months), continued the treatment for further 4 years (T4) (ILO group). Other 19 patients, although they continued the same cyclic intravenous iloprost treatment as the previous group, also received bosentan 125 mg twice a day for 4 years (ILO+BOS group), due to digital ulcers. DT was yearly evaluated by both US (18 MHz probe, MyLab 25, ESAOTE, Italy) and mRSS at the level of the usual seven skin areas (zygoma, fingers, dorsum of hands, forearms, upper-arms, chest, abdomen, thighs, legs, and feet). Non-parametric tests were used for the statistical analysis.

Results: A statistically significant decrease of DT, measured by US, was observed in the ILO+BOS group from T0 (median DT 1.135 mm) to T4 (median DT 1.088 mm) (p=0.01). No statistical significant variation of mRSS was observed during the follow-up in this group of patients (median mRSS at T0 12.5/1 and at T4 11.5/1, p=0.70). Conversely, in ILO group, a statistically significant increase of DT was observed after four years, as measured by US (median DT at T0 1.070 and at T4 1.258, p<0.0001), as assessed by mRSS (median mRSS at T0 4.5/1 and at T4 8/5, p=0.0001). Transient increase of transaminases was managed by temporary bosentan discontinuation.

Conclusions: In this open study, the long-term treatment with ET-1 receptor antagonist in combination with iloprost seems to be associated with a decrease of DT in SSc patients, in contrast to the treatment with iloprost alone. DT evaluated by US over long term seems to be more susceptible to change than by mRSS.

REFERENCES:

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