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Case Report

급성괴사성궤양성 치은염을 닮은 재발성 허피스 구내염

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Abstract

Recurrent Herpes-Stomatitis Mimicking Acute Necrotizing Ulcerative Gingivitis

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Herpes simplex is caused by viruses of the herpesvirus hominus family. HSV have four categories: type 1, 2, 6, and 8. Generally HSV-1 affects the mouth. Once infected by HSV, the person's infection is permanent. Retrograde transport through adjacent neural tissue to sensory ganglia leads to a lifelong latent infection. Recently, we treated a patient with recurrent herpes-stomatitis mimicking acute necrotizing ulcerative gingivitis (ANUG). The results were satisfactorty so we report this case. 31 years old male patient showed sore throat, gingival ulceration, palpable both submandibular lymph node, and sulcular pus formation around posterior decayed teeth. This is the third time he has suffered from this symptom. Tentative diagnosis was acute necrotizing ulcerative gingivitis. Antibiotic therapy was started. But, intraoral symptom got worse in process of time. Especially ulcer of marginal gingiva got worse. Viral disease was suspected. We carried out viral cultivation. At the same time topical application of antiviral ointment (herpecid®) was performed on oral ulcer unilaterally for the purpose of diagnosis and reducing pain experimentally. The next day pain was decreased dramatically on application area. Basing on the viral cultivation and clinical effect of antiviral ointment (herpecid®), we have diagnosed it as a recurrent herpes-stomatitis and concluded that viral infection was major cause of disease and bacterial infection was secondary.

Key words: Acute necrotizing ulcerative gingivitis (ANUG), Recurrent Herpes-Stomatitis

Introduction

Herpes simplex is caused by viruses of the herpesvirus hominus family. There are four groups of herpes viruses that cause disease in humans: herpes simplex, varicella-zoster, cytomegalo virus, and Epstein-Barr virus. HSV

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have four categories: type 1, 2, 6, and 8. Generally HSV-1 affects the mouth. Infection with HSV-1 is almost universal. Most individuals bocome infected during infancy or childhood, usually as a result of close contact with family members or friends who carry the virus. Once infected by HSV, the person's infection is permanent. Retrograde transport through adjacent neural tissue to sensory ganglia leads to a lifelong latent infection[1,2]. The individual lesion begins as a vesicle that appears gray or white. The vesicles quickly burst, leaving a punctate ulceration with a red base[3]. Of course, differential diagnosis is necessary from other ulcerative lesion.

Recently, we treated a patient with recurrent herpes-stomatitis mimicking acute necrotizing ulcerative gingivitis (ANUG). The results were satisfactorty so we report this case.

Case Report

31 years old male patient showed sore throat, gingival ulceration (Fig. 1), palpable both submandibular lymph node, and sulcular pus formation around posterior decayed teeth.

He said this symptom was started five days ago. In CT

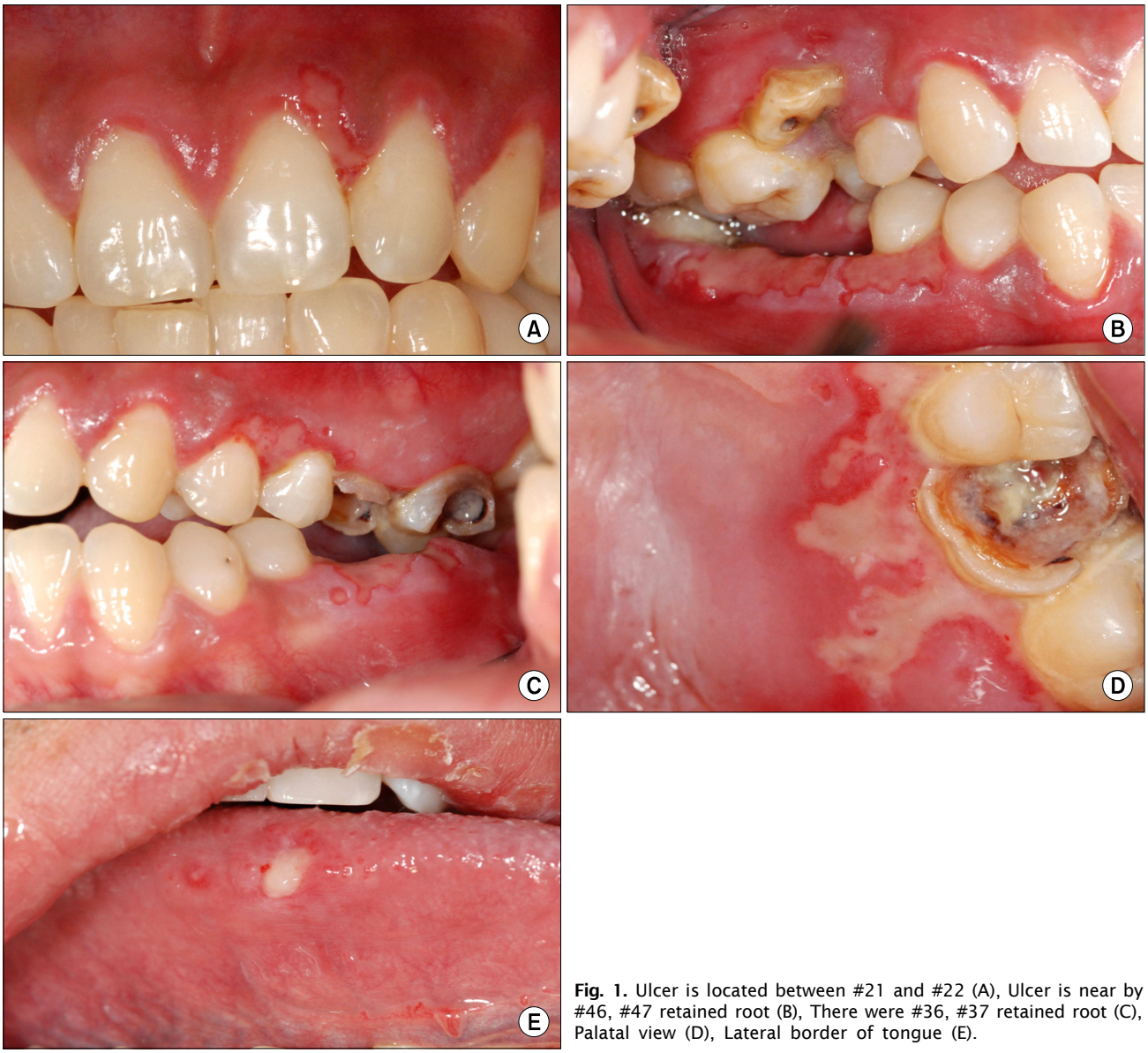

Fig. 1. Ulcer is located between #21 and #22 (A), Ulcer is near by #46, #47 retained root (B), There were #36, #37 retained root (C), Palatal view (D), Lateral border of tongue (E).

view slight platysama muscle swelling was seen. In history taking he had fear of dental treatment, recently he was under stress with a matter of marriage. This is the third time he has suffered from this symptom. First time was in his military service period, second time was when discharged from military service and this time is more serious than the others. Unexpectedly in familial history his maternal grandmother suffered from oral cancer.

After examination infection was suspected. First day of hospitalization, antibiotic therapy was started; cefazolin, isepamicin, metronidazole. The patient showed elevated body temperature 38°C. Tentative diagnosis was acute necrotizing ulcerative gingivitis. In lab test there was no oddity in number of leucocyte but ESR and CRP figure were high (respectively 7,100/mm^3, 26 mm/hr, 10.95 g/dl). Second day of hospitalization, sore throat was improved and size of palpable lymph node was decreased. But new ulcerative lesion was appear on upper labial mucosa around vermilion border. The same day of hospitalization, smear was done for diagnosis of ANUG. Third day of hospitalization, bacterial culture was done on ulcer area. But, intraoral symptom got worse in process of time. Especially ulcer of marginal gingiva got worse. Generally ulcer moved from mandibular alveolar mucosa to maxillary alveolar mucosa according to his sensation. Viral disease was suspected. Fourth day of hospitalization, we carried out viral cultivation and blood sampling for detection of herpes virus. Palatal ulcer was targeted. At the same time topical application of antiviral ointment (herpecid®) was performed on oral ulcer unilaterally for the purpose of diagnosis and reducing pain experimentally[4]. The next day pain was decreased dramatically on application area. Clinically it was diagnosed as HSV-1. Sixth day of hospitalization, general condition was improved and patient wanted discharge. When discharging antiviral ointment (herpecid®) was applicated on the whole ulcer. On follow up treatment he said piercing pain on left palate was disappeared the day after ointment application. A few days later result of bacterial culture was *Streptococcus Mitis*. This microbes are known as commensal bacteria which may play some role in dental caries[5-7].

In the light microscopic analysis of smears from the lesion there were not fusiform and spirochete microbes. Viral culture showed positive about herpes simplex virus type 1. The virus was not found in blood sample. For the present, the symptoms of the patient has been disappeared. Basing on the viral cultivation and clinical effect of antiviral ointment (herpecid®), we have diagnosed it as a recurrent herpes-stomatitis and concluded that viral infection was major cause of disease and bacterial infection was secondary.

Discussion

Clinically, recurrent intraoral herpetic lesions are almost always located on the oral mucosa that is tightly bound to periosteum. The hard palate, near the posterior aspect at the reflections of the vault, is the most common location, followed by the attached gingiva of alveolar ridge of the maxilla or mandible. The individual lesion begins as a vesicle that appears gray or white. The vesicles quickly burst, leaving a punctate ulceration with a red base[3]. At first, this case was misunderstood as ANUG because of interdental gingival ulceration, pain, elevated BT, poor oral hygiene, emotional stress, smoking habit[8,9]. But oral smear didn't express fusiforms and spirochetes. Nevertheless, bacterial infection must have played a role in this case.

The oral smear was the primary tool used to provide laboratory confirmation of clinical diagnosis of oral ulcerations of RHSV. When patients had ulceration that seemed to satisfy the clinical criteria for lesions of RHSV, the lesions were photographed and cytologic smear was done[3]. According to Griffin et al[4,10,11], under the light microscope, the presence of "balloon degeneration" of epithelial cells and the presence of syncytial multinucleated giant cells with molded nuclei and peripherally marginated chromatin were used as the criteria for posive finding for cytopathogenic effect of herpes simplex virus. The presence of herpetic cytopathogenic effect on oral smear necessarily implies an active infection[3]. In addition, Dwight and his colleagues[3] pointed out ulcerations three days old or less appear optimum for detection of virus and cytologic change. They supposed that the cytopathogenic effect is transient and that the herpes virus is isolable most easily in the early stages of the infection[3]. In this study, however, we could not find "balloon degeneration" in oral smear. This negative result maybe related to transient char-

acteristic of the cytopathogenic effect.

Ryan[1] said gold standard for HSV-1 diagnosis is viral isolation by tissue culture. In our study, rapid shell vial method was used as a way of viral isolation. The result was positive. However, isolation of herpes virus does not, per se, prove an active infection since virus can occasionally be isolated in the saliva of apparently normal individuals[12]. When virus is consistently found together with characteristic lesions, this fact is significant[3].

As a result of history taking and clinical examination presuming it as ANUG, we priscribed metronidazole and cefazolin etc, which had been usually used for head and neck infection. After antibiotic medication the patient was on the mend. But intraoral ulcer and pain was getting worse, we doubted viral disease. Oral smear and viral isolation was carried out. At the same time antiviral ointment was applied experimentally. Response of patient was dramatic. Finally we could diagnose that it was recurrent herpes-stomatitis mimicking ANUG by clinically and laboratorially.

References

1. Ryan WA. Herpes simplex virus: clinical presentation and treatment. Dent Today 2001;20:64-7.
2. Paolo G. Herpes Simplex Virus Type 1 infection: overview on relevant clinico-pathological features. J Oral Pathol Med 2008;37:107-21.
3. Weathers DR, Griffin JW. Intraoral ulcerations of recurrent herpes simplex and recurrent aphthae: two distinct clinical entities. J Am Dent Assoc 1970;81:81-7.
4. Griffin JW. Recurrent intraoral herpes simplex virus infection. Oral Surg Oral Med Oral Pathol 1965;19:209-13.
5. Rudney JD, Larson CJ. Identification of oral mitis group streptococci by arbitrarily primed polymerase chain reaction. Oral Microbiol Immunol 1999;14:33-42.
6. Elliott D, Harrison E, Handley PS, et al. Prevalence of Csh-like fibrillar surface proteins among mitis group oral streptococci. Oral Microbiol Immunol 2003;18:114-20.
7. Morita E, Narikiyo M, Nishimura E, et al. Molecular analysis of age-related changes of Streptococcus anginosus group and Streptococcus mitis in saliva. Oral Microbiol Immunol 2004;19:386-9.
8. Johnson BD, Engel D. Acute necrotizing ulcerative gingivitis. A review of diagnosis, etiology and treatment. J Periodontol 1986;57:141-50.
9. Shields WD. Acute necrotizing ulcerative gingivitis. A study of some of the contributing factors and their validity in an Army population. J Periodontol 1977;48:346-9.
10. Blank H, Burgoon CF, Baldridge GD, Mcarthy PL, Urbach F. Cytologic smears in diagnosis of herpes simplex, herpes zoster, and varicella. J Am Med Assoc 1951;146:1410-2.
11. Cooke BE. Exfoliative cytology in evaluating oral lesions. J Dent Res 1963;2:343-7.
12. Rustigian R, Smulow JB, Tye M, Gibson WA, Shindell E. Studies on the latentinfection of skin and oral mucosa in individuals with recurrent herpes simplex. J Invest Derm 1966;47:218-21.