

运动性高血压的研究进展

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摘要

高血压是心脑血管疾病重要的风险因素, 故筛选防治高血压的重点对象有重要的临床意义。运动性高血压能为高血压病的早期诊断及高血压危险性的评价提供简单可靠的指标。本文旨在探讨运动性高血压的概念、诊断标准、发病机制、影响因素及临床意义, 来提高临床对高血压的认识和关注。

关键词

运动性高血压, 运动性高血压反应, 高血压病

Research Progress of Exercise Hypertension

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Abstract

Hypertension is an important risk factor for cardiovascular and cerebrovascular diseases. Therefore, the key target of screening prevention and control of hypertension has important clinical significance. Exercise hypertension provides simple and reliable indicators for early diagnosis of hypertension and evaluation of hypertension. This article aims to explore the concepts, diagnostic standards, pathogenesis, influencing factors and clinical significance of motor hypertension, to improve the clinical understanding and attention of hypertension.

Keywords

Exercise Hypertension, Hypertensive Response to Exercise, Hypertension



1. 引言

我国高血压患病率随着社会老龄化而逐渐增加,而高血压及其所致的心脑血管疾病仍是全球最主要的死亡原因之一[1]。故筛选高血压重点防治对象有重要的临床意义,研究表明运动性高血压是未来发生高血压、心脑血管疾病重要的风险因素,因此提高对运动性高血压的认识,能有效预防或延缓心脑血管等疾病的发生和发展。近年来随着运动试验在临床中的应用增加,对运动高血压的研究也越来越多,现对国内外运动高血压的研究情况进行综述报告。

2. 运动性高血压的概念及诊断标准

运动性高血压(exercise hypertension, EH)的概念最早在 1983 年由 Dlin 等[2]提出,是指在一定的运动负荷下,运动过程中和(或)运动结束后恢复期内,血压超出正常生理反应范围而异常增高的征象,又称为运动性高血压反应(hypertensive response to exercise, HRE)。由于运动中的血压反应受到多种因素的影响,包括运动方式、运动负荷、研究人群、年龄、性别等,因此对于运动性高血压的诊断标准并没有统一的规定。关于运动性高血压的诊断标准在国内外研究中有很多,目前尚未得到统一的标准,以下三种诊断标准较为常见:1) 美国心脏协会(AHA)标准:峰值收缩压(SBP)男性 ≥ 210 mmHg (1 mmHg = 0.133 kPa)、女性 ≥ 190 mmHg;和(或)峰值舒张压(DBP) ≥ 90 mmHg 或 DBP 升高幅度 ≥ 10 mmHg [3]。2) Framingham 心脏研究标准:峰值 SBP 男性 ≥ 210 mmHg、女性 ≥ 190 mmHg [4]。3) Dlin 标准:峰值 SBP ≥ 200 mmHg,和(或)峰值 DBP ≥ 90 mmHg 或 DBP 升高幅度 ≥ 10 mmHg [2]。

3. 运动性高血压的发病机制

目前为止对于运动性高血压的发病机制尚未完全了解。根据目前国内外的研究,运动性高血压的发病机制主要可以归纳为以下几点:

3.1. 交感神经系统亢进

Miyai 等[5]对 54 名血压正常且久坐不动的男性进行了踏车运动试验研究,发现虽然 EH 组与非 EH 组血浆肾上腺素水平相似,但是运动引起的血浆去甲肾上腺素水平明显高于非 EH 组。Mert 等[6]的研究表明,基于 β 受体阻滞剂的单一或联合治疗,与高血压患者较低的运动血压相关。

发现接受 β 受体阻滞剂治疗(OR: 0.637; 95% CI: 0.428~0.949)是运动性高血压的独立预测因子。另有研究认为 EH 患者较非 EH 患者交感神经兴奋性增强,迷走神经兴奋性减弱,可能对迷走神经抑制作用更强[7]。由此可见,在运动期间机体交感神经系统的亢进对于运动时血压的升高起着重要作用,并且为运动性高血压的治疗提供了重要的参考方向。

3.2. 肾素 - 血管紧张素 - 醛固酮系统(RAAS 系统)过度激活

Kim 等[8]研究表明正常血压组的血管紧张素 II 水平较运动性高血压组显著降低。血管紧张素 II 活性与 EH 相关。运动后,非 EH 组的 NO 水平没有变化,但 EH 组的 NO 水平降低。NO 水平降低与 EH 相关。在 Shim 等[9]的研究中发现运动性高血压患者在休息状态下的血压、肾素、醛固酮和儿茶酚胺水平与正常人没有明显差异。在运动过程中两者的肾素、醛固酮和儿茶酚胺水平都有相应的增加,但仍然没

有明显差异。唯独血管紧张素 II 在运动性高血压患者中明显升高。RAAS 过度激活在 EH 发生中有重要价值。

3.3. 血管内皮功能损伤与动脉硬化

国外学者 Kader [10]等采用血流介导扩张以及扩张率评价内皮功能,研究发现运动后出现血压异常升高患者的血流介导扩张以及扩张率要明显低于血压正常的患者。Chung 等研究报道[11],运动中收缩压和舒张压升高与血流介导性动脉扩张减弱等参数有关,提示 EH 患者血管内皮受损,使生成、激活和释放的舒张因子(一氧化氮、前列环素等)减少和收缩因子(内皮素等)增多,导致动脉的内皮依赖性舒张功能减弱。无 Kayrak 等研究[12]在运动性高血压的患者中其血浆中的非对称性二甲基精氨酸(asymmetric dimethyl arginine, ADMA)含量远高于正常者;而 ADMA 对一氧化氮的合成具有抑制作用,可增加心脑血管事件的发生。Sarma [13]等研究报道同样证实了在一般健康人群中, EH 患者较非 EH 者相比,有更差的血管内皮功能和动脉硬化。近期国内房慧雯[14]等研究将原发性高血压患者进行次极量跑台运动负荷试验,结果发现运动性高血压组的踝臂脉搏波传导速度(baPWV)、颈动脉内膜中层厚度(IMT)及高于运动血压正常组,且 baPWV 与发生运动性高血压之间呈正相关关系,是影响 EH 发生的独立危险因素,次极量运动时 SBP 随着 baPWV 增快而越高。提示 EH 的患者比非 EH 的患者动脉硬化更明显,运动性高血压可能会加速动脉粥样硬化的进展。杨雪[15]等研究也探讨了运动性高血压与动脉硬化的相关性。根据既往是否有高血压病史及是否符合运动性高血压的诊断标准,分为 4 组,4 组间比较动脉硬化指数(arterosclerosis index, AI)、颈股脉搏波传导速度(cfPWV)、踝臂指数(ankle-brachial index, ABI)差异均有统计学意义($p < 0.05$)。运动中最高收缩压与 AI、cfPWV、ABI 成正相关。Bitigen 等[16]通过计算主动脉僵硬度和进行超声心动图检查,发现相比于运动血压正常组,运动性高血压组的主动脉僵硬程度显著增高($p = 0.001$),主动脉扩张型则显著降低($p = 0.001$)和左室质量明显增加。此外,他们还发现左室质量与主动脉僵硬指数及次极量运动时收缩压密切相关。这些发现表明在临床表现出高血压之前,运动性高血压就可能加速动脉硬化和弹性功能减退,并促进左室肥大的发生。

3.4. 遗传方面

在 Alioglu 等[17]的研究中,探讨 α -内收蛋白 Gly460Trp 基因 Trp460 的多态性对高血压患者的影响,分析发现,携带至少一个 α -内收蛋白基因的 Trp460 等位基因的患者运动高峰期和恢复期(3 分钟的收缩压反应高于没有相关等位基因者。两个突触前去甲肾上腺素转运相关的等位基因 T-182C 和 A-3081T 与运动性高血压相关,Kohli 等[18]的研究表明在 145 名健康受试者中,T-182C 和 A-3081T 的变异率可达 44% 和 58.9%,并且经过多变量因素校正后的收缩压峰值和收缩压区域均明显高于无相关等位基因者。

3.5. 炎症反应

Jae 等[19]研究显示,相较于运动中血压正常反应者,运动性高血压者的外周血白细胞计数明显更高,而且白细胞计数与运动血压水平呈而且白细胞计数与运动过程中收缩压的增高成正相关。Kilicaslan 等[20]研究通过比较 EH 组与非 EH 组炎症指标,发现 CRP、白介素 6、纤维蛋白原与 EH 密切相关。

4. 影响因素

Huang 等[21]运动血压会随着 BMI 水平增高而增高。同样在国内相关研究中[22]也发现高 BMI (BMI $\geq 28.0 \text{ kg/m}^2$)与运动性高血压显著相关,另有研究纳入了接受减肥手术的严重肥胖患者,在减肥手术前以及术后 3 个月和 6 个月进行运动测试,结果表明,减肥会一定程度降低运动血压[23]。巴俊强等[24]通过稳态模型 HOMA-IR 法进行 2 型糖尿病患者的胰岛素抵抗评估,发现运动性高血压组 HOMA-IR 指数明

显高于运动血压正常组, 并通过多因素 Logistics 回归分析发现胰岛素抵抗的概率是运动血压正常组 1.48 倍, 证实了胰岛素抵抗与运动性高血压的相关性。既往研究发现, 在心血管疾病风险较高的 2 型糖尿病患者中, 运动性高血压的患病率为 50% [25]。Laurinavicius [26] 等研究发现非酒精性肝脂肪变性(HS)与运动性高血压反应独立相关。Sengul [27] 等研究对血压正常的受试者进行运动测试, 发现心外膜脂肪厚度(EFT)与运动血压的升高有关, 并且 EFT 的超声心动图测量值可作为预测未来高血压风险增加的指标。综上, EH 与心血管疾病的影响因素相似, 包括静息血压、年龄、性别、饮酒、体质指数、胆固醇水平、肥胖、内脏脂肪、胰岛素抵抗等[28]。

5. 运动性高血压的临床意义

5.1. 运动性高血压与高血压

有研究表明运动性高血压与未来新发高血压相关[29] [30]。Miyai 等[31]过长期随访发现 EH 与高血压发生风险独立相关, EH 是高血压前期患者的独立危险因素。另一方面, 国外有研究发现 EH 中隐匿性高血压比例高达 58% [32]。同样也有研究证实运动高峰时测量的舒张压是 EH 患者隐匿性高血压的独立预测因子[33]。

5.2. 运动性高血压与心血管疾病

有研究表明[34]运动收缩压与左室质量、左室质量指数、相对室壁厚度、后壁厚度和室间隔厚度的增加相关。EH 患者与无 EH 的患者相比发生左室肥厚的风险高 2.6 倍, 平均左室质量、左室质量指数、相对室壁厚度、室间隔厚度(0.78 ± 0.17 mm)和左心房直径更高。唐念等[35]发现运动性高血压是高血压患者心肌微循环损伤的独立危险因素。Carneiro 等[36]对 2066 名 Framingham 后代研究的参与者进行了活动平板试验, 随访 16.8 年, 运动期间舒张压升高、运动后收缩压恢复和心率恢复减慢是射血分数降低型心力衰竭的标志。病例对照研究得出, 心肌梗死、卒中发生的危险度均更大, 揭示了运动血压对心脑血管事件、心血管病发病率和死亡率的预测价值, 有利于高血压危险程度的评估[37]。

5.3. 运动性高血压与脑血管疾病

有研究发现[38], 有脑白质病变(WML)的受试者 EH 发生率较高。尤其是在血压正常的受试者中, 运动峰值 SBP 升高与 WML、亚临床脑部小血管疾病显著相关。Spartano [39]等表明在高血压患者中, 运动 SBP 近 20 年后较小的脑容量相关。有国外研究[40]对健康中年男性随访 35 年。发现中等和最大运动负荷时的 SBP 以及卒中风险之间存在显著的正相关关系, 并且随着静息收缩压和最大运动收缩压之间差异的增加, 脑卒中风险增加, 提示运动中血压过度反应可能是脑卒中的独立预测因素。Wijkman [41]等研究还指出运动 SBP 异常升高可作为预测未来脑卒中的一种辅助手段。还有进一步研究[42]通过 44 年长期随访发现, 运动最大收缩压每增加 24.6 mmHg, 卒中的风险就会增加 34%, 静息收缩压 ≥ 140 mmHg 且运动最大收缩压 ≥ 210 mmHg 的男性卒中风险最高。

5.4. 其他疾病

运动性高血压是心血管危险因素, 但是运动性高血压与肾脏疾病的关系存在争议。Çoner [43]等对 170 例中年健康志愿者进行极量平板运动试验, 发现 EH 组尿白蛋白与肌酐比值明显升高($p = 0.002$), 提示 EH 可能与亚临床肾病有关。国内研究发现[14] EH 组的 UACR 高于运动血压正常组, 说明 EH 与肾损害有一定相关性。但两组间 SCr、BUN 并无明显差异, 但 UACR 已经升高, 说明 UACR 比 SCr、BUN 更有利于发现高血压早期肾功能损害。Tanaka [44]等研究提示 UACR 比尿微量白蛋白更能反映肾脏血管的早期

损害。另有研究[45]对 2 型糖尿病患者进行轻中度运动, 通过静息时和运动后 30 分钟的白蛋白 - 肌酐比值(ACR)评估白蛋白尿, 发现运动性高血压反应组的患者比正常血压组的运动后 30 分钟 ACR 值明显升高。运动中血压反应过高可能还会增加发生慢性肾脏病的风险[46]。Zhou 等[47]研究对没有抑郁症状的受试者, 平均随访 3.9 年后, 发现运动性高血压可能通过增加大脑脉动压力负荷, 而与抑郁症的发生率较高相关。另外, 近期国外一项研究提示感染 COVID-19 的运动员的 EH 发生率更高[48]。

6. 小结

综上所述, 运动性高血压与高血压、心脑血管等疾病相关, 具有重要的临床意义。既往研究表明运动性高血压与交感神经系统亢奋、肾素 - 血管紧张素 - 醛固酮系统的过度激活、血管内皮功能受损和动脉硬化、遗传因素、炎症反应等有关。但目前运动性高血压未得到像静息性高血压一样的重视, 对于运动性高血压的发病机制的研究不多, 其发病机制尚未完全明了。并且运动性高血压的诊断标准尚未确定, 目前多数研究都是以外国人群为基础。因此, 需要我们继续努力建立以我国人群为基础的运动性高血压的诊断阈值与治疗方案, 进一步探索其发病机制和临床意义, 使运动性高血压在临床工作中得到重视和普及[14]。

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