Cerebral Vasospasm and Eclampsia

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We describe a patient who experienced focal cerebral and brainstem ischemia in the setting of postpartum eclampsia. Cerebral angiography showed spasm of large- and medium-caliber arteries. This case provides rare documentation that vasospasm may account for cerebral ischemia in eclamptic women with focal signs. This observation suggests that in such patients cerebral angiography may be informative and useful. (Stroke 1988;19:326-329)

The neurologic manifestations of preeclampsia-eclampsia include headache, visual disturbances including cortical blindness, generalized seizures, depressed alertness, and coma.1,2 Focal neurologic abnormalities are rare and, in the absence of parenchymal hemorrhage, their mechanism is uncertain.3 We describe a patient with focal deficits in the context of eclampsia in whom we documented spasm of large- and medium-caliber cerebral vessels. This angiographic finding provides rare documentation that in such eclamptic patients vasospasm may be a mechanism of ischemic injury.

Case Report

A 27-year-old woman, gravida one (G1), para zero (P0) in the 42nd week of gestation by dates, was admitted shortly after the onset of irregular contractions. Her blood pressure on admission was 120/70 mm Hg. She had 2+ pitting edema, mild hyperreflexia, and 2+ proteinuria and was without complaint of headache, visual disturbance, or epigastric pain. Pitocin was administered because of minimal progress of labor. She had a normal spontaneous vaginal delivery 12 hours after admission. Fifteen minutes postpartum, she complained of headache, nausea, and epigastric pain. Blood pressure was 172/88 mm Hg. She had 2+ pitting edema, mild hyperreflexia, and 2+ proteinuria and was without complaint of headache, visual disturbance, or epigastric pain. Pitocin was administered because of minimal progress of labor. She had a normal spontaneous vaginal delivery 12 hours after admission. Fifteen minutes postpartum, she complained of headache, nausea, and epigastric pain. Blood pressure was 172/88 mm Hg. Magnesium sulfate was begun promptly with a loading dose of 4 mg i.v. followed by an infusion of 1.5 mg/hr, which was later increased to 2 mg/hr. Blood pressure initially rose to 190/102 mm Hg and then remained in the range of 160-170/85-90 mm Hg. The following morning, she was found stuporous. Blood pressure was 140/80 mm Hg; respirations were regular. She made no response to verbal stimuli but had purposeful spontaneous movements. No papilledema was evident. The pupils were 2 mm bilaterally and reacted to light. Left lateral gaze paralysis was noted with the oculocephalic (doll’s head) maneuver and persisted after ice water caloric stimulation. Muscle stretch reflexes were slightly brisk and symmetric. The plantar responses were flexor. Magnesium sulfate was discontinued. A magnesium level was 5 (therapeutic range 3–6) meq/l. A cranial computed tomogram (CT scan) obtained urgently showed no intracranial hemorrhage. She regained awareness over the following 24–48 hours. A repeat CT scan on the second postpartum day showed evidence of radioluency in the midbrain and upper pons. Her blood pressure remained elevated (140/80–160/100 mm Hg) until the eighth postpartum day, when it returned spontaneously to normal (130/70 mm Hg). A cerebral angiogram obtained on the fourth postpartum day and the CT scan performed immediately following are described below. She complained at that time of diplopia on left lateral gaze. Her examination showed adductor lag on left gaze and vertical nystagmus on upgaze. Blood pressure at the start of the angiogram was 159/98 mm Hg. Her hemoglobin was remarkable for thrombocytopenia (lowest platelet count 54,000 on the second postpartum day, with return to 315,000 by the fifth day). Serum hepatic enzymes (in IU/l) were elevated as follows: lactate dehydrogenase 1,045 (normal 0–200), serum glutamicoxaloacetic transaminase 445 (normal 0–41), and alkaline phosphatase 264 (normal 0–115). Coagulation profile included a hematocrit of 38.3, prothrombin time of 12.0 (control 12.1) seconds, partial thromboplastin time of 32 (control 30) seconds, fibrinogen of 295 (normal 200–400), and fibrin split products of 10 (normal 0–9). She was discharged on the ninth postpartum day, normotensive and with a normal neurologic examination. She remained normal 1 month later.

Results

Cerebral angiography on the fourth postpartum day showed diffuse vasospasm. The left vertebral injection (Figure 1) showed moderate spasm of the posterior cerebral arteries, with severe segmental narrowing of the right superior cerebellar branch and failure to fill the left superior cerebellar branch. Bilateral carotid injections showed beading in multiple branches of the anterior and middle cerebral arteries, with almost complete occlusion of some of the terminal branches (Figure 2). There was no evidence of atherothrombosis, aneurysm, or vascular malformation.

CT scan on the fourth postpartum day showed discrete areas of low density in the brainstem, thalamus, and internal capsules bilaterally. Neither hemorrhage nor contrast enhancement was seen (Figure 3).

Discussion

This patient had postpartum eclampsia as indicated by headache, epigastric pain, hypertension, edema,
hyperreflexia, and depressed alertness. The elevation of her blood pressure was rapid, sustained, and well in excess of any of the stated criteria for pregnancy-induced hypertension. Laboratory data supporting the diagnosis of eclampsia included proteinuria, hepatocellular dysfunction, and thrombocytopenia. Her course was complicated by the appearance of focal neurologic abnormalities (lateral gaze paralysis, vertical nystagmus). Such focal signs in eclampsia suggest underlying hemorrhage or ischemia. Cranial CT scan demonstrated midbrain and pontine lucencies that were interpreted as ischemic changes. In our investigation into the pathophysiologic mechanism for her cerebral ischemia, we documented the first case of which we are aware of angiographically proven cerebral vasospasm in eclampsia.

The pathogenesis of the neurologic manifestations of eclampsia is poorly understood. Many authors state that eclampsia induces an acute hypertensive encephalopathy. Insight into this unsettled issue may
FIGURE 3. Cranial computed tomogram obtained following cerebral angiography on fourth postpartum day. Diffuse hypodensity is seen throughout brainstem, thalamus, and internal capsules bilaterally. There is no evidence of abnormal intracranial enhancement or acute hemorrhage.

derive from the special features of this case. In hypertensive encephalopathy, as in eclampsia, focal neurologic findings are rare. A prospective study of the neurologic manifestations of malignant hypertension led Heaton et al to conclude that the presence of focal signs indicates a second diagnosis (e.g., stroke) in addition to hypertensive encephalopathy. Similarly, the pathologic studies of Sheehan and Lynch revealed that in eclampsia focal signs are most commonly due to cerebral hemorrhage and less commonly due to ischemic infarction, with sites of predilection in the basal ganglia, midbrain, and pons.

Reports of patients with eclampsia can now be found in which cranial CT demonstrates areas of low density in the cortex and basal ganglia. The lack of angiographic data in these cases precludes an understanding of the vascular mechanism. There has been a tendency to speculate that spasm of large cerebral arteries or vasoconstriction with forced vasodilatation of cerebral arterioles is the underlying mechanism. Cross et al are alone in presenting a series of patients who had ischemic infarcts associated with pregnancy and the puerperium who were then studied by angiography or at necropsy. Of their 31 patients with hemiplegia of varying degree, five had "pre-eclamptic toxemia with fluctuating hypertension." The nature of the arterial lesion, if any, in this subset was not specified.

Cerebral angiography in our patient on the fourth postpartum day demonstrated diffuse vasospasm of large- and medium-caliber cerebral vessels. The angiogram revealed severe segmental narrowing of larger branch arteries and beading of smaller peripheral arteries with occlusions of some terminal branches. We could not justify a repeat cerebral angiogram since her neurologic signs resolved over the following days. Although we conclude that the vasospasm of intracerebral arteries seen in our patient was related to her eclampsia, we recognize that her angiographic findings could be compatible with vasospasm due to subarachnoid hemorrhage or arterial wall changes due to cerebral aneurysm. As to the former, subarachnoid hemorrhage can be excluded only by timely lumbar puncture. Recognizing that we cannot definitely exclude the possibility of hemorrhage, the evidence as a whole is persuasive that subarachnoid hemorrhage did not occur. Clinically, meningismus was absent. On serial cranial CT scans, no intracranial blood was apparent, and extensive vasospasm has been correlated with the quantity of blood on CT scan. On four-vessel angiography, no aneurysm or vascular malformation was demonstrated. Finally, an alternative diagnosis (that is, eclampsia) had both clinical and laboratory corroborations whereas little of her syndrome or laboratory data could be accounted for by subarachnoid hemorrhage. As to the latter, we recognize that angiographic vasculitis confined to the central nervous system cannot be excluded with absolute certainty. However, isolated angitis of the brain is rare, usually affects an older age group, and most often proves grave or fatal, not rapidly reversible and benign.

Our patient made a gratifying recovery without treatment of her hypertension. Initially, her systolic blood pressure rose >70 mm Hg above her baseline value and, at angiography, her diastolic blood pressure was still elevated by 30 mm Hg above her baseline. The level of her rapid and sustained hypertension well exceeded any of the criteria for the diagnosis of eclampsia but did not approach the malignant levels that are characteristic of hypertensive encephalopathy. Her neurologic symptoms and signs cleared well before her blood pressure returned to normal. These observations suggest that her neurologic symptoms did not result exclusively from hypertensive encephalopathy but do not confute a commonality of mechanism. We do not know if her moderately elevated systemic blood pressure afforded her some measure of protection from further ischemia due to vasospasm. We do know that she tolerated angiography well and that a specific vasculopathy was revealed.

References

**KEY WORDS**
- cerebral ischemia
- cerebral vasospasm
- eclampsia
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