Scalpellum
JOURNAL OF SURGERY & CLINICAL RESEARCH

Review Article

A Comprehensive Review of Meningioma Pathogenesis, Grading, and Prognostic Stratification

Silmina Suhdi[1], **Andrea Valentino**[1,2*]
[1]Departement of Surgery, Faculty of Medicine, Universitas Riau, Pekanbaru, Indonesia
[2]Division of Neurosurgery, Arifin Achmad General Hospital, Riau, Indonesia

*Corresponding author: andrea.valentino@lecturer.unri.ac.id

Abstract

Meningioma is an intracranial tumor derived from arachnoid meningothelial cells and the most common primary tumor of the central nervous system, accounting for approximately 36.3% of all brain tumors. Most meningiomas are benign and slow growing, but a small number can show atypical or malignant behavior. The main risk factors include radiation exposure, advanced age, female gender, and genetic disorders such as neurofibromatosis type 2. The diagnosis of meningioma is typically established through magnetic resonance imaging (MRI) and subsequently confirmed by histopathological examination. Management of meningioma involves observation, surgery, and/or radiation therapy, depending on the size, location, and clinical symptoms of the patient. The prognosis is generally good, especially in cases of benign meningioma that are successfully completely resected, although the risk of recurrence remains depending on the degree of malignancy and extent of resection. Long-term monitoring is still needed to detect recurrence and complication after treatment.

Keywords: Meningioma, Brain Tumor, Prognostic Stratification

OPEN ACCESS
Submitted: 17 July 2025
Revised: 28 November 2025
Accepted: 16 December 2025
Published: 25 December 2025

Corresponding author
Andrea Valentino
andrea.valentino@lecturer.unri.ac.id

Academic editor
Siti Nurkasanah, M.Biomed

Data Availability Statement
All relevant data are within the paper and its Supporting Information files.

Introduction

Meningioma is a type of primary tumor originating from the meningeal layer of the brain or spinal cord.[1] Meningioma, which is the most common tumor in adults, according to WHO meningioma is generally benign (WHO Grade I) with a lower percentage classified as atypical (WHO Grade II) and rarely anaplastic (WHO Grade III).[2] As reported by histology, meningioma comprises 37.6% of all primary CNS tumors and 53.3% of all benign central nervous system (CNS) tumors. The incidence of meningioma increases with age, with a mean age at diagnosis of 66 years.[3] The etiology of meningioma is not fully understood, but several factors that play a role in the development of meningioma have been identified, both genetic and environmental. Factors that play a role in the development of meningioma can include ionizing radiation, obesity, occupation, diet, hormones, and family factors.[3,4] The clinical manifestations of this tumor vary depending on the location of the tumor. Clinical manifestations that appear can be headaches due to increased intracranial pressure (ICP), focal or global neurological deficits due to local mass effects.[5,6]

This literature review aims to provide a comprehensive understanding of meningioma, by reviewing aspects of epidemiology, etiology, classification, diagnosis, and therapeutic approaches based on the latest scientific evidence.

Methodology for Literature Review

The method used in this literature review is to search and review various literatures sourced from online databases such as PubMed, Google Scholar, and MDPI, from 2020 to 2025. Keywords used in the literature search process include "Meningioma", "Epidemiology of Meningioma", "Diagnosis and Management of Meningioma", "Pathophysiology of Meningioma", “Primary CNS Tumor”, “NF2 Gene”, “Radiotherapy”, “Simpson Grade”, “Surgical Resection”. Inclusion of a small number of older references was accepted if necessary to provide evidence. The literature search procedure is considered appropriate and relevant to the topic.

Discussion

Definition

Meningioma is a type of primary tumor that originates from the meningeal layer of the brain or spinal cord.[1] Originating from meningothelial cells or arachnoid cap, these tumors are often found in the cranial vault, skull base, tentorium cerebelli, and falx cerebri.[7]

Epidemiology

As reported by histology, meningiomas account for 37.6% of all primary CNS tumors and 53.3% of all benign central nervous system (CNS) tumors. The incidence of meningioma increases with age, with a mean age at diagnosis of 66 years. The incidence rate in patients aged 40 years was 18.69/100,000 and in those aged 0–19 years was 0.16/100,000. In patients aged 40+ years, aged 15–39 years, and aged 0–14 years, meningiomas accounted for 43.6%, 15.6%, and 1.7% of all CNS tumors, respectively. Meningioma occurs more frequently in females than in males, with a female-to-male ratio ranging from 2:1 to 3.5:1.[1] Benign and malignant meningiomas are more common in women, with incidence ratios of 2.33 and 1.12, respectively.[3] According to WHO, the grade for meningiomas, 80% to 81%, is considered typical or grade 1. While 17% to 18% of them are atypical or grade 2, and 1.7% are anaplastic or grade 3 meningiomas.[1]

Risk Factor

Meningiomas develop from the meningeal layer of the CNS, specifically, the arachnoid barrier cells located within the leptomeninges, with approximately 90% found above the tentorium and the remaining 10% distributed between the area below the tentorium and along the spinal cord. Most meningiomas are sporadic, benign, and slow-growing. Meningiomas usually show one or more focal chromosome deletions, however, malignant meningiomas often have multiple chromosomal mutations. More genetic mutations are associated with accelerated growth and increased tumor grade.[2,8]

1. Ionizing radiation

Exposure to ionizing radiation is one of the risk factors for meningioma. This type of radiation can directly damage the double helix structure of DNA, triggering the activation of DNA damage detection mechanisms that can then lead to cell senescence, apoptosis, or necrosis. In addition, radiation also disrupts the normal mitosis process and damages various subcellular structures such as the cytoplasmic membrane, endoplasmic reticulum, ribosomes, mitochondria, and lysosomes, thereby affecting the regulation of tumor cell activity.[9]

2. Hormonal (endogenous and exogenous)

Estrogen hormones that are physiologically produced in women's bodies are also known to have carcinogenic potential. Several studies have shown that a person's risk of developing cancer can increase due to the influence of estrogen and progesterone hormones. One example is the use of diethylstilbestrol (DES), a form of synthetic estrogen given to pregnant women in the United States between 1940 and 1971 to prevent miscarriage and other pregnancy complications. Later studies showed that women who received DES had an increased risk of developing cancer.9 Several studies have attempted to link endogenous and exogenous hormone exposure to meningioma due to the higher incidence in women of reproductive age, expression of hormone receptors on tumors, association with breast cancer, and changes in meningioma size during pregnancy, menstrual cycle, and menopause. Longer exposure to exogenous progesterone in women is associated with lower levels of progesterone receptor (PR) and NF2 mRNA in the blood and is thought to be associated with a higher risk of meningioma.[10]

3. Obesity

Proposed mechanisms for the association of obesity with increased meningioma risk include chronic inflammation and increased adipokine-mediated signaling, as well as insulin and insulin-like growth factor (IGF) signaling. IGF-1 is known to suppress apoptosis and stimulate tumor growth. Higher levels of IGF-1 have been observed in obesity and meningioma, suggesting a role in the development of these tumors. Unfortunately, no clear molecular pathways specific to meningioma have been associated with obesity. A positive association with meningioma risk has been identified in body mass index (BMI) and percentage body fat. The relative risk (RR) of meningioma in relation to BMI was 1.48 (95% CI, 1.30–1.69) for obese and 1.18 (95% CI, 1.07–1.31) for overweight.[3]

4. Genetics

Genetic mutations on chromosome 22 in neurofibromatosis type 2 are one of the most common predisposing conditions seen in sporadic meningioma. Patients with NF2 are also more likely to develop grade 2 and 3 or multiple meningiomas. Other chromosome mutations reported in meningiomas are 1p, 6q, 14q, and 18q. There are also genes found in inherited meningiomas such as CREB-binding protein, patched protein homolog 1, phosphatase and tensin homolog, cyclin-dependent kinase inhibitor 2A, Von Hippel–Lindau, and neurofibromatosis.[2,8,10]

Histopathology and WHO Classification

1. Grade 1 meningioma (benign)

Grade 1 meningioma accounts for more than 80% of all cases and consists of nine histological variants. This variant does not show anaplastic features as found in higher grades. Subtypes included in this category include meningothelial, fibroblastic, transitional (or mixed), psamomatous, angiomatous, microcystic, secretory, lymphoplasmacyte-rich, and metaplastic. Each subtype has its own characteristics. More than 70% of meningiomas show progesterone receptor expression. Histopathologically, one of the characteristics of meningioma is the proliferation of meningothelial cells that undergo a mineralization process to form psamoma bodies. Sometimes hyperostosis is also found in the bone around the tumor. WHO grade 1 meningiomas have a low mitotic index (<4/10 high power field), no brain invasion and <3 atypical features (necrosis, small cell change, sheet architecture, macronucleus and hypercellularity).[2,7]

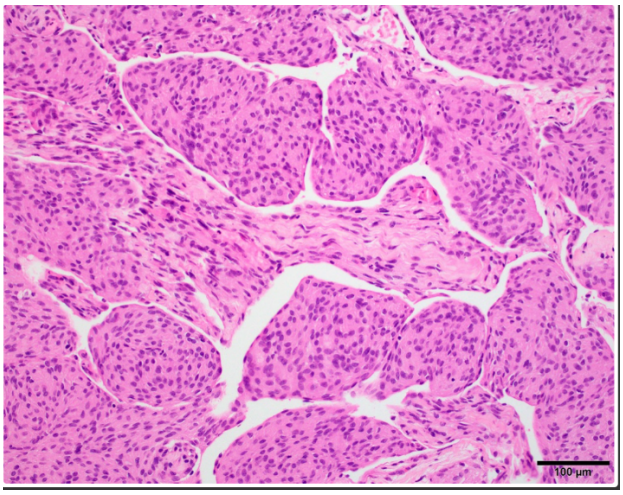

Figure 1. Meningothelial meningiomas form classic meningothelial nests or cellular whorls that resemble arachnoid cap cells.[8]

2. Grade 2 meningioma (atypical)

Grade 2 meningioma is characterized by increased mitosis (4 - 19/10 high power field), increased cellularity or areas of small cell clusters, sheet-like growth pattern, areas of spontaneous necrosis, macronucleoli. Brain invasion is defined as irregular projection of tumor cells into the adjacent CNS parenchyma without a layer of leptomeninges between them at the tumor-brain interface. Meningioma with brain invasion is also called grade 2 meningioma.[1,11]

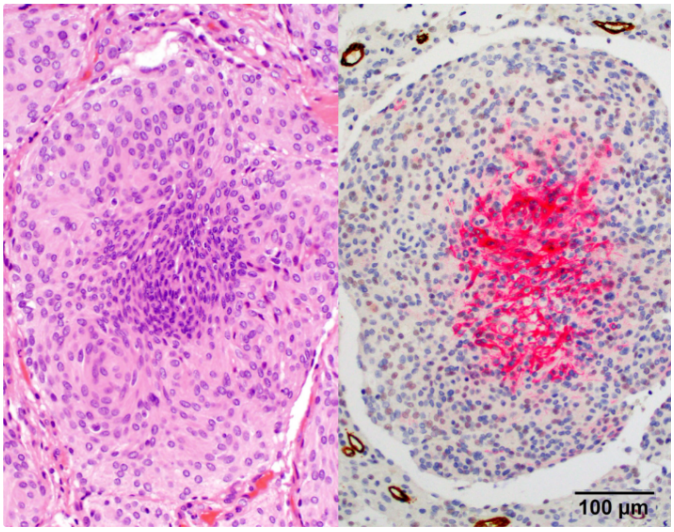

Figure 2. Small cell changes tend to occur in the center of the avascular meningothelial nests. Left panel shows H&E and right panel shows the vascular markers CD34 (brown) and carbonic anhydrase IX (red) which are hypoxic makers from the same field.[11]

3. Grade 3 (anaplastic) meningioma

Grade 3 is an anaplastic malignant lesion that can resemble high-grade sarcoma, carcinoma, or melanoma with a high rate of distant metastasis. High mitotic activity (≥ 12.5 mitoses/mm2, ≥ 20 mitoses/10 HPF each 0.16 mm2.) will also indicate a grade 3 lesion. Histologic variations of this grade include papillary and rhabdoid subtypes. At least focal meningothelial rings and nuclear pseudoinclusions are useful in establishing meningothelial origin, Psammoma bodies may be present, necrosis and brain invasion may be present.[1,12]

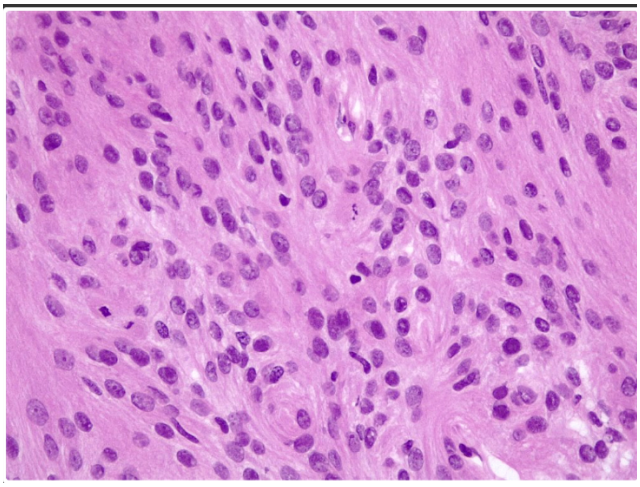

Figure 3. Frozen section of anaplastic meningioma with mitosis.[12]

Molecular Biology of Meningioma

The most frequently observed molecular alteration in sporadic meningiomas is biallelic inactivation of the NF2 tumor suppressor gene located on chromosome 22. Loss of NF2 function results in the absence of Merlin protein, a critical regulator of cell proliferation that normally inhibits the mammalian target of rapamycin complex 1 (mTORC1) and modulates the PI3K/AKT signaling pathway. In the absence of Merlin, these growth-promoting pathways become constitutively active, contributing to tumorigenesis and progression. In addition to NF2, several other driver mutations have been identified in non-NF2 meningiomas, including TRAF7, KLF4, AKT1, SMO, PIK3CA, POLR2A, TERT, and SMARCE1. These mutations are often mutually exclusive and tend to correlate with specific histopathologic subtypes and anatomical locations, particularly within the skull base. For instance, SMO and AKT1 mutations are commonly associated with meningothelial meningiomas located at the anterior skull base, whereas KLF4 and TRAF7 co-mutations are characteristic of secretory subtypes.[13,14]

Epigenetic alterations, particularly aberrant DNA methylation, have emerged as important contributors to meningioma pathogenesis. Recent studies have classified meningiomas into six distinct methylation classes with varying degrees of aggressiveness, independent of histological grade. These methylation-based classifications have demonstrated superior prognostic accuracy compared to traditional WHO grading, particularly in distinguishing biologically aggressive tumors among histologically benign lesions. At the signaling level, tumorigenesis is driven by dysregulation of several key intracellular pathways, notably the RAS/MAPK, PI3K/AKT/mTOR, and Sonic Hedgehog pathways. These cascades play critical roles in promoting cellular proliferation, survival, angiogenesis, and inhibition of apoptosis. Additional pathways implicated in higher-grade meningiomas include the Rb/p53 tumor suppressor axis, Wnt/β-catenin, and Notch signaling. Furthermore, peritumoral brain edema (PTE), observed in approximately 40–60% of meningioma cases, represents a significant pathophysiological phenomenon associated with increased perioperative morbidity. The development of PTE has been linked to overexpression of vascular endothelial growth factor (VEGF), matrix metalloproteinases (particularly MMP-9), and aquaporin-4, all of which contribute to increased vascular permeability and fluid accumulation in peritumoral brain tissue.[13,14]

In conclusion, the pathophysiology of meningioma encompasses a wide spectrum of molecular and cellular abnormalities that influence tumor behavior, prognosis, and potential therapeutic responsiveness. The integration of molecular profiling into routine diagnostic workflows holds promise for enhancing the precision of meningioma classification and informing individualized treatment strategies.[13,14]

Diagnosis

Clinical symptoms of meningioma are often unclear, unless the tumor is quite large. This is because the tumor grows slowly. Some meningiomas may not show symptoms throughout life. Tumors are usually detected accidentally during a brain scan. The signs and symptoms that appear depend on the size and location of the tumor. Symptoms experienced can include headaches, hearing disorders such as ringing in the ears, visual disturbances and nausea and vomiting.[5] The symptoms experienced by meningioma patients also depend on the specific location of the tumor in the brain:

- Falx and parasagittal: Can cause impaired brain function such as decreased memory and thinking ability. If the tumor is in the midline, the patient is at risk of experiencing seizures and muscle weakness.
- Convexity (outer surface of the brain): Generally triggers seizures, headaches, and impaired cognitive function.
- Sphenoid: Can cause impaired vision, facial paralysis, and seizures due to pressure on the surrounding nerve structures.
- Olfactory groove: Causes loss of the ability to smell due to tumor pressure on the olfactory nerve. If the tumor grows larger, pressure on the optic nerve can cause impaired vision.
- Suprasellar: This location is often associated with visual impairment due to its proximity to the optic nerve pathway.

- Posterior fossa: Can trigger hearing loss, difficulty walking in balance (gait ataxia), and impaired body coordination.
- Cerebellopontine angle: Usually causes hearing loss and facial muscle paralysis.

The main imaging modality used in the evaluation of meningioma is Magnetic Resonance Imaging (MRI) with the use of gadolinium contrast. This technique is the gold standard for initial diagnosis and periodic monitoring of meningioma patients. Computed Tomography (CT) Scan with contrast can be an alternative, especially in certain conditions such as contraindications to MRI or for evaluation of bone structure. On CT examination, meningioma generally appears isodense to the brain parenchyma, but in some cases it can be seen as a hyperdense or slightly hypodense lesion. CT Scan has advantages over MRI in detecting psammomatous calcifications, which are found in about 25% of meningioma cases. Overall, both CT Scan and MRI play an important role in establishing a diagnosis, monitoring disease progression, and evaluating the response to therapy, both post-surgery and after other adjuvant therapies.[3,10]

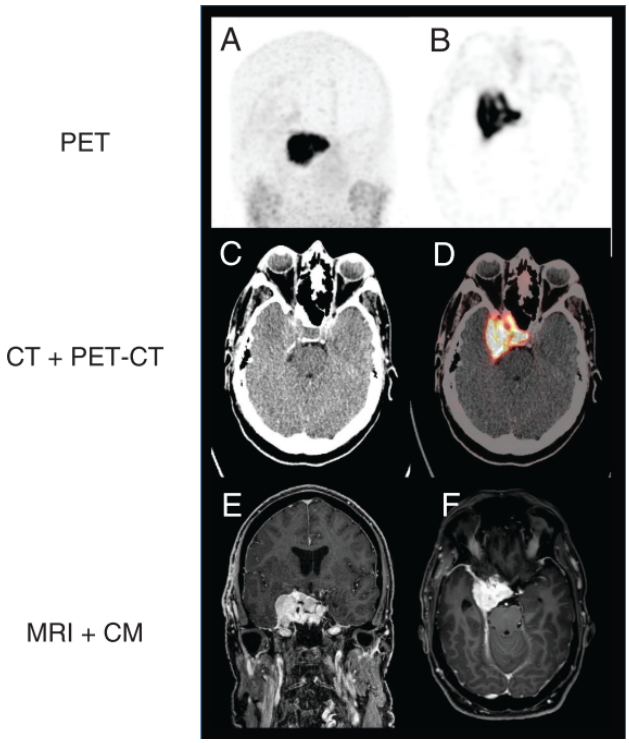

Figure 4. PET CT and MRI of meningioma.[10]

Management

1. Observation

The observation approach is applied to meningioma patients who are asymptomatic and/or have a tumor size ≤3 cm in diameter. Monitoring is carried out periodically using MRI until the patient begins to show symptoms or the tumor grows in size. Based on guidelines from the European Association of Neuro-Oncology (EANO), the first MRI examination is performed six months after the diagnosis is made. If the patient remains asymptomatic, further monitoring is carried out annually for five years. Meanwhile, patients with benign meningioma who have limited life expectancy due to advanced age or severe complications do not need to undergo further observation.[3,10]

2. Surgery

Surgery is the main option for meningioma patients who show symptoms, patients who fail the observation phase, or patients with large tumors that are expected to cause symptoms. Surgery aims to perform a complete total tumor resection or Gross Total Resection (GTR). However, the implementation of GTR can be limited by factors such as the location of the tumor being difficult to reach, involvement of the venous sinuses, or surrounding neurovascular structures. The ultimate goal of surgery is total tumor removal. As with other brain tumors, the success of resection is evaluated by CT scan or MRI within 72 hours after surgery. Postoperative assessment is also based on MRI results and histopathology analysis used in the Simpson grading system—a prediction system for the risk of meningioma recurrence. In this system:[3,10,15]

- Grade 0 indicates complete tumor removal plus removal of an additional 2–3 cm from tumor insertion site *(proposed addition)*
- Grade 1 indicates GTR including tumor, dura, and affected bone (extradural extension).
- Grade 2 indicates GTR of tumor with coagulation of the dura.
- Grade 3 includes GTR of tumor without resection or coagulation of the dura and without removal of the extradural part.
- Grade 4 indicates partial resection of the tumor.
- Grade 5 only biopsy is performed.

If total resection is not possible, planned subtotal resection should be performed to preserve neurological function. Residual meningioma can then be monitored or treated with stereotactic radiosurgery (SRS) or conformal fractionated radiotherapy postoperatively.[3,10]

3. Radiosurgery

Radiosurgery has been established as an alternative therapy to surgery in well-defined cases with small tumors in elderly or critically ill patients. Local control of small intracranial meningiomas measuring 3 cm or less after SRS is comparable to Simpson Grade I resection. Two retrospective series found that tumor size reduction after SRS or hypofractionated RT was predictive of long-term tumor control after 5 and 10 years. The 10-year recurrence-free survival was 93.4% and 95.7%, respectively, using doses above 13 Gy. The

use of stereotactic radiotherapy for the management of meningioma continues to be developed, using linear accelerators, Leksell Gamma knives. Stereotactic radiotherapy is considered appropriate for small tumors (<35 mm).[3,10]

4. Fractionated external beam radiotherapy

Fractionated external beam radiotherapy remains an important component of the therapeutic arsenal for the management of meningioma. For patients with meningiomas that are unsafe for surgery, or after incomplete surgical resection, several large retrospective studies published over the past 3 years have confirmed the current EANO guidelines providing class III evidence with recommendations B and C, for the use of fractionated RT. In a series of 7811 patients with WHO grade 2 meningiomas and 1936 patients with WHO grade 3 meningiomas obtained from the US National Cancer Database who underwent surgical resection and/or RT from 2004 to 2014, the 5-year overall survival (OS) was 75.9% in patients with grade 2, and 55.4% in patients with grade 3 meningiomas (P < .0001). In patients with grade 2 meningiomas, gross total resection and postoperative fractionated RT were independent predictors of improved survival.[10]

5. Chemotherapy

Despite extensive investigation over the past three decades, no chemotherapeutic agent has demonstrated consistent efficacy for refractory or recurrent meningiomas, and none are currently considered standard of care. Among the agents studied, hydroxyurea was the most frequently administered, typically dosed at 1000–1500 mg/day (approximately 20 mg/kg/day), yet its benefits were modest and accompanied by frequent haematological toxicities. Limited partial radiographic responses were reported with agents such as hydroxyurea, mifepristone (200 mg/day), tamoxifen (30–40 mg/day), and combination regimens involving cyclophosphamide, doxorubicin, and vincristine. However, these responses were generally not corroborated in randomized trials, and six-month progression-free survival rates varied widely from 6% to 100%. Current research is shifting toward molecularly targeted therapies rather than conventional cytotoxic chemotherapy, underscoring the need for personalized, mechanism-driven approaches to treatment.[16]

Prognosis And Recurrence

The prognosis and recurrence of meningioma are strongly influenced by multiple interrelated factors including histopathological grade, extent of resection, proliferative index, and the tumor's anatomical location. WHO grade I meningiomas, typically benign and slow-growing, are associated with a favourable prognosis and low recurrence rates, particularly when gross total resection (Simpson grade I–II) is achieved. However, even among these, long-term follow-up remains essential, as recurrence can occur many years postoperatively. In contrast, WHO grade II (atypical) and grade III (anaplastic) meningiomas exhibit a substantially higher risk of recurrence and poorer overall survival, often requiring multimodal management including postoperative radiotherapy. The Simpson grading system continues to serve as a valuable prognostic tool; while Simpson grades I through IV are not significantly different in recurrence rates, Simpson grade V (biopsy or decompression only) is independently associated with markedly increased risk of tumor recurrence. Moreover, elevated MIB-1 (Ki-67) labelling index values—particularly >3%—have been associated with greater proliferative activity and a higher likelihood of recurrence, especially when combined with high-grade histology. Advanced imaging modalities such as 68Ga-DOTATATE PET and improved surgical techniques have enhanced detection and management of residual disease, yet no current strategy fully eliminates the risk of relapse. Importantly, recurrence is not confined to the early postoperative years; studies have identified a critical recurrence interval at approximately 5.5 years post-surgery, beyond which tumor reappearance remains a considerable concern. Consequently, the management of meningioma should adopt a long-term, individualized surveillance strategy, particularly for patients with subtotal resection, high-grade tumors, or elevated proliferative indice.[17,18]

Meningioma in Indonesian Context

Meningioma is one of the most commonly diagnosed primary intracranial tumors in Indonesia, mirroring global patterns of higher prevalence in adult females, particularly in the 40 to 60-year age group.[19] A recent retrospective study conducted at the Dharmais National Cancer Center described the epidemiological features of WHO grade II and III meningiomas between 2011 and 2022, reporting that while these high-grade tumors constitute a minority of total meningioma cases, they are associated with increased recurrence and worse prognosis.[18] Most grade II cases were histologically atypical (95%), and most grade III were anaplastic (76.9%), frequently located in convexity or skull base regions. According to Indonesia's National Clinical Practice Guidelines for Brain Tumors (Pedoman Nasional Pelayanan Kedokteran, PNPK), the standard management includes total surgical resection followed by adjunct radiotherapy when needed, supported by advanced imaging such as MRI and CT with contrast.[20] However, access to diagnostic imaging, neurosurgical care, and histopathological services is

unevenly distributed, posing challenges in early diagnosis and optimal management, especially in non-urban regions. Despite these challenges, research on meningioma in Indonesia is not only feasible but also highly relevant. Several national referral centers such as RS Kanker Dharmais and RSUP Dr. Hasan Sadikin have the clinical caseload, technical infrastructure, and academic collaborations necessary for conducting robust epidemiological, clinical, and translational studies. Such research would be instrumental in generating context-specific data to inform national cancer policy, optimize patient outcomes, and address existing disparities in care delivery.

Conclusion

Meningioma is the most common primary tumor of the central nervous system, predominantly comprising benign WHO Grade I lesion, yet it includes aggressive subsets (Grade II and III) that demand specialized attention. Diagnosis hinges on state-of-the-art imaging, particularly contrast-enhanced MRI, followed by histopathological confirmation. Current management principles are multimodal, involving observation for small, asymptomatic cases, Gross Total Resection (GTR) as the therapeutic cornerstone, and adjuvant radiotherapy for residual, recurrent, or higher-grade disease. Prognosis is highly influenced by the WHO malignancy grade, the completeness of surgical resection (Simpson Grade), and the MIB-1 proliferative index. Moving forward, meningioma management is shifting toward personalized medicine by integrating molecular-based classification (such as DNA methylation classes) into routine diagnostic workflows, given its superior prognostic accuracy. Future research must prioritize validating therapies that target aberrant signaling pathways (such as PI3K/AKT/mTOR and RAS/MAPK) and elucidating the molecular mechanisms driving peritumoral brain edema (PTE), facilitating a transition from general histology-based treatment to targeted, individualized interventions aimed at improving long-term disease control and patient quality of life.

Ethics approval

Not required.

Acknowledgments

I would like to express my deepest gratitude to my supervisor, dr. Andrea Valentino, Sp.BS, Subsp. N-Onk, for his continuous guidance, direction, and support during this research process. I would also like to express my gratitude to all parties who have helped in the preparation of this article, including in terms of language assistance, writing, and manuscript revision. All input and assistance provided are very meaningful for the completion of this research

Competing interests

All the authors declare that there are no conflicts of interest.

Funding

This study received no external funding.

Underlying data

Derived data supporting the findings of this study are available from the corresponding author on request.

References

1. Alruwaili AA, De Jesus O. Meningioma. [Updated 23 Aug 2023]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK560538/
2. Antonica F, Aiello G, Soldano A, Abballe L, Miele E, Tiberi L. Brain Tumor Modeling: A Perspective Review of Organoid and In Vivo Models. Front Mol Neurosci. 2022;15(May):1-19.
3. Ogasawara, C., Philbrick, B. D., & Adamson, D. C. (2021). Meningioma: A Review of Epidemiology, Pathology, Diagnosis, Treatment, and Future Directions. Biomedicine, 9(3), 319.
4. Boetto J, Birzu C, Kalamarides M, Peyre M, Sanson M. Meningioma: A review of current knowledge. La Rev Médecine Interne. 2021;43(2):3-31.
5. Putra AF, Farah A, Putri A, Oriesto B, Sari EE, Hidayah GP, et al. Education about meningioma, risk factors and treatment efforts. J Pengabdi Masy Trimedika (JPMT). 2024;1(1):16-23.
6. Talawo V, Kaelan C, Juniarsih J, Zainudin A, Ihwan A, Cangara M, et al. Clinical Characteristics and Histopathology of Meningioma in Makassar. J Kesehatan Tadulako. 2023;9(1):81-6.
7. Awuah WA, Ben-Jaafar A, Karkhanis S, Nkrumah-Boateng PA, Kong JSH, Mannan KM, et al. Cancer stem cells in meningioma: new insights and therapeutic implications. Clin Transl Oncol [Internet]. 2024;(1-22).
8. Cai C. Pathology Outlines. 2021. Meningioma. Available from: https://www.pathologyoutlines.com/topic/cnstumormeningiomageneral.html
9. Putri TAK, Prihatin LM, Priyanto B. Meningioma: A Literature Review. J Biol Trop. 2023;23(1):364-70.
10. Goldbrunner R, Stavrinou P, Jenkinson MD, Sahm F, Mawrin C, Weber DC, et al. EANO guideline on the diagnosis and management of meningioma. EANO Guidel meningioma. 2021;23(11):1821-34.
11. Cai C, Kresak J, Yachnis A. Pathology Outlines. 2021. Atypical meningioma. Available from: https://www.pathologyoutlines.com/topic/cnstumoratypicalmeningioma.html
12. Barresi V. Pathology Outlines. 2022. Anaplastic meningioma. Available from:

https://www.pathologyoutlines.com/topic/cnstumoranaplasticmeningioma.html.

13. Delgado-López PD, Cubo-Delgado E, González-Bernal JJ, Martín-Alonso J. A Practical Overview on the Molecular Biology of Meningioma. Curr Neurol Neurosci Rep. 2020;20(12).
14. Youngblood MW, Günel M. Molecular genetics of meningiomas. Handb Clin Neurol. 2020;169:101–19.
15. Wilson TA, Huang L, Ramanathan D, Lopez-Gonzalez M, Pillai P, De Los Reyes K, et al. Review of Atypical and Anaplastic Meningiomas: Classification, Molecular Biology, and Management. Front Oncol. 2020;10(November).
16. Shahbandi A, Shah DS, Hadley CC, Patel AJ. The Role of Pharmacotherapy in Treatment of Meningioma: A Systematic Review. Cancers (Basel). 2023;15(2):1–25.
17. Fotakopoulos G, Georgakopoulou VE, Spandidos DA, Angelopoulou E, Menis AA. Prognostic factors for first recurrence following meningioma surgery. 2025;1–7.
18. Corniola MV, Meling TR. Management of Recurrent Meningiomas: State of the Art and Perspectives. Cancers (Basel). 2022;14(16):1–14.
19. Singh A, Firdaus M, Widarta OA, Sugiarto YK, Halim D, Faried A. Epidemiological Characteristics of WHO Grade II and III Intracranial Meningioma Cases at Dharmais Cancer Hospital. Malahayati Health Student Journal. 2023;3(11):3741–3752.
20. Kementerian Kesehatan Republik Indonesia. Pedoman Nasional Pelayanan Kedokteran Tumor Otak. Jakarta: Kementerian Kesehatan RI; 2019