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Hypothalamic Hamartoma Presenting With Gelastic and Dacrystic Seizures at Three Months of Age: A Case Report

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Abstract

Hypothalamic hamartomas (HH) are rare congenital, non-neoplastic malformations composed of disorganized but mature neuronal and glial tissue native to the hypothalamic region. They are strongly associated with gelastic and dacrystic seizures and may also present with additional focal seizure types and cognitive, behavioral, sleep, and endocrine manifestations. We report the case of a two-year-old girl who had experienced recurrent, stereotyped, context-inappropriate episodes of laughter and crying since three months of age. These early manifestations were initially not recognized as epileptic. Over time, additional focal seizure types emerged, characterized by behavioral arrest, impaired consciousness, automatisms, right-sided clonic activity, ocular deviation, and tonic posturing. Brain magnetic resonance imaging demonstrated a non-enhancing lesion involving the floor of the third ventricle and hypothalamic region, consistent with HH. Four months after diagnosis, the patient was admitted because seizure frequency had increased to six to nine episodes per day despite antiseizure treatment. Her pharmacological regimen was adjusted, with a short-term reduction in seizure frequency during hospitalization. Following neurosurgical evaluation, lesion-directed intervention was deferred, and clinical and neuroimaging follow-up was recommended. Long-term outcomes could not be established because the patient was subsequently lost to follow-up. This case highlights that recurrent, stereotyped, context-inappropriate episodes of laughter or crying beginning in early infancy should prompt neurological evaluation and high-resolution brain MRI with careful assessment of the hypothalamic region, particularly when additional focal seizure manifestations subsequently emerge.

Categories: Neurology, Radiology, Pediatrics
Keywords: colombia, dacrystic seizures, epilepsy in children, focal epilepsy, gelastic seizures, hypothalamic hamartoma, hypothalamic tumor, pediatric refractory epilepsy

Introduction

Gelastic and dacrystic seizures are uncommon, typically brief seizure manifestations characterized by inappropriate laughter and crying, respectively [1]. In the 2025 International League Against Epilepsy (ILAE) classification, gelastic and dacrystic manifestations are described as mimic automatisms within the complex motor semiological descriptors used to characterize focal seizures [2]. They may occur in isolation or in clusters and have been associated with epileptogenic networks involving the temporal, frontal, and parietal regions, as well as with hypothalamic hamartomas (HH) [1].

HH are rare congenital, non-neoplastic malformations composed of disorganized but mature neuronal and glial tissue in the hypothalamic region, most commonly involving the inferior hypothalamus and tuber cinereum [3]. Their clinical presentation is heterogeneous, ranging from asymptomatic lesions to epilepsy, central precocious puberty, cognitive impairment, behavioral disturbances, and sleep disorders [4,5]. Reported frequency estimates range from approximately one in 50,000 to one in 1,000,000 individuals [6]. Clinical manifestations generally begin in early childhood, although the age at onset varies according to the presenting phenotype [7].

Gelastic seizures are among the most characteristic epileptic manifestations of HH and have been reported in a high proportion of affected patients [8]. As the epileptic network evolves, additional seizure types may emerge, sometimes accompanied by impaired consciousness and increasing seizure burden [9]. Reported seizure types include dacrystic, atonic, tonic, atypical absence, and focal-to-bilateral tonic-clonic seizures [4,9,10]. Other ictal manifestations may include automatisms, autonomic symptoms, epigastric sensations, déjà vu, and focal motor features [6].

Although the association between HH and gelastic seizures is well established, recognition can be challenging when seizures begin very early in infancy and their semiology evolves over time. This case is noteworthy because gelastic and dacrystic seizures began at three months of age and were subsequently followed by additional focal seizure types, contributing to delayed recognition of the underlying HH. By

presenting this clinical course, we aim to emphasize the importance of considering HH in infants with recurrent, stereotyped, context-inappropriate episodes of laughter or crying, particularly when additional focal seizure manifestations emerge, in order to facilitate earlier diagnosis and appropriate management.

Case Presentation

A two-year-old girl presented with recurrent episodes of spontaneous laughter and crying that had begun at three months of age and were initially not investigated. These episodes were brief, stereotyped, and inappropriate to the surrounding context, consistent with gelastic and dacrystic seizures. Over time, additional focal seizure types emerged. She developed focal seizures with impaired consciousness characterized by staring and behavioral arrest, followed by right-hand gestural automatisms, lasting less than one minute. She also presented focal seizures with impaired consciousness characterized by behavioral arrest followed by clonic activity involving predominantly the right hemibody, including the right hemiface and perioral region, associated with rightward ocular deviation and lasting approximately 15 seconds. A further seizure type consisted of impaired consciousness followed by unprovoked crying and tonic posturing with flexion of the right upper limb, lasting approximately 15 seconds.

She received a diagnosis of focal epilepsy. A six-hour video-EEG showed generalized-appearing interictal epileptiform activity, characterized by high-amplitude, bilaterally synchronous spike-and-wave and polyspike-and-wave discharges, without consistent focal predominance on scalp EEG. In the clinical context of HH-associated epilepsy, these findings were not considered sufficient to support a diagnosis of primary generalized epilepsy (Figure *1*).

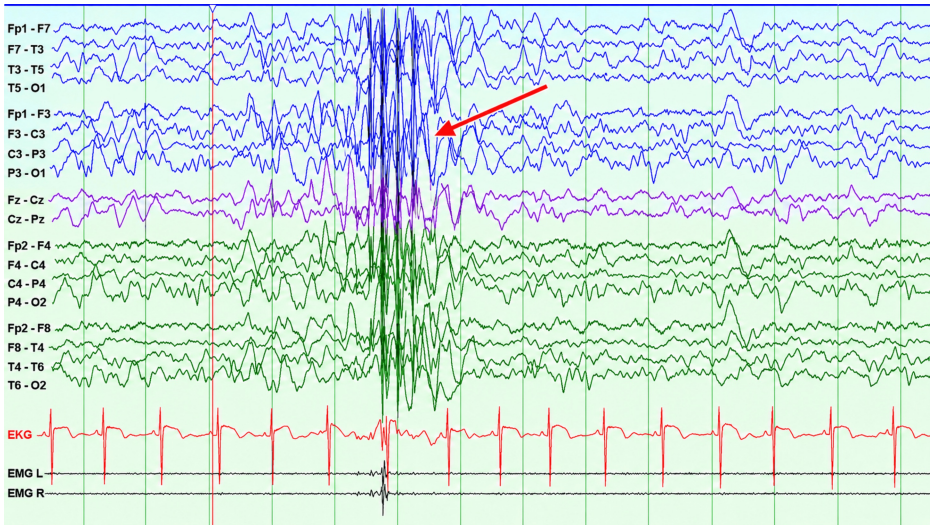


FIGURE 1: Representative interictal epileptiform abnormalities on six-hour video-electroencephalography (EEG)

Representative bilaterally synchronous polyspike-and-wave discharges without consistent focal predominance on scalp EEG. These epileptiform abnormalities exhibited a generalized-appearing distribution in a patient with structural focal epilepsy associated with a hypothalamic hamartoma. The red arrow indicates a representative epileptiform discharge.

Technical parameters: Longitudinal bipolar montage (double banana); sensitivity: 7 µV/mm; time base: 30 mm/s; low-frequency filter (LFF): 1 Hz; high-frequency filter (HFF): 70 Hz; notch filter: 60 Hz (enabled).

Brain magnetic resonance imaging (MRI) demonstrated a non-enhancing left parasagittal lesion involving the floor of the third ventricle, with attachment to the hypothalamic region near the tuber cinereum and mammillary bodies. The lesion measured approximately 6.9 × 6.9 × 5.4 mm and was identified on T1-weighted sagittal, coronal, and axial images. Its anatomical location and attachment were consistent with a hypothalamic hamartoma (Figure *2*).

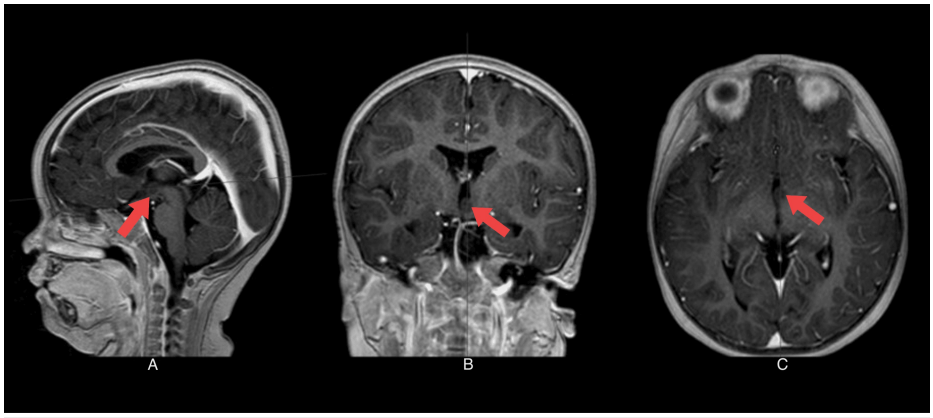


FIGURE 2: T1-weighted magnetic resonance imaging findings of a hypothalamic hamartoma

(A) Sagittal MRI showing a small lesion involving the floor of the third ventricle, with attachment to the hypothalamic region near the tuber cinereum and mammillary bodies; (B) Coronal MRI demonstrating the left parasagittal location of the lesion and its relationship with the hypothalamic floor; (C) Axial MRI illustrating the lesion in relation to adjacent hypothalamic structures.

The lesion measured approximately 6.9 × 6.9 × 5.4 mm. No appreciable contrast enhancement was reported. Its anatomical location and attachment are consistent with a hypothalamic hamartoma. Arrows indicate the hypothalamic lesion.

Following neurosurgical evaluation, lesion-directed intervention was deferred, and clinical follow-up with serial neuroimaging was recommended. Neurology initiated outpatient antiseizure therapy with levetiracetam at 40 mg/kg/day and topiramate at 3.2 mg/kg/day. Four months later, the mother brought her to the pediatric emergency department due to increased seizure frequency (six to nine per day over the preceding week).

On admission, the patient was alert and in good general condition. Central hypotonia was noted, with no other abnormalities on neurological examination. She also had raised hyperpigmented lesions on the thighs and extremities related to previously diagnosed cutaneous mastocytosis, for which she was receiving tapering prednisone and loratadine.

Pediatric neurology considered the patient to have structural focal epilepsy associated with HH, characterized initially by gelastic and dacrystic seizures, followed by additional focal seizure types, based on the clinical presentation and neuroimaging findings. Oxcarbazepine was added and progressively titrated to 30 mg/kg/day, topiramate was increased to 5 mg/kg/day, and levetiracetam was continued at 40 mg/kg/day. During hospitalization, irritability and insomnia were documented, and melatonin was initiated.

The patient was re-evaluated by neurosurgery. Lesion-directed intervention remained deferred, and clinical follow-up with annual brain MRI was recommended. The available medical record did not provide a detailed rationale for deferring intervention.

During hospitalization, the patient showed a progressive reduction in seizure frequency and tolerated the prescribed antiseizure medications without documented adverse effects. She was discharged with continuation of pharmacological treatment and outpatient follow-up. However, long-term clinical follow-up was limited because the family lived in another city and was subsequently lost to follow-up; therefore, sustained seizure control, persistence or resolution of gelastic/dacrystic seizures, developmental and behavioral outcomes, sleep disturbances, adverse effects, and endocrine evolution could not be reliably assessed.

Discussion

HH-associated epilepsy has a distinctive clinical spectrum in which gelastic and dacrystic seizures may be the earliest manifestations, followed over time by additional focal seizure types and, in some patients, cognitive, behavioral, and sleep disturbances [4,9,11]. In the present case, gelastic and dacrystic seizures began during early infancy and were subsequently accompanied by focal seizures characterized by impaired consciousness, automatisms, clonic activity, and tonic posturing.

The pathophysiology of gelastic seizures associated with HH remains incompletely understood. However, current evidence supports a model involving small gamma-aminobutyric acid (GABA)ergic neurons within the HH that exhibit spontaneous electrical activity. These neurons project synaptic connections to other

small and large neurons within the HH, which in turn project to structures that play a key role in limbic epilepsies, such as the hippocampus and thalamus [4-6,9]. Additionally, it has been proposed that excessive growth of neurons derived from the lateral tuberal nucleus may lead to disinhibition of neurons of the parvalbumin-containing PV1 nucleus, thereby triggering the laughter associated with gelastic seizures [10].

In most patients with HH, gelastic seizures represent the earliest seizure type and are commonly recognized during late infancy or early childhood, with onset typically around the first year of life. According to established clinical descriptions, these characteristic episodes of inappropriate laughter often prompt the initial diagnosis. Supporting this pattern, a well-characterized clinical cohort study by Pridmore et al. reported that gelastic seizures were the predominant seizure type and that seizure onset occurred before one year of age in more than half of patients, with a mean age of onset of 10 months [11].

In contrast, the remarkably early presentation observed in this case, beginning at three months of age, underscores the need to consider HH even in very young infants presenting with recurrent, stereotyped, context-inappropriate episodes of laughter or crying.

Furthermore, epilepsy may be accompanied by progressive cognitive deterioration, behavioral disorders, and psychiatric symptoms [6,9,10]. Hyperactivity, attention deficits, anger attacks, and aggressive behavior are common [10]. Cognitive impairment has been reported in more than 80% of cases, and its severity has been associated with early onset of epilepsy, higher seizure burden, and greater use of antiseizure medications [6,9,10]. Additionally, epilepsy may disrupt sleep and impair sleep quality [10]. During hospitalization, irritability and insomnia were documented and melatonin was initiated. Given the limited follow-up and potential clinical and pharmacological confounders, these manifestations cannot be definitively attributed to HH-associated epilepsy.

HH is also well associated with central precocious puberty (CPP). CPP related to HH typically presents at an earlier age than idiopathic CPP, with a mean age of onset of 3.7 years in males and 2.5 years in females [6,12]. Its pathophysiology is thought to involve premature activation of pulsatile gonadotropin-releasing hormone (GnRH) secretion [6,9,10]. No clinical signs suggestive of CPP were documented during the available period of observation.

Neuroimaging plays a central role in the diagnosis and anatomical characterization of HH. On MRI, these lesions are typically isointense to gray matter on T1-weighted sequences and isointense or mildly hyperintense on T2-weighted sequences, with no significant contrast enhancement [13]. MRI also defines the relationship of the hamartoma to the floor of the third ventricle, tuber cinereum, mammillary bodies, and adjacent hypothalamic structures, which is important for clinical characterization and treatment planning. Sessile or intrahypothalamic attachments are more commonly associated with epilepsy, whereas pedunculated or parahypothalamic lesions are more frequently associated with central precocious puberty [4,13,14]. In the present patient, the lesion involved the floor of the third ventricle and mammillary region, an anatomical relationship consistent with the epilepsy-associated intrahypothalamic phenotype.

EEG may have limited sensitivity in HH-associated epilepsy because gelastic and dacrystic seizures arise from a deep-seated epileptogenic source and may occur without a clear scalp EEG correlate. Interictal recordings may be normal or may show nonspecific focal, bilateral, or generalized epileptiform abnormalities, particularly as the epileptic network evolves [4]. In the present case, the six-hour video-EEG demonstrated generalized-appearing interictal epileptiform activity, with bilaterally synchronous spike-and-wave and polyspike-and-wave discharges without consistent focal predominance. These findings were interpreted as nonspecific network-level abnormalities rather than evidence of primary generalized epilepsy. In this setting, the bilaterally synchronous discharges may reflect secondary bilateral synchrony or propagation through an HH-related epileptic network rather than primary generalized epilepsy.

High-resolution 3-T epilepsy-protocol MRI is essential for the diagnosis and anatomical characterization of HH. Additional studies such as fluorodeoxyglucose positron emission tomography (FDG-PET), ictal single-photon emission computed tomography (SPECT), and magnetic resonance spectroscopy (MRS) are not recommended as routine components of HH evaluation [14-16].

Management of HH depends on the clinical phenotype, seizure burden, lesion anatomy, and available expertise [10]. Antiseizure medications may provide symptomatic seizure control. Current international consensus supports early evaluation for lesion-directed treatment in patients with HH-associated epilepsy, with intervention recommended after failure of two appropriately selected antiseizure medication regimens [8,15]. Current interventional options include minimally invasive ablative or disconnective procedures, such as stereotactic radiofrequency thermocoagulation (RFTC) and magnetic resonance-guided laser interstitial thermal therapy (MRgLITT); endoscopic disconnection; and, in selected cases, open microsurgical approaches. Stereotactic radiosurgery may also be considered in carefully selected patients, although its therapeutic effect is delayed. The choice of technique depends on the size, morphology, and anatomical attachment of the hamartoma, the relationship to adjacent hypothalamic and neurovascular structures, the clinical phenotype, and the expertise and technological resources available at the treating center [17-20].

Potential complications of lesion-directed treatment include weight gain, endocrine dysfunction, memory impairment, oculomotor deficits, and other neurological complications, depending on the therapeutic approach and the anatomical relationship of the hamartoma to adjacent hypothalamic structures [10]. The patient underwent neurosurgical evaluation, and lesion-directed intervention was deferred with a recommendation for clinical follow-up and annual brain MRI. Because the detailed rationale was not documented in the available medical record, the relative influence of lesion anatomy, lesion size, short-term seizure response, or local availability of minimally invasive treatment options could not be determined. Furthermore, long-term follow-up was unavailable; therefore, sustained seizure control and subsequent neurological, developmental, behavioral, and endocrine outcomes could not be reliably assessed.

Conclusions

HH are rare congenital, non-neoplastic malformations that should be considered in infants and young children with recurrent, stereotyped, context-inappropriate episodes of laughter or crying. In this case, gelastic and dacrystic seizures began at three months of age and were followed by additional focal seizure types, contributing to delayed recognition of the underlying HH. When this characteristic seizure semiology is identified, early neurological evaluation and high-resolution brain MRI are important, even before cognitive, behavioral, sleep, or endocrine manifestations become apparent. Management decisions require individualized multidisciplinary assessment of seizure burden, lesion anatomy, developmental and endocrine status, and response to antiseizure therapy. Early recognition may facilitate timely epilepsy management and longitudinal monitoring for neurological, developmental, behavioral, and endocrine complications.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Supervision: Silvia N. Suarez Mantilla, Yuli E. Rojas, Leidy Forero, Sergio F. Gárces, Laura S. Carvajal, Santiago Arrieta, Yully A. Rangel

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. Universidad Industrial de Santander issued approval 03022026. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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