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Histopathological Findings in Placentas from Pregnancies Complicated with Preeclampsia

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Abstract

Pathological appearance in preeclampsia placenta giving us information on how the pathophysiology of placental insufficiency as a key role of this syndrome. This study aims to describe the placenta's histopathological features using hematoxylin and eosin staining procedure from block paraffin on placental tissue. An observational study of 51 placentas samples were collected from preeclampsia patients and stained with hematoxylin and eosin. Results were adjusted for patient's age at delivery, parity state, gestational age at delivery, and hypertension history before pregnancy. Out of 51 preeclampsia patients that mostly in age of 20-35 years old (64,7%), 34 of them (66,4%) are multiparity, 41 of them with gestational age at delivery in 37-42 weeks, 35 patients (68.6%) without prior history of having hypertension before pregnant and 16 patients (31,4%) with previously hypertension history before pregnant. Villous necrosis, increasing of syncytial knots, and thrombosis of spiralis artery were found in all of the 51 placental samples (100%) while calcification was found in 46 samples (90,2%). Placenta from patients with preeclampsia showed characteristic morphological features of fibrinoid necrosis of the villous, increased syncytial knots, calcification of the villous, and thrombosis of spiralis artery.

Keywords: Placental; preeclampsia; histopathological features.

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1. Introduction

Preeclampsia and eclampsia have been shown to have a global incidence, making them the third most common causes of maternal mortality globally [1]. In Indonesia, where the maternal mortality rate (MMR) is still rather high, hypertension is the fourth leading cause of maternal death [2].

Preeclampsia is a very complex disorder and involves multiple organ systems [3]. Preeclampsia has placental abnormalities that begin with insufficient cytotrophoblast cell invasion and ending with severe maternal endothelial dysfunction [4]. Production of placental anti-angiogenic factors are released into the maternal circulation and disrupt the maternal endothelium and result in hypertension, proteinuria, and the other systemic manifestations of preeclampsia [4]. These variables are thought to be the main reason why the placental mass cannot be sufficiently perfused by the normal placental blood supply, leading to a relative hypoperfusion [3]. According to numerous studies, maternal hypoperfusion causes the utero-placental flow of blood in hypertensive diseases of pregnancy to decrease. This also causes higher risk of preterm birth which has a significant mortality risk [5,6].

In order to determine how preeclampsia affects the placenta's morphology, a thorough study of the placenta is required. As a result, the purpose of this study focuses on the placenta's histological morphology in preeclampsia and reviews the clinical features of the preeclampsia patients in Makassar, South Sulawesi.

2. Materials and Methods

2.1. Collection of samples

The Ethics Committee of Hasanuddin University approved this descriptive study that was performed on the 51 samples of placenta from preeclampsia patients. The samples were collected from March 2018 to September 2018 from patients clinically diagnosed with preeclampsia. After delivery, the placenta samples fixed in 10% neutral buffered formalin, processed, sectioned and stained with Haematoxyllin and eosin. The histological features was examined using Olympus CX43 light microscopy by two experienced gynecologic pathologists.

2.2 Data Processing

SPSS 18 for Windows was used to process the data for this investigation. Descriptive statistical methods were used to describe the characteristics of the frequency data collected on age, parity, gestational age at delivery, history of chronic hypertension, and the histological findings in placenta.

3. Result

51 samples from preeclampsia placenta were investigated in the current investigation. The clinical characteristics data on age, parity, gestational age at delivery, chronic hypertension history are shown in table 1. The microscopic features of preeclampsia cases' placenta specifically villous stromal necrosis, the proliferation of trophoblastic cells by the increasing of syncytial knots, calcification, and thrombosis of spiral artery are shown on table 2.

Table 1: Baseline characteristic of preeclampsia patients (n=51).

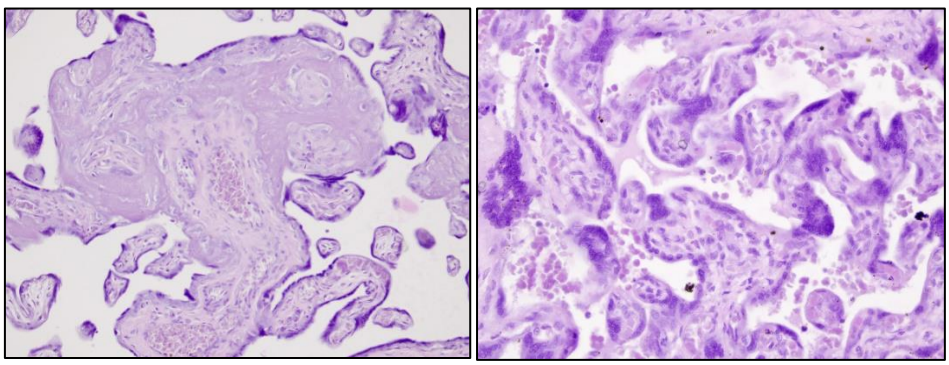
Sample Characteristics	Total (%)
Age	
<20 years	5 (9.8%)
20-35 years	33 (64.7%)
>35 years	13 (25.5%)
Parity	
Primigravida	15 (29.4%)
Multigravida	34 (66,7%)
Grand multiparity	2 (3.9%)
Gestational age at delivery (weeks)	
<37 weeks	10 (19,6%)
37-42 weeks	41 (80,4%)
Chronic Hypertension before pregnancy	
Has prior history	16 (31,4%)
Has no history	35 (68.6%)

Table 2: Histological findings of preeclampsia placenta.

Histological findings	Total (%)
Villous necrosis	51 (100%)
Increased syncytial knots	51 (100%)
Calcification	46 (90.2%)
Thrombosis of spiral artery	51 (100%)

Based on table 1, the prevalence of the preeclampsia patients in this study based on age were mostly in range 20-35 years, which was 33 patients (64,7%).

Five patients (9,8%) were in age <20 years and 13 patients (25,5%) were in age of >35 years. Parity status was mostly founded in multigravida patients account for 34 patients (66,7%), primigravida category in 15 patients (29,4%) and the grand multiparity category were 2 patients (3,9%). Based on gestational age at delivery, there were 10 patients (19,6%) in the category of <37 weeks and 41 patients (80,4%) in the category of 37-42 weeks. 16 patients (31,4%) have had prior chronic hypertension history before pregnancy, while 35 patients (68,6%) had no prior chronic hypertension history before pregnancy.



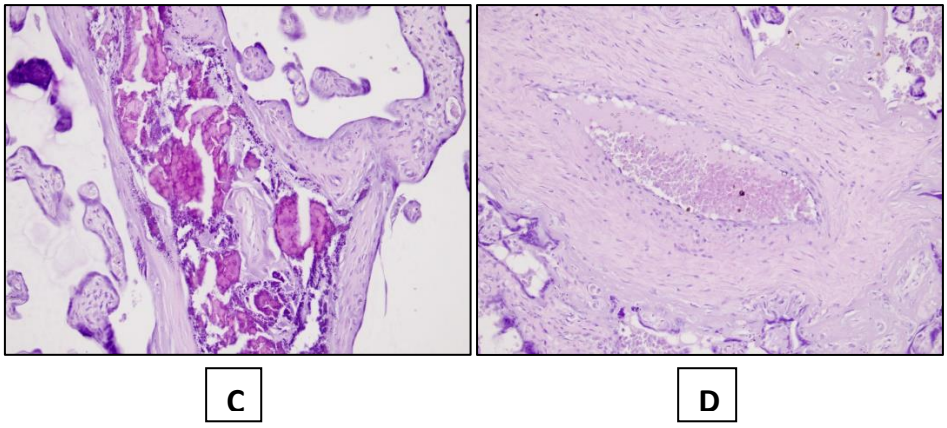


Figure 1: Histomorphological findings of preeclampsia placenta. Necrosis of the villous (Magnification 100x) (A). Increased syncytial knots (Magnification 400x). (B). Calcification of the villous (Magnification 100x) (C). Thrombosis of spiralis artery (Magnification 200x) (D).

The histopathological findings of preeclampsia placenta are shown in figure 1. All the 51 placenta samples (100%) shows villous stromal necrosis, the proliferation of trophoblastic cells by the increasing of syncytial knots, and thrombosis of spiral artery. While calcification was founded in 46 samples (90,2%).

4. Discussion

A total of 51 samples from preeclampsia patients were included in this study, with 33 patients (64,7%) in the 20- to 35-year-old age group representing the majority of the sample population. According to one epidemiological study, pregnant women older than 35 have a 4.5-fold increased probability of developing preeclampsia during pregnancy compared to pregnant women between the ages of 25 and 29 [7]. This is contrary to the findings of the study.

The majority of the pregnant women in this study's sample, which included up to 34 samples, (66,7%) had multigravida pregnancy status, and 15 samples (29,4%) had primigravida status. This is not in line with studies that conclude that the risk of preeclampsia is 3 times higher in primigravida [8].

According to one study, the risk of preeclampsia decreases as gestational age increases. It was determined that the risk of preeclampsia increased with younger gestational age [9]. Our study's results were different because we examined 41 samples from pregnant women whose gestational ages ranged from 37 to 42 weeks.

35 of the study's samples (68,6%) had no previous history of hypertension before pregnancy, whereas 16 of the samples (41,4%) had a previous hypertension history. This is not relevant with one study that conducted in Ethiopia, 2015, which suggests that a personal or family history of hypertension, old age, and a family history of diabetes are risk factors associated with preeclampsia [7]. Another study discovered that gestational hypertension, hypertension, and other pre-existing maternal illnesses were linked to a 22.3% rise in preeclampsia among other nulliparous pregnant women [10].

As one of the histopathological results, we discovered villous stromal necrosis in all of the placenta samples (n=51, 100%). This characteristic results from decreased fetal blood flow through the villi, and prolonged hypoperfusion conditions ultimately lead to ischemia and the obstruction of maternal arteries, which causes the surrounding villi to become infarcted [11]. Within the ischemic region, the intervillous space is initially decreased, and villi have a tendency to congregate and get clogged with dilated capillaries. After long time, villous structures are frequently referred to as "ghost villi" because they resemble the old villi's shadow [11].

The proliferation of trophoblastic cells by the increasing of syncytial knots were observed in all the placenta samples (n=51, 100%). This is consistent with a prior study that demonstrated how preeclampsia placenta is linked with the decreased of blood flow, which accelerates the growth of the placental villi and increases the formation of syncytial knots. Syncytial knots may be observed in mature placentas, although they are more common in preeclampsia placentas due to the placenta's exposure to hypoxia. Hypoxia alters the morphology of the tip or terminal villi and increases the risk of trophoblast accumulation, which may result in the formation of a syncytial knot [11,12].

The spiral arteries alter in preeclampsia due to insufficient perfusion of the placenta. In this situation, the placenta experiences hypoxia, oxidative stress, and ischemia, which lead to necrosis and infarction. The spiral arteries, which are the arteries supplying the villi, are affected by the irregular blood flow in preeclampsia. As a consequence of the villi's decreased oxygenation, smooth muscle cells proliferate and deposit extracellular matrix in the tunica intima, converting fat into fibrofatty atheroma and contributing to the progression of atherosclerotic lesions [13]. This is coherent with our study that observed all the placenta samples (n=51, 100%) with thrombosis of spiral arteries.

Calcification was founded 42 samples (90,2%). Stress pathways should also be considered as potential mediators of placental calcification. Preeclampsia is associated with an increased risk of cardiovascular disease later in life, although the reasons and consequences of placental calcification in placental malfunction and preeclampsia are unclear. Placental calcification may be a physiological response to a variety of stimuli that is created by both shared and unique processes. Both placental calcification and cardiovascular disease may be predisposed by maternal phenotypes and trigger mechanisms [14].

More research samples are required to be able to adequately describe the clinical and pathological relationships in preeclampsia placenta, which is another limitation of this study. Additional research is required, particularly into the specifics of clinical symptoms, associated laboratory tests, and continued observation of the status of the fetus delivered after delivery.

5. Conclusion

We draw the conclusion from this study that preeclampsia is more common in the 20–35 age range and more samples found in multigravida status. Younger gestational ages and prior histories of hypertension before pregnancy do not raise the risk of preeclampsia. Placenta from patients with preeclampsia showed characteristic morphological features of necrosis of the stromal villous, increased syncytial knots, calcification of the villous,

and thrombosis of spiralis artery. The placenta's morphology is adversely affected in preeclampsia. Understanding the precise cause of placental malfunction requires a thorough and quantitative evaluation of placental alterations. If this process is completely understood, more specific intervention tactics may be developed, which can lead to future medicines that are more successful.

6. Suggestions

Additional research is required to examine the proteins associated with morphological characteristics that may be connected to placenta ischemia variables, particularly to predict the likelihood for future preeclampsia pregnancies.

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