CLINICAL STUDY

Bilateral Hyperplasia of the Coronoid Process in Pediatric Patients: What is the Gold Standard for Treatment? A Systematic Review of the Literature

Marco Farronato, DDS, PD, Alberta Greco Lucchina, DDS,† Carmen Mortellaro, MD, DDS,‡ Andrea Fama, DDS,* Guido Galbiati, DDS, PD,* Giampietro Farronato, MD, DDS,* and Cinzia Maspero, MD, DDS, PD, AP**

Objectives: The aim of this systematic review of the literature is to describe treatment options for bilateral coronoid process hyperplasia in pediatric patients, to describe etiologic and diagnostic correlations with the treatment, and to evaluate long-term follow-up treatment outcomes.

Methods: A systematic revision of the literature was performed in the Medline, PubMed, Cochrane library, and Embase database up to December 5, 2017. Predetermined Medical Subject Heading keywords were used: "bilateral" or "monolateral" and "coronoid" or "coronoid process" and "hyperplasia" and "temporomandibular joint" or "tmj" and "ankylosis" or "trismus" and "treatment". Results were recorded following PRISMA guidelines.

Results: The systematic research produced 1459 results excluding duplicates. Two additional studies from "Grey literature" were also considered. After application of inclusion and exclusion criteria, 38 articles were selected for a qualitative synthesis. Data regarding sex and age of presentation were collected and summarized in a study flow diagram.

Conclusion: It is possible to conclude that early diagnosis is fundamental to restore stomatognathic multifunction. There is lack of longitudinal studies presenting long-term follow-up to determine treatment stability. Coronoidectomy might be considered gold standard treatment for this pathologic condition.

Key Words: Coronoid hyperplasia, coronoid process, coronoidectomy, coronoidotomy, temporomandibular joint ankylosis, trismus

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The purpose of this study is to define treatment possibilities by analyzing the literature, considering the etiology and diagnosis of coronoid process hyperplasia (CPH).

From the *Università degli studi di Milano; †Università degli Studi del Piemonte Orientale; and ‡Department of Medical Science, University of Eastern Piedmont, Novara, Italy.

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Address correspondence and reprint requests to Marco Farronato, DDS, PD, Università degli studi di Milano, Via Francesco Sforza 28, Milano 20122, Italy; E-mail: marcofarronato@msn.com

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The CPH, also defined as giant coronoid syndrome, is a pathologic condition[1] first described by Langenback in 1853. It consists in an abnormal volumetric increment of the mandibular coronoid process, without interposition of synovial tissue,[2,3] and it presents as a mechanical problem, limiting mouth opening.

The abnormal apposition of bone occurs as a result of hypermetabolic growth nuclei that do not present anomalous aspects of dysplasia but paraphysiologic hyperplasia and absence of findings in histologic examination.[1]

This condition could be mono or bilateral, although the bilateral form seems to be more frequent. Unilateral hyperplasia is usually a consequence of traumas or pathologies, for example, osteochondromas (known as Jacob disease), and it is frequently associated with facial asymmetry.[4] Bilateral form affects both genders[2,5] with a male to female rate of 5:1[6] and a peak of prevalence at 25 years.[7,8]

Etiopathogenesis is not totally clear but it seems to be related to external or acquired factors such as dysfunctional and traumatic outcomes, as asserted by Tucker et al,[2] post-radiant therapy sequences, inflammatory events (eg, juvenile rheumatoid arthritis and puerile infections), and neoplastic cases, exostosis, osteochondromas, and various secondary events derived from other pathologies (eg, Nevoid Basal Cell Carcinoma Syndrome or NBCCS). The main clinical finding is a progressive and painless difficulty in mouth opening resulting into trismus.

METHODS

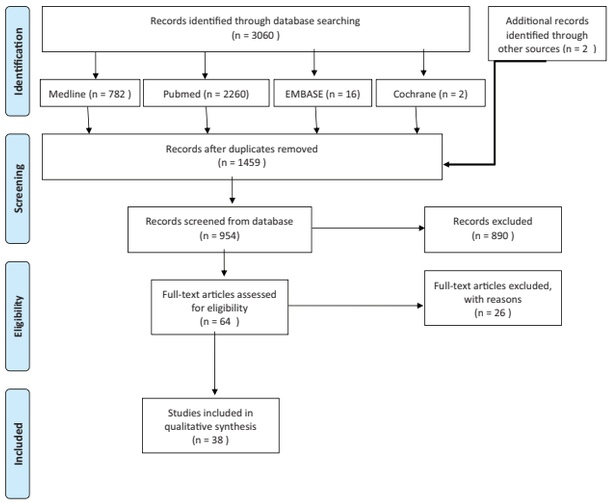
The protocol set for this systematic investigation observed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses PRISMA statement.[9] A flow chart of the article selection process at each stage of the review is presented in Figure 1.

Search Strategy

The systematic review is conducted through the following electronic databases: Medline, PubMed, Embase, and Cochrane Library. The Medical Subject Heading (MeSH) was applied for finding the keywords. Keywords recorded for selecting the data were "bilateral" or "monolateral" and "coronoid" or "coronoid process" and "hyperplasia" and "temporomandibular joint" or "tmj" and "ankylosis" or "trismus" and "treatment".

Inclusion Criteria

Articles between 1963 and December 5, 2017 were selected. Using the "limits" option, only articles referring to "Humans" were considered. Topics considered for inclusion were: reliability or accuracy of the diagnostic method, information about etiology, diagnosis and therapy during pediatric ages, and specific articles regarding the role of surgical and rehabilitative treatment. The following types of studies were included: multiple or single case reports, literature reviews, and trials referring to clinical research on



AQ14 **FIGURE 1.** Review results flow diagram.

humans. Articles with abstracts missing full length text manuscript were also considered. Commentaries, letters to the editor, short communication, articles referring to non-dentistry topic, and those published before 1963 were not contemplated.

Manuscript Selection

Two independent reviewers among the authors were responsible for article selection following the same criteria. Disagreements in the selection were discussed until agreement, and in case of lack of consensus, a third reviewer among the authors was selected.

Data Extraction

The following parameters were used to assembly a database of the research.

1. Parameters of the studies considered:
   - "author": referred to the first surname of the author of the study.
   - "year": indicated the year of final publication, or the year of last record when not applicable.
2. Qualitative and quantitative parameters of the cases described in the articles:
   - "sex": referred about the gender of the patients.
   - "age presented": referred to the first appearance of CPH.
   - "age diagnosed": referred to the age were CPH was diagnosed.
   - "Treatment": referred IOBC = intra oral bilateral coronoidectomy; EOBC = extra oral bilateral coronoidectomy; No treatment: untreated; Physio = physiotherapy; Coronal = coronal flap; reoperation: 2-phase treatment; Multiple operation: more than 2-phase treatment.
   - "Preoperatively": referred to mouth opening before treatment.
   - "Postoperatively": referred to mouth opening posterior to the treatment where WNL stands for within normal limits when units were not specified.
   - "Δ": referred to the delta before and after treatment.
   - "Follow-up": When specified in original articles referred to follow-up described.
   - "Miscellaneous": referred to other relevant information collected.
3. Qualitative synthesis of study design, therapeutic outcomes, and author's conclusion.

Parameters data extracted were collected in a spreadsheet (Excel, Microsoft).

Limitations of the Review

A meta analysis was not possible due to the heterogeneity of the diagnostic tools assessed and the variability in study designs. No reportable limitations exist as the PRISMA guidelines were followed.

Risk of Bias in Individual Studies

The Quality Assessment of Diagnostic Accuracy Studies tool-2 (QUADAS-2) was used to evaluate risk of bias.[10] The reviewers assessed the risk of bias of each study independently during the article selection. Any discrepancies in bias risk assessment were resolved by a third reviewer among the authors.

RESULTS

About 3062 articles were found in total. After elimination of duplicates (n = 1459), the primary search resulted in 954 articles. Two additional studies from non-peer-reviewed journals (Gray literature) were also considered. Successively 890 articles were marked as non-relevant on the basis of abstract, title, and study design. Abstracts of 64 articles were read to exclude additional irrelevant studies. Thirty-eight articles met the inclusion criteria and were included in this review for qualitative analysis. The PRISMA flow chart (Fig. 1) illustrates search methodology and results.

DISCUSSION

Etiopathogenesis

Several theories for the etiopathogenesis of the CPH have been described in the literature. Wenghoefer et al found 3 cases with temporalis hyperplasia and 2 cases with hypertonic masticatory muscles due to a neurologic disorder.[11] Lyon and Sarnat suggest that a hyperactivity of the temporalis muscle is able to exercise constant mechanical microstimulations producing a sort of reactive hypertrophy of the coronoid process.[12]

Other authors hypothesize an etiology of the giant coronoid process as a result of temporomandibular disorders,[5] traumas[4] associated to consequent unilateral bone, cartilaginous or connective tissue proliferation in the area of the coronoid, and due to the persistence of an active growth center at the same level of the coronoid process.[13]

Other studies disagree and show normal electromyographic characteristics of the temporalis and masseter muscles in patients with diagnosis of CPH as evidence to the contrary.[4,14–16]

The CPH was associated with Moebius syndrome in 2 cases, characterized by facial musculature paralysis at birth. In these patients, authors describe that hypoplasia of the mandible and masticatory muscles was present, which also contradicts the temporalis muscle hyperactivity theory.[17]

Genetic influence in the CPH has been demonstrated by multiple studies as reported in the following. Puche et al confirmed the association between the temporalis muscle hyperactivity and coronoid hyperplasia in a series of patients with syndromic diagnosis of Kabuki syndrome and Pena-Shokeir syndrome.[18]

Mandibular CPH has been related to other genetic disorders, such as Gardner,[19] Jacob,[8] and Hecht syndromes.[20,21]

A study conducted by Khandavilli et al on monozygomatic twin sisters presenting bilateral coronoid hyperplasia shows the strong relationship between genetic transmission and CPH.[22]

Rowe first suggested a relationship between CPH and endocrine system alterations in the peripuberal period as the result of abnormal hormonal stimulus on the tissues of the coronoid process.[23] Cases of muscular trismus had also been described even if in absence of muscular hypertrophy.[24] Therefore the background of this pathologic condition could be a mechanical problem[5] caused by friction between osseous surfaces.

Differential Diagnosis

Giant coronoid syndrome is clinically accompanied by a painless and latent symptomatology. It is easily confused with a variety of disorders of the temporomandibular joint (TMJ) such as muscle and joint pathology, head and neck trauma, TMJ dysfunction, infection, soft and hard tissue pathology, muscle contracture, and neurologic disorders.[18]

Therefore, it is important to provide a differential diagnosis, an accurate anamnesis, a complete objective examination, and acquisition of pathognomonic signs such as the progressive and painless difficulty in opening the mouth because of the interfering contact with the zygomatic bone temporal surface or with the medial plane of the zygomatic arch itself.[25]

Diagnosis and Clinical Features

Mandibular hypomobility involves a series of negative consequences, as affirmed by Costello and Edward.[26] A reduced oral opening could cause respiratory problems, delayed growth, malnutrition, speech delay, and difficulties in maintaining good oral hygiene. Moreover, considering that this syndrome affects young patients in particular during the growth phase, the combination of muscular contraction, the atrophy resulting from hypomobility, and coronoid hypertrophy could cause both dental and skeletal developmental irregularities.[2] Nevertheless occlusion is unaffected.[27]

Levandoski analysis[28] in the orthopanothomograpy (Fig. 2) is useful for measuring the length of the coronoid process (Fig. 2), where Cd (condyle), Go (gonion), and Kr (coronoid process) points and 4 lines are considered: line 1 is the vertical median passing across the nasal septum; lines 2, 3, and 4 are perpendicular to line 1 and intersect the inferior edge of the mandibular symphysis (line 2), the extreme superior limit of condyle (line 3), and the coronoid (line 4). According to this analysis, the ratio between Cd-Go and Kr-Go distances (Kr-Go/Cd-Go), on both sides of the patient, should normally have a value <1.07.[29]

In addition, magnetic resonance imaging analyses the presence of an eventual limitation of the condylar excursion, the condyle-meniscus relationship, and in general, the glenoyd fossa, to exclude an anterior meniscus dislocation. The CBCT remains the gold standard, both during the diagnostic phase and during therapeutic approach: in fact, it provides information about coronoid morphology and its relationship to the zygomatic arch.[6] Yura et al propose that an open mouth 3D scan can prove and depict the exact location of impingement.[30]

Therapeutic Options and Outcomes

The CPH is treated by surgery, because the limited mandibular movements are mainly caused by a mechanical obstacle. The aim of the therapeutic plan is to restore the physiologic mouth opening. Our review of the literature (Table 1) reports increased mouth AQ7 opening from 5 to 25 mm postoperatively. This includes the review by McLoughlin et al.[7]

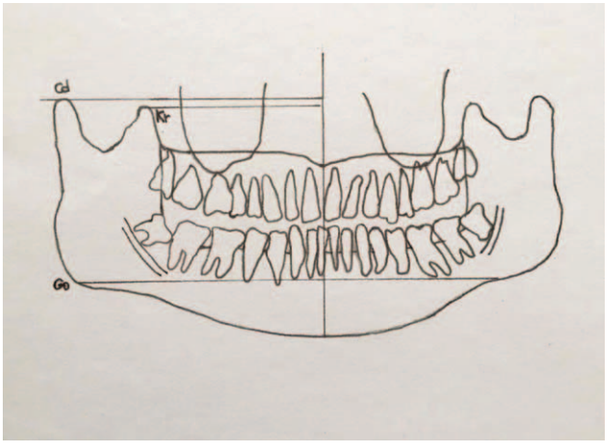


FIGURE 2. Levandoski layout. Cd (condyle), Go (gonion), and Kr (coronoid process) points are considered along with four lines: line 1 is the vertical median passing across the nasal septum; lines 2, 3, and 4 are perpendicular to line 1 and intersect the inferior edge of the mandibular symphysis (line 2), the extreme superior limit of condyle (line 3), and of the coronoid (line 4).

Both intra-oral and extra-oral approaches have been described in the literature. Two types of surgery are possible: coronoidectomy and coronoidotomy.

The intra-oral approach usually provides enough exposure to allow the removal of the hyperplastic process without visible scarring. The major risk is postoperative hematoma and fibrosis. Several extra-oral approaches have been proposed, such as submandibular, pre-auricular, (bi)temporal,[31] or endoscopically assisted.[32] The advantages of the extra-oral surgical approach are: less fibrosis and/or hematoma formation and better exposure to resect the coronoid process and release the temporalis muscle. The damage of the facial nerve and a visible scar during healing process are the main risks of the extra-oral technique. Many authors suggest that the elective treatment is coronoidectomy with intraoral access.[7,12]

Hayter and Robertson recomend a histologic examination on coronoidal removed section, to exclude any aspect of malignity.[33]

According to a study by Ostrofsky and Lownie, a submandibular approach is indicated in cases of anchylosis between coronoid and zygomatic processes.[34]

Coronal access should be elective in cases of more serious coronoid hyperplasia and associated lesions as osteomas.[33,35]

Daniel et al assert that the coronal transzygomatic coronoidect- AQ8 omy represents the optimal approach for the radical resection of the coronoids and distal temporalis inserction, in Hecht syndrome.[21]

Mohanty et al state that coronoidotomy is more straightforward adjunct to arthroplasty than coronoidectomy in the management of TMJ ankylosis.[36]

With regards to when the treatment of children or adolescents should begin, authors agree that it should be effectuated once the growth phase of the patient is completed to prevent recidivism. Exception to this approach are in cases where the patient has severe difficulty in opening the mouth making feeding problematic as is reported by Mano et al[24] or Satoh et al.[37]

Wallender et al suggest that prompt diagnosis and treatment is essential to prevent acute and long-term consequences.[38]

Gibbons and Abulhoul underlines the importance of following a postsurgical rehabilitative protocol, once restored normal values of mouth opening have been restored. It consists of the combination of passive and active physiotherapy in order to reach stable results.[39]

AQ12 **TABLE 1.** Review Results of the Literature

	Year	Sex	Age Presented	Age Diagnosed	Treatment	Preoperatively	Postoperatively	Δ	Follow-Up	Miscellaneous
Ginestet	1957	M	7	19	IOBC	5	NS		NS	
Rowe	1963	M	12	15	IOBC	15	35	20		
Nickerson	1969	M	11	22	EOBC	9	24	13		
Van Hoof	1973	M	12	12	Physio	13	20	7		TPS F
	1973	M	3	32	EOBC (reoperation)					TPS F
Rusconi	1974	M	12	21	IOBC	18	38	20		
Monks	1978	M	11	50	EO/IOBC	8	37	25		
York	1983	F	7		IOBC	<10	WNL	25		
	1983	F	12		IOBC	<10	WNL	25		
Bernstein	1984	M	12	17	IOBC	13	30	17		
Giacomuzzi	1986	M	11	19	No treatment	11				
Hall	1989	M	11	23	IOBC	9	38	27		
Totsuka	1991	M	10	13	IOBC	29	45	16		
Azaz	1994	M	–	3.5	IOBC (5)	–	23			TPS F
Smyth	1994	M	8	15	Coronal/IOBC	4	23	19		
McLoughlin	1995	M	1	7	IOBC	12	17	5		
		F	7	8	IOBC	15	39	24		
		M	8	13	IOBC	NS	NS			
		M	11	13	IOBC	14	29	15		
		M	1	21	IOBC	10	21	11		
Karras	1995	F	5	14	IOBC (multiple operations) (reoperation)	10	20	10		TPS F
Fabie	2002	F	Birth	8	IOBC	6	30	24		
Mano	2005	M	5	6	IOBC (8 y)	17	40	23		
Carlos	2005	M	Birth	6	IOBC	23				
Wenghoefer	2008	F		2	IOBC + Physio	10	25		Stable	
		M		45	IOBC + Physio	10	31		Stable	
		M		16	IOBC + Physio	14	40		Stable	
		M		18	IOBC + Physio	16	>30		Stable	
		M		24	IOBC + Physio	18	40		Stable	
		F		5	IOBC + Physio	4	32		Stable	
		M		35	IOBC + Physio	5	31		Stable	
		M		23	IOBC + Physio	10	23		Stable	
		M		38	IOBC + Physio	7	35		Stable	
		F		53	IOBC + Physio	–	>30		Stable	
		M		4	IOBC + Physio	–	>30		Stable	
		M		14	IOBC + Physio	15	33		Stable	
		M		28	IOBC + Physio	–	>30		Stable	
		F		18	IOBC + Physio	–	>30		Stable	
		M		52	IOBC + Physio	22	–			
		M		56	IOBC + Physio	25	–			
Gerbino	1997	M	14	15	IOBC + Physio	15	46		41 (12 mo)	
		M	15	32	IOBC + Physio	20	42		38 (5 y)	
		M	12	14	IOBC + Physio	12	46		48 (5 y)	
		M	10	13	IOBC + Physio	18	40		38 (15 mo)	
		M	14	16	IOBC + Physio	20	38		45 (5 y)	
Puche	2012	M		8 mo	EOBC + Physio	9	22		27	
		M		10 mo	EOBC + Physio	11	24		25	
Oliveira	2016	M	Birth	12	IOBC (multiple operations)	5	45			
Balkin	2015	M	Birth	7 mo	EOBC + Physio	5	10		Stable	
Wallender	2015	M	Birth	2 mo	IOBC + Physio	4	25			
Baraldi	2010	F		20	IOBC + Physio	12	35			

EOBC, extra oral bilateral coronoidectomy; IOBC, intra oral bilateral coronoidectomy.

McLoughlin et al asserted, in a review of 31 cases, that only half of treated patients has re-established a physiologic mandibular opening (>30 mm at least). Failure of the other treatments was mainly due to postsurgical errors or lack of rehabilitation.[7]

To sum up, successful treatment of coronoid hyperplasia depends on a correct surgical approach associated with early postsurgical physiotherapy.[40]

CONCLUSION

The pathology manifests by mouth opening reduction (<20 mm) and can be confirmed by radiographic assessment of head and neck region. The CBCT can be considered the gold standard for radiologic diagnosis.

Depending on the severity of the pathology, patients should be treated once growth is completed to prevent recidivism, excluding the cases presenting severe oral opening reduction.

There is no scientific consensus in the literature about electromyographic changes of masticatory muscles.

Primary aim of surgical treatment is to re-establish a correct mandibular mobility to permit normal functional growth.

The success of surgery is quantified considering mandibular opening degree immediately after treatment and its maintenance over time and it is strictly related to postsurgical physiotherapy, best results have been observed when intraoral coronoidectomy was performed. However, the literature is limited to short-time studies; therefore, more studies are needed to objectively measure success and treatment stability percentage presenting long-term follow-up in longitudinal studies.

REFERENCES

1. Fernandez Ferro M, Fernandez Sanrom an J, Sandoval Gutierrez J, et al. Treatment of bilateral hyperplasia of the coronoid process of the mandible. Presentation of a case and review of the literature. *Med Oral Patol Oral Cir Bucal* 2008;13:E595–E598
2. Tucker MR, Guilford WB, Howard CW. Coronoid process hyperplasia causing restricted opening and facial asymmetry. *Oral Surg Oral Med Oral Pathol* 1984;58:130–132
3. Maspero C, Pitto I, Salvadori S, et al. Malocclusioni di classe II prima divisione: considerazioni sulla scelta di eseguire una o due fasi di trattamento. *Mondo Ortod* 2011;36:166–174
4. Hall RE, Orbach S, Landesberg R. Bilateral hyperplasia of the mandibular coronoid processes: a report of two cases. *Oral Surg Oral Med Oral Pathol* 1989;67:141–145
5. Isberg A, Isacsson G, Nah KS. Mandibular coronoid process locking: a prospective study of frequency and association with internal derangement of the temporomandibular joint. *Oral Surg Oral Med Oral Pathol* 1987;63:275–279
6. Takahashi A, Hao-Zong W, Murakami S, et al. Diagnosis of coronoid process hyperplasia by three-dimensional computed tomographic imaging. *Dentomaxillofac Radiol* 1993;22:149–154
7. McLoughlin PM, Hopper C, Bowley NB. Hyperplasia of the mandibular coronoid process: an analysis of 31 cases and a review of the literature. *J Oral Maxillofac Surg* 1995;53:250–255
8. Capote A, Rodriguez FJ, Blasco A, et al. Jacob's disease associated with temporomandibular joint dysfunction: a case report. *Med Oral Patol Oral Cir Bucal* 2005;10:210–214
9. Moher D, Liberati A, Tetzlaff J, et al. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. *Ann Intern Med* 2009;151:264–269
10. Whiting P, Rutjes AW, Reitsma JB, et al. The development of QUADAS: a tool for the quality assessment of studies of diagnostic accuracy included in systematic reviews. *BMC Med Res Methodol* 2003;3:25
11. Wenghoefer M, Martini M, Allam JP, et al. Hyperplasia of the coronoid process in patients with ankylosing spondylitis (Bechterew disease). *J Craniofac Surg* 2008;19:1114–1118
12. Lyon LZ, Sarnat BG. Limited opening of the mouth caused by enlarged coronoid processes: report of case. *J Am Dent Assoc* 1963;67:644–650
13. Shira RB, Lister RL. Limited mandibular movements due to enlargement of the coronoid processes. *J Oral Surg (Chicago)* 1958;16:183–191
14. Gerbino G, Bianchi SD, Bernardi M, et al. Hyperplasia of the mandibular coronoid process: long-term follow-up after coronoidotomy. *J Craniomaxillofac Surg* 1997;25:169–173
15. Yamaguchi T, Komatsu K, Yura S, et al. Electromyographic activity of the jaw-closing muscles before and after unilateral coronoidectomy performed on a patient with coronoid hyperplasia: a case study. *Cranio* 1998;16:275–282
16. Allison M, Wallace R, von Wyl H. Coronoid abnormalities causing limitation of mandibular movement. *J Oral Surg* 1969;27:229–233
17. Turk AE. Moebius syndrome: the new finding of hypertrophy of the coronoid process. *J Craniofac Surg* 1999;10:93–96
18. Puche M, Guijarro-Martínez R, Pérez-Herrezuelo G, et al. The hypothetical role of congenital hypotonia in the development of early coronoid hyperplasia. *J Craniomaxillofac Surg* 2012;40:e155–e158
19. Oliveira MR, Rodrigues WC, Gabrielli MF, et al. Gardner syndrome with unusual maxillofacial manifestation. *J Craniofac Surg* 2016;27:1253–1255
20. Jaskolka MS, Eppley BL, van Aalst JA. Mandibular coronoid hyperplasia in pediatric patients. *J Craniofac Surg* 2007;18:849–854
21. Balkin DM, Chen I, Oberoi S, et al. Bilateral coronoidectomy by craniofacial approach for hecht syndrome-related trismus. *J Craniofac Surg* 2015;26:1954–1956
22. Khandavilli SD, Pattni N, Naredla PR, et al. First case of bilateral coronoid hyperplasia in monozygotic twin sisters-a new aetiological perspective? *Oral Maxillofac Surg* 2016;20:441–443
23. Rowe NL. Bilateral developmental hyperplasia of the mandibular coronoid process. A report of two cases. *Br J Oral Surg* 1963;1:90–104
24. Mano T, Ueyama Y, Koyama T, et al. Trismus due to bilateral coronoid hyperplasia in a child: case report. *J Oral Maxillofac Surg* 2005;63:399–401
25. Maspero C, Galbiati G, Giannini L, et al. Correlazione tra espansione rapida del palato e funzionalità respiratoria. *Dent Cadmos* 2010;78:87–105
26. Costello BJ, Edward SP. Pediatric mandibular hypomobility: current management and controversies. *Oral Maxillofac Surg Clin North Am* 2005;17:455–466
27. Shultz RE, Theisen FC. Bilateral coronoid hyperplasia. Report of a case. *Oral Surg Oral Med Oral Pathol* 1989;68:23–26
28. Kubota Y, Takenoshita Y, Takamori K, et al. Levandoski panographic analysis in the diagnosis of hyperplasia of the coronoid process. *Br J Oral Maxillofac Surg* 1999;37:409–411
29. Jamal BT, Taub D, Gold L. Controlateral coronoid hyperplasia in patients undergoing hemimandibulectomy with disarticulation: a case series. *J Oral Maxillofac Surg* 2009;67:1821–1825
30. Yura S, Ohga N, Ooi K, et al. Mandibular coronoid hyperplasia: a case report. *Cranio* 2009;27:275–279
31. Pregarz M, Fugazzola C, Consolo U, et al. Computed tomography and magnetic resonance imaging in the management of coronoid process hyperplasia: review of five cases. *Dentomaxillofac Radiol* 1998;27:215–220
32. Mavili E, Akyurek M, Kayikcioglu A. Endoscopically assisted removal of unilateral coronoid process hyperplasia. *Ann Plast Surg* 1999;42:211–216
33. Hayter JP, Robertson JM. Surgical access to bilateral coronoid hyperplasia using the bicoronal flap. *Br J Oral Maxillofac Surg* 1989;27:487–493
34. Ostrofsky MK, Lownie JF. Zygomatico-coronoid ankylosis. *J Oral Surg* 1977;35:752–754
35. Hernández-Alfaro F, Escuder O, Marco V. Joint formation between an osteochondroma of the coronoid process and the E zygomatic arch (Jacob disease): report of case and review of literature. *J Oral Maxillofac Surg* 2000;58:227–232
36. Mohanty S, Kohli S, Dabas J, et al. Fate of the coronoid process after coronoidotomy and its effect on the interincisal opening: a clinical and radiologic assessment. *J Oral Maxillofac Surg* 2017;75:1263–1273

37. Satoh K, Ohno S, Aizawa T, et al. Bilateral coronoid hyperplasia in an adolescent: report of a case and review of the literature. *J Oral Maxillofac Surg* 2006;64:334–338
38. Wallender A, Ahson I, Steinberg B. Neonatal coronoid hyperplasia: a report of a case and concepts to promote early diagnosis and treatment. *J Oral Maxillofac Surg* 2015;73:1615.e1–1615.e7
39. Gibbons AJ, Abulhoul S. Use of a Therabite appliance in the management of bilateral mandibular coronoid hyperplasia. *Br J Oral Maxillofac Surg* 2007;45:505–506
40. Baraldi CE, Martins GL, Puricelli E. Pseudoankylosis of the temporomandibular joint caused by zygomatic malformation. *Int J Oral Maxillofac Surg* 2010;39:729–732

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