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Postpartum HELLP syndrome associated with posterior reversible encephalopathy syndrome: a case report and literature review

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ABSTRACT

Background. Haemolysis, elevated liver enzymes, low platelets (HELLP) syndrome is a life-threatening pregnancy complication. Various mechanisms may lead to diffuse endothelial damage which causes multi-organ dysfunctions, with possible brain involvement. We report a rare case of postpartum HELLP syndrome associated with posterior reversible encephalopathy syndrome (PRES) and conducted a literature review from the PubMed, Embase, and Cochrane databases to summarize the clinical, laboratory and radiological characteristics. We found only 10 papers describing this association occurring during pregnancy and 7 articles in the postpartum.
Case presentation. Our patient developed brief hypertensive peaks followed by two episodes of generalized tonic-clonic convulsions during postpartum. Brain magnetic resonance imaging allowed the diagnosis of PRES and an association with HELLP syndrome was established after blood exams. After medical therapy, the follow-up was marked by full recovery.
Conclusions. Patients with PRES do not always show typical manifestations and MRI is crucial for its diagnosis. Cases of postpartum PRES with involvement of anterior brain regions after HELLP syndrome are rare but must be considered and they should be kept in mind as a differential diagnosis in women presenting with seizures, headache and altered mentation. Clinicians should be aware of this neurological syndrome because prompt diagnosis and treatment may result in complete resolution.

INTRODUCTION

Haemolysis, elevated liver enzymes, low platelets (HELLP) syndrome is a life-threatening pregnancy complication, that is often associated with pre-eclampsia, although it has also been described in women without this diagnosis [1]. It usually develops in the last trimester of pregnancy or, more rarely, during the postpartum [2]. Various mechanisms are associated with gestational hypertensive disorders [3] and may lead to diffuse endothelial damage which causes multi-organ dysfunctions, with possible brain involvement [1]. We report a rare case of postpartum HELLP syndrome associated with posterior reversible encephalopathy syndrome (PRES). It is an uncommon disorder characterized by a broad range of non-specific neurologic manifestations, such as seizures, headache, altered mental status, and visual loss [4]. The neuro-imaging methods are essential to confirm the diagnosis [5]. It is caused by multifactorial aetiologies and its pathophysiology is unclear. It is considered a reversible condition if promptly diagnosed and treated, although irreversible brain damage might occur if therapy is delayed [6]. We also conducted a systematic review in the MEDLINE (PubMed), EMBASE, Cochrane Central Register of Controlled Trials, IBECS, BIOSIS, Web of Science, SCOPUS, congress abstracts, and Grey literature (Google Scholar; British Library) from inception to June 2022 searching for records reporting HELLP syndrome associated with PRES. The following keywords were used: "Posterior Leukoencephalopathy Syndrome", "PRES", "reversible posterior leukoencephalopathy syndrome" and "RPLS". These terms were further combined with "HELLP". We found only 17 records describing this combination, occurring during pregnancy in 10 papers [7-16] (**Table 1**) and in the postpartum in the other 7 [17-23] (**Table 2**).

CASE PRESENTATION

We report the case of a 19-year-old Italian primigravid woman, admitted to San Marco Hospital for premature rupture of membranes at 38 gestational weeks. Her family history was negative for hypertension and epilepsy, and she denied having had any problems before and during pregnancy, with normal arterial blood pressure (BP) during antenatal care. On admission, the patient had a BP of 132/84 mmHg and exhibited clear leaking amniotic fluid and cervical dilatation of 2 cm. Antepartum cardiotocography showed a reassuring foetal heart rate pattern, and the initial laboratory evaluation was unremarkable. On the same day, a female infant was born by uncomplicated spontaneous delivery in good health conditions, weighing 2,904 g and with a one- and five-minute APGAR scores of 9 and 10, respectively. At one hour after delivery, the patient developed a single, generalized tonic-clonic (GTC) seizure and a second episode after another hour, both stopped spontaneously within a few minutes and were associated with mild hypertensive peaks with BP of 140/90 mmHg. An emergent non-contrast brain computed tomography (CT) scan did not detect any anomalies (**Figure 1**). Brain magnetic resonance imaging (MRI) was performed after three and a half hours the delivery, revealing focal cortical-subcortical hypersignal on T2-weighted imaging (WI) and fluid-attenuated inversion recovery (FLAIR) sequences, involving the left occipital lobe, both hemispheres of parietal and frontal lobes bilaterally, and the cerebellum bilaterally (**Figure 2**).

These images were suggestive of PRES. Blood biochemistry showed haemolysis with hemoglobinemia (Hb) = 10.4 g/dL, lactate dehydrogenase (LDH) = 527 U/L (normal value ≤ 248 U/L), hypertransaminemia with aspartate aminotransferase (AST) = 123 U/L (normal value ≤ 35 U/L) and alanine aminotransferase (ALT) = 62 U/L (normal value ≤ 35 U/L), low platelet count (PLT) of $59 \times 10^3/\mu L$, and antithrombin III (ATIII) = 53%. Serum electrolytes, renal function, glucose, creatinine, prothrombin time, partial thromboplastin time, bleeding time and proteinuria were normal. A diagnosis of postpartum HELLP syndrome was established. Drug therapy during hospitalization included seizure prophylaxis with levetiracetam and magnesium sulphate, antihypertensive treatment with methyldopa and nifedipine, and ATIII replacement. The evolution was marked by the normalization of both neurological status and biological analysis. She was discharged in good condition on the fourth day with antihypertensive and antiepileptic outpatient therapy. A follow-up brain MRI performed on day 9 (**Figure 3**) showed significant radiological improvement; there were only small areas of oedema in the right frontal lobe. The outpatient follow-up confirmed the full recovery. The timeline of the salient events is illustrated in **Figure 4**.

Table 1. *HELLP syndrome and PRES occurring during pregnancy.*

Study	Age (years)	Gestational age (weeks)	Symptoms and signs	Laboratory findings	Brain TC	Brain MRI		Outcomes
						T2 and FLAIR	Diffusion	
Ates *et al.* 2015 [7]	29	35	Headache, peripheral oedema, blurred vision, hypertension	Class 1 HELLP, hypertransaminasemia, thrombocytopenia, elevated LDH, schistocytes, altered renal function, proteinuria	Not mentioned	Hyperintensities in the right occipital lobe, left cerebellum and right thalamus	No restriction	Complete recovery
Hegde *et al.* 2009 [8]	21	Not mentioned	GTC seizures, altered sensorium, generalized oedema, naso-oral bleeding, hypertension, oliguria	Class 1 HELLP, hypertransaminasemia, thrombocytopenia, elevated LDH, altered renal function, proteinuria	Bilateral ill-defined hypodensities in the posterior temporal, parieto-occipital, high frontal, and parietal lobes; bilateral watershed territory hypodensities with focal doubtful loss of gray-white matter differentiation	Not mentioned	Not mentioned	Complete recovery
Marano *et al.* 2003 [9]	30	32	GTC seizures, drowsiness, tiredness, anuria	Class 2 HELLP, hypertransaminasemia, thrombocytopenia	Not mentioned	Bilateral asymmetric hyperintensities in the subcortical parieto-occipital lobes	Not mentioned	Complete recovery
Morton *et al.* 2005 [10]	26	28	GTC seizures, confusion, tongue lacerations, epigastric pain, oedema, visual disturbance, hypertension	Class 2 HELLP, hypertransaminasemia, thrombocytopenia, altered renal function, proteinuria	Hypodensities in the right frontal and both occipital lobes	Not mentioned	Not mentioned	Complete recovery
Orgul *et al.* 2017 [11]	40	24	Seizures, headache, visual loss, hypertension	Class 1 HELLP, hypertransaminasemia, thrombocytopenia, proteinuria	Bilateral hypodensity in the occipital lobes and hyperaemia in the falx cerebri	Hyperintensities in both posterior hemispheres	Not mentioned	Complete recovery
Özden *et al.* 2020 [12]	29	36	Headache, nausea, vomiting, epigastric pain, visual loss, hypertension	Class 1 HELLP, hypertransaminasemia, thrombocytopenia, elevated LDH, hyperbilirubinemia, altered renal function, proteinuria	First interpretation: bilateral hypodensities in white matter substance, especially in the occipital lobes, and general oedema. Second interpretation: no signs of PRES	Not mentioned	Not mentioned	Complete recovery
Sarbu *et al.* 2018 [13]	24	35	Drowsiness, weariness in the limbs with gait difficulties, hypertension	HELLP, hypertransaminasemia, thrombocytopenia	Bilateral symmetric hypodensity of the basal ganglia	Symmetric hyperintensities in the lenticular and caudate nuclei, and internal and external capsule	Mild restriction in some areas	Complete recovery

Study	Age (years)	Gestational age (weeks)	Symptoms and signs	Laboratory findings	Brain TC	Brain MRI		Outcomes
						T2 and FLAIR	Diffusion	
Tetsuka *et al.* 2017 [14]	38	29	Headache, drowsiness, delirium, abdominal pain, hypertension	Class 1 HELLP, hypertransaminasemia, thrombocytopenia, elevated LDH, hyperbilirubinemia, altered renal function	Not mentioned	Hyperintensities in the cortical-subcortical white matter in the occipital lobes, basal ganglia and callosal splenium	No restriction	Complete recovery
Vijayalakshmi *et al.* 2010 [15]	28	16 triploid pregnancy	Headache, peripheral oedema, puffiness of the fingers, morning sickness, hemiparesis, epistaxis, epigastric pain, visual disturbance, hypertension	Class 1 HELLP, hypertransaminasemia, thrombocytopenia, hyperbilirubinemia, reduced haptoglobin, altered renal function, proteinuria	Bilateral hypodensities in the basal ganglia and internal capsule, more on the right side	Bilateral, almost symmetrical, hyperintensities in the head of the caudate nucleus, external capsules, and basal ganglia	Minor restriction in the right paraventricular area	Complete recovery
Zemple *et al.* 2017 [16]	21	32-35	GTC seizures, hypertension	Class 1 HELLP, hypertransaminasemia, thrombocytopenia, altered renal function	Hypodensities in the parieto-occipital lobes and indistinct areas of basal ganglia, putamen, and thalamus	Not mentioned	Not mentioned	Complete recovery

CT: computed tomography; FLAIR: fluid-attenuated inversion recovery; GTC: generalized tonic-clonic; HELLP: haemolysis, elevated liver enzymes, low platelets; LDH: lactate dehydrogenase; MRI: magnetic resonance imaging; PRES: posterior reversible encephalopathy syndrome.

DISCUSSION

We report a rare case of HELLP syndrome occurring in the immediate postpartum that was associated with clinical and neuroradiological findings consistent with PRES.

HELLP syndrome is a severe pregnancy disorder that occurs in the third trimester of pregnancy or, in one-third of the cases, postpartum [2], as in the present case. The incidence of HELLP syndrome is 7.6 events per 1,000 live births in tertiary obstetric centres [1]. It is thought to be due to microvascular injury caused by hypertension or by immunopathological mechanisms. In our case, the patient did not suffer from chronic hypertension, and this is unusual for HELLP syndrome in which only 20% of patients do not present with hypertension [1]. Feske *et al*. [18] showed that endothelium dysfunctions are likely important in the genesis of the syndrome, based on observing the similarities of HELLP and other vascular pathologies, such as thrombotic thrombocytopenic purpura and haemolytic-uremic syndrome. In these syndromes, the endothelium would be directly damaged by factors such as vasospasm, ischemia, platelet activation, increase in the levels of vasoactive molecules such as serotonin and thromboxane A-2, activation of the coagulation system, and impairment in the action of the normal placentally mediated adaptations to prostacyclin, nitrous-oxide, as well other endothelial mediators of the vascular tone and of vascular permeability. These mechanisms lead to multi-organ dysfunctions, with possible brain involvement. Organ dysfunction is highlighted by altered blood exams, such as anaemia with haemolysis, elevated liver enzymes (AST, ALT), low platelet count and ATIII levels [24], through which we established a diagnosis of post-partum HELLP syndrome. Platelet count is a prognostic factor, making a differentiation into three classes of decreasing severity according to the Mississippi-Triple Class System: class 1 (platelets $\leq$ 50,000/μl), class 2 (platelets > 50,000/μl $\leq$ 100.000/μl), and class 3 (platelets > 100,000/μl $\leq$ 150,000/μl) [9]. Therefore, our patient developed a class 2 HELLP syndrome with a platelet count of 59×10^3/μl. The only definitive treatment for gestational hypertensive disorders is childbirth. The disease usually regresses within 24-48 hours after delivery. However, timing is important to avoid premature births in planning the delivery. The criteria for determining delivery timing include the

Table 2. *HELLP syndrome and PRES occurring in the postpartum.*

Study	Age (years)	Gestational age (weeks)	Symptoms and signs	Laboratory findings	Brain TC	Brain MRI		Outcomes
						T2 and FLAIR	Diffusion	
Babahabib *et al.* 2015 [17]	31	38	GTC seizures, headache, hypertension	Class 1 HELLP, hypertransaminasemia, thrombocytopenia, proteinuria	Not mentioned	Hyperintensities in the parieto-occipital lobes	Not mentioned	Complete recovery
Feske *et al.* 1997 [18]	36	39	Lethargy, cranial nerve abnormalities, hypertension	Class 2 HELLP, hypertransaminasemia, thrombocytopenia, increased LDH, schistocytes, proteinuria	Hypodensities in the thalami, midbrain, pons, and cerebellum	Hyperintensities in the occipital lobes, thalami, midbrain, pons, and cerebellum	Not mentioned	Complete recovery
Grzesiuk *et al.* 2009 [19]	34	34	GTC seizures, drowsiness, blurred vision, hypertension	Class 1 HELLP, hypertransaminasemia, thrombocytopenia, increased LDH, hyperbilirubinemia	Not mentioned	Hyperintensity in the cortical-subcortical white matter, more intense in the occipital, parietal, and frontal lobes	No restriction	Complete recovery; small areas of oedema in the occipital and frontal lobes on MRI
Miyoshi *et al.* 2014 [20]	31	37	Headaches, nausea, loss of consciousness, backache, dyspnoea, peripheral oedema, blurred vision, hypertension	Class 1 HELLP, hypertransaminasemia, thrombocytopenia, increased LDH, hyperbilirubinemia, proteinuria	Slight hyperdensity in the cerebral base and diffuse brain oedema in the brainstem	Hyperintensity in the parietal and occipital lobes	Not mentioned	Complete postsurgical recovery
Negro *et al.* 2005 [21]	37	39	GTC seizures, stupor, confusion, visual loss, hypertension	Class 2 HELLP, hypertransaminasemia, thrombocytopenia, increased LDH, hyperbilirubinemia, reduced haptoglobin, schistocytes, proteinuria	Not mentioned	Diffuse cortical-subcortical hyperintensities in both hemispheres, the thalamus, cerebellum, and mesencephalon	Not mentioned	Complete recovery
Peng *et al.* 2008 [22]	36	38	GTC seizures, hypertension, loss of consciousness, general edema	Class 2 HELLP, hypertransaminasemia, thrombocytopenia, elevated LDH, proteinuria	Not mentioned	Hyperintensities in the right frontal lobe and bilateral basal ganglia	Not mentioned	Complete recovery
Takagi *et al.* 2016 [23]	37	38	GTC seizures, epigastric pain, vomiting, giddiness, hypertension	Class 2 HELLP, hypertransaminasemia, thrombocytopenia, elevated LDH, hyperbilirubinemia, reduced haptoglobin, schistocytes	Discreet bilateral hypodensity in the frontal lobes	Hyperintensities in the superior frontal gyrus, the precentral gyrus, and the cingulate gyrus	Not mentioned	Complete recovery
Present study	19	38	GTC seizures, hypertension	Class 2 HELLP, hypertransaminasemia, thrombocytopenia, increased LDH	Normal	Focal cortical-subcortical hyperintensities in the left occipital lobe, parietal and frontal lobes bilaterally, and the cerebellum bilaterally	Not mentioned	Complete recovery; small areas of oedema in the right frontal lobe on MRI

CT: computed tomography; FLAIR: fluid-attenuated inversion recovery; GTC: generalized tonic-clonic; HELLP: haemolysis, elevated liver enzymes, low platelets; LDH: lactate dehydrogenase; MRI: magnetic resonance imaging; PRES: posterior reversible encephalopathy syndrome.

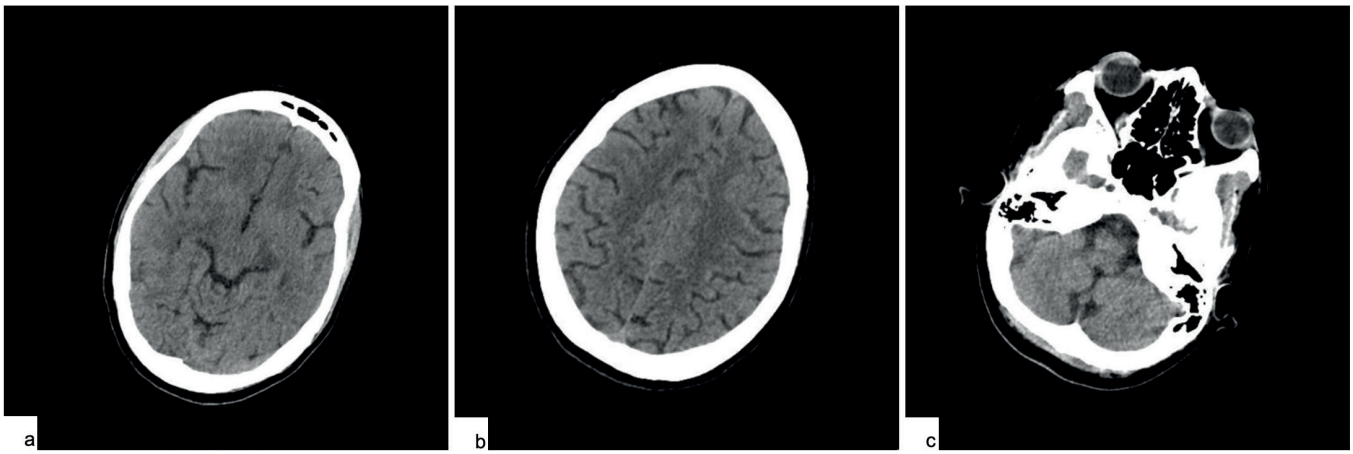

Figure 1. *Emergent non-contrast brain CT scan did not detect any anomalies in the occipital **(a)**, frontal and parietal **(b)** lobes, and in the cerebellum **(c)**.*

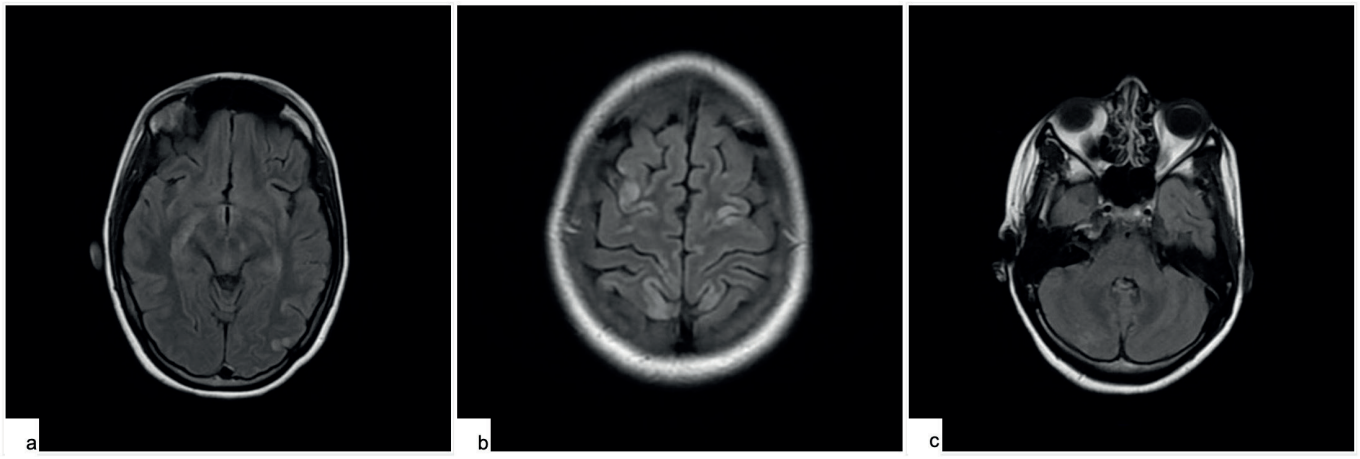

Figure 2. *Brain MRI performed after three and a half hours the delivery revealed focal cortical-subcortical hypersignal on T2-WI and FLAIR sequences, involving the left occipital lobe **(a)**, both hemispheres of parietal and frontal lobes bilaterally **(b)**, and the cerebellum bilaterally **(c)**.*

severity of the disease, gestational week, and maternal and foetal conditions. Expectant management with a close arterial blood pressure check and clinical monitoring should be considered in non-severe cases [25]. However, there is not wide consensus regarding the blood pressure cut-offs for the diagnosis and follow-up. The 2017 American College of Cardiology/American Heart Association (ACC/AHA) guideline changed the criteria for the diagnosis of hypertension in non-pregnant adults, moving the blood pressure cut-off from 140/90 mmHg to 130/80 mmHg [26]. Moreover, there is evidence from recent studies regarding the possibility of using 130/80 mmHg cut-off for pregnant women against the actual one of 140/90 mmHg [27]. Delayed diagnosis or inappropriate treatment account for the poor overall prognosis increasing the maternal and perinatal death rates [1]. The most common emergencies are haematological events (*e.g.*, disseminated intravascular coagulation, haemorrhage) and cardiopulmonary complications (*e.g.*, pulmonary oedema, adult respiratory distress syndrome, acute heart failure). Neurological disorders are infrequent, since they occur in 4.5% of class 1 patients, but they pose a high risk of morbidity and mortality [1]. They include seizures, ischemic and haemorrhagic strokes, cerebral venous thrombosis, eclampsia, cerebral artery dissection, encephalitis, and PRES. The last one, also known as reversible posterior leukoencephalopathy syndrome (RPLS), is a rare clinical and neuroradiological entity first described by Hinchey *et al*. in 1996 [28]. Its global incidence is unknown. Most cases have been reported in young to middle-aged adults with a marked female preponderance [4], as in the present case. The exact pathophysiology of PRES remains incompletely understood and is likely multi-factorial. Although most patients present severe hypertension, others only show mild hy-

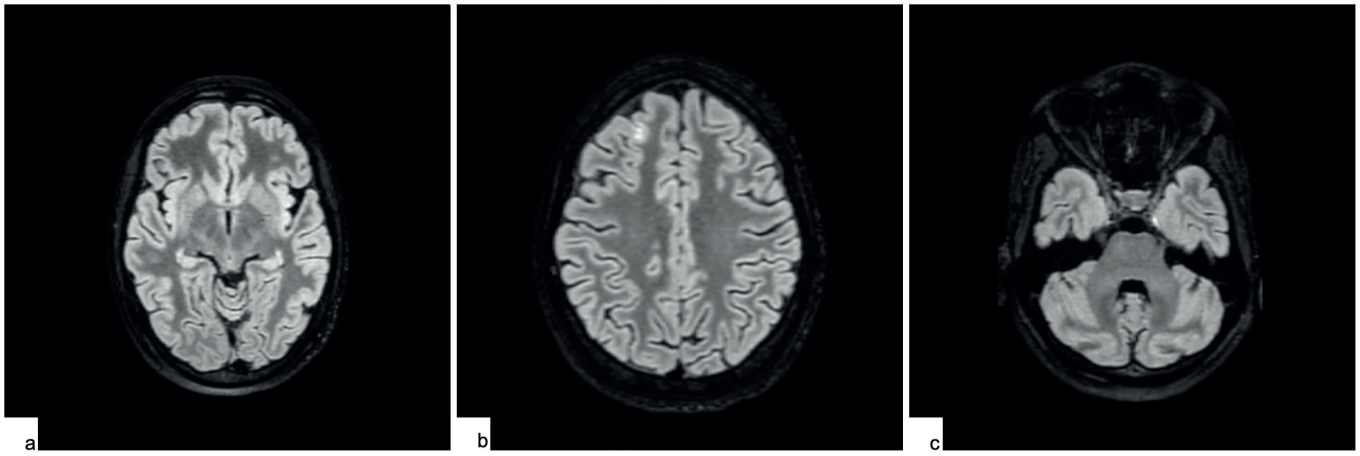

Figure 3. *Follow-up brain MRI performed on day 9 showed significant radiological improvement and did not detect any anomalies in the occipital lobe* ***(a)*** *and in the cerebellum* ***(c)****. There were only small areas of edema in the right frontal lobe* ***(b)****.*

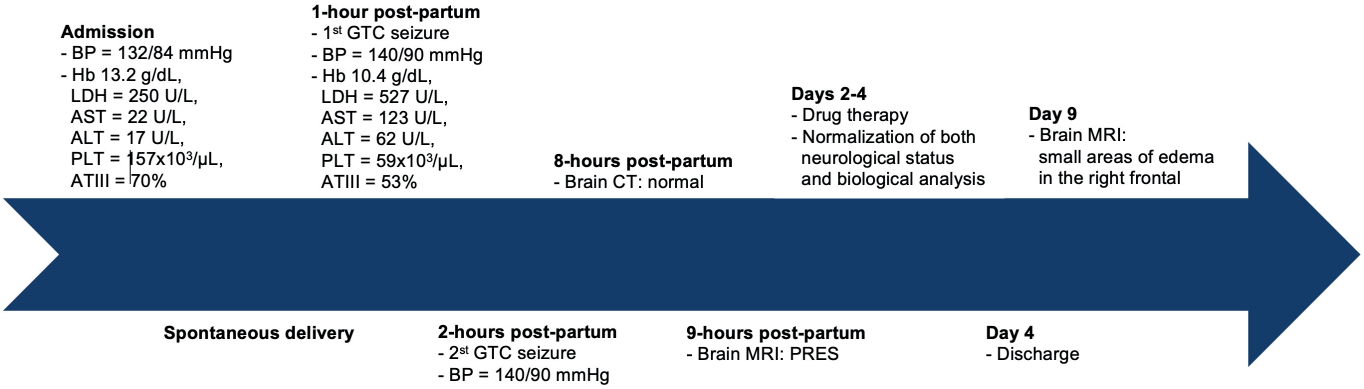

Figure 4. *Timeline of the salient events.*

pertension or normal BP [29]. In fact, it is associated with several medical conditions besides hypertension and preeclampsia/eclampsia, *e.g.*, renal failure, post-transplantation (allogeneic bone marrow transplantation and solid organ transplantation), immunosuppressive therapy (cyclosporine and tacrolimus), autoimmune diseases (systemic lupus erythematosus, systemic sclerosis, granulomatosis with polyangiitis, polyarteritis nodosa), post-cancer chemotherapy, electrolyte disturbances (hypomagnesemia, infections, sepsis, and shock) [30]. These causative factors of PRES were excluded in the present case by clinical and laboratory evidence. The pathophysiology of PRES is still under debate, but two possible hypotheses have been proposed, both caused by an abrupt rise in BP. According to the more widely accepted hyperperfusion theory, the brain tries to maintain a constant cerebral blood flow by autoregulation through sympathetic and arteriolar systems. As a result of loss of autoregulation, vasoconstriction – which occurs to protect the brain after a rise of systemic BP – cannot be sustained and passive dilatation of cerebral arterioles and consequent cerebral vasogenic oedema develop [4]. The second theory states that acute episodes of hypertension could cause cerebral vasospasm. Ischemia and cytotoxic oedema are thought to occur due to sympathetic nerve stimulation and cerebral vasoconstriction [31]. It must be highlighted that in the case of PRES, an elevation in BP might occur even in patients without chronic hypertension and the postulate is that it is a rapid increase in BP and not the absolute value that is important for the development of this condition [29]. The BP values at which cerebral symptoms emerge are often modest. In this case, the systolic BP was 140 mmHg and the diastolic one was 90 mmHg during the acute phase of her illness. This degree of hypertension often occurs without serious sequela, suggesting that

other factors may contribute to the pathogenesis of the brain lesions and the breakdown of the blood-brain barrier was probable because HELLP syndrome provokes endothelial damage. In the case of peripartum, there are other additional mechanisms, especially capillary hyperpermeability of pregnancy and substances modulating the vascular tone, such as the hypersensitiveness to endogenous vasopressor agents, and the reduction of prostaglandins and cytotoxic factor of placental origin, responsible for endothelial cell dysfunction [32]. Moreover, in the normotensive cases occurring after immunosuppressive or cytotoxic treatment, a toxic effect on vascular endothelium is hypothesized [33]. Preferential involvement of posterior brain regions may be due to relatively reduced sympathetic innervations in the posterior cerebral arterial circulation compared to the anterior vessels. This sympathetic innervation protects the brain from increases in systemic BP. Consequently, this autoregulation affords better protection against damaging hypertension to the anterior than the posterior circulation. However, the protective sympathetic mechanism of the anterior circulation also may be overcome in severe cases and the brain stem, basal ganglia, frontal and temporal lobes, and cerebellum may also be involved [7, 8, 10, 13-16, 18, 19, 21-23]. In our case, the involvement of the left occipital lobe, both hemispheres of parietal and frontal lobes bilaterally, and the cerebellum bilaterally indicates a severe disturbance in cerebral autoregulation. PRES is a clinical condition characterized by temporary neurological symptoms such as GTC seizures, sometimes complicated by status epilepticus, headaches, confusion (encephalopathy), nausea, and vomiting. There may be a focal neurological deficit, such as cortical blindness, cerebellar syndrome, or hemiparesis. These presentations may lead to coma and death [4]. Our patient presented brief hypertensive peaks followed by two episodes of GTC convulsions. However, she did not show focal neurological deficit, such as vision abnormalities, unlike other previously reported cases [7, 10-12, 15, 18-21]. There should be a high degree of clinical suspicion whenever there are predisposing causes and should be confirmed by neuroimaging modalities. Brain CT is the imaging modality of choice in the emergency department, but it may be normal, as in our case. In contrast, MRI is considered the gold standard in diagnosing PRES, and it has been demonstrated to be particularly sensitive to changes in the distribution of water in the brain. It can detect white matter oedema early, and reliably differentiate between vasogenic and cytotoxic oedema. It shows symmetrical white matter or cortical oedema more in the posterior cerebral hemispheres, particularly the parieto-occipital regions, with characteristic hypointense signals in T1-weighted and hyperintense signals in T2-weighted and FLAIR images [5]. Asymmetric appearance has also been noted [7, 22]. The MRI alterations seem to represent vasogenic oedema and not ischemic lesions, since they are usually reversible and there are areas which are spared, such as the medial part of the occipital lobe and the calcarine fissure [5].

The differential diagnosis for seizures in the postpartum includes eclampsia, subarachnoid haemorrhage, intracerebral haemorrhage, cerebral venous thrombosis, intracranial neoplasm, head trauma, idiopathic epilepsy, infection (meningo-encephalitis), amniotic fluid embolism, and postpartum angiopathy [4]. Based on the distinctive clinical and neuroradiological features of our patient and their reversibility, a diagnosis of PRES was clearly established. The mainstay of treatment of PRES is recognition and removal of the etiological causes which are the factors that triggered the event, *e.g.*, control of BP and seizures [4]. Our patient's seizures were controlled with levetiracetam and magnesium sulfate, and the BP was managed with methyldopa and nifedipine. Magnesium sulfate represents an important drug in many obstetric conditions [34, 35], but it can be burdened by toxicity, so magnesemia was monitored to ensure adequate serum level. PRES is not always reversible though the name suggests, and its outcome is variable. Prompt diagnosis and proper treatment can lead to a complete reversal of clinical and neuro-radiological findings with favourable outcomes [7-23]. However, in a few patients, it bears a risk of permanent cerebral damage if therapy is delayed [6]. The ideal timing for the repetition of brain imaging to document recovery is unclear, but in the largest reported clinical series with confirmed neuro-imaging improvement, resolution typically occurs in the range of several days [4]. Our patient underwent a follow-up MRI 3 days later with a complete resolution of the brain lesions, supporting the previous diagnosis of PRES. A full recovery was confirmed at a second follow-up MRI performed on day 9 and the absence of clinical anomalies at the outpatient follow-up. Strengths

of the present study are the rarity of the present case reporting the association between HELLP syndrome and PRES; the systematic review, describing the clinical, biochemical and radiological characteristics of the cases previously described both during pregnancy and in the postpartum and comparing the similarities and differences between our case and the others. Limitations include the few cases described in the previous literature and the lack of a wide consensus regarding the therapeutical approach.

CONCLUSIONS

We raised attention to a rare association between postpartum HELLP syndrome and PRES. Postpartum PRES is still largely unknown. Its pathophysiology remains controversial, and the list of conditions known to be associated is increasing steadily. Patients with PRES do not always show typical manifestations and MRI is crucial for its diagnosis. Cases of postpartum PRES with involvement of anterior brain regions after HELLP syndrome are rare but must be considered and they should be kept in mind as a differential diagnosis in women presenting with seizures, headache and altered mentation. We believe that clinicians should be aware of this neurological syndrome because prompt diagnosis and treatment may result in complete resolution.

COMPLIANCE WITH ETHICAL STANDARDS

Authors contribution

G.G.I.: Conceptualization, writing – original draft. F.G., D.I., A.V., P.C., C.S.: Writing – review and editing. M.P.: Supervision.

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Study registration

N/A.

Disclosure of interests

The authors declare that they have no conflict of interests.

Ethical approval

Not required.

Informed consent

Informed consent was obtained from the patient for publication of this case report and any accompanying images.

Data sharing

Data are available under reasonable request to the corresponding author.

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