Intracranial Meningioma Surgery Outcome – the Impact of Preoperative Neuroimaging

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Abstract: The present study is aimed at finding radiological parameters which could provide indirect information on invasive growth of meningioma, relevant enough to predict the possible risk of postoperative neurological deficit development. The cohort was composed of 40 consecutive adult patients of comparable general condition parameters (age 18–75 years, KRS 70–100, ASA 1–2) with meningiomas attacking with the whole of their volume solely the brain tissue. As follows from the outcome, meningioma growth in the eloquent area and the presence of peritumoral oedema are the two adverse parameters predicting the development of postoperative neurological deficit. In contrast, dural type of vascularisation, visible tumour-brain interface, meningioma growing in a non-eloquent area and the absence of peritumoral oedema are favourable predictive parameters. According to our results, if the last two of those parameters are present, the patient need not to be exposed to the risks of invasive selective angiography.

Introduction

In the overwhelming majority of cases, meningiomas are benign tumours (WHO degree I) arising from arachnoid cells, growing extracerebrally and constituting about 20 % of all intracranial tumours. To prevent potential relapse radical surgical resection of meningioma is the optimal therapeutical method [1, 2, 3, 4]

There is general consensus on factors which affects the patient most: age, physical condition, site of meningioma, degree of its growth invasiveness, and the radicalism of resection [2, 5, 6, 7, 8, 9]. In terms of localisation and attainable degree of radicalism of the resection, intracranial meningiomas represent a heterogeneous group of tumours. Hence, the results of surgery for superficially localised meningiomas (convexity, falcine, parasagittal) can hardly be compared with those for skull base meningiomas (suprasellar, cavernous sinus, petroclival sites). This is mainly because in the latter group of meningiomas the radicalism of resection is limited beforehand, and any postoperative deficit is often caused by damage done to structures other than the brain tissue (cranial nerves and vessels). However, differences in the outcome of surgical treatment are found even in the group of meningiomas which, theoretically speaking, can be removed by radical resection (Simpson degree I–III) and whose expansion involves exclusively cerebral tissue. Brain tissue eloquence in the meningioma neighbourhood and invasiveness of the tumour growth are the main hazards of surgery there.

Our project presents a cohort of intracranial meningioma patients enrolled in a prospective longitudinal study who met the above listed criteria (radical resecability, involvement of brain tissue alone). The aim of the study was to identify, on the basis of a particular diagnostic algorithm, factors able to predict the risk of postoperative neurological deficit. It is important primarily for the patient, because without his consent the surgical operation cannot be undertaken. The diagnostic algorithm is made up of the following radiological parameters: MRI-defined localisation, the tumour surface – brain interface, the size of peritumoral oedema, oedema signal

intensity, and DSA-established type of the meningioma vascular supply (selective angiography of the ACI and ACE) [10, 11, 12, 13, 14].

Materials and methods

Patient cohort

40 consecutive patients (from 01/012004 to 31/05/2005) with MRI-diagnosed intracranial meningioma were enrolled in the non-randomised longitudinal study. Patients were included if the tumour was clinically symptomatic, the whole of its volume was supratentorially engulfed by brain tissue alone, and the tumour was, surgically speaking, fit for radical resection (Simpson degree I–III). In terms of their general condition, the enrolled patients were required to meet the following other criteria: age 18–75 years, ASA 1–2 [15], KRS ≥ 70 [16]. Other criteria for non-enrolment in the study were as follows: multiple meningiomas including those of spinal localisation, familial genetically defined meningioma-related pathological involvement, localisation in the posterior fossa and anywhere on the skull base relative to cranial nerves and vascular structures of the cranial base, relapsing meningioma, other CNS diseases, calcifications and cysts inside the meningioma, generally applicable contraindications of MRI scanning.

The cohort of 40 patients enrolled in the study included 28 women and 12 men aged 24 to 75 years (mean = 54). Their preoperative neurological picture was rated with the KPS score as follows: 70 2×, 80 9×, 90 11× and 100 18× ; their general physical health according to ASA: I 25× a II 15×. (Table 1).

The meningiomas under study exhibited the following clinical manifestations: epileptic seizure in 16 patients, headache in 14, mental impairment in six, hemiparesis in three, and hemianopsia in one patient. (Table 2)

Table 1 – Patients' characteristics

	No. (%)
Age (yr)	
Median	54
Range	24–75
Sex	
Female	28 (70%)
Male	12 (30%)
KPS score	
70	2 (5%)
80	9 (22.5%)
90	11 (27.5%)
100	18 (45%)
ASA class	
I	25 (62.5%)
II	15 (37.5%)

KPS: Karnofsky Performance Scale, ASA: American Society of Anesthesiology

Table 2 – Clinical symptoms

Symptom	No. (%)
Seizure	16 (40%)
Headache	14 (35%)
Mental impairment	6 (15%)
Hemiparesis	3 (7.5%)
Hemianopsia	1 (2.5%)

Radiological parameters (Table 3)

Magnetic Resonance Imaging
MRI scans of the brain were taken standard-wise on a Signa Exite II, 1,5 Tesla set: plain T1WI in the axial, coronary and sagittal planes, plain T2WI in all three planes. In addition, this standard imaging was supplemented with plain T1WI in the axial plane at 3.5 hours after the first post-contrast T1WI. MR imaging helped to establish the following radiological parameters: localisation, tumour-brain interface, peritumoral oedema size, and oedema signal intensity

Localisation
Out of 40 meningiomas in our cohort, 27 tumors were superficially localised (convexity, parasagittal, falcine), the remaining 13 (sphenoid wing lateral, sphenoid wing middle, frontobasal) were growing up from the skull base. 22 of the expanding tumours in both groups attacked the eloquent area of the brain (central area, speech centres, optic radiation and visual cortex).

Tumor-brain interface
Meningioma is an extracerebral tumour. If well circumscribed, it is separated from the brain tissue by an interface barrier made up of the arachnoid, subarachnoid space, pia mater and cerebral surface. Provided this barrier is complete all along the tumour-brain interface, T1WI will show it as a hypointense and T2WI as a hyperintense border discernibly separating the tumour from the surrounding brain.

Depending on the technique of imaging (T1WI, T2WI), the border signal is consistent with that of the cerebrospinal fluid [13, 14]. Out of the forty meningiomas, the border enveloping the whole tumour was identifiable in 11 cases

Table 3 – Radiological parameters

	No. (%)
Localization	
Convexity	16 (40%)
Parasagittal	8 (20%)
Falx	3 (7,5%)
Frontobasal	2 (5%)
Sphenoid wing lateral	7 (17,5%)
Sphenoid wing middle	4 (10%)
Eloquence	
Eloquent area	22 (55%)
Non-eloquent area	18 (45%)
Tumor-brain barrier	
Visible	11 (27,5%)
Non-visible	29 (72,5%)

	No. (%)
Peritumoral edema	
None	14 (35%)
Mild	10 (25%)
Moderate	10 (25%)
Severe	6 (15%)
Edema signal intensity	
Increased	24 (60%)
Non-increased	16 (40%)
Vascularization type	
Dural	19 (47.5%)
Pial+dural	21 (52.5%)

(27.5%). A close examination of the remaining 29 tumours (72.5%) failed to find a border or else found it poorly visible all over the tumour surface.

Peritumoral oedema

The oedema size can be expressed using the oedema index designed to compare the respective tumour and oedema volumes. The meningioma volume was calculated by means of the following equation for ellipsoid volume calculation:

$Vt = 4/3p \times a \times b \times c$ (with *a* and *b* as the longest meningioma diameters lying perpendicularly to one another in the axial plane, and *c* as the longest diameter in the coronary plane) as shown no the post-contrast T1WI; the same equation was used for oedema volume (Ve) calculation on T2WI. The oedema index (EI) was established using the equation $EI = (Ve+Vt)/Vt$. If $EI = 1$, no peritumoral oedema is present, $EI = 1–2$ means a mild, 2–3 moderate, and EI >3 severe oedema [10, 14]. Out of our 40 oedemas cases – 14 (35%) were free from oedema, 10 (25%) had mild, 10 (25%) had moderate, and 6 (15%) had severe oedema.

Oedema signal intensity

Meningiomas with peritumoral oedema were measured for their oedema signal intensity at a distance of 3 mm from the region of interest (ROI) using the Signal Profile method on T1WI performed at 3,5 ±0,4 hours after contrast medium application. A modification of the technique described in detail by Bitzer et al. was used for the purpose [11]. Increased values of oedema signal intensity were found in 24 patients (60%), not increased values in 16 of the group (40%).

Digital Subtraction Angiography

The meningioma vascular supply was measured by means of selective ACI and ACE angiography. The type of meningioma vascularisation was established according to the AP and lateral angiograms, using the semiquantitative method [12, 14]. 19 (47.5%) of the meningiomas were supplied solely from ACE branches (dural type), common brain and meningioma supply (pial + dural type) was seen in 21 (52.5%) cases.

Results

Out of the whole cohort, radical resection of the meningioma (SI–III) was attained in 33 (82.5%) patients, subtotal resection in seven (17.5%). The post-operative state was neurologically estimated at post-operative day 7, and then again at 3 months after the surgery. Ten (25%) patients at 7 days after the operation had their neurological findings worse than before. Seven were found to have a new neurological deficit (hemiparesis 3×, aphasia 3×, hemianopsia 1×), and there were three cases of progression of the existing neurological

symptoms (hemiparesis 2×, hemianopsia 1×). Three patients (7.5%) were worse off neurologically than before the operation as long as 3 months after surgery (hemiparesis, aphasia, hemianopsia) while seven had their neurological condition restored ad integrum. All of the ten patients with post-operatively worsened neurological findings had their meningiomas localised in the eloquent area. (Table 4)

Statistical analysis

The Pearson chi-square test was used for statistical evaluation. A correlation was found between the eloquent area and neurological deficit, and also between the presence of peritumoral oedema (small, medium, large) and neurological deficit. Some interdependence was detected between a discernible tumour-brain interface and absence of oedema, between a discernible tumour-brain interface and dural type of vascular supply, and between the dural type of vascularisation and absence of oedema. (Table 5)

Discussion

The factors of significance for the outcome of surgical treatment for intracranial meningioma are generally known and recognised; in particular: age, preoperative physical health, and severity of neurological deficit, meningioma site, and degree of resection radicalism attained [1, 2, 17]. At present, attention is centred on factors which can help predict the mode of meningioma expansion (circumscribed or invasive) and the resulting scope for extrapial or, on the contrary, need for subpial resection.

The method of resection is crucial for the outcome of the operation. While in extrapial exposure of the tumour capsule the brain remains intact, subpial resection is marked by the development of cortico-subcortical foci of minute ischaemias and bleeding, the main cause of any post-operative neurological deficit [18]. The pial

Table 4 – Operative data (according to eloquence)

Localization	Removal		Neurological deficit	
	S I-III	S IV	Day 7	3 Months
Eloquent area	15 (37.5%)	7 (17.5%)	10 (25%)	3 (7.5%)
Non-eloquent area	18 (45%)	–	–	–

Table 5 – Statistical analysis – results of Pearson Chi-Square Test

	Value	df	Asymp. Sig. (2-sided)
eloquence × neurological deficit	10,909	1	0,001
edema index × neurological deficit	9,524	3	0,023
brain-tumor barrier × edema index	28,177	3	0,000
brain-tumor barrier × vascularisation type	16,770	1	0,000
edema index × vascularisation type	24,628	3	0,000

df- degree of freedom

type of tumour vascularisation and the presence of peritumoral oedema, in particular, are currently seen as the main adverse radiological parameters predictive of an invasive growth and need for subpial resection of the meningioma [8, 18, 19, 20].

The very rigorous criteria for enrolment in our study enabled us to establish a homogeneous cohort. The results of surgical treatment were unaffected by the patients' general physical health or by the severity of preoperative neurological findings (KRS ?≥70, ASA 1–2) or by the meningioma expansion in intimate relation to cranial nerves or vascular structures (arteries, venous sinuses) on the skull base. All meningiomas attacked in their growth solely the brain tissue and, from the surgical point of view, all were fit for radical resection (SI–III).

Preoperative radiology entails a sum of parameters such as are currently studied relative to foretelling the meningioma expansion invasiveness and, consequently, to the actual outcome of surgical intervention [10, 12, 13, 14, 18, 19, 20]. The results in our cohort confirmed an interdependence involving the neurological deficit, the growth of meningioma in the eloquent area, and the presence of peritumoral oedema.

As for the other correlations found in our cohort, highly important is the absence of oedema in the presence of an MRI-visible interface, and the dural type of vascular supply seen on selective angiography. Similar relevance has the interdependence between an MRI-visualised tumour-brain interface and the dural type of vascularisation on selective AG.

As follows from our results, high-risk patients threatened with the need of subpial resection and consequently with the likelihood of a post-operative deficit are necessarily those whose meningioma is situated in the eloquent area and surrounded by peritumoral oedema. This outcome is confirmed by findings presented by the above listed authors. The interdependence between the dural supply of meningioma and the absence of oedema enables us to consider whether or not the currently used invasive examination – selective angiography – is indicated in patients with meningiomas expanding in a non-eloquent area in the absence of the peritumoral oedema. There were 14 (35%) such patients in our cohort, and none of them experienced any neurological deficit worsening in the wake of radical resection (SI–III).

Conclusion

As results of our prospective study suggest, meningioma expanding in the eloquent area and the presence of peritumoral oedema are adverse parameters predictive for the outcome. In contrast, favourable parameters include the dural type of supply, discernible tumour-brain interface and absence of peritumoral oedema. Our results justify the conclusion that patients with meningiomas in the non-eloquent area free from signs of peritumoral oedema need not be exposed to the hazards of invasive selective antiography.

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