Brain attack: Imaging of non traumatic neuro emergencies, what radiologists should know

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Learning objectives

"BRAIN ATTACK" is an emergency code entitled to the patients presenting with sudden onset of neurological symptoms, without history of recent trauma. Neuroimaging is vital for the diagnosis and subsequent management. Radiologists should be aware about the imaging features and possible differential diagnosis of various neuroemergencies. The aim of this poster is thus directed to:

1. Review the protocols and current state of imaging for evaluation of non-traumatic neuro emergencies, with emphasis to stroke.

2. Briefly discuss the possible clinical entities and their pertaining imaging features, other than stroke presenting with acute neurological symptoms.
Background

"Brain attack" is a term used to describe non traumatic acute presentation of neurologic symptoms, may be secondary to stroke or any other neurological conditions/stroke mimics. Different imaging techniques and protocols have been developed to aid in determining the cause of these acute neurologic symptoms and to assess brain tissue status. A standardized imaging approach is proven to be practical and efficient in the prompt diagnosis of Brain attack cases so that urgent and appropriate action may be undertaken. Important considerations in developing an imaging protocol for these cases include time urgency, availability of expertise, and availability of possible intervention such as endovascular therapy.

Imaging protocol:

The imaging protocol/algorithm that will be discussed below are in concordance with the American College of Radiology Appropriateness Criteria. (Fig. 1 on page 26)

CT-scan

The high specificity and sensitivity of plain CT-scan to the presence or absence of acute blood made it an accepted standard-of-care imaging technique in emergent evaluation of acute intracranial hemorrhage. If intraparenchymal hemorrhage is detected, as in 15% of all strokes, the imaging evaluation in the acute phase may include CT angiography (CTA) of the intracranial arteries for evaluation of an underlying vascular malformation (i.e. aneurysm and AVM).

MRI

The primary goals of imaging evaluation in the acute phase (0 to 4.5 hour time window) of a suspected stroke patient are to exclude the presence of intracranial hemorrhage and assess the presence and extent of ischemic changes. As "time is brain", imaging should not delay administration of IV thrombolytic if indicated. In our institution, a comprehensive and fast (≤ 15 mins) MRI protocol is followed to prevent these delays in diagnosis and management.

Parenchymal imaging: sequences directed to identify parenchymal pathologies such as areas of irreversible infarcted core in ischemic stroke, presence of hemorrhage or edema, white matter changes and mass lesions.
DWI/ADC (axial and coronal)

- Diffusion weighted imaging (DWI) is a MR sequence based on the measurement of random Brownian motion of water molecules within a voxel of tissue. Impedance of water molecules diffusion can be assessed quantitatively using the apparent diffusion coefficient (ADC) value.
- High tissue cellularity or those with cellular swelling exhibit lower diffusion coefficients, hence, these sequences are particularly useful in tumor characterization and cerebral ischemia.

T2WI/FLAIR (axial)

- T2WI: pathologic processes increase the water content in tissues, rendering high signal changes are noted on T2WI (i.e. tumors, edema, ischemia, gliosis, demyelination).
- FLAIR (Fluid Attenuation Inversion Recovery) is a MR sequence that removes the signal effects of CSF fluid from the resulting image. This sequence is particularly useful in the detection of subtle white matter changes/edema. FLAIR is also useful in the demonstration and aging of a region of infarction and shows changes related to ischemia earlier than T2W sequence. FLAIR is also the most sensitive MR sequence for the detection of subarachnoid hemorrhage.

GRE: Gradient Recalled Echo

- Bleeding breakdown products due to its large paramagnetic effect result in signal loss on GRE sequences (blooming, darkening). This sequence is helpful in identifying blood breakdown product. Other components that produce GRE susceptibility due to large paramagnetic effect includes calcification and iron deposition.

Vascular imaging:

MRA (MR Angiography)

Part of the workup of patients with suspected acute ischemic stroke is the imaging of both the intracranial and extracranial/neck vasculature to detect the site of arterial disease. This is crucial in determining the subsequent management.

Distinct cases will be discussed briefly on remaining part of this article, which presented to the emergency room as non traumatic neuro emergencies encoded with brain attack protocol. These are broadly representative cases for stroke, central nervous system infections, inflammation/demyelination and tumors.
CEREBROVASCULAR DISEASE OR STROKE

Stroke remains a leading cause of mortality and morbidity worldwide. Of all strokes, 87% are ischemic and 13% are hemorrhagic. The primary goal of imaging in patients with acute stroke symptoms is to distinguish between hemorrhagic and ischemic stroke.

(Case no. 1)

ISCHEMIC STROKE:

Deprivation of oxygen and glucose to the brain due to interruption of blood flow through an intracranial artery leads to a cascade of events that may lead to cell death. Patients with ischemic infarction usually presents with rapid onset neurological deficit that is determined by the area of brain involved.

Imaging Features:

Non contrast CT-scan (NCCT):

The limitation of CT in assessment of ischemic stroke is its low sensitivity in the hyper acute and acute setting. The role of plain CT in this early setting is to exclude intracranial hemorrhage in patients who are candidates for thrombolysis.

Early signs of ischemic infarction on NCCT includes:

- "Hyper dense vessel sign": the vessels may appear dense due to blood clots or thrombus.
- Loss of gray-white differentiation and hypoattenuation of the deep nuclei as well as cortical hypodensity with sulcal effacement.

MRI:

MRI has superior sensitivity and specificity in the diagnosis of acute ischemic infarction. Correlation of signal changes in different sequences also serves to age the infarction which has implications in the management and course of the disease (Fig. 2 on page ). This modality also has higher sensitivity and specificity than CT in the detection of "stroke mimics" such as cerebral edema, vascular malformations, neoplasm, infections, inflammatory diseases, and toxicometabolic disorders.
Diffusion-Weighted Imaging (DWI)/ ADC:

Restricted diffusion seen as DWI bright signal areas with corresponding signal drop on ADC may be seen within minutes following the onset of ischemia.

T2-weighted imaging and FLAIR:

Less sensitive than DWI in the first few hours to parenchymal change.
T2W/FLAIR high signal is exhibited by the infarcted tissue after 6-12 hrs.

Gradient Recall Echo (GRE):

Detection of intracranial hemorrhage/hemorrhagic conversion of infarcted areas.

CT angiography/ MR angiography:

Identification of vascular stenosis/occlusion, thrombus, aneurysm or dissection and vascular malformations.

(Case no. 2)

CEREBRAL VENOUS THROMBOSIS

Cerebral venous thrombosis is rare and accounts for 0.5 to 1 % of total stroke cases and commonly seen among young adults.

Risk factors:

Pregnancy, puerperium, trauma, surgery, coagulopathies, infection, inflammatory, cancers and many others.

Clinical Features
Non-specific and secondary to increased intracranial pressure (20-40 %) or stroke like features. The common symptoms are headache, seizure, vomiting, blurring of vision, isolated change in mental status, sixth nerve palsy, hemiparesis or aphasia.

**ANATOMY**

Brain and meningeal veins are divided into two groups (Fig. 3 on page 27):

1. Dural sinuses
2. Cerebral veins- Cerebral veins are further classified as Superficial and Deep veins

*What really happens in the brain?*

Initially intraluminal thrombus causes increased venous pressure leading to disruption of the blood brain barrier and vasogenic cerebral edema. These changes may be reversible in the presence of adequate venous collaterals, however lack of these collaterals leads to further increase in intracerebral pressure and intravascular pressure predisposing to diminished arterial perfusion with ischemia and cytotoxic edema. Persistent of these ischemic changes leads to cell death or hemorrhage and are irreversible. Both forms of cerebral edema may be seen with venous thrombosis.

Anatomical location - commonly involves the superficial venous system where superior sagittal sinus is most commonly involved, where as deep venous system accounts for 16 % of the cases.

**Imaging Features:**

*Non contrast CT-scan:*

Findings mainly relies on direct visualization of the thrombus within the sinus.

- *Hyperdense sinus*- Thrombus within the sinus.
- *Cord sign*- Hyperdense thrombus within the transverse sinus.
- *Filled delta sign*- Hyperdense thrombus within the posterior superior sagittal sinus.

(High index of suspicion should be made when infarction is seen with hemorrhagic components but without arterial territorial distribution.)
Other findings on non contrast CT are subarachnoid hemorrhage, rare and occurs in 0.5 to 0.8 % of the patients. Likewise intracranial hemorrhage is noted in approximately 30 to 40 % cases.

**Contrast enhanced CT scan:**

- 'Empty delta sign’ or filing defect on the posterior sagittal sinus seen.
- Direct visualization of thrombus: Enhancement of the dura with filling defect of the affected sinus.

(These CT findings may not appear for initial days but persists for days to weeks.)

**MRI:**

Greater sensitivity than CT-scan for detecting thrombosis and brain parenchymal changes.

Non specific edema is noted and the location may help in identifying the involved vessels.

- Frontal, parietal and temporal lobes - superior sagittal sinus
- Temporal lobe - transverse and sigmoid sinus
- Deep parenchymal involvement including the thalami - vein of galen or straight sinus.

**Time Of Flight (TOF) MRV:** most commonly used method for evaluation of thrombosis. However, contrast enhanced CT or MR venogram is required for confirmatory diagnosis.

**DWI/ADC:** DWI restriction with signal drop out on ADC was also noted in acute thrombus (40 % of cases).

*(Case no. 3)*

**POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME (PRES)**

**PRES** is a neurotoxic state with unique pattern on imaging and is associated with number of complex clinical conditions. Initially, described for involvement of the posterior cerebral lobes, hence coined as PRES, however other regions of the brain are also involved. The parietal and occipital lobes are most commonly involved, followed by frontal lobes, inferior temporal-occipital junction and cerebellum. Infrequently brain Stem and basal ganglia are also involved.
Risk factors:

Preeclampsia and eclampsia, autoimmune disease, renal disease, cancer chemotherapy, transplantation, hypertension and sepsis.

Clinical presentation:

Headache, seizure, visual disturbance, paresis, hemianopsia, nausea and altered mental status.

Etiology- two theories are described

1. Failure in auto-regulatory mechanism secondary to severe hypertension leads to hyperperfusion and endothelial damage and subsequent vasogenic edema.

2. Vasoconstriction and hypo perfusion leads to brain ischemia and subsequent vasogenic edema.

Imaging features:

Typically vasogenic edema is noted at different locations and is usually symmetric and reversible on follow-up imaging.

- T1 - hypointense,
- T2 and FLAIR - hyperintense,
- DWI - usually normal,
- ADC- increased signal.

Rarely, hemorrhage may be noticed as GRE susceptible focus.

Post contrast- contrast enhancement of the adjacent cortical vessels may be seen.

Three different patterns of involvement of the brain parenchyma are described:

1. Domina NT parietal-occipital pattern: Parietal and occipital cortex and white matter with or without involvement of the temporal lobes.
2. **Holohemispheric watershed pattern:** Typically involves at the watershed zones as linear pattern of vasogenic edema involving the frontal, parietal, occipital lobes and less frequently temporal lobes.

3. **Superior frontal sulcus pattern:** Linear pattern along the mid to posterior aspect of the superior frontal sulcus.

**Partial or asymmetric expression of the primary pattern:** Incomplete involvement of the above described patterns is also noted and may be either partial or asymmetric expression or combined (partial and asymmetric) expression.

**HEMORRHAGIC STROKE**

Intracranial hemorrhage can be subdivided into two distinct types based on the site and origin of blood: subarachnoid and intracerebral hemorrhage. Vascular anomalies are noted at younger patients presenting as hemorrhagic stroke. These anomalies are broadly classified as slow flowing and high flowing. Developmental venous anomaly also known as venous angioma and cavernoma are slow flowing anomalies whereas, fistula and arteriovenous malformations are high flowing vascular anomalies. CT or MR angiogram helps to identify the exact veins draining and arteries supplying the lesion.

**Imaging features:**

*Non contrast CT-scan:*

Acute blood appears as areas of high attenuation/hyperdensities relative to the brain parenchyma.

*MRI:*

MRI is usually requested if an underlying abnormality is suspected to be the cause of intracerebral hemorrhage like vascular anomalies, tumors and infections.

*(Case no. 4)*

**CEREBRAL ARTERIOVENOUS MALFORMATIONS**
Cerebral AVM's are abnormal vascular connections in the brain, which may either be pial or dural, depending on the location of the shunt. Classical or pial AVMs are characterized by a tangle of abnormal vessels located in the brain parenchyma called the *nidus*, which is the transition between the abnormally communicating arteries and veins. AVM are thought to be congenital lesions.

**Imaging features:**

*CT /MR angiography:*

The feeding arteries, nidus and draining veins appear like "bag of worms" after administration of contrast and image rendering. Demonstration of weak points in the angioarchitectural framework of the intracranial vessels (i.e. intranidal aneurysms, venous ectasia, venous stenosis) by imaging helps in assessing the risk of future hemorrhage.

*Non contrast CT-scan:*

The role of non-contrast CT is to demonstrate possible complications of the AVM such as acute hemorrhage.

*Cranial MRI:*

Fast and turbulent flow in the abnormal vascular connections produce signal voids that are easily seen on T2 weighted images. Complications including previous hemorrhage and adjacent edema are likewise demonstrated in Cranial MRI.

*(Case no. 5)*

**CEREBRAL ANEURYSM**

Cerebral aneurysm is an abnormal focal dilatation of cerebral artery with attenuation of the vessel wall. It is seen in 3-5 % of the general population.

**Pathogenesis:**
The major cause of aneurysm is age related degenerative changes, however, hereditary conditions like polycystic kidney disease, Ehlers-Danlos syndrome among may others bear increased risk of aneurysm formation. Other risk factors are hypertension, diabetes, smoking and female sex. Mycotic aneurysms are secondary to infectious etiology.

Classification:

according to the size of the aneurysm:

- Small \( \leq 5 \text{ mm} \),
- medium 5 to < 15 mm,
- large 15 to < 25 mm,and
- giant \( \geq 25 \text{ mm} \).

according to the size of the neck of aneurysm:

- Small neck aneurysms : neck size < 4 mm and
- large neck aneurysms : neck size > 4 mm.

Morphologically:

- saccular (berry aneurysm),
- fusiform, and
- blood blister like aneurysm.

Saccular aneurysm is the most common form.

Clinical features:

Unruptured small aneurysms are mostly asymptomatic. Larger aneurysms may exert mass effect to the adjacent structures like compression of the cranial nerves causing cranial nerve palsies (particularly third nerve), or to the brain parenchyma leading to seizure, motor or sensory deficit.

Complications:

Majority of cerebral aneurysm remains stable without complications. Approximately 0.25 to 0.95 % of the aneurysm will rupture. Ruptured aneurysm will further complicate to cerebral hematoma, ventricular hemorrhage, hydrocephalus, vasospasm and infarction (most common cause for morbidity and mortality), brain herniation and re-bleeding.

Risk factors for rupture:
1. **Size:** Larger the size of aneurysm (usually > 7 mm) greater the risk for rupture. Giant aneurysm (> 25 mm) has the highest risk of rupture and approximately, 33.4 %.

2. **Location:** Aneurysm arising from posterior and anterior communicating arteries.

3. **Aneurysm with daughter sac or multi-lobed aneurysm is prone to rupture.**

**Imaging Features:**

CT Angiography is the primary method for evaluation of aneurysm.

**Non contrast CT scan:**

Unruptured aneurysm may be demonstrated as relatively hyperdense focal bulge along the course of the cerebral vessels.

Ruptured aneurysm presents with subarachnoid hemorrhage and location of pool of blood may aid in identification of the involved vessel.

- Interhemispheric fissure - anterior communicating aneurysm.
- Sylvian fissure and temporal lobe hematoma - middle cerebral artery aneurysm.
- Diffuse within the cisterns - Posterior communicating aneurysm or aneurysm from the posterior circulation.

**CT Angiography:**

It is the best method for identification of aneurysm. MR angiography has almost similar sensitivity and specificity.

**Digital Subtraction angiography:**

Gold standard and performed for all preoperative assessment. It is particularly identifying peripheral small vessel aneurysm.

The following are to be reported in an Angiography reports. (Fig. 4 on page 26)

- Site of aneurysm.
- Size of the aneurysm - described in three dimensions (length, width/dome and height) and
- Size of the neck of aneurysm.
(Case no. 6)

MYCOTIC ANEURYSM

Infectious aneurysm or mycotic aneurysm are uncommon and represents approximately 2-5% cases of total intracerebral aneurysm.

Causative agents: Bacterial infections particularly by streptococcus and staphylococcus are common whereas, fungal infections are rare.

Risk factors:

Infective endocarditis, septicemia, intravenous drug abuse, immunosupression, particularly HIV AIDS and rheumatic heart disease. Reports show 1-10% of patients with infective endocarditis have mycotic aneurysm and approximately 65% of patients with mycotic aneurysm have infective endocarditis.

Clinical features:

Headache, focal neurological deficit, fever.

Imaging features:

Plain CT-scan:

Subarachnoid hemorrhage, infarct or intraparenchymal hemorrhage.

CT Angiogram:
Variable size of aneurysm (1 to 15 mm) and usually located at the distal middle cerebral artery branches. More than sixty percent of the aneurysm is ruptured at initial presentation.

**CNS INFECTION**

Majority of the central nervous system infections are secondary to bacteria, virus, fungus or protozoas. The diagnosis relies on clinical findings, laboratory tests, particularly CSF analysis and imaging manifestations. Prompt diagnosis is crucial for management of the disease. Acute changes secondary to the mass effect (exhibited by various forms of infection) and stroke like features (secondary to vasculitis) are the major complications.

The detailed discussion of every forms of infection is beyond the scope of this article. Cerebral manifestations of Mycobacterium tuberculosis and dengue virus infection will be discussed.

*(Case no. 7)*

**CNS TUBERCULOSIS:**

Mycobacterium tuberculosis typically affect the lungs, however extra-pulmonary involvement is also noted. CNS (Central Nervous System) tuberculosis accounts for 2-5 % of the total cases and approximately 10% of the patients with HIV.

**Pathogenesis**

- Hematogenous transmission from the lungs or other sites like gastrointestinal tract or lymph nodes to the brain occurs.
- “Rich focus”: Initial focus of infection at the brain, similar to Ghon’s focus in the lungs.

**Various pattern of involvement to the central nervous system are noted and as follows**

1. **Tubercular Meningitis**: Meningitis is secondary to either rupture of the rich focus in the brain into the subarachnoid space or direct dissemination of the tubercular disease to the meninges from distant site.
2. **Vasculitis and infarction**: Exudates along the cisterns causes panarteritis and leads to thrombosis and occlusion of the vessels predominantly, the small sized arteries, particularly the lenticulo striate and thalamoperforating arteries. This causes infarction at the region of basal ganglia.
3. **Tuberculoma**: Most common parenchymal form.
4. **Tubercular abscess**: Common at the gray-white matter junction.
5. **Hydrocephalus**: Common in early stage of the illness.

Both communicating as well as non-communicating hydrocephalus is noted with tubercular infection. Exudates at the basal cisterns obstructs the CSF flow causing communicating hydrocephalus, whereas obstruction to the flow by tubercular abscess or tuberculoma at the ventricular system, leads to non-communicating hydrocephalus.

**IMAGING FEATURES**

*Plain CT:*

- **Hyper dense basal cisterns** is described as highly specific feature of tubercular meningitis.
- Tuberculoma or tubercular abscess appears as hypo or hyper dense areas with surrounding vasogenic edema, however edema in abscess is relatively less extensive as compared to pyogenic abscess.
- Cerebral ischemia or infarct are demonstrated as areas of hypo densities, predominantly at the basal ganglia.
- Cerebritis is demonstrated as non specific areas of edema without adjacent tuberculoma or abscess.

*Post contrast CT/MRI:*

- Nodular and thick enhancement of the basal cisterns, predominantly along the location of M1 segment of middle cerebral artery and at the sylvian fissure. The remaining meninges also demonstrates nodular enhancement.
- Tuberculoma are demonstrated as well defined varying size lesion with either diffuse enhancement or peripheral rim enhancement.
- **Target sign**: Central area of calcification or hyperdensity in a peripherally enhancing may be seen in tuberculoma, however not specific.
- Tubercular abscess is uncommon and appears as peripherally enhancing hypo dense lesion with surrounding edema. Liquefied caseation of tuberculoma may have similar imaging appearance like tubercular abscess, rendering them indistinguishable. Thus, histopathologic confirmation is required.

*MR spectroscopy:*

- **Elevated lipid peaks** due to high lipid content of the mycolic acid at the tuberculcar cell wall.
- MR spectroscopy for pyogenic abscess demonstrates amino acid resonance at 0.9 ppm, which is not seen in tubercular abscess.
CEREBRAL MANIFESTATIONS OF DENGUE VIRUS INFECTION:

Dengue is second most common mosquito borne disease following malaria which is caused by single stranded RNA, *Flavi virus*. The disease is endemic in certain regions of Asia, central and south America, whereas not commonly seen in the European countries.

Pathogenesis

Neurological manifestations are secondary to liver failure, cerebral hypoperfusion, electrolyte imbalance, cerebral edema, vasculopathy and leak with subsequent hemorrhage or infarction.

Clinical Features

Fever, headache, retro-orbital pain, abdominal and joint pain, vomiting, rashes, epistaxis, GI bleeding and finally leading to shock syndrome.

WHO classifies dengue infection as:

- dengue without warning signs
- dengue with warning signs
- severe dengue.

(Central nervous system (CNS) manifestations of dengue is categorized as severe dengue.)

Neurological manifestation in dengue infection ranges from 0.5 to 5 % and presents with diverse clinical conditions in the form of encephalopathy, encephalitis, cerebrovascular disease, neuromuscular dysfunction and neuro-ophthalmic disorders. Brief imaging features of the cerebral manifestations will be discussed.

*Encephalitis*: Direct invasion of the dengue virus to the brain is presumed to cause encephalitis. No specific imaging findings has been described in the literature. Few case reports described non-specific areas of edema or focal hemorrhage. Encephalitis is a pathological diagnosis and confirmed only by histopathology.
Encephalopathy: Most common cerebral manifestation and is multifactorial in etiology as described above. The imaging features are again non-specific and demonstrates areas of edema, venous congestion or hemorrhage.

Cerebrovascular disease: Platelet dysfunction leading to thrombocytopenia causes hemorrhage with dengue infection. Likewise, Dengue associated coagulopathy and intravascular thrombosis leads to ischemia or infarction.

Imaging Findings: Hemorrhage either parenchymal or extra-axial, infarction and edema with non-specific pattern and location.

Other neurologic manifestations are Acute Disseminated Encephalomyelitis, Guillain-Barre syndrome and mononeuropathies.

Major differential diagnosis: Japanese Encephalitis and confirmed by specific laboratory analysis.

(Case no. 9)

MULTIPLE SCLEROSIS

Multiple sclerosis is one of the most common chronic inflammatory demyelinating diseases mostly seen among 30 to 40 age group females.

Pathogenesis:

The exact etiology is not known, however believed to be auto immune process with various stages of evolution from early-acute, sub-acute to chronic stages.

Clinical Features:

Non-specific and may range from sensory, motor and autonomic dysfunction to cranial nerves involvement.

Multiple sclerosis is further classified clinically depending on the presentation and course of the disease as:
1. Clinically Isolated Syndrome (CIS): Presents as isolated involvement of the brain stem, optic nerve or spinal cord.

2. Relapsing and remitting disease: Most common form, disease evolves, stabilizes and often improves clinically.

3. Secondary progressive disease: Symptoms will relapse and the disease may be progressive in between.

4. Primary progressive disease: Less common, in 15% of cases where there is steady progression of the disease from its onset without evidence of relapses.

**Imaging Features:**

**CT-scan:**

Limited role on the diagnosis and demonstrates non-specific area of hypo densities or even appears normal without any changes.

**MRI:**

MRI along with clinical symptoms is a key to the diagnosis and follow up monitoring of multiple sclerosis, as described on the Mc Donald's Criteria.

- Morphologically, lesions may be linear, ovoid, round or mass like typically at the periventricular white matter parallel to lateral ventricles, corpus callosum, calloso-septal interface, subcortical regions, U fibers, brain stem, spinal cord or cranial the optic nerves.
- Spinal cord lesions are short segment (3 or less segments) and typically located dorsolaterally

**T1W:**

Iso to hypointense. "Black holes" may be seen and represents myelin destruction

**T2 and FLAIR:**

- Hyper intense signals at the locations as described above.
- *Ependymal dot dash sign*: early changes represents as small hyper intense foci at the calloso-septal interface.
- "*Dawson fingers*" - abnormal signals radiating outwards perpendicular to the lateral ventricles, best visualized at parasagittal views.

*Post contrast MRI:*
• Active plaque may enhance and persists for 2-6 wks.
• Peripheral incomplete ring like enhancement also known as "open ring sign" is a common feature. However, solid or nodular enhancement is also seen.

_DWI and ADC_ acute lesions may show restricted diffusion.

**MR spectroscopy:**

Reduced NAA, sign of neuronal loss or degradation and elevated choline, secondary to glial turnover is noted. These findings are non-specific and even seen in glial tumors.

**MR Perfusion:**

Low CBV and CBF is noted and helps to distinguish it from neoplasm which in contradictory demonstrate increase in CBV and CBF

**Diagnosis**

Mc Donald's criteria should be followed. It describes Clinical features, MR imaging characters and positive CSF examination (oligoclonal bands and elevated IGg index).

**MR imaging descriptors for Mc Donald's criteria:**

• _Dissemination in time:_ Simultaneous presence of asymptomatic gadolinium-enhanced and nonenhanced lesions at any time, or a new T2 and/or gadolinium-enhancing and non enhancing lesion(s) on follow up MR images irrespective of timing of baseline study.
• _Dissemination in space:_ One or more lesion in atleast two 2 of 4 MS typical region of the CNS (periventricular, juxtacortical, posterior fossa, spinal cord).

Differential Diagnosis:

Neuromyelitis optica, Acute Disseminated Encephalomyelitis (ADEM), CADASIL, infections like Lyme disease, small vessel ischemic disease.

*(Case no. 10)*

**NEUROMYELITIS OPTICA(NMO) or DEVIC's DISEASE**
NMO is an autoimmune demyelinating disease induced by specific antibody NMO-IgG, targeted against water channel aquaporin 4 (AQP4) antigen. The highest concentration of AQP4 is around the ventricles. NMO has female predilection, similar to multiple sclerosis. Initially it was presumed to be one of the variant of Multiple Sclerosis (MS), but currently described as a distinct clinical entity. The prompt identification of the disease is crucial since it carries poor prognosis and has relatively aggressive management as compared to MS.

**Diagnosis**

Diagnostic criteria for the diagnosis of NMO

| • Optic Neuritis  |
| • Acute myelitis  |
| • Atleast 2 of the following 3 supportive criteria: |

1. Contiguous spinal cord MRI lesion extending over at least 3 vertebral segments.
2. Onset brain MRI findings not meeting the diagnostic criteria for MS.
3. NMO-IgG seropositivity status.

**Imaging features**

- **MR imaging of the spinal cord:** T1 hypointense and T2 hyper intense long segment (greater than 3 vertebral segments) signals preferably involving the gray matter. Slight cord expansion with enhancement on post contrast studies may be seen.
- **MR imaging of the optic nerve:** Long segment involvement of the optic nerve with expansion and enhancement, preferably bilateral and extending beyond the optic chiasm.
- **MR imaging of the brain:** The lesions are typically located at the region with high concentration of AQP4, particularly periependymal region surrounding the third ventricle, cerebral aqueduct and fourth ventricle.

**Post Contrast MRI:**

- Demonstrates no discrete contrast enhancement, however, enhancement with blurred margins described as "cloud like enhancement" is relatively specific for NMO.
- "Pencil thin enhancement" of the ependymal lining is also noted with NMO, not seen in MS.

**MRS:**
Normal without demonstrable abnormal metabolites.

**Diffusion Tensor Imaging:**

Normal appearing white matter, however DTI parameters shows axonal loss and demyelination.

Few important notes regarding *NMO*

1. Periependymal/periventricular lesions adjacent to the lateral ventricles are common. However, unlike MS, the radially oriented Dawson’s finger like pattern is not common.
2. Large confluent white matter lesions are also noted and sometimes with involvement of the corpus callosum. These are usually not associated with mass effect and vanish on steroid treatment.
3. Unilateral or bilateral lesions longitudinally along the cortico-spinal tract is seen in Korean population.
4. Unlike MS, Cortical grey matter involvement is not a feature of NMO.

**Differential Diagnosis**

Multiple Sclerosis, ADEM, Spinal cord infection/ intramedullary neoplasm, Sarcoidosis.

*(Case no. 11)*

**MOYA MOYA DISEASE**

Moya moya disease is an idiopathic non-inflammatory cerebrovascular condition predisposing patients to stroke with progressive narrowing of the vessels at the circle of Willis, predominantly the intracranial internal carotid arteries and its proximal branches.

- *Moya moya syndrome* - moya moya vasculopathy with well recognized associated clinical conditions.
- *Moya moya disease* - moya moya vasculopathy without associated risk factors.
- *Moya moya phenomenon or pattern* - known clinical entities with imaging appearance similar to moya moya disease.

**Demography**
• Bimodal distribution (children usually 5 yrs of age and adults at mid 40s)
• Twice as common in females than males.
• Relatively more common in Asian population, however, even seen among other ethnic groups.

Pathogenesis

Both genetic and acquired condition are described as the probable etiology of the disease. Smooth muscle hyperplasia and luminal thrombosis is responsible for vessel narrowing. There is no associated atherosclerosis or inflammatory changes. There is concentric stenosis of the bilateral internal carotid arteries and its proximal branches. Less commonly posterior cerebral vessels are also involved.

Clinical Features

• Stroke like presentation in the form of transient ischemic attack, infarct or hemorrhage, seizure or headache are common symptoms.
• Hemorrhage is usually seen in adults.
• Ischemia may be triggered by cry, cough or exercise among pediatric age group.

Clinical conditions associated with Moya Moya syndrome are Down syndrome, NF1, cardiac anomaly, renal artery stenosis, cervico fascial hemangiomas and hyperthyroidism.

Imaging Features:

CT-scan:

• Hypodense areas, suggesting infarcts (depending on the vessels involved.
• Cerebral atrophy, demonstrated as prominence of the cerebral sulci, cisterns and ventricles
• Hemorrhage (usually in adults)

MRI:

• Infarcts: usually on the watershed zones. Although the disease involves large cerebral vessels, extensive territorial infarctions are uncommon.
• Ivy sign: Flair and post contrast studies demonstrate linear pattern of increased signal in the leptomeninges and perivascular spaces which represents retrograde engorged pial vessels from leptomeningeal anastomosis.

MR OR CT Angiogram:
Diagnostic angiographic criteria:

Occlusion or stenosis of the distal internal carotid arteries and proximal part of the anterior and middle cerebral arteries as well as presence of collateral vessels at the base of the brain, without causal disease.

- Parenchymal collaterals are noted along the course of lenticulo striate arteries and thalamostriate arteries, transdural collaterals from the external carotid artery and leptomeningeal collaterals from the posterior cerebral artery supplying the ischemic brain are also demonstrated

Conventional Angiogram/Digital Substraction Angiography

Gold standard for the diagnosis, however, is invasive with risk of complications.

- “Puff of smoke appearance”: Moya moya disease originally described in Japan was classically described for its angiographic appearance of collateral vessels forming "puff of smoke" like appearance. These findings can even be identified on MR or CT angiograms, however small vessels are better visualized on conventional angiograms.

MR/CT perfusion:

- Decreased perfusion in ischemic areas.
- Suzuki staging is used for the assessment of changes related to Moya moya disease.

(Case no. 12,13 & 14)

BRAIN TUMORS

The world wide incidence of primary malignant brain tumor is 3.4 per 100,000 persons. Patients with brain tumors are predisposed to the following emergency complications.

- **Brain herniation**: Any form of brain herniation can occur and includes subfalcine, uncal, ascending and descending transtentorial herniation. Vital centers at the brainstem may be compressed and are fatal.

- **Cerebrovascular disease**: Individuals with brain tumors are in immuno-compromised state with increased risk of infection and also in
hypercoagulable state. These predisposes the patient to hemorrhage or infarction. Likewise, increased risk of sinus thrombosis with direct tumor invasion and direct compression of the vessels also predisposes the patient to cerebrovascular accidents.

- *Post treatment toxicity/changes*: Chemotherapeutic agents and post radiation effects may lead to inflammatory changes in the brain or narrowing of the cerebral vessels (post radiation moya phenomenon).

Few key points for characterizing brain tumors are described in Fig. 5 on page 28 & Fig. 6 on page 29.
Images for this section:

**Fig. 1: BRAIN ATTACK PROTOCOL**

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Fig. 4: Location of aneurysm: 1- middle cerebral artery (proximal or distal to bifurcation), 2- carotid terminus, 3- anterior choroidal, 4- superior hypophyseal, 5- anterior communicating, 6- posterior communicating, 7- ophthalmic, 8- basilar terminus, 9- superior cerebellar, 10- V4 segment, vertebral, 11- posterior inferior cerebellar, 12- pericallosal.

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Fig. 3: MR venogram (MRV) demonstrating normal venous anatomy: 
a. Internal cerebral vein 
b. Vein of Labbe 
c. Vein of Galen 
d. Straight sinus 
e. Vein of Trolard 
f. Superior sagittal sinus 
g. Sigmoid sinus 
h. Transverse sinus 
i. Jugular bulb

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**BRAIN TUMOR FACTS**

Incidence: one third metastatic, one third gliomas and one third non glial tumors

Age and location:  
Supratentorial  
Pediatric: astrocytoma, pleomorphic xanthoastrocytoma, PNET, DNET, ganglioglioma  
Infratentorial: pilocytic astrocytoma, medulloblastoma, ependymoma, brainstem glioma

Adults: Metastasis is the most common supratentorial and infratentorial tumors; meningioblastoma is the most common infratentorial primary brain tumor.

Pineal region: pineocytoma, germ cell tumors, PNET, germinoma, meningioma, dermoid, arachnoid cyst

Intraventricular: ependymoma, subependymoma, choroid plexus papilloma, central neurocytoma, colloid cyst, meningioma (most common in adult)

CP angle tumors: schwannoma, meningioma, epidermoid, arachnoid cyst, paraganglioma, metastasis

Sellar and parasellar: pituitary adenoma, craniopharyngioma, meningioma, Rathke's cyst, chiasmatic glioma, dermoid, epidermoid, germinoma, schwannoma, metastasis

Skull base: chordoma, chondrosarcoma, esthesioneuroblastoma, lymphoma, metastases, myeloma, paraganglioma, sinonasal carcinoma

Midline crossing tumors: GBM, lymphoma, metastases, meningioma, epidermoid tumors

Calcified tumors: most common intra axial tumor with calcification is oligodendroglioma (90%). Other tumors with calcifications are astrocytoma (20%), meningioma (25%), craniopharyngiomas (90%), ependymoma (50%), choroid plexus papilloma (25%), ganglioglioma (40%)

Hemorrhagic tumors: Common primary tumors are glioblastoma, pituitary adenoma, ependymoma, central neurocytoma, choroid plexus carcinoma, pilocytic astrocytoma. Tumors with hemorrhagic metastases are melanoma, renal cell carcinoma, choriocarcinoma, thyroid malignancy, breast and lung carcinoma.

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**Fig. 5:** Features of brain tumor

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**MRI features of Importance for Brain Tumors**

- Highly cellular tumors with DWI restriction: Lymphoma, meningioma, PNET, germinoma, GBM, oligodendroglioma

- Radiation necrosis versus recurrent tumor: Increased cerebral blood volume (CBV) is the most sensitive findings to suggest recurrent tumor against radiation necrosis, which has no increased blood volume.

**MR SPECTROSCOPY**

<table>
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<tr>
<th>Tumors</th>
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<th>Lipid</th>
<th>Myo</th>
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<td>Glioma, low grade</td>
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Fig. 6: Advanced MRI features of Brain tumors

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Findings and procedure details

The examiantions were all performed on either 16,64 or 256 slice CT scanner and 1.5T or 3.0 T MRI machines depending on the indication and availability.

Case no 1. (Fig. 7 on page 33)

60 yrs old male, Hypertensive with acute onset left-sided hemiparesis presented to the ER within 1 hour of the symptoms.

Case no 2. (Fig. 8 on page 33 & Fig. 9 on page 34)

28 yrs old female and 6 wks pregnant, presented with severe headache since one week, two episodes of seizure, left-sided weakness and loss of consciousness few hours prior to consult.

Case no 3. (Fig. 10 on page 35, Fig. 11 on page 36 & Fig. 12 on page 37)

48 yrs old female, known case of endometrial cancer and under chemo/radiotherapy presented with sudden onset of seizure, unresponsivness, blank stare and no verbral output.

Case no 4. (Fig. 13 on page 38)

32 yrs old male, with sudden onset of seizure and no significant past morbidities.

Case no 5. (Fig. 14 on page 39 & Fig. 15 on page 40)

60 yrs old hypertensive, with severe headache and loss of consciousness since last 2 hrs.

Case no 6. (Fig. 16 on page 41 & Fig. 17 on page 42)

29 yrs old female, known patient of rheumatic heart disease with 2D echo findings of infective endocardiastis, presented with sudden history of right-sided weakness and headache.
Case no 7. (Fig. 18 on page 43 & Fig. 19 on page 44)

5 yrs old boy, presented to the emergency room, with history of seizure, cough, fever and neck stiffness, few hrs prior to consult. Past history of tuberculosis in immediate family member.

Case no 8. (Fig. 20 on page 45, Fig. 21 on page 46, Fig. 22 on page 47 & Fig. 23 on page 48)

32 yrs old male, known case of dengue fever, presented with sudden onset of loss of consciousness, seizure and unresponsiveness.

Case no 9. (Fig. 24 on page 49 & Fig. 25 on page 50)

39 yrs old female, Known case of Multiple Sclerosis under treatment since 2 yrs, with sudden onset of headache, seizure and right-sided weakness. No other co-morbidities noted.

Case no 10. (Fig. 26 on page 50, Fig. 27 on page 51 & Fig. 28 on page 52)

20 yrs old female, with headache, blurring of vision on right eye, left upper and lower extremity weakness since last 6 hrs.

Case no 11. (Fig. 29 on page 53 & Fig. 30 on page 53)

23 yrs old female with sudden onset left sided weakness with similar episodes of weakness at the past. The evaluation for autoimmune and possible vasculitis was negative.

Cases no 11,12 and 13. (Fig. 31 on page 54)

Three individual cases with known primary malignancies, presenting to ER for sudden neurological deterioration.
Images for this section:

Fig. 7: DWI and ADC demonstrating acute right middle cerebral artery infarct. Time of Flight MRA sequence demonstrate initially occluded right middle cerebral artery with reperfusion following thrombectomy. There is significant regression of the DWI and ADC signals.

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**Fig. 8:** Axial DWI image demonstrate areas of restricted diffusion at the right central sulcus. Axial flair image demonstrates hyper intense signal suggestive of sub arachnoid hemorrhage at both central sulci. GRE susceptible foci were noted at the same region, suggestive of hematoma. Axial T2W images demonstrate increase signals suggestive of edema.

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Fig. 9: MR venogram demonstrates absence of normal flow at the superior sagittal sinus, suggestive of superior sagittal sinus thrombosis.

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**Fig. 10:** Axial FLAIR images demonstrate multiple hyper intense signals in the cortical-sub cortical both occipital, parietal and frontal lobes.

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Fig. 11: Axial DWI images (a & b) shows hyper intense signals but without corresponding low signal on ADC (c & d).

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Fig. 12: Comparative study with prior axial FLAIR study shows significant regression of abnormal signals at the frontal, parietal and occipital lobes, consistent with PRES.

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**Fig. 13:** T2W and FLAIR demonstrate prominent flow voids at the right frontal parasaggital region. A fluid collection with rim exhibiting GRE susceptibility is noted adjacent to the prominent flow voids. Findings are suggestive of a pial AVM with adjacent parenchymal hemorrhage.

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Fig. 14: Axial non contrast CT images demonstrates hyper densities outlining the particularly the basal cisterns and posterior fossa cisterns compatible with sub arachnoid hemorrhage.

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**Fig. 15:** Axial post contrast CTA (a) and 3D reformatted images of the circle of willis demonstrate a bilobed aneurysm arising at the tip of the basilar artery. The length is 0.6 cm, dome is 0.5 cm and neck is 0.6 cm.

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Fig. 16: Axial non contrast images of the brain demonstrates a lobar hemorrhage at the left temporal and parietal lobes with surrounding vasogenic edema. There is mass effect with effacement of the adjacent cortical sulci and left lateral ventricle.

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Fig. 17: MIP images (a), 3D reformatted (b) and coronal post contrast CTA of the circle of Willis demonstrates sacular aneurysm at the distal cortical branches of the left middle cerebral artery. The larger aneurysm appears irregular, suggestive of rupture.

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**Fig. 18:** Contrast enhanced CT scan of the brain demonstrates multiple enhancing nodules (blue arrows) at the right deep temporal lobe and pons. Ring enhancing nodules (red arrows) are noted at the right thalamus and left frontal lobe. White matter edema or ischemia (yellow arrow) is noted at the left frontal and temporal lobe. The lesions are concentrated along the perisylvian and basal cisterns.

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Fig. 19: Axial post contrast images demonstrate classic features of CNS tuberculosis with meningitis (yellow arrow), tuberculoma (blue and red arrow), cerebritis or ischemia (green arrow) and infarction (black arrow).

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Fig. 20: Axial DWI and ADC images demonstrating restricted diffusion with corresponding signal drop out on ADC, suggestive of acute infarcts at the right parietal lobe and smaller lacunar on the left frontal lobe. Other lacunar infarcts were also noted, not included in the images.

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Fig. 21: GRE susceptible focus at the left parietal lobe with surrounding edema suggestive of acute parenchymal hemorrhage. Similar focus was noted at adjacent to the infarct of the right parietal lobe. Subarachnoid hemorrhage is also noted on the left frontal region.

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Fig. 22: Axial plain CT images of the brain in between the interval of 6 hrs was done and demonstrates interval progression in the size of hematoma and surrounding edema at the left parietal lobe. Right parietal lobe infarct and left frontal subarachnoid hemorrhage is also noted.

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Fig. 23: Normal intracranial MRA and MRV

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**Fig. 24:** Sagittal and axial FLAIR images demonstrates classic radially oriented triangular Flair signals at the calloso-septal interface (Dawson’s finger like appearance indicated by blue arrows) and also at the periventricular white matter of both cerebral hemisphere, corpus callosum and cerebellum.

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**Fig. 25:** Same patient with classic features of multiple sclerosis develops interval appearance of an avidly enhancing mass at the left parietal lobe involving the splenium of the corpus callosum with perilesional edema. Interval regression of previously noted white matter signals are noted. The patient was diagnosed as multiple sclerosis following McDonald's criteria. The mass was biopsy proven lymphoma.

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Fig. 26: Axial FLAIR images demonstrate periependymal high intense signals surrounding the third ventricle (a), right peripheral pons, right cerebellar peduncle adjacent to the fourth ventricle and dorsal medulla (b and c)

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**Fig. 27:** Sagittal T2W (a) and post contrast (b) images of the cervical spine demonstrate extensive cervical cord expansile lesion with faint enhancement from C2 down to C5-C6 level in the same patient with abnormal signals in the brain and brain stem.

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**Fig. 28:** Axial T2W image demonstrates relatively enlarged intraorbital segment of the right optic nerve with peripherally increased signals. The normal appearing left optic nerve is also noted in the same patient. Laboratory findings were positive for NMO IgG antibody.

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**Fig. 29:** Axial DWI images demonstrate an area of restricted diffusion with corresponding low signal at ADC at the right frontal and parietal lobes. T2W image demonstrates a chronic infarct at the right parietal lobe. GRE images demonstrate no abnormalities.

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**Fig. 30:** Left vertebral (LVA) and internal carotid angiogram (LCA) typical "puff of smoke" like appearance. TOF MRA demonstrates narrowing of both internal carotid arteries (red arrows) and attenuated bilateral anterior and middle cerebral arteries (white arrows). Multiple collaterals are also noted.

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Fig. 31: Various forms of brain tumors with mass effect. Desmoplastic infantile ganglioglioma in an infant involving the right cerebral hemisphere with subfalcine and uncal herniation. Metastatic thyroid carcinoma with hemorrhage causing mass effect to the pons. Primary CNS lymphoma with subfalcine herniation.

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Conclusion

Stroke represents the majority of non-traumatic neuro emergency, however, several other clinical conditions like infection, inflammation, demyelination or tumors present with similar clinical features as stroke. Conventional as well as advanced neuro imaging plays a vital role in timely diagnosis of the disease and subsequent management. Radiologists should be well knowledge about the imaging features of stroke and non-stroke related diseases presenting with acute neurological symptoms.
References

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16. Traboulsee et al. Revised Recommendations of the Consortium of MS Centers Task Force for a standardized MRI Protocol and Clinical


