Arteriographically Visualized Extravasation in Hypertensive Intracerebral Hemorrhage

REPORT OF SEVEN CASES

BY MASAHIRO MIZUKAMI, M.D.,* GORO ARAKI, M.D.,* HIROSHI MIHARA, M.D.,* TAKASHI TOMITA, M.D.,† AND RYOZO FUJINAGA, M.D.*

Abstract:
Arteriographically Visualized Extravasation in Hypertensive Intracerebral Hemorrhage

We advance the theory that continuous bleeding from the ruptured artery with mechanical destruction and displacement of cerebral tissue is the cause of massive hematoma formation, and discuss the possibility of surgical treatment of the acute stage of hypertensive intracerebral hemorrhage.

Additional Key Words
massive hematoma hypertension cerebral angiography

We have been performing cerebral angiography in the early stage of hypertensive intracerebral hemorrhage. Recently, extravasation of the contrast medium from the lateral lenticulostriate artery has been demonstrated in seven of our cases. Only a few cases have been reported briefly by others in which the bleeding could be visualized during the acute stage of hypertensive intracerebral hemorrhage; no detailed reports have been made previously.

The sequence of events observed in our cases illustrates a mechanism for massive hematoma formation, and elucidates possible surgical approaches to treatment of the acute stage.

We feel that continuous bleeding from the ruptured artery, with mechanical destruction and displacement of cerebral tissue, is the cause of massive hematoma formation in these patients. In support of this theory, we present the following case studies and analysis.

Case Reports

Case 1
A 54-year-old man, with known hypertension, suddenly became unconscious following lunch. Initial examination two hours later revealed semicoma, a right hemiplegia, and a blood pressure of 240/110 mm Hg.

Six hours after the onset, the patient was in coma, the extremities exhibited decerebrate rigidity to painful stimuli, the oculocephalic reflex was...
absent, and respiration was of the Cheyne-Stoke type.

Arteriography
Left carotid arteriography, utilizing 10 ml boluses of 60% Conray and hand injection with single exposures, was performed seven hours after the onset. In the A-P view of the arterial phase, the lateral lenticulostriate artery was slightly displaced medially and was corrugated with a spotty stain of Indian-bean size evident at its periphery (fig. 1A). Lateral view of the arterial phase taken five minutes later showed the lateral lenticulostriate artery displaced by pressure from the posterosuperior direction, with extravasation of the contrast medium at its periphery, spreading posteriorly (fig. 1B). A repeat study was carried out ten minutes later. Extravasation of the contrast medium was clearly shown in both the A-P and the lateral views of the capillary phase. A lateral view performed one hour later revealed further expansion of the extravasation, accompanied by the formation of a fluid level, due to the patient lying in the supine position (fig. 1C). The contrast medium was mixed with the hematoma, revealing the hematoma cavity as a light stain.

FIGURE 1A
The A-P view of Case 1 shows extravasation of contrast medium as an Indian-bean-sized spotty stain (arrow).

FIGURE 1B
The lateral view of Case 1 shows extravasation as a spotty stain (single arrow), and spreading of the contrast medium as a soy-bean-sized stain (double arrow).

FIGURE 1C
The lateral view of Case 1 shows expansion of the extravasation with the formation of a fluid level (arrows).
Operative findings
Osteoplastic craniotomy of the left pars temporalis was performed at ten hours after the onset. The brain was very swollen and the hematoma was found to extend 3-cm deep from cortex to lateral ventricle. The hematoma consisted mostly of clots which were evacuated almost totally. Marked bleeding in the area of the lateral lenticulostriate artery was noted, as well as bleeding from the wall of the hematoma, but no definite hemorrhaging vessel could be found. Bleeding was controlled, a Penrose drain was inserted into the hematoma cavity, and closure was accomplished.

Postoperative course
The patient remained in coma without improvement. The right pupil was dilated immediately following surgery and it was felt that uncal herniation had developed during surgery. The patient expired two hours postoperatively and 15 hours after the onset.

Autopsy
The brain was markedly swollen with an operative wound from the cortex of the left temporal lobe into the hematoma cavity. Hemorrhagic lesions were distributed as far as the lateral ventricle but centered around the left putamen. Since part of the thalamus remained, the hemorrhagic source was thought to be the lateral lenticulostriate artery which had shown extravasation on cerebral angiography. However, due to the destruction of the brain tissues around the hematoma by surgical manipulation, no ruptured vessel could be definitely identified.

CASE 2
A 49-year-old man, with known hypertension for several years, complained of headache and was noted to have right-sided weakness, which was followed by confusion 20 minutes later. Initial examination three hours after the onset showed him to be in coma with absent ciliospinal and oculocephalic reflexes, constricted and nonreactive pupils, and a blood pressure of 210/110 mm Hg.

Arteriography
Left carotid arteriography, performed five hours after the onset, revealed extravasation from the lateral lenticulostriate artery as a rice-sized stain in both the anterior and the lateral views. A four-second delayed film showed further expansion to Indian-bean size (fig. 2A). The patient expired 17 hours after the onset.

Autopsy
The intracerebral hematoma reached to the lateral ventricle and was centered at the putamen. A ruptured vessel felt to be the lateral lenticulostriate artery was found, confirming the ultrasonic x-ray finding (fig. 2B). Additional ruptured vessels of 150 μ and 165 μ in diameter were found at the periphery of the hematoma on histological examination.

CASE 3
A 51-year-old man, with known hypertension for several years, had a sudden collapse at noon. Examination one hour after the onset revealed semicomatose, with a left hemiplegia and decerebrate rigidity. The blood pressure was 220/110 mm Hg. Aniscoria (the right pupil larger than the left) and absent oculocephalic and ciliospinal reflexes were noted.

Arteriography
Right carotid arteriography was performed one and a half hours after the onset. In both the anterior and the lateral views of the arterial phase, extravasation was observed as a stain of soya-bean size (fig. 3). This extravasation was noted to be...
FIGURE 2B
Postmortem x-ray findings of Case 2 shows the ruptured vessel with extravasation (arrow).

FIGURE 3
The lateral view of Case 3 shows a soya-bean-sized extravasation (arrow).

530

larger on the four-second delay film. The patient expired six hours after the onset. Autopsy was not performed.

CASE 4
A 39-year-old man, with known hypertension for one year, complained of headache and rapidly developed a left hemiparesis. Examination 30 minutes after the onset revealed semicoma, anisocoria (the right pupil larger than the left), and a blood pressure of 210/130 mm Hg.

Arteriography
Right carotid arteriography performed at four and a half hours after the onset revealed extravasation in the periphery of the lateral lenticulostriate artery in both the anterior and the lateral views of the arterial phase (fig. 4). Further expansion was noted in the four-second delay film, with diffuse spread through the hematoma cavity.

Operative findings
A craniotomy was performed six hours after the onset. The hematoma was found to extend from 3 cm below the cortex to the lateral ventricle. Following evacuation of the hematoma, a vessel, which was felt to be the lateral lenticulostriate artery with a bleeding globe, was identified and noted to be still bleeding. Hemostasis was

Stroke, Vol. 3, September-October 1972

MIZUKAMI, ARAKI, MIHARA, TOMITA, FUJINAGA
ARTERIOGRAPHICALLY VISUALIZED EXTRAVASATION

FIGURE 4
The A-P view of Case 4 shows a soya-bean-sized extravasation (arrow).

obtained by clipping the artery, and bleeding from the hematoma wall was controlled. No additional swelling occurred and brain pulsation became apparent. A Penrose drain was inserted and the operation was completed.

Postoperative course
Anisocoria was less postoperatively, then disappeared completely by the second day. The state of consciousness gradually improved to the point of response to vocal commands by the third postoperative day. However, urinary output began to decrease on the third day, and the patient became anuric on the fourth day. He expired of acute renal insufficiency on the fourth postoperative day.

Autopsy
The brain was swollen with the hemorrhagic lesion centered around the right putamen and extending to the lateral ventricle. There was little residue of the hematoma, and the lateral lenticulostriate artery which had been clipped during the surgery was identified as the ruptured vessel.

CASE 5
A 70-year-old woman, with known hypertension and a right intracerebral hemorrhage eight years previously and with residual left hemiparesis, suddenly collapsed and went into coma. Examination 30 minutes after the onset revealed coma with decerebrate rigidity and a blood pressure of 230/120 mm Hg. Anisocoria (the left pupil larger) was noted, and the left oculocephalic and cold caloric responses were absent.

Arteriography
Left carotid arteriography was performed one and a half hours after the onset, revealing the lateral lenticulostriate artery to be displaced medially with a fingertip-sized extravasation observable at its periphery. This was well seen in both the anterior and the lateral projections (fig. 5). Four-second delay films showed further expansion and spread of contrast medium into the hematoma.

Autopsy
A hematoma extending from the putamen to the lateral ventricle was found. A ruptured vessel with a bleeding globe, felt to be the lateral lenticulostriate artery that showed extravasation on the arteriogram, was identified.

CASE 6
A 41-year-old man with hypertension developed a headache followed shortly by a right hemiparesis. Examination two and a half hours after the onset revealed a blood pressure of 220/130 mm Hg and stupor, which rapidly deepened to coma with dilation of the left pupil within one hour.

Arteriography
Left carotid angiography performed three and one-half hours after onset revealed extravasation from the lateral lenticulostriate artery in both the anterior and the lateral views of the arterial phase (fig. 6). Four-second delay films showed expansion of the extravasation.

Operative findings
A craniotomy was performed four and a half hours after the onset. The hematoma was found to extend from 3 cm below the cortex to the ventricle. Arterial bleeding was encountered in the area of the lateral lenticulostriate artery following evacuation of the hematoma. This bleeding was controlled by clipping the artery that had a bleeding globe. Brain swelling disappeared and brain pulsations became evident following the internal decompression. The hematoma cavity was drained with a Penrose drain.
The A-P view of Case 5 shows a fingertip-sized extravasation (arrow), and medially displaced lateral lenticulostriate artery.

Postoperative course
Pupillary equality and oculocephalic reflexes returned initially. Again, anisocoria developed and death occurred four and a half hours after operation.

Autopsy
The brain was very swollen and exhibited hemorrhagic lesions extending from the putamen to the lateral ventricle, with residual hematoma present in the lateral ventricle. The clipped vessel was identified as the lateral lenticulostriate artery, confirming that rupture of this vessel had been the cause of the hemorrhage.

CASE 7
A 63-year-old man, in whom hypertensive intracerebral hemorrhage resulting in left hemiparesis had been diagnosed one year previously, became unconscious. Examination 30 minutes after the onset revealed a blood pressure of 220/120 mm Hg and semicoma with right hemiplegia. Oculocephalic and ciliospinal reflexes were present.

FIGURE 6
The A-P view of Case 6 shows an Indian-bean-sized extravasation (arrow).

Postoperative course
During the first two postoperative weeks, the state of consciousness gradually improved, and the
FIGURE 7

The A-P view of Case 7 shows an extravasation (arrow) and medially displaced lateral lenticulostriate artery.

The A-P view of Case 7 shows an extravasation (arrow) and medially displaced lateral lenticulostriate artery.

The A-P view of Case 7 shows an extravasation (arrow) and medially displaced lateral lenticulostriate artery.

The A-P view of Case 7 shows an extravasation (arrow) and medially displaced lateral lenticulostriate artery.

The A-P view of Case 7 shows an extravasation (arrow) and medially displaced lateral lenticulostriate artery.

The A-P view of Case 7 shows an extravasation (arrow) and medially displaced lateral lenticulostriate artery.

The A-P view of Case 7 shows an extravasation (arrow) and medially displaced lateral lenticulostriate artery.

The A-P view of Case 7 shows an extravasation (arrow) and medially displaced lateral lenticulostriate artery.

The A-P view of Case 7 shows an extravasation (arrow) and medially displaced lateral lenticulostriate artery.

The A-P view of Case 7 shows an extravasation (arrow) and medially displaced lateral lenticulostriate artery.

The A-P view of Case 7 shows an extravasation (arrow) and medially displaced lateral lenticulostriate artery.

The A-P view of Case 7 shows an extravasation (arrow) and medially displaced lateral lenticulostriate artery.
resulted in death within 27 hours. Autopsy revealed hemorrhagic lesions extending from the putamen to the lateral ventricle in all cases, with the ruptured artery identified at surgery in three cases and at autopsy in two cases.

Discussion

Theories of the mechanism of hypertensive intracerebral hemorrhage can be roughly divided into two groups, according to whether priority is given to vascular rupture or to disturbances of the cerebral parenchyma. Debate on these points has been continuous. However, the predominant theory is that which attributes bleeding to the so-called angionecrosis of the intracerebral arteries or to the rupture of microaneurysms resulting from the angionecrosis. Opinion is divided as to the mechanism of the formation of massive intracerebral hematoma.

Our view is that large hematomas can be formed by continued bleeding from the ruptured vessel.

Extravasation of contrast medium in hypertensive intracerebral hemorrhage has been observed by Westerberg, Huckman et al., Leeds and Goldberg, and Yamaguchi et al., who have reported their cases briefly without attention to the significance of the formation of a massive hypertensive intracerebral hematoma.

The extravasations of the contrast medium observed in our seven cases were all from the lateral lenticulostriate artery. In Case 1, extravasation was observed from the artery over a period of approximately one hour. This is felt to be firm evidence for the formation of massive hematoma by continuous bleeding from a ruptured artery. During approximately a two-second delay between injection and exposure, a stain of about Indian-bean size, on the average, was formed. If this size stain develops during the period of time until exposure, the formation of a substantial hematoma by continuous bleeding is quite possible. The sizes of the hematomas visualized agree roughly with this calculation. However, additional ruptured vessels of 150 µ and 165 µ in diameter were found at the periphery of the hematoma in Case 2. This indicates that fragmentation is possible in the small vessels around a hematoma, due to acute circulatory disturbance as shown by Yamaguchi’s Case 4. But these vessels could not play a leading role in the formation of a massive hematoma.

Extremely important is the problem of whether the massive hematoma in hypertensive intracerebral hemorrhage is formed simply by mechanical elimination of the cerebral parenchyma, as we believe, or by fusion of necrotic lesions of the cerebral parenchyma. This has great support on the adequacy of surgical removal of the hematoma as a means of treatment. In view of our operative and autopsy findings, bleeding is postulated to develop in the direction of least resistance, by disruptive mechanical force. This would appear to be along a plane external to the putamen and between it and the caudatum. This plane corresponds to the watershed area between the distribution of the artery concerned in the rupture, and the cortical and subcortical branches of the middle cerebral artery. It is evident in the radiograph of bleeding from the putamen in figure 8 that the hematoma advanced, eliminating the surrounding parenchyma by pressure. The vessels were displaced by the hematoma, allowing a rough estimate of the location of the site of the hemorrhage from the shape and the direction of the vascular deformity.

VanderArk’s experiments support our view. He injected a mixture of Thorotrast and blood into monkeys’ brains, and found that the hematoma developed in the direction of least resistance, as if eliminating neural tissue. As we did, he placed the same interpretation on his cases in whom clinical signs improved dramatically after removal of the hematoma.

It is necessary here to reflect briefly on the relationship between cerebral angiography and extravasation. In all of our seven cases, the clinical course was deteriorating with a certain tempo, as mentioned by Fisher. There was no evidence that the tempo was accelerated by cerebral angiography. Among Fisher’s 2,500 cases of acute cerebral apoplexy in whom cerebral angiography was performed, no cases of secondary bleeding were recognized. Therefore, it is reasonable to assert that extravasation in these cases cannot readily be attributed to secondary bleeding.

We feel that in cases rapidly progressing to death from hypertensive intracerebral hemorrhage, bleeding from the ruptured artery continues over several hours at the least. We feel that the proper application of surgical treatment is to attempt to control bleeding at as
<table>
<thead>
<tr>
<th>A: Theory with priority to vascular rupture</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Rupture of microaneurysm</td>
</tr>
<tr>
<td>2. Angionecrosis → rupture of multiple vessels → fusion of small hematomas (2) (6)*</td>
</tr>
<tr>
<td>3. Cerebrovascular lesion (aneurysm, arteriovenous malformation, primary angionectosis, arteriosclerosis) → primary bleeding due to ruptured vessel, increasing venous pressure and other factors → secondary bleeding by extravasation or rupture of smaller vessels (8)</td>
</tr>
<tr>
<td>4. Rupture of cerebral vein</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>B: Theory with priority to disturbance of cerebral parenchyma</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Ischemic infarction of cerebral parenchyma due to angiospasm → extravasation or rupture of vessels (1)</td>
</tr>
<tr>
<td>2. Brain softening → rupture of ischemic vessels (3)</td>
</tr>
<tr>
<td>3. Disturbance of venous circulation → disturbance of cerebral parenchyma and vessels → extravasation or rupture of vessels (7)</td>
</tr>
</tbody>
</table>

*Numbers in parentheses indicate the reference which reported the theory.
early a stage as possible and to attempt removal of the hematoma following control of the isolated bleeding source. The fact that the clinical signs were improved, though temporarily, by early operation in Case 4, and that Case 7 survived, would suggest the possibility of surgery as a lifesaving device in certain cases which would have been felt to have a hopeless prognosis in the past. Discovery of seven cases in 19 months indicates that these findings would not be as rare if cerebral angiography were performed in the early stage in severe cases.

**Summary**

Seven cases are reported in which extravasation of contrast medium was observed from the lateral lenticulostriate artery on cerebral angiography performed in the early stage (one and one-half to seven hours) after the onset of hypertensive intracerebral hemorrhage.

It is felt that these findings will not be found to be exceptional if cerebral angiography is performed in the early stage in severe cases.

The mechanism of formation of a massive hematoma in hypertensive intracerebral hemorrhage is believed to be a hemorrhage resulting from a rupture of the main stem of a perforating artery, which progresses in the direction of least resistance in the cerebral tissue eliminating white matter mechanically.
ARTERIOGRAPHICALLY VISUALIZED EXTRAVASATION

In severe cases which rapidly progress to death, the bleeding is felt to continue from the single ruptured artery over a period of several hours.

Hematoma removal and control of hemorrhage were carried out in four of the seven cases, but six of the seven died.

The one case that survived with surgical treatment is felt to support the view that early surgery may be of benefit in severe cases.

Addendum

After we reported these seven cases, we had another case who survived, a 60-year-old man in coma. Angiography performed six hours after onset revealed extravasation. Surgery was performed 12 hours after onset. The patient is in good condition and walking with the aid of a cane.

Acknowledgments

We would like to express our thanks to Professor Tatsuyuki Kudo, Chairman of the Department of Neurosurgery, Keio University School of Medicine, Tokyo, Japan, for his advice, and to Associate Professor Yoichi Yashida, Department of Pathology, Gunma University School of Medicine, Maebashi, Japan, for his autopsy report.

Also, we wish to thank Professor Arthur G. Waltz, Department of Neurology, University of Minnesota Medical School, Minneapolis, Minnesota, for his review.

References

Arteriographically Visualized Extravasation in Hypertensive Intracerebral Hemorrhage
MASAHIRO MIZUKAMI, Goro Araki, Hiroshi Mihara, TAKASHI TOMITA and Ryozo Fujinaga

Stroke. 1972;3:527-537
doi: 10.1161/01.STR.3.5.527

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/3/5/527

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org/subscriptions/