

Review Article

DEATH DUE TO SMOKE INHALATION IN A COW

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Abstract: Cattle producers face serious threats to their livestock in face of sudden fires following accidental or suspicious causatives either within the housing or in establishments around the barn; as a result they bear severe economic loss owing to the death of livestock. The department of Veterinary Pathology received an adult female Jersey crossbred cow carcass for postmortem examination with history of fire accident death. Grossly, highly congested mucous membranes, scald wounds, linear abrasions on thoracic vertebra, amorphous dark black soot on nostrils and trachea, pulmonary congestion, edema, atelectasis, collapse, grey black fluid in the bronchiole lumen of lung, epicardium revealed petechiae and ecchymosis, cerebral and meningeal blood vessels were congested. Histopathological examination of lung revealed pulmonary emphysema, edema, atelectasis, fibrosis, charred carbon particles on the tracheal lumen and submucosa and third degree cutaneous burns. Animal died due to asphyxia which might be caused by inhalation of smoke and toxic gases.

Keywords: Cow, pathology, asphyxia, fire accident, smoke inhalation.

INTRODUCTION

Most of the fire related deaths in livestock in urban areas are related either solely or in conjunction with nearby household fires which involves human casualties. Fatal residential fires account for 10% of all accidental deaths in developed countries (Perananthamet *al.*, 2014). Life threatening burns and inhalation injuries are seen with increasing frequency, as a result of which a forensic approach is needed to aid in the comparative diagnosis of the human counterparts involved in the crisis.

Asphyxia is any condition which leads to tissue oxygen deprivation (Richards and Wallis, 2005). Common causes include drowning, smothering, obstruction of foreign body in the respiratory tract, inhalation of smoke or poisonous gases, pathology of the respiratory system etc. Smoke inhalation alone can cause lung injury, or the combination of smoke injury with body surface burn can markedly increase respiratory complications compared with burn alone (Hales *et al.*, 1985; Thompson *et al.*, 1986). All smoke contains carbon monoxide, carbon dioxide and particulate matter (PM or soot). Smoke can contain many different chemicals,

including aldehydes, acid gases, sulfur dioxide, nitrogen oxides, polycyclic aromatic hydrocarbons (PAHs), benzene, toluene, styrene, metals and dioxins. The type and amount of particles and chemicals in smoke varies depending on what is burning, how much oxygen is available, and the burn temperature (NYS, 2016). Inhalation of smoke causes immediate irritation to the lining of the respiratory system, including nasal passages, trachea and lungs. This can lead to inflammation, pulmonary edema and emphysema, with the severity determined by the duration of inhaled smoke (Fries *et al.*, 2008). As a result, there is disruption of respiratory epithelium leading to exudation in the terminal bronchioles and pulmonary compromise, asphyxiation and death (Jutkowitz, 2008). Hence, the present paper describes in detail about the smoke inhalation induced death in an adult cow.

CASE PRESENTATION

An adult female jersey cross bred cow was presented for post mortem examination to the Department of Veterinary Pathology, Madras Veterinary College, and Chennai-7 with a history of death due to fire accident in an apartment adjacent to the shed. The mortality of the presented cattle accompanied human casualties.

Gross examination revealed rigor mortis, highly congested mucous membranes, multiple scald wounds ranging from triangular to irregular patches were seen on epidermis of skin of pelvis region, scapula and right lateral nostril (Fig. 1). A patchy linear abrasion was noticed over the region of thoracic vertebrae. An amorphous dark black soot smeared the entire oral cavity from within. The dorsum of the tongue was swollen and appeared black in color. Internal examination of the carcass revealed striking features at the level of nasopharynx, trachea and lungs. Nasal mucosa of both the nostrils had a thick coating of black particles (soot) (Fig. 2). The tracheal mucosa was diffusely coated with an amorphous thick layer of soot extended into the bronchi and bronchioles bilaterally (Fig. 3). The terminal bronchioles were severely congested and contained oedematous grey black fluid (Fig. 4). On incision, the lung parenchyma was highly oedematous. The pericardial sac contained about 50 ml of sero-sanguineous fluid. Stray petechiae and ecchymotic hemorrhages were noticed over the epicardial surface and the coronary blood vessels were congested (Fig. 5). The other visceral organs revealed congestion. The meningeal and the cerebral vasculature were highly congested (Fig. 6).

HISTOPATHOLOGICAL EXAMINATION

Microscopical examination of the lung revealed diffuse oedema (Fig. 9) and emphysema which is in agreement with earlier reports (Fries *et al.*, 2008). Few areas revealed atelectasis. Haemorrhages were also noticed in some of the areas. Few bronchioles were lost. Increased

amount of fibrous tissue observed extending from the pleural membrane to the parenchyma. The tracheal lumen contained charred carbon particles deposited throughout the mucosal lining with few focal invasion of carbon particles into the submucosa (Fig. 7). Scald wounds revealed dermal and epidermal detachment and epidermolysis. Mild congestion and infiltration of inflammatory cells in dermis were noticed. It indicated that the animal had third degree burn. From the above pathological findings, death was ascertained to be peracute and due to asphyxia which had occurred post five minutes exposure to smoke based on the history.

CONCLUSION

In this case report, we have signified the acute adverse effects of smoke inhalation injury in a cow. Smoke inhalation produces a progressive pulmonary derangement which can ultimately prove fatal. There is a strong correlation between duration of exposure and severity of pathological injuries. The morbidity and mortality increase greatly when associated with thermal burns. In such a case, the resultant pulmonary oedema becomes more severe. In the present case, early effects of smoke inhalation were evident from gross and histopathological observations which might be due to systemic effects of carbon monoxide, methaemoglobin, cyanide and various irritant gases.

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Fig. 1–Jersey cross cow - Multiple scald wounds were noticed.



Fig. 2 - Jersey cross cow- Nasal mucosa of both the nostrils had a thick coating of black particles (soot)



Fig. 3 - Jersey cross cow - The tracheal mucosa was diffusely coated with an amorphous thick layer of soot



Fig. 4 - Jersey cross cow - The terminal bronchioles were severely congested and contained edematous greyish black fluid

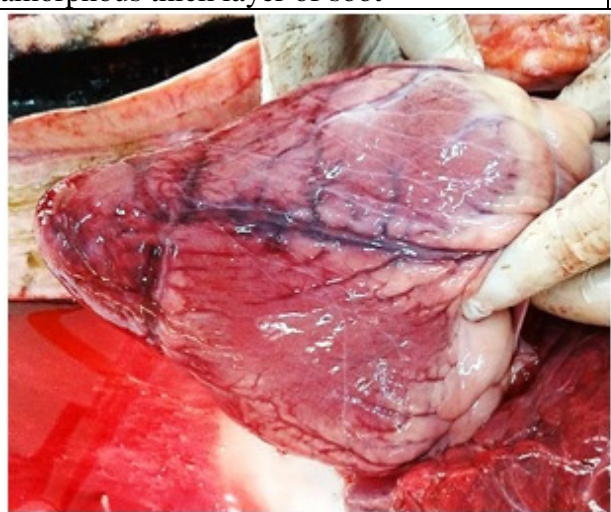


Fig. 5 - Jersey cross cow – Epicardium revealed petechiae and ecchymotic hemorrhages and the epicardial vessels were congested



Fig. 6 - Jersey cross cow - Highly congested meningeal and cerebral blood vessels

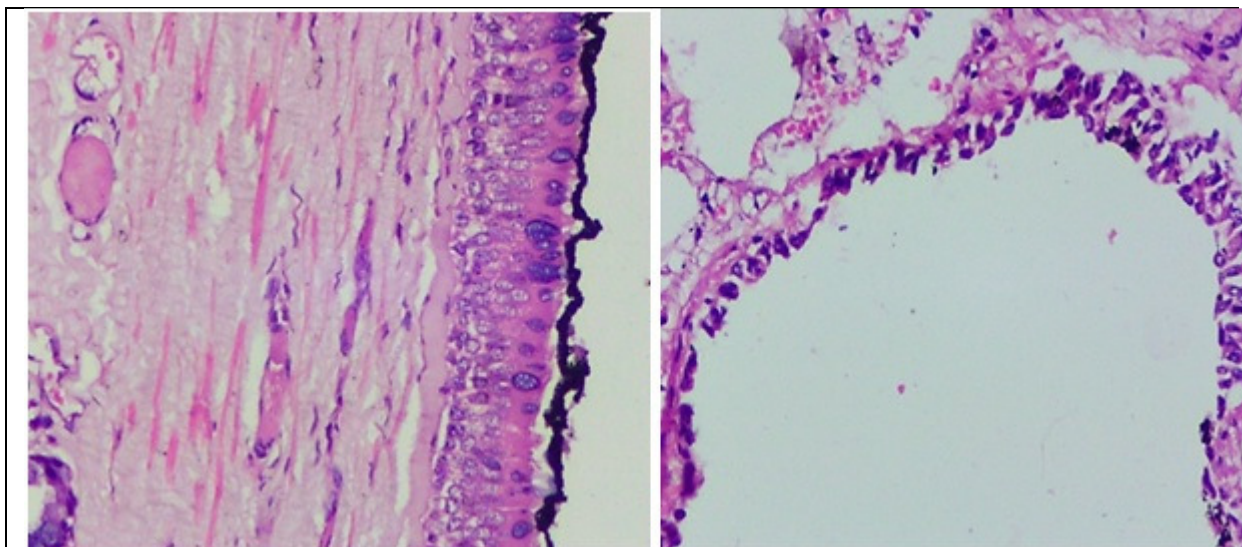


Fig. 7 - Jersey cross cow- Tracheal lumen contained charred carbon particles deposited throughout the mucosal lining. H & E x 40X

Fig. 8 - Jersey cross cow- Lung – Pulmonary emphysema. H & E x 40X