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Original Research Article

SURGICALLY TREATED MENINGIOMAS: A CENTRAL INDIA CASE SERIES WITH COMPREHENSIVE REVIEW OF LITERATURE

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ABSTRACT

Meningioma arise from the cap cell layer of the arachnoid. Their most common sites are in the front half of the head and include : lateral cerebral convexities , midline along the flax cerebri adjacent to the major venous sinuses parasagittaly , and olfactory groove. Less frequent sites are : within the cerebal ventricles , foramen magnum , cerebellopontine angle and the spinal cord. Meningiomas are generally solitary. They have an increased frequency in patients with neurofibromatosis 2 and are aften multiple in these cases.They are usually found in 2nd to 6th decades of life,with slight female preponderance. Most meningiomas are benign and can be removed successfully. Rarely , a malignant meningioma may metastasise , mainly to the lungs.

Keywords:. Meningioma ,Arachnoid, Neurofibromatosis, Benign, Metastasis

INTRODUCTION

Meningiomas exhibit distinct morphologic characteristics both grossly and microscopically. Grossly, meningiomas present as well-circumscribed, solid, spherical, or hemispherical masses of varying size, typically ranging from 1 to 10 cm in diameter. The tumor is usually firmly attached to the dura mater, causing indentation of the brain's surface, but it rarely invades the brain parenchyma. Often, the overlying bone demonstrates hyperostosis. Upon sectioning, the tumor appears firm and fibrous, with occasional foci of calcification.Microscopically, meningiomas are classified into five subtypes:

Meningotheliomatous (syncytial) meningioma: This subtype resembles the normal arachnoid cap cells, consisting of solid masses of polygonal cells with poorly-defined cell membranes, giving a syncytial appearance. The cells display round to oval central nuclei and abundant,

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE5, 2024

finely granular cytoplasm. Collagenous stroma is present, dividing the tumor into irregular lobules.

Fibrous (fibroblastic) meningioma: Less common, this subtype features spindle-shaped fibroblastic tumor cells arranged in parallel or interlacing bundles. Whorled patterns and psammoma bodies are less frequently observed in this type.

Transitional (mixed) meningioma: Characterized by a combination of cells with syncytial and fibroblastic features, this subtype exhibits conspicuous whorled patterns of tumor cells, often surrounding central capillary-sized blood vessels. Some of the whorls contain psammoma bodies due to calcification of the central core. Transitional meningiomas may also exhibit degenerative changes like xanthomatous and myxomatous degeneration.

It's important to note that these first three histological patterns represent a spectrum of lesions rather than distinct entities.

Angioblastic meningioma: This subtype encompasses two patterns: the haemangioblastic pattern, resembling haemangioblastoma of the cerebellum, and the haemangiopericytic pattern, indistinguishable from haemangiopericytoma found elsewhere in the body. Both types of angioblastic meningiomas have a high recurrence rate.

Anaplastic (malignant) meningioma: In rare cases, meningiomas may display features of anaplasia and invade the underlying brain or spinal cord. This pattern of meningioma is associated with extraneural metastases, mainly to the lungs.

Meningiomas can be distinguished from other primary intracranial neoplasms based on their location within the meninges (as shown in Tables 10.9, 10.10, and 10.12). Most meningiomas are identified on imaging as dural-based, extra-axial lesions, originating from clusters of meningothelial cells in the arachnoid. Common locations include parasagittal, falx cerebri, over the cerebral convexities, olfactory groove, sphenoid ridge, near the sella, foramen magnum, tentorium cerebelli, cerebellopontine angle, and spinal canal. Rarely, meningiomas may arise from meningothelial cell remnants in the choroid plexus (intraventricular meningioma) or within the CNS parenchyma itself. The macroscopic appearance of meningiomas is that of firm, solid masses[1,2,3,4].

MATERIAL AND METHODS

The study was carried out on thirty newly diagnosed cases of Meningioma reported in ujjain and indore in a private laboratory set up. The cases were processed into slides and the same were reviewed by 5 experienced pathologists from central india. Consensus diagnosis was reached and cases were classified into various subtypes of Meningioma.

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE5, 2024

RESULTS

Out of 80 cases of CNS tumors operated, menigioma consists of 30 cases accounting for 37.5% of total CNS tumors. The average age of presentation was 44.1 years. M:F ratio of 1:1.3.Most common affected age group was 30-50 years comprising 24 cases (80%).

The most common clinical symptoms for intracranial meningioma were headache followed by vomiting and seizures. Most common histopathological variety encountered was meningothelial in 42.72% cases. WHO grade I consists of majority of the tumors (94%) while grade II consists of 6 %.

DISCUSSION

There are currently 13 histologic variants of meningioma recognized by the WHO classification system. Due to their diverse histologic patterns, few generalizations can be applied to all meningiomas. However, the most common growth patterns are (a) meningothelial with syncytial and epithelioid cells and indistinct cell borders, (b) fibroblastic with spindle-shaped cells and indistinct cell boundaries, and (c) transitional, showing features of both meningothelial and fibroblastic patterns, often with classic whorls. Psammoma bodies are a common but highly variable feature among cases[3].

Certain features can serve as evidence of a meningioma in any craniospinal tumor. A syncytial appearance is a distinctive feature of meningothelial meningiomas and is also present in other subtypes. Whorls and psammoma bodies are characteristic of meningiomas. Meningiomas usually express EMA, distinguishing them from gliomas, but EMA expression may be reduced or lost in anaplastic meningiomas.

Meningothelial Meningiomas (Syncytial Meningioma): This is the classic syncytial meningioma, resembling small clusters of meningeal cells found normally in the meninges and choroid plexus. It is typically distinguishable from other neoplasms based on its syncytial appearance, whorls, psammoma bodies, and usual meningeal attachment. However, in some cases, it may appear epithelioid, leading to confusion with other tumors. EMA reactivity can help identify meningiomas from other tumor types.

Fibroblastic Meningiomas (Fibrous Meningioma): These firm tumors consist of spindle cells and may resemble schwannomas, fibrillary astrocytomas, and pilocytic astrocytomas. Thick collagen bundles within the tumor parenchyma can differentiate them from schwannomas and astrocytomas. Focal EMA expression is a distinguishing feature[5].

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE5, 2024

Transitional Meningioma: Comprising both syncytial and fibroblastic components, transitional meningiomas are among the easiest to identify. Prominent whorls, psammoma bodies, and clusters of syncytial cells are common[6].

Psammomatous Meningiomas: Often found in the spinal region, these meningiomas are crowded with psammoma bodies. Syncytial cells between the psammoma bodies help confirm the diagnosis.

Angiomatous Meningiomas: These tumors have features typical of benign meningiomas but contain many small or large vascular channels. Differentiating them from hemangioblastomas can be challenging, but progesterone receptor (PR) and/or EMA staining can help confirm the meningioma diagnosis[6].

Other Meningioma Subtypes: Various histologic subtypes of meningioma have been proposed, including microcystic, lymphoplasmacytic-rich, metaplastic, secretory, papillary, rhabdoid, clear cell, and chordoid meningiomas. Distinguishing these variants from other entities, including carcinomas and sarcomas, is crucial, and ultrastructural confirmation may be necessary in difficult cases[6,7,8,9,10].

Clear Cell Meningioma (World Health Organization Grade II): Clear cell meningiomas are considered biologically aggressive and are characterized by a mixture of clear cells and meningothelial features. Cytoplasmic glycogen can help confirm the diagnosis[11].

Chordoid Meningioma: This variant, similar to chordoma, is more common in childhood and can be distinguished by its focal meningothelial features and absence of CK and brachyury expression seen in chordomas. Chordoid meningiomas [12] are classified as WHO Grade II due to their tendency to recur.

Rhabdoid Meningioma: An uncommon morphologic variant, rhabdoid meningiomas are recognizable by the presence of barely cohesive cells with abundant eosinophilic cytoplasm[13]. They were previously considered highly aggressive but are now graded similarly to other meningioma subtypes.

Papillary Meningioma (World Health Organization Grade III): Papillary configurations in meningiomas are rare but associated with high rates of local recurrence and metastases. The presence of papillae with a vascular core can help differentiate papillary meningiomas from other tumors. Use of EMA and, if necessary, EM can aid in the diagnosis [6].

GRADING OF MENINGIOMAS

Mitotic index . Meningiomas, regardless of histologic subtype , are graded based on the number of mitoses identified in 10 hpfs. Meningiomas with 4 or more mitoses per 10 hpfs are classified as WHO grade 2. Meningiomas with 20 or more mitoses per 10 hpfs are classified as WHO grade 3. Ki-67 labeling indices , although not a criterion for increasing the grade of the tumor, usually follow the mitotic index and are high in most atypical (grade 2) and

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE5, 2024

anaplastic (grade 3) meningiomas (14). Discordant tumors in which the mitotic count appears low , but the Ki-67 is elevated, should be graded carefully.

Brain invasion. Invasion of adjacent cerebral parenchyma is considered to connote a higher risk of recurrence of a meningioma . meningiomas with brain invasion are assigned a WHO grade 2 . Dural or bone invasion does not impact grading . In contrast to brain , the overlaying dura and bone can be easily removed surgically, and invasion of these structures has not been associated with an increased risk of recurrence.

Histologic features . The presence of certain histologic features has been associated with more aggressive behavior in meningiomas. Meningiomas with any three of the following five histologic features : (a) prominent nucleoli , (b) necrosis, (c) growth in sheets,(d) small cell formation , or (e) hypercellularity are classified as WHO grade 2. Certain histologic variants of meningioma , such as clear cell, chordoid,rhabdoid , or papillary variants , have been associated with more aggresive behavior and were assigned a WHO grade 2 to 3. Recent evidence has put into question the association of at least some of the histologic variants with more aggressive behavior in the absence of increased mitotic activity (15).

The designation of meningioma has been extended through the years to diverse neoplasms sharing only tendency to arise within the histogenetically complex tissues of the leptomeninges or dura mater. Thus, such dissimilar entities as the meningeal hemangiopericytoma and hemangioblastoma - currently accorded separate Nosologic status among tumors of the CNS and its coverings - were once yoked under the regrettable rubric of "angioplastic" meningioma and widely assumed to derive from a common progenitor. Neuropathologists now label as meningiomas only the neoplasms exhibiting morphology or immunophenotypic evidence of an origin from meningothelial cells, Specialised elements that popular the Arachnoid membranes and cap the arachnoidal villi associated with intradural venous sinuses and their tributaries .

Meningomas, may make their appearance in childhood or adolescense,(16,17) But most are encountered in Middle or later adult life(18,19,20). Females are afflicted more commonly than males (especially at spinal levels), and some studies suggest a particularly increased prevalence in women with mammary carcinomas,(19) rare meningiomas actually harboring metastatic deposits derived from breast Primaries (22) couple with their frequent expression of progesterone as well as androgen receptors(20) and the rapid enlargement of some examples

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE5, 2024

during pregnancy or the little phase of the menstrual cycle this observations indicate that the growth of meningiomas is subject to true hormonal influence. Not working is the association of multifocal meningiomas with type 2(central) neurofibrometosis (NF-2) (20) the genetic locus for which resides on chromosome 22q12. Allelic loss involving this band is a frequent feature of meningomas including sporadic variants as are NF-2 gene mutation (particularly common in fibroblastic and transitional variants). The presentation of meningioma in childhood or adolescence should trigger investigation for underlying NF-2(17). Familial examples occurring outside the setting of classic NF-2 have also been described (20). Ionizing cranial irradiation emerges from a number of epidemiologic studies are conferring significant risk for subsequent meningioma development(19), radiation related lessons being more often multiple histologically atypical and clinically aggressive then those arising in sporadic fashion(24,20). Less clear is the etiologic role of craniocerebral trauma(19), but the presentation of select meningiomas in the immediate vicinity of a prior skull fracture or enclose physical association with traumatically implanted foreign bodies has been convincingly documented(25,26). Also on record are meningiomas found to lie just over a glioblastoma or other glioma(27). Most collision tumors of this type are undoubtedly fortuitous lesions, but it is conceivable that the occasional meningioma evokes a hyperplastic glial reaction that subsequencely progress to neoplasia. As noted in our discussion of gliomesenchymal neoplasmas the term sarcoglioma has been extended to some mixed tumors postulated to have a arisen in this fashion(28). As a mentioned in our prior discussion of hamartomatous lesions, meningiomas very occasionally arise in association with foci of meningioangiomatosis(29,30).

Most meningiomas arise within the cranial cavity are dura based and are found in the vicinity of the superior sagittal sinus over the cerebral convexities or in contact with the Falx cerebri. Basally positioned examples favour the sphenoid ridge olfactory grooves tuberculum ,sellae and paracellar region.. Intacranial meningioma may also originate within the stroma of the choroid plexus and rest entirely within the ventricular system. Also recognised are epidural (intradiploic),calvarial, and intrapetrous meningomas as well as variants located entirely outside the craniospinal confines. The latter are usually uncountered in the head and neck region and include orbital, glabellar sinonasal,oropharyngeal,subgaleal,juxtaparotid,and cutaneous examples(31,32). Rarely meningiomas are situated at even greater removes from the central neuraxis (e.g.,in the mediastinum(33)lung(34,35),or brachial plexus(36)(23)).

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE5, 2024

On neuroradiologic and gross assessment the typical meningioma is a solid lobulated or globose mass that is broadly anchored to the duramater. Cystic variants although uncommon are well recognised and the term meningioma in plaque maybe invoked for the occasional reason that presents (usually over the sphenoid ridge) as a poorly deleted blanket like growth. Neuro radiology features that should prompt concern include indistinct tumoral margins , a mushrooming growth pattern characterized by multi nodular projections (this representing regions of necrosis), and edema of the neighbouring brain on CT or MR study(37).

On sectioning most meningiomas are greyish -tan and soft but collagenized having a rubbery texture and whorled trabeculated cut surface (resembling that of the Leiomyoma) where areas variants rich in stromal mucopolysaccharides of somewhat gelatinous consistency.

Meningiomas are Notorious for the variety of their cytology and histology presentations but most viewed of several prototypical guises(18,20)."Meningiotheliomatous" Variant are characterized by a lobular microarchitecture and are populated by cells having delicate round of oral nuclei in inconspicuous nucleoli lightly yersenophilic cytoplasm and indistinct cytoplasmic borders. Common to this (and other subtypes) are tumor cells consentrically wrapped in tight whorls, nuclear clearing and pale nuclear "pseudoinclusions"consisting of invaginated cytoplasm, and the calcospherules known as psemmoma bodies. While none of this features are pathognomonic of meningioma, there demonstration in the setting of an extra axial dura based mass carries considerable diagnostic weight. In contrast to the epithelioid appearance of meningoliometous variants fibrous (or fibroblastic) meningiomas adopt mesenchymal profile being variable and consisting of tumor cells in fascicular or storiform array. An example containing crystalline structures rich in tyrosin has been depicted(38). These are often particularly reach in compact cellular whorls and endowed with psammoma bodies in conspicious numbers. When the letter are present in profusion the term psammomatous meningoma may be applied to tumors of this type characteristically occur in middle aged women and exhibit a particular predilection for the intraspinal compartment.

The **microcytic meningioma** (39,40) named for its content of variable sized intercellular vacuoles this often appearing empty but in some instance containing a lightly PAS- positive fluid derived in all likelihood from the transudation of plasma across a characteristically rich and frequently hyalinized stromal vasculature. Some examples actually progress to the formation of microcysts and harbor only minor solid components. Constituent cells exhibit

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE5, 2024

cytoplasmic clearing due to glycogen or lipid accumulation and often assume stellate profiles that along with their tendency to disaggregation can promote consideration of a low - grade microcytic astrocytoma in the differential diagnosis.

The **Secretary meningioma**,(42) a variant of the meningotheliomatous subtype, is distinguished by its content of "pseudopsammoma bodies"-globular hyaline inclusions that are eosinophilic, intensely PAS positive, and diastase resistant . On ultrastructure and study this can be shown to lie within microvillus-lined intracellular lumina and maybe immunolabeled for human secretary component, IgM,IgA,and CEA. Nearly always benign, the secretary meningioma may yet masquerade as a malignant neoplasm by virtue of its occasional association with elevated serum CEA levels, (43)an especially confounding phenomenon in the patient with a prior history of systematic cancer and with progressive neurologic deficits referable to severe edema of juxtatumoral cerebral tissues. The lattar ,generally foreign to conventional meningioma and usually uncountered as a complication of malignant Meningeal neoplasms (primary or metastatic), maybe associated with curious pericytic proliferation occurring in the tumoral vascular bed(44).

The **lymphoplasmacyte- rich meningioma** is a tumor infiltrate by chronic inflammatory elements at times so heavily that it's meningothelial (and neoplastic) nature is obscured(45,46). There can be no doubt that at least some reported cases would be better classified as dura based examples of inflammatory pseudotumor ("plasma cell granuloma") aur sinus histocytosis with massive lymphadenopathy (Rosai - dorfman disease). Peritumoral lymphoplasmacytic infiltrates with germinal centre formation are also conspicuous features of a peculiar meningeal tumor noteworthy for its **choroid histology** and presentation in childhood or adolescence with manifestations of the, **castleman syndrome** - polyclonal dysgammaglobulinemia, iron-refractory anemia,hepatosplenomegaly and retarded growth and sexual maturation(47,48).

The latter, also reported as **"myxoid"**or **"mucinous meningiomas(49)** Proved in one analysis to be more prone to local regrowth following sub total resection (49)

Metaplastic meningiomas can contain bone, cartilage, aur adipocytic elements(18,20). Progressive and xanthomatous change, non specific cytoplasmic lipidiztion, rather than true metaplasia seems to account for so called **lipomatous meningioma (50).** We would call particular attention to rare variants that may be misconstrued as sarcoma owing to potentially

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE5, 2024

lipoblastic cytology features and the occurrence in some examples of troubling nuclear abnormalities that are almost certainly degenerative in nature (51,50). Neoplasms exhibiting meningothelial and rhabdomyosarcomatos (52)or leiomyosarcomatous (53) differentiation are also on record. (54), nesting arrangement reminiscent of paraganglioma;or a hyalinized stromal vasculature so exuberant and domineering as it to suggest a malformative process **(angiomatous meningioma(18,20)** A **sclerosing subtype** that appears to undergo progressive fibrous obliteration is notewothy for its presentation in the pediatric group and favorable outcome despite a tendency to foci of disturbing hypercellularity, pleomorphism and cerebrocortical infiltration(778). Some observers have suggested that at least a subset of sclerosing childhood meningiomas are pseudoinvasive lesions arising within cortical foci of meningioangiometosis (29).

A Subset of meningothelial neoplasms evidence clear cell, oncocytic or rhabdoid features. The significance of such such alterations when uncountered only focally is unsettled, but tumors exhibiting prominent changes of this sort merit distinct designation owing to their potential for mischief. **Clear cell meningimas** are the most deceptive of the lot(56,57,22). These are characterized by a predilection for young subjects (including children) and are most often found in the spinal canal, cerebellopontine angle, or foramen Magnum region. They are usually extra exile and dura- based, but maybe associated with cranial nerves, spinal roots are the cauda equine. Composed of glycogen rich water clear cells that are often disposed in pattern less sheets traversed by bands of hyalinized collagen, this unusual tumors typically manifest little or nothing in the way of classic meningothelial attributes(e.g. whorls and nuclear pseudo inclusions) and maybe only focally and faintly immuno reactive for epithelial membrane antigen (EMA), a marker of specialized arachnoidal cells expressed by most meningomas in diffuse fashion. High recurrence rates and increased mortality characterize this intrinsically aggressive meningoma variant, this despite the fact that most examples do not evidence conspicuously increased mitotic activity, necrosis or other histology features that would a arouse suspicion.

That rhabdoid cytology is, in general, a maker of increased biologic potential is born out by the discouraging behaviour of most meningioma displaying this profile, which may be evidence on enitial presentation or appear only in recurrent material(20,59). Rhabdoid meningomas in fully developed from usually retain meningotheliomatous regions but boast a prominent

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE5, 2024

population of sales with vascular nuclei, prominent nucleoli , and Globose paranuclear inclusion-like bodies representing compacted cytoplasmic vimenting filaments. This cells usually lie in lobules or sheets, butter papillary growth pattern may be encountered(60). Conspiciously increased mitotic activity is the rule, and brain invasion common, most such tumors qualifying as atypical or frankly anaplastic by the WHO/Mayo clinic criteria presently discussed. In one reported series, 87% of afflicted patients experienced at least one recurrence and 53% died of tumor progression(59). The prognosis for examples evidencing rhabdoid change in the absence of elevated proliferative activity or other aggressive indicators does not appear to be as poor, but this need close watching. Similar consideration seems to apply to oncocytic meningiomas, i.e., meningothelial neoplasms demonstrating fine cytoplasmic granularity due to an accumulation of mitochondria(61). High mitotic rates, necrosis, brain invasion and recurrence have been overrepresented in the few cases recorded to date and in examples that we have uncountered in consultation.

A final variant meriting specific comment in view of its distinctive histology and clinical biology is the **papillary meningioma**, a tumor characterised by the ependymoma - like peri vascular structureing of its constituent cells(62,63,64). The latter can be seen to extend variable elongated cytoplasmic processes toward vessel walls, fashioning pseudorosette - like structures that disaggregate and come to float unanchored in tissue section. Regions exhibiting a more conventional meningothelial appearance are nearly always identifiable but usually depart from the typical in evidencing worrisome hypercellularity,mitotic activity, and, in some cases,foci of coagulative necrosis. Stubborn local recurrence, capacity for extraneural metastatic, and often fatal outcome characterize papillary meningioma. An excess of reported cases have presented in childhood or adolescence.

Distinction of the meningioma from potential counterfeits occasionally requires the use of the electron microscope or immuno histochemical assay. The most constant and distinctive ultrastructural feature of meningothelial Neoplasms is the interdigitation of tumor cell processes without intervening basal lamina material (elaborated by both the meningeal hemangiopericytoma and schwannoma), Although fibroblastic variants tend to a more parallel alignment. Intercellular junctional complexes are frequent and include well developed desmosomes conspicuous cytoplasmic complement of intermediate filaments rounding out the ultra structural picture. The latter consist of vementin, regulary demonstrable by immuno

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE5, 2024

histochemical methods regardless of tumor subtype(20). Of particular diagnostic utility is the observation that all large majority of meningomas exhibit(at least focally) membraneous, as well as diffuse, cytoplasmic immuno labelling for EMA(20), a features foreign to the hemangiopericytoma, To nerve sheath tumors other than the rare perineurioma the solitary fibrous tumor, and other fibroblastic neoplasms. Nuclear immuno reactivity for progesterone receptors is also common(20). Reactivity for S-100 protein, if present, is usually limited to the cytoplasm of a subset of neoplastic cells but may occasionally be encountered is diffuse form. As regards cytokeratin expression, meningomas often share with native arachnoidal lining cells (and with epithelial cancers) immuno labelling for CK18, but typically CK20-negative regardless of histology pattern(65). Focal reactivity for CK 7, CK 8 (CAM 5.2), CK 19 and EA1/3 maybe encountered (and is a consistent feature of the inclusion - bearing elements that define meningiomas of secretary type), but widespread labelling for these antigen suggests that are dura - based mass represents metastatic carcinoma(65). GFAP-labelling meningomas of papillary(58) and rhabdoid (60,59,66)type have been depicted but are curiosities . Whether this phenomenon actually reflects GFAP expression is open to question. In fact, one report depicting GFAP reactivity in a tumor interpreted as meningothelial(67) seems instead to represent the first account of the neoplasm now recognised as chordoid glioma of the third ventricle(68). As previously noted, meningomas really exercise an ability to differentiate along myogenic lines (52,53)and some otherwise conventional example focally label for muscle associated actins(69).

Meningima's of conventional histology type grow slowly and are amenable tu surgical cure when complete excision can be affected, as is usually the case for examples arising over the cerebral convexities or along the spinal axis. Even tumors so favourably situated , however, main recur following gross total resection, magnitude of this risk emerging only on long term observation. In one study for example respective 5-,15-, and 25 year relapse rates for histologically benign and completely excised intracranial meningiomas were 3%, 15% and 21%(37). By all accounts, the likelihood of regrowth is considerably higher for the less accessible olfactory groove sphenoid wing meningiomas, In plaque examples and lesions invasive of the cranial floor proving particularly troublesome(70).

Application of the WHO/Mayo clinic criteria outline below broadly stratifies meningothelial tumors into three tiers of increasing biological potential **meningioma, atypical meningioma,**

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE5, 2024

and anaplastic meningioma (WHO grades l,ll,and lll, respectively). The stated criteria are applied whether present as focal finding (often the case)or in more diffuse form. In this scheme, a typical meningomas are defined as :

1). Containing 4 or more mitotic figures per 10 high power microscopic fields (0.19mm)2.

2). Exhibiting at least three of the following features :

• hypercellularity

• patternless, sheet like growth

• macronucleoli

• small cell components with high nuclear :cytoplasmic ratio

• zones of necrosis

Anaplastic meningiomas are defined as:

1). Containing 20 or more mitoses per 10 high power microscopic fields.$(0.16mm)^{2.}$

2). Exhibiting a loss of differentiated features resulting in carcinoma,Melanoma or sarconoma like appearance.

We would emphasize that the preoperative embolization of meningiomas, Undertaken to reduce their blood supply and facilitate removal, often results in regions of necrosis with evident mitotic activity (and increased MIB - 1 reactivity) in adjoining tumor tissue(74). Potentially rendering problematic application of the foregoing criteria, this phenomenon is often, though not invariably, signaled by the presence of clearly foreign material within the tumoral vasculature. The finding of multiple zones of necrosis that appear to be at the same acute stage of development suggests sach intervention but, in any case, pathologist should consult their clinical colleagues on this issue prior to releasing reports. The study cited above suggests that WHO/Mayo clinic mitotic criteria remain valid in this circumstances, but confirmation in additional large series would be reassuring.

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE5, 2024

Brain invasive meningomas, in fact, span the spinal histology spectrum, some being of otherwise typical appearance(20,72). The demonstration of brain invasion per se adds relatively little to the predictive power of the WHO/Mayo clinic scheme as a prognostic model once a given tumor has satisfied the listed criteria for designation as atypical or anaplastic. Meningothelial tumors of conventionally benign histology aspect that infiltrate brain, on the other hand, appear to behave much in the manner of morphologically atypical meningioms (see below) and so we endorse the Mayo clinic suggestion (72) that thay be so designated, though this is not specifically advocated by the WHO(20).

In the experience that served as the basis of this recommendations, respective 5 year recurrence rates for conventional versus atypical meningomas (the latter including brain invasive but otherwise benign examples) were 12% and 14% following gross total resection(72,73). Atypical tumors carried 5 year mortality rate of approximately 20%. Anaplastic meningiomas, by contrast recurred in the large majority of cases (ostensibly complete excision not withstanding),were associated with a 68% 5 year mortality rate and a median survival of only 18 months. Other findings that have been correlated which worrisome histology and increased biologic potential in the setting of meningothalial neoplasia include elevated MIB 1 labeling indices and in some studies failure to express progesterone receptors(20). Regarding the former, meningomas of conventional type and indolent behaviour usually have MIB 1 labeling indices below 4 to 5% but there is overlap in the values recorded for Benign atypical and anaplastic lesions as well as for recurring versus non recurring tumors(75,76,77).

Consequently, the predictive power of this assessment in the individual case in some what compromised. Noteworthy is the observation that distributing histology features, including elevated mitotic MIB 1 indices and brain invasion, same to be over represented among meningiomas occurring in the paediatric group(17). as detailed elsewhere(17), most studies have found this morphologic attributes to correlate with an increased risk of recurrence and fatal outcome in this patient cohort, but this has not been the experience of all. Progression-associated genetic markers, such as chromosome 1 p and 14 p deletions, may also be more prevalent in meningiomas of childhood and adolescence compared to adult one set examples(17). The reader is referred Elsewhere for more comprehensive discussion of genetic alterations associated with meningioma progression(20). At the time of this writing, these have no generally accepted role in patient management.

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE5, 2024

While application of the WHO/Mayo clinic guidelines discussed above can be recommended as a means of stratifying affected patients into groups at increasing risk of recurrence and meningioma related death, pathologist and attending clinicians must realise that prognostic certainly is not to be found in the proposed criteria-particularly in the mitotic threshold that separate the typical, atypical and anaplastic. It has long been our practice to specifically comment with confronted by any meningioma containing more than the very occasional mitosis found on careful scrutiny, close neuroradiologic surveillance being prudent in this circumstances even if a growth total excision has been affected. It also bears emphasizing that within the broad spectrum of meningomas that qualify as merely"atypical"by WHO/Mayo clinic criteria are to be found examples (e.g. the tumor exhibiting 14 mitosis per 10 high power microscopic field,necrosis, and cerebrocortical invasion) that may not be as predictably virulent as Anaplastic variants but that are capable of frankly malignant clinical behaviour. The last words have not been written to these issue.

Although local regrowth is the major pattern of treatment failure, aggressive meningioma variants can spread via the CSF and on occasion travel to extraneural sites such as the lung, liver, bone and lymph node (18,20) . We would point out however that an excess of distant metastates recorded in the literature have derived from "angioblastic"variants that would now be classified as meningeal hemangiopericytomas . Examples of "**benign metastasizing meningioma** "have been well documented but remain Curiosity (75). As noted, Venous sinus invasion is a feature of maningiomas and is not predictive of hematogenous dissemination. In a similar vein, little significance attaches to foci of pronounced nuclear pleomorphism, provided that nucleolar enlargement , mitotic activity, or other atypical features are not in evidence. X- chromosome inactivation studies and NF-2 gene mutation analysis suggest that at least some sporadic cases of "multi focal" meningioma (particulary those a characterized by the presence of three or more spetially distinct tumors) actually represent clonal proliferations with subarachnoid spread.(20).

CONCLUSION

This study provides a comprehensive examination of the histological features of meningiomas, highlighting the characteristic morphological patterns and cellular characteristics of these tumors. Our findings demonstrate the importance of accurate histological diagnosis, as the subtype and grade of meningioma can have significant implications for patient prognosis and treatment. The histological features observed in this study, including the presence of whorl formations, psammoma bodies, and nuclear pseudoinclusions, are consistent with the expected

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE5, 2024

pathological features of meningiomas. Notably, the identification of specific histological subtypes, such as transitional, fibroblastic, and atypical meningiomas, underscores the heterogeneity of these tumors. These findings have important implications for the development of effective treatment strategies and prognostic models. Further research is warranted to explore the molecular mechanisms underlying the histological changes observed in meningiomas, as well as to identify novel therapeutic targets for these tumors. Ultimately, this study contributes to a deeper understanding of the complex histological features of meningiomas, informing the development of more accurate diagnostic and prognostic models to improve patient outcomes.

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