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Review Article

THE ROLE OF OSTEOMEATAL COMPLEX ANATOMICAL VARIANTS IN CHRONIC RHINOSINUSITIS*

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Abstract

Currently, computed tomography is the method of choice for assessment of paranasal sinuses, nasal fossae and their anatomical variants. Presumably, these variations might induce osteal obstruction, preventing mucus drainage and predisposing to chronic rhinosinusitis. However, this concept is still controversial and the presence of any anatomical variant does not necessarily establish an etiology for rhinosinusitis. Among three subtypes of concha bullosa, only the bulbous type seems to be strongly associated with symptoms. Size and obliteration of osteomeatal complex drainage pathways may be relevant as well. Variations and tomographic signs of sinusal disease occurring on the same side reinforce the likelihood of interference with

the mucus drainage process. Computed tomography offers detailed study of anatomical variations and is an invaluable tool for managing clinical decisions and planning surgical strategies. Imaging assessment must be based on identification of variants, definition of their dimensions, as well as on their association with obstruction of drainage ostia and tomographic signs of sinus disease.
Keywords: Anatomical variations; Paranasal sinuses; Sinusitis; Computed tomography.

INTRODUCTION

The approach to patients with chronic rhinosinusitis has been changed with the arrival of the endoscopic functional surgery of paranasal sinus and nasal cavity. As far as it is concerned, the computed tomography (CT) has become indispensable to the surgical planning, since it allows a detailed study of the whole structure of this region, which would not be possible with plain X-rays. In most cases, the endoscopic surgery aims at removing the obstruction of the main drainage pathway – the osteomeatal complex –, based essentially on the concept that such obstruction perpetuates the sinus disease[1–3].

Paranasal sinus and nasal cavity anatomical variants are usual findings with an estimated prevalence of 65%[4]. Some authors propose the hypothesis that anatomical variants may be obstructive factors, predisposing to sinusitis[5]. Notwithstanding, a consensus on this matter has not been reached yet but many recent studies have contributed with new information. This article provides an updated review, showing points that seem to be of agreement about what remains undefined on this theme.

DEVELOPMENT AND ANATOMY OF PARANASAL SINUS AT CT

The development of paranasal sinus starts early in the fetal period as nasal cavity invaginations. Only the maxillary and ethmoidal sinuses are present and can pneumatize at birth. The sphenoidal and frontal sinuses develop from the first years of life[6], expanding progressively and maturing up to the age of 12–14 years[7,8]. The ethmoidal cells pneumatization process may originate some variant cells like agger nasi, concha bullosa and Haller's[7,8].

The anatomical relations of relevant bone structures and soft tissues with sinus drainage ostia can be more easily understood on coronal tomographic images[9] (Figure 1). The nasal septum is an osseocartilagenous wall dividing the nasal cavity into right and left sides. The lateral nasal wall consists of inferior and middle turbinates and, occasionally, a superior or supreme turbinate bone with their respective meatus. The middle meatus is the most important of them, with an opening — the semilunar hiatus — which receives the main drainage pathways

from the paranasal sinuses. The drainage to this fissure is made by the frontal sinus, through the frontal recessus, and the maxillary sinus, through the infundibula (medially limited by the unciform process and laterally limited by the ethmoidal bulla), middle and anterior ethmoidal cells. The semilunar hiatus and surrounding structures together compose the osteomeatal complex[9]. One believes that the obstruction of this narrow region is a key factor in the development of chronic sinusitis[1–3]. The drainage of the sphenoidal sinus and posterior ethmoidal cells is performed through the sphenoethmoidal recess and through the superior meatus. Only occasionally this posterior group of sinuses is affected by inflammatory processes[3,4,6,9].

ANATOMICAL VARIANTS

The role of anatomical variants in the sinusitis genesis is controversial. Theoretically, these variants could shift and compress osteomeatal complex components, determining an obstruction to the paranasal sinuses mucus drainage[9,10]. Researches on this theme consider that, if anatomical variants really predispose to sinusitis, one should expect that these variants were more frequently found at CT in patients with sinus disease (symptomatic) than in the general population (asymptomatic). Findings of several studies on the theme are summarized in Tables 1 to 6. Tonai and Baba[1] have analyzed tomographic studies of 75 adult patients. Comparison of anatomical variants prevalences in the symptomatic and asymptomatic groups has showed no significant difference (Table 1). In another study[4], of all the evaluated anatomical variants (Table 2), only one specific type of middle concha bullosa has presented association with the clinical disease (Table 3). Four studies have described the anatomical variants prevalence on CT examinations in children with chronic or recurrent sinusitis.(Table 4)[6,11–13]. However, in these studies there was not a control group for statistical correlation. Liu *et al.*[14] have demonstrated that the greater the size of the anatomical variant, the higher the frequency of association with paranasal sinus mucosal alterations at CT (Table 5). Scribano *et al.*[10] have studied patients with anatomical variants by means of CT, aiming at identifying patients in which an anatomical variant determined a contact between the mucosal surfaces of the osteomeatal complex (aerial space obliteration). One has observed that the maxillary sinus opacification was significantly more frequent in cases where the concha bullosa determined osteomeatal complex obliteration (Table 6), when compared with cases of concha bullosa without osteomeatal complex obliteration. For these authors, the contact of mucosal surfaces would be more significant for the sinusitis pathogeny than the size of concha bullosa.

Different types of anatomical variants present distinct relations with either clinical or tomographic sinus disease. Main anatomical variants are middle concha bullosa, Haller and agger nasi cells, nasal septum deviation and enlarged ethmoidal bulla.

The middle concha bullosa is a result of pneumatization of the osseous plate due to ethmoidal extention (Figures 2 and 3). A prevalence up to 80% is observed[4] and, besides Haller and agger nasi cells and deviated nasal septum, the middle concha bullosa is one of the most frequent anatomical variants[1,4,6,11–13] (Tables 1 and 2). In children diagnosed with sinusopathy, the frequency of this anatomical variant ranged between 4.2% and 24% of cases[6,11–13] (Table 4). Milczuk *et al.* have found an association with ipsilateral ethmoid-maxillary sinusopathy in the childhood in 63% of cases studied[13] (Figure 2). Contradictorily, Lusk *et al.*[12] have not observed any association of concha bullosa with sinus disease in children (Figure 3). Tonai and Baba[1] (Table 1) and Zinreich *et al.*[15] also have not detected any relation of concha bullosa with sinusopathy. It is important to note that the degree of pneumatization found in the population studied by Tonai and Baba[1] was low, which they attribute possibly to a racial factor (the study has been developed in Japan). The concha pneumatization may occur at several degrees, from that affecting only the bulbous portion (distal) (Figure 2) or lamellar portion (proximal), or the called true variant where there is pneumatization of both portions[4] (Figure 4). In one of these studies[4], the bulbous-type middle concha bullosa was the only anatomical variant presenting a relation with sinusopathy (Table 3). The association of anterior ethmoidal cells and maxillary sinuses with tomographic alterations may depend not only on the subtype, but also on dimensions of the concha bullosa[14]. Scribano *et al.*[10] have demonstrated that when the concha bullosa does not interfere with the amplitude of paranasal sinuses drainage paths at CT, it possibly cannot cause a sinus disease, and probably the rhinosinusitis genesis involves an osteomeatal complex aerial column obliteration by an anatomical variant (Table 6).

Haller cells are found in up to 45% of the general population[1,4,16,17] and in 1.4% to 18% of children[6,11–13] (Tables 1, 2 and 4). Haller cells are anterior ethmoidal cells that project beyond the limits of the bulla ethmoidal under the orbital floor, forming the infundibulum lateral wall between the papyracea lamina and the unciform process[1,13,18] (Figure 4). There was no difference in its prevalence between patients with and without sinusopathy (Tables 1 e 2)[1,4]. However, larger Haller cells are more likely related to tomographic alterations of maxillary sinuses[14,18] (Table 5).

Middle concha may present a curvature contrary to the one normally seen; this is another anatomical variant named paradoxical concha (Figure 5) and present at CT in 30% of patients (Tables 1 e 2)[1,4]. In children, its prevalence is lower, ranging from 4.4% to 10%[6,11–13] (Table

4). Depending on the curvature degree, the infundibulum may be compressed, causing sinusal obstruction[1,9]. There is no consistent data about its relation with sinusopathy[1,4,11–13].

The agger nasi cell is the most anterior ethmoidal cell, situated below the frontal sinus, next to the frontal recess, representing the lacrimal bone pneumatization due to ethmoidal extension (Figure 6). This may be an important factor in the genesis of symptoms like lacrimation and frontal sinus disease. Reported prevalence is quite variable (10% to 98.5%)[1,4,9] (Tables 1 and 2), possibly due to two factors: 1) different definitions are assigned to this anatomical variant; 2) the small size of the agger nasi cell might make its detection difficult in former researches involving anatomic pieces. Therefore, higher frequencies are described in CT studies in which agger nasi cell can be easily identified[1,4]. A positive relation between the cell size and the presence of frontal sinus disease has been described by Liu *et al*. (Table 5)[14]. Nassar Filho *et al.*[19] also have observed that this cell presented hyperpneumatized more frequently in the group with sinusopathy than in the control group. However, this anatomical variant has not been considered as an obstructive factor in the study of Voegels *et al.*[20].

In the junction of the nasal cartilage with the vomer, an acute angulation occurs in 20% to 30% of the population[7,16], constituting the nasal septum deviation (Figure 7), one of the most frequent anatomical variants[19,11,12]. Its prevalence in children ranges from 10.4% to 14%[6,11,12]. In older children (on average 9 years of age) septum deviation tends to be more pronounced[12]. This last observation might indicate an acquired nature of this condition[11]. It may determine compression of the middle concha with consequential infundibulum obstruction[9]. One has demonstrated the association of higher grades of septum deviation with ipsilateral sinusopathy in adults[9,21,22]. Some authors[1,4] do not mention this anatomical variant in their studies.

An enlarged ethmoidal bulla may obstruct the infundibulum or the middle meatus. The exact prevalence of enlarged ethmoidal bulla is not known[9,23]. Its size is an important factor when associated with opacification of anterior ethmoidal cells at CT in patients diagnosed with sinusopathy[14]. However, one has not found in the literature an objective description of what could be considered an enlarged ethmoidal bulla[9,10,19,23]. Late in its development, the ethmoidal sinus measures on average 36 x 18 x 14 mm (length, height and width) in measurements performed in MRI studies[24]. These measures were similar in cadaver skulls[25]. The ethmoidal bulla, however, has not been separately evaluated in theses studies. In measurements at CT in adults, the average area of each ethmoidal cell is 0.73 ± 0.42 cm², the larger ones, situated in the posterior portion of ethmoid, measure 1.46 ± 0.64 cm²[26]. Again, the size of the ethmoidal bulla has not been described separately. Since the ethmoidal bulla is the largest anterior cell[24], it is implicit that its average area should not exceed 2.1 cm².

Other anatomical variants appear less frequently. The superior extremity of the unciform process may deviate laterally, medially or anteriorly and may interfere in the middle meatus drainage[14]. Its prevalence was of 6.9% in a study with children[11]. The unciform process pneumatization also is hardly frequent (prevalence up to 2.5%)[1,4,11,12,16]. Hypoplastic maxillary sinus appears in about 6.9% to 17.5% of the pediatric population[11–13]. According to Lusk *et al.*[12], the hypoplasia may be a result of the loss of pneumatization by the infundibulum. The correlation of these anatomical variants with sinusopathy has been not determined[4,9,12,13].

DISCUSSION

Most probably, the rhinosinusitis genesis is multifactorial, and the physiological factor (mucociliary clearance disorders) possibly is as much significant as the mechanical obstructive factor[19]. Indications for surgical correction of the sinusal drainage deal with the possibility of an anatomical variant constituting an obstructive factor, principally at the level of the osteomeatal complex, but there is no reference to objective parameters like anatomical variant dimensions or drainage pathways amplitude as specific indicators[27,28].

The role of anatomical variants in the chronic or recurrent sinusitis pathogenesis can be evaluated by comparison between anatomical variants prevalence in populations with sinusopathy and prevalence in populations free from sinusal problems. If anatomical variants determine any effect on the chronic rhinosinusitis genesis, it is assumed that they are found more frequently in groups of patients with sinusopathy. Some studies on the prevalence of anatomical variants have failed in identifying a significant relation with rhinosinusitis symptoms or with mucosal alterations of paranasal sinus at CT[1,4,19,20]. However, Bolger *et al.*[4] have found out that the pneumatization of the bulbous portion of the middle concha presented a prevalence significantly increased in patients with sinusopathy (Table 3). Likewise, larger anatomical variants present higher probability of association with tomographic alterations of paranasal sinus (Table 5)[14]. Finally, even disregarding factors like subtype or size, Scribano *et al.*[10] have observed that, if the anatomical variant determines obliteration of the aerial space of the osteomeatal complex drainage paths, the sinusal disease is more frequently detected at CT (Table 6) than when the anatomical variant does not obstruct these pathways.

The prevalence of anatomical variants seems to increase with the age (Table 4). Lower prevalences are found in the study including lower age ranges (1 to 7 years of age)[6]. These data suggest the hypothesis of some anatomical variants being of acquired nature. Besides the fact that in children the sinus disease is usually bilateral and symmetrical[12], one may infer that anatomical variants have less influence on the sinusitis etiopathogenesis in this age range than in adults.

Some disparities between frequencies in different studies[1,4,6,10–14,19,20] can be explained by some controversial factors, definitions and ratings of the anatomical variant[4,9], the utilization of evaluation methods with different sensitivities (anatomical pieces *versus* CT) and also racial or population factors[1]. Also, it is necessary establish the difference between clinical sinusopathy and tomographic sinusopathy, since sinusal alteration at CT does not mean necessarily clinical disease[4,29–33]. Finally, since each variant seems to have a different influence on the development of the sinus disease, it would be convenient to determine the risk of each variant independently. A few studies involve a sufficient number of cases for a statistically satisfactory data analysis, as some anatomical variants present a very low incidence. Lusk *et al.*[12], for example, have examined 115 children and observed that the frequency of anatomical variants was not sufficiently high to allow a statistical correlation with sinusopathy.

CONCLUSION

There is no consensus in the literature on the role of anatomical variants in the chronic rhinosinusitis physiopathogenesis. The single detection of an anatomical variant itself does not establish the genesis of the disease. Before the suggestion of a causal relation between the anatomical variant and the sinusopathy in the tomographic analysis of a patient with sinusopathy and one anatomical variant, these conditions should be considered in conjunction with the clinical picture, its type and size, its association with obliteration of osteomeatal complex drainage paths and the presence of ipsilateral sinusal mucosa alterations.

REFERENCES

1. Tonai A, Baba S. Anatomic variations of the bone in sinonasal CT. Acta Otolaryngol Suppl 1996;525:9–13.

2. Isaacson G. Sinusitis in childhood. Pediatr Clin North Am 1996;43:1297–1318.

3. Zinreich J. Imaging of inflammatory sinus disease. Otolaryngol Clin North Am 1993;26:535–547.

4. Bolger WE, Butzin CA, Parsons DS. Paranasal sinus bony anatomic variations and mucosal abnormalities: CT analysis for endoscopic sinus surgery. Laryngoscope 1991;101:56–64.

5. Stammberger H, Wolf G. Headaches and sinus disease: the endoscopic approach. Ann Otol Rhinol Laryngol Suppl 1988;134:3–23.

6. Dutra LD, Marchiori E. Tomografia computadorizada helicoidal dos seios paranasais na criança: avaliação das sinusopatias inflamatórias. Radiol Bras 2002;35:161–169.

7. Kronemer KA, McAlister WH. Sinusitis and its imaging in the pediatric population. Pediatr Radiol 1997;27:837–846.

8. Scuderi AJ, Harnsberger HR, Boyer RS. Pneumatization of the paranasal sinuses: normal features of importance to the accurate interpretation of CT scans and MRI images. AJR Am J Roentgenol 1993;160:1101–1104.

9. Laine FJ, Smoker WR. The ostiomeatal unit and endoscopic surgery: anatomy, variations, and imaging findings in inflammatory diseases. AJR Am J Roentgenol 1992;159:849–857.

10. Scribano E, Ascenti G, Cascio F, Racchiusa S, Salamone I. Computerized tomography in the evaluation of anatomic variations of the ostiomeatal complex. Radiol Med (Torino) 1993;86:195–199.

11. April MM, Zinreich SJ, Baroody FM, Naclerio RM. Coronal CT scan abnormalities in children with chronic sinusitis. Laryngoscope 1993;103:985–990.

12. Lusk RP, McAlister B, el Fouley A. Anatomic variation in pediatric chronic sinusitis: a CT study. Otolaryngol Clin North Am 1996;29:75–91.

13. Milczuk HA, Dalley RW, Wessbacher F, Richardson M. Nasal and paranasal sinus anomalies in children with chronic sinusitis. Laryngoscope 1993;103:247–252.

14. Liu X, Han D, Zhou B. Relationship between anatomic variations of nasal sinus and chronic sinusitis. Zhonghua Er Bi Yan Hou Ke Za Zhi 1998;33:149–152.

15. Zinreich SJ, Mattox DE, Kennedy DW, Chisholm HL, Diffley DM, Rosenbaum AE. Concha bullosa: CT evaluation. J Comput Assist Tomogr 1988;12:778–784.

16. Arslan H, Aydinlioglu A, Bozkurt M, Egeli E. Anatomic variations of the paranasal sinuses: CT examination for endoscopic sinus surgery. Auris Nasus Larynx 1999;26:39–48.

17. Kennedy DW, Zinreich, SJ. Functional endoscopic approach to inflammatory sinus disease: current perspectives and technique modifications. Am J Rhinol 1988;2:89–96.

18. Stackpole SA, Edelstein DR. The anatomic relevance of the Haller cell in sinusitis. Am J Rhinol 1997;11:219–223.

19. Nassar Filho J, Anselmo-Lima WT, Santos AC. Participação das variações anatômicas do complexo ostiomeatal na gênese da rinossinusite crônica, analisadas por tomografia computadorizada. Rev Bras Otorrinolaringol 2001;67:489–495.

20. Voegels RL, Goto EY, Chung D, Nita LM, Lessa MM, Butugan O. Correlação etiológica entre variações anatômicas na tomografia computadorizada e a rinossinusite crônica. Rev Bras Otorrinolaringol 2001;67:507–510.

21. Kainz J, Stammberger H. The roof of the anterior ethmoid: a place of least resistant in the skull base. Am J Rhinol 1989;3:191–199.

22. Elahi MM, Frenkiel S, Fageeh N. Paraseptal structural changes and chronic sinus disease in relation to the deviated septum. J Otolaryngol 1997;26:236–240.

23. Castro Jr NP, Takara CK. Anatomia cirúrgica da parede lateral do nariz. Rev Bras Otorrinolaringol 1998;64(Supl 2):5–10.

24. Dos Santos RM. Desenvolvimento dos seios paranasais: estudo por ressonância magnética do crânio. (Tese de Mestrado). São Paulo: Universidade Federal de São Paulo, 2002.

25. Wolf G, Anderhuber W, Kuhn F. Development of the paranasal sinuses in children: implications for paranasal sinus surgery. Ann Otol Rhinol Laryngol 1993;102:705–711.

26. Saheki T. The investigation of the structures in the ethmoidal air cells on CT. Nippon Jibiinkoka Gakkai Kaiho 1989;92:827–836.

27. Kennedy DW, Zinreich SJ, Rosenbaum AE, Johns ME. Functional endoscopic sinus surgery: theory and diagnostic evaluation. Arch Otolaryngol 1985;111:576–582.

28. Zinreich SJ, Kennedy DW, Rosenbaum AE, Gayler BW, Kumar AJ, Stammberger H. Paranasal sinuses: CT imaging requirements for endoscopic surgery. Radiology 1987;163:769–775.

29. Havas TE, Motbey JA, Gullane PJ. Prevalence of incidental abnormalities on computed tomography scans of the paranasal sinuses. Arch Otolaryngol Head Neck Surg 1988;114:856–859.

30. Manning SC, Biavati MJ, Phillips DL. Correlation of clinical sinusitis signs and symptoms to imaging findings in pediatric patients. Int J Pediatr Otorhinolaryngol 1996;37:65–74.

31. Diament MJ, Senac MO, Gilsanz V, Baker S, Gillespie T, Larsson S. Prevalence of incidental paranasal sinus opacification in pediatric patients: a CT study. J Comput Assist Tomogr 1987;11:426–431.

32. Lesserson JA, Kieserman SP, Finn DG. The radiographic incidence of chronic sinus disease in the pediatric population. Laryngoscope 1994;104:159–166.

33. Araújo SA, Souza AS, Sanches RA, *et al*. Significado clínico da opacificação incidental dos seios da face à TC em crianças: resultados preliminares. Radiol Bras 2001;34(Supl 1):45.

O PAPEL DAS VARIANTES ANATÔMICAS DO COMPLEXO OSTIOMEATAL

Tabelas e Figuras

Table 1 Prevalence of anatomical variants at CT (%). (Tonai & Baba[1]).

	Total (n = 75)	Symptomatic (n = 57)	Asymptomatic (n = 18)	*p*
Agger nasi cell	86.7	86.0	88.9	> 0.05
Haller cell	36.0	33.3	38.9	> 0.05
Middle concha bullosa	28.0	28.1	27.8	> 0.05
Middle paradoxical concha	25.3	29.8	11.1	> 0.05
Pneumatized uncinate	0	0	0	> 0.05

Table 2 Prevalence of anatomical variants at CT (%). (Bolger *et al*.[4]).

	Total (n = 202)	Symptomatic (n = 166)	Asymptomatic (n = 36)	*p*
Agger nasi cell	98.5	[a]	[a]	[a]
Haller cell	45.1	45.9	41.6	> 0.05
Middle concha bullosa	53.0	53.6	50.0	> 0.05
Middle paradoxical concha	26.1	27.1	22.3	> 0.05
Pneumatized uncinate	2.5	3.0	5.6	> 0.05

[a] Not reported by the authors.

Table 3 Prevalence of middle concha bullosa subtypes in symptomatic and asymptomatic patients at CT (%). (Bolger *et al*.[4]).

	Total (n = 202)	Symptomatic (n = 166)	Asymptomatic (n = 36)	*p*
Lamellar	46.2	45.9	47.2	0.99
True	15.7	17.4	8.4	0.38
Bulbous*	31.2	35.3	13.9	0.04*

* Statistically significant difference.

Table 4 Prevalence of anatomical variants in symptomatic children at CT (%).

	Dutra & Marchiori[6]	Lusk *et al*.[12]	Milczuk *et al*.[13]	April *et al*.[11]
Haller cell	1.4	10.0	5.3	18.0
Middle concha bullosa	4.2	10.0	9.6	24.0
Middle paradoxical concha	[a]	8.5	4.4	10.0
Pneumatized uncinate	1.4	0	[a]	[a]
Nasal septum deviation	14.1	10.4	[a]	13.0

[a] Not reported by the authors.

Table 5 Size of the anatomical variant in relation to the tomographic alteration of respective paranasal sinus (between brackets in the first column). (Liu *et al*.[14]).

	Altered sinus	Normal sinus	*p*
Agger nasi cell (frontal)	11.7 mm	8.5 mm	< 0.01*
Haller cell (maxillary)	91.6 mm^2	41.6 mm^2	< 0.05*

* Statistically significant difference.

Table 6 Relation of the mucosal contact in the osteomeatal complex over the opacification in the presence of anatomical variants[a]. (Scribano *et al*.[10]).

	Maxillary opacification	Normal maxillary	*p*
With contact (n = 44)	35	9	< 0.05*
Without contact (n = 69)	17	52	< 0.05*

[a] n = 73 (113 anatomical variants). * Statistically significant difference.

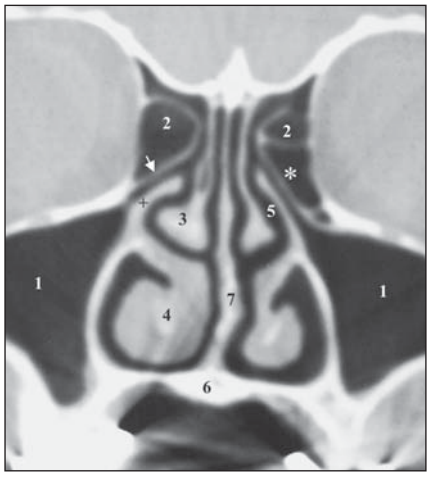

Figure 1. Coronal CT slice at the level of the osteomeatal complex. Maxillary sinus (1), ethmoidal cells (2), middle concha (3), inferior concha (4), middle meatus (5), hard palate (6), nasal septum (7), uncinate process (+), ethmoidal *bulla* (*), infundibulum (arrow). Note the slight nasal septum deviation to the left.

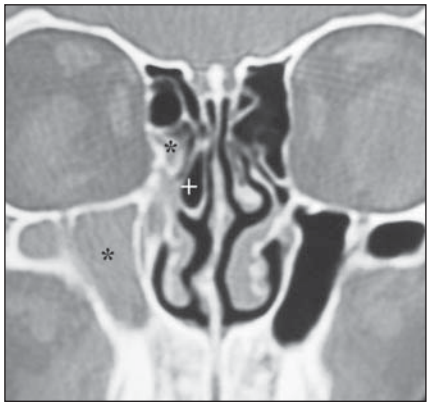

Figure 2. Coronal CT at the level of the osteomeatal complex. Right ethmoid-maxillary sinusopathy (*), probably determined by obstruction of the osteomeatal complex by bulbous-type middle concha bullosa (+).

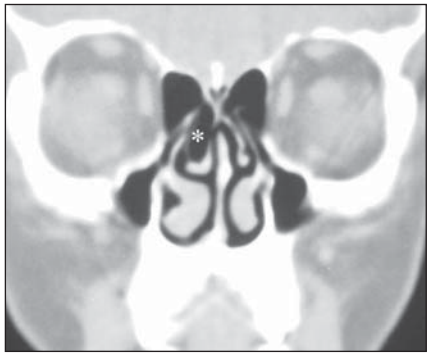

Figure 3. Coronal CT at the level of the osteomeatal complex. Child with a true middle concha bullosa to the right (*), with dimensions similar to that presented in Figure 2, without associated sinus disease.

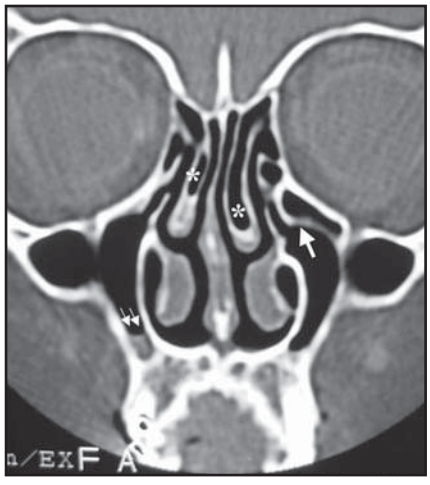

Figure 4. Coronal CT. Haller cell to the left (arrow), associated with bilateral conchas bullosas (*), a true concha bullosa at the left and a lamellar concha bullosa to the right, both small-sized. The osteomeatal complexes are free from obstruction. Minimal mucosal disease can be observed in the right maxillary sinus (double arrows).

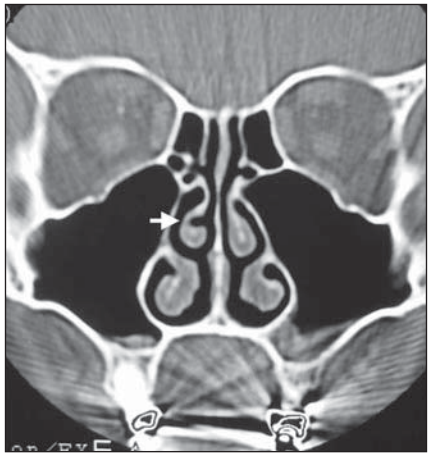

Figure 5. Coronal CT. Paradoxical middle concha to the right (arrow), posteriorly to the osteomeatal complex. There is a discrete mucous thickening on the floor of the maxillary sinus.

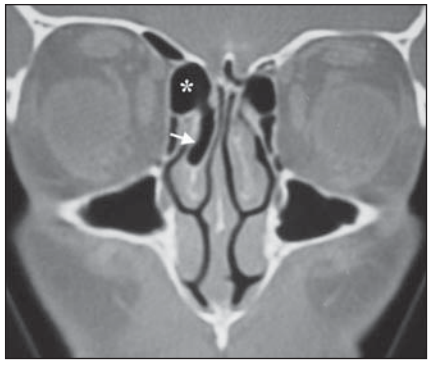

Figure 6. Coronal CT. Lamellar-type middle concha bullosa to the right (arrow), whose pneumatization comes upon a prominent agger nasi cell (*), next to the right frontal recess. There is no sinus disease.

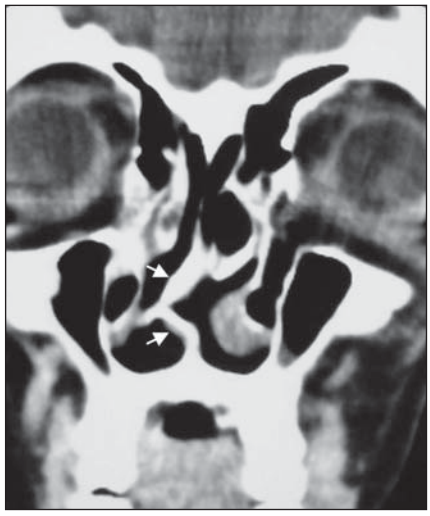

Figure 7. Coronal CT. Accentuated nasal septum deviation to the right (arrows), with formation of a spur.