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## ANEURYSMAL RUPTURE DUE TO CEREBRAL ANGIOGRAPHY SUBSEQUENT TO SUBARACHNOID HEMORRHAGE

BY

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### ABSTRACT

A report is made of a case of rupture of intracranial aneurysm by angiography which was done four hours after the subarachnoid hemorrhage. This is a twenty-eighth reported case of the ruptured aneurysm by angiography. The criteria of this complication are presented. The cause and mechanism of this complication (especially vasospasm, change of intraarterial pressure and peeling out of the thrombus) is discussed. Clinical factors of this complication are shown by summarizing previous reports. Cerebral angiography within a few hours after subarachnoid hemorrhage is very dangerous although it is said that angiography should be done as early as possible.

### INTRODUCTION

Cerebral arteriography first reported by Egas Moniz<sup>1)</sup> is now important as a diagnostic measurement of neurological diseases, especially in cerebrovascular disorder. Unfortunately, however, many kinds of complications of cerebral angiography have been reported. Rupture of intracranial aneurysm is one of the most serious and regretful complications. This is very rare but sometimes described because of its seriousness and important matter for neurosurgeons. In this paper, we report a case of ruptured aneurysm during angiography, summarize previously reported cases from many standpoints and discuss some important factors and problems.

### CASE

A 31-year-old male patient was brought to our outpatient's clinic by ambulance because of loss of conscious and convulsive seizure on February 5, 1974. Lumbar puncture showed grossly hemorrhage in the CSF. He was

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hospitalized immediately and administered anticonvulsants, hypotensive drugs, antifibrinolytic agents, and corticosteroid. His signs and symptoms on admission were mild headache, nausea, nuchal rigidity, anisocoria (rs > ls), and left hyperreflexia. This attack was his second. He first was a bleeding attack on January 30, 1974. Four hours after this attack cerebral angiography was done under local anesthesia with heavy premedication. Puncture of common carotid arteries were uneventful. Three injections of 60% Conray, at the dose of 8 ml each, were given and three films were taken. Few minutes after injections, he suddenly became unconscious and ceased respiration for a few seconds. Blood pressure was 210 mmHg at systolic, although 120 mmHg two minutes before. Immediately resuscitation started. His respiration reappeared within 0.5 min. and his vital signs gradually improved. We stopped examination. When returned to his bed, right pupil dilated and optic fundi showed bleeding. Arteriography showed a large dumbbell shaped aneurysm at the trifurcation of the right middle cerebral artery but no finding of hematoma (Fig. 1). We decided emergency operation at once. When the operation was started his both pupils dilated, and B.P. was very low.

*Operation:* Right side large front lateral craniectomy was done. Large subdural hematoma (Fig. 2), severe diffuse subarachnoid hemorrhage (Fig. 3), and intracerebral hematoma were found. Aneurysmal neck clipping was successfully done.

*Postoperative course:* His level of consciousness was semicomatous but his state gradually deteriorated, and he died 1 week after the operation.

*Autopsy:* There was severe edema in both cerebra. The brain stem, especially interbrain, midbrain, and pons, had fallen into softening or the so-called respirator brain.

#### DISCUSSION

All of the reported cases are summarized in Table 1 which shows that the incidence is very low, but we think it is more frequent. It is very difficult to say clearly that angiography is responsible for aneurysmal rupture. Our criteria for aneurysmal rupture due to angiography are as follows:

- (A) 1. Cerebral angiography shows an aneurysm.
2. During or just after the cerebral angiography there are some signs or symptoms of sudden onset of bleeding attack.
3. Fresh aneurysmal bleeding is confirmed by surgery or autopsy within a short time after the attack.

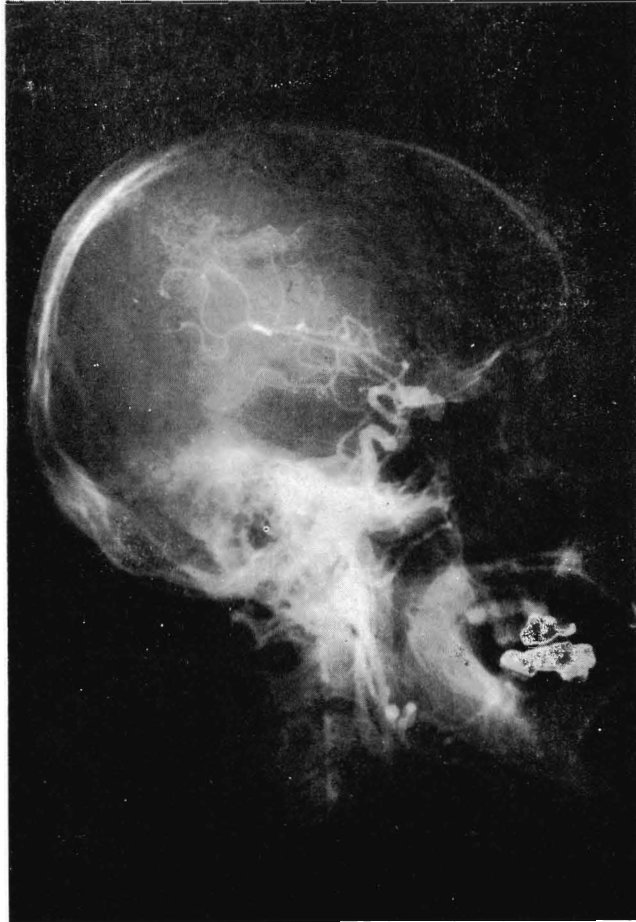


Fig. 1. Arteriogram of right carotid shows a large dumbbell shaped aneurysm and no signs of hematoma or vasospasm.

(B) 1. (A)-2

2. Cerebral angiography shows extravasation from aneurysm.

Therefore, extravasation from aneurysm is neither necessary nor sufficient.

*Incidence:*

Sex: Female is preponderant to male. (Table 2-A:B)

Position of aneurysm: Rupture of internal carotid aneurysm is the most frequent, and cases of anterior cerebral aneurysm have not been reported. This fact will be closely related to the cause of rupture (Table 2-A:D). Aneurysmal rupture due to angiography has occurred only in the case of aneurysms of circle of willis and trifurcation of middle cerebral artery. Aneurysm of peripheral branches never ruptured during angiography. In-

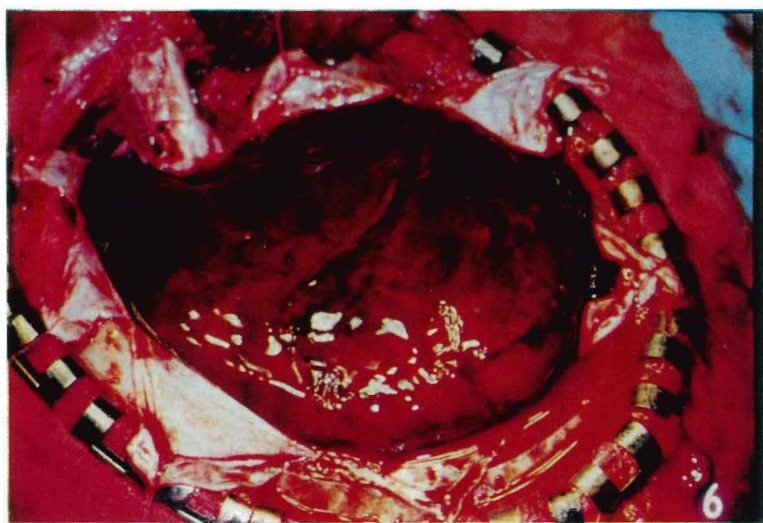


Fig. 2. Operative film shows massive subdural hematoma as a result of aneurysmal bleeding.

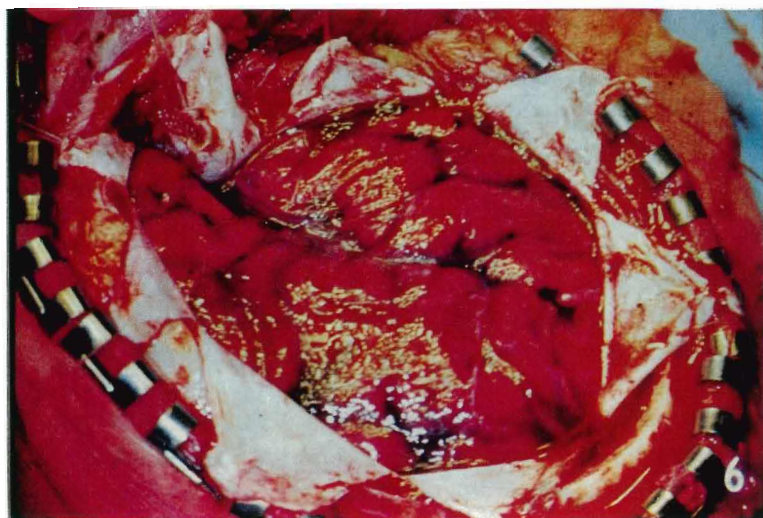


Fig. 3. Operative film shows severe subarachnoid hemorrhage as a result of aneurysmal bleeding.

Table 1. Cases of rupture of intracranial aneurysm due to angiography

Author	Year reported	No. of cases reported	Site of ruptured aneurysm
Abott <sup>6)</sup>	1952	1	IC-PC
Jenkinson <sup>13)</sup>	1954	1	IC-PC
Jamieson <sup>8)</sup>	1954	1	MCA
Gallagher <sup>3)</sup>	1956	1	ACCA
Jackson <sup>14)</sup>	1960	1	IC-PC
Triska <sup>15)</sup>	1962	1	MCA
Wright <sup>11)</sup>	1962	3	ACCA, IC-PC, IC
Field <sup>16)</sup>	1962	2	IC-PC, IC
Pool <sup>17)</sup>	1965	1	BA
Liu <sup>12)</sup>	1965	1	IC-PC
Perret <sup>1)</sup>	1966	2	?
Murphy <sup>18)</sup>	1967	1	IC
Goldstein <sup>19)</sup>	1967	1	IC-PC
Hoff <sup>20)</sup>	1969	1	BA
Beamer <sup>21)</sup>	1969	1	ACCA & IC
Ferrari <sup>7)</sup>	1969	1	IC
Tsuda <sup>22)</sup>	1970	1	ACCA (two)
Freemon <sup>23)</sup>	1970	1	IC-PC
Vines <sup>24)</sup>	1971	1	IC-PC
Sakamoto <sup>25)</sup>	1972	1	IC-PC
Lehrer <sup>4)</sup>	1972	1	ACCA
Karadayi <sup>26)</sup>	1973	1	MCA
Waga <sup>27)</sup>	1973	1	IC-PC
Ohata	1974	1	MCA

IC: Internal carotid artery

IC-PC: Internal carotid-posterior communicating artery

ACCA: Anterior communicating artery

MCA: Middle cerebral artery

BA: Basilar artery

tracranial internal carotid artery receives most directly the pressure of contrast medium injection for angiography. We routinely irrigate the intra-arterial punctated needle with 2-3 ml of saline containing anticoagulants to prevent formation of a clot but at the time of injection of saline containing anticoagulants, the concentration of anticoagulants is the highest in the internal carotid artery.

Lin and Chase<sup>2)</sup> proved the rise of intra-arterial pressure by brachial injection. Undoubtedly rapid injection of a large amount of contrast medium increases intra-arterial pressure. For example, aneurysmal rupture occurred by the common carotid injection of 20 ml of contrast medium<sup>3)</sup> or by the brachial injection of 50 ml of contrast medium<sup>4)</sup> (Table 2-B:D). Meanwhile, Bakay and Sweet<sup>5)</sup> reported that, in their clinical experiment, any rapid injection of 10-12 ml of contrast medium into the common caro-

Table 2-A. Summary of reported case

A. Age distribution		D. Sites of ruptured aneurysm	
Age	No. of cases	Site	No. of cases
~35	6	IC	17
36~60	16	ACA	0
61~	2	MCA	4
		ACCA	5
		BA	2
B. Sex distribution		E. Number of aneurysms	
Sex	No. of cases		No. of cases
Male	7	Single	20
Female	15	Multiple	6
C. Surgical risk grade (Hunt & Hess)			
Grade	No. of cases		
I, II	9		
III~	11		

Table 2-B. Summary of cases

A. Duration after previous hemorrhage		D. Volume of injection	
Day	No. of cases	Volume	No. of cases
0	6	~10 ml	14
1~7	6	11~15 ml } I.C. or C.C.	2
8~14	5	20 ml }	1
15~21	2	50 ml (Brachial)	1
22	2		
B. No. of previous ruptures		E. Timing of rupture	
No. of ruptures	No. of cases	Timing	No. of cases
One	9	Arterial puncture of needle	2
Over two times	8	During injection	7
		Just after injection	13
		Arterial compression	6
C. Artery injected		F. Mortality	
Artery	No. of cases		No. of cases
I.C.	4	Alive	6
C.C.	15	Dead	19
Brachial	1		

tid did not change the internal carotid pressure, though there were some reported cases of rupture by common carotid injection of less than 10 ml of contrast medium. Bakay et al.<sup>5)</sup> concluded that this was coincidental but we cannot agree with their view. As Abott<sup>6)</sup> or Ferrari<sup>7)</sup> stated, reflex vasoconstriction by needle puncture or vasospasm by chemical irritation of contrast medium is very important as a factor for aneurysmal rupture by angiography.

Jamieson<sup>8)</sup> stressed the significance of chemical irritation by contrast medium. The development of non-irritant and good contrast medium is very important. Intra-arterial concentration of a contrast medium is highest in the internal carotid artery. It is thought that in the present case, each volume of 8 ml of contrast medium injected slowly into the common carotid artery was not responsible for increasing the intraarterial pressure.

If vasospasm was responsible for rupture, early spasm should have occurred. Although it is said that early spasm occurs within a few minutes, angiograms taken during 20 min. showed no spasm. Ferrari<sup>7)</sup> and Rowbotham<sup>9)</sup> stressed the number of injections and briefness of the intervals between them. In this case, the number of injections and the intervals between them did not matter.

Interval between the last bleeding attack and angiography can be seen in Table 2-B:B. Aneurysmal rupture by angiography is most frequent when angiography is performed on the day of the rupture, and becomes less frequent when angiography is performed more than 2 weeks after the previous rupture. However, this tendency is not clearly defined because not only there are small number of cases but also many lack detailed reports. It is hoped that more chronologically detailed course would be reported. In the present case, mechanism of rupture is thought to be that infirm, fresh, easily coming-off clot was peeled out from the dome of the aneurysm because of a very mild increased intra-arterial pressure or slightest vasospasm, and produced a small cleft in the aneurysm and this cleft gradually enlarged and massive bleeding occurred. Because the interval between previous bleeding and examination was only 4 hr., clearly increased intra-arterial pressure would not occur because of the position of needling and the amount of contrast medium injected, and angiograms showed no vasospasm. It was also thought that irrigation with saline and anticoagulants were responsible for peeling out of the clot from the dome of aneurysm. Rowbotham<sup>9)</sup> and Sedizimir<sup>10)</sup> said that the irrigation with saline and anticoagulants decreased angiographic complication, but it might be possible that even small amount of anticoagulants would peel out the feebly fresh clot from the dome of aneurysm as a focal effect.

Table 2-A:E shows that the frequency of rupture of multiple aneurysm was higher than that of a single one. There were fairly many cases in which carotid compression was performed in order to prevent bleeding from the point of puncture or to see the cross filling (Table 2-B:E). The mechanism of these cases is clearly the increased intra-arterial pressure. This phenomenon was clearly proved by Wright<sup>11</sup>). In the present case judging from its clinical course, blood pressure of the patient, who had no hypertension in his early history, showed a striking and abnormal increase (120 mm Hg to 210 mm Hg) within a few minutes and this was considered to be the result of increased intracranial pressure because of aneurysmal bleeding and was not considered to be the cause of rupture.

Clinical grade (by Hunt and Hess) at the time of examination or previous number of rupture had no relation to angiographic rupture (Table 2-A:C, 2-B:B). Table 2-B:F shows that the mortality rate of this complication is very high. It is thought that prognosis depends on the amount of bleeding. Liu<sup>12</sup>) reported a case successfully relieved by immediate ligation of the carotid artery. However, there are still some cases in which aneurysmal rupture and angiography were purely coincidental when various factors are considered.

However, it is thought that following factors, which would result in increased intraarterial pressure, dissolving or peeling out of clots, or production of vasospasms, have important relation to the induction or mechanisms of angiographic aneurysmal rupture.

1. Location of aneurysm (especially internal carotid)
2. Duration between the last rupture and angiography (especially few hours)
3. Kind of contrast medium used
4. Speed and amount of each injection
5. Presence of contralateral compression
6. Injection of anticoagulants

It will be possible that the cause and mechanism of angiographic aneurysmal rupture will be solved more clearly and confirmingly by detailed and accurate clinical observation and experiments. At least we emphasize that cerebral angiographic examination within a few hours after rupture of aneurysm is very dangerous although it is said that angiography should be done as early as possible.

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