International Journal of Contemporary Pediatrics
Encarnacion-Santos DA et al. Int J Contemp Pediatr. 2026 May;13(5):765-769
http://www.ijpediatrics.com
pISSN 2349-3283 | eISSN 2349-3291

DOI: https://dx.doi.org/10.18203/2349-3291.ijcp20261143

Case Report

A preterm infant with intraventricular hemorrhage (IVH stage 2), central nervous system injury and hydrocephalus: case report and literature review

Daniel A. Encarnacion-Santos[1]*, Ghezal Dabiry[2], Gennady Chmutin[1,3], Sultan Mujib Dabiry[4], Egor Chmutin[1], Kamolov Mehriddin[1], Emmanuel Batista-Geraldino[5]

[1]Department of Neurosurgery of People of Friendship University, Moscow, Russia
[2]Faculty of Medicine, Ankara Yıldırım Beyazıt University, Ankara, Türkiye
[3]Moscow State Budgetary Healthcare Institution, Morozovskaya Children's City Clinical Hospital of the Moscow City Healthcare Department, Moscow, Russia
[4]Department of Emergency Medicine, Medical Park Ankara Hospital, Ankara, Türkiye
[5]Deparment of Neurosurgery, University teaching Hospital, Juan Pablo Pina, San Cristóbal, Dominican Republic

Received: 10 March 2026
Accepted: 09 April 2026

***Correspondence:**
Dr. Daniel A. Encarnacion-Santos,
E-mail: danielencarnacion2280@gmail.com

ABSTRACT

Intraventricular hemorrhage (IVH) is a type of bleeding in newborns that occurs after germinal matrix hemorrhage. The aim of this report is to determine the causes and pathologies associated with HIV and the postoperative outcomes with conventional or surgical management, based on bleeding findings through neurographic diagnostic means and care in the intensive care unit (ICU) during the development of the disease. A spontaneously born premature infant at 27 weeks gestation, delivered in cephalic presentation, was admitted to the emergency department with ruptured membranes and two umbilical cord entanglements. There was no spontaneous breathing. Resuscitation was performed in the delivery room with mask ventilation for 1 minute and intubation for 2 minutes. Surfactant replacement therapy was administered with positive results. The Apgar score was 3/4/5, and weight was 1190 g. The infant was transferred to the NICU for further care and monitoring. A premature infant was transferred to the neonatal unit at 1 month and 14 days of age (33 weeks gestation) in moderate condition due to respiratory distress; oxygen dependence due to bronchopulmonary dysplasia (BPD); neurological symptoms, including central nervous system depression syndrome due to hypoxic-hemorrhagic CNS damage; and ventriculomegaly.

Keywords: Intraventricular hemorrhage, Preterm infant, Hydrocephalus, CNS injury (HIV)

INTRODUCTION

Intraventricular hemorrhage (IVH) is a type of bleeding in newborns that occurs after germinal matrix hemorrhage. It can cause morbidity or mortality in very low birth weight infants (at least <1500 g). With a 22% rate of prematurity, these infants are at high risk of germinal matrix injury or hemorrhage, or high-grade intraventricular hemorrhage. Between 25% and 30% of these infants tend to develop post-hemorrhagic hydrocephalus. Untreated hydrocephalus can cause white matter damage, including seizures and cognitive impairment, which can lead to death.[1] IVH is considered multifactorial, requiring interventions at different time points, such as during the prepartum, intrapartum, and postpartum periods, with the aim of reducing intraventricular hemorrhage. Most IVHs occur within the first three days of life, making it crucial to evaluate the effectiveness of interventions as they arise. The incidence of IVH in some studies was 34%, with severe cases reaching 15% in infants with a gestational age

of less than 28 weeks.[2] Cranial ultrasounds is considered the primary diagnostic tool for detecting HIV. Following certain screening tests, especially cranial ultrasound, can help pinpoint the onset of bleeding. Using other tests, however, can delay the detection of this onset, which may lead to complications such as increased risk of neurological damage or delayed treatment interventions.[3] Factors that tend to cause HIV include prenatal, perinatal, and postnatal events. HIV is considered acute, although, because it is asymptomatic, the onset of the event is unknown in most cases. The first cranial ultrasound should be performed on the third postpartum day. HIV is believed to occur within the first 12 hours before or during labor; therefore, when it occurs after 12 hours, it may be related to postnatal risks.[4] Infections tend to be more frequent in shunts following intraventricular hemorrhage in premature infants compared to other types of hydrocephalus. At an early age, shunt placement is considered a risk factor. Although this theory is relatively limited, the infection rate can reach 18%, taking into account modifiable risk factors in the high-risk premature infant.[5]

CASE REPORT

A premature infant was admitted to the Moscow City Clinical Children's Hospital from the Moscow Regional Clinical Hospital, Morozovskaya Children's Hospital, at 27 weeks' gestation, with a diaphragmatic oxygen cannula due to the severity of his condition, which included moderate respiratory distress, neurological disorders, profound morphofunctional immaturity, and hemorrhagic damage to the central nervous system. On admission, the complete blood count (CBC) was normal, and inflammatory markers were negative. X-rays showed signs of bronchopulmonary dysplasia (BPD), with areas of infiltrative changes in the right supradiaphragmatic regions. The mother was 38 years old and had autoimmune thyroiditis and pulmonary sarcoidosis in remission. Her medical history included a hematoma in the first trimester; COVID-19 and cervical insufficiency in the second trimester (with surgical correction—sutures); and threatened miscarriage throughout the third trimester. The first spontaneous labor was premature, with a cephalic presentation at 27 weeks, requiring emergency hospitalization due to rupture of membranes mixed with blood and two umbilical cord entanglements. Spontaneous breathing was absent. Resuscitation was provided in the delivery room, with mask ventilation for 1 minute and intubation for 2 minutes. Surfactant replacement therapy was administered with a positive effect. The Apgar score was 3/4/5, weight was 1190 g, length was 33 cm, head circumference was 27 cm, and chest circumference was 26 cm. At 30 minutes, the baby was transferred to the intensive care unit in critical condition and placed on a ventilator. On the first day of life, the baby was switched to bilevel positive airway pressure (BiPAP); on the fifth day, to diffuse oxygenation; and on the eighth day, to CPAP. On the seventeenth day of life, oxygenation was administered via nasal cannula. Antibacterial therapy was administered with rotation on the fourth heartbeat. On the ninth day, hyperglycemia developed, and glucose levels were normalized with Actropid. On the thirteenth day, signs of NEC were detected, and GVL was established. Metronidazole therapy was initiated with an enteral pause, and the condition normalized. On the seventh day, neurosonography was performed: ventriculomegaly in the context of grade 2 HIV. A lumbar puncture was performed on the 9th day (xanthochromic cerebrospinal fluid); after 7 days, the cerebrospinal fluid was yellow. Blood transfusion was administered 6 days after the ventricular punctures; ventriculomegaly and parenchymal atrophy persisted. The following therapy was administered: therapeutic and protective regimen (incubator + servocontrol), and respiratory support with 24-hour mechanical ventilation; (VRAP), 5 days of O_2 in the incubator diffusely two days later, continuous positive airway pressure (CPAP), from the 4th day later; O_2 was administered in the incubator (12 hours); CPAP: O_2 was repeated via nasal cannula for nine days from 24 hours until the present, infusion therapy as needed, taking into account the second day of last month until the present; antibacterial therapy; and to prevent fungal infections, Fluconazole was used for 11 days and Fluconazole intravenously (Figures 1 and 2).

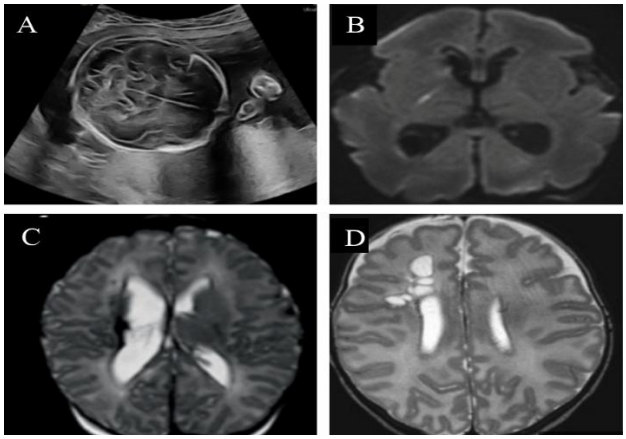

Figure 1: (A) Neurosonography with irregular contours and dilation of the ventricles, probable hemorrhage, and hydrocephalus; (B) brain MRI of a premature infant; (C) HIV, diffusion restriction in the midportion of the posterior limb of the internal capsule (PLIC); and (D) MRI of a premature infant shows hemorrhage with a multicystic pattern on the T2-weighted sequence.

Diagnosis

Diagnosis included - primary diagnosis: prematurity of 27 weeks, VLBW; hypoxic-hemorrhagic CNS injury (HIV stage 2, spontaneous SAH), depressive syndrome; ventriculomegaly replacement; morphofunctional immaturity; urinary tract infection; mild bronchopulmonary dysplasia; retinopathy of prematurity stage II, active phase; and anemia of prematurity (blood transfusions).

Neurosonography

First day, ultrasound results indicate physical immaturity of brain structures and dilated lateral ventricles as a result of intraventricular hemorrhage (stage 2 IVH). The brain's structures are positioned appropriately.

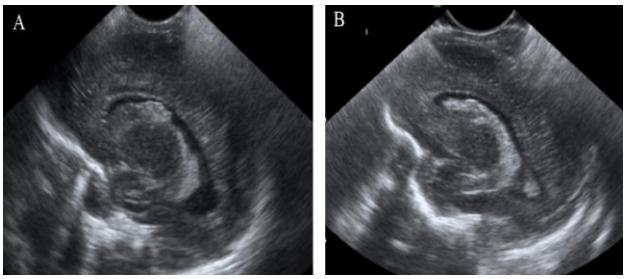

Figure 2: (A) Echographic signs of dilated lateral ventricles associated with intraventricular hemorrhages, and (B) positive dynamics and morphological immaturity of brain structures.

Two days later, the color filling of the visible parts of the cerebral sinuses (sagittal sinus) was entirely evident during a color Doppler test in the intensive care unit. Intraluminal lesions were not found.

Three days later, there were echographic indications of lateral ventricle dilatation linked to intraventricular hemorrhages with positive dynamics and immature brain morphology. A single small periventricular cystic inclusion in the left temporoparietal projection, a collection of echographically heterogeneous content in the projection of the meningeal spaces of the left cerebellar hemisphere, a marked morphofunctional immaturity of the brain tissue, and moderately dilated lateral ventricles due to intraventricular hemorrhages.

Seven days later, there were echographic indications of a single small periventricular cystic inclusion in the projection of the left temporoparietal region, a collection of sonographically heterogeneous contents in the projection of the meningeal spaces of the left cerebellar hemisphere, a marked morphofunctional immaturity of the brain tissue, and moderately pronounced dilation of the lateral ventricles (without a significant increase in size compared to previous ultrasound examination data).

34 days later, an ultrasound revealed a small cystic inclusion in the projection of the caudothalamic notch on the right, a small anechoic inclusion in the projection of the temporal region on the left, and a slight dilation of the lateral ventricles (no increase in size compared to the ultrasound).

Electroencephalography

The recording was obtained with a background of numerous artifacts. Cerebral bioelectrical activity is disorganized and characterized by the suppression of cortical rhythms in the posterior regions of the brain. In addition, poorly synchronized slow-wave activity was recorded in the theta-delta range, including a sharp-wave component, followed by the suppression of cortical rhythms: the "alternating newborn curve." Rhythmic activity was periodically observed in the frontocentral brain regions at frequencies up to 5 Hz, likely forming sleep spindles.

MRI

The MRI shows post-ischemic leukopathy, mixed replacement hydrocephalus, sequelae of intraventricular hemorrhage and subacute hemispheric hemorrhage, immaturity of the cerebral cortex, and venous angiomas in the cerebellar cortex.

Two days later, MRI showed post-ischemic leukopathy, mixed replacement hydrocephalus, sequelae of intraventricular hemorrhage and subacute hepatitis C, immaturity of the cerebral cortex and venous angiomas in the cerebellar cortex.

DISCUSSION

Intraventricular hemorrhage is associated with germinal matrix hemorrhage (LVH-GMH), similar to a periventricular hemorrhagic infarction, and its main complication is post-hemorrhagic ventricular dilation. Neonatal morbidities often occur in premature infants and can lead to adverse cognitive outcomes. These hemorrhagic types are described in cranial ultrasound, along with the underlying anatomical structures and their mechanisms.[6]

Epidemiology

Currently, there have been significant advances in the treatment of premature infants with intraventricular hemorrhage and parenchymal infarction with germinal matrix injury. The incidence in this population ranges from 20 to 25%. In very low birth weight infants, it increases with decreasing gestational age and occurs in premature newborns born at 24 weeks' gestation. The risk of grade III IVH and parenchymal infarction is estimated at 10–25% in surviving newborns born at 28 weeks' gestation, with severe injuries diagnosed in <5% of cases. Late IVH hemorrhage presenting at 32 weeks' gestation can cause venous thrombosis in newborns or premature infants.[7]

Pathology

The pathogenesis of IVH and parenchymal hemorrhage, with gestational age as a risk factor, is multifunctional and complex. The germinal matrix can reach its maximum volume within 25 weeks of gestational age; it tends to regress, remaining a persistent mass until 36 weeks of gestation. The microvascularity of the germinal matrix tends to be fragile and intrinsic, as demonstrated by some postmortem venous studies. This is attributed to the

immaturity of the vessel walls and their fluctuations in cerebral blood flow, which lack autoregulation and appear to reflect important factors.[8] Fluctuations in venous pressure, along with anatomical variations in venous systems and genetic factors, with complex integration, tend to be associated with IVH and parenchymal hemorrhage; perinatal hypoxic ischemia; inflammation; and cardiovascular instability, such as in severe respiratory illness, pneumothorax, and the use of inotropic drugs. The risk of intrahepatic and parenchymal hemorrhage has been shown to decrease with the use of prenatal glucocorticoids, and postnatal indomethacin reduces severe intrahepatic hemorrhage, especially in male newborns, but without neurological improvement. Data on the preventive use of umbilical cord clamping are contradictory.[9]

Intraventricular hemorrhage in preterm infants

Intraventricular hemorrhage of the germinal matrix in preterm newborns is known as the most common intracranial hemorrhage, while other types of hemorrhage are less frequent. The neonatal intensive care unit has been remodeled over time, thanks to current technological advances, which have increased survival rates but also increased morbidity in preterm newborns with intraventricular hemorrhage. IVH can cause ruptures of vessels in the germinal matrix, which is highly vascularized in regions such as the periventricular subependymal area and is the source of neuronal and glial cells in the immature brain, which migrate during fetal brain development.[10] Oligodendrocytes are glial precursors, which in turn give rise to astrocytes of the white matter and GABAergic neurons of the thalamus and cerebral cortex. The germinal matrix surrounds the entire ventricular system and begins to involute around 28 weeks of gestation, disappearing completely at term. This involution occurs after 32 weeks of gestation and carries a risk of hemorrhage. In a premature brain (<32 weeks of gestation), the white matter is occupied by premature oligodendrocytes and their precursor cells, which are more sensitive to light, excitotoxicity, and oxidative stress than mature oligodendrocytes. Glial precursors tend to migrate to the cerebral cortex in low-birth-weight infants.[11]

IV-associated brain injury

Skeletal muscle possesses a system of blood vessels that, when ruptured, release blood that deposits red blood cells into the intraventricular space. These red blood cells then lyse, releasing hemoglobin into the cerebrospinal fluid (CSF) and periventricular white matter. Once hemoglobin levels are high and reactive, it spontaneously auto-oxidizes from oxyhemoglobin to methemoglobin and then to superoxide. Hemosiderin is converted into hemosiderin, released from the CSF, and deposited in the brainstem and cerebellar surface, potentially damaging these anatomical structures and altering the cerebellar cortex. The degradation of heme produces bilirubin, carbon monoxide, and free radicals.[12] Free iron can release reactive oxygen species and damage lipids, proteins, and DNA. It also has the capacity to invade cell membranes with cytolytic effects that can lead to periventricular cell death. IVH: This entire process can damage cell progenitors and white matter through oxidative stress and pressure, which may contribute to the pathogenesis of periventricular leukomalacia. Therefore, there may be negative effects due to small hemorrhages, which can affect the migration of neuronal and glial cells in the brains of premature infants. Cognitive complications can arise after the development of hydrocephalus.[13]

CONCLUSION

A premature infant was transferred to the neonatal unit at 1 month and 14 days of age (33 weeks' gestation) in moderate condition due to respiratory distress; oxygen dependence due to bronchopulmonary dysplasia (BPD); neurological symptoms, including central nervous system depression syndrome due to hypoxic-hemorrhagic CNS damage; and ventriculomegaly. Due to brief episodes of desaturation and a decrease in heart rate, caffeine was reintroduced at a dose of 10 mg/kg/day, and oxygen therapy was continued via nasal cannula at a rate of 1 l/min. Oxygen dependence decreased dynamically, oxygenation stabilized, and the infant was switched to diffused oxygen (34 weeks' gestation). Given the absence of apneic episodes, respiratory stimulation treatment was completed after 7 days (35 weeks' gestation). Oxygen weaning is being performed dynamically, with monitoring of oxygen saturation. A consultation was held with the pulmonary department. Since 1 month and 10 days of age (37 weeks' gestational age), the infant has been oxygen-independent. During the first few days of admission, a loss of tolerance to enteral feeding was observed, manifested by abdominal distension with significant gas. An antifoaming agent was added to the feeding regimen, fecal stimulation was performed, and enteral feeding is now being absorbed more effectively. The infant is receiving breast milk, and the dose of fortifying formula has been gradually increased. Independent sucking began after two days (35 weeks' gestational age) and has been independently sucking the entire volume of feed since 8 days (36 weeks' gestational age). Newborn screening revealed elevated 17-OPG levels of 78.3 nmol/l, which subsequently decreased to 62 nmol/l. The child was followed by an endocrinologist. ACTH and cortisol levels were within reference limits. At discharge at 2.5 months of age (postnatal growth restriction at 37 weeks), the child's condition is satisfactory. He is oxygen-independent. There are no signs of infectious toxemia. Blood and urine tests show no inflammatory changes, and the anemia is compensated. The child tolerates enteral nutrition at a rate of 55-60 ml of breast milk, plus one sachet of NAN fortifier per feeding. He is gaining weight. His physical development corresponds to the 25th percentile for weight and height and the 3rd percentile for head circumference, according to the intergrowth postnatal growth scale. The child does not require hospitalization or further evaluation and is discharged in good general condition.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was carried out according to the latest revision of the Helsinki Declaration regarding medical research involving human subjects

REFERENCES

1. Holste KG, Xia F, Ye F, Keep RF, Xi G. Mechanisms of neuroinflammation in hydrocephalus after intraventricular hemorrhage: a review. Fluids Barriers CNS. 2022;19(1):28.
2. Stoll BJ, Hansen NI, Bell EF, Walsh MC, Carlo WA, Shankaran S, et al. Trends in Care Practices, Morbidity, and Mortality of Extremely Preterm Neonates, 1993-2012. JAMA. 2015;314(10):1039.
3. Balakrishnan N, Colton T, Everitt B, Piegorsch W, Ruggeri F, Teugels JL, editors. Wiley StatsRef: Statistics Reference Online. 1st edition. Wiley. 2014.
4. Krediet TG, Kavelaars A, Vreman HJ, Heijnen CJ, Van Bel F. Respiratory distress syndrome-associated inflammation is related to early but not late peri/intraventricular hemorrhage in preterm infants. J Pediatr. 2006;148(6):740-6.
5. Han RH, McKinnon A, CreveCoeur TS, Baksh BS, Mathur AM, Smyser CD, et al. Predictors of mortality for preterm infants with intraventricular hemorrhage: a population-based study. Childs Nerv Syst. 2018;34(11):2203-13.
6. Parodi A, Govaert P, Horsch S, Bravo MC, Ramenghi LA. Cranial ultrasound findings in preterm germinal matrix haemorrhage, sequelae and outcome. Pediatr Res. 2020;87(S1):13-24.
7. Tajdar M, Orlando C, Casini A, Herpol M, De Bisschop B, Govaert P, et al. Heterozygous FGA p.Asp473Ter (fibrinogen Nieuwegein) presenting as antepartum cerebral thrombosis. Thrombosis Res. 2018;163:185-9.
8. Ballabh P. Pathogenesis and Prevention of Intraventricular Hemorrhage. Clin Perinatol. 2014;41(1):47-67.
9. Tarnow-Mordi W, Morris J, Kirby A, Robledo K, Askie L, Brown R, et al. Delayed versus Immediate Cord Clamping in Preterm Infants. N Engl J Med. 2017;377(25):2445-55.
10. Mukerji A, Shah V, Shah PS. Periventricular/Intraventricular Hemorrhage and Neurodevelopmental Outcomes: A Meta-analysis. Pediatrics. 2015;136(6):1132-43.
11. Gilard V, Chadie A, Ferracci FX, Brasseur-Daudruy M, Proust F, Marret S, et al. Post hemorrhagic hydrocephalus and neurodevelopmental outcomes in a context of neonatal intraventricular hemorrhage: an institutional experience in 122 preterm children. BMC Pediatr. 2018;18(1):288.
12. Morita T, Morimoto M, Yamada K, Hasegawa T, Morioka S, Kidowaki S, et al. Low-grade intraventricular hemorrhage disrupts cerebellar white matter in preterm infants: evidence from diffusion tensor imaging. Neuroradiology. 2015;57(5):507-14.
13. Romantsik O, Agyemang AA, Sveinsdóttir S, Rutardóttir S, Holmqvist B, Cinthio M, et al. The heme and radical scavenger α1-microglobulin (A1M) confers early protection of the immature brain following preterm intraventricular hemorrhage. J Neuroinflammation. 2019;16(1):122.

Cite this article as: Encarnacion-Santos DA, Chmutin G, Chmutin E, Mehriddin K. A preterm infant with intraventricular hemorrhage (IVH stage 2), central nervous system injury and hydrocephalus: case report and literature review. Int J Contemp Pediatr 2026;13:765-9.