Effervescent agents in acute esophageal food impaction

1 Section of Gastroenterology, 1 Department of Internal Medicine, Banner University Medical Center Phoenix, University of Arizona College of Medicine Phoenix, Phoenix, and 3 Division of Gastroenterology and Hepatology, Mayo Clinic, Scottsdale, Arizona, USA

SUMMARY. Acute esophageal food impaction (AEFI) occurs frequently. Few data are published describing the use of effervescent agents (EAs) for treatment of AEFI. We aimed to evaluate the effectiveness, cost, and safety of EAs in the treatment of AEFI. We retrospectively identified patients aged 18 years and older who were seen in the emergency department of 2 hospitals in 1 metropolitan area from January 1, 2011, through April 4, 2016, who had a clinical diagnosis of AEFI. We collected and analyzed data on outcomes and cost associated with the use of EAs, glucagon, and no pharmacologic therapy. During the study period, 239 patients with AEFI met the inclusion criteria. Of the 45 patients who received EA monotherapy, 25 (55.6%) responded successfully, compared with 11 of 62 (17.7%) who received glucagon monotherapy (P < .001) and 16 of 93 (17.2%) who had no therapy (P < .001). Ten of 39 patients (25.6%) who were given both glucagon and EA responded successfully. The other 177 patients had endoscopy, which was successful in all cases. Median hospitalization charges for patients who responded successfully to EA alone were $1,136, compared with $2,602 for responders to glucagon alone (P < .001) and $1,194 for those who cleared their bolus spontaneously (P < .001). All patients who received EA monotherapy had lower median hospitalization costs ($2,384) than all patients who received glucagon monotherapy ($9,289; P = .03) and all patients who received neither ($8,386; P = .02). Effervescent agents are a safe, effective, and cost-saving initial strategy in the treatment of acute esophageal food impaction.

KEY WORDS: dysphagia, endoscopy, esophagus, foreign bodies, swallowing disorders.

INTRODUCTION

Acute esophageal food impaction (AEFI) is a common occurrence and frequently requires emergent endoscopy. The annual incidence rate was estimated to be 13.0 per 100,000 in the mid-1990s 1 and has been increasing in association with the increasing incidence of eosinophilic esophagitis. 2,3 The most recent American Society for Gastrointestinal Endoscopy guidelines from 2011 recommend emergent endoscopy in patients with AEFI who have complete esophageal obstruction and are not able to manage secretions. Despite mixed results in studies of the use of glucagon for AEFI, the guidelines also state that 1 mg of intravenous glucagon is relatively safe and remains an acceptable option, as long as definitive endoscopic therapy is not delayed. Effervescent agents (EAs), however, may be a more effective means of treating AEFI than glucagon and may be equally safe and cost effective. The guidelines do not comment on the use of EAs. 4

EAs consist of granulated sodium bicarbonate, citric or tartaric acid, and simethicone; when combined with water, they immediately form CO2 gas. When given to a patient with a food bolus impaction, the assumed mechanism of action is that the gas increases intraluminal pressure in the esophagus against a closed upper esophageal sphincter, thereby propelling the food bolus downward into the stomach.
The existing data on EAs are older, and study sample sizes are small. A few small case series from the 1980s and early 1990s evaluated the use of EAs and demonstrated good success rates.5,6

We hypothesized that EAs could be used to successfully treat AEFI, which would thereby avoid the need for emergent endoscopy and allow for earlier disimpaction. Therefore, we sought to evaluate the efficacy of EAs in patients with AEFI seen in the emergency department (ED).

Secondary goals were to compare the efficacy of EAs versus glucagon; determine and compare costs associated with the use of EAs, glucagon, or no pharmacologic therapy; and determine whether any patient characteristics can predict successful treatment of AEFI with EAs.

**MATERIALS AND METHODS**

**Data extraction**

This was a retrospective cohort study at a large, tertiary medical center (Banner University Medical Center, Phoenix, Arizona) involving all adult patients (age ≥ 18 years) seen in the ED between January 1, 2011, and April 4, 2016, who had AEFI. AEFI was identified by using pertinent International Classification of Diseases (ICD), Ninth Revision and Tenth Revision codes: ICD-9 code 935.1 (foreign body in esophagus) and ICD-10 codes T18.128A (food in esophagus causing other injury-initial encounter), T18.198A (other foreign object in esophagus causing other injury-initial encounter), and T18.108A (unspecified foreign body in esophagus causing other injury-initial encounter). We obtained the same data from a smaller, affiliated community hospital (Banner Estrella Medical Center, Phoenix, Arizona) from January 1, 2015, through April 4, 2016, because the same emergency physician group staffed both hospitals during these time periods and had been introduced to the concept of using EAs for AEFI several years earlier. We excluded cases in which the ingestion was not boneless food impaction. The institutional review board of Banner University Medical Center-Phoenix approved the study protocol on April 5, 2016.

**Data collected**

We collected data on demographics, clinical characteristics, treatments, outcomes, and costs in all patients with AEFI during the study period at each hospital. AEFI was defined as the inability to tolerate swallowing liquids without regurgitation in patients who presented with an appropriate clinical history. Demographic and clinical data included age, sex, race, body mass index (BMI), history of gastroesophageal reflux disease, history of prior AEFI, and time from impaction to intervention. We also collected data on prior endoscopic findings, endoscopic findings from the examined encounter, and adverse events. Treatments for AEFI were categorized as EAs (E-Z-GAS II; Bracco Diagnostics), glucagon, both, or neither of these agents.

**Outcomes**

A successful treatment outcome for AEFI was defined as clearance of the food impaction avoiding the need for emergent endoscopy. Clearance was measured by the patient’s reporting a sensation of bolus passage and the witnessed ability to swallow secretions and liquids. Cost was measured as the total hospital charges as reported by the medical records department.

**EA administration technique**

Emergency medicine physicians who were originally interested in using EAs were instructed to pour 1 or 2 packets of E-Z-GAS II into a medicine cup. The patient, once placed fully upright and with an emesis basin in hand in case of failure, was instructed to pour the contents quickly onto the back of their tongue and immediately chase it with water, swallowing with and maintaining a chin-tuck position. Patients were encouraged to try to hold the mixture in their esophagus, resisting the urge to belch or regurgitate, for as long as possible.

**Statistical analysis**

We first stratified the overall study sample of AEFI cases into 4 groups on the basis of initial treatment: EAs, glucagon, no pharmacologic therapy (i.e. neither EAs nor glucagon), and EAs plus glucagon. We compared demographics, clinical characteristics, outcomes, and costs among the groups. For all inferential statistics, we analyzed only the 3 groups that received either no treatment or only 1 medication (monotherapy). We used independent t tests or 1-way analysis of variance (or Kruskal-Wallis test when appropriate) to compare continuous variables and χ² tests (or Fisher exact tests, when appropriate) for categorical variables. Variables associated with treatment success (avoidance of endoscopy) with \( P < .10 \) were entered into multiple logistic regression to determine independent predictors of treatment success. The final multivariable models included only variables with a final adjusted \( P \) value < .05. For univariate analyses, a two-tailed \( P < .05 \) was considered significant. IBM SPSS Statistics for Mac, version 24.0. (IBM Corp) was used for analysis.

**RESULTS**

**Demographics**

The search identified 291 ED encounters for AEFI, 52 of which were excluded; 239 met all inclusion
Table 1 Demographics and clinical characteristics stratified by treatment group

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>EA monotherapy (N = 45)</th>
<th>Glucagon monotherapy (N = 62)</th>
<th>No treatment (N = 93)</th>
<th>Glucagon + EAs (N = 39)</th>
<th>(P) value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, year</td>
<td>55.6 (19.5)</td>
<td>51.9 (18.5)</td>
<td>61.7 (20.1)</td>
<td>54.0 (19.5)</td>
<td>0.01(\dagger)</td>
</tr>
<tr>
<td>Male</td>
<td>30 (66.7)</td>
<td>44 (71.0)</td>
<td>62 (66.7)</td>
<td>24 (61.5)</td>
<td>0.81</td>
</tr>
<tr>
<td>BMI, kg/m(^2)</td>
<td>28.2 (7.8)</td>
<td>27.2 (7.2)</td>
<td>26.8 (4.5)</td>
<td>28.0 (5.7)</td>
<td>0.56</td>
</tr>
<tr>
<td>Prior AEFI</td>
<td>19 (45.7)</td>
<td>27 (43.5)</td>
<td>42 (45.7)</td>
<td>15 (39.5)</td>
<td>0.94</td>
</tr>
<tr>
<td>Time to therapy, hour</td>
<td>10.0 (15.5)</td>
<td>10.0 (11.7)</td>
<td>– 9.4 (8.0)</td>
<td>9.4 (8.0)</td>
<td>0.96</td>
</tr>
<tr>
<td>GERD history</td>
<td>15 (33.3)</td>
<td>14 (22.6)</td>
<td>33 (35.5)</td>
<td>12 (30.8)</td>
<td>0.39</td>
</tr>
<tr>
<td>White(\S)</td>
<td>31 (68.9)</td>
<td>44 (71.0)</td>
<td>66 (71.0)</td>
<td>33 (84.6)</td>
<td>0.34</td>
</tr>
</tbody>
</table>

\(\dagger\) Values are mean (SD) or number of patients (%);  
\(\S\) No treatment versus any treatment;  
\(\S\) Race was reported by the patients.

AEFI, acute esophageal food impaction; BMI, body mass index; EA, effervescent agents; GERD, gastroesophageal reflux disease.

Fig. 1 Flow chart of therapy and outcomes. EA indicates effervescent agent; EGD, esophagogastroduodenoscopy.

criteria. About two-thirds of patients were men (160, 67%), and most were white (174, 73%). The mean (SD) age was 56.8 (19.8) years, and the mean body mass index was 27.4 (6.1) kg/m\(^2\). Approximately 31% \(n = 74\) had a history of gastroesophageal reflux disease, and 43% \(n = 103\) had had prior AEFI. The cause of AEFI, when identified, was determined by documented history or endoscopic findings. Peptic strictures accounted for most impactions (75, 31.4%), followed by erosive esophagitis (47, 19.7%) and eosinophilic esophagitis (47, 19.7%). Schatzki rings were present in 8.8% of patients \(n = 21\). The rest of the patients had no documented history or pertinent endoscopic findings recorded. Median (range) time from food impaction to intervention was 5.0 (0.5–72.0) hours.

Treatment for AEFI was EA monotherapy in 45 patients, glucagon monotherapy in 62, both EA and glucagon in 39, and no treatment in 93. Demographics stratified by mode of treatment are shown in Table 1. Notably, patients in the ‘No Treatment’ group were significantly older than patients who received at least one form of medical therapy.

**Outcomes**

Overall, 62 patients (21.3%) cleared their food impactions without the need for endoscopic intervention (Fig. 1). More than 3 times as many patients who received EA monotherapy had a successful outcome (55.6%) as patients who received glucagon monotherapy (17.7%; \(P < .001\)) and patients who were observed while awaiting endoscopic therapy (17.2%; \(P < .001\)) (Fig. 2). Twice as many patients with EA monotherapy (55.6%) had a successful outcome as patients who received both glucagon and EA (25.6%; \(P = .008\)). Data were incomplete regarding the order of medication administration, but in the majority with data available, glucagon was given first, followed by EA once glucagon was deemed to have failed.

Among the 177 patients who required endoscopic intervention, 100 had endoscopy after failure of EA
and/or glucagon treatment, and 77 had endoscopy without having been given either EA or glucagon. Endoscopy was successful in clearing the food impaction in all patients (Fig. 1). There were no reported instances of the endoscopy having been affected by the administration of EA or glucagon.

**Cost**

Total hospital charges for patients who had successful outcomes with any medical therapy were lower than for those who required endoscopy. Furthermore, EA administration was associated with lower cost than non-administration. The 25 patients who had a successful outcome with EA monotherapy had median (range) total charges of $1136 ($438–$4127), compared with $2602 ($1464–$4939) for the 11 patients who responded to glucagon monotherapy (P < .001) and $1194 ($555–$10,033) for the 16 patients who passed their impaction without specific therapy (P < .001). A cost advantage for EAs persisted when outcome was disregarded. Patients who received EA monotherapy had median (range) total charges of $2384 ($438–$60,781), compared with $9289 ($1464–$61,930) for all patients who received glucagon monotherapy (P < .001) and $8386 ($555–$59,055) for patients who received no pharmacologic therapy (P = .02).

**Predictors of response**

When we combined the EA monotherapy and glucagon monotherapy data sets (n = 107), independent predictors of response included shorter duration between food impaction and intervention, no prior AEFI, and use of EAs (Table 2). In multivariable adjusted analysis, EA monotherapy was more than 6 times more likely to be associated with treatment success compared with glucagon monotherapy (odds ratio, 6.53; 95% CI, 2.42–17.6; P = .003) (Table 2).

Among only patients who received EA monotherapy, several factors were significantly different between those who did and did not have successful outcome: BMI (P = .02) and time to therapy (P = .002) (Table 3). Higher BMI (P = .03), no prior AEFI (P = .03), and shorter time to therapy (P = .02) were also independent predictors on unadjusted analysis (Table 4). On multiple regression analysis, however, only shorter time to intervention independently predicted treatment success (odds ratio per 1-hour increase, 0.85; 95% CI, 0.74–0.98; P = .02) (Table 4).

**Adverse events**

Only 1 adverse event occurred in the cohort. A mucosal tear occurred in an 85-year-old woman with dementia who was given EAs (with unsuccessful results) after seeking care 4 hours after her impaction. Chest radiography performed about 1 hour after she was given EAs was unremarkable. She subsequently

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**Table 3** Demographics and clinical characteristics stratified by outcome after treatment with EA monotherapy (N = 45)

<table>
<thead>
<tr>
<th>Clinical characteristic</th>
<th>Success (n = 25)</th>
<th>Failure (n = 20)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, year</td>
<td>51.2 (18.5)</td>
<td>61.1 (19.8)</td>
<td>0.09</td>
</tr>
<tr>
<td>Male</td>
<td>15 (60.0)</td>
<td>15 (75.0)</td>
<td>0.35</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>30.6 (8.7)</td>
<td>25.3 (5.2)</td>
<td>0.02</td>
</tr>
<tr>
<td>Prior AEFI</td>
<td>7 (29.2)</td>
<td>12 (60.0)</td>
<td>0.07</td>
</tr>
<tr>
<td>Time to therapy, hour</td>
<td>6.3 (14.5)</td>
<td>14.7 (15.8)</td>
<td>0.002</td>
</tr>
<tr>
<td>GERD history</td>
<td>6 (24.0)</td>
<td>9 (45.0)</td>
<td>0.21</td>
</tr>
</tbody>
</table>

AEFI, acute esophageal food impaction; BMI, body mass index; EA, effervescent agents; GERD, gastroesophageal reflux disease.

**Table 2** Multivariable adjusted logistic regression analysis of predictors of treatment success with EA or glucagon monotherapy (N = 107)

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Unadjusted</th>
<th>Multivariable adjusted</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Odds ratio (95% CI)</td>
<td>P value</td>
</tr>
<tr>
<td>Age (per 1 year increase)</td>
<td>0.99 (0.97–1.01)</td>
<td>0.20</td>
</tr>
<tr>
<td>BMI (per 1.0 kg/m² increase)</td>
<td>1.04 (0.99–1.10)</td>
<td>0.13</td>
</tr>
<tr>
<td>Prior AEFI</td>
<td>0.32 (0.13–0.77)</td>
<td>0.01</td>
</tr>
<tr>
<td>Time to therapy (per 1 hour increase)</td>
<td>0.95 (0.91–0.99)</td>
<td>0.04</td>
</tr>
<tr>
<td>EA therapy</td>
<td>5.80 (2.41–13.9)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

AEFI, acute esophageal food impaction; BMI, body mass index; EA, effervescent agents.
underwent esophagogastroduodenoscopy, and an 8-cm full-thickness tear in the mid esophagus was described, but no food impaction was seen. On subsequent computed tomography, a mediastinal leak was demonstrated. She was treated nonoperatively with no oral intake and intravenous antibiotics and had a full recovery.

DISCUSSION

To our knowledge, this study represents the largest reported experience with the use of EAs in the treatment of AEFI. The results suggest that using EAs as initial therapy to dislodge AEFI is effective in more than 50% of cases. These numbers support previous results from the literature that also suggest high success rates. In 1983, Rice et al. described a 100% success rate in 8 patients treated with tartaric acid and sodium bicarbonate with no adverse events. In a retrospective review, Zimmers et al. described a 65% success rate in 26 patients who received a combination of tartaric acid and sodium bicarbonate. Reports from the radiology literature have described combining contrast agents, EAs, and glucagon, with success rates of about 70%. Patients in these studies were also given either barium or water-soluble contrast agents for esophagography before therapeutic intervention. It was postulated that the weight of the barium column could have contributed to clearance of the food impaction. Current recommendations are to avoid using barium as it can hinder subsequent endoscopy, if necessary.

The results of this study showed clear superiority of EAs over glucagon in the treatment of AEFI. In fact, glucagon efficacy was comparable to that of observation and spontaneous passage in our data set. The use of glucagon for AEFI has long been debated in the literature since it was first described in 3 patients in 1977. In healthy patients, glucagon has been demonstrated to decrease lower esophageal sphincter resting pressure and relaxation without affecting amplitude or duration of esophageal contraction. More recent studies have demonstrated a wide but suboptimal range of efficacy for glucagon from 9.2% to 39.2%. In this study, glucagon worked only 17% of the time, which was the same success rate as observation. Patients who received both glucagon and EAs had success rates higher than glucagon alone, but lower than those who received EAs alone. One possible explanation is that glucagon could theoretically impair the ability of EAs to generate sufficient intraluminal pressure to dislodge the bolus.

Any success with pharmacologic therapy was associated with a significant decrease in cost because of the avoidance of urgent endoscopy and its associated costs. Many of these patients, however, should be advised to undergo elective endoscopy. Theoretically, an elective endoscopy, especially one done in an ambulatory surgery center, would be less costly than one done in the hospital setting during an emergency visit. Also, some patients who present with AEFI have already had endoscopy and have well-established esophageal diagnoses making subsequent elective endoscopy less important in this group.

Success with EAs was less costly than success with the use of glucagon or spontaneous passage, possibly because in addition to avoidance of endoscopy, the results of EA administration are known immediately, thereby decreasing the patient’s time spent in the ED, if successful. This compares with the several hours it can take for glucagon or observation to work. Most likely, the longer a patient stays in the ED, the higher the chances of ancillary testing that would add to the cost. This testing, however, usually adds little, if anything, to the patient’s clinical outcome. In this study, practice patterns varied among the treating ED staff for ordering laboratory tests, electrocardiography, and imaging studies, all of which added to the cost of the visit but did not seem to affect clinical outcome.

The only independent predictor of successful response to EAs that remained significant on multivariable adjustment with logistic regression was shorter time to intervention. Nevertheless, successful outcomes occurred with EA administration as much as 72 hours after onset of impaction. The aforementioned publications that showed high rates of efficacy for EAs also showed a correlation of shorter time intervals and success with EAs.

The only adverse event in our study was a mucosal tear in an 85-year-old woman who was treated unsuccessfully with EA (4 hours after impaction), followed by endoscopy. The literature describes only 1 other mucosal tear in a 60-year-old woman who was given

Table 4 Multivariable adjusted logistic regression analysis of predictors of EA monotherapy treatment success (N = 45)

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Unadjusted Odds ratio (95% CI)</th>
<th>P value</th>
<th>Multivariable adjusted Odds ratio (95% CI)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (per 1 year increase)</td>
<td>0.97 (0.94–1.01)</td>
<td>0.10</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI (per 1.0 kg/m² increase)</td>
<td>1.13 (1.01–1.26)</td>
<td>0.03</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prior AEFI</td>
<td>0.28 (0.08–0.96)</td>
<td>0.04</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time to therapy (per 1 hour increase)</td>
<td>0.85 (0.74–0.98)</td>
<td>0.02</td>
<td>0.85 (0.74–0.98)</td>
<td>0.02</td>
</tr>
</tbody>
</table>

AEFI, acute esophageal food impaction; BMI, body mass index; EA, effervescent agents.
EAs 18 hours after impaction.\textsuperscript{6} It is important to note that EAs may not be entirely without risk given the proposed mechanism of action. Consideration to withhold it should be given to patients who would be at higher risk of aspiration or perforation. This may include patients who are very elderly or demented, as well as those with known eosinophilic esophagitis or those who present with longer time interval from food impaction.

A strength of our study is the large, consecutive series of patients from 2 sister institutions staffed by the same ED physician group that had been familiar with using EAs. We postulate that success with EAs depends not only on patient characteristics, but also on proper technique of administration, coaching on the part of the ED physician, and patient motivation to swallow and hold the EAs as long as possible.

A limitation of this study is its retrospective design. Also, practice patterns within the ED physician group were heterogeneous as to use of EAs, glucagon, and other agents as well as their propensity to order ancillary laboratory tests and imaging studies. This made it difficult to control for those variables, which could have affected outcomes in terms of success and cost. Further, since patients in the 'no treatment' group were significantly older than those who received at least one form of medical therapy, it stands to reason that the treating ER physicians may have selected against giving older patients EA or glucagon perhaps due to a fear of adverse events. It is possible this could have affected the success rates of EAs and/or glucagon.

Regarding the use of EAs, there was little, if any, description of the technique by the ED physician as to how many packets were administered, whether the patient was given proper instructions on how to take it and whether those instructions were followed, all of which may have affected the potential success of its use. Also, we did not consider possible effects of other agents that were used, including nitrates, benzodiazepines, calcium channel blockers, narcotics, antiemetics, and sodas. In one study, the use of benzodiazepines and nitrates did not affect the success rates of glucagon.\textsuperscript{13} Moreover, these agents were used in relatively few patients in our study, so we elected not to account for them.

CONCLUSION

EAs are an effective, cost-saving, and safe initial treatment strategy for AEFI. We recommend using EAs in adult patients with AEFI. Prospectively collected data using a standardized EA administration protocol with trained providers, and a lack of age disparity among treatment groups, would improve the quality of data with regard to EA use in AEFI and would potentially strengthen this recommendation.

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References