

Case Report

Acute Prevertebral Abscesses Caused by Bacterial-infected Traumatic Tooth Fractures

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We report a case of acute prevertebral abscess caused by traumatic tooth fractures in a 77-year-old Japanese man. After being transferred to our hospital the patient was initially diagnosed with a neck hematoma; however, blood culture showed *Streptococcus parasanguinis*, an oral bacterium, and an MRI examination suggested prevertebral abscesses. Tooth fractures, severe periodontitis, and peri-implantitis with *Streptococcus parasanguinis* were observed. Antibiotics were administered and fractured teeth were extracted. The patient's condition then gradually improved. We concluded that bacteremia caused by traumatic tooth fractures induced the acute prevertebral abscesses.

Key words: prevertebral abscess, deep neck infection, periodontal disease, peri-implantitis, *Streptococcus parasanguinis*

Head and neck infections are severe infectious diseases that present with serious complications such as airway obstruction, mediastinitis, mediastinal abscesses, sepsis, disseminated intravascular coagulation, jugular septic thrombophlebitis, and pericarditis [1]. The head and neck infections include submandibular space infections, lateral pharyngeal space infections, parapharyngeal infections, danger space infections, spinal epidural abscesses, retropharyngeal abscesses, and prevertebral abscesses.

A prevertebral abscess is a deep neck infection and is formed in the prevertebral space, between the prevertebral fascia and vertebral bodies, extending from the base of the skull caudally to the coccyx. This type of abscess often occurs secondarily to osteomyelitis, disci-

titis, or hematogenous spread [2]. Gram-positive organisms such as *Staphylococcus aureus* are the predominant microorganisms in prevertebral abscess [3]. The major complication of these deep infections is spinal cord compression, and irreversible paralysis occurs in 4-22% of patients [4]. Therefore, the early detection and treatment of prevertebral abscess is important, but such diagnosis can be difficult because only 75% of patients present with back or neck pain, 50% present with fever, and 33% develop neurological deficits ranging from nerve root pain to paralysis [5]. Indeed, half of all prevertebral abscess cases are initially misdiagnosed [6]. With respect to treatment, there are 2 main approaches: surgical drainage of prevertebral space infections, and conservative therapy with antibiotics. In either case, a precise diagnosis is required to locate

and identify the source of infection.

Odontogenic infections (e.g., dental caries, periodontitis, and periapical periodontitis) may spread to the deep neck spaces [7]. Acute odontogenic maxillofacial infections may require lengthy hospitalization [8]. However, odontogenic infections are difficult to diagnose because basic medical tests do not identify oral infections such as periodontitis and periapical periodontitis. In the present case report, the patient was initially diagnosed with a hematoma in front of the cervical vertebrae but was eventually re-diagnosed with a prevertebral abscess upon the detection of *Streptococcus parasanguinis* during blood culture examinations, with the confirmation of an odontogenic infection as the source of infection. He was treated with antibiotics and the infected teeth were then extracted, resulting in the gradual improvement of his symptoms.

Patient

The patient was a 77-year-old Japanese man who was transferred to the hospital due to a head injury after he fell when exiting a car. He had no hospitalization or surgery history. He smoked ~12 cigarettes/day from the age of 20 to 40 years old. He was a social drinker. He had a family dentist from whom he had received implants and routine dental checkups. Periodontal disease in his maxillary molars was suggested, but he did not undergo aggressive dental treatment because of the lack of symptoms. He lived with his wife and had no significant family medical history. He had owned and run a business that was later taken over by his son.

Physical findings at admission. On his admission, the patient's temperature was 35.9°C, his blood pressure was 99/60 mmHg, and his pulse was 88 beats per minute. The other results of laboratory tests are shown in Table 1. He was alert and his vital signs were normal. He had an approximately 3-cm laceration above his left eyebrow from the fall. His blood test results were within the normal limits. A head computed tomography (CT) scan revealed acute subdural hematomas on the left side. The laceration was stitched, and his progress was closely monitored.

Treatment progress. The next day, the patient had fever, headache, and gradually increasing pain in his left shoulder and posterior cervical region. A lung CT scan did not indicate any inflammation. He did not present any symptoms indicating infection—e.g.,

increased sputum, dyspnea, or pain during urination. On the 3rd day post-injury, his fever reached 39.6°C (Fig. 1A). A blood test revealed high levels of C-reactive protein (CRP), white blood cells (WBC), and neutrophils (Neut) (Fig. 1B). A blood culture examination was then conducted because of the suspected infection (Table 2). On the 4th day post-injury, a neck MRI examination indicated high-intensity signals on T2-weighted images and no-intensity signal on T1-weighted images at the front of the cervical vertebrae (Fig. 2A-C). A hematoma was suspected rather than an infectious disease such as a retropharyngeal abscess. On the 5th day post-injury, blood culture examination results confirmed the presence of *S. parasanguinis*, and we treated him with ceftriaxone (Rocephin; Hoffman-La Roche, Basel, Switzerland). On the 6th day post-injury, as the patient's liver function decreased (Fig. 1C), the antibiotics were changed to ampicillin/sulbactam (Yucion-S; Sawai Pharmaceutical, Osaka, Japan).

On the 9th day post-injury, another neck MRI indicated an edema in the soft tissue in front of the cervical vertebrae in addition to a bilateral mass in the longus capitis muscles (Fig. 2E, F). His symptoms deteriorated from those observed during the previous examinations. Based on these results, he was finally diagnosed with prevertebral abscesses, and dentists in the hospital were asked to investigate whether there was any source of infection in his oral cavity because the bacterium was one of the major early colonizers of oral biofilms on the tooth surface [9].

Oral examinations revealed maxillary anterior tooth fractures due to the patient's fall (Fig. 3A, B), which resulted in severe periodontitis in the maxillary molar on both sides and peri-implantitis on the left maxillary premolar region (Fig. 3D). The fractured teeth were extracted on the same day (Fig. 3C). A bacteriological examination was performed in the gingival pockets of all areas, revealing the presence of *S. parasanguinis*. The numbers of bacteria were higher around the dental implants than the fractured teeth and within the periodontitis area. Other molar teeth with severe periodontitis were also extracted on the 20th and 23rd days post-injury, and the patient's condition then gradually improved (Fig. 1B). He was discharged on the 38th day post-injury.

The patient's dental implants were extracted 90 days post-injury at another hospital. Ampicillin/sulbactam

Table 1 The results of laboratory findings

Complete Blood Count

| Test name | Reference | Units | Day 0 | Day 37 | |
|-----------|-------------|------------------|-------|--------|------|
| WBC | 3,500~8,950 | / μ L | 4,600 | 3,520 | |
| RBC | 440~560 | 10^4 / μ L | 449 | 362 | Low |
| Hb | 13.4~17.5 | g/dL | 15 | 11.6 | Low |
| Hct | 40~52 | % | 43.9 | 35.1 | Low |
| PLT | 12.9~37.2 | 10^4 / μ L | 16.8 | 23.8 | |
| MCV | 85~100 | fl | 97.8 | 97 | |
| MCH | 27~36 | pg | 33.4 | 32 | |
| MCHC | 32~36 | g/dL | 34.2 | 33 | |
| Neut | 45~71 | % | 51.6 | 56 | |
| Lymph | 25~45 | % | 38.7 | 29.3 | |
| Mono | 3~7 | % | 8 | 11.6 | High |
| Eosin | 1~5 | % | 1.5 | 3.1 | |
| Baso | 0~1 | % | 0.2 | 0 | |
| Neut# | 1,575~6,355 | / μ L | 2,370 | 1,970 | |
| Lymph# | 875~4,028 | / μ L | 1,780 | 1,030 | |
| Mono# | 105~627 | / μ L | 370 | 410 | |
| Eosin# | 35~448 | / μ L | 70 | 110 | |
| Baso# | 0~89.5 | / μ L | 10 | 0 | |

#: number

Liver function

| Test name | Reference | Units | Day 0 | Day 37 | |
|---------------|-----------|-------|-------|--------|------|
| AST(GOT) | 5~40 | IU/L | 44 | 17 | High |
| ALT (GPT) | 5~40 | IU/L | 30 | 15 | |
| ALP | 102~349 | IU/L | 224 | 211 | |
| γ -GTP | 0~73 | IU/L | 25 | 28 | |
| LD (LDH) | 120~240 | IU/L | 432 | 127 | High |
| Alb | 3.9~5.8 | g/dL | 4.8 | | |

Renal function

| Test name | Reference | Units | Day 0 | Day 37 | |
|-----------|-----------|-------|-------|--------|--|
| UN | 8~20 | mg/dL | 17.4 | 9.9 | |
| CRE | 0.61~1.04 | mg/dL | 0.83 | 0.81 | |
| eGFR | | | 68.4 | 70.2 | |

Urine alysis

| Test name | Reference | Units | Day 1 | Day 21 | |
|-------------------------------------|-----------|-------|---------|---------|------|
| Urine protein | (-) | | 1+ | - | High |
| Urine blood | (-) | | - | - | |
| Urine sugar | (-) | | \pm | - | |
| Urine urobilinogen | (\pm) | | normal | normal | |
| Urine ketone bodies | (-) | | - | - | |
| Urine bilirubin | (-) | | - | - | |
| Urinary sediment (RBC) | | | under 1 | under 1 | |
| Urinary sediment (WBC) | | | 1~4 | 1~4 | |
| Urinary sediment (epithelial cells) | | | under 1 | 1~4 | |

Immune serum inspection

| Test name | Reference | Units | Day 0 | Day 37 | |
|--------------|-----------|-------|-------|--------|--|
| CRP | 0~0.3 | mg/dL | 0.028 | 0.217 | |
| HBs antigen | | | - | | |
| HCV antibody | | | - | | |

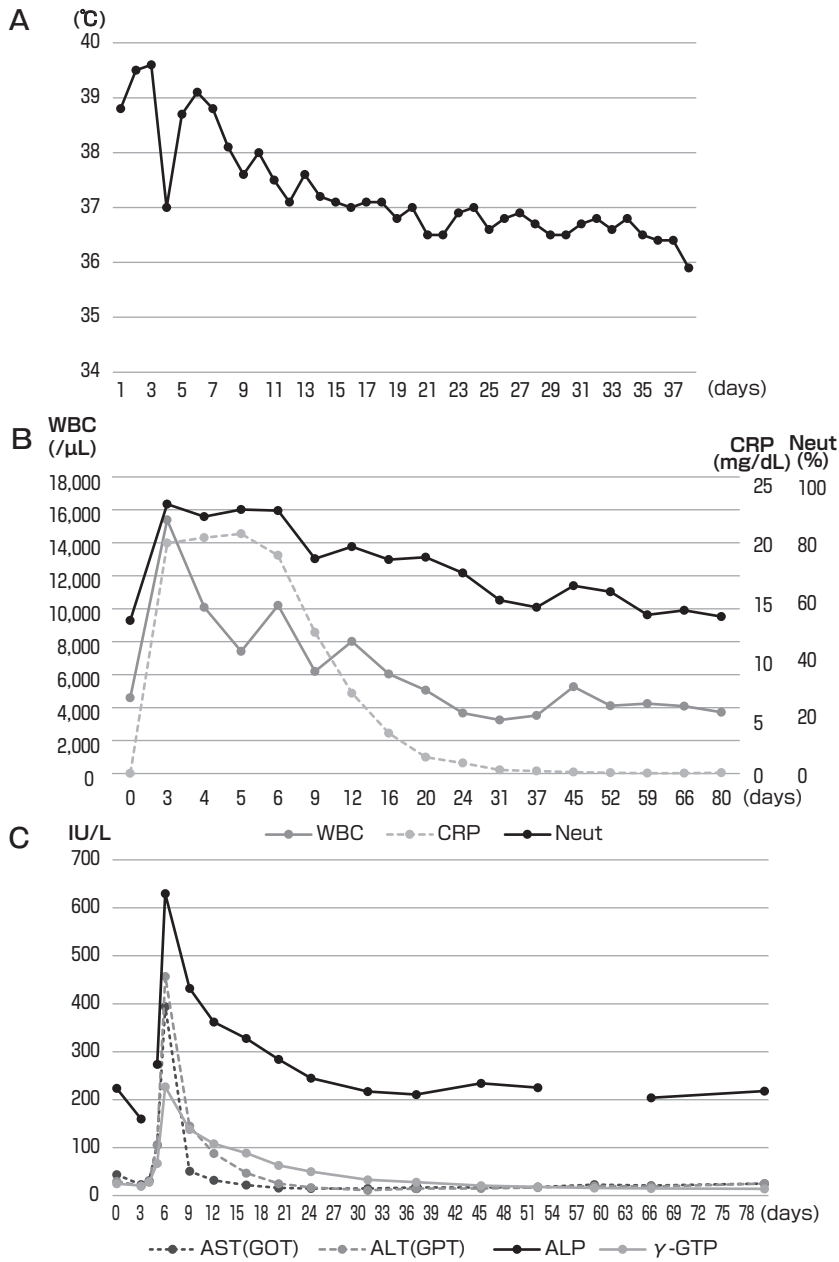


Fig. 1 Hematological and temperature changes during treatments. The changes in the patient's (A) temperature; (B) C-reactive protein (CRP), white blood cell (WBC), and neutrophil (Neut) levels; and (C) aspartate aminotransferase (AST [GOT]), alanine aminotransferase (ALT [GPT]), alkaline phosphatase (ALP), and γ -glutamyl transpeptidase (γ -GTP) during the treatments.

was administered for 33 days after discharge, followed by cefcapene pivoxil hydrochloride hydrate (Flomox; Shionogi & Co., Osaka, Japan) until 59 days. After 59 days, the treatment was changed to amoxicillin/clavulanic acid (Augmentin; GlaxoSmithKline, London) because of the onset of skin rashes. The amoxicillin/clavulanic acid was terminated 125 days after the injury because of the patient's full recovery. *S. parasanguinis* was not detected by bacteriological examinations of the

oral cavity were performed on the 90th, 118th, and 125th day post-injury.

Discussion

In this case, we performed not only a radiographic analysis but also a bacteria examination and oral examination for the diagnosis and treatment of deep-neck disease. Based on the results, we suspected that

Table 2 Bacterial culture examination

| Day | Place | Specimen | Method | Bacteria |
|---------|------------------------------------------------|------------------------|--------------------|----------------------------------------------------------------------------------------------------------------------------|
| Day 3 | left upper extremity | blood | aerobic, anaerobic | <i>S. parasanguinis</i> (+) |
| | right upper extremity | blood | aerobic, anaerobic | <i>S. parasanguinis</i> (+) |
| Day 5 | left upper extremity | blood | aerobic, anaerobic | <i>S. parasanguinis</i> (+) |
| | right upper extremity | blood | aerobic, anaerobic | <i>S. parasanguinis</i> (+) |
| | | urine | aerobic | <i>Staphylococcus spp.</i> (+) <i>Streptococcus sp.</i> (+) |
| Day 6 | lumber | spinal fluid | aerobic, anaerobic | (-) |
| Day 9 | maxillary anterior tooth | dental plaque | aerobic, anaerobic | <i>S. parasanguinis</i> (+) <i>Staphylococcus spp.</i> (+) <i>Capnocytophaga sp.</i> (+) <i>Neisseria sp.</i> (+) |
| Day 12 | left upper extremity | blood | aerobic, anaerobic | (-) |
| | right upper extremity | blood | aerobic, anaerobic | (-) |
| Day 13 | gingival pocket on right maxillary molar | gingival crevice fluid | aerobic, anaerobic | <i>S. parasanguinis</i> (+) <i>Capnocytophaga sp.</i> (+) |
| | gingival pocket on left maxillary molar | gingival crevice fluid | aerobic, anaerobic | <i>S. parasanguinis</i> (+) <i>Staphylococcus spp.</i> (+) <i>Neisseria sp.</i> (+) |
| Day 21 | | urine | aerobic | (-) |
| Day 24 | left upper extremity | blood | aerobic, anaerobic | (-) |
| | right upper extremity | blood | aerobic, anaerobic | (-) |
| Day 27 | dental implants on right maxillary premolar | gingival crevice fluid | aerobic, anaerobic | <i>S. parasanguinis</i> (3+) <i>Neisseria sp.</i> (3+) |
| | mandibular molar | dental plaque | aerobic, anaerobic | <i>Enterococcus faecalis</i> (+) <i>S. parasanguinis</i> (+) <i>Neisseria sp.</i> (+) |
| Day 90 | mandibular molar | gingival crevice fluid | aerobic | <i>S. mitis group</i> (+) <i>S. sanguis</i> (+) <i>Neisseria sp.</i> (+) |
| | maxillary molar | dental plaque | aerobic | <i>S. mitis group</i> (3+) |
| Day 118 | maxillary molar | dental plaque | aerobic | <i>S. mitis group</i> (3+) <i>Neisseria sp.</i> (2+) |
| Day 125 | mandibular molar | dental plaque | aerobic | <i>S. mitis group</i> (2+) <i>S. sanguis</i> (2+) <i>Neisseria sp.</i> (2+) |
| | maxillary molar | dental plaque | aerobic | <i>S. mitis group</i> (2+) <i>S. sanguis</i> (2+) <i>Neisseria sp.</i> (2+) |

S. parasanguinis might be the causative pathogenic bacterium of the prevertebral abscess in this case. The combination of antimicrobial administration and extraction of the affected teeth in order to immediately

remove the source of infection prevented the progression into another space and recurrence of the abscess.

Oral infections such as caries and periodontal disease cause bacteremia, followed by various complicat-

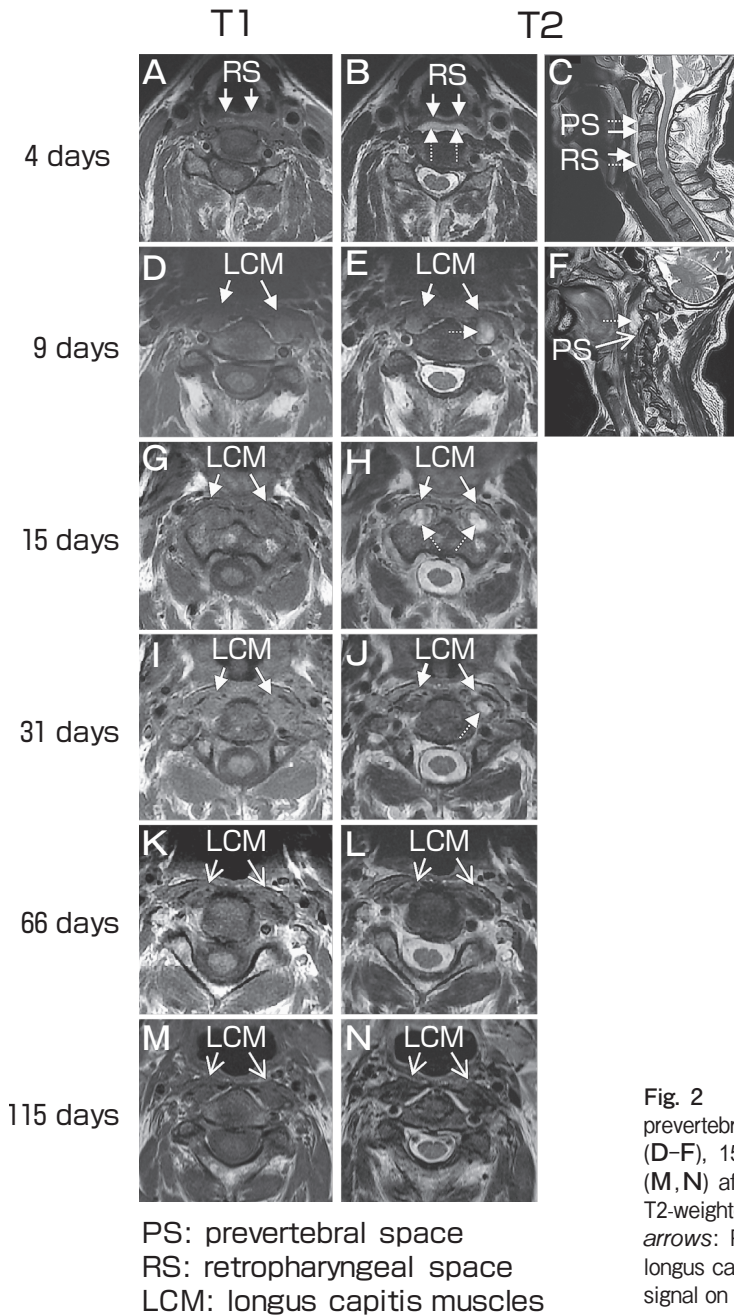


Fig. 2 Neck MRI (T1 and T2-weighted images) and changes in prevertebral abscesses. Neck MRI images at 4 days (A-C), 9 days (D-F), 15 days (G,H), 31 days (I,J), 66 days (K,L), and 115 days (M,N) after fall injury. T1-weighted images (A,D,G,I,K,M) and T2-weighted images (B,C,E,F,H,J,L,N) are shown. *White solid arrows*: Prevertebral space (PS), retropharyngeal space (RS), and longus capitis muscles (LCM). *White dotted arrows*: High-intensity signal on T2-weighted images.

ing diseases such as infectious endocarditis. However, it is difficult to harvest a specimen from such a deep infected area and to detect pathogenic bacteria, and thus a causal relationship usually cannot be confirmed between oral infections and bacteremia. We therefore usually perform various examinations such as radiographic analysis and bacteria examination for an accurate diagnosis.

It can be difficult to clearly distinguish between the retropharyngeal space and the prevertebral space with the use of MRI, but in our patient edemas or fluid collections seemed to be present in both spaces by MRI images on day 4 post-injury. The edema disappeared from the retropharyngeal space, and MRI showed that the abscess clearly remained in the prevertebral space on day 4. We suspected that the infection might spread

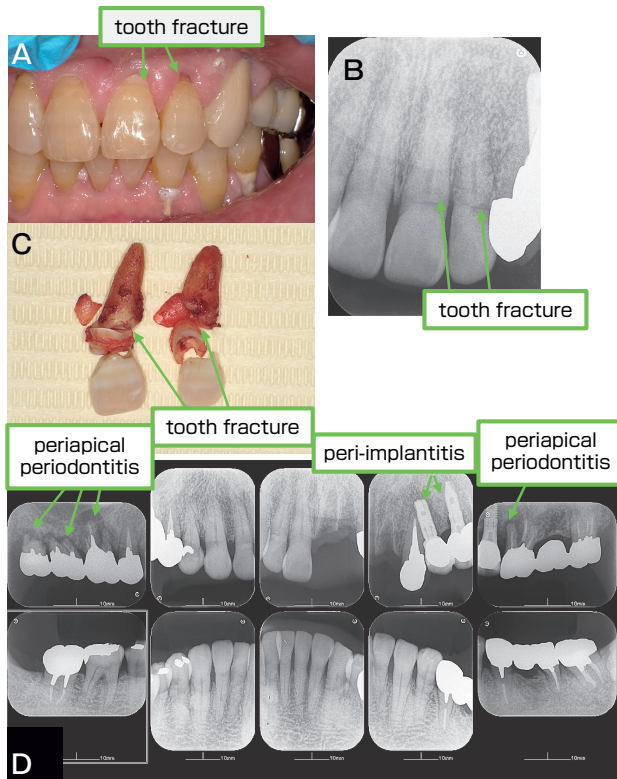


Fig. 3 Dental photography and X-rays. Dental photograph before the extraction of fractured teeth, (B) dental X-ray photo, (C) extracted fractured teeth, and (D) full mouth dental X-ray photos after the extraction of fractured teeth. *Green arrows*: Tooth fracture lines (A–C), periapical periodontitis, and peri-implantitis (D).

hematogenously because it is rare for such a lesion to extend across the prevertebral layer. Another possibility was that the infection spread directly from the lateral pharyngeal space. Unfortunately, we did not perform gadolinium-enhanced T1-weighted MRI in this patient's case, although such imaging might have helped identify the precise location of the disease.

Generally, retropharyngeal abscesses are located in the tissues in the back of the throat behind the posterior pharyngeal wall. These abscesses are caused by pharyngitis and purulent lymphadenitis, and they lead to pharyngeal pain. It rarely happens that the abscesses spread from the prevertebral space across the deep fascial layer. In contrast, prevertebral abscesses are located in the tissues in the back of or surrounding the prevertebral deep layer. They are caused by pyogenic spondylitis and tuberculous spondylitis, and they lead to neck pain. In our patient's case, the abscesses were located in prevertebral muscle and the pericervical space, and the symp-

tom of neck pain supported our diagnosis.

S. parasanguinis is the major early colonizer of dental surfaces in the human oral cavity; it enters the bloodstream by oral surgery or trauma and causes bacteremia [9]. *S. parasanguinis* has been detected in infective endocarditis, lung abscess, and brain abscess [10]. In our patient's case, we considered that *S. parasanguinis* infected the injured prevertebral muscles at the patient's fall, because we detected *S. parasanguinis* in both oral cavity samples and blood samples. This was not direct evidence, because we did not detect *S. parasanguinis* from the prevertebral abscess. We speculate that bacteremia caused by oral bacteria may have induced the patient's deep neck abscesses. We could not detect *S. parasanguinis* by culture examination after removal of the infected teeth. Consequently, the deep neck abscesses progressed toward recovery. It is thus reasonable that the oral infections were the source of the deep neck abscesses. We could not detect any anaerobic bacteria because of the culture difficulties. We therefore suspect that multiple bacteria (especially some anaerobic bacteria) were involved in the infection.

From the results of the follow-up bacteria culture examination during the patient's treatment, we observed that antimicrobial administration was effective against the bacteria in the patient's blood, but it seemed to induce drug resistance against the bacteria in the oral cavity (Table 3). We thus propose that the combination of antimicrobial treatment and the removal of the source of infection by extracting the affected teeth was useful as a treatment for prevertebral abscesses and for preventing the recurrence of abscesses by the drug-resistant bacteria.

We also observed a transient elevation of liver function test results and a skin rash by the antimicrobial treatment in our patient. We diagnosed ceftriaxone-induced liver dysfunction because of the transient elevation of liver function values. We thus changed the antimicrobial drug to ampicillin/sulbactam. We also changed the antimicrobial agent from cefcapene pivoxil hydrochloride hydrate to amoxicillin/clavulanic acid because of the onset of skin rashes. A patient-specific drug administration and an adequate drug change may be necessary on a case-by-case basis.

Oral bacteria are thought to be able to cause a deep-neck infection, and it is thus crucial to conduct an early oral examination and provide appropriate dental treatment to prevent deterioration and recurrence of this

Table 3 The antimicrobial susceptibility of *S. parasanguinis*

| Day | Day 3 | | Day 5 | | Day 9 | Day 13 | | Day 27 | |
|----------|----------------------|-----------------------|----------------------|-----------------------|--------------------------|------------------------------------------|-----------------------------------------|---------------------------------------------|------------------|
| | left upper extremity | right upper extremity | left upper extremity | right upper extremity | maxillary anterior tooth | gingival pocket on right maxillary molar | gingival pocket on left maxillary molar | dental implants on right maxillary premolar | mandibular molar |
| Specimen | blood | blood | blood | blood | dental plaque | gingival crevice fluid | gingival crevice fluid | gingival crevice fluid | dental plaque |
| EM | R | R | R | R | R | R | R | R | R |
| CAM | R | R | R | R | R | R | R | R | I |
| AZM | R | R | R | R | R | R | R | R | I |
| CAZ | I | R | R | R | R | R | I | R | R |
| CPFX | I | I | I | I | R | R | R | R | R |
| CTM | S | S | S | S | R | R | R | R | R |
| FMOX | S | S | S | S | R | R | R | R | I |
| ST | S | S | S | S | R | R | R | R | R |
| PZFX | S | S | S | S | R | R | I | R | R |
| TFLX | S | S | S | S | R | R | I | R | I |
| CEZ | S | S | S | S | I | R | I | R | I |
| PCG | S | S | S | S | I | I | I | I | I |
| PIPC | S | S | S | S | I | I | I | I | I |
| SBT/ABPC | S | S | S | S | I | I | I | I | I |
| SBT/CPZ | S | S | S | S | I | I | I | I | I |
| TAZ/PIPC | S | S | S | S | I | I | I | I | I |
| LVFX | S | S | S | S | I | I | I | I | I |
| AMPC | S | S | S | S | I | I | S | I | S |
| AMPC/CVA | S | S | S | S | I | I | S | I | S |
| CPDX-PR | S | S | S | S | S | S | S | S | I |
| CTRX | S | S | S | S | S | S | S | S | S |
| CFPN-PI | S | S | S | S | S | S | S | S | S |

EM, Erythromycin; CAM, Clarithromycin; AZM, Azithromycin; CAZ, Ceftazidime; CPFX, Ciprofloxacin; CTM, Cefotiam; FMOX, Flomoxef; ST, Sulfamethoxazole-Trimethoprim; PZFX, Pazufloxacin; TFLX, Tosufloxacin; CEZ, Cefazolin; PCG, Penicillin G; PIPC, Piperacillin; SBT/ABPC, Sulbactam/Ampicillin; SBT/CPZ, Sulbactam/Cefoperazone; TAZ/PIPC, Tazobactam/Piperacillin; LVFX, Levofloxacin; AMPC, Amoxicillin; AMPC/CVA, Amoxicillin/Clavulanate; CPDX-PR, Cefpodoxime-Proxeti; CTRX, Ceftriaxone; CFPN-PI, Cefcapene-Pivoxil
S, Susceptible; I, Intermediate; R, Resistant

disease.

In conclusion, bacteremia caused by traumatic tooth fractures induced acute prevertebral abscesses, and *S. parasanguinis* present in the oral cavity caused the abscess formation.

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