Pregnancy-Associated Acceleration of Meningiomas: A Three-Patient Case Series

Maxwell A Marino, DO, MPH[1]; Ali O Jamshidi, MD[2];
[1]Riverside University Health System, [2]Kaiser Permanente Woodland Hills

Abstract

Meningiomas are typically indolent tumors but may demonstrate accelerated growth during pregnancy due to hormonal and hemodynamic changes. We present three patients with skull base meningiomas that exhibited symptomatic progression temporally associated with pregnancy.

All lesions involved the paraclinoid or cavernous sinus region and were WHO Grade I on pathology. Two tumors demonstrated progesterone receptor positivity, and molecular analysis identified PIK3CA mutations in two cases. Patients presented with visual disturbance, cranial neuropathy, or tumor recurrence during pregnancy. Surgical management included frontotemporal craniotomy with anterior clinoidectomy or subtotal resection when cavernous sinus involvement limited safe removal.

Despite radiographic progression during pregnancy, all patients achieved favorable maternal outcomes, and no adverse fetal outcomes were observed. These cases highlight the potential for pregnancy-associated meningioma acceleration, particularly in skull base lesions, and underscore the importance of multidisciplinary management and individualized timing of surgical intervention.

Introduction

Meningiomas are the most common primary intracranial tumors in women and frequently express progesterone receptors. Although typically slow-growing, case reports suggest that pregnancy may accelerate tumor progression due to hormonal fluctuations, increased blood volume, and tumor-associated edema.

Skull base meningiomas, particularly those involving the paraclinoid and cavernous sinus regions, present unique challenges given proximity to the optic apparatus and internal carotid artery. Symptomatic progression during pregnancy creates complex management decisions balancing maternal neurologic risk with fetal safety.

The literature remains limited to isolated reports, and optimal timing of surgical intervention during pregnancy is not well defined.

Even benign WHO Grade I meningiomas may demonstrate clinically significant progression during pregnancy.

Methods and Materials

A retrospective review was performed of three pregnant patients with radiographic and/or symptomatic progression of intracranial meningiomas. Clinical presentation, gestational timing, imaging characteristics, operative details, histopathology, molecular profiling, and maternal-fetal outcomes were analyzed.

Serial MRI studies were reviewed to assess interval tumor growth relative to pregnancy. Surgical timing and extent of resection were evaluated in the context of multidisciplinary management involving neurosurgery and high-risk obstetrics.

Variable	Case 1	Case 2	Case 3
Age	40	33	41
Gestational Timing	Postpartum	37 weeks	Early pregnancy
Location	Left spheno-cavernous	Left spheno-cavernous	Cavernous sinus
Optic/ICA Involvement	Yes	Yes	Yes
WHO Grade	I	I	I
PR Status	Positive	Not reported/Negative	Positive
PIK3CA Mutation	Present	Not tested/Absent	Present
Extent of Resection	GTR	STR	STR
Maternal Outcome	No deficit	No deficit	No deficit
Fetal Outcome	Favorable	Term delivery	Favorable

Table 1. Clinical and Pathologic Characteristics of Pregnancy-Associated Meningiomas

Results

Tumor Characteristics
- Skull base location in all cases
- WHO Grade I in 3/3
- PR positive in 2/3
- PIK3CA mutation in 2/3

Management
- Pterional approach ± anterior clinoidectomy
- Subtotal resection when cavernous sinus involved

Outcomes
- No permanent neurologic deficits
- Favorable maternal and fetal outcomes

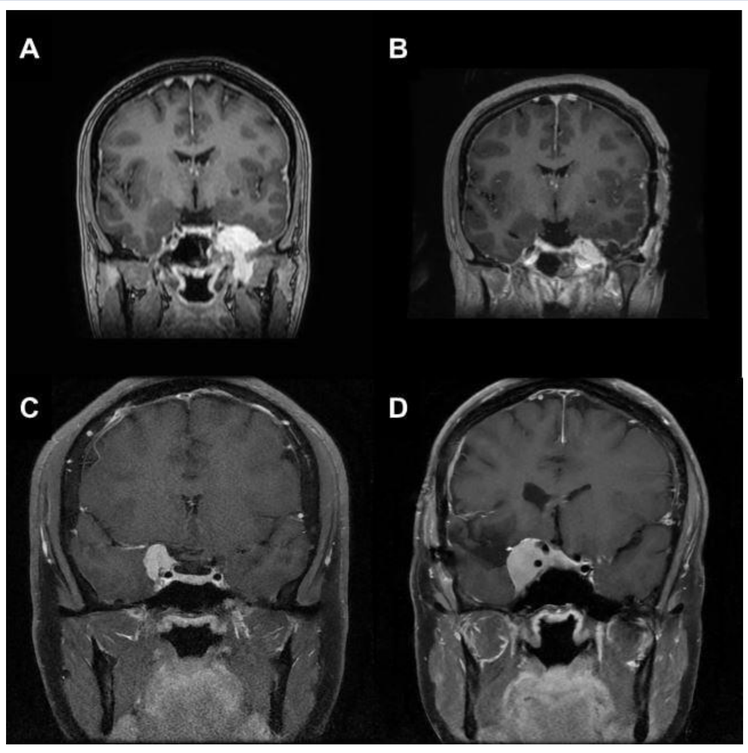

Figure 1. Pre- and postoperative imaging of pregnancy-associated cavernous sinus meningiomas.
(A) Preoperative left spheno-cavernous meningioma.
(B) Postoperative imaging demonstrating interval resection.
(C) Preoperative cavernous sinus meningioma.
(D) Postoperative imaging showing residual tumor within the cavernous sinus.

Discussion

This series highlights the potential for pregnancy-associated acceleration of otherwise indolent WHO Grade I meningiomas. All lesions involved the skull base, where limited surgical corridors and proximity to the optic apparatus and internal carotid artery increase management complexity.

Hormonal influence is supported by progesterone receptor expression in two cases and aligns with prior literature suggesting progesterone-mediated tumor proliferation. Increased vascularity and hemodynamic changes during pregnancy may further contribute to tumor expansion and symptomatic progression. The identification of PIK3CA mutations in two patients raises the possibility that molecular drivers may interact with hormonal signaling pathways.

Management decisions during pregnancy require careful balancing of maternal neurologic risk against fetal safety. In this series, surgical intervention was safely performed when clinically indicated, with favorable maternal and fetal outcomes. Subtotal resection was appropriately chosen when cavernous sinus involvement limited safe gross total removal.
These cases reinforce the importance of close surveillance of known meningiomas in women of reproductive age and emphasize the need for multidisciplinary coordination between neurosurgery and high-risk obstetrics.

Conclusions

- Pregnancy may be associated with accelerated growth or symptomatic progression of otherwise benign WHO Grade I meningiomas.
- Skull base location increases clinical complexity due to optic nerve and internal carotid artery involvement.
- Hormonal receptor expression and molecular alterations such as PIK3CA mutation may contribute to tumor behavior during pregnancy.
- With multidisciplinary coordination and individualized timing of intervention, favorable maternal and fetal outcomes are achievable.

Contact

Ali O. Jamshidi, MD
Kaiser Permanente Medical Center, Woodland Hills
5601 De Soto Ave, Woodland Hills, CA 91367
ALI.O.JAMSHIDI@kp.org
818-719-2000

References

1. Lusis EA, Scheithauer BW, Yachnis AT, et al. Meningiomas in pregnancy: a clinicopathologic study of 17 cases. *Neurosurgery*. 2012;71(5):951–961.
2. Pravdenkova S, Al-Mefty O, Sawyer JR, Husain M. Progesterone and estrogen receptors: opposing prognostic indicators in meningiomas. *J Neurosurg*. 2006;105(2):163–173.
3. Perry A, Louis DN, Budka H, von Deimling A, Sahm F. Meningioma. In: WHO Classification of Tumours of the Central Nervous System. 5th ed. Lyon: IARC; 2021.
4. Claus EB, Bondy ML, Schildkraut JM, et al. Epidemiology of intracranial meningioma. *Neurosurgery*. 2005;57(6):1088–1095.
5. Hortobágyi T, Bencze J, Varkuti B, et al. Meningioma recurrence. *Open Med (Wars)*. 2016;11:168–173.
6. Mawrin C, Perry A. Pathological classification and molecular genetics of meningiomas. *J Neurooncol*. 2010;99(3):379–391.
7. Peyre M, Stemmer-Rachamimov A, et al. PIK3CA mutations in meningioma: molecular characterization and clinical relevance. *Acta Neuropathol*. 2017;134(6):1021–1023.
8. Stevenson CB, Thompson RC. Management of intracranial tumors during pregnancy. *Clin Obstet Gynecol*. 2013;56(3):562–575.

© Poster Template by Genigraphics® 1.800.790.4001 www.genigraphics.com