Cracked tooth syndrome. Part 1: aetiology and diagnosis

S. Banerji,[1] S. B. Mehta[2] and B. J. Millar[3]

VERIFIABLE CPD PAPER

PRACTICE

IN BRIEF

- Updates and clarifies the definition of the term 'cracked tooth syndrome', including an overview of the typically associated signs and symptoms of this syndrome complex.
- Provides an account of the epidemiology, aetiology and diagnosis of the condition, including a description of available special clinical tests to form a positive diagnosis.
- Details the factors which may influence the prognosis of affected teeth.

Symptomatic, incompletely fractured posterior teeth can be a great source of anxiety for both the dental patient and dental operator. For the latter, challenges associated with deriving an accurate diagnosis together with the efficient and time effective management of cases of cracked tooth syndrome are largely accountable for the aforementioned problem. The aim of this series of two articles is to provide the reader with an in-depth insight into this condition, through the undertaking of a comprehensive literature review of contemporarily available data. The first article will provide details relating to the background of cracked tooth syndrome including the epidemiology, patho-physiology, aetiology and diagnosis of the syndrome, together with a consideration of factors which may influence the prognostic outcome of teeth affected by incomplete, symptomatic fractures. The second article will focus on the immediate and intermediate management of cracked teeth, and also provide a detailed account of the application of both direct and indirect restorations and restorative techniques used respectively in the management of teeth affected by this complex syndrome.

INTRODUCTION

The term 'cuspal fracture odontalgia' was first used by Gibbs in 1954,[1] to describe a condition which is better now known as 'cracked tooth syndrome' or 'cracked cusp syndrome'. The latter concept was coined by Cameron in 1964,[2] who proceeded to define the condition as 'an incomplete fracture of a vital posterior tooth that involves the dentine and occasionally extends to the pulp'. In more recent times the definition has been amended to include, 'a fracture plane of unknown depth and direction passing through tooth structure that, if not already involving, may progress to communicate with the pulp and or periodontal ligament'.[3]

The term 'incomplete fracture of posterior teeth' is often used interchangeably with that of cracked tooth syndrome,[4] while the terms 'green-stick fracture' or 'split tooth syndrome' have also been used synonymously.[5]

Patients suffering from cracked tooth syndrome (CTS) classically present with a history of sharp pain when biting, or when consuming cold food/beverages.[6] It has been suggested that the symptom of pain on biting increases as the applied occlusal force is raised.[7] A detailed assessment of the symptoms may reveal a history of discomfort that may have been present for several months previously. Other symptoms may include pain on release of pressure when fibrous foods are eaten, 'rebound pain'.[8] Pain may also be elicited by the consumption of sugar containing substances[5] and also by the act of tooth grinding or during the undertaking of excursive mandibular movements.[9] While some patients are able to specify the precise tooth from which the symptoms may be arising, the latter is not a consistent feature. The absence of heat induced sensitivity may also be a feature.

Where the fracture line may eventually propagate into the pulp chamber ('complete fracture'), symptoms of irreversible pulpitis or apical periodontitis may ensue, while fractures which progress further towards the root may be associated with areas of localised periodontal breakdown or at worst culminate in vertical tooth fracture[5]

Fig. 1 Shows an example of a tooth with a vertical root fracture, where the patient initially presented with symptoms of cracked tooth syndrome. Any delays in instituting therapy may result in such an outcome, which may happen where there is doubt over the diagnosis of the condition

as shown by Figure 1. Table 1 provides a summary of the commonly associated signs and symptoms associated with CTS.

The physiological basis of pain on chewing has been hypothesised by Brannstrom *et al.*[10] to be accounted for by the sudden movement of fluid present in dentinal tubules which occurs when the fractured portions of the tooth move independently of one another. It is thought that the latter results in the activation of myelinated A-type fibres within the dental pulp, thereby accounting for the acute nature of the pain. It has also been suggested that the perception of hypersensitivity to cold may occur as a result of the seepage of noxious irritants through the crack, which results

[1]Senior Clinical Teacher; [2]General Dental Practitioner and Clinical Teacher, [3]*Professor, Consultant in Restorative Dentistry, Department of Primary Dental Care, King's College London Dental Institute, Bessemer Road, London, SE5 9RW
*Correspondence to: Professor B. J. Millar
Email: brian.millar@kcl.ac.uk

Refereed Paper
Accepted 25 March 2010
DOI: 10.1038/sj.bdj.2010.449
©British Dental Journal 2010; 208: 459–463

in the subsequent release of neuropeptides which cause a concomitant lowering in the pain threshold of unmyleinated C-type fibres within the dental pulp.[11]

An alternative hypothesis has been proposed, whereby it has been postulated that the symptoms are caused by the alternating stretching and compressing of the odontoblast processes located within the crack.[12]

The aim of this article is to provide an overview of the condition of 'cracked tooth syndrome', with regards to its epidemiology, aetiology and diagnosis.

Figure 2 illustrates a tooth which has an incomplete fracture, which was revealed upon the removal of an existing silver amalgam restoration. An incomplete tooth fracture is often difficult to visualise before a restoration is removed, but transillumination can be used from different aspects to show the presence of an interface within the tooth (Fig. 3). Tooth fractures can be highlighted by the use of stains although this may be difficult to remove and colour the final aesthetic restoration.

EPIDEMIOLOGY OF CRACKED TOOTH SYNDROME

Epidemiological studies of the incidence of cracked tooth syndrome are conflicting;[13,14] however, it would be appropriate to state that CTS is a condition which generally affects adult dental patients, typically in the age range of 30 to 60 years. While the results of an early epidemiological survey by Cameron in 1976[6] seemed to suggest that the condition was much more prevalent among female dental patients, it has since been shown by more recent studies that both sexes seem to be equally affected.[15]

Geursten *et al.*[16] have reported that tooth fractures are a potential major cause of tooth loss in the industrialised world. The availability of incidence data on the condition of cracked tooth syndrome is largely lacking. A study by Krell *et al.*[17] has reported an incidence rate of 9.7% among 8,175 patients referred to a private endodontic practice over a period of six years. It would be logical to assume that as more patients are retaining their teeth into older age incomplete fractures of posterior teeth are more likely to be observed to be occurring at an even higher frequency in the future.

Table 1 The commonly presenting signs and symptoms seen in cases of cracked tooth syndrome (CTS)

Sudden, sharp pain on biting/chewing and in some cases on release: 'rebound pain'
Sensitivity to cold thermal stimuli; in some cases hyper-reactivity to hot/sugary stimuli may also occur
Symptoms may be present for periods ranging from weeks to months
Inconsistent ability to localise the affected tooth
Pain may be elicited by lateral cusp pressure, as evoked by 'bite tests' and tooth grinding
Fracture lines may be seen clinically (sometimes upon removal of the restoration), aided by magnification, dyes or transillumination
Positive response to vitality tests; exaggerated response to cold thermal stimuli
Radiographs; usually inconclusive

While incomplete posterior tooth fractures are most likely to be seen to be occurring in teeth which have carious lesions or contain dental restorations, a study by Hiatt[13] reported that 35% of the cases presenting with CTS among their sample were among teeth which were sound and caries free.

Mandibular molar teeth appear to be the most commonly involved teeth by this condition,[13] followed by maxillary premolars and maxillary molar teeth - while mandibular premolar teeth seem to be least affected. It has been hypothesised that since lower first molar teeth are usually the first permanent teeth to erupt into the dental arch, they are most likely to be affected by the condition of dental caries, followed by the need of subsequent restorative intervention.[14] These teeth are therefore more likely to be rendered with large, deep restorations, making them more vulnerable to the process of subsequent fracture. It has also been proposed that the 'wedging effect' inflicted upon lower first molar teeth from the prominent mesio-palatal cusp of maxillary first molar teeth may also be contributory.[14]

AETIOLOGY OF CRACKED TOOTH SYNDROME

The aetiology of incomplete fractures of posterior teeth is multi-factorial. In an article by Guersten *et al.*[14] it is stated that 'excessive forces applied to a healthy tooth or physiologic forces applied to a weakened tooth can cause an incomplete fracture of enamel or dentine'.

Lynch *et al.*[18] have subdivided the causes of cracks into four major causative categories, hence: 'restorative procedures', 'occlusal factors', 'developmental conditions' and 'miscellaneous factors'.

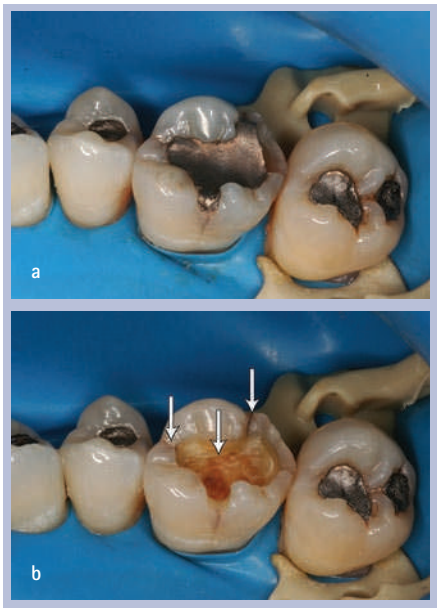

Fig. 2 Shows an example of a tooth which has an incomplete fracture (a) which was revealed upon removal of an existing silver amalgam restoration; (b) the arrows illustrates the path of the fracture line running around the mesiopalatal cusp

Fig. 3 Transillumination of cracked cusp showing mesial midline and lingual fractures. The transmission of the light beam has been 'stopped' mesio-lingually by the presence of the fracture

'Restorative procedures' such as the placement of 'friction lock' or 'self threading dentine pins',[19] the non-incremental application of composite resin, excessive

hydraulic pressure when luting inlays, onlays, crowns or bridges[5] (in particular where the restorations may be 'tight-fitting'), can all induce stresses onto the residual tooth structure culminating in a possible fracture.[20] Likewise, the placement of poor quality dental amalgam alloys, the contamination of freshly placed dental amalgam by moisture and excessive condensation pressures when placing amalgam may also induce fractures.[21] It is ironic, however, that dentine pins are often used to restore fractured cusps among teeth which have been lost through cracked tooth syndrome.

Other aspects of 'restorative practice' which may contribute to crack formation include the excessive removal of tooth tissue during cavity preparation which indeed has been shown to significantly lower tooth rigidity.[22] Deep cusp-fossa relationships which arise as a result of the over-contouring of restorations may also contribute to the fracture of the non-functional cusp.[5] The preparation of vital teeth to receive MOD amalgam restorations (with the loss of both marginal ridges) has been shown to significantly reduce relative cuspal rigidity, on average by 63%.[23] It has been proposed that a cavity of width in excess of one quarter of the intercuspal distance should be considered to be at an increased risk of fracture.[24]

Ratcliff *et al.*[25] have estimated that the presence of an intra-coronal restoration can predispose the tooth to a risk of fracture 29 fold times greater than that of a healthy, un-restored tooth! Differences in the co-efficients of thermal expansion between that of the tooth tissue and restorative material may also have the potential to induce fracture.[26]

'Occlusal causitive factors'; Trucshowksy[5] has stated that the most common cause of cracked tooth syndrome is that of 'a masticatory accident' - biting suddenly on a hard object such as bone with excessive force. Other commonly attributing food items/objects include betel nut chewing, inadvertent biting of lead shot, cherry stones and 'granary' bread.[27]

Trauma from the occlusion may also lead to fracture. Helkimo *et al.*[28] determined the maximum biting force between natural molars to range from 10 to 73 kg with an average of 45.7 kg for males and 36.4 kg for females. It has been estimated that the ratio of force on molars, premolars and incisors is 4 : 2 : 1 respectively, with far higher forces being applied the closer the tooth is to its posterior occlusal determinant, (the temporo-mandibular joint).[29] Occlusal interferences on vulnerable cusps can also lead to eventual fractures as may do non-working side occlusal interferences.[30] The loss of anterior guidance may also lead to the generation of harmful eccentric forces.

Parafunctional tooth grinding habits may also lead to the generation of considerable occlusal forces, in particular the habit of nocturnal bruxism, possibly due to cortical inhibitors being suppressed during sleep, thus allowing greater forces to be applied.[31]

So called 'developmental factors' include the possibility of areas of localised structural weakness within a tooth, arising as a result of the incomplete fusion of areas of calcification.[13]

Morphological factors associated with the increased risk of cracked tooth syndrome include deep occlusal grooves, pronounced vertical radicular grooves or bifurcations, extensive pulp spaces, steep cusp angles, prominent mesio-palatal cusps of upper maxillary first molars as well as the presence of lingually inclined mandibular molar teeth, which are thought to be the be most likely to suffer the complete loss by fracture of both lingual cusps.[32]

Under the category of 'miscellaneous factors' are included factors such as the effect of lingual barbells; the cracking/crazing of tooth tissue which arises from the use of high speed rotary instruments; erosive tooth wear and the factor of thermal cycling, which may induce enamel cracks.

An ageing dentition may also be more predisposed to cracking as dental hard tissues become more brittle and less elastic with age, whereby forces applied may exceed the elastic limits of dentine.[7]

THE DIAGNOSIS OF CRACKED TOOTH SYNDROME

The diagnosis of cracked tooth syndrome is often problematic and has been known to challenge even the most experienced of dental operators, accountable largely by the fact that the associated symptoms tend to be very variable and at times bizarre.[33] Indeed it has been reported[34] that 20% of patients referred to specialist endodontists with diagnostic uncertainties are eventually diagnosed with incomplete tooth fractures. The importance of an early diagnosis has being linked with successful restorative management and prognosis.[35]

A careful history and assessment of the symptoms, in particular that of cold sensitivity and sharp pain on biting hard or tough food which ceases on the release of pressure, is an important indicator. Symptoms may vary according to the depth and orientation of the crack.[4]

According to Homewood,[36] fractures tend to occur in a direction parallel to the forces on the cuspal incline; thus with larger restorations, cracks tend to be more superficial and thereby produce fewer symptoms, while with smaller restorations cracks tend to be deeper and closer to the pulp. It has also been suggested that most cracks tend to run vertically (as opposed to horizontally).[33] Vertical cracks usually run in a mesio-distal direction along the occlusal surface and may involve one or both of the marginal ridges respectively.[33]

Diagnosis is often complicated by the fact that several other dental conditions may readily be mis-diagnosed as cracked tooth syndrome. Such conditions include: acute periodontal disease, reversible pulpitis, dentinal hypersensitivity, galvanic pain associated with the recent placement of silver amalgam restorations, post-operative sensitivity associated with micro-leakage from recently placed composite resin restorations, fractured restorations, 'high spots' or areas of hyper-occlusion from dental restorations, occlusal trauma from the process of parafuntional tooth grinding, orofacial pain arising from conditions such as trigeminal neuralgia and psychiatric disorders such as atypical facial pain.[20]

While occasionally cracks may be detected by visual inspection, they are not always readily apparent. The use of magnifying loupes and trans-illumination with the aid of a fibre-optic device (Fig. 3) may be helpful.[18] The use of a sharp straight probe may also help detect 'catches' in the cracks, while the application of the latter dental instrument at the margins of heavily restored teeth which are suspected to be involved by an incomplete fracture may elicit symptoms of sharp pain should a fracture be possibly present. The removal

of existing restorations may also help to reveal fracture lines.

An un-authenticated technique, which has often been used by the authors of this article particularly where there may be doubt over the precise diagnosis, is one which involves the placement of composite resin over the affected tooth without etching and bonding; material is added at a minimal thickness of 0.5 mm and wrapped over across the external line angles of the tooth onto the axial walls. The set material acts as a splint and the patient can bite down on this with an intervening bite test; although high, it may result in greatly reduced symptoms as the fracture no longer opens on clenching, perhaps confirming the diagnosis of an incomplete fracture. It must be emphasised, however, that there is no evidence in the available literature (to the knowledge of the authors) to scientifically validate this approach.

The use of stains to highlight fracture lines such as gentian violet or methylene blue[33] have been described by several authors. However, it is imperative to note that the technique of using stains to delineate cracks may take several days to be effective and may require the placement of a provisional restoration in the cavity, which may further undermine the structural integrity of the tooth and thereby aid in the process of crack propagation. Another complication with the use of delineation dyes is the subsequent difficulty associated with the placement of a definitive aesthetic restoration. Periodontal probing may also be helpful, as localised isolated periodontal probing defects may be seen where fracture lines may have extended subgingivally.

The use of so called 'bite tests'[18] to mimic the symptoms associated with incomplete fractures of posterior teeth may also prove helpful. However, it is important to gain prior consent from the patient as the use of such a test may cause cuspal fragmentation! Objects that have been traditionally used for this purpose include: orange wood sticks, cotton wool rolls, rubber abrasive wheels such as a Burlew wheel or the head of a number 10 round bur in a handle of cellophane tape.

The technique for the use of wood sticks has been described.[37] It is advocated that the stick is rested on the suspected tooth and the patient asked to bite; by the subsequent application of the stick to each individual cusp in turn it may be possible to localise the affected cusp. The use of cotton wool rolls involves placing the roll on the suspected tooth and requesting the patient to bite down and then suddenly releasing the pressure. Pain perceived upon release of pressure has been suggested by Kruger[38] to confirm the diagnosis of cracked tooth syndrome. The use of rubber plungers of anaesthetic carpules suspended from a length of floss, to be used in a similar fashion to cotton wools rolls, to aid in the diagnosis of cracked tooth syndrome, has also been described.[39]

Commercially available diagnostic tools to undertake 'bite tests' include products such as 'Fractfinder (Denbur, Oak Brook, IL, USA) and 'Tooth Slooth II' (Professional Results Inc., Laguna Niguel, California, USA). Figure 4 shows the Tooth Slooth which comprises two small plastic pyramidal plastic bite blocks attached to a handle at either end (20 by 10 mm). One block has a small concavity at its apex which can accommodate the cusp of a tooth.[40] The slooth is placed either between the cusps of a tooth or onto the cusp tip and the patient is asked to close together. Pain on biting or release on the specific cusp identifies the offending/involved cusp. Ehrmann *et al.*[37] have advocated the use of this method as one with a higher level of sensitivity than that associated with the use of wood sticks; furthermore, it is thought to allow for the more accurate identification of the affected/involved cusp.

Vitality tests for involved teeth are usually positive,[5] although sometimes affected teeth may display signs of hypersensitivity to cold thermal stimuli due to the presence of pulpal inflammation; a feature that may help to confirm a diagnosis of CTS. Teeth affected by the condition are seldom tender to percussion (when percussed apically).

Radiographs tend to be of limited use as fractures tend to propagate in a mesio-distal direction,[18] parallel to that of the plane of the film. However, they may be of value in detecting more rarely occurring fractures which may be running in a bucco-lingual direction and for excluding other dental pathology.[20]

The use of copper rings, stainless steel orthodontic bands and acrylic provisional crowns to confirm the diagnosis of an incomplete fracture of a posterior tooth, have been discussed in more detail in second of the two articles on cracked tooth syndrome.

Fig. 4 Tooth Slooth (Professional Results, USA)

THE PROGNOSIS OF TEETH AFFECTED BY CRACKED TOOTH SYNDROME

The prognosis of teeth affected by CTS is dependent on a multitude of factors. The location and extent of the crack is a key determinant. It has been reported that incomplete cracks generally run in a mesio-distal direction (81.1%); rarely are vertical or orovestibular cracks seen,[41] For cracks that are confined to the dentine layer, that run in a horizontal direction not involving the dental pulp, or for those fractures which are limited to a single marginal ridge which do not extend more than 2-3 mm below the periodontal attachment, the prognosis has been reported by Clark *et al.*[42] to be 'excellent'; while the prognosis for fractures that involve both marginal ridges, communicating with the dental pulp or those fractures that extend vertically through the pulp or involve the sub-pulpal floor has been described as being 'poor'. Affected teeth which present with complete mesio-distal fractures, or one where the fractured segment cannot be removed or be exposed by gingivoplasty or by an alveoplasty procedure, have been described as having a 'hopeless prognosis'.[42]

Other factors which may impact on the prognosis include the anatomy of the tooth and roots, the previous operative/restorative history of the tooth and the functional forces applied to the tooth (during both functional and parafunctional activity).[18]

Early recognition will also help to prevent further cracking, in particular, helping to avoid propagation of the crack into the pulp chamber or sub-gingivally. It has been postulated that the loss of pulp vitality will have a poor effect on the prognosis of the

tooth, as endodontically treated cracked teeth have been shown to display a relatively high failure rate of 14.5% after an evaluation period of two years.[43] Indeed, it has been reported that approximately 20% of teeth with cracked tooth syndrome will require root canal therapy.[44] The skill and experience of the operator are also important factors.[18]

Finally, the technique used to manage the condition will also have an important impact on the ultimate prognosis of the affected tooth. The second paper in this series will discuss the latter issue in considerable detail.

1. Gibbs J W. Cuspal fracture odontalgia. *Dent Dig* 1954; **60:** 158-160.
2. Cameron C E. Cracked tooth syndrome. *J Am Dent Assoc* 1964; **68:** 405-411.
3. Ellis S G. Incomplete tooth fracture - proposal for a new definition. *Br Dent J* 2001; **190:** 424-428.
4. Geurtsen W, Schwarze T, Gunay H. Diagnosis, therapy and prevention of the cracked tooth syndrome. *Quintessence Int* 2003; **34:** 409-417.
5. Trushkowsky R. Restoration of a cracked tooth with a bonded amalgam. *Quintessence Int* 1991; **22:** 397-400.
6. Cameron C E. The cracked tooth syndrome: additional findings. *J Am Dent Assoc* 1976; **93:** 971-975.
7. Signore A, Benedicenti S, Covani U. Ravera G. A 4 to 6 year retrospective clinical study of cracked teeth restored with bonded indirect resin composite onlays. *Int J Prosthodont* 2007; **20:** 609–616.
8. Stanley H R. The cracked tooth syndrome. *J Am Acad Gold Foil Oper* 1968; **11:** 36-47.
9. Griffin J. Efficient, conservative treatment of symptomatic cracked teeth. *Compendium* 2006; **27:** 93-102.
10. Brannstrom M, Astrom A. The hydrodynamics of dentine: its possible relation to dentinal pain. *Int Dent J* 1972; **22:** 219-227.
11. Davis R, Overton J. Efficacy of bonded and non-bonded amalgams in the treatment of teeth with incomplete fractures. *J Am Dent Assoc* 2000; **131:** 496-478.
12. Dewberry J A. Vertical fractures of posterior teeth. Lieve F S(ed). *Endodontic therapy*, 5th ed. pp 71-81. St Louis: Mosby, 1996.
13. Hiatt W H. Incomplete crown-root fractures in pulpal periodontal disease. *J Periodontol* 1973; **44:** 369-379.
14. Geurtsen W. The cracked tooth syndrome: clinical features and case reports. *Int J Periodontics Restorative Dent* 1992; **12:** 395-405.
15. Roh B D, Lee Y E. Analysis of 154 causes of teeth with cracks. *Dent Traumatol* 2006; **22:** 118–123.
16. Geurtsen W, Garcia-Godov F. Bonded restorations for the prevention and treatment of the cracked tooth syndrome. *Am J Dent* 1999; **11:** 266–270.
17. Krell K, Rivera E. A six year evaluation of cracked teeth diagnosed with reversible pulptitis; treatment and prognosis. *J Endod* 2007; **33:** 1405-1407.
18. Lynch C, McConnel R. The cracked tooth syndrome. *J Can Dent Assoc* 2002; **68:** 470-475.
19. Fuss Z, Lustig J, Katz A, Tamse A. An evaluation of endodontically treated vertical root fractured teeth: impact of operative procedures. *J Endod* 2000; **27:** 46-48.
20. Turp C, Gobetti J. The cracked tooth syndrome: an elusive diagnosis. *J Am Dent Assoc* 1996; **127:** 1502-1507.
21. Rosen H. Cracked tooth syndrome. *J Prosthet Dent* 1982; **47:** 36-43.
22. Goel V, Khera S, Gurusami S, Chen R. Effect of cavity depth on stresses in a restored tooth. *J Prosthet Dent* 1992; **67:** 174-183.
23. Plotino G, Buono L, Grande N, Lamorgese V, Soma F. Fracture resistance of endodontically treated molars restored with extensive composite resin restorations. *J Prosthet Dent* 2008; **99:** 225-232.
24. Mondelli J, SteagallL, Ishikiriama A. Fracture strength of human teeth with cavity preparations. *J Prosthet Dent* 1980; **433:** 419-422.
25. Ratcliff S, Becker I, Quinn L. Type and incidence of cracks in posterior teeth. *J Prosthet Dent* 2001; **86:** 168-172.
26. Bearn D, Saunders E, Saunders W. The bonded amalgam restoration – a review of the literature and report of its use in the treatment of four cases of cracked tooth syndrome. *Quintessence Int* 1994; **25:** 321-326.
27. Talim S T, Gohil K S. Management of coronal fractures of permanent posterior teeth. *J Prosthet Dent* 1974; **31:** 172-178.
28. Helkimo E. Bite force and functional state of the masticatory system in young men. *Swed Dent J* 1978; **2:** 167-175.
29. Arnold M. Bruxism and the occlusion. *Dent Clin North Am* 1981; **25:** 395-407.
30. Sweptson J H, Miller A W. The incompletely fractured tooth. *J Prosthet Dent* 1986; **55:** 413-415.
31. Attansio R. Nocturnal bruxism and its clinical management. *Dent Clin North Am* 1991; **35:** 245-252.
32. Bader J D, Martin J A, Shugars D A. Incidence rates for complete cusp fracture. *Community Dent Oral Epidemiol* 2001; **29:** 346-353.
33. Liu H H, Sidhu S K. Cracked teeth – treatment rational and case management: case reports. *Quintessence Int* 1995; **26:** 485-492.
34. Brady B V, Maxwell E H. Potential for tooth fracture in restorative dentistry. *J Prosthet Dent* 1981; **45:** 411-414.
35. Agar J R, Weller R N. Occlusal adjustments for initial treatment and prevention of cracked tooth syndrome. *J Prosthet Dent* 1988; **60:** 145–147.
36. Homewood C I. Cracked tooth syndrome – incidence, clinical findings and treatment. *Aust Dent J* 1998; **43:** 217-222.
37. Ehrmann E H, Tyas M J. Cracked tooth syndrome: diagnosis, treatment and correlation between symptoms and post-extraction findings. *Aust Dent J* 1990; **35:** 105-112.
38. Kruger B F. Cracked tooth syndrome. Letter to the editor. *Aust Dent J* 1984; **29:** 55.
39. Liebenburg W H. Esthetics in the cracked tooth syndrome: steps to success using resin bonded ceramic restorations. *J Esthet Dent* 1995; **7:** 155-166.
40. Fox K, Youngson C C. Diagnosis and treatment of the cracked tooth. *Prim Dent Care* 1997; **4:** 109-113.
41. Mittal N, Sharma V, Minocha A. Management of cracked teeth - a case report. *Endodontology* **2007;** 39-44.
42. Clark L L, Caughman W F. Restorative treatment for the cracked tooth. *Oper Dent* 1984; **9:** 136-142.
43. Tan L, Chen N N, Poon C Y, Wong H B. Survival of root filled cracked teeth in a tertiary institution. *Int Endod J* 2006; **39:** 886-889.
44. Gutman J L, Rauskin H. Endodontic and restorative management of incompletely fractured molar teeth. *Int Endod J* 1994; **27:** 364–365.