干眼与心理因素双向影响的研究进展

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【摘要】 干眼是一种严重影响患者生活质量的多因素眼表疾病。近年来,干眼与焦虑、抑郁、压力及睡眠障碍等心理因素之间的双向关联日益受到关注。本文旨在系统综述干眼与心理因素的相互作用及其深层机制,基于“心理-神经-内分泌-免疫(psycho-neuro-endocrino-immune, PNEI)网络”,重点探讨了自主神经失衡、中枢敏化、炎症通路及神经内分泌功能障碍的作用,同时分析了“脑-肠-眼轴”这一新兴前沿观点;基于“生物-心理-社会”医学模式的综合诊疗策略,提出整合医学模式下的多学科诊疗建议,为精准联合治疗提供新思路。

【关键词】 干眼;心理因素;抑郁;睡眠障碍

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Research progress on the bidirectional influence of dry eye disease and psychological factors

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【**Abstract**】 Dry eye disease (DED) is a multifactorial ocular surface disorder. The disease seriously affects the quality of life of patients. In recent years, the bidirectional relationship between DED and psychological factors such as anxiety, depression, stress and sleep disorders is increasingly receiving attention. This article systematically reviews the interaction between DED and psychological factors as well as its underlying mechanisms. Based on the Psycho-Neuro-Endocrino-Immune (PNEI) network, the roles of autonomic nervous system dysregulation, central sensitization, inflammatory pathways and neuroendocrine dysfunction are focused. Furthermore, the emerging concept of the Brain-Gut-Eye Axis is examined as a frontier perspective. Based on the comprehensive diagnosis and treatment strategy of the "bio-psycho-social" medical model, suggestions of multidisciplinary diagnosis and treatment under the integrated medical model are proposed. This may provide new ideas for precise combination therapy.

【**Key words**】 Dry eye disease; Psychological factors; Depression; Sleep disorders

干眼是一种多因素、有症状的疾病,其特征是泪膜和/或眼表稳态的失衡,其中泪膜不稳定和高渗透压、眼表炎症和损伤以及神经感觉异常是其病因学因素[1]。其病情程度可从初期的轻度功能性不适,逐步迁延加重,甚至存在失明风险[2,3]。当患者兼具焦虑、抑郁、压力、睡眠障碍等心理障碍时,可出现“症征分离”现象,即诊断干眼的客观指标并不总是与症状相匹配[2,4]。多项研究提示,干眼与心理因素存在密切且双向的关联,构成了一个自我强化的“恶性循环”。本文从干眼与心理因素的双向影响及其机制展开综述,为构建多学科协作下的干眼个性化诊疗模式提供理论支撑。

1 流行病学

1.1 干眼的流行病学 干眼是全球范围导致眼部不适的常见眼病,其全球患病率 5%~50%,中国患病率 21.0%~52.4%,而女性在所有年龄段的临床诊断性患病率均高于男性[5~7]。随着人口老龄化和生活方式数字化,干眼的患病率仍将持续攀升,这已成为公共卫生领域亟待解决的重要问题。

1.2 心理因素的流行病学 第二次世界大战后,研究发现干眼是一种与心身医学领域密切相关的眼部疾病,故提出“心身眼科”[8]。多项研究指出,干眼与心理因素存在关联,尤其在负面情绪方面表现明显[9]。为及时诊疗“症征不符”的干眼患者并找到共病线索,应将“生物-心理-社会”模型纳入干眼的诊疗框架。

2 干眼与心理因素的双向影响

多种因素与干眼密切相关,研究发现抑郁、焦虑、压力、睡眠障碍及其治疗药物与干眼存在关联。

2.1 干眼与抑郁、焦虑的双向影响 干眼患者焦虑和抑郁的概率显著高于普通人群,且干眼与抑郁、焦虑状态呈显著正相关。研究显示,哭泣可促进心身恢复,但干眼患者泪液分泌减少,干扰对负面情绪的处

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理[10]。接受有效的干眼治疗可改善焦虑情绪,表明干眼症状的缓解可能对焦虑状态具有积极的反馈作用[11]。抑郁症患者的泪液分泌量和泪膜稳定性下降,眼表健康受到负面影响[12];伴有焦虑症的干眼患者,常表现出更严重的眼部不适和难治性症状[13, 14]。

2.2 干眼与压力的双向影响 心理压力作为应对环境挑战时的应激状态,与干眼严重程度呈显著正相关[15]。干眼患者因长期的眼部不适、频繁就医及经济负担,共同构成持续的心理应激源,加重心理压力,通过中枢神经系统通路增加对疼痛的敏感性,患者放大对眼部的不适感[4]。

2.3 干眼与睡眠障碍的双向影响 睡眠障碍属于心理特征的精神生物学异常,与干眼症状显著相关,而干眼本身也可能是睡眠障碍的驱动因素[4]。睡眠障碍的干眼患者泪膜破裂时间显著缩短、睑板腺形态异常[9]。研究证实,缺乏睡眠可诱导泪液高渗和减少泪液分泌,直接导致干眼的发生[16]。部分患者夜间眼睑闭合不全导致的眼表疼痛也会干扰睡眠,干眼的严重程度与失眠的严重程度呈正相关[17]。睡眠障碍还会破坏大脑高水平的认知功能,与抑郁、焦虑等高度共病,从而加重干眼[18, 19]。

2.4 治疗药物的影响 若干眼患者对滴眼液的成分过敏,可能引起眼部瘙痒、红肿、刺痛等不适,进而引起焦虑;而精神类药物可能通过其抗胆碱能作用影响泪膜稳定性与泪液分泌功能,从而引发干眼[7]。

2.5 积极情绪的影响 研究表明,笑能有效缓解抑郁、焦虑、压力和慢性疼痛;能通过改善泪膜稳定性、脂质层厚度和睑板腺功能来缓解干眼。面部模仿式笑声能放大或引发幸福感,而主观幸福感与干眼症状存在负相关[20]。积极的情绪,影响个体对内部躯体感觉的注意和解读,所以主观幸福感较高的人对不适的感知较低,不易患干眼或其他心理疾病。

3 机制

3.1 神经生物学机制 从自主神经系统失衡、神经病理性疼痛、中枢敏化及脑功能网络连接异常可以发现干眼与心理因素之间在神经生物学中的深层机制。既往研究表明,应激、睡眠障碍等心理因素可通过自主神经系统平衡失调引起泪腺功能障碍及眼部炎症,参与干眼的发生发展。交感神经过度激活可降低外周微循环灌注,使泪腺和角膜上皮更易处于缺氧与高渗环境,启动眼表免疫激活与组织损伤;可升高儿茶酚胺及减少乙酰胆碱,使促炎因子释放,形成促炎微环境[4]。

干眼患者外周或中枢神经系统出现超敏反应,角膜神经功能失调,可出现神经性眼痛,使生理及心理呈双重应激状态[4]。而心理压力、焦虑及抑郁等负面情绪也可出现中枢敏化,直接影响大脑对疼痛的处理,导致眼部疼痛阈值显著降低,增加干眼易感性,成为心理应激源,进一步诱发或加重负面情绪[21]。大脑在处理眼部的痛觉信号时,神经投射通路与负责情绪调控的脑区存在高度重叠,所以干眼引起的不适可能通过刺激大脑某些区域来引发抑郁焦虑等负面情绪。在产生伤害感受的相关区域中,刺激岛叶皮层会引发角膜等眼部组织的伤害感受,诱发焦虑与抑郁。干眼的局部眼表炎症可引发系统性炎症反应,破坏血脑屏障完整性,使神经毒性物质进入中枢,损害前额叶皮层、海马体等情绪调节关键脑区的神经元功能和神经递质平衡,最终导致抑郁样行为[4]。

3.2 免疫炎症与细胞损伤 干眼与心理因素在分子水平的交互聚焦于炎症因子增多、氧化应激失衡及细胞焦亡,造成两者之间的交互激惹。研究发现,抑郁症患者的泪液和结膜中 IL-6、IL-17、TNF-α、IFN-γ 等促炎细胞因子水平显著升高,反映了局部免疫变化的程度,并影响干眼临床症状的严重程度[22]。持续的焦虑或压力状态使交感神经兴奋,减少具有抗炎作用的乙酰胆碱,释放促炎细胞因子,形成炎症微环境[4]。抑郁症或焦虑症患者的患者外周血中可观测到类似炎症标志物的异常增高,且泪液和结膜中 $CD4^+$ 淋巴细胞数量增加,提示神经免疫炎症可能作为共同病理基础[23]。

焦虑、抑郁状态过度激活交感神经系统,抑制副交感神经,造成泪液分泌减少,加重眼表氧化应激和细胞凋亡,从而引起神经内分泌和代谢产物改变,引发两者之间的恶性交互[24~26];睡眠障碍状态下,褪黑素的缺乏剥夺了眼表的抗氧化保护,解除对线粒体氧化及 NLRP3/caspase-1/GSDMD 细胞焦亡通路的抑制,加剧了角膜上皮和杯状细胞的炎症与焦亡,加重干眼症状[27, 28]。

3.3 神经内分泌与激素调节 干眼与心理因素的交互涉及多条神经内分泌通路的协同调控,包括慢性压力过度激活交感-肾上腺髓质(SAM)轴与下丘脑-垂体-肾上腺(HPA)轴介导的应激效应,以及性激素失衡在免疫调节与眼表屏障功能中的作用。SAM 轴持续兴奋可引发小血管痉挛与微循环灌注不足,加重泪腺及角膜组织的缺氧状态,使眼表更易进入炎症放大循环[29, 30]。HPA 轴介导的皮质醇是应激反应的核心激素,睡眠障碍激活 HPA 轴,使机体处于高渗脱水状态和炎症易感环境[31~34]。这种应激状态通常伴随雄激素水平下降,从而削弱泪腺与睑板腺的分泌功能,影响泪液分泌量与脂质层的稳定性,导致泪腺、睑板腺功能障碍以及水液缺

乏型干眼;雄激素的缺乏也可引起抑郁症和睡眠障碍[35~39]。研究数据显示,雌激素可上调人角膜上皮细胞中包括 MMP-9 在内的 促炎细胞因子表达,较高的雌激素水平与更严重的抑郁症状相关[40, 41]。

3.4 药物治疗的影响 情绪稳定剂(如碳酸锂)会显著缩短泪膜破裂时间,直接破坏泪膜的结构完整性[42]。苯二氮䓬类抗焦虑药及三环类抗抑郁药(TCAs)可干扰神经递质信号直接影响泪液分泌与眼表稳态,引发干眼[43];也可通过影响结膜杯状细胞表面受体,阻断人泪腺副交感神经的神经支配,减少泪液、睑板腺脂质和泪液黏蛋白分泌,激活泪腺的炎症反应,从而导致泪膜不稳定、泪液渗透压升高[44, 45]。尽管新一代药物(如选择性 5-羟色胺再摄取抑制剂 SSRIs 和 5-羟色胺-去甲肾上腺素再摄取抑制剂 SNRIs)的抗胆碱能副作用较弱,但仍会增加干眼风险[46]。长期接触组胺和 5-羟色胺会改变分泌功能,比如神经元释放的 5-羟色胺可能影响泪液分泌的急性调控[47]。SSRIs 可能导致多种促炎细胞因子(TNF-α、IL-1β、IL-6、IL-10 等)与促凋亡基因(AIF、BAD、BAX)表达升高,提示抗抑郁药可在泪腺与眼表组织中诱导炎症及凋亡程序,造成泪液分泌减少和腺体炎症反应亢进[31, 32, 43, 48~50]。这些药物还可通过增加血清素和炎症介质的分泌,导致角膜神经末梢敏感度升高、疼痛阈值降低,从而引发与干眼样表现[51]。

3.5 潜在的微生态背景 肠道菌群通过调控肠道与中枢免疫反应,参与神经炎症、影响情绪稳态,从而改变眼部症状感知,提示“脑-肠-眼轴”在干眼与心理因素相互作用中发挥关键调控作用[52]。压力、睡眠障碍等心理因素引起 HPA 轴失调,增加潜在致病菌,破坏肠道屏障,使肠道共生细菌及其产物进入循环,诱发病理性炎症反应,影响眼表组织稳态,导致功能障碍;还可通过自身免疫反应、Th17/Treg 失衡等,释放促炎因子,驱动眼表微环境持续炎症与角膜屏障损伤,推动干眼发展[53~55]。肠道菌群紊乱还会导致肠道神经元释放神经肽 Y 和 P 物质等关键信号分子,通过稳态循环分布到眼表面和泪腺,影响泪液分泌[56];还可导致短链脂肪酸(SCFAs)水平下降,降低巨噬细胞抗炎能力、减少泪液分泌[57]。

4 小结

针对干眼伴随心理疾病的患者,需要采取综合治疗,包括心理支持、生活方式干预、肠道干预等。通过详细的疼痛评估并综合考量患者心理健康状况,可制定更具个性化且更有效的综合管理策略,同时改善身心健康。未来研究应针对干眼躯体与情感层面的双重干预,提供更精准完备的治疗方案。

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