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Case Report

In utero embolization for placental chorioangioma and neonatal multifocal hemangiomatosis

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ABSTRACT

Placental chorioangioma is a limited non trophoblastic vascular tumour that may causes fetal complications as well as post-natal ones. We reported in here the first case of an in utero embolization of chorioangioma diagnosed at 22 W G with a post-natal diagnosis of neonatal multifocal hemangioma with a good outcome. The chorioangioma was embolized using GLUBRAN 2 ® (cyanolacrylate) a biologic surgical glue at 26 W G. Premature rupture of membrane occurred at 28 W G. A cesarean section at 32 W G was performed for retro placental hematoma. The neonate was 1400 g healthy girl with an anemia (hemoglobin 9.7 g/dl). After one month of life, the child met a neonatal multifocal hemangioma (skin and liver were involved) with superficial erosion of skin hemangiomas that required post-natal transfusions. We propose a literature review related to the various technics of in utero treatment of placental chorioangioma and the links with neonatal multifocal hemangiomatosis as well. The girl is now 7 year old and has a normal neurodevelopmental outcome.

Case presentation

A primigravid 26 year-old patient was referred at 26 W G for prenatal care of a placental chorioangioma. She had no specific past history and an abnormal hormonal profile for Down screening with Alpha foetal protein at 10.54 MoM (402.0UI/ml) HCG 3.55 MoM (38 ng/mL) and UE3 1.07 MoM (5.35 nmol/L within an overall risk at 1/950. Initial diagnosis was made at 22 W G. An amniocentesis was performed at 24 W G before patient was referred to our center (Normal results 46 XX) because of short femur length associated with a chorioangioma which size was 50*46*39 mm. It was vascularized by 2 pedicles, one coming from the umbilical cord located close to the chorionic plate. The higher systolic speed of the middle cerebral artery (MCA) (ie 45 cm/sec ie 1.46 MoM). At 26 W G ultrasound showed short long bones measurements with maintained growth with associated with increased amniotic fluid. The chorioangioma size was 60*50*95 mm. It was vascularized by a deep arterial pedicle with insertion of umbilical cord close to the chorioangioma (Fig.1 a and supplementary files).

Invasive procedure

At 26 + 3 WG, ultrasound showed a moderate polyhydramnios with increased MCA (ie 90 cm/sec 2.67 MoM) and a slight cutaneous oedema. The team underwent embolization of the placental chorioangioma under ultrasound control. Red blood cells were available if fetal transfusion was required. The first attempt with a 20 gauges needle enabled to take a sample of fetal blood and catheterize the feeding vessel of the chorioangioma. This first attempt was a failure as the needle cannula was plugged by the product that was used. Just after, we used with a transmaniotic approach a 18 gauges needle within a flexible vascular catheter (Microcatheter Level 10). Through this catheter, 1 ml of a biologic lipodol-glue blend (GLUBRAN 2 ®, Biologic surgery glue-Diluted 1 by 1) was injected. At the end, the chorioangioma was no more vascularized (supplementary files). The MCA speed deeply decreased to 40 cm/s (1.18 MoM). Fetal blood sampling showed an hemoglobin of 9.2 g/dL and blood platelet level of 188 G/l. At 28 + 4 WG, premature rupture of membranes occurred. Amoxicillin (1 g 3 times a day during 48 h) and antenatal corticoisteroids were given (bethametosone 12 mg

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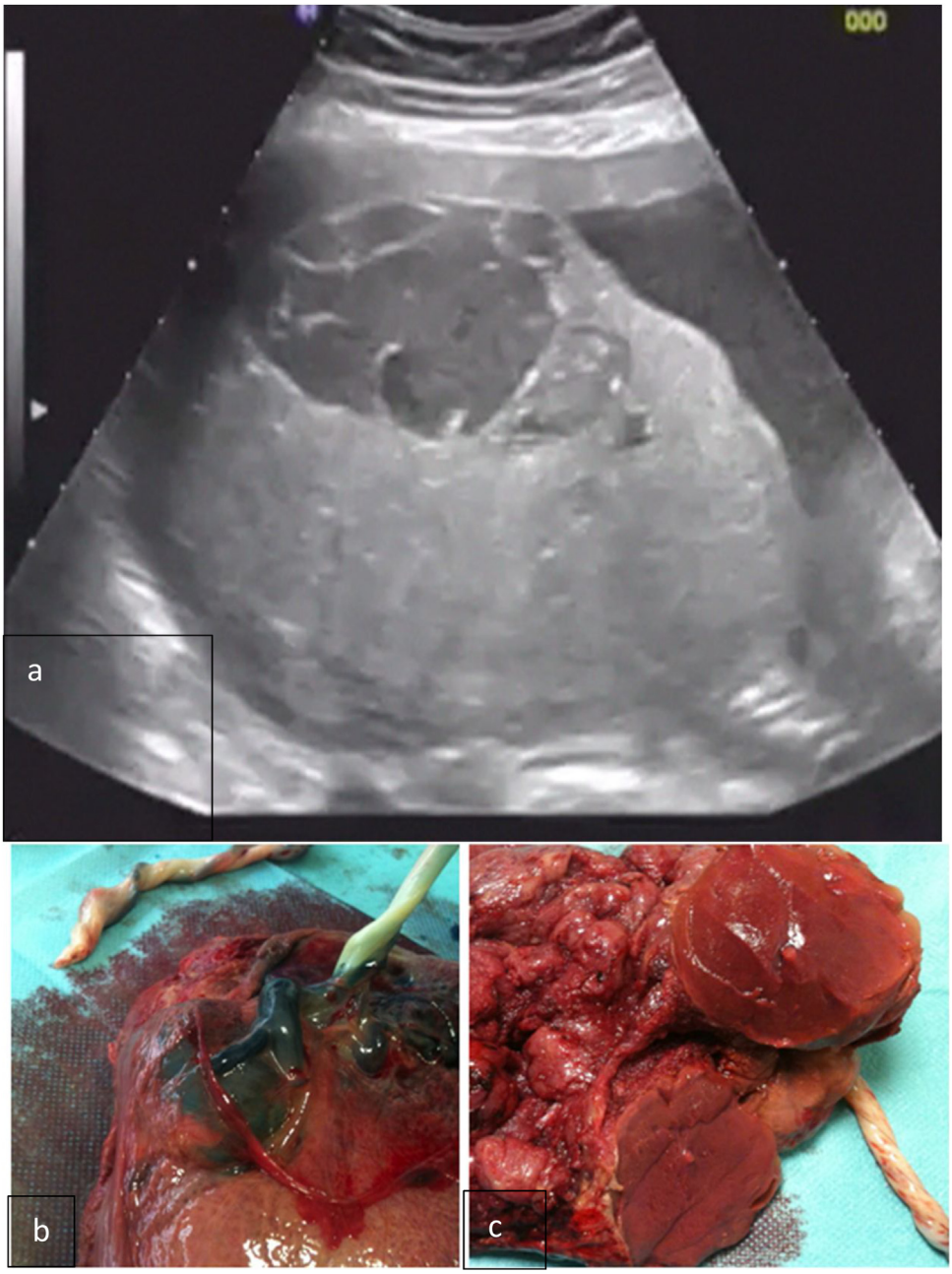

Fig. 1. a. Placental Chorioangioma at ultrasound at 26 WG; b. placenta aspect after delivery; c. chorioangioma aspect after birth.

twice). At 31 + 5 WG, an emergency cesarean section was performed for retro-placental hematoma (RPH). Magnesium sulfate was set up for neuroprotection. The baby girl weight 1400 with Apgar 8/10/10 and pH 7.33. The chorioangioma that seemed to have a bigger size than highlighted by the ultrasound control (Fig. 1b). The placental anatomy pathology found four chorioangiomas from 1 to 5 cm of big axis and partially infarcted. The baby presented a hyaline membrane disease. The neonatal sampling showed a regenerative anemia with 9.7 g/dl of red cells and 255 000/mm3 platelets.

Outcome and follow up

On the 3 day, the clinical examination found a lumbar haemangioma of 3 cm diameter, tuberous, heterogeneous colored, not pulsatile with no blowing during the medical control (Fig. 2). The size of the lumbar haemangioma that gradually increased by about 1 cm per week, starts ulcerating as from the 3rd week of life.Two other haemangiomas of $^{1}/_{2}$ cm diameter appeared at the level of vulva and the right arm as well. Many episodes of abdominal ballooning occurred without signs of necrotizing enterocolitis enteropathy. At 36 WG, the lumbar haemangioma size was 4.5 x 3 cm. The abdominal ultrasound control, pointed out hypo-echogenic intrahepatic lesions highly vascularized and compatible with hemangiomas as well (Fig. 3 and 4). The child was transfused twice with red blood cells. Two sepsis episodes occurred of urinary origin whereas the second one was of skin origin. At 38 WG (corrected age), a systemic treatment with propranolol at 2 mg/kg/day during 7 days then 3 mg/kg/day, which was well tolerated. The lumbar haemangioma size began to decrease.

Fig. 2. Lumbar haemangioma at Day 15, Lumbar haemangioma and right forearm at Day 45 before treatment by propranolol.

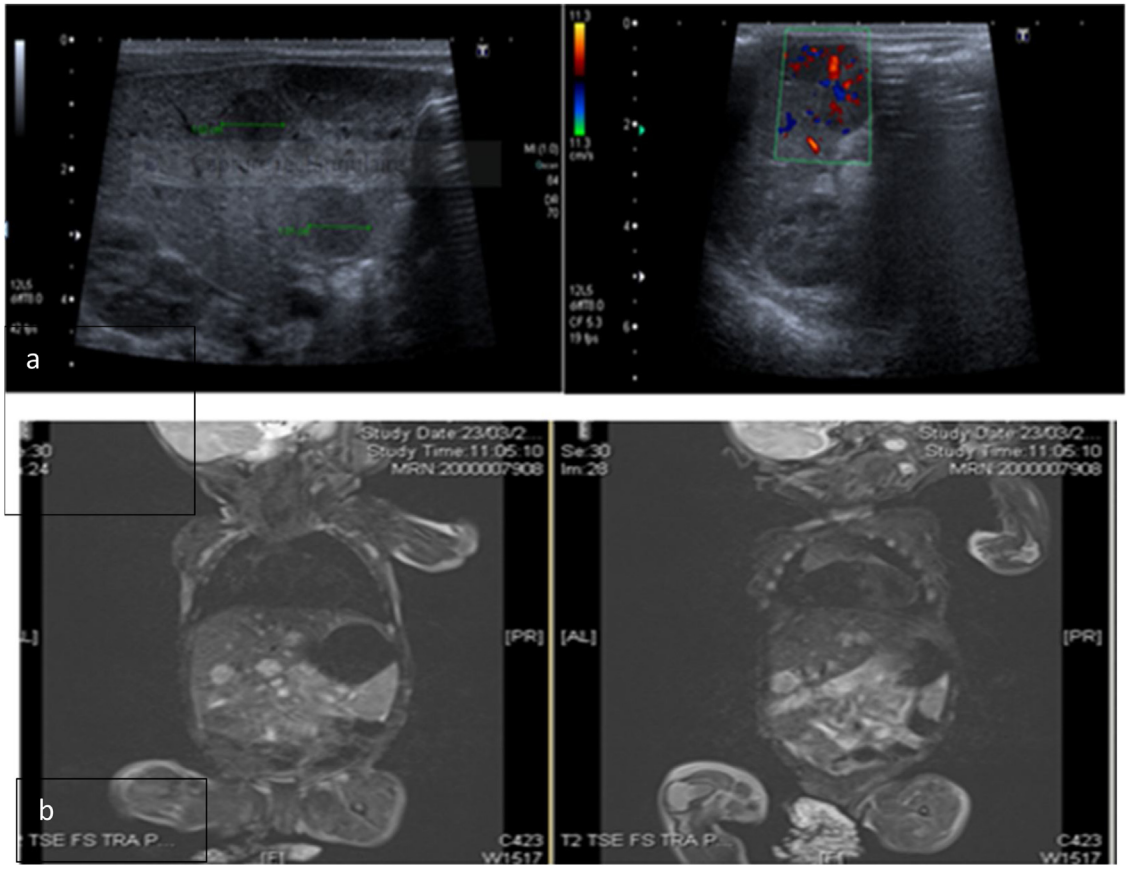

Fig. 3. a. Hepatic hemangiomas at ultrasound and b. at IRM of the whole body at 39 W G (corrected age).

After two months of life, a rectorragia episode linked to discover intestinal angiomas. At 43 W G (corrected age), after one month of treatment by β blocking, the abdominal ultrasound control points out a decrease of the angiomatous lesions sizes. The Doppler exam showed a highly vascularized lesion near the born trunk with a draining vein of big size. The lumbar hemangioma size was 5.7 x 3.7 cm. The blood check-up points out an aregenerative anemia of 7.7 g/dL that was well clinically tolerated, blood platelet count was 405G/L. The baby girl was transfused for the third time. The evolution was then favorable within a good outcome with a disparition of haemangioma. The propranolol treatment was maintained till the age of 3. The girl is now 7 year old and has a normal neuro-developmental outcome.

Discussion

Chorioangiomas (CA) are benign tumors present in roughly 1% of placentas. We reported in here the association between chorioangioma and neonatal multifocal hemangiomatosis/hemangiomas (NMH) according to Holden and Alexander [1]. In 2012, a new classification was proposed taking into account clinic, biologic and anatomopathologic criteria. Two entities were therefore identified: the “Infantile Hemangioma (IH)” or neonatal multifocal hemangiomatosis (NMH) with or without extra cutaneous impact and the “Multifocal lymphangioendotheliomatosis with thrombocytopenia” (MILT) that is the most frequent and had the worst prognosis [2]. In brief hemangioma is a cellular endothelial

Fig. 4. Lumbar Hemangioma at 43 W G (corrected age) after one month of treatment with propranolol.

proliferation begnin tumor feed by vessels. Endothelial cells express GLUT-1 which is an endothelial marker also found in placenta. GLUT-1 is absent in IH and in vascular malformations. IH and MILT should be differenciated in order to optimize the treatment and to assess the prognosis (IH = 5% of death rate against 65% in the MILT, IC 5,6-387,6, $p < 0{,}0001$) [2]. There are two new criteria in the diagnosis of NMH: absence of thrombocytopenia and presence of a convector of glucose type GLUT-1, highlighted by immunohistochemistry [3].

Although most of CA are asymptomatic, they can be associated to fetal anomalies as a polyhydramnios, hydrops fetalis, in utero growth delay, fetal anemia or fetal demise up to 30–40% [4]. Antenatal complications are, most of the time, linked with chorioangiomas size more than 4 cm as in our case where the fetus had signs of fetal anemia with high MCA and hydrops. Literature showed that antenatal care can be either symptomatic with in utero transfusion or amnioreduction or invasive with procedures such as laser therapy, embolization with microcoils or alcohol [5] (Table 1). Hosseinzadeh showed after systematic literature analysis that further studies are needed to determine the optimal antenatal care [5]. Chorioangioma embolization with biologic glue could be a less invasive way to treat the patient (Table 1). The embolization process using biologic glue was applied twice before our case and one the same year. Lau and al in 2005 used enbucrilate at 24 W G with PROM after 2 weeks followed by delivery and the death of the child after one day of life [6]. Gajewska used Glubran [7]. This case was very similar to ours, with in utero treatment at 22 W G of a chorioangioma close to the umbilical cord with a feeder vessel of large diameter. The baby was full-term born with growth retardation. This process with Glubran (cyanolacrylate) was consequently used for our patient. A third and fourth cases of embolization were reported after [8,9]. The Glubran 2 was chosen as this is biologic glue that, on contact with chemical human substances such as blood or vascular cells, fully plugs the vessels without embolizing the foetal or maternal peripheral vessels. Its main molecule is based on cyanoacrylate which is polymerized through contact with biologic liquids implying creation of a waterproof soft elastic tissue. This procedure is tricky as the needle has to be inserted exactly in the chorioangioma feeder vessel. As regards the teratogenicity of this biologic glue, there are no valid scientific data supporting the Glubran nontoxicity. Nevertheless, this treatment is extensively used by the adults without any proven unwanted effects.

Several authors tried stopping chorioangioma vascularization with other in utero methods [5] (Table 1). Quintero, for the first time, in 1996, stopped vascularization of the placental tumor by endoscopic control at 24 SA by ligaturing the feeder vessel and by making an electro-cauterization of the superficial vessels. Nevertheless, the baby died three days later [10]. Afterwards, other etiologic treatments less invasive were tested with more or less success. Sepudeva in 2009, after conducting three coagulations through chorioangioma feeder vessels endoscopy with a term birth, a premature birth at 28 W G (With a baby death after one year life) and a intra uterine demise at 29 W G [11]. Then a less invasive technic with a chemical sclerosis with high degree alcohol was tested. Among the six described cases, two were born full-term, 2 were premature and 2 were dead because of the procedure [12–16]. The technic developed by Lau and al, described in 2003 with obstruction by coils [17], was in fact less invasive, but the process had to be weekly repeated with multiple In utero transfusion (IUT). Indeed, peripheral vascularization of the chorioangioma could not have been fully blocked and the baby was born at 30 W G and died two days later. In 2003, Bhide and al [18] attempted coagulation by laser through interstitial way whilst being placed inside the chorioangioma nucleus at 24 W G. The coagulation was renewed two weeks later because of a chorioangioma revascularization with fetal anaemia with preterm birth.

IUT was extensively discussed for our patient. We decided not to perform IUT as it could increase fetal loss risk and the fetal blood volume. MCA strongly decreased after embolization. Amniocentesis was realized before referral to our center. This procedure was not indicated and increased the overall risk of complication. Moreover, in case of large sizes chorioangioma frequent ultrasounds are needed. In our case the next ultrasound was realized 1 month after initial diagnosis which is too late.

This association between placental chorioangioma and neonatal multifocal Hemangiomatosis (NMH) is rare but not harmless. Indeed, at least twelve similar cases were previously reported without any invasive antenatal procedures such as described in here (Table 2). Nevertheless, the relationship between these two disorders is not solved yet. Immunohistochemical surveys demonstrated the presence of a glucose conveyor (GLUT-1) in case of diffuse neonatal hemangiomatosis [23].

The post-natal treatment by propanolol was efficient in our case, this is in agreement with previous studies [24,25]. The propranolol, a no-cardio selective beta-blocker, would act by inhibiting the catecholamine upon the receivers' β1 and β2. These latter ones are the origin of cellular cascades inducing production of angiogenesis factors such as VGEF-A or other pro-angiogenic cytokines [26]. Duration of treatment by propranolol is between 4

Table 1
Chorioangioma antenatal treatments and neonatal outcomes. IUFD intra uterine fetal death. PROM premature rupture of membranes. PA : placental abruption.

Authors, year	Ultrasound	WG at Treatment	Treatment	Neonatal outcome
Quintero 1996 [10]	Chorioangioma 8.5 cm, anasarque, heart failure, fœtal anaemia	24	Foetoscopy : clipping of the main vessel and coagulation of the superficial vessels	IUFD
Nicolini 1999 [12]	Chorioangioma 6 cm, fœtal anaemia	27	Alcool injection	Term delivery, survival
	Chorioangioma 5 cm, fœtal anaemia	24 and 25	Alcool injection	Term delivery, survival
Jauniaux 2000 [15]	Chorioangioma 10 cm, anasarque, heart failure, anaemia	32	Alcool injection	Caesarean at 32 W G, death
Wanapirak 2002 [13]	Chorioangioma 8 cm, hydramnios, anasarque, fœtal anaemia	27	Alcool injection	PROM, delivery at 32 W G, Survival
Sepulveda, 2003 [16]	Chorioangioma 7.5 cm, anasarque, hydramnios	26	Alcool injection	IUFD
Bhide, 2003 [18]	Chorioangioma 5.3 cm, light heart failure	24 and 26	Interstitiel percutaneous laser	Preterm delivery at 32 W G due to placental failure, Survival
Lau 2003 [17]	Chorioangioma 10 cm, heart failure, fetal anemia	24 and 25	Embolization by microcoils	4 IUT, premature delivery at 29 SA
Lau 2005 [6]	Chorioangiome 9 cm, anasarque, heart failure	24	Embolization with embucrilate	PROM and premature delivery at 26 SA, Early neonatal birth
Quarello 2005 [19]	Chorioangioma de 4.4 cm, hydramnios	25	Foetoscopy : laser coagulation	Term delivery, survival
Deren 2007 [14]	Chorioangioma 8.3 cm, heart failure, hydramnios	25 and 26	Alcool injection	Preterm delivery at 28 W G, survival
Bermudez 2007 [20]	Chorioangioma 6.7 cm, hydramnios, anasarque	24	Foetoscopy : laser coagulation	PROM at 25 W G, Amniopatch, IUT, IUFD trombosis of the umbilical vein
Sepulveda 2009 [11]	Chorioangioma 6.7 cm, hydramnios, heart failure	26	Foetoscopy : laser coagulation	Term delivery, Survival
	Chorioangioma 5.8 cm, hydramnios, heart failure	27	Foetoscopy : laser coagulation	IUT, preterm birth at 28 W G, renal failure. Death at 1 year old.
	Chorioangioma 8.5 cm, anasarque, heart failure,	28	Foetoscopy : laser coagulation	IUFD
Gajewska 2010 [7]	Chorioangioma 8 cm, fetal anemia, heart failure	23	Embolization with Glubran	2 IUT, 22 W G, Growth delay in utero, Term Caesarean, Survival
Jones 2012 [21]	NR		Foetoscopy : laser coagulation	Amnioreduction
Ercan 2012 [22]	Chorioangioma, fetal anemia, hydramnios	25	Alcool injection	Amnioreduction, Transfusion in utero, Survival
Babic 2012 [8]	NR	22	Embolization with enbucrilate	2 in utero transfusions Cesarean section 30 W G
Cheng 2017 [9]	NR	22	Embolization with enbucrilate	Heart failure Cesarean section 30 W G
Hosseinzadeh 2015 [5]	Chorioangioma 6 cm, fetal anemia, hydramnios	22	Laser	Term delivery, Survival
Our case	Chorioangioma 6 cm, fetal anaemia	26	Embolization with Glubran 2 cyanolacrylate	PROM at 28 W G, Cesarean section delivery at 31 SA, Survival

Table 2
Chorioangioma Association (CA): neonatal multifocal placental/ Hemangiomatosis (NMH), IUT : In Utero Transfusion,PROM : Premature rupture of membranes, BCBA : bichorial biamniotic twin pregnancy, WG weeks of gestation; NR non reported.

	Diagnosis (WG)	Size (mm)	Antenatal period	Delivery (WG)	outcome
Kung 1997 [27]	36	70*70	polyhydramios, IUGR PROM 37 WG	37	NDH (Lever,skin) cyst lever ablation survival
	27	40*90	polyhydramios – IUGR	37	Anemia Thrombocytopenia survival
Haak 1999 [28]	30	40*68	Fetal anemia – TIU	27 + 3	NMH (Skin) ventricular hypertrophy Survival
Maymon 2003 [29]	20	50*50	Cardiomegaly - polyhydramios	Release 35	NMH (Skin) ventricular hypertrophy Survival
Witters 2003 [30]	30	70	polyhydramios	35	NMH (Skin) Regression in 3 months
Bakaris 2004 [31]	39	70*50*50	polyhydramios	39	NMH (Skin, under skin, lever)
Capelle 2008 [32]	29 + 6	58	Fetal anemia- TIU-HRP 30 + 1	30 + 1	NMH (Skin, lever) Corticotherapy Survival
Hoeger 2009 [33]	NR	80	Amniodrainage	>37	NMH (Skin) Survival
	NR	80	Amniodrainage	>37	NMH (Skin) Corticotherapy Survival
	NR	80	Amniodrainage	>37	NMH (Skin, lever) Corticotherapy Survival

and 6 months with sometimes a recurrence implying sometimes a second period of treatment.

In case of chorioangioma, the prognosis has to be discuss before delivery with search of thrombocytopenia and other lesions in utero. In case of multiple child haemangioma suspicion with thrombocytopenia prenatal care should include an extensive information of the parents.

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Conflict of interests

None.

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Appendix A. Supplementary data

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