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A Change of Computed Tomographic Findings of Two Cases of Giant Internal Carotid Artery Aneurysms after Cervical Carotid Artery Occlusion

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ABSTRACT

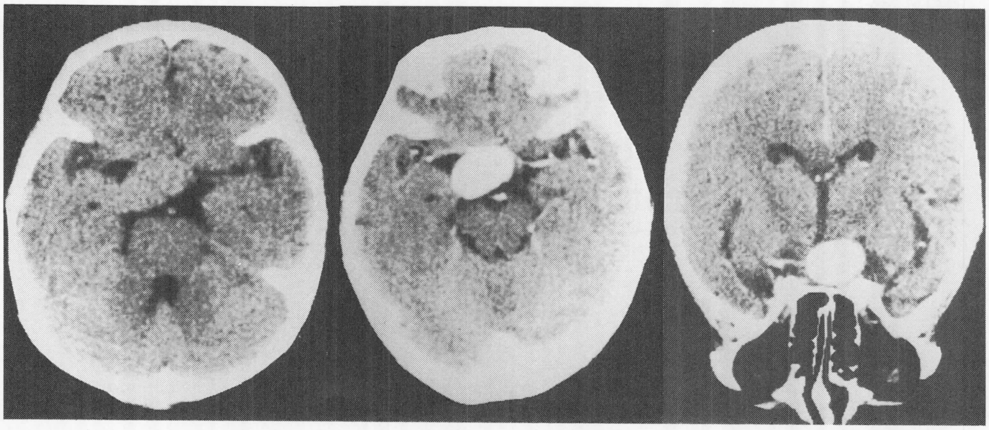
The effects of cervical carotid artery occlusion on two giant internal carotid artery aneurysms were studied by computed tomography (CT). In both cases, the density on CT findings of the thrombosed parts in the aneurysms were changed with time from hyperdensity in the immediate post operative period to isodensity and finally to decreased density. For giant aneurysms treated with occlusion of the proximal artery, CT scanning is a reliable and atraumatic method for follow-up review, providing precise information concerning actual size, thrombosis, and pathological changes.

Giant intracranial aneurysms have been defined as those over 2.5 cm in diameter[20], and they usally present clinically as intracranial masses, frequently producing dementia and cranial nerve deficits[2,7,8,31]. Partially or completely thrombosed giant aneurysms may easily be mistaken for neoplasms on the computed tomography (CT) scan unless attention is paid to its detail. On the other hand, giant internal carotid artery (ICA) aneurysms have continued to challenge the expertise of the most capable surgeons. In recent years the ligation of the ICA with or without extracranial-intracranial (EC-IC) bypass has been employed for the treatment surgically inaccessible or technically difficult intracranial aneurysm[9,12,13,19,29,30]. The purpose of this report is to describe the CT characteristics of giant ICA aneurysms and the changes in CT findings after the proximal ICA occlusion.

CASE REPORTS

Case 1

A 58-year-old woman was admitted to the Shimane Prefectural Central Hospital in July 14, 1981, because of a decrease in visual acuity and visual field defect on the left side of the left eye. Neurological examination at admission revealed left optic disc atrophy, visual acuity of 1.0 on the right and 0.1 on the left, and temporal hemianopsia of the left eye. General physical examination revealed no abnormalities and the results of the laboratory test were normal. CT scans revealed an isodense, oval mass in the suprasellar region, which was intensely and homogenously enhanced after the administration of the contrast material (Fig. 1). Left caroid angiography disclosed a giant aneurysm, measuring 3 × 3 × 2.5 cm, arising from the left internal carotid artery distal to the ophthalmic artery (Fig. 2). Right carotid angiography showed sufficient collateral flow via the anterior communicating artery on left carotid compression in the neck. Matas' test was well tolerated. On September 8, 1986, the ICA was surgically exposed in the neck, and electroencephalographic (EEG) monitoring and regional



(A) (B) (C)

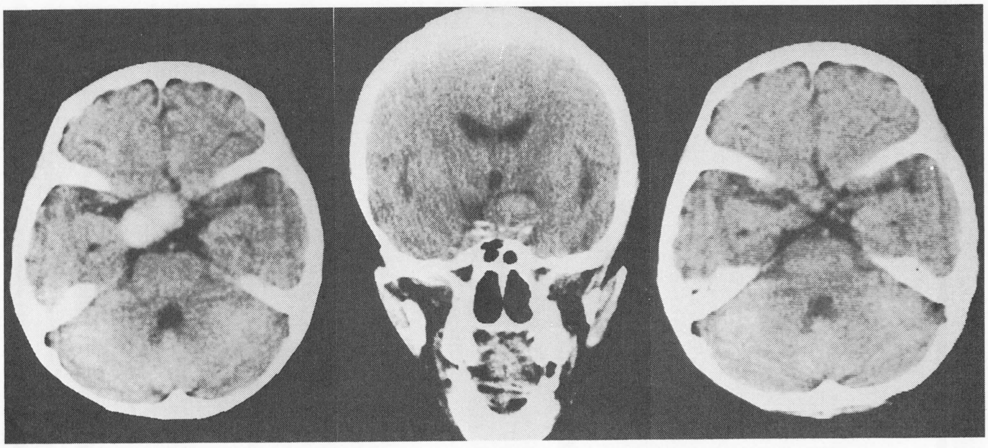
Fig. 1. CT scans before ligation of the cervical ICA in Case 1. A plain CT scan reveals an isodense, ovale mass in the suprasellar region (A), which is intensely and homogenously enhanced after the administration of the contrast material (B, C).

(A) (B)

Fig. 2. Left carotid posteroanterior (A) and lateral (B) angiograms demonstrate a giant aneurysm measuring 3 × 3 × 2.5 cm, arising from the left ICA distal to the ophthalmic artery in Case 1.

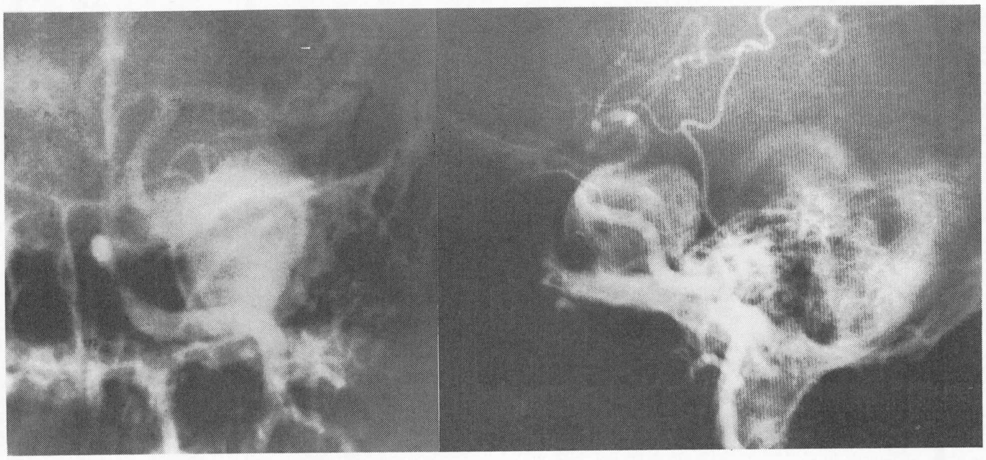
cerebral blood flow (rCBF) measured by the ^{133}Xe inhalation method during temporary ICA clamping were made. The patient's clinical condition and EEG pattern did not change during ICA occlusion for 30 min. The left rCBF was within the normal range (56 ml/100g/min) during ICA occlusion. Stump pressure was measured while the ICA was clamped and opened. The mean pressure with the ICA opened was 130 mmHg and with the ICA clamped, the pressure fell to 60 mmHg. Therefore, we completely ligated the ICA in the neck on September 19.

An axial plain CT scan 2 weeks after complete occlusion of the ICA showed that the isodense mass had changed to a hyperdense mass, suggesting that the aneurysm had thrombosed (Fig. 3-A). Contrast enhancement was not observed. Subsequently, CT scans were performed repeatedly. A coronal plain CT scan on October 27 showed that the aneurysm gradually lost its well-defined margin, and the density continued to decrease, more markedly in the periphery (Fig. 3-B). The density became isodense compared with nearly brain matter with a slight



(A) (B) (C)

Fig. 3. Repeat plain CT scans after ligation of the cervical ICA in Case 1. The isodense mass has changed to a hyperdense mass 2 weeks after carotid occlusion (A). A coronal CT scan 5 weeks after ligation shows that the aneurysm gradually loses its well-defined margin, and the density is decreased (B). The density becomes isodense with a slight reversal of the mass effects 6 months after surgery (C).



(A) (B)

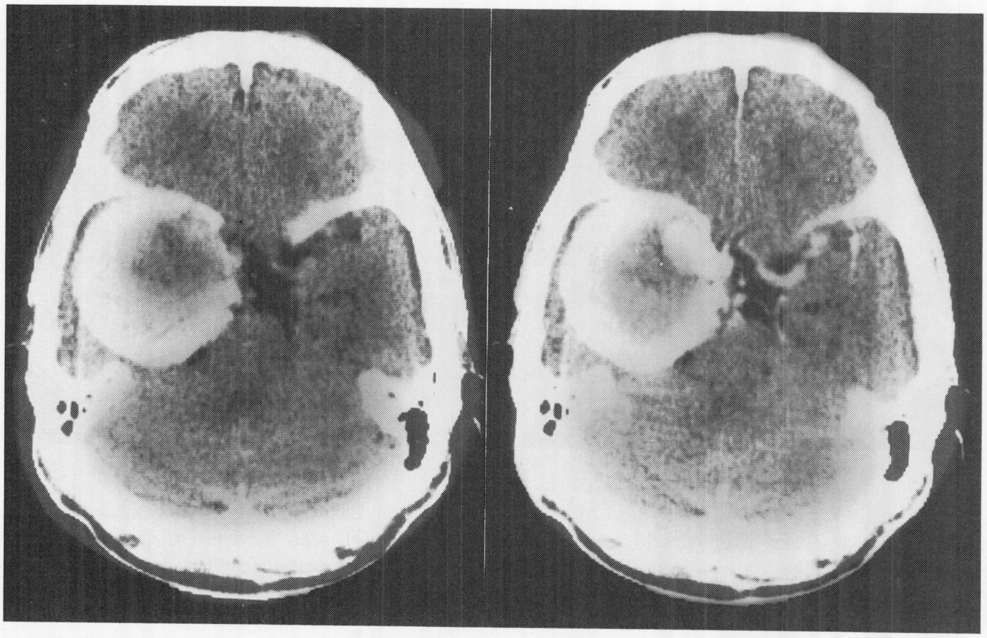
Fig. 4. Left anteroposterior (A) and lateral (B) carotid angiograms obtained in 1970 demonstrate a giant aneurysm arising from the intracavernous portion of the ICA in Case 2.

reversal of the mass effects six months after surgery (Fig. 3-C). No enhancement was noted after administration of contrast medium. Digital subtraction angiography conducted on September 29 showed that the left anterior and middle cerebral arteries were filled from the right carotid system, and the aneurysm was not visualized any longer. The patient was discharged on October 30, 1986 and at present one year after carotid occlusion the visual field defect improved, probably due to shrinkage of the aneurysm and a decrease in the mass effect on the adjacent optic pathways.

Case 2

A 43-year-old man underwent left carotid angiography at the Hiroshima Prefectural Hiroshima Hospital in 1970 due to visual disturbance of the left eye, which disclosed a giant aneurysm on the intracavernous portion of the ICA (Fig. 4). No special treatment was provided at that time.

For the purpose of re-examining the giant aneurysm, the patient was admitted to the Shimane Prefectural Central Hospital on May 10, 1982. CT scans showed that there was a huge, partly calcified, globoid lesion of high density in



(A) (B)

Fig. 5. CT scans obtained in 1982 on admission demonstrate a giant aneurysm measuring 6 × 6 × 7 cm in size in Case 2. Plain CT scan (A) shows a spherical lesion with a partially calcified capsule occupying a substantial part of the left middle cranial fossa. Postcontrast scan (B) shows enhancement of an eccentric area and a rim of peripheral region.

(A) (B)

Fig. 6. Left carotid posteroanterior (A) and lateral (B) angiograms on admission in Case 2. A large partially thrombosed aneurysm of the ICA is shown associated with upward displacement of the M-1 portion of the MCA.

the left middle cranial fossa, which eroded the basal bone structures including the sphenoidal ridge and the petrous pyramid (Fig. 5). The lesion was 6 × 6 × 7 cm in size. Following the administration of the contrast medium, the eccentric and peripheral zones were intensely enhanced (so-called target sign)[17,25]. Left carotid angiography disclosed a partially thrombosed giant aneurysm from the left ICA with a marked elevation of M-1 portion of the middle cerebral artery by a large avascular mass (Fig. 6). The patient was treated by combined ICA occlusion

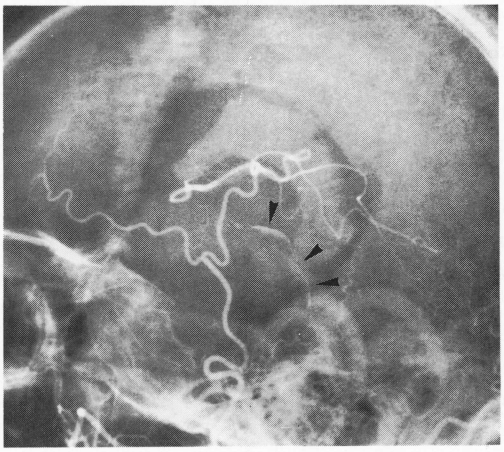
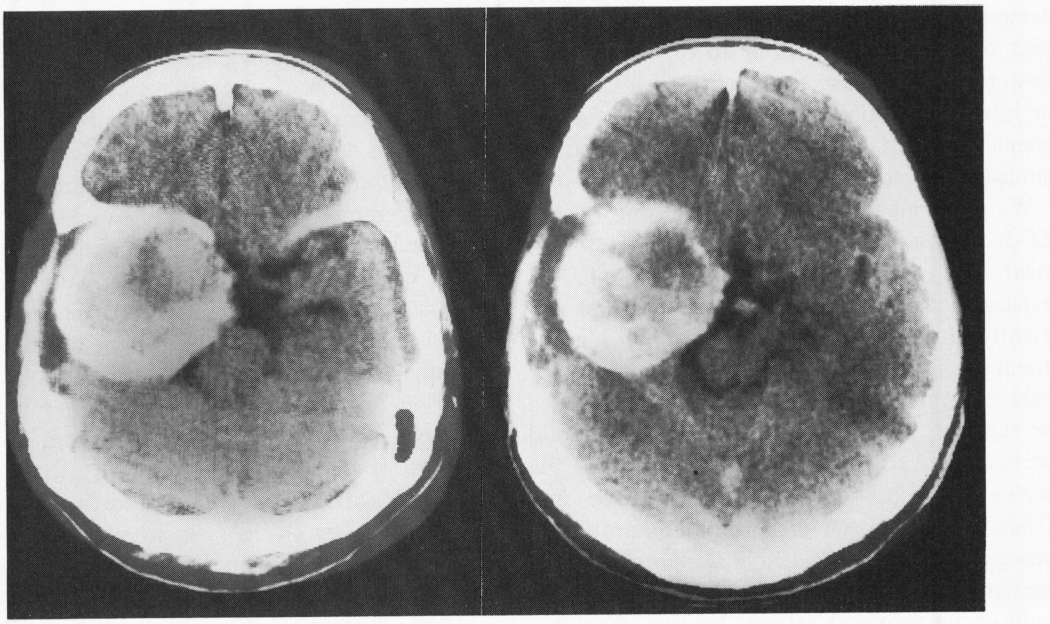


Fig. 7. Left lateral carotid angiography in Case 2 demonstrates that the MCA is filled by STA-MCA anastomosis. A shell-like calcification (arrowheads) is visible.



(A) (B)

Fig. 8. CT scans after complete closure of the cervical ICA in Case 2. A plain CT scan 18 days after occlusion shows the density of the eccentric area is slightly increased, indicating that the lumen is thrombosed (A). A post-contrast CT scan 4 months after complete occlusion demonstrates that the attenuation values of the eccentric round part and the peripheral zone decreased with time (B).

in the neck and superficial temporal artery to middle cerebral artery anastomosis (Fig. 7). A plain CT scan made 18 days after complete closure of the Selverstone clamp showed that the attenuation values of the eccentric part had increased (Fig. 8-A), suggesting that the aneurysm had thrombosed. Repeat CT scans with contrast medium enhancement showed that the attenuation values of the eccentric round part and the peripheral zone had decreased with lapse of time (Fig. 8-B). The aneurysm had gradually lost its well-defined margin and inhomogenous appearance was observed inside the ring enhancement. The patient is doing well at present.

DISCUSSION

Although giant aneurysms have been detected in infancy and in childhood[16,18], there are, generally, two hypotheses on the explanation of the mechanisms involved in the growth of giant aneurysms; one presumes that the volume of an aneurysm increases due to rupture[34], and the other suggests that the volume increases without rupture[33,36]. Artmann et al[1] in reviewing 23 reported cases of growing giant aneurysms including their own case described that in cases with repeated subarachnoid hemorrhage, the change in the size of the aneurysm could be presumed to result from the rupture, but in cases without rebleeding, changes can result from slow and steady growth or by a sudden expansion. Although the giant aneurysm of Case 2 was large from the beginning some 12 years ago, the hemodynamic injuries to the wall due to the impact of the blood stream or to turbulent blood flow may have caused the repeated intramural or intrathrombotic hemorrhages and the subsequent processes of thrombosis and thrombus organization, leading to enlargement.

We performed cervical ICA occlusion without EC-IC bypass in Case 1 and with in Case 2. Cervical ICA occlusion with or without EC-IC bypass has become used in the treatment of surgically inaccessible or unclippable giant aneurysms of the ICA[9,12-14,19,29,30]. However, not only ischemic complication[5,7,10,11] but also growth or rupture[4,6-8,12,28] of the aneurysm after carotid occlusion have been sometimes reported. In the series of Heros et al[12] and Ferguson and Drake[7,8], Some patients showed deterioration of vision, which was presumably attributable to enlargement of the aneurysm due to thrombosis induced by proximal arterial ligation. Aneurysmal enlargement may be ascribed to progression of thrombosis in the aneurysm[4,6]. Ischemic complication is one of the most important events after carotid occlusion for an aneurysm, but it is controversial to perform EC-IC bypass as a prophylactic measure in all patients with planned carotid occlusion[12,13,21,24]. Heros et at[12] and Peerless and Durward[24] have recommended that determination should be made whether a patient could tolerate acute occlusion of the ICA and if the collateral flow was judged to be satisfactory by angiography, rCBF, etc., EC-IC bypass should not be carried out. On the contrary, Spetzler et al[29,30] and Gelber and Sundt[9] have used an EC-IC bypass in conjunction with abrupt or staged occlusion of the ICA in all patients. Spetzler et al[29,30] have advocated that the question of delayed neurological complication after carotid occlusion has not yet been resolved and that EC-IC bypass had the theoretical advantage of increasing a pressure head distal to the aneurysm, thus further reducing the flow in the region of the aneurysm orifice.

The patterns of CT scans with and without contrast medium enhancement vary depending on whether the aneurysm is nonthrombosed, partially thrombosed, or completely thrombosed[1,15,19,22,23,25-27,31,32,35,37]. Nonthrombosed giant aneurysms appear as sharply delineated round or oval masses with a slightly increased homogenous density compared to the nearly normal brain on plain CT scans and they are intensely and homogenously enhanced after administration of contrast medium[15,19,26]. Partially thrombosed giant aneurysms appear as areas of mixed density, where the vascular lumen and the thrombosed part may be sometimes difficult to differentiate due to the protean density of the thrombus. On postcontrast CT scan, the central or eccentric area shows an intense and homogenous enhancement, corresponding to the residual lumen[15,19,23,25-27,32,37]. This pattern of partially thrombosed giant aneurysms is called a target sign[17,25]. The CT scan of completely thrombosed giant aneurysms shows a roundish lesion of slightly increased density with a mottled appearance, the well or ill defined limits being dependent on the presence of a calcified wall before administration and no enhancement after administration of contrast medium[15,19,25-27]. In the present study, the preoperative CT scans showed a nonthrombosed giant aneurysm of the left ICA in Case 1, and partial thrombosis of the aneurysm with the typical target sign in Case 2. Usually, after carotid occlusion, plain CT scans show a marked increase in attenuation values of the aneurysmal lumen, reflecting complete thrombosis immediately after the operation, and thereafter the aneurysm gradually loses its well-defined margin and the density continues to decrease more markedly in the periphery to the center of the aneurysm, but no enhancement is noted after contrast infusion during this course[15,19,26,27]. In our patients, after complete occlusion of the ICA almost similar changes in CT scans were observed. In partially or completely thrombosed giant aneurysms, ring-shaped contrast medium enhancement of the aneurysmal wall or shell-like calcification may be observed on CT scans, and the former characteristic is deemed to have resulted from enhancement of the vascularized wall[1,15,23,25-27,31,35]. Recently it has been shown that extracranial vessels contained an extensive vasa vasorum network[3], but this system was not demonstrated in intracranial vessels[3,38]. In the histological examination of the giant aneurysmal specimens obtained at operation or necroscopy, many small vessels resembling vasa vasorum were observed in the aneurysmal wall[2,14,19,25,27,31,32,36,37] and small vascular channels without endothelial lining or wall structure were occasionally observed inside the aneurysmal thrombus[14,32,35,36]. Pinto et al[25] have postulated that the thickened fibrous wall surrounding the aneurysmal lumen containing many small vessels was formed from multipotential cells of the adjacent meninges in response to the subarachnoid mass[25]. Furthermore, we have proposed that the wall of the giant aneurysm was nourished not only by vasa vasorum due to meningeal reaction outside the aneurysm but also internally by vasa vasorum derived from the lumen of the parent artery itself[14].

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