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RESEARCH ARTICLE

CASE REPORT: NEPHROTIC SYNDROME IN AN INFLUENZA B POSITIVE 35 DAYS OLD BOY, CLINICAL PRESENTATION AND TREATMENT

^{1,*}Raymonda Chahrour, M.D., ¹Pielly Hanna, M.D., ¹Farah Beih, M.D., ²Kamal Kanso, M.D. and ³Abir Gaith, M.D.

¹Pediatrics rotating resident at Sacré Coeur Hospital, affiliated with the Lebanese University- Faculty of Medical Sciences

²Pediatric intensive care unit at Sacré Coeur Hospital

³Pediatric nephrology department at Sacré Coeur Hospital

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ABSTRACT

Influenza infection is responsible for a wide range of respiratory illnesses (1), and complicated by otitis media, lower respiratory tract infections mainly (2). Nephrotic syndrome rarely occurs following influenza infection (2). In fact, till date, only 2 cases were reported in the literature (4)(5). We report here a third case of nephrotic syndrome secondary to influenza B infection.

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INTRODUCTION

Influenza viruses are members of Orthomyxoviridae family and are large single-stranded RNA viruses divided into 3 types: A, B and C. Seasonal epidemics are caused mainly by influenza A and B viruses (primary human pathogens) whereas type C is responsible for the sporadic cases of mild upper respiratory tract illnesses (Centers for Disease Control and Prevention, 2019). Influenza infection is generally self-limited in healthy children. However, it may be complicated by otitis media in the first place, followed by pneumonia and less frequently encephalitis, myelitis (Healthline Editorial Team, 2019). As for renal system involvement, only 2 cases were reported in pediatric age till the date of writing of this case (Wenderfer, 2015; Ferrara, 2012; Kim Sae Yoon et al., 2010).

CASE REPORT

A 35 days old baby, presenting to emergency department for one episode of fever (38.5), grunting, along with hypoactivity and decreased PO intake.

*Corresponding author: Raymonda Chahrour, M.D., Pediatrics rotating resident at Sacré Coeur Hospital, affiliated with the Lebanese.

No associated respiratory symptoms (cough or rhinorrhea), no gastrointestinal symptoms (no vomiting nor diarrhea). Neonatal history: the infant was born at 36 weeks of gestation (late preterm), by normal vaginal delivery, for a G2P2 healthy mother, well followed, uncomplicated pregnancy (no diabetes mellitus or hypertension, no infections). Group B streptococcus and TORCH status undetermined. He was admitted to the neonatal intensive care unit (ICU) for hypoglycemia. The admission was complicated by early onset neonatal sepsis for which he received a full course (14 days) of meropenem (blood culture grew E.coli.ESBL). The baby is breast and formula fed, on vitamin D supplements (400 IU). Upon arrival to the ER, the baby was hemodynamically stable, except for a temperature of 38.2. He was mottled, sleepy, had a non-bulging fontanelle, with normal cardiopulmonary auscultation and a soft, non-distended abdomen. Blood tests showed a white count of 3630, neutrophils 38% (ANC: 1380), mild thrombocytopenia (platelets of 120000), positive CRP (3.9mg/dl), normal electrolytes. Respiratory syncytial virus (RSV) test was negative. Chest XR, urine and CSF analysis results were unremarkable. After taking pancreatic cultures, patient was started on IV Cefotaxime. Throughout the day, the patient got better and was tolerating PO feeding.

The next day, high grade fever was noted again, and all of a sudden he became hypoactive, cyanotic, mildly edematous and desaturating. He was transferred urgently to pediatric intensive care unit (PICU), intubated, labs repeated including cultures, and influenza test was taken. Antibiotics were switched to Meropenem and Amikin. Labs were significant for thrombocytopenia (74000), anemia (Hb:7.7) and hypoalbuminemia (1.4). The transaminases were within normal limits, chest x-ray remained unchanged, and influenza B test turned out to be positive. Oseltamivir was added. During his stay in PICU, edema increased and was generalized, urine output decreased to less than 0.5cc/kg/hour, BP readings were 120/60 mmHg and 130/80 mmHg, associated with proteinuria (500mg/dL) and hypoalbuminemia (Albumin level was 2 post transfusion). Nephrologist was consulted and considered acute glomerulonephritis secondary to viral infection and congenital nephrotic syndrome as differential diagnosis.

Spot urine for protein to creatinine ratio was high > 2 (91/8.7 mg/dL=10.7), high triglycerides (195mg/dl), high cholesterol (185mg/dl) levels. EBV and TORCH serologies (Toxoplasmosis, Rubella, CMV, Herpes simplex, Parvovirus, Syphilis), immune profile (IGG, IGA, IGM) were negative, TSH was normal. Moderate intraperitoneal fluid was noted on abdominal ultrasound.

Treatment and prognosis: Albumin transfusion was given for 48hrs (last albumin level:3.2) and furosemide 1mg/kg/dose every 6 hours then tapered. The baby improved after treatment. He became normotensive, non-edematous, with normal urine output and proteinuria decreased to 100mg/dl upon discharge home. He was then followed one week after discharge in clinic by nephrologist with marked improvement.

DISCUSSION

This is a case of 35 days old boy admitted for hypoactivity and low grade fever; found to have influenza B positive infection, complicated by nephrotic range proteinuria. The latter treated by albumin transfusion and furosemide. As a review of literature, only 2 cases of nephrotic syndrome following H1N1 influenza were reported in pediatric age group (Ferrara, 2012; Kim Sae Yoon, 2010).

This case is the third one reporting renal complications following influenza infection and the first to include type B virus. In fact, 3 possible mechanisms contribute to viral - induced glomerulopathies (Wenderfer, 2015).

Viral particles (5-300nm diameter) get trapped in glomerular filtration apparatus leading to immune complex formation.

Possible autoimmune reaction against cross-reactive glomerular cell epitopes as influenza virus acts as an antigenic stimulus to immune system.

Kidney damage secondary to influenza virus induced muscle breakdown (You Jihye, 2017).

Conclusion

Glomerular diseases can develop following viral infections such as influenza viruses (Wenderfer, 2013). So, searching for viral etiologies, especially in younger patients and when the disease has atypical course, should be considered. Treatment of the causative agent can lead to a permanent and complete remission. But in case of persistence of nephrotic range proteinuria for more than 6 months, completing the work up with a biopsy should be considered.

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