

• 综述 •

癫痫与睡眠障碍关系的相关机制研究进展

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[摘要] 睡眠障碍可能诱发或加重癫痫,而癫痫发作也可能干扰患者的睡眠状况。失眠、阻塞性睡眠呼吸暂停、发作性睡病、不宁腿综合征等睡眠障碍与癫痫共病的机制十分复杂,发作性睡病和癫痫可能具有相同的发病机制,非快速眼动睡眠相关异态睡眠与睡眠相关性过度运动性癫痫的诊断需相互鉴别。本文在阐述癫痫与睡眠障碍关系相关机制的同时,还阐述了睡眠结构对癫痫的影响,以期为进一步控制癫痫发作和治疗该类患者的睡眠障碍提供参考。

[关键词] 癫痫;睡眠觉醒障碍;入睡和睡眠障碍;睡眠呼吸暂停,阻塞性;发作性睡病;不宁腿综合征;睡眠,快速眼运动;睡眠结构;综述

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Research advances in the mechanism of the relationship between epilepsy and sleep disorders HU Ya-ting, KONG Qingxia, YAN Huan (Jining Medical University, Jining 272000, China)

[ABSTRACT] Sleep disorders may induce or exacerbate epilepsy, and seizures may interfere with the status of sleep. There are complex mechanisms for the comorbidity between sleep disorders (such as insomnia, obstructive sleep apnea, narcolepsy, and restless legs syndrome) and epilepsy; narcolepsy and epilepsy may share the same pathogenesis, and non-rapid eye movement sleep-related parasomnias and sleep-related hypermotor epilepsy need to be differentiated from each other. This article describes the mechanism of the relationship between epilepsy and sleep disorders and elaborates on the influence of sleep architecture on epilepsy, in order to provide a reference for effective seizure control and the treatment of sleep disorders in such patients.

[KEY WORDS] Epilepsy; Sleep wake disorders; Sleep initiation and maintenance disorders; Sleep apnea, obstructive; Narcolepsy; Restless legs syndrome; Sleep, REM; Sleep structure; Review

癫痫是由多种病因引起的慢性脑部疾病,由大脑神经元高度同步化异常放电所致,具有反复刻板性发作的临床特征,常伴随神经功能损害。睡眠障碍是癫痫患者的常见并发症。研究显示,癫痫和睡眠质量之间的关系是双向的,因为反复发作的癫痫会显著影响患者的睡眠,而良好的睡眠可以缓解癫痫的发作^[1]。癫痫患者常伴有睡眠障碍,常见的睡眠障碍类型有失眠、阻塞性睡眠呼吸暂停(OSA)、发作性睡病、不宁腿综合征(RLS)以及非快速眼动睡眠(NREM)相关异态睡眠。癫痫患者中显著升高的睡眠障碍,构成癫痫发作频率增加及不良预后的独立风险因素。尽管人们对癫痫共患睡眠障碍的认识逐步提高,但目前仍缺乏癫痫与睡眠障碍关系相关机制的研究。现对近年关于睡眠结构对癫痫的影响、癫痫与睡眠障碍关系相关机制进行综述。

1 癫痫与睡眠结构

癫痫会扰乱睡眠结构,因此癫痫患者睡眠障碍的患病率更高。睡眠结构由 NREM 期和快速眼动睡眠(REM)期组成。在 REM 期,脑电图(EEG)呈现低幅快波模式,并伴随骨骼肌张力的完全抑制,REM 期通过增强前脑抑制性神经

递质释放及抑制谷氨酸能传递,可以显著降低皮质神经元兴奋性,其抑制效应可达到:①痫样放电传播阻断;②发作间期癫痫样放电(IEDs)发生率降低 $\geq 70\%$ (部分患者可以完全抑制)^[2]。根据反映神经元活动的特定脑电波模式,NREM 期可分为 N1、N2 和 N3 三个阶段。NREM 期与 REM 期以 90~120 min 为周期交替循环,且周期结构呈动态演变:前半夜以 N3 阶段慢波睡眠为主,后半夜 REM 期持续时间逐次延长^[3]。在 NREM 期,脑干网状上行激活系统对丘脑的兴奋性输入减弱,会导致丘脑皮质神经元超极化和同步化振荡。该同步化状态通过促进神经元集群的同步放电,显著增加 IEDs 的发生概率(较清醒期时增加 3~5 倍)。需要注意的是,REM 期持续存在的 IEDs 具有较高的致痫灶定位的价值——因其能在 REM 期显著抑制性神经生理环境(高胆碱能张力、脑电去同步化、感觉门控增强)下持续存在,该现象可直接反映致痫灶兴奋性的异常情况^[4]。基于此,REM 期痫样放电的空间分布可作为判断癫痫发作起始区定位的可靠依据^[5]。REM 期患者神经元 ATP/二磷酸腺苷(ADP)比值显著下降表明此时大脑处于极高的能量消耗状态,从而致大脑能量供需失衡,通过激活腺苷 A1 受体抑制大脑皮质的兴奋性,使 REM 期局灶性癫痫发作频率较 NREM 期降低 83.2%^[6]。因此,NREM 期同步化振荡网络促进癫痫样放电扩散,而在 REM 期则是通过能量代谢依赖的腺苷能抑制通路发挥内源性抗痫作用。GIULIANO 等^[7]及 YEH 等^[8]研

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究发现,局灶性癫痫患者 REM 期占比显著降低,且 REM 期占比降低程度与颞叶痫样放电负荷呈现显著负相关,提示 REM 时期时长缩短可通过减弱腺苷能抑制作用,促进致痫网络的兴奋性;同时发现 NREM 期癫痫发作频率为 REM 期的 4.22 倍,揭示 NREM 期脑电同步化会让癫痫放电更容易扩散,而 REM 期的自我保护机制则能压制癫痫发作。

2 癫痫与失眠

癫痫与失眠存在双向病理促进关系。一项癫痫与睡眠紊乱的前瞻性横断面研究显示,睡眠紊乱加剧了癫痫的发作^[9]。MACÊDO 等^[10]对成年与儿童癫痫患者睡眠质量进行研究,发现癫痫患者更易出现失眠症状,其中最常见失眠症状是睡眠维持困难(6.9%~79.0%),其次是睡眠起始困难(12.9%~38.1%)。癫痫发作时外周和脑内的免疫细胞会异常活跃,释放危险信号,并产生促炎介质,包括细胞因子、补体因子、前列腺素等,引发一系列的神经炎症持续反应,最终可致神经变性^[11]。同时,促炎介质的产生也会引发睡眠障碍,进而导致失眠^[12]。而睡眠障碍也可促进神经元的丢失,致癫痫的发作频率增加和症状加剧^[13]。综上,癫痫与失眠通过神经炎症介导形成双向恶性循环。

3 癫痫与 OSA

OSA 的主要特点为发作性的呼吸暂停/低通气和觉醒状态,伴发间歇性低氧血症和睡眠剥夺。同时 OSA 也会导致睡眠结构发生变化,即 NREM 中的 N2 阶段增长、N3 阶段和 REM 时期时长缩短。癫痫发作呈显著睡眠依赖性,在 NREM 期 N2 阶段发生率最高,其机制在于丘脑-皮质纺锤波振荡促进了神经元同步放电。OSA 通过延长 N2 阶段,扩大丘脑-皮质振荡网络的兴奋性时间窗,致使局灶性癫痫发作风险增加约 3.2 倍^[14]。研究表明,未经治疗的 OSA 具有间歇性低氧血症、睡眠结构紊乱、睡眠碎片化及睡眠效率降低等临床症状,可通过激活 HIF-1 α /NF- κ B 介导的神经炎症通路并诱发线粒体功能障碍,降低癫痫发作阈值,使发作风险增加约 3.2 倍^[15]。一项针对 84 例癫痫患儿进行的前瞻性研究显示,在难治性癫痫(每月发作次数大于 1 次)患儿中,有 43.8%同时患有 OSA,而在发作频率为每月 0 次或 1 次的患儿中,约有 30.7%患有 OSA^[16]。越来越多的研究证明,OSA 的低氧血症可能会引起氧化应激,进而激活炎症通路,产生白细胞介素-6 和肿瘤坏死因子- α ,从而引起癫痫发作。然而,也有一些研究认为,低氧血症不是导致癫痫发作的主要原因,因为患有 OSA 的癫痫患者的血氧饱和度降低不够明显^[17]。基于上述研究,建议未来应加强对低氧血症能否致癫痫发作的关注,以期进一步阐明低氧血症影响癫痫发作的机制。

OSA 会加重癫痫,同样癫痫也会加重 OSA。癫痫可以通过以下方式加重 OSA:①额颞叶异常放电会抑制脑干的呼吸中枢,从而导致呼吸暂停^[18]。②抗癫痫药物会降低患者呼吸中枢的反应能力,降低呼吸道的张力,从而导致呼吸

困难^[19]。PHABPHAL 等^[20]针对 166 例癫痫患者的横断面多中心研究结果显示,OSA 的发生比例为 38%。在成人癫痫患者中,OSA 的发生率是正常对照组的 2 倍,且有随着年龄的增长而增加的趋势^[21]。

4 癫痫与发作性睡病

发作性睡病是一种原因不明的慢性睡眠障碍,其特点是白天过度嗜睡、出现无法控制的频繁睡眠发作以及睡眠片段,还可能伴有猝倒、睡眠瘫痪和睡眠幻觉。该病有两种类型:发作性睡病 1 型(以前称为发作性睡病,伴猝倒)和发作性睡病 2 型(以前称为发作性睡病,无猝倒)。发作性睡病是一种罕见且诊断不足的疾病,据分析,发作性睡病被误诊的概率高达 71.4%,其中被误诊为癫痫的概率达 35%^[22],特别是当其与癫痫共存时非常可能被忽视^[23]。YANG 等^[24]的研究发现,癫痫与发作性睡病均具有自身免疫性疾病的病理特征,自身免疫反应可能是两者共病的潜在机制。发作性睡病的发生主要受免疫相关基因的影响,该疾病与人类白细胞抗原 DQA1 * 01:02/DQB1 * 06:20 表型密切相关。癫痫和发作性睡病具有相同的 HLA-DQB1 * 06:02 发作性睡病相关等位基因,因此这可能是发作性睡病易被误诊为癫痫,且发作性睡病与癫痫共存的原因^[25]。

5 癫痫与 RLS

RLS 又称不安腿综合征、Willis-Ekbom 病,是一种神经系统疾病,其特征是无法控制移动腿部的欲望和腿部存在有不适感,尤其是在夜间,从而导致睡眠障碍。RLS 是一种因中枢铁储备降低引起的昼夜节律相关的感觉运动障碍,病变部位位于壳核和黑质。癫痫发作后海马及新皮质区铁浓度的升高会引发神经炎症,进而导致铁的重新分布,即增加了大脑新皮质中的铁沉积,减少了壳核、苍白球、黑质以及红核中的铁储备,因此癫痫患者可共患 RLS^[26]。SAFARPOUR LIMA 等^[27]分析了 70 例癫痫患者,发现有 28.6%的癫痫患者共患 RLS。KHACHATRYAN 等^[28]研究显示,癫痫患者中 RLS 的发生率(20.6%)明显高于非癫痫患者(6.1%)。

6 睡眠相关过度运动性癫痫(SHE)与 NREM 睡眠相关异态睡眠

NREM 相关异态睡眠以夜间行为异常、意识障碍以及自主神经系统激活为特征,其原因是觉醒受损,故也称为 NREM 相关觉醒障碍。SHE 又称夜间额叶癫痫,是一种罕见的异质病因综合征,其特征是与睡眠相关的刻板化的局灶性癫痫。其癫痫发作主要发生在睡眠期间,更确切地说是发生在 NREM 期。乙酰胆碱作为一种兴奋性神经递质,可以增强大脑皮质神经元的兴奋性,进而诱发觉醒,研究表明胆碱能系统在睡眠以及觉醒中起着关键作用^[29]。SHE 最常见的致病基因为与神经元烟碱型乙酰胆碱受体(nAChR)结合的基因突变^[30]。nAChR 作为配体门控阳离子通道,在中枢神经系统中具有多离子通透性,同步介导 Na⁺、K⁺跨膜流

动并显著通透 Ca^{2+} , nAChR 通过增强神经递质的兴奋性传递及提升抑制性神经传递效能来调控神经递质动态平衡。编码 nAChR 的基因若发生突变,可通过改变受体构象或通道功能,破坏神经元环路平衡,从而导致癫痫发作^[31]。邹鑫平^[32]研究发现编码 nAChR 的基因突变影响患者乙酰胆碱的代谢,进而使睡眠时间减少,睡眠碎片化,睡眠质量下降。BISULLI 等^[33]研究显示,SHE 家族中的觉醒障碍发生率较高。SHE 的发作呈刻板性运动多模式谱系:以肌张力障碍性姿势及过度运动性自动症为核心,可伴发情感性先兆以及口咽部复杂自动症^[34]。觉醒障碍通常于 NREM 期的 N3 阶段发作,SHE 主要于 NREM 期的 N2 阶段发作,N3 阶段以外的主要运动事件(过度运动性癫痫发作)对 SHE 的诊断具有较高提示价值^[35]。与觉醒障碍患者相比,SHE 患者的睡眠质量明显受损,表现为较低的睡眠效率和 N3 阶段缩短。SHE 患者在睡眠期间的运动活动度明显增加,该类患者每晚睡眠相关运动次数高于觉醒障碍患者^[36]。研究表明,与健康对照组相比,SHE 与觉醒障碍患者的循环交替模式(CAP)发生率显著升高,并且 SHE 的癫痫性事件与觉醒障碍的异常觉醒事件均与 CAP 的 A 相存在明确的时序关联性,CAP-A 相触发的皮质下-皮质网络短暂激活是两类事件共同的病理基础^[37]。

7 小结

癫痫与各种睡眠障碍之间的关系十分复杂,本文阐述了睡眠结构对癫痫的影响,总结了失眠、OSA、发作性睡病、RLS、NREM 相关异态睡眠等睡眠障碍与癫痫之间关系的相关机制,但仍需更加深入的研究以明确两者之间的具体影响机制,为更好控制癫痫发作和改善患者预后提供参考。

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动并显著通透 Ca^{2+} , nAChR 通过增强神经递质的兴奋性传递及提升抑制性神经传递效能来调控神经递质动态平衡。编码 nAChR 的基因若发生突变,可通过改变受体构象或通道功能,破坏神经元环路平衡,从而导致癫痫发作^[31]。邹鑫平^[32]研究发现编码 nAChR 的基因突变影响患者乙酰胆碱的代谢,进而使睡眠时间减少,睡眠碎片化,睡眠质量下降。BISULLI 等^[33]研究显示,SHE 家族中的觉醒障碍发生率较高。SHE 的发作呈刻板性运动多模式谱系:以肌张力障碍性姿势及过度运动性自动症为核心,可伴发情感性先兆以及口咽部复杂自动症^[34]。觉醒障碍通常于 NREM 期的 N3 阶段发作,SHE 主要于 NREM 期的 N2 阶段发作,N3 阶段以外的主要运动事件(过度运动性癫痫发作)对 SHE 的诊断具有较高提示价值^[35]。与觉醒障碍患者相比,SHE 患者的睡眠质量明显受损,表现为较低的睡眠效率和 N3 阶段缩短。SHE 患者在睡眠期间的运动活动度明显增加,该类患者每晚睡眠相关运动次数高于觉醒障碍患者^[36]。研究表明,与健康对照组相比,SHE 与觉醒障碍患者的循环交替模式(CAP)发生率显著升高,并且 SHE 的癫痫性事件与觉醒障碍的异常觉醒事件均与 CAP 的 A 相存在明确的时序关联性,CAP-A 相触发的皮质下-皮质网络短暂激活是两类事件共同的病理基础^[37]。

7 小结

癫痫与各种睡眠障碍之间的关系十分复杂,本文阐述了睡眠结构对癫痫的影响,总结了失眠、OSA、发作性睡病、RLS、NREM 相关异态睡眠等睡眠障碍与癫痫之间关系的相关机制,但仍需更加深入的研究以明确两者之间的具体影响机制,为更好控制癫痫发作和改善患者预后提供参考。

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