INTRODUCTION

Preeclampsia is systemically characterized by widespread vascular endothelial dysfunction and microangiopathy in the mother, but not in the fetus. At present it is believed that preeclampsia starts with abnormalities in the development of blood vessels in the placenta that cause an effect on the maternal endothelium [1, 4, 5]. Knowledge of cellular and molecular processes of human trophoblast invasion is based on in vitro research and animal models; there is evidence that decidual natural killer (dNK) cells are very important in successful placentation. They are key mediators of the interaction of the mother’s immune system with fetal cells. dNK cells are also involved in modulation of invasion of extravillous trophoblasts (EVT) and remodeling of the maternal spiral arteries. They express various surface receptors and signaling molecules, and their function in modulating EVT migration, invasion, and change from epithelial phenotype to endothelial are beginning to be revealed [6].

MATERIALS AND METHODS

This study is a comparative analytic study with a cross-sectional study. Sampling was done with a convenience method specifically by taking pregnant women with preeclampsia in severe stages and normal pregnancy (controls) at RSUD dr. Pirngadi Medan and Faculty of Medicine, University of North Sumatra Networking Hospital from November 2015 to April 2016. The sample of the study was 23 pregnant women suffering from severe preeclampsia and 33 pregnant women with a term gestational age that fulfilled the study criteria. The research subjects were pregnant women aged 18-35 years, singleton pregnancy, severe preeclampsia, and term and exclusion criteria ie damaged placenta samples; who came for pregnancy control to General Hospital H. Adam Malik Medan, RSUD dr. Pirngadi Medan, and Faculty of Medicine, University of North Sumatra Networking Hospital from November 2015 to April 2016. The sample of the study was 46 women, who met the inclusion criteria.

RESULTS

Characteristics of research subjects

Table 1: Distribution characteristics of research subjects in severe preeclampsia case group and normal pregnancy group

| Characteristics | Research subjects | % | Normal pregnancy | % | Total | % | p value |
|-----------------|------------------|---|-----------------|---|-------|---|---------|
| Age (years old) | Severe preeclampsia |  | Normal pregnancy |  |       |   |         |
| 15-25           | 3                | 13 | 8               | 34.8 | 11 | 23.9 | 0.062** |
| 26-35           | 16               | 69.6 | 15              | 65.2 | 31 | 67.4 |         |
| >35             | 4                | 17.4 | 0               | 0   | 4  | 8.7  |         |
| Parity          | Primigravida     | 7  | 30.4            | 5   | 8.7 |       |         |

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Table 2: Differences in dNK cell expression between severe preeclampsia case group and normal pregnancy group

| Research subjects | N  | CD56 expressions | p value |
|-------------------|----|------------------|---------|
|                   |    | Mean             | Std. deviasi |    |
| Severe Preeclampsia | 23 | 2.55             | 2.31    | 0.031 |
| Normal Pregnancy  | 23 | 8.66             | 3.16    |       |

Table 3: Differences in dNK cell expression between severe preeclampsia case group and normal pregnancy group based on Normal birthweigth and low birth weight (LBW)

| Birth weight | N  | CD56 expressions | p value |
|--------------|----|------------------|---------|
|              |    | Mean             | Std. deviasi |    |
| Normal       | 41 | 6.9451           | 5.20322  | 0.003 |
| LBW          | 5  | 2.4000           | 2.00468  |       |

Table 4: Differences in expression of dNK cells based on proteinuria

| Proteinuria | N  | CD56 expressions | p value |
|-------------|----|------------------|---------|
|             |    | Mean             | Std. deviasi |    |
| Negative    | 23 | 9.6957           | 4.24997  |       |
| (+2)        | 9  | 3.8889           | 4.90075  | 0.001 |
| (+3)        | 11 | 2.7045           | 2.93645  |       |
| (+4)        | 3  | 3.0000           | 3.46410  |       |

Fig. 1: Boxplot showing mean number of dNK cells in placenta with severe preeclampsia and normal pregnancy
CONFLICT OF INTERESTS

All the authors have contributed equally. Nil

AUTHORS CONTRIBUTIONS

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therapy in severe preeclampsia that in the future it can become a reference basis for immunological addition, researchers are aware of the need for further research so in this research. Beloved wife, Dr. Fithria Aldy, M. Ed (Oph), Sp. M, Alisha Hanifa Lubis, and M. Chairuddin Martua Lubis. May Allah

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CONCLUSION

Immunohistochemistry examination of dNK cell in severe preeclampsia case group generally gave a picture of expression with a mean of 2.55±2.31 while in the normal pregnancy control group had a higher mean with 8.66±3.16. Which showed significant

Immunohistochemistry examination results

The results of this study found that the results of immune-histochemistry dNK cells examination in the severe preeclampsia case group generally gave a description of expression with a mean of 2.55±2.31 while in the normal pregnancy control group had a higher mean with 8.66±3.16. Statistically obtained p value<0.05 which indicates there is a significant difference in the expression of immunohistochemistry dNK cells between severe preeclampsia case group and normal pregnancy group.

In another study by Charles et al. stated that dNK cells in women with preeclampsia were significantly less than controls (normotension). Closely related between dNK cells and blood vessels, dNK cells trigger angiogenic factors on the effects of IFN-γ to facilitate remodeling of normal blood vessels [57, 70, 71]. Rieger et al. in their study found that there was a significant relationship between CD56+/CD16+dNK cell counts that increased in the normal pregnancy group than in severe preeclampsia group (7.3% vs 5.3%, p<0.01) [58]. Williams et al. Observed that CD56+NK cells (p = 0.01) decreased in placental bearing biopsies in PB pregnancy women compared to normal third trimester pregnancy [59].

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AUTHORS CONTRIBUTIONS

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CONFLICT OF INTERESTS

There is no conflict of interest in this research.

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