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LETTER TO THE EDITOR

Shock wave therapy for systemic sclerosis

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Sir,

The recently published paper of Tinazzi et al. [1] on the effects of shock wave therapy (ESWT) on the skin of patients with systemic sclerosis (SSc) prompted us to report our data.

We studied the effects of ESWT in 8 SSc female patients who received 4 consecutive ESWT sessions (Dermagold–MTS Germany defocused lithotripter device, 10.000 shots/session applied at the energy level of 0.06 mJ/mmq, frequency of 5 Hz), weekly applied to both hands and to one forearm, keeping the contralateral forearm as control.

Circulating vascular endothelial growth factor (VEGF) and von Willebrand factor (vWf) were measured before the treatment, immediately after the first session and at the end of the cycle. Skin involvement was assessed by Rodnan Skin Score (RSS) and by durometry. A visual analogic scale (VAS) was used to evaluate skin wellness.

Immediately after the first ESWT session, VEGF and vWf significantly decreased ($P = 0.007$ and $P = 0.004$, respectively), but remained stable thereafter. At the end of treatment, while total RSS did not change, RSS at fingers was significantly reduced ($P = 0.018$); durometer analysis showed a significant decrease at finger-pads and at treated forearm ($P < 0.0001$ and $P = 0.021$, respectively). VAS for skin wellness was found significantly improved ($P < 0.001$).

Although the present study was performed on a smaller group of SSc patients and the observation was concluded at the end of the cycle, our data confirm the observations of Tinazzi et al. [1] on the benefit of ESWT on SSc skin, as assessed by durometer analysis and VAS skin wellness.

Interestingly, VEGF and vWF, higher in SSc in agreement with previous observations [2, 3], significantly decreased immediately after the first ESWT session, but tended to return to baseline at the end of the entire cycle. These data may be in accord with Tinazzi et al. [1] who did not find any change in these as well as in other circulating markers of endothelial damage, probably because measured only later after the conclusion of the treatment.

The reason of the behavior of VEGF and vWF is not clear, but is consistent with several experimental studies that showed an early release of VEGF after ESWT [4, 5]. The intriguing point is the unexpected tendency for these markers to return to baseline at the end of treatment, in apparent association with the skin improvement.

Our data might be consistent with previous observations on hindlimb-induced ischemia in rabbits, where, at 28 days after ESWT, VEGF was unchanged, but capillary density was significantly increased [6].

Therefore, in agreement with the recent finding of increased capillary patency starting as early as 1 h after ESWT application in mouse striated skin muscle [7], the release of angiogenic growth factors seems to be a very early feature of ESWT. It might be conceivable that ESWT first stimulates the early and probably short-living expression of endothelium-derived factors and only subsequently

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induces the mechanism(s), still unknown, responsible for tissue regeneration and repair [8].

In conclusion, ESWT might represent a non-invasive, effective and safe strategy for ischemic conditions, including SSc. However, more studies are needed to further clarify the underlying mechanisms, since other systems than the complex interaction between endothelium, VEGF and angiogenesis may be involved.

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