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A Case Report of Sporadic Cerebellar Hemangioblastoma Post Cesarean Section

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ABSTRACT

Background: Hemangioblastomas are rare, benign tumors of the central nervous system. Usually they develop sporadically, but 25% are associated with von Hippel-Lindau disease. Symptoms include headache, nausea, vomiting, dizziness, and sensory/motor deficits.

Case: 36-year-old patient G2P2002 presented 5 weeks after cesarean section endorsing headaches, dizziness, ataxia, nausea, vomiting. CT brain revealed a 4.9cm lesion consistent with a cerebellar hemangioblastoma, obstructive hydrocephalus and tonsillar herniation. The patient underwent emergent surgical resection with complete resolution of symptoms.

Conclusion: Cerebellar hemangioblastomas may exhibit growth during pregnancy due to increased blood volume, growth factors, and hormonal changes. Physicians should include cerebellar hemangioblastoma in their differentials as it can mimic classical pregnancy pathologies. To our knowledge, this is the first reported case of sporadic cerebellar hemangioblastoma presenting shortly after pregnancy.

INTRODUCTION

Hemangioblastomas are benign, slow growing, highly vascularized tumors that make up less than 3% of all primary tumors of the central nervous system. They usually occur sporadically in the posterior fossa of adults, but can occur as a component of von Hippel-Lindau (VHL) disease 20-30% of the time. Von Hippel-Lindau is an inherited, autosomal dominant disease characterized by the formation of tumors throughout the body, such as hemangioblastomas, renal cell carcinomas, pheochromocytomas, retinal angiomas, and pancreatic cysts. Sporadic hemangioblastomas make up about 75% of hemangioblastomas, are usually solitary, and diagnosed in the fourth or fifth decade of life, later than in patients with VHL. The typical symptom manifestations depend on the location and size of the tumor, but they commonly cause headache, nausea, vomiting, dizziness, ataxia, oculomotor nerve dysfunction, and sensory/motor deficits due to the mass effect of the tumor compressing neural structures, edema, obstructive hydrocephalus, or hemorrhage. Histologically, these tumors are made of neoplastic stromal cells of unknown origin with large, dark nuclei and lipid containing vacuoles dispersed throughout abundant cytoplasm. These clear cells contrast with the vast, thin-walled vascular network that gives these tumors their deep red appearance, after which they derived their name. Rosenthal fibers may also be seen due to long standing gliosis **(Figure 1)**. Surgical removal is the treatment of choice, particularly in sporadic hemangioblastomas, but radiation therapy and antiangiogenesis therapy has shown increasing promise for tumors refractory to surgery, or in patients with multiple tumors [1].

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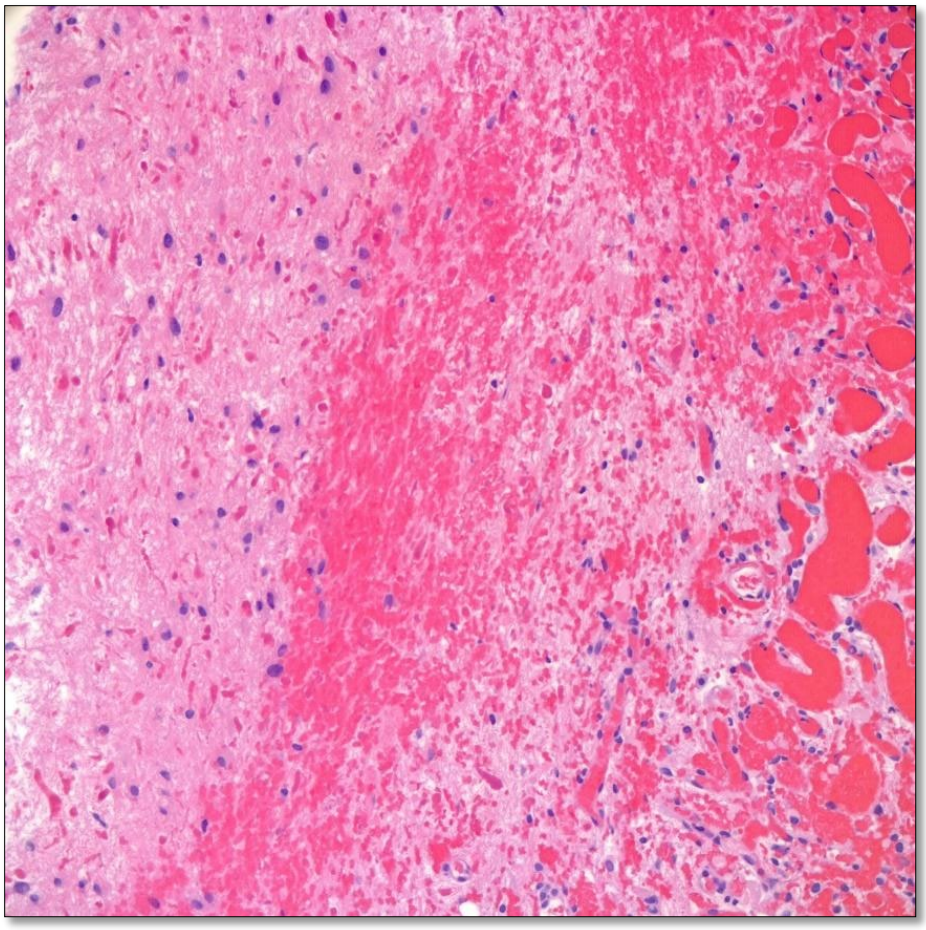

Figure 1. Pathological sample magnified 200x demonstrating stromal cells with lipid filled cytoplasm, capillary proliferation, and Rosenthal fibers, associated with gliosis.

CASE

Patient is a 36-year-old patient G2P2002 with past medical history positive for hypertension and obesity and no significant family history who presented to labor and delivery at 39 weeks gestation for a repeat cesarean section with bilateral salpingectomy. Her postpartum course was uncomplicated, and she was discharged home on postoperative day 2. She presented to the ED 38 days later for intermittent headaches and dizziness for 2 weeks. At the ED, she received acetaminophen 1000 mg, prochlorperazine 10 mg injection, and hydralazine 10 mg, and was discharged the same day with nifedipine 30 mg, ibuprofen 600 mg, and ondansetron 4 mg. She was instructed to follow up with her PCP and OBGYN.

Three days later, she presented to the OBGYN with headache, nausea, vomiting, dizziness, unsteady gait, and hypertension. She was referred to the ED for immediate management of suspected preeclampsia. Upon arrival her BP was 162/108, her urine protein/creatinine ratio was WNL, but her WBC count, RBC count, and hematocrit were slightly elevated. She received $MgSO_4$ for 24 h for seizure prophylaxis and labetalol and nifedipine for sustained hypertension. She was sent home the next day with nifedipine 30 mg after symptom improvement.

Four days later, she presented to the OBGYN for a follow up visit with worsening of her previous symptoms, now with altered mental status. She was once again referred to the ED for suspected pre-eclampsia. Brain CT scan findings were positive for a 4.9cm intra axial posterior fossa cystic lesion involving the right cerebellum with significant mass effect on the fourth ventricle, brainstem, and cerebral aqueduct, causing acute obstructive hydrocephalus and transtentorial and tonsillar herniation **(Figure 1).** Subsequent characterization with MRI demonstrated a 4.7 x 4.6 x 4.1 cm mass with a 0.9cm enhancing mural nodule along the right superolateral margin along the mass, a finding suggestive of a hemangioblastoma **(Figures 2 & 3)**. There was a 12 mm downward cerebellar tonsillar herniation below the level of the foramen magnum. The next day, the patient underwent a craniectomy with for excision of a tumor in the posterior fossa and an external ventricular drain with a long tunnel emerging from the chest.

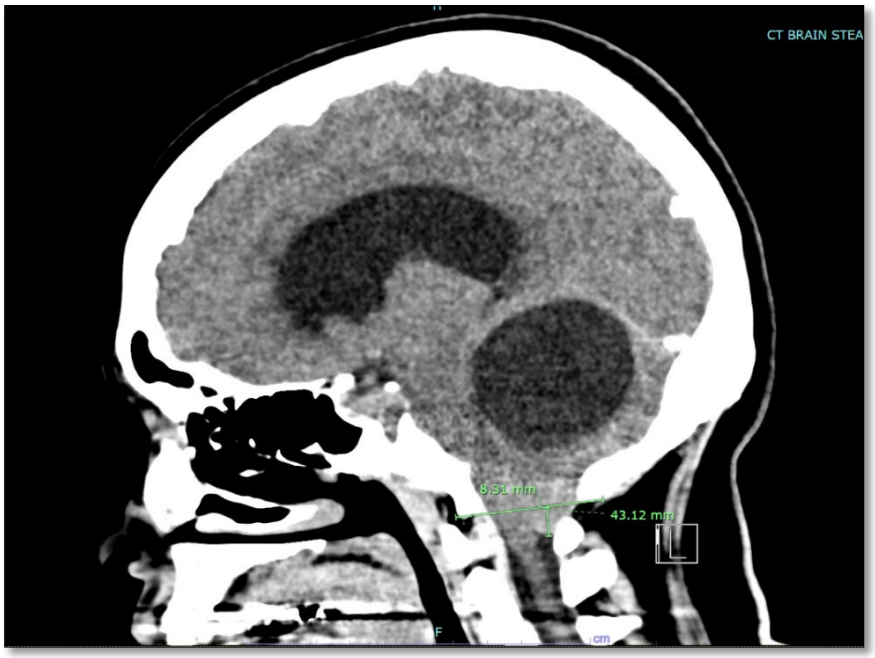

Figure 2. Preoperative CT brain demonstrating 4.9 cm intra-axial posterior fossa lesion causing significant mass effect, hydrocephalus, and transtentorial and tonsillar herniation.

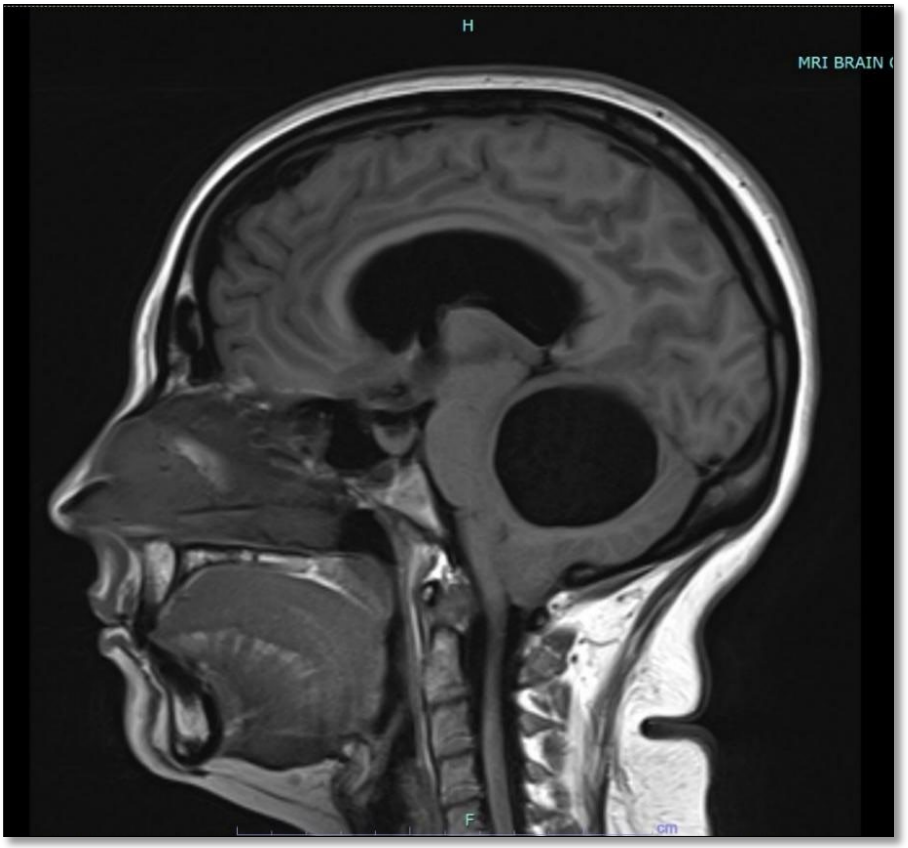

Figure 3. Preoperative MRI brain redemonstrating 4.7 x 4.6 x 4.1 cm mass with a 0.9cm mural nodule along the right superolateral margin along the mass, suggestive of a hemangioblastoma.

Repeat brain CT after surgery on post-operative day 0 showed improvement of mass effect on the brainstem and posterior fossa with decreased downward tonsillar herniation and expansion of the fourth ventricle **(Figure 4)**. On post-operative day 3, the patient denied headache, dizziness, nausea, and vomiting. She was discharged on post-operative day 6 with full resolution of her symptoms and no residual deficits.

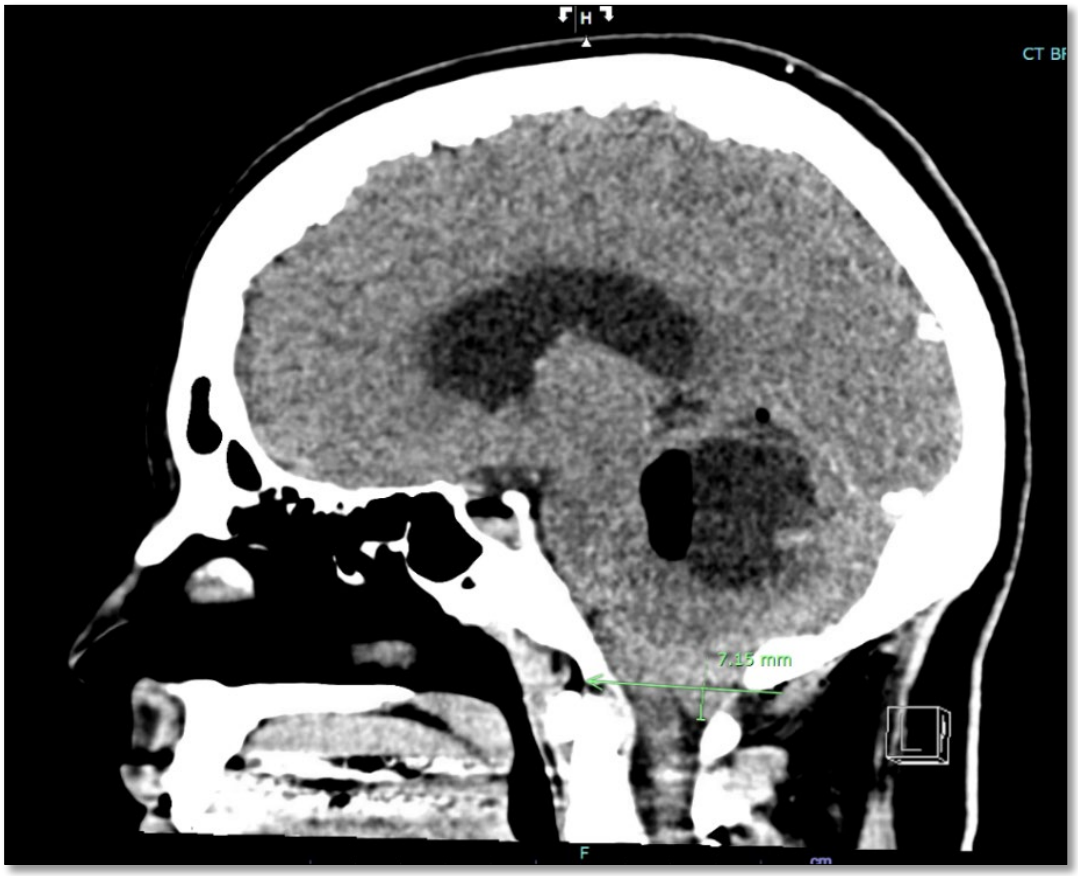

Figure 4. Postoperative CT brain demonstrating resection cavity in the posterior fossa at the site of previous mass and decreased tonsillar herniation.

DISCUSSION

Thorough clinical examination and differential diagnosis during pregnancy is of great importance due to the possibility of rare diseases that may manifest similarly to diseases of pregnancy, such as preeclampsia. Availability bias is one obstacle that can obscure a physician's ability to consider all possible differentials, especially when more common diagnoses associated with the physiology of pregnancy take up the majority of their focus. In current literature, conflicting evidence exists regarding the progression of hemangioblastomas during pregnancy compared to non-pregnant patients. Some studies suggest growth of hemangioblastomas during pregnancy, citing the increased maternal blood volume and hormonal changes of pregnancy as potential causes of tumor development. Laviv [2], Frantzen [3] & Özdem [4] presented a case report that strongly suggested that pregnancy encouraged their growth when he discussed a patient that had the cystic portion of a sporadic cerebellar hemangioblastoma aspirated during her seventh month of pregnancy, only to return 17 days later with a recurrence of the cyst to 80% of its original size. Laviv [2] & Özdem [4] reported that the physiological increase in placental growth factor and its receptor, vascular endothelial growth factor receptor 1, stimulate the growth of peritumoral cysts, which are responsible for the mass effect and obstructive symptoms caused by cerebellar hemangioblastomas.

Furthermore, it is well established that estrogen induces endothelial cell migration and proliferation [5], and this may provide another potential pathway for the progression of existing hemangioblastomas. Beyond estrogen, Brown [6] found that hemangioblastomas demonstrated progesterone receptor immune-reactivity and offered another pathway that pregnancy hormones may stimulate hemangioblastoma development, especially later in pregnancy when progesterone peaks. Yet another mechanism that could explain progression of hemangioblastomas during pregnancy is CXCR4 up regulation. CXCR4 levels physiologically

increase during pregnancy for its role in placental development, and Chatterjee [7] found that CXCR4 over expression is associated with tumor growth and angiogenesis.

Interestingly, Ye [5] found that pregnancy was not associated with hemangioblastoma progression, however, the same study found that the growth of peritumoral cysts seen with hemangioblastomas was significantly increased for patients in the pregnancy cohort during the time that they were not pregnant, when compared to the non-pregnant cohort. While this peculiar finding is not fully understood, it adds to the significance of the present case, who only experienced symptoms of her cerebellar hemangioblastoma several weeks postpartum. Current literature surrounding cerebellar hemangioblastomas during pregnancy focuses on patients with VHL, necessitating further research on sporadic cerebellar hemangioblastomas, to better elucidate the pathophysiology of the latter and their association with pregnancy [8].

CONCLUSION

We report a case of a 36-year-old patient with symptomatic exacerbation of a sporadic cerebellar hemangioblastoma several weeks after the cesarean birth of her child. This case is unique because, to our knowledge, it is the first reported case of a sporadic cerebellar hemangioblastoma associated with pregnancy causing obstructive hydrocephalus that presented shortly after an uncomplicated pregnancy. Following a craniectomy for excision of the cerebellar hemangioblastoma and an external ventricular drain for resolution of obstructive hydrocephalus, the patient made a full recovery and, upon follow up, had complete resolution of her symptoms.

REFERENCES

1. Wong ET, Joseph J, Wu JK, Wen PY, Tung GA, et al. (2024) Hemangioblastoma. UpToDate.
2. Laviv Y, Wang JL, Anderson MP, Kasper EM (2019) Accelerated growth of hemangioblastoma in pregnancy: The role of proangiogenic factors and upregulation of hypoxia-inducible factor (HIF) in a non-oxygen-dependent pathway. Neurosurg Rev 42: 209-226.
3. Frantzen C, Kruizinga RC, van Asselt SJ, Zonnenberg BA, Lenders JW, et al. (2012) Pregnancy-related hemangioblastoma progression and complications in von Hippel-Lindau disease. Neurology 79(8): 793-796.
4. Özdem N, Arslan I, Faz MG (2012) Recurrence of Cystic Part of Cerebellar Hemangioblastoma at Early Postoperative Period and its Spontaneous Resolution: A Pregnant Patient with Serial Magnetic Resonance Imaging Findings. Neurol Sci Neurophysiol 29(2): 385-392.
5. Ye DY, Bakhtian KD, Asthagiri AR, Lonser RR (2012) Effect of pregnancy on hemangioblastoma development and progression in von Hippel-Lindau disease: Clinical article. J Neurosurg 117(5): 818-824.
6. Brown DF, Dababo MA, Hladik CL, Eagan KP, White CL, et al. (1998) Hormone receptor immunoreactivity in hemangioblastomas and clear cell renal cell carcinomas. Modern Pathol 11(1): 55-59.
7. Chatterjee S, Behnam Azad B, Nimmagadda S (2014) The intricate role of CXCR4 in cancer. Adv Cancer Res 124: 31-82.
8. Oviedo PJ, Sobrino A, Laguna-Fernandez A, Novella S, Tarín JJ, et al. (2011) Estradiol induces endothelial cell migration and proliferation through estrogen receptor-enhanced RhoA/ROCK pathway. Mol Cell Endocrinol 335(2): 96-103.