Radiation-induced organized hematoma after gamma knife radiosurgery for arteriovenous malformation

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Kyu Seon Chung

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ABSTRACT

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Purpose

This study aimed to evaluate the incidence of a late complication, radiation-induced organized hematoma (RIOH), after gamma knife radiosurgery (GKRS) for arteriovenous malformation (AVM).

Materials and Methods

Between May 1991 and June 2012, 694 patients underwent GKRS for AVM. All patients were administered a marginal dose of 12–18 Gy with 50% isodose line. When the AVM was not obliterated after 3 years, additional GKRS was performed.

Results

Thirteen (1.9 %) patients with RIOH were analyzed in our study. Except for 1 patient, 12 patients underwent multiple GKRS for remnant AVM (twice in 6 patients, thrice in 2 patients, four times in 2 patients, after external radiation therapy in 1 patient, and after Novalis stereotactic radiosurgery in 1 patient). The mean duration of RIOH development was 12.6 years (range, 7.7–21 years). Of the 13 patients, 9 (69.2%) needed surgery because RIOH enlargement was detected on follow-up magnetic resonance imaging.

Conclusions

Not only bleeding from AVM during the latent period after GKRS but also RIOH should be carefully followed up because of the necessity for decompression.

Key words : gamma knife radiosurgery, radiation-induced organized hematoma, arteriovenous malformation, gkrs complication

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I. INTRODUCTION

Cerebral arteriovenous malformations (AVMs) are rare, but they require careful treatment because they can cause neurological symptoms by chronic vascular steal effect and intracranial hemorrhage by nidal rupture. There are several treatment modalities for the treatment of AVMs, including microsurgical resection, embolization, and gamma knife radiosurgery (GKRS). Studies have shown that GKRS has been increasingly used for the treatment of AVMs. Within 2–3 years after GKRS treatment, 70% patients achieve complete obliteration of the nidus, the ultimate goal of AVM treatment. GKRS is a safe and minimal invasive method, but some complications have been reported with its use.[1-3]

Because the vascular change after GKRS is gradual, the risk of rebleeding or new intracranial hemorrhage by nidal rupture is well known as a latent period complication. Moreover, radiation-induced edema, cyst formation, and organized hematoma may develop after GKRS treatment during follow up. Among these, radiation-induced organized hematoma (RIOH), which can cause critical morbidity in patients, has been reported sporadically, and there is a lack of systematic reports on its mechanisms and clinical course.[3-7]

Here, we retrospectively analyzed data from our institution on the clinical course and management of RIOH after GKRS.

II. MATERIALS AND METHODS

Patients with AVM who underwent GKRS between May 1991 and June 2012 at our institution were included in this study. A total of 844 GKRS procedures were performed for AVMs in 694 patients. We treated each case with GKRS model B (until May 1992), model C (until November 2008), and Perfexion (after November 2008; Elekta, Stockholm, Sweden). Leksell stereotactic frame fixation was performed in all patients. Except for contraindications, magnetic resonance imaging (MRI) with gadolinium enhancement and cerebral digital subtraction angiography (DSA) were performed for treatment planning. All patients were administered a marginal dose of 12–18 Gy with 50% isodose line. After the first treatment, patients underwent MRI and DSA during periodic follow ups. If complete obliteration was not observed within 3 years, additional GKRS was repeatedly performed.

MRI findings show RIOH as a mass lesion similar to cavernous malformation. However, enhancing lesions are observed in RIOH, in gadolinium-enhanced T1-weighted magnetic resonance imaging. The RIOH has an incomplete popcorn-like appearance, partial hemosiderin rims and perilesional edema.[8]

Of the 694 patients with AVM, 13 had RIOH. These patients were followed clinically and radiologically until August 2019. In our retrospective analysis, we examined the characteristics of patients, AVMs, and GKRS treatment. Furthermore, we evaluated the clinical course and radiological and pathological findings.

III. RESULTS

1. Incidence of RIOH

Thirteen (1.9%) patients (6 women and 7 men; median age at first treatment: 28.2 years [range, 13–45 years]) with RIOH were analyzed in our study. Regarding the location of AVM nidus, the occipital lobe was the most common

(6 patients). The Spetzler–Martin grade at the initial diagnosis of AVM was as follows: 2 (1 patient), 3 (5 patients), and 4 (7 patients). In patients whose data were analyzed, the mean AVM nidus volume at the time of initial treatment was 28.9 cm3 (range, 6.2–61.6 cm3). Twelve patients were repeatedly treated with GKRS for remnant AVM (twice in 6 patients, thrice in 2 patients, four times in patients, after external radiation therapy in 1 patient, and after Novalis stereotactic radiosurgery [SRS] in 1 patient). AVM embolization was performed in 4 patients before the first GKRS. Moreover, there were 3 patients with intracranial hemorrhage before treatment. In patient no. 8, intracranial hemorrhage developed before the nidus was completely obliterated between the first GKRS and second GKRS. No surgical resection for AVM nidus was performed before GKRS (Table 1).

Table 1. Patients, AVMs, and GKRS Characteristics

Case No.	Sex /Age†(yr)	AVM location	SM grade	AVM volume (cm^3)	Repeated GKRS ‡	GKRS plan Marginal dose (Gy)	AVM embolization before GKRS	AVM rupture before GKRS
1	F/25	Left occipital	S2V0E1	46.2 33.4 5.3 3.9	#1 1999 #2 2003 (56mo) #3 2007 (42mo) #4 2009 (27mo)	15 12.5 15 13	No	No
2	M/44	Left occipital	S2V0E1	6.2 6.3	#1 1996 #2 2000 (40mo)	15 14	No	No
3	M/30	Right cerebellum	S2V1E1	33.7 5.7	#1 1997 #2 2011 (168mo)	13 13.5	Yes	Yes
4	F/39	Left parietal	S2V1E1	57.1 46.3 9.0 1.9	#1 1999 #2 2004 (60mo) #3 2007 (32mo) #4 2014 (77mo)	14 13.5 12 15	No	No
5	M/21	Right occipital	S2V1E1	10.0 1.6	#1 2000 #2 2003 (36mo)	17.5 14	Yes	No
6	F/13	Left temporal	S2V0E1	15.6 4.6	#1 2000 #2 2003 (40mo)	16 14	No	No
7.	M/39	Left parietal	S2V1E1	23.0 6.1 5.7	#1 2001 #2 2004 (36mo) #3 2006 (24mo)	14 15 12.5	No	No
8	F/19	Left occipital	S2V1E1	61.6 39.7	#1 2003 #2 2005 (29mo)	12.5 13	No	Yes[§]
9	F/45	Left occipital	S1V1E1	21.0	#1 2003	15	Yes	Yes
10	M/18	Right parietal	S2V1E1	10.6 2.7	#1 2005 #2 2009 (42mo)	15 13	No	No
11	M/31	Right temporo -occipital	S2V0E1	55.1 89.3 43.0	#1 1997 #2 2009 (144mo) #3 2013 (51mo)	12.5 13 14.5	No	No
12	M/24	Left parietal	S1V0E1	* 3.0	#1 1993 (EFRT) #2 1996 (34mo)	5400 cGy[1] 14	No	No
13	F/19	Right parietal	S2V1E1	6.3 1.3	#1 2009 (Novalis) #2 2012 (34mo)	20[2] 16	Yes	Yes

†, age at the first treatment
SM, Spetzler–Martin; S, size; V, venous drainage; E, eloquence
‡, year when GKRS performed (interval from previous GKRS)
§, AVM rupture after the first GKRS and before the second GKRS
*, no data
1, external radiation therapy, total radiation dose
2, Novalis SRS, 80% isodose line

2. Clinical course of RIOH

DSA confirmed total obliteration of the nidus in all the remaining patients, except 2 patients. In these 2 patients, both RIOH and remaining AVM nidus were

removed at the same time. In a mean of 12.6 years (range, 7.7–21 years), RIOH was identified after the initial treatment. Nine of the 13 patients had severe brain edema associated with RIOH, which caused neurologic symptoms. They underwent surgical resection of RIOH. Three of the 9 patients were initially diagnosed with symptom development and immediately underwent surgical treatment. Perilesional brain edema and other symptoms improved after surgery. Four patients who did not receive surgical treatment have not complained of worsening neurological symptoms, and aggravation on radiological examination was not observed after the first diagnosis of RIOH. In patient no. 8, the RIOH was large and located deep in the thalamus; hence, it was partially resected. No exacerbation of the remnant lesion was observed postoperatively in the 5-year follow up. Patient no. 2 had de novo RIOH on MRI 1 year after total resection, and there was no specific interval change in 2.8 years (Table 2).

Table 2. Clinical course of RIOH

Case No.	Time to angiographic obliteration after the first GKRS (years)	Symptom of RIOH	First detection of RIOH after the first GKRS (years)	Surgical intervention after the first GKRS (years)	Postoperative follow-up duration (years)	RIIC confirmed before RIOH detection	Cyst formation
1	12.5	Headache	12.5	12.5	6	Yes	Yes
2	9.6	Visual disturbance	17.7	18.7	3.8	Yes	No
3	17.7	No	18.7	No	2*	No	No
4	†	Right-sided weakness	14	15	3.6	Yes	No
5	5.1	Visual disturbance	11.5	11.5	6.2	No	Yes
6	5.6	No	13	No	5.1*	Yes	Yes
7	13.1	No	13.1	No	4.5*	Yes	Yes
8	5.4	Gerstmann syndrome	8	Cyst: 8 ‡ Resection: 11.5	5	Yes	Yes
9	8.6	Mental change	7.9	11.9	1.9	Yes	Yes
10	7	No	9.5	No	4.4*	Yes	Yes
11	†	Visual disturbance	21	21	0.5	Yes	Yes
12	7.6	Seizure	10.1	25.6	0.1	Yes	Yes
13	5.7	Seizure	6.7	Cyst: 7.3 ‡ Resection: 8.5	1	Yes	Yes

RIIC, radiation-induced imaging change
†, remnant AVM nidus was totally resected with RIOH
‡, surgical treatment for cyst before RIOH resection
*, follow-up period after first detection

3. Radiological course of RIOH

The MRI images of patients who did not need immediate surgical resection when RIOH was initially detected were checked in chronological order. In some cases, the size of the lesions has gradually increased, and in other cases, the size of the lesions has suddenly increased (Figure 1).

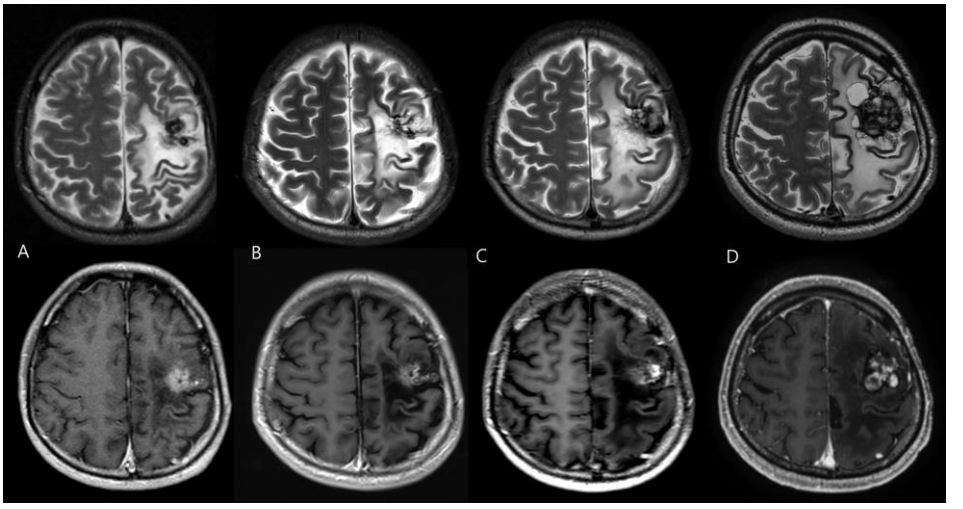

Figure 1. Changes in RIOH over the years. We followed the patient no. 12 regularly after GKRS. A) MRI scan 2.5 years after angiography confirmed total obliteration of AVM nidus. MRI showed RIOH and perilesional edema at the left parietal lobe. He did not complain of neurologic symptoms. Thus, we observed the patient regularly without treatment. B) MRI scan 4 years after initial diagnosis of RIOH. C) MRI scan 10 years after initial diagnosis of RIOH. As the RIOH continued to grow, the surrounding edema aggravated. D) The patient had seizure 15.5 years after initial diagnosis of RIOH. MRI showed rapidly enlarged RIOH and severe edema. Therefore, surgical resection of RIOH was performed.

Severe edema was observed surrounding RIOH when patients needed surgical resection. Perilesional edema was resolved after surgical resection (Figure 2).

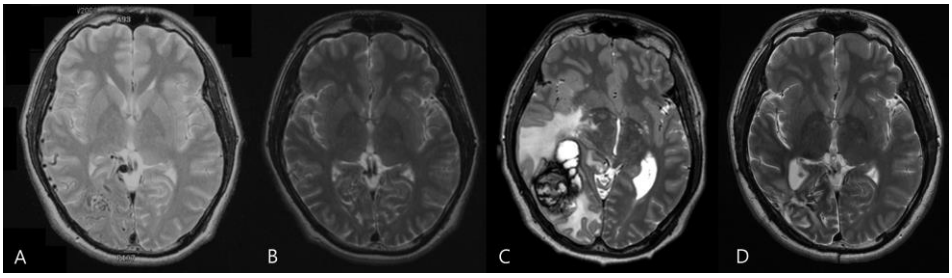

Figure 2. A 21-year-old man (case 5) presented with seizure. A) MRI scan showed AVM at the left occipital lobe. He underwent GKRS twice for AVM. B) MRI scan showed total obliteration of AVM 5 years after the first GKRS. C) He presented with headache and diplopia 11.5 years after the first GKRS. RIOH and severe cerebral edema were identified. He underwent surgical resection of RIOH. D) Follow-up MRI scan at 1 year postoperatively showed improved cerebral edema and no evidence of recurrence.

RIOH was identified in two patients with remnant AVM nidus. In the DSA conducted at this time, the RIOH had no AVM nidus and was not stained by the contrast (Figure 3).

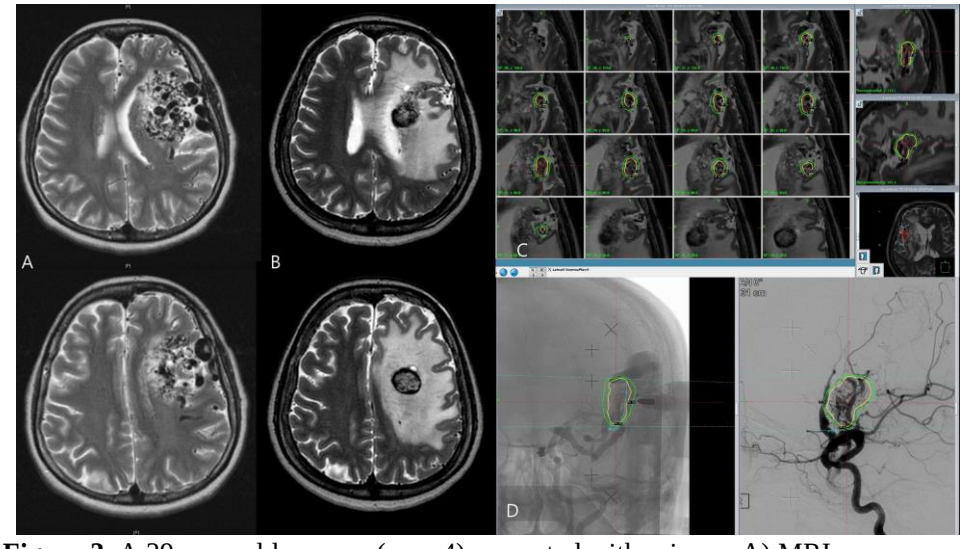

Figure 3. A 39-year-old woman (case 4) presented with seizure. A) MRI scan showed large AVM on the left parietal lobe. She underwent repeated GKRS. B)

RIOH and remnant AVM nidus were observed 14 years after the first GKRS (32 months after the third GKRS). She had no neurologic symptom. The fourth GKRS was performed. C, D) Image from the fourth GKRS planning. On cerebral angiography, there was no nidus lesion in the RIOH. Therefore, we excluded RIOH in the GKRS planning field.

In 10 (76.9 %) patients, RIOH and cysts were simultaneously identified. In two patients, cysts were treated first by aspiration and Ommaya reservoir insertion, and then symptoms improved. However, during the follow-up observation, they underwent a second surgery because of newly developed cysts and RIOH worsening (Figure 4).

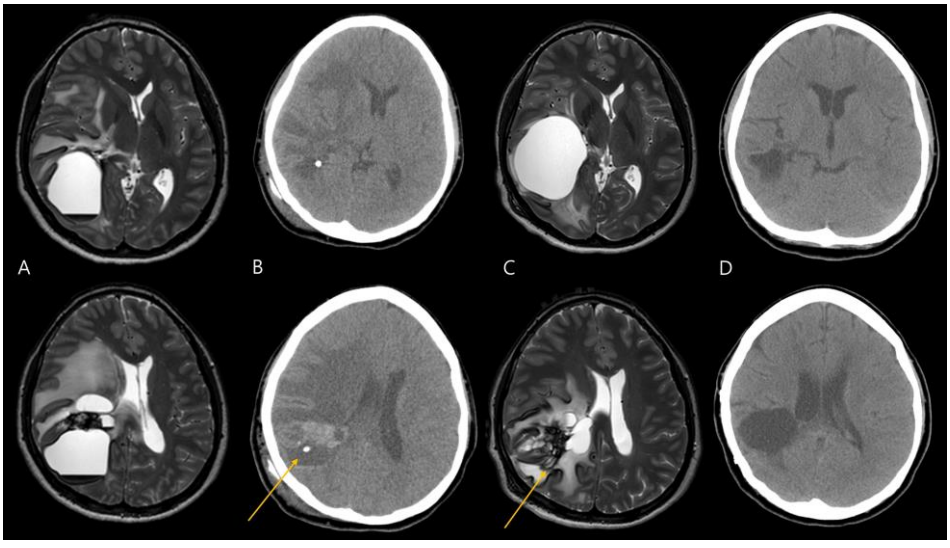

Figure 4. Case (case 13) that shows RIOH accompanied with cyst. A) Multiple cysts and RIOH were observed 7.4 years after the total obliteration of the AVM nidus. The patient presented with seizure. She underwent Ommaya reservoir insertion for large cyst that was located posterior to the RIOH. B) On computed tomography (CT), the cyst became smaller 1 day postoperatively. Her symptoms also improved. C) Seizure recurred 1 year postoperatively. There are newly developed large cyst and growing RIOH. Surgical resection of the RIOH and cysts was performed. D) CT images showed absence of cysts and RIOH. There were

also no symptoms. Arrow, Ommaya reservoir catheter

Radiation-induced imaging change (RIIC) was confirmed in 11 (84.6 %) patients before the development of RIOH.

4. Pathological finding of RIOH

Gross RIOH showed homogenous hematoma-like lesions with dense collagenous tissue capsule. Microscopically, various staged clots with hemosiderin deposits were observed. Furthermore, recent hemorrhage and irregular compressed capillary-sized vascular channels were observed. There were radiation necrotic tissues without vascular anomaly.

IV. DISCUSSION

There are several known complications that develop late after treating AVM with GKRS. Cyst formation is the most common late complication. The incidence of cyst formation varies from 1.17% to 5%.[1,3,9-11] RIOH is discussed in several case reports, but studies on its incidence are insufficient. RIOH is often accompanied by cysts. Ten (76.9 %) patients in our study had cyst. Nakajima et al. reported that 20 of 404 patients had cysts, and 11 of them had RIOH.[3] Shuto et al. analyzed the presence of cyst in 18 of 775 patients and confirmed that 5 of them had RIOH.[10] Their study did not include RIOH without cysts, making it difficult to determine the exact incidence. In this study, 13 (1.9 %) of 694 patients were diagnosed with RIOH. The results seem quite high compared to those in several known cases. Pan et al. reported that the incidence of cyst formation in GKRS cases was 1.6%, but this incidence increased to 3.6% when they extended their follow-up duration to >5 years after GKRS. Therefore, complications after GKRS, such as cyst formation, are more likely to develop over a long period of time.[11] Likewise, RIOH is one of the late complications. In this study, we reviewed data on patients treated from May 1991 to June 2012 and who were followed up until August 2019. Thus, the incidence results are meaningful.

The mechanism of RIOH formation following GKRS in the treatment of cerebral AVM is still unclear and controversial. Some authors have suggested that repeated minor bleeding from fragile vessels in radionecrotic brain tissue is the cause of RIOH, similar to the formation of chronic subdural hematoma.[4,5,7] RIOH did not

show pathological findings of vascular malformation, such as cavernous malformation.[7,8] Nakamizo et al. reported the role of vascular endothelial growth factor (VEGF) in RIOH formation while analyzing RIOH associated with incompletely obliterated AVM. Hypoxic state and minor bleeding from the incompletely obliterated AVM nidi may draw the focal activation of VEGF pathway. Moreover, it may lead to neovascularization in the granulation layer of the hematoma capsule that serves as a continual source of bleeding and perifocal edema.[6] Shuto et al. described the lesion with contrast enhancement on MRI following GKRS as radiation-induced angiomatous change, which is the origin of cyst and RIOH. They explained that, if the bleeding from this angiomatous lesion spreads mainly into the brain parenchyma, a cyst develops, and repeated bleeding within this angiomatous lesion produces RIOH. Therefore, the authors suggested that the causes of cyst and RIOH formation are essentially similar as they often develop simultaneously.[10] Some authors have reported that RIOH after GKRS is not specific to an AVM.[8,10] In some cases, RIOH developed within the brain parenchyma adjacent to the irradiated nidus without continuity with occluded nidus.[10] Cha et al. analyzed the RIOH after GKRS treatment in AVM and glioma simultaneously. They explained that when target lesions shrink by GKRS, a small cavity is formed and hemorrhage fills the destructive tissue in the cavity, resulting in RIOH.[8] When we examined our cases, vascular malformation was not visible, and we agreed with previous reports that repeated bleeding in the brain parenchyma destroyed by radiation caused RIOH. Therefore, radiated intermingled normal brain tissue in AVMs may be associated with RIOH development. In 11 patients, RIOH developed after total obliteration of AVM nidus, and in 2 patients, RIOH was identified with partially obliterated AVM nidus. AVM nidus was not observed in RIOH lesion in the DSA of these two patients. As a result, it is considered that RIOH developed where AVM nidus was obliterated. However, the cause of the sudden increase and exacerbation of RIOH is unknown. Additional research is needed in this area.

The risk factors for causing RIOH are still unknown. Park et al. suggested that large-volume AVM (longest diameter >3 cm in 4 cases), repeated radiosurgery, and a large cumulative radiation dose are the risk factors.[7] In our cases, the longest diameter of AVM in 11(84.6 %) patients was >3 cm, and repeated radiosurgery was performed in 12 (92.3 %) patients. These results correlate with a previous study.

After treatment of AVM with GKRS, the newly appearing increased signal surrounding the treated AVM nidus on MRI T2-weighted imaging is called RIIC. Moreover, 33.8% of patients had RIIC after GKRS treatment, and large AVM and

single draining vein are risk factors for the development of RIIC.[12] Pomeraniec et al. observed RIIC in 64.7% patients with cysts after SRS and 36.1% patients without cysts and suggested that longer duration of RIIC could be a risk factor of cyst formation.[9] In our study, RIIC was observed in 11 patients (84.6 %) on MRI prior to the development of RIOH. Considering the mechanism of cyst and RIOH formation discussed above, RIIC can be regarded as the risk factor of RIOH.

Nine (69.2 %) patients required surgical resection of RIOH. Six of 9 patients underwent surgical resection several years after the initial RIOH identification. The period varied from 1 to 15.5 years. It is impossible to predict when RIOH suddenly worsens, so the four patients without symptoms should be carefully monitored. Cyst and RIOH were observed in 10 (76.9%) patients. In two of these, we initially treated the cyst through aspiration and Ommaya reservoir insertion. However, during the follow-up, new cysts were formed and RIOH increased and worsened, so we surgically resected RIOH and cysts. Moreover, there is no recurrence of cyst postoperatively. If surgical intervention is necessary because of neurological symptoms in patients with cyst accompanied by RIOH, RIOH resection should be considered, rather than merely resolving cyst, considering RIOH lesion as origin.

The retrospective design with a small number of patients is the limitation of our study. It is necessary to study the pathogenesis, risk factors, and clinical course of RIOH, which is the late complication arising after treating AVM with GKRS, using large data from multiple GKRS centers.

V. CONCLUSION

Although the incidence of late complications that develop after treating AVM with GKRS is low, it is important to monitor patient symptoms with periodic imaging after treatment. Even if imaging findings show complete AVM obliteration, late complications, such as RIOH, can develop. If there is a lesion that is suspected to be RIOH on imaging, it should be carefully observed because surgical resection should be considered in patients in whom the size of the lesion increases.

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ABSTRACT(IN KOREAN)

뇌내 동정맥 기형의 감마나이프 방사선 수술 치료 후 발생한 방사선에 의한 조직화된 혈종

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정 규 선

목적 : 감마나이프 방사선 수술을 통해 뇌동정맥기형을 치료하고나서 뒤늦게 발생하는 합병증으로 방사선에 의한 조직화된 혈종이 있다. 이 조직화된 혈종의 발병률 및 임상 경과에 대해 연구하였다.

재료 및 방법 : 1991년 5월부터 2012년 7월까지 총 694명의 환자들이 본원에서 뇌동정맥기형에 대해 감마나이프 방사선 수술을 받았다. 모든 환자들은 12Gy에서 18Gy의 경계선량으로 치료받았다. 치료 이후 3년 내외로 뇌동정맥 기형의 폐색이 되지 않은 경우, 반복적인 감마나이프 방사선 수술이 시행되었다.

결과 : 13명 (1.9 %) 의 환자가 방사선에 의한 조직화된 혈종이 진단되었다. 1명의 환자를 제외한 12명의 환자들은 남아있는 뇌동정맥기형에 대해 반복적인 감마나이프 방사선 수술을 받았다. 2번 받은 환자는 6명, 3번받은 환자는 2명, 4번 받은 환자는 2명이었으며, 한 명은 타병원에서 시행한 체외방사선 치료 후 추가 감마나이프 방사선 수술이 이루어졌으며, 다른 한 명은 타병원에서 Novalis 정위방사선 수술 이후 추가 감마나이프 방사선 수술을 받았다.

방사선에 의한 조직화된 혈종이 진단된 시기의 평균값은 12.6년(범위 7.7-21년) 이었다. 13명의 환자 중 9명 (69.2 %)

환자는 방사선에 의한 조직화된 혈종이 커지고 이로 인하여 증상이 악화되어 수술적 치료를 받았다.

결론 : 감마나이프 방사선 수술로 뇌동정맥기형을 치료 후 완전 폐색이 되기 전 발생하는 뇌출혈 또한 중요하지만, 방사선에 의한 조직화된 혈종 또한 수술적 치료가 높은 비율에서 필요할 수 있기 때문에 주의 깊게 경과관찰 하여야 한다.

핵심되는 말 : 감마나이프 방사선 수술, 방사선에 의한 조직화된 혈종, 뇌동정맥 기형, 감마나이프 방사선 수술 후 합병증