STELLATE GANGLION PATHOMORPHOLOGY IN PATIENTS WITH THERMAL BURNS

I. M. Isaev

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The stellate ganglion is formed by fusion of the inferior cervical and first thoracic ganglia of the lateral sympathetic trunk. In man this fusion is the usual event, and only in a small percentage of cases do the two ganglia remain separate. Fusion of these ganglia in man is observed in 75% of cases [14]. A difference is often observed in the volume and connections of the stellate ganglion on the two sides. Sometimes the second and even the third thoracic ganglia also take part in the formation of the stellate ganglion [3, 7]. The inferior cervical ganglion as a rule is laid down in the embryo in the form of the cranial part of the thoracic sympathetic trunk [2]. These ganglia, as peripheral centers of nervous regulation, play a very important role in the pathogenesis of different types of cardiovascular diseases [9]. Accordingly, the study of the pathomorphology of the stellate ganglia in burns is particularly interesting, for it has been shown that burn trauma is often complicated by cardiovascular and pulmonary failure [5, 6, 8]. Similar investigations have been undertaken in diseases of the cardiovascular system [1, 4, 12]. However, this problem is still far from having been solved, and the available information on the possibility of studying nervous structures in cadaveric material are contradictory [11, 13]. Meanwhile analysis of structural changes in the stellate ganglia in thermal burns, using a combination of modern neurohistochemical and classical neurohistologic methods has not hitherto been undertaken. This was accordingly the aim of the investigation to be described below.

EXPERIMENTAL METHOD

Autopsy material from 80 patients dying from burns of different duration (with shock, toxemia, septicotoxemia, and burn emaciation) was studied. The victims were aged from 20 to 80 years. Autopsy was performed at various times from 2 to 6 h after death. The stellate ganglia were chosen as test objects. Afer fixation in a 10% solution of neutral formalin the material was embedded in paraffin wax. Sections were stained with heamtoxylin and eosin, by Van Gieson's and Nissl's methods, and impregnated by the Bielschowsky–Gros methods. Neurohistochemical methods of staining adrenergic structures also were used, by incubating the sections in a 2% solution of glyoxylic acid in the modification in [10]. Cholinergic nerve structures were demonstrated by the method of Karnovsky and Roots. The diameter of the cells was measured with an ocular micrometer and the results were subjected to statistical analysis.

EXPERIMENTAL RESULTS

Marked congestion of capillaries and ectasia of lymphatics were observed in the stroma of the stellate ganglion of patients dying in the period of burn shock. Signs of acute ganglionitis were present in the parenchyma of the ganglion. In preparations stained by Nissl's method various stages of dystrophy and atrophy of neurons were observed, with signs of swelling and shrinking. In the later stages of burns various degrees of chromatolysis were often observed, or even complete lysis of the chromatophilic substance. The cytoplasm of the neurons under these circumstances was stained pale violet with thionine.

Department of Pathological Anatomy, N. Narimanov Azerbaijan Medical Institute, Baku. (Presented by Academician of the Academy of Medical Sciences of the USSR D. S. Sarkisov.).
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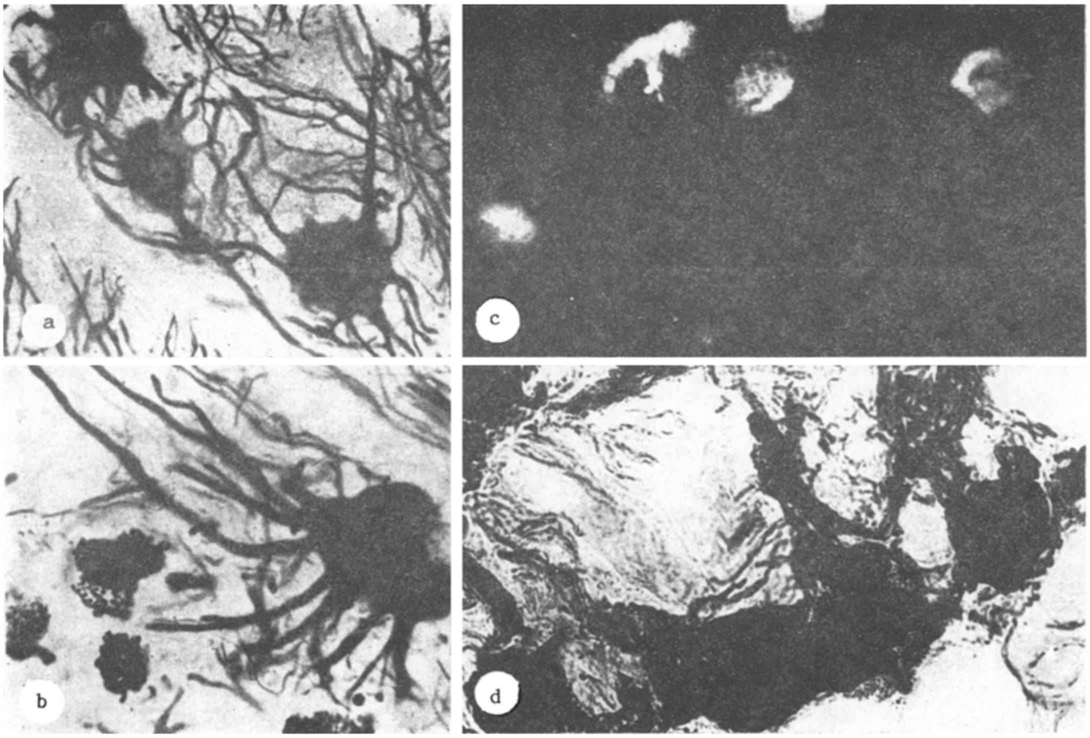

Fig. 1. Changes in stellate ganglia at various periods of burns. a) Hypertrophied neurons with thickening of outgrowths, enlargement of nuclei clearly visible — burn toxemia (impregnation after Bielschowsky—Gros. 300 ×); b) atrophy and necrosis of most neurons, cloudy-swelling degeneration of body of hypertrophied neuron on 40th day after burning — period of septicotoxemia (impregnation after Bielschowsky—Gros, 600 ×); c) bright fluorescence of catecholamines in cytoplasm of individual neurons of stellate ganglion, sharp decrease in content or absence of catecholamines in outgrowths and nerve fiber—burn shock (sections incubated in 2% solution of glyoxylic acid 200 ×); d) high concentration of AChE and lipofuscin granules in cytoplasm of individual neurons and alongs the course of nerve fibers — burn toxemia (staining after Karnovsky and Roots, 200 ×).

In preparations impregnated with silver various dystrophic and also regenerative changes were observed in the bodies and processes of the nerve cells. Only individual neurons were round in shape, with a clearly outlined nucleus and nucleolus. In many cells the nucleus was not clearly outlined and these cells were converted into cell ghosts; at the site of death of a cell, coarse granules of lipofuscin were visible.

In the period of burn toxemia marked disturbances of the circulation were present in the vessels of the stellate ganglion. Neurons of the ganglion were unevenly impregnated with silver: in some neurons, which were greatly enlarged, impregnation was intense, the diameter of the nerve cells was as much as 80-90 μ, and long axons emerged from the bodies of these neurons, in which the nuclei were also enlarged (Fig. 1). Hypertrophied neurons were interwoven by a dense network of preganglionic fibers, which formed synapses on the bodies of the nerve cells. Besides hypertrophy, atrophic changes were observed also in some neurons. They were characterized by a decrease in diameter of the nerve cells to 40-50 μ and by thinning and disappearance of the dendrites. The nuclei of these cells also were reduced in size and became hyperchromic.

The data obtained by the study of the stellate ganglia in the later stages of burns (the septicotoxemic stage) were varied. Cases in which compensatory processes in the nerve tissue of the ganglion were sufficiently well defined, although there were also considerable degenerative changes, were included in one group. This group included patients who died 15-20 days after burning. In these cases the stellate ganglion as a rule was divided by layers of connective tissue into lobules, in which two types of neurons were concentrated. Neurons of the first type were greatly enlarged, intensely impregnated with silver, and their nuclei were clearly outlined; cells of the second type resembled atrophic neurons, found in the ganglia of patients dying in the period of burn toxemia. These were pale, swollen cells, round in shape and with granular cytoplasm, and their nuclei intensely impregnated with silver, and often located eccentrically. Binuclear cells also were observed, and in some cells the second nucleus, which was much smaller, was displaced toward the periphery of the cell. The bodies of many neurons were elongated and deformed, and the nuclei of these cells were pycnotic or their outlines could not be distinguished at all; many cells were undergoing necrosis (Fig. 1b). Against the background of these compensatory and degenerative changes in the neurons, bundles of preganglionic myelinated fibers were disintegrating. Varicose thickenings were seen in those myelinated fibers which remained. In patients dying in the later stages (25-40 days after trauma) dystrophic changes predominated in the structure of the ganglion during the septicotoxemic period.

Analysis of the results of the neurosurgical investigation indicates that different adrenergic structures, which undergo considerable changes in burns, are represented in the stellate ganglia. These structures also undergo changes depending on the patient's age, the stage of development of the disease, and treatment given. Toward old age the catecholamine concentration in many adrenergic structures of the stellate ganglia falls appreciably. Meanwhile, in cases of burn toxemia and septicotoxemia catecholamine fluorescence in nerve structures of the stellate ganglia was observed quite clearly, although in many areas of the ganglion, weakly luminescent neurons, having lost their catecholamines, could be distinguished against this background. A variegated picture of a combination of brightly luminescent nerve cells of adrenergic nature and dying cells was characteristic. Incidentally, in ganglia of the human sympathetic nervous system luminescence of neurons is usually relatively uniform and does not attain a high level [9]. In patients with burns we observed for the first time how brightly luminescent are the sympathetic neurons, and in their degree of luminescence in some places they recalled SIF cells [1, 14]. In shock there is evidently a sudden flow of impulses from presynaptic nerve endings to neurons of autonomic ganglia. As a result, synthesis of noradrenalin and other mediators is strongly activated and reactive changes take place in the neuron. This state of the adrenergic neurons develops at different times, as is shown by the different degrees of luminescence of the neurons composing the ganglia (Fig. 1c). With the onset of the period of toxemia the number of brightly fluorescent adrenergic neurons falls. Incidentally, the number of luminescent nerve fibers in the composition of the ganglia chosen for study was relatively small. The distribution of luminescent nerve cells at the periphery of the ganglion was largely related to the particular architectonics of many sympathetic ganglia, in which many of the preganglionic fibers enter the ganglion in the form of bundles and are partly distributed among the cells, to which they give off presynaptic terminals [1]. As a rule, asymmetrically distributed lipofuscin concentrations, with yellow luminescence, could be found in the neurons studied. For the first time we demonstrated enlargement of the lipofuscin inclusions in adrenergic neurons during the transition from burns to toxemia, and an even greater degree of their enlargement with the onset of the period of septicotoxemia. In the study of the dynamics of an increase in the content of pigments in sympathetic neurons during the development of burn state, age differences in involution of neurons of the sympathetic ganglia must certainly be taken into consideration.

We observed for the first time an increase in acetylcholinesterase (AChE) activity at the beginning of the burn syndrome not only in the period of shock, but also with the onset of subsequent periods of toxemia and septicotoxemia. The study of the pathomorphology of the stellate ganglia showed that in burns the structure of the sympathetic nerve ganglia is very easily made out. As a result of pathological changes in the sympathetic nerve ganglia which we studied, the structure of individual neurons can be made out particularly clearly. Large cholinergic nerve cells are characterized by high AChE activity (Fig. 1d). The difference in size of the nerve cells and different levels of AChE activity in them will be noted. In burns AChE activity varies in the neurons, and it corresponds basically to the mosaic pattern of the lesion in the ganglia described above, with respect to their catecholamine concentrations. Many intraganglionic nerve fibers possess high AChE activity simultaneously. The vascular system of the ganglion also is clearly visible. On the whole, the contrasting state of the AChE activity in the ganglia studied is a noteworthy feature, evidence of the heterogeneity of the processes taking place in them, for besides reactive changes in some neurons, degenerating nerve cells and fibers of varied caliber also are found.

To conclude, changes in the structure of the stellate ganglia reveal a definite dynamics during different periods of the course of burns. In the period of burn shock, against the background of circulatory stasis, many neurons in the stroma of the ganglion die. In the period of toxemia, compensatory and adaptive changes predominated in the structure of the ganglion, in the form of hypertrophy of the neurons. Meanwhile, in the tissues of the stellate ganglion, many atrophic neurons, sharply reduced in size, were found. In the period of septicotoxemia and in the syndrome of burn exhaustion, degenerative changes were observed predominantly in the structure of the stellate ganglion.

Data obtained with the aid of modern neurohistochemical techniques are in agreement with the results obtained by classical neurohistologic methods of investigation.

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