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Histological demonstration of haemosiderin deposits in lungs and liver from victims of chronic physical child abuse

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Abstract In the context of chronic physical child abuse, two entities have been described based on macroscopical and radiological criteria: the battered baby syndrome and the shaken baby syndrome. However, in some autopsy cases, clinico-radiological information may not be available. In these cases, histological examinations are necessary to look for sequelae of repeated haemorrhages, particularly in organs likely to have suffered traumatisms such as the lungs, or in organs belonging to the mononucleated macrophage resorption system, such as the liver and the spleen. We examined a series of 15 young children who died from proven chronic child abuse and compared them with 15 sex and age-matched control subjects who died from natural causes with no history of child abuse. Using Perl's stain for iron, we identified haemosiderin deposits in pulmonary, hepatic and splenic samples and the deposits were evaluated qualitatively and quantitatively. Haemosiderin deposits were significantly ($P < 0.001$) more abundant in the lungs and liver of the chronic abuse victims than in those of the control subjects. However, they were not significantly more abundant in the spleens of child abuse victims than in controls. We conclude that haemosiderin deposits in lungs and liver could be proposed as a marker for chronic physical child abuse. This study stresses the importance of systematic histological examination to look for pulmonary and hepatic haemosiderin deposits in cases in which chronic child abuse is suspected.

Key words Child abuse · Lung · Liver · Haemosiderin deposits · Histopathology

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Introduction

The battered baby syndrome is classically described in young children who have suffered repeated abuse (and who may have died subsequently). The symptoms include chronic subdural haematomas, multiple long bone fractures of various ages, periosteal bleeding and ossification, bruises and behavioural disturbance [1–4]. More recently, another entity, the shaken baby syndrome, was described by Caffey [5] and Chiocca [6] including retinal haemorrhages and macroscopical evidence of subarachnoid haemorrhages and subdural hematomas, in the absence of external evidence of traumatism. However, post mortem findings are not always clear and there are cases of repeated physical child abuse where no macroscopical or radiological changes can be identified [7]. In such cases, conclusions are usually assessed with respect to the child's environment. Furthermore, in some cases clinical and radiological information are not available and in these cases, histology could be important to demonstrate the sequelae of relatively recent haemorrhages.

In order to test this hypothesis we looked for indirect microscopical evidence of repeated traumatisms such as haemorrhages of various ages, particularly in organs likely to have suffered traumatisms, such as the lungs, or organs belonging to the mononucleated macrophage resorption system, such as the liver and the spleen. We used a histochemical technique to identify haemosiderin resulting from resorption of haemorrhages.

Material and methods

Between 1st January 1986 and 31st December 1996, the cases of 15 Caucasian infants and young children who were victims of from homicide in the context of chronic child abuse as assessed by police investigations, post mortem and microscopical examination of visceral organs, were reviewed in the Department of Forensic Medicine of Hôpital R. Poincaré, Garches (group A). This group included 8 males and 7 females whose average age at death was 7 months, ranging from 1 month to 48 months. Epidemiological data concerning this group are summarized in Table 1. These cases were compared with 15 age and sex-matched Caucasian control subjects who died from proven natural or accidental causes, mostly sudden infant death, with no suspicion of child abuse (group B). These infants presented staturo-ponderal development within normal limits up to +1 to +3 standard deviation in most cases and had no medical history (case 3: rhinopharyngitis 1 month before death, case 11: treatment for gastrooesophagal reflux). Of these infants 13 died at the child-minder's home, which led to police investigations, always with negative results. Mostly the infants were found in ventral decubitus and the biological and radiological findings were normal except for one case of carbon monoxide intoxication (case 12, carboxyhaemoglobin 59.3%). Apart from this last case, the final diagnosis was that of sudden infant death. Post mortem examination was performed less than 24 h after death and samples were fixed in a 10% formalin solution for at least 4 days, embedded in paraffin wax, cut into 5 μm sections and stained with HES (haematoxilin-eosin-safran) for microscopical examination. Haemosiderin deposits were detected using a Prussian blue histochemical technique (Perls' stain for iron to demonstrate ferric ferrocyanate, allowing identification of ferric ions Fe+++). For the two series of stainings we used a solution of 100 ml 2% hydrochloric acid and 100 ml 2% potassium ferrocyanide.

We examined four randomly selected specimens from the lungs and one each from the liver and the spleen. Sections were examined blind by two pathologists (A.D., M.D.) and the evaluation was qualitative (±) for the spleen. Haemosiderin deposits were evaluated in the whole liver section using Deugnier's qualitative, quantitative and topographic score [8, 9]. Briefly, this score allows a topographic evaluation of the iron load in the whole liver, in the hepatocytes, in the sinusoids and in the interstitial connective tissue. It allows the differentiation of centrolobular and periportal areas. and a quantitative evaluation of the intensity and number of the deposits. In the lungs, haemosiderin deposits were evaluated according to a method derived from the Golde's score [10] adapted to lung parenchyma. This score allows both a quantitative and a qualitative evaluation which was calculated on an average of 200 cells from the 4 samples and on the 2 fields with most deposits.

Table 1 Epidemiological data and medical history in group A: case with chronic child abuse

Case	Age months	Sex	Type of abuse	Medical history	Post mortem investigation	Death circumstances
1	2	M	Battered baby syndrome	No medical history	Meningo cerebral haemorrhage, chronic leg fractures	Died in critical care unit, no obvious coagulopathy
2	9	M	Battered baby syndrome	No medical history	Meningo cerebral haemorrhage. Chronic rib fractures. Recent rib cage injury	Found dead at home
3	3.5	M	Battered baby syndrome	No medical history	Meningo cerebral haemorrhage. Chronic rib fractures	Found dead at home
4	4	F	Battered baby syndrome	No medical history	Meningo cerebral haemorrhage. Bruises of differentages. Hypotrophic child (–2 standard deviation)	Found dead at home
5	3	M	Battered baby syndrome	No medical history	Meningo cerebral haemorrhage. Bruises and wounds of different ages	Died in critical care unit, no obvious coagulopathy
6	3.5	F	Battered baby syndrome	No medical history	Meningo cerebral haemorrhage. Chronic rib fractures	Found dead at home
7	12	F	Battered baby syndrome	Admitted to hospital 1 month before death for infection	Meningo cerebral haemorrhage. Chronic rib fractures	Found dead at home
8	1	F	Battered baby syndrome	No medical history	Meningo cerebral haemorrhage and vertebral fracture. Chronic rib fractures	Found dead at home
9	2.5	F	Battered baby syndrome	Admitted to hospital 2 months before death for infection	Meningo cerebral haemorrhage. Chronic rib fractures	Found dead at home
10	6	M	Battered baby syndrome	No medical history	Meningo cerebral haemorrhage. Chronic leg fractures	Died in critical care unit, no obvious coagulopathy
11	2	M	Battered baby syndrome	No medical history	Meningo cerebral haemorrhage. Chronic leg fractures	Found dead at home
12	48	F	Battered baby syndrome	Admitted to hospital 24 months before death for infection, suspicion of battered baby syndrome	Abdominal haemorrhage. Chronic rib fractures	Found dead at home
13	4	F	Shaken baby syndrome	No medical history	Meningo cerebral haemorrhage	Found dead at home
14	4	M	Shaken baby syndrome	No medical history	Meningo cerebral haemorrhage	Died in critical car unit, no obvious coagulopathy
15	1	M	Shaken baby syndrome	Admitted to hospital 10 days beforedeath for gastro-esophageal reflux	Meningo cerebral haemorrhage	Found dead at home

* case 1–15: Police investigation in all cases revealed chronic maltreatement, later confessed by the parents

An analysis of the results was performed using a univaried statistical method, the Fischer Student test for the spleen and the *t* test comparing averages for lungs and liver which were evaluated quantitatively. The significance was set at 5%.

Results

The gross examination of the visceral organs did not disclose any significant abnormality in either group. The lungs, spleen and liver had weights within normal limits. The microscopical examination of the lungs revealed inflammatory changes in the alveolae with macrophages of variable density on routine histological staining in all cases from group A except cases 1, 5, 6 and 7, which were normal. Case 12 also had moderate focal bronchopneumonia. The liver and spleen did not show any significant structural abnormality.

In the lung specimens from group A, haemosiderin deposits were found in alveolar macrophages which were diffuse in the lung parenchyma in cases 10–15 (Figs. 1, 2) but were more focally distributed in cases 2, 3, 4, 8 and 9. In group B, only occasional foci of alveolar siderophages were observed in cases 3, 6 and 8 (Fig. 3).

In the liver of most of the subjects from group A, haemosiderin deposits were diffuse in Kupffer's cells and hepatocytes (Fig. 4). In group B, haemosiderin deposits were observed in cases 3, 5–8, 10–12 and 14. They were mostly found in Kupffer's cells and were more common in periportal areas. The values of the different scores in lungs and liver in both groups are summarized in Table 2. The statistical analysis of these scores showed that haemosiderin deposits were significantly greater in group A than in group B ($P < 0.001$) (Table 2).

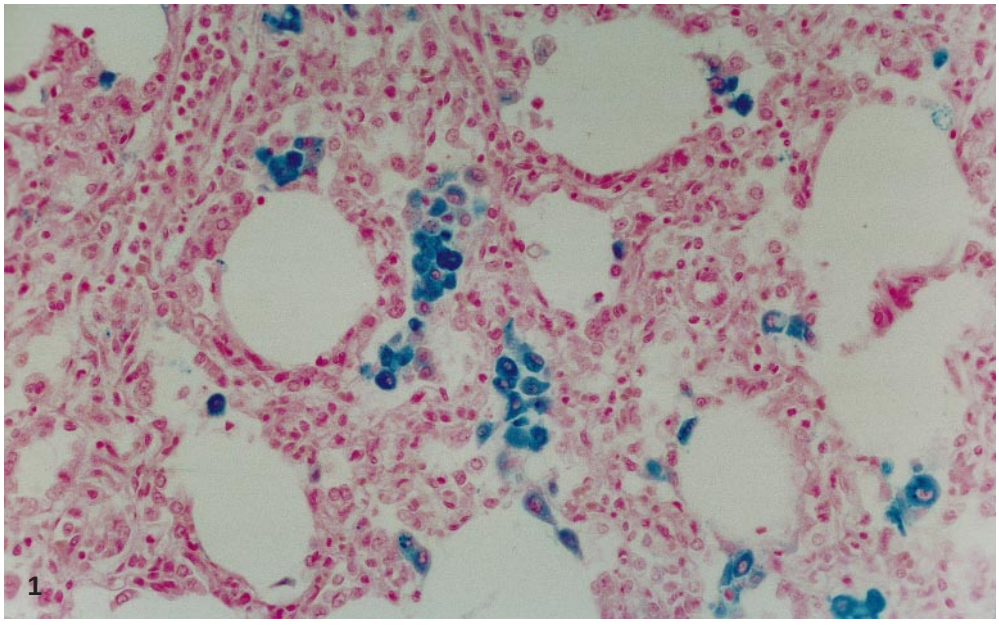

Fig. 1 Case 11 group A. In lungs diffuse and intense (Golde's score: 255) iron staining in the majority of alveolar macrophages (Perls stain for iron × 20)

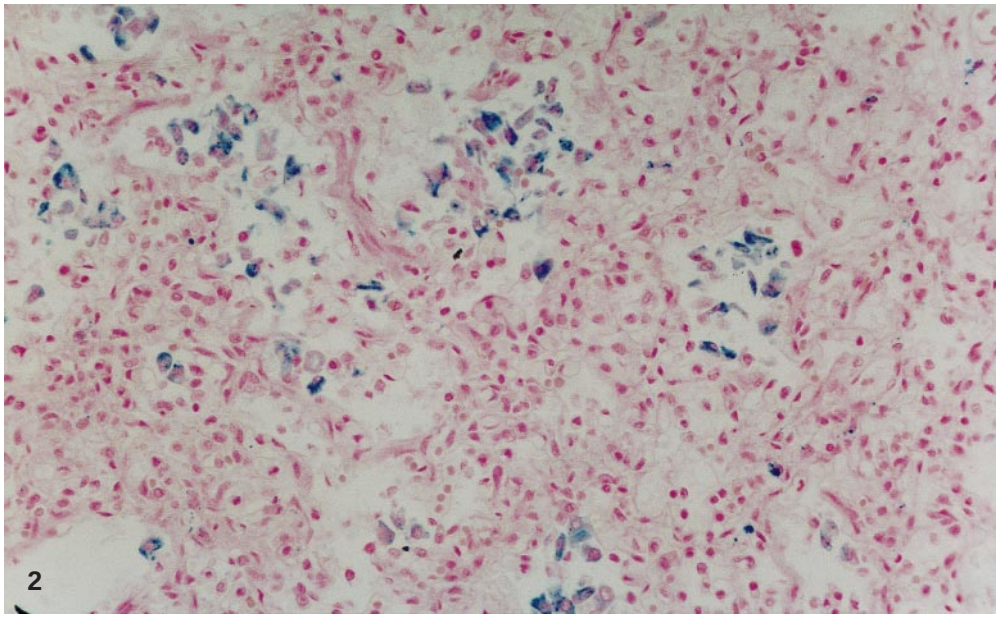

Fig. 2 Case 15 group A. In lungs marked less dense than in figure 2 (Golde's score: 66) iron pigment deposits in alveolar macrophages (Perls stain for iron × 20)

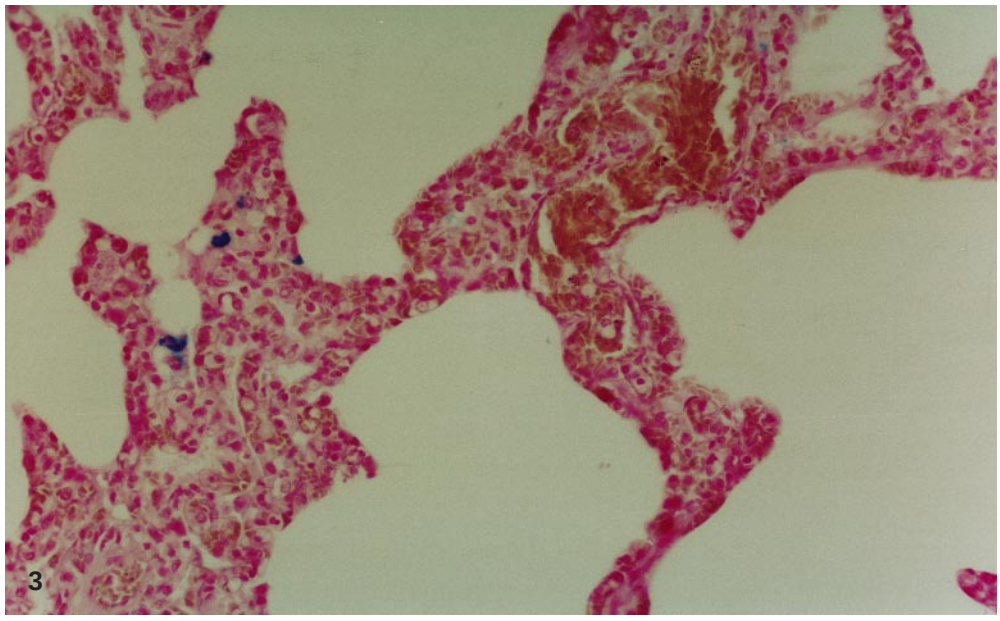

Fig. 3 Case 8 group A. In lungs mild and rare (Golde's score: 25) iron pigment deposits in alveolar macrophages (Perls stain for iron × 20)

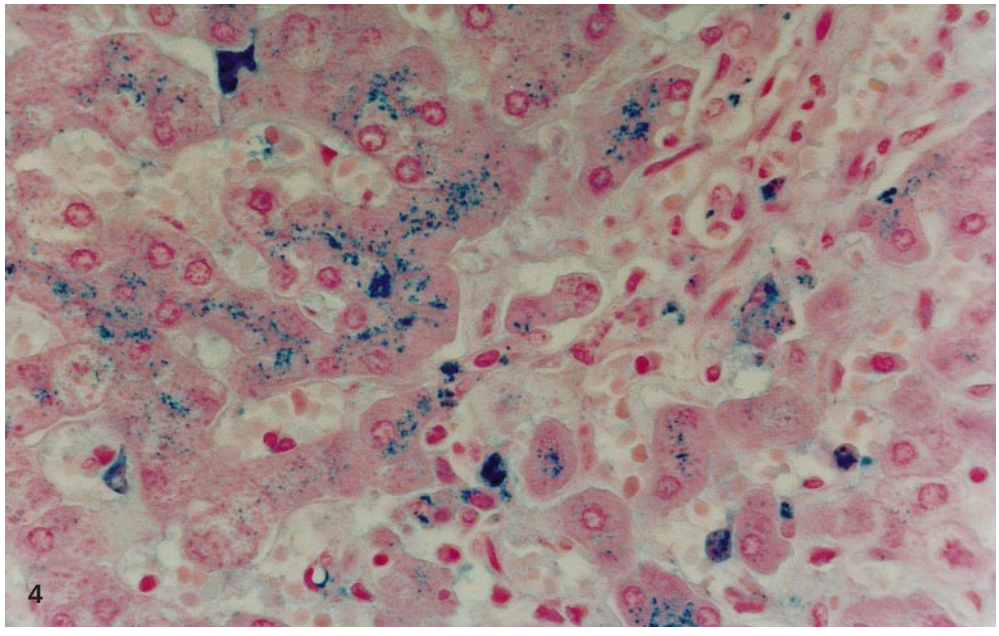

Fig. 4 Case 10 group A. Abundant iron pigment in Kupffer's cells and, to a lesser extent in hepatocytes (Perls stain for iron × 20)

Evaluation of the abundance of haemosiderin deposits in the spleen was only qualitative as these were found only in rare cases and were extremely weak. We did not find any significant discrepancy between the two groups.

Discussion

Chronic child abuse in the absence of radiological images or macroscopical lesions characteristic of the battered baby syndrome and the shaken baby syndrome has been stressed by some authors and represents a difficult problem in forensic practice [7]. In these cases, the child's environment context and police investigations are usually decisive. Although macroscopical visceral haemorrhages in lungs or liver have not been described in this context [11, 12], an isolated contusion of the sternum being reported in only one case [13], our findings suggest that haemosiderin deposits in the lungs and liver may be a feature of chronic child abuse which should be looked for microscopically in cases of suspicious death of infants and young children. This line of research concerning deposits in organs such as the liver, lungs and spleen has been less explored than deposits in skin, intracerebral and soft tissue. Indeed it has been far more usual to study haemosiderin deposits in the latter organs [14]. In cases of repeated microscopical haemorrhages, the haemoglobin contained in extravascular erythrocytes is physiologically catabolized. Consequently, there is an excess of divalent ferrous ions which can remain in the injured tissue or spread into the systemic circulation due to macrophage resorption. In both situations, these ions are oxidized into trivalent ferric ions,

Table 2 Scores of haemosiderin deposits in lungs and liver with mean values and statistical comparison from both groups. Group A: chronic child abuse, Group B: control

Case number	Golde's score		Deugnier's score	
	Group A	Group B	Group A	Group B
1	0	0	3	0
2	5	0	6	0
3	5	0.5	14	4
4	15	0	37	0
5	0	0	10	10
6	0	5	27	8
7	0	0	0	10
8	25	0.5	18	6
9	35	0	4	0
10	155	0	52	10
11	250	0	43	4
12	80	0	17	8
13	10	0	18	0
14	38	0	12	10
15	66	0	10	0
Median	15	0	14	4
Average	45.2	0.4	18.07	4.67
Range	70.482	1.285	15.337	4.386
Significance	$P < 0.001$		$P < 0.001$	

These scores summarize the quantitative and qualitative values obtained from morphometric examination of lung and liver samples

which combine with aposiderin to form haemosiderin which is a pigment forming brown refringent granulations and easily identified by the histochemical reaction with Prussian blue [15]. Therefore, the presence of haemosiderin deposits is evidence of chronic bleeding and when it is found in organs likely to have suffered violence or belonging to the macrophage resorption system, it may be suggestive of previous child abuse.

The delay between the initial bleeding and the formation of haemosiderin can be fixed only approximately. Usually, these pigments are formed rapidly within 24 h following red blood cell degradation and disappear between the second or third month after cleansing by the macrophage resorption system [16, 17]. Thus, this presence does not allow a precise conclusion on the severity and the number of traumatisms. In addition there are some pitfalls that should be avoided. In delayed post mortem examination putrefactive gas may oxidize normal Fe++ ions into Fe+++ giving rise to false positive results [18] and fixation with picric acid may cause false negative results as it dissolves Fe+++ ions [19]. In our study we used the more sensitive variation of the Prussian Blue staining method recommended by Lockemann and Püschel [20]. To date no better technique has been developed and it has the advantage of being easily used routinely with no significant variation in the early post mortem period.

It is also necessary to rule out medical treatment or pathological conditions which can cause diffuse haemosiderin deposits in visceral organs, such as iron suppletive treatment, blood transfusions, erythroblastosis, haemolysis or coagulopathies of various types. Haemosiderin in the lungs is a feature of primary idiopathic haemosiderosis. This may be suspected in patients with a history of recurrent episodes of haemoptysia and coughing and chest radiographs show bilateral reticulo-nodular images predominant around the hila. Microscopical examination shows marked fibrosis of the lung parenchyma and thickening of the arterial and arteriolar wall. Lung haemosiderosis may also be secondary to repeated blood transfusions or occur in patients with cardiopathy and severe pulmonary arterial hypertension. In these cases, unlike in ours, haemosiderin deposits were mostly found in interstitial macrophages [21–23]. In infants, repeated pulmonary physical therapy and lung infections may also cause microscopical haemorrhages in the lungs.

Haemosiderin deposits in the liver may be a feature of neonatal idiopathic haemochromatosis, and may also be found in patients who have taken iron supplement tablets. These conditions have to be ruled out by a questionnaire on the patient's medical history. Microscopical examination may disclose an angiomatosis of the liver or intrahepatic erythropoiesis, it may also confirm neonatal idiopathic hemochromatosis [24]. Hereditary autosomal recessive haemochromatosis differs from the neonatal form and usually occurs in adults. Adams et al. [25] examined 255 children from families affected by haemochromatosis and performed liver biopsies only in children with abnormal serum iron levels but did not find any iron deposits in the hepatocytes. The latter carried haemochromatosis genes of either homozygote or heterozygote forms. They concluded that investigations should not be carried out before the age of ten [25].

Some methodological points need to be stressed. This study study shows that haemosiderin deposits are not present in all cases, therefore it is important to analyse several samples from the lungs as the deposits may be irregular and minute. Indeed four cases of infants who died from proven chronic maltreatment had no detectable haemosiderin deposists in the four samples examined initially; however, in these cases it also possible that the last traumatic episodes before the one which caused their death took place so long previously that the haemoglobin catabolism was total.

The Golde score, which is largely used in pathology, is considered reliable and reproducible [26]. We also found the more recent Deugnier score that we used to evaluate the abundance of liver deposits to be reliable and reproducible. The validity of this score has been assessed by one of the authors using image analysis.

None of the children in group A had a history of pulmonary disorder, eight of them had been admitted to hospital before death (Table 1), but no abnormality of the lungs or liver had been detected in any case and routine biological investigations had given normal results. There were no clinical or pathology data suggestive of a haemoglobinopathy [27]. In two children belonging to a family from North Africa, where haemoglobinopathy may occasionally occur, no significant haemosiderin deposits were identified in the visceral organs (cases 1 and 5). We could not

rule out with certainty a history of hereditary autosomal recessive haemochromatosis in the family, however, all the children were younger than 10. In addition, in our cases, liver haemosiderin deposits were most often found both in hepatocytes and Kuppfer's cells unlike in cases of incipiens haemochromatosis where they have only been described as present in hepatocytes. Macroscopical and microscopical examination of the visceral organs in the patients of group A did not show any significant abnormality with the exception of moderate focal bronchopneumonia in case 12 which could have been responsible for microvascular abrasion. There were no changes suggestive of arterial or venous active congestion. The vessels of the bronchus, hilus of the lungs and septa did not show any significant abnormality.

After having ruled out the major differential diagnoses we concluded that the presence of haemosiderin deposits in the lungs and liver which were significantly greater in group A than in control group B were very likely the consequence of haemoglobin catabolism from old microscopical bleeding caused by trauma.

There are very few comparable studies previously reported in the literature. Stewart and Fawcett [17] found haemosiderin deposits without inflammatory changes in the lungs of 10 children from a series of 24 cases of sudden infant death. These changes were thought to be the consequence of hypoxemic episodes preceding death, the mechanism of which was not detailed. Regrettably, these authors did not describe precisely the technique used to quantify haemosiderin deposits which makes it difficult to compare results. Byard et al. [28] also found that haemosiderin deposits were more abundant in the lungs of children who died from SIDS (sudden infant death syndrome) than in controls with known non-SIDS and non-traumatic causes of death. However, they did not find any significant discrepancies between SIDS cases with a history of ALTE (apparent life threatening event) and those without such a history. Interestingly, control children with a history of thoracic traumatism had significant amounts of haemosiderin deposits in their lungs. Possible previous hypoxemic and/or traumatic episodes as the origin of haemosiderin deposits in the lung were also hypothesized by Becroft and Lockett [29] in a study of five cases and it was suggested that the deposits may represent a possible marker to differentiate true sudden infant death from asphyxic homicides in the context of repeated child abuse in "Münchhausen by proxy syndrome" [30, 31].

Our study confirms the results of these studies and stresses the importance of looking for haemosiderin deposits in the lungs of infants and young children who died under suspicious circumstances. It also suggests that, apart from asphyxic phenomena which may be responsible for micro-haemorrhages, the deposits may result from the resorption of multiple small bleedings caused by pressure, shaking, or direct shock related to chronic child abuse. The resorption of excess haemoglobin may be focal in organs which have suffered traumatisms, such as the lungs. After eliminating all classic differential diagnoses, in particular pathological conditions, siderophages demonstrate the presence of haemorrhage, strongly indicating chronic traumatic lesions. However this can never be totally certain. To our knowledge, this resorption can also occur in systemic organs belonging to the mononucleated macrophage resorption system, such as the liver but this has not been underlined previously. Failure to demonstrate increased haemosiderin deposits in the spleen of victims of chronic physical abuse compared to normal controls in our study may be explained by an increased haematopoietic turnover.

In conclusion our study demonstrates on statistically assessed morphometric grounds, that haemosiderin deposits in the lungs and liver are significantly greater in children with a proven history of chronic child abuse than in matched control subjects who died from natural causes, with no suspicion of child abuse. It stresses the interest of histological detection of haemosiderin deposits in the lungs and liver of infants and young children dying under suspicious circumstances after possible chronic child abuse. Once other pathological conditions have been ruled out, the detection of haemosiderin deposits may provide some supporting evidence of chronic violence independently of the traumatic mechanisms. It seems crucial to extend this study and systematically examine the visceral organs of children who have died from proven chronic child abuse and also of children who die under suspicious circumstances. Histochemical results should be analysed morphometrically using criteria reproducible by other pathologists such as the Golde score [10] for the lungs and the Deugnier score [8, 9] for the liver. Our study also suggests that evaluation of the number of siderophages on broncho-alveolar washings from children admitted to hospital for suspected chronic child abuse could perhaps supply additional arguments to support this diagnosis in vivo.

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