Supplementary Motor Area Syndrome in Neurosurgical Practice

Mohammad Hamza Bajwa[1], Zunaira Pathan[2], Muhammad Shahzad Shamim[3]

Abstract

Supplementary motor area (SMA) syndrome is a distinct clinical entity observed following surgery of the medial frontal cortex particularly in procedures targetting intra-axial lesions and epileptogenic zones. Characterized by transient, contralateral hemiparesis and, with dominant hemisphere involvement and speech initiation deficits, SMA syndrome presents a significant but reversible postoperative challenge. With the integration of advanced neuroimaging and intraoperative neural tract mapping techniques, our understanding of neuroplasticity in the clinical trajectory and recovery following SMA syndrome has substantially progressed. This review aims to synthesize recent, high-impact studies in the context of brain tumour and epilepsy surgery outlining contemporary insights into SMA function, surgical planning and recovery mechanisms. We contextualize these developments in the light of the foundational work of Laplane et al. (1977), who first characterized the syndrome and explore novel findings on the role of the frontal aslant tract (FAT) in SMA connectivity. The aim is to provide neurosurgeons and neuroscientists with an evidence-based understanding of SMA syndrome and highlight implications for improving functional outcomes in neuro-oncological and epilepsy surgery.

DOI: https://doi.org/10.47391/JPMA.26-39

Introduction

The supplementary motor area (SMA) is located on the medial surface of the superior frontal gyrus anterior to the primary motor cortex and is intricately involved in initiation and planning of voluntary movements with co-ordination of complex motor sequences. It plays a role in speech initiation and inter-limb coordination notably during bi-manual tasks. Historically, SMA lesions were presumed to result in irreversible motor impairments; this view shifted significantly following the landmark 1977 study by Laplane et al. who systematically documented the clinical evolution of deficits following mapped cortical resections involving the SMA.[1] Their work, based on stereotactic resections in epilepsy patients, described a tri-phasic recovery: an initial stage of global akinesia and mutism followed by partial recovery with persistent contralateral hypokinesia and speech reduction, and near-complete recovery with subtle coordination deficits. This study laid the groundwork for conceptualizing SMA syndrome as a reproducible, largely reversible postoperative phenomenon emphasizing the separation between voluntary and spontaneous motor behaviour in terms of cortical networks.

[1,3]Section of Neurosurgery, The Aga Khan University Hospital, Karachi, Pakistan
[2]Dow International Medical College, Karachi, Pakistan
Correspondence: M. Shahzad Shamim. e-mail: shahzad.shamim@aku.edu
ORCID ID: 0000-0001-8305-8854

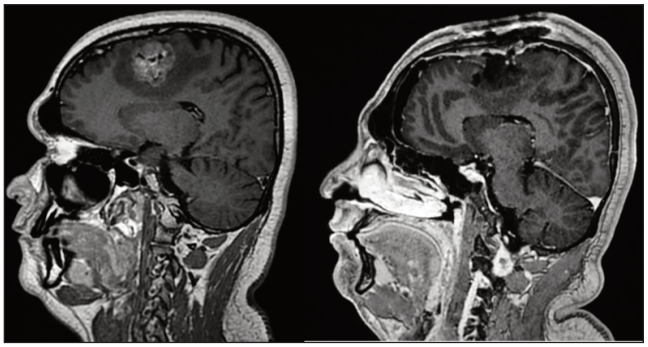

Figure: (Left) MRI brain with contrast (sagittal sequence) showing a contrast-enhancing intra-axial lesion within the pre-motor cortex, (Right) post-operative imaging showing gross-total resection of the lesion.

SMA syndrome is often encountered following resection of low- or high-grade gliomas and drug-resistant epilepsy when epileptogenic foci involve the mesial frontal regions (Figure 1). With evidence supporting supramarginal resection in glioma surgery and expanding indications for resective epilepsy procedures, a nuanced understanding of prognostic implications and recovery mechanisms is critical. This review presents evidence organized around key studies that have contributed to shaping our current paradigm. We also introduce emerging neuro-anatomical insights into white matter connectivity; particularly the involvement of the frontal aslant tract (FAT), as a basis for SMA-associated deficits.

Review of Evidence

Krainik et al. (2001) conducted a prospective functional MRI (fMRI) study on mechanisms of postoperative recovery following SMA resection in patients with gliomas.[2] The study involved 23 patients undergoing medial frontal lobe surgery with serial fMRI used to monitor motor network activation pre- and post-operatively. They found that patients demonstrating postoperative recruitment of the contralateral SMA and lateral premotor cortex had a more rapid and complete recovery of motor function. These findings were among the first to provide neuro-imaging

evidence for interhemispheric compensation as a key mechanism in SMA syndrome recovery. This work remains foundational in establishing contralesional SMA involvement in postoperative recovery pathways. Nakajima et al. (2019) presented a retrospective analysis of 20 patients who underwent glioma resections involving the SMA. This study documented the duration and recovery trajectory of postoperative SMA syndrome.[3] In this cohort, 95% of patients developed some degree of SMA syndrome, predominantly characterized by contralateral akinesia and speech arrest, with 81.8% achieving complete or near-complete functional recovery within five weeks. The authors noted that lesions encroaching upon the cingulate gyrus or involving the pre-SMA were associated with more prolonged deficits, suggesting a topographic vulnerability gradient within the SMA complex. This study provides robust clinical data affirming the transient nature of SMA syndrome – this evidence is crucial in pre-operative patient counselling and planning resections in this region.

Kasabeh et al. (2012) extended the exploration of SMA syndrome into epilepsy surgery context. This retrospective review included 39 paediatrics patients with resistant epilepsy attributed to the mesial frontal lobe who underwent SMA resections. The authors reported that while a significant proportion of patients with postoperative neurological deficits (17 of 23) experienced transient motor and speech deficits consistent with SMA syndrome, 82% of patients with SMA syndrome achieved resolution of symptoms 1 month after surgery.[4] Complete resolution of the syndrome was seen in all patients 6 months after epilepsy surgery. This study is pivotal in demonstrating that resection of epileptogenic SMA cortex can yield favourable seizure outcomes without incurring permanent functional compromise. It also underscores the trade-off between seizure control and transient functional morbidity in such cases.

Functional Subdivisions of the SMA

Hiroshima et al. (2014) created functional maps of the SMA by employing direct cortical and subcortical stimulation during awake craniotomies in six patients. Their findings demonstrated functional segregation within the SMA: the posterior SMA was more directly involved in motor execution and speech initiation, containing the so-called "SMA core". Intraoperative stimulation of these areas reliably reproduced components of SMA syndrome such as speech arrest and contralateral akinesia.[5] These results support the utility of tailored intraoperative mapping in preserving function while maximizing resection. This study provides compelling evidence that SMA syndrome is not simply a consequence of bulk resection, but of targetted disruption of specialized sub-regions. Briggs et al. (2021) employed diffusion tensor imaging (DTI) to investigate the contribution of white matter tracts, particularly the frontal aslant tract (FAT), in the development of speech disturbances and deficits in motor initiation post-SMA resection. The FAT is a recently characterized association pathway linking the pre-SMA and SMA with Broca's area in the inferior frontal gyrus. The authors analyzed 45 patients undergoing SMA-area glioma resections and found a strong correlation between postoperative SMA syndrome and disruption of the FAT.[6] This study underscores the importance of considering subcortical connectivity in surgical planning, as damage to the FAT may account for non-aphasic speech arrest seen in SMA syndrome. While tractography-based insights are compelling, they require validation through intraoperative mapping to ensure anatomical and functional congruence. The FAT has emerged as a key structure implicated in the speech-related features of SMA syndrome and represents a promising focus for future connectome analyses.

Recurrent SMA Syndrome After Repeat Resection

Further expanding on the mechanisms of SMA syndrome recurrence and brain plasticity, Abel et al. (2015) explored six cases where patients underwent repeat resections in the SMA region. All six experienced SMA syndrome following both surgeries, despite anatomical preservation of the eloquent motor cortex. The authors proposed that functional recovery after the first surgery occurred due to intra-hemispheric reorganization within nearby SMA or perilesional cortex. The recurrence of symptoms post-second surgery suggested that these newly adapted regions were also disrupted. This study challenges the long-standing assumption that recovery predominantly relies on contralesional compensation, instead highlighting the vulnerability of locally reorganized functional networks. These findings underscore the importance of comprehensive preoperative counselling and meticulous surgical planning when considering repeat interventions involving the SMA.[7]

Conclusion

Supplementary motor area syndrome represents a complex, yet largely reversible, constellation of deficits that pose significant perioperative challenges in neurosurgical practice. Contemporary evidence highlights that functional recovery is mediated not solely by spared cortical regions but also by contralesional and peri-lesional plasticity within a distributed motor network. Studies employing functional imaging, intraoperative stimulation, and tractography have elucidated both cortical and subcortical contributors to SMA syndrome, refining our risk stratification and surgical strategies. The reviewed literature underscores the necessity for a multimodal, individualized approach to

SMA-involving surgeries, integrating preoperative imaging, intraoperative mapping, and postoperative rehabilitation.

Importantly, the foundational observations by Laplane et al. remain relevant, particularly in defining the natural history of SMA syndrome and its recovery phases. Modern imaging and stimulation studies have expanded upon this foundation by delineating precise functional subregions and critical white matter connections, such as the frontal aslant tract, that mediate motor and speech functions. Moving forward, future research should prioritize prospective, longitudinal studies with standardized imaging and electrophysiological assessments to further characterize the temporal dynamics of recovery. The integration of connectomes and real-time intraoperative feedback holds promise for improving the precision of SMA resections. As surgical goals increasingly favour maximal safe resection, a sophisticated understanding of SMA syndrome and its nuances will remain essential for optimizing both oncological and functional outcomes.

References

1. Laplane D, Talairach J, Meininger V, Bancaud J, Orgogozo J. Clinical consequences of corticectomies involving the supplementary motor area in man. J Neurol Sci. 1977;34:301–14.
2. Krainik A, Lehéricy S, Duffau H, Vlaicu M, Poupon F, Capelle L, et al. Role of the supplementary motor area in motor deficit following medial frontal lobe surgery. Neurology. 2001;57:871–8.
3. Nakajima R, Kinoshita M, Yahata T, Nakada M. Recovery time from supplementary motor area syndrome: relationship to postoperative day 7 paralysis and damage of the cingulum. J Neurosurg. 2019;132:865-74.
4. Kasasbeh AS, Yarbrough CK, Limbrick DD, Steger-May K, Leach JL, Mangano FT, Smyth MD. Characterization of the supplementary motor area syndrome and seizure outcome after medial frontal lobe resections in pediatric epilepsy surgery. Neurosurg. 2012;70:1152-68.
5. Hiroshima S, Anei R, Murakami N, Kamada K. Functional localization of the supplementary motor area. Neurologia med-chirurg. 2014;54:511-20.
6. Briggs RG, Allan PG, Poologaindran A, Dadario NB, Young IM, Ahsan SA, Teo C, Sughrue ME. The frontal aslant tract and supplementary motor area syndrome: moving towards a connectomic initiation axis. Cancers. 2021;13:1116.
7. Abel TJ, Buckley RT, Morton RP, Gabikian P, Silbergeld DL. Recurrent supplementary motor area syndrome following repeat brain tumor resection involving supplementary motor cortex. Operative Neurosurg. 2015 ;11:447-56.