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REVIEW

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Humps and bumps of head: review of meningiomas of the scalp

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Abstract

Meningiomas are a type of tumor that arises from meningothelial cells and primarily develops in intracranial space, being some of the most common benign tumors of the central nervous system. However, meningiomas can rarely occur on the scalp and are called *primary cutaneous meningiomas*. Since the pathogenesis of these lesions is still uncertain, these tumors still pose challenges in terms of histopathological diagnosis. In this review, we will discuss the main cases of scalp meningiomas in the literature, their classification, pathological and immunohistochemical diagnosis, differential diagnosis with other scalp lesions and the most effective treatment. This study highlights the importance of immunohistochemistry in the differential diagnosis of skin lesions located on the scalp.

Keywords: type I cutaneous meningioma, scalp meningioma, primary cutaneous meningiomas, scalp lesions.

Introduction

Meningiomas are a type of tumor that arises from meningothelial cells [1]. They represent 37.6% of all primary tumors of the central nervous system (CNS) and 53.3% of all benign tumors of the CNS [2]. According to the *World Health Organization* (WHO), these tumors are categorized into three pathological grades, with grade 1 being the most benign and grade 3 being the most malignant, constituting the group of anaplastic (malignant) meningiomas. The incidence of atypical and malignant meningiomas is less than 10% [3, 4], and the incidence of grade 3 malignant meningioma is 1.7% among all meningiomas [2]. Both benign and malignant meningiomas can metastasize, with a probability of 0.1% [5]. Meningiomas can metastasize to distant organs like bone, spinal cord, lungs and liver, and the rate of distant metastasis of anaplastic meningiomas is 30% [6, 7]. Regarding subcutaneous metastases of intracranial meningiomas, the literature has reported several cases [6, 8–11].

In addition to these, another rare type of meningiomas is those that develop *de novo* on the skin of the head, face, or paraspinous regions, with no subcutaneous metastases of an intracranial meningioma [12]. Named *primary cutaneous meningiomas*, these lesions are much rarer than intracranial meningiomas, are slowly growing tumors, and usually have a benign nature [12]. However, primary cutaneous meningiomas pose problems in histopathological (HP) diagnosis, making it difficult [13], especially since the pathogenesis of these lesions is still uncertain [14].

Due to the rarity of these cutaneous meningiomas, the literature is mostly based on case reports [13–18]. The first cutaneous meningioma was reported in 1904 by Max Winkler and published in *Virchows Archiv für pathologische Anatomie und Physiologie und für klinische Medizin* [19]. Subsequently, a literature review conducted in 1974 by Lopez *et al.* reported that of the 140 cases of ectopic meningiomas, 64% were primary extracranial lesions and 36% were secondary extensions from underlying intracranial meningiomas [18].

Much like intracranial meningiomas, their *de novo* extracranial variants are slow-growing, well-circumscribed tumors, and the clinical presentation generally consists of the appearance of a mass on the scalp, accompanied or

not by alopecia [12] or overlying hair tufts [17]. The most common locations of cutaneous meningiomas are on the midline, in the occipital region of the scalp [18], but they can also occur in the orbit [20, 21], glabella [21, 22], nose [22], maxillary and frontal sinuses [22], or ear [18].

Several theories have been proposed to explain the development of cutaneous meningiomas, including: (*1*) head injuries which can lead to the displacement of superficial meningeal tissue and subsequent growth in the area where it was displaced, (*2*) the blockage of heterotopic rests of arachnoid between layers of soft tissue during embryonic development, (*3*) migration of arachnoid cells to the skin along cranial and spinal nerves, (*4*) persistence of an atretic encephalocele with intracranial extension, or (*5*) vascular turbulence that allowed the detachment and embolization of arachnoid islets [8, 23–26].

Classification of scalp meningiomas

Based on the pathological and clinical characteristics of cutaneous meningiomas that develop outside the CNS, Lopez *et al.* classified them into three types in 1974 [18]. This classification is still widely used today (Table 1) [8].

Table 1 – ***Characteristics of type I, II and III meningiomas according to Lopez's classification***

Features	Type I	Type II	Type III
Origins	Ectopic arachnoid cells (meningothelial cells)	Remnant of arachnoid cells extended along nerves	Extension from a meningioma that involves neuroaxis
Pathology	This focus was trapped outside the cranial cavity along in the dermis and subcutis, along lines of embryonic fusion	Ectopic soft tissue meningioma extending to the skin along nerves (in the distribution of the cranial and spinal nerves)	Primary meningiomas that secondarily involve the skin by direct extension through bone
Congenital type	Yes	No	No
Anatomical region	Scalp (along suture lines, especially occipital area), forehead, paravertebral regions	Around eyes, nose, mouth and ears	Anywhere on the skull (especially on the calvarium)
Associated intracranial meningioma	No	No	Yes
Neurological symptoms	No	May cause neurological symptoms	Yes
Age of diagnosis	Children and young adults that have present since birth	Develop later in life (adults)	More common in adults
Prognosis	Excellent	Good	Poor

Type I – primary cutaneous meningiomas

These tumors represent congenital meningiomas that occur on the face, scalp, or paravertebral region, especially in children and young people. Typically, these conditions are present from birth and typically follow a non-threatening course [27]. Regarding the occurrence of this type of cutaneous meningioma, there are currently two theories. According to the first theory, some authors believe that these tumors are present at birth and originate from an ectopic focus of arachnoid cells located outside the skull, because of abnormal cranial bone development [16], during embryogenesis [28]. Usually, the precursors are located primarily on the scalp and forehead skin [13]. In this case, meningothelial cells are ectopically located in the dermis or subcutaneous tissue [16, 29] (Figure 1, A and B).

This theory is the most widespread and widely accepted. Other authors who have formulated a second theory believe that meningiomas of skin are not real tumors but rather sequestered heterotopic meningeal nodules or meningoceles located in the skin [30], considering that they probably develop from a rudimentary meningocele that did not maintain connection with the CNS [13, 17, 27, 30, 31].

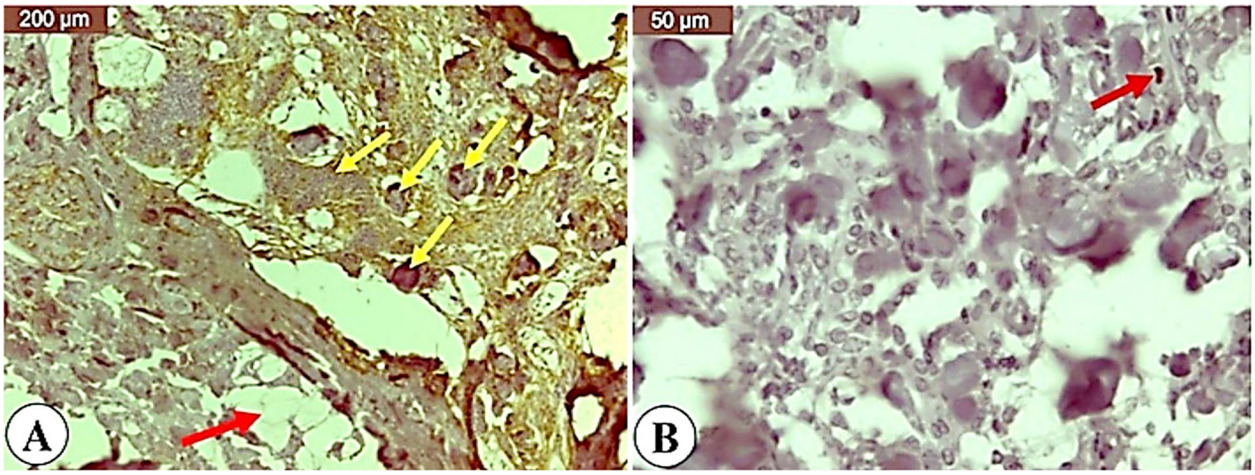

Figure 1 – ***(A) Photomicrograph showing in the lower left part the hypodermis with groups of adipocytes (red arrow), and in the upper right part the tumor is present, whose cells are immunopositively for EMA (yellow arrow), which demonstrates the meningothelial origin of the cell's tumors; (B) Photomicrograph showing a single tumor cell whose nucleus shows immunopositivity for Ki67, so the Ki67 labeling index was less than 1% (red arrow). Anti-EMA antibody immunomarking: (A) ×100. Anti-Ki67 antibody immunomarking: (B) ×400. EMA: Epithelial membrane antigen.***

Type II – meningiomas of soft tissue with extension into the skin

These lesions are ectopic soft tissue meningiomas that extend into the skin around the sensory organs [32]. They generally occur around the sensory organs of the head and can develop periorbitally, peribucally, perinasally, and periauricularly, or along cranial or spinal nerves. These tumors occur from ectopic arachnoid cells that extend to the skin by contiguity along the cranial nerves. Thus, the most frequent anatomical locations are the forehead, scalp, or angle of the mandible [16]. Like type I, type II cutaneous meningiomas are extracranial tumors and are not associated with intracranial meningiomas [16, 18, 33]. They appear *de novo* and represent a cutaneous extension of an ectopic soft tissue meningioma originating from an arachnoidal cell rests displaced along nerve sheaths [27]. Type II cutaneous meningiomas are more common in adults but can occur at any age. They usually grow slowly and become symptomatic when they increase in size [16].

Type III – CNS meningiomas with extension into the skin

These lesions represent the direct extension under the scalp [27] of an intracranial meningioma that infiltrates the skull through a bony defect (traumatic or surgical) or directly through local invasion of the bone [32]. These meningiomas are aggressive, and their treatment involves extensive surgery [16], primarily of the intracranial tumor. Sometimes, the extracranial component can also have significant dimensions, Morina *et al.* reporting the resection of an extracranial meningioma up to 45 cm in size, weighing 3.5 kg [34].

As this review is only about cutaneous meningiomas located on the scalp (type I cutaneous meningiomas), in the following, we will thoroughly address this topic.

Primary cutaneous meningiomas (type I cutaneous meningiomas)

The majority of scalp meningiomas are located in the occipital region or along the cranial sutures [16, 29]. Various authors have suggested that a rudimentary meningocele may represent the origin of some type I cutaneous meningiomas [17, 18], and furthermore, some patients have associated multiple congenital anomalies, such as mandibular asymmetry, abnormal segmentation of the vertebrae, cleft palate, lip, and nose [17], or even finger malformations, especially in neurofibromatosis type I [35].

The possibility of a genetic component may exist. In this regard, Tron *et al.* reported the familial occurrence of type I cutaneous meningiomas with autosomal dominant transmission [36]. In their study, they observed that these tumors were identified on the scalp of 11 of 24 family members over four generations [33, 36]. Additionally, other authors have reported the presence of cutaneous meningiomas in a pair of adult Japanese siblings [36].

Regarding imaging diagnosis, even though they are cutaneous lesions, authors still recommend magnetic resonance imaging (MRI) or contrast-enhanced computed tomography when MRI is contraindicated [37] to better define the cutaneous lesion and exclude the presence of an intracranial meningioma.

Histopathology

Regarding the histological variants of type I cutaneous meningiomas, most have been reported as pathological findings of meningothelial cells, with psammoma bodies grouped in nests (Figure 2, A and B).

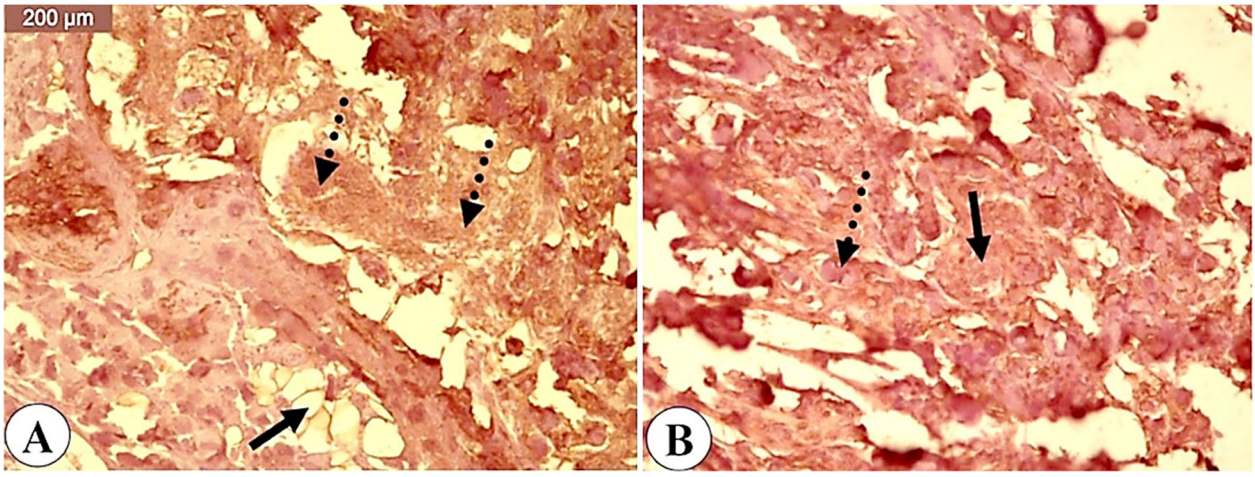

Figure 2 – ***(A) Photomicrograph showing in the lower left the hypodermis with groups of adipocytes (continuous black arrow), and in the upper right the tumor is present, whose cells are immunopositively for vimentin, which demonstrates that meningothelial origin of the tumor cells; (B) Photomicrograph showing the immunopositivity of the tumor cells for vimentin, also highlighting the "swirling" arrangement of the tumor cells (solid black arrow), as well as the arrangement of collagen in the form of pseudopsammomatous bodies (dotted black arrow). Anti-vimentin antibody immunomarking: (A and B) ×100.***

Other reported variants have been acellular meningeal hamartomas, which are less circumscribed, with foci of hyperplastic meningothelial cells and psammoma bodies [33]. A third variant is represented by rudimentary meningoceles due to the presence of a rudimentary cystic cavity or stalk with nests and strands of meningothelial cells [33]. Although most authors consider these lesions to be both histologically and immunohistochemically identical to their meningeal counterparts [13, 33, 38], some authors have identified some HP differences. Thus, although type II or III meningiomas have similar characteristics to type I meningiomas, they usually have less collagen, are more cellular and lobulated, and extend more into the dermis [33]. In contrast, well-developed collagen bodies are more typical for type I

cutaneous meningiomas [18]. Regarding hyperostosis, it has been reported in cases of type I cutaneous meningiomas [12, 39], not just in intracranial meningiomas with or without extracranial extension.

Differential diagnosis

Since clinical and imaging characteristics are often inconclusive, especially in types I and II, the pathological examination, and especially the immunohistochemical (IHC) examination, represent the most important tool in the diagnosis of these lesions.

The most frequent pathological form is the meningothelial histological subtype [40], with nests and sheets of polygonal or oval meningothelial cells, with collagen or psammoma bodies (*calcification foci*), characteristics that are helpful in pathological diagnosis [13]. Regarding the pathological characteristics for the three types in Lopez's classification (1974), type I (congenital) meningiomas tend to be centered in the subcutaneous fat and have collagenous stroma [40]. On the other hand, type II and III (acquired) lesions extend higher into the dermis, are more cellular, and more frequently associated with ulceration or atrophy of the epidermis. Furthermore, HP differentiation between type II and type III is not possible without neuroimaging, which provides information about the existence of intracranial meningioma [18, 40]. Regarding IHC characteristics, these are usually common, reported by various authors (Table 2). These characteristics are represented by positivity for vimentin and epithelial membrane antigen (EMA) (Figure 2, A and B), and negativity for desmin, S100 protein, cytokeratins (CKs), cluster of differentiation (CD)34, CD68, CD31, and smooth muscle actin [8, 13, 16, 27].

Cutaneous meningiomas located on the scalp have an insidious onset, and the clinical manifestations are those of a subcutaneous lesion. For this reason, the diagnosis is difficult, mostly based on IHC examination, and must differentiate cutaneous meningioma from other lesions, such as lipomas [18], sebaceous cysts [5], melanocytic nevi, embryonic cyst [13, 27], angiomas, benign fibrous histiocytomas, muscle tumors [13], dermoid cysts, lymphangiomas, deep *granuloma annulare*, calcified cephalhematomas, meningoceles with a fibrohistiocytic reaction [15, 41], verrucous hamartoma [42], adnexal tumor (dermal and subcutaneous epithelioid neoplasm of the scalp) [40] or cutaneous metastasis from tumor cell seeding during surgery for an intracranial meningioma.

Clinical examinations of the head and face play an important role in the early detection of various pathologies that can be localized at this level, including cutaneous meningioma. In some such cases, a multidisciplinary approach involving neurosurgeons, ophthalmologists, maxillofacial surgeons, dentists, dermatologists, and pathologists may even be considered.

To make a differential diagnosis of a cutaneous meningioma from another lesion, IHC stains are very helpful, especially EMA and vimentin, because positivity for these supports the diagnosis of meningioma [43]. CK staining is used to exclude epithelial lesions [27], which will be negative in the case of meningiomas. A lack of staining for Melan-A and S100 will exclude melanocytic lesions [27]. To exclude myogenic, histiocytic or vascular tumors, muscle markers actin and desmin can be used, as well as cell markers, such as CD34, CD31 and CD68 [13]. However, EMA can also be positive in adnexal tumors [40]. Furthermore, CK reactivity can be diffuse or focally positive in meningiomas, ranging from 24% in benign meningiomas to 75% in malignant meningiomas, and can also be diffusely positive in adnexal tumors [44, 45]. Thirdly, it has been demonstrated that p63 exhibits positivity in both cutaneous meningiomas and malignant cutaneous adnexal tumors [46–48]. Therefore, we recommend a diverse panel of IHC stains for the diagnosis of cutaneous meningioma and differentiation from other cutaneous lesions.

Association with other lesions

Several authors have reported an association between cutaneous meningiomas and other scalp lesions [49, 50]. Tong *et al.* reported the coexistence of cutaneous meningioma and steatocystoma [50]. They suggest that immunological events in the meningioma microenvironment could contribute to the appearance of another tumor, such as steatocystoma in their case [49]. Their explanation is based on studies in literature that support the presence of B- and T-cells, macrophages, and plasma cells in meningiomas, indicating substantial immune cell infiltrates [50–53]. In 2006, Zeikus *et al.* [14] also reported the coexistence of a cutaneous meningioma and a *sinus pericranii*, an uncommon vascular anomaly characterized by abnormal communication between intra- and extracranial venous systems.

Treatment

The "gold standard" for the treatment of these meningiomas is complete surgical resection [8, 54]. In some rare cases, when the patient's clinical condition does not allow for complete excision, a biopsy may be performed [16]. Type I meningiomas have a better prognosis than type II or III meningiomas [8], as type III meningiomas can be inoperable in some cases [8]. The prognosis of cutaneous meningiomas is largely determined by the Lopez lesion type. Type I has a favorable prognosis if are achieved clear surgical margins intraoperatively [55]. Furthermore, no tumor recurrences have been reported after the complete surgical resections [33]. On the other hand, type II and III have a poorer prognosis, especially if the lesions are large and involve vital vasculo-nervous structures [33, 56]. In such cases, targeted therapies have been considered, especially for non-surgical treatable meningiomas, particularly type III meningiomas [33]. These treatments encompass platelet-derived growth factor and vascular endothelial growth factor inhibitors [33] and are currently the subject of ongoing research [57] (Table 2).

Conclusions

Cutaneous meningiomas are rare tumors that can be challenging in terms of diagnosis. Accurate diagnosis requires a combination of clinical experience, imaging techniques, and pathological diagnosis. Of these, pathological diagnosis and especially IHC one plays a major role in the precise diagnosis of these lesions, and especially in their differential diagnosis with other scalp lesions. The most effective treatment remains surgical resection, ensuring complete tumor removal until clear margins are achieved. This study highlights once again the importance of immunohistochemistry in the differential diagnosis of cutaneous lesions located on the scalp.

Table 2 – ***The main cases of scalp meningiomas (type I) reported in the literature and their characteristics***

Authors	Year	Gender/ Age [years]	Region of scalp	Dimensions [mm]	Macroscopic features	Treatment	Recurrence	Histological grade (*WHO*)	IHC characteristics
Li & Qi [5]	2022	M/50	Forehead	42×41	Lobulated nodule Close to bone surface Irregular in shape (US, MRI)	Resection	No	Grade 3 meningioma (malignant meningioma)	Ki67 10% (+) EMA (+) PR (+) SMA (-)
Jaiswal *et al*. [56]	2018	F/7	Occipital region	10×10×0.8	Well-defined margins (skull X-ray)	Resection	Not specified	Grade 1 meningioma	EMA (+)
Kishore *et al*. [58]	2017	F/40	R parietal-occipital	35×30×3	Firm, fixed, nontender, non-fluctuant; bone underlying erosion (X-ray, CT)	Initial – biopsy Second – resection	Not specified	Not specified	EMA (+) Vimentin (+) S100 (+)
Konopinski *et al*. [41]	2017	F/30	L posterior scalp	28×18	Not specified	Resection	Not specified	Grade 2 meningioma	Ki67 21.6% (+) EMA (+) S100 (-) CD34 (-) SMA (-) STAT6 (-) CD31 (-) ERG (-) Desmin (-)
Brantsch *et al*. [27]	2009	F/33	R parietal	35	Large subcutaneous node (MRI)	Resection with periosteum Re-excision (three months later)	No	Grade 1 meningioma	Vimentin (+) EMA (+) S100 (-) Melan-A (-) CK (-)
Hussein & Abdelwahed [13]	2007	F/ newborn baby	L posterior occipital	30×20, protruding 20 mm above the scalp level	Well-circumscribed ovoid mass (CT, MRI)	Resection	No	Grade 1 meningioma	Vimentin (+) EMA (+) CKs (-) CD34 (-) CD31 (-) CD68 (-) Alpha-1 antitrypsin (-) SMA (-) Desmin (-) S100 (-)
Courville *et al*. [30]	2000	M/6	Vertex (midline)	10	(US, MRI)	Resection	Not specified	Grade 1 meningioma	Vimentin (+) EMA (+) S100 (-) NSE (-) GFAP (-) Actin (-) CK (KL1, MNF116) (-)
Ragoowansi *et al*. [16]	1998	M/77	L parietal	50×50	Well-localized (CT)	Biopsy	Not specified	Not specified	Vimentin (+) EMA (+) CAM 5.2 (-) LP34 (-) CEA (-) CD34 (-) Factor VIII-related antigen (-)
Theaker & Fleming [59]	1987	M/33	Vertex	20 (diameter)	Firm, non-tender (CT)	Resection	Not specified	Not specified	Vimentin (+) EMA (+) S100 (-) CK (-)
Serwatka & Mellette [12]	1984	M/45	Vertex (parietal-occipital area)	30×30	Firm, not hard, nonmobile subcutaneous tumor; important hyperostosis of the posterior parietal bones bilaterally (CT)	Initial – biopsy Second – resection (parietal craniectomy with cranioplasty)	Not specified	Not specified	S100 (-), weakly positive immunostaining

CD: Cluster of differentiation; CEA: Carcinoembryonic antigen; CK: Cytokeratin; CT: Computed tomography; EMA: Epithelial membrane antigen; F: Female; GFAP: Glial fibrillary acidic protein; IHC: Immunohistochemical; L: Left; M: Male; MRI: Magnetic resonance imaging; NSE: Neuron-specific enolase; PR: Progesterone receptor; R: Right; SMA: Smooth muscle actin; STAT6: Signal transducer and activator of transcription 6; US: Ultrasonography; WHO: *World Health Organization*.

Conflict of interests

The authors declare that they have no conflict of interests.

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