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Journal of Pediatrics & Neonatal Care

Case report

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Placentary chorioangioma, perinatal management, report of two clinical cases

Summary

Introduction: Several cases of chorioangiomas have been described, however, there is little or no bibliography about the neonatal management of children born to mothers with this condition. Therefore, it is of utmost importance to have an approach to this type of pathology in order to venture a little more in the possible optimal management of these patients.

Clinical case: Two patients with high cardiac output, who were given a high cardiovascular risk profile, however, extreme measures were taken at birth, especially physiological clamping of the umbilical cord, which improved cardiac output according to the decrease in vascular resistance at birth, improving the outcome of the patients, with minimal requirements for ventilatory support and short in-hospital stays.

Conclusions: Physiologic clamping is a practice that should be made routine in patients with this prenatal history, who show combined cardiac output and elevated cardiovascular risk.

Keywords: chorioangioma, cardiovascular risk profile, late clamping

Volume 15 Issue 1 - 2025

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Received: October 22, 2024 | **Published:** January 24, 2025

Introduction

Chorioangiomas (Cas) were first described by Jhon Clarke in 1978 and are defined as a delimited placental mass composed of capillaries and surrounding stromal and trophoblast cells arising from the chorionic villi and are the most common placental tumor (0.5-1%).[1]

Risk factors include: high altitude, maternal age at risk (>35 years), hypertensive disorders of pregnancy, including preeclampsia, multiple gestation and is rarely recurrent in subsequent pregnancies.

They are divided into two groups: small chorioangiomas (less than 5 cm in diameter), which are the most frequent, and giant chorioangiomas (larger than 5 cm).[2]

Larger chorioangiomas, larger than 4 cm in size, are associated with a negative clinical impact, particularly when they grow rapidly.[3]

Neonatal mortality is between 30-40%.[4]

As shown in Figure 1, placental development is affected by variations in factors such as VEGF or PaO_2 levels, so the development of these lesions originates more frequently in areas of hypo perfusion.

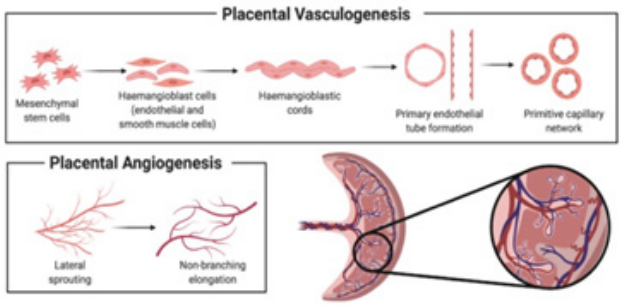

Figure 1 Normal Placental Vasculogenesis (taken from Byford, Abigail. Don't sugar coat it: the effects of gestational diabetes on the placental vasculature).

Placental changes related to hypoxia may be caused by hypoxemia secondary to decreased ambient oxygen pressure (in high altitude pregnancies), decreased oxygen binding capacity of maternal blood (maternal anemia), environmental pollution, maternal smoking, increased uterine distension (multiple pregnancy), maternal diabetes mellitus (abnormal oxygen-hemoglobin dissociation curve), absent or incomplete physiological remodeling of uterine spiral arteries (preeclampsia).[1]

In preeclampsia, placental hypoxia is probably related to focal rather than global injury depending on the number of spiral arteries with a second failed phase of trophoblast invasion. Decreased oxygen metabolism of the intervillous space due to decidual arteriopathy may lead to accelerated chorionic villus maturation and villus ischemia.[1]

The occurrence of vascular lesions together with other fetal organ hemangiomas, other fetal anomalies and syndromes such as Beckwith Wiedemann, suggest a possible genetic component.[4]

Chorioangiomas are characterized by numerous narrow, tortuous and partially thrombosed vascular networks, which can easily damage erythrocytes and trap platelets, resulting in chronic microangiopathic hemolytic anemia and severe thrombocytopenia, while large chorioangiomas may release prothrombotic factors and cause thrombosis.[1]

They are associated with arteriovenous shunts of the placenta and can be linked to multiple complications during pregnancy, including fetal anemia, thrombocytopenia, non-immune fetal hydrops, polyhydramnios, antepartum hemorrhage with placental abruption, preterm labor, IUGR and increased perinatal mortality.[1]

There are multiple theories about polyhydramnios, by pathological transudate through the abnormal capillaries of the tumor or by mechanical compression of the umbilical cord due to the tumor.[2]

High-output heart failure is the main reason for neonatal mortality, and there is a 30-40% mortality rate in large chorioangiomas. Increased blood flow through AV shunts within the tumor alters fetal hemodynamic circulation, resulting in high output heart failure and fetal death. Compression of fetal erythrocytes within the tumor vessels causes hemolysis, microangiopathic anemia, and reduced

oxygen-carrying capacity of the blood, resulting in hydrops. There is maternal-fetal hemorrhage due to leakage through the tumor capillaries causing elevated AFP levels in maternal blood and possible maternal hemolysis. Several theories have been postulated for the pathogenesis of polyhydramnios, including excess amniotic fluid transudating through the abnormal vessel walls of the tumor, or due to mechanical obstruction of the umbilical vein by the large tumor mass. Premature delivery is a common outcome. Other complications include rupture of the tumor sinusoids leading to sudden deterioration.[3]

Fetal anemia has been described in up to 15% of cases and has led to the use of serial measurements of middle cerebral artery peak systolic velocity to detect this complication. Intrauterine intravascular transfusion was the only invasive therapy used.[5]

Statiel et al, mention that, the cardiovascular profile score (CVPS) has been used to characterize cardiovascular well-being and has been related to fetal outcomes in other conditions. The objective was to test the hypothesis that elevated combined cardiac output (CCO) in fetuses with high output lesions may be associated with worsening cardiovascular status, as evidenced by a lower CVPS. Eighty percent of fetuses had a CVPS <8. CVPS is a summary score to describe fetal cardiovascular well-being. [6]

Often as the heart attempts to compensate for the increased blood flow and cardiac output, cardiovascular changes can be observed in the fetus.[6]

Among the complications, polyhydramnios can be resolved by amniodrainage. Uterine transfusions by cordocentesis resulted in an improvement of the hydropic fetuses. Suture ligation of the arterial supply of the chorioangioma by operative fetoscopy (fetoscopic devascularization) has been attempted with positive results, however, a side effect is utero-placental insufficiency.[3]

Prenatal treatment can be classified as: Supportive (i.e., intrauterine transfusion, amnioreduction or through the use of transplacental pharmacotherapy) and Definitive (i.e., surgical ligation/clipping, fetoscopic laser ablation, embolization, alcohol injection and radiofrequency ablation).[7]

Clinical case 1

Preterm newborn of 34.2 SDG by capurro, low birth weight, appropriate weight for gestational age, pulmonary adaptation syndrome, child of mother with giant placental chorioangioma, maternal age at risk, urinary tract infection, cervico-vaginitis under treatment, Ostium Secundum type interatrial communication, patent foramen ovale.[7]

Son of a 35-year-old mother, native and resident of Mexico City. Hemotype O +.

Gesta 3. Delivery 2.

Maternal-fetal medicine evaluation on October 6, 2023: Single live fetus, average fetometry 32.0 weeks of gestation, fetal heart rate of 148 beats per minute, estimated fetal weight 2147 grams, in the 16th percentile for had lock, amniotic fluid qualitatively normal. On the left lower edge of the placenta there is a hypoechogenic tumor of 9.6x8.2 cm, with regular borders, with a large caliber nutrient vessel. Normal fetal hemodynamics.

Assessed by fetal cardiology on October 06, 2023:

Situs solitus in levocardia. There is dilatation of the umbilical vein and portal system. Cardiothoracic index of 0.41, cardiac axis pointing to the left. Atrioventricular connection concordant type perforated mode. Ventriculoarterial connection concordant type, perforated mode. Pulmonary artery trunk with adequate antegrade flow. Mild tricuspid insufficiency not holosystolic. Combined cardiac output of 825mlkgmin (previous 612). Diagnosis: Heart failure with high cardiac output (825mlkgmin).[3] Cardiovascular score 9/10.

Neonatal resuscitation: Fetus is obtained via abdominal route after epidural block, clear amniotic fluid, vigorous, physiological clamping for 4 minutes, initial resuscitation steps are taken with the cord intact, due to suboptimal saturations at 5 minutes of life, it is decided to place bubble CPAP with continuous airway pressure of 6 cm H_2O and FiO_2 21%. Apgar 8/9, Silverman Anderson.[1]

Somatometry: Weight 2170 (P 55.3), Height 43 cm (P 19.34), PC 31.5cm (P 62.8), PB 9 cm, PT 29.5 cm, PA 26cm, SS 25 cm, Foot 7.5 cm, IP 2.7 (P 50-75).

Physical examination: Normal.

Postnatal assessments:

Pediatric Cardiology 10.10.23: Echocardiogram with preserved systemic cardiac output with adequate biventricular systolic function. For the moment she does not require aminergic treatment, only monitoring. If adequate pulmonary vascular resistance is not reduced, there is a risk of pulmonary hypertension.

Clinical course:

Ventialtorily with bubble CPAP for 3 days, with maximum FiO_2 requirement of 30% and continuous airway pressure of 5 cm H_2O, without requiring initiation of any other noninvasive ventilation device after withdrawal.

Hemodynamically without need of aminergic support, with fluid intake according to days of life. Echocardiogram report with normal CO.

Gastronutrition was started on the enteral route at 12 hours of life, without eventualities, tolerating the enteral route adequately, with no data of food intolerance.

Metabolic with no reports of hypoglycemia suggestive of a genetic syndrome.

Neurological: The first tranfontanelar ultrasound was reported with decreased resistance index suggestive of cerebral vasodilation.

Labs 11.10.23 (12 hours after birth): Hb 13.8, Hto 41.6, Plaq 192 000, Leuc 9,800, Neu 4,500, Lymph 600, retis 5.3%, BT 8.2. Direct Coombs negative, ORh+.

Clinical case 2

Early term newborn 37.2 SDG by LMP, adequate birth weight, high weight for gestational age, excess body mass, transient tachypnea of newborn, mild bilateral ventriculomegaly, bilateral pyelocaliceal bilatation, high output heart failure secondary to placental chorioangioma, patent ductus arteriosus, patent foramen ovale, mild pulmonary hypertension.

Son of a 32-year-old mother, native and resident of Mexico City. Hemotype A +.

Gestation 3, cesarean 2.

Maternal fetal medicine 22.08.23: Large fetus for gestational age, bilateral mild ventriculomegaly, right pyelocaliceal dilatation,

Citation: Corrales YG, Chang EAJ. Placentary chorioangioma, perinatal management, report of two clinical cases. *J Pediatr Neonatal Care*. 2025;15(1):31–34. DOI: 10.15406/jpnc.2025.15.00578

high cardiac output failure secondary to chorioangioma, severe polyhydramnios. Currently the fetus is found alive, pelvic, left dorsum, FHR 152, amniotic fluid qualitatively increased, with ILA 50cm, upon evaluation of the placenta a mass is identified in the lower placental border of 5.6 x 4.7cm, with increased vascularity, cord insertion is observed next to it and a vessel coming out of the cord into the mass.[8]

Genetics 01.09.23: pregnancy of 33.6 SDG by FUM. Adequate prenatal control extrainper with first trimester combined screening at low risk for chromosomal alteration. Structural USG was not performed and she was referred for USG in August due to the above mentioned findings. The findings and the possibility of the fetus having an overgrowth syndrome such as Beckwith-Wiedemann syndrome were explained. Due to the gestational age, assessment at birth is indicated.[9]

Fetal cardiology 22.09.23: High output heart failure secondary to placental chorioangioma, Cardiovascular Score 7/10. Late clamping is suggested.

Neonatal resuscitation: Abdominal approach was obtained, amniotic fluid was clear, placenta with chorioangioma, female fetus was obtained, vigorous, physiological umbilical cord clamping was performed, initial resuscitation steps were taken, with irregular respiratory effort, so CPAP was started with T-piece with continuous airway pressure of 8cm H_2O, without adequate recovery, so orotracheal intubation was performed.

Somatometry: Weight 3590 Kg (P 96.9), Height 50 cm (P 94.9), PC 39 cm (P >90), PB 11 cm, PT 34 cm, PA 35 cm, SS 29 cm, Foot 7.5 cm. IP 2.8 (P 75-90).

Physical examination: Hypertelorism, low implantation of the pinnae, bilateral dimpling of the pinnae, retrognathia, overlapping of the toes.

Postnatal assessments:

Evaluation by pediatric cardiology: History of placental chorioangioma with heart failure with high cardiac output / patent ductus arteriosus / patent foramen ovale / preserved systemic cardiac output / adequate biventricular systolic function with borderline cardiac output at 158 ml/kg/min, with patent ductus arteriosus with bidirectional shunt which is expected.

Ventilatory: She is maintained on conventional volume controlled synchronized ventilation, cycled by time with adequate evolution, programmed extubation is performed at 24 hours of life, she is transferred to high flow nasal prongs with flow at 5 lts/min until October 11, 2023.

Hemodynamic: Dobutamine was started at 10 mcg/kg/min for 24 hours with subsequent re-evaluation of the CO with an increase to >200 ml/kg/min, for which reason it was suspended 24 hours after starting, later without any eventuality, basal fluid intake was decided without presenting hemodynamic repercussions.

Gastro nutrition: With initiation of enteral route at 18 hours of life, with adequate tolerance, without presenting later gastrointestinal repercussions.

Metabolic: Due to prenatal suspicion of Beckwith Wiedemann syndrome, capillary glucometry controls were performed, which were within normal ranges for the hours of life, so this suspicion was ruled out (in addition to not having any other characteristic of this syndrome).

Neurological: Without presence of abnormal body movements during her evolution, transfontanellar ultrasounds were reported with presence of mild bilateral ventriculomegaly, with progressive decrease of area in subsequent control ultrasounds.

Labs 23.10.23 (12 hours after birth): Hb 10 Hto 28.7% Leuc 7,800 Neu 1,600 Lymph 4,400 Plaq 460,000 Retis 1.6%. BT 0.5. Gpo B Rh +, negative direct coombs.

Discussion

In recent years there has been increased interest in chorioangioma due to increased opportunities for prenatal diagnosis resulting from advances in echocardiography and Doppler imaging.

Many of the chorioangiomas had demonstrated adverse perinatal outcomes, however, in these two case reports, we evaluated the evolution of the patient postnatally in which we observed how the physiological clamping of the umbilical cord allowed a gradual decrease in cardiac output without abruptly modifying the cardiac output when performing early umbilical cord clamping, thus generating greater hemodynamic changes in our patients. It is worth mentioning that our patients did not present previous fetal pathology (hydrops, polyhydramnios requiring amniodrainage, fetal anemia, etc). This is why prenatal diagnosis is of utmost importance, and the fact of routinely performing physiological umbilical cord clamping in cases in which we do not know of this pathology prenatally.

Conclusions

To favor an adequate transition period in which there are no abrupt changes in cardiac output, we suggest physiological umbilical cord clamping.

Acknowledgments

None.

Funding

None.

Conflicts of interest

The authors declare no conflicts of interest.

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