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Changes in the upper respiratory tract after burn injuries

U.R Usmonov

M.T Qayimov

BSTRACI

Dysfunctional changes in the upper respiratory tract in most cases develop as a postburn pathology. (1,2,12). After a burn, in 30-40% of patients, the general condition worsens and vital organs fail. (,3,6,8,11,13,16). The most important factors that damage the mucous membrane of the respiratory tract during IT are various combustion products in a confined space. The development of mucosal edema is primarily dependent on mediators such as histamine, bradykinin, prostacyclin and bronchospasm, and pulmonary vascular resistance is associated with the release of thromboxane and complement components (8.15).

Keywords:

Multiple organ disorders, burn disease, inhalation injury.

Acute respiratory failure in burned patients, regardless of the cause that caused it, often leads to multiple organ failure and mortality. Respiratory failure is one of the most common forms of organ failure in the PON syndrome and, progressing, leads to death in combination with dysfunction of other organs, regardless of the presence of inhalation injury (12,16). The disorder of the respiratory function of the lungs, in turn, disrupts the nutrition of the alveolar tissue, reduces the production of surfactant, contributing to the development of atelectasis (4.14), and increases venous blood shunting (2.5). In burn disease, along with respiratory disorders. non-respiratory functions of the lungs suffer: blood purification mechanical impurities and destruction. enzymatic participation in hemostasis and fibrinolysis. water and electrolyte metabolism, metabolism proteins, fats, biologically active substances (1, 7,9,10).

Purpose of the study: To study the pathology of the respiratory organs in burn disease

Materials and methods of research: For the period 2017-2021, there were 2136 patients in

the combust ology department of the Bukhara branch of the RRCEM. Of these, 156 patients had impaired function of the respiratory system. Of these, 96 women (61.5), men 60 (38.5). The age of the patients ranged from 2 to 70 years.

Frank index (IF) more than 60-90 units in 76 (48.7) patients, IF more than 90 units in 80 (51.3%) patients. In 55% burns with flames, 43% with boiling water, in 2% - electrical injury.) To study the dysfunction of the respiratory system in terms of systemic inflammatory (SIR), we analyzed the clinical indicator of tachypnea and features of the X-ray picture of the lungs (Table No. 1).

Chest radiography is an important diagnostic method for monitoring pulmonary complications and evaluating treatment from day 1 of a burn injury (493).

Results and discussions: First of all, it was noted that among 156 patients with identified disorders of the respiratory system, the vast majority (128 patients) with tachypnea 21-24 in 1 minute had signs of venous plethora of the lungs, expressed to varying degrees and indicating an increase in pressure in the pulmonary artery system. According to the

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literature data, the pressure in the pulmonary artery is increased during all periods of burn disease, depending on the severity of the burn disease, and the greatest increase in pressure was found in the stages of shock and toxemia with a gradual decrease by the time of healing of burn wounds (11.16).

Hyperserotonemia plays a role in the genesis of increased pulmonary resistance, hypertension the pulmonary circulation, impaired and exchange. perfusion gas causing vasoconstriction and bronchial constriction (2,4). The development of venous plethora, and with-it tachypnea in burned patients, we associate with manifestations of systemic microcirculation disorders and capillaryalveolar diffusion of blood gases against the background of developed SIRS (17.18.19.20).

Table №.1

	Number of patients			groups	
Pathological changes	1-18	вг] II-40	III - 56	IV-42	Total 156
Venous plethora	7	30	52	39	128
Interstitial	-	3	10	10	23
Alveolar edema	-	1	3	6	10
ARDSV	-	-	1	2	3
bacterial	-	1	8	11	20
Infarct pneumonia	1	5	6	3	15
Atelectasis	1	3	8	6	18
Diaphragm dome position	-	5	7	8	20
Pneumothorax	-	-	1	-	1

Distribution of patients with different severity of burn injury in accordance with x-rav changes in the lungs in most patients, signs of venous plethora appeared on the 1-2 day and persisted for 7-14 days, and in some patients it was much longer during the SVR, which correlated with the severity of the burn injury. With an uncomplicated course of a burn disease, venous plethora, according radiography, gradually decreased and

disappeared, with a complicated one, it appeared again or increased, while some patients developed signs of increased vascular permeability in the microvessels of the lungs, which manifested itself as interstitial or even alveolar edema. Among 128 patients, more than half (5, 8, 11) had tachypnea combined only with circulatory disorders in the lungs.

Signs of increased vascular permeability against the background of venous plethora in the form of interstitial and alveolar pulmonary edema were detected in 23 and 10 patients, respectively. An additional negative role in the formation of edema can be played by excessive infusion therapy.

Conclusions: We assume that interstitial edema, caused by a systemic increase in vascular permeability in SVR, occurred in a larger number of patients with tachypnea, but its severity was minimal and insufficient to detect this sign on radiographs. A more pronounced swelling of the interstitium, and even more so the sweating of edematous fluid into the alveoli, caused more severe dysfunction and even respiratory failure.

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Volume 18 | March, 2023

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