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Case Report

Pitts-Hoppkins Syndrome Post Severe Exacerbation of Asthma: About a Case: 3-Year-Old Child

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Introduction

In some children recovering from an acute asthma attack, flaccid paralysis similar to poliomyelitis has been observed [1-3]. This condition, first discovered in Australia in 1974, is known as Hopkins Syndrome (HS) [4]. No specific virus was identified in these patients, who were usually correctly vaccinated against poliomyelitis [5]. This disorder primarily affects cells in the anterior horn of the spinal cord. It is characterized by a rapid progression of paralysis usually affecting one limb, leaving the child with severe and permanent weakness. Sensation is usually preserved, and Cerebrospinal Fluid (CSF) analysis often shows a slight increase in white blood cells and a slight increase in protein [6-8]. Magnetic Resonance Imaging (MRI) alterations in the anterior horn have been documented [9]. Some children show signs of an underlying immune deficiency [10]. We report the case of a 3-year-old boy who presented with an episode of Hopkins syndrome.

Case Report

This is a 3-year-old child, known to be asthmatic since the age of 9 months, with good therapeutic compliance, treated on an outpatient basis several times for wheezing dyspnoea attributed to a moderate exacerbation of his asthma, including one requiring hospitalization in France of known asthmatic paternal family.

He was well vaccinated according to the National Immunization Program (PNI), and had good psychomotor development.

Initially admitted to the Children's Hospital in Rabat, in the P1 respiratory disease department, for wheezing dyspnoea, put under nebulization of beta-2 mimetics with bolus of corticosteroids and antibiotic therapy based on C3G, aminoside associated with an antiviral.

Following the non-improvement, the patient was transferred to intensive care for additional care.

At the clinical examination of his admission, we found a tachypneic child, desaturating at 89% on ambient air, signs of respiratory struggle such as suprasternal and intercostal indrawing and thoraco-abdominal rocking; auscultation found bilateral snoring and sibilant rales; tachycardia at 170 bpm; neurologically confused.

A gasometry made showed capnia at 63 mmHg; with a saO2 at 93% under a high concentration mask under 15l of O2. A chest X-ray showing horizontalization of the ribs.

Faced with the aggravation, continuous nebulization was added based on an atropine anticholinergic with adrenaline, injections of magnesium sulphate at 30 mg/kg and beta dose adrenaline at 0.1 gamma/min.

Despite all the therapies deployed, there was a persistence of all the clinical signs mentioned above, hence the decision to mechanically ventilate the patient, by oro-tracheal intubation with sedation.

The evolution was marked by a pulmonary superinfection with the occurrence of a febrile peak at 39.5, an appearance of a pulmonary image not initially existing on the X-ray and the isolation of a Gram-negative Bacillus on the sample. Distal protected, hence a therapeutic escalation to ceftazidime and aminoside.

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Discontinuation of sedation was considered after clinical stability, reduction in oxygen requirements with regression of wheezes.

After gasometric correction and complete awakening, the child's extubation was decided.

The awakening is thus manifested by a regression of the muscular strength noticed in the 4 limbs listed at 2/5.

An electromyogram carried out showing motor axonal polyneuropathic damage affecting the L4/L5 roots bilaterally, which may be part of L4/L5 polyradicular damage or part of the motor form of Guillain Barré syndrome (GBS).

A lumbar puncture in addition performed returned to normal showing no cytological albumin dissociation, GBS therefore ruled out.

A 5-day course of immunoglobulins was started in the patient, with a total recovery of the overall muscle strength of the 4 limbs.

The diagnosis of PITT HOPKINS syndrome post severe exacerbation of asthma was thus made in consultation with the pediatricians of the hospital.

Discussion

Hopkins syndrome is an uncommon condition that affects cells in the anterior horn after an acute asthma attack, usually seen in children with atopic asthma [11]. Usually, this disease manifests itself a few days to a few weeks after an acute asthma attack, characterized by the sudden onset of soft paralysis and, in most cases, progressing to severe muscle degeneration of the affected limb [12].

According to previous studies, during these episodes, the response to corticosteroids is often ineffective and recurrences are usually absent [13]. Some cases show predominantly ventral root involvement on Magnetic Resonance Imaging (MRI), while others show extension of lesions into the anterior parts of the spinal cord [14-16]. Thus, lesions in Hopkins syndrome are not strictly limited to the anterior horn. In some previous studies, it has been reported that peripheral blood lymphocytes of asthma patients produce more cytokines when exposed to allergens than those with skin allergies [17]. However, to date, Atopic myelitis (AD) has similarities to Hopkins syndrome. Numerous reports have confirmed the presence of signs in the central or peripheral nervous system in AD patients with elevated total IgE and concomitant atopic disease [18]. Hopkins syndrome and atopic myelitis differ in the age at which they preferentially manifest, neurological manifestations, and preferential sites of spinal cord involvement. However, the two conditions agree on one essential point: myelitis develops in the presence of atopic disorders, suggesting a link between atopy and inflammation of the spinal cord. Hopkins syndrome is frequently associated with epilepsy. Previous reports do not provide detailed descriptions of the Electroencephalogram (EEG), but slow-wave brain activity was observed in patients of Pitt and Hopkins (1978), while "generalized seizure activity" was present in Singh's patient (1993).

Horuchi et al. described the case of a 22-year-old woman who had an additional episode of myelitis after another asthma attack, although relapses are not usually reported in cases of Hopkins syndrome [4,19,20]. In the differential diagnosis of patients with symptoms similar to our patient, consideration should be given to Joubert's syndrome, Rett's syndrome and Angelman's syndrome [21].

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