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Delayed Massive Cystic Transformation Following Germinal Matrix Hemorrhage Presenting as Hydrocephalus in a Preterm Infant: A Case Report

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ABSTRACT

Germinal matrix hemorrhage/intraventricular hemorrhage (GMH-IVH) is a significant neurological complication of prematurity, particularly in infants born before 32 weeks of gestation. Severe GMH-IVH may lead to long-term sequelae including post-hemorrhagic ventricular dilatation, white matter injury, cystic degeneration, and neurodevelopmental impairment. Delayed large cystic transformation with progressive hydrocephalus remains an uncommon and underrecognized complication.

We report the case of a 10-month-old premature female infant born at 32 weeks of gestation with a birth weight of approximately 1700 g from a twin pregnancy. Neonatal cranial ultrasonography demonstrated right-sided Grade III germinal matrix/intraventricular hemorrhage. After an initial period of relatively acceptable growth and neurodevelopment, the patient progressively developed macrocephaly, developmental delay, poor feeding, and lethargy between 6 and 10 months of age. Brain magnetic resonance imaging (MRI) revealed a large right parieto-occipital intra-axial cystic cavitary lesion with cerebrospinal fluid signal intensity, peripheral hemosiderin staining on susceptibility-weighted imaging, marked thinning of the adjacent cortical mantle, ipsilateral white matter loss, and severe supratentorial hydrocephalus. Imaging findings were most consistent with delayed post-hemorrhagic cystic transformation, likely representing porencephalic evolution secondary to prior periventricular hemorrhagic injury. The patient subsequently underwent ventriculoperitoneal shunt placement with early postoperative reduction in ventricular size. However, during follow-up, the patient developed a severe systemic infection complicated by septic shock. No definite evidence of shunt malfunction, recurrent intracranial hemorrhage, or progression of the intracranial lesion was identified. Despite intensive medical management, the patient died approximately two months after surgery.

This case highlights a rare delayed complication of severe neonatal GMH-IVH with progressive cystic cavitary transformation and hydrocephalus presenting months after the initial hemorrhagic insult. The report emphasizes the importance of long-term neuroimaging surveillance and careful neurodevelopmental follow-up in premature infants with severe GMH-IVH, particularly those at risk for periventricular hemorrhagic injury and post-hemorrhagic ventricular dilatation.

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Introduction

Germinal matrix hemorrhage is a well-established complication of prematurity, particularly in neonates born before 32–34 weeks of gestation. The germinal matrix is a highly vascular, metabolically active structure located in the subependymal region, characterized by fragile capillary networks lacking adequate structural support and autoregulatory capacity [1–3]. The classification system proposed by Papile et al. categorizes GMH into four grades, reflecting increasing severity and extent of hemorrhage [2]. Higher-grade hemorrhages are associated with a greater risk of long-term neurological impairment and structural brain injury [3]. The natural evolution of GMH is variable. While many low-grade hemorrhages resolve without sequelae, more severe cases may lead to post-hemorrhagic ventricular dilatation, white matter injury, and, less commonly, cystic degeneration [4]. In rare instances, progressive liquefaction of injured brain tissue results in the formation of intraparenchymal cystic cavities, known as porencephalic cysts [5]. Although early complications of GMH are well documented, delayed cystic transformation presenting months after the initial insult remains insufficiently described, particularly when associated with significant mass effect and hydrocephalus.

Case Presentation

A 10-month-old female infant was referred to our center because of progressive head enlargement, poor feeding, lethargy, and developmental delay. The patient was born prematurely at 32 weeks of gestation as part of a dichorionic twin pregnancy via cesarean section due to preterm labor. Birth weight was approximately 1700 g. Apgar scores were 6 and 8 at 1 and 5 minutes, respectively. Following delivery, the patient was admitted to the neonatal intensive care unit (NICU) for approximately one month because of complications related to prematurity, including respiratory distress syndrome requiring noninvasive ventilatory support and supplemental oxygen therapy. No neonatal seizures were documented during hospitalization. Initial cranial ultrasonography performed during the first week of life demonstrated right-sided germinal matrix hemorrhage with intraventricular extension, compatible with Grade III intraventricular hemorrhage.

After discharge from the NICU, early developmental milestones were reportedly comparable to those of the co-twin sibling. Head circumference measurements initially remained within expected corrected-age percentiles. Between 6 and 10 months of age, however, the parents gradually noted delayed neurodevelopmental progression compared with the co-twin, including reduced social interaction, decreased environmental responsiveness, delayed motor milestone acquisition, and diminished activity level. Progressive macrocephaly also became clinically apparent during this interval. No definite seizure activity was reported by the parents. Neurological examination at admission demonstrated reduced spontaneous activity and mild generalized hypotonia without clear focal hemiparesis or overt spasticity. Ophthalmologic examination did not reveal definite papilledema, although clinical suspicion for increased intracranial pressure was raised because of progressive head enlargement and poor feeding.

Two to three days before hospitalization, the infant developed poor oral intake, lethargy, irritability, and decreased feeding tolerance. The patient was initially admitted to the infectious disease service with a presumptive diagnosis of sepsis. Laboratory evaluation revealed elevated inflammatory markers, and empirical intravenous antibiotic therapy was initiated. Because of progressive macrocephaly and concerning neurological findings, brain magnetic resonance imaging (MRI) was subsequently performed. MRI demonstrated a large right parieto-occipital intra-axial cystic cavitary lesion with cerebrospinal fluid signal characteristics on all sequences, including T1 hypointensity, T2 hyperintensity, and suppression on FLAIR imaging. No restricted diffusion was identified on diffusion-weighted imaging (DWI), and corresponding apparent diffusion coefficient (ADC) maps demonstrated facilitated diffusion. No enhancing solid component was observed following gadolinium administration. Susceptibility-weighted imaging (SWI) demonstrated peripheral hemosiderin-related blooming, compatible with prior hemorrhagic injury (Figures 1–3). The lesion was associated with marked thinning of the adjacent cortical mantle, surrounding white matter volume loss, distortion of the right lateral ventricle, and severe supratentorial ventricular dilatation consistent with advanced hydrocephalus. Imaging findings were considered most consistent with delayed post-hemorrhagic cystic transformation related to prior germinal matrix/intraventricular hemorrhage, likely representing porencephalic evolution associated with periventricular hemorrhagic injury (Figures 4–6). The patient subsequently underwent ventriculoperitoneal shunt placement (Figure 7) because of progressive hydrocephalus and mass effect. Early postoperative imaging demonstrated partial reduction in ventricular caliber and mild

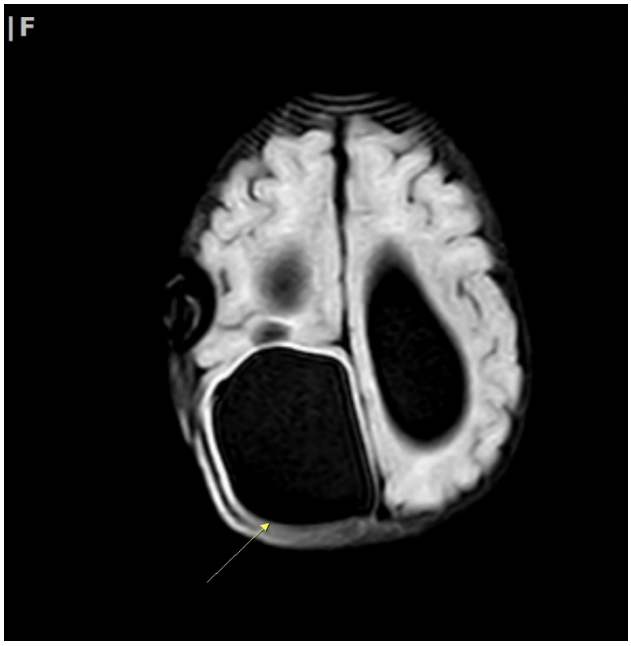

Fig. 1. Axial FLAIR MRI shows a large low FLAIR signal intensity lesion developed adjacent to occipital horn of right lateral ventricle. The lesion demonstrates CSF-like signal intensity, appearing hypointense on FLAIR and hyperintense on T2-weighted imaging.

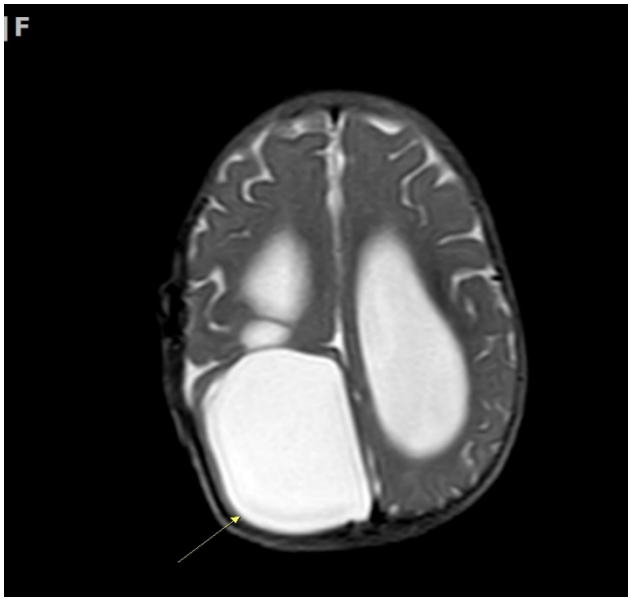

Fig. 2. Axial T2 image reveals that there is a round lesion with CSF signal intensity located adjacent to occipital horn of right lateral ventricle without any solid part or any septation.

improvement in mass effect. Initial postoperative clinical assessment also demonstrated mild improvement in feeding tolerance and alertness. However, during follow-up, the patient developed a severe systemic infection complicated by septic shock. No definite evidence of shunt malfunction, recurrent intracranial hemorrhage, or progression of the intracranial lesion was identified. Despite intensive medical management, the patient died approximately two months after surgery.

Discussion

This case illustrates a rare and delayed complication of germinal matrix hemorrhage, characterized by extensive cystic transformation and late-onset hydrocephalus. Germinal matrix–intraventricular hemorrhage (GM-IVH) remains one of the most important neurological complications of prematurity, particularly among infants born before 32 weeks of gestation and those with very low birth weight

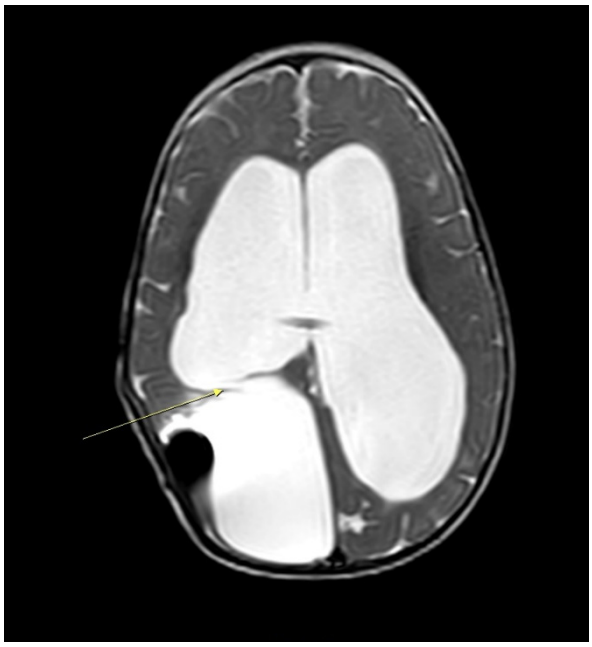

Fig. 3. Axial T2 image reveals that the cystic structure located posterior to occipital horn of right lateral ventricle has a connection to the right lateral ventricle (yellow arrow). This connection between mentioned cyst and the lateral ventricle favors a porencephalic cyst rather than other differential diagnoses such as arachnoid cyst or simple cystic encephalomalacia.

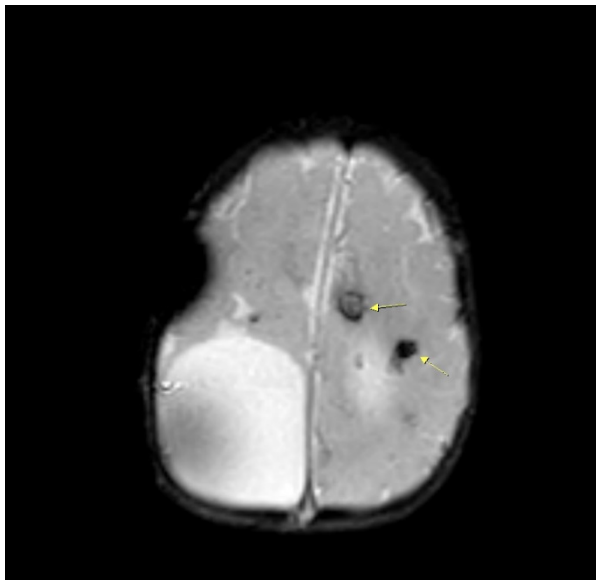

Fig. 4. Evidence of several foci of blooming in GRE sequences(arrows) representative of old intraparenchymal hemorrhage leading to hemosiderin deposition.

[11,12]. The germinal matrix is a highly vascularized and metabolically active region located in the subependymal zone adjacent to the lateral ventricles. Due to the structural immaturity of its capillary network, poor vascular support, and impaired autoregulation of cerebral blood flow in premature neonates, this region is highly susceptible to hemorrhage during periods of hemodynamic instability [11,12]. The susceptibility of the germinal matrix to hemorrhage is well established and is primarily related to its immature vascular architecture and limited autoregulatory capacity [10]. Following hemorrhage, a cascade of pathological processes—including hemoglobin degradation, inflammatory activation, and oxidative stress—leads to secondary neuronal and glial injury. Volpe and others have emphasized that this process may result in progressive tissue necrosis and subsequent liquefaction, ultimately

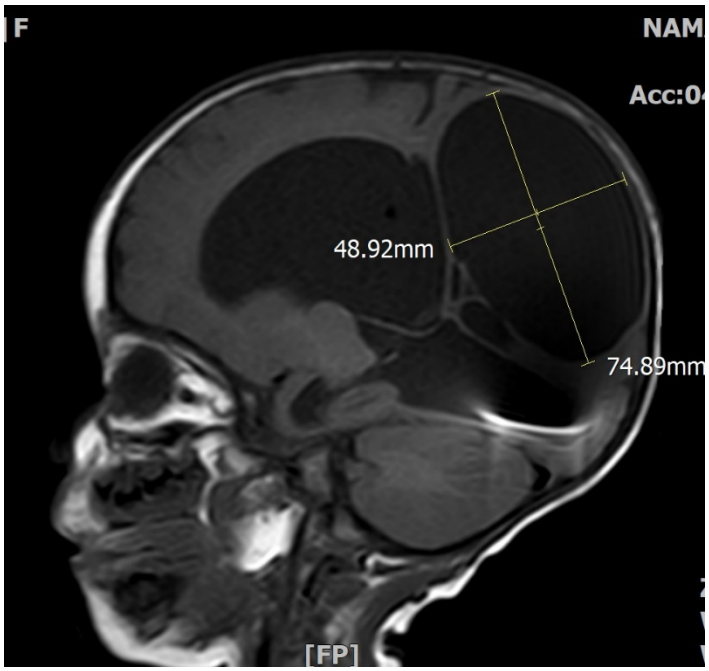

Fig. 5. Sagittal T1 image shows a 74 × 48 mm cystic structure adjacent to the occipital horn of right lateral ventricle connected to the right lateral ventricle associated with hydrocephalus.

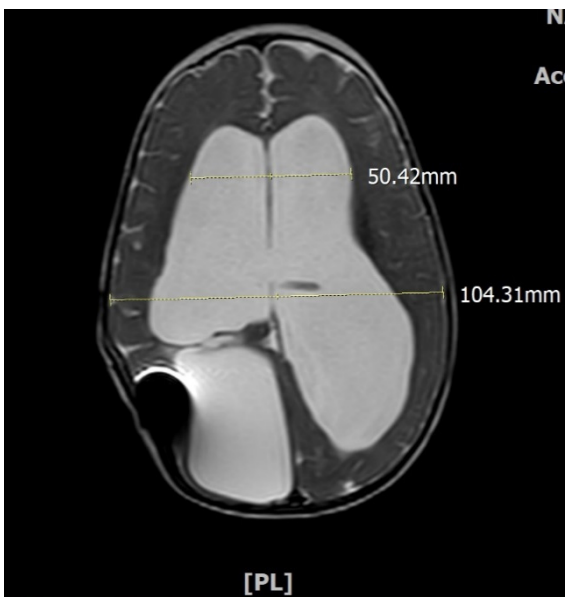

Fig. 6. Axial T2 image shows significant ventriculomegaly, the Evans index measured 0.5, indicating marked ventriculomegaly in conjunction with clinical and imaging findings of hydrocephalus.

forming cystic cavities within the brain parenchyma [3,4]. These cavities, when filled with cerebrospinal fluid and communicating with the ventricular system, are referred to as porencephalic cysts [5]. Also, the pathogenesis of severe GM-IVH is now understood to extend beyond simple intraventricular bleeding. Contemporary literature emphasizes the major role of venous congestion and periventricular hemorrhagic infarction (PVHI/PHI) in the development of subsequent parenchymal injury and cystic degeneration. Parodi et al. described that obstruction of the deep medullary and terminal veins by germinal matrix hemorrhage may result in venous ischemia, secondary hemorrhagic infarction, white matter destruction, and eventual cavitary transformation [12]. These lesions may progressively evolve into unilateral persistent cystic cavities communicating with the ventricular system, representing porencephalic evolution following hemorrhagic injury rather than a primary congenital malformation [12].

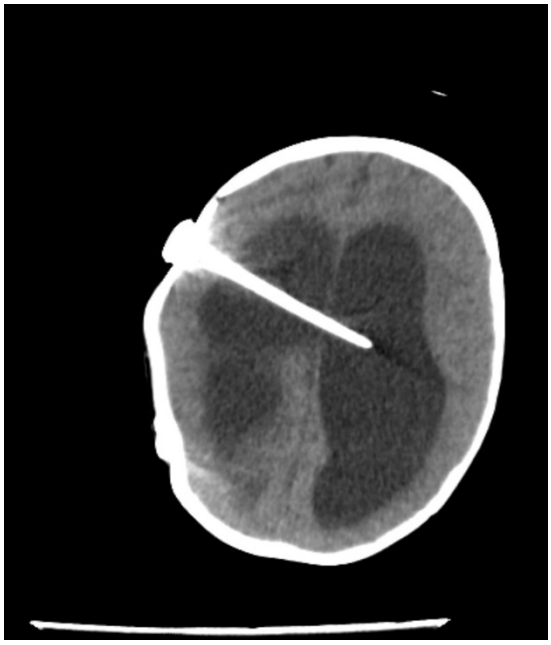

Fig. 7. Early postoperative image following ventriculoperitoneal shunt placement demonstrating the catheter tip within the left lateral ventricle.

Large post-hemorrhagic cavitary lesions are therefore increasingly considered part of a spectrum of periventricular hemorrhagic injury rather than isolated entities [11,12]. The evolution from acute hemorrhage to delayed cyst formation may occur over weeks to months as necrotic white matter undergoes liquefactive degeneration and progressive encephalomalacic change [11]. In many patients, the resulting cavity remains connected to the lateral ventricle and is associated with adjacent white matter loss and cortical mantle thinning, imaging findings that support porencephalic transformation. However, because the boundaries between porencephalic cyst, post-hemorrhagic cavitation, and cystic encephalomalacia may overlap radiologically, several authors recommend cautious terminology and clinicoradiologic correlation [12]. Post-hemorrhagic ventricular dilatation (PHVD) represents another important complication of severe GM-IVH. Approximately one-quarter of infants with severe IVH develop progressive ventricular enlargement, particularly following grade III IVH or PVHI. The mechanism is multifactorial and includes impaired cerebrospinal fluid (CSF) resorption due to inflammatory fibrosis of arachnoid granulations, obstruction of CSF pathways by intraventricular blood products or fibrin debris, and distortion of ventricular anatomy by adjacent parenchymal injury [12,13]. One of the most notable features of this case was the delayed clinical presentation at 10 months of age. Most complications of GMH, particularly post-hemorrhagic ventricular dilatation, occur within the early neonatal period [9,10]. In the present case, the large unilateral cystic cavity likely contributed to hydrocephalus through combined mechanisms of ventricular distortion, altered CSF dynamics, and post-hemorrhagic ventricular obstruction. The delayed progression of macrocephaly and neurodevelopmental decline several months after the initial neonatal hemorrhage highlights the importance of long-term neuroimaging surveillance in premature infants with severe GM-IVH, even in those who initially appear clinically stable.

Hydrocephalus following GMH is typically attributed to impaired cerebrospinal fluid absorption due to obstruction of arachnoid granulations by blood products [10]. However, in this case, the presence of a large cyst introduces an additional mechanism.

The cyst likely contributes to hydrocephalus through:

- Direct compression of cerebrospinal fluid pathways
- Distortion of ventricular anatomy
- Secondary impairment of CSF circulation

This represents a less common, mass-effect–related hydrocephalus, distinguishing it from classic post-hemorrhagic hydrocephalus. The differential diagnosis of a large intracranial cystic lesion in a preterm infant with a prior history of

germinal matrix/intraventricular hemorrhage includes porencephalic cyst formation, post-hemorrhagic cystic cavitation, cystic encephalomalacia, arachnoid cyst, and severe ex vacuo ventricular enlargement. Arachnoid cysts are typically extra-axial lesions that displace adjacent brain parenchyma without causing significant parenchymal destruction or white matter loss [6,7]. In the present case, the lesion appeared intra-axial with marked thinning of the surrounding cortical mantle and adjacent white matter loss, making an arachnoid cyst less likely.

Cystic encephalomalacia and post-hemorrhagic cavitary degeneration were also considered. These entities may demonstrate CSF-like signal intensity and develop following ischemic or hemorrhagic injury; however, they are more commonly associated with volume loss and ex vacuo dilatation rather than a large expansile cystic cavity with substantial mass effect and progressive hydrocephalus [8,12]. Furthermore, the persistent unilateral cavitary lesion with probable communication to the ventricular system and surrounding chronic hemorrhagic changes favored a post-hemorrhagic porencephalic evolution. The distinction between porencephalic cyst, post-hemorrhagic cavitation, and severe cystic encephalomalacia may be challenging because these processes likely represent overlapping components of the same spectrum of periventricular hemorrhagic injury in premature infants [12]. Therefore, in the current case, the imaging findings should be interpreted cautiously within the clinical context rather than regarded as pathognomonic for a single entity.

The history of severe prematurity, documented neonatal grade III GM-IVH, peripheral hemosiderin deposition on susceptibility-weighted imaging, adjacent white matter loss, cortical thinning, and ventricular communication collectively support delayed post-hemorrhagic cystic transformation, likely representing porencephalic evolution secondary to periventricular hemorrhagic injury. This case remains notable because of the delayed presentation several months after the initial hemorrhagic insult, the development of a large unilateral cystic cavity with progressive hydrocephalus, and the significant diagnostic overlap with other cystic intracranial lesions. These findings underscore the importance of long-term neuroimaging follow-up in premature infants with severe GM-IVH and suggest that delayed structural sequelae of periventricular hemorrhagic injury may be underrecognized.

Conclusion

Delayed cystic transformation following germinal matrix hemorrhage is a rare but clinically important entity that may present later in infancy with progressive neurological signs and hydrocephalus. This case emphasizes that post-hemorrhagic brain injury is not always a static process and may evolve over time into significant cystic lesions with mass effect. Awareness of this delayed spectrum is essential for radiologists and clinicians to ensure accurate diagnosis, appropriate differentiation from other intracranial cystic lesions, and timely neurosurgical management when indicated.

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This case report was obtained from a patient at Namazi Hospital.

Ethical Considerations

Ethical statement

We confirm that this work was conducted in accordance with the Declaration of Helsinki and that written informed parental consent for publication was obtained.

Data availability statement

Data are available from the corresponding author upon reasonable request via email.

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No funding was received to assist with the preparation of this manuscript.

Conflict of Interests

The authors have no conflict of interest to declare.

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