

DISSECTION OF THE ANTERIOR AND MIDDLE CEREBRAL ARTERY WITH FATAL ISCHEMIA FOLLOWING KICKS TO THE HEAD*

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Summary

A 19-year-old male received kicks to his head in the course of a fight with other young people. On admission to hospital he became drowsy and developed a hemiparesis and a facial paralysis on the right. After 3–4 days of continuous improvement the patient suddenly fell unconscious again and died on the seventh day after the trauma due to an increase of the intracranial pressure. Neuropathologic examination revealed an extensive dissection of the wall of the left anterior and middle cerebral artery. The age of the recurring infarction in the dependent areas of blood supply corresponded to the biphasic clinical course.

Key words: Blunt head trauma; Cerebral arterial dissection; Stroke

Introduction

Laminar dissection of cerebral arteries is a well-known form of vascular lesion which is often of traumatic origin [8,10,18,20,21,25,27,30,35,41,44,53,55,56] but may also occur spontaneously, with and/or without preceding vascular disease such as inflammation and others [1,3,4,6,7,9,11,17,19,21,22,33,34,38–40,44,50,53].

Traumatic or mechanical origin of cerebral arterial dissection has been reported under various conditions, such as traffic accidents [18,25,27,43,49], sports and fight injuries [10,25,30,48,52,55,56], and manipulation of the cervical spine [2,14–16,23,26,29,31,32,46,47,51]. In the observations reported in the literature, the vertebro-basilar arteries were most frequently involved [8,10,14–16,26,31,32,46–48,56] followed by the carotid arteries and their branches [2,29,30,35,37,45,49,55].

The laminar arterial dissection is, as a rule, followed by intramural bleeding, often with the formation of an intramural hematoma in the dissected space. The

*Dedicated to Professor Dr. Günter Ule, Director emeritus of the Institute of Neuropathology of the University of Heidelberg on the occasion of his 70th birthday.

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hematoma may compress the vascular lumen, thus causing ischemia in the dependent territories of blood supply [2,5—7,10,13,28,44,54].

Here we report the observation of a surprisingly extensive laminar arterial dissection in the brain of a young man who had received kicks to his head.

Case Report

A 19-year-old normally constituted drunken male became involved in a fight with other youngsters. Witnesses said that he had fallen down and received kicks to his head.

He had to be admitted to the hospital where he arrived in a drowsy state and exhibited facial paralysis and hemiparesis on the right. On general examination he did not show any signs of traumatic impact to his body or head; he only exhibited two bite-lesions of his tongue. To pain stimuli he answered with extension spasms on the right and non-coordinated reactions on the left. He could only breathe with respirator support.

Hematologic studies revealed a leukocytosis of 14 000/ml. All other findings were normal.

CT scans obtained shortly after the admission only showed brain edema of unknown origin. However, 1 day later control scans revealed signs of ischemic infarction in the left frontal lobe, centered to the supply territories of the left anterior and middle cerebral artery. Some further hypodense foci were found in the left temporo-parietal region.

Doppler sonographic examination of the cephal-ad vessels did not provide evidence of vascular occlusion. In addition, the results of an ultrasound echocardiography rendered cardiogenic embolism unlikely.

From the third day on, after admission, his condition improved significantly; he became alert and could breathe spontaneously.

However, on the sixth day he suddenly deteriorated again, fell unconscious and exhibited signs of raised intracranial pressure. CT scans revealed severe edema of the left cerebral hemisphere with a shift of the midline structures to the right. The intracranial pressure rise could not be managed. On the seventh day no further brain activity could be registered by electroencephalography. Respirator treatment was stopped on the eighth day, when he showed clear signs of coma dépassé.

General medico-legal examination revealed only some slight, several days-old excoriations of his elbows and a minor bleeding into the left temporal muscle. Further, a recent thrombotic occlusion of the jugular veins and a purulent tracheobronchitis were found.

Neuropathological Findings

On gross inspection his brain (1240 g) showed flattening of the gyri and narrowing of the sulci. The coloration of the cortical grey matter varied considerably from pale-white to dark-brownish, in particular in the fronto-parietal region of the left hemisphere. The latter exhibited clear features of recent ischemic infarction with hemorrhage of the left basal ganglia (putamen and dorsal part of the

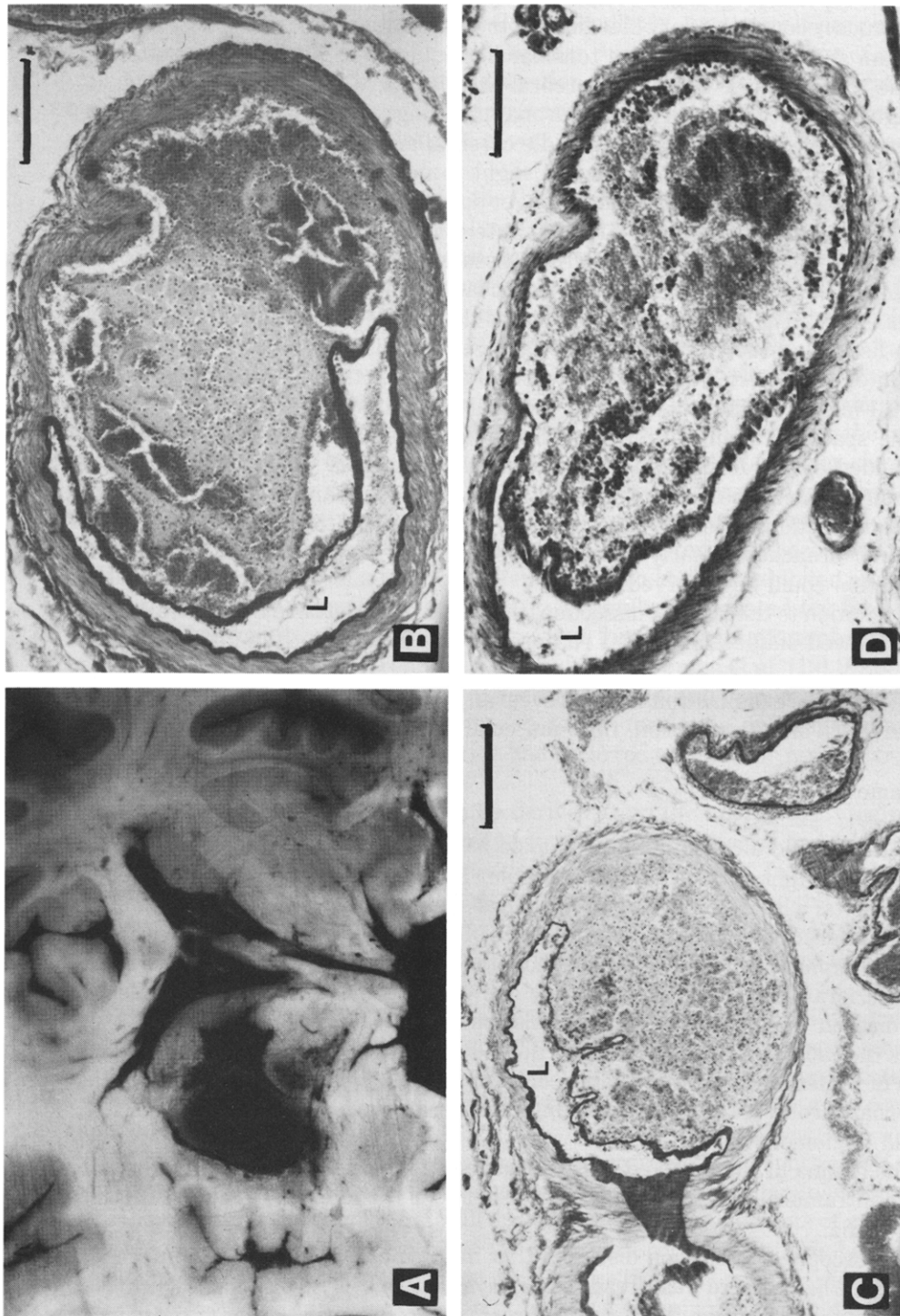


Fig. 1. (A) Acute ischemic cerebral infarction with recent hemorrhage in the basal ganglia. (B—D) Laminar dissection of branches of the left middle cerebral artery (B,C) and the anterior cerebral artery (A). The internal elastic lamina is detached from the media. The dissected space is distended by intramural clots (B) and recent hemostasis (C,D), the vascular lumen (L) is compressed. (B,C) Elastic van Gieson; bar represents 200 μ m. D PAS stain; bar represents 320 μ m.)

internal capsule) (Fig. 1A). The midbrain appeared symmetrically compressed with numerous recent secondary bleedings in the tegmentum and in the pons. The cerebellum showed signs of acute tonsillar herniation with hemorrhagic softening of the tonsillar tips. The basal cerebral arteries were slightly hypoplastic.

Light microscopically, recent neuronal palloring was found in the supply territories of the left anterior and middle cerebral artery, mostly still without any glial reaction. Only in some places a slight to moderate gliosis had occurred indicating a longer duration of the damage in these areas.

Microscopic cross sections of the left anterior and middle cerebral artery in the interhemispheric cleft and the Sylvian fissure displayed a striking laminar dissection of their walls, the intima and elastic lamina being detached from the media (Figs. 1B,C). The three major branches of the middle cerebral artery in the Sylvian fissure all showed this kind of damage. The dissection extended from the origin of the anterior and middle cerebral artery to the small peripheral branches. Finally, the trunk of the left internal carotid artery was also involved.

The space between the internal elastic lamina and the media was markedly distended by the formation of an intramural hematoma which compressed the vascular lumen. The intramural bleeding was predominantly acute without any cellular reactions from the vessel wall. Only in some of the dissected peripheral vascular branches an early cellular response to the damage and intramural clot formation could be observed.

In addition to the laminar dissection, the origin of the left middle cerebral artery also showed single transversal ruptures of the intima and the internal elastic lamina.

Microscopic examination did not reveal any signs of a pre-existing vascular disorder, such as inflammation, fibro-muscular dysplasia, fibro-elastosis or others.

Comments

A 19-year-old alcohol-intoxicated male had sustained kicks to his head in the course of a fight with other young people. Thereafter, he had to be admitted to the hospital where he had exhibited clinical symptoms of a left-sided ischemic cerebral infarction in the supply territories of the internal carotid artery. After a short interval of improvement throughout the first 5 days after admission, he rapidly deteriorated again on the sixth day due to a rise of the intracranial pressure. He died on the eighth day after admission.

As the cause of the cerebral infarction a surprisingly extensive laminar dissection of his left anterior and middle cerebral artery, beginning in the trunk of the internal carotid artery and extending peripherally into small arterial branches, could be demonstrated.

The lumina of the involved vessels had been compressed due to the formation of an intramural hematoma in the space between the internal elastic lamina and the media.

Microscopic examination disclosed that in some areas the ischemic infarction of the left hemisphere was already some days old as indicated by gliosis reaction to the damage. This was in agreement with the first onset of the clinical

symptoms (drowsiness, right-sided hemiparesis) a few hours after the fight when he had received the kicks to his head. Correspondingly, some small peripheral arterial branches already showed a slight cellular response to the damage of their wall, while in the majority the vascular dissection was still non-reactive, indicating a short duration of the damage. This corresponded with the fact that the major part of the left-sided cerebral infarction was also still non-reactive and at most 2 days old.

It is noteworthy that compared with the thorough arterial dissection, the deceased had only exhibited minor superficial signs of blunt impact to his body and head. Recent bleeding into the left temporal muscle was the only alteration found at autopsy, which indicated the mechanical impact to his head. Nevertheless, the immediate timely relation between the kicks to his head and the onset of clinical symptoms, together with the absence of pre-existing vascular disease do not leave any doubts that the extensive arterial dissection was of traumatic origin. The absence of superficial contusion marks could later be explained by the fact that the aggressor had worn jogging shoes with a soft sole. However, in general, the lack of external evidence of head injury despite severe intracranial vascular change is not so unusual. Dratz and Woodhall [12] described an observation very similar to the present one. In a young lady aged 21, a dissecting aneurysm of the left internal carotid, and the middle and anterior cerebral arteries had occurred due to blunt head trauma from a traffic accident. She also had not exhibited any external signs of head injury. The vascular dissection had separated the internal elastic lamina from the media. The same was true for the observations of de Veer et al. [54], Linell et al. [28], Brenner and Wasl [5] and Duman et al. [13]. In some of these observations signs of external head injury had also been missing. Like in our observation, vascular occlusion due to the formation of an intramural hematoma followed by cerebral infarction had occurred at intervals ranging from 1 h to several days.

In the present observation it could be derived from the microscopic findings that obviously at first only minor damage to the cerebral arteries had occurred as a cause of the older parts of the left-sided ischemic infarction, from which the patient first seemed to recover until the sixth day after admission to the hospital. At that time the major arterial dissection must have occurred as a second, spontaneous event causing the recent infarction which resulted in death due to severe brain swelling.

In the reviewed literature we scarcely found reports of traumatically induced cerebral arterial dissection of an extent similar to that in the present observation. Dissections of comparable extent have mostly been observed in pre-existing vascular disease, such as fibro-muscular dysplasia, where they may even occur spontaneously [3,22,38,39].

Traumatic laminar arterial dissection may be interpreted as a "splitting-off effect" due to laminar shear stress. This means the occurrence of a laminar disconnection of the layers of the arterial wall due to their different moments of inertia. As a result, on sudden acceleration of the head with rotation and side-bending the laminae will become shifted against each other. This may result in dissection of the mural layers, e.g. disconnection of the elastic lamina from the media or

the media from the adventitia etc. In addition, transversal rupture of mural layers may occur due to stretching.

Nedwich [36] and Ritchie [42] emphasized that on mechanical impact to the head of alcohol-intoxicated individuals the ethanol-dependent dilation of the vasa vasorum may favor the formation of an intramural hematoma in the dissected vascular space. In addition, the limitation of defense reactions and reflexes in ethanol-intoxication may contribute significantly to the occurrence of a laminar arterial dissection.

By the analysis of 31 observations of traumatic cerebral arterial rupture, Krauland [24] could demonstrate that in alcohol-intoxicated individuals vascular lesions resulting in death may already occur due to minor non-penetrating head trauma, even without external signs of injury. Such observations may suggest that in the present observation the alcohol-intoxication of the victim may have played a significant role in the origin of the thorough intracranial arterial dissection.

The observation reported may serve as a further example in that, a lack of external evidence of head injury after closed trauma does not exclude the occurrence of severe damage to intracranial arteries which may result in either subarachnoid hemorrhage [10,24,25] or vascular occlusion with stroke [5–7, 13,28,29,54,55].

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