Neurochirurgie 58 (2012) 146–150

Disponible en ligne sur
SciVerse ScienceDirect
www.sciencedirect.com

Elsevier Masson France
EM|consulte
www.em-consulte.com

Report 2012: Intracranial aneurysms: clips or coils

Strategical implications of aneurysmal cranial nerve compression

F. Scholtes*, D. Martin

Service de neurochirurgie, université de Liège, CHU de Liège, Sart Tilman B35, 4000 Liège, Belgium

ARTICLE INFO

Article history:
Received 27 February 2012
Accepted 27 February 2012

Keyword:
Intracranial aneuvrysm

ABSTRACT

Intracranial aneurysms may manifest clinically by inducing neurological symptoms, including cranial nerve dysfunction. In unruptured aneurysms, this may result from mass effect and the pulsation of the sac. Aneurysm rupture and sudden expansion of a pseudo-sac may precipitate the appearance of cranial nerve deficits. Symptomatic aneurysms should be treated. Surgery reduces mass effect and arterial pulsations, and removes clot after rupture. Endovascular treatment decreases pulsatility of the sac. Recovery has been reported after both treatments. It appears more reproducible after surgery, but the data of current literature remains weak. The possible advantage of surgery is an argument among others that must be considered in the choice of the most adequate therapeutic approach.

1. Introduction

Ruptured as well as unruptured aneurysms may induce neurological manifestations due to their direct effect on adjacent cerebral parenchyma, including epilepsy, or cranial nerves (Friedman et al., 2001).

Oculomotor deficits are the most common cranial nerve dysfunction, especially those due to third nerve compression by aneurysms at the carotid origin of the posterior communicating artery (Barnes et al., 1981). Oculomotor symptoms can also be induced by intracavernous, especially when they are large, or posterior circulation aneurysms (Jefferson, 1947; Ajtai et al., 2004; Nistri et al., 2007; Huang et al., 1996; Kim et al., 2003).

Visual deficits are the second most common aneurysm-induced cranial nerve dysfunction, due mostly to conflict between the anterior visual pathways and anterior circulation aneurysms, usually paraophthalmic, clinoidal or of the anterior cerebral artery.

Hemifacial spasm and other cranial nerve dysfunctions are rare.

De novo cranial nerve deficits or functional aggravations, including visual loss, have been described after both surgical (Almer and Miller, 2008) and endovascular (Kim et al., 2003; Nishino et al., 2009; Turner et al., 2008; Lee et al., 2011) treatment.

This manuscript concerns those symptomatic aneurysms that require a choice between open surgical and endovascular treatment in usual clinical settings, excluding intracranial arterial dolichoectasia, arterial dissections, fusiform aneurysms and intracavernous aneurysms of the internal carotid artery.

2. Oculomotor palsy

There is a large body of literature concerning aneurysmal oculomotor palsy. In 1996, one review assembled several hundred surgically treated cases (Leivo et al., 1996). But, although third cranial nerve palsy due to the presence of an aneurysm at the origin of the posterior communicating artery is the most common aneurysmal cranial nerve dysfunction (Fig. 1), the literature is purely retrospective and heterogeneous. In many publications, various aneurysms and deficits are grouped (Guresir et al., 2011). Systematic analysis of these data is further complicated by methodological differences (including surgical technique, such as aneurysm sac resection with IIIrd nerve manipulation; definitions of complete or partial deficits or recovery; exceptional use of standardised recovery scales. . ., (Chen et al., 2006; Zhang et al., 2010; Richling, 2010)).

In any case, the presence of a symptomatic aneurysm presenting with oculomotor palsy, even unruptured, is associated with an increased risk of haemorrhage.

2.1. Treatment indications

Ruptured aneurysms, as proven by CT or lumbar puncture, must be treated.

In unruptured aneurysms, the occurrence of oculomotor palsy may be the manifestation of an enlargement of the sac. It is often associated with retrobulbar and upper trigeminal pain that may precede the palsy, resulting from irritation of trigeminal fibres in the oculomotor nerve (Lanzino et al., 1993). This thus makes an unruptured aneurysm symptomatic, and risk of rupture is increased (Wermer et al., 2007; Komotar et al., 2008). Unruptured aneurysms of the posterior communicating artery, as those of the posterior circulation, are at an increased risk of rupture (Wermer et al., 2007;

* Corresponding author.
E-mail addresses: felix.scholtes@chu.ulg.ac.be (F. Scholtes), didier.martin@chu.ulg.ac.be (D. Martin).

0028-3770/$ – see front matter © 2012 Elsevier Masson SAS. All rights reserved.
doi:10.1016/j.neuchi.2012.02.019

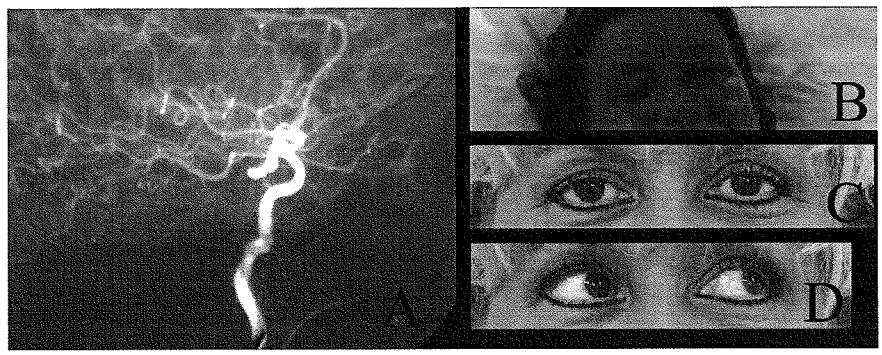

Fig. 1. Ruptured posterior communicating artery aneurysm. The patient had presented with sudden headache and ptosis. A. Angiography demonstrating the presence of the posterior communicating artery aneurysm. B. Preoperative photograph of the patient. Ptosis. Ophthalmological examination revealed complete oculomotor paralysis. C et D. Post-operative follow up showing recovery of ptosis (C) and oculomotor function (D).

Komotar et al., 2008; International Study of Unrupted Intracranial Aneurysms Investigators, 1998). Thus, when symptomatic they should all be treated.

2.2. Oculomotor recovery

Oculomotor palsy may recover without any treatment (Jefferson, 1947; Guresir et al., 2011; Foroozan et al., 2002), after surgical clipping of the aneurysm (Leivo et al., 1996; Guresir et al., 2011) (Fig. 1) and after endovascular treatment (Zhang et al., 2010; Birchall et al., 1999; Panagiotopoulos et al., 2011; Kassis et al., 2010). Most recoveries occur soon after treatment (Leivo et al., 1996), but recovery has been described after several months, up to a year and a half (Kassis et al., 2010). Patients should be informed of this.

Although it is not possible to modify factors that influence recovery and are related to the patient or the aneurysm itself, such as age or diabetes mellitus (Ahn et al., 2006), aneurysm size (Yanaka et al., 2003), the severity of the palsy (Chen et al., 2006; Zhang et al., 2010; Panagiotopoulos et al., 2011; Kyriakides et al., 1989), or the presence of subarachnoid haemorrhage (Zhang et al., 2010; Kassis et al., 2010; Albuquerque, 2010), two factors are modifiable: treatment modality and delay.

2.2.1. Surgical versus endovascular treatment

The question of the advantage of one treatment modality over the other has been addressed by a series of recent publications. It is particularly interesting for posterior communicating artery aneurysms which are accessible to both open surgical and endovascular treatment. All published investigations are limited by significant confounding factors in patient selection. Still, four papers attempt to compare open surgical and endovascular treatment (Guresir et al., 2011; Chen et al., 2006; Ahn et al., 2006; Nam et al., 2010).

2.2.1.1. Direct comparison of treatment approaches. One literature review published in 2011 (Guresir et al., 2011) has assembled and analysed methodologically acceptable previous investigations published up to 2008, concerning oculomotor palsy due to posterior communicating artery aneurysms. Another 15 aneurysms were added from local experience. In this analysis of approximately 200 aneurysms, complete as well as "complete or partial" recovery appeared to occur significantly more often after open surgical than after endovascular treatment. When compared to untreated aneurysms, recovery from complete oculomotor deficits was significantly increased after open surgery but not after endovascular treatment.

The previously cited analysis includes a series of 12 patients published by Spetzler's group in 2006 (Chen et al., 2006). This paper described complete recovery in 85% of open surgical cases versus 33% of endovascular cases after six months, and partial recovery in 15% versus 67%. In those cases followed for one year or more, complete recovery was seen in all open surgical cases and half the endovascular cases.

Two other recent, comparative, methodologically less convincing series (Ahn et al., 2006; Nam et al., 2010) were not included in the 2011 review. They do not add any compelling data.

2.2.1.2. Recent endovascular series. Recent series describing outcome after endovascular treatment of posterior communicating artery aneurysms with third nerve palsy show recovery in many cases. Nevertheless, recovery remains less common than in many open surgical series (Zhang et al., 2010; Panagiotopoulos et al., 2011; Kassis et al., 2010). Recanalisations were common, often moderate, and were usually not associated with oculomotor palsy recurrence (Kassis et al., 2010).

2.2.2. Summary

Considering the limited decisional impact of this kind of epidemiological investigations (Raymond et al., 2011), one may conclude that:

- recovery from oculomotor palsy occurs in many cases, after both surgical and endovascular treatment. No treatment can guarantee complete recovery;
- the intuitive notion that surgical third nerve decompression may be more efficient than endovascular filling of the aneurysmal sac seems to be confirmed by the available data. On the basis of open surgical series and the observed high rates of recovery, this treatment is indicated when safe.

2.3. Delay to treatment

Classic literature data indicates the usefulness of early surgery (Feely and Kapoor, 1987). In 1996, Leivo et al. reviewed 283 cases of posterior communicating artery aneurysms for which the delay between occurrence of symptoms and treatment was known, and added 28 of their own cases. Though without precise juxtaposition of the severity of the preoperative deficit and the degree of recovery, the shortest times to treatment were still clearly correlated with complete recovery (Leivo et al., 1996). Despite the fact that a statistically significant correlation is not found in all studies (Chen et al., 2006; Kyriakides et al., 1989), the majority of publications describe higher proportions of complete recovery after early treatment (Zhang et al., 2010; Kassis et al., 2010; Yanaka et al., 2003;

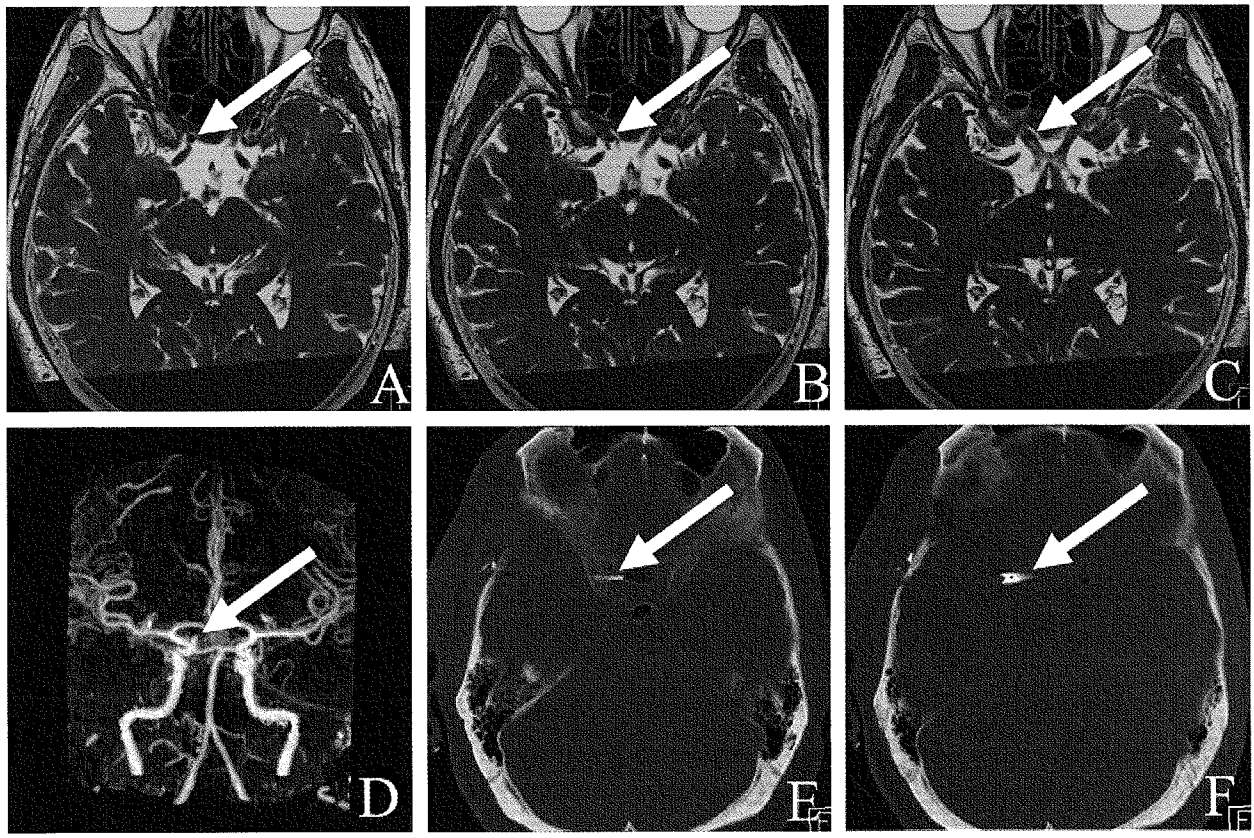

Fig. 2. Left carotido-ophthalmic aneurysm. The patient presented with headache and vague visual symptoms, without haemorrhage at CT or lumbar puncture. A–C. CT-T2 weighted MRI fusion, transverse plane. The arrow shows the conflict between the aneurysmal sac and the optic nerve. D. TOF MRI showing the aneurysm (arrow), directed upwards from the ophthalmic segment of the carotid artery. E et F. Postoperative transverse CT images showing the position of the clip (arrow) above the anterior clinoid process.

Feely and Kapoor, 1987; Perneczky and Czech, 1984; Giombini et al., 1991; Yang et al., 2008).

3. Visual deficits

The optic nerve and chiasm may be compressed (Schuss et al., 2011), or even penetrated and split (Beatty, 1986; Kanamaru et al., 2001; Fujita et al., 2002; Jea et al., 2003; Joo and Kim, 2007; Wang et al., 2010) by anterior circulation aneurysms, typically of the ophthalmic segment of the internal carotid artery (Fig. 2).

The resulting visual deficit must not be confused with others that may occur in the presence of an intracranial aneurysm, especially after haemorrhage. They may be due to vitreous haemorrhage, ischemic optic neuropathy (Kang et al., 1997; Hara et al., 2003), sometimes during vasospasm (Carney and Oatey, 1983). In addition, visual deficits may appear after paraclinoid aneurysm surgery even in the absence of manipulation of or damage to the optic nerve or its vascular supply (Rizzo, 1995).

3.1. Surgical versus endovascular treatment

Early treatment of aneurysm responsible for visual deficits is known to be efficacious for preventing definitive visual loss (Tawk et al., 2006; Sundt and Whisnant, 1978; Winn et al., 1977). In many cases, recovery occurs after surgical decompression (Heros et al., 1983; Ferguson and Drake, 1981; de Oliveira et al., 2009), as in third nerve compression, but also, to a certain degree, after endovascular treatment (Vargas et al., 1994).

The literature reporting open surgical and endovascular treatment of anterior circulation aneurysms responsible for visual deterioration up to 2010 has also been recently reviewed and completed by local experience (Schuss et al., 2011), in order to compare open surgical and endovascular efficacy on visual symptoms. Most of the analysed aneurysms were unruptured, since visual deficits due to other causes than aneurysmal nerve compression had to be formally excluded from this review. Again, all studies were retrospective with the associated limitations. Nevertheless, this analysis speaks clearly in favour of open surgical optic nerve decompression. Also, visual deterioration has been seen after endovascular treatment, probably of multifactorial origin with inflammatory and compressive components (Tawk et al., 2006).

This analysis excludes open surgical treatments other than clipping, like complex anastomoses (Kim et al., 2006). Also, certain aneurysms may have to be treated with a combination of surgery and endovascular approaches (Arnautovic et al., 1998). Finally, when clipping of an aneurysm is not feasible, wrapping of the aneurysm may be performed. When muslin is used, this may lead to visual loss (Carney and Oatey, 1983; Gruber et al., 1983; Repka et al., 1984; McFadzean et al., 1991; Felsberg et al., 1993; Berger et al., 2003).

3.2. Summary

Symptomatic aneurysmal optic nerve compression indicates prompt treatment. Choice of open surgical or endovascular treatment is often dictated by various factors independent of the visual prognosis. Nevertheless, based on current clinical understanding

and the literature, open surgical aneurysm treatment and optic nerve decompression should be offered when feasible and as safe as endovascular treatment.

4. Facial hemispasm and trigeminal neuralgia

Facial hemispasm and palsy, and even trigeminal neuralgia, may be seen in the presence of aneurysms, particularly fusiform (Nakagawa et al., 2011; Choi et al., 2008; Uchino et al., 2005; Sato et al., 2001), but also sacciform aneurysms of the vertebro-basilar circulation, though the latter are uncommon (Moriuchi et al., 1996; Maroon et al., 1978; Neimat et al., 2005; Trotter and Choksey, 2000; Terao et al., 2001). Treatment should be tailored to each case according to clinical judgement.

5. Conclusion

Symptomatic aneurysms require treatment. A cranial nerve deficit can influence the choice of one therapeutic modality over another. It is one argument among many, including aneurysm shape and location, its relation to the parent and surrounding vessels, which all influence initial degree and long term stability of endovascular occlusion (Songsaeng et al., 2011, 2010).

The deficit is due to aneurysm mass effect on the nerve, transmission of arterial pulsations, change in shape in case of enlargement of the sac, and, in case of rupture, presence of the clot. Open surgery can address all these aspects. Endovascular treatment decreases pulsatility.

Oculomotor palsy due to a posterior communicating artery aneurysm may influence the choice of open surgical over endovascular treatment if other factors are equivocal (Golshani et al., 2010). If open surgery can be offered safely, it should be.

Optic nerve compression by an aneurysm should be treated promptly, if possible by open surgery. If this carries unacceptable risk, endovascular occlusion might also result in visual stabilisation or recovery.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

References

Ahn, J.Y., Han, I.B., Yoon, P.H., et al., 2006. Clipping vs coiling of posterior communicating artery aneurysms with third nerve palsy. Neurology 66, 121–123.

Ajtai, B., Fine, E.J., Lincoff, N., 2004. Pupil-sparing, painless compression of the oculomotor nerve by expanding basilar artery aneurysm: a case of ocular pseudomyasthenia. Arch Neurol 61, 1448.

Albuquerque, F.C., 2010. Coiling versus clipping for posterior communicating artery aneurysms associated with oculomotor nerve palsy: only time will tell. World Neurosurg 74, 250–251.

Almer, Z.E., Miller, N.R., 2008. Delayed third cranial nerve palsy after aneurysm wrapping. J Neuroophthalmol 28, 359.

Arnautovic, K.I., Al-Mefty, O., Angtuaco, E., 1998. A combined microsurgical skull-base and endovascular approach to giant and large paraclinoid aneurysms. Surg Neurol 50, 504–518 [discussion 518–20].

Barnes, M.P., Hunt, B.J., Williams, I.R., 1981. The role of vertebral angiography in the investigation of third nerve palsy. J Neurol Neurosurg Psychiatry 44, 1153.

Beatty, R.A., 1986. Splitting of the optic nerve by a carotid-ophthalmic artery aneurysm. Case report. J Neurosurg 65, 560–562.

Berger, C., Hartmann, M., Wildemann, B., 2003. Progressive visual loss due to a muslinoma–report of a case and review of the literature. Eur J Neurol 10, 153–158.

Birchall, D., Khangure, M.S., McAuliffe, W., 1999. Resolution of third nerve paresis after endovascular management of aneurysms of the posterior communicating artery. AJNR Am J Neuroradiol 20, 411–413.

Carney, P.G., Oatey, P.E., 1983. Muslin wrapping of aneurysms and delayed visual failure. A report of three cases. J Clin Neuroophthalmol 3, 91–96.

Chen, P.R., Amin-Hanjani, S., Albuquerque, F.C., McDougall, C., Zabramski, J.M., Spetzler, R.F., 2006. Outcome of oculomotor nerve palsy from posterior communicating artery aneurysms: comparison of clipping and coiling. Neurosurgery 58, 1040–1046 [discussion 1040–6].

Choi, S.K., Rhee, B.A., Park, B.J., Lim, Y.J., 2008. Hemifacial spasm caused by fusiform aneurysm at vertebral artery-posterior inferior cerebellar artery junction. J Korean Neurosurg Soc 44, 399–400.

de Oliveira, J.G., Borba, L.A., Rassi-Neto, A., et al., 2009. Intracranial aneurysms presenting with mass effect over the anterior optic pathways: neurosurgical management and outcomes. Neurosurg Focus 26, E3.

Feely, M., Kapoor, S., 1987. Third nerve palsy due to posterior communicating artery aneurysm: the importance of early surgery. J Neurol Neurosurg Psychiatry 50, 1051–1052.

Felsberg, G.J., Tien, R.D., Haplea, S., Osumi, A.K., 1993. Muslin-induced optic arachnoiditis ("gauzoma"): findings on CT and MR. J Comput Assist Tomogr 17, 485–487.

Ferguson, G.G., Drake, C.G., 1981. Carotid-ophthalmic aneurysms: visual abnormalities in 32 patients and the results of treatment. Surg Neurol 16, 1–8.

Foroozan, R., Slamovits, T.L., Ksiazek, S.M., Zak, R., 2002. Spontaneous resolution of aneurysmal third nerve palsy. J Neuroophthalmol 22, 211–214.

Friedman, J.A., Piepgras, D.G., Pichelmann, M.A., Hansen, K.K., Brown, R.D.J., Wiebers, D.O., 2001. Small cerebral aneurysms presenting with symptoms other than rupture. Neurology 57, 1212–1216.

Fujita, A., Tamaki, N., Yasuo, K., Nagashima, T., Ehara, K., 2002. Complete penetration of the optic chiasm by an unruptured aneurysm of the ophthalmic segment: case report. Surg Neurol 57, 130–134.

Giombini, S., Ferraresi, S., Pluchino, F., 1991. Reversal of oculomotor disorders after intracranial aneurysm surgery. Acta Neurochir (Wien) 112, 19–24.

Golshani, K., Ferrell, A., Zomorodi, A., Smith, T.P., Britz, G.W., 2010. A review of the management of posterior communicating artery aneurysms in the modern era. Surg Neurol Int 1, 88.

Gruber, D., Brasseur, G., Charlin, J.F., Creissard, P., Langlois, J., 1983. Optochiasmatic compression by a giant intracranial aneurysm. Treatment and favorable visual results. Rev Otoneuroophtalmol 55, 289–295.

Guresir, E., Schuss, P., Setzer, M., Platz, J., Seifert, V., Vatter, H., 2011. Posterior communicating artery aneurysm-related oculomotor nerve palsy: influence of surgical and endovascular treatment on recovery: single-center series and systematic review. Neurosurgery 68, 1527–1533, discussion 1533–4.

Hara, N., Mukuno, K., Ohtaka, H., Shimizu, K., 2003. Ischemic optic neuropathy associated with subarachnoid hemorrhage after rupture of anterior communicating artery aneurysm. Ophthalmologica 217, 79–84.

Heros, R.C., Nelson, P.B., Ojemann, R.G., Crowell, R.M., DeBrun, G., 1983. Large and giant paraclinoid aneurysms: surgical techniques, complications, and results. Neurosurgery 12, 153–163.

Huang, L.T., Shih, T.Y., Lui, C.C., 1996. Posterior cerebral artery aneurysm in a two-year-old girl. J Formos Med Assoc 95, 170–172.

International Study of Unruptured Intracranial Aneurysms Investigators. Unruptured intracranial aneurysms–risk of rupture and risks of surgical intervention. N Engl J Med 1998; 339:1725–33.

Jea, A., Baskaya, M.K., Morcos, J.J., 2003. Penetration of the optic nerve by an internal carotid artery-ophthalmic artery aneurysm: case report and literature review. Neurosurgery 53, 996–999 [discussion 999–1000].

Jefferson, G., 1947. Isolated oculomotor palsy caused by intracranial aneurysm. Proc R Soc Med 40, 419–432.

Joo, S.P., Kim, T.S., 2007. Splitting of the optic nerve associated with ruptured anterior communicating artery aneurysm. Neurol Med Chir (Tokyo) 47, 501–502.

Kanamaru, K., Ishida, F., Taki, W., 2001. Splitting and penetration of the optic nerve by an aneurysm arising from the anterior wall of internal carotid artery: case report. J Neurol Neurosurg Psychiatry 71, 525–527.

Kang, S., Yang, Y., Kim, T., Kim, J., 1997. Sudden unilateral blindness after intracranial aneurysm surgery. Acta Neurochir (Wien) 139, 221–226.

Kassis, S.Z., Jouanneau, E., Tahon, F.B., Salkine, F., Perrin, G., Turjman, F., 2010. Recovery of third nerve palsy after endovascular treatment of posterior communicating artery aneurysms. World Neurosurg 73, 11–16 [discussion e2].

Kim, D.J., Kim, D.I., Lee, S.K., Kim, S.Y., 2003. Unruptured aneurysms with cranial nerve symptoms: efficacy of endosaccular Guglielmi detachable coil treatment. Korean J Radiol 4, 141–145.

Kim, K., Mizunari, T., Mizutani, N., et al., 2006. Giant intracranial aneurysm of the anterior communicating artery treated by direct surgery using A3-A3 side-to-side anastomosis and A3-RA graft-STA anastomosis. Acta Neurochir (Wien) 148, 353–357 [discussion 357].

Komotar, R.J., Mocco, J., Solomon, R.A., 2008. Guidelines for the surgical treatment of unruptured intracranial aneurysms: the first annual J. Lawrence pool memorial research symposium–controversies in the management of cerebral aneurysms. Neurosurgery 62, 183–193 [discussion 193–4].

Kyriakides, T., Aziz, T.Z., Torrens, M.J., 1989. Postoperative recovery of third nerve palsy due to posterior communicating aneurysms. Br J Neurosurg 3, 109–111.

Lanzino, G., Andreoli, A., Tognetti, F., et al., 1993. Orbital pain and unruptured carotid-posterior communicating artery aneurysms: the role of sensory fibers of the third cranial nerve. Acta Neurochir (Wien) 120, 7–11.

Lee, S.H., Koh, J.S., Lee, C.Y., Kwon, G.Y., Ryu, C.W., 2011. Penetration and splitting of the optic apparatus by intrasaccular coils within an anterior communicating artery aneurysm. Clin Neurol Neurosurg 113, 578–581.

Leivo, S., Hernesniemi, J., Luukkonen, M., Vapalahti, M., 1996. Early surgery improves the cure of aneurysm-induced oculomotor palsy. Surg Neurol 45, 430–434.

Maroon, J.C., Lunsford, L.D., Deeb, Z.L., 1978. Hemifacial spasm due to aneurysmal compression of the facial nerve. Arch Neurol 35, 545–546.

McFadzean, R.M., Hadley, D.M., McIlwaine, G.G., 1991. Optochiasmal arachnoiditis following muslin wrapping of ruptured anterior communicating artery aneurysms. J Neurosurg 75, 393–396.

Moriuchi, S., Nakagawa, H., Yamada, M., Kadota, T., 1996. Hemifacial spasm due to compression of the facial nerve by vertebral artery-posterior inferior cerebellar artery aneurysm and elongated vertebral artery–case report. Neurol Med Chir (Tokyo) 36, 884–887.

Nakagawa, I., Takayama, K., Kurokawa, S., et al., 2011. Hemifacial spasm due to contralateral aneurysmal compression of the facial nerve successfully treated with endovascular coil embolization: case report. Neurosurgery 69, E768–E771 [discussion E771–2].

Nam, K.H., Choi, C.H., Lee, J.I., Ko, J.G., Lee, T.H., Lee, S.W., 2010. Unruptured intracranial aneurysms with oculomotor nerve palsy: clinical outcome between surgical clipping and coil embolization. J Korean Neurosurg Soc 48, 109–114.

Neimat, J.S., Hoh, B.L., McKenna, M.J., Rabinov, J.D., Ogilvy, C.S., 2005. Aneurysmal expansion presenting as facial weakness: case report and review of the literature. Neurosurgery 56, E202–E205.

Nishino, K., Ito, Y., Hasegawa, H., Shimbo, J., Kikuchi, B., Fujii, Y., 2009. Development of cranial nerve palsy shortly after endosaccular embolization for asymptomatic cerebral aneurysm: report of two cases and literature review. Acta Neurochir (Wien) 151, 379–383.

Nistri, M., Perrini, P., Di Lorenzo, N., Cellerini, M., Villari, N., Mascalchi, M., 2007. Third-nerve palsy heralding dissecting aneurysm of posterior cerebral artery: digital subtraction angiography and magnetic resonance appearance. J Neurol Neurosurg Psychiatry 78, 197–198.

Panagiotopoulos, V., Ladd, S.C., Gizewski, E., et al., 2011. Recovery of ophthalmoplegia after endovascular treatment of intracranial aneurysms. AJNR Am J Neuroradiol 32, 276–282.

Perneczky, A., Czech, T., 1984. Prognosis of oculomotor palsy following subarachnoid hemorrhage due to aneurysms of the posterior communicating artery. Zentralbl Neurochir 45, 189–195.

Raymond, J., Darsaut, T.E., Kotowski, M., Bojanowski, M.W., 2011. Unruptured intracranial aneurysms: why clinicians should not resort to epidemiologic studies to justify interventions. AJNR Am J Neuroradiol 32, 1568–1569.

Repka, M.X., Miller, N.R., Penix, J.O., Trant III, J.H., 1984. Optic neuropathy from the use of intracranial muslin. J Neuro Ophthalmol 4, 147.

Richling, B., 2010. Endovascular therapy for symptomatic aneurysms. World Neurosurg 74, 252–253.

Rizzo, JFr., 1995. Visual loss after neurosurgical repair of paraclinoid aneurysms. Ophthalmology 102, 905–910.

Sato, K., Ezura, M., Takahashi, A., Yoshimoto, T., 2001. Fusiform aneurysm of the vertebral artery presenting hemifacial spasm treated by intravascular embolization: case report. Surg Neurol 56, 52–55.

Schuss, P., Guresir, E., Berkefeld, J., Seifert, V., Vatter, H., 2011. Influence of surgical or endovascular treatment on visual symptoms caused by intracranial aneurysms: single-center series and systematic review. J Neurosurg 115, 694–699.

Songsaeng, D., Geibprasert, S., Willinsky, R., Tymianski, M., TerBrugge, K.G., Krings, T., 2010. Impact of anatomical variations of the circle of Willis on the incidence of aneurysms and their recurrence rate following endovascular treatment. Clin Radiol 65, 895–901.

Songsaeng, D., Geibprasert, S., ter Brugge, K.G., Willinsky, R., Tymianski, M., Krings, T., 2011. Impact of individual intracranial arterial aneurysm morphology on initial obliteration and recurrence rates of endovascular treatments: a multivariate analysis. J Neurosurg 114, 994–1002.

Sundt, T.M.J., Whisnant, J.P., 1978. Subarachnoid hemorrhage from intracranial aneurysms. Surgical management and natural history of disease. N Engl J Med 299, 116–122.

Tawk, R.G., Villalobos, H.J., Levy, E.I., Hopkins, L.N., 2006. Surgical decompression and coil removal for the recovery of vision after coiling and proximal occlusion of a clinoidal segment aneurysm: technical case report. Neurosurgery 58, E1217 [discussion E1217].

Terao, S., Hara, K., Yoshida, K., Ohira, T., Kawase, T., 2001. A giant internal carotid-posterior communicating artery aneurysm presenting with atypical trigeminal neuralgia and facial nerve palsy in a patient with autosomal dominant polycystic kidney disease: a case report. Surg Neurol 56, 127–131.

Trotter, M.I., Choksey, M.S., 2000. Facial pain with intracranial aneurysm. J R Soc Med 93, 479–480.

Turner, R.D., Byrne, J.V., Kelly, M.E., et al., 2008. Delayed visual deficits and monocular blindness after endovascular treatment of large and giant paraophthalmic aneurysms. Neurosurgery 63, 469–474 [discussion 474–5].

Uchino, M., Nomoto, J., Ohtsuka, T., Kuramitsu, T., 2005. Fusiform aneurysm of the vertebral artery presenting with hemifacial spasm treated by microvascular decompression. Acta Neurochir (Wien) 147, 901–903.

Vargas, M.E., Kupersmith, M.J., Setton, A., Nelson, K., Berenstein, A., 1994. Endovascular treatment of giant aneurysms which cause visual loss. Ophthalmology 101, 1091–1098.

Wang, Y.Y., Thani, N.B., Han, T.F., 2010. Optic nerve penetration by a carotico-ophthalmic artery aneurysm. J Clin Neurosci 17, 931–933.

Wermer, M.J., van der Schaaf, I.C., Algra, A., Rinkel, G.J., 2007. Risk of rupture of unruptured intracranial aneurysms in relation to patient and aneurysm characteristics: an updated meta-analysis. Stroke 38, 1404–1410.

Winn, H.R., Richardson, A.E., Jane, J.A., 1977. The long-term prognosis in untreated cerebral aneurysms: I. The incidence of late hemorrhage in cerebral aneurysm: a 10-year evaluation of 364 patients. Ann Neurol 1, 358–370.

Yanaka, K., Matsumaru, Y., Mashiko, R., Hyodo, A., Sugimoto, K., Nose, T., 2003. Small unruptured cerebral aneurysms presenting with oculomotor nerve palsy. Neurosurgery 52, 553–557 [discussion 556–7].

Yang, M.Q., Wang, S., Zhao, Y.L., Zhang, D., Zhao, J.Z., 2008. Postoperative recovery from posterior communicating aneurysm complicated by oculomotor palsy. Chin Med J (Engl) 121, 1065–1067.

Zhang, S.H., Pei, W., Cai, X.S., Cheng, G., 2010. Endovascular management and recovery from oculomotor nerve palsy associated with aneurysms of the posterior communicating artery. World Neurosurg 74, 316–319.