Insular glioma

see also insuloopercular glioma.

Insular gliomas have specific histology and natural history, are most often low grade \textsuperscript{1}.

Insular gliomas represent a unique category within intrinsic brain tumors in terms of their presentation and behavior. These tumors usually arise in areas of white matter adjacent to allocortex or mesocortex. The insula is a mesocortical structure (3-5 cellular layers) along with the temporal pole, caudal orbitofrontal cortex, and cingular and parahippocampal gyri. The cytoarchitecture of this region of the brain explains the growth pattern and expansion of these tumors. Thus, during the initial phases of growth, the tumor tends to be confined within the allo- and mesocortical areas, respecting the neocortical areas, central nuclei, and ventricles \textsuperscript{2}.

**Epidemiology**

They are quite frequent, however treatment of patients with this pathology still remains a challenging and controversial issue of neurosurgery.

The insular lobe and paralimbic region represent a common location for gliomas. Up to 25% of low-grade and 10% of high-grade newly diagnosed gliomas are found in this region \textsuperscript{3}.

**Classification**

see Insular tumor classification.

**Clinical features**

They are typically large at the time of presentation and frequently cause epilepsy as their only initial symptom \textsuperscript{4,5,6}.

Patients with insular gliomas have significant impairment of autonomic functions with left insular glioma showing sympathetic dominance. Suppression of autonomic function is more in those presenting with seizures \textsuperscript{7}.

**Diagnosis**

Magnetic resonance imaging (MRI) with T2-weighted and Fluid Attenuated Inversion Recovery (FLAIR) images best delineates the extent of tumour infiltration, which can be limited to the insular lobe (Yasargil type 3a) or reach the perisylvian opercula (type 3b) and other paralimbic areas, namely the orbitofrontal and temporopolar regions (type 5), with or without involvement of core limbic structures \textsuperscript{8}.

**Treatment**

Frequently managed conservatively regardless of their nature and clinical evolution, even if impending infiltration of nearby eloquent areas will further promote dysfunction, but surgical resection of these lesions is nonetheless feasible since tumour burden often displaces more than it incorporates eloquent sites.
see **Insular glioma surgery**.

**Surgical treatment**

see **Insular tumor surgery**

**Outcome**

Prognosis of insular tumor resection is still controversial. Further analysis of subgroup characteristics of insular grade II gliomas based on clinical and molecular analysis is required to reliably determine patients' survival rates.

Although a number of studies have shown that greater extent of resection improves overall patient survival, few studies have documented postoperative seizure control after insular tumor resection.

**Complications**

The interruption of the lenticulostriate arteries during resection of insular tumors may result in hemiparesis and that these arteries should be preserved to prevent infarction in the corona radiata.

Three dimensional magnetic resonance imaging based on time of flight magnetic resonance angiography was performed pre- and postoperatively in patients with insuloopercular gliomas. This 3D 3-T TOF MR imaging clearly visualized the lenticulostriate arteries (LSAs) and the relationships with the tumor margins. These findings were confirmed intraoperatively. Three-dimensional 3-T TOF MR imaging of the LSAs in patients with insuloopercular gliomas can help to maximize the extent of resection without neurological complications, preserve the LSAs during surgery, and assist in patient selection.

M1 segment of the middle cerebral artery, M2 segment of the middle cerebral artery, lenticulostriate arteries, basal ganglia, and internal capsule involvement, causes a high rate of postoperative complications in these patients.

**Recurrence**

While the benefits of an extensive initial resection of a insular glioma have been widely demonstrated, the best management of residual tumor still represents an open question.

Only 3 investigations analyzed the role of second surgery in case of tumor recurrence (TR). Only 3 investigations analyzed the role of second surgery in case of tumor recurrence (TR).

For the first time Schmidt et al. provided clinical evidence of the safety of a second surgery in 40 patients.

Martino and coworkers analyzed the clinical outcomes of 19 patients with recurrent LGGs in eloquent areas, strengthening the concept of possible functional reshaping occurrence after the first surgical procedure.

In line with these findings, Ius et al., showed that a second surgery is a safe and effective procedure, even for recurrent insular low grade gliomas.

Another possible reason for the positive outcome after a second surgery may be the smaller tumor volume at relapse.
The investigation of Ius et al., also highlights that seizure recurrence in patients who were seizure-free after the first surgery is associated with tumor progression.

From a strictly surgical point of view, there are some technical key points to take into consideration at second surgery. At recurrence, there is no intracranial hypertension. Tumor recurrence volume is smaller than the volume at first surgery and the cavity left by the previous operation allows a larger surgical field. The recurrent mass of tumor tissue mainly regrows from the walls of the previous resection into the cavity.

Ius et al., have noticed, also, a better definition between the healthy parenchyma and the tumor tissue, which is softer and, consequently, easier to remove. Moreover, at second surgery, the risk of damaging the vascular structures is much lower, because dissection of the middle cerebral artery (MCA) and its branches has already been performed during the first surgical procedure.

The only difficulty of second surgery is represented by the adhesions. They may cause pain during the opening; moreover, adhesions between dura mater and cortex, on the dominant side, may represent a risk of damage to the cortical language areas. In conclusion, the newly infiltrated deep tumoral tissue is not resected if it has been shown to still be functional based on brain mapping results.

The study of Ius et al., has potential limitations. First, it is a retrospective study; thus it is limited in nature. Patients with recurrence insular LGGs that are suitable for second surgery are per se highly selected. Thus, the number of our samples is limited, but, if we consider the papers, mentioning insular second surgery, the overall number of patients is 32; thus the study population (23 patients) is not considerably small and it is statistically sufficient to draw some preliminary considerations, which need to be confirmed by enlarging the case study. Moreover, insular surgery is rare at first diagnosis and even rarer at second surgery, so it is not easy to find large population in literature. In any case, it is unlikely that a prospective, randomized study will be designed to address these issues; thus, they believe retrospective, matched studies or prospective observational trials may be a more practical solution, as previously described.

The findings should be validated in a wider series, using multi-institutional cohort to create a potential model able to stratify the risk of TR after the first surgery. In this way, it would be possible to anticipate adjuvant postoperative treatments, also in patients with a diagnosis of pure LGG.

The timing of second surgery has not been well defined yet. Anyway, as previously remarked by Martino et al, it is better to “overindicate” an early second surgery than performing a late surgery when the tumor has already transformed into high-grade gliomas, especially in consideration of the low morbidity profile associated with reoperation.

Case series

see Insular glioma case series.

2) Yaşargil MG: Microneurosurgery. Stuggart: Georg Thieme Verlag, 1994
3)


