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Guillain-Barré Syndrome after Malignant Pertussis: A Case Report

Muñoz-Bonet JI[1], Montero Yéboles R[1], Gil Gimeno R[2], Sebastián Cuevas FJ[1], Roselló Millet P[1*] and Flor Macián EM[1]

[1]PICU, Clinic Hospital, Valencia, Spain

[2]Department of Neurology, Clinic Hospital, Valencia, Spain

[*]**Corresponding author:** Muñoz-Bonet JI, PICU, Clinic Hospital, Valencia, Avenida de Moratalaz 187, 1C. Madrid, Spain, Tel: +55 16 3878-9700; Fax: +33(0)383 646 158; E-mail: coepacheco@gmail.com

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Introduction

Pertussis is an acute respiratory infection produced by *Bordetella pertussis* (BP), affecting the paediatric population. Among the complications associated with this case, "*malignant pertussis*" is the most serious and occurs most commonly in infants less than 3 months associated with pneumonia, serious hypertensive pulmonary disease and a neurological decrement of brain conscious or seizures [1].

The Guillain-Barre syndrome (GBS) is an acute inflammatory polyradiculoneuropathy often associated with a previous condition like an infection, surgery, malignant process or vaccination [2]. The GBS was described after the lockjaw vaccination, influenza, hepatitis and in the combination of lockjaw and diphtheria vaccination [2-8]. However, there are no published reports after the pertussis disease. To our knowledge, we describe the first case with diagnosis based on the symptomatology, electromyography, the chemistry of the cerebrospinal fluid (CSF) and clinical evolution.

Keywords: Bordetella pertussis; Guillain-Barre syndrome; Malignant pertussis; polyradiculoneuropathy; Critically ill polyneuropathy; Tetraparesis

Case Presentation

A child of 44-day-old is admitted to our center showing an acute respiratory failure in the context of diagnosed pertussis (through PCR for BP), undergoing treatment with azithromyzin after diagnosis.

The patient was born by C-section as a result of mother's preeclampsia. She did not receive pertussis vaccination during pregnancy. The patient has a history of low weight for the gestational age (1970 g) and prematurity (35+4) which required his admission during the first 48-hours of life to guarantee intake. Mixed feeding since birth. Mother with productive cough during a month. Other family antecedents not considered.

The physical examination on admission to our hospital was alarming: Weight 3.100 g. HR 164 bpm. RR 60 bpm. SaO_2 69% (with oxygen therapy of high flow and with FiO_2 of 0.85). Blood pressure 100/45 mmHg. General bad appearance with grey paleness color and cross-linking skin. Perioral cyanosis and distal extremities. Present and symmetric pulse. Cardiopulmonary examination showed bad entrance of bilateral air and upper airway transmission noise with noticeable subcostal and intercostal recession. Heart examination normal, no heart murmurs or effleurage.

Blood test showed 45.260 leukocytes/mm^3 with 18.030 neutrophils/mm^3 and 22.590 lymphocytes/mm^3. Hemoglobin 10.3 g/dl with hematocrit of 33% and 570.000 platelets. The chemistry showed CRP of 49.9 mg/dl and the coagulation study showed a prothrombin time of 23%.

On admission, because of hypoxemia and general bad condition, we proceeded to increase blood volume, intubation and connection to mechanical ventilation. Despite this, the patient presented steady hypercapnic acidosis that required an increase of respiratory assistance and inotropic support with a high dose of vasoactive drugs. The evolution to acute respiratory distress syndrome (low PaO2/FiO2 ratio of 63 and high oxygen index of 30, after 72 hours of admission) and refractory pulmonary high blood pressure needing inhaled nitric oxide, inotropic support in dose of cardiopulmonary reanimation and treatment with continuous veno-venous hemofiltration (CVVH) because of renal failure and anasarca. Owing to refractory hypercarbia and the direct relation we had observed between his hypercarbia and worsened pulmonary hypertension, his ventilation support is modified to a high frequency oscillation ventilation (HFOV) with good response in ventilation and subsequent control of pulmonary hypertension. Later satisfactory evolution with better respiratory pattern and hemodynamics.

Since admission, the patient was treated with midazolam and fentanyl for 20 days, in maximum dose of 0.33 mg/kg/h and 3.3 mcg/kg/h respectively, and muscular relaxant with rocuronium for 15 days. Upon completion of the treatment with rocuronium, there were no spontaneous movements with generalized saggy muscular paralysis, absent tendon reflexes, absent corneal reflex but with light reactive pupils. Normal CPK and cholinesterase. Brain ultrasound showed slight tetra-ventricular hydrocephalus with discreet increase of periventricular echogenicity and the EEG showed noticeable

signs of brain light bioelectrical distress-irritation, with discreet reactivity to painful appeals. Auditory and somatosensory evoked potentials were negative. Due to the patient's age, the EMG was more complex, but showed acute denervation and low amplitudes of motor evoked potentials with normal distal latency (tibial nerve). All previous were compatible with axonal sensorimotor polyneuropathy. Considering the results, the EMG and the clinical features, two diagnostic options were propounded: Critical illness polyneuropathy (CIP) or an axonal variant of GBS.

The lumbar puncture showed a CSF slightly xanthochromic with glucose of 40 mg/dL, proteins of 308 mg/dL, 40 erythrocyte/uL, 0 nucleated cells/uL, IgG of 10.2 mg/dL, absent oligoclonal bands IgG and PCR for Bordatella Pertussis and negative HSV 1 and 2. Owing to result of lumbar puncture one month after admission, treatment with I.V. gammaglobulin (2 g/Kg) was initiated. As no progress was evidenced, we proceeded with plasma exchange therapy through plasmapheresis (5 sessions). Following first session, there was small evidence of slow clinical improvement, with rhythmical movements of bilateral blinking, beginning of facial muscles movement with body progress, but with lower limb hypotonicity and limited mobility. There was a gradual improvement throughout the following sessions of plasmapheresis. When the patient was discharged from the PICU, the head support was not complete, but the visual contact and social laugh were normal. The patient showed normal eye movement and could handle objects with both hands and scoop them into mouth. Three months later, the EMG was repeated showing sensory-motor polineuropathy with more motor than sensory affectation, axonal predominance inactive, with moderate or severe subacute reinervation changes in the distal muscles (tibial and median nerves).

Patient´s progress has been good. He required mechanical ventilation through tracheostomy for 5 months. Currently, he is 40-months-old and presents normal neurological development except for a distal paresis in both lower limbs, though not compromising patient's ability to crawl, stand up and wander with support.

Discussion

Guillain Barre syndrome involves symptoms of flaccid areflexic tetraparesis with scarce sensitive symptoms. The evolution of paresis is highly changeable and, in acute cases, can reach complete palsy resulting in breathing disability owing to the weakness of the diaphragm or intercostal muscles [9-11]. This will require an extended hospital stay, in the PICU, with the use of a ventilator. With initial symptoms there can be distal paresis, but there is no deficit of marked sensitivity [9] as in our patient.

The diagnosis is clinically supported by additional tests such as the CSF study showing the albumin-cytological disassociation (high proteins without cells). Proteins are generally normal throughout the initial stages of the illness, increasing after the first week and remaining consistent for several months, even after the clinical recovery [10,11]. In the case of our patient, the CSF study was performed three weeks after being admitted with results matching with this syndrome. We also considered neurophysiological studies. In 80% of patients with GBS there was a deceleration of the conduction velocity and an increase of distal latency (demyelination). However, there is also an axonal variant of the GBS. The EMG results are compatible with this showing an axonal motor polyneuropathy with less sensory affectation and less demyelinization.

Conclusion

The differential diagnosis in our patient is largely based on ruling out the critically ill polyneuropathy (CIP). In both entities (GBS and CIP) axonal affectation can occur, but in the CIP the CSF is normal. CIP has sensitive affectation more often than GBS. Additionally, the clinical damage has a faster recovery in comparison to our patient who was discharged with neurological motor impairment. Finally, the rapid and sustained response to plasma exchange, suggests the intervention of some immune mechanism.

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