

Case Report

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SMOKE INHALATION INJURIES IN A DOG - CASE REPORT

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Abstract

Smoke inhalation injuries are relatively uncommon in veterinary pathology and the current literature on spontaneous carbon monoxide intoxication in companion animals is limited. The majority of available data are derived from isolated case reports or extrapolated from human forensic studies. Consequently, there is a need for systematic documentation and analysis of such cases to improve diagnostic accuracy and support legal investigations involving animal fatalities in fire-related incidents. In this case, five mixed-breed dogs (two males and three females), ranging in age from one to five years, were found deceased as a result of a residential structure fire. All animals were in good nutritional condition and did not have microchip identification. One dog, at the request of the public prosecutor, was submitted for necropsy to the Department of Forensic Veterinary Medicine and Legislation, Faculty of Veterinary Medicine, University of Belgrade, Serbia, in order to determine the cause of death. Gross examination revealed discoloration of the coat due to soot deposition, cherry-red oral mucosa and the presence of soot particles on the oral mucosa, including the teeth and tongue, as well as in the external ear canal. No external thermal

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injuries, such as burns or signs of carbonization were found. Histopathological findings demonstrated pulmonary edema, soot particles in airways and generalized hyperemia. Toxicological examination of postmortem blood samples collected from the heart and major vessels demonstrated markedly elevated carboxyhemoglobin concentrations (68.67%), consistent with fatal carbon monoxide poisoning.

This case emphasizes the importance of a comprehensive forensic methodology integrating anamnestic data, post-mortem macroscopic and microscopic findings, and toxicological analysis, as essential and reliable tools for investigating fire-related fatalities in companion animals.

Key words: carbon monoxid, poisoning, pet animals, fire-related fatalities, necropsy, toxicology

POVREDE USLED UDISANJA DIMA KOD PSA – PRIKAZ SLUČAJA

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Kratak sadržaj

Povrede nastale usled udisanja dima su relativno retke u veterinarskoj patologiji, a literatura o spontanoj intoksikaciji kućnih ljubimaca ugljen-monoksidom je i dalje oskudna. Većina dostupnih podataka potiče iz pojedinačnih izveštaja ili je zasnovana na rezultatima studija sprovedenih na ljudima. Shodno tome, postoji potreba za sistematskom dokumentacijom i analizom takvih slučajeva kako bi se unapredila pouzdanost dijag-

noze i pružila relevantna podrška pravnim postupcima u vezi sa smrtnim slučajevima životinja u incidentima povezanim sa požarima. U ovom slučaju, pet pasa mešanaca (dva mužjaka i tri ženke), starosti između jedne i pet godina, pronađeno je u stambenoj kući u kojoj je izbio požar. Svi su bili u dobroj kondiciji i bez mikročipa. Jedan pas je, na zahtev javnog tužioca, dostavljen na nekropsiju na Katedru za sudsku veterinarsku medicinu i zakonske propise, Fakulteta veterinarske medicine, Univerziteta u Beogradu, Srbija, radi utvrđivanja uzroka uginuća. Makroskopskim pregledom konstatovana je promena boje dlake usled taloženja čađi, crvenkasta sluzokoža usne duplje i prisustvo čestica čađi na sluzokoži usne duplje, uključujući zube i jezik, kao i u spoljašnjem ušnom kanalu. Spoljašnjim pregledom nisu ustanovljene termičke povrede u vidu opekotina ili karbonizacije. Histološki, ustanovljen je edem pluća, čestice čađi u disajnim putevima i generalizovana hiperemija. Toksikološka analiza uzoraka krvi iz srca i glavnih krvnih sudova nakon uginuća pokazala je značajno povišene koncentracije karboksihemoglobina (68,67%), što je u skladu sa fatalnim trovanjem ugljen-monoksidom.

Ovaj slučaj ističe značaj forenzičke metodologije koja obuhvata anamnestičke podatke, makroskopske i histopatološke nalaze nakon uginuća i toksikološku analizu, kao bitne i pouzdane alate za istraživanje slučajeva uginuća kućih ljubimaca u požarima.

Ključne reči: trovanje ugljen dioksidom, kućni ljubimci, fatalne povrede izazvane požarom, obdukcija, toksikologija

CASE PRESENTATION

Anamnestic Data

In this case, a mixed-breed female dog, approximately two years old, weighing 13.2 kg, with gray coat, was examined. The coat was heavily contaminated with soot, particularly in the head region and on the distal parts of the forelimbs and hindlimbs, suggesting that the dog was alive at the time of the fire and most likely attempting to escape from the smoke-filled environment (Figure 1a). This external finding indicates active contact with smoke and combustion products, while respiratory and circulatory functions were still preserved. Notably, the area around the neck, corresponding to where a collar would have been, remained clean and retained its original white color.

Macroscopic examination

Examination of the oral mucosa revealed no macroscopic lesions. However, it exhibited a cherry-red discoloration and contained soot particles, which were also present on the teeth and tongue (Figure 1b). A dark red discharge, composed of blood and soot particles, was observed flowing spontaneously and under pressure from the nostrils. The skin of the left ear displayed prominent soot deposition and erythema, indicative of thermal and smoke exposure. In contrast, the right ear remained unaffected, suggesting the animal had been positioned on its right side at the time of exposure. The skin on the ventral surface of the abdomen exhibited a cherry-red discoloration and was covered with soot particles.

The tongue is blackened and covered with a substantial amount of soot (Figure 1c). The esophageal mucosa was intact but hyperemic, with a small amount of soft, mushy content containing soot particles. The trachea was completely filled along its entire length with a dark, black-gray, foamy substance (Figure 1d). The lungs were dark red with mottled areas of lighter red, exhibited a rubbery consistency, and had rounded, blunt edges. Upon sectioning, the lung parenchyma was moist, with moderate amounts of frothy, blood-tinged fluid present in the airways.

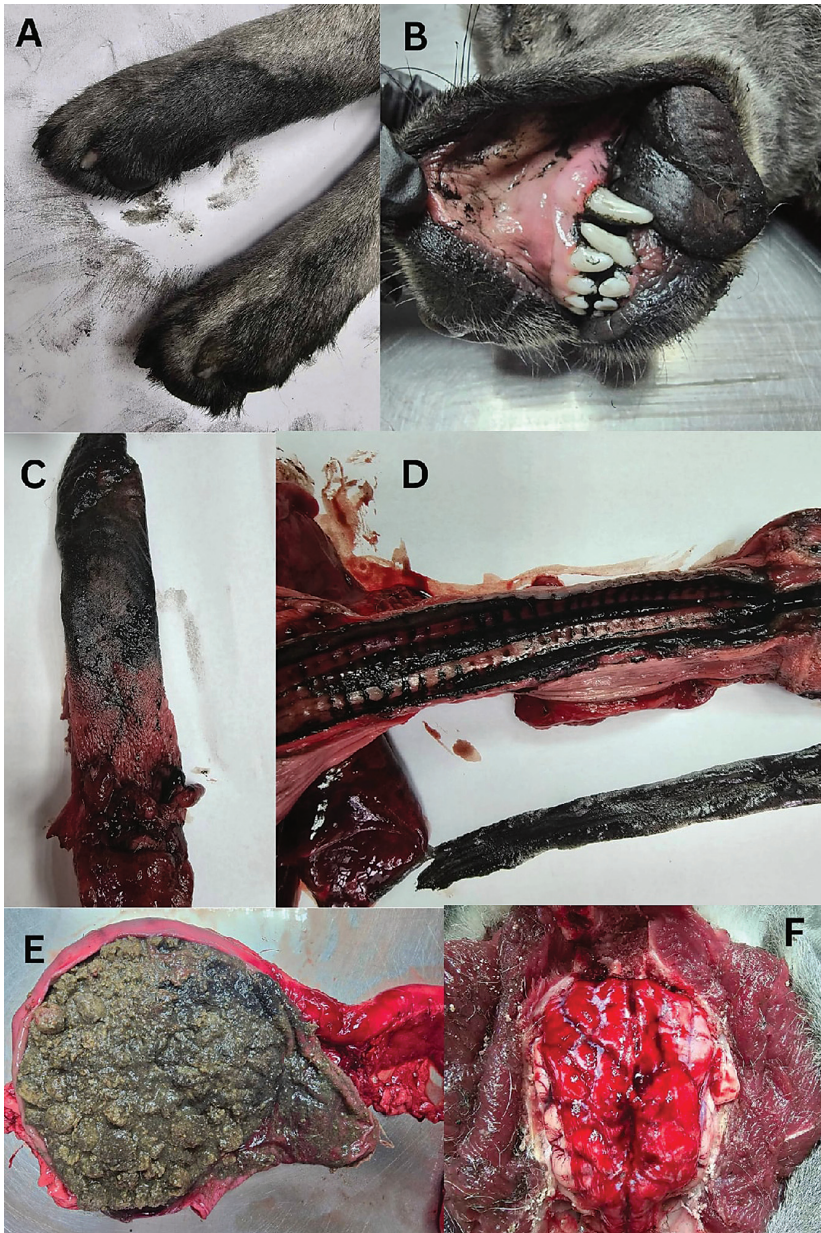


Figure 1. 1a) heavily contaminated coat with soot on the distal parts of the forelimbs; 1b) cherry-red discoloration and contained soot particles in oral mucosa; 1c) blackened and soot on the tongue; 1d) The trachea completely filled with a dark black-gray, foamy material; 1f) meningeal hyperemia, characterized by diffuse vascular congestion.

Gross examination of the stomach revealed black pigmentation of the gastric contents (Figure 1e), indicative of ingested soot, likely resulting from the animal's inhalation and subsequent swallowing of combustion by-products during the fire. Examination of the brain revealed marked meningeal hyperemia, characterized by diffuse vascular congestion, indicative of an acute hypoxic event, most likely resulting from smoke inhalation (Figure 1f).

Following necropsy, tissue samples (lungs, heart, liver, kidney, gaster, trachea) were collected for histopathological analysis and fixed in 10% neutral buffered formalin. The samples were embedded in paraffin, sectioned at 5 μm , and stained with hematoxylin and eosin (H&E). For the toxicological evaluation of potential carbon monoxide poisoning, postmortem blood samples were collected from the heart and major blood vessels in order to determine carboxyhemoglobin (COHb) concentrations via spectrophotometric analysis.

Microscopic examination

Microscopic examination revealed that the respiratory system is predominantly affected. In the lungs, pulmonary edema (Figure 2a) is evident, characterized by the accumulation of homogeneous eosinophilic fluid within the alveolar spaces. Anthracotic pigment (coal dust particles) is observed in the alveoli (Figure 2b), along with desquamated alveolar epithelial cells and intra-alveolar hemorrhages, indicated by the presence of extravasated erythrocytes. The vasculature of the alveolar septa and the larger interstitial vessels exhibited marked hyperemia with luminal spaces densely packed with erythrocytes. In the areas unaffected by edema, alveolar emphysema was observed, characterized by rupture of alveolar septa and coalescence of adjacent alveoli.

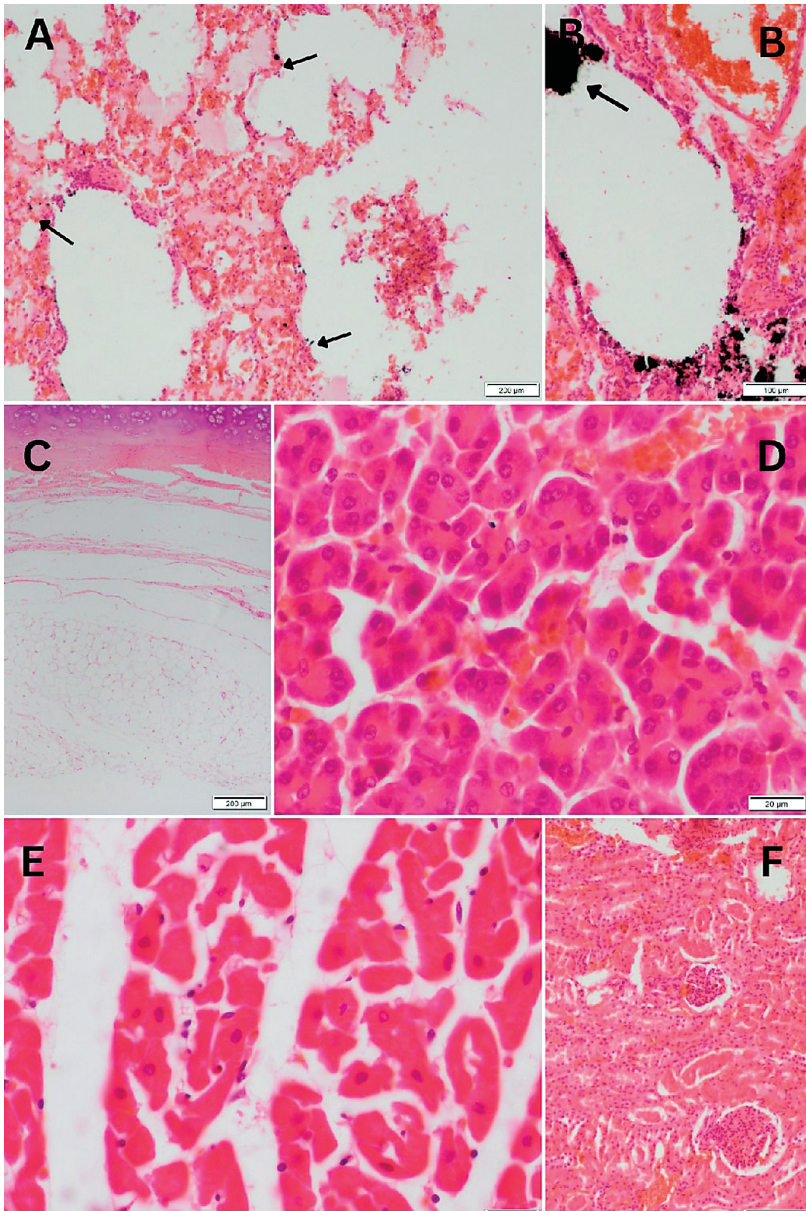


Figure 2. 2a) pulmonary edema characterized by the accumulation of homogeneous eosinophilic fluid within the alveolar spaces; 2b) desquamated alveolar epithelial cells and intra-alveolar hemorrhages; 2c) pronounced hyperemia and epithelial architectural distortion in trachea; 2d) pyknosis in the hepatocytes which are notably swollen; 2e) myocardial tissue revealed multifocal to coalescing areas of coagulative necrosis, accompanied by interstitial edema and neutrophilic infiltration.

The tracheal mucosa displayed significant structural damage, including pronounced hyperemia and epithelial architectural distortion. Surface accumulation of desquamated epithelial cells and particulate carbonaceous material is noted (Figure 2c). Additionally, a homogeneous, partially coagulated eosinophilic exudate was present on the epithelial surface.

In the hepatic tissue, hepatocytes are notably swollen, with cytoplasmic vacuolation and mildly cloudy appearance. Cell nuclei were prominent, with some displaying features consistent with pyknosis (Figure 2d). The sinusoidal capillaries are engorged with erythrocytes, indicating sinusoidal congestion.

Microscopic examination of the myocardial tissue revealed multifocal to coalescing areas of coagulative necrosis, accompanied by interstitial edema and neutrophilic infiltration (Figure 2e). Myocardial fibers exhibited wavy degeneration and occasional contraction band necrosis, suggestive of catecholamine-induced injury and hypoxia. Vascular congestion and perivascular hemorrhage were also noted.

Microscopic examination of renal tissues revealed mild interstitial edema and mononuclear inflammatory infiltrates along with occasional glomerular congestion (Figure 2f). The kidneys exhibited features consistent with acute tubular necrosis, including epithelial cell desquamation, cytoplasmic vacuolation, and loss of brush borders.

Histopathological examination of cerebral tissues revealed changes consistent with acute hypoxic-ischemic encephalopathy. Neurons, particularly within the hippocampus and cerebral cortex, showed eosinophilic cytoplasm and pyknotic nuclei, indicating irreversible injury (Figure 3a). Perivascular and perineuronal edema (Figure 3b) were common, along with congestion of meningeal and parenchymal vessels. Microglial nodules and reactive astrogliosis (Figure 3c) were observed, indicating an early glial response to injury.

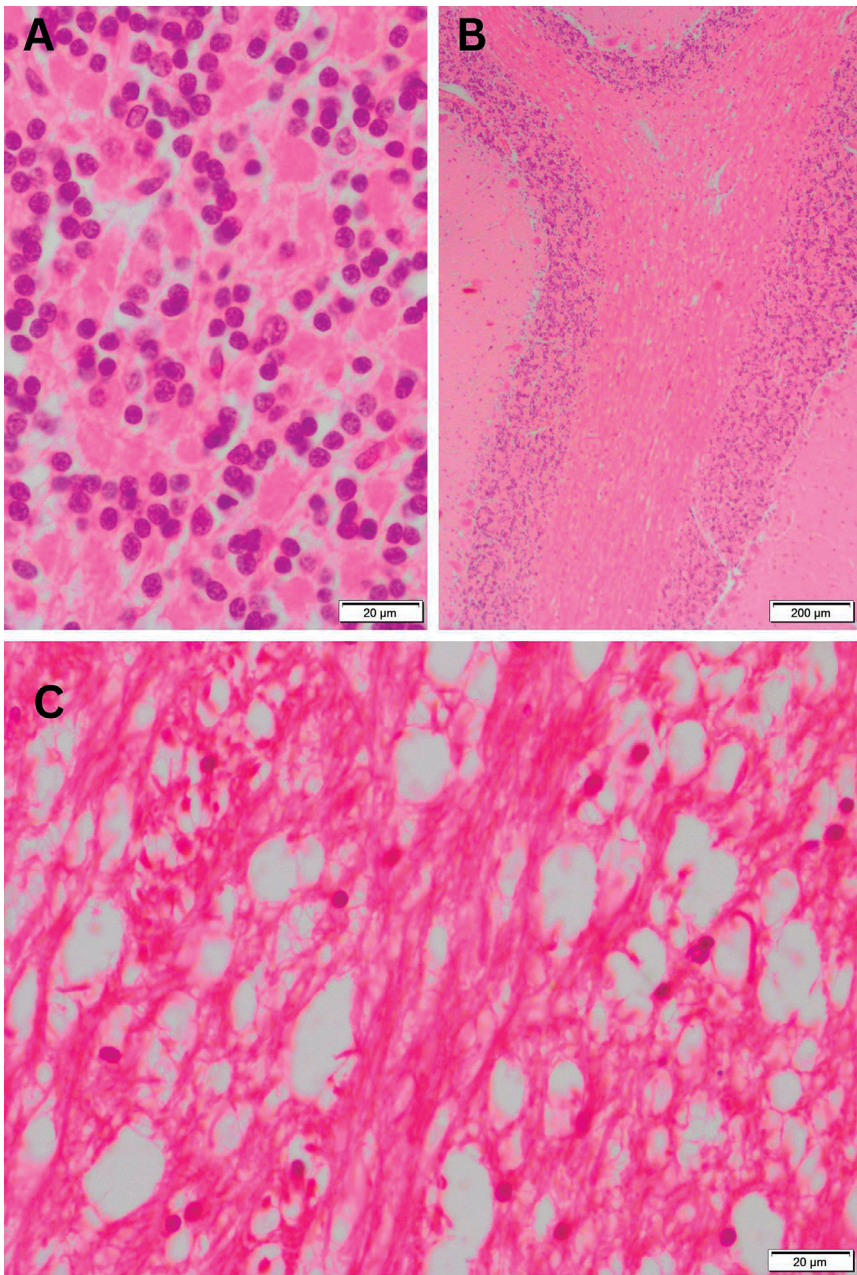


Figure 3. Histopathological examination of the brain

Toxicological analysis

Carboxyhemoglobin (COHb) levels were determined using a spectrophotometric method. To 0.5 mL of blood, 2 mL of distilled water was added. 1 mL of obtained hemolysate was taken and mixed with 4 mL of acetate buffer pH = 5 (5 mol/dm³ acetic acid and 3 mol/dm³ sodium-acetate (1:3)) and sample was heated at 55 °C for 5 minutes. The sample was then cooled under a stream of water and subsequently centrifuged at 3000 rpm. Liquid sample was filtered and absorbance was measured at 533 nm. The concentration of COHb was calculated using the following formula: %COHb = Abs/0,0405.

DISCUSSION

Thermal injuries, although relatively uncommon in veterinary pathology (Munro and Thrusfield, 2001), pose a significant challenge in the field of veterinary forensics. In animals, this type of injuries can be caused by open fire, boiling water, chemicals, ionizing radiation, as well as by improper use of electric heaters or insufficiently grounded plates of electric scooters (Zhenikhova et al., 2024). In fire-related fatalities (FRFs), death may result directly from extensive thermal trauma, including cutaneous burns or indirectly from the inhalation of smoke and the consequent exposure to toxic combustion products (Madea et al., 2004, Jordaan et al., 2020).

Inhalation of hot and toxic gases is one of the primary contributors to mortality in fire victims, underscoring the importance of comprehensive toxicological investigations. During fires, smoke and combustion gases contain a complex mixture of toxic substances, including carbon monoxide (CO), cyanide (CN), acrolein and other pulmonary irritants (Alarie, 2002; Madea et al., 2004). These substances are produced during the combustion or smoldering of various materials (furnishings, clothing and plastics) leading to chemical injury of the tracheobronchial airways and pulmonary parenchyma (Farrow, 1983; Geor and Ames, 1991). Exposure to these toxic compounds can cause rapid unconsciousness or death, often occurring before any visible external injuries become apparent. Smoke inhalation injury (SII) refers to the inhalation of superheated gases, steam, chemical vapors, and particulate matter, leading to primary damage to the respiratory tract. Smoke inhalation induces three distinct physiological types of injury: (a) thermal injury, predominantly affecting the upper airway; (b) chemical injury, impacting both the upper and lower respiratory tract; and (c) systemic effects of toxic gases, such as CO and CN (Gupta et al., 2018) or any combination of these. The prevalence of smoke

inhalation injury (SII) in small animals remains unclear, primarily due to high prehospital mortality rates and the lack of a centralized reporting database (Rosati et al., 2020).

Forensic indicators such as the presence of soot in the airways, esophagus or stomach can provide valuable information regarding the animal's physiological status at the time of the fire. Additionally, toxicological findings, especially the elevated levels of COHb or CN, are pivotal in establishing the cause of death (Madea et al., 2004).

The veterinary forensic expert plays a vital role in determining whether an animal was alive at the time of fire exposure - an essential factor in establishing the cause and manner of death during legal investigations. Evidence of vital reactions to fire, such as the inhalation of hot gases or steam, often coexists with varying degrees of postmortem thermal damage (Madea et al., 2004). One of the most significant indicators is the presence of soot on the mucous membranes of the oral and nasal cavities, as well as throughout the respiratory and upper digestive tracts, including the stomach, which suggests the animal was breathing and swallowing during the fire (Stern et al., 2014). However, the interpretation of soot deposition requires careful consideration. While the presence of soot in the deep airways and digestive tract serves as a strong indicator of life during the fire, its absence does not necessarily rule out antemortem exposure (Madea et al., 2004). Soot particles can passively enter the oral cavity of a deceased animal, potentially depositing on the tongue, pharynx or even the glottis. In contrast, the presence of soot deeper within the trachea or in the stomach provides more definitive evidence of vital exposure (Knight, 1996; Madea et al., 2004). Additional respiratory findings, including epithelial damage, loss of cilia, increased mucus production, and edema (particularly in the proximal trachea) further support the diagnosis of smoke inhalation and are most effectively evaluated through histological sampling at multiple levels (Galeiras, 2021).

Soot was identified within both airways and alveolar spaces in this case report. These findings are similar to those reported by Stern et al. (2014). The presence of pulmonary edema, characterized by homogeneous eosinophilic fluid accumulation within alveoli, aligns with observations in both canine and bear models. For instance, a study by Tunç and Kutsal (2023) on bears revealed similar pulmonary lesions, including edema and emphysema, following smoke exposure. These findings suggest that the severity of pulmonary damage is dose-dependent and can vary based on species and exposure duration.

Significant damage to the tracheal mucosa, including hyperemia, epithelial irregularities, and soot particle deposition, has been documented in this case

report. A case report by Rosati et al. (2020) describing a dog with obstructive tracheal necrosis secondary to smoke inhalation documented similar mucosal injuries. Such lesions are indicative of the direct toxic effects of inhaled smoke on airway epithelium. The findings of surface accumulation of desquamated epithelial cells and particulate carbonaceous material in trachea in this case report are similar to those reported by Wohlsein et al. (2016).

The observed hepatic congestion, characterized by swollen hepatocytes and sinusoidal erythrocyte accumulation, is in line with the findings commonly reported in cases of smoke exposure. A study on toxic smoke inhalation in fire dog victims by Stern et al. (2014) reported hepatic congestion and cellular alterations consistent with the effects of systemic hypoxia and circulatory disturbances.

The skeletal muscle changes observed in this case report, including coagulative necrosis and interstitial edema, are notable findings this case report. These alterations are often attributed to ischemic injury and inflammatory responses following smoke inhalation.

Microscopic examination of renal tissues from dogs exposed to smoke inhalation in this case report revealed acute tubular necrosis, characterized by tubular epithelial cell necrosis, loss of brush borders, cytoplasmic vacuolation, and occasional glomerular congestion. Interstitial edema and mild infiltration of inflammatory cells were also noted. These alterations are likely secondary to systemic hypoxia, carbon monoxide toxicity, and oxidative stress resulting from exposure to smoke constituents.

The extent of renal damage appeared to correlate with the duration and severity of smoke exposure.

Myocardial lesions may reflect a combination of hypoxia, oxidative stress and sympathetic overactivity. Cerebral damage appears to be primarily hypoxic in nature; however, the potential for direct neurotoxic effects from combustion byproducts cannot be excluded. These findings are in line with changes reported in human and experimental models of smoke exposure (Gorgun et al., 2017).

CO is the primary cause of fatalities in residential and room fires (Rodkey et al., 1974). CO poisoning, a common and potentially fatal consequence of smoke inhalation during fires, occurs when CO binds to hemoglobin with an affinity approximately 250 times greater than that of oxygen, forming COHb and thereby severely impairing oxygen delivery to tissues. Clinical signs of CO toxicity typically emerge when COHb levels exceed 10% (Tintinalli et al., 2016), with progression to lethargy, disorientation and eventually cardiac arrest at concentrations above 50%. There is no direct correlation between

COHb levels and the presence or intensity of clinical, gross, or histologic signs, though levels above 10% are considered to be indicative of toxicity (Ashbaugh et al., 2012). The central nervous system and heart are particularly vulnerable to hypoxic injury (Merck and Miller, 2013). In animals which have higher metabolic rates than humans, lethal COHb levels can be reached more rapidly. Postmortem detection of elevated COHb levels serves as a reliable indicator that the animal was alive during the fire and had inhaled toxic gases (Rose et al., 2017; Ashbaugh et al., 2012; Piantadosi, 1999). CO has a high affinity for myoglobin, and binding to cardiac myoglobin can lead to myocardial depression, hypotension and arrhythmias. Cardiac decompensation further exacerbates tissue hypoxia, which ultimately leads to death (Blumenthal, 2001).

In forensic fire investigations involving animals, laboratory testing plays a critical role, providing crucial evidence for establishing the cause of death. The detection of COHb in postmortem blood samples serves as a key marker of antemortem inhalation, thereby reinforcing conclusions derived from gross pathology and histopathological findings.

The integration of gross pathology, microscopic examination and toxicology is indispensable for accurately distinguishing between various forms of fire-related trauma and for providing objective, scientifically grounded evidence essential for legal investigations into animal fatalities (Wohlsein et al., 2016). In order to establish a definitive diagnosis in cases of fire-related fatalities in animals, a thorough evaluation of the anamnestic data, in conjunction with both macroscopic and microscopic findings, as well as comprehensive toxicological analysis, is crucial.

This case highlights the importance of a thorough forensic veterinary approach in the investigation of animal deaths related to fire incidents and underscores the critical importance of a comprehensive forensic veterinary approach in determining the cause of death in animals exposed to fire-related incidents. In this case, the presence of soot on the mucosal surfaces of the trachea, esophagus and stomach indicates exposure to open fire (Wohlsein et al. 2016).

The presence of soot on the mucosal surfaces of the trachea, esophagus and stomach suggests vital reactions of the organism and confirms exposure to smoke and combustion products during the agonal or pre-mortem phase. This finding serves as a key indicator of vitality and respiration during the fire (Stern et al., 2014). The presence of inhaled smoke particles in the stomach further corroborates the likelihood of life during the smoky phase of the fire (Knight, 1996; Madea et al., 2004). Additionally, the detection of soot in the airways or upper gastrointestinal tract is nearly always associated with toxic

or lethal concentrations of COHb and CN (Stoll et al., 2016), as confirmed by spectrophotometric analysis. This report also underscores the value of post-mortem blood as a reliable sample for diagnostic confirmation.

In this case, the absence of thermal injuries, combined with the presence of soot particles in the respiratory and digestive tract and elevated COHb levels strongly supports the conclusion that the dog deceased due to indirect effects of fire - smoke inhalation and the associated exposure to toxic combustion products.

CONCLUSION

This case highlights the critical importance of comprehensive forensic veterinary examination integrating gross pathology, histopathology and toxicology for determining the cause of death in fire-related animal fatalities. It also underscores the evidentiary value of soot distribution and COHb quantification in differentiating antemortem from postmortem fire exposure.

Author's Contribution

JAR - performed the necropsies and wrote the manuscript, EN - English proof reading, ID - preparation of histological slides using the standard hematoxylin and eosin (H&E) staining method and the analysis and interpretation of histopathological findings, VN performed necropsy, DV participated in writing of the manuscript, SĐ performed toxicological analysis, DD designed the study and participated in writing of the manuscript.

Competing interest

The authors declare no conflict of interest.

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