Labelling-Based Information Management Systems in Disasters and Emergencies

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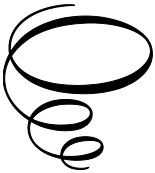
Edited by

Mehmet Sinan Başar

Cambridge Scholars Publishing



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This book first published 2022

Cambridge Scholars Publishing

Lady Stephenson Library, Newcastle upon Tyne, NE6 2PA, UK

British Library Cataloguing in Publication Data
A catalogue record for this book is available from the British Library

ISBN (10): 1-5275-8232-9
ISBN (13): 978-1-5275-8232-3

TABLE OF CONTENTS

Contributors vii

Chapter One 1
Traumatic Injuries in Disasters
Ayça Çalbay

Chapter Two 20
General Characteristics of Disasters
Gülşen Çiğşar

Chapter Three 36
Special Groups in Disasters
Atif Bayramoğlu and Fatma Çakmak

Chapter Four 50
Infectious Diseases and Water Supply in Disasters
Handan Çiftçi and Murat Özdemir

Chapter Five 64
Neuropsychiatrical Disorders in Disasters
Mehmet Nuri Koçak and Fatma Çakmak

Chapter Six 77
Information Needed about Affected People in Emergency and Disaster Management
Ali Utku Şahin and Kemal Koçak

Chapter Seven 87
Artificial Intelligence and Data Analysis in Disasters
Hilal Kübra Sağlam

Chapter Eight 105
Artificial Intelligence Applications in Disaster and Emergency Management
Emrah Şimşek and Emre Çintaş

Chapter Nine.....130
System Analysis and Design
Sinan Kul

Chapter Ten 150
Web and Mobıle based Dısaster Management Systems
Ferhat Bozkurt

Chapter Eleven 168
Use of Geographic Information Systems in Disasters
Fatih Alaeddinoğlu

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CHAPTER ONE

TRAUMATIC INJURIES IN DISASTERS

AYÇA ÇALBAY

Introduction

Disasters are events that stop or interrupt normal life for a certain period of time. They can be of natural, technological or human origin. The cause of the disaster is not the event, but the consequences are after the event. The consequences are at a level that society cannot handle, causing physical, economic or social losses (Afet & Acil Durum Yönetimi Başkanlığı, 2021).

Disaster types are categorized under five main headings: geological, climatic, biological, social and technological disasters. Earthquakes, landslides, tsunamis, tornados, cold waves, fire, epidemics, mine accidents, wars, and CBRN (chemical, biological, radioactive, nuclear) events are destructive disaster types that cause large masses to be affected.

As with all trauma patients, an "ABCDE" evaluation of a disaster victim should be made and the patient should be examined from top to toe. Before these procedures, the appropriate triage should be done. If needed, decontamination should be provided. After the patients are stripped, precautions should be taken to protect the patient from hypo/hyperthermia and dehydration. Resuscitation interventions for life-threatening injuries should be initiated without delay.

In this section, life-threatening traumatic injuries that are frequently observed in disasters will be mentioned.

Crush Injury/Crush Syndrome

Crush injuries occur when the trunk, extremities, or any part of the body is exposed to an external crushing force. Compression caused by the force applied creates a direct trauma in the tissue. In major earthquakes, 3-20% of those patients injured by the collapse of buildings have crush injuries (Bartels, S.A. & VanRoyen, M.J., 2012). Crush injuries with traumatic

asphyxia can also be observed in traumatic injuries where large masses are affected (de Almeida, M.M. & von Schreeb, J., Human Stampedes, 2019).

Impaired perfusion of injured tissue leads to the development of ischemia. Sequelae that occur after tissue reperfusion also cause crush injuries. Systemic symptoms occurring after crush injury may result in organ dysfunction and death. This condition is defined as crush syndrome (Slater, M.S. & Mullins, R.J., 1998; Oda, J., Tanaka, H. & Yoshioka, T., 1997; Peiris, D., 2017). After the crushing force is removed, muscle damage and swelling occur with possible muscle necrosis and neurological dysfunction at the injury site. The compressive force that occurs with compression causes direct tissue damage by preventing venous outflow. Cellular death due to prolonged compression can lead to myonecrosis and crush syndrome. Potassium, phosphorus, and myoglobin released from the injury site within 20 minutes of recovery can cause ventricular fibrillation and sudden death. This situation is called "smiling death". While the patient rejoices that he has survived, he faces death. Death together with acute renal failure due to crush syndrome has been observed many times after major earthquakes (Peiris).

The most common presentation is acute renal failure. In order to prevent this situation, clinicians should quickly recognize the crush injury and start appropriate fluid replacement as soon as possible in a short time interval. Crush syndrome can also develop after prolonged immobility, burns and electrical injuries. It is the second direct cause of death after trauma in earthquakes.

Rescue and Field management

Crush injuries are often trapped in the wreck field. In cases where the number of people affected is relatively small, victims are evacuated with the help of lay rescuers or trained professional rescuers. If there is an incident that affects large masses, a rescue team suitable for the countries' own disaster programs is used for rescue.

The rescue time is related to the earthquake-related mortality rate. When victims are affected for >24 hours, the risk of death increases significantly (Macintyre, A.G., Barbera, J.A. & Smith, E.R., 2006). Survivors are those found in spaces in collapsed structures. Children and older people are more vulnerable. There is a possibility that collapsed structures will collapse again. In this respect, rescuers should be very careful during interventions.

Confined Space Medicine

Tunneling and support debris are required for the rescue of survivors. In this case, the intervention and transfer relating to the patient will not be in the form of the usual container. For the same reason, the first intervention of patients with crush injuries will be done by rescuers inside the collapsed structure. Interventions made in the current situation are called confined space medicine (Petinaux, B., Macintyr, A.G. & Barbera, J.A., 2014). Providing patient care in such areas is dangerous and difficult. It is necessary to wear the necessary equipment and make preparations to manage and protect dangerous situations such as dust, excessive temperature, dangerous substances and gas, explosion, fire, and the possibility of re-collapse. If the crush injury is caused by non-earthquake situations, such as bombing or mine collapse, it is also necessary to protect victims from blast injury, fire and inhalation of toxic agents.

Airway Support, Breathing, and Circulation

A crush injury in the thorax is a major cause of respiratory failure and associated death. Traumatic asphyxia, irritant and toxic gases and respiratory tract irritation and damage may cause an advanced airway and sometimes surgical airway requirement.

During rescue procedures, if the patient has injuries such as pneumothorax, hemothorax, flail chest, or pulmonary contusion along with the crush injury, a chest decompression may be required (needle/finger decompression, tube thoracostomy).

Hyponatremia may be observed in patients who are under a weight for a long time. Dehydration is the most important and fatal cause of hypovolemia. Bleeding and burns are other causes of hypovolemia. Bleeding from the trauma itself may not manifest itself as long as the injury site is under pressure. Findings can be preserved until the moment of recovery.

The first intervention in preventing deaths due to hypovolemia and crush injury is fast access to isotonic solutions and at least one large lumen vein. Fluids containing K should be avoided while performing fluid replacement to the patient. Adequate and rapid fluid resuscitation will protect the patient from crush syndrome. The observation of urine output in the patient is an important evaluation criterion in terms of patient monitoring, if long-term field care is provided to the victim. For this, equipped field rescuers use algorithms to prevent acute kidney injury. Initially, the patient is given a bolus of 1000 ml/hour of normal saline solution intravenously within 2

hours. Afterwards, maintenance treatment is started at a dose of 500 ml/hour. If the patient has known heart failure, kidney failure, or chronic obstructive pulmonary disease, hydration is planned at low doses such as 10 cc/kg.

Extreme Reperfusion and Hyperkalemia

A crushed extremity may have mild symptoms after the overwhelming force is gone. There may be only erythematous findings. Or it may be a mottled appearance due to ecchymosis, ischemia. K release from crushed muscles and necrotized tissues may cause rapid hyperkalemia and ventricular fibrillation in the patient. Application of iv crystalloid before the hospital is the most important protective intervention for the patient. If electrocardiography can be performed on the patient before the hospital, T spikes due to hyperkalemia and enlarged QRS waves can be detected. In this case, Ca gluconate/Ca chloride treatment can be given to the patient. Inhaled albuterol therapy and iv insulin therapy are other treatment options used in the treatment of hyperkalemia.

It is not recommended to apply a tourniquet to prevent the release of K and other cellular contents from the extremity. There is no evidence level showing the usefulness of this method. Tourniquet application is only used in extremity bleeding that cannot be stopped by pressure. Fasciotomy at the scene is not recommended due to the high risk of infection (Sever, M.S. & Vanholder, R., 2013). It has been observed that the amputation of severely crushed parts or limbs at the scene does not reduce the risk of crush syndrome, but it does increase the risk of stump infection in survivors.

Field Amputation

When the extremities under load cannot be removed from the wreck, field amputation is performed as a last solution. Manual or electric saws are used for this, considering the available range of motion, resources, and presence of explosive risk. Some rescue teams have a trained emergency surgery team that can perform this intervention. If possible, this team is enabled to reach the patient. For the intervention, a tourniquet is applied and a guillotine amputation is applied from the distal of the injury. Safe drugs that can be used indoors are preferred for anesthesia and analgesia (Macintyre, A., Kramer, E.B. & Petinaux, B., 2012).

Extremity Injuries

As in many types of injuries, extremities are among the most frequently injured body parts in disasters. Injuries can be in the form of simple soft tissue damage or require a multidisciplinary approach that requires more serious interventions. Amputation, brachial plexus injury, long bone fractures that can cause serious blood loss, open fractures and associated fat emboli are the injuries of crush syndrome. It should be kept in mind that casualties with this type of serious injury may have other injuries that often involve other parts of the body.

The presence of bleeding/open wounds at the injury site, vascular/nerve injury and circulatory disturbance in the affected area should be evaluated. In the presence of bleeding, direct pressure should be placed on the bleeding area. In the presence of vascular injury or amputation, a tourniquet can be applied just proximal to the injury site. If a tourniquet is applied to the injured person, they should be followed closely. The hour and minute that the tourniquet is applied should be noted. Blood flow should be provided to the distal of the intervention area by loosening the tourniquet at intervals of 10-15 minutes. In this way, oxygenation of the tissue is attempted to be achieved.

Deformity, tissue loss and limitation of movement observed in an extremity may be symptoms of fractures that can occur in bones and joints. If there is blood mixed with oil droplets at the injury site, this may be an indication of an open fracture. In this case, the injured area should be cleaned so that it does not contain any foreign objects and covered with a moist sterile dressing until the debridement is completed. Tetanus prophylaxis and early antibiotherapy will reduce the risk of infection.

The area with the possibility of fracture should be fixed so as to include one lower and one upper joint. Compartment syndrome should be kept in mind in order not to miss the diagnosis. Early fasciotomy can prevent further increases in compartment pressure that can lead to muscle necrosis and neuropathy. Findings suggestive of compartment syndrome include disproportionate or severe pain, pain caused by passive elongation of the muscle, and stiffness of subfascial tissues. Measuring the compartment pressure will be helpful in diagnosis.

Acute Compartment Syndrome

This is characterized by increased pressure in extremity sections covered by the fascia and the abdomen. For this reason, it is examined under two headings: abdominal and extremity compartment syndrome. Capillary

perfusion is impaired in extremity, and bone tissue may also be affected. Increased pressure may affect the respiratory cardiovascular system and renal system, causing a multisystemic picture.

Extremity Compartment Syndrome

The muscle groups surrounding the bones, vessels and nerve packages are divided into sections and compartments with strong and rigid membranes. Compartment syndrome occurs due to the increase in pressure within these compartments for any reason. The increased pressure will disrupt the circulation of the tissues as well as cause their functions to be impaired. The most common cause of acute occurrence is trauma. Acute compartment syndrome (ACS) is an emergency requiring an emergency surgery indication.

Epidemiology and Risk factors: ACS is seen especially with trauma accompanied by long bone fractures (Shore, B.J., Glotzbecker, M.P. & Zurakowski, D., 2013; Park, S., Ahn, J. & Gee, A.O., 2009). Compartment syndrome is mentioned in any situation that decreases the volume capacity of a compartment or when the volume increases, causing the compartment volume to increase. The most common regions are the legs and forearms (Park, S. et al.). In addition, the foot, thigh, and gluteal region are other areas where it is seen.

Its incidence is higher under the age of 35. Especially, it is more common in tibia diaphysis and distal radius fractures that have more muscle mass and have completed their growth period (Shore, B.J. et al., McQueen, M.M., Gaston, P. & Court-Brown, C.M., 2000). Fractures constitute approximately 75% of the cases (Shore, B.J. et al.; Patel, R.V. & Haddad, F.S., 2005). The risk is higher in fragmented fractures.

Among the traumas that can cause ACS without fracture, we can list the following:

- Severe thermal burns (pay attention to circular ones)
- Tight bandage, splint, plaster applications (pay attention to circular ones)
- Penetrating traumas
- High pressure injection
- Vascular injuries
- Animal bites and stings
- Direct trauma to the body area (e.g., crush injury).

Patients with rhabdomyolysis, including after excessive exercise, are at risk for ACS (Boland, M.R. & Heck, C., 2009; Bhalla, M.C. & Dick-Perez,

R., 2014). Victims who have bleeding diathesis, who have to use the injured, crushed body area, and who continue to use it, carry a risk in terms of ACS.

Vascular injuries, especially arterial injuries, are an important cause of ACS (Olson, S.A. & Glasgow, R.R., 2005; Suzuki, T., Moirmura, N., Kawai, K. & Sugiyama, M., 2005). Compartment internal pressure increases after arterial bleeding. Ischemia develops in the circulating muscle and becomes susceptible to reperfusion damage. Swelling due to reperfusion increases the compartment pressure even more. It has been observed that the risk of ACS is also increased in venous injuries (Modrall, J.G., Sadjadi, J. & Ali, A.T., 2004).

Pathophysiology: In all cases, the common cause is cellular anoxia due to local ischemia. The cause of ischemia is that the metabolic needs of the affected area cannot be met by the current blood flow. As the tissue pressure increases to the mean arterial pressure, muscle oxygenation decreases (Olson, S.A., et al). Hypotensive patients will have a lower tolerance to this condition.

Clinical Features: The emergence of signs and symptoms of ACS shows a gradual clinic. More specific findings have different times of emergence. The most important clues are that symptoms and signs progress rapidly within a few hours, and patients at risk have more than one finding compatible with the diagnosis. For these reasons, patient follow-up and physical examination should be done closely, in detail, and in a repetitive manner. When a tense and painful muscle compartment is identified, compartment pressure should be measured, and circulatory and neurological examinations should be performed. Findings suggestive of ACS are:

- Pain disproportionate to the injury,
- Persistent deep or burning pain,
- Paresthesias (paresthesias between 30 minutes and 2 hours are indicative of ischemic nerve damage and suggest ACS),
- Pain caused by passive stretching of the muscles in the affected compartment (early finding),
- A tense compartment that feels hard “like wood”,
- Pallor due to vascular insufficiency (not always),
- Decreased sensory sensitivity,
- Weakness of the affected muscles (starts within about two to four hours of ACS),
- Paralysis (late finding).

Although a physical examination is important in diagnosing ACS, it will be insufficient alone, and compartment pressure must be measured. Surgical consultation should be done.

Weakness and paralysis in the affected muscles are late signs of ACS and may indicate permanent damage.

If ACS is left untreated, it may cause infection, sensory defects, paralysis, muscle contracture, nonunion of the fracture and even amputation (McQueen, M.M. et al.). Rhabdomyolysis may develop due to muscle ischemia, myoglobinuria and renal insufficiency may require dialysis.

Laboratory Tests: The diagnosis of ACS is made by examination findings and the measurement of compartment pressure. Laboratory tests are not used for diagnosis. Surgical consultation of patients and measurement of compartment pressure are not delayed for laboratory tests. However, as tissue destruction due to ischemia occurs in patients, muscle enzymes increase and CK increases. Myoglobinuria can be observed 4 hours after the development of ACS.

Compartment Pressure Measurement: This is an important aid in the diagnosis of ACS. Compartment pressures can be measured to prevent unnecessary fasciotomy in patients who cannot be diagnosed fully. If necessary, patients can be followed up with repeated measurements. In cases suggesting complete ACS, fasciotomy should not be delayed by wasting time with pressure measurement.

Direct Measurement Techniques: The three most common methods are hand-held manometer (for example, the Stryker device), a simple needle manometer system, and a wick or slit manometer system. All manometers measure resistance from tissue pressure after a small amount of saline is injected into a closed compartment. Compartment pressure measurements can be made by connecting 18-gauge needles directly to the devices which measure arterial pressure in places where there is no hand monitor or measuring device. The delay that may occur due to the lack of a device is thus prevented.

If the measurement is made close to the fracture site, incorrect measurements can be made (Heckman, M.M., Whitesides Jr., T.E., Grewe, S.R. & Rooks, M.D., 1994).

Interpretation of Measurements: The pressure of a normal compartment varies between 0 and 8 mmHg (Klenerman, L., 2007). ACS findings in the affected areas vary according to the proximity of the pressure in the measured area to the systemic pressure:

- Capillary blood flow is compromised when the tissue pressure approaches the mean arterial pressure by 25 to 30 mmHg.

- Pain may develop when tissue pressures reach between 20 and 30 mmHg.
- Ischemia occurs when tissue pressures approach diastolic pressure.

Abdominal Compartment Syndrome (AbCS)

Abdominal compartment syndrome (AbCS) is a clinical syndrome characterized by intra-abdominal organ dysfunction as a result of increased intra-abdominal pressure. Its incidence is 6-14% in trauma patients and 1-20% in burn patients. Regardless of the incident that initiates AbCS, fluid extravasation into the interstitium and massive intestinal wall edema occur due to capillary leak syndrome. The fluid-holding capacity of the intestinal wall is high. In this way, liters of fluid will be sequestered in the intestinal wall and mesentery. This edema will cause a decrease in renal and intestinal perfusion and a loss of ventilatory capacity by increasing the pressure in the abdominal cavity (Carr, J.A., 2013). Although surgical decompression is considered as the only treatment option for AbCS, nonoperative medical treatments can also be used in the prevention and treatment of organ failure due to AbCS (Cheatham, M.L., Malbrain, M.L., Kirkpatrick, A., Sugrue, M., Parr, M. & De Waele, J., 2007).

Blast Injury

Some initial factors that should be known in order to be able to make an effective intervention in blast injuries are:

- Mechanism of injury
  Primary injury
  Secondary injury
  Tertiary injury
  Various
- The injury itself
- To have a command of management suitable for injuries.

Primary blast injuries are caused by the blast wave and have severe effects. Air-filled and luminal organs are more affected by these injuries. The lungs, ears, and gastrointestinal tract are the most sensitive. As the wave passes through the body, it causes damage through four mechanisms: particle, explosion, non-interaction (acceleration/deceleration) and pressure differences. The human body usually resists being thrown, but injuries occur when it hits the ground or surrounding objects.

Secondary blast injuries are caused by the acceleration of small fragments caused by the explosion. In terrorist bombings, injuries from flying glass, shrapnel and debris are responsible for most illnesses.

Tertiary injuries occur after victims hit a hard surface with the impact of the explosion.

Sudden burns caused by the intense but short-term heat of the explosion, which can reach 3000°C, and inhalation injuries due to the gas released from the bomb itself are among the other injuries that may occur.

The proximity of the casualties to the explosion, the objects around them, the type of explosive and the additional materials it contains (such as shrapnel) are factors that affect the effect of injury.

In blast injuries, the area most likely to be damaged is the head and neck. The five most common injuries in deaths are; brain injury (66%), skull fracture (51%), diffuse lung contusion (47%), tympanic membrane rupture (45%) and liver laceration (42%).

Inhalation Injury

Inhalation injuries describe the damage caused by the air, particles, chemicals, heat and smoke taken into the airway by inspiration; often used synonymously with smoke inhalation. It not only affects the lungs, but also causes systemic poisoning. The severity of the injury will depend on many factors: the size and diameter of the particles in the smoke, the duration of exposure, the content of the gas exposed, the solubility of the gases and the ignition source. Toxin damage that may occur directly depends on the low molecular weight contents of the gases, their pH, their ability to create free radical damage and their ability to reach the alveoli (Enkhbaatar, P., Pruitt Jr., B.A. & Suman, O., 2016; Demling, R.H., 1993). Depending on the localization of the damage, injuries are classified as upper airway, tracheobronchial system or lung (AC) parenchyma injuries.

Upper Airway Injury

The upper airway describes the region above the vocal cords. Injuries to this area are thermal burns caused by heat exchange in the oropharynx and nasopharynx. Acute damage is seen as erythema, ulceration and edema (Sheridan, R.L., 2016). In injuries with combined burns and inhalation damage, aggressive fluid therapy required for burn shock treatment may accelerate the formation of early edema. Burns that occur in the face and neck region can cause obstructions in the upper airway by creating external compression and deformity (Demling, R.H.). Ciliary dysfunction secondary

to the inflammatory process may increase the risk of bacterial infection. The formation of thick secretions that are difficult to remove can disrupt the gas exchange and cause the formation of plugs and atelectasis.

Tracheobronchial Injury

Injury to this area is often caused by the smoke itself and the chemicals it contains. Clinical symptoms include persistent cough, erythema, hyperemia, wheezing, and hypoventilation.

The tracheobronchial area is rich in vasomotor and sensory nerve endings. Stimulation of these areas causes neuropeptide release. Neuropeptides cause bronchoconstriction and increase vascular permeability. Inflammatory response occurs due to increased vasodilation and bronchial blood flow increases. The loss of intact bronchial epithelium and the formation of reactive oxygen species cause fluid loss from the intravascular space to the alveoli and bronchioles. Consequently, alveolar collapse occurs (Walker, P.F., Buehner, M.F. & Wood, L.A., 2015; Murakami, K. & Traber, D.L., 2003). This process is the primary cause of hypoxemia after inhalation injury. Thus, ventilation-perfusion mismatch occurs.

Parenchymal Injury

Lung parenchyma damage occurs later. This period is proportional to the severity of the damage (Rehberg, S., Maybauer, M.O. & Enkhbaatar, P., 2009). Parenchymal damage is manifested by atelectasis and alveolar collapse, leading to impaired oxygenation. Alveolar hemostasis is disturbed, fibrinolytic activity decreases, and massive fibrin accumulation occurs. This situation causes ventilation/perfusion incompatibility (Rehberg, S. et al.). This situation is exacerbated by the formation of nitric oxide and the blood flow of bronchioles with impaired oxygenation increases (Dyamenahalli, K., Garg, G. & Shupp, J.W., 2019). Airway plugs, atelectasis, activation of the inflammatory system and impaired mucociliary clearance increase the risk of pneumonia.

Systemic Toxicity

The direct systemic effects of inhalation injuries develop as a result of the burning and decomposition of burning materials. The two most common gases that increase morbidity and mortality are carbon monoxide and hydrogen cyanide (Rehberg, S. et al.).

Carbon monoxide poisoning: This is one of the most common causes of death after inhalation injury (Rehberg, S. et al.). The hemoglobin affinity of carbon monoxide is 200 times higher than that of oxygen. In the presence of carbon monoxide, oxygen release to tissues is impaired and tissues remain hypoxic. The fact that it is a colorless and odorless gas makes it difficult to distinguish.

As carboxyhemoglobin cannot be distinguished from oxyhemoglobin, the values measured by pulse oximetry have no value in the diagnosis and follow-up of the patient. Until proven otherwise, all patients with inhalation injury or fire exposure should be treated as carbon monoxide intoxication.

Hydrogen cyanide poisoning: This is a colorless gas with a bitter almond odor. The diagnosis is difficult because the symptoms that may occur in the early period are not specific and cannot reach clinically measurable levels. It is an important diagnosis that should be kept in mind with every patient coming from the fire area. Although the diagnosis is not confirmed by the laboratory, treatment for cyanide poisoning can be initiated in burn patients with loss of consciousness, cardiac arrest and cardiac decompensation.

Cold Injuries

Cold injuries vary according to exposure to heat, duration of exposure and individual factors. Frostbite, also known as a cold bite, is the prototype of cold injuries. Cold injuries seen without freezing are observed after being in wet environments. Trench foot and pernio are injuries in this group. While permanent damage to the tissues is observed in the presence of frostbite, mild but disturbing skin lesions are observed in cold injuries that are not accompanied by frostbite.

Cold Physiology

The cardiovascular response to cold is in the form of deep peripheral vasoconstriction and an increase in heart rate and blood pressure. This is followed by progressive bradycardia, hypotension and myocardial irritability. Below 32°C, the risk of arrest increases as malignant cardiac arrhythmias increase. Atrial fibrillation and flutter are expected arrhythmias (Brown, D.J., Brugger, H., Boyd, J. & Paal, P., 2012). Usually, classical Osborn J waves occur.

The response of the kidneys to hypothermia is called cold diuresis. Rhabdomyolysis is a complication that should not be forgotten. Pseudo rigor mortis may occur. Coagulopathy is an important problem especially below 34°C. Coronary and cerebral artery occlusion and disseminated

intravascular coagulation (DIC) are among the problems that can be encountered.

Frostbite

The body areas most affected by cold bites are the head, hands and feet (Ervasti, O., Juopperi, K. & Kettunen, P., 2004; DeGroot, D.W., Castellani, J.W., Williams, J.O. & Amoroso, P.J., 2003). Although most of these cases are mild (frostnip), some (12%) are severe.

Risk factors include age and gender, temperature and wind, behavioral and psychological factors (alcohol-smoking, improper dressing style, staying in the same position for a long time), and diseases (peripheral vascular disease, Raynaud's disease, atherosclerosis, DM, hypovolemia).

Freezing alone is not sufficient for tissue damage. The consequences of thawing also affect the damage that will occur. The depth of freezing in the tissue depends on the temperature, exposure time and freezing rate. Tissue sensitivity also varies. If we sort by the least damaged tissue according to the most sensitive tissue, this will be as follows: cartilage, ligament, vessels, cutis, epidermis, bone, muscle, nerves and bone marrow.

An arachidonic acid cascade is formed immediately after the tissue begins to freeze and thaw. This condition causes vasoconstriction, platelet aggregation, leukocyte collapse, and erythrostasis. Therefore, venule and arterial thrombosis, ischemia, necrosis and dry gangrene occur (Bourne, M.H., Piepkorn, M.W., Clayton, F. & Leonard, L.G., 1986). Tissue necrosis is observed secondary to cellular or vascular injury.

Affected tissue can be divided into three areas. The coagulation zone is the most severe area of exposure and is usually distal and the damage caused is irreversible. The area of hyperemia is mostly superficial, and typically proximal, and the least cellular damage is seen in this area; the injury usually resolves without treatment within <10 days. The stasis zone is the middle zone, possibly where reversible severe cell damage is observed. If the circulation of the frozen area can be restored, the area where the treatment will benefit most is the stasis area.

First degree injury is characterized by partial frostbite, erythema, mild edema, blistering, and skin desquamation that occurs after a few days. The patient complains of tingling, burning and throbbing. Its prognosis is very good.

Second degree injury is characterized by full thickness skin frostbite, severe edema formation within 3-4 hours, and the formation of thromboxane and prostaglandin rich blisters. The formation of the bullae is observed within 6-24 hours, progresses towards the fingertips and usually turns into

hard-black eschar within a few days. Patients complain of numbness following pain and throbbing. It has a good prognosis.

Third degree injury is characterized by progressive damage to the subdermal plexus. Hemorrhagic bubbles form on the damaged area and blue-gray discoloration is observed on the skin. These are associated with skin necrosis. Patients associate the injury site to a piece of wood. It has pains like burning and throbbing. Its prognosis is generally worse.

Fourth degree injury is characterized by its extension to subcutaneous tissues, muscles, bone and tendons. There is very little edema. A deep, dry, black, and mummified eschar tissue is formed. Often the vesicles do not form or appear late, they may be small/bloody blisters but do not extend to the fingertips. The patient may complain of a deep joint pain. The prognosis is often poor.

Pre-hospital field management needs much protection from cold exposure. Hypothermia and dehydration should be prevented. Wet and tight-shrunken clothing should be removed. The patient must be protected from the wind. In mild cases of consciousness, warm drinks can be given. The frozen area should not be heated directly. This can cause the damage to worsen. Heating should be done after the risk of refreezing is eliminated (Handford, C., Buxton, P. & Russell, K., 2014; McIntosh, S.E., Hamonko, M. & Freer, L., 2011). Since reheating will be a very painful process, it is recommended to apply analgesia beforehand. Rubbing the frozen area directly or with snow may increase the injury. Home-use creams which are used at home are not recommended in the field. The frozen zone must be fixed.

The first step of treatment in emergency departments is rapid reheating. For pain management, iv opiates can be given before the intervention. Local care is provided for tissue preservation and infection. It should not be applied to hemorrhagic blisters as debridement will cause tissue drying and poor prognosis. Care with aloe vera is recommended for all types of bullae. Extremity elevation and wearing loose clothing are recommended to reduce edema formation. Wound closure with dry sterile gauze and loose bandages is performed. Intra-arterial tissue plasminogen activator and thrombolysis treatment are controversial. Sympathectomy and sympathetic blockage are among the recommendations to reduce vasospasm and edema.

Drowning

Water-borne natural disasters such as floods and tsunamis occur with the effect of a great force. An underwater earthquake, volcanic eruption,

landslide or fall of high-volume meteorites results in the displacement of a significant amount of water in a rapid process.

Patients removed from the water should be rescued in a supine position to reduce the risk of orthostatic and hydrostatic changes. Most of the drowning happens when the patient is immediately in the water and suffers from hypoxic cardiac arrest, after which hypothermia develops.

The cause of a cardiac arrest observed after an avalanche disaster is asphyxia or trauma (Boyd, J., Brugger, H. & Shuster, M., 2010). Hypothermia that may occur after avalanche is much slower than when drowning in cold water. For this reason, hypothermia is not found to be among the reasons for the arrest in patients returning from cardiac arrest in <35 minutes. Hypothermia may be considered to be among the causes of arrest in patients with a snow-filled airway, reached in cardiac arrest in >35 minutes, and a core temperature >32°C. In these patients, prolongation of resuscitation is recommended.

Dry drowning occurs in 10-20% of drowning incidents. This is due to laryngospasm secondary to hypoxia. The most common situation is wet drowning caused by aspirating water into the lungs. The pulmonary surfactant dilutes in wet suffocation. Alveolar gas exchange is disturbed and atelectasis occurs. If drowning occurs in fresh water, hemodilution, hemolysis and hyponatremia are observed (Boyd, J. et al., 2010). Hypernatremia, hyperkalemia and hemoconcentration occur in cases of drowning in salt water. Bacteria, chemicals, stomach contents, and foreign substances that enter the lungs of people during suffocation will have negative effects on the prognosis.

Resuscitation of the injured should be started as soon as possible before reaching the hospital. As there are no data on spinal injury during the removal of the victim from the water, there has to be acceptance of injury. Protection from hypothermia is necessary after leaving the water.

After entering the emergency room, an advanced airway should be provided if needed. Resuscitation should be directed by determining factors such as the water in which the person drowned, how long he stayed there, and the core temperature (Boyd, J. et al., 2010). Seizures may occur due to hyponatremia in those who drown in fresh water. Those who drown in warm water have a lower chance of returning to spontaneous circulation than those who drown in cold water. Hypothermic patients should be warmed by internal and external methods. The decision for exitus should not be made before reaching the core temperature, and resuscitation should be prolonged. Patients with good general conditions should be asymptomatic for at least 4-6 hours before discharge from the emergency department and have an arterial oxygen saturation >95% at room air.

Psychological Problems

Natural disasters are a serious source of stress in the moment of the disaster and during the next process. Living with anxiety for both themselves and their relatives creates psychological burdens that people cannot cope with. Many negativities such as injuries during the moment and after the disaster, observing what is happening in the environment, not being able to meet other victims in need, not being able to reach their loved ones or losing their loved ones are a trauma element for individuals (Boyd, J. et al., 2010). For this reason, a psychological assessment should be made of each individual who experiences and shares these adverse conditions, whether they are the victim, relatives or rescuers. Frequent nightmares, survival guilt, depression, suicidal thoughts and anxiety disorders should be determined. The stress experienced will reduce the body's resistance and adversely affect the immune system (Boyd, J. et al., 2010). It will adversely affect the outcome of the required medical interventions after the rescue from the debris.

Conclusion

People's management of stress and trauma factors is another issue. For some people, a negative event is easy to overcome, while for others it is much more difficult. Sometimes this process can take months or years. In the recovery process, people may make negative orientations such as substance use and alcohol consumption in order to cope with these problems.

The fact that psychological evaluations are a part of the service provided to the victims can contribute positively to people's efforts to return to normal life.

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CHAPTER TWO

GENERAL CHARACTERISTICS OF DISASTERS

GÜLŞEN ÇIĞŞAR

Introduction

A disaster happens suddenly and unexpectedly. It disrupts the normal order of life. It causes desperation, a feeling of pain and disrupts the socioeconomic structure of the region in which it occurs.

Disasters may occur for many different reasons, and in some cases, they may occur as a result of the coexistence of more than one cause (Varol and Gültekin 2016; Jha et al.2010). In this section, firstly, the definition and general characteristics of disasters will be explained.

Disaster definition: A disaster can be defined as an unpredictable and often sudden situation or event that exceeds the local capacity, requires a request for foreign aid at the national or international level, and causes great damage, destruction and human suffering.

Disasters occur as a result of the convergence of hazards with security vulnerabilities in the region. Therefore, an increase in physical, social, economic or environmental vulnerabilities may mean an increase in the frequency of disasters (Varol and Gültekin 2016; Jha et al.2010). The World Health Organization defines a disaster as “an ecological phenomenon large and sudden enough to require external assistance” (Singh 2020).

General effects of disasters

The effects that occur after disasters can be direct or indirect. Disasters have different effects according to their mechanism of occurrence and size, the affected population and the capacity of local organizations to deal with them. Economic crisis, famine, epidemics, migration, homelessness and deaths, and orphaned children as a result of these deaths are among the problems that occur in almost all disasters. Along with these effects, one or

more of the following can be seen in disasters in general (Singh 2020; Balsubramanıan 2014);

- Loss of life
- Injury
- Loss of property (movable/immovable)
- Loss of farmland/agricultural products
- Interruption of production
- Disruption of people's living standards
- Disruption of transportation
- Loss of jobs and livelihoods
- Loss of basic needs such as electricity and natural gas
- Infrastructure damage
- Damage to communication networks
- Disruption of state institutions
- Limited access to clean water
- Food shortage
- Epidemic diseases
- Economic effects
- Sociological influences
- Post-disaster psychological effect.

Causes of Disasters

The increases in world population, global warming and vectors carrying disease cause a global increase in the frequency and severity of disasters that cause great loss of life and material damage. Disasters can be natural or man-made (Singh 2020).

Natural Origin Disasters

Volcanic eruptions: During volcanic eruptions, cracks and earthquake-like tremors are seen and experienced on the earth. During the eruption, lava, ash, toxic gases, soil and rock fragments can be released into the environment. These vibrations and cracks can cause serious damage and destruction to structures. The spreading lava can cause forest fires as well as damage buildings and destroy all living things living in it, by engulfing cultivated areas such as fields and gardens (Balsubramanıan 2014; Hansell and Oppenheimer 2004). Ashes scattered in the air affect the skin and mucous membranes, while toxic gases can cause serious respiratory problems.

These ashes enter the engines of vehicles and aircraft, causing damage, affecting local water supplies and making clean water supplies difficult. When combined with heavy rainfall, mud flows occur (Balsubramanıan 2014; Hansell and Oppenheimer 2004). In volcanic eruptions, various air pollutants are emitted, including gases and fine particles. Harmless water vapor is the most abundant volcanic gas. Carbon dioxide (CO2), sulfur dioxide (SO2), hydrogen sulfide (H2S) and, to a lesser extent, hydrogen halides (hydrogen chloride [HCl] and hydrogen fluoride [HF]) and carbon monoxide (CO) are the other gases emitted. Some volcanoes emit radon, a radioactive gas produced by the decay of uranium deep within the earth. Volcanic gases are all colorless (invisible), but they have different odors (Hansell and Oppenheimer 2004).

Sulfur dioxide (SO2) reacts in the atmosphere to form volcanic sulfate. It is acidic and goes deep into the lungs. Acid rain can occur in volcanic eruptions. Acid rain pollutes clean water sources. Cultivated agricultural lands are damaged, making food supply difficult (Hansell and Oppenheimer 2004). Volcanic eruptions cause the mass death of people and animals, have negative effects on human health and psychology, and cause serious destruction in all fields of agriculture, animal husbandry, and technology and socioeconomic damage.

Earthquakes: Earthquakes are tremors of the earth due to seismic waves formed as a result of the movement of the earth's tectonic layers. Due to their movement, stress occurs in these plates and rocks; when this stress exceeds the capacity of the rock, a high-energy fracture occurs with an earthquake. The greater the stress load and the longer it lasts, the greater is the intensity of the earthquake (Balsubramanıan 2014; Below et al. 2009). Earthquakes are the most devastating natural disasters, killing hundreds of thousands of people in earthquake-prone regions of the world (Bhosale 2015).

Earthquakes are sudden and unpredictable disasters. They may result in extensive damage, loss of life or injury. However, earthquake zones can be determined and precautions can be taken in these zones. Smaller aftershocks can be felt after large earthquakes (Balsubramanıan 2014). The magnitude of earthquake damage; The depth and duration of the earthquake and the population density in the region are affected. An earthquake causes buildings to collapse or be damaged and dams, bridges, tunnels and roads to collapse, causing thousands of people to die and become homeless. After the earthquake, it is necessary to establish safe, equipped health facilities that will not be affected by aftershocks for food and clean water supply, hygiene to protect against epidemic diseases, shelter, and care of the injured. Affected individuals should be provided with psychological support. An