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Case Report

Encephalotrigeminal Angiomatosis - Syndrome of Dental Interest: A Case Report

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Abstract

Encephalotrigeminal Angiomatosis or Sturge-Weber Syndrome is a rare malformation, which has several systemic manifestations. Its clinical features include neurological, ophthalmological, dermatological and stomatological alterations. One of the main craniofacial and stomatological manifestations is the presence of angiomatous lesion, characterized by "port wine" stains (nevus flammeo) on the skin of the face accompanying one or more paths of the trigeminal nerve branches. Because of the possibility of the occurrence of angiomatous lesion in the oral cavity, the dental surgeon's knowledge is essential for the performance of procedures, especially surgical ones. Considering the possibility of mental retardation, the patient with Encephalotrigeminal Angiomatosis becomes a special care patient, requiring the most adequate and specific dental treatment to your case. The purpose of this article is to present a clinical case of a patient with Encephalotrigeminal Angiomatosis focusing on the clinical characteristics and approach of the stomatological manifestations. Periodontal treatment was performed, with oral hygiene instruction and scaling and root planing sessions. Although the patient presented mild mental retardation and was not very responsive and collaborative to the periodontal treatment, the control of periodontal disease was achieved, with a 6-year follow-up.

Keywords: *Sturge-Weber Syndrome; Angiomatosis; Hemangioma; Dental Care; Oral Manifestations; Dentistry*

Introduction

Encephalotrigeminal Angiomatosis, also known as Sturge-Weber Syndrome, is a congenital malformation of developmental angiomatous, neurocutaneous, nonhereditary origin involving the brain and mesenchymal tissues of the face [1-5].

The combination of skin and brain angiomatous malformations can result from the poor development of vascularization that usually occurs in the 4th to 8th week of intrauterine life, when the ectoderm forms the upper part of the face near the neural tube [4,6]. The individual affected by the syndrome may present neurological, ophthalmological, dermatological and stomatological alterations that result from the probable disorder in migration and differentiation of tissues originating from the neural crest [7-10]. The Roach scale [11-14] classifies the angiomatous lesion levels, as well as the degree of complexity and areas affected in the patient who presents the vascular lesion on the face, and is summarized in table 1.

Type	Features/Involvement
I	Facial angioma; leptomeningeal involvement; possible glaucoma.
II	Facial angiomatosis; no Central Nervous System involvement; possible glaucoma.
III	Isolated involvement in leptomeningeal; unlikely glaucoma.

Table 1: *Roach scale [9-12] that classifies the degree of complexity and involvement of vascular lesions in encephalotrigeminal angiomatosis.*

The main dermatological alteration includes the presence of "port wine" stains (nevus flammeo) on the skin of the face accompanying one or more paths of the trigeminal nerve branches. The angiomatous lesion can extend to the labial commissure, maxillary region, orbit and portions of the frontal and parietal regions. In the oral cavity, the vascular lesion may involve hard and soft palates, alveolar ridge and oral mucosa, usually respecting the midline. Ipsilateral immature eruption of permanent teeth, macrodontia and ipsilateral lingual hypertrophy can also be found. Other stomatological manifestations can be observed as facial asymmetry and mouth rhyme deviation, caused by vascular hypertrophy [1-5,7,11-19,21-23].

Among the neurological manifestations, venous angiomatous masses can be found in the leptomeninge above the unilateral cerebral cortex, intracranial gyriform calcifications, varying degrees of mental retardation, epileptic seizures, hemiplegia or contralateral hemiparesis with hemisensory déficit [4,17,20,24-28]. Anticonvulsant drugs are usually administered. Neurological surgeries are reserved for refractory cases [3,4,26,27,29].

Among the ophthalmological disorders, glaucoma, buftalmos, coloboma, vascular malformations of the conjunctiva, sclera, retina and choroid can be verified [11,20,23,24,26].

The treatment of Encephalotrigeminal Angiomatosis is multiprofessional and its early diagnosis is fundamental for a better control of systemic manifestations, particularly neurological ones. It is worth mentioning that dental surgeon must have the knowledge of the clinical stomatological characteristics of this pathology, because to perform invasive procedures in patients with the syndrome, surgical planning is necessary, since hemorrhage can be a significant problem [4,10].

The purpose of this article is to present a clinical case of a patient with Encephalotrigeminal Angiomatosis focusing on the clinical characteristics and approach of the stomatological manifestations.

Case Report

A Caucasian male, 36-year-old, patient with Sturge-Weber Syndrome came to the clinic for periodontal treatment complaining of gingival bleeding and halitosis.

In the intraoral clinical examination, inadequate plaque control was observed, with the presence of biofilm and dental calculus, characterizing periodontitis. No intraoral hemangiomatous lesions were observed (Figure 1).

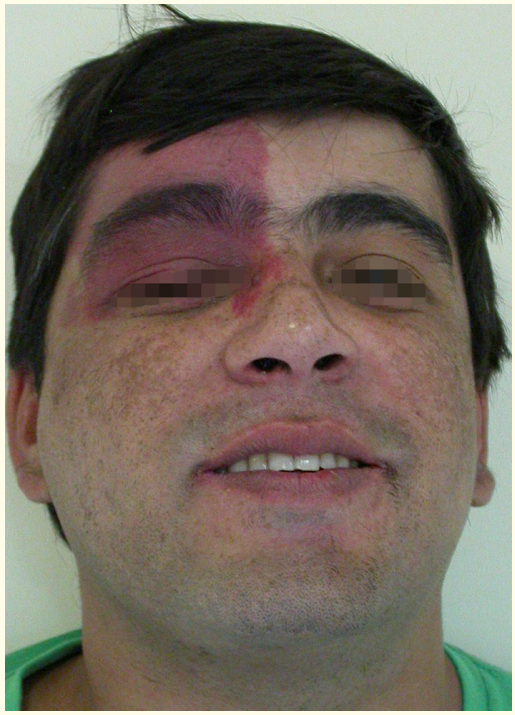

Figure 1: Intraoral clinical aspects: presence of biofilm and dental calculus, characterizing periodontitis.

Regarding the medical history, it was reported the occurrence of convulsive crises since 6 months of age, which were currently controlled with the daily administration of anticonvulsant drugs (Carbamazepine 200 mg; Primidone 100 mg; Topiramate 100 mg). However, the patient's mother reported sporadic seizures, often once a month.

The extraoral physical examination revealed the presence of congenital "port wine" stains on the face, in the region of ophthalmic nerve innervation on the right side. Bilateral angular queillitis was also observed. The patient presented hemiplegia on the right side of the face, exophthalmia on the left, strabismus, dystopia, absence of lip seal and mild mental impairment (Figure 2).

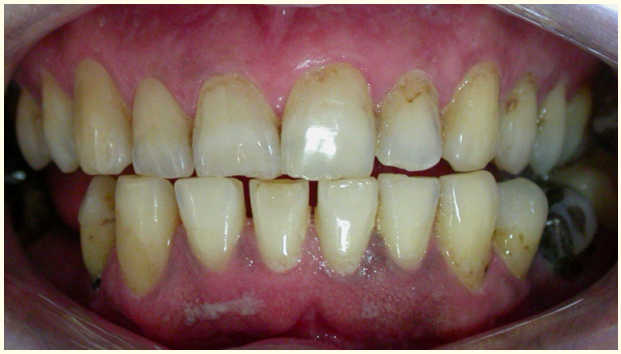

Figure 2: Extraoral clinical aspects: note the "port wine" stains on the face, in the region of ophthalmic nerve innervation on the right side.

Regarding the routine radiographic examination, the radiolucent images generalized on panoramic radiography refer to bone loss caused by periodontitis (Figure 3).

The patient was systematically evaluated and no preventive therapy for hemorrhage was required. Serological and biochemical laboratory tests were performed and no marked alterations (blood dyscrasias) were presented.

Before periodontal treatment, the depth of the periodontal pocket was measured, generally ranging from 3 to 4 mm. It was recommended the orientation of oral hygiene to the patient and to his mother, being evidenced with erythrosine (Figure 4) and instructing him with electric brush and dental floss. The improvement of oral hygiene procedures was essential to perform the periodontal procedure, bringing about an improvement in the gingival bleeding due to gingival inflammation.

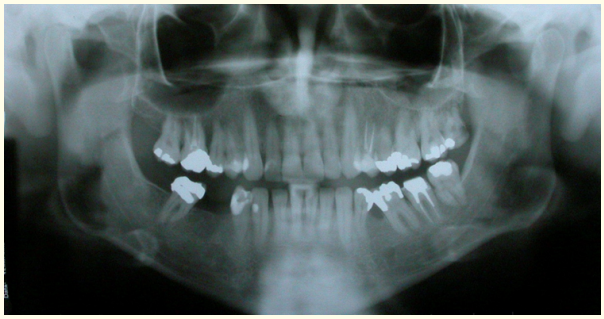

Figure 3: Radiolucent images inherent to bone loss due to periodontitis.

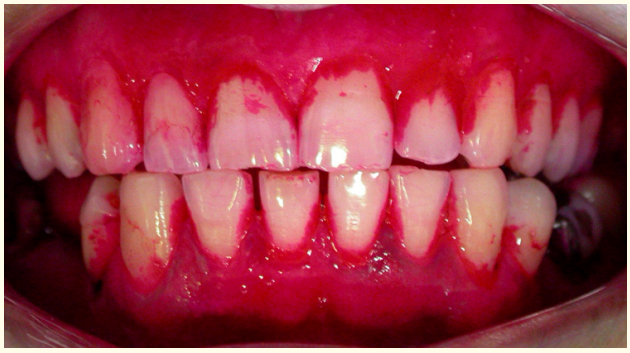

Figure 4: Instruction of oral hygiene after use of erythrosine.

Although the patient had mild mental retardation, he presented a cognitive response, although not very responsive and collaborative. The patient has been followed for 6 years, without signs of recurrence of periodontal changes, but under strict periodontal control, especially reinforcements in oral hygiene orientation. After periodontal treatment, the depth of the periodontal pocket was

measured, ranging from 2 to 3 mm. However, clinical characteristics such as the absence of bacterial plaque were more indicative of periodontal control.

Discussion

Encephalotrigeminal Angiomatosis was first described in 1879 by Willian Allen Sturge, who reported a case of a patient who presented as clinical features, facial skin nevus, convulsions, ocular alterations and mental retardation. In 1922, Francis Parker Weber published the radiographic alterations that can be found, such as intracranial calcifications. Since then, the disease has been known as Sturge-Weber Syndrome [10,15,19].

The diagnosis of Encephalotrigeminal Angiomatosis in this case was confirmed by the presence of the "Port Wine" stain, and classified as grade I of the Roach Scale, by the presence of neurological alterations or other associated lesions. The "Port wine" stains are representative signs of the disease, and comprise the vascular malformation of the dermis capillarity. These stains accompany topographically the innervation of one or more trigeminal nerve branches. In a retrospective study of 106 cases of facial "Port wine" stains, it was shown that the location in the ophthalmic branch was the most incident in Encephalotrigeminal Angiomatosis, followed by the location in maxillary and mandibular branches [7]. Unilateral involvement is more common and staining can range from pink to purple-red. In the present case, the patient presented involvement of the right ophthalmic branch with violaceous stains, with clear limits.

Among the oral manifestations of the syndrome, the occurrence of vascular malformations in the oral mucosa is one of the main findings. These angiomatoses occur preferentially in the labial mucosa, jugal mucosa and alveolar ridge [8,18,28,29]. The clinical presentation of these lesions is variable. In the present report, there was no involvement of the angiomatous lesion in the oral cavity. Additionally, it is worth remembering that the concomitant presence of gingival overgrowth due to the use of anticonvulsant drugs is possible. This patient also did not show gingival overgrowths. If necessary, basic (how it was done in this case) and surgical (gingivoplasty) periodontal treatment must be performed. It is also important to emphasize the importance in the differential diagnosis between the volumetric increase caused by lesions of vascular origin (angiomatoses) of the inflammatory gingival overgrowth - caused secondarily by the administration of anticonvulsant drugs. Such distinction is important, since hypervascularized lesions require special care in their management [11-14,20,22].

The treatment methods described for angiomatous lesions include surgical excision, sclerosing agent injection, electrodissection, radiotherapy and CO_2 laser, which should be particularized case by case [19,30,32]. Of all the proposed treatments, the surgical one is the one that promotes the fastest and most definitive resolution for the case. However, special precaution should be given when performing the surgical procedure, since hypervascularization presents a high risk of hemorrhage that is difficult to control trans-surgery. Sometimes, angiographically controlled vascular embolization is necessary, being useful in these conditions in the prevention of undesirable hemorrhagic accident [33].

The patient has been followed for 6 years, presenting no gingival or angiomatous lesions. It must be emphasized that the treatment of the patient with Encephalotrigeminal Angiomatosis must be multiprofessional, and certain cares such as the accomplishment of an adequate anamnesis, correct diagnosis of the oral manifestations and their respective conducts, and prevention of hemorrhagic accidents during the surgical procedure (when necessary), favor the dental treatment that privileges the safety and the well-being of the patient with this disease.

Conclusion

Patients with Encephalotrigeminal Angiomatosis must receive multidisciplinary treatment for the clinical manifestations presented. From the most basic procedures, such as the orientation of oral hygiene and periodontal treatment itself, to advanced surgical procedures - when affected by the angiomatous lesion - must be rigorously evaluated to prevent possible surgical complications.

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Volume 4 Issue 3 March 2021

A mandibular molar tooth with acute irreversible pulpitis is one of the most disconcerting situations to be encountered in an endodontic clinic [1]. Treatment options for these situations are limited to either emergency access opening or prescribing potent analgesics [1]. Achieving satisfactory anesthesia and reducing the incidence of post-treatment discomfort are the difficulties encountered in clinical treatment of these teeth [1]. Multiple strategies have been explored for attaining profound anesthesia and to control post-operative pain in these situations [2]. Pre-operative use of analgesics has been the most investigated strategy in the literature [2,3]. However, there is a lack of scientific evidence regarding the efficacy of oral pre-operative analgesics in controlling both the intra-operative and post-operative pain following single visit root canal treatment for mandibular teeth with acute irreversible pulpitis. An earlier report from the concluded that pre-operative ketorolac tromethamine was not effective in reducing the intra-operative pain for mandibular molar teeth with acute irreversible pulpitis when inferior alveolar nerve blocks (IANB) with both lignocaine and articaine anesthetic agents were used. However, pre-operative ketorolac tromethamine was effective in reducing post-operative pain in the lignocaine anesthetic group [4]. Furthermore, pre-operative ketorolac tromethamine prior to single visit root canal treatment showed no significant difference in post-treatment pain between the different irrigation groups [4]. These findings are in agreement with reports on the role played by root canal irrigants in controlling post-operative pain in teeth with acute irreversible pulpitis being inconclusive [5-8].

A literature search has shown that there is a gap in understanding about the efficacy of pre-operative ketorolac tromethamine in improving the success of local anaesthetics and for the management of post-operative pain following single visit root canal treatment in mandibular molar teeth with acute irreversible pulpitis. Furthermore, no scientific evidence is available on the role played by different local anesthetic agents on post-treatment pain following single visit root canal treatment.

Aim of the Study

The present study was planned with the primary aim to compare the anesthetic efficacy of lignocaine and articaine IANB with additional injections for single visit root canal treatment for mandibular molar teeth with acute irreversible pulpitis following pre-operatively administered oral ketorolac tromethamine. A second objective was to explore the efficacy of these two different local anesthetics in controlling post-operative pain following single visit root canal treatment when employed with three different root canal irrigants.

Materials and Methods

A sample size of 126 patients was calculated to be sufficient to detect clinical data difference (alpha error of 0.05, power of 95% and effect size 0.4) (G power 3.1.9.2. software, Germany). The period of study was from November 2018 to January 2020 and 128 patients were recruited. After gaining approval from the institutional ethics committee [CSICDSR/IEC/0052/2018] and the trial was registered with the Clinical Trial Registry of India [CTRI/2019/10/021597]. The patients (or where appropriate, parents or guardian) were informed about the nature of the treatment and the study, and they were asked to sign an informed consent form. The methodology adopted for this study is similar to a previous experiment in the authors' department with the same operator.

Patients referred to the Department of Conservative Dentistry and Endodontics with pain due to acute irreversible pulpitis from carious mandibular first and second molar teeth requiring root canal treatment were evaluated as possible candidates for this study. Subjects aged between 13 and 70 years with no intake of medications for pain relief in the previous 10 days prior to treatment were included in the study. All patients reported mild to severe pain that was continuous, spontaneous, radiating, nocturnal or throbbing in nature. All teeth included in this study responded to cold pulp sensibility testing (Endo-Frost, Coltene Whaledent, Switzerland) with exaggerated pain, with or without lingering. They also had tenderness on percussion. In addition, profuse bleeding was evident upon gaining access into the pulp chamber. The teeth included in the study also did not have any evidence of periapical bone changes in the pre-operative periapical radiographs.

Exclusion criteria were teeth with poor periodontal or restorative prognosis, patients with systemic ailments or conditions hindering single visit root canal treatment, patients not willing to participate in the post-operative recall evaluation, any anatomic variation such as extra roots or root canals, C-shaped roots, and patients with a history of allergy.

In the period from November 2018 to April 2019, 64 patients underwent single visit root canal treatment with 2.5 mL of 2% lignocaine containing 1:80,000 adrenaline (Lignox, Warren Pharmaceuticals, Mumbai, India) for an inferior nerve alveolar nerve block (IANB) plus a 1.5 mL buccal infiltration and 0.1 to 0.2 mL intra-ligamentary injection using the same anesthetic agent (lignocaine group). The intra-ligamentary injections were given at four sites for each tooth on the buccal and lingual sides. From May 2019 to January 2020, another 64 patients underwent single visit root canal treatment using 2.5 mL of 4% articaine with 1:100,000 adrenaline (Septodont Healthcare Pvt Ltd, Raigod, India) for an inferior alveolar nerve block (IANB) plus buccal infiltration and intra-ligamentary injections as described above (articaine group). Subjects were allotted to three different irrigation groups - saline, sodium hypochlorite, or dexamethasone. Allocation to the irrigation groups was done by block randomization according to pre-operative pain intensity (mild, moderate or severe). Randomisation was done by a dentist who was not involved in the study. Figure 1 explains the methodology flowchart.

All the root canal procedures were done by a single operator blinded to the irrigation allotment. The levels of pre-, intra- and post-operative pain at 24 hrs and 48 hrs for each patient were recorded using a 10-point visual analog scale (VAS). The participants indicated the intensity of their pain by choosing a number using the following values: levels 1 - 3, mild pain; levels 4 - 7, moderate pain; and levels 8 - 10, severe pain. All patients in this study were given a 10 mg of ketorolac tromethamine tablet (Dr. Reddy's Laboratories Ltd, Solan (HP) India) which was taken orally 45 mins prior to local anaesthetic administration.

An intra-dermal injection of 0.2 mL of the local anesthetic agent to be used was given prior to the IANB in order to rule out any allergy to the anaesthetic agents. Local anesthesia with 2.5 mL of 2% lignocaine containing 1:80,000 adrenaline or 4% articaine with 1: 100,000 adrenaline was administered seven minutes prior to commencing the root canal procedure. Cold pulp sensibility tests and percussion evaluations were performed after enquiring about the level of lower lip numbness and before the access opening was initiated. Responses to these tests were also recorded. If sufficient anesthesia was not attained, an additional IANB with the same anesthetic agent was administered.

Working length was determined using a Root ZX Mini Apex Locator (J Morita, Kyoto, Japan) and Aurum Profiles (Meta Biomed, Co. Ltd, Incheon, Korea) were used for root canal preparation according to the manufacturer's instructions with an Endomate DT motor (NSK Inc.,Tochigi, Japan). Canal lubrication and smear layer management were done with EDTA (10%) and carbamide peroxide (15%) (Endoprep RC, Anabond Stedman Pharmaceuticals, Chennai, India). Depending on the participant's allotment to the irrigation groups, saline (NS 500 mL, Sodium chloride 0.9%, Fresenius Kabi, Pune, India Pvt. Ltd), 3% sodium hypochlorite (Septodont Healthcare India Pvt. Ltd, Raigad, India) or dexamethasone sodium phosphate (Dexalab Inj 2 mL, Laborate Pharmaceuticals, Sahib (H.P), India) were used as irrigants during the root canal preparation procedures. In all cases, 2 mL of saline with 2% povidone-iodine (Puradine, Leeford Healthcare ltd, Mumbai, India) was used as the initial irrigant and this was followed by the interventional irrigation solutions - saline, sodium hypochlorite or dexamethasone, according to the group allocation- as mid-treatment rinses using 1.5 mL for each canal. Then, a final irrigation of each canal was performed with 2 mL of saline with 2% povidone-iodine solution. Initial irrigation of the root canal was done after glide path establishment upto size 20 or 25K-file (Mani, Co., Tokyo, Japan). Mid-treatment rinses and final irrigation were employed after the use of rotary instruments. A total of 6 mL of irrigation solution was used in each canal during the treatment. The interventional irrigation solutions were delivered inside the root canal using a side-vented 25-gauge needle

Section/Topic		Item No	Checklist item	Reported on page No
	Title and abstract			
		1a	Identification as a randomised trial in the title	
	1b	Structured summary of trial design, methods, results, and conclusions (for specific guidance see CONSORT for abstracts)	1	
	Introduction			
Background and objectives		2a	Scientific background and explanation of rationale	3
	2b	Specific objectives or hypotheses	4	
	Methods			
Trial design		3a	Description of trial design (such as parallel, factorial) including allocation ratio	6
	3b	Important changes to methods after trial commencement (such as eligibility criteria), with reasons	Not applicable	
Participants		4a	Eligibility criteria for participants	4, 5
	4b	Settings and locations where the data were collected	4	
Interventions		5	The interventions for each group with sufficient details to allow replication, including how and when they were actually administered	5 - 8
Outcomes		6a	Completely defined pre-specified primary and secondary outcome measures, including how and when they were assessed	8
	6b	Any changes to trial outcomes after the trial commenced, with reasons	Not applicable	
Sample size		7a	How sample size was determined	4
	7b	When applicable, explanation of any interim analyses and stopping guidelines	Not applicable	
Randomisation				
Sequence generation		8a	Method used to generate the random allocation sequence	6
	8b	Type of randomisation; details of any restriction (such as blocking and block size)	Not applicable	
Allocation concealment mechanism		9	Mechanism used to implement the random allocation sequence (such as sequentially numbered containers), describing any steps taken to conceal the sequence until interventions were assigned	6
Implementation		10	Who generated the random allocation sequence, who enrolled participants, and who assigned participants to interventions	6

Blinding		11a	If done, who was blinded after assignment to interventions (for example, participants, care providers, those assessing outcomes) and how	Not applicable
	11b	If relevant, description of the similarity of interventions	Not applicable	
Statistical methods		12a	Statistical methods used to compare groups for primary and secondary outcomes	9
	12b	Methods for additional analyses, such as subgroup analyses and adjusted analyses	9	
	Results			
Participant flow (a diagram is strongly recommended)		13a	For each group, the numbers of participants who were randomly assigned, received intended treatment, and were analysed for the primary outcome	10
	13b	For each group, losses and exclusions after randomisation, together with reasons	10, Figure 1	
Recruitment		14a	Dates defining the periods of recruitment and follow-up	5, 6
	14b	Why the trial ended or was stopped	6	
Baseline data		15	A table showing baseline demographic and clinical characteristics for each group	Not applicable
Numbers analysed		16	For each group, number of participants (denominator) included in each analysis and whether the analysis was by original assigned groups	10
Outcomes and estimation		17a	For each primary and secondary outcome, results for each group, and the estimated effect size and its precision (such as 95% confidence interval)	10, 11
	17b	For binary outcomes, presentation of both absolute and relative effect sizes is recommended	Not applicable	
Ancillary analyses		18	Results of any other analyses performed, including subgroup analyses and adjusted analyses, distinguishing pre-specified from exploratory	11
Harms		19	All important harms or unintended effects in each group (for specific guidance see CONSORT for harms)	Not applicable
	Discussion			
Limitations		20	Trial limitations, addressing sources of potential bias, imprecision, and, if relevant, multiplicity of analyses	16
Generalisability		21	Generalisability (external validity, applicability) of the trial findings	15
Interpretation		22	Interpretation consistent with results, balancing benefits and harms, and considering other relevant evidence	11 - 16

	Other information			
Registration		23	Registration number and name of trial registry	4
Protocol		24	Where the full trial protocol can be accessed, if available	http://ctri.nic.in/Clinicaltrials/pmaindet2.php?trialid=27074&EncHid=&userName=CSI%20College%20of%20Dental%20Sciences
Funding		25	Sources of funding and other support (such as supply of drugs), role of funders	Nil

**We strongly recommend reading this statement in conjunction with the CONSORT 2010 Explanation and Elaboration for important clarifications on all the items. If relevant, we also recommend reading CONSORT extensions for cluster randomised trials, non-inferiority and equivalence trials, non-pharmacological treatments, herbal interventions, and pragmatic trials. Additional extensions are forthcoming: for those and for up to date references relevant to this checklist, see www.consort-statement.org.*

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