

Iatrogenic Dissecting Aneurysm of the Internal Carotid Artery

K. Kinugasa¹, T. Yamada¹, T. Ohmoto¹, and K. Taguchi²

Departments of ¹Neurological Surgery and ²Pathology, Okayama University Medical School, Okayama, Japan

Summary

A secondary dissecting aneurysm after arterial injury has been reported as a complication of surgery. We encountered this phenomenon in a patient with a meningioma of the sphenoid ridge. Despite successful clipping of a small rupture in the anterior wall of the proximal portion of the internal carotid artery, the patient became drowsy 30 days after surgery and developed right hemiparesis followed by fatal bleeding from the internal carotid artery. Histological examination revealed a traumatic dissecting aneurysm which had developed from the tear in the intima and extended into the dural ring and the bifurcation of the carotid artery. The characteristic clinical and histopathologic findings are shown. Methods for early recognition and the possibility of immediate treatment are briefly discussed.

Keywords: Dissecting aneurysm; internal carotid artery; ischaemia; traumatic aneurysm.

Introduction

Most traumatic intracranial aneurysms occurring after a head injury and surgical trauma to a cerebral artery have been commonly reported to be saccular aneurysms [3, 10, 21]. Nontraumatic dissecting aneurysms of a cerebral artery rarely occur in young patients with no previous significant clinical history [6, 7], as they result from a number of aetiologic factors and pathological mechanism [8, 14, 15, 17, 19, 20]. Traumatic dissecting aneurysms of an intracranial artery are unusual [1, 8, 13, 20]. Most reported cases have been fatal, and almost all patients died within 1 week of the onset of symptoms. This statistic most likely results from the difficulty of diagnosing dissecting aneurysms or cerebral dissection based on angiographic findings alone. We discuss the case of a patient with a massive intracerebral haemorrhage presumably induced by iatrogenic injury to emphasize the rapid course of this phenomenon and the necessity of early recognition.

Case Material

A 56-year-old man was hospitalized in our institution with a 2-month history of progressively deteriorating of visual acuity in his left eye.

Examination

No abnormality was found during the physical examination. A neurologic examination disclosed hemianopsia of the lower temporal quadrant without loss of central vision in the left eye. Examination of the optic fundi revealed neither papilloedema nor optic atrophy. The oculomotor, abducens and trochlear nerves were intact. Magnetic resonance imaging (MRI) revealed a large enhancing tumour arising from the sphenoid ridge with attachment to the left internal carotid artery, the optic nerve and the lateral wall of the cavernous sinus (Fig. 1). Left internal carotid angiograms showed superior displacement of the supraclinoid portion of the internal carotid artery, with superior displacement of the proximal M1 seg-

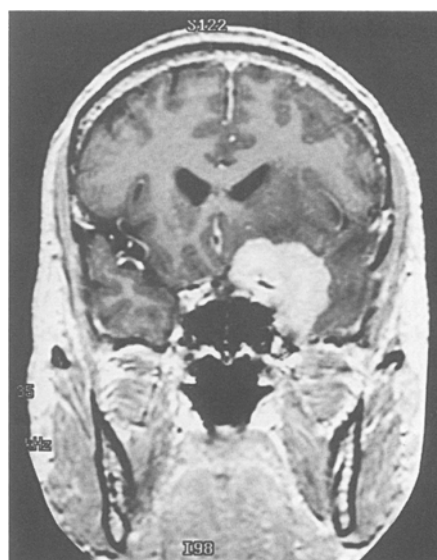


Fig. 1. Gadolinium-enhanced MRI, coronal view, showing a sphenoid ridge meningioma extending to the cavernous sinus



Fig. 2. Left internal carotid angiograms, anteroposterior (A) and lateral (B) views, showing superior displacement of the internal carotid artery and faint tumour stain (arrowheads). The tumour is fed by the dural branches of the internal carotid artery

ment of the middle cerebral artery (Fig. 2 A, B). External carotid angiograms revealed no vascular Blush from the dural branches of the external carotid artery.

Operation

A left frontotemporal craniotomy was carried out, and a well-circumscribed hard tumour extending into the anterior territory of the middle cranial fossa was found originating from the inner sphenoid ridge. The tumour was contiguous with the proximal portion of the internal carotid artery, the M1 segment of the middle cerebral artery, and the optic nerve. The tumour was easily separated from the lateral wall of the cavernous sinus, carotid artery and optic nerve, and removed piecemeal. During dissection of the tumour capsule from the internal carotid artery, unexpected vigorous bleeding occurred from a 0.5×0.5 mm tear in the anterior wall of the internal carotid artery. No aneurysmal wall or bulge was seen in the artery. Compressive haemostasis was attempted several times with small pieces of Bemsheet (Kawamoto Bandage Material Co., Ltd., Osaka, Japan) but could not be obtained. To control bleeding, temporary Sugita clips were gently applied to the proximal internal carotid artery, the proximal A1 segment of the anterior cerebral artery, and the proximal M1 segment of the middle cerebral artery.

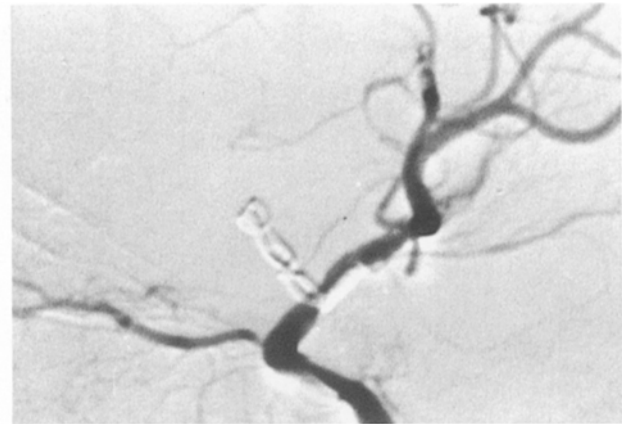


Fig. 3. An angiogram reveals narrowing of the internal carotid artery at the site of the tear caused by a clip

With silk ligature it was attempted to close the hole, but it failed because of the friable nature of the arterial wall, and the hole grew larger. Three-quarters of the circumference of the artery then were wrapped with larger pieces of Bemsheet. (Wrapping the entire artery would have damaged important perforating vessels in the ventral wall of the internal carotid artery). A right-angled Sugita clip, with a fenestration 5 mm in diameter and blades 6 mm long, was used to secure the Bemsheet, sparing the perforating arteries and the internal carotid artery. The intention was to apply the fenestrated portion of the clip to compress the Bemsheet on the hole in the vessel. The temporary clips were removed in less than 6 minutes. Inspection and palpation of the internal carotid artery indicated that its blood flow was functioning well. The tumour was completely removed along with its dural attachment and the roof and lateral wall of the cavernous sinus were coagulated. Intra-operative angiography was carried out to confirm the patency of the internal carotid artery. This patency was sufficiently maintained through the segmental narrowing of the carotid artery by the application of a clip.

Postoperative Course

The patient's postoperative course was uneventful. A left internal carotid angiogram taken 1 week after surgery showed the same configuration of the supraclinoid internal carotid artery as at the time of the intra-operative angiogram (Fig. 3). Follow-up computed tomogram (CT) showed low density of the surgical bed with resolution of the mass. Thirty days after surgery, the patient suddenly developed hemiparesis on the right side and became lethargic. A CT scan showed a large, low density lesion around the Sylvian fissure. We believed this change resulted from cerebral infarction associated with narrowing of the carotid artery, and anticoagulant therapy for cerebral infarction was begun. The patient's symptoms showed some improvement. On the second day, he suddenly became comatose and unresponsive to verbal stimuli and had fixed, dilated pupils and decerebrate posture, followed in a few minutes by irregular respiration. A CT scan demonstrated a haemorrhage in the left Sylvian fissure and intracerebral haemorrhage involving the left frontal lobe. The patient remained deeply comatose and died 6 days later.

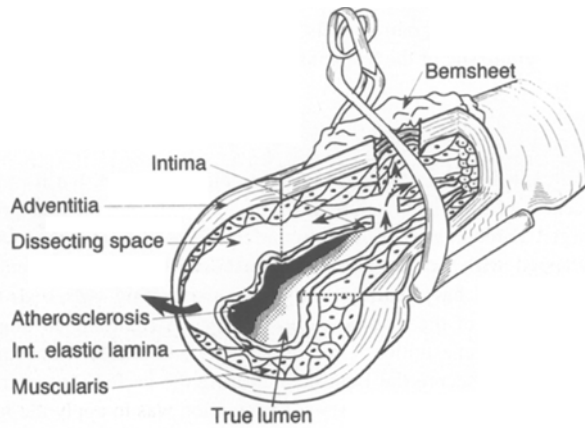
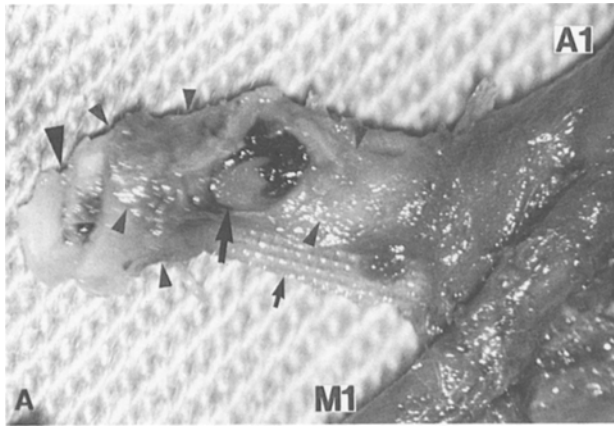


Fig. 4. (A) Gross dissection of the paraclinoid segment of the internal carotid artery. The darkened segment (small arrowheads) is caused by the intramural haematoma. A clip-crush artifact (small arrow) is seen in the adventitia distal to the hole (large arrow). The cut end of the internal carotid artery is split (large arrowhead). *A1* A1 segment of the anterior cerebral artery; *M1* M1 segment of the middle cerebral artery. (B) Schematic representation of the paraclinoid segment of the internal carotid artery. The plane of dissection is submedial

Pathologic Examination

An autopsy was performed, and pertinent observations were limited to the paraclinoid portion of the left intracranial internal carotid artery and the central nervous system. The brain was markedly oedematous, with marked transtentorial herniation on the left side and bilateral tonsillar herniation. Ischaemic infarction had occurred within the distribution of the left middle cerebral artery. A massive haemorrhage occurred on the orbital surface of the left frontal lobe, and a moderate amount of blood remained in the left Sylvian fissure and the basal cisterns. The anterior and middle cerebral arteries appeared normal. The tear in the vessel wall was surrounded by Sugita's fenestrated clip and Bemsheet measuring 7 × 7 mm at level 5 mm distal to the carotid dural ring of the internal carotid artery. The fatal bleeding did not result from the original tear in the internal carotid artery during surgery. After the clip and Bemsheet were removed, the media appeared through the hole in the artery wall. A part of the wall opposite the tear was pinched by the clip.

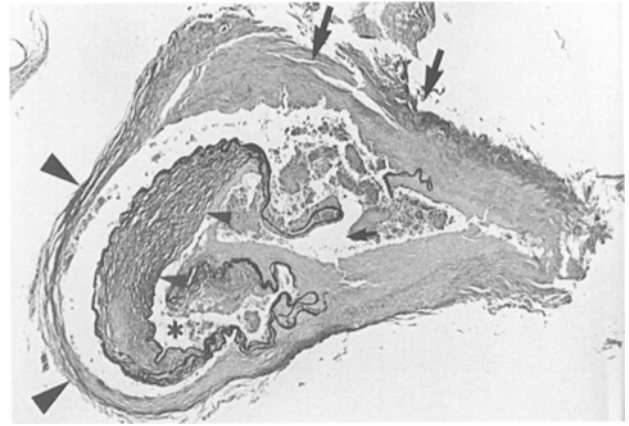


Fig. 5. Microscopic view of a transverse section of the proximal portion of the left internal carotid artery. The tear (small arrow) is seen in the internal elastic lamina in normal thickness. A marked intimal thickening (small arrowheads) can be seen on the left side of the true lumen (asterisk). The crescent of the dissecting aneurysm is surrounded by one thick (large arrows) and one thin (large arrowhead) layer of smooth muscle and the internal elastic lamina, respectively (elastic V.G. stain × 40)

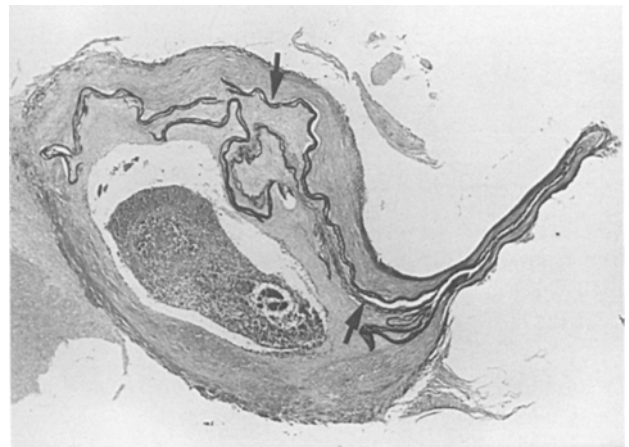


Fig. 6. Microscopic view of a transverse section of the distal portion of the left internal carotid artery. The intima is compressed by the media including the mass of blood, and the lumen has collapsed (arrowheads) (elastic V.G. stain × 49)

A length of the vessel 5 mm below and 3 mm above the hole was firm and dark brown in a third of the circumference of the artery, but the vessel was normal in size except for the portion held by the clip. This discolouration appeared to continue to the tear and to result from the accumulation of clot in the intramural lumen. The dissection cleft of the wall was found at the cut end of this abnormal segment (Fig. 4 A, B). The dissecting space, including the thrombus, extended for a short distance from the tear toward both the carotid dural ring and the bifurcation of the internal carotid artery. The point of fatal bleeding, as evidenced by the subadventitial haematoma, was just distal to the dural ring of the internal carotid artery, indicating a slit in the adventitia.

Microscopic study of serial sections of the left internal carotid

artery just proximal to the hole revealed a large crescent-shaped lumen of the dissecting aneurysm, which had separated the media from the internal elastica. The separation involved two-thirds of the circumference of the vessel. A part of the intima was markedly hypertrophic, with a calcified and cholesterol cleft, but its adventitia and media were thin and atrophic. The internal elastica, without hypertrophy of the intima, was interrupted on the opposite side, where a thick layer of smooth muscle and a thin layer of adventitia were present (Fig. 5). The lumen of the dissecting aneurysm gradually appeared on the surface of the more proximal sections. Near its proximal end, the haematoma had focally penetrated the loose media with adjacent rupture of the thinner adventitia. The section just distal to the hole revealed that the lumen of the dissecting aneurysm was located between the internal elastica and the adventitia, and was generally surrounded by a layer of smooth muscle and filled with blood. The lumen of the vessel was completely occluded by folds, and a convoluted slit of the internal elastica on one side was surrounded by an intramural thrombus. Portions of all the layers were compressed by the clip, but the intima was intact (Fig. 6). There was no evidence of an antecedent abnormality, such as invasion by tumour cells, in the wall of the artery or adjacent to the dissection. The finding of the neuropathologic examination was a ruptured dissecting aneurysm without dilation of the paraclinoid segment of the internal carotid artery.

Discussion

Yonas *et al.* [23] separated the clinical manifestations of dissecting aneurysms into two groups, which correlate with the plane of dissection. In the first group, blood is found between the internal elastica and the media. This type can result from trauma and is ascribed to a defect in the internal elastica. In the second group, dissection occurs within the media or adventitia and presumably results from rupture of the vasa-vasorum.

When the tumour in our patient was dissected from the internal carotid artery, no definite aneurysmal wall or discolouration was seen, but the bleeding originated from a tear in this artery. This bleeding was believed to result from injury to the arterial wall or compression by the tumour. Otherwise, we thought that a small feeding artery from the internal carotid artery might have avulsed while the tumour capsule was being dissected. This bleeding was controlled by wrapping and a clip.

The cause of the fatal bleeding in our patient was not apparent until histological sections of the involved artery were examined. There had been no hint of true, false or dissecting aneurysms of the internal carotid or middle cerebral arteries on either intra- or postoperative angiograms. The autopsy revealed that the dissecting cavity was caused by the luminal extension of blood between the internal elastic lamina and the smooth muscle, and had extended mainly to

the dural ring of the internal carotid artery. However, the intramural haematoma extended into the smooth muscle toward the carotid bifurcation.

The destruction of the internal elastica and intima was probably caused by the interruption of the carotid artery during tumour removal, or by the shearing stress on the arterial wall from applying the temporary clip. The disrupted site was found in the internal elastica without atheromatous degenerative changes and near the hole in the wall. The dissection resulted presumably because the injury in the internal elastica allowed blood from the lumen to force its way between the elastica and the smooth muscle, and especially because of the increased flow velocity in the carotid, which was narrowed by the fenestrated clip. The accumulating blood pushed the internal elastica toward the vessel lumen, narrowing or occluding the lumen. Perhaps the patient's initial hemiparesis was caused by the collapse of the lumen when blood, entering the wall outside the internal elastic lamina, longitudinally dissected the artery. The adventitia and smooth muscle were markedly thin or atrophic at the site of intimal hypertrophy or atherosclerosis in the proximal carotid artery, and the external elastic membrane was absent. Thus, the restricted blood flow advanced proximally in the dissecting space and fatal bleeding originated through these thinner outer layers of the vessel wall.

Many authors have reported the angiographic findings of intracranial dissecting aneurysms, including the string sign, irregular tapering or narrowing, occlusion, a rosette, an intimal flap, proximal and distal dilation, a double lumen, retention of contrast medium and intramural pooling [4, 12, 22]. Arteriography performed 5 to 7 days after a penetrating head injury seems to be the best method of demonstrating a traumatic aneurysm [3, 4, 6, 12, 22].

Diagnosing a dissecting aneurysm is not easy, even with angiography. The difficulty arises in differentiating dissection from vasospasm and an atherosclerotic lesion [12, 22]. In our patient, the dissecting aneurysm was not seen on radiography. Except for slight narrowing of the carotid artery, intra- and postoperative carotid angiograms showed the same configuration of the supraclinoid internal carotid artery as in the initial angiogram. The luminal narrowing was construed as contraction of the vessel from compression with cotton and the fenestrated clip. In fact, the narrowing occurred in the adventitia with the intact elastica of the clip-crush artifact, as revealed in the microscopic

section. An angiogram may not show the characteristic findings of intracranial dissecting aneurysms because either thrombus forms in the small lumen of the lesion or the dissecting aneurysm may occur later than 1 week after arterial injury. Cerebral arteriography should be employed repeatedly once this diagnosis is ascertained.

Some authors advocate conservative and anticoagulant therapy for patients with a cervical carotid dissecting aneurysm [5, 16, 18]. Anticoagulant therapy used in this case, however, might have accelerated bleeding because of the possibility of extending a pre-existing dissection [2]. If all parts of the arterial wall – the intima, elastica, media, and adventitia – could have been repaired with a microvascular suture, perhaps no blood would have flowed into the lumen of the dissecting aneurysm through the tear. This procedure would have protected against extension of the dissection.

The possibility of a postoperative dissecting aneurysm should be kept in mind if an arterial wall is damaged or bleeding occurs during an operation. A dissecting aneurysm should be suspected in any patient whose clinical condition deteriorates within 2 weeks after trauma [10]. Evaluations using MRI and digital subtraction angiography can detect a dissecting aneurysm soon after surgery. If a dissecting aneurysm is seen on these images, a surgical bypass of the aneurysm should be carried out immediately.

We might also conjecture that re-establishing blood flow, perhaps with an immediate external carotid-middle cerebral anastomosis followed by ligation of the left internal carotid artery, might have halted the rebleeding while maintaining perfusion of the cerebrum. If the tumour infiltrates the cavernous sinus, sella, clivus and the internal carotid artery, the treatment outlined by Sekhar, *et al.* [11], should be considered. This treatment entails reconstruction using a saphenous vein graft to replace the internal carotid artery. Covering the saphenous vein with an artificial vascular graft might help prevent the pseudoaneurysm and aneurysmal dilatation and secure patency of the graft [9]. The internal carotid artery, when involved by tumour of the skull base, is a significant impediment to aggressive resection of this lesion. We have to carefully work around the artery and avoid injury to the vessel. If a small tumour remnant cannot be resected, subsequent radiosurgery may be considered.

The diagnosis of a dissecting aneurysm is often

delayed or overlooked because the surgeon's attention is drawn toward the accompanying intracranial haematoma [6]. Even if a postoperative angiogram does not show true or false aneurysms, a dissecting aneurysm should be considered in every patient whose condition deteriorates a few weeks after the removal of a tumour adhering to cerebral arteries or when vessels are injured during surgery [10]. These lesions can be recognized earlier using angiography more frequently and by more sophisticated scanning.

Acknowledgements

The authors thank Hideki Wakimoto for editorial assistance and Seiji Awai for the histological preparations.

References

1. Amagasa M, Sato S, Otabe K (1988) Post-traumatic dissecting aneurysm of the anterior cerebral artery: case report. *Neurosurgery* 23: 221–225
2. Berger MS, Wilson CB (1984) Intracranial dissecting aneurysms of the posterior circulation. Report of six cases and review of the literature. *J Neurosurg* 61: 882–894
3. Burton C, Velasco F, Dorman J (1968) Traumatic aneurysm of a peripheral cerebral artery: review and case report. *J Neurosurg* 28: 468–474
4. Friedman AH, Drake CG (1984) Subarachnoid hemorrhage from intracranial dissecting aneurysm. *J Neurosurg* 60: 325–334
5. Friedman WA, Day AL, Quisling RG, Sybert GW, Rhoton AL Jr (1980) Cervical carotid dissecting aneurysms. *Neurosurgery* 7: 207–214
6. Grosman H, Fornasier VL, Bonder D, Livingston KE, Platts ME (1980) Dissecting aneurysm of the cerebral arteries: case report. *J Neurosurg* 53: 693–697
7. Hart RG, Easton JD (1983) Dissections of cervical and cerebral arteries. *Neurol Clin North Am* 1: 155–182
8. Johnson AC, Graves VB, Pfaff JP Jr (1977) Dissecting aneurysm of intracranial arteries. *Surg Neurol* 7: 49–52
9. Kinugasa K, Sakurai M, Ohmoto T (1993) Contralateral external carotid-to-middle cerebral artery graft using the saphenous vein: case report. *J Neurosurg* 78: 290–293
10. Parkinson D, West M (1980) Traumatic intracranial aneurysms. *J Neurosurg* 52: 11–20
11. Sekhar LN, Sen CN, Jho HD (1990) Saphenous vein graft bypass of the cavernous internal carotid artery. *J Neurosurg* 72: 35–41
12. Senter HJ, Sarwar M (1982) Nontraumatic dissecting aneurysm of the vertebral artery: case report. *J Neurosurg* 56: 128–130
13. Shaw C-M, Foltz EL (1968) Traumatic dissecting aneurysm of middle cerebral artery and carotid-cavernous fistula with massive intracerebral hemorrhage: case report. *J Neurosurg* 28: 475–479
14. Shillito J Jr (1964) Carotid arteritis. A cause of hemiplegia in children. *J Neurosurg* 21: 540–551
15. Stehbens WE (1972) Pathology of the cerebral blood vessels. Mosby, St Louis, pp 351–470

16. Stringer WL, Kelly DL Jr (1980) Traumatic dissection of the extracranial internal carotid artery. *Neurosurgery* 6: 123–130
17. Turnbull HM (1915) Alterations in arterial structures, and their relation to syphilis. *QJ Med* 8: 201–254
18. Watridge CB, Muhlbauer MS, Lowery RD (1989) Traumatic carotid artery dissection: diagnosis and treatment. *J Neurosurg* 71: 854–857
19. Wisoff HS, Rothballer AB (1961) Cerebral arterial thrombosis in children: review of literature and addition of two cases in apparently healthy children. *Arch Neurol* 4: 258–267
20. Wolman L (1959) Cerebral dissecting aneurysms. *Brain* 82: 276–291
21. Yamaura A, Makino H, Hachisu H, Takemiya S (1978) Secondary aneurysm due to arterial injury during surgical procedures. *Surg Neurol* 10: 327–333
22. Yamaura A, Watanabe Y, Saeki N (1990) Dissecting aneurysms of the intracranial vertebral artery. *J Neurosurg* 72: 183–188
23. Yonas HY, Agamanolis D, Takaoka Y, White RJ (1977) Dissecting intracranial aneurysms. *Surg Neurol* 8: 407–415

Correspondence: Dr. K. Kinugasa, Department of Neurological Surgery, Okayama University Medical School, 2-5-1 Shikata-cho, Okayama 700, Japan.