

Kadar 2,3-dinor-6-keto-prostaglandin-F1, dalam urin wanita pascamenopause alami dan premenopause yang minum aspirin 100mg = Urinary 2,3-dinor-6-keto-prostaglandin-fia in healthy postmenopausal women and premenopausal women receiving aspirin 100 mg

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Abstrak

Prevalensi penyakit kardiovaskular meningkat dengan tajam pada wanita pasca menopause. Pada wanita pasca menopause terjadi peningkatan produksi trombosit dan penurunan produksi prostasiklin. Aspirin dosis rendah (75 - 150 mg) telah lama dikenal sebagai penghambat agregasi trombosit. Aspirin bekerja dengan menghambat produksi tromboksan (suatu zat proagregasi trombosit dan vasokonstriktor poten) serta produksi prostasiklin (suatu zat antiagregasi trombosit dan vasodilator poten).

Studi ini merupakan uji klinik tidak tersamar dengan 2 kelompok paralel. Kelompok pertama terdiri dari 15 orang wanita premenopause (34-40 tahun) dan kelompok kedua 15 orang wanita pasca menopause yang telah henti haid selama 3 - 5 tahun. Urin 24 jam dikumpulkan dari setiap subyek sebelum dan sesudah minum aspirin 100 mg selama 7 hari berturut-turut. Kadar prostasiklin dalam urin dalam bentuk metabolitnya, 2,3-dinor-6-keto-prostaglandin-F1.1, dianalisis menggunakan metode EIA (Enzyme immunoassay).

Tromboksan, dalam bentuk metabolitnya (11-dehidro-tromboksan-B2), juga diukur dalam sampel urin ini pada studi terdahulu.

Studi terdahulu menunjukkan bahwa aspirin menurunkan kadar tromboksan secara bermakna pada kedua kelompok dengan persentase penurunan yang lebih besar secara bermakna pada wanita pasca menopause dibandingkan wanita premenopause. Hasil studi ini menunjukkan bahwa aspirin menurunkan kadar prostasiklin secara bermakna pada wanita premenopause (selisih = 78,44 ng/g kreatinin; $p = 0,001$) maupun wanita pasca menopause (selisih = 35,71 ng/g kreatinin; $p < 0,001$), namun persentase penurunan antara kedua kelompok tidak berbeda bermakna (46,26% vs 40,94%; $p = 0,574$). Penurunan kadar tromboksan dan prostasiklin oleh aspirin perlu dibandingkan (dalam bentuk penurunan rasio kadar 11-dehidro-tromboksan-B2 / 2,3-dinor-6-keto-prostaglandin-Fu, dalam urin) untuk menilai efikasi aspirin sebagai antitrombotik. Perhitungan rasio kadar 11-dehidro-tromboksan-B2 / 2,3-dinor-6-keto-prostaglandin-Fm sebelum pemberian aspirin jauh lebih tinggi pada wanita pascamenopause dibandingkan wanita premenopause (4,09 vs. 1,13; $p = 0,001$). Penurunan rasio kadar 11-dehidro-tromboksan-Bd 2,3-dinor-6-keto-prostaglandin-Fm oleh aspirin jauh lebih besar pada wanita pasca menopause dibandingkan wanita premenopause (1,91 vs. 0,17; $p = 0,022$).

Dengan demikian disimpulkan bahwa aspirin menurunkan kadar prostasiklin secara bermakna pada masing-masing kelompok dengan persentase penurunan yang tidak berbeda antara kedua kelompok, namun menurunkan rasio kadar 11-dehidro-tromboksan-BJ 2,3-dinor-6-keto-prostaglandin-F1., yang jauh lebih besar pada wanita pasca menopause dibandingkan pada wanita premenopause.

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The prevalence of cardiovascular diseases in women increases sharply after menopause. In postmenopausal women, thromboxane production increases while prostacyclin production decreases. Low dose aspirin (75 - 150 mg) has long been known as an antiplatelet aggregator. Aspirin reduces the production of both thromboxane (potent thrombocyte aggregator and vasoconstrictor) and prostacyclin (anti thrombocyte aggregator and potent vasodilator).

The present study was an open-label clinical trial with 2 parallel groups. One group consisted of 15 premenopausal women (age 20-40 years) while the other group 15 postmenopausal women (for 3 - 5 years). Twenty-four hours urine was collected from each subject before and after aspirin 100 mg daily for 7 days. The concentration of prostacyclin was measured as its metabolite (2,3-dinor-6-keto-prostaglandin-F_{2α}) in urine using EIA (Enzyme immunoassay). Thromboxane as its urinary metabolites (11-dehydro-thromboxan-B₂) was also measured in these same urine samples in the previous study.

Previous study showed that aspirin significantly reduced thromboxane in both groups, with significantly larger percentage reduction in postmenopausal women compared to premenopausal women. Results of the present study showed that aspirin reduced prostacyclin significantly in both premenopausal women (mean difference = 78.44 ng/g creatinine; p = 0.001) and postmenopausal women (mean difference = 35.71 ng/g creatinine; p < 0.001), but the percentage reduction between the groups was not significantly different (46,26% vs. 40,94%; p = 0,574). The decrease in thromboxane and prostacyclin should be compared (as the decrease in the ratio of 11-dehydro-thromboxan-B₂ / 2,3-dinor-6-keto- prostaglandin-F_{2α}) to assess aspirin efficacy as an antithrombotic. Calculation of the ratio of 11-dehydro-thromboxan-B₂ / 2,3-dinor-6-keto- prostaglandin-F_{2α}, before aspirin consumption was much higher in postmenopausal women compared to that in premenopausal women (4.09 vs. 1.13; p = 0.001). The decrease in 11-dehydro-thromboxan-B₂ / 2,3-dinor-6-keto- prostaglandin-F_{2α} ratio by aspirin was found much larger in postmenopausal women compared to that in premenopausal women (1.91 vs.0.17; p = 0.022).

It was concluded that aspirin reduced prostacyclin significantly in each group with nonsignificant percentage reduction between groups, but reduced the 11-dehydro-thromboxan-B₂ / 2,3-dinor-6-keto- prostaglandin-F_{2α} ratio much larger in postmenopausal women compared to that in premenopausal women.