EUROPEAN JOURNAL OF
MODERN MEDICINE AND PRACTICE
Vol. 6 No. 02 (Feb - 2026) EJMMP ISSN: 2795-921X

https://inovatus.es/index.php/ejmmp

INTERPRETATION OF MORPHOLOGICAL CHANGES IN INTERNAL ORGANS DURING TRAUMATIC SHOCK

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Abstract: This scientific article investigates the morphological changes resulting from traumatic shock based on the study of 27 autopsy cases using wet archive materials. The objects of study included data from re-commission examinations and forensic histological reports conducted between 2024 and 2025 at the Republican Scientific and Practical Center of Forensic Medical Examination for cases diagnosed with shock of various etiologies. The analysis revealed a series of common pathomorphological changes in the autopsy materials of individuals who died from traumatic shock. It was found that morphological changes in internal organs during severe and irreversible traumatic shock develop not only due to the direct impact of the injury but also as a result of systemic microcirculatory failure, hypoxia, and disseminated intravascular coagulation (DIC syndrome).

Keywords: Traumatic shock, wet archive, parenchymal organs, morphological changes.

Relevance

Shock is a severe pathological process characterized by acute failure of the systems supporting vital functions. Statistics indicate that mortality rates for various types of shock (traumatic, hypovolemic, septic, anaphylactic, or cardiogenic) remain high, ranging from 30% to 80% [1,2,3]. Its high prevalence among the young, working-age population causes significant medical and economic damage.While clinical presentations vary, the underlying pathogenetic mechanism is systemic microcirculatory failure[4,5,6]. Traumatic shock: Intense pain impulses and blood loss play a leading role.Septic shock: Effects of infectious toxins on vascular walls and metabolism take priority.Cardiogenic shock: Primary damage to the heart's pumping function occurs [7,8,9].All types eventually lead to disseminated intravascular coagulation (DIC), tissue hypoxia, and cellular

destruction. Experts face the "macro-microscopic dissociation" phenomenon; for instance, organs may appear intact externally in traumatic shock, but microscopic signs like "Shock lung" (hyaline membranes, interstitial edema), "Shock kidney" (acute tubular necrosis), and dystrophic changes in the liver and heart serve as morphological confirmation[10,11,12].Shock is now viewed as Multiple Organ Dysfunction Syndrome (MODS). Currently, there is a necessity to apply ultrastructural and immunohistochemical methods alongside standard H&E staining to detect early cellular changes, such as mitochondrial lysis and receptor degradation.

Aim of the Work

The study aims to analyze morphological changes in cases diagnosed with shock of various etiologies based on re-commission examinations and forensic histological data from 2024–2025 at the Republican Scientific and Practical Center of Forensic Medical Examination.

Materials and Methods

The primary materials for this scientific research consisted of 27 autopsy cases involving wet archive specimens from individuals whose cause of death was confirmed as traumatic shock. The objects of the study included, Data from forensic histological departments and re-commission examinations conducted during the 2024–2025 period at the Republican Scientific and Practical Center of Forensic Medical Examination.Supplemental referral documentation provided for forensic analysis.Wet archive samples of parenchymal organs for detailed microscopic evaluation.

Results and Discussion

The most characteristic feature of traumatic shock is the redistribution of blood, which was especially clearly observed in the lung tissue. Sludge phenomenon and stasis were identified in numerous capillaries and venules, manifested by aggregation of erythrocytes, i.e., adhesion of red blood cells to one another.Disseminated intravascular coagulation (DIC) syndrome was also detected, characterized by the formation of fibrin thrombi in small blood vessels. These changes were particularly prominent in the capillaries of the kidneys, lungs, and liver.Increased vascular permeability, resulting from damage to the vascular walls, led to plasmorrhagia and interstitial edema. Thus, blood redistribution remains the most characteristic morphological feature of traumatic shock.

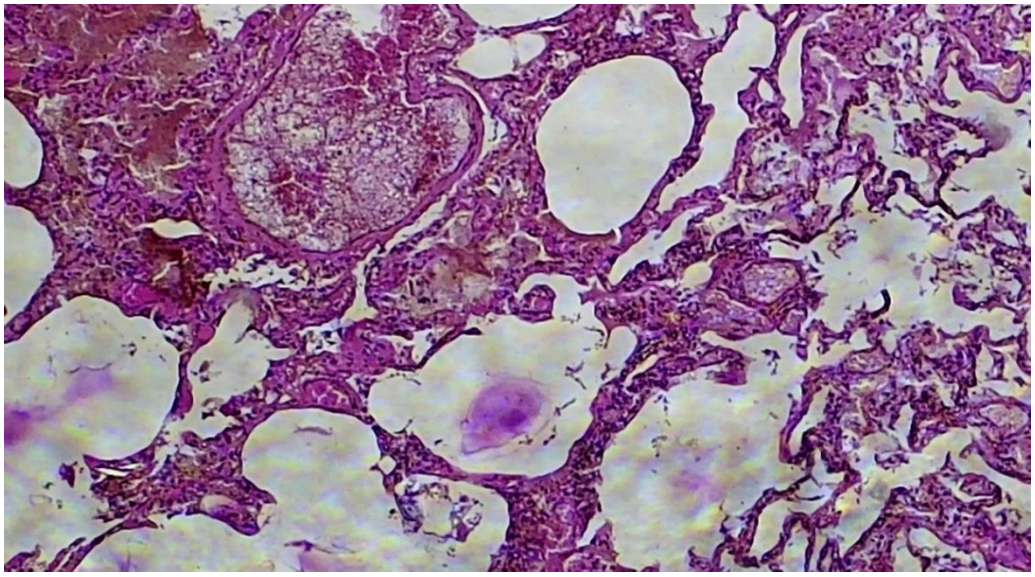

Fig. 1. Lung tissue in traumatic shock: development of emphysematous foci, marked vascular congestion, and focal hemorrhages in the stroma. Stain: Hematoxylin and Eosin (H&E). Magnification: ×100 (10×10).

Autopsy materials from individuals who died due to traumatic shock revealed a number of common pathomorphological changes. In cases of severe and irreversible traumatic shock, morphological alterations in internal organs were found to result not only from the direct impact of trauma, but also from systemic microcirculatory disturbances, hypoxia, and disseminated intravascular coagulation (DIC syndrome) (Figure 2).

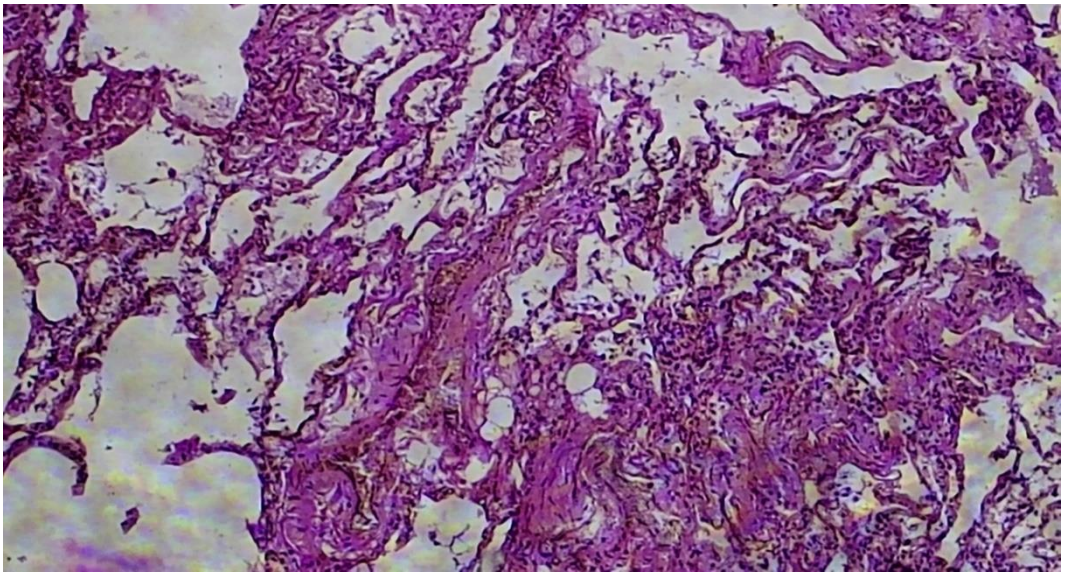

Fig 2. Lung tissue in traumatic shock: presence of emphysematous foci and stromal hemorrhages. Stain: Hematoxylin and Eosin (H&E). Magnification: ×100 (10×10).

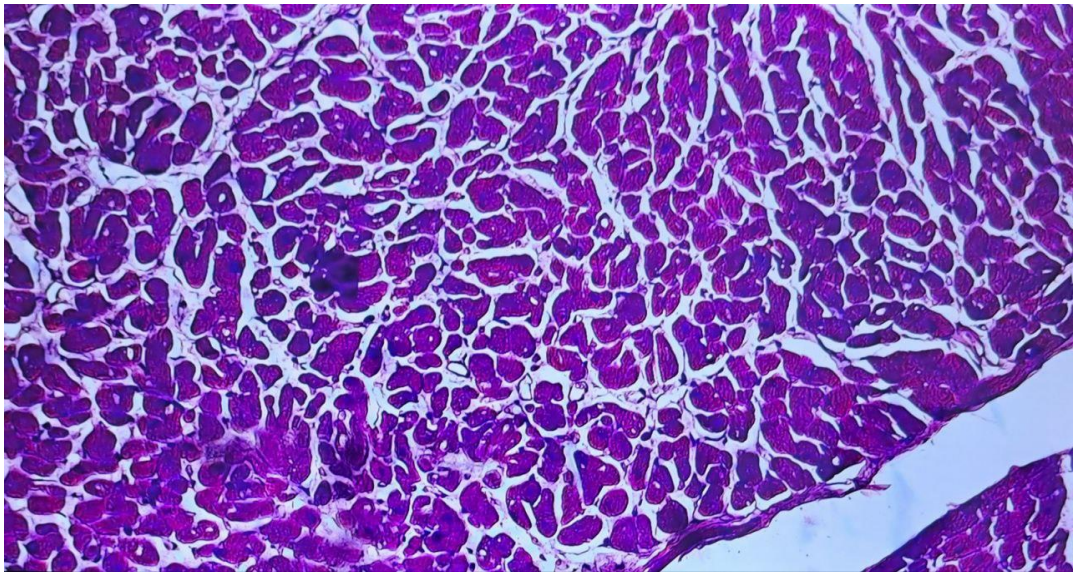

Figure 3. Destructive and dystrophic changes of cardiomyocytes, fragmentation of muscle fibers, and interstitial edema. Stain: Hematoxylin and Eosin (H&E). Magnification: ×400 (10×40).

Pronounced dystrophic and destructive processes were observed in the myocardial cells. Cardiomyocyte hypertrophy and dystrophy were evident, along with uneven thickening of muscle fibers and granular (protein) degeneration of the cytoplasm.Nuclei of cardiomyocytes appeared hyperchromatic and polymorphic. Fragmentation of myofibrils was present. Wavy deformation and segmentation of muscle fibers were clearly identified, reflecting acute contractile insufficiency of the myocardium during shock.Interstitial edema fluid was detected between cardiomyocytes, leading to separation of muscle fibers from one another.

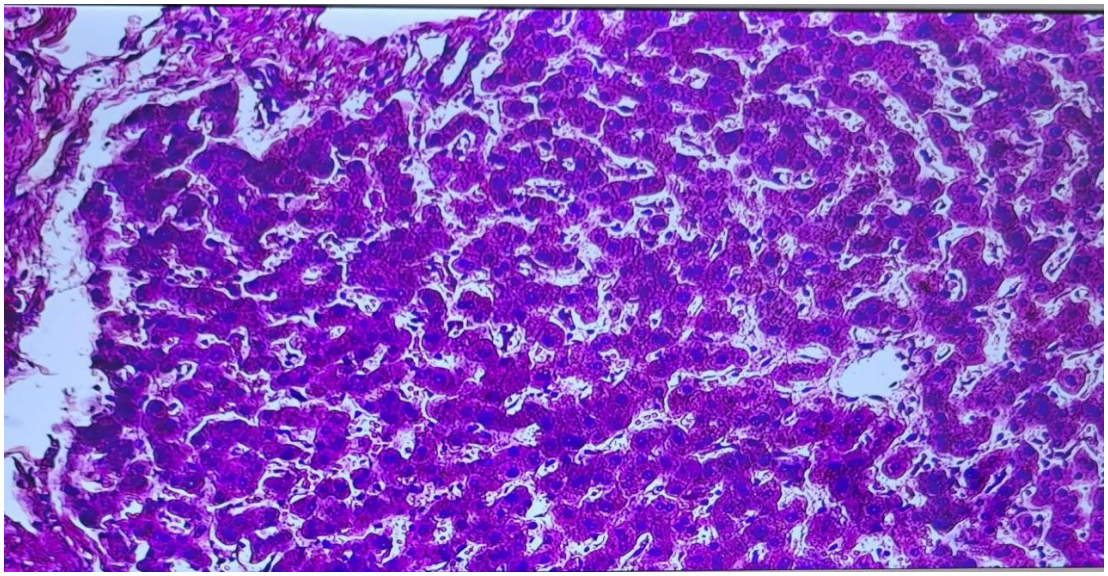

Figure 4. Hypoxic and dystrophic changes in hepatocytes with centrilobular necrosis. Stain: Hematoxylin and Eosin (H&E). Magnification: ×400 (10×40).

Pronounced hypoxic and dystrophic alterations were observed in hepatocytes. Centrilobular necrosis was identified, characterized by necrotic changes in hepatocytes located in the central zones of hepatic lobules, accompanied by nuclear fragmentation (karyorrhexis).Marked blood stasis was present within hepatic sinusoids. Morphological features consistent with disseminated intravascular coagulation (DIC syndrome), including fibrin thrombi in the microvasculature, were also detected.

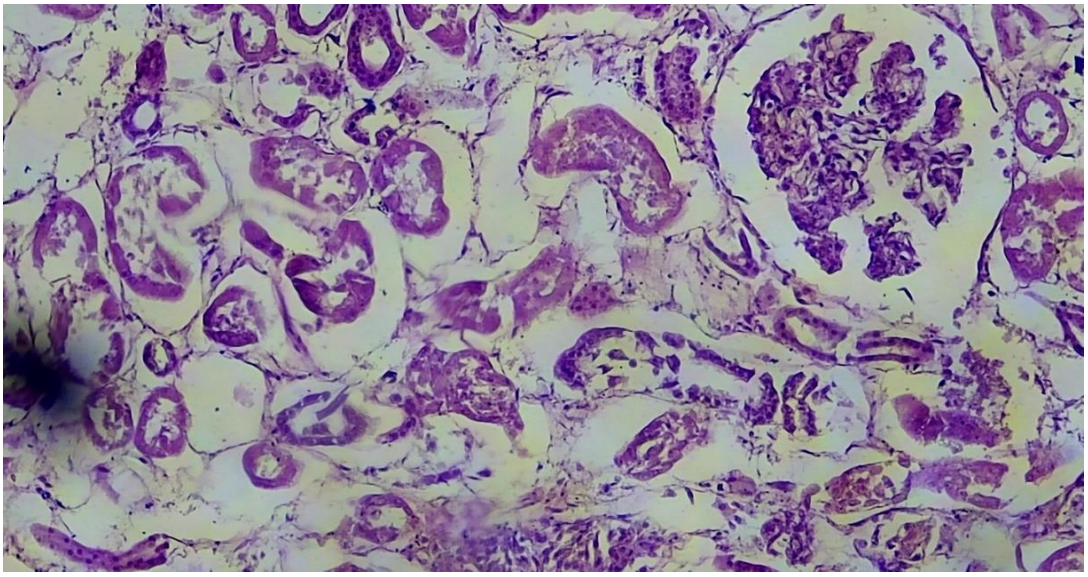

Figure 5. Kidney tissue in traumatic shock: massive necrosis of convoluted tubules, microvascular alterations, and glomerular atrophy. Stain: Hematoxylin and Eosin (H&E). Magnification: ×400 (10×40).

Acute tubular necrosis (Figure 5) was clearly identified. The epithelium of both proximal and distal tubules appeared markedly swollen; nuclei showed lysis and, in some cells, pyknotic condensation, appearing dark and shrunken. These findings indicate acute hypoxic injury leading to cellular death.Narrowing of the tubular lumen was observed due to swollen epithelial cells obstructing the tubular pathways. In certain areas, cellular debris (detritus) was present within the lumen.Glomeruli were relatively preserved; however, slight widening of Bowman's capsule space was noted, suggesting reduced renal perfusion and impaired filtration.Interstitial edema fluid was detected in the connective tissue between tubules, reflecting microcirculatory disturbances associated with traumatic shock.

Conclusion

Based on the comprehensive pathomorphological analysis of 27 autopsy cases, the following conclusions were drawn. Systemic Response: morphological changes in internal organs during traumatic shock are not merely a direct result of mechanical injury but a consequence of systemic microcirculatory collapse, severe hypoxia, and the progression of DIC syndrome.Pathognomonic Markers: the redistribution of blood, intravascular sludge phenomenon, plasmorrhagia, and interstitial edema are the most characteristic morphological markers of the shock state.Target Organs, the most prominent manifestations of shock are observed in the lung ("shock lung") and kidney ("shock kidney") tissues. Hyaline membranes in the alveoli and acute tubular necrosis serve as the primary pathomorphological substrates of the terminal mechanism.Forensic Significance, the morphological interpretations presented in this study are of critical importance for the objective substantiation of traumatic shock diagnosis and the clarification of the cause of death in forensic medical practice.

References

1. Avdeev, A. I. Forensic medical examination in blunt chest trauma / A. I. Avdeev. – Khabarovsk: Publishing House of Far Eastern State Medical University, 2005. – 164 p.
2. Zhelev, V. A. Clinical and biochemical markers of myocardial injury in premature newborns / V. A. Zhelev et al. // *Bulletin of Siberian Medicine*. – 2007. – No. 4. – P. 86–90.
3. Pigolkin, Yu. I. Forensic Medicine: National Guidelines / Yu. I. Pigolkin. – Moscow: GEOTAR-Media, 2015. – 640 p.
4. Romanov, A. B. Ablation of incisional atrial flutter in an adolescent after surgical correction of Tetralogy of Fallot / A. B. Romanov et al. // *Journal of Arrhythmology*. – 2021. – Vol. 28, No. 4 (106). – P. 52–56.
5. Rudaeva, E. V. Congenital heart defects and pregnancy / E. V. Rudaeva et al. // *Fundamental and Clinical Medicine*. – 2019. – Vol. 4, No. 3. – P. 102–112.
6. Rudyuk, L. A. Macroscopic placental changes in pregnancies complicated by congenital heart defects / L. A. Rudyuk, O. S. Reshetnikova // *Clinical and Experimental Morphology*. – 2019. – Vol. 8, No. 1. – P. 19–26.
7. Ergashbaeva, D. A. Cardiointervalographic assessment of early neonatal adaptation / D. A. Ergashbaeva et al. // *Obstetrics and Gynecology*. – 2008. – No. 2. – P. 19–21.

8. Maron, B. J. Commotio cordis / B. J. Maron, N. A. Estes III // *New England Journal of Medicine*. – 2010. – Vol. 362, No. 10. – P. 917–927.
9. Link, M. S. Commotio cordis: ventricular fibrillation triggered by chest wall impact / M. S. Link // *Cardiovascular Pathology*. – 2014. – Vol. 23, No. 1. – P. 2–6.
10. Geddes, L. A. Evolution of our knowledge of sudden death due to commotio cordis / L. A. Geddes, R. A. Roeder // *American Journal of Emergency Medicine*. – 2005. – Vol. 23, No. 1. – P. 67–75.
11. Saukko, P. Knight's Forensic Pathology / P. Saukko, B. Knight. – 4th ed. – London: CRC Press, 2015. – 692 p.