IS EARLY ESOPHAGOSCOPY NECESSARY IN THE PEDIATRIC INGESTED CAUSTIC SUBSTANCES? A RETROSPECTIVE CLINICAL STUDY

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ABSTRACT

Ingestion of the corrosive substances can cause serious, even fatal, injuries in the children. There are several approaches in the treatment of the patients with esophageal injuries, including early esophagoscopy. The aim of this study was to evaluate the endoscopic findings retrospectively and correlate them with clinical progress in children ingested caustic substances, as well as to investigate whether the early esophagoscopy is necessary. In this retrospective cohort study the patients were divided into two groups: G1-patients underwent esophagoscopy and G2-patients did not undergo esophagoscopy. We obtained 103 patients (68 male (66 %) and 35 female (34 %)) with a median age 41 months (range: 4-144 months; Mean age: 41.01 ± 31.24 months). Sixty-six children (64%; Age: 42.7 ± 29.3 months) indicated as G1 underwent early esophagoscopy (within 12-24 h postingestion), whereas 37 patients indicated as G2 did not undergo esophagoscopy (36 %; Age: 38.1 7 ± 34.1 months). In G1, two patients (3.03 %) the endoscopic findings were identified as grade Ia and Ib. On the other hand, in G2 one patient (2.70 %) was observed with stricture requiring repeated dilatation. In both groups, stricture prevalence was found similar. Among the patients, 73 of them (70.9 %) were inspected with oral burns, whereas 30 patients (29.1 %) were not showed any oral burns. No relation between the esophagoscopy findings and stricture development was observed (Pearson Chi-Square test, 2(1) = 0.01, p=0.924). No relation was determined between the stricture development and the presence of oral burns (Pearson Chi-Square test, 2(1) = 0.03, p=0.871). In our study, the prevalence of the stricture development was identified similar in both groups. As the incidence of the stricture development is regardless of esophagoscopy findings. Therefore, our results suggest that early esophagoscopy is not necessary for the caustic ingested pediatrics.

INTRODUCTION

Intentional or accidental ingestion of caustic substances may lead to life-threatening injuries such as esophagus, pharynx, larynx, mouth and stomach (Turner and Robinson, 2005). Household bleach, dishwasher detergents, and other cleaning agents are all mildly alkaline substances, and the household cleaning products are used daily are the most common caustic substances (Rigo et al., 2002; Stiff et al., 1996). Presentations to the emergency departments due to the ingestion of caustic substances are not the most common; however, more than 5,000 cases of caustic substance ingestion in USA annually (Erdoğan et al., 2003). Caustic substances are chemicals that can induce tissue injury on direct contact.

An 80% of the cases were indicated to involve the pediatrics, whereas the ingestion of the caustic substances is more serious due to the intention of suicide (Bussen, 1993). Injury patterns for acid burns and alkali burns differ, as acids cause coagulative necrosis leading to a self-limiting burn pattern, while alkalis lead to generation of liquefactive necrosis followed by saponification of the fats and solubilization of the proteins (Weigert and Black, 2005). Even small amount of ingestion of the caustic substance by the pediatrics may lead to morbidity and mortality (Weigert and Black, 2005). Therefore, a good prediction of the presence and severity of the esophageal lesions, as well as the caustic reagent is a necessity. The ingestion of the caustics may lead to catastrophic effects or it may not lead to any complications (Mamede and De Mello Filho, 2002). There are divergent approaches on the diagnosis and treatment of the lesions caused by pediatric ingestions.
cases (Contini and Scarpignato, 2013). Previously, several investigators attempted to correlate the initial signs and symptoms with the severity of the lesions to avoid any unnecessary esophagoscopy operations; however, the latter remains as an open question. The debate can be briefly explained by the fact that the lack of oropharyngeal lesions or pharyngoesophageal symptoms does not rule out the presence of esophageal or gastric injury. Gaudreault et al. concluded that symptoms do not predict the presence or the severity of esophageal damage (Gaudreault et al., 1983). Others indicated that the patients at risk of severe esophageal injury and subsequent stricture always show signs and symptoms after the ingestion of a caustic substance (Lupa et al., 2009). On the other hand, Crain et al. reported that the presence of two or more symptoms is a better predictor of esophageal injury than only one symptom (Crain et al., 1984). The major problem for the physician is to resist the temptation to perform an endoscopy in children with no visible lesions, no signs and symptoms. The question is that the completely asymptomatic patients who are seen after unintentional ingestion of a caustic substance really have to undergo an esophagoscopy. The aim of the present study was to evaluate whether an esophagoscopy is always mandatory in children visited the emergency department due to a caustic ingestion, and whether the symptoms at presentation can predict the presence of esophageal lesions. Development of esophageal strictures is closely related to severity of the initial injury, and early esophagoscopy is widely recommended to assess the severity and extent of aerodigestive lesions and appropriate therapeutic regimens.

MATERIALS AND METHODS

Patient selection and experimental design

A group of 103 patients who admitted to Emergency Room were transferred to Pediatric Surgery unit after caustic substance ingestion during the period of January 2001 - December 2003. The oral intake was stopped and provision of 2000 cc/m² maintenance fluids and intravenous antibiotics administration was started. Use of the steroids were avoided in any patients. The patients were divided into two groups as the ones underwent the esophagoscopy (G1) and who did not (G2). Rigid esophagoscopy was chosen for the evaluation of esophagus for the patients in G1, and was performed by a pediatrics surgeon within 12 to 24 hours post-ingestion by using a pediatric-type rigid esophagoscope. When circumferential lesion was demonstrated, endoscopy was ended to avoid the risk of esophageal disruption by carrying on beyond the circumferential injury. Esophageal burns were classified by using the Zargar’s endoscopic grading classification with small modifications as indicated in Table I (Zargar et al., 1991). Among the patients who has lesions, oral intake was avoided for 72 h and then they were fed a liquid diet. When the period of being fed a liquid diet, the patients were discharged to their home with an appointment for 3 weeks later for follow-up examination. The patients who did not present any lesions after esophagoscopy were fed within 3-4 h after the effect of anesthesia was over, and were discharged to their home. The esophagoscopy procedure could not be applied in the patients in G2 due to various reasons (i.e. Upper respiratory tract infection, no-permission of the family and other system disorders). These patients were accepted as if they were presenting mucosal injury. The oral intake was avoided for 72 h and then they were fed a liquid diet. When the period of being fed a liquid diet, the patients were discharged to their home with an appointment for 3 weeks later to conduct the barium swallow test. The patients who did not present any lesions after esophagoscopy were fed within 3-4 h after the effect of anesthesia was over, and were discharged to their home. In addition to the esophageal examinations, leukocytes and platelets were counted as blood parameters from 102 patients. Leukocytosis (leukocyte counts > 10,000) and thrombocytosis (platelet counts > 300,000) were determined accordingly.

Statistical analysis

Statistical analyses were conducted by using the SPSS statistical software package (IBM). Statistical tests conducted for the data are indicated in the results section for the respective result. The p value lower than 0.05 was considered as statistically different.

Ethics committee approval

The study data were retrospectively analyzed. No treatment was given to patients, nor any comparison regarding treatment modalities was done. No test or treatment apart from routine management approach was used. Therefore, we felt no need to obtain ethics committee approval or families’ informed consents.

RESULTS

We obtained 103 patients (68 male (66%) and 35 female (34%)) with a median age 41 months (range: 4 – 144 months; Mean age: 41.07 ± 31.24 months). Among the patients, 73 of them (70.9%) were inspected with oral burns, whereas 30 patients (29.1%) were not showed any oral burns. Sixty-six children (64%; Age: 42.7 ± 29.3 months) underwent early esophagoscopy (within 12 – 24 h post-ingestion), whereas 37 patients did not undergo esophagoscopy (36%; Age: 38.1 ± 34.1 months). In order to evaluate the treatment outcome and prognosis of patients with caustic injuries, mucosal lesions were scored as grade I (erythema or petechiae), grade II (noncircumferentialIIa, circumferential IIb ulcers), or grade III (mucosal necrosis). In 25 children (38%) normal endoscopic findings, in nine children (14%) grade I, in 32 children (48%) grade II and 11 children (18%) grade III were identified. Children with no lesions were accepted as normal, and discharged home with normal alimentation. Others were allowed to start oral intake in 72 hours. Requiring repeated dilatation, esophageal stricture developed in two cases (3.03%) who had esophagoscopy. In these cases, with esophageal stricture, the endoscopic findings were identified as grade IIa and IIb, and they were given caustic substances ingestion treatment protocol (no oral intake before 72 hours, broad-spectrum antibiotics, steroid). In one case who did not undergo esophagoscopy (2.70%), stricture was observed. No relation between the esophagoscopy and stricture development was observed (Pearson Chi-Square test, \( \chi^2(1) = 0.01, p = 0.924 \)). Among those who developed stricture, two patients were observed with oral burns and one without oral burns. No relation was determined between the stricture development and the presence of oral burns (Pearson Chi-Square test, \( \chi^2(1) = 0.03, p = 0.871 \)). There was no gender difference in relation with the severity of the injury (Pearson Chi-Square test, \( \chi^2(3) \))
Mucosal injury was observed in 43 patients (65.15%) among the patients in G1 and in 30 patients (81.08%) in G2 (Pearson Chi-Square test, $\chi^2(1) = 2.91$, $p = 0.088$). Distribution of the developmental period of patients did not differ between G1 and G2 (Table 2; Pearson Chi-Square test, $\chi^2(4) = 5.68$, $p = 0.224$). Leukocytosis was observed in 44 patients (66.67%) in G1 and in 25 patients (75.00%) in G2, and no association was identified between esophagoscoppy and leukocytosis (Pearson Chi-Square test, $\chi^2(1) = 0.364$). Among the patients, 43 (41.7%) and 59 (57.1%) of them were determined as ingested alkaline and acidic agents, while the type of the ingested caustic of one patient was unknown. The barium swallow test was conducted on the 5 patients (13.5%), which were in G2. In total, eighty-nine patients (86.4%) did not attend the follow-up examinations, while the rest were examined. Among the 89 patients, mucosal injury of the 23 (25.8%), 7 (7.9%), 27 (30.3%) patients were scored as grade 0, grade I and grade II (IIa and IIb), respectively, while the 32 (36%) patients did not undergo for the esophagoscopy procedure. On the other hand, esophagoscopic examinations of the patients presenting oral burns (73 patients) revealed that the mucosal injury grade of 14 patients (19.2%) were grade 0, five patients (6.4%) were grade I, 24 patients (32.9%) were grade II (IIa and IIb). A group of 30 patients with oral burns (41.1%) did not undergo the esophagoscropy. Highest frequency of the patients (n = 40 (54.79%)) with burns were determined within the developmental period of toddlerhood (Table 2). Among the alkaline caustic ingested patients, 31 (72.1%) of them were inspected with burns, whereas the incidence of burns in the

Table 1. Endoscopic grading of mucosal injury

<table>
<thead>
<tr>
<th>Grade</th>
<th>Finding</th>
<th>N=43 (% of the total)</th>
<th>N=59 (% of the total)</th>
<th>Grade 0 (n=25) N</th>
<th>Grade I (n=9) N</th>
<th>Grade II (n=16) N</th>
<th>Grade III (n=37) N</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Normal esophagus</td>
<td>11 (16.67%)</td>
<td>11 (15.06%)</td>
<td>2</td>
<td>0</td>
<td>3</td>
<td>9</td>
</tr>
<tr>
<td>I</td>
<td>Mucosal edema and hyperemia</td>
<td>10 (14.81%)</td>
<td>7 (9.96%)</td>
<td>3</td>
<td>1</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>IIa</td>
<td>Friability, erosions, hemorrhage blisters, exudates, whitish membrane and shallow ulcers</td>
<td>11 (16.67%)</td>
<td>11 (15.06%)</td>
<td>3</td>
<td>1</td>
<td>7</td>
<td>9</td>
</tr>
<tr>
<td>IIb</td>
<td>Grade IIa lesions with deep or circumferential lesions</td>
<td>5 (7.54%)</td>
<td>11 (15.06%)</td>
<td>3</td>
<td>1</td>
<td>7</td>
<td>9</td>
</tr>
<tr>
<td>III</td>
<td>Mucosal necrosis</td>
<td>6 (8.84%)</td>
<td>11 (15.06%)</td>
<td>3</td>
<td>1</td>
<td>7</td>
<td>9</td>
</tr>
</tbody>
</table>

Table 2. Frequency and distribution of the ingested compound and the oral burns according to the esophageal grades and the developmental periods

<table>
<thead>
<tr>
<th>Developmental period</th>
<th>Type of the ingested corrosive</th>
<th>Patients underwent esophagoscopy N (%)</th>
<th>Frequency of oral burns (n=73) N (% of the total burns)</th>
<th>Severity of the injury of the patients according to Zargar’s grading system</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Alkaline N=43 (%)</td>
<td>Acidic N=59 (%)</td>
<td>Grade 0 (n=25) N</td>
<td>Grade I (n=9) N</td>
</tr>
<tr>
<td>Infancy (0-1 year)</td>
<td>4 (9.30)</td>
<td>12 (20.34)</td>
<td>7 (10.61%)</td>
<td>11 (15.06%)</td>
</tr>
<tr>
<td>Toddlerhood (1-3 years)</td>
<td>31 (72.09)</td>
<td>26 (44.07)</td>
<td>39 (59.00%)</td>
<td>40 (54.79%)</td>
</tr>
<tr>
<td>Early childhood (3-6 years)</td>
<td>3 (6.98)</td>
<td>11 (18.64)</td>
<td>9 (13.64%)</td>
<td>11 (15.06%)</td>
</tr>
<tr>
<td>Middle childhood (6-12 years)</td>
<td>5 (11.63)</td>
<td>10 (16.95)</td>
<td>11 (16.67%)</td>
<td>11 (15.06%)</td>
</tr>
<tr>
<td>Adolescence (12-18 years)</td>
<td>n.a.</td>
<td>n.a.</td>
<td>0 (0.0%)</td>
<td>0 (0.0%)</td>
</tr>
</tbody>
</table>

Table 3. Leukocytosis and platelet counts of the patients on different developmental periods

<table>
<thead>
<tr>
<th>Developmental period</th>
<th>N (% of the total)</th>
<th>Leukocytosis (Mean ± S.D.) / # of cases</th>
<th>Platelet counts (Mean (10^9/µL) ± S.D.) / # of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infancy (0-1 year)</td>
<td>16 (15.5)</td>
<td>13.59 (± 4.87) / 16</td>
<td>436.13 (± 138.78) / 16</td>
</tr>
<tr>
<td>Toddlerhood (1-3 years)</td>
<td>57 (53.3)</td>
<td>12.67 (± 5.18) / 55</td>
<td>407.75 (± 162.51) / 56</td>
</tr>
<tr>
<td>Early childhood (3-6 years)</td>
<td>14 (13.6)</td>
<td>12.97 (± 4.21) / 14</td>
<td>374.07 (± 77.89) / 14</td>
</tr>
<tr>
<td>Middle childhood (6-12 years)</td>
<td>15 (14.6)</td>
<td>14.25 (± 6.76) / 15</td>
<td>398.133 (± 126.06) / 15</td>
</tr>
<tr>
<td>Adolescence (12-18 years)</td>
<td>1 (1.0)</td>
<td>6.9 / 1</td>
<td>309.20 / 1</td>
</tr>
</tbody>
</table>
Analyses revealed that there was no relation between leukocytosis and the type of the corrosive ingested (p > 0.05; Figure 1a), as well as the type of corrosive ingested and platelet counts (p > 0.05; Figure 1b). Also, no significant differences were observed when the effect of gender on leukocytosis (p > 0.05; Figure 1c) and platelet counts (p > 0.05; Figure 1d) were investigated. Also, platelet counts did not have any relation with the severity of the injury (Pearson Chi-Square: $\chi^2(3) = 4.96, p = 0.175$). Then, we compared the leukocytosis and platelet counts on different pediatric developmental period. Leukocytosis and platelet counts did not differ from each other depending on the developmental period (Table 3). On the other hand, only patient on the adolescence period exhibited relatively lower leukocytosis and platelet counts.

**DISCUSSION**

The present study investigated the effect of the caustic substance ingestion on platelet counts and leukocytosis in the pediatric patients. A total of 103 patients, 64 of which underwent early esophagoscopy, were recruited. Caustic ingestion is generally accepted as the ingestion of the strong acidic or alkaline household products, such as drain openers, bleaches, toilet bowl cleaners, detergents, battery fluids, and commercial pesticides, as well as industrial products (Cheng et al., 2008). Depending on the time of exposure to the agent and caustic agent concentration, the full-thickness of the injury may vary, as well as the time of the symptoms occur (Gumaste and Dave, 1992). Similar to a recent study (Quingking et al., 2013), there was no relation between the severity of the injury and the ingested compound in our study. Previously, white blood cell count was found to be significantly higher in the patients with high grade upper gastrointestinal tract injury compared to low grade ones (Havanond and Havanond, 2007). On the other hand, the sole patient on the adolescence period was observed with grade III injury but low leukocytosis and platelet counts. This might be an indicator of that blood counts do not always correlate with the grade of the injury; however, one should be careful about the conclusion, as there was only one case in this study. In our study, we did not observe any relation between the leukocytosis and the compound ingested. Leukocytosis is accepted as a biological marker of the severity after ingestion of a corrosive agent by some researchers (Rigo et al., 2002), whereas some considers them as unreliable for prognosis (Turner and Robinson, 2005; Chen et al., 2003). Also, platelet counts did not either differ depending on the gender, type of substance or the developmental period of the patients. A previous study indicated a significantly lower platelet level as the severity of burns increased, suggesting a prognostic role of platelet count for the burn patient (Gajbhiye et al., 2013). On the other hand, another study suggested that the normal platelet counts may not be sufficient after a severe trauma (Brown et al., 2011). The caustic ingestion may lead to a severe esophageal injury, and our study showed that there was no relation between the severity of the injury and thrombocytosis, suggesting that the platelet counts are not useful for the determination of the severity of the esophageal injury. Necessity of esophagoscopy on the asymptomatic patients after caustic ingestion remains as a question. It was previously stated that the rigid esophagoscopy seems to be unnecessary on the children with no or low grade injuries, whereas it might be useful to reduce the hospitalization period and assess the esophageal injury as the result of the ingestion of the strong caustics (Bosnali et al., 2017). On the other hand, an earlier study also, Lamireau et al. concluded that early esophagoscopy is not necessary as the children with severe lesions always show symptoms in the developed countries.
Betalli et al. stated that esophagoscopy could be avoided in the asymptomatic patients, but should be applied on the symptomatic patients (Betalli et al., 2008). In parallel with the studies suggesting avoidance of esophagoscopy, we observed that the patients developed strictures regardless of esophagoscopy. Instead of esophagoscopy, one approach might be to apply elective endoscopy to prevent the unnecessary damage caused by the urgent esophagoscopy. Previously, it was stated that urgent endoscopy did not reduce the length of stay of the patients with acute non-variceal upper-gastrointestinal lesions (Bjorkman et al., 2004). Additionally, discovery of better predictors to conduct an elective evaluation is a necessity and to avoid the unnecessary esophagoscopy. In conclusion, our study showed that the stricture developed regardless of the esophagoscopy, suggesting that the esophagoscopy is not necessary to prevent the stricture development. We also showed that grade of the injury and type of the ingested caustic agent are not correlated. Moreover, the platelet counts and the leukocytosis are not useful biomarkers to determine the severity of the esophageal injury.

REFERENCES


