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# Placenta - the "black box" for the evolution of gestation and evidence in forensic expertise of pregnancy

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**Abstract:** The authors highlight the informational value of placenta in the understanding of antepartum, intrapartum and postpartum pathology within the fetal-placenta-maternal biosystem. The study, conducted on 120 placentas, selected from 1450 term pregnancies, draw the attention over issues related to maternal, fetal and newborn pathology that undergo simultaneously with phenotype transformations of placental trophoblastic, vascular and fibrin structures. Macro- and microanatomic analysis was conducted on fetal-placenta and maternal-placenta circulatory systems in order to evaluate placental reaction synergisms in the presence of umbilical vascular thrombosis and/or massive intra- and perivillous fibrin substance deposition.

Anatomic examination of placenta allows the understanding of physiopathological processes within the fetal-placenta-maternal system and provides objective evidences for forensic expertise in order to estimate the approximate age of local lesions, to assess perinatal asphyxia and neurocongenital development perturbations.

**Key Words:** placenta, phenotype transformations, trophoblast, fibrin, vascular thrombosis.

Forensic expertise of abortion and gestation require morphologic analysis, of both the fetus and placenta. Placenta is not always receiving the attention it deserves. It represents an important source of information that offers a better understanding of physiopathological processes occurring either antepartum, intrapartum or postpartum. Anatomic analysis of placenta is seldom utilized and its importance underestimated. The identification of some new anatomic-clinical entities draws the attention over current anatomical examination of placenta (Benirschke 1961 [1], Benirschke and Driscoll 1967 [2]; Fox 1967 [3]; Benirschke and Kaufmann 1995 [4]; Redline and Pappin 1995 [5]).

The purpose of this paper is to provide arguments for the use of information obtained through anatomic examination of placenta, relevant for the assessment of structural elements phenotype transformations involved in pathological-morphogenesis of fetal lesions. Placenta owns the "trimester diary" of gestation and represents "the black box" of maternal-fetal-placenta system evolution.

The aim of this paper is to achieve a macro- and microanatomic analysis regarding, the effects of thrombotic vasculopathy over blood flow within fetal-placenta circulatory system and the effects of massive fibrin deposition around or inside villosities.

## MATERIALS AND METHODS

The study was conducted on 120 placentas, selected from 1450 cases of pregnancies at term: 85 cases

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with fetal pathology (intrauterine growth retardation, intrauterine death, congenital malformations), 15 cases with neonatal pathology (perinatal asphyxia with Apgar score lower than 6, newborn death), 5 cases with maternal pathology (hypertension, last trimester metrorrhagia) and 15 cases with placenta pathology (placenta acreta, size abnormalities, retroplacental hematoma).

Macroanatomic examination analyzed the morphology of: umbilical cord, membranes, chorial plate, basal plate and transplacental section surfaces.

Microanatomic examination of seriate sections after paraffin inclusion and Hematoxylin- Eosin, picrofuxine Weigert, PAS, and Gomori staining, allowed the visualization of phenotype transformations at the level of chorial plate, placenta parenchyma and basal plate.

Macroanatomic imaging was done using Canon EOS 1ds Mark II Digital Camera, equipped with Macro-Ultrasonic Lens EF 100mm F/2.8. Microanatomic images were achieved with Nikon 80i research microscope, using Nikon DS Fi 1 Digital Camera.

## RESULTS

Macro- and microanatomic analysis was performed for fetal-placenta and maternal-fetal circulatory systems, in order to assess the consequences of vascular thrombosis as well as the massive fibrin substance deposition inside or around villosities.

***A. Anatomic analysis of "fetal thrombotic vasculopathy" syndrome, which determines blood flow perturbations within fetal-placenta circulatory system***

The external configuration of placental disk, fetal and maternal faces was macroscopically studied. The umbilical cord inserts paracentral: more frequently marginal or velamentous (Fig. 1 A-C). It is twisted and endured at the level of placental extremity (Fig. 1 B, C). Alanto-chorial blood vessels are dilated and hardened at palpation (Fig. A-E).

On the placental disk section surface are often found: occlusive thrombosis, at the level of alanto-chorial (Fig. 1 G-I), troncular (Fig. 1 H, I) and peduncular (Fig. 1I) blood vessels, as well as fibrin substance deposits at the level of chorial blade, around peduncular villosities and within pars basalis placentae (Fig. 1 J).

Microanatomic examination of seriate sections through placenta fragments, allowed the visualization of: fibrin fascicles within chorial blade structure and around subchorial blood vessels (Fig. 1 K), fibromuscular sclerosis of subchorial blood vessels middle tunic (Fig. 1 K, N), thrombosis of alanto-chorial, troncular and peduncular blood vessels (Fig. 1 M, N), as well as endothelial pads.

***B. Anatomic analysis of phenotype transformations undergone by placenta structures that determine blood flow perturbations within fetal-placenta circulatory system***

The microscopic examination of seriate sections through placenta structures showed the participation of fibrin substance in the phenotype transformations of placenta villosities and peduncular villosities (Stem villi). Following these transformations, intervillous space is collapsed (Fig. 2C) and accentuated by trophoblast which generates syncitial knots inside intervillous space (Fig. 2 E, F, K). Peduncular villous stroma is dissociated by thick fibrin bands (Fig. 2 E, F).

At the level of intermediary and terminal villous stroma, we identified the disappearance of blood capillaries, secondary to a massive intravillous fibrin accumulation which provides the aspect of villous fibrin necrosis (Fig. 2 J-O). In the periphery of these villosities, a trophoblast thin layer is still persisting, which contributes to the formation of syncitial knots (Fig. 2 J-O). Perivillous fibrin accumulation leads to intermediary and terminal villosities agglutination (Fig. 2 D, G-I).

Massive fibrin deposits are also visible at macroscopic examination on the maternal and fetal surfaces of the placenta (Fig. 1D), as well as on macroscopic seriate sections. When analyzing the section surfaces, a great variability regarding fibrin deposits location was noticed: marginal (Fig. 2B), subchorial (Fig. 1 J), juxtabasal (Fig. 2 B), transplacental (Fig. 1 J) and in pseudolobular spots (Fig. 2 B).

## DISCUSSIONS

Placenta, as an organ with ephemeral existence, represents the border between fetus and maternal host and acts like a macro-membrane between two blood circulations: fetal-placenta and maternal-placenta. Due to its villous and vascular structures, placenta assures the development of breathing, endocrine secretion, metabolic changes and immune processes.

Phenotype transformations that appear during placental genesis are anatomic markers, irreversibly stocked inside its vascular and/or villous structures (Dragoi *et al.* 2009, 2010 [6-8]; Zimta *et al.* 2012 [9]; Melinte *et al.* 2015 [10]; Fox 1967, [11], 1970 [12]). They assure the placenta role of information storage, regarding determinant factors of both fetal-placenta and maternal-placenta blood circulations perturbations. Anatomic analysis of this information contributes to the morphologic-functional assessment of circulatory perturbations effects over the whole fetal-placenta biosystem, especially over the evolution of fetal intrauterine development ("intrauterine growth retardation") and over antepartum, intrapartum or postpartum fetal death.

In this context, we highlight the importance of placenta examination, in all cases like: fetal growth retardation, preterm birth, fetal death, newborn

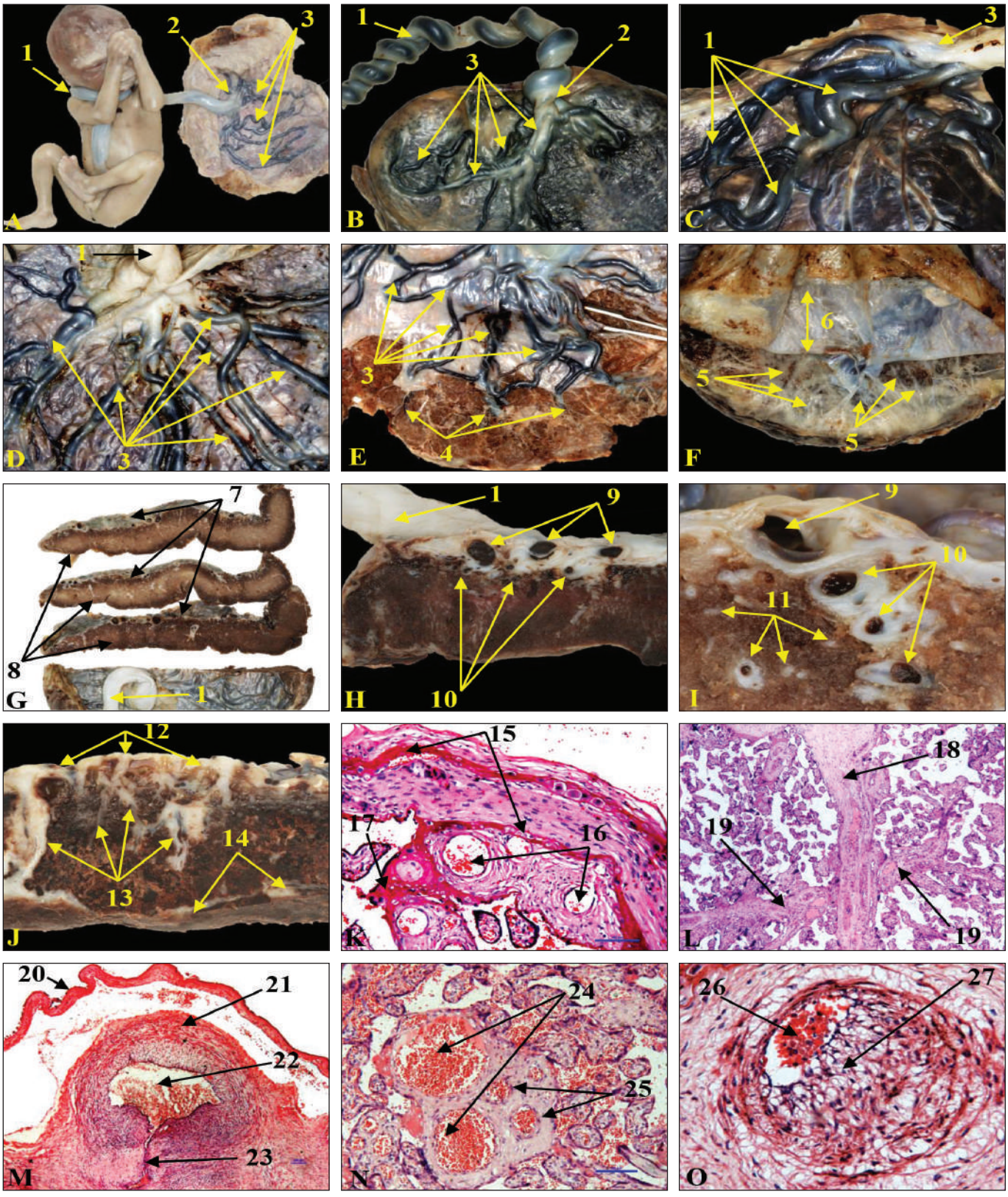


**Figure 1.** Fetal thrombotic vasculopathy. 1.Funiculus umbilicalis; 2. Insertio marginalis; 3. Alanto-chorial vascular network; 4. Blood vessels within troncular villosity; 5. Subchorial villous network; 6. Chorial lamina; 7. Placenta fetal face; 8. Placenta maternal face; 9. Alanto-chorial thrombosis; 10. Troncular villous trombosis; 11. Peduncular villosities thrombosis; 12. Fibrinoid chorial lamina; 13. Fibrinoid around peduncular villosity; 14. Fibrinoid within pars basalis placentae; 15. Fibrinoid fascicles within chorial lamina; 16. Subchorial vessels; 17. Subchorionic perivascular fibrinoid; 18. Villus peduncularis; 19. Mature intermediary villosity; 20. Amnion; 21. Perivascular connective tissue within chorial lamina; 22. Organized thrombus inside alanto-chorial artery; 25. Peduncular villous thrombosis; 26. Adherent thrombus onto peduncular artery wall; 27. Intimal pad. A – J : Macrophotographs taken with Canon EOS 1 ds Mark II Digital Camera. Macro Ultrasonic Lens, 100 mm, f/2,8. K – O: Paraffin sections. Hematoxyline Eosene Stain. Microphotographs taken with Nikon Sight DS-Fi1 Hight Definition Color Camera Head. x70 (L,M); x140 (K,N); x280 (0).

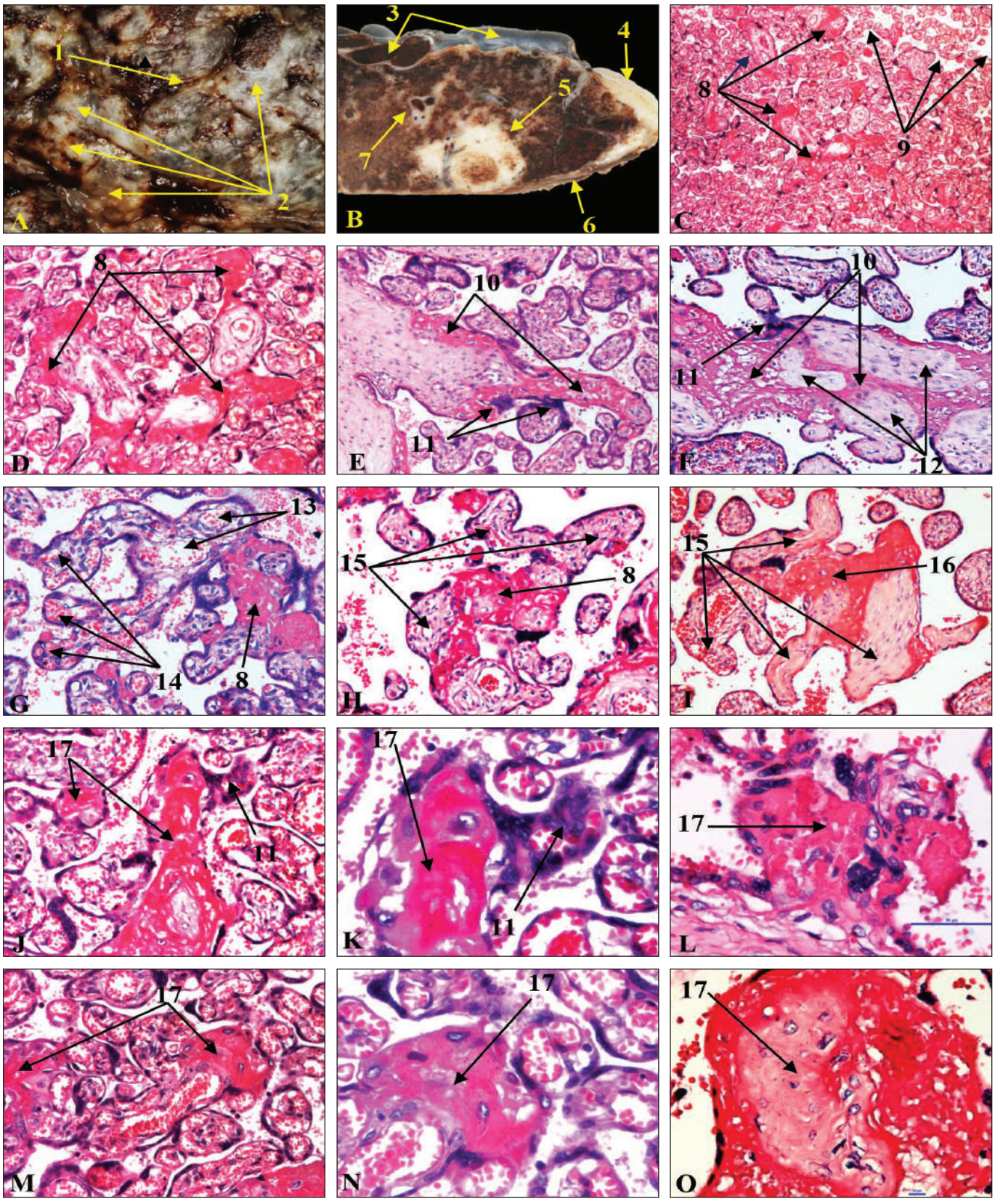


**Figure 2.** Maternal floor infarction (MFI) prin Massive fibrin deposition (MFD). 1. Interlobular ditch; 2. Fibrinoid onto the maternal face of placenta; 3. Alanto-chorial thrombosis; 4. Marginal location of fibrinoid; 5. Spheroid fibrinoid within pars basalis placentae; 6. Fibrinoid blade within pars basalis placentae; 7. Villus peduncularis thrombosis; 8. Intervillous fibrinoid with placental villi agglutination; 9. Colaps of intervillous space; 10. Intervillous fibrinoid; 11. Syncitial Knots; 12. Villous mesenchyme dissociated by fibrinoid fascicles; 13. Intermediary mature villi with vascular stasis; 14. Terminal villi with sinusoid capillaries stasis; 15. and 16. Intermediary mature villi aglutionation; 17. Avascular placenta villi with fibrinoid intravillous deposits. A,B : Macrophotographs taken with Canon EOS 1ds Mark II Digital Camera.Macro Ultrasonic Lens, 100 mm, f/2,8. C-O : Paraffin sections. Hematoxyline Eosene Stain. Microphotographs taken with Nikon Sight DS – Fi1 Hyight Definition Color Camera Head. x28 (C); x70 (D,G – J, M); X140 (E,F,K,N); x280 (L,O).

encephalopathy and/or convulsions. Anatomic-functional studies regarding determinant factors of blood flow perturbations within maternal-placenta and fetal-placenta circulatory systems led to the formation, in the last decades, of two type of morphologic syndromes: 1) "Massive fibrin deposition" (MFD) with implications in "Maternal floor infarction" (MFI) for maternal-placental circulatory system [1, 2] and 2) "Fetal thrombotic vasculopathy" (FTV) for fetal-placental circulatory system [5].

Blood flow perturbations within maternal-placenta circulatory system are determined by a perivillous fibrino substance accumulation. Benirschke (1961 [1], Benirschke and Driscoll 1967 [2]) nominated the presence of fibrin excess inside pars basalis placentae using the term "Massive basal plate fibrin deposition", with implications in "Maternal floor infarction". This concept was extended to pars chorionica [3] and placenta parenchyma, located between chorial and basal plates [4]. In the case of fibrinoid substance perivillous accumulation, as "laminate plates", in the infrachorial hystologic-topographic region, Fox [11] uses the term "Subchorionic fibrin plaque". For fibrin substance deposits around villosities inside placental parenchyma, Bernirschke and Kaufmann [4] created the term of "Perivillous fibrin deposition".

Blood flow perturbations within fetal-placental circulatory system are mainly determined by thrombosis of umbilical vessels and its branches. Redline and Pappin [5] proposed the term "Fetal thrombotic vasculopathy" for blood flow perturbations within umbilical, alanto-chorial, troncular and villous vessels. For this system, authors described six types of lesions: 1) partial or total vascular thrombosis; 2) avascular villosities; 3) fibrin deposits within blood vessels wall; 4) endothelial cushions associated with fibrinoid cushions within blood vessels intima; 5) hemorrhagic endovascularitis and 6) fiber-muscular sclerosis.

In this context, we consider that anatomic examination of placenta offers a support in the understanding of physiopathological processes within fetal-placental system, in confirmation or information of a diagnosis, in elaboration of a preventive strategy in recidivate pathology cases and last but not least, in evidence collection for forensic expertise, in order to approximate the age of acute or chronic lesions with the purpose to assess perinatal asphyxia and neurocongenital development perturbations in cases with medical responsiveness implications (L'Hermine Coulomb 2005 [14]; Boog 2001 [15]).

Macro- and microanatomic examination of placenta should become mandatory in order to evaluate fetal-maternal-placental biosystem (Cornelis 2008[16]; Redline *et al.* 2005 [17].

The main indications for the morphologic analysis of placenta are: 1) fetal (intrauterine growth retardation, severe oligoamnios, prematurity, premature membranes rupture for over 36 hours, congenital malformations, multiple pregnancies, spontaneous abortion, intrauterine fetal death); 2) newborn (newborn sepsis, perinatal asphyxia- Apgar score lower than 6, umbilical artery pH lower than 7, neurologic signs, newborn death); 3) maternal (infectious syndrome, hypertension, preeclampsia, last trimester metrorrhagia) and 4) placental (placenta acreta, size abnormalities, ultrasound abnormalities, suspicion of retroplacental hematoma).

## CONCLUSIONS

1. Three fundamental structures contribute to the development, in time and space, of fetal-placental biosystem functions, during its epigenesis dynamic: trophoblast, blood vessels and fibrin substance.

2. They represent the basis for the occurrence and evolution of three groups of pathologic processes: trophoblastic, vascular and fibrinoid.

3. Phenotype transformations of placenta fundamental structures are anatomic markers for embryo and fetus evolution or involution and they are highlighted by postpartum placenta examination – a real "black box of gestation".

4. Existence of reciprocal induction and interrelations between trophoblast, blood vessels and fibrin determined the occurrence of five new syndromes: "Fetal thrombotic vasculopathy", "Perivillous fibrin deposition", "Massive basal plate fibrin deposition", "Maternal floor infarction" and "Subchorionic fibrin plaque".

5. Elementary lesions discovered in the context of these syndromes, represent the basis for antepartum, intrapartum, postpartum and newborn pathological assessment in all cases involving medical responsiveness.

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