·综 述·

老年慢性肾脏病合并肌少症的研究进展

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【摘 要】 肌少症是以骨骼肌质量、力量及功能降低为主要特征的退行性综合征。肌少症与衰老密切相关,但是,慢性肾脏病等疾病可以加速肌肉消耗,增加肌少症的发生率。慢性肾脏病患者易合并肌少症,其机制涉及炎症反应、蛋白质能量消耗、运动减少及维生素D缺乏等。早期识别肌少症的危险因素并对其进行干预,对于慢性肾脏病合并肌少症患者生活质量的改善至关重要。目前,干预措施主要有体育锻炼、营养补充及药物治疗等。本文就近年对老年慢性肾脏病合并肌少症的发病情况、机制及治疗的研究进展作一综述。

【关键词】 老年人;慢性肾脏病;肌少症
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Research progress in chronic kidney disease complicated with sarcopenia in the elderly

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【Abstract】 Sarcopenia is a degenerative syndrome characterized by reduced skeletal muscle mass, strength and function. It is closely related to aging, but some diseases like chronic kidney disease can accelerate muscle wasting and increase the incidence of sarcopenia. Those patients suffering from chronic kidney disease are prone to sarcopenia, and its mechanisms include inflammatory responses, protein energy wasting, reduced exercise and vitamin D deficiency. Early identification and intervention for sarcopenia is essential for improving the quality of life in the patients with chronic kidney disease complicated with sarcopenia. At present, the intervention measures mainly include physical exercise, nutrition supplement and drug treatment. This paper reviews the recent research progress in the pathogenesis, mechanism and treatment of chronic kidney disease complicated with sarcopenia.

【Key words】 aged; chronic kidney disease; sarcopenia
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慢性肾脏病(chronic kidney disease,CKD)是一种以肾脏排泄和内分泌功能逐渐丧失为特征的慢性代谢性疾病,在疾病进展过程中通常会合并肌量减少、肌力下降等。肌少症(sarcopenia)是以骨骼肌质量、力量以及功能降低为主要特征的退行性综合征,其会增加老年人的住院率及医疗费用,严重影响老年人的生活质量。CKD等慢性疾病会加快肌少症的进展过程,而肌少症也会对CKD患者的预后产生不良的影响。近年来,肌少症已成为研究热点。本文就近年对老年CKD合并肌少症的发病情况、机制及治疗的研究进展作一综述。

1 肌少症的定义及诊断标准

肌少症于1989年由Rosenberg首次提出,它被认为是一种与年龄相关的疾病,表现为骨骼肌质量、力量或功能不同程度的丧失,增加跌倒、残疾等不良事件发生为特征的"老年综合征"[1],是老年人生理功能逐渐减退的重要原因和表现之一。2010年,欧洲老年肌少症工作组(European Working Group on Sarcopenia in Older People,EWGSOP)发表了肌少症

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共识[2]。此后,国际肌少症会议工作组(International Sarcopenia Consensus Conference Working Group, ISCCWG)、亚洲肌少症工作组(Asian Working Group for Sarcopenia, AWGS)先后就肌少症的定义及诊断标准发布新共识[3,4]。我国在2016年达成了肌少症专家共识,建议肌少症的筛查及诊断如下。(1)先行步速测试,若步速≤0.8 m/s,则进一步测评肌量;步速>0.8 m/s,则进一步测评手部握力。(2)若静息情况下,优势手握力正常(男性握力>25 kg,女性握力>18 kg),则排除肌少症;若肌力低于正常,则要进一步测评肌量。(3)若肌量正常,则排除肌少症;若肌量减低,则诊断为肌少症[5]。

2 CKD合并肌少症的发病情况

肌少症,是一种重要的与年龄有关的生理变化。据美国和欧洲部分国家报道,老年人60~70岁肌少症患病率为5%~13%,>80岁为50%~60%[6];2000年美国花费在肌少症的直接医疗费用接近18.5亿美元,占总医疗费用的1.5%[7]。肌少症虽然是一种增龄性疾病,但近来研究发现,CKD等慢性疾病可以加速这个过程,肌少症也会对CKD的预后产生不良的影响,两者相互作用共同使得老年人的运动能力、生活质量下降,死亡率增加。有研究表明,CKD患者可能也经历与年龄有关的过程。然而,其肌肉质量的损失更为严重。此外,与同龄人相比,在CKD患者中更易观察到肌少症的早期症状[8]。肌少症被证明与CKD患者的死亡率增高密切相关[9]。肌少症是CKD患者常见的并发症之一,长期以来,各项研究都表明接受血液透析的患者中肌少症的发病率更高[10]。另外,最近的一项研究表明,未接受透析的CKD患者肌少症的发生率高于那些非CKD患者[11]。

3 CKD合并肌少症的临床研究

肌少症虽然最初被认为是一种与衰老有关的疾病,但国际社会目前都认识到代谢病(如CKD)在肌少症的病因学中所起的重要作用。CKD固有的代谢紊乱导致蛋白质分解代谢增加,肌肉质量和功能下降[12]。因此,CKD患者更容易合并肌少症。合并肌少症的CKD患者常常表现为肌力减退、活动能力下降,易出现疲劳、乏力等。Ishikawa等[13]纳入了260名非透析CKD患者研究肌少症的患病率和危险因素,结果表明25%的研究对象患有肌少症,多变量分析显示,肌少症的风险增加与年龄、性别、体质量指数、糖尿病及袢利尿剂使用显著相关(OR=4.59,95%CI 1.81~11.61;P<0.001)。同样,大量研究表明维持性血液透析患者肌少症的发生率也显著升高。Honda等[14]系统综述结果发现,肌少症和缺乏运动在CKD患者中协同进展,是该人群死亡率的预测因子。

4 CKD合并肌少症的发生机制

目前关于CKD合并肌少症发病机制的研究主要集中在炎症反应、蛋白质能量消耗、运动减少及维生素D缺乏等方面。

4.1 炎症反应

微炎症状态在CKD患者中较为常见。有研究表明[15],在CKD早期阶段循环中炎症标志物C-反应蛋白(C-reactive protein, CRP)、白细胞介素-6(inerleukin 6, IL-6)、肿瘤坏死因子α(tumor necrosis factor-α, TNF-α)水平即开始升高。这些炎症因子与肌少症的发生发展密切相关[16]。CKD患者的炎症状态通过增加蛋白质分解代谢、减少蛋白质合成或肌肉祖细胞增殖受损等途径导致肌肉萎缩。有研究表明[17],注入TNF-α、IL-6、IL-1β、干扰素(interferon-γ, IFN-γ)会促进肌肉蛋白降解,而中和这些因素会通过遗传或药理方法减少肌少症的发生。据报道,核因子-κB(nuclear factor-κB, NF-κB)是炎症反应的关键调控者,TNF-α即通过NF-κB通路增强肌肉消耗,其激活对于细胞因子诱导的骨骼肌蛋白损失是必需的。此外,Verzola等[18]研究发现,CKD还可以通过上调Toll样受体4(Toll like receptor 4, TLR4)激活下行的炎症信号如TNF-α和NF-κB调控基因,促进肌肉的炎症。

4.2 蛋白质能量消耗

传统观念认为,CKD营养不良可能与患者营养物质摄入不足或蛋白质丢失过多有关,但近年来大量研究表明CKD患者存在多种蛋白质能量代谢异常,其中骨骼肌消耗是最主要特征,这被称为“蛋白质能量消耗(protein energy wasting, PEW)”[19]。最近的一项研究发现,31%的CKD(包括透析和非透析)患者普遍存在PEW[20],造成肌肉蛋白质合成、分解的不平衡。目前,低蛋白饮食被普遍认为可以有效延缓CKD进展,改善预后,但是减少CKD患者蛋白质摄入的安全性一直受到质疑。两项安全性研究表明给予低或极低蛋白饮食并辅以酮酸的CKD患者中营养不良的发生率很低[21],这说明CKD患者合理应用低蛋白饮食不仅不会引起营养不良,反而可以改善营养状况。但是由于CKD疾病本身包括厌食症、能量消耗增加以及透析相关因素等,使

CKD患者易发生PEW,导致肌肉蛋白质消耗增加和合成相对不足,引起肌肉量减少,进而导致肌少症的发生。其中厌食症在CKD患者中很常见,可能是由于刺激食欲和抑制食欲激素的改变、肾衰竭时体内代谢废物的累积、味觉异常和药物对味蕾的影响等[22]。

4.3 运动减少

CKD的重要并发症之一是骨骼肌减少及萎缩,导致疲劳、肌肉抽筋、衰弱和低体能等。这些症状在CKD非透析患者中就已经存在,随着时间的推移,肾功能逐渐下降进展至晚期肾功能衰竭时,可能会导致参与体育活动减少[23]。先前的文献报道[24],在CKD尤其是接受肾替代治疗的终末期肾病(end-stage renal disease,ESRD)患者中,缺乏运动是很常见的。而大量研究表明,运动锻炼特别是抗阻运动可以增加肌肉量,所以缺乏体育锻炼的CKD患者更容易合并肌少症。

4.4 维生素D缺乏

CKD患者普遍存在维生素D缺乏和不足[25],有数据显示,其发生率在CKD人群中竟高达70%~80%,尤其在维持性血液透析患者中发生率更高[26]。观察性研究表明,循环中25-羟维生素D[25(OH)D]下降的水平与肌肉症状的严重程度相关[27]。同样,最新证据表明,维生素D受体在肌肉细胞中表达,调节基因表达及25(OH)D在骨骼肌细胞中的吸收,可作为维生素D的储存场所[28,29]。也有证据表明维生素D缺乏通过干扰胰岛素敏感性来影响肌肉收缩功能和肌肉代谢[30]。而补充维生素D可刺激成肌细胞,促进卵泡抑制素、抑制肌生成抑制素的表达,增加肌肉蛋白质的合成。

除此之外,CKD合并肌少症的发生机制还涉及胰岛素信号通路异常、生肌调节因子异常、线粒体功能障碍等[31]。

5 CKD合并肌少症的治疗进展

5.1 体育锻炼

体力活动是延缓肌少症发生的主要干预措施。研究表明抗阻运动可以通过克服阻力来达到肌肉增长和力量增加。Watson等[32]对38例CKD患者进行随机对照研究,实验组进行了为期8周的渐进性抗阻运动,对照组进行常规的身体活动,结果表明,渐进性抗阻运动可以增加肌肉解剖横截面积、肌肉体积、膝伸肌力量及运动能力。同样,在一组36名透析前CKD患者的研究中发现,进行为期1年的有氧运动和抗阻运动后,身体活动显著增加,肌肉力量增强[33]。这都表明,对CKD患者进行运动干预,可改善肌肉功能、增加肌肉力量、减少肌少症的发生。

5.2 营养补充

CKD患者特别是终末期肾衰竭患者,多存在营养不良和代谢紊乱。增加机体蛋白质和氨基酸的摄入,对肌少症的预防甚至治疗有一定作用。Yoshimura等[34]将44名老年肌少症患者随机分成干预组和对照组,干预组给予富含亮氨酸的氨基酸,对照组未予特殊干预,8周后干预组肌少症患者的骨骼肌质量、力量及肌肉功能较对照组明显改善。目前普遍认为,适量的蛋白质、必需氨基酸和其他营养成分的补充,对老年人肌肉内环境稳态的维持非常重要。为平衡CKD患者低蛋白饮食与补充蛋白质及氨基酸以避免发生蛋白质能量消耗的关系,CKD患者的饮食推荐如下:对于CKD 3-5期病情稳定的非透析患者予摄入蛋白质0.6~0.8 g/(kg·d)(优质蛋白质>50%),能量30~35 kcal/(kg·d),可加用α酮酸或必需氨基酸。对于透析患者而言,其在透析过程中的炎症刺激、氨基酸和白蛋白丢失等都可以促进蛋白质分解,所以蛋白质的摄入量一般高于非透析患者,因此在透析患者中蛋白质和能量摄入量分别为1.2 g/(kg·d)(优质蛋白质>50%)和30~35 kcal/(kg·d),可加用α酮酸或必需氨基酸[35]。

5.3 激素

性激素(睾酮、雌激素)及生长激素具有促进肌肉合成的作用,使肌肉力量明显提高。研究表明,CKD患者在肾功能下降的不同阶段都可能出现性腺功能减退,其中大约50%发生在血液透析阶段[36]。对性腺功能低下者雄激素补充疗法可使肌肉蛋白合成率增加,使肌肉力量增强、肌肉体积增大等。但其是否可为肌少症患者带来获益,其有效性和安全性还需要大规模临床试验进一步证实。

6 总结与展望

综上所述,肌少症是CKD的一种常见并发症,与CKD患者的预后密切相关。近年来的研究表明,CKD合并肌少症的发病机制涉及炎症反应、蛋白质能量消耗、运动减少、维生素D缺乏等。早期识别CKD合并肌少症的危险因素,并采取积极有效的干预措施,如鼓励患者积极加强体育锻炼、进行营养支持等是至关重要的。

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·消 息·

致“一带一路”沿线国家和地区医学机构

《中华老年多器官疾病杂志》是由中国工程院院士、老年心脏学专家王士雯教授于2002年创办的全世界惟一一本以老年心脏病和老年心脏病合并其他器官疾病为主要内容的杂志,月刊,由中国人民解放军总医院老年心血管病研究所主办。杂志已被“中国科技论文统计源期刊”(中国科技核心期刊)收录。本杂志的摘要、图表和参考文献,均为中、英文双语对照,方便国外读者顺利阅读。为促进中国与“一带一路”沿线国家和地区的医学及文化交流,本刊将免费刊登其来稿,并赠送当期杂志。欢迎“一带一路”沿线国家和地区的老年心脏病和老年病学医生、学者踊跃投稿。

To medical academic institutions of all countries along the Belt and Road

The Chinese Journal of Multiple Organ Diseases in the Elderly (*Zhonghua Laonian Duoqiguan Jibing Zazhi*) is founded in 2002 by Shiwen Wang, Member of Chinese Academy of Engineering, a renowned geriatric cardiologist in China. The journal is published monthly by the Institute of Geriatric Cardiology (IGC), Chinese PLA General Hospital in Beijing, China. The journal, the only one in the world currently, focuses on both basic research and clinical practice to the diagnosis and treatment of cardiovascular disease in the aged people, especially those with concomitant disease of other major organ-systems, like the lungs, kidneys, liver, central nervous system, gastrointestinal tract or endocrinology, *etc*. The journal has been listed in the most authoritative Chinese database, the Chinese Scientific and Technical Papers and Citations Database (Chinese Core Sci-Tech Periodical). For convenience of foreign readers, the main parts of the paper, including abstract, tables, figures and references, are expressed in Chinese-English bilingually. To facilitate the cultural and academic communication between China and countries or regions along the Belt and Road, the journal welcomes the manuscripts from these areas. If reviewed qualified, the manuscript would be published without charging, and the authors would receive a complimentary copy of the current issue.

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