Caustic Ingestions Mimicking Anaphylaxis: Case Studies and Literature Review

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abstract

Anaphylaxis presents in children with rapid involvement of typically 2 or more organ systems including cutaneous, gastrointestinal, and respiratory. Caustic ingestions (CI) may also present with acute involvement of cutaneous, gastrointestinal, and respiratory systems. We present 2 cases of "missed diagnosis" that illustrate how CI presenting with respiratory symptoms can be mistaken for anaphylaxis owing to these similarities. Both of these patients had delay in appropriate care for CI as a result. These cases demonstrate the importance of considering CI in children who have gastrointestinal symptoms, respiratory distress, and oropharyngeal edema.

Anaphylaxis presents with a combination of gastrointestinal, cardiovascular, respiratory, and cutaneous symptoms.[1] The estimated incidence of pediatric anaphylaxis is ~1 per 1000 children per year.[2] Caustic ingestions (CI) also present with respiratory distress, vomiting, and oral edema. The incidence of CI is lower, at ~1 per 100 000 children per year.[3] Many CI cases are accidental; bleach, disinfectants, and laundry detergents are the most common agents.[4] We report 2 children misdiagnosed with anaphylaxis after unrecognized CI.

CASE DESCRIPTIONS

Patient A

Patient A was a 5-year-old boy who had asthma and eczema who presented with coughing and lip and tongue swelling immediately after eating dinner. Over the following 4 to 6 hours, he developed vomiting, drooling, and audible wheezing. His mother called emergency medical services and reported that he was having trouble breathing. Initial emergency department (ED) examination revealed respiratory distress, stridor at rest, drooling, and an oxygen saturation of 89% by pulse oximetry. In the ED, neck films revealed minimal tracheal narrowing and epiglottal thickening. He was given dexamethasone, diphenhydramine, and nebulized racemic epinephrine without improvement. His respiratory status worsened, requiring intubation. A swollen glottis was noted during the procedure.

He was transferred to our tertiary care hospital for intensive care management. Because of his atopic medical history and presentation, he was tentatively diagnosed with refractory anaphylaxis and given diphenhydramine and intramuscular (IM) epinephrine. Examination revealed tongue edema and evidence of stomatitis, atypical for anaphylaxis but concerning for infection or trauma. His mother denied awareness of chemical ingestions or recent fever. A rapid screen for group A β-hemolytic *Streptococcus* was positive. A diagnostic evaluation for allergic reaction returned a negative tryptase level and negative serum-specific

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immunoglobulin E to foods he recently ingested. The working diagnosis changed from anaphylaxis to streptococcal oropharyngeal infection, amoxicillin was started, and antihistamines and steroids were discontinued after 48 hours of treatment. On the third day of admission he was extubated to room air and transferred to the hospital medicine service for further observation. The stomatitis failed to improve despite appropriate antibiotic therapy. On day 10 of illness, the team attempted a nasogastric tube insertion for supplemental nutrition; however, the patient immediately developed hemoptysis. Then, for the first time, the patient's mother disclosed that he had inadvertently ingested detergent before the initial presentation.

A social worker learned that the mother's former reluctance to reveal the ingestion was based on a fear that her children would be removed from her care. An urgent esophagogastroduodenoscopy (EGD) showed diffuse circumferential esophageal ulceration and exudate consistent with CI. Microlaryngoscopy and nasopharyngoscopy were performed, identifying epiglottal and pharyngeal ulceration and friable mucosa consistent with this diagnosis (Figs 1 and 2). He was started on sucralfate and omeprazole for gastric protection. A nasojejunal (NJ) tube was placed for long-term nutritional supplementation. Over a year later he has had no recovery of esophageal motility.

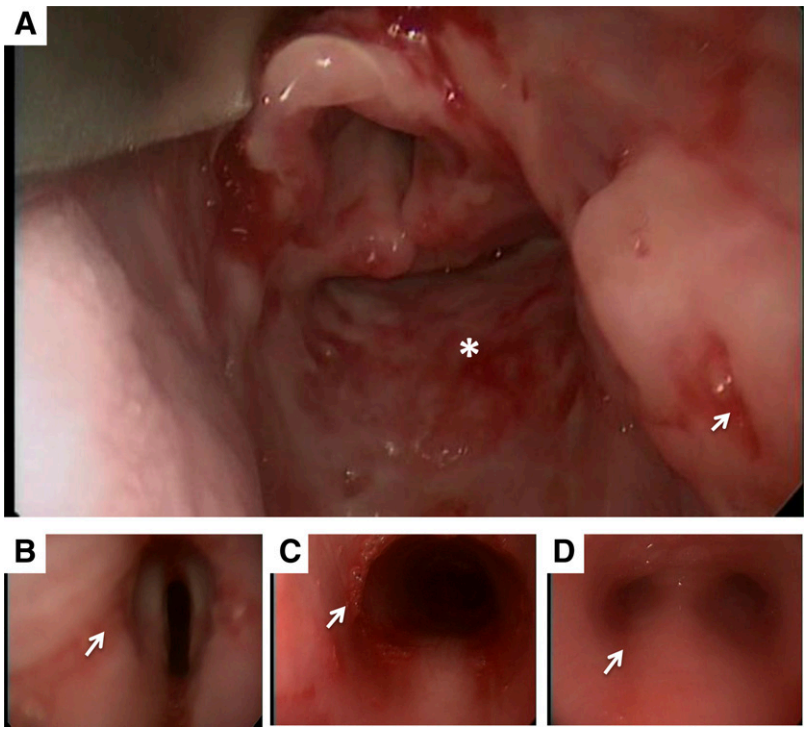

FIGURE 1

Microlaryngoscopy and rigid bronchoscopy at the initial presentation of Patient A depicting A, the supraglottic view; B, true vocal folds; C, subglottis; and D, carina. Blood-tinged secretions were noted throughout the examination (indicated by arrows) as well as ulceration and friable mucosa (indicated by the asterisk).

Patient B

Patient B was a 3-year-old boy who did not have any medical history. On the evening of presentation, he drank from a bottle of "peach tea" that was left on the kitchen counter. He quickly developed severe discomfort, retching, emesis, and lip and tongue edema. After an episode of hematemesis, his caregiver took him to an outlying hospital where he demonstrated stridor, retching/vomiting, petechiae, and oral mucosa and lip edema. Because of concern for anaphylaxis he was given a dose of nebulized racemic epinephrine, a treatment of nebulized albuterol, solumedrol, diphenhydramine, and ondansetron. A chest radiograph was obtained and showed aryepiglottic and epiglotic mucosal edema and tracheomalacia.

He was transferred to our ED where he received a dose of IM epinephrine owing to persistent respiratory distress. The family provided the bottle of "peach tea" to ED staff, which was then tested for pH, with a normal result of 5. Because of continued stridor, he was given a second dose of nebulized racemic epinephrine with some improvement in general appearance but he developed an oxygen requirement. He was admitted to the hospital medicine service for further monitoring.

After arrival to the floor he had continued respiratory distress and stridor that did not improve with an additional dose of IM epinephrine. He developed a fever. Examination was significant for denuded buccal mucosa with white exudate and drooling. This prompted urgent evaluation by nasopharyngolaryngoscopy owing to concern for CI. Direct visualization revealed erythematous and edematous tongue base, posterior pharyngeal wall, epiglottitis, aryepiglottic folds, and esophageal inlet. An EGD was performed and revealed burns to the oral mucosa and tongue, swelling of the epiglottis, diffuse thickening and sloughing of the esophagus, a large eschar on the posterior stomach wall, and pyloric edema, consistent with CI. He was started on pantoprazole, sulcralfate, and total parenteral nutrition to lower the risk for gastric perforation. One week later, a repeat EGD noted gastric ulcerations, total parenteral nutrition was stopped, and an NJ tube was placed for further nutrition. After

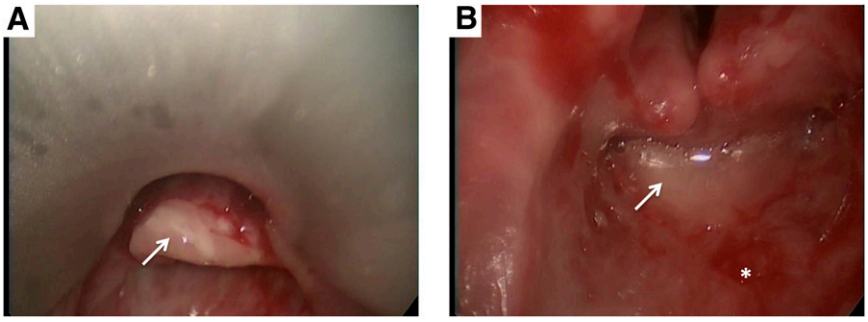

FIGURE 2
Close-up images of early mucosal injury after caustic ingestion in Patient A. A, Lingual surface of the epiglottis demonstrating white eschar (indicated by arrow). B, Posterior pharyngeal wall and hypopharynx demonstrating demucosalized surfaces (indicated by asterisk) and white eschar (indicated by arrow).

3 months, he had the NJ tube removed and no longer has dietary restrictions, and his last EGD showed evidence that the ulcer was healing. The family disclosed to a social worker that the patient had a previous accidental ingestion (oil) that did not undergo medical evaluation. The exact substance in the current case was never identified.

DISCUSSION

These patients demonstrate that, in the setting of an unclear history, CI can be mistaken for anaphylaxis owing to overlap in symptoms. Up to 53% of children who have anaphylaxis have gastrointestinal symptoms including nausea, vomiting, and trouble swallowing.[1] These symptoms are also present in over half of children who have CI with grade 2 or 3 esophageal lesions.[5] Likewise, respiratory symptoms are common to both diagnoses, with shortness of breath present in at least 30% of anaphylaxis patients and up to two-thirds of patients who have CI, particularly with severe damage.[1,5] Lastly, 33% of systemic allergic reactions present with gastrointestinal symptoms and 18% of CI have airway injury.[6,7] Ultimately these studies show that these 2 pathologies have similar symptoms and symptom rates (Table 1).

A literature review for this case series found that anaphylaxis was part of the differential diagnosis for a CI in 1 source; no sources identified CI as an alternative diagnosis to anaphylaxis.[8] Potential alternative diagnoses for anaphylactic symptoms were shock, carcinoid syndrome, excess histamine syndromes, hereditary angioedema, panic attack, and pheochromocytoma. Epiglottitis, esophagitis, gastroesophageal reflux disease, and perforated ulcer were the suggested alternative diagnoses for CI. Even with an overlap in presentation the differential diagnoses of CI and anaphylaxis rarely reference each other in the literature. This biases the clinician to think of these 2 pathologies as unrelated.

However, because of their similarities in presentation, CI should be considered an alternative explanation for presumed anaphylaxis, especially when patients fail to respond to appropriate management. Even if the clinician considers CI in the differential, there is currently no way to establish the diagnosis in the emergent setting if the caregiver does not report the ingestion. Chest and lateral neck radiographs can help provide information regarding perforation during the initial evaluation; however, this modality will not definitively diagnosis CI, as the findings may be similar to those expected with anaphylaxis.[7] The definitive diagnosis for CI occurs with endoscopy that identifies characteristic pathology. When performed within the first 72 hours after ingestion, this procedure stages pathology and identifies complications in symptomatic patients.[9,10] A promising alternative might be an emergent microlaryngoscopy and nasopharyngoscopy, a quick procedure that in both cases adequately identified the correct etiology. This procedure can be useful in the emergent setting, because it provides information about the patient's airway, a concern in almost a quarter of CI.[7] Unfortunately there is insufficient evidence to promote this procedure to rule out CI in either the emergent or long-term periods; microlaryngoscopy also carries risks associated with general anesthesia.

A prompt diagnosis is important in both of these conditions. Current data indicate that instituting early anaphylaxis therapy, including IM epinephrine, improves outcomes.[1] No such treatment protocol exists for CI. The only management that has reached consensus is to avoid ipecac, because it leads to esophageal

TABLE 1 A Comparison of Common Symptom Percentages in Caustic Ingestions and Anaphylaxis

	Caustic Ingestion			Anaphylaxis	
	Gaudreault et al[15]	Gupta et al[10]	Riffat et al[7]	de Silva et al[6]	Ross et al[16]
Number of total cases	298	28	50	123	141
Vomiting	33	11	48	26	16
Dysphagia	25	—	—	—	—
Abdominal pain	24	—	—	3	—
Refusal to eat/drink	20	14	76	—	—
Respiratory symptoms	—	11	—	97	30
Mucosal edema	—	—	40	55	—
Drooling	24	75	56	—	—

All data are presented as percentages unless otherwise noted.

re-exposure of the ingested substance, and to initiate acid-blocking medications early to prevent reflux-associated injury.[11] In this setting, nasogastric tubes are necessary for nutritive and protective purposes; however, nasogastric tube insertion should be cautiously performed immediately after ingestion owing to the perforation risk. Corticosteroids have unproven efficacy in this setting and should be cautiously used.[7] Although they may reduce strictures in third-degree esophageal injuries, corticosteroids may also increase the chance of esophageal perforation.[12–14] Social workers were involved in both cases to facilitate discussion about the patient's care after discharge, an important part of many CI cases.

Furthermore, the ability to distinguish these 2 pathologies is also needed in developing nations as well as the United States. In these countries CI are more common than in westernized nations, in part because of the use of secondary containers and the availability of chemicals around the home.[15] In addition, with the westernization of developing nations and a rise in atopic diseases where food allergy was previously uncommon, the need to differentiate between CI and anaphylaxis in these settings is increasingly important.[16]

CONCLUSIONS

Thorough history and physical examination will correctly identify many cases of CI and anaphylaxis. The 2 reported cases demonstrate the importance of evaluating for potential CI in patients who present with presumed anaphylaxis, particularly when there is no history of food allergy or an atypical course, because both may present with gastrointestinal and respiratory symptoms. Lastly, these cases indicate the need for a diagnostic test, such as a microlaryngoscopy and nasopharyngoscopy, to quickly distinguish anaphylaxis from CI.

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