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Intracranial Dissecting Aneurysm

Report of a Case

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Summary. The case of a 47-year-old man who died one month after a history of paroxysmal occipital headaches, vertigo, vomiting, weakness, and sweating is presented. The death was due to a pontine softening caused by a subintimal dissecting aneurysm of the two vertebral, the basilar and the right posterior inferior cerebellar arteries. No etiological factor of the illness could be found. The clinical signs resembled those of a flap-valve tumor of the IIIrd ventricle.

Key words: Dissecting aneurysm – Clinical signs – Etiology.

Zusammenfassung. Der Fall eines 47jährigen Patienten wird beschrieben. Der Kranke hatte occipitale Kopfschmerzen, Schwindelgefühl, Erbrechen, Schwitzen und ist nach einem Krankheitsverlauf von einem Monat verstorben. Der Tod war durch eine pontine Malacie verursacht. Der pontine Erweichungsherd entwickelte sich als Folge eines dissezierenden subintimalen Aneurysma der Aa. vertebrales, der A. basilaris und der rechten A. cerebelli inf. post. Keine ätiologischen Faktoren konnten gefunden werden. Die klinischen Symptome waren denen einer Ventil-Geschwulst des III. Ventrikels ähnlich.

Schlüsselwörter: Dissezierendes Aneurysma – Klinische Zeichen – Ätiologie.

Introduction

The occurrence of dissecting aneurysms of the intracranial cerebral vessels is very rare. There are no more than 15 reported cases of dissecting aneurysms in the vertebro-basilar system [1—10, 12, 13, 15—17].

Case Report

A 47-year-old man was admitted to our Department on 4th of August 1977 suffering a sudden loss of consciousness. He had complained of headaches for years. Three times between the 10th

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of July and 3rd of August 1977 he had severe occipital headache, vertigo, weakness, vomiting, and sweating lasting for a short time after bending forward. There was no previous history of hypertension, head injury or lues. On the day of admission he became unconscious with bilateral peripheral facial and abducens paresis, and hypotonic tetraplegia. After dehydration the neurological signs were peripheral facial weakness on the right side and left side hemiparesis. On the same day, ventriculocisternostomia was performed because of a sudden tetraparesis with pyramidal signs and brain stem fits. It was thought to be a tumor of the IIIrd ventricle, hippocampal, and tonsillar herniation. The C.S.F. was clear and normal. W.R. was negative. Six days after the operation the patient died.

Neuropathological Findings

The autopsy revealed a bronchopneumonia. The brain weighed 1,430 g. There was swelling in the right half of the cerebral pons. In the basis of the pons, mainly on the right side, there was a recent, partly hemorrhagic softening from the plane of the superior colliculi to the medulla (Fig. 1). Microscopically we found a dissecting aneurysm in the wall of the macroscopically normal, non-atherosclerotic vertebral arteries, in the basilar artery and in the right posterior inferior cerebellar artery. The most severe dissection was in the proximal part of the left vertebral artery (Fig. 2), where the original lumen was connected to the remaining wall of the vessel only by a small stem. The remaining medial coat was very thin or partly absent. In the newly formed lumen there was a recent thrombus. A fibrotic thickening was found in the intima of the collapsed original lumen. In the distal part of the left vertebral artery the dissection involved only 50% of the circumference. The original lumen was a thin cleft with an intensive fibrotic intimal thickening, the new lumen was filled with fresh blood. In the right vertebral artery the dissection involved 70% of the circumference. The medial

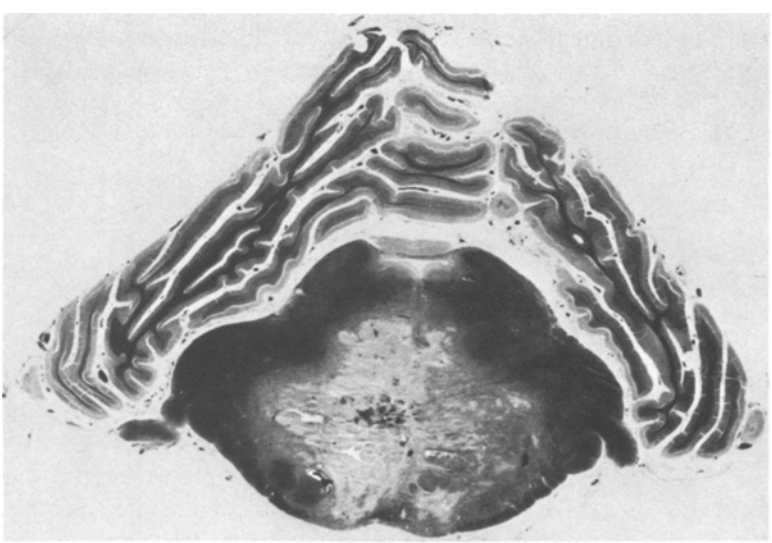

Fig. 1. Softening in the basis of the pons. Heidenhain myelin stain

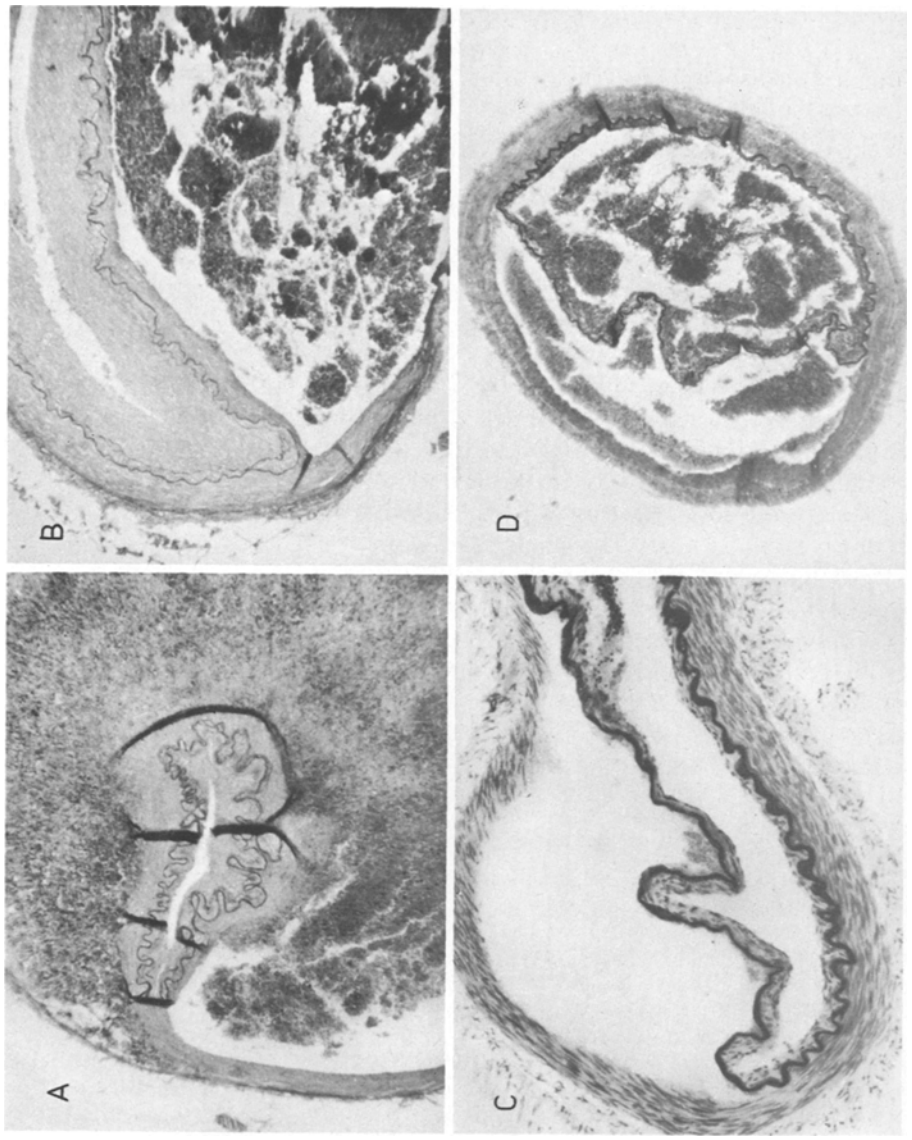

Fig. 2. **A**: Dissection in the proximal part of the left vertebral artery. Elastic-van Gieson, original magnification ×32. **B**: The right vertebral artery. Elastic-van Gieson, original magnification ×63. **C**: Dissection in the wall of the basilar artery. Luxol fast blue, original magnification ×63. **D**: The right inferior posterior cerebellar artery. Elastic-van Gieson, original magnification ×32

coat of the original lumen was normal but on the other side there were foci of intramural hematomas (Fig. 2b). The basilar artery was affected in its total length. There was a slight intimal fibrosis in the original lumen. The media was thin at the newly formed lumen (Fig. 2c). The dissection extended into the right inferior posterior cerebellar artery too (Fig. 2d).

The other cerebral arteries, including the posterior cerebral arteries, were normal. In all dissected arteries the internal elastic lamina was fragmented, thickened, and the dissection was located between the internal elastic lamina and the media.

Discussion

In this case a dissecting aneurysm affecting the two vertebral and the basilar arteries was found in a young man one month after the beginning of the clinical history of paroxysmal headaches and six days after the beginning of the neurological signs. From the clinical point of view it is interesting and instructive to find that the dissecting aneurysm of the vertebro-basilar arteries might mimic the signs of a ventile tumor.

As the dissection was most severe in the left vertebral artery, we can assume that it began in the wall of this vessel. Because neither the general pathologist nor we thought of this kind of illness, we could not investigate the extracranial parts of these arteries.

The long history (one month) of the disease suggests that circulation was maintained in the dissected arteries for a long time and the intramural bleeding may have spread permanently or in attacks from the vertebral arteries.

The etiology of the disease is unknown. In the literature there are various etiological factors mentioned, e.g., syphilis (Turnbull, 1915; Szabó, 1939), head injury (Brihaye, 1971), mucoid medial degeneration (Hyland, 1933; Watson, 1956), legal and accidental electrocution (Hassin, 1933, 1937), homocystinuria (Campiche, 1969), atherosclerosis (Crosato, 1961; Scholefield, 1924), as well as unknown causes (DeBusscher, 1952; Wolman, 1959). In our subject we did not find any evidence of preexisting or acute arterial disease.

According to Sinclair (1953) migraine may result in the dissection of the cerebral vessels, but in our case it seems more reasonable that it was the dissection itself that caused the occipital headaches.

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