

Decreased expression of heat shock proteins may lead to compromised wound healing in type 2 diabetes mellitus patients.

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Abstract

BACKGROUND:

Heat shock proteins (HSPs) are inducible stress proteins expressed in cells exposed to stress. HSPs promote wound healing by recruitment of dermal fibroblasts to the site of injury and bring about protein homeostasis. Diabetic wounds are hard to heal and inadequate HSPs may be important contributors in the etiology of diabetic foot ulcers (DFU).

OBJECTIVE:

To analyze the differential expression of HSPs and their downstream molecules in human diabetic wounds compared to control wounds.

METHODS:

Expressional levels of HSP27, HSP47 and HSP70 and their downstream molecules like TLR4, p38-MAPK were seen in biopsies from 101 human diabetic wounds compared to 8 control subjects without diabetes using RT-PCR, western blot and immunohistochemistry.

RESULTS:

Our study suggested a significant down regulation of HSP70, HSP47 and HSP27 (p value=<0.001 for HSP70; p value=0.007 for HSP47; p value=0.007 for HSP27) in DFU along with their downstream molecules TLR4 and p38-MAPK (p value=0.006 for p38-MAPK; p value=0.02 for TLR4). HSP70 levels were significantly lower in male subjects and their levels increased significantly with the grades of wound on Wagner's scale. Infection status of the wounds was found to be significantly associated with the increased levels of HSP70 and HSP27 in infected diabetic wounds.

CONCLUSIONS:

Our study demonstrates that the down regulation of HSPs in diabetic wounds is associated with wound healing impairment in T2DM subjects.

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KEYWORDS:

Diabetic foot ulcer; Heat shock proteins; MAPK; TLR4; Type 2 diabetes mellitus(T2DM); Wound healing impairment

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