

An Anatomical-Computerized Tomography (CT Scan) Study on the Arteriovenous Malformations (AVMs) in the brain of Iraqi Patients

Nameer T. George * MBChB. MSc. PhD

Summary

Background Arteriovenous malformations (AVMs) of the brain are anomalies affecting different age groups of the population, and predisposing patients to significant neurological disability from stroke, epilepsy, or other clinical manifestations. Noninvasive modalities are revealing these lesions more frequently, and with more accuracy. Previous studies on Iraqi subjects with intracranial AVMs are scarce.

Objectives The aim of the study is to correlate the CT findings of intracranial AVMs with the clinical presentations, anatomic locations, the size, and the predictable origin of the arteries feeding these lesions and their venous drainage.

Patients and Methods The charts and CT scans of fifty-four Iraqi patients with an AVM, 31 males and 23 females (male to female ratio 1.3: 1), ranging in age from 6-74 years (mean 37.7) who were seen at the Neurosurgical Hospital-Baghdad from October 1998 to August 2002 were reviewed.

Results Supratentorial AVMs were present in 53 patients; one patient had a left cerebellar AVM. The lesion was solitary, and directly localized in a single lobe, with more in the right lobes (mainly the parietal and temporal) in the non-haemorrhagic lesions, and in the left lobes of the AVMs presented with haemorrhage. The diameter of the lesion varied from less than 2.5 cm to ≥ 6.5 cm.

Conclusion AVM may present symptomatically at any age. The arterial and venous components of the AVM could be explained by the site of the lesion. The size of the AVM could be evaluated as a potential factor predicting future AVM haemorrhage risk. Long-term follow-up evaluation is necessary for assessing the natural history and prognosis for such lesions.

Key words: arteriovenous malformation. Computerized tomography (CT). Brain. Anatomical localization

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Introduction

to August 2002 were reviewed. They had one or more CT scans of the head. Intravenous contrast media was used routinely. In our series, of the thirty patients presenting with hemorrhage, in fifteen patients the plain and enhanced scans were performed within one week from hemorrhage; in three patients: from one to four weeks after hemorrhage, and in twelve patients: four weeks or longer after hemorrhage.

CT images were reviewed by neuroradiologist for radiological diagnosis and by neurosurgeons for further assessment. For the lesion size, data on maximum diameter of the AVMs were available for 36 patients. In 18 malformations, the precise size was unknown as the CT scan of the head was not available for review.

Results

Thirty patients (55%) presented to hospital with a recent or remote intracranial hemorrhage, nineteen (36%) with a seizure disorder, and five (9%) with a migraine-like headache or other neurological symptoms (Table 1).

As there are distinct histopathological features that allow distinction between four types of vascular malformations (arteriovenous malformations, cavernous malformations, dural venous malformations, and telangiectasias) (4), there are specific features on CT that correspond to their distinction as well. In AVM, the most common CT

An AVM is defined as a congenital non-neoplastic vascular abnormality comprised essentially of a coiled mass of arteries and veins partially separated by thin strips of gliotic nervous tissue, lying in a bed formed by displacement rather than by invasion of normal brain tissue (1). It is a fistulous malformation or a shunt that permits arterial blood to enter the venous system without passing through an arteriole-capillary bed. To date, the cause of this local angioblastic mistake is unknown.

Accuracy, as high as 100%, has been reported for the CT demonstration of intracerebral AVMs, with or without contrast media are performed (2).

The aim of this work is to define the clinical presentation, anatomical localization, and the size of AVMs in the brains of fifty-four Iraqi patients using CT scan for their evaluation. By systematically reviewing the literature, we have found that there is very little information about the frequency and clinical course of AVMs of the brain in Iraqis.

Patients and Methods

The charts and CT scans of fifty-four Iraqi patients with an AVM, 31 males and 23 females (male to female ratio 1.3: 1), ranging in age from 6-74 years (mean 37.7) who were seen at the Neurosurgical Hospital-Baghdad from October 1998

* Department of Anatomy, College of Medicine-University of Baghdad

enhancing high density lesion was visible in 8 patients.

Table3. CT Findings in 19 patients of AVM presenting with seizure disorders

<i>CT Findings</i>	<i>No. of Cases</i>
<i>high density lesion without mass effect</i>	9
<i>low density lesion</i>	2
<i>dilatation of ipsilateral lateral ventricle/ventricular abnormality</i>	7
<i>infarction</i>	1
Total	19

Of the 5 patients in the heterogeneous group; four had a normal plain CT scan. With infusion study, however, all 4 patients showed vessel enhancement or demonstrated a lesion not previously seen on a plain CT scan. One patient, who exhibited ataxia, had a large AVM of the left cerebellar hemisphere (Table 4)

Table4. CT Findings in 5 patients of AVM presenting with a non-haemorrhagic, non-seizure presentation

<i>CT Findings</i>	<i>No. of Cases</i>
<i>high density lesion without mass effect</i>	2
<i>low density lesion</i>	1
<i>dilatation of ipsilateral lateral ventricle/ventricular abnormality</i>	2
<i>infarction</i>	1
Total	5

Location of the AVM (Table 5)

Supratentorial AVMs were found in 53 patients; one patient had a left cerebellar AVM. Although lesions were detected in the frontal, temporal, and occipital lobes, the sites most commonly involved were the right parietal lobe (in 13 patients), and the left parietal lobe (in 9 patients).

There were slightly more lesions on the right than on the left side (33 versus 21), although the opposite was seen in haemorrhagic lesions (17 versus 13). AVM at the area of the right basal ganglia was noted in the CT scan of two patients.

Table 5. Location of AVMs based on CT scan

Location	Right side	Left side
<i>Frontal lobe</i>	7	4
<i>Parietal lobe</i>	13	9
<i>Temporal lobe</i>	10	7
<i>Occipital lobe</i>	1	0
<i>basal ganglia</i>	2	0
<i>cerebellum</i>	0	1
Total	33	21

findings included (2,3): a) a high density lesion, b) a low density lesion, c) ventricular abnormalities, d) a mass effect secondary to the AVM, e) a shift of midline cranial structures, and f) the presence of calcification within the malformation.

Table 1. Clinical presentation of 54 cases of brain AVM

presentation	No. of cases	Percent
haemorrhage		
<i>coma</i>	7	
<i>drowsiness</i>	3	
<i>hemiparesis</i>	11	
<i>incoordination/altered mental state</i>	3	
<i>somatosensory deficit</i>	6	55
Total	30	
Seizure		
<i>generalized</i>	8	
<i>focal</i>	11	
Total	19	36
heterogeneous group		
<i>headache</i>	2	
<i>focal symptoms</i>	2	
<i>ataxia</i>	1	9
Total	5	

In patients presenting with hemorrhage (Table 2), a haematoma was noticed in 9 patients; intraventricular haemorrhage was seen in 2 patients, subarachnoid bleeding was seen in 2 patients. Two patients had a small parietal AVM associated with hemosiderosis of the surrounding brain tissue. The plain CT scan showed a high density that did not enhance with infusion in 13 patients.

Table 2. CT Findings in 30 patients of AVM presenting with intracranial haemorrhage

<i>CT Findings</i>	<i>No. of Cases</i>
high density lesion without mass effect	
low density lesion	
haematoma	9
subarachnoid bleed	2
high density lesion with compression of ipsilateral lateral ventricle	
calcification	2
ventricular abnormality	2
oedema around a lesion	
Total	30

The findings on plain CT of 19 patients with AVM presenting with a seizure disorder is shown in Table 3. There was dilatation of the ipsilateral lateral ventricle, and on the infused study an

or the temporal lobe (in 17 patients), or the occipital lobe (1 patient), or in the basal ganglia (2 patients). The AVMs in the frontal lobe (11 patients), and the parietal lobes, could be supplied exclusively or in combination by the anterior or the middle cerebral artery. This requires the confirmation by angiographic studies.

The parent arterial trunk feeding these malformations is usually a normally placed but enlarged channel (10). In the embryo 20 to 40 mm in length (11), this is probably the determinant stage of these lesions; the terminal branches of the main arteries are continuous on the medial and pallial walls of the embryonic hemisphere. Thus, more than one main cerebral artery could participate in many of these malformations (12).

Venous drainage can also be predicted from the site of the AVM in most instances. This depends on the most adjacent venous sinus to the AVM; the instance of a lesion bypassing the most adjacent venous sinus to drain into a more distant one is scarce (13). Thus, the superior sagittal sinus may participate in draining AVMs in the frontal, parietal, occipital, and temporal; the great cerebral vein (of Galen), the parasellar plexus, and/or the petrosal sinus can participate in draining AVMs located in the basal ganglia. The arterial and venous components of AVM signify the site and extent of the chance interposition of the angioblastic error resulting in an AVM.

The size of the AVM has been evaluated as a potential factor predicting future AVM haemorrhage. In arteriographic studies, without long-term follow-up, some have noted the size to be an important factor in presentation with haemorrhage (14,15), while others have not noted AVM size to be a predictor of presentation with haemorrhage (16, 17).

In our series, the diameter of the lesion varied from less than 2.5 cm to ≥ 6.5 cm. The size of the AVM may be helpful in predicting the apparent growth of the AVMs in follow-up studies. It has been reported that AVM can change over time; while some do increase in size or may remain unchanged, others get smaller or thrombose completely (18, 19). Hamby (20) pointed out that the malformations are most certainly found early in the development of the cerebral mantle, and remain separable from normal brain tissue so that lesion growth would be expected to occur between definitive boundaries without involvement of originally normal vasculature. Thus, AVMs may have a predetermined ultimate size. This would account for the occasional finding of a very small lesion in an older person. On the other hand, the size of AVM was found to be of no value in predicting rupture (21).

It should be pointed out that AVM are detected with more sensitivity with MRI than CT (2). However, CT is often the first imaging study obtained, particularly in acute clinical presentations. MRI is

***AVMs were either on the right or the left side. No lesions were seen on both sides**

Size of AVM (Table 6);

The diameter of the lesion varied from less than 2.5 cm to ≥ 6.5 cm. Only 3 of 36 lesions measuring 3.4 cm or less manifested as haemorrhage compared with 13 of 20 lesions measuring 3.5 to 5.4 cm and 8 of 11 that were 5.5 cm or larger.

Table 6. The Size (diameter) of AVMs based on CT scan

Location	Haemorrhagic AVMs	Non-Haemorrhagic AVMs
<2.5cm	1	1
2.5-3.4cm	2	1
3.5-4.4 cm4	6	4
4.5-5.4cm	7	3
5.5-6.4	6	2
$\geq 6.5cm$	2	1
Total	24	12

Discussion

This study defined the clinical presentation, the location, and size intracranial of AVMs in a random group of 54 Iraqi patients; these were diagnosed by CT scan and in 26 of them before a clinically evident intracranial haemorrhage occurred (two patients in the non-haemorrhagic group showed evidence of infarction on CT). Our study confirmed previous reports that AVM may present symptomatically at any age (5, 6). However, the present study lacked consistent long-term follow-up evaluation which is necessary for assessing the natural history and prognosis, in particular the risk of rupture.

The lesion is usually, if not always, directly localized in a single lobe, with more on the right side and mainly in the parietal, temporal and frontal lobes. In this study, the site of the lesion was used to localize the AVM without regard to the extent of the feeding arteries or draining veins. Correlation of signs and symptoms by location of the lesion indicates that the site of the AVM is usually responsible for the resulting neurological deficit (7). However, some signs and symptoms may be due to the effect at a distance from the lesion, such as from vascular steal (8), or the mechanical effect of a large feeding or draining vessel (9).

The arterial component of the AVM could be explained by the site of the lesion; in 53 cases the AVMs were supratentorial; the arterial trunk feeding these malformations could be the middle cerebral artery and/or the posterior cerebral artery where the AVM were located in the parietal lobe (22 patients),

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superior to CT and angiography for demonstrating the size and precise anatomic location of the AVM nidus, and the relationship of its feeding and draining vessels to deep structures such as the basal ganglia, the ventricular system, and corpus callosum. The introduction of MRI with its high sensitivity and specificity makes it the imaging examination of choice in the evaluation of AVM in future studies.

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