

ASSESSMENT OF CHRONIC FETAL HYPOXAEMIA

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ABSTRACT

Chronic fetal hypoxaemia (CFH) can occur in many clinical conditions and can have maternal, fetal or placental origin. The last, due to placental obliterative vasculopathy, is the more frequent. In this condition nutrient supply to the fetus is reduced and later oxygen supply can be also affected. In fact CFH is encountered in 30-35 % of IUGR fetuses. When progressing, the oxygen reduction can lead to hypoxia (cellular and tissue damage). In order to prevent the permanent damage a timely recognition of CFH is crucial. The methods currently used for that purpose are presented and discussed.

Key Words: Doppler, cardiotocography, intrauterine growth restriction, IUGR.

REZUMAT

Hipoxia fetală cronică (HFC) poate apărea în multiple condiții clinice și poate avea origine maternă, fetală sau placentară. Ultima cauză este cea mai frecventă datorită vasculopatiei placentare obliterante. În acest caz transferul nutritiv către făt este redus și ulterior poate fi afectat și cel de oxigen. Această lipsă survine la 30-35% din feții cu restricție de creștere intruterină (RCIU). Reducerea transferului de oxigen către făt poate duce la hipoxie (afectare celulară și tisulară). Pentru a preveni o afectare permanentă recunoașterea din timp a HFC este crucială. Metodele curente utilizate în acest scop sunt prezentate și discutate în studiul de față.

Cuvinte cheie: Doppler, cardiotocografie, restricție de creștere intrauterina, RCIU

INTRODUCTION

Chronic Fetal Hypoxaemia (CFH) is the cause of the adverse perinatal outcome in many clinical conditions. CFH can be the consequence of abnormal situations affecting the mother, the fetus or the placenta.

Hypoxaemia is defined as a reduced content of oxygen in the blood that in MOST severe cases, can lead to hypoxia (reduced oxygen supply to the tissues). This last condition generated cellular and organ permanent damage. Therefore the timely recognition and monitoring of the CFH is of paramount importance for preventing hypoxia and its consequences.

In order to achieve this result it is necessary to consider the characteristics of CFH, its aetiology, the effects on fetal vital functions and the methods and possibilities to recognize and monitor it in order to improve the clinical management and the outcome.

ETIOLOGY

Fetal chronic hypoxaemia can be the consequence of reduced capacity of oxygen transport by the maternal

or fetal blood (Anemic hypoxaemia). This situation can be the consequence of severe maternal anemia, or fetal as it is in the case of fetal haemolytic disease.

Hypoxaemia can be also be the consequence of reduced blood flow (ischaemic hypoxaemia). The last is the most common cause of CFH. Placental obliterative vasculopathy, consequence of insufficient secondary trophoblastic invasion that occurs at early stages of the pregnancy, reduces the blood flow from the placenta to the fetus. Maternal-fetal exchanges are affected. First nutrient supply to the fetus is reduced and later on also oxygen supply is compromised. The consequence is fetal growth restriction followed by CFH and acidaemia.

In fetuses showing intrauterine growth restriction (IUGR) due to placental insufficiency CFH is observed in 30-35 % of the cases and it is the principal reason of the unfavourable perinatal outcome.¹

FETAL RESPONSE AND ADAPTATION

When affected by CFH the fetus adapts to the adverse situation by altering many of his vital functions. Blood flow redistribution occurs, heart rate can be altered and movements and amniotic fluid amount can be reduced. The instrumental evaluation of these changes are the basis of the objective assessment and monitoring of the fetal wellbeing and represents the guide for the clinical management.

1. Blood flow redistribution

In case of CFH fetal somatic and splanchnic

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arteries undergo peripheral vasoconstriction while the cerebral arteries can depict a vasodilatation. The finality is to reserve more oxygenated blood to the central nervous system and it is called "brain sparing effect". This phenomenon is considered a defense mechanism. It has been postulated that a sparing effect is also observable at the level of coronary, splenic and adrenal arteries.

2. Fetal heart rate

When CFH is present the fetal heart rate patterns show changes. The variability can be reduced and its reduction is inversely proportional to the level of oxygenation. Bradycardia can occur and in most severe cases late deceleration are observable.

3. Amniotic fluid

The amount of amniotic fluid can be reduced. This reduction is the consequence of restricted fetal urine production caused by reduced blood perfusion of the fetal kidneys due to the increased peripheral resistance observable in the renal arteries.

4. Fetal movements and tone

In an attempt to spare energy fetal movements are reduced in frequency and intensity.

CFH RECOGNITION AND MONITORING

As already said the study of the changes in fetal vital functions represent the method of choice for recognizing the presence of CFH and assessing its level. The most commonly used methods are:

1. Doppler study of fetal and umbilical hemodynamics.
2. Cardiotochography (CTG).
3. Ultrasonic assessment of amniotic fluid amount.
4. The fetal biophysical profile (FBP).

1. DOPPLER

Doppler technology allows the study of haemodynamic characteristics and changes in arterial and venous systems as well. Therefore by applying this technique to umbilical and fetal vessels it is possible to study both the cause (umbilical arteries) and the effects (fetal vessels) of CFH.

The Doppler Velocity Waveform (DVWF) of the arterial vessels is mainly influenced by the peripheral impedance to blood flow downstream the explored segment and significant changes can be observed particularly in the diastolic phase of the cardiac cycle. By comparing systolic and diastolic velocities it is possible to obtain the so called angle-independent parameters. The most commonly used is the Pulsatility Index (PI). Its value increases in proportion to the increase in peripheral resistance. In very severe cases

the flow in diastole cannot be recorded (Absent End Diastolic Flow - EDFA) or be reversed (Reverse Flow - RF). These patterns are called ARED flow.

DVWF at the level of venous system is mainly influenced by the characteristics of the cardiac activity.

a. Umbilical arteries (UA).

In normally evolving pregnancies the PI in the umbilical arteries is progressively reduced. In case of placental obliterative vasculopathy the PI value increases and in most severe cases ARED flows are observable. The values of the PI are proportional to the obliteration of the placental vascular bed but at least 60 % of it must be obliterated before DVWF become apparent.^{2,3} In case of ARED flow the compromise of the placental vasculature is always very important.

Therefore by studying the haemodynamic characteristics of the UA it is possible to recognize the cause of the CFH consequence of reduced maternal-fetal exchanges. It is also necessary to remember that evidence exists that Doppler study improves the outcome only for the UA.⁴

b. Fetal Arteries.

In normal pregnancies the PI of the fetal arteries is fairly constant. In case of CFH somatic and splanchnic arteries undergo peripheral vasoconstriction and cerebral arteries show vasodilatation as a consequence of the blood flow redistribution that is the first defence mechanism operated by the fetus. The PI value increases at the level of the fetal aorta, mesenteric, renal, adrenal, splenic and limb arteries. In very severe condition ARED flow can be observed in the fetal thoracic descending aorta.

On the contrary at the level of cerebral arteries (internal carotid, middle and anterior cerebral) the PI first is reduced as a compensation mechanism in order to supply more blood to the brain. In case of worsening situation decompensation can occur and the PI value increases returning to the previous value.

This aspect must be remembered because a single observation can induce errors in evaluating the fetal condition.

c. Fetal venous system.

As already said the Doppler patterns of the venous fetal vessels are mainly influenced by the characteristics of the fetal heart activity. The most investigated fetal veins are: the inferior vena cava (IVC), the umbilical vein (UV) and the ductus venosus (DV). In presence of CFH, if severe, pulsation is observable in IVC and UV. At the level of the DV reverse flow can be observed.

2. FETAL HEART RATE (CTG)

Cardiotochography is the most widely used

method for assessing fetal wellbeing antepartum and intrapartum as well. CTG can be used without stimuli (Non Stress Test NST) or after inducing contractions, usually by oxytocin perfusion (OCT). The NST at eyeball evaluation has shown to have insufficient sensibility and specificity for assessing high risk pregnancies as it is the case of CFH, due to the large intra- and inter-observer variability.⁵ In order to overcome this problem computerised evaluation of the CTG has been introduced in clinical practice. The most diffused system is that produced by the late Professor G. Dawes at the Nuffield Institute of Research. This system allows the on-line assessment of the fetal heart rate variability. This parameter is strongly influenced by the level of fetal hypoxemia and offers also the possibility to study the trend of the variability along time.⁶

3. AMNIOTIC FLUID AMOUNT

Amniotic fluid amount can be evaluated by using obstetrical ultrasound. Currently 2 methods are used. One is the semiquantitative obtained by measuring the deepest pocket inside the amniotic sac. The second one is represented by the Amniotic Fluid Index (AFI) that is obtained by measuring the depth of the pockets in four uterine quadrants.

4. FETAL BIOPHYSICAL PROFILE (FBP)

This is a scoring system that take into consideration CTG, fetal tone and movements, amniotic fluid amount and fetal respiratory movements.

Also if largely used, there is no evidence of his efficacy in the management of high risk pregnancies.⁷

CLINICAL MANAGEMENT

First of all it is necessary to remember that the only available treatment of the CFH, caused by placental insufficiency, is represented by the delivery. As the perinatal outcome is mainly dependent on gestational age, it becomes clear that the choice of the timing of the delivery, when CFH occurs at low gestational age, represents a compromise between the risks of intrauterine demise or permanent damage and those of prematurity.

Therefore the management is different according to the GA, if less or more than 32-33 weeks.

The data that are usually taken into consideration for choosing the timing of the delivery are those obtained by Doppler study and CTG whenever possible computerized. What is crucial in managing this critical situation is to remember the pathophysiological background of the changes observable by the two

methods.

1. Umbilical arteries

Increased PI over the 2nd Standard Deviation indicates reduction of blood supply to the fetus and that CFH is likely to occur. EDFA is a sign of severely reduced blood supply and CFH is already present. RF indicates an extreme reduction of blood supply, CFH is severe and the fetus can be already compromised. Intrauterine death can be expected in few days, perinatal mortality is very high and the prevalence of severe handicaps in survivors is also very high.⁸

2. Fetal arteries

Blood flow redistribution is a sign of adaptation to CFH. EDFA in the fetal thoracic aorta indicates very severe CFH. RF in aorta is a sign of severe compromise.

3. Fetal veins

Significant alteration of the DVWF in fetal veins is a strong predictor of adverse perinatal outcome.

At the moment there is no evidence that its clinical use can be recommended in the management of CFH.^{9,10} A randomized clinical trial (TRUFFLE Study) is ongoing.

4. CTG

Significant reduction of fetal heart rate variability is best assessed by using computerized CTG. A short variability below 3.5 milliseconds indicates the presence of CFH and acidemia and predicts a 29 % probability of intrauterine demise. The observation of the trend of the short term variability (STV) offers the possibility to evaluate the progress of the fetal deterioration.

Therefore the following recommendations according to different situation, can be presented.

I. BEFORE 32-33 WEEKS

A. PI over 2 SD in UA, fetal blood flow redistribution, with diastolic flow present, STV more than 3.5 milliseconds.

Clinical and instrumental close surveillance. Maternal corticosteroids administration to enhance fetal lung maturity.

B. EDFA in UA, fetal blood flow redistribution with diastolic flow present, STV over 3.5 milliseconds.

Close surveillance. Corticosteroids maternal administration. Consider delivery when fetal lung maturity is achieved. Immediate delivery not necessary as the fetus can sustain this haemodynamic condition for a few days.

C. RF in UA. In this situation the fetal Doppler and STV are always showing abnormal values.

Consider delivery after exhaustive counselling with the family regarding early and late outcome. Non aggressive management can be considered.

II. 34 WEEKS AND MORE

A. UA PI over 2 SD with diastolic flow present. Fetal blood flow redistribution with diastolic flow present, STV over 3.5 milliseconds. .

Close surveillance. Timing of the delivery in case of deterioration. Vaginal delivery possible in 24-40 % of the cases.^{1,11}

B. EDFA in UA

If fetal lung maturity is warranted timing of the delivery should be considered.

C. RF in UA. In this condition fetal Doppler and STV are abnormal.

Delivery can be considered after exhaustive counselling with the family regarding early and late outcome.

Whenever possible the birth of these critical infants should take place at tertiary level centers where the optimal newborn management is available.

CONCLUSIONS

CFH is a severe complication of pregnancy. In the majority of the cases the etiological factor is obliteration of the placental vascular bed leading first to fetal growth restriction and later on to

reduced oxygen supply. This condition is often called “placental insufficiency”. As the only available treatment is represented by the delivery an early recognition is crucial for optimizing management and outcome. Instrumental fetal semiology, Doppler and CTG, preferably computerized, are the principal used methods .

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