

Case Report

Successful Resection of Hypothalamic Hamartoma with Intractable Gelastic Seizures - by Transcallosal Subchoroidal Approach

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A 19-year old female with intractable gelastic seizures was found to have 0.7 x 1.8 x 1.8 cm elliptical mass on the floor of the third ventricle. The signal intensity on the Magnetic Resonance Imaging (MRI) was consistent with the Hypothalamic Hamartoma (HH). Ictal EEG demonstrated rhythmic 7 Hz waves over Fp2, F4, and C4 with spreading to the right temporal region and then bilaterally. Ictal Single Photon Emission Computerized Tomography (SPECT) showed hyperperfusion at hypothalamic and medial frontopolar regions. The patient underwent surgical resection using Trans Callosal Subchoroidal Approach (TCSA) to the third ventricle. Pathological finding confirmed the diagnosis of hypothalamic hamartoma. Following the operation, she has been seizure free up to 12 months. Thereafter, provoked seizures seldom occurred and there has been improvement in her memory, emotional control and independence. This appears to be the first report of this surgical approach for HH, which is less likely to disturb memory function compared to previously described interfoniceal approach.

Keywords: Hypothalamic hamartoma, Gelastic seizure, Transcallosal, Subchoroidal, SPECT, Epilepsy

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Although Hypothalamic Hamartoma (HH) was first reported in 1934 by Le Marquand and Russel⁽¹⁾ and successful surgical removal of HH causing "pubertas praecox" was first published by Northfield and Russell⁽²⁾ in 1967, the epileptogenic potential of HH has not been widely appreciated until recently^(1,3-9). The majority of seizures caused by HH eventually become medically intractable^(5-7,9,10). Seizure free is an exception⁽⁵⁾. Gonadotropin Releasing Hormone (GnRH) analogue administration and Gamma knife have been

reported to provide seizure control in a limited number of patients^(3,6,7). Vagal nerve stimulation has little effect on seizure control in patients with HH⁽¹¹⁾. Surgical removal of HH, therefore, plays a significant role in treating patients with this disorder at present^(1,5,6,12-16). The authors herein present a case of HH where the epileptogenicity revealed during presurgical ictal SPECT and successful surgical removal achieved by Trans Callosal Subchoroidal Approach (TCSA) with review of the related literature.

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Case Report

A 19 year-old Thai female was first noted to have 1-minute duration laughing at the age of 2 months. Since then, she has had several spontaneous laughing spells on a daily basis. At the age of 4 years, she started

having episodes of unmotivated laughter followed by Generalized Tonic Clonic (GTC) seizures lasting 3-5 minutes. She had 2-3 attacks per year. At 9 years of age, the laughing episodes were occasionally followed by unresponsiveness, blank staring. She had daily hand automatism lasting 1- 2 minutes. Her birth history, growth, and development were unremarkable. She had normal intellectual function and behavior. Physical examination showed no abnormality. The patient underwent comprehensive presurgical evaluation. High resolution MRI of the brain revealed a midline elliptical lesion, 7 mm. in width, 1.8 cm. in height and A-P dimension, occupying the inferior part of the third ventricle. The mass was slightly hypersignal on T2WI

and FLAIR (Fig. 1a-d). Electro EncephaloGram (EEG) showed rare bilateral T3T4 interictal discharges. Ictal EEG showed rhythmic 7 Hz waves over Fp2, F4, and C4; discharges then spread to the right temporal region; and then bilaterally. Early ictal SPECT using Tc-99m Ethyl Cysteinate Dimer (ECD) injected 24 seconds after the clinical onset of a gelastic seizure showed hyperperfusion of hypothalamus and medial frontopolar regions. Interictal SPECT demonstrated hypoperfusion of the hypothalamic area (Fig. 2). She has been on several antiepileptic drugs without satisfactory improvement. Prior to operation, she experienced more than 10 laughing spells per month followed by Complex Partial Seizure (CPS) or GTC.

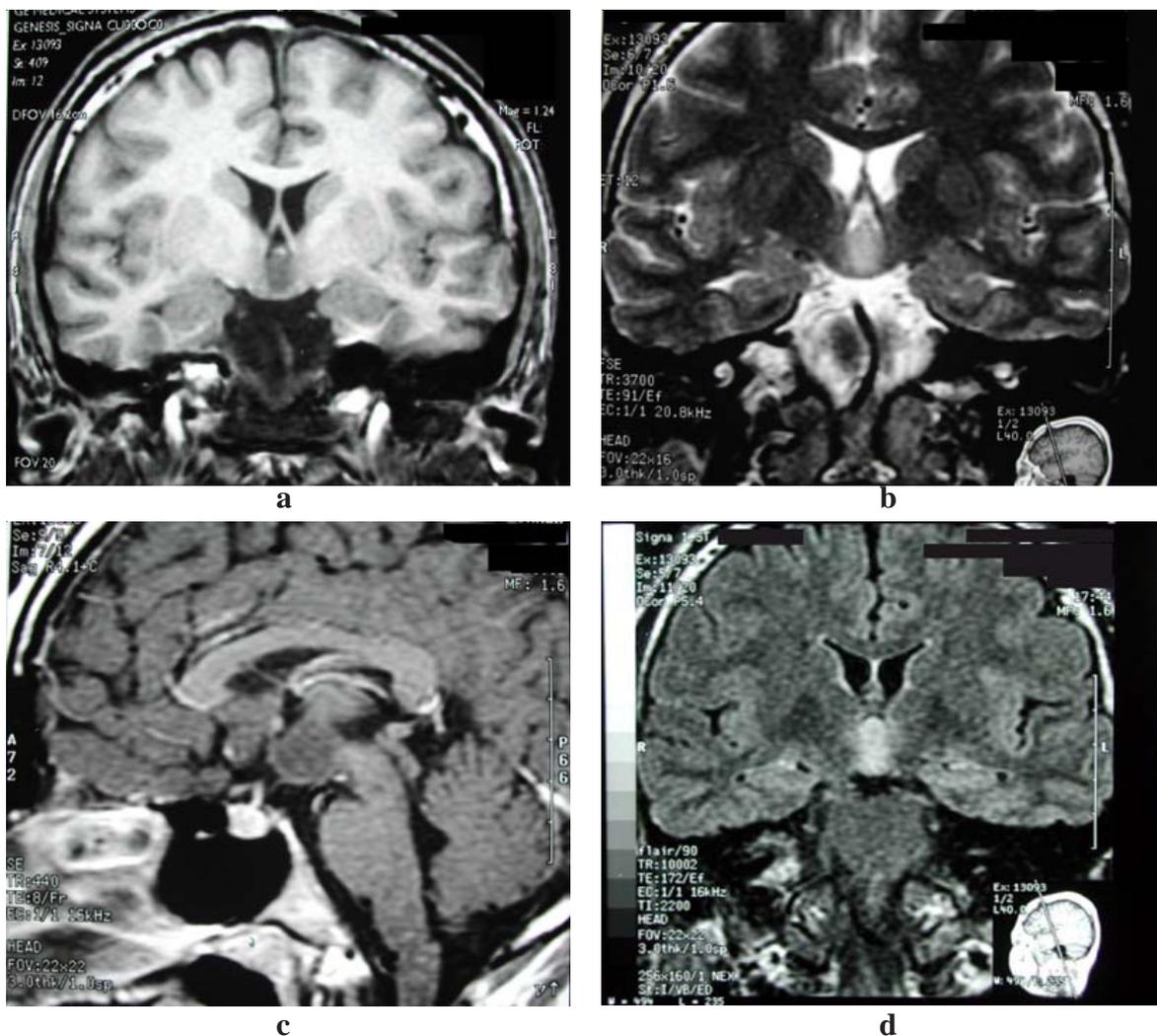


Fig. 1a-d Coronal and sagittal MRI reveal hypothalamic hamartoma in the third ventricle with broad attachment to tuber cinereum

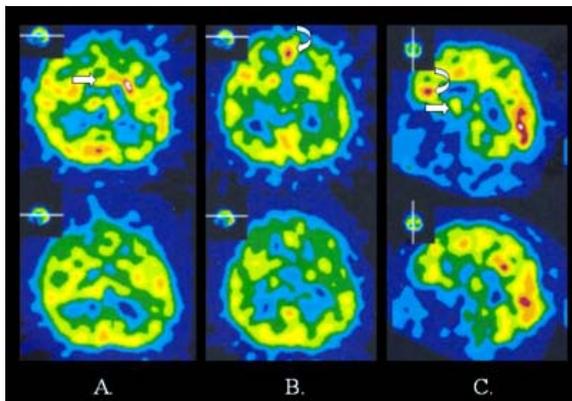


Fig. 2 Ictal (upper row) and interictal (lower row) SPECT images: The midline hypothalamic region showed hyperperfusion on the ictal study compared to hypoperfusion on the interictal study (straight arrow). The medial frontal area near the frontal pole showed hyperperfusion on the ictal and normoperfusion on the interictal study (curved arrow)

At operation, the patient was placed in the supine position with head in rigid fixation in zero degree rotation and her neck was slightly flexed. Right frontal craniotomy was made with bone flap extending across the midline; posterior border extended just posterior to the coronal suture. Dura was opened based on the midline. Under microscope magnification, interhemispheric fissure was gradually split; the dissection was deepened until the corpus callosum was reached. Entry

to the frontal horn of the right lateral ventricle was made through a 2.5 centimeter opening on the anterior corpus callosum. Once the right frontal horn was entered, the foramen of Monroe was identified. The size of the foramen was normal and did not allow adequate visualization of the third ventricle (Fig. 3a). Therefore, choroid plexus emerging from the foramen of Monroe was gently elevated and retracted toward the midline. The tethered thalamostriate vein at the posterior limit of the foramen of Monroe and the velum interpositum were divided. With further retraction of choroids plexus toward the midline, third ventricle and its contents were clearly visualized (Fig. 3b). There was an oval shiny white mass attached to the floor of the third ventricle, and to a lesser extent, to walls of the ventricle. The mass was approximately 2 cm in anterior-posterior dimension and 1 cm in transverse dimension. The massa intermedia was identified a few millimeters posterior to the mass. The mass was removed flush with the inner walls of the third ventricle.

Results

Postoperative period was uneventful. There was no neurological deficit. Pathological findings confirmed the hypothalamic hamartoma. The patient had no seizures during the 12 months of post operative period until rare provoked gelastic seizures occurred (Engel Class IIa)⁽¹⁷⁾. Post operatively; there have been improvements in memory, emotional control, independence and her quality of life.

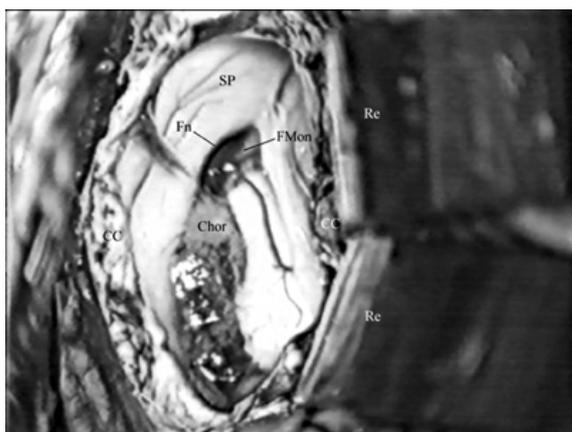


Fig. 3a Entering right lateral ventricle, foramen of Monroe comes into view. *CC.* corpus callosum; *Chor.* choroid plexus; *Fmon.* foramen of Monroe; *Fn.* fornix; *Re.* retractor; *SP.* septum pellucidum

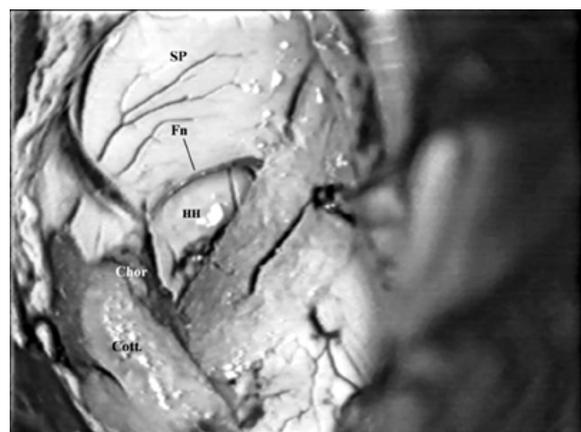


Fig. 3b After dividing the thalamostriate vein and retracting the choroid plexus, hypothalamic hamartoma comes in to view. *Chor.* choroid plexus; *Cott.* cottonoid; *Fn.* fornix; *HH.* hypothalamic hamartoma; *SP.* Septum pellucidum

Discussion

HH may present with Gelastic Seizure (GS), Precocious Puberty (PP), or in combination^(9,12). GS usually start in the early years of life (< 1yr) and may be followed by drop attacks, complex partial seizures, and generalized motor seizures^(3,5,6,18). Other features include mental retardation or behavioral abnormality. Palmieri, et al reported all 13 patients in their series also had delayed developmental milestones either from birth or after the onset of seizures⁽¹⁸⁾. The present case had epilepsy at a young age of onset, but without any other associated abnormality or intellectual impairment, which is interestingly uncommon.

There has been some evidence supporting the epileptogenicity of the HH^(13,19). The early injected Tc-99m ECD ictal SPECT in the present study in one GS without CPS or GTC demonstrated hyperperfusion at the midline hypothalamic region compared to the interictal injection, suggesting the HH itself as being the ictal onset zone for GS. Hyperperfusion within the hamartoma and hypothalamic regions has also been shown by others using ictal SPECT⁽¹⁹⁻²¹⁾. Moreover, stereotactic depth EEG recordings have consistently demonstrated that the ictal discharges of GS were originated and confined within the hamartoma, and stimulation of the HH could produce gelastic episodes^(19,22,23). These findings suggest that GS associated with HH is generated in the hypothalamus. The other types of seizures such as CPS, atonic or GTC, were found to be associated with cortical involvement, either frontal or temporal lobes⁽²³⁻²⁵⁾.

The frontomesial area is involved in triggering motor component of laughter in many studies. Restricted pericingulate premotor cortex has been found to underlie ictal laughing in patients without HH⁽²⁶⁻²⁸⁾. Ictal SPECT in the presented patient also showed hyperperfusion at the frontopolar region, which is likely to represent the symptomatogenic zone for ictal laughter. The increased perfusion to the occipital lobe is considered physiologic uptake usually found in Tc-99m ECD studies as described elsewhere^(29,30).

Since Northfield and Russel reported the first successful surgical treatment of HH in 1967⁽²⁾, surgical series have been published with varying outcomes^(1,5,13-16,18,20,31-33). The surgical approaches for GS vary among neurosurgeons. These approaches can be divided into suprahypothalamic and infrahypothalamic. Suprahypothalamic techniques include transcallosal resection⁽¹³⁾ and endoscopic disconnection^(16,20,31). Infrahypothalamic techniques include frontotemporal^(14,18,32,33), subtemporal⁽¹⁾, subfrontal^(1,2) and translaminar terminalis⁽⁵⁾ approaches.

Early surgical procedures were limited to infrahypothalamic routes^(1,5,14,32,33). With the advent of MRI, the configuration of HH and the relation of HH to the hypothalamus can be visualized with unprecedented details⁽⁴⁾. This has led to suprahypothalamic approaches by some authors^(13,16,31).

For the HH with most of the lesion located in the third ventricle, corresponding to Delalande type II, the suprahypothalamic approach appears to be most suitable^(13,16). Rosenfeld elegantly pioneered transcallosal interfoniceal approach for intraventricular HH with good outcomes⁽¹³⁾. However, transient memory loss due to bilateral foniceal injury is a potential complication with this approach varying from 20-57% depending on the nature of the lesions⁽³⁴⁻³⁶⁾. Harvey et al reported short-term memory impairment in 14 out of 29 patients following transcallosal interfoniceal approach, with four being persistent⁽³⁷⁾.

Delalande, et al reported on 17 HH patients. Nine patients underwent endoscopic disconnection and two had an intraventricular lesion (Delalande type II). Seizure outcomes according to Engel classification⁽¹⁷⁾ were I and III respectively. For the patients with combined intraventricular and intrahypothalamic HH (Delalande type III), Engel class I were obtained in four out of nine cases using combined pterional and endoscopic approaches. This might be attributable to the extent of resection versus the boundary of epileptogenic zone beyond the HH.

In the authors' opinion, the TCSA appeared to be feasible and effective for strictly intraventricular HH (Delalande type II HH). The authors decided to use this technique for resection of the lesion in the presented patient as it provided superior visualization of the intraventricular HH compared to other infrahypothalamic approaches. It also required much less manipulation of fornices compared to Transcallosal Inter Forniceal Approach (TIFA), thus minimizing the possibility of postoperative memory deficit. Although, TCSA enters the third ventricle slightly more lateral compared to TIFA (Fig. 4a), with the brain relaxation from the cerebrospinal fluid drainage and changing the angle of the microscope, the HH, third ventricular wall, and HH interface can be visualized in small and medium size HHs'. However, in the large HH, the HH interface on the opposite site could be obscured by the column of the fornix (Fig. 4b), which makes TIFA more suitable (Fig. 4c).

Although resection of the thalamostriate veins has been a concern by many authors⁽³⁸⁻⁴⁰⁾, unilateral resection is well tolerated in most cases^(34,41,42).

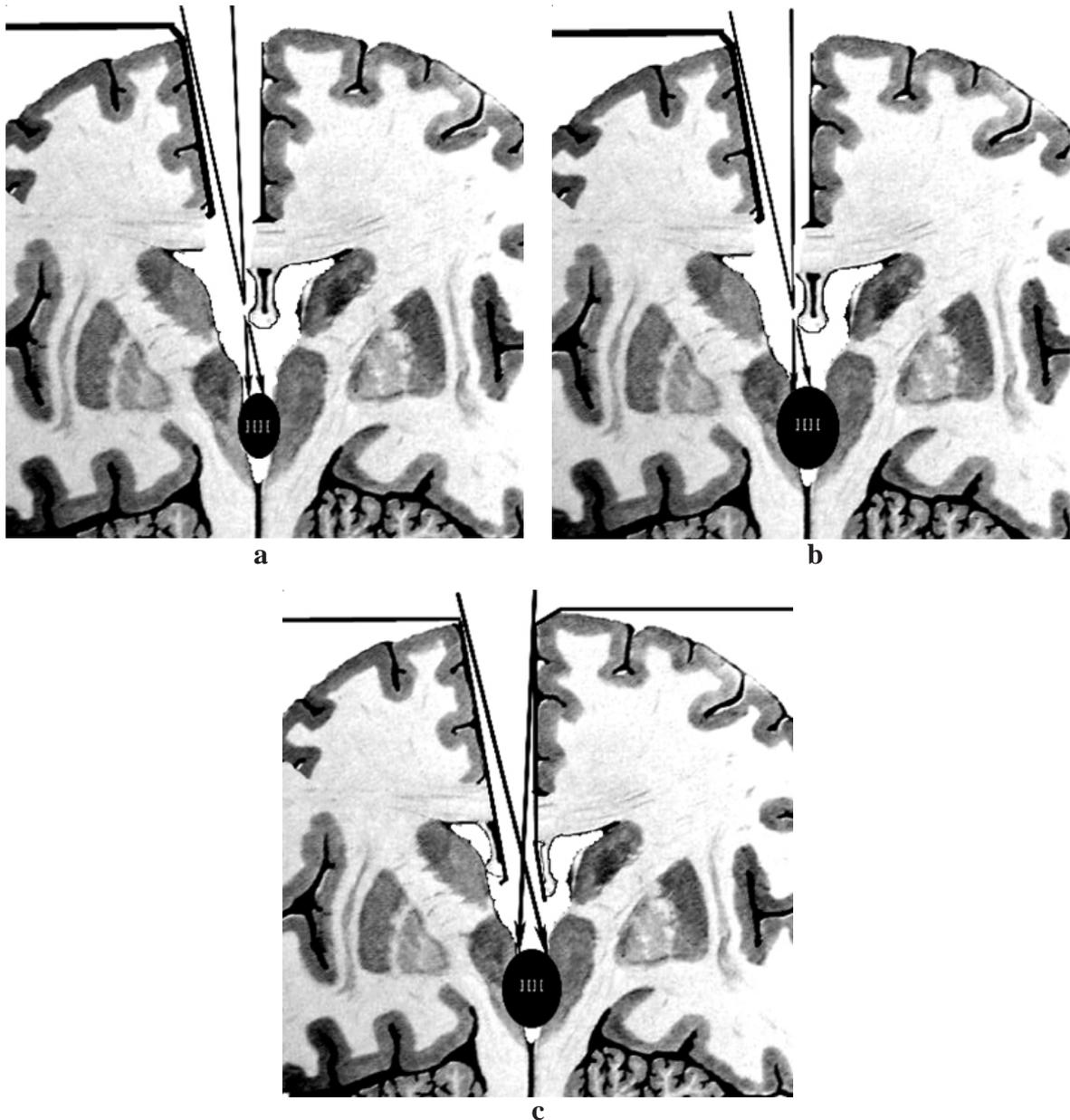


Fig. 4 Comparing transcallosal subchoroidal approach in medium (a) and large (b) HH to transcallosal interfonticeal approach in large HH (c). *HH*. hypothalamic hamartoma

Successful surgery without complication is demonstrated in the presented case.

Surgical outcome of the patient with HH associated with GS varies among series^(1,2,5,13-16,18,31-33). Potential complications of resection of HH include thalamic infarct, third nerve palsy, hyperphagia, hypopituitarism, diabetic incipidus and hemiparesis^(6,18,31). The extent of resection correlates directly to the seizure control. Most of the patients who have become

seizure free have had total or near total resection of the HH^(13,14,16,18,32). The patients who remain seizing after resection are probably due to incomplete removal of the epilepto-genic zone. Alternative hypotheses are secondary epileptogenesis or more diffuse brain lesion indicated by the existence of HH⁽⁶⁾.

In the presented patient, good seizure control can be attributable to several factors. Firstly, the pre-surgical evaluations, including EEG and ictal SPECT,

indicated the HH to be the ictal onset zone. Secondly, the circumscribed configuration, the midline symmetrical, and the almost separable intraventricular location of the HH were amenable to complete resection. Thirdly, the convergence of the GS and the SPECT finding help in confirming the role of HH in ictal genesis. Lastly, the absence of associated abnormality and normal intellectual function in the presented patient may also be responsible for the good surgical outcome.

Conclusion

The ictal SPECT findings in the presented patient confirmed the likeliness of HH as the ictal origin and the mesial frontopolar region as the symptomatogenic zone for GS. Although, the complications associated with surgical resection of hypothalamic hamartoma may be disabling^(6,18,31), surgical treatment still plays an important role in HH associated with medically intractable seizures. The surgical option seems justified in the presented patient who had no other associated abnormality and solely, refractory epilepsy impaired her quality of life. The transcallosal, subchoroidal approach has been demonstrated to be an effective surgical technique to the HH located in most of in the third ventricle. The authors' key to successful resection is the profile of the patient and the surgical strategy chosen⁽¹⁸⁾.

References

1. Sato M, Ushio Y, Arita N, Mogami H. Hypothalamic hamartoma: report of two cases. *Neurosurgery* 1985; 16: 198-206.
2. Northfield DW, Russell DS. Pubertas praecox due to hypothalamic hamartoma: report of two cases surviving surgical removal of the tumour. *J Neurol Neurosurg Psychiatry* 1967; 30: 166-73.
3. Unger F, Schrottner O, Haselsberger K, Korner E, Ploier R, Pendl G. Gamma knife radiosurgery for hypothalamic hamartomas in patients with medically intractable epilepsy and precocious puberty. Report of two cases. *J Neurosurg* 2000; 92: 726-31.
4. Hahn FJ, Leibrock LG, Huseman CA, Makos MM. The MR appearance of hypothalamic hamartoma. *Neuroradiology* 1988; 30: 65-8.
5. Kramer U, Spector S, Nasser W, Siomin V, Fried I, Constantini S. Surgical treatment of hypothalamic hamartoma and refractory seizures: a case report and review of the literature. *Pediatr Neurosurg* 2001; 34: 40-2.
6. Regis J, Bartolomei F, de Toffol B, Genton P, Kobayashi T, Mori Y, et al. Gamma knife surgery for epilepsy related to hypothalamic hamartomas. *Neurosurgery* 2000; 47: 1343-51.
7. Zaatreh M, Tennison M, Greenwood RS. Successful treatment of hypothalamic seizures and precocious puberty with GnRH analogue. *Neurology* 2000; 55: 1908-10.
8. Berkovic SF, Kuzniecky RI, Andermann F. Human epileptogenesis and hypothalamic hamartomas: new lessons from an experiment of nature. *Epilepsia* 1997; 38: 1-3.
9. Arita K, Ikawa F, Kurisu K, Sumida M, Harada K, Uozumi T, et al. The relationship between magnetic resonance imaging findings and clinical manifestations of hypothalamic hamartoma. *J Neurosurg* 1999; 91: 212-20.
10. Nguyen D, Singh S, Zaatreh M, Novotny E, Levy S, Testa F, et al. Hypothalamic hamartomas: seven cases and review of the literature. *Epilepsy Behav* 2003; 4: 246-58.
11. Brandberg G, Raininko R, Eeg-Olofsson O. Hypothalamic hamartoma with gelastic seizures in Swedish children and adolescents. *Eur J Paediatr Neurol* 2004; 8: 35-44.
12. Romner B, Trumpy JH, Marhaug G, Isaksson HJ, Anke IM. Hypothalamic hamartoma causing precocious puberty treated by surgery: case report. *Surg Neurol* 1994; 41: 306-9.
13. Rosenfeld JV, Harvey AS, Wrennall J, Zacharin M, Berkovic SF. Transcallosal resection of hypothalamic hamartomas, with control of seizures, in children with gelastic epilepsy. *Neurosurgery* 2001; 48: 108-18.
14. Valdueza JM, Cristante L, Dammann O, Bentele K, Vortmeyer A, Saeger W, et al. Hypothalamic hamartomas: with special reference to gelastic epilepsy and surgery. *Neurosurgery* 1994; 34: 949-58.
15. Berkovic SF, Andermann F, Melanson D, Ethier RE, Feindel W, Gloor P. Hypothalamic hamartomas and ictal laughter: evolution of a characteristic epileptic syndrome and diagnostic value of magnetic resonance imaging. *Ann Neurol* 1988; 23: 429-39.
16. Delalande O, Fohlen M. Disconnecting surgical treatment of hypothalamic hamartoma in children and adults with refractory epilepsy and proposal of a new classification. *Neurol Med Chir (Tokyo)* 2003; 43: 61-8.
17. Engel J Jr, Van Ness PC, Rasmussen TB, Ojemann LM. Outcome with respect to epileptic seizures. In: Engel J Jr, editor. *Surgical treatment of the epilepsies*. 2nd ed. New York: Raven Press; 1993:

- 609-21.
18. Palmi A, Chandler C, Andermann F, Costa DC, Paglioli-Neto E, Polkey C, et al. Resection of the lesion in patients with hypothalamic hamartomas and catastrophic epilepsy. *Neurology* 2002; 58: 1338-47.
 19. Kuzniecky R, Guthrie B, Mountz J, Bebin M, Faught E, Gilliam F, et al. Intrinsic epileptogenesis of hypothalamic hamartomas in gelastic epilepsy. *Ann Neurol* 1997; 42: 60-7.
 20. Fohlen M, Lellouch A, Delalande O. Hypothalamic hamartoma with refractory epilepsy: surgical procedures and results in 18 patients. *Epileptic Disord* 2003; 5: 267-73.
 21. DiFazio MP, Davis RG. Utility of early single photon emission computed tomography (SPECT) in neonatal gelastic epilepsy associated with hypothalamic hamartoma. *J Child Neurol* 2000; 15: 414-7.
 22. Arroyo S, Lesser RP, Gordon B, Uematsu S, Hart J, Schwerdt P, et al. Mirth, laughter and gelastic seizures. *Brain* 1993; 116(Pt 4): 757-80.
 23. Kahane P, Ryvlin P, Hoffmann D, Minotti L, Benabid AL. From hypothalamic hamartoma to cortex: what can be learnt from depth recordings and stimulation? *Epileptic Disord* 2003; 5: 205-17.
 24. Leal AJ, Moreira A, Robalo C, Ribeiro C. Different electroclinical manifestations of the epilepsy associated with hamartomas connecting to the middle or posterior hypothalamus. *Epilepsia* 2003; 44: 1191-5.
 25. Munari C, Kahane P, Francione S, Hoffmann D, Tassi L, Cusmai R, et al. Role of the hypothalamic hamartoma in the genesis of gelastic fits (a videostereo-EEG study). *Electroencephalogr Clin Neurophysiol* 1995; 95: 154-60.
 26. Chassagnon S, Minotti L, Kremer S, Verceuil L, Hoffmann D, Benabid AL, et al. Restricted frontomesial epileptogenic focus generating dyskinetic behavior and laughter. *Epilepsia* 2003; 44: 859-63.
 27. Garcia A, Gutierrez MA, Barrasa J, Herranz JL. Cryptogenic gelastic epilepsy of frontal lobe origin: a paediatric case report. *Seizure* 2000; 9: 297-300.
 28. Striano S, Meo R, Bilò L, Cirillo S, Nocerino C, Ruosi P, et al. Gelastic epilepsy: symptomatic and cryptogenic cases. *Epilepsia* 1999; 40: 294-302.
 29. Leveille J, Demonceau G, Walovitch RC. Intra-subject comparison between technetium-99m-ECD and technetium-99m-HMPAO in healthy human subjects. *J Nucl Med* 1992; 33: 480-4.
 30. Inoue K, Nakagawa M, Goto R, Kinomura S, Sato T, Sato K, et al. Regional differences between 99mTc-ECD and 99mTc-HMPAO SPET in perfusion changes with age and gender in healthy adults. *Eur J Nucl Med Mol Imaging* 2003; 30: 1489-97.
 31. Breningstall GN. Gelastic seizures, precocious puberty, and hypothalamic hamartoma. *Neurology* 1985; 35: 1180-3.
 32. Nishio S, Morioka T, Fukui M, Goto Y. Surgical treatment of intractable seizures due to hypothalamic hamartoma. *Epilepsia* 1994; 35: 514-9.
 33. Takeuchi J, Handa H, Miki Y, Munemitsu H, Aso T. Precocious puberty due to a hypothalamic hamartoma. *Surg Neurol* 1979; 11: 456-60.
 34. Apuzzo ML, Litofsky NS. Surgery in and around the anterior third ventricle. In: Apuzzo ML, editor. *Brain surgery*. Vol. 1. New York: Churchill Livingstone; 1993: 541-79.
 35. Berkovic SF, Arzimanoglou A, Kuzniecky R, Harvey AS, Palmi A, Andermann F. Hypothalamic hamartoma and seizures: a treatable epileptic encephalopathy. *Epilepsia* 2003; 44: 969-73.
 36. Polkey CE. Resective surgery for hypothalamic hamartoma. *Epileptic Disord* 2003; 5: 281-6.
 37. Harvey AS, Freeman JL, Berkovic SF, Rosenfeld JV. Transcallosal resection of hypothalamic hamartomas in patients with intractable epilepsy. *Epileptic Disord* 2003; 5: 257-65.
 38. McKissock W. The surgical treatment of colloid cyst of the third ventricle; a report based upon twenty-one personal cases. *Brain* 1951; 74: 1-9.
 39. Ono M, Rhoton AL, Jr., Peace D, Rodriguez RJ. Microsurgical anatomy of the deep venous system of the brain. *Neurosurgery* 1984; 15: 621-57.
 40. Viale GL, Turtas S. The subchoroid approach to the third ventricle. *Surg Neurol* 1980; 14: 71-4.
 41. Lavyne MH, Patterson RH. Subchoroidal transvelum inerspositum approach. In: Apuzzo ML, editor. *Surgery of the third ventricle*. 2nd ed. Vol. 1. Baltimore: Lippincott William & Wilkins; 1998: 453-69.
 42. Hirsch JF, Zouaoui A, Renier D, Pierre-Kahn A. A new surgical approach to the third ventricle with interruption of the striothalamic vein. *Acta Neurochir (Wien)* 1979; 47: 135-47.

รายงานการผ่าตัดก้อนไฮโปธาลามิคฮามาโตมาในผู้ป่วยโรคลมชักแบบเจลาสติกโดยการผ่านคอร์ปัสแคโลสัสน์ และลอดใต้คอร์รอยด์

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ผู้ป่วยหญิง อายุ 19 ปี ป่วยเป็นโรคลมชักแบบเจลาสติกซึ่งต้องการรักษาทางยา จากการตรวจสมองด้วยเครื่องตรวจสนามแม่เหล็กพบมีก้อนขนาด 0.7 x 1.8 x 1.8 เซนติเมตรที่บริเวณด้านล่างของช่องโพรงน้ำสมองที่ 3 โดยลักษณะเข้าได้กับก้อนไฮโปธาลามิคฮามาโตมา จากการตรวจคลื่นสมองขณะชักพบว่ามีคลื่นความถี่ 7 เฮิรท์ ที่บริเวณ Fp2, F4 และ C4 กระจายไปยังสมองกลีบขมับข้างขวาและกระจายต่อไปทั้งสองข้าง จากการตรวจด้วยสเปกสแกนพบการเพิ่มการเรืองสารที่บริเวณไฮโปธาลามิคและบริเวณกลีบสมองส่วนหน้า ผู้ป่วยได้ เข้ารับการผ่าตัดฮามาโตมาโดยผ่านคอร์ปัสแคโลสัสน์และลอดใต้คอร์รอยด์ หลังผ่าตัดผู้ป่วยไม่มีอาการชักเลยเป็นเวลา 12 เดือน หลังจากนั้นมีการชักบ้างแต่ไม่บ่อย หลังการผ่าตัดผู้ป่วยมีความจำ การควบคุมอารมณ์และการช่วยเหลือตัวเองดีขึ้น รายงานนี้เป็นรายงานแรกที่ใช้การผ่าตัดวิธีนี้เพื่อรักษาไฮโปธาลามิคฮามาโตมาเพื่อลดผลกระทบต่อความจำจากการผ่าตัดเข้าระหว่างโพนิกส์ซึ่งมีการรายงานมาก่อนหน้านี้
