Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

ischemia and bowel infarction [9]. When the diagnosis is delayed, it carries a mortality rate of up to 67% [10]. The management of intestinal obstruction generally requires definitive intervention, and early determination of the etiology may minimize subsequent morbidity and mortality.

Conventionally, the diagnosis of small bowel volvulus is made by upper gastrointestinal series showing an abnormal position of duodenojejunal junction. Barium enema may also show an abnormal position of the cecum. However, the results of radiographic studies are not always conclusive [5,11], and barium upper gastrointestinal studies poorly predict the risk for midgut volvulus [12]. These studies, such as barium enema and upper gastrointestinal series, are time-consuming and not available 24 hours each day 7 days a week. They may be valid if the patient can tolerate the procedure and the clinical condition permits performance of the test.

Ultrasonography is another tool for the emergency physician in evaluating patients with acute abdominal pain. The appearance of the characteristic whirlpool sign can facilitate the preoperative diagnosis of midgut volvulus and malrotation [4]. The disadvantage of ultrasonography is that it is operator-dependent. A detailed ultrasonographic survey of the abdomen, especially of the superior mesenteric vessel to their mesenteric artery, can increase diagnostic accuracy and give better evaluation of SMV and SMA relationship. However, this needs to be weighed against the impracticality of obtaining a detailed ultrasound examination under these circumstances. We demonstrated a patient with the initial presentation of abdominal pain with ileus pattern on initial plain radiogram. The whirlpool sign and the SMV using ultrasonography, was twisted around the SMA with 270° of rotation and with the whirlpool sign being observed, and small bowel volvulus was diagnosed promptly and is valuable for the preoperative diagnosis of mesenteric vessel malrotation and midgut volvulus.

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Herpes simplex virus esophagitis in an immunocompetent teenaged girl

A previously healthy 14-year-old teenaged girl presented to the emergency department (ED) complaining of dysphagia associated with liquids and solids and difficulty swallowing her own saliva. The patient also reported a "pain in the chest" for the previous 2 days that appeared to begin after eating a turkey sandwich. The pain was substernal, sharp, and pleuritic. She reported fever and chills, but denied abdominal pain, vomiting, diarrhea, or hematemesis. She denied any history of gastroesophageal reflux, nonsteroidal anti-inflammatory drug, tobacco or alcohol use, unusual ingestions, trying to induce vomiting, or having ever been sexually active.

On arrival, her vital signs were as follows: temperature, 39.4°C; blood pressure, 105/60; pulse rate, 80 beats per minute; respiratory rate, 22 breaths per minute; oxygen saturation, 97% on room air.

On physical examination, she was sitting upright, non–toxic-appearing, and spitting her saliva into a cup. Her oropharynx had no lesions or exudates. There was no evidence of lymphadenopathy. The lungs were clear to auscultation. Her cardiac examination demonstrated no murmur, rub, or gallop. The abdominal examination was benign and there was no flank tenderness. The skin examination revealed no rashes or lesions.

The white blood cell count was 14.1, with 80% neutrophils and 7% bands. While in the ED, her serum electrolytes, serum urea nitrogen, creatinine, and dipstick urinalysis were unremarkable.

The patient had a radiograph of the chest and a lateral soft tissue of the neck. Both of these studies were normal.

A pediatric gastroenterologist was consulted, and the patient was taken to operating room for removal of a suspected esophageal foreign body. The patient underwent esophagogastroduodenoscopy (EGD) with biopsies. There was no evidence of foreign body or *Candida* esophagitis. The entire esophagus was erythematous and edematous with linear, superficial lesions throughout (Fig. 1). The mucosal surface was friable. Biopsies of the proximal esophagus showed acute esophagitis with ulceration, and biopsies of the distal esophagus demonstrated the viral cytopathic changes of herpetiform esophagitis. A slide made from tissue of the esophagus showed inclusion bodies consistent with herpes simplex virus (HSV) infection. She was admitted to the hospital and started on intravenous famotidine, fluconazole, and acyclovir. Follow-up EGD 2 days later was once again consistent with HSV esophagitis (HSVE).

A pediatric infectious disease consult was obtained. T-cell studies, including CD4, and immunoglobulin (Ig) studies (IgG, IgA, and IgM) were normal. HIV testing was refused.

She was continued on intravenous acyclovir for 1 week. At the time of discharge she had no fever and was able to swallow without difficulty. She was discharged on oral acyclovir and famotidine.

Johnson et al first reported HSVE in 1940, and subsequent autopsy studies have demonstrated an incidence of 1.8%. The overwhelming majority of these were known to have been immunocompromised patients [1]. Visceral disease caused by HSV in the immunocompromised host most commonly affects the esophagus [2]. It is a common opportunistic infection in patients with HIV/AIDS, solid organ and bone marrow transplant recipients, patients on immunosuppressive therapy or systemic corticosteroids, diabetic patients, alcoholics, or anyone who is otherwise immunocompromised. Another group of patients that are prone to HSVE are those with motility disorders, esophageal diverticulum, or esophageal mucosal injury. Gastroesophageal reflux and nasogastric tube placement are commonly cited as precipitants to HSVE [3].

Herpes simplex virus esophagitis is infrequently reported in the immunocompetent host. Case series on these patients reveal an age range from 1 to 76 years, with a median age of 29 $\pm$ 21 years. Pediatric cases (<18 years of age) represent a distinct minority (24%). A male predominance is often cited in reviews, representing 90% of the pediatric population [2].

The clinical presentation of HSVE in the immunocompetent host is often subtle. Only a minority of patients who are diagnosed with HSVE present with concurrent oropharyngeal lesions (21%) or a prodrome of systemic manifestations (24%). Fever is reported in many cases (45%-63%) [1,2], but the absence of an elevated temperature should not discourage the clinician to consider this diagnosis. A minority of patients have coexistent herpes infections of the oropharynx or labia. In addition, family members with concurrent HSV infection are found in only 22% of the cases [2].

Esophagitis is infrequently reported in the pediatric age group. Rodrigues et al [4] looked at 6 children who were diagnosed with HSVE. Most presented with fever, odynophagia, dysphagia, and retrosternal pain. The median time from onset of symptoms to diagnosis was almost 7 days. In the pediatric immunocompromised population, HSV viremia is important in the pathogenesis of disseminated HSV infection, but the same is not true with the immunocompetent host [5].

The most likely source of transmission of HSVE in the immunocompetent host is gastroesophageal reflux with local tissue damage and superimposed herpes infection from a previously affected oral or labial herpes. Our patient denied any previous oral herpes, but the patient may have been unaware of the infection or forgotten about the occurrence. In addition, oral sex and sexual abuse must be considered in these cases. Finally, the possibility still exists that this patient has congenital transmission of HIV and is, in fact, immunocompromised.

Herpes simplex virus esophagitis in the immunocompetent host is usually self-limiting, although it is reported that antivirals may hasten recovery. Because this is a rarely reported clinical entity there have not been many studies that have looked at the improvement of immunocompetent patients with HSVE when treated with antivirals. Kato et al [1] reports that patients receiving antiviral therapy improve almost 5 days faster than patients who did not receive similar therapy (4.6 $\pm$ 4.7 and 10 $\pm$ 6.8 days). Several authors, including Ramanathan et al [2], have suggested that there is evidence to support the early use of antiviral therapy in both primary and recurrent HSV infections and, therefore, this same approach may be reasonable in the immunocompetent patient with HSVE.

Evaluation of a sore throat in the pediatric population can be challenging because dysphagia and odynophagia are

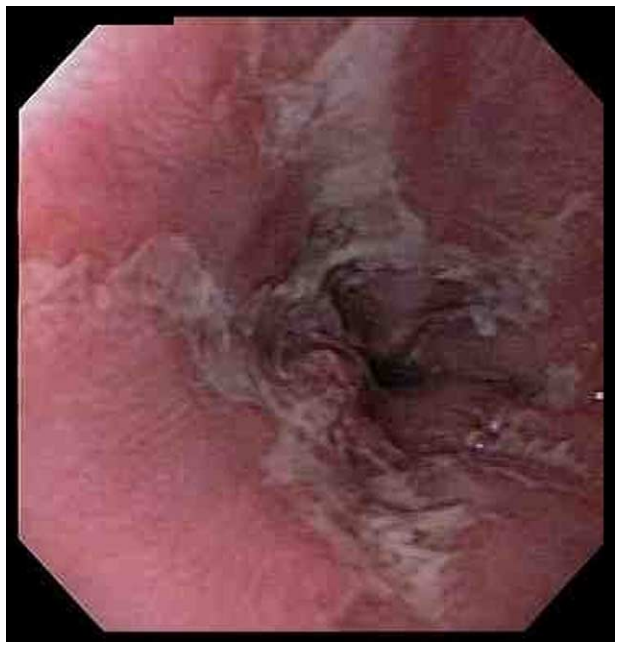

Fig. 1 Esophagus with superficial lesions seen on EGD.

often symptoms of a disease process unrelated to the pharynx. Consequently, a meticulous history and physical examination are required to properly distinguish among the myriad causes of a sore throat.

The most common etiologies of the pediatric sore throat are infectious in origin, with viral pharyngitis accounting for 40% of all cases. The most frequent causative pathogens in viral pharyngitis include adenovirus, coronavirus, coxsackievirus, cytomegalovirus, echovirus, Epstein-Barr virus (EBV), HSV, HIV, parainfluenza virus, and rhinovirus. Primary infection with bacteria accounts for 30% of infectious pharyngitis in children, with group A β-hemolytic streptococcus (*Streptococcus pyogenes*) predominating. Additional pyogenic causes include group C streptococci, group G streptococci, *Arcanobacterium haemolyticum*, *Chlamydia pneumoniae*, *Corynebacterium* species, *Francisella tularensis*, *Mycoplasma pneumoniae*, and *Neisseria gonorrhoeae*. If a patient is immunocompromised it may render them susceptible to any of the aforementioned conditions and more specifically to opportunistic infections such as HSV and candidiasis [6-9].

A detailed history and physical examination are often sufficient to make the diagnosis and proceed with treatment; however, the ED workup for a basic sore throat may include a pharyngeal swab and culture. Sensitivity and specificity of the results of these tests vary depending on collector technique, sampling obtained, and media used. More specialized diagnostic modalities, such as dedicated media for viruses or serologies for HSV and EBV, are also sometimes used [9].

Conditions that could potentially compromise the airway of a pediatric patient presenting with a sore throat are foreign body, epiglottitis, EBV-induced tonsillar hypertrophy, abscesses (lateral pharyngeal, peritonsillar, retropharyngeal), and Lemierre syndrome (mixed anaerobic pharyngitis complicated by sepsis). In addition to respiratory distress and stridor, signs that should alert a health care provider to any of these life-threatening conditions include toxic appearance, drooling, uvular deviation, trismus, submandibular edema, neck tenderness to palpation, high fever, and hypotension [6,7].

The causes of a chronic sore throat are varied. They include chemical exposure (usually alkaline), irritation (postnasal drainage, hot-air heating), neoplasms, vasculitides, systemic inflammatory diseases (Behçet's syndrome, Kawasaki disease, Steven-Johnson syndrome), referred pain (cervical adenitis, dental disease, ear infections), and psychogenic pharyngitis (anxiety, globus hystericus) [7].

Further ancillary studies, such as blood work, radiographic imaging (including computed tomography scans), EGD, or consultation with a specialist (such as a gastroenterologist or otorhinolaryngologist) may be warranted in the workup of a life-threatening or chronic sore throat. The patient's disposition hinges on the severity of their clinical presentation, availability of diagnostic modalities in your ED, and opportunities for proper outpatient follow-up.

In our ED, the percentage of patients with a sore throat who ultimately require admission is very small. In 2005, we admitted 2232 of the 14 023 pediatric patients (15.9%) who presented to our ED, but only 11 (0.49% of those admitted) had a diagnosis of sore throat, dysphagia, or odynophagia. The challenge is to distinguish between those cases that are benign and serious. When patients present with dysphagia or odynophagia and the diagnosis is not straightforward or obvious, the ED provider should consider esophagitis, including HSVE, regardless of the patient's immune status.

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