

against further adjustments. Conclusions- PAF-AH activity is not an indicator of the systemic inflammation that accompanies acute coronary syndromes. PAF-AH activity is affected by a number of cardiovascular drugs; however, after such medication use was accounted for, PAF-AH activity was associated with angiographic CAD, complementary to sCRP and independently of established risk factors such as LDL cholesterol.

血小板活化因子乙酰水解酶活性可独立于全身炎症和其他危险因素提示血管造影阳性冠状动脉疾病：
Ludwigshafen 风险和心血管健康状况研究

背景：血小板活化因子乙酰水解酶 (PAF-AH)，也称脂蛋白相关性磷脂酶 A₂，是可能参与炎症和动脉粥样硬化过程的脂蛋白结合酶。本研究探讨 PAF-AH 活性与血管造影阳性冠状动脉疾病 (CAD)、心血管药物的应用以及其他已知危险因素的关系。方法和结果：测定 2454 例经血管造影证实的 CAD 患者和 694 例对照者的 PAF-AH 活性、脂蛋白、高敏 C 反应蛋白 (sCRP)、纤维蛋白原、血清淀粉样蛋白 A 和白细胞计数。PAF-AH 活性与 LDL 胆固醇 ($r=0.517$)、载脂蛋白 B ($r=0.644$) 和非 HDL 胆固醇 ($r=0.648$) 高度相关，但与 sCRP 和纤维蛋白原无关。女性 PAF-AH 活性较男性低，且受降脂药 (-12% ; $P < 0.001$)、阿司匹林 (-6% ; $P < 0.001$)、 β 受体阻断剂 (-6% ; $P < 0.001$) 和洋地黄 (+7% ; $P < 0.001$) 的影响。与 sCRP、纤维蛋白原和血清淀粉样蛋白 A 不同，PAF-AH 活性在不稳定型心绞痛、非 ST 段抬高心肌梗死或 ST 段抬高心肌梗死时不升高。检测未使用降脂药者时，PAF-AH 活性与 CAD 的严重程度及显著狭窄的冠状动脉数目相关。对未使用降脂药物者，经校正阿司匹林、 β 受体阻断剂和洋地黄的使用后，CAD 与 PAF-AH 活性增高相关的优势比为 1.39 (95% CI 1.26 ~ 1.54, $P < 0.001$)，经进一步校正后更明显。结论：PAF-AH 活性不是伴有急性冠状动脉综合症的全身炎症的标志。PAF-AH 活性受很多心血管药物的影响；然而，本研究表明，经相关药物治疗后，作为对 sCRP 的补充，PAF-AH 活性与血管造影阳性 CAD 相关，而独立于诸如 LDL 胆固醇的已知危险因素。

0575. Aliskiren, a novel orally effective renin inhibitor, provides dose-dependent antihypertensive efficacy and placebo-like tolerability in hypertensive patients

Gradman A. H. / Schmieder R. E. / Lins R. L. et al.
[Dr. M. P. Bedigian, Cardiovasc. and Metab. Clin.

Res., Novartis, One Health Plaza, East Hanover, NJ 07936, United States] - CIRCULATION 2005, 111/8 (1012-1018)

Background- Stopping the detrimental effects of the renin-angiotensin system at the most upstream point of the cascade offers theoretical advantages for cardiovascular protection. This study compares the antihypertensive efficacy and safety of the novel oral renin inhibitor aliskiren with placebo and an active comparator. Methods and Results- The study was a randomized, multicenter, double-blind, placebo-controlled, active-comparator 8-week trial in patients with mild-to-moderate hypertension (mean sitting diastolic blood pressure [DBP] ≥ 95 and < 110 mm Hg). After a 2-week, single-blind placebo run-in, 652 patients were randomized to receive double-blind treatment with once-daily oral doses of aliskiren (150, 300, or 600 mg), irbesartan 150 mg, or placebo. Aliskiren 150, 300, and 600 mg effectively lowered both trough mean sitting DBP and systolic blood pressure (SBP) ($P < 0.001$ versus placebo for both variables). The least-squares mean reductions in trough DBP were 9.3 ± 0.8 , 11.8 ± 0.8 , and 11.5 ± 0.8 mm Hg, respectively, versus 6.3 ± 0.8 mm Hg for placebo, and the least-squares mean reductions in trough SBP were 11.4 ± 1.3 , 15.8 ± 1.2 , and 15.7 ± 1.2 mm Hg, respectively, versus 5.3 ± 1.2 mm Hg for placebo. The antihypertensive effect of aliskiren 150 mg was comparable to that of irbesartan 150 mg (8.9 ± 0.7 and 12.5 ± 1.2 mmHg, least-squares reduction in mean sitting DBP and SBP, respectively, for irbesartan). Aliskiren 300 and 600 mg lowered mean sitting DBP significantly more than irbesartan 150 mg ($P < 0.05$). Aliskiren showed safety and tolerability comparable to those of placebo and irbesartan; the incidence of adverse events and number of patients discontinuing therapy were similar in all groups. Conclusions- Once-daily oral treatment with aliskiren lowers blood pressure effectively, with a safety and tolerability profile comparable to that of irbesartan and placebo, in patients with mild-to-moderate hypertension. Aliskiren 150 mg is as effective as irbesartan 150 mg in lowering blood pressure.

新型口服肾素抑制剂阿利吉仑对高血压患者有剂量依赖性抗高血压作用及与安慰剂相当的耐受性

背景：从理论上讲，在级联反应的源头阻断肾素血管紧张素系统不良效应有利于保护心血管系统。本研究比较了新型口服肾素抑制剂阿利吉仑和安慰剂及一种

活性对比药物的抗高血压安全性和有效性。方法和结果:本试验采用随机、多中心、双盲、安慰剂对照和活性药物比较的方法,对轻中度高血压患者[平均坐位舒张压(DBP) ≥ 95 mmHg、 < 110 mmHg]进行为期8周的研究。经第一个2周单盲安慰剂导入期后,652例患者被随机分配接受双盲治疗——阿利吉仑(150 mg、300 mg或600 mg)、依贝沙坦(150 mg)或安慰剂每日一次口服。阿利吉仑150 mg、300 mg和600 mg可以明显降低平均坐位DBP和收缩压(SBP)的谷值(两变量与安慰剂相比, P 均 < 0.001)。用最小二乘法计算,DBP谷值的降低值分别为 9.3 ± 0.8 mmHg、 11.8 ± 0.8 mmHg和 11.5 ± 0.8 mmHg,安慰剂为 6.3 ± 0.8 mmHg;SBP谷值的降低值分别为 11.4 ± 1.3 mmHg、 15.8 ± 1.2 mmHg和 15.7 ± 1.2 mmHg,安慰剂为 5.3 ± 1.2 mmHg。150 mg阿利吉仑的降压效果与150 mg依贝沙坦相似(使用最小二乘法计算,依贝沙坦使坐位DBP和SBP分别下降 8.9 ± 0.7 mmHg和 12.5 ± 1.2 mmHg)。阿利吉仑300 mg和600 mg的降低平均坐位DBP效果显著强于依贝沙坦150 mg($P < 0.05$)。试验表明,阿利吉仑的安全性和耐受性与安慰剂和依贝沙坦相近,且各组不良事件发生率和中止试验的患者数相近。结论:每日一次口服阿利吉仑治疗轻中度高血压可有效降压,且其安全性和耐受性与依贝沙坦和安慰剂相近。阿利吉仑150 mg与依贝沙坦150 mg降压效果相当。

0576. Troponin elevation after percutaneous coronary intervention directly represents the extent of irreversible myocardial injury: Insights from cardiovascular magnetic resonance imaging

Selvanayagam J. B. / Porto I. / Channon K. et al.
[Dr. A. P. Banning, Department of Cardiology, John Radcliffe Hospital, Oxford, OX3 9DU, United Kingdom] -
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Background- Although troponin elevation after percutaneous coronary intervention(PCI) is common, uncertainties remain about the mechanisms of its release and its relationship to the volume of myocardial tissue loss. Delayed-enhancement MRI of the heart has been shown to reliably quantify areas of irreversible myocardial injury. To investigate the quantitative relationship between irreversible injury and cardiac troponin release, we studied the incidence and extent of new irreversible injury in patients undergoing PCI and correlated it to postprocedural changes in cardiac troponin I. Methods and Results- Fifty patients

undergoing PCI were studied with preprocedural and postprocedural(24 hours) delayed-enhancement MRI for assessment of new irreversible myocardial injury. Cardiac troponin I measurements were obtained before PCI and 24 hours after PCI. Of these 50 patients, 24 underwent a further third MRI scan at a median of 8 months after the procedure. Mean patient age was 64 ± 12 years. After the procedure, 14 patients(28%) had evidence of new myocardial hyperenhancement, with a mean mass of 6.0 ± 5.8 g, or $5.0 \pm 4.8\%$ of total left ventricular mass. All of these patients had raised troponin I levels(range 1.0 to 9.4 $\mu\text{g/L}$). Thirty-four patients(68%) had no elevated troponin I and no evidence of new myocardial necrosis on MRI. There was a strong correlation between the rise in troponin I measurements at 24 hours and mean mass of new myocardial hyperenhancement, both early($r = 0.84$; $P < 0.001$) and late($r = 0.71$; $P < 0.001$) after PCI, although there was a trend for a reduction in the size of PCI-induced myocardial injury in the late follow-up scan($P = 0.07$). Conclusions- In the setting of PCI, patients demonstrating postprocedural elevation in troponin I have evidence of new irreversible myocardial injury on delayed-enhancement MRI. The magnitude of this injury correlates directly with the extent of troponin elevation.

对心血管 MRI 研究发现经皮冠状动脉介入术后肌钙蛋白浓度升高直接代表心肌不可逆损伤的程度

背景:虽然经皮冠状动脉介入术(PCI)后肌钙蛋白常常升高,但肌钙蛋白释放的机制及其与心肌组织丧失的关系仍不确定。有资料显示,心脏延迟增强 MRI 能可靠定量不可逆心肌损伤的范围。为研究心肌不可逆损伤与肌钙蛋白释放量的关系,本研究检测了 PCI 术后患者新发不可逆心肌损伤的发生率和范围,并将其与术后肌钙蛋白 I 的变化建立联系。方法和结果:50 例接受 PCI 的患者在术前和术后(24 h)进行心脏延迟增强 MRI 检查以检测新发不可逆心肌损伤。PCI 前和 PCI 后 24 h 分别测量心肌肌钙蛋白 I。50 例患者中,24 例在术后 8 个月(中位值)接受第 3 次 MRI 扫描。患者平均年龄为 $64(\pm 12)$ 岁。术后 14 例患者(28%)出现新发心肌高增强表现,平均质量为 6.0 ± 5.8 g,占左心室总质量的 $5.0\% \pm 4.8\%$ 。所有患者肌钙蛋白 I 水平均升高($1.0 \sim 9.4 \mu\text{g/L}$)。34 例患者(68%)无肌钙蛋白 I 升高,且 MRI 显示无新发心肌坏死。尽管晚期随访扫描表明 PCI 引起的心肌损伤面积有减小趋势($P = 0.07$),但不论 PCI 术后早期($r = 0.84$, $P < 0.001$)还是晚期($r = 0.71$, $P < 0.001$),24 h 肌钙蛋白 I 升高与新发心肌高