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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 pneumonia*; 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.

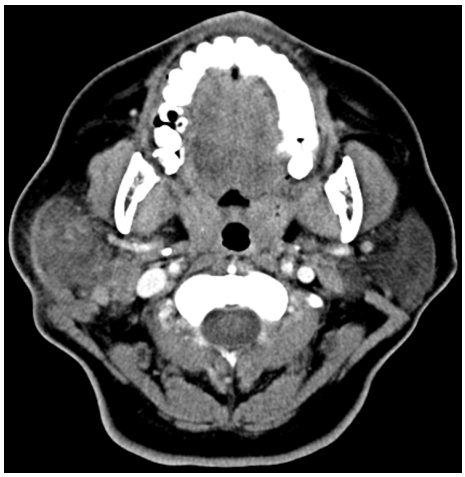

Figure 1: Computed tomography with contrast exhibited heterogeneous right parotid gland and peripheral enhancement.

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 19.8×10^9/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.

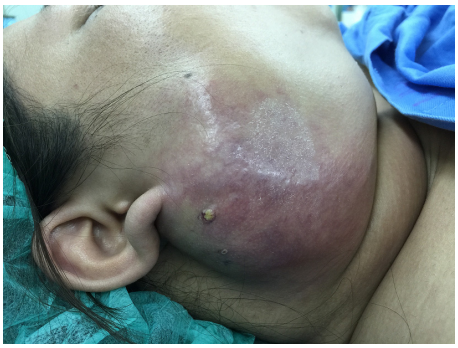

Figure 2: Swelling over right parotid gland and cervical region, and two small apertures were left after needle aspiration.

Initially, parenteral fluid hydration, antimicrobial therapy with amoxicillin/clavulanic acid and medications for symptom relief, such as NSAIDs, were prescribed. Because the symptoms and signs did not subside, we arranged a series of examinations for adjusting diagnosis and therapeutic plan. Hyperglycemia and HbA1c of 8.7% were found. In addition to controlling blood sugar level, we kept antibiotic therapy, and she slowly had symptomatic improvement in consecutive days.

On the hospital day 14, she complained of odynophagia and severe tenderness over right cervical region. Fluctuance of right parotid gland and swelling over right neck were noted (Figure 2). Ash gray pus was evacuated by needle aspiration. We arranged incision and drainage immediately for suspected deep neck infection. We performed the modified Blair incision, and the incision was extended to the neck. Large amount of pus were evacuated from anterior cervical triangle during surgery. 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 debridement and changed wet dressing every day. Pus 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. After the surgical intervention and changing coverage of antibiotics, the patient recovered progressively and discharged without complication.

3. Discussion

Most extracranial infections of head and neck are self-limited, and just a few have life-threatening potential by facial spaces. Lin et al. reviewed the 161 cases of patients with deep neck infection from 2002 to 2012, only 5.6% of patients was caused by sialoadenitis [5]. When we talk about CNF, it is clinically described as extensive necrosis of superficial fascia and an uncommon clinical entity in head and neck region. The common nidi are pharyngeal and dental infection. The gland infection, such as parotitis, is 14% in distribution of sources of CNF [3].

Some factors predispose patients to CNF, including immunocompromise and low nutritional status [1,6]. Diabetes mellitus is the systemic illness most frequently associated with CNF, being reported in 72% of patients by Lin et al. In the review of literature, diabetics display disturbance of cell-mediated immunity, alterations in opsonization and decreased bactericidal function of granulocytes and monocytes [7]. Due to this immunocompromised status, along with vascular insufficiency, diabetic patients are prone to have an invasive infection.

There is not a definite standardized diagnosis of CNF. It is usually based on history, laboratory finding, radiography, and surgical exploration. At the early stage, it is difficult to distinguish CNF from superficial cellulitis. Both present local swelling, erythematous change, and tenderness. Crepitus is a sign of subcutaneous gas, but does not be presented in all cases of CNF. 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]. Redebridement 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.

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