Congenital nephrotic syndrome secondary to pertussis

Síndrome nefrótico congénito secundario a la tos ferina

Dear Editor,

Nephrotic syndrome shows itself as massive proteinuria, hypoalbuminemia, edema and hypercholesterolemia. In the first year of life, nephrotic syndrome is a rare but serious disease. The physio-pathology of congenital nephrotic syndrome (CNS), especially in the first 3 months of life, is different from other nephrotic syndromes of childhood. CNS may develop as a result of primary or secondary causes. NPHS1 or NPHS2 genes are detected in primary CNS, while secondary CNS often presents with intrauterine infections, drug reactions, and infantile systemic lupus erythematosus. The diagnosis of CNS should be based on various criteria such as clinical presentation, family history, laboratory findings, and genetic testing. Although primary CNS progresses to end-stage renal failure despite various treatments, the treatment of secondary CNS is more pleasing.[1]

Pertussis is an acute and contagious infectious disease characterized by overlapping and spasmodic coughing attacks. Pertussis can be seen in all age groups.[2] CNS secondary to pertussis is very rare in the literature.[3,4] In this study, we present a case with CNS that secondary to the pertussis in infant and improved with antibiotherapy.

A 45-day-old male applied to hospital with complaints of fever and recurrent coughing attacks. He was born at 3120 g with cesarean section after 39 weeks of pregnancy. It was learned from history that his mother had persistent cough attacks before one month ago. Physical examination revealed arterial tension was 92/51 mmHg, body weight 4.8 kg (25–50 percentile), height 53 cm (25th percentile), body temperature 36.2 °C, oxygen saturation 80%, pulse 160/min and respiration 66/min. There were intercostal retractions and rales, liver and spleen were palpable.

The patient's white blood cell was 56,000/mm^3 (64% polymorphonuclear leukocyte, 28% lymphocytes, 8% monocytes). Blood gases were normal. Chest X-ray examination revealed pneumonic infiltration. The patient was given 100 mg/kg ceftriaxone IV and 15 mg/kg clarithromycin IV. *Bordetella pertussis* PCR was detected as positive in nasopharynx swabs.

On the 5th day of follow-up, edema was detected on the eyelids. In laboratory tests, albumin was reported as 2.8 g/dl (*N*: 2.5–4.4), total protein 4.3 g/dl (*N*: 4.6–7.4) and triglyceride 150 mg/dl (0–150). In complete urinalysis pH was reported as 8, protein 2+. Protein/creatinine in spot urine was 3.6 ($N < 0.7$). Urine culture was negative. Renal ultrasonography revealed no pathology. Anti HCV(−), Anti HIV(−), HBsAg(−), Toxo IgM(−), CMV IgM(−), Rubella IgM(−), and VDRL(−) were detected. Genetic analysis revealed no mutation in NPHS1 and NPHS2 genes. Cough attacks significantly reduced, and edema and rales improved, and the antibiotics were discontinued on the 10th day. On discharge total protein was normal, protein/creatinine in spot urine was 1.6. At the age of 6 months development of the patient was normal and protein/creatinine was 0.5 in spot urine.

Massive proteinuria is caused from mutations in genes encoding structural or regulatory proteins of the renal filtration barrier in the glomerular capillary wall. Proteinuria is caused by the loss of size and load selectivity provided by this barrier.[5] CNS is often caused by primary causes, the most common cause being mutations in the NPHS1 and NPHS2 genes, respectively. Renal biopsy cannot explain the etiology of the disease, and patients should be examined for these two mutations especially for definite diagnosis.[6] In our study, no mutation was detected in NPHS1 and NPHS2 genes.

Secondary CNS is associated with many diseases such as cytomegalovirus infection, toxoplasmosis, congenital rubella, hepatitis B and human immunodeficiency virus infection. This type of CNS develops due to the triggering of nephropathy either directly or through immunomimetic mechanisms.[3] These diseases, which can be recycled and treatable, should be investigated in all cases thought.[7] One of the factors of secondary CNS is pertussis infection, which is rarely reported in the literature.[3,4]

Coughing adolescents and adults are the most important source of pertussis in childhood pertussis. In infants, the concentrations of transplacental pertussis antibodies decrease with a half-life of about 6 weeks and in 2–6 months of age the antibody against *B. pertussis* becomes undetectable.[8] Based on the anamnesis, the mother of our case was accepted as the source of pertussis.

Pertussis is diagnosed by culture or polymerase chain reaction (PCR). Culture is gold standard in diagnosis, but this method may give false negative results in the late period of infection, in vaccinated individuals and in antibiotic therapy areas. In recent years PCR has become more popular.[2]

The treatment of secondary CNS is directed at the underlying cause, and nephropathy is improved with the treatment directed against the agent. Macrolides are used in the treatment of pertussis. Macrolides have been shown to reduce the symptoms, especially when they are given in the early stages of the disease, to prevent contamination by

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increasing the elimination of nasopharynx[9] and to correct nephropathy.[4]

As a result, pertussis is a rare cause of secondary CNS, therefore edema or proteinuria, which can be detected in infants who are followed due to pertussis, should be stimulating for CNS.

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Conflict of interest

The authors declare that there is no conflict of interest.

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Renal damage secondary to checkpoint inhibitors☆

Fracaso renal agudo asociado a inhibidores check-point

Dear Editor,

The treatment of renal carcinoma (CR) has been revolutionised thanks to the use of antiproliferative and immunomodulatory drugs. Sunitinib (SUN) is a tyrosine kinase (TK) inhibitor with antiproliferative and anti-angiogenic effect.[1] Nivolumab (NIV) is a human monoclonal antibody, a checkpoint inhibitor, which prevents the cancer cells from evading the immune system, enhancing the patient's immune response.[2] Both drugs have been shown to increase RCC survival, but have also been linked to toxicity in different organs. We describe here the case of a patient who developed renal damage associated with the use of SUN and NIV.

This was a 70-year-old male with a pacemaker due to Mobitz AV block, hypertension (HTN) and chronic kidney disease (baseline creatinine 1.4–1.5 mg/dl) due to loss of nephron mass after right nephrectomy due to RC (pT3b Nx Mx) in 2014.

After recurrence of the cancer, he received first-line treatment with SUN, which was discontinued because toxicity, including renal toxicity, leaving serum creatinine (sCr) levels of 1.4–1.8 mg/dl. Six months later, the cancer progressed,

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