Chapter

Ophthalmic Disorders in Posterior Reversible Encephalopathy Syndrome Associated with Preeclampsia

Katarina Cvitkovic, Anita Pusic Sesar, Antonio Sesar and Ivan Cavar

Abstract

Posterior reversible encephalopathy syndrome (PRES) is a clinicoradiological entity presented with different symptoms such as visual disturbances, headaches, seizures, severe hypertension and altered mental status. It has been recognized in a different pathological conditions, although preeclampsia/eclampsia is the most common cause of PRES. The pathogenesis of PRES is still not fully understood, but it seems that failure of cerebrovascular autoregulation causing vasogenic edema, cerebral vasoconstriction, and disruption of the blood brain barrier plays an important role. Cortical blindness, hypertensive retinopathy, serous retinal detachment (SRD), central retinal artery and vein occlusions, retinal or vitreous hemorrhages, anterior ischemic optic neuropathy (AION) and Purtscher's retinopathy are ophthalmic disorders that may occur in PRES associated with preeclampsia. Among these, cortical blindness is the best documented complication of preeclampsia. Magnet resonance imaging (MRI) is a gold standard to establish the diagnosis of PRES because clinical findings are not sufficiently specific. Typically, there are bilateral cortical occipital lesions with hyperdensity on T2-weighted MRI. Blindness due to occipital lesions is reversible and the vision loss is usually regained within 4 h to 8 days.

Keywords: preeclampsia, eclampsia, posterior reversible encephalopathy syndrome, ophthalmological disorders, cortical blindness

1. Introduction

Posterior reversible encephalopathy syndrome (PRES) is a clinicoradiological entity presented with different symptoms, such as headaches, seizures, visual disturbances, severe hypertension, and altered mental status [1]. Previously, it has been known by various names such as reversible posterior leukoencephalopathy syndrome, reversible posterior cerebral edema syndrome, and reversible occipital parietal encephalopathy [2, 3]. PRES was first described in 1996 by Hinchey et al. in patients with acute neurological symptoms and since then it has been recognized in different pathological conditions such as preeclampsia, eclampsia, hypertensive

encephalopathy, autoimmune diseases, renal failure, infection, and the use of cytotoxic or immunosuppressive drugs [1, 3–9]. Among these, preeclampsia and eclampsia are the most common causes of PRES. Preeclampsia is pregnancy-specific disorder clinically characterized by a new onset of hypertension and proteinuria that appear after the 20th week of gestation and up to 6 weeks postpartum in a previously normotensive woman [5, 6]. Preeclampsia and its variants affect approximately 5% of pregnancies and is the leading cause of both maternal and fetal morbidity and mortality worldwide [10]. It is characterized by impaired organ perfusion that occurs as a result of vasospasm and activation of the coagulation system [11]. Eclampsia is an acute cerebral complication of preeclampsia, presented with the occurrence of tonic-clonic convulsions in pregnant or recently postpartum women [12]. Severe intracranial vasospasm, local ischemia, intracranial hypertension, and endothelial dysfunction associated with vasogenic and cytotoxic edema are possible causes of seizures in PRES [11]. Rare cases of PRES in pregnant women with normal blood pressure and without preeclampsia have also been described in the literature [13]. Early recognition of PRES is essential in order to timely apply the medication, which typically includes drugs that lower blood pressure, act as antiedematous and interrupt tonic-clonic convulsions [7].

2. Pathogenesis of PRES

The exact pathophysiological mechanisms of PRES are not precisely known. Failure of cerebrovascular autoregulation, cerebral vasoconstriction, and disruption of the blood brain barrier due to endothelial disfunction are possible mechanisms involved in the pathogenesis of PRES [2]. Failure of cerebrovascular autoregulation causing vasogenic edema is the most accepted one. It seems that hyperperfusion plays a crucial role in disorders where hypertension is a key feature, such as in preeclampsia [14]. During fluctuations of systemic blood pressure, cerebrovascular autoregulation maintains cerebral blood flow, leading to vasodilation during systemic hypotension and vasoconstriction during systemic hypertension. The rapid development of hypertension can exceed the capacity of cerebral blood flow autoregulation leading to hyperperfusion [14]. It is supposed that posterior brain regions are more vulnerable to hyperperfusion, which is explained by better autoregulation of the anterior circulation due to better sympathetic innervations as compared to the posterior circulation [15]. Another theory suggests spasm of cerebral arteries in response to acute hypertension, thus resulting in decreased cerebral blood flow, intraarterial thrombosis, and cerebral ischemia leading to cytotoxic edema, especially in the border zones between arterial territories [16–18]. Breakdown of the blood brain barrier and endothelial dysfunction occurs in PRES with fluid and macromolecule extravasation into the interstitium. Further, increased concentrations of circulating cytokines activate endothelial cells and allow adhesion of circulating leukocytes. On the other hand, the tight junctions are disrupted and vascular endothelial growth factor expression is increased, leading to increased vascular permeability and vasogenic edema [19].

3. Neuroimaging features of PRES

The diagnosis of PRES cannot be established exclusively on clinical findings [1]. Brain lesions are usually located in the white matter, although rarely, overlying cortex may also be affected [20]. The parieto-occipital regions of the brain are main foci of changes, that are usually bilateral and symmetric. However, the lesions can

also extend to other brain structures such as the frontal and temporal lobes, cerebellar hemispheres, basal ganglia, brain stem, and deep white matter [21]. A multislice computed tomography (MSCT) scan is often normal or shows cortical-subcortical hypodensities, predominantly in posterior brain regions. However, MSCT scans in PRES show lesions in only of about 50% cases [22]. Because of that, magnet resonance imaging (MRI) is a gold standard for the diagnosis of PRES and the follow-up of these patients. Neuroimaging studies showed hypointense or isointense signal changes on T1-weighted images. The typical neuroimaging feature is a high signal intensity on T2-weighted images predominantly in the posterior regions, which is caused by subcortical white matter vasogenic edema [1]. Abnormalities are more observable on fluid-attenuated inversion recovery imaging (FLAIR), which increases the ability to detect subtle lesions in PRES [15]. Supplemental diffusion-weighted imaging (DWI) and apparent diffusion coefficient (ADC) map images are helpful in distinguishing vasogenic oedema from cytotoxic edema [15–19]. Cytotoxic edema appears hyperintense on DWI with a low signal intensity image on the corresponding ADC sequence. A predominantly low signal on DWI and a high signal on ADC image indicates vasogenic edema. Vasogenic edema and cytotoxic edema can also coexist in eclampsia [23].

4. Ocular disorders in PRES associated with preeclampsia

Visual disorders in pregnancy can be highly variable and range from mild symptoms such as transient blurred vision, photopsia, and different types of visual field defects to transient or permanent total blindness [24]. Vision loss during pregnancy has been documented in 1–3% of cases and a possible causes are cortical blindness, central retinal artery and vein occlusions, retinal detachment, ischemic optic neuropathy, retinal or vitreous hemorrhages, and Purtscher's retinopathy [24–26]. Approximately 25% patients with severe preeclampsia and 50% patients with eclampsia present different visual symptoms including blurred vision, homonymous hemianopsia, visual neglect, visual anosognosia, and cortical blindness [27, 28]. They seem to be a consequence of the cerebral edema located in the occipital cortex or in the temporal and parietal association cortices. In most patients, the visual impairment is reversible, but in rare cases, permanent blindness has been described [29].

4.1 Cortical blindness

Cortical blindness is among the best-documented complications of preeclampsia/eclampsia and affects almost 15% of eclamptic women [30, 31]. Cortical blindness is caused by a dysfunction in the visual pathway that conducts visual information from the lateral geniculate nucleus of the thalamus to the cerebral visual cortex [20]. Vasogenic edema is the main cause of cortical blindness. It appears that the primary visual cortex in the occipital lobes is more susceptible to the breakdown of cerebrovascular autoregulation and subsequent hyperperfusion than other brain regions [31]. Cortical blindness has been described to occur several hours before or after eclamptic seizures, rarely several days or weeks postpartum [32–34]. The bilateral vision loss often begins with blurry vision and progresses within a couple of hours to bare light perception [35]. Sometimes, the woman is unaware of her blindness and feels that she can see, which is known as visual anosognosia or Anton syndrome, indicating involvement of the visual association cortex [34, 36]. However, cortical blindness is associated with an intact pathway from the eye to the lateral geniculate body and therefore ophthalmological

examination including the pupillary reflexes, ocular motility, and fundoscopic findings is normal in these patients [29]. Blindness due to occipital lesions is reversible and the vision loss is usually regained within 4 h to 8 days [27].

5. Other ocular disorders in pregnancy complicated with preeclampsia

5.1 Hypertensive retinopathy

According to some studies, the prevalence rate of hypertensive retinopathy in women with hypertensive disorders in pregnancy is 32.5% [37]. Generally, the retinal vascular changes correlate with the severity of systemic hypertension. At the pathophysiological level, increased blood pressure leads to focal or diffuse vasoconstriction. In addition, increased vascular permeability leads to the extravasation of fluid to the extravascular spaces. Clinically, the most common abnormality seen during fundoscopy is narrowing of retinal arterioles [38]. Other retinal changes that may be present are decreased retinal artery to vein ratio, cotton wool spots, hemorrhages and Elschnig spots [39]. Vasospastic manifestations are reversible, and the retinal vessels rapidly return to normal after delivery [38].

5.2 Serous retinal detachment

Serous retinal detachment (SRD) is a rare complication of hypertensive disease in pregnancy, affecting 1–2% of preeclamptic and 10% of eclamptic women [40]. It is characterized by separation of the neurosensory retina from the pigmented retinal epithelium and it is usually observed in the absence of significant retinal vascular abnormalities or retinal breaks [41]. It may be present either before or after delivery [42]. Clinically, patients report loss of visual acuity and visual field defects [42, 43]. The detachments are often bullous and bilateral [27]. The exact pathophysiology of SRD in cases of preeclampsia is not well known, but it seems to be related with choroidal ischemia, which is secondary to an intensive arteriolar vasospasm [27]. The choroidal vascular insufficiency can lead to lesions in retinal pigment epithelium, fluid transudation, and focal retinal detachment. The majority of women who manifest SRD during pregnancy have a gradually improvement of visual acuity in few weeks after delivery, ending with complete recovery of vision [31]. Management of SRD in preeclampsia is conservative and involves treating the underlying condition [27].

5.3 Purtscher's retinopathy

Purtscher's retinopathy is a rare cause of visual loss during pregnancy and has been mostly described in association with complicated labor [24]. However, isolated cases during normal spontaneous labor have also been described in the literature [44]. Clinically, it presents with decreased visual acuity and a different types of visual field defects such as central or paracentral scotoma. The retinal changes include ischemia at the posterior pole with white patches of edema known as Purtscher's flecken, which represent areas of capillary bed infarction. In the initial phase, the optic disc is normal, but in the later phases, disc pallor and optic atrophy occur [27]. According to some researchers, these fundoscopic changes may be caused by embolic occlusion of the precapillary arterioles of the retina by fat, air, platelets, and leukocyte aggregates [45, 46]. The diagnosis is established on the basis of clinical findings and confirmed by intravenous fluorescein angiography [47]. The majority of patients recover some of their visual function without

treatment. Use of systemic steroids may improve visual outcome in some patients, but momentary specific medication is not available [47].

5.4 Anterior ischemic optic neuropathy

Anterior ischemic optic neuropathy (AION) is a rare ophthalmological disorder in preeclampsia that has been described to occur before and after delivery. Clinically, it is presented with sudden vision loss and unilateral or bilateral disc edema [48, 49]. The exact pathophysiology of AION in preeclampsia remains unclear, but it is suggested that uncontrolled hypertension leads to vasoconstriction or ischemia in the posterior ciliary artery circulation [49].

5.5 Central retinal vein occlusion

Central retinal vein occlusion (CRVO) is also described in preeclamptic women and was presented with bilateral vision loss up to 21 days postpartum. Ophthalmologic examination reveals multiple retinal hemorrhages in all 4 quadrants, venous dilatation, and macular edema. Improvement of visual acuity is significant but not complete [31]. The exact pathophysiological mechanism of CRVO in preeclampsia is not fully understood; however, it is proposed that central retinal artery thickening is thought to cause compression of the central retinal vein, thereby leading to venous occlusion [50, 51].

5.6 Central retinal artery occlusion

Central retinal artery occlusion (CRAO) is rare in young people, and it is usually associated with a predisposing pathological disorders such as cardiac valvular disease, systemic vascular disease, and hypercoagulable disorders. According to some studies, CRAO is rarely described in women with eclampsia [52, 53]. Clinically, it is presented with sudden, painless, and persistent vision loss. Fundoscopy shows typical changes such as pallor of posterior pole with cherry-red spot. It seems that the activation of coagulation system could be a cause of multiple emboli and vascular occlusion in these patients [53].

5.7 Retinal and vitreous hemorrhages

Retinal and vitreous hemorrhages are rare disorders that may precede the appearance of preeclampsia. They are presented as a sudden vision loss in a normotensive pregnant women, who usually develop preeclampsia within 2 weeks after labor [54, 55].

6. Conclusion

Preeclampsia and eclampsia are the most common conditions associated with PRES. Due to the high predilection of pathological lesions in white matter of the occipital lobes, PRES could be manifested with different types of ocular disorders. Some of these complications can be serious including cortical blindness, SRD, CRVO, CRAO, AION, and vitreal and retinal hemorrhages. Clinicians should be aware of these ocular manifestations, and careful ophthalmological, neurological, and neuroradiological evaluation should be carried out to ascertain the various causes of vision loss in pregnancy. In most cases, visual prognosis is usually good with permanent vision recovery. Effective treatment of preeclampsia/eclampsia along with termination of pregnancy is the mainstay of treatment.

Conflict of interest

The authors declare they do not have any conflict of interest.

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