Transient Blindness in a Preeclamptic Patient Secondary to Cerebral Edema

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Blindness associated with a cerebral lesion has been described as cortical blindness. This is the first reported case in which computerized tomography has documented cerebral edema to be the cause of cortical blindness in a preeclamptic patient.

Blindness associated with preeclampsia is rare and, in most cases, is a result of ophthalmological changes such as retinal edema, retinal detachment or central retinal artery thrombosis. Grimes et al[1] described the first case of cortical blindness in a preeclamptic patient based on a normal ophthalmology examination, and the presence of bilateral cerebral lesions in the occipitoparietal cortex demonstrated by computed tomographic scan. Due to limitations in the CT scanning equipment of that generation, the lesions were thought to represent either cerebral edema or hemorrhage. Using improved CT scanning equipment, we determined that the lesions in the occipitoparietal cortex in our patient with preeclampsia and transient blindness were found to be from cerebral edema.

Case Report

A 34-year-old primiparous Chinese woman was admitted to the hospital at 32 weeks of gestation with a diagnosis of preeclampsia. She presented with a blood pressure of 155/100 mmHg and 3+ proteinuria. Her highest blood pressure during her prenatal visits was 110/70 mmHg, and urine was negative for protein at every visit. Examination of her eyes revealed normal disks, maculae, and vessels. Her neurologic examination was normal, and there was no evidence of hyperreflexia. Admission hemoglobin was 12.3 gm/100 ml, uric acid 5.5 mg/100 ml, platelet count 197,000 per mm3, creatinine 0.7 mg/100 ml, blood urea nitrogen 9 mg/100 ml, and serum glutamic oxaloacetic transaminase 21 mg/100 ml. A 24-hour urine examination revealed proteinuria of 8 gm. On bedrest, her blood pressure returned to 120/80 mmHg.

The second day of admission she began to complain of headaches and her vision acuity was reduced to light perception only. Her blood pressure had risen to 130/90 and she was infused with 4 gm of magnesium sulfate over 20 minutes, followed by continous infusion of 2 gm per hour.

Both pupils were normal in diameter and reacted fully to light. A repeat fundoscopic examination revealed normal vessels, disks, and maculae in both eyes. Her blood pressure rose to 170/105 mmHg and a cesarean section was performed under general anesthesia. A female infant weighing 1338 gm was delivered with Apgar scores of 7 and 9.

A CT scan using a General Electric Advantage Highlight System was obtained following the cesarean section and showed hypodensity in both occipitoparietal lobes. This was diagnosed as cerebral edema (Fig. 1). Magnesium sulfate was continued for the next 48 hours at 2 gm per hour, and her blood pressure fell to 130/80 mmHg. Her vision returned to normal over the next 24 hours although she developed a problem with depth perception. Her vision was completely normal by the third postoperative day, and she was discharged on the fourth postoperative day. A repeat CT scan 6 weeks after delivery was normal (Fig. 2). A neurologic and ophthalmologic examination 6 months following delivery was normal.

Discussion

Antepartum transient blindness with preeclampsia in the presence of normal ophthalmologic findings has been reported in three patients prior to this case (Table 1). Although Grimes et al[1] postulated the etiology as a cerebral lesion by demonstrating hypodensity of the occipitoparietal region of the cortex on CT scan, Nishimura et al[2] failed to demonstrate any abnormalities on CT scan. Arulkuraran et al[3] found the CT scan to be normal in the most recent reported case of cortical blindness. In our case, the CT scan clearly demonstrated hypodensities of the occipitoparietal region of the cortex confirming the etiology of cortical blindness as a cerebral lesion.

Grimes et al[1] postulated that the observed lesions represented cerebral edema or cerebral vascular accident. Beeson et al[4] said cerebral edema was responsible for the findings of hypodensity in the occipital lobes in an eclamptic patient with cortical blindness since these lesions caused symmetric compression of the lateral ventricles and resolved with diuresis. A more recent report by Brown et al[5] suggested that the areas of hypodensity represented petechial hemorrhages accompanied by cerebral edema. Using improved equipment, the CT scan in this case clearly demonstrated that the hypodensities in the occipitoparietal region of the cerebral cortex was due to cerebral edema rather than hemorrhage.

Cortical blindness associated with preeclampsia or eclampsia has

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Table 1.—Reported Cases of Antepartum Cortical Blindness in Preeclampisa

Author	Year	Gestational age (wk)	Preclampsia	Computed Tomographic Scan	Resolution of Blindness Following Delivery
Grimes et al[1]	1980	41	Severe	Occipitoparietal lesion	24 hours
Nishimura et al[2]	1982	36	Mild	Normal	72 hours
Arulkumaran et al[3]	1985	36	Severe	Normal	4 hours
Shieh et al	1995	32	Severe	Ocipitoparietal Cerebral Edema	24 hours

Fig. 1.—A CT scan showing cerebral edema in both occipitoparietal lobes of the cerebral cortex with the right more than the left (arrow).

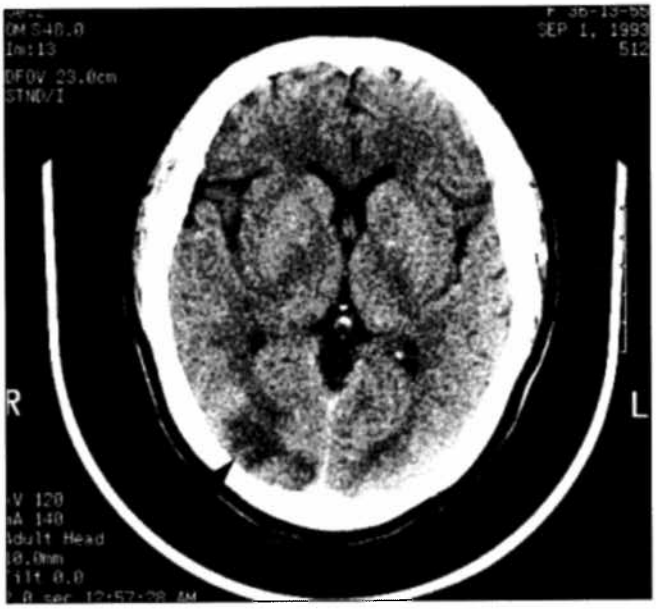
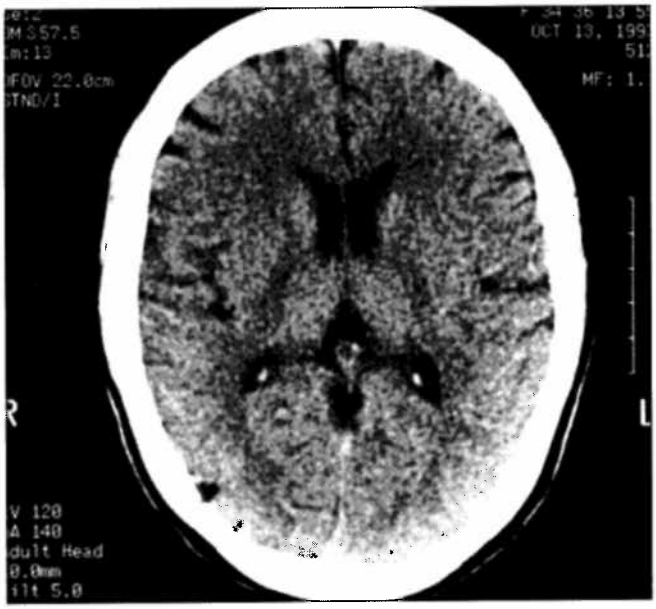


Fig. 2.—Normal CT scan 6 weeks following delivery.



been reported in 12 patients in the English literature. Seven of these patients developed blindness prior to delivery[1-4, 6-7] and four following delivery.[3,8] Blindness occurred in seven patients with preeclampsia[1-4, 6-7] and in five eclamptic patients.[2,4, 6-8] Every case of cortical blindness resolved spontaneously. The results from the CT scan of our patient with cortical blindness corroborates the CT results reported by Grimes et al,[1] who first postulated the etiology as a cerebral lesion. It further confirms the work of Beeson et al,[4] who suggested that the lesion represented cerebral edema.

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