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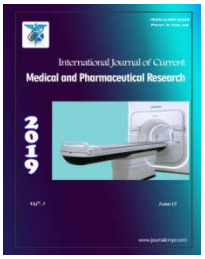

Case Report

HYPERTENSIVE ENCEPHALOPATHY AS A COMPLICATION OF ACUTE DIFFUSE GLOMERULONEPHRITIS POST-STESTRECOCCAL: CASE REPORT IN PEDIATRICS

Marcella Lopes Abitbol[1], Fabrícia Louzada Depizzol Vito Sobrinho[2], Rafaela Monique Mendonça de Barros[1], Juliana Vieira de Oliveira Brasil[1], Laís Viana Lopes Sato[1], Mário Jorge dos Santos Noel Filho[1], Renata da Silva Almeida[3] and Romulo Novellino[4]

[1]Resident Physician of Pediatrics at the Foundation of Tropical Medicine Doctor Heitor Vieira Dourado (FMT/HVD) - Manaus/AM
[2]Pediatric Physician and Master's Degree in Child and Adolescent Health from the Faculty of Medicine of Ribeirão Preto (FMRP/USP) - Manaus/AM
[3]Resident Physician of Pediatrics at the State University of Amazonas (UEA) - Manaus/AM
[4]Academic of the Medical Course of Nilton Lins University (UNL) - Manaus /AM

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ABSTRACT

Acute diffuse glomerulonephritis has as a most frequent cause in pediatrics, infection by hemolytic β Streptococcus of Group A. Of patients with post-streptococcal glomerulonephritis, 95% evolve well with or without treatment and few present serious complications during the condition. The objective of this report is to provide a pediatric case of acute diffuse glomerulonephritis post infectious by Streptococcus pyogenes that evolved with complications of hypertensive encephalopathy. It is of paramount importance to pay attention to post-streptococcal glomerulonephrites, because even though most evolve satisfactorily, there is the possibility of more aggravating complications related to the pressure uncontrollable ness of patients.

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INTRODUCTION

Acute diffuse glomerulonephritis (ADGN), also called post-streptococcal glomerulonephritis, is one of the most prevalent causes of glomerulonephritis in the world. This condition predominantly affects developed countries (97%), reaching an annual incidence of 9.5 to 28.5 cases per 100,000 inhabitants per year. Preschool children are the most affected age group, and it is rare to find cases between children under 2 years of age and in adults over 40 years of age. There is also a slight predominance inmales.[1.4]

ADGN is generally preceded by a superior airway infection or after a pyodermitis[2,3]. The classically clinical symptoms acute nephritis, attending with edema, arterial hypertension, macroscopic hematuria. Treatment is symptomatic, and prognosis is favorable in the most majority ofcases.[2.3]The most frequent causein pediatrics, is a β-hemolytic *Streptococcus* infection of Lancefield Group A.[5]

The deposit of streptococcal components in glomerulus stimulates the production of antibodies by the host, forming in situ immune complexes that alter the permeability of the basal membrane, allowing subsequent deposition of preformed immunocomplexes. [6]

CASE REPORT

Pediatric patient KSM, female, 5 years old, natural and coming from Manaus (Brazil), of low socioeconomic level, with history of diffuse impetiginous lesions in the legs. The post 5 days after initial condition, child evolved with bilateral periorbital edema, in the legs, abdominal distension, fever and macroscopic hematuria. She had no significant pathological history.

On the same day of clinical worsening, she goes to the municipal emergency hospital for medical care. At the first medical care, child was in regular general condition, hypocorated, with general edema, hypertensive for the age group (PA 160 x 98 mmHg), oliguric and with fever measured at 38.6° Celsius. Admission tests showed hematuria, pyuria, increased creatinine, leukocytosis and elevated

Corresponding author:* **Marcella Lopes Abitbol
Resident Physician of Pediatrics at the Foundation of Tropical Medicine Doctor Heitor Vieira Dourado (FMT/HVD) - Manaus/AM

Creactive protein. Reduced serum complement dosage (C3 and C4).

Due to the severity of the condition, child was hospitalized, with restriction of sodium and water (400ml/m²), initiated loop diuretics (furosemide 2mg/kg/day), control of blood pressure levels with Anlodipine at a dose of 0.06 mg/kg/day and intravenous antibiotic therapy (Ceftriaxone 100mg/kg/day). She remained hospitalized for 5 days, with improved edema and blood pressure levels. She was discharged from hospital with antihypertensive and oral antibiotic with new doctor appointment after 7 days.

Thepost 5 days of hospital discharge the child was entered in the emergency room, edemaciated, with lowering of level of consciousness (Glasgow Coma Scale <8 points), presenting emergency hypertensive crisis and with history of successive seizures at home initiated with 4 hours of admission. He required mechanical ventilation, anticonvulsants for seizure refractory and hydralazine in continuous infusion (0.2mg/kg/dose). Laboratory tests reconfirmed renal dysfunction and treatment for GNDA was again started. Patient evolved with improvement of the condition and referred to the pediatric nephrology care for follow-up.

DISCUSSION

In the case described, the patient presented acute diffuse glomerulonephritis secondary to pyoderma itis. The kidney symptoms manifested about 5 days after the onset of the condition, incubation period considered short for the installation of glomerular lesion.[7] As a late complication of acute diffuse glomerulonephritis, a child evolved with hypertensive encephalopathy and seizures, because he had undergone irregular treatment of antihypertensive drugs and control of sodium intake at home.

Laboratory tests showed progressive worsening of renal function, presence of hematuria and piuria, leukocytosis, anemia and increased evidence of inflammatory activity. These findings are compatible with the acute period of the disease.[8]Treatment performed with restriction of water and sodium supply in the oral, loop diuretic, antihypertensive and intravenous antibiotic for 5 days, allowed rapid and satisfactory recovery of the condition.

Due to clinical improvement and no hypertensive episodes in the 48 hours, children were discharged from the hospital with guidance of maintaining restriction in the diet, the use of antihypertensive, oral antibiotic to terminate treatment and with medical reassessment scheduled for 7 days after release. However, due to the correct non-performance of medications at home and by non-restriction of water mainly, the child evolved with the situation of hypertensive encephalopathy.

When it occurs elevation of the blood pressure levels above the99th or 20-30mmHg percentile above 95, severe hypertensive or hypertensive crisis is defined, with a risk iminente of complications. Hypertensive encephalopathy occurs when blood pressure exceeds the limits of control of blood flow cerebral (vasoconstriction of cerebral arteries and arterioles). When these limits are exceeded, blood vessels suddenly dilate, causing liquid leakage to the perivascular space, determining the appearance of cerebral edema and its manifests. At the same time, there is loss of capillary permeability, with worsening of the condition.[9]

The prognosis of hypertensive crisis in children depends on the early recognition of the crisis, the underlying cause, the systemic repercussions and the appropriate treatment through the use of antihypertensive drugs. The presence of severe encephalopathy or of chronic disease (renal insufficiency) has a determining factor in prognosis, but the speed of recognition and control of the crisis favorably influences the evolution of the ground. [10]

CONCLUSION

Acute diffuse glomerulonephritis is a common pathology in pediatrics, has benign evolution and simple treatment of clinical support, with water and sodium restriction, blood pressure control, and current causal infection, ensures complete recovery in 95% of patients.[11] Hypertensive encephalopathy is not a common complication of glomerulonephritis, but in the child is the main hypertensive emergency, when there is increased blood pressure above the 99th percentile and is usually associated with renal dysfunction and hypervolemia. The patient should be guided well on the maintenance of treatment and the need for specialized clinical follow-up.

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