

# Incidence and risk factors of postoperative headache after endovascular coil embolization of unruptured intracranial aneurysms

Kyu-Sun Choi · Jung-Hyun Lee · Hyeong-Joong Yi ·  
Hyoung-Joon Chun · Young-Jun Lee · Dong-Won Kim

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## Abstract

**Background** Endovascular coil embolization for unruptured intracranial aneurysms (UIAs) has gained popularity because of its low morbidity and mortality in a short-term context. However, Headache is sometimes brought about or worsened after endovascular treatment, and this complaint may lead to perplexing situations, albeit infrequently. The aim of this study is to estimate the practical incidence and risk factors of postoperative headache in patients with endovascular embolization of UIAs.

**Method** One hundred and thirty patients who underwent endovascular treatment of UIAs between March 2006 and May 2012 were enrolled according to inclusion criteria. From a retrospective chart review, the patients who had worsening or newly developed headache from postoperative day 1 to in-hospital stay were investigated for analyzing risk factors of post-embolization headache. Factors based on patients' demographics, anatomical and radiological features of the lesions, treatment, utilized devices and outcome were investigated, and statistically verified.

**Results** Headache occurred or was exacerbated in 32 patients (24.6 %). Of these, 30 patients showed improvement within days, but two patients with previous migraine history complained of intermittent headache over 3 months after the embolization. Univariate comparison between the headache

group and the non-headache group showed that internal carotid artery (ICA) segment aneurysm, stent-assisted coiling, and no history of hypertension were associated with post-embolization headache ( $p < 0.05$ ). However, stent-assisted coiling and no history of hypertension were significantly associated with post-embolization headache in logistic regression analysis ( $p < 0.05$ ).

**Conclusions** In the current study, stent-assisted coiling and no history of hypertension were important risk factors for headache in patients undergoing endovascular coil embolization for UIAs. Further investigations are still necessary to confirm the correlation of other factors which did not reach statistical significance in post-embolization headache in this limited study.

**Keywords** Embolization · Endovascular coiling · Intracranial aneurysm · Headache

## Introduction

Intracranial aneurysms are relatively common vascular lesions, and present in approximately 3–6 % of the population over the age of 30 years [27]. Recently, the incidence of such aneurysms have been further increasing, mainly because of emerging new technology and changing life patterns with resultant early detection and recognition of dormant lesions. In most cases, these remain asymptomatic and are only diagnosed through imaging studies or after spontaneous rupture. Headache is a frequent symptom in patients with intracranial aneurysms, often leading to the detection of the aneurysm whether ruptured or not [20, 26]. Following treatment of intracranial aneurysms, there may be changing degrees in headache, with most patients improving or unchanged, while others having onset of new headaches or worsening of existing headaches. Several retrospective studies analyzed

K.-S. Choi · J.-H. Lee · H.-J. Yi (✉) · H.-J. Chun  
Department of Neurosurgery, Hanyang University Medical Center,  
17 Haengdang-dong, Seongdong-gu 133-792, Seoul, Korea  
e-mail: hjyi8499@hanyang.ac.kr

Y.-J. Lee  
Department of Radiology, Hanyang University Medical Center,  
Seoul, Korea

D.-W. Kim  
Department of Anesthesia and Pain Medicine, Hanyang University  
Medical Center, Seoul, Korea

that, although many patients showed improvement of headache after intracranial aneurysm treatment, others have no competent change, with some having worsening or new onset of headache [7, 18, 21].

Postoperative headaches are often found in patients with craniotomy. The prevalence of craniotomy-related headache varies from 0 to 50 % in retrospective studies of patients who had aneurysmal neck clipping for treatment of intracranial aneurysm [2, 9, 22, 23]. Over the last 15 years, endovascular treatment of intracranial aneurysms has evolved significantly, and this minimally invasive therapy can now be applied to a greater number of aneurysms [17]. Therefore, endovascular treatment using coiling is currently used in patients worldwide as the first-line therapy to occlude UIAs. The efficacy has been proved when compared to craniotomy, but functional outcome such as headache in symptomatic aspects was not thoroughly evaluated yet. We performed this study to identify incidence and risk factors of headache after endovascular treatment for UIAs.

## Material and methods

### Patient population

This study was approved by the institutional review board at the author's institute (HYUH IRB 2011-R-5). Endovascular coil embolization was performed by two interventionists (HJY, YJL). One hundred and thirty consecutive patients treated with endovascular coil embolization for UIAs from March 2006 to May 2012 were enrolled in the present study. Some patients were excluded according to the given criteria. We reviewed the medical charts and radiographic materials of the included patients.

### Exclusion criteria

Patients were excluded from this analysis: 1) if they had a non-saccular (fusiform or dissecting) aneurysm; 2) if they had a previous history of aneurysmal subarachnoid haemorrhage; 3) if they had a symptomatic thromboembolic complication that could cause a headache; 4) if they had a history of recent severe headache for a month before embolization, severe head injury, or accompanying intracranial lesions such as arteriovenous malformation, tumour, cerebral infarction, or trigeminal neuralgia. The cases that were included in this study were identified from a retrospective chart review.

### Risk factors

Patients were classified on the basis of several associated risk factors including age, sex, location, size and multiplicity of aneurysm, intraoperative rupture, a previous history of

headache leading to aneurysm diagnosis, volumetric coil packing density, treatment method with regard to stenting. Location of aneurysms consisted of anterior/posterior circulation and internal carotid artery (ICA) segment/other segment. ICA segment was defined as the proximal portion of the carotid T bifurcation. All patients were also divided into two groups according to their age at the level of 60 years. Size of aneurysm was classified according to their maximum diameter as small (<5 mm), medium (5–10 mm), and large ( $\geq 10$  mm).

The coil packing density is thought to have a strong influence in post-coiling headache. The aneurysm volume was calculated under the assumption that aneurysm shape was spherical, using the following formula; Aneurysm Volume =  $4 \times \pi \times (\text{radius})^3 / 3$ . Coil volumes and coil lengths were calculated using the following equation; Coil Volume =  $\pi \times (\text{radius})^2 \times \text{length}$ . Packing density is defined as the ratio between the inserted coils and aneurysm volume. The coil packing density was calculated using the following formula; Packing density = (coil volume/aneurysm volume)  $\times 100$  %. In this study, we used packing density calculating software (<http://www.angiocalc.com>).

### Post-embolization headache

When patients had a headache, they informed on the quality, severity, and frequency of the headache. Patients with prior history of headache were assigned a headache diagnosis according to the International Classification of Headache Disorder II (ICHD-II) criteria. It is important to distinguish between the pre- and post-procedural headache. For the purpose of this study, worsening of headache is defined as at least a 50 % increasing headache severity and frequency on follow-up period, whereas improvement was defined as decreasing. The ratio of patients with postoperative headaches who showed improvement, worsening or newly developed headache were calculated. A patient was considered to have a new onset headache if they had one at least from postoperative day 1 to in-hospital stay. From medical record reviews, we could confirm whether prior headaches exist or not.

### Coiling procedures and supporting techniques

All aneurysms were coiled with patients under a general anesthesia. Heparinized saline was flushed continuously through the catheters during procedure. A bolus of heparin from 3,000 IU was administered after placement of the arterial introducer sheath, followed by an additional 1,000 IU per hour. Embolization of aneurysms was performed on a commercially available biplane angiographic unit equipped with an image-intensifier matrix of 1024x1024 (Philips Medical Systems, Eindhoven, the Netherlands). Each aneurysm embolization was performed using several

detachable coils: TruFill™ (Cordis Neurovascular, Miami, FL, USA), Axium™ (EV3, Irvine, CA, USA), Guglielmi™ (Stryker, Fremont, CA, USA), MicroPlex™ (MicroVention, Aliso Viejo, CA, USA) coils. Neck remodeling techniques (stent-assisted or balloon-assisted) were used for aneurysms with unfavorable angioarchitecture for the simple technique if necessary: Enterprise stent (Cordis Neurovascular, Miami, FL, USA); Neuroform stent (Boston Scientific, Fremont, CA, USA).

The aim of coiling was to pack the aneurysm as densely as possible without compromising the lumen of the parent artery and branch. Embolization was stopped when the last coil could not be further introduced into the sac, when angiographically satisfactory obliteration was achieved including dome rarefaction, or when there was a danger of occluding the normal vascular branch near to the aneurysm or parent artery. Angiograms were obtained immediately after deployment of the last coil to detect thromboembolic complication.

After coil embolization, all patients were admitted to the intensive care unit (ICU) until they were stable. Vital signs, especially blood pressure, were checked each hour, and neurologic examination was also regularly checked. Anticoagulation protocol consisted of subcutaneous administration of heparin, maintaining activated clotting time (ACT) of two to three times above the normal value. Heparin was continued the day after the procedure. Antiplatelet medication including aspirin and clopidogrel was used 2 days before endovascular treatment after loading dosage in all cases. Patients treated with embolization were generally given aspirin 100 mg and clopidogrel 75 mg for 3 to 6 months according to whether a stent was deployed or not.

#### Statistical analysis

All retrieved data were converted into categorical variables, either dichotomization or stratification. Difference in parametric value (volumetric coil packing density) accompanying intracranial aneurysm size was analyzed using univariate analysis of variance (ANOVA). Categorized data (sex, age, intracranial aneurysm location, intraoperative rupture, stent used or not) were analyzed using Fisher's exact or chi-square tests. When appropriate, continuous scale/score data (size of intracranial aneurysm, volumetric coil packing density) were analyzed using a linear by linear association method. To identify independent risk factors of post-embolization headache, logistic regression analysis with backward stepwise regression was performed to select an appropriate model, and a  $p$  value  $>0.20$  was used for removal. We considered a  $p$  value  $<0.05$  to be significant for all analyses. All statistics were performed with SPSS version 18.0 (SPSS, Chicago, Illinois, USA).

## Results

### Incidence and clinical feature of post-embolization headache

During the recent 6 years, 130 patients who met the inclusion criteria underwent endovascular embolization for UIAs. There were 32 male patients (24.6 %) and 98 female patients (75.4 %). The mean age was 62 years old (range, 29–85 years old), constituting  $58.2 \pm 10.73$  years old in the headache group (worsening or newly developed headache), compared to the non-headache group (headache relief or no headache) of  $62.5 \pm 9.48$  years old. There were 39 patients who had experienced preoperative headache, including episodic tension-type headache (10 patients), chronic tension-type headache (14 patients), episodic migraine (9 patients), and chronic migraine (6 patients). Post-embolization headache occurred in 32 cases (24.6 %). Among them, 26 patients had new onset headache and six patients had worsening headache as compared to preoperative status. Headache characteristics were expressed as diffuse, tightened, or bursting. Some patients had cluster and migraine-like headache. In most patients, headache was improved within postoperative 3 months. But intermittent headache persisted for 6 months in 2 patients who had a previous history of chronic migraine.

### Risk factors related to headache development

The characteristics of variables, such as patient-related factors, aneurysm size and location, and procedure-related factors according to presence of post-embolization headache are summarized in Table 1. There were no significant differences in demographic features between the headache group and the no headache group. Of the 39 patients with preoperative headache, most patients (33 patients) experienced headache relief. In a similar vein, a previous history of headache was not significantly associated with development of post-embolization headache ( $p=0.262$ ). The aneurysms located in the ICA segment were more frequent in patients with worsening or newly developed headache than in those without headache (62.5 % versus 49.0 %, OR 2.045, 95 % CI 1.092–4.639;  $p=0.048$ ). Moreover, patients who had a previous history of hypertension tended to have no experience of post-embolization headache (59.2 % versus 34.4 %, OR 0.361, 95 % CI 0.157–0.831;  $p=0.015$ ). In the procedural aspect, the use of a stent device was more frequent in the headache group (53.1 % versus 30.6 %, OR 2.569, 95 % CI 1.135–5.812;  $p=0.021$ ). Packing density was distributed 9–37 %. Volumetric packing density was not significantly associated with development of headache ( $p=0.313$ ). Packing density and headache according to size of the aneurysm had not shown a significant correlation ( $p=0.061$ ). Correlation between packing density and headache was not found if the aneurysm location was dichotomized as internal carotid artery

**Table 1** Comparison of characteristics of variable factors between the patients with headache and without headache after coil embolization of unruptured intracranial aneurysms

	Headache (-) (n = 98)	Headache (+) (n = 32)	P value	Odds ratio (95 % CI)
Age in years	62.5±9.48	58.2±10.73		
<60	60 (61.2 %)	21 (65.6 %)		
≥60	38 (38.8 %)	11 (34.4 %)	0.656	0.827 (0.359–1.906)
Female	73 (74.5 %)	25 (78.1 %)	0.679	1.223 (0.471–3.173)
Hypertension	58 (59.2 %)	11 (34.4 %)	0.015	0.361 (0.157–0.831)
Diabetes mellitus	10 (10.2 %)	5 (15.6 %)	0.405	1.630 (0.512–5.182)
Hyperlipidemia	18 (18.4 %)	9 (28.1 %)	0.315	1.739 (0.690–4.385)
Location of aneurysm				
Anterior circulation	91 (92.9 %)	27 (84.4 %)		
Posterior circulation	7 (7.1 %)	5 (15.6 %)	0.150	2.407 (0.707–8.198)
Location of aneurysm				
ICA segment	44 (44.9 %)	20 (62.5 %)	0.048	2.045 (1.092–4.639)
Other segment	50 (51.0 %)	12 (37.5 %)		
Treatment modality				
Only coiling	74 (75.5 %)	17 (53.1 %)		
Stent-assisted coiling	24 (24.5 %)	15 (46.9 %)	0.021	2.569 (1.135–5.812)
Intraoperative rupture	2 (2.0 %)	1 (3.1 %)	1.000	NA
Size (mm)	5.6±1.87	5.9±2.66		
<5	13 (13.3 %)	2 (6.2 %)	0.233	1.000
5–10	78 (79.6 %)	25 (78.1 %)		2.083 (0.402–14.377)
≥10	7 (7.1 %)	5 (15.6 %)		4.643 (0.547–47.638)
Packing density (%)				
10–20	8 (8.2 %)	5 (15.6 %)	0.313	1.000
20–30	49 (50.0 %)	12 (37.5 %)		0.392 (0.092–1.692)
30–40	41 (41.8 %)	15 (46.9 %)		0.585 (0.141–2.474)

ICA internal carotid artery, CI confidence interval, NA not applicable

and other locations ( $p=0.281$ ). The use of a stent device was significantly associated with the development of headache. There were no statistically significant differences in size of the aneurysm, intraoperative rupture, location of the aneurysm (anterior or posterior circulation), or other coexistent medical illness (diabetes mellitus or hyperlipidemia) between the two groups.

#### Independent risk factors of post-embolization headache

Independent risk factors for the occurrence of post-embolization headache were assessed by logistic regression analysis to control for possible confounding variables. Only two variables had a statistically significant relationship with development of post-embolization headache: no previous history of hypertension (OR 2.806, 95 % CI 1.197–6.574;  $p=0.018$ ) and the use of stent (OR 2.607, 95 % CI 1.127–6.031;  $p=0.025$ ) (Table 2). However, there was no statistically significant association between detailed location of aneurysm and development of post-embolization headache ( $p=0.192$  and  $p=0.137$ , respectively).

#### Discussion

This study suggests that a substantial number of patients experience aggravation or new development of headache after endovascular coil embolization for UIAs. In most patients, post-embolization headaches were transient and resolved at least within 3 months after the procedure. Stent-assisted coiling and no previous history of hypertension were significantly associated with post-embolization headache.

**Table 2** Independent risk factors of headache development after coil embolization of unruptured intracranial aneurysms

	P value	Odds ratio (95 % CI)
No hypertension	0.018	2.806 (1.197–6.574)
Posterior location	0.192	2.598 (0.619–10.898)
ICA segment	0.137	1.912 (0.814–4.492)
Stent-assisted coiling	0.025	2.607 (1.127–6.031)

ICA internal carotid artery, CI confidence interval

## Headache incidence

Development of headache after coil embolization for UIAs has been described in previous studies [7, 18, 20, 24]. Hwang et al. reported that 50 of 90 patients (55.6 %) without history of headache experienced post-embolization headache immediately postoperative that resolved within several days [7]. In the current study, 32 of 130 patients (24.5 %) experienced post-embolization headache. This discrepancy might be due to different time intervals according to the definition of post-embolization headache. We found that the patients had headache after postoperative day 1, suggesting that they occurred beyond the influence of general anesthesia [5]. In a previous study, headache development after minor outpatient surgery under general anesthesia was 43 % between 4 and 6 h postoperatively. A substantial number of patients might experience headache on the day of coil embolization under general anesthesia.

## Mechanism of post-embolization headache

Endovascular therapy can induce headaches and edema surrounding the aneurysm by several mechanisms such as local thrombosis, dilation of the vessel wall or inflammation within the aneurysm sac after placement of coils [14, 25]. After the endovascular procedure, increased sympathetic tone from irritation of vessels develops headache. Some authors described that the patients who underwent the endovascular procedure experience headache with a similar mechanism as stated above [1, 12]. The headache might be resolved completely when the edema surrounding the aneurysm disappears.

In this study, the aneurysm located in the ICA segment was associated with post-embolization headache in univariate comparison. Of 64 patients with ICA segment aneurysm, substantial numbers of patients were treated with balloon-assisted (13 patients, 20 %) or stent-assisted coiling (22 patients, 34 %). This trend seems to be directly related to the stimulation of the arterial wall which is possible cause of post-embolization headache. However, correlation between ICA segment aneurysm and headache according to the use of balloon or stent was not found and there was no statistically significant difference between two groups in multivariate analysis ( $p=0.284$  and  $p=0.137$ , respectively).

In a previous study, absence of a history of hypertension was correlated with susceptibility to post-embolization headache in patients with unruptured aneurysms, as was also found in this study [7]. Because of the more elastic and distensible nature of healthy vessels, the walls of aneurysms without hypertension could be further modified by pressure increases within aneurysmal sacs [7]. These findings might be associated with post-embolization headache in patients without a history of hypertension.

## Use of stent and headache development

The intracranial arteries, especially the proximal portions of the major vessels, are innervated by sensory nerves that could potentially be activated by aberrant blood flow and structural changes of the vessel wall [10, 13, 15, 19]. According to Irima et al., the traction on sensitive trigeminal and parasympathetic fibers with an inflammatory process in the cavernous sinus after aneurysm coiling is suggested to be the cause of headache [8]. The implantation of a stent within a parent artery near the cavernous segment might stimulate a surrounding nerve fiber by a similar mechanism. In a recent study by Piotin and colleagues, evaluating stent-assisted coiling in both ruptured and unruptured aneurysms, they found that the use of a stent is related to a low prevalence of angiographic recurrences, but with an increased risk of complications [16]. Stent-assisted coiling can be associated with post-embolization headache via numerous possible mechanisms to include underlying differences in aneurysm characteristics, activation of vascular sensory afferents by the stent, and remodeling of the aneurysm neck and parent vessel [28]. The implantation of a stent within a parent artery may straighten the vessel, altering the flow dynamics within the aneurysm. The magnitude of this effect is affected primarily by the rigidity of the stent. However, the rigidity of the stent (open cell or closed cell) was not statistically correlated with post-embolization headache ( $p=0.333$ ) in this study. The post-embolization headache is significantly different when we apply the stent comparing with the one with coil alone in both groups.

## Coil packing density and headache development

It is well known that geometry of the aneurysm affects coil packing and the angiographic result. Although the exact mechanism by which occlusion after coil embolization is unknown, the factors affecting aneurysm occlusion are thought to be dome-to-neck ratio, dome-to-height ratio, aneurysm size, and absolute neck diameter. Clot maturation and neoendothelial coverage require the precondition that the coil is packed enough in the aneurysm and around the neck, and that the coil mesh tolerates hemodynamic stress during clot organization [11]. Intra-aneurysmal thrombosis and the coil mass itself lead to local inflammation associated with headache [3, 4]. In a related move, Hwang et al. described that post-embolization headache develop more frequently with a high packing attenuation [7]. However, packing density was not correlated with post-embolization headache in the current study. Although some advocated packing density has an effect on headache differently according to the size of the aneurysm, it was not proved in this study, and the correlation between packing density and headache according to the location of the aneurysm was not significantly. As stated above, the important

factor in the development of headache after coil embolization may be the inflammatory response or an increase in aneurysm size related to packing density [14, 25]. Oppositely, coil embolization may reduce the pulsatile expansion or may result in retraction of the aneurysm sac, leading to the improvement of headache [18]. The packing density can be affected by the changes of the flow hemodynamics and same numerical value of the packing density could cause the different result depending on the coil type and physician's technique. Therefore, further large studies are required to assess the effect of packing density on post-embolization headache.

### Limitations

While the findings of the current study were important, there were several limitations. First, the retrospective nature of patient collection is a critical limitation of the current study. Retrospective studies may underestimate headache incidence through the introduction of investigator recall bias [6]. Second, there was significant case complexity and limitation by a small number of cases in our patient population. Therefore, these findings may not be generalized to a wider group of endovascular treatment patients. Intraoperative rupture or aneurysms in posterior circulation headache was not statistically associated with post-embolization in this study, but the reliability is low due to the lack of a sufficient number of cases. Third, several kinds of coils were used in most cases. Therefore, it is difficult to evaluate how each coil effects post-embolization headache. Fourth, we overlooked psychological effects about headache. It was already proved that post-embolization headache could be developed by psychological causes, such as depression or anxiety disorder [20]. Several psychologic factors would be confounded with organic causes. Finally, calculation of volumetric coil packing density seems to have been exaggerated. In this study, volumetric coil packing density was calculated high compared to the averages of other literature. It was thought that the aneurysm volumes were not calculated using a three-dimensional method.

### Conclusions

We have studied the influence of coil embolization for UIAs on post-embolization headache. The use of a stent and no prior history of hypertension were two important predictors of post-embolization headache in patients with UIAs. In future large studies, it would be interesting to quantify or to qualify the strength of association between several risk factors and post-embolization headache. Identification of risk factors and a practical pattern of headache might allow us to be provided information on endovascular treatment of unruptured aneurysms for many interventionists and patients. Furthermore,

such risk factors would be estimated as modifiable, so alleviation of headache might be possible.

**Conflict of interest** None

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