



A Rare Case of Pediatric Intraventricular Hemorrhage

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Abstract

Background: Intraventricular hemorrhage (IVH) is a subtype of intracerebral bleeding confined to the ventricular system, predominantly affecting preterm infants and the elderly. While its prevalence in term neonates is less documented, it is estimated to occur in 3.5% to 5.0% of cases. Potential etiologies for IVH in term infants include arteriovenous malformations (AVM), bleeding disorders, and trauma, particularly from instrumental vaginal deliveries. Management typically involves a multidisciplinary approach, including pediatricians, neurosurgeons, neurologists, hematologists, and neuroradiologists. External ventricular drainage (EVD) is the primary treatment method for IVH complicated by obstructive hydrocephalus.

Case Description: A 1-month and 19-day-old male infant presented with decreased consciousness following a seizure, accompanied by a history of vomiting and fever. After thorough examination, laboratory tests, and CT scans, he was diagnosed with IVH likely due to a vascular malformation, along with communicating hydrocephalus and suspected Dandy-Walker Syndrome. An emergency EVD was performed, and the patient showed significant improvement, leading to his discharge 11 days post-surgery.

Conclusion: This case highlights the successful management of a rare instance of IVH in a term infant through CSF drainage with EVD, resulting in improved clinical outcomes. Although vascular studies such as MRI or MRA have yet to be conducted, they are planned for future follow-up to further evaluate the underlying vascular pathology.

Keywords: Intraventricular Hemorrhage, External Ventricular Drainage, Term Neonate, Hydrocephalus, Vascular Malformation

INTRODUCTION

Pediatric intraventricular hemorrhage (IVH) is a critical condition that predominantly affects neonates and infants, often leading to significant morbidity and mortality. It is characterized by bleeding into the ventricles of the brain, which can arise from various etiologies, including traumatic brain injury, vascular malformations, and other underlying medical conditions. In children, particularly those under one year of age, isolated IVH is frequently observed, distinguishing it from adult presentations where such occurrences are less common and often linked to more complex vascular issues. ^{[1]–[3]} The prevalence of IVH in the pediatric population necessitates a thorough understanding of its causes, risk factors, and potential outcomes. Notably, brain arteriovenous malformations (AVMs) are recognized as a leading cause of spontaneous intracranial hemorrhage in children, contributing significantly to cases of IVH. ^{[4]–[6]} The clinical implications of IVH are profound, as it can lead to long-term neurological deficits and developmental challenges, underscoring the importance of early diagnosis and intervention. ^{[7], [8]}

Intraventricular haemorrhage (IVH) is defined as a subtype of intracerebral bleeding limited to the ventricular system. ^[9] This type of intracerebral haemorrhage is more common in preterm infants due to bleeding in the fragile and



immature capillaries of the germinal matrix and in the elderly due to a variety of other mechanisms.^[10] In term neonates with a normal perinatal course, IVH is uncommon and other causes should be ruled out.^[11] The prevalence of IVH in preterm neonates is almost 30%, while in term neonates is only around 3.5% to 5.0%. This gap may result from the brain's increased maturity at term and the lower prevalence of underlying risk factors for bleeding, such as coagulative diseases.^[12] This also shows that the younger the gestational age, the more susceptible it is for the infant to experience IVH.

IVH occurrence as an extension of intracerebral haemorrhage in adults is an independent indicator of a worse outcome. While in the neonatal populations, post-haemorrhagic hydrocephalus (PHH) is a common comorbidity and is an independent predictor of poor prognosis.^[13] IVH in both preterm and term neonates may bear serious consequences, including poor neurodevelopment and mortality, especially in cases of early onset IVH.^[12] IVH in extremely preterm infants has a mortality rate of over 50%, and it is accompanied with serious neurological sequelae such as cerebral palsy, developmental delay, deafness, and blindness.^[10]

Treatment options for both adult and neonatal IVH are focused on methods of CSF drainage to prevent PHH.^[10] An external ventricular drainage (EVD) remains the current primary method for treating primary IVH with obstructive hydrocephalus, for EVD may drain CSF out of the ventricular system and reducing intracranial pressure.^[9] Paediatric patients with IVH should be managed with multidisciplinary team including paediatricians, paediatric neurosurgeons, neurologists, hematologists, neuroradiologists, and others.^[14] Pharmacological therapy by lowering the PaCO₂ may be possible to sustain cerebral perfusion and reduce ICP in the case of acute bleeding. However, it is revealed that acetazolamide and furosemide, which reduce CSF production, may result in worse outcome for preterm infants.^[15]

We present a rare case of intraventricular haemorrhage in a term infant with clinical manifestations of seizure and decreased level of consciousness. The patient's head CT-scan showed evidence suggesting an intraventricular haemorrhage, communicating hydrocephalus, and Dandy-Walker Syndrome. The patient was treated with an emergency EVD Kocher D procedure and was seizure-free and discharged eleven days post-surgery.

CASE REPORT

A one month and 19 days old male baby was referred to Dr. Soetomo Hospital (26/11/2022) from Muhammadiyah Lamongan Hospital due to gradual decreased level of consciousness post seizure. The patient experienced a generalized seizure with the duration of one minute, and the seizure conceded with anticonvulsive medicine. Patient had a history of vomiting twice the day before and fever two days prior. History of trauma or shaken baby syndrome was denied.

Patient was born from a 27 years-old G2P0A1 female, with the first pregnancy terminated due to prematurity. Patient was born at 37 weeks by spontaneous normal vaginal delivery, with birthweight of 2.7 kg and head circumference of 32 cm. Maternal history of hypertension during pregnancy or consumption of herbal medicine was denied. Patient's mother consumed folic acid and iron supplementation routinely during pregnancy and antenatal ultrasonography did not show any abnormality. The baby had received vitamin K injection at birth.

Following the examination, patient displayed weak general condition, stable vital signs, and positive corneal reflex on both eyes. Motoric examination showed no signs of lateralisation. Physical examination revealed an open and convex anterior fontanelle, with frontal occipital circumference of 39 cm (between -2 and 2 SD) and no frontal bossing or venectations (Figure 1). Laboratory results (27/11/2022) showed anaemia, leucocytosis, hypoalbuminemia, and hyperkalaemia (Hb 7.4 g/dL; white blood cell 13.39x10³ cells/mm³; albumin 3.31 g/dL; kalium 5.4 mEq/L). Liver function tests showed increased SGOT (72 U/L) with normal SGPT (32 U/L), and an increased total (6.2 mg/dL) and direct bilirubin (3 mg/dL). Renal function and coagulation profile were normal.



Supporting examinations in the form of babygram (Figure 2), head CT-scan (Figure 3, left) were carried out (26/11/2022). The patient's babygram showed no signs of abnormality. While the head CT-scan showed a hyperdense lesion in bilateral lateral ventricles suggesting an intraventricular haemorrhage, dilatation of bilateral lateral ventricles, third and fourth ventricles suggesting a communicating hydrocephalus, and a hypodense lesion in the posterior fossa with hypoplasia of vermis cerebellum suggesting a Dandy-Walker syndrome.

The patient was assessed with decreased level of consciousness and seizure, intraventricular haemorrhage due to suspected vascular malformation, communicating hydrocephalus with suspected Dandy-Walker Syndrome, anaemia, hyperbilirubinemia, leucocytosis, hypoalbuminemia, and hyperkalaemia. The patient was scheduled for an emergency external ventricular drain (EVD) Kocher D (28/11/2022). Two days after the procedure, the patient's frontal occipital circumference was reduced to 37 cm with an open and flat anterior fontanelle. CT scan was performed one week after surgery with the impression of slit ventricle and bilateral infarct on temporoparietal region (Figure 3, right). EVD was removed two days later. Eleven days postoperative (9/12/2022), patient was seizure-free and discharged for routine neurosurgery outpatient visits.

DISCUSSION

Intraventricular haemorrhage (IVH) is a subtype of intracerebral bleeding limited to the ventricular system, more common in preterm infants and elderly. In term neonates with a normal perinatal course, IVH is uncommon and other causes should be ruled out. ^[11] In a French cohort study, approximately 20 to 30% of infants with gestational age below 29 weeks were diagnosed with IVH, where one-third developed post-haemorrhagic hydrocephalus (PHH) and 10 to 20% require shunt insertion. ^[1] In neonates with birthweight less than 1500 g, the incidence of IVH is similar to preterm neonates which is around 27%. ^[16] The prevalence of IVH in term neonates is rarely documented, and is thought to range between 3.5% and 5.0%. ^[12] Incidence rate of advanced IVH was 5.5 per 100.000 live term births. ^[14]

IVH in preterm infants is mostly caused by immature central nervous system and hemodynamic instability. ^[14] IVH in preterm infants originates from the rupture of germinal matrix haemorrhage through the ependyma into the lateral ventricle, thus the term germinal matrix haemorrhage (GMH)-IVH. ^[17] The veins in the germinal matrix are arranged in a way that predisposes them to blood flow turbulence, platelet aggregation, and vascular injury. ^[15] Fluctuating cerebral blood flow have a significant role in the pathogenesis of GMH-IVH. In preterm infants, sudden changes in blood pressure, ischemia, reperfusion, and venous congestion may cause injury to the germinal matrix. Cerebral vasodilatation which are frequently observed in hypoxic and hypoglycaemic preterm infants also increases the risk of IVH. ^[18] Other frequently documented risk factors for IVH in preterm infants include absence of antenatal steroid exposure, early sepsis, hypotension requiring therapeutic intervention, treatment for acidosis, and maternal infection. ^[19]

Several risk factors associated with the incidence of IVH in term neonates include maternal history of preeclampsia, urogenital tract infections, chorioamnionitis, and neonatal risks such as asphyxia, traumas, prothrombin complex deficiency, thrombocytopenia, and sinovenous thrombotic events. The source of bleeding in term neonates with IVH are thought to originate partly from the residual periventricular germinal matrix tissue, and mostly from ischemia or venous thrombosis in the choroid plexus or the thalamus. Other areas include the watershed area of the foramen of Monro near the caudate nucleus. ^{[12], [14]} Asphyxia is typically regarded as a risk factor for IVH due to an interruption of placental blood flow and decreased blood flow to the brain, impairing the cerebral autoregulation. However, it is challenging to prove that hypoxia is a significant contributor to IVH pathogenesis given that IVH itself may result in respiratory difficulty. ^[14]



Probable etiology of IVH in term infants include arteriovenous malformations (AVM), bleeding disorders, and trauma which may result from vaginal birth process, especially instrumental deliveries. AVM is reported by Flint et al. to be the most common vascular abnormality in adult population with IVH (54.5%), followed by aneurysm (39.4%) and Moyamoya disease (5.1%). This finding is quite similar in paediatric population from a study by Guo et al, where 66.7% paediatric patients with IVH also presented with AVM. ^[10] Bleeding disorders such as thrombocytopenia may be drug-induced, infectious, genetic, or immune related. Other coagulation factors deficiencies such as vitamin K-dependent coagulation factors deficiency and haemophilia may also increase risk of IVH. Children with severe congenital heart diseases are also at increased risk for IVH due to altered haemodynamic or anticoagulant administration. ^[14]

Clinical manifestations of IVH differs in preterm and term infants. In the term infants, IVH generally presents with seizures, apnea, irritability or lethargy, and vomiting with dehydration. Meanwhile in the preterm infants, signs such as flaccidity, loss of pupillary reaction, extraocular movements, coma, irritability, vomiting, shrill cry, central facial weakness, opisthotonic posturing, fever or hypothermia, hypo or hyperglycaemia, decreased lower extremity tone, neck flexor hypotonia, head lag, and brisk reflexes are more common findings of IVH. However, approximately 25% of IVH may be asymptomatic and only discovered from imaging procedures. ^[14] IVH commonly occurs within the first 3 days of life, and 20 to 40% of IVH extend during the first week of life. ^[16]

The first diagnostic method to diagnose an IVH is ultrasonography due to its portability, low cost, and lack of ionizing radiation. Although a more detailed imaging such as computed tomography (CT) might be required for USG cannot clearly visualize haemorrhage on the cerebellum and small injuries in the white and grey matter. ^[18] A grading system for IVH was established by Papile in 1978 using CT findings (Table 1). Another grading system was later developed by Volpe in 2008 using ultrasonography (Table 2). ^[18] The grades were described as follows:

Grade I and II involve mild bleeding that is often self-limiting. ^[17] Meanwhile, grade IV IVH was previously categorized as periventricular haemorrhagic infarction, and along with grade III were defined as advanced grades. ^[18] The source of bleeding for higher grade IVH is thought to emanate from the posterior tufts at the glomus in the choroid plexus. ^[14] IVH of this severity is commonly associated with the male sex, the IL-6-177CC genotype, and a complication following extremely preterm birth. ^[17] Advanced IVH has a rate of cerebral palsy of over 50%, and a special education is required in 75% patients. ^[18] Progressive ventricular dilatation resulting in obstructive hydrocephalus is also a common finding secondary to advanced grade IVH, although this is an uncommon finding in term infants. ^{[11], [17]}

Paediatric patients with IVH should be managed with multidisciplinary team including paediatricians, paediatric neurosurgeons, neurologists, hematologists, and neuroradiologists. ^[14] IVH can cause an increase of intracranial pressure secondary to the mass effect produced from the haemorrhage itself or the corresponding hydrocephalus due to obstruction of the ventricular outflow. The most common neurosurgical procedure for the monitoring and managing of ICP is the placement of external ventricular drain (EVD). ^[20] EVD remains the current primary method for treating primary IVH with obstructive hydrocephalus, for EVD may drain CSF out of the ventricular system and therefore reducing ICP. ^[9] EVD may be indicated if pharmacological ICP lowering treatment such as sedation and osmotic diuretics are incapable of reducing ICP to the preferred target. Other indications for insertion of EVD in IVH include the presence of hydrocephalus and deteriorating neurologic condition. The standard technique of EVD insertion commonly used is placement of right frontal EVD at the Kocher's point. This technique is contraindicated in the presence of right lateral ventricle hematoma and AVM in the trajectory of the EVD. ^[20]

One of the unwanted side effects of EVD placement is the formation of blood clots which occludes the catheter resulting in failure to relieve communicating hydrocephalus. ^{[10], [20]} EVD also removes tissue plasminogen activator released from the clot into the CSF and subsequently slow the rate of IVH clearance. ^[20] One of the solution to this



problem is the administration of antifibrinolytics to reduce the complications of EVD. A study by The Clot Lysis Evaluating Accelerated Resolution of Intraventricular Hemorrhage (CLEAR-IVH) trial showed that rtPA administered through an EVD increases the speed of IVH resolution, but it has a less prominent effect on blood in the posterolateral sections of the lateral ventricles.^[10] Other undesirable effects of EVD placement include mechanical complication such as dislodgement, and infective complications such as ventriculitis and meningitis. The latter has the highest incident rate (as high as 40%), difficult to treat, and significantly increase hospital costs. However, despite the possibility of infection and other recognized side effects, the benefit of an indicated placement of EVD may outweigh the risks.^[21]

Treatment choice for IVH depends on the volume of bleeding and presence of obstructive hydrocephalus. In patient with mild IVH, small hematoma, and no apparent hydrocephalus may be treated conservatively. While patients with grade III to IV IVH may consider to be treated with EVD. If the IVH is total and accompanied with intracranial hypertension, craniotomy and neuroendoscopic procedure might be recommended.^[10] Pharmacological treatment with acetazolamide and furosemide to reduce CSF production proves to worsen outcome. A study showed that compared to the 10 neonates treated with various conventional methods, the 19 neonates with IVH treated with neuroendoscopic lavage had less shunt, fewer infections, and multiloculated hydrocephalus.^[15]

Screening for IVH is recommended for all preterm neonates with gestational age below 30 weeks using ultrasonography at two time points. The first time point is when the infant is between 7 to 14 days old to detect signs of IVH, and the second time point is at 36 to 40 weeks postmenstrual age to detect central nervous system lesions such as ventriculomegaly or periventricular leukomalacia.^[19] There is no recommendation for routine screening of IVH in term infants.

Mortality and sequelae due to IVH ranged from 5% in grade I to 90% in grade IV.^[12] Although lower grades of IVH are attributed to lower risk of cerebral palsy and mental retardation, children with grade I and II IVH are also at risk for developmental disability. According to the US Census Bureau and the Centers for Disease Control, there are more than 3600 cases of mental retardation secondary to IVH in the United States every year, and the total cost of treatment for these children over their lifetimes is more than 3.6 billion dollars. The two most important sequelae of IVH is post-haemorrhagic hydrocephalus (PHH) and periventricular leukomalacia (PVL). PHH resulted from IVH are communicating in the majority of cases, due to an impaired CSF absorption.^[19]

CONCLUSION

This case report described a rare incidence of IVH with post-haemorrhagic hydrocephalus (PHH) in a term infant, manifesting after the first month of birth, with an uneventful perinatal history. The patient presented with rapidly increasing frontal occipital circumference, dilatation of ventricles on radiologic examination, and signs of increased intracranial pressure. EVD remains the primary management for IVH patients presenting with communicating hydrocephalus. Although the incidence of IVH in term infants is much lower than in preterm infants, if not treated accordingly may result in mortality and neurological sequelae in the future. The patient in this case has not assessed with vascular study, such as MRI, MRA, or Perfusion MR, yet. For further evaluation, we planned vascular studies in the next follow up visit. From this case report, it is important for physician to consider the possibility of IVH in term infant presenting with seizure and anaemia.

REFERENCES

- [1] J. T. Kleinman *et al.*, "Evaluation of Intraventricular Hemorrhage in Pediatric Intracerebral Hemorrhage," *J. Child Neurol.*, vol. 27, no. 4, pp. 526–531, Apr. 2012, doi: 10.1177/0883073811422272.
- [2] O. Romantsik *et al.*, "Severe Intraventricular Hemorrhage Causes Long-Lasting Structural Damage in A Preterm Rabbit Pup Model," *Pediatr. Res.*, vol. 92, no. 2, pp. 403–414, Aug. 2022, doi: 10.1038/s41390-022-02075-y.



- [3] K. G. Holste, F. Xia, F. Ye, R. F. Keep, and G. Xi, "Mechanisms of Neuroinflammation in Hydrocephalus After Intraventricular Hemorrhage: A Review," *Fluids Barriers CNS*, vol. 19, no. 1, p. 28, Dec. 2022, doi: 10.1186/s12987-022-00324-0.
- [4] R. M. Starke *et al.*, "International Multicenter Cohort Study of Pediatric Brain Arteriovenous Malformations. Part 2: Outcomes After Stereotactic Radiosurgery," *J. Neurosurg. Pediatr.*, vol. 19, no. 2, pp. 136–148, Feb. 2017, doi: 10.3171/2016.9.PEDS16284.
- [5] T. Lee, D. Nam, W. S. Chang, and H.-Y. Mun, "Recurrence of Pediatric Cerebral Arteriovenous Malformation After Obliteration by Radiosurgery: A Case Report," *J. Korean Soc. Stereotact. Funct. Neurosurg.*, vol. 19, no. 2, pp. 65–70, Sep. 2023, doi: 10.52662/jksfn.2023.00080.
- [6] T. Blauwblomme *et al.*, "Long-Term Outcome of 106 Consecutive Pediatric Ruptured Brain Arteriovenous Malformations After Combined Treatment," *Stroke*, vol. 45, no. 6, pp. 1664–1671, Jun. 2014, doi: 10.1161/STROKEAHA.113.004292.
- [7] U. Ciochon *et al.*, "Causes and Risk Factors of Pediatric Spontaneous Intracranial Hemorrhage - A Systematic Review," *Diagnostics*, vol. 12, no. 6, p. 1459, Jun. 2022, doi: 10.3390/diagnostics12061459.
- [8] L. A. Beslow *et al.*, "Predictors of Outcome in Childhood Intracerebral Hemorrhage," *Stroke*, vol. 41, no. 2, pp. 313–318, Feb. 2010, doi: 10.1161/STROKEAHA.109.568071.
- [9] L. Afsharkhas, N. Khalessi, and M. Karimi Panah, "Intraventricular Hemorrhage in Term Neonates: Sources, Severity and Outcome," *Iran. J. child Neurol.*, vol. 9, no. 3, pp. 34–9, 2015, [Online]. Available: <http://www.ncbi.nlm.nih.gov/pubmed/26401151>
- [10] D. Bhattacharya, I. K. Sharawat, and L. Saini, "Intraventricular Haemorrhage and Obstructive Hydrocephalus in A Term Neonate: An Uncommon Presentation of Haemophilia B," *BMJ Case Rep.*, vol. 9, no. 3, pp. 34–39, May 2018, doi: 10.1136/bcr-2018-225341.
- [11] M. Dey, J. Jaffe, A. Stadnik, and I. A. Awad, "External Ventricular Drainage for Intraventricular Hemorrhage," *Curr. Neurol. Neurosci. Rep.*, vol. 12, no. 1, pp. 24–33, Feb. 2012, doi: 10.1007/s11910-011-0231-x.
- [12] T. Garton, Y. Hua, J. Xiang, G. Xi, and R. F. Keep, "Challenges for Intraventricular Hemorrhage Research and Emerging Therapeutic Targets," *Expert Opin. Ther. Targets*, vol. 21, no. 12, pp. 1111–1122, Dec. 2017, doi: 10.1080/14728222.2017.1397628.
- [13] V. Gilard, A. Tebani, S. Bekri, and S. Marret, "Intraventricular Hemorrhage in Very Preterm Infants: A Comprehensive Review," *J. Clin. Med.*, vol. 9, no. 8, p. 2447, Jul. 2020, doi: 10.3390/jcm9082447.
- [14] R. Guo *et al.*, "Primary Intraventricular Hemorrhage in Pediatric Patients: Causes, Characteristics, and Outcomes." *World Neurosurgery*, Feb. 05, 2019. doi: 10.21203/rs.2.296/v1.
- [15] A. Kirmani, A. Sarmast, and A. Bhat, "Role of External Ventricular Drainage in The Management of Intraventricular Hemorrhage; its Complications and Management," *Surg. Neurol. Int.*, vol. 6, no. 1, p. 188, 2015, doi: 10.4103/2152-7806.172533.
- [16] M.-F. Kuo, "Surgical Management of Intraventricular Hemorrhage and Posthemorrhagic Hydrocephalus in Premature Infants," *Biomed. J.*, vol. 43, no. 3, pp. 268–276, Jun. 2020, doi: 10.1016/j.bj.2020.03.006.
- [17] H. J. McCrea and L. R. Ment, "The Diagnosis, Management, and Postnatal Prevention of Intraventricular Hemorrhage in The Preterm Neonate," *Clin. Perinatol.*, vol. 35, no. 4, pp. 1–17, Dec. 2008, doi: 10.1016/j.clp.2008.07.014.
- [18] C. Siffel, K. D. Kistler, and S. P. Sarda, "Global Incidence of Intraventricular Hemorrhage Among Extremely Preterm Infants: A Systematic Literature Review," *J. Perinat. Med.*, vol. 49, no. 9, pp. 1017–1026, Nov. 2021, doi: 10.1515/jpm-2020-0331.
- [19] D. Szpecht, D. Frydryszak, N. Miszczyk, M. Szymankiewicz, and J. Gadzinowski, "The Incidence of Severe Intraventricular Hemorrhage Based on Retrospective Analysis of 35939 Full-Term Newborns-Report of Two Cases and Review of Literature," *Child's Nerv. Syst. Off. J. Int. Soc. Pediatr. Neurosurg.*, vol. 32, no. 12, pp. 2447–2451, Dec. 2016, doi: 10.1007/s00381-016-3164-5.
- [20] D. Szpecht, M. Szymankiewicz, I. Nowak, and J. Gadzinowski, "Intraventricular Hemorrhage in Neonates



- Born Before 32 Weeks of Gestation-Retrospective Analysis of Risk Factors,” *Child’s Nerv. Syst. Off. J. Int. Soc. Pediatr. Neurosurg.*, vol. 32, no. 8, pp. 1399–404, Aug. 2016, doi: 10.1007/s00381-016-3127-x.
- [21] S. G. Kersin, “Prematürede Intraventriküler Kanama,” *Türk Pediatr. Arşivi*, vol. 55, no. 3, pp. 215–221, 2020, doi: 10.14744/TurkPediatriArs.2020.66742.

Ethical approval

Institutional Review Board approval is not required.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Conflicts of interest

There are no conflicts of interest.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.



Figure 1: Clinical Pictures from Physical Examination (26/11/2022)



Figure 2: Babygram (26/11/2022)

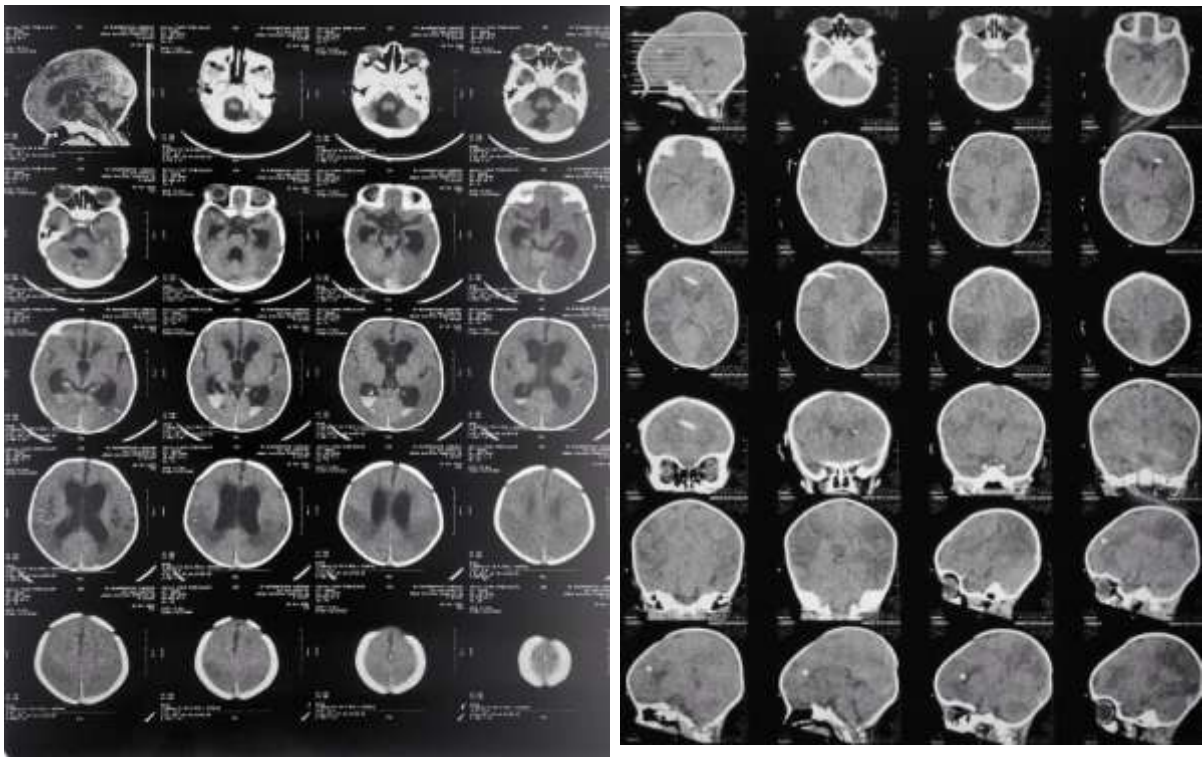


Figure 3: Head CT-scan, non-contrast (left 26/11/2022; right 4/12/2022)

Table 1. Intraventricular haemorrhage grading using cranial computed tomography (from: Özek *et al.* 2020)

Grade I	Germinal matrix haemorrhage
Grade II	Haemorrhage fills less than 50% of the lateral ventricle
Grade III	Haemorrhage fills and enlarges the lateral ventricle
Grade IV	Intraparenchymal haemorrhage

Table 2. Intraventricular haemorrhage grading using ultrasonography (from: Özek *et al.* 2020)

Grade I	Germinal matrix haemorrhage (no or minimal haemorrhage in the ventricle)
Grade II	IVH filling 10-50% of the ventricle at the parasagittal section
Grade III	IVH filling more than 50% of the ventricle and causing ventricular enlargement
Grade IV	Periventricular echodensity