Parasellar tumor

Although invasive pituitary tumors are the most common neoplasms encountered within the parasellar region, other tumoral (and cystic) lesions can also be detected. Craniopharyngiomas, meningiomas, as well as Rathke's cleft cysts, chordomas, and ectopic pituitary neuroendocrine tumors can primarily originate from the parasellar region. Except for hormone-producing ectopic pituitary tumors, signs and symptoms of these lesions are usually nonspecific, due to a mass effect on the surrounding anatomical structures (i.e. headache, visual disturbances), while a clinically relevant impairment of endocrine function (mainly anterior hypopituitarism and/or diabetes insipidus) can be present if the pituitary gland is displaced or compressed. Differential diagnosis of parasellar lesions mainly relies on magnetic resonance imaging, which should be interpreted by neuroradiologists skilled on base skull imaging. To date, neurosurgery is the main treatment, alone or in combination with radiotherapy. Of note, recent studies have identified gene mutations or signaling pathway modulators that represent potential candidates for the development of targeted therapies, particularly for craniopharyngiomas and meningiomas. In summary, parasellar lesions still represent a diagnostic and therapeutic challenge. A deeper knowledge of this complex anatomical site, the improvement of imaging tools, as well as novel insights in the pathophysiology of presenting lesions are strongly needed to improve the management of parasellar lesions 1).

3D FIESTA magnetic resonance imaging is useful for detection of oculomotor nerve compression, especially in the field of parasellar region lesions ²⁾.

Treatment

Stereotactic radiosurgery (SRS) is an accepted treatment option for patients with benign parasellar tumors. Graffeo et al. objective were to determine the risk of developing new or progressive internal carotid artery (ICA) stenosis or occlusion after single-fraction SRS for cavernous sinus meningioma (CSM) or growth hormone-secreting pituitary neuroendocrine tumor (GHPA).

They queried their prospectively maintained registry for patients treated with single-fraction SRS for CSM or GHPA in the period from 1990 to 2015. Study criteria included no prior irradiation and ≥ 12 months of post-SRS radiological follow-up. Pre-SRS grading of ICA involvement was applied according to the 1993 classification schemes of Hirsch for CSM or Knosp for GHPA.

The authors conducted a retrospective review of 283 patients, 155 with CSMs and 128 with GHPAs. Ninety-three (60%) CSMs were Hirsch category 2 and 3 tumors; 97 (76%) GHPAs were Knosp grade 2-4 tumors. Median follow-up after SRS was 6.6 years (IQR 1-24.9 years). No GHPA or category 1 CSM developed ICA stenosis or occlusion. Three (5.2%) patients with category 2 CSMs had asymptomatic ICA stenosis (n=2) or occlusion (n=1); 1 (1.1%) category 2 CSM patient had transient ischemic symptoms. Five (14.3%) category 3 CSMs progressed to ICA occlusion (4 asymptomatic, 1 symptomatic). The median time to stenosis/occlusion was 4.8 years (IQR 1.8-7.6). Five- and 10-year risks of ICA stenosis/occlusion in category 2 and 3 CSM patients were 7.5% and 12.4%, respectively. Five- and 10-year risks of ischemic stroke from ICA stenosis/occlusion in category 2 and 3 CSM patients were both 1.2%. Multivariate analysis showed patient age (HR 0.92, 95% CI 0.86-0.98, p=0.01), meningioma pathology (HR and 95% CI not defined, p=0.03), and pre-SRS carotid category

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(HR 4.51, 95% CI 1.77-14.61, p=0.004) to be associated with ICA stenosis/occlusion. Internal carotid artery stenosis/occlusion was not related to post-SRS tumor growth (HR and 95% CI not defined, p=0.41).

New or progressive ICA stenosis/occlusion was common after SRS for CSM but was not observed after SRS for GHPA, suggesting a tumor-specific mechanism unrelated to radiation dose. Pre-SRS ICA encasement or constriction increases the risk of ICA stenosis/occlusion; however, the risk of ischemic complications is very low ³⁾.

1)

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3)

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