

INTRAVENTRICULAR HEMORRHAGE DUE TO LEFT FRONTAL LOBE ARTERIOVENOUS MALFORMATION

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Key-word: Arteriovenous malformations, cerebral.

Background: A 23-year-old woman was admitted to a general hospital with severe headache of three days duration. The patient was 16 weeks pregnant. Previous medical history was unremarkable. Within hours after admission, the patient lost consciousness and was transferred to our hospital. Upon arrival, the patient was comatose with intact pupillary light reflexes. Laboratory findings were within normal range.

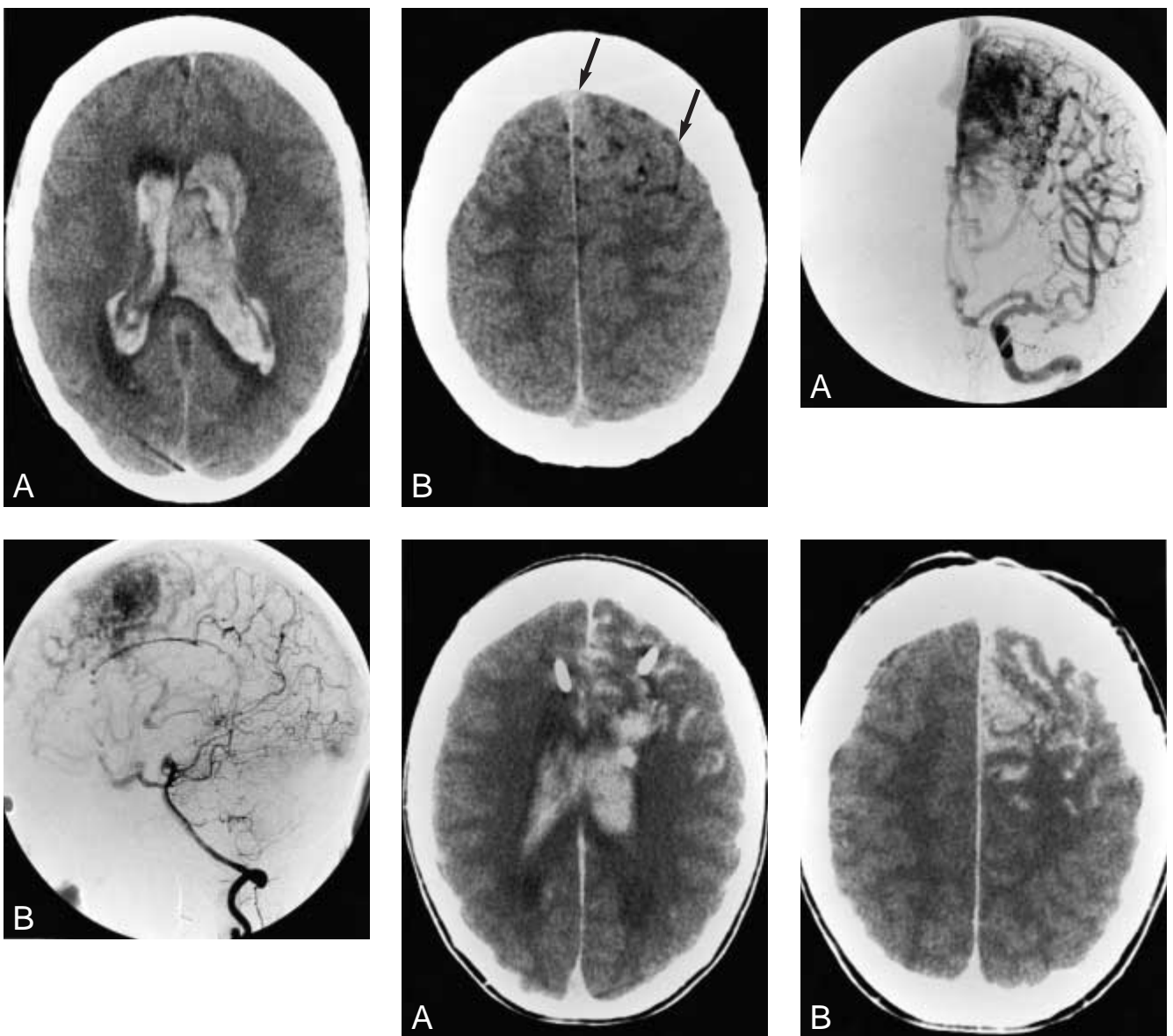


Fig.

1A	1B	2A
2B	3A	3B

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Work-up

A non-contrast CT scan revealed an extensive intraventricular hemorrhage. After immediate placement of ventricular drains, cerebral angiography followed by (contrast-enhanced) CT scan was performed.

Radiological diagnosis

Plain CT scan of the brain (fig. 1) includes an axial slice through the lateral ventricles (A) and an axial slice through the centrum semiovale (B). It shows massive intraventricular hemorrhage with hydrocephalus (A). There is also an ill-defined area of branching hypodensities in the left frontal region (B, arrows).

Cerebral angiography consisted of a selective angiography of the left internal carotid artery, AP view (A) and a selective angiography left vertebral artery, lateral view (B). It shows an extensive arteriovenous malformation (AVM) in the left frontal lobe with cortical-subcortical and subependymal extension.

The feeding arteries originate from the anterior cerebral artery, middle cerebral artery and posterior choroidal arteries. Venous drainage is basically through superficial veins as well as through the deep venous system.

On CT scan of the brain after angiography (fig. 3, A: axial slice through the lateral ventricles and B: axial slice through the centrum semiovale), a large inhomogeneous contrast enhancing area confirms the AVM. Multiple ventricular draining catheters are seen in place (A).

The diagnosis of *left frontal lobe arteriovenous malformation (AVM) with intraventricular hemorrhage* was made.

Discussion

AVMs are the most commonly encountered type of vascular malformation. They are congenital lesions with abnormal arterial supply and venous drainage that bypass the capillary system. The pial AVMs typically consist of enlarged supplying arteries, a nidus, and dilated draining veins.

Prevalence of AVMs in the population is described in the literature varying from 0.1% to 0.8%. AVMs typically manifest themselves in the third to fourth decade. The most common initial symptom is related to intracranial hemorrhage, most often intraparenchymal. Intraventricular hemorrhage is seen less often, however literature states that an intraventricular hemorrhage should always suggest an underlying AVM, provided that trauma and hypertensive bleeding have been ruled out.

Larger AVMs present more often with seizures. Headache is another frequently described symptom of AVMs.

For unruptured AVMs the incidence of hemorrhage is about 2% to 4% per year. For a ruptured AVM the incidence increases to 6% in the year after and gradually decreases in the following year to the 2% to 4% of the non-ruptured AVMs.

A ruptured AVM has a mortality rate of 10% to 15% and a morbidity rate for permanent neurological deficit of 20% to 30%.

In our case we have to consider that the patient was pregnant. Literature states that pregnancy does not increase the risk of first hemorrhage of AVMs. However after a ruptured AVM there is a higher risk of repeated hemorrhage in pregnancy compared to non-pregnancy.

Angiography should be performed to document the size and location, the number of feeding arteries and draining veins, and the presence of associated arterial, nidal or venous aneurysms. This information is crucial for treatment planning.

Bibliography

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