Case Report

Intraventricular hemorrhage possibly extending from an infratentorial subdural hematoma via a perforated suprapineal recessus in a full-term neonate

Yoichiro Serita, Takato Morioka, Nobuya Murakami, Nobuko Kawamura, Yasushi Takahata, Ryutaro Kira

Departments of Neonatology, 1Neurosurgery, 2Clinical Radiology, 3Pediatric Neurology, Fukuoka Children’s Hospital, Fukuoka, Japan

E-mail: Yoichiro Serita - serita.y@fcho.jp; Takato Morioka - takato@ns.med.kyushu-u.ac.jp; Nobuya Murakami - murakami.n@fcho.jp; Nobuko Kawamura - kawamura.n@fcho.jp; Yasushi Takahata - takahata.y@fcho.jp; Ryutaro Kira - kira.r@fcho.jp

*Corresponding author

Received: 12 April 17  Accepted: 21 June 17  Published: 01 November 17

Abstract

Background: Although intraventricular hemorrhage (IVH) is very rarely reported in full-term neonates, it may occur in children with perinatal trauma, asphyxia, and coagulation disorders, and may originate in the choroid plexus and residual subependymal germinal matrix layer.

Case Description: We present the case of a full-term baby with IVH. She had no perinatal problems or coagulation disorders. Sagittal views of neuroimages demonstrated that the IVH possibly extended from a subdural hemorrhage (SDH) in the infratentorial area via a perforated suprapineal recessus. This was barely visible on a conventional axial view of a computed tomographic scan.

Conclusion: When the etiopathogenesis of IVH in a full-term baby with an uncomplicated delivery cannot be clearly defined, multi-directional and multi-modal neuroimaging may be useful.

Key Words: Full-term birth, infratentorial subdural hemorrhage, intraventricular hemorrhage, suprapineal recessus

INTRODUCTION

Intraventricular hemorrhage (IVH) in the subependymal germinal matrix layer is the most common type of neonatal IVH. It is characteristically observed in premature infants, and the younger the gestational age, the more often it occurs. IVH is very rarely reported in full-term neonates. However, it may occur in these children with various clinical conditions, and is mostly related to perinatal trauma, asphyxia, and coagulation disorders. Often, IVH in full-term neonates originates in the choroid plexus and residual subependymal germinal matrix layer.

Here, we report the case of a full-term baby with IVH, which possibly extend from a subdural hemorrhage (SDH) in the infratentorial area via a perforated suprapineal recessus.

This is an open access article distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 3.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as the author is credited and the new creations are licensed under the identical terms.

For reprints contact: reprints@medknow.com

How to cite this article: Serita Y, Morioka T, Murakami N, Kawamura N, Takahata Y, Kira R. Intraventricular hemorrhage possibly extending from an infratentorial subdural hematoma via a perforated suprapineal recessus in a full-term neonate. Surg Neurol Int 2017;8:266.

http://surgicalneurologyint.com/Intraventricular-hemorrhage-possibly-extending-from-an-infratentorial-subdural-hematoma-via-a-perforated-suprapineal-recessus-in-a-full-term-neonate/
CASE REPORT

A full-term (37 weeks and 4 days) baby girl was born in good general condition by vaginal delivery. Her 22-year-old mother (gravida 1, para 1) experienced an unremarkable pregnancy, apart from testing positive for chlamydia antibody at 28 weeks, for which she received azithromycin. The girl’s birth weight was 2862 g, length 48 cm, head circumference 33 cm, and Apgar score 9/10. Thirty minutes after her birth, sporadic ventricular premature contractions were frequently observed. She started breastfeeding 8 hours after birth, and she vomited a couple of times. She developed apneic attacks during the night. Subsequently, generalized seizure was observed in the morning of the next day and she was transferred to our hospital.

On arrival, the girl was not very active. There were no trauma-related lesions, including cephalohematoma, on her outer surface. Her seizure was controlled using intravenous administration of phenobarbital. Peripheral blood count and coagulation tests, including prothrombin time and activated partial thromboplastin time, were within normal limits. Ultrasound imaging of her head showed a high echoic lesion in the lateral ventricles. Axial computed tomographic (CT) images revealed IVH in the bilateral lateral ventricles, the 3rd ventricle and the 4th ventricle. However, SDH with subarachnoid hemorrhage (SAH) was barely visible compared with the IVH [Figure 1a]. Nonetheless, mid-sagittal views clearly demonstrated SDH with SAH in the infratentorial area and the tentorial incisura, which was seen to be connected to the IVH in the 3rd ventricle via the superior pineal recessus [Figure 1b]. The density of the IVH was more intense than that of SDH.

Because her fontanel was not tense and general conditions were good, conservative therapy for the intracranial hemorrhage was selected. Although protein C activity was slightly decreased (probably due to her intracranial hemorrhage), protein S activity, antithrombin III, and factor XIII were within normal limits.

Around day 13 after birth, the girl’s head circumference had gradually increased in size. On day 18, magnetic resonance (MR) images showed marked enlargement of the entire ventricular system [Figure 2a]. Although most of the IVH was resolved [Figure 2a], the infratentorial SDH persisted [Figure 2b]. The 3D heavily T2-weighted imaging (3D-hT2WI) showed internal cerebral veins (ICVs) visualized as a linear signal void at the roof of the 3rd ventricle and an enlarged suprapineal recessus [Figure 2c]. MR venography revealed an intact Galenic venous system, including ICVs. T2*-weighted imaging and MR angiography failed to reveal the causative bleeding point, which included the subependymal layer and the choroid plexus.

On Day 19, the girl underwent a ventricle-peritoneal (VP) shunt through the right anterior horn. Her postoperative course was uneventful and her development corresponded to her age.

DISCUSSION

In this full-term baby, without coagulation disorders, both massive IVH and infratentorial SDH was observed...
at first CT scan. Although most of the IVH was resolved, infratentorial SDH persisted at follow-up MRI. However, the causative bleeding spot of the IVH was not noted. These findings might indicate that the SDH was the primary hemorrhagic site and the IVH was an extension of the infratentorial SDH.

The most common etiology of infratentorial SDH is disruption of the tentorium or falx with tearing of the bridging veins due to mechanical compression and distortion of the head during birth.\[^2\-^7,^11\] Massive SDH may also occur after dural sinus and rupture of the Galenic venous system. Because infratentorial SDH in term infants is frequently associated with breech delivery, use of forceps, and prolonged labor with cranial molding, infratentorial SDH has been considered to be a traumatic lesion related to difficult parturition.\[^2\-^4,^7,^11\]

However, recent studies using modern neuroimaging techniques have concluded that the presence of SDH is not necessarily indicative of excessive birth trauma and may occur as the sequela of an otherwise uncomplicated delivery.\[^1\-^3,^12\] Nonetheless, one should be alert to possible underlying causes, including coagulation abnormalities.\[^1,^2\]

Because neither difficult parturition nor coagulation abnormalities were observed, the present case could be diagnosed as a “spontaneous” infratentorial SDH. MR venography and 3D-hT2WI did not show any rupture of the Galenic venous system, therefore, the possible causative bleeding spot was injury of the bridging cerebellar veins.

Spontaneous SDH located in the infratentorial region is difficult to see on axial CT scans, since the axial plane is not completely parallel to the tentorium, and it can therefore be overlooked.\[^12\]

In our case, from the axial view, the infratentorial SDH was barely visible, while the massive IVH was clearly visible. However, mid-sagittal views clearly demonstrated that the infratentorial SDH could be connected to the IVH in the 3rd ventricle via the superior pineal recessus [Figure 1b]. The suprapineal recessus projects posteriorly between the upper surface of the pineal gland and the lower layer of the tela choroidea in the roof of the 3rd ventricle.\[^10\] The ependymal tissue of the suprapineal recessus is thought to be thin in neonates. Thus, it is reasonable that the extension and pressure of the infratentorial SDH may have directly perforated the ependyma of the suprapineal recessus [Figure 3]. If the bleeding spot was adjacent to the suprapineal recessus, it could be speculated that the direct pressure of the SDH would be large enough to perforate the suprapineal recess, with a large hematoma extending into the ventricles. This is a possible reason why the density of the IVH was greater than that of the SDH at the initial CT scan. While our hypothesis was not proved and the possibility of the simultaneous occurrence of IVH and SDH cannot be completely excluded, when the etiopathogenesis of IVH cannot be defined clearly on conventional axial CT scans, multi-directional and multi-modal neuroimaging may be recommended.\[^10\]

The outcome of the infratentorial SDH depends on the parenchymal and brainstem involvement.\[^4,^7\] When the infratentorial SDH is massive, the large hematoma extends to the cerebellar parenchyma, compressing the 4th ventricle and the brainstem.\[^4,^7\] In the present case, no parenchymal or brainstem involvement were observed because the infratentorial SDH probably drained into the ventricles through a perforated suprapineal recessus. Because a VP shunt successfully controlled the patient’s post-IVH communicating hydrocephalus, her prognosis is expected to be good.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

REFERENCES

1. Ahmad K, Zafar Janjua N, Bin Hamza H, Imran Khan M. Spontaneous subdural haemorrhage in new born babies. Lancet 2004;363:2001-2.
2. Brouwer AJ, Groenendaal F, Koopman C, Nievelstein R-JA, Han SK, de Vries LS. Intracranial hemorrhage in full-term newborns: A hospital-based cohort study. Neuroradiology 2010;52:567-76.
3. Chamnanvanakij S, Rollins N, Perlman J-M. Subdural hematoma in term infants. Pediat Neurol 2002;26:301-4.
4. Hayashi T, Hashimoto T, Fukuda S, Oshihama Y, Moritaka K. Neonatal subdural hematoma secondary to birth injury. Clinical analysis of 48 survivors. Childs Nerv Syst 1987;3:23-9.
5. Ichiyama M, Ohga S, Ochiai M, Fukushima K, Ichimura M, Torio N, et al. Fetal hydrocephalus and neonatal stroke as the first presentation of protein C deficiency. Brain Dev 2016;38:253-6.
6. Moriooka T, Hashiguchi K, Nagata S, Miyagi Y, Mihara F, Hikino S, et al.
7. Perrin RG, Rutka JT, Drake JM, Maltzer H, Hellman J, Jay V, et al. Management and outcome of posterior fossa subdural hematomas in neonates. Neurosurgery 1997;40:1190-200.

8. Rhoton AL, ed. The lateral and third ventricles. In: RHOTON Cranial anatomy and surgical approaches. Maryland: Lippincott Williams & Wilkins; 2003. p. 235-99.

9. Sahriarian S, Akbari P, Amini E, Dalili H, Esmaeilnia Shrivany T, Niknafs N, et al. Intraventricular hemorrhage in a term neonate: Manifestation of protein S deficiency. A case report. Iran J Public Health 2016;45:531-4.

10. Szpecht D, Frydryszak D, Miszeszk N, Szymankiewicz M, Gadzinowski J. The incidence of severe intraventricular hemorrhage based on retrospective analysis of 35939 full-term newborns. Report of two cases and review of literature. Childs Nerv Syst 2016;32:2447-51.

11. Usul H, Karaarslan G, Cakir E, Kuzeyl K, Mungan I, Baykal S. Conservative management of spontaneous posterior fossa subdural hematoma in a neonate. J Clin Neurosci 2004;12:196-8.

12. Whitby EH, Griffiths PD, Rutter S, Smith MF, Springa A, Ohadike P, et al. Frequency and natural history of subdural hematomas in babies and relation to obstetric factors. Lancet 2003;362:846-50.