Hyperbaric oxygen potentiates diabetic wound healing by promoting fibroblast cell proliferation and endothelial cell angiogenesis

Author links open overlay panel[XuHuang](a)[PengfeiLiang](b)[BimeiJiang](c)[PihongZhang](b)[WenchangYu](b)[MengtingDuan](b)[LeGuo](b)[XuCui](b)[MitaoHuang](b)[XiaoyuanHuang](b)
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Highlights

- HBOT facilitated wound healing in DFU.
- HBOT promotes angiogenic activities endothelial cells.
- HBOT activates HIF-1α signaling.
- HBOT promoted the expression of VEGF/SDF-1 in fibroblast and the expression of VEGFR/CXCR4 in endothelial cells.

Abstract

Background

Diabetic foot ulcer (DFU), one of the diabetic complications, brings high burden to diabetic patients. Hyperbaric oxygen therapy (HBOT) has been proven to be an effective clinical method for the treatment of DFU. However, the mechanisms still to be elucidated.

Methods

Diabetic foot mice model was established, and treated with hyperbaric oxygen. Haematoxylin & eosin (H&E) staining and Masson's trichrome staining were used for the analysis of wound healing. Human skin fibroblast (HSF) and human umbilical vein endothelial cell (HUVECS) were exposed to high glucose and hyperbaric oxygen for studying the mechanism of hyperbaric oxygen promoted wound healing *in vitro*. Wound healing assay, reactive oxygen species (ROS) assay, cell proliferation assay and tube formation assay were used for the analysis of wound healing. Quantitative-polymerase chain reaction (Q-PCR), Western blotting and enzyme-linked immunosorbent assay (ELISA) were used for the analysis of gene expression.

Results

HBOT facilitated wound healing in DFU mice model, and promoted the expression of HIF-1α, NF-κB, VEGFA, SDF-1, VEGFR2 and CXCR4. Hyperbaric oxygen promoted the proliferation, migration and ROS production, as well as the expression of SDF-1 and VEGFA in HSF. HBOT stimulated the proliferation, migration and tube formation, as well as the expression of CXCR4 and VEGFR2 in HUVECS.

Conclusion

Hyperbaric oxygen potentiates angiogenesis and diabetic wound healing by activating HIF-1α signaling, so as to promote the expression of VEGF/SDF-1 in HSF and the expression of VEGFR/CXCR4 in HUVECS, ultimately to promote the proliferation of HSF and the angiogenesis of HUVECS.