Postpartum Cerebral Angiopathy With Cerebral Infarction Due to Ergonovine Use

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Background and Purpose: Earlier cases of stroke due to postpartum cerebral angiopathy have been reported. The mechanism of this angiopathy has not been explained.

Case Description: We present a case of cerebral infarction with evidence of occlusive change in the vertebrobasilar system as a complication of intravenous ergonovine use after cesarean section delivery.

Conclusions: Ergonovine therapy is a likely cause of so-called postpartum cerebral angiopathy and stroke. (Stroke 1992;23:1364–1366)

Key Words • cerebral infarction • cerebral vasospasm • vertebrobasilar circulation • women

Ergonovine is an ergot alkaloid derivate commonly used during delivery labor. Several drugs, such as methylergonovine and bromocriptine, are related to ergonovine. Myocardial infarction, arterial hypertension, and stroke have been reported as complications of the use of ergonovine and bromocriptine. A few cases of stroke due to ergonovine use have been noted, including one case of intracerebral hemorrhage. We report a case of occlusive change in the vertebrobasilar arterial system secondary to the use of ergonovine after an uncomplicated cesarean section delivery, with arteriographic findings compatible with postpartum cerebral angiopathy.

Case Report

A 28-year-old woman was admitted for cesarean section delivery. She had no history of heart disease, migraine, arterial hypertension, or diabetes mellitus. There was no hypertension or proteinuria during pregnancy. Physical examination on admission revealed no abnormalities, and cesarean section was uncomplicated. Following delivery of the placenta, the patient received 0.2 mg i.v. ergonovine. After a few seconds, the patient developed sudden and severe headache with bilateral amaurosis and mild confusion. There was no elevation of blood pressure during the procedure.

The neurological examination showed a confused patient with normal ocular fundi. Both pupils were 5 mm and reacted slowly to light. Extraocular movements were full. There was bilateral amaurosis. Motor function was normal. There was a right hemisensory deficit to all modalities.

Brain computed tomographic scan performed at 20 hours of evolution showed a hypodense area in the left occipital lobe. Magnetic resonance imaging performed on day 4 showed bilateral temporo-occipital infarction, more prominent on the left, and left thalamic infarction (Figure 1). Cerebral arteriography showed marked narrowing of the distal basilar artery, with irregular narrowing of the right posterior inferior cerebellar artery and the middle and distal thirds of the basilär artery (Figure 2, left panel). There was no filling of the posterior cerebral arteries (Figure 3, left panel).

Various blood studies (including those for hematologic antithrombin III, proteins C and S, immunologic rheumatoid factor, lupus erythematosus preparation, antinuclear antibodies, erythrosedimentation rate, and C3-C4 concentrations) were normal. Results of electrocardiography and bidimensional and contrast echocardiography were normal. Treatment with oral nimodipine and aspirin was started.

Three months later, a second cerebral arteriogram was performed that showed normalization of the caliber of the right posteroinferior cerebellar artery, a lesser degree of narrowing of distal third of the basilar artery (Figure 2, right panel), and a partial filling of the left posterior cerebral artery (right panels of Figures 2 and 3).

Discussion

The cardiovascular side effects of oxytocic drugs in obstetric patients have long been recognized. In 1949 Greene and Brachman reported a series of patients with arterial hypertension after use of vasopressors and an oxytocic agent; one patient developed hemiplegia. The ergot derivates have been documented as a cause of peripheral and visceral arteriopathies. Cerebral vascular complications are rarely reported, most often related to excessive doses. The adverse effects produced by ergonovine are more severe when the drug is given by the intravenous route. Intravenous injection of ergonovine has been associated with transient hypertension, seizures, and retinal detachment. Other vascular side effects include coronary artery spasm and myocardial infarction as well as intracerebral hemorrhage.

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FIGURE 1. Axial T2-weighted (repetition time, 2,160 msec; echo time, 90 msec) magnetic resonance imaging scan showing high-intensity signal in area corresponding to the distribution of the posterior cerebral arteries bilaterally, indicating extensive infarction, including the left thalamic region. Findings are consistent with acute ischemic stroke.

FIGURE 2. Left panel: Left vertebral arteriogram, anteroposterior view, showing irregular narrowing of right posteroinferior cerebellar artery (PICA; small arrow) and both narrowing and dilatation of the distal third of the basilar artery (large arrow). Right panel: Right vertebral arteriogram, anteroposterior view, performed 3 months later, showing a normal right PICA (small arrow), narrowing in the distal third of the basilar artery (large arrow), and partial filling of the left posterior cerebral artery (double arrow).

FIGURE 3. Left panel: Vertebrobasilar arteriogram, lateral view, showing irregular narrowing of the distal third of the basilar artery (solid arrow) and no filling of the posterior cerebral artery (PCA; small arrow). Right panel: Vertebrobasilar arteriogram, lateral view, performed 3 months later, showing less narrowing of the distal basilar artery (double arrow) and partial filling of the PCA (single arrow).
To date, seven cases of cerebral arteriopathy associated with ergonovine use have been described. In three of the four cases of "postpartum cerebral angiopathy" reported by Rascol et al, ergonovine was administered, but the authors did not consider this relation. The three single cases reported by Dupuy et al, Henry et al, and Bogousslavsky et al were comparable with our case in respect to arteriographic findings. The temporal relation to drug administration was obvious in the case reported by Henry et al, in which neurological symptoms appeared 10 minutes after the administration of ergonovine. Ergonovine has a rapid onset of action (less than 40 seconds) when administered by the intravenous route.

In our case, other potential causes of stroke seem unlikely. There was no evidence of an embolic source of stroke. Spontaneous basilar artery dissection is a plausible alternative etiology, but this is a rare condition and is not associated with arteriographic beading. There are several reports of diffuse vasospasm associated with toxemia, but there was no clinical evidence of toxemia in this patient.

Ergonovine must be added to the list of drugs that can produce cerebral infarction and should be given with caution to patients with migraine, Raynaud's phenomenon, or other conditions that may predispose patients to vasospasm. Concomitant use of ergonovine and other drugs with similar actions (e.g., bromocriptine or ergotamine) conceivably may also increase the risk of cerebrovascular complications.

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