Assessment of triamcinolone treatment using a keloid-derived fibroblast-seeded collagen sponges

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Keloids are scars that arise from often quite minor trauma. They are characterized by high and unabating inflammation, which provokes their fibroblasts to proliferate and produce abundant extracellular matrix. This causes the scar to grow relentlessly both vertically and horizontally. The pathohistological characteristics of keloids include the production of thick irregular collagen fiber bundles in the reticular layer of the dermis, between which lie multiple fibroblast-like spindle cells. Given the intractability of keloids and the high risk of recurrence, effective therapies for these scars generally involve a multidisciplinary approach that combines surgical resection, electron beam radiation, and various conservative treatments. Some conservative treatments are sometimes initially used on their own. An example is local administration of triamcinolone acetonide. However, its effectiveness is limited and variable and the risk of keloid recurrence remains. In this study, a collagen sponge was seeded with an established human keloid-derived fibroblast cell line to generate a highly reliable and reproducible keloid fibroblast model. We then used immunohistochemistry to examine the effect of triamcinolone acetonide on fibroblast proliferation and their production of characteristic keloid molecules. We found that triamcinolone acetonide reduced overall fibroblast numbers and their production of type Ⅲ collagen, IL-6, and TGF-β. However, the numbers of fibroblasts that were positive for α-SMA and produced type I collagen did not change. Thus, triamcinolone acetonide only partially inhibits keloid fibroblast proliferation. This may explain why this monotherapy can have poor and variable effects.