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Purely cystic meningioma: extremely rare clinical and radiographic mimic of intra-axial glioblastoma multiforme with systematic review of the literature. Illustrative case

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BACKGROUND Purely cystic meningiomas are extremely rare extra-axial neoplasms that can mimic aggressive malignancies, like glioblastoma multiforme (GBM), because radiologically and intraoperatively they lack a visible solid component and demonstrate postcontrast enhancement and significant vasogenic edema. The authors present a case of a purely cystic intra-axial meningioma mimicking GBM with an accompanying systematic review of the literature.

OBSERVATIONS An 84-year-old female presented with expressive dysphasia. MRI revealed a 3-cm inhomogeneously enhancing intra-axial left temporal lobe mass with apparent MRI presentation of central necrosis, suggesting a high-grade glioma. Intraoperatively, the lesion appeared vascular and infiltrative like a GBM; however, histopathological and immunohistochemical analyses (somatostatin receptor 2 antigen positive, progesterone receptor positive, glial fibrillary acidic protein negative) confirmed an angiomatous meningioma (WHO grade 1). Postoperatively, the patient's symptoms resolved, and 10 years of annual follow-up MRI studies confirmed no recurrence. Only 3 other purely cystic meningioma cases (extra-axial) were identified in the literature.

LESSONS Cystic meningiomas are rarely GBM "imitators." Surgeons should consider cystic meningioma when classic radiological hallmarks are absent. Despite an aggressive imaging profile, these tumors are biologically and clinically benign. Gross-total resection remains the gold-standard treatment, offering excellent long-term prognosis and the potential for a permanent cure. This case highlights the need for histological tissue diagnosis before final treatment plans are considered.

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KEYWORDS purely cystic meningioma; glioblastoma mimic; case report; systematic review

Meningiomas are the most prevalent primary nonglial intracranial neoplasms, representing approximately 13%–53.3% of all primary brain tumors.[1,2] While typically recognized as slow-growing, well-demarcated, and solid extra-axial lesions, their clinical and radiological presentation can vary significantly based on histological subtype and associated secondary changes. Among meningiomas, the cystic meningioma is a rare and distinct variant, occurring in only 1.6%–11.7% of all cases.[3,4] These tumors are characterized by the presence of macroscopic intratumoral, peritumoral, or intraparenchymal cysts, which often obscure the classic dural-based architecture of the lesion.[5] However, purely cystic meningiomas are extremely rare, with only 3 published cases, all of which were extra-axial and one of which was intraosseous.

The diagnostic complexity is further intensified in the purely cystic meningioma. Although biologically benign, meningiomas are notorious for their extreme vascularity and the induction of disproportionately extensive perifocal vasogenic edema. When these vascular features coexist with large cystic components, the resulting radiological profile—characterized by heterogeneous enhancement and apparent central "necrosis"—frequently mimics more aggressive malignancies, such as glioblastoma multiforme (GBM) or metastatic tumor.[1] This radiographic mimicry often leads to preoperative misclassification, potentially altering the surgical strategy and patient counseling.

In this report, we present a rare case of a purely cystic angiomatous intra-axial meningioma in an 84-year-old patient, which was initially diagnosed radiologically and intraoperatively as a high-grade glioma. We describe the successful gross-total resection via a left anterior temporal craniotomy and a unique 10-year longitudinal follow-up confirming the absence of recurrence. Furthermore, we provide a systematic review of the literature to analyze the radiological pitfalls and surgical nuances required to differentiate these extremely rare benign entities from their malignant counterparts.

ABBREVIATIONS FLAIR = fluid-attenuated inversion recovery; GBM = glioblastoma multiforme; PR = progesterone receptor; SSTR2A = somatostatin receptor 2 antigen.

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Illustrative Case

An 84-year-old female presented with isolated expressive dysphasia and word-finding difficulties; a neurological examination was otherwise within normal limits. Initial neuroimaging via noncontrast CT and multiparametric MRI revealed a 3-cm inhomogeneous mass in the left temporal lobe (Fig. 1). The lesion exhibited hyperintensity on T2-weighted and fluid-attenuated inversion recovery (FLAIR) MR sequences, significant perifocal vasogenic edema, and heterogeneous enhancement following gadolinium administration. Given the presence of apparent central necrosis and mass effect on the left lateral ventricle, a preoperative diagnosis of high-grade glioma was suspected.

Surgical intervention was performed by the senior author (K.I.A.). The patient was placed in the supine position with the head rotated approximately 30° to the right and secured in a Mayfield head frame to optimize access to the left temporal lobe. A standard left-sided curvilinear skin incision was performed, starting superiorly to the zygomatic arch and extending posteriorly toward the ear. A temporal craniotomy was performed in standard fashion. Following a bone flap elevation, a dural incision was made in a semicircular fashion, revealing a soft, gray-purple highly vascularized mass.

The tumor demonstrated an atypical seemingly infiltrative border within the surrounding brain parenchyma, which, combined with the presence of prominent peritumoral edema, closely mimicked the appearance of a high-grade glioma (WHO grade 4). Using microdissection techniques and bipolar coagulation for hemostasis, we debulked the lesion and then dissected it from the surrounding temporal parenchyma. Gross-total resection was achieved and a whole specimen was submitted for histological analysis. The patient tolerated the procedure without intraoperative complications. Intraoperative frozen biopsy was not suggestive of high-grade glioma, and we awaited the final report.

Final histopathological and immunohistochemical analysis corrected the diagnosis to meningioma (WHO grade 1). Postoperative recovery was excellent, with complete resolution of speech deficits.

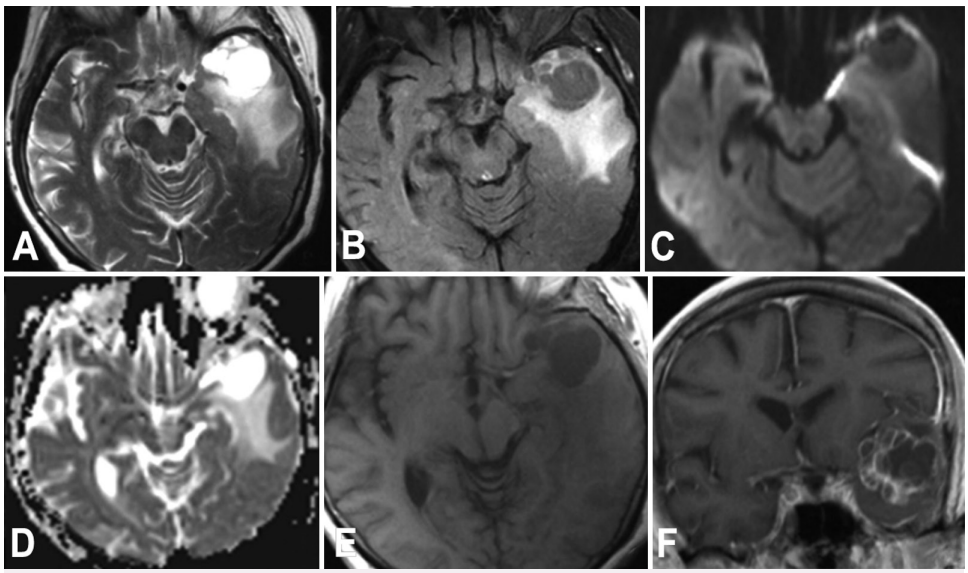

FIG. 1. Preoperative MR images obtained in the patient: axial T2-weighted image (**A**), axial FLAIR image (**B**), axial diffusion-weighted image (**C**), axial apparent diffusion coefficient map (**D**), axial T1-weighted image (**E**), and coronal T1-weighted spin-echo image after intravenous contrast administration (**F**).

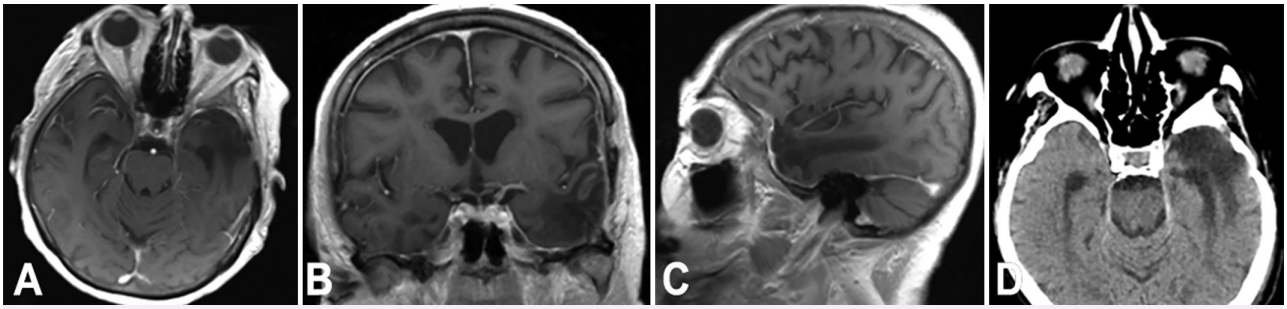

FIG. 2. Follow-up neuroimages obtained to evaluate the extent of resection and monitor for disease recurrence. **A–C:** Axial (A), coronal (B), and sagittal (C) contrast-enhanced T1-weighted MR images demonstrating a stable postoperative surgical bed with no evidence of pathological nodular or linear enhancement—consistent with gross-total resection—and no signs of tumor recurrence. **D:** Axial noncontrast CT scan confirming the expected postoperative changes, including at the craniotomy site, with no evidence of high-density masses or significant perifocal edema.

Long-term surveillance, including MRI and a 10-year follow-up CT study (Fig. 2), confirmed a stable surgical bed with no evidence of recurrence, illustrating the favorable prognosis of this rare meningioma subtype despite its aggressive radiological mimicry. The patient was followed with yearly MRI studies for the next 10 years without neurological deficiencies and without tumor recurrence.

Histological Findings

Histological examination of the resected specimen revealed a highly vascularized neoplasm composed of a biphasic population of meningothelial and spindle cells interspersed among a prominent network of irregular, variably sized thin-walled blood vessels (Fig. 3). Immunohistochemical profiling demonstrated strong and diffuse cytoplasmic positivity for vimentin and nuclear immunoreactivity for progesterone receptor (PR). The diagnosis was further supported by the robust and diffuse membranous expression of somatostatin receptor 2 antigen (SSTR2A) and focal membranous staining for epithelial membrane antigen. To exclude morphological mimics, a broad panel was utilized; the tumor cells were negative for CD34 (ruling out solitary fibrous tumor), glial fibrillary acidic protein (excluding glial origin), and cytokeratin and inhibin (excluding metastatic carcinoma and hemangioblastoma, respectively). These morphological and immunophenotypic features are diagnostic of an angiomatous meningioma, consistent with WHO grade 1 criteria, indicating a benign biological behavior.

Systematic Review

A systematic literature review was conducted in accordance with PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines.[6] A search was performed of the PubMed database using the following Boolean operators and MeSH (Medical Subject Headings) terms: "cystic and meningioma." The search period was restricted to articles published between December 1, 2021, and March 31, 2026.

Studies were included for analysis based on the following criteria: 1) studies published exclusively in the English language; 2) case reports or series that presented purely cystic meningiomas (defined as lesions without a macroscopically visible solid tumor component on imaging); 3) studies with available preoperative MRI data that demonstrated the cystic nature of the lesion; 4) studies that included definitive histopathological confirmation of meningioma, regardless of histological subtype or WHO grade; and 5) studies that included primary intracranial (cranial) localization. Studies were excluded based on the following criteria: 1) published animal studies and veterinary reports, 2) studies published in languages other than English, 3) cases that featured mixed cystic-solid tumors or purely solid meningiomas, 4) studies that featured spinal or extracranial meningiomas, and 5) studies with incomplete radiological or pathological documentation.

Following the initial identification of records, duplicates were removed and titles/abstracts were screened for relevance. Full-text articles were then assessed against the eligibility criteria to ensure a strict focus on purely cystic intracranial meningiomas in human subjects (Fig. 4). The literature search and subsequent screening of titles and abstracts were performed independently by two primary reviewers (B.R. and Z.M.). Any discrepancies or disagreements regarding the eligibility of a specific publication were resolved through consultation and final adjudication by the senior author (K.I.A.).

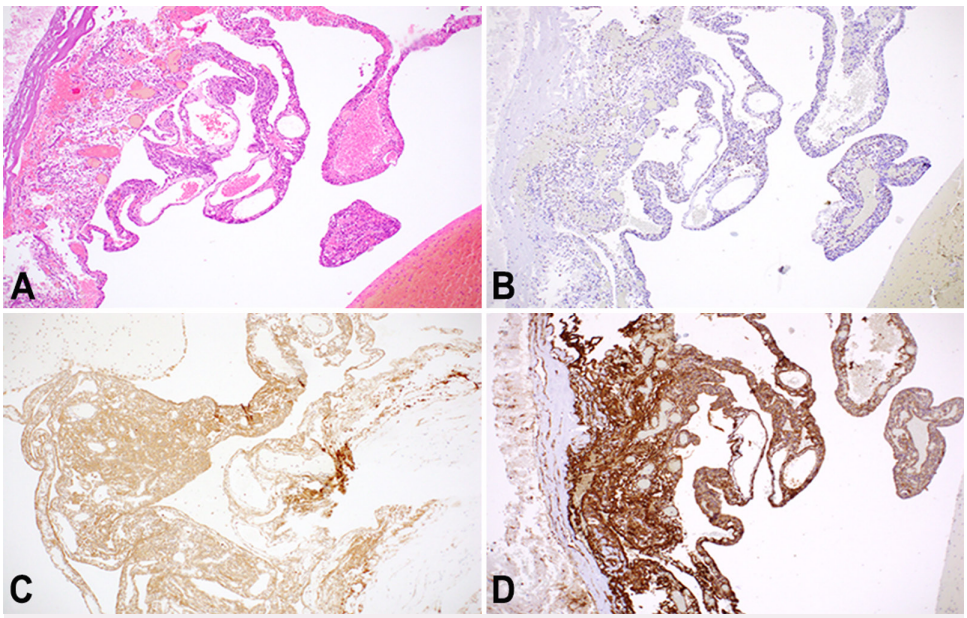

FIG. 3. Histopathological and immunohistochemical characterization of the tumor. Representative photomicrographs (original magnification ×10). **A:** Hematoxylin and eosin section demonstrating a highly vascularized neoplasm characterized by a dual population of meningothelial and spindle cells arranged in a perivascular distribution around irregular, variably sized vascular channels. **B:** SSTR2A stain showing robust, diffuse membranous and cytoplasmic immunoreactivity, confirming the meningothelial origin of the lesion. **C:** PR stain showing strong and diffuse nuclear expression observed across both the spindle and meningothelial cell components. **D:** Intense, diffuse cytoplasmic vimentin stain highlighting the mesenchymal intermediate filaments within the neoplastic cells.

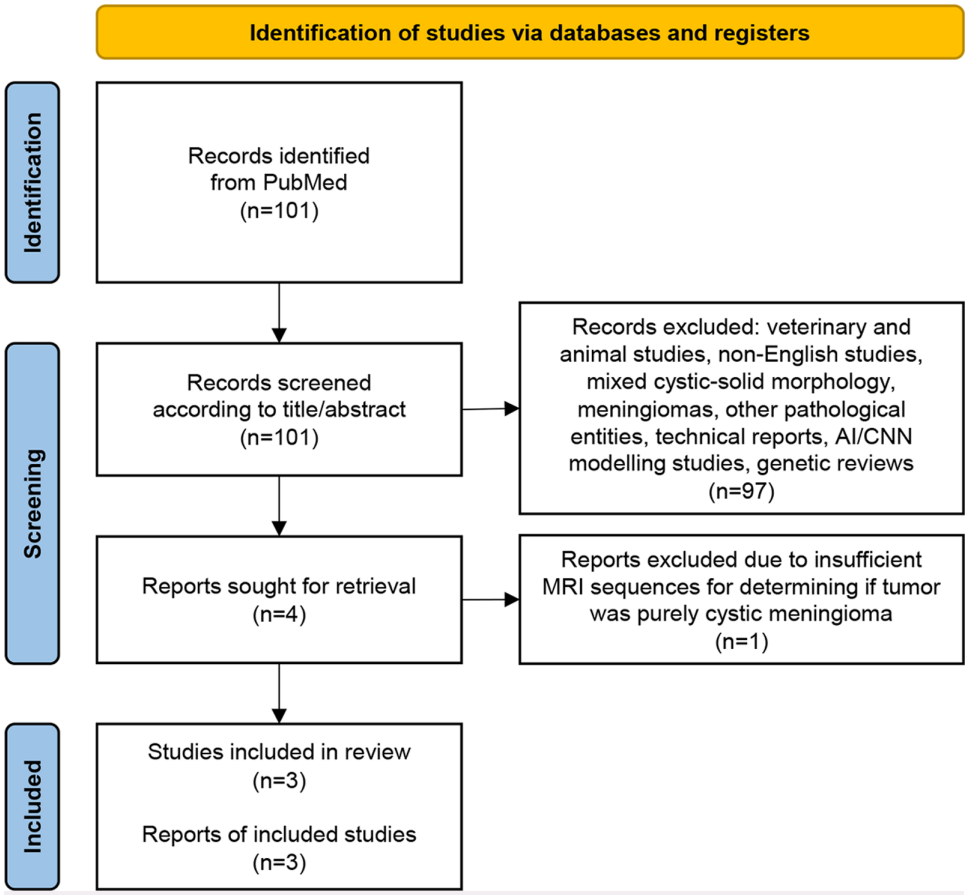

FIG. 4. PRISMA flow diagram. AI = artificial intelligence; CNN = convolutional neural network. Data added to the PRISMA template (from Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. *BMJ*. 2021;372:n71) under the terms of the Creative Commons Attribution (CC BY 4.0) License (https://creativecommons.org/licenses/by/4.0/).

Only 3 other cases were identified in the literature that reported extra-axial purely cystic intracranial meningiomas in younger individuals (Table 1),[1,7,8] one of which was intraosseous.[8] Gkasdaris et al. reported a single case of purely cystic intracranial meningioma;[1] the authors also performed a systematic review of purely cystic intracranial meningioma by searching the PubMed database to December 2021 and found only 1 case, by Ergun et al.[7] Rojas et al. reported a single case of intraosseous purely cystic meningioma;[8] in their accompanying

TABLE 1. Summary of purely cystic meningiomas

Authors & Year	Age (yrs)	Sex	Follow-Up	Location	MRI Findings
Intracranial meningioma					
Ergun et al., 2015[7]	67	F	NA	Parietal	Dural-based extra-axially located rt parietal vertex cystic lesion w/ a dural tail, demonstrating peripheral rim enhancement
Gkasdaris et al., 2022[1]	59	F	3 yrs	Parieto-occipital	Cystic lesion grew progressively to 36 × 55 mm on final preop imaging, compared to 32 × 40 mm on 1st cerebral MRI exam
Present case	84	F	10 yrs	Temporal	Atypical, seemingly infiltrative border w/ surrounding brain parenchyma, which, combined w/ the presence of prominent peritumoral edema, closely mimicked the appearance of a high-grade glioma
Intraosseous meningioma					
Rojas et al., 2025[8]	41	F	6 mos	Frontal	4.0 × 4.5–cm cystic lesion near the dura, w/ expansion of the intradiploic space; an intradiploic arachnoid cyst was suspected

NA = not available.

systematic review, performed by searching the PubMed database to December 2021, they found 2 additional intraosseous meningioma cases. However, these were excluded from our study because they were not purely cystic;[9,10] they had a solid component and insufficient MRI studies to declare the lesion purely cystic.

Informed Consent

The necessary informed consent was obtained in this study.

Discussion

Observations

This case highlights an extremely rare presentation of a purely cystic intracranial meningioma, a diagnostic challenge given its ability to closely mimic a high-grade glioma. Our systematic review further underscores the scarcity of this entity, identifying only 3 previously documented cases in the literature, all of which were female.

A broad differential diagnosis exists for intracranial cystic lesions, often presenting a significant diagnostic dilemma. The diverse etiology of these cysts necessitates a careful evaluation to distinguish them from other neoplastic and nonneoplastic processes.[11] The presence of a cystic component poses a significant challenge for radiological differentiation. Owing to shared imaging features—specifically contrast-enhancing mural nodules—these lesions are frequently misidentified as intra-axial pathologies, including high-grade gliomas, metastatic deposits, hemangioblastomas, or pilocytic astrocytomas. Meningiomas are the most prevalent benign intracranial neoplasms, typically presenting as solid, hypercellular, and well-vascularized masses; their incidental discovery is common.[12]

Histologically, meningiomas arise from the meningothelial cells of the leptomeninges.[13] Their classic MRI profile involves T1 isointensity and strong homogeneous enhancement; however, the presence of a dural tail, although seen in 72% of cases, is not exclusively diagnostic. Diagnostic complexity is further compounded by various meningioma mimics, most notably solitary fibrous tumors, metastatic lesions, and glial tumors.[5] Radiographic differentiation is not always definitive, as GBMs can adopt the hallmarks of a meningioma, including a dural tail and a CSF cleft. Even cerebral angiography, which can reveal tumor feeders common to meningiomas, may fail to distinguish between the two, contributing to a high degree of diagnostic uncertainty in atypical cases.[14] While these mimics typically distribute across the convexity and skull base, our case of a purely cystic variant represents an even rarer diagnostic pitfall that defies these standard radiographic expectations.

In most cases, meningiomas are solid tumors that can be diagnosed using CT scans with a sensitivity of 100% and a specificity of 90%, but in cases of cystic forms, the number of differential diagnoses widens and specificity drops below 50%. Using additional MRI with contrast achieves a preoperative diagnostic accuracy of 80%.[15] Cystic meningiomas were divided into four groups by Nauta et al. in 1979, based on the site of the cystic cavity:[16] 1) centrally located intratumoral cysts, 2) peripherally located intratumoral cysts, 3) peritumoral cysts in the adjacent parenchyma, and 4) peritumoral cysts between the tumor and the adjacent parenchyma.

Our patient was an 84-year-old woman with mild and unspecific symptoms. The necrotic cavities were found inside the larger tumorous mass on brain CT and MRI with irregular margins. The initial radiological diagnosis was a high-grade glioma. The patient and her family members questioned the rationale for any treatment but eventually decide for biopsy and/or resection of the lesion.

The etiology of cystic meningiomas remains a subject of debate. One reported theory focuses on degenerative macrocavitation. Alternatively, the proposed "oasis phenomenon" suggests an ischemic origin, characterized by arteriolar hyalinization in necrotic regions.[17] Other authors have suggested that intratumoral hemorrhage may serve as a precursor to cyst development. Additional authors have suggested peritumoral demyelination, the transudation of cerebral edema into a peritumoral cyst, and secretion by tumor cells or reactive astrocytes.[17] Several mechanisms have been suggested for cystic formation in meningiomas, such as the loculation of widened subarachnoid spaces, ischemic necrosis, peritumoral demyelination, cystic degeneration with or without hemorrhage, the transudation of cerebral edema into a peritumoral cyst, and secretion by tumor cells or reactive astrocytes.[4]

The incidence of cystic meningioma is remarkably low; the current literature has reported a frequency ranging from 1.6% to 11.7%. There are currently no clear data about the incidence of purely cystic meningioma. Ergun et al. only first reported a rare instance of a purely cystic meningioma in 2015.[7] The purely cystic variant deviates significantly from this classic radiographic appearance, often leading to diagnostic confusion. As previously mentioned, we have identified only 3 cases that were purely cystic meningiomas, all extra-axial and one intraosseous.

The management of purely cystic meningiomas remains consistent with that of other histological subtypes of meningiomas, with total resection remaining the primary therapeutic strategy.

Lessons

Purely cystic intracranial meningiomas represent an extremely rare clinical entity that may defy standard radiographic expectations. Because of their ability to mimic intra-axial malignancies, such as high-grade gliomas, they remain a significant diagnostic pitfall, and patients and families may question the rationale for any further surgical treatment—or even a biopsy—given the high suspicion for GBM. This clinical case highlights again the importance of exact histological diagnosis and waiting for histological verification before assuming malignant disease and a dismal prognosis. However, despite their atypical neuroradiological and intraoperative presentation, the surgical objective remains unchanged. Total resection is the definitive treatment, offering a favorable prognosis consistent with more common meningioma variants.

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Disclosures

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author Contributions

Conception and design: Arnautović, Rovčanin, Merhemić, Čampara. Acquisition of data: Arnautović, Rovčanin, Merhemić, Čampara. Analysis and interpretation of data: all authors. Drafting the article: Arnautović, Rovčanin, Čampara. Critically revising the article: Arnautović, Rovčanin, Merhemić, Čampara. Reviewed submitted version of manuscript: Arnautović, Rovčanin, Čampara. Approved the final version of the manuscript on behalf of all authors: Arnautović. Statistical analysis: Arnautović. Administrative/technical/material support: Arnautović, Rovčanin. Study supervision: Arnautović, Merhemić.

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