Acta Derm Venereol 2015; 95: 614–615

SHORT COMMUNICATION

A Male with Group B Streptococcal Necrotizing Fasciitis at Multiple Sites Secondary to Multifocal Septic Arthritis

Hiroshi Umemura[1], Keita Hiragushi[2], Susumu Sasaki[2], Hiroko Doi[1], Naofumi Shiota[3], Koji Kabutan[4] and Kenji Asagoe[1]

Departments of [1]Dermatology, [2]General Medicine, [3]Orthopedic Surgery, and [4]Anesthesiology, National Hospital Organization Okayama Medical Center, Okayama, Japan. E-mail: UGN11252@nifty.com

Accepted Nov 13, 2014; Epub ahead of print Nov 18, 2014

Necrotizing fasciitis (NF) is a severe soft tissue infection characterized by high mortality rates (1–3). Group A *Streptococcus* is one of the most common causative agents of this condition (1–3). Group B *Streptococcus* (GBS), on the other hand, is a virulent pathogen causing invasive infections, including NF in infants and pregnant women (4). However, several cases of GBS causing NF in non-pregnant adults have also been reported (5–7).

We describe here a case of a male patient with NF at multiple sites and septic arthritis of multiple joints. He survived with aggressive surgical intervention, antimicrobial therapy and intensive care.

CASE REPORT

In August 2013, a 63-year-old man presented to an orthopaedic centre with general malaise, high fever, and pain in several large joints. He had no history of illness or medication use other than that for hypertension. His white blood cell count (WBC) was 15,360 cells/μl (normal 3,500–8,500) and his C-reactive protein (CRP) level was 15.93 mg/dl (<0.30) when he was admitted to hospital. On day 3, his left knee was swollen, and the culture from synovial fluid yielded *S. agalactiae* (group B). No other microorganism was detected. On day 5, his CRP level had increased to 26.97 mg/dl, and his WBC was 11,770 cells/μl. According to the results of antibiotic sensitivity testing, he was treated with cefepime and piperacillin. On day 8, his CRP level had decreased to 13.46 mg/dl and his WBC had increased to 12,460 cells/μl. However, he was still febrile and swelling had spread to his left shoulder, left wrist and right ankle.

On day 10, he was referred to our hospital. Physical examination revealed purpura, bulla formation and significant necrosis accompanied by erythematous swelling on the skin overlying the medial surface of the left arm, lateral surface of the right forearm, right ankle and right great toe (Fig. 1). In addition, his left shoulder, left hip and left knee were swollen. Contrast-enhanced computed tomography (CT) detected low-density areas in these joints, indicating abscesses (Fig. S1[1]). No gas shadows in the joints or soft tissue were observed on CT. He was diagnosed as having NF at multiple sites, which was considered to be secondary to septic arthritis in multiple joints. His vital signs were: body temperature 37.7°C, pulse rate 98 beats/min, and blood pressure 114/70. Initial blood examination showed the following findings: WBC 16,000 cells/μl, CRP level 13.13 mg/dl, procalcitonin level 4.21 ng/ml (normal <0.05), and immunoglobulin G (IgG) level 1,568 mg/dl (870–1,700). A screening test for human immunodeficiency virus was negative. Blood gas analysis was as follows: pH 7.52, partial pressure of carbon dioxide ($PaCO_2$) 31.9 mmHg, partial pressure of oxygen (PaO_2) 55.3 mmHg, HCO_3^- 26.1 mEq/l, base excess 2.7 mEq/l, and arterial oxygen saturation (SaO_2) 92.5%.

On the day of admission to our hospital, we performed emergency debridement of the affected tissues. Necrotic tissues could be removed at the deep fascia level (Fig. S2[1]). After the operation, he was admitted to the intensive care unit. Antibiotic therapy was immediately started with meropenem and vancomycin. Blood culture and culture of excised tissue yielded *S. agalactiae*, which exhibited no resistance to antibiotics. No other microorganism was detected. The antibiotics were changed to ampicillin, clindamycin and vancomycin. He also developed acute renal failure and was treated with continuous hemodiafiltration or intermittent haemodialysis. On day 3, transthoracic echocardiography detected no vegetation, and there was no evidence of infectious endocarditis. On day 9, arthrotomies and drainage of the left shoulder, left hip and left knee were performed. All 3 joints discharged pus and *S. agalactiae* was solely detected from pus culture. The inflammation in these joints gradually ameliorated. The skin wounds of the extremities granulated well with daily irrigation followed by negative-pressure wound therapy. With improvement in the patient's general condition, we covered the skin wounds with split-thickness skin grafts on day 50 of hospitalization. These grafts were successful and the ulcers epithelialized one month after grafting. On day 59, his immunological screening was as follows: IgG level 1,299 mg/dl; total haemolytic complement activity (CH50) 52.3 U/ml (normal 25.0–48.0), and complement C3 level 127 mg/dl (50–130), complement C4 level 40 mg/dl (10–50). Approximately 4 months after the initial operation, the patient was discharged from our hospital.

[1]https://doi.org/10.2340/00015555-2015

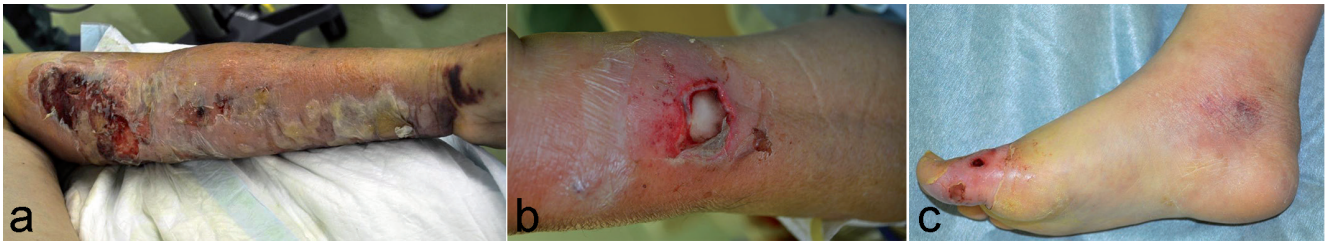

Fig. 1. Skin findings during initial physical examination and after surgical debridement. (a) Left arm, (b) right forearm, and (c) right ankle and great toe.

DISCUSSION

NF has been categorized into 2 types (8). Our case belongs to type II NF, based on monomicrobial infection and the lack of underlying disease or antecedent surgery. Compared with the previous cases of GBS-induced NF, our patient showed a different presentation with involvement at multiple sites (5–7). Despite severe cutaneous manifestations, his vital signs were relatively stable when he was transferred to our hospital and the patient slowly, but extensively, deterioration. Prior antibiotic therapy and delay of surgical intervention in the former clinic may have affected this presentation. In addition, virulent factors, including GBS serotype and host genetic factors, possibly affected the severity of NF (8).

GBS is isolated from approximately 3% of septic arthritis cases (9). To our knowledge, GBS has been attributed to 2 previous cases of NF accompanying septic arthritis (5, 10). Tang et al. (5) described a case of a 75-year-old woman who showed NF on her left lower limb and septic arthritis in her right knee. Similar to our patient, she had no underlying disease other than hypertension. Despite an above-knee amputation of the left lower limb, she died 48 h after operation. Yu et al. (10) reported a similar case in a 30-year-old man with no underlying disease who survived after amputation. It is of note that none of these 3 cases, including ours, were in an immunosuppressive state; however, they had critical infections with GBS and needed drastic surgical intervention.

Prior research has revealed that β-haemolysin/cytolysin and the exopolysaccharide capsule play important roles in GBS infection. Beta-haemolysin/cytolysin is a pore-forming toxin and has the ability to damage lung epithelial cells, endothelial cells and cardiomyocytes (11–13). The cytolytic effects of this toxin may have the potential to induce soft tissue damage. In addition, this toxin has been correlated with the severity of GBS-induced septic arthritis (14). The exopolysaccharide capsule inhibits complement deposition, activates the bacterial surface, and reduces opsonophagocytic clearance (15). Sendi et al. (16) differentiated GBS from an NF patient into 2 types according to expression levels of the capsule. They hypothesized that a low capsule expression facilitates toxin-mediated direct tissue injury and proinflammatory effects, whereas a high capsule expression facilitates resistance to phagocytic clearance. Such virulence factors may have contributed to the severity and the involvement of multiple sites in our case.

The authors declare no conflicts of interest.

REFERENCES

1. McHenry CR, Piotrowski JJ, Petrinic S, Malangoni MA. Determinants of mortality for necrotizing soft-tissue infections. Ann Surg 1995; 221: 558–565.
2. Anaya DA, McMahon K, Nathens AB, Suillivan SR, Foy H, Bulger E. Predictors of mortality and limb loss in necrotizing soft tissue infections. Arch Surg 2005; 140: 151–158.
3. Kihiczak GG, Schwartz RA, Kapila R. Necrotizing fasciitis: a deadly infection. J Eur Acad Derm Venereol 2006; 20: 365–369.
4. Doare KL, Heath PT. An overview of global GBS epidemiology. Vaccine 2013; 315: D7–D12.
5. Tang WM, Ho PL, Yau WP, Wong WK, Yip DKH. Report of 2 fetal cases of adult necrotizing fasciitis and toxic shock syndrome caused by streptococcus agalactiae. Clin Infect Dis 2000; 31: e15–e17.
6. Ogawa D, Shikata K, Wada J, Matsuda M, Makino H. Successful treatment of necrotizing fasciitis associated with diabetic nephropathy. Diab Res Clin Pract 2003; 60: 213–216.
7. Wong CH, Kurup A, Tan KC. Group B streptococcus necrotizing fasciitis: an emerging disease? Eur J Microbiol Infect Dis 2004; 23: 573–575.
8. Sendi P, Johannson L, Norrby-Teglund A. Invasive group B streptococcal disease in no-pregnant adults. A review with emphasis on skin and soft-tissue infections. Infection 2008; 36: 100–111.
9. Ross JJ, Saltzman CL, Carling P, Shapiro DS. Pneumococcal septic arthritis: review of 190 cases. Clin Infect Dis 2003; 36: 319–327.
10. Yu KH, Luo SF, Liou LB, Wu YJJ, Tsai WP, Chen JY, et al. Concomitant septic and gouty arthritis – an analysis of 30 cases. Rheumatology 2003; 42: 1062–1066.
11. Nizet V, Gibson RL, Chi EY, Farmson PE, Hulse M, Rubens CE. Group B streptococcal beta-hemolysin expression is associated with injury of lung epithelial cells. Infect Immun 1996; 64: 3818–3826.
12. Gibson RL, Nizet V, Rubens CE. Group b streptococcal β-hemolysin promotes injury of lung microvascular endothelial cells. Pediatr Res 1999; 45: 626–634.
13. Hensler ME, Miyamoto S, Nizet V. Group B streptococcal β-hemolysin/cytolysin directly impairs cardiomyocyte viability and function. PLoS One 2008; 3: e2446–e2452.
14. Puliti M, Nizet V, Hunolstein C, Bistoni F, Mosci P, Orefici G, et al. Severity of group B streptococcal arthritis is correlated with β-hemolysin expression. J Infect Dis 2000; 182: 823–832.
15. Marques MB, Kasper DL, Pangburn MK, Wessels MR. Prevention of C3 deposition by capsular polysaccharide is a virulence mechanism of type III group B streptococci. Infect Immun 1992; 60: 3986–3993.
16. Sendi P, Johansson L, Dahesh S, Van Sorge NM, Darenberg J, Norgern M, et al. Bacterial phenotype variants in group B streptococcal toxic shock syndrome. Emerg Infect Dis 2009; 15: 223–232.