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Chapter

Meningiomas

İsmail Kaya and Hüseyin Yakar

Abstract

Meningiomas are among the most common central nervous system (CNS) tumors worldwide. These extra-axial lesions, which usually originate from neoplastic arachnoidal (meningothelial) cells, often appear in mid-late adulthood and are more common in women. Due to their heterogeneous morphology, the World Health Organization (WHO) divided meningiomas into three main groups, and these three main groups are divided into nine subgroups with histopathological differences according to their biological behavior. Clinical signs and symptoms, as in other central nervous system tumors, vary considerably depending on the compression or invasion of the neurovascular structures in the compartment where the meningioma is located. Meningiomas that are presented as benign lesions often have the potential to grow slowly, but could be associated with morbidity, such as poor quality of life, depending on the histopathological grade and localization of the lesion. Although fractionated radiotherapy or stereotactic radiosurgery is an alternative treatment option for meningiomas that cannot be completely removed (surgically inaccessible, or recurrent (atypical or anaplastic)) the primary treatment for these lesions is surgery. In this context, we have detailed meningiomas in this section.

Keywords: central nervous system tumors, intracranial meningioma, clinic, diagnosis, treatment

1. Introduction

Meningiomas, i.e., nonglial and extra-axial tumors of the central nervous system (CNS), are benign primary brain tumors that arise from neoplastic arachnoid (meningothelial) cells [1]. These epithelial cells, usually located in the arachnoid villi of the brain and spinal cord (rarely in the ventricles and extracranial area), are a component of the meninges that protect the brain [1]. Meningothelial cells, which form the interface between the parenchymal neurons and the cerebrospinal fluid (CSF), have an important barrier function for the CNS [1]. Thus, these cells are involved in removing waste products from the CSF with the protection of the optic nerve microenvironment and play a role in immunological processes through the secretion of proinflammatory cytokines in response to pathological stress conditions [2].

Felix Plater first defined meningioma in autopsy reports in 1614 as a roundish, fleshy, hard tumor with holes and the size of a medium-sized apple, covered with its membrane, and interspersed with venous lesions [3]. In the following years, different names were used for these pathological extra-axial structures, such as fungus durae matrix (Antoine Louis, 1774) and psammoma (Virchow, 1847), epithelioma
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(Bouchard, 1864), endothelioma (Golgi, 1869) [4–7]. In 1922, Harvey Cushing's current definition of "meningiomas" prevailed in the literature to eliminate the diversity and confusion of this definition and to group many different pathologic tumor types arising from the meninges [6].

Meningiomas are common tumors, accounting for approximately 36% of all CNS tumors and 53% of non-malignant CNS tumors [8].

While meningiomas present as benign lesions they are often associated with treatable focal neurologic deficits and epileptic seizures, they may rarely be associated with morbidity, such as poor quality of life, depending on the histopathologic grade and location of the lesion [8].

Although the treatment principles are almost the same, spinal meningiomas and meningiomas of childhood are not discussed in this section to maintain the integrity of the topic. In this section, we aimed to give basic information about meningiomas, the importance of which we briefly mentioned.

2. Epidemiology, incidence, and prevalence

The incidence of meningiomas is based on hospital- or population-based information [8]. In parallel with developments in neuroradiology and increasing accuracy of disease reporting, the incidence has gradually increased over the years [9]. Hospital-based brain tumor series reported approximately 20% of all intracranial tumors, whereas autopsy studies found an overall incidence of almost 30% [10]. Meningiomas have the highest incidence rate among CNS tumors [10, 11]. They account for 38% of all intracranial tumors in women and 20% in men [10, 11]. Population-based studies have found an overall annual incidence of 6/100,000 [10, 11]. The incidence increases significantly with age. It is considered an age-related incidence, being 0.3/100,000 in childhood and 8.4/100,000 in the elderly [12, 13]. Intracranial meningiomas are most common in adults between the fourth and sixth decade [13]. While the incidence increases in patients with breast tumors and after head trauma, it reaches the highest rates after 50 years [9, 14, 15]. Although intracranial tumors have an overall higher prevalence in men than in women, the situation is reversed for meningiomas (women/men: 2/1) [9]. It is suggested that this increase is due to steroid receptors activating tumor growth [9]. However, female dominance of meningiomas, which is reported to be more common in Black people, has not been demonstrated in Black people [9]. Prevalence rates vary from 50.4/100,000 to 70.7/100,000 [16, 17]. Asymptomatic meningiomas are estimated to be discovered incidentally in 2–3% of the population and more than one in 8% of these cases [17]. Interestingly, non-malignant meningiomas are predominantly female, whereas atypical and anaplastic meningiomas are more common in men [18].

3. Neuropathological features and classification

Meningiomas are neoplastic changes of meningothehial cells tasked with barrier-like functions [2]. They originate from any region where the dura mater is located, often from the skull base and rarely from the extensions of the dura mater such as falx cerebri, tentorium cerebelli, and rarely from the optic nerve sheath and internal choroid plexuses in the ventricles [1]. Meningiomas, an extra-axial lesion, have a slow growth character, and macroscopically, a CSF cleft may be present adjacent to the...
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These masses often present as a single, lobulated, and solitary extra-axial lesion [19]. Multiple lesions defined as “meningiomatosis” in syndromic patients, such as neurofibromatosis type 2 (NF2), can be seen [19].

Meningiomas can be classified according to their dural site of origin, involvement of adjacent tissues (e.g., bone, venous sinus, nerves, and brain), and histologic grading [20]. According to the World Health Organization (WHO), meningiomas are classified into three main groups based on their heterogeneous morphology: WHO Grade 1 (80% most common), WHO Grade 2 (10–18%), WHO Grade 3 (2–4% most aggressive) [20]. These three main groups were also subdivided into subgroups with histopathological differences [20]. This classification is based on pathologic criteria and is used to estimate the tumor progress (Table 1) [19, 20]. WHO Grading depends on brain invasion, specific histopathological features, or mitotic rate [21]. Although grade 1 meningiomas are designated as benign histopathologically, and they have a low recurrence rate of 5 years after surgery, the lifetime recurrence rate is approximately 30% [22]. In contrast, the 5-year recurrence rate for atypical (WHO 2) and anaplastic (WHO 3) meningiomas can be as high as 50% and 80% [21, 22]. The increase in recurrence rate after surgery is related to factors such as high-grade meningioma, brain or bone involvement, and a high proliferation index [23, 24]. However, it is impossible to determine which tumors will recur, based on the histologic criteria alone [20]. In addition, grade I and grade II meningiomas can progress to grade III by malignant transformation, but it is still unclear in which cases such progression occurs [20]. To this end, molecular characterization of meningiomas has defined several genetic biomarkers that can hopefully predict tumor behavior, and clinical trials are underway to address genetic subtypes [20]. These include BAP1 (rhabdoid and papillary subtype), SMARCE1 (clear cell subtype), TERT promoter mutation, and/or homozygous deletion of CDKN2A/B (CNS WHO grade 3), H3K27me3 loss of nuclear expression (potentially worse prognosis), KLF4/TRAF7 (secretory subtype) mutations, and methylome profiling (prognostic subtyping) [20].

3.1 WHO classification

3.1.1 WHO grade 1 (benign)

The most common meningiomas are classified into nine subgroups (Table 1). WHO grade 1 meningiomas generally have a good clinical course and a low risk of recurrence [20]. They rarely present with a histopathologic feature characterized by the presence of mitotic figures with pleomorphic nuclei [20, 25, 26].

<table>
<thead>
<tr>
<th>WHO grade 1</th>
<th>WHO grade 2</th>
<th>WHO grade 3</th>
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<tbody>
<tr>
<td>Meningothelial</td>
<td>Chordoid</td>
<td>Papillary</td>
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<td>Fibrous</td>
<td>Clear cell</td>
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<td>Metaplastic</td>
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Table 1. WHO classification of meningiomas.
3.1.2 WHO grade 2 (atypical)

WHO grade II meningiomas, referred to as the atypical group, have increased mitotic activity and a recurrence rate of up to 40% in 5 years [20, 25, 26].

3.1.3 WHO grade 3 (anaplastic)

This group represents malignant tumors with a very high recurrence rate as well as high mortality and morbidity. The 5-year progression-free survival for anaplastic variants is only 10% [20, 25, 26].

As mentioned before, CNS meningiomas are also named after the regions from which they arise. There are several classifications on this subject, but one of the most important is the one established by Yaşargil in 1966 [27]. According to Yaşargil, they are divided into six main groups (Table 2) [27].

3.2 Meningiomas, according to their localization

3.2.1 Olfactory groove meningiomas

This meningioma develops from arachnoid cap cells around the cribiform plate and crista galli [28, 29]. They may occur unilaterally or bilaterally [29]. Olfactory

<table>
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<th>Table 2.</th>
<th>Yaşargil meningioma classification.</th>
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<td>A. Basal meningiomas</td>
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<td>1. Median</td>
<td>2. Paramedian</td>
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<td>Olfactory groove</td>
<td>Orbital ceiling</td>
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<td>Tuberculum sella</td>
<td>Inner sphenoid wing</td>
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<td>Dorsum sella</td>
<td>Infracavernous</td>
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<td>Clivus</td>
<td>Cavum Meckel</td>
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<td>Foramen magnum</td>
<td>Cerebellopontine</td>
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<td>B. Fissural meningiomas</td>
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<td>Falcin</td>
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<td>Tentorial</td>
<td>Sphenoidal</td>
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<td>C. Dorsal meningiomas</td>
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<td>1. Supratentorial</td>
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<td>A. Parasagittal</td>
<td>B. Paramedian</td>
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<td>Temporal</td>
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<td>D. Intraventricular</td>
<td>E. Orbital</td>
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<td>Lateral ventricles</td>
<td>Foraminal</td>
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<tr>
<td>Third ventricle</td>
<td>Canalicular</td>
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<tr>
<td>Fourth ventricle</td>
<td>Infraorbital</td>
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groove meningiomas generally cause compression of the olfactory nerve and grow into the hemispheres [29]. Larger ones grow in the epidural plane and invade the ethmoid and sphenoid sinuses [29]. The most common findings in these cases are headache, personality changes, epilepsy, memory visual, and olfactory disturbances [28–30]. The arterial supply is usually through the ethmoid, meningeal, and ophthalmic arteries [29]. For this reason, central enucleation of the tumor should be performed after occluding the dural arterial supplies during surgery [27]. When removing the tumor, attention should be paid to the optic nerve and the anterior communicating arterial complex [27]. The operation is performed in two ways. One is the unilateral pterional transsylvian approach, and the other is bifrontal craniotomy [29, 31]. In the bifrontal craniotomy approach, less frontal lobe retraction and direct tumor intervention are easily possible, but more scar tissue is formed [29, 31]. With the pterional approach, although it provides relatively minor surgery, frontal lobe resection may sometimes be required [27, 30, 32]. To prevent CSF leakage from the cribriform plate after surgery, it should be covered with a pericranial flap, and fibrin glue should be used if necessary [27]. Postoperative CSF fistula may still occur [31]. It is recommended that lumbar drainage be attempted first, and if this attempt fails, radical dural repair is recommended [31].

3.2.2 Tuberculum sella meningiomas

These meningiomas arise from the tuberculum sellae, limbus sphenoidale, chiasmatic sulcus, and planum sphenoidale [33]. They account for 3% of all intracranial meningiomas [33]. Tuberculum sellae meningiomas most commonly originate from the optic nerves and chiasm [33]. Tuberculum sella meningiomas push the optic nerves upward and sideways [33]. The tumor grows posteriorly and superiorly, compressing the anterior cerebral artery and the anterior communicating artery complex [33]. Again, because of its growth, the tumor may invade the optic canal and frontobasal interhemispheric fissure [33]. When they reach a larger volume, they can compress the hypothalamus and cause an upward displacement of the third ventricle [33]. Very rarely, they can cause carotid artery dislocation [33]. In their clinic, they most commonly cause asymmetric vision loss [27]. In such cases, unilateral optic atrophy is noted [27]. This situation is in itself an indication of surgery [27]. Depending on the size and direction of the tumor, subfrontal, unilateral, supraorbital, or pterional transsylvian surgery may be performed [27, 33–36]. In the postoperative period, a 42–64% improvement in vision is observed [27, 30, 37, 38]. Vision impairment is observed in 10–20% of cases [27, 30, 37, 38]. The duration of visual impairment before surgery, tumor size, extent of visual loss, and advanced optic atrophy are key factors in postoperative recovery [9, 30, 37, 38]. In recent series, recurrence rates are less than 3% [39–44].

3.2.3 Sphenoid wing meningiomas

According to Al-Mefty, meningiomas of the sphenoid and parasellar regions are classified into five regions, including:

1. optic nerve, orbit, anterior visual pathways.
2. tuberculum sella.
3. clinoidal.
4. cavernous sinus

5. middle and outer wing meningiomas [33].

All of these have been mentioned in the classification of Yaşargil later on.

3.2.4 Clinoidal meningiomas

They are also meningiomas of the internal or medial sphenoid wing and arise from the anterior sphenoid process and the periphery of the lesser sphenoid wing [45]. As tumors grow, they compress the optic nerve, internal carotid artery, and branches, causing displacement [45]. Sometimes they can encircle these entities. Al-Mefty divided this group of meningiomas into three groups [45].

Group 1: These tumors originate on the underside of the anterior clinoid process and mainly involve the adventitia of the carotid artery [45]. For this reason, it may not be possible to distinguish this group of tumors from the branches of the carotid and middle cerebral arteries [45].

Group 2: These tumors originate at the superior or lateral projection of the anterior clinoid process [45]. The arachnoid of the carotid cistern separates it from the tumor adventitia [45]. Therefore, it is easy to separate the tumor from the carotid artery [45].

Group 3: The origin of these tumors is the optic foramen, so it grows toward the tip of the optic canal and anterior clinoid process [45]. These are relatively small tumors [46]. They are more easily scraped from the carotid arteries [45]. Clinically, unilateral optic atrophy is the most important finding [46–48]. In some cases, papilledema (Foster-Kennedy syndrome) may be observed on the opposite side of the eye [28, 49]. Depending on the size and orientation of the tumor, mental changes, hemiparesis, anosmia, and epileptic seizures may be observed [46–48]. It is more common in women and may enlarge during pregnancy [49]. The headache often manifests as orbital pain [49].

Pterional craniotomy is often preferred in treating this tumor group [50]. With advancing surgical techniques, vascular structures can be scraped microsurgically, and cranial nerves can be preserved safely, as with cavernous intrasinusal spread [50–52]. Despite all these options, the surgical cure of meningiomas of the middle sphenoid wing is still problematic [53, 54]. Radical resection is difficult despite all the developments [27, 31, 36, 45, 53, 54]. In Al-Mefty’s series, it was reported that complete resection was not possible in group 1 cases, while group 2 and 3 cases could be treated without problems [45].

3.2.5 Lateral and middle sphenoid wing meningiomas

Rosal et al. divided this group of meningiomas into seven groups by extending the classification of Brotchi and Bonnal (Figure 1) [53].

Brotchi and Bonnal classification modified by Rosal et al.: Group 1: Medial sphenoid wing no cavernous sinus infiltration; Group 2: Medial sphenoid wing with cavernous sinus infiltration; Group 3: Middle sphenoid wing; Group 4: Lateral sphenoid wing; Group 5: En-plaque no cavernous sinus infiltration (first third and fourth areas combined); Group 6: En-plaque with cavernous sinus infiltration (first second third and fourth areas combined); Group 7: Pure intraosseous tumor infiltration.

They form clinics according to their location. While the second group tends to cause exophthalmos, the third and fourth groups cause hemiparesis and epilepsy [53]. In the
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In the fifth group, there is localized and prolonged pain due to the spread of the plaques [53]. This sometimes occurs with the orbit and sphenoid bone invasion without mass effect [53]. In the case of invasion of the cavernous sinus, paralysis of the third, fourth, and sixth nerves may be observed [53]. When meningiomas in either group grow into the Sylvian fissure, they may encircle the middle cerebral artery [53]. In this case, differentiation from important structures is difficult. It is necessary to leave some tumor tissue [27]. Bone resection may be required to achieve a complete cure, especially because of bone invasion in groups 5, 6, and 7 [27]. The most appropriate surgical approach is the pterional approach [27]. The lateral sphenoid wing should be straightened to obtain an optimal field of view of this area [27].

3.2.6 Cavernous sinus meningiomas

While cavernous meningiomas can arise outside the cavernous sinus and invade it, they can also arise primarily from the cavernous sinus and invade externally [54]. The cavernous sinus is frequently invaded by meningiomas located in the orbital apex, internal sphenoid wing, middle fossa, tentorium, and superior clival region [54]. The reverse is also true [54]. Cavernous meningiomas were classified into five groups by Sekhar [55]. This classification was based on the location of the tumor in the sinus and the condition of the cavernous portion of the internal carotid artery [55]. Clinical findings refer to 3, 4, first and second branches of 5, and 6 nerves [55]. Therefore, diplopia and ophthalmoplegia can be seen [55]. The disease generally progresses slowly and worsens over time [55]. Angiography should be performed in all cases [55]. Patients should undergo a balloon occlusion test to determine collateral circulation [55]. After this test, cases are classified into low, intermediate, and high risk [55]. Because of the high likelihood of potential morbidities, adequate information and appropriate patient selection are important [55]. The main indication of surgery is a progressive deterioration of neurologic findings and evidence of radiographic growth [55]. While the indication for surgery is straightforward in large tumors and young
patients, the decision to operate should be cautioned in elderly and high-risk patients with balloon occlusion tests [55]. Parkinson first performed surgery in 1965 [55]. Many authors have reported new surgical approaches and techniques [51, 52, 55–57]. Frontotemporal craniotomy and orbitozygomatic osteotomy are mostly used in surgery [51, 52, 55–57]. If the tumor infiltrates Meckel’s cavity, tumor dissection should be performed [50, 56, 57].

3.2.7 Foramen magnum meningiomas

They are studied in two groups. The craniospinal ones arise from the basal groove in the lower part of the 1/3-clivus and extend from anteriorly and anterolaterally of the medulla to the foramen magnum [58]. The spino cranial, on the other hand, begins in the upper cervical region and extends upward from the posterior and postero-lateral aspect of the medulla to the cerebellomedullary cistern [58]. The most common finding is neck pain that is unilateral and occurs primarily with coughing, Lhermitte’s phenomenon, cold dysesthesia due to the eleventh nerve compression, progressive sensorineural and motor deficits that begin in one arm and spread to the other extremities, and atrophy of intrinsic limb muscles [58–60]. Lower cranial nerve palsies, Horner syndrome, respiratory problems, sphincter disturbances, nystagmus, and papilledema are observed less frequently [58–60]. To reach foramen magnum meningiomas, there are four main entry sites [44, 61–67]. These are posterior, postero-lateral, anterior, and transcervical approaches [44, 61–67]. The most commonly used approaches are posterior ones and among them, transcondylar and inferior suboccipital approaches are widespread [44, 61–67]. Most lesions can be resected simply with the inferior suboccipital approach [44, 61–67]. The most important factor to complicate the approach is the venous plexus [44, 61–67]. For those originating from the anterior aspect of foramen magnum, the postero-lateral (far lateral) approach is beneficial, especially when the tumor is large, opening a corridor to the anterior aspect of the brain stem and upper spinal cord [44, 61–67]. The transcondylar approach is associated with a higher morbidity rate than the far lateral approach [44, 61–67]. But it gives a wider viewing angle and accesses hard-to-reach areas [44, 61–67]. It should be applied if the benefit of the transcondylar approach is greater when weighed against the risks associated with CN XI dissection, VA transposition, and condyle drilling [44, 61–67].

3.2.8 Cerebellopontine angle meningiomas

Cerebellopontine angle meningiomas, which arise from the dorsal part of the petrous bone, are divided into two parts according to their location [67, 68]. These are antero-medial angle meningiomas and postero-lateral angle meningiomas [67, 68]. While the first group originates from the anteromedial side of the internal meatus acusticus, the tumors of the second group originate from the postero-lateral side [68]. These tumors spread toward the jugular foramen and hypoglossal foramen [68]. These can cause compression of the cerebellar hemispheres and pons, which results in their displacement [68]. Cerebellopontine angle meningiomas may cause erosions in the petrous bone [68]. About half of all meningiomas of the posterior fossa consist of cerebellopontine angle meningiomas [67]. While their incidence peaks in the fifth decade cerebellopontine angle meningiomas are 2–4 times more common in women than men [68]. Symptoms it causes include hearing loss, tinnitus, vertigo, headache, trigeminal neuralgia, long-track findings, and increased cerebral pressure (ICP) [68].
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Meningiomas of this region invade the fifth and seventh nerves more frequently than acoustic neuromas [68]. Lower cranial nerve findings are also seen in large lesions [68]. It is difficult to differentiate meningiomas in this region from acoustic neuromas [42, 68]. In the differential diagnosis, subarachnoid hemorrhage, which Yaşargil noted at a rate of 6%, may be a clue to angle meningiomas [67–70]. Another distinguishing feature for meningiomas could be the dural tail [68–70]. Angiographic staining is common in meningiomas but not in acoustic neuromas [68–70]. While intracanalicular tumors are rare in meningiomas, they are always found in acoustic neuromas [68–70]. As well as there is no hyperostosis in acoustic neuromas, it is found in meningiomas [68–70]. Additionally, the internal auditory canal is enlarged in acoustic neuromas, it is normal in meningiomas [68–70]. Despite these differences, diagnosing is not always easy [68–70]. While the facial nerve is anterior or anterosuperior to the tumor in acoustic neuromas, it can be located anywhere in the tumor in meningiomas [68–70]. For this reason, it is relatively easy to preserve hearing in meningiomas, but this is often impossible in acoustic neuromas [69, 70]. To reach these tumors, petrosal procedures in the anteromedial location of the tumor and retrosigmoid suboccipital procedures in the cerebellopontine angle location are used [70]. The surgeon's choice of surgical approach mainly depends on the characteristics of the tumor and the surgeon's personal preferences and the patient's clinic [70]. Most neurosurgeons prefer a single approach, while others use combinations. However, there are some points that need to be mentioned [70]. Petroal procedures should be performed in patients with hearing loss because hearing preservation is not possible with this technique [70]. Retrosigmoid suboccipital procedures can be used in lesions that have serviceable hearing and can be resected posteriorly [70]. However, in the retrosigmoid approach, it is relatively more difficult to protect the adjacent cranial nerves [70].

3.2.9 Petroclival meningiomas

Petroclival meningiomas arise from the upper part of the ⅔ clivus and petrous region medial to the fifth nerve [71]. The brainstem and basilar artery complex are typically pushed to the opposite side [71]. Clival meningiomas originate in the superior part of the clivus ⅔ and midline [71]. They cause backward displacement of the brainstem and basilar artery complex [72–74]. Another group is the sphenopetrosclival meningiomas defined by Yaşargil [71]. This type occurs when Meckel's cave is invaded by petroclival meningiomas [71]. In addition to the features of petroclival meningiomas, they invade the lateral wall of the cavernous sinus [71–75]. In the typical clinic of these tumors, headache and ataxia are observed with a frequency of 70%, while spastic paraparesis and somatosensory deficits are less common [71–75]. In these tumors, the fifth and eighth nerves are involved in ⅔ of the cases, the seventh nerve in half of the cases, and inferior cranial nerves in ⅓ of the cases [67, 72, 76]. Commonly used approaches for resection of these tumors are petrosal procedures, mid-fossa base procedures, or extended petrosal procedures that combine these two procedures [72, 76, 77].

3.2.10 Falx meningiomas

They can originate from any part of the falx [78]. Depending on their location, they are examined in three sections called anterior, middle, and posterior [78]. Anterior falx meningiomas are found in the part of the falx between the crista galli and the coronal suture [78]. Meningiomas of this region show an insidious
course clinically and become noticeable only when they have reached a large size [78]. Common findings include headaches, visual disturbances, personality changes, and dementia [78]. Seizures are observed less frequently than in other regions [78].

Meningiomas of the middle falx arise in the falx region between the coronal suture and the lambdoid suture [78]. This region’s most common clinical finding is focal motor or Jacksonian spasms [78]. Similarly, motor deficits may be seen [78].

Posterior falx meningiomas are meningiomas consisting of the falx portion between the lambdoid suture and the torcula [78]. They most commonly present with headaches [78]. Visual hallucinations and homonymous hemianopsia may occur in these patients [78].

For the resection of these tumors, various approaches can be used depending on the site of onset [27, 78].

3.2.11 Parasagittal meningiomas

These tumors are meningiomas that infiltrate the sagittal sinus, surrounding convex dura, and falx [79]. Bone involvement may also occur. According to the classification system of Sindou and Alvernia, sinus invasion is studied in six types (Figure 2)[79].

Type I: Lesion attachment to the outer surface of the sinus wall.
Type II: Tumor fragment inside the lateral recess.
Type III: Invasion of the ipsilateral wall.
Type IV: Invasion of the lateral wall and roof.
Types V and VI: Complete sinus occlusion with or without one wall free, respectively [79].

As with falx meningiomas, they are studied as anterior, middle, and posterior. Again, the symptoms and findings are the same as in Falx tumors [78, 79].

The most important question in surgical treatment is the condition of the sinuses [27, 78, 79]. Anterior meningiomas can be resected even if the sinus is open, but excision cannot be performed in intermediate and posterior tumors without complete closure of the sinus [27, 78, 79]. In such cases, subtotal resection (STR) is performed [27, 78, 79].

Surgical intervention for these lesions can be performed in various ways, depending on the location of the tumor [27, 78, 79]. All surgical methods used in other meningioma lesions can be used singly or in combination [27, 78, 79].

![Figure 2](image_url)

**Figure 2.** Sindou and Alvernia classification.
3.2.12 Convexity meningiomas

Convexity meningiomas are meningiomas that are not associated with the dura of the skull base and do not invade the dural venous sinuses [80, 81]. They account for 15% of all meningiomas [80, 81]. They have been classified into precoronal, coronal, postcoronal, paracentral, parietal, occipital, and temporal subgroups by Cushing and Eisenhardt [28]. Clinically, the main findings are headache, mental symptoms, visual disturbances, and epilepsy [80, 81]. The surgical approach differs depending on the origin of the lesion [35, 82, 83]. Complete excision is simple compared with other groups, and mortality is negligible [35, 82, 83].

3.2.13 Tentorial meningiomas

Tentorial meningiomas are divided into three groups as medial, lateral, and falciotentorial meningiomas [84]. Tentorial meningiomas grow below or above the tentorium [84]. In most cases, the tumor grows infratentorial and typically causes headaches and ataxia [84].

The subtemporal approach is recommended for medial and lateral tentorial meningiomas [84]. An interhemispheric or supratentorial approach is recommended for posterior tentorial and falciotentorial meningiomas, and a combined supratentorial approach is recommended for the infratentorial portion of lateral tentorial meningiomas [27, 84].

3.2.14 Intraventricular meningiomas

Intraventricular meningiomas account for 5% of all meningiomas [85]. They originate from the tela choroidea or the choroid plexus, and 80% are in the lateral ventricle, 15% in the third ventricle, and 5% in the fourth ventricle [85]. Most of the meningiomas in the lateral ventricle are located in the trigone region [85]. Headache, vomiting, speech disorders, homonymous hemianopsia, and sensorimotor hemiparesis are observed in meningiomas at the trigonal region [85]. Papilledema, vomiting, and hypothalamic disturbances are common in third ventricular meningiomas [85]. Obstructive hydrocephalus findings occur in the fourth ventricle meningiomas [85]. Intraventricular meningiomas are supplied by the choroidal arteries and their venous drainage is through the ependymal veins [85]. They are often of the fibroblastic type [85]. Since 90% of intraventricular meningiomas are WHO grade I, the prognosis is favorable [85]. Surgery is considered curative if gross total resection is possible [85]. However, 10% of patients have intraventricular meningiomas WHO grades II and III and may require additional radiosurgery [85]. According to the WHO grading for all types of meningiomas, survival rates are valid for intraventricular meningiomas, but the recurrence and mortality rates are lower due to their generally lower grade and a better rate of complete surgical resection [85].

Transcortical interventions (middle frontal gyrus, posterior-middle temporal gyrus, superior parieto-occipital fissure) and interhemispheric transcallosal approaches are used in the surgery of meningiomas located in the lateral ventricle [44]. While the interforniceal approach is preferred for meningiomas in the third ventricle, the suboccipital route is preferred for meningiomas in the fourth ventricle [44].
3.2.15 Orbital meningiomas

Meningiomas in orbit are divided into primary and secondary meningiomas [49]. Whereas primary orbital meningiomas arise from the optic nerve sheath and are located throughout the orbit, secondary tumors arise from the dura around the orbit and grow into the orbit [49]. Orbital meningiomas account for 9% of all orbital tumors [49]. They commonly occur in childhood [49]. Loss of vision is the most important symptom [49]. In addition, optic disk changes, visual field loss, proptosis, and pain may also occur [49]. The recurrence rate ranges from about 17–42% [86]. The recurrence rate is lower in patients receiving postoperative radiation therapy, depending on the WHO grade [86]. Surgical interventions include transorbital and transcranial procedures [86].

3.2.16 Calvarial meningiomas

This group is a rare tumor that arises from the calvarium [27]. They do not have intradural components [27]. Cases have been reported in the scalp, temporal bone, jugular foramen, orbit, paranasal sinuses, infratemporal fossa, and parotid gland [27]. They are more common in childhood and among the elderly [27].

4. Etiology

Compared with malignant glial tumors, there are fewer studies on the etiologic risk factors for meningiomas. Although the exact etiology of meningiomas is still unknown, some recognized risk factors are present.

4.1 Molecular etiology (genetic)

Although meningiomas have benign pathophysiology, they are thought to arise from clonal growth from a single cell, which is a characteristic of carcinomas [87, 88]. Sporadic meningiomas are generally associated with one or more focal chromosomal deletion(s) [87, 88]. In contrast, atypical and malignant meningiomas usually have more than one chromosomal replica number change [89]. It is now known that the complexity of genetic abnormalities also leads to an increase in tumor grade in meningiomas [90]. The most common genetic disorder associated with an increased risk of meningiomas is NF2 [91]. These patients are more likely to develop second- and third-grade meningiomas or multiple meningiomas [91]. Gorlin, von Hippel-Lindau, Li-Fraumeni, multiple endocrine neoplasia (MEN), and Cowden disease are also syndromes that predispose to the development of meningiomas [92].

4.2 Ionizing radiation

Exposure to ionizing radiation is the most important risk factor for developing meningiomas [92, 93]. It has been found that there is an increased risk for the development of meningiomas when ionizing radiation is used in the context of indications for the treatment of various diseases (e.g., cranial irradiation for tinea capitis, dental radiography) [93]. This risk is increased not only in patients who have been exposed to ionizing radiation for treatment but also in people who have been exposed to the effects of the atomic bomb [93]. Ionizing radiation is a risk factor for the
predisposition of meningiomas, with a six- to tenfold relative risk after a delay and without a dose relationship [93]. Based on this risk, ionizing radiation of radiographic examinations was recalculated and reduced [93].

4.3 Hormone

Because of the high incidence of meningiomas in women of reproductive age and women with breast cancer as well as the changes in meningioma size found in studies during pregnancy, the menstrual cycle, and menopause, it has been suggested that the increased risk for meningiomas may be related to hormones [94]. However, no association has been found between the use of oral contraceptives and the development of meningiomas [95, 96]. In addition, some other studies find no association between meningioma development and hormonal factors [95, 96].

Among the etiologically explained risk factors, head trauma, the presence of breast cancer, smoking, and cell phone use are mentioned. However, the causal relationship between these factors and meningioma development has not been established. For this reason, future studies will clarify these issues and uncover new developmental/acquired etiologic factors.

5. Diagnosis

These dural-based tumors are routinely discovered incidentally by neurologists, neurosurgeons, and other clinicians because of the wider use of computed tomography (CT) and magnetic resonance imaging (MRI) [97]. Nowadays, radiologic imaging with contrast-enhanced cranial CT or MRI provides very useful information not only in the diagnosis of meningiomas but also in monitoring asymptomatic cases, deciding on surgical/systemic treatment, and distinguishing between tumor recurrence and radiologic changes [97]. Histopathologic analysis by biopsy or resection is required for definitive diagnosis. However, thanks to evolving neuroradiology, MRI results have become the standard method for radiologic diagnosis and follow-up of meningiomas [97]. Contrast-enhanced CT can be performed in patients whose MRI is contraindicated for patient-related reasons (e.g., pacemakers, in-body metallic implants, claustrophobia) [97]. In addition, CT is superior to MRI in radiologic diagnosis by revealing the chronic effects of the tumor such as intra-tumoral calcification (25% of cases seen) and changes in the bone structure such as hyperostosis and interosseous bone growth [97]. However, the simultaneous use of both diagnostic tools provides more detailed information before and after surgery in most cases [97]. When imaging findings suggest meningioma, a biopsy is not required in these patients [97].

Benign meningiomas are usually isointense or mildly hypointense on T1-weighted brain imaging and hyperintense on T2-weighted/FLAIR sequences of MRI [97]. They have a characteristic thickened, contrast-enhancing dural tail (60%), and contain a CSF cleft [97]. Additionally, they have clearly defined margins and homogeneous enhancement (95%) [97]. Meningiomas displace the brain away from the overlying dura [97]. The dural tail is a useful radiologic finding at diagnosis to distinguish meningiomas from other lesions such as schwannomas [97]. However, the dural tail is not pathognomonic for meningiomas and may also be seen in metastases or hemangiopericytomas [97]. Again, it should be remembered that approximately 10–15% of meningiomas may have an atypical appearance on MRI images that mimics metastases or malignant gliomas [98]. Central necrosis, which is specific for
malignant gliomas (hypointense, non-enhancing central necrotic area in the lesion at the T1-weighted images), can interestingly be found in both benign and malignant variants of meningiomas [98]. A cystic appearance is a rare radiologic finding for this tumor [98]. Although uncertain, peritumoral edema can be seen on T2-weighted and FLAIR images [98]. Peritumoral edema is attributed to more aggressive meningiomas invading the brain [98]. In particular, significant peritumoral edema may be present in secretory meningiomas [98].

MRI spectroscopy (MRIs) can be used for differential diagnosis of meningiomas [99]. This modality can be particularly beneficial in patients who cannot undergo surgery [99]. Compared with normal brain tissue, MRIs usually reveal decreased N-acetyl-aspartate and creatinine peaks and increased choline and alanine peaks. In contrast, atypical meningiomas may have an increased lactate peak caused by necrotic tumor tissue [99, 100]. Buhl et al. reported a characteristic lactate peak in more than 63% of patients with atypical meningiomas on preoperative MRIs [100].

Depending on radiology, WHO grading degrees may be suspected [101]. However, there are currently no imaging criteria for preoperative differentiation of the various WHO grades of intracranial meningiomas [101]. Therefore, there is still uncertainty about which patients should be followed up or operated on early [102]. Although it is not possible today to determine the variants of meningiomas radiologically, invasion of the adjacent brain parenchyma and bone tissue in its location, heterogeneous contrast enhancement, intense peritumoral edema, seen in T2-weighted and FLAIR sequences, and central necrosis seen as hypointense in the T1-weighted sequence (non-enhancing tumor area) are also considered indicative of high-grade meningiomas [102, 103]. In addition, meningiomas with calcifications on cranial CT (hyperdense) and T2-weighted MRI imaging (hypointense) have been associated with a slower growth rate [104, 105].

Recently, the role of radiomics has been investigated in meningiomas. Radiomics consists of the correlation of quantitative radiological features with pathological and molecular features of the tumor [106]. This novel method has the potential to increase knowledge of the tumor, which is beneficial given the tumor’s hard-to-access location [106]. Several studies showed a potential role of radiomics in predicting the pathological grade and subtypes of meningiomas [106–109]. However, it is not currently in standard clinical use.

Because meningiomas easily invade the cerebral veins and cerebral venous sinuses, the MRI venogram is useful to visualize the relationship of the tumor to the lateral or superior sagittal sinus (direct invasion or compression) to determine the degree of tumor invasion and to reveal collateral venous outflow [107].

Conventional angiography no longer has a place in diagnosing meningiomas [110]. However, this technique can be used for intravascular embolization or to clarify the diagnosis when the appearance on CT or MRI remains unclear [110]. Angiographic findings suggest that meningioma includes dural arteries supplying the central tumor and pial arteries supplying the tumor periphery and bilateral vasculature [110].

Although positron emission tomography (PET) is not routinely used in clinical practice, it can be useful for meningiomas at the skull base, which are often difficult to detect with standard imaging modalities CT and MRI [111, 112]. Furthermore, PET(68-Ga-DOTATATE) can aid in the diagnosis during follow-up of recurrent meningiomas in cases where biopsy specimens cannot be obtained easily or undecided ones [111, 112].
6. Clinic

We have briefly mentioned above the clinical appearance caused by meningioma subgroups specifically. In general, clinical findings in meningiomas result from the tumor tissue compressing adjacent neural, vascular structures or occluding CSF flow pathways, cortical veins, and venous sinuses, depending on the compartment in which meningioma originated [113, 114]. Symptoms and signs of ICP such as papilledema, headache, nausea, and vomiting can occur not only in anterior skull base meningiomas that reach giant sizes (6 cm in diameter) but also in small tumors that cause severe reactive vasogenic edema [113, 114]. Although not common, they may present with clinical signs of transient ischemic attack or intracranial hemorrhage [113, 114]. Usually, meningiomas commonly cause peritumoral edema and epileptic seizures episodes (27–67%), which are thought to be site-dependent and may be partial (37%), complex partial (8%), generalized (60%), or a combination thereof [115, 116].

7. Treatment

The treatment of meningiomas varies widely and depends on patient-related factors such as age, performance status, concomitant medical conditions, and the targeted treatment modality (observation, symptomatic treatment, surgical treatment). Currently, the main treatment modality is observation with intermittent radiologic imaging for asymptomatic meningiomas, whereas complete surgical resection is sought for meningiomas that progress or cause symptoms (Table 3) [117].

7.1 Observation

Because the tumor growth rate for asymptomatic intracranial meningiomas is 2–4 mm/year, they can be treated conservatively. However, close surveillance is required clinically and radiologically, especially in young patients, because they can grow rapidly [117]. When a patient with a meningioma is planned for follow-up, the gold standard for it is intermittent MRI [117]. Contrast-enhanced T1-weighted sequences provide images suitable for evaluating volume increases in tumor mass [117]. In a study to determine tumor growth behavior in 64 patients with asymptomatic meningiomas, no patient had tumor-related symptoms in a 5-year follow-up [117]. During this 5-year follow-up period, 48 (75%) of the 64 patients experienced an increase in tumor size of 15% or more [117]. Therefore, serial imaging can follow asymptomatic meningiomas until permanent tumor growth is detected radiologically or symptoms develop [117]. However, even if an increase in tumor size is detected on serial volume measurements in the follow-up, the decision to proceed

| Grade 1: Complete tumor resection, including dura and ingrown bone |
|------------------|------------------|
| Grade 2: Gross total resection (dura coagulation is present) |
| Grade 3: Macroscopic resection (no dural excision or coagulation) |
| Grade 4: Subtotal resection |
| Grade 5: Biopsy only [121] |

Table 3. SIMPSON grade.
with surgery still depends on the patient’s age, symptomatology, and comorbidities [118, 119]. This is because the morbidity rate in surgically treated asymptomatic meningiomas is not negligible, especially in patients older than 70 [118, 119]. Therefore, the natural history of incidentally discovered tumors remains a concern for physicians and patients.

7.2 Surgery

Surgery is the primary treatment for meningiomas with volume increase on symptomatic or neuroradiologic follow-up [117–120]. The main goal of surgery is:

1. Preservation of existing neurological functions.
2. Elimination of neurological symptoms caused by the mass effect.
3. Prevention of recurrence with low morbidity (the recurrence rate is inversely proportional to the width of the resection); there should be maximal resection of the tumor, including all relevant dural and bony structures [120].

The basic principles of surgical treatment are central debulking and peripheral dissection, which facilitates resection in hard and calcified tumors, with good control of bleeding during surgery [27, 119]. Often, an arachnoid plane allowing reliable differentiation of the tumor from normal structures is discovered during surgery [27]. If this arachnoid plane is preserved, bleeding and injury to neurovascular structures are largely avoided [27].

As with other CNS tumors, the primary goal of surgical treatment for meningiomas is complete resection of the pathologic tissue [120]. However, several patient-related factors, such as tumor location, invasion of adjacent brain parenchyma, venous sinuses, encasement of arteries and cranial nerves by tumor tissue, or age and cardiovascular disease, may prevent complete resection from achieving good outcomes [27, 119, 120]. Therefore, complete resection is impossible without compromising functional outcomes for meningiomas in near-critical neurologic structures or surrounding neurovascular structures in some patients [27, 119, 120]. While resection for convexity meningiomas is relatively simple, resection for parasagittal tumors is more complicated because they often invade the sagittal sinus [120]. In cases where the tumor invades the sinus and venous flow persists, the portion of the tumor within the sinus should not be resected because of the risk of air embolism, hemorrhage, and acute sinus thrombosis [120]. Skull base meningiomas in the tuberculum sella, sphenoid wing, cerebellopontine angle, olfactory groove, or petroclival region require advanced surgical techniques [120].

Endoscopic endonasal procedures have been described that allow safe access without retraction of the parenchyma, especially for tumors located in the anterior midline region of the skull base [120]. Where necessary, an attempt should be made to achieve gross total resection (GTR) using all available modalities.

As with other intracranial tumors, it is possible to assess the success of the surgical resection with contrast-enhanced CT or MRI in the first 72 h after surgery [121]. In addition, neuro-radiologic imaging forms the basis for the Simpson grading system, which can predict recurrence after surgery [121]. According to Simpson’s criteria, the extent of resection is considered a factor for progression-free and disease-free survival [121]. As the grade increases from Simpson grade 1 to grade 5, recurrence rates also increase (Table 4) [121]. Another factor affecting recurrence rates is the
histopathologic grading of meningiomas [122, 123]. The 5-year recurrence rate after
total gross resection of WHO grade I meningiomas was 7–23%, whereas WHO grade
2 meningiomas were 50–55%, and WHO grade 3 was 72–78% [122, 123]. However, the
15-year recurrence rate in patients who underwent GTR for all types of meningiomas
was 24–60%, whereas this rate was over 70% in patients who underwent STR [124].
Other factors affecting survival include patient age and tumor location.

Preoperative embolization may be performed before surgery in very large or diffi-
cult-to-remove tumors with complex vascular feeding [125, 126]. However, because of
cardiovascular complications, preoperative embolization is not a routine procedure and
is not recommended in every case [125, 126]. In addition to shortening the operative

Table 4.
Overall evidence-based treatment algorithm.
time, preoperative embolization may be beneficial in cases where it is difficult to reach the feeding arteries, such as petroclival meningiomas [125, 126]. Therefore, the surgical team should evaluate the decision individually in each case [125, 126].

7.3 Endovascular treatment

The increase in interventional neuroradiologic applications and developments in microvascular catheters have also raised hopes for endovascular treatment of meningioma, which is a vascular tumor [127]. Some studies on this topic have found that the benefits of endovascular therapy are uncertain [128]. Therefore, significant obstacles remain to accepting endovascular intervention as a treatment modality. Selective microcatheter embolization of the meningeal arterial supply with various agents can be remarkably effective in the devascularization of the tumor, and preoperative embolization reduces perioperative bleeding [129]. However, there is still uncertainty about when preoperative embolization before resection is appropriate [129]. Furthermore, because atypical histopathologic features are more common in patients undergoing endovascular embolization, embolization could induce atypical histologic changes associated with benign (WHO 1) meningiomas [130, 131]. For the above reasons, endovascular embolization may be considered an alternative treatment option for managing meningiomas, but only for patients in whom surgical intervention is not feasible. It is not a stand-alone treatment modality.

7.4 Radiotherapy

Although radiation therapy has been used to treat tumors for many years, there are not as much clinical studies on treating meningiomas with radiation therapy as other pathologies. While radiotherapy (RT) is usually a secondary treatment modality to surgical resection to prevent higher-grade progression of meningiomas and reduce the recurrence rate, it may be considered a primary treatment option in a well-defined, inoperable small group of patients [132]. For this purpose, both fractionated external beam radiation therapy (EBRT) and stereotactic single-fraction radiation therapy (SRS) are used as adjuvant treatment tools [132]. SRS is increasingly used for lesions and may better protect the surrounding brain parenchyma from potential radiation toxicity [132]. Single-fraction SRS is usually limited to tumors <30 mm diameter and for meningiomas not directly adjacent to (or compressing) sensitive structures such as the hypothalamus [132]. Multifractional SRS can be used for bigger tumors [132]. Local control of meningiomas of a diameter of 3 cm or less after SRS was the effect of Simpson Grade I resection [133]. Two retrospective series found that a reduction of tumor size after SRS or EBRT provided tumor control after 5 and 10 years [134, 135]. The 10-year recurrence-free follow-up is 93.4% and 95.7%, respectively [135]. WHO grade II and III meningiomas are aggressive tumors [135]. The 1- and 4-year progression-free survival of these lesions after the first SRS is 92% and 31%, respectively [135]. Radiosurgery may be an important adjuvant and salvage therapy for lesions that will likely require more than one treatment [135]. Although discussions continue, combined treatment approaches that include surgery and fractionated RT are increasingly preferred.

7.5 Systemic treatments

There is no effective medical treatment for meningiomas because the beneficial effects of systemic agents have not been fully demonstrated in clinical trials.
Currently, conventional cytotoxic agents are not thought to have a beneficial effect on the tumor. Data about that subject are generally based on observational or retrospective data from a small group of patients rather than prospective studies.

Systemic therapy is currently an alternative treatment option for a small group of patients with recurrent/progressive diseases who cannot be treated with RT or for whom further surgical resection is not possible. Thus, systemic treatment is not the initial treatment for meningiomas but the final treatment step. These can be studied as follows.

7.5.1 Hormone therapy

Knowing that 70% of meningiomas have progesterone receptors, 60% have prolactin receptors, 30% have estrogen receptors, and female predominance suggests the growth of such tumors may be hormone-dependent [136–139]. Knowledge of the presence of hormone receptors has led to the idea that hormone antagonists can also be used in treating meningiomas. To this end, Koide et al. used mifepristone (progesterone antagonist) at 200 or 400 mg daily doses [140]. Although they reported improvement in 25% of the patients included in the study, they noted a decrease in tumor size in 35% of fourteen patients using the same hormone antagonist at similar doses [140]. Grunberg et al. used similar doses of mifepristone and had five out of 14 patients show a meaningful decrease in tumor size [141]. However, continuing studies with larger patient groups failed to achieve the cure mentioned above rates [140–142]. The trial with tamoxifen, an estrogen receptor modulator, conducted with 19 patients also failed to provide positive results [142].

7.5.2 Chemotherapy

Hydroxyurea, a drug commonly used in cancers, inhibits proliferation by inhibiting the S phase of replication [143, 144]. Swinnen et al. reported a decrease in tumor size in three of four patients in their study with hydroxyurea [143]. However, these responses could not be replicated in phase II clinical trials, and Chamberlain's study also failed to demonstrate efficacy [143, 144]. Currently, there is no routine clinical use.

7.5.3 Somatostatin analogs

Somatostatin receptors were found to be expressed in approximately 90% of meningiomas using single-photon emission computed tomography (SPECT) scanning [145]. Although 44% of 16 patients treated with a somatostatin agonist with a high affinity for somatostatin receptors (Sandostatin LAR) had positive results in terms of progression-free survival at 6 months, this agonist was found to have no effect in recurrent high-grade meningiomas [146]. In a study conducted with pasireotide, another agonist with higher receptor affinity than Sandostatin, no improvement in progression-free survival (PFS) at 6 months was observed [147]. The studies remain controversial, with no consensus. Currently, there is no routine clinical use.

7.5.4 Targeted agents

In recent studies, bevacizumab, an antibody against vascular endothelial growth factor (VEGF), has been shown to inhibit growth in meningiomas [148, 149]. Bevacizumab shows this effect possibly by blocking angiogenesis [148, 149]. Overall,
bevacizumab appears to be an effective therapeutic approach for patients with atypical and anaplastic meningiomas who have exhausted surgical and radiation therapy options [148, 149]. Also because of the programmed death receptor (PDL1) in solid organ tumors outside the CNS, immunotherapeutics such as nivolumab and ipilimumab are quite effective [150]. After Du et al. presented evidence of PDL1 receptors in meningiomas, the use of nivolumab in recurrent meningiomas and pembrolizumab in atypical and anaplastic meningiomas paved the way for promising clinical trials [151]. There is currently an intense debate on the subject [151]. There is still no opinion due to the studies concluded in both directions [151]. Currently, there is no routine clinical use.

8. Conclusion

Within the framework of the rules of the book, we have tried to write this section without going into unnecessary detail. We hope it is useful for the reader.

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Conflict of interest

The authors declare that they have no known competing financial interests or personal relationships between any person/persons or institution/institutions and the authors that could have appeared to influence the work reported in this paper.

Other declarations

Finally, we dedicate it to the great Türk nation, which sheds light on our future with its thousands of years of history.

Appendices and nomenclature

CNS central nervous system
CSF cerebrospinal fluid
CT computed tomography
EBRT external beam radiation therapy
GTR gross total resection
ICP increased cerebral pressure
MEN multiple endocrine neoplasia
MRI magnetic resonance imaging
MRIs magnetic resonance imaging spectroscopy
NF2 neurofibromatosis type 2
Meningiomas

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PET positron emission tomography
PDL1 programmed death receptor 1
RT radiotherapy
SPECT single-photon emission computed tomography
SRS stereotactic radiosurgery
STR subtotal resection
VEGF vascular endothelial growth factor
WHO World Health Organization

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