Extravasation of angiographic contrast material in hypertensive intracerebral hemorrhage

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Leakage of contrast material during angiography is an uncommon phenomenon in hypertensive intracerebral hemorrhage. Intraparenchymal extravasation was demonstrated in five of 12 patients in whom serial carotid angiography was carried out within 5 hours of hemorrhage. Extravasated material, first noted in the arterial phase, grew in size and density until the early venous phase and was still visible on the last 10-sec film. Three of the four in whom the hematoma was evacuated surgically showed clinical improvement.

KEY WORDS: hypertensive intracerebral hemorrhage, extravasation of contrast material, cerebral angiography, surgical treatment of stroke

Case Reports

Case 1

A 69-year-old woman had a sudden onset of aphasia and loss of consciousness on October 28, 1970. On admission the systemic blood pressure was 180/110 mm Hg. The patient was comatose and displayed a right hemiplegia with Babinski sign. A left serial carotid angiogram demonstrated a medial shift of the lenticulostrate arteries and displacement of the anterior cerebral artery to the contralateral side. A collection of contrast medium, noted in the arterial phase, grew in size and density until early in the venous phase (Fig. 1) and still remained on the last film (10 sec). Operation 4 hours after the stroke demonstrated a large hemorrhage in the region of the basal ganglia with dissection into the left lateral ventricle; a hematoma 120 mL in size was evacuated. The patient regained consciousness 10 days after
M. Kowada, K. Yamaguchi, S. Matsuoka and Z. Ito

FIG. 1. Case 1. Left carotid angiogram, lateral view (3.5 sec), showing elevation of the branches of the middle cerebral artery and marked leakage of contrast material.

craniotomy and was discharged with motor aphasia and a right hemiparesis on March 1, 1971.

Case 2

A 37-year-old hypertensive man was admitted after the onset of speech disturbance and right hemiparesis on November 16, 1970. The blood pressure was 200/120 mm Hg, and there was a right hemiparesis with Babinski sign. A left serial carotid angiogram showed slight lateral shift of insular branches of the middle cerebral artery and medial displacement of the internal cerebral vein. An intraparenchymal leakage of contrast medium was revealed in the parietal region in the arterial phase at 3 sec. The contrast shadow increased in size and opacity in the venous phase (7 sec) (Fig. 2). The patient was discharged improved after operation on January 8, 1971.

Case 3

A 41-year-old hypertensive man developed sudden onset of right hemiparesis with subsequent progression to coma. When admitted on February 5, 1971, the blood pressure was 220/120 mm Hg, and the patient was slightly responsive to painful stimuli. Left serial carotid angiogram demonstrated a marked shift of the anterior cerebral artery to the right side and lateral bowing dislocation of the middle cerebral artery. Increased sweep of the pericallosal artery suggested ventricular enlargement. The leakage of contrast material in the parietal region was revealed in the arterial phase at 3 sec and was still distinct in the last film at 13 sec (Fig. 3). Evacuation of a hematoma was carried out 2 hours after onset. The operation demonstrated extensive hemorrhage involving the basal ganglia with dissection into the lateral ventricle; a hematoma 110 mL in size was removed. The patient is still in the hospital with impairment of consciousness 1 month after the operation.

Case 4

A 57-year-old hypertensive man entered the hospital after the sudden onset of weakness of the right arm and leg with impairment of consciousness in June, 1970. He was semicomatose on admission, with a systemic blood pressure of 160/110 mm Hg, and a right hemiparesis and Babinski sign. A left serial carotid angiogram revealed lateral displacement of insular branches of the middle cerebral artery, medial shift of the lenticulostriate artery, and contralateral displacement of the internal cerebral vein. Leakage of contrast medium was demonstrated close to the lenticulostriate artery in the arterial phase at 1.5 sec. A distinct contrast fleck was still noted on the last film at 10 sec. The patient was discharged improved after removal of a hematoma of 40 mL.

Discussion

Intraparenchymal extravasation in hypertensive intracerebral hemorrhage may be
Angiographic extravasation in intracerebral hemorrhage
demonstrated more frequently than expected when angiography is carried out a short time after onset. Cerebral angiography was done within 1 hour after the onset of hemorrhage in Cases 1 and 3, within 2 hours in Case 2, and within 5 hours in Case 4. Huckman, et al., did not report the interval between angiography and onset in their four cases. Kudo performed angiography twice after stroke in the same patient and noted more leakage at 8 hours than at 7 hours.

The initial leakage of contrast material appeared in the arterial phase within 1.5 to 5 sec. The shadow grew in size and density until the arterial delineation disappeared or venous filling began. Extravasated material diffused slowly at the marginal zone and was still clearly visible in the last film at 10 sec. Bergström and Lodin did not observe a diffuse contrast zone in the region of the basal ganglia. Fujino and Hara described an unusual transient appearance resembling extravasation; there was no such transitory phenomenon in our five cases.

The leakage of contrast medium seemed to differ in our cases. Multiple dissections were observed in Cases 1 and 2, and relatively isolated spots in Cases 3 and 4. This variation may be due to local differences in the necrotic brain tissues around which the leakage extends. It is not possible, however, to explain differences in the shape of the angiographic extravasation because pathogenesis of this phenomenon is not clear, even in cases of head injury. Before operation a very grave prognosis was indicated in our Cases 1 and 3 because of the large mass lesions dissecting from the basal ganglia into the lateral ventricle; the patient in Case 1 did not regain consciousness until 10 days after removal of the hematoma, but both recovered. Both of our other two patients improved after operation.

We have operated on 25 cases of hypertensive intracerebral hemorrhage in the last 22 months with an operative mortality of 8% within 1 month after evacuation. It is conceivable that the more expansive the hemorrhage, the more likely the possibility of demonstrating leakage of contrast medium shortly after onset of the hemorrhage, and early removal of the hematoma might save the patient's life even in the presence of extravasation.

References
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