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Root resorption – a clinical problem following tooth injuries

Root resorption is one of possible complications following tooth injury. This process can lead to the loss of the affected tooth. In order to avoid this situation, appropriate treatment within an adequate timeframe is of the utmost importance.

The etiology of root resorption requires two phases: injury and stimulation. Injury is related to less mineralized tissues covering either the external surface of the root, known as the precementum, or the internal surface of the root canal, the predentin. Denuded mineralized tissue is colonized by multinucleated cells, which initiate the resorption process. However, without further stimulation of the resorption cells, the process will end spontaneously. Repair with cementum-like tissue will occur within 2–3 weeks (1).

Recent studies indicate the significant role of the inflammatory cytokines interleukin-1 (IL-1) and tumor necrosis factor alpha (TNF α) in the course of mechanically induced root resorption (2).

We can recognize a number of factors which will induce this process after tooth injury, with the most frequent being pressure or bacterial infection. Therefore, injury should be classed into three groups:

I. The damage to the hard tissues without displacement of the tooth.

II. The damage to the periodontium: a. moderate: concussion, subluxation, b. medium: extrusive luxation, lateral luxation, c. severe: intrusive luxation, avulsion.

III. The damage to both the hard tissues of the tooth and to the periodontium.

In the first group infection is the main factor, while in the second group it is nonphysiological pressure related to tooth displacement (with the exception of subgroup "a") and the possibility of damage to the neurovascular bundle. The third group includes more complex injury. There resorption may be caused by pressure and bacterial infection.

Pulp necrosis post-trauma has been shown to occur in infected teeth or after damage to the neurovascular bundle. Clinical diagnosis of pulp necrosis was made when there was a lack of sensitivity to stimulation and at least one additional sign, such as grey discoloration of the coronal tooth structure, spontaneous pain, tenderness to percussion which persists for a few weeks after injury or periapical radiolucency. X-ray images should be taken several times during the treatment (3–5).

Sometimes after injury, we observed normal or intensified pulp reaction to stimulation, but after a few days, the same examination resulted in a lack of pulp sensitivity. There is a possibility that in these instances, injury had caused damage to the pulp vascularization, however, the function of the nervous system was still sustained. Atrophy of the nerves had taken place some days after trauma.

In another case, we observed no pulp response just after injury, but within a few days, normal pulp sensitivity returned. We call this situation "a shock" after injury. It is impossible for the nerves to transmit due to pressure from hematoma which subsides several days after trauma (6).

Transient apical breakdown is also a cause for temporary lack of pulp reaction to stimulation. As a result of displacement of the tooth following luxation, the vascular supply at the pulpoperiodontal interface could be either partially or totally severed, leading to degenerative changes in the pulp. Subsequent osteoclastic resorption increases the diameter of the apical foramen and the vascular ingrowth into the root canal. Transient apical breakdown could be seen within the first months after injury in 10% of extruded or laterally luxated incisors with completed root formation. Therefore, it is appropriate to take time to observe the progress or regression of both clinical and radiographic disturbances, especially in the young adult age group (approximately 15 years of age and older). There the risk of aggressive inflammatory root resorption is minimal (3, 7).

Approximately 50% of teeth after injury, most especially luxation, responded incorrectly to pulp sensitivity testing. Positive reaction may occur within two months after injury or even later. This is observed in teeth with incomplete root formation. Regeneration of nerve fibers require more time than revascularization, so basic sensitivity tests are not adequate to recognize the present status of the pulp.

If correct root development is observed on x-ray imagery, we can be sure that the process of revascularization has taken place. However, the most conclusive examination is blood flow testing, which diagnoses the vitality of the pulp by measuring its blood flow using Doppler effect.

Pulp reaction to electrical testing is not always accurate. Lack of pulp response is related to the damage to the nervous supply and it may be a temporary status lasting a few weeks. Extended reaction is observed in the teeth with uncompleted root formation and in teeth with resorption. We obtained false positive results during necrosis teeth examination. The positive response is the reaction of periodontium. These factors indicate the necessity of long-term, detailed investigation in order to avoid unnecessary root canal treatment. Preserving the vitality of the pulp is essential for maintaining the condition of the tooth's root by keeping the right elasticity of dentin and function of prioprioreceptors. In 80% of the permanent teeth with incompletely formed roots after root canal treatment, the root or the crown-root fracture was observed in the follow-up period (3). Loss of the pulpal-periodontal prioreceptors is discussed as the reason for such fractures by diminishing the biting force control.

Stormer et al. investigated the negative role of calcium hydroxid in long-term treatment. Andreasen noticed that calcium hydroxid interferes in the natural balance between the main components of the dentin. Part of the organic matrix acts as a bonding agent for collagen and hydroxyapatite. Calcium ions alkalize acid proteins and proteoglycans containing phosphorus and carboxyl groups and reduces their function as bonding agents, and in the same, attenuates the dentin (8).

Before starting any treatment, careful examination, diagnosis, and prognosis are necessary. The type of injury, time elapsed post-injury, the stage of root development, and the risk of complication should be determined. According to Moorees' classification, teeth in the 2^{nd}, 3^{rd} or 4^{th} stage of development after replantation or intrusion are prone to very rapid inflammatory resorption. According to Professor Andreasen, this resorption proceeds about 0.1 mm/24 h and can be observed in radiological examination 2–3 weeks after injury (9). The rate of replacement resorption is proportional to the patients' rate of bone remodelling – occurring more quickly in adolescents, and more slowly in adults (bone remodeling in adolescents occurs at the rate of 50% per year; in adults it is 2% per year) (10) (Fig.1).

Due to dentin mineralization grade, the walls of root canals are thicker, and dentin tubules narrower in mature teeth, resorption progress is significantly lower.

Fig. 1. The case of inflammatory resorption of the left central upper incisor in a patient, two weeks after replantation (image from our own dental practice)

Periodic radiological examinations are essential in the diagnostic approach to traumatized teeth. The first x-ray examination of a traumatized tooth should be evaluated on the day of injury, the next after three weeks, then three months, six months, and once a year for a period of five years post-injury. For diagnostic usefulness, it is important to take subsequent radiographs in the same projection. During the assessment of the radiographs, it is important to remember that resorption of the labial or palatal root site cannot be visualized (9).

In radiological and histological investigations conducted on the human premolars from autopsy, the first symptoms of resorption visualized by radiological examinations were detected with diameter 0.6 mm of depth and 1.2 mm of extension (11) (Fig. 2).

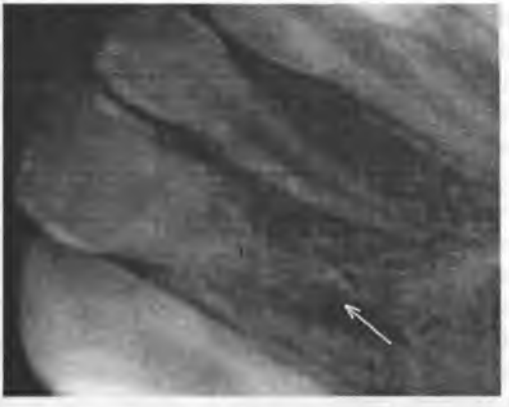

Fig. 2. The case of replacement resorption in mature teeth, a few years after injury (image from our own dental practice)

Traumatized teeth need long-term follow-up examination, and the cooperation of the patient is highly important for treatment success.

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SUMMARY

Root resorption is a serious clinical problem following tooth injuries. The appropriate timing of therapeutic management will aid dentists in avoiding numerous complications. The authors followed the proper procedure for treatment based on the published literature and on their own experience. Most of the available literature recommends a thorough examination and observation of the pulp's condition. Misdiagnosis of this may result in unnecessary endodontic treatment. The authors discerned those categories of tooth injury with the highest risk of root resorption. The age of the patient and the stage of development of the injured tooth determined the intensity of the process. The aim of this study was to evaluate the problem of proper diagnosis of the pulp condition and the stage of root resorption.

Resorpcja korzenia jako powikłanie pourazowych uszkodzeń zębów

Resorpcja korzenia jest poważnym klinicznym problemem występującym po urazach zębów. Podjęcie leczenia stomatologicznego we właściwym czasie pomaga uniknąć wielu niepowodzeń terapeutycznych. Autorzy na podstawie dostępnego piśmiennictwa i własnych doświadczeń prezentują właściwy algorytm postępowania z zębami po urazach. Główną uwagę poświęcono wnikliwemu badaniu i obserwacji stanu miazgi. Pominięcie tego faktu może prowadzić do przedwczesnego leczenia endodontycznego. Wyodrębniono grupy urazów, w których ryzyko resorpcji jest największe. Podkreślono wpływ wieku pacjenta i stopnia rozwoju zęba na intensywność procesu. Szczególną uwagę poświecono trudnościom diagnostycznym pojawiającym się przy ocenie stanu żywotności miazgi oraz stopnia resorpcji korzenia.