

## Original Research Article

**To study the correlation of clinico –pathological and radiological finding in the atypical meningiomas- A retrospective study****Dr. Nilesh Potdar<sup>1</sup>, Dr. S Suresh Kumar<sup>2</sup>, Dr. Bhavadasan K<sup>3</sup>**<sup>1</sup>PG 3<sup>rd</sup> yr, Department of Neurosurgery, Amala Institute of Medical Sciences, Amala nagar, Thrissur, Kerala – 680555<sup>2</sup>Senior Consultant Neurosurgeon, Associate Professor & Unit Chief Department of Neurosurgery, Amala Institute of Medical Sciences, Amala nagar, Thrissur, Kerala - 680555<sup>3</sup>HOD And Professor, Senior Consultant Neurosurgeon, Professor & Unit Chief Department of Neurosurgery, Amala Institute of Medical Sciences, Amala nagar, Thrissur, Kerala - 680555**\*Corresponding author**

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**Abstract:** The aim is to study of neuro radiological finding of atypical meningiomas and correlation of intra operative finding and histopathological diagnosis and prognosis after surgery. Fourty eight cases of meningioma were treated surgically in our hospital between January 2014 and dec 2016. Eleven (22%) were histologically identified as aggressive variant of meningioma. Here study cases of aggressive variant of meningiomas operated, according to their neuro radiological finding and histopathological and intra operative findings .We found 11 cases of meningiomas found to be of atypical variety and aggressive in nature. Six of them were recurrent meningiomas. Bony erosion were found in five cases, three cases having heterogeneous contrast enhancement, four cases having brain invasion. HPR result showing, three cases having malignant meningiomas and six cases having atypical meningioma. CT and MRI gave useful information for the possible diagnosis of the atypical meningioma before surgery. Predicting histological nature meningioma would aid in surgical and treatment planning.

**Keywords:** Meningioma; atypical, brain invasion.

**INTRODUCTION**

Most meningiomas are benign and classified as grade I according to World Health Organization (WHO) standards [1]. Since malignant meningioma was first recognized by Cushing and Eisenhardt in 1938, [2] there have been diverse criteria for histopathologically grading atypical and anaplastic meningiomas. To improve this situation, the 2000 WHO classification recommends much more stringent and objective criteria [1, 2]. In the 2000 WHO classification, some important diagnostic variables were amended, particularly proliferation index, brain invasion and mitotic activity.

However, subtypes such as atypical, clear cell, chordoid, and malignant meningiomas display less favorable clinical outcomes and are classified as grades II and III [1, 3-5]. Atypical meningiomas account for between 4.7 and 7.2% of all meningiomas [1]. Malignant meningiomas are less common, comprising between 1.0 and 2.8% [1]. Malignant and atypical meningiomas are more prone to recurrence and rapid growth [1]. The distinction between benign and atypical or malignant meningioma represents important surgical information, because surgical and treatment planning as well as prognostication will depend on those pathologic types.

Recent studies into the cytogenetic alteration of meningioma have provided tools for understanding the mechanisms underlying malignant progression [6, 7]. These advances may be useful in improving our ability to predict clinical outcome and develop therapeutic strategies to improve outcomes in patients with high-grade meningiomas.

This study was primarily motivated by growing concerns about the validity of treatment based on both histological grading and radiological finding of meningiomas. To address this issue, by adopting the 2000 WHO criteria, we reclassified previous atypical and anaplastic meningiomas, reanalyzed their treatment outcomes and re-evaluated prognostic factors by clinicopathological and radiological aspects.

**MATERIALS AND METHODS**

Fourty eight cases of meningioma were treated surgically in our hospital between January 2014 and dec 2016. Out of 48 patient 32 (66%) female, 16 (34%) were male. Youngest patient age was 35 years and oldest was 83 years. Mean age presentation was 60 year; median age was around 55 years.

Eleven (22%) were histologically identified as aggressive variant of meningioma. The patients comprised 3 males and 8 females, ranging in age from 36 to 76 years (mean, 62.7 years). Neurological symptoms such as headache, loss of consciousness, and numbness of the extremities were reported. Duration of symptoms ranged from 1 month to 16 years, with a mean of 4 years 11.4 months. Six cases represented recurrent disease subsequent to resection of benign meningotheelial meningiomas. Computed tomography (CT) was performed before and after contrast administration in five cases. Magnetic resonance imaging (MRI) including pre- and post-contrast T1-weighted imaging using spin-echo (SE) sequences and T2-weighted imaging using fast spin-echo (FSE) sequences was performed in seven cases. On CT, attenuation of the tumor compared to normal gray matter, presence of calcification, pattern of contrast enhancement, and presence of bony changes were analyzed. On MRI, signal intensity of the tumor compared to normal gray matter, pattern of contrast enhancement, characteristics of tumor margin, and extent of surrounding edema were analyzed. Extent of edema was divided into three grades: -, not seen; +, smaller than the tumor; and 2 +, larger than the tumor.

For characteristics of the tumor margin, the CSF interface between the tumor and the brain surface, the ‘peri tumoral band’, shows a hypointense rim on T1W and a hyperintense rim on T2W. Presence of the peri tumoral band was evaluated. Presence of the dural tail sign with thickened enhancing dura extending from the tumor on post contrast T1W was also assessed.

**RESULTS**

Findings for all patients are summarized in Table. Five tumors were located in the falx, three in the convexity, two in the orbital region, and one of en plaque meningioma. Tumor size ranged from 25.0 to 85.0 mm in maximum diameter (mean, 51.4 mm). Out Of These 11 Cases, 6 Meningiomas represented as recurrent meningiomas two were found on medial sphenoid wing and at the Orbit. Three were found on the falx and parasagittal area. One was on convexity meningiomas (table 1).

Bony Erosion was found in 5 cases. One Case Has extra Cranial involvement, presented as extra cranial swelling. Dural tail sign was absent in one case. Brain edema was found in every case. Administration of contrast material shows heterogeneous enhancement was found in three patients, they were having malignant type of meningioma. During intra operative finding, we found four cases of meningiomas having brain invasion and recurrent meningiomas having more brain edema (Table 2)

Histopathological finding of these aggressive variant of meningiomas shows, three cases of them showing malignant meningiomas, six cases were showing grade 2 type of meningiomas, one were showing rhabdoid variant of meningiomas, and one case showing microcystic pattern of meningiomas, six cases showing recurrence of disease during the interval of the first and third year of the post-operative period (table2).

Of the three cases in which CT was utilized, tumor attenuation appeared heterogeneous in two (Fig. 1), although homogeneous hyper density was observed in one. Calcification was seen in one tumor (Fig. 1). One tumor homogeneously enhanced to the same degree as blood vessels in port contrast CT, and two were heterogeneously enhanced. Bony changes such as erosion and hyperostosis were seen on four tumors. On T1W MRI, tumors were homogeneously isointense in eight cases .Three tumors displayed heterogeneous signal intensity with hypo-, iso- and hyperintensity (Figs. 1 and 2). On T2W MRI, tumors were homogeneously hyperintense in two and isointense in one. One tumor appeared in homogeneously hyperintense (Fig. 2). Heterogeneous intensity was seen in three cases. Peri tumoral band was complete in one tumor. On post contrast T1W, all tumors were enhanced after administration of contrast material, with heterogeneous enhancement in three (Figs. 1 and 2) and homogeneous enhancement in eight. Eight of the eleven cases displayed edema larger than the tumor. No perifocal edema was seen in one case. Although, a peri tumoral band was only completely present in one case, it was partially apparent in some other tumors (Four). Post contrast T1W showed a dural tail sign not seen in only one case.

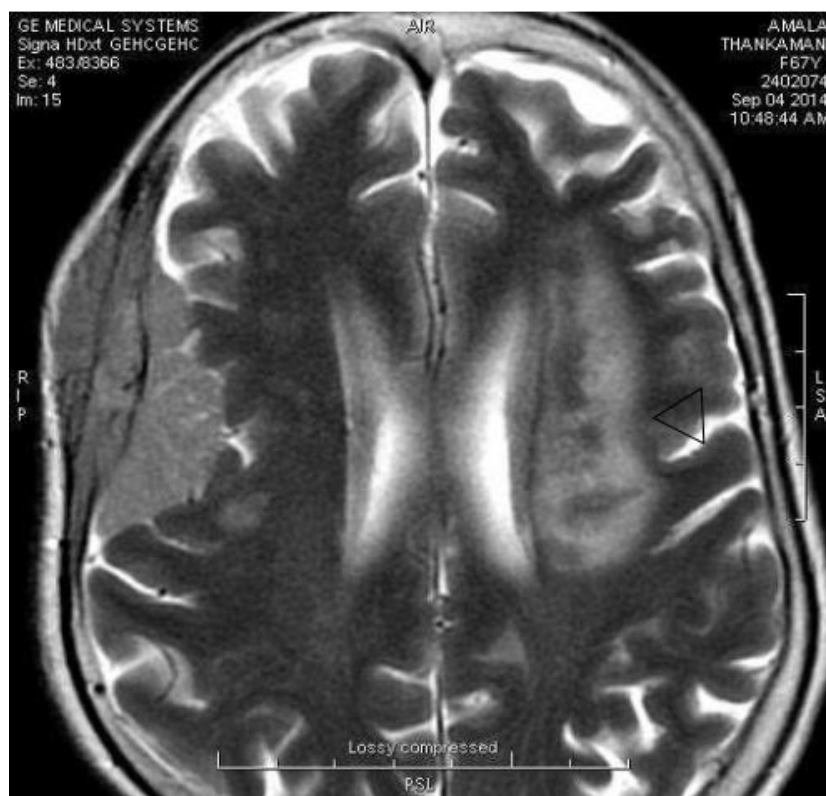
**Table 1: Showing CT and MRI finding**

Case	Age	Sex	CT		MRI		
			Attenuation	CE	INTENSITY T1W	T2W	CE
1	67	M	ND		HOMO ISO	HOMO ISO	HOMO ++
2	62	F	ND		HOMO ISO	HOMO ISO	HOMO ++
3	67	F	ND		HETERO ISO	HETERO ISO	HETERO ++
4	36	M	HOMO+	+	HOMO ISO	HOMO ISO	HOMO ++
5	40	F	ND		HOMO ISO	HOMO ISO	HOMO ++
6	67	F	ND	+	HOMO ISO	HOMO ISO	HOMO ++
7	76	M	HETRO	+	HETERO ISO	HETERO ISO	HETERO ++
8	48	M	ND		HOMO ISO	HOMO ISO	HOMO ++
9	53	F	ND		HETERO ISO	HETERO ISO	HETERO +
10	55	F	ND		HOMO ISO	HOMO ISO	HOMO +
11	59	F	HOMO +	+	ND	ND	ND

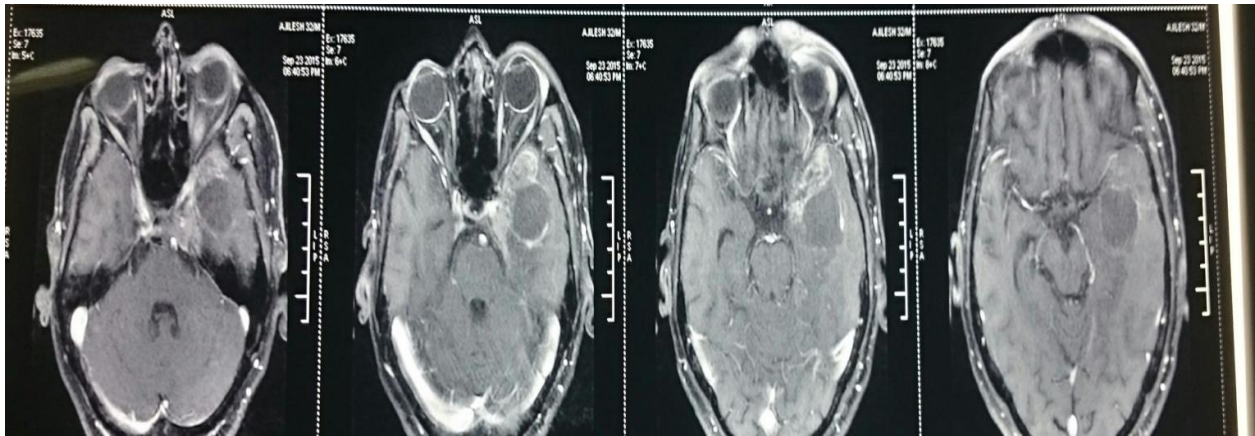
CE:-contrast enhancement

**Table 2:- Operative and HPR finding**

Case	Age	OPERATIVE FINDING		MRI FINDING			HPR	RECURRENT
		LOCATION	BONE EROSION	EDEMA	DURALTAIL	PERITUMOR BAND		
1	67	Convexity	-	+	+	+	Grade 2	No
2	62	Convexity	+	++	+	Incomplete	Rhabdoid	No
3	67	Orbital	++	++	+	Not seen	Malignant	Yes / 2 times
4	36	Temporal near cavernous sinus	-	+	-	-	Micro cystic	No
5	40	Falx	-	++	+	Incomplete	Grade 2	Yes
6	67	Convexity	Extra cranial	++	+	-	Grade 1	No
7	76	Falx	-	++	++	-	Grade 1	3
8	48	Falx	-	+	+	Complete	Malignant	No
9	53	Orbital/medical sphenoidal wing	++	++	+	-	Grade 2	1
10	55	Falx	++	++	+	Incomplete	Grade 2	3
11	59	falx	-	+	+	Incomplete	Grade 2	1



**Fig-1: MRI SCAN: In the right hemisphere a space occupying intracranial extra axial lesion buckling the right**



**Fig 2: MRI scan showing left temporal meningioma with cavernous sinus infiltration**



**Fig 3 MRI scan – temporal meningioma having bone erosion**



**Fig 4: MRI scan- sphenoid meningioma having orbital extension**

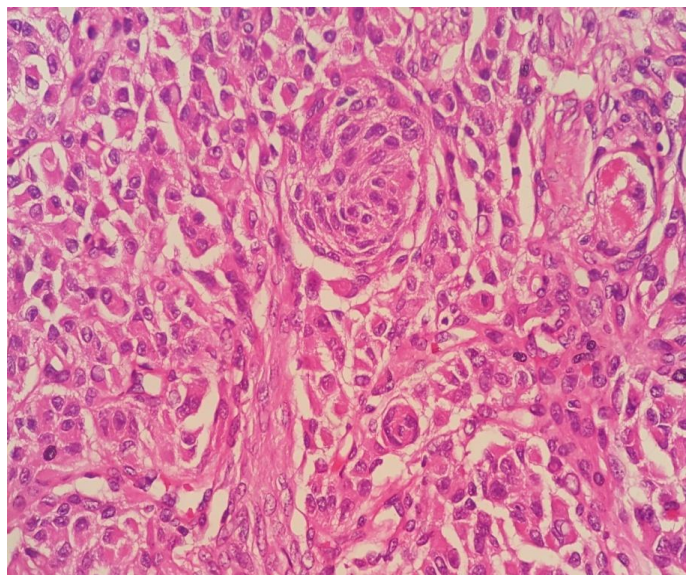


Fig 5: HPR E&H of atypical meningiomas (Rhabdoid meningioma)

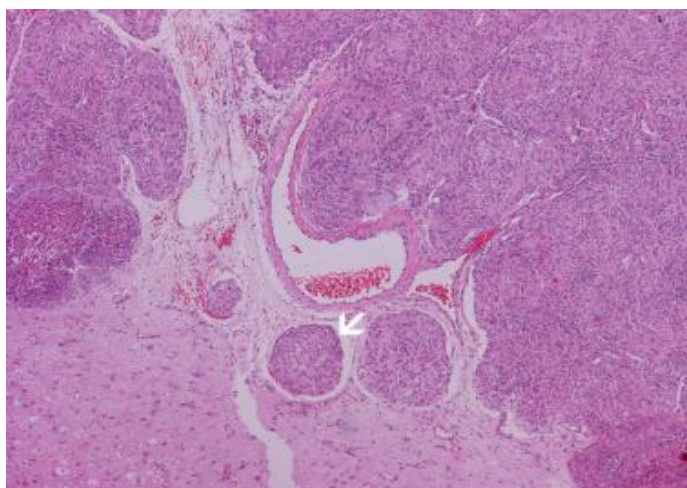


Fig 6: HPR showing atypical meningiomas with invasion

#### DISCUSSION:

The radiological diagnosis of meningioma is not difficult in the majority of cases. CT and MRI play important roles in the diagnosis of meningioma. Typically, meningiomas are sharply demarcated and hyperdense on CT. On MRI, the tumor is iso- or hypointense on non-contrast T1-W, and iso or hyperintense on T2-W. Homogeneous enhancement is observed after contrast administration. Unusual radiological findings are present in about 15% of all meningiomas and can include cystic, necrotic, or fatty changes [8]. Cystic components, which can be partially necrotic, were seen in four of the present cases. Fatty change was not seen. Only five of our cases radiologically displayed findings similar to benign meningioma. The other three tumors demonstrated

Non-homogeneous CT density or MRI intensity, in addition to heterogeneous contrast enhancement. Dural tail sign was seen in only one of these five tumors. Calcification was also found in only one of the present series. A previous report considered

the absence of calcification in malignant meningiomas [9]. The peri tumoral band represents the border between the tumor and the brain surface, and demonstrates the extra axial nature of the tumor [8]. A complete peri tumoral band was seen in only one tumor. Partial or complete disappearance of the peri tumoral band was seen in other tumors. Although, histological proof was not obtained, this finding is attributable to tumor invasion of the pia mater [10]. The amount of edema surrounding meningioma varies in the literature, but some reports [11, 12] have found no correlation between edema and histological type. However, amount of edema was relatively large in more than half of the tumors in the present series. Statistical analysis could not be performed due to the small number of patients in the present study.

Recently, the effectiveness of diffusion-weighted imaging (DWI) in differentiating malignant or highly atypical from benign meningiomas has been reported [13]. In that report, apparent diffusion coefficient (ADC) values were low in malignant or

atypical meningioma, but sample size was small. ADC could be effective in differentiating malignant or atypical from benign meningiomas, because ADC reflects degree of cellularity and amount of extracellular space in various tumors other than meningioma. DWI was not performed in the present series due to the retrospective study. The utility of DWI should be further studied.

The present study showed that partial or complete disappearance of the peri tumoral band was seen in a majority of tumors. More than half of the tumors exhibited lack of dural tail sign and a relatively large amount of perifocal edema. CT, MRI gave useful information for the possible diagnosis of the atypical meningiomas before surgery. Predicting histologic nature meningiomas would aid in surgical and treatment planning, because recurrence rate and prognosis in atypical meningiomas are different from those in benign meningiomas. If a meningioma radiologically has the above characteristics before surgery, surgeons may need to prepare to remove it as completely as possible.

Although a poor prognosis may be associated with a high MIB-1 labelling index, significant overlap exists in the MIB-1 labelling ranges for benign, atypical and anaplastic meningiomas [14]. Moreover, inter institutional and inter observer variation has been reported in meningioma grading [15]. Therefore, MIB-1 labelling cannot be a single parameter to establish meningioma grade in the 2000 WHO classification [16, 17]. Brain invasion has long been considered a worrisome feature in meningioma resection specimens, but it has been debated whether brain invasion constitutes a single criterion of malignancy. Recent molecular genetic investigations have failed to show genetic changes that are characteristic of non-benign meningiomas in histologically benign meningiomas that display brain invasion [18, 19]. Furthermore, the presence of brain invasion does not correlate with an aggressive course of anaplastic meningioma and only increases the likelihood of recurrence such as that of atypical meningioma, not anaplastic meningioma. Like some other studies [20, 21]. Our study revealed that the presence of brain invasion was a powerful predictor of reduced recurrence-free survival, but the worst prognosis had a close relationship with meningiomas with frank histological anaplasia, whether invasive or not.

In atypical meningioma surgery has been the primary treatment modality for meningiomas, regardless of subtype or grade. Similar to benign meningiomas, gross total resection of an atypical meningioma is associated with lower recurrence rates and increased survival than with subtotal resection [22]. Simpson Grade I, II resection without adjuvant radiotherapy might be sufficient to achieve durable local control and a longer survival period. Furthermore, adjuvant radiotherapy did not improve patient survival, regardless of the extent of

resection. However, this result should be interpreted with caution. Invasive meningiomas are often adherent or intertwined with cortical vessels and therefore more difficult to excise [23]. Moreover, microscopic brain invasion emerged as the most powerful predictor of reduced recurrence-free survival [17]. Adjuvant radiotherapy contributed significantly to improvement in overall survival and recurrence-free survival in the brain-invasive meningioma. Several studies have demonstrated that adjuvant radiotherapy improves overall and recurrence-free survival in atypical meningiomas following incomplete tumour resection [20–24]. Based on the above description, if the atypical meningioma is completely resected (Simpson Grade I, II) and does not reveal brain invasion, we do not recommend adjuvant radiotherapy. However, if the atypical meningioma was incompletely resected or showed brain invasion, adjuvant radiotherapy may be helpful for improved patient outcome.

#### CONCLUSION:-

CT and MRI gave useful information for the possible diagnosis of the atypical meningiomas before surgery. Predicting histologic nature meningiomas would aid in surgical and treatment planning, because recurrence rate and prognosis in atypical meningiomas are different from those in benign meningiomas. If a meningioma radiologically has the above characteristics before surgery, surgeons may need to prepare to remove it as completely as possible. Adjuvant radiotherapy may be helpful for improved patient outcome.

#### REFERENCES

1. Louis DN, Scheithauer BW, Budka H, Kepes JJ. In: Kleihues P, Cavenee WK, editors. World Health Organization Classification of Tumours. Pathology and Genetics. Tumours of the Nervous System. Lyon, France: IARC Press; 2000: 176–84.
2. Cushing H, Eisenhardt L. Meningiomas; their classification, regional behaviour, life history, and surgical end results. Leslie B. Adams for the Classics of Neurology & Neurosurgery Library; 1988.
3. Nowell PC. Tumor progression: a brief historical perspective. In: Seminars in cancer biology 2002 Aug 31 (Vol. 12, No. 4, pp. 261-266). Academic Press.
4. Al-Mefty O, Kadri PA, Pravdenkova S, Sawyer JR, Stangeby C, Husain M. Malignant progression in meningioma: documentation of a series and analysis of cytogenetic findings. *Journal of neurosurgery*. 2004 Aug; 101(2):210-8.
5. Simpson D. The recurrence of intracranial meningiomas after surgical treatment. *Journal of Neurology, Neurosurgery & Psychiatry*. 1957 Feb 1; 20(1):22-39.
6. Jääskeläinen J, Haltia M, Servo A. Atypical and anaplastic meningiomas: radiology, surgery, radiotherapy, and outcome. *Surgical neurology*. 1986 Mar 31; 25(3):233-42.

7. Perry A, Stafford SL, Scheithauer BW, Suman VJ, Lohse CM. Meningioma grading: an analysis of histologic parameters. *The American journal of surgical pathology*. 1997 Dec 1; 21(12):1455-65.
8. Ginsberg LE. Radiology of meningiomas. *J Neurol Oncol* 1996; 29: 229–38.
9. Servo A, Porras M, Jääskeläinen J, Paetau A, Haltia M. Computed tomography and angiography do not reliably discriminate malignant meningiomas from benign ones. *Neuroradiology*. 1990 Mar 1; 32(2):94-7.
10. Scheithauer BW, Perry A, Stafford SL, Lohse CM, Wollan PC. "Malignancy" In Meningiomas: A Clinicopathologic Study Of 116 Patients With Grading Implications. *Journal of Neuropathology & Experimental Neurology*. 1999 May 1; 58(5):524.
11. Abe T, Black PM, Ojemann RG, Hedley-White ET. Cerebral edema in intracranial meningiomas: evidence for local and diffuse patterns and factors associated with its occurrence. *Surgical neurology*. 1994 Dec 31; 42(6):471-5.
12. Tanaka Y, Matsuo M. Role of MR imaging in the differentiation of benign and nonbenign intracranial meningiomas: the utility of contrast-enhanced T1-weighted images. *Nihon Igaku Hoshasen Gakkai zasshi. Nippon acta radiologica*. 1996 Jan; 56(1):1-8.
13. Filippi CG, Edgar MA, Uluğ AM, Prowda JC, Heier LA, Zimmerman RD. Appearance of meningiomas on diffusion-weighted images: correlating diffusion constants with histopathologic findings. *American Journal of Neuroradiology*. 2001 Jan 1; 22(1):65-72.
14. Karamitopoulou E, Perentes E, Tolnay M, Probst A. Prognostic significance of MIB-1, p53, and bcl-2 immunoreactivity in meningiomas. *Human pathology*. 1998 Feb 1; 29(2):140-5.
15. Kleihues P, Louis DN, Scheithauer BW, et al. The WHO classification of tumors of the nervous system. *J Neuropathol Exp Neurol* 2002;61:215–25; discussion 26–9.
16. Amatya VJ, Takeshima Y, Sugiyama K, Kurisu K, Nishisaka T, Fukuhara T, Inai K. Immunohistochemical study of Ki-67 (MIB-1), p53 protein, p21 WAF1, and p27 KIP1 expression in benign, atypical, and anaplastic meningiomas. *Human pathology*. 2001 Sep 30; 32(9):970-5.
17. Perry A, Stafford SL, Scheithauer BW, Suman VJ, Lohse CM. The prognostic significance of MIB-1, p53, and DNA flow cytometry in completely resected primary meningiomas. *Cancer*. 1998 Jun 1; 82(11):2262-9.
18. Weber RG, Boström J, Wolter M, Baudis M, Collins VP, Reifenberger G, Lichter P. Analysis of genomic alterations in benign, atypical, and anaplastic meningiomas: toward a genetic model of meningioma progression. *Proceedings of the National Academy of Sciences*. 1997 Dec 23; 94(26):14719-24.
19. Simon M, von Deimling A, Larson JJ, Wellenreuther R, Kaskel P, Waha A, Warnick RE, Tew JM, Menon AG. Allelic losses on chromosomes 14, 10, and 1 in atypical and malignant meningiomas: a genetic model of meningioma progression. *Cancer research*. 1995 Oct 15; 55(20):4696-701.
20. Coke CC, Corn BW, Werner-Wasik M, Xie Y, Curran WJ. Atypical and malignant meningiomas: an outcome report of seventeen cases. *Journal of neuro-oncology*. 1998 Aug 1; 39(1):65-70.
21. Dziuk TW, Woo S, Butler EB, Thornby J, Grossman R, Dennis WS, Lu H, Carpenter LS, Chiu JK. Malignant meningioma: an indication for initial aggressive surgery and adjuvant radiotherapy. *Journal of neuro-oncology*. 1998 Apr 1; 37(2):177-88.
22. Modha A, Gutin PH. Diagnosis and treatment of atypical and anaplastic meningiomas: a review. *Neurosurgery*. 2005 Sep 1; 57(3):538-50.
23. Milosevic MF, Frost PJ, Laperriere NJ, Wong CS, Simpson WJ. Radiotherapy for atypical or malignant intracranial meningioma. *International Journal of Radiation Oncology Biology Physics*. 1996 Mar 1; 34(4):817-22.
24. Jääskeläinen J, Haltia M, Servo A. Atypical and anaplastic meningiomas: radiology, surgery, radiotherapy, and outcome. *Surgical neurology*. 1986 Mar 31; 25(3):233-42.