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Letters to the Editor

Pneumonitis Due to Hypersensitivity in Infants

Neumonitis por hipersensibilidad en el niño

To the Editor:

Hypersensitivity pneumonitis (HP) is an inflammatory immune illness that affects lung interstice, bronchioli and alveoli in people susceptible to the disease as a result of repeatedly inhaling organic substances.[1-3] This disease is uncommon in children,[2,4] and is often a result of exposure to avian proteins.[2]

A child, 12 years old who has various pets at home, among said pets were pigeons, attended the Emergency Department for a high fever, wheezing, rales as well as intense difficulty breathing. He showed a 3 month clinical history of dry cough, progressive dyspnoea and weight loss. Analysis showed 14,470 leukocytes (83.5% neutrophilis, 11% lymphocytes, and 0.6% eosinophil count) and C-reactive protein at 2.28mg/dl. A chest x-ray showed a diffuse and bilateral reticulonodular infiltrate. Treatment with ceftriaxione, clarithromycin and prednisolone was initiated. A high resolution CT scan showed a bilateral interstitial pattern of diffuse distribution, with multiple hypersensitivity regions with centrilobular pattern and some others in "ground glass opacity" (Figure). Lymphocytosis (82%) was observed in the bronchoalveolar lavage, as well as a CD4/CD8 ratio less than 1%. The lung function tests showed a forced vital capacity (FVC) at 79.5% of predicted value, a forced expiratory volume in the first second (FEV_1) at 70% of predicted value and an FEV_1 /FVC ratio of 74%. The diffusion capacity of carbon monoxide was found to be moderately reduced.

The patient was discharged from hospital and given oral and inhaled corticosteroids. He also said that his family moved the birds out of the house and cleaned and disinfected the cages.

Two months later, the symptoms worsened. The birds had been removed from the patient's home, but there were birds living in adjacent patios. As a result, the local health authorities were alerted of the situation, and they carried out an inspection of the area.

From the moment exposure to the antigen disappeared completely, the patient became asymptomatic. A new chest CT scan was performed that has shown no new alterations, and the functional tests showed only a mild obstructive pattern, with no other manifestations.

A diagnosis of HP is based on clinical findings, which are radiological, physiological and immunological in nature. Pigeon specific precipitine concentration (immunoglobin G) was not determined because it is not essential in the diagnosis or determination of the aetiological agent. If this concentration proves positive, it only confirms exposure to the antigen, if it is negative, diagnosis still may not be excluded because the tests only detect the most frequent antigens and may not detect the implicated antigen.[1,5]

In this case it was not difficult to identify the aetiological agent, because of the previously mentioned environmental context and the positive response to treatment following removal of the birds. The recurrence of symptoms was related to persistence of exposure, due to both the presence of the pigeons in adjacent patios and the fact that the pigeon antigens remained in the environment for 18 months, even after their removal from the house and deep cleaning of the cages.[1]

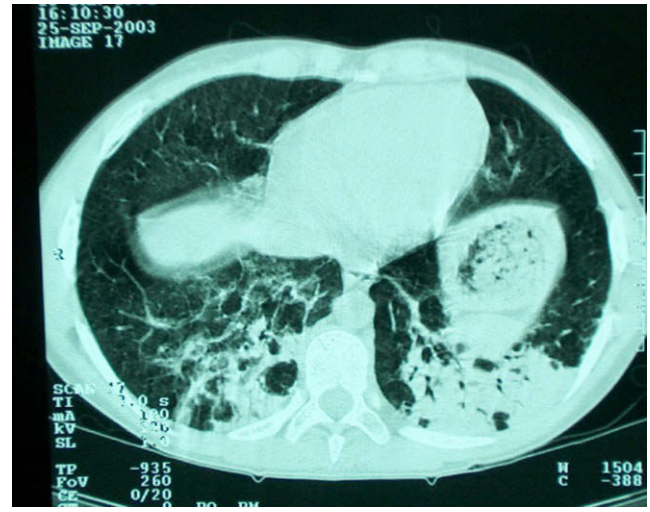

Figure. High resolution CT scan: bilateral interstitial pattern of diffuse distribution, with multiple regions of hypersensitivity with centrilobular pattern and some others in "ground glass opacity".

HP is an uncommon disease in children with unspecific symptoms. The most important factor and the key to diagnosis was the medical history, which allowed us to find the aetiological agent and thus carry out direct and precise treatment, which is fundamental when avoiding serious and irreversible complications such as pulmonary fibrosis.

Acknowledgements

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Maria José Dinis,* Andreia Teles, and Isabel Carvalho

Centro Hospitalar Vila Nova Gaia/Espinho, Vila Nova de Gaia, Portugal

*Corresponding author.
E-mail address: majodinis@gmail.com (M.J. Dinis).

Horner Syndrome Due to First Rib Fracture

Síndrome de Horner causado por la fractura de la primera costilla

To the Editor:

Horner syndrome or oculosympathetic palsy occurs as a result of an interruption in the sympathetic chain which extends from the hypothalamus to the eyeball. It is characterized by a clinical profile of palpebral ptosis, myosis, enophtalmos and at its full stage, hemifacial anhidrosis.

We present the case of a 61 year old patient with antecedents of stable ischaemic cardiopathy and coronary revascularisation surgery 6 years ago. He was seen in the Emergency Department after suffering craneoencephalic and thoracic trauma without losing consciousness, owing to an accidental fall from a height of 2m. Upon admission, the neurological exam proved normal, without thoracic deformities, and presented a haematoma in the upper right area of the rib cage. Cardiorespiratory ausculation was normal, as was that of the abdomen and the extremities. Regarding complementary tests carried out, computerized tomography scan (CT) of the thorax revealed a fracture from the first to the ninth lower right arcs, close to the costovertebral angle, lacking haemopneumothorax images; the cranial CT showed no findings. After 24 h in the observation area, he underwent thoracic surgery, where, at 48 h, signs of right-sided Horner syndrome became noticeable (Figure). An ophthalmologic consultation was requested, which confirmed the diagnosis. Following treatment with intravenous analgesia and respiratory rehabilitation, the patient showed favourable progress, and was discharged from hospital after 5 days. In follow-up, partial recovery from symptoms was observed after 3 months, with no further changes noted at 6 months.

First rib fracture associated with Horner syndrome as the result of thoracic trauma is a rarely occurring pathological phenomenon: following a search of the English-language bibliographic data base MEDLINE (key words: *Horner, chest trauma, fracture rib*) we found only 6 cases published since 1975, one of which was bilateral.[1] Although the lesion mechanism is well known, it is important to emphasise the possible non-immediate or unrecorded presentation of symptoms, as well as the need to understand its presentation in cases of thoracic trauma.

This cause of Horner syndrome is of great clinical relevance due to its rare occurrence and importance in differential diagnosis regarding central neurological lesions in polytraumatised patients.

The causes of this pathological process are numerous. Congenital and postsurgical reasons are among the most common. Additional causes include; penetrating lesions in the spinal region, spinal cord lesions, thoracic epidural anaesthesia, brachial plexus and subclavical artery lesions, superior sulcus tumours, inadequate positioning of the thoracic tube and first rib fractures or tumours.

The sympathetic chains descend vertically in front of the costovertebral joints on each side of the vertebral column. They connect in the front, crossing the intercostal vessels and nerves and are encased by the parietal pleura.

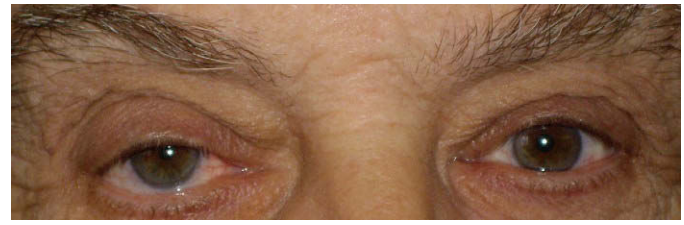

Figure. Traumatic Horner syndrome of the right eye.

The first order sympathetic nerve fibres extend from the posterolateral region of the hypothalamus and end in the intermediolateral column of the spinal cord at level C8-T2. The second order pupillomotor preganglionic fibres exit the spinal cord at the T1 level and enter the cervical sympathetic chain, where they are in close proximity to the subclavian artery and the apex pulmonis. In this section of the trajectory, the sympathetic trunk and the cervicothoracic or stellate ganglio are vulnerable to thoracic trauma, which explains the physiopathological lesion in our clinical case. At times, the lesions are not permanent and the result is full recovery, however; in our case the lesion has remained and the recovery from the syndrome has been only partial.

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Florencio Quero Valenzuela,* Sebastián Sevilla López, and Antonio Cueto Ladrón de Guevara

Departamento de Cirugía Torácica. Hospital Virgen de las Nieves, Granada, Spain

*Corresponding author.
E-mail address: florencioquero@msn.com
(F. Quero Valenzuela).