F2α-isoprostane, Na+-K+ ATPase and membrane fluidity of placental syncytiotrophoblast cell in preeclamptic women with vitamin E supplementation

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Abstrak

Latar belakang: Penelitian ini bertujuan untuk menganalisis kadar F2α-isoprostan, aktivitas enzim Na+-K+ ATPase dan fluiditas membran sel sinsitiotrofoblas plasenta penderita pre-eklampsia yang diberi vitamin E.
Metode: Penelitian dilakukan pada bulan September 2003 ? Februari 2005 di Rumah Sakit Bersalin Budi Kemuliaan, Jakarta Pusat. Sampel penelitian adalah 6 wanita pre-eklampsia yang mendapatkan vitamin E, 6 wanita pre-eklampsia yang tidak mendapat vitamin E dan 6 wanita hamil normal. F2α-isoprostan diukur dengan ELISA Reader pada λ = 450 nm. Fluiditas diukur dengan membandingkan rasio molar kolesterol total dan kadar fosfolipid membran sel. Kolesterol diukur menggunakan Modular C800 dengan reagen Roch. Fosfolipid diukur menggunakan spektrofluorometer Shimadzu RF5301PC dengan filter eksitasi 267 nm dan emisi 307 nm. Aktivitas Na+-K+ ATPase dihambat dengan ouabain. Produksi Pi diukur dengan metode Fiske dan Subbarow menggunakan spektrofotometer pada λ = 660 nm. Data dianalisis menggunakan uji F melalui ANOVA 1 arah.
Hasil: Pemberian vitamin E pada penderita pre-eklampsia menurunkan stres oksidatif dengan indikasi turunnya F2α-isoprostan secara bermakna (26,72 ± 11,21 vs 41,85 ± 7,09 ng/mL, p = 0.017). Vitamin E mampu menangkal radikal bebas sehingga peroksidasi fosfolipid dapat dihambat dan fluiditas membran sel dapat dipertahankan pada 0,39 ± 0,08 dibandingkan tanpa pemberian vitamin E yaitu 0,53 ± 0,14 (p = 0,024). Aktivitas enzim Na+-K+ ATPase membran sel sinsitiotrofoblas tidak dipengaruhi oleh vitamin E (p = 0,915).
Kesimpulan: Suplementasi vitamin E pada wanita pre-eklampsia menurunkan kadar F2α-isoprostan, mempertahankan fluiditas membran sel, namun tidak meningkatkan aktivitas enzim Na+-K+ ATPase sel sinsitiotrofoblas.
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Abstract
Background: The aim of our study was to analyze F2α-isoprostane level, Na+-K+ ATPase activity and placental syncytiotrophoblast cell membrane fluidity in preeclamptic women who received vitamin E supplementation.
Methods: The study was conducted between September 2003 and February 2005 at Budi Kemuliaan Maternity Hospital, Central Jakarta. Samples were 6 preeclamptic women with vitamin E supplementation, 6 preeclamptic women without vitamin E supplementation and 6 normal pregnant women. The dose of vitamin E was 200 mg daily. F2α-isoprostane was measured with ELISA reader at λ of 450 nm. Cell membrane fluidity was measured by comparing the molar ratio of total cholesterol and cell membrane phospholipid concentration. The cholesterol was measured by Modular C800 using Roche reagent. Phospholipid was measured by Shimadzu RF5301PC spectrofluorometer (excitation 267 nm, emission 307 nm). Na+-K+ ATPase activity was inhibited by ouabain. Pi production was measured with Fiske and

Subbarow method using spectrophotometer at λ of 660 nm. Data was analyzed using F test with one-way ANOVA.
Results: Vitamin E supplementation in preeclamptic women decreased the oxidative stress, indicated by significantly lower level of F2α-isoprostane compared to those without vitamin E (26.72 ± 11.21 vs 41.85 ± 7.09 ng/mL, respectively, p = 0.017). Membrane fluidity in syncytiotrophoblast cell of preeclampsia with vitamin E group was maintained at 0.39 ± 0.08 while in those without vitamin E was 0.53 ± 0.14 (p = 0.04). Na+-K+ ATPase activity in syncytiotrophoblast cell membrane was not affected by vitamin E (p = 0.915).
Conclusion: Vitamin E supplementation in preeclamptic women decreases F2α-isoprostane level and maintains cell membrane fluidity of syncytiotrophoblast cells; however, it does not increase Na+-K+ ATPase enzyme activity.