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Case Report

Naevus Sebaceous with Tumour of the Follicular Infundibulum, Trichilemmoma, Desmoplastic Trichilemmoma, Apocrine Adenoma and Syringocystadenoma Papilliferum: Report of a Case

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Introduction

Due to the increasing prevalence of so called "life style diseases", such as diabetes, obesity or hypertension, the number of associated vascular and nerve lesions increases. In the lower limbs in particular, bagatelle trauma causes wounds that lead to wound healing disorders and chronic wounds [1]. All these patients are reliant on drugs for the treatment of cardiovascular or diabetic disease. While drugs such as cortisone or non-steroidal anti-inflammatory drugs (NSAIDs) are postulated to have a negative effect on wound healing, it is unknown whether the intake of antihypertensive drugs and antidiabetics, respectively influences wound healing as well. As this is difficult to analyze systematically in clinical investigations, a comparative *in-vitro* study with human skin cells in 2D and 3D models is performed.

Methods

A systematic literature review on the topic "Wound healing & antihypertensive drugs" and "Wound healing & antidiabetics" was carried out. Based on these findings and according to the latest drug report of a major German health insurance, the effect of the five antihypertensive drugs and four antidiabetics most frequently administered in Germany were analyzed in human fibroblasts and keratinocytes. For the antihypertensive drugs a representative sample of the groups β1-receptor blocker, calcium channel blocker, angiotensin-converting enzyme inhibitors, diuretics and the sartan group was selected. For the antidiabetic drugs representatives of the substance classes biguanides, sulfonylureas, glinides and dipeptidylpeptidase inhibitors (DDPIs) were chosen. In 2D cultures, cell metabolism, cell viability and migration ability were histologically evaluated. In addition, substances were investigated in a 3D wound model according to Timpson [2], regarding epidermal thickness (µm) and fibroblast density (n/µm) at the wound margin and wound bed. Here, cell proliferation, cell migration and apoptosis were immunohistochemically investigated after 12 days of application of antihypertensive drugs and antidiabetics in serum equivalent doses.

Results

There are only few scientific studies investigating the effect of antihypertensive drugs and antidiabetics on wound healing. In addition, their findings are partly contradictory. Among antihypertensive drugs, calcium channel blockers like amlodipine improve the tensile strength, but not the epithelialization of the skin [3, 4, 5]. β-blockers like propranolol or metoprolol have a systemic and locally accelerating effect on wound healing [6, 7].They activate the keratinocytes, which have a positive effect on wound closure but a negative effect on a possible existing psoriasis [8, 9].

ACE inhibitors like ramipril have a negative effect on cell proliferation and cell migration [10, 11, 12]. This was confirmed by reduced healing processes in the 3D wound model. Ramipril inhibits collagen biosynthesis as well [7]. Among the antidiabetics, metformin belonging to the biguanides impairs wound healing processes qualitatively and quantitatively *in-vivo* and *in-vitro* [13, 14]. The negative influence on cell metabolism is particularly noteworthy here. In contrast, the quite new dipeptidyl peptidase inhibitor sitagliptin has a positive effect on the wound and its blood circulation [15, 16]. Sulfonylureas like glibenclamide do not appear to have a positive or negative effect on wound healing [17, 18]. Own results in the in-vitro 3D wound model largely support this above-mentioned effects, with keratinocytes reacting much more sensitive to antihypertensive drugs and antidiabetics. In summary, the negative influence of hydrochlorothiazide and metformin is particularly worth mentioning here.

Discussion

Most patients with chronic wounds also suffer from hypertension and/or diabetes. It is therefore astonishing that "side effects" of drug therapy on wound healing are hardly considered in medical and scientific research. Almost all antidiabetics and antihypertensive drugs have a distinct measurable *in-vitro* effect on wound healing. There are huge variations among the substance classes: In contrast to systemic application, the application of metformin in serum-equivalent concentration was associated with negative effects on wound healing d in *in-vitro* analyzes [19]. Repaglinide also tended to have a negative effect on the metabolism of skin cells. In contrast, sitagliptin and glibenclamide had a slightly positive effect. Interestingly, antidiabetics led to an increased rate of apoptosis in the tissue of the wound bed (fibroblasts) when applicated directly. In the case of antihypertensive drugs, β-receptor blockers improve wound healing in *in-vivo* and *in-vitro* studies. The results after local application of timolol are particularly noteworthy [4]. Calcium channel blockers showed a positive effect on wound healing. However, the opposite is the case for ACE inhibitors and thiazide diuretics (e.g. HCT): The wound healing in *ex-vivo* and *in-vitro* 3D skin model is delayed by HCT in serum equivalent doses [20]. In addition, a possible association between thiazide diuretics and skin cancer should not be neglected. The results presented here should be considered due to their potential relevance for patients with chronic wounds who do not respond to adequate wound therapies. In particular, the negative results of metformin, the first choice therapy for type II diabetes, which does not induce hyperglycemia or weight gain, increases insulin sensitivity and reduces mortality and morbidity, are remarkable and incomprehensible, so that a clinical study is indispensable.

Legends to photomicrographs

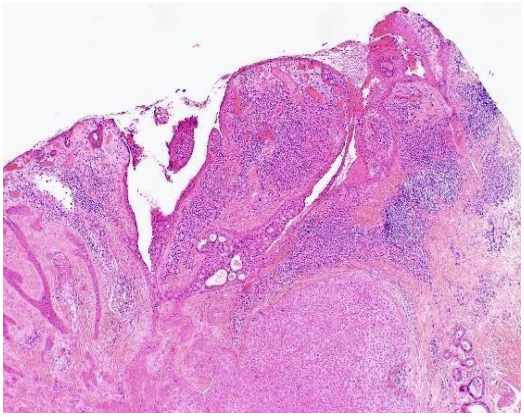

Figure1. Features of a naevus sebaceous.

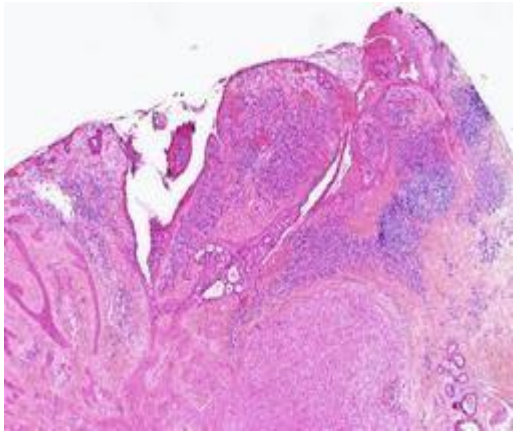

Figure2. Basaloid follicular hamartoma.

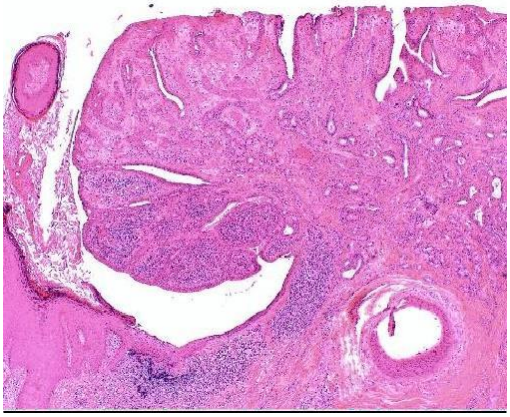

Figure3. Desmoplastic trichilemmoma**.**

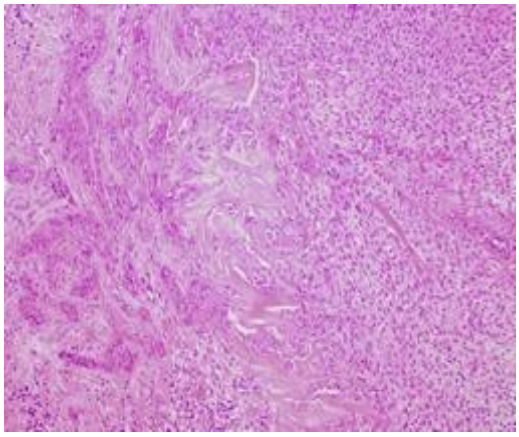

Figure 4. Trichilemmoma. and syringocystadenoma.

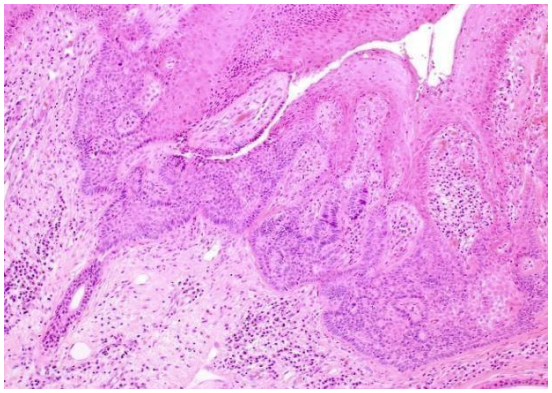

Figure 5. Syringocystadenoma papilliferum and apocrine adenoma.

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