Interdisciplinary Neurosurgery: Advanced Techniques and Case Management 27 (2022) 101379

Contents lists available at ScienceDirect

Interdisciplinary Neurosurgery: Advanced Techniques and Case Management

journal homepage: www.elsevier.com/locate/inat

Research Article

Comparing the frequency of symptomatic cerebral vasospasm and vasospasm-related ischemia in Fisher 3 grade ruptured anterior communicating artery aneurysms treated via microsurgical clipping or endovascular coiling

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ARTICLE INFO

Keywords:
Endovascular coil
Microsurgical clip
Subarachnoid hemorrhage
Symptomatic vasospasm
Ischemia

ABSTRACT

Background and purpose: Ischemia due to cerebral vasospasm still remains an important cause of morbidity and mortality after aneurysmal SAH. Studies that compared the incidence of ischemia due to vasospasm between clip and coil therapies are still controversial. We compared vasospasm-induced ischemia rates between clip and coil therapies for ruptured anterior communicating artery aneurysms.

Materials and methods: We retrospectively analyzed cases of ruptured anterior communicating artery treated with clips or coils within 36 h after rupture. Patients with Glasgow Coma Scale scores < 13; Fisher grades of 1, 2, or 4; multiple aneurysms; aneurysms associated with an arteriovenous malformation; stent- or balloon-assisted coiling; and complications during or after the clipping and coiling procedure were excluded from the study.

Results: Although the incidence rates of ischemia and symptomatic vasospasm were higher in the coil group, no statistically significant difference was found between the two groups (P = 0.278 and P = 0.270, respectively).

Conclusion: We found no significant difference in the incidence of vasospasm-induced ischemia between the patients treated with clipping and those treated with coil therapy who had Fisher grade 3 SAH and good clinical conditions. The amount of blood in basal cisterns on computed tomography, vascular manipulation, and brain retraction had the same triggering effect on vasospasm-induced ischemia.

1. Introduction

The main complications after aneurysmal subarachnoid hemorrhage (SAH) are rebleeding and ischemia due to cerebral vasospasm [1]. Early occlusion of the aneurysm eliminates rebleeding development, but ischemia due to cerebral vasospasm remains an important cause of morbidity and mortality [2]. The pathophysiology of vasospasm is extremely complex, and a significant number of experimental and clinical studies have been conducted to identify and prevent its development. The amount of cisternal blood on computed tomography (CT) has been considered by many authors as the most important predictive factor of vasospasm [3,4], in addition to other factors including vessel manipulation during surgery, low Glasgow Coma Scale (GCS) score on admission, smoking, hypertension before SAH development, and being older than 50 years of age [5–7].

Ruptured aneurysms can be treated with clipping or coiling. However, which of these treatments results in lower incidence for vasospasm or vasospasm-related ischemic complication(s) is unknown. Some authors have reported that early surgery reduces the incidence of vasospasm and ischemic complications due to the removal of blood clots in the basal cistern [8–10]. However, advocates of endovascular treatment have concluded that the incidence rates of vasospasm and vasospasm-related ischemic complications were lower with this approach, due to the absence of vascular manipulation and brain retraction [11,12]. There are also studies which, despite demonstrating a significant difference in the frequency of all-cause ischemic infarction with the use of different approaches, have found similar frequencies of vasospasm-related ischemic infarction in patients treated with the clipping or

Abbreviations: SAH, subarachnoid hemorrhage; CT, computed tomography; GCS, Glasgow Coma Scale; AcoA, anterior communicating artery; A1, precommunicating segment of the anterior cerebral artery; DSA, digital subtraction angiography.

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https://doi.org/10.1016/j.inat.2021.101379
Received 26 May 2021; Received in revised form 11 September 2021; Accepted 12 September 2021
Available online 20 September 2021

coiling methods, indicating a need for further studies on this topic [13].

As such, conducting further studies that compare treatment models in ruptured AcoA aneurysms can provide more accurate results in terms of the risks and advantages of treatments, thereby enabling better selection of approach on a case-by-case basis. The purpose of this study was to compare the frequency of vasospasm-related ischemic complications in patients who underwent early treatment with clipping or coiling for ruptured AcoA aneurysms, in order to identify which method may have lower risks.

2. Material and methods

We retrospectively analyzed cases of ruptured AcoA aneurysms in patients treated with clips or coils by two hybrid vascular neurosurgeons, between January 2015 and December 2020, at the Neurosurgery Departments of two University Hospitals, Adnan Menderes University Faculty of Medicine and Biruni University Medicana International Istanbul Hospital. In both institutions, the patient registry and national population management software were used to obtain required data.

During this period, a total of 166 patients had been treated for AcoA aneurysms. Inclusion criteria were, being older than 18 years of age at time of intervention, having Fisher grade 3 lesion, and having only received treatment for AcoA aneurysm during the procedure. The decision to include only Fisher grade 3 lesions was made because we aimed to compare the two methods in patient groups with similar baseline risk for cerebral vasospasm (and related ischemia). It is well established that the amount of blood present in the cisterns (which is also the basis of Fisher grading) is a primary factor associated with vasospasm risk [3,14]. Patients with Fisher grade 1 or 2 lesions are unlikely to suffer from vasospasm, while those with grade 3 or 4 lesions have increased likelihood. Thus, in order to standardize the vasospasm risks of the two groups, we chose to include only patients with Fisher grade 3 lesions.

Patients with Glasgow Coma Scale (GCS) scores of <13; Fisher grades of 1, 2, or 4; those with multiple aneurysms or aneurysms associated with an arteriovenous malformation; subjects who had been treated with stent- or balloon-assisted coiling or had undergone the procedure after more than 36 h had passed following SAH development; and individuals who had developed complications during or after the clipping or coiling procedure were excluded from the study. Of the 166 patients, 92 met the inclusion/exclusion criteria. The patients were divided into two groups according to treatment type; 52 patients were treated with endovascular coiling, while 40 were treated with microsurgical clipping.

2.1. Management of aneurysmal SAH

All the patients underwent a CT scan for the confirmation of SAH diagnosis, which was followed by 4-vessel cerebral angiography and three-dimensional rotational angiography for the diagnosis of AcoA aneurysm. After the three-dimensional angiographic evaluation, the decision for the treatment method (treatment planning) was made by the hybrid vascular neurosurgeons in accordance with the following properties (and with respect to the particular characteristics of each patient): aneurysm dome-neck ratio, aneurysm geometric shape and projection, and carotid and anterior cerebral artery tortuosity.

All patients were hospitalized in the intensive care unit before the procedure, and dexamethasone (4 × 4 mg), paracetamol (4 × 1 mg) and glycerol trinitrate treatments were started with intravenous fluids (100 ml/hour) for those with blood pressure values exceeding 120/80 mmHg. Lumbar drainage was applied to all patients. After intervention, each patient underwent an immediate postoperative CT scan. Following the securing of the aneurysm with a clip or coil, standard hemodilution, hypertension, hyperhydration (triple-H) treatment [15], and intravenous nimodipine were administered to all patients for a minimum of 10 days. Angiography was performed in patients with clinical signs and symptoms of cerebral vasospasm (new neurological deficit, paralysis or paresthesia in the face or extremities, disruptions in motor or sensory function, headache progression or onset after initial regression, decreased consciousness) with respect to risk factors, including the identification of excessive subarachnoid blood or large blood clots on CT imaging and comorbidities (hypertension, diabetes mellitus, heart disease) [16]. In all suspected cases, cerebral vasospasm was confirmed via angiography. Papaverine was injected in the affected vessels during angiography in any patients who developed symptomatic vasospasm [17]. CT imaging was ordered immediately after surgery and immediately after symptom detection, and it was repeated whenever necessary when symptoms did not regress after initial inconclusive imaging. The detection of a new hypodense area was defined as vasospasm-related cerebral infarction, given that findings were unassociated with the surgery or the initial injury.

2.2. Microsurgical clip procedure

The standard pterional approach was utilized in all patients. The head was fixed with a Mayfield holder, and a right or left approach was decided based on the shape and geometric structure of the dome and neck and the precommunicating segment of the anterior cerebral artery (A1) that supplied the aneurysm. The Sylvian fissure was opened widely starting from the distal part. The lamina terminalis was opened for additional relaxation of the brain. Approximately 2 mm of the gyrus rectus was removed, and the frontal lobe was gently retracted for better exposure of the aneurysm and the AcoA complex.

After the aneurysm was clipped, careful attention was given to ensure that normal vessels did not remain in the clip via microscopic examination –confirmed with micro-Doppler imaging. After clipping, the cisterns were irrigated with plenty of saline, the blood clots were removed as much as possible, and sodium nitroprusside-impregnated surgicel was laid on the vessels.

2.3. Endovascular coil procedure

Under general anesthesia, coiling was performed using a standard transfemoral transarterial single-microcatheter technique. Anticoagulation was initiated by an intravenous heparin bolus injection of 5000 IU at the beginning of the procedure, and the anticoagulation state was monitored throughout the procedure by way of measuring aPTT which was kept between 26 and 35 s. After coiling, control angiographies were taken, and all the vessels were shown to be patent.

2.4. Statistical analyses

All statistical analyses were performed using IBM SPSS version 25.0 (SPSS Inc., Chicago, Illinois, USA). Continuous variables were found to conform to normal distribution in both groups (Shapiro-Wilk test and Q-Q plots). In the tables, continuous variables are presented as mean ± SD, while categorical variables are presented as number (n) and percentage (%). Comparisons between the groups were made using the independent samples *t*-test for continuous variables, while chi-square tests were used for the comparison of the distribution of categorical variables. A p value of < 0.05 was considered to show statistical significance.

3. Results

Of the 40 patients in the clip group, 23 were male and 17 were female, with ages ranging from 31 to 75 years (mean ± SD: 51.01 ± 11.87). The GCS score at hospital admission was 13 in 6 patients, 14 in 10 patients, and 15 in 24 patients (mean ± SD: 14.45 ± 0.74). Eleven patients developed symptomatic vasospasm (27.5%), and vasospasm-related ischemia was identified in five patients (12.5%).

Of the 52 patients in the coil group, 35 were male and 17 were female, with ages ranging from 20 to 79 years (mean ± SD: 56.90 ± 11.77). The GCS score at hospital admission was 13 in 17 patients, 14 in 22 patients, and 15 in 13 patients (mean ± SD: 13.92 ± 0.76). Ischemia

due to vasospasm was observed in 11 of the 52 patients treated with coiling (21.15%). Symptomatic vasospasm was observed in 20 patients (38.46%) (Table 1).

Ischemia due to vasospasm and symptomatic vasospasm were observed in 16 and 31 of the 92 patients included in the study, respectively. Although the incidence rates of ischemia (21.15%) and symptomatic vasospasm (38.46%) were higher in the coil group, no statistically significant difference was found between the two groups (P = 0.278 and P = 0.270, respectively; Table 2).

4. Discussion

In patients presenting with aneurysmal SAH, complications due to vasospasm are still among the most important causes of mortality and morbidity even when the aneurysm is successfully treated with coiling and clipping [18]. The amount of cisternal blood on CT within 3 days after SAH is the primary factor that has been associated with vasospasm development [3,14]. However, there is no conclusive evidence regarding other factors suggested to contribute to vasospasm and ischemia risks. One such debated factor is the approach to treatment. In the present study, we found no differences between the coiling and clipping procedures in terms of vasospasm or vasospasm-related ischemic complications.

Prior studies comparing treatment methods have revealed conflicting findings. While the clearance of blood clots in the cisterns has been associated with a decrease in the possibility of developing vasospasm in some studies [8,19], other researchers have suggested that the retraction of the brain and manipulation of vessels during early microsurgery increases the likelihood of vasospasm [11]. A multicenter randomized clinical trial (the International Subarachnoid Aneurysm Trial, ISAT) that compared clipping and coiling revealed better outcome (defined as death or dependency) in coiled patients at 1-year follow-up, which caused an increase in the proportion of patients who undergo endovascular coiling. However, unfortunately, the study did not publish any results regarding the frequency of vasospasm or related ischemic complications [20].

According to Kanamaru et al., vasospasm-induced cerebral infarction occurred more frequently after clipping than after coiling. Although age, sex, World Federation of Neurosurgical Societies (WFNS) scale scores, and Fisher grades were similar in the two groups, aneurysm locations were distributed heterogeneously and coiling was used more frequently in patients with posterior circulation aneurysms [21]. Rabinstein et al., in their study including 415 patients with aneurysmal SAH, had applied clipping in 339 patients, while coiling was used in 76 patients. The patients treated with endovascular coil had worse admission clinical grades at presentation, whereas recipients of clipping had a higher frequency of delayed aneurysm treatment (≥4 days after onset of SAH). Nonetheless, the authors concluded that, in patients with WFNS scores of I to III at presentation, the coiling approach carried lower risks for symptomatic vasospasm and permanent deficits caused by vasospasm when compared to clipping [12]. However, in both studies, the presence of patients who had received delayed treatment eliminated the possible advantages gained by early clearance of blood clots, which may be particularly important for patients with symptomatic vasospasm and vasospasm-related ischemia. In our study, both groups included only patients who had received treatment within 36 h after SAH.

Zaidat et al. reported that coiling resulted in lower incidence rates for angiographic and clinical vasospasms [22]. They used transcranial Doppler (TCD) to diagnose vasospasms. Patients with no radiological or clinical evidence of vasospasm were maintained in normovolemic and normotensive states. However, it must be noted that increased TCD velocities may not indicate vasospasm in presence of impaired cerebral autoregulation, a situation in which the increase in circulatory volume and hypertension can lead to misclassification due to alterations in cerebral blood flow. Considering that conventional angiography is established as the gold standard approach to vasospasm diagnosis [23], the results of studies utilizing other methods to diagnose cerebral vasospasm should be approached with caution, and direct comparisons should be avoided.

Dumont et al. found that clipping was independently associated with angiographic vasospasm and delayed ischemic neurological deficit; however, their results demonstrated that a greater frequency of patients in the coil group were treated with calcium channel antagonists [24], indicating a baseline bias that could influence results. In another study, by Gruber et al., the overall findings favored the use of clipping versus coiling, and ischemia was associated with various factors including higher Fisher and Hunt-Hess grades. But when patients determined as Fisher grade 4 and Hunt-Hess V were excluded from both groups, the statistical significance in the incidence of infarction between the two groups was found to have disappeared [25].

There are a number of factors that could explain the high degree of conflict in the literature: the heterogeneity of patient characteristics, the differences in treatment decisions and methods before or after SAH, the variations among studies in terms of aneurysm sites, the fact that microsurgical clipping techniques are yet to be fully and clearly defined, and the use of different criteria or methodology to diagnose vasospasm. Notwithstanding the fact that these factors may have also influenced our results and comparisons with prior studies, it appears that our results are in agreement with the majority of comparable literature on this topic –which have often demonstrated no significant difference between these two methods with respect to the incidence of symptomatic vasospasm and infarction [26–29].

In the present study, the groups were similar in terms of demographic characteristics. Only ruptured AcoA aneurysms were analyzed (Fisher grade 3) to be able to ensure consistency in comparisons due to the fact that the incidence of vasospasm and ischemia vary with respect to the site of aneurysm [30]. Also, vascular manipulation and brain retraction are performed at a greater frequency during the clipping procedures for AcoA aneurysms [31], and therefore, analysis of this subgroup of patients provides grounds for a reliable comparison of the two long-running hypotheses for cerebral vasospasm after SAH treatment (manipulation vs. clot removal). One particularly important point to note is that, in this study, the management of SAH, pre- and post-operative care, and the conduct of the clipping and coiling procedures were undertaken by dual-trained neurosurgeons. Balanced experience in both modalities has several evident advantages when evaluating endovascular and surgical series from different institutions, including the restriction of variances attributable to pre- and post-operative care, patient population, outcome assessment, and capabilities of the facilities. Despite these strengths, our study also has some notable limitations. Firstly, the patients' smoking status, which is known to be associated with vasospasm, were not known because this characteristic was not consistently recorded in medical records. Secondly, data regarding the overall frequency of post-treatment vasospasm cannot be drawn from this study, since angiography was not performed in patients without symptoms. Finally, the lack of statistical significance in the comparisons may have been associated with the limited number of patients investigated.

5. Conclusions

In this study, the incidence rate of cerebral vasospasm-related ischemia showed no significant difference between the clipping and coiling treatment groups which were comprised of patients admitted with good clinical condition and were found to have Fisher grade 3 lesions. Larger patient populations and prospective randomized controlled studies are needed for more-precise results. Additionally, researchers must be aware that it is exceedingly likely that there will be various factors that could cause significant variance between compared groups, as demonstrated by the conflicting literature on this topic, and thus, results should be meticulously assessed for possible biases.

CRediT authorship contribution statement

Eyüp Baykara: Conceptualization, Methodology, Software, Visualization, Investigation, Supervision, Writing – review & editing. **Abdullah Topçu:** Data curation, Writing – original draft, Software, Validation.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

References

[1] T.J. Leipzig, K. Redelman, T.G. Horner, Reducing the risk of rebleeding before early aneurysm surgery: a possible role for antifibrinolytic therapy, J. Neurosurg. 86 (2) (1997) 220–225, https://doi.org/10.3171/jns.1997.86.2.0220.

[2] N.F. Kassell, T. Sasaki, A.R. Colohan, G. Nazar, Cerebral vasospasm following aneurysmal subarachnoid hemorrhage, Stroke 16 (4) (1985) 562–572, https://doi.org/10.1161/01.str.16.4.562.

[3] C.M. Fisher, J.P. Kistler, J.M. Davis, Relation of cerebral vasospasm to subarachnoid hemorrhage visualized by computerized tomographic scanning, Neurosurgery 6 (1) (1980) 1–9, https://doi.org/10.1227/00006123-198001000-00001.

[4] A. Pasqualin, L. Rosta, R. Da Pian, P. Cavazzani, R. Scienza, Role of computed tomography in the management of vasospasm after subarachnoid hemorrhage, Neurosurgery 15 (3) (1984) 344–353, https://doi.org/10.1227/00006123-198409000-00009.

[5] C.G. Harrod, B.R. Bendok, H.H. Batjer, Prediction of cerebral vasospasm in patients presenting with aneurysmal subarachnoid hemorrhage: a review, Neurosurgery 56 (4) (2005) 633-54; discussion 633-54. 10.1227/01.neu.0000156644.45384.92.

[6] T.M. Lasner, R.J. Weil, H.A. Riina, J.T. King Jr., E.L. Zager, E.C. Raps, E.S. Flamm, Cigarette smoking-induced increase in the risk of symptomatic vasospasm after aneurysmal subarachnoid hemorrhage, J. Neurosurg. 87 (3) (1997) 381–384, https://doi.org/10.3171/jns.1997.87.3.0381.

[7] J.M. Findlay, R.L. Macdonald, B.K. Weir, M.G. Grace, Surgical manipulation of primate cerebral arteries in established vasospasm, J. Neurosurg. 75 (3) (1991) 425–432, https://doi.org/10.3171/jns.1991.75.3.0425.

[8] Y. Handa, B.K. Weir, M. Nosko, R. Mosewich, T. Tsuji, M. Grace, The effect of timing of clot removal on chronic vasospasm in a primate model, J. Neurosurg. 67 (4) (1987) 558–564, https://doi.org/10.3171/jns.1987.67.4.0558.

[9] T. Inagawa, M. Yamamoto, K. Kamiya, Effect of clot removal on cerebral vasospasm, J. Neurosurg. 72 (2) (1990) 224–230, https://doi.org/10.3171/jns.1990.72.2.0224.

[10] M. Taneda, The significance of early operation in the management of ruptured intracranial aneurysms–an analysis of 251 cases hospitalized within 24 hours after subarachnoid haemorrhage, Acta Neurochir. (Wien.) 63 (1–4) (1982) 201–208, https://doi.org/10.1007/bf01728873.

[11] K. Hosoda, S. Fujita, T. Kawaguchi, Y. Shose, S. Hamano, M. Iwakura, Effect of clot removal and surgical manipulation on regional cerebral blood flow and delayed vasospasm in early aneurysm surgery for subarachnoid hemorrhage, Surg. Neurol. 51 (1) (1999) 81–88, https://doi.org/10.1016/s0090-3019(97)00508-9.

[12] A.A. Rabinstein, M.A. Pichelmann, J.A. Friedman, D.G. Piepgras, D.A. Nichols, J. I. McIver, L.G. Toussaint 3rd, R.L. McClelland, J.R. Fulgham, F.B. Meyer, J. L. Atkinson, E.F. Wijdicks, Symptomatic vasospasm and outcomes following aneurysmal subarachnoid hemorrhage: a comparison between surgical repair and endovascular coil occlusion, J. Neurosurg. 98 (2) (2003) 319–325, https://doi.org/10.3171/jns.2003.98.2.0319.

[13] J.J. Heit, R.L. Ball, N.A. Telischak, H.M. Do, R.L. Dodd, G.K. Steinberg, S.D. Chang, M. Wintermark, M.P. Marks, Patient outcomes and cerebral infarction after ruptured anterior communicating artery aneurysm treatment, AJNR Am. J. Neuroradiol. 38 (11) (2017) 2119–2125, https://doi.org/10.3174/ajnr.A5355.

[14] M. Mizukami, T. Takemae, T. Tazawa, T. Kawase, T. Matsuzaki, Value of computed tomography in the prediction of cerebral vasospasm after aneurysm rupture, Neurosurgery 7 (6) (1980) 583–586, https://doi.org/10.1227/00006123-198012000-00008.

[15] R. Meyer, S. Deem, N.D. Yanez, M. Souter, A. Lam, M.M. Treggiari, Current practices of triple-H prophylaxis and therapy in patients with subarachnoid hemorrhage, Neurocrit. Care 14 (1) (2011) 24–36, https://doi.org/10.1007/s12028-010-9437-z.

[16] T. Inagawa, K. Yahara, N. Ohbayashi, Risk factors associated with cerebral vasospasm following aneurysmal subarachnoid hemorrhage, Neurol. Med. Chir. (Tokyo) 54 (6) (2014) 465–473, https://doi.org/10.2176/nmc.oa.2013-0169.

[17] P. Vajkoczy, P. Horn, C. Bauhuf, E. Munch, U. Hubner, D. Ing, C. Thome, C. Poeckler-Schoeninger, H. Roth, P. Schmiedek, Effect of intra-arterial papaverine on regional cerebral blood flow in hemodynamically relevant cerebral vasospasm, Stroke 32 (2) (2001) 498–505, https://doi.org/10.1161/01.STR.32.2.498.

[18] K. Li, C.D. Barras, R.V. Chandra, H.K. Kok, J.T. Maingard, N.S. Carter, J.H. Russell, L. Lai, M. Brooks, H. Asadi, A review of the management of cerebral vasospasm after aneurysmal subarachnoid hemorrhage, World Neurosurg. 126 (2019) 513–527, https://doi.org/10.1016/j.wneu.2019.03.083.

[19] T. Tsuji, D.A. Cook, B.K. Weir, Y. Handa, Effect of clot removal on cerebrovascular contraction after subarachnoid hemorrhage in the monkey: pharmacological study, Heart Vessels 11 (2) (1996) 69–79, https://doi.org/10.1007/bf01744506.

[20] A.J. Molyneux, R.S. Kerr, L.M. Yu, M. Clarke, M. Sneade, J.A. Yarnold, P. Sandercock, International subarachnoid aneurysm trial (ISAT) of neurosurgical clipping versus endovascular coiling in 2143 patients with ruptured intracranial aneurysms: a randomised comparison of effects on survival, dependency, seizures, rebleeding, subgroups, and aneurysm occlusion, Lancet 366 (9488) (2005) 809–817, https://doi.org/10.1016/s0140-6736(05)67214-5.

[21] K. Kanamaru, H. Suzuki, W. Taki, Risk factors for vasospasm-induced cerebral infarct when both clipping and coiling are equally available, Acta Neurochir. Suppl. 120 (2015) 291–295, https://doi.org/10.1007/978-3-319-04981-6_49.

[22] O.O. Zaidat, C.C. Ionita, S.I. Hussain, M.J. Alexander, A.H. Friedman, C. Graffagnino, Impact of ruptured cerebral aneurysm coiling and clipping on the incidence of cerebral vasospasm and clinical outcome, J. Neuroimaging 19 (2) (2009) 144–149, https://doi.org/10.1111/j.1552-6569.2008.00285.x.

[23] L. Letourneau-Guillon, B. Farzin, T.E. Darsaut, M. Kotowski, F. Guilbert, M. Chagnon, A. Diouf, D. Roy, A. Weill, M. Lemus, C. Bard, M. Belair, D. Landry, L. Nico, A. Tellier, R. Jabre, C. Kauffmann, J. Raymond, Reliability of CT angiography in cerebral vasospasm: a systematic review of the literature and an inter- and intraobserver study, AJNR Am. J. Neuroradiol. 41 (4) (2020) 612–618, https://doi.org/10.3174/ajnr.A6462.

[24] A.S. Dumont, R.W. Crowley, S.J. Monteith, D. Ilodigwe, N.F. Kassell, S. Mayer, D. Ruefenacht, S. Weidauer, A. Pasqualin, R.L. Macdonald, Endovascular treatment or neurosurgical clipping of ruptured intracranial aneurysms: effect on angiographic vasospasm, delayed ischemic neurological deficit, cerebral infarction, and clinical outcome, Stroke 41 (11) (2010) 2519–2524, https://doi.org/10.1161/strokeaha.110.579383.

[25] A. Gruber, K. Ungersböck, A. Reinprecht, T. Czech, C. Gross, M. Bednar, B. Richling, Evaluation of cerebral vasospasm after early surgical and endovascular treatment of ruptured intracranial aneurysms, Neurosurgery 42(2) (1998) 258-67; discussion 267-8. 10.1097/00006123-199802000-00032.

[26] J.G. de Oliveira, J. Beck, C. Ulrich, J. Rathert, A. Raabe, V. Seifert, Comparison between clipping and coiling on the incidence of cerebral vasospasm after aneurysmal subarachnoid hemorrhage: a systematic review and meta-analysis, Neurosurg Rev 30(1) (2007) 22-30; discussion 30-1. doi: 10.1007/s10143-006-0045-5.

[27] A.J. Goddard, P.P. Raju, A. Gholkar, Does the method of treatment of acutely ruptured intracranial aneurysms influence the incidence and duration of cerebral vasospasm and clinical outcome? J. Neurol. Neurosurg. Psychiatry 75 (6) (2004) 868–872, https://doi.org/10.1136/jnnp.2003.033068.

[28] A.R. Dehdashti, B. Mermillod, D.A. Rufenacht, A. Reverdin, N. de Tribolet, Does treatment modality of intracranial ruptured aneurysms influence the incidence of cerebral vasospasm and clinical outcome? Cerebrovasc. Dis. 17 (1) (2004) 53–60, https://doi.org/10.1159/000073898.

[29] B.L. Hoh, M.A. Topcuoglu, A.B. Singhal, J.C. Pryor, J.D. Rabinov, G.A. Rordorf, B. S. Carter, C.S. Ogilvy, Effect of clipping, craniotomy, or intravascular coiling on cerebral vasospasm and patient outcome after aneurysmal subarachnoid hemorrhage, Neurosurgery 55(4) (2004) 779-86; discussion 786-9. 10.1227/01.neu.0000137628.51839.d5.

[30] A.A. Abla, D.A. Wilson, R.W. Williamson, P. Nakaji, C.G. McDougall, J. M. Zabramski, F.C. Albuquerque, R.F. Spetzler, The relationship between ruptured aneurysm location, subarachnoid hemorrhage clot thickness, and incidence of radiographic or symptomatic vasospasm in patients enrolled in a prospective randomized controlled trial, J. Neurosurg. 120 (2) (2014) 391–397, https://doi.org/10.3171/2013.10.Jns13419.

[31] L.H. Yu, P.S. Yao, S.F. Zheng, D.Z. Kang, Retractorless surgery for anterior circulation aneurysms via a pterional keyhole approach, World Neurosurg. 84 (6) (2015) 1779–1784, https://doi.org/10.1016/j.wneu.2015.07.055.