SWALLOWINGNEWS

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Director's Forum

GASTROESOPHAGEAL REFLUX: EXTRAESOPHAGEAL COMPLICATIONS

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The complications of gastroesophageal reflux are among the most common problems seen by primary care physicians and gastroenterologists. The clinical manifestations of acid reflux sequelae include the classical symptoms and signs (heartburn and regurgitation) that both patients and physicians recognize readily. There are, however, some less apparent manifestations, both esophageal and extraesophageal, that may not be recognized unless careful history and certain specialized studies are performed. This presentation will focus on complications of acid reflux that involve the upper aerodigestive tract, including the mouth, pharynx, sinuses, larynx and lungs. Loss of enamel from the teeth also is reported due to acid injury. For many years, the primary interest in acid reflux has been centered around damage caused to the esophagus resulting in heartburn as the most common manifestation. Severe acid reflux complications include erosive esophagitis, esophageal stricture, the columnar-lined (Barrett) esophagus with its predisposition to precancerous changes of dysplasia, and ultimately in a few patients, to adenocarcinoma of the esophagus.

The typical reflux symptom of heartburn, at times associated with regurgitation, is widely recognized as the result of acid irritation or injury to the esophageal mucosa. A diagnostic problem arises when patients without telltale symptoms and signs of simple esophageal reflux present with complications that are a direct result of acid reflux and regurgitation. The mechanism for the sensation of heartburn is not fully understood, but it is clear that the damaging effects of acid reflux may cause both severe esophageal and extraesophageal disease without producing either heartburn or detectable regurgitation as a clue to its presence.

The frequency of upper aerodigestive and pulmonary symptoms in children and adults caused by reflux is not known. There is increasing evidence that asthma, recurrent pneumonia, bronchitis, hoarseness, chronic cough, sore throat and post-nasal drip may in some individuals be caused by the silent regurgitation of acid reflux into the mouth and pharynx, as well as aspiration of this material into the larynx and lungs. There is also evidence that acid reflux into the esophagus may induce a neural (nerve related) reflex constriction of the bronchi or smaller air passages in the lung that may result in chronic cough and/or asthma. In children, recurrent middle ear infections, episodes of laryngospasm, respiratory arrest and sudden infant death syndrome may occur with aspiration or induction of the esophago-bronchial vagal nerve-mediated reflex mentioned above.

Some laryngologists have suggested that acid reflux may play a role as a co-factor with tobacco and alcohol in the etiology of squamous cell carcinoma of the larynx. Laryngeal and tracheal stenosis has been found related to acid regurgitation into the pharynx in over 50% of one group of patients studied.

Acid reflux is commonly associated with asthma, being reported in up to 80% of patients in some studies. This relationship should be suspected, particularly in adult onset asthma and in patients who have asthmatic symptoms primarily during periods of recumbency, especially at night, when acid reflux is more likely to occur. In some patients, a direct relationship to acid reflux can be demonstrated by a dramatic improvement in cough and/or asthma after adequate antireflux therapy by either medication or antireflux surgery.

Although an etiologic relationship between reflux and recurrent bouts of pneumonia is clearly documented in some patients, its frequency is relatively rare. On the other hand, chronic cough is one of the more frequent sequelae, acid reflux being cited as the primary cause in as many as 10% of adults in one study. The mechanism for chronic cough may be due solely to aspiration, especially micro aspiration of regurgitated stomach acid, or perhaps in some cases only by the stimulation of the esophagobronchial reflex initiated by acid refluxed into the esophagus.

The possibility of reflux-related symptoms also should be considered in a differential diagnosis in all patients with chronic laryngitis, hoarseness (especially if the hoarseness tends to improve or disappears later in the day), repeated throat clearing, ear pain or recurrent ear infections (especially in children), morning sore throat, or even bad breath.

Clinical studies have demonstrated that only 30-50% of patients with upper aerodigestive complications of gastroesophageal reflux will have heartburn, and only 40% or less will have evidence of esophagitis on endoscopic examination. Since at least half of individuals with acid reflux-related upper airway problems have no significant symptoms typical for acid reflux, it is impor-

(continued)

tant to utilize studies other than the usual barium x-ray and endoscopic investigations. The use of 24 hour ambulatory pH monitoring in such patients often provides proof of acid reflux into the proximal esophagus and hypopharynx in both the upright and recumbent positions. Dual channel pH studies have provided clear evidence that gastric acid reflux occurs as high as the proximal esophagus, and even into the pharynx and may lead to aspiration of acid into the larynx, trachea and lungs. Animal studies have revealed that even small quantities of acid can cause damage to the larynx, trachea and bronchial tree. Recent human studies suggest that about 80% of asthmatics have gastroesophageal reflux based on pH monitoring, but as noted above, less than half of these will have typical esophageal symptoms or signs of acid injury.

Ambulatory 24 hour pH monitoring is the diagnostic procedure of choice to determine the relation of extraesophageal disease symptoms to acid reflux. This study has a sensitivity and specificity of about 90% This procedure allows correlation between the patient's symptoms and the presence of acid in the upper esophagus or in the pharynx. Esophageal pH study is also necessary to determine whether a certain dose of acid suppression drugs is providing adequate acid control. For the study, acid-sensing electrodes, positioned in a single thin plastic tube about 15 cm (6 in.) apart, are used to evaluate the extent of acid reflux into the esophagus. The upper electrode is usually placed just below the upper esophageal sphincter at mid-neck level to prove the abnormal presence and duration of acid exposure at this unusually high level. Ideally, one of the electrode sensors should be placed above the upper esophageal sphincter in the pharynx, however, technical difficulties occur at this level due to electrode drying that causes difficulty in interpretation of the tracing. The true sensitivity of pH monitoring for this purpose remains to be elucidated.

Extraesophageal complications of acid reflux should be suspected in all cases with upper aerodigestive pulmonary symptoms. Obviously this etiology becomes much more likely in those cases with a negative conventional evaluation for the other causes of these symptoms, and a failure to respond to the usual therapeutic measures. Laryngoscopy, bronchoscopy or esophagoscopy may reveal evidence of unexplained mucosal disease. Twenty-four hour ambulatory pH monitoring offers the best chance for establishing a relationship between acid reflux and these extraesophageal sequelae.

The medical treatment of these atypical upper aerodigestive acid reflux syndromes is not nearly as predictably successful as it is with the typical esophageal symptoms. Although antireflux measures such as bed elevation and diet modification may be at least partly helpful in most, the optimum treatment must include maximum acid suppression by medication, preferably a proton-pump inhibitor (Prilosec or Prevacid). The optimum therapeutic challenge includes a double dose of proton-pump inhibitor acid suppression therapy for a period of three to six months to evaluate for clinical evidence of symptom reduction or resolution. In some patients, the optimum response will not be apparent for up to six months. The role of a prokinetic drug, such as Propulsid, is uncertain but may provide added benefits in some patients. In those cases with documented proximal acid reflux proven to benefit from acid suppression therapy, an antireflux surgical operation may offer the best long-term treatment. If patients do not respond on acid suppression pharmacotherapy, adequate esophageal and gastric acid suppression should be documented by 24 hour pH study prior to calling medical treatment a failure. Up to 20% of patients will have inadequate acid suppression on even a twice daily dosage of a proton-pump inhibitor.

Clinicians should be alert to the possibility of acid reflux-induced oropharyngeal, laryngeal and pulmonary symptoms in patients who are not found to have another specific cause for their complaint. This is one clinical circumstance in which clinical diagnosis of causation is difficult and laboratory diagnosis by 24 hour ambulatory pH monitoring proves most accurate. Proper evaluation and adequate therapy often will lead to dramatic improvement.

SCLERODERMA

Umesh Choudhry, M.D.

Scleroderma, as the name suggests, is a disorder that involves sclerosis (fibrosis or hardening) of the skin. When several systems in the body such as blood vessels, gastrointestinal tract, lungs, heart and kidneys, etc. are involved, the disorder is called systemic sclerosis. The exact cause of this disorder is unknown. It has a worldwide distribution and occurs equally in all races. The onset of the disease is usually in the third to fifth decade. Women are affected nearly three times as often as men. The role of heredity and environmental factors in its causation is not proven but possible. Exposure to silica dust in mining industry and polyvinyl chloride, epoxy resins, benzene, etc. may result in scarring similar to that seen in scleroderma. As stated above, the disease may be classified as diffuse type or limited type, based upon the extent of skin involvement. The limited disease primarily results in skin thickening in the fingertips, toes and face. In addition, this form of the disease affects swallowing more often and forms the focus of this discussion.

SWALLOWING DIFFICULTY AND SCLERODERMA

The basic pathology of this condition involves replacement of normal cutaneous (skin) or smooth muscle tissue by fibrous tissue. When such replacement occurs in the food pipe (esophagus), its ability to transport food in a normal fashion is compromised. This results in swallowing difficulty or dysphagia due to an abnormal movement pattern of the esophagus (esophageal dysmotility). The characteristic motility disorder seen in patients with scleroderma is a loss of peristalsis or synchronized movement in the lower half of the esophagus along with a complete loss of tone or pressure in the lower esophageal sphincter muscle. The lax sphincter in turn allows severe and prolonged acid reflux into the esophagus, resulting in acid related injury to the esophagus. When acid reflux goes untreated for a long period of time, the esophagus lining undergoes scarring and stricture formation, thus worsening the dysphagia. Initially, these patients have a very slowly progressive difficulty in swallowing solids and liquids. As the lumen of the esophagus becomes narrower, the swallowing difficulty becomes more prominent and food impaction may result. Some patients with scleroderma may have skin tightening around the mouth and may not be able to open their mouth enough to take normal-sized bites.

When scleroderma involves other areas of the gastrointestinal tract, decreased or hypomotility is seen in the small and large intestine. This results in bloating, abdominal pain and constipation. At other times, decreased intestinal motility results in excess colonization of bacteria in the intestine and consequent diarrhea.

DIAGNOSIS

Scleroderma is often diagnosed clinically by thickening of the skin of hands, loss of wrinkling on the face, telangiectasias or dilated tiny veins in the skin of the face, chest, arms and hands. Fingers and toes often turn blue, white and painful when exposed to cold temperature (Raynaud's phenomenon). Due to thickening of the skin of the fingertips, there may be decreased sensation and consequent increased chances of injury and delayed healing. These features, in conjunction with dysphagia and calcium deposits in tissues, are collectively called by an acronym, CREST syndrome. Diagnosis is further confirmed by looking for anti-Scl 70 antibody in the blood and performing an esophageal manometry study and recording the movement pattern in the esophagus. Biopsies from the involved organs may reveal characteristic findings.

THERAPY

Although no therapy is currently available that can reverse the sclerosis, numerous drugs have been tried with variable results. These include d-penicillamine, calcium channel blockers, steroids, colchicine, etc. Therapy specifically aimed towards swallowing difficulty includes adopting antireflux precautions, a set of life style changes that prevent acid reflux. Potent acid suppression therapy such as omeprazole (Prilosec) or lansoprazole (Prevacid) is often needed to reduce/heal the esophagitis that occurs in these patients. In addition, drugs that improve stomach emptying (prokinetic agents) such as metoclopromide (Reglan) and cisapride (Propulsid) may be used in conjunction with acid suppression therapy.

Things To Remember

1. **OFFICE HOURS:** 8:00 a.m. 'til 4:30 p.m. Monday through Friday.

   Our office is closed on weekends and some holidays so it is important to make sure any medication refills are called to us during our regular office hours.

   Also, our emergency telephone number for after hours is (813) 974-2201.

2. **BILLING: The University of South Florida Physicians Group, as most doctors offices, expects payment for services rendered at the time of your visit. Please be prepared to pay any co-payments due at the time of your visit to the Center for Swallowing Disorders.**

   Patients who have problems with their physician or facility fee bills should contact Laura Fussner, Financial Specialist, at the University of South Florida Medical Clinics at (813) 974-4659 between the hours of 9:00 a.m. and 4:00 p.m. Monday through Friday.

   It is the patient's responsibility to get authorizations or pre certifications from their insurance company **before** any treatment occurs. Laura is available to help with insurance authorizations when problems arise.

   For those patients who are from out-of-town, a new toll-free number has been added for you to call with billing questions. The number is 1-888-873-3627. This number is for calls originating in Florida and is only for billing questions and help with insurance authorizations.

3. **DILATIONS:** For our patients who receive periodic esophageal dilations: Please try to anticipate and contact our office at least 2 to 3 weeks in advance of your need for dilation, if at all possible. We have been having to schedule return cases 3 to 4 weeks in advance due to our heavy patient load. We do not want any of you to suffer unnecessarily, so please help us with your appointment needs.

Dilation or stretching of the wall of the esophagus may be needed when stricture formation causes narrowing of the esophageal lumen. This is an outpatient procedure for which tapered bougies or balloons are used to gradually stretch the esophageal wall resulting in widening of the lumen. Based upon the original caliber of the esophageal lumen, this process may take several sessions. Muscle exercises and rehabilitation measures are helpful when scleroderma affects the mouth opening. These improve the bite size and prevent injury to the angle of the mouth.

In conclusion, scleroderma is a serious condition involving several organ systems in the body. Skin thickening and tightening is the most overt of these manifestations. Gastrointestinal and esophageal manifestations are common. Once the disease is diagnosed, it may progress at a variable pace and may be very insidious. None of the available therapeutic modalities can alleviate some symptoms, improve blood supply to fingers, but cannot reverse the underlying disorder. When the esophagus is involved, swallowing difficulty and severe reflux results. Potent acid suppression and promotility agents are useful. When stricture formation occurs, esophageal dilation is needed.

CONTINUING MEDICAL EDUCATION

During the past several months members of the Center for Swallowing Disorders staff have continued their active participation in graduate medical education at regional, national, and international meetings and by contributions to the medical literature.

Lecture Presentations by CSD Staff

July 28-29, 1997: Diagnosis and treatment of patients with dysphagia. Greenville Hospital's Family Practice Residency Program. Greenville, SC. (Boyce)
September 10-13, 1997: 1) Esophagogastric junction anatomy and chromoendoscopy in the diagnosis of columnar-lined esophagus; 2) Medical manage ment of complex benign esophageal strictures. New Techniques in Diagnostic and Therapeutic Endoscopy Conference. Milwaukee, WI. (Boyce)

3. October 4, 1997: A long look down your throat. Mini Med School, USF College of Medicine. Tampa, FL. (Boyce)
4. October 15-18, 1997: Management of refractory peptic strictures. Tenth International Course on Therapeutic Endoscopy. Toronto, Canada. (Boyce)
5. October 25, 1997: GERD and its complications – treatment strategies. USF: Common Gastrointestinal Disorders: Clinical Update. Tampa, FL. (Boyce)
6. November 1, 1997: Treatment of benign and malignant strictures of the esophagus. ACG Annual Postgraduate Course. Chicago, IL. (Boyce)
7. November 2, 1997: Utilization of chromoendoscopy and high resolution endoscopy in the evaluation of the dyspeptic patient. ACG Satellite Symposium: The evaluation and treatment of non-ulcer dyspepsia. Chicago, IL. (Boyce)
8. November 12, 1997: 1) An approach to dysphagia; 2) Atypical manifestations of GERD. Dayton Gastroenterology Symposium. Dayton, OH. (Boyce)
9. December 12-14, 1997: Esophageal strictures. Astra Symposium: Clinical Challenges in Gastroenterology. Vienna, Austria. (Boyce)
10. February 5-8, 1998: 1) Esophageal cancer; 2) Columnar-lined (Barrett) esophagus: diagnosis and treatment, surveillance; 3) Achalasia: chemical (Botox) denervation therapy; 4) Endoscopic ultrasonography diagnosis and staging. Caracas Red Cross Hospital – 30th Anniversary Cancer Conference. Caracas, Venezuela. (Boyce)
11. February 11, 1998: Management of complex esophageal strictures. Pinellas County Cut Club Inaugural Meeting. Clearwater, FL. (Boyce)
12. February 20-22, 1998: Techniques in esophageal dilation. Interactive video case presentations EGD: Dilation, chromoendoscopy, MPEC for Barretts. 22nd Gastrointestinal Assistants Seminar: The Techniques of Endoscopy. Los Angeles, CA. (Boyce)

February 20-22, 1998: Swallowing Disorders: The multidisciplinary approach to assessment. Interactive video case presentations EGD: Dilation, chromoendoscopy, MPEC for Barretts. 22nd Gastrointestinal Assistants Seminar: The Techniques of Endoscopy. Los Angeles, CA. (Jones)
March 9-13, 1998: 1) Anatomy of the esophagogastric junction; 2) Therapy for malignant esophageal obstruction; 3) Medical management of complex esophageal strictures. Solutions in Gastroenterology: A Clinical and Pathological Approach. Dorado, Puerto Rico. (Boyce)
March 20-22, 1998: 1) Diagnostic endoscopy for GERD; 2) Therapeutic endoscopy for Barrett esophagus; 3) Gastric endoscopy and the dyspeptic patient. New Frontiers in Gastroenterology: Advanced Diagnostic and Therapeutic Workshops. Video Case Presentations. Orlando, FL. (Boyce)
March 25-27, 1998: 1) Complex esophageal strictures: diagnostic clues and therapeutic options; 2) Esophagology for internists: update on diagnosis and therapy. Brooke Army Medical Center Visiting Consultant. San Antonio, TX. (Boyce)
April 17-19, 1998: 1) Diagnostic endoscopy for GERD; 2) Therapeutic endoscopy for Barrett esophagus; 3) Gastric endoscopy and the dyspeptic patient. New Frontiers in Gastroenteology: Advanced Diagnostic and Therapeutic Workshops. Video Case Presentations. New York, NY. (Boyce)
May 1, 1998: 1) Adventures in the esophagus: an update on diagnosis and therapy; 2) Complex esophageal stricutres: pathogenesis as a guide to therapy and prognosis. St. Louis University Visiting Professor Program. St. Louis, MO. (Boyce)

Contributions To Medical Literature and Clinical Research Abstracts

Chak A, Canto M, Rosch T, Dittler HJ, Hawes RH, Tio TL, Lightdate CJ, Boyce HW, Jr. et al: Endoscopic differentiation of benign and malignant stromal cell tumors. Gastrointest Endosc 1997;45(6):468-473.

Boyce HW, Jr.: Therapeutic approaches to healing esophagitis. Am J Gastroenterol 1997;92(4):22S-29S.

Boyce HW, Jr.: Editorial: Drug-induced esophageal damage: diseases of medical progress. Gastrointest Endosc (in press).

Acosta MM, Boyce HW, Jr.: Chromoendoscopy – where is it useful? Jnl of Clinical Gastroenterology (in press).

Johnson MC, Boyce HW, Jr.: Endosonography: a new frontier in endoscopy. Contemporary Internal Medicine 1997;9(10):46-54.

Choudhry U, Boyce HW, Jr., Robinson BE: Inlet Patch: is it really an innocent bystander? Gastrointest Endosc 1997;45(4):AB65.

Choudhry U, Boyce HW, Jr., Robinson BE: Inlet Patch: a propsective comparison of its occurrence in achalasia and gastroesophageal reflux disease (GERD). Gastrointest Endosc 1997;45(4):AB65

Sherman F, Baig Z, Choudhry U, Boyce HW, Jr., et al: The prevalence of specialized columnar epithelium in patients with symptoms and/or signs of gastroesophageala reflux. Gastrointest Endosc 1997;45(4):AB82.

Baig Z, Choudhry U, Sherman F, Boyce HW, Jr., et al: Columnar-lined esophagus (CLE) and esophageal stricture (ES): how commonly do they co-exist? Am J Gastroenterol 1997;92(9):1584.

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