MORPHOLOGY AND PATHOMORPHOLOGY

Early Bronchoscopic and Morphological Diagnostics and Prognosis in Thermal Inhalation Trauma

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Thermal injury is diagnosed by fibrobronchoscopy within the first 5 days after inhalation burn trauma in 73 patients (34% of the total number of patients hospitalized during the last 2 years). Pathological changes of the mucosa with predominant multiple ulceration at different levels of the tracheobronchial tree are detected in 44% patients and in more than 70% patients with acute respiratory deficiency. These changes determine variation and severity of thermal inhalation injury in the patients, and, together with morphofunctional characteristics, provide objective basis for the early diagnostics and prognosis.

Key Words: *thermal inhalation trauma; diagnostics; prognosis*

Thermal inhalation trauma determines a high level of lethality (50-80%) among flame-affected patients [1,4,10,13]. This trauma is characterized by latent development and high variability. The early diagnostic criteria of evaluation of this trauma are necessary to avoid inadequate therapy under the conditions of rapidly developing respiratory deficiency. Traditionally, the early prognosis of the burn trauma is based on the patients' age and the total area of burns [7,16]. These risk factors, as well as duration of the prehospital period, are considered as critical factors [11]. When inhalation burn trauma is taken into account, the accuracy of prognosis increases [18].

The characteristics of such combined trauma were revealed in the study of respiratory system several hours later after a burn accident. An objective method to prove the presence of inhalation trauma are the early fibrobronchoscopy (FBS), which sometimes restores the passability in the upper respiratory tract (URT). Damage to the entire bronchopulmonary system is not diagnosed at the early stage, although in many cases (for example, in explosion trauma), the injury at the alveolar level is fatal [8,10,12].

In order to solve of this problem, the mechanisms of smoke-induced instant development of URT edema and pulmonary pathology with early manifestations of irreversible respiratory distress syndrome have been studied [9,13,14,17]. An increase in the respiratory deficiency is caused by destruction of the alveolar capillary membranes and rapid development of toxic edema under conditions of microcapillary permeability. A burn trauma is characterized by disturbances in microcirculation and neuroendocrine regulation [3]. Inhalation of toxic components leads to stimulation of chemotaxis and accumulation of polymorphonuclear leukocytes, induction of damaging mechanism, and increase in the transvascular liquid flow [17]. Rapid development of perivascular and peribronchial, and then intra-alveolar edema within the first 2 days after smoke inhalation are reflected by the early changes in roentgenograms of the thoracic organs which correlate with the degree of respiratory dysfunction [15]. An objective estima-

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tion of the severity of thermal trauma should be based on the data related to the early pathomorphological changes in the bronchopulmonary system revealed during life and postmortem. However, there are no comprehensive characteristics of survived patients and those died soon after inhalation burn trauma. Our objective was to study the early morphological and bronchoscopic alterations in the bronchopulmonary system after inhalation burn trauma and to determine the most objective diagnostic and prognostic criteria.

MATERIALS AND METHODS

Morphological study included analysis of the severity of tissue damage in bronchi and lungs based on the autopsy material of 19 burnt patients with inhalation burn trauma, which died during the first 5 days. We also studied mucous FBS biopsies from trachea and primary and superlobar bronchi on the 4th-5th day after trauma in 15 patients by curative-diagnostic FBS [6]. In early FBS, inhalation burn injuries were diagnosed within the first 5 days in 73 patients (34% of the patients hospitalized during the last 2 years in the Thermal Traumatic Center, Russian Academy of Medical Sciences). The flame burns were inflicted in close compartments due to ignition of inflammable substances or a burst, which could be accompanied by a deep penetration of fumes into the respiratory tract. The patients' age varied from 16 to 80 years, the burnt area from 20 to 90% of the body surface (70±20%). FBS was used for visual determination of changes in URT, i.e., oronasal pharynx and tracheobronchial tree (TBT) to the extent depending on its passability. In each case the severity of damage was estimated by complex examination. The method for serial FBS was described previously [2].

RESULTS

The early FBS-diagnostics of inhalation burn trauma showed that lesions spread over the entire respiratory tract (Fig. 1). Focal ulcerations and necrosis of the URT were found in 64% patients and those of TBT in 44% patients. At different levels of TBT, FBS detected dispersed fine carbon particles, which are known to carry adsorbed fume toxic components, testifying to chemical mechanism of TBT lesion. Several hours after trauma, all patients developed respiratory deficiency. In 35% patients, this deficiency was acute, with prevalence of multiple ulceration of the TBT mucosa. Morphological analysis of vital biopsy of TBT showed pronounced destructive changes in the mucosa [6]. The disturbances of mucus formation, cell metabolism, and reparation ability of

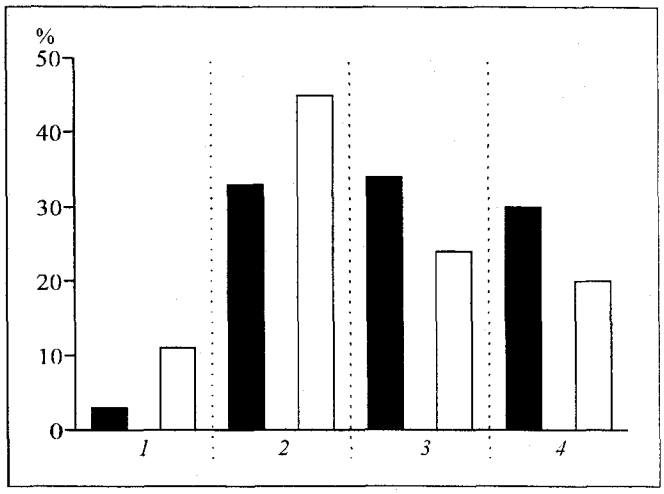

Fig. 1. Distribution of patients according to duration and intensity of inhalation burn trauma of the air pathways according to the early bronchoscopy data. Mean occurrences of different mucosal alterations: *1*) catarrhal; *2*) erosive-ulcerated; *3*) ulceronecrotic; and *4*) necrotic. Black bars: oronasal pharynx; white bars: tracheobronchial tree.

the preserved epithelium were confirmed by radioautographic data on suppression of proliferate activity and reduced vitality of epitheliocytes. The same phenomena underlie ulceration [5]. Detection of ulceration in tracheobronchial mucosa and modified morphofunctional characteristics of its cells provide the most objective presentation of pathogenic features of the inhalation burn trauma.

After serial FBS had been carried out, the lethality within the first 5 days after inhalation burn trauma decreased to 27%. The deceased patients had vast skin burns (more than 50% of the body surface area), while the survived patients had burns of smaller area. To determine the contribution of FBS to the early prognosis, it was necessary to assess the severity of damage both in survived patients and patients who died soon after trauma.

Purulent focal necrotic tracheobronchitis was diagnosed in patients who survived 1 day after trauma. Hyperemia, necroses, and pronounced edema were detected in the affected mucosa of trachea and large bronchi. Although edema had developed, the lumen of lobar bronchi was preserved just past the carina. At the same time, there was obturation of most distal lobar and segmentary bronchi by dense viscous secret containing soot and formation of purulent mucous plugs, which were gradually shifted in the distal direction by the repeated FBS. Aspiration to the level of subsegmentary bronchi and reduction of edema led to positive dynamics of external respiration indices. In most cases fibrinopurulent tracheobronchitis was diagnosed in FBS biopsy on the 4th day after trauma.

By contrast, in patients who died within the first and following few days drastic changes were detected

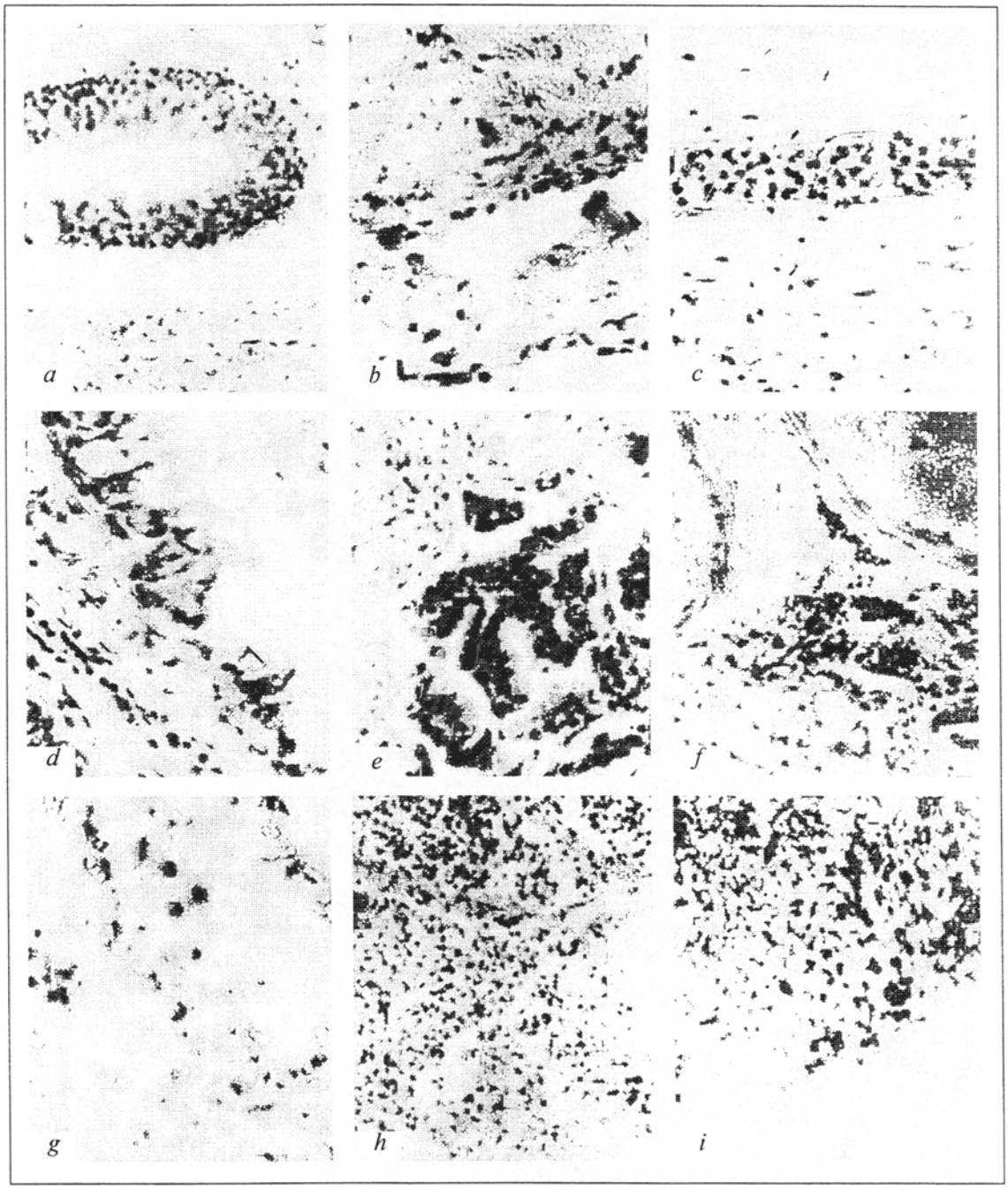

Fig. 2. Histological study of the bronchi and lungs of the patients with inhalation burn trauma who died within the first day. *a*) desquamative epithelium, edema, and moderate leukocyte infiltration of the connective tissue basis of the main bronchus; ×160; *b*) deep ulceration of mucosa in lobar bronchus; preserved epithelium is a flattened cell monolayer with destroyed cilia, ×250; *c*) plethora with stasis of erythrocytes and leukocytes in the capillaries of the main bronchus wall, edema and lymphocyte infiltration in *lamina propria* of the mucosa, ×250; *d*) surface ulceration of mucosa in the segmental bronchus with boundary hyperplasia of goblet cells and complete obturation of the lumen, ×320; *e*) spasm of small bronchus with complete obturation of the lumen, ×320; *f*) plethora and stasis of erythrocytes in the pulmonary vessels; perivascular accumulation of the soot particles in the pulmonary parenchyma, ×160; *g*) intra-alveolar edema with hemosiderophages, ×250; *h*) focal pneumonia with leukocyte exudate in the alveoli, ×160; *i*) large accumulation of soot particles in intrapulmonary lymphatic node, ×160. Hematoxylin and eosin staining.

in the entire URT 4-6 h after the inhalation burn trauma. Pronounced edema markedly decreased the lumens of lobar and segmentary bronchi. Disintegration and exfoliation of mucosa were observed in the edematous walls of trachea and large bronchi, which was characterized by vast erosions with contact bleeding and ulceration with rough necrotic edges and fibrin-covered bottom. Fibropurulent masses, secretion, and

soot were found in the bronchi both before and after FBS-sanitation. Irrespective of several attempts of URT sanitation, bronchial obturation increased. In these patients, the impairment of drainage function of TBT aggravated the diffuse ulceropurulent tracheobronchitis.

Autopsy detected severe morphological damage to the entire bronchopulmonary system (Fig. 2). There were pronounced changes in main and lobar bronchi. Fibrinous deposits with fragments of desquamative epithelium, mucus, and soot drastically decreased the bronchial lumen. The preserved fragments of mucosa were characterized by dystrophic changes and flattened mono- or bilayer epithelium with lost cilia. In the walls of large bronchi, deep ulcerations of the mucosa were detected, which stretched to the muscular layer. The epithelial lining of segmental bronchi was less affected, although it contained large areas of hyperplasia, hypersecreting goblet cells, and ulcers. Edema, stagnate plethora, focal lymphocyte infiltrations, and spasm of the terminal bronchi were observed in bronchial walls (Fig. 2). Functional changes in most elements of bronchial mucosa deteriorated mucociliary clearance and pulmonary ventilation and led to formation of atelectases. Stagnant plethora and blood stasis in pulmonary microcapillary vascular bed testified to disturbed hemodynamics in the lungs. Deterioration of gas exchange in the lungs was aggravated by development of interstitial and intra-alveolar edema with accumulation of hemosiderophages in edema. On the one hand, hemodynamic disturbances and diffuse edema indicate that inhalation trauma affects not only the bronchial tree, but also pulmonary tissue, and, on the other hand, together with progressive leukocyte exudation, they are the signs of bronchopneumonia developing within the first hours after trauma. The depositions of soot around the vessels and along the interalveolar septa promoted the primary alterations of pulmonary parenchyma. Soot was also accumulated in the intrapulmonary lymph nodes (Fig. 2).

Thus, within the frames of differential morphological diagnostics and FBS, the main indicator of severity of inhalation burn trauma is the duration and intensity of changes in TBT, while among the pathogenic criteria of damage, the main one is ulceration against the background of drastic hemodynamic disturbances. In contrast to favorable prognosis related to small-sized focal character of TBT affection, the total damage of respiratory system with pronounced edema, particularly at orifices of large bronchi, and irreversible obturation, detected by FBS, are accompanied by the early development of bronchopneumonia and indicate an unfavorable prognosis. Complex estimation of severity of inhalation burn damage as a risk factor of the burn disease enhances the objectivity of the prognosis at the early stage of its development.

REFERENCES

1. P. F. Ganzha and V. V. Yudenich, *Sov. Med.*, No. 2, 62-64 (1983).
2. V. Yu. Moroz, A. A. Alekseev, Sh. I. Kurbanov, and G. B. Yakovlev, *Vrach*, No. 6, 38-41 (1993).
3. D. S. Sarkisov, V. K. Sologub, R. I. Kaem, and I. I. Kolker, *Klin. Med.*, No. 2, 14-21 (1986).
4. V. K. Sologub, L. I. Gerasimova, L. P. Loginov, *et al.*, *Khirurgiya*, No. 11, 77-80 (1980).
5. D. S. Sarkisov (Ed.), *A Manual on Structural Basis of Adaptation and Compensation of the Disturbed Functions* [in Russian], Moscow (1987).
6. T. S. Ustinova, N. V. Panova, E. V. Glushchenko, *et al.*, *Byull. Eksp. Biol. Med.*, **115**, No. 6, 662-665 (1993).
7. C. C. Berry, T. L. Wachtel, and H. A. Frank, *Burns*, **8**, 38-45 (1982).
8. H. G. Bingham, T. J. Gallanger, and M. D. Powell, *J. Trauma*, **27**, 1286-1288 (1987).
9. F. S. Bongard, M. Matthay, R. C. Mackersie, and F. R. Levis, *Surgery*, **96**, 395-401 (1984).
10. W. R. Clarc and G. F. Nieman, *Burns*, **14**, 473-494 (1988).
11. N. D'Arpa, B. Napoli, and M. Masellis, *Ann. Medit. Burns Club*, **6**, No. 1, 15-19 (1993).
12. R. H. Demling and C. La Londe, *Burn Trauma*, Stuttgart (1989).
13. D. N. Herndon, R. E. Barrow, H. A. Linares, *et al.*, *Burns*, **14**, 349-356 (1988).
14. D. N. Herndon, R. E. Barrow, D. L. Traber, *et al.*, *Surgery*, **102**, No. 2, 341-348 (1987).
15. A. B. Peitsman, G. T. Shires, H. S. Teixidor, *et al.*, *J. Trauma*, **29**, 1232-1239 (1989).
16. V. M. Peterson, T. R. Murphy, T. Haddix, *et al.*, *Ibid.*, **28**, 632-637 (1988).
17. D. L. Traber, H. A. Linares, D. N. Herndon, and T. Prien, *Burns*, **14**, 357-364 (1988).