Skull base meningioma

- Massive Foramen Magnum Meningioma Mimicking a Stroke
- Management of skull base meningiomas with extracranial extension: resection, recurrence, and prognostic factors
- Impact of tumor size and peritumoral edema on outcomes and complications in anterior midline skull base meningiomas
- Face the Pain: Radiobiological and Clinical Considerations of Re-radiosurgery to the Trigeminal Nerve Following Irradiation of an Abutting Petroclival Meningioma
- Effect of routine extradural optic canal decompression performed by skull base trained surgeons on visual outcomes in patients with anterior skull base meningiomas
- Patient nutritional status is associated with surgical site infections in meningioma patients undergoing craniotomy for tumor resection
- Efficacy and safety of stereotactic radiosurgery for large meningiomas: A comprehensive systematic review and meta-analysis
- Oral contraceptives with progestogens desogestrel or levonorgestrel and risk of intracranial meningioma: national case-control study

Type of Intracranial meningioma

Epidemiology

Skull base meningioma epidemiology.

Classification

see Anterior skull base meningioma.

see Central skull base meningioma.

see Middle skull base meningioma.

see Posterior fossa meningioma.

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see Pediatric Skull Base Meningioma.

Etiology

Genetic aberrations (TRAF7, KLF4, AKT1, and SMO) and the effects of genetic aberrations on the expression of immune checkpoint inhibitory molecules (PD-L1, IDO, and TDO2) in skull base meningiomas are still unclear.

Genetic alterations in the four genes were identified in 92 skull base meningiomas by Sanger sequencing. The expression differences in immune checkpoints between mutant and wild-type (WT) tumors were determined by immunohistochemistry (IHC) and Western blot (WB).

The four mutations were not concurrently detected in the patients with skull base meningiomas. Among the tumors from the KLF4-mutated group, almost half were petroclival meningiomas. KLF4- and TRAF7-mutated tumors were predominantly secretory meningiomas. SMO-mutated tumors exhibited higher calcification, and half of these tumors were observed in the brain midline. Receiver operating characteristic curve analysis indicated that tumor volume can predict KLF4 and TRAF7 mutation status with high sensitivity and specificity, respectively. The IHC and WB analyses indicated that PD-L1, IDO, and TDO2 levels in tumors with TRAF7 mutations were significantly higher than those in WT tumors. Meanwhile, there was a significant difference in TDO2 between tumors with AKT1 mutations and WT tumors. Specifically, TRAF7 mutations could play a key role in skull base meningiomas by regulating the expression of inhibitory immune checkpoints and thus suppressing immune responses.

Checkpoint inhibitors may be potential strategies for targeted immunotherapies of these mutant meningiomas [1].

Treatment

Skull base meningioma treatment.

Outcome

Skull base meningioma outcome.

Case series

see Skull base meningioma case series.

1)
Hao S, Huang G, Feng J, Li D, Wang K, Wang L, Wu Z, Wan H, Zhang L, Zhang J. Non-NF2 mutations have a key effect on inhibitory immune checkpoints and tumor pathogenesis in skull base meningiomas. J Neurooncol. 2019 Jun 8. doi: 10.1007/s11060-019-03198-9. [Epub ahead of print] PubMed PMID: 31177425.