

Eosinophilic esophagitis in a dog

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Case Description—A 4-year-old spayed female mixed-breed dog with a history of allergic skin disease was examined because of regurgitation, coughing, and dysphagia that began 15 days after abdominal surgery for correction of gastric dilatation and volvulus.

Clinical Findings—Severe diffuse esophagitis, esophageal dysmotility, and a benign esophageal stricture at the level of the base of the heart were identified via contrast videofluoroscopy and esophagoscopy. Severe diffuse eosinophilic ulcerative esophagitis was confirmed by histologic examination of esophageal biopsy specimens and cytologic evaluation of specimens obtained by use of a cytology brush. Esophageal eosinophils were evident (14% to 50% of the inflammatory cell population and > 25 eosinophils/hpf).

Treatment and Outcome—No clinical or endoscopic improvement was evident after treatment with antireflux medications, including a proton-pump inhibitor, following an initial esophageal bougienage procedure. An excellent response characterized by resolution of dysphagia and regurgitation with marked improvement of the esophageal mucosa was evident following intralesional and systemic administration of glucocorticoids, 2 additional esophageal bougienage procedures, and feeding of an elimination diet.

Clinical Relevance—To our knowledge, the information reported here is the first description of eosinophilic esophagitis (EE) in a dog. Many similarities exist between the condition in the dog reported here and EE in humans. This clinical report highlights the need to consider EE as a differential diagnosis for esophagitis and esophageal strictures in dogs. When appropriate, esophageal biopsy or cytologic specimens should be obtained and examined to investigate the possibility of EE. (*J Am Vet Med Assoc* 2009;235:61–65)

A 4-year-old spayed-female mixed-breed dog that weighed 24.6 kg (54.1 lb) was examined at our veterinary medical teaching hospital because of regurgitation, coughing, and dysphagia (day of initial examination was designated as day 1). Twenty-five days before examination at our facility, the dog underwent abdominal surgery to correct GDV. Acute vomiting associated with the GDV resolved within 24 hours after surgery; there were no complications during surgery or the recovery period, and the dog was eating and drinking normally by 5 days after surgery. Daily regurgitation of food and sometimes liquids, in addition to dysphagia characterized by exaggerated swallowing, stretching of the neck, and licking of the lips associated with eating, began 10 days prior to examination (ie, 15 days after surgical correction of GDV). The dog also had a productive cough, which began 3 days before examination.

Other problems in the medical history included pedal pruritus, nonseasonal otitis externa, dermatitis attributable to *Malassezia* spp, and superficial pyoderma associated with atopic dermatitis or food allergies. A board-certified veterinary dermatologist made the dermatologic diagnoses 10 weeks before day 1. Treatment initiated at the time of the dermatologic diagnoses included clindamycin (5.4 mg/kg [2.45 mg/lb], PO, q 12 h for 4 weeks), ketoconazole (7.1 mg/kg [3.23 mg/lb], PO, q 24 h for 2

ABBREVIATIONS

EE	Eosinophilic esophagitis
GDV	Gastric dilatation and volvulus
GERD	Gastroesophageal reflux disease
PEG	Percutaneous endoscopic gastrostomy

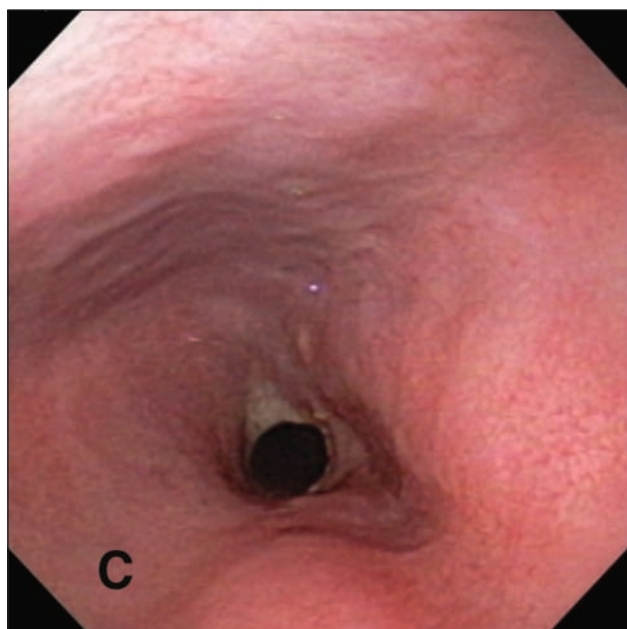
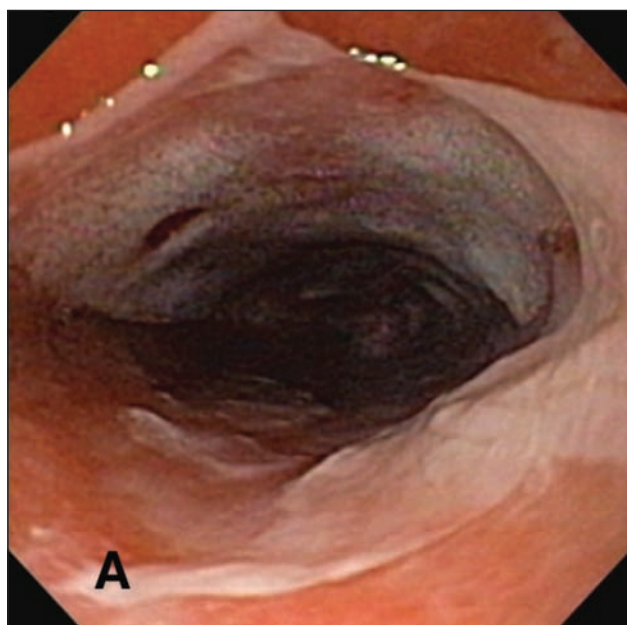
weeks), prednisone (0.5 mg/kg [0.23 mg/lb], PO, q 24 h for 1 week, then 0.2 mg/kg [0.091 mg/lb], PO, q 24 h), topical antimicrobial medications, and feeding of a commercial elimination diet,^a all of which resulted in noticeable dermatologic improvement. The owner stopped feeding the elimination diet a few weeks before day 1, and an increase in pruritus was detected after that diet was discontinued. The only medication being administered on day 1 was an intermittent low dose of prednisone (0.2 mg/kg, PO, q 24 to 72 h).

Physical examination on day 1 revealed that the dog was alert and active with a body condition score of 5 (scale of 1 to 9). Palpation of the dog's trachea and neck resulted in coughing and discomfort manifested by exaggerated swallowing and licking of the lips. The only other abnormalities detected were pruritus, erythema, and hyperpigmentation of the skin in the perineal, interdigital, and aural regions. Results of a CBC, serum biochemical analysis, and urinalysis were unremarkable. Thoracic radiography revealed focal dilatation of the esophagus with gas cranial to the heart base; radiography also revealed alveolar infiltrates of the right middle lung lobe that were consistent with aspiration pneumonia. Evaluation of a fluoroscopic esophagram revealed dilatation in the cranial portion of the thoracic

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esophagus and severe dysmotility; the esophagus was narrow (approx 9.2 mm at the heart base), which was consistent with a stricture.

Gastroesophagoscopy performed on day 2 confirmed a benign esophageal stricture, but the most striking finding was severe, diffuse esophagitis characterized by exceedingly friable and hyperemic mucosa, with large areas of exudative and ulcerated mucosa caudal to the stricture site (**Figure 1**). The stricture was easily torn when a 8.9-mm gastroscope^b was gently manipulated through the narrowed area. The lower esophageal sphincter and gastric mucosa were visibly normal. Multiple biopsy specimens were obtained by use of the gastroscope, and 2 biopsy specimens from the proximal and distal portions of the esophagus were acquired. The specimens were easy to collect because of the friable nature of the esophageal mucosa. A PEG feeding tube was placed.



The dog was discharged to the owner with instructions for feeding and medicating the dog via the PEG tube. Initial treatment included antireflux medications (famotidine [0.6 mg/kg {0.27 mg/lb}, PO, q 12 h for 5 days], omeprazole [1 mg/kg {0.45 mg/lb}, PO, q 24 h], cisapride [0.6 mg/kg, PO, q 8 h], and sucralfate [41 mg/kg {18.64 mg/lb}, PO, q 8 h]), amoxicillin-clavulonic acid [20 mg/kg {9.1 mg/lb}, PO, q 8 h for 3 weeks]), and prednisone (0.2 mg/kg, PO, q 24 h). Another commercial elimination diet^c was prescribed because of the dermatologic disease. It was recommended that the dog be returned to our facility 7 days later for reexamination.

Histologic examination of the esophageal and gastric biopsy specimens obtained on day 2 revealed severe, diffuse eosinophilic and neutrophilic ulcerative esophagitis and minimal to mild lymphocytic gastritis of unknown importance. In most sections of the esophageal biopsy specimens, the ulcerated mucosa was replaced with a bed of granulation tissue containing an inflammatory infiltrate of approximately 50% eosinophils, with 25 to 40 eosinophils/hpf.

The dog was not reexamined until day 28. At that time, coughing had ceased; however, the dysphagia was similar to that reported previously, and the regurgitation had progressed to complete intolerance of solid food. Despite our recommendations, the PEG tube had been used only intermittently and the elimination diet had been fed for only a few days after discharge. Medications being administered by the owner on day 28 included omeprazole, cisapride, sucralfate, and prednisone at the aforementioned dosages and intervals.

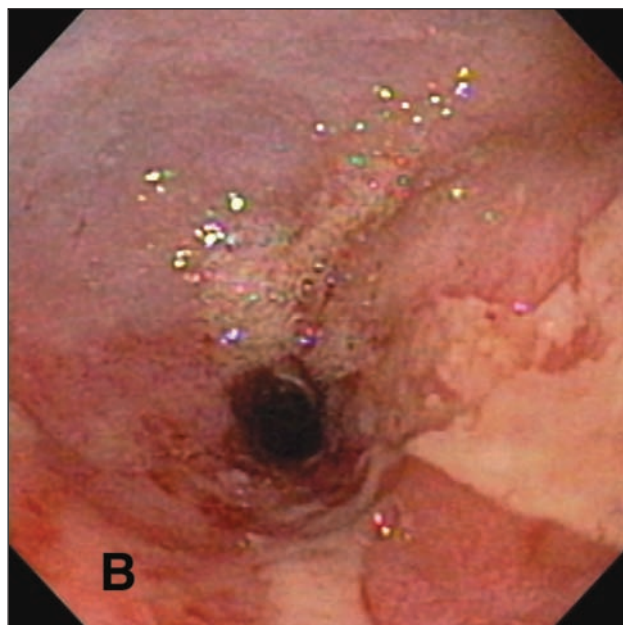


Figure 1—Photographs of the esophagus of a dog examined because of regurgitation, coughing, and dysphagia that began 15 days after abdominal surgery for correction of GDV. **A**—The image was obtained on day 2 (day 1 = day of initial examination because of regurgitation, coughing, and dysphagia). Notice the diffuse exudative and ulcerative esophagitis distal to the stricture site. **B**—The image was obtained on day 28. Notice the esophagitis and a benign esophageal stricture after administration of antireflux medications. **C**—The image was obtained on day 40. Notice the esophageal mucosa and a benign esophageal stricture after treatment for EE.

Thoracic radiography revealed resolution of the aspiration pneumonia, but the esophagus remained dilated with gas cranial to the heart base. Esophagoscopy was performed to evaluate severity of the esophagitis and assess stricture recurrence. Grossly, the severe, diffuse esophagitis on day 28 appeared similar to that on day 2 (Figure 1), although the esophageal stricture was smaller (diameter, 7 mm). Triamcinolone (0.4 mg/kg [0.18 mg/lb]) was diluted 1:1 with saline (0.9% NaCl) solution, and the dose was divided into 4 aliquots and injected into the lesion (each of 4 quadrants around the stenotic area) by use of a 23-gauge sclerotherapy needle. A guidewire^d was passed into the esophagus and through the stricture site by use of an endoscope. The endoscope was then withdrawn, and bougienage was performed by gently passing rigid bougies^d (diameter, 7, 9, and 11 mm) over the guidewire.

A 2.5-mm-diameter endoscopic cytology brush^e was used to obtain samples from the esophageal mucosa at a location distal to the stricture site. Cytologic analysis revealed a highly cellular sample with 84% mild to moderately degenerative neutrophils, 14% eosinophils (approx 33 eosinophils/hpf in monolayer regions), 1.7% small mature lymphocytes, and 0.8% macrophages (Figure 2). Cytologically, a few unremarkable squamous epithelial cells and some intracellular and extracellular bacteria were also detected. No cytologic evidence of fungal elements, parasites, or neoplastic conditions was observed. The dog was discharged with recommendations to the owner to continue administration of the cisapride and sucralfate as previously instructed, increase the dosage of omeprazole (1.5 mg/kg [0.68 mg/lb], PO, q 24 h) and prednisone (1 mg/kg, PO, q 24 h), commence administration of cephalexin (30 mg/kg [13.6 mg/lb], PO, q 12 h for 3 weeks) and tramadol (4 mg/kg [1.8 mg/lb], PO, q 12 h as needed), and strictly adhere to feeding of a commercial elimination diet because of the possibility that an adverse food reaction could potentially explain the dermatologic and esophageal abnormalities. It was recommended that the dog be returned to our facility 7 days later for repeat esophagoscopy and esophageal bougienage.

The dog was returned to our facility 12 days later (day 40). The PEG tube had been inadvertently dis-

lodged, but the dog was now tolerating oral ingestion of canned food with no dysphagia or regurgitation (regurgitation was only evident when kibble was fed). Administration of prednisone and cephalexin and feeding of the elimination diet had been discontinued within the preceding 2 days.

Esophagoscopy revealed a 12-mm-diameter stricture and noticeable improvement of the esophagitis (Figure 1). With the exception of the stricture and 1 small area distal to the stricture, most of the esophageal mucosa appeared grossly normal or had only mild mucosal irregularities. The esophageal stricture was dilated with rigid bougies^d (diameter, 14, 15, and 17 mm).

The owner was advised to continue administration of the omeprazole, cisapride, and sucralfate and to resume administration of the prednisone (1 mg/kg, PO, q 24 h) and feeding of the elimination diet. The ultimate objective was to determine whether the esophagitis and dermatologic condition could be controlled with dietary management alone. Although information obtained during a telephone conversation with the owner 2 weeks later indicated that the dog was doing well with no dysphagia or regurgitation of canned food (kibble had not been fed), the owner admitted to discontinuing the elimination diet a few days before the telephone call. Efforts to obtain additional follow-up information during the next 6 months were unsuccessful.

Discussion

The most common causes of esophagitis in dogs are gastroesophageal reflux secondary to anesthesia, hiatal hernia, persistent or chronic vomiting (reflux esophagitis), ingestion of caustic substances, medication retention within the esophageal lumen (pill esophagitis), thermal injury as a result of radiation therapy, and esophageal foreign bodies.^{1,2} Reflux esophagitis associated with Zollinger-Ellison syndrome and pyrogranulomatous esophagitis, secondary to pythiosis and spirocercosis, has also been reported in dogs.³⁻⁵ The information reported here describes a dog with EE in association with allergic skin disease that was presumed to be secondary to food allergies or aeroallergens. Eosinophilic esophagitis is a relatively new and emerging disease in humans, and it is commonly associated with allergies.^{6,7} To the authors' knowledge, this report represents the first description of EE in a dog.

Eosinophilic esophagitis was first reported in humans in the late 1970s.^{8,9} Since that time, the body of literature regarding EE has grown rapidly, and there is evidence of an increasing prevalence of EE.^{6,10,11} Eosinophilic inflammation of the esophagus is not specific for EE because systemic eosinophilic disorders (eg, eosinophilic gastroenteritis and hypereosinophilic syndrome) and various noneosinophilic disorders (eg, reflux esophagitis, parasitic and fungal infections, drug injury, and neoplastic conditions) may also result in the recruitment of eosinophils to the esophageal mucosa.^{7,11} Currently, the diagnosis of EE in humans relies on characteristic clinical features and endoscopic findings, large numbers of eosinophils within the esophageal mucosa (≥ 15 to 20 eosinophils/hpf in 1 or more biopsy specimen), persistence of clinical signs despite acid suppression, and exclusion of other causes of eo-

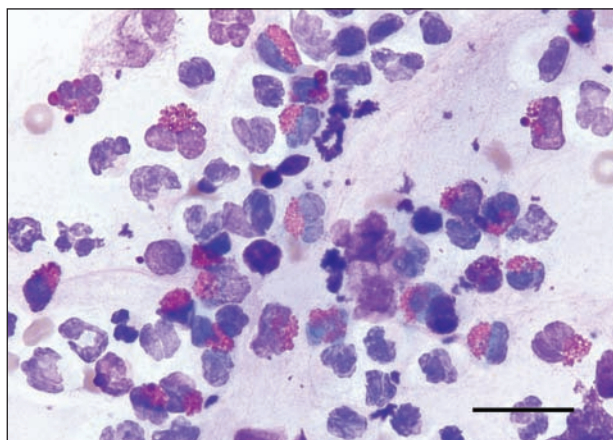


Figure 2—Photomicrograph of a cytologic specimen obtained from the esophagus of the dog on day 28. Notice the eosinophilic and neutrophilic inflammation. Wright-Giemsa stain; bar = 20 μ m. See Figure 1 for remainder of key.

sinophilic infiltration.^{6,12,13} Eosinophilic esophagitis in humans is predominantly found in males and young adults, with common clinical signs including dysphagia, food impaction, heartburn, and chest or epigastric pain.^{6,14,15} Endoscopic features are numerous and include attenuation of the subepithelial vascular pattern, linear furrowing (fissure lines), mucosal rings or proximal strictures, surface exudates (microabscesses or plaques), erosions or ulcers, and mucosa that readily tears during dilation of the esophagus.^{14–16} However, gross esophageal lesions may be subtle or not apparent. In addition, extremely fragile, delicate, and inelastic mucosa (termed crepe-paper mucosa) has also been described.¹⁷ Causes of esophageal eosinophils other than primary EE can usually be elucidated from the history and initial clinical signs, histologic examination of specimens obtained from the esophagus or other sections of the gastrointestinal tract, and failure to resolve clinical signs or the persistence of esophageal eosinophils following treatment with antisecretory medications.¹² After EE has been diagnosed, affected humans typically undergo exhaustive allergy testing to elucidate associated environmental and food allergens.^{7,11,12}

Eosinophilic esophagitis was diagnosed in the dog of this report on the basis of clinical signs, endoscopic and histologic findings, failure to respond to treatment with antireflux medications, and a favorable clinical response to administration of glucocorticoids and feeding of an elimination diet. The proximal location of the esophageal stricture, surface exudates, extremely fragile mucosa, and > 25 eosinophils/hpf in esophageal mucosal samples (before and after administration of antireflux medications) are consistent with a diagnosis of EE in humans. The allergic skin disease is also consistent in that 70% of humans with EE are described as having allergies (currently or in the medical history).⁷ However, the degree of esophageal ulceration in the dog reported here is not typical of EE in humans.¹⁶ Potential reasons for the esophageal ulcers include a late stage of the condition when diagnosed, species differences in disease expression, or concurrent GERD.

Gastroesophageal reflux associated with acute vomiting and anesthesia at the time of the surgery to correct GDV was initially thought to be the main cause of the esophagitis and esophageal stricture in the dog. However, failure to respond to treatment with antireflux medications and the density of eosinophilic inflammation evident during evaluation of esophageal biopsy specimens ultimately led to a diagnosis of EE. Because the onset of regurgitation began 15 days after the diagnosis and surgical treatment of GDV, it appeared likely that gastroesophageal reflux may have played a role in the esophagitis and stricture formation of this dog. In humans, there appears to be a complex and intricate relationship between GERD and EE.¹³ These 2 disorders may coexist but be unrelated, or each disorder may contribute to or cause the other. Therefore, responses to treatment with antireflux medications are expected, and differentiating EE from GERD can be problematic.^{13,18} Although the histologic and cytologic findings of > 25 eosinophils/hpf in the esophageal tissues of the dog reported here are most supportive of a diagnosis of EE, it is possible that gastroesophageal reflux asso-

ciated with GDV may have exacerbated the EE (and caused the stricture), which led to the close temporal relationship of GDV and regurgitation. Although reflux esophagitis can be associated with esophageal eosinophils in humans, the infiltration is typically mild (< 7 eosinophils/hpf).^{11,13} Other mammals have been used to study histologic changes associated with GERD, and neutrophils, plasma cells, and lymphocytes are typically evident, whereas eosinophils are rarely reported.^{19–22} Although the exact number of eosinophils in an esophageal biopsy specimen that will be confirmatory of EE in humans is currently unknown, there is a growing consensus that ≥ 15 to 20 eosinophils/hpf in at least 1 specimen is diagnostic.^{6,12,13,23}

In addition to determining the density of esophageal eosinophils, clinicians must rule out other diseases associated with eosinophilic infiltration of the esophagus before a diagnosis of primary EE can be established. In the dog reported here, clinical signs and results of histologic examination of gastric biopsy specimens did not support a diagnosis of diffuse eosinophilic gastrointestinal disease, and results of the CBC and imaging examinations did not support a diagnosis of hypereosinophilic syndrome. Furthermore, esophagoscopy and histologic and cytologic examination of specimens failed to reveal evidence of fungal, parasitic, or neoplastic disease. Although examination of a fecal sample was not performed for this dog, *Spirocerca lupi* was considered highly unlikely given the lack of esophageal nodules and granulomatous inflammation.⁵ The only disorder that could not be excluded for this dog was reflux esophagitis. However, the lack of response to treatment with antireflux medications, as determined on the basis of clinical signs, esophagoscopy, and results of cytologic evaluation, suggested that ongoing gastroesophageal reflux was an unlikely cause of esophagitis in this dog.

Although the optimal treatment of humans with EE has yet to be defined, current recommendations include a combination of avoidance of allergens and systemic or topical administration of corticosteroids.^{6,12} Antisecretory treatment, esophageal dilation, and drugs such as purine analogues or monoclonal antibody against interleukin-5 may also be indicated in some patients.^{6,12} Avoidance of dietary allergens, as determined on the basis of results of skin prick or atopy patch testing, is common, and multiple reports^{6,7,24} indicate support for the use of dietary manipulation in controlling EE. In the dog reported here, atopic dermatitis or food allergies had been diagnosed on the basis of dermatologic signs. Unfortunately, we were unable to determine whether the EE or dermatologic disease could be maintained in remission with dietary modification alone because the owner was not compliant in feeding an elimination diet. Treatment with oral administration of prednisone and intralesional administration of triamcinolone was used in conjunction with dietary manipulation in this dog to control esophageal inflammation and decrease fibroblast proliferation. In humans, systemic administration of glucocorticoids is effective at decreasing the severity of clinical signs associated with EE, number of esophageal eosinophils per hpf, serum IgE concentrations, and eosinophil counts.^{6,12} However, topical treatment with orally administered fluticasone propionate

or viscous budesonide is more commonly used because of the adverse effects of systemically administered corticosteroids.^{6,12,25} Although the dog reported here was only given modest doses of prednisone (administered orally) and triamcinolone (administered intralesionally at the stricture site), substantial clinical and endoscopic improvement was evident after glucocorticoid treatment. Topical administration of glucocorticoids was not attempted.

Although treatment with antireflux medications is not typically effective as the sole course of treatment in patients with EE, acid suppression may be beneficial in some patients.^{6,12,13} Because of the complex association between EE and GERD, some clinicians argue that treatment with antireflux medications is indicated in the management of patients with both disorders.¹³ For the dog reported here, treatment with antireflux medications was ineffective when used as the sole treatment, but it was continued because of the possibility of concurrent EE and GERD.

To the authors' knowledge, this is the first report of a dog with EE. Although we cannot determine whether the esophageal stricture was secondary to the EE or was a reflux injury associated with the GDV, strictures in the proximal portion of the esophagus are consistent with EE in humans. As is common for humans with EE, the dog of this report had signs of allergic disease and responded to glucocorticoid treatment, esophageal bougienage, and dietary manipulation. Unfortunately, owner compliance was poor, and the long-term outcome for this dog remains unknown. The information provided here illustrates the need to consider EE as a differential diagnosis for dogs with esophagitis and esophageal strictures and highlights the importance of obtaining biopsy or cytologic specimens from the esophagus in animals with esophageal disease.

- a. Iams Response Diet KO, The Iams Co, Dayton, Ohio.
- b. Olympus GIF-VET PQ 140 gastroscope, Center Valley, Pa.
- c. Purina Veterinary Diets LA, Nestlé Purina, St Louis, Mo.
- d. Savary-Gilliard rigid dilators and guidewire, Wilson Cook, Winston-Salem, NC.
- e. ConMed Endoscopic Technologies, Billerica, Mass.

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