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ORIGINAL PAPER

Subdural hematoma in infants: can it occur spontaneously? Data from a prospective series and critical review of the literature

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Abstract
Background Subdural hematomas (SDH) in infants often result from nonaccidental head injury (NAHI), which is diagnosed based on the absence of history of trauma and the presence of associated lesions. When these are lacking, the possibility of spontaneous SDH in infant (SSDHI) is raised, but this entity is hotly debated; in particular, the lack of positive diagnostic criteria has hampered its recognition. The role of arachnoidomegaly, idiopathic macrocephaly, and dehydration in the pathogenesis of SSDHI is also much discussed.
Purpose We decided to analyze apparent cases of SSDHI from our prospective databank.
Materials and methods We selected cases of SDH in infants without systemic disease, history of trauma, and suspicion of NAHI. All cases had fundoscopy and were evaluated for possible NAHI. Head growth curves were reconstructed in order to differentiate idiopathic from symptomatic macrocrania.
Results Sixteen patients, 14 males and two females, were diagnosed with SSDHI. Twelve patients had idiopathic macrocrania, seven of these being previously diagnosed with arachnoidomegaly on imaging. Five had risk factors for dehydration, including two with severe enteritis. Two patients had mild or moderate retinal hemorrhage, considered not indicative of NAHI. Thirteen patients underwent cerebrospinal fluid drainage. The outcome was favorable in almost all cases; one child has sequels, which were attributable to obstetrical difficulties.
Conclusion SSDHI exists but is rare and cannot be diagnosed unless NAHI has been questioned thoroughly. The absence of traumatic features is not sufficient, and positive elements like macrocrania, arachnoidomegaly, or severe dehydration are necessary for the diagnosis of SSDHI.

Keywords Infantile subdural hematoma · External hydrocephalus · Idiopathic macrocrania · Pathophysiology · Medico legal evaluation

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Introduction

The diagnosis of subdural hematoma (SDH) in infants always raises the question of its cause. Having eliminated nontraumatic causes like vascular diseases and systemic causes of bleeding, the differential diagnosis is mostly between accidental head injury (AHI) and nonaccidental head injury (NAHI). This may represent a medico legal dilemma when the child presents without evidence of abuse and of any suspicious context, but without a history of trauma either. The possibility of spontaneous SDH in infants (SSDHI) has been considered by several authors [5, 19, 25, 33], but published cases of SSDHI are rare in the literature, often poorly documented, and the evidence is sometimes of questionable value. With increased recognition of NAHI [27] and critical review of the published

cases, this entity has shrunk to the point where one no longer knows whether it exists or not. Although many clinicians occasionally make a diagnosis of SSDHI, these observations generally do not meet the present standard of evidence for publication; as a result, the literature may not accurately reflect the clinical experience.

One of the main obstacles to the recognition of the concept of SSDHI is that it is defined mostly by negative features (absence of systemic disease, of signs of trauma, and of suspicious context); in addition, providing proof of the absence of trauma is virtually impossible. Identifying positive elements, like factors predisposing to SSDHI, would be of great interest for this discussion. In particular, the role of arachnoidomegaly (a.k.a. external hydrocephalus) and macrocephaly and of severe dehydration has been supported by several observations in the literature. However, few cases have been documented convincingly enough for NAHI to be eliminated.

We decided to review cases of SSDHI collected from our prospective register and analyze documented cases from the literature in order to try to define this clinicopathological entity and identify predisposing factors.

Materials and methods

We collected prospectively cases of infants admitted in our hospital for apparent head injury with or without history of trauma, as reported earlier [42]. Cases of SDH caused by surgery, shunting, or infection were not included. We selected children with a SDH, defined as a fluid collection between the dura mater and the arachnoid membrane, diagnosed on imaging or during surgery. SDH caused by birth trauma or systemic causes like clotting deficiency or metabolic disorder was excluded. The systematic evaluation included clinical examination, with record of all signs of impact, on the head as well as on other parts of the body; extensive clotting study; skeletal X-ray in case of suspicion of NAHI; and systematic fundoscopy by an ophthalmologist, with repeated study by a neuro-ophthalmologist in case of retinal hemorrhage (RH). The head circumference growth chart was plotted for each patient and expressed in standard deviations (SD) for the age and sex. This allowed us to differentiate idiopathic macrocrania (defined by generally high value of head circumference at birth and regular progression to +2 SD and beyond), from symptomatic macrocrania, defined by discontinuity in the curve at the time of presentation in relation with raised intracranial pressure (ICP).

NAHI was suspected based on the incoherence between the lesions and the history of trauma, and/or the child's developmental level; the absence of history of trauma; and/or lesions of different ages. Whenever a suspicion of NAHI arose, a social inquiry was performed, and the child abuse team met to decide whether the case should be brought to justice. NAHI was thus defined as a medical diagnosis leading to report the case to the legal authorities, whatever the legal outcome (which was often unknown or pending at the time of the study, and depended on other, nonmedical considerations).

We defined SSDHI as a clinically significant subdural hemorrhagic collection without a history of trauma or associated traumatic lesions, caused neither by infection nor by surgery, occurring without any local nor systemic cause, and for which not element of suspicion of child abuse was found. The SSDHI group was compared with cases of traumatic SDH in infants (TSDHI), for which a trauma was identified on the basis of clinical history, other traumatic lesions, or a context of child abuse.

We performed a review of the literature, looking for cases of SDH in infants considered spontaneous by the authors; in particular, we sought to identify risk factors for SSDHI and to analyze on which basis NAHI was eliminated.

Statistics were calculated on SPSS 14 software, using the Chi-square and Fischer's exact test for nominal variables, Student's t test for continuous variables, and Wilcoxon's z test for nonparametric tests.

Results

Our clinical material included 16 cases of SSDHI, 14 males and two females (M/F ratio=7.0), with a mean age at diagnosis of 3.5 months (1.4–8.1 months; 95% CI, 3.0–4.0). Nine of these (56.2%) had a history of obstetrical problems, 12 (75.0%) had idiopathic macrocephaly, and 7 (43.7%) had arachnoidomegaly diagnosed previously by ultrasound or computed tomography (CT) scanner performed on average 52 days (median 62, extremes 10 to 138 days) before the diagnosis of SDH. No patient in the study group had a stepparent or a history of dysfunctional family (substance abuse, psychiatric disorder, and history of violence), opposed to 14.3% and 29.5% of the TSDHI group, respectively. Constitution of the group and predisposing factors are detailed in Table 1.

All patients were hospitalized for an SDH, with a mean delay of 30.0 days after clinical onset (0.33–138 days; 95% CI, 19.6–40.3). Symptoms of raised ICP were present in the majority of cases. One patient presented with status epilepticus and was kept in intensive care unit for 9 days. One patient presented with severe dehydration related to enteritis and underwent head CT because of somnolence. All patients underwent at least one CT scanner, after a mean delay of 21.1 days after clinical onset (0.1–85 days; 95% CI, 14.9–27.3); six patients also underwent cerebral magnetic resonance imaging (MRI). All patients underwent fundoscopy, after a mean delay of 31.9 days after clinical onset

Table 1 Past medical history and predisposing factors in the study group

Case	Sex	Age at Dx (months)	Past medical history	Obstetrical problem	Head circumference (SD)[a]	Documented arachnoidomegaly	Delay since Dx of arachnoidomegaly (days)[b]	Dehydration
1	M	3.2	None		0			
2	F	2.9	Forceps delivery	1	1			
3	M	2.0	Reflux; arachnoidomegaly		0	1	18.5	
4	M	2.8	Maternal diabetes, C-section; macrocrania with arachnoidomegaly	1	3	1	23.8	
5	F	1.4	Hydramnios, maternal diabetes, C-section; macrocrania	1	3			
6	M	2.0	C-section; macrocrania	1	4	1	74.0	
7	M	5.4	Macrocrania		3			
8	M	3.8	Macrocrania		2			
9	M	2.0	Macrocrania		2			
10	M	3.1	Low birth-weight, emergency C-section; fever, lumbar puncture	1	0			1
11	M	1.5	Prematurity; macrocrania	1	3	1	10.0	
12	M	7.0	Enteritis, dehydration		2			1
13	M	3.0	C-section; reflux, macrocraina with arachnoidomegaly, acetazolamide	1	4	1	87.0	1
14	M	5.5	Vacuum extraction; enteritis dehydration	1	3			1
15	M	8.1	Macrocrania with arachnoidomegaly, acetazolamide hypotonia	1	4	1	138.6	1
16	M	3.0	Umbilical cord nuchal loop; macrocrania with arachnoidomegaly	1	2	1	62.0	

ICP intracranial pressure, *SD* standard deviation from the mean for the age and sex

[a] Head circumference represents the baseline head circumference in SD for the age and sex; idiopathic macrocrania is defined by a baseline head circumference at or above +2SD

[b] Delay between diagnosis of arachnoidomegaly and the diagnosis of subdural hematomas

(0–140 days; 95% CI, 21.3–42.4). No patient had severe RH, but two (cases 2 and 14) had mild and moderate RH, respectively, which were not considered indicative of NAHI, in accordance with our previous experience [41]. Clinical and radiological findings are summarized in Table 2.

Eleven patients underwent subdural puncture, and 11 underwent subduroperitoneal drainage, of which two required subsequent craniotomy. Only three patients did not undergo any form of drainage of the SDH. All patients were discharged home after a mean hospital stay of 11.4 days (2–37; 95% CI, 9.4–13.3). The mean duration of follow-up was 18.8 months (1.9–71.5 months; 95% CI, 14.0–23.6); the mean age at last control was 23.4 months (5.0–77.3 months; 95% CI, 18.6–28.1). Fourteen children were normal, one had hypotonia requiring physiotherapy, and one had motor delay, epilepsy, and decreased vision, which were considered sequels of pre- and per-partum difficulties. Early outcome, surgical management, and delayed outcome are detailed in Table 3.

The control group (TSDHI) was composed of 148 cases, 111 males and 37 females (M/F ratio=3.0), with a mean age of 5.5 months (0.5–21.5; 95% CI, 5.2–5.8). One hundred seven were caused by NAHI, 31 by household accidents, and eight by traffic accidents or accidents having occurred in public places. Comparison between SSDHI and TSDHI regarding medical history, clinical and radiological findings, and immediate and delayed outcome is detailed in Table 4. The distribution of baseline head circumference in the SSDHI and TSDHI groups is presented in Fig. 1.

Review of cases of supposed SSDHI published in the literature are summarized in Table 5. Overall, among 81 reported cases, only 22 had systematic fundoscopy and evaluation by the medico legal team. In many cases, arachnoidomegaly was the suspected cause, but in only 15 (including our seven cases) was it documented previous to the occurrence of the SDH.

Discussion

Analysis of biases

Although patient accrual was prospective in our series, we could not escape several biases. Our surgical selection bias

Table 2 Clinical and radiological findings in the study group

Case	Delay to Dx (days)[a]	Clinical presentation[b]	Delay to CT (days)[c]	Mixed density	Thickness SDH (mm)
1	5.6	Malaise, seizure, raised ICP	2.0	1	6
2	7.1	Seizure, raised ICP	0.1	1	5
3	24.5	Macrocrania	24.5	0	6
4	23.8	Macrocrania, vomiting	24.4	1	6
5	3.8	Macrocrania, raised ICP	3.0	0	10
6	117.0	Macrocrania, raised ICP	47.8	0	14
7	15.0	Macrocrania, raised ICP	45.1	0	12
8	2.0	Raised ICP, vomiting	2.2	1	7
9	0.3	Raised ICP	0.4	0	15
10	2.0	Hypotonia, irritability, status epilepticus	2.3	1	4
11	42.0	Macrocrania	32.4	0	5
12	1.5	Dehydration, somnolence	2.0	1	10
13	138.0	Macrocrania, raised ICP	85.5	0	9
14	2.4	Macrocrania	0.4	0	12
15	2.0	Macrocrania, anorexia, malaise, fever	2.1	0	14
16	93.0	Macrocrania, hypotonia, torticollis	63.5	1	9

[a] Delay between the clinical onset and admission to our hospital

[b] Symptomatic macrocrania, as opposed to idiopathic (cf. Table 1), represents head enlargement caused by raised intracranial pressure

[c] Delay between the clinical onset and the time of the first CT scanner. The delay to CT was shorter than the delay to diagnosis when the CT was performed in a peripheral center hospital before referral

is reflected by the high proportion of patients requiring drainage, compared with the literature (Table 5). On the other hand, some cases seen in clinics with a very mild SDH were not hospitalized, healed spontaneously, and are not accounted for in our study. We may also assume that many more SDH were so mild that these were never diagnosed. The accrual pattern in any study presupposes a threshold for patient inclusion.

Circular reasoning is another bias not obviated by our study. For example, the large proportion of patients with idiopathic macrocephaly in our series may result from the preconception that SSDHI is caused by macrocrania. Another example is the absence of familial dysfunction in the SSDHI group, compared with a sizeable proportion of cases in the TSDHI group, which may reflect prejudice from our part; but we considered that not reporting to the judiciary unresolved cases of SDH in a suspicious context would have been unethical (and unlawful under the French law). A final example of circularity bias in our study is the absence of severe RH in the SSDHI group, because all cases with severe RH were considered suspect, based on previous studies [41]. On the other hand, we chose not to eliminate cases of supposed SSDHI with mild RH, because we had found that only severe RH was indicative of NAHI.

Negative elements, like the absence of skeletal lesions and of coagulopathy, were taken in account in our analysis. We consider that the use of negative findings is legitimate only if these features are systematically and prospectively searched for. This was the case in our study for skeletal and clotting surveys, but not for systemic diseases like glutaric aciduria, which was searched for in only four cases (all negative). How extensive the biological evaluation for medical causes of SDH in infants should be is open for discussion. It should also be pointed out that fundoscopy can be falsely negative, especially when performed late [27, 31], which was the case in several of our cases, in relation with the slowly progressive clinical presentation. The same holds true for the absence of bruises, which can clear in a week or two. Overall, the negativity of finding which are known to have a short lifespan has little value for the diagnosis of SSDHI. For this reason, we think that the diagnosis of SSDHI cannot rely just on negative findings, and positive elements, like predisposing factors, are necessary for the diagnosis.

Among the clinical features, macrocephaly deserves special mention because of its ambiguity: it is either idiopathic, associated with arachnoidomegaly, in which case the diagnosis of SSDHI may be considered; or symptomatic of an SDH, associated or not with arachnoidomegaly, manifesting in a progressive fashion. For that reason, it is very important that the head growth chart is plotted carefully for each patient (corrected for gender and gestational age), in order to differentiate idiopathic from symptomatic macrocephaly [16]. In many studies reporting on SDH and arachnoidomegaly, especially retrospective

Table 3 Immediate and delayed outcome in the study group

Case	Hospital stay (days)	Surgery	Follow-up (months)	Age at last control (months)	Sequels	GOS
1	12	Drainage	10.6	14.0	None	GOS 1
2	11	Drainage	18.9	22.0	None	GOS 1
3	16	Drainage	11.7	14.5	None	GOS 1
4	5	None	5.0	8.6	None	GOS 1
5	12	Drainage	69.0	70.5	None	GOS 1
6	8	Drainage	71.5	77.3	None	GOS 1
7	15	Drainage	54.5	60.5	None	GOS 1
8	11	Drainage	13.4	17.2	None	GOS 1
9	9	Drainage	18.2	20.2	None	GOS 1
10	37	Drainage, then craniotomy	1.9	5.0	Hypotonia, epilepsy, decreased vision	GOS 3
11	2	None	3.9	6.8	None	GOS 1
12	16	Puncture	12.2	19.0	None	GOS 1
13	7	Drainage	16.3	23.8	None	GOS 1
14	2	None	16.4	21.9	None	GOS 1
15	16	Drainage	6.1	14.3	Motor delay	GOS 2
16	3	Drainage, then craniotomy	8.3	14.4	Hypotonia	GOS 1

GOS Glasgow outcome score, according to the World Federation of Neurosurgical Societies (1=normal life, 5=dead)

series, this distinction is not clear [5, 11, 35, 44], which has contributed to cloud the issue. Even in a prospective series like ours, the distinction may be difficult, when measurements are scarce or when the patient is very young.

After thorough review of our series, we are confident of the spontaneous nature of 14 of our cases; however, two cases (cases 1 and 2) are tainted with some doubt about possibly concealed NAHI. Of note is the fact that these two cases lacked of any predisposing factor like macrocrania, arachnoidomegaly, or dehydration, and that one of these (case 2) had mild RH. Both cases, however, fared well, and no new incident occurred during 11 and 19 months of follow-up, respectively. Whatsoever, we chose not to exclude these two cases from the series because we feel that doubt cannot be totally eliminated from the concept of SSDHI, as reported earlier [13], and these cases illustrate the difficulties of defining this entity.

Pathophysiology

Role of macrocrania and arachnoidomegaly

Arachnoidomegaly (a name considered more accurate than external hydrocephalus) is a common feature in infants even in the absence of macrocrania [9, 29], and is considered a characteristic of the normal infant [37]. It is more common in males [2, 4, 16, 28, 29], and the mean age at diagnosis is around 8 months [4, 5, 28]. It may be the result of a mismatch between increasing cerebrospinal fluid (CSF) output and delayed maturation of CSF absorption pathways [8, 9, 24]. This transition period coincides with the acquisition of the erect posture and the closure of the greater fontanel, which allow intracranial venous pressure to drop below atmospheric pressure [37]; negative pressure may be the stimulus that triggers the development of the arachnoid villi [20].

All series of infantile SDH have found a male predominance. However, in our recent experience with 389 head injuries in infants, although intradural lesions (SDH or subarachnoid hemorrhage) occurred more often in males (M/F ratio=2.7), lesions not involving the intradural compartment (fractures and epidural hematomas) were found almost evenly in boys and in girls (M/F ratio 1.36); the difference between intra- and extradural sex-ratios was statistically significant (p=0.003). These data suggest that head injuries are not much more frequent in boys but cause more often intradural lesions (unpublished data); we interpret these data as a greater fragility of meningeal structures in males, possibly in relation with a larger head with enlarged arachnoid spaces.

Overall, the epidemiology of arachnoidomegaly coincides with that of SDH in infants, and this coincidence has led several authors to consider a causal relationship between the two [18, 19]. The hypothesis of increased fragility of the corticodural veins caused by stretching overexpanded subarachnoid spaces has been widely admitted [18, 19, 30]; however, this theory has been challenged recently by Raul et al., who showed that the enlargement of

Table 4 Comparison between spontaneous and traumatic subdural hematomas

	Traumatic	Spontaneous	*p*
Patients			
N	148 (90.2.0%)	16 (9.8.0%)	
M/F ratio	3.0	7.0	0.37
Mean age (95% CI)	5.5 (5.18-5.87)	3.5 (3.06-4.03)	0.034
Stepparent	15 (14.3%)	0	0.36
Social/psy context	43 (29.5%)	0	0.013
Obstetrical problem	58 (39.2%)	9 (56.2%)	0.20
Baseline head circumference			
SD: median	0	2.5	
SD: mean (95% CI)	0.60 (0.51-0.68)	2.25 (1.90-2.59)	<0.001
Macrocrania[a] ≥+2 SD	30 (20.3%)	12 (75.0%)	<0.001
Clinical presentation			
Days to Dx (95% CI)	6.06 (4.8-7.3)	29.9 (19.6-40.3)	<0.001
Seizures	60 (40.5%)	3 (18.8%)	0.09
Deficit	50 (33.8%)	3 (18.8%)	0.22
Vigilance	110 (74.3%)	5 (31.3%)	0.028
Raised ICP	93 (62.8%)	11 (68.8%)	0.64
Symptomatic macrocrania[b]	44 (30.1%)	11 (68.8%)	0.002
Signs of impact	58 (39.2%)	0	Criterion
Lesions			
Skull fracture	24 (16.8%)	0	Criterion
Mixed density	100 (67.6%)	7 (43.8%)	0.057
Other fracture	25 (18.8%)	0	Criterion
Transfusion	21 (14.2%)	1 (6.25%)	NS
Life threat	6 (4.05%)	0	NS
Retinal hemorrhage	93 (64.1%)	2 (11.1%)	0.0001
Delay to fundoscopy (days)	7.6 (6.3-8.9)	31.9 (21.3-42.5)	< 0.001
Mild	21 (36.5%)	1 (6.25%)	NS
Moderate	14 (9.5%)	1 (6.25%)	NS
Severe	58 (39.2%)	0	0.0018
Immediate outcome			
Subdural puncture	111 (75.0%)	11 (68.8%)	NS
Subdural drainage	99 (66.9%)	11 (68.8%)	0.90
Days in ICU (95% CI)	2.05 (1.69-2.41)	0.56 (0-1.23)	0.036
Days in hospital (95% CI)	13.7 (13.2-14.3)	11.4 (9.4-13.3)	0.10
Delayed outcome			
Months follow-up (95% CI)	20.6 (18.8-22.4)	21.2 (15.9-26.3)	0.49
GOS 1	119 (80.4%)	14 (87.5%)	0.41
GOS 2	11 (7.43%)	1 (6.25%)	
GOS 3	7 (4.73%)	1 (6.25%)	
GOS 4	4 (2.70%)	0	
GOS 5	7 (4.73%)	0	
Developmental delay	22 (15.6%)	3 (18/8%)	NS
Motor delay	32 (22.7%)	3 (18/8%)	0.66
Deficit	7 (5.0%)	0	1
Epilepsy	26 (18.4%)	1 (6.25%)	0.31
Behavior	10 (7.1%)	0	0.60
Vision	23 (16.3%)	1 (6.25%)	0.47

NS no statistics calculated because of small numbers, *Criterion* no statistics calculated because the data are inclusion criteria

[a] Constitutional macrocrania

[b] Symptomatic macrocrania (cf. Tables 1 and 2)

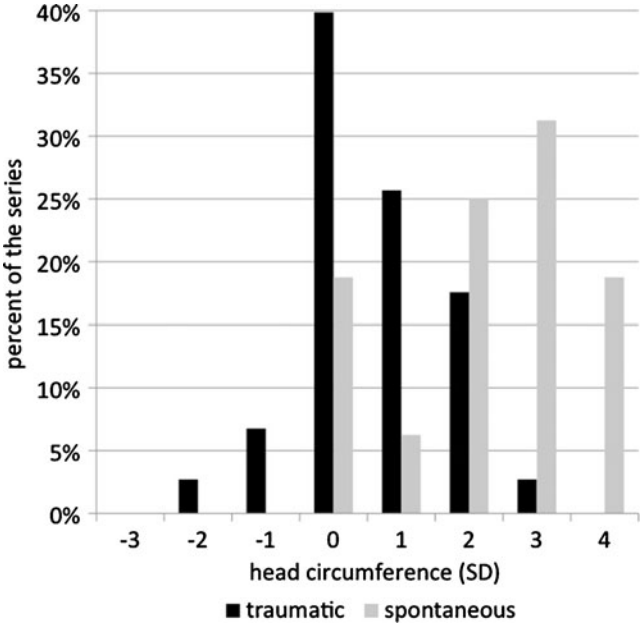

Fig. 1 Comparison of the distribution of baseline head circumference in the traumatic and spontaneous subdural hematoma (SDH) groups. Because numbers were quite different in the two groups, ordinates are expressed as percent of the total series in each group. This histogram shows that patients with spontaneous SDH in infant had often, but not always, idiopathic macrocephaly

the subarachnoid space has in fact a buffering effect [34]. Alternative sources of bleeding have also been demonstrated, like the dura mater itself [24, 39]. In addition, the relationship between idiopathic macrocrania and occurrence of an SDH has eluded previous studies [17]. Moreover, the association of arachnoidomegaly with an SDH does not indicate that one preceded the other (Fig. 2); for Ravid, "the question [is] whether the external hydrocephalus is responsible for the subdural hematoma or is secondary to it" [35]. Our data indicate a strong correlation between idiopathic macrocrania and SSDHI; however, SSDHI were also found in normocephalic infants (Fig. 1). We conclude that arachnoidomegaly, which is also found in many normocephalic infants [9, 29], only reflects a fragile hydrodynamic balance, and that SDH is a consequence of rupture of this balance rather than a complication of arachnoidomegaly. This hydrodynamic fragility exposes infants, especially males, to a higher risk of developing SDH after meningitis, craniotomy, shunting, head trauma, and even spontaneously. We concur with the opinion formulated by Hellbusch: "infants with benign extracerebral fluid collections can have subdural hematoma/hygroma complication with slight trauma or none" [16]. However disturbing this finding may be, it does not diminish the fact that the majority of SDH are caused by trauma, especially NAHI, as attested by confessions of perpetrators [6, 27, 40, 41].

Other predisposing factors

The relation between birth injury and SDH occurring later in infancy has been raised by several authors, who hypothesized that asymptomatic head injury at birth could cause chronic SDH later during infancy [12, 26, 38]. Several authors have since studied asymptomatic newborns with MRI and found traces of subdural bleeding in up to 67% of vaginal deliveries [23, 36, 43]. However, the meningeal bleeding was always in the posterior regions, unlike SDH in infants, and traces of blood disappeared in all cases after 4 weeks to 3 months. Rooks et al. reported one case of frontal SDH appearing 11 days after the initial MRI at birth had shown subdural bleeding in the posterior locations, associated with arachnoidomegaly [36]. Nonetheless, these authors considered SDH in this case to be unrelated to the neonatal bleeding and concluded "SDH after 1 month of age is unlikely to be birth-related." In our series, obstetrical problems were common, both in the SSDHI and TSDHI groups, without statistically significant difference. Birth difficulties may be related to increased fetal head circumference. On the other hand, several studies have identified prematurity and obstetrical problems as risk factors for NAHI, in relation with a defective parental bond [27, 42]. The hypothesis of a predisposition to new bleeding by scarring tissue residual of neonatal hemorrhage has thus not received confirmation from the literature nor from our study.

Other intercurrent factors should be considered when evaluating possible SSDHI, like dehydration caused by enteritis or diuretic treatment, intracranial hypotension, especially caused by lumbar puncture. In the literature, intracranial hypotension caused by lumbar puncture (LP) appears responsible for SDH only in adults or ambulating children [1, 7, 22], and as far as we know, no case has been reported in infants. This may be simply related with their posture and short stature, which limit the impact of hydrostatic pressure. In our series, the cases of SSDHI after LP or diuretic treatment were also associated with arachnoidomegaly (Fig. 3). SDH caused by severe dehydration is documented by rare cases [15, 21, 25]. It can result from enteritis, like in our cases 12 and 14, or diabetes insipidus [25]. Overall, these patients present with a severe clinical status, which is caused by dehydration rather than by intracranial hypertension, and the SDH tends to recede without surgery. A differential diagnosis in this context is cerebral thrombophlebitis [10], which can complicate severe dehydration and also cause SDH because of impaired CSF absorption.

Nosology: does SSDHI exist?

Our review of literature, summarized in Table 5, yielded few cases of SSDHI. However, we felt it does not reflect the clinical experience of many pediatric neurosurgeons, who

Table 5 Summary of cases of spontaneous subdural hematomas in infant published in the literature

Author, year	Study	Cases/series	M/F	Mean age (months)	Antecedent	Pre-existent arachnoidomegaly	Surgery	Follow-up (months)	Retinal hemorrhage	Medico legal team meeting	Note
Azaïs, 1992	Prospective	41	33/8	7	Familial macrocrania 13/41	No	2/41	19.1	Not studied	No	Arachnoidomegaly cause or consequence of SDH?
Wilms, 1993	Retrospective	8/19	?	4.5	Obstetrical problems 3/8	No	8/8	?	Not studied	No	Arachnoidomegaly cause or consequence of SDH?
Gout, 1997	Case report	2	1/1	7.0	Macrocephaly 1/2	Yes 2/2	No	12.0	Studied 1/2: present, severe	No	One case suspect of NAHI
Fung, 2001	Retrospective	2/9	1/1	13.0	?	No	2/2	?	Present 1/2	Yes	One case suspect of NAHI
Eidlitz-Markus, 2003	Case report	1	0/1	6.0	Prematurity NICU	No	Yes	18	Not studied	No	Arachnoidomegaly cause or consequence of SDH?
Ravid, 2003	Case report	3	2/?*	4.7	Macrocephaly 3/3	Yes 1/3	1/3	5.0	Absent 3/3	No	Arachnoidomegaly cause or consequence of SDH?
Amodio, 2005	Case report	1	1/0	3.5	Prematurity NICU	Yes	0	?	Absent	Yes	
McNeely, 2006	Retrospective	5	3/2	4.5	Macrocrania 3/5	Yes 2/5	2/5	?	Studied 2/5: normal	Yes	
Hellbusch, 2007	Retrospective	1/39	1/0	4.5	?	Yes	Yes	80	Not studied	No	
Rooks, 2008	Prospective	1/101	?	0.5	Meningeal bleeding at birth	Yes	No	5	Absent	Yes	Arachnoidomegaly at birth
present study	Prospective	16/164	16/2	3.5	Obstetrical problems 10/16	Yes 7/16	11/16	18.8	Present 2/16, Not severe	Yes	

Case/series: number of cases of SSDHI among the whole series

NICU neonatal intensive care unit

Fig. 2 Case 7: 5-month male with preexisting idiopathic macrocrania (+3 SD), without other past medical history, who presented with a 10-day duration syndrome of raised intracranial pressure with increased head circumference (symptomatic macrocrania with discontinuity of the growth curve). computed tomography showed a purely hypodense collection, which magnetic resonance imaging (axial view, T2-weighted) showed to be both subdural and subarachnoid. Other explorations were all negative. Subdural subtraction of 20 ml of bloody cerebrospinal fluid was followed with subduroperitoneal drainage and uneventful recovery. The coexistence of subdural hematoma (SDH) with arachnoidomegaly shows that both compartments are under pressure and suggests that preexisting hydrodynamic fragility was decompensated with the occurrence of an SDH

occasionally diagnose an isolated SDH in an infant without any context of AHI, but without element of suspicion of NAHI either. These cases are unlikely to be published in the absence of positive criteria allowing identification as a clinical entity. Cases of SSDHI reported in the literature are rare, often old, and analyzed without concern about possible NAHI [5, 11, 13, 14, 16, 35, 44]. Some case reports, however, mention fundoscopy and formal meetings by the medico legal team [3, 25, 36], and these appear bona fide cases of SSDHI. Cases of SSDHI reported in the literature were often associated with arachnoidomegaly, mild hypotonia, occipital flattening, and motor delay [5, 26], representing the "benign hydrocephalus syndrome." This background explains that the outcome is not always completely favorable, like in our case 15.

In summary, our experience and the literature review suggest that SSDHI do exist, but are quite rare, and this diagnosis should not be accepted without a thorough investigation in order to eliminate NAHI. Predisposing or precipitating factors like idiopathic macrocephaly (ideally with documented arachnoidomegaly) or severe dehydration are important elements in favor of this diagnosis. Our opinion confirms the statement made by Pittman who concluded, based on the principle of presumption of innocence, that if a SDH is associated with arachnoidomegaly but not with traumatic lesion, the diagnosis of NAHI could not be made [32].

Conclusion

The issue of SSDHI in infants has been for many years an elusive and vexing problem. For long, believing or not in the existence of this entity has been a matter of opinion rather than science. Progress on the definition of this concept was hampered by the absence of systematic

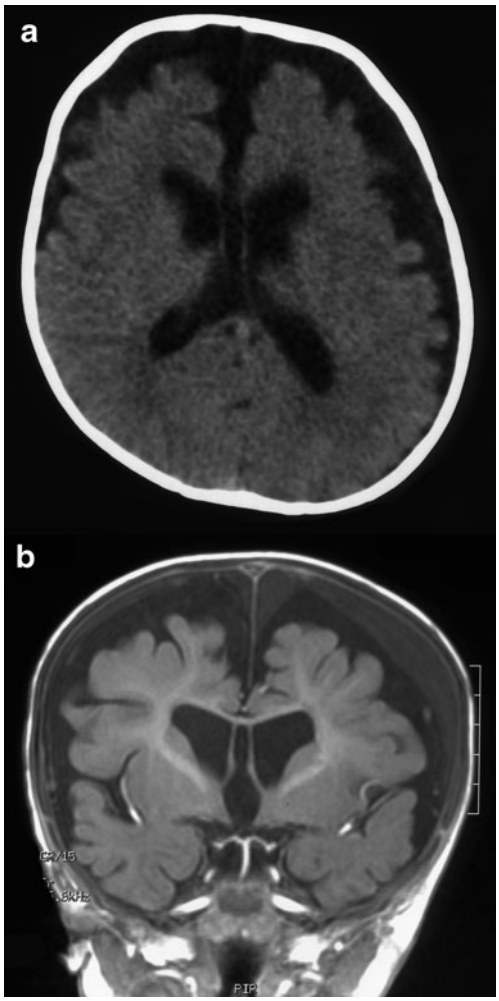

Fig. 3 Case 15: 8-month male with severe macrocrania (+4 SD) and arachnoidomegaly documented on computed tomography scan (**a**); note right-sided occipital flattening in relation with excessive mechanical constraint. The patient was treated with Acetazolamide®. He presented in emergency 4 months later for fever, malaise, and symptoms of raised intracranial pressure. Magnetic resonance imaging (frontal view, T1-weighted) showed a mixed, subarachnoid, and subdural collection (**b**). Percutaneous subtraction of 60 ml of bloody subdural cerebrospinal fluid was followed with subduroperitoneal drainage, with uneventful recovery. We think this case illustrates the filiation between arachnoidomegaly and subdural hematoma, with the possible adjuvant role of dehydration by a diuretic treatment

investigations aiming at asserting or eliminating NAHI, and ambiguity in the evaluation of macrocephaly. Our study suggests that SSDHI do exist, but even in a prospective series with systematic evaluation, some shadowy areas remain. Careful investigation of cases of SDH in order to eliminate NAHI and attention to predisposing factors like idiopathic macrocephaly/arachnoidomegaly or severe dehydration are mandatory in the quest for this rare diagnosis.

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LETTER TO THE EDITOR

Subdural hematoma in infants: can it occur spontaneously? Data from a prospective series and critical review of the literature by Vinchon et al

Horace B. Gardner

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Dear Editor,

Now that Vinchon [1] has validated Piatt's [2] observation that the diagnosis of abuse is unsafe in infants with pre-existing enlarged cerebrospinal fluid spaces, the entire abuse literature needs revision to remove all such cases from the abuse category to the non-abuse or at least the undetermined categories. Rather than comparing "spontaneous" to "abuse" cases, Vinchon apparently has sufficient clinical material to compare those cases with either rapid head growth (crossing two percentile lines) or increased space between the brain and skull (McNeely [3] used 5 mm) on presentation to those without. This may be the more informative comparison.

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LETTER TO THE EDITOR

Reply to Dr H Gardner

Matthieu Vinchon

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Dear Editor,

I appreciate the interest manifested by Dr. Gardner for our study. However, I would like to urge caution against rushing to the conclusion that all subdurals in infants with large subarachnoid spaces are spontaneous. I do not think that "the entire abuse literature needs revision", however, criticism should be exercised when reading past articles, especially regarding the basis on which cases of subdural hematomas were declared accidental or not.

Our previous paper [1], based on abusers' confessions, confirmed that violent shaking is the most common cause of subdural in infants. In our total series (unpublished data), the vast majority of subdurals in infants without evidence of trauma were diagnosed as child abuse, very often confirmed by confession and/or judicial sanction.

What we wanted to stress in the present paper is that not all subdurals are traumatic, and that clinicians should exercise much caution when evaluating an infant with subdural hematoma for possible child abuse. This evaluation must include complete physical examination by a pediatrician, early ophthalmological examination by an ophthalmologist, coagulation study, skeletal survey, and reconstruction of the head circumference curve. In the presence of positive features (bruises, severe retinal hemorrhage, or bone fractures), the diagnosis of abuse (or at least the need to refer the case to the judiciary) is clear. In the absence of all of these features, the question is still open, and many of these cases were found to be abusive trauma [2].

Since negative features are not sufficient to assert the absence of trauma, we tried to identify positive features in favor of spontaneous bleeding, like arachnoidomegaly, pre-existing macrocrania or dehydration. This effort was made in a strictly medical perspective; we leave it to the competent authorities to ponder the legal implications of these findings.

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LETTER TO THE EDITOR

Response to Vinchon et al.

John Melville · Sandeep Narang

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Dear Editor,

We read with great interest Vinchon et al's [1] report proposing spontaneous subdural hematoma in infants (SSDHI) as an important differential in the workup of suspected non-accidental head injury (NAHI)/abusive head trauma (AHT). While we appreciate Dr. Vinchon's contributions to the medical literature involving AHT, there are, unfortunately, some methodological and reporting problems that limit the clinical utility of this article.

The authors report "We collected prospectively cases of infants admitted in our hospital for apparent head injury with or without history of trauma, as reported earlier" [1 referring to 2]. However, the authors provide no clarification on how they recruited a sample which differs significantly from the authors' prior work [2]. A review of the results demonstrates that the two samples must represent different populations. For example, the proportion of non-hemophilic children with subdural hematoma (SDH) thought to have SSDHI nearly doubles in the second study (4/72 or 6% versus 16/164 or 10%). No explanation is given for the remarkable change in incidence of SSDHI between the two studies.

Additionally, the description of case selection from the author's prospective register is incomplete. If this were simply a consecutive case series, then one would expect the incidence of SSDHI to remain relatively constant across the author's multiple studies of the same population. However, as the authors' data suggest, this did not happen. Other features, such as the identification of a "control group" in the results section and the different treatment of children suspected of being abused, suggest that the groups were selected separately, and that case control analysis may have been more appropriate.

Furthermore, the evaluation of children suspected for abuse is unclear, inconsistent across the sample, and, in certain important aspects (the screening for retinal hemorrhages), inadequate. In the methods section, the authors state "*Whenever a suspicion of NAHI arose,* a social inquiry was performed, and the child abuse team met to decide whether the case should be brought to justice," and the authors performed "skeletal X-ray *in case of suspicion for NAHI."* [1, emphasis added] The authors fail to clarify the relevant criteria the child abuse team used in deciding which cases "should be brought to justice". Had there been uniform application of evaluation criteria (social inquiry and, if necessary, skeletal x-rays), it is entirely possible, and likely, that additional cases of NAHI would have been identified, or, at the minimum, additional "dysfunctionality" identified in some social histories which would have precipitated further evaluation.

The authors report that all children, appropriately, underwent fundoscopy by an ophthalmologist to look for retinal hemorrhages; however, retinal exams were delayed, with a mean of 31.9 days in the SSDHI group and significantly ($p<0.001$) longer than in the traumatic group. Sezen [3] reports that retinal hemorrhages following birth resolve quickly, often within days. He reports only one of 178 infants had a retinal hemorrhage persisting past 3 weeks. That many of the SSDHI children were examined much later than those with traumatic injury, and well beyond when retinal hemorrhages are

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known to resolve, challenges our confidence in the results of such examinations.

Finally, the authors identify circularity bias as a problem in this study, and report that due to the nature of NAHI research, some circularity is inevitable. However, the failure to address the methodological problems in this study does little to minimize such bias.

We agree with the authors that much doubt still surrounds the purported diagnosis of SSDHI. Due to the methodological and reporting limitations identified above, we respectfully submit that the authors have failed to execute the "proactive and systematic [search]" for non-accidental trauma that they initially set out to do. Consequently, while we applaud the authors' efforts to tackle this difficult diagnosis, we cannot accept the results or conclusions purported by this study.

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Reply to Dr John Melville: the scientific controversy over abusive head trauma in infants

Matthieu Vinchon

Childs Nerv Syst 2011 ; 27 ; 207-208

Dear Editor,

We greatly appreciate the interest showed by Dr. Melville for our study, and the care he took to analyze our results. We feel compelled to clarify our stances because of the perceived contradictions pointed to by Dr. Melville.

Regarding the apparently conflicting results in the different paper [1, 2], separated by 5 years, probably our phrasing in the latest “as reported earlier” was too vague, leading to ambiguity. What was meant was that the structure of the database was the same (with only new cases entered between the two studies), and the procedure to diagnose abusive or accidental trauma did not change; however the matter of the later study was a subgroup from this database, as detailed in the *materials and methods* section. Whereas the first study addressed *all types* of traumatic lesions (intradural in many cases, but also purely extradural lesions), including obstetrical traumas [1]; in the second, only *intradural* lesions were selected, comparing cases with a history ...

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LETTER TO THE EDITOR

Response to Vinchon

Charles J. Hyman · David Ayoub · Marvin Miller

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Dear Editor:
Vinchon maintains that alleged abusers' "confessions" and/or judicial decisions can confirm violent shaking as a cause of subdural hemorrhages in infants [1].

We suggest that it is time for physicians, especially medical journal reviewers, to disabuse themselves from accepting "alleged" perpetrator confessions and judicial outcomes as per se scientific confirmation of child abuse.

Certainly, some perpetrator confessions do accurately explain abusive acts causing subdural hemorrhage. However, it is common knowledge that there are "confessions" of those who maintain their innocence made to obtain a plea bargain to avoid the vagaries of a trial. In some cases, one parent will make a false admission in order to try and save his/her mate. Some, for multiple reasons, will confess to an act they did not commit. In reading and viewing interviews of alleged perpetrators, we find statements of resuscitative shaking or playful bouncing, or other playing similar to this being construed as the cause of a subdural hemorrhage [2]. This certainly should not be considered "violent" shaking". Whether shaking alone can cause subdural hemorrhage is still a zealously debated hypothesis [3, 4]. As medicine has proven through the ages, experience alone is not a substitute for the scientific method and does not prove a hypothesis [4].

We agree with Turkheimer that it is untenable to suggest that this particular scientific dispute should be decided in courts. In our adversarial system, the presentation of scientific evidence is dependent on the constrictions imposed by the court, on the legal competency of the attorney, on the knowledge and communication skills of the expert, if they can afford one, and on the educational background and the capacity of the triers of fact to evaluate science. While judges, many of whom are learned legal minds, make decisions of cases brought before them, they do not have the scholarly standing in the biomedical community to determine the scientific validity of competing medical evidence [5, 6]. Sadly, we have encountered judges who have abrogated judicial impartiality by failing to consider the possibilities of competing medical science and advocating for the omniscience of the state's child abuse experts.

The natural course of scientific evolution has resolved many medical conflicts separating scientifically valid arguments from opinion and speculation. Until that time, confessional data and judicial decision must be considered with caution and cannot be taken as per se determinants of child abuse.

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