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ORIGINAL ARTICLE

Subpial Hemorrhage

A Distinctive Neonatal Stroke Pattern

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Abstract

Background and Purpose Subpial hemorrhage is a rare form of neonatal stroke, still poorly understood. The aim of this study was to characterize a cohort of term and preterm neonates with subpial hemorrhages and contribute to a better knowledge of this condition.

Material and Methods Clinical records and magnetic resonance (MR) imaging data of all neonates with subpial hemorrhage followed at a pediatric hospital between 2010 and 2020 were retrospectively reviewed.

Results A total of 10 patients were included in the analysis, 40% of whom were term neonates. Operative vaginal delivery was registered in 30%. Temporal was the most common location of subpial hemorrhage (70%), and all patients displayed underlying brain infarction. A characteristic yin-yang pattern was present in 90% of the study cohort, and ingurgitation of medullary veins on susceptibility weighted imaging in 80%. Cerebellar microbleeds were observed in 60% of neonates, both term and preterm. When available, MR angiography and venography were unremarkable. Patients' clinical outcome was variable, with early prematurity not associated to worse outcomes.

Conclusion Subpial hemorrhage has a distinctive MR pattern, with underlying parenchymal venous infarction, and can occur in term and preterm neonates. This study results suggest an association between subpial hemorrhage and cerebellar microbleeds but further studies are required to confirm it and better understand the pathophysiology of subpial hemorrhage.

Keywords Perinatal stroke · MRI · Neonate · Bleeding · SWI

Background

Neonatal stroke, occurring in the first 28 days of life, comprises both ischemic and hemorrhagic stroke [1]. Among neonatal hemorrhagic stroke, a particular subpial hemorrhage subtype has been seldom described. Although the first descriptions of this entity date back to 1972 [2], it was not until 2004 that subpial hemorrhage was confirmed on brain magnetic resonance imaging (MRI) in seven patients in the study by Huang and Robertson [3]. Since then, there has been an increasing awareness of this neonatal stroke pattern [4]. Cain et al. [5] and Assis et al. [6] reported the cases of 17 and 16 neonates with this hemorrhage subtype, respectively, contributing to increasing the knowledge of this entity. Subpial hemorrhage is clinically important because it is associated with injury to adjacent brain parenchyma. It mainly occurs in term and late pre-term neonates [3, 5] and, despite the exuberant presentation on brain MRI, the outcome is usually good [5]. Nevertheless, the literature

Availability of Data and Material All data are available on request.

Code Availability Not applicable.

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on this subject is still scarce and the pathophysiology of subpial hemorrhage remains poorly understood.

The aim of this study was to characterize a cohort of neonates with subpial hemorrhage followed at a pediatric hospital over a 10-year period, increasing knowledge of this entity, improving the understanding of its specific image patterns, and ultimately raising awareness of this rare stroke presentation.

Material and Methods

This was a single-center retrospective study of clinical records and MRI data of all neonates presenting with intracranial bleeding, as registered in the neuroimaging teaching database of Hospital Dona Estefânia—Centro Hospitalar Universitário de Lisboa Central (CHULC, Lisbon, Portugal), between 2010 and 2020. Patients with subpial hemorrhage were identified by a senior pediatric neuroradiologist with 19 years of experience. The following criteria described by Cain et al. [5] were used to confirm the presence of subpial hemorrhage: (1) hemorrhage closely opposing the underlying sulci and (2) pooling of blood products relatively localized rather than spreading along the convexity. This study was approved by the Clinical Research Ethical Committee of the CHULC. Informed consent was waived.

Clinical Data

Demographic and clinical data, laboratory findings and clinical outcome of all patients were retrieved from clinical records.

MRI Acquisition

All examinations were performed on a *Signa* 3.0T MR system (GE Healthcare, Chicago, IL, USA) with an 8-channel phased array head coil.

Due to the extension of the period under analysis (from 2010 to 2020), acquisition parameters slightly varied over the years.

Still, the following sequences were acquired in all patients: axial T1-weighted imaging (WI), axial and coronal fast spin echo (FSE) T2-WI, axial diffusion-weighted imaging (DWI) and corresponding apparent diffusion coefficient (ADC) map, and axial gradient recalled echo (GRE) T2*-WI. In more recent scans, gradient-echo susceptibility weighted imaging (SWI; SWAN) was performed instead of T2*-WI. Some patients were additionally assessed with 3D time-of-flight (TOF) MR angiography (MRA) and 2D TOF MR venography (MRV). Follow-up images were analyzed whenever available.

Image Analysis

All images were analyzed by a resident neuroradiologist with 4 years experience supervised by a senior neuroradiologist. Both were blinded to all clinical information and outcomes.

Hemorrhage location, size, morphology, and signal intensity were assessed as well as brain parenchyma for signal intensity changes.

Results

Patient Cohort

A total of 10 neonates were included in the analysis, 6 of whom were male and 4 female. Of the patients, 4 were born at term (≥ 37 weeks), another 4 were moderate-to-late preterm (≥ 32–< 37 weeks), and 2 were early preterm (≥ 28–32 weeks).

Pregnancies were complicated by intrauterine growth restriction in two cases, chorioamnionitis in one case, oligohydramnios in two cases, hydramnios in two cases and gestational diabetes in one case. Among these pregnancies, two had a previous history of neonatal hemorrhage (one idiopathic and one related to perinatal alloimmune thrombocytopenia). Vaginal delivery occurred in six cases (three of which were instrumental deliveries), and cesarean delivery in four. The mean APGAR score at 1 min and 5 min was 7 and 9, respectively.

The most common clinical presentation was respiratory distress and apneic events in seven patients, followed by seizures in two. Thrombocytopenia was reported in six patients (related to perinatal alloimmune thrombocytopenia in one) but no coagulation disorders were observed.

At follow-up, four patients presented with hemiparesis, and one patient with tetraparesis. One patient additionally developed ipsilateral hemianopia. Global and language development delay were present in two patients each. One patient with complex cardiopathy (probable CHARGE syndrome, all clinical picture was according, but the diagnosis was not genetically confirmed) died at the age of 1 month due to progressive heart failure. Only two neonates became completely asymptomatic.

Early preterm did not present worse outcomes compared to term or moderate-to-late preterm (both groups had mild hemiparesis).

Demographic and clinical data of the study cohort are depicted in Table 1.

Table 1 Demographic and clinical data of the study cohort

Characteristic		
Sex (*n*)	Male	6/10
	Female	4/10
Gestational age at birth (*n*)	Term (≥ 37 weeks)	4/10
	Moderate-to-late preterm (≥ 32 and < 37 weeks)	4/10
	Early preterm (≥ 28 and < 32 weeks)	2/10
	Extreme preterm (< 28 weeks)	0/10
Delivery (*n*)	Vaginal eutocic	3/10
	Vaginal dystocic	3/10
	Cesarean	4/10
Clinical presentation (*n*)	Apnea/respiratory distress	7/10
	Myoclonic seizures	2/10
	Hypotension and metabolic acidosis	1/10
Apgar score (mean (range))	1 min	6.7 (2–9)
	5 min	8.9 (5–10)
Laboratory findings (*n*)	Coagulopathy	0/10
	Thrombocytopenia	6/10
	Low hemoglobin	1/10
Outcomes (*n*)	No neurologic deficits	2/10
	Motor neurologic deficits	5/10
	Language delay	2/10
	Global development delay	2/10
	Epilepsy	1/10
	Death	1/10
Age at last follow-up (median months (range))	–	26 (2–72)

Table 2 Brain MRI findings in the study cohort

Location (lobes involved, more than one possible) (*n*)	
Temporal	7/10
Frontal	3/10
Parietal	3/10
Occipital	2/10
Parenchymal cytotoxic edema (*n*)	10/10
Parenchymal hemorrhage (*n*)	9/10
Prominent medullary veins (fan-shaped) (*n*)	8/10
Yin-yang sign (*n*)	9/10
Cerebellum microbleeds (*n*)	6/10
MRA	*n* = 4, all negative
MRV	*n* = 2, all negative

MRA magnetic resonance angiography, *MRI* magnetic resonance imaging, *MRV* magnetic resonance venography

Imaging Findings

Detailed description of brain MRI findings are presented in Table 2. The temporal lobe was most commonly affected (70%), followed by frontal, parietal and occipital locations (Fig. 1). No infratentorial subpial hemorrhage was identified. The extension of hemorrhage ranged from as small as 1.8 cm to extensive, almost hemispheric bleeding. Regarding morphology, subpial hemorrhage presented as blood collections that tended to pool rather than spread along the convexity (unlike subarachnoid hemorrhage). Subpial bleeding extended into the adjacent cerebral sulci, widening them, as well as displacing and flattening the underlying parenchyma (Fig. 2). Importantly, subpial hemorrhage overlayed the cortex but was separated from adjacent cerebrospinal fluid (CSF), which further helped to distinguish it from subarachnoid hemorrhage (Fig. 2).

Three patterns of subpial hemorrhage were identified (Fig. 3): in pattern 1, the most common, blood collection presented with T2 hypointense and T1 hyperintense signal; in pattern 2, blood collection presented with T2 hyperintense and T1 hyperintense signal; and in pattern 3, the least common, blood collection presented with mixed T2 signal. A pooled simple fluid interposed between the subpial hemorrhage and the displaced cortex was observed in pattern 3.

On DWI and ADC maps, blood collections showed increased diffusivity, consistent with acute or subacute bleeding (Fig. 4).

Regarding the underlying parenchyma, in addition to the previously described morphological change, mixed-signal changes mainly characterized by T2 hyperintense areas were present in both cortical and subcortical white matter regions (Fig. 5). In some cases, T2 hypointense foci were found, reflecting brain parenchymal hemorrhage. On DWI and ADC maps, peripheral cortical and subcortical restricted diffusion was seen in all patients, consistent with brain parenchymal infarction with cytotoxic edema (Fig. 4).

In nine patients, signal changes from the blood collection and underlying infarcted parenchyma originated a characteristic pattern on T2-WI resembling the yin-yang symbol of the Chinese philosophy, with a T2 hypointense side corresponding to the focal subpial collection of blood extending into cerebral sulci (consistent with acute bleeding) and a T2 hyperintense side reflecting the underlying compressed and infarcted cerebral cortex and white matter (Fig. 5).

An additional distinctive pattern of subpial hemorrhage was identified, characterized by radiating fan-shaped linear T2 hypointensity along the infarcted parenchyma, consistent with hemorrhage and ingurgitation of the medullary veins and displaying blooming on SWI (Fig. 6). Finally, focal cerebellar microhemorrhages were identified on SWI in six neonates. In these patients, apart from brain parenchyma underlying the subpial hemorrhage, no other microbleeds were identified across the supratentorial parenchyma.

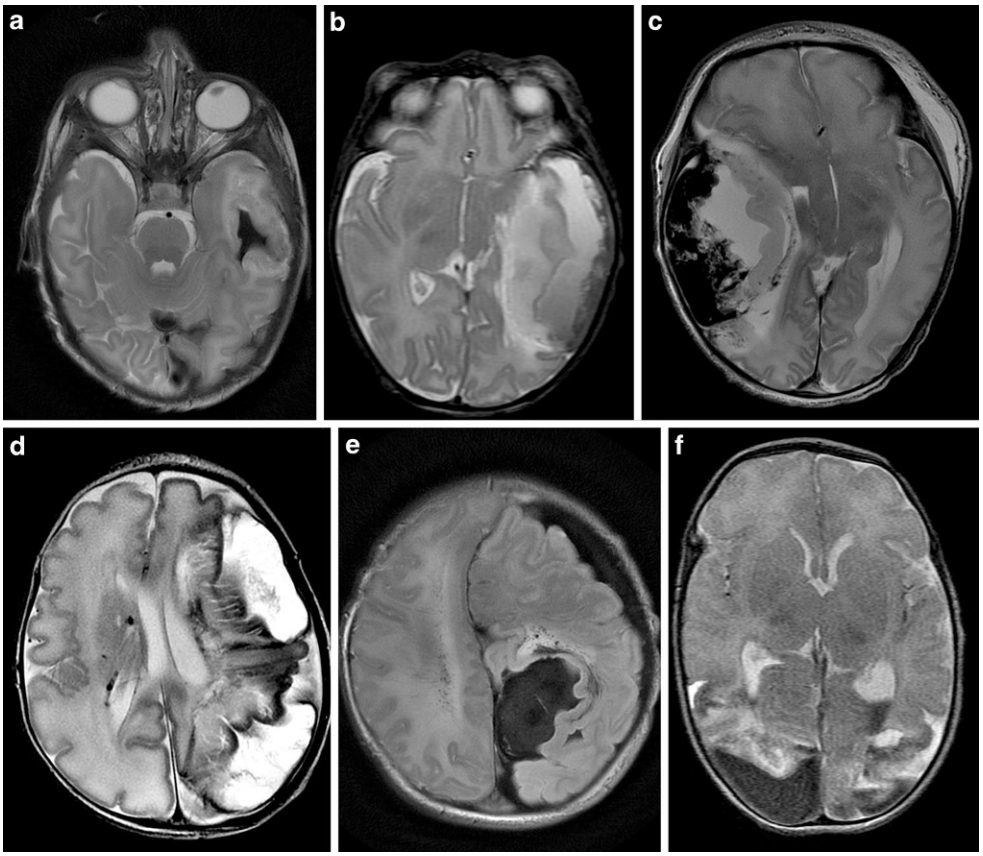

Fig. 1 Axial T2-weighted imaging showing different presentations and locations of subpial hemorrhage in six neonates. The most common location is temporal (**a**–**c**), followed by frontal (**d**) and parietal (**e**), sometimes coexisting (**d**). The less common occipital location was also observed (**f**)

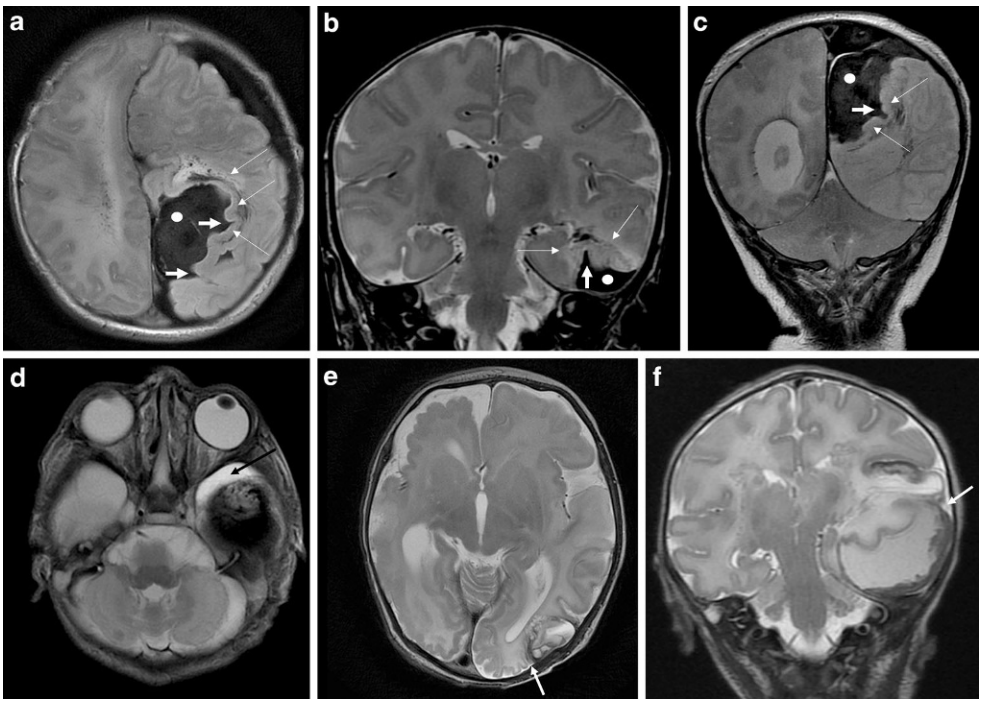

Fig. 2 T2-weighted imaging on axial (**a**, **d**, **e**) and coronal (**b**, **c**, **f**) plans. Subpial hemorrhage tends to pool (*white circle*, **a**–**c**) and extend into the adjacent cerebral sulci (*bold arrows*, **a**–**c**). In addition, cerebral sulci are widened, while the underlying parenchyma is displaced and flattened (*long, thin arrows*, **a**–**c**). Subpial hemorrhage also typically overlays the cortex, although keeping separated from adjacent cerebrospinal fluid (*black arrow* on **d**, *white arrows* on **e**–**f**)

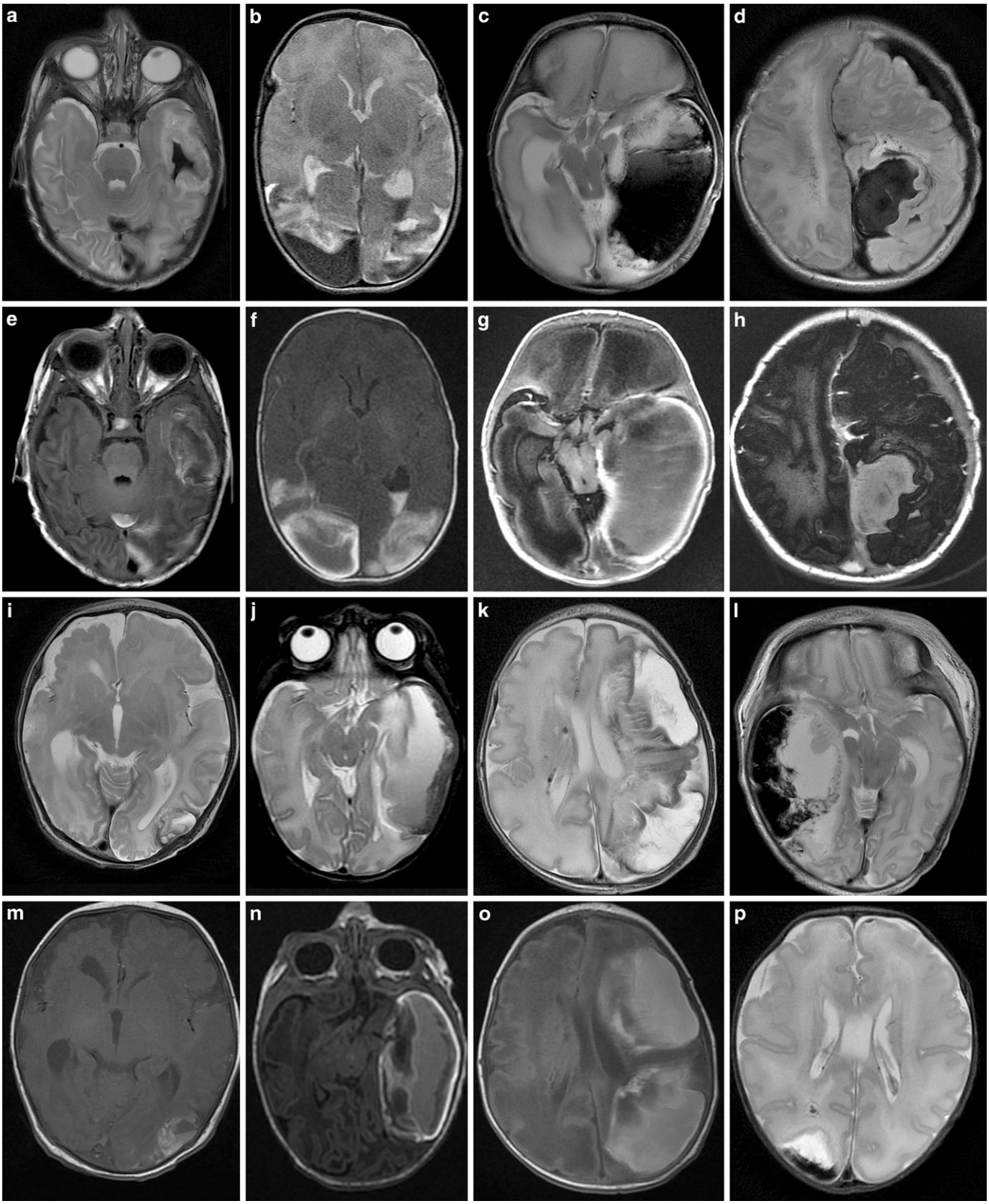

Fig. 3 Patterns of subpial hemorrhage. Pattern 1: top row, axial T2-weighted imaging showing T2 hypointense signal within blood collection (**a**–**d**), and second row, T1-weighted imaging showing T1 hyperintense signal within blood collection (**e**–**h**). Pattern 2: axial T2-weighted imaging showing hyperintensity (**i**–**k**) and T1-weighted imaging also showing hypersignal (**m**–**o**). Pattern 3: T2-weighted imaging showing mixed signal intensity (**l** and **p**), with hypersignal adjacent to the cortex and hyposignal on the periphery

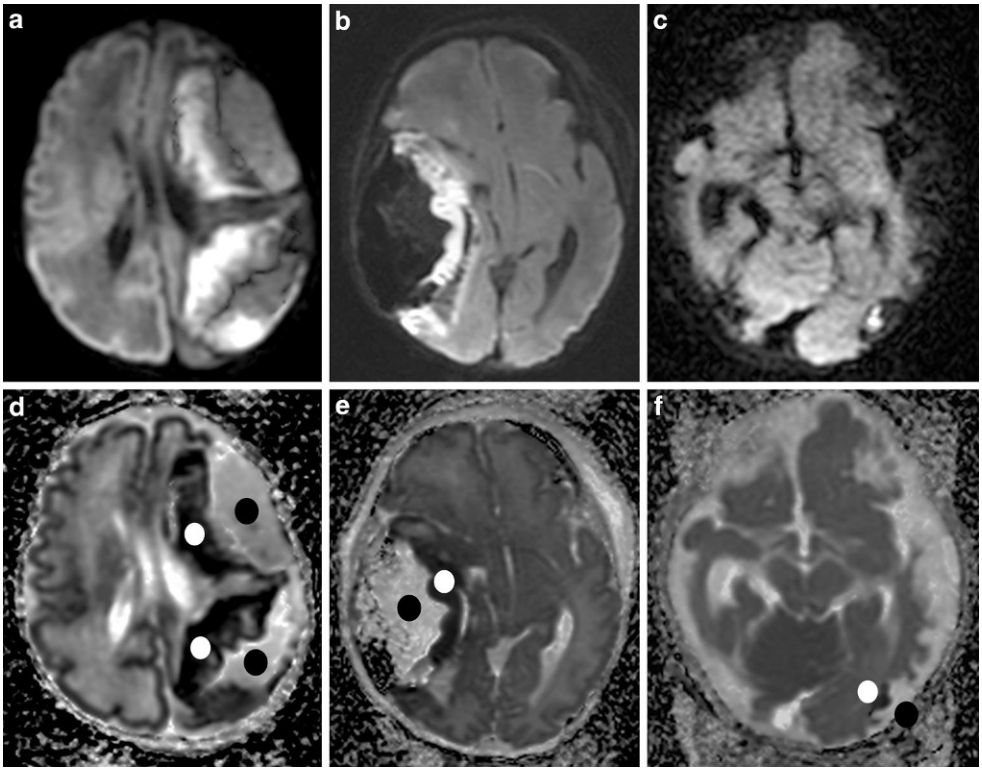

Fig. 4 Axial diffusion-weighted imaging (DWI) trace images (**a**–**c**) showing low signal intensity in blood collection, corresponding to high signal intensity on apparent diffusion coefficient (ADC) maps (*black circles*, **d**–**f**). DWI high signal intensity on parenchyma (**a**–**c**), corresponding to low signal on ADC maps (*white circles*, **d**–**f**), reflecting restricted diffusion due to acute brain parenchyma infarction

The MRA and MRV, available in four and two patients in this cohort, respectively, were unremarkable, with no evidence of arteriovenous shunt or dural venous sinus or cerebral venous thrombosis.

Follow-up MRI was available in five patients and showed a peculiar aspect of subpial hemorrhage sequelae, with a thin T2 hypointense capsule with cystic appearance filled with fluid content along the superficial surface, in addition to residual damaged brain tissue (encephalomalacia) along the inner cavity surface (Fig. 7).

Discussion

The present retrospective study described clinical and imaging features of 10 neonates presenting with subpial hemorrhage, further contributing to increase the body of knowledge about this entity.

Results confirm previous reports that most of these types of stroke occur in term and moderate-to-late preterm neonates [3, 5], who accounted for 80% of patients in this cohort; however, subpial hemorrhage was also identified in two early preterm (with 28 and 31 weeks of gestation), supporting findings of the recent study by Assis et al. [6] and raising awareness to the fact that neonatologists should consider subpial hemorrhage even in early preterm. Moreover, this study showed that early preterm did not have worse outcomes compared to the remaining. This disagrees with the findings of Assis et al. [6], reporting that early preterm infants did not survive hospital discharge. Because subpial hemorrhage is a rare presentation, studies to date focusing on the condition have been based on case series with small patient cohorts. Although the cohort included in this study was also small, which represents a limitation to draw conclusions, results retrieved support the notion that subpial hemorrhage also occurs in early preterm and that, unlike previously believed, the outcomes are not necessarily worse than those of later preterm or term neonate.

In previous reports by Huang et al. [3] and Assis et al. [6], maternal and perinatal histories were unremarkable. In this cohort however, pregnancy complications were found in six neonates (60%), representing a slightly higher incidence than previously reported by Cain et al. [5].

Similarly to previous reports [5, 6], the type of delivery did not seem to have an impact on the occurrence or severity of subpial hemorrhage in this study, as the condition was present both in cesarean and vaginal as well as eutocic and dystocic deliveries. Cole et al. [7] showed that neonatal hemorrhagic stroke was not associated with traumatic delivery, which further supports this study findings.

Regarding clinical presentation, apnea and respiratory distress were the most frequent clinical presentation, in agreement with reports in the literature [3, 5, 6].

Although no coagulation disorders were found, 60% of neonates had thrombocytopenia, which represents a slightly higher percentage than reported by Cain et al. [5].

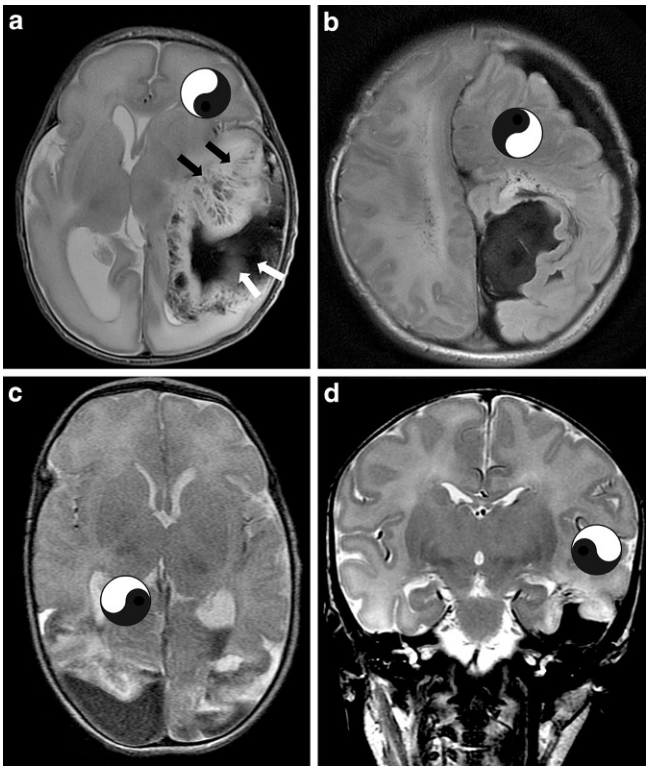

Fig. 5 Axial (**a**–**c**) and coronal (**d**) T2-weighted imaging showing a yin-yang sign. Displaced and flattened brain parenchyma (*black arrows*) underlies the subpial hemorrhage (*white arrows*). T2 hypointense "black side" represents the focal subpial collection of blood extending into cerebral sulci, with signal intensity consistent with acute bleed. T2 hyperintense "bright side" reflects the underlying compressed cerebral cortex and subcortical white matter, with signal intensity consistent with acute venous infarct. The combination of these phenomena originates a characteristic pattern on T2-weighted imaging, resembling the yin-yang symbol

Subpial hemorrhage always presented with underlying parenchyma infarction, even in small hemorrhages, also similarly to Cain et al. [5] and Assis et al. [6] series. The authors believe that adjacent brain infarction cannot be dissociated from subpial hemorrhage and should be considered an integral part of this distinctive stroke subtype presentation. Subpial hemorrhage has been ascribed different names over the years, including: superficial lobar hemorrhage, leptomeningeal hemorrhage, pial hemorrhage, subpial hemorrhage with underlying cerebral infarct, extra-axial bleed with underlying infarct, temporal lobe hemorrhagic venous infarct, hemorrhagic stroke and venous stroke [3, 8–11]. Although the descriptive name subpial hemorrhage with underlying cerebral infarct seems to better reflect this presentation, the simpler subpial hemorrhage designation is more widespread in the literature and hence easier to recognize; however, it should be acknowledged that this pattern of neonatal stroke includes both hemorrhage and brain parenchymal infarction. Regardless of the most accurate term, both experts and the medical community should make an effort to standardize the nomenclature, allowing to establish a common background of knowledge.

Neither brain infarction nor subpial hemorrhage followed a typical arterial territory in this study. Moreover, available MRA results were normal, further supporting the non-arterial nature of this infarction-hemorrhage combination. Like previously reported by Cain et al. [5] and Assis et al. [6], also in this study a fan-shaped hemorrhage was documented within the infarcted parenchyma in addition to the subpial hemorrhage and brain infarction. As described, this hemorrhage followed the medullary venous territories, and enlarged medullary veins were present on SWI with associated blooming, denoting congestion and eventually thrombosis of these veins [12].

The pathophysiology of subpial hemorrhage remains elusive, and although it cannot be confidently assessed in this small retrospective study, image findings support that medullary venous congestion and consequent venous infarction and bleeding may be the most reasonable etiologies for this neonatal stroke subtype.

In a very nice work by Khalatbari et al. [13], the authors discussed different perinatal venous stroke presentations and put forward potential explanations for this type

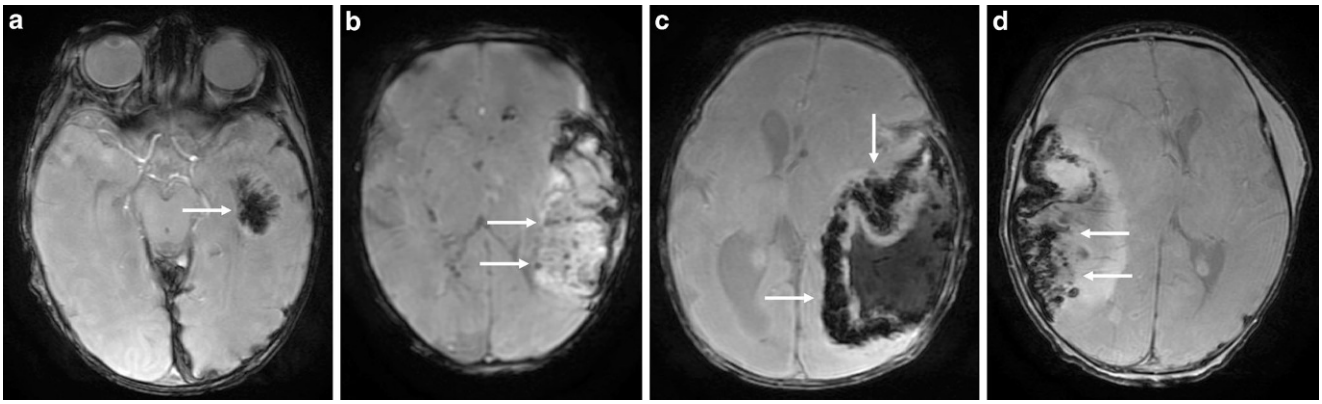

Fig. 6 Axial susceptibility weighted imaging (SWI) scans from different patients (**a–d**) showing ingurgitation/prominence of the medullary veins (*arrows*) underlying the cortical ribbon

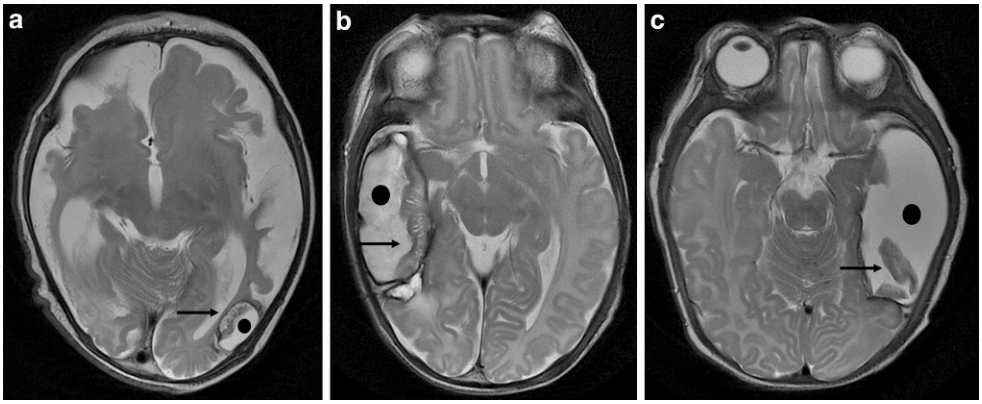

Fig. 7 Subpial hemorrhage axial T2-weighted imaging follow-up from different patients (**a–c**) showing a peculiar pattern, with thin T2 hypointense capsule and capsuled cystic cavity filled with fluid along the superficial surface (bright on T2, *black circle*). Residual damaged brain tissue (encephalomalacia) is seen along the cavity inner surface (*black arrows*)

of infarction. First, the veins coursing through the subpial space are not covered by pia mater, which means that they can bleed directly into the subpial space. Why these subpial vessels rupture and bleeding is still not understood, but one possible explanation could be an insult to the glia limitans with consequent stretching and rupture of the small subpial vessels [14]. Once the bleeding starts, subpial hemorrhage cannot be reabsorbed, since the pia mater is impermeable, and as a result blood accumulates in the contained space under the pia mater and may compress the underlying brain parenchyma. Subpial veins are also compressed, as they have lower blood pressure and lack leptomeningeal coating, increasing pial pressure and obstructing venous outflow, and leading to venous congestion, medullary vein thrombosis, and cortical/subcortical venous infarction [14].

Regarding subpial hemorrhage patterns, the authors speculate that they merely represent different times of evolution of the hemorrhage. The typical pattern of acute/recent bleeding can be recognized in pattern 1, while pattern 2 probably depicts evolution to a chronic state, where blood components have been reabsorbed and only a serous collection persists. Pattern 3 probably reflects a middle stage between the previous two. T2 bright fluid must reflect degradation of blood products and entrapment of serous-hematic fluid between the still recent blood collection, which is dark on T2 and parenchyma (blood usually starts to reabsorb from the parenchymal surface to the periphery, as the pia is impermeable).

A characteristic imaging pattern on T2-WI resembling the yin-yang symbol of Chinese philosophy was found in all patients except one, probably due to the small hemorrhage size (this neonate had the smallest hemorrhage). This makes this yin-yang pattern very reliable to identify this type of stroke, as previously reported [6].

Interestingly, bilateral cerebellar microbleeds were identified in 60% of neonates. As far as the authors are aware, no association between subpial hemorrhage and cerebellar hemorrhage has been previously described. It is acknowledged that cerebellar microhemorrhages are common in preterm neonates [15], but in this cohort such microbleeds were observed both in term and preterm neonates and were not present in all preterm neonates. For this reason, the authors hypothesize that the presence of cerebellar microbleeds in the setting of subpial hemorrhage may be independent of gestational age at birth. Although the pathophysiology of cerebellar microbleeds in preterm neonates is not fully understood, two theories are pointed out as most probable [16]: (i) primary cerebellar hemorrhage may result from subpial bleeding within the germinal matrix of the external granular cell layer and the germinal matrix of the fourth ventricle or (ii) is the result of a vaso-occlusive injury in the cerebellar vascular distribution, with venous congestion [17]. In either case, the mechanism may somehow resemble the previously described pathophysiology of subpial hemorrhage, explaining the link between the two entities. The immaturity of cerebellar vasculature comparing to the supratentorial one is pointed out as an important factor contributing to isolated cerebellar microhemorrhages [18, 19]. Nevertheless, further studies are warranted to confirm the association between subpial hemorrhage and presence of cerebellar microbleeds.

Due to the retrospective nature of this study, the length of follow-up greatly varied among patients. Nevertheless, the median follow-up was 26 months, with 4 patients over 3 years of age at the last follow-up. Only one child died due to heart failure in the context of a cardiac malformation unrelated to subpial hemorrhage. Study results support Cain et al. [5] findings that neonatal death from subpial hemorrhage is an uncommon event. On the other hand, the fact that only two neonates became completely asymptomatic suggests that subpial hemorrhage is not benign, and these patients' outcomes may not be as good as previously suggested. The remaining seven children with neurologic sequelae had various clinical presentations, ranging from mild to severe motor impairment. As previously discussed, clinical outcomes did not correlate with gestational age in the present study.

Subpial hemorrhage long-term clinical outcomes are unclear, with Barreto et al. [14] suggesting that the severity of sequelae might be related to the extent of cortical injury and overall hemorrhage volume.

This study has limitations that should be acknowledged, mainly related to its retrospective design and small sample size. Follow-up was not standardized, and a control group was lacking. Still, the authors hope that it can help to better characterize subpial hemorrhage as a rare type of neonatal stroke, raise awareness of the condition, and improve its recognition in the clinical practice. Future studies are required to better understand the pathophysiology of subpial hemorrhage and assess long-term clinical outcomes and radiographic determinants of prognosis.

Conclusion

Subpial hemorrhage is a rare subtype of neonatal stroke, characterized by a distinctive pattern on MR images. Subpial bleeding and parenchymal infarction coexist, probably originating from venous congestion. Although traditionally believed to occur mainly in term neonates, these study results show that it can also present in late and early preterm. The outcome is variable, with preterm babies not displaying worse outcomes that older neonates. Study results suggest an association between subpial hemorrhage and cerebellar microbleeds, but more studies are required to confirm it, as well as to better understand the pathophysiology of this condition.

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Declarations

Conflict of interest C. Pinto, B. Cunha, M.M. Pinto and C. Conceição declare that they have no competing interests.

Ethical standards This retrospective study was performed after consultation with the institutional ethics committee and in accordance with national legal requirements. This study was approved by local Ethics Committee. *Consent to participate*: Informed consent was waived. *Consent for publication*: Informed consent was waived.

References

1. Dunbar M, Kirton A. Perinatal stroke: mechanisms, management, and outcomes of early cerebrovascular brain injury. Lancet Child Adolesc Health. 2018;2:666–76.
2. Friede RL. Subpial hemorrhage in infants. J Neuropathol Exp Neurol. 1972;31:548–56.
3. Huang AH, Robertson RL. Spontaneous superficial parenchymal and leptomeningeal hemorrhage in term neonates. AJNR Am J Neuroradiol. 2004;25:469–75. Erratum in: AJNR Am J Neuroradiol. 2004;35:666.
4. Lee S, Mirsky DM, Beslow LA, Amlie-Lefond C, Danehy AR, Lehman L, Stence NV, Vossough A, Wintermark M, Rivkin MJ; International Paediatric Stroke Study Neuroimaging Consortium and the Paediatric Stroke Neuroimaging Consortium. Pathways for Neuroimaging of Neonatal Stroke. Pediatr Neurol. 2017;69:37–48.
5. Cain DW, Dingman AL, Armstrong J, Stence NV, Jensen AM, Mirsky DM. Subpial Hemorrhage of the Neonate. Stroke. 2020;51:315–8.
6. Assis Z, Kirton A, Pauranik A, Sherriff M, Wei XC. Idiopathic Neonatal Subpial Hemorrhage with Underlying Cerebral Infarct: Imaging Features and Clinical Outcome. AJNR Am J Neuroradiol. 2021;42:185–93.
7. Cole L, Dewey D, Letourneau N, Kaplan BJ, Chaput K, Gallagher C, Hodge J, Floer A, Kirton A. Clinical Characteristics, Risk Factors, and Outcomes Associated With Neonatal Hemorrhagic Stroke: A Population-Based Case-Control Study. JAMA Pediatr. 2017;171:230–8. Erratum in: JAMA Pediatr. 2017;171:602.
8. Bergman I, Bauer RE, Barmada MA, Latchaw RE, Taylor HG, David R, Painter MJ. Intracerebral hemorrhage in the full-term neonatal infant. Pediatrics. 1985;75:488–96.
9. Hanigan WC, Powell FC, Palagallo G, Miller TC. Lobar hemorrhages in full-term neonates. Childs Nerv Syst. 1995;11:276–80.
10. Slaughter L, Egelhoff J, Balmakund T. Neurologic outcome in neonatal temporal lobe hemorrhagic venous infarcts. J Child Neurol. 2009;24:1236–42.
11. Bruno CJ, Beslow LA, Witmer CM, Vossough A, Jordan LC, Zelonis S, Licht DJ, Ichord RN, Smith SE. Haemorrhagic stroke in term and late preterm neonates. Arch Dis Child Fetal Neonatal Ed. 2014;99:F48–53.
12. Kersbergen KJ, Groenendaal F, Benders MJ, de Vries LS. Neonatal cerebral sinovenous thrombosis: neuroimaging and long-term follow-up. J Child Neurol. 2011;26:1111–20.
13. Khalatbari H, Wright JN, Ishak GE, Perez FA, Amlie-Lefond CM, Shaw DWW. Deep medullary vein engorgement and superficial medullary vein engorgement: two patterns of perinatal venous stroke. Pediatr Radiol. 2021;51:675–85.
14. Barreto ARF, Carrasco M, Dabrowski AK, Sun LR, Tekes A. Subpial Hemorrhage in Neonates: What Radiologists Need to Know. AJR Am J Roentgenol. 2021;216:1056–65.
15. Villamor-Martinez E, Fumagalli M, Alomar YI, Passera S, Cavallaro G, Mosca F, Villamor E. Cerebellar Hemorrhage in Preterm Infants: A Meta-Analysis on Risk Factors and Neurodevelopmental Outcome. Front Physiol. 2019;10:800.
16. Limperopoulos C, du Plessis AJ. Disorders of cerebellar growth and development. Curr Opin Pediatr. 2006;18:621–7.
17. Johnsen SD, Tarby TJ, Lewis KS, Bird R, Prenger E. Cerebellar infarction: an unrecognized complication of very low birthweight. J Child Neurol. 2002;17:320–4.
18. Limperopoulos C, Benson CB, Bassan H, Disalvo DN, Kinnamon DD, Moore M, Ringer SA, Volpe JJ, du Plessis AJ. Cerebellar hemorrhage in the preterm infant: ultrasonographic findings and risk factors. Pediatrics. 2005;116:717–24.
19. Volpe JJ. Cerebellum of the premature infant: rapidly developing, vulnerable, clinically important. J Child Neurol. 2009;24:1085–104.