

## Horner Syndrome: A Rare Complication of Cervical and Thoracic Herpes Zoster Infection<sup>☆</sup>



### Síndrome de Horner: una rara complicación del herpes zóster cérvico-torácico

To the Editor:

Postherpetic neuralgia is the most frequent and best-known complication of herpes zoster. Lesser-known complications include encephalitis, meningitis, and peripheral motor paralysis.<sup>1</sup> Horner syndrome (miosis, ptosis, enophthalmos, and/or anhidrosis) is a rare complication of cervical and thoracic herpes zoster.

We report the case of a 74-year-old woman who presented to our emergency department with a 10-day history of progressively appearing painful lesions on her left arm, left chest, and left upper back. Past history included hiatal hernia and endometrial adenocarcinoma treated 11 years earlier with total hysterectomy, bilateral adnexectomy, bilateral pelvic and para-aortic lymphadenectomy, and adjuvant radiotherapy. She reported no systemic symptoms and no motor symptoms involving the arm. Physical examination revealed clustered vesicles and bullae on an erythematous base with some areas of surface erosion, in a C8 and T1 dermatomal distribution on the left side (Fig. 1). In addition, the patient had ptosis of the left upper eyelid; her left pupil was smaller than the right, and her skin was drier on the left side of her face (Fig. 2A), with no other focal neurologic abnormalities. Biochemistry, a complete blood count, and a chest radiograph yielded no findings of interest. The patient was diagnosed with



**Figure 1** Vesicles and bullae forming herpetiform clusters on an erythematous base in the C8-T1 dermatomal distribution on the left side.

cervical and thoracic herpes zoster associated with Horner syndrome of the left eye. Antiviral therapy was initiated with 1 g of valacyclovir every 8 hours for 7 days, together with an alternating regimen of 1 g of acetaminophen every 8 hours and 575 mg of metamizole every 8 hours for pain. The skin lesions gradually improved, and Horner syndrome resolved within 15 days with no sequelae (Fig. 2B).

The best-described ocular complications of herpes zoster include swelling of the eyelid, keratitis, conjunctivitis, iridocyclitis, episcleritis, retinal necrosis, central retinal artery occlusion, and ophthalmoparesis.<sup>2</sup> Horner syndrome, or oculosympathetic palsy, is characterized by ptosis, miosis, anhidrosis, and/or enophthalmos, and was classically attributed to space-occupying lesions in the vertex of the lung, such as Pancoast tumor. It has seldom been described as an ophthalmic or thoracic complication of herpes zoster.<sup>3</sup>

In the spinal cord, the ocular sympathetic pathway originates in the cells of the intermediolateral cell column at the level of segments C8 to T2. Preganglionic fibers emerge from the spinal cord following the ventral roots to form the sympathetic chain, pass through the stellate ganglion, and synapse in the superior cervical ganglion.<sup>4</sup> Given that the agent of herpes zoster infection lies dormant in the dorsal roots of the sensory ganglia of the central nervous system, the most plausible theory for the pathogenesis of Horner syndrome secondary to thoracic herpes zoster is that either the spinal cord or its ventral roots become inflamed owing to viral activation that spreads by contiguity.<sup>5,6</sup> However, in the case of herpes zoster ophthalmicus, other potential mechanisms have been considered, including ischemic neuritis of the third, fourth, or sixth cranial nerves; a mesencephalic lesion caused by axonal spread of the virus; inflammation by contiguity leading to myositis; or even immune-mediated demyelination due to local or remote viral spread.<sup>7</sup>

Our search of the literature for herpes zoster associated with Horner syndrome revealed 11 reported cases (Table 1) of patients aged 24 to 79 years, including 7 men and 4 women. Horner syndrome was associated with herpes zoster involvement of branches of the fifth cranial nerve in 5 cases,<sup>8</sup> and with involvement of cervical and/or thoracic dermatomes in 6 cases. The right side was involved in 7 cases and the left side in 4. Most patients received antiviral therapy (acyclovir or famciclovir). However, Horner syndrome persisted for weeks or years in 6 cases, while recovery was partial in 2 cases and complete in 2 others.<sup>9,10</sup> None of the authors discuss potential factors predicting the persistence of Horner syndrome after herpes zoster resolution. In our view, early antiviral treatment, whether oral or intravenous, reduces the risk of persistence over weeks or years.

We have reported a new case of Horner syndrome as a rare complication of cervicothoracic herpes zoster. This case is significant in that it underscores the need for dermatologists to examine patients for Horner syndrome and recognize it, in order to initiate early antiviral treatment to prevent other ocular complications and so that neurologists and/or ophthalmologists can be involved in a multidisciplinary management approach.

<sup>☆</sup> Please cite this article as: Lobato-Berezo A, Estellés-Pals MT, Gallego-Valdés MÁ, Torres-Perea R. Horner Syndrome: A Rare Complication of Cervical and Thoracic Herpes Zoster Infection. *Actas Dermosifiliogr.* 2015;106:595–597.



**Figure 2** A, The left eye shows miosis, ptosis of the upper eyelid, and greater dryness of the skin (the characteristic triad of Horner syndrome). B, Ocular signs resolved.

**Table 1** Reported Cases of Horner Syndrome Associated with Herpes Zoster.

Case	Sex	Age, y	Dermatome	Side	Treatment	Course
Jarrett, 1967	Male	64	Trigeminal, V2-V3	Right	Topical analgesics and antibiotics	Cleared within 3 weeks
Wimalaratna, 1987	Male	46	T2-T3	Right	—	Persisted
Smith, 1993	Male	53	Trigeminal, V1	Right	Acyclovir, 800 mg 5 times a day, 5 days	Persisted after 1 month
Tola-Arribas, 1997	Male	60	Trigeminal, V1	Left	Acyclovir, 800 mg 5 times a day, 5 days	Persisted after 9 months
Poole, 1997	Female	57	T3-T4	Right	Acyclovir, 200 mg 5 times a day, 5 days	Persisted after 3 years
Gabriel, 2003	Male	79	C7-T1	Left	Oral acyclovir	Persisted after 3 years
	Female	79	C8-T1	Left	Oral famciclovir	Persisted after 10 months
Agudo, 2004	Male	58	T2-T3	Right	Topical acyclovir	—
Pandey, 2005	Female	24	Trigeminal, V1	Right	Acyclovir, 800 mg 5 times a day for 3 weeks, and prednisone, 80 mg a day in tapering doses	Improved after 3 weeks
Sedehizadeh, 2010	Male	64	C3-C4	Right	Intravenous acyclovir and dexamethasone	Cleared in 12 weeks
Falzon, 2011	Female	70	Trigeminal, V2	Left	Intravenous acyclovir, 10 mg/kg every 8 hours	Improved after 3 weeks

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## Acute Disseminated Paracoccidioidomycosis with Molluscoid Lesions in a Young Woman<sup>☆</sup>



### Paracoccidioidomycosis aguda diseminada moluscoide en mujer joven

To the Editor:

Paracoccidioidomycosis (PCM) is a systemic mycosis caused by a dimorphic fungus.

The fungus *Paracoccidioides brasiliensis* lives in the ground and on plants in tropical and subtropical regions. It is endemic to South America and is the most common of the deep mycoses in Paraguay.

Infection occurs by the inhalation of spores during the first decades of life; these spores reach the lungs and can spread to other parts of the body via the blood or lymphatics. The fungus can affect the skin, mucosae, adrenal glands, and other organs.<sup>1-4</sup>

The 2 main clinical presentations of PCM are the chronic form and the acute/subacute form. The chronic form, which accounts for up to 90% of cases, typically affects male farmers between 30 and 60 years of age. The higher frequency in men is due to the presence of 17- $\beta$ -estradiol receptors in the cytoplasm of *P. brasiliensis*. The interaction between the receptors and the female hormone inhibits transformation of the fungus from the mycelial form to the yeast form; this transformation is essential for infection to develop.<sup>3-7</sup>

The acute/subacute form (juvenile type) accounts for less than 10% of cases and is mainly observed in young individuals with a depression of cell-mediated immunity. This form has a rapid clinical course (4 to 12 weeks), with alterations of the monocyte-macrophage system and polymorphous skin lesions (nodular, furunculoid, verrucous, ulcer-granulomatous, molluscoid).<sup>5,7-9</sup>

The gold standard for diagnosis is culture, which must be performed under strict biosafety measures. The fungus grows as mycelia at room temperature and as the yeast in vitro and in tissues at 37°C.<sup>1-5</sup> Other diagnostic methods include serology (immunodiffusion), and molecular studies such as polymerase chain reaction.<sup>1,2,10</sup> Histopathology reveals fungal structures, with well or poorly organized granulomas, depending on the immune response.<sup>1,7</sup>

Commonly associated infectious diseases include tuberculosis (5%-10%), intestinal parasitoses, Chagas disease, syphilis, other superficial and deep mycoses, and AIDS. Important noninfectious diseases that may be associated with PCM are non-Hodgkin lymphoma and some carcinomas.

An association with smoking has been reported in a high percentage of patients.<sup>1,5,8</sup>

Patients with severe forms of PCM, with weight loss of over 10%, respiratory difficulty, and neurological symptoms, must be hospitalized.

The treatment of choice is itraconazole, 200-400 mg/d, by mouth for 6 to 12 months; this regimen achieves cure in 88% to 100% of cases, though there is a recurrence rate of 3%. In severe forms, amphotericin B, 0.8-1 mg/kg/d, is administered by intravenous infusion until an improvement is achieved in the clinical manifestations, after which the patient can be changed to oral treatment.<sup>1-3,7</sup> Other alternatives include trimethoprim-sulfamethoxazole, voriconazole, and fluconazole. Periodic follow-up of clinical, mycologic, radiologic, and immunologic criteria must be performed to determine whether a favorable response to treatment is being achieved. Delays in the initiation of treatment can elevate mortality to up to 30%, and increase the risk of potentially disabling sequelae, such as pulmonary fibrosis.<sup>1-3,5,7</sup>

We present the case of a 21-year-old housewife, an indigenous woman from a humid and wooded rural region of Paraguay. She had a 2-month history of skin lesions on her back, face, neck, abdomen, and limbs. The patient described a feeling of fever, mainly in the evening, jaundice, dyspnea, asthenia, anorexia, and unquantified weight loss. She denied any underlying diseases, smoking, alcohol consumption, or use of recreational drugs.

Physical examination revealed erythematous-violaceous papules and plaques, some with a central depression and bloodstained scab, mainly on the face (Fig. 1). In addition, jaundice, ascites, and submandibular and cervical lymph nodes of 1.5 to 3 cm diameter were detected. Breath sounds were absent in both lung bases. The patient presented fever of 38.5°C, but her vital signs were stable.

Blood test results were as follows: hemoglobin, 7.4 g/dL; hematocrit, 21%; white cell count, 11 800/ $\mu$ L (neutrophils, 82%; lymphocytes, 10%; monocytes, 6%; and eosinophils, 2%); platelets, 124  $\times 10^3$ / $\mu$ L; aspartate aminotransferase, 127 U/L (normal value, 32 U/L); alanine aminotransferase 75 U/L (normal value, 33 U/L); alkaline phosphatase, 4694 IU/L (normal value, 300 IU/L); total bilirubin, 10.1 mg/dL; direct bilirubin, 4.8 mg/dL; indirect bilirubin, 6.1 mg/dL; albumin, 1.3 g/dL (normal value, > 3.5 g/dL); prothrombin index, 50%; activated partial thromboplastin time, 45 seconds. Serology for human immunodeficiency virus, syphilis, hepatitis C virus, hepatitis B surface antigen,

<sup>☆</sup> Please cite this article as: Di Martino Ortiz B, Moreno T, Galeano G, Rodríguez M. Paracoccidioidomycosis aguda diseminada moluscoide en mujer joven. *Actas Dermosifiliogr.* 2015;106:597-599.