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Surgical Treatment of Lesions Involving the Supplementary Motor Area: Clinical Results of 12 Patients

Suplementer Motor Alan Lezyonlarının Cerrahi Tedavisi: 12 Hastanın Klinik Değerlendirmesi

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ABSTRACT

AIM: Surgical resection of lesions involving the dominant supplementary motor area (SMA) may result in immediate postoperative motor and speech deficits which in most cases are reversible.We report 12 patients with frontal lesions involving the SMA and aim to analyse the clinical data and the correlation of neurological deficit with the extent of SMA resection.

MATERIAL and METHODS: 12 patients (5M/7F, mean age 30.5) harbouring lesions involving the SMA who underwent surgery between 2002-2007 were evaluated retrospectively. Eloquent cortical areas were determined by functional MRI studies and/or invasive monitoring with subdural grids and depth electrodes and/or awake craniotomy with continuous intraoperative monitoring.

RESULTS: The evaluation of postoperative MRI's revealed total and subtotal resection of the lesion in 9(75%) and 3(25%) patients respectively. The extent of SMA resection was complete in 5 patients (41,6%) and incomplete in 7 patients (58,3%). Immediately postoperatively, all patients where the resection of SMA was complete displayed the typical characteristics of SMA syndrome. The degree of deficits was consistent with the extent of the SMA resection in all patients.

CONCLUSION: Proper diagnostic methods aiming to localise the sensorimotor area can minimize the risk of deficit in the surgical treatment of SMA lesions. The results suggest a relationship between the incidence of SMA syndrome and the extent of SMA resection.

KEY WORDS: Supplementary motor area, Surgery, Functional MRI, Invasive monitoring, Aphasia, Hemiparesia

ÖZ

AMAÇ: Dominant suplementer motor alanı (SMA) içeren lezyonların cerrahi tedavisinde postoperatif erken dönemde görülen ve çoğunlukla geri dönen motor ve konuşma işlev kayıpları olabilir.Bu çalışmada, SMA'nı içeren frontal lezyon nedeniyle opere edilen 12 olguluk seri sunulmaktadır ve klinik verilerin analizi ve postoperatif nörolojik kayıp ile SMA rezeksiyon düzeyi ilişkisinin araştırılması amaçlanmıştır.

GEREÇ ve YÖNTEM: 2002-2007 yılları arasında SMA ilişkili frontal lezyon nedeniyle opere edilen 12 olgunun (5 E/7K,ortalama yaş 30,5) verileri retrospektif olarak incelendi.Bütün hastalara preoperatif ve postoperatif kontrastlı-kontrastsız MR incelemesi uygulandı.Bütün hastalarda rezeksiyon öncesi hassas kortikal alanlarlar fonksyonel MR ve/veya subdural grid ve derinlik elektrodu ile invazif monitorizasyon ve/veya lokal anestezi altında uyanık kraniotomi ile intraoperatif monitorizasyon kullanılarak belirlendi.

BULGULAR: Postoperatif MR incelemeleri değerlendirildiğinde lezyonların 9 hastada total (75 %), 3 hastada ise (25 %) subtotal olarak çıkartıldığı saptandı. SMA rezeksiyon düzeyi 5 hastada tam (41,6 %), 7 hastada ise kısmendi (58,3 %). SMA rezeksyonu tam olan bütün hastalarda postoperatif erken dönemde SMA sendromunun tipik bulguları görüldü. Bütün hastalarda nörolojik hasar görülme derecesi SMA rezeksyonu derecesi ile ilişkili bulundu.

SONUÇ: SMA'yı içeren lezyonları cerrahi tedavisinde cerrahi öncesi sensorimotor alanın tespiti amacıyla uygun yöntemlerin kullanılması gelişebilecek komplikasyonları en aza indirgeyecektir. Sonuçlar, SMA sendromunu insidansı ile SMA rezeksiyon düzeyi arasında bir ilişkinin bulunduğunu düşündürmektedir.

ANAHTAR SÖZCÜKLER: Supplementer motor alan, Cerrahi, Fonksyonel MRI, İnvasif monitorizasyon, Afazi, Hemiparezi

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INTRODUCTION

The supplementary motor area (SMA) occupies the medial portion of Brodmann cortical area 6 and is located in the superior frontal gyrus (14,28,37). The anatomical limits of the SMA are the primary motor cortex posteriorly, the cingulate sulcus and genu of corpus callosum inferiorly and the edge of the medial cortex laterally (30,32,36). It has been shown to be involved in several aspects of motor control, including movement selection, preparation, initiation, execution, and feedback-monitoring of a motor program as well as in motor learning and in planning of complex sequences of movement (6,15, 16,18,27,38). Moreover, clinical and electrophysiological studies in patients also support a role of the SMA in speech (8,12). Electrical stimulation performed rostral to the supplementary motor representation of the face resulted in vocalization and speech arrest or slowing of speech (8,12).

Surgical resections of tumors of the medial frontal lobe may result in immediate postoperative motor and speech deficits which in most cases are reversible (4,11,29,33,34,39). However postoperative motor deficit is usually unpredictable, while it is more common when the resection limit extends in caudal parts of SMA (20,39). Thus, it is of utmost importance for the neurosurgeon to determine the anatomical and functional limits of the surgical resection and the characteristics and the cause of the deficit. This is also important in the preoperative period for informing the patient about the risk of its occurrence and its typical course of recovery.

We report a series of 12 patients who experienced various degrees of neurological deficits related to partial or complete SMA syndrome after partial or total resection of SMA had been performed to remove a frontal lesion. We aimed to analyse the clinical data with respect to diagnosis, surgical technique and prognosis. Our subsequent intend was to evaluate the correlation between the pattern of immediate postoperative clinical symptoms and the extent of SMA resection.

MATERIAL and METHODS

This clinical study includes 12 patients harbouring lesions involving SMA who were operated between 2002 and 2007 by the senior author (MU). There were 5 male and 7 female patients with ages ranging from 10 to 60 years (mean age 30,5 years). All of the patients, but one were right handed. The primary presenting symptoms were seizure in 10 patients and hemiparesia in 2 patients. Of the 10 patients presenting with seizures, 4 had medically intractable epilepsy. These patients were evaluated preoperatively according to our epilepsy surgery team's preoperative protocol. All patients underwent preoperative cranial MR imaging with and without gadolinium.

The determination of the central and precentral gyrus on MRI was accomplished by examining the typical anatomical landmarks as described before (2). For further demonstrating the eloquent cortical areas, 9 patients underwent preoperative functional MRI (fMRI) imaging while each performed language and motor tasks. In 4 patients who were operated for medically intractable epilepsy, subdural grids and/or depth electrodes were used for the same purpose. Two of these 4 patients also underwent fMRI imaging. In the remaining 2 patients, awake craniotomy was done where fMRI was done in one of them.

Lesion resection was performed along the visible lesion borders with careful dissection under the microscope to avoid injuring eloquent cortical areas. In 4 patients in whom subdural grids and/or depth electrodes were placed, lesionectomy was performed in the second operation after brain mapping with cortical stimulation. In two patients the surgery was performed awake and under local anesthesia as the posterior lesion border reached or exceeded the precentral sulcus.

The neurological status of each patient was assessed preoperatively as well as postoperatively at 1-day intervals until the patient was discharged. The postoperative motor and language deficits were reviewed and analysed from the patient records. Motor deficits were rated using a standard grading scale: 5/5, normal power; 4/5, active movement against gravity and resistance; 3/5, active movement against gravity; 2/5, active movement only with gravity eliminated; 1/5, trace contraction; or 0/5, no contraction. Hemiplegia or impaired movement contralateral to the side of lesion which improved rapidly was classified as an SMA injury. Immediate speech deficits, including mutism, reduced spontaneous speech, and anomia, that recovered during the postoperative early follow-up period were also considered to be due to SMA injury.

In all patients, cranial MRI with and without gadolonium was performed in the postoperative period. In 6 patients postoperative MRI's were acquired within 24 hours. The extent of the resection of the lesion and the SMA was evaluated on postoperative MR images and classified as total or subtotal and complete or incomplete, respectively.

RESULTS

The histopathological evaluation of the specimens disclosed tumoral lesions in 9 patients (75%) and nonneoplastic focal lesions in three patients (25%). The distribution of the tumoral lesions were oligodendroglioma in 4 patients (3 WHO grade II, 1 WHO grade III), oligoastrocytoma in 1 patient (WHO grade II), astrocytoma in 1 patient (WHO grade II), anaplastic astrocytoma in 1 patient, glioblastoma multiforme in 1 patient and dysembryoplastic neuroepithelial tumor in 1 patient. Among the 3 nontumorous focal lesions, 1 was focal cortical dysplasia, 1 was cortical dysgenesis with multilobular polymicrogyrus and the remaining lesion was a hamartoma.

The evaluation of the postoperative MRI's revealed total resection of the lesion in 9 patients (75%) and subtotal resection in 3 patients (25%). The extent of SMA resection was complete in 5 patients (41.6%) and incomplete in 7 patients (58.3%). Immediately postoperatively, 5 patients with a dominant left SMA, displayed severe hemiparesis, neglect of the contralateral extremities, and mutism. Mutism without hemiparesia was observed in a patient with a dominant right SMA.

In the series presented, a typical SMA syndrome was encountered in 5 patients (Cases # 3,5,6,11,12). In all these cases, various degrees of hemiparesia were observed in the early postoperative period immediately after surgery. Whenever motor deficits were present, the upper and lower extremities were similarly affected and motor function was later regained in both extremities simultaneously. Moreover, the muscle tones were typically preserved in the affected extremities of SMAS cases. In 3 patients (Case #3,5,11) the motor deficit were hemiplegia, while in the remaining two (case #6 and #12) the motor deficit grade were 2/5 and 3/5 respectively. In case #3, the hemiplegia rapidly recovered to 4/5 motor power in 3 days on both right upper and lower extremities. He was back to full recovery at postoperative 1 month follow-up. In Case #5, the motor power of the right lower extremity recovered to 4/5 in the postoperative 3rd week, while the right upper extremity was 3/5. At her last follow up at postoperative 1 year, she had recovered completely except for a mild deficit in the right hand grasping function. Case #11 also had a fast recovery period from hemiplegia to 4/5 motor power in both upper and lower extremities in postoperative 10th day. However the tumor recurred and he presented with hemiplegia 4 months after the first operation. He was lost 1 month later. In cases #6 and #12 the hemiparesia recovered in 1 month while there were mild deficits in the finger movements in both patients at their 1 year control. When the extent of the resection of the SMA was evaluated, the degree of postoperative motor deficit was in concordant with the resection of SMA. In cases where the SMA syndrome was observed the SMA was totally removed either because the lesion was purely localised to that area (Case #5 and #12) or the lesion was extending towards it. In contrast, motor deficits were not present at all in cases which the SMA had been incompletely removed. As for the 7 patients where the SMA resection was incomplete, only one patient experienced postoperative neurological deficit as mutism without hemiparesia. The clinical data of the patients are summarized in (Table I). Some of the cases in the present series are illustrated in Figures 1-6.

DISCUSSION

A characteristic syndrome of immediate postoperative contralateral motor and speech deficits emerges following complete or incomplete resection of the SMA (33,34,39). One of the main characteristics of this syndrome, namely the SMA syndrome, is a complete or almost complete recovery within several weeks or months (20,33). The specific evolution of this syndrome has been reported to occur in three stages (22): 1) immediately after surgery there is a global akinesia, which is more prominent contralaterally with an arrest of speech; 2) sudden recovery a few days later, with persistent reduction in contralateral motor activity, emotional facial palsy, and reduction in spontaneous speech; and; 3) within weeks to months after operation, the only sequel is disturbance of the alternating movements of the hands. Typically, the muscle tone of the paralyzed extremities are preserved. These observations have been confirmed by other series of surgical resection of the SMA (4,11,29,33,34,39).

Table I. Clinical and radiographic data of 12 patients who underwent resection of SMA lesions

Case #	Age/ Sex	Pres. Sx	MRI fx - Adjacent to	Excision Lesion/SMA	Pathology	Preop NE	Postop NE
1	33 / M	Seizure	LPG	ST / I	OD grade II	Deficit (-)	Deficit (-)
2	31 / M	Seizure	LPG 1cm ant.	T / I	OD grade II	Deficit (-)	Deficit (-)
3	60 / M	Seizure	LPG 1cm ant.	T / C	OD grade III	Tremor, N	SMAS
4	10 / F	Seizure	RF	ST / I	MLP	Deficit (-)	Deficit (-)
5	23 / F	Seizure	LPG	T / C	FCD	Deficit (-)	SMAS
6	30 / M	HP	LPG	T / C	OD grade II	R HP	SMAS
7	11 / F	Seizure	RPG	T / I	DNET	Deficit (-)	Deficit (-)
8	14 / F	Seizure	LPG 1cm ant.	T / I	Hamartoma	Deficit (-)	Deficit (-)
9	24 / F	Seizure	RFCG	T / I	OA grade II	Deficit (-)	Mutism
10	35 / F	HP	RPG	ST / I	A grade III	Deficit (-)	Deficit (-)
11	55 / M	Seizure	LF	T / C	GBM	Deficit (-)	SMAS
12	41 / F	Seizure	LPG	T / C	A grade II	Deficit (-)	SMAS

Abbreviations: R:Right; L:Left; Pres.Sx:Presenting symptom; HP:Hemiparesia; LPG:Left precentral gyrus; RPG: Right precentral gyrus; RF: Right frontal; RFCG:Right frontal cingulate gyrus; MRI fx: MRI findings; T: Total; ST:Subtotal; C:Complete; I: Incomplete; OD: Oligodendroglioma; FCD: Focal cortical dysplasia; MLP: Multilobular polymicrogyrus; DNET: Dysembryoplastic neuroepithelial tumor; OA: Oligoastrocytoma; A:Astrocytoma; GBM: Glioblastoma Multiforme; NE: Neurological examination; N: Nystagmus; SMA: Supplementary motor area; SMAS: Supplementary motor area syndrome

Typical SMA syndrome have been encountered in 5 of the patients in our series with various degrees of hemiparesia occurring in the early postoperative period immediately after surgery. The degree of postoperative motor deficit was concordant with the resection of SMA as SMA syndrome was observed in all cases where the SMA was totally removed and it was not encountered in cases which the SMA had been incompletely removed. Thus, the location and severity of SMAS were proportional to the anteroposterior extent of the resection and thus to the amount of SMA preserved. Following surgery, the motor deficit remains unpredictable, although it is more frequent when the resection extends in caudal parts of the SMA (20,39).

Postoperative speech disorders as the component of SMAS in this series were observed as a transient aphasia followed by a stage of constant improvement in speech fluency. This finding is similar to the previous reports (1,17,19,13,22,23,33, 35,39). It has been suggested that only the SMA in the dominant hemisphere is involved in language function, thus aphasia occurs as a result of the resection of dominant SMA (7,23,26,39). However, there are reports where the resection of SMA in the nondominant hemisphere resulted in speech dysfunction (22,33). In our series, postoperative aphasia was only observed as a component of SMAS except for one patient (Case # 9). Thus there was no postoperative speech disorder in 6 patients. As all SMAS cases in this series had lesions in the dominant hemisphere, one cannot argue that only the resection of the dominant SMA leads to postoperative aphasia. Moreover, we note that postoperative mutism was also observed in case #9, although the lesion was in the nondominant hemisphere. Thus we agree with the previous reports which find the role of SMA dominancy in speech production controversial (19,22,33).

Identification of eloquent areas of the brain to avoid resection-induced damage is of utmost importance for minimizing the neurological deficit and postoperative quality of life. Thus in patients harbouring lesions associated with SMA, the anatomical and functional association must be defined and shown preoperatively. Invasive and non-invasive methods are used for this purpose. Awake craniotomy with stimulation and continuous monitoring of neurological function and brain mapping with the use of subdural grids and depth electrodes are two reliable invasive methods (3, 10,

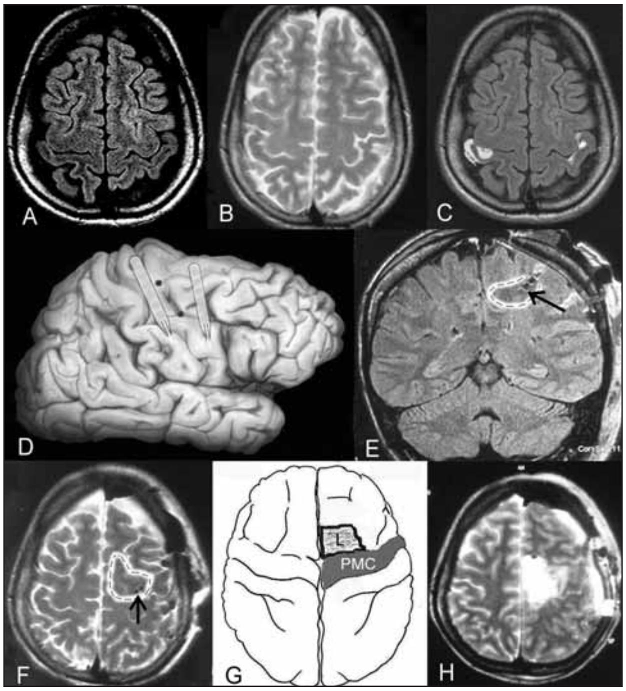

Figure 1: *(Case # 5): Preoperative Flair (**A**) and T-2 weighted (**B**) cranial MRI sections reveales no evidence of pathology. The left and right precentral gyrus is demonstrated on fMRI (**C**). Two subdural grids with 6 contacts and two depth electrodes (black dots) were placed as shown in the diagram (**D**). After stimulation and brain mapping the suspected epileptogenic area is identified in dotted lines as shown in coronal T1 (**E**) and axial T2 (**F**) weighted MRI sections. The black arrows show the tip of the depth electrode. (**G**) The schematic drawing shows that the lesion (**L**) is confined to the borders of the SMA (Black lines) in front of the primary motor cortex (PMC). (**H**) Postoperative T2 weighted cranial MRI demonstrates total excision of the lesion.*

21). However, awake craniotomy necessitates the performance of surgery in the awake patient, which can be a great strain for the patient and the surgeons in a procedure that takes several hours, and even carries the added risk of producing an epileptic seizure. Brain mapping with electrodes and grids requires a two staged procedure where the resection is done in the second operation. As a non-invasive method, fMRI has been used as an alternative procedure in estimating the location of the SMA (5,9, 24,25,31,35). The value of fMR imaging in preoperative mapping of the SMA in patients with tumors in the medial frontal lobe brain has been reported previously (20,25). Krainik et al. retrospectively evaluated motor deficits in 23 patients who had undergone resection of tumors involving the medial frontal lobe and concluded that

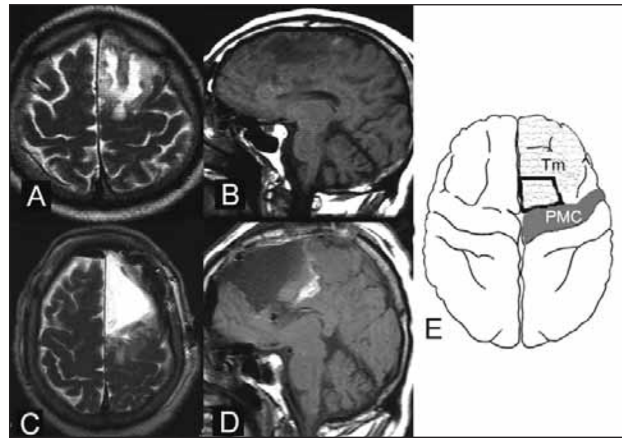

Figure 2: *(Case # 3): The preoperative cranial MRI sections (**A-B**) demonstrate a left frontal lesion extending posteriorly to the precentral gyrus. Postoperative axial (**C**) and sagittal (**D**) cranial MRI sections reveal total excision of the lesion. (**E**) The diagram shows that the tumor (Tm) involves all the SMA area (black lines) and extends posteriorly towards the primary motor cortex (PMC).*

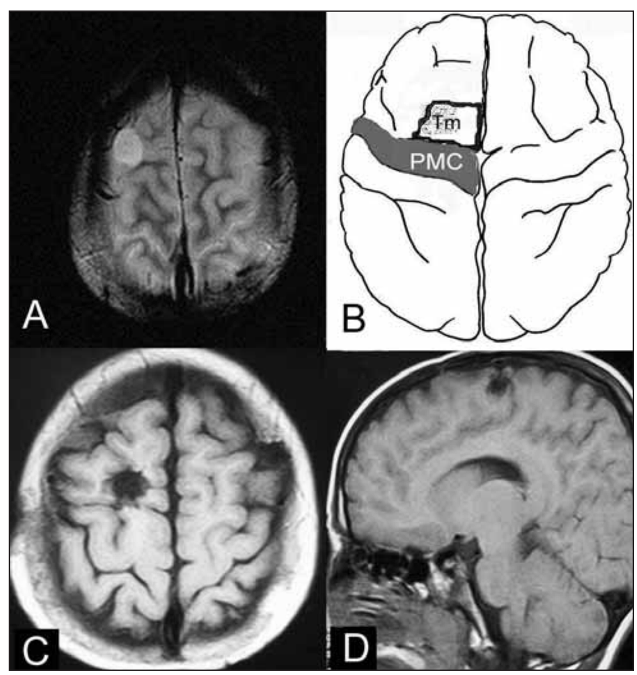

Figure 3: *(Case # 7): (**A**) The preoperative axial flair MRI section shows a demarcated lesion located on the right superior frontal gyrus. (**B**) Although not occupying all the SMA, the tumoral lesion (Tm) was confined to SMA borders (black lines) anterior to the primary motor cortex (PMC), as shown in this diagram. Postoperative axial (**C**) and sagittal (**D**) MRI sections demonstrate total exsicion of the tumor.*

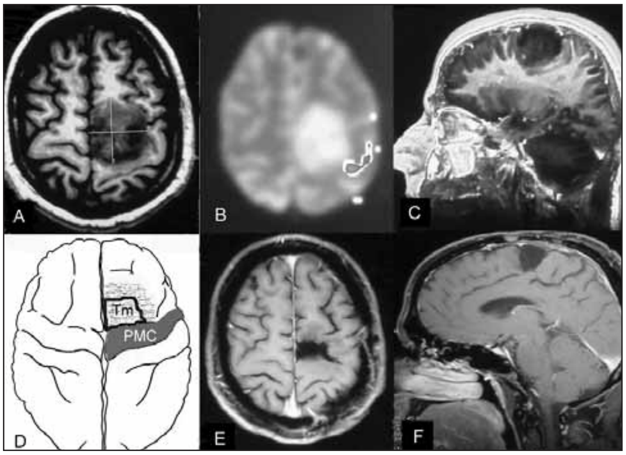

Figure 4: *(Case # 1): The preoperative axial (**A**) and sagittal (**C**) cranial MRI sections reveal a well demarcated left frontal lesion located in close proximity to the left primary motor strip as also shown in fMRI section (**B**). (**D**) The diagram shows that the tumor (Tm) is located in the SMA (black lines), in front of the primary motor cortex (PMC). The patient underwent awake craniotomy and the tumor was subtotally excised as shown in postoperative axial (E) and sagittal (F) contrasted cranial MRI sections.*

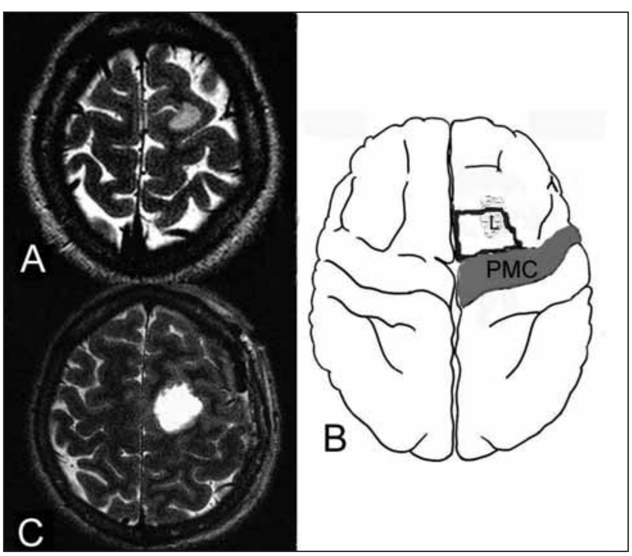

Figure 5: *(Case #8): (**A**) Preoperative axial T-2 weighted cranial MRI section shows a lesion located in the left superior frontal gyrus. (**B**) Part of the lesion (L) was located in the boundaries of the SMA (black lines) in front of the primary motor cortex (PMC) as shown in the diagram. (**C**) The postoperative axial T2-weighted cranial MRI section shows total removal of the lesion.*

the risk of developing postoperative weakness increased when surgical resection included the SMA as demonstrated on preoperative fMR image mapping (20). Similar findings has been reported by Nelson et al., who measured the distance between

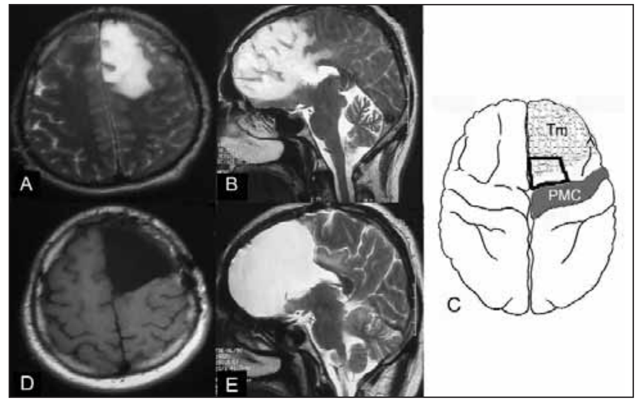

Figure 6: *(Case # 2): The preoperative axial (**A**) and sagittal (**B**) T-2 weighted cranial MRI sections reveal a left frontal tumoral lesion. The tumor (Tm) was extending towards the boundaries of the SMA (black lines) and involved part of it as shown in the diagram (PMC: Primary motor cortex). The postoperative axial (**D**) and sagittal (**E**) cranial MRI sections show total removal of the tumor.*

the edge of the tumor and the center of SMA activation in fMRI of 12 patients preoperatively and correlated these results with the risk of postoperative neurological deficit (25). We have used invasive methods in 6 patients where 4 of them were operated for medically intractable epilepsy. In these 4 patients subdural grids and depth electrodes were placed for the purpose of localising the origin and extension of the epileptogenic focus. In 6 patients only fMRI was done preoperatively and the results were useful in the preoperative mapping of the eloquent areas prior to resection of lesions. We believe that fMRI should be performed in every patient having suspected lesions involving the eloquent areas. In cases where the preoperative epilepsy surgery work up necessitates invasive monitoring, fMRI can be used as an additional procedure for further estimating the motor strip and eloquent areas.

In conclusion, presurgical planning in patients having lesions involving SMA requires the proper identification of eloquent areas that are very important for minimizing the postoperative neurological deficit. The occurrence and severity of SMAS is associated with the extent of SMA resection and patients should be informed preoperatively about the risks and natural course of this syndrome. Whenever possible fMRI should be performed preoperatively in every patient, even in cases where invasive monitoring is required, as it can further aid in mapping cortical function.

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REFERENCES

1. Arseni C, Botez MI: Speech disturbances caused by tumours of the supplementary motor area. Acta Psychiatr Neurol Scand 36: 279–299, 1961
2. Berger MS, Cohen WA, Ojemann GA: Correlation of motor cortex brain mapping data with magnetic resonance imaging. J Neurosurg 72: 383–387, 1990
3. Berger MS, Kincaid J, Ojemann GA, Lettich E: Brain mapping techniques to maximize resection, safety, and seizure control in children with brain tumors. Neurosurgery 25: 786–792, 1989
4. Bleasel A, Comair Y, Lüders HO: Surgical ablation of the mesial frontal lobe in humans. Adv Neurol 70: 217–235, 1996
5. Braun V, Dempf S, Tomczak R, Wunderlich A, Weller R, Richter HP: Functional cranial neuronavigation. Direct integration of fMRI and PET data. J Neuroradiol 27: 157–163, 2000
6. Cadoret G, Smith AM: Comparison of the neuronal activity in the SMA and the ventral cingulate cortex during prehension in the monkey. J Neurophysiol 77: 153–166, 1997
7. Chauvel P, Bancaud J, Buser P: Participation of the supplementary motor area in speech. Exp Brain Res 58: (abstr).A14, 1985.
8. Chauvel PY, Rey M, Buser P, Bancaud J: What stimulation of the supplementary motor area in humans tells about its functional organization. Adv Neurol 70: 199–209, 1996
9. Chung GH, Han YM, Kim CS: Functional MRI of the supplementary motor area: comparison of motor and sensory tasks. J Comput Assist Tomogr 24: 521–525, 2000
10. Ebeling U, Schmid UD, Ying H, Reulen HJ: Safe surgery of lesions near themotor cortex using intra-operative mapping techniques: A report on 50 patients. Acta Neurochir (Wien) 119: 23–28, 1992
11. Fontaine D, Capelle L, Duffau H: Somatotopy of the supplementary motor area: Evidence from correlation of the extent of surgical resection with the clinical patterns of deficit. Neurosurgery 50: 297–305, 2002
12. Fried I, Katz A, McCarthy G, Sass KJ, Williamson P, Spencer SS, Spencer DD: Functional organization of human supplementary motor cortex studied by electrical stimulation. J Neurosci 11: 3656–3666, 1991
13. Gelmers HJ: Non-paralytic motor disturbances and speech disorders: the role of the supplementary motor area. J Neurol Neurosurg Psychiatry 46: 1052–1054, 1983
14. Goldberg G: Supplementary motor area structure and function: review and hypotheses. Behav Brain Sci 4: 567–615, 1985
15. Hikosaka O, Sakai K, Miyauchi S, Takino R, Sasaki Y, Pütz B: Activation of human presupplementary motor area in learning of sequential procedures: a functional MRI study. J Neurophysiol 76: 617–621, 1996
16. Humberstone M, Sawle GV, Clare S, Hykin J, Coxon R, Bowtell R, Macdonald IA, Morris PG: Functional magnetic resonance imaging of single motor events reveals human presupplementary motor area. Ann Neurol 42: 632–627, 1997
17. Jonas S: The supplementary motor region and speech emission. J Commun Disord 14: 349–373, 1981
18. Kansaku K, Kitazawa S, Kawano K: Sequential hemodynamic activation of motor areas and the draining veins during finger movements revealed by cross-correlation between signals from fMRI. Neuroreport 9: 1969–1974, 1998
19. Krainik A, Lehéricy S, Duffau H, Capelle L, Chainay H, Cornu P, Cohen L, Boch AL, Mangin JF, Le Bihan D, Marsault C: Postoperative speech disorder after medial frontal surgery: role of the supplementary motor area. Neurology 60(4): 587-594, 2003
20. Krainik A, Lehéricy S, Duffau H, Vlaicu M, Poupon F, Capelle L, Cornu P, Clemenceau S, Sahel M, Valery CA, Boch AL, Mangin JF, Bihan DL, Marsault C: Role of the supplementary motor area in motor deficit following medial frontal lobe surgery.Neurology 57(5): 871-878, 2001
21. Kral T, Kurthen M, Schramm J, Urbach H, Meyer B: Stimulation mapping via implanted grid electrodes prior to surgery for gliomas in highly eloquent cortex. Neurosurgery 58(1 Suppl):ONS36-43, 2006
22. Laplane D, Talairach J, Meininger V, Bancaud J, Orgogozo JM: Clinical consequences of corticectomies involving the supplementary motor area in man. J Neurol Sci 34: 301–314, 1977
23. Masdeu JC, Schoene WC, Funkenstein H: Aphasia following infarction of the left supplementary motor area. A clinicopathologic study. Neurology 28: 1220–1223, 1978
24. Mueller WM, Yetkin FZ, Hammeke TA, Morris GL 3rd, Swanson SJ, Reichert K, Cox R, Haughton VM: Functional magneticresonance imaging mapping of the motor cortex in patientswith cerebral tumors. Neurosurgery 39: 515–521, 1996
25. Nelson L, Lapsiwala S, Haughton VM, Noyes J, Sadrzadeh AH, Moritz CH, Meyerand ME, Badie B: Preoperative mapping of the supplementary motor area in patients harboring tumors in the medial frontal lobe. J Neurosurg 97(5): 1108-1114, 2002
26. Pai MC: Supplementary motor area aphasia: A case report. Clin Neurol Neurosurg 101: 29–32, 1999
27. Passingham RE: Functional specialization of the supplementary motor area in monkeys and humans. In: H. O. Luders, ed. Advances in Neurology, Vol. 70: Supplementary Sensorimotor Area. Lippincott-Raven, Philadelphia: 1996: 105–116
28. Penfield W, Welch K: The supplementary motor area of the cerebral cortex: a clinical and experimental study. Arch Neurol Psychiatry 66: 289–317, 1951
29. Peraud A, Meschede M, Eisner W, Ilmberger J, Reulen HJ: Surgical resection of Grade II astrocytomas in the superior frontal gyrus. Neurosurgery 50: 966–977, 2002
30. Picard N, Strick PL: Motor areas of the medial wall: a review of their location and functional activation. Cereb Cortex 6: 342–353, 1996
31. Puce A, Constable RT, Luby ML, McCarthy G, Nobre AC, Spencer DD, GoreJC, Allison T: Functional magnetic resonance imaging of sensory and motorcortex: Comparison with electrophysiological localization. J Neurosurg 83: 262–270, 1995
32. Rizzolatti G, Luppino G, Matelli M: The classic supplementary motor area is formed by two independent areas. In: Lüders H, ed. Supplementary sensorimotor area. Philadelphia: Lippincott-Raven, 1996: 45–56

33. Rostomily RC, Berger MS, Ojemann GA, Lettich E: Postoperative deficits and functional recovery following removal of tumors involving the dominant hemisphere supplementary motor area. J Neurosurg 75: 62–68, 1991
34. Russell SM, Kelly PJ: Incidence and clinical evolution of postoperative deficits after volumetric stereotactic resection of glial neoplasms involving the supplementary motor area. Neurosurgery 52(3): 506-516, 2003
35. Sailor J, Meyerand ME, Moritz CH, Fine J, Nelson L, Badie B, Haughton VM: Supplementary motor area activation in patients with frontal lobe tumors and arteriovenous malformations. AJNR Am J Neuroradiol 24(9): 1837-1842, 2003
36. Talairach J, Bancaud J: The supplementary motor area in man: anatomo-functional findings by stereo-electroencephalography in epilepsy. Int J Neurol 5: 330–347, 1966
37. Tanji J: The supplementary motor area in cerebral cortex. Neurosci Res 19: 251-258, 1994
38. Weilke F, Spiegel S, Boecker H, von Einsiedel HG, Conrad B, Schwaiger M, Erhard P: Time-resolved fMRI of activation patterns in M1 and SMA during complex voluntary movement. J Neurophysiol 85: 1858–1863, 2001
39. Zentner J, Hufnagel A, Pechstein U, Wolf HK, Schramm J: Functional results after resective procedures involving the supplementary motor area. J Neurosurg 85: 542–579, 1996