Acute Hypertensive Subdural Hematoma from Arterial Rupture Shortly After the Onset of Cerebral Subcortical Hemorrhage: Leakage of Contrast Medium During Angiography

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SUMMARY  A case is reported of a Japanese female in whom acute right subdural hematoma due to the spontaneous rupture of the posterior branch of the right central artery occurred shortly after the onset of hypertensive subcortical hemorrhage of the right occipital lobe. Marked hypertension persisted. There was no evidence of subdural hematoma when a small collection of extravasated contrast medium from the cortical artery was revealed during right carotid angiography. Soon thereafter the patient became comatose and developed decerebrate posturing. Computed tomography scan was done immediately and a large subdural hematoma was identified. There was a gratifying response to prompt recognition and neurosurgical therapy of the condition. The pinpoint rupture of the cortical artery is considered to have occurred during marked hypertension. Previous 39 cases with subdural hematomas from arterial rupture, 27 traumatic and 12 spontaneous nontraumatic, are reviewed.

SUBDURAL HEMATOMAS FROM ARTERIAL RUPTURE have been even rarely reported. Out of 39 reported cases of such hematomas, 27 were traumatic and 12 spontaneous nontraumatic. In most of the reported cases, bleeding came from cortical arteries in the vicinity of the sylvian fissure. The source of bleeding was disclosed by autopsy or by operation. The mode of onset of signs and symptoms of acute spontaneous nontraumatic subdural hematomas from arterial rupture was acute in 8 patients, and their clinical course was just like cerebrovascular accidents.

Our present case differs from the reported cases in that acute subdural hematoma from arterial rupture occurred during marked hypertension shortly after the onset of hypertensive subcortical hemorrhage. The source of bleeding was verified during cerebral angiography as well as at operation.

Case Report
A 50-year-old Japanese woman was admitted to Kuwana Hospital at 2:00 p.m., on April 19, 1979, because of sudden onset of severe headache followed by nausea and vomiting. The symptoms had started about 4 hours prior to admission.

There was a past history of myoma uteri and hysterectomy had been performed at another hospital at the age of 38. The patient had had hypertension since 42 years of age, but had rarely undergone medical management. There was no history of head trauma.

Examination on admission revealed alert consciousness and normal mental functions. The patient had nausea and showed agonized facial expression due to an extremely severe headache of bursting or crushing sensation. No other neurological abnormalities were found. Blood pressure was 248/132 mmHg and pulse rate was 72/min. Lumbar spinal tap yielded clear and colorless cerebrospinal fluids (CSF). The initial pressure was 200 mmHg. CSF examination showed normal protein, glucose and chlorides, no red blood cells and one lymphocyte per mm³.

Computed tomography (CT) scan disclosed subcortical hematoma which occupied the right occipital lobe and measured about 14 cm³. A thin low density area was seen adjacent to the hematoma. The posterior horn, posterior two thirds of the body of the lateral ventricle and the sylvian fissure were closed on the right side. Displacement of midline structures was not found. Abnormal contrast enhancement was not observed (fig. 1).

Right carotid angiography (direct common carotid puncture using 19 gauge Teflon canula) performed at about 5:30 p.m. disclosed no aneurysms nor arteriovenous malformations. The posterior cerebral artery was not filled. Arteriosclerotic elongation of the internal carotid artery and arteriosclerotic elongation and dilatation of the middle cerebral and the anterior cerebral arteries were demonstrated. Leakage of contrast medium was identified in the venous phase in the first series of the angiography. The stain of a small collection of the extravasated contrast medium persisted and was revealed to be located over the surface of the precentral gyrus just adjacent to the posterior branch of the central artery. No evidences of subdural hematoma were found (fig. 2).

To examine the territory of the posterior cerebral artery, transfemoral vertebral angiography was performed using Kifa’s guide wire and catheter. The catheter tip slipped easily into the left common carotid artery, but did not enter the vertebral artery. Then, left transbrachial vertebral angiography was performed. No aneurysms nor arteriovenous malformations were found.

In spite of osmotherapy and administration of antihypertensive drugs, blood pressure was 222/122 and
240/140 mmHg during these angiographic procedures.

While performing transbrachial vertebral angiography, the patient began not to respond to verbal orders. The pupils were round and equal in size measuring 4 × 4 mm in diameter. Light response was prompt. There was no papilledema. Paresis was not present. The patient became comatose at 9:30 p.m. when these angiographic procedures were completed. Both pupils were unresponsive to light, the right being markedly larger than the left. Right eye ball did not move to the left side by doll’s head maneuver. Left Babinski’s and Chaddock reflexes were present. The situation was thought critical and the patient was rushed to CT scanning immediately followed by surgery.

CT scan disclosed right subdural hematoma, marked midline shift to the left, and closing of the third and right lateral ventricles (fig. 3).

The patient developed decerebrate posturing.

Operation started at 10:55 p.m. A large right frontoparietotemporal craniotomy revealed an extensive subdural fresh clot over most of the hemisphere. There was no neomembrane. A fine stream of blood jetted into the field from a pinpoint opening in the posterior branch of the central artery of the middle cerebral artery on the surface of the cortex when the overlying clot was removed. The bleeding was arrested using bipolar electrocoagulation. The underlying brain was normal: there was no cerebral contusion, no subarachnoid blood staining, no aneurysm, no arteriovenous malformation nor angioma. The opening in the artery was considered to be responsible for the cause of the
Figure 3. Computed tomogram demonstrating right acute subdural hematoma following right occipital subcortical hemorrhage. Contrast enhancement of the brain was seen because computed tomography was performed soon after cerebral angiography.

subdural hematoma and for the source of leakage of contrast medium during cerebral angiography. The hematoma weighed about 90 gm. Extremely large patch-grafting of the dura was performed. Bone flap was removed for decompressive purposes and was preserved for delayed implantation.

By 10 o'clock the following morning the patient was alert and had a slight left hemiparesis. Decompressive area was very tense and bulged. Two days after the surgery, generalized convulsions occurred and were controlled with anticonvulsive drugs. The patient showed excellent recovery and was discharged on August 6, 1979. No neurological deficits were present. Blood pressure returned to normal levels with control of diet and drug therapy. On May 8, 1980 an autogenous skull cranioplasty was performed because bulging of the decompressed area disappeared. The patient has remained well.

Discussion

It is well recognized that subdural hematomas develop most frequently after head trauma and the source of bleeding is thought to be venous in origin. Subdural hematomas are less frequently secondary to other pathological lesions such as tumors, infections, rupture of intracranial aneurysms, ventricular decompression for hydrocephalus, pneumoencephalography, blood dyscrasias, complication of anticoagulant therapy and alcoholism.7, 12-17

On the other hand, attention has been also directed to subdural hematomas from arterial rupture since the report of Werkgartner10 in which autopsy and histological examination disclosed the source of the acute fatal subdural hematoma following head trauma to be a ruptured cortical artery on the right temporal lobe.

Thirty-nine cases of subdural hematomas from arterial rupture have been reported in the literature.1-4, 6-8 These hematomas are divided into two groups, i.e., traumatic and spontaneous nontraumatic.

Twenty-seven cases were traumatic (table 1). Fifteen patients were males, 5 females, and sex was not described in 7. Nine were between 60 and 69 years of age, 7 between 70 and 79 years, 3 between 30 and 59 years, 3 between 40 and 49 years, 2 between 30 and 39 years, 2 between 20 and 29 years and 1 was 3 years of age. The symptoms were acute in 17 cases, subacute in 7 and chronic in 2. Three had a history of alcoholism, 1 suffered from hypertension, 1 from serum sickness and 1 from chromophobe adenoma. Ruptured artery was disclosed by autopsy and histological examination in 10 cases7, 9, 10 and by autopsy in 1.13 The source of bleeding was verified at operation in 16 patients.2, 4, 6, 8 Operative results were well in 8, hemiparetic in 1 and dead in 4 out of 13 acute or subacute patients in whom operative results were described.

Twelve cases have been reported of spontaneous nontraumatic subdural hematomas from arterial rupture (table 1). Ten patients were males and 2 were females; 6 were between 50 and 59 years of age, 4 between 60 and 69 years, 1 was 48 years and 1 was 37 years. The symptoms were acute in 8 patients, subacute in 2 and chronic in 2. Two had a history of hypertension, 1 suffered from alcoholism and hypertension, 1 from lung tuberculosis 24 years before and hypertension 6 months before, 1 from chronic bronchitis, 1 from epileptic seizures for many years, 1 from polycythemia rubra vera, 1 from head trauma 2 years before and 1 from a single generalized seizure. The source of bleeding was verified at operation in all the 12 patients. Five died postoperatively out of 7 acute patients in whom operative results were described.

Because the hematoma exists outside the cerebrum, prompt neurosurgical intervention may save the patients with even severe neurological deficits such as in the state of coma with decerebrate rigidity. Removal of hematoma with large decompressive craniectomy and large patch-grafting of the dura which allows the edematous swollen brain to expand away from the brain stem postoperatively, may lead operative results more favorable than without decompressive craniectomy.
Angiographic extravasation of contrast medium in subdural hematomas from arterial rupture is rare. The occurrence was reported in only 1 out of 16 cases with acute or subacute subdural hematomas from arterial rupture in which cerebral angiography was performed. Ito et al. reported a case of the acute traumatic subdural hematoma in which extravasation of the contrast medium from an ascending branch of the middle cerebral artery was shown by angiography and the leak was verified at operation.

As to the possible mechanism of the occurrence of subdural hematomas from arterial rupture, Vance divided these traumatic subdural hematomas into two groups. He presumed that a branch of the ruptured vessel was a part of a bridging trunk from the subarachnoid space to the dura and was torn by the forcible oscillation of the brain at the time the injury was inflicted. The other was what he termed "fire hose" rupture, as it occurs in arteries on the lateral cerebral surface which possess small arterial twigs coming off the outer wall of the vessel at right angles just under the arachnoid. A point of weakness is therefore present which is ill fitted to withstand any increase of pressure inside the vascular lumen. Drake assumed that the

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<th>Age (yr)</th>
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arterial bleeding stemmed from the gliding rotatory movement of the brain within the skull upon injury, tearing an artery or arterial twig from a dural attachment, leaving a tiny rent in the vessel. Talalla and McKissok assumed that subclinical subdural clots may form a possible etiology for spontaneous subdural hemorrhages. When such a small clot resolves spontaneously, this must in some instances lead to the formation of significant adhesions between the surface of the brain and the inner surface of the dura. With sudden movement of the head, tension can be put on these adhesions and these adhesions can rupture. O'Brien et al. presumed that the junction of arterial twigs as they emerge from the parent vessel represents an anatomically weak point where any stress, whether it is minor trauma or a hemorrhagic diathesis, may cause rupture. Byun and Patel stated that spontaneous subdural hematoma from arterial bleeding was probably related to the formation of adhesions between the cortical artery and the arachnoid. Minor motions of the brain, which are usually insignificant, could cause tearing of both the vessel wall and the arachnoid at the site of adhesion and subsequent hemorrhage into the subdural space.

In our present case, it may be assumed from the viewpoints of clinical course and angiographical as well as operative evidences that the pinpoint rupture of the cortical artery occurred during marked hypertension, but the bleeding point adhered to the arachnoid membrane, then the bleeding was easily arrested. Soon thereafter rebleeding occurred into the subdural space, tearing the arachnoid membrane adherent to the bleeding point.

Acknowledgments

The author is indebted to Dr. Komei Ueki, Professor Emeritus of Niigata University, for helpful criticism of the manuscript, to Dr. Kenzio Ozaki, Dr. Shigeaki Ohsugi, Dr. Hiroaki Hondo and Dr. Teruo Miyakawa for care of the patient, to Mr. Kiyoshi Uesugi for technical assistance in photography and to Miss Ryuko Sato for preparation of the manuscript.

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Acute hypertensive subdural hematoma from arterial rupture shortly after the onset of cerebral subcortical hemorrhage: leakage of contrast medium during angiography.

H Arai

Stroke. 1983;14:281-285
doi: 10.1161/01.STR.14.2.281

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