Infectious diseases, hormone abnormalities or fluctuations, some drugs and foods, and even neoplasias have been linked to EAC lesions. Annually recurring EAC may also involve seasonal environmental factors such as increased temperature or insect bites. A clear causal agent, however, cannot be identified in most cases (idiopathic EAC).

With regard to treatment, topical and systemic corticosteroids may alleviate the pruritus, but they cannot halt the progress of the lesions, which may involve the entire chest, back or neck. Characteristic of EPSR and annually recurring EAC is the gradual and spontaneous regression of the lesions with the arrival of cooler seasons. Long-term follow-up has recorded recurrences in the first 2-5 years, with subsequent definitive resolution. Other publications, however, suggest a longer duration of the disease.

Although EPSR has been described and subsequently reported in high-impact scientific journals, some authors question that it has sufficient clinical pathologic entity to be considered as an independent disease and they prefer to consider it as a peculiar variant of recurring figurate erythemas such as annually recurring EAC.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

Abdominal Pseudohernia Due to Herpes Zoster

Pseudohernia abdominal por herpes zóster

To the Editor:

After attending the emergency department of another hospital, an 84-year-old man was diagnosed with left abdominal herpes zoster (HZ). Because clinical signs had developed more than 72 hours earlier, no earlier treatments were administered. One week later, the patient came to our outpatients due to the sudden appearance of an asymptomatic mass in the area affected by HZ. Five years earlier he had developed a rectal neoplasm that was treated with surgery and radiation therapy. Physical examination revealed hyperesthesia and lesions in the crusting phase on dermatomes T10 to T12. Painless, reducible bulging of the abdominal wall that increased with Valsalva maneuvers was evident in the area affected by HZ (Fig. 1). A midline laparotomy scar showed no signs of complication. An abdominal computed tomography scan was requested to rule out abdominal mass or hernia. The results revealed thinning of the abdominal wall without evidence of hernia. An electroneuromyographic study revealed no alterations. Given the temporal relationship between the appearance of the rash and the protrusion, the case was oriented as abdominal pseudohernia due to HZ. After 8 months, the patient showed a complete clinical recovery (Fig. 2).

References


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While sensory alterations are one of the main neurologi-
cal complications of HZ, motor nerves can also be affected,
resulting in paralysis of the facial muscles, extremities,
diaphragm, or abdominal muscles, in some cases with vis-
ceral involvement.

Abdominal pseudohermia is a protrusion limited to the
abdominal wall without an actual underlying defect. The
first case of paralysis of the abdominal musculature caused
by HZ was described in 1886 by Broadbent.2 Despite the high
incidence of HZ, this entity is only observed in between
0% and 2% of cases.3 It manifests as abdominal distention
with unilateral bulging on the affected side. The T11 der-
matome is the most affected, followed by T12 and T10.4
Symptoms can appear 2 to 6 weeks before the rash, but
usually develop about 2 weeks after.5 It has been pre-
dominantly described in elderly patients and those with
hematologic neoplasms, as well as immunocompromised
individuals.5

The exact underlying mechanism is not entirely clear, but
it is thought to involve viral infection at the level of the ante-
rior horn of the spinal cord as a consequence of neural spread
of the varicella-zoster virus from the dorsal root ganglia.6
Pathological studies5,7 have demonstrated ganglion lesions
combined with degeneration of the sensory and motor
roots together with severe neuritis, which may explain
the electrophysiological findings characteristic of the
disease.

Diagnosis is primarily clinical, based on temporal corre-
lation of HZ with the appearance of abdominal distension.
Physical examination may reveal decreased or absent seg-
mental reflexes.5 An electroneuromyographic study can be
useful to confirm diagnosis, although alterations are ob-
served in only 35%6,8 of cases. Abdominal computed
tomography shows a thinned abdominal wall and rules out
the presence of an abdominal mass or hernia. Gadolinium-
diethylenetriamine penta-acetic acid (DTPA) nuclear mag-
netic resonance imaging can help define the extent of
inflammation and exclude compression of the spinal nerve
roots.9

The differential diagnosis should include diseases that
present with alterations in the innervation of the abdom-
inal wall musculature and can cause pseudohermia, such as
lumbar hernia, polyradiculoneuropathy, diabetic neuropa-
thy, and syringomyelia.

Treatment is the same as for HZ, with antiviral drugs and
analgesia if required. Short courses of corticosteroids10 have
also been used for their anti-inflammatory effects, as well as
multiple vitamin preparations,5,7 which can help restore
damaged nerve fibers, although there is little evidence to
support the use of these treatments.

The prognosis for motor weakness is usually good, with
complete or near complete recovery in 55% to 75%10 of
cases within a period of 2 to 18 months.3 The most
common complication of pseudohermia is constipation,4
although other complications, including paralytic ileus
and voiding disorders,1 have been described in 19.4%6 of
patients.

In conclusion, abdominal pseudohermia is a rare compli-
cation of HZ that usually has a good prognosis. Although
the suspected diagnosis is clinical, it is advisable to perform
a noninvasive imaging test to rule out a true hernia.

Figure 2 Complete resolution of pseudohermia after 8 months
of follow-up.

Conflicts of interest
The authors declare no conflict of interest.

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Mucous membrane pemphigoid: When the mouth can give a clue to the diagnosis of an esophageal stenosis of unknown origin

Penfigoide de mucosas: cuando la mucosa oral puede ser la clave para el diagnóstico de una estenosis esofágica de origen desconocido

Dear Editor:

Mucous membrane pemphigoid (MMP) is an uncommon heterogeneous group of autoimmune subepidermal blistering disorders which predominantly involves mucosal membranes. Diagnosis and treatment are challenging and delay may cause severe scarring and complications such as esophageal and urethral stenosis, conjunctival synechia and blindness.

An 88-year-old woman with a 9-year history of dysphagia secondary to an indeterminate esophageal stenosis, requiring several endoscopic dilatations (Fig. 1), was referred to our clinic for evaluation of oral erosions. Examination revealed erosive gingivitis, and extensive oral and genital erosions (Fig. 2). Biopsies of vulvar lesions were non-specific and direct immunofluorescence (IFD) of non-affected genital and labial mucosa were negative. Indirect immunofluorescence on salt-split skin (IIF) revealed IgG antibodies binding to the epidermal side of the blister. IIF showed IgG deposition at the basement membrane. ELISA tests were negative for anti Dg1, Dg3 and BP180 antibodies. Immunoblotting of non-affected epidermal extracts was negative for IgG: BP230, BP180, 210 kDa envelopakin, 190 kDa periplakin, Dg1 and Dg3. Immunoblotting of recombinant protein of C-terminal domain of BP180 (BP180ct) detected IgG reactivity of patient serum. A diagnosis of MMP was established and treatment with prednisone (30 mg/day) in a tapering regimen, dapsone 50 mg/day and tacrolimus in a 2 mg/liter mouth rinse formulation was initiated. Dysphagia, oral and genital erosions remitted, but the patient has developed a scarring fibrosis of the vulva with fusion of labia and urethral meatus.

Erosive esophagitis (EE) is a common finding in esophagogastroduodenoscopy (EGD) of patients with gastroesophageal reflux disease (GERD), drug-induced mucosal damage, infections, malignancies and autoimmune disorders. Among autoimmune disorders, a possible under-diagnosed pathology is MMP. The frequency of esophageal involvement in MMP is between 2% and 30%, and this may be an underestimation as EGD is only performed on symptomatic patients.

In patients with MMP and esophageal lesions, a mean of another 3 mucosal areas are involved, and the oral cavity is affected in 86% of the cases. Dysphagia can signal esophageal involvement, although clinically it can be difficult to distinguish it from odynophagia. For all the above-mentioned reasons, performing an EGD on every newly diagnosed patient with MMP has been suggested. Although, EGD is not free of complications and not always available, we agreed with other authors that it should be especially indicated in symptomatic patients or patients with involvement of several mucous membranes.

The mouth is the beginning and the most accessible portion of the digestive tract, and as EGD is performed with a transnasal videogastroscope in these patients, oral exploration may be omitted. In any patient with esophageal erosions, scarring or stenosis, the oral cavity must be clinically explored. The presence of gingivitis or erosions makes examining the anogenital area, nose, throat, eyes and skin necessary in order to rule out MMP, and to evaluate the severity of the disease.

Diagnosis and treatment of MMP can be challenging. In our patient, IIF revealed an epidermal side positivity, which is compatible with bullous pemphigoid, lichen planus pemphigoides and MMP. This finding excludes the diagnosis of acquired bullous epidermolysis, P200 pemphigoid and MMP anti-laminin 332. Finally, immunoblotting was positive for BP180ct, a very specific finding of MMP. On the basis of clini-

Figure 1  Esophagogastroduodenoscopy. Friable esophageal mucosa with erosions and strictures.