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CLINICAL CASE

Posterior reversible encephalopathy syndrome (PRES) and hemolytic anemia: two severe and unusual complications of acute post-streptococcal glomerulonephritis

Síndrome de encefalopatía posterior reversible (PRES) y anemia hemolítica: dos complicaciones graves e inhabituales de glomerulonefritis aguda post estreptocócica

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What do we know about the subject matter of this study?

Acute post-streptococcal glomerulonephritis (APSGN) is the main cause of nephritic syndrome, and one of its manifestations is hypertension. PRES is a clinical-radiological diagnosis, and its treatment is based primarily on controlling the underlying disease and stabilizing blood pressure.

What does this study contribute to what is already known?

We report the association of two rare and serious complications of APSGN, such as PRES, and autoimmune hemolytic anemia. Both should be considered in the differential diagnosis of cases with an atypical course and neurological or hematological complications. In this context, ongoing evaluation of neurological symptoms together with monitoring of erythrocyte counts is important.

Abstract

The most frequent cause of nephritic syndrome in the pediatric population is acute post-infectious glomerulonephritis (PIGN). A rare complication is posterior reversible encephalopathy syndrome (PRES), characterized by subcortical vasogenic cerebral edema associated with variable neurological symptoms. The development of autoimmune hemolytic anemia is an atypical clinical presentation. **Objective**: To report the coexistence of two unusual and serious extrarenal complications of PIGN and to discuss potential mechanisms involved in their development. **Clinical Case**: A 4-year-old male patient, with a 5-day history of hematuria and edema, headache, nausea, and vomiting. He was admitted in convulsive status and hypertensive crisis, laboratory showed C3 hypocomplementemia and high titers of Antistreptolysin O, which was interpreted as PIGN. Due to the presence of encephalopathy, PRES secondary to hypertensive emergency was suspected, which was confirmed by brain MRI. He also presented autoimmune hemolytic anemia, with hemoglobin up to 5 g/dL. The

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treatment was based on antihypertensive therapy, neuroprotective measures, and steroid treatment. He was discharged 31 days after hospitalization and remained asymptomatic 6 months after discharge. **Conclusions**: There must be a high suspicion index of PRES before the appearance of nonspecific neurological symptoms during the evolution of a PIGN. The existing anemia on occasion can be autoimmune.

Introduction

The most frequent cause of nephritic syndrome in the pediatric population is acute post-streptococcal glomerulonephritis (APSGN). This pathology can cause severe acute complications with associated morbidity and mortality, such as hypertensive emergency (21.5%), congestive heart failure (12.3%), encephalopathy (4.6%), and retinopathy (1.5%)[1].

Posterior reversible encephalopathy syndrome (PRES) is a clinical-radiological entity characterized by reversible subcortical vasogenic edema in patients with acute neurological symptoms such as epileptic seizures, encephalopathy, headache, and visual disturbances[2]. The imaging study usually shows symmetric bilateral vasogenic edema that mainly affects areas of the posterior cerebral circulation, predominantly subcortical white matter, usually in the parieto-occipital region. In children, it develops in the context of secondary hypertension (HT)[3,4]. In PRES, abrupt changes in blood pressure and/or the direct effect of cytokines result in endothelial dysfunction causing vasogenic edema at the level of the central nervous system.

The occurrence of severe anemia is uncommon in patients with APSGN and remains unexplained in some cases[1].

The objective of this report is to describe the coexistence of two unusual and severe complications, PRES and autoimmune hemolytic anemia (AIHA), in a patient with APSGN and to review the possible pathophysiological mechanisms involved.

Clinical Case

A 4-year-old male patient with history of nasal impetigo two weeks before admission (treated with topical mupirocin and oral cefadroxil; dosage, duration, and adherence to treatment unknown), with no other morbid history, who presented macroscopic glomerular hematuria associated with a 5-day history of lower extremity edema, in addition to headache, nausea, and vomiting in the last 20 hours before consultation. He went to the emergency department (ED) in status epilepticus after 20 minutes of generalized tonic-clonic seizures.

On admission to the ED, the patient had no fever, with unmeasurable blood pressure, a quantitative altered state of consciousness associated with generalized hypertonia, and bi-palpebral and pretibial edema. Endotracheal intubation was decided, and phenobarbital (10 mg/kg) was administered to manage the status epilepticus.

At the time of physical examination in the intensive care unit (ICU), blood pressure 134/94 mmHg (MAP 110 mmHg) was observed (p95 for patient 108/66 mmHg; p95+12 120/78 mmHg).

Initial laboratory parameters included complete urinalysis which showed hematuria > 100 erythrocytes per high-power field (HPF), proteinuria 3+, and leukocyturia 10-25 per HPF; serum creatinine 0.3 mg/dL, anemia with hematocrit (Hto) 21%, hemoglobin (Hb) 7 g/dL, normal mean corpuscular volume (MCV) and mean corpuscular hemoglobin concentration (MCHC), leukocytosis 23,900 cells/mm^3, thrombocytosis 756,000/mm^3, no increase in acute phase reactants levels, hypocomplementemia with complement level C3 at 25 mg/dL (normal value, NV: 80-150 mg/dL), and normal C4 level. The rapid antigen detection test for group A beta-hemolytic Streptococci (*Streptococcus pyogenes*) in the pharynx was positive as well as the Antistreptolysin O (ASO) test (table 1). A brain CT scan without contrast showed no acute changes. Renal ultrasound showed bilateral nephromegaly with increased cortical echogenicity and decreased corticomedullary differentiation.

The patient was diagnosed with nephritic syndrome due to APSGN complicated with hypertensive emergency and status epilepticus.

Within the first 24 hours of his stay in the ICU, the patient required mechanical ventilation (MV) and anticonvulsant therapy with phenobarbital. He progressed without epileptic seizures, with a normal electroencephalogram (EEG) (the day after his admission), and normal cerebrospinal fluid analysis. Antibiotic therapy was started for eradication of *Streptococcus pyogenes* with cefotaxime and diuretic therapy with furosemide.

The following day, he progressed with deterioration of renal function presenting creatinine increase up to 0.99 mg/dL, hypertension, and 24-hour proteinuria of 36.6 mg/m^2/h, without oliguria. He started antihy-

Table 1. Temporal evolution of laboratory parameters in a patient with post-streptococcal glomerulonephritis complicated by posterior reversible encephalopathy syndrome and hemolytic anemia

Parameter	Normal Value	Admisión	Day 2	Day 5	Day 15
Hemoglobin (g/dL)	10.5-13.5	7.1	6.6	8.9	11.1
Platelet count (x10^3/mm^3)	140-400	683	531	519	400
RST	Negativo	Positivo	-	-	-
ASO	< 200 U/ml	Positivo	-	-	-
Haptoglobin (mg/dL)	41-165	-	-	151.8	-
LDH (U/L)	195-349	322	231	-	197
Bilirrubin (mg/dL)	0.3-1.2	< 0.15	0.19	-	0.18
Urea nitrogen (mg/dL)	9-22	10	18	12	6
Creatinin (mg/dL)	0.36-0.63	0.3	0.65	0.34	0.2
PCI (mg/mg)	< 0.2	6.7	2	1.3	0.23
C3 (mg/dL)	80-150	25	-	46	152
C4 (mg/dL)	12-36	31.2	-	24	41.8
Direct coombs	Negativo	-	Positivo	-	Positivo
ANA	Negativo	-	1/160	-	-

RST: Rapid streptococcus test; ASO: antiestreptolisin O; g/dL: grams per decilitre; mg/dL: milligrams per deciliter; U/ml: units per mililiter; LDH: lactate dehydrogenase; PCI: Protein/Creatin urine index; mg/mg: milligrams per miligrams; C3: complement C3; C4: complement C4; ANA: antinuclear antibodies.

pertensive treatment with amlodipine and intravenous labetalol, with good initial control.

Given a favorable progression, extubation was performed after 48 hours, being well tolerated from a ventilatory point of view. However, 24 hours after extubation, the patient's state of consciousness worsened, with eye opening and limb withdrawal reflex only in response to painful stimuli and poor verbal response (Glasgow Coma Scale score 8); and blood pressure >p95+12 despite receiving continuous infusion of labetalol (up to 3 mg/kg/h), amlodipine (10mg/day), and furosemide. It was necessary to start MV again and sodium nitroprusside infusion (up to 3 mcg/kg/min), in order to gradually reduce blood pressure (25% daily) to prevent secondary neurological damage. Given the presence of acute neurological symptoms associated with hypertension in a patient with glomerulonephritis, the diagnosis of PRES was suspected and confirmed by brain MRI (day 5), which showed increased subcortical signal (figure 1) in the bilateral and symmetrical occipital region, without restricted diffusion, compatible with vasogenic edema. The ophthalmological evaluation was normal, and a new EEG revealed occasional episodes of generalized voltage depression.

During the following days, blood pressure continued to be unstable, without achieving control of blood pressure levels, therefore, enalapril was added to the treatment. Finally, after 10 days with a slow pharmacological withdrawal, blood pressure normalization was achieved. The control MRI (day 12) showed regression of the previously described findings (figure 1). Successful extubation was achieved after 5 days.

During his stay in the ICU, the hemoglobin level decreased to 5 g/dL, with normal MCV and MCHC, and no thrombocytopenia, so hemolytic anemia was suspected due to positive direct Coombs test and hemoglobinuria. He required red blood cell transfusions twice. It was decided to initiate steroid therapy with methylprednisolone (1 mg/kg/d) for 72 hours. The stool culture was negative, as was the urinary antigen test for *Streptococcus pneumoniae*. Serology for Epstein-Barr virus and Parvovirus B19, Extractable Nuclear Antigen (ENA) profile, antineutrophil cytoplasm antibodies (ANCA), anti-DNA antibodies, anti-B-2 glycoprotein 1 antibodies, anti-cardiolipin antibodies, and lupus anticoagulant were negative. All cultures were negative (blood, urine, endotracheal aspirate, and pharyngeal). Only antinuclear antibodies (ANA) were positive (1/160).

The patient progressed with improved blood pressure, increased complement level, and urine test without proteinuria or hematuria. The direct Coombs

test remained positive on the 9th day of hospitalization.

On day 31, the patient was discharged with normal blood pressure, without anemia, with preserved renal function, without proteinuria or hematuria, with normalization of C3 levels, and asymptomatic regarding neurological aspects. He was discharged with pharmacological therapy with prednisone, amlodipine, enalapril, and folic acid. The patient had no recurrence and remained asymptomatic at 6 months post-discharge.

Figure 2 summarizes the clinical course of the patient.

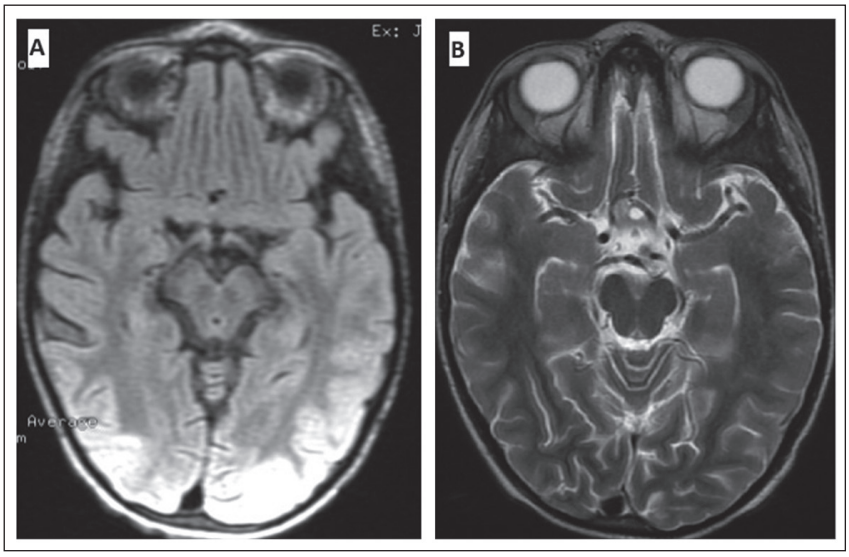

Figure 1. Initial and follow-up brain magnetic resonance imaging (MRI). Image **(A)** corresponds to the patient's first MRI, where an axial projection of the FLAIR sequence was evident, showing hyperintensity of the subcortical signal in the occipital region. Control image one week after the previous one **(B)** shows axial section of T2 sequence, where complete resolution of the hyperintensities is observed. FLAIR: *Fluid attenuated inversion recovery.*

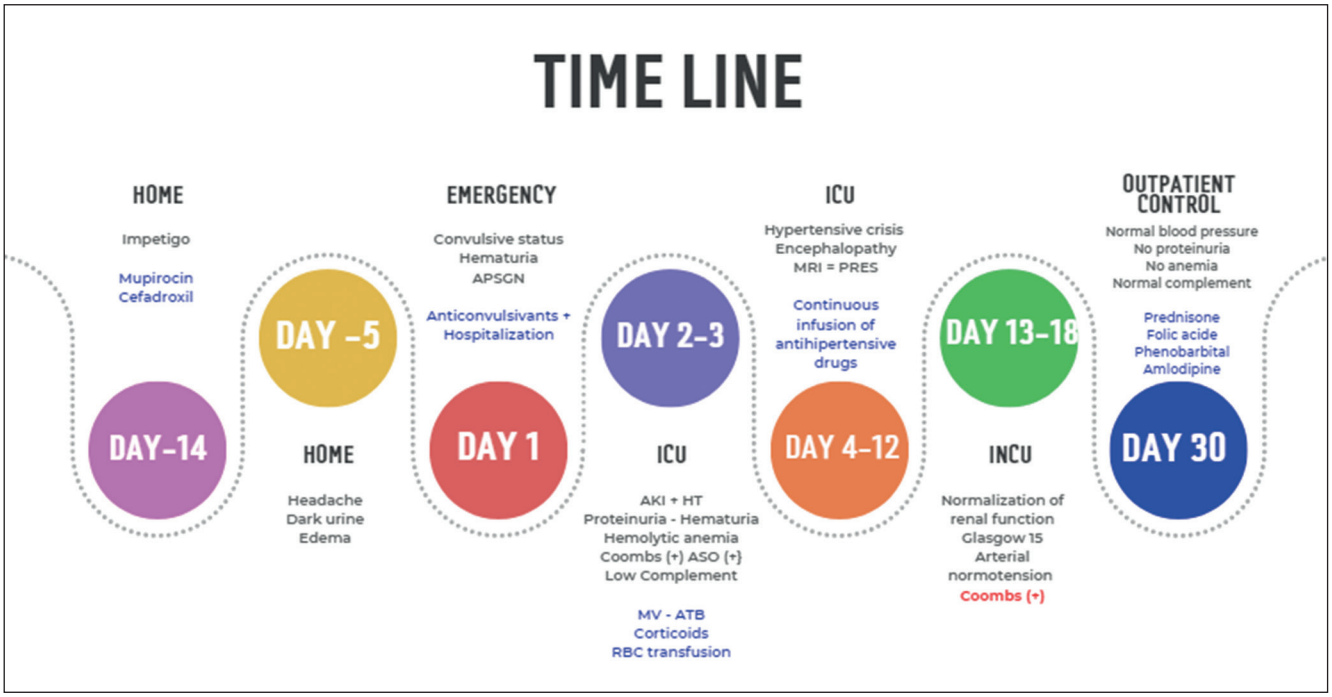

Figure 2. Timeline with diagnoses, most relevant clinical and laboratory data of a patient with post-streptococcal glomerulonephritis complicated by posterior reversible encephalopathy syndrome and hemolytic anemia. ASPGN: Acute post-streptococcal glomerulonephritis, ICU: Intensive care unit, AKI: Acute kidney injury, HT: Arterial Hypertension, MV: Mechanical ventilation, RBC: Red blood cells, ATB: Antibiotics, ASO: Anti streptolisin O, MRI: Magnetic Resonance Imaging, PRES: Posterior reversible encephalopathy syndrome, INCU: Intermediate care unit.

Discussion

The reported case presents unusual characteristics to observe in a frequent pathology such as APSGN in pediatrics. These were the development of two serious complications such as PRES and AIHA. Both required treatment and the first one was potentially associated with significant morbidity.

Few studies have been published on PRES in children[5]. The approximate incidence in the general pediatric population is 0.04%[6], however, in pediatric patients admitted to the ICU, this is estimated at 0.4%[5]. In addition, the incidence of PRES in patients with kidney disease varies between 4-9%[6].

In a recent systematic review in a pediatric population, where patients with PRES of oncologic cause were excluded, the main etiology was nephrological[7]. Within kidney diseases, both glomerulonephritis[7] and hemolytic uremic syndrome[8] have been described. Notably, the association between PRES and AIHA[7] has also been reported.

The most recognized risk factors for the development of PRES are the presence of HT and thrombotic microangiopathy (TMA) with non-immune hemolytic anemia[9].

HT is common in pediatric PRES, being the main associated factor in 80% of cases of APSGN[10,11].

PRES in the pediatric population may present some differences with adults in terms of etiology, precipitating factors, and clinical manifestations[7]. The main symptoms are seizures (90%) and encephalopathy, which were present in our case. Others frequently found are headache, visual disturbances, and focal neurological deficits[12].

The imaging study is characterized by the presence of bilateral vasogenic edema affecting the subcortical white matter, with paramedian preservation of the occipital lobe[13]. The localized involvement pattern observed in the superior frontal sulcus and the parieto-occipital sulcus have similar frequencies (39%)[11]. Atypical lesions on MRI are found in 61-84% of children with PRES, compared to 10-58% of adult patients[12,14]. PRES can lead to intracranial hemorrhage, both intraparenchymal and subarachnoid. This complication is seen in 8-28% of pediatric PRES[11].

Its pathophysiology is controversial, as both vasomotor and immunological mechanisms have been described[15] (figure 3). The first mechanism involves the direct effect of HT on cerebral vasculature, surpassing the limits of cerebrovascular autoregulation, leading to increased blood flow and the development of vasogenic edema[16].

In most of the reported PRES cases associated with APSGN, the patients had HT, which supports this hypothesis. Sympathetic innervation, which serves a protective role in this context, is lower in the vertebrobasilar system, which could explain the predominantly posterior localization of the observed brain lesions[17].

Regarding the proposed immunological mechanism, interactions between streptococcus antigens and antibodies activate the immune system, including the complement system and cytokine/chemokine production, which increases the expression of vascular endothelial growth factor (VEGF), a key regulator of vascular permeability[18]. This represents the initial step in its pathogenesis.

PRES usually has a good prognosis and both symptoms and anatomical alterations are reversible if it is diagnosed and treated promptly, with recovery within days to weeks[11,19]. A complete or near-complete imaging resolution of cerebral edema has been described in 90% of patients after one month of progression[12]. However, if diagnosis or treatment is delayed, relevant neurological sequelae may occur (10-20%)[20] or even death, especially in concomitance with intracranial hemorrhage or cerebral infarction[21], with a 3.2% case fatality rate[22]. A recent publication indicated that underlying end-stage renal disease, the presence of status epilepticus, the existence of atypical lesions on MRI, and increased inflammatory biomarkers could be prognostic factors for PRES in children[23].

Another particular feature of the case presented was the development of AIHA. The most frequent hematologic complication in patients with APSGN is anemia, however, this is generally mild and is mainly due to hemodilution[24]. The existence of AIHA has been infrequently reported. Greenbaum et al.[25] reported three cases of APSGN with concomitant hemolytic anemia. All of them presented positive Coombs test; however, two of them interestingly progressed with bilirubin and LDH in the normal range as observed in our patient, which suggests that hemolysis could be subacute and eventually not perceived in the absence of a high index of suspicion.

Cachat et al.[26] reported the case of a patient who presented severe AIHA during anuric AKI secondary to APSGN, observing a good response to steroids. The role of complement activation caused by APSGN, a mechanism also involved in the pathophysiology of PRES, in the induction or severity of hemolysis was postulated.

Recently, Gong et al.[27] attributed the presence of anticardiolipin antibodies a role in the development of AIHA in a patient with APSGN. It has been demonstrated that *Streptococcus* infection is the cause of the generation of antiphospholipid antibodies[27], as well as that they play a direct role in the pathogenesis of hemolytic anemia. In the case reported, testing for antiphospholipid antibodies was negative.

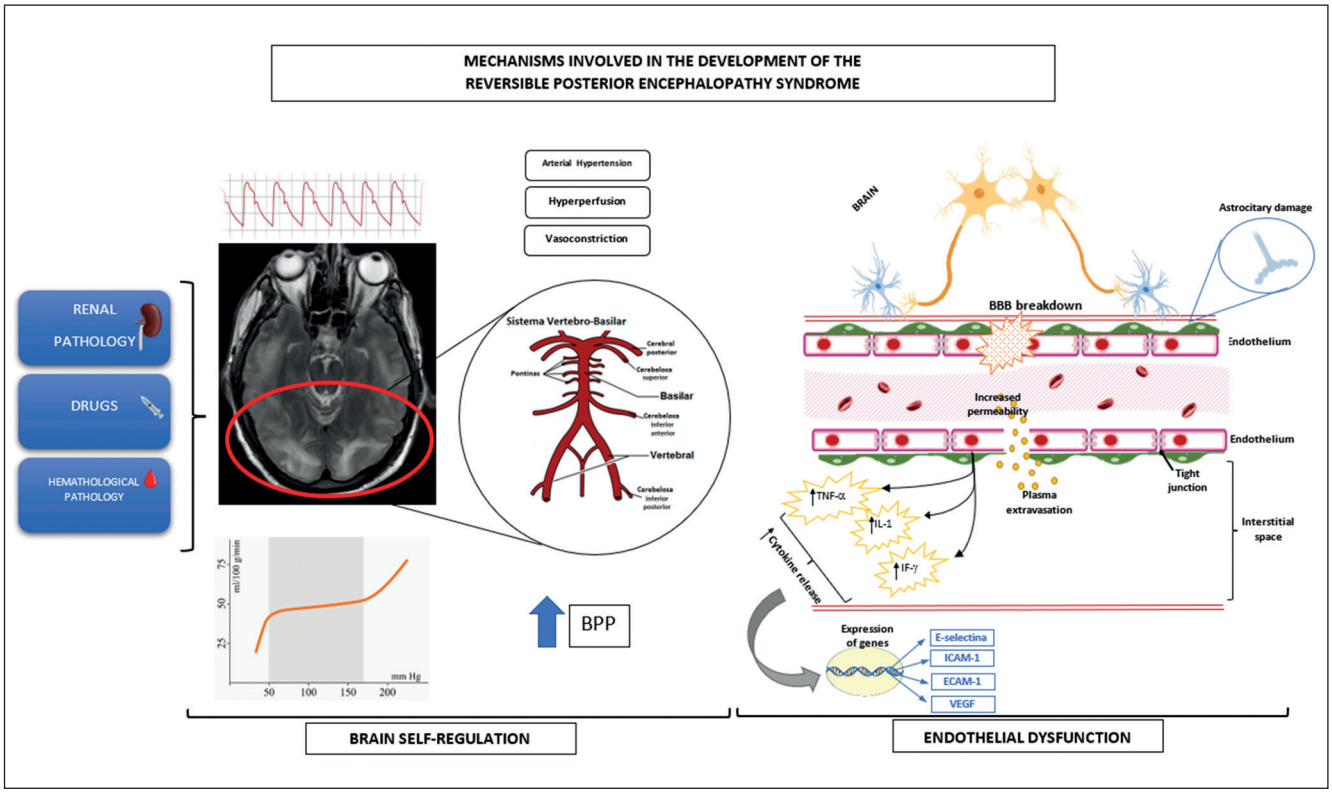

Figure 3. Pathophysiological mechanisms involved in the development of posterior reversible encephalopathy syndrome. Proposed hypotheses. The first one postulates that arterial hypertension would cause arterial vasoconstriction, causing ischemia and cytotoxic edema. The second considers that an alteration of cerebral vascular autoregulation would occur secondary to uncontrolled arterial hypertension, which in turn would cause vasodilation, endothelial dysfunction and vasogenic edema. The dilation of the arterioles and the opening of the endothelial tight junctions will determine the dysfunction of the blood-brain barrier, causing interstitial transudation, edema, hypoperfusion and vasoconstriction with cytotoxic damage. Likewise, endothelial activation and dysfunction entails reactive changes with increased expression of vascular endothelial growth factor and T lymphocytes in the intra- and perivascular space with activation of the immune system, including complement, leukocyte infiltration and production of cytokines/chemokines, stimulating angiogenesis and increasing vascular permeability, contributing to vasogenic edema. Classically, hydrostatic edema affects the white matter of the posterior portions of both cerebral hemispheres. It is usually a relatively symmetrical pattern with preservation of the calcarine and paramedian cortex of the occipital lobes. However, other structures such as the brain stem, cerebellum, and the frontal and temporal lobes may also be affected. BPP, Brain perfusion pressure; BBB, blood brain barrier; TNF-α, alfa tumor necrosis factor; IL-1, interleukin-1; IF-γ, gamma interferon; ICAM-1, intercellular adhesion molecule 1; ECAM-1, epithelial cell adhesion moleculesl 1, VEGF: Vascular Endothelial Growth Factor.

The pathogenic mechanism of AIHA in patients with APSGN is unknown. A cross-reaction between antibodies induced by streptococcus infection and red blood cells has been proposed, however, this has not been confirmed[28].

The streptolysin O toxin can interact with the lipid layer of red blood cells and insert itself into the membrane as a large, polymerized molecule. Likewise, the membrane-bound toxin can form immune complexes by capturing human immunoglobulin G (IgG). These complexes formed by the binding of streptolysin O antigen and IgG antibodies are powerful activators of the classical complement pathway[28]. In addition, NAD-glycohydrolase (NADase) may also play a role[29]. Therefore, both enzymes may contributed to the hemolysis in our patient.

It is known that infections caused by *Streptococcus pneumoniae*, a neuraminidase producer, are a cause of TMA. This enzyme transiently exposes T antigens on the erythrocyte surface, platelets, and endothelial cells. Naturally, IgM antibodies exist in the infected host, which bind to the exposed T antigen, resulting in endothelial damage, and initiating the cascade of events leading to the development of TMA[30]. However, although less known, *Streptococcus pyogenes* is also a neuraminidase producer, capable of causing the same pathogenic mechanism[31].

Finally, both PRES and the occurrence of AIHA share similar immunologic pathogenic mechanisms[28] that could explain the concurrence of these unusual complications in a patient with APSGN.

Conclusions

In this report, we present a four-year-old boy with an uncommon association of two serious complications such as PRES and AIHA. Both should be suspected in cases of APSGN of unusual course with neurologic or hematologic complications.

When severe anemia appears in a child with APSGN, the possible existence of an acute hemolytic phenomenon of immunologic cause should be evaluated.

Responsabilidades Éticas

Protección de personas y animales: Los autores declaran que los procedimientos seguidos se conformaron a las normas éticas del comité de experimentación humana responsable y de acuerdo con la Asociación Médica Mundial y la Declaración de Helsinki.

Confidencialidad de los datos: Los autores declaran que han seguido los protocolos de su centro de trabajo sobre la publicación de datos de pacientes.

Derecho a la privacidad y consentimiento informado: Los autores han obtenido el consentimiento informado de los pacientes y/o sujetos referidos en el artículo. Este documento obra en poder del autor de correspondencia.

Conflicto de intereses

Los autores declaran no tener conflicto de intereses.

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