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LARYNGOLOGY

New approach to diagnose arytenoid dislocation and subluxation using three-dimensional computed tomography

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Abstract Understanding the complex three-dimensional (3D) arrangement of the arytenoid cartilage is necessary for diagnosing arytenoid dislocation (AD) and arytenoid subluxation (AS). We examined the 3D arrangements of AD and AS (AD/AS) cases by region and considered their new diagnoses. This retrospective study included 2 patients with AD, 10 with AS, and 23 with unilateral vocal fold paralysis (UVFP) for comparison. The etiologies were intubation-induced and idiopathic. We classified the AD/AS position into four joint regions: mediocaudal, laterocaudal, mediocranial, and laterocranial. We generated 3D computed tomography (3DCT) images during rest and phonation to analyze functional movements. We attempted to compare the endoscopic findings and 3DCT images of patients with UVFP and AD/AS. To examine the joint status, we especially focused on the position and movements of the muscular process (MP) on the joint because the arytenoid facet is mainly located on the back of the MP. We were able to obtain endoscopic and 3DCT findings characteristic of each AD/AS region. The dislocated MPs were localized to the mediocaudal, mediocranial, and laterocranial regions. Two AD cases were diagnosed due to complete separation of the joint surfaces during rest and phonation. The finding of MPs displacing partially outside the cricoid facet is common to both severe UVFP and AS. The most important differentiation point was that the MP in UVFP cases was located on both the medial and lateral side regions of the joint, but that of AS was on one side region only. Furthermore, no cases of passive gliding movements characteristic of UVFP that have been described previously by us were observed in AD/AS cases. AD can be diagnosed by findings of complete joint separation. AS can be diagnosed based on positions and movements distinct from those of UVFP.

Keywords Arytenoid dislocation · Arytenoid subluxation three-dimensional computed tomography · 3DCT · Larynx

Introduction

Arytenoid dislocation (AD) and arytenoid subluxation (AS) are rare disorders that occur mainly after external or internal injury (particularly post-intubation) to the larynx. Hoarseness, swallowing disorders, and pharyngeal pain emerge, and timely reduction is necessary. An accurate diagnosis involves the correct differentiation of AD and AS (AD/AS) from unilateral vocal fold paralysis (UVFP), but doing so is difficult based on endoscopic observation alone.

Many studies have reported on the diagnosis of AD or AS [1–6]. Among the representative tests performed are endoscopic observation, laryngeal electromyogram, passive mobility test, and computed tomography (CT). Although electromyograms are important for a conclusive diagnosis, they cannot identify the region of the cricoarytenoid joint (CAJ) in which an AD or AS has occurred. A diagnosis of the AD or AS position provides crucial information for determining the method of the closed reduction. Furthermore, a case in which AD and paralysis occur concurrently can only be diagnosed as UVFP when analyzed by electromyography and endoscopy. Reports using CT [1, 2, 5] have focused on the asymmetry of

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bilateral arytenoid cartilages, but because the asymmetry is also observed in UVFP [7–10], one must show the difference between AD/AS and UVFP. In brief, no report to date has clearly demonstrated the optimal strategy for diagnosing AD or AS.

Imaging of cartilage is now possible using three-dimensional (3D) CT [7–10], and we have previously reported the movements and characteristic 3D arrangements of the arytenoid cartilages in UVFP using 3DCT [10]. In addition, we can see the joint status of patients with AD/AS.

The term AD is appropriately used when complete separation of the joint surfaces occurs, whereas AS implies some contact between joint surfaces. Therefore, AD can be diagnosed with a complete separation of the joint surfaces on 3DCT during rest and phonation. However, the CAJ capsule is large, and our research has revealed that in cases of severe UVFP, the paralyzed arytenoid cartilage is displaced far from the joint surface in the cranial direction [9, 10]. As the finding of arytenoid cartilage displacing partially outside the cricoid facet is common to both severe UVFP [9, 10] and AS, the diagnosis of AS requires more detailed observations.

In this study, we were able to diagnose patients with AS because they exhibited arrangements and movement patterns that differed from those of patients with UVFP that we had previously researched. We retrospectively analyzed the difference in the 3D arrangements of the arytenoid cartilage between AD/AS and UVFP, and suggest a new approach to diagnose AD/AS using 3DCT.

Materials and methods

Patients

The subjects were patients who had been diagnosed with unilateral AD or AS among cases of unilateral vocal fold immobility for which 3DCT was performed between April 2005 and May 2008. Cases after external injuries to the larynx were excluded. The present study included two male patients with AD and ten patients with AS (4 women, 6 men). In four patients, electromyograms (Neuropackμ MEB-9102; Nihon Kohden, Tokyo, Japan) were also performed. Their ages ranged from 39 to 88 years (mean 65 years). This 3DCT study included eight cases of intubation and four idiopathic cases.

We present 23 patients with UVFP whom we had previously researched [10] for comparison with the patients with AS. Their ages ranged from 44 to 83 years (mean 67 years). These patients with UVFP were diagnosed using intraoperative arytenoid adduction and electromyography findings with paralysis (electromyography of the thyroarytenoid muscle was performed from the thyroplasty type I window during surgery). We also found normal movement of the arytenoid cartilage because we experienced one patient who recovered from UVFP.

3DCT images

CT was performed in the horizontal plane using a multi-slice CT scanner (Sensation Cardiac 64; Siemens, Munich, Germany). Scans were made during rest and phonation. During phonation, each subject was asked to sustain the vowel /e/. Data were transferred using the Dicom system. The Mimics ver. 11.0 software (Materialise, Leuven, Belgium) was used to create 3DCT images through surface-rendering protocols using Windows XP (Microsoft, Redmond, WA, USA). Our previous study concerned the characterization of thresholds for arytenoids and cricoids [10]. The laryngeal lumen was visualized using a 3D endoscopy procedure [11].

Analysis

AD can be diagnosed by the finding of a complete separation of the joint surface on 3DCT. AS should be diagnosed not only by arytenoid cartilage displacement but also by movement.

The primary goal in diagnosing AD or AS is the clear definition of its position. In past reports, the terms “anterior,” “posterior,” “medial,” and “lateral” have been used to refer to the position of an AD or AS. However, these terms do not indicate the position in the *vertical* direction. We divided the cricoid facet into four areas: mediocaudal, laterocaudal, mediocranial, and laterocranial, plus an area outside of the cricoid facet. We classified the AD/AS position into four regions. One region consisted of an area of the cricoid facet, plus an area outside of the cricoid facet (Fig. 1).

At first, we focused on the position of the muscular process (MP) to determine the relationship with the cricoid facet because the main portion of the arytenoid facet is located on the back of the MP. We present one normal case to help describe the MP displacement analysis.

The patient was a 48-year-old man who recovered from UVFP (left side) 3 months after onset. After recovery, a normal rocking motion was observed, located symmetrically on both sides (Fig. 2a, oral view; Fig. 2b, frontal view). During rest, the arytenoid facet (right side) widely contacted with the cricoid facet (Fig. 2c; areas 1, 2, 3, and 4). When we focused on the part of the MP during rest, it was similarly located in areas 1, 2, 3, and 4, and during phonation, it was located in areas 1 and 3. We selected the cricoid facet area in which the MP mainly exists to analyze the difference in the position of the affected MP between patients with AS and those with UVFP. In this normal case,

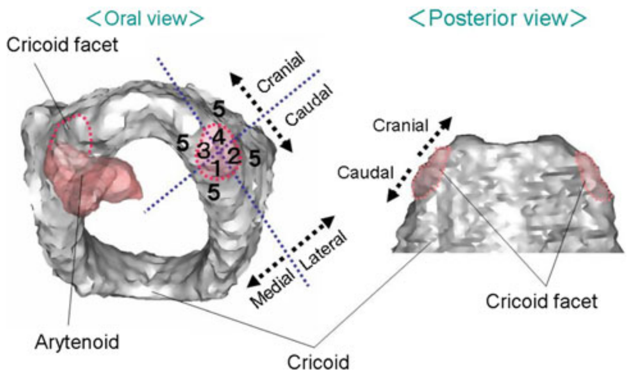

Fig. 1 The cricoid facet areas. The cricoid facet, viewed on 3DCT, has a cylindrical dimension, as shown by the *dotted lines* in the figure. This cricoid facet is identifiable as protruding in relation to its surroundings. To define the positions of the AD/AS, we classified the AD/AS position into four regions. One region consisted of an area of the cricoid facet, plus an area outside of the cricoid facet as follows: areas *1* and *5* mediocaudal region, areas *2* and *5* laterocaudal region, areas *3* and *5* mediocranial region, area *4* and *5* laterocranial region

the MP moved primarily from area 2 during rest to mainly area 1 during phonation.

Next, we performed 3DCT under conditions of rest and phonation and detected changes in MP position as movements. Previously, we reported that in UVFP, the paralyzed arytenoid showed a passive gliding movement, which displaced in the cranial direction during phonation. This passive gliding movement was observed in 49/54 (90.7%) patients, and we concluded that this movement was characteristic of UVFP [10]. In the present study, we investigated whether this movement could be observed in patients with AD/AS, and whether the movement was useful for distinguishing AD/AS from UVFP.

We also focused on the position of the vocal process (VP) to clarify vocal fold position (visualization of the vocal process on 3DCT is usually limited to its base because of the elastic cartilage), and the apex of the arytenoid cartilage to determine the arytenoid hump position. From these findings, we attempted to compare endoscopic findings and 3DCT images of the arytenoid cartilage.

We used maximum phonation time (MPT) and mean flow rate (MFR) to evaluate the aerodynamic function of each position in AD/AS cases; MFR was measured in the most comfortable condition using a phonation analyzer (PS-77; Nagashima, Tokyo, Japan).

Results

We classified AD/AS cases based on the MP position (Table 1). Two AD cases (mediocaudal, case 4; mediocranial, case 1) were diagnosed due to complete separation of the joint surfaces during rest and phonation. We observed

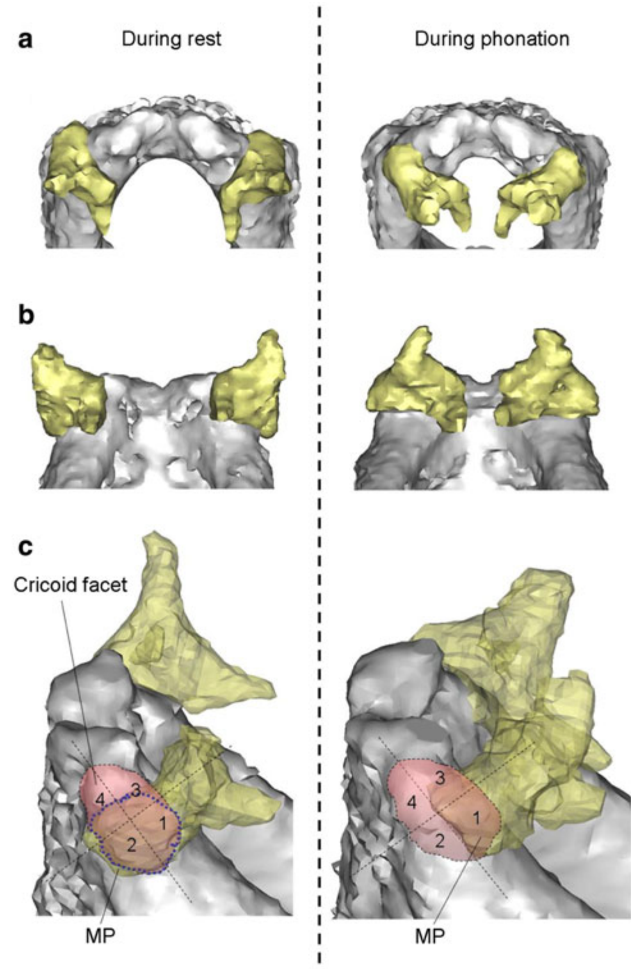

Fig. 2 Normal case. *MP* muscular process. *Yellow coloration* healthy arytenoid cartilage. **b** shows a three-dimensional image processed to make the arytenoid cartilage radiolucent, allowing cricoid facet to be visualized. The area surrounded by blue *dotted line*, The area in which the arytenoid facet contacts with the cricoid facet

three AS locations: the mediocaudal (5 cases), mediocranial (4 cases), and laterocranial (1 case) regions. We did not observe AD/AS in the laterocaudal region. Table 1 shows the patient profiles, MPT, and MFR based on the AD/AS region.

Table 2 shows the position of the MP (we selected one or two cricoid facet areas in which the affected MP mainly exists: asterisk) of UVFP and AS cases during rest and phonation. The cricoid facet areas in which the MP existed were different in AS and UVFP cases. Importantly, during rest and phonation, the affected side MP of UVFP cases was located in both medial and lateral areas, whereas the dislocated side MP of AS cases was located in either the medial or lateral area. Additionally, the dislocated side showed no passive gliding movement in any patient with AD/AS.

Table 1 Subject profiles, MPT, and MFR, divided by region

No./sex/age	Side	Etiology/period from onset	Diagnosis	MPT (S)	MFR (ml/s)	Treatment/outcome
Mediocaudal region						
1/M/76	L	Intubation/6 months	Subluxation	6	360	Thyroplasty type 1/improved
2/M/76	R	Intubation/6 years	Subluxation	15	–	None/no f/u
3*/F/88	L	Intubation/2 weeks	Subluxation	26.5	82.9	Closed reduction/unchanged
4/M/68	R	Intubation/4 months	Dislocation	17	93.3	None/no f/u
5/M/72	L	Intubation/6 years	Subluxation	15	–	None/unchanged
6/F/52	R	Intubation/17 years	Subluxation	12	199.8	None/unchanged
Mediocranial region						
1*/M/66	L	Idiopathic/3 months	Dislocation	3	595.1	Closed reduction/unchanged
						Thyroplasty type 1/improved
2*/F/46	L	Idiopathic/3 months	Subluxation	15	267.5	None/no f/u
3/M/64	L	Intubation/4 months	Subluxation	4.8	587.7	None/spontaneous reduction
4/M/80	L	Idiopathic/5 months	Subluxation	2	1,131.5	None/no f/u
5*/M/39	R	Idiopathic/13 months	Subluxation	4.5	450.7	Closed reduction/unchanged
						Thyroplasty type 1/improved
Laterocranial region						
1/F/62	L	Intubation/1 months	Subluxation	3	–	None/spontaneous reduction

L left, *R* right, *MPT* maximum phonation time, *MFR* mean flow rate, *no f/u* patient lost to follow-up

* Laryngeal electromyography was performed

Table 2 The areas which the MP exists in UVFP and AS cases

	UVFP ($n = 23$)	Mediocaudal AS ($n = 5$)	Mediocranial AS ($n = 4$)	Laterocranial AS ($n = 1$)
Healthy side				
Rest	1,2*,3,4	1,2*,3,4	1,2*,3,4	1,2*,3,4
Phonation	1 (3/23)	1*,3 (1/5)		
	1*,3 (20/23)	1,3* (2/5)	1*,3 (4/4)	1*,3 (1/1)
		1*,3* (2/5)		
Affected side				
Rest	3*,4 (4/23)			
	3*,4* (3/23)			
	3*,4,5 (3/23)	1,5 (2/5)		
	1,3*,4 (6/23)	1*,3,5 (3/5)	3,5 (4/4)	4,5 (1/1)
	1,2,3*,4 (3/23)			
	1,2,3*,4* (4/23)			
Phonation	3*,4 (1/23)			
	3*,4* (1/23)	1,5 (2/5)		
	3*,4,5 (12/23)	1*,3,5 (3/5)	3,5 (4/4)	4,5 (1/1)
	3*,4*,5 (9/23)			

Number indicates area number, *MP* muscular process

* We selected one or two cricoid facet areas in which the affected MP mainly exists

We present representative examples and variations (Table 3) of the endoscopic and 3D findings for UVFP and AD/AS of each region. First, we present UVFP cases to compare the displacements and movements of the affected side arytenoid cartilages, including endoscopic findings in UVFP cases compared to those with AD/AS.

UVFP (Case 2)

We present a case of severe UVFP with a MFR of >1,000 mL/s (Fig. 3). This patient was an 80-year-old man who suffered paralysis following surgery for an aortic aneurysm.

Table 3 The endoscopic and 3DCT findings of the affected side for each type of AD/AS and UVFP

	VP position (rest)	Shape of VF (rest)	Arytenoid hump position (phonation)	Arytenoid apex position (phonation)	Higher side of VP (phonation)	Over-adduction (phonation)
UVFP	Median (6/23) Paramedian (11/23) Intermediate (6/23)	Bowing (21/23)	Posterior (19/23) Same level (4/23)	Posterior (23/23)	Affected side (23/23)	Healthy side (23/23)
Mediocaudal AD (AS) ($n = 6$)	Median (6/6)	Bowing (1/6) Straight (5/6)	**Anterior (3/6)** Same level (3/6)	**Anterior (6/6)**	**Healthy side (6/6)**	**Affected side (6/6)**
Mediocranial AD (AS) ($n = 5$)	Paramedian (1/5) Intermediate (4/5)	Bowing (4/5) Straight (1/5)	**Anterior (5/5)**	**Anterior (5/5)**	Same level (3/5) Healthy side (1/5) Affected side (1/5)	Healthy side (5/5)
Laterocranial AS ($n = 1$)	Lateral (1/1)	**Hyperextension (1/1)**	Posterior (1/1)	Posterior (1/1)	Affected side (1/1)	Healthy side (1/1)

The items shown in bold type are findings that are different from those of the UVFP cases

VF vocal fold, *VP* vocal process

In the endoscopic oral view (Fig. 3a), the arytenoid hump on the paralyzed side (left) was displaced anteriorly, and the vocal process (VP) was hidden during rest. Bowing of the vocal fold (VF) (Table 3) was observed. The paralyzed arytenoid hump was displaced posteriorly relative to the healthy arytenoid hump during phonation. The VF on the paralyzed side appeared to be immobile.

In the 3D endoscopic tracheal view (Fig. 3b), the paralyzed side VP was positioned medially during rest but was displaced laterally during phonation, and the glottal gap was large. Therefore the VF on the paralyzed side was actually not immobile. The healthy side VP exhibited over-adduction during phonation (it moved over the midline on the tracheal view).

The MP (Fig. 3c, d; Table 2) of the healthy side moved primarily from area 2 during rest to mainly area 1 during phonation. During rest, the paralyzed side MP was located in areas 3* and 4. During phonation, the MP glided cranially (posterior at the same time) relative to its position during rest (passive gliding movement) and was located in areas 3*, 4, and 5. In this case, the affected side MP was displaced far from the joint, and the cricoid facet was extremely exposed during phonation. However, the MP existed within the joint during rest.

In 23 UVFP cases, the position of the MP varied, but the MPs of the affected side were located in both medial and lateral areas and tended to exist in the cranial areas (areas 3 and 4) during rest and phonation (Table 2); the affected side showed a passive gliding movement in all patients.

During phonation, the apex of the paralyzed arytenoid cartilage was displaced in the cranial and posterior directions relative to the healthy side. The base of the paralyzed side VP was displaced cranially and laterally during phonation, and the higher side of the VP was the affected side.

Mediocaudal region AD/AS

Of six cases, one (case 4) had an AD and the others had ASs. All cases were intubation-induced. Five patients were initially misdiagnosed as having UVFP. Phonatory function in this group was satisfactory (Table 1). Representative examples are shown in Fig. 4 (case 6, a–d; case 1, e).

In the endoscopic oral view (Fig. 4a) during rest, the VP on the dislocated side (right) was positioned medially (Table 3) and the VF did not display bowing (straight). The glottal closure was satisfactory during phonation. The arytenoid hump (white asterisk) on the dislocated side was displaced anteriorly during phonation relative to that on the healthy side. When observed in close proximity during phonation, the VF on the dislocated side (arrow) was displaced caudally relative to the VF on the healthy side.

During rest and phonation in the tracheal view (Fig. 4b), the dislocated VP exhibited over-adduction (it had moved over the midline), and glottal closure was satisfactory during phonation.

The dislocated MP (Table 2) was in areas 1 and 5 during both rest and phonation and adducted more than the healthy side during phonation. The apex of the arytenoid was displaced slightly anteriorly to that of the healthy arytenoid during phonation. The bases of the VP and VF (Fig. 4d) on the dislocated side were lower than those on the healthy side during phonation (higher side of VP is the healthy side).

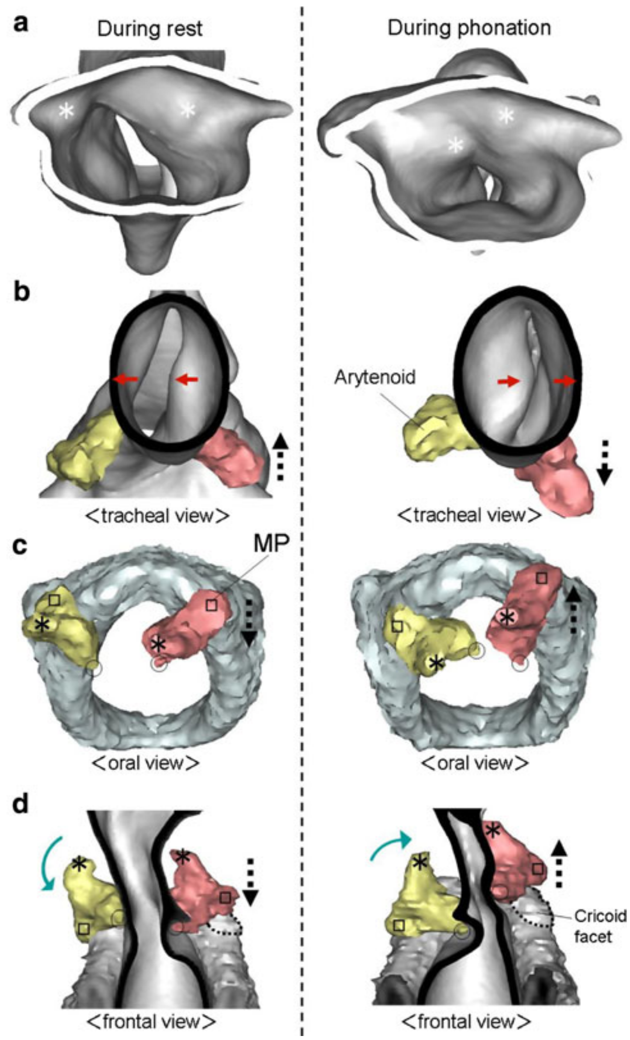

Fig. 3 UVFP case. *Yellow coloration* healthy arytenoid, *red coloration* paralyzed arytenoid. *White asterisks* arytenoid humps, *black asterisks* apices of the arytenoids. *Circle* base of the vocal process. *Square* muscular process. *Arrows* movements. **d** The cartilage and 3D endoscopic images were taken simultaneously

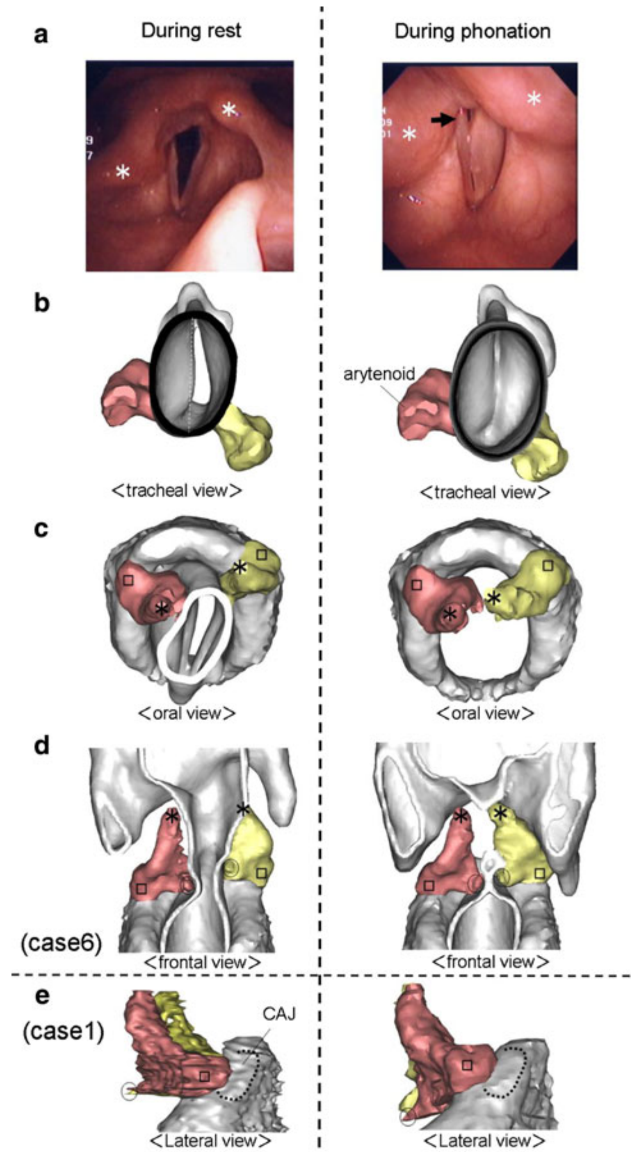

Fig. 4 Mediocaudal AS. *Yellow coloration* healthy arytenoid, *red coloration* dislocated arytenoid. *White asterisks* arytenoid hump, *black asterisks* apices of the arytenoids. *Circle* base of the vocal process. *Square* muscular process. **d** The cartilage and 3D endoscopic images were taken simultaneously

In two cases (cases 1 and 4) during phonation, the dislocated base of the VP was displaced farther medially and caudally than that observed during rest (Fig. 4d).

Mediocranial region AD (AD/AS)

Of five cases, one (case 1) had an AD and the others had ASs. Four were idiopathic cases and one was intubation-induced. Four patients were initially misdiagnosed as having UVFP, and mild-to-severe MFRs were observed. A representative example is shown in Fig. 5 (case 1, AD).

In the endoscopic oral view (Fig. 5a), the VP on the dislocated side (left) was at an intermediate position (Table 3) and the VF did not display bowing. During both rest and phonation, the arytenoid hump on the dislocated side was displaced anteriorly relative to that on the healthy side.

On the tracheal view (Fig. 5b), the VP on the dislocated side during phonation was displaced farther laterally from its position during rest (in all cases), and the glottal gap was large. The healthy VP exhibited over-adduction during phonation (dotted line, midline).

The apex (Fig. 5c) of the dislocated arytenoid (black asterisk) was displaced in the anterior and medial directions during both rest and phonation. On the frontal view (Fig. 5d), the apex of the dislocated arytenoid crossed the

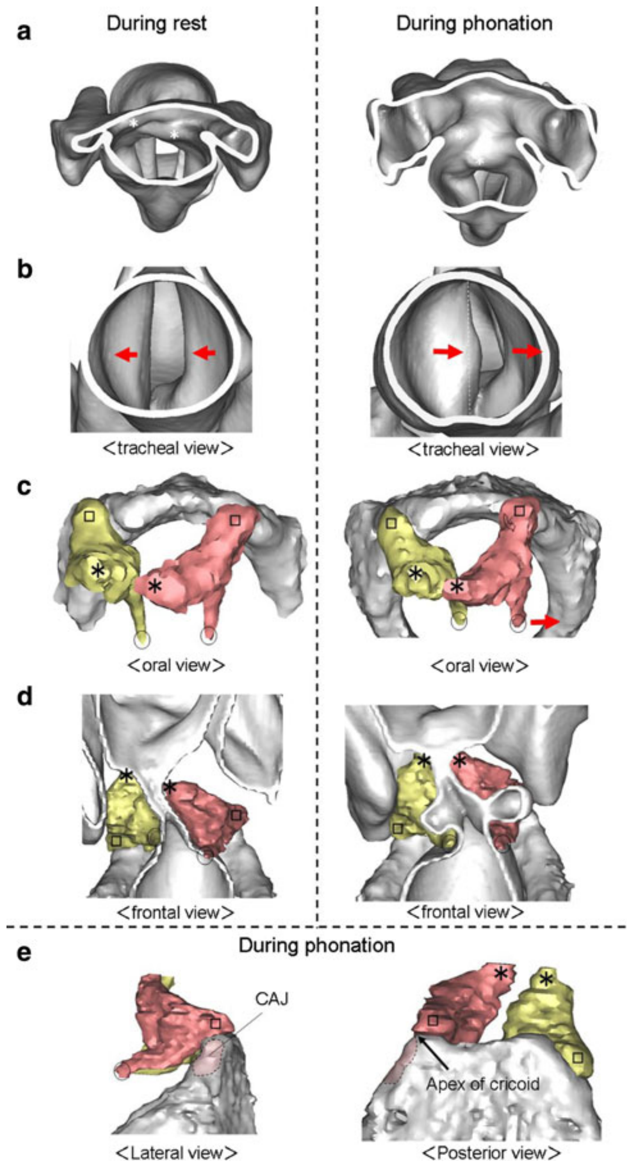

Fig. 5 Mediocranial AD. *Yellow coloration* healthy arytenoid, *red coloration* dislocated arytenoid. *White asterisks* arytenoid humps, *black asterisks* apices of the arytenoids. *Circle* base of the vocal process. *Square* the muscular process. *Arrows* movements. **d** The cartilage and 3D endoscopic images were taken simultaneously

midline (in all cases) during rest. During phonation, the MP rotated slightly (Fig. 5c) to the same position, and the apex of the dislocated arytenoid and the base of the VP shifted laterally relative to their positions during rest as the healthy side arytenoid shifted toward the midline and pushed the dislocated side.

The dislocated side MP was positioned in area 5 of the mediocranial region during both rest and phonation. The dislocated arytenoid (Fig. 5e) reached the apex of the cricoid, and the joint surfaces did not contact one another. When examined on frontal views, the dislocated MP was displaced medially, and the base of the VP was displaced caudally. Therefore, the positions of the base of VP (Fig. 5d, circle) on the dislocated and healthy sides during phonation were at the same level, despite the MP on the dislocated side being located cranially.

In case 3, we observed a sudden improvement, and a spontaneous reduction was confirmed with repeat 3DCT (both arytenoids were symmetrical during phonation). Soon afterward, the MPT improved from 4.8 to 9 s, and the MFR improved to the normal value (151.9 mL/s) by 6 months.

Laterocranial region AS

We observed one case of an AS in this region (Fig. 6). This case was intubation-induced, and the MFR was 890 mL/s.

In the endoscopic oral view (Fig. 6a) during rest, the VP on the dislocated side (left) was at a lateral position (Table 3) and the VF was hyperextended. The dislocated arytenoid hump was displaced posteriorly relative to that of the healthy side during phonation. The glottal gap was large during phonation.

On the tracheal view (Fig. 6b), the dislocated VF was opened wide during rest and was shifted somewhat medially during phonation. The healthy side VP exhibited over-adduction during phonation (dotted line, midline).

The MP of the dislocated side was positioned in areas 4 and 5 (Table 2) during rest and phonation. The apex of the dislocated arytenoid moved from a lateral position (during rest) to a medial position (during phonation). The apex on the dislocated side (Fig. 6c, asterisk) was positioned in the posterior direction relative to that on the healthy side during phonation.

On the frontal view (Fig. 6d), the base of the VP was located laterally and cranially relative to that on the healthy side (higher side of VP was the affected side) and the arytenoid facet surface was partly exposed during phonation (Fig. 6d). The AS became clear during rest due to dislocated side over-abduction, resulting in a cricoid facet that was largely exposed.

This patient improved suddenly on the 46th day (spontaneous reduction). Figure 6e shows the 3DCT data just after this improvement. The arytenoid cartilage was repositioned within the CAJ during rest and phonation. The bilateral arytenoids became symmetrical during rest and phonation (Fig. 6e). The MPT improved from 3 to 8 s immediately after spontaneous reduction and then increased gradually to 28 s on the 72nd day.

Discussion

Clearly defining the AD/AS region is important. The AD and AS were located in the mediocaudal, mediocranial, and laterocranial regions in this study.

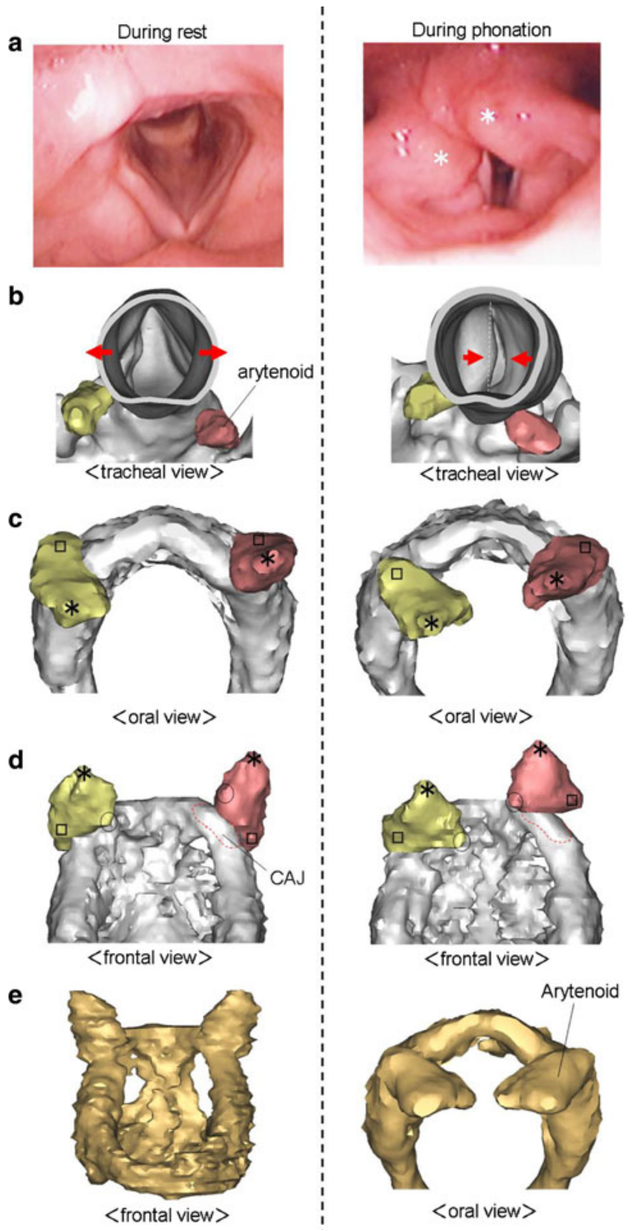

Fig. 6 Laterocranial AS. *Yellow coloration* healthy arytenoid, *red coloration* dislocated arytenoid. *White asterisks* arytenoid humps, *black asterisks* apices of the arytenoids. *Circle* base of the vocal process, *arrows* movements. *Square* muscular process

During diagnosis, the correct differentiation of AD/AS from UVFP is essential. AD can be diagnosed by the finding of a complete separation of the joint surfaces during rest and phonation, whereas AS can be diagnosed based on areas and findings distinct from those of UVFP. Therefore, we needed to compare the displacements and movements of the affected side arytenoid cartilages in UVFP and AD/AS cases.

The most important differentiation point (Table 2) is that during rest and phonation, the MP is located on both medial and lateral side areas in UVFP, but that of AS exists on one side area (medial or lateral area). Furthermore, we conclude that detection of the passive gliding movement [10] is important for the diagnosis of AS, as this movement was not observed in the patients with AS examined in the present study.

By examining the properties of the AD/AS by region, we were able to obtain endoscopic and 3DCT findings that were characteristic for each region, as follows. Moreover, on the basis of Table 2 findings, we present a simplified scheme (Fig. 7) of the MP displacement to help develop an understanding of the arrangements of AS and UVFP.

UVFP

The important points for diagnosing UVFP are as follows:

- In severe cases, the affected side MP was displaced partially outside the cricoid facet.
- The affected side MP existed in both medial and lateral areas (Table 2).
- The affected side MP tended to exist in the cranial area (areas 3 and 4) during rest and phonation.
- Passive gliding movement occurred.

The arytenoid cartilages are thought to come into contact with each other during phonation because the actual size of the arytenoid cartilage is bigger than the arytenoid cartilage imaged using 3DCT. In patients with UVFP, the paralyzed arytenoid cartilage undergoes cranial displacement, which is caused by contact with the healthy side (Fig. 7, dotted arrow) during phonation and as a result of expired air. We call this a passive gliding movement. Note that the healthy side experienced over-adduction during paralysis due to the lack of muscle tension on the opposite side [10].

In Fig. 7, the arrow shows the muscular vector acting on the MP, and X shows the paralysis condition. We believe that constriction of the interarytenoid muscle (IA), which is innervated by both recurrent laryngeal nerves, also pulls the paralyzed arytenoid in the direction of the healthy side; thus, it is displaced cranially (posteriorly at the same time) even further [10]. As a result, the apex of the paralyzed arytenoid cartilage and arytenoid hump was displaced in the posterior direction during phonation, relative to the healthy side.

Mediocaudal AD and AD/AS

AD/AS in this region has been reported as being anteromedial AD/AS. The AD case (case 4) could be diagnosed by the finding of a complete separation of the joint surfaces during rest and phonation. The important points for diagnosing AS are as follows:

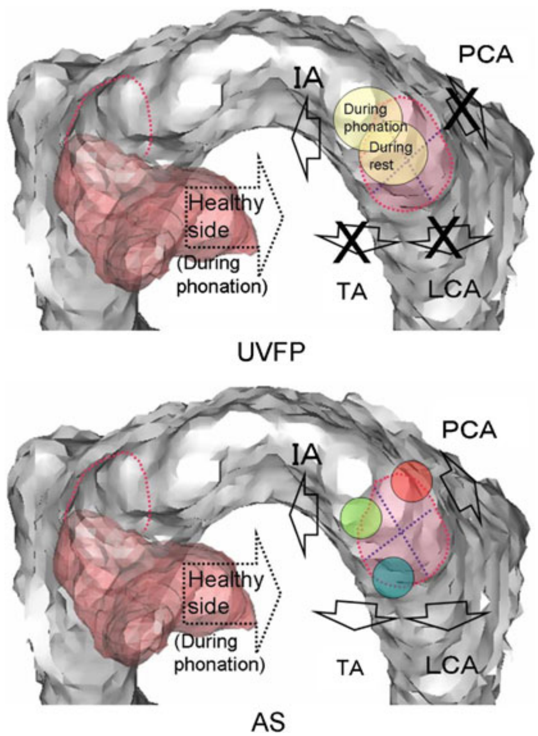

Fig. 7 Muscular process displacement for each condition (unilateral vocal fold paralysis vs. arytenoid subluxation). On the basis of the Table 2 findings, we present the MP displacement of UVFP and AS. *Yellow circle* MP position of UVFP. *Blue circle* MP position of mediocaudal AS. *Green circle* MP position of mediocranial AS. *Red circle* MP position of laterocranial AS. *TA* thyroarytenoid muscle, *LCA* lateral cricoarytenoid muscle, *PCA* posterior cricoarytenoid muscle, *IA* interarytenoid muscle. *Arrow* the muscular vector acting on the MP. *Dotted arrow* vector acting on the affected arytenoid by contacting the healthy side. *X* paralysis condition

- The dislocated side MP existed in medial and caudal areas during rest and phonation (Table 2).
- The dislocated arytenoid cartilage adducted more than the healthy side during phonation.
- No passive gliding movement occurred.
- Active movements were observed in two cases in this study.

If these were UVFP cases, the affected side cartilage would be displaced laterally by contact with the healthy side (Fig. 7, dotted arrow) during phonation, but the dislocated side MP in this group never existed in a lateral area (areas 2 and 4). Furthermore, the dislocated side adducted more than the healthy side and tended to exist in the caudal area, whereas in UVFP, the affected side MP tended to exist in the cranial area during rest and phonation.

As a result of this MP position, the apex of the arytenoid was displaced slightly anterior to that of the healthy side. Furthermore, the dislocated side VP was positioned medially and caudally during rest and phonation, and glottal closure and MFR (Table 1) were satisfactory compared to the other positions in AD/AS cases.

When case 6 was examined more closely using endoscopy, the VP (VF) of the dislocated side was displaced caudally compared to the healthy side. This is an important endoscopic observation when a mediocaudal AD/AS is suspected because in UVFP, paralyzed VPs are displaced cranially (Table 3).

The dislocated side arytenoid cartilage during phonation was displaced slightly mediocaudally in cases 1 and 4. This can be interpreted as being due to active movement. If this active movement is detected clearly, an electromyogram may not be required.

In this retrospective study, five patients in this group were misdiagnosed as having mild UVFP. In one case, a type I thyroplasty was performed and the others received no treatment after the AD/AS diagnosis because the phonatory functions were satisfactory. Therefore, an electromyogram was not performed because we lost them to follow-up.

Note that when combined with complete paralysis, repositioning of the AD or AS may worsen phonatory function because the MP will be displaced cranially and laterally. Thus, using an electromyogram is advisable to ensure an accurate assessment of concurrent paralysis before treatment.

Mediocranial AD (AD/AS)

AD/AS in this region has not been clearly defined in the past, but 3D analyses are useful to understand their complex arrangements. The AD case (case 1, Fig. 5) could be diagnosed by the finding of a complete separation of the joint surfaces during rest and phonation. The important points for a diagnosis of AS are as follows:

- The dislocated MP existed in medial and cranial areas during rest and phonation (Table 2).
- No passive gliding movement occurred.

Active movements were not observed because displacements in areas 3 and 5 on the dislocated side MP antagonized the posterior cricoarytenoid (PCA) and lateral cricoarytenoid muscle (LCA) (Fig. 7). However, we observed passive movements in which the dislocated side was pressed outward by the healthy arytenoid during phonation because the upper structure of the dislocated side was displaced across the midline during rest. Although the dislocated side was pressed outward by the healthy side during phonation, the MP was never displaced into lateral areas (areas 2 and 4), and the passive gliding movement seen in patients with UVFP was not observed. (The dislocated MP was slightly rotated at the same position.) Thus, an electromyogram is required for diagnosing concurrent paralysis.

In our previous study of UVFP, the case was more severe as the displacement of the paralyzed side MP in the cranial direction during phonation became greater. Generally, in severe UVFP cases, a large difference is seen at the vocal folds level. Although the cranial displacement of the dislocated side MP was large in mediocranial AD/AS, the base of the dislocated side VP was located at the same level as the healthy side during phonation in three cases and was displaced caudally compared to the healthy side in one case. This is because the direction of MP displacement was not only cranial but also medial.

The arytenoid apex shifts in the anterior direction during phonation because of this medial displacement. As a result, the dislocated side arytenoid hump is displaced in the anterior direction during phonation. This is an important endoscopic finding that is different from UVFP (Table 3). As a result of this anterior displacement, the AD/AS in the mediocaudal and mediocranial regions may be confused with those in the anteromedial region.

We observed four idiopathic cases (electromyograms were performed in 3 of 4 cases), without any prior history of external injuries or intubation. This is an intriguing result that warrants further analyses with more cases and an investigation of the cause.

Laterocranial AS

This type is known as posterolateral AD/AS. The important points for the diagnosis of AS are as follows:

- The MP existed in lateral and cranial areas during rest and phonation (Table 2).
- The dislocated arytenoid cartilage abducted more than the healthy side during rest.
- No passive gliding movement occurred.
- Active movement was observed in this case.

As a result of the MP position (areas 4 and 5), the apex of the paralyzed side and the arytenoid hump were displaced posteriorly relative to the healthy side during phonation. A large glottal gap and severe MFR were observed. These findings are observed in cases of UVFP. However, hyperextension of the VF on the dislocated side during rest is an important observation when AD/AS is suspected because it is not observed in cases of UVFP. This is an over-abduction that results from the PCA acting in the context of subluxation (Fig. 6d). In the case of UVFP, the paralyzed side does not exhibit over-abduction during rest.

Displacements in areas 4 and 5 on the dislocated side MP antagonize LCA and TA (Fig. 7). As a result of IA and PCA activity, the apex of the dislocated arytenoid moved from a lateral position (during rest) to a medial position (during phonation), and these movements were considered to be active (Fig. 6c). However, the dislocated side MP did not shift to the medial area during phonation because this movement occurred in the subluxation position.

An electromyogram was not performed in this patient because of spontaneous reduction before attempting a closed reduction; however, the bilateral arytenoids became symmetrical with repeated CT just after reduction. Thus, this patient obviously had AS.

Only one case of AS was seen at this position. In the future, more information about this dislocation can be found by the examination of an increased number of patients, such as those with concurrent paralysis.

We were able to obtain the endoscopic and 3DCT findings listed above, which were characteristic for each AD/AS region. However, VF bowing and the VP position are not specific to AD/AS because of the variability observed.

Primarily, we detected the corniculate and cuneiform cartilage as the arytenoid hump; left–right positional differences (asymmetry) in the arytenoid humps sometimes occur during phonation even in normal cases. In many cases, however, the left–right positional difference of the arytenoid humps during endoscopy indirectly reflects the relative positional relationship between the apices of both arytenoid cartilages. Therefore, the arytenoid hump is a useful marker (Table 3) when an AD/AS is suspected in endoscopic findings, although AD/AS should be diagnosed based on joint status determined by 3DCT.

Dividing the cricoid facet precisely into a medial area and a lateral area was difficult; however, in this study, the MP of the AS was clearly located on one side area (medial or lateral area). In addition, the passive gliding movement characteristic of UVFP is a useful finding. If this movement is detected, we can diagnose UVFP even when the cricoid facet is extremely exposed during phonation.

Table 3 presents the characteristics of each type of AD/AS and UVFP from the endoscopic and 3DCT findings. AD/AS in the same region have many findings in common. Based on these, we can estimate whether an AD or AS exists, and its position, even when 3D visualizations of the MP and cricoid facet are poor.

We observed a spontaneous reduction with repeat 3DCT and a return of normal phonatory function for AD/AS of the mediocranial and laterocranial regions. The MPT value doubled immediately in cases of spontaneous reduction. However, in neither case did normal phonatory function improve immediately after repositioning. This may have been due to impairment of adduction, which was caused, for example, by a hematoma [12] within the joint after AD/AS.

We did not observe AD/AS in the laterocaudal region in the present study. A possible reason is that this is a position for normal abduction, so it is less likely to receive a load in the lateral direction. However, the possibility also exists that AD can occur in any direction, as in, for example, extracapsular AD induced by an external injury.

Of the 12 patients with AD/AS, eight had been misdiagnosed with UVFP at the time of initial assessment. Eight patients were long-standing cases (3 months or more). Thus, the risk of missing the critical period for closed reduction warrants the use of early 3DCT screening.

Conclusions

AD can be diagnosed by the finding of a complete separation of the joint surfaces during rest and phonation. AS can be diagnosed based on areas and findings distinct from those of UVFP.

The endoscopic observations, 3DCT findings, and MFR severity may vary according to the AD/AS position. Depending on the AD/AS region and by understanding both the 3D arrangement of the arytenoid cartilage and the endoscopic findings, one can initially estimate whether an AD or AS exists, and its position, via the endoscopic findings.

Although a decision as to the presence or absence of concurrent paralysis may require electromyography, we believe AD/AS should be diagnosed based on joint status.

In this study, the MP of the AS was located on one side area (medial or lateral area) during rest and phonation, and no cases of passive gliding movements were observed. Therefore, if cases satisfy these two conditions, an AS is very likely. In the future, prospective studies with electromyography will contribute to the establishment of more detailed diagnostic methods.

Conflict of interest statement The authors declare that they have no conflict of interest.

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