Necrotizing fasciitis after peritonsillar abscess in an immunocompetent patient

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Abstract

Cervical necrotizing fasciitis (CNF) is a rapidly progressive, severe bacterial infection of the fascial planes of the head and neck. Group A beta haemolytic *Streptococcus* spp. (GABHS), *Staphylococcus* spp., or obligatory anaerobic bacteria are the most common causative pathogens. The disease usually results from a dental source or facial trauma. Extensive fascial necrosis and severe systemic toxicity are common manifestations of CNF. Review of the literature reveals only seven such cases, with four successful outcomes.

The authors present the case of a 50-year-old immunocompetent female with CNF arising from a peritonsillar abscess. Intravenous immunoglobulins in conjunction with surgery and antibotics were used successfully.

The authors also suggest the importance of the early diagnosis, aggressive surgical debridement, broadspectrum antibiotics, and possible usefulness of the intravenous immunoglobulins in the treatment of CNF, especially when the disease is associated with toxic shock syndrome.

Key words: Fasciitis, necrotizing; Immunoglobulins, intravenous

Introduction

Necrotizing fasciitis (NF) is a rare soft tissue infection characterized by progressive destruction of fascia and adipose tissue that may not involve the skin (Sellers et al., 1996). Alterations in immune functions, such as diabetes mellitus, burns and malnutrition, are common predisposing factors. Craniocervical necrotizing fasciitis (CNF) initially involves the superficial musculoaponeurotic system (SMAS) and superficial fascial planes of the head and neck, or it may result from a deep soft tissue infection, such as pharyngitis or dental infection that spreads along the deep fascial planes. Causative organisms include Group A beta-haemolytic streptococci (GABHS), Staphylococcus spp., or obligate anaerobic bacteria. These virulent bacteria, alone or in synergistic combination, produce a severe necrotizing infection of the fascia and soft tissues of the head, neck, and scalp.

If a treatment is not initiated early, the infection can rapidly spread to involve the great vessels or mediastinum, producing life-treating systemic toxicity and sepsis (Lalwani and Kaplan, 1991; Shindo *et al.*, 1997). Recent studies report an apparent increase of CNF incidence all over the world (Chelsom *et al.*, 1994; Henrich *et al.*, 1995; Curtis, 1996). The basis of successful treatment consists of early recognition of CNF combined with aggressive surgical debridement and drainage of the involved necrotic fascia and tissue along with intensive patient support and intravenous broad-spectrum antibiotic coverage.

We report an unusual and successfully treated case of CNF originating from a peritonsillar abscess in an otherwise immunocompetent patient.

Case report

A 50-year-old female was admitted to the department with a right peritonsillar abscess lasting six days. Prior to admission she had been treated at home by her general



FIG. 1

A CT-scan of the patient demonstrated diffuse infiltration of the neck fat, and a huge formation surrounded by remarkable oedema, suggesting an abscess.

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practitioner by means of intramuscularly administered penicillin for five days (throat swab was not performed). On admission, the patient was alert and cooperative, her body temperature was 39.2°C, the blood pressure was 14/ 10 kPa, the respiratory rate was 22/min and the pulse was 104/min.

Physical examination revealed a remarkable erythematous, nonfluctuant and noncrepitant oedema of the right side of the neck with extension to the anterior cervical region. An examination of the throat revealed erythema and exudative tonsillitis with the typical clinical picture of peritonsillar abscess.

Computed tomography (CT) of the patient's neck demonstrated diffuse oedema involving the soft tissue of the right tonsillar pillar, right masseter muscle and right submandibular gland. A CT scan also demonstrated diffuse infiltration of the fat tissue of the neck, and a huge formation surrounded by remarkable oedema, suggesting an abscess (Figure 1).

An ultrasound examination of the neck revealed a large heterogenous mass measuring 85×55 millimetres (mm) surrounding the right jugular vein (Figure 2).

Systolic blood pressure after the patient's deterioration was 12 kPa and pulse rate was 130/min.

A diagnosis of CNF was presumed. Intravenous ampicillin/sulbactam and gentamicin along with massive fluid and electrolyte resuscitation were administered immediately. The patient was taken urgently to the operating theatre.

A modified face lift incision was made anterior to the right ear, extending inferiorly up to the cricoid cartilage. Exploration of the right side of the neck revealed a grey, boiled-like and necrotic external layer of deep cervical fascia which was covering the internal pterygoid muscle and the sternocleidomastoid muscle (SCM). The SCM was indurated on section but without the formation of microabscesses. These findings included a necrotic middle layer of deep cervical fascia.

A blunt incision was performed in all directions. Separation of the necrotic fascia from surrounding tissues was easy. There was also necrotic tissue along the right thyroid cartilage, anteromedial to the carotid sheath. Thin 'dishwater' pus and necrotic fascia were encountered and sent for aerobic and anaerobic culture. The necrotic fascia and tissue were debrided. The parapharyngeal, retropharyngeal and carotid sheath spaces were opened, copiously irrigated, and drained. Two Penrose drains were placed into the parapharyngeal space. The tissues at the inferior margin of the dissection, just below the clavicle, appeared healthy, therefore the mediastinum was not explored.

Immediately after surgery, intravenous immunoglobulin G (IVIGG) treatment was started at a dose of 150 mg/kg and was administered daily for five days. After surgery, on the day of the procedure itself, the patient's condition gradually improved. During the afternoon of the next day, her condition stabilized and no further progression of the oedema could be detected. Intra-operatively taken cultures revealed only methicillin – sensitive *Staphylococcus epidermidis*. Imipenem was used instead of ampicillin/ sulbactam and gentamicin.

Antistreptolysin titre testing of blood samples taken on admission, then 10 days and four weeks later revealed antistreptolysin O (ASO) titre 1: 1280, 1: 640, 1:80, respectively. The antideoxyribonuclease B (anti-DNase B) titer was 400.

Her wounds have healed normally and she has resumed normal activities. After 21 days she was discharged from the hospital.



FIG. 2

An ultrasound of the neck did not reveal thrombosis of the right jugular vein.

Discussion

Necrotizing fasciitis (NF) is a relative uncommon but aggressive soft tissue infection. It is associated with extreme systemic toxicity (Reed and Vinod, 1992). The first clear description of necrotizing fasciitis was published during the American Civil War by Joseph Jones (1871). Recent interest has been stimulated by several articles in the lay press about 'flesh-eating bacteria' which have caused several deaths in the United States (Henrich *et al.*, 1995), England (Curtis, 1996), and Norway (Chelsom *et al.*, 1994).

NF can develop in patients of all ages and has no sex or race predilection (Kronish and McLeish, 1991). Minor trauma, such as abrasions, lacerations, insect bites, and hypodermic needle injections, are the most common initiating factors. The onset of infection may also follow surgery or blunt injuries, and occasionally, develop without apparent cause (Kronish and McLeish, 1991; Henrich *et al.*, 1995) as seemed to be the case in our patient. In most of the reported cases the lower extremities were involved, but the upper extremities, trunk and genitalia also are frequently affected (Chelsom *et al.*, 1994; Sellers *et al.*, 1996).

NF of the head and neck, as it was in our patient, is relatively uncommon and may have a poor prognosis. The most common cause of craniocervical necrotizing fasciitis (CNF) is dental infection from the mandibular molars (30 per cent), followed closely by trauma (28 per cent) (Goodnight *et al.*, 1994; Greiwald *et al.*, 1995).

Group A streptococcus and staphylococcus species are the most common aetiologic microorganisms, and are usually found in synergistic combination with other facultative bacteria, such are enterobacteriacea, or anaerobic bacteria (Lalwani and Kaplan, 1991; Miles *et al.*, 1992; Henrich *et al.*, 1995).

Our patient is a rare case where *Streptococcus* spp. could not be isolated from the wound. Perhaps the five-days antibiotic course prior to taking the bacteriological sample might have suppressed the growth of the pure culture (Kronish and McLeish, 1991). Still, we proved the existence of this microorganism by means of serological analysis (ASO titre 1:1280, 1:640, 1:80, respectively). The accuracy of this serological proof was additionally confirmed by means of anti-DNase B.

The pathogenesis of NF has been studied by numerous investigators, but the exact mechanism of this rapidly spreading gangrenous infection has not been established.

The release of enzymes, such as hyaluronidase and proteolytic portions of cell membranes, has been shown to be a contributing factor in the necrosis (Greiwald et al., 1995). The relative lack of vascularity of the relevant fascial planes has also been hypothesized as a factor in NF. An additional factor in extensively necrotizing processes is that the diffusion of antibacterial agents into the infectious foci will be greatly hampered, which emphasizes the need for removal of all necrotic tissue. On the other hand, superantigens, such as SPE-A, SPE-B, and SPE-C, secreted by some strains of GABHS cause millions of clones of T cells to be activated instead of just few of them. The resultant excessive activation of cytokines, complement, and clotting cascades, plus production of oxygenfree radicals and nitric oxide cause the shock and multiorgan failure. Key roles are played by T4 cells and tumour necrosis factors (TNF), both alpha and beta. The depletion of T lymphocytes that carry specific variable regions of beta chains also supports the role of a superantigen phenomenon (Lamothe et al., 1995). The discovery that IVIG can reverse the hyperproliferation of T-cells, neutralize superantigens, and down-regulate the production of TNF prompted us to use IVIGG to treat NF (Yong, 1994).

The key to successful treatment of NF is early diagnosis which, when combined with aggressive treatment, can substantially improve the outcome (Grant, 1994). Extensive excision, debridement, and drainage of the involved necrotic skin, fascia, and muscle are the most important aspects of therapy. This should be done early, not only to control the primary infectious process, but also to remove the necrotic tissue which is highly susceptible to secondary infection. To the best of our knowledge, our case is the first in which a patient with NF of the head and neck received combined treatment with antibiotics, surgery and IVIGG.

References

- Chelsom, J., Halstensen, A., Haga, T., Hoiby, E. A. (1994) Necrotizing fasciitis due to group A streptococci in western Norway: incidence and clinical features. *Lancet* **344**: 1111–1115.
- Curtis, N. (1996) Invasive group A streptococcal infection. Current Opinion in Infectious Diseases 9: 191–202.
- Goodnight, J. W., Sercarz, J. A., Wang, M. B. (1994) Cervical and mediastinal emphysema secondary to third molar extraction. *Head and Neck* 16: 287-290.

- Grant, I. S. (1994) Necrotizing fasciitis (Letter). Lancet 344: 1770–1771.
- Greiwald, J. H., Wilson, J. F., Haggerty, P. G. (1995) Peritonsillar abscess: An unlikely cause of necrotizing fasciitis. Annals of Otology, Rhinology and Laryngology 104: 133–137.
- Henrich, D. E., Smith, T. L., Shockley, W. W. (1995) Fatal craniocervical necrotizing fasciitis in an immunocompetent patient: A case report and literature review. *Head and Neck* 17: 351–357.
- Jones, J. (1871) Investigation upon the nature, causes and treatment of hospital gangrene as it prevailed in the Confederate Armies 1861–1865. In Surgical memories of the War of Rebellion. US Sanitary Commission, New York, pp 146–170.
- Kronish, J. W., McLeish, W. M. (1991) Eyelid necrosis and periorbital necrotizing fasciitis. Ophthalmology 98: 92–98.
- Lalwani, A. K., Kaplan, M. J. (1991) Mediastinal and thoracic complications of necrotizing fasciitis of the head and neck. *Head and Neck* 13: 531–539.
- Lamothe, F. D., Amico, P., Ghosn, P., Tremblay, C., Braidy, J., Patenaude, J. V. (1995) Clinical usefulness of intravenous human immunoglobulins in invasive group A streptococcal infections: case report and review. *Clinical Infectious Diseases* 21: 1496–1470.
- Miles, L. T., Jacobs, J. B., Gittelman, P. D., Lebowitz, A. S. (1992) Streptococcal gangrene of the head and neck: A case report and review of the literature. *Head and Neck* 14: 143–147.
- Reed, J. M., Vinod, K. A. (1992) Odontogenic cervical necrotizing fasciitis with intrathoracic extension. *Otolaryn*gology – *Head and Neck Surgery* 107: 596–600.
- Sellers, B. J., Woods, M. L., Morris, S. E., Saffle, J. R. (1996) Necrotizing group A streptococcal infections associated with streptococcal toxic shock syndrome. *American Journal* of Surgery **172**: 523–528.
- Shindo, M. L., Nalbone, V. P., Dougherty, W. R. (1997) Necrotizing fasciitis of the face. *Laryngoscope* 107: 1071–1079.
- Yong, J. M. (1994) Necrotizing fasciitis (Letter). Lancet 343: 1427.

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