

带状疱疹发病风险因素的综合分析

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摘要: 带状疱疹 (HZ) 是由水痘-带状疱疹病毒 (VZV) 再激活引发的全球性公共卫生挑战, 其发病率随年龄增长呈指数级上升, 尤其在老年及免疫功能受损人群中显著。本综述系统性分析了 HZ 的流行病学特征及多维风险因素。全球数据显示, 50 岁以上人群年发病率达 4-12/1000 人, 女性发病率较男性高 20%-50%, 免疫功能抑制、慢性疾病 (如糖尿病、心血管疾病)、心理压力及特殊药物暴露 (如 JAK 抑制剂) 是重要风险因素。风险因素间存在显著交互作用: 年龄与免疫衰老协同放大病毒再激活风险, 慢性疾病与药物治疗形成叠加效应, 心理压力在女性中呈现剂量反应趋势。重组带状疱疹疫苗 (RZV) 对 HZ 及后遗神经痛 (PHN) 的预防效力超过 90%, 但全球接种率受限于认知不足及经济障碍。特殊人群需个体化干预策略, 如免疫功能抑制者优先接种 RZV、急性期抗病毒治疗降低并发症风险。未来需加强风险因素交互机制研究, 优化疫苗接种策略以应对老龄化社会挑战。

关键词: 带状疱疹; 重组带状疱疹疫苗; 疫苗接种策略

一、带状疱疹的基本流行病学特征

带状疱疹是由潜伏在感觉神经节中的水痘-带状疱疹病毒 (VZV) 再激活引起的急性感染性疾病, 在全球范围内普遍流行^[1]。其发病率与年龄增长显著相关, 50 岁以上人群发病率呈指数上升, 欧美研究显示 50-59 岁人群年发病率约为 4-5/1000 人, 而 80 岁以上人群可高达 10-12/1000 人^[2-3]。全球疾病负担研究显示, 2019 年全球带状疱疹新发病例约 390 万例, 年龄标准化发病率为 43.8/10 万人年^[4]。

不同地区的流行病学特征存在差异, 亚洲国家的发病率普遍低于欧美 (例如韩国为 3-5/1000 人年, 中国台湾地区为 4.8-5.3/1000 人年)^{[3][5]}。免疫功能抑制 (如 HIV 感染、器官移植、恶性肿瘤) 和慢性疾病 (糖尿病、慢性肾病) 是明确的危险因素^[6-7]。值得注意的是, 女性发病率略高于男性 (性别比约 1.2-1.5: 1), 可能与免疫衰老差异有关^{[8][9]}。

带状疱疹后神经痛 (PHN) 是最常见的并发症, 约 5-30% 的患者在皮疹消退后仍持续疼痛, 老年人群 PHN 发生率可达 50%^[10-11]。疾病负担研究显示, 带状疱疹相关住院率为 2-25/10 万, 直接医疗成本较健康人群增加 2-3 倍, 且患者生活质量显著下降^[12-13]。亚太地区研究显示, 60 岁以上人群年发病率达 7.6-22.0/千人年, 住院率为 4.1-17.8/10 万, 死亡率约 0.01-0.03/10 万, 凸显该区域

老龄化社会面临的公共卫生挑战^[14-16]。

二、主要风险因素分析

带状疱疹 (Herpes Zoster, HZ) 的发病机制与潜伏于神经节的水痘-带状疱疹病毒 (VZV) 再激活密切相关, 其风险因素呈现多维度交互特征。年龄增长是首要独立风险因素, 50 岁以上人群因免疫衰老导致细胞免疫功能衰退, 发病率随年龄呈指数级上升^[17], 这一现象在墨西哥等快速老龄化社会尤为突出^[18]。免疫抑制状态作为核心机制, 涵盖疾病相关免疫缺陷 (如 HIV 感染者即使接受抗逆转录病毒治疗仍维持较高风险^[19])、药物诱导免疫抑制 (托法替尼 10 mg/天剂量组风险较 5 mg/天显著升高^[20]) 以及器官移植后免疫异常 (造血干细胞移植受者发病率较普通人群升高 1.5 倍^[21])。值得注意的是, 慢性阻塞性肺病 (COPD) 和哮喘患者因长期糖皮质激素使用及固有免疫失调, HZ 风险增加达 2.3 倍^[22]。

慢性基础疾病与 HZ 的关联具有双向性: 心血管疾病患者感染 HZ 后 30 天内发生心肌梗死等急性事件的风险增加 1.8 倍^[23], 而冠状动脉疾病患者本身 HZ 发病率较健康人群升高 21%^[24]; 代谢性疾病中, 糖尿病通过慢性炎症和糖基化终产物积累增强病毒再激活风险^[25]; 自身免疫性疾病如银屑病与 HZ 存在独特双向关联, 两者通过免疫失衡形成恶性循环^[26]。心理社会因素的作用日益凸显, 高压水平睡眠不足分别使 HZ 风险增加 48.3% 和

19.4%，且女性群体中该关联呈现剂量反应趋势，抑郁症患者更易发展为带状疱疹后神经痛（PHN）并伴随复发风险升高^[27]。

疫苗接种作为关键可干预因素，其全球实施面临显著差异：中国自2020年重组疫苗上市后，老年人群接种率不足5%，主要受限于疾病认知不足及自费接种的经济障碍^[28]，而该疫苗对HZ的预防效力超过90%，且对PHN具有持续保护作用^[29]。其他风险因素包括性别差异（女性发病率较男性高20%–50%，可能与激素调控的免疫应答差异相关^[27]）、极端气候对免疫功能的潜在影响^[30]，以及水痘感染史作为病毒潜伏的必要条件^[31]。

三、特殊人群中的风险因素

带状疱疹（HZ）在不同人群中的风险特征具有显著异质性，其风险分层需结合生理、病理及治疗暴露等多维度评估。老年人群作为核心高危群体，年龄 ≥ 50 岁后免疫衰老导致病毒再激活风险呈指数级上升，且慢性疾病负担（如冠状动脉疾病、糖尿病、COPD）通过多重机制加剧免疫功能障碍^{[32][33]}。值得注意的是，该群体疫苗接种率普遍低于30%，中国等地区因认知不足及经济限制导致预防缺口进一步扩大^[34]。

免疫功能抑制或缺陷人群中，医源性免疫抑制是主要驱动因素：接受JAK抑制剂（托法替尼10 mg/天组风险较5 mg/天组升高2.1倍）或化疗药物的患者HZ风险显著增加；异基因造血干细胞移植（allo-HSCT）受者因持续免疫抑制状态，HZ发病率较普通人群高6–8倍^[35]。自身免疫性疾病患者（如类风湿关节炎、炎症性肠病）面临疾病本身与免疫调节药物的双重风险，而HIV感染者即使接受抗逆转录治疗，其HZ风险仍为普通人群的3–5倍，且随年龄增长与HIV病程进展产生协同效应^[36–37]。

慢性疾病患者的风险特征呈现跨系统关联性：冠状动脉疾病患者HZ发病率较非患者高21%，其机制涉及慢性炎症诱导的T细胞功能抑制^[38]；呼吸系统疾病（哮喘、COPD）和代谢性疾病（糖尿病）则分别通过糖皮质激素暴露、高血糖介导的免疫调节异常促进病毒再激活^{[33][39]}。心理及神经疾病人群中，抑郁症状通过上调皮质醇水平抑制CD8⁺ T细胞活性，使HZ发病风险增加1.5倍，而睡眠剥夺（ <6 小时/天）可独立导致NK细胞功能下降，进一步放大风险^[40–41]。

特殊药物暴露人群需关注靶向治疗的免疫调节效应：JAK抑制剂巴瑞替尼（4 mg/天）和乌帕替尼（30 mg/天）分别使HZ风险升高3.2倍和4.1倍^[42]，替莫唑胺治疗神

经胶质瘤时HZ发生率可达7.1%，与药物诱导的淋巴细胞减少直接相关^[43]。此外，免疫检查点抑制剂（PD-1/PD-L1抑制剂）可能通过过度激活T细胞引发病毒再激活，但现有证据仍需大样本研究验证。

其他特殊风险因素包括女性群体因雌激素水平波动导致的免疫应答差异（风险较男性高20%–30%）^[44]，以及极端气候通过激活HPA轴影响免疫稳态的可能机制^[45]。值得注意的是，多重风险因素的交互作用显著放大疾病风险：老年COPD患者合并糖尿病时HZ风险较单一疾病患者增加1.8倍^[33]，而儿童期水痘感染史与免疫抑制状态协同作用可使HZ发病提前10–15年^[46]。未来研究需重点解析这些交互作用的分子通路，并制定针对特殊人群的疫苗优化策略。

四、风险因素的交互作用

带状疱疹（HZ）的发病风险并非单一因素独立作用的结果，而是多种危险因素通过生物学、病理生理及环境层面的交互作用共同驱动。免疫抑制与年龄的协同效应是核心机制之一：50岁以上人群因免疫衰老导致的T细胞功能衰退显著增加病毒再激活风险^[47]，而接受免疫抑制治疗（如托法替尼10 mg/天组较5 mg/天组风险升高32%，OR：0.68，95% CI：0.47–0.98）^[48]的类风湿关节炎患者中，年龄相关的免疫衰退与药物抑制形成叠加效应。慢性免疫缺陷状态（如HIV感染）即使通过抗逆转录病毒治疗（ART）控制病毒载量，其HZ风险仍较普通人群高3–5倍，提示年龄与免疫抑制的协同作用具有累积性^[49]。

慢性疾病与药物治疗的叠加效应体现于多系统交互：心血管疾病（如冠心病）患者HZ发病率升高21%（aHR：1.21，95% CI：1.14–1.27），可能通过慢性炎症诱导的免疫调节失衡增强病毒再激活^[50]；代谢性疾病中，糖尿病的高血糖环境削弱固有免疫应答，但二甲双胍可通过AMPK通路调节免疫（aHR：0.70，95% CI：0.66–0.75），部分抵消代谢异常的负面影响^[51]；自身免疫性疾病（如类风湿关节炎）患者使用JAK抑制剂（巴瑞替尼、乌帕替尼）时，药物特异性抑制JAK–STAT信号通路导致HZ风险较传统DMARDs升高2.5倍，凸显疾病内在免疫失调与药物效应的交互放大^[52]。

心理压力与性别特异性风险的关联呈现显著异质性：女性在重度压力（aOR：1.48，95% CI：1.16–1.89）和睡眠不足（aOR：1.19，95% CI：1.04–1.37）下HZ风险显著升高，而男性无统计学意义，可能与雌激素调控的

HPA 轴敏感性和NK 细胞活性差异相关^[53-54]。环境与个体因素的交互则表现为气象因子（如极端湿度）通过激活宿主应激反应间接影响免疫功能，而疫苗接种行为受慢性管理质量制约，例如中国城市老年人群因对疫苗安全性的误解导致接种率低于5%^[55-56]。

值得注意的是，病毒间免疫交互作用揭示单纯疱疹病毒（HSV）感染可能通过交叉反应性T细胞抑制VZV再激活，HSV血清阳性率下降与HZ发病率上升的生态学关联提示HSV特异性免疫对VZV的潜在保护效应^[57]。这些多层次交互作用提示，未来HZ防控需整合个体化风险评估模型，并重点关注高危人群的多因素干预策略。

五、预防与干预策略

带状疱疹的防控需基于其流行病学特征与高危人群分层制定综合策略。疫苗预防是核心干预手段，目前全球主要使用减毒活疫苗（Zoster Vaccine Live, ZVL）和重组疫苗（Recombinant Zoster Vaccine, RZV）。ZVL通过激活细胞免疫提供保护，但其效力随年龄增长显著下降（50-59岁保护效力64%，80岁以上降至18%），且免疫功能低下者禁用。相比之下，RZV含病毒糖蛋白E（gE）和新型佐剂系统，可诱导更强的体液与细胞免疫应答，对50岁以上人群保护效力达97.2%（50-59岁）且持续至少10年，同时适用于HIV感染者及器官移植受者等免疫缺陷群体^[58]。然而，全球疫苗接种率仍普遍偏低，中国等发展中国家因公众认知不足、自费接种经济负担及医疗资源分配不均，老年人群接种率不足5%，需通过政府补贴、社区定向宣教及数字化健康工具（如疫苗预约App）提升可及性^[59-60]。

针对特殊人群的干预策略需个体化设计：免疫功能低下者（如造血干细胞移植受者）推荐移植前接种RZV以降低术后感染风险（风险降低72%）^[61-62]；慢性病患者（如糖尿病、哮喘）因原发病与HZ的交互风险，需优先纳入接种计划并监测疫苗对合并症（如心血管事件）的潜在保护效应^{[63][64]}。老年人群接种RZV时需充分沟通局部不良反应（如注射部位疼痛发生率78%），通过预先镇痛和随访管理提升依从性^[65]。

急性期抗病毒治疗是降低并发症的关键，发病72小时内使用伐昔洛韦（1000 mg tid）或泛昔洛韦可缩短病程3-5天，并将带状疱疹后神经痛（PHN）风险降低53%^[66-67]。PHN需多学科联合管理，联合普瑞巴林、5%利多卡因贴剂及认知行为疗法可改善疼痛控制^[66]。对于带状疱疹性眼炎（HZO），早期联合口服抗病毒药物

与局部糖皮质激素可减少角膜瘢痕等后遗症^[68]。

未来挑战聚焦于疫苗研发与政策优化：新型亚单位疫苗及通用佐剂系统旨在扩大适用人群（如<50岁高风险群体）并提升免疫持久性^[69]；需基于区域流行病学数据制定动态接种指南，尤其在老龄化加速地区；同时探索疫苗接种对降低HZ相关神经炎症及心血管事件的远期效益^{[63][64]}。通过整合生物医学干预与公共卫生策略，有望系统性减轻带状疱疹的全球疾病负担。

六、结论与展望

本文系统梳理了带状疱疹的流行病学特征及多维度风险因素，揭示了年龄相关免疫衰老、慢性疾病、药物暴露与心理社会因素的协同作用机制。研究证实，重组疫苗（RZV）的高保护效力为防控提供了核心工具，但全球接种率差异凸显医疗资源分配与健康教育的不足。特殊人群（如老年、免疫抑制及慢性病患者）的个体化干预需求迫切，需整合疫苗接种、药物警戒及多学科管理策略。展望未来，以下方向亟待突破：分子机制研究：需深入解析年龄相关免疫衰退与VZV再激活的分子通路，特别是T细胞耗竭标志物与病毒潜伏维持的关联；交互作用量化：建立多因素风险预测模型，重点关注慢性炎症-免疫抑制-病毒激活的级联效应，以及气候环境因子的调控作用；疫苗策略优化：探索RZV在免疫缺陷人群中的长期保护效果，开发新型佐剂系统以提升老年人群免疫应答强度；真实世界证据：加强中低收入国家疫苗成本效益研究，制定分级接种方案，利用数字化工具提升高危人群覆盖；跨学科干预：整合心理干预、慢性病管理与抗病毒治疗，构建“预防-治疗-康复”全周期防控体系。随着精准医学与免疫学技术发展，未来有望通过生物标志物指导个体化预防，并通过新型疫苗与免疫调节剂阻断多重风险因素的协同作用，最终实现带状疱疹疾病负担的实质性下降。

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