

The Implant Retrograde Peri-implantitis Induced Neighboring Healthy Tooth

Chronic Apical Periodontitis : A Case Report

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BACKGROUND

Retrograde peri-implantitis (RPI) is defined as a clinically symptomatic periapical lesion that develops following implant placement while maintaining normal bone-to-implant contact at the coronal aspect. RPI can lead to dental implant failure or trigger periapical periodontal inflammation in adjacent natural teeth. Managing periapical infection in a natural tooth adjacent to an implant with RPI is challenging. This is because RPI can originate from various factors, including infection in adjacent natural teeth, bone damage during the implant procedure, implant overloading, or pre-existing alveolar bone infection. Correct diagnosis is crucial for the appropriate treatment of RPI, whether it arises from the dental implant or a natural tooth.

AIM

This rare case report describes an instance where RPI at implant site #46 induced pulp necrosis and periapical infection in the neighboring second premolar (#45).

MATERIALS&METHODS

A 69-year-old male patient underwent extraction of the lower right first molar (#46) due to a vertical root fracture (Figure 01). After 6 months, a dental implant (Camlog 5.0 x 11 mm) was placed in the #46 site, achieving adequate primary stability at the time of placement (Figure 02,03). However, two months postoperatively, RPI was observed around implant #46 on a periapical radiograph. Additionally, the gingiva surrounding the adjacent second premolar (#45) showed signs of edema and mild swelling. Pulp vitality testing with an electric pulp tester (EPT) revealed a gradual loss of vitality in #45, leading to total pulp necrosis. Following endodontic treatment, the symptoms subsided, and the gingival sinus tract disappeared. The final prosthesis for #46 was completed, but radiographic evidence of RPI presence (Figure 04).



Figure 1: Tooth 46 has a VRF .(20140529) (L)

Figure 2: Tooth 46 was extracted. (20140901) (R)

Nevertheless, 22 months later, the gingival swelling and sinus tract around #45 reappeared (Figure 05). Radiographic examination revealed the gutta-percha material tracing back to the RPI area of implant #46 (Figure 06, 07). Despite endodontic re-treatment and subsequent extraction of #45, the symptoms persisted. Surgical periapical curettage was performed around implant #46, during which small fragments of black soft tissue were removed, and the alveolar bone around the implant apex was curetted.

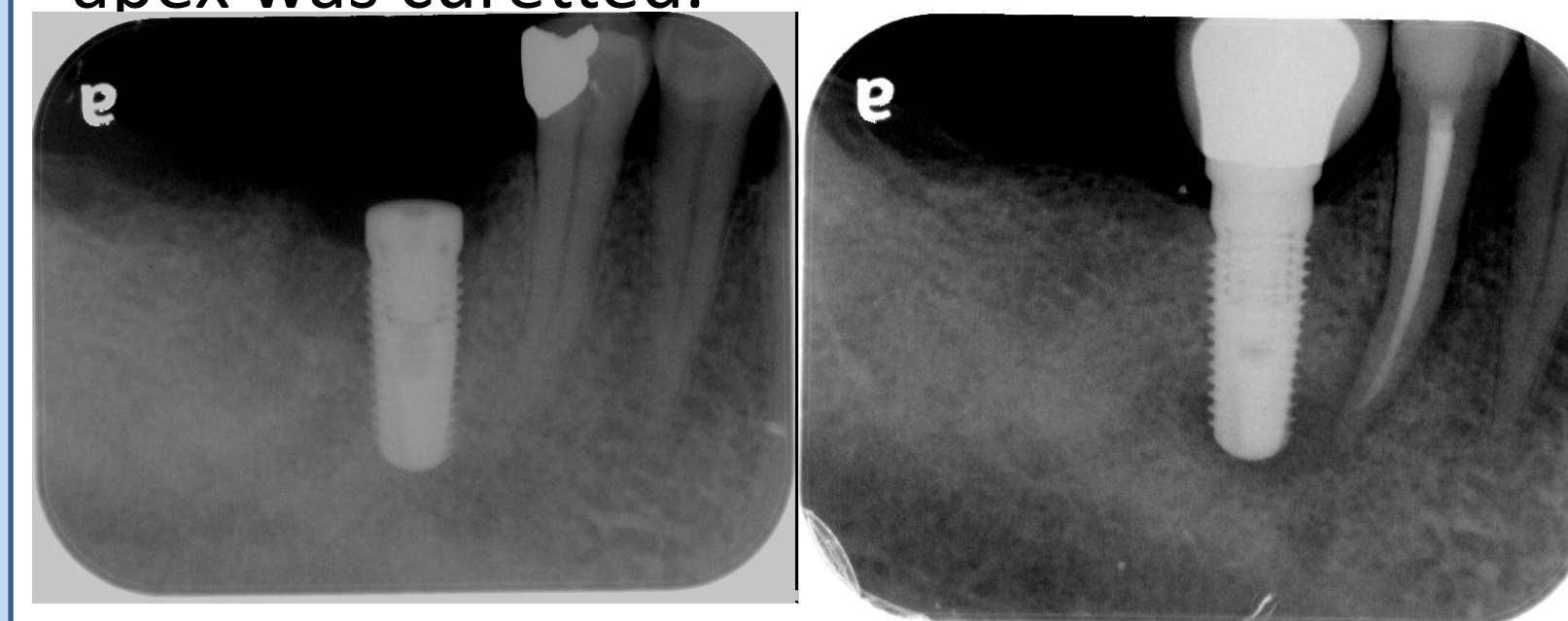


Figure 3: An implant was placed.(20150225) (L)

Figure 4: Tooth 45 underwent endo tx. (R)
(20161027)(R)

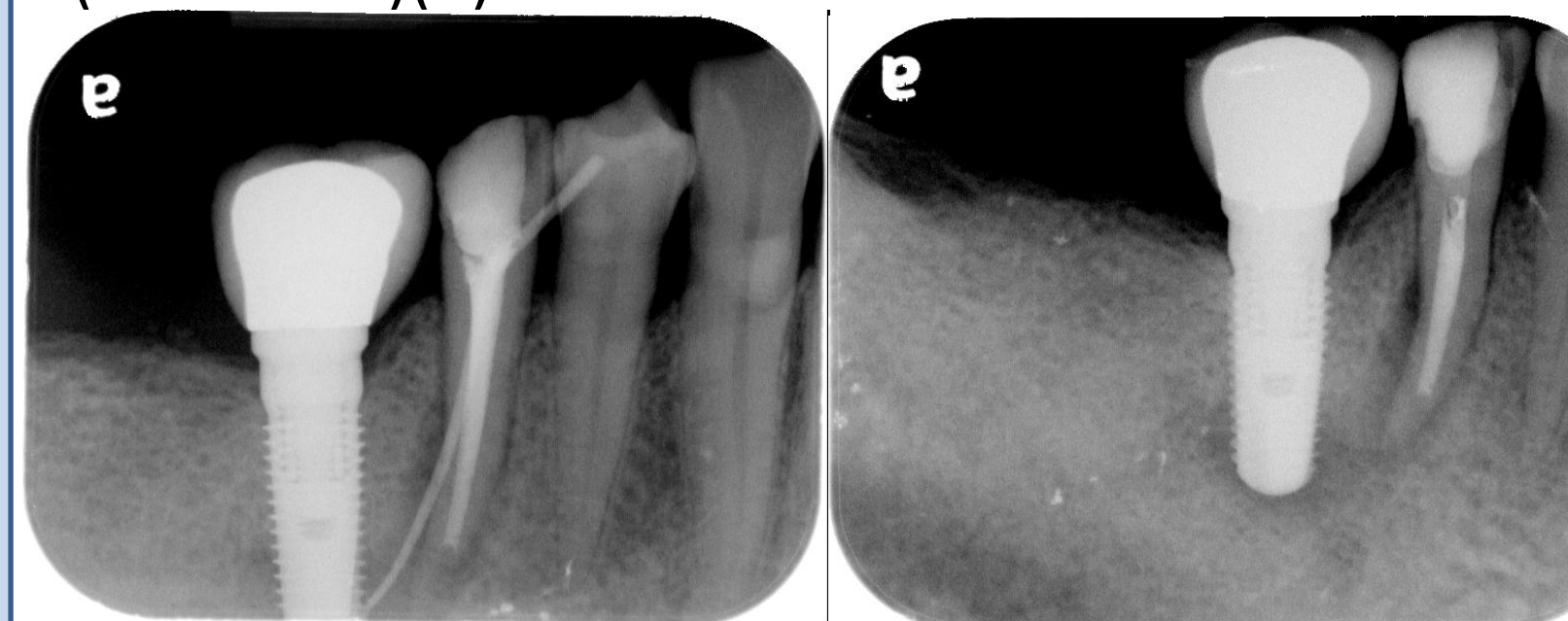


Figure 5: A sinus tract has appeared around the implant.(20170612)(L)

Figure 6: Tooth 45 was extracted (20170927)

Freeze-dried bone allograft (FDBA) was packed into the alveolar bone space at the implant apex. This intervention ultimately resulted in symptom relief.

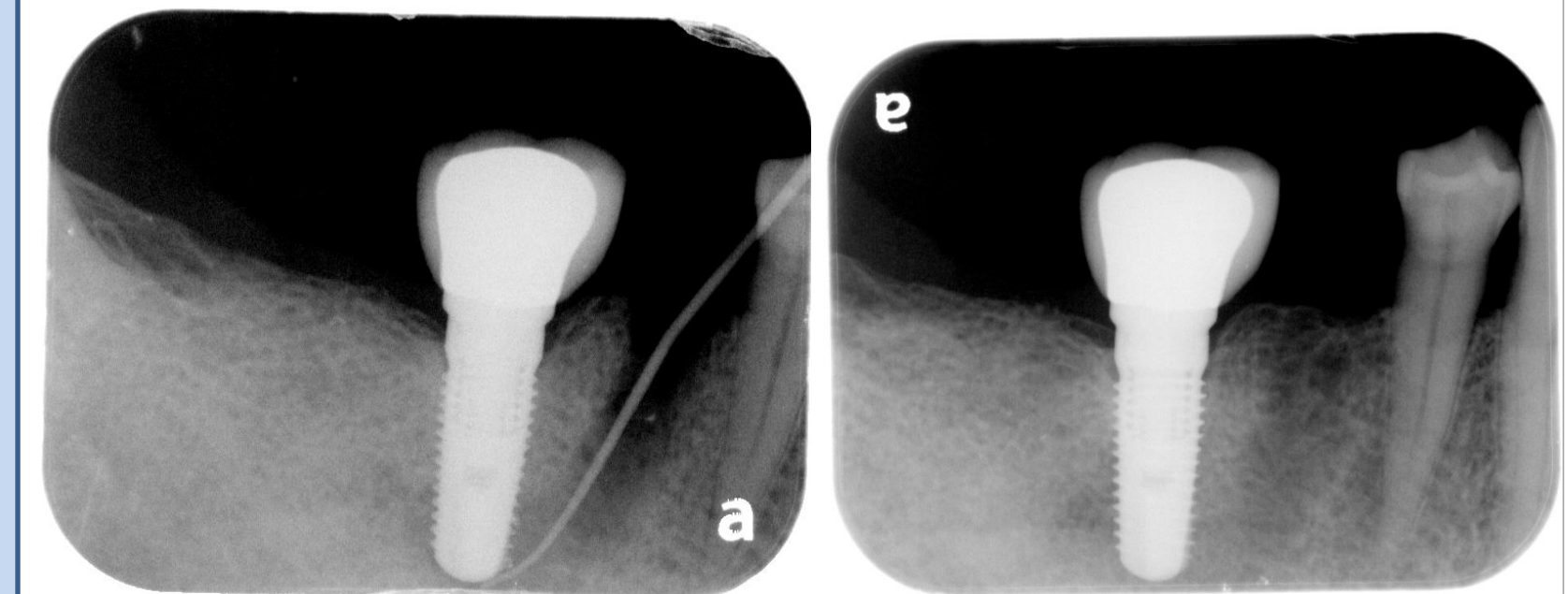


Figure 7: The sinus tract was traced by a GP point, which led to the implant.(20180115)(L)

Figure 8: New bone growth was noted.(20210321)

RESULTS

The lower right second premolar (#45) was extracted. The implant at site #46 remained functional and healthy after six years of follow-up (Figure 08).

CONCLUSIONS

This case report demonstrates that RPI at a dental implant site can induce pulp necrosis and chronic apical periodontitis in adjacent natural teeth. Therefore, treating RPI should be prioritized over endodontic treatment of the affected natural tooth."

DISCLOSURE OF INTEREST

No funding was available for this case report.