

Necrotizing Fasciitis (Flesh-Eating Disease)

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Editorial

Since the mid-1980s, concern has grown that invasive group A Streptococci (GAS) has been increasing in incidence and severity. Invasive infections caused by group A *Streptococcus* or *Streptococcus pyogenes* include sepsis, arthritis, pneumonia, meningitis, necrotizing fasciitis (NF) and streptococcal toxic shock syndrome (STSS). GAS also causes noninvasive suppurative disease as pharyngitis and otitis media, and nonsuppurative post streptococcal sequelae (acute rheumatic fever and acute glomerulonephritis [1]).

Necrotizing fasciitis (flesh-eating disease) is a soft tissue infection involving the subcutaneous fat and fascia. It was first described in 1952 by Wilson [2]. Facial necrosis precedes muscle and skin involvement, hence its namesake. The overall mortality rate is 25%-30% more than 70% of cases are associated with toxic shock syndrome [3]. The organisms related to NF are group A beta-hemolytic Streptococci (type II NF), with or without staphylococcal co infection [4]. The rarer form (type I NF) caused by polymicrobial infections especially obligate and facultative anaerobes, affects mainly immunocompromised or debilitated patients [5]. Severe pain is the most common clinical presentation, presence of bullae in the skin and gas in the soft tissue on plain X-ray [6]. The risk factors of necrotizing fasciitis are diabetes mellitus, malnutrition, trauma operative procedures, and nonsteroidal anti-inflammatory drugs [7]. In addition, necrotizing fasciitis has also been reported in renal transplant recipient treated with FK506 (Tacrolimus) [8]. Information on bacteriology from wound and blood cultures remains important for fine-tuning the antibiotic selection over empirical treatment [6]. A delay in diagnosis can result in progressive advancement highlighted by widespread infection, multiple-organ involvement, and ultimately, death [9]. Clinical management of invasive GAS infection focuses on accurate diagnosis and timely appropriate use of antimicrobial therapy [10]. Bacterial attachment to host tissue is the first step leading to colonization and subsequent development of invasive disease. Binding to fibronectin promotes adherence to epithelial cells [11]. Invasion by specialized adhesion mechanisms, through a particular adhesion factor might confer a certain site or tissue specificity to group A Streptococcus [12].

The study of extracellular proteins that play a role in the pathogenesis of GAS and known as "streptococcal pyrogenic toxins" using PCR technique showed that they were nonuniformly distributed among the GAS causing different types of disease [13]. Therefore several molecular typing systems have been introduced and reported as alternative tools. Serotyping of GAS based on protein M, a major surface virulence factor, has long been used as the gold standard for the epidemiological surveillance of the infections caused by this pathogen. In recent years it has been widely replaced by similar techniques based on sequencing the hyper variable region of the *emm* gene encoding the M protein. However, recent studies show that *emm* typing alone is not sufficient to unambiguously identify GAS clones and that it must be

complemented with other typing methods such as pulsed-field gel electrophoresis (PFGE) or multilocus sequence typing (MLST) [14]. Obszańska et al. [15] reported two new methods: MLVF (multiple locus variable number tandem repeat fingerprinting) based on the amplification of several loci of variable size and comparison of generated band patterns with a reference and MLVA (multilocus variable tandem repeat analyses) based on the same principles as MLVF, but instead of pattern analysis, number of repeated sequences within each locus is used to generate unique code that can be stored in the database. Both methods are cheap, fast and simple and give results within 10 hours when compared with PFGE. On 2011 Dale et al. [16] constructed a new 30-valent vaccine containing M protein peptides from GAS serotypes prevalent in North America and Europe.

References

1. O'Loughlin RE, Roberson A, Cieslak PR, Lynfield R, Gershman K, et al. (2007) The epidemiology of invasive group A streptococcal infection and potential vaccine implications: United States 2000-2004. *Clin Infect Dis* 45:853-862.
2. Wilson B (1952) Necrotising Fasciitis. *Am Surg* 18: 416-431.
3. Gelaw Y, Abateneh A (2014) Periocular necrotizing fasciitis following retrobulbar injection. *Clin Ophthalmol* 8: 289-292.
4. Lazzeri D, Lazzeri S, Figus M, Tascini C, Bocci G, et al. (2010) Periorbital necrotizing fasciitis. *Br J Ophthalmol* 94: 1577-1585.
5. Hayek S, Ibrahim A, Atiyeh B (2011) The diagnosis and management of necrotizing fasciitis. *Wounds Int* 2: 4.
6. Goh T, Goh LG, Ang CH, Wong CH (2014) Early diagnosis of necrotizing fasciitis. *Br J Surg* 10: e119-125.
7. Sehgal VN, Sehgal N, Sehgal R, Khandpur S, Sharma S (2006) Necrotizing fasciitis. *J Dermatolog Treat* 17: 184-186.
8. Tang S, Kwok TK, Ho PL, Tang WM, Chan TM, et al. (2001) Necrotizing fasciitis in a renal transplant recipient treated with FK 506: The first reported case. *Clin Nephrol* 56: 481-485.
9. Kanuck DM, Zgonis T, Jolly GP (2006) Necrotizing fasciitis in a patient with type 2 diabetes mellitus. *J Am Podiatr Med Assoc* 96: 67-72.
10. Sendur MA, Aksoy S, Ozdemir NY, Zengin N (2014) Necrotizing fasciitis secondary to bevacizumab treatment for metastatic rectal adenocarcinoma. *Indian J Pharmacol* 46: 125-126.
11. Goodfellow AM, Hibble M, Talay SR, Kreikemeyer B, Currie BJ, et al. (2000) Distribution and antigenicity of fibronectin binding proteins (SfbI and SfbII) of *Streptococcus pyogenes* clinical isolates from the northern territory, Australia. *J Clin Microbiol* 38: 389-392.
12. O'Brien KL, Beall B, Barrett NL, Cieslak PR, Reingold A, et al. (2002) Epidemiology of invasive group a streptococcus disease in the United States, 1995-1999. *Clin Infect Dis* 35: 268-276.
13. Vlamincx BJ, Mascini EM, Schellekens J, Schouls LM, Paauw A, et al. (2003) Site-specific manifestations of invasive group a streptococcal disease: type distribution and corresponding patterns of virulence determinants. *J Clin Microbiol* 4: 4941-4949.

14. Carriço JA, Silva-Costa C, Melo-Cristino J, Pinto FR, de Lencastre H, et al. (2006) Illustration of a common framework for relating multiple typing methods by application to macrolide-resistant *Streptococcus pyogenes*. *J Clin Microbiol* 44: 2524-2532.
15. Obszańska K, Borek AL, Hryniewicz W, Sitkiewicz I (2012) Multiple locus VNTR fingerprinting (MLVF) of *Streptococcus pyogenes*. *Virulence* 3: 539-542.
16. Dale JB, Penfound TA, Chiang EY, Walton WJ (2011) New 30-valent M protein-based vaccine evokes cross-opsonic antibodies against non-vaccine serotypes of group A streptococci. *Vaccine* 29: 8175-8178.