

RECURRENT INTRACRANIAL MENINGIOMA

Essay

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ABSTRACT

Recurrence of intracranial meningiomas or tumor progression after surgical excision ranged between 10-32% of cases within 10 years.

Recurrence of intracranial meningioma is generally controlled by many factors including patient's age and sex, location and histopathologic type of the tumor, period of follow-up, extent of surgical excision and adjuvant therapy. The extent of surgical removal is an important factor affecting recurrence or progression of meningiomas. Those who had complete removal achieved the longest mean free-interval compared to those who had incomplete meningioma removal.

Key Word:

The Meninges, Etiology of Meningiomas, Embryology and development, Pathology of Meningiomas

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LIST OF ABBREVIATIONS

AgNORs	: Silver nuclear organizer regions
AR	: Androgen
BUDR	: Bromodeoxyuridine
CNS	: Central nervous system
CSF	: Cerebro-spinal fluid
CT	: Computerized tomography
DNA	: Deoxyribonucleic acid
EGF	: Epidermal growth factor
ETA	: Endothelin A
FGR-1	: Fibroblast growth factor receptor-1
IFN α	: Interferon alpha
IGF	: Insulin like growth factor
IL6	: Interleukin 6
LI	: Labeling index
PAS	: Periodic acid-Schiff
PCNA	: Proliferative cell nuclear antigen
PGF- β	: Platelet derived growth factor beta
PR	: Progesterone
RNA	: Ribonucleic acid
SAS	: Subarachnoid space
SS	: Somatostatin
TGF- β	: Transforming growth factor beta

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***Introduction and
Aim of the Work***

INTRODUCTION AND AIM OF THE WORK

Meningiomas are common tumors and account for 15% of all primary intracranial neoplasms (**Munshi et al., 2007**) which originate from meninges covering the brain, derived from arachnoid cap cells (**Rimenschneider et al., 2006**).

They are commonly detected in the fifth decade of life, and there is a clear predominance in women (**Caroll et al., 2000**).

The only definitive method for cure of meningiomas is complete surgical resection. The more complete the resection the less the chance for recurrence (**Youmans, 2004**).

The incidence of recurrence range from 10-32% even after complete removal (**Ildan et al., 2007**).

The most important factor in recurrence of meningiomas is postoperative tumor residual. In **1957, Simpson** introduced five-grade classification of surgical removal of meningiomas. Recurrence rates for grade I are about 10%, those for grade II are twice as high. The rate of recurrence are understandably higher in the higher Simpson grades (**Youmans, 2004**).

Palma and coworkers (1997) found that location was a significant predictor of recurrence when completeness of resection was not precisely controlled.

Nakasu and colleagues (1999) divided meningiomas into two groups, near major sinus and distant from major sinus, and found that the recurrence rate was different between the two groups (tumors in the near sinus group are more likely to recur than the distal sinus group).

Mushroom-shaped meningiomas were associated with significantly higher incidence of recurrence than those with smooth borders (**Alvarez et al., 1987**).

Factors cited as being associated with meningioma recurrence include:

- a. Incomplete surgical resection.
- b. Atypical and malignant histological types.
- c. Heterogeneous tumor contrast enhancement on CT scan.
- d. Presence of nucleolar prominence on microscopy.
- e. Presence of two or more mitoses per 10 high power field on microscopy (**Sekhar, 2001**).

Because the recurrence rate is invariably higher with atypical or malignant meningiomas, thorough follow up is essential. The earlier a recurrence is detected, the smaller the tumor size and the better the

chance that subsequent treatment will succeed. This is the case even with benign tumors when re-operation is the treatment of choice in case of recurrence, higher grade meningioma show increased glucose utilization and are noted on positron emission tomography scan. In this case reoperation followed by radiotherapy is performed (**Singh et al., 2000**).

Aim of the Work:

- To evaluate the extent of recurrence in different locations of the brain.
- To study the risk factor increasing the incidence of recurrence of meningiomas.
- To suggest a protocol for the best management.

Review of Literature

THE MENINGES

Embryology and Development of the Meninges

The meninges originate from cells of the neural crest and from cells that migrate into the area of the developing neural tube from the mesoderm. The precursors of the meninges are seen early in development and have reached their basic adult form by about the end of the first trimester. There are some differences in meningeal development between the brain and spinal cord and between different regions of the brain at various stages (**O'Rahilly and Moller, 2007**).

The neural folds along medial portions of the embryo have fused dorsally and the primitive neural tube is being formed. The initial fusion of the neural folds takes place at what will become the approximate junction of the spinal cord with the rhombencephalon: this closure proceeds in rostral and caudal directions. At this stage (22-24 days), usually single, layer of cells surrounds the developing neural tube. This cellular monolayer has some continuity with neural crest cells that are migrating ventrolaterally, it is likely that these cells originate, from the neural crest. A second collection of loosely arranged cells begins to form around the developing spinal cord and brain by about 24-28 days and basically surrounds the cord by 30-31 days (8-9 mm) and most of the brain by 41 days. This layer is thicker

than the cellular monolayer on the surface of the neural tube, and is composed of mesenchymal cells that originate from mesoderm. At this stage of differentiation is identifiable as the meninx primitive (primary meninx). Some cells of the primary meninx become closely associated with the layer of cells immediately surrounding the neural tube (**O'Rahilly and Moller, 2007**).

During the interval of about 37-44 days, the primary meninx around the brain continues to organize and the structures that will become the tentorium cerebelli and flax cerebri are forming mesenchymal condensations that will give rise to the cranial meninges to appear first around lateral aspects of the developing brain, then they are seen dorsally and ventrally. In this regards, there are some regional variations concerning when the cranial primary meninx appears and have its differentiation (**Haines and Frederickson, 1991**).

At stages about 44-48 days for the brain the primary meninx begins to organize itself into an outer portion that is more compactly arranged, around the brain, containing venous channels, and an inner portion that is loosely structured. The outer more condensed, layer represents the ectomeninx while the inner, more loosely organized part, plus the cells on the surface of the neural tube represent the endomeninx. A thin darkly staining cell layer (the dural limiting layer) is located at the interface of the outer compact region

(ectomeninx) and the inner more reticulated area (endomeninx). The dural limiting layer is first seen at about 40 days, initially located in ventral areas, and then appears into other regions as development progresses, it contributes to the ultimate formation of the dura and possibly the arachnoid (**O'Rahilly and Moller, 2007**).

In the interval of 45-55 days portion of the endomeninx (that portion of neural crest originated on the neural tube) begins to form a definitive cell layer that will become the pia. Vessels located adjacent to the neural surface are also covered by a thin layer of cells from the endomeninx. The outer part of the endomeninx becomes progressively more loosely arranged while the overlying ectomeninx becomes more compact. Cavitations appearing in the endomeninx at this time represent the primitive subarachnoid space (**Haines and Frederickson, 1991**).

By about 50 days enlarged areas of the subarachnoid space, form presumptive cisterns, and these are becoming obvious by 55 days. The ectomeninx is more compact in its appearance, contains venous channel that will form clearly recognizable dural sinuses by 55 days, and the ectomeninx is continuous with the cellular condensations that will form the skull around the brain. The skull will originate from the skeletogenous layer. The ectomeninx around the brain maintains a close apposition to the developing skull. Its inner portion, and probably the dural limiting layer, will condense of