

# Bilateral astrocytomas: Clinicoradiological characteristics and endoscopic management

Sanjay Behari, Vivek Vaid, Namit Singhal, S Jaypal Reddy, Deepu Banerji

**Abstract:** Bithalamic astrocytoma is an extremely rare condition associated with progressive neurobehavioural syndrome and dementia. We report a 17-year-old boy who presented with headache and memory loss. His magnetic resonance imaging revealed large bithalamic, symmetrical, non-enhancing lesions with moderate hydrocephalus and tonsillar herniation. Magnetic resonance spectroscopy revealed high choline and creatinine peaks. An endoscopic guided biopsy and ventriculoperitoneal shunt placement was followed by radiotherapy. The biopsy revealed grade II fibrillary astrocytoma. He was initially relieved of raised pressure symptoms but developed tremors and progressive neurobehavioural dysfunction and died after 3 years. (p98-103)

**Key words:** Astrocytomas, brain tumours, magnetic resonance imaging, magnetic resonance spectroscopy, thalamus, thalamic tumours and endoscopy

## Introduction

This 17-year-old boy had headache for one year, memory loss and decreased scholastic performance for 6 months prior to his admission. On examination, he was conscious and oriented but with recent memory loss. There were no other motor or sensory deficits or cerebellar signs. T1- and T2-weighted MRI revealed bilateral large thalamic gliomas (dimension 7 cm × 5 cm × 5 cm) with interconnection through the interthalamic adhesion. The lesions had occupied the third ventricle reaching up to the posterior third ventricular region and the foramen of Monro. Only a small part of the anterior third ventricle was free. There was spread on the right side to the head of the caudate nucleus. The tumours were uniformly isointense on T1-weighted image except for a small area of hypointensity on the posterior part of the tumour on the right side. The lesions were reaching up to the midbrain with no well-defined plane from the neuraxis. There was no perifocal oedema. There was no spread of the lesion to the medial temporal or the frontal region. The lesions were hyper-

intense on T2-weighted image. There was no contrast enhancement on gadolinium administration. The lateral ventricular system was slightly dilated but with no associated periventricular lucency. There was associated tonsillar herniation (Figs. 1a, b, c and d).

The water-suppressed proton MRS of the tumours done with a point-resolved spectroscopic sequence (PRESS) revealed choline and creatinine peaks with the creatinine peak appearing greater than the choline peak. The N-acetylaspartate signal was decreased. A small lactate peak was also observed (Fig. 2). Two months prior to admission, he developed severe headache and tremors of the right upper limb. The lapses of recent memory worsened. He also had bilateral papilloedema with bilateral VI<sup>th</sup> nerve paresis. A repeat MR scan revealed increased lateral ventricular dilatation but with no obvious increase in the size of the thalamic lesions.

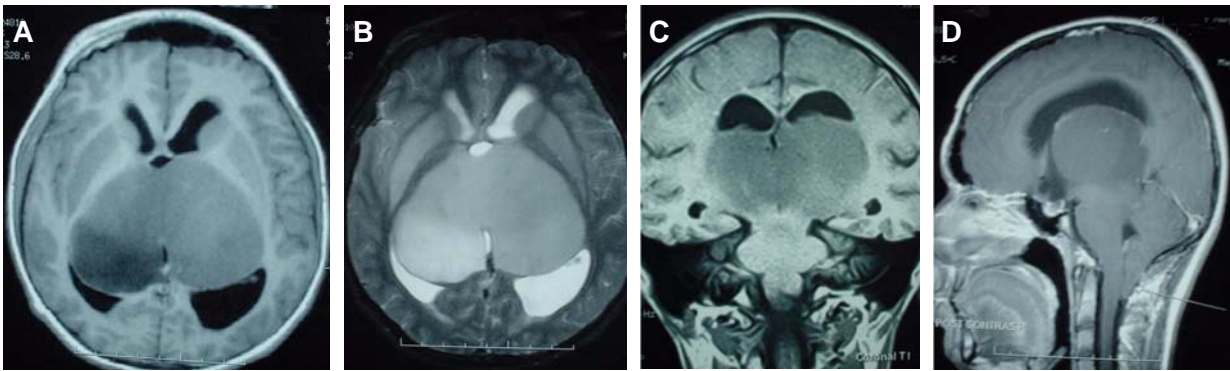
The patient was placed on phenytoin 100 mg three times a day and injection dexamethasone 4 mg 6 hourly prior to surgery.

At surgery, left and right parietal burr holes were made. Through the right parietal burr hole, a neuroendoscope was introduced into the right lateral ventricle. The tumour was identified bulging into the lateral ventricle. The ventricular ependymal line had been breached at a few places revealing a grayish-pink avascular lesion (Fig. 3). Biopsy was taken using a biopsy forceps introduced through the endoscope. Through the same burr hole, a ventricular catheter of the “Chhabra” medium pressure ventriculoperitoneal shunt was introduced into the right lateral ventricle and its position

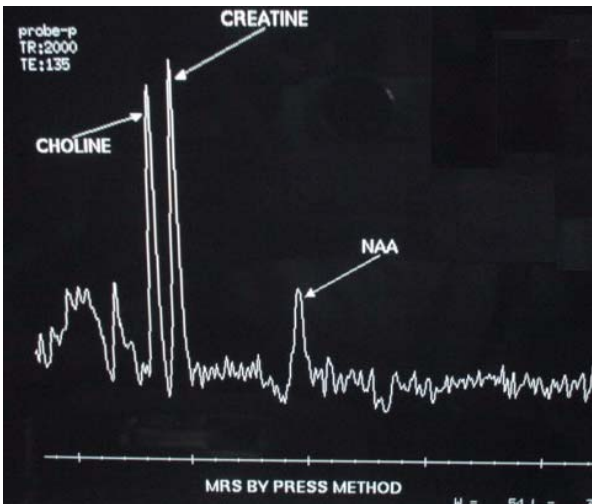
Department of Neurosurgery  
Sanjay Gandhi Institute of Postgraduate Medical Sciences,  
Lucknow  
India

### Correspondence:

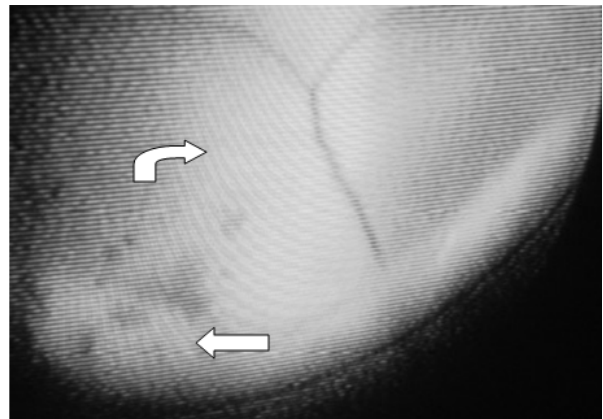
Dr. Sanjay Behari  
Department of Neurosurgery  
Sanjay Gandhi Postgraduate Institute of Medical Sciences  
Rae Bareilly Road  
Lucknow, UP - 226014  
India  
Tel: (91 52) 2266 8700 Ext. 2107 / 2741  
Fax: (91 52) 2266 8078 / (91 52) 2266 8017  
Email: sbehari@sgpgi.ac.in / sbehari27@yahoo.com



**Figure 1a** - Axial T1-weighted MRI showing bilateral large thalamic gliomas with interconnection through the interthalamic adhesion. There was spread on the right side to the head of the caudate nucleus. The tumours are uniformly isointense on T1-weighted image except for a small area of hypointensity on the posterior part of the tumour on the right side. **(b)** Axial MRI showing nearly uniform hyperintensity on T2-weighted image. **(c)** Coronal T1-weighted MRI showing the two lesions relatively confined to the thalamus with no well defined plane of cleavage from the surrounding neuraxial structures. **(d)** Postcontrast T1-weighted MRI showing no contrast enhancement. There is evidence of tonsillar herniation.



**Figure 2** - The MRS revealed choline and creatinine peaks with the creatinine peak appearing greater than the choline peak. The N-acetylaspartate signal was decreased. A small lactate peak was also observed.



**Figure 3** - A neuroendoscope introduced into the right lateral ventricle showed the bulging tumour (curved arrow) with the ventricular ependymal line breached at a few places revealing a grayish-pink avascular lesion (straight arrow) from where the biopsy was taken

confirmed using the endoscope. Through the left parietal burr hole, another ventricular catheter was placed in the left lateral ventricle and also confirmed using the endoscope. The two ventricular ends were connected to the “Chhabra” medium pressure ventriculoperitoneal shunt using the Y connector. The abdominal end of the shunt was placed in the peritoneal cavity using a para-umbilical incision.

The biopsy of the lesion revealed a fibrillary astrocytoma [World Health Organization (WHO) grade II]. There was no evidence of necrosis, mitotic figures or endothelial proliferation.

Following the ventriculoperitoneal shunt procedure, he was relieved of headache and vomiting. However, the VI<sup>th</sup> nerve palsy, tremors in right hand and memory deficits persisted.

He was also administered 55 Gy radiotherapy in the tumoural area. A 1-year follow-up revealed a stable neurological status with persisting memory disturbance and tremors. However, after approximately 1.5 years following the procedure, there was progression of cognitive disturbances and disorientation. He also became completely apathetic to his surroundings. He finally died after lapsing into altered

sensorium 3 years after the initial diagnosis.

## Discussion

### Incidence

Thalamic astrocytomas account for 0.84 - 5.2% of all cerebral tumours.<sup>5,18,24</sup> A review of literature has revealed that to date, 40 cases (including the present case) of bilateral thalamic astrocytomas have been reported (Table 1).<sup>5,6,9-15,17-19,23,25</sup> Thirteen of these 40 patients (32%) were less than 18 years of age.<sup>5,9,14,17,19</sup> The majority of lesions with bithalamic involvement have been low grade astrocytomas (WHO grade II), as in present study.<sup>5,9,12-14</sup> However, grades III and IV have also been encountered and malignant transformation of a low grade lesion may also occur.<sup>5,9,11,14,17,19,23</sup> In the present case, the bithalamic tumours had a contiguous spread to the opposite side through the interthalamic adhesion, completely filling up the body of the third ventricle. Spread to the opposite side across the enlarged midbrain has also been reported.<sup>5</sup> In cases of strictly

symmetrical tumours localized in both thalami, a bilateral origin has also been proposed.<sup>3,14</sup>

### Clinical presentation

Our patient having a bithalamic astrocytoma grade II was fortunate to survive for approximately 3 years after the initial diagnosis, as was seen in the case reported by Kouyialis, et al.<sup>12</sup> The histological grade of the tumour forms the most important prognostic indicator in thalamic astrocytomas and both our patient, as well as the one reported by Kouyialis, et al had a low grade astrocytoma (grade II).<sup>12,18,25</sup> The literature, however, reports a very poor prognosis for the majority of bithalamic low grade astrocytomas.<sup>5,18</sup> One striking feature in both these cases (with a relatively longer survival) was the presence of early headache even when hydrocephalus had not developed. Perhaps recurrent cerebrospinal fluid pathway obstruction had led to the headache and had facilitated early detection of the tumours before the latter had infiltrated into the

**Table 1** - Review of case studies of bilateral thalamic gliomas

No. of cases	Author, year	Age (in years)	Symptoms	Glioma grade	Hydrocephalus
1	Ziegler, et al 1977 <sup>25</sup>	59	Memory impairment	IV	None
1	Gutmann, et al 1990 <sup>10</sup>	27	Personality change	-	None
2	Ruel, et al 1992 <sup>19</sup>	70 8	Disorientation Personality change	IV III	None None
8	Partlow, et al 1992 <sup>17</sup>	8 15 15 35 49 53 67 68	Slow mentation Personality change Personality change Memory loss, loss of interest Memory loss, confusion Personality change, memory loss Progressive dementia Disorientation, confusion, memory loss	I, II - III, IV I, II III, IV II I, II -	None None Mild Mild Moderate Mild Mild Mild
12	Reardon, et al 1998 <sup>18</sup>	-	-	75% I & II	-
2	Esteve, et al 1999 <sup>6</sup>	31 21	Decreased visual acuity, hemianopia, personality change Decreased visual acuity, personality change, memory loss	II II	None None
1	Hirano, et al 2000 <sup>11</sup>	63	Headache, memory impairment	III	Mild
1	Nagratnam, et al 2001 <sup>15</sup>	64	Personality change, ataxia	-	None
1	Uchino, et al 2002 <sup>23</sup>	35	Hemidysaesthesia, memory loss, slow mentation	III	None
4	Di Rocco, et al 2002 <sup>5</sup>	10 6 3months 10	Headache, vomiting, right hemiparesis Headache, vomiting Bulging fontanelles, nystagmus, intentional tremor, torticollis Tremors	II II II III	None None Mild None
2	Messing-Junger, et al 2002 <sup>14</sup>	2 10	Ataxia, dysarthria, personality change Headache	II III	None None
2	Gudowius, et al 2002 <sup>9</sup>	20 months 11	Psychomotor regression, progressive ataxia Ataxia, headache, vomiting	II III	Moderate None
1	Lagares, et al 2004 <sup>13</sup>	67	Personality change, memory disturbance	II	None
1	Kouyialis, et al 2004 <sup>12</sup>	65	Personality change	II	None
1	Behari, et al 2006 (present study)	17	Memory loss, headache	II	Moderate

surrounding thalamic nuclei and caused severe neuro-behavioural disability. This unexpectedly long survival made it possible for us to observe the progressively evolving, complex and varied symptomatology due to the preferential involvement of different thalamic nuclei in our patient. The headache was due to raised intracranial pressure as evident by papilloedema and tonsillar herniation. The mass effect of the large thalamic tumours; the intermittent cerebrospinal fluid pathway blockage, during the initial course of the illness and its subsequent complete obstruction at the level of foramen of Monro, or the anterior third ventricular region leading the hydrocephalus were probable contributing factors. The placement of a biventricular shunt brought about a temporary relief in headache in our patient.

The significant memory loss was perhaps due to the infiltration of the anterior thalamic nuclei and the disruption of the mamillothalamic tract that form an integral part of the Papez circuit of memory. The bilateral tumours in our case did not extend to the other structures that are also crucial for memory namely, the medial temporal-amygdalohippocampal region or the prefrontal cortex. The tremor in our patient was perhaps due to the extension of the tumour into the ventroanterior and ventrolateral thalamic nuclei and the dentato-rubro-thalamic tract infiltration. The apathy and lack of initiation in the final stages seemed to be due to the involvement of midline and interlaminar nuclei and their connections with the prefrontal cortex that form an integral part of the reticular activating ascending tracts of the cerebral cortex.<sup>7,10,12,13,17,25</sup> Uncompensated raised intracranial pressure could have also contributed to the clinical presentation in the terminal stages. Our patient did not have sensory symptoms corresponding to the sensory component of ventral nuclei, or visual deficits corresponding to the lateral geniculate body or the pulvinar.<sup>12</sup>

Thalamic astrocytomas initially remain confined to two different areas of the thalamus.<sup>6,21,23</sup> The group I tumours start in the peduncular and capsulothalamic areas and invade the lateral and ventrolateral thalamus and, group II tumours originate in the subependymal glia of the third ventricle and expand laterally from the medial nuclei. In the case reported by Kouyialis, et al the tumour could be shown to be clearly belonging to group II based on the evolving neurobehavioural syndrome.<sup>12</sup> In our patient, however, a clear distinction between the two groups was not possible since the patient progressively developed manifestations attributable to the anterior nuclei and mamillothalamic tract, the motor component of the ventral nuclei and subsequently the dorsomedial and interlaminar nuclei. Raised intracranial pressure as well as bilaterality of the tumours resulting in the involvement of different sets of nuclei in the two thalami could have contributed to the

nebulousness in the progression of clinical manifestations in our patient. Thus, we were unable to classify him in one of the predefined groups based on the progression of neuro-behavioural manifestations.

### Radiology

The T1-weighted MRI showed bilateral thalamic tumours without contrast enhancement. The T2-weighted images showed the almost homogenous hyperintense lesions that respected the grey-white matter boundaries and essentially remained confined to the thalamus (except the spread to the head of the right caudate nucleus, as has also been observed by Esteve, et al).<sup>6</sup> In our patient, the tumours had a large volume with no significant mass effect and the margins were indistinct from the surrounding brain pointing to the infiltrative nature of the tumour.<sup>4</sup> Serial imaging in these tumours often does not show any difference in size or progression to other areas even when rapid clinical deterioration is evident, as was also observed in the present case.<sup>5,9</sup> The MRS of the tumours supported the observation made by Esteve, et al in their 2 patients with bithalamic gliomas.<sup>6</sup> In our patient, there was the presence of the distinct creatinine and choline peaks with the creatinine peak being slightly higher than the choline peak. This was in contrast to the increased MR choline peak (in comparison to the creatinine peak) usually observed in low grade astrocytomas. The MR N-acetyl aspartate signal was decreased and there was a small lactate signal, as has also been observed in low grade gliomas. In addition, Esteve, et al had also shown an increase in the phosphocreatinine peak in a patient with bilateral thalamic tumours who had undergone P-MRS.<sup>31</sup> This had led them to conclude that these tumours have a unique metabolism even when their histology is indistinguishable from that of the low grade astrocytomas.<sup>6</sup> The creatinine peak in the MRS findings of the patient reported by Hirano, et al was not higher than that of choline; nevertheless, the creatinine and choline peaks were distinctly high and there was decrease in N-acetyl aspartate.<sup>11</sup>

### Outcome

Reardon, et al treated 36 consecutive paediatric patients with thalamic astrocytomas and found that bithalamic involvement exerted an independent and significant negative impact on progression free and overall survival of patients with low grade tumours; however, the bithalamic involvement was not associated with outcome among patients with high grade tumours.<sup>18</sup> The review of literature has also revealed a progressive and relentless course with a dismal survival beyond 2 - 3 years for the majority of patients with bilateral thalamic astrocytomas, even when most of these patients have a WHO astrocytoma grade I, II or III.<sup>5,18</sup> In contrast to this, a subpopulation of unilateral low grade astrocytomas behaves in a very benign manner

and remains dormant for several years.<sup>25</sup>

Thus, an obvious contradiction is apparent while dealing with bilateral thalamic astrocytomas. Despite the usually benign histological grade of the tumours and the fact that they do not seem to be enlarging or infiltrating into surrounding areas on serial imaging, they are still associated with a dismal prognosis. This may be explained on the basis of two postulations, both of which may be working in tandem. One of these relates to their "unique metabolism" and the other to their "crucial bilateral compensatory interconnectivity".

The proton MRS choline signal correlates with cell membrane biosynthesis and with choline turnover, especially in proliferating tissues. The creatinine signal is caused by metabolites involved in cellular energetics. The N-acetyl aspartate signal is a marker of neuronal tissue.<sup>6,11,23</sup> In the healthy brain tissue and the usual low grade gliomas, the choline and creatinine peaks have a much lower signal than has been observed in the cases of bilateral thalamic astrocytomas.<sup>6,11</sup> The higher creatinine peak in bilateral thalamic astrocytomas, when compared to their unilateral counterparts, also points towards a higher cellular energetics in the former condition. The decrease in N-acetyl aspartate was nearly the same in bilateral thalamic gliomas as seen in the usual low grade gliomas. Thus, the cellular metabolism as well as proliferative potential in bilateral thalamic gliomas is much higher than seen in the usual low grade gliomas while the neuronal tissue loss is nearly the same. The poorer prognosis in the former condition may be due to this local aggressiveness and altered metabolism of the tumour cells that causes rapid and progressive functional loss of vital thalamic interconnections even while the blood brain barrier (evidenced by lack of enhancement) and a benign histology is maintained. Therefore, bilateral thalamic gliomas need to be graded into a higher grade of aggressiveness based on their MRS findings than their histology indicates.

The thalamus has several nuclei and serves as a strategic connecting portal between the cerebral cortex, the corpus striatum, the cerebellum, brain stem and the spinal cord.<sup>12</sup> It is possible that the two thalami have a coordinated action so that when there is loss of function on one side due to an infiltrating astrocytoma, the contralateral thalamus compensates by serving as a back-up facility. However, their bilateral involvement causes an early decompensation of vital neuronal interconnectivity in the brain leading to early neurological deterioration. This functional impairment may explain the progressive neurobehavioural deterioration of patients with bilateral thalamic gliomas, even when the tumours appear confined to the thalami on imaging.

### Surgical management

Unilateral thalamic lesions may be radically treated with a

variety of approaches including a transcortical transventricular, an interhemispheric transcallosal and occasionally, an infratentorial supracerebellar or transsylvian transinsular approach.<sup>1,16,22</sup> Neuronavigational volumetric resection is a useful adjunct for the resection of these deep seated tumours.<sup>5,14</sup> In the cases of bilateral thalamic astrocytomas, however, almost all the studies have utilized stereotactic biopsy and radiotherapy.<sup>5,11-13,17,23</sup> This conservative approach has been adopted because the lesions are bilateral, non-enhancing and lack a clearly distinguishable tumour-brain interface.<sup>14</sup> Messing-Junger, et al have performed image-guided stereotactic biopsy from bilateral thalamic astrocytomas by using MRS and positron emission tomography using O-(2-<sup>18</sup>fluoroethyl)-L-tyrosine and found diagnostic "hot spots" after metabolic imaging that enhanced the diagnostic yield of the biopsy.<sup>14</sup> We preferred an endoscopic biopsy and a simultaneous cerebrospinal fluid diversion under endoscopic guidance for our patient.<sup>2,8,20</sup> While using the endoscope, the orientation of the lateral ventricle was determined by tracing the choroid plexus and its relationship to the medial ventricular wall and the foramen of Monro. A direct visualization of the tumour bulging into the lateral ventricle was possible; the biopsy could be monitored and obtained from the most representative area of ependymal breach, haemostasis could be achieved under visual control and precise guidance could be obtained for shunt placement. Conformational and stereotactic techniques in radiotherapy and combination chemotherapy may contribute to improving the prognosis.<sup>5</sup> The outcome of thalamic gliomas is poor with only 28% of paediatric patients surviving progression free for more than 4 years after the diagnosis.<sup>18</sup> Bilateral thalamic tumours contribute to further worsening of prognosis of these patients.

### Conclusion

To conclude, a unique case of bilateral thalamic astrocytoma has been reported and literature reviewed. Despite being histologically benign, these lesions represent a rapidly progressive and downhill course for the patient. This may be explained by their unique metabolism evident on MRS analysis. A neuroendoscope was a useful adjunct in obtaining biopsy to decide the further course of action and in guiding successful ventriculoperitoneal shunt placement to temporarily ameliorate the symptoms of raised intracranial pressure.

### References

1. Albright AL: Feasibility and advisability of resections of thalamic tumours in pediatric patients. *J Neurosurg* 2004, 100 (5 Suppl Pediatrics): 468-72
2. Auer LM, Holzer P, Ascher PW, Heppner F: Endoscopic neurosurgery. *Acta Neurochir (Wien)* 1988, 90: 1-14
3. Burger PC, Cohen KJ, Rosenblum MK, Tihan T: Pathology of diencephalic astrocytomas. *Pediatr Neurosurg* 2000, 32: 214-9

4. Colosimo C, di Lella GM, Tartaglione T, Riccardi R: Neuroimaging of thalamic tumors in children. *Childs Nerv Syst* 2002; 18(8): 426-39
5. Di Rocco C, Iannelli A: Bilateral thalamic tumors in children. *Childs Nerv Syst* 2002, 18(8): 440-4
6. Esteve F, Grand S, Rubin C, Hoffmann D, Pasquier B, Graveron-Demilly D, Mahdjoub R, Le Bas JF: MR spectroscopy of bilateral thalamic gliomas. *AJNR* 1999, 20(5): 876-81
7. Fernandez C, Maues de Paula A, Colin C, Quilichini B, Bouvier-Labit C, Girard N, Scavarda D, Lena G, Figarella-Branger D: Thalamic gliomas in children: an extensive clinical, neuroradiological and pathological study of 14 cases. *Childs Nerv Syst* 2006, 22(12): 1603-10; [Epub 2006, Sep 2]
8. Gaab MR, Schroeder HWS: Neuroendoscopic approach to intraventricular lesions. *J Neurosurg* 1998, 88: 496-505
9. Gudowius S, Engelbrecht V, Messing-Junger M, Reifenberger G, Gartner J: Diagnostic difficulties in childhood bilateral thalamic astrocytomas. *Neuropediatrics* 2002, 33(6): 331-5
10. Gutmann DH, Grossman RI, Mollman JE: Personality changes associated with thalamic infiltration. *J Neurooncol* 1990, 8(3): 263-7
11. Hirano H, Yokoyama S, Nakayama M, Nagata S, Kuratsu J: Bilateral thalamic glioma: case report. *Neuroradiol* 2000, 42(10): 732-4
12. Kouyialis AT, Boviatis EJ, Prezerakos GK, Korfiatis S, Sakas DE: Complex neurobehavioural syndrome due to bilateral thalamic glioma. *Br J Neurosurg* 2004, 18: 534-7
13. Lagares A, de Toledo M, Gonzalez-Leon P, Rivas JJ, Lobato RD, Ramos A, Cabello A: Bilateral thalamic gliomas: report of a case with cognitive impairment. *Rev Neurol* 2004, 38(3): 244-6
14. Messing-Junger AM, Floeth FW, Pauleit D, Reifenberger G, Willing R, Gartner J, Coenen HH, Langen KJ: Multimodal target point assessment for stereotactic biopsy in children with diffuse bithalamic astrocytomas. *Childs Nerv Syst* 2002, 18(8): 445-9
15. Nagaratnam N, Ting A, Jolley D: Thalamic tumour presenting as frontal lobe dysfunction. *Int J Clin Pract* 2001, 55(7): 492-3
16. Ozek MM, Ture U: Surgical approach to thalamic tumors. *Childs Nerv Syst* 2002, 18(8): 450-6
17. Partlow GD, del Carpio-O'Donovan R, Melanson D, Peters TM: Bilateral thalamic glioma: review of eight cases with personality change and mental deterioration. *AJNR* 1992, 13(4): 1225-30
18. Reardon DA, Gajjar A, Sanford RA, Heideman RL, Walter AW, Thompson SJ, Merchant TE, Hao LI, Jenkins JJ, Langston J, Boyett JM, Kun LE: Bithalamic involvement predicts poor outcome among children with thalamic glial tumors. *Pediatr Neurosurg* 1998, 29(1): 29-35
19. Ruel JH, Broussolle E, Gonnaud PM, Jouveta A, Rousselle C, Chazot G: Bilateral thalamic glioma. A clinicopathological study of 2 cases. *Rev Neurol (Paris)* 1992, 148(12): 742-5
20. Selvapandian S: Endoscopic management of thalamic gliomas. *Minim Invasive Neurosurg* 2006, 49(4): 194-6
21. Smyth GE, Stern K: Tumors of the thalamus, a clinicopathological study. *Brain* 1938, 61: 339-74
22. Steiger HJ, Gotz C, Schmid-Elsaesser R, Stummer W: Thalamic astrocytomas: Surgical anatomy and results of a pilot series using maximum microsurgical removal. *Acta Neurochir (Wien)* 2000, 142(12): 1327-37
23. Uchino M, Kitajima S, Miyazaki C, Shibata I, Miura M: Bilateral thalamic glioma-case report. *Neurol Med Chir (Tokyo)* 2002, 42(10): 443-6
24. Yoshida M, Fushiki S, Takeuchi Y, Takanashi M, Imamura T, Shikata T, Morimoto A, Konishi K, Miyazaki A, Sawada T: Diffuse bilateral thalamic astrocytomas as examined serially by MRI. *Childs Nerv Syst* 1998, 14(8): 384-8
25. Ziegler DK, Kaufman A, Marshall HE: Abrupt memory loss associated with thalamic tumor. *Arch Neurol* 1977, 34(9): 545-8

### Comments:

In this well written report, the authors present a relatively rare case of a patient with bilateral thalamic astrocytoma. I agree with the overall management as presented by the authors. The aim ought to be to obtain diagnosis (either a stereotactic biopsy or transventricular endoscopic biopsy as performed by the authors), CSF diversion in the presence of hydrocephalus, followed by adjunctive treatments, depending on the specific grade of the tumour and available institutional protocols (chemotherapy and/or radiation). I do not recommend surgical resection of thalamic gliomas, as the risk of neurological deficits outweighs the theoretical benefit of decompressing a thalamic astrocytoma.

Saleem I Abdulrauf  
 Associate Professor, Neurological Surgery  
 Secretary General, World Federation of Skull Base Societies  
 Director, Cerebrovascular and Skull Base Surgery Program  
 Saint Louis University School of Medicine  
 St. Louis, Missouri  
 USA  
 Tel: (1 314) 268 7888  
 Fax: (1 314) 268 5061  
 Email: abdulrsi@slu.edu