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Spontaneous occlusion of cerebral arteriovenous malformation accompanied by distal flow-related aneurysm after partial embolisation – a case report

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Background:

Summary

We report the case of a 48-year-old woman with right occipito-temporal AVM associated with two flow-related aneurysms. One of the aneurysms was located on the junction of P1/P2 segments of the right posterior cerebral artery (PCA), the second one- on the right carotid internal artery (ICA). The patient suffered from migraine headache, epilepsy (resistant to treatment) and left hemiparesis. The decision to treat the AVM and the aneurysms with endovascular embolisation was made. The purpose of the treatment was to occlude the flow-related aneurysm which was located on the main access route to the AVM first, and then to carry out the embolisation of the latter. An attempt of aneurysm embolisation failed due to its large neck as well as high, turbulent flow inside the sac which prevented from anchoring any coil. An embolisation of an AVM via right ICA and posterior communicating artery was undertaken and resulted in 80% reduction of the AVM nidus volume. Control angiogram after 2 months showed complete occlusion of the AVM and lack of filling of the right P1/P2 junction aneurysm.

The right ICA aneurysm was occluded by embolisation with GDC coils.

Key words:

arteriovenous malformation • aneurysm • embolisation

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Background

The arteriovenous defects of the brain, commonly known as angiomas, belong to the developmental defects of the vascular system [1]. The formation starts in the fetal life and despite its often observed functional and volumetric enlargement in life, it is completely developed before the delivery [2].

Intracranial angiomas are characterized by abnormal arteriovenous connections of high blood flow, which take place of normal capillary vessels. Such junctions occur locally in the form of a ball of tangled vessels called the nidus of

angioma, to which the blood flows through supplying arteries (usually functionally enlarged) and is diverted through the veins which are enlarged as well [2].

The occurrence of intracranial angiomas is estimated for 0,02–0,05% of the population [3]. They can be situated in every part of the central nervous system. In 90% they are located supratentorially. In about 12 % of cases the arteriovenous angiomas are accompanied by the so-called flow-related aneurysms, as their formation is a result of a pathologically increased blood flow in the arteries supplying the lesion [4, 5, 6, 7]. The risk of intracranial hemorrhage in patients with cerebral angiomas is estimated for 1–2% per year, though if it is complicated with the presence of an



Figure 1. Left vertebral artery angiography (DSA) in lateral view before (A) and after (B) AVM embolisation. Fig. 1b showing decrease (app. 80%) of AVM nidus. Both angiographies show large aneurysm in the proximal part of the right posterior artery (PCA) (P1/P2).

aneurysm – the risk rises to 7% a year [3, 8]. The most common sources of bleeding are aneurysms located in the nidus of angioma [4, 8].

Case report

In march 2000, the patient, P.T., aged 48 was admitted to the Neurosurgery Clinic in Lublin for intensifying epileptic attacks and migraine headaches, which had occurred for around 20 years and were resistant to pharmacologic treatment. The neuroradiological examination revealed mild left hemiparesis with superficial sensation disorders and weakening of the muscle strength, positive Romberg's test and positive Babinsky's sign on the left side. The MR, angioMR, digital subtraction angiography (DSA) revealed intracranial angioma of the right cerebral hemisphere, located deeply on the junction of occipital and temporal lobes. The angioma was mainly supplied by temporal or occipital branches of the right posterior cerebral artery. Additional supply was provided by temporal branches of the middle cerebral artery. Blood outflow from angioma was through deep veins to Galen's vein and to straight sinus.

Angiographic examination also revealed two saccular aneurysms – the first one with diameter of 10mm, located on P1/P2 junction of the right posterior cerebral artery, the second – with diameter of 2.5 mm in the suprasphenoid part of the right interior carotid artery (fig. 2a, 3a). They were both flow-related.

The patient was referred to treatment of all the mentioned vascular changes by means of endovascular embolisation. In June 2000 an attempt for embolisation of the right posterior cerebral artery was made, as its location enhanced the risk for selective catheterization of the artery which was a natural access route to the nidus.

The embolisation was carried out in general anesthetic, with puncture of the right femoral artery. Heparin was injected – 5000 IU as a bolus, 1500 IU per hour in intravenous infusion. Guiding catheter 6F was inserted into the left verte-

bral artery and angiographic examination was performed. Afterwards, the catheter (Tracker Excel 14, Target, Freemont, California, USA) was inserted with the use of a microguide (Transend 14, Target, Freemont, California, USA) and its ending was placed inside the aneurysm. An attempt of inserting GDC-10 spiral with diameter of 12 mm and 30 cm of length (Tracker Excel 14, Target, Freemont, California, USA) was made. Because of the wide base of aneurysm and a very fast turbulent blood flow in its sac, the attempt was unsuccessful. The following attempt of inserting the microguide (Magic 1,8 F, Balt, Montmorency, France) into the distal segment of the right posterior cerebral artery in order to carry out the embolisation of the angioma on the side of the vertebral artery was impossible due to the presence of an aneurysm and tortuosity of the P1 segment. The catheter was inserted through the right interior carotid artery, then through anterior communicating artery to its posterior temporal branch. Its tip was placed selectively inside the angioma nidus. The control angiography examination was performed in this position, followed by an administration of 0.7 ml of a 25% mix of Histoacryl and Lipiodol. The control angiography performed through a guiding catheter revealed an occlusion of 80% of the volume of angioma (fig. 1a, b). The operation did not cause any complications.

The next stage of the treatment took place 2 months after the embolisation of the angioma. Clinical state of the patient improved. Angiographic control exam of cerebral vessels showed no signs of the nidus filling up along with complete spontaneous occlusion of the flow-related aneurysm and P1 segment of the left posterior cerebral artery (fig. 2a-f).

Small aneurysm of the right interior carotid artery was closed with one GDC-10 Soft-type spiral (2x8 mm) by means of embolisation. The control angiographic examination revealed a complete exclusion of aneurysmal sac from circulation (fig. 3a-d).

The patient was discharged from hospital after 3 days, in an unchanged clinical state, with persistent minor left side paresis and prescription of control angiography after 6 months.

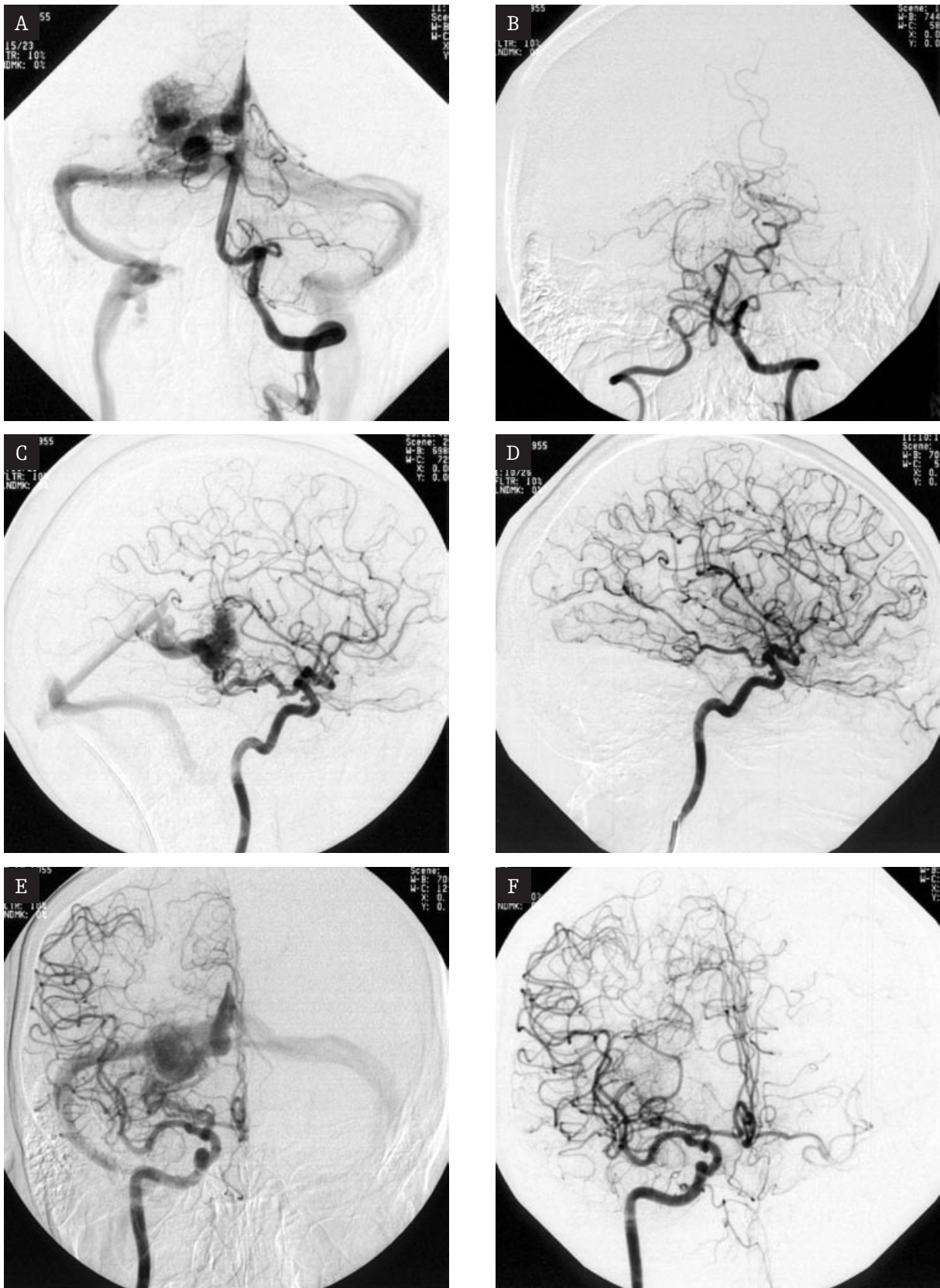


Figure 2. Right posterior artery (PCA) and right ICA angiographies with the AVM and aneurysms (before embolisation) – left vertebral artery injection in standard AP view (A) and right ICA AP view (B) and lateral view (C). Note, DSA left vertebral artery AP view (D) and ICA AP view (E) and lateral view (F) 2 months after partial embolisation of the AVM. Complete Occlusion of the AVM nidus and an aneurysm located in the proximal part of the right posterior artery. Spontaneous self-occlusion of the P1 segment of the right PCA fills through the right posterior communicating artery.

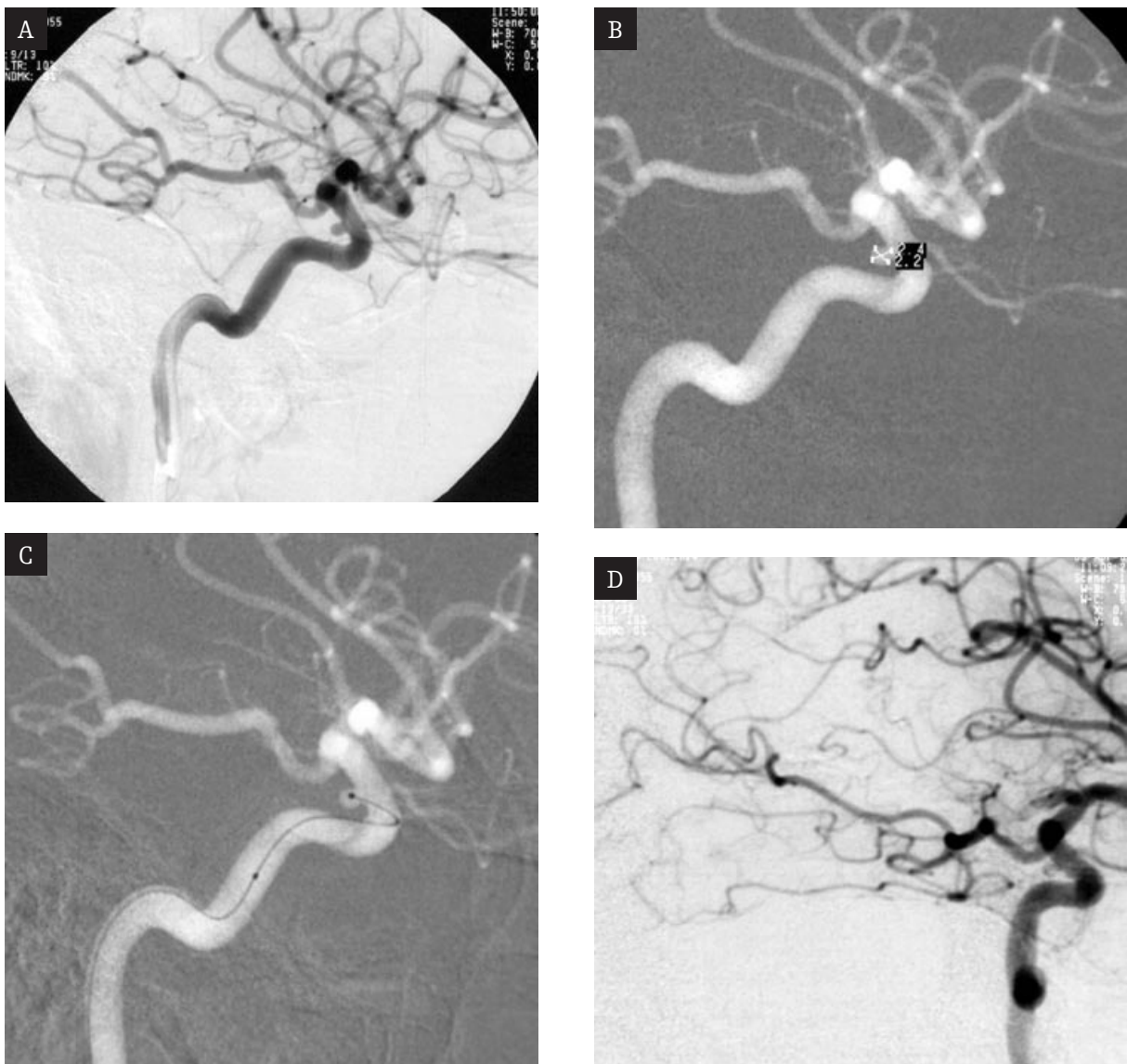


Figure 3. DSA right ICA arteriography in oblique view (A and B) showing "baby" aneurysm (app.diam.2,5mm) on the "road-map" view – micro-catheter in the aneurysmal sac (C). Note DSA right ICA in oblique view (D) aneurysmal sac completely packed by CDC-10 soft coils (2 mm x 8 cm).

Discussion

Spontaneous, complete or partial occlusion of the intracerebral angiomas is rarely observed – 1–3%. In literature there have only been 50 such cases described so far, including 29 cases of adult patients [9, 10, 11].

It is supposed that in 70 % of cases the reason for spontaneous process of occlusion of the brain angioma is an intracerebral hemorrhage, which causes pressure on the draining veins in their proximal segment. Decelerating the blood flow leads to progressive thrombosis [12, 13].

In our case significant hemodynamic lowering of the blood flow was a result of occlusion of about 80% of the nidus with the initial part of the main single draining vein by means of embolisation. The presence of the single draining vein is considered to be a favourable factor for the

process of spontaneous recovery of the intracranial angioma [11, 14]. Based on the analysis of 29 cases, Krapf et al. stated that the spontaneous occlusion relates to cerebral angiomas of small nidus (< 3 cm), located in the occipital lobe (16 cases out of 29) and mainly to the patients aged over 40 years [12]. The described patient matched those conditions.

In the described case a probable mechanism that lead to the spontaneous occlusion of the angioma was the lowering of blood flow by the rest of nidus and its consequent clotting. Moreover, regional proliferation of smooth muscle cells along with the cells of connective tissue was also probable [15]. This process leads first to narrowing the vessel and then to its complete occlusion. In the described case both of the phenomena occurred and resulted in spontaneous exclusion of the angioma from circulation.

The coexistence of aneurysms and cerebral angiomas has long been known and has been corroborated by numerous authors. The occurrence fluctuates between 3 to 58%, on average – 12% [4, 5, 6, 8, 16, 17].

Aneurysms of the type build up as a result of an elevated blood flow caused by the presence of arteriovenous angioma – that is why they are called „flow-related” angiomas. They are usually located within the arterial circle of Willis, in proximal segments of supplying arteries, in their distal segments or/ and in the nidus of angioma. Batjer et al. and Pötting et al. stated that rupture of aneurysm concomitant with angioma is the reason of hemorrhage in about 80% of cases of arteriovenous angiomas [5, 18]. This is the reason why most experts suggest treating the aneurysm first, and then the angioma [14, 16]. Such procedure is bound to lower the risk for aneurysm rupture in the hemodynamic conditions changed after partial embolisation of the nidus. Our course of action followed that rule. However, because of the wide base of aneurysm and turbulent blood flow inside of it, the operation was impossible to conduct. In order to that, the decision of preliminary embolisation of the angioma nidus was made in order to lower the blood flow and then to make another attempt to treat the aneurysm. The case of decrease or total disappearance of the aneurysm after excluding the angioma from circulation was described by many researchers [6, 17, 19].

The control angiography examination carried out before the next planned stage of treatment, 2 months after the

first operation, revealed complete lack of filling up of the cerebral angioma and massive aneurysm located on the junction of P1/P2 segments of the right posterior cerebral artery. The P1 segment was also occluded while the whole posterior artery was filling up with the right connective posterior artery, as was described in the previous examination.

Such reaction is an interesting example of spontaneous exclusion of functionally unneeded elements, such as an aneurysm with congenital vessel – the P1 segment, from circulation. The main route of blood inflow to the right posterior cerebral artery was always the interior carotid artery together with posterior connective artery on the same side. This route was used during the first operation in order to insert the microcatheter into the nidus.

Complete exclusion of two aneurysms and middle-size intracerebral angioma from circulation was achieved in the course of treatment by means of intravascular embolisation.

Conclusions

The intravascular embolisation is an effective and safe method for treating the intracerebral angiomas as well as the related aneurysms. In favorable hemodynamic and anatomical conditions partial embolisation of nidus of the angioma can lead to spontaneous clotting of its remaining part.

References:

1. Szczerbo-Trojanowska M, Szajner M. Rozdział: Neuroradiologia Zabiegowa. Neuroradiologia; Upowszechnianie Nauki – Oświata „UN-O” Warszawa 2000: 346–347.
2. Berenstein A, Lasjaunias P. Surgical Neuroangiography. Berlin: Springer-Verlag, 1992, Vol. 4.
3. Crawford P, West CR, Chadwick DW. Arteriovenous malformations of the brain: natural history in unoperated patients. *J Neurol Neurosurg Psychiatry* 1986; 49: 1–10.
4. Lasjaunias P, Piske R. Cerebral arteriovenous malformation and associated arterial aneurysms. Analysis of 101 CAVM cases, with 37 AA in 23 patients. *Acta Neurochir (Wien)* 1988; 91(1–2): 29–36.
5. Pötting M, Ross IB, Weill A. i wsp. Intracranial arterial aneurysms associated with arteriovenous malformations: endovascular treatment. *radiology* 2001; 220: 506–513.
6. Redekop G, TerBrugge K. Arterial aneurysms associated with cerebral arteriovenous malformations: classification, incidence, and risk of hemorrhage. *J Neurosurg* 1998; 89(4): 539–46.
7. Stapf C, Mohr JP. Concurrent arterial aneurysms in brain arteriovenous malformation with hemorrhagic presentation. *J Neurol Neurosurg Psychiatry* 2002; 73(3): 294–8.
8. Brown RD, Jr, Wiebers DO, Forbes GS. Unruptured intracranial aneurysms and arteriovenous malformations: frequency of intracranial hemorrhage and relationship of lesions. *J Neurosurg* 1990; 73: 859–863.
9. Abdulrauf SI, Malik GM, Awad IA. Spontaneous angiographic obliteration of cerebral arteriovenous malformations. *Neurosurgery* 1999; 44: 280–287.
10. Ezura M, Kagawa S. Spontaneous disappearance of a huge arteriovenous malformation: case report. *Neurosurgery* 1992; 30: 595–599.
11. Wakai S, Chen CH, Wu KY, Chiu CW. Spontaneous regression of a cerebral arteriovenous malformation: report of a case and review of the literature. *Arch Neurol* 1983; 40: 377–380.
12. Krapf H, Siekmann R, Freudenstein D i wsp. Spontaneous occlusion of a cerebral arteriovenous malformation: angiography and MR imaging follow-up and review of the literature. *Am J Neuroradiol* 2001; 22: 1556–1560.
13. Sartor K. Spontaneous closure of cerebral arteriovenous malformations demonstrated by angiography and computed tomography. *Neuroradiology* 1978; 15: 95–98.
14. Szajner M., Szczerbo-Trojanowska M. Przeznaczyniowa embolizacja jako nowoczesna metoda leczenia malfornacji naczyńiowych mózgu. *Magazyn Medyczny* 2001; 7: 26–36 15.
15. Pile-Spellman J, Baker KE, Liszczak TM, i wsp. High-flow angiopathy: cerebral blood vessel changes in experimental chronic arteriovenous fistula. *Am J Neuroradiol* 1986; 7: 811–815.
16. Cuhna MJ, Bennett M. The treatment of associated intracranial aneurysms and arteriovenous malformations. *J Neurosurg* 1992 Dec; 77: 853–859.
17. Perata HJ, Tomsick TA, Tew JM, Jr. Feeding artery pedicle aneurysms: association with parenchymal hemorrhage and arteriovenous malformation in the brain. *J Neurosurg* 1994; 80: 631–634.
18. Batjer H, Suss RA, Samson D. Intracranial arteriovenous malformations associated with aneurysms. *Neurosurgery* 1986; 18: 29–35.
19. Turjman F, Massoud TF. Aneurysms related to cerebral arteriovenous malformations: superselective angiographic assessment in 58 patients. *Am J Neuroradiol* 1994 Oct; 15(9): 1601–5.