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Fire Related Respiratory Injury

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Abstract

Background: Fire-related respiratory injury is a complex clinical condition caused by thermal trauma to the respiratory tract, chemical irritation from combustion products, and systemic toxicity from hazardous gases. This injury is a critical predictor of mortality in burn victims, increasing the risk of death by up to 24-fold. Despite its severity, disaster management often faces significant challenges, including delayed rescue services, limited access to medical supplies, and initial assessments by medical personnel who may lack specific experience in treating burn and inhalation trauma.

Methods: This study was conducted as a comprehensive literature review by searching and selecting relevant, up-to-date academic sources from databases such as Google Scholar, Scopus, and PubMed. The selection focused on scientific journals, textbooks, and articles published within the last five years. Each source was evaluated for quality and relevance, then organized into key themes to provide a comprehensive perspective on the etiology, pathogenesis, and management of fire-related respiratory injuries.

Results: The findings indicate that inhaled particles reaching the terminal bronchioles trigger intense inflammatory reactions, leading to mucosal edema, decreased alveolar surfactant activity, bronchospasm, and pulmonary edema. Pathophysiological changes also include the release of chemical mediators and reactive oxygen species (ROS) that increase vascular permeability and cause widespread tissue hypoxia. Assessment relies on clinical indicators such as facial burns, carbonaceous sputum, and hoarseness. While initial thoracic radiographs may appear normal, diagnostic tools like fiberoptic bronchoscopy and CT scans are essential to determine the severity of the injury and identify distal airway damage.

Conclusion: Fire-related respiratory injury remains a leading cause of fire-related morbidity and mortality. Prompt initial assessment using primary and secondary survey, followed by immediate on-site stabilization and prioritized hospital referral, is essential. Failure to provide immediate and appropriate treatment—including airway management and fluid resuscitation—frequently results in severe complications such as acute respiratory distress syndrome (ARDS) and respiratory failure requiring prolonged ventilator support.

Keywords: Fire-related respiratory injury.

1. Introduction

The 9/11 incident that occurred in New York, United States is remembered as one of the most terrible events. At that time, a group of terrorists collapsed the twin towers of the World Trade Center (WTC) on September 11, 2001. This event resulted in Fire-related respiratory injury or inhalation injury by exposure to heat and caused death. Fire-related respiratory injury increases mortality by 24 times and risk factors that increase morbidity and mortality.[1] Research by R.H. El-Helbawy and F.M. Ghareeb in Egypt in 2011 found that the mortality rate in people with inhalation trauma was around 41.5%.[2] A study in Tokyo evaluated the impact of inhalation trauma on burn mortality and found a mortality rate of about 33.6%, so they concluded that inhalation trauma was the main predictor of burn mortality in Tokyo.[3]

World Health Organization (WHO) data in 2008 shows that the mortality rate in burns in Southeast Asian countries is the highest in the world compared to other countries. The mortality rate increased to 184,000 per year, with a mortality rate of about 11.6 per 100,000 population and a global mortality rate of 59%.[4] Research at Ciptomangunkusumo Hospital Jakarta stated that the average hospitalization victim with inhalation trauma for 15 days caused a mortality rate to reach 25.8%. Inhalation injuries significantly increase the mortality rate. Half of all fire deaths are caused by smoke inhalation.[5]

Problems encountered during disasters such as inefficient services in disaster situations, including delayed rescue and transportation services, poor access to medical supplies, poor initial assessment by doctors inexperienced in treating burn victims, and lack of medical resources.[2] Based on this background, the author is interested in discussing Fire-related respiratory injury, which is expected to be used as knowledge to better understand the etiology, pathogenesis, immunopathogenesis, signs and symptoms, pre-hospital and hospital management and its complications.

METHOD

This method of writing literature studies is carried out through searching and selecting relevant and up-to-date literature sources from various academic databases such as Google Scholar, Scopus, and PubMed. The sources used include scientific journals, books, and articles published in the last five years. Each selected source is evaluated based on its quality and relevance to the research topic. The authors then organize the literature obtained into key themes related to the study topic, analyze, and compare existing findings to provide a comprehensive perspective. In the preparation of this reference, the author also ensures the use of citations in accordance with the standards set by the journal to be published, as well as referring to the applicable writing format to ensure conformity with scientific publication guidelines.

RESULTS

Definition and etiology

Fire-related respiratory injury is an inhalation injury caused by inhalation of heat, toxic gases or irritants that cause damage to the airways and lung parenchyma.[6,16] Inhalation injuries caused by exposure to heat and inhalation of toxic products of imperfect combustion. Inhalation injuries are classified into.[3]

a. Thermal injury to the upper respiratory tract
b. Chemical irritation and injury to the respiratory tract

c. Systemic toxicity due to exposure to hazardous gases. Some of the toxic compounds present in smoke are carbon monoxide (CO), hydrogen cyanide (HCN), phosgene, ammonia, sulfur dioxide, hydrogen sulfide (HS), formaldehyde, and acrylonitrile.[1]

Pathophysiologists

Inhalation injuries from fire are the result of a combination of direct exposure, systemic effects of inhaled toxins, endobronchial debris buildup and secondary infections. Inhalation injuries will result in the victim breathing through the mouth due to nasopharyngeal irritation.[7] Particles larger than 10 μ are retained in the nasopharynx, but particles 1–2 μ in size can enter the alveoli.[1] Small particles and chemicals contained in the smoke can reach the terminal bronchioles, the inflammatory reaction will cause bronchospasm and edema. Chemical irritants cause damage to epithelial cells in the airways, mucosa edema, decrease the activity of alveolar surfactants, cause bronchospasm, obstruction of airflow, atelectasis, pulmonary edema and change the properties of proteins.[3]

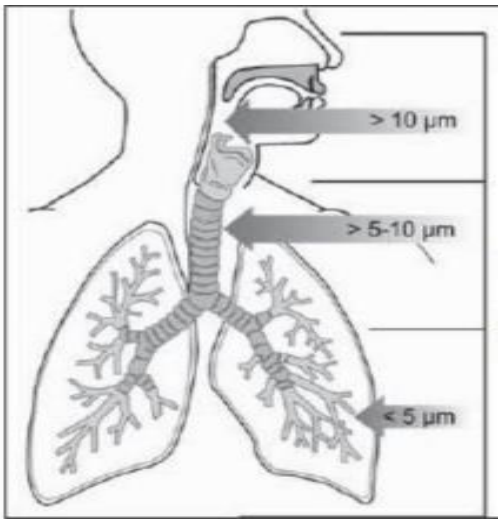

Figure 1. Distribution of irritant gases and injury sites, particle size and solubility in water

Quoted from (14)

Disorders of the ciliary mucosa and exfoliation of the bronchial columnar epithelium, thus causing obstruction of the airway and alveoli. This bronchial blockage increases the risk of infection in the lungs. The toxic chemicals present in the smoke lower the levels of surfactants, resulting in widespread microatelectasis and ventilation-perfusion mismatches. The irritants found in the smoke stimulate sensory nerve endings in the tracheobronchial, release neuropeptides that cause bronchoconstriction and activate nitric oxide synthase (NOS).[1]

Immunopathogenesis

Heat injury will result in the release of chemical mediators such as substance P, calcitonin gene-related peptides, eicosanoids, nerve endopeptidase, and interleukin8 (IL8), which attract polymorphonuclear cells and release proteases. The inflammatory reaction that occurs results in bronchial blood circulation increasing 10-20 times within 3 hours after inhaling smoke and becoming hyperemic. This increased bronchial blood flow leads to airway edema, fluid exudation, and the flow of inflammatory mediators. Circulating inflammatory mediators increase bronchial blood vessel permeability and pulmonary transvascular fluid flow and pulmonary edema.[6]

Heat destroys the epithelial layer, alters the properties of proteins and activates a cascade of complements that lead to the release of histamine and the formation of xanthine oxidase. This enzyme catalyzes the breakdown of purine into uric acid and releases reactive oxygen species (ROS) such as superoxide as described in figure 1. Superoxide is a highly reactive molecule, which is physiologically stabilized by its formation into hydrogen peroxide catalyzed by superoxide dismutase. The formation of nitric oxide (NO) by endothelial cells is enhanced by histamine stimulation. Reactions with NO to form reactive nitrogen species (RNS), such as peroxynitrite. [8]

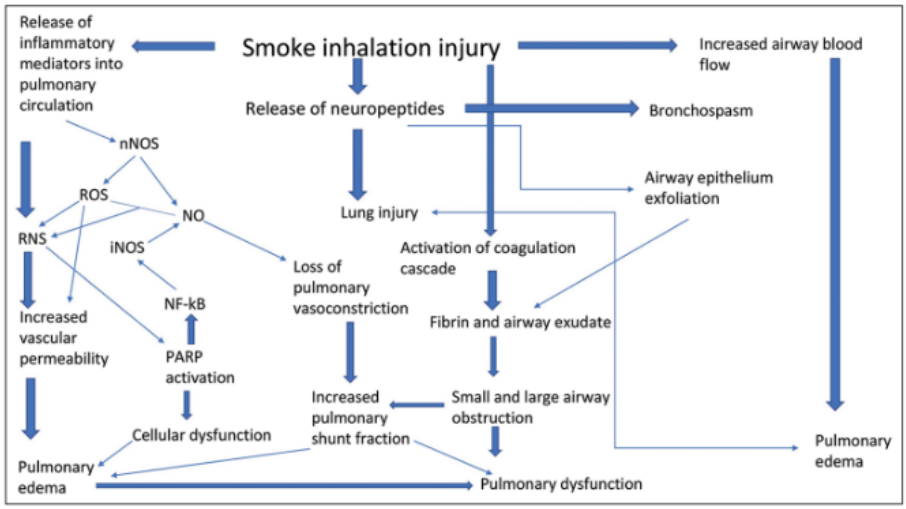

Figure 2. Pathophysiology of inhalation injuries due to heat exposure

Quoted from (3)

The ROS and RNS reactions lead to increased endothelial permeability to proteins, resulting in the formation of edema. Eikosanoids and IL-8 are released after injury, which causes the attraction of polymorphonuclear cells, which strengthens inflammatory processes, for example through the production of ROS. Activation of pulmonary C-fiber receptors by inflammation and smoke irritants causes vasodilation by increasing NO production, resulting in edema.[9]

Neutrophils are activated to release proteolytic enzymes (such as elastase), which damage the pulmonary parenchyma. Increased neutrophil infiltration also results in pulmonary interstitial edema and decreased compliance. The infarction reaction will trigger an increase in inflammatory mediators such as IL6, IL8, IL10, and tumor necrosis factor (TNF) alpha. The enzyme polyADP-ribose polymerase (PARP) is activated, thereby depleting ATP, resulting in cell dysfunction and apoptosis. Increased production of NO inhibits pulmonary vasoconstriction, hypoxia resulting from loss of airspace, increasing the risk of developing into acute respiratory failure.[10]

Toxic gases

Carbon monoxide (CO) and hydrogen cyanide (CN) cause hazardous gas injuries. Carbon monoxide is an odorless, colorless gas, having an affinity to hemoglobin (Hb) 200 times higher than oxygen (O). Severe carbon monoxide poisoning causes hypoxia in the brain and impaired consciousness.[7] Asphyxia due to poisoning (CO) is the leading cause of death due to inhalation injuries in fires. Incomplete combustion dominates the production of CO that will compete with oxygen to bind to Hb. The lower the partial pressure of oxygen (pO), the greater the success of CO in Hb binding. [1,17]

CO binding shifts the dissociation curve of oxyhemoglobin (O Hb) to the left as shown in figure 2,

increasing the affinity of O2 to Hb binding, thus, greater tissue hypoxia will occur and oxygen delivery to organs of the body such as the brain and heart which have high levels of O2 use.[1] The binding of CO and intracellular cytochrome causes mitochondrial oxidative stress and membrane damage due to lipid peroxidation, the effect of which is extensive asphyxia.[7.18]

Cyanide (CN) is a colorless gas with a bitter almond-like odor, but CN is difficult to detect at the scene of a fire.[6] Cyanide is abundant in plastics. Cyanide binds to mitochondria, interferes with oxidative phosphorylation, causes tissue damage and leads to hypoxia.[3] CN poisoning inhibits mitochondrial cytochrome C oxidase, a component of the respiratory chain, thereby inhibiting oxidative phosphorylation and causing tissue hypoxia due to the inability to use the O2 that delivers.[1]

CN poisoning affects the central nervous system, respiratory system, and cardiovascular system, comparable to the concentration of inhaled cyanide.[1] The amount of fatal CN varies from 1 to 5 mg/l. Davies and colleagues described a concentration of CN in smoke as 250 ppm, which drops to below 10 ppm by the 8th minute. Short-term exposure limits (15 ppm), as well as short-term lethal concentrations (350 ppm) were not exceeded.[8.19]

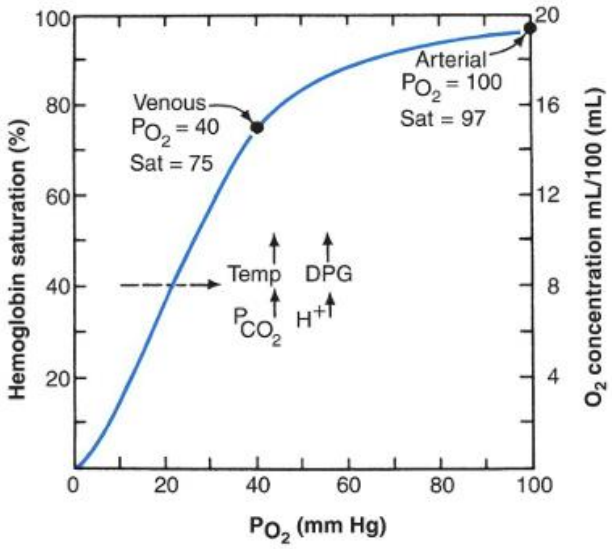

Figure 3. Oxygen dissociation curve

Quoted from (11)

Clinical symptoms

The clinical symptoms of CO toxicity often vary depending on the concentration of COHb, duration of exposure, and the morbidity of the victim. Saturation of oxygen, pulse and arterial blood gas may be normal at the beginning of treatment because it cannot distinguish between O Hb and COHb.[1] Clinical symptoms mainly involve neurological and cardiovascular manifestations. Masimo set 57 is one of the non-invasive tools that can be used to measure COHb levels.[7]

Symptoms of CN poisoning vary from tachycardia, tachypnea, dyspnea, drowsiness, and headache at low concentrations to cardiac arrhythmias, hypotension, seizures, paralysis, cardiorespiratory collapse, and coma at high concentrations (>100 ppm) on blood tests found increased lactate levels and metabolic acidosis. The level of cyanide in the blood can be measured to ensure exposure to CN.[1,20] Diagnosis of inhalation injury based on:[3]

a. History of fires in enclosed spaces
b. Burns on the face
c. Scorched nose hair
d. Soot in the mouth or nose
e. Hoarseness
f. Carbon-containing phlegm
Expiratory wheezing.
g. Upper airway edema can occur quickly, but airway edema is clinically visible after 24 hours.

Edema can also be seen on the nose, posterior pharynx and larynx. Patients may complain of hoarseness and difficulty speaking with more severe laryngeal injuries and sometimes stridor on physical examination. Careful auscultation of the lungs is necessary to detect stridor, wheezing and ronki. Headaches and balance disturbances accompanied by chest pain and vomiting can indicate shimmic poisoning, such as cyanide or hydrogen sulfide. Victims who are unconscious and found in an enclosed space should be thought to receive longer inhalation exposure than conscious victims due to unprotected airways and concentrated exposure.[14,21]

Pre-hospital management

The initial assessment of burn victims is divided into primary surveys and secondary surveys. Primary airways breathing circulation (ABC) surveys should be assessed and stabilized.[3.15]

a. Airways: ensuring a patented airway without any obstructions or foreign objects.[2]
b. Breathing: checking for upper airway disorders. Shortness of breath, difficulty breathing, stridor, and coughing. A 100% oxygen mask must be installed immediately from the site of injury to the hospital due to possible carbon monoxide poisoning.[3]
c. Ciculation: monitoring of oxygen saturation with oximetry, pulse, and blood pressure should be done regularly.[8]

Airway management is very important, as the airways swell rapidly, even though the victim can speak and initially does not experience shortness of breath. The victim should be examined for signs of inhalation injuries, such as burns on the face, carbonaceous phlegm, charred nose hair, and soot in the mouth. Evidence of airway disorders accompanied by swelling of the neck, burns in the mouth, or wheezing, intubation should be performed. A secondary, directed history should be obtained from the victim and emergency medical service personnel. These include exposure to burns of the agent, involvement of chemicals, duration of exposure, exposure to fire in an open or enclosed space, the presence or absence of an explosion, other trauma, and loss of consciousness. [3]

Airway edema so as to indicate direct bronchoscopy should be performed using topical anesthesia. The airway appears normal, intubation may be delayed, but requires close observation or airway re-evaluation if the condition worsens.[3] Early intubation is considered in conditions

1. Victims with extensive burns (i.e., involving> 40% of the total body surface area) if there is no inhalation injury due to the formation of edema can develop into airway disturbance or occlusion within minutes to hours.[3]
2. The victim with heat and smoke inhalation injuries combined with extensive burns to the face or neck.[7]
3. Victims with smoke inhalation injuries but no burns of the face or neck can be closely observed and then intubated, if conditions worsen.[3]

Tracheal intubation is performed using topical anesthesia with conscious victim and moderate sedation.[3] Prophylactic endotracheal intubation is generally not recommended in individual victims and depends on the technical skills of the physician or paramedic. It is important to consider that airway management will deteriorate over time. In cases of mouth burns without inhalation injuries, an early secured airway is the safest approach.[7] Victims with moderate to severe burns must be immediately taken to a complete health facility within 24 hours.[2]

Management in hospitals

Hospital management carried out examinations that included complete blood counts, serum electrolytes, creatinine, arterial blood gas analysis, including carboxyhemoglobin levels, thoracic and electrocardiogram photos, fire-specific toxicology examinations such as blood CN and CO.[1]

a. Thoracic photo

Thoracic photographs are taken upon arrival at the hospital to be used as a basis to see the progression of smoke inhalation injuries.[2] Thoracic photographs initially appear normal, but later may reveal atelectasis, consolidation, and/or pulmonary edema.[3] Figure 3 describes the condition when he was admitted to the hospital, he had a cough but there was no hoarseness, stridor, wheezing, or signs of burns on his face. The thoracic photograph results showed no abnormalities. Fiber optic bronchoscopy showed a slight soot but no edema or erythema in the larynx. The patient experienced dyspnea on day 2.[3]

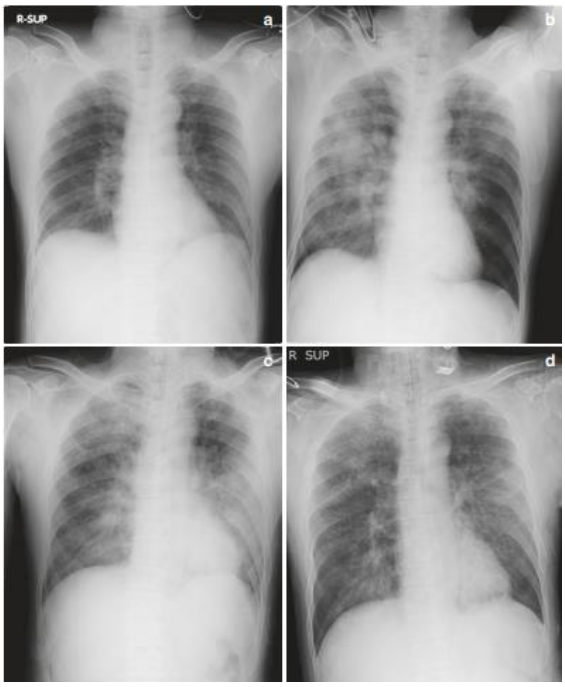

Figure 4. Serial thoracic photograph of a patient with thermal injury.

Quoted from (3)

The diagnosis of smoke inhalation injury without thermal injury is established. The patient was intubated on day 3 showing bilateral infiltrative shadow. The infiltrative shadow seen on the thoracic photograph begins to disappear on day 5. The patient was successfully extubated on day 15, showing clean lungs, except for inflammatory lesions in the left middle lung field on day 19 shown in figure 4.[3]

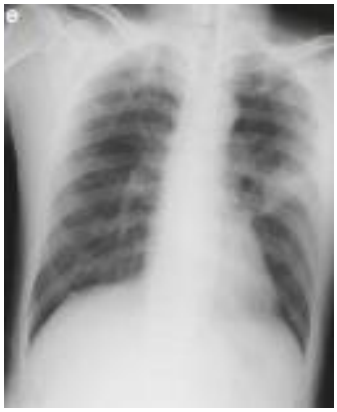

Figure 5. Thoracic photo of day 19

Quoted from (3)

b. CT Scan toraks

Chest computed tomography on day 3 showed bilateral infiltrative shadowing, ground-glass cloudiness, and bronchial stenosis. Thoracic CT scan examination shows bilateral infiltrative shadow, ground-glass opacity in peribronchial distribution, and bronchial stenosis within a few hours after inhalation injury and can identify the extent of the injury to the distal airway.[3]

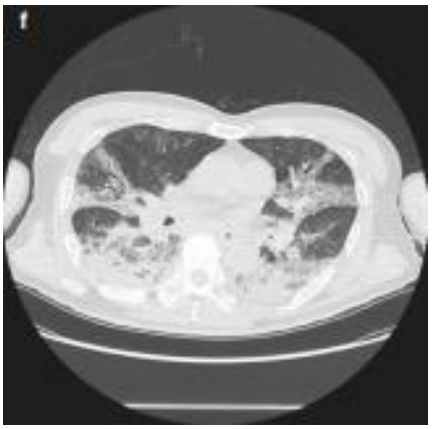

Figure 6. CT scan on day 3 post thermal injury.

Quoted from (3)

c. Bronchoscopy

Fiber optic bronchoscopy provides direct information about the entire respiratory system. Bronchoscopy in addition to its diagnostic function is also therapeutic and investigative used to determine the severity of inhalation injuries. Severity of inhalation injuries:[2]

1. Degree I: no laryngeal edema
2. Degree II: laryngeal edema and minimal erythema
3. Degree III: mild mucosal edema of the trachea and erythema
4. Degree IV: moderate mucosal edema of the trachea and erythema
5. Degree V: severe tracheal edema and erythema

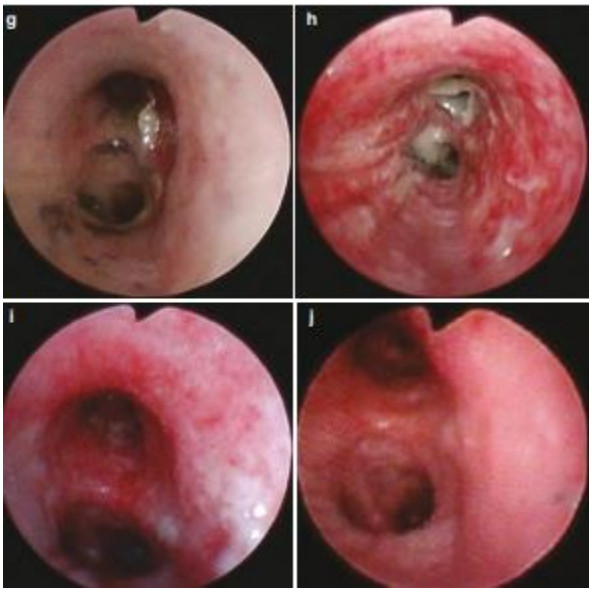

Figure 7. Bronchoscopy series of day one (g), day three (h), day five (i) and day twelfth (j).
Quoted from (12)

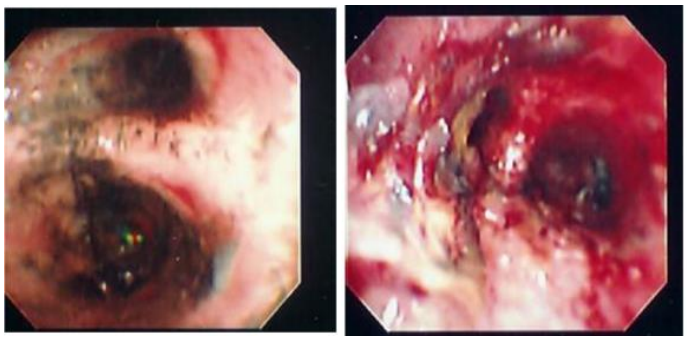

Figure 8. Bronchoscopy in H-0 and H-4 inhalation trauma patients
Quoted from (12)

Bronchoscopy is performed on a thermally injured patient to evaluate the condition of the patient. Figure 7 shows a series picture of the first day bronchoscopy showing some soot but no edema or erythema in the larynx in fig. g. Bronchoscopy on day 3 in figure h, day 5 in picture i, and day 12 in picture j showed mucosal edema, exfoliation of necrotic tissue. endobronchial fragments, and complete degeneration of the bronchial mucosa. Fiber optic bronchoscopy on day 12 (j) showed healing of bronchial mucosal lesions.[12]

The victim's problem is intense pain, which can lead to neurogenic shock and fluid loss, which can lead to hypovolemic shock. Liquid therapy with Ringer acetate 500 cc, with Evans formula: 2 cc × body weight (kg) × percentage of burns.[2] The Baxter or Parkland formulas are commonly used for resuscitation guidelines for burn victims.[3] Calculation of fluid balance with urinary catheter and record urine, which should be 1 cc/kg body weight/hour. Burns of more than 50% of the total surface area of the body are central venous (Swan-Ganz catheter) for continuous measurement of central venous pressure.[2]

Inhalation injuries are given a 100% oxygen mask installed continuously.[2] Early intubation should be done with a fiberoptic bronchoscope, which is a prudent course of action before airway edema occurs. Bronchial hygiene is maintained, which includes therapeutic coughing, chest physiotherapy, deep breathing exercises, and early ambulation. Pharmacological therapies such as beta-2 agonists, epinephrine

rasape, N-acetyl cysteine, and aerosol heparin are used to improve lung oxygenation.[13]

Inhalation injury treatments include moisturized oxygen, intubation and ventilation, bronchodilators, and bronchial toilets. Bronchoscopy at the subacute stage can detect tracheobronchial mucosal necrosis and hemorrhagic tracheobronchitis. Therapeutic bronchoscopy should be performed to alleviate airway obstruction caused by necrotic remains of the tracheobronchial. Half of the victims with inhalation injuries will develop a lung infection. Victims who experience fever and discoloration of sputum must be treated with antibiotics according to the results of sputum cultures. About half of the burn victims who were intubated developed ARDS. Fluid resuscitation should be done with caution, to avoid exacerbating pulmonary edema and ARDS. The treatment of victims with severe burns involves intensive care over a long period of time.[3]

Complications

Victims of inhalation injuries by heat exposure can develop acute respiratory distress syndrome and respiratory failure that require ventilator support. Risk of ventilator-related complications such as barotrauma and pneumonia. Complications of infection such as tracheobronchitis, bronchiectasis, bronchiolitis obliterans, and pneumonia can develop in 38%–60% of victims, after 3–10 days of smoke inhalation injury, and are associated with a mortality rate of up to 60%.[1]

The airways can remain hyperreactive for up to 6 months after extubation. Damage to the larynx from inhaled toxins or prolonged intubation can lead to hoarseness or long-term dysphonia. Injury to the upper airway epithelium from an initial injury can lead to a tracheoesophageal fistula, tracheomamalasia, advanced subglottic stenosis, or tracheobronchial polyps. Smoke inhalation injuries can lead to restrictive ventilation dysfunction, obstructive ventilation dysfunction, and decreased diffusion capacity, which can persist for months.[1]

Conclusion

1. Fire-related respiratory injury is an inhalation injury caused by inhalation of heat, toxic gases or irritants that cause damage to the airways and lung parenchyma.
2. Heat injury will result in the release of chemical mediators such as substance P, calcitonin gene-related peptides, eicosanoids, nerve endopeptidase, and interleukin8 (IL8), which attract polymorphonuclear cells and release proteases.
3. Diagnosis of inhalation injury based on a history of fire in an enclosed space, burns on the face, scorched nasal hair, soot in the mouth or nose, hoarseness, carbon-containing phlegm, expiratory wheezing and upper airway edema.
4. The initial assessment of burn victims was divided into primary and secondary surveys and continued with hospital management
5. Complications of inhalation injuries can include acute respiratory distress syndrome and respiratory failure requiring ventilator support.

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Conflict Of Interest

None.

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