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Posteroinferior septal defect due to vomeral malformation

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Abstract

Purpose Vomeral malformation may lead to a posteroinferior septal defect (PISD). It is usually found incidentally, without any characteristic symptoms. The purpose of this study was to evaluate its clinical implications.

Methods In this study, we included 18 patients with PISD after reviewing paranasal sinus computed tomography scans and medical records of 2655 patients. We evaluated the shape of the hard palate and measured the distances between the anterior nasal spine (A), the posterior end of the hard palate (P), the posterior point of the vomer fused with the palate (V), the lowest margin of the vomer at P (H), and the apex of the V-notch (N).

Results None of the PISD patients had a normal posterior nasal spine (PNS). Six patients lacked a PNS or had a mild depression (type 1 palate), and 12 had a V-notch (type 2 palate). The mean A–P, P–H, and P–V distances were 44.5 mm, 15.3 mm, and 12.4 mm, respectively. The average P–N distance in patients with type 2 palate was 7.3 mm. There were no statistically significant differences between the types of palates in A–P, P–H, or P–V distances. In patients with type 2 palate, there was a significant correlation between P–V and P–N distances ($r = 0.664$, $p = 0.019$).

Conclusions PISD due to vomeral malformations was identified in 0.7% of the cases in this study. None of the subjects had a normal PNS, which suggests that the development of the vomer is closely related to that of the hard palate.

Keywords Vomer · Palate · Malformation · Septum · Defect

Introduction

Congenital vomer defects (CVDs) were first reported by Morhi and Amatsu in 2000 [1]. Since then, many cases have been reported in English literature. Endoscopic examinations and computed tomography (CT) are used to confirm the defect of the posteroinferior nasal septum that coincides with the location of the vomer and does not have another cause such as trauma, infection, inflammation, irritation, granulomatous disease, or neoplasia [2]. In the past, CVDs were thought to be rare, but the widespread use of endoscopy and improved diagnostic techniques have proved these to be more common than previously thought [3, 4].

Because CVDs have no characteristic symptoms, most are found incidentally [1, 3]. These may be accompanied by a deviated nasal septum and compensatory hypertrophy of the posterior parts of the inferior turbinates; adenoid hypertrophy may also be observed [5–8]. It is thought that turbulent airflow in the nasopharynx due to the defect may cause otitis media with effusion [1]. No problems regarding velopharyngeal functions or structural abnormalities in the palatal or craniofacial region have been observed. However, in some cases, hypernasality and bifid uvula were present [3, 9].

Developmental deformities of the vomer have been studied in relation to cleft palate. In particular, Grzonka et al. reported that in 30 patients with symptomatic submucous cleft palate (SMCP) who had undergone surgery, the vomer was hypoplastic in all cases. In 97% of cases, the defect was not covered with mucosa and was open; in 7% of cases, it was covered with mucosa [10]. Ren et al. also reported that 91.7% of SMCP patients who had not undergone surgery had a vomer partially fused with the palate [11]. Therefore,

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a typical criterion should include the defects of the vomer as well as the classic triad of SMCP: a bifid uvula, a palpable V-notch of the posterior palate, and a midline diastasis of the palatal muscles. The abnormality of the vomer is found in almost all cases in patients with SMCP and is one of the important considerations in preoperative evaluation and operation.

We have questioned whether posteroinferior septal defect (PISD) due to vomeral malformation was more frequently encountered as an isolated and independent vomeral bone defect, which is reported as CVD, or one of the anatomic variations associated with asymptomatic SMCP. Because almost all patients diagnosed with cleft palate are found to have hypoplasia or anomalies of the vomer, it should be checked whether the deformities of the vomer, even in patients without symptoms related to velopharyngeal inadequacy (VPI), are related to the structural malformation of the hard palate. However, there have been no studies on the clinical implication of vomer deformities that are discovered incidentally.

The purpose of this study was to assess the incidence of PISD due to vomeral malformation using clinical paranasal sinus CT and nasal endoscopy and to investigate the variations of the surrounding anatomical structures. In particular, we investigated the shape of the subjects' posterior bony palates by applying the grading system used to describe the hard palates of SMCP patients.

Materials and methods

We retrospectively reviewed paranasal sinus CT and medical records from January 2017 to July 2018 of patients who visited the otorhinolaryngology department. All paranasal sinus CT images were acquired with 1.0 mm collimated slice width intervals without contrast enhancement. We examined the PISD, which is shown as separation of the nasal septum from the floor in front of the posterior border of the palate and choanae on coronal scans and shortening of the nasal septum in the posterior area on axial scans.

When the defect was identified, the shape of the hard palate was evaluated. According to the classification used by Sommerlad et al. and Mori et al., the type of the bony defect of the hard palate was 0 in the case of a normal posterior nasal spine, 1 in the case of an absent posterior nasal spine or mild depression, 2 in the case of a V-notch, and 3 in the case of a V-notch extended to the incisive foramen [12, 13]. In addition, the anatomic variations of the nasal septum and turbinates were examined. A defective area of the PISD extending from the choanae to the mid-portion of the middle turbinate was designated as type I; a partial defect in the caudal portion was designated as type II [3].

The anterior nasal spine was defined as A. The point at which the vomer separated from the palate was defined as V. The posterior end of the palate was defined as P. The lowest end of the vomer that meets the imaginary line perpendicular to the palate at point P was defined as H. The apex of the V-notch was defined as N. The distances between A–P (on the axial plane), V–P (on the sagittal plane), P–H (on the coronal plane), and N–P (on the axial plane) in types 2 and 3 were measured on the CT image using a picture archiving and communication system (Fig. 1).

The endoscopic findings of the PISD were confirmed by two authors (Y.W. Lee and Y.H. Yoon), who used endoscopy to examine the posterior area of the nasal septum and record the presence and type of any defect; the defect shape was classified as either wedge or oval. Furthermore, anatomical abnormalities of the uvula were verified and symptoms

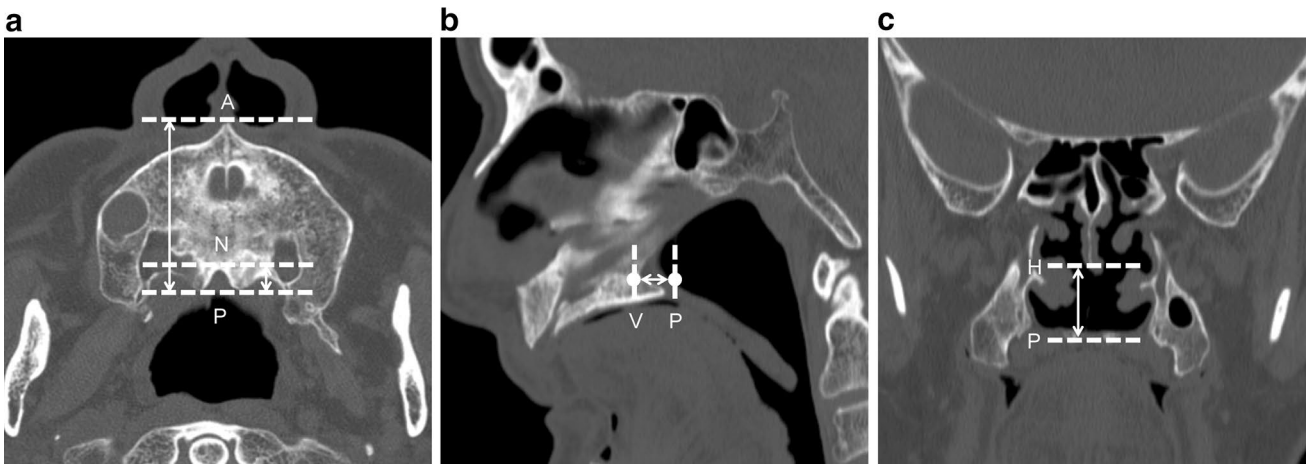

Fig. 1 Measurement between the assigned points. **a** Axial computed tomography scan showing distances from the anterior nasal spine (A) and apex of the V-notch (N) to the posterior end of the hard palate (P). **b** The distance from point P to the posterior end of the vomer fused with the palate (V). **c** The lowest end of the vomer (H) from point P was measured on the coronal scan

were checked, along with medical/surgical and familial histories. To exclude the association with SMCP as much as possible, patients who underwent cleft palate/lip surgery or who complained of nasal regurgitation, nasal emission, or hypernasality were not included in the study. Patients who underwent nasal septal surgery and may have been injured at the vomer were also excluded.

Statistical analysis was performed using SPSS 16.0 (Chicago, IL, USA). Statistical significance between groups was tested using the Mann–Whitney *U* test, and correlation was evaluated using Spearman's Rho test.

Results

We reviewed the paranasal sinus CT and medical records of 2655 patients and found 18 patients (0.7%) who could be included in this study (Table 1). The age of the patients (12 males and 6 females) ranged from 16–83 years (mean age, 62.9 years). Among them, two underwent endoscopic middle meatal antrostomy for fungal maxillary sinusitis, one underwent uvulopalatopharyngoplasty, and three had minor nasal-bone trauma that did not require surgical treatment. The others denied a history of facial surgery or trauma. No patient had a family history of cleft lip or palate.

Patients presented various symptoms such as nasal obstruction, rhinorrhea, posterior nasal drip, epistaxis, an itching sensation in the nose, coughing, sputum, oral pain, and a foreign-body sensation in the throat; no patients complained of otologic symptoms such as aural fullness. Two patients had a type I defect, and the others had type II. A wedge-shaped defect was observed in three patients (one type I, and two type II), and an oval defect was observed in 15 patients. None of the patients had a normal posterior nasal spine (type 0) in the posterior hard palate. Six patients had type 1 palates and 12 had type 2 palates (Figs. 2, 3). None of the patients had a type 3 palate.

Fifteen patients had a deviated nasal septum: five of the six type-1 patients and 10 of the 12 type-2 patients. In five patients, posterior bony spurs were observed (one of six with type 1, four of 12 with type 2). Hypertrophy of the inferior turbinate was observed in eight patients (three of six with type 1, five of 12 with type 2). Two patients had a bifid uvula (1 with each type of palate), and one patient who underwent uvulopalatopharyngoplasty had a type 2 palate. Type I defects and wedge-shaped defects were all found in patients with type 2 palates.

In all patients, the mean A–P distance was 44.5 mm, the P–H distance was 15.3 mm, and the P–V distance was 12.4 mm (Table 2). In patients with V-notch defects, the average value of the P–N distance was 7.3 mm. The mean distances of the A–P, P–H, and P–V in patients with type 1 palate were 44.0 mm, 15.3 mm, and 12.1 mm, respectively; those same distances in patients with type 2 palate were

Table 1 Characteristics of patients with posteroinferior septal defect due to vomeral malformation

Patient no.	Symptoms	Type of hard palate	Type of PISD	Shape of PISD	Septal deviation	PBS	ITH	Uvula
1	Nasal obstruction	1	II	O	−	−	+	N
2	Oral pain	1	II	O	+	−	−	N
3	Nasal obstruction	1	II	O	+	−	+	N
4	Nasal obstruction	1	II	O	+	−	−	N
5	Rhinorrhea	1	II	O	+	+	+	N
6	Itching sensation in the nose	1	II	O	+	−	−	bifid
7	Sputum	2	I	O	+	−	+	N
8	Epistaxis	2	I	W	+	+	−	bifid
9	Nasal obstruction	2	II	O	−	−	−	N
10	Rhinorrhea	2	II	O	+	−	+	N
11	Nasal obstruction	2	II	O	+	+	+	N
12	Posterior nasal drip	2	II	W	+	−	−	N
13	Throat discomfort	2	II	W	+	+	−	N
14	Rhinorrhea	2	II	O	+	−	+	N
15	Cough	2	II	O	+	−	−	N
16	Nasal obstruction	2	II	O	+	−	+	s/p UPPP
17	Posterior nasal drip	2	II	O	−	−	−	N
18	Epistaxis	2	II	O	+	+	−	N

PISD posteroinferior septal defect, *PBS* posterior bony spur, *ITH* inferior turbinate hypertrophy, *O* oval, *W* wedge, *N* normal, *s/p* status postoperative, *UPPP* uvulopalatopharyngoplasty

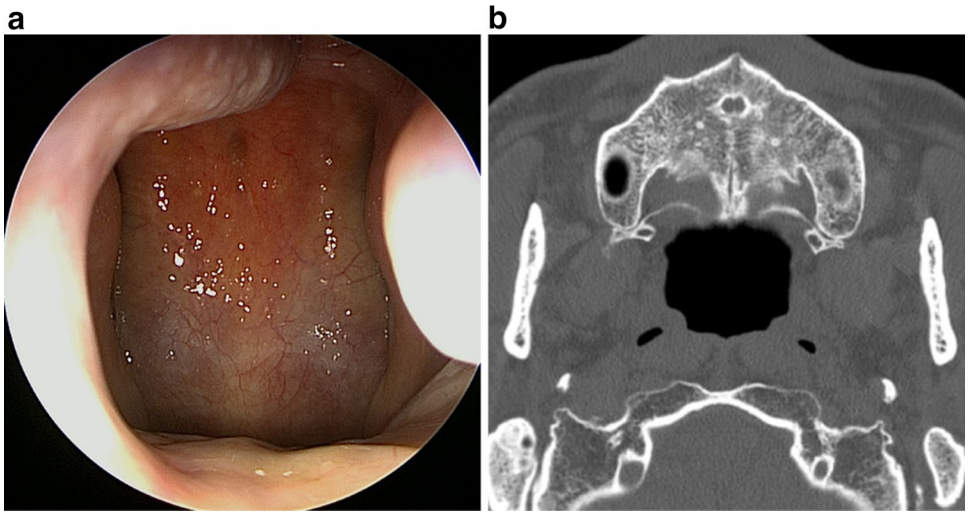

Fig. 2 **a** Oval-shaped type II posteroinferior septal defect and **b** type 1 hard palate, which lacks the posterior nasal spine

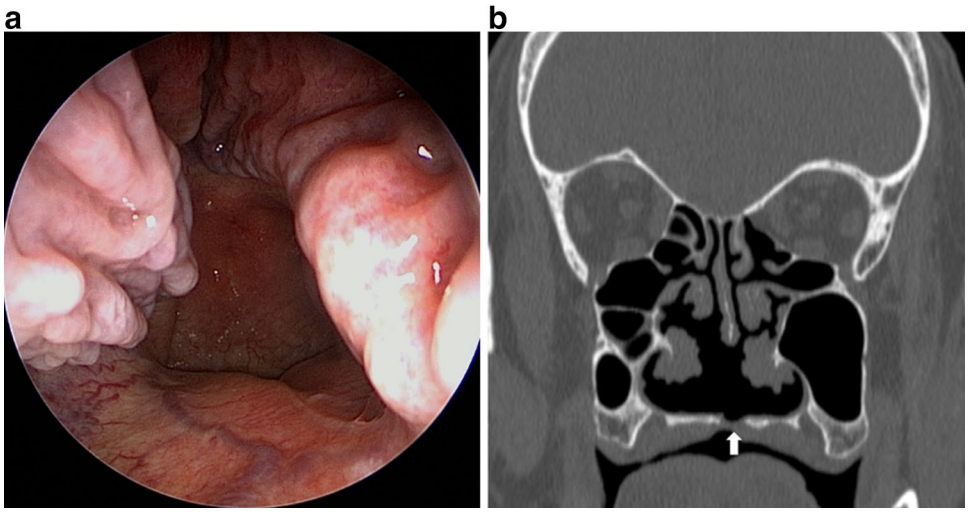

Fig. 3 **a** Wedge-shaped type I posteroinferior septal defect and **b** type 2 hard palate, which shows the cleft (arrow)

44.7 mm, 15.2 mm, and 12.5 mm, respectively. The P–N distance in type 2 patients was 7.3 mm (Table 3). There were no statistically significant differences in A–P, P–H, or P–V distances between palate types. There was a significant correlation between P–V and N–P distances in type 2 patients ($r=0.664$, $p=0.019$).

Discussion

Abnormal development of the vomer may cause defects in the posteroinferior nasal septum and is usually found incidentally. No other structural abnormalities of the palate or epipharynx have been found. However, sporadic cases have been reported in the literature, and no retrospective cohort studies have been performed. In the present study, we investigated the incidence, associated symptoms, and concomitant structural abnormalities of this condition.

The vomer begins to form after the appearance of the ossification center on both sides of the posterior lower part of the cartilaginous nasal septum in the eighth week of gestation. By that time, the palatal shelves rise and meet the nasal septum above the tongue. At about the seventeenth week of gestation, the ossification centers of both sides meet at the lower part of the septal cartilage and become U-shaped. After the nineteenth week, the vomeral bone develops into a Y shape and thickens laterally [14, 15]. PISD, as one of forms of nasal septal perforation, occurs due to various causes. In this study, we excluded secondary and extrinsic causes and included only those patients with congenital deformities. It is believed that CVDs occur when the vomer does not develop completely; if the gap with the palate is not covered by the mucosa, it appears as a PISD.

Endoscopic examination showed that the shape of the PISD could be classified as either a wedge or an oval. In this study, three wedge-shaped defects were found in patients with type 2 palates (one with type I and two with type II

Table 2 Measured distances between landmarks for each type of hard palate

Type of hard palate	Patient no.	A–P (mm)	P–H (mm)	P–V (mm)	P–N (mm)
1	1	47.8	19.0	12.0	–
	2	29.9	16.4	9.0	–
	3	45.4	17.6	17.2	–
	4	45.8	15.4	17.9	–
	5	45.2	12.7	8.1	–
	6	49.9	10.6	8.4	–
2	7	39	16.1	27	10.2
	8	45.4	20.0	25.8	16.8
	9	49.7	16.5	7.1	3.5
	10	46.0	20.4	15.0	6.6
	11	41.9	9.9	10.1	7.0
	12	28.7	11.7	6.0	5.0
	13	44.7	11.3	7.4	6.0
	14	47.8	13.0	8.8	8.4
	15	46.8	17.9	11.6	6.3
	16	44.6	20.0	11.6	5.3
	17	49.2	15.5	9.5	4.2
	18	52.4	10.6	10.6	8.4
Mean ± SD		44.5 ± 6.3	15.3 ± 3.5	12.4 ± 6.1	7.3 ± 3.5

A anterior nasal spine, *P* posterior end of the hard palate, *H* lowest end of the vomer at point P, *V* posterior end of the vomer articulated with hard palate, *N* apex of the V-notch, *SD* standard deviation

Table 3 Comparison of the measured parameters (mean ± SD) between the types of hard palate

Type of hard palate	A–P (mm)	P–H (mm)	P–V (mm)	P–N (mm)
1 ($n=6$)	44.0 ± 7.1	15.3 ± 3.1	12.1 ± 4.4	–
2 ($n=12$)	44.7 ± 6.2	15.2 ± 3.9	12.5 ± 6.9	7.3 ± 3.5
p value	0.963	0.925	0.815	0.019*

*Statistically significant correlation with P–V in type 2 palate ($r=0.663$)

n number of patients, *A* anterior nasal spine, *P* posterior end of the hard palate, *H* lowest end of the vomer at point P, *V* posterior end of the vomer articulated with hard palate, *N* apex of the V-notch

defects). The shape of the defect is thought to be influenced by the degree of the malformation of the vomer and hard palate, angulation between the posterior border of the vomer and the palate, and the surrounding mucosa.

As previously mentioned, the patients presented with a variety of manifestations in the present study. Patients with nasal obstruction were the most frequent (6 of 18), followed by three patients with rhinorrhea. It is difficult to determine whether these symptoms were associated with PISD because they are atypical among the symptoms treated in the otorhinolaryngology department. In addition, two patients each complained of posterior nasal drip and epistaxis, and many other patients showed a variety of symptoms. Therefore, we suspect that there are no characteristic symptoms related to PISD.

A deviated nasal septum was observed in most patients (15/18). In addition, a bony spur was observed in the posterior part of the nasal septum in five patients (1/6 with type 1 palate and 4/12 with type 2 palate). If the vomer is connected to one side of a cleft palate, the posterior bony nasal septum may show marked deviation [16, 17]. However, there was no patient in this study with a severe deviation of the posterior bony septum. This is thought to be because this study was performed on patients who had incomplete fusion between the posterior bony palate and the vomer. Sixteen patients had the vomer articulated with the hard palate in the midline. Two patients showed the vomer articulated with the left side before the V-notch, and one of them had a left-sided posterior bony spur (Fig. 4).

Fetuses with cleft palates between 8 and 21 weeks menstrual age (MA) exhibit a more rapid growth rate in the anteroposterior length and the volume of the vomer than fetuses without cleft palates. In addition, nasal airway volume growth decreases significantly by 21 weeks MA in fetuses with cleft palate, which seems to be due to enlargement of the nasal septal cartilage and vomer. The reasons for this remain unclear, but it may be explained by unrestricted growth of the nasal septum following palatal clefting [18, 19]. In addition, the reduced posterior maxillary height and the narrow nasal airway could lead to vomer deviation [20]. The deviated nasal septum and posterior bony spur in PISDs are influenced by many factors, and the embryological factor can be one of them, as in the case of cleft palate.

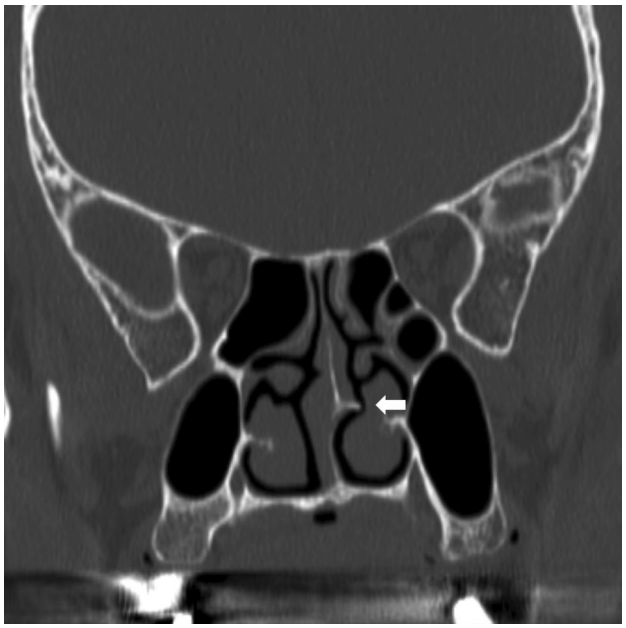

Fig. 4 Coronal scan demonstrating articulation of the vomer with the palate, which is deviated to the left side, and a posterior bony spur of the nasal septum (arrow)

This study did not include patients with symptoms related to velopharyngeal function, because it focused on the anatomical and structural connections between the vomer and its surrounding structures. The relationship between the development of VPI and the anatomical variations of cleft palate is still controversial. There are reports that the severity of the anatomical defect of the hard palate is not significantly related to the severity of VPI, and that the preoperative speech parameters, particularly hypernasality and nasal emission, have no correlation with the severity of the hard palate notch, uvula clefting, or muscle abnormality [12, 13, 21]. However, other reports claim that the greater and wider the extent of the cleft at the hard palate, the more likely the symptoms will develop [22, 23]. This is partly explained by an association of functional and anatomical abnormality. Although it is possible for patients with PISD with type 3 palate to present with no VPI symptoms, there were no such patients in this study. This may be because of the relatively small sample size; moreover, patients with severe anatomical abnormality are likely to be diagnosed and treated earlier.

In addition, there were no patients with type 0 palates in this study, which indicates PISD detected in the preoperative evaluations for palate surgery may help to detect abnormality of the hard palate. Hansen et al. showed that, in fetuses with cleft palate, U-shaped ossification of the vomer occurs normally at 14–15 weeks MA. However, the ossification centers of both sides at the lower part of the U-shaped vomer, corresponding to the footplate, do not appear until 18–19 weeks MA, depending on the coalescence of the soft tissue palatal shelves with the nasal septum. Moreover, the absence of the footplate may occur not only in the area of the cleft palate, but also in the anterior region of the cleft, where both palatal shelves are fused in the midline and the ossified vomer is U-shaped. It is not known whether this is a permanent deficiency or a delayed appearance [24].

Therefore, the absence of the bilateral ossification centers of the vomeral footplate, which would be connected to the palate, is thought to contribute to the occurrence of PISD. When the footplate does not develop in the anterior area of the cleft, a mild deformity, such as the absence of a PNS or a mild depression, seems to be enough to cause the failure of fusion of the vomer with the most posterior part of the palate.

Among the measured parameters, there were no statistically significant differences between the types of palate in the P–H and P–V distances. This suggests that type of palate may not be significantly related with the malformation of the vomer. These results are consistent with results of previous studies on the relation of the hard palate and the vomer. It is reported that the extent of deformity of the vomer is not related to the extent of the defect of the hard palate in symptomatic patients with cleft palate, and that the sagittal extent of the fusion rate of the vomer and hard palate is greatly varied [10, 11, 25].

However, in this study, there was a correlation between the P–V distance and P–N distance in patients with type 2 palate. This result seems to indicate that the V-point is always in front of the N-point, as the vomer was separated in front of the apex of the notch in all subjects, and the measured values of two patients with type I PISD showed a relatively big difference compared to the type II PISD. Additional studies are needed in this area, because the number of type I defects included in this study was insufficient.

Mohri and Amatsu proposed two theories to explain the causes of vomeral defects. The immature ossification theory states the vomer is not completely developed or later absorbed due to an imperfect ossification center. The incomplete downward growth theory states the primary nasal septum does not extend to the posteroinferior direction [1]. Verim et al. suggested the incomplete touch theory, which states the defect of the vomer occurs due to the delay of contact between the ossification center cell and the mesenchymal cell that becomes the future vomeral bone as the result of interfering mineralization [3]. We believe that, along with these possibilities, the bilateral absence of a footplate, which is considered to be closely related to the cleft palate, may be a cause of PISD.

In previous reports, patients with PISD due to vomer malformation were reported to have no structural abnormality of the epipharynx or other craniofacial regions. However, in this study, which used a grading system to describe the bony defect of the hard palate, normal PNS was not observed in

any of the subjects. Although the present study was retrospective in nature, with a relatively small sample size due to the low incidence of PISD, the results show that vomeral malformation is closely related to the deformity of the hard palate.

Conclusion

PISD due to vomeral malformation without VPI-related symptoms can present with a variety of manifestations. Because it is found incidentally without any characteristic symptoms, diagnosis can be performed by nasal endoscopy and paranasal sinus CT. In this study, 0.7% of the subjects had PISD due to vomeral malformations, and none had a normal PNS. Therefore, anatomical variation of the hard palate should be anticipated even in the absence of velopharyngeal insufficiency. This condition may be due to the bilateral absence of the vomeral footplate, which plays an important role in the fusion of the vomer and hard palate.

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Compliance with ethical standards

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

Ethical approval retrospective studies This study was approved by the institutional review board of Chungnam National University Hospital (reference number: CNUH201807013-HE001) and complies with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

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