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论文

吲哚布芬对血小板聚集及血栓形成的影响

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摘要：

目的 通过动物实验，研究注射剂型吲哚布芬静脉给药后对血小板聚集及血栓形成的影响。方法 每项实验用动物50只，分5组，每组10只，动物按体质量随机分为生理盐水对照组（NS组）、吲哚布芬低、中、高剂量组及阿司匹林（ASA组）对照组，药后30min，分别检测由二磷酸腺苷（ADP）、花生四烯酸（AA）及血小板活化因子（PAF）诱导的Wistar大鼠血小板1min、5min的聚集率及最大聚集率;胶原-肾上腺素诱发的KM小鼠血栓性偏瘫甚至死亡的时间及电刺激Wistar大鼠颈动脉血栓形成的时间。结果 大鼠血小板聚集实验显示，与NS组比较，吲哚布芬中、高剂量组和ASA组能抑制由ADP、AA及PAF诱导的大鼠血小板不同时间点的聚集（P<0.05或P<0.01），与ASA组比较，在ADP及AA诱导的实验中，吲哚布芬高剂量组对血小板1min/5min聚集率的抑制作用有统计学差异（P<0.05）；胶原-肾上腺素诱导的小鼠肺栓塞实验显示，与NS组比较，吲哚布芬中、高剂量组及ASA组小鼠存活时间明显延长（P<0.01或P<0.05），与ASA组比较，吲哚布芬高剂量组的小鼠存活时间延长有统计学差异(P<0.05)；电刺激大鼠颈动脉血栓形成实验显示，与NS组比较，吲哚布芬中、高剂量组和ASA 组均能明显抑制血管栓塞时间（P<0.05或P<0.01），与ASA组比较，吲哚布芬抑制血管栓塞时间虽无统计学差异，但吲哚布芬高剂量组抑制血栓形成时间明显高于ASA组。结论 吲哚布芬具有显著的抑制血小板聚集和血栓形成作用，且呈现一定的量效关系，吲哚布芬抑制血小板聚集及血栓形成作用强于ASA。

关键词： 吲哚布芬；血小板聚集；血栓形成；大鼠，Wistar；小鼠，KM

Indobufen on platelet aggregation and thrombosis

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Abstract:

Objective To investigate the effects of Indobufen on platelet aggregation and thrombosis in animal models. Methods In each study, 50 animals were randomly divided into five groups, i.e. group NS, group L(low concentration), group M(medium concentration)， group H (high concentration) of Indobufen and group ASA (Aspirin, positive control）, according to their body weight. After being treated intravenously for 30 minutes, one-minute and five-minute platelets′ aggregation rate and the maximum aggregation rate, which were induced by adenosine diphosphate （ADP）, arachidonic acid （AA）or platelet activating factor（PAF）, were assayed. Moreover, the time of thrombotic paralysis or even death caused by collagen-epinephrine and the time of carotid artery thrombosis caused by electrical stimulation during the whole process were tested, too. Results The platelet aggregation study showed that Indobufen group of M, H and ASA 22group, compared with group NS, demonstrated the ability to inhibit ADP, AA and PAF-induced platelet aggregation at different time points（P<0.05 or P<0.01）. In the inducement study of ADP and AA, compared with group ASA, Indobufen group of H showed a significant discrepancy in marked inhabitation of the one-minute/five-minute platelet aggregation rate（P<0.05）. Based on the pulmonary embolism study induced by collagen adrenaline in mice, the survival time of the mice in the Indobufen groups of M， H and group ASA increased much more than group NS（P<0.01 or P<0.05）. Meanwhile, the survival time of group H was considerably longer than group ASA（P<0.05）. According to the results of the carotid artery thrombosis study, Indobufen groups of M，H and group ASA could significantly inhibit the thrombosis time（P<0.05 or P<0.01）， compared with group NS. Although the thrombosis time inhibited by Indobufen did not show a significant difference, Indobufen group of H presented a longer time to inhibit thrombosis than group ASA. Conclusion Indobufen is a very potential inhibitor of platelet aggregation and thrombus formation in vivo, and the effect is dose-dependent. To inhibit the

platelet aggregation and thrombosis, Indobufen is better than ASA.
Keywords: Indobufen; Platelet aggregation; Thrombosis; Rats， Wistar; Mice， KM

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