Nutritional Assessment of Patients Undergoing Esophageal Dilatation Program for Post Corrosive Esophageal Injury

Thesis

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بسم الله الرحمن الرحيم

وَقُلِ اعْمَلُوا فَسَيَرَى اللَّهُ
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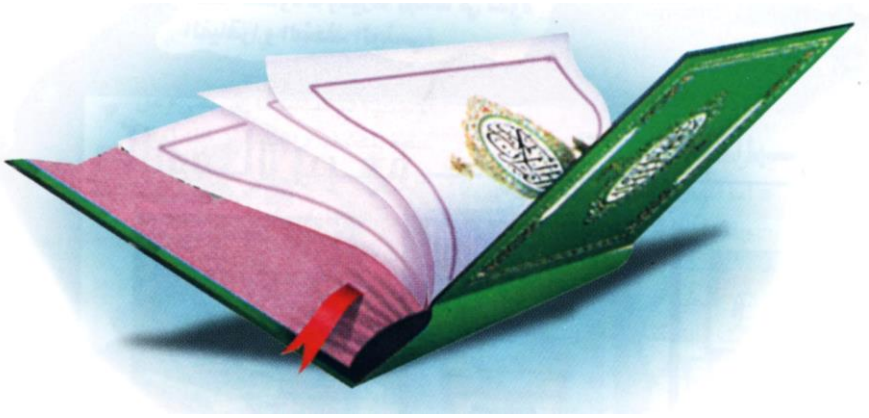

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Mona Ismail Mahmoud Mohammad

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List of Abbreviations

Abb.	Full term
ALP	*Alkaline phosphatase*
BMI	*Body Mass Index*
CT	*Computed tomography*
ES	*Endoscopic*
ESPEN	*European Society for Parental Enteral Nutrition*
GIT	*Gastro Intestinal Tract*
HCT	*Hematocrit test*
LOS	*Length of stay*
MCV	*Mean Corpuscular Volume*
NRS	*Nutritional Risk Score*
PA view	*Poster anterior view*
PEJ	*Percutaneous endoscopic Jejunostomy*
PLT	*Platelet*
PNRS	*Pediatric Nutritional Risk Score*
PNST	*Pediatric Nutrition screening tool*
PYMS	*Pediatric York hill Malnutrition score*
SGNA	*Subject global Nutritional Assessment*
STAMP	*Screening Tool for the Assessment of Malnutrition*
TIBC	*Total Iron Binding Capacity*
WBC	*White Blood Cell*

INTRODUCTION

Caustic ingestion continues to be a major health hazard in developed and developing countries. At the end of 19th century and the beginning of 20th century, dye products became commercially available for domestic use. These agents are highly corrosive if ingested ***(Scheers et al 2015).***

The ingestion of caustic substance induces a wide range of injury to the lips, oral cavity, pharynx, esophagus, stomach and upper airway. The peak is seen in children aged 1 to5 years, with most of these ingestions being accidental ***(Chibishev et al 2016).***

Swallowing of corrosives is intensively expressed on (mucosa), submucosa and muscle layer of the esophagus, stomach and duodenum. Extensive damages of gastrointestinal tract disable the physiologic nutrition of these patients. They are in a very difficult general condition either because of hypercatabolic condition or negative nitrogen balance ***(Heuschkel et al 2015).***

Acute intoxications with corrosive substances can cause severe chemical injuries of the upper gastrointestinal tract, most often located in the mouth, pharynx, esophagus, stomach and duodenum ***(Braunschweig et al 2012).***

If a patient survives the acute phase of intoxication, regenerative response may result in esophageal and/or gastric

stenosis, and increased risk of esophageal and gastric cancer. Such intoxication may be fatal due to perforation or tracheal necrosis. Unlike pediatric population where corrosive intoxications are most often accidental, corrosive intoxications in adult patients are intentional or suicidal in more than 90% of the cases ***(Campbell et al 2012).***

Severity of the resulting chemical injuries depend on several factors: nature of the corrosive substance, quantity, concentration, duration of exposition, swallowing, food presence, gastroesophageal reflux, or various previous pathological conditions of the upper gastrointestinal tract ***(Cereda et al 2015).***

Post-corrosive injuries can be reversible and irreversible. Injured mucosa, submucosa and muscular layer are regenerating poorly due to the surrounding inflammation, necrosis and secondary complications. Resulting fibrosis and adhesions or circular stenosis greatly impair the upper gastrointestinal function such as impaired peristaltic and passage. Latter leads to further deterioration of patient's general condition, inability for physiologic nutrition, weight loss, prostration and cachexia. Such development can result in fatal outcome due to inadequate nutrition, i.e. inability for normal feeding caused by complications ***(Spiers et al 2012).***

Severe GIT injuries prevent normal nutrition of patients. Patients are in poor general condition, characterized by

hypercatabolic state and negative nitrogen balance. Therefore, early nutritional support is of great importance for the treatment and outcome of these patients. Effects of the nutrition substitution in patients with life-threatening disorders, including the reduction of infection risk, aspiration pneumonia or pulmonary embolism, as well as stimulation and facilitated healing of injuries, are main reasons for use of artificial nutritional support in patients with acute corrosive intoxications ***(Ferrante et al 2012).***

The aim of the treatment of acute corrosive intoxications is to prevent perforation and progressive fibrosis, and esophageal and gastric stenosis. Such treatment mainly consists of anti-secretory treatment, antibiotics and intensive hyper-alimentation, aiming to prevent late post-corrosive intoxications ***(Sahn et al 2014).***

It is considered that nutritional support plays a major role in maintenance of metabolic processes and prevention of severe metabolic complications that could additionally aggravate the condition and impair the treatment ***(Russell et al 2014).***

Aim of the Work

This thesis aims at assessment of the nutritional status of pediatric patients with post corrosive esophageal strictures on regular endoscopic dilatation protocol as compared to those patients undergoing feeding gastrostomy after corrosive ingestion, in order to evaluate the efficiency of this technique in maintaining good nutrition.

Chapter 1

POST CORROSIVE EFFECT

Corrosives are a group of chemicals that have the capacity to cause tissue injury on contact by a chemical reaction. They most commonly affect the gastrointestinal tract (GIT), respiratory system and eyes. Corrosives and caustics are synonyms, both mean 'something that eats away'. Acids and alkalis are the two primary types of agents most often responsible for caustic exposures ***(Raghu and VadIvelan 2012).***

Exposure to corrosive agents continues to be a leading toxicological source of injury for children. An average home contains a dozen different cleaning products. These account for a large number of accidental and intentional poisonings ***(Chang et al 2011).***

The estimated prevalence of corrosive poisoning is 2.5-5% while the morbidity is above 50% and the mortality is 13%. Eighty percent of corrosive poisoning occurs in children below five years ***(AAPPCC 2015).***

1- Common corrosive agents:

The common caustic agents include: Strong acids and alkalis, concentrated weak acids and alkalis Oxidizers (with neutral pH) and Alkylating agents, Dehydrating agents Halogens and organic halides Phenol ***(Plackova et al 2013).***

Acids includes Car battery fluid (sulfuric acid) Descalers (hydrochloric acid) Metal cleaners (nitric acid) Rust removers (hydrogen fluoride). Alkalis includes Bleach (hypochlorite) Sodium hydroxide (liquid lye) ***(Mrazova et al 2012).***

Uses of common caustic agents:

Hydrochloric acid-metal/toilet bowl cleaner, Sulfuric acid-automobile batteries, Sodium hydroxide-paint remover/drain cleaner and Phenol-antiseptic ***(Zakharov et al 2013).***

Factors Determining Corrosiveness

Factors that determine corrosiveness include:

Solid/liquid, Duration of contact with tissue, Concentration of agent, Quantity of agent pH of agent: pH, are more corrosive Food: Presence or absence of food in stomach, Titratable acid or alkali reserve (TAR): This quantifies the amount of neutralizing substance required to bring the pH of a caustic agent to physiological pH of the tissue ***(Olson 2014).***

Mechanism of Action of Corrosive Agents

Historically, the rate and severity of caustic ingestion injury increased markedly with the introduction of liquid drain cleaners, because liquid alkalis can be swallowed in a significant quantity, before the subject recognizes the mistake and experiences the symptoms ***(Toxbase 2016).***

Prior to this time, the ingestion of a solid crystalline or powder forms of these products tended to occur in smaller amounts because it was difficult to ingest much before severe pain ensued. The liquid forms also tend to cause more extensive circumferential burns of the esophagus ***(Johnson and Brigger 2012).***

It has been said that strong alkalis "bite the oesophagus and lick the stomach" while strong acids "lick the oesophagus and bite the stomach" ***(Huska et al 2010).***

Alkali ingestion:

Causes liquefaction necrosis. This process includes protein dissolution, collagen destruction, fat saponification, cell membrane emulsification, submucosal vascular thrombosis and cell death ***(Park 2014).***

Acid ingestion: Causes coagulation necrosis. In this process, hydrogen (H+) ions desiccate epithelial cells producing an eschar. This process leads to edema, erythema, mucosal sloughing, ulceration and necrosis of tissues. Both acids and alkalis cause fibrosis and cicatrization (stricture formation) ***(Urganci et al 2014).***

Consequences of Caustic Injury

Acute exposure to cleaning products in children constitutes a major problem for healthcare specialists worldwide ***(Manzar et al 2010).***

This injury occurs mostly by accident, and the accidental ingestion of cleaning products instead of a soft drink or water is very common**.** Careless storage and improper safeguarding of chemical is the single reason for accidental ingestion. Consecutively, caretakers' education about intoxication prevention seems necessary ***(University of Chicago 2016).***

Labeling of cleaning products plays an important role in the prevention and treatment of exposure, since the label should be a source of toxicological information and instructions to first aid for parents and adults and health professionals ***(Ekpe and Ette 2012).***

Caustic injury may cause the following: 1- Necrosis: Occurs within seconds of exposure to caustic agent 2- Ulceration and perforation: Occurs within 24-72 hours of exposure 3- Fibrosis: Occurs within 14-21 days of exposure 4- Stricture: Occurs after weeks to years of exposure, 5- Carcinoma formation: Occurs after decades of alkali exposure ***(Vezakis et al 2016).***

Clinical Presentation in Corrosive Poisoning

1- Effect on digestive tract:-

Corrosive injuries to the upper gastrointestinal tract are still a major concern in developing countries where the corrosive substances are easily accessible to common people including children. These substances are used commonly in the