

Fetal intracranial hemorrhage: is minor maternal trauma a possible pathogenetic factor?

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ABSTRACT

Objective The occurrence of fetal intracranial hemorrhage before labor has been repeatedly observed. The aim of this study was to evaluate the sonographic appearance of fetal intracranial hemorrhage in relation to its location. Possible causative factors were also evaluated.

Design Five consecutive cases of fetal intracranial hemorrhage were identified at a single ultrasound unit between 1996 and 1999. In utero magnetic resonance imaging was also performed in four of these cases. Autopsy was performed after pregnancy termination or intrauterine fetal death (one case of each), and neurological follow-up was initiated in the three surviving infants.

Results Hydrocephaly was the predominant sonographic finding associated with intraventricular or subependymal hemorrhage; sonography provided the correct diagnosis in the former (two cases), whereas magnetic resonance imaging was necessary in the latter. Massive intraparenchymal hemorrhage was depicted as an irregular echoic mass, whereas extradural hemorrhage had a cystic appearance. History of minor maternal physical trauma without maternal or placental injury was elicited in all cases. Ultrasound examinations performed before or shortly after the trauma were available in all cases and showed normal fetal anatomy.

Conclusions The sonographic appearance of fetal intracranial hemorrhage is variable, depending on its location. Even though sonography detected an intracranial anomaly in all cases, magnetic resonance imaging was necessary to establish the hemorrhagic nature of isolated subependymal and extradural hemorrhage. The similarity of histories involving minor maternal physical trauma in all cases, together with the

absence of any known factor predisposing to fetal hemorrhage, may suggest that trauma is at least a contributing factor to the pathogenesis of fetal intracranial hemorrhage.

INTRODUCTION

Intracranial hemorrhage, and intraventricular hemorrhage in particular, is not uncommon in preterm infants; its incidence has decreased in the last years, but still ranges between 15 and 20% of the infants born before 34 weeks' gestation¹. Postnatal ischemic insults are the major cause of intraventricular hemorrhage in preterm infants, whose germinal matrix is particularly vulnerable to this type of injury. However, the pathogenesis of intraventricular hemorrhage is believed to be multifactorial, with several intravascular, vascular and extravascular factors and combinations of these playing a variable role in different patients¹. Recent studies have shown that intracranial hemorrhage is not rare even in term infants. As in preterm newborns, disorders of cerebral blood flow, vascular integrity and coagulation may cause intracranial hemorrhage, but perinatal asphyxia accounts for at least 50% of the cases, and the role of birth trauma is even more important¹.

With the widespread utilization of obstetric ultrasound, many cases of fetal intracranial hemorrhage have been detected *in utero* before the onset of labor. As many of these cases were associated with placental abruption^{2,3} or intrauterine growth restriction (IUGR)^{4–10}, the same hypoxic/asphyctic pathogenesis that is often relevant in newborns was hypothesized to be so also in fetuses. In other cases, fetal intracranial hemorrhage has been linked to fetal thrombocytopenia^{7,11} or coagulation disorders^{12–14}. Hemorrhage in a subdural location has been mainly related to

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maternal trauma, often also causing uteroplacental or other fetal injuries¹⁵. Minor maternal trauma resulting from, for example, abdominal massage has also been related to intracranial, and especially subdural, hemorrhage^{16,17}. Furthermore, fetal intracranial hemorrhage has also been described in association with a variety of maternal pathologies, such as pancreatitis¹⁸, cholecystectomy⁵, seizures¹⁹ and cytomegalovirus infection²⁰ and has also been associated with amniocentesis²¹. However, many cases remain unexplained¹⁰.

With the increased number of fetal intracranial hemorrhages reported in the literature, it has become evident that this diagnosis should be considered whenever an intracranial anomaly which cannot be explained on the basis of maldevelopment is detected. Magnetic resonance imaging (MRI) may be helpful for both differential diagnosis and estimation of the time of the bleeding^{10,22–24}. This is because of the different magnetic susceptibility of oxyhemoglobin, deoxyhemoglobin and hemosiderin, which results in different signals on various pulse sequences even though the changes over time of the MRI signal have not yet been completely elucidated in the fetus. When hemorrhage is acute the signal is usually deeply hypointense with respect to brain tissue in T2-weighted images and intermediate in T1-weighted images, subacute hemorrhage is hyperintense in both T1- and T2-weighted images, and chronic hemorrhage becomes deeply hypointense in both T1- and T2-weighted images.

With the aim of providing some clues as to the correlation between the sonographic appearance and the location of the hemorrhage, we studied five cases of fetal intracranial hemorrhage. The examination of these cases, in which no sign of hypoxia or coagulopathy was evident, also led to the incidental finding of similar maternal histories of minor physical trauma, which might be regarded as a possible pathogenetic factor.

METHODS

The present study deals with five consecutive cases of fetal intracranial hemorrhage observed at the ultrasound unit of the Division of Obstetrics and Gynecology of the University of Pisa, Italy, over the years 1996–99. In our division about 3500 obstetric sonographic examinations are performed every year. Of them, about 500 examinations per year are performed by one of the authors (F.A.L.S.) in women referred because of known risk factors for fetal structural anomalies or suspicious scan results in other ultrasound units. The scanner used during the study period (Au4, Esaote Biomedica, Genova, Italy) was equipped with color flow imaging, pulsed Doppler and convex probes (3.5–5 MHz for transabdominal examinations and 5–6.5 MHz for transvaginal examinations). Since 1996, the cases with a diagnosis of central nervous system anomalies have been referred to the MRI unit of Stella Maris Scientific Institute for fetal cranial MRI, whenever it is felt that further information may be relevant for counseling or management. Magnetic resonance imaging was performed using a 1.5-T imager (Signa Advantage, GE Medical Systems Milwaukee, WI, USA). After the sagittal scout images of the maternal abdomen, spin echo T1-

weighted (TR/TE 700/13) and single shot fast spin echo T2-weighted (TR/TE 18,000/180) multiplanar 5–7-mm thick images were obtained, with a 40 × 40 field of view and 256 × 192 matrix. The mother was starved during the 8 h before the examination and was placed in a left lateral decubitus position. No drugs were administered. Each single shot fast spin echo (SSFSE) pulse sequence required about 10–12 s.

All cases of fetal intracranial hemorrhage suspected and/or diagnosed by routine or targeted sonography were included in the study. After the diagnosis of fetal intracranial hemorrhage, blood samples were obtained in all mothers for TORCH, platelet count and coagulation parameters (fibrinogen concentration, prothrombin time, activated partial thromboplastin time, antithrombin III activity). A further ultrasound examination was performed 1 week later to check for possible rapid worsening of hydrocephaly; subsequent follow-up examinations were repeated at 7–15-day intervals. In each case the diagnosis was always confirmed by either postnatal MRI or autopsy. In continuing pregnancies, elective Cesarean section was planned after 36 weeks' gestation, depending on the ultrasound findings, to reduce the possible risk of further intracranial bleeding during labor. After neonatal and neurosurgical treatment, a long-term neurological follow-up was carried out, by means of a standardized neurological examination and Griffiths developmental scale²⁵.

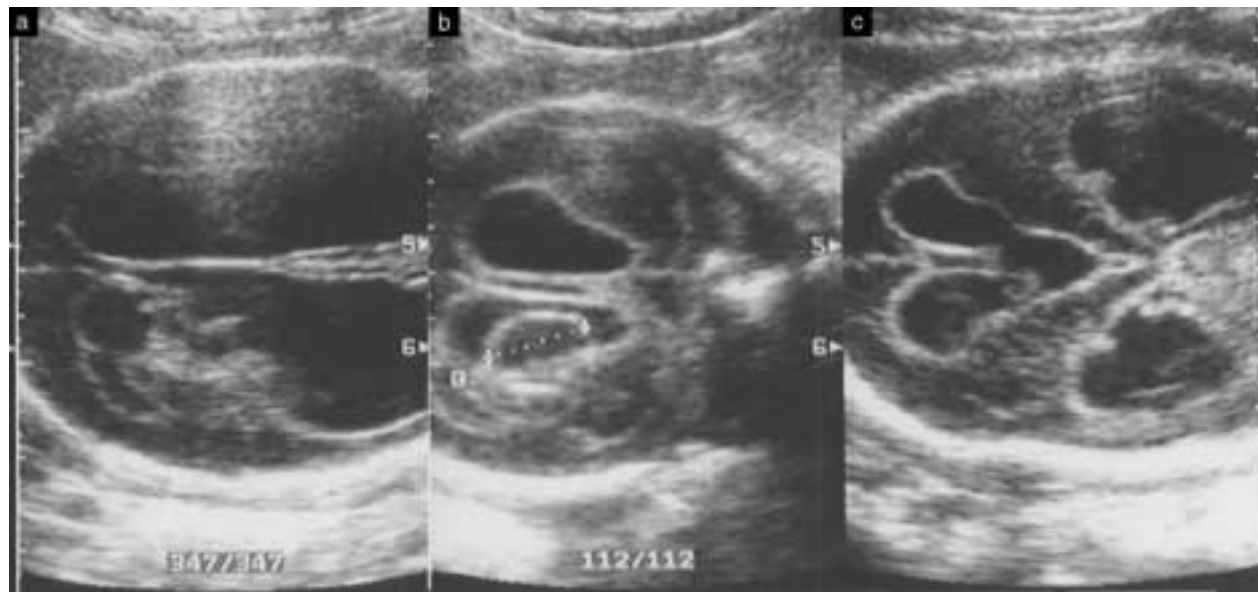
RESULTS

Apart from the five fetuses included in the study, over the same period no further cases of prenatal intracranial hemorrhage were suspected in newborns delivered in our division, nor found at autopsies following spontaneous miscarriages, terminations of pregnancy or intrauterine fetal deaths. Four of the five cases of fetal intracranial hemorrhage occurred in otherwise uncomplicated pregnancies in primiparous women aged 17–31 years; the women were not taking vasoactive or anticoagulant drugs, and denied using illicit substances. In Case 4, the 42-year-old woman, gravida 6 para 2, suffered from insulin-dependent diabetes mellitus and Gilbert's syndrome, and had repeated hypoglycemic attacks resulting in unconsciousness during the first and early second trimesters, possibly linked to hyperemesis gravidarum. She was treated at home with glucagon, which resolved the hypoglycemia and resulted in rebound hyperglycemia. She was hospitalized at 14 weeks and good metabolic control was achieved.

Hydrocephaly was the most obvious sonographic finding in three of five cases (Table 1). The diagnosis of intraventricular hemorrhage was straight forward in two of these (Cases 3 and 5), based on the detection of echogenic material within the ventricular system; in Case 3, intraparenchymal hemorrhage was diagnosed in addition to intraventricular hemorrhage (Figures 1 and 2). In contrast, in Case 1, which has been previously reported²⁴, neither transabdominal nor transvaginal sonography could detect the cause of hydrocephaly, even if signs of hemorrhage were looked for, because of the late and sudden onset of ventricular dilatation; MRI was necessary in this case to demonstrate a subependymal hemorrhage. Subsequent ultrasound examinations in Cases 1

Table 1 *In utero* imaging findings in the five cases of fetal intracranial hemorrhage

Case	Gestation (weeks)	Sonographic findings	Sonographic provisional diagnosis	Magnetic resonance imaging findings
1	34	Triventricular hydrocephaly	Hydrocephaly of unknown cause	Subependymal hemorrhage; hydrocephaly
2	28	Complex echoic mass disrupting left cerebral hemisphere	Intraparenchymal hemorrhage? Cerebral neoplasm?	Intraparenchymal or intratumoral hemorrhage; intraventricular hemorrhage
	32	Colliquated intraparenchymal mass; hydrocephaly	Massive intraparenchymal and intraventricular hemorrhage	
3	34	Triventricular hydrocephaly; echoic material within and lateral to the left anterior horn	Intraventricular hemorrhage; intraparenchymal hemorrhage	Same as sonography
4	17	Posterior anechoic cyst with echoic focus; displacement of normal structures	Astrocytoma?	Hemorrhagic cyst
5	38	Triventricular hydrocephaly; echoic material with cavitation within the ventricles	Intraventricular hemorrhage	

**Figure 1** Case 3: (a) Axial scan of the fetal head showing dilatation of the frontal horns, bodies, and atria of the lateral ventricles. An intraparenchymal hemorrhage is also depicted as a dishomogeneous lesion lateral to the left frontal horn. (b) A cavitated clot is seen impinging on the lateral side of the left frontal horn. (c) A small clot can be depicted between the left frontal horn and the third ventricle.

and 3 did not show a disproportionate increase in biparietal diameter, even though the atrial width showed a 27% increase in Case 1. The middle cerebral artery pulsatility index was within two standard deviations above the mean and it did not show any striking change over time in Cases 1 and 3; it was increased in Case 5, in which an enlarged biparietal diameter was also documented.

In the two remaining cases, ventriculomegaly was not the predominant finding (Table 1). In Case 2, first observed at 28 weeks, the left cerebral hemisphere was filled with an irregular echogenic mass, which displaced the midline towards the right side, although the tentorium appeared normal, as did the cerebellum and brainstem (Figure 3). Asymmetrical IUGR was also diagnosed, with the biparietal diameter and femur length being within the normal range for gestational age and the abdominal circumference measure-

ment corresponding to four standard deviations below the mean; the umbilical artery systolic–diastolic ratio as well as the amniotic fluid index were normal. Four days later, MRI indicated the presence of a hemorrhagic component of the mass; it also showed intraventricular hemorrhage and displacement of the ventricular system towards the right side. At 29 weeks, ultrasound showed cavitation within the previously observed mass, and dilatation of the third ventricle; it was not possible to distinguish with certainty the lateral ventricles from the intraparenchymal lesion, possibly evolving towards porencephalic cysts (Figure 3). At 32 weeks, a triventricular hydrocephalus was shown together with the previously described intraparenchymal lesion.

In Case 4, a posterior triangular-shaped anechoic mass (24 × 23 × 22 mm) was observed in the left hemisphere, adjacent to the midline and above the tentorium (Table 1). The

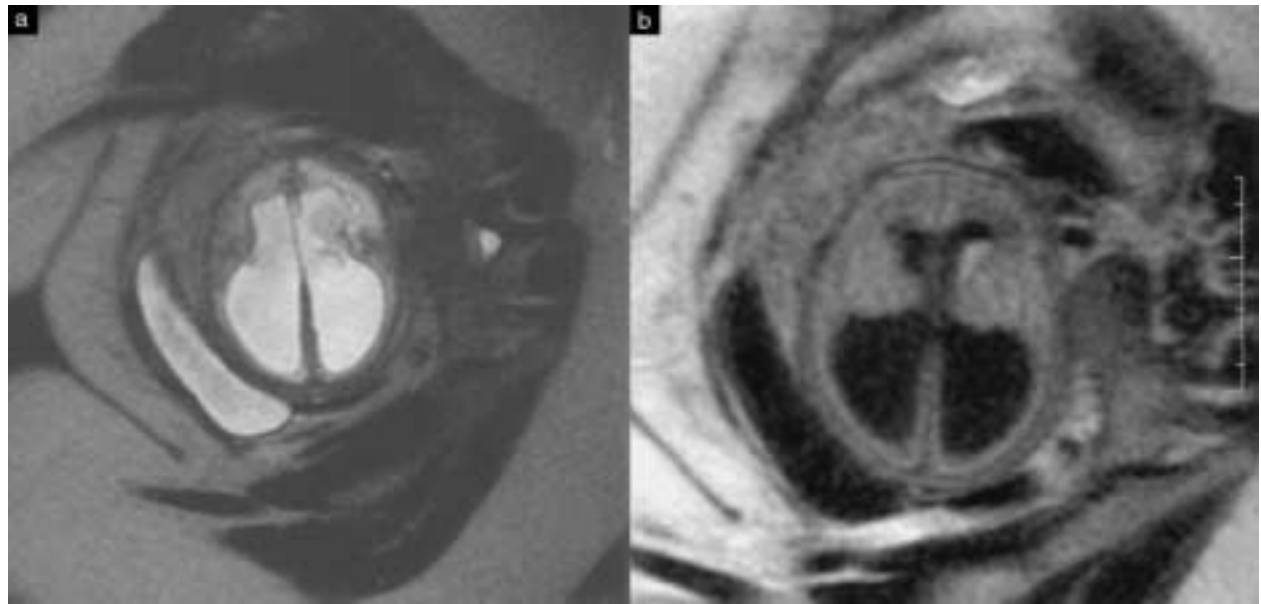


Figure 2 Case 3: Fetal brain magnetic resonance images showing subacute hemorrhage. A subependymal clot is depicted within the lateral wall of the left frontal ventricular horn, impinging on the enlarged ventricular cavity. It appears mildly hyperintense with respect to brain tissue in both T2-weighted (a) and T1-weighted (b) magnetic resonance images.

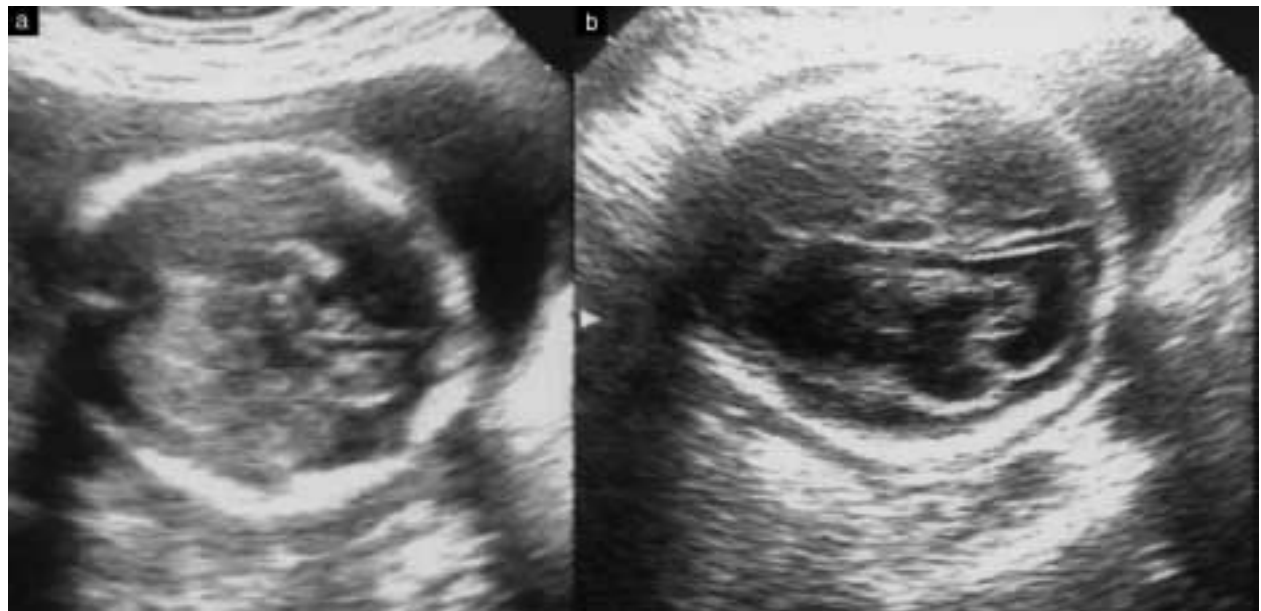


Figure 3 Case 2: (a) Axial scan of the fetal head showing an echogenic irregular mass disrupting the normal echoanatomy of the left hemisphere. The midline is displaced towards the right. (b) After 1 week, the echogenic lesion previously shown had undergone liquefaction. Dilatation of the third ventricle and left atrium is also depicted.

cyst contained an echogenic focus (6 mm) without posterior shadowing, adjacent to the occipital bone (Figure 4). The atrium of the ipsilateral ventricle was displaced anteriorly and was mildly dilated (10 mm). The pattern was dissimilar from any known cerebral malformation, and a neoformation (either hemorrhagic or tumoral) was suspected; the echogenic focus within the lesion could suggest the diagnosis of astrocytoma. Magnetic resonance imaging confirmed the presence of the cyst, but not the solid structure observed within it by

ultrasound; the cyst content appeared hematic (Figure 4). A further ultrasound examination at 19 weeks did not show any modification but an enlargement of the third ventricle (5 mm). Color-flow mapping did not show any signal within the cystic wall or the echogenic focus.

In Cases 1, 3 and 5, fetal movements were normally perceived by the mothers and appeared normal during sonographic examinations; cardiotocography showed normal fetal heart rate variability, without decelerations. The

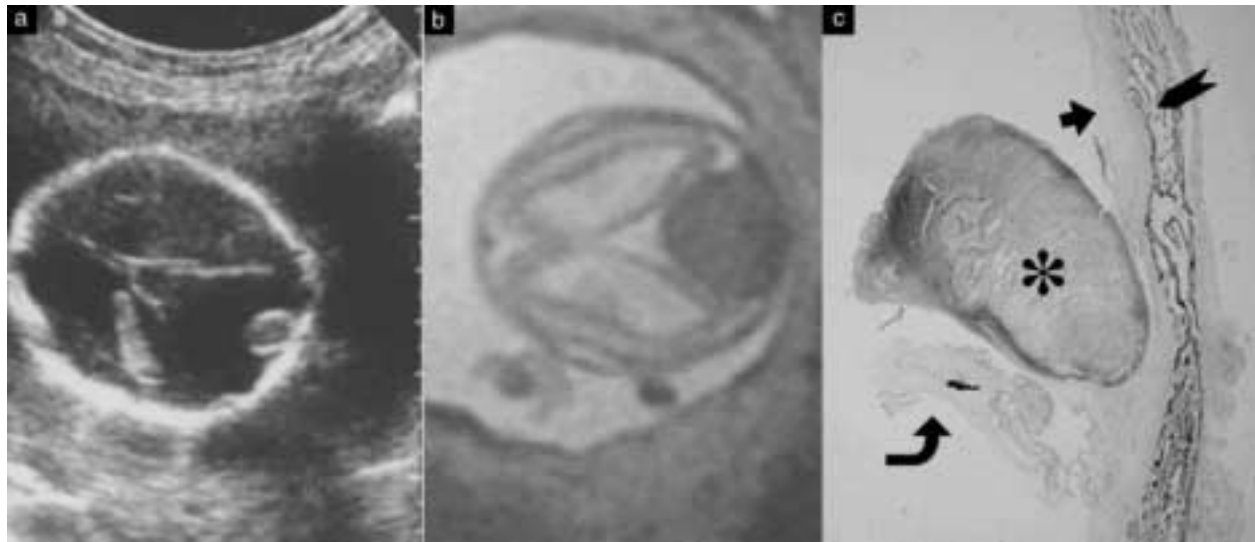


Figure 4 Case 4: (a) Sonographic image of the fetal head above the tentorium, showing a posterior cystic lesion with an echoic focus adjacent to the occipital bone. The left lateral ventricle is displaced anteriorly, with a dangling choroid plexus. (b) Axial SSFSE T2-weighted magnetic resonance image of fetal brain. The posterior extracerebral round mass impinging on both ventricular walls is deeply hypointense due to the magnetic susceptibility effect of deoxyhemoglobin. Note the hydrocephalic ventricular enlargement. (c) Histological specimen showing the fibrin clot (*) between the periosteum (straight arrow) of the occipital bone (arrow head) and leptomeninges (curved arrow). (Hematoxylin and eosin stained.)

Table 2 Pregnancy outcome and postnatal follow-up

Case	Outcome	Gestation (weeks)	Sex	Birth weight (g)	Postnatal follow-up
1	CS	38	Male	3310	Ventriculoperitoneal shunt on day 7; normal development at 1 year
2	IUFD	33	Female	1190	—
3	CS	36	Male	2790	Ventriculoperitoneal shunt on day 10; mild right hemiparesis; normal mental development at 1 year
4	TOP	19	Female	300	—
5	CS	38	Male	3310	Ventriculoperitoneal shunt at 2 months; motor retardation; hypertonia at 6 months

CS, Cesarean section; IUFD, intrauterine fetal death; TOP, termination of pregnancy.

women were hospitalized just before elective Cesarean section, which was performed between 36 and 38 weeks. All three cases required a ventriculoperitoneal shunt (Table 2).

In Case 2, no intrauterine or extrauterine therapy seemed feasible, given the magnitude of the lesion. Fetal movements, which appeared normal at 28 weeks, were markedly increased at 29 weeks, when they were judged similar to those usually observed in anencephalic fetuses; at 32 weeks they were markedly reduced, and intrauterine fetal death occurred 10 days later. Labor was then induced, and a stillborn small-for-gestational age infant was delivered. Fetal maceration prevented the detailed study of the brain, but hemorrhage was confirmed.

In Case 4, the parents elected to terminate the pregnancy, because of the large size of the cyst relative to the fetal head, the displacement of intracranial structures suggesting compression, and the initial dilatation of the ventricular system. At autopsy, a subperiosteal extradural hemorrhage was

demonstrated, with a fibrin clot attached to the internal surface of the occipital bone (Figure 4).

In no case did maternal or neonatal blood tests reveal any possible cause of fetal hemorrhage. On the other hand, detailed history revealed that all mothers had undergone accidents during pregnancy, even if none of them reported any injury (Table 3). No fetal abnormality had been detected at ultrasound examinations performed 2 weeks before the accident in Case 3, or shortly thereafter in Cases 2, 4 and 5; in Case 1, it cannot be stated whether the last normal examination was performed before or after the accident, because the latter was not reported on the mother's clinical notes as it was regarded as irrelevant. The time lag between the accident and the detection of fetal lesions ranged between 3 days and 5 weeks. Before referral, an anomaly of the fetal central nervous system had been detected in all cases in the same ultrasound unit as that in which the last normal ultrasound scan had been obtained, thus making it unlikely that a pre-existing lesion had been

Table 3 Timing of events

Case	Last normal sonogram		Accident		First abnormal sonogram	
	Week	Indication	Week	Modality	Week	Indication
1	31	Routine	Unknown (3rd trimester)	Fall from motorcycle	34	Suspected placenta previa at previous examination
2	25	Accident (within 24 h)	25	Car accident	28	Routine
3	31	Routine	33	Car accident	33	Accident (after 3 days)
4	14	Diabetes mellitus	10–14	Repeated falls during hypoglycemic attacks	17	Amniocentesis
5	33	Accident (within 24 h)	33	Fall while gardening	38	Amniotic fluid index

missed because of a poorer quality previous ultrasound examination.

DISCUSSION

The different location of the bleeding in the fetuses described above can explain the different ultrasound images obtained. In the cases of intraventricular hemorrhage, the echogenic clots within the anechoic dilated cerebral ventricles were clearly evident and typical in appearance (Cases 3 and 5). *In utero* MRI confirmed the diagnosis in Case 3, whereas it was not performed in Case 5, and obstetric management was decided on the basis of ultrasound alone. In contrast, in Case 1, periventricular hemorrhage was not recognized: the clot was adjacent to the ventricular wall, instead of impinging or floating within the fluid-filled ventricles, so that there was no clear contrast which could have allowed ultrasound depiction. Moreover, unlike in Case 3, the sonographic diagnosis was further hampered by the absence of modifications of intraparenchymal texture, as confirmed by MRI. In fact, only a few cases of isolated fetal periventricular hemorrhage have been described^{4,26}, further supporting the difficulty of the sonographic depiction of periventricular hemorrhage in comparison to intraventricular, intraparenchymal or subdural hemorrhages. In cases of unexplained hydrocephaly, *in utero* MRI is therefore warranted, to search for signs of possible etiological factors, including hemorrhage.

The echoic mass corresponding to recent massive intraparenchymal hemorrhage might have suggested cerebral neoplasia², and even MRI could not exclude a hemorrhagic neoplasm in our Case 2, because of the disruption of the cerebral anatomy. However, the evolution of the ultrasound appearance, showing cavitation and hydrocephaly, has been previously described^{2,27}, and is typical of massive hemorrhage.

The most intriguing findings were detected in the case of subperiosteal, extradural hemorrhage (Case 4). Subperiosteal hemorrhage has been reported as an autoptic finding in fetuses with concomitant intracranial hemorrhage detected in other locations by obstetric sonography; however, the subperiosteal component was missed *in utero*^{19,28}. In our case, the sonographic appearance of an echoic cyst was not suggestive of a hemorrhagic content, and the echoic focus suggested a cystic neoplasm, even if color Doppler imaging showed no blood flow. The correct diagnosis of a hemorrhagic lesion was provided by MRI, even if it did not detect

the fibrin clot, which was found at autopsy and could explain the echoic focus seen at ultrasound.

Our sonographic findings are consistent with the different appearance of intracranial hemorrhage according to their location described in newborns. *In vitro* studies were able to explain the different sonographic appearance on the basis of a different production of thromboplastin, which is maximal in intraparenchymal hemorrhage, and minimal in subdural hemorrhage²⁹. It is conceivable that cerebral thromboplastin is not present in this location, thus explaining the anechoic content we observed.

Similarly to other authors who reported on flow velocity waveforms of the middle cerebral artery in cases of fetal hydrocephaly and/or intracranial hemorrhage^{10,23,30}, we found either normal or increased values of the pulsatility index; moreover, we did not find any relevant modification over time. An increased pulsatility index was observed in Case 5, which subsequently showed the worst neurological outcome. However, other variables may have been more relevant in determining the outcome, and no general agreement has yet been reached on its relevance in newborns³¹.

In four of five cases (Cases 1, 2, 3 and 5), a history of accident was offered by the mother, and in the fifth case (Case 4) the mother was diabetic and we witnessed at least one maternal fall following a hypoglycemic attack. Fetal intracranial hemorrhage has been described following maternal accidents, even if the majority of the reported cases involve either fetal coagulopathy or conditions linked to fetal hypoxia, such as IUGR or pre-eclampsia. It has been suggested that accidents can cause maternal injury, with consequent cardiovascular shock, or placental abruption, thus causing acute fetal hypoxia¹⁵. However, in the present series, all mothers were apparently uninjured and no sign of placental abruption was detected. Interestingly, in all third-trimester cases the hemorrhagic lesion was in the fetal cerebral hemisphere that was furthest from the maternal abdominal wall, suggesting that the counterstroke, rather than the direct trauma, was the cause of the intracranial bleeding. However, we cannot be certain that the fetuses had not changed position between the time of the accident and the time of ultrasound examination.

In Case 2, the intraparenchymal hemorrhage was associated with IUGR. This association is not uncommon^{4–10}, and it is usually explained on the basis of hypoxia leading to both IUGR and intracerebral circulatory changes with secondary hemorrhage⁴. However, it might also be hypothesized that

the extensive brain damage subsequent to intraparenchymal hemorrhage could cause IUGR. This hypothesis is supported by the observation that the birth weight of anencephalic fetuses is significantly lower than that of normal infants, even after correction for the absence of the brain³²; moreover, removal of the fetal rat brain can cause a reduction of the body weight³³.

In the case of subperiosteal extradural hemorrhage, the only relevant risk factor was the repeated hypoglycemic attacks in the mother. However, bleeding is not regarded as a complication of severe hypoglycemia in the preterm newborn. Although multiple intracranial hemorrhages were described in a preterm asphyxial neonate delivered by a mother with diabetic ketoacidosis, the time of onset and cause of the bleeding are unclear³⁴; moreover, no sign of ketoacidosis was ever found in our patient, in spite of the rebound hyperglycemia. In this case the hemorrhagic site was different from that in the other cases; as gestational age was much lower, a different fetal susceptibility to the same trauma can be hypothesized. In fact, germinal matrix hemorrhage has never been reported before 22 weeks¹⁰. After that age, subependymal germinal matrix, the main site of intraventricular hemorrhage in fetuses and preterm infants, undergoes a progressive process of involution, which increases the vulnerability of its tenuous capillary network.

In conclusion, it is not possible to state with certainty that the intracranial hemorrhages observed in our cases were caused by the maternal accidents. However, the similarity of the histories is striking, involving an ultrasound examination performed before or shortly after the accident which showed normal fetal anatomy, and the subsequent demonstration of the intracranial hemorrhage. As previously discussed, several concomitant factors play a role in the pathogenesis of intracranial hemorrhage¹. Minor maternal trauma may act as a trigger in a chain of events leading to hemorrhage in a vulnerable period of fetal development. A similar hypothesis has been suggested for hemorrhage as well as other central nervous system damage in a pediatric series³⁵; the present study, based on *in utero* diagnosis, allows for certain exclusion of any causative role of periparturient events. All these cases recall many old tales of fetal death or child handicap linked to maternal accidents, which are often offered in the family history and are generally disregarded because no autopsy or imaging studies were available. On the other hand, a recall bias can be present when a fetal injury is demonstrated. The hypothesis of a traumatic cause for many fetal intracranial bleedings is disturbing, since its frequency cannot be at present evaluated, whereas minor accidents are obviously very common. The prevalence of fetal intracranial hemorrhage cannot be established based on the present data, because of the high percentage of referred high-risk pregnancies. Similarly, the sensitivity of sonography for fetal intracranial hemorrhage cannot be established since delivery or miscarriage may have occurred at other hospitals. Even if the present data are not sufficient to demonstrate an unequivocal cause-consequence relationship between maternal trauma and fetal intracranial hemorrhage, further, controlled studies are warranted, also to evaluate whether fetal damage is dependent on severity and modality of the trauma or on fetal

characteristics. It is difficult to infer when (and if) a follow-up fetal ultrasound examination should be performed after maternal accidents, considering that the time of occurrence of posthemorrhagic ventricular dilatation is variable in newborns³⁶, also depending on the amount of intraventricular bleeding. In two of our cases (Cases 2 and 5), in which the patient had an ultrasound examination within the 24 hours following the accident, no fetal anomaly was detected at that time; in the third case (Case 3), hydrocephaly was already evident 3 days later.

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