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Diagnostics of Toxic Lung Injuries

Islamov Shavkat Erjigitovich
DSc, Associate Professor, Samarkand state medical university, Samarkand, Uzbekistan

Makhmatmuradova Nargiza Negmatullaevna
PhD, Associate Professor, Samarkand state medical university, Samarkand, Uzbekistan

Khazratov Bekmurod Sherboevich
Samarkand state medical university, Samarkand, Uzbekistan

Annotation: The article is devoted to the diagnostics of morphogenesis of toxic lung damage and the possibilities of its diagnostics are currently more promising than before, since researchers have histochemical, immunohistochemical, electron microscopic, morphometric and other precision methods at their disposal, the use of which contributes to a more complete disclosure of the essence of this pathology. All this allows us to conduct a study of the pathological morphology of the lungs in poisoning at a new level and is a guarantee that a properly planned and well-supported study in terms of method can end with the receipt of results that are promising both in theoretical and practical terms.

Keywords: lungs, toxic damage, diagnostic methods.

Introduction. The lungs are quite often the target of toxic damage. The structural and functional features of the lungs, significant transit of atmospheric air and blood determine the high intensity of organ damage when exposed to various exogenous and endogenous chemical factors. The most common and frequently encountered is poisoning with carbon monoxide, volatile organic compounds (carbon tetrachloride, ethers, aldehydes, aromatic alcohols, etc.), volatile acids and alkalis. Poisoning can occur both at work and at home - through carelessness or for suicidal purposes. According to the authors, in large cities of the world, acute toxic lung damage accounts for from 3 to 8% of all fatal poisonings (Vdovina, N.V., 2024; Colunga Biancatelli, R.M.L., 2021) [3,14].

Developing intoxication quickly leads to tissue damage, microcirculatory disorders and contributes to the formation of acute respiratory failure. This condition is described as "adult respiratory distress syndrome", "toxic pneumopathy", "toxic pulmonary edema" and ultimately often determines thanatogenesis (Kirillov, Yu.A., 2020; Cereda, M., 2014) [5,11].

The two main routes of endotoxin entry into the lung tissue are fundamentally different: inhalation and non-inhalation. At the same time, the form of their non-inhalation penetration may differ somewhat depending on the localization of the primary source of toxins - the gastrointestinal tract, lymph nodes, foci of tissue damage in soft tissues, blood (Avdeev, S.N., 2019; Choi, J., 2020) [1,13]. It is well known that the mechanisms of subsequent damage to lung tissue with the described routes of entry are somewhat different. Recently, the attention of researchers has been attracted by toxic pneumonia; developing when bacterial lipopolysaccharide enters the lung tissue through the air: This is possible not only due to the product entering from the atmosphere (including non-sterile air circuits), but also due to the death of microorganisms in the upper respiratory tract or oral cavity, followed by penetration of the toxin through the surfactant layer into the lung tissue (Aleksandrov, N.S., 2017; Bolourani, S., 2021) [2,10].

The interalveolar septum contains epithelial cells of type 1 and type 2 (epithelial cells), alveolar macrophages, endothelium; a small number of fibroblasts and transient leukocytes: Their representation, localization in relation to the lumens of capillaries and alveoli, and receptor apparatus differ significantly (Dvorakovskaya, I.V., 2021; Childs, B.G., 2019) [4,12]. This should have

consequences in the form of differences in the morphogenesis of inhalation and non-inhalation entry of toxins into the lung tissue, but work in this direction is rare and contradictory.

From this perspective, an experimental study of morphofunctional changes in the lungs during acute inhalation and non-inhalation penetration of toxins of various natures into them seems relevant.

At present, the main causes, precursors and conditions, the unfavorable combination of which can cause toxic lung damage, can be considered established and sufficiently studied. The few studies contain information on the intermediate and final phases of atelectasis morphogenesis, after which atelectasis either resolves with the restoration of lung airiness to one degree or another, or passes into a chronic stage with transformation into fibroatelectasis and sclerosis of lung tissue. As for the initial stages of intoxication morphogenesis, that is, the period when most of the emerging general pathological processes, including damage and circulatory disorders, are reversible and restitution of lung tissue is possible, they represent a kind of “terra incognita” (Jarzebska, N., 2021) [16]. All this is fully characteristic of pathologies arising as a result of radiation-induced exposure to lung tissue (Kirillov, Yu.A., 2020) [5]. Recent assumptions about the role of oxidative stress and endothelial dysfunction in the occurrence and development of pathologies are generally not supported by morphological evidence (Li, F., 2021) [19]. For the treatment of the initial stage of atelectasis, the morphological manifestations of which are hypoxia-induced interstitial and intraalveolar edema of varying severity, antiexudative agents and glucocorticoids are used (Avdeev, S.N., 2019) [1]. At the same time, the specificity of radiation damage to the lungs dictates the need to use drugs with antioxidant, radioprotective and antifibrotic effects (Judge, J.L., 2017; Mungunsukh, O., 2021) [17,20].

The causes of lung injury are quite wide, ranging from traumatic chest injury, consequences of extensive lung surgery, viral pneumonia, body surface burns, inhalation trauma, general anesthesia, bronchial obstruction and various exogenous effects, including chemical or radiation damage to the lungs. Recently, due to the widespread use of radiation exposure as a method of treating patients with malignant neoplasms of the thoracic and intrathoracic localization, including breast cancer, included in clinical guidelines for the management of patients with this pathology (Clinical guidelines "Malignant neoplasm of the bronchi and lung", MH of the RF, 2021), the incidence of lung pathologies has slightly increased (Kavanaugh, J., 2019) [6,18]. Clinical observation data indicate that in approximately a third of cases, various signs of pulmonary pathology may appear after radiation therapy (Sumita, K., 2016) [21]. In manuals on pulmonology and human pathological anatomy, in the section on the morphology and pathogenesis of atelectasis, obstructive, compression and neurogenic atelectasis are distinguished (Chernyaev, A.L., 2000) [8]. Research in recent years, while unconditionally recognizing the dominant role of obstruction and/or significant limitation of the lumen of the airways in the formation of pathologies, nevertheless, state, firstly, the presence of a significantly larger number of factors influencing patho- and morphogenesis and, secondly, their unconditional conjugacy and implementation in different time periods (Hur, W., 2017) [15]. At the same time, the small number of works on modeling toxic lung damage in an experiment makes it difficult to understand and evaluate the initial changes in the lung tissue that constitute the essence of the first links in the morphogenesis of atelectasis. Therefore, the identification of specific mechanisms of alteration underlying the emergence and implementation of pathologies seems to be an urgent task (Rosenberg, O.A., 2014) [7]. In contrast to clinical observations, in which intravital morphological diagnostics of toxic lung damage is usually difficult, and the study of autopsy material in most cases makes it extremely difficult to use precision methods for studying biological objects, including electron microscopic examination of the material, experimental models make it possible to study and describe the morphogenesis of this pathology completely, starting from the stage of initial damage and ending with the resolution of atelectasis or pulmonary fibrosis (Beike, L., 2019) [9]. In addition, the problem of developing and using new methods, technologies and drugs for the prevention and treatment of toxic lung damage is also relevant.

Заключение. Анализ морфогенеза токсических повреждений легких и возможностей его диагностики в настоящее время является более перспективным, нежели ранее, поскольку в распоряжении исследователей имеются гистохимические, иммуногистохимические,

электронно-микроскопические, морфометрические и иные прецизионные методы, применение которых способствует более полному раскрытию существа данной патологии. Все это позволяет проводить исследование патологической морфологии легких при отравлениях на новом уровне и является гарантом того, что правильно спланированное и хорошо обеспеченное в методическом отношении исследование может завершиться получением результатов, перспективных как в теоретическом, так и практическом отношениях.

Conclusion. The analysis of the morphogenesis of toxic lung damage and the possibilities of its diagnostics is currently more promising than before, since researchers have histochemical, immunohistochemical, electron microscopic, morphometric and other precision methods at their disposal, the use of which contributes to a more complete disclosure of the essence of this pathology. All this allows us to conduct a study of the pathological morphology of the lungs in poisoning at a new level and is a guarantee that a properly planned and well-supported study in terms of method can end with the receipt of results that are promising both in theoretical and practical terms.

References.

1. Avdeev, S.N. The role and significance of inhaled glucocorticosteroids in patients with chronic obstructive pulmonary disease: current recommendations. Conclusion of the expert council. / S.N. Avdeev, Z.R. Aisanov, V.V. Arkhipov, A.S. Belevsky, I.V. Leshchenko, A.I. Sinopalnikov // Pulmonology. - 2019. - Vol. 29 (5) - P. 632–636.
2. Aleksandrov, N.S. Using Raman fluorescence spectroscopy for the diagnosis of clear cell renal cell carcinoma / N.S. Aleksandrov, S.T. Avraamova, Yu.A. Kirillov, T.O. Babicheva, V.I. Kukushkin, D.N. Artemyev // Clinical and experimental morphology. – 2017. – T. 4(24) – P. 59–65.
3. Vdovina, N. V. The human body: life processes and their regulation: monograph / N. V. Vdovina. - 2nd ed., revised. and add. - Moscow: Yurait Publishing House, 2024. - 391 p.
4. Dvorakovskaya, I. V. Pathological anatomy of vascular lesions in influenza and viral-bacterial pneumonia / I. V. Dvorakovskaya, O. N. Titova, B. M. Ariel, I. S. Platonova, V. A. Volchkov, N. A. Kuzubova // Russian Medical Journal. - 2021. - Vol. 3 - P. 3–7.
5. Kirillov, Yu.A. Morphogenesis of pulmonary atelectasis under experimental radiation-induced exposure / Yu.A. Kirillov, I.A. Chernov, E.M. Malysheva, S.E. Timofeev, et al. // Clinical and experimental morphology. - 2020. - Vol. 9 - № 1.
6. "Clinical guidelines "Malignant neoplasm of the bronchi and lung" (approved by the Ministry of Health of Russia) [Electronic resource] / "Clinical guidelines. - 2021. - URL: https://cr.minzdrav.gov.ru/recomend/30_3 (date of access: 11/12/2021).
7. Rosenberg, O.A. Pulmonary surfactant preparations in acute and chronic lung diseases (part I) / O.A. Rosenberg // General reanimatology. - 2014. - Vol. 10 (4) - P. 51-73.
8. Chernyaev, A.L. Pulmonary atelectasis. In the book: Cellular biology of the lungs in health and pathology: a guide for doctors / Ed. V.V.Erohin, L.K.Romanova. / A.L. Chernyaev. - M .: Medicine, 2000. - Vol. 1 - P. 384-385.
9. Beike, L. Surfactant dysfunction and alveolar collapse are linked with fibrotic septal wall remodeling in the TGF-β1-induced mouse model of pulmonary fibrosis / L. Beike, C. Wrede, J. Hegermann, E. Lopez-Rodriguez, C. Kloth, J. Gauldie, M. Kolb, U.A. Maus, M. Ochs, L. Knudsen // Laboratory Investigation. – 2019. – Vol. 99 – № 6 – P. 830–852.
10. Bolourani, S. The interplay of DAMPs, TLR4, and proinflammatory cytokines in pulmonary fibrosis / S. Bolourani, M. Brenner, P. Wang // Journal of Molecular Medicine. – 2021. – Vol. 99 – № 10 – P. 1373–1384.
11. Cereda, M. Hyperpolarized gas diffusion MRI for the study of atelectasis and acute respiratory distress syndrome / M. Cereda, Y. Xin, S. Kadlecek, H. Hamedani, J. Rajaei, J. Clapp, R.R. Rizi // NMR in biomedicine. – 2014/06/11 ed. – 2014. – Vol. 27 – № 12 – P. 1468–1478.

12. Childs, B.G. Cellular Identification and Quantification of SenescenceAssociated β-Galactosidase Activity In Vivo. / B.G. Childs, T.J. Bussian, D.J. Baker // Methods in molecular biology (Clifton, N.J.). – 2019. – Vol. 1896 – P. 31–38.

13. Choi, J. Inflammatory Signals Induce AT2 Cell-Derived DamageAssociated Transient Progenitors that Mediate Alveolar Regeneration / J. Choi, J.-E. Park, G. Tsagkogeorga, M. Yanagita, B.-K. Koo, N. Han, J.-H. Lee // Cell Stem Cell. – Elsevier, 2020. – Vol. 27 – № 3 – P. 366-382.e7.

14. Colunga Biancatelli, R.M.L. Age-Dependent Chronic Lung Injury and Pulmonary Fibrosis following Single Exposure to Hydrochloric Acid. / R.M.L. Colunga Biancatelli, P. Solopov, C. Dimitropoulou, J.D. Catravas // International journal of molecular sciences. – 2021. – Vol. 22 – № 16.

15. Hur, W. Molecular Pathogenesis of Radiation-Induced Cell Toxicity in Stem Cells / W. Hur, S.K. Yoon // International Journal of Molecular Sciences. – 2017. – Vol. 18 – № 12.

16. Jarzebska, N. Scarred Lung. An Update on Radiation-Induced Pulmonary Fibrosis / N. Jarzebska, E.S. Karetnikova, A.G. Markov, M. Kasper, R.N. Rodionov, P.M. Spieth // Frontiers in Medicine. – 2021. – Vol. 7 – P. 1100.

17. Judge, J.L. The Lactate Dehydrogenase Inhibitor Gossypol Inhibits Radiation-Induced Pulmonary Fibrosis. / J.L. Judge, S.H. Lacy, W.-Y. Ku, K.M. Owens, E. Hernady, T.H. Thatcher, J.P. Williams, R.P. Phipps, P.J. Sime, R.M. Kottmann // Radiation research. – 2017. – Vol. 188 – № 1 – P. 35–43.

18. Kavanaugh, J. Anatomical Adaptation-Early Clinical Evidence of Benefit and Future Needs in Lung Cancer. / J. Kavanaugh, G.D. Hugo, C. Robinson, M. Roach // Seminars in radiation oncology. – 2019. – Vol. 29 3 – P. 274–283.

19. Li, F. Risk factors for radiation pneumonitis in lung cancer patients with subclinical interstitial lung disease after thoracic radiation therapy / F. Li, H. Liu, H. Wu, S. Liang, Y. Xu // Radiation Oncology. – 2021. – Vol. 16 – № 1 – P. 70.

20. Mungunsukh, O. Captopril reduces lung inflammation and accelerated senescence in response to thoracic radiation in mice / O. Mungunsukh, J. George, E.A. McCart, A.L. Snow, J.J. Mattapallil, S.R. Mog, R.A.M. Panganiban, D.L. Bolduc, W.B. Rittase, R.M. Bouten, R.M. Day // Journal of Radiation Research. – 2021. – Vol. 62 – № 2 – P. 236–248.

21. Sumita, K. Re-irradiation for locoregionally recurrent tumors of the thorax: a single-institution, retrospective study / K. Sumita, H. Harada, H. Asakura, H. Ogawa, T. Onoe, S. Murayama, S. Nakamura, N. Tanigawa, T. Takahashi, T. Nishimura // Radiation Oncology (London, England). – 2016. – Vol. 11 – P. 104.