Blindness associated with preeclampsia and eclampsia

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OBJECTIVE: Over a 14-year period at Parkland Hospital, the clinical courses of 15 women with severe preeclampsia or eclampsia were further complicated by blindness. Our purpose is to describe their management and outcome, as well as to offer insight to the pathophysiologic characteristics of blindness complicating pregnancy-induced hypertension.
STUDY DESIGN: Prospective ascertainment of women with blindness and pregnancy-induced hypertension was done. These cases were managed according to the standardized preeclampsia-eclampsia regimen used at our hospital since 1955. Briefly, this regimen includes magnesium sulfate given intramuscularly to prevent or control seizures, hydralazine to lower dangerously elevated blood pressure, intravenous fluid restriction, and delivery.
RESULTS: There were 15 women with blindness that persisted from 4 hours to 8 days; it subsequently resolved completely in all. Of the 13 women who underwent computed tomography, 8 had low-density areas localized predominantly in the occipital lobes. Five of these 13 subsequently underwent magnetic resonance imaging and 2 showed corresponding hyperintense lesions in the occipital areas.
CONCLUSIONS: On the basis of previously published experiences with computed tomography in women with eclampsia, as well as the experiences described here, we conclude that cortical blindness associated with preeclampsia-eclampsia results from petechial hemorrhages and focal edema in the occipital cortex. These lesions are likely stimulated by disparity in cerebral regional blood flow that is characterized by vasospasm and diminished flow primarily affecting the posterior circulation. (Am J Obstet Gynecol 1995;172:1291-8.)

Key words: Eclampsia, preeclampsia, blindness, computed tomography, magnetic resonance imaging

Except when overt cerebral hemorrhage is identified, the neuropathologic mechanisms for eclampsia are unclear. Some factors that have been implicated include cerebral vasospasm, hemorrhage, ischemia, and edema, as well as hypertensive and metabolic encephalopathy.[1, 2] Recently, Schwartz et al.[3] proposed that findings of preeclampsia-eclampsia can be explained by the loss of autoregulation of the posterior cerebral circulation. Aside from convulsions, other dramatic neurologic effects, albeit uncommon, include blindness, an altered state of consciousness, and coma. There are a number of case reports that describe women with these findings, and in some, abnormalities were seen on neuroimaging studies. In this article we report the clinical courses of 15 women in whom preeclampsia-eclampsia was further complicated by blindness. Abnormal findings in these women were commonly seen by computed tomography or magnetic resonance imaging techniques. Our purpose is to correlate these radiologic findings with clinical and pathologic changes.

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Material and methods

During the 14-year period from 1977 to 1990, 15 women with preeclampsia or eclampsia whose infants were delivered at Parkland Hospital had a clinical course that was further complicated by blindness. During this same period approximately 165,000 women were delivered of infants and the incidence of pregnancy-associated hypertension ranged from 12% to 14% annually. Also during this time 70 women were diagnosed as having eclampsia. These cases were managed according to the standardized treatment regimen for preeclampsia-eclampsia in use at our hospital since 1955.[4] Preeclampsia and eclampsia were defined conventionally,[5] and all 15 women had proteinuric hypertension. Women with these complications were ascertained prospectively and followed up clinically by at least one of the authors. All of these women were examined by a neurologic and ophthalmologic consultant; 13 had cranial computed tomography, and 5 had magnetic resonance imaging. None had a diagnosis of seizure disorder, migraine headaches, or collagen-vascular disease.

Table I. Demographic, clinical, and laboratory data in 15 women with preeclampsia-eclampsia complicated by blindness

						Clinical findings			
						On admission		*At central nervous system event*	
Patient No.	*Age (yr)*	*Race*	*Parity*	*Diagnosis*	*Neurologic complications and duration*	*Blood pressure (mm Hg)*	*Proteinuria*	*Blood pressure (mm Hg)*	*Proteinuria*
1	32	Black	4	Antepartum eclampsia	Cortical blindness, 24 hr	240/140	4+	N/A	N/A
2	23	White	0	Antepartum eclampsia	Cortical blindness, 62 hr	230/120	2+	160/80	3+
3	23	Latin	1	Preeclampsia	Cortical blindness, 58 hr	140/80	2+	170/92	4+
4	24	Latin	0	Preeclampsia	Cortical blindness, 48 hr; stupor, bilateral retinal detachment	140/100	1+	180/120	–
5	19	Latin	0	Postpartum eclampsia	Cortical blindness, 30 hr	150/94	4+	170/90	–
6	34	Black	1	Preeclampsia	Cortical blindness, 192 hr; obtunded	210/94	3+	194/120	4+
7	18	Latin	0	Antepartum eclampsia	Cortical blindness, 16 hr	150/100	4+	164/124	–
8	22	Latin	1	Antepartum eclampsia	Cortical blindness, 96 hr; hallucinations	100/70	4+	170/110	–
9	19	Latin	0	Antepartum eclampsia	Cortical blindness, 21 hr	160/110	3+	130/100	2+
10	23	Latin	1	Preeclampsia	Blindness caused by retinal detachment, 96 hr	170/108	3+	160/100	2+
11	20	Latin	1	Preeclampsia	Cortical blindness, 72 hr; obtunded	140/90	3+	132/86	4+
12	16	Latin	0	Intrapartum eclampsia	Cortical blindness, 24 hr	176/110	2+	170/120	–
13	16	Latin	0	Postpartum eclampsia	Cortical blindness, 22 hr	140/90	2+	250/150	1+
14	18	Black	1	Antepartum eclampsia	Cortical blindness, 42 hr	190/110	2+	200/120	–
15	18	Latin	0	Antepartum eclampsia	Cortical blindness, 4 hr	150/90	2+	150/104	–

N/A, Not available.

Results

Demographic, clinical, and laboratory findings from these 15 women are summarized in Table I. Of the 15 women, 14 (93%) had presented for prenatal care, 1 had chronic hypertension, and 3 had prior preeclampsia. The racial distribution was not reflective of the general obstetric population at Parkland Hospital (73% of the study women were Hispanic, 20% were African-American, and 7% were white, compared with 33%, 39%, and 27% of all obstetric patients during the 14-year period). The mean age was 21.7 ± 5.3 years (range 16 to 34), and 13 (87%) were younger than 25.

Laboratory values (nadir or peak value)			*Neuroimaging studies*		
Platelets ($10^3/\mu l$)	*Aspartate aminotransferase (U/L)*	*Serum creatinine (mg/dl)*	*Computed tomography*	*Magnetic resonance imaging*	*Comments*
90	270	1.4	–	–	No follow-up
295	77	0.9	–	–	No follow-up
284	22	0.7	Day 6: Bilateral nonenhancing occipital low-density area	–	Normotensive post partum, prior preeclampsia, recurrent preeclampsia
78	96	1.0	Day 0: Bilateral low-density areas in occipital and bifrontoparietal regions Day 5: Resolving nonenhancing low-density areas	–	No follow-up
285	93	0.8	Day 0: Low-density lesions in occipitoparietal areas	–	Recurrent preeclampsia, subsequent chronic hypertension
39	172	2.1	Day 0: Scattered low-density areas, most prominent in occipital areas; cerebral edema with flattened sulci and compressed ventricles Day 4: Partial resolution Day 15: Normal	–	Normotensive post partum
192	110	1.6	Day 1: Multiple low-density areas, bifrontal, parietal, and occipital areas Day 2: Partial resolution	–	Normotensive post partum
272	31	0.6	Day 0: Low-density areas in left parietal occipital lobe	Day 4: Hyperintense lesion in left occipital lobe	Normotensive post partum, subsequent idiopathic seizure disorder
273	111	0.8	Day 0: Normal Day 3: Normal	–	Postpartum psychosis, normotensive
230	27	0.9	Day 0: Normal	Day 1: Normal	Normotensive post partum, prior severe preeclampsia
237	76	0.8	Day 1: Multiple low-density areas in white matter, effacement of sulci consistent with edema	Day 4: Small high-signal lesion in lateral basal ganglia on right	Twin gestation at 18 wk, prior chronic hypertension
167	138	0.9	Day 0: Right large occipital low-density area, edema on left Day 5: Partial resolution	–	No follow-up
227	64	0.9	Day 0: Normal	Day 1: Normal	Normotensive 2 mo post partum
306	201	0.8	Day 0: Normal Day 5: Normal	Day 8: Normal	No follow-up
166	225	1.0	Day 0: Normal	–	Normotensive post partum

Eight were nulliparous, 6 were primiparous, and only 1 was multiparous. Their gestational ages when preeclampsia-eclampsia developed ranged from 18 to 40 weeks (mean 34 ± 5.3) with 10 women < 37 weeks and 5 between 27 and 32 weeks.

Of these 15 women with blindness, 5 had severe preeclampsia and 10 had eclampsia (7 antepartum, 1 intrapartum, and 2 post partum). At the time of admission 5 (33%) had diastolic blood pressure ≥ 110 mm Hg and 6 (40%) had systolic blood pressure ≥ 170 mm Hg. At the time of the central nervous system changes, 7 women (47%) had diastolic blood pressure ≥ 110 mm

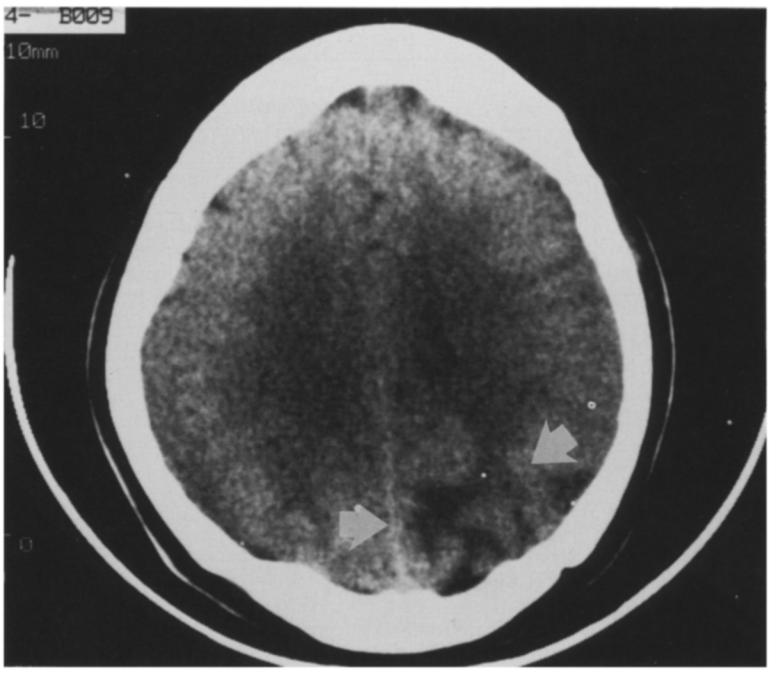

Fig. 1. Tomographic findings of left occipital low-density area *(arrows)* seen in patient 8 from Table I.

Hg and 8 (53%) had systolic blood pressure ≥ 170 mm Hg.

Shown in Table I are data from these 15 women. Ten had isolated blindness with eclampsia, 2 had isolated blindness with preeclampsia, and 3 had blindness along with an altered state of consciousness (e.g., obtundation or stupor). Two women had retinal detachments, and one of them also had concomitant evidence of cortical blindness. Nine women had premonitory symptoms of a severe headache before the onset of blindness or seizures. In 3 of 10 with eclampsia cortical blindness was the primary clinical presentation and preceded seizures by 4 to 7 hours. In the remaining 7, blindness developed 1 to 32 hours after seizures had been controlled with magnesium sulfate. The duration of blindness in these 10 eclamptic women ranged from 4 to 96 hours (mean 34.1 ± 26.7). Cortical blindness developed in 4 preeclamptic women, antepartum in 3 and 24 hours post partum in 24 hours. The duration of blindness ranged from 48 to 192 hours (mean 92.5 ± 67.1). Two of the 13 women with pure cortical blindness also had an altered state of consciousness, which we described as being obtunded. In both of these women the duration of the visual disorders surpassed that of the brain dysfunction.

To assess possible effects of overhydration, we evaluated fluid therapy and volume status during the critical antepartum, intrapartum, and postpartum periods. In 6 women blindness was apparent at the time of admission and the possibility of fluid overload was discounted. The remaining 9 women were managed according to our protocol that calls for fluid restriction.[4, 5] Specifically, they had been given a mean of 55 ml/hr lactated Ringer's solution before becoming blind.

As shown in Table I there were varying but usually mild degrees of laboratory abnormalities in 12 of these women. Three women had evidence of renal dysfunction with serum creatinine concentrations ranging from 1.6 to 2.1 mg/dl. Twelve women had elevated serum aspartate aminotransferase levels that ranged from 64 to 270 U/L. Three women had thrombocytopenia with concentrations ranging from 39,000 to 90,000 platelets/μl.

Eleven of the 13 women with pure cortical blindness underwent computed tomography. Seven had focal low-density modifications that were observed most frequently in the occipital lobe region (Fig. 1). These hypodense lesions commonly were seen bilaterally. Follow-up scans performed in 3 of these women showed partial resolution of the low-density areas within 2 to 5 days. The 2 women with pure cortical blindness who also had altered states of consciousness (patients 6 and 11) had more diffuse tomographic abnormalities that included widespread edema reflected by effacement of sulci and compressed ventricles. Patient 6 had partial radiologic resolution on day 4 and complete resolution by day 15.

Magnetic resonance imaging was performed in 4 of

the 11 women with pure cortical blindness who underwent tomography (patients 8, 11, 13, 14). Two of these had high-signal-intensity lesions corresponding to the low-density areas seen on computed tomography (Figs. 1 and 2). In the other two both studies were normal.

Two women with blindness had evidence of retinal detachment seen on ophthalmologic examination. One of these (patient 4) also had computed tomographic evidence of a low-density lesion in the occipital lobe. The other woman had both normal computed tomography and normal magnetic resonance studies.

Comment

According to Dieckman,[6] although visual disturbances develop in perhaps 25% of women with severe preeclampsia, blindness is rare, and he cited an incidence with eclampsia of 1% to 3%. From our experiences now described blindness is much more common and developed in almost 15% of women with eclampsia whom we cared for over this 14-year period. There have been a number of case reports of women with blindness complicating preeclampsia-eclampsia, and these are summarized in Table II. In the past most cases of blindness were attributed to retinal abnormalities that include edema, vascular changes, and detachment. More recently, case reports have emphasized cortical blindness, which is characterized by intact pupillary light reflexes and normal ophthalmoscopic findings. Importantly, techniques that have enhanced the resolution of neuroimaging studies have been revolutionized in the past two decades.

In 1980, Grimes et al.[7] reported the first case in which computed tomographic scanning was used to demonstrate reversible cortical lesions in a woman with preeclampsia and temporary blindness. Others have since reported low-density areas predominantly in the occipital lobes of women with blindness associated with severe preeclampsia or eclampsia. Radiologic findings have ranged from normal to documentation of widespread low-density areas. The latter are nonenhancing and have been attributed to localized areas of decreased perfusion associated with arterial spasm, infarction, or cerebral edema. We found that 7 of 11 women with pure cortical blindness in whom computed tomography was done had similar occipital lobe hypodensities. Others have reported partial resolution of these radiologic hypodensities by 3 to 5 days, with complete resolution within 14 days.[7-12] We observed a similar time course for their resolution and observed that clinical recovery precedes normalization of x-ray findings.

There is other evidence that these lesions are induced by vascular changes. Using Doppler velocimetry, Williams and McLean[13] showed that cerebral blood flow velocity is increased in pregnancy-induced hypertension, suggesting an increased resistance to flow. Velocity increased even more in the immediate puerperium. Thus the nature and duration of these reversible focal neurologic lesions and transient cortical blindness are in concert with the characteristic reversible pathophysiologic changes of preeclampsia-eclampsia.

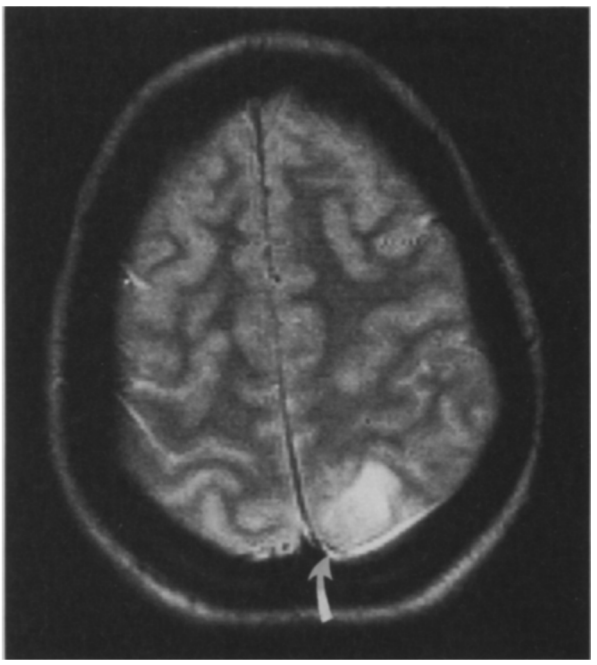

Fig. 2. Magnetic resonance scan showing corresponding high-signal lesion *(arrow)* in left occipital lobe of patient 8 from Table I.

As it has become more readily available, magnetic resonance imaging has been used to study cerebral lesions associated with preeclampsia. Duncan et al.[14] correlated high-signal magnetic resonance–imaged lesions (hyperintense signal lesions on T_2-weighted images and hypointense T_1-weighted images) with low-density lesions seen on x-ray tomography in 3 women with eclampsia and cortical blindness. Sanders et al.[15] corroborated these findings and demonstrated these subcortical lesions in the occipital lobes bilaterally. These high-signal lesions are consistent with those seen in transient ischemic lesions and indicate focal cerebral edema, reflected by increased water content. As shown in Figs. 1 and 2, in 2 of the 4 women with pure cortical blindness described here who underwent magnetic resonance imaging these corresponding high-signal lesions were demonstrated.

To correlate these clinical and radiologic abnormalities with pathologic findings, we compared them with the neuropathologic studies of Sheehan and Lynch.[16] In their large autopsy series of eclamptic women, they reported that 60% who died with 2 days of convulsions had evidence of cerebral hemorrhages or "softenings."

Table II. Cases of blindness associated with preeclampsia-eclampsia described in literature

*Source**	*Case No.*	*Clinical diagnosis*	*Cause*
Sommerville-Lange, 1950	1	Preeclampsia	Bilateral retinal detachment, transverse optic neuritis
Carpenter, 1953	1	Preeclampsia	Central retinal artery occlusion
Gandhi, 1978	1	Preeclampsia	Retinal vasospasm and edema
Grimes, 1980	1	Preeclampsia	Cortical
Beck, 1980	1	Eclampsia	Ischemic optic neuropathy
Singh, 1980	1	Eclampsia	Cortical
	2	Eclampsia	Cortical
Sibai, 1981	1	Eclampsia	Cortical
Beeson, 1982	1	Eclampsia	Cortical
McNamee, 1982	1	Eclampsia	Cortical
Nishimura, 1982	1	Preeclampsia	Cortical
	2	Eclampsia	Cortical
Goodlin, 1983	1	Preeclampsia	Cortical
Liebowitz, 1984	1	Eclampsia	Cortical
Hill, 1985	1	Preeclampsia	Cortical
Moodley, 1985	1	Eclampsia	Bilateral retinal detachment, cortical
	2	Eclampsia	Cortical
Arulkumaran, 1985	1	Preeclampsia	Cortical
	2	Preeclampsia	Cortical
Hauswald, 1987	1	Eclampsia	Cortical
Levavi, 1987	1	Eclampsia	Cortical
Lau, 1987	1	Preeclampsia	Cortical
	2	Preeclampsia	Cortical
Chew, 1988	1	Preeclampsia	Cortical
	2	Preeclampsia	Cortical
	3	Preeclampsia	Cortical
	4	Preeclampsia	Cortical
Seaward, 1989	1	Hypertension	Cortical
	2	Preeclampsia	Cortical
	3	Preeclampsia	Cortical
	4	Eclampsia	Cortical
Duncan, 1989	1	Eclampsia	Cortical
	2	Eclampsia	Cortical
	3	Eclampsia	Cortical

*A complete list of references is available from the authors on request.

These areas of hemorrhage were less evident with longer survival. The principal postmortem lesions found were cortical petechial hemorrhages, occurring most commonly in the occipital lobes. The characteristic lesions were focal groups of petechiae arranged in streaks that were radially aligned in the cortex. Histologically, these lesions were composed of numerous small hemorrhages 0.3 to 1.0 mm in diameter, capillary stasis and thromboses, and microinfarcts 0.3 to 1.0 mm in diameter. The hypodense areas seen in the occipital lobes with tomography and the corresponding high-signal lesions seen by magnetic resonance imaging are similar in configuration and distribution to these cortical petechiae. Similar pathologic findings have been correlated with those of severe hypertensive encephalopathy in nonpregnant patients in whom hemorrhage is a result of prolonged cerebral ischemia.[4, 17]

Another explanation for occipital blindness was pro-

Computed tomography	*Magnetic resonance imaging*	*Resolution*
None	None	Permanent blindness
None	None	Permanent blindness
None	None	Normal vision, 5 days
Day 0: Symmetric occipital low-density areas and left occipitoparietal region Day 5: partial resolution	None	Improved, 24 hr
Normal	None	3 days
None	None	4 days
Normal	None	4 days
Not reported	Not reported	Not reported
Day 0: Multiple low-density areas, more prominent in occipital lobes Day 14: Normal	None	24 hr
None	None	2 days
Normal	None	3 days
Normal	None	3 days
Day 0: Bilateral low-density areas in occipital cortex Day 4: Partial resolution	None	36 hr
Day 0: Basal ganglia and occipital lobe hypodensities Day 3: Partial resolution	None	6 hr
Day 0: Multiple hypodensity areas in white matter of occipital lobes, posterior parietal lobes, and posterior limb of right internal capsule Day 14: Normal	None	54 hr
Day 0: Diffuse occipital edema, slight narrowing of lateral ventricles Day 14: Normal	None	3 days
Bilateral occipital lobe edema	None	36 hr
None	None	5 hr
None	None	4 hr post partum
None	None	"Several days"
None	None	10 hr post partum
Day 0: Low-density areas in occipital lobes, wedge-shaped hypodensities in posterior temporal regions, slight narrowing of ventricles	None	3 days
Day 0: Bilateral low-density areas in occipital lobes	None	3 days
None	None	24 hr
None	None	24 hr
None	None	6 hr post partum
None	None	6 hr post partum
Normal	None	7 days
Normal	None	3 days
Normal	None	Within 8 days
Day 10: Left occipital cortex infarct	None	17 days
Day 0: Normal	Day 3: Hyperintense lesions (T_2-weighted images) in both occipital lobes	24 hr
Day 0: Bilateral occipital low-density lesions	Day 0: Hyperintense lesions (T_2-weighted images) in both occipital lobes and temporal cortex	36 hr
Day 0: Normal	Day 3: Hyperintense signal lesion in right occipital lobe	3 days

*A complete list of references is available from the authors on request.

posed by Schwartz et al.[3] in their report on hypertensive encephalopathy. They cite two possible mechanisms by which normal cerebral autoregulation may be overcome by severe hypertension. The first, called *overregulation,* proposes that severe cerebral vasoconstriction leads to the lesions seen. The second theory, called *breakthrough,* proposes that the loss of cerebral vascular regulation with resultant dilatation leads to focal vasogenic edema. Importantly, they cite a study to show that sympathetic innervation of the cerebral vasculature is differential. This study shows that the anterior circulation is well innervated whereas the posterior circulation is less so.[18] These observations can be used to explain the preferential predilection of symptoms and neuroimaging findings concentrated in the posterior circulation. In either case these radiologic abnormali-

ties localized to the occipital lobe appear to reflect petechial hemorrhages with accompanying focal edema and account for the clinical manifestations of scotomas and cortical blindness in women with pregnancy-induced hypertension.

The management guidelines are straightforward for women with severe preeclampsia or eclampsia in whom cortical blindness develops. Generally, they are the same as for women without this sequela and include magnesium sulfate for seizure prophylaxis, control of severe hypertension, and fluid restriction to attempt to avoid worsening of cerebral edema. Ophthalmologic and neurologic consultation, along with neuroimaging, is undertaken; however, delivery should not be delayed unnecessarily.

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