European Journal of Obstetrics & Gynecology and Reproductive Biology 185 (2015) 103–107

Contents lists available at ScienceDirect

European Journal of Obstetrics & Gynecology and Reproductive Biology

journal homepage: www.elsevier.com/locate/ejogrb

Intraoperative findings, placental assessment and neonatal outcome in emergent cesarean deliveries for non-reassuring fetal heart rate

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ARTICLE INFO

Article history:
Received 30 August 2014
Received in revised form 27 November 2014
Accepted 5 December 2014

Keywords:
Emergent cesarean deliveries
Intraoperative findings
Neonatal outcome
Non-reassuring fetal heart rate
Placental histopathology

ABSTRACT

Objective: To correlate between intraoperative findings, placental histopathology and neonatal outcome in emergent cesarean deliveries (ECD) for non-reassuring fetal heart rate (NRFHR).
Study design: Data on ECD for NRFHR were reviewed for labor, documented intraoperative findings, neonatal outcome parameters and placental histopathology reports. Results were compared between those with and without intraoperative findings. Placental lesions were classified to those related to maternal underperfusion or fetal thrombo-occlusive disease, and those related to maternal (MIR) and fetal (FIR) inflammatory responses. Neonatal outcome consisted of low Apgar score ($\leq$7 at 5 min), cord blood pH $<$ 7.0, and evidence of respiratory distress, necrotizing enterocolitis, sepsis, transfusion, ventilation, seizure, hypoxic–ischemic encephalopathy, phototherapy, or death.
Results: Intraoperative findings were observed in 49.5% of 543 women, mostly cord complications (77%). Placental lesions were more common in those without intraoperative findings as compared to those with intraoperative findings: placental lesions related to maternal under-perfusion, vascular lesions, 9.1% vs. 4.1%, $p = 0.024$, and villous changes, 39.2% vs. 30.7%, $p = 0.047$, lesions consistent with fetal thrombo-occlusive disease, 13.6% vs. 7.4%, $p = 0.024$, and inflammatory lesions, MIR and FIR, $p = 0.033$, $p = 0.001$, respectively. By using multivariate logistic regression analysis, adverse neonatal outcome was found to be dependent on maternal age, gestational age, preeclampsia placental weight $<$10th%, and MIR.
Conclusion: NRFHR necessitating ECD may originate from different underlying mechanisms. In about half, the insult is probably acute and can be identified intraoperatively. In the remaining half, underlying placental compromise may be involved.

Introduction

Low fetal oxygenation during labor may lead to neonatal morbidity and mortality [1]. Transfer of oxygen to the fetus during labor may be altered by various mechanisms [2], leading to hypoxic changes and acidosis, such as abnormal perfusion within the utero-placental vasculature or compression of the spiral arteries or umbilical cord during contractions that result in cessation of the umbilical cord blood flow. In addition, pregnancy complications, such as hypertensive disorders, diabetes mellitus (DM), inflammatory diseases, chorioamnionitis, preterm labor, and fetal growth restriction (FGR), all may further exaggerate the compromised blood supply to the fetus during labor.

In an attempt to prevent hypoxic–ischemic morbidities to the neonate, non reassuring fetal heart rate (NRFHR) monitoring during labor is one of the major indications for prompt delivery by performing an emergent cesarean delivery (ECD) [3,4]. Intraoperative findings, such as tight cord entanglements, uterine rupture or placental abruption may explain why fetal hypoxia that is expressed by NRFHR occurred. However, in many situations no explanation can be found, suggesting that the placenta could be the key, and therefore should be sent for assessment [5]. Few studies

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http://dx.doi.org/10.1016/j.ejogrb.2014.12.007
0301-2115/

have investigated the role of placental histology in association with NRFHR; Salafia et al. [6,7] reported an association between abnormal FHR patterns and acute inflammation of the placenta. Our group also demonstrated that histological chorioamnionitis was associated with cord blood acidosis in neonates delivered by ECD [8]. By contrast, Moberg et al. [9] observed an increased prevalence of FHR tracings consistent with umbilical cord compression in women with preterm premature rupture of the membranes, with no increase in placental findings suggestive of chorioamnionitis, and Robinson et al. [10] demonstrated that the presence or extent of placental inflammation does not appear to differ between women who delivered by ECD for category II FHR and those who delivered by ECD for labor arrest. In addition to these contradictive findings on the association between NRFHR and placental histology, currently, no information exists on the association between NRFHR, ECD, intraoperative findings and placental assessment. Therefore, the present study was aimed to correlate between intraoperative findings, placental histopathology and neonatal outcome, in ECD for NRFHR.

Materials and methods

The medical records of all women who underwent ECD for NRFHR, at the labor ward at Edith Wolfson Medical Center, Holon, Israel, from 1.2009 to 6.2013, were reviewed. Cases eligible for the study were identified from our computerized data system. The study group included women who delivered a singleton, between 24 and 42 weeks, by ECD for NRFHR as the only indication, and their placentas were sent to histopathological evaluation. Excluded from the study were patients with additional indications for ECD, as abnormal progress in labor, initial maternal refusal for CS, patients with evidence of fetal or neonatal malformations, or cases with missing data.

As part of our departmental protocol, surgeons are instructed to document any findings recognized by them as possible causes for the NRFHR. Such findings included: cord entanglements (including various positions of entanglement and number of loops), tight true knots of the umbilical cord, short umbilical cords (<50 cm), umbilical cord prolapse, macroscopic placental abruption and uterine rupture.

The study group was divided into two subgroups; those in which macroscopic intraoperative findings suggestive of causes for NRFHR were found (Group 1), and those without findings (Group 2).

Approval was obtained from the Local Ethics Committee.

Data collection

The clinical data for the present study was collected from the patients' medical and surgical files and included: demographic and labor characteristics: age, gravidity and parity, BMI, pre-gestational DM, gestational DM, thrombophilia, preeclampsia, TOLAC attempt, gestational age at delivery, oligohydramnious (amniotic fluid index <5 cm), cervical dilatation, the presence of intrapartum fever >38 °C, and meconium. Gestational age was confirmed by first-trimester ultrasonography. Active labor was defined as regular painful uterine contractions and cervical dilatation of >4 cm.

Immediately after birth, all neonates were examined by pediatricians. Birth weight percentile for gestational age was assigned using the updated Israeli growth charts [11]. FGR was defined as actual birth weight <10th% for gestational age. The following information was collected from the neonatal records: Apgar scores, cord blood pH, sepsis (positive blood or cerebrospinal fluid culture), need for blood transfusion, need for phototherapy, respiratory distress syndrome, need for mechanical ventilation, necrotizing enterocolitis, intraventricular hemorrhage, hypoxic ischemic encephalopathy, seizures, and death.

Placental examination

As part of our departmental protocol, in every case of ECD for NRFHR placentas are sent to histopathological evaluation. Placental pathology examinations were performed using our standard protocol, by a single pathologist (author L.S), who was blinded to neonatal outcome [8,12]. Placental lesions were classified by maternal or fetal origin according to the criteria that were adopted by the Society for Pediatric Pathology [13].

Briefly, placental weight was determined 24 h after delivery, and the percentile was determined according to placental weight charts [14]. Each placenta was fixed in formalin, and at least 5 samples were embedded in paraffin blocks for microscopic assessment.

Lesions of maternal vascular supply included placental hemorrhages (marginal, and retro-placental), vascular changes associated with maternal underperfusion (acute atherosis and mural hypertrophy), and villous changes associated with maternal underperfusion (increased syncytial knots, villous agglutination, increased intervillous fibrin deposition, and villous infarcts). Lesions of fetal vascular supply were defined as findings consistent with fetal thrombo-occlusive disease: vascular lesions (thrombosis of the chorionic plate and stem villous vessels) and villous changes (hypovascular, fibrotic and avascular villi).

Findings consistent with chorioamnionitis were defined by the presence of an inflammatory neutrophil infiltrate at two or more sites on the chorionic plate and extra-placental membrane. The maternal inflammatory response (MIR), was divided into three stages; stage 1 – characterized by the presence of a few scattered neutrophils in the subchorionic space; stage 2 – characterized by many neutrophils (11–30 per HPF) in the lower half of the chorionic plate; and stage 3 – characterized by dense infiltrates of neutrophils (more than 30 per HPF) throughout the chorionic plate. The fetal inflammatory response (FIR) was also divided into 3 stages: stage 1 – early, umbilical phlebitis; stage 2 – intermediate, umbilical arteritis; and stage 3 – concentric umbilical perivasculitis (necrotizing funisitis). Placental histologic examination also included the detection of meconium stained membranes associated with columnar change in amniotic epithelium or the appearance of pigmented macrophages.

Statistical analysis

Data were analyzed with SPSS software, version 15.0. Continuous variables were calculated as mean ± SD or median and range, as appropriate. Categorical variables were calculated as rate (%). Continuous parameters were analyzed by Student's *t*-test and categorical variables by chi-square test or by Fisher exact test, as appropriate. A *p*-value of <0.05 was considered statistically significant.

Composite neonatal outcome was defined as one or more of the following complications: Apgar scores < 7 in 5 min, cord blood pH < 7.0, sepsis, blood transfusion, phototherapy, respiratory distress syndrome, transient tachypnea of the newborn, mechanical ventilation, intra-ventricular hemorrhage, seizures, hypoxic–ischemic encephalopathy, NEC, or death.

To identify independent risk factors for adverse neonatal outcome, a multivariate stepwise forward logistic regression analysis was performed. Composite neonatal outcome served as a dependent variable. The various maternal demographics, pregnancy complication, and the various placental lesions served as independent variables.