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Full lenght article

Immaturity for gestational age of microvasculature and placental barrier in term placentas with high weight

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ABSTRACT

Objective: Villous immaturity for gestational age is a multifactorial developmental deviation associated with unexpected placental insufficiency, fetal hypoxia and term fetal death. In our previous work we have shown that immature CD15+/CD31+/CD34+ endothelial cells were an important indicator of placental villous immaturity and chronic insufficiency. The aim of this study was to perform a comparative analysis of CD15-marked immaturity in the vessel walls between normal and pathological term placentas of clinically and structurally heterogenous groups with normal, low and high weight.
Study design: 165 clinically normal and pathological placentas of gestational age 39–42 with normal weight (25–75 percentile), low weight (<10 percentile) and high weight (>90 percentile) were structurally and immunohistochemically analyzed. Excluded were placentas with a severe form of placental insufficiency associated with intrauterine fetal death, low APGAR-score, genetic and chromosomal diseases or placental inflammations. The distribution patterns of CD15, CD31 and CD34 were assessed separately in the macrovasculature, microvasculature and placental barrier (PB) – associated capillaries.
Results: All placental groups with normal weight, low weight and high weight include normal, accelerated villous maturation or villous immaturity independent of their weight. However, a significant increase of immature CD15+/CD31+/CD34+ endothelial cells was detected in microvasculature and PB –associated capillaries in high weight-placentas (63.5%/52.2%), compared to those of normal weight (13.8%/8.2%) and low weight (16.1%/17.8%). The distribution of macrovascular immature CD15+/CD31+/CD34+ endothelial cells did not show such marked differences.
Conclusion: We have identified the immaturity of microvasculature and PB –associated capillaries with a pathological persistency of immature CD15+/CD31+/CD34+ endothelial cells and a reduction of terminally differentiated CD15-/CD31+/CD34+ endothelial cells in a structurally and clinically heterogeneous group of high weight-placentas. We assume that immaturity of placental vessels are part of prenatal adaptational processes that can be recruited in different emergency situations and may provide potential targets of therapeutic correction of placental growth and chronic insufficiency. We therefore recommend the use of CD15-based immunophenotyping as a method to identify latent unfavorable conditions of fetal development in the intrauterine life and individual risk of disease in the postnatal period.

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Introduction

An actual problem for obstetricians and neonatologists are clinically latent development disorders of the placenta in late pregnancy complicated in some cases by unexpected placental insufficiency and a perinatal morbidity and mortality [1–3]. Postnatal assessment of the pathological placental phenotype may help to identify unfavourable conditions in intrauterine life and early stratification of a heterogeneous population of newborns with the detection of personalised risk of disease in postnatal life.

Maturation disorders of placentas can be a cause of chronical placental insufficiency and fetal hypoxia [2–5]. Two main contrary types of structural disturbances of villous maturation have been described: accelerated for gestational age and immature for gestational age. Latter is an important cause of latent placental insufficiency [2,3,5]. There is only an insufficient development of

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the terminal villi and vasculo-syncytial membranes (VSM), both constituting the main components of the placenta barrier (PB) [4,5]. This immaturity for gestational age of the PB prolongs the intravillious feto-maternal diffusion distances in comparison to a mature placenta [5]. Maturation disorders of placentas may correlate with severe forms of placental weight deviations and reflect the prenatal pathological and adaptational processes in the feto-placental unit [4–6]. Recent studies have proven the existence of different populations of resident progenitor cells in the vascular walls, which carry an important role in physiological and pathological adaptational mechanisms of vessel walls in the pre- and postnatal period [7–11]. Our group has characterized the maturation process of progenitor cells in the placental vascular walls from multi-lineal hemangioblasts to terminally mature endothelial cells during different gestational ages (4–42nd week of pregnancy) [12,13]. The balance between immature CD15-positive endothelial cells (CD15+ endothelial cells) and terminally differentiated CD15-negative endothelial cells (CD15- endothelial cells) changes in a stage-specific manner during the normal process of placental maturation. In a previous study, we have shown that stage-specific populations of immature CD15+ endothelial cells were an important indicator of persisting placental immaturity and chronic insufficiency in term placentas [14]. We observed a site-specific expansion of vascular CD15+ endothelial progenitor cells in the term placentas with chronic fetal stress response [14].

Novel diagnostic and therapeutic strategies are warranted to successfully predict and treat placental diseases of the perinatal stage, but require a better understanding of the pathogenesis behind the maturation and growth of the placenta and of the placental vasculature.

CD15 (called Lewis X or stage-specific embryonic antigen-1) is a carbohydrate adhesion molecule [15–17]. It acts as a key ligand of selectin and plays an important role in migration and differentiation of the stem cells [18]. Furthermore, CD15 is stage-specific expressed in several embryonal, neuronal and vascular stem cells [13,19–21]. Yue et al. has shown that CD15 expression occurs in an intermediate stage of differentiation of embryonic stem cells into endothelial cells in vitro [22].

The aim of this study was to perform a comparative analysis of CD15-marked immaturity in the vessel walls between normal and pathological term placentas of clinically and structurally heterogenous groups of normal, low and high weight.

2. Materials and methods

2.1. Tissue specimens and histology

165 placentas of gestational age 39–42 were analyzed in this retrospective study. Excluded were placentas with a severe form of placental insufficiency associated with fetal death, low APGAR-score, chromosomal diseases or inflammations. All placentas underwent pathological examination in the Department of Pathology, University Medical Center Mainz within a period from June 2014 until December 2015. For this study excess placental material was used in accordance with the regulations of the Local Ethical Committee. In all cases advanced written informed consent was obtained from the pregnant women.

Placentas were collected immediately after delivery and weighed without membranes or the umbilical cord. Representative tissues were fixed in 4% neutral buffered formalin. In this study we have analysed only those severe forms of weight deviations (<10 and > 90. percentile). Data regarding placentas of less severe weight deviations have already been published in our previous work [12,14].

All placentas were divided into 3 groups: normal weight (25–75 percentiles), low weight (<10 percentile) and high weight (>90 percentile) [2,23]. The main clinical and morphological data of the placentas are depicted in Table 1.

Macroscopic and histological observations were performed in accordance to the Vogel principles [14,24]. Every group with

Table 1
The main clinical and morphological data of the groups.

Characteristics of groups	Groups (39–42 pregnancy week)		
	Normal weight (n = 42)	Low weight (n = 60)	High weight (n = 63)
Characteristics of placenta and umbilical cord			
Weight, g	499,1 ± 34,5	309,4 ± 53,3	697,5 ± 56,4
Inflammatory changes, n	0	0	0
Circulatory disorders			
Infarction, n	4	7	3
Hematoma, n	1	5	2
Tumors, n	0	0	0
Features of umbilical cord, n	0	0	0
Characteristics of newborn			
APGAR score	8–10	8–10	8–10
Genetic/chromosomal anomalies, n	0	0	0
Intrauterine fetal death, n	0	0	0
Intrauterine growth restriction (IUGR), n	12	37	3
Large for gestational age (LGA), n	3	0	25
Small for gestational age (SGA), n	0	9	0
Clinical characteristics			
Norma, n	23	0	0
GDM, n	0	7	22
DM I type, n	0	0	4
HELLP-syndrome, n	2	3	0
Preeclampsia (late-onset), n	6	9	9
Caeserean section, n	2	26	20
Premature placental abruption, n	0	8	1

gestational diabetes mellitus (GDM); diabetes mellitus (DM); Hemolysis, Elevated Liver enzymes, and Low Platelet count (HELLP)

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