Tracheobronchial Stenosis From Acid Aspiration Presenting as Asthma*

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We report a case of a 42-year-old man who fell in a vat of hydrochloric acid, resulting in ingestion and aspiration of acid. Initially, he suffered from a chemical pneumonitis and GI burns. He was released from the hospital without complications, only to return with signs and symptoms consistent with asthma. Evaluation revealed multiple areas of large airway stenosis, resulting from the chemical burns. The stenoses were treated with multiple stents.

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Key words: aspiration; hydrochloric acid; stenosis; stenting

Abbreviations: DLCO = diffusion capacity of the lung for carbon monoxide; PFT = pulmonary function test; RADS = reactive airways dysfunction syndrome; RV = residual volume

Reactive airways dysfunction syndrome (RADS) is commonly reported after exposure to toxic chemicals. Included in the definition of RADS are asthma-like symptoms such as cough, wheezing, and dyspnea. These symp-

toms usually present within hours of exposure and dissipate over time. Pulmonary function test (PFT) results reveal obstructive defects that on repeat measurements resolve. Several reports of RADS following exposure to chlorine gas have been reported in the literature. Despite chlorine gas and hydrochloric acid having common toxic pathways, different injury patterns are possible. We present an unusual case of a man exposed to hydrochloric acid resulting in airway strictures, not RADS, requiring airway stenting for treatment.

CASE REPORT

The patient is a 42-year-old African-American steel mill worker without a significant medical history, except for a 22-pack-year history of tobacco use, who was referred to pulmonary clinic for treatment of uncontrolled asthma. The patient was referred by his primary care physician, complaining of progressive shortness of breath and wheezing 2 months after an industrial accident.

At that time, the patient fell into a vat of 35% hydrochloric acid. He was completely submerged, and ingested and aspirated some of the acid. He denied loss of consciousness and was able to remove himself from the acid, and coworkers rapidly decontaminated him with large volumes of water. He was hospitalized for further treatment.

His main complaint at the time of presentation was dyspnea and generalized first-degree burns. Examination revealed stridor, at which point intubation was attempted. Examination revealed significant edema and bleeding of his oropharynx. Due to the extent of swelling, an emergent cricothyroidotomy was performed to establish airway access. The patient was admitted to the burn unit and stabilized. CT of the chest, abdomen, and pelvis ruled out a perforation but revealed bilateral lower lobe pneumonia (Fig 1, top left; A; center left; D; bottom left, G). The patient’s cricothyroidotomy was converted to a tracheostomy, during which direct laryngoscopy and fiberoptic bronchoscopy were performed. The patient was noted to have a large amount of edema of the posterior oropharynx, subglottic wall, and uvula. Large amounts of blood mixed with pulmonary secretions were in the airways, but no obvious mucosal lesions were seen in the distal tracheal or segmental bronchi. Esophagogastroduodenoscopy revealed swollen and partially denuded portions of the esophagus and stomach with some ulcerations. Despite significant injuries, the patient did well clinically. His hematocrit fell acutely from 44 to 27% due to chemical burns in the GI tract. He was initially kept on the ventilator for airway protection and chemical pneumonitis. He was liberated from the ventilator within 3 days, and his tracheostomy was decannulated on day 13. The patient was discharged home on hospital day 14 performing all activities without distress or shortness of breath. He was eating and speaking without difficulty. His only medication was acetaminophen.

The patient did well for approximately 1 month, when he began experiencing intermittent shortness of breath with exertion. Given these complaints, his primary physician started him on inhaled bronchodilators for reactive airways disease, without improvement. His symptoms progressed to the point that he became short of breath with simple activities such as walking across a room or getting dressed. He was reevaluated and found to be in mild distress with significant inspiratory and expiratory wheezing and diminished air movement. He was started on prednisone and continued on bronchodilators with the presumed diagnosis of reactive airway disease/asthma induced by his chemical burns vs COPD flare given his history of tobacco use. The patient had no prior history of asthma or COPD. He was started on broad-spectrum antibiotics to treat pneumonia vs tracheo-

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Spirometry revealed an FEV1 of 1.33 L (31% predicted) and FVC of 4.93 L (77% predicted), with a ratio of 67%. Total lung capacity was 7.96 L (95% predicted), with a ratio of 67%. DLco was 24.5 mL/min/mm Hg (70.6% predicted), total lung capacity was 7.96 L (95% predicted), and RV was 2.99 L (112% predicted). These tests revealed normal limits. Spironometry revealed an FEV1 of 1.33 L (31% predicted) and FVC of 4.93 L (77% predicted), with a ratio of 67%. Total lung capacity was 7.96 L (95% predicted), with a ratio of 67%. DLco was 24.5 mL/min/mm Hg (70.6% predicted), total lung capacity was 7.96 L (95% predicted), and RV was 2.99 L (112% predicted). These tests were normal.

Admission laboratory test results were all within normal limits. Spironometry revealed an FEV1 of 1.33 L (31% predicted) and FVC of 4.93 L (77% predicted), with a ratio of 67%. Total lung capacity was 7.96 L (95% predicted), with a ratio of 67%. DLco was 24.5 mL/min/mm Hg (70.6% predicted), total lung capacity was 7.96 L (95% predicted), and RV was 2.99 L (112% predicted). These tests were normal.

On examination, vital signs were normal, with an oxygen saturation of 98% on 2 L of oxygen via nasal cannula. There was no respiratory distress at rest, but with exertion there was dyspnea with accessory muscle use. Chest examination revealed diffuse inspiratory and expiratory wheezing throughout with diminished air movement. Cardiovascular examination revealed a grade 1 systolic murmur. No residual oropharyngeal or cutaneous scarring was noted.

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Pertinent history includes only a 22-pack-year history of smoking with none since the accident. Medications were prednisone, 30 mg qd; albuterol/ipratropium via metered-dose inhaler qid; and amoxicillin/clavulanate, 875 mg bid.

The patient failed to respond and was hospitalized for management after evaluation in the pulmonary clinic. Pertinent history includes only a 22-pack-year history of smoking with none since the accident. Medications were prednisone, 30 mg qd; albuterol/ipratropium via metered-dose inhaler qid; and amoxicillin/clavulanate, 875 mg bid.

Approximately 1 week later, the patient returned with increasing shortness of breath. Repeat CT scan revealed persistent stenoses at several sites. Given his rapidly progressive dyspnea, the patient was emergently taken to the operating room, where self-expandable metal wall stents were inserted to maintain airway patency. The patient then underwent several successive rounds of bronchoscopy with stent placement. Eventually, the patient had five self-expandable metal wall stents placed, located in the left mainstem bronchus extending to the distal trachea, left lower lobe, right bronchus intermedius, and right middle and lower lobes (Fig 1, top right, C; center right, F; and bottom right, I). Currently, the patient feels well, performing all activities without difficulty. He was scheduled to return for follow-up and repeat dilatation.

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During this time, the patient underwent several successive rounds of bronchoscopy with stent placement. Eventually, the patient had five self-expandable metal wall stents placed, located in the left mainstem bronchus extending to the distal trachea, left lower lobe, right bronchus intermedius, and right middle and lower lobes (Fig 1, top right, C; center right, F; and bottom right, I).

CT images immediately after the accident (top left, A; middle left, D; bottom left, G), on presentation 2 months after the accident (top center, B; middle center, E; bottom center, H), and status after placement of multiple stents (top right, C; middle right, F; bottom right, I). The representative images are taken at the levels of the distal trachea (top left, A; top center, B; top right, C), the main carina (middle left, D; middle center, E; middle right, F), and the right middle/lower lobe bronchi (bottom left, G; bottom center, H; bottom right, I). Note the pneumonitis present predominantly in the lower lobes immediately after the accident (bottom left, G) that resolved subsequently (bottom center, H; bottom right, I). The marked narrowing of the airways are best seen at the trachea (top center, B) and the carina (middle center, E) when compared to his initial presentation (top left, A; and middle left, D, respectively). The corrections of these strictures by stenting are seen in images at top right, C, and middle right, F.

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were consistent with a mild obstructive defect and a mild gas transfer defect. There were significant improvements in flows and air trapping.

**Discussion**

To our knowledge, this is the first report of hydrochloric acid aspiration resulting in distal tracheobronchial structures. The initial event of acid immersion was clearly associated with a chemical pneumonitis and acid damage to the oropharynx, GI, and respiratory tracts, and was treated acutely with tracheostomy and brief mechanical ventilation. Approximately 6 weeks after the accident, following resolution of his initial symptoms, dyspnea on exertion developed along with wheezing unresponsive to therapy for asthma. PFT results revealed a moderate obstructive ventilatory defect, elevated RV, and a moderate gas transfer defect consistent with asthma and/or COPD. However, chest CT revealed multiple areas of stenosis in large airways. The degree of stenosis was of sufficient severity to account for many of the patient's symptoms and lung function abnormalities. Following an attempt at dilation that brought only temporary relief, multiple stents were placed during several procedures with marked improvement in symptoms, wheezing, and dyspnea on exertion. Tests of lung function improved with decreased airflow obstruction and RV.

Figure 1 presents CT views of the distal trachea (top left, A; top center, B; top right, C), main carina (middle left, D; middle center, E; middle right, F), and right middle/lower lobe bronchi (bottom left, G; bottom center, H; bottom right, I), Top left, A; middle left, D; and bottom left, G reflect the findings seen at the initial presentation; top center, B; middle center, E; and bottom center, H, show findings at the time of new-onset asthma; and top right, C; middle right, F; and bottom right, I show findings following multiple airway stent placements.

Prior reports of acid exposure have only included a few cases of liquid aspiration, none involving hydrochloric acid. Case reports have described exposure to hydrofluoric acid and formic acid resulting from splashes. Victims in these case reports describe chemical pneumonitis and reactive airways disease that resolve over time.1,2

The most common exposure reported in the literature is acid exposure occurring by inhalation of gas fumes. A prior report describes exposure to nitric acid fumes resulting in the death of three pulp mill workers as a result of fatal pulmonary edema.3 Though there are no prior reports of hydrochloric acid inhalation, it is believed that hydrochloric acid and chlorine gas share a final common toxic pathway.4 The sequelae of such inhalation exposures to chlorine gas have included chemical pneumonitis and airway hyperactivity with RADS. Complete resolution of symptoms is usual, often within hours of exposure. Hasan et al5 describe 18 individuals exposed to chlorine gas leaking from a storage tank. The major respiratory complaints were cough or shortness of breath. All 18 individuals had evidence of airway obstruction and impaired gas exchange. Obstructive abnormalities resolved within 1 week in patients with cough, while slower resolution occurred in those patients whose main complaint was dyspnea. Limited long-term follow-up in 9 of the 18 subjects 5 months after exposure revealed normal spirometry and DLco findings. These findings are similar to those in a study by Abhyankar et al6 that describes chlorine gas exposure in 14 individuals. In the nine patients without a history of obstructive lung disease, a 26% reduction in FEV1 occurred compared to values obtained after 8 weeks. In the group of patients with a history of COPD, a decrease of 24% in the FEV1 to 53% predicted occurred, compared to an FEV1 of 76% predicted 6 months later. These articles clearly describe the transient obstructive abnormalities or RADS that resolves within months of acute exposure. Our case is unique in that it was a liquid exposure resulting in airway strictures accounting for long-term dyspnea.

The cause for the progressive narrowing of the large airways in this case is unknown but is likely due to the direct aspiration of concentrated liquid acid into the airways. Subsequent attempts at healing and airway repair lead to progressive narrowing. Although damage to regions of the lung distal to the large airways was documented, this damage did not result in sequela observed in the large airways. On CT, there was no evidence of persistent pneumonitis, fibrosis, or bronchiectasis. However, the persistent obstructive defect and decreased DLco noted on lung function testing may represent persistent physiologic abnormalities in the distal airways and/or lung parenchyma related to acid injury.

Recently, stent placement has played a major role in palliative management for airway obstructions due to malignancy. In local benign strictures, however, the “gold standard” is surgical resection.7,8 In nonsurgical patients, Dumen stent (Novatech; Marseilles, France) placement is preferred. Self-expandable metal stents for benign stenosis should be applied cautiously because granulation tissue formation, scarring, and restenosis occur frequently, making later management by other means difficult if not impossible.

In our patient, airway narrowing was too diffuse and associated with active inflammation, which made surgical resection or Dumen stenting less desirable.9 Instead, to relieve the severe dyspnea, multiple self-expandable metal stents were placed after initial dilatation by rigid bronchoscopy.

Usual expected complications of self-expandable stents include granulation tissue formation, restenosis by scar tissue, stent migration, and stent covering bronchial openings. All these complications occurred in this patient, and were managed successfully by numerous bronchoscopic procedures. The first left main bronchus stent migrated distally. It was removed and replaced with another one slightly protruding into the terminal trachea in order to cross the narrowed left main bronchus opening. Restenosis occurred at the left main bronchus opening inside the stent, which was dilated with a balloon two times. Granulation tissue at the proximal and distal edges of the stent and inside the stent was laser-coagulated at three bronchoscopic procedures. Finally, the part of the right main bronchus stent covering the right upper lobe orifice was opened by laser, and the portion of the left main bronchus stent protruding to the terminal trachea removed by laser.10

This case typifies an old saying, “all that wheezes is not..."
Pulmonary Toxicity in Patients Receiving Low-Dose Amiodarone*

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Rationale: Although there have been reports of pulmonary toxicity with low-dose amiodarone, it is generally believed that low-dose therapy is safe.

Methods: The clinical data for eight patients identified from a retrospective chart review are presented.

Results: All of the patients were receiving amiodarone, 200 mg/d, for an average of 2 years. The average age was 77 years (range, 65 to 89 years). Seven of the eight patients were male. Seven of the eight patients presented with dyspnea on exertion, and three of the eight patients presented with cough. All of the patients had a clinical diagnosis of amiodarone-induced pulmonary toxicity. Open-lung biopsies were obtained on two patients that were consistent with amiodarone-induced pulmonary toxicity. None of the patients were in congestive heart failure. Treatment involved cessation of amiodarone. In addition, three patients received corticosteroids. Five of the patients improved symptomatically with this conservative approach, and four patients improved radiographically. One patient died with progressive respiratory insufficiency (presumably from amiodarone pulmonary toxicity). One patient was unavailable for follow-up.

Conclusion: Amiodarone-induced pulmonary toxicity can occur at a daily dose of 200 mg. Clinicians must remain alert to this possibility even with this low-dose therapy.

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Key words: amiodarone; antiarrhythmics; drug toxicity; pulmonary medicine

Abbreviations: BOOP = bronchiolitis obliterans organizing pneumonia; CABG = coronary artery bypass grafting; DLCO = diffusing capacity of the lung for carbon monoxide; TLC = total lung capacity

Pulmonary complications, including pneumonitis and ARDS, are well-documented risks of long-term amiodarone use, especially in the perioperative setting. Although there have been reports of pulmonary toxicity with low-dose amiodarone use (defined here as < 200 mg/d), it is generally believed that low-dose therapy is safe. In addition, amiodarone has been associated with ARDS following coronary artery bypass grafting (CABG). One study has concluded that low-dose amiodarone is safe for cardiac surgery. We report a series of eight patients with pulmonary toxicity associated with low-dose amiodarone to emphasize that even low-dose therapy may have serious adverse pulmonary effects.

Materials and Methods

The clinical records of eight patients are presented. After receiving institution review board approval, a series of five case reports were compiled from the cardiology and pulmonology practices at Mayo Clinic in Jacksonville, FL, a referral-based, multispecialty group practice. In addition, we performed a retrospective electronic chart search of our outpatient clinic population using the key words amiodarone pulmonary toxicity and amiodarone-induced pulmonary toxicity from 1994 to 2001. Approximately 5,890,000 patient records from 520,000 patients were searched with this method. Sixty clinical notes involving 28 patients were identified as potential matches and were reviewed. Thirteen of these 28 patients identified were receiving amiodarone at the time of the evaluation. Nine of these 13 were receiving at least 400 mg/d. The authors reviewed the clinical charts of the four patients receiving low-dose amiodarone at length to evaluate for the diagnosis of amiodarone-induced pulmonary toxicity, recognizing that this is usually a diagnosis of exclusion. Radiographs, biopsies, bronchoscopies, laboratory work, echocardiograms, and documented symptoms/examination findings were considered in making the presumed diagnosis. An example of this review is presented in the “Results” section.

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