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Kajian Faktor Risiko Sindrom Ovarium Polikistik terkait Metabolisme Lemak: Fokus kepada Metilasi DNA dan Ekspresi mRNA Fat Mass And Obesity Associated (FTO) Gene serta Asupan Lemak = Study of Polycystic Ovarian Syndrome's Risk Factors Related to Fat Metabolism: Focusing on DNA Methylation and mRNA Expression of Fat Mass and Obesity associated (FTO) Gene and Fat Intake

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Abstrak

Sejumlah faktor risiko diketahui berperan dalam patofisiologi terjadinya SOPK, termasuk metabolisme lemak. Metilasi DNA Fat Mass And Obesity Associated (FTO) Gene sebagai salah satu faktor epigenetik dipikirkan bertanggung jawab dalam patofisiologi SOPK. Namun hingga saat ini belum ada studi yang meneliti metilasi DNA gen FTO sebagai faktor risiko SOPK. Oleh karena itu, penelitian ini bertujuan untuk mengkaji faktor risiko SOPK terkait metabolisme lemak, terutama fokus pada metilasi DNA dan ekspresi mRNA gen FTO, serta asupan lemak. Dilakukan studi potong lintang pada 80 responden yang terbagi menjadi 4 kelompol fenotip, yaitu SOPK obese, kontrol obese, SOPK nir-obese, dan kontrol nir-obese. Pemeriksaan dilakukan pada sejumlah variabel seperti usia, indeks masa tubuh, kadar testorteron, SHBG, nilai FAI, tingkat metilasi DNA dan ekspresi mRNA gen FTO, serta asupan harian lemak (lemak total, PUFA, asam lemak jenuh, asam lemak tidak jenuh) yang berasal dari Food Frequency Questionaire, kemudian dianalisis secara statistik. Didapatkan perbedaan bermakna pada variabel usia, IMT, kadar SHBG dan tingkat metilasi DNA gen FTO pada keempat fenotip SOPK ($p<0,05$). Tidak terdapat perbedaan bermakna pada variabel kadar testosteron, FAI, ekspresi mRNA gen FTO, asupan lemak, PUFA, asam lemak jenuh dan asam lemak tidak jenuh pada keempat fenotip ($p>0,05$). Tingkat metilasi DNA gen FTO pada fenotip SOPK obese (6,47%) lebih rendah dibandingkan dengan kontrol obese (16,77%) sehingga dapat disimpulkan bahwa fenotip SOPK obese mengalami hipometilasi. Sedangkan, tingkat metilasi DNA pada SOPK nir-obese (13,08%) lebih tinggi dibandingkan dengan kontrol nir-obese (6,02%) sehingga SOPK nir-obese mengalami hipermetilasi. Selain itu, hasil uji multivariat menunjukkan bahwa SHBG dan tingkat metilasi DNA merupakan faktor risiko dari SOPK pada kelompok nir-obese. Didapatkan kenaikan tingkat metilasi DNA gen FTO meningkatkan risiko kejadian SOPK pada kelompok nir-obese sebesar 1,085 kali. Sehingga dapat disimpulkan bahwa perubahan tingkat metilasi DNA gen FTO dan SHBG dapat berperan sebagai marka biologis tatalaksana SOPK dengan IMT normal dalam aplikasi klinis.
.....A number of risk factors are known to play a role in the pathophysiology of PCOS, including fat metabolism. DNA methylation of FTO gene, as an epigenetic factor, is thought to be responsible for the pathophysiology of PCOS. However, until now there has been no research examining the DNA methylation of the FTO gene as a risk factor for PCOS. Therefore, this study aims to investigate the risk factors for PCOS related to fat metabolism, especially focusing on DNA methylation and mRNA expression of the FTO gene, as well as daily fat intake. A cross-sectional study was conducted on 80 respondents who were divided into 4 phenotypic groups, namely obese PCOS, obese controls, non-obese PCOS, and non-obese controls. The examination was carried out on a number of variables such as age, body mass index, testosterone levels, SHBG, FAI values, DNA methylation levels and mRNA expression of the FTO gene, as

well as daily intake of fat (total fat, PUFA, saturated fatty acids, unsaturated fatty acids) derived from from the Food Frequency Questionnaire. Statitic analysis was performed. There were significant differences in the variables of age, BMI, SHBG levels and DNA methylation levels of the FTO gene in the four PCOS phenotypes ($p<0.05$). There were no significant differences in the variables of testosterone levels, FAI, FTO gene mRNA expression, fat intake, PUFA, saturated fatty acids and unsaturated fatty acids in the four phenotypes ($p>0.05$). The level of DNA methylation of the FTO gene in the obese PCOS phenotype (6.47%) was lower than the obese control (16.77%) so it can be concluded that the obese PCOS phenotype was hypomethylated. Meanwhile, the level of DNA methylation in non-obese PCOS (13.08%) was higher than non-obese controls (6.02%) so that the non-obese PCOS phenotype was hypermethylated. In addition, multivariate test results showed that SHBG and DNA methylation levels were risk factors for PCOS in the non-obese group. It was found that an increase in the level of DNA methylation of the FTO gene increased the risk of PCOS in the non-obese group by 1.085 times. Therefore, it can be concluded that the changes in DNA methylation levels of FTO gene and SHBG might be useful as biological markers for the management of PCOS with normal BMI in clinical applications.