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VETERINARY PATHOLOGY OF ORGANOPHOSPHATE TOXICITY IN DOGS

ANNOTATION

Organophosphorus compounds are widely used in agriculture as insecticides and acaricides. However, their improper use or violation of safety regulations can lead to animal poisoning. This study is devoted to the investigation of pathological and histopathological changes in dog that died as a result of acute organophosphorus poisoning.

The object of the study was the carcass of a dog submitted for veterinary-sanitary examination. The necropsy revealed typical macroscopic signs: pronounced rigor mortis, cyanosis of the skin and mucous membranes, hyperemia and pulmonary edema, multiple hemorrhages under serous membranes, in muscles and mucous membranes, as well as erosive and ulcerative lesions in the gastrointestinal tract. The liver, kidneys, and heart also showed signs of dystrophy and congestion.

Histological examination confirmed the presence of edema, hemorrhages, and dystrophic and necrotic changes in the tissues of the lungs, liver, kidneys, and myocardium. Inflammatory and necrotic processes were observed in the stomach and intestines. The identified morphological changes correlate with the known mechanism of toxic action of organophosphorus compounds, which is based on the inhibition of acetylcholinesterase and disruption of vital organ functions.

The obtained results can be used in veterinary diagnostics, forensic examinations, and in the development of preventive measures and the safe application of organophosphorus compounds in animal husbandry.

Key words: *organophosphates, dog, pathological changes, histology, poisoning.*

Introduction. Organophosphate poisoning in animals, particularly pets, represents a significant veterinary concern due to the toxic effects of these compounds, primarily used in pesticides and insecticides. Organophosphates act as acetylcholinesterase inhibitors, leading to the accumulation of acetylcholine at synapses, which subsequently causes severe cholinergic toxicity. Dogs and cats are both at risk for acute poisoning, which presents with various clinical signs such as hypersalivation, tremors, seizures, and respiratory distress [1, 2].

The mechanisms of organophosphate toxicity involve a range of pathophysiological responses. Upon exposure, animals may exhibit acute cholinergic signs indicative of overstimulation of cholinergic pathways, including bronchospasm, miosis, lacrimation, and gastrointestinal disturbances like diarrhea and vomiting [3, 4]. In severe cases, respiratory failure can occur due to the paralysis of respiratory

muscles or bronchoconstriction, necessitating mechanical ventilation in critical care settings [5]. Previous studies emphasize the critical role of prompt treatment, including cholinergic antagonists like atropine and reactivators such as pralidoxime, in reducing morbidity and mortality associated with organophosphate poisoning [6, 7].

The incidence of organophosphate poisoning in pets can be exacerbated by improper product usage, such as the inappropriate application of pesticide sprays or accidental ingestion of treated materials. For instance, cases of poisoning in dogs following the ingestion of cattle ear tags impregnated with organophosphates have been documented, highlighting the need for awareness regarding potential sources of exposure. Moreover, the rising prevalence of such poisoning cases requires veterinary practitioners to remain vigilant. In Nigeria, for example, the incidence of organophosphate and carbamate poisoning in dogs has been reported as a significant public health concern due to persistent agricultural practices favoring these chemicals [8].

From an epidemiological perspective, organophosphate poisoning remains a pressing issue in veterinary medicine, particularly in regions where regulations regarding chemical usage are lax. Studies suggest that dogs are often exposed to organophosphates through environmental contamination or direct application in pest control practices [9]. Due to the high morbidity associated with organophosphate poisoning in animals, it is vital for veterinarians and pet owners alike to understand the symptoms, causes, and treatment protocols associated with such toxicological incidents.

Organophosphate poisoning in dogs is a significant veterinary concern due to the widespread use of organophosphate compounds in agricultural and domestic settings. These compounds function as acetylcholinesterase inhibitors, causing a buildup of acetylcholine at synapses, which leads to overstimulation of the nervous system, resulting in a range of acute clinical signs. The manifestations can include salivation, lacrimation, urination, diarrhea, gastrointestinal distress, and respiratory depression [10].

Studies have documented the prevalence of organophosphate poisoning not only in developed countries but also in regions with less stringent regulatory controls on pesticide usage, such as in Maiduguri, Nigeria, where poisoning incidents were notably linked to deliberate or accidental exposure to agricultural chemicals [11]. Dogs are often exposed to organophosphates through various routes, including ingestion of contaminated food, inhalation during pesticide application, and dermal exposure from treated surfaces [12, 13]. In some cases, immediate clinical signs might emerge, while in others, delayed symptoms, such as paralysis due to intermediate syndrome, can occur several hours after initial exposure.

The treatment of organophosphate poisoning in dogs typically involves the administration of atropine to counteract the cholinergic crisis, along with pralidoxime, which acts to reactivate AChE if administered early enough [14]. However, the prognosis can vary significantly depending on the timing of treatment initiation and the severity of symptoms at presentation. For instance, a retrospective study indicated that the survival rate could be influenced significantly by the promptness of treatment initiation after exposure, where delays could substantially heighten mortality risks [15].

Complications from organophosphate poisoning can also extend to persistent neurological effects or the development of respiratory failure, sometimes necessitating mechanical ventilation [16]. Therefore, veterinary practitioners must maintain a high index of suspicion for organophosphate poisoning in symptomatic dogs, particularly in areas where agricultural chemical use is prevalent, and provide timely and appropriate treatment [17].

Materials and methods. The study was conducted on an animal that died from suspected acute poisoning with organophosphorus compounds. The study included the carcass of a dog that was sent for veterinary and sanitary examination.

The pathological autopsy was performed according to the standard procedure approved by the instructions for forensic veterinary examination. During the autopsy, the state of rigor mortis, the color of the skin and mucous membranes, the presence of hemorrhages, edema, and pathological changes in internal organs were taken into account (Figure 1).



Figure 1 – Internal organs of the dog

To assess the condition of internal organs, a macroscopic examination of the lungs, heart, liver, kidneys, spleen, gastrointestinal tract, and lymph nodes was performed. Attention was paid to blood count, consistency, and color, presence of hemorrhages, erosions, and necrosis.

Selected organ samples (liver, lungs, heart, kidneys, brain, and stomach) were fixed in 10% neutral formalin, poured into paraffin, and stained with hematoxylin and eosin according to the standard protocol for histological analysis.

In addition, in some cases, to confirm the diagnosis, biochemical blood analysis and toxicological examination of the stomach and liver contents for the presence of organophosphorus compounds were performed using gas-liquid chromatography.

Data analysis was performed using descriptive statistics. Comparative data from literature sources and current veterinary standards were used to interpret the results.

Results. At necropsy, dog that succumbed to organophosphate poisoning exhibited marked rigor mortis and pronounced cyanosis of the skin and visible mucous membranes, indicating severe hypoxemia. One of the most consistent findings was generalized hyperemia, especially prominent in the lungs, which were markedly edematous, heavy, and dark red, often exuding frothy, blood-tinged fluid upon sectioning - hallmarks of acute pulmonary edema (Figure 2).



Figure 2 – Dog lungs during necropsy

Petechial and ecchymotic hemorrhages were frequently observed in the mucous membranes, under serous surfaces (particularly of the heart, liver, and intestines), in skeletal muscles, and subcutaneous

tissues. These hemorrhagic manifestations reflect acute vascular damage and coagulopathy associated with systemic toxicity.

The gastrointestinal tract exhibited extensive changes: the gastric and intestinal mucosa were hyperemic and swollen, with multifocal to confluent hemorrhages. Erosions and ulcerations were commonly seen, indicating corrosive action of ingested toxins or their metabolites. The contents often consisted of a mixture of partially digested feed, mucus, and blood. Mesenteric lymph nodes were enlarged and edematous, reflecting systemic inflammatory response and immune activation.

The liver was congested, with an accentuated lobular pattern, and in some cases showed early parenchymal degeneration. The spleen was often slightly enlarged, with pulpy consistency. The kidneys were congested, with petechiae on the cortical surface, and in some instances, cloudy swelling of tubular epithelium was noted, indicating nephrotoxicity.

The heart frequently exhibited epicardial and endocardial hemorrhages, while histological examination typically reveals interstitial edema, myocardial fiber degeneration, and vascular stasis.

These gross lesions collectively reflect the systemic nature of organophosphorus toxicity, characterized by cholinesterase inhibition, overstimulation of the parasympathetic nervous system, and subsequent circulatory, respiratory, and gastrointestinal collapse.

Table 1 – Pathological changes in organophosphorus poisoning

Organ	Pathological anatomical changes
Common characteristics	Pronounced rigor mortis, cyanosis of the skin and mucous membranes
Lungs	Hyperemia, edema, presence of foamy bloody fluid in the bronchial lumen, congestion, hemorrhages
Heart	Punctate and patchy hemorrhages on the epicardium and endocardium
Vascular system	Massive hemorrhages in the mucous membranes, under the serous membranes, in the muscles, and in the subcutaneous tissue
Digestive tract	Hyperemia, erosion, ulceration of the stomach and intestinal mucosa, hemorrhages, presence of mucus and blood
Liver	Congestion, accentuated lobular pattern, dystrophic changes
Kidneys	Congestion, petechiae in cortex, cloudy swelling of tubular epithelium
Spleen	Slight enlargement, pulpy consistency
Lymph nodes	Enlarged, edematous, congested

Table 1 presents the main pathological and anatomical changes observed in various organs and tissues of a dog that died from acute organophosphorus poisoning. The findings reflect the systemic toxic effects caused by inhibition of acetylcholinesterase, resulting in severe disturbances in circulation, respiration, and digestion.

Microscopic examination of the organs of an animal that died from poisoning with organophosphorus compounds revealed pronounced dystrophic and vascular-inflammatory changes. Alveolar and interstitial edema, capillary hyperemia, diapedetic hemorrhages, and accumulation of fibrinous exudate in the alveolar lumen were observed in the lung tissue. The bronchiolar epithelium was desquamated in places, with signs of necrosis.

In the liver, granular and fatty degeneration of hepatocytes, sinusoid enlargement, hyperemia of the central veins, foci of parenchymal necrosis, and periportal lymphohistiocytic infiltrates were detected.

The kidneys were characterized by cloudy swelling and partial breakdown of the epithelium of the convoluted tubules, enlargement of the glomerular capsules, and venous hyperemia. In some cases, there were clusters of erythrocytes and protein casts in the lumen of the tubules.

In the myocardium, there was cardiomyocyte dystrophy, interfibrillar edema, and venous hyperemia. In the stomach and intestines, there was necrosis of the mucosal epithelium, hemorrhages in the lamina propria, and lymphoplasmacytic infiltration.

The histological changes identified reflect the systemic toxic effects of organophosphorus compounds, leading to disturbances in metabolism, microcirculation, and tissue respiration.

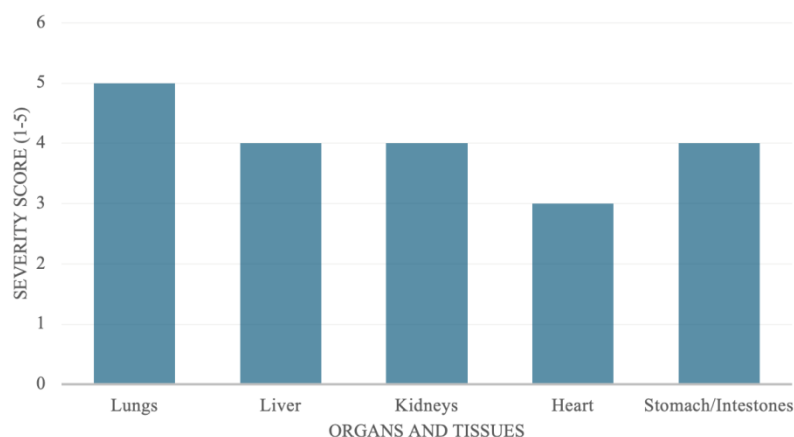


Figure 3 – Severity of histopathological changes in poisoning with organophosphorus compounds

Figure 3 shows comparative severity scores of histopathological changes in various organs of a dog following organophosphate poisoning. The lungs showed the most severe damage (score 5), followed by the liver, kidneys, and gastrointestinal tract (score 4), while the heart was less affected (score 3).

Discussion. The results of pathological autopsies of dead animals suspected of poisoning with organophosphorus compounds indicate systemic lesions, which is consistent with the literature data on the toxicodynamics of these substances. The main mechanism of action of organophosphorus compounds is the inhibition of acetylcholinesterase, leading to the accumulation of acetylcholine and the development of persistent excitation of cholinergic receptors, which causes multiple organ dysfunction [18, 19].

The most pronounced changes are observed in the respiratory system: hyperemia, edema, and foamy bloody bronchial contents indicate the development of acute toxic pulmonary edema, which is typical of organophosphorus poisoning due to parasympathetic hyperstimulation and vascular permeability [20].

Similar lesions have also been described in cattle and small domestic animals [21]. Massive hemorrhages in the mucous membranes, muscles, and serous membranes reflect severe disturbances in microcirculation and vascular tone caused by acetylcholinergic hyperactivity [22].

These changes are consistent with studies indicating the development of hemorrhagic diathesis in acute organophosphorus poisoning [23].

Damage to the gastrointestinal tract in the form of hyperemia, erosions, and ulcerations may be caused by both the direct toxic effect of organophosphorus compounds and ischemia due to vasospasm [24]. The presence of blood and mucus in the contents of the gastrointestinal tract indicates deep inflammatory damage to the mucous membrane.

Dystrophic changes in the liver and cloudy swelling of the renal tubule epithelium reflect the involvement of the liver and kidneys in the metabolism and excretion of toxins, which is also confirmed by experimental data [25]. Enlargement of the spleen and lymph nodes is probably associated with a systemic inflammatory response.

Thus, the results obtained confirm the polyorgan nature of lesions in organophosphorus compound intoxication and can serve as a diagnostic criterion in veterinary and sanitary examinations.

Conclusion. The results of the pathological examination of an animal that died from organophosphorus compound poisoning indicate the development of severe polyorgan damage affecting the respiratory, cardiovascular, digestive, and excretory systems. The most characteristic changes are edema and hyperemia of the lungs, multiple hemorrhages in various tissues, destructive-inflammatory processes in the gastrointestinal tract, as well as dystrophic changes in the liver and kidneys. These pathological signs reflect the typical mechanism of toxic action of organophosphorus compounds— inhibition of cholinesterase and subsequent overstrain of cholinergic transmission, leading to disruption of vital organ function. The data obtained can be used to diagnose cases of acute poisoning, as well as in forensic veterinary examinations and the development of preventive measures in the agricultural and industrial use of organophosphorus compounds.

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ТҮЙІН

Фосфорорганикалық қосылыстар ауыл шаруашылығында инсектицидтер мен акарицидтер ретінде кеңінен қолданылады. Алайда олардың дұрыс қолданылмауы немесе қауіпсіздік талаптарының бұзылуы жануарлардың улануына әкелуі мүмкін. Бұл жұмыс фосфорорганикалық қосылыстармен жедел улану салдарынан өлген иттегі патологоанатомиялық және гистопатологиялық өзгерістерді зерттеуге арналған.

Зерттеу нысаны ретінде ветеринариялық-санитарлық сараптамаға түскен иттің өлекесі алынды. Жарып сою кезінде келесі макроскопиялық белгілер анықталды: айқын өлгеннен кейінгі сіресу, тері мен шырышты қабаттардың цианозы, өкпенің гиперемиясы мен ісінуі, серозды қабықшалар астындағы, бұлшықеттер мен шырышты қабықтардағы көпшілік қан құйылу, сондай-ақ асқазан-ішек жолдарындағы эрозиялық-ойық жаралы зақымданулар. Бауыр, бүйрек және жүрек тіндерінде дистрофия мен қанға толу белгілері байқалды.

Гистологиялық зерттеу нәтижелері өкпе, бауыр, бүйрек және миокард тіндерінде ісіну, қан құйылу, дистрофиялық және некроздық өзгерістердің бар екенін растады. Асқазан мен ішек тіндерінде қабыну және некроздық үдерістер байқалды. Анықталған морфологиялық өзгерістер фосфорорганикалық қосылыстардың ацетилхолинэстеразаны тежеп, өмірлік маңызды ағзалардың қызметін бұзуға негізделген белгілі уытты әсер ету механизмімен сәйкес келеді.

Алынған нәтижелер ветеринариялық диагностикада, сот-сараптамалық тәжірибеде, сондай-ақ фосфорорганикалық қосылыстарды мал шаруашылығында қауіпсіз пайдалану мен алдын алу шараларын әзірлеуде қолдануға болады.

РЕЗЮМЕ

Фосфорорганические соединения широко применяются в сельском хозяйстве в качестве инсектицидов и акарицидов. Однако их неправильное использование или нарушение требований безопасности может привести к отравлению животных. Настоящая работа посвящена изучению патологоанатомических и гистопатологических изменений у собаки, павшей в результате острого отравления фосфорорганическими соединениями.

Объектом исследования стал труп собаки, поступивший на ветеринарно-санитарную экспертизу. При вскрытии были выявлены типичные макроскопические признаки: выраженное трупное окоченение, цианоз кожи и слизистых оболочек, гиперемия и отёк лёгких, множественные кровоизлияния под серозными оболочками, в мышцах и слизистых оболочках, а также эрозивно-язвенные поражения желудочно-кишечного тракта. Печень, почки и сердце также демонстрировали признаки дистрофии и полнокрывия.

Гистологическое исследование подтвердило наличие отёка, кровоизлияний, дистрофических и некротических изменений в тканях лёгких, печени, почек и миокарда. В желудке и кишечнике отмечались воспалительные и некротические процессы. Выявленные морфологические изменения коррелируют с известным механизмом токсического действия фосфорорганических соединений, основанным на ингибировании ацетилхолинэстеразы и нарушении функции жизненно важных органов.

Полученные результаты могут быть использованы в ветеринарной диагностике, судебной экспертизе, а также при разработке профилактических мер и безопасного применения фосфорорганических соединений в животноводстве.