A 44-year-old man was admitted to our institution because of the appearance of painful cutaneous, oral, and genital vesicles. His previous medical history was unremarkable. He had had pharyngitis treated with amoxicillin 1 month before the hospitalization. On physical examination, symmetrically erythematous plaques, covered with vesicles, were seen. Because of subsequent dysphagia for solid food, he underwent esophagogastroduodenoscopy.

The endoscopic examination revealed esophageal mucosal exfoliation with multiple linear and confluent furrows in the upper and middle esophagus (Fig. 1) that were less severe in the lower esophagus. The gastric and duodenal mucosa was preserved, without mucosal breakage. Biopsy specimens were taken from lesions in the upper and middle esophagus, with stripping of the mucosa on withdrawal of the biopsy forceps (Video 1). The histologic examination showed suprabasal clefting, cells with a tombstone appearance, and prominent acantholysis, suggestive of pemphigus vulgaris (Fig. 2). At the same time, a skin lesion biopsy was performed, which confirmed the diagnosis (Fig. 3).

Systemic corticosteroid treatment was started but was unsuccessful; during subsequent immunotherapy and the intravenous administration of monoclonal antibodies, the symptoms and signs decreased.

Pemphigus vulgaris is a rare autoimmune disease in which bullae of the skin and oral mucosa form as a result of acantholysis mediated by circulating immunoglobulin G autoantibodies against intercellular antigens of stratified epithelia. Pemphigus vulgaris can involve other mucosal surfaces that have squamous epithelium, such as those of the esophagus, nasopharynx, conjunctivae, cervix, and anus [1]. The prevalence of esophageal involvement in autoimmune bullous skin disease is 67.8% [2], but esophageal involvement may be underrecognized or misdiagnosed without a proper endoscopic evaluation.
Commonly, it is characterized by vesi-
cles, superficial erosions, red and erythe-
matus longitudinal lines, a circumferen-
tial crack with peeling, and a whitish mu-
cosa with extensive bleeding along the
entire organ. Pemphigus vulgaris may also
be induced by drugs, including d-penicilla-
mine and angiotensin-converting enzyme
inhibitors [4, 5]. Corticosteroids and im-
munosuppressive agents are considered
the first-line treatment. If untreated, pem-
phigus vulgaris can be fatal.

Endoscopy_UCTN_Code_CCL_1AB_2AC_3AH

Competing interests: None

References
1 Bystryn JC, Rudolph JL. Pemphigus. Lancet
2005; 366: 61–73
2 Galloro G, Migagnola M, de Werra C et al. The
role of upper endoscopy in identifying
oesophageal involvement in patients with
oral pemphigus vulgaris. Dig Liver Dis 2005;
37: 195–199
3 Hokama A, Yamamoto Y, Taira K et al. Esoph-
agitis dissecans superficialis and autoimmune
bullous dermatoses: a review. World
J Gastrointest Endosc 2010; 2: 252–256
4 Duhra P, Foulds I. Penicillin-induced pem-
phigus vulgaris. Br J Dermatol 1988; 118:
307
5 Ruocco V, Satriano R, Guerrera V. “Two step”
pemphigus induction by ACE-inhibitors. Int

Bibliography
DOI http://dx.doi.org/
Endoscopy 2015; 47: E271–E272
© Georg Thieme Verlag KG
Stuttgart · New York
ISSN 0013-726X

Corresponding author
Paolo Cecinato, MD
Unit of Gastroenterology and Digestive Endoscopy
Arcispedale Santa Maria Nuova-IRCCS
Reggio Emilia 42123
Italy
Fax: +39-0522-295941
paolocecinato@libero.it