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# 短暂单侧神经痛样头痛发作的诊疗进展

董青<sup>1</sup>综述, 李明欣<sup>2</sup>审校

**摘要:** 短暂单侧神经痛样头痛发作(SUNHA)是罕见致残性原发性头痛,属于三叉神经自主神经性头痛(TACs),含短暂单侧神经痛样头痛发作伴结膜充血及流泪(SUNCT)和短暂单侧神经痛样头痛发作伴脑自主神经症状(SUNA)两个亚型。其特征为单侧(多V1分布)剧烈刺痛/电击样痛(持续1~600 s),发作频繁(日数次至数百次),伴显著同侧脑自主神经症状(如结膜充血、流泪、鼻塞等)。86%患者存在触发因素。诊断需符合国际头痛疾病分类第三版(ICHHD-3)标准(≥20次发作),并以头部MRI(尤其垂体/后颅窝等)排除继发性病因(如血管压迫、垂体瘤)。预防性治疗首选拉莫三嗪预防,在预防性治疗起效前可用利多卡因作为过渡性治疗,难治性病例可考虑枕神经刺激、深部脑刺激或微血管减压术。提高认识、精准鉴别(如三叉神经痛/其他TACs)及个体化治疗至关重要。

**关键词:** 短暂单侧神经痛样头痛发作; 三叉神经自主神经性头痛; 脑自主神经症状; 三叉神经-下丘脑通路  
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**Advances in the diagnosis and treatment of short-lasting unilateral neuralgiform headache attacks** DONG Qing, LI Mingxin. (Department of Neurology, the Third Affiliated Hospital of Sun Yat-sen University, Guangzhou 510630, Guangdong, China)

**Abstract:** Short-lasting unilateral neuralgiform headache attacks (SUNHA) are a rare type of disabling primary headache within the category of trigeminal autonomic cephalalgias (TACs), and it has two subtypes of SUNCT (with conjunctival injection and tearing) and SUNA (with other autonomic features). SUNHA is characterized by severe unilateral (often V1) stabbing/shock-like pain (lasting for 1–600 s), high frequency (2–600 attacks a day), and prominent ipsilateral cranial autonomic symptoms (such as conjunctival injection, tearing, and nasal obstruction). Trigger factors are observed in 86% of patients. The diagnosis of SUNHA should meet the ICHD-3 criteria (≥20 attacks), and brain MRI (especially for the pituitary gland/posterior cranial fossa) should be performed to exclude secondary causes (such as neurovascular conflict and pituitary tumor). Lamotrigine is used as first-line prophylaxis, while lidocaine aids acute relief in the transitional phase; occipital nerve stimulation, deep brain stimulation, or microvascular decompression can be used for refractory cases. It is of great importance to enhance awareness, achieve precise differentiation (from trigeminal neuralgia or other types of TACs), and provide individualized treatment.

**Key words:** Short-lasting unilateral neuralgiform headache attacks; Trigeminal autonomic cephalalgias; Cranial autonomic symptoms; Trigeminothalamic pathway

国际头痛疾病分类第三版(international classification of headache disorders, 3rd edition, ICHD-3)中,从集性头痛(cluster headache, CH)、阵发性偏侧头痛(paroxysmal hemicrania, PH)、短暂单侧神经痛样头痛发作(short-lasting unilateral neuralgiform headache attacks, SUNHA)、持续性偏侧头痛同属于三叉神经自主神经性头痛(trigeminal autonomic cephalalgias, TACs)<sup>[1]</sup>。SUNHA是罕见的致残性原发性头痛,疼痛通常位于三叉神经第一支(V1)分布区,其特征是严重的固定单侧的短暂性头痛,伴有同侧突出的脑自主神经症状和体征。疼痛持续时间通常1~600 s,疼痛性质为尖锐、刺痛、灼烧样或电击样痛。根据相关自主神经症状,SUNHA又被分为2个亚型,分别叫伴结膜充血/流泪的短暂单侧神经痛样头痛发作(short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing, SUNCT)和伴其

他自主神经症状的短暂单侧神经痛样头痛发作(short-lasting unilateral neuralgiform headache attacks with autonomic symptoms, SUNA)<sup>[1-4]</sup>。1978年首次描述了SUNCT。后来SUNA被报道。虽然SUNCT表现出更突出的脑自主神经特征,且比SUNA更容易触发,很多学者认为这2种综合征之间可能没有差异,而且这两种表型可能属于同一谱系。本文根据Arca等<sup>[2]</sup>、Duggal等<sup>[3]</sup>、Kang等<sup>[4]</sup>及其他相关文献进行整理形成。

## 1 流行病学

由于SUNHA很少见,其基于人群的患病率尚不

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作者单位:(1. 中山大学附属第三医院神经内科,广东 广州 510630;

2. 山东大学齐鲁医院神经内科,山东 济南 250012)

通信作者:李明欣, E-mail: leemingsin@163.com

明确,现有的证据主要来自临床观察性研究。从1978年的第1次描述到2003年,仅报告了50例病例<sup>[5]</sup>。SUNHA的估计患病率为0.32%<sup>[6]</sup>。典型的发病年龄为40~70岁;平均发病年龄为57岁<sup>[5]</sup>。SUNHA从儿童到老年都有报道<sup>[7]</sup>。最年轻的SUNCT患者为3岁,最年长的患者为88岁<sup>[8,9]</sup>。开始的研究曾经认为SUNA主要发生在女性身上,而SUNCT更常见于男性,但后来的报告并不支持SUNHA存在性别方面的差异<sup>[10-13]</sup>。家族性SUNCT综合症的病例也有报告<sup>[14]</sup>。

## 2 病理生理机制

SUNHA的确切病理生理机制尚不清楚。参与SUNCT发病机制的主要结构是三叉神经血管系统、脑自主神经系统和下丘脑<sup>[15]</sup>。SUNCT和三叉神经痛之间的临床相似性,以及影像学上许多SUNCT患者出现同侧三叉神经血管压迫,都表明有外周机制的参与<sup>[16]</sup>。神经血管接触可能导致三叉神经局灶性脱髓鞘,导致自发异位冲动,从而导致短暂的自发和皮肤触发的发作<sup>[15]</sup>。SUNCT患者的MRI纤维束成像显示,血管压迫后三叉神经的结构存在变化。对SUNHA的高分辨率MRI研究发现,与无症状侧相比,有症状侧的三叉神经有更多的血管接触。特别是在近端和上部,症状侧与动脉的接触更大<sup>[17]</sup>。三叉神经尾侧核与C1-C2背角组成三叉神经颈复合体(trigemino-cervical complex, TCC),三叉神经被激活后,其传入信号投射到TCC的二级神经元,后者再投射到脑桥上泌涎核。上泌涎核发出节前副交感神经纤维,沿面神经及其分支岩浅大神经,最终在翼腭神经节形成突触<sup>[18,19]</sup>。随后节后副交感神经纤维沿着面神经第二支到达泪腺,并通过尾侧鼻支和腭支支配鼻黏膜和腭<sup>[2]</sup>。三叉神经自主反射造成与SUNHA相关的自主神经症状<sup>[20]</sup>。

SUNCT和SUNA的功能成像研究显示<sup>[21-23]</sup>,三叉神经和下丘脑之间存在三叉神经下丘脑通路,三叉神经尾侧核可以激活下丘脑后部。下丘脑后部可以调节三叉神经尾侧核的活动。下丘脑功能障碍可能导致伤害性食欲素B等的过度产生,从而激活三叉神经下丘脑通路<sup>[7,24,25]</sup>。TCC二级神经元上行投射到丘脑,最后传递到调节疼痛的大脑,皮质区域就会随之激活,导致剧烈疼痛<sup>[26]</sup>。延髓背外侧梗死患者表现出继发性SUNCT,支持三叉神经下丘脑通路部分性功能障碍可能导致TAC症状<sup>[27,28]</sup>。SUNHA患者头痛发作同侧的腹侧被盖区(ventral tegmental area, VTA)接受脑深部电刺激(deep brain stimulation, DBS)治疗后,发作频率和发作严重程度均得到改善,支持VTA参与SUNHA的病理生理过程。而学者们认为VTA和下丘脑后部实际上可能是同一个区域。

## 3 临床表现

SUNHA的特征是通常位于三叉神经第一支(V1)分布区、突然出现、短暂、单侧的严重疼痛,伴有同侧脑自主神经症状。62%的SUNCT患者和56%的SUNA患者可能会出现不安和躁动<sup>[29,30]</sup>。

3.1 脑自主神经症状 SUNHA与三叉神经痛(TN)的区别在于其相关的自主神经特征,SUNCT必须伴有结膜充血和流泪,也可以有或没有其他自主神经症状,而SUNA仅伴一种或无结膜充血或流泪,但有其他自主神经症状<sup>[1]</sup>。其他自主神经症状包括鼻塞、流涕、瞳孔缩小、上睑下垂、眼睑水肿、额头和面部出汗、和面部发红,以及耳朵有闷胀感<sup>[7,31-33]</sup>。上睑下垂和流涕在SUNCT中更常见。颅自主神经症状往往与头面部疼痛同侧,但约15%的病例颅自主神经症状是双侧的<sup>[19,30]</sup>。V1分布的疼痛与更严重的结膜充血和流泪有关,而V2和V3分布的疼痛则与更多的流涕和鼻塞有关。

3.2 疼痛部位 SUNHA的疼痛通常是单侧的,通常位于V1分布区,如眼眶、眶后、眶上或颞区。某些患者疼痛可能始于另一个区域,并辐射到V1分布区域,也可以从一侧鼻孔开始,放射到眉毛的内侧边缘,进入眶上区。有时候疼痛可能在V2分布区,较少在V3分布区。很少出现三叉神经区域外的疼痛,如C2-C3感觉分布区的疼痛<sup>[30]</sup>。大多数患者为固定于一侧的疼痛,但10%~15% SUNHA患者可能有双侧交替发作<sup>[11,19,30]</sup>。

3.3 疼痛性质 SUNHA的疼痛通常很重,患者常描述为其经历过的最痛苦的疼痛,SUNCT通常比SUNA更重<sup>[11,29,30]</sup>。部分患者在发作期间更喜欢保持静止不动,可能是为了避免皮肤触发疼痛发作<sup>[30]</sup>。疼痛通常为刺痛、尖锐痛、枪击样或电击样疼痛<sup>[11,29,30]</sup>。有的患者同时描述为搏动痛或跳痛<sup>[30]</sup>。

3.4 发作模式 很多SUNHA患者可能有不止一种发作。最常见的是单个刺痛模式,平均持续时间为58 s(1~600 s),近40%患者为这种模式;另一种为成组刺痛模式,刺痛快速连续发生,单个刺痛持续时间相同,但成组刺痛可能持续长达20 min;还有一种为锯齿模式,疼痛发作没有无痛间隔,刺痛之间的疼痛不恢复到基线,平均持续时间为1 160 s(5~12 000 s)的背景性疼痛,并伴有典型的刺痛<sup>[7,34]</sup>;另有一种为平顶模式,疼痛在2~3 s内达到最大值,平均持续时间约为300 s;甚至在平顶模式的基线痛上随机叠加持续1~2 s的超短刺痛的叠加超短刺痛的平顶模式<sup>[35]</sup>。

3.5 发作频率 频率2~600次/d,但平均<100次/d。一些患者可能几乎持续发作,发作次数超过30次/h,可能会在1 d的大部分时间里引起疼痛,无

疼痛间隔很短,导致几天严重失能,成为SUNHA的一种持续状态样表现<sup>[3]</sup>。尽管发作可能存在季节性变化,但是通常没有年度的周期性<sup>[30,36]</sup>。SUNHA可以在1 d中的任何时间发生,没有明显的昼夜节律。大多数SUNHA患者在清醒时(54%)发作,在清醒和睡眠时(44%)也会发作。纯粹夜间发作的患者很少(<2%)<sup>[29,30]</sup>。

**3.6 发作性和慢性** 绝大多数SUNHA(约90%)患者,发作时间超过1年,没有缓解或缓解期持续不到1个月。约10%的病例平均每年1~4次发作,持续时间为1周~10个月。随着病情的进展,发作性SUNA可能会在平均8.4年内转变为继发性慢性形式<sup>[29]</sup>。

**3.7 触发因素和不应期** 86%患者既有自发发作,也有触发性发作<sup>[29]</sup>。12%~14%的SUNCT和27%的SUNA患者仅经历自发发作。SUNCT患者偶尔可能只有触发性发作(<5%)<sup>[29]</sup>。触发区域几乎总是位于疼痛的同侧。常见的触发因素包括三叉神经的任何刺激,触摸面部或头皮、洗澡或淋浴、洗头、刮胡子、擤鼻涕、咀嚼或进食、温度变化、噪声暴露、鼻呼吸、说话、咳嗽、运动和光线<sup>[7,11,29,30,37]</sup>。与三叉神经痛相比,大多数SUNCT/SUNA患者没有不应期。在近80%~95%的SUNCT/SUNA患者,发作可以在前一次发作停止后立即触发<sup>[11,29,30,38]</sup>。但2%~4%的SUNCT、3%~18%SUNA在某些情况下可能存在不应期<sup>[38]</sup>。

**3.8 间期疼痛** 两次疼痛之间常常没有疼痛。一些SUNHA患者在发作间期可能会有与发作期疼痛同侧的无特征性的背景性疼痛。这种发作间期疼痛通常在刺痛发作的时候就开始出现。47%~50%的患者会出现发作间期疼痛<sup>[11,29,30,38]</sup>。

**3.9 偏头痛特征** 一些SUNHA患者可能有偏头痛样症状,如恶心和呕吐。约27%的患者有畏光和畏声<sup>[29]</sup>。很少患者可能有先兆,面部同侧有刺痛感的感觉先兆更常见,但也有报道1例患者有视觉先兆<sup>[30]</sup>。

## 4 诊断

ICHD-3头痛国际分类中,SUNHA的诊断是基于临床的诊断<sup>[1]</sup>,需至少20次发作,发作期超过1/2的时间头痛发作频率至少为1次/d,中重度单侧通常位于三叉神经分布区的持续1~600 s的单个刺痛、连续刺痛或锯齿样模式,伴有自主神经症状。根据相关自主神经症状,SUNHA又被分为SUNCT和SUNA 2个亚型。SUNCT同时出现结膜充血和流泪,也可以有或没有其他自主神经症状,而SUNA只具有一种或没有结膜充血或流泪,但有其他自主神经症状<sup>[1]</sup>。根据有无缓解期,缓解期以3个月为界分为

发作性或慢性类型。神经系统检查通常是正常的,偶尔出现同侧三叉神经感觉异常。三叉神经V1或V2分布中的针刺样异常感觉在SUNCT患者中占88%,在SUNA患者中占78%<sup>[29]</sup>。如果存在明显的局灶性神经功能缺损,要考虑查找继发性原因。鉴于继发性原因的复杂性,所有患者都必须接受详细的头部MRI检查,特别是检查垂体、海绵窦和三叉神经。

## 5 鉴别诊断

SUNHA跟其他的TACs相鉴别。虽然SUNHA的典型持续时间<10 min,但与PH有一些重叠,后者通常持续时间为2~30 min。重复刺痛或锯齿型发作会持续更长时间,需与CH鉴别<sup>[1]</sup>。临床上持续时间较短的模式可能被误诊为三叉神经痛,而持续时间较长的模式可能会被误诊为PH和CH<sup>[35]</sup>,CH或PH患者不会出现重复刺痛或锯齿型头痛,因此一个重要鉴别点是,不光要看每次发作的持续时间,更重要的是注意每天的疼痛中以分钟计的发作,即单个刺痛的持续时间,平均持续时间为1 min<sup>[29]</sup>。与CH不同,大多数SUNHA患者在发作期间更喜欢保持静止不动,可能是为了避免皮肤触发疼痛发作<sup>[30]</sup>。对吡啶美辛或100%氧气缺乏反应的可疑病例更支持SUNHA的诊断<sup>[39,40]</sup>。

原发性针刺样头痛通常不在三叉神经支配的区域,发作中疼痛位置可以发生变化,并且非常短暂,持续时间不超过1 s<sup>[41]</sup>。自发性疼痛是原发性刺痛样头痛的主要症状,通常没有任何伴随的自主神经症状。而SUNHA遵循单侧三叉神经分布,大多数持续时间超过10 s,且伴有自主神经症状,三叉神经支配区域的皮肤刺激可触发发作<sup>[42,43]</sup>。

头皮闪痛是引起超级短暂头痛的另一个原因,其特征是短暂的(1~50 s)过电样、针刺样、烧灼性疼痛发作,从前到后或从后到前呈线性或之字形放射。特征性的线性或之字形放射性疼痛和缺乏脑自主神经症状有助于与SUNHA鉴别<sup>[3]</sup>。

SUNHA最重要的鉴别诊断是三叉神经痛(TN)。TN和SUNHA之间存在相当大的临床重叠,甚至一种观点认为TN和SUNHA可能是同一疾病的连续体<sup>[16]</sup>。三叉神经痛主要影响三叉神经第二或第三分布区。三叉神经痛通常缺乏自主神经症状,且有不分期,在此期间触发因素几乎不可能引发另一次发作。

## 6 继发性SUNHA

SUNHA最常见的继发性原因是颅后窝病变,如压迫三叉神经根移行区的血管环、动静脉畸形、海绵状血管瘤、多发性硬化、梗死、感染、占位性病变,甚至颅骨异常<sup>[29,33,44-46]</sup>。一项文献综述中201例经头部

MRI评估的SUNHA病例,34例患者有三叉神经血管受压的证据<sup>[47]</sup>。压迫三叉神经的常见血管是小脑前下动脉或小脑上动脉<sup>[47]</sup>。延髓背外侧缺血性损伤也导致继发性SUNCT<sup>[27,28]</sup>。垂体微腺瘤和大腺瘤也可以引起SUNCT样头痛。头痛症状可能比垂体症状早3~10年<sup>[29,48-51]</sup>。垂体病变通常位于疼痛侧的同侧,机械性和神经激素机制可能是导致SUNCT的原因<sup>[48,52]</sup>。

## 7 治疗

由于这些疾病的罕见性,目前还没有随机对照试验研究,大多数治疗都是基于观察性研究、病例系列和病例报告。SUNHA的发作持续时间很短,治疗主要是预防性治疗,其效果可能会有几天至几周的延迟。在等待预防性治疗起效前,过渡性治疗可以在有限的时间内快速抑制发作。

**7.1 过渡性治疗** 静脉注射或皮下注射利多卡因对SUNHA有效<sup>[5,53]</sup>。系统综述文献中发现,利多卡因对94%的患者有效,其中80%的患者可实现完全无痛<sup>[32]</sup>。静脉注射利多卡因对周围神经末梢有影响,也可能减少中枢致敏。口服或静脉注射皮质类固醇的疗效存在争议<sup>[13,38,54]</sup>。短期使用类固醇治疗SUNCT,特别是甲基泼尼松龙,成功率各有不同<sup>[32]</sup>。

枕神经阻滞(greater occipital nerve block, GON)对SUNHA的疗效存在争议。枕神经阻滞使用80 mg甲基泼尼松龙和2 ml 2%利多卡因,有效率为27%<sup>[13]</sup>。推测枕神经阻滞可能引起TCC的神经调控并减轻疼痛。

肉毒杆菌毒素疗法也可能对难治性SUNHA有效,其疗效与治疗三叉神经痛相似<sup>[37]</sup>。抗降钙素基因相关肽单克隆抗体erenumab和galcanezumab对SUNCT的作用,表明二者可能有治疗难治性SUNHA的作用<sup>[55,56]</sup>。

**7.2 预防性治疗** 拉莫三嗪是预防性治疗最有效的药物,被认为是SUNHA管理中的一线药物。使用拉莫三嗪时需要缓慢滴定剂量。有研究报告5例SUNCT患者对拉莫三嗪的反应良好,并发现剂量效应关系,在200 mg/d的较高剂量下效果显著<sup>[57]</sup>。拉莫三嗪在68%的SUNCT患者中有良好的反应,但在SUNA患者中效果较差(25%)<sup>[13,30]</sup>。拉莫三嗪对发作性SUNCT的疗效更好,但对慢性SUNHA的疗效可能较差,如果无效可能需要高剂量的药物<sup>[47]</sup>。

卡马西平也是SUNHA中常用的药物,但只有33%的SUNCT患者对卡马西平有反应<sup>[5]</sup>。奥卡西平对近59%的SUNHA患者有效<sup>[58]</sup>。在开放标签研究

中,托吡酯对约50%的SUNHA患者有效<sup>[11,59]</sup>。与SUNA患者相比,托吡酯对SUNCT患者更有效<sup>[38]</sup>。加巴喷丁反应率为27%~48%,SUNA患者比SUNCT患者可能获益更多<sup>[5,32,60]</sup>。

当潜在的病理是血管压迫三叉神经时,微血管减压术是继发性SUNHA的治疗方法,微血管减压术在75%的病例中有效<sup>[47,61,62]</sup>。在2例难治性SUNCT中,神经调控方法失败后,微血管减压术是有益的<sup>[63]</sup>。

枕神经刺激(occipital nerve stimulation, ONS)被认为是难治性SUNCT和SUNA的潜在选择。在2个小病例系列中,几乎所有患者都对枕神经刺激有反应<sup>[3,64]</sup>。在31例患者的大型病例系列中,在平均随访44.9个月时,枕神经刺激的50%反应率为77%,发作频率下降了69%<sup>[34]</sup>。

下丘脑后部或腹侧中脑被盖的DBS对3例顽固性SUNCT患者产生了良好的效果,治疗后12~18个月内几乎没有疼痛<sup>[65,66]</sup>。另一项研究,平均随访29个月,50%的应答率为82%,每日发作平均减少78%<sup>[67]</sup>。9例难治性SUNHA患者接受翼腭神经节脉冲射频术,随访6个月,有效率为78%<sup>[68]</sup>。

## 8 总结与展望

SUNCT/SUNA作为罕见却高度致残的三叉神经自主神经性头痛,其临床特征已逐渐明确:单侧V1分布区短暂剧痛(1~600 s)、高频发作(日数次至数百次)、伴同侧脑自主神经症状(SUNCT必备结膜充血+流泪)是核心诊断依据。病理生理机制涉及三叉神经血管压迫的外周机制与三叉神经-下丘脑通路的中枢调控紊乱,继发性病因需重点排查颅后窝血管压迫及垂体病变等。拉莫三嗪是一线预防性药物,难治性病例可考虑神经调控(ONS/DBS)或微血管减压术。未来需要进一步深入探索SUNHA的病理生理机制,多学科协作提高诊断的准确性,优化治疗策略,提高对该病的认识、实现个体化分层治疗改善患者预后。

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