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Research Paper

A Review on Endodontic Management in Traumatized Permanent Teeth

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ABSTRACT: *The majority of accidents affect children and adolescents, often when root development of the injured teeth is not completed. Teeth with immature root development, necrotic pulps, and apical periodontitis present multiple challenges for successful treatment. The infected root canal space cannot be cleaned and disinfected with the standard root canal protocol using an aggressive procedure with endodontic files.After the disinfecting phase of treatment has been completed, filling the root canal is difficult because the open apex provides no barrier for containing the root filling material without impinging on periodontal tissues.Even after successfully completing the endodontic procedure, the roots of these teeth are still thin and have a significant risk of subsequent fracture.These problems can be managed using a disinfection protocol that minimizes root canal instrumentation, by stimulating the formation of a hard tissue barrier or providing an artificial apical barrier to allow for optimal filling of the canal, and by reinforcing the weakened root against fracture both during and after an apical barrier has been provided.*

Keywords:*Traumatized teeth, Endodontic management, Permanent teeth, Teeth fracture.*

I. INTRODUCTION

Traumatic injuries of teeth are the main cause of emergency treatment in dental practice. It occurs most commonly in young patients, and varies in severity from fracture of teeth or avulsion. Management of traumatic injuries is done by restorative as well as endodontic procedures. Traumatic dental injury occurs more frequently in permanent teeth, mostly the maxillary central incisors. The physiologic and especially severe overjet predisposes maxillary incisors to fracture, especially if there is lack of lip coverage. Traumatic injuries of face can occur due to violence, road traffic accidents or sports activities. The word 'trauma' implies a reasonable severe, non-physiological lesion to any part of the body. Any thermal, chemical or mechanical lesion that affects the dentition should be analysed as a dental trauma and its effect, as a traumatic dental injury.[1]Trauma to the teeth may result either in injury of the pulp, with or without damage to the crown or root, or partial or complete avulsion. When the crown or root is fractured, the pulp may recover and survive the injury, it may succumb immediately, or it may undergo progressive degeneration, resulting in the death of the pulp.[2] Traumatic injuries if among the age group of 8-10yrs involving the dentition[3], the chances of retention of teeth by vital pulp therapy will be more successful due to good blood supply. The disadvantage with children's pulp is that the root development may be affected and the teeth with damaged pulps, leaving the roots thin and weak.It has been claimed that the high percentage of dental trauma and its sequelae will probably exceed dental caries and periodontal disease among the young population.[3] Proper dental and systemic history is essential for the success of the treatment. Hence through evaluation of the patient's medical history and immunization status and a general appraisal of the patient's physical condition and neurologic status are necessary.[4] Dental treatment should be performed only after potential life threatening injuries such as neurological damage, bleeding or aspiration of foreign bodies/teeth, are treated or excluded. Management of traumatic dental injury could be divided into primary, secondary and tertiary phases. Primary care provides the emergency treatment required immediately after trauma.[5,6] This can be divided into acute, sub-acute and delayed priorities. Acute priority deals with problems such as haemostasis, replanting avulsed (dislodged) teeth and fixing alveolar fractures. Sub-acute

priority could be executed within hours after injury, like treating various types of teeth luxations (displacement), complicated crown fractures, root fractures and removal of foreign bodies from the wound.[7] Tetanus toxoid or antibiotic prophylaxis is also provided at this stage.[8]The secondary phase of treatment usually starts two weeks after the primary phase. This includes monitoring of pulpal status and healing of soft and hard tissues, root canal treatment, and endo-perio surgery.[9]The tertiary phase of treatment commences a few months to years after injury, which includes the final restoration of missing teeth, monitoring of reimplanted teeth and other dental specialty treatment.[10]The success of treatment of traumatic injuries depends on the use of ideal restorative materials. Protection of pulp is more important as researchers develop new methods to promote pulpal function in young, traumatized teeth.[11] Biomimetics, or tissue engineering, may allow replacement of necrotic pulp with tissue capable of assuming the interrupted functions of a traumatized pulp.[7]Prevention has been a goal in dentistry for a long time and has resulted in remarkable success. Use of preventive gadgets such as well-fitting mouthguards, properly designed headgear, face-protection, and seat belts can be very effective in preventing traumatic injuries.

Crown fractures with dentin exposure

Healing and pathology: Crown fractures involving dentin result in exposure of dentinal tubules to the oral environment. Exposure of dentin, as such, causes only insignificant changes in the pulp which may resolve and the exposed dentinal tubules be sealed off by formation of secondary dentin.[8]However, if deeply exposed dentin is left unprotected, bacteria and their components in dental plaque on the fracture surface may penetrate the tubules and cause inflammation in the underlying pulp.[14] Judging from experimental studies, the subsequent reparative or degenerative changes depend on the time that has elapsed since injury and the distance between the fracture surface and pulp. In young permanent teeth, wide dentinal tubules may be an additional factor. If irritation is eliminated by treatment of the exposed dentin, localized inflammation in the pulp may resolve, with damaged tissue being replaced reparative dentin[15].When deeply exposed dentin is left unprotected over a longer period of time, the pulp may in some cases become necrotic and the crown discoloured. However, a more frequent cause of pulp necrosis in crown-fractured teeth is probably impaired blood circulation in the pulp due to a concomitant luxation injury.

Treatment: A crown fracture is usually an isolated injury and the crown can be restored at the first visit, normally with a resin composite material and dental bonding techniques. Modern dentin bonding systems have been reported to increase the bonding strength of composite restorations, reduce microleakage, decrease permeability of dentin and possess an antibacterial effect. The occasional occurrence of pulp inflammation has been explained by various factors, but poor bonding, which permits microleakage and penetration of bacteria into the pulp seems to be the most common. It ahs, therefore, been recommended that in case of deep carious lesions, the pulp should be protected, e.g. by the lining of the deepest part of the cavity before a dentin bonding agent is placed over the rest of the dentin. The value of lining with calcium hydroxide preparations beneath permanent restorations has been questioned. However, in cases where the color of the pulp can be seen through a very thin dentin layer or when a deep fracture is left untreated over a couple of days, it may be advisable that the dentin be initially treated with calcium hydroxide and the crown temporarily restored for 2-3 weeks.[14]In such teeth, treatment with calcium hydroxide may remove bacteria from the fracture surface and give an eventually inflamed pulp a chance to recover and seal off the exposed dentinal tubules by deposition of reparative dentin. Moreover calcium hydroxide significantly decreases the permeability of dentin to penetration by bacterial components into the pulp, which may provide additional pulp protection under a subsequent restoration.If, for some reason, crown restoration cannot be performed immediately, e.g. due to incorporation in a splint, the pulpo-dentinal complex should be protected. Before the splint is adapted, the exposed dentin may be covered with hard-setting calcium hydroxide compound or glass ionomer cement.

Prognosis: Restoration of a fractured crown with resin composites and dental bonding techniques can be regarded as an effective tooth repair. However, it should be noted that bacterial leakage has been demonstrated in the gaps between composites and dentinal walls in experimental cervical cavities. But most uncomplicated fractures do not involve the cervical area; and, according to clinical studies, the risk of later pulpal damage seems to be minimal.[8,15]

Crown fractures with pulp exposure:

Healing and pathology: A crown fracture through the pulp chamber causes laceration and exposure of the pulp to the oral environment. Healing does not occur spontaneously and untreated exposures ultimately lead to pulp necrosis, a process in which bacteria are the dominant factor. The fibrin clot that forms over the wound surface resolves after couple of days.[8] The subsequent changes can either be proliferative, such as pulp polyp, or destructive, such as abscess formation or pulp necrosis. In various experiments pulpal changes were characterized by a proliferative response, invariably associated with only superficial inflammation, extending not more than 2 mm from the exposure site.A proliferative response of the pulp is probably favoured by an

exposure which permits salivary rinsing and prevents impaction of contaminated debris, as occurs in caries or experimentally induced cavities[12].Necrosis of an inflamed pulp is a rare occurrence. A common etiological factor seems to be plaque and contaminated debris that are permitted to accumulate over the exposure, which may allow bacteria to settle into injured or necrotized pulp tissue.

Treatment: The aim of the treatment should be the preservation of a vital, non-inflamed pulp, biologically walled off by a continuous hard tissue barrier. In most cases, this can be achieved by pulp capping or pulpotomy.When these treatment alternatives are not possible, the pulp must be extirpated and the root canal filled with an adequate root filling material.The maturity of the tooth is of utmost importance in the choice of treatment. It is generally agreed that the exposed pulp should be maintained in young teeth with incomplete root formation; while it can be removed in mature teeth where constriction of the apical foramen allows adequate obturation of the root canal. However, maturation of a tooth is not complete with constriction of the apical foramen. Removal of the pulp in children and adolescents deprives the tooth of physiologic dentin apposition which, together with mechanical cleansing, leaves thin dentinal walls, which may increase the risk of alter cervical root fractures, a problem that should be considered in treatment planning.A concomitant luxation injury compromises the nutritional supply to the pulp and, in principle, contraindicates conservative treatment. However, in luxated immature teeth the chance of pulp survival is considerable and conservative treatment may allow further root development. Treatment should therefore be determined considering severity of the periodontal injury and maturity of the tooth.The effect of age is controversial. Experimentally, an inferior response to injury and treatment has been observed in pulps of old rats compared to young ones. Yet, successful treatment of pulp exposure due to either trauma or caries in older patientshas repeatedly been reported. For various reasons, degenerative changes in the pulp undoubtedly increase with age. Thus, removal of the pulp could be a more successful procedure, although no age limit can be set for either pulp preservation or removal. However, conservative treatment, i.e. capping or pulpotomy should not be performed if degenerative or inflammatory changes are anticipated, e.g. in teeth with reduced pulpal lumen due to trauma or age, or in periodontally involved teeth in adults.Surgical pulp procedures invariably cause further injury to the remaining pulp and should be kept to a minimum.[14] Various instruments have been recommended for pulpal amputation, suchas spoon excavators, slowly rotating round burs and high-speed abrasive diamonds. Of these, the spoon excavator, successfully used in molars, has proven unsuitable in young incisors. Slowly rotating instruments are known to inflict significant injury to the remaining pulp, limiting the chance of survival.

However, it has been shown that injury to the underlying tissue is minimal when abrasive diamond is used at high speed to remove part of the pulp, provided that the bur and tissues are adequately cooled. If effective cooling is not possible, e.g. when the amputation site is deep in the root canal, a round bur at low speed should be used in order to avoid overheating the pulp.The commonly used wound dressing is calcium hydroxide. This material has been widely used for accidentally exposed pulps since the first histological appeared decades ago. When placed over the vital pulp, pure calcium hydroxide causes a superficial tissue necrosis, approximately 1-1.5 mm in depth. This necrosis consists of several layers, including a layer of firm coagulation necrosis in close contact with vital tissue, which seems not to be appreciably affected. Based on the observations in experiments in sound human teeth, it has been suggested that it is neither calcium hydroxide nor its components, but the low-grade irritation from coagulation necrosis that induces defensive reactions in the pulp, resulting in formation of a demarcating hard-tissue barrier. The underlying tissue seems to react to this irritation by producing collagen that is subsequently mineralized, while the coagulated tissue is calcified, which is later followed by differentiation of dentin. This hypothesis of low grade irritation that is not strong enough to destroy the pulp tissue but is sufficient to elicit defensive reactions was strongly supported by findings of the formation of hard tissue barriers after only 10 mins treatment with calcium hydroxide or capping with cyanoacrylate, a material possessing none of the calcium or hydroxyl ions that are thought to be responsible for induction of hard tissue.Cyanoacrylate is degradable in a biological environment and it is probable that some released substance(s) may exert just enough irritation to elicit defensive reactions in the pulp leading to formation of hard tissue barrier. Similar results have been reported in experiments with light-cured composite, zinc phosphate and silicate cements. On the other hand when a biologically inert material, such as Teflon, has been brought into contact with pulpal tissue no hard tissue formation could be found in relation to this material. Thus, the role of calcium hydroxide appears to be limited to its chemical effect that is able to elicit defensive processes in the pulp by exerting a low-grade irritation, either through the induction of coagulation necrosis or directly on the pulp tissue without a visible necrosis when hard-setting compounds are used[12]. Accordingly, formation of the hard tissue barrier itself can be regarded as a defensive, probably stereotypic, reaction of the pulp to a non-specific low-grade irritant.Calcium hydroxide seems to be a suitable and well-tested pulp dressing which repeatedly gives predictable results in the form of a non-inflamed pulp under a well-formed hard-tissue barrier. However, it should be stressed that calcium hydroxide as such has no beneficial effect on healing of a chronically inflamed pulp. In clinical terms, this means that the compound shouldbe placed against vital pulp tissue with intact vascularity in teeth which respond to sensibility testing. Internal dentin resorption and

dystrophic calcifications, reported to occur after dressing of the pulp with calcium hydroxide, seem to be related to the presence of an extra-pulpal blood clot or to the damage caused by operative procedures.[16]

Pulp capping: Capping of the pulp is indicated when a small exposure can be treated shortly after injury which, according to experimental studies, seems to mean within 24 hours. Pulp capping implies that the pulpal wound healing caused by the injury is covered by calcium hydroxide. It is though that, in small exposures treated soon after injury, the mechanical damage and inflammation in the pulp cannot be deeper than the necrotizing effect of calcium hydroxide.[1,12] Thus, the effect actually is exerted on healthy pulp tissue, while bacteria on the dentin fracture and the wound surfaces are eliminated by the action of calcium hydroxide. Accordingly, the primary contamination of the pulp should not be critical for healing. However, pulp healing may be threatened by later contamination due to microleakage of defective restorations, since all calcium hydroxide compounds gradually lose their antibacterial property. Furthermore, the hard tissue barriers may include structural defects which may increase their permeability and, in the case of 'tunnel' formation, due to vascular inclusions, offer a direct contact with the under lying pulp tissue.[2]

Treatment procedure: The fracture surface and pulpal wound are washed with saline. When bleeding has ceased, the exposed pulp is covered with a soft- or a hard-setting calcium hydroxide compound. If the pulp is covered with a soft calcium hydroxide, the exposed dentin should be protected with glass ionomer cement or a hard-setting calcium hydroxide liner before the crown is restored. If the definitive restoration of the crown must be postponed, a temporary crown restoration should be placed with a material that does not allow microleakage, e.g. zinc oxide eugenol or polycarboxylate cement.[2,13]

Pulpotomy: Pulpotomy involves removal of damaged and inflamed tissue to the level of clinically healthy pulp, followed by a calcium hydroxide dressing. Depending on the size of exposure and time elapsed since injury, different levels of pulpal amputation have been recommended, i.e. partial or deep pulpotomy. It has been shown, however, that neither exposure size nor time interval between injury and treatment are critical for healing when only superficial layers of pulp are removed[14].

Treatment procedure: Pulpotomy should be performed with a diamond bur, of a size corresponding to the exposure, in a high speed contra-angle handpiece. Effective cooling is essential. To avoid injury to the pulp due to insufficient cooling, the tooth and cutting instrument should be flushed continuously with water or saline by means of a syringe or the turbine spray. Furthermore cutting should be performed intermittently for brief periods and without unnecessary pressure.[13]The level of amputation should be about 2 mm below the exposure site. This level is deep enough to remove inflamed tissue and provide an adequate cavity for both the dressing and the sealing material. The pulpal wound is rinsed with saline until bleeding has ceased. The wound surface is covered with calcium hydroxide and adapted with cotton pellets using light pressure, whereby water from the paste is also removed. The surplus calcium hydroxide is then easily removed with an excavator.[1,2] The coronal cavity is closed with tight-sealing material, e.g. zinc oxide-eugenol cement. However as eugenol may interfere with the polymerisation of composites, it should be covered with a liner or glass ionomer cement before restoration with composites. Alternatively, the cavity can be sealed with prader's zinc-sulphate cement. This material does not interfere with polymerisation, but possesses sealing properties equal to those of zinc oxide-eugenol cement.The advantage of partial pulpotomy lies in the minimal injury to the pulp and undisturbed physiologic apposition of dentin, especially in the critical cervical area of the tooth. The limited loss of crown substance offers continued opportunity for sensitivity testing and, in most cases, a post in root canal will not be required for crown restoration. Compared with pulp capping, this procedure implies better wound control and, by sealing off the cavity with material which does not allow microleakage, provides effective protection for the pulp. The frequency of failures is low and, judged from clinical observations, depends on factors, such as misjudged pulp diagnosis, overheating pulp at the time of treatment and poor restoration of crown, which allows microleakage and penetration of the bacteria into the pulp. When necrotic tissue or obviously impaired vascularity is present at the exposure site of immature teeth, the pulp should be amputated to a level at which fresh bleeding tissue is found, i.e. a cervical or deep pulpotomy should be performed. Due to problems with adequate cooling of the diamond at high speed at that level, a round carbide bur at low speed should be used. In mature teeth, a pulpectomy is the treatment of choice.[12]

Pulpectomy: Pulpectomy, i.e. removal of the entire pulp to the level of 1-2 mm from the apical foramen, is performed in mature teeth when conservative pulp treatment is not indicated. After removal of the pulp, the root canal is cleansed chemo-mechanically and obturated with gutta-percha points and a suitable sealer.[1,2] There are, however, instances when interim dressing with calcium hydroxide is the treatment of choice, i.e. when periapical healing and closure of the apical foramen with hard tissue is desired before obturation, or when adequate treatment is not possible due to the presence of splints. In the latter instance, it is difficult to adapt rubber dam and access to the root canal can be limited. In such cases, calcium hydroxide can be used as an interim dressing and treatment continued once the splint has been removed.[12]

Prognosis: The frequency of healing after pulp capping, partial or cervical pulpotomy treatments varies from 72-96%. The reported frequency of success after pulpectomy and filling of the root canal with various materials varies from 80-96%.[9]

Root fractures

Healing and pathology: Pulp necrosis with subsequent periradicular involvement occurs with a relatively low frequency, in about 25% of the root fractured teeth. It is a characteristic of a root fracture that only the coronal fragment is dislocated and that pulpal circulation in the apical fragment is not severely disturbed. Thus, pulp necrosis occurs only in the coronal fragment, while the apical fragment remains vital. However, if the coronal fragment is left untreated, the bacteria from its necrotic pulp may spread and cause inflammation and necrosis of the pulp in the apical fragment as well.[8]

Diagnosis of pulp necrosis: Clinical symptoms, such as changes in crown colour and pulp sensibility are interpreted in the same way as in other traumatically injured teeth. The first radiographic sign of pulp necrosis is often a progressive widening of the space between two fragments, later followed by pathologic changes in the adjacent periradicular bone, seen as a widened and diffusely outlined periodontal space or radiolucency, usually present within 3 months after injury. If tooth is not splinted, these changes can make the coronal fragment loose and tender to percussion. Internal surface or tunneling resorption seem to be part of healing processes and normally do not require endodontic intervention. On the other hand, external inflammatory root resorption indicates necrotic and infected pulp tissue.[17]

Treatment procedure: Endodontic treatment in teeth with a fracture involving the cervical part of the root has been shown to be of a poor value. When coronal fragment in a mature tooth should, for some reason, be removed and the length of the apical fragment is judged to be able to support a prosthetic crown, the root canal can be filled with gutta-percha and the fragment extruded orthodontically into the desired position.[18]Conservative endodontic treatment of teeth with fracture in the middle or apical part of the root can be divided into treatment of the coronal fragment alone or both fragments. If these treatments are unsuccessful, surgical removal of the apical fragment can be indicated.The choice of treatment depends on radiographic findings, such as periradicular changes, width of the pulp lumen and separation between the two fragments, as well as on the clinical finding of pulp vitality in the apical fragment at the time of treatment (i.e. pulp canal obliteration). The location of the fracture in the middle or apical part of the root does not affect the choice of treatment.When the root canal is wide, as in very young teeth or in fractures in the middle of the root, it can be difficult to achieve satisfactory cleansing and adequate obturation with gutta-percha. The coronal fragment can be instead be treated initially with calcium hydroxide and filled with gutta-percha after a hard tissue barrier has formed apically in the coronal fragment and periradicular healing has taken place.Root canal treatment of both the fragment s can be performed when the entire pulp is necrotic.[19] The treatment is complicated, as it is difficult to avoid impacting necrotic tissue and filling debris between the fragments during mechanical cleansing as well as overfilling with gutta-percha. This could explain the poor prognosis of this kind of treatment. However, once the root has healed, leaving no empty space between the fragments, this type of treatment may have a better prognosis, in case of a late secondary pulp necrosis.Root canal treatment of the coronal fragment and surgical removal of the apical fragment is indicated in teeth in which the apical fragment with necrotic pulp is not accessible for treatment or when prognosis is poor due to a wide space between the two fragments, as well as in teeth in which either of the first two treatments has not been successful.In case of concomitant crown fracture, the exposed dentin is covered with hard-setting calcium hydroxide or glass ionomer cement, followed by a layer of composite material. The final restoration of the crown is performed after splint removal, i.e. periodontal healing has taken place. An exposed pulp is treated by pulp capping or partial pulpotomy, provided that vital and fresh bleeding is found at the exposure site. Root canal treatment isinstituted later in the event of pulp necrosis.[20]

Prognosis: In a recent study, types of conservative endodontic treatment were evaluated in 98 teeth with fracture in the middle or apical part of the root. It was found that frequency of healing was minimal (0%) in teeth in which the root canal in both fragments was filled with gutta-percha. The failures were judged to be due to extrusion of filling material and necrotic debris in between the fragments, resulting in persistent radiolucency in adjacent tissues. In teeth treated by filling with gutta-percha of the root canal in only the coronal fragment, healing occurred in 76% of teeth. In teeth in which the coronal fragment was filled with gutta-percha and the apical fragment was removed surgically, healing was seen in 68%.periodontal healing with formation of hard tissue barrier apically in the coronal fragment, treated by calcium hydroxide and subsequent obturation with gutta-percha, was found in 86% of 65 teeth, 4 years after the last treatment.[21]

Luxated avulsed or replanted teeth

Healing and pathology: The risk of pulp necrosis increases with increasing stages of root development and severity of root development. A sudden, complete break in pulpal circulation leads to

infarction and coagulation necrosis of the pulp. In mature teeth and in absence of bacteria, such tissue may persist without clinical symptoms or obvious radiographic change for long period of time. The infracted tissue can also be revascularized, i.e. replaced by ingrowth of mesenchymal tissues. This is followed by deposition of hard tissue along the canal walls and, sometimes, continued root development. The presence of micro-organisms was the predominant reason for absence of complete revascularization. Two particular sources were the presence of blood clots harbouring bacteria in the apical area of the pulpal lumen, most probably contaminated during the extra-alveolar period, the other was mechanically damaged cervical root surfaces through which bacteria from dental plaque could penetrate into the necrotic pulp tissue.[4]Anachoretic contamination of the pulp by blood-borne bacteria appears less likely in healthy individuals, although it cannot be excluded.When treated topically with doxycycline for 5 mins before replantation, the frequency of revascularization was significantly increased and the presence of bacteria in pulp lumen decreased.In most experimentally replanted immature teeth with necrotic and contaminated pulps, vital pulp tissue and sometimes formation of new hard tissues can be seen in the apical portion of the pulpal lumen, despite adjacent inflammation and abscess formation either in root canal or periapically. In some teeth, formation of dentin can be seen, indicating that a part of the original pulp has survived, probably due to the diffusion of nutrients from periapical tissues. The presence of vital tissue in the apical part of the pulpal lumen is important from a therapeutic point of view, as this may ensure a more rapid formation of a hard tissue barrier and sometimes continued root development after treatment of non-vital immature teeth with calcium hydroxide.[22]

Treatment of immature teeth: Wide or funnel-shaped root canals make endodontic treatment in immature teeth difficult. The difficulties lie in removing all necrotic tissue from the dentinal walls and achieving adequate obturation of the root canal.[23]

Timing of endodontic procedure after replantation: The endodontic procedure should ideally prevent development of inflammatory resorption or treat this condition at a stage. At the same time PDL healing should be sufficiently advanced that the use of substances such as calcium hydroxide cannot interfere with the healing process and cause apical ankylosis due to its high pH. According to various studies, the optimal time for pulp extirpation appears to be 7-14 days after replantation. Naturally the tooth needs to be splinted or stabilized during the procedure as PDL healing is not complete at this time. The pulp extirpation as performed as a prophylaxis against root resorption (e.g. in the case of root canal closed teeth or teeth with incomplete root formation, or such a long extraoral storage that pulp revascularization is not likely).[22,24]

External inflammatory root resorption:

Pathology: External root resorption in luxated or replanted teeth has been described as surface, inflammatory or replacement resorption, of which only inflammatory resorption is related to necrotic and infected pulp. When dentinal tubules are exposed by resorption of the damaged tissues on the root surface, bacteria and toxins from the root canal may via dentinal tubules diffuse to the adjacent periodontal tissues causing inflammation and progressive root resorption. This seems to be more frequent in immature teeth, most probably due to the thin dentinal walls and wide tubules. The use of systemic antibiotics to prevent inflammatory resorption in replanted teeth is controversial.Radiographically, external inflammatory root resorption is characterized by a progressive loss of tooth substance associated with a persistent or progressive radiolucency in the adjacent alveolar bone. The critical period for onset of these changes is about 2-8 weeks after injury.[5]

Treatment: Dressing the root canal with calcium hydroxide before filling with inert gutta percha has been shown to give high frequency of healing. Elimination of bacteria from the root canal and dentinal tubules appears to be the main factor. It has also been reported that calcium hydroxide from the root canal may raise the pH at the root surface to such a level that the tissue along the resorption cavity, including the resorbing cells, can be damaged. The hydroxyl ions passing through the dentinal tubules denature or precipitate its protein contents, whereby further release of hydroxyl ions towards the periodontium is prevented or reduced to harmless concentrations.It has been suggested that short-lasting or repeated release of calcium hydroxide from the root canal may contribute to occurrence of ankylosis by damaging periodontal cells. But, the initial periodontal injury, inflicted by injury or desiccation, rather than calcium hydroxide should be blamed for occurrence of ankylosis, subsequent to healing of inflammatory resorption.[25]

Root canal resorption (internal resorption)

Pathology: Root canal resorption is related to amount of vital tissue in the pulp lumen. It is often seen in root fractured, but seldom in only luxated teeth. The processes have been described as transient or progressive surface or tunnelling resorption. Surface resorption is seen radiographically as a limited loss of hard tissue, usually at the fracture site or near the apical foramen in luxated teeth. Tunnelling resorption is characterized by the loss of root substance behind the predentin border which slowly progresses in a coronal direction, followed by obliteration of both resorption lesions and the pulpal lumen. Although the triggering mechanisms have not

been assessed, these changes can possibly be looked upon as a part of healing processes in which resorption of damaged or altered tissue is necessary before repair can take place. Thus, endodontic treatment is not indicated unless other changes occur, such as periradicular inflammatory changes[25].Progressive internal resorption is a late and rare complication that usually occurs in the cervical area of the root canal in luxated teeth. The process seems to be elicited by irritation from bacteria or its components in dentinal tubules, originating from a mechanical damage, dilaceration or cracks in the cervical area of the root or by metaplasia of pulp to bone.Occasionally, progressive dentin resorption may also occur in root-filled teeth. A conceivable reason for this could be presence of bacteria in the root canal and communication with the periodontium via an accessory canal, from which soft tissue may proliferate into the root canal and resorb contaminated dentin.[4]

Treatment: It is complicated by the difficultly in removing tissue from a resorption cavity. Soft tissue remnantsmay impede healing if communication exists with the periodontium. However, soft tissue in the lesion can be dissolved by means of repeated filling with calcium hydroxide at 2- to 3- week intervals. If treatment with calcium hydroxide is maintained for a couple of months, communication with the periodontium may be closed by apposition of hard tissue and thus overfilling with gutta percha can be avoided.[4,25]

II. CONCLUSION

Traumatic injuries in children and adolescents are a common problem and several studies have reported that the prevalence of these injuries has increased during past few decades. The goal of treatment for traumatically injured teeth is to return the teeth to acceptable function and aesthetics. Regardless of whether a patient has an isolated injury or a combination of traumatic injuries, the outcome of dental injuries is influenced by patient age, severity, treatment and timely follow-up. In the recent years progress has been made to improve the understanding of the biological considerations involved in both diagnosis and treatment principles. Improved Diagnostic aids (like LDF, various advanced imaging modalities) have helped in close monitoring, that discloses any loss of vitality, pathologic changes, or disturbed development of the root. Similarly, advancement in the material science (such as MTA, Emdogain, adhesive resins etc.,) have allowed for improved treatment options.The management of trauma to the dentition depends primarily on the tissues affected and the extent of damage inflicted by the injury on these tissues. Injuries involving only the hard tissues are managed to promote pulpal healing to an insult. Injuries involving the supporting structures are managed to promote pulpal and periodontal healing. The extent of the damage and management guides the long-term prognosis, which relies on appropriate assessment of the type and severity of the injury.The incidence of pulpal necrosis depends on the mode of injury and stage of root formation, and ranges from 100% in intrusive luxation of mature roots to only 5% in concussion of immaturely formed roots. In case of an avulsed tooth, readily available transport media and appropriate endodontic treatment can make the difference in whether the tooth is retained or ultimately lost. Follow-up evaluation is essential for a good long term prognosis. Frequent review following any form of dental trauma is mandatory. Increased knowledge on the mechanism of various complications and of methods to prevent the process will greatly enhance the treatment of dental injuries. Finally, best way to prevent is the education of how to avoid injuries as well as how to manage those at the site of injury, and all these should lead to progress towards the goal of reducing trauma as a cause of tooth loss.

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