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Original Article

Descent of the anterior communicating artery after removal of pituitary macroadenoma using transsphenoidal surgery

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Abstract

Background: After removal of pituitary macroadenoma, the anterior communicating artery (AComA) descends toward the original position. However, the process and contributing factors of this descent are not elucidated.

Methods: This retrospective study included 102 patients who underwent transsphenoidal surgery (TSS) for macroadenomas with maximum diameters of >2 cm. Sequential T2-weighted magnetic resonance images were used to assess the AComA flow void and its distance from the planum sphenoidale before and after TSS. The AComA position in relation to the adenoma was divided into four groups as follows: anterior, anterosuperior, superior, and posterior. The descent was compared to the presence of intratumoral hemorrhage or adenoma extension into the sphenoid sinus.

Results: One week after TSS, the AComA descent was more pronounced than when originally in the superior position (6.5 ± 3.7 mm vs 4.4 ± 3.5 mm, $P < 0.0001$). The postoperative descents of the AComA were well correlated with those of residual adenomas only when in the superior position ($P = 0.030$). The AComA descent was more significant at 1 week (4.4 ± 3.5 mm) than at 1 week to 3 months (0.7 ± 1.0 mm) in all the groups. Both intratumoral hemorrhage and sphenoid sinus extension of adenoma did not affect the AComA descent in each group.

Conclusion: AComA descent was most influenced when it was superior to the macroadenoma and progressed mostly within 1 week after TSS, probably initiating during TSS. The position of the AComA in relation to a macroadenoma should be considered preoperatively to avoid vascular injury.

Key Words: Anterior communicating artery, magnetic resonance imaging, pituitary adenoma, transsphenoidal surgery

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INTRODUCTION

Transsphenoidal surgery (TSS) is the surgical procedure of choice for sellar and parasellar lesions because of its effectiveness and safety. Although unusual, hemorrhagic vascular complications associated with TSS can lead to serious morbidity and mortality. Pituitary adenoma and the pituitary gland itself are located in the center of the circle of Willis and, accordingly, are well vascularized. Therefore, vascular complications can occur not only intraoperatively after surgical manipulation but also intraoperatively and postoperatively because of the shift of the main trunks in the circle of Willis, which is markedly displaced by extension of the macroadenoma into the suprasellar cistern and cavernous sinus.[2,10]

Pituitary macroadenomas with suprasellar extension can elevate the anterior communicating artery (AComA) and optic chiasm. During and after the removal of macroadenomas, these two structures can descend toward the original positions. However, the process of AComA descent has not been investigated previously. If the AComA is involved in vascular anomalies such as cerebral aneurysms, AComA descent might result in vascular complications during or after TSS, which are life-threatening consequences.[1,3,5,6,9,11-13] Vascular complications during and after TSS are the result of abrupt tumor capsule collapse into the sella turcica following adenoma removal, which may cause traction of the AComA.[9,13]

The goal of this study was to quantitatively evaluate AComA descent after TSS to clarify its process and contributing factors after removal of macroadenomas.

MATERIALS AND METHODS

This retrospective study included 102 patients who underwent TSS for removal of a pituitary macroadenoma with maximum diameters of >2 cm at the Kanazawa University Hospital between 2006 July and 2014 December. Endoscopic techniques were used in all the surgeries performed for these patients. Small sections of fat and fascia were used as packing materials when cerebrospinal fluid leakage was detected. The sellar floor was reconstructed with nasal bone fragment. Patients were enrolled with the approval of the institutional review board of Kanazawa University.

Patient demographics, including sex, age, and tumor histology, and the presence of atherosclerotic risk factors, such as hypertension, diabetes mellitus, and hyperlipidemia, were obtained from the clinical records of the patients. This study consisted of 51 men and 51 women, with age at diagnosis ranging from 20 to 82 years (mean, 56.6 years; median, 58 years). Tumor histology was used to confirm the diagnosis of a pituitary adenoma in 102 patients (nonfunctioning in 92, prolactin secreting in 9, and thyroid-stimulating hormone secreting in 1). In this study, hypertension, defined as an average systolic or diastolic blood pressure of ≥130/85 mmHg, was present in 38 patients. Diabetes mellitus, defined as an elevated fasting plasma glucose level ≥100 mg/dl, was present in 23 patients. Hyperlipidemia, defined as a high serum triglyceride level of ≥150 mg/dl, and a low high-density lipoprotein-cholesterol level of <40 mg/dl in men or <50 mg/dl in women, was present in 34 patients.[14]

Neuroradiological evaluation

Magnetic resonance (MR) images were obtained by using a 3.0-Tesla-magnet-strength scanner (Signa HDx 3T, GE Medical Systems, Milwaukee, WIS). MR imaging was performed with fast spin-echo T2-weighted sequences (repetition time, 2500–3500 ms; echo time, 98–104 m; flip angle, 90°; field of view, 14 × 14 cm; matrix, 288 × 224 or 256 × 192; section thickness, 2.0–3.0 mm; and section gap, 0.5 mm).

Sequential T2-weighted MR images were used to determine the position of the AComA in relation to the pituitary macroadenoma on both coronal and sagittal sections before and at intervals after TSS. The AComA was defined as the artery between the bilateral A1-A2 junctions. In the case of an AComA too short to be detected by using MR imaging and angiography, the lower A1-A2 junction was substituted for the AComA. Because these MR images only showed topological information regarding the AComA and its surrounding artery such as A1 and A2, preoperative MR angiography was performed in all the patients in this study. The course of the AComA and surrounding arteries was matched with the flow-void patterns of the arteries on the T2-weighted MR images.

The height of the AComA was defined as the vertical distance of the lower margin of the AComA flow void from the planum sphenoidale, as indicated in the study by Park *et al*.[8] In this study, the heights of the AComAs on the midline of the sagittal section of the T2-weighted MR images were considered "line a," and the maximum heights of the adenomas from the planum sphenoidale were considered "line b" [Figure 1a]. Adenomas with maximal diameters of ≧10 mm were classified as macroadenomas. The AComA locations with respect to the adenoma was divided into four groups as follows: A (anterior), AS (anteriorsuperior), S (superior), and *P* (posterior). The location of each group was defined by the angle crossed at the midpoint of the intratumoral portion of an imaginary line that extends from the planum sphenoidale to the lower margin of the AComA. The angles were divided into groups according to the following ranges; 0–30° (group A), 31–60° (group AS), 61–90° (group S), and 91–180° (group P) [Figure 1b]. Among the participants of this study, 18 patients were in

group A, 30 in group AS, 45 in group S, and 8 in group P [Figure 2a-d].

We investigated if intratumoral hemorrhage could affect the AComA descent after TSS for pituitary adenoma. In 27 patients, a massive intratumoral hemorrhage was identified and defined as a hematoma, which accounted for >50% of the adenoma volume. The volumes of the adenoma and hematoma were determined by multiplying their maximum height, width, and depth. The sphenoid sinus extension of the adenomas was also examined because the AComA could fall into the cavity in the sphenoid sinus formed after tumor removal, which is beyond the original position, as the AComA can be stretched by a macroadenoma. In the present study, sphenoid sinus extension could be detected and was defined as a tumor downward extension of more than two-thirds of the height of the sphenoid sinus on the sagittal section of the MR image.

Statistical analyses

The Chi-square test was used to determine if the patients' demographic characteristics, including sex, age, arteriosclerotic risk factors (e.g., hypertension, diabetes mellitus, and hyperlipidemia), intratumoral hemorrhage, and sphenoid sinus extension affected the AComA descent 1 week after TSS. One-factor analysis of variance (ANOVA) and *post hoc* analysis were used to compare the preoperative heights of the AComA, the maximum diameter above the planum sphenoidale of the adenoma, and the AComA descent 1 week after TSS among the four groups. One-factor ANOVA and *post hoc* analysis were also used to compare the sequential AComA descent between 1 week and 3 months after TSS in each group. The correlation between the postoperative AComA descent and the heights of the residual adenoma on the midline was assessed by using Pearson's correlation coefficient. These statistical analyses were performed by using Microsoft StatView ver. 5 (SAS institute Inc.). A P value of <0.05 was considered statistically significant.

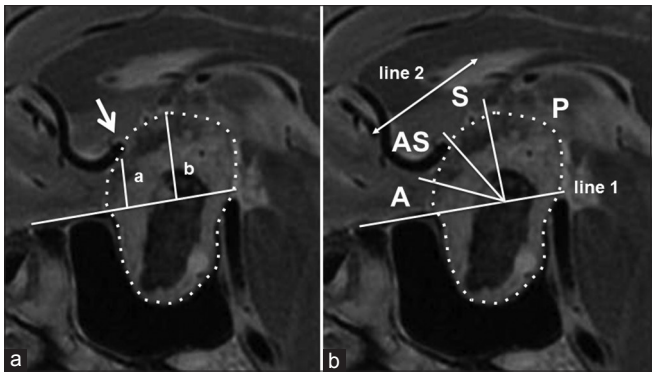

Figure 1: (a) T2-weighted magnetic resonance image of the sagittal section, including the AComA, which is remarkably elevated by the pituitary macroadenoma together with the optic chiasm. Line a indicates the height of the AComA with respect to the planum sphenoidale, and line b indicates the maximum height of the portion above the planum sphenoidale in the pituitary macroadenoma. (b) Pituitary macroadenomas were divided into four groups in this study based on the position of the AComA in relation to the pituitary macroadenoma. (a) Position was determined using the crossing angle between line 1 and line 2 in the adenoma on the sagittal section of the MR image that imaged the AComA. Line 1 indicates the extension of the anterior skull base from the planum sphenoidale. Point C is the midpoint of line a in the adenoma. Line 2 connects the inferior margin of the AComA with Point C. (b) The positions of the AComA on the macroadenoma, which were determined by the angle between line 1 and line 2 in each group, were as follows; Group A; 0–30°, Group AS; 31–60°, Group S; 61–90°, Group P; 91–180°

RESULTS

Patient characteristics

Among the 102 patients included in the analysis, a flow void in the AComA was observed on T2-weighted MR images both on coronal and sagittal sections before and 1 week and 3 months after TSS. Cerebrovascular anomalies combined with pituitary adenomas in this analysis included four unruptured aneurysms (3.8%) located at the AComA in 1 patient, the internal carotid artery (ICA) in 2 patients, and the middle cerebral artery (MCA) in 1 patient. One patient with an aneurysm at the ICA was treated preoperatively, however, the other 3 patients were not treated for aneurysms before TSS and were observed as outpatients after TSS.

In all the patients, the mean AComA descent was 4.4 ± 3.5 mm at 1 week after TSS and 5.1 ± 3.8 mm at 3 months after TSS. The associations of AComA descent with sex and age (≤60 or >60 years) were evaluated. No significant difference in AComA descent at 1 week after TSS was found between the patients aged ≤60 years (56 patients, 4.9 ± 3.7 mm) and those aged >60 years (46 patients, 3.8 ± 3.9 mm; P = 0.072). In 51 male patients, the AComA descent at 1 week after TSS was 4.6 ± 3.1 mm, which was not statistically different from that in 51 female patients, whose AComA descent was 4.1 ± 3.8 mm (P = 0.245). Three arteriosclerotic risk factors, namely hypertension, diabetes mellitus, and hyperlipidemia, did not affect the AComA descent at 1 week after TSS. The AComA

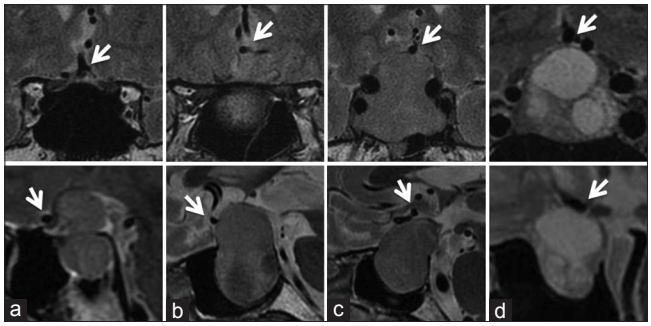

Figure 2: Representative cases of each group, as classified by the positions of the AComA in relation to the pituitary adenoma both on coronal and sagittal sections of T2-weighted MR images. (a) Group A, (b) Group AS, (c) Group S, and (d) Group P

descent was 3.7 ± 2.5 mm in the patients with hypertension (38 patients) and 4.8 ± 3.9 mm in the patients without hypertension (64 patients; $P = 0.346$). The AComA descent was 4.8 ± 4.1 mm in the patients with diabetes mellitus (23 patients) and 4.2 ± 3.3 mm in the patients without diabetes mellitus (79 patients; $P = 0.651$). The AComA descent was 5.0 ± 3.8 mm in the patients with hyperlipidemia (34 patients) and 4.1 ± 3.3 mm in the patients without hyperlipidemia (68 patients; $P = 0.157$) [Table 1].

Preoperative heights of the AComA and pituitary adenoma in each group

AComA descent was assessed according to its position in relation to the adenoma. The patients in this study were divided into four groups, as described in the Materials and Methods section. First, the preoperative heights of the AComA were measured in each group. The height of the AComA on T2-weighted MR images on sagittal sections was most significant in group S (9.5 ± 2.9 mm), followed by group AS (7.4 ± 3.5 mm), group P (6.3 ± 2.9 mm), and group A (3.5 ± 2.6 mm; $P < 0.0001$). The preoperative maximum height of this macroadenoma above the planum sphenoidale was also measured in each group. The difference in height among the groups were not significant ($P = 0.278$) as follows: group S (8.2 ± 3.7 mm), group AS (11.6 ± 4.3 mm), group P (8.1 ± 4.4 mm), and group A (12.3 ± 8.2 mm) [Table 2].

Sequential changes in AComA descent after TSS in each group

The AComA descent in each group was assessed at 1 week and 3 months after TSS. The results obtained at 1 week after TSS were most significant ($P = 0.0001$) in group S (6.5 ± 3.7 mm), followed by group AS (4.1 ± 2.5 mm), group P (3.7 ± 2.7 mm), and group A (1.0 ± 2.0 mm). Furthermore, the AComA descents in these four groups were significantly different [Table 2]. This result implies that the higher the position of the AComA in relation to the adenoma, the larger its descent within 1 week after TSS.

The percentages of the AComA descent at 1 week after TSS to that at 3 months after TSS in each group were compared. Consequently, AComA descent was found to be the most significant at 1 week after TSS in all the groups as follows: A (78.6%), S (89.1%), *P* (88.1%), and AS (83.7%). Among the groups, no statistically significant difference was found in the percentage of AComA descent at 1 week or 3 months after TSS. This result shows that the AComA descent after TSS can occur as quickly as within 1 week after TSS, regardless of the position of the AComA in relation to the adenoma [Table 3]. The correlation between postoperative AComA descent and residual adenomas on the midline was most significant in group S both at 1 week ($P = 0.030$) and 3 months after TSS ($P = 0.007$). The correlation was significant in group AS at 3 months after TSS ($P = 0.018$), but not at 1 week after TSS ($P = 0.068$). However, the correlation was not significant in groups P and A.

Table 1: Correlations of the AComA descents with the factors about the patients' demographic features

Variables	Numbers	AcomA descent (mm)	*P*
Age (yrs)			
60 and less	56	4.9±3.7	0.072
60 over	46	3.8±3.9	
Sex			
Male	51	4.6±3.1	0.2451
Female	51	4.1±3.8	
HT			
W	38	3.7±2.5	0.3465
W/o	64	4.8±3.9	
DM			
W	23	4.8±4.1	0.651
W/o	79	4.2±3.3	
HL			
W	34	5.0±3.8	0.1567
W/o	68	4.1±3.3	

Table 2: Preoperative heights of AComA and pituitary adenoma with the classification of AComA positions on pituitary adenomas

Group	Numbers	Heights of AcomA (mm)	Heights of Adenoma (mm)
A	18	3.5±2.6	12.3±8.2
AS	30	7.4±3.5	11.6±4.3
S	46	9.5±2.9	8.2±3.7
P	8	6.3±2.9	8.1±4.4
Total	102	7.5±4.3	11.4±6.8
P		< 0.0001	0.2788

Table 3a: Sequential changes of the AComA descents after transsphenoidal surgery in each group

Group	Numbers	1w (%) (mm)	3m (mm)
A	18	1.0±2.0 (78.6)	1.4±2.4
AS	30	4.1±2.5 (83.7)	4.9±2.9
S	46	6.5±3.7 (89.1)	7.3±4.0
P	8	3.7±2.7 (88.1)	4.2±2.9
Total	102	4.4±3.5 (86.2)	5.1±3.8
P		< 0.0001	<0.0001

AComA descents with or without intratumoral hemorrhage and sphenoid sinus extension in each group

AComA descent was compared between the groups "with or without intratumoral hemorrhage" and those "with or without sphenoid sinus extension of the adenoma" at 1 week after TSS. AComA descent was

not significantly different (P = 0.983) between the patients with (n = 27, 4.4 ± 3.4 mm) and those without intratumoral hemorrhage (n = 75, 4.4 ± 3.5 mm). In addition, AComA descent was not significantly different (P = 0.833) between the patients with (n = 39, 4.5 ± 3.9 mm) and those without sphenoid sinus extension (n = 63, 4.3 ± 3.2 mm) [Table 4]. These results indicate that AComA descent is dependent on the position, regardless of intratumoral hemorrhage and sphenoid sinus extension of the adenoma.

Representative case

A 65-year-old woman who presented with visual field defect consulted a local hospital for further examination. MR imaging revealed a pituitary macroadenoma extending to the suprasellar region. However, three-dimensional computed tomographic angiography (3-D CTA) revealed a small, unruptured AComA aneurysm, whose height was 13.7 mm from the planum sphenoidale. As the AComA aneurysm was small, we did not think the aneurysm would have considerable potential for intraoperative rupture. Therefore, we only performed TSS for this macroadenoma, resulting in subtotal removal. The patient's clinical course during and after TSS was uneventful. Although the postoperative 3-D CTA, obtained 1 week after TSS, revealed that the size of the aneurysm was unchanged; the height of the AComA was 5.6 mm. The AComA descent was 8.1 mm, and the running course of both A1 had changed remarkably, thereby suggesting that the hemodynamic stress to the AComA aneurysm had changed. On the MR angiographic follow-up of the aneurysm once a year, no enlargement was detected. In our analyses, this aneurysm belonged to group S and had a high risk of AComA descent. This case prompted us to investigate the risk of AComA descent in other patients [Figure 3].

Table 3b: Correlation between the postoperative AComA descents and the heights of residual adenomas on the midline

Group	Numbers	1w	3m
A	18	0.6763	0.4542
AS	30	0.0675	0.0176*
S	46	0.0299*	0.0066**
P	8	*	*
Total	102	0.004	0.003

Table 4: Postoperative AComA descents in aspects of with or without intratumoral hemorrhage and sphenoid sinus extension in each group

Variables	Numbers	AcomA descent	*P*
Intratumoral hemorrhage			
W/	27	4.4±3.4	0.9829
W/o	75	4.4±3.5	
Sphenoid sinus extension			
W/	39	4.5±3.9	0.8331
W/o	63	4.3±3.2	

DISCUSSION

In this study, the contributing factors to the process of AComA descent after TSS, such as the position of the AComA on the pituitary adenoma, massive intratumoral hemorrhage, and extension of the adenoma to the sphenoid sinus. The descent of the AComA was demonstrated to be statistically different based on the AComA position in relation to macroadenoma. Specifically, a higher AComA position might to be the strongest factor of AComA descent after TSS for macroadenoma.

Because AComA descent after TSS is considered a vascular-related event, it is thought to be influenced by atherosclerosis-related factors such as the patients' sex, age, and vascular risk factors (e.g., hypertension, diabetes mellitus, and hyperlipidemia). However, no significant difference in AComA descent was observed.

If pituitary macroadenoma coexists with some vascular anomalies such as cerebral aneurysms or arteriovenous malformations in the AComA, vascular injuries could be induced by the traction of AComA descent into the tumor cavity along with the abrupt collapse of the adenoma during or after TSS.[9,13] Such vascular injuries are hypothesized to be caused by the dissection of the adhesion between the adenoma and vasculature. This change in the hemodynamic stress on the vasculature

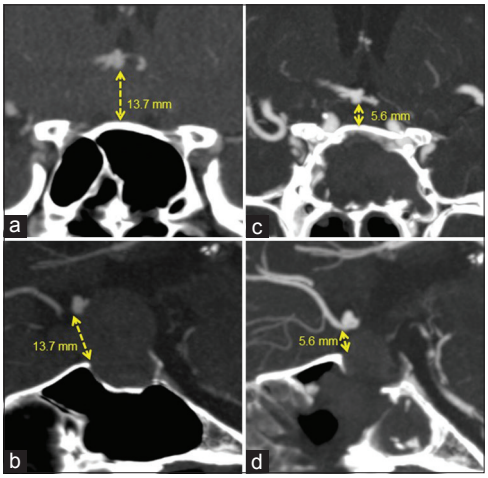

Figure 3: A 65-year-old woman presented with a visual field defect. MR imaging revealed a pituitary macroadenoma that extended to the suprasellar region. A 3-D CTA revealed a small, unruptured AComA aneurysm, with the height of the aneurysmg being 13.7 mm from the planum sphenoidale. The AComA aneurysm was detected preoperatively (a: Coronal section; b: Sagittal section). The AComA descent was 8.1 mm during the first week after TSS. A 3-D CTA revealed not only significant descent of the AComA, but also a change in the course of both A1 and A2, thereby suggesting an alteration of the hemodynamic stress in the aneurysm (c: Coronal section; d: Sagittal section)

could be induced by its decompression after removal of the adenoma or due to changes in the course of the parent artery such as A1.[9,13] In fact, many authors have reported the coexistence between pituitary adenoma and cerebral aneurysms.[3,4,7,15] Wakai *et al*. indicated that the incidence of such coexistence was significantly higher than that of other brain tumors (7.4% vs. 1.1%).[15] In such cases, predicted AComA descent after TSS could be a rough indicator of the risk of occurrence of these vascular events.

Tsuchida *et al*. reported a case of a ruptured AComA aneurysm during TSS for pituitary adenoma.[13] The preoperative angiogram of the right ICA showed remarkable elevation of both sides of the A1 portion, and the subsequent angiogram obtained 3 weeks after TSS showed an apparent AComA descent. An AComA positioned in relation to the pituitary adenoma was assumed to belong to either group S or AS, which has the potential to induce remarkable AComA descent intraoperatively or postoperatively. The authors speculated that the mechanism of the rupture of the aneurysm involved the intraoperative abrupt collapse of the tumor capsule after removal of the adenoma, thereby resulting in AComA descent into the tumor cavity.

As shown in Figure 3, the case involving AComA colocalization with the pituitary macroadenoma (such that it was classified into group S) showed significant AComA descent 1 week after TSS. The preoperative and postoperative 3-D CTA clearly demonstrated changes in the course of A1, thereby suggesting changes in hemodynamic stress, which can harbor the potential of aneurysm rupture. Fortunately, the postoperative clinical course of our patient was uneventful, and the aneurysm was observed on the MR angiography performed annually.

To the best of our knowledge, no previous reports resemble our analysis or have examined AComA descent after TSS. In our study, the AComA descent in all the groups occurred as early as within 1 week after TSS, regardless of the position of the AComA in relation to the adenoma. These results suggest that AComA descent probably is initiated immediately after the removal of the adenoma. As described earlier, if pituitary macroadenoma coexists with some vascular anomalies in the AComA, its descent could lead to intraoperative and postoperative immediate changes of the hemodynamic stress on the vasculature of the anomalies after its decompression by removal of the adenoma or due to changes in the course of the parent artery. According to our results, reevaluation of the size or shapes of vascular anomalies should be performed as early as possible postoperatively; otherwise, the patients might be exposed to subsequent vascular events, including rupture or embolism, while not being unaware of it. In addition, a second TSS might be required for residual adenoma. The position of the AComA in groups S and AS should be monitored carefully because it would change immediately after the first TSS. In conclusion, pituitary neurosurgeons should take notice of the preoperative position of the AComA in relation to the macroadenoma to avoid possible vascular injury.

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Conflicts of interest
There are no conflicts of interest.

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