International Journal of Science and Research (IJSR)
ISSN (Online): 2319-7064
Index Copernicus Value (2015): 78.96 | Impact Factor (2015): 6.391

Endovascular Embolisation of a Complex Arteriovenous Malformation

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1. Introduction

Arteriovenous brain malformations are congenital diseases, considered anomalies in the development of the cerebral vessels. AVM can be localized in any brain region, their most frequent localization being in the basin of the middle cerebral arteries. They have different size starting from few millimeters to affecting the entire brain hemisphere. The supplying arteries may be one or several, and may be with a dilated lumen or just conform to the remaining normal cerebral arteries. The draining vein may also vary depending on their number, so they can be deep or cortical.[1]

From anatomic viewpoint they represent a complex net of afferent arteries and efferent (draining) veins, which are connected by a pathological capillary base – the so called nidus.

Clinically the brain AVMs are cause for mortality or long-lasting disablement of patients due to intracranial hemorrhage or epilepsy, for which they are the underlying reason.

2. Clinical Case

19year old female patient was admitted into the hospital in comatose condition following a seizure. The computer tomography scan visualized subarachnoid hemorrhage with an existence of intraparenchymal hematoma infratentorially in the left.The performed CT angiography diagnosed AVM infratentorially, in the left. The subsequent conventional angiography visualized fill-out of the pathological nidus from the super-cerebellar cerebellum artery,the latter being drained by a dilated venous vessel in direction of the transversal sinus.

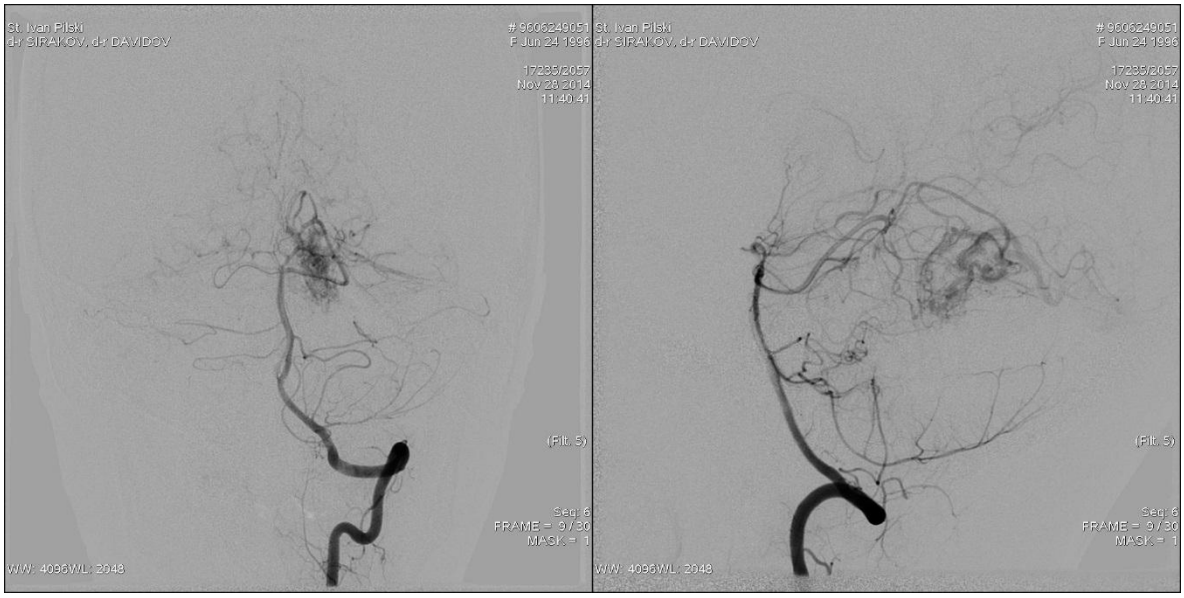

Figure 1: Visualization of the arteriovenous malformation

Following consultation by a multidisciplinary team of radiologists and neurosurgeons a decision was taken for endovascular treatment of the malformation due to better prognostic and therapeutic result for the patient.

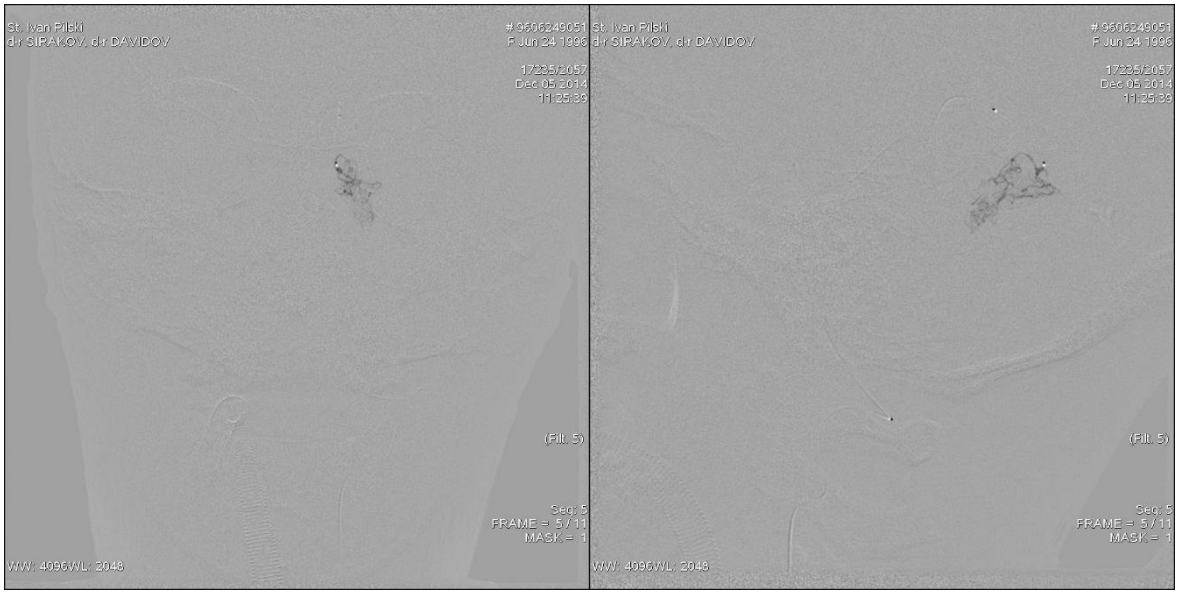

Figure 2: Selective catheterization of the pathological vascular nidus

The goal of this endovascular treatment undertaken as an emergent procedure was to isolate the malformation that had bleeding from the normal blood circulation. The pathologic nidus was reached by placement of 6F guiding catheterEnvoy, its tip positioned into the left vertebral artery at the level of PICA. Into the lumen of the latter a micro-catheterAppolo was supplied with 3 cm,mechanically detachable' tip, placed in the malformation. Total nidus embolization was thus accomplished.

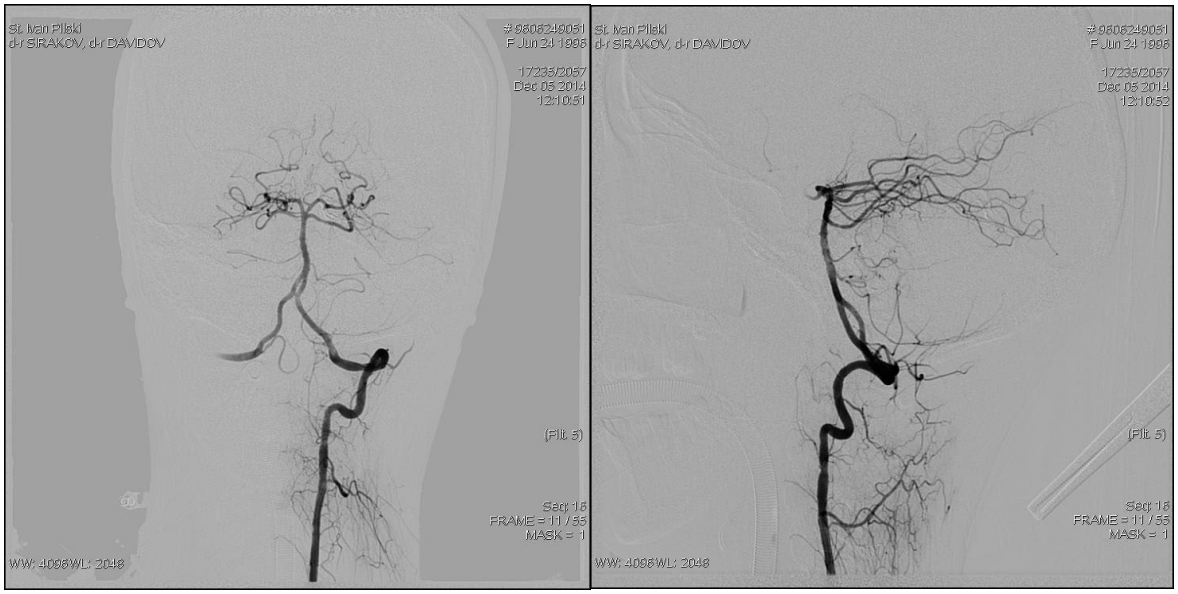

Figure 3: Control angiography with no data of residual malformation

The patient overcame the postoperative period and subarachnoid hemorrhage with no residual neurologic deficiency and was discharged 7 days after the endovascular procedure. During the last 6 months the patient has no seizures or new episodes of subarachnoid hemorrhage (SAH).

3. Discussion

Some authors think that AVMs are associated with Rendu-Osler-Webersyndrome as in 30% of the population with such a syndrome there is an AVM. Other authors are of the opinion that a mutation in the gene, localized in 9qchromosome causes abnormity in,endoglin' – the transforming growth factor of β- connecting protein of the endothelial cells.[2]Yet another reason for this malformation is considered to be a variant in the 12qchromosome, causing mutation in the activin-receptor kinase (A LK – 1 gene) which provokes expression of the growth of endothelial cells. [3] The brain arteriovenous malformations are often symptomatic in young people, usually before reaching 40 years of age[4].

From anatomic point of view the natural course of arteriovenous malformations may in rare cases include enlargement, shrinkage or regression.Surprisingly, in a small group of 20 patients, monitored by angiography for periods ranging from 5 to 28 years, *Minakawa*witnessed

enlargement in the size of arteriovenous malformations in 4 patients, shrinkage in four and full regression in another four. Enlargement of the brain arteriovenous malformations was observed in young patients (below the age of 30) and particularly in child age[6,7].

Intracranial hemorrhage is the most common clinical manifestation of the arteriovenous malformation, its incidence ranging between 30% and 82%[8].It is of prime importance to identify the factors, increasing the risk of bleeding of brain arteriovenous malformation regarding the treatmentstrategy.

It is assumed that there is connection between the size of the arteriovenous malformation and its susceptibility to rupture. Within the groups of *Graf et al. (1983)*[9],the risk of hemorrhage for a period of 5 years have been 10% for large arteriovenous malformations (>3.0 cm in dimeter) and 52% for small arteriovenous malformations (<3 cm in diameter). In the groups of *Spetzler et al. (1992)*[10], in 82% of the patients with small arteriovenous malformations (less than 3 cm), 29% of the patients with medium size arteriovenous malformations (3–6 cm) and 12% of the patients with large arteriovenous malformations (larger than 6 cm) hemorrhage has occurred.

The multivariate analysis in large groups of patients, studied by *Mansmann et al. (2000)*[11],has also found out that arteriovenous malformationsize larger than 3 cm is a factor, negatively associated with intracranial hemorrhage.

Nevertheless the absolute risk of sudden intracranial hemorrhage in small and large brain arteriovenous malformations is still controversial. In the groups of *Crawford et al (1986)*[12], 21% small and 18% large arteriovenous malformation bleed again within 5 year period. The small and large arteriovenous malformations may have identical risk of bleeding. Large arteriovenous malformations may more frequently be expressed in other ways different from the hemorrhage (convulsions, progressive paralysis, headache), which could lead to acceleration of the bleeding incidence in the small arteriovenous malformations.

Deep venous drainage is associated with higher risk of bleeding[13,14] . Superficial and deep venous drainage differ from anatomic point of view. The veins from the central drainage have one common end path, which is the vein of *Galen*and sinus rectus. On other hand the superficial veins have more bifurcations and can be drained posteriorly through the upper sagittal sinus and anteriorly through the Sylvian vein. The superficial venous system is probably more flexible in adapting to the hemodynamic situation, established by the presence of arteriovenous malformation.

4. Conclusion

The endovascular embolization of arteriovenous malformations, even when they are of complex anatomic structure remain the key method for their treatment, which is of proven effectiveness and low risk of complications.

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