OMCR

Oxford Medical Case Reports, 2020;2,59–61

doi: 10.1093/omcr/omaa007
Case Report

CASE REPORT

Congenital nephrotic syndrome as a complication of whooping cough: a case report

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Abstract

Bordetella organisms are responsible for whooping cough, which is an extremely contagious respiratory illness with substantial morbidity in infants. It is also considered one of the 10 predominant reasons for childhood decease globally, particularly before vaccination was available. Congenital nephrotic syndrome (CNS) presents within the first 3 months of life. It is classified as primary or as secondary to other etiologies, such as infections, drug reactions, toxins, mercury exposure, diabetes mellitus and autoimmune diseases. This article describes the rare presentation of CNS as an outcome to Bordetella infection. That is treating pertussis resulted in CNS to resolve, so it was classified as secondary. This case is the first documented in Syria and the second worldwide.

INTRODUCTION

Humans are the reservoir for Bordetella coccobacilli, which are the causative of whooping cough or pertussis. It is very contagious, basically by aerosolized droplets [1].

Major symptoms in infants are apnea, cyanosis and paroxysmal cough. It could also be complicated by pneumonia, pulmonary hypertension, seizures and encephalopathy [2].

Nephrotic syndrome (NS) is a constellation of clinical findings. It comprises proteinuria, hypoalbuminemia, hyperlipidemia and edema [3]. When presenting in the first 3 months of life, it is considered as congenital [1].

Primary NS has a poorer prognosis compared to secondary NS, as it is usually incurable [4].

In this article, we introduce the incidence of congenital nephrotic syndrome (CNS) as a secondary outcome to Bordetella infection.

CASE REPORT

A male newborn aged 25 days was hospitalized after evaluation in the ER at Aleppo University Hospital (AUH) because of choking, consecutive forceful coughing and posttussive emesis (Day 0). He had been delivered by C-section in 37th week of gestation. His birthweight was 2500 g. Mother's obstetric history included (Gravida 2 Para 1) and that she had encountered premature rupture of membranes. She reported a history of repeated bouts of rapid, spasmodic, intermittent coughs followed by hurried, deep inspirations both she and her daughter had experienced ~3 weeks before delivery. The neonate had been admitted in a newborn intensive care unit for 7 days because of transient tachypnea of newborn and jaundice. He also had an upper respiratory infection on Day (−4) and had been treated with unknown antibiotic. He was then referred to AUH because of not improving and developing leukocytosis with elevated

Received: September 9, 2019. **Revised:** January 16, 2020. **Accepted:** January 22, 2020

C-reactive protein (CRP) level, which were proved by preadmission testing.

Assessment of vital signs on Day (0) revealed blood pressure (BP) 95/50 mmHg, heart rate (HR) 130/min, respiratory rate (RR) 56/min and body temperature of 36.1°C. His weight was 2.5 kg, his head circumference was 34 cm and his height was 45 cm. We reported moderate respiratory distress and fine rales on physical examination, so we did a chest X-ray. Bilateral perihilar shaggy infiltrates obscuring the heart borders were identified. Occasional cough and cyanotic spells with apnea were also documented. Lab works showed a leukemoid reaction of 57.800/µl white blood cells, lymphocyte 57.4%, hemoglobin 10.7 g/dl, hematocrit 31.1%, platelets 355.000/mm^3 and CRP level of 109 mg/dl. The patient was started on Cefotaxime (50 mg/kg IV tid) for the acute inflammatory state, in addition to empiric treatment with Clarithromycin (7.5 mg/kg IV bid) for atypical pneumonia secondary to suspected Bordetella infection.

The follow-up on day (+3) included blood work (leukocytes 77.300/µl, lymphocytes 61.5%, hemoglobin 7.1 g/dl, hematocrit 19.8% and platelets 411.000/mm^3) and physical examination, which revealed a temperature of 38°C and BP 100/60 mmHg. Significant weight gain was noticed (2.5 → 2.86) kg, with development of severe pitting edema in lower extremities and periorbital region. Therefore, we did a urinalysis (Protein +++, PH 5, Specific gravity 1020), biochemical testing (Na 125 mEq-L, serum albumin 2.2 g/dl, total protein 3.7 g/dl, urea 46 mg/dl, creatinine 0.51 mg/dl and triglycerides 86 mg/dl), an echocardiography (a 3 mm-ASD, which is considered normal, with otherwise no abnormalities), liver enzymes (SGPT 25 U/L and SGOT 40 U/L) and renal ultrasonography (slightly echogenic kidneys). Because of these results, the patient was diagnosed as CNS. Syphilis and TORCH panel were done in the context of differential diagnosis and they were negative.

CNS was determined as secondary to Bordetella infection. The patient was treated with Captopril (0.5 mg/kg IV when needed), Spironolactone (2 mg/kg IV bid) and Furosemide (0.5 mg/kg IV bid). He developed oliguria, hypertension and edema. Blood and albumin transfusions were also needed.

By the Day (+12), CRP level fell to 1.8, whereas hypertension and edema were totally diminished by the Day (+24). The pneumonia was also cured. His laboratory results and urinary output were almost normal hereafter.

Eventually, the patient was discharged on day (+26) with Spironolactone (2 mg/kg bid) and supplementary vitamins.

DISCUSSION

Whooping cough includes three classic stages: catarrhal, paroxysmal and convalescent. These stages are usually unclear in infants younger than 3 months of age [4].

Pertussis is mainly transmitted by aerosolized droplets [2]. In our case, the infant's mother and sister were apparently the sources of infection. Mother did not receive any prior immunization against Bordetella.

Infants experience severe cough, which leads to hypoxia and cyanosis. They could also suffer from feeding difficulties. Respiratory distress and pneumonia are also common complications [1].

Leukocytosis is a characteristic finding in pertussis, which happens usually on the behalf of lymphocytes in ~25% of infants infected with Bordetella and aged <6 months [4]. It is also considered an indicator of the disease's severity and may be associated with fatal outcome [5]. In our case, the peak reached was 87.470/µl with lymphocytes' percentage 61.5%. Fortunately, the prompt presumptive treatment for pertussis succeeded in decreasing this number and saved the patient's life.

Diagnosing pertussis depends on clinical presentation and features. Polymerase chain reaction and serology are not frequently available, yet considered further diagnostic tests [6]. They were not also affordable by the family of our patient. Meanwhile, they are limited in our city Aleppo.

CNS is defined as NS starting within the first 3 months after birth. It is categorized as primary or as secondary to a number of etiologies.

Secondary CNS results from systemic diseases such as in utero infections (cytomegalovirus, toxoplasmosis, syphilis, hepatitis B and C and HIV), rubella, malaria, drug reactions, toxins, infantile systemic lupus erythematosus or mercury exposure [4]. These diseases affect the glomerular capillaries creating an immune response as a part of a systemic reaction or by immediately damaging the renal tissue [7].

In our case, the infant showed signs of CNS: proteinuria, hypoalbuminemia, hyperlipidemia and edema. We defined the case as secondary CNS for the absence of early-onset NS, the normal appearance of the placenta, the negative familial history for renal diseases and the occurrence of proteinuria right after the infection.

Curing secondary NS implies treating the causative condition [4]. The improvement of CNS after treating Bordetella infection in our patient supported this approach.

The infant showed significant physical growth during follow-up.

ACKNOWLEDGMENT

We are greatly thankful to Miss Nour Ajam for her valuable help in the revision process of our article.

CONFLICT OF INTEREST

None to disclose.

FUNDING

We have no source of funding to disclose.

ETHICAL APPROVAL

The research meets ethical guidelines and adheres to the local legal requirements.

CONSENT

Agreement by Patient's parents was obtained prior to submission.

GURANTOR

Dr. Mohamad Bassel Mouti.

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