Pengaruh asam asetilsalisilat dosis tunggal terhadap agregasi trombosit pada orang sehat = Effect of single dose of acetylsalicylic acid on healthy human platelet aggregation

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Abstrak

ABSTRAK
Ruang Lingkup dan Cara Penelitian: Kemampuan asam asetilsalisilat (ASA) dalam menghambat agregasi trombosit, sering dikaitkan dengan pencegahan infark jantung. Dewasa ini, dalam upaya menurunkan resiko terjadinya infark jantung, ada kecenderungan menggunakan ASA dengan dosis makin kecil. Sehubungan dengan itu, dilakukan penelitian yang bertujuan untuk mengetahui berapa lama, dan apakah ada perbedaan yang bermakna antara intensitas antitrombotik beberapa tingkat dosis ASA (50 mg, 100 mg, 200 mg, dan 300 mg). Kemampuan agregasi trombosit diukur dengan metode baru yang berdasarkan intensitas transmisi cahaya. Hasil pemeriksaan tercermin sebagai suatu kurva agregasi trombosit. Disain yang dipakai adalah rancangan pola silang, dengan 11 orang sukarelawan sehat yang setelah diacak, masing-masing mendapat 4 tingkat dosis ASA dengan selang waktu 2 minggu. Bahan pemeriksaan terdiri dari 'platelet rich plasma', 'platelet poor plasma' dan adenosin difosfat yang berkadar akhir 10 uM, sebagai agregator. Parameter hambatan agregasi trombosit adalah berkurangnya nilai agregasi maksimal dan atau meningkatnya reversibilitas kurva agregasi trombosit, disbanding nilai sebelum mendapat ASA. Data dianalisis dengan ANOVA dua arah dan 'Planned comparison'. Untuk data dengan distribusi tidak normal, dipakai tes non parametrik (tes Friedman).

Hasil dan Kesimpulan: Bila berdasarkan adanya salah satu parameter hambatan agregasi trombosit, maka ASA 50 mg, 100 mg, dan 200 mg per oral dapat menghambat agregasi trombosit selama 4 hari, sedangkan ASA 300 mg selama 5 hari (p > 0,01). Namun bila berdasarkan adanya kedua parameter hambatan agregasi trombosit, maka ASA 50 mg dapat menghambat agregasi trombosit pada 3 jam sesudah pemberian obat, sedangkan ASA 100 mg dan 200 mg, sampai 4 hari sesudah pemberian ASA. Intensitas antitrombotik ke empat dosis ASA, pada hari yang sama setelah makan obat, tidak menunjukkan perbedaan yang bermakna (p}0,07). Untuk menyatakan hambatan agregasi trombosit, kriteria peningkatan reversibilitas kurva agregasi lebih peka di-banding kriteria pengurangan nilai agregasi maksimal.
<hr><i>ABSTRACT
Scope and Method of Study: The ability of acetylsalicylic acid (ASA) to inhibit the platelet aggregation is related with its use to the prevention myocardial infarction. Currently there is a trend to use small doses of ASA for this purpose. In this context, the present trial was conducted to find out how long the antithrombotic effect persist after small oral doses of ASA, and also to observe whether in the same days different small doses of ASA exert significant difference in their anti-thrombotic intensity. The antithrombotic effect of ASA was measured according to the method described by Born which was based on light transmission. The results were recorded as platelet aggregation curve. Eleven healthy volunteers participated in this trial after giving their' informed consents. Each subject received single doses (i.e. 50, 100, 200 and 300 mg) of ASA in a randomized and cross-over design. Wash out period between doses was 2

weeks. Materials being tested included platelet rich plasma, platelet poor plasma and adenosine diphosphate (aggregating agent) with final concentration of 10 uM. Inhibition of platelet aggregation by ASA was evaluated using two parameters, i.e. decrease of maximal aggregation and/or increase of aggregation curve's reversibility (compared to their pre-ASA values). Data was analysed with two way ANOVA and planned comparison test. Friedman test was used for non-Gaussian data.

Results and conclusions: If criterion of platelet aggregation inhibition is based on one of the two criteria mention above, ASA 50, 100, and 200 mg inhibited platelet aggregation for four days; meanwhile the 300 mg dose did it for five days ($p < 0,01$). If criterion of platelet aggregation inhibition is based on both of the above mentioned criteria, however, ASA 50 mg inhibited plate-let aggregation at 3 hours after dosing; meanwhile the 100 and 200 mg doses did it for four days. There is no significant difference in antithrombotic intensity between the four doses in the same days after drug administrations ($p > 0,01$). In addition, reversibility of platelet aggregation curve is a more sensitive parameter than maximal aggregation for measuring platelet aggregation.</i>