

## Multiple Meningiomas: Case Report and Review of Literature

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## 1. Introduction

We present a case of a 63-year old man with multiple meningiomas, with no evidence of neurofibromatosis 2.

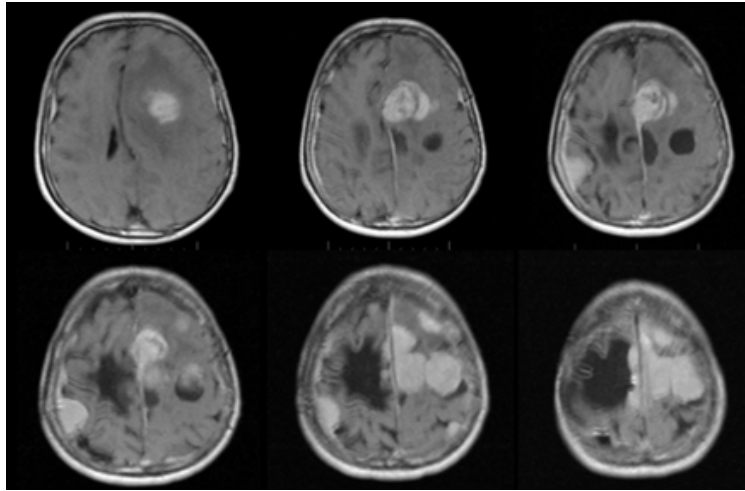
## 2. Case Presentation

A 63-year old man presented with complaints of a sudden left-sided weakness, propulsion, urinary incontinence. He had a history of craniotomy for right-sided parasagittal meningioma 18 years ago. After the surgery the patient developed left-sided spastic hemiparesis, which partially resolved in six months. After operation the patient developed also focal epileptic seizures twice a month. In May 2011 he noticed progressive worsening of his symptoms and 20 days before admission suddenly developed right-sided hemiparesis, which gradually resolved.

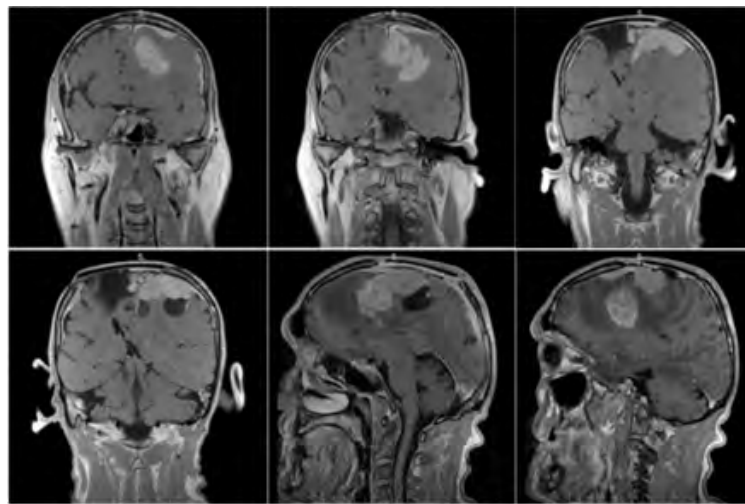
MRI of the brain was obtained which revealed 12 meningiomas. All of them had supratentorial localization and one falcine meningioma appeared to have intratumoral hemorrhage (Figure 1, 2). MRI of the whole spine was the obtained which revealed another

nidus on the level of C4 predominantly on the left side which was consistent with meningioma versus schwannoma (Figure 3).

On examination, the patient was awake, alert and oriented. However, he was a little depressive. He didn't have any meningeal symptoms and signs. Cranial nerves were WNL. He had left-sided hemiparesis. The muscle force on the right side was 4/5, on the left side 3/5. Left-sided Babinski sign. Patient had hyperostosis on the right temporal region and postoperative scar on the central parietal region. The patient had no evidence of neurofibromatosis 2. In our clinic patient received dexamethasone 5mg TID for 10 days, after that we performed the operation: left parietal craniectomy for removal of left parasagittal and falcine meningiomas. On first and seventh post-op days head CT scan was done (Figure 4, 5). 14 days after the operation patient was discharged from the clinic with slight spastic tetraparesis. The histological examination of the removed meningiomas showed, that all meningiomas were fibroplastic (WHO grade I).



**Figure 1:**



**Figure 2:**



**Figure 3:**

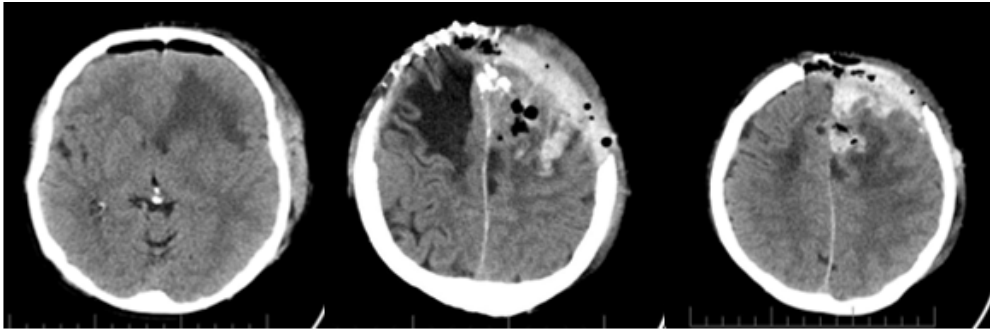


Figure 4: POST OP Day 1

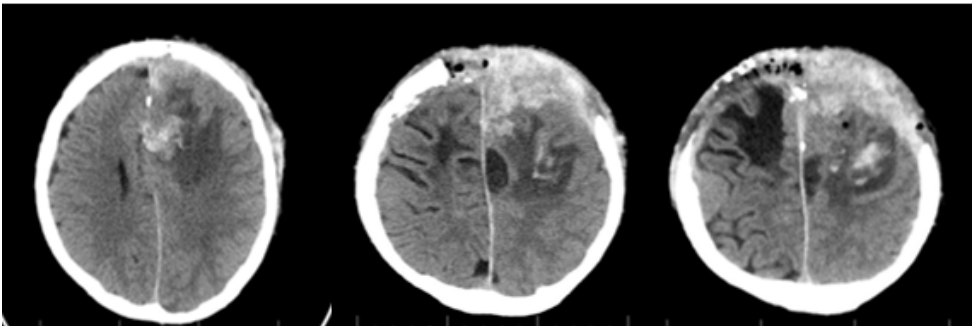


Figure 5: POST OP DAY 7

### 3. Discussion

Meningiomas are the most common, non-glioma, primitive intracranial tumors, their prevalence among operated tumors is around 13-19%. They may occur at any age but have a peak incidence around 45 years of age; 60% occur in females. Meningiomas may occasionally have an atypical appearance and atypical enhancement pattern secondary to necrosis, scarring, previous hemorrhage, or fat deposition [1].

Meningiomas accounts for approximately 34% of primary brain tumors (tumors that start in the brain) in the United States and occurs in approximately six of every 100,000 people. Meningiomas are rare in children. The overall five-year survival rate (the percentage of people who survive at least five years after the meningioma is detected, excluding those who die from other diseases) for meningioma is 69% (70% for benign and 55% for malignant). Symptoms of meningioma can be general (caused by the pressure of the tumor on the brain or spinal cord), or specific (caused by the tumor stopping the normal functioning of a specific part of the brain or by pressure on nerves or blood vessels). Generally, meningioma is not diagnosed until symptoms begin [2]. The incidence of multiple meningiomas has increased since the introduction of computed tomographic (CT) scan and a high incidence of up to 8.0% has been reported (2). Meningioma is classified into subtypes based on the location of the tumor:

- Falx and parasagittal meningioma (accounts for 25% of meningiomas).
- Convexity meningioma (20%).
- Sphenoid wing meningioma (20%).
- Olfactory groove meningioma (10%).

- Posterior fossa meningioma (10%).
- Suprasellar meningioma (10%).
- Spinal meningioma (less than 10%).
- Intraorbital meningioma (less than 10%).
- Intraventricular meningioma (2%) [2].

Meningiomas are classified according to the World Health Organization (WHO) schema, which is based upon morphologic criteria. The 2000 and 2007 versions of the WHO classification system divides meningiomas into three groups [8]

- WHO grade I – Benign meningiomas (WHO grade I) are subdivided into a number of subtypes. WHO grade I meningiomas do not meet any of the criteria for a higher grade lesion based upon morphologic criteria. The treatment approach is the same for all of the subtypes of benign meningiomas.
- WHO grade II – WHO grade II meningiomas have increased mitotic activity ( $\geq 4$  mitoses per ten high powered fields) and three or more of the following features: increased cellularity, small cells with a high nuclear cytoplasmic ratio, prominent nucleoli uninterrupted patternless or sheet-like growth, or foci of spontaneous or geographic necrosis. Chordoid, clear cell, and atypical meningiomas are classified as WHO grade II.
- WHO grade III – WHO grade III meningiomas have  $\geq 20$  mitoses per ten high powered fields and/or malignant characteristics resembling carcinoma, sarcoma, or melanoma. Features that support the diagnosis of malignant meningiomas are:

nant meningioma include the loss of usual meningioma growth patterns, infiltration of underlying brain, abundant mitoses with atypical forms, and multifocal microscopic foci of necrosis. Papillary, rhabdoid, and anaplastic meningiomas are classified as WHO grade III.

It is used also the Simpson classification [9].

Grade I- This is a macroscopically complete removal of the tumor, with excision of its dural attachment, and of any abnormal bone. Where the tumor arises from the wall of a dural venous sinus, such an operation necessarily entails resection of the sinus.

Grade II -This denotes a macroscopically complete removal of the tumor and of its visible extensions, with endothermy coagulation (usually to the point of charring) of its dural attachment.

Grade III -This denotes a macroscopically complete removal of the intradural tumor, without resection or coagulation of its dural attachment, or alternatively, of its extradural extensions, e.g., an invaded sinus or hyperostotic bone.

Grade IV -This denotes a partial removal, leaving intradural tumor in situ.

Grade V -This is a simple decompression, with or without biopsy.

Multiple meningiomas mostly consist of benign tumors, in which a combination of differential histological types of meningioma is observed in approximately 30% of cases. However, the simultaneous occurrence of benign and anaplastic histological types is extremely rare [3]. In 1938 Cushing applied the term of multiple meningiomas to a condition in which a patient has more than one meningioma with different localizations. Confluent ‘en plaque’ meningiomas are usually reported as diffuse meningiomatosis which is generally considered an extreme form of multiple meningioma [5]. There are 2 distinct hypotheses for the occurrence of multiple meningiomas. The first suggests that tumors arise independently and this is supported by histological and cytogenetic examinations that have revealed microscopic and karyotypic differences in multiple tumors from the same patient. Another hypothesis suggests that a single transforming event occurs and the original clone of cells spreads throughout the meninges resulting in the formation of multiple, clonally related tumors [3]. Primary tumors are deemed to be multiple when they occur wholly separate from each other in different parts of the central nervous system. They can develop at the same time or independently [4]. Sometimes, despite initial treatment, the meningioma may not go into remission (the temporary or permanent disappearance of a tumor) or it recurs (comes back after treatment) [2].

It divides recurrent meningiomas into:

1) True or local recurrences when the new growth is either within the limits of the insertion area of the previously resected meningioma or, if outside this zone, in direct continuity with (in practice, the recurrent tumor is usually within the limits of the previous craniotomy) and

2) False or regional recurrences, when the new growth is contiguous to but independent of the attachment surface of the primary meningioma and outside the previous craniotomy site. Those regional recurrences should be considered as new primary lesions originating from the multicentric tumor foci in the contiguous dura mater and the fibrous fringe. This pathological surrounding dura mater should be considered as part of attachment surface of ‘solitary’ meningiomas [6]. The distinction is important because recurrence and regrowth represent two separate phenomena. The diagnosis of recurrence was based on computed tomography or magnetic resonance imaging findings [7] A number of factors have been studied for a possible relationship to the development of meningiomas and other brain tumors. They are Ionizing radiation, radiation therapy, genetic factors: neurofibromatosis type 2, hormonal factors, breast cancer, head trauma, cell phone use). Multiple meningiomas can be associated with neurofibromatosis 2 [5]. Neurofibromatosis type 2 (NF2) is an autosomal dominant disorder predisposing to multiple neoplastic lesion. This disorder is due to a mutation in the NF2 gene, a tumor suppressor gene on chromosome 22 which encodes a membrane cytoskeletal protein called merlin or schwannomin that appears to be involved in actin-cytoskeleton organization [8].

Treating brain and spinal cord tumors can be challenging. Surgery is the most common type of treatment, but it can be difficult if the tumor is near a delicate portion of the brain or spinal cord. The blood-brain barrier, which normally serves to protect the brain and spinal cord from damaging chemicals, also keeps out many types of chemotherapy. Meningioma grows outside the blood-brain barrier, so some drugs do reach these tumors; however, they are very resistant to currently available chemotherapy [2]. Surgery is the removal of the tumor and surrounding tissue during an operation. For meningioma, it is the most common type of treatment and is often the only treatment needed for benign tumors that are able to be completely removed by surgery [2]. It can use radiation therapy and chemotherapy. The goal of chemotherapy can be to destroy any tumor remaining after surgery, slow the tumor’s growth, or reduce symptoms. However, chemotherapy is rarely used to treat meningioma, although researchers are studying this form of treatment. It is also important to keep in mind that a treatment plan may change over time if it is no longer working. Most patients with a brain tumor will be prescribed steroids to help relieve swelling of the brain. Steroids occur naturally in the body in tiny amounts. In larger amounts, they are very powerful anti-inflammatories (drugs that help swelling). You will most likely receive steroids when you

are first diagnosed, before and after surgery, before and after radiation therapy, and if you have an advanced brain tumor. Steroids have many side effects, which include weight gain and water retention, increased appetite, difficulty sleeping, changes in mood, and stomach irritation.

**Anticonvulsant medication.** A person with a CNS tumor may experience seizures, and this type of medication helps to control the frequency of them.

**Antidepressants.** Depression can be common in people with a CNS tumor, but it is often undiagnosed. However, this does not mean that all people with a CNS tumor are depressed. For those who have symptoms of depression, the health care team may decide to prescribe an anti-depressant to help with the symptoms [2].

#### 4. Conclusions

Multiple meningiomas consist more than one meningioma with different localizations. Multiple meningiomas mostly consist of benign tumors. The role for development of multiple meningiomas play radiation therapy, head trauma, hormonal factors, ionization, genetic factors, especially neurofibromatosis type 2. But in our case multiple meningiomas were recurrent meningiomas, development who didn't play the factors who have sign above. The patient didn't have any signs of neurofibromatosis.

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