Predictors of Neurological Deficit after Endovascular Treatment of Cerebral Arteriovenous Malformations and Functional Repercussions in Prospective Follow-Up

JOSE JORDAN, JUAN CARLOS LLIBRE, FRANK VAZQUEZ

Interventional Neuroradiology Unit, CIMEQ, Institute of Neurology and Neurosurgery; La Habana, Cuba

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SUMMARY – Endovascular therapy is a well-established approach to the treatment of cerebral arteriovenous malformations (AVMs). The objective of this study was to determine the predictive factors of neurological deficit following endovascular procedures. Seventy-one patients with cerebral AVMs who underwent 147 embolization sessions from 2006 to 2011 were followed up prospectively (average 31.1 ± 17.5 months). Functional neurological condition was documented by means of the modified Rankin scale. Factors found to be predictors of neurological deficit were the partial obstruction of drainage veins (OR = 197.6; IC = 2.76 - 1416.0; $P = 0.015$), a positive result in the Propofol test (OR = 50.2; IC = 6.18 - 566.5; $P = 0.000$), AVM diameter under 3 cm (OR = 21.3; IC: 1.71 - 265.6; $P = 0.018$), the presence of intranidal aneurysms (OR = 11.2; IC = 1.09 - 114.2; $P = 0.042$), the absence of post-procedure hypotension (OR = 10.2; IC = 1.35 - 77.7; $P = 0.003$), deep venous drainage (OR = 7.14; IC = 1.15 - 44.4; $P = 0.035$), and devascularization in excess of 40% per session (OR = 3.3; IC = 1.11 - 16.8; $P = 0.056$). Fifty-six patients (78.9%) did not experience changes in their neurological condition after the treatment and 13 patients (18.3%) showed a new neurological deficit related to the treatment; 95.8 % of the patients did not show significant long-term incapacity. Partial obstruction of drainage veins, small AVMs, intranidal aneurysms, faulty hemodynamic control and extensive devascularization were found to be predictors of neurological deficit. A significant number of patients with neurological deficit improved in the long term.

Introduction

Arteriovenous malformations (AVMs) are the cause of 1 to 2% of strokes, 3% of ictuses in young adults, 9% of subarachnoid hemorrhages, 4% of intracerebral primary hemorrhages and at least one third of the intracerebral primary hemorrhages in young adults¹. The incidence of hemorrhage from AVMs is 0.42/100 000 persons-years². The annual mortality rate fluctuates in the 1-1.5% range, with a 2% yearly risk of hemorrhage of non ruptured AVMs, 18% rebleeding risk in the first year and 2-4% every consecutive year¹. Mortality from a hemorrhagic episode is 10-15% and morbidity, 20-50%³⁻⁵.

Current therapeutic options for AVMs include microvascular neurosurgery, radio neurosurgery, and endovascular embolization. Endovascular treatment is usually the primary treatment option and is combined with surgery or radiosurgery.

The reported incidence of complications from endovascular treatment varies between 3% and 25%⁶⁻¹⁸. Permanent morbidity and mortality rates fluctuate in the 3.8-14% and 1.0-3.7% ranges, respectively¹⁹.

Several studies^{11,12,14,15,18-20} have evaluated the predictive factors of complications in endovascular treatment of AVMs: age, the number of embolization sessions, a normal neurological condition in the initial evaluation, number of

embolized pedicles, deep venous drainage, localization in an eloquent area, venous embolization and nidus diameter.

The objective of this study was to determine the predictive factors of neurological deficit, and to evaluate the functional repercussions of long-term complications prospectively in the endovascular treatment of AVMs.

Materials and Methods

A longitudinal prospective study was carried out in 71 patients with cerebral AVMs after 147 embolization sessions with n-butyl cyanoacrylate (n-BCA) between April 2006 and April 2011, in the endovascular therapy unit of the Medical Surgical Research Center. To determine the predictive factors of neurological deficit and evaluate the functional repercussions of long-term complications in endovascular treatment of AVMs (average follow-up: 31.1 ± 17.5 months), demographic, morphological and clinical data of the treatment and post-embolization results were collected.

The functional neurological condition was documented before embolization and after each endovascular procedure until the end of the treatment, by means of the modified Rankin's scale (mRS)²¹. Any decline in the patient's condition with respect to the pre-embolization Rankin score was considered a neurological deficit associated with the procedure. This included worsening in patients with pre-existing neurological deficit. The new neurological deficit was classified as functionally irrelevant (mRS = 1), non-incapacitating (mRS = 2), and incapacitating (mRS $\geq$ 3).

Endovascular Technique

Cerebral pan-angiography was carried out applying the transfemoral approach in angiographs featuring digital subtraction and road-mapping (Philips Integris and Siemens Artis). A flow-guided Magic 1.2 or 1.5 microcatheter and Balt Sorcerer 0.009 microguide were inserted, placing the former as close as possible to the nidus or intranidally, so as to carry out superselective angiography, followed by a superselective test with Propofol. Then embolization with n-BCA was performed.

We initially carried out extensive devascularization, 40-60% per session, by embolizing multiple arterial pedicles, without strict post-procedure hemodynamic control. After the on-

set of hemorrhagic complications, devascularization was restricted to 25-30% per session and strict post-embolization hemodynamic control was established, involving a 20% reduction of the mean arterial pressure with respect to basal values and continuous monitoring.

Statistical Analysis

A univariate analysis was carried out to describe the effect of demographic factors, presentation symptoms, pre-embolization neurological condition, morphological parameters, number of embolization sessions, percentage devascularization per session, number of pedicles embolized, hemodynamic control and other factors relevant to the risk of post-embolization neurological deficit.

The predictive factors and all the variables with a significant relationship in the univariate analysis ($P < 0.05$), or according to expert criteria, were included in the multiple logistic regression model to evaluate their independent association with post-procedure neurological deficit, controlling sex and age. The analysis includes Spetzler & Martin's components and general score.

The analyses were carried out using Stata 9.2, 2007 (Stata Statistical Software: release 10; StatCorp, College Station, TX, USA).

Results

The clinical, demographic, morphological and general characteristics of the treatment for AVMs in the studied series were reported in previous papers^{22,23}.

The predictive factors of procedure-related neurological deficit given by the univariate analysis were: the presence of intranidal aneurysms (OR: 4.17; IC: 1.1–17.9; $P = 0.055$); AVM smaller than 3 cm (OR: 3.05; IC: 0.74–12.6; $P = 0.124$), partial obstruction of drainage veins (OR: 24.2; IC: 2.03–288.2; $P = 0.012$), devascularization above 40% (OR: 3.21; IC: 1.49–6.92; $P = 0.003$), absence of post-procedure hypotension (OR: 4.77; IC: 1.08–14.2; $P = 0.017$) and positive result of the Propofol test (OR: 11.3; IC: 2.93–43.8; $P = 0.000$). Post-procedure hypotension was a protective factor (OR: 0.27; IC: 0.08–0.86; $P = 0.027$) (Table 1).

The multiple logistic regression analysis (Table 2) confirmed as predictive factors of neurological deficit: the partial obstruction of drainage veins, with OR = 197.6 (IC: 2.76–1416.0; $P =$

Table 1 Predictive factors of neurological deficit in univariate analysis.

Characteristics	No. (%)	OR	IC: 95 %	P
Males (n = 71)	41 (57.7)	1.08	0.47 – 2.48	0.860
Racial morphotype black/mixed race (n = 71)	26 (36.7)	1.88	0.59 – 5.93	0.283
Embolization sessions > 3 (n = 47)	24 (33.8)	1.17	0.24 – 5.72	0.845
Absence of pre-procedure neurological deficit (n = 71)	63 (45.6)	1.87	0.39 – 8.86	0.430
Initial presentation (n = 71)				
Hemorrhage	41 (57.7)	1.49	0.44 – 5.09	0.524
Epileptic seizures	8 (11.3)	0.57	0.07 – 4.69	0.604
Convulsions	13 (18.3)	1.08	0.28 – 4.21	0.905
Headache	51 (71.8)	1.14	0.33 – 3.89	0.837
High tension	5 (7.0)	1.08	0.28 – 4.21	0.905
Deep afferences (n = 71)	8 (11.3)	–	–	–
Associated aneurysms (n = 71)				
Any of the above-mentioned	9 (12.7)	2.38	0.59 – 9.63	0.224
Flow-related	–	–	–	–
Intranidal	5 (7.0)	4.17	0.97 – 17.9	0.055
Spetzler-Martin components (n = 71)				
Small AVM (< 3 cm)	12 (16.9)	3.05	0.74 – 12.6	0.124
Average-sized AVM (3 – 6 cm)	50 (70.4)	0.59	0.18 – 1.91	0.378
Large AVM (> 6 cm)	9 (12.7)	0.76	0.16 – 3.6	0.725
Deep venous drainage	25 (35.2)	2.7	0.83 – 8.67	0.098
Location in eloquent area	48 (67.6)	0.34	0.11 – 1.08	0.066
Spetzler-Martin grades (n = 71)				
Grade 1	2 (2.8)	–	–	–
Grade 2	13 (18.3)	2.21	0.55 – 8.89	0.263
Grade 3	42 (59.2)	1.11	0.35 – 3.59	0.856
Grade 4	12 (16.9)	0.29	0.04 – 2.31	0.242
Grade 5	2 (2.8)	1.78	0.19 – 16.0	0.608
Post-procedure hypotension (n = 147)	99 (67.3)	0.27	0.08 – 0.86	0.027
Partial obstruction of drainage veins (n = 147)	3 (2.8)	24.2	2.03 – 288.2	0.012
Devascularization > 40 % (n = 147)	35 (23.7)	3.21	1.49 – 6.92	0.003
More than one pedicle per session (n = 147)	19 (13)	1.25	0.26 – 6.14	0.782
Positive Propofol test result (n = 147)	12 (8.2)	11.3	2.93 – 43.8	0.000
Afferences of more than two major vessels of the Willis polygon (n = 71)	32 (45.1)	2.69	0.79 – 9.17	0.113
Post-procedure hemorrhage (n = 147)	6 (4.1)	–	–	–
Post-procedure infarction (n = 147)	5 (3.4)	–	–	–

0.015); a positive Propofol test, with OR = 50.2 (IC: 6.18 – 566.5; P = 0.000); diameter smaller than 3 cm, with OR = 21.3 (IC: 1.71–265.6; P = 0.018); the presence of intranidal aneurysms, with OR = 11.2 (IC: 1.09–114.2; P = 0.042); the

absence of post-procedure hypotension, with OR = 10.2 (IC: 1.35–77.7; P = 0.003); deep venous drainage, with OR = 7.14 (IC: 1.15–44.4; P = 0.035) and devascularization larger than 40% per session, with OR = 3.3 (IC: 1.11–16.8; P = 0.056).

Table 2 Multiple logistic regression model evaluating the association of demographic and morphologic parameters with a neurological deficit.

<i>Parameters</i>	<i>OR</i>	<i>IC: 95 %</i>	<i>P</i>
Absence of pre-embolization neurological deficit	1.12	0.58 – 7.47	0.739
Absence of post-procedure hypotension	10.2	1.35 – 77.7	0.003
Positive Propofol test result	59.2	6.18 – 566.5	0.000
Diameter under 3 cm	21.3	1.71 – 265.6	0.018
Deep venous drainage	7.14	1.15 – 44.4	0.035
Intranidal aneurisms	11.2	1.09 – 114.2	0.042
Partial obstruction of drainage veins	197.6	2.76 – 1416.0	0.015
Devascularization > 40 % per session	3.3	1.11 -16.8	0.056

Table 3 Time behavior of the general functional results.

<i>Score in Rankin scale</i>	<i>Initial evaluation (%)</i>	<i>Post-embolization n (%)</i>	<i>Long-term follow-up</i>
0	56 (78. 9)	43 (60. 6)	60 (84. 5)
1	13 (18. 3)	19 (26. 8)	7 (9. 9)
2	–	4 (5. 6)	1 (1. 4)
0 – 2	69 (97. 2)	66 (92. 9)	68 (95. 8)
3	2 (2. 8)	3 (4. 2)	1 (1. 4)
4	–	–	–
5	–	–	–
6	–	2 (2. 8)	2 (2. 8)

No statistically significant relationship in the regression was found in the multiple logistic analysis between the Spetzler and Martin score or its components and the risk of procedure-related neurological deficit.

In the initial evaluation by the modified Rankin scale, 78.9% of the patients did not show neurological deficit (mRS = 0); 18.3%, 13 patients presented functionally irrelevant neurological deficits (mRS = 1), 12 of them as a consequence of hemorrhagic episodes and one secondary to ischemic ictus and 2.8% of the cases (two patients) showed incapacitating deficits (Rankin = 3) due to intracranial hemorrhage.

According to the modified Rankin scale, 56 patients (78.9%) did not experience changes in neurological condition after the treatment. A total of 13 patients (18.3%) showed a new treatment-related neurological deficit, including two (2.8%) who died from a complication of the procedure, one (1.4%) with incapacitating deficit (mRS = 3), four (5.6%) suffered non-incapacitating deficit (mRS = 2), and six (26.8%) presented a functionally irrelevant neurological deficit (mRS=1).

After the embolization session that caused

the new neurological deficit, 11 patients (15.5%) continued the endovascular therapy; in four patients (5.6%), all with cerebral infarction, complete devascularization of the AVM was achieved at the time of this complication, with total recovery of the new neurological deficit after rehabilitation, achieving an mRS similar to the pre-embolization value. The other two patients who developed neurological deficit died as a consequence of procedural complications.

In the long-term follow-up (mean of 31.1±17.5 months), 95.8% of the patients did not exhibit significant incapacity; irrelevant neurological deficit (mRS = 1) persisted in four patients; and both non-incapacitating (mRS = 2) and incapacitating neurological deficit (mRS = 3) affected one patient; the remaining patients regained their pre-procedure Rankin score (Table 3).

Discussion

Predictive factors for neurological deficit in our series were the partial obstruction of drainage veins, AVMs smaller than 3 cm, intranidal

aneurysms, a positive value of the Propofol test, faulty hemodynamic control and extensive devascularization. Some of these factors have been reported by other authors.

Hartmann et al.¹² evaluated neurological outcomes by the Rankin scale in 233 patients who had undergone 545 embolization sessions, multivariate analysis finding that age, the number of embolization sessions and a normal neurological condition in the initial evaluation exhibited a significant relationship with complications related to the endovascular treatment. The other variables assessed did not show a significant relationship. Neither the general score of the Spetzler-Martin scale ($P = 0.163$) nor its three separately evaluated components predicted the results of the treatment. Restricting the univariate analysis to incapacitating neurological deficit and death, they found significant relationships for age, initial presentation with hemorrhage, small AVM size and the presence of deep arterial afferences.

Jayaraman et al.¹⁹, in their series, reported the factors associated with new deficits included the number of embolization sessions and the absence of pre-treatment neurological deficit. They put forward the hypothesis that the absence of pre-treatment neurological deficit is due to the fact that new deficits are easier to identify in patients without pre-existing deficits. They did not find that the Spetzler-Martin score or any other specific morphological characteristic predicted the risk of permanent deficit.

Haw et al.'s¹⁸ multivariate analysis reported that the factors associated with complications included the presence of high-flow fistulas, a nidus with a fistulous component, involvement of the membrane and venous embolization with the polymer.

Ledezma et al.¹⁴, in a multivariate analysis, found that peri-procedure hemorrhage and grades 3 and 4 of the Spetzler-Martin scale were predictors of post-embolization complications.

Kim et al.¹⁵ reported that the number of embolized pedicles was significantly related to the onset of neurological deficits. Spetzler-Martin high grades were apparently related to a high risk of new neurological deficits, but this was not statistically significant.

Starke et al.¹¹ reported that the predictive factors of new neurological deficits due to embolization in a univariate analysis ($P < 0.15$) were the execution of more than one embolization procedure and location in an eloquent area. Moreover, patients with AVMs of average diameter (3 – 6 cm) had a smaller risk of neu-

rological deficit due to embolization, while patients with large AVMs (> 6 cm) had a higher probability. In a multivariate analysis controlling gender, the following pre-embolization variables were predictors of new neurological deficit: performing more than one embolization procedure, deep venous drainage, location in an eloquent area and small AVM diameter. Although they had four patients with wide diameter AVMs, a size above 6 cm also significantly predicted new post-embolization deficits.

In a univariate analysis of another series of 119 patients with 240 superselective embolizations, Hartmann et al.²⁴ reported that the high frequency of embolizations, the absence of basal neurological deficit, the non-hemorrhagic presentation of the AVM, large AVM diameters, Spetzler and Martin score and its elements, "deep venous drainage" and "location in eloquent area" were associated with neurological deficits related to long-term treatment, regardless of the fact that no significant relationship was found in the multivariate analysis for some variables²⁴.

The fact that extensive devascularization and faulty hemodynamic control are predictive factors of neurological deficit is linked to the increased hemorrhage rate associated with them, in agreement with the findings of Heidenreich et al.²⁰, that extensive devascularization, over 60% per session, increases the risk of post-procedure hemorrhage, constituting a risk factor for hemorrhage (OR = 18.8; IC: 95%). In addition, better results were obtained in patients cared for in postoperative intensive care units that in those looked after in medical intensive care units or in ictus units due to better post-procedure control of arterial pressure.

On the other hand, Jayaraman et al.¹⁹ reported only 1.6% hemorrhagic complications with intensive blood pressure monitoring for 24-48 h, and MAP of 65-75 mmHg, which in their opinion minimizes the sudden elevation of post-embolization systolic arterial pressure and may reduces the risk of post-embolization hemorrhage.

In our study, similar to others^{11,12,18,19}, none of the grades of the Spetzler and Martin classification, and none of its three components predict treatment-related complications. This system was originally designed and validated to predict the results of surgical treatment^{25,26} and its application in endovascular therapy is not well-defined^{1,3,12}.

The rates of post-procedure morbidity-mortality in our study are within the ranges described elsewhere^{6,7,18,19}. Restoration of the neurological deficits in the long-term follow-up is

noteworthy, though in most cases functionally irrelevant deficits persist.

In their series using the Rankin scale, Hartmann et al.¹² reported that 200 patients subjected to endovascular treatment did not experience changes in their neurological state after the treatment, and in 92% the deficit was not incapacitating. Of the 200 patients that did not experience changes in their functional neurological condition, 41 had pre-existing neurological deficits that remained unchanged after the endovascular treatment.

Jayaraman et al.¹⁹ reported that in their series 171 patients (89%) presented a pre-embolization mRS of 0–2. After embolization, 172 patients (90%) showed mRS in the same range. However, four patients (2.3%) with pre-treatment mRS <2 showed post-embolization mRS >2. Three of these patients presented complications related to the endovascular procedure.

Hartman et al.²⁴ reported that in a series of 119 patients undergoing 246 superselective embolizations, in the initial evaluation by the

modified Rankin scale 96% of the patients did not present functionally relevant neurological deficit (mRS = 0 or 1), and in 99% of them it was non-incapacitating (mRS = 2).

Starke et al.¹¹ reported that before the first embolization 91% of the patients did not present significant incapacity (mRS = 0-2) and, after the last embolization, 86% of the patients did not present significant incapacity. In the long-term follow-up (mean of 43.4±34.6 months), 94% of the patients did not show significant incapacity.

Conclusions

Our study found as predictive factors of neurological deficit the partial obstruction of drainage veins, AVMs smaller than 3 cm, intranidal aneurysms, a positive result of the Propofol test, faulty hemodynamic control and extensive devascularization. A significant number of patients with neurological deficit improved in the long-term prospective follow-up.

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Juan Carlos Llibre, MD
Interventional Neuroradiology Department
Institute of Neurology and Neurosurgery
29 y D vedado, Plaza
La Habana, 10400 Cuba
E-mail: pereraelianne@infomed.sld.cu