Case Report

*Klebsiella pneumoniae* Cervical Necrotizing Fasciitis Secondary to Bacterial Parotitis: A Case Report

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Abstract Cervical necrotizing fasciitis is a fulminant infection that spreads along the fascial planes, causing subcutaneous tissue death characterized by rapid progression and systemic toxicity. Dental infection is the most common nidus of cervical necrotizing fasciitis. *Streptococcus* and *Staphylococcus* species are found to be the most commonly isolated organisms in many bacteriological analyses of cervical necrotizing fasciitis. We describe a case of a 29-year-old female who was diagnosed with acute suppurative parotitis first. After admission, her illness progressed to cervical necrotizing fasciitis. She underwent surgery of incision and drainage, and pus culture yielded *Klebsiella pneumoniae* (*K. pneumoniae*). To the best of our knowledge, cervical necrotizing fasciitis is seldom secondary to bacterial parotitis, and rarely caused by *K. pneumoniae*.

Keywords bacterial parotitis; cervical necrotizing fasciitis; *Klebsiella pneumoniae*; diabetes mellitus

1. Introduction

Necrotizing fasciitis is a fulminant infection of soft and connective tissues. Tracing back to the American Civil War in 1871, this overwhelming infection was first described by Joseph Jones [1]. It is defined as an extensive necrosis of superficial fascia, which causes surrounding tissue involvement and rapid spreading associated with systemic toxicity. The parotid gland is invested in a dense fibrous capsule derived from the superficial layer of the deep cervical fascia, which communicates with deeper layers of cervical fascia by parapharyngeal space. Anatomically, bacterial parotitis has the potential to cause a rapid and diffuse spread of infection throughout multiple fascial planes in head and neck region.

In our review of the literature, neither acute suppurative parotitis (ASP) nor cervical necrotizing fasciitis (CNF) is primarily caused by *Klebsiella pneumonia* (*K. pneumonia*) [2,3]. Besides, parotid gland is not the primary source of CNF [3,4]. Herein, we share our clinical experience of CNF secondary to ASP associated *K. pneumoniae*.

2. Case report

A 29-year-old female visited our emergency department because of progressive enlargement of the right side of her face with severe tenderness for a couple of days. She was in her usual health status without remarkable systemic disease. Her mother has non-insulin-dependent diabetes mellitus. She denied sore throat, toothache, and odynophagia. There was no febrile episode and other toxic signs.

The right side of her lower face and submandibular region were swollen, with maximum swelling near the angle of the mandible. The skin overlying the swollen area was erythematous and blanched with finger pressure. There was no fluctuance or crepitus, nor was expression of the pus from Stensen’s duct. The laboratory findings showed a white blood cell count of $19.8 \times 10^9/L$, neutrophil of 85% and C-reactive protein of 4.45 mg/dL. Electrolyte levels were normal. Contrast-enhanced computed tomography exhibited heterogeneous right parotid gland and peripheral enhancement (Figure 1). Under the impression of ASP, she was admitted to our ward for further treatments.

Figure 1: Computed tomography with contrast exhibited heterogeneous right parotid gland and peripheral enhancement.
cultures yielded amoxicillin/clavulanic acid-resistant K. pneumoniae, which are susceptible to cefoxitin. We changed the antibiotics to cefoxitin in line with the result of culture. Although the symptoms and signs did not subside, we performed the aspiration. We arranged incision and drainage immediately and noted (Figure 2). Ash gray pus was evacuated by needle aspiration. On the hospital day 14, she complained of odynophagia and severe tenderness over right cervical region. Fluquitude of right parotid gland and swelling over right neck were noted (Figure 2). Necrotic fascia of sternocleidomastoid muscle was revealed. According to the surgical finding, we confirmed the diagnosis of cervical necrotizing fasciitis. Over the next week, we performed the surgical finding, we confirmed the diagnosis of cervical necrotizing fasciitis. According to the anterior cervical triangle during surgery. Necrotic fascia to the neck. 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One reliable indicator is mentioned that too much or too little pain is out of proportion to the physical examination [6]. According to a previous study, neither white blood cell count nor other laboratory-based diagnostic tools are useful in identifying CNF [8]. The cervical computed tomography might provide initial clue for diagnosing CNF by identifying some features, such as air-fluid level, diffuse gas bubble, and fascial plane dissection [1,3,6]. Routine thoracic computed tomography is suggested to rule out mediastinitis.

Streptococcus and Staphylococcus species are the most common pathogens isolated from patients with CNF. Nevertheless, many reports have called attention to the presence of anaerobic species [1,3,9]. Polymicrobial infections are not only in nature, but its synergistic interactions lead to more invasive infection. Similarly, the importance of anaerobes and mixed flora is mentioned in early study of ASP [2]. When patient has the underlying disease with diabetes mellitus, K. pneumoniae is found to be the most commonly isolated organism in many bacteriological analyses of deep neck infection in Asia [10]. This predominance could be explained by increased oropharyngeal K. pneumoniae colonization in immunocompromised host. Besides, much more virulent K. pneumoniae strains has been mentioned before in Asia [11]. The invasive K. pneumoniae strains are associated with serotypes K1 and K2 and the regulator of mucoid phenotype A gene (rmpA), which present hypermucoviscous phenotype [12]. The hypermucoviscous phenotype provides resistance to phagocytosis and immune system. Almost all patients with severe infection are exclusively associated with these serotypes. Although highly virulent K. pneumoniae strains are primarily found in Asia, sporadic case was reported in Western hemisphere [13].

Once the diagnosis of CNF is confirmed, broad-spectrum intravenous empiric antibiotics should be initiated.
immediately. Antibiotic coverage can be narrowed after yielding the culture result. As mentioned previously, for diabetic patient, we have to consider the antibiotics to cover the *K. pneumoniae*, especially in Asia. Most of *K. pneumoniae* strains are susceptible to narrow-spectrum cephalosporins, although amoxicillin/clavulanic acid has reduced effectiveness among *K. pneumoniae* strains in early study [14]. Aggressive surgical intervention is mandatory, involving wide incision, adequate exploration of deep neck spaces and debridement of necrotic tissue until healthy bleeding tissue is encountered. Mediastinitis is the common CNF-induced complication. If mediastinal involvement is noted, advanced management has to be performed, such as mediastinostomy, thoracotomy, and thoracic drainage [15]. Rededbriment should be considered if patient still has toxic signs and poor-controlled wound infection.

Some studies advocate hyperbaric oxygen (HBO) therapy in the treatment of CNF. It could increase polymorphonuclear cell function and tissue oxygenation, which is bactericidal to the anaerobic bacteria [6,15]. HBO also enhances the transport of antibiotics across the bacterial cell wall, particularly the aminoglycosides [6]. However, the use of HBO is still controversial, because of no standard regimen and considering the availability in different regions [4,6,15].

Patient is diagnosed of CNF, who requires intensive care unit (ICU) management to reduce morbidity and mortality. The average length of ICU stay needs at least one week for patients without thoracic extension. In the group with thoracic extension, the length of ICU stay is 2–3 times as much longer as patients without thoracic extension [3,15]. In Taiwan, the hospital stay is an important issue, due to our national health insurance. Nougué et al. showed that the occurrence of severe complications of CNF was independently associated with oral glucocorticoid intake before admission and a pharyngeal source of CNF [3]. In our clinical practice of otolaryngology, glucocorticoid is often used to eliminate local edema. We should be more careful to prescribe glucocorticoid to prevent the undesired result.

In conclusion, CNF does not have a definite standardized diagnosis, and it is usually based on history, laboratory finding, radiography, and surgical exploration. In spite of the uncommon nidus of CNF, ASP still has a certain degree of probability in distribution. We should not neglect any kind of infection with potential to develop more fulminant infection in head and neck region.

**Conflict of interest** The authors declare that they have no conflict of interest.

**References**


