INTRODUCTION

Scars are visible and palpable skin changes that remain after healing various types of damage to the integrity of the skin. Scarring is a pathophysiological process of skin regeneration, which is directed at closing of defect. Atypical wound healing may be accompanied by excessive scarring. In this case, randomly (chaotically) located fibers of dense connective tissue form hypertrophic or keloid scars. The regulation of process of scar formation depends on many iatrogenic and somatic factors. The problem of excessive scar formation deserves a particular attention, primarily in the open areas of the human body [1-4].

Long-term treatment of wounds, purulent inflammatory processes, the presence of various pyogenic microorganisms in them, as well as a weakening of the reactivity and resistance of the body, including tissue immunity, lead to atypical development of connective tissue with the formation of an altered musculoskeletal structure – scars. They cause not only cosmetic, but also physiological disturbances [5-8].

Therefore, the problem of differential diagnosis and comprehensive rehabilitation treatment of head and neck scars remains an urgent problem of modern medicine.

THE AIM

Study of the influence of local oxygen deficiency on the features of the formation of keloid scars of the head and neck.

MATERIALS AND METHODS

The research material was 17 incisional biopsy specimens of keloid scars, which were the highest age categories of patients from 19 to 63 years.

To achieve the established goals, the studied tissues were stained using the Mallory method, Hart + Van Gieson (in our modifications) and amydo-black (in our modifications) [9-11]. A digital camera was used for systematic documentation.

RESULTS AND DISCUSSION

Based on the literature, a visually keloid scar is characterized by a protruding part of the skin covered with the epidermis, under which there are hyaline masses [12, 13].

The histochemical studies were performed to determine the histochemical properties of fibrous structures of keloid scar. It was revealed that at coloring according to Mallory’s technique, sections of fibrinoid swelling of collagen fibers were noted near central homogeneous hyaline masses. The lateral areas of the dermis that adjacent to keloid scar tissue, at coloring by the Hart method, in our modification, were characterized by the separation of the hyalinosis zone from the undamaged dermis by coarse bundles of elastic fibers that are colored in dark purple. The undifferentiated dysplasia of the connective tissue has caused the replacement and obstruction of single vascular components, which has complicated the local oxygen deficiency of keloid-altered tissues.

Conclusions: Thus, plasmaragia and the accumulation of protein deposits in the perivascular space determine the inhibition of local hemodynamics, which explains the decrease in oxygen transport to tissues. Decreased oxygenation and increased permeability of the vascular wall causes local hemocirculatory hypoxia.

KEY WORDS: keloid scar, hyalinosis, histotopographic features
were characterized by the separation of the hyalinosis zone from the undamaged dermis by coarse bundles of elastic fibers that are colored in dark purple.

Hyperelastosis, that was observed in the lateral areas of the keloid scar, in our opinion, should be regarded as an adaptive-compensatory process for formation of hyalinosis.

It has been established that the basis of the keloid scar reaches the borders of dermis, which consists of coarse bundles of collagen fibers were painted in red color. These bundles were separated from each other by bundles of elastic fibers that were painted in dark purple.

Between the aforementioned fibrous structures it was noted the vascular bundle, which was represented by arteries and veins. In the arteries, the external and internal membranes were clearly defined, painted in purple.

The circular smooth muscle layer was located between the membranes, which was colored green. Unlike arteries, bundles of collagen and elastic fibers in the veins were intertwined.

It is obvious that precisely due to the presence of a vascular bundle in the basis of the keloid; a compensatory-adaptation process in the form of hyperelastolysis is carried out around it. The undifferentiated dysplasia of the connective tissue has caused the replacement and obstruction of single vascular components, which has complicated the local oxygen deficiency of keloid-altered tissues (Figure 2).

It should be noted that in some cases the neuromas were visualized on serial histological sections at the base of the keloid scar at stained according to the Hart method, in our modification. The latter were represented by concentric, spiral-like myelin fibers, which were colored black. Between them bundles of collagen fibers colored in red were noted. In our opinion, the partial reparative regeneration of myelin fibers occurs is happened during the growth

![Fig. 1. Plots of fibrinoid swelling of keloid scar. Colored by Mallory. Ob.: x 40; Ok.: x 10.](image1)

1-fragmented collagen fibers; 2-protein composites; 3-regressed vessels.

![Fig. 2. Areas of the dermis that border to keloid scars. Colored by Hart (in our modification). Ob: x 20; Ok.: x 10:](image2)

1-intact dermis; 2-zone of hyalinosis; 3-elastic fibers.

![Fig. 3. The formation of a neuroma in the main zone of keloid scars. Colored by Hart (in our modification). Ob.: x 100; Ok.: x 10:](image3)

1-neuroma; 2-collagen fibers.

![Fig. 4. Change of tintorial properties of collagen fibers. Colored by amido-black (in our modification). Ob.: 40; Ok.: x 10.](image4)

1-basal membrane; 2-tintorially modified collagen fibers; 3-core zone; 4-vascular components.
of the latter. Obviously, the presence of a neroma in the thickness of the keloid scar falling under compression of collagen and elastic fibers causes the subjective sensations of patients (local pain, paresthesia and itching) (Figure 3).

It was found that at coloring by amido-black, in our modification, in the areas of keloid scars adjacent to the epithelium, the papillary layer of the dermis was replaced by individual bundles of collagen fibers. The structure of the epidermis was preserved with the presence of horny scales.

Separate bundles of collagen fibers have stacked together and gradually have replaced the papillary layer, which included arcade microvessels. There were single cell infiltrations between bundles of collagen fibers. It was established that the keloid scar in case of this technique of coloring have consisted of thin fibrillar structures.

The bundles of fibrous structures during histological examination at coloring by amido-black were colored red due to changes in their tinctorial properties. In our opinion, a change in these properties when applying the above-mentioned color technique was due to the phenomenon of fibrinoid swelling.

Fibrinoid swelling, which was observed in keloid scars, has indicated its constant progression in the chronic course in the presence of various exogenous factors. Between the individual arteries and venules, there were light homogeneous structures of the protein deposit, among which there were single elongated fibriloblast nuclei (Figure 4).

Therefore, at the first stage of the formation of a keloid scar, a gradual replacement of the papillary dermis with bunches of collagen fibers, which were formed in case of presence of cellular infiltrates, was observed.

The second stage of keloid morphogenesis was characterized by fibrinoid swelling and necrosis of collagen fibers.

It should be noted that in conditions of progression of the keloid scar at its base, along with the phenomena of fibrinoid swelling of collagen fibers, plasma hemorrhage from the vessels was noted. The connective tissue hyalinosis was observed due to fibrinoid swelling and plasmoragia from blood vessels in the central zone of the keloid scar.

CONCLUSIONS

Thus, plasmoragia and the accumulation of protein deposits in the perivascular space determine the inhibition of local hemodynamics, which explains the decrease in oxygen transport to tissues. Decreased oxygenation and increased permeability of the vascular wall causes local hemocirculatory hypoxia. Therefore, in our opinion, to eliminate the local oil-deficient state, emoxipin should be used, which stabilizes the vascular wall and reduces tissue hypoxia by suppressing lipid peroxidation processes.

REFERENCES

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