Review Article

Intracranial Meningioma

Azmy M. Al-Hadidy,[1] Waleed S. Maani,[2] Waleed S. Mahafza,[1] Mahasen S. Al-Najar [1] and Mustafa M. Al-Nadii[2]*

Abstract

Meningiomas are the most common benign intracranial tumor, accounting for 15-30 % of all primary intracranial tumors. They are usually diagnosed between 40-60 years and they are more common in females, but the aggressive type is more common in men and children. Pathologically, Meningiomas are divided into three grades:
1- Benign (90 %) 2- Atypical (5-7%) 3- Anaplastic (3-5%).

The gross appearance of Meningioma is usually globular mass with regular surfaces and attached to the dura, but there is a certain type called meningioma en plaque, has a flattened appearance that conforms to the curves of the brain and the inside of the skull. Studying the immunohistochemistry and the biological activity of this tumor showed the presence of Epithelial Membrane Antigen (EMA) in 80 % of cases, in addition to the presence of progesterone receptors in 57- 67 % of cases which is associated with a good histological grade, lower frequency of recurrence, and overall favourable prognosis.

Many causes are thought to be associated with meningioma like trauma, viral infection, radiation and genetic factors. The signs and symptoms are variable, and some of them are specific to the location of the tumor. The diagnosis of meningioma is done by CT scan and MRI, in addition to nuclear imaging and MRSpectroscopy. In many cases, angiography is also done either conventional or via MRAngiography. The treatment methods depend on the size and site of tumor, patient's age and clinical presentation starting by clinical observation and ending by surgical management.

This article will discuss all of the above in detail with review of the medical literature.

Keywords: Meningioma, Meningioma type, Progesterone receptors, radiation.

(J Med J 2007; Vol. 41 (1): 37- 51)

Received	***Accepted***
April 23, 2006	*November 19, 2006*

1- Department of Radiology, Jordan University Hospital, Amman, Jordan.
2- Department of Neurosurgery, Jordan University Hospital, Amman, Jordan.
*** Correspondence should be addressed to:**
Azmy M. Al- Hadidy, MD
Department of Diagnostic Radiology, Jordan University Hospital.
P.O. Box: 13046 Amman 11942, Jordan.
E-mail: ahadidy@ju.edu.jo.

Introduction

Meningiomas have left their mark, in the form of hyperostosis, on human skulls as far back as prehistoric times. The recognition of these tumors as pathologic entities started in the seventeenth century.[1] In 1614, Felix Plater first described meningiomas at an autopsy.[2]

In the eighteenth and nineteenth centuries, meningiomas were diagnosed during life only if they caused changes on the overlying skull that could be appreciated by inspection or palpation. Only few attempts were made to remove these lesions surgically, and few were beneficial to the patient. Of 13 such operations performed between 1743 and 1896, nine ended in death.[1]

In 1864, John Cleland, Professor of Anatomy in Glasgow, described two tumors adherent to the deep surface of dura mater; the tumors took their origin from the arachnoid rather than the dura.[3,4] In 1915, Cushing and Weed reconfirmed Cleland's opinion that meningiomas were indeed derived from arachnoid cell clusters.[5] Harvy Cushing proposed the term meningiothelioma. Later, he opted for the term meningioma. In 1922, he reported 85 cases of meningiomas. He wrote "There is today nothing in the whole realm of surgery more gratifying than the successful removal of meningioma with subsequent perfect functional recovery…..".[4] In 1938, Cushing and Eisenardt published *meningiomas: Classification, Regional Behavior, Life History, and Surgical End Results,* in which they reported in detail 313 patients encountered between 1903 and 1932.[2, 6, 7]

Epidemiology

Meningiomas are the most common benign intracranial tumor, accounting for 15-30 % of all primary intracranial tumors. [8] They have an annual incidence of 6 per 100,000 population. [9] They are twice as common in the female as in the male population [8] but a reverse male-to-female preponderance of 3:1 has been reported in the malignant form. The incidence increases with age with peak incidence between the age of 40-60 years. A slight drop after the eighth decade was noted.[10] An estimated 2-3 % of the population has an incidental asymptomatic meningioma, and in autopsy studies, 8 % of these are multiple.[11] Meningiomas are more prevalent in blacks, but female preponderance may be less pronounced in the black population than in other groups. Although overall meningiomas are more common in female patients, they tend to be more aggressive with a poorer prognosis in men as well as in children; as malignant meningiomas and meningeal sarcoma accounted for 28% of all primary meningeal tumors in children.

Meningiomas constitute 1- 4 % of all childhood (< 18 years) brain tumors.[12, 13] The average age at presentation is 11.6 year. The incidence of males to females is equal in childhood meningiomas.

In children, they may arise in unusual sites where intraventricular childhood form about 11-20 %. Meningiomas are extremely uncommon in infancy, with a male predominance of 71 %.

The incidence of multiple tumors is in the range of 1-16 % of meningiomas. 60-90 % of patients with multiple tumors are women. The average age at presentation is 50 to 60 years, except for patients with neurofibromatosis (NF2) where a younger age is common.[6]

Pathology

Table 1: WHO Classification of MENINGIOMAS.

Grade	Examples	Remarks
***I**, Benign (90%), low risk of recurrence (7-20%) and aggressive growth*	*Meningothelial(syncytial), fibrous (fibroblastic), transitional (mixed) , psammomatous, angiomatous, lymphoplasmacyte-rich, microcystic, secretory, calcified, metaplastic , meshy meningioma*	*Occasional mitotic figures, pleiomorphic nuclei do occur, tumor cells express epithelial membrane antigen. Characteristic histological features of cellular whorls and psammoma bodies*
***II**, Atypical (5-7%), increased likelihood of recurrence (29-40%) and/or aggressive behavior*	*Atypical , clear cell (intracranial), chordoid meningioma*	*Specific histological features: mitotic rate of at least four mitotic figures/10 hpf(most important) or at least three of the following: 1. increased cellularity; 2. small cells with a high ratio of nucleus to cytoplasm; 3. prominent nucleoli; 4. sheet-like growth pattern; and 5."geographic" necrosis .*
***III**, Anaplastic (3-5%), increased likelihood of recurrence (56%) and/or aggressive behavior*	*Rhabdoid , papillary , anaplastic (malignant) , any meningioma subtype or grade with brain invasion and /or high proliferation index*	*Papillary: perivascular pseudo-tumor pattern,highly aggressive, metastasize in 20%. Rhabdoid:contains rhabdoid cells with eccentric nuclei, abundant globular eosinophilic cytoplasm, and paranuclear inclusions, and they show focal immunoreactivity for epithelial membrane antigen. Anaplastic: malignant cytology , a high mitotic rate (20 or more mitotic figures/ 10 hpf), or both. a high frequency of local and brain invasion, recurrence, and metastases*

Meningiomas are thought to originate from arachnoidal cap cells (cells forming the outer lining of the arachnoid membrane),[14] in addition to related cells such as arachnoidal fibroblasts or perhaps the precursor cell of the meninges as a whole –the so-called meningioblast. Although they can potentially occur at any site in the meninges, about 90 % are supratentorial and 10 % are infratentorial. Certain intracranial locations are more common than others (Table 2).

Uncommonly sited tumors include intraosseous meningiomas and extraneuraxial meningiomas. 16 % of the reported primary extraneuraxial tumors occurred in the skin and the subcutaneous tissue; others have been reported in the lungs, mediastinum and adrenal gland.

The gross appearance of meningiomas is mostly well-demarcated, round or oval, frequently lobulated tumors and attached to the dura (globular meningiomas). The fibroblastic and transitional types are firm to hard in consistency whereas the angiomatous and meningothelial types are soft in consistency.

A certain type, called meningioma en plaque, has a flattened appearance that conforms to the curves of the brain and the inside of skull.

Meningiomas may invade surrounding tissues, including the dura, adjacent bone, soft tissue of orbit and the paranasal sinuses. This makes complete excision of the tumor difficult and; consequently, increases the incidence of local recurrence after operation.

Immunohistochemistry

Immunohistochemistry can help diagnose meningiomas, which are positive for Epithelial Membrane Antigen (EMA) in 80% of cases. They stain negative for anti-Leu 7 antibodies (positive in schwannomas) and for Glial Fibrillary Acidic Protein (GFAP). Progesterone receptors can be demonstrated in the cytosol of meningiomas; the presence of other sex hormone receptors is much less consistent. Somatostatin receptors also have been demonstrated consistently in meningiomas.

Biological Activity

Expression of proliferation markers such as MIB-1 and Ki67 has generally shown progressive increases in labelling index with WHO grade from 1.00–1.35% for grade I, to 1.90–9.30% for grade II or the atypical, and 5.60–19.5% for grade III or the anaplastic meningioma. [8, 15- 17] Tumors that recur tend to have higher labelling indices than those that do not (2·30–10·9% *vs* 0·39–3.80%).[17]

Expression of other proliferation-associated markers, such as mitosin and topoisomerase IIα, correlates well with MIB-1 labelling index.[16, 18] Conversely, other studies have found no relation with tumour grade [18, 19] or did not find the labelling index for it to be an independent prognostic marker. [20]

Other biological factors, such as apoptosis-related proteins (p53, p21WAF1, and p27Kip1)[15] or growth factors (transforming growth factors α and β, and platelet-derived growth factor),[21] are likely to be important in meningioma progression or recurrence.

Many tumors (57–67%) express progesterone receptors (PR).[22,23] Several studies have shown associations between high PR expression and good histological grade, lower frequency of recurrence, and overall favourable prognosis.[20,21,24] Somatostatin receptors are also expressed by many other tumors (70–100%).[25,26]

The biological function of these receptors, predominantly type 2a (hsst2a) receptors, is unknown.[26]

Causes

- Trauma and viruses have been investigated as possible causative agents for the development of meningioma. However, no definitive proof has yet been found.
- There is a role for radiation in the genesis of meningioma. Patients subjected to low-dose irradiation for tinea capitis may develop multiple meningiomas decades later in the field of irradiation. High-dose cranial irradiation may induce meningioma after a short latency period.
- Genetic causes have been implicated in the development of meningioma. The best characterized and most common genetic alteration is the loss of the NF-2 gene on chromosome 22q. NF2 encodes a tumor suppressor known as merlin (or schwannomin). The meningioma locus is close to but probably different from the gene responsible for NF-2. Up to 60% of sporadic meningiomas were found to harbor *NF2*. The next most common genetic mutations seen in meningiomas after loss of 22q are deletions of 1p, 3p, 6q, 9p, 10q, and 14q.[27] Loss of chromosome 10 is associated with an increased tumor grade, shortened time to recurrence and shortened survival.

Progression to anaplastic form has been associated with involvement of the chromosomal site 17q. The following events were found to be associated with higher grades of meningiomas: loss of the Tumor Suppressor in Lung Cancer-1 gene (*TSLC-1*), loss of progesterone receptors, increased expression of cyclo-oxygenase 2 and ornithine decarboxylase. Monosomy of chromosome 7 is a rare cytogenetic change, however, it is frequently reported in radiation-induced meningioma. The invasive potential of meningioma cells seems to be reflected by a balance between the expression of Matrix Metallo-Proteinases (MMPs) and Tissue Inhibitors of MMPs (TIMPs). The most consistent chromosomal abnormality isolated is on the long arm of chromosome 22.

Clinical Presentation

Meningiomas produce their symptoms by several mechanisms. They may cause symptoms by irritating the underlying cortex, compressing the brain or the cranial nerves, producing hyperostosis and/or invading the overlying soft tissues, or inducing vascular injuries to the brain. The secondary signs and symptoms may appear or become exacerbated during pregnancy, but usually abate or improve in the postpartum period. By irritating the underlying cortex, meningiomas can cause seizures. Some symptoms and signs are specific to tumor's location and the tumor may be gigantic before it becomes symptomatic. (Table 2).

The presentation may be due to vascular events. Tumors of the skull base may narrow and even occlude important cerebral arteries, possibly presenting as Transient Ischemic Attack (TIA)–like episodes or as a stroke.

Another presentation is in the form of Intraventricular meningioma that may present with obstructive hydrocephalus but can also rarely present as intraventricular hemorrhage, especially if it is of the fibroblastic type. [28]

Tumors in the vicinity of the sella tursica may produce panhypopituitarism. Tumors that compress the visual pathways produce various visual field defects, depending on their location. Patients with chordoid meningiomas may exhibit the presence of iron refractory anemia and polyclonal gammopathy (Castleman's syndrome); both of which remit with resection but reappear upon recurrence. [29] One of the rare presentations is subdural hemorrhage caused by angiomatous meningioma. Anaplastic tumors may rarely be exhibited as metastasis especially to the lung, abdominal viscera and bones.

Compression of the underlying parenchyma may give rise to pyramidal signs that are exemplified by pronator drift, hyperreflexia, positive Hoffman sign, and presence of the Babinski sign. Compression of the dominant (usually left) parietal lobe may give rise to Gerstmann syndrome:dysgraphia/ agraphia, dyscalculia/acalculia,left-right disorientation and finger agnosia. Raised intracranial pressure leads to papilledema, decreased mentation and, ultimately, to brain herniation.

Diagnostic Studies

Plain X-ray may show calcification within the tumor, hyperostosis (which may be localized or diffused as in en plaque meningiomas) or blistering of the skull and enlargement of vascular grooves. Indirect signs are those of increased intracranial pressure such as demineralization of sella turcica and brain shift (displacement of calcified pineal gland). Rare osteolysis is associated with the benign and aggressive types.

CT with and without contrast detects about 85-95% of meningiomas respectively. The typical non-contrasted CT appearance is of sharply demarcated mass attached to the dura with an obtuse angle that is isodense (25 %) or slightly hyperdense (75 %) relative to adjacent brain, [30] but few appear hypodense (lipoblastic and xanthomatous variants).

Table 2: Intracranial locations that are more common than others for Meningioma, and the Symptoms.

Location	*Symptoms*	*Percentage*
Parasagittal	*Monoparesis of the contralateral leg*	*24.4%*
Subfrontal	*Change in mentation, apathy or disinhibited behavior, urinary incontinence*	*1.5%*
Olfactory groove	*Anosmia with possible ipsilateral optic atrophy and contralateral papilledema (this triad termed Kennedy-Foster syndrome)*	*10%*
Cavernous sinus	*Multiple cranial nerve deficits (II, III, IV, V, VI), leading to decreased vision and diplopia with associated facial numbness*	*7.3%*
Occipital lobe	*Contralateral hemianopsia*	*1.5%*
Cerebellopontine angle	*Decreased hearing with possible facial weakness and facial numbness*	*10.5%*
Optic nerve	*Exophthalmos, monocular loss of vision or blindness, ipsilateral dilated pupil that does not react to direct light stimulation but might contract on consensual light stimulation; often, monocular optic nerve swelling with optociliary shunt vessels*	*1.2%*
Sphenoid wing	*Seizures; multiple cranial nerve palsies if the superior orbital fissure involved*	*16%*
Tentorial	*May protrude within supratentorial and infratentorial compartments, producing symptoms by compressing specific structures within these 2 compartments*	*3.6%*
Foramen magnum	*Paraparesis, sphincteric troubles, tongue atrophy associated with fasciculation*	*1%*
Convexity	*Epilepsy and focal neurological signs*	*23%*

It appears as a homogenous vividly enhancing mass, with a collar of thickened enhancing tissue that surrounds their dural attachment; this is known as a dural tail which may be either reactive or neoplastic. A dural tail occurs in approximately 65% of meningiomas and 15% of other peripheral tumors, it appears to be highly suggestive of meningiomas, but it is not pathognomonic. Cerebral edema is absent in 50% of patients because of slow growth.

There may be only mild to moderate degree of cerebral edema as seen mainly in fibroblastic and transitional cell tumors, or it may be marked which tends to be associated with tumors of syncytial or angioblastic cell type and may extend throughout the white matter of the entire hemisphere. [31] Intraventricular tumors produce extraventricular edema in 50 % of cases. Generally, edema is more apparent on MRI than on CT scan. Studies have indicated that the presence of edema correlates well with either the tumor blood supply coming from cerebral pial arteries, or with its venous drainage into the cortical cerebral veins, or with tumor infiltration into adjacent brain parenchyma. [31]

CT is effective in showing calcification of meningiomas (Figure 1), diffuse or focal (25%-50%) and hyperstosis (which may be present in up to 15%-20 %). Calcification is seen in as many as 50 % of the cases of intraventricular meningiomas. [28] CT is also effective in showing bone destruction, erosion at the site of dural attachment, acute tumoral hemorrhage and widened vascular grooves in the calvarium.

MRI was initially thought to be poor in the detection of meningiomas, nevertheless, current MRI can show most meningiomas on T2 WI unless it is nearly totally calcified, so it is the investigation of choice as it can show the dural origin of the tumor in most cases.

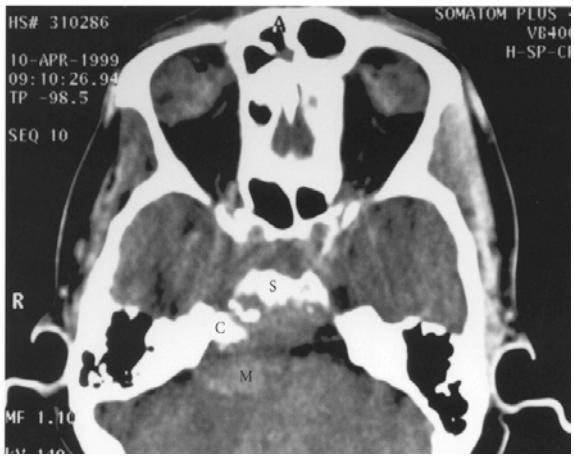

Fig 1: Axial CT-Scan showing Rt Sphenoidal ridge meningioma (M) with calcification (C) and hyperostosis(S).

Characteristic MR Findings are: 1) Cortical buckling – inward displacement of the cortical grey matter. 2) Cerebrospinal fluid clefts which are identifiable in 80%[47] of the cases. 3) Broad base against the dural surface. 4) Pseudocapsule of displaced vessels of subarachnoid space. 5) Secondary intraaxial vasogenic edema. 6) Dural tail sign.

Other lesions with adjacent meningeal enhancement may mimic meningioma, such as neuroma, chloroma, metastasis, lymphoma, glioma, pituitary disease, granulomatous disorders, or cerebral Erdheim-Chester disease.[32] Several other disease processes have a propensity for primary involvement of the dura matter or subdural space giving a meningioma -like appearance, [33] including metastatic disease (lymphoma and adenocarcinoma), inflammatory lesions (sarcoidosis, Wegener's granulomatosis), and infections (tuberculosis).

Histologic subtypes may have different MRI appearances, but this is not sufficient for a histologic diagnosis by using MRI. Tumor signal on T1 images were rather similar regardless of the histologic subtype of the tumor which was most commonly isointense (Figure 2B) or slightly hypointense to brain.

On T2WI, hypointense tumors were mainly fibroblastic and hyperintense tumors (Figure 2A) were mainly syncytial, angioblastic, chordoid and partly transitional. Isointense tumors were mainly transitional and partly fibroblastic and syncytial, [34] while sclerotic meningiomas were isointense relative to the gray matter on T1WI and markedly hypointense on T2WI.

Meningiomas enhance intensely and homogeneously after injection of gadolinium gadopentate with T1 sequence (Figure 2C). Post contrast FLAIR sequence is not valuable in meningiomas in contrast to other extraaxial diseases (meningoencephalitis and leptomeningeal metastases). [35] Heterogeneity of signal intensity (T1 &T2) may also be seen if calcification, high vascularity, cystic or necrotic changes are present. Gadolinium enhancement is found especially useful in delineating en-plaque meningiomas. Chen et al. [36] found that aggressive meningiomas were more vascular, but that there was no correlation between the degree of surrounding edema or contrast enhancement and the histopathologic findings.[31] Servo et al. [37] and younis et al. [38] determined that CT can't reliably distinguish malignant tumors from benign ones. There are, however, some CT or MRI trends that point in favor of the malignant meningiomas: **1**) the absence of visible calcium aggregates. **2**) "mushrooming"or the presence of a prominent pannus of tumor extending well away from the globoid mass **3**) non-homogeneous enhancement **4**) necrosis **5**) the presence of indistinct tumor margins.

If angiography is performed, AV shunting is a feature that suggests malignancy. Controversial features in relation to malignancy [47] include marked peritumoral edema, osteolysis (Figure 4A), intrinsic cyst like areas and tumor density. Elster et al. [39] couldn't detect any significant difference on either TI or T2W studies which allowed differentiation of malignant from benign meningiomas.

As with other slow-growing extra-axial tumours, meningiomas can cause reactive arachnoid cysts of variable sizes in 5% of the cases, particularly in tumors involving the basal CSF cisterns, [27] and these may contribute to the mass effect. Some meningiomas show central cystic degeneration (Figure 3) or have an associated cyst that can mimic schwannomas or intra-axial tumours.[28]

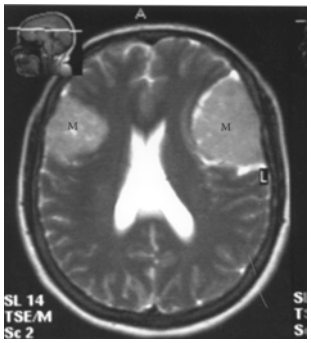

Fig 2 A.

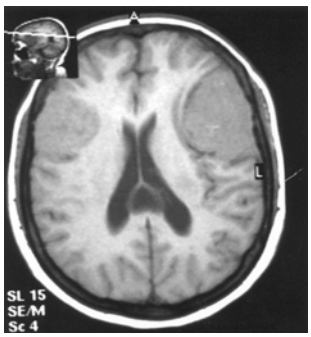

Fig 2 B.

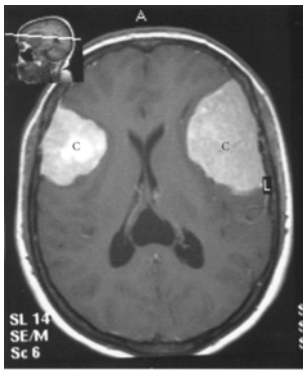

Fig 2 C.

Fig 2: Bilateral symmetrical parietal meningioma (M) seen in axial MR Images appears in T2 (A) hyperintense and isointense in T1 (B) while showing strong enhancement (C) after contrast injection.

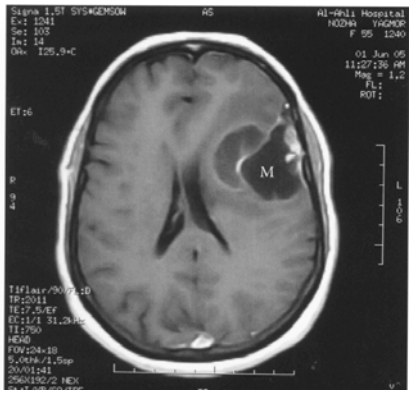

Fig 3: Cystic meningioma(M) of Lt parietal region seen in axial T1 contrasted MRI.

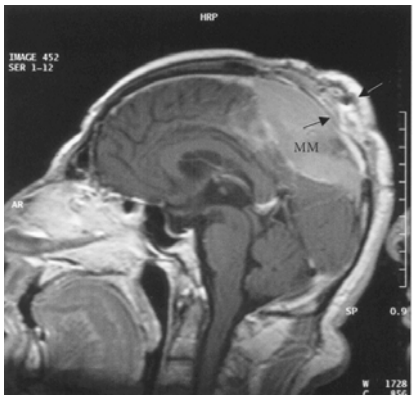

Fig 4 A.

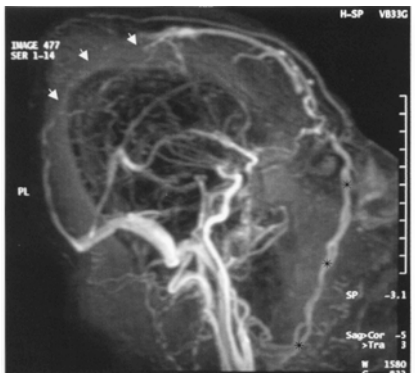

Fig 4 B.

Fig 4: Malignant parasagittal meningioma (MM) seen in T1 sagittal contrasted MRI(A) while the MRV (B) is showing invasion of the skull vault scalp(Arrows) and occlusion of superior sagittal sinus(Arrow heads) and collateral circulation(stars).

MR Spectroscopy: Although diagnosis of meningioma is usually straight-forward in MRI images, proton MR Spectroscopy may be useful in the diagnosis of difficult cases. The most common proton spectrum found in typical meningiomas is high choline peak (up 300 times of normal) with low or absent N-acetylaspartate (NAA), creatine and phosphocreatine (Cr) and variable amounts of lactate. Most important is an unusually high ratio of alanine (Ala) to creatine because of high Ala and low Cr content and this is a relatively specific finding for meningiomas. [42, 43] Atypical and malignant meningiomas may show resonance in the location of NAA, and differentiating them from astrocytomas may prove difficult; [43] but meningioma was the only tumor that showed higher sensitivity and specificity at MRS with long TE (≥ 130 ms) rather than short TE (≤30 ms) which was better for intraxial tumors. [44]

Angiography is useful in delineating the blood supply of the external versus internal carotid arteries and can show encasement of intracranial vessels. It is useful to establish patency of major dural sinuses, to facilitate preoperative embolisation and to confirm diagnosis by the prolonged homogeneous tumor blush. Meningiomas characteristically have external carotid artery feeders. Classic findings include: 1) Enlarged and tortuous afferent vessels (from external carotid). 2) Abnormal arborisation of the afferent arteries.[45] 3) Sunburst appearance of the arteries at hilus, which is the tumor's site of origin. 4) Cork screw appearance of the small arteries in the interstices of the lesion. 5) Usually normal circulation time. 6) Dense tumor capillary blush in the late venous phase.[46] 7) Mother-in-law blush, i.e comes early and leaves late.[47]

Usually meningiomas don't exhibit drainage veins, but angioblastic types can display it. MR Arteriography well documents those arteries encased by parasellar and cavernous sinus meningiomas to have a reduced diameter but remain hypointense in all imaging sequences.

MRI- Venography, especially when using contrast enhanced 3D turbo-flash MRA, [48] is an extremely useful non-invasive means of demonstrating patency, narrowing or occlusion of major vessels (accuracy in predicting sinus involvement is about 90%)[49], which helps in the selection of appropriate surgical management. Venous sinus invasion is most common in parasagittal tumors (Figure 4B).

Nuclear Medicine: Early somatostatin receptor scintigraphic studies have shown repeatedly, in vitro and invivo, that meningiomas, regardless of their histologic grading, express human somatostatin receptor with a sensitivity of about 100%. Therefore, somatostatin receptor scintigraphy (^{111}In-DTPA octreotide) has been suggested for the preoperative differentiation of meningiomas from neurinomas, which show a predilection for similar sites and do not express somatostatin receptors. This exclusion procedure became questionable with reports of histologically proven meningiomas and negative scintigraphic studies.[25] These false negative scintigraphic studies were attributed primarily to the presence of a more-or-less intact blood-tumor barrier especially in small tumors. Recently, a somatostatin receptor analogue (DOTA-TOC) has been labeled with a positron emitter ^{68}Ga, this allows for the detection of small tumors due to increased spatial resolution of PET.[50] In contrast to ^{18} FDG,^{68} Ga-DOTA-TOC shows high meningiomas-to-background ratio and provides valuable additional information on the extent of meningioms beneath osseous structures, especially at the base of the skull.[50]

Clinically, somatostatin receptor imaging techniques may be applied to improve the characterization of skull-base tumors when MRI findings are unclear and the tumor is to be treated by radiosurgery alone, or when streotactic biopsy is risky.[50] Furthermore, the intraoperative radiodetection of somatostatin receptors with hand held gamma probe may be helpful to guide the surgical removal of bone invasive en plaque meningioma[51] and scintigraphy has significant impact in post radio surgical follow-up.[52]

Intraoperative Ultrasonography will show hemorrhage, cystic changes or calcification within or outside the tumor. It can also show tumor outline and brain parenchymal invasion in cases of malignant meningiomas, while Duplex ultrasound will show the degree of tumor vascularity.

Management

The management of a meningioma depends on the signs and symptoms it produces, the age of the patient, and the site and size of the tumour.[53,54] A small incidental meningioma that is discovered in a patient who is undergoing neuroradiological investigations for other reasons can be safely managed conservatively,[55,56] especially if the patient is elderly or has a medical disorder that would increase the potential morbidity of surgical excision.[57] If the lesion is calcified on CT or hypointense on T2-weighted MRI, it is likely to remain asymptomatic.[55]

Medical care has been disappointing. It is restricted either to perioperative drugs or to medications that are used after all other means of treatment have failed. The use of corticosteroids preoperatively and postoperatively has significantly decreased the mortality and morbidity rates associated with surgical resection. Antiepileptic drugs should be started preoperatively in supratentorial surgery and continued postoperatively for no less than 3 months. The current experience with chemotherapy is disappointing. This modality of treatment is reserved for malignant cases after failure of surgery and radiotherapy to control the disease.

With recent advances in design of interventional neuroradiology catheters and microvascular techniques, endovascular therapy has increased substantially. [58] Selective microcatheter embolisation of the meningeal arterial supply can be achieved with several different agents, including glue, coils and small particles (150-300μ) of polyvinyl alcohol.

These can be highly effective at devascularising the tumour, and preoperative embolisation reduces peroperative blood loss [59] especially those with a complex presentation, giant meningiomas, malignant or angioblastic character, or those involving skull base, scalp or critical vascular structures. Only 2% of patients have complications that result in neurological deficit, but it may be the only treatment required or possible in older or high risk patients, in addition, it may reduce the likelihood of recurrence. [60]

Surgical excision of the tumor and its dural base is the most common primary management. The constant principles in meningioma resection are the following: If possible, all involved or hyperostotic bone should be removed. The dura involved by the tumor as well as a dural rim that is free from the tumor should be resected (duraplasty is performed). Dural tails that are apparent on MRI are best removed, even though some of them may not be involved in the tumor. A provision is made for harvesting a suitable dural substitute (pericranium or fascia lata). The surgeon can also use commercially available dural substitutes. In general, postoperative results are better in patients with few concomitant diseases, smaller meningiomas, less edema, short surgery times, and a more accessible location (ie; convexity rather than skull base). [60] Simpson [61] described the recurrence rates of meningiomas after surgical excision. He proposed a grading system based on the degree of surgical excision. A grade 1 excision involved the removal of the tumor bulk, its surrounding dural attachment, and any involved bone; grade 2 excision was the removal of the tumor with diathermy of its dural attachment; grade 3 removal was a macroscopic tumor resection with small foci left in situ (eg, in a major venous sinus); grade 4 was an extended biopsy with macroscopic residual disease; and grade 5 was a decompression with or without biopsy. The recurrence rate at 5 years was 9% for grade 1 excision; 19% for grade 2 excision; and 29% for grade 3 excision. The rate of meningioma recurrence increased when the follow-up period was extended.

Even after Simpson grade 1 resection, recurrence rates of 20% at 10 years have been reported.[62] Although a total excision (Simpson grade 1) is the ideal goal, many tumours cannot be totally excised because they are enveloping vital neural or vascular structures or are en plaque.[54, 61]

Significant factors contributing to recurrence include the following: 1) Incomplete surgical resection (Simpson classification). 2) Atypical and malignant histologic types (WHO classification). 3) Presence of nucleolar prominence. 3) Presence of more than 2 mitoses per 10 high-power fields. 4) Heterogenous tumor contrast enhancement on CT scan. [60]

Patients without any of these features showed low recurrence rates of 4% and 18% at 5 and 10 years, respectively, while among patients older than 70 years who underwent surgery for meningiomas, the neurologic complication rate reached approximately 23%, and was approximately 3% in younger patients. [60] Although most meningiomas grow slowly and have a low mitotic rate, clinical benefit has been reported in many case series with either tumor regression or stasis after radiotherapy; however, these results have not been confirmed in randomized trials. Radiotherapy is mainly used as adjuvant therapy for incompletely resected, high grade and/or recurrent tumors. It can also be used as primary treatment in some cases (optic nerve meningiomas and some unresectable tumors).[63- 68]

Stereotactic radiosurgery has been shown to provide excellent local tumor control with minimal toxicity. It is mainly used for small (<3 cm in diameter) residual or recurrent lesions when surgery is considered to carry a significantly high risk morbidity. It has been advocated as an effective management strategy for small meningiomas and for meningiomas involving the skull base or the cavernous sinus. It is used primarily to prevent tumor progression.[69,70.] Factors that may be predictive of a high post- operative morbidity rate include:

1) Patient-related factors (advanced age, comorbid states as diabetes and coronary artery disease, pre-operative neurological status).
2) Tumor factors (location, size, consistency, vascularity, vascular or neural involvement).
3) Previous surgery or radiation therapy. [47]

Despite advances in imaging, interventional neuroradiology, neuropathology, microsurgery, and radiotherapy, many meningiomas remain a challenging clinical problem that is increasingly being managed by a multidisciplinary team approach.[53, 54] Patients who undergo operation for meningioma should receive regular follow-up with enhanced MRI to check for possible recurrences. Patients whose meningiomas are completely resected usually have an excellent prognosis. The types of meningioma that are most likely to recur are: incompletely excised, malignant, or multiple tumors.

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الورم السحائي داخل الجمجمة

عزمي الحديدي، وليد المعاني، وليد محافظة، محاسن النجار، مصطفى النادي، قسم الأشعة، مستشفى الجامعة الأردنية؛ قسم جراحة الدماغ والأعصاب، مستشفى الجامعة الأردنية، عمان، الأردن.

الملخص:

يحتل الورم السحائي داخل الجمجمة المرتبة الأولى في قائمة الأورام الدماغية الحميدة؛ إذ يشكل نسبة 15- 30 % من كل الأورام الدماغية الأولية، وعادة يتم تشخيصه في عمر متوسط بين 40- 60 سنة، وهو يسود أكثر لدى النساء ولكن ذلك لا ينطبق على النوع الخبيث منه الذي وجد بنسبة أكبر لدى الرجال والأطفال. وقد تم تقسيم الورم السحائي نسيجيا إلى ثلاث درجات هي:

1- الحميد بنسبة 90%

2- اللانمطي بنسبة 5-7%

3-الكشمي بنسبة 3-5%

وتزيد احتمالية الرجوع و التصرف العدواني في النوعين الأخيرين.

يظهر الورم السحائي عادة بشكل كروي منتظم الحواف وملتصق بالأم الجافية، غير أن هناك نوعاً خاصاً منه يسمى باللوحي ويتخذ شكل منحنيات الدماغ وتجويف الجمجمة. ولقد تمت دراسة الكيمياء النسيجية المناعية والنشاط البيولوجي لذلك الورم، الذي أظهر وجود المستضد الظهاري (EMA) في 80 % من الحالات، إضافة لوجود مستقبلات البروجستيرون في 57-67% من الحالات التي لها علاقة بدرجة نسيجية جيدة واحتمالية أقل للرجوع.

هناك عوامل مختلفة يعتقد أنها قد تؤدي إلى ظهور ذلك الورم السحائي مثل الرضخ، والفيروسات، والتعرض للإشعاع خاصة بجرعات عالية، والعوامل الوراثية.

تتعدد الأعراض والإشارات التي يسببها ذلك الورم، وقد تتفاقم خلال الحمل، وبعض هذه الأعراض له علاقة وثيقة بموقعه داخل الجمجمة. ويتم تشخيص الورم السحائي شعاعيا باستخدام الصور الطبقية والرنين المغناطيسي، إضافة إلى الصور النووية والتنظير الطيفي المغناطيسي، وفي كثير من الحالات يضاف أيضا تصوير الأوعية الدموية بواسطة الرنين أو حتى التصوير التداخلي الملون. ولقد تفاوتت نتائج الدراسات التي تشير إلى علاقة نوع نسيج الورم السحائي بمظهره على صور الرنين المغناطيسي. أما بالنسبة لطرق العلاج فإنهاتعتمد على حجم وموقع الورم وعلى عمر المريض والأعراض الظاهرة ابتداء بالعلاج التحفظي وانتهاء بالعلاج الجراحي.

وهذا التقرير يتعرض بتفصيل لما ورد أعلاه مع مراجعة الأدب الطبي في ذلك.

الكلمات الدالة:

الورم السحائي، درجات الورم السحائي، مستقبلات البروجسترون، التشخيص الشعاعي.