Fire-associated bear mortalities - Histopathological study

Research Article

ABSTRACT

In the study, it was aimed to describe the histopathological findings in bears that died as a result of smoke poisoning in natural fire deaths, unlike model studies. Himalayan (n=3) and brown bears (n=7) that died due to smoke in the fire were brought for necropsy. Macroscopically, there were no burns or injuries on the bearskins. The lumens of the trachea were filled with edema and had petechial to ecchymotic hemorrhages. There were hemorrhage areas ranging from the size of a pinhead to large areas of ecchymosis, spreading diffusely in all lung lobes. Pulmonary emphysemas were found in varying sizes, especially in the distal lobes. In all bronchi, bronchioles, most alveoli, and under the pleura were detected filled areas with erythrocytes. The interalveolar septal regions were noted to be thickened by erythrocyte/inflammatory cell infiltration. Desquamation of lamina epithelialis, edema, emphysema, and carbon pigment in alveolar macrophages and/or free were observed. Since, many studies on smoke inhalation are experimental, in this respect, the death findings noted in this study are thought to be very valuable since the bears died naturally. Additionally, symptoms caused by acute smoke inhalation in bears have been described. To the authors' knowledge, this is the first fire-related study in bears, and it is considered that bears have died from carbon monoxide inhalation.

Keywords: Bear, fire, histopathology, mortality, smoke inhalation

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INTRODUCTION

As it is known, many animals die in fires, especially wildfires. While some animals die from smoke poisoning, some die from burning in the fire (Jordaan et al., 2020) Inhalation of smoke can cause various acute and chronic lung diseases in surviving animals. In addition to experimental animals (Dong et al., 2015), various animal models have been developed, including both small and large animals (Reczyńska et al., 2018). Thus, these models have enabled a better understanding of the mechanism, pathogenesis and pathophysiology of damage caused by smoke inhalation and the development of new treatments. However, unfortunately, these models do not fully reflect the natural and real consequences of fire.

Animals can tolerate a maximum body temperature of approximately 50°C. As the temperature increases, cells denature proteins and become inactive rapidly, thus disrupting the membrane structure (Schmidt-Nielson, 1979). The length of time that an animal is exposed to high temperature, anoxia, or smoke is crucial (Engstrom, 2010; Whelan, 1995). Increased carbon dioxide and carbon monoxide resulting from smoke inhalation cause decrease in oxygen binding to hemoglobin, thus improving oxygenation in the tissue. This causes lung damage (Rutter et al., 2013). Early and late complications occur in the respiratory system due to smoke inhalation. Pneumonia and airway obstruction are from early complications. Bronchiolitis obliterans, tracheal stenosis, sepsis, acute lung injury and acute

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respiratory distress syndrome are also delayed complications (Reczyńska et al, 2018; Wohlsein et al., 2016).

Early and late complications occur in the respiratory system due to smoke inhalation. Pneumonia and airway obstruction are from early complications. Bronchiolitis obliterans, tracheal stenosis, sepsis, acute lung injury and acute respiratory distress syndrome are also delayed complications (Reczyńska et al, 2018; Wohlsein et al., 2016). The thermal damage occurring here activates the inflammatory response. The functions of ciliary cells and alveolar macrophages decrease, and surfactant production is impaired. Thus, predisposing those affected by smoke inhalation to develop respiratory infections (Dries and Endorf, 2013; Wohlsein et al., 2016).

The aim of the study is to describe the histopathological findings in bears that died as a result of smoke poisoning in natural fire deaths, unlike model studies.

MATERIALS AND METHODS

It is unknown how the fire started, but it was reported that 10-bears died due to smoke inhalation in the fire that broke out in the municipal shelter where circus animals were kept. The chipboards in the shelter where the bears stayed caught fire, and the animals inside were affected by the smoke. According to the information given by the authorities, it was reported that 9 bears died there due to smoke intoxication in the fire that broke out at night. The bears were brought for necropsy in the afternoon of the same day. However, the 10th bear, which was affected by the fire but survived, died 5 days later and was brought for necropsy. Himalayan bears (n=3) and brown bears (n=7) were 1 (n=2), 7 (n=2), 9 (n=2), 19 (n=1), and 24 (n=3) years old. Three of the bears were males and 7 of them were females. After the fire, all the bears died and were brought to the Pathology Department for necropsy. After necropsy, tissue samples were fixed in 10% neutral buffered formalin. Then routine process, the samples were embedded in paraffin, sectioned at 5 µm, and stained with hematoxylin-eosin (HE).

RESULTS

Macroscopic findings

There were no burns or injuries on the bearskins. The fattening status of the cadavers was normal, and their death stiffnesses occurred. The tracheal mucosa showed markedly hyperemic and petechial to ecchymotic hemorrhages. Additionally, the lumens of the trachea were filled with foamy exudate (Figure 1A).

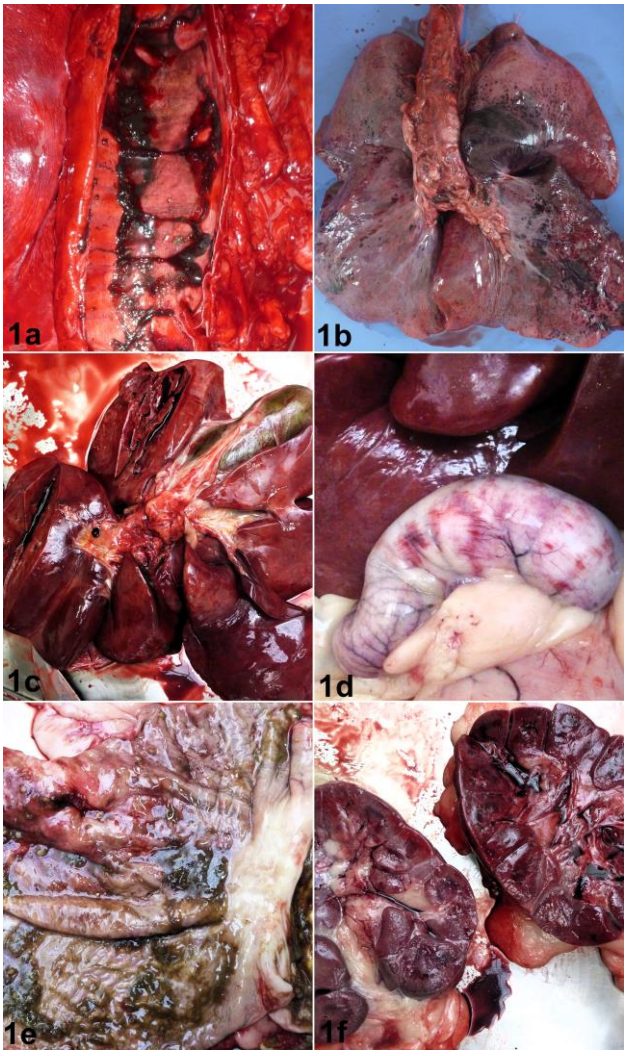

Figure 1. The lumen of the trachea filled with edema and hemorrhage (A), hemorrhage areas ranging from the size of a pinhead to large areas of ecchymosis, spreading diffusely in all lung lobes (B). The blood coming from the cut sections of the liver (C) and kidneys (F), hemorrhage areas at gastric (D) and intestinal mucosa (E).

There were hemorrhage areas ranging from the size of a pinhead to large areas of ecchymosis, spreading diffusely in all lung lobes, including

cyanotic areas in places (Figure 1B). Pulmonary emphysemas were found in varying sizes, especially in the distal lobes. Bullous emphysema was observed in only one of the animals. The livers were swollen, and blood was coming from the cut sections of the livers (Figure 1C). In addition, gastric and intestinal mucosa were hyperemic and had hemorrhage areas (Figure 1D-E). The blood vessels of the brains were swollen and hyperemic. The cut sections of the kidneys were also hyperemic (Figure 1F).

Microscopic findings

In all bronchi, bronchioles, most alveoli, vessels and under the pleura were detected filled areas with erythrocytes (Figure 2A) (n=10).

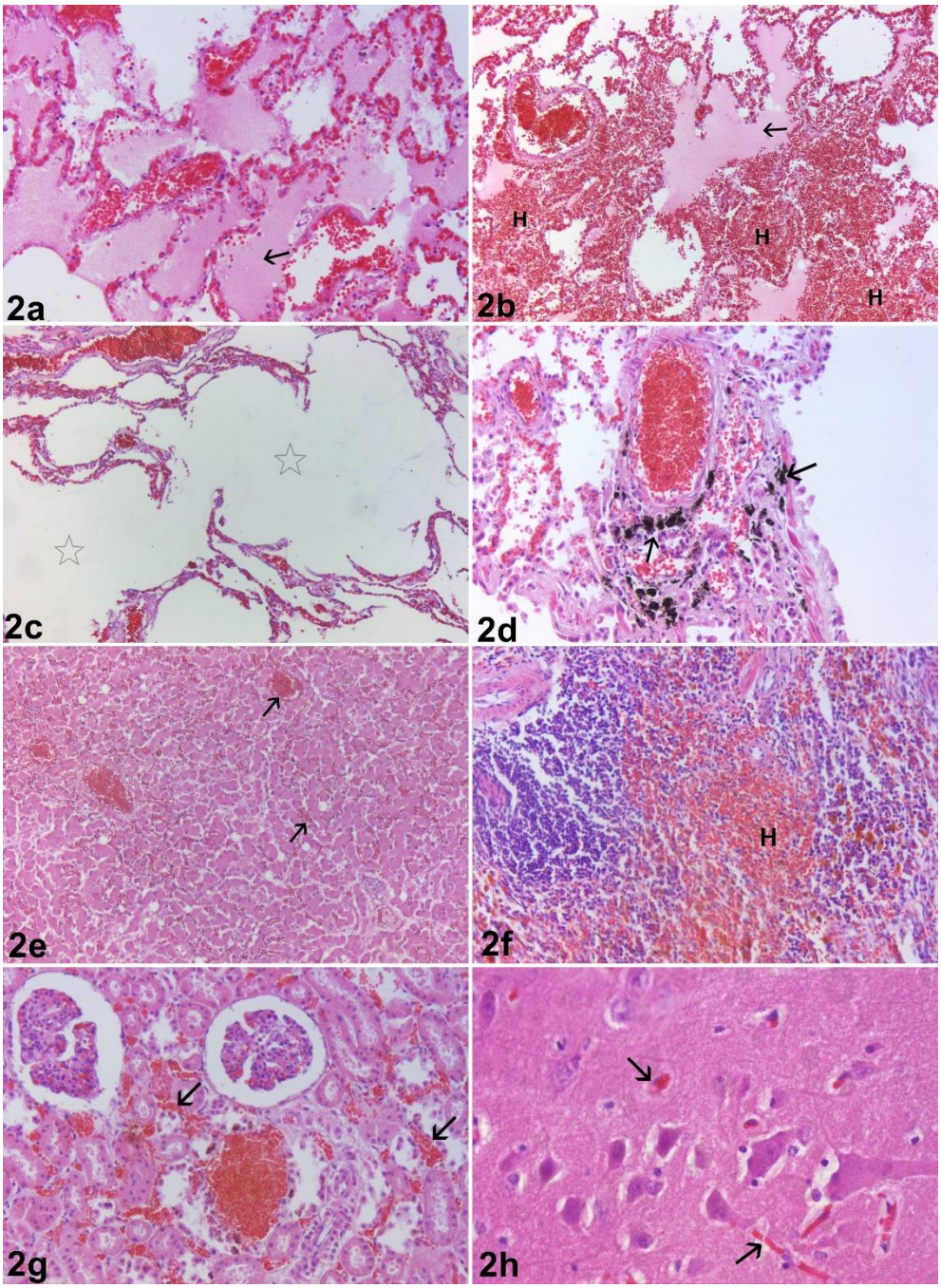

Figure 2. Edema (black arrows) and erythrocyte-filled vessels, bronchioles, alveoli, and hemorrhage (H) areas (A-B), emphysema (stars) in alveoli (C) and carbon pigments (anthracosis) (black arrows) as free and/or in alveolar macrophages (D). Vessels and sinusoids in the liver filled with erythrocytes (black arrows) (E), hyperemic vessels (black arrows) and hemorrhages (H) areas in the spleen (F) and kidneys (G), hyperemic vessels (black arrows) in the brain (H).

The interalveolar septal regions were noted to be thickened by erythrocyte/inflammatory cell infiltration (n=10). The lamina epithelialis layers in some areas were shed to lumens of the bronchi and bronchiole (n=5). Edema was observed in the lumens of some alveoli (n=8) (Figure 2B). The alveoli had an emphysematous appearance in many areas (Figure 2C). In addition, the presence of carbon pigment in alveolar macrophages and/or free (Figure 2D) was determined around the vessels and in interalveolar regions in some areas (n=6).

The vessels and sinusoids in the livers of all animals were filled with erythrocytes. Remak cords were disorganized, and hepatocytes were quite swollen (n=10) (Figure 2E).

In addition, the vessels of the intestine, stomach, spleen (Figure 2F), kidneys (Figure 2G), and heart were hyperemic, and hemorrhages were observed in some areas of these organs (n=10).

The vessels of all brains were severely hyperemic (Figure 2H). While hemorrhage areas were encountered in some areas, perivascular edema was noted in many areas. Vacuolar degeneration was frequently observed in neurons (n=10).

DISCUSSION

Gas inhalation and smoke, followed by respiratory failure, are the important causes of death in living things due to fire (Reczyńska et al., 2018). The degree of inhalation injury depends on the burning material and the components of the inhaled gas. Lower airway damage from smoke inhalation is attributed to toxic gases causing asphyxiation, systemic toxicity, or direct damage to respiratory tissue (Savolainen et al., 1997; Walker et al., 2015). Two known toxic gases are very important in fires, carbon monoxide (CO) and cyanide. Of these, CO is released in slowly burning fires and is lethal to all animals as well as humans. The main cause of death in house and room fires is CO poisoning. CO has approximately 250 times greater affinity for hemoglobin than oxygen, leading to anoxia or hypoxia resulting from failure to oxygenate the blood (Rodkey et al., 1974; Wohlsein et al., 2016). In the study, a fire broke out in the room where the bears were kept, and they were found dead. Therefore, first, CO poisoning was come to mind. Because bearskins were not affected by the fire. There were no wounds or burn marks. Histopathological examinations showed that the bears likely succumbed to smoke inhalation during the fire, as carbon particles were present in their lungs and respiratory tract. The limitation of the study is that since the animals were brought to necropsy dead, unfortunately, their blood could not be examined for carbon monoxide poisoning.

Sheep is the most common animal used in smoke inhalation studies. Hubbard et al. (1991), in a study on 57 sheep, evaluated them for periods ranging from 15 minutes to 4 weeks after smoke inhalation. They stated that necrosis and shedding of the respiratory tract epithelium are the most important lesions caused by smoke inhalation. These findings were seen even in sheep exposed for 15 minutes. Mongrel dogs (Brizio-Molteni et al., 1984) and the New Zealand white rabbits (Thorning et al., 1982) exposed after inhalation of pinewood smoke. Lungs of dogs were examined after 30 min and rabbits up to 72 h after smoke exposure. Extensive hemorrhage and oedema of canine lungs and focal necrosis of rabbit lungs were detected. But, after such a short time, no significant changes to tracheal and/or airway epithelium were found. in dogs, the alveolar spaces were filled with erythrocytes and edema. Several neutrophilic granulocytes were present at some areas. However, no necrosis was encountered in the lungs in this study. But the presence of large amounts of erythrocytes and edema in the lumens of the majority of bronchi, bronchioles and alveoli in the lungs was noted. This was similar to the findings of the mentioned studies. On the other hand, in this study, not only the lung findings in the animals, but all organs

(liver, brain, kidney etc.) were examined in detail and hemorrhage areas and hyperemia were found. In this case, it adds difference to the study.

In the other experimental study (Zhu et al., 2012), for experiment group (9 min time of smoke inhalation) used 48 rats. After smoke inhalation, six rats were killed each time in the at 2 h, 4 h, 6 h, 24 h, 48 h, 96 h, 7 days, and 28 days. When lung histopathologically examined for the effects of smoke inhalation, rat lungs had extensive hemorrhage and accumulation of black particles. 24 hours after smoke inhalation, inflammatory exudates and diffuse hemorrhage were noted, with marked edema in the lungs. At 96 hours, these findings disappeared, and alveolar collapse occurred with partial thickening of the alveolar septum (Zhu et al., 2012). The findings encountered in this study are consistent with those observed in the first 24 hours. Anthracosis pigments, edema, emphysema, and hemorrhage were the most prominent findings. But there was no alveolar septum thickening. It is thought that this situation was not seen because there were no living bears after the fire. Maybe if they had lived for 96 hours, similar findings would have been seen. However, the findings seen in bears that died quickly from smoke inhalation at the fire scene are quite similar to the findings seen in rats in the first 24 hours of the experimental study.

CONCLUSION

Many studies on smoke inhalation are experimental. In this respect, the death findings noted in this study are thought to be very valuable since the bears died naturally. Additionally, symptoms caused by acute smoke inhalation in bears have been described. To the authors' knowledge, this is the first fire-related study in bears, and it is considered that bears have died from carbon monoxide inhalation.

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Conflict of interest: The authors declared that there is no conflict of interest.

Ethical statement: This study was approved by the Ankara University Animal Experiments Local Ethics Committee, Ankara, Türkiye (Approval no: 2023-18-161).

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