

7 Shi Y, Hon M, Evans RM. The peroxisome proliferator-activated receptor delta, an integrator of transcriptional repression and nuclear receptor signaling. *Proc Natl Acad Sci USA*, 2002, 99: 2613 - 2618.

8 Liliane M, Beatrice D, Wahli W. Peroxisome-proliferator-activated receptor beta/delta: emerging roles for a previously neglected third family member. *Curr Opin Lipidol*, 2003, 14: 129 - 135.

9 Tan NS, Shaw NS, Vinckenbosch N, et al. Selective cooperation between fatty acid binding proteins and peroxisome proliferator-activated receptors in regulating transcription. *Mol Cell Biol*, 2002, 22: 5114 - 5127.

10 Tan NS, Michalik L, Noy N, et al. Critical roles of PPAR beta/delta in keratinocyte response to inflammation. *Genes Dev*, 2001, 15: 3263 - 3277.

11 Schmuth M, Haqq CM, Cairns WJ, et al. Peroxisome proliferator-activated receptor (PPAR)- β/δ stimulates differentiation and lipid accumulation in keratinocytes. *J Invest Dermatol*, 2004, 122: 971 - 983.

12 Tan NS, Michalik L, Di-Poi N, et al. Essential role of Smad3 in the inhibition of inflammation-induced PPARbeta/delta. *EMBO J*, 2004, 23: 4211 - 4221.

13 Michalik L, Desvergne B, Tan NS, et al. Impaired skin wound healing in peroxisome proliferator-activated receptor (PPAR) alpha and PPARbeta mutant mice. *J Cell Biol*, 2001, 154: 799 - 814.

14 Planavila A, Laguna JC, Vazquez-Carrera M. Nuclear factor-kB activation leads to down-regulation of fatty acid oxidation during cardiac hypertrophy. *J Biol Chem*, 2005, 280: 17464 - 17471.

15 Di-Poi N, Michalik L, Tan NS, et al. The anti-apoptotic role of PPARbeta contributes to efficient skin wound healing. *J Steroid Biochem Mol Biol*, 2003, 85: 257 - 265.

16 Di-Poi N, Michalik L, Desvergne B, et al. Functions of peroxisome proliferators-activated receptors (PPAR) in skin homeostasis. *Lipids*, 2004, 9: 1093 - 1099.

17 Di-Poi N, Tan NS, Michalik L, et al. Antiapoptotic role of PPARbeta in keratinocytes via transcriptional control of the Akt1 signaling pathway. *Mol Cell*, 2002, 10: 721 - 733.

18 Grose R, Hutter C, Bloch W, et al. A crucial role of beta 1 integrins for keratinocyte migration in vitro and during cutaneous wound repair. *Development*, 2002, 129: 2303 - 2315.

19 Tan NS, Michalik L, Desvergne B, et al. Peroxisome proliferator-activated receptor (PPAR)-beta as a target for wound healing drugs: what is possible? *Am J Clin Dermatol*, 2003, 4: 523 - 530.

20 Tan NS, Michalik L, Desvergne B, et al. Peroxisome proliferator-activated receptor-beta as a target for wound healing drugs. *Expert Opin Ther Targets*, 2004, 8: 39 - 48.

(收稿日期: 2005-01-31)

(本文编辑: 赵敏)

· 病例报告 ·

银屑病患者烫伤二例

张军 王凌峰 王宏 路玮

例 1 男, 53 岁, 患银屑病 10 年, 病灶集中于双下肢, 以角化不全病变为主。左足被热水烫伤 1.0%, 其中浅 II 度 0.5%、深 II 度 0.5% TBSA, 足部多处散在的银屑病皮损亦被烫伤, 伤后 1 h 来笔者单位就诊。立即以冷水冲洗创面 10 min, 低位引流水泡液, 保留疱皮, 碘伏纱布外敷后加压包扎。伤后 3 d, 水泡液部分被吸收, 银屑病皮损区烫伤部位水泡内均有血性渗液, 疱皮开窗探查见皮损烫伤区基底脆弱易出血、颜色较周围艳红、边界清楚, 继续以碘伏纱布包扎。伤后 6 d, 大部分疱液已被吸收, 银屑病皮损区痂皮下可见明显黑色淤斑, 而疱液吸收情况与周围正常皮肤烫伤区相同。大部分浅 II 度创面于伤后 8 ~ 10 d 愈合。原银屑病皮损区呈粉红色斑片状, 突出于周围皮肤, 皮损形态仍存在。深 II 度创面痂皮较早溶解, 清除痂皮后包扎治疗, 伤后 27 d 愈合形成瘢痕, 原银屑病皮损消失。患者出院 3 个月后随访, 见原深 II 度创面愈合区瘢痕充血现象消失, 瘢痕处无银屑病皮损, 浅 II 度愈合区可见银屑病皮损。

例 2 男, 37 岁, 患银屑病 6 年, 皮损集中于双上肢肘关节处。右上肢被铁水烫伤, 面积 0.5% TBSA, 深 II 度。银屑病皮损区因被烧伤, 表皮剥脱, 基底艳红有出血。早期清创后以碘伏纱布包扎, 伤后 3 d 创面结痂, 外涂磺胺嘧啶银治疗。伤后 16 d 痂皮溶解, 继续采用包扎治疗。伤后 24 d 创面愈合, 愈合区充血呈紫红色。患者出院 2 个月后随访, 原

银屑病皮损区瘢痕增生, 未再见银屑病皮损。

讨论 银屑病为全身性的皮肤病, 至今病因不清^[1]。银屑病皮损区烧伤较为少见, 本组两例患者创面有以下特点: (1) 银屑病皮损区创面基底脆弱易出血, 有血性渗液。(2) 浅 II 度烧伤创面愈合后银屑病皮损仍存在, 深 II 度烧伤创面愈合后形成瘢痕, 银屑病皮损消失。(3) 银屑病皮损区烧伤创面愈合过程与正常皮肤烧伤一致, 未出现延迟愈合。由于银屑病病变范围局限于表皮和真皮乳头层^[2], 浅 II 度烧伤后, 银屑病皮损区的病变细胞尚有部分存活, 这部分病变细胞参与了创面的修复, 且修复的能力与正常皮肤细胞相似。所以, 临床上银屑病皮损区与正常皮肤区的烧伤创面同时愈合, 原银屑病皮损仍然存在。深 II 度烧伤后, 银屑病皮损区的病变细胞全部坏死, 创面愈合后形成瘢痕, 所以原有银屑病皮损消失。银屑病仅表现为皮肤组织角化不全, 部分表皮细胞和真皮细胞并无改变, 创面具备愈合的条件。通过对以上两例的观察, 笔者认为银屑病皮损区烧伤创面的治疗可按正常皮肤烧伤创面处理。

参 考 文 献

1 魏双平, 四荣联, 张晓光, 等. 银屑病发病因素的 Logistic 回归分析. *中华皮肤科杂志*, 2004, 37: 665 - 666.

2 方光起, 郑增强, 程兵, 等. 应用银屑病微粒皮移植治愈大面积烧伤一例. *中华烧伤杂志*, 2002, 18: 165.

(收稿日期: 2005-06-22)

(本文编辑: 赵敏)

作者单位: 014010 包头, 内蒙古医学院第三附属医院烧伤科