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Professional paper

PARASAGITTAL MENINGIOMAS SYMPTOMS

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Abstract

The historical perspective of meningiomas begins in early times since 1614. Felix Plater, Professor Cushing in 1922 and Eisengardt 1938 proposed the term parasagittal meningiomas along with SSS. The purpose of this study is to familiarize ourselves with the experience of Neurosurgery service of Mother Theresa University Hospital TIRANA, by Prof. Mentor Petrela (PU - PH Paris) in the treatment of parasagittal meningiomas as well as our experience in the Tetovo Clinical Hospital although it is very short as well as some cases of meningiomas from the "8 September" General Hospital Skopje. This is a result of the collaborative work performed by the Neurosurgery, Neuroanesthesia - Resuscitation and Neuroradiology team at these three Clinics.

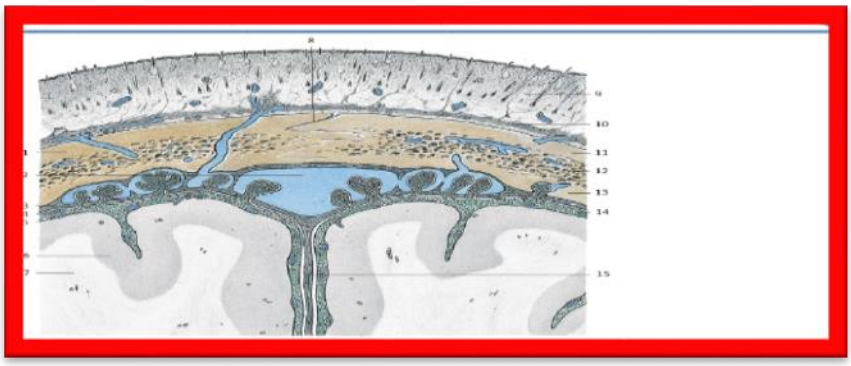
Keywords: Symptoms, tumor, craniotomy, teratment.

1. Purpose of The Study

The purpose of this study is to see the symptomatology of meningioma in its development from stages without clinical sings to symptomatology with deformity of the calvaria (1)

1.1. Historical retrospective: Meningioma is in many ways the soul of Neurosurgery, progress in the treatment of meningiomas reflects advances in Neurosurgery. These advances have been put to maximum use to improve the treatment of meningiomas. Meningiomas are female-dominated tumors and mainly affect middle age 90% of whom are benign, 6% are atypical, and only 2% are malignant, in most cases patients with meningioma diagnosis decide to surgically remove, and are advised to do so based on neurological symptoms (2). In most cases, complete removal of the tumor surgically results in a cure, when the meningioma is undetectable or when all other medications have failed (Surgery-Radiotherapy). Immunochemistry can be successful (3). Cushing proposed in 1922 the term parasagittal meningiomas together with SSS, these tumors originating from the arachnoid pockets that insert into the SSS and involving the convexity extending toward the flax and the wall (4).

1.2. Superior Sagittal Sinus (SSS) –Anatomy: The upper sagittal breast (sinus sagittalis superior) is the breast on which it is placed duplication of the upper convex lip of the falx cerebrum. This sinus extends, sulcus sinus superior sagittalis, from foramen cecum to protuberantia occipitalis interna, where flows into confluent sinuum, or less commonly into sinus transversus. The frontal and parietal part of the upper sagittal breast has lateral extensions (lacunae laterales), into which the granulationes are inserted arachnoidales of Arkanoid. In the superior sinus sagittalis, the following veins flow vv. superior cerebri, vv. diploicae, and mater veins. V. superior anastomotica connects this sinus with sinus cavernosus (5, 6).



1. Os parietale,
2. Sinus sagittalis superior,
3. Dura mater,
4. Arachnoidea mater,
5. Pia mater,
6. Cortex cerebri,
7. Substantia alba,
8. Sutura sagittalis,
9. Cutis,
10. Galea aponeurotica,
11. Calvaria, lamina externa,
12. Calvaria, diploica,
13. Calvaria, lamina terna,
14. Spatium suarchnoideum,
15. Falx cerebri,
16. Granulationes arachnoidales.

Figure 1. Front cut of calvary and meninges. Calvaria and meninges. Frontal incision

1.3. Meninges: Meninges are complex and highly complex brain and spinal cord covers, essentially consisting of three closely related layers: dura mater, arachnoidea and pia mater. The dura mater is the thick layer, it consists of the periosteal and meningeal site, the subdural spaces also serve as a barrier because the archnoidal cells extend throughout its extension, while the vascular wall system is essential because meningiomas receive vascularization from neighboring dural spaces, whereas the archnoidea is a thin layer but varies depending on the region, its thickness varies, and the pia mater is of varying thickness depending on the region, and its vascular system is virtually non-existent. The World Health Organization (World Health Organization) WHO has classified nervous system tumors in 2000 and classifies meningiomas as meningeal tumors. The classification describes three scales based on pathological criteria that present relapse risk or rapid growth (7).

Table 1-1 - WHO Grading of meningiomas 2007

Grade I
Meningothelial Meningioma
Fibroblastic Meningioma
Miksod Meningioma
Psamomatous Meningioma
Angimatous Meningioma
Microcytic Meningioma
Methaplasic Meningioma

Grade II
Atypical Meningioma
Clear cell Meningioma
Cordiod Meningioma

Grade III
Rhabdoid Meningioma
Papilar Meningioma
Anaplastic Meningioma
Non graded WHO Meningioma
Gaint cell Meningioma
Meningioma with intracytoplasmic eosinophilic involvement
Sclerosing meningioma
Oncocytic meningioma

Meningiomas are tumors of a globular and encapsulated nature. They attack the wall by exerting pressure on the brain tissue without infiltrating it, while also exerting pressure on neighboring structures. Although meningiomas also involve the dura or the dural sinuses, they can easily be prepared by the drink.
Meningiomas are female-dominated tumors and mainly affect middle age 90% of whom are benign, 6% -8% are atypical, and only 2% are malignant, in most cases patients with meningioma diagnosis decide to remove surgical and are advised to do so based on neurological symptoms.
AL-Mefty and co-workers have also documented that the tumor progresses irreversibly. The progress of TU is explained by the cloning theory which initiates the formation of TU by the cell, which carries mutants and which prioritizes the growth of new TU. The difference between malignant transformation and de novo new malignant tumor mass has been the subject of recent research, and this study has shown differences in clinical behavior of TU, hormonal receptor status, proliferative indexes, and cytogenetic profile between these two subgroups. tumor.

1.4. Epidemiology of meningiomas: The discussion about meningiomas affects two groups of patients: 1. Group of patients with TU, hospitalized and 2. Group of patients in a limited general population. A US study shows that the incidence of symptomatic meningiomas is about 2 cases per 100,000 population, and accounts for about 20% of primary intracranial TU. However, the true prevalence is greater than what we said above because studies performed on patients' autopsies showed a 2.3% higher rate because it was also found in people who developed undiagnosed asymptomatic meningioma.

1.5. Etiology of meningiomas:

1.5.1. Molecular etiology: Meningioma cells exhibit a striking resemblance to arachnoid coil cells (meningiomas), which are possible cells of tumor origin, although meningioma has a good pathophysiology in 95% of cases.

1.5.2. Risk factors :

Ionizing radiation
Hormones
Head trauma
Mobile phones
Breast cancer
Family connection

1.5.3. Genetic aspects of meningiomas: Genetic changes in meningiomas are known to be factors for tumor growth, and become more aggressive, in general, karyotypic abnormalities are more widespread in atypical and anaplastic meningiomas including multiple location changes between different chromosomes and multiple chromosomal monosomes, in addition to chromosome abnormalities. , which are among the first cytogenetic changes known as solid TU.

Table 1-2. A and B. Genetic model of tumor genesis of meningioma and progression to malignancy according to WHO-2007 (81).

Arachnoidal (meningothelial cell) or precursor cell		
Benign meningioma	Atypical meningioma	Anaplastic meningioma
−22q (40%–70%)	−1p (40%–75%)	−1p (70%–100%)
NF2 mutations (30%–60%)	−6q (30%)	−6q (50%)
Loss of 4.1B expression (20%–50%)	−10 (30%–40%)	−9p21 (60%–80%)
Loss of TSLC 1 expression (30%–50%)	−14q (40%–60%)	−10 (40%–70%)
PR expression (50%–90%)	−18q (40%)	−14q (60%–100%)
EGFR, PDGFRB activation	+1q, 9q, 12q, 15q, 17q, 20q (30%–50% each)	−18q (60%–70%)
	Loss of TSLC 1 expression (70%)	NDRG2 hypermethylation (70%)
	Loss of PR expression (60%–80%)	Loss of TSLC 1 expression (70%)
	Telomerase/hTERT activation (60%–95%)	Loss of PR expression (80%–90%)
	Notch, WNT, IGF, VEGF activation	17q23 amplification (40%)

1.5.4. Meningiomas and Receptors: Meningiomas can become symptomatic during pregnancy. Symptoms may occur during the proliferative phase of the menstrual cycle. It is not yet clear that these changes occur as a result of hormonal activity or vascular hypertrophy.

1.6. Image on CT and RM meningiomas: Meningiomas in large numbers display different stereotypes in their imaging features, which you combine with intracranial localization as well as dural adhesions, often becoming easily diagnosed without the need for other diagnostic methods that are often invasive. CT and RM are the most commonly used.

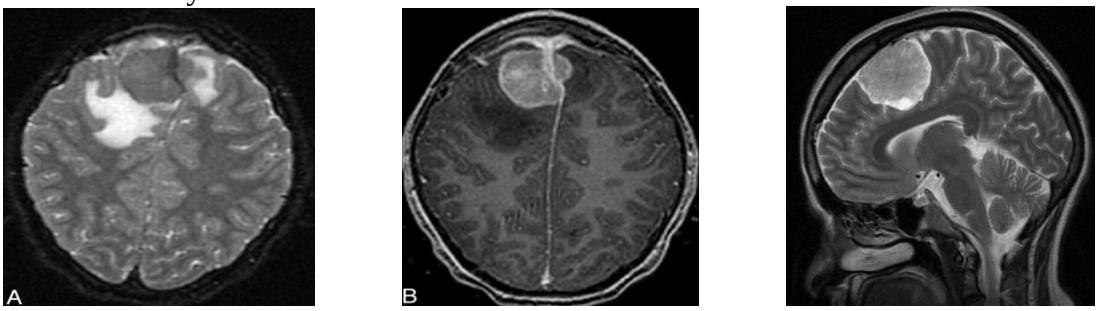


Figure 2. MRI imaging in parasagittal meningiomas.
a-Axial T2 b-MRI with contrast axial T1 MRI; c- FLAIR MRI and sagital T2.

Diagnostic features during RM and CT

HIPERDENSITY
CALCIFICATION
HIPEROSTOZIS
DURAL BEAST
ANGIOGRAPHICAL VASCULAR TEMPLATE (AVT)

1.6.1. General Anatomical Considerations (SSS): From the SSS cross-section, a triangular base is found that extends forward and backward, the SSS laterally communicating with irregular intradural cavities and veins, which extend to either side of the wall and are occasionally accompanied by arachnoid granulation. These structures often appear as intrasinusal defects. Tigliev studied collateral venous flow in 242 cases of parasagittal meningiomas, showing that 52.1% of TU had venous flow from the anterior 1/3 of the SSS

collateral, whereas collateral venous flow was 67% from the middle 1/3 or posterior, whereas 56% of cases had collateral venous flow (CFV) despite localization along the sinus. Oka also conducted a study, which identified that blood flow could also be through the meningeal veins and anastomoses of the superficial veins of the brain.

1.6.2. The natural course of meningiomas: In 1957 Simpson described SSS infiltration as a major factor associated with the recurrence of meningiomas. Since then it has been known that the rate of surgical extravasation and histopathologic classification of meningiomas correlates with the possibility of recurrence, although sometimes due to the inability to remove it radically, the need for some to remain undiagnosed may remain stable for a long time. although predictive factors remain to be determined in the future.

2. The Clinic

The presentation of clinical symptoms to parasagittal meningiomas is largely related to the proximity of the lesion to the Rolandic tribe as described by Cushing's case of General Leonard Wood. These patients usually present with sensory or motor neurologic deficits, including lower contralateral extremities, following the above neurological deficits, with more frequent secondary symptoms being paresthesia, papillae, and dementia disorders.

Presentation of symptoms in parasagittal meningiomas

1. Focal seizures
2. General convulsions
3. Headache
4. Confusion
5. Monoparesis of the lower extremities
6. Visual symptomatology
7. Calvary deformity
8. Dysphasia
9. Cerebrovascular Insult
10. Vertiginous syndrome
11. Mental symptoms

1. Operational exposure should be as wide as possible. Skin unfolding and craniotomy should extend along the entire midline to allow visualization of both sides of the sinus and approximately 3 cm outside the borders of the occluded sinus. However, such a large approach should be reconsidered if the skin, pericranium, or collateral veins can be damaged during such an opening. A bi-coronary incision is preferred because it allows for maximum vascularization in the skin, especially if subsequent craniotomies are to be performed.
2. Afferent tumor arteries within the wall must be coagulated or clipped before being cut.
3. The dura is cut peripherally around the tumor mass in the convex form to the convex margins, along the border of the superior SSS sagittal sinus. Multiple holes are made close to each other on the periphery of the tumor.
4. Craniotomy (Bur holes) around the tumor and SSS allow bone preparation for the bone.
5. The microscope is installed.
6. Microsurgery separation of the tumor capsule from the surrounding cortex is performed by preserving the vessels that overlap in the normal cortex.

7. Adhesion of the meningioma to the lateral sinus wall and adjoining phalanx structures should be discontinued using the method of incision with a bipolar clamp for coagulation, cutting off the tumor from the meningeal supply.

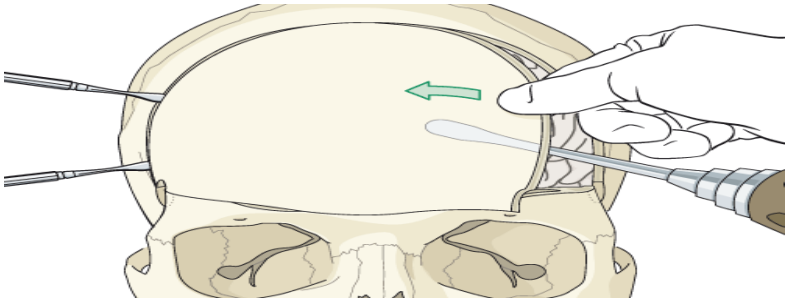


Figure 2.1. Anterior 1/3 bicoronary craniotomy

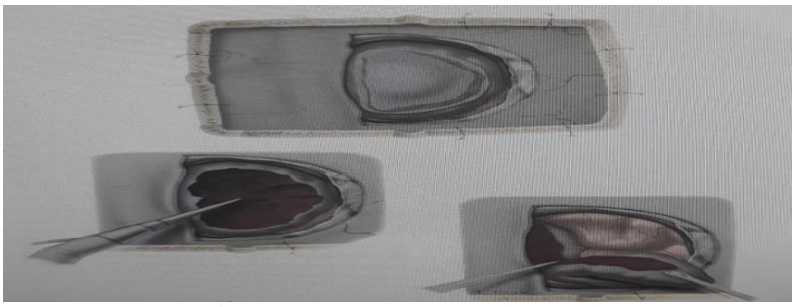


Figure 2.2. 1/3 posterior craniotomy

3. Material and Methods

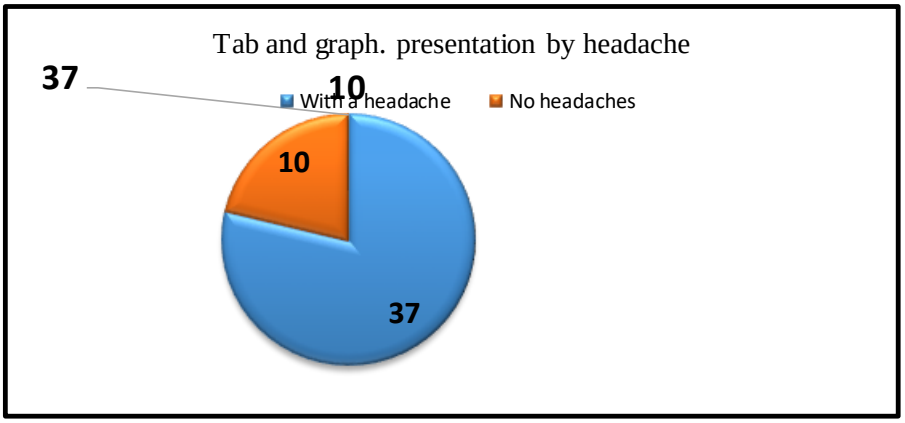
This is a retrospective study, conducted between January 2012 and December 2019, 47 cases were included, treated in Neurosurgery Service, University Hospital Center "Mother Teresa", Tirana, Clinical Hospital Tetovo and the Hospital "September 8" Skopje. Our cohort is homogeneous and unselected. For this kind. Our pathology center is a reference center for Albania, Albanians in Kosovo, Montenegro, and North Macedonia. The patients were led by the same surgeon Prof. Mentor Petrela PU - PH Paris, eliminating thereby the effects of experiential learning on the outcomes of interventions. The study included patients with a histologic diagnosis of meningioma and localized to the anterior 1/3, middle 1/3 and 1/3 posterior throughout the length of the Sagital Superior SSS Sinus. Their dentition was performed by examination. Intervention logs and admission cards. Intervention logs indicate the date of the intervention, patient generalities, preoperative diagnosis, intraoperative diagnosis, a short history of illness, the operator act. Based on the patient list, drawn from the operator registers, the files were found in clinical and imaging files of the follow-up of patients.

4. Results

Table and graphical representations by age, out of a total of 47 patients, mean age is 57.8 years, with the youngest age ranging from 32 to 77 years. Table 4, graph 1.

Total patients studied by headache	Total 47	100 %
Headache	37	78,72 %
No headche	10	21,27 %

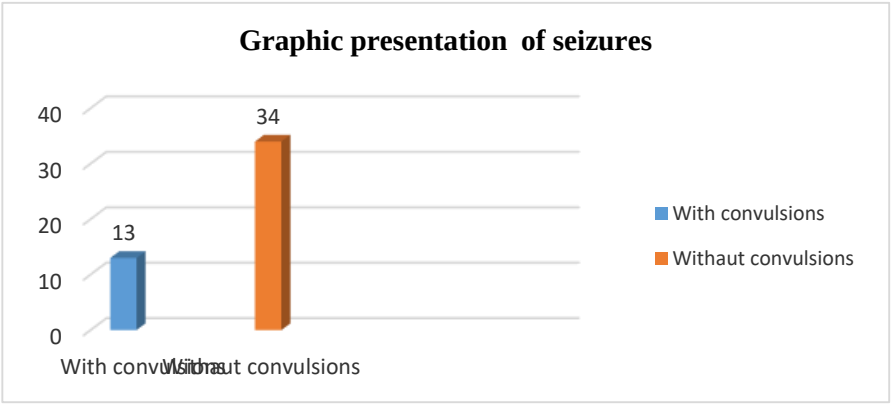
Tabular and graphical presentation of the headache symptom, out of a total of 47 headache patients, is present in 37 patients and in 10 cases there is no headache.



Tabular and graphical presentation of the headache symptom, out of a total of 47 headache patients, is present in 37 patients and in 10 cases there is no headache.

Table 4.1. Table showing the presence of focal and general seizures

Table showing the presence of focal and general seizures	Total 47	100 %
With the presence of focal and general seizures	13	27,65 %
Without the presence of focal and general seizures	34	72,34 %

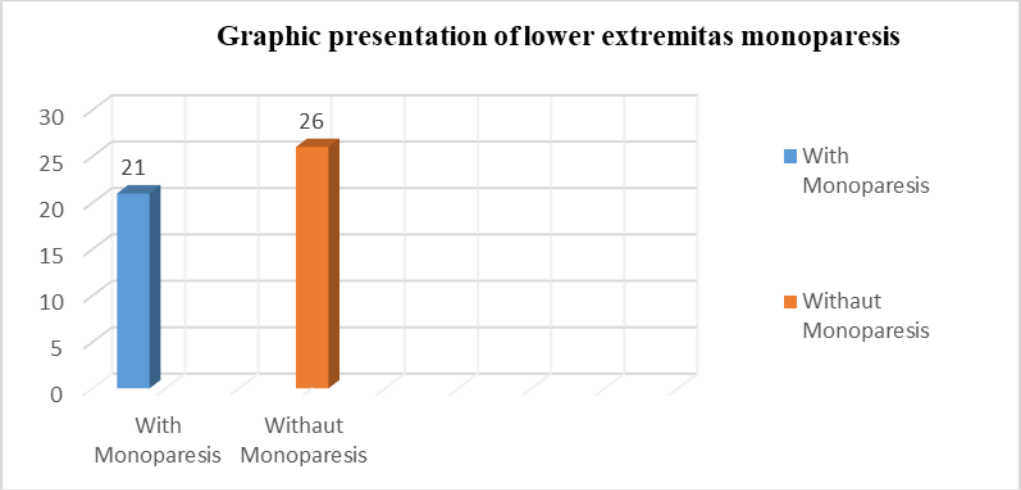


Graph 4.2. Graphic presentation presence of convulsions

Table and graphical presentation of limb monoparesis, from a total of 47 patients with limb limb. The lower extremities have appeared in 21 cases and in 26 cases we have no presence of monoparesis. showing the presence of monoparesis of extremities, out of a total of 47 patients with lower extremity monoparesis appeared in 21 cases, and in 26 cases no presence of monoparesis.

Tabular presentation presence of monoparesis of the lower extremities	Total 47	100 %
With the presence of monoparesis of the lower extremities	21	44,68 %
Without the presence of monoparesis of the lower extremities	26	55,31 %

Tabular presentation presence of monoparesis of the lower extremities



Graphic presentation of lower extremitas monoparesis.

4.1. Unilateral neurological deficit: The neurological deficit also manifests itself in most of our cases. This clinic manifesting patients becomes the leading cause of rapid diagnosis, to the exclusion of other pathologies not related to tumor masses, where in most cases treatment begins as a vascular insult.

4.1.1. Visual symptoms: We refer to three patients with visual symptomatology one of these with bilateral amaurosis as a result of tumor mass in the frontoparietal region with optic chiasm damage according to Ophthalmologists, where after the intervention the patient's vision was impossible in one case and two cases in the other. others have decreased vision sharpness.

4.1.2. Calvary deformity: Calvary deformity refers to two patients, one of whom has marked calvary deformity in the parietal region, this consequence of not being diagnosed on time, not addressing on time has caused the patient to be deformed by head infiltration. the tumor in the lower bone wall of the Tabula Interna, as well as causing artificial bone (MethylMetacrylat) which also causes infections and fistulas which is an increased risk of these patients, refer a patient with such complications.

4.1.3. Dysphasia: In one case, we had immediate expressive dysphagia, where the patient at the clinical examination cut words without any grammatical meaning, resulting in a tumor mass in the brain.

4.1.4. Cerebrovascular insult: No case of ischemic stroke is reported.

4.1.5. Vertiginous syndrome: Dizziness, a very common sign, in more than 17 cases we have SY Vertiginosum with imbalance, a symptomology that directs the patient to the doctor.

4.1.6. Mental symptoms: Very common symptomatology in meningioma patients, mental changes consist of confusion, anterograde amnesia, depressive syndrome and signs of paranoid schizophrenia

4.1.7. Epilepsy: In 16 patients (20.5), complex partial epileptic seizures with secondary generalization and generalized epileptic seizures were reported. Depakine was used in the first group and, Dipedane in the second group. Treatment with AED was continued for 6 months and was discontinued after EEG examination in 12 patients and continued in 4 patients maintained periodically in control every 6 months.

CLINIC	No. of patients
Headache	36
Focal-generalizing seizures	25
Epilepsy	17
Lower extremitas monoparesis	5
Unilateral neurological deficit	26
Calvary deformity	2
Disphasy	4
Cerebrovascular Insult	2
Vertiginous Syndrome	17
Visual Symptom	4

Clinic of parasagittal meningiomas

5. Discussion

After modern technological development and progressive popularization, as well as improved images from CT, RM and DSA it has become possible to clearly define, diagnose, qualitatively, meningiomas of all regions. Regarding the natural progression of meningiomas are not sufficient Among the largest studies conducted with 75 patients, no severe complications were observed such as thrombotic occlusion of the SSS, no mortality, neurological deficits reported in only 5 cases, edema brain in only one case, venous stroke in 1 case, liquid in 2 cases, and systemic complication in 1 case. Small-sized meningiomas, lack of edema, and the presence of calcifications are well progressed. In parasagittal meningiomas, indications of surgery consist in removing neurological deficit, reducing intracranial pressure, decompression of surrounding tumor structures, histologic grade, the relation between venous and arterial blood vessels,

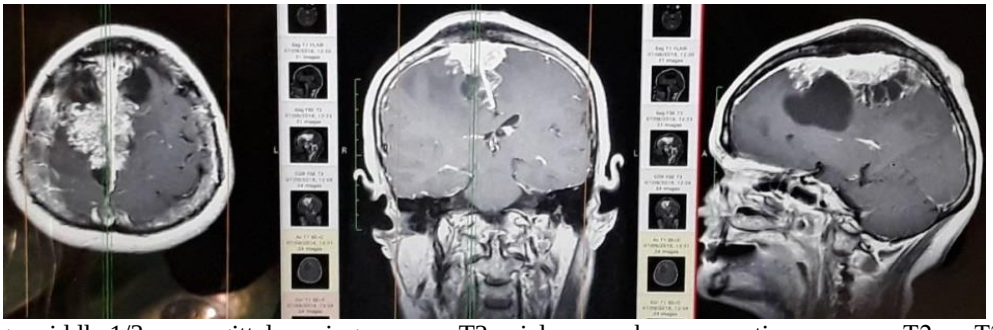


Figure 5.1.-A Large middle 1/3 parasagittal meningeoma a - T2 axial pre op; b pre operative coronary T2; c- T2 sagittal pre op.

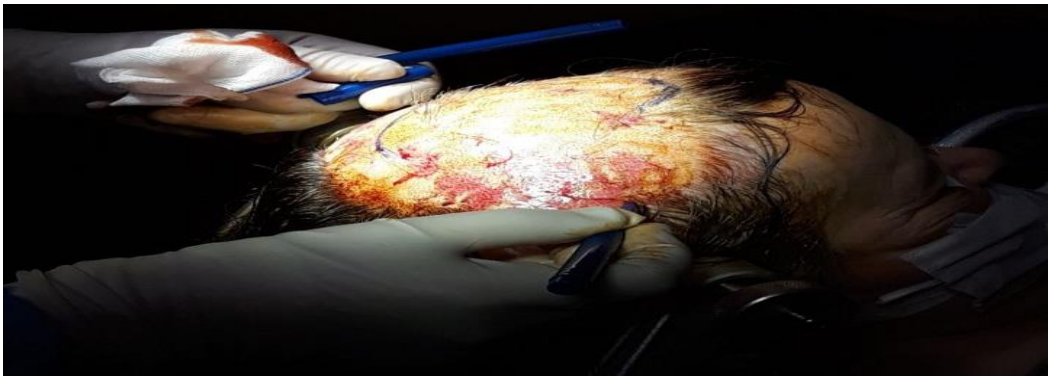


Figure 5.2. Marking of skin incision

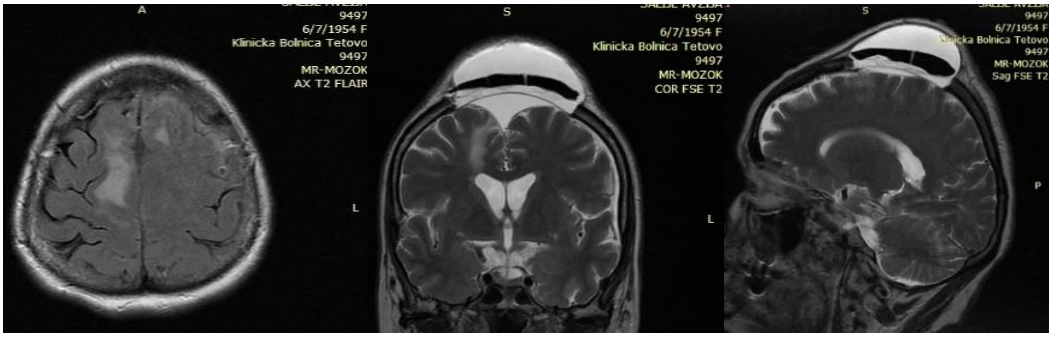


Figure 5.3.-B: Large middle 1/3 parasagittal meningioma a- T1 axial with Gadolinium postop; b1 postop coronary T1; c- T1 sagittal post op.

6. Conclusions

Parasagittal meningiomas in our study from 2012 to 2019 were 47 out of 262 meningiomas in total or 18% which corresponds to the percentage of parasagittal meningiomas in the world literature.
The dominant clinic is headache and neuromotor deficit
The time from onset of complaints to diagnosis is several weeks to 120 months. The Medline in diagnosis was 10 months.
At the moment of diagnosis, a large of them are very large tumors.
Life has increased qualitatively after tumor removal.
Recidiv has been low.
The incidence of parasagittal meningiomas in various literature varies from 16.8% to 25.6%, according to the initial classification based on morphological criteria, whereas the latter classifications are based on tumor location along the SSS. The report is 2.3 cases per 100,000 citizens per year (1.5 for males and 3.1 for females). They make up about 20% of all intracranial tumors.
Almost 50% attack SSS, 50% tend to attack falx and only 25% are bilateral, 25% are associated with skeletal hyperostosis and are a valuable indicator for diagnosis.

7. Summary

Meningioma is in many ways the soul of neurosurgery, progress in the treatment of meningiomas reflects advances in neurosurgery, these advances have been put to maximum use to improve the treatment of meningiomas (8). Meningiomas are female-dominated tumors and mainly affect middle age 90% of whom are benign, 6% are atypical, and only 2% are malignant, in most cases patients with meningioma diagnosis

decide to surgically remove, with minimal neurological symptoms. In most cases, complete removal of the tumor surgically results in a cure, a cure for meningioma, or when all other medications have failed (surgery-radiotherapy). Immunochemiotherapy may work. In 2000, the World Health Organization (WHO) classified nervous system tumors and classified meningiomas as meningeal tumors. The classification describes three scales based on pathological criteria that present the risk of recidiv, rapid growth, and malignity.

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