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Anaesthesia Management Of Patient Undergoing Esophagogastroduodenoscopy For Haematemesis And Haematochezia Post Drowning

[1]Dr. Shubhajyothi Swamiadke*, [2]Dr. Neha Rai, [3]Dr. Nikita Ranjan, [4]Dr. Deepika Sathe
[1]Junior Resident, [2]Senior Resident, [3]Assistant Professor, [4]Professor,
Department of Anaesthesiology, MGM Medical College

***Corresponding Author:**
Dr. Shubhajyothi Swamiadke
Junior Resident, Department of Anaesthesiology, MGM Medical College

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Abstract

Acute bleeding from the upper and lower gastrointestinal system post drowning is a medical emergency which, despite availability of modern diagnostic methods and treatment, it still follows a high mortality rate. Bleeding from the upper and lower gastrointestinal system are mainly characterized by occult bleeding, while profuse bleeding rarely occurs accompanied by hemorrhagic shock. In our study we present this unique case of a 58-year-old female patient who had drowned for about 15 minutes a month back. She was now admitted for the third time in a month of the drowning incident, as a case of asphyxia, haematemesis and haematochezia which caused breathlessness, and profuse bleeding through vomit and through rectum respectively, thus projecting a clinical picture of severe hemorrhagic shock. Histopathological examination indicated very low blood counts. Urgent esophagogastroduodenoscopy under sedation or local anaesthesia was a sensitive and specific diagnostic and therapeutic method of choice for further investigations.

Keywords: Haemorrhagic Shock, Asphyxia, Haematemesis, Haematochezia, Cardiopulmonary Resuscitation (CPR), Esophagogastroduodenoscopy (OGD Scopy)

Introduction

According to the World Health Organization (WHO), approximately 0.7 %, or 500,000, deaths each year are due to unintentional drowning.[1 2.] Drowning is the second leading cause of unnatural death worldwide after road traffic accidents. Immediate resuscitation with rescue breaths and relief of hypoxia is fundamental to survival. Fluid aspiration, hypoxia, hypercarbia, and hypothermia cause multiple organ dysfunction requiring intensive care management. Frequent pathologic and neurologic assessment should be done. A protective ventilation strategy should be adopted in order to manage the acute lung injury/acute respiratory distress syndrome that may follow successful resuscitation after drowning.

In its 2010 resuscitation guidelines, the American Heart Association recommends according to the Utstein guidelines, drowning refers to: "A process resulting in primary respiratory impairment from submersion or immersion in a liquid medium". The Utstein guidelines further suggest that ambiguous or confusing terms such as "near-drowning," "secondary drowning," and "wet drowning" should not be used.

All drowning victims should have 100 % oxygen during their initial evaluations. Early use of intubation CPAP / bilevel positive airway pressure (BiPAP) in the awake, cooperative, and mildly hypoxic individual is warranted if asphyxia persists followed by further diagnostic procedures to ascertain the location of bleeding using noninvasive procedures like esophagogastroduodenoscopy. However, to date, no study has systematically investigated the impact of drowning-induced asphyxia on hemostasis followed by haematemesis and haematochezia.

Case Presentation

A 58 year old female patient, a housewife, a case of asphyxia, haematemesis and haematochezia after one month post drowning, was posted for esophagogastroduodenoscopy on 12 July 2022 in order to evaluate the cause of bleeding.

She was apparently alright one month back when she accidentally drowned in a beach in Konkan area for about 15 to 20 minutes. Subsequently she was conservatively managed in outside hospital for one week in view of dyspnea. After discharge, she again developed breathlessness with loose stools. She was treated for a suspected infection in the lungs in outside hospital and was discharged after a week on being stable. She was at her home for a week after which she developed vomiting with blood and breathlessness. She took homeopathic medicine which temporarily relieved her symptoms.

Four days later, she presented in Emergency Department on 09 July 2022, with asphyxia, heamatemisis and haematochezia. She was a known hypertensive since 8 years, and known diabetic since one month, not on any medication. She was also a known case of rheumatoid arthritis since 2 years, with swan neck deformity of fingers. She has a past surgical history of tubal ligation 30 years back under spinal anaesthesia.

On presentation, BP was 90 / 60 mmHg, PR was 90 per minute, RR was 28 per minute, SPO2 was 98% on room air and severe pallor was present. On auscultation, air entry was bilaterally reduced with bilateral crepts present. The patient was shifted to MICU for monitoring and further evaluation.

Primary investigations of 10 July 2022 revealed severe anaemia with Hb of 2.5 gm%, and a raised PT/INR of 18.9 / 2.47. She was transfused with 2-pint packed cell volume (PCV) and 4 pint fresh frozen plasma (FFP) on 09 July and 10 July 2022. The patient's 2D Echo report of 14 June 2022 reported dilated cardiomyopathy with ejection fraction (EF) of 25%, severe left ventricular (LV) systolic and diastolic dysfunction, moderate functional mitral regurgitation (MR), right ventricular (RV) dysfunction and significant pulmonary hypertension.

On arrival in the ICU, she was started on Inj. Noradrenine (16/50) at 2 ml/hr, Inj Telmipressin 2 mg six times a day, Inj Pantaprozole 200 / 50 @ 2 ml / hour and Inj Vitamin K 10mg OD.

On 12 July 2022, the patient was shifted from ICU to endoscopy room for esophagogastroduodenoscopy. On arrival, pulse rate was 130 per minute, BP was 110 /70 mmHg on Inj Noradrenaline (16 / 50) at 4 ml / hour and Inj Vasopressin @ 2.4 ml / hour. SPO2 was non recordable. On auscultation bilateral crepts were present. Most recent preoperative investigations revealed Hb 3.4 gm / dl, Platelet count of 81000, and ejection fraction of 25%. The case was discussed with the gastroenterologist, was explained the high risk and possibility of cardiopulmonary arrest. It was decided that the patient will be taken for OGD Scopy under local anaesthesia. Sedation and total intravenous anaesthesia was ruled out due to high risk to the patient.

For local anaesthesia application, LOX 10% was sprayed over the posterior pharyngeal wall and the procedure was started. The patient tolerated the procedure well and was stable up to 5 minutes. Subsequently she developed bradycardia for which Inj atropine 0.6 mg IV was given. The procedure was stopped as the patient became unresponsive. Carotid pulse could not be felt, CPR was initiated as per the American Heart Association advanced cardiac life support protocol. Rhythm was recognized as asystole, Inj adrenaline 1mg was given and CPR was continued for 2 minutes. Next rhythm again was asystole, CPR was continued, and advanced airway was secured with endotracheal tube no 7. Bilateral air entry was confirmed. The return of spontaneous circulation was achieved within 4 minutes. ECG showed sinus rhythm. Patient was shifted to ICU intubated, sedated and paralysed, for further management.

Discussion

Globally, every year, drowning accounts for at least 500,000 deaths worldwide, including approximately 4000 fatalities in the United States. Statistics for nonfatal drowning are more difficult to obtain, but nonfatal drowning events may occur several hundred times as frequently as reported drowning deaths.

The immersion or more precisely the presubmersion phase is the one in which the individual maintains the face above water by treading and will continue until the point of fatigue in deep watery environments.

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This is followed by a submersion phase in which the covering of the nose and mouth will trigger a series of physiological responses beginning with voluntary breath holding or apnea. The entire head need not be submerged and thus drowning can occur in as little as a few inches of water that could exist in buckets, washbasins, and shallow natural bodies of water like streams.

Breath-holding continues until a certain breakpoint or critical blood O2 concentration and CO2 concentration is reached, causing states of hypoxia, hypoxemia, and hypercarbia. This breakpoint triggers an involuntary gasping response, which would ideally lead to inhalation of air if head is above water, but by being submerged in the now hostile watery environment, water and any admixed debris enters the airway. There is interindividual variability in the amount of water inhaled and as little as 1 mL/kg up to 11 mL/kg of water can precipitate the drowning process. Swallowing of water may occur simultaneously, increasing the risk of vomiting with aspiration of gastric contents, which can cause further alveolar injury. Transient laryngospasm or bronchospasm may occur as a result of stimulation of the innervated mucosa of the oropharynx and larynx by water. Whether this actually limits or precludes further entry of water into the lungs leading to so-called "dry drowning" remains unclear and unsubstantiated, prompting the World Congress on Drowning to abandon use of the term. Subsequent relaxation of the larynx will eventually permit water to be inhaled into the lungs. The worsening hypoxia and hypoxemia initiated by the impaired oxygen diffusion in the lungs leads to anoxia, depletion of brain energy reserves with deterioration of brain function, failure in brain energy metabolism, loss of consciousness, and irreversible neuronal cell injury. Irreversible neuronal cell injury may begin approximately four to six minutes after sustained oxygen deprivation and the degree of partial or total recovery after a submersion event is dependent on the extent and regions of the brain affected and the promptness of resuscitative efforts. Aside from the neurological effects arising from pulmonary oxygen impairment, worsening hypoxia may also trigger an asphyxial or hypoxic cardiac arrest. Profound systemic hypoxia coupled with hypercarbia also leads to respiratory and metabolic acidosis, cardiovascular collapse with multisystem organ failure, and eventually death. [2]

Researchers have recognized that aspiration of more than 11 mL/kg of body weight must occur before blood volume changes occur, and more than 22 mL/kg before electrolyte changes take place. It is unusual for nonfatal drowning victims to aspirate more than 3 to 4 mL/kg, and the distinction between salt water and fresh water drowning is no longer considered important. Both types of nonfatal drowning result in decreased lung compliance, ventilation-perfusion mismatching, and intrapulmonary shunting, leading to hypoxemia that causes diffuse organ dysfunction. The temperature of the water and the presence of contaminants may affect patient outcomes.

Eighty percent of patients with drowning-induced asphyxia developed overt disseminated intravascular coagulation within 24 hours. When compared with nondrowning cardiac arrest patients, drowning patients had a 13 times higher prevalence of overt disseminated intravascular coagulation at admission. Most drowning patients develop overt hyperfibrinolytic disseminated intravascular coagulation, partly caused by hypoxia induced tissue plasminogen activator release. Antifibrinolytics and heparinase partially reverse the abnormal clotting patterns. Severe activated partial thromboplastin time prolongation may be a marker of combined hyperfibrinolytic afibrinogenemia and autoheparinization in drowning-related asphyxia.[3 4]

The management of specific complications of nonfatal drowning are namely, hypothermia, acute respiratory distress syndrome, and bradycardia. The management of specific complications of fatal drowning are namely asphyxia, haematemesis and haematochezia. The damage to the gastrointestinal system needs further investigation. Stomach mucosa tears were found in 21.1% of the cases of drowning, mostly on fundus (54.5%). [5 6]

The usual asphyxia, haematemesis and haematochezia conditions as associated in fatal drowning or other methods of suppressing respiration, is not a simple case of oxygen want. The factor of carbon dioxide accumulation is always associated. [7]

Conclusion

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Drowning occurs in a predominantly healthy and young population and results in significant morbidity and mortality. The primary event is hypoxia due to aspiration of liquid. Secondary pulmonary and neurological injury after cardiac arrest determines patient survival and subsequent quality of life.

While the tendency to presume that the patient has undergone a water-related drowning by accident or other means, comprehensive medical investigations for sufficient period must be performed to ensure accurate mode of revival, while also unearthing any criminality. Multidisciplinary and multiagency efforts are required for the gathering of the vital information, which provide context within which the pathologist can interpret any toxicological findings. Most drowning patients develop overt hyperfibrinolytic disseminated intravascular coagulation, partly caused by hypoxia induced tissue plasminogen activator release. Further detailed investigations are essential for effective revival and sustainable recovery of the patient. Non Invasive or Invasive diagnostic procedures or surgeries undertaken need to be reviewed by anesthesiologist after evaluation of the patient suitability for appropriate selection of anaesthesia technique.

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