Pattern of bone resorption in vertically fractured, endodontically treated teeth

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Purpose. To evaluate the clinical pattern of alveolar bone resorption associated with vertically fractured, endodontically treated teeth in correlation to clinical symptoms.

Material and methods. The pattern of bone resorption was evaluated in 66 maxillary premolars, 13 mandibular premolars, and 31 mesial roots of mandibular molars extracted during an 18-month period because of vertical root fractures. Type and duration of symptoms were recorded and correlated to the pattern of bone resorption.

Results. A V-shaped pattern osseous defect (dehiscence) was typical (91%) to the buccal plate rather than a U-shaped shallow, rounded, slow grade resorption in the palatal or lingual plate. Fenestration of the buccal plate was observed in 10 patients (9%). A positive correlation between type of symptoms and amount of buccal bone resorption was found (P < .0001). The resorptive defect was always facing the fracture line.

Conclusions. A typical pattern of bone resorption in vertical root fracture cases as shown in this study can be helpful to the clinician in diagnosing vertical root fracture when an exploratory full flap surgical procedure is performed.

Material and methods

From June 1996 to January 1998, a total of 110 endodontically treated maxillary (n = 66) and mandibular (n = 13) premolars and mesial roots (n = 31) of mandibular molars with a clinical diagnosis of VRF were referred for extraction (Table I). Teeth with VRF were not included in the 110 teeth if patient records did not supply accurate information regarding tooth history and previous symptoms or if periapical surgery had previously been performed as a result of misdiagnosis and the bone loss pattern could not be attributed to only the VRF.

Signs and symptoms before extraction were evaluated by the oral surgeon and symptoms recorded for each case as follows:

• Chronic pain that is mild and intermittent when biting with the tooth, or purulent suppuration from a sinus tract or from an osseous defect
• Acute or persistent pain, swelling, or both
• Exacerbation of chronic symptoms: patient’s tooth history showed previous chronic symptoms, becoming acute before extraction
• Asymptomatic: a suspicious radiographic feature for VRF was detected on routine examination with no symptoms.

Teeth were extracted either by simple procedure (75 cases) or during a surgical exploratory procedure (35 cases). Surgical exposure was performed only in cases of uncertain diagnosis or surgical need caused by loss of coronal tooth structure. The tooth or root was extracted if the fracture was visualized at this stage. The soft granulomatous tissue was curetted and the socket was rinsed with saline solution, followed by hemostasis procedures. The clean socket was inspected for type of bone resorption on the buccal and palatal or lingual plates and interproximal walls with a periodontal probe. For each case, bone loss was recorded
graphically on separate paper during and after the surgical procedure or extraction.

Chi-square statistical analysis was carried out to correlate the patient’s symptoms and the pattern of buccal plate resorption.

**RESULTS**

Out of 110 patients, 63 had chronic symptoms, 18 had acute symptoms, 17 had exacerbation of chronic symptoms, and 12 were asymptomatic (Fig 1). There was a significant positive correlation \( (P < .0001) \) between the combination of chronic symptoms and those of exacerbation type to the amount and pattern of bone resorbed. Significantly more bone was resorbed in patients with chronic symptoms than those of the acute type.

Two main types of alveolar bone resorption were observed in the buccal plate: dehiscence and fenestration. Dehiscence, in which the bone loss had a triangular V-shape, was observed in 100 cases (91%) (Table I). The tip of the triangle was at the apical point of the resorption, which was also the most apical point of the root fracture. The base was an imaginary line between the 2 most distant points of the coronal bone loss (Fig 2). Fenestration, in which bone loss had an oval shape and a bridge of bone preserved coronally (Fig 3) was present in 10 cases (9%). In these cases, the fracture line appeared only approximate to the bone resorption area and did not extend coronally as in dehiscence.

Lingual resorption was noted in some of the cases in addition to the buccal side. The total amount of resorption was less than on the buccal, and the tip of the triangle was rounded, similar to the letter U.

In the maxillary premolars (n = 66), 59 (89.3%) showed the dehiscence type and 7 (10.7%) showed fenestration resorption (Table I). Only 1 case with the fenestration type was shown in the mandibular premolars (Table I). Most of the mesial roots of the mandibular molars (n = 31) had a dehiscence pattern of bone resorption (29 cases, 93.5%) and only 2 cases (6.5%) had the fenestration pattern.

The correlation between the 100 cases of dehiscence resorption and the types of symptoms before extraction is shown in Fig 1. In 72 cases of dehiscence, “wide” bone resorption was observed, including resorption of the interproximal bone. “Narrow” bone resorption was seen in the remaining cases.

Chi-square statistical analysis showed that a “wide” bone resorption pattern was typical to the chronic and exacerbation types of symptoms and the “narrow”

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**Table I.** Type of buccal plate resorption according to tooth type

<table>
<thead>
<tr>
<th>Type of Resorption</th>
<th>Maxillary premolars</th>
<th>Mandibular premolars</th>
<th>Root mandibular molars</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dehiscence (%)</td>
<td>59 (89.3)</td>
<td>12 (92.3)</td>
<td>29 (93.5)</td>
<td>100 (100)</td>
</tr>
<tr>
<td>Fenestration (%)</td>
<td>7 (10.7)</td>
<td>1 (7.7)</td>
<td>2 (6.5)</td>
<td>10 (100)</td>
</tr>
<tr>
<td>Total (%)</td>
<td>66 (60.0)</td>
<td>13 (11.8)</td>
<td>31 (28.2)</td>
<td>110 (100)</td>
</tr>
</tbody>
</table>

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Fig 1. Patients’ symptoms and pattern of buccal plate resorption (dehiscence).

Fig 2. “Narrow” type resorption of buccal plate (dehiscence) in maxillary second premolar, after complete removal of granulation tissue. Intact bone can be seen in interproximal areas.
type of bone loss ($P < .0001$) was seen more often in acute cases.

**DISCUSSION**

Tamse et al\(^2\) evaluated 92 cases of VRF in endodontically treated teeth of various tooth types and found the typical deep osseous defect in 67.4% of the cases. This was considered to be a characteristic sign in endodontically treated vertically fractured teeth. Meister et al\(^5\) documented 32 cases of VRF verified with surgical procedures. A bony defect was found in 30 cases. Because premolar teeth and mesial roots of mandibular molars are the 2 major groups of teeth and roots most prone to vertical root fracture,\(^6-8\) it was decided to evaluate the amount and type of resorption and the correlation to the patients' symptoms in these groups.

The most typical pattern of bone resorption seen in the present study was the “dehiscence” in the buccal plate, which is prone to rapid resorption. Initially, when the thin buccal plate is resorbed, a narrow bony cleft develops and resorbs in an apicocoronal rather than in a lateral direction, that is, propagate with the fracture to form an oblong triangle\(^9\) (Fig 2). At a later stage, it becomes wider in a diagonal direction and resorbs the interproximal areas (Fig 4). This was the typical feature seen after reflection of the flap and cleaning the soft tissue (Fig 5). On the lingual side, the spongy bone and the thicker cortex created a “shield phenomenon” in which the spongy bone resorbed backwards first and later propagated laterally, forming a shallow rounded U-shape resorption while the height of the plate is preserved.

The “fenestration” type of bone resorption occurs when the VRF is located somewhere along the root, usually on the buccal side, but not exceeding to the coronal margin or the apical part. In this case, because bone loss is opposite the fracture site, the bone in the other areas is intact (Fig 3). The only clinical sign in the 10 cases of fenestration was an abscess, similar to a dentoalveolar abscess of endodontic origin. There was no pocketing in the gingiva, and no osseous defect could be detected before flap reflection. In these cases, direct bacterial invasion through the periodontal sulcus was not the cause of the inflammation. The source of inflammation was probably the root canal itself, as suggested by Walton et al.\(^10\)

Rud and Omnell\(^11\) tried to correlate the amount of bone resorption in VRF cases and the radiographic appearance. They stated that a fracture on the lingual and buccal sides of the root (which is the usual site) would take some time before bone destruction extends interproximally where it could be seen in the radiograph. Therefore, radiographic evaluation is limited when resorption is “narrow.” Only when resorption starts to exceed laterally to the interproximal bone, or when the VRF is more oblique because the bone resorbs opposite to it, will it be possible to see on the radiograph. Because it is difficult to make a diagnosis from a radiograph,\(^11,12\) a surgical evaluation is essential.\(^4\)

The mechanism of bone resorption is related to bacterial infection, followed by chronic inflammation in which granulation tissue replaces the bone as observed in all the VRF cases (Fig 5). It is speculated that the inflammatory process progresses apically, followed by rapid bone resorption in this specific area.\(^5\) Although Walton et al\(^10\) could not determine the precise etiologic factor which caused the osseous defect, they suggest that bacterial infection from the
oral cavity probably entered the area when the fracture communicated with the periodontal sulcus; granulation tissue in these cases replaces the resorbed bone along the fracture line and fills the entire defect (Fig 5). Bacterial infection from the root canal and excess sealer are other irritants that could add to the inflammatory process.10 When the fracture starts to separate, fragments communicate with the periodontium, and percolation of tissue fluids occurs.10

Cases of VRF associated with acute symptoms showed minimal interproximal bone loss in this study. It is conceivable that in these cases, the exposure time to infection was minimal and consequently, the resorption process did not proceed further. The 70 patients who appeared with chronic symptoms had a long exposure time to inflammation; thus, a large amount of bone was already resorbed. In some cases with chronic symptoms, the lingual aspects were also involved.

Bone loss in periodontal disease and the dehiscence type resorption in VRF cases both originate from the periodontal sulcus apically.13 However, bone loss in VRF progresses much faster than in periodontal disease. Differences in the bone resorption pattern14 and progression in cases of VRF could be attributed to rapid bacterial invasion through the epithelial attachment in the fracture area.15 The fracture line allows a high concentration of irritants to bypass the defense line of epithelial attachment. This creates easy passage to the unlined tissues and intensifies the inflammation, resulting in resorption of the adjacent thin buccal plate. In periodontal disease, the epithelial attachment prevents ingress of irritants to the apical periodontal tissues. Therefore, bone resorption progresses slowly around the root.

Because there is no single pathognomonic sign to verify VRF, exploratory surgery is essential at an early stage. When extraction is needed, the involved tooth (or root) should be removed without delay to prevent further resorption of the bony plates. The pattern of bone resorption in VRF cases, as shown in this study, will be helpful when an exploratory surgical flap procedure is performed.

REFERENCES


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