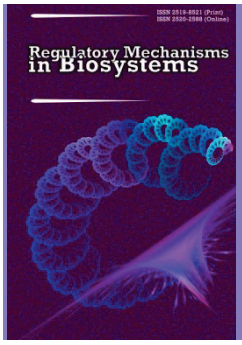

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Forensic veterinary diagnosis of systemic hemostatic disorders and sudden death in cats and dogs: Thanatogenetic aspect of critical conditions

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During the forensic veterinary examination of corpses of dogs and cats that died suddenly due to myocardial dystrophy, in refractory critical conditions, and as a result of systemic coagulopathy, Simpson's principle and Sutton's rule were adapted in compliance with the standard of "object examination at different structural levels" of evidence-based veterinary medicine. The corresponding thanatogenetic models were experimentally, clinically, and laboratory-instrumentally substantiated. To achieve the research goal, the corpses of dogs and cats of various ages were grouped into cohorts, and autopsy, laboratory-instrumental, and philosophical-gnoseological research methods were applied. It was proven that among the morphological criteria determining the cause of death in animals, it is advisable to distinguish specific patterns, which are reflected when substantiating a forensic veterinary diagnosis. It was shown that in cases of "acute cardiac death" in animals, myocardial fiber contracture deformation, hemocapillary hyperemia, erythrocyte stasis, vascular spasm, and plasma imbibition of the walls of microcirculatory vessels in the myocardium were observed. Together, these can be considered morphological criteria for cardiac death resulting from chronic heart hypoxia. It was argued that in cases of systemic coagulopathy, animal death occurs directly as a result of hemorrhagic hypovolemia against the background of distributive shock, which is confirmed by Minakov's sign. It was proven that the micromorphological patterns of sepsis are focal lesions of the microcirculatory vessels of the kidneys, providing diagnostically informative morphological criteria for the probability of a systemic inflammatory response. It was argued that the application of clinical logic concepts narrows the differential diagnostic scope in cases of death due to refractory terminal conditions. Future research prospects include a comprehensive forensic veterinary assessment of the diagnostic informativeness of biotransformation phenomena in dog and cat corpses in various conditions.

Keywords: forensic veterinary examination; coagulopathy; systemic inflammatory response; sepsis; cardiac death; terminal conditions; distributive shock.

Introduction

Analysis of statistical data indicates a fairly high prevalence of heart pathologies (14.9%) among the total number of all diseases diagnosed in dogs and cats during primary examination at veterinary medical facilities. In dogs, the most common condition is mitral valve endocardiosis (Iakovlev & Petrov, 2023). The formation of excess valve tissue, which mainly affects the entire anterior leaflet, leads to its thickening and prolapse, causes stretching and rupture of the chordae tendineae, and contributes to cardiac death (O'Brien et al., 2021).

The manifestation of the hypertrophic cardiomyopathy phenotype remains the leading cause of sudden death at the subclinical stage in cats due to complications, primarily pulmonary thromboembolism and cardiogenic pulmonary edema (Inoue et al., 2016). Despite the prevalence of hypertrophic cardiomyopathy in cats, the prognostic question of developing fatal complications at the subclinical stage remains unresolved (März et al., 2015). Dilated cardiomyopathy is the dominant condition with a poor prognosis in dogs, characterized by rapidly progressive left-sided or bilateral dilation of the heart chambers and a decrease in systolic function. This cardiomyopathy phenotype is the main cause of sudden death in clinically healthy dogs due to the progression of congestive heart failure syndrome (Jaffey et al., 2019) and pulmonary hypertension (Reinero et al., 2020).

Animals with severe injuries, sepsis, or a history of inflammatory lung diseases should be considered at risk of developing acute pulmonary failure (Zamorska et al., 2021). Based on randomized studies, a classification of forms of pulmonary edema depending on the etiological factor has been established. Cardiogenic edema occurs due to increased hydrostatic pressure in the pulmonary capillaries caused by left-sided heart failure, which is triggered by dilated cardiomyopathy, acquired mitral regurgitateon, and hypertrophic cardiomyopathy (Kittleson & Côté, 2021). Common causes of non-cardiogenic pulmonary edema include upper airway obstruction and sepsis. Acute respiratory distress syndrome leads to a decrease in colloid-osmotic pressure or changes in the resistive density of pulmonary capillaries, as indicated by Petrushko & Grushanska (2022).

In cases of sudden cardiac death in animals, as noted by Szeremeta et al. (2023), characteristic morphological changes in organs and tissues do not have time to develop. In this context, relying solely on forensic autopsy data, particularly in unclear cases, can, according to Touroo et al. (2020), lead to establishing incorrect causal relationships and, accordingly, to a mistaken expert conclusion, as demonstrated by Fedyk & Besaha (2023) in the context of judicial proceedings.

When determining the thanatogenesis of sudden death in humans, special logical methods adapted in forensic medicine, particularly cardiology, are applied (Kim et al., 2022). Simpson's principle emphasizes the need for diagnostic testing only if the predicted result alters the clinical hypothesis regarding the cause of death, while Sutton's rule states that in diagnostics, one should first consider the obvious and conduct studies that confirm the most probable fatal diagnosis.

It is widely known that the onset of sudden death, even despite emergency veterinary assistance, reflects the critical condition of the animal.

According to the generally accepted triage algorithm, a veterinarian determines the priority of assistance based on an evaluation of the animal's clinical status using functional and biochemical parameters. Today, it is believed that the key to a successful therapeutic strategy for urgent conditions is the timely prevention of fatal complications, primarily systemic coagulopathy, systemic inflammatory response, distributive shock, etc. (Sharp et al., 2019).

Drawing on observations by Gando & Otomo (2015), the detailed pathomorphogenesis of systemic coagulopathy syndrome has been described. The researchers distinguish the following stages of the syndrome: hypercoagulation and aggregation of blood elements, consumption coagulopathy, deep hypocoagulation and fibrinolysis, and the stage of residual thrombosis and blockages. It is stated that the syndrome manifests due to widespread thrombosis resulting from excessive consumption of coagulation factors and the activation of fibrinolysis. The Starling model, which is now accepted by most researchers, explains the occurrence of systemic coagulopathy through the damage to the endothelial glycocalyx observed during severe traumatic disease and septic conditions in humans (Kravets et al., 2023).

In the modern understanding of systemic inflammatory response syndrome and sepsis, it is important to detail some aspects of their pathogenesis due to the high mortality rate. In this direction, Singer et al. (2016) summarized observations of critical human conditions, noting that the clinical criteria for systemic inflammatory response are nonspecific and are more likely a result of morphofunctional adaptation rather than a pathological reaction of the body. On the other hand, systemic inflammation may be accompanied by septic shock or, conversely, trigger it, which requires differential diagnosis with the obligatory identification of the bacterial component (Fernando et al., 2018).

An interesting randomized study was conducted by Rebollada-Merino et al. (2020), in which the structured nosology of violent deaths, including accidents and poisonings, in dogs and cats over five years was analyzed based on fatal injury factors. Significant contributions to forensic veterinary toxicology were made by Schmid et al. (2017), who demonstrated the fatal role of hemostatic disorders in dog poisoning with isoniazid compounds, and by Kotsyumbas & Vretsona (2021), who defined their morphological criteria.

One of the controversial issues in clinical veterinary medicine from a bioethical perspective remains euthanasia, which involves a "weighed decision" by the veterinary professional. In this context, Bubeck (2023) argues that forced animal death through euthanasia takes various legal forms and presents complex ethical problems due to the involvement of different parties and the wide range of responsibilities of the veterinarian.

Given the above, the aim of this work is to substantiate the causal relationships in the thanatogenesis of sudden animal death and the adverse outcomes of critical conditions and systemic coagulopathy based on an original approach, avoiding overdiagnosis, as supported by the results of original research.

Materials and methods

In the experimental part, modern clinical research standards were applied: cohorts of subjects, patterns, inclusion and exclusion criteria, statistical sampling, representativeness, randomization, evidence levels, and digitization methods.

The theoretical part of the study was a multicenter retrospective analysis of forensic veterinary expert reports from 2013 to 2023. The subjects of the empirical part of the study were dogs and cats and their corpses (60 dogs and 40 cats aged from 1 day to 10 years). The inclusion criteria for the study were sudden death of dogs and cats, fatal outcomes of critical conditions, and systemic coagulopathy of various etiopathogenesis. The exclusion criteria were non-fatal outcomes of critical conditions and systemic coagulopathy in dogs and cats.

Prospective studies were conducted in veterinary medical institutions from 2020 to 2023 by observing changes in the clinical status of animals during the interval from hospital admission to the resulting outcome, averaging 3 ± 1 days. Animals with a common clinical diagnosis were sorted into cohorts. In this study, quantitative levels of blood biochemical markers, except for lactate, were not considered, as anamnesis morbi revealed that the values varied across a statistically wide range depending on the criticality of the status praesens objectivus.

All diagnostic procedures were performed, and some phenomena explained, following bioethical principles in veterinary medicine. The animals' hemostatic system was evaluated using activated partial thromboplastin and prothrombin time indices, determined using "Granum-activated partial thromboplastin time" (Ukraine) and "Granum-prothrombin time" (Ukraine) test kits. These indicators were used to interpret hyper/hypocoagulatory shifts, thrombosis/thromboembolisms, and coagulopathies of various etiologies. For sample preparation, blood samples were taken from the v. saphena vein into a 4 cm^3 plastic tube (Ukraine) with 3.2% sodium citrate solution at a 9:1 blood-to-citrate ratio. The blood was centrifuged at 3000 rpm for 15 minutes using a "Sigma 1-7" centrifuge (Germany, 2020). Centrifugation was performed immediately after blood collection, and plasma was extracted immediately after centrifugation for further analysis. According to good laboratory practice standards, reference values for activated partial thromboplastin time were 23–39 seconds, and for prothrombin time, 8–15 seconds, calculated by averaging data from clinically healthy individuals. Hemolyzed, lipidemic, clotted, or samples older than 1 hour (for activated partial thromboplastin time) and 2 hours (for prothrombin time) were not used in the study. Attention was drawn to the fact that the study evaluated the state of the hemostatic system and assessed the coagulation potential of different target organs. This methodological approach was chosen because analyzing coagulation-lytic system parameters in blood coagulograms alone cannot adequately assess the potential for local thrombus formation, particularly in initiating the coagulation cascade and consequent ischemic tissue damage.

In each expert case, autopsy of animal corpses was performed following the authors' methodology no earlier than two hours after death (Yatsenko & Kazantsev, 2023). Metodyka sudovo-veterynarnoho doslidzhennya trupiv tvaryn [Methods of forensic veterinary examination of animal corpses (code 18.1.02), https://rmpse.minjust.gov.ua/page/29).

For microscopic examination of tissue loci, cylindrical necropsy samples were taken during autopsy using a Panch device (China, 2020), histological slides were prepared, and then stained with hematoxylin and eosin to study the structure, and Sudan Black B to detect lipid compounds in the myocardium. Histological techniques followed standard methods (Horalskyi et al., 2015). The obtained histotopograms were evaluated under a "Granum R50" optical microscope (China, 2021). The most representative changes were recorded using a "ToupCam UCMOS03100KPA" camera (China, 2021) integrated with the microscope. The data were interpreted according to the recommendations of Raskin et al. (2022).

For rapid detection of free fluid in cavities, targeted ultrasonographic examinations of the abdomen and chest were performed using the AFAST and TFAST methods on a "CHISON CBit 9" machine (China, 2023), digital radiography with an "X-ray Device PCMAX-40HBP" (Korea, 2023) in the lateral projection, and X-ray tomography with a "32GE Optima CT 540" (Netherlands, 2020). Chemical-toxicological studies were performed on a "Waters XEVO TQ-S micra" mass spectrometer (USA, 2020) using liquid chromatography.

The obtained photograms of forensic objects were detailed using the "RadiAnt DICOM Viewer 2023.1" software package. All photos were processed on a personal computer using the "Photo Frame Studio 3.0" software.

The crime scene inspection was conducted together with law enforcement officers, who involved the authors of this publication as veterinary specialists. Photogrammetry was performed using a scale ruler designed for forensic investigation photography. Statistical analysis of the obtained data was carried out by calculating the percentage ratio of expert cases. For the first time in Ukraine, two concepts of clinical logic have been introduced into the practice of forensic veterinary examinations of animal corpses to prevent overdiagnosis while remaining within the bounds of evidence-based veterinary medicine.

Results

When an animal dies in a veterinary clinic without an established clinical diagnosis or under unclear circumstances, it often becomes necessary to determine the cause of death. This helps to rule out errors in diagnosis

and treatment protocols, and in some cases, even to investigate the possibility of a violent cause of death. The following are the main reasons that trigger the need for forensic examinations of animal corpses: lack of proper communication between the pet owner and the veterinarian; the veterinarian's uncertainty during consultations, leaving the owner with doubts about the professional's competence; the owner's hyperdiagnostic approach to the illness and the veterinarian's polypharmacy approach, leading to unfavorable outcomes for the animal; sudden death or the absence of an obvious cause of death and a justified clinical diagnosis.

Logical challenges often arise when drafting an expert conclusion about the cause of death, particularly if death occurred after some time following treatment, due to fatal complications. For instance, after a certain period following the extirpation of a dog's internal reproductive organs, subcutaneous phlegmon developed due to contamination of the postoperative wound with anaerobic bacteria, leading to systemic inflammation and death. In such cases, to formulate a well-grounded conclusion about the cause of death, it is essential to compare the pathogenesis with thanatogenesis, understanding the death mechanism in their causal relationships to establish a direct/indirect link between iatrogenic influence and the animal's death.

Depending on the main etiopathogenetic factor of sudden animal death, two primary thanatogenetic scenarios can be distinguished: "acute cardiac" and non-cardiac. During the observation period, "acute cardiac death" caused by complications of chronic coronary insufficiency, such as fibrosis, intramural myocardial ischemia, hemopericardium, etc., was diagnosed in 10% of examined animal corpses aged over 8 years without a history of heart disease. It is important to note that a forensic veterinary diagnosis of cardiac death is a diagnosis of exclusion, established only when there are no other injuries or pathologies with independent thanatogenetic significance or other competing causes, as "acute" chest injuries can trigger reflex asystole, ventricular fibrillation, and fatal myocardial paralysis.

It is worth emphasizing that the foundation of any phenotype of organic cardiomyopathy is age-related heart involution during the animal's ontogeny, including macro-morphologically expressed heart muscle dystrophy. In cases of death in cats with a history of myocardial hypertrophy, the most common location of ischemia was the left ventricular wall (Fig. 1a).

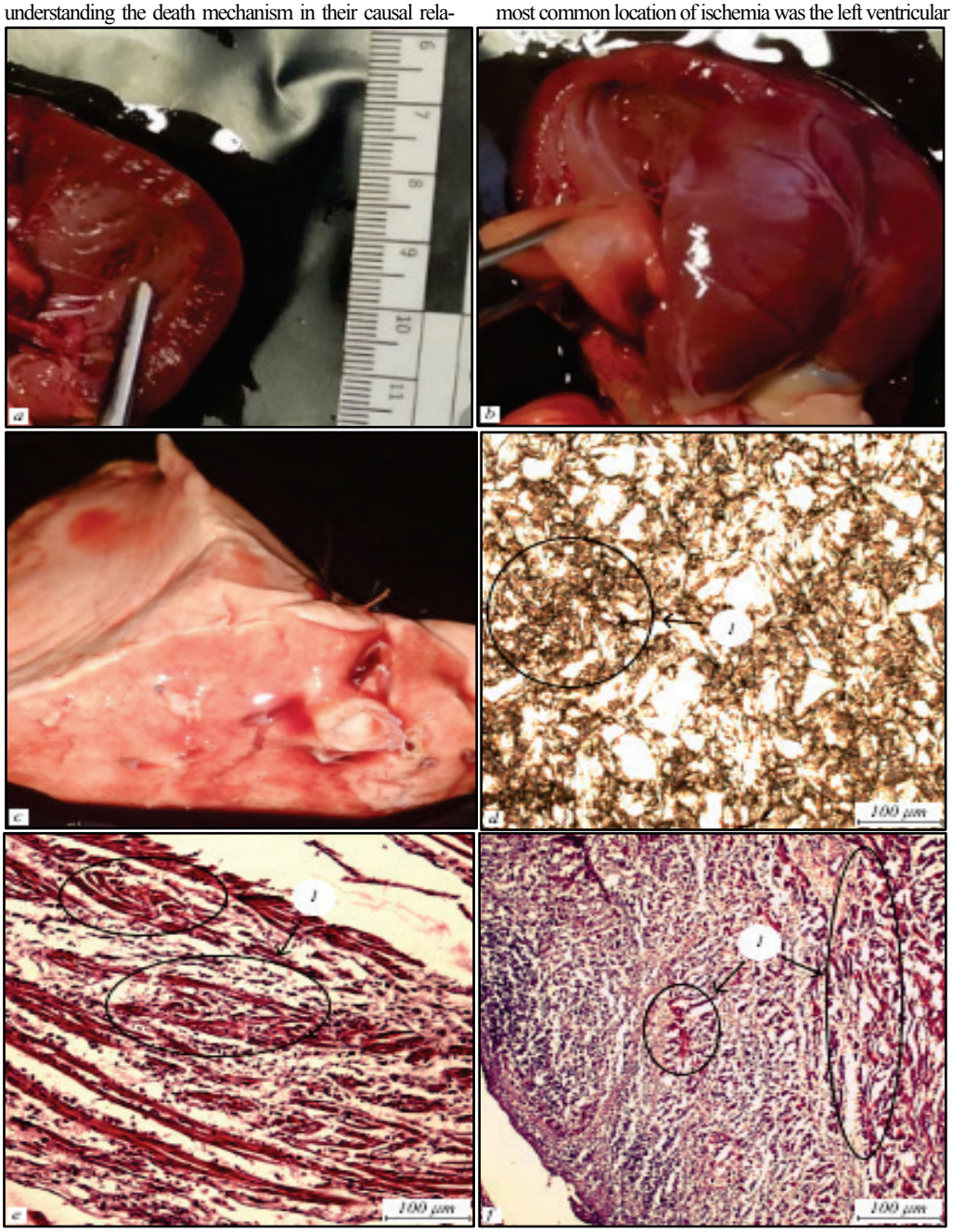

Fig. 1. Morphological changes in the organs of the thoracic cavity of a cat cadaver: *a* – fragment of the heart with intramural ischemia of the left ventricle; *b* – fragment of the heart with a white thromboembolus in the lumen of the pulmonary artery; *c* – fragment of the lung showing anemia in the case of cardiogenic edema; *d* – histotopogram of the heart, lipoid dystrophy (*1*), Sudan black B stain; *e* – histotopogram of the heart, loci of muscle fiber deformation (*1*), hematoxylin and eosin stain; *f* – histotopogram of the heart, hemocapillary erythrocyte stasis (*1*), hematoxylin and eosin stain

Visually, these areas appear dirty-gray and round, sometimes surrounded by pinpoint hemorrhages, with the myocardium either soft or firm to the touch. Due to myocardial hypertrophy, the left ventricular lumen narrows, physiologically leading to thromboembolism of large-caliber vessels, which is the primary cause of death, while the white color of the embolus indicates a prolonged agonal period (Fig. 1b). It should be noted that the firm consistency and adhesion of the embolus to the pulmonary artery trunk wall proves its premortem morphogenesis.

Although the lungs in these cases appear anemic (Fig. 1c), pink froth freely flows from the terminal bronchioles, indicating cardiogenic pulmonary edema as the definitive cause of death. Sutton's rule applies here, as confirmation of sufficient morphological changes in the myocardium leading to pulmonary edema suggests the fatal complications have a cardiogenic etiology.

In cases of excessive pressure overload in any chamber due to dilated cardiomyopathy with underlying myomalacia, wall rupture may occur, followed by cardiac tamponade due to hemopericardium.

Histological studies of cardiomyocytes in areas of lipoid myocardial dystrophy (Fig. 1d) confirmed the loss of striations and wavy fragmentation, corresponding to local contracture microdeformation of myocardial muscle fibers (Fig. 1e). This likely led to transient deterioration in vascularization of the left ventricular endocardium and subendocardial cardiomyocytes.

In ischemic myocardial regions, hemocapillary hyperemia, erythrocyte stasis, vascular spasm, and plasma imbibition of the microcirculatory vessels were observed (Fig. 1f). Together, these patterns indicate ischemia progression, creating conditions for further necrotization. In the left ventricle, focal thickening, sagging, and fragmentation of the endocardial folds were also noted, corresponding clinically to the morphological and functional manifestations of valvular heart failure (endocardiosis) in animals.

Therefore, the forensic conclusion in the case of cardiac death in a cat, based on the thanatogenetic principle, can be formulated as follows. The initial cause of death: hypertrophic myocardial dystrophy, thromboembolism, left ventricular endocardiosis, corresponding to acute myocardial hemodynamic disorders. The definitive (immediate) cause of death: transmural ischemia of the left ventricular wall with a pronounced demarcation line, myocardial paralysis, interstitial pulmonary edema, physiologically corresponding to chronic myocardial hypoxia and cerebral anoxia.

Undoubtedly, urgent conditions require the hospitalization of animals in veterinary clinics and the administration of appropriate syndromic therapy. During this period, the clinical status dynamically changes, progressively worsening, which affects the final pathomorphological picture leading to a fatal outcome. Often, pet owners bring animals to the hospital in a syncopal or shock state, exhibiting signs of clinical death. These conditions, especially when there is no time to gather anamnesis vitae et morbi, require veterinarians to make an emergency assessment of the priority level of assistance/resuscitation based on a combination of parameters of status praesens objectivus, such as blood pressure and blood oxygen saturation, due to the high probability of a negative prognosis.

Clinical veterinary practice confirms that critical conditions in animals can quickly lead to death due to the decompensation of a wide range of syndromes, monocausal pathologies, and their comorbidities. These include anaphylactoid reactions, profuse hemorrhage, chronic cardiomyopathy (including hypertrophic, dilated, and restrictive phenotypes), thromboembolism, reflex myocardial paralysis, vasomotor collapse, hypo- and hypervolemia, respiratory distress syndrome (including pulmonary edema and alveolar emphysema), encephalopathy and myelopathy of various origins, syncope, dysuria (due to oliguria or acute urinary retention), dystocia, passive hyperthermia, active pyrexemia, prolonged hypothermia, persistent arterial hypo- and hypertension, hypoxia (hypoxemia) of various etiologies, hematemesis and/or profuse diarrhea, prolapse of internal organs through hernial openings, dyspnea, tonic-clonic seizures, epileptic status, acute pain, shock of various etiologies, acute gastric dilation syndrome, intoxication (nutritional, medicinal), severe injuries, paraneoplastic syndrome, hypo- and hyperkalemia of various etiologies, congenital or acquired coagulopathy, systemic inflammatory response, pathological anastomoses, certain endocrinopathies (diabetic ketoacidosis, Cushing's/ Addison's syndrome, hyperthyroidism), acute kidney injury, and toxic liver dystrophy.

Empirical evidence shows that the basis of fatal complications in critical conditions is shock, which arises due to total dysfunction of the hemocirculatory system. However, the monocausal thanatogenetic significance of shock is debatable, as shock is always a consequence or background condition. In our view, shock's critical state is determined by three essential components: hemodynamic disorders, hypoxia, and hemostatic dysfunction. We believe this pathophysiological triad triggers a cascade of reactions leading to multiple organ failure, which, in turn, potentiates a fatal outcome.

Therefore, a gnoseological analysis of the interrelationship between structural changes and organ dysfunction in the pre-mortem period allows us to identify informative links, visualize the sequence of cause-and-effect relationships, and predict the occurrence of "brain death" in the animal.

It is well known that systemic coagulopathy is realized in the body through disseminated intravascular coagulation. It is now undisputed that this response occurs de novo as a complication of various syndromes, such as polytrauma, destructive pancreatitis, chemical and thermal burns, sepsis, systemic inflammation, etc. The syndrome develops as a fatal complication of an underlying pathology due to the decompensation of the hemostatic system, initiated and accelerated by various factors. It is important to note that the onset of DIC in systemic inflammatory response syndrome has a high probability, as endothelial damage in the microcirculatory system activates intravascular coagulation. Consequently, a microcirculatory block occurs, leading to hypoxemic multi-organ failure.

Our research has shown that animal death in cases of systemic coagulopathy occurs directly due to refractory hypovolemic shock caused by blood loss. This is detected during routine radiological (Fig. 2a) and ultrasonographic (Fig. 2b) examinations of the cadaver and later confirmed by laboratory tests following fine-needle aspiration biopsy. During the ultrasonographic screening of a cat cadaver, free fluid was found between the liver lobes (Fig. 2c), and in a dog cadaver, around the left kidney (Fig. 2d).

We assert that the development of disseminated intravascular coagulation drives a tendency towards functional decompensation and, as a result, establishes a negative prognostic outlook. Clearly, due to prolonged ischemia and tissue hypoperfusion, the accumulation of toxic metabolites leads to uremic syndrome and regional organ disorganization, which predominantly worsens the clinical status of the animal and results in the terminal lethal triad: acidosis, hypothermia, and hypocoagulation. It is known that the most common cause of metabolic acidosis is lactic acidosis, which is induced by reduced tissue perfusion, leading to anaerobic metabolism. Therefore, the increase in lactate levels observed in critically ill animals indicates the development of transorgan hypoxia. Furthermore, with prolonged disturbances in hemomicrocirculation, specific organs, considered "shock organs," become the primary targets of damage. Success in addressing these cases is achievable only through a detailed analysis of the fundamental propaedeutics: the pathophysiological processes and pathomorphological phenomena underlying the development of multivisceral trigger lesions.

During autopsy examinations of animal corpses, we observed that, in addition to the specific signs of the underlying pathology, there was a consistent pattern of pathological phenomena in target organs (Table 1). The data in Table 1 indicate that in all expert cases, the dominant trigger organs exhibiting macroscopic structural changes are the pancreas, kidneys, stomach and intestines (along with the mesentery).

Table 1

Correlation of structural changes in target organs of cat (n = 20) and dog (n = 30) cadavers with disseminated coagulopathy

Target organ	The pathological changes ratio, %	
	macrostructural	microstructural
Brain	6	10
Heart	8	12
Lungs	70	76
Pancreas	92	94
Stomach	90	96
Intestine	94	98
Liver	82	86
Spleen	56	58
Adrenal glands	86	88
Kidneys	92	98

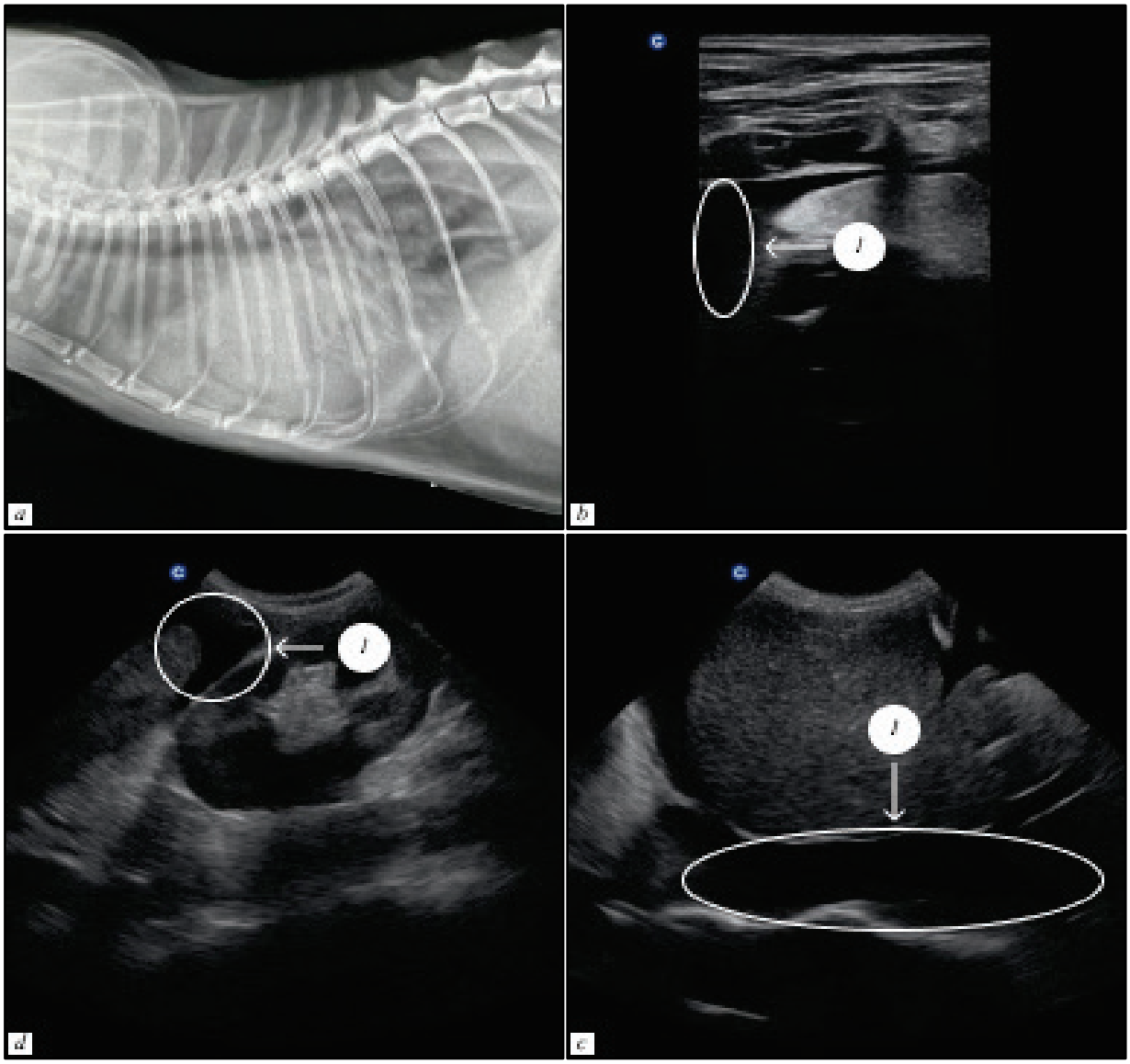

Fig. 2. Instrumental and visual signs of internal bleeding: *a* – X-ray of a dog's chest in RL projection, showing signs of free fluid in the thoracic cavity; *b* – T-Fast of a cat cadaver, ultrasonographic signs of free fluid in the mediastinum (*1*); *c* – A-Fast of a cat cadaver, ultrasonographic signs of free fluid between the liver lobes (*1*); *d* – A-Fast of a dog cadaver, ultrasonographic signs of free fluid around the kidney (*1*)

To clarify the logic of a forensic veterinary expert and the algorithm for conducting expert examinations of animal corpses, let's present some cases of "empirically complex" scenarios. From registration records, it is known that the dog was free-roaming the day before its death. The dog's body was found by police officers in an unnatural position and posture (Fig. 3a), indicating that the death occurred under unclear circumstances. No external injuries were found, but there were notable signs such as free-flowing non-clotted blood from the medial corner of the left eye (Fig. 3b) and prominent blood vessels in the hypodermis (Fig. 3c).

During the autopsy, the following forensic veterinary diagnoses were established: hemoperitoneum, macroscopic hematuria, and bladder hematoma (Fig. 3d). Ultrasonographic examination of the status localis of the injury revealed detachment of the bladder's mucosal layer from the underlying tissues (Fig. 4a), which was further confirmed histologically: perivascular edema and erythrodiapedesis in the bladder wall (Fig. 4b).

So, such a hematoma could have been caused by a blunt object strike in the perineal area. However, according to Sutton's rule, the hemostasis tests performed on the animal's corpse show a prolonged prothrombin time, which does not exclude external pharmaceutical stimulation and, therefore, requires chemical-toxicological verification. This also highlights the potential for diagnostic errors during a superficial analysis of the case. Considering this, the expert's final conclusion should indicate the initial cause of death in a probable form (trauma from a blunt object in the perineal area) and the immediate cause – hemoperitoneum resulting from systemic coagulopathy. The key links in the process of death can be illustrated as a fatal chain: hemostasopathy – hypocoagulation – hypotension – hypothermia – hypoxia – early decompensated shock stage – terminal shock stage – animal death, confirmed by relevant physical parameters.

It is essential to emphasize that acute kidney injury was observed in all animals, clinically characterized by rapid oliguria or anuria along with a sharp rise in renal biochemical markers, manifesting as renal uremic syndrome. Most likely, the decrease in blood filtration rate in the glomeruli is the starting pathological link of "shock kidney." Regardless of the primary factor, kidney injuries were localized. It is known that during shock, glomerular vascularization slows down and is carried out through juxtamedullary shunting, visually confirmed on a sagittal section of the organ (Fig. 4c). It can now be stated that the pathomorphogenesis of disseminated intravascular coagulation at the tissue level is realized through the formation of fibrin microclots and sludges of blood elements, which coagulate in the renal capillaries, causing blockages due to thrombosis and local hemoconcentration (Fig. 4d).

It is likely that widespread glomerular thrombosis determines the development of cortical tubular necrosis and can be considered a primary morphological pattern of "shock kidney," directly indicating the need for microscopic examination of additional histological slides to identify patterns of metastatic septic processes, even in isolated foci. Despite the fact

that the histomorphological architecture of the urinary tubules remained within functional norms, hemodynamic disorders were detected, manifested as dilation and excessive blood filling of the capillaries, especially in the subcapsular zone of the kidneys. At the same time, glomerular capillaries were dilated and filled with formed blood elements, causing renal corpuscles to increase in size. Our observations show that in animals with histologically confirmed generalized inflammatory reactions in the form of septicemia, pathological changes in renal vessels were characterized by erythrocyte sludging, microdiapedetic hemorrhages, and perivascular edema, which allow these patterns to be used for assessing the critical state from a diagnostic-prognostic perspective of the likelihood of generalized infectious-septic processes.

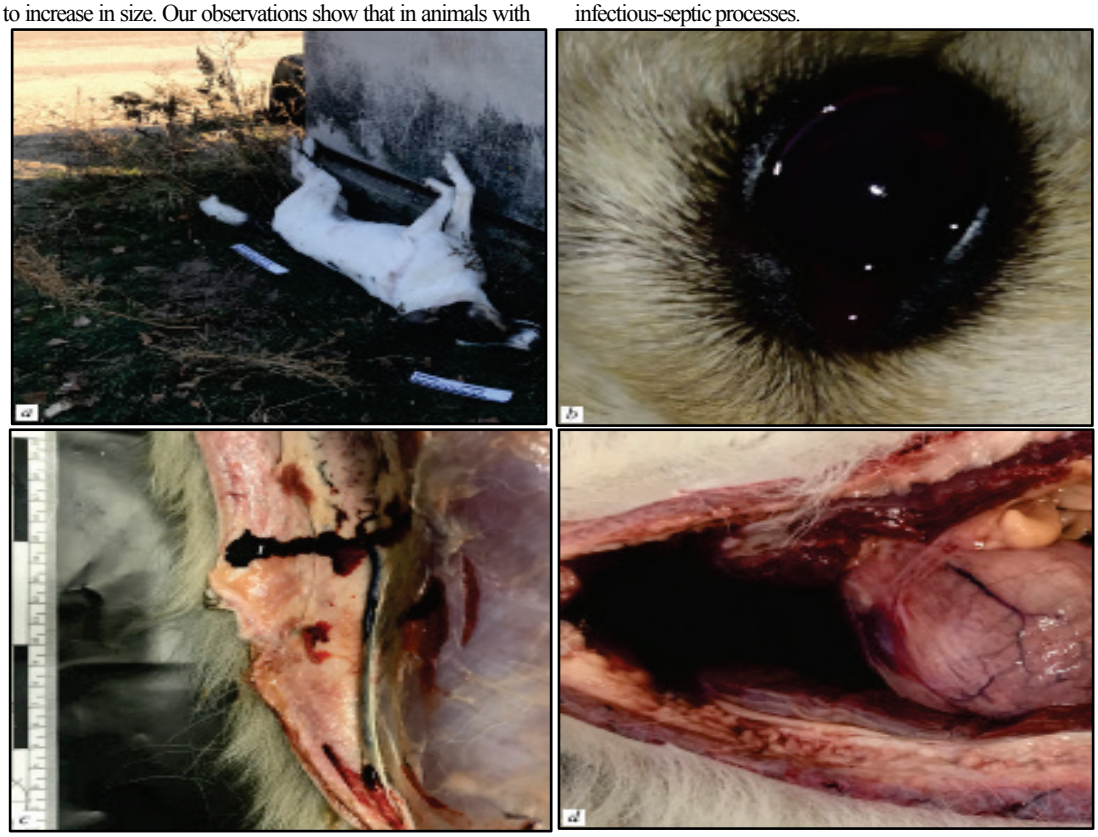

Fig. 3. Forensic veterinary autopsy of a dog's cadaver: *a* – inspection of the cadaver at the site of discovery; *b* – blood leakage from the medial corner of the left eye; *c* – pronounced leakage of non-clotted blood from hypodermal vessels; *d* – fragment of the abdominal cavity, bladder hematoma, hemoperitoneum

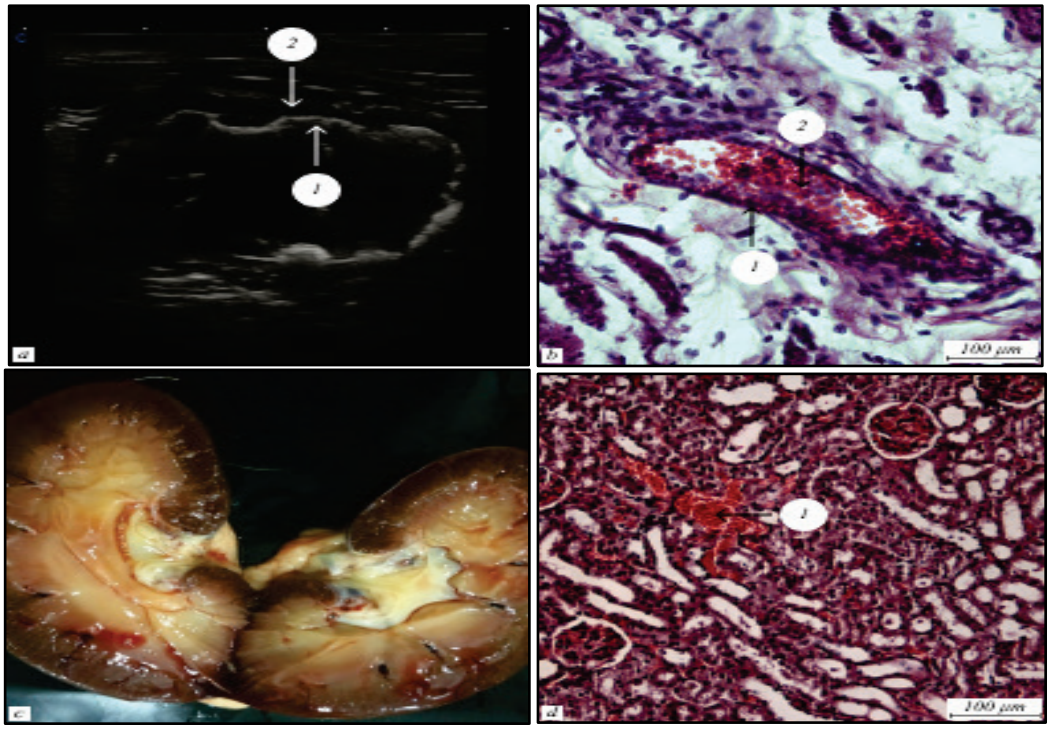

Fig. 4. Morphological changes in the bladder and kidneys of a dog's cadaver: *a* – ultrasonographic examination of the bladder, mucosal layer (*1*), desquamation (*2*); *b* – histotopogram of the bladder wall, perivascular edema (*1*), erythrodiapedesis (*2*), hematoxylin and eosin stain; *c* – kidneys on sagittal section, diapedesic hemorrhages in the interstitial layer; *d* – histotopogram of the kidney, glomerular thrombosis (*1*), hematoxylin and eosin stain

The morphological patterns of sepsis are not limited to classical, macroscopically evident exudative foci, observed in animals with clinically confirmed sepsis, such as serous-fibrinous polyserositis, but also during thorough microscopic examination of focal lesions in the renal microcirculatory vessels, providing informative morphological criteria for the likelihood of sepsis. Accordingly, the presence of sepsis in animals without morphological signs of renal microcirculation damage is doubtful, even with a reliable clinical-laboratory picture.

Thus, simultaneous multiorgan disorganization in the form of a combination with prerenal uremia can be considered one of the signs of the clinical manifestation of sepsis, which should be taken into account when formulating the expert's final conclusion.

When septic and toxic processes are involved, circulatory shock ensues, the basis of which is changes in the peripheral hemocirculation system. The disruption of microcirculation during the shock state of the animal is the direct cause of the premortal status. It is emphasized that the underlying factor in fatal disseminated intravascular coagulation in animals is always intravascular coagulation, with its direct (microthrombi) and indirect (hemorrhages and necroses) manifestations.

In advance, it is detailed that during the examination of animal corpses, hemorrhages into body cavities, interstitial lung edema (sometimes with foci of hemorrhagic pneumonitis), cortical necrosis and edema of the kidneys, glomerular capillary thrombosis, acute hemorrhagic glomerulonephritis, interstitial adrenal edema, ischemic and hemorrhagic infarctions of the adrenal cortex, liver dystrophy and congestive hyperemia, acute pancreatitis (with sepsis – pancreatic destruction), erosive-ulcerative gastroenterocolitis, venous hyperemia, and thrombosis of mesenteric vessels were observed. These patterns were discovered de novo, regardless of the primary cause.

A regional organ manifestation of disseminated intravascular coagulation is the "shock lung" syndrome, which we view as evidence of a microcirculatory crisis in the pulmonary circulation system.

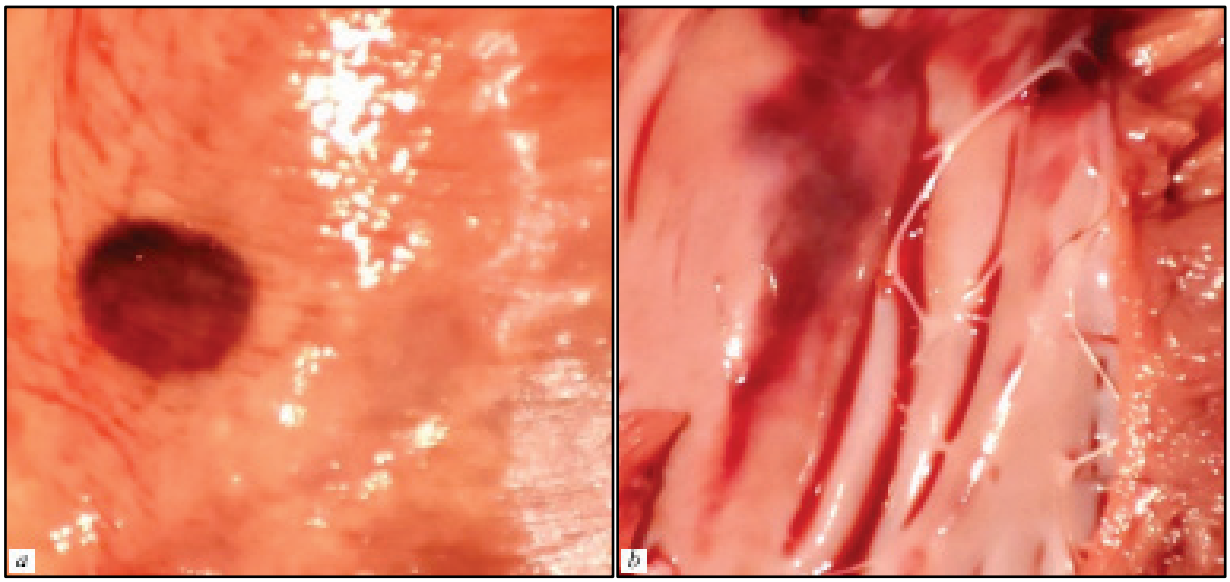

Fig. 5. Focal hemorrhages in a dog's cadaver: *a* – lung fragment, hemorrhagic pneumonitis; *b* – endocardium fragment, Minakov's spots

This condition is caused by a distributive defect in capillary blood flow within the pulmonary acini, which, along with microthrombi obstruction, leads to oxygen hypoperfusion. This, in turn, is the direct cause of multivisceral hypoxemic failure. Pathological changes in "shock lung" are non-specific and result from hemocapillary shunting in the microcirculatory system into the "third space." Interstitial lung edema, along with the aforementioned factors, is evidently also due to increased drainage function during the onset of metabolic acidosis, which always accompanies hemomicrocirculation blockage under hypoxemic conditions. Therefore, disseminated intravascular coagulation in the lungs is ultimately confirmed by the detection of microthrombi, which macroscopically manifest as round-shaped subpleural hemorrhages (Fig. 5a). Externally, the lungs appear anemic. As for the strip-shaped subendocardial hemorrhages in the left ventricle (Minakov's sign), they are a constant, reliable pattern associated with death from acute blood loss (Fig. 5b).

Hemodynamically-induced structural changes in the gastrointestinal tract wall are explained by microthrombosis of the vessels and their destabilization due to increasing hyperlactatemia. This results in erosions of the parietal structures, accompanied by hematemesis, melena, or profuse diarrhea. It is logical to assume that subsequent dehydration leads to the loss of sodium and potassium cations, causing transient arrhythmias and increasing chloride anion levels. This slows the overall capillary perfusion rate as fluids shift into "third spaces," exacerbating metabolic hyperlactatemia. Reduced capillary perfusion in the kidneys leads to circulatory hypoxemia and tissue hypoxia.

The targeted "shock organ" for coagulopathy syndrome is the duodenum, which, in our opinion, plays a key role in the development of circulatory shock through mesenteric artery microthrombosis (Fig. 6a). Ulcerative enteritis develops in the small intestine (Fig. 6b), followed by necrosis of the intestinal wall. Lysosomal enzymes likely play a dominant role in this process, as their activation due to cytolysis contributes to hemorrhagic pangastritis, erosion (Fig. 6c), and focal detachment of the mucosa, leading to profuse melena. Histomorphological examination of duodenal samples from dog cadavers with catarrhal-hemorrhagic duodenitis revealed that the lamina propria of the mucosa between the gland ducts and the submucosa was intensely infiltrated by plasma cells and permeated by small-diameter blood elements, which locally form conglomerates, indicating the progression of alterative-exudative processes. The glandular secretion was markedly increased. Externally, the mucosa was covered by a single-layer columnar glandular epithelium with foci of desquamated cells in the intestinal lumen and in the area of the duodenal glands (Fig. 6d).

It has been proven that the pathomorphological changes in the pancreas are generally typical regardless of the etiological factor, and the progression of acute pancreatitis (Fig. 7a), serous-hemorrhagic pancreatitis (Fig. 7b), and pancreatic necrosis (Fig. 7c) stages depends only on the duration of the damaging impact on the organ. For example, in cases of ultrasonographically confirmed acute pancreatitis, hypercoagulation with prolonged prothrombin time was registered in animals within a day if adequate stabilization was not provided. The detected disorders not only persisted but also progressed, reaching a peak by the end of the second day of observation.

Regarding the histomorphology of pancreatic acini in cat cadavers, it was found that the glandular clusters, in a state of pronounced secretion, were enlarged, and their lumen was filled with a large number of tightly arranged aggregates of blood elements (Fig. 6e), formed due to erythroleukostasis, and were paralyzed and dilated (Fig. 7d). The identified pathohistological picture of the pancreas clinically corresponds to the exudative stage of acute pancreatitis.

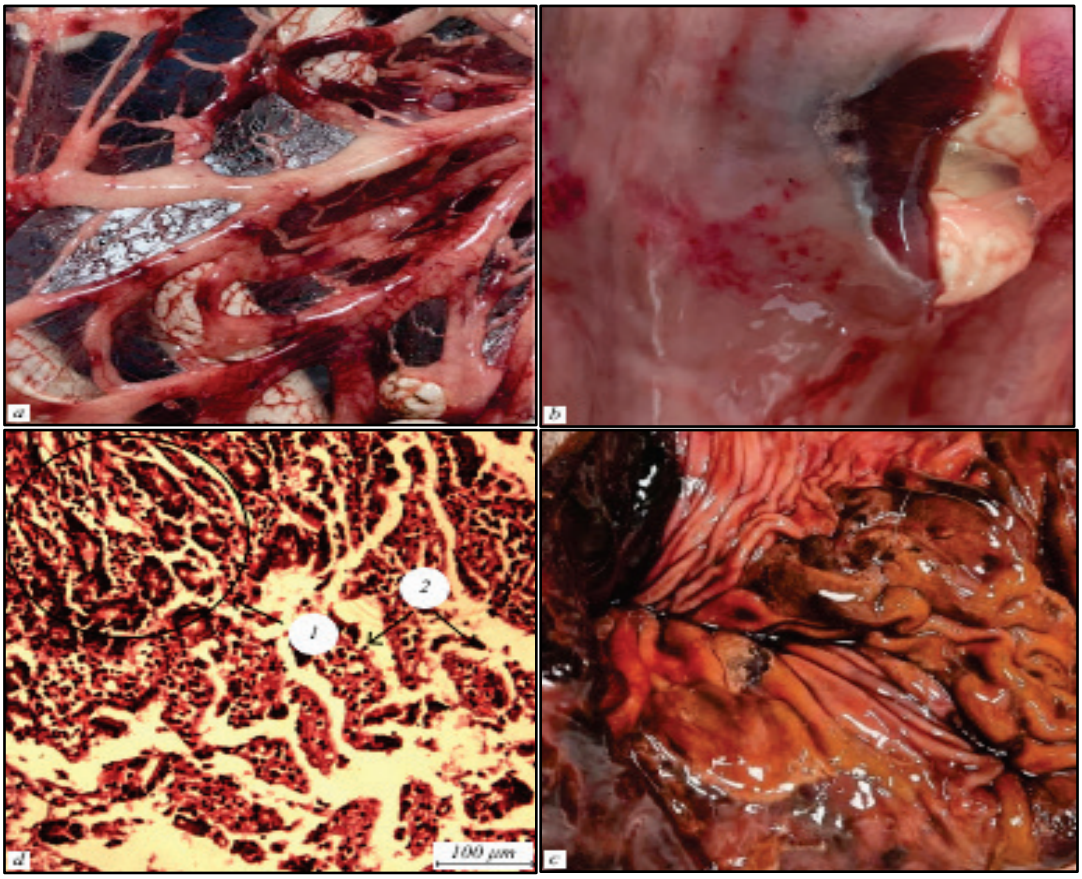

Fig. 6. Morphological changes in the gastrointestinal tract of animal cadavers: *a* – fragment of the mesentery from a dog cadaver, multifocal thrombosis; *b* – fragment of the duodenum from a cat cadaver, perforating ulcer; *c* – fragment of the stomach mucosa from a dog cadaver, erosive-hemorrhagic pangastritis; *d* – histotopogram of the duodenal mucosa from a dog cadaver, marked blood cell infiltration (*1*), epithelial desquamation (*2*), hematoxylin and eosin stain

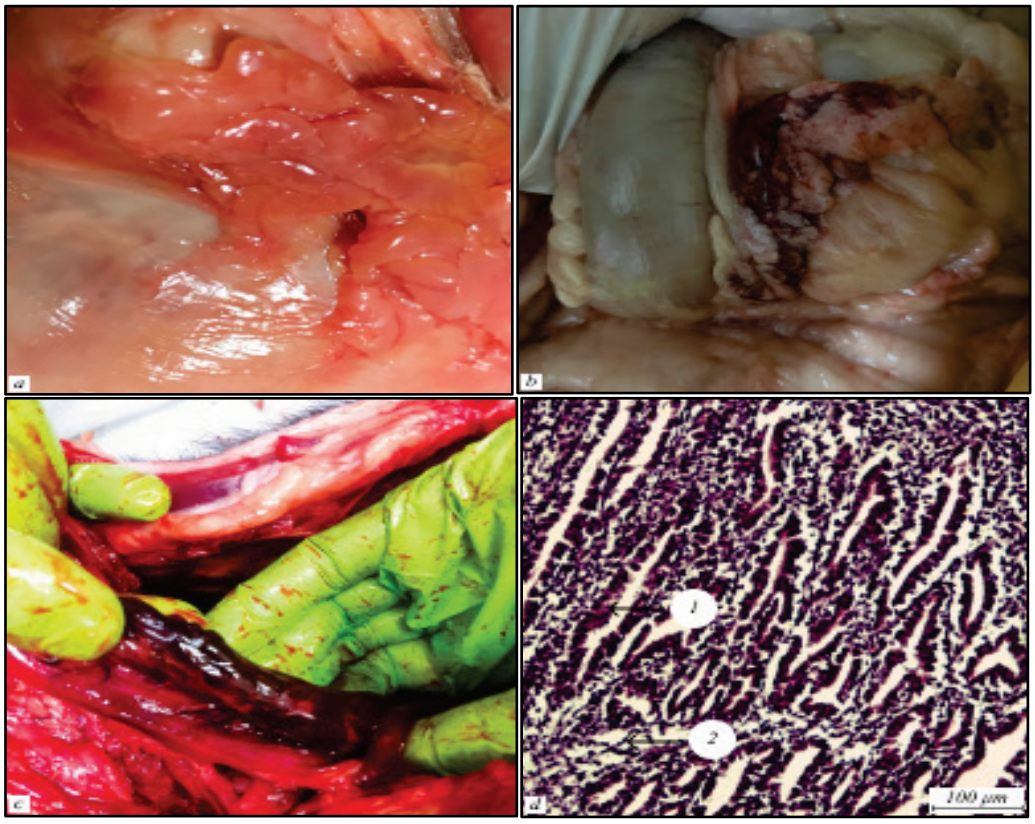

Fig. 7. Morphological changes in the pancreas of animal cadavers: *a* – pancreas of a cat cadaver, acute destructive pancreatitis; *b* – pancreas of a cat cadaver, subtotal chronic pancreatitis with fibro-hemorrhagic foci; *c* – pancreas of a dog cadaver, total sequestrating pancreatic necrosis; *d* – histotopogram of the pancreas of a cat cadaver, erythrocyte stasis (*1*), dilated ducts of duodenal glands (*2*), hematoxylin and eosin stain

It is known that the thrombosis of acini in the microcirculatory bed and the development of ductal hypertension lead to local and, over time, generalized activation of proteolytic enzymes, which causes de novo fibrinolysis and results in "mucosal shock" in the form of hematochezia or hematemesis. The progression of pancreatic lesions is accompanied by sequestration, parapancreatic fluid formations, and diffuse omentitis, which indirectly contribute to systemic inflammatory response syndrome. This clearly supports the hypothesis that not only endogenous toxins but also general hypoxia with hemocoagulation changes play a crucial role in the development of systemic inflammatory response syndrome.

It has been established that the onset of disseminated intravascular coagulation in conjunction with liver alteration occurs independently of the etiopathogenesis of hepatopathy. The most common pathological conditions, which are macroscopically detected during autopsy of animal cadavers, include a wide range of primary and underlying liver lesions: marked hyperemia against the background of lymphoplasmacytic hepatitis (Fig. 8a), hepatobiliary cirrhosis with mucocele (Fig. 8b), and toxic hepatodystrophy associated with amyloidosis (Fig. 8c). These conditions are consistently linked to impaired synthesis of albumins and globulins, particularly fibrinogen. Furthermore, the decrease in albumin levels in the blood of animals due to protein-synthesizing disorders of the liver serves as a predictor of plasma transudation outside the vascular system, which is also important to consider in the final conclusions.

When correlating the nature of pathological changes in the liver at the tissue level, the macroscopic findings of the forensic veterinary diagnosis were confirmed. Histomorphological studies of liver samples from cats with toxic hepatopathy revealed signs of hemodynamic disturbances: the sinusoidal spaces were dilated, and a significant number of venous vessels were filled with blood elements (Fig. 8d), while the overall trabecular structure of the organ was not well defined. Dystrophic changes in hepatocytes were characterized by karyorrhexis and variability in the sizes of dark nuclei, with cloudy cytoplasm containing fine granularity. In cases of feline lipidosis, hepatocytes located mainly in the central acini exhibited a ring-like shape with a predominant displacement of the nucleus to the periphery. The cytoplasm was filled with fat droplets, which optically corresponded to vacuolated spaces.

It has been established that ischemia of the adrenal cortex, caused by vascular thrombosis, stimulates the development of a sympatho-adrenal crisis, which clinically manifests as acute adrenal insufficiency, followed by coma and rapid death.

Based on a comparison of anamnesis, clinical, and post-mortem data for a sufficient number of animals treated in veterinary medical facilities, the authors concluded that regardless of the reason for an animal's admission and the etiology of the disease, typical complications such as organ failure and the development of critical conditions with unfavorable prognoses arise during pathogenesis.

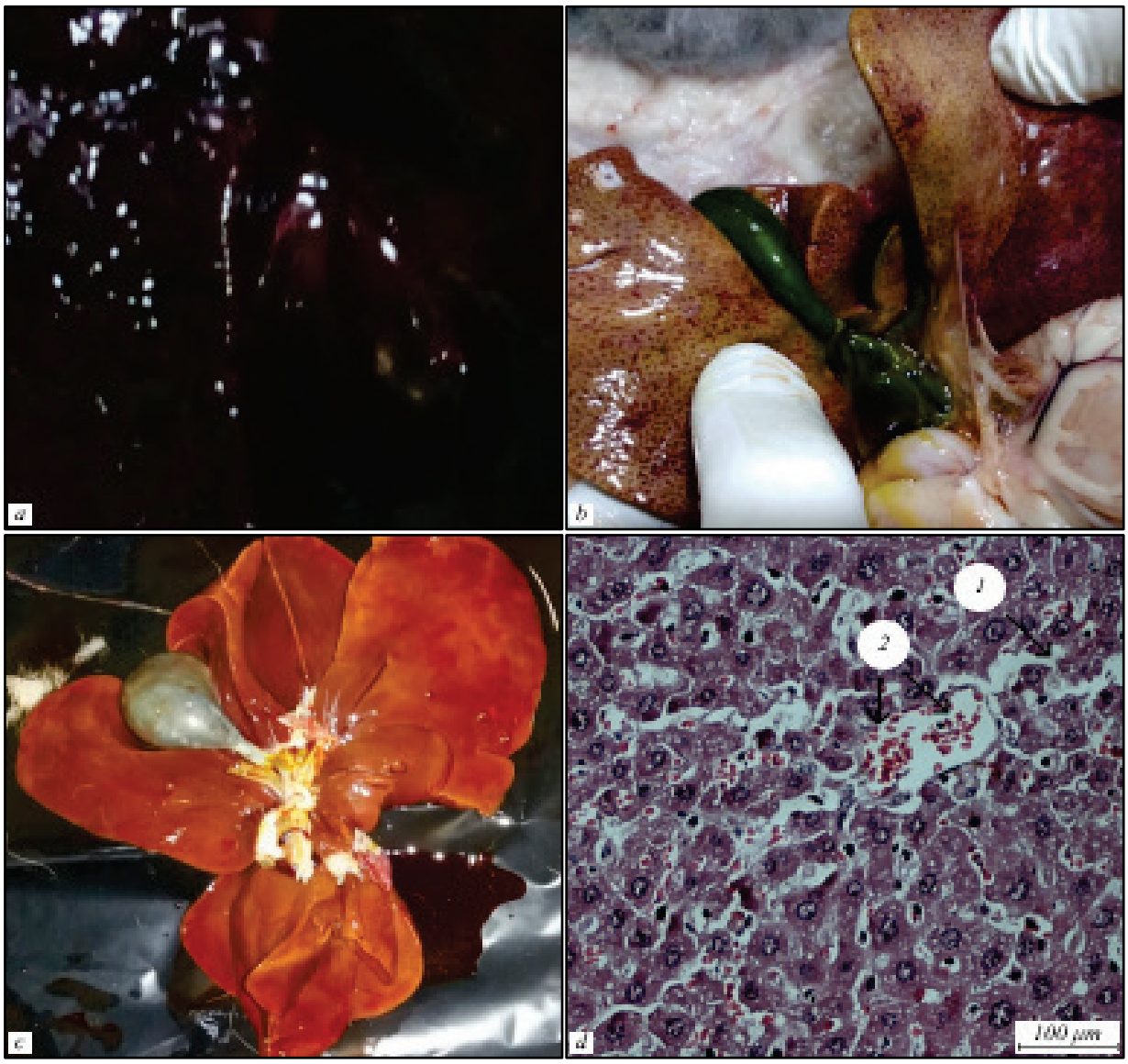

Fig. 8. Morphological changes in the liver of animal cadavers: *a* – fragment of the liver from a dog cadaver, marked alterative-exudative reaction; *b* – fragment of the liver from a cat cadaver, hepatobiliary cirrhosis in the area of the gallbladder bed; *c* – liver from a dog cadaver, amyloid dystrophy; *d* – histotopogram of the liver from a cat cadaver, dilated sinusoidal ducts (*1*), erythrocyte stasis (*2*), hematoxylin and eosin stain

In a retrospective analysis of clinical and morphological case summaries, the authors identified a thanatogenetic focus within hospital mortality statistics for animals. For example, 22% of animals aged 6 months to 6 years died from infectious and parasitic diseases, compared to only 2% of older animals (aged 5–10 years) in which tissue metaplasia with morphological manifestations of paraneoplastic syndrome was recorded. On the one hand, the high mortality rate among neonates with contagious diseases is likely due to irreversible hemodynamic disturbances in vital organs and intoxication with acidic metabolic by-products, leading to cerebral anoxia through venous hyperemia and brain swelling. On the other hand, similar phenomena may predict antenatal hypoxia during gestation due to uterine dystocia.

In forensic veterinary examinations of 15% of animal cadavers, deaths occurred at different stages of veterinary care, including recurrent postoperative complications, iatrogenic overdoses due to incorrect risk/benefit evaluations, nosocomial infections, and others. There was a necessary direct connection between the completeness of diagnostics, the scope of treatment, and the death of the animal. However, it would be fundamentally incorrect to assert that death occurred due to a lack or defect in diagnostic or therapeutic veterinary procedures. For instance, concluding that "the delayed surgical intervention for gastric dilation in a Central Asian Shepherd was directly related to the cause of death" conflicts with the principles of evidence-based veterinary medicine. The reasoning would be flawed because the event (the surgery) did not occur, and thus the consequence (death) cannot be attributed to it. Therefore, the absence of timely intervention cannot be considered the cause of death since death results from a material cause, and timely treatment prevents the onset of death in emergency cases.

Mechanical injuries incompatible with life, such as polytrauma from a vehicle, severe anatomical damage to bones, crushed soft tissues, massive internal bleeding, traumatic amputation, etc., caused the death of 10% of animals of all age groups. Death due to pathological processes and conditions associated with decompensated hypoxia occurred in 7% of animals from different age groups.

The largest proportion of fatal outcomes, according to our data, was due to intoxication of various etiologies, accounting for 33% of all animal cadavers studied. This was most common in animals aged 3–4 and 7–8 years. Among these cases, exogenous pharmaceutical stimulation was a significant cause, which also led to hemodynamic disorders and hypoxia.

In cases of isoniazid poisoning in dogs or combinations with prokinetics, clinical evaluation was based on analyzed symptoms. Premortem history indicated that a few hours after free-roaming, the animals' behavior changed, leading to mental disturbances. Neurological deficits such as ataxia, apraxia, seizures, muscle hyperkinesia, nystagmus, paresthesia, hyperesthesia, tachypnea, paraplegia, and proprioceptive disorders were recorded. In the most severe cases, hematemesis was not observed. Death occurred within four to six hours after the onset of the first muscle hyperkinesia. Due to the variable clinical presentation, a definitive clinical diagnosis was difficult, but using Simpson's principle narrowed down the differential diagnosis to poisonings by substances like metaldehyde, ethylene glycol, ivermectin, ketoprofen, metronidazole, diphenhydramine, bromethalin, methylxanthine, and strychnine. Other conditions not related to toxicosis, such as idiopathic epilepsy, encephalopathies, some infectious diseases, hydrocephalus, brain tumors, hypoglycemia, and hyponatremia, must also be ruled out.

During external examinations of poisoned dogs, no injuries were found. However, in cases of pronounced neurological symptoms, small ecchymotic hemorrhages were sometimes observed in the subcutaneous tissue at sites of self-injury. The visible mucous membranes of the oral cavity and conjunctiva were mostly hyperemic.

Forensic autopsies of the dogs revealed distinct morphological findings. The primary cause of death (I) was distributive shock and cerebral anoxia. Secondary conditions (II) included generalized venous hyperemia, acute dilation of the right heart, and acute catarrhal-hemorrhagic gastroenteritis. Background conditions (III) included metabolic dystrophy of the liver and kidneys and chronic focal pancreatic fibrosis.

These findings are characteristic of "rapid death," and while the clinical picture and autopsy results are not pathognomonic for a specific type of poisoning, chemical verification is required. To establish the primary causal relationship between the detected pathological changes and the death of the dogs, chemical-toxicological analysis of gastrointestinal contents was conducted, identifying isoniazid as the poison.

In cases of brodifacoum poisoning in cats, the clinical picture before death was non-specific, including lethargy, anemic mucous membranes, macroscopic hematuria, hematochezia, hematemesis, epistaxis, petechial rashes, limb weakness, and syncope. Such a broad symptomatology does not point to a specific clinical diagnosis but, according to Sutton's rule, suggests the possibility of external pharmacological stimulation with fibrinolytics and angio-stabilizers.

Forensic autopsies of the cats identified hemoperitoneum, hemothorax, and alveolar pulmonary edema as primary causes of death (I), with additional findings (II) of acute hemorrhagic gastroenterocolitis, Minakov's spots, generalized anemia, and venous hyperemia in the kidneys, liver, and spleen. Background conditions (III) included unspecified hepatopathy, nephropathy, and acute pancreatitis.

The forensic diagnosis confirmed systemic coagulopathy, corroborated by prolonged activated partial thromboplastin time (< 40 sec.), prothrombin time (< 20 sec.), and critical thrombocytopenia. The coagulopathy hypothesis was supported by opposite signs of peripheral anemia and visceral venous hyperemia, indicative of distributive shock.

To confirm the causal relationship between pathological findings and the cats' deaths, chemical-toxicological analysis of gastrointestinal contents was conducted, identifying brodifacoum. Based on a combination of anamnesis, external and internal examination findings, and chemical-toxicological research, the forensic conclusion of violent death due to chemical trauma was substantiated.

In comparing the forensic veterinary diagnoses of acute isoniazid and brodifacoum poisonings, refractory hemodynamic disturbances (distributive shock) and subsequent fatal hypoxia – either cerebral anoxia or alveolar pulmonary edema – are the dominant factors in the thanatogenetic aspect.

Summarizing the findings, in cases of general hemostatic pathology in forensic veterinary autopsies, the possibility of violent death due to poisoning should always be considered during the determination of the primary cause of death.

In cases where there are no specific changes in the corpse, which may occur during sudden death or pronounced post-mortem transformation, the forensic veterinary expert, even after a thorough investigation, may not be able to determine the cause of death. It is advisable to carefully review the case materials: the circumstances of death, the clinical picture of the dying process, laboratory test results, and so on, and substantiate a probable cause of death in the conclusion. Such an explanation must be argued with established facts. Cases where it is impossible to determine the cause of death of an animal should be accompanied by a well-founded explanation. For example, expert cases with an unknown cause of sudden death in animals, with a formulated probable statement like "it is impossible to establish the cause of death, though the possibility of... cannot be excluded," accounted for only 1% of all expert studies. However, it should be noted immediately that the violent nature of the animals' deaths was excluded during the autopsy stage, as the identified morphological findings had an independent thanatogenetic role, and signs of bodily injury were absent. For example, during the internal examination of a cat's corpse, pyothorax was found (Fig. 9a), and death resulted from sepsis, or a phenomenon of mucogranulosis in the form of numerous cysts (Fig. 9b) filled with clear fluid, which together caused fatal compression of the animal's internal organs.

Decompensation of congenital malformations evidently caused the death of a cat in another expert case due to the atypical anatomical positioning of the gas-distended stomach located in the thoracic cavity, resulting from prolapse through the diaphragmatic hiatus and subsequent fatal displacement of thoracic organs (Fig. 9c). Similarly, the presence of a portocaval shunt in a dog cadaver (Fig. 9d) led to hepatic encephalopathy caused by the decompensation of metabolic pathways, specifically the blockade of deamination processes.

Establishing the primary cause of death in animals remains one of the most complex tasks for forensic veterinary experts. This process should ideally be approached in several logical stages. First, the examination of the animal cadaver must always be thorough and comprehensive, regardless of the apparent or seemingly obvious cause of death. In the next stage,

the forensic expert must envision the post-mortem morphological picture across various types, species, and categories of death. A retrospective analysis helps the expert work backwards – from the observed signs to the cause of death. Finally, all collected factual data must be comprehensively and rationally integrated without prioritizing certain methods or findings.

The number of specific patterns detected during the examination dictates the strength of the expert conclusion. It is important to note that non-pathognomonic signs, such as cyanosis of mucous membranes, venous congestion of internal organs, hypostatic spots, intestinal paralysis, pulmonary edema, and brain swelling, do not necessarily point to a specific cause of death. These signs, often observed during internal examinations, can result from various causes that occurred before death. Therefore, the thanatogenetic diagnostic process can be challenging due to the wide range of possible causes and symptoms with no clear indicators. In such cases, it is logical to construct a series of "expert hypotheses."

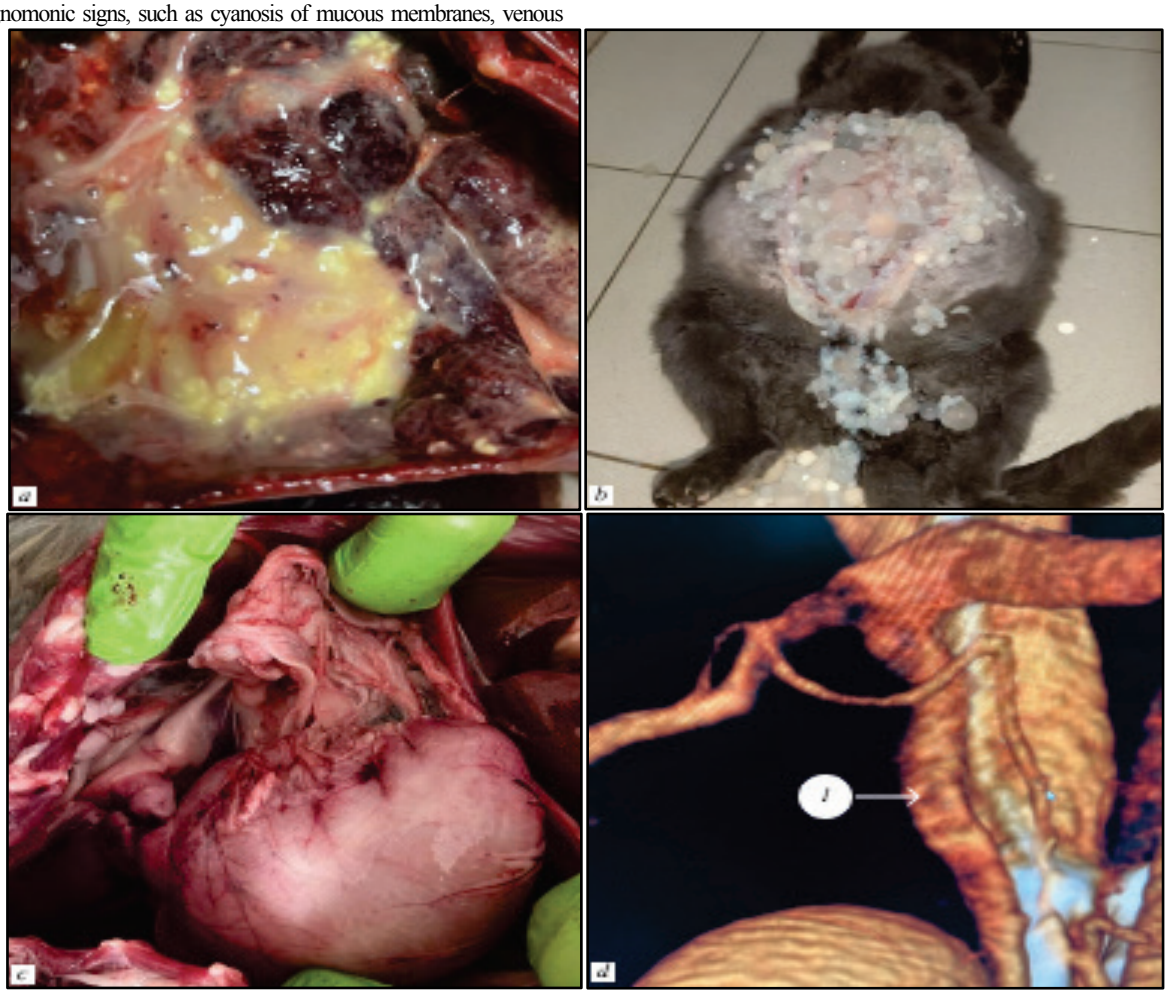

Fig. 9. Forensic cases of sudden animal death: *a* – accumulation of exudate in the thoracic cavity of a cat cadaver; *b* – abdominal cavity of a cat cadaver, signs of visceral muco-granulosis; *c* – thoracic cavity of a cat cadaver, anatomically atypical stomach location with gas stasis; *d* – radiographic tomogram of the abdominal cavity of a dog cadaver, portocaval shunt (*1*)

When encountering "competing" causes of death, it is advisable to apply a "thanatogenetic" approach during the forensic analysis, based on the principles of evidence-based veterinary medicine. This approach helps to identify the primary and most significant links in the process of dying, highlighting the initial cause of death and its fatal complication amidst many pathological phenomena. However, in practice, there is often no need to differentiate between the primary and direct causes of death unless explicitly requested of the forensic veterinary expert.

Discussion

The primary goal of forensic veterinary autopsy is to resolve a range of questions posed by investigative authorities, with the most important being the immediate cause of death. The forensic expert must determine the cause of death based solely on objective data: thorough analysis of case materials, forensic veterinary, and additional examinations. To establish legal liability for crimes against the health and life of animals, there must be a direct causal relationship between the action (or event) and the outcome. Therefore, the justification of the cause of death should lead to an unequivocal legal qualification of the act.

Under the definition of the primary cause of death in animals, it is proposed to consider a nosology, injury, or condition that initiates the sequence of thanatogenic processes leading to death from bodily harm or accidental events. Pathological changes caused by or resulting from the primary nosology and subsequently leading to life-incompatible disorders should be regarded as the immediate cause of death. However, it is important to note that dividing the causes of animal death into primary and immediate may not always help establish the objective truth in investigations involving crimes against animal health.

In certain cases, it may be appropriate to apply the "nosological" principle, whereby the cause of death is formulated as a nosological diagnosis. However, experience shows that an expert's conclusion based on the "nosological" principle often fails to reflect the main stages of thanatogenesis in an ontogenetic sequence. As a result, the forensic veterinary diagnosis may not represent objective reality, as it does not adhere to the morphological requirements of evidence-based veterinary medicine. Over-reliance on the "nosological" principle in forensic conclusions about the cause of death often leads to erroneous outcomes, emphasizing "background" pathologies (e.g., pulmonary, cardiac, or cerebral) over more immediate causes. It is essential to avoid interpreting "immediate" cause of death as merely the cessation of brain activity, as that only confirms clinical death rather than explaining the cause of death at an organismal level.

The complication of such expert cases stems from the fact that, during the dying process of an animal in critical condition, pathophysiological

compensatory mechanisms may produce comorbid morphological positions with equal thanatological informativeness. For example, pulmonary edema may be either cardiogenic or non-cardiogenic, depending on the primary cause of death. Therefore, identifying only the initial pathology as the cause of death may be insufficient. While death is ultimately caused by one factor, determining the immediate cause requires understanding what specifically should be considered the direct cause of death. Despite the limitations, the "nosological" approach can be valid if supported by research findings from the forensic examination.

In clinical veterinary practice, myocardial paralysis is often cited as the cause of sudden death under unexplained circumstances. In our view, except for cases of immediate bodily fragmentation, biological death is always preceded by the cessation of cardiac activity, which may not be correct from a forensic thanatogenetic perspective. From this standpoint, the cause of death cannot be reduced to the cessation of heart function alone, as this contradicts the basic principles of evidence-based veterinary medicine. Instead, it is important to specify heart-related causes of death, except for primary heart diseases that can independently cause sudden death.

Currently, no morphological criteria reliably confirm the occurrence of sudden death due to myocardial dystrophy in animals, unlike the well-documented cases in humans. For instance, Brugada-Terradellas et al. (2021) assert that dilated cardiomyopathy is the dominant morphological cause of sudden cardiac death in dogs, while hypertrophic cardiomyopathy is more common in cats. The authors emphasize the importance of future research on heart pathologies, including channelopathies and ischemic disorders, which have independent thanatogenetic significance. Ultrasound examination of the thoracic organ complex in critically ill animals can help differentiate between cardiac and respiratory pathologies (Loughran et al., 2019). In another systematic review, the immediate causes of cardiac death in dogs were detailed, focusing on the physiological mechanisms associated with premature ventricular complexes, ventricular tachycardia, ventricular fibrillation, polymorphic ventricular tachycardia, and agonal electrical activity with bradyarrhythmia (Santilli et al., 2021). However, these findings are useful primarily when a cardiological history is known, with microscopic assessment and histopathological examination of the myocardium being crucial (Herman & Eldridge, 2019).

In our accumulated data, we can confirm that a predictor of critical premortem conditions in animals is the presence of complicating pathologies leading to organ failure, characterized by reversible biochemical-functional shifts during acute conditions (e.g., acute respiratory, renal, or hepatic failure). In contrast, terminal conditions are characterized by irreversible morphological changes in multiple organs observed in acute organ failure. Therefore, it is logical to adopt the concept of equating the pathogenesis of functional failures in critical animal conditions with the thanatogenesis of organ disorganization during clinical death. From this perspective, we argue that premortem changes in the body are dialectically linked to the dominant stage of thanatogenesis, and the dying process begins during clinical death in a state of refractory critical condition. Consequently, it is necessary to distinguish between the cause of death that initiates and ends the dying process.

The use of euthanasia for animals in terminal conditions remains a topic of debate from both ethical and forensic veterinary perspectives (Mota-Rojas et al., 2023).

The final synthesis in an expert opinion should logically reveal the key morphological links in thanatogenesis, reconstructing the mechanism of death from the onset of morphological changes to premortem outcomes. The final forensic conclusion should objectively and impartially explain the direct cause-and-effect relationships between the animal's premortem state and its death. The conclusion, integrating clinical and pathomorphological data, serves to clarify the mechanism and specifics of the animal's death for all parties involved in the legal process.

Histopathological changes in organs during sepsis are well-studied in humans (Garofalo et al., 2019), and some researchers suggest modeling the inflammatory response in animal organs, particularly the kidneys (Kosaka et al., 2016). Our research corroborates the morphological changes in the kidneys of dogs and cats during systemic inflammatory responses. Recent studies have focused on the activation of the hemostatic system in systemic inflammation, as the role of thrombin and fibrin generation was previously viewed merely as one determinant of multiple organ failure and death during septic shock (Delabranche et al., 2017). Macrophages directly activate the coagulation cascade early in sepsis (Okamoto et al., 2016). DeLaforcade et al. (2019) reported that hypercoagulation was identified in dogs with sepsis, indicating a potential risk for systemic hemostasis disorders. The predisposition to hypercoagulation has been demonstrated in critically ill dogs and, to a lesser extent, in cats (Pfaff et al., 2020). These findings were corroborated by Sotos et al. (2023), who identified sepsis as a primary factor in the development of thrombosis in dogs. A population of parvovirus-infected dogs was selected for sepsis modeling because parvovirus enteritis predisposes dogs to sepsis by promoting bacterial translocation and immunosuppression (Alves et al., 2020). This concept was further supported by Corda et al. (2023), who used canine parvovirus enteritis to model systemic inflammatory responses. Despite this progress, there is still no consensus in veterinary medicine regarding sepsis and systemic inflammatory response syndrome (Cortellini et al., 2024). Marchetti et al. (2021) experimentally demonstrated that corticosteroid insufficiency associated with critical illness, including systemic inflammation, affects 48% of dogs with sepsis.

If we assume that more than one cause could have contributed to the animal's death, and the expert has doubts about the primary one, then a conclusion about the competition of causes of death based solely on this doubt cannot be considered justified. Such a conclusion is not built on evidence-based cause-and-effect relationships, but rather on a statement of certain positions from the forensic veterinary diagnosis, and therefore, cannot take the form of a categorical statement. For example: a poisoned animal in an unconscious state was submerged in cold water. Analyzing the thanatogenesis in this expert case, it can be concluded that each damaging factor could independently cause death, but the fatal outcome would be due to the prolonged effect of each factor individually. In our opinion, even in such " competing" cases, the expert must determine the cause and specify the role of other determinants in the thanatogenesis.

In our view, the wide range of etiological factors, nosological forms, and pathogenic conditions that lead to disseminated intravascular coagulation involves all organ systems. According to our hypothetical model, the syndrome is consistently accompanied by various forms of shock, complicating the forensic diagnosis of the cause of death, particularly in cases of profuse hemorrhage. It is likely that characteristic morphological patterns in the brain and heart of animal cadavers may not have had time to develop, whereas histomorphological changes occur before macroscopic changes in the organs presented for examination. Unfortunately, the sources mentioned contain only fragmentary morphological confirmations, which require further investigation and refinement.

Conclusions

Among the morphological criteria used to determine the cause of death in animals, it is essential to highlight specific patterns that contribute to the forensic veterinary diagnosis. In cases of "acute cardiac death" in animals, structural changes in the myocardium, such as contractural deformation of the fibers, hemocapillary hyperemia, erythrocyte stasis, vasospasm, and plasma imbibition of the walls of microcirculatory vessels, can collectively be considered a morphological indicator of cardiac death resulting from chronic myocardial hypoxia, representing the physiological end of ontogenesis. Critical states of dogs and cats are prone to rapid decompensation. In forensic veterinary autopsies of animals showing patterns of general coagulopathy, the expert must always consider the possibility of violent death through poisoning, which should be factored into the diagnosis. Animal death in cases of systemic coagulopathy occurs directly due to hemorrhagic hypovolemia amid distributive shock, confirmed by the Minakov sign. Morphological patterns of sepsis are not limited to classic, macroscopically evident lesions but are also found during detailed microscopic examination of focal damage to the microcirculatory vessels in the kidneys, providing diagnostically informative criteria for systemic inflammatory response. The chosen approach allows the use of these identified patterns to substantiate systemic coagulopathy in the final expert conclusion. The application of clinical logic concepts narrows the differential-etiological framework in cases of death resulting from refractory critical conditions.

The authors declare that the research was conducted in the absence of any relationships that could be construed as a potential conflict of interest.

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