CHANGES IN THE SIZE OF INTRACRANIAL ARTERIAL ANEURYSMS

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Despite the fact that intracranial arterial aneurysms occasionally are found in children, their occurrence is rare before the age of 15 years. But since they are comparatively common in adults, they must have begun to develop at some stage after childhood. Since little is known about how and when aneurysms grow, we have tried to elucidate the problem by a survey of a series of intracranial arterial aneurysms.

PATIENTS

Up to Jan. 31, 1962, some 700 patients with intracranial arterial aneurysms were seen in the Department of Neurological Surgery of the Helsinki University Central Hospital. On 19 of these, a second carotid angiography was performed after an interval varying between 2 weeks and 10 years, but without any decisive surgical procedures having been carried out in the meantime.

There were 8 men and 11 women. Their age at first hemorrhage ranged from 17 to 59 years, the mean being 39.5 years. All had had at least one verified subarachnoid hemorrhage, though not necessarily from the aneurysm considered in this report.

The aneurysms were located as follows:

<table>
<thead>
<tr>
<th>Artery</th>
<th>No. of Patients</th>
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<tbody>
<tr>
<td>Internal carotid</td>
<td>3</td>
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<tr>
<td>Bifurcation of internal carotid</td>
<td>3</td>
</tr>
<tr>
<td>Middle cerebral</td>
<td>9</td>
</tr>
<tr>
<td>Anterior communicating</td>
<td>3</td>
</tr>
<tr>
<td>Multiple aneurysms (right callosomarginal + left pericallosal)</td>
<td>1</td>
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Eleven patients were treated conservatively. On primary admission, an intracranial ligature of the aneurysm was attempted on 4 patients. However, postoperative angiography showed that the attempt had been futile, the aneurysm filling as before. Three patients with an aneurysm of the middle cerebral artery were operated upon for a second aneurysm: in 2 cases an aneurysm of the internal carotid artery, and in 1 an aneurysm of the anterior communicating artery. One patient with an aneurysm of the internal carotid artery also had an aneurysm of the internal carotid artery on the opposite side; this aneurysm was operated upon on first admission.

RESULTS

In 10 patients the aneurysm definitely had grown between angiographies, the interval being 3, 4, 5, 13, 13, 20, 47, 48, 62, and 125 months. Six of these 10 had suffered recurrent hemorrhage in the interval (3, 4, 13, 48, 62, and 125 months). One of these 6 had undergone intracranial exploration after the first angiography. In the remaining 4, there had been no symptoms suggesting recurrent hemorrhage during the interval (5, 13, 20, and 47 months). Two of these patients had undergone exploratory craniotomy after the first angiography.

In 8 patients, the size of the aneurysm was unchanged 4, 10, 11, 12, 15, 20, 22, and 88 months after the primary angiography. None of these patients had had a verified or clinically probable subarachnoid hemorrhage in the meantime.

In 1 patient, the aneurysm had disappeared during the interval; this patient had been subjected to an attempt at clipping of the aneurysm on primary admission.
SELECTED CASE HISTORIES

a) Growth of Aneurysm With Recurrent Hemorrhage (6 cases).

Case 1. Woman, born 1910. Subarachnoid hemorrhage with right hemiparesis and aphasia, Sept. 25, 1950. Left carotid angiography, May 11, 1951: small nubbin arising from the junction of the internal carotid and posterior communicating artery (Fig. 1). Recurrent hemorrhage, September 1961. Left carotid angiography, Oct. 10, 1961: large aneurysm of the internal carotid-posterior communicating junction (Fig. 2). Right carotid angiography, Oct. 18, 1961: widening of the infundibulum of the posterior communicating artery (Fig. 3).

Case 2. Woman, born 1902. Verified subarachnoid hemorrhage and left ophthalmoplegia in Spring of 1952. Left carotid angiography, June 4, 1952: aneurysm of the internal carotid artery (Fig. 4). Recurrent subarachnoid hemorrhage April 22, and May 24, 1955. In 1957, slowly progressing ophthalmoplegia. Left carotid angiography, Aug. 17, 1957: considerable growth of aneurysm (Fig. 5).

b) Growth of Aneurysm Without Recurrent Hemorrhage (4 cases).

Case 3. Woman, born 1895. Subarachnoid hemorrhage and slight aphasia, April 9, 1955. Left carotid angiography, May 6, 1955: aneurysm on left middle cerebral artery (Fig. 6). No symptoms of recurrent hemorrhage during interval. Left carotid angiography, April 18, 1959: slight increase in size of aneurysm (Fig. 7).

c) Size of Aneurysm Unchanged; No Recurrent Hemorrhage (8 cases).

change in size of callosomarginal aneurysm. Blood pressure 150/70.

d) Disappearance of Aneurysm (1 case).

Case 5. Man, born 1917. Subarachnoid hemorrhage with right hemiparesis and aphasia, Sept. 24, 1953. Left carotid angiography, Oct. 8, 1953: aneurysm of internal carotid bifurcation. Clipping was attempted, but control angiography on Nov. 2, 1953 showed that the aneurysm was still filling and the clip was visible behind the aneurysm (Fig. 10). No recurrent hemorrhage during interval. Left carotid angiography, June 5, 1958: no filling of aneurysm; position of clip changed (Fig. 11).

COMMENTS

Very little seems to be known about changes in the size of unoperated intracranial arterial aneurysms. Mount and
Taveras\(^7\) stated that in 16 patients not operated upon the aneurysm was the same size, or larger, in repeated angiograms performed after some lapse of time; they did not see any case of spontaneous thrombosis in untreated aneurysms. Poppen and Fager\(^9\) reported a case of carotid ligation for an intracranial arterial aneurysm, with subsequent expansion of another aneurysm on the opposite side. There is 1 case on record of a truly traumatic arterial aneurysm: \(^2\) a meningioma arising from the roof of the orbit was removed after preoperative angiography showing no vascular anomaly. During the operation, a small branch of the middle cerebral artery was torn and carefully clipped so as not to occlude the parent vessel. Postoperatively, the patient suffered a subarachnoid hemorrhage, and an aneurysm on the middle cerebral artery was found at the origin of the torn vessel. Taylor\(^12\) commented that a sudden decrease in intracranial pressure encourages the development and growth of an aneurysm, and may lead to

Figs. 8 and 9. Case 4. (Left) Aneurysm of the pericallosal artery. (Right) One year later: no change in size of the aneurysm.

Figs. 10 and 11. Case 5. (Left) Aneurysm of the internal carotid bifurcation after an attempt at clipping. (Right) Five years later: the aneurysm has disappeared.
subarachnoid hemorrhage if an aneurysm is already present.

Any apparent angiographic changes in the size of an aneurysm may be caused by differences in the roentgenological views and the degree of filling of the aneurysm. During recent years, we have paid particular attention to this, and have tried to reproduce the previous views as faithfully as possible, in order to eliminate this source of error.

In our series, there is a considerable difference between the development of those aneurysms that have bled and those that have not. The former generally have grown much more, whereas the growth of the latter, if any, has been slight. It should be pointed out that 2 out of the 4 showing growth without bleeding had undergone intracranial exploration after the first angiography; such exploration may be a trauma to the aneurysm, and may further its growth. In our small series, there is no instance of aneurysm not having grown, despite subarachnoid hemorrhage in the interval.

Five of the 6 aneurysms on the internal carotid artery and its bifurcation showed growth, and 3 of these were the most spectacular instances of growth after subarachnoid hemorrhage. The 6th aneurysm in this group, an aneurysm on the bifurcation of the internal carotid artery, disappeared in the interval. Two of the aneurysms of the anterior communicating artery had not changed, but the 3rd was the best instance of growth without bleeding, albeit after exploration with muscle wrapping. Among the aneurysms of the middle cerebral artery, 5 remained the same size, 3 had grown after bleeding, and 1 had grown without bleeding.

The spectacular growth of the 3 aneurysms on the internal carotid artery (illustrated by our Cases 1 and 2) suggests that a free-hanging aneurysm in a subarachnoid cistern has a better opportunity of growth than an aneurysm embedded in cerebral tissue, as aneurysms of the anterior communicating and middle cerebral arteries often are. Taylor's comment on the importance of the general intracranial pressure also may be relevant in this connection.

Our Case 1 shows that a small nubbin may grow into a large aneurysm, but this development is not certain, since we have 3 similar cases in which the nubbin was the same size after periods of up to 88 months. However, these nubbins were located on the callosomarginal and the middle cerebral arteries. It is interesting to compare these clinical findings with the investigations of Hassler and Saltzman on widening of the infundibulum of the posterior communicating artery. Our Case 1 had such a widening on the "normal" side, but this side was not investigated on first admission.

Our Case 5 must be regarded as a very rare one; it is the only case of spontaneous thrombosis in our aneurysm series. However, it shows that such thrombosis does occur, though a case reported by Hamilton and Falconer suggests that intracranial arterial aneurysms are not very prone to thrombosis. In their paper on experimental aneurysms, Black and German showed that the ratio between the width of the neck and the volume of the aneurysm must be within certain limits. If the volume is disproportionately large, or the neck is very narrow, spontaneous thrombosis occurs, and the aneurysm "stabilizes" at a smaller volume. However, in our total series of aneurysms there were several cases in which the neck of the aneurysm had been constricted at operation, but postoperative angiograms still showed good filling of the aneurysm, and no decrease in size.

In our Case 5, not even constriction of the comparatively broad neck of the aneurysm was achieved at operation, and we cannot explain why the aneurysm thrombosed; certainly no decrease in size was found immediately after the exploratory craniotomy. Höök and Johanson reported 1 case of spontaneous thrombosis of an intracranial arteriovenous malformation; Norlén reported a case of partial thrombosis, and so did Paterson and McKissock.

This study has shown how an arbitrarily selected group of aneurysms had developed at repeated angiography. It seems that aneurysms of the internal carotid artery
show a more marked increase in size after bleeding than do aneurysms in other locations; but it is debatable whether this depends on the factor we have proposed above. We have not found any definite trend in aneurysms in other locations. The development of an intracranial arterial aneurysm is influenced by many factors, chief among which are hypertension and degenerative vascular disease, though we still know far too little about these. It is, for instance, quite possible that the aneurysms in Case 4 neither bled nor grew because the patient had undergone an operation for aortic coarctation, with consequent lowering of the blood pressure in the upper half of the body.

We do not know why certain aneurysms bleed and others do not, nor, indeed, do we know exactly at what stage the increase in size takes place. The bleeding may be a consequence of an increase in size, causing a weakness of the aneurysmal wall, but it is, of course, also possible that the growth of the aneurysm takes place during the hemorrhage.

**SUMMARY AND CONCLUSIONS**

A series of 19 patients with angiographically verified intracranial arterial aneurysms was subjected to a second angiography after an interval varying from 2 weeks to 10 years, without any decisive surgical procedures having been carried out in the meantime.

Ten aneurysms definitely had grown during the interval. Six of these had caused a recurrent hemorrhage; 4 had not. Two of these 4 had been explored surgically in the meantime. Eight aneurysms had remained unchanged, whereas 1 had disappeared entirely.

We conclude that intracranial arterial aneurysms may grow even without subarachnoid hemorrhage, although the growth is much more marked in patients who have suffered hemorrhages between angiographies. Among aneurysms that have bled, those of the internal carotid artery showed the most spectacular increase in size.

Spontaneous thrombosis of an arterial aneurysm apparently is a comparatively rare occurrence.

**REFERENCES**