Extravasation of contrast material into subdural space from internal carotid aneurysm during angiography

Case report

KUNIYUKI SOMEDA, M.D., NOBUYUKI YASUI, M.D., YOSHIKUMI MORIWAKI, M.D., YASUO KAWAMURA, M.D., AND HIROSHI MATSUMURA, M.D.

Department of Neurosurgery, Kansai Medical School, Osaka, Japan

A case of multiple aneurysms is reported in which rupture of the internal carotid aneurysm occurred during carotid angiography, opacifying the subdural space. The patient recovered and was successfully treated by clipping of the aneurysm neck.

KEY WORDS • aneurysm • contrast material, extravasation • subdural space opacification • Urografin • angiography

There have been a number of reports of rupture of an aneurysm during angiography opacifying the subarachnoid space, the ventricles, or an intracerebral mass. The outcome of this rare complication is grave and only four of the 16 cases so far reported have recovered. We are reporting a unique case in which rupture of the aneurysm occurred during carotid angiography and opacified the subdural space. The patient recovered following successful operative obliteration of the aneurysm.

Case Report

On April 24, 1974, this 48-year-old woman suffered a headache with nausea and vomiting; her headache persisted for about a week. On May 2, she suddenly had severe neck pain, nausea, and vomiting, and lost consciousness. She was taken to a hospital in a semicoma state, with weakness of the right leg. Spinal tap revealed grossly bloody cerebrospinal fluid (CSF). She gradually recovered consciousness and on the following day was alert and well oriented; weakness of the right leg persisted.

On May 9, she again became unconscious for about an hour. The following day she was admitted to our clinic, conscious and well oriented. She complained of headache and nausea and vomited frequently. The pupils were equal and reacted promptly to light. She had a stiff neck and slight weakness of the right leg, but tendon reflexes were normal and symmetrical. Neurological examination was otherwise normal. Spinal tap was performed; the opening pressure was 260 mm H2O, CSF was grossly bloody, and the supernatant was xanthochromic after centrifugation.
FIG. 1. Right retrograde brachial angiogram revealing the internal carotid-posterior communicating aneurysm.

Angiography. Left carotid angiography performed on the third hospital day demonstrated an internal carotid (IC) aneurysm 4 x 3 mm in diameter at the origin of the posterior communicating (PC) artery. On the fifth hospital day, right retrograde brachial angiography was carried out to examine the right carotid and vertebrobasilar systems; another IC-PC aneurysm was found, with spasm at C1, M1, and M2 suggesting recent rupture (Fig. 1). The patient’s condition was unchanged throughout the procedure.

Since the need for prompt surgical treatment necessitated successive right carotid angiography for further evaluation of the aneurysm, 10 ml of 60% Urografin was injected by hand into the right common carotid artery. Immediately following injection, the patient became unconscious with upward deviation of the eyes, and respiration ceased. After cessation of the tonic phase, respiration started, at first irregular and superficial, and gradually returned to normal. Fifteen minutes later, she responded to painful stimuli by moving her right extremities; she had apparent left hemiplegia involving her face. Respiration was regular and normal. The level of consciousness improved rapidly and 1½ hours later she could follow simple verbal commands. In 2 hours she had become almost fully conscious.

The right carotid angiogram showed that a large amount of contrast material had leaked out from the aneurysm, filling the Sylvian fissure at the bifurcation (Fig. 2 upper). Successive films showed the contrast material flowing under the base of the temporal lobe to the dorsal surface of the tentorium (Fig. 2 middle and lower). Plain skull films taken 15 minutes later still demonstrated the presence of the contrast material above the tentorium; there was also faint opacification above the sella turcica and in the Sylvian fissure on the lateral film (Fig. 3).

Operation. On the day following angiography, right frontal craniotomy was performed under hypothermia. There was a thin subdural clot about 5 mm thick, and the brain was moderately tense. The aneurysm was exposed by gentle retraction of the frontal lobe, and clipped with the aid of an operative microscope. The fundus of the aneurysm was buried in fresh hematoma within the Sylvian fissure and fairly thick membranous adhesion was found around its neck and over surrounding brain. The adhesion seemed to have prevented blood leaking from the ruptured aneurysm from reaching the chiasmatic and basal cisterns, which were loosely filled with grayish, partially-organized old clots. Then through a left frontal craniotomy, the left IC-PC aneurysm was also successfully clipped at the same time. There was no sign of rupture on this side.

Postoperative Course. Postoperative recovery was uneventful and the left hemiparesis improved. The postoperative carotid angiograms showed that the aneurysms were obliterated; arterial spasm was still present at C1 and M2 on the right side. A narrow avascular area around the right hemisphere indicated thin subdural clots. The patient was discharged on the 22nd postoperative day, with mild left hemiparesis.

Discussion

Leakage of contrast material from an aneurysm during angiography has been reported before. Bakay and Sweet1 believed that this demonstrated the purely coincidental rupture of the aneurysm during injection of the contrast material; however, according to Lin, et al.,14 intraarterial pressure in the carotid artery is temporarily increased during injection of contrast material into the brachial artery. Such a definite hydrodynamic change could initiate rupture of a susceptible aneurysm. The incidence of rupture of
Contrast material leak into subdural space

Fig. 2. Right serial carotid angiograms showing the extravasation and spread of contrast material. Anteroposterior projections are on the left (A, B, and C) and lateral views on the right (D, E, F). Upper: Immediately after injection of 10 cc of Urografin. Middle: 1.5 seconds after injection. Lower: 6 seconds after injection.
aneurysms caused by injection of contrast material is small in comparison to the frequency with which angiography is done at present. However, this procedure poses a definite hazard in patients who have had repeated subarachnoid hemorrhages at short intervals, and in such patients angiography should be performed as early as the patient’s condition permits. In addition to the deleterious effect of hemorrhage, contrast material per se is toxic once it enters a subarachnoid space. Appropriate precautions include a low injection pressure and the use of the minimum necessary amount of contrast material.

Rupture of an aneurysm usually causes subarachnoid hemorrhage; more than 80% of subarachnoid hemorrhages are presumed to be due to aneurysmal rupture. Occasionally, rupture gives rise to intracerebral hematoma and intraventricular hemorrhage, and there have been reports of aneurysmal rupture during angiography with opacification of ventricles or demonstration of a mass. The presence of subdural clots due to rupture of aneurysms is not an uncommon finding at craniotomy, and incidence of subdural hematoma is estimated between 0.5% and 7.9%. The amount of blood in the subdural space from aneurysmal rupture varies from thin blood clots to a rapidly fatal massive hematoma. Strang, et al., reported that the site of the aneurysm has some importance in determining the associated formation of a subdural hematoma, and that aneurysms of the carotid and middle cerebral arteries have a relatively high incidence.

In our case the distribution of the contrast material as seen on successive films and the patient’s rapid recovery without epileptic seizures suggests that the rupture of the aneurysm resulted in hemorrhage in the subdural space; this was confirmed by the finding of subdural clots at craniotomy. We assume that the membranous adhesions around the aneurysmal sac and extending over the surrounding brain prevented the contrast material from leaking from the aneurysm into the subarachnoid space, so that it spread underneath the temporal lobe. Boop, et al., reported a case of ruptured intracranial aneurysm complicated by subdural hematoma in which no apparent hemorrhage into the cerebrospinal fluid was recognized. Clarke and Walton mentioned that hemorrhage into the subdural space was more likely to occur in the second or a subsequent rupture because of formation of adhesions around the sac of the aneurysm. Strang, et al., reported two cases of subdural hematoma due to aneurysmal rupture and discussed mechanisms by which the aneurysmal blood reached the subdural space. They mentioned direct rupture into the subdural space with tear of arachnoid membrane which, as a result of previous leakage and the development of adhesions, had become a part of the sac wall. This seems to be the more probable explanation in our case. It is also interesting to know the speed with which blood spreads in the subdural space.

Toxicity of water-soluble contrast materials in the ventricles and subarachnoid space has been investigated by Heimburger, et al., and it is well known that Conray 60 is among the least toxic. Although it is safely used for ventriculography, epileptic seizures often ensue when it gains access to the cerebral cortex. In our case Urografin, which is more toxic than Conray 60, extravasated into the subdural space, and the intervening subarachnoid space with cerebrospinal fluid fortunately protected the cerebral cortex from its toxic action.

References
Contrast material leak into subdural space


Address reprint requests to: Kuniyuki Someda, M.D., Department of Neurosurgery, Kansai Medical School, Osaka, Japan.