PICTORIAL ESSAY | Neuro/Head and Neck Radiology

# Bilateral thalamic lesions: a pictorial essay

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## ABSTRACT

The aim of this pictorial review is to familiarize radiologists with the numerous pathologies that can affect bilateral thalami while demonstrating their several neuroimaging manifestations. Vascular etiologies include infarcts of the artery of Percheron, tip of the basilar syndrome, venous infarcts, hypoxic-ischemic encephalopathy, PRES, hypertensive microbleeds, and CADASIL; infectious etiologies include Creutzfeldt-Jakobs disease and encephalitides, while demyelinating disorders include ADEM and MS. Bilateral thalamic involvement may also be seen in metabolic & toxic etiologies such as Wernicke encephalopathy, osmotic myelinolysis, Fabry disease, Fahr disease, Wilson disease, and Leigh disease. Furthermore, low- and high-grade gliomas may originate or infiltrate bilateral thalami while gadolinium deposition can be a mimicker of disease. Radiological features that can be used in the assessment and differential approach include MR signal characteristics, calcifications, exact location within the thalamus, symmetry, presence of synchronous extra-thalamic involvement, and presence of expansion. Additional imaging tools such as DWI, MRA/MRV/CTA/CTV, MRS, PWI, and correlation with clinical and laboratory findings may narrow the differential diagnosis.

KEY WORDS
Thalamic; Bilateral; Basal ganglia; Central Nervous System; CNS; Radiology; CT; MRI; Diagnostic; Imaging;

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## *Introduction*

The thalami are paired deep grey matter (GM) structures situated amongst the midbrain and cerebral white matter (WM), on both sides of the third ventricle. Even though bilateral involvement is unusual, the thalami can be affected by an extensive range of diseases. As they are responsible for several functions, when facing certain pathologies making a timely diagnosis can be of utmost importance. The aim of this pictorial review is to familiarise radiologists with the numerous pathologies that can affect bilateral thalami **(Table 1)** while demonstrating their several neuroimaging manifestations. The approach of an accurate diagnosis can be attained by combining the radiological, clinical, and laboratory findings.

## Clinical entities

### Vascular Aetiologies

*Arterial Infarcts*

Blood supply of the thalami is derived from numerous arteries originating from the posterior communicating artery and P1/P2 segments of the posterior cerebral arteries [1]. Bilateral thalamic infarcts occur rarely. The artery of Percheron (AOP) is an uncommon anatomic variant where a single trunk arising from one P1 segment, supplies both paramedian thalami and/or rostral midbrain. If occluded, it causes infarcts in the aforementioned territories [2] with imaging manifestations following those of a brain infarct **(Fig. 1)**. The main differential diagnosis is top of the basilar syndrome (bithalamic ischaemia owing to top of the basilar artery occlusion), however ischemic infarcts in this entity may also occur in vascular territories of the posterior cerebral, and/or the superior cerebellar arteries [3].

*Venous Infarcts*

Cerebral venous thrombosis (CVT) has been associated with several causes (i.e. oral contraceptives, infection, dehydration, pregnancy), with the aetiology occasionally remaining unidentified [4]. A hyperdense venous thrombus can sometimes be seen on non-contrast CT. On MRI thrombus signal might vary depending on its age, while an absence of normal flow void in dural venous sinuses can also be noted. CT venography (CTV) and MR venography (MRV) can display the filling defect and the lack of flow, respectively. Venous infarcts will appear hyperintense in T2/FLAIR sequences and will not comply with arterial territories, while peripheral enhancement around the clot or absence of enhancement might be displayed in T1 post gadolinium images [5]. CVT can cause both vasogenic and cytotoxic oedema, with or without haemorrhage [5]. Cytotoxic oedema superimposed on pre-existing vasogenic oedema will lead to patchy areas of restricted and increased diffusion [6]. Bithalamic concomitant enlargement may also be seen [5], with the thalami most often being affected when internal cerebral veins have been occluded.

*Hypoxic-ischemic Encephalopathy (HIE)*

HIE is encountered in numerous settings (i.e. cardiac arrest, drowning, asphyxiation) due to severe global hypoxic-ischemic injury, usually with devastating neurological sequelae. Due to increased metabolic requirements making them prone to injury, GM structures are primarily damaged. Diffusion-weighted imaging (DWI) is the first imaging modality to showcase anomalous findings, owing to early cytotoxic oedema **(Fig. 2)** [7].

*Posterior Reversible Encephalopathy Syndrome (PRES)*

PRES results from a cerebral vascular autoregulation malfunction, usually affecting the occipital and parietal regions and less frequently, the brainstem, basal ganglia, thalami, and cerebellum. Typical imaging findings include T2/FLAIR hyperintensity, and increased diffusivity, while contrast enhancement, haemorrhage, and diffusion restriction are uncommon **(Fig. 3)**. The vast list of aetiologies includes, although is not restricted to, severe hypertension and drug toxicity (i.e. cyclosporine) [8].

*Haemorrhage*

Hypertension can cause thalamic and basal ganglia microbleeds, often encountered bilaterally [9].

*Cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL)*

CADASIL is an autosomal dominant disorder caused

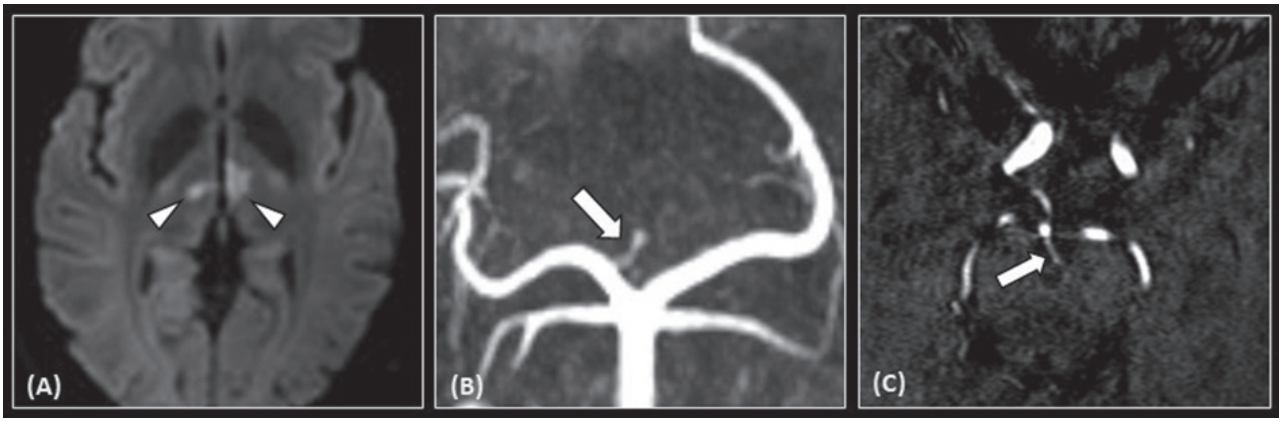


***Fig. 1. Artery of Percheron infarct.*** *(**A**) Both paramedian thalami showcase restricted diffusion in an asymmetric fashion (arrowheads), in a 29-year-old male patient. (**B**), (**C**) MRA-TOF reconstructions demonstrate the artery of Percheron arising from the right P1 segment and exhibiting abrupt occlusion (arrow).*

**Table 1. Categorisation of the numerous pathologies that can affect bilateral thalami according to their aetiology. CADASIL: Cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy, PRES: Posterior reversible encephalopathy syndrome**

| Vascular | Infectious-Inflammatory | Demyelinating | Metabolic/Toxic | Neoplastic | Other |
|---|---|---|---|---|---|
| Arterial infarcts | Infectious Encephalitis | Multiple sclerosis | Wernicke encephalopathy | Glioma | Gadolinium deposition |
| Venous infarcts | Autoimmune Encephalitis | Acute disseminated encephalomyelitis | Osmotic myelinolysis | | |
| Hypoxic-ischemic encephalopathy | Creutzfeldt-Jakob disease | | Fabry disease | | |
| PRES | | | Fahr disease | | |
| Haemorrhage | | | Wilson disease | | |
| CADASIL | | | Leigh disease | | |

by small and middle-sized arterial vasculopathy, secondary to fibrotic thickening of their basement membrane. MRI is the examination of choice, often displaying white matter hyperintensities (WMHs), lacunar infarcts and microbleeds. Diffuse involvement of the subcortical WM may be seen, with the anterior temporal lobes and external capsules representing preferential sites for WMHs. Furthermore, hyperintense lesions might also be seen in the basal ganglia, thalamus and pons **(Fig. 4)**. Nonetheless, definite diagnosis mandates genetic identification of the mutated NOTCH3 gene [10].

## Infectious Aetiologies

*Creutzfeldt-Jakob disease (CJD)*

CJD is a spongiform encephalopathy caused by prions. It is comprised of four types, the sporadic form (most common), the variant form (associated with bovine spongiform encephalopathy - aka mad cow disease), the familial and iatrogenic form. It may lead to rapidly progressive dementia or other less characteristic non-specific neurologic manifestations. A definitive CJD diagnosis requires a brain biopsy, however for a non-intrusive diagnosis of the sporadic form, periodic sharp wave complexes on electroencephalo-

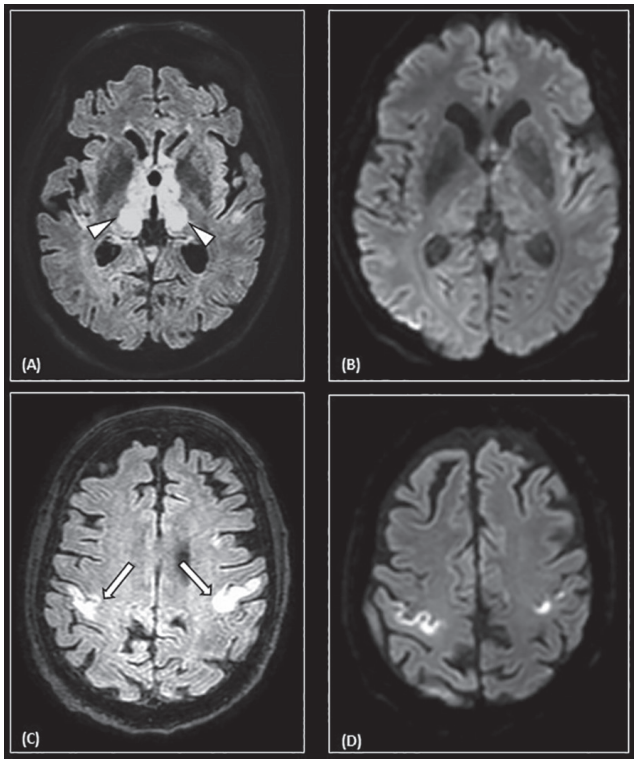


***Fig. 2. Hypoxic-ischemic encephalopathy.*** *(**A**) Axial FLAIR image demonstrates extensive hyperintensity including bilateral thalami (arrowheads), in a 74-year-old male patient with pancreatic adenocarcinoma. (**B**) Axial DWI displays only mildly increased signal in the aforementioned territories, indicative of the subacute phase of their involvement. (**C**) Axial FLAIR image in the same patient demonstrates hyperintense signal around bilateral central sulci (arrows). (**D**) Axial DWI correspondingly displays increased signal in the perirolandic cortex, signifying acute involvement. The above findings imply that in this patient the cortex was affected subsequently to the thalami.*

gram, and detection of 14-3-3 protein in the CSF are required. On MRI basal ganglia, thalami and cerebral cortex are typically involved seen as restricted diffusion in DWI/ADC sequences (more sensitive) and increased signal in T2 sequences. Variant CJD typically causes bilateral thalamic lesions in the pulvinar nuclei (pulvinar sign), or the pulvinar and dorsomedial thalamic nuclei (hockey stick sign). However thalamic involvement can be seen in the sporadic form, as well **(Fig. 5)** [11, 12, 13].

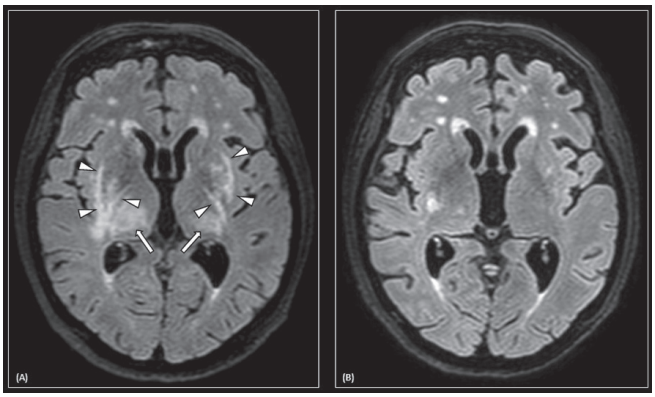


***Fig. 3. PRES.*** *(**A**) Axial FLAIR image demonstrates increased signal intensity in bilateral thalami (arrows) as well as bilateral external and internal capsules (arrowheads), in a 58-year-old female patient with a history of systemic lupus erythematosus (SLE). Lesions were attributed to Posterior Reversible Encephalopathy Syndrome. (**B**) Follow up imaging obtained 20 days later displays subsidence of imaging findings in the aforementioned regions.*

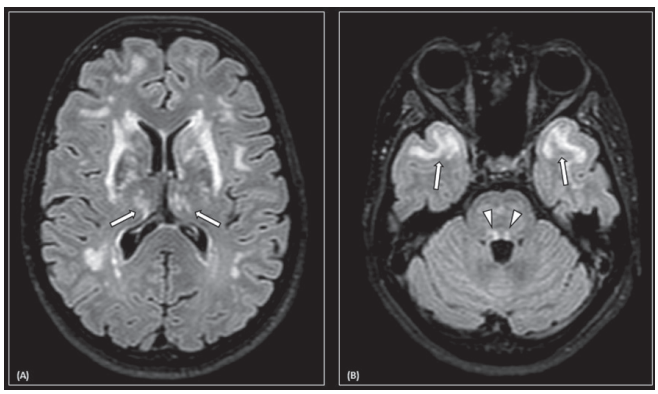


***Fig. 4. CADASIL.*** *(**A**) Axial FLAIR image demonstrates hyperintensities in bilateral external capsules and thalami (arrows A), as well as diffuse involvement of the subcortical, and to a lesser extent, the periventricular white matter, in a 40-year-old male patient. (**B**) Axial FLAIR image at a lower level, displays bilateral characteristic subcortical anterior temporal lobe involvement (arrows B) as well as additional lesions in the pons (arrowheads), in the same patient.*

*Encephalitis*

Several viral forms of encephalitis (e.g. West Nile virus, Japanese encephalitis, Herpes encephalitis) may affect thalami and deep GM structures usually symmetrically **(Fig. 6)**. Lesions are usually T2/FLAIR hyperintense

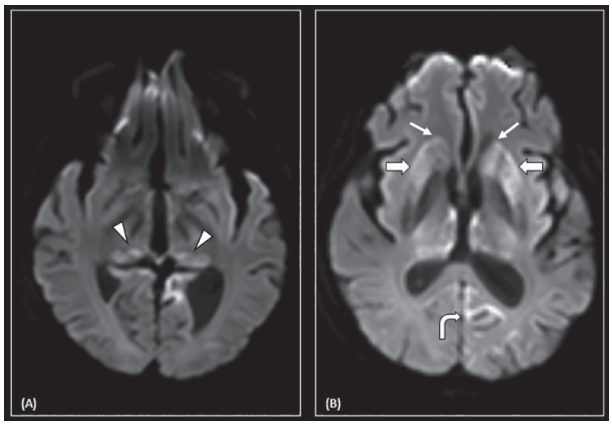


***Fig. 5. Creutzfeldt-Jakob disease.*** *(**A**), (**B**): DWI images: Bilateral pulvinar nuclei diffusion restriction (pulvinar sign) (arrowheads) is demonstrated in a 68-year-old female patient with sporadic CJD. Diffusion restriction is also noticed in bilateral caudate nuclei (thin arrows), bilateral putamina (thick arrows), and left occipital cortex (curved arrow).*

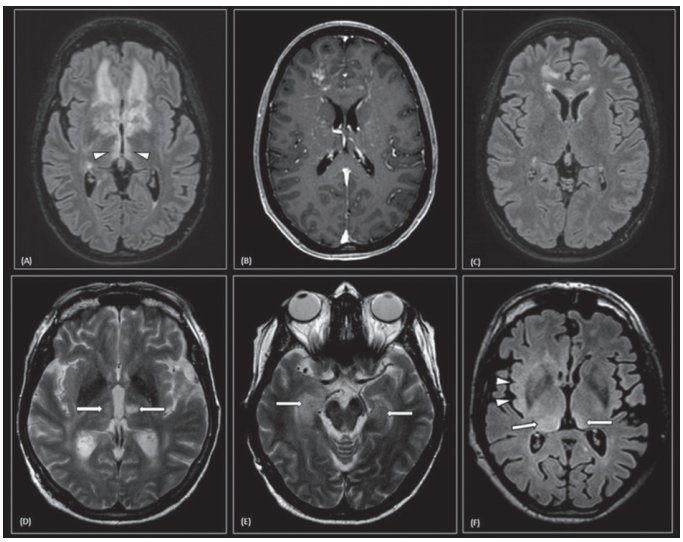


***Fig. 6. Encephalitis.*** *(**A**) Axial FLAIR image showcases diffuse and confluent abnormal signal hyperintensity involving white matter, basal ganglia and the medial aspect of bilateral thalami (arrowheads), in a 37-year-old female patient who has received chemotherapy (G-CHOP) due to Nodular lymphocyte-predominant Hodgkin's lymphoma. (**B**) T1 contrast-enhanced image reveals bilateral areas of linear or nodular enhancement in the same patient. Human Herpesvirus (HHV7) was detected in CSF sampling. (**C**) Axial FLAIR image demonstrates significant subsidence of the signal abnormalities 1 month later, after the patient had received appropriate antiviral treatment. Axial T2 images (**D, E**) of a different patient reveal asymmetric bilateral thalamic involvement (arrows D), and asymmetric bilateral hippocampal abnormalities (arrows E) in a 37-year-old female with anti-gaba-b receptor autoimmune encephalitis. Axial FLAIR image (**F**) (also in a different patient) shows bithalamic hyperintensities (arrows F) in anti-Ma2 (paraneoplastic) encephalitis, as well as right insula involvement (arrowheads F).*

with or without DWI restriction, haemorrhage, or contrast-enhancement. Symptoms and clinical presentation may widely vary, and serologic markers are necessary for a definitive and specific diagnosis [11]. Bithalamic involvement may be likewise seen in autoimmune (i.e. paraneoplastic) encephalitis **(Fig. 6)**.

## Demyelinating disorders

*Multiple Sclerosis (MS)*

Although exceptional, bithalamic involvement (seen as T2/FLAIR hyperintense lesions) can be displayed in MS. Identifying concurrent typical demyelinating MS lesions is essential for the diagnosis **(Fig. 7)** [14].

*Acute Disseminated Encephalomyelitis (ADEM)*

Imaging features partly overlap with MS, however with thalamic and basal ganglia involvement being more frequent in ADEM. Differentiation amongst the two is aided by the monophasic postinfectious/post-vaccination nature of ADEM and by simultaneous enhancement seen in ADEM lesions [14]. However, absence of enhancement does not exclude the diagnosis.

## Metabolic and Toxic Aetiologies

Metabolic and toxic (i.e. medications) **(Fig. 8)** entities normally affect bilateral thalami symmetrically.

*Wernicke Encephalopathy*

Wernicke encephalopathy can result from thiamine (vitamin B1) deficiency due to malnutrition/malabsorption, hence most typically seen in alcoholics. Symmetric signal intensity alterations are characteristically seen in the periaqueductal GM, mammillary bodies, medial thalamic nuclei, and tectal plate **(Fig. 9)** [11].

*Osmotic Myelinolysis*

Osmotic myelinolysis is an acute demyelinating pro-

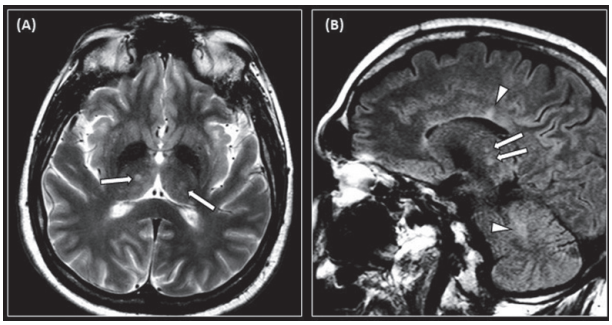


***Fig. 7. Multiple sclerosis.*** *Axial T2 image (**A**) reveals demyelinating lesions in bilateral thalami (arrows A) in a 37-year-old male patient. Sagittal FLAIR image shows two lesions in the left thalamus (arrows **B**) and characteristic MS periventricular - callosal and infratentorial lesions (arrowheads).*

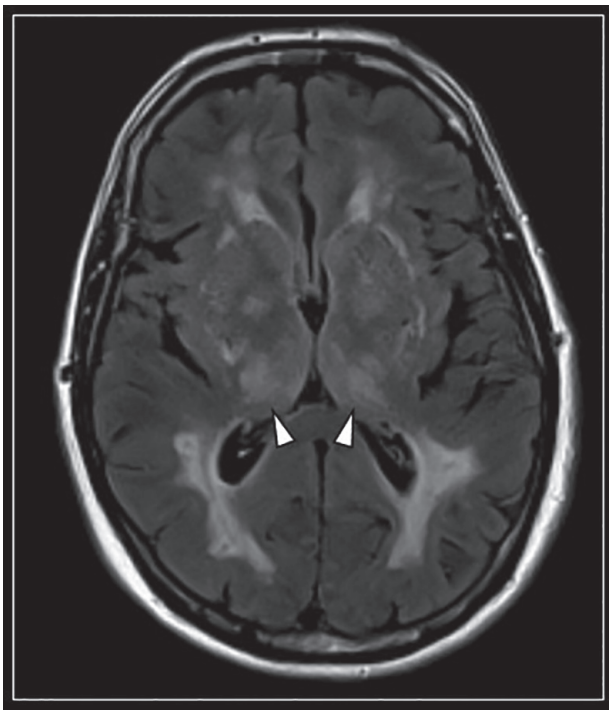

***Fig. 8. Thalamic lesions of toxic etiology.*** *Axial FLAIR image shows chemotherapy induced leukoencephalopathy with diffuse symmetric bilateral involvement of the basal ganglia and thalami (arrowheads), in a 60-year-old male patient with urinary bladder cancer.*

cess instigated by rapid correction of hyponatremia. The central pons is typically affected although extrapontine involvement can also be seen in the thalami, basal ganglia, and WM. Lesions are T2 hyperintense/T1 hypointense whereas restricted diffusion can be seen in the early stages [15, 16].

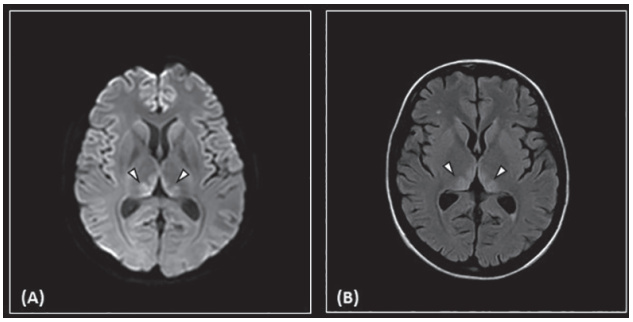


***Fig. 9. Wernicke encephalopathy.*** *DWI (**A**) and FLAIR (**B**) sequences display symmetrically increased signal in bilateral medial thalami (arrowheads) in a 27-year-old female patient with hyperemesis gravidarum.*

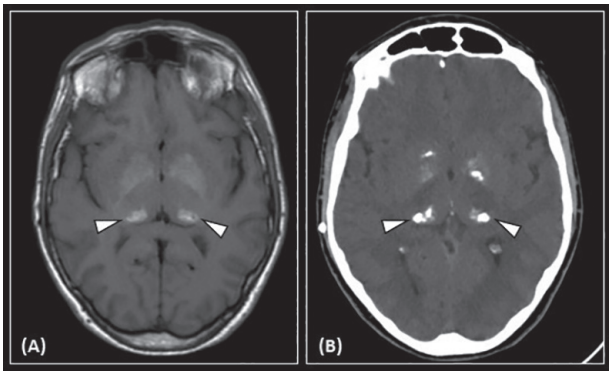


***Fig. 10. Fabry disease.*** *MRI (**A**) and CT (**B**) reveal T1 hyperintensities (**A**) and calcifications (**B**) in the lateral aspects of both pulvinar nuclei (arrowheads) and putamen in a 37-year-old male patient.*

*Fabry Disease*

Fabry disease is an X-linked disorder of glycosphingolipid catabolism due to deficient activity of α-galactosidase with consequential abnormal accumulation of sphingolipids in numerous organ systems. The most sensitive modality to showcase CNS manifestations is MRI. Demonstration of T1 lateral pulvinar hyperintensity is considered pathognomonic [17], although increased T1 signal might also be displayed in deep grey nuclei **(Fig. 10)**. Added findings include T2/FLAIR hyperintensities in periventricular WM and deep GM [18].

*Fahr Disease*

Fahr Disease is an inherited disorder characterised by abnormal vascular calcium deposition, without concurrent calcium metabolism anomaly. CT displays

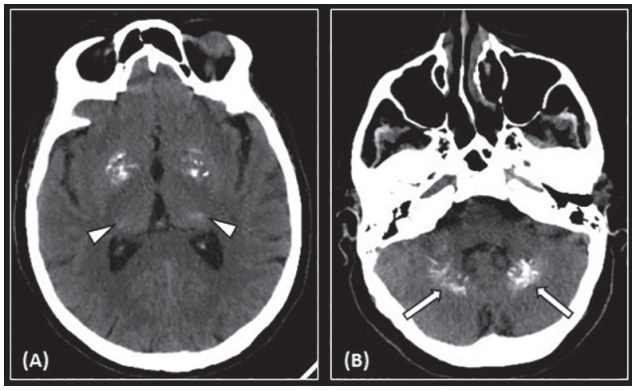


***Fig. 11. Fahr disease. (A)*** *Bilateral calcifications in the basal ganglia and thalami (arrowheads) in a 62-year-old female patient.* ***(B)*** *Dense calcifications are also noted in the dentate nuclei (arrows).*

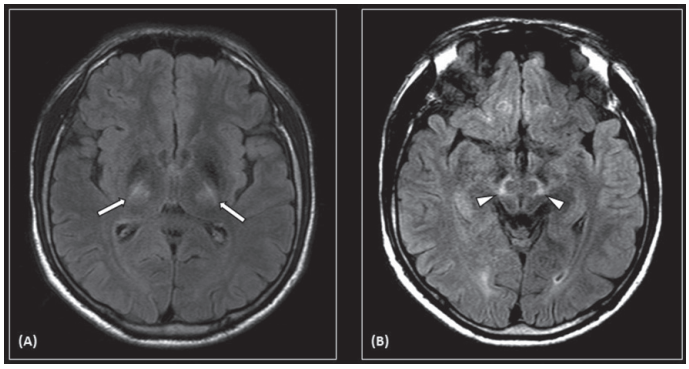


***Fig. 13. Wilson disease.*** *(A) Axial FLAIR sequence demonstrates bilateral symmetric areas of hyperintensity in the ventrolateral thalami (arrows) in a 17-year-old male patient.* ***(B)*** *Axial FLAIR image at midbrain level demonstrates the "face of the giant panda" sign seen as a result of T2/FLAIR hyperintense signal surrounding the unaffected red nucleus and substantia nigra (arrowheads).*

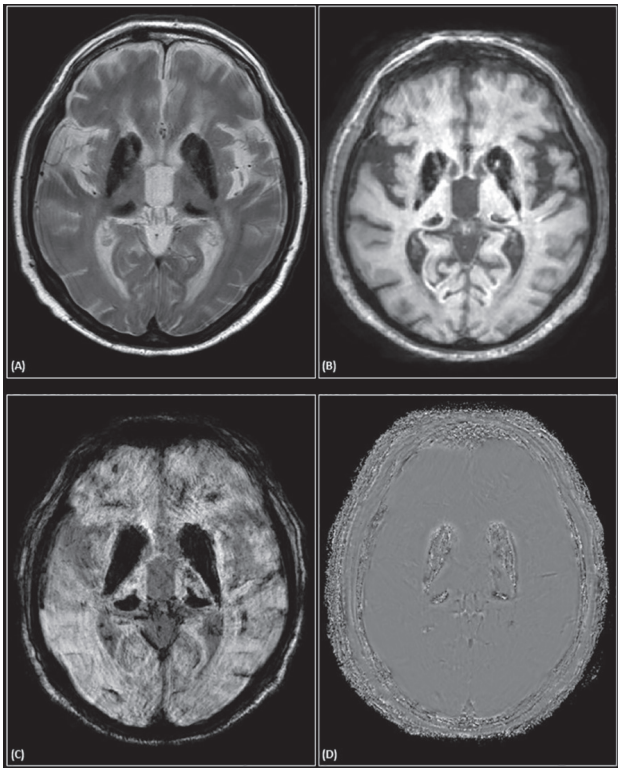


***Fig. 12. Primary hyperparathyroidism.*** *(A) Axial T2 and* ***(B)*** *axial T1 images demonstrate markedly decreased signal intensity affecting bilateral basal ganglia (including bilateral thalami), in a 72-year-old male patient.* ***(C)*** *Susceptibility weighted imaging (SWI) likewise displays signal loss in the aforementioned regions, which seems to correspond to calcifications on altered-phase imaging* ***(D)****.*

dense symmetrical calcifications and atrophy in the basal ganglia, thalami, dentate nuclei, and subcortical WM **(Fig. 11)**. On MRI, these areas will demonstrate high T1/low T2 signal. Important differentials include hyperparathyroidism **(Fig. 12)**, hypoparathyroidism, pseudohypoparathyroidism, and pseudopseudohypoparathyroidism [11, 15].

*Wilson Disease*

In Wilson Disease (autosomal recessive disorder) the abnormal copper metabolism causes its subsequent toxic accumulation in several systems. Patients may demonstrate dysarthria, dystonia, tremor, and choreoathetosis. Kayser-Fleischer rings seen in the cornea on slit-lamp examination, are characteristic. MRI findings include areas of increased T2 signal, with the putamen, pons, and ventrolateral thalamus **(Fig. 13 A)** being most frequently affected [19]. A characteristic, although not exclusive, finding of Wilson disease is the "face of the giant panda" sign which can be seen at midbrain level when unaffected red nucleus and substantia nigra are surrounded by high T2/FLAIR signal **(Fig. 13 B)**.

*Leigh Disease*

Leigh Disease (neurodegenerative mitochondrial disorder) leads to lactic acid build-up. Bilateral T2 hyperintensity of the putamina, caudate nuclei, globi

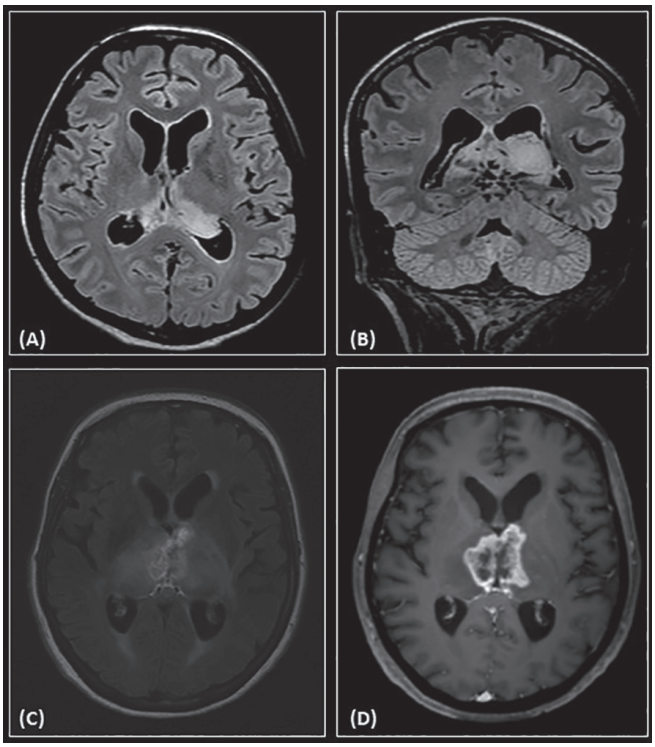


***Fig. 14. Bithalamic glioma.*** *Axial* ***(A)*** *and coronal* ***(B)*** *FLAIR images show diffuse anaplastic astrocytoma infiltrating bilateral thalami. Different patient with glioblastoma involving bilateral thalami seen on axial FLAIR* ***(C)*** *and T1 post-contrast administration images* ***(D)****.*

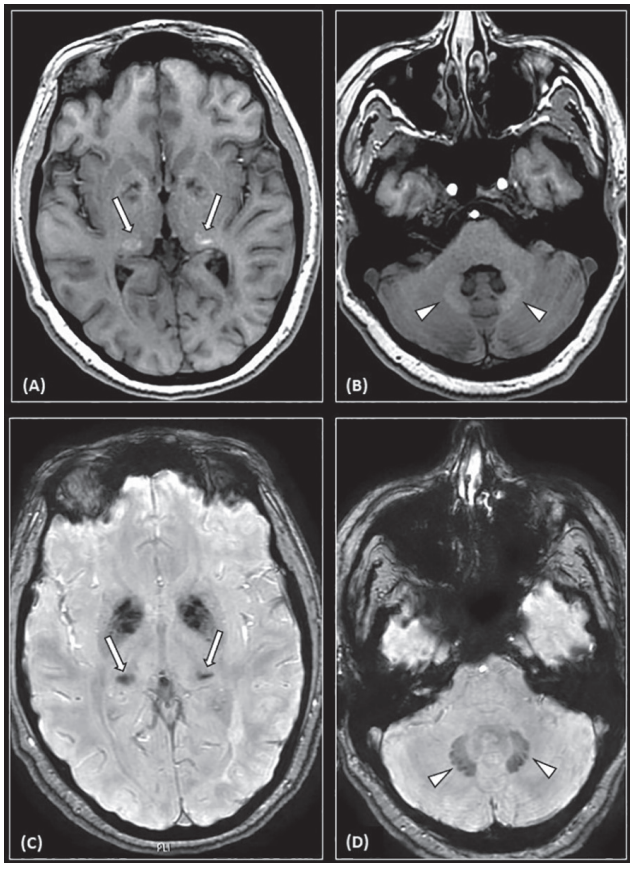


***Fig. 15. Gadolinium deposition.*** ***(A, B)*** *Axial T1 unenhanced images demonstrate high T1 signal intensity in bilateral thalami (arrows A) and bilateral dentate nuclei (arrowheads B) in a 36-year-old female patient with longstanding MS repeatedly receiving linear contrast agents on follow-up MRIs.* ***(C), (D)*** *T2* axial sequences display corresponding low signal in the aforementioned areas.*

pallidi, thalami, and brainstem can be displayed on MRI, occasionally also accompanied by diffuse supratentorial T2 WM hyperintensities. MR spectroscopy (MRS) may reveal increased lactate levels in the basal ganglia [11].

**Neoplastic**

*Bilateral Thalamic Glioma*

Bilateral Thalamic Glioma are rare, diffuse low-grade astrocytomas (WHO grade II), with variable presentation. They can be seen in children, typically producing intracranial hypertension symptoms, while in adults they usually present with behavioural alterations or dementia. Even with therapy, the prognosis is poor, owing to the deep location of the lesions [15, 20]. Moreover, high-grade gliomas (diffuse anaplastic astrocytoma and glioblastoma) have the potential to infiltrate both thalami **(Fig. 14)**. CT/MRI demonstrate either symmetrical or asymmetrical thalamic expansion, which may cause associated hydrocephalus [15, 20]. High-grade tumours originate from nearby structures or infiltrate them. MRS and MR perfusion-weighted imaging (PWI) may assist in differentiating from other similar appearing lesions [20].

**Other**

*Gadolinium deposition*

Several studies have demonstrated that numerous previous administrations of linear non-ionic gadolinium-based contrast agents have been associated with gadolinium deposition in the deep brain structures, including thalami and dentate nuclei. Gadolinium

deposition can be displayed on unenhanced images as increased T1 signal intensity in the aforementioned regions **(Fig. 15)** [21].

### Conclusion

Radiologists should be aware of how bithalamic processes might manifest on imaging. Radiological features that can be used in the assessment and differential approach include MR signal characteristics, calcifications, exact location within the thalamus, symmetry, presence of synchronous extra-thalamic involvement, and presence of expansion. Additional imaging tools such as DWI, MRA/MRV/CTA/CTV, MRS, and PWI may enhance characterisation. Correlating with clinical and laboratory findings must be sought at all times. At last, follow-up imaging might frequently offer valuable clues, especially when initial imaging is inconclusive. **R**

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### Conflict of interest

The authors declared no conflicts of interest.

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Arkoudis NA, Filippiadis DK, Toulas P, Velonakis G. Bilateral thalamic lesions: a pictorial essay. *Hell J Radiol* 2021; 6(3): 40-49.