MORPHOLOGY AND PATHOLOGICAL ANATOMY

THE DEGENERATIVE AND REGENERATIVE PROCESSES IN THE OLFACTORY ORGAN OF MAMMALS

Ya. A. Vinnikov

From the Department of General Biology of the Turkmen State Medical Institute (Ashkhabad)

(Received December 28, 1955. Submitted by Acting Member of the Academy of Medical Sciences of the USSR N. G. Khlopin)

The degenerative and regenerative processes were studied in the olfactory organ of 40 white rats in two series of experiments. In the first series (20 rats) the receptor layer of the olfactory lining of the right half of the nasal cavity was injured with a sharp knife. In the second series (20 rats) the olfactory bulb of the brain was injured or extirpated in the same way (also on the right side).

Injury of the olfactory lining caused the degeneration of the surrounding tissues, hemorrhage and a local inflammatory reaction. The olfactory receptor layer, which degenerated in the traumatized area as a result of the inflammatory edema, sloughed off for a large area around the necrotic region. Scaling from the underlying connective tissue is observed at the external basal membrane. In the scaled areas, which are connected with the unscaled olfactory receptor layer, the olfactory and supporting cells show a number of atrophic changes.

Soon (in 2-3 days) the elements which scaled off degenerate, uncovering the underlying connective tissue. They detach themselves from the retained areas of the receptor layer, which form a unique bulb-shaped protuberance, or roll, at this spot which soon builds up and moves onto the uncovered surface of the connective tissue.

In such border areas of the receptor area which are moving onto the connective tissue it is possible to find on the 3-5th day a number of changes of a reactive nature. Some supporting cells swell, round and multiply karyokinetically, others collect granules of secreta. The neighboring olfactory cells stain intensively, especially after osmium fixation. Their olfactory hairs, apparently in connection with "paralysis" of mitosis, elongate and seem to hang down into the lumen, which is bordered by the olfactory lining. Soon such "paralyzed" olfactory cells disintegrate, as a rule. Their remnants are eliminated by numerous phagocytes, which penetrate into the receptor layer from the underlying connective tissue (partly, most likely, at the expense of the phagocytic function of the supporting elements).

In Bowman's glands, which may be preserved both in the traumatized area and, especially, at some distance, proliferative processes are observed, along with degenerative phenomena. The first attempts to close the defect on the part of the bordering areas of the receptor layer, which were observed from the 4-8th day, increase. The above-mentioned roll of the preserved border area of the receptor layer moves and spreads over the connective tissue which had been uncovered as a consequence of the preliminary scaling. At this time the onmoving roll thins down to a single layer of flattened cells, which gradually creeps over the area of the direct trauma, freed of the bloody scab.

If the surface of the wound is large, both the proliferating excretory ducts and the terminal sections of the Bowman's glands can participate in covering it. Fairly often the defect is closed by proliferation of the respiratory epithelium, which is often present in the depths of the folds of the reticulate labyrinth of rodents [1, 2].

Thus on the 8-10th day the traumatic defect is always covered by an epithelioid coat which developed either from the supporting elements and Bowman's glands of the receptor layer or from the respiratory epithelium. In both cases Bowman's glands may be found in this coat. Part of them are the preserved glands, part are undoubtedly newly formed, especially in the areas covered by supporting elements.

Of course, the regenerative process occurred simultaneously in the underlying connective tissue. The proliferation of the covers can change the contour of the olfactory organ considerably.

Prolonged observation of the newly-formed covers (up to 3-6 months) in our experiments on white rats did not uncover even once the new formation of olfactory cells. The latter were preserved only in the uninjured areas of the olfactory organ. In this respect our results agree with some data [3, 4, 5] in the literature and disagree with others [6].

On the 2-6th day after the trauma, the central growths of the destroyed and degenerated olfactory cells also showed initial hyperimpregnation, then varicose thickening, fragmentation and degeneration.

The destruction of individual nerve fibres which surround the olfactory bulbs and the first signs of changes in the olfactory glomeruli, as a result of the indicated process of retrograde degeneration, were already observed on the 3-4th day after trauma.

Many glomeruli were wholly or partly involved in the degenerative process. It was evidenced by the edema around the glial capsule and the proliferation of its elements. Later (on the 6-8th day), the substance of the glomerulus could disintegrate into separate, intensely-staining lumps, and after some time (10-15 days) such glomeruli could disappear completely, which could be watched especially clearly in the olfactory bulbs of rats.

The disintegration of the olfactory glomeruli—of the first olfactory synapse—affected the mitral neurons connected with the glomeruli also. Already on the 4-5th day signs of "irritation" could be seen in them. On the 8-10th day pericellular edema occured around the neurons. Part of the neurons disintegrated and, apparently, were subjected to neuronophagy at the expense of the adjacent glia. By the 28-30th day after the operation, if the defect in the olfactory lining was great, the layer of mitral neurons in the corresponding olfactory bulb abruptly atrophied both due to the decrease in the size of the individual neurons and due to the decrease in the total number in the layer. In some cases the neurons could completely disappear in the area of the disintegrated glomeruli.

It should be observed that after injury to the receptor layer of the right half of the nasal cavity, on the 20-25th day signs of destruction and atrophy of individual olfactory glomeruli and neurons were usually observed in the left bulb also. At the same time destruction of groups or whole nerve fibers in the olfactory nerve and of olfactory cells in the receptor layer of the left half of the nasal cavity was observed also.

In spite of the exact localization of the trauma, the projected reflection of the atrophic process could not be located in either the olfactory nerve nor in the olfactory glomeruli and neurons of the olfactory bulbs [5]. Individual degenerating tufts and fibers were found in the most unexpected areas of the olfactory lining or in fila olfactoria which were quite far from the injured area. This also relates to the olfactory glomeruli and to the mitral neurons whose degeneration was followed not locally, but in many areas of the olfactory bulbs.

In experiments with section or extirpation of the olfactory bulbs it was also possible to follow the regular cycle of degenerative and regenerative processes. At the cut, destruction of the cerebral tissue and hemorrhages were observed. On the 10-14th day reparative processes were found in the olfactory bulbs at the location of the injury.

The first reaction of the olfactory nerve and receptor layer of the olfactory lining on one side could be observed on the 2-4th day. It was evidenced first as intensive infiltration of the receptor layer by leucocytes. The infiltration extended to the underlying connective tissue with its Bowman's glands and tufts and fibers of the olfactory nerve. The infiltration could also be followed in the fila olfactoria. Then selective staining of the olfactory cells, which usually are made evident with difficulty, was observed. The number of varicose thickenings in the olfactory fibers increased sharply. The olfactory cells showed signs of "paralysis" of miosis—their hairs elongated. Later such olfactory cells began to contract. Their nucleus lost its rounded shape and became pycnotic, the peripheral and central outgrowths disintegrated. Such olfactory cells died, were subjected to phagocytosis and soon disappeared; supporting cells which were located alongside, on the contrary, were preserved well. The destruction of the olfactory cells in the receptor layer could include considerable areas, which could be located easily in various parts of the olfactory lining. Such areas could be solitary also. If the trauma in the olfactory bulbs was extended or if the bulb was extirpated completely, areas of the receptor layer with dead olfactory cells were found more frequently and covered a greater area, even the entire half of the nasal cavity; however a regular distribution of destroyed areas in the receptor layer, which would reflect the defect in the

olfactory bulb, was not found. With injury of the olfactory bulbs, degeneration of elements in Jacobson's organ was also observed.

Destroyed areas were soon replaced by proliferating supporting cells. The new formation of olfactory cells could not be found here. Bowman's glands, their ducts opening into the areas where the olfactory cells disappeared, kept their structure and, apparently, continued their usual function.

On the 16-18th day a clear picture of degeneration could be observed in the fibers of the olfactory nerve which were connected with the degenerated olfactory cells.

In conclusion it should be pointed out that during injury of the right olfactory bulb, degenerative processes in part of the mitral neurons and individual olfactory glomeruli were observed in the left one also. In addition, in the left half of the receptor layer, individual olfactory cells or small groups of them were destroyed and disappeared.

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