]. The epileptogenic feature among these would be the cortical base of the tumor. High-grade gliomas [Figure 10] can also frequently cause seizures due to their large size and extensive surrounding edema. Hypothalamic hamartoma is known to cause gelastic seizures [Figure 11].[28]

Extra-axial tumors such as craniopharyngioma [Figure 12], meningioma [Figure 13], and dermoid cyst [Figure 14] can also present with seizures due to mass effect and vasogenic edema in the adjacent neuroparenchyma. Intracranial dermoid cysts may remain asymptomatic until they rupture and cause chemical meningitis.[29]

Although arachnoid cysts are a common benign incidental finding on MR, they may very rarely cause seizures in case of large cysts with significant mass effect [Figure 15].[30]

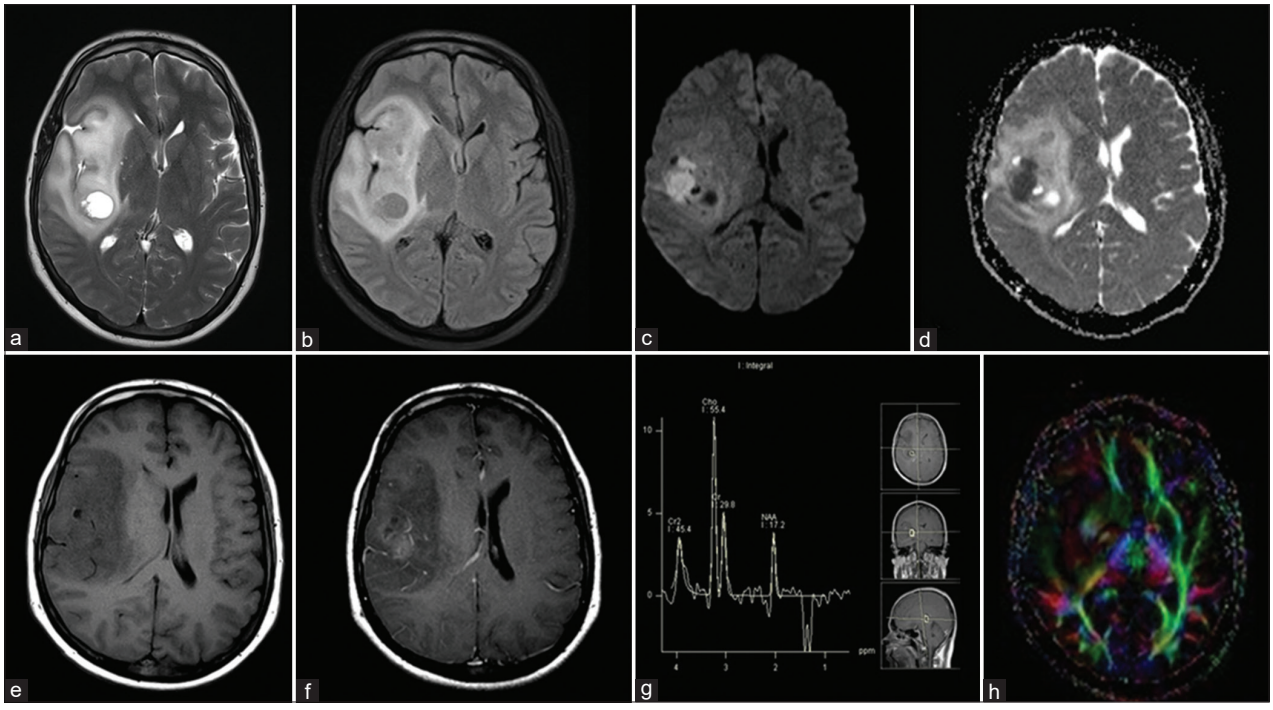

Figure 10: High-grade glioma in a 35-year-old male patient who presented with GTCS. Magnetic resonance imaging brain (a and b) axial T2W and fluid-attenuated inversion recovery images showing ill-defined intra-axial hyperintense lesion with cystic changes. (c and d) Patchy areas of high signal on DWI with corresponding low signal on apparent diffusion coefficient, suggestive of true diffusion restriction. Magnetic resonance imaging brain (e and f) pre- and post-contrast T1W images demonstrate the ill-defined hypointense lesion with patchy areas of contrast enhancement. (g) Magnetic resonance spectroscopy showing elevated choline peak and lipid lactate inversion on intermediate TE. (h) Diffusion tensor imaging showing anisotropy of the right inferior occipitofrontal fasciculi.

INFECTIONS

Acute infections causing meningitis [Figure 16], viral encephalitis [Figure 17] and chronic infective etiologies such as fungal/tubercular granulomas [Figure 18], pyogenic abscess, and parasitic infections such as neurocysticercosis [Figure 19] are frequently associated with seizures.[31]

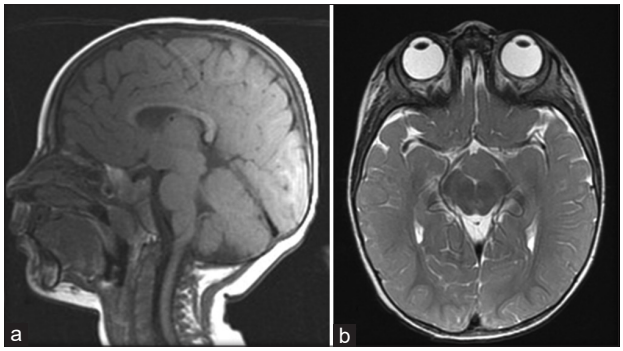

Figure 11: Hypothalamic hamartoma in a 5-year-old female child with gelastic seizures. Magnetic resonance imaging brain (a and b) sagittal T1W and axial T2W images, respectively, show extra-axial well-defined isointense lesion in the region of hypothalamus. There was no enhancement on post contrast images.

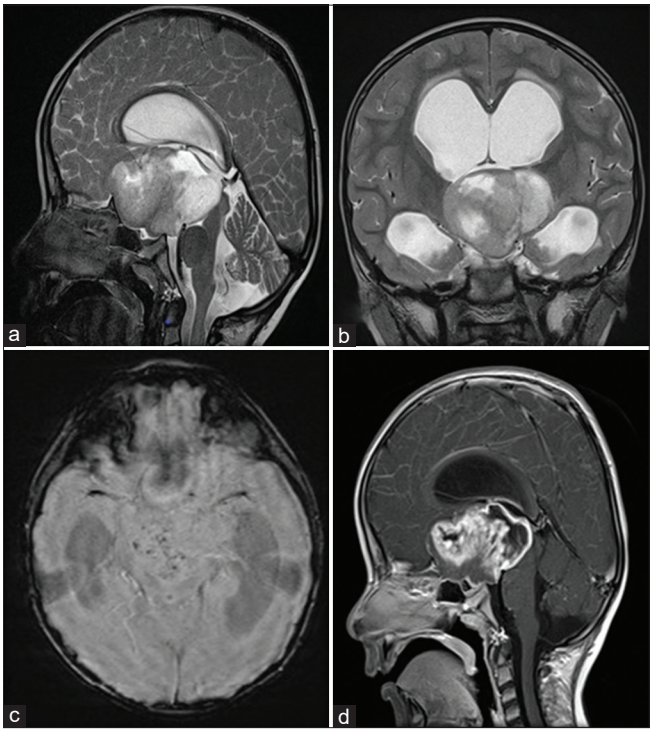

Figure 12: Craniopharyngioma in a 4-year-old male child presented with GTCS. (a and b) Magnetic resonance imaging brain axial and coronal T2W image demonstrates an extra-axial lobulated heterogeneously hyperintense solid cystic lesion in suprasellar region causing obstructive hydrocephalus. The pituitary gland is seen separately. (c) Susceptibility-weighted image reveals foci of blooming within the lesion suggesting calcifications. (d) Post-contrast T1W image shows heterogeneous enhancement.

VASCULAR MALFORMATIONS

Most frequently involved as a cause of seizure are cerebral arteriovenous malformation (AVM) and cerebral slow flow venous malformations. Seizures can occur *de novo* given

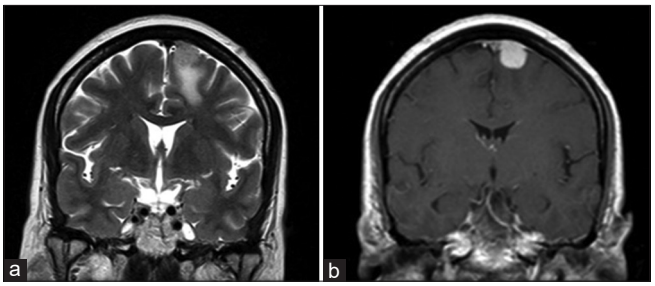

Figure 13: A 61-year-old female patient with frontal meningioma who presented with complex partial seizures. Magnetic resonance imaging brain (a) coronal T2W image reveals a well-defined extra-axial isointense lesion in the left frontal region with edema in the surrounding neuroparenchyma. (b) Post-contrast T1W image shows intense homogeneous enhancement.

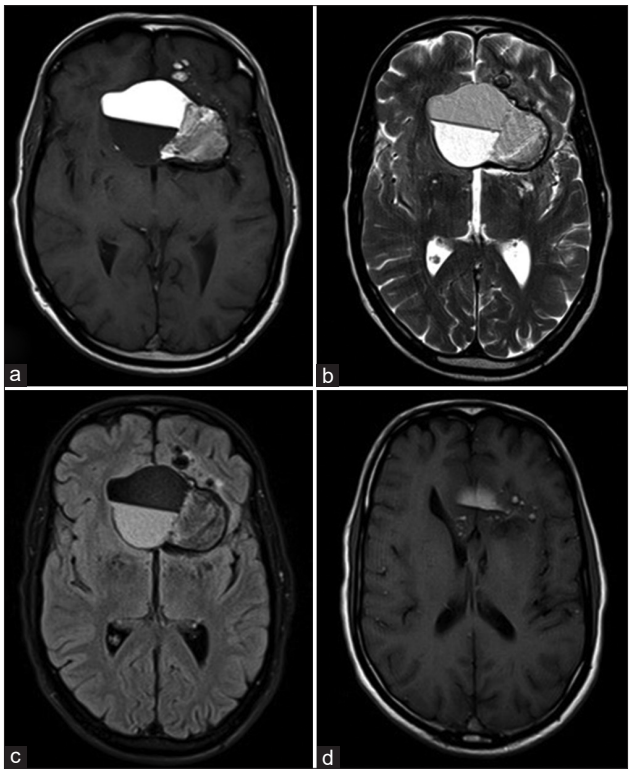

Figure 14: Ruptured intracranial dermoid cyst in a 55-year-old male patient with headache and seizures. Magnetic resonance imaging brain (a and b) axial T1W and T2W images show an extra-axial lobulated heterogeneous hyperintense lesion with hypointense rim and fat-fluid level involving bilateral frontal regions. (c) In axial fluid-attenuated inversion recovery image with fat suppression the T1W hyperintense layer appears hypointense due to fat suppression. (d) Axial T1W image at a section superior than (a) reveals multiple hyperintense fat locules seen scattered in subarachnoid space suggestive of rupture.

their supratentorial location and involvement of the cortex but also secondary to intracerebral hemorrhage.[32]

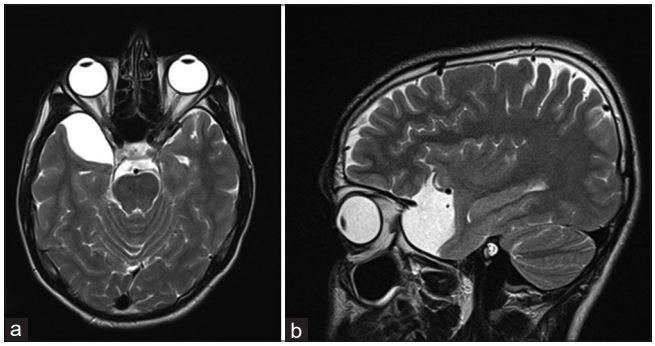

Figure 15: Arachnoid cyst in an 18-year-old male patient with seizures. (a and b) Magnetic resonance imaging brain T2W axial and sagittal images showing extra-axial CSF signal intensity cystic lesion with mass effect in the right anterior temporal region.

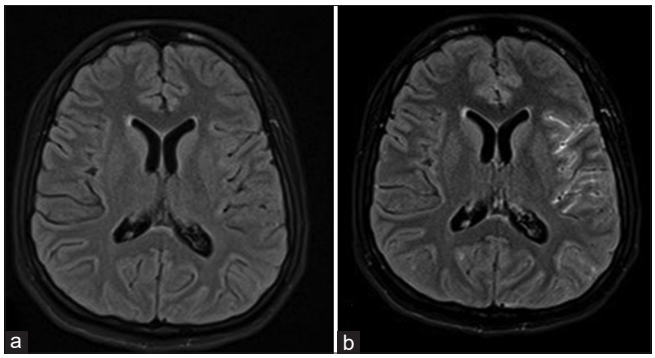

Figure 16: A 26-year-old woman with leptomeningitis presented with generalized tonic clonic seizures and fever. (a) Magnetic resonance imaging brain axial pre-contrast fluid-attenuated inversion recovery sequence shows no demonstrable abnormality. (b) Post-contrast axial fluid-attenuated inversion recovery image demonstrates leptomeningeal enhancement in the left Sylvian fissure and left temporo-occipital cortical sulci.

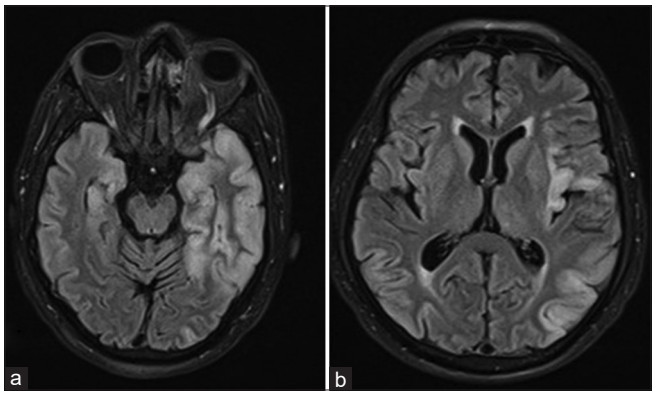

Figure 17: HSV encephalitis in a 61-year-old male patient with altered sensorium and seizures. (a and b) Magnetic resonance imaging brain axial fluid-attenuated inversion recovery sequence shows asymmetric cortical swelling with hyperintensity involving bilateral temporal and parietal lobes.

AVMs are characterized by an abnormal tangle of flow voids representing a nidus, with multiple prominent arterial feeders and dilated early draining veins [Figure 20].

Cerebral slow flow venous malformations, previously called cavernous malformations [Figure 21], are composed of blood-filled vascular spaces with blood in different stages of

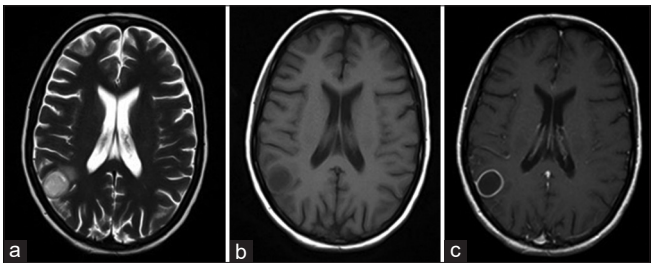

Figure 18: Tuberculoma in a 26-year-old male patient presenting with fever, headache, and seizures. Magnetic resonance imaging brain (a and b) axial T2W and T1W images, respectively, showing well-defined intra-axial lesion with central hypointensity and isointense rim. (c) Post-contrast T1W image reveals ring enhancement.

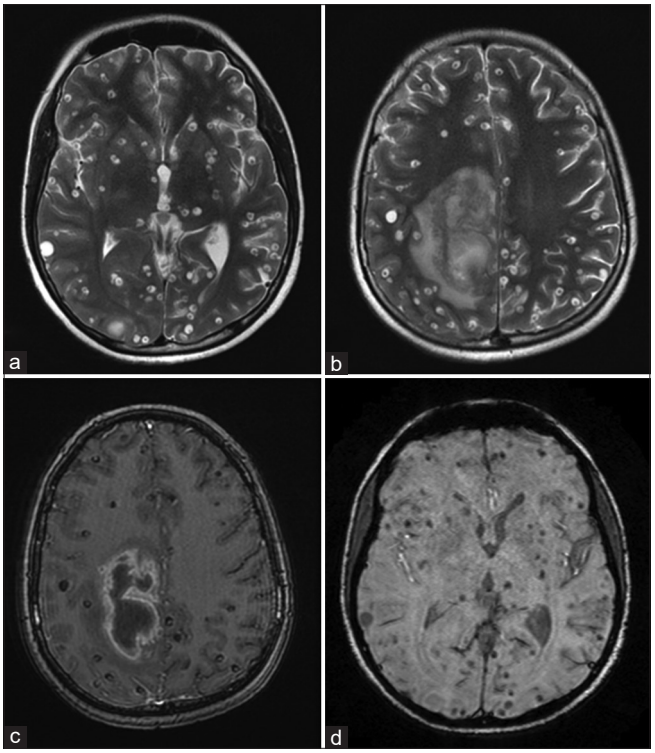

Figure 19: Neurocysticercosis in a 17-year-old male patient with seizures. Magnetic resonance imaging brain (a) axial T2W image shows multiple small cystic lesions with small eccentric hypointense foci suggestive of scolex in a vesicular stage. The scolices are even better appreciated on heavily T2W sequences like FIESTA/CISS. (b) Axial T2W image at a higher level shows an irregular hyperintense lesion with hypointense rim and extensive surrounding edema. (c) Axial T1W post-contrast image shows peripheral enhancement suggesting colloidal stage. (d) Susceptibility-weighted image shows multiple foci of blooming suggesting calcifications.

maturation. They range from Type I to Type IV according to the Zabramski classification of cavernous malformation based on the stage of hemorrhage.[27] They are commonly associated with developmental venous anomalies as a part of mixed vascular malformations.

ENCEPHALOMALACIA AND GLIOSIS

It occurs as a result of a variety of CNS injury including infarction, infection, trauma, and hemorrhage. It can act as a potential epileptogenic focus and is one of the common findings on MR evaluation of patients with focal seizures.[33]

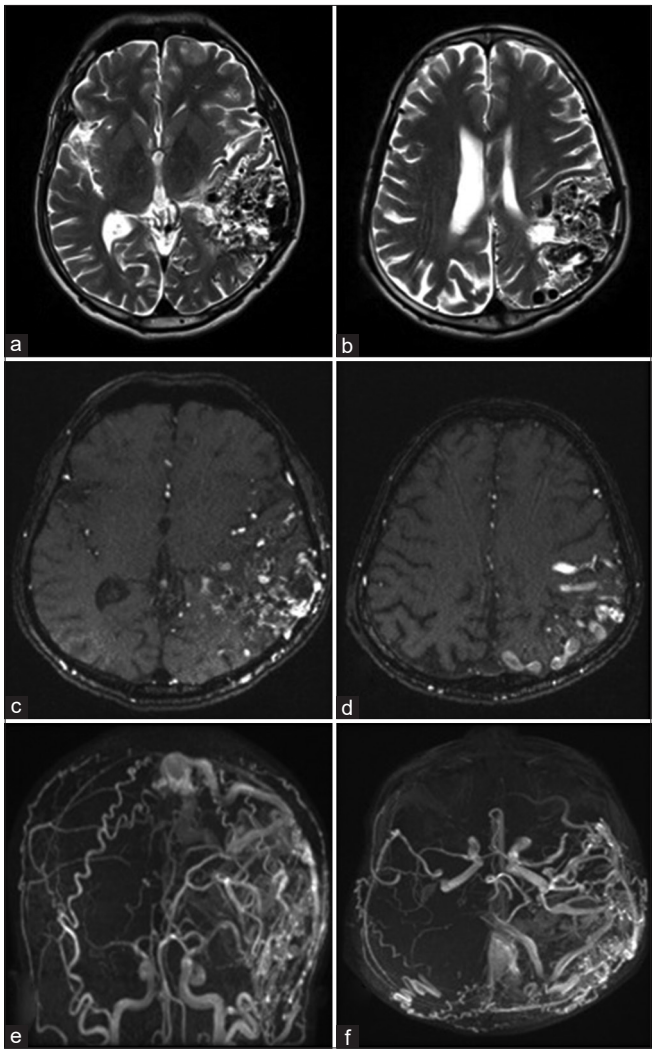

Figure 20: Cerebral arteriovenous malformation in a 33-year-old male patient presenting with GTCS. (a and b) Magnetic resonance imaging brain T2W axial images show a compact tangle of flow voids in the left parietotemporal lobe. Time-of-flight MRA source (c and d) and 3D reconstructed (e and f) images reveal prominent feeding arteries from the left MCA and early dilated draining veins, draining into the superior sagittal sinus, and left sigmoid sinus.

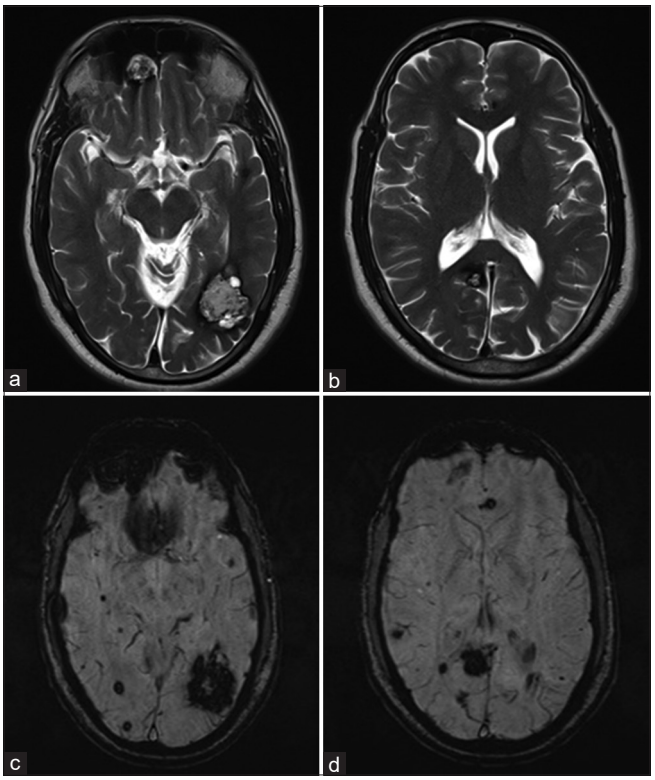

Figure 21: Cerebral cavernous malformation in a 40-year-old male patient presented with headache and seizures. Magnetic resonance imaging brain (a and b) axial T2W images showing heterogeneously hyperintense lesions with hypointense rim with classic popcorn ball appearance in bilateral cerebral hemispheres. (c and d) Corresponding SWI images showing intense blooming in the above lesions and multiple additional foci of blooming – consistent with Type II and Type IV cavernoma, Zabramski classification.

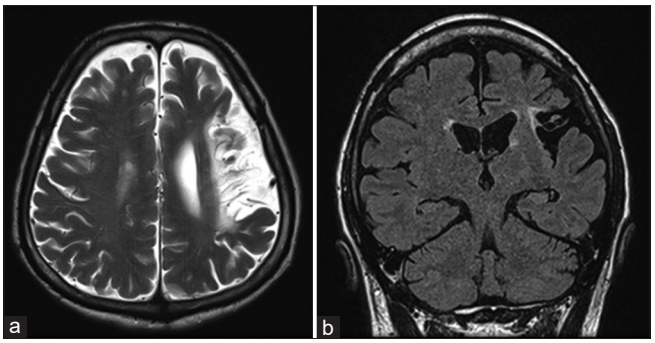

Figure 22: Encephalomalacia and gliosis in a 48-year-old male patient with seizures. Magnetic resonance imaging brain (a and b) axial T2W and coronal oblique fluid-attenuated inversion recovery images, respectively, show encephalomalacic changes with adjacent hyperintensity suggesting gliosis in the left frontoparietal lobe and associated mild ex vacuo dilatation of the left lateral ventricle.

It is characterized by the presence of focal or diffuse volume loss representing encephalomalacic changes with associated T2W/FLAIR hyperintense signal which suggests gliosis [Figure 22]. The margin may be lined by hemosiderin due to a break in the blood-brain barrier better seen on susceptibility-weighted imaging.

DYKE-DAVIDOFF-MASSON SYNDROME

A rare entity, characterized by hemicerebral atrophy secondary to primary CNS insult in fetal or early infantile. It is frequently associated with compensatory hyperostosis of adjacent calvarium and hyperpneumatization of ipsilateral paranasal sinus [Figure 23]. This needs to be differentiated from Rasmussen encephalitis which is another rare cause of seizures and hemicerebral atrophy by the absence of calvarial and paranasal sinus changes [Figure 24]. A probable immunological cause has been proposed to be the etiology of Rasmussen encephalitis.[34]

FAHR DISEASE

Fahr disease is characterized by the presence of extensive bilateral symmetrical intracerebral calcifications predominantly involving the deep gray nuclei [Figure 25]. The usual presentation of Fahr disease is that of extrapyramidal and behavioral symptoms but can rarely present with an epileptic seizure.[35]

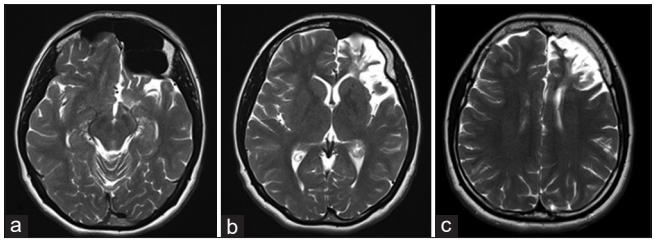

Figure 23: Dyke-Davidoff-Masson syndrome in a 17-year-old female patient presenting with complex partial seizures. (a and b) Magnetic resonance imaging brain axial T2W image demonstrates atrophy of the left frontal lobe with ipsilateral enlargement of the left frontal sinus and hyperostosis of the left frontal bone.

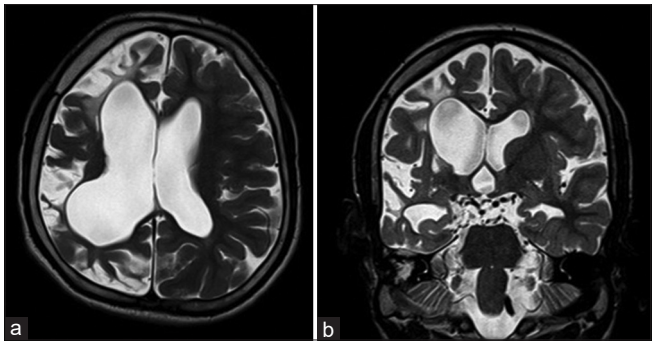

Figure 24: Rasmussen encephalitis in a 25-year-old male patient presenting with seizures. (a) (b) Magnetic resonance imaging brain axial and coronal T2W images demonstrate right hemicerebral atrophy with ex vacuo dilatation of the right lateral ventricle. There was no hypertrophy of adjacent calvarium/frontal sinus enlargement, differentiating it from Dyke-Davidoff-Masson syndrome.

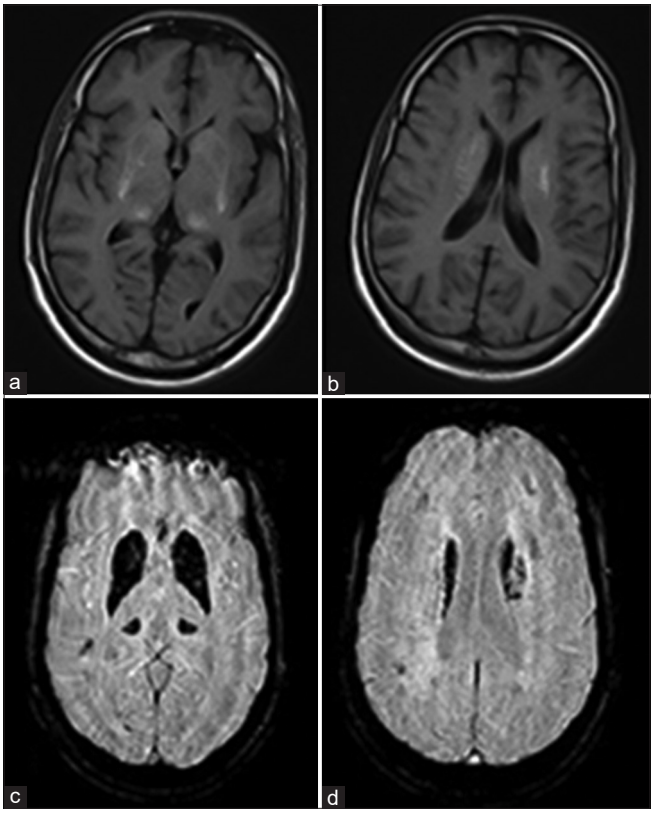

Figure 25: Fahr disease in a 48-year-old male patient presenting with prolonged history of movement disorder and seizures. Magnetic resonance imaging brain (a and b) axial T1W images show patchy hyperintensities involving bilateral basal ganglia, dorsal thalami nuclei, and bilateral corona radiata. (c and d) Susceptibility-weighted imaging showing extensive blooming corresponding to the T1 hyperintense areas. CT brain (not shown) revealed extensive cerebral calcifications.

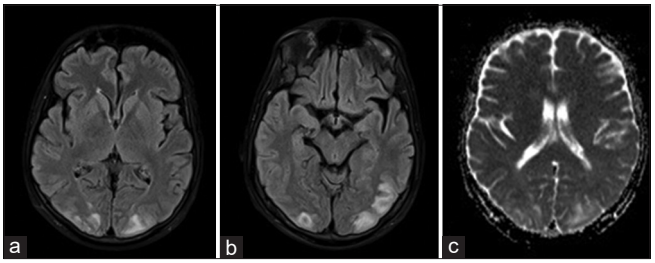

Figure 26: Posterior reversible encephalopathy syndrome in a 36-year-old hypertensive male patient presenting with seizures. Magnetic resonance imaging brain (a and b) axial fluid-attenuated inversion recovery images reveal bilateral symmetrical hyperintensities involving the cortex and subcortical white matter of bilateral parieto-occipital lobes. (c) Axial apparent diffusion coefficient image shows T2 shine through.

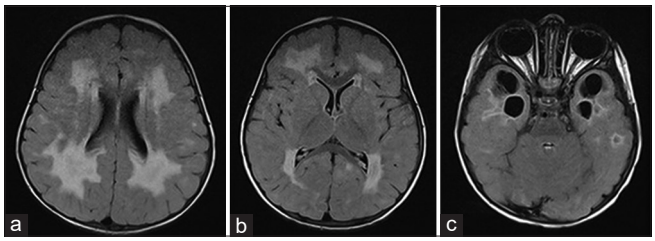

Figure 27: Megalencephalic leukoencephalopathy with subcortical cysts/Van der Knaap disease in a 1 1/2-year-old male child with seizures and global developmental delay. Magnetic resonance imaging brain (a and b) axial fluid-attenuated inversion recovery images reveal extensive symmetrical confluent white matter hyperintensity involving periventricular, deep, and subcortical white matter. (c) Subcortical cysts in bilateral temporal lobes.

POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME (PRES)

PRES can be associated with a variety of conditions such as hypertension, eclampsia, toxin exposure, and autoimmune diseases and can also have a range of presentations such as headache, altered sensorium, visual disturbances, and rarely seizures. It is manifested by the presence of vasogenic edema in bilateral parieto-occipital lobes and T2 shine through in diffusion-weighted imaging [Figure 26].[36]

SKULL BASE MENINGOENCEPHALOCELES

It can be spontaneous or traumatic. Spontaneous skull case meningoencephaloceles can be associated with idiopathic intracranial hypertension and central obesity which impair cerebral venous return to the heart which, in turn, increases the intracranial CSF pressure. They usually present with CSF rhinorrhea, otorrhea, or can remain asymptomatic and can be incidentally detected. They can rarely present as temporal lobe epilepsy due to herniation and traction of the involved cortex.[37-39]

MEGALENCEPHALIC LEUKOENCEPHALOPATHY WITH SUBCORTICAL CYSTS

Previously called Van der Knaap disease is another rare slowly progressive neurodegenerative disorder characterized by infantile macrocephaly, mild neurological symptoms, seizures and cerebral leukoencephalopathy. MRI shows early confluent involvement of subcortical and deep white matter with subcortical cysts predominantly in anterior temporal poles [Figure 27].[40]

CONCLUSION

Both the radiologist and the referring clinician must be updated and keep refreshing their current knowledge given the myriad causes of seizures encountered in clinical practice. Employing dedicated imaging protocols and systematically reviewing the images, taking the relevant clinical history and EEG findings into consideration, radiologist plays a significant role in the evaluation of patients presenting with seizures. A dedicated coronal oblique T2W and FLAIR sequences planned along the axis of the hippocampus, T2 relaxometry, and MR volumetry are ideal in the diagnosis of early MTS. Moreover, the T1 inversion recovery sequence aids in the diagnosis of malformations of cortical development by a better depiction of gray-white matter junction.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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