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Case Series

Posterior reversible encephalopathy syndrome and pregnancy: a Moroccan retrospective study of a patient's oligo series

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ABSTRACT

Posterior reversible encephalopathy syndrome (PRES) is an entity combining reversible central nervous system damages with characteristic magnetic resonance imaging (MRI) brain imaging, it can occur in peripartum regardless of any preexisting pathology. PRES in peripartum is a poorly understood phenomenon. Early diagnosis and management are essential to prevent irreversible neurological sequelae. We report 6 cases series of PRES, collected at obstetrics and gynecology department of the military hospital Mohamed V in Rabat, between 2000 and 2019, in order to describe and analyze epidemiological, clinical, paraclinical and therapeutic aspects. Average age was 27 years old. Found symptoms were: high blood pressure, seizures, headaches, visual disturbances, consciousness disorders, vivid osteotendinous reflexes, vertigo, nausea and vomiting. Associated complications were status epilepticus, eclampsia, haemolysis, elevated liver enzymes, low platelet count (HELLP) syndrome and acute lung edema. Radiologically, found lesions are suggestive of PRES. After management, evolution was favorable in all patients.

Keywords: PRES, Pregnancy, Retrospective studies, Morocco

INTRODUCTION

Posterior reversible encephalopathy syndrome (PRES) is a clinical and radiological entity that has been revealed rather recently, it combines a reversible damage of central nervous system with characteristic brain imaging. Physiopathology remains a source of disagreement. It is a unique neurological entity that can occur in peripartum, regardless of any pre-existing pathology. Diagnosis calls for an evocative context, a characteristic symptomatology, a typical MRI appearance with a clinical and radiological reversibility.

CASE SERIES

We report a 6 cases retrospective series of PRES, collected in department of obstetrics and gynecology and managed in collaboration with both departments of intensive care and neurology in the military hospital of instruction Mohamed V Rabat, between 2000 and 2019, In order to describe and analyze epidemiological, clinical, paraclinical, therapeutic and evolutionary aspects of this pathological entity.

Analyzed data were extracted from patients' clinical report. PRES diagnosis is retained in front of a characteristic symptomatology, an evocative context, a typical aspect of MRI with a clinical and radiological reversibility. We eliminated all incomplete reports, patients without a sure PRES diagnosis, and patients lost to follow-up.

All data was collected on operating sheet as follows: 1. Age, 2. Medical history, 3. Gynecological and obstetrical history, 4. Evolution of pregnancy and gestational age, 5. Clinical parameters: Onset of childbirth signs, temperature (°C), Systolic blood pressure (SBP), diastolic blood pressure (DBP) in mm Hg, lower limbs edema, proteinuria, Neurological signs: Consciousness status assessed by Glasgow score, Headache (onset time), Convulsive seizures and/or status epilepticus existence (onset time, and blood pressure levels during), Presence or

not of neurological deficit, Osteotendinous reflexes examination, Visual disturbances existence, 6. Neuro-imaging (Brain (CT) and/ MRI), 7. Biological data, 8. Electroencephalogram, 9. Associated complications, 10. Therapeutic and obstetric care, 11. Immediate evolution and control data and 12. Newborn's state.

Obtained information have been compiled in Table 1.

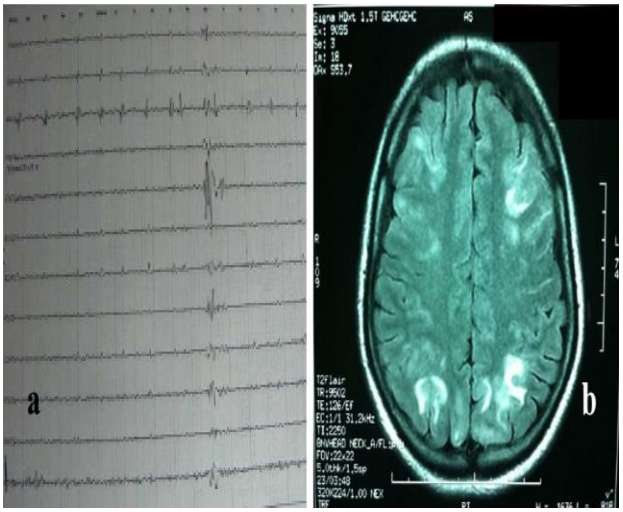

Figure 1 (A and B): Electroencephalogram: a diffuse paroxysmal activity for short time and brain MRI, axial section, FLAIR sequence: hypersignals of the subcortical white matter at the frontal and bilateral parietal areas.

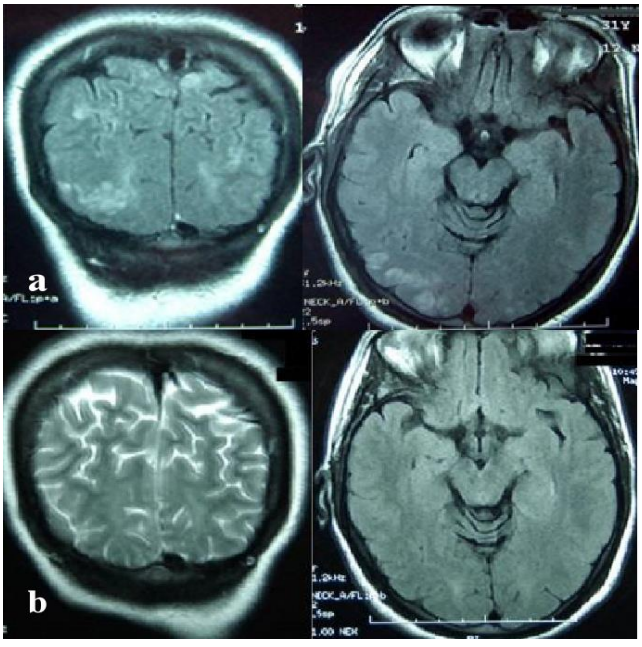

Figure 2 (A and B): Brain magnetic resonance imaging (MRI), axial and coronal section, FLAIR sequence: bilateral and asymmetric cortico-subcortical hypersignals, in parieto-occipital area and the 2 months brain MRI control: complete resolution of the brain damages.

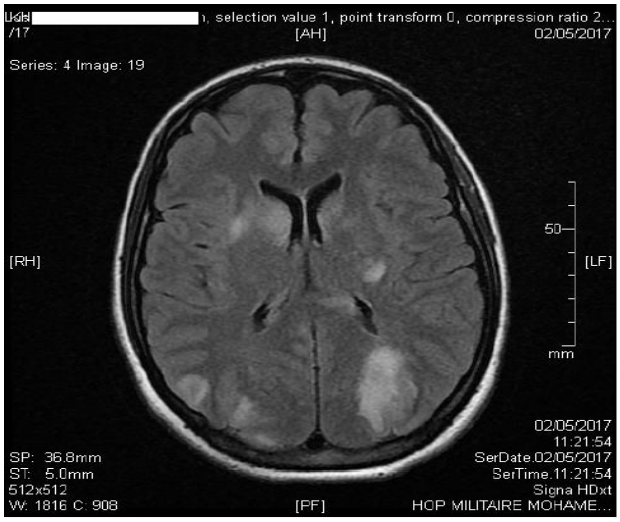

Figure 3: Brain MRI, axial section, FLAIR sequence: Bilateral and asymmetric cortico-subcortical hypersignals, in parietal, capsulo-lenticulars and caudate nuclei heads.

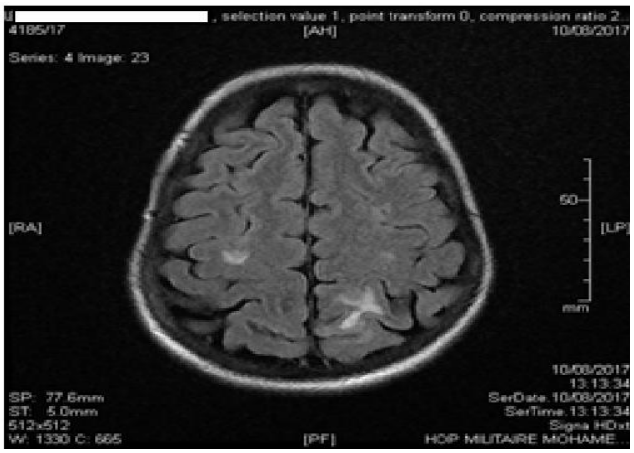

Figure 4: Brain MRI, axial section, FLAIR sequence: hypersignals of subcortical white matter in right frontal and left parietal areas.

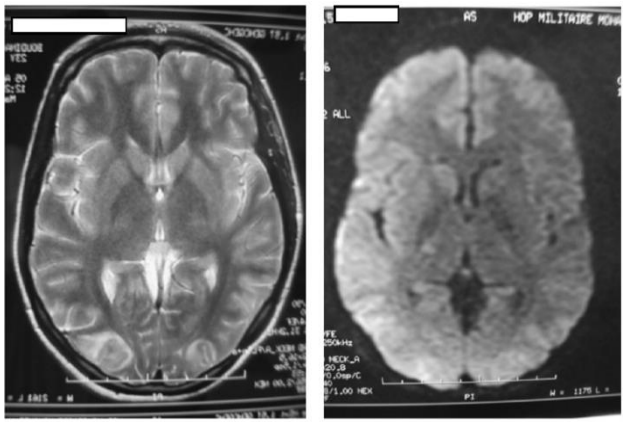

Figure 5: Brain MRI, axial sections, T2 and diffusion sequences: Cortico-subcortical hypersignals at bilateral occipital area, more marked on the right.

Table 1: The clinical, paraclinical, therapeutic and evolutionary aspects in the PRES patients.

Variables	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5	Patient 6
Age (years)	23	31	25	26	28	29
Gestity	Primigest	Third gest	Primi gest	Primi gest	Primi gest	Primi gest
Parity	Primiparous	Multiparous	Nulliparous	Primiparous	Primiparous	Nulliparous
Medical history	None	None	Appendicectomy 8 years ago	None	None	None
Pregnancy term (AW)	38 AW + 2 days	38 AW + 5 days	27 AW	36 AW	37 AW	33 AW
Evolution of pregnancy	Threat premature labor due to urinary infection à 34 AW	Normal	Normal	-Gemelar pregnancy -High BP at 34 AW without proteinuria treated by alpha-methyldopa	Membranes premature rupture at 37 AW	-Contractions at 33 AW -Severe fetal growth restriction + oligo-hydramnios
Proteinuria	Negative	++	+++	+++	Negative	+++
Edema	Discreet lower limbs edema	Discreet lower limbs edema	Discreet lower limbs edema	Lower limbs edema	None	Discreet lower limbs edema
Blood pressure BP (mmHg)	140/85	180/100	167/110	174/100	140/90	180/110
Headaches	Helmet-like headaches	Intense helmet-like headaches	Invalidant helmet-like headaches	Intense headaches	Intense helmet-like headaches	Helmet-like headaches
Headaches onset of the time	Postpartum 7th day	Post-partum 12th hour	27 AW	36 AW	Postpartum 7th day	33 AW
Convulsive seizures	Generalized convulsive seizures	3 tonic-clonic generalized seizures	2 generalized convulsive seizures	Many tonic-clonic generalized seizures	Tonic-clonic generalized seizures	Tonic-clonic generalized seizures followed by convulsive state
Seizures on set time	Postpartum 7th day	Postpartum 12th hour	27 AW (many hours after headaches)	Postpatum 11th hour	Postpartum 7th day	33 AW
Blood pressure during seizures (mmHg)	180/100	140/90	180/110	150/90	230/120	210/110
Other clinical signs	Vertigo Dyspnea	Nil	Bilateral hemianopsia with vivid osteotendinous reflexes, Nausea	Nil	Vertigo	-Tinnitus -Consciousness disorders
Complication	Acute lung edema	-HELLP syndrome -Eclampsia	-Eclampsia	-Eclampsia	Nil	Oligohydramnios -Eclampsia -Convulsive State
Brain CT scan	Normal				Normal	

Continued.

Variables	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5	Patient 6
Brain MRI	Hypersignals of subcortical white matter at frontal and bilateral parietal level. Figure 1 A	Bilateral and asymmetric cortico-subcortical hypersignals, in parieto-occipital area. Figure 2 A	Bilateral and asymmetric cortico-subcortical hypersignals, in parietal, capsulo-lenticulars and caudate nuclei heads areas. Figure 3	Hypersignals of subcortical white matter in right frontal and left parietal area. Figure 4	Cortico-subcortical hypersignals at bilateral occipital area, more marked on the right. Figure 5	Bilateral posterior parietal hypersignals.
EEG	Diffuse paroxysmal activity for short time. Figure 1 B	Normal			Neurological distress signs	
Other analysis	Brain natriuretic protein at 457 pg/ml	HELLP syndrome	Normal	Normal	Normal	Normal
Delivery	Caesarean section for dynamic dystocia	Prophylactic caesarean section for surgical pelvis	Caesarean section for eclampsia	CS for 1st twin breech presentation and dynamic dystocia	Caesarean section for dynamic dystocia	Caesarean section for eclampsia
Treatment	-Midazolam, -Phenobarbital -Sodium valproat -Magnesium sulfate -Furosemid	-Diazepam, Phenobarbital - Nicardipin	-Midazolam -Nicardipin -Alpha-methyldopa -Magnesium sulfate -Corticosteroids	-Diazepam -Magnesium sulfate -Alpha-methyldopa	-Diazepam -Nicardipin	-Diazepam -Magnesium sulfate -Nicardipin
Exit prescription	-Sodium valproate -Heparin	-Sodium valproate -Heparin	-Sodium valproate -Heparin -Alpha-methyl dopa	-Sodium valproate -Acetyl salicylic acid -Alpha-methyl dopa	-Sodium valproate -Acetyl salicylic acid	-Acetyl salicylic acid
Evolution	Complete symptoms disappearance.	Normalization of blood pressure and biological assessment, return to initial neurological state	Blood pressure normalization and neurological signs improvment.	Blood pressure normalization and seizures disappearance.	Satisfying clinical evolution	Clinical signs improvment
Control	Clinical and radiological control at 9 weeks is without abnormalities.	2 months check-up: no clinical anomaly. Control MRI: complete brain damage resolution. Figure 2 B	Control at postpartum second month: normal clinical examination and control brain MRI showed disappearance of radiological lesions.	Control at postpartum 45th day; normal clinical exam. radiological brain MRI control showed disappearance of radiological lesions.	3 months check-up: no clinical anomaly.	3rd week check-up: no clinical anomaly. Brain MRI control: complete brain damage resolution.

Continued.

Variables	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5	Patient 6
Newborn	-Apgar 10/10 -3100 g -Alive	-Apgar 10/10 -2900 g -Alive	-Apgar 08/10 -950g -Neonatal intensive care hospitalization for 65 days	-Apgar 10/10 -2550 and 2400g -alives	-Apgar 10/10	-Apgar 4/10 -1050 g -Died after 3 hours

DISCUSSION

PRES actual impact is still unclear. Besides, there is no information about PRES incidence in peripartum.[1] Average age of PRES onset is around 45 years, it is less common in men than women.[2] Average age in three Moroccan series is around 29 years old.[3] Our patients are between 23 and 31 years old, with an average of 27±1.56 years. PRES can occur in much younger women; 21.4 years old as average age in Cunningham series, 21.8 years old in Brewer series and 20 years old in Loureiro series.[3-5]

PRES is more common in primigravida.[3] In our study, 5 patients (83.33%) were primiparous. Our patients' average term was 35 amenorrhea weeks (AW) with extremes of 27 AW and 38 AW and 5 days. There is not much difference with Araqi-Houssaini et al, Harandou et al and Brewer et al studies. [4,6,7]

In obstetrics, the majority of PRES cases are due to preeclampsia with 66.67% of cases in our series, or to one of its complications, specifically eclampsia, with 66.67% in our series or even less HELLP syndrome, with 16.67% in our series. In Zeeman's study, out of 27 eclampsia, 15 had developed PRES (55.5%).[8] In addition, the Araqi-Houssaini et al study focused on 298 cases of eclampsia, from which 89 images are showing PRES lesions in 13 cases (14.6%). [6]

In Brewer study, PRES occur in 50% of patients in prepartum, 76% of cases in Loureiro study and 61.5% of cases in Araqi-Houssaini et al study.[4-6] In our series, PRES occurred in prepartum in 2 out of 6 patients (33.33%). This could be explained by the fact that most eclampsia occurs during pregnancy or childbirth.

In postpartum, PRES occurs in 2/3 of cases within the first week after childbirth.[4] In our series, 6/6 of patients (100%) with PRES did not go beyond the first week of postpartum. This can sometimes happen later. However, PRES can occur during childbirth and immediate postpartum, favored by vasoactive drugs used in epidural anesthesia, postpartum hemorrhage and inhibition of lactation.[9]

Neurological manifestations occur suddenly, they are various and frequently include convulsion seizures, headaches, confusion, nausea and vomiting. These manifestations can lead to coma.[10]

Classically, headaches set in progressively, often isolated, bilateral and starting in posterior areas before becoming diffuse.[11] Thunderclap headaches are also possible.[6,11] All our patients (100%) presented severe "helmet-like" headaches.

Seizures are reported in 92% of cases, most often generalized.[10] In our series, 6 patients (100%) presented generalized tonic-clonic seizures. A status epilepticus is also possible, as presented by one of our patients (16.67%).[12]

Visual disturbances are present in more than 50% of cases. They vary from simple visual blurring to total cortical blindness, including hemianopsia.[13] In our series, one of our patients presented a bilateral hemianopsia (16.67%).

High blood pressure occurs during headache attacks. Thus, about a third of patients have high blood pressure during painful headache episodes.[10,12,13] All patients in our series had high blood pressure. The 4 patients (66.67%) presented a hypertension peak ranging from 167 mmHg/110 mmHg to 180 mmHg/110 mmHg on admission or at delivery. during seizures, 4 patients presented hypertensive peaks ranging from 180/100 mmHg to 230/120 mmHg.

Other neurological manifestations such as psychomotor slowing, confusion, lethargic state and agitation have been reported.[13] Two of our patients presented vertigo (33.33%), another presented consciousness troubles (16.67%).

There are also severe forms with hemorrhage or massive edema in posterior fossa, leading to hydrocephalus or brainstem compression. Finally, focal deficit signs can also exist such as hemiparesis or aphasia.[12]

In retrospective studies, brain computed tomography (CT) is performed in a high number of patients (65% to 100%).[10,14] Examination is generally normal or shows non-specific abnormalities in 66% of cases.[14] Lesions could appear as hypodensities of evocative topography which do not take contrast. Edema may be visible on CT scan but with imperfect sensitivity. Scanner is not a performant examination for diagnosis because it is falsely reassuring in 40% of cases. Brain CT was performed in 2 patients in series (33.33%) showing no abnormalities.

Brain MRI is the gold standard for both diagnosis and monitoring of PRES, it provides images with very high resolution, and detect invisible small focal lesions on CT scan.[15] The most commonly observed anomaly is cerebral edema without infarction, typically affecting subcortical white matter in posterior areas of cerebral hemispheres bilaterally and symmetrically, for the most part, in parietal-occipital regions.[3] Brainstem and cerebellum injuries are frequent, while frontal lobe damage is rare, with T1 hyposignal, T2 and Fluid-attenuated inversion recovery (FLAIR) hypersignal appearance. There is usually no enhancement after injection of contrast product.[16] MRI in diffusion sequence is the best diagnosis tool allowing an adapted and rapid treatment in order to prevent appearance of irreversible neurological lesions and permanent sequelae. Brain MRI was performed in 6/6 patients (100%), it showed bilateral subcortical foci, T1 hypointense appearance, T2 and FLAIR hypersignal in all patients (100%). Brain lesions predominate parietal and occipital posterior territories (6/6 cases) (100%), then in frontal areas (2/6 cases) (33.33%).

Angiography reveals vascular anomalies with focal and diffuse vasodilation and vasoconstrictions, generally causing a "pearl necklace-like" appearance, even in the absence of significant high blood pressure. However, it may be normal when it is done early. Other brain imaging techniques can be used such as proton magnetic resonance spectroscopy, brain scintigraphy/single-photon emission computed tomography (SPECT), apparent diffusion coefficient mapping (ADC) and 3D MRI angiography.

Diagnosis of PRES, as its name suggests, can only be made after a reversibility proof, by vascular imaging at three months (CT, MRI or arteriography) showing a return to normal/ at least a clear regression of vascular involvement.

Biology does not contribute to PRES positive diagnosis.[9] Lumbar puncture is almost systematic, in order to eliminate subarachnoid hemorrhage if brain CT scan is normal.[17] Biological assessment carried out in our patients, showing a significant proteinuria in 4 of them (66.67%), 1 patient presented HELLP syndrome (16.67%).

Electroencephalogram (EEG) usually shows slow and unresponsive traces with no focalization signs.[18] EEG was performed in 3 of our patients (50%), objectifying signs of neurological suffering in 2 patients (66.66%).

Both etiology and clinical presentation of PRES will determine therapeutic strategy. Stopping the triggering or aggravating factor is the first therapeutic measure.[7] there is no standardized therapy guidelines.[19] In fact, many treatments have been detailed, with very different and sometimes opposing outcomes.[20] However, controlling hypertension is the crucial part of management. It uses habitual antihypertensive agents: calcium channel blockers (nicardipin and diltiazem), beta blockers (labetolol) and diuretics. The objective is to maintain an average arterial pressure between 105 and 125 mmHg, without reducing this pressure more than 25% during first hour. Magnesium sulfate has a vasodilator effect, which increases blood flow to the brain, serving to prevent ischemic damage that causes seizures.[21] Corticosteroids are the most commonly used drugs against vasospasm and headaches. Association of anti-edematous treatment, mannitol in this case, should be discussed on case-by-case basis and may only be beneficial in a number of situations.[7] However, spontaneous regression of PRES makes the assessment of treatments efficacy difficult. Moreover, hypertension treatment should be careful because hypotension should not be induced when there is already a cerebral vasospasm reducing cerebral flow. In the occurrence of a seizure, antiepileptic treatment must be started urgently. Benzodiazepines should be administered as first line treatment.[22]

From a therapeutic perspective, our patients benefited from symptomatic treatment as follows: 1) anticonvulsant treatment: all patients (100%) received benzodiazepines (diazepam 4/6 or midazolam 2/6) in acute phase. Magnesium sulfate was administered in 4/6 of cases (66.67%). Sodium valproate and Phenobarbital were respectively received in 1/6 cases (16.67%). 2) antihypertensive treatment: in acute phase, 5/6 patients (83.33%) received antihypertensive monotherapy, one patient received bi-therapy (16.67%). As intensive care measures, patient who presented acute lung edema received non-invasive ventilation. Fluid-electrolyte re-balance, blood and platelet concentrates transfusion were administered to patient with HELLP syndrome. Fetal extraction as etiological treatment was carried out in 2 patients with prepartum eclampsia (100%).

One of PRES specific features is reversibility. Correct and early diagnosis and treatment of PRES can prevent neurological irreversible lesions.[23] Despite severity of initial clinical presentation (coma, status epilepticus), evolution is usually favorable, subject to early and appropriate management.[19] In the majority of cases, a complete regression of clinical and radiological abnormalities is observed within few days or even few weeks.[13] Nevertheless, normalization is sometimes only obtained after one year. It should be emphasized that lack of early treatment can lead to clinical aggravation.[23] PRES, because of its relatively recent description, has not yet been subject of prolonged longitudinal studies; clinical evolution of patients after regression is therefore very poorly understood.

In our series, clinical, biological and radiological evolution was favorable in 6/6 patients (100%). It was marked by complete clinical remission without recurrence. A control brain MRI was performed in all our patients, demonstrating a complete resolution of lesions, confirming PRES diagnosis.

Several cases of death during PRES have been described.[24] 30.7% in Araqi-Houssaini et al series and 30% in Salami et al series.[6,25] In our series, there were no deaths.

In our series, stillbirth rate did not exceed 15% (1/7 newborns died after third hour birth). While in Araqi-Houssaini et al study, stillbirth rate is 6/13 (46%).[6]

CONCLUSION

PRES is a poorly understood phenomenon. It is a neurological entity that can occur in peripartum, regardless of any pre-existing pathology. This syndrome should be correctly and quickly identified by clinicians and specifically radiologists, in order to prevent complications which can sometimes be extremely serious. Primary therapeutic action consists on eliminate a possible triggering or aggravating factor and initiate antihypertensive and anticonvulsant treatments. However, in some severe forms, reversibility is not always assured, in spite of a timely diagnosis and consequent therapy.

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