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Abdominal-wall postherpetic pseudohernia

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Abstract

Herpes zoster affects 10–20% of the general population. Motor complications sometimes occur in the segments corresponding to the involved sensory dermatomes causing abdominal wall pseudohermias. We present a case of a 57-year-old woman with herpes zoster characteristic rash following T11–T12 right dermatomes. Ten days after dermatologic manifestations onset, she had developed a protrusion at the abdominal wall on the right flank. The electroneuromiography confirmed axonal motor commitment, and morphological defects were ruled out by ultrasonography. The bulge totally disappeared after 4 months of observation. Postherpetic pseudohermia must be suspected when a patient develops signs and symptoms of motor dysfunction that coincide with or follow a herpes zoster eruption resulting in abdominal-wall herniation. A review of the literature concerning these extremely exceptional sequelae of herpes zoster is presented.

Keywords

Pseudohermia - Herpes zoster - Abdominal wall hernia - Paresis - Abdominal distention

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Abdominal-wall postherpetic pseudohernia

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35 **Keywords** Pseudohernia · Herpes zoster · Abdominal
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Introduction

37
38 Herpes zoster affects 10–20% of the general population
39 [1] and is caused by reactivation of a latent neurotropic
40 virus (Varicella-zoster) many years after initial infec-

tion. The virus have a predilection for the posterior root
ganglia; thus, the majority of neurological complications
are sensory. However, motor complications sometimes
occur in the segments corresponding to the involved
sensory dermatomes [2], causing abdominal wall weak-
ness that can present with abdominal or flank bulges
mimicking abdominal-wall hernias [3].

The first case of motor weakness following herpes
zoster was reported in 1886 by Broadbent [4]; and until
today, there are only 20 cases reported in the medical
literature. We report an original case of dermatomal
herpes zoster infection with subsequent abdominal
muscle weakness and a pseudohernia formation. The
current literature is summarized, and management rec-
ommendations are suggested (Table 1).

Report of case

A 57-year-old woman came to the dermatology doctor's
office complaining of vesicles and pain in her right flank.
On physical examination, she was noted to have con-
fluent vesicles with erythematous base, and a charac-
teristic rash that followed the distribution of T11 and
T12 dermatomes at her right flank. Clinical diagnosis of
herpes zoster was done, and treatment with acyclovir
3,200 mg/day for 7 days was initiated. Seventeen days
after the onset of dermatologic manifestations, the
patient noticed a progressive bulge in her right flank,
which increased with cough and straining. The patient
denied pain, nausea or alterations in bowel movements.
The patient consulted a surgeon, who requested an
abdominal ultrasound; however, no morphologic altera-
tions were found. She came back to our office; at this
point, we could see herpetic hypertrophic scars at the
abdominal wall without active lesions (Fig. 1), and also
confirmed the bulge of the right flank that protruded
even more with Valsava's maneuver (Fig. 2). There was
no pain during abdominal palpation, and no viscerome-
galy was observed. The patient was asked to accept
the performance of an electroneuromiography (EMG) at

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Table 1 Comparison of reported cases of postherpetic abdominal-wall pseudohermias in the last 10 years

Reported case	Age/Sex	Beginning	Treatment	Remission (month)
Hanakawa et al. [7]	54 years/♀	15 days After cutaneous eruption	Acyclovir 1,500 mg/10 days	1
Healy et al. [2]	84 years/♂	14 days	Corset	6
Vincent and Davis [1]	64 years/♂	14 days	Amitriptyline 25 mg/dia*	3
Zuckerman and Siegel [11]	78 years/♀	21 days	Observation	12
Hindmarsh et al. [9]	64 years/♀	14 days	Observation	1.5
Barroso (2002)	72 years/♂	20 days	Observation	12
Kesler et al. [13]	82 years/♂	30 days	Surgery (Refused)	4
Safadi [3]	57 years/♂	90 days	Observation	5

79 the site of lesions, which showed positive acute waves in
 80 the right external oblique muscle, indicating acute
 81 muscle denervation. These findings, associated with
 82 physical examination, allowed us to conclude that the
 83 protrusion was a consequence of muscle paralysis due to

motor axon lesion of T11 and T12 dermatome enervation. After 3 months of observation the bulge had remitted entirely (Fig. 3).

Discussion

Herpes zoster infection typically involves the posterior root ganglia, and therefore most of the symptoms are sensory. Motor involvement can occur in the same distribution, but is uncommon [3], with an estimated incidence of 1–5%, although this may be underestimated [1, 5]. Thomas and Howard [6] studied 1,210 patients with cutaneous herpes zoster diagnosis, and found 61 with motor involvement and two with abdominal-wall paresis.

The pathogenesis of segmental zoster paresis remains uncertain, but the most likely cause is direct spread of virus from the sensory ganglia to the anterior horn cells or anterior spinal nerve roots, or both [7]. The resulting weakness usually develops in those muscles innervated by the affected cord segment that corresponds to the cutaneous manifestation [5]; there are reports of only a few patients with such a remote topographic dissociation between the skin lesion and the paretic segment [8].

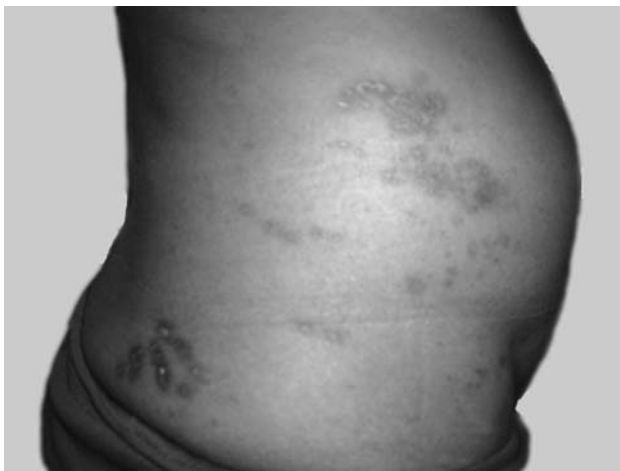


Fig. 1 Hypertrophic herpetic scars following T11 and T12 dermatomes enervation



Fig. 2 Right flank abdominal protrusion 30 days after the onset of herpes zoster



Fig. 3 Complete resolution of abdominal protrusion after 3 months

106 Segmental zoster paresis affects diaphragmatic and
 107 abdominal-wall muscles, face, upper and lower limb,
 108 bladder, urinary and gastrointestinal viscera, resulting
 109 variously in urinary retention, cystitis and colonic
 110 pseudo-obstruction. Only abdominal-wall muscle
 111 paresthesia was present in our patient [2].

112 Symptoms of focal muscle paresis usually appear
 113 within 2–3 weeks of the appearance of the rash [9], and
 114 the onset of weakness is usually abrupt, reaching peak
 115 levels within hours or days [1]. Herpes zoster paralysis
 116 has been described in middle-aged or elderly persons,
 117 patients with underlying hematological malignancies,
 118 and immunocompromised individuals [2, 9–11]. Our
 119 patient had no history of immunodeficiency or hema-
 120 tological malignancies but the age, 57 years, was com-
 121 patible with the epidemiology.

122 Diagnosis is suspected by previous or present history
 123 of herpes zoster associated with abdominal-wall herni-
 124 ation. Physical examination shows reduced or absent
 125 segmental reflexes [2]. To confirm paralysis, a nerve-
 126 conduction study must be done; electroneuromiogra-
 127 phy is used for this [10]. MRI with gadolinium-DTPA
 128 can help to delineate the extent of the inflammation,
 129 and to exclude the local entrapment of spinal nerve
 130 roots, known to be a predisposing factor of herpes
 131 zoster [7, 8].

132 Differential diagnosis includes lumbar hernias, that
 133 can occur spontaneously through the inferior lumbar
 134 triangle of Petit or the superior triangle of Grynfeltt [12],
 135 and other conditions such as diabetic truncal neuropathy,
 136 Lyme's disease, polyradiculoneuropathy, syringo-
 137 myelia and prolapsed L1–L2 intervertebral disc [13].

138 The prognosis for this motor weakness is good, with
 139 complete recovery in 55–75% of patients within
 140 6–12 months of the onset, but some patients remain with
 141 permanent weakness [1, 7, 10, 14]. Our patient was
 142 completely recovered after 4 months. Apparently, there
 143 is not a relationship between the degree of paralysis and
 144 complete recovery [5], and also there does not seem to
 145 be any pharmacologic way of hastening recovery. The
 146 only things to do are observation, use of corsets and
 147 expectation.

Conclusion

Postherpetic pseudohernia must be suspected when a patient develops signs and symptoms of motor dysfunction that coincide with or follow a herpes zoster eruption resulting in abdominal-wall herniation. Recognizing this entity is important, because it is a potentially reversible disease and does not require surgical intervention.

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