

Review Article

The Talon Cusp Anomaly – Review of The Literature And Treatment Options

Dinesh Francis Swamy¹, Elaine Savia Barretto², Sapna Sada Raut Dessai³, Kathleen Manuela D'Souza⁴, Kennedy Mascarenhas⁵, Sheen Ann Alex⁶

^{1,2} Department of Pedodontics & Preventive Dentistry, Goa Dental College & Hospital, Bambolim, Goa

³ Department of Oral Medicine and Radiology, Goa Dental College & Hospital, Bambolim, Goa

^{4,5} Department of Prosthodontics, Goa Dental College & Hospital, Bambolim, Goa

⁶ Department of Pedodontics & Preventive Dentistry, PMS College of Dental Science & Research, Thiruvananthapuram, Kerala

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ABSTRACT

Talon cusp is a developmental anomaly which occurs as a result of disturbances in the complex process of tooth development. The clinical problems caused by the anomaly may be diagnostic, functional, aesthetic or pathological. Treatment should be planned depending on the configuration and size of the talon cusp and during tongue of occlusal interferences, irritation associated clinical problems such as attrition, speech or mastication, development of dental caries and it ranges from nil treatment, conservative treatment such as cusp reduction and restoration, to intentional endodontics. This paper reviews the literature associated with the anomaly and their conservative management.

Introduction

Aberrations occurring in the morpho-differentiation stage of tooth development can result in unique manifestations such as mulberry molars, peg laterals, and also, talon cusps. The Talon cusp anomaly, according to Hattab^[1], may originate as a result of outward folding of inner-enamel epithelial cells (precursor of ameloblasts) and/or transient focal hyperplasia of the mesenchymal dental papilla (precursor of odontoblasts).

This phenomenon was first described by Mitchell in 1892^[2] and the term “Talon cusp” was coined by Mellor and Ripa in 1970^[3] because of its resemblance to an eagle’s talon. The accessory cusp, if connected to the incisal ridge may form a ‘T’ shape, or if attached more cervically, a ‘Y’ shape may result. Varying degrees of the anomaly exist and have attracted various names – ‘enlarged/prominent cingula’ for lesser cusp-like formations in the cingulum area, ‘hyperplastic cingulum’, ‘cusped cingulum’, Leong’s

* Corresponding author: *Dr. Dinesh Francis Swamy, Department of Pedodontics & Preventive Dentistry, Goa Dental College & Hospital, Bambolim, Goa*

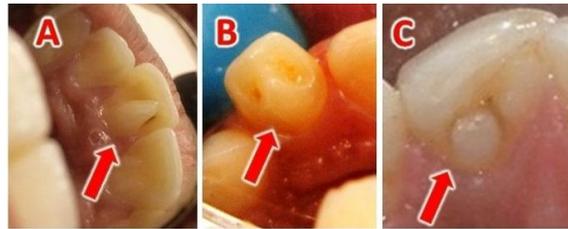


Fig 1: Different degrees of Talon Cusp: A – Type 1/Major Talon; B – Type 2/Minor Talon; C – Type 3/Trace Talon (from Mull P, 2016^[63])

premolar, occlusal pearl, evaginatus odontoma and ‘supernumerary lingual tubercle’. Electronic literature search of the MEDLINE database via Pubmed was done for scientific papers published in English from 1990 to 2016. Eligible studies were Case Reports, Case Series and Epidemiological studies describing the management of talon cusp. The search equation combined free text words and controlled vocabulary pertaining to the condition (Talon Cusp, Dens Evaginatus, talon AND cusp). The search yielded 99 articles. Manual checking of the reference list was done for relevant studies. This article attempts to reviews the literature associated with the anomaly and discusses the various clinical problems encountered and their conservative management.

Prevalence:

Talon cusps occur more frequently in the permanent dentition than the primary dentition – three times more according to a 1996 study by Hattab.^[4] Numerous studies have attempted to estimate the prevalence of the talon cusp anomaly. Prevalence ranging from 0.06% in Mexico, 0.17% in the United States, 5.2% in Malaysia, 1-2.4% in Pakistan and 7.7% in a north-Indian population have been reported.^{[5],[6],[7],[8],[9]} However, race seems to influence the incidence of Talon cusp considerably, with greater incidence seen in Mongoloid populations.^[10] The gender distribution shows more frequent occurrence in males than in females, with a ratio

of 1.8:1 in the permanent dentition and 3.5:1 in the primary dentition.^[11] Literature reports describing the occurrence of the anomaly indicate that the site-distribution involves the maxilla predominantly, with the maxillary lateral incisors most frequently involved (67%), followed by the maxillary central incisors (24%) and canines (9%).^[11] About one fifth of all cases are bilateral^[12].

In the literature, numerous conservative or radical treatment modalities have been advocated to manage Talon cusps. The chief objective of treatment is the avoidance of occlusal interference, and the main treatment consideration is the avoidance of damage to the dentine-pulp complex. This paper reviews the literature associated with the anomaly and discusses the various clinical problems encountered and the various modalities of management of Talon Cusp anomaly.

Classification

Since the beginning, there has been significant heterogeneity in the description of the talon cusp anomaly. Some authors believe that the term ‘dens evaginatus’ can be used in place of the Talon anomaly – as it is more descriptive of the pathology; while others believe its use should be exclusively confined to similar features affecting the posterior dentition.^{[13],[14],[15]}

The anomaly has been more consistently described following the use of classification systems. With the

recognition of talon cusp occurrence on facial surfaces, in addition to lingual surfaces, the older classification of Hattab and co-workers was modified by Hsu & co-workers based on the degree of their formation and extension as:^{[4],[16],[17],[18]}

Type 1 – Major talon: A morphologically well-defined additional cusp that projects from the facial or palatal/lingual surface of an anterior tooth and extends at least half the distance from the cemento-enamel junction to the incisal edge. (Fig 1A)

Type 2 – Minor talon: A morphologically well-defined additional cusp that projects from the facial or palatal/lingual surface of an anterior tooth and extends more than one fourth, but less than half the distance from the cemento-enamel junction to the incisal edge. (Fig 1B)

Type 3 – Trace talon: Enlarged or prominent cingula and their variations which occupy less than one fourth the distance from the cemento-enamel junction to the incisal edge. (Fig 1C)

Etiopathogenesis

Cusp morphogenesis is controlled initially, by the primary enamel knot during bud-stage, and thereafter, by the secondary enamel knot during late-bell stage, through expression of certain signalling proteins.^[19] These specific molecules diffuse from the mesenchymal cells and interact with various cell types and are thought to be responsible for orderly formation of the various parts of the tooth. Hence, defects occurring at various points in this developmental sequence can be implicated in the genesis of anomalies of cusp number and size.^[20]

The talon cusp anomaly has been reported in patients with certain syndromes; in non-syndromic patients along with additional dental malformations; and, in an isolated manner in normal individuals with no associated abnormalities – with the latter constituting the usual

mode of occurrence. Cases of Talon cusps have been reported in patients with Rubinstein-Taybi syndrome, Mohr syndrome, Sturge-Weber syndrome, Ellis van Creveld Syndrome.^{[21],[22],[23],[24]} Given the fact however, it has not been conclusively proven that an association between such syndromes and talon cusp exists, and talon cusp is not reported as an integral part of any syndrome. Similarly, occurrence along with other dental malformations such as peg-shaped lateral incisors, impacted mesiodens, complex odontoma, supernumerary teeth, megadont, dens invaginatus, shovel shaped maxilla incisors, bifid cingula, exaggerated cusps of Carabelli have been reported in the literature.^{[4],[14],[15],[25],[26],[27],[28],[29]}

These facts suggest that genetics may be a major causative factor. However, the usual sporadic occurrences of the abnormality are speculated to be caused by trauma, local pressure effects from adjacent tooth germs, or other localized insults affecting the tooth germ.^{[12],[30],[31]} Notwithstanding the exact underlying mechanism, there is undoubtedly an abnormal folding of a region of the inner-enamel epithelium and adjacent ectomesenchymal cells of the dental papilla into the stellate reticulum of the enamel during the bell-stage. It is thought that the high incidence of occurrence in the lateral incisors is due to compression of the tooth germ during the morpho-differentiation stage between the adjacent central incisors and canine, which develop several months earlier.^[18] The focal outfolding of the inner-enamel epithelium does not affect the architecture of the rest of the tooth and there is no alteration in the root canal system of the tooth. Also, there is no communication between the pulp and the oral cavity unless concomitantly involved with other invaginating anomalies.

Complications

The complications of talon cusp are diagnostic, functional, aesthetic and pathological. These are elaborated below. Additionally a summary of the literature is presented. (Table 1)

1. Diagnostic complications:

The outline of a talon cusp can mimic a supernumerary tooth, and since most supernumeraries occur in the maxillary anterior region it is essential to differentiate between the two. Reports on misidentification of talon cusp as supernumerary teeth, treated by accidental extraction serve to highlight the need for meticulous pre-operative assessment.^{[18],[32]} In the clinic, it has been suggested that this differentiation may be done by employing a shift-cone technique, whereby the talon cusp presents as incisally-converging radio-opacity in multiple angulations, whereas the supernumerary presents as separate images of teeth and follicles.^[23]

The presence of a horn of pulpal tissue within the talon cusp has clinical implications for any operative procedures, but the reliable presence of such pulpal extensions are not evident in several histologic studies of extracted talon cusp teeth.^{[14],[33]} To further complicate clinical-planning, radiographic tracing of the pulpal configuration inside the talon cusp is inherently difficult as the cusp is superimposed over the affected tooth crown, making accurate pre-operative estimation of the extent of permissible cusp removal rarely possible.^[33] This can be overcome with the use of newer imaging techniques such as Cone Beam Computed Tomography that offer higher resolution, greater sharpness and permit volume rendering.^{[34],[35]}

2. Functional complications:

Occlusal interference when present can produce numerous functional complications affecting both the

hard and soft tissues. The goal of treatment is therefore to reduce the offending cusp, while preserving the normal vitality and continued-growth of the tooth and managing any complications encountered.

Functional complications to soft tissues include irritation to the tongue or compromised tongue space, speech disturbances or difficulty in feeding.^{[1],[36],[37],[38]}

The prominent cusp may also come into premature contact during occlusion – such hyper-occlusion can result in damage to the cusp itself – resulting in varying degrees of trauma, ranging from attrition to cusp-fracture, to temporomandibular joint disorders due to the deflecting premature contact.^[39] Hyper-occlusion may also cause damage to the opposing tooth, causing attrition and primary trauma from occlusion, and subsequently damage to the periodontium (i.e. secondary trauma from occlusion) accompanied by tenderness, mobility and attachment loss.^[40]

3. Aesthetic complications:

Unlike the evaginatus-anomaly on posterior teeth, the talon cusp is much less likely to encounter crushing masticatory-forces. Rather than resulting in cusp-fracture, the oblique vector of forces acting on both the talon cusp-affected tooth and its opposing counterpart are more likely to produce their displacement and/or malocclusion.^[41] Talon cusp could also present an obstacle for mandibular growth, holding the mandible back and causing and causing Class II malocclusions.^[37] Such malocclusions are treated by a removal of the interfering part/entire Talon cusp followed by appropriate appliance therapy, either removable or fixed depending on the malocclusion.^{[42],[43]}

4. Other pathological complications:

Developmental grooves are susceptible to plaque accumulation and predispose to dental caries.^[12] Their extension onto the root can also lead to gingival inflammation and/or attachment loss or pocketing necessitating periodontal and restorative treatment.^{[1],[44]}

Management:

Management of talon cusp ranges from nil-intervention to simple cusp-reduction to cusp-reduction with pulp-therapy. The degree of pulpal involvement and status of the pulp would influence the choice of either conservative pulp procedures such as pulp-capping or pulpotomy, or more radical pulp procedures. The various variables to consider while planning treatment are elaborated.^{[37],[45],[46],[47]}

1. Extent of cusp-reduction:

Cusp reduction is carried out with a high-speed handpiece using abundant coolant. In performing the reduction, it is essential to check the contact in maximum intercuspation and also in protrusive movements. The extent of cusp reduction that is permissible varies according to literature. Pitts and Hall^[43] have reported the removal up to 3mm of anomalous cusp in one visit, without pulp exposure, while Hattab and co-workers have reported the removal of 1-1.5mm of cusp in several instances.^{[1],[33],[36]} A systematic review found that in 56% of case reports analysed complete reduction was done in a single appointment; periodic reduction in 26% of cases; abstinence in 13% and extraction in 5%.^[48] However, since the pulp-horn may infrequently extend up to the dentino-enamel junction such techniques may not be relied upon exclusively, or without adequate imaging.

Excessive grinding may result in inadequate remaining-dentine-thickness placing the pulp at risk of insult and subsequent necrosis. In such cases, the layering of an acid-etched flowable light-cured resin or glass ionomer applied after cusp-reduction on any exposed dentin is indicated. Reports exist where cusp-reduction without subsequent restoration has resulted in early pulpal involvement.^[49]

2. Interval between cusp-reduction appointments:

Studies have shown that the rate of tertiary-dentin formation varies from about 0.74µm to 3.5µm daily (Average daily reparative dentin formation has been reported to be 2.85µm for primary and 1.5 µm for permanent teeth.^[31] Mechanical stimulation that recruits the maximum number of odontoblasts is desirable. Since the odontoblasts are oriented along the length of the cusp, reduction done on the side of the cusp, rather than at the tip, is likely to yield maximum reactionary dentin deposition.^[50] It is important to note however that process produces a 'narrowing' of the pulp horn, rather than a 'recession' of the pulp. Ideally, after every instance of cusp-reduction, a sensitizing/remineralizing agent should be applied to occlude open dentinal tubules. The interval of 6-8 weeks in between appointments allows sufficient time for tertiary dentine deposition, and additional time-interval produces further pulp-horn obliteration. Extensive reduction in excess of 2/3rd of talon cusp over a prolonged treatment period of 5yrs (four-month recall intervals) has been reported in literature.^[51]

Levitan^[52] has suggested that this technique be employed at 6-month intervals when the tooth in question has a mature apex and a normal (non-inflamed) pulp, and at 3- or 4-month intervals in teeth with immature apices. However, in cases where there the pulp is inflamed or necrotic, radical pulp therapy is

recommended. This entails performing conventional root-canal treatment in teeth with mature apices, or apexification procedure in cases with immature apices, using Mineral Tri-Oxide Aggregate (MTA) or Calcium Hydroxide (CaOH).^{[23],[47],[53]} In obtaining access to the root canal, the access-cavity outline can involve the talon cusp so that the offending cusp is removed during the course of root-canal treatment. Also, since the anomaly is a localized enamel outfolding, the subsequent procedure of the root-canal is uneventful and routine in term of canal negotiation, irrigation and obturation.

Between these two often used endodontic medicaments, MTA has proven to be superior to CaOH when employed for pulp-capping, pulpotomy and apexification procedures. It has largely overcome the concerns of CaOH use i.e. time-duration for barrier induction, CaOH-related weakening of dentin structure, vascular inclusions in barrier, and formation of zones sterile pulp necrosis. MTA shows a more bacteria-tight seal by induction of a hard tissue bridge with lesser defects or porosities, superior adhesion to dentin, and greater radicular-dentine reinforcement.^{[54],[55]} Also, MTA has a faster rate of induction and is more biocompatible. In a study by Tziafas^[56], MTA was able to induce crystalline directly at the pulp-MTA interface without any intervening zone of necrosis within only 1 week, and could induce cellular dentin within 3-4 weeks. Additionally, the success rates of MTA are superior to those of CaOH in clinical studies – in pulpotomies (CaOH – 95% avg. success-rate vs. MTA – 100% avg. success-rate), pulp-capping's (MTA 98% success-rate), and apexifications (CaOH – 95% avg. success-rate Vs. MTA – 89% avg. success-rate). In cases of

Talon cusp, the use of MTA over CaOH has been suggested by Koh et al.^[57]

Regarding the choice of material after occlusal grinding and pulp-medicament, incremental layers of micro-hybrid and micro-fill flowable light-cured resin materials offer the best abrasion resistance, with ease of applicability. Resin modified glass ionomers also offer similar benefits. In cases where exposure/near-exposure is not a concern, acid-etching is also thought to accelerate the rate of tertiary-dentin formation.^[31]

Prophylactic Maintenance:

Maintenance of oral hygiene, even in trained patients, is challenging owing to the grooves present on either side of the junction of the talon cusp and palatal (or labial) surface of the tooth. These sites are predisposed to plaque build-up, staining and have higher caries susceptibility. Patients benefit from emphasis on plaque-control and counselling on periodic check-ups, and the application of fluoride and/or sealants is justified in patients whose etiologic factors and disease determinants are not well controlled. When required, pre-prophylaxis with an abrasive slurry and/or invasive sealant (with fissurotomy/enameloplasty) application may also be employed.^[42]

The application of an agent such as fluoride or GC Tooth Mousse™ (GC Corporation, Tokyo, Japan), promotes remineralisation, reduces sensitivity, stimulates reparative dentin formation, and increases the local resistance of the enamel to acid dissolution.

In cases where the talon cusp is small, not compromising aesthetics, not interfering with occlusion or contributing to malocclusion or soft-tissue irritation, and the site is deemed sufficiently maintainable by the patient, then 'no-intervention' is perfectly acceptable.

CONCLUSION

The aim of this case report was to illustrate the conservative management of a case of Talons cusp, to avoid complications related with this condition. A review of the literature is also presented to further an

understanding of the anomaly, its associated pathoses and their management. The patient's degree of compliance must also be taken into account during treatment planning as conservative management

Table 1: Summary review of literature with problems reported and treatments proposed

Problems reported in cases of Talon Cusp	Treatments proposed in Literature
<ul style="list-style-type: none"> • Small, inconsequential 	<ul style="list-style-type: none"> • No treatment^{[12],[36]}
<ul style="list-style-type: none"> • Radiographic misinterpretation (as supernumerary, compound odontoma, dens in dente)^[13] 	<ul style="list-style-type: none"> • Care in diagnosis^[18]
<ul style="list-style-type: none"> • Susceptibility to Caries 	<ul style="list-style-type: none"> • Prophylactic cleaning of grooves^[11] + Sealant^{[11],[39],[58]} • Placement of Sealants alone^[18]
<ul style="list-style-type: none"> • Compromised aesthetics^[11] 	<ul style="list-style-type: none"> • Aesthetic Restorations
<ul style="list-style-type: none"> • Dental Caries^{[12],[58]} 	<ul style="list-style-type: none"> • Enameloplasty and Restoration:^[58] <ul style="list-style-type: none"> ○ With GIC^{[11],[39]} ○ With RMGIC^[59] ○ With Composite^[11] ○ With Full-coverage Crown^[60]
<ul style="list-style-type: none"> • Occlusal interferences causing: <ul style="list-style-type: none"> ○ Attrition^[11] ○ Pathological mobility/Traumatic occlusion^[40] ○ Tongue irritation/Compromised tongue space^{[11],[36],[61]} ○ Cusp fracture^[52] ○ Speech disturbances^[37] ○ TMJ Disorders due to premature contact, deflecting contact^{[11],[39]} 	<ul style="list-style-type: none"> • Occlusal grinding: <ul style="list-style-type: none"> ○ Sequential Grinding alone^[11] ○ Sequential Grinding + Fluoride-application/desensitizing-agent^{[11],[11],[33],[37],[39],[42]} ○ Grinding followed by restoration with: <ul style="list-style-type: none"> ▪ GIC ▪ Composite^[37]
<ul style="list-style-type: none"> • Risk-of or Actual Pulp-exposure, Pulpal Irritation (Pulpitis) or Necrotic Pulp <ul style="list-style-type: none"> ○ Secondary to Attrition/Traumatic occlusion^{[3],[52]} ○ Secondary to Dental Caries 	<ul style="list-style-type: none"> • Pulp Capping <ul style="list-style-type: none"> ○ Hard-setting CaOH-based medicaments^[37] ○ MTA^[45] • Partial Pulpotomy (When immature) <ul style="list-style-type: none"> ○ CaOH Pulpotomy^{[23],[47],[53]} ○ MTA^[46] • RCT (When mature)^{[3],[52]} • Apexification (When immature) <ul style="list-style-type: none"> ○ CaOH/MTA^[52]
<ul style="list-style-type: none"> • Malocclusion & Displacement of opposing/affected teeth 	<ul style="list-style-type: none"> • Orthodontic correction with: <ul style="list-style-type: none"> ○ Removable appliance^{[18],[42],[43]}

<ul style="list-style-type: none"> ○ Tooth rotation^[62], displacement^{[37],[58]}, infra-occlusion^{[1],[18]} ○ Open Bite^[11] • Hinder anterior retraction during fixed appliance therapy^[41] 	<ul style="list-style-type: none"> ○ Fixed appliance therapy^{[33],[43]} • Periodic follow-up to prevent cross-bite^[58]
<ul style="list-style-type: none"> • Damage to periodontium/attachment^{[1],[44]} 	<ul style="list-style-type: none"> • Perio/Resto treatment^[33]
<ul style="list-style-type: none"> • Hypersensitivity 	<ul style="list-style-type: none"> • Restoration, Desensitizing agents, Fluoride-agent application
<ul style="list-style-type: none"> • Difficulty in Feeding^{[36],[38]} 	<ul style="list-style-type: none"> • Same as above

necessitates the prolonged involvement of the patient. More extensive research into this anomaly needs to be conducted, because despite knowing the influence of genetic and environmental factors, the particular cause for an individual case remains unidentified. Also, despite numerous reports of such cases, there has been no controlled clinical trial conducted to evaluate the effectiveness of a particular type of treatment for this anomaly.

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