Long-term surgical results of supplementary motor area epilepsy surgery

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OBJECTIVE Supplementary motor area (SMA) epilepsy is a well-known clinical condition; however, long-term surgical outcome reports are scarce and correspond to small series or isolated case reports. The aim of this study is to present the surgical results of SMA epilepsy patients treated at 2 reference centers in Mexico City.

METHODS For this retrospective descriptive study (1999–2014), 52 patients underwent lesionectomy and/or corticectomy of the SMA that was guided by electrocorticography (ECoG). The clinical, neurophysiological, neuroimaging, and pathological findings are described. The Engel scale was used to classify surgical outcome. Descriptive statistics, Student t-test, and Friedman, Kruskal-Wallis, and chi-square tests were used.

RESULTS Of these 52 patients, the mean age at epilepsy onset was 26.3 years, and the mean preoperative seizure frequency was 14 seizures per month. Etiologies included low-grade tumors in 28 (53.8%) patients, cortical dysplasia in 17 (32.7%) patients, and cavernomas in 7 (13.5%) patients. At a mean follow-up of 5.7 years (range 1–10 years), 32 patients (61%) were classified as Engel Class I, 16 patients (31%) were classified as Engel Class II, and 4 (8%) patients were classified as Engel Class III. Overall seizure reduction was significant (p = 0.001). The absence of early postsurgical seizures and lesional etiology were associated with the outcome of Engel Class I (p = 0.05). Twenty-six (50%) patients had complications in the immediate postoperative period, all of which resolved completely with no residual neurological deficits.

CONCLUSIONS Surgery for SMA epilepsy guided by ECoG using a multidisciplinary and multimodality approach is a safe, feasible procedure that shows good seizure control, moderate morbidity, and no mortality.

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KEY WORDS epilepsy surgery; outcome; supplementary motor area; long-term; mesial frontal epilepsy

THE supplementary motor area (SMA), or supplementary sensorimotor area, was first described by Penfield and Welch.40 Penfield described epileptic seizures arising in this area.39 The SMA, which is located in the medial part of the frontal lobe, has variable anatomical limits and has been associated with voluntary motor control, such as the generation of complex movements (both initiation and execution), while the more rostrally located primary–supplementary motor area is associated with planning, sequencing, and the selection of movements.14,22,32,33,48 The SMA has a somatotopic organization, with a rostrocaudal representation of language, face, forelimbs, and lower extremities.32,36 The semiology of epileptic seizures originating in the SMA is variable and often represents a clinical challenge.1,6,11,20,22,32,36,46,49,51 Considering this confusing semiology and the fact that scalp electroencephalography (EEG) and video-EEG studies are notoriously inaccurate, subtle, or nonexistent, these seizures are often misdiagnosed. This is especially relevant because in nonlesional cases invasive procedures are more often required.1,4,32
Some case reports or small series of patients who underwent resective surgery of SMA lesions have been reported; however, not all patients in these series had intractable epilepsy. Furthermore, short- and long-term follow-up data for patients with intractable epilepsy who underwent surgery are lacking. The few studies with a small number of patients show that surgically treated patients with intractable SMA seizures are seizure free in up to 75% of cases. The aim of our study was to present the clinical, neuroimaging, neurophysiological, pathological features, and surgical results of surgically treated patients with intractable SMA epilepsy who were treated at 2 referral centers.

**Methods**

We performed a retrospective, descriptive study at 2 referral centers in Mexico City: National Institute of Neurology and Neurosurgery and Centro Neurologico ABC, Centro Medico ABC Santa Fe. We included all patients older than 18 years who consented to undergo SMA epilepsy surgery during the period from 1999 to December 2014. The study was approved by the institutional committees of each center.

All patients underwent a protocolized presurgical evaluation that included clinical evaluation by epileptologists, neurosurgeons, neuropsychologists, neuropsychiatrists, and neuroimaging studies (3.0-T MRI and T1-weighted, T2-weighted, FLAIR, and SPGR sequences), including functional MRI (movement initiation and planning paradigms) as well as PET and SPECT studies in selected cases. The studies were analyzed independently by 2 neuroradiologists. Scalp EEG monitoring and ictal and interictal video-EEG monitoring were performed in all cases. Patients with no MRI evidence of lesions had invasive monitoring using bilateral, 24-contact, recording grid meshes (6 x 4 contacts with 1-cm spacing [Auragen Precision Monitoring Electrodes, Integra LifeSciences]) that were placed on the frontal interhemispheric sulcus for 3 to 5 days in order to capture enough clinical seizures to determine the ictal zone and perform cortical motor mapping. We recorded the following sociodemographic and clinical variables: manual dominance, age, medical history, age at first seizure, age at time of surgery, duration of epilepsy, preoperative mean seizure frequency, psychiatric comorbidities, histopathological reports, preoperative neurological deficits, postsurgical complications, early postoperative seizures (that occurred at 1–7 days), and mean seizure frequency at 1 year and at the last follow-up visit.

**Surgical Procedure, Cortical Mapping, and Intraoperative Electrocorticography**

The same neurosurgeon with specialized epilepsy surgery training performed the surgery in all patients. The epileptogenic zone/lesion was resected using the subpial/endopial technique upon localization of the primary motor area by bipolar stimulation. The asleep-awake-asleep anesthetic technique for intraoperative mapping was used. All resections of the SMA were guided by intraoperative electrocorticography (ECoG) as described previously, thus resulting in a particular size, shape, and extension of the neocortical resection for each patient. ECoG was performed using a 64-channel digital EEG system and the Brain Explorer amplifier (both manufactured by EB Neuro S.p.A.). Signals were filtered at 0.03 to 70 Hz, with sensitivity set at 50 μV/second, and a 60-Hz notch filter. Twenty-four–contact recording grid meshes (6 x 4 contacts with 1-cm spacing [Auragen Precision Monitoring Electrodes, Integra LifeSciences]) were placed on the frontal interhemispheric sulcus. A reference electrode was placed at the ipsilateral mastoid apophysis. The anesthesia parameters were recorded and suspended regularly for 10 minutes prior to each ECoG recording with a mean duration of 15 minutes. A preresection ECoG recording site was deemed satisfactory when epileptiform activity originating in the SMA was recorded without a burst suppression pattern. Following resection, the mesh was replaced on the preresection location for the first postresection recording. Further recordings were performed after shifting the mesh at 1-cm increments over the mesial frontal neocortical areas until the epileptiform activity of the frontal lobe was almost or totally absent. Cortical mapping by direct cortical stimulation was performed according to a previously described technique.

**ECoG Analysis**

All ECoG recordings were carried out by a certified clinical neurophysiologist. We used the spike and wave definitions and terminology set forth by the Terminology Committee of the International Federation for EEG Societies and Clinical Neurophysiology. No epileptiform induction techniques were used during the ECoG recordings.

**Surgical Follow-Up**

Histopathological studies were performed by a certified neuropathologist at each medical center. Cortical dysplasias and tumors were characterized according to the Palmini and WHO classifications, respectively. All patients underwent follow-up at 1, 3, 6, 12, 18, and 24 months following surgery, in which postsurgical seizure control was staged using the Engel outcome scale, and the surgical complications were recorded.

**Statistical Analysis**

The results are expressed in percentages, standard deviations, and means. We used the Student t-test and chi-square, Friedman, and Wilcoxon signed-rank tests for paired samples. The analysis of the differences between the means according to the 3 etiologies was performed with the Kruskal-Wallis test, with post hoc analysis of the Mann-Whitney test adjusted with the Bonferroni correction. We used SPSS software (version 23.0, IBM). Statistical significance was set at p < 0.05.

**Results**

A total of 67 patients met the inclusion criteria, but 15 cases were excluded due to incomplete medical records or loss on follow-up. The final analysis included 52 patients—20 (38.4%) female and 32 (61.6%) male patients—with a mean age of 37 ± 10.5 years, and 52 patients (98%) were left-handed. The level of schooling in years was as...
Table 1. Seizure semiology in 52 patients who underwent SMA epilepsy surgery

<table>
<thead>
<tr>
<th>Semiology</th>
<th>No. of Patients (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sudden onset w/ loss of consciousness</td>
<td>36 (69)</td>
</tr>
<tr>
<td>Pericranial headache</td>
<td>16 (31)</td>
</tr>
<tr>
<td>Contralateral hemibody paresthesias</td>
<td>16 (31)</td>
</tr>
<tr>
<td>Fencing posture</td>
<td>33 (63)</td>
</tr>
<tr>
<td>Vocalizations</td>
<td>1 (2)</td>
</tr>
<tr>
<td>Speech arrest</td>
<td>1 (2)</td>
</tr>
<tr>
<td>SPS w/ secondary generalization</td>
<td>8 (15)</td>
</tr>
<tr>
<td>Generalized tonic-clonic seizures</td>
<td>11 (21)</td>
</tr>
</tbody>
</table>

Trigger factors

- Sleep deprivation: 7 (14)
- Stress: 6 (11)

Circadian pattern

- Nocturnal: 37 (71)
- No circadian predominance: 15 (29)
- Did not report time of seizures: 1 (2)

SPS = simple partial seizures.

Follows: 19 (37%) patients had 18 years of education, 17 (33%) patients had 15 years, 7 (13%) patients had 12 years, 6 (11%) patients had 22 to 24 years, 2 (4%) patients had 24 to 30 years, and 1 (2%) patient had no formal education. The relevant data in the medical history included a history of brain trauma in 5 patients (9.6%), high blood pressure in 3 patients (5.6%), diabetes mellitus in 2 patients (4%), allergies in 2 patients (4%), and neuroinfection, neonatal hypoxia, alcoholism, and rheumatoid arthritis in 1 patient each (2%).

Presurgical Evaluation of Frontal SMA Epilepsy

The mean age at seizure onset was 26.3 ± 14.7 years, and the mean durations of epilepsy and pharmacoresistance were 10.9 and 37.9 years, respectively. The basal preoperative seizure frequency was 7.5 years. The distribution of patients according to the seizure type was as follows: 20 (38%) patients had hypermotor seizures, 18 (35%) patients had generalized tonic-clonic seizures, 10 (19%) patients had hypermotor seizures with secondary generalization, and 3 (6%) patients had simple partial seizures with secondary generalization. Details of seizure semiology are shown in Table 1.

EEGs and interictal/ictal video-EEGs with epileptiform activity were scarcely localizing in the majority of patients (28 of 52 patients; 54%). The neurophysiological findings are shown in Table 2. MR images showed abnormalities in 42 of 52 patients (81%). All patients with normal MRI findings underwent functional studies such as PET-CT and/or ictal/interictal SPECT and invasive monitoring. Lesions were found in the left SMA in 77% of patients, while 23% of patients had right-sided lesions. Abnormal presurgical neurological examination findings were noted in 9 patients, including 5 patients with hemiparesis (3 left and 2 right), 2 patients with right hemihyposthesia, 1 patient with ideomotor apraxia, and 1 patient with dysarthria.

Table 2. Ictal and interictal scalp EEG findings in 52 patients with SMA epilepsy

<table>
<thead>
<tr>
<th>Localization</th>
<th>Epileptiform Discharges</th>
<th>Focal Slowing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rt frontocentral</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Lt frontocentral</td>
<td>15</td>
<td>1</td>
</tr>
<tr>
<td>Rt frontal</td>
<td>1</td>
<td>7</td>
</tr>
<tr>
<td>Lt frontal</td>
<td>3</td>
<td>11</td>
</tr>
<tr>
<td>Rt frontotemporal</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Lt frontotemporal</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Bifrontal</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>27 (52)</td>
<td>25 (48)</td>
</tr>
</tbody>
</table>

Data are presented as the number of patients (%).

Surgical Procedure and Early Complications

ECoG showed epileptic activity of the SMA in all cases and was used to guide the resection of the epileptogenic lesion/area using a subpial/endopial technique that resulted in no residual postresection epileptogenic activity (Figs. 1 and 2). No intraoperative complications were encountered.

The following complications were registered in the immediate postoperative period (24 hours) in 26 of 52 (50%) patients: 21 of 26 (80.7%) patients had new, mild (Medical Research Council Scale Grade 4 of 5) contralateral hemiparesis (15 right-sided and 6 left-sided) and returned to baseline status in 3 to 6 months; and 3 of 26 (11.6%) patients had akinetic mutism and returned to baseline status in 4 weeks. Finally, 2 of 26 (7.7%) patients developed severe surgical and anesthesia-reversible complications: 1 subdural hematoma and 1 case of respiratory acidosis.

FIG. 1. T1-weighted sagittal MR images obtained in 2 patients: 1 patient with low-grade gliomas (A and B), and the other patient with cavernoma (C and D). Presurgical (A) and postsurgical (B) MR images obtained in a 36-year-old male with Grade I astrocytoma in the left SMA. The double-headed arrow (A) indicates the size of the tumor, and the single-headed arrows (B) indicate the tumor margins. Presurgical (C) and postsurgical (D) MR images with gadolinium obtained in a 33-year-old female with a left-sided SMA cavernoma. At the 1-year follow-up, both patients were classified as Engel Class I. Figure is available in color online only.
Two patients with previous (1 right and 1 left) hemiparesis improved in the immediate postoperative period. In terms of etiology, the neurological complications were distributed as follows: 15 of 26 (57%) patients had low-grade tumors, 8 of 24 (33%) patients had cortical dysplasia, and 3 of 26 (10%) patients had cavernomas. Seventeen of 26 (65%) patients underwent operations on the dominant hemisphere, and 7 of 26 (27%) patients had normal findings on presurgical head MRI.

Seizures were present in the first 3 postsurgical days in 12 of 52 patients (23%), and 4 of these 12 patients (33%) continued to have seizures at the 6- and 12-month follow-up visits. Overall, 28 of 52 (54%) patients were seizure free (Engel Class IA) at the last visit (mean 5.7 years; range 1–10 years).

Etiology

All of our patients had a lesional epileptic etiology that was confirmed pathologically. Low-grade tumors, which were found in 28 of 52 (53.8%) patients, were the most common lesions, followed by cortical dysplasia in 17 of 52 patients (32.7%) and cavernomas in 7 of 52 patients (13.5%); 1 patient had multiple cavernomas (Fig. 2). All neoplastic lesions were low-grade tumors and classified as follows: 12 of 28 (42.8%) patients had WHO Grade II oligoastrocytomas, 9 of 28 (32.1%) patients had WHO Grade II astrocytomas, 3 of 28 (10.7%) patients had WHO Grade II oligodendrogliomas, 2 of 28 (7.1%) patients had primitive neuroepithelial tumors, and 2 of 28 (7.1%) patients had gangliogliomas. According to the Palmini classification, 17 cases of cortical dysplasia were classified: 10 of 17 cases were Class IIB, 5 of 17 cases were Class IIA, and 2 of 17 cases were Class IB. Cortical dysplasia was the underlying etiology in all cases with normal MRI and was evidenced only in 7 patients by this imaging method.

Long-Term Postsurgical Results

The mean seizure frequency at the 1-year postsurgical follow-up was 3.5 seizures/month and 3 seizures/month at the time of the last visit (range 1–10 years). The overall seizure reduction was significant at the follow-up (p = 0.001), and the seizure-free Engel IA stage was observed in 28 of 52 (53.8%) patients.

Based on etiology, the baseline frequencies of the seizures between the 3 etiologies (cortical dysplasia, glioma, and cavernoma) using the Kruskal-Wallis test were significantly different (p = 0.001), specifically between the patients with cortical dysplasia and gliomas (p = 0.001; Mann-Whitney test). However, we did not find any differences in the frequencies of the seizures between these groups at the follow-up (p = 0.064) using the Kruskal-Wallis test. The seizure reduction showed statistical significance for each group (p = 0.012 for cortical dysplasia; p = 0.001 for gliomas; p = 0.028 for cavernomas; according to the Wilcoxon signed-rank test) (Fig. 3).

In total, 16 of 28 (57.1%) patients with low-grade tumors, 7 of 17 (41.1%) patients with cortical dysplasia, and 5 of 7 (71.4%) patients with cavernomas were seizure free. According to the Engel classification at a mean follow-up of 5.7 years, 61% of patients were Engel Class I, 31% were Engel Class II, and 8% were Engel Class III.

A significant relationship between early postsurgical seizures and a seizure-free condition was revealed ($\chi^2 = 8.7994$, p = 0.05). In particular, the presence of postsurgical early seizures was seen in 12 of 52 (23%) patients, and of these 2 of 12 (17%) patients were seizure free in the follow-up period. Moreover, 26 of 40 (65%) patients without early postsurgical seizures were seizure free. Also, a significant relationship between lesional etiology and the seizure-free condition was exhibited ($\chi^2 = 7.4421$, p = 0.05). No significant statistical relationship between the side of the lesion on MRI and seizure-free condition, type of seizure and seizure-free condition, or sex and seizure-free condition was found.

Discussion

Our study showed that 61% of patients with lesional SMA epilepsy who underwent surgery guided by ECoG were in Engel Class I at a mean follow-up of 5.7 years with moderate morbidity and no instances of mortality. These
findings confirm the results of previous small-series studies on frontal epilepsy surgery in adults and children that reported seizure freedom in 50% to 75% of patients.\textsuperscript{23,37,41,54} To the best of our knowledge, this is the largest series that reports long-term seizure outcome in surgically treated adult patients with SMA epilepsy.

Surgical control of seizures is primarily dependent on the careful selection of patients whose epilepsy is pharmacologically resistant, in whom the convergence of information gathered through neurophysiological, neuroimaging, and psychological studies and complemented by nuclear medicine and invasive monitoring in selected cases, allows the precise identification of the epileptogenic area. The placement of subdural mesh is essential for better localization and delimitation of the epileptogenic area, specifically in nonlesional cases.\textsuperscript{7,14,18,20,22,29,32,35,46}

Fifty percent of our patients exhibited a transitory postsurgical neurological deficit (akinet mutism and hemiparesis) that resolved in 3 to 6 months, corresponding to the so-called SMA syndrome\textsuperscript{22,50,52} described by Laplante et al. in 1977.\textsuperscript{28} The syndrome is characterized by mild-to-moderate transitory hemiparesis/hemiplegia or motor apraxia with variable degrees of speech arrest followed by a rapid (3–8 months) recovery of neurological dysfunction.\textsuperscript{53} SMA syndrome has been reported in 23% to 100% of surgical cases. Most often, this was observed hours after surgery and improved gradually over the next few days, with restoration of motor strength within 1 day to 1 week of surgery in 41% of cases. The vast majority of cases (82%) resolve by the 1st month.\textsuperscript{3,8,10,16,23,25,28,41,43–45,50,54} However, residual fine motor deficits in the form of the impairment of alternating movements of the hands after 1 year of follow-up has been reported in small series (range 3–12 patients).\textsuperscript{28,50}

A few factors have been associated with an increased risk for the development of SMA syndrome or irreversible neurological deficits, including the resection of the eloquent areas of the SMA involved in ictal onset, increased threshold or lack of motor responses on presurgical cortical mapping,\textsuperscript{23} resections within 1 cm of the eloquent area or precentral gyrus,\textsuperscript{23,62} resections in the dominant hemisphere,\textsuperscript{6,7,14,22,35} and normal presurgical MRI findings.\textsuperscript{23} In our study, 81% of patients showed abnormalities on presurgical MRI and no resection of the eloquent areas was needed to complete lesional resections. On the other hand, ECoG aided in the delimitation of irritative zones in cases with normal MRI findings, enabling complete resections that were pathologically confirmed as cortical dysplasias. Previous studies have demonstrated the utility of ECoG guidance in frontal epilepsy surgery for preserving the eloquent areas and improving surgical outcome in cases of cortical dysplasias that were not evidenced on MRI.\textsuperscript{3,5,9,13,21,27} Some authors recommend including cortical mapping and intraoperative somatosensory evoked potentials as invaluable neurophysiological tools for preserving neural function in SMA epilepsy surgery.\textsuperscript{23,41,54}

In a recent retrospective study that included 35 pediatric patients who underwent mesial frontal lobe surgery, 59% of patients exhibited postsurgical neurological deficits, of which 74% corresponded to SMA syndrome and 15% developed permanent neurological deficits. Symptoms were resolved in the majority of patients within 1 month after surgery.\textsuperscript{23} Our adult patients showed no new permanent neurological deficits or deaths, which supports the safety of the SMA surgical procedure using the subpial/endopial technique.\textsuperscript{23,41,54} In fact, 2 patients with previous hemiparesis improved postsurgically.

Histopathological studies revealed an underlying lesion in all our cases, with low-grade tumors representing the most frequent etiology followed by cortical dysplasias, and these 2 causes combined represent 86% of cases. Our remaining patients had cavernomas as the underlying etiology, showing a distribution that is similar to that of other reported frontal epilepsy surgical series.\textsuperscript{54} Independent of etiology, all patients showed a significant reduction in seizures at the follow-up. Interestingly, 50% of the patients who developed SMA syndrome underwent resections for low-grade tumors followed by cortical dysplasia (33%), and this finding could be related to the infiltrative biology of these pathologies.\textsuperscript{2,41,44}

The clinical and sociodemographic characteristics of our patients are similar to those of other SMA surgical series.\textsuperscript{23,41,54} Only 47% of our patients had SMA clinical seizures, although lesonal SMA epilepsy was confirmed pathologically in 100% of patients. In concordance with previous studies, we found significant relationships between variables: early postsurgical seizure versus seizure free, and lesional diagnosis versus seizure free.\textsuperscript{23}

The noninvasive neurophysiological findings were generally nonlateralizing or/and without epileptiform activity in many cases. Most abnormal studies showed lateralized, mild-to-moderate focal slowing in the frontal lobes, and when epileptiform activity was present it was seen over the frontocentral regions. It has been previously reported that the value of scalp EEGs in localizing seizures arising from the SMA is limited due to restricted access to the mesial frontostructures, rapid transcallosal propagation of electric activity to neighboring structures, involvement of the contralateral hemisphere, and the interference of motor artifacts.\textsuperscript{24} When sharp waves and interictal spikes are present, they are generally confined to the vertex and may be confused with normal EEG patterns, especially during
sleep, or they can show false lateralization.\textsuperscript{32} It has been reported that in the majority of clinical SMA seizures, the epileptogenic zone is localized in other mesial frontal and even extra frontal structures located primarily in the mesial parietal cortex.\textsuperscript{10,12,26,30,35} Intercital video-EEG studies generally show rapid paroxysmal activity or electrodecrement at the moment of ictal onset, and epileptiform fronto-central discharges have been described in 50\% of patients with asymmetrical bilateral tonic seizures.\textsuperscript{24} Localization of the epileptogenic area in patients who exhibit clinical seizures in the SMA correlates to EEG and imaging findings in only 20\% of cases.

Intraoperative cortical mapping and ECoG allow the precise definition of the extension of epileptic activity and eloquent areas of the SMA that are interconnected to the contralateral SMA, cingulum, premotor area, dorsolateral prefrontal cortex, cerebellum, basal ganglia, and parietal association areas.\textsuperscript{11,26} Some limitations of our study are the lack of a postsurgical neuropsychological profile; heterogeneous pathologies; the different sizes, shapes, and extensions of neocortical resection in each patient; and the age at onset of symptoms between the different etiologies, which potentially could impact neurological functional outcome. The need for further studies that define the prognostic factors and the value of ECoG in SMA epilepsy has been previously mentioned.\textsuperscript{37} Concomitantly, larger series reports on subdural and deep electrodes are needed to characterize the electrophysiological profile of SMA seizures.\textsuperscript{35} Furthermore, the use of structural, higher resolution MRI, new methods\textsuperscript{26,47} and functional images with newly developed paradigms for complex movement initiation and sequencing, and SMA tractography will provide further information for characterizing lesional epilepsies and aid in neurosurgical planning in order to reduce the incidence of complications and improve impact on seizure control.

Conclusions

In our series, 61\% of patients who underwent lesional SMA epilepsy surgery guided by ECoG were Engel Class I at a mean follow-up of 5.7 years, and 31\% showed only rare seizures (Engel Class II) with moderate morbidity and no instances of mortality.

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References

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Disclosures

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