Acta Biomed 2023; Vol. 94, Supplement 1: e2023054 DOI: 10.23750/abm.v94iS1.14011

Case report

Cardiotocography pattern: not always a true friend

Carmen Imma Aquino[1,2], Roberta Amadori[1], Elisabetta Vaianella[1], Silvia Bonassisa[1], Alessandro Libretti[1], Daniela Surico[1], Valentino Remorgida[1]

[1]Department of Gynaecology and Obstetrics, University of Piemonte Orientale, Ospedale Maggiore della Carità, Novara, Italy; [2]Department of Translational Medicine, University of Piemonte Orientale, Novara, Italy.

Abstract. Fetal well-being in labor could be assessed trough cardiotocography (CTG). Some doubts have been raised about its unequivocal applicability. Pathological CTG is in most cases connected to fetal acidosis at birth, but other potential causes must be considered in the differential diagnosis. A 31-years-old G2P1 patient referred to our Department of Obstetrics and Gynecology for her scheduled post-term CTG at 40 weeks and 3 days of gestation. The pregnancy was uneventful. CTG was classified as suspicious, and after pharmacological induction, it switched as pathological: an emergency cesarean section was performed. Venous and arterial blood sample taken from the umbilical cord were normal. The next assessments revealed that Atrial Flutter (AFL) occurred at birth. Suspicious CTG is not always associated to neonatal asphyxia. Cardiotocography can help not only in the evaluation of fetal distress, but also in the assessment of global fetal cardiac activity. The presence of a fetal heart defect should be considered when CTG is suspicious. (www.actabiomedica.it)

Key words: tachycardia, neonatal asphyxia, cardiotocography, fetal monitoring, arrhythmia, operative delivery

Introduction

Cardiotocography (CTG) is a standard test used during pregnancy and delivery. The procedure is based on a continuous electronic record of baby's heart rate and uterine contractions. It is the best indicator for fetal surveillance during labour and prevention of intra-uterine demise, birth asphyxia, newborns' neurological defects and neonatal mortality. Despite a global use of this technique, some doubts have been considered about its unequivocal applicability. Its evaluation requires, anyway, high expertise to indicate whether the trace is Normal, Suspicious or Pathological (1, 2).

A good interobserver concordance was not always found in the analysis of CTG traces (3), i.e., in case of bradycardia, reduced variability, saltatory pattern, absence of accelerations and decelerations. Despite it is the most widely antepartum assessment, it is characterized by low specificity for fetal acidosis and poor perinatal outcomes (3).

There is no unequivocal evidence that antenatal CTG improves perinatal outcome, and a suspicious CTG does not always justify operative intervention during pregnancy or labor (1-3). Although interventions on Pathological CTG aim to avoid sequelae of fetal acidosis at birth, differential diagnosis with other potential causes of CTG changes must be carefully evaluated.

Case report

A 31-years-old G2P1 patient without comorbidities presented at 40 weeks and 3 days of gestation to our Department of Obstetrics and Gynecology for her scheduled post-term CTG. The pregnancy was uneventful. CTG was classified as suspicious (Fig.1) due to the presence of fetal heart rate 160 bpm, low variability and absence of accelerations. The gynecologist on duty decided for induction of labor. After the

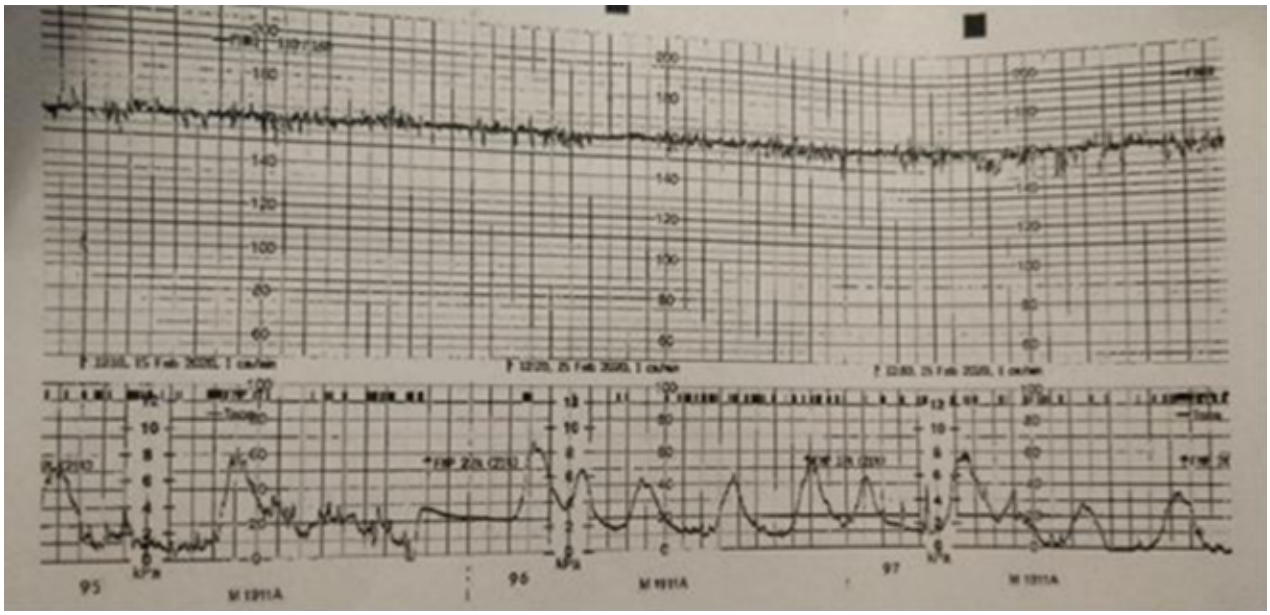

Figure 1. Suspicious CTG before induction.

first vaginal administration of Dinoprostone 2 mg, CTG was evaluated as pathological due to persistence of fetal heart rate 160 bpm, a non-variable trace, and absence of accelerations for more than 30 minutes. An emergency caesarean section was then performed.

Venous and arterial samples from the umbilical cord were collected. pH was 7.372 for the venous sampling (VS) and 7.318 for the arteriosus one (AS); pCO2 (mmHg) was 38.2 for the VS and 52.4 for the AS; pO2 (mmHg) was 29.2 for the VS and 14.0 for the AS; cLac (mmol/L) was 1.2 for the VS and 1.1 for the AS; Anion gap (mmol/L) was 5.1 for the VS and 4.1 for the AS. The newborn was a female of 3750g, with head circumference and length in normal range. The baby was eupneic, with normo-transmitted vesicular murmur and hypo-sphygmic peripheral pulses. Apgar score was 9/9 at 1 and 5 minutes. The normal values on the collected samples allowed exclusion of neonatal asphyxia. An atrial flutter (AFL) was therefore suspected to justify the anomalies of the CTG trace.

The newborn was then admitted to the Neonatal Intensive Care in spontaneous breathing with oxygen supplementation. The chest X-ray excluded pneumonitis; heart size was normal. Echocardiography revealed normal heart anatomy.

At birth, the baby had an arrhythmic heart rate of 260 beats per minute already suggestive of tachyarrhythmia. The ECG described a supraventricular tachycardia with a narrow QRS atrial rate. On the first day of life, she was treated with Adenosine, with a momentary clinical stability. Next ECGs confirmed the AFL. After the administration of 3 boluses of Adenosine, her heart rate starting to present various recurrent episodes of AFL. Given the recurrence and the tachycardiac aspect, according to the pediatric protocols of our department, it was decided to threat the newborn with Amiodarone, with a dose of 0.5 mg/kg/h. Unfortunately, despite an increase in dosage (0.7/mg/kg/h), this drug did not normalize the heart rate that continued to present an AFL pattern on the ECG. Therefore, it was decided for electrical cardioversion, successfully performed with 3J.

After 24h, in the absence of other episodes of arrhythmia, intravenous Amiodarone therapy was replaced with oral treatment. The baby was discharged on the nineteenth day of life. As AFL did not occur again during the 6-month follow-up, therapy was discontinued, with the clinical attention reserved for any other healthy newborn. Today the baby is completely healthy.

Discussion

Not all suspected CTGs are a prelude of neonatal asphyxia: here we present a case of Atrial Flutter with CTG alterations.

AFL is an uncommon supraventricular tachyarrhythmia (1/3 of fetal arrhythmias), characterized by a fast, irregular, atrial activity of 280–500 BPM on ECG trace. This arrhythmia is described by saw-tooth flutter waves with an atrial rate of up to 500 beats/min and it is frequently associated with 2:1 atrioventricular (AV) conduction by macro-reentry within the atrial wall. It may be induced by myocarditis, positive SSA/SSB autoantibody, congenital heart disease (i.e., Ebstein's anomaly, transposition of the great arteries, complex cyanotic heart defect, atrial septal defect, valve diseases) or cardio-surgery (4). AFL could be documented in the last trimester of pregnancy. This might be related to the higher vulnerability to atrial extrasystoles, due to the maximum size reached by the atrial fibers. AFL may cause cardio-vascular failure and death (4).

This communication wishes to focus on the fact that not all suspected CTG traces are linked to neonatal asphyxia.

The inter- and intra-observer variability and the subjectivity of CTG interpretation must be evaluated for each case . A suspicious admission CTG, an increased or decreased amniotic fluid index (AFI), and an altered doppler ultrasound study can predict the stress that the fetus is experiencing, but they are not always diriment (4).

Cardiotocography helps in the evaluation of fetal distress, not only in case of asphyxia, but also in the assessment of global fetal cardiac activity. The aim of this report is to remind that, in the case of alterations of the CTG trace, cardiological evaluation of the fetus/newborn is essential.

Informed Consent Statement: Newborn's parents have given consent to the use of her clinical data for research purposes.

Ethic Committee: not applicable

Conflict of Interest: Each author declares that he or she has no commercial associations (e.g. consultancies, stock ownership, equity interest, patent/licensing arrangement etc.) that might pose a conflict of interest in connection with the submitted article.

Authors Contribution: 1. Conception and design of the work: Carmen Imma Aquino, Roberta Amadori. 2. Data collection: Carmen Imma Aquino, Roberta Amadori, Elisabetta Vaianella. 3. Data analysis and interpretation: Carmen Imma Aquino, Roberta Amadori, Elisabetta Vaianella, Silvia Bonassisa, Alessandro Libretti, Daniela Surico, Valentino Remorgida. 4. Drafting the article: Carmen Imma Aquino, Roberta Amadori, Alessandro Libretti, Daniela Surico. 5. Critical revision of the article: Carmen Imma Aquino, Roberta Amadori, Alessandro Libretti, Daniela Surico, Valentino Remorgida. 6. Final approval of the version to be published: Carmen Imma Aquino, Roberta Amadori, Elisabetta Vaianella, Silvia Bonassisa, Alessandro Libretti, Daniela Surico, Valentino Remorgida.

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Correspondence:
Received: 2 December 2022
Accepted: 7 January 2023
Full first name Aquino CI, MD
Department of Gynecology and Obstetrics,
University of Piemonte Orientale,
Ospedale Maggiore della Carità,
Via Mazzini 18, 28100, Novara, Italy.
Tel: 0321 3731.
E-mail: 20033548@studenti.uniupo.it