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Correlation of Interleukin-33 and Imunoglobulin E with Risk of Asthma in Pregnant Woman

Harun Iskandar[1], Dewi Setiawati[2]

[1]Pulmonology Division Departement Internal Medicine of Hasanuddin University, Wahidin Sudirohusodo Hospital Makassar Indonesia

[2]Medical Faculty Alauddin State Islamic University Makassar Indonesia

ABSTRACT

Background: Interleukin-33 (Il-33) is involved in the pathogenesis of atopic asthma by activating Th2 cells. Immunoglobulin E (IgE) attached to mast cells and degranulation which cause bronchoconstriction, mucus secretion and vasodilation in asthma. The pathomechanism of asthma in pregnancy not clearly understood. The main of this study to evaluate the level of IL-33 and IgE in asthma pregnancy.

Method: This was a case control study conducted in Makassar Indonesia from November 2020 until June 2021. Interleukin 33 and IgE levels were analyzed from serum samples using the Enzyme-linked Immunosorbent Assay (ELISA) method and the level of asthma control determined with GINA Criteria

Result: Subject were 80 pregnant women: 40 subject with asthma in pregnancy and 40 non asthma. Subject of asthma in pregnancy were 22 uncontrolled, 18 well controlled asthma. The mean age was not significantly different between the subjects of pregnant women with asthma (28.025 $\pm$ 5.17 years) and subjects of non-asthmatic pregnant women (28.025 $\pm$ 4.26 years). Mean serum IL-33 levels were not higher in pregnant women with asthma than non-asthmatic pregnant women (1.77 vs 1.60 pg/mL, p=0.43) but mean serum IgE level were higher (529.8 vs 66.77 IU/mL, p=0.001). Mean serum IL-33 levels were not higher in uncontrolled asthma compared to well controlled (1.62 vs 1.98 pg/mL, p=0.44) but mean serum IgE levels were higher in uncontrolled asthma compared to controlled asthma (745.76 vs 264.6 IU/mL, p=0.01).

Conclusion: Levels of IL-33 serum were not higher asthma in pregnancy and also not different in level of asthma controlled, but levels IgE serum were higher in subjects with asthma in pregnancy and also uncontrolled asthma

KEYWORDS: Asthma in pregnancy, Il-33, IgE.

ARTICLE DETAILS

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BACKGROUND

Asthma in pregnancy is one of common respiratory problems in pregnant women with varies prevalence about 4-12% worldwide (Barman, 2013, Murphy et al. 2003; Wang et al. 2014).

The pathogenesis of asthma in pregnancy is not clearly understood. One mechanism that is thought to play a role is through Th2 inflammation and another theory via hormonal and elevation diagfragma due to pregnancy. Physiological pregnancy is a condition with a Th2 predominance to protect the fetus from the mother's immune response.

Interleukin 33 which is localized in the cell nucleus will be released when there is cell damage due to allergens and then will provoke local inflammation as alarmin. Interleukin 33 activates Th2 cell production by naive T cells through the IL-33 receptor (ST2). (Yagami et al. 2010) Interleukin (IL)-33 is one of the earliest-released signaling molecules following epithelial damage and can orchestrate the recruitment and activation of the cells responsible for asthma. IL-33 activity leads to activation of T-helper type 2 cells, mast cells, eosinophils, and basophils, ultimately leading to increased expression of cytokines and chemokines in asthma. (Larry et al 2011)

Interleukin 33 which is localized in the cell nucleus will be released when there is cell damage due to exposure to allergens and will then provoke local inflammation as alarmin. Cytokines secreted by Th2 cells will stimulate specific B lymphocytes against foreign antigens to differentiate into plasma cells which then produce IgE. Allergens will bind to IgE attached to mast cells and mast cell degranulation occurs. The degranulation releases histamine, proteases, leukotrienes and prostaglandins which cause mucus secretion, vasodilation and bronchoconstriction in the airways. (Thomas et al. 2001; Lee et al. 2009).

METHODS

The study was conducted in Makassar from November 2020 to June 2021. This was a cases control study with cases of 40 women with asthma in pregnancy and 40 controls of non-asthmatic pregnant women. Asma ditentukan dengan gejala klinis dan pemeriksaan spirometry serta tes bronchodilators. Derajat control asma ditentukan using GINA Criteria. Interleukin 33 and IgE levels were analyzed from serum samples using the Enzyme-linked Immunosorbent Assay (ELISA) method.

RESULTS

Subject were 80 pregnant women where 40 with asthma in pregnancy and 40 non asthma as subjects control.

Table 1. Characteristic of subjects

Variable	Pregnant woman with asthma in pregnancy n = 40	Pregnant woman non asthma n = 40	p value
Age (years)	28,025 ± 5.17	28,025 ± 4.26	p=0,96
Uncontrolled asthma	22	-	
Controlled asthma	18	-	

* t test

Table 2. Comparison of IL-33 and IgE levels asthma in pregnancy and pregnant without asthma

Levels	Pregnant woman with asthma in pregnancy n = 40	Pregnant woman non asthma n = 40	p
IL-33	1.77	1.60	0.43
IgE	529.27	66.7	0.001

In this study (table 2), it was found that the mean IL33 levels were not significantly different between asthma in pregnancy compared to non-asthma in pregnancy. The mean IgE level was significantly higher in asthma in pregnancy (529.27) compared to non-asthma in pregnancy (66.7) (p=0.001).

Table 3. Comparison of IL-33 and IgE levels in asthma in uncontrolled and non-asthmatic pregnancies pregnant

Levels	Asthma uncontrolled in pregnancy n=22	Asthma well controlled in pregnancy n=18	p
Kadar IL-33	1.62	1.98	0.44
Kadar IgE	745.76	264.6	0.01

t test

This study found (table 2) that IL-33 levels did not differ between uncontrolled asthma in pregnancy and controlled asthma in pregnancy (1.62 vs 1.98; p=0.44). Meanwhile, serum IgE levels were higher in uncontrolled asthma in pregnancy than in controlled asthma in pregnancy (745.76 vs 264.6 p=0.01).

Discussions

This study found that the mean IgE levels were significantly higher in asthma in pregnancy (529.27) compared to pregnant woman non asthma (66.7) (p=0.001). Amina et al reported the same thing where IgE was a predictive factor for asthma and its levels were higher in asthmatic subjects compared to non-asthmatic subjects. (Amine) Penelitian ini mendapatkan kadar IL-33 tidak berbeda antara kasus asma dalam kehamilan dan kontrol non-asma hamil (p=0,43).

Rui et al. reported a meta-analysis on 633 asthma subjects compared to 379 healthy subjects who found IL-33 levels were higher in severe asthma compared to mild asthma. Likewise, IL-33 levels are higher in asthma than non-asthmatics. IL-33 is expressed by several cells including mast and Th2 cells. IL-33 further activates eosinophils resulting in airway inflammation (Rui et al. 2014)

In pregnant women IL-33 is also expressed by the perivascular tissue. ST2 which is a soluble receptor can be released by the placenta due to hypoxia. The more hypoxia, the more IL-33 receptors will be released thereby increasing the inflammation that occurs (Ingrid et al. 2011). In this study, there were no differences in IL-33 levels between pregnant asthmatic and non-pregnant asthmatic subjects. This is possibly because the mechanism of asthma in pregnancy is multifactorial, not just through the IL-33 pathway found in atopic asthma. Besides that, IL33 is also influenced by the levels of IL-33 receptors, namely ST2, which is released by the placenta in pregnant women.

Comparison of Serum IL-33 and IgE Levels with Degree of Asthma Control in Pregnancy

The study found (table 3) that IL-33 levels did not differ between uncontrolled asthma in pregnancy and controlled asthma in pregnancy (1.62 vs 1.98; p=0.44). Meanwhile, serum IgE levels were higher in uncontrolled asthma in pregnancy than in controlled asthma in pregnancy (745.76 vs 264.6 p=0.01). Kittipong et al reported the same thing that serum IgE was significantly higher in uncontrolled atopic asthma than in control. (1075.4 + 420 vs 703.5 + 355 IU/mL, $p < 0.001$).

Based on the various analyzes above, it can be concluded that asthma in pregnancy is slightly different compared to atopic asthma in that there is an increase in IL33 in cases of atopic asthma. As a risk factor for asthma in pregnancy, it is not through the IL-33 and IgE pathways but possibly through other pathways, including hormonal factors

Tan et all in asthmatic subjects who received the hormone progesterone caused a reduction in the density and response of β2-AR and a reduction in the response of cyclin-AMP (Tan et al. 1997). Estrogen Stimulates Th2 Cytokine Production and Regulates the Compartmentalisation of Eosinophils during Allergen Challenge in Asthma (Cai Y et al 2012).

CONCLUSIONS

IgE levels were higher in pregnant women with asthma and uncontrolled asthma in pregnancy. However, IL-33 levels did not differ between pregnant women with asthma and those without asthma, nor did they differ based on the degree of asthma control.

Conflict of interest: Statement

The outhors declare no There was no conflict interest

Author's contributions: Harun Iskandar (concept, design, materials, sources, data processing, interpretation and analysis, literature research, and manuscript editing); Dewi Setiawaty (concept, planning, guidance, interpretation, analysis and literature research).

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