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Research

Posterior Reversible Encephalopathy Syndrome (PRES): Experience in the National Institute of Pediatrics

Darmendra Singh Urias[1*], Samuel Zaltzman[1], Gilberto Gómez Garza[1] and Chiharu Murata[1]

[1]National Institute of Pediatrics, Mexico

Abstract

Background

Posterior reversible encephalopathy syndrome (PRES) is characterized by neurological disorders such as headache, nausea, vomiting, visual disturbances, altered consciousness, paresthesia or seizures. The diagnosis is clinical and radiological because a predominantly symmetric vasogenic edema in the parietal and occipital lobes is observed in the cranial CT or MRI scans. The risk factors for PRES include, among others, hypertension, use of immunosuppressant drugs, steroids or chemotherapy. The treatment is aimed to reduce or remove the risk factors for PRES. The objective of this study is determining the risk factors for PRES, as well as their clinical and radiological features and their evolution.

Method

A review was performed on the clinical and radiographic records of those patients with a probable diagnosis of PRES at the National Institute of Pediatrics in Mexico City from January 2006 through January 2013. The neurological symptoms were categorized as mild, moderate or severe according with the clinical features of each patient. The radiological findings were classified into three grades, according to the extension of vasogenic edema in the imaging study. Blood pressure, the use of steroids, immunosuppressant drugs or chemotherapy was also analyzed.

Results

Twenty-two patients were confirmed with PRES. All were classified with severe neurological symptoms. Seven patients received immunosuppressive therapy, (6 with calcineurin inhibitors), 12 with steroids, and 5 also received chemotherapy. Twenty-one patients had vasogenic edema beyond the white matter.

Conclusion

An MRI scan should be performed in all patients with severe hypertension and other risk factor for PRES, which would help to initiate an early treatment to reduce the risk of complications.

Keywords: Reversible Encephalopathy; Hypertensive Encephalopathy; Vasogenic Edema

Introduction

The posterior reversible encephalopathy syndrome was first described in 1996 by Hincheyet al., who observed in 15 patients the presence of headaches, altered consciousness, seizures and visual loss which were reversible in short time. A neuroimaging study was practiced in all the patients where characteristics of edema predominantly on the posterior region of the brain were observed, reason why it was named reversible posterior leukoencephalopathy syndrome [1, 2].

From the various reports of reversible posterior leukoencephalopathy syndrome have been suggested different nomenclatures as reversible occipital parietal encephalopathy, PRES and hypertensive encephalopathy [2]. PRES is an uncommon condition characterized by neurological disorders such as headaches, visual disturbances, alterations of consciousness, paresthesia or seizures and vasogenic edema in the brain parenchyma with parieto-occipital dominance.

The parenchyma abnormalities can be seen both in a Cranial Computed Tomography (Cranial CT) and magnetic resonance imaging (MRI) scan; therefore the diagnosis is clinical and radiologic[1- 4]. Although the radiological changes are most common in the white matter, they can also found within the anterior brain, anterior and posterior cortex, brain stem, cerebellum, or in the spinal cord; the alterations may be symmetrical or asymmetrical. The lesion (vasogenic edema) in the MRI appears as a hypo intense image in T1 sequence, while in T2, FLAIR and proton density it is hyper intense [4- 10].

There are multiples factors may cause PRES, among them are hypertension (AHT), immunosuppressant drugs, high doses of steroids and chemotherapy [2,4,6-13]. The treatment is aimed to reduce or remove the factors that could have triggered the syndrome, start with antihypertensive drugs and anticonvulsants for patients with seizures. The goal of therapy is reducing the risk of irreversible damage.

***Corresponding author:** Darmendra Singh Urias, Pediatric Nephrologist, National Institute of Pediatrics, mexico, E-mail: darmendra_s@hotmail.com

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The purpose of the study is determine the risk factors found in patients with PRES at the National Institute of Pediatrics in Mexico City from January 2006 to January 2013; in addition to describe the clinical and radiological characteristics, and their evolution.

Materials and Methods

Case studies. Clinical and radiographic records (cranial MRI) of patients with a suggestive diagnosis of PRES from departments of Nephrology, Immunology, Hematology, Oncology and Gastroenterology at the National Institute of Pediatrics in Mexico registered between January 2006 and January 2013 were reviewed. 29 patients with diagnosis of PRES were found in the institute database, but only 22 patients met both clinical and radiologic features for the diagnosis. Age of patients, sex, signs and symptoms such as hypertension, headaches, nausea, vomiting, visual disturbances, paresthesia, altered consciousness, and seizures were recorded. As well, risk factors for PRES such as immunosuppressant or steroid drugs and history of chemotherapy were researched. The clinical picture in each patient was graded according to their severity, similar to what Yamada and Ueda [13] did in their study. In that study, they graded the neurological signs and symptoms, as mild presentation, when there were headaches, nausea, vomiting or tremor; moderate, in the presence of visual disturbances, and severe when were mental disorders, cerebellar symptoms, seizures, stupor or coma. Blood pressure (BP) was assessed and graded since the beginning of the symptoms until they were controlled. At the same time BP was classified as severe hypertension when was above 99th percentile for blood pressure according to height. All patients underwent cranial MRI with the techniques of T1, T2 / FLAIR, diffusion and ADC. Radiographic findings were graded as grade 1: subtle changes; grade 2: large abnormal areas; grade 3: an entire region affected, as it was described by Yamada and Ueda[13]in the same study (Figures 1, 2 and 3). A follow-up MRI could not be performed on all patients for different reasons. In those patients whom had the follow-up MRI was performed in different times of each patient. The image changes were recorded to assess the reversibility of lesion, as well the presence of sequelae.

Results

Twenty-nine patients were registered with diagnosis of PRES in the Radiology and Imaging Department, whom underwent to a cranial MRI to confirm the diagnosis. Only 22 of the 29 patients with radiology studies met the criteria for the syndrome. The remaining 7 patients, in three of them the radiologic records were not found, and the medical record in one. The last three patients did not met the criteria (Fig.4).

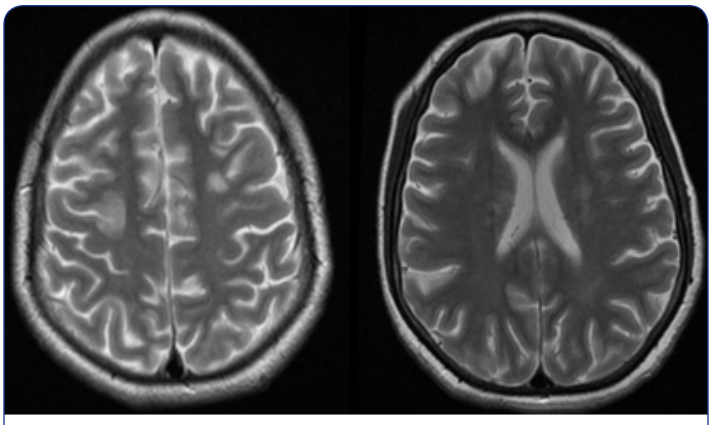


Fig 1: MRI classified as grade 1 (mild vasogenic edema)

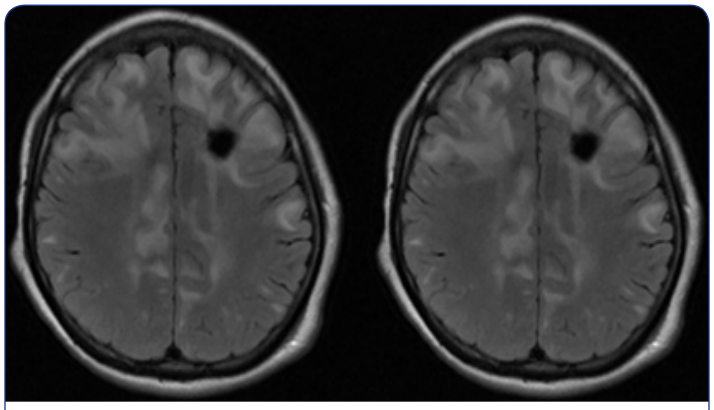


Fig 2: MRI classified as grade 2 (moderate vasogenic edema) because it does not compromise the entire region of a lobe.

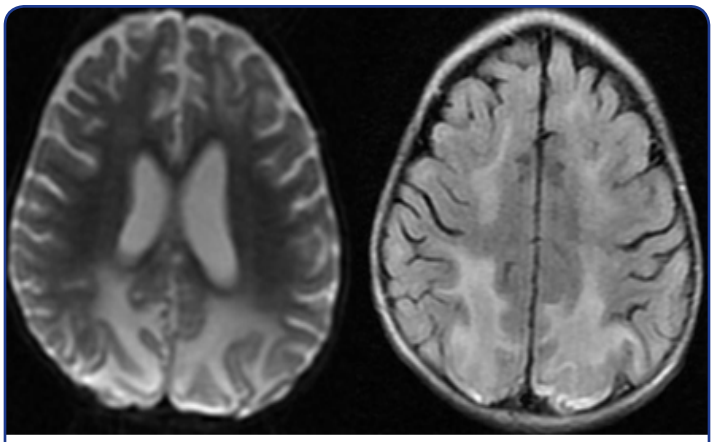
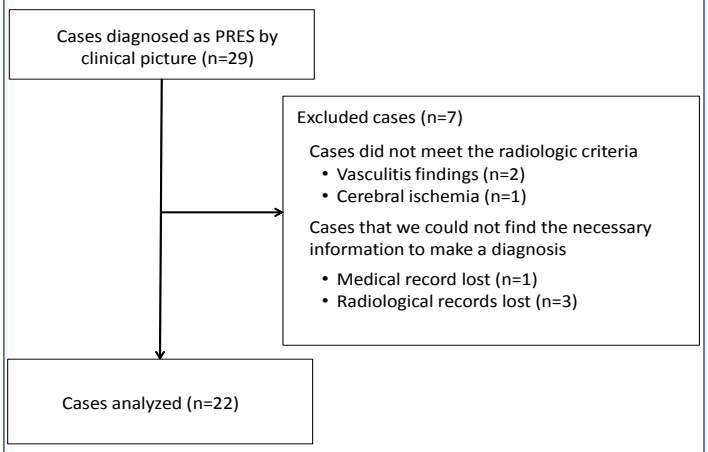


Fig 3: MRI classified as grade 3 (severe vasogenic edema) because we can observe the entire occipital lobe affected.

Fig. 4. Flowchart of the application of inclusion and exclusion criteria



Clinical and Demographic Characteristics

Clinical and demographic characteristics of patients are shown in the table 1. The different diseases in each patient at the time of diagnosis of PRES were: acute lymphoblastic leukemia in 6 cases, 4 with lupus nephropathy, 2 post kidney transplant, 2 more had hemolytic uremic syndrome, and one case of each of the following diseases: thymic carcinoma, cavernomatous malformation of the portal vein, acute lymphoblastic lymphoma, Henosh-Shonlein purpura, autoimmune thrombocytopenic purpura, liver sarcoma, hemophagocytic syndrome and testicular tumor. Therefore, from the 22 patients confirmed with PRES, the 64% were patients from the Nephrology and Oncology departments, 32% each one. The remaining patients are,18%,from hematology department; 14%, immunology department; and 4%, from the gastroenterology department.The age of the patients was from 1 year with 10 months to 16 years with 7 months old (mean 8.5 years old).

Table 1: Demographic information, risk factors and clinical findings of patients with PRES

Case	Age	Gen	MDx.	BP mm/Hg	Use IST	Type IST	Use SD	Type SD	Clinical findings
1	2ª1ᵐ	F	HUS*a	140/100	No		No		GTC Sz*q, irritability Somnolence
2	1ª10ᵐ	F	HUS	135/85	No		Yes	MEP*m	Irritability, somnolence
3	16ª8ᵐ	F	PRTx*b	166/95	Yes	CSA*j AZA*k	Yes	PRED*n	Headache, GTCSz
4	5ª	M	PRTx	147/92	Yes	CSA	Yes	PRED	Tremor, GTC Sz somnolence
5	7ª9ᵐ	M	LN*c	142/86	No		Yes	PRED	GTC Sz
6	16ª7ᵐ	F	LN	194/129	No		Yes	MEP	GTC Sz
7	2ª4ᵐ	F	CMPV*d	112/63	No		No		Tremor, CP Sz*r
8	7ª	M	ITP*e	130/100	Yes	CSA	Yes	MEP	headache, CP Sz
9	7ª1ᵐ	M	LN	120/70	Yes	CSA MMF*l	Yes	MEP	CPSz
10	14ª11ᵐ	F	LN	195/140	Yes	CSA	Yes	PRED	Headache, nausea, vomiting Somnolence, GTCSz
11	3ª2ᵐ	F	HLH*f	100/90	Yes	CSA	No		CPSz
12	8ª8ᵐ	M	ALL*g	129/85	No		No		GTCSz
13	7ª	M	ALL	120/90	No		No		GTCSz
14 m	6ª	M	ALL	113/85	No		No		CPSz, lethargy,somnolence
15	13ª9ᵐ	M	Thymic carcinoma	130/80	No		Yes	DXM*o PRED	GTCSz
16	7ª5ᵐ	M	LBL*h	130/70	No		Yes	HC*p	CP Sz, lethargy
17	11ª8ᵐ	F	ALL	110/70	No		No		Nausea, vomiting, CP Sz
18	7ª9ᵐ	M	ALL	150/100	No		No		Headache, somnolence
19	6ª5ᵐ	M	Liversarcoma	140/79	No		No		GTCSz
20	15ª6ᵐ	M	Testicular tumor	120/80	No		No		Vomiting, GTC Sz, amaurosis somnolence, aggressiveness
21	12ª5ᵐ	F	ALL	174/100	No		Yes	HC	CPSz, somnolence
22	7ª5ᵐ	M	HSP*i	160/114	Yes	MMF	Yes	PRED	Irritabilidad, Desorientación

Gen: gender, MDx: main diagnosis, BP: Blood Pressure, IST: Immunosuppressant, SD:Steroids.

*a Hemolytic Uremic Syndrome, *bPost renal transplant, *c Lupus Nephritis, *dCavernomatous malformation of the Portal vein, *e Autoimmune Thrombocytopenic Purpura, *f HemophagocyticLymphohistiocytosis, *g Acute Lymphoblastic Leukemia, *h Lynphoblastic Lymphoma, *i Henoch-Schonlein Purpura, *j Ciclosporina A, *k Azathioprine, *lMycophenolato, *m Methylprednisolone, *n Prednisone, *oDexamethasona, *p Hydrocortisone, *qGeneralized tonic clonic seizures, *rcomplex partial seizures.

Neurological Symptoms and Neuroimaging Studies

The most common clinical presentation were seizures in the 86% of the cases, followed by disturbances of consciousness 45%, headache 18%, nausea/vomiting 13%, tremor 9% and visual disturbances 4% (Table 1). Generalized seizures were observed in 11 patients while 8 had partial seizures. There was not recurrence of seizures in any patient. The hyper intense image in the sequence of T2 / FLAIR was observed in 20 patients in the parietal and occipital lobes with bilateral dominance. In the other 2 patients, one had the image only in the parietal lobes while in the other one, the image was in the occipital lobes and in one of the temporal lobes. Other areas affected were: the frontal and temporal lobes in 14 and 8 patients, respectively. The cerebral ganglia, cerebellum and brain stem were less involved. The lesion reversibility could be assessed only in 11 patients (50%), because not all the patients underwent to a follow-up MRI. From the 11 patients only 4 had reversibility of the vasogenic edema and without any sequelae. In the other 7 patients, 4 did not have any more edema, but they already had another type of parenchyma damage; Encephalomalacia in 2 patients, one with gliosis, and another one with multiple lesion as laminar necrosis, petechial hemorrhages, and gliosis. The last 3 patients still had the edema, but 2 of them together with another lesion as laminar necrosis and encephalomalacia.

Therefore, we can say that only 4 cases of PRES were reversible according to the assessment of the follow-up MRI (Table 2).

Predisposing Factors, Hypertension Severity, Clinical and Radiographic Picture

None of the patients had history of arterial hypertension (AHT). From the 22 patients diagnosed with PRES, 20 presented high blood pressure of which 80% was classified as severe. (Table 3) The median of systolic and diastolic pressure were: 132.5 and 88 mmHg.

On the other hand, 7patients were taking immunosuppressant drugs (31%), six with calcineurin inhibitors (cyclosporine A) and one with mycophenolatemofetil. Two of the patients with cyclosporine were also taking a second immunosuppressant, azathioprine and mycophenolate, respectively. Regarding the use of steroids, twelve patients (54.5%) were taking them when they developed the syndrome, 4 with pulses of methylprednisolone.The remaining patients with steroids were using prednisone, dexamethasone or hydrocortisone at a rate of 1mg/kg/day. Finally, only five patients (22.7%) received chemotherapy before the syndrome. Two with methotrexate; one, vincristine/L-aspartate;other vincristine/ cisplatin; and the last one, etoposide/cisplatin/ifosfamida.

All the clinical pictures were graded as severe, because all the patients had alterations of consciousness.

The severity of the lesion in the MRI was classified as second grade in 11 patients (50%); 8 (36.3%) grade 1; and 3 (13.6%) grade 3. There was no statistically significant association between the variables of

Tabla 2: Radiological findings on MRI from our patients

Caso	DPIMR	RFIMR	RFIMR	Grade of lesion	DPFMR	Reversibilidad FMR	Secuela FMR
1	12 d*a	PL*b bilateral OL*c bilateral	FL bilateral TL unilateral	3°	90 d	No	Encephalomalacia
2	20 d	PL bilateral OL bilateral	FL unilateral	2°	38 d	Yes	-
3	1 d	PL bilateral OL bilateral	FL bilateral	1°	No FMR	-	-
4	11 d	PL bilateral OL bilateral FL*d bilateral	TL bilateral BGL*f BSL*g	3°	24 d	No	Encephalomalacia
5	2 d	PL bilateral OL bilateral	TL unilateral	1°	24 d	No	Stillwith Vasogenic edema
6	3 d	PL bilateral OL bilateral FL bilateral	TL bilateral BGL CL*h	2°	23 d	Yes	-
7	7 d	PL bilateral OL bilateral	FL bilateral TL unilateral	2°	No FMR	-	-
8	3 d	PL bilateral OL bilateral		2°	No FMR	-	-

9	2 d	PL bilateral OL bilateral	FL bilateral	2°	No FMR	-	-
10	14 d	PL bilateral OL bilateral		1°	No FMR	-	-
11	4 d	PL bilateral OL bilateral		1°	No FMR	-	-
12	8 d	PL bilateral OL bilateral	FL unilateral	1°	No FMR	-	-
13	2 d	PL bilateral OL bilateral	FL unilateral TL bilateral	1°	17 d	No	Gliosis
14	2 d	PL bilateral		1°	No FMR	-	-
15	0 d	PL bilateral OL bilateral	FL bilateral	2°	17 d	Yes	-
16	1 d	PL bilateral OL bilateral	FL unilateral TL unilateral	3°	28 d	No	Still with Vasogenic edema and Laminar necrosis
17	1 d	PL bilateral OL bilateral	FL bilateral	2°	No FMR	-	-
18	1 d	PL bilateral OL bilateral	CL bilateral	1°	21 d	-	-
19	0 d	OL bilateral TL*e unilateral		2°	121 d	Yes	-
20	12 d	PL bilateral OL bilateral	CL bilateral	2°	13 d	No	Laminar necrosis Petechial Hemorrhages Gliosis
21	8 d	PL bilateral OL bilateral	FL unilateral	2°	22 d	No	Still with vasogenic edema and Encephalomalacia
22	14 d	PL bilateral OL bilateral	FLunilateral	2°	No FMR	-	-

DPIMR: Dayselapsed to perform the initial MR, RFIMR: radiological findings in initial MR, DPFMR: Days elapsed to perform the follow MR, FMR: follow MR. *a days, *b Parietal lesion, *c Occipital lesion, *d Frontal lesion, *e Temporal lesion, *f Basal ganglia lesion, *g Brainstem lesion, *h cerebellum Lesion.

age and sex with the severity of the lesion in the MRI neither the grade of lesion with sequelae. We did not find either association between the number of risk factors in the patients to develop PRES and the grade of lesion or sequelae.

Discussion

Posterior reversible encephalopathy syndrome is not very common. The incidence or prevalence in the pediatric population or adults is unknown. Due to the clinical picture varies, acute or sub acute, with signs and symptoms that go from subtle to severe, it is possible that is misdiagnosed, given that in those cases where only headaches and nausea are present as only symptoms of the clinical picture, a routine MRI scan is probably not done.[5,14]

In 1897, Vazquez and Nobecourt, made the first description of neurological disorders associated with pregnancy, but it wasn't until 1928 when the hypertensive encephalopathy was described by Oppenheimer and Fishberg. Subsequently, Tamaki et al [15-17] described the reduction of cerebral flow, preceded by blood brain barrier dysfunction and the subsequent cerebral edema, as it occurs with the posterior reversible encephalopathy syndrome (PRES). In recent years, there has been controversy over the use of

Tabla 3: Risk factors for PRES. Classification of the hypertension, andclinical and radiological picture

Case	BP Mm/Hg	GradeAHT	IST	Steroid	CRT	Grade ofNSx	Grade of RF
1	140/100	Sev*	-	-	-	Sev	3
2	135/85	Sev	-	MEP*d	-	Sev	2
3	166/95	Sev	CSA*a/AZA*b	PRED*e	-	Sev	1
4	147/92	Sev	CSA	PRED	-	Sev	3
5	142/86	Sev	-	PRED	-	Sev	1
6	194/129	Sev	-	MEP	-	Sev	2
7	112/63	Leve	-	-	-	Sev	2
8	130/100	Sev	CSA	MEP	-	Sev	2
9	120/70	Leve	CSA/MMF*c	MEP	-	Sev	2
10	195/140	Sev	CSA	PRED	-	Sev	1
11	100/90	Sev	CSA	-	-	Sev	1
12	129/85	Sev	-	-	VCR*h / L-asp*i	Sev	1
13	120/90	Sev	-	-	-	Sev	1
14	113/85	Leve	-	-	MTX*j	Sev	1
15	130/80	Leve	-	DXM*f/PRED	-	Sev	2
16	130/70	Sev	-	HC*g	-	Sev	3
17	110/70	-	-	-	-	Sev	2
18	159/100	Sev	-	-	MTX	Sev	1
19	140/79	Sev	-	-	VCR / CPT*k	Sev	2
20	120/80	-	-	-	IFO*l / CPT/ VP-16*m	Sev	2
21	174/100	Sev	-	HC	-	Sev	2
22	160/114	Sev	MMF	PRED	-	Sev	2

BP: Tensión Arterial, AHT: arterial hypertension, IST: Immunosuppressant, CRT: chemotherapy, NSx: Neurological symptoms, RF: Radiological findings.

* Severe, *a Cyclosporine, *b azathioprine, *c MicophenolatMofeti, *d Methylprednisolone, *e Prednisone, *f Dexamethasone, *g Hydrocortisone, *h Vincristine, *i L-aspartate, *j Methotrexate, *k Cisplatin, *l Ifosfamide, *m Etoposide.

the name of hypertensive encephalopathy or posterior reversible leukoencephalopathy for this neurological disorder condition, since the clinical and radiologic manifestations are similar. However, after Hinchey et al, reported other causes that develop the same clinical radiologic symptoms, such as kidney, oncological, hematological, immunological diseases, the use of immunosuppressive drugs, steroids (mainly in high doses), the treatment with chemotherapy, as well as arterial hypertension, that although in most cases is severe, the clinical picture may occur in up to 20% of the cases without hypertension or with mild hypertension; that is the reason why Hinchey decided to name this pathology as posterior reversible leukoencephalopathy and not as hypertensive encephalopathy [1,2]. Casey et al. proposed to modify the name in the year 2000 to posterior reversible encephalopathy (PRES) because they found that it could be presented not only in white matter but also in gray matter.[3]In view of the above described, in this study, mostly, the patients had kidney, oncological, immunological or hematological diseases (just one corresponded to the gastroenterology area due to cavernous transformation of the portal vein). Different hypothesis of physiopathological mechanisms for this syndrome were proposed: the first was linked to arterial hypertension. Currently, the most accepted is that this syndrome is manifested as vasogenic edema more than cytotoxic or that in some cases both problems may exist. In this hypothesis is proposed that the physiopathological mechanisms is produced due to a loss of autoregulation of cerebral blood flow that leads to arterial vasodilation and endothelial dysfunction with blood brain barrier dysfunction and capillary transudation. Plasma and cells are accumulated in the extracellular compartment, mainly in the white matter [4,10,12,18-23]. On the other hand, the physiopathological mechanisms of the posterior reversible encephalopathy syndrome linked with the use of immunosuppressive drugs, mainly calcineurina inhibitors such as cyclosporine, cytotoxic or both, is even more uncertain. However, it has been thought that these drugs may have a direct toxic effect over the vascular endothelium and cause vasospasm, reduction of the tissue perfusion, activation of the coagulation cascade and in

turn extravasations of liquid [4, 5,7,10,19].

Yamada et al [13] investigated the association of the severity of the clinical picture of the PRES and its sequelae with the age and sex of the patient. They observed that the younger ones had greater recurrence of seizures, and that the neurological symptoms were more severe in female patients; however, this data was not statistically significant since the study sample was very small (11 cases). In our study there was a predominance of males of 59% and there was no relation to the severity of the symptoms, since all patients were classified as having severe symptoms. Of the patients younger than 5 years, 3 had grade 3 of the lesion in the MRI, and some type of sequelae in the MRI control, which could suggest that this age group could have a larger cerebral edema, as well as higher risk of sequelae; on the other hand, 2 patients of the same age group had grade 1 and 2 of the lesion, respectively and nosequelae. No relations of the grade of the lesion with sequelae were found, since from the 6 patients with sequelae, 3 had edema grade 3. Of the predisposing factors, almost all of the patients had more than one factor to develop posterior reversible encephalopathy syndrome, and almost all of them presented some kind of disease that has been linked with this syndrome. On the other hand, 90% of the cases had arterial hypertension, from which 80% were severe. Other factors were the use of immunosuppressive drugs in 7 patients, some kind of steroids in 12 patients and chemotherapy background in 5. Only one patient has as sole factor to develop PRES his/her disease which is acute lymphoblastic leukemia that could be linked to immunological alteration. With this we could confirm that this entity is of multifactorial origin, for which the most suitable name is posterior reversible encephalopathy syndrome.

Among the most linked immunosuppressive drugs with the development of posterior reversible encephalopathy syndrome are the calcineurin inhibitors, mainly cyclosporine stand out due to its toxic effect over the endothelium; six of our 7 patients with immunosuppressive received cyclosporine [1, 3,4,8,10]. The most frequent clinical picture presented was seizures, mainly generalized clonic seizures, or it started as complex partial and evolved to generalized, that may be linked mostly with arterial hypertension [2,4,10,23]. From the cases that presented seizures, 58% started as generalized tonic-clonic and the rest as complex partial, even though all evolved to generalized tonic-clonic. The vasogenic cerebral edema observed, through an MRI or a CAT scan, is found more frequently in the posterior region of the hemispheres of the brain, mainly the parieto-occipital and bilateral manner; however, there are cases that also present it in the cerebellum, brain stem, basal ganglia, among other areas. It may also be presented asymmetrically and it's called atypical posterior reversible encephalopathy syndrome [3, 7, 12, 15, 19 22]. From our 22 patients, 18 had vasogenic edema beyond the parieto-occipital region, mostly bilaterally; only 4 had limited edema to the posterior region as the literature describes it with greater frequency. Although all presented early reversibility of the neurological symptoms, upon reviewing the follow-up MRI in the 11 patients who had it, only 4 had reversibility of the vasogenic edema with any sequelae. In the other 7 patients; 4 did not have any more edema, but they already presented other type of parenchymal damage like Encephalomalacia in 2 patients, one more with gliosis, and the last one with multiple lesion as laminar necrosis, petechial hemorrhages, and gliosis.The other 3 patients still had the edema, but 2 of them together with other lesion as laminar necrosis and encephalomalacia, respectively. Therefore, we can say that only 4 cases of PRES were reversible according to the assessment of the follow-up MRI. However, we cannot make any conclusion with these numbers because the population in our study is small, as well as because not all the patients underwent to the follow-up MRI.

Conclusion

PRES is not a different entity to what is known as hypertensive encephalopathy, but rather a pathological entity of multifactorial causes and not only for hypertension. The action to take when we suspect of PRES is to perform early imaging studies in order to rule out cerebral vascular events and confirm the syndrome in presence of vasogenic edema. Once PRES is diagnosed, it is essential assess the adjustment or removing drugs that are related to PRES, in addition to initiate hypertension treatment if there; all with the aim of reducing the risk of irreversible damage.

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