Treatment of postangiographic hemiplegia with vasopressors

Case report

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Postangiographic hemiplegia can be successfully treated with vasopressors. A case is presented and the method of treatment discussed and analyzed.

Key Words • angiography • hemiplegia • vasopressor

The creation of a neurological deficit is a feared complication of cerebral angiography. This may be related to emboli from the catheter, direct damage to the cannulated artery, or the contrast medium which may cause changes in vasomotor regulation, clumping and agglutination of red blood cells, changes in blood flow, and disturbances in enzyme function.

Hemiparesis following angiography may persist despite maintenance of normal blood pressure. However, if the blood pressure is elevated by the use of vasopressors, neurological function can be restored. We describe here the successful use of hypertension to reverse a neurological deficit.

Case Report

This 50-year-old man was admitted complaining of dizziness when he stood up quickly, and grand mal seizures which had first appeared 6 months previously.

Examination. The blood pressure was 120/90 mm Hg and the pulse 84 and regular. General physical and neurological examinations were within normal limits except for a right Babinski sign. The electrocardiogram was interpreted as normal. The electroencephalogram was normal awake and asleep.

Angiography. A left carotid angiogram was performed under local anesthesia with preoperative sedation of Valium 10 mg, Demerol 100 mg, and atropine 0.4 mg, given 1 hour before the procedure. We injected 10 ml of sodium methylglucamine diatrizoate (Renografin 60) by hand. A series of anteroposterior films were taken, and the patient suffered no ill effects. A second injection was given for a series of lateral films; the patient immediately became aphasic and hemiplegic on the right side. The angiogram was normal. The carotid pulses remained full, equal, and without bruits. The blood pressure was 90/60 mm Hg and the apical pulse 54 and regular.
Intravenous metaraminal bitartrate (Levorphan) was started approximately 4 minutes after the onset of the complications. The blood pressure rose to 160/110 mm Hg, and the pulse to 84. The patient fully recovered from the right hemiplegia and aphasia within 30 seconds.

Through careful monitoring, the therapeutic range of blood pressure in this patient was found to be between 160/110 and 170/120 mm Hg. A blood pressure above 170/120 mm Hg brought on severe headaches, nausea, and flushing. If the pressure dipped below 160/110 mm Hg, symptoms of cerebral ischemia reappeared; if it fell to 155 mm Hg systolic, the patient became confused. Below 140 mm Hg, the right arm and leg became weak, and the patient became aphasic. At 130 mm Hg systolic, marked hemiplegia was evident. On 12 occasions, the blood pressure fell below 160/110 mm Hg, and each time this happened, the deficit corresponded in severity to the drop in blood pressure; each time the blood pressure was elevated to 160/110, there was virtually instantaneous and complete recovery.

Twelve hours after the angiogram, it was possible to lower the blood pressure slightly without any symptoms of cerebral anoxia. Over the next 6 hours, the medication was gradually reduced so that the patient was able to tolerate his usual blood pressure of 120/90 mm Hg, without any neurological abnormalities. Except for the initial bradycardia, the pulse rate ranged between 78 and 84.

Postangiographic Course. The patient’s recovery was uneventful. A brain scan taken 2 days later was normal. At a follow-up examination 3 months after angiography the patient was completely normal neurologically. He still had occasional dizziness when he stood up quickly, but had no seizures. He had complete amnesia for the postangiogram episodes of hemiplegia and aphasia. The EEG was normal awake and asleep.

Discussion

The exact cause of this patient’s neurological deficit is not known. The slight drop in blood pressure and heart rate caused by preoperative sedation, the contrast medium, or emboli from the tip of the cannula were considered the most likely causes that led to a marginal lowering of cerebral blood flow so that the neurons in the involved area became dysfunctional but not necrotic. Elevation of the blood pressure is thought to have increased flow to the hypoxic area, thus preventing infarction and restoring function.

Autoregulation is known to be lost for as long as 2 days after ischemia; it then gradually returns. This could explain why hypertensive therapy was needed for only 48 hours.

Others have reported similar experience with vasopressor therapy. Farhat and Schneider noted successful alleviation of neurological deficits in four cases, two of which were postangiographic. One occurred in a patient who had stenosis of the intracranial portion of the internal carotid artery, and another in a patient with a subarachnoid hemorrhage and spasm of the internal carotid artery. The other two cases involved intracranial surgery. One patient awoke from surgery with hemiplegia after undergoing a craniotomy for removal of a chromophobe adenoma, wherein the tumor was dissected from the internal carotid artery. The other patient had surgery for an aneurysm of the internal carotid artery at the posterior communicating artery, which was trapped intracranially; vasopressors were instituted immediately. She had no postoperative deficit until the next day, when the blood pressure was lowered; this deficit was alleviated when the pressure was again elevated. All patients in that series required elevated blood pressures for 3 days.

Wise reported two cases in which postangiographic neurological deficits were reversed. One patient suffered left hemiplegia following an angiogram which revealed complete occlusion of the right internal carotid artery at the carotid bifurcation. He was successfully treated with vasopressors and was able to tolerate his usual blood pressure after 5 hours. The other patient developed right hemiplegia following an angiogram which revealed complete occlusion of the left common carotid artery and severe stenosis of the right internal carotid artery at the carotid bifurcation. Vasopressor therapy was successful in reversing the deficit, but the blood pressure had to be maintained at elevated levels until a right carotid endarterectomy could be performed 2 days later.

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VanderArk and Pomerantz\(^6\) reported a case in which, after an aneurysm had been clipped, the patient awoke with a deficit which was reversed when the blood pressure was elevated. But, in addition, the cardiac output had to be increased with atropine before the patient could tolerate his usual blood pressure without symptoms of cerebral ischemia.

It is significant to note that in these cases of neurological deficit following cerebral angiography, all of the patients had symptoms of cerebral ischemia, cerebrovascular disease, or cerebrovascular spasm. Also, in the cases involving intracranial surgery, the carotid artery had been manipulated in some way. This leads us to believe that patients with cerebrovascular disease who undergo cerebral angiography or intracranial surgery are the ones most likely to require the maintenance of their blood pressures at elevated levels, either during or immediately after the procedure.

We feel that hypertensive therapy for postangiographic deficits is a safe and highly effective method of restoring neurological function providing that close monitoring, preferably in the intensive care unit, is carried out. The therapeutic blood pressure must be individualized. From our experience and that of others, the blood pressure can be safely lowered to usual levels in approximately 24 to 48 hours. If this does not prove possible, other factors causing decreased cerebral blood flow must be investigated and corrected before hypertensive therapy can be safely discontinued.

References

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