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Radiology Section

Pictorial Review of Basal Ganglia and Thalamic Lesions

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Basal ganglia (BG) are paired symmetric subcortical (deep grey matter) nuclei that form the core of the extrapyramidal system and control motor activity. BG pathologies manifest clinically as movement and tone dysfunction; some may present with alteration in higher mental functions such as behavioural problems, memory and other thought processes [1].

Magnetic Resonance (MR) imaging is the modality of choice for evaluating the BG. Computed Tomography (CT) may well be the primary investigation, mainly in emergency situations in which patients present with altered sensorium or acute onset seizures [2]. Based on the clinical history, involvement of other brain structures in MRI and the relative laboratory findings, accurate diagnosis is reached. Clinical and laboratory findings correlation is essential to reach accurate diagnosis [2].

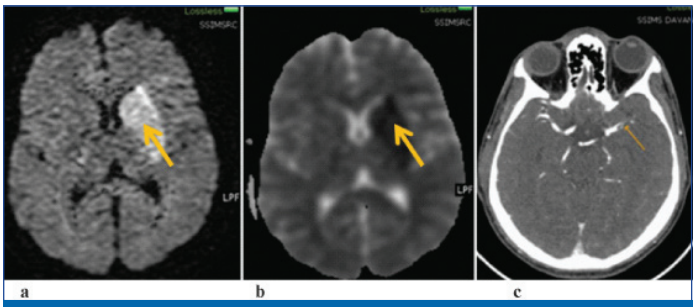
In this pictorial review, we have described different cases affecting BG and thalamus in our Institute. Vascular conditions like infarcts, haemorrhage, cerebral venous thrombosis, Hypoxic Ischaemic Encephalopathy (HIE), toxic conditions like methanol and Carbon monoxide (CO) poisoning, metabolic conditions like hypo and hyper glycaemia, pontine demyelinosis and few infectious conditions like dengue encephalitis and Acute necrotising encephalitis were encountered [Table/Fig-1] [3-11].

Sl no	Age/ Sex	Chief complaint and its duration	Clinical Findings	Radiological findings	Radiological differentials	Diagnosis	Treatment	Findings published in literature
1	26 years/ male	Sudden onset of right sided weakness	Right motor power 2/5	Diffusion restriction in left caudate and putamen with CT angiography revealing thrombus in M2 portion of left Middle Cerebral Artery (MCA) [Table/Fig-2]	Usually unilateral, and no differential to be considered	Acute non haemorrhagic stroke in left MCA territory	Thrombolysis.	
2	31 years/ female	History of altered sensorium and loss of consciousness	Stuporous and altered sensorium	Diffusion restriction with T2, Fluid Attenuated Inversion Recovery (FLAIR) hyperintensity in bilateral median thalami, in anterior midbrain region.[Table/Fig-3]	Osmotic demyelinosis (Pontine involvement is characteristic in this, in artery of Percheron infarct pons will be spared)	Artery of percheron infarct	Thrombolysis.	Sandvig A et al., presented a 56-year-old female who presented with history of over consumption of alcohol and altered sensorium and MRI showed infarcts in bilateral paramedian thalamic nuclei and pons [3]
3	48 years/ male	Sudden onset of loss of consciousness and high blood pressure	Glassgow Coma Scale (GCS) 3/15, intubated.	Hyperdensity (HU: 73) involving left basal ganglia and internal capsule region [Table/Fig-4]	---	Acute intraparenchymal haemorrhage	Surgery	
4	5 days/ male	Preterm and birth asphyxia presents with h/o seizures	At birth low APGAR, Spo_2: 98% Reflexes: abnormal	Diffusion restriction in bilateral basal ganglia and thalami [Table/Fig-5]	Hypoglycaemia	HIE	Conservative management	A study done by Ghei SK et al., on Hypoxic Ischaemic Encephalopathy (HIE), they concluded that two patterns of HIE is seen in neonates, peripheral and basal ganglia thalami, peripheral pattern involving sub cortical white matter and cortex, seen in mild hypoxia, where as basal ganglia and thalami involvement suggests severe hypoxia [4]
5	83 years/ male	H/o chest pain three days back diagnosed as inferior wall MI, presented with altered sensorium and weakness	Bilateral upper and lower limb power 3/5, Altered verbal command	NCCT shows bilateral hypodense swollen basal ganglia caudate and lentiform nucleus, MRI image shows diffusion restriction with corresponding FLAIR hyperintensity [Table/Fig-6]	Any elderly with h/o hypoxic insult bilateral BG showing diffusion restriction, HIE needs to be considered.	HIE adult onset	Treatment of underlying cause.	Muttikkal TJ, Wintermark M, studied 151 cases with HIE in adults and they concluded, patterns associated with relatively favourable clinical outcome were: a) watershed pattern; and b) basal ganglia without cortical involvement and pattern associated with poor clinical outcome were: a) diffuse cortical and deep grey matter pattern, with and without perirolandic sparing; (b) medial occipital with perirolandic involvement c) precentral gyrus involvement; d) diffuse white matter involvement; e) brainstem involvement; f) cerebellar involvement; and g) hippocampal involvement [5]

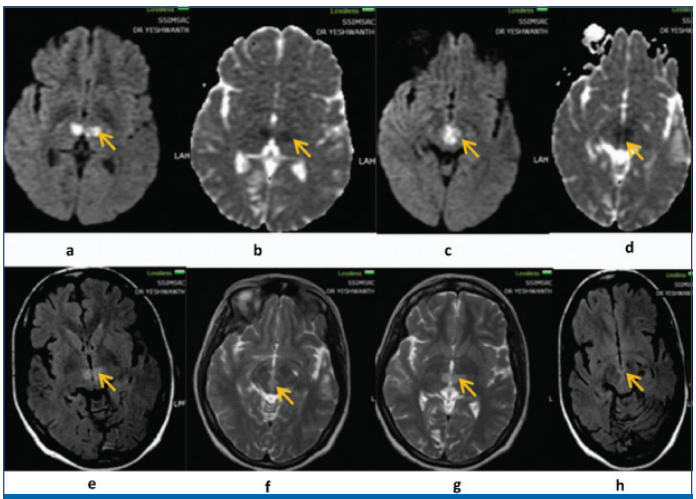
6	44 years/ female	H/o unknown compound poisoning presents with altered sensorium	Stuporous, non responding to verbal commands	Bilateral symmetrical diffusion restriction with FLAIR hyperintenity in basal ganglia, thalami and fronto-parieto-occipital lobe cortex. [Table/ Fig-7]	HIE and drug abuse	CO poisoning	Conservative managements and Chelating agents	Hopkins RO et al., assessed 73 patients with CO poisoning and concluded nearly 4-88% of patients will have bilateral basal ganglia lesions [6]
7	28 years/ male	H/o ingestion of spirit (Methanol) presented with altered sensorium	Stuporous, non responding to verbal commands	MRI images shows symmetrical diffusion restriction with FLAIR high signal involving bilateral basal ganglia typically globus pallidus and caudate [Table/ Fig-8]	HIE and drug abuse. Bilateral haemorrhagic pallidal necrosis is the key imaging feature	Methanol intoxication	I.V Ethanol	Elkhamary SM et al., studied 58 patients with methanol intoxication and concluded that Bilateral putamen necrosis was present in 45 cases (77.6%), Optic nerve enhancement and atrophy were detected in 33 cases (56.9%), 21 cases (36.2%) showed combination of bilateral putamen necrosis and optic nerve enhancement [7]. Although in our case putaminal necrosis was not there
8	83 years/ male	H/o of seizures and dysarthria. Treatment for hypernatremia was started	No obvious weakness, at time of admission TC: normal, electrolytes: Na+: 127 mg/dL, K+: 4.2 mg/dL, Urea: 26 mg/dL, Cr: 0.6 mg/dL.	MRI brain demonstrated diffusion restriction with T2, FLAIR hyperintensity in bilateral basal and pons, classically trident shaped [Table/Fig-9]	Pontine ischemia-infarction	Osmotic demyelination syndrome	Slow sodium correction.	In a case study done by Garg P et al., they concluded that diffusion restriction is the first signal abnormality to appear. Second, the caudate nucleus and putamen signal abnormalities preceded the pontine signal abnormalities, i.e., Extrapontine Myelinolysis (EPM) preceded, possibly by a few hours, the Central Pontine Myelinolysis (CPM). Third, the territorial involvement, although characteristic once the imaging findings have evolved, is initially non specific especially if extrapontine precedes pontine involvement and needs a high index of clinical and radiological suspicion for accurate diagnosis [8]
9	13 years/ female	H/o fever, convulsions and altered sensorium since three days. clinically imaging wise necrosis of basal ganglia or thalami helps in narrowing d/d	Investigations: TLC: 15,000, Platelets: 1.3 lacs/ cumm, Electrolytes: normal, Dengue serology: negative, WIDAL: negative	CT images showing symmetrical hypodensities with MRI showing diffusion restriction with corresponidng T2, FLAIR hyperintensities in bilateral thalamus, posterior putamen and midbrain [Table/ Fig-10 a,b]	Any viral encephalitis involves basal ganglia on imaging, rapid clinical deterioration may raise suspicious of acute necrotising encephalitis	Acute Necrotising Encephalitis (ANE)	Conservative only, poor prognosis	Mizuguchi M et al., studied 13 and 28 patients with acute encephalopathy concluded in ANE bilateral thalamic swelling and haemorrhage is the most significant finding [9]
10	21 years/ female	H/o fever, retro bulbar pain and altered sensorium.	Platelet count of 30,000 and Serum Ig M positive (Dengue virus). CSF finding shows normal to abnormal pleocytosis	MRI diffusion restriction in bilateral symmetrical swollen thalami with T2, FLAIR hyperintensity, no blooming on SWAN sequence indicating no haemorrhage. [Table/Fig-11]	Bilateral thalamic involvement is diagnostic of dengue.	Dengue encephalitis.	Platelet transfusion, IV fluids, IV Paracetomol infusion	
11	38 years/ male	Acute onset of confusion and drowsiness	BP at time of admission: 116/ 80 mmHg, RBS (Random blood sugar) was found to be 40 mg/dL	MRI revealed diffusion restriction with corresponding T2, FLAIR hyperintensities involving bilateral basal ganglia region. [Table/Fig-12]	-	Hypoglycaemia	IV Dextrose.	
12	60 years/ female	Acute onset of abnormal movements of both hands, at time of admission	RBS was found to be 220 mg/ dL, HbA1c was 7.8 IU	CT shows tiny calcifications in bilateral lentiform nucleus with MRI showing bilateral symmetrical T2, T1, and FLAIR hyperintensities, on SWAN blooming noted in bilateral lentiform nucleus. [Table/ Fig-13]	Fahr's disease	Non ketotic hyperglycaemic / diabetic striatopathy.	Correction of electrolyte and IV fluids	Hansford BG et al., concluded that radiologists should be alert to the possibility of non ketotic hyperglycaemia in patients with asymmetric/lateralising basal ganglia lesions even in the absence of a movement disorder [10]
13	55 years/ male	H/O dementia and seizures	No weakness	CT shows bilateral dense basal ganglia calcifications. [Table/Fig-14]	Non ketotic hyperglycaemia	Fahr's disease	Irreversible phenomena	Ooi HW et al., concluded that radiological findings of bilateral symmetrical basal ganglia and dentate nuclei calcifications are significant [11]

14	36 years/ female	History of severe headache	Headache, no papiloedema, no motor deficit	Axial FLAIR images shows bilateral thalamic hyperintensity and corresponding MR VENOGRAM shows non visualisation of straight sinus consistent with thrombosis (white arrows) [Table/Fig-15]	Thalamic edema is the imaging hallmark of this condition	Cerebral Vein Thrombosis (CVT)	Anticoagulants	

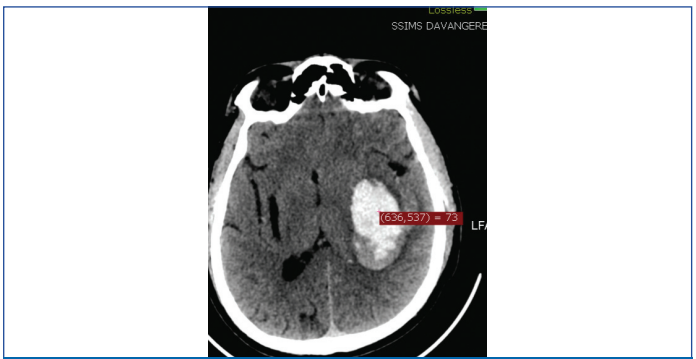
[Table/Fig-1]: Table showing the features of the cases encountered [Table/Fig-2-15] [3-11].



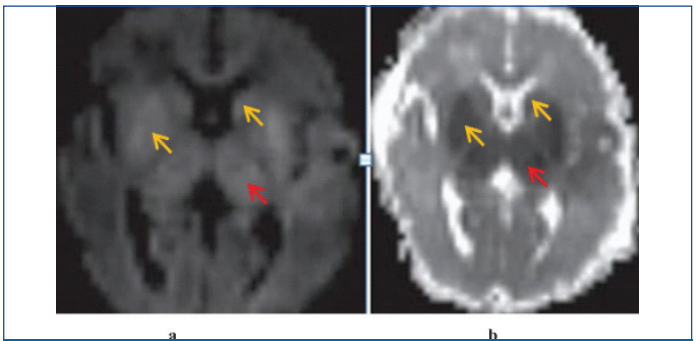
[Table/Fig-2]: MRI image: (a) arrow demonstrates high signal intensity in left caudate and putamen with corresponding ADC shows restriction on image; (b). image; (c), CT angiography of same patient revealed thrombus in M2 portion of left MCA (Yellow arrow).



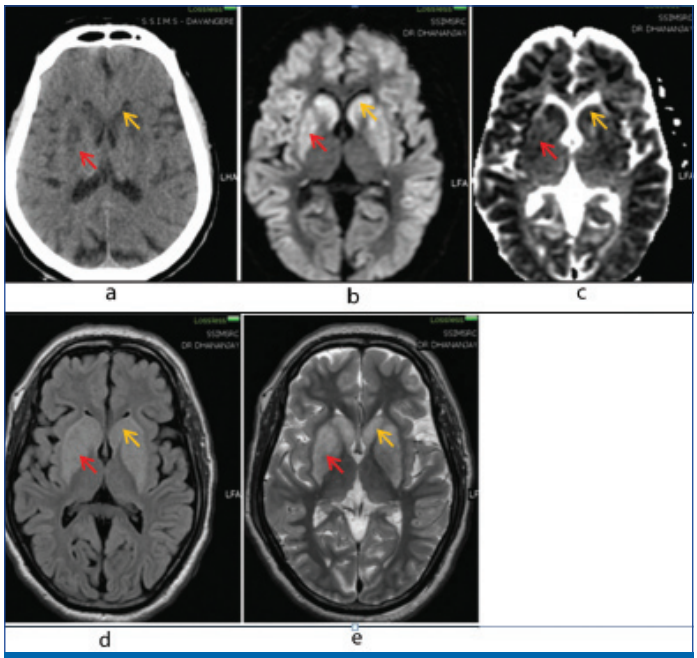
[Table/Fig-3]: MRI brain demonstrates, DWI (a) with ADC (b) shows diffusion restriction with T2, FLAIR hyperintensity (g) and (e) and in bilateral median thalami; (c) and (d) DWI and ADC; (f) and (h) in anterior midbrain region.
DWI: Diffusion weighted imaging; ADC: apparent diffusion coefficient



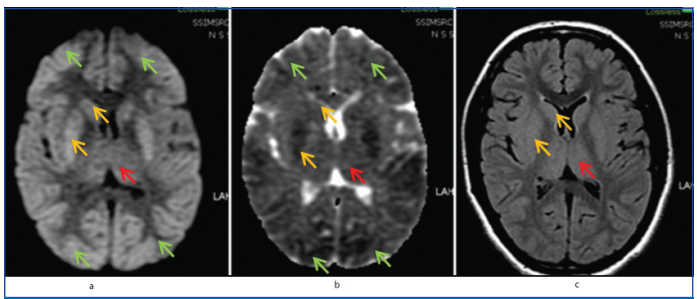
[Table/Fig-4]: Non Contrast Computed Tomography (NCCT) brain shows hyperdense (HU: 73) bleed involving left Basal Ganglia (BG) and internal capsule region.



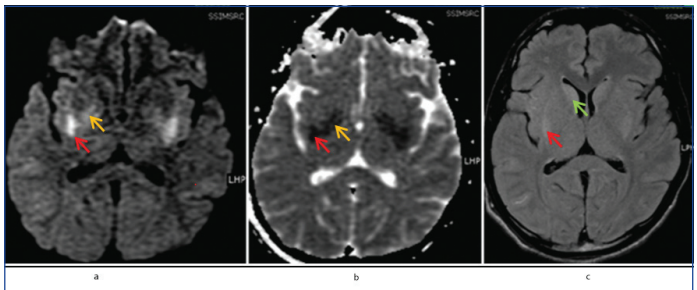
[Table/Fig-5]: Image (a) demonstrates high diffusion in bilateral Basal Ganglia (BG) (yellow arrows) and thalami (red arrows) with corresponding low ADC on image (b).



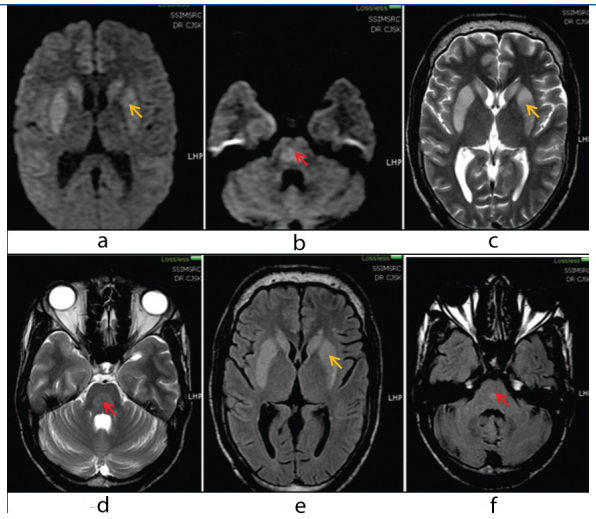
[Table/Fig-6]: Image (a) NCCT shows bilateral hypodense swollen Basal Ganglia (BG) caudate (yellow arrows) and lentiform nucleus (red arrows), MRI image shows high diffusion (b) and corresponding low ADC image (c), and corresponding FLAIR (d),T2 (e) hyperintensity.



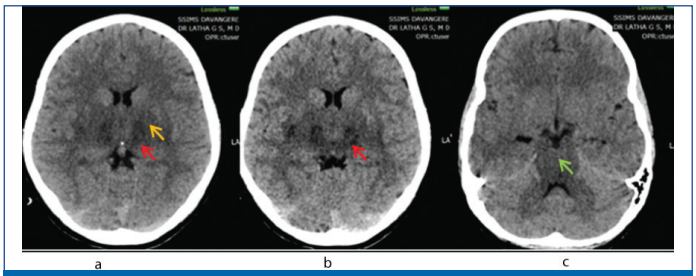
[Table/Fig-7]: MRI shows, image (a) high signal on diffusion, (b) low on ADC involving bilateral symmetrical Basal Ganglia (BG) (yellow arrows), thalami (red arrows) and fronto-parieto-occipital lobe cortex (green arrows), FLAIR high signal intensity on image (c)-Features suggestive of toxic encephalopathy later proven to be CO poisoning.



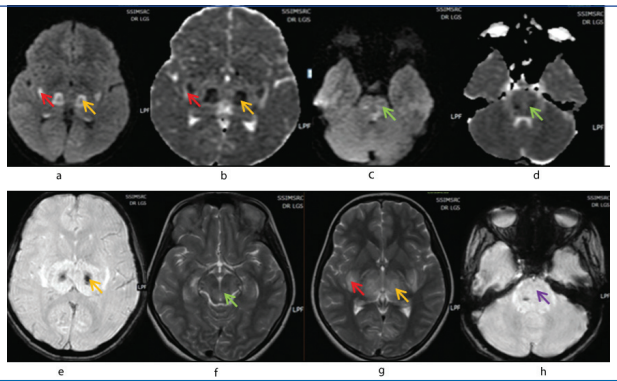
[Table/Fig-8]: MRI images shows symmetrical high signal intensity on diffusion image (a), with low ADC image (b) and high signal on FLAIR (image c) involving bilateral Basal Ganglia (BG) typically globus pallidus (red arrow), putamen (yellow arrow) and caudate (green arrows).



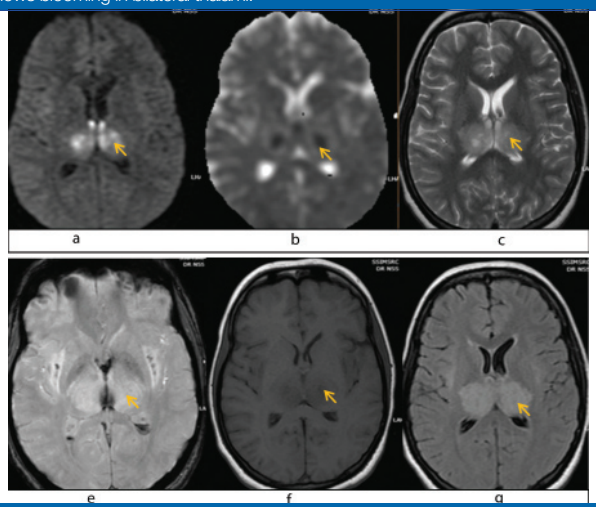
[Table/Fig-9]: MRI brain demonstrates, DWI (a), (b) shows high signal intensity with T2 (c) and (d) and FLAIR (e) and (f) hyperintensity in bilateral Basal Ganglia (BG) (yellow arrows) and pontine (red arrows), classically trident shaped.



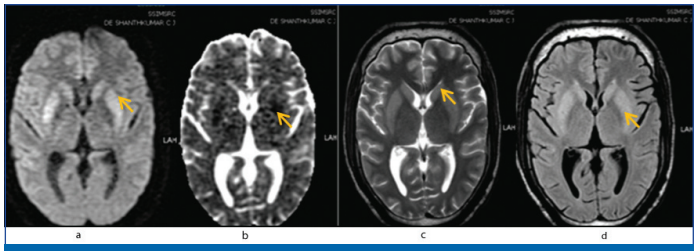
[Table/Fig-10a]: CT images showing symmetrical hypodensity seen in bilateral thalamus (yellow arrow), posterior putamen (red arrows (images a and b) and mid-brain (green arrows).



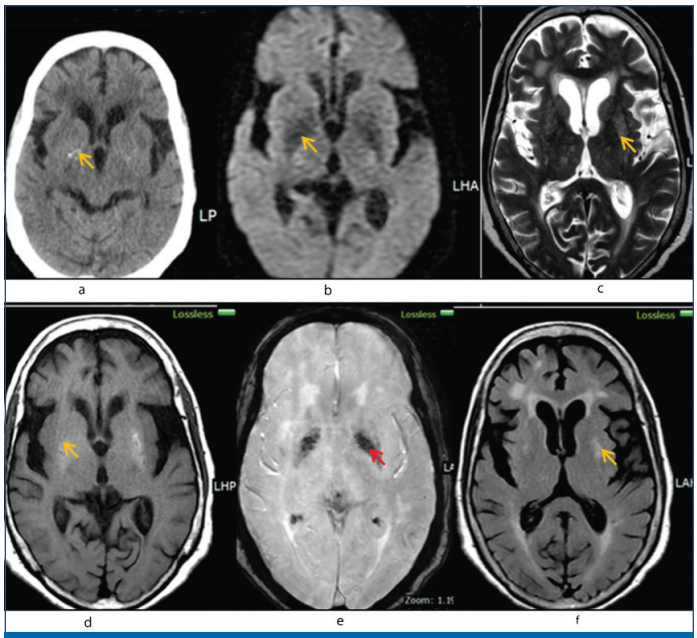
[Table/Fig-10b]: MRI brain demonstrates, DWI (a), (c) shows high signal intensity with low signal on ADC on images (b) and (d) with high intensity on T2WI (f) and (g) involving bilateral thalamus (yellow arrows), posterior putamen (red arrows), midbrain and pons (purple arrow) and on Susceptibility-Weighted Angiography (SWAN) sequence on image (e) shows blooming in bilateral thalami.



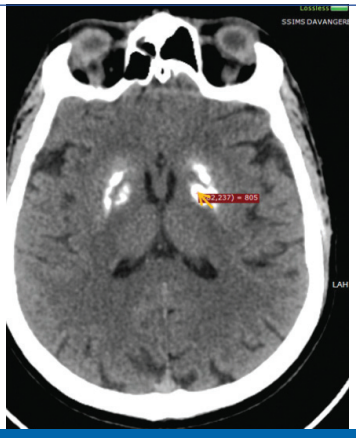
[Table/Fig-11]: MRI revealed diffusion restriction (a) with low ADC (b), bilateral symmetrical swollen thalami with T2 (c) FLAIR (d) hyperintensity, T1 (e) hypointensity no blooming on SWAN sequence (f) indicating no haemorrhage.



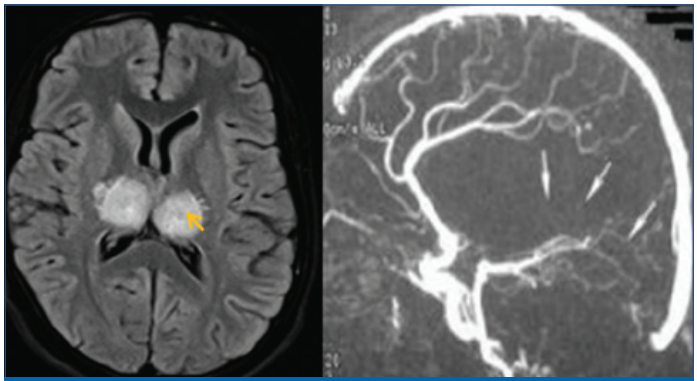
[Table/Fig-12]: MRI revealed diffusion restriction i.e., high signal on DWI (a) low on ADC (b), with Corresponding T2 (c), FLAIR (d) hyperintensities involving bilateral Basal Ganglia (BG) region (yellow arrows).



[Table/Fig-13]: CT (image a) shows tiny calcifications in bilateral lentiform nucleus (yellow arrow), MRI shows no diffusion restriction (image b), bilateral symmetric T2 (image c), T1 (image d), and FLAIR (image f) hyperintensities, on SWAN (image e) blooming noted (red arrows) in bilateral lentiform nucleus (yellow arrow).



[Table/Fig-14]: CT image reveals dense calcifications (HU: 805) in bilateral ienti-form nucleus (yellow arrow).



[Table/Fig-15]: Axial FLAIR images show bilateral thalamic hyperintensity (yellow arrows) and corresponding MR VENOGRAM shows non visualisation of straight sinus consistent with thrombosis (white arrows).

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