



Ruptured Intracranial Dermoid Cyst: A Rare Cause of Post-Partum Seizures

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Abstract

In this case report we will discuss a 34-year-old female who presented six months post-partum with headaches, auras and burning in her arm. Progressive weakness ultimately lead to confusion and generalized tonic-clonic seizure on arrival to the emergency department. After stabilization, she was found to have a ruptured, intracranial dermoid cyst which had spread sebum throughout her central nervous system. She developed chemical meningitis as a result, and had a myriad of neurological symptoms including word finding difficulties, decreased sensation and seizures. Her cyst was resected and she has been doing well. In a post-partum patient, ruptured intracranial dermoid cyst is an abnormal cause of headaches and seizures. Going unrecognized, severe neurological outcomes are probable. In this case report we will discuss her presentation, identification of her mass and therapeutic measures undertaken.

Case Presentation

A 34-year-old female was brought to the Emergency Department (ED) for right sided hemiparesis, expressive aphasia and confusion. She was six months post-partum and had a history of depression, GERD, chronic urinary tract infections and pyelonephritis. For the past three months she began to have daily headaches characterized as dull and worse in the afternoon without associated nausea/vomiting or visual changes/auras. Over the past week she had been having difficulty holding her baby due to sensations of extreme heat in her right arm with associated weakness. The day she presented she was found by her neighbors banging her hand against a wall and confused.

Upon arrival to an outlying ED, she experienced two episodes of generalized tonic-clonic seizures with rightward gaze preference, each lasting approximately four minutes. Her seizures were treated with diazepam and a loading dose of Levetiracetam. Head CT demonstrated a 4.4cm extra-axial mass extending from the suprasellar region with mass effect on right basal ganglia, right lateral and third ventricles (Figure 1). There was also the presence of sebum in the subarachnoid space and ventricles.

Upon arrival in our ED at the tertiary care referral center, her symptoms and mental status had improved. She was able to provide further history given above and initial vitals were as follows: T 37C (98.6F), HR 81, Resp 19, BP 118/81, SpO2 98% on room air. Exam was remarkable for 5/5 strength in all extremities, but scattered areas of decreased sensation on the dorsum of the right hand, arm and foot. Her cranial nerves were intact, her speech was remarkable for subtle word finding difficulties though she remained alert and oriented to person, time and place. She went

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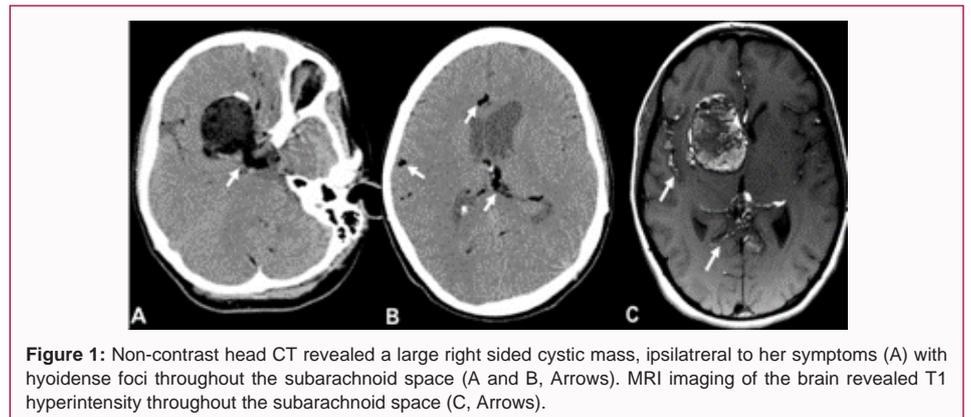


Figure 1: Non-contrast head CT revealed a large right sided cystic mass, ipsilateral to her symptoms (A) with hypodense foci throughout the subarachnoid space (A and B, Arrows). MRI imaging of the brain revealed T1 hyperintensity throughout the subarachnoid space (C, Arrows).

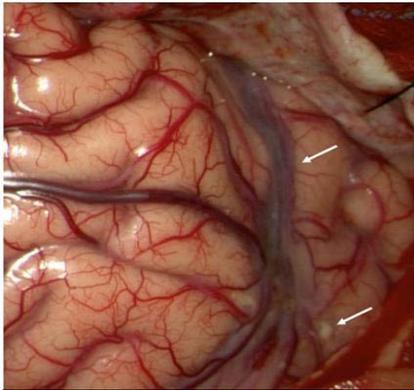


Figure 2: Surgical resection of the mass revealed sebum in the subarachnoid space (E, Arrow).

for MRI with findings likely representing ruptured dermoid cyst with significant FLAIR signal abnormalities within the left cerebral hemisphere (Figure 2).

She was admitted to the neurosurgical service for a planned resection. Over the course of her hospitalization she developed SIADH which was successfully treated with salt tablets. Her cyst was resected without incident and she was discharged on post-op day 6.

Discussion

Intracranial dermoid cysts are benign growths most commonly found in the supra/parasellar, or posterior fossa regions. They characterize only 0.04% to 0.6% of all intracranial neoplasms [1-4]. They are most commonly discovered during the third to fifth decades of life and usually incidentally, after spontaneous or traumatic rupture or when symptoms present from mass-effect. Average sizes tend to be 4 cm to 4.5cm and they grow overtime from the secretion of sebaceous material into the cyst, along with sloughing of epidermal cells [1,5]. The clinical signs of rupture include headaches, seizures, sensory or motor hemiparesis and chemical meningitis which may have sequelae of cerebral ischemia and possibly death [3,4]. While this case reports a 34-year-old female presenting with classic features of headache, altered mental status, seizures and hemisensory deficits after spontaneous rupture of intracranial dermoid cyst; the existence of these symptoms in a post-partum female and the clinical correlation of neurologic exam findings in relation to neuroimaging findings made this case unique.

Dermoid cysts have characteristic CT and MRI findings. On CT (Figure 1) they have well-demarcated and hypodense appearances sometimes seen with calcified borders [2,6]. On T1-weighted MRI sequencing they reveal a hyperintense cyst which is secondary to the high cholesterol content of the cyst [1,6]. They will typically appear heterogeneous on T2-weighted images (Figure 1). The fat intensity signal witnessed on MRI is secondary to sebaceous secretions and cholesterol [1,4,6]. When a dermoid tumor ruptures, fat droplets-appearing hypodense on CT or hyperintense on T1 MRI-may be seen scattered and floating within the nondependent portions of the ventricular system and/or subarachnoid space. Recognition of rupture makes the availability of a dedicated neuroradiologist invaluable as this is a rare phenomenon - 5 out of 2707, or 0.18% of all new CNS tumors operated on during a 12-year period at a major tertiary care center [8]. Furthermore, rupture can have grave clinical consequence due to associated aseptic chemical meningitis that

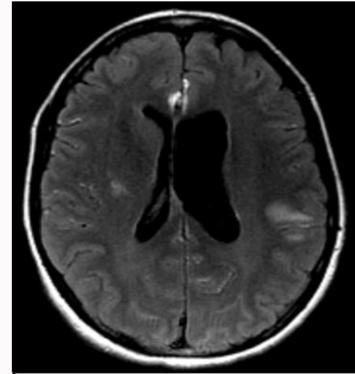


Figure 3: FLAIR hyperintensity of MRI within the left (contralateral) cerebral hemisphere (D, *) suggesting subarachnoid spread of sebum and cerebral edema as an etiology for her symptoms.

produces irritative effects from the disseminated cholesterol debris [9] (Figure 2). Chemical meningitis can also lead to cerebral ischemia due to vasospasm with resultant infarction and death [10,11]. Our patient had evidence of all of the classic findings associated with a ruptured intracranial dermoid cyst.

Clinically in a patient with no history of epilepsy, post-partum seizures are generally eclampsia until proven otherwise. Despite the common misconception that delivery is the 'cure' for preeclampsia and eclampsia, there have been reported seizures from eclampsia up to 5 weeks post-partum [7]. This patient was 6 months post-partum however and given the patient's blood pressure and CT/MRI findings usual etiologies such as eclampsia, cavernous sinus thrombosis and CNS infections became unlikely diagnoses in her case.

After her seizures the patient in the ED only had subtle right arm sensory deficits and expressive aphasia. In a report by El-Bahy et al. [12] symptoms seen after cyst rupture are typically - headache (32.6%), seizures (26.5%), cerebral ischemia with sensory and/or motor hemisindrome (16.3%) and aseptic meningitis (8.2%). Yet, the inconsistency observed clinically was that her sensory deficits were ipsilateral to her cortical lesion seen on imaging. Pathophysiologically this could only be explained by three main mechanisms - mass effect on neural structures, subarachnoid spread from sebum and cerebral edema from cyst content or the effect of hydrocephalus. The mass effect seen on imaging is actually on the R sided cortical tracts and if affected these fibers would decussate in the medulla and affect structures on the L side and thus not likely. Though obvious mass effect was seen on imaging the slow growth of this lesion most likely allowed structures to shift appropriately without clinical effect. This slow growth is also the likely reason why there is no associated hydrocephalus and thus an unlikely explanation of R sided symptoms. Figure 3 shows clear edema in the contralateral L cerebral hemisphere with provides a plausible explanation for R sided sensory deficits, subtle expressive aphasia appreciated on exam alongside a potential nidus for the initiation of generalized tonic clonic seizure. Postictal Todd's paralysis is also plausible but the MRI findings make localized cerebral edema a more likely culprit. In conclusion, an emergent MRI in this patient really helped settle the discrepancy between the clinical and imaging findings and reveal the etiology of symptoms not apparent on CT imaging.

The timing of her symptoms made her post-partum state clinically intriguing. Her headaches first started approximately

three months ago, followed by heat sensations in her arm all the way to her acute worsening of headache associated with seizures that led to her eventual presentation. The exact pathophysiology of dermoid cyst rupture remains poorly understood. Hypotheses have included glandular secretions due to age dependent hormones [13], trauma as well as head movements and brain pulsations [14]. To the best of these authors knowledge, pregnancy and the well described post-partum hormonal changes have not been well described as a possible etiology for spontaneous intracranial dermoid cyst rupture. Furthermore the literature reports that symptom onset from time of rupture can vary anywhere from three months to 6.5 years since the irritative effects of leaked contents require time to develop [15]. It is hypothetically possible that the known post-partum hormonal changes and specifically their known impact on neuro-hormonal balance may have led to the rupture of this cyst and/or accelerated the manifestations of cyst rupture such as headache, aseptic/chemical meningitis, seizures and cerebral edema.

Conclusion

Intracranial dermoid cyst rupture is a rare etiology of headache, seizures, aseptic/chemical meningitis and neurological deficits. Due to its rare incidence, a multi-disciplinary approach including acute seizure and headache management in the ED, prompt diagnosis on advanced neuroimaging such as MRI by seasoned neuroradiologists and definitive management via resection from an experienced neurosurgery team is of paramount importance. Pregnancy may or may not play a role in the pathophysiologic mechanism of cyst rupture and the rate of manifestations of its associated symptoms.

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