

Perioperative Stroke in the Brain and Spinal Cord Following an Induced Hypotension

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A 49-year-old woman presented with stupor and paraplegia following an induced hypotension. The temporal relationship to the induced hypotension and the absence of a clear embolic source on diagnostic tests support a causal association between the hypotensive episode and the ischemic infarct. However, despite the association, a cause-and-effect relationship could not be automatically inferred.

Key Words: Induced hypotension, anesthesia, stroke

Deliberate lowering of the arterial blood pressure during surgery has been used routinely in surgery for over fifty years in order to minimize intraoperative blood loss and decrease operating time.^{1,2} However, there have been a few reports of cerebral complications.^{3,4}

Here we report a case of infarcts of the brain and spinal cord following an induced hypotension.

CASE REPORT

A 49-year-old woman with a history of angina pectoris was scheduled for decompressive total laminectomy with an autologous iliac bone graft for spinal stenosis at segments L3-L4 and L4-L5. The patient had no prior history of hypertension, diabetes mellitus, hyperlipidemia or cerebral ischemic events. Four years previously, the patient had been diagnosed with stable angina pectoris

because of exertional chest discomfort, and she had been taking aspirin orally at a daily dosage of 100 mg. A preoperative evaluation revealed no neurologic deficit. Her height, weight, blood pressure (BP), and heart rate were 167 cm, 65 kg, 140/80 mmHg and 80 bpm (regular rate and rhythm), respectively. Preoperative laboratory tests, including transthoracic echocardiography (ejection fraction 68%) and 24 hours Holter monitoring, were normal.

General anesthesia was induced with 250 mg thiopental and 50 mg succinylcholine. The trachea was intubated and the lung ventilated with 60% nitrous oxide in oxygen. Anesthesia was maintained with 0.5-2.0 vol. % enflurane and a further increment of muscle relaxant. To induce hypotension, a mixture of 100 ml normal saline with 100 mg esmolol hydrochloride was administered. Five minutes after the injection, the patient's BP had fallen to 70/35 mmHg (MAP 47). Consequently, the hypotensive agent was discontinued and 500 ml of a 5% dextrose solution with 200 mg of dopamine was given. The hypotensive episode lasted for approximately 60 minutes and the arterial blood pressure averaged 80/50 during this period.

Twelve hours after the operation, the patient became stuporous and was unable to follow commands. Her pupils were equal and reacted to light. Corneal reflexes were present, and oculocephalic reflexes showed normal. She did not respond to deep pain.

One day after operation, her mental condition recovered and she complained of muscle weakness and a loss of sensation in both lower extremities. On examination, she was completely insensitive below the T6 level and displayed

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flaccid paraplegia. Deep tendon reflexes in the lower extremities were not recorded.

Laboratory studies including blood gas analysis, complete blood cell and platelet count, erythrocyte sedimentation rate, blood electrolytes, creatinine, liver enzymes, cholesterol, triglycerides, prothrombin and partial thromboplastin time, antithrombin III-protein C and protein S activity were all normal. In addition, autoantibodies screens were also normal. Spinal fluid examination showed no abnormality.

Diffusion-weighted brain MRI showed multifocal ischemic infarcts involving the posterior regions of both cerebellar hemispheres, both parieto-occipital lobes and both superior frontal cortices (Fig. 1). An MRI scan of the thoracic spine 5 days after the onset of symptoms showed a diffuse ischemic infarct in the spinal cord segment T5-L1 (Fig. 2). Magnetic resonance angiograms of the brain and thoracic and abdominal aorta were normal. Neither the electrocardiogram nor transesophageal echocardiography revealed any cardiac abnormalities. In addition, both the carotid duplex and transcranial Doppler sonograms also showed no abnormalities.

DISCUSSION

The incidence of perioperative stroke is less than 1% in the general surgical population.^{5,6} The majority have a thromboembolic origin and occur commonly during the postoperative period.⁵ Risk factors for developing perioperative stroke include previous stroke history, hypertension, diabetes, smoking, obesity, and cardiac and peripheral vascular disease.⁵⁻⁹ Hypotension, especially in the postoperative period, has also been identified as a risk factor for perioperative stroke.^{9,10} However, intraoperative induced hypotension as the cause of perioperative stroke is rare and has not been established.^{3,9,11}

Our patient developed symptoms of diffuse cerebral dysfunction and paraplegia one day after a hypotensive episode during general anesthesia. Although a causal association between the hypotensive episode and the infarcts is uncertain, it is unlikely that other etiologies were responsible. Our patient's only risk factor for ischemia was a

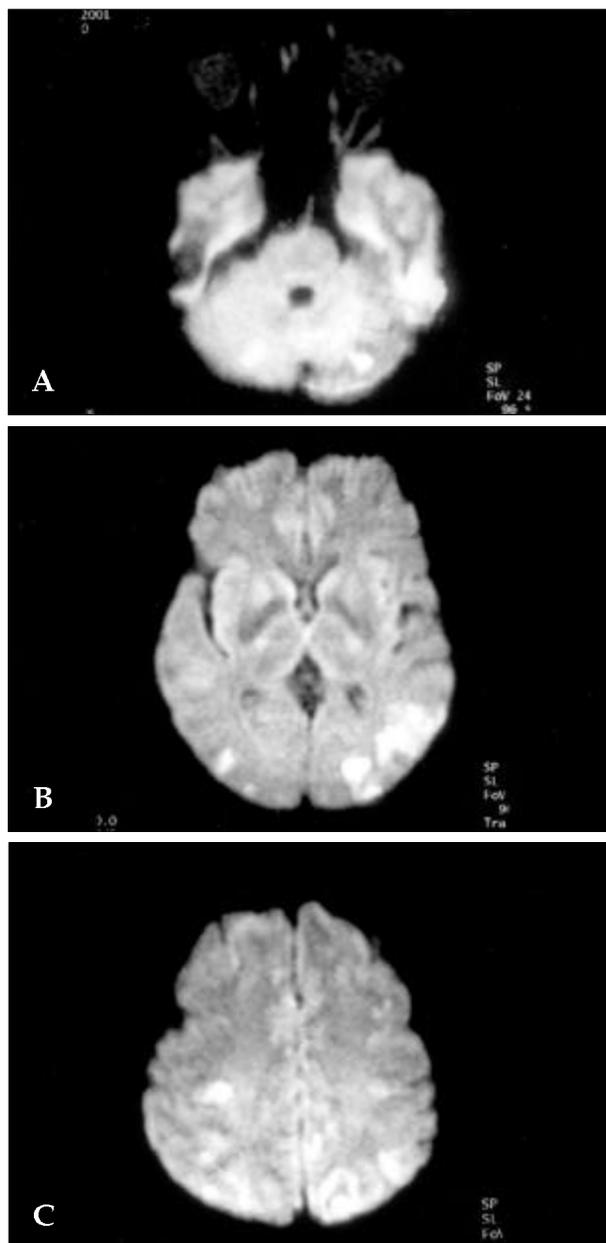


Fig. 1. Diffusion-weighted brain MRI obtained one day after the operation show multifocal ischemic infarcts involving the posterior regions of both cerebellar hemispheres (A), both parieto-occipital lobes (B) and both superior frontal cortices (C).

past history of angina pectoris. However, laboratory evidence was not documented in either the electrocardiogram or the echocardiogram. The patient showed no evidence of cardiac arrhythmia or diabetes mellitus. The temporal relationship to the induced hypotension and the absence of a clear embolic source on the diagnostic tests



Fig. 2. A sagittal MRI scan of the thoracic spine showing a diffuse ischemic infarct in the spinal cord segment T5-L1.

(preoperative transthoracic echocardiography and postoperative transesophageal echocardiography, electrocardiography and 24 hours Holter monitoring, carotid duplex sonogram, transcranial Doppler sonogram, brain magnetic resonance angiogram, and aorta magnetic resonance angiogram) support a causal association between the hypotensive episode and the multiple cerebral infarcts and an ischemia in the spinal cord.

It is of course possible that the ischemic stroke in this patient may have been unrelated to perioperative hypotension. Another possible source of infarcts is an embolic focus. As previously reported,¹² fibrocartilaginous embolism cannot be ruled out in relation to this condition. In addition, the possibility that the embolic foci were not detected should be considered because laboratory evaluations searching for embolic foci had been performed during the two week period after

operation.

Therefore, we think that a profound, induced hypotension can result in multiple infarcts; however, a cause-and-effect relationship usually cannot be established with certainty. It is of course possible that an embolic phenomenon might be one of the proposed etiologies. The indication for hypotensive anesthesia should be applied more rigorously by pre-anesthetic evaluation, and the adequate intra- and post-operative monitoring should be requested in patients with risk factors for perioperative stroke.

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