Spectrum of imaging findings of brain in neurological disorders in patients with end stage renal failure on hemodialysis

Poster No.: C-2507
Congress: ECR 2019
Type: Educational Exhibit
Authors: S. Shaikh, S. M. Faiq, A. Javed Qureshi, B. sheikh; Karachi/PK
Keywords: Ischaemia / Infarction, Education and training, Acute, Imaging sequences, Education, Complications, MR, CT, Neuroradiology brain, Kidney, Emergency
DOI: 10.26044/ecr2019/C-2507

Any information contained in this pdf file is automatically generated from digital material submitted to EPOS by third parties in the form of scientific presentations. References to any names, marks, products, or services of third parties or hypertext links to third-party sites or information are provided solely as a convenience to you and do not in any way constitute or imply ECR's endorsement, sponsorship or recommendation of the third party, information, product or service. ECR is not responsible for the content of these pages and does not make any representations regarding the content or accuracy of material in this file.

As per copyright regulations, any unauthorised use of the material or parts thereof as well as commercial reproduction or multiple distribution by any traditional or electronically based reproduction/publication method ist strictly prohibited.

You agree to defend, indemnify, and hold ECR harmless from and against any and all claims, damages, costs, and expenses, including attorneys' fees, arising from or related to your use of these pages.

Please note: Links to movies, ppt slideshows and any other multimedia files are not available in the pdf version of presentations.

www.myESR.org
Learning objectives

- To enlist the common and uncommon imaging findings in brain in renal failure patients on hemodialysis.
- To address basic risk factors of the disease process in this particular group of patients.
- To discuss the imaging features of individual diseases.
Background

Chronic Kidney Disease

Chronic kidney disease (CKD) is increasingly recognized as a major public health problem. It is indicated by decreased kidney function, abnormal albumin excretion or, quantified by estimated glomerular filtration rate that remains for more than three months. To promote evaluation of severity of CKD, the National Kidney Foundation developed criteria for its staging which is categorized into five stages of increasing severity.

Hemodialysis:

Hemodialysis is the routinely administered therapy for end-stage renal disease (ESRD) patients. Although hemodialysis technique has shown improvement in the last decades, mortality and morbidity of chronic hemodialysed patients is persistently high. These patients tend to develop neurological complications frequently which often remain undetermined and under treated. Cerebrovascular manifestations in hemodialysed patients develop for the following reasons:

1) Disease processes leading to end stage renal failure.
2) Secondary to Uremia.
3) Due to hemodialysis itself.

The introduction of dialysis lead to pronounced decrease in the incidence and severity of neurologic complications caused by uremia; however many of these are unaltered by dialysis. Early detection and diagnosis of cerebrovascular incidents is important for quality of life and prognosis in these patients.
Findings and procedure details

Overview:

Neurological complications developing in hemodialysis patients are multifactorial. Neurological examination alone is usually not adequate to determine the underlying pathologies. Imaging i.e. MRI and CT scan of the brain is the most valuable tool frequently required for evaluation of neurological complications in symptomatic patients. It is of great importance in not only evaluating the cause of neurological complains when the clinical assessment is inconclusive but also used for treatment response assessment. The imaging spectrum of this particular group of patients is infrequently studied. We will address cause/risk factors, clinical manifestations and imaging findings in this particular group of patients.

- CEREBRAL ISCHEMIA AND INFARCTION:

Risk Factors:

- The occurrence of stroke is considerably higher among hemodialysis patients. CKD greatly affects severity and outcome of both ischemic and hemorrhagic stroke.
- Traditional risk factors include hypertension, diabetes, hypercholesterolemia, anemia, atherosclerosis, thromboembolic disease and intradialytic hypotension.

Imaging Findings:

- Conventional MRI i.e. T2WI and FLAIR are usually normal for the first six hours of acute infarct. Afterwards an increase in intensity, swelling of gyri and effacement of sulci are noticed. DWI shows restriction with low value on ADC.
- CT may be normal for many days in small infarcts.
- Cerebral changes include acute infarction Fig. 1 on page 13 Fig. 2 on page 13 Fig. 3 on page 14 Fig. 4 on page 15, white matter changes Fig. 5 on page 16, and lacunar ischemic lesions. Chronic changes of infarction represented by encephalomalacia are often seen. Fig. 6 on page 17

- NON-TRAUMATIC INTRACRANIAL HEMORRHAGE:

Cause/Risk factors:

- ESRD patients are at increased risk of spontaneous intracerebral hemorrhage. The pathophysiology of bleeding diathesis is complex that
ranges from intrinsic platelet defects to uremic toxins, infections and anticoagulant therapy during dialysis.

Imaging Manifestation:

- Non traumatic cerebral hemorrhage may be intraparenchymal, intraventricular Fig. 7 on page 18 , subarachnoid Fig. 8 on page 19 and subdural Fig. 9 on page 20 in nature. Fig. 10 on page 21
- On CT scan, acute blood appears hyperdense with gradual decrease in its attenuation towards resolution.
- MRI can very well demonstrate location, evaluation and extension of intracerebral hemorrhage. It also helps in assessing its cause.

- NON-TRAUMATIC CEREBRAL MICROBLEEDS (CMBs):

Cause/ Risk factors:

- Chronic Kidney disease is considerably linked to the presence and number of CMB, as well as presence of CMB in deep locations.
- Hypertensive microangiopathy also plays an important role as a causative factor of CMBs in hemodialysis patients.

Clinical Manifestations:

CMB are clinically silent in general, but considered as a risk factor for future intracranial hemorrhage.

Imaging findings:

- Cerebral microbleeds are defined as small, round or oval homogeneous low signal intensity lesions in T2-weighted gradient-echo (GRE) MRI and susceptibility-weighted imaging (SWI).
- They are not commonly seen on conventional MRI or CT
- CMBs secondary to hypertensive microangiopathy are characteristically present in deeper locations i.e. pons, basal ganglia, cerebellar hemispheres. Fig. 11 on page 22
- This is the same distribution as hypertensive intracerebral hemorrhages.

- INTRACRANIAL INFECTIONS:

Cause /Risk factors:

- Dialysis techniques also introduce additional risk factors for infection. Patients having central venous access catheters are at the highest risk of access-related infection.
- Systemic opportunistic and non-opportunistic infections are also identified.
Organisms:

Opportunistic bacterial infections include pathogens as Nocardia, and Listeria monocytogenes. Fungi often constitute Aspergillus, Cryptococcus, Candida, Pneumocystis and Mucor species. **Mycobacterium tuberculosis** is a principal cause in endemic areas.

Clinical manifestation:

The usual signs of infection in immunosuppressed patients are often attenuated so early diagnosis is sometimes challenging.

Imaging Manifestations:

- Plain and contrast-enhanced MRI helps to illustrate the cerebral infections better than CT scan. Such complications include meningitis Fig. 12 on page 23, subdural empyema, cerebritis/abscess, ventriculitis Fig. 13 on page 24, Fig. 14 on page 25 Fig. 15 on page 26 Fig. 16 on page 27 Fig. 17 on page 28 venous and arterial infarcts, venous thrombosis, hydrocephalus, and cerebral edema.
- Secondary cerebral infections from the contiguous extension of orbital and sinus infections are also well depicted on imaging. Fig. 18 on page 29 Fig. 19 on page 30

- **CEREBRAL VENOUS SINUS THROMBOSIS (CVST):**

  Cause / risk factors:

  - Hypercoagulable events are also not uncommon among ESRD patients.
  - Infections are traditionally recognized as common cause of CVST.
  - In addition nontraditional risk factors for thrombosis, such as hyperhomocysteinemia, malnutrition, inflammation and endothelial dysfunction are present in a significant number of chronic dialysis patients.

  Clinical manifestation:

  Initial symptoms include a headache, raised intracranial pressure and seizures.

  Imaging findings:
- A non-contrast CT scan of head is usually the first diagnostic imaging assessment performed in an emergency setting. **Hyperdense thrombosed sinus** can be easily demonstrated.
- On conventional MR sequences Thrombus may manifest as **absence of normal flow void**.
- **Secondary Intraparenchymal hemorrhages** evolve through its traditional stages.
- Post Contrast imaging can show classic "**Empty Delta Sign**" Fig. 20 on page 31

- **OSMOTIC DEMYELINATION SYNDROME (ODS):**

  **Cause/ Risk factors:**
  - **Rapid correction of chronic hyponatremia** is a well-recognized cause.
  - Most patients with ESRD are subject to rapid osmotic fluctuations after hemodialysis which also attributes to ODS.

  **Clinical presentation:**
  
  Acute progressive quadriplegia, dysarthria, dysphagia followed by alteration of consciousness.

  **Distribution:**
  
  - **Central Pons** is the most common location.
  - **Extrapontine regions** including the midbrain, thalamus, basal nuclei and cerebellum can be affected as well.

  **Imaging findings:**
  
  - On MRI brain classic **"Trident shape appearance"** is found in central pons. Fig. 21 on page 32
  - Extrapontine regions also demonstrate similar **bilateral symmetric edema and demyelination**.

- **PROGRESSIVE REVERSIBLE ENCEPHALOPATHY SYNDROME (PRES):**

  **Cause/ Risk factors:**
  
  - PRES is often associated with an **abrupt rise in blood pressure** and it is a frequent finding in ESRD patients.
  - The uremic environment of ESRD has also been suggested as an independent precipitating agent.
  - The **reversibility** of this syndrome is well established.
Clinical presentation:

It includes severe headaches, nausea, vomiting, seizures and visual and mental changes attribute to increase blood pressure.

Imaging findings:

- It is characterized by distinctive radiological findings in the cortical and/or subcortical part of the brain supplied by posterior circulation, which may spread to basal ganglia, brain stem, and cerebellum.
- CT shows hypodensity in posterior brain while signals on MRI represent vasogenic edema. Fig. 22 on page 33
- Frank Intracranial hemorrhage and microhemorrhages is known to occur in PRES in approximately 15-64%.
- PROGRESSIVE MULTIFOCAL LEUKOENCEPHALOPATHY (PML):

Cause/ Risk factors:

PML is a demyelinating disease caused by the reactivation of JCV Polyomavirus and infection of oligodendrocytes. PML had been reported with the HIV patients with CD4 count less than < 200/uL. However profound immunosuppression is a notable cause and it has also been reported in few cases with ESRD patients on hemodialysis.

Clinical manifestation:

Insidious onset and steady progression of focal symptoms that include cognitive, motor, speech, behavioral, and visual impairment.

Imaging findings:

- There are white matter abnormalities that are multifocal, confluent, involve cortex and subcortical areas with invariable involvement of Sub-cortical U-fibers in bilateral asymmetrical distribution. Basal ganglia and cerebellum involvement has also been reported. Frontal and parieto occipital lobes are frequently involved. Absence of mass effect. Enhancement is not commonly observed.
- CT brain reveals hypodense lesions of the affected white matter.
- MRI is far more sensitive to the presence of the white matter lesions. MRI shows hyperintense lesions on T2-weighted images and FLAIR images in the affected regions. Hypointensity is seen on T1WI. Fig. 23 on page 34
• **METABOLIC ENCEPHALOPATHIES:**

Encephalopathy is a common problem in ESRD patients that may be caused by variable factors. Cerebral imaging is not necessarily required; but it can exclude other structural causes of neurologic symptoms. Some of these having typical imaging appearances are considered here.

**Uremic Encephalopathy:**

**Clinical manifestation:**

It is characterized by, instability of gait, dysarthria, action tremor, asterixis, multifocal myoclonus and unconsciousness. Variation of clinical signs from day to day is typical.

**Cause/ Risk factors:**

The cause has been linked to uremic neurotoxins and it is improved by hemodialysis.

**Distribution:**

It typically shows **basal ganglia**, **thalami** and **midbrain** involvement with uncommon involvement of cortical subcortical areas of parieto-occipital region.

**Imaging findings:**

- **Lentiform Fork sign** is its characteristic feature. [Fig. 24 on page 35](#)
- On CT scan confluent hypodensity seen in these particular regions.

**Hypoglycemic Encephalopathy**

**Cause / Risk factors:**

CKD is an independent risk factor for hypoglycemia, and exacerbate the risk already present in diabetics.

**Clinical manifestation:**
Typical symptoms of hypoglycemia are associated by measured plasma glucose concentration **70 mg/dL**. There are focal neurologic symptoms including, aphasia, hemiplegia and convulsion. Hypoglycemic encephalopathy occurs in prolong and sever hypoglycemia.

**Distribution:**

- The characteristic findings occur in cortex of the **bilateral cerebral hemispheres**. It can also be seen in hippocampus, basal ganglia and corpus callosum. **Fig. 25 on page 36 Fig. 26 on page 37**
- Cerebellum, thalami and brain stem are usually spared in adults.

**Imaging findings:**

Earliest findings are shown by DWI which shows restriction. High signal intensity is seen on T2WI and Flair and low signals on T1WI.

**Wernicke's encephalopathy**

**Cause/ Risk factors:**

- It is not common in uremic patients, but hemodialysis increases the risk because not only low thiamine intake but apparently also promoted loss of thiamine.
- Early identification is paramount because timely thiamine administration can reverse the clinical features.

**Clinical manifestation:**

It typically presents with the triad of **ophthalmoplegia, ataxia** and **cognitive symptoms**. It often remains obscured with high mortality.

**Distribution:**

Typically abnormal signals are seen in **thalami**, **mammillary bodies**, **tectal plate**, and **periaqueductal area** in symmetric distribution.

**Imaging findings:**
CT is usually normal. On MRI reversible vasogenic edema is seen. Restricted diffusion may be seen. **Fig. 27** on page 38

- **BRAIN ATROPHY:**

  **Cause / Risk Factors:**

  - Patients on hemodialysis show significant cerebral atrophy. This corresponds to longer hemodialysis duration and cognitive deficits.
  - The causes can be assigned to *aluminium containing dialysate, small vessel disease, uremic intoxication, malnutrition* and *anemia.*

  **Imaging findings:**

  CT and MRI are equally able to demonstrate generalized cortical atrophy, but MRI is more sensitive. Prominent sulci and ventriculomegaly without bulging of the third ventricular recesses are seen. **Fig. 28** on page 39

- **CEREBRAL METASTATIC CALCIFICATIONS:**

  **Cause/ Risk factors:**

  The most common causes of these calcifications with a striatum-pallidus-dentate distribution are the *calcium- phosphorus metabolism* disorders due to secondary hyperparathyroidism as a consequence of CKD.

  **Clinical manifestations:**

  - Symptoms can range from delirium, dementia to seizures and focal neurological complains.
  - Occurrence of *extraskeletal metastatic calcifications* increases with hemodialysis.

  **Imaging findings:**

  - There is symmetric involvement of *basal ganglia, thalami, basal ganglia* and *dentate nuclei.* Subcortical white matter can also be involved.
  - Areas of calcifications are best seen on non-contrast CT scan. **Fig. 29** on page 40

- **RENAL OSTEODYSTROPHY (RO):**
Cause/Risk factors:

The musculoskeletal disease associated with CKD is complex and multifactorial and are frequently seen.

Clinical Manifestations:

Varying neurological complains have been documented, mainly related to the cranial nerves compression.

Imaging findings:

- Involvement of the cranial bones causes calvarial hypertrophy and narrowing of the neural foramina. Widening of the diploic space is a common radiological finding.
- Brown tumors are also not uncommon. Fig. 30 on page 41 Fig. 31 on page 42
- INTRACRANIAL NEOPLASMS:

The immunosuppressive condition of patients with CKD is invariably associated with an increased incidence of neoplasia. Although incidence of intracranial neoplasms is uncommon. primary CNS lymphoma Fig. 32 on page 43 have been described in ESRD patients.
Fig. 1: CT scan brain plain demonstrates White cerebellar sign. There is diffuse decrease in density of supratentorial brain parenchyma with increase attenuation of cerebellum representing hypoxic ischemic insult.

© Sindh institute of urology and transplantation - Karachi/PK
Fig. 2: Acute ischemic infarct of ACA/MCA territory. Restriction is seen on DWI with significantly low signals on ADC map.

© Sindh institute of urology and transplantation - Karachi/PK
Fig. 3: CT brain: There are multiple non-enhancing cortical based hypodense lesion consistent with border zone infarcts.

© Sindh institute of urology and transplantation - Karachi/PK
**Fig. 4:** Widespread symmetric hyperintense signals in cortex and deep grey matter. On T1WI gyriform hyperintensities in similar locations corresponding to cortical laminar necrosis. No diffusion restriction representing its chronic stage.

© Sindh institute of urology and transplantation - Karachi/PK
**Fig. 5**: MRI brain shows extensive deep white matter changes with ex-vacuo dilatation of lateral ventricles.

© Sindh institute of urology and transplantation - Karachi/PK
**Fig. 6:** Extensive loss of brain parenchyma in left middle cerebral artery distribution with cortical laminar necrosis.

© Sindh institute of urology and transplantation - Karachi/PK
Fig. 7: Extensive intraventricular hemorrhage with intraparenchymal extension is seen in this CT scan.

© Sindh institute of urology and transplantation - Karachi/PK
**Fig. 8:** Subarachnoid hemorrhage in left sylvian fissure (black arrow)

© Sindh institute of urology and transplantation - Karachi/PK
Fig. 9: MRI brain shows crescent shaped right subdural hematoma.

© Sindh institute of urology and transplantation - Karachi/PK
**Fig. 10:** Adult polycystic kidney disease as a cause of renal failure. These are associated with aneurysm rupture and hemorrhage. MRI brain shows intraparenchymal hemorrhage in left frontal lobe with intraventricular extension.(blue circles) Aneurysm present at the presumed junction of left Anterior cerebral and Anterior communicating artery.(green circle). SWI shows areas of blooming.

© Sindh institute of urology and transplantation - Karachi/PK
Fig. 11: MRI brain showing findings of hypertensive microangiopathy with microhemorrhages characteristically present in deeper locations i.e. pons and basal ganglia. Best seen on SWI.

© Sindh institute of urology and transplantation - Karachi/PK
Fig. 12: MRI brain demonstrate thick enhancing leptomeninges consistent with meningitis.

© Sindh institute of urology and transplantation - Karachi/PK
Fig. 13: Multiple brain abscesses in left fronto-parietal region with subtle contralateral midline shift. DWI is showing restriction with low values on ADC.

© Sindh institute of urology and transplantation - Karachi/PK
Fig. 14: Disease processes leading to end stage renal failure can also lead to neurological complications. A young female patient. Chronic renal failure secondary to lupus nephritis. Extensive edema in the cortex and subcortical white matter of the cerebral hemispheres, diagnosed as Lupus cerebritis.

© Sindh institute of urology and transplantation - Karachi/PK
**Fig. 15:** Same patient as in Fig.12: Marked clinical and radiological improvement is seen on follow up brain MRI.

© Sindh institute of urology and transplantation - Karachi/PK
**Fig. 16:** Angio-invasive fungal cerebritis. There is extensive bilateral signal abnormality involving deep white matter of fronto-parietal region. Microhemorrhages are present. Subtle enhancement of the affected areas along with left ventriculitis is also noticed.

© Sindh institute of urology and transplantation - Karachi/PK
**Fig. 17:** In a young female patient on hemodialysis, MRI brain is showing multiple cerebral abscesses in left occipital region with associated with enhancing meninges representing meningitis. Subdural empyema is also seen in frontal region on left side.

© Sindh institute of urology and transplantation - Karachi/PK
**Fig. 18:** Intraparenchymal extension of extra cerebral infection. Fungal sinusitis involving left nasal cavity, maxillary sinus and ethmoid sinuses. Erosion and destruction of walls of maxillary sinus is also visualised. On post contrast study (c) filling defect representing thrombus is noted in distended left cavernous sinus. (black arrow).

© Sindh institute of urology and transplantation - Karachi/PK
Fig. 19: MRI brain: Right eye proptosis with extensive infiltration and enhancement in retro-orbital region representing orbital cellulitis. Intra-cerebral extension of the infection is seen along right temporal lobe and down to the brain stem which is showing edema with peripheral enhancement. (white arrows)

© Sindh institute of urology and transplantation - Karachi/PK
**Fig. 20:** Cerebral venous sinus thrombosis in a 51 years old female patient. Conventional MRI sequences and MR venography often provide sufficient information to make a diagnosis of CVST or at least make a suspicion. (a) On T1WI Hyperintensity seen in superior sagittal sinus with bilateral symmetrical abnormal signal intensity in parasagittal region on T2WI(b). On post contrast (d,e) empty delta sign is seen with increased vascularity of cortical vein secondary to congestion. TOF MRV shows filling defect at confluence and distal superior sagittal sinus

© Sindh institute of urology and transplantation - Karachi/PK
**Fig. 21:** Characteristic symmetrical high T2/FLAIR signal abnormality centrally in the pons is shaped like a trident. Midbrain also demonstrates similar bilateral symmetric edema.

© Sindh institute of urology and transplantation - Karachi/PK
Fig. 22: This 62 years old patient has typical history of sudden rise of blood pressure during dialysis. There is cortical / subcortical edema in the areas supplied by posterior circulation. DWI doesn’t show restriction representing its vasogenic nature. Microhemorrhages can also be seen in PRES which are more evident on SWI. (circle)

© Sindh institute of urology and transplantation - Karachi/PK
**Fig. 23:** A 41 years old male patient on hemodialysis for 6 years presented with speech problem and difficulty walking that rapidly progressed to loss of coordination and altered level of consciousness. On investigation profound immunosuppression is noted with CD4 count of 150/ul. On MRI there are multifocal bilateral confluent white matter signals with involvement of U-fibres. These findings combined with clinical presentation and lab investigation strongly raised the possibility of PML. Unfortunately patient expired shortly after this MRI.

© Sindh institute of urology and transplantation - Karachi/PK
Fig. 24: A 39 years old ESRD patient who is not compliant with hemodialysis, came to ER in obtunded state. MRI brain shows typical findings of Uremic encephalopathy with bilateral almost symmetrical involvement of basal ganglia, internal and external capsule giving Lentiform Fork sign.

© Sindh institute of urology and transplantation - Karachi/PK
**Fig. 25:** A 35 years old ESRD patient on hemodialysis develop seizures and presented with altered state of consciousness. MRI brain demonstrate abnormal signals in bilateral cerebral cortex. Severe hypoglycemia was noticed, serum glucose level was 20 mg/dl.

© Sindh institute of urology and transplantation - Karachi/PK
**Fig. 26:** Same patient as in Fig. 25. After glucose administration previously noted signal abnormalities has returned to normal

© Sindh institute of urology and transplantation - Karachi/PK
Fig. 27: MRI brain shows hyperintense signals in dentate nuclie, peri-aqueductal grey, around third ventricle and thalami (white arrows). These abnormalities are showing bilateral symmetric distribution and are more conspicuous on DWI. These findings are highly suggestive of thiamine deficiency. Patient showed remarkable clinical improvement after thiamine administration.

© Sindh institute of urology and transplantation - Karachi/PK
Fig. 28: A 45 years old patient on hemodialysis for 6 years. There is significant sulcal widening and prominence of extra axial CSF spaces with minimal ventricular dilatation. Brain atrophy in this patient is out of proportion to his age. ESRD is known to be associated with accelerated brain atrophy leading to cognitive decline.

© Sindh institute of urology and transplantation - Karachi/PK
Fig. 29: A 45 years old male patient on hemodialysis presented to us with the complains of headache, vertigo and imbalance. CT scan brain plain done a. Bilateral symmetrical calcifications in basal ganglia and thalami. b. Ectopic calcifications are also seen in bilateral dentate nuclei of cerebellum. Secondary Fahr’s Syndrome (bilateral striatopallidodentate calcinosis) can be seen in patients with chronic renal failure.

© Sindh institute of urology and transplantation - Karachi/PK
Fig. 30: A 20 years old female patient on hemodialysis complained of severe frontal headache and hard swelling along the left temporomandibular joint. CT scan brain plain done: a. There are lytic expansile lesions in left mandibular condyle and within the right maxillary sinus. Cortical break is seen along the right lateral wall of maxillary sinus. b. Widening of diploic space in the skull with multiple tiny lytic lesions. Features are highly suggestive of renal osteodystrophy with brown tumors.

© Sindh institute of urology and transplantation - Karachi/PK
**Fig. 31:** Radiographs still remain the initial investigation for evaluation of bone disease, however patients presenting with varying neurological complaints often go directly to cross-sectional imaging of brain. Significant calvarial thickening and widening of diploic space is seen in this brain MRI which is a part of renal osteodystrophy.

© Sindh institute of urology and transplantation - Karachi/PK
**Fig. 32:** MRI brain shows characteristic enhancing infiltrative mass showing subependymal extension and crossing the corpus callosum. Primary lymphomas are commonly found in setting of transplant patients however one can also encounter these in the setting of other immunosuppressive states.

© Sindh institute of urology and transplantation - Karachi/PK
Conclusion

The **cerebrovascular complications** either secondary to the uremic state or its treatment is a significant cause of mortality and morbidity. **Imaging** displays utmost importance in evaluation of these neurological complains. The neurological findings in this group of patients demonstrate **multifactorial trend** and knowledge of imaging findings linked to the neurological complains and **multidisciplinary approach** aid in timely analysis of patient.
References