Bilateral Thalamic and Right Fronto-temporo-parietal Gliomas in a 4 Years Old Child Diagnosed by Magnetic Resonance Imaging

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ABSTRACT

We report the neuroimaging findings of a 4-year-old girl with biopsy-proven bilateral thalamic and right fronto-temporo-parietal cortical gliomas, which are uncommon tumours involving the central nervous system. Despite their benignity, the prognosis is usually poor because of involvement of the thalamic nuclei and difficulty in surgical excision. These lesions have limited differential diagnoses that include metabolic, toxic, infective, vascular and neoplastic. Imaging characteristics on conventional Magnetic Resonance (MR), Magnetic Resonance Spectroscopy (MRS) and Diffusion tensor imaging (DTI) can further narrow the differential diagnosis and also provide additional information regarding the degree of involvement of adjacent brain tissue and white matter tracts around the lesions.

CASE REPORT

CASE REPORT

A 4-year-old girl presented with two episodes of afebrile seizures with tonic and clonic jerking of the upper limbs. The seizures responded to antiepileptics and subsided. The child has been previously well before the episodes. No history of trauma head and family history was negative. Cerebrospinal fluid (CSF) analysis was negative.

Imaging findings

The patient was admitted and underwent non- contrast CT scan of the brain that showed bilateral thalamic enlargement with mass effect and midline shift to the left by 9 mm (Fig. 1). Mild hydrocephalus was also noted. Urgent contrast enhanced conventional magnetic resonance imaging (MRI) of the brain with diffusion tensor imaging was performed (Fig.2, 3, 4). MRI data was processed to obtain fractional anisotropy (FA) maps using Siemens Neuro 3D task card software. MR images were analysed for lesion location and extension as well as displacement or destruction of brain tissue and white matter tracts (Fig.5). MRI revealed bilateral thalamic masses, right larger than left. They measure about 5.0 x 2.6 cm and 3.3 x 2.1 cm on the right and left thalamus respectively. The lesions were iso-hypointense on T1- weighted images (T1W) (Fig.2C, D) and hyperintense on T2- weighted (T2W) (Fig. 2A, B)/ fluid attenuated inversion recovery (FLAIR) images (Fig. 4D) relative to cerebral cortex. No enhancement after contrast administration was appreciated (Fig. 3). No restricted diffusion or susceptibility artifact was noted in the lesions (Fig. 4A-C). Asymmetrical dilatation of the ventricles with distortion of the lateral and third ventricles was also demonstrated. In addition, there was diffuse swelling and increased T2W (Fig.2) and FLAIR (Fig. 4D) signal in the right fronto-temporo-parietal cortex, with no obvious enhancement of the lesion (Fig.3).

DTI and FA demonstrated that the fiber tracts surrounding the lesions were displaced but not disrupted by the lesions. Fractional anisotropy maps showed no significant change in the perilesional white matter (Fig.5).

The MRS of the thalamic and right cortical lesions showed reduced N- acetyl aspartate (NAA) level, without prominent choline peak (Fig. 6).

Management

The patient underwent endoscopic biopsy of the thalamic masses and ventriculo-peritoneal shunt insertion, followed by open craniotomy biopsy of the right cortical lesion. The right cortical and thalamic lesions proved to be low grade gliomas (grade 2 of WHO classification). The intervening white matter between the right cortical and thalamic lesions appeared normal at the initial presentation and was not biopsied. In serial follow-up MRI imaging a new small right frontal periventricular white matter lesion was detected, separated from the initial cortical lesion.

The patient then received two cycles of chemotherapy and repeat MRI showed an increase in size of the tumours. At the time of this report no decision has been taken to give radiotherapy.

DISCUSSION

Etiology & Demographics

Pediatric supratentorial tumours represent about 50% of all intracranial neoplasms. The most frequent tumours of the cerebral hemispheres are gliomas that arise from astrocytes [1]. Primary tumors of the thalamus account for only 1-1.5% of all intracranial tumors and approximately 25% of them arise in children aged 15 years or under. However, their actual incidence has not yet been established and two recent reports provide figures ranging from 0.84 to 5.2% of all intracranial tumors [2,3,4].

The frequency of bilateral thalamic gliomas is less defined and about 66 cases have been reported till now [5]. Bilateral thalamic glioma with associated cortical glioma is extremely uncommon; has not been reported in English literature till date.

The majority of bilateral thalamic gliomas are usually low grade tumours (WHO grade II). However, grades III, IV and malignant transformation have also been encountered [2, 6].

Bilateral thalamic tumours have typical features and are considered to be distinct from unilateral tumours, probably they are not simply unilateral thalamic tumors and grow on both sides, as proven by their specific neuroradiological and metabolic properties, as well as a rapidly fatal clinical evolution. The unresponsiveness of these tumors to radiotherapy and chemotherapy treatment contributes further to distinguish these uncommon tumors from the relatively more common unilateral thalamic neoplasms [2]. However, the bilateral onset of a neoplastic process at the level of both thalami is not widely accepted. Some authors consider that bilateral involvement of the thalamic nuclei can occur due to spread of the tumour from either of the sides to the other. Others attribute this to the lateral spread of a tumour originating in the subependymal region of the third ventricles. However, some hypotheses report a connection across the midline through the posterior portion of the corpus callosum in autopsy series [2].

The age of presentation is between 20 months to 80 years with a mean of 39 years [4]; and it is less common in the paediatric age group. Few cases have been reported below the age of six years [2].

No gender predilection has been reported for these lesions.

Our case is a 4 year old girl who presents with the uncommon and atypical findings of bilateral thalamic and right fronto-temporo- parietal cortical gliomas, which are less common in these regions and less often seen in a bilateral manner with cortical involvement.

Clinical & Imaging findings

The clinical presentation is quite benign, symptoms and signs are mild inspite of the patient having large bilateral tumours. Raised intracranial pressure is usually not due to hydrocephalus, as hydrocephalus is generally absent or mild. The lesion causes mass effect on adjacent brain structures [2].

The clinical symptoms and signs are variable, depending on the involvement of different nuclei or tracts in this region. Hemiparesis, sensory disturbances, dysmetria, unsteady gait, torticolis, and nystagmus are the usual clinical features. In adults, severe dementia and personality modification were observed and attributed to involvement of the dorsomedial nuclei of the thalami and their connections with the temporal and frontal lobes. Furthermore, memory dysfunction and disorientation are attributed to anterior thalamic nuclei and mammillothalamic tracts involvement [2, 4].

In our case, the main clinical presentation was of afebrile seizure, which was related to the cortical involvement.

On CT scan, the tumours appear as bilateral, symmetrical, isodense, non enhancing thalamic lesions, with little or no mass effect. The absence of mass effect makes CT scan insufficient in delineating the extent of these tumours. On MRI, the lesions appear to be iso to hypointense on T1W and hyperintense on T2W and FLAIR images (relative to cerebral cortex), with no diffusion restriction or enhancement on post contrast study [2, 4].

MR spectroscopy can help to differentiate bilateral thalamic gliomas from other lesions [2, 4]. The intervening white matter between the abnormal right cortical and thalamic lesions was normal in serial follow-up MRI imaging.

In our case, the right cortical-subcortical lesion has an MRS pattern similar to those seen in the thalamic lesions. MRS showed increased creatine-choline peak, (creatine peak greater than choline peak) with decreased peak of NAA,

suggesting a low grade glioma. Neuronal loss is indicated by decrease in the NAA peak [4,7].

Diffusion tensor imaging (DTI) imaging and fiber tractography are relatively new MR imaging techniques based on the concept of anisotropic water diffusion in myelinated fibers, which enable visualization of white matter tracts and provide information about the relationship of these fiber tracts to the mass [8]. Tractography and FA values have been used to identify a tumour's effect on fiber tracts, such as disruption, displacement, infiltration or oedema. This can be used to improve presurgical/ biopsy planning by minimizing iatrogenic injury of important functional tracts and also can help to classify the histopathologic and behavioural characteristics of the tumour.

In our case, there was a cortical and subcortical right fronto-temporal lobe lesion, which had MRI signal characteristics similar to those in the thalamic lesions with no diffusion restriction. Mild hydrocephalus and midline shift to the left were also appreciated. MRS showed decreased NAA peak with no choline elevation. DTI and FA demonstrated displacement and distortion rather than disruption of the descending motor fiber tracts, on the right side more than the left, with fiber integrity being maintained. This is in keeping with a lower grade or less aggressive neoplasm. These MRI characteristics can help to differentiate between bilateral thalamic glioma and other thalamic lesions in this age group such as vascular, infectious or metabolic causes.

Histologically, most cases have been low grade gliomas (grade 2 of WHO classification) [9]. In our case, the cortical and thalamic lesions proved to be low grade gliomas (grade 2 of WHO classification).

Treatment & Prognosis

Because of diffuse and bilateral involvement of the thalamus, surgical excision is very difficult and no case of radical removal has been reported. Thus, the only role of surgery is to get tissue for histological diagnosis, which can be obtained by open surgery, endoscopically or under stereotactic guidance [2]. In cases of a large tumour causing significant mass effect, debulking of the lesion can be attempted. If hydrocephalus exists, biventricular shunting is an option. The role of adjuvant radiotherapy or chemotherapy is not established and the outcomes are usually poor [2, 3]. In our case, the patient underwent endoscopic biopsy of the thalamic lesions and open craniotomy for biopsy of the right fronto-tempro-parietal cortical lesion. A ventriculo-peritoneal shunt was also inserted. Two cycles of chemotherapy were given and repeat MRI showed increase in the size of the tumours. At the time of this report no decision has been taken to give radiotherapy.

The prognosis is usually poor irrespective of treatment [2].

Differential Diagnosis

The lesions in our case had MRI features described for bilateral thalamic and right fronto-temporo-parietal and these lesions have a differential diagnosis that includes metabolic, infection and vascular lesions, although these each have distinct imaging features when considering multiple imaging modalities (Table 2) [3, 4, 9].

Infection: Flavivirus such as Japanese encephalitis presents with prodromal phase of fever, rigors, headache, rashes, and body aches followed by CNS symptoms like dyskinesia, dystonia, tremors, drooling, dysarthria, altered consciousness, seizures, and coma. The diagnosis depends on the detection of antibodies in the serum and CSF. MR characteristics are T2 hyperintensity, with bilateral involvement of the posteromedial thalamus. However, basal ganglia, substantia nigra, red nucleus, pons, hippocampi, cerebral cortex and cerebellum can be involved. Also, intralesional hemorrhages and restricted diffusion have been described [10].

Wernicke Encephalopathy: Frequently it associated with alcohol abuse. The classic clinical triad is ataxia, altered consciousness and abnormal eye movements. MRI features show symmetric T2-weighted high signal intensity in the mammillary bodies, medial aspects of the thalami, tectal plate, periaqueductal gray matter, and dorsal medulla. Contrast enhancement can be seen in mammillary bodies. It can have restricted diffusion owing to ischemia-like changes in the thalami [11,12].

Leigh Disease: This is a genetically heterogeneous mitochondrial disorder that causes progressive neurodegeneration leading to respiratory failure and death in childhood. Elevation of the lactate in the CSF, serum, and urine is noted. On T2-weighted images hyperintensity may be noted in brain stem, periaqueductal grey matter, medulla, midbrain and putamen [13]. MR spectroscopy shows a decreased level of NAA with elevated choline and lactate levels. Contrast enhancement is uncommon. Restricted diffusion may be seen in the acute stage.

Vascular: Bilateral thalamic arterial infarcts are uncommon. Deep venous thrombosis typically results in bilateral symmetric involvement of the thalami and occasionally the basal ganglia. Causes include pregnancy, oral contraceptives, infection, trauma, and dehydration. A hyperdense vein may be seen on CT scan, and corresponding T1 hyperintensity from clot in the sinuses may be seen on MR images. High T2 signal intensity with loss of normal signal void in deep venous sinuses can also be identified. CT and MR venography show no areas of contrast enhancement or signal intensity in the deep venous sinuses. Diffusion restriction on DWI has been described [14]. Patchy contrast enhancement may be also seen.

In conclusion, clinical presentation and characteristic MRI and MRS findings can help to diagnose thalamic glioma; particularly if it is not associated with diffusion restriction, marked enhancement or rise of choline on MRS.

TEACHING POINT

MRI is the modality of choice for diagnosis of bilateral thalamic and cortical gliomas based on a combination of findings; they appear iso- hypointense on T1W and hyperintense on T2W (relative to cerebral cortex) with no perilesional oedema, enhancement or diffusion restriction demonstrated by the lesions. In addition, there is elevation of the creatine –NAA ratio on MRS sequences with displacement, rather than disruption of the adjacent white matter fiber tracts on tractography and fractional anisotropy imaging.

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FIGURES

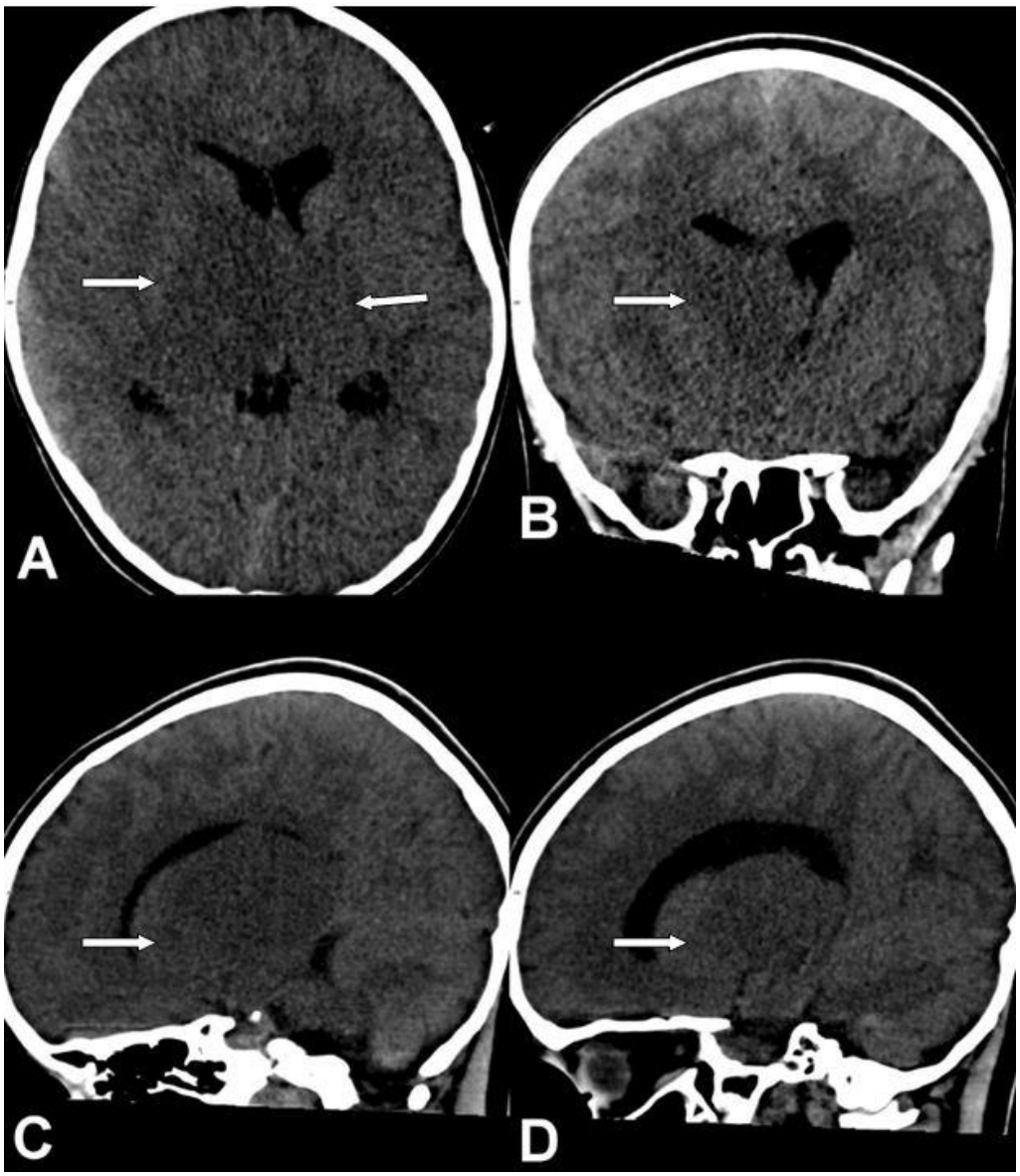

Figure 1: A 4- year-old female with bilateral thalamic and right fronto-temporo-parietal cortical gliomas.
Findings: axial(A), coronal (B), right parasagittal (C) and left parasagittal (D) images demonstrating bilateral thalamic enlargement with hypodense lesions (white arrows) causing mass effect and midline shift to the left. Mild hydrocephalus is also noted.
Technique: 64 slice CT (Toshiba, Aquilion 64 system), 3 mm slice thickness, 100 KV, 178 mAs, non-contrast.

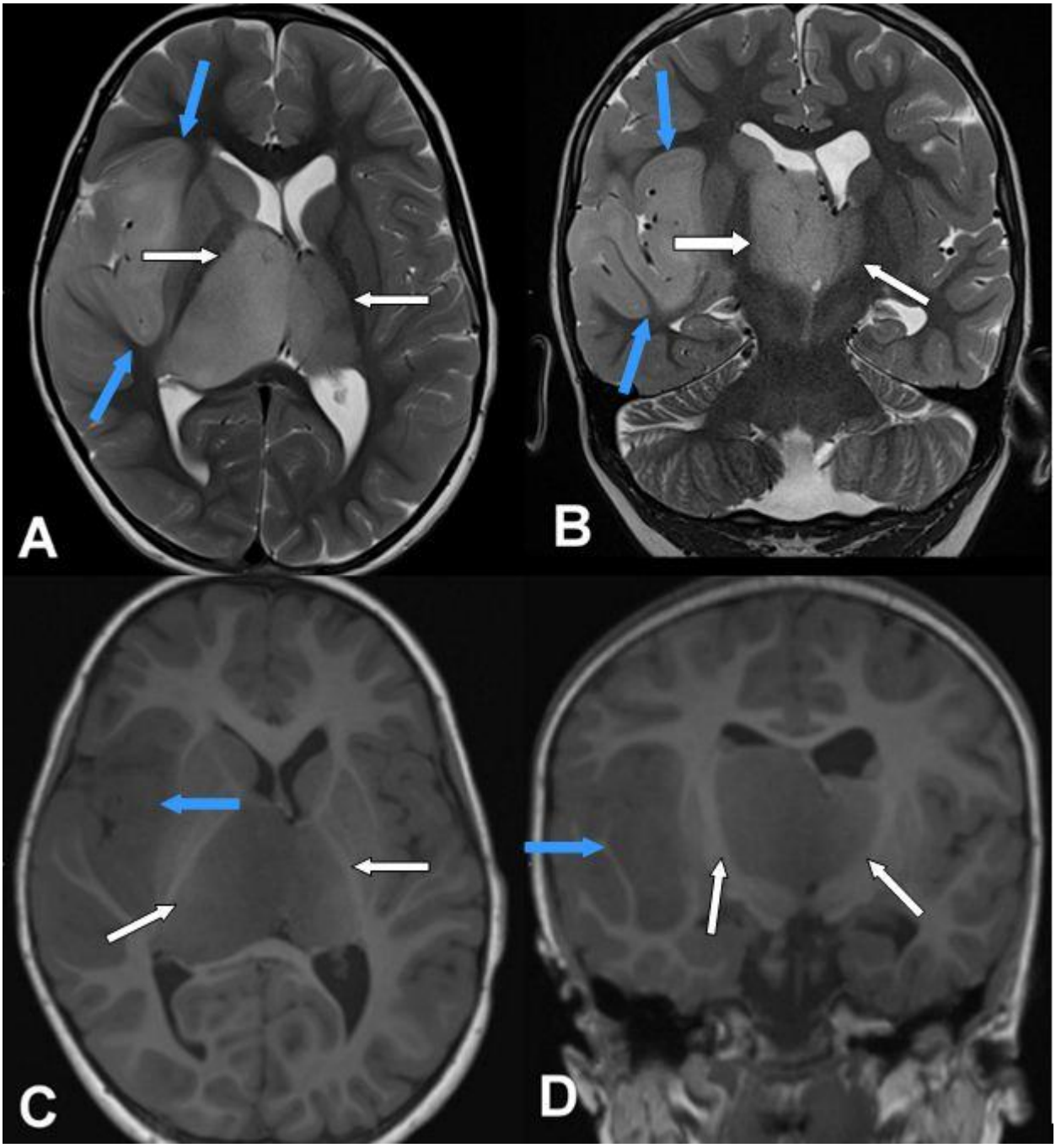

Figure 2: A 4- year-old female with bilateral thalamic and right fronto-temporo-parietal cortical gliomas.
Findings Fig. A&B:
Axial (A) and coronal (B) T2W images demonstrate homogenous swelling and enlargement of both thalami, more on the right. They are showing diffuse hyperintense T2W signal, relative to cerebral cortex (white arrows). Midline shift to the left with mild asymmetrical dilatation of the lateral ventricles is appreciated. The right lesion measures approximately 5.0 x 2.6 cm, while the left lesion measures 3.3 x 2.1 cm. In addition, there is diffuse swelling and increased T2W signal intensity in the right fronto-temporo- parietal cortex (blue arrows). No evidence of associated vasogenic oedema.
Technique: Acquisition 3.0 Tesla MRI (Siemens system). T2W axial A and coronal B images Repetition time/Echo time TR/TE: 5000/96 msec., 5mm slice thickness with field of view (FOV) 21 cm and 2 mm slice thickness with FOV 17cm, for T2W axial and coronal images respectively, non-contrast.
Findings Fig. C&D:
Axial (C) and coronal (D) non- contrast T1W images demonstrate the lesions, which appear to be iso-hypointense to the cerebral cortex (white and blue arrows).
Technique: Acquisition 3.0 Tesla MRI (Siemens system).non- contrast T1W axial C and coronal D images repetition time/echo time TR/TE: 1900/900 msec., 1mm slice thickness, non-contrast, field of view 21 cm.

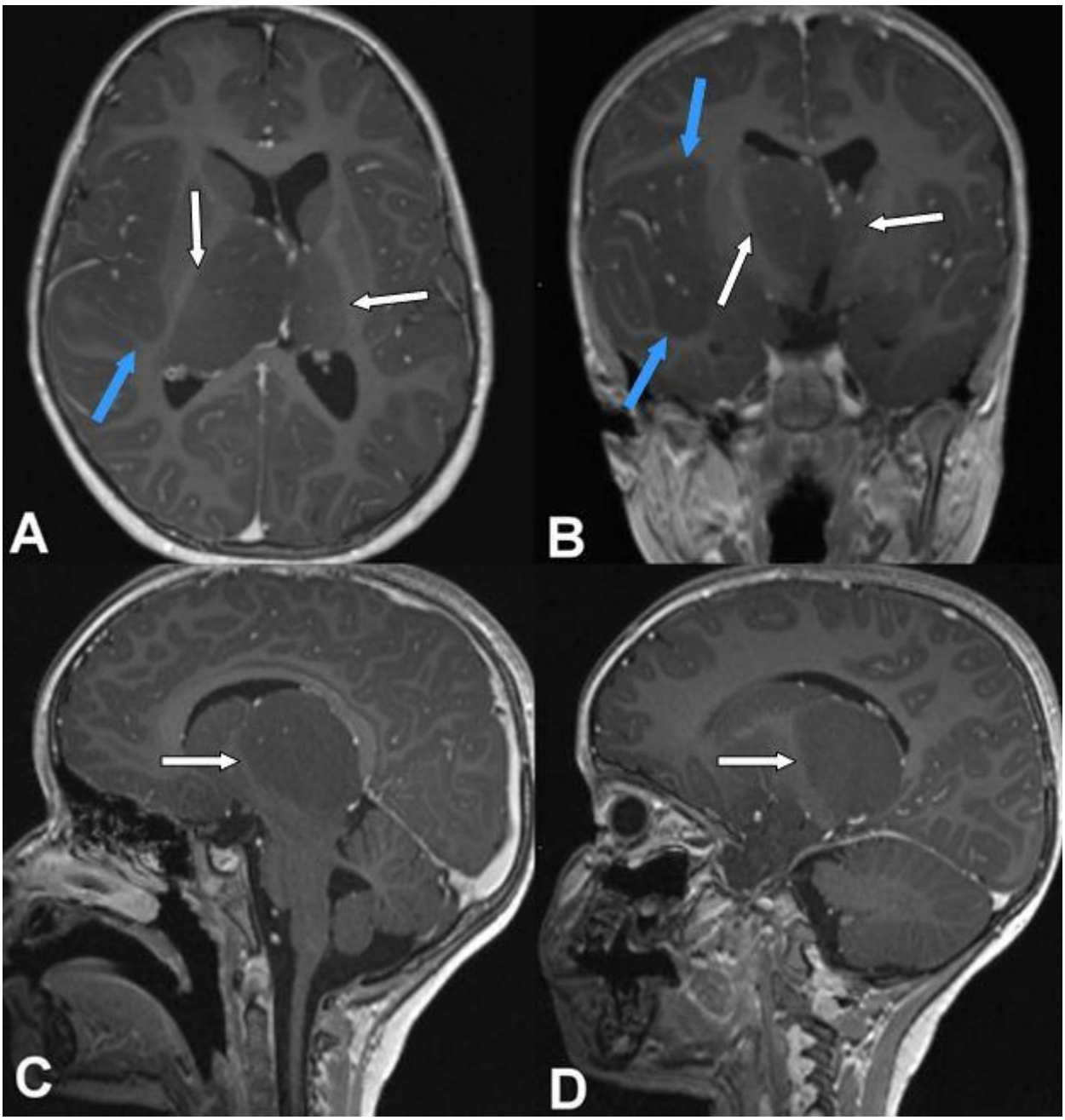

Figure 3: A 4- year-old female with bilateral thalamic and right fronto-temporo-parietal cortical gliomas.
Findings Fig. A, B, C& D: Post-contrast axial (A), coronal (B), sagittal (C) and right para-sagittal (D) T1-weighted images illustrate bilateral thalamic (white arrows) and right fronto-temporo- parietal cortical lesions (blue arrows), with no definite contrast enhancement. Mild hydrocephalus is noted.
Technique: Acquisition 3.0 Tesla MRI (Siemens system), T1W post- cont. axial, coronal, sag.& right parasag. TR/TE: 1900/900 msec., 1mm slice thickness for axial and coronal and 0.9 mm slice thickness for sagittal images, field of view 21 cm, Gadavist: 0.1 mmol/kg.).

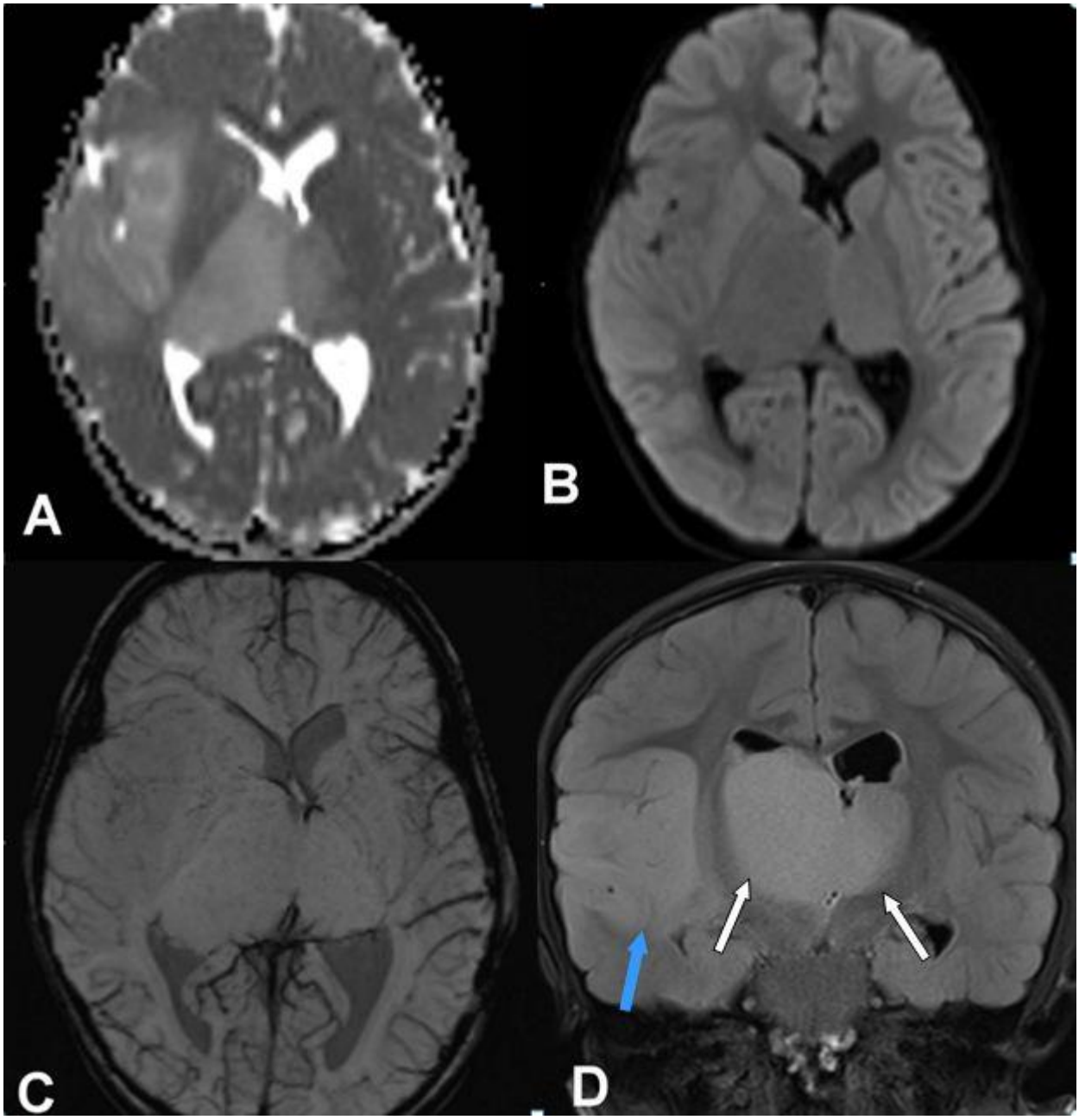

Figure 4: A 4- year-old female with bilateral thalamic and right fronto-temporo-parietal cortical gliomas.
Finding Fig. A&B: Axial ADC (A)& DWI (B) demonstrate no definite restricted diffusion of the bilateral thalamic and right fronto-temporo-parietal cortical lesions.
Technique: acquisition 3.0 Tesla MRI (Siemens system). ADC (A) and DWI(B) images, TR/TE: 6800/74 msec., 5mm slice thickness, field of view 21 cm, B value =1000,non-contrast.
Findings Fig. C:
Axial SWI (Susceptibility Weighted Imaging) illustrate no signal loss or `blooming` artifacts, indicating no evidence of calcification or haemorrhage within the lesions.
Technique: acquisition 3.0 Tesla MRI (Siemens system). SWI (C) images TR/TE: 27/20 msec., 2mm slice thickness, field of view 21 cm, non-contrast.
Findings Fig. D:
Coronal FLAIR images. The lesions appear to be slightly hyperintense relative to cerebral cortex (white arrows for thalamic lesions and blue arrow for right cortical lesion). Mild hydrocephalus and mid-line shift to the left are also demonstrated.
Technique: acquisition 3.0 Tesla MRI (Siemens system). Coronal FLAIR (D) images. TR/TE: 8000/108 msec., 5mm slice thickness, non-contrast, field of view 21 cm.

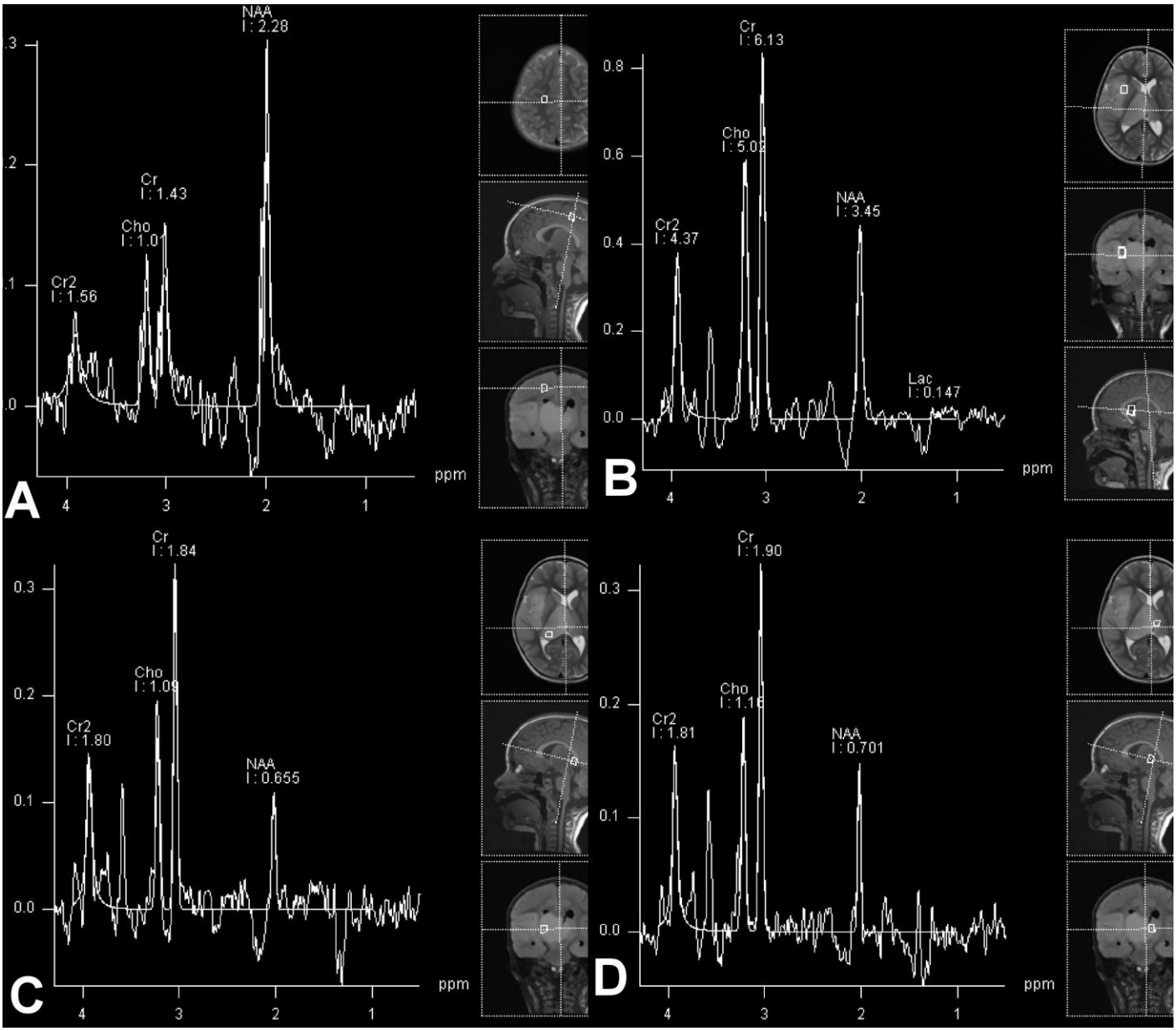

Figure 5: A 4- year-old female with bilateral thalamic and right fronto-temporo-parietal cortical gliomas.
Findings (A): MR spectroscopy of the normal right parietal white matter (for comparison) (A) showing normal N- acetyl aspartate (NAA) peak.
Findings (B),(C)&(D):The right fronto-temporo-parietal cortical glioma (B), right (C) and left (D) thalamic lesions illustrating reduced N- acetyl aspartate (NAA) level without dominant choline peak, consistent with neural damage.
Technique: Acquisition 3.0 Tesla MRI (Siemens system). MR spectroscopy was performed by using csislaser, selecting voxel size 10.0 x 10.0 x 15.0 mm^3, TR/TE: 1700/135 msec., non-contrast.

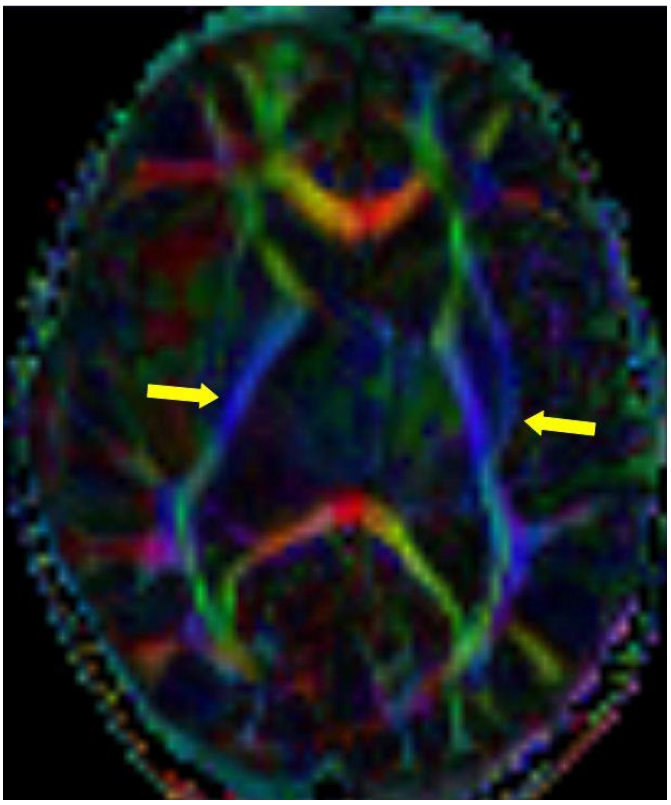

Figure 6 (left): A 4- year-old female with bilateral thalamic and right fronto-temporo-parietal cortical gliomas.
Findings: Axial diffusion tensor images with Fractional Anisotropy (FA) map showing deformed and displaced but intact white matter descending motor fiber tracts which are curving around and surrounding the lesions (yellow arrows).
Technique: Acquisition FA was processed with Siemens Neuro 3D task card software after acquiring data from 3.0 Tesla MRI, Siemens system, TR/TE: 6800/74, 2 mm slice thickness, field of view 25.6 cm, b-value1000 s/mm², number of directions 30.

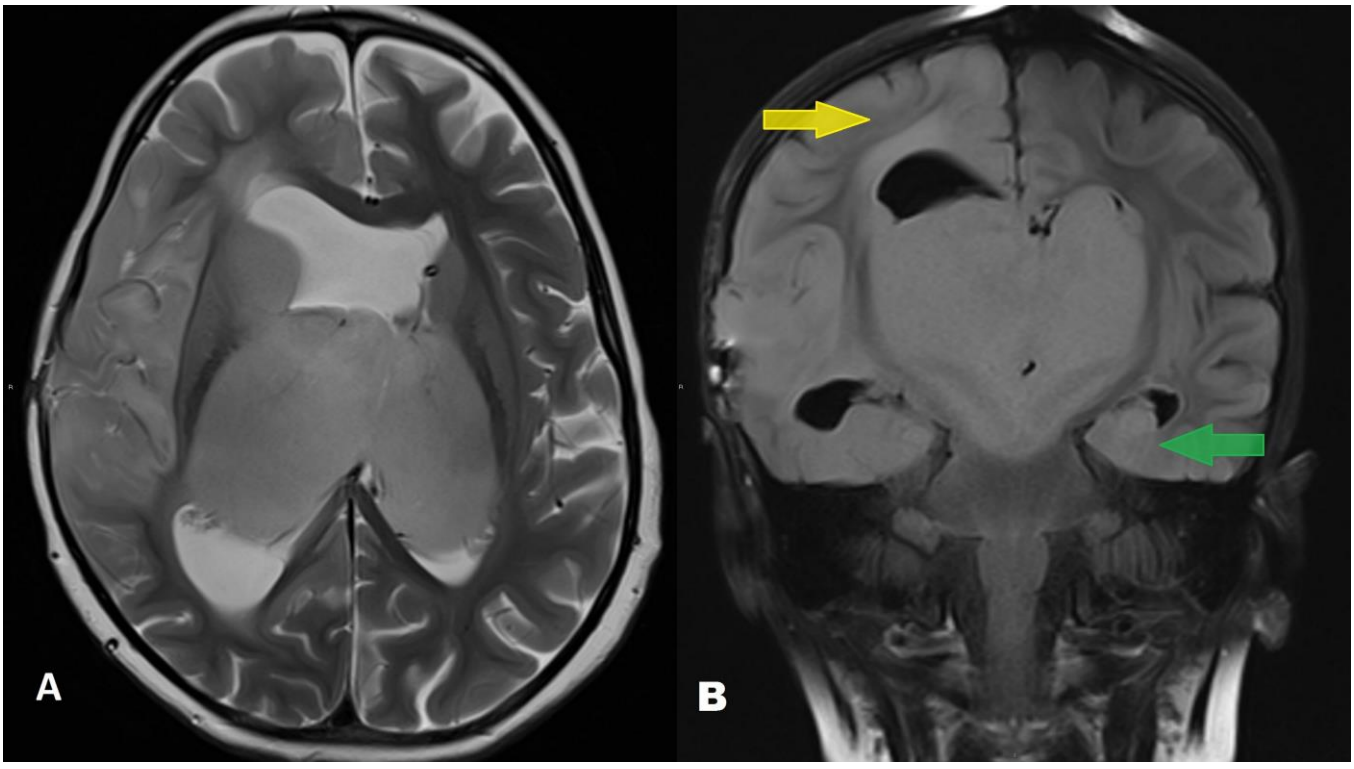

Figure 7: A 4- year-old female with bilateral thalamic and right fronto-temporo-parietal cortical gliomas.
(Follow up MRI after one year of chemotherapy).
Findings Fig. A: Axial (A) T2W images. There is interval increase in size of the bilateral thalamic and right fronto-temporo-parietal masses. There is new involvement of the right caudate nucleus. The hydrocephalus appears more prominent.
Technique: Acquisition 3.0 Tesla MRI (Siemens system). T2W axial A B images Repetition time/Echo time TR/TE: 5000/96 msec., 5mm slice thickness with field of view (FOV) 21 cm and 2 mm slice thickness with FOV 17cm, for T2W axial and coronal images respectively, non-contrast.
Findings Fig. B: Coronal (B) FLAIR images. The lesions appear larger with new involvement of the right frontal periventricular white matter (yellow arrow) and medial left temporal lobe (green arrow).
Technique: acquisition 3.0 Tesla MRI (Siemens system). Coronal FLAIR (D) images. TR/TE: 8000/108 msec., 5mm slice thickness, non-contrast, field of view 21 cm.

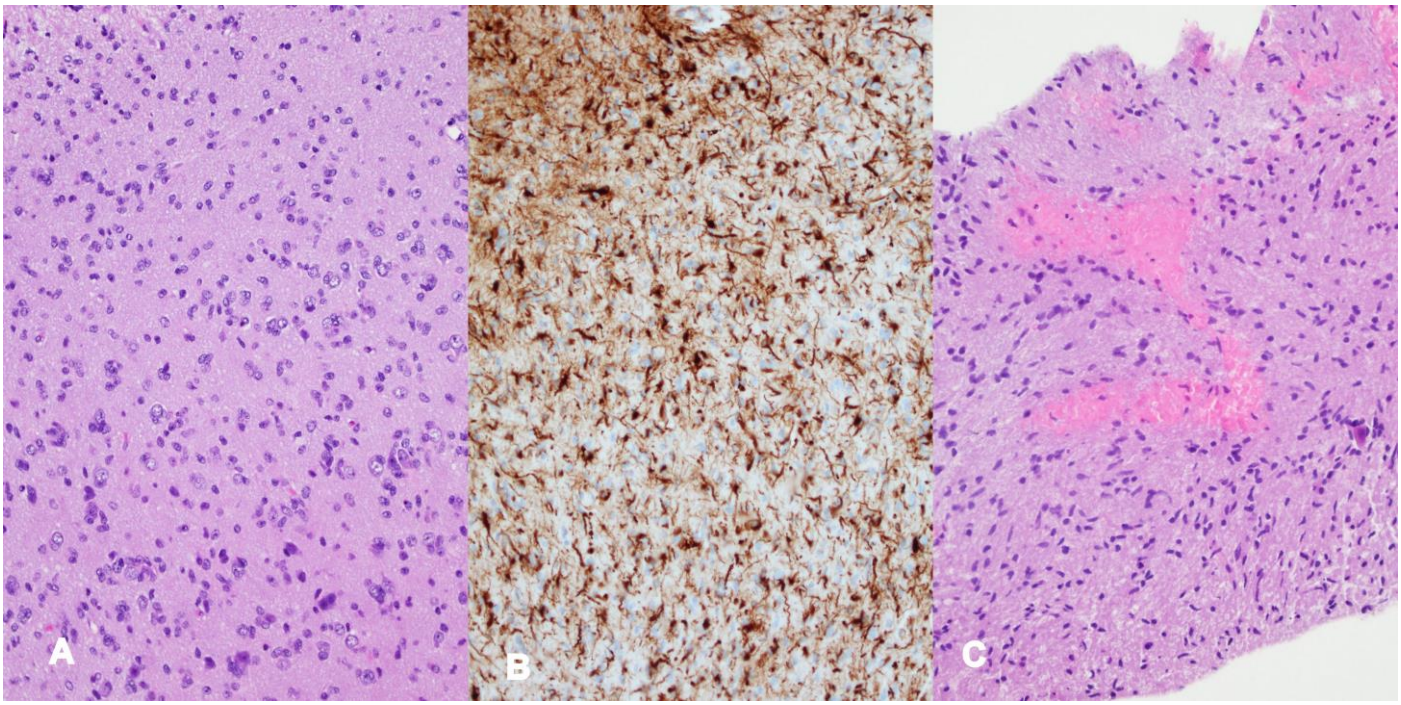

Figure 8: A 4- year-old female with bilateral thalamic and right fronto-temporo-parietal cortical gliomas.
(A): Temporal lobe cortex infiltrated by diffuse astrocytoma (WHO grade II) with subpial spread and perineuronal and perivascular satellitosis, hematoxylin and eosin (H&E), high power (200x).
(B): Temporal lobe cortex. Infiltrating tumor cells show strong diffuse immunereactivity for WT1, high power (200x).
(C): Thalamus infiltrated by diffuse astrocytoma with perineuronal satellitosis, (H&E), high power (200x).

Aetiology	• Usually diffuse low-grade astrocytoma (World Health Organization grade II).
Age of predilection	• Primary tumors of the thalamus account for only 1-1.5% of all intracranial tumors and approximately 25% of them arise in children aged 15 years or under. • The frequency of bilateral thalamic gliomas is less defined and about 66 cases have been reported till now. The age of presentation varies from 20 months to 80 years, but is less common in the paediatric age group and few cases have been reported below six years of age. • Bilateral thalamic glioma with associated cortical glioma is uncommon and has not been previously reported.
Gender ratio	• No gender predilection.
Risk factors	• Unknown.
Treatment	• Chemo and radiotherapy and decompression for hydrocephalus.
Prognosis	• Poor outcome due to location.
Presentation	• Children: Present with signs of increased intracranial pressure and movement disorder. • Adult: Present with mental changes and deterioration.
Imaging findings	• MRI: Typically, swelling and expansion of both thalami accompanied by abnormal hyperintensity on T2W and FLAIR images and iso-hypointensity on T1W images (relative to cerebral cortex). No associated contrast enhancement or restricted diffusion. The cortical lesion has the same MRI signal characteristics. Hydrocephalus depends on the degree of mass effect. • MRS: low NAA peak, without dominant choline peak. • DTI: Displacement without disruption of the white matter fiber tracts.

Table 1: Summary table for bilateral thalamic and cortical gliomas.

Entity	Clinical features	CT findings	MRI findings
Bilateral thalamic and cortical gliomas	Depending on the involvement of different nuclei or tracts in this region. Hemiparesis, sensory disturbances, dysmetria, unsteady gait, torticollis, and nystagmus are the usual clinical features in children.	Bilateral, symmetrical, isodense, non-enhancing thalamic lesions,	• T1 iso-hypointense. • T2/FLAIR hyperintensity involving the thalami. • No enhancement. • DWI shows no diffusion restriction. • MRS shows increased creatine-choline peak, with decreased peak of NAA, suggesting a low grade glioma. • The cortical lesion has MRI characteristics similar to the thalamic lesions. • DTI &FA can be used to improve presurgical/ biopsy planning
Infection	Viral encephalitis mostly Japanese encephalitis (JE). It presents with prodromal phase of fever, rigors, headache, rashes, and body aches followed by CNS symptoms like dyskinesia, dystonia, tremors, drooling, dysarthria, altered consciousness, seizures, and coma. The diagnosis depends on the detection of antibodies in the serum.	Ill-defined low density in both thalami.	• T1 hypointensity. • T2 hyperintensity involving the postero-medial thalami, basal ganglia, hippocampi, substantia nigra, cerebral cortex and cerebellum. • Enhancement not usually observed. • Intralesional hemorrhages and restricted diffusion have been described
Vascular occlusion	Usually due to deep venous thrombosis (bilateral thalamic arterial infarcts are uncommon). This may be due to pregnancy, oral contraceptive, trauma and dehydration	Hyperdense vein due to intraluminal clot.	• T1 hyperintensity from clot in the sinuses. • T2 hyperintensity in both thalami and occasionally in the basal ganglia, with loss of normal signal void in deep venous sinuses. • MR venography shows filling defect in the deep sinuses. • DWI shows restricted diffusion. Patchy contrast enhancement may be seen.
Leigh Disease	Onset usually before age of 2 years with psychomotor delay / regression leading to respiratory failure and death in childhood. There is elevation of the lactate in the CSF, serum, and urine.	Regions of low density matching areas of abnormal T2 signal on MRI. Occasionally some of these areas can demonstrate contrast enhancement	• T2W: hyperintense signal in brain stem, periaqueductal grey matter, medulla, midbrain and putamen. • T1W: usually demonstrates reduced signal in T2 abnormal areas, although some areas of hyperintensity and enhancement can be seen. • DWI: in the acute stage some restricted diffusion may be evident. • MR spectroscopy: - features include elevated lactate and choline peaks and reduced NAA.
Wernicke Encephalopathy	Older age group, with alcohol abuse. Characterized by triad of acute confusion, ataxia and ophthalmoplegia	Usually Normal	• T2W: hyperintense signal in mammillary bodies, medial aspect of the thalami, tectal plate, dorsal medulla, and periaqueductal grey matter. • DWI: Can have restricted diffusion. Contrast enhancement can be seen in mammillary bodies

Table 2: Differential diagnosis for bilateral thalamic and cortical gliomas.

ABBREVIATIONS

ADC = Apparent coefficient map
Cho = Choline
Cr = Creatine
CSF = cerebrospinal fluid
CT = Computed Tomography
DWI = Diffusion-Weighted Imaging
Fig = Figure
FLAIR = Fluid Attenuated Inversion Recovery
FOV = Field Of View
H&E = Hematoxylin and Eosin
KV = kilovoltage
mAs = milliampere second
MRI = Magnetic resonance imaging
MRS = Magnetic Resonance Spectroscopy
msec = millisecond
NAA = N-acetyl-aspartate
parasag = Parasagittal
post-cont = post-contrast
sag = Sagittal
SWI = Susceptibility-Weighted Imaging
T1W = T1 weighted
T2W = T2 weighted
TE = Time to echo
TR = Repetition time

KEYWORDS

Bithalamic gliomas; cortical gliomas; intracranial mass; paediatric seizures; magnetic resonance imaging

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