

Are your **MRI contrast agents** cost-effective?

Learn more about generic **Gadolinium-Based Contrast Agents**.



FRESENIUS
KABI

caring for life

AJNR

Rupture of an anterior thalamoperforating artery aneurysm: cause of basal ganglia hemorrhage.

A J Kumar, S J Zinreich and T J Preziosi

AJNR Am J Neuroradiol 1982, 3 (5) 581-582

<http://www.ajnr.org/content/3/5/581.citation>

This information is current as
of April 16, 2024.

Rupture of an Anterior Thalamoperforating Artery Aneurysm: Cause of Basal Ganglia Hemorrhage

Ashok J. Kumar,¹ S. James Zinreich,¹ and Thomas J. Preziosi²

The bulk of spontaneous basal ganglia hemorrhages result from rupture of the small perforating intracerebral arteries and a clinical background of hypertension. Charcot and Bouchard [1] noted that most microaneurysms occurred in hypertensive brains due to miliary microaneurysms involving branches of lenticulostriate and thalamoperforating arteries. Such microaneurysms were subsequently demonstrated on direct magnification carotid angiography [2].

Rupture of an arteriovenous malformation, bleeding into a neoplasm, and rupture of circle of Willis aneurysms are also known causes for hemorrhage within the basal ganglia. On review of the literature, no previous angiographic demonstration of a macroaneurysm arising from a thalamoperforating artery has been uncovered and serves as the basis of our report.

Case Report

A 48-year-old woman was admitted to The Johns Hopkins Hospital after sudden onset of right temporal headaches, followed by nausea, vomiting, photophobia, and lethargy. There was no history of trauma and no evidence of hypertension on previous medical examinations. On admission her blood pressure was 105/70 mm Hg and her pulse rate 80 beats/min and regular. Positive neurologic findings included mild weakness of the left arm and leg with decreased tone. The plantar response was flexor bilaterally. Cranial nerve examination evidenced only a central left seventh nerve palsy.

Computed tomography (CT) immediately after admission revealed a 3.3 × 1.3 cm hematoma involving the right putamen, internal capsule, and thalamus anteriorly (fig. 1A). Vertebrobasilar and right internal carotid angiography also performed on the day of admission showed a round aneurysm arising from the terminal part of an anterior thalamoperforating artery (figs. 1B–1D). Subsequent studies including echocardiography, blood cultures, and antinuclear antibody were negative.

Repeat cerebral angiography 18 days after admission was performed to observe the status of the aneurysm and any vascular changes that may have suggested an etiologic factor. The angiogram again showed normal vascularity and the aneurysm was no longer seen. In the interim, the patient had been administered Decadron (dexamethasone) with gradual improvement in the neu-

rologic deficit. The patient has been followed for a period of 5 years since this episode without the development of new central nervous system symptoms or findings related to other organ systems.

Discussion

Charcot and Bouchard [1] in 1868 proposed that intracerebral hemorrhage resulted from the rupture of small arterial aneurysms. Their work was based on 60 cases of cerebral hemorrhage in mostly elderly patients. In Russel's x-ray microangiography of 54 autopsy cases [3], 16 had been hypertensive (none were younger than age 50 and only two were younger than age 60) and 38 normotensive. Fifteen of the hypertensive cases and 10 of the normotensive cases had miliary aneurysms of small diameter (100–300 μm) arteries. The diameter of the concomitant aneurysms ranged from 300–900 μm . In a postmortem study of 200 brains by Cole and Yates [4], 100 (half) patients had been hypertensive and 46 of those had had microaneurysms compared with 7% of normotensive subjects. The occurrence of the microaneurysms in hypertensive patients was also found to be age-related since only two of the hypertensive patients were younger than 50 years of age and the involved normotensive patients were older than 65 years. Leeds and Goldberg [2] were the first to demonstrate microaneurysms in hypertensive patients by direct magnification angiography.

Our report describes a nonhypertensive patient in whom the hemorrhage resulted from rupture of a *macroaneurysm* involving the distal part of an anterior thalamoperforating branch. The precise etiology of this aneurysm remains undisclosed both because of its deep location, which precluded surgery and histopathologic assessment, and because the aneurysm was not visualized on follow-up examination.

ACKNOWLEDGMENT

We thank Ellen Ellis for assistance in manuscript preparation.

Received October 26, 1981; accepted after revision February 23, 1982.

¹ Department of Radiology and Radiological Science, Johns Hopkins Medical Institutions, 600 N. Wolfe St., Baltimore, MD 21205. Address reprint requests to A. J. Kumar.

² Department of Neurology, Johns Hopkins Medical Institutions, Baltimore, MD 21205.

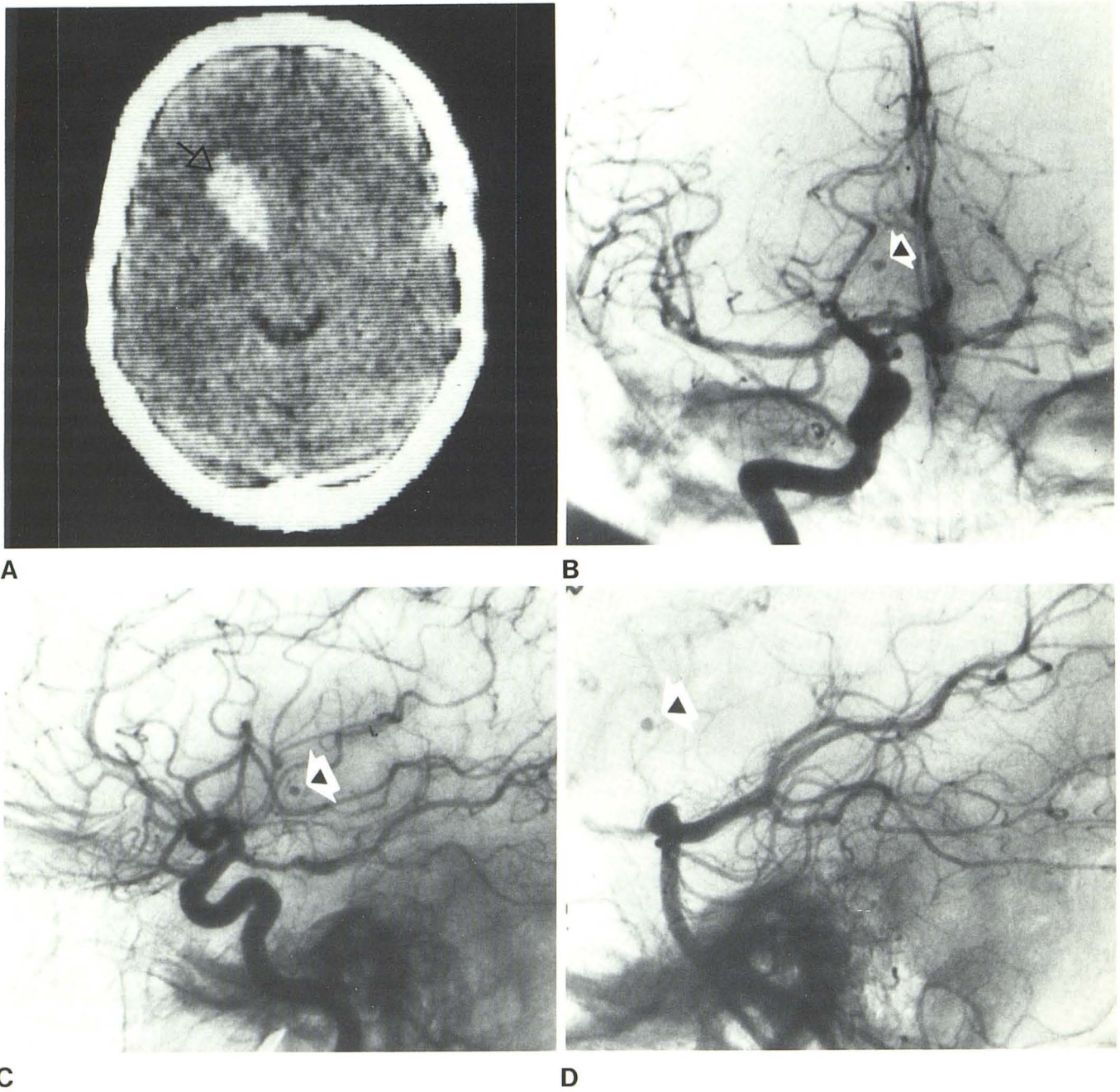


Fig. 1.—A, Noncontrast CT scan. Basal ganglia hemorrhage (arrow). B–D, Cerebral angiography. B, Selective internal carotid angiography. Frontal projection. Small aneurysm just medial to right posterior cerebral artery. C,

Lateral projection. Rounded collection represents aneurysm. D, Selective vertebral angiography, lateral projection. Aneurysm derived from anterior thalamoperforating branch.

REFERENCES

1. Charcot JM, Bouchard C. Nouvelles recherches sur la pathogenie de l'hémorragie cerebrales. *Arch Physiol Norm Pathol* **1868**;1:110–127, 643–665
2. Leeds NE, Goldberg HI. Lenticulostriate artery abnormalities.

Radiology **1970**;97:377–383

3. Russell RWR. Observations on intracerebral aneurysms. *Brain* **1963**;86:425–442
4. Cole FM, Yates PO. Intracerebral microaneurysms and small cerebrovascular lesions. *Brain* **1967**;90:759–768