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Letter to the Editor

Massive feto-maternal hemorrhage: Two cases

Dear Editors,

We would like to report two cases of massive feto-maternal hemorrhage that occurred in our Hospital within the last few months.

Case 1

A 28-year-old primipara patient, with a negative AB blood type, was admitted at 38 weeks gestation (WG), for decreased active fetal movements visualized in the ultrasound without any other highlighted anomaly, which justified the induction of labor by Prostaglandin (PROPESS®). The pregnancy showed normal progress. Considering the premature appearance of anomalies of the fetal heart rhythm, the type of sinusoidal rhythm, and deceleration (Fig. A1a), a caesarean section was performed as a matter of urgency green code, allowing the birth of a girl weighing 2670 g, with a positive A blood type. Apgar scores were 3, 7, and 7 within 1, 5, and 10 min of life with an anemia of 2.7 g/dL, for a standard of 16.5 g/dL for the birth. Blood arterial blood pressure at birth found a pH of 7.20 and lactates at 9.7 mmol/L. The child underwent three red blood cell transfusions. The analysis of irregular agglutinines in the mother showed negative results, and the Kleihauer test found 825 fetal red blood corpuscles/mm^3, justifying the realization of a maternal injection of 1900 µg anti-D immunoglobulin (Rhophylac®). No other causes of anemia have been found (infectious (parvovirus B19, cytomegalovirus), hemoglobinopathy, no ultrasound sign of chronic anemia). The transfontanellar ultrasound and Amplitude-integrated electroencephalography (aEEG) were normal, so there was no MRI. The clinical and psychomotor examination was normal at 2 months of life.

Case 2

A 31-year-old primipara patient with an A negative blood type, availed a biopsy of trophoblasts in the 1 st quarter of her pregnancy for a nuchal translucency of 3.6 mm, which did not reveal an abnormality. The patient consulted a 37 W G for a decrease in active fetal movements. The recording of the fetal heart rhythm indicated a sinusoidal rhythm (Fig. A1b), justifying the fetal extraction as a matter of urgency via caesarean section, allowing the birth of a girl weighing 2370 g, with a positive A blood type. Apgar scores of 1, 3, 5 were reported within 1, 5, and 10 min of life, with which the amniotic liquid was tinged. Blood arterial blood pressure at birth found a pH of 7.22 and lactates at 10.7 mmol/L. The blood balance sheet a fetal anemia of 1.7 g/dL and the Kleihauer test (48 h after childbirth) found 1000 fetal red blood corpuscles /mm^3, justifying the realization of a maternal injection of 1600 µg anti-D immunoglobulin (Rhophylac®). A brain MRI was performed in the first week of life of the child. It did not find ischemic or haemorrhagic sequelae. At one year of life, psychomotor examination and development were normal. The clinical outcomes of the mother has been favorable.

Discussion

The definition of macro transfusion, also called feto-maternal massive bleeding, varies according to the authors. Renaer et al. reported that macro transfusion was considered when the threshold exceeds 10 mL of fetal blood, the threshold from which the passage is neutralized by 100 µg of anti-D immunoglobulin [1]. Almeida *et al.* considered the threshold to be 80 mL, the rate at which a neonatal anemia is noted [2]. Finally, others, in particular, Samadi *et al.* considered a threshold of 100 mL, the threshold from which there is a fetal morbi-mortality [4]. Therefore, according to the used definition, the frequency varies from 1 to 5 for every 1000 births and its prevalence among unexplained fetal deaths is approximately 3–5 % [3].

To diagnose and quantify this bleeding, we use the Kleihauer test, which estimates the number of fetal red blood corpuscles for every 10,000 red blood corpuscles in the maternal blood. Thus, a rate of one fetal red blood corpuscle in 10,000 mature red blood corpuscles, corresponds to the passage of 0.5 mL of fetal blood in the maternal blood. Knowing that the fetal blood volume at birth is around 85–90 mL/kg, we can conclude that children in your cases lost respectively 412 mL and 500 mL of their total blood volume [1].

The main complication involves fetal anemia, which could be asymptomatic, but in the most severe cases, she can entail the neonatal death, according to the arrival of this anemia, the importance of the transfused blood volume, the term of the pregnancy, and the time of the feto-maternal bleeding diagnosis [4]. It's therefore necessary to search signs of anemia, acute or chronic (depending on its degree of installation). The first sign of appeal in yours cases is a decrease in active fetal movements. In this case, this is essential to calculate the peak velocity of systolic blood flow in the proximal middle cerebral artery (PSV-ACM) on ultrasound. If this calculation is superior than 1,5 multiples of median, it is the most reliable sign of anemia. In case of acute anemia, this will be the only ultrasound sign but if it is a chronic anemia, it will be associated to effusion of serous or hydrops. Some anomalies of the fetal heart rhythm must make suspect anemia, especially sinusoidal rhythm and pseudo sinusoidal rhythm, sometimes with decelerations. In the two cases presented, there is a misinterpretation of the fetal heart rate. The sinusoidal rhythm was either not diagnosed or diagnosed with several hours of lag. Therefore, the PSV-ACM on ultrasound was not initially realized which would have made it possible to make the diagnosis immediately and in the first case to allow a caesarean rather

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Macro-transfusion that consists of a fetal extraction involves urgency, as a neonatal resuscitation or transfusions of red blood cells may be necessary.

The main causes of fetal anemia are maternal-fetal alloimmunizations, in particular anti-RhD, but also anti-Kell and anti-c, which are detected in the first trimester of pregnancy by a positive Irregular Agglutinin Test, leading to close-up ultrasound (search for anasarca, PSV-ACM) and biological (dosage-titration) of the pregnancy. This type of chronic anemia causing anasarca. The other main cause of fetal anemia is maternal-fetal infections with Parvovirus B19 or cytomegalovirus. Infection with Parvovirus B19 causes central damage to fetal erythropoiesis, which causes deep anemia. The spontaneous evolution is towards the cure or the resumption of the erythropoiesis, if the severity of the anemia did not cause in utero fetal death. Cytomegalovirus infection may also be diagnosed by the discovery of fetal hydrops, but the fetal prognosis is related to fetal brain damage by Cytomegalovirus more than to anemia. More rarely anemia may be due to chronic maternal-fetal hemorrhage (trauma, fetal invasive action, placental pathologies such as choriocarcinoma [5] or certain fetal hemoglobinopathies such as homozygous alpha-thalassemia, congenital syphillis or pathologies of the erythrocyte membrane. Even if in 82% of the cases, no cause is found.

A sinusoïdal fetal heart rhythm is a late and insensitive sign of severe anemia and the decrease in active fetal movements also occurring at very severe stages of fetal anemia suggests that there are 2 cases of acute fetal anemia, which is confirmed by the results of blood arterial blood pressure at birth and the absence of neurological sequelae

Conclusion

Before all these data, we realized the difficulty in establishing a diagnosis of feto-maternal bleeding, both by its clinical presentation and by the specific diagnostic elements. Thus, it is necessary to diagnose it widely, in particular in case of decrease of active fetal movements, which involve the realization of an ultrasound (PSV-ACM) and known recognize a sinusoidal rhythm. It will also eliminate other causes of fetal anemia.

Annexes

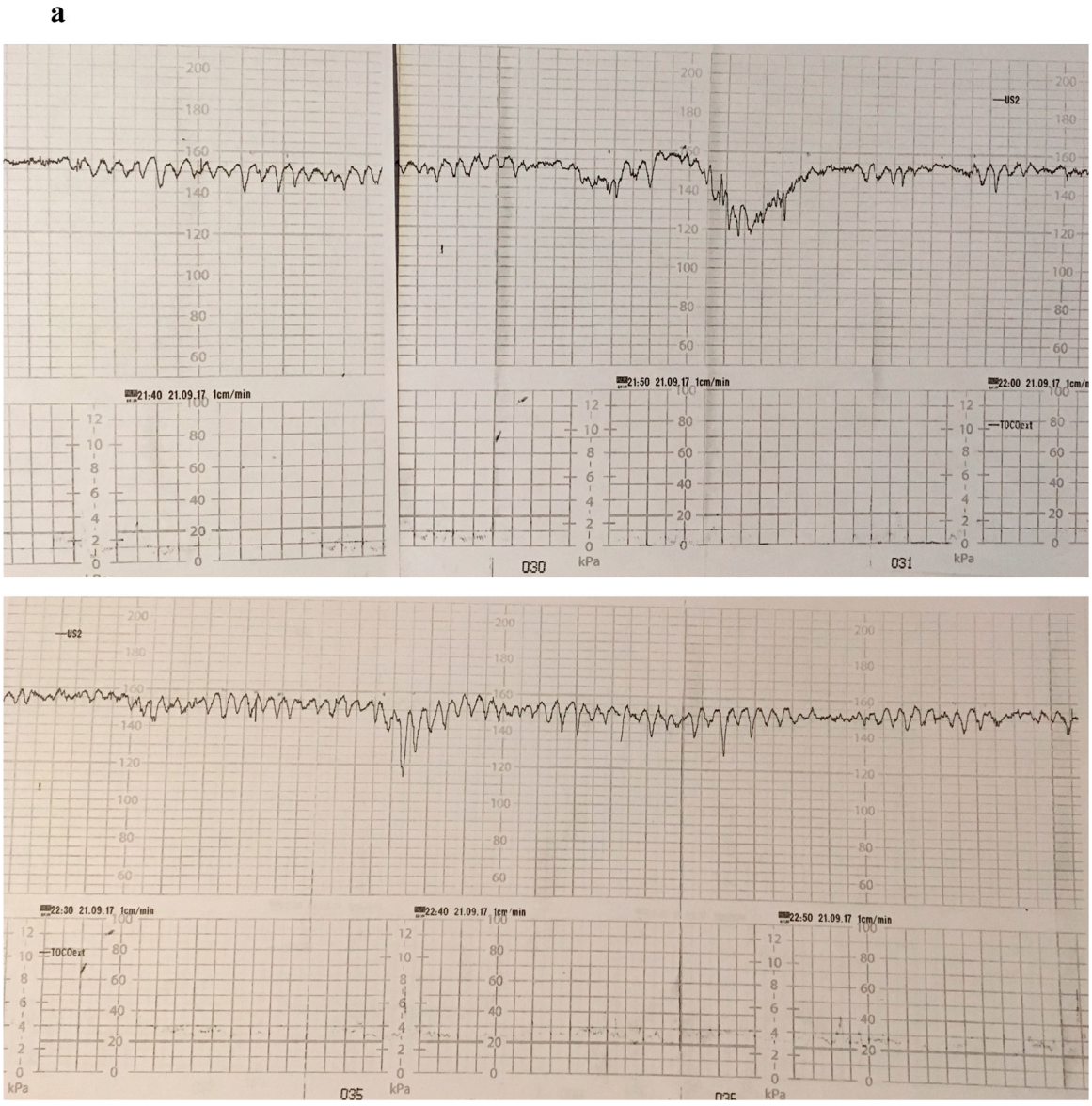

Fig. A1. a. Sinusoidal rhythm with decelerations b. Sinusoidal rhythm.

b.

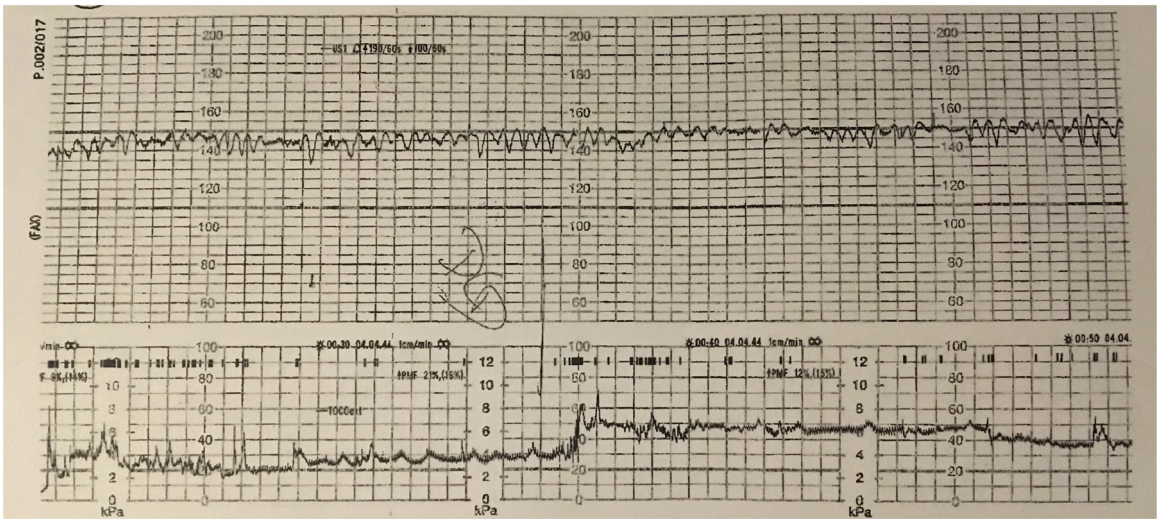

Fig. A1. (Continued)

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