Journal of Medical Microbiology (2008), **57**, 246–248 DOI 10.1099/jmm.0.47453-0

Case Report

Necrotizing fasciitis with ruminococcus

Murat Livaoğlu,[1] Gürdal Yilmaz,[2] Servet Kerimoğlu,[3] Kemalettin Aydin[2] and Naci Karacal[1]

[1]Department of Plastic and Reconstructive Surgery, Karadeniz Technical University School of Medicine, Trabzon 61080, Turkey

[2]Department of Infectious Disease and Clinical Microbiology, Karadeniz Technical University School of Medicine, Trabzon 61080, Turkey

[3]Department of Orthopedics, Karadeniz Technical University School of Medicine, Trabzon 61080, Turkey

Correspondence
Murat Livaoğlu
mliva@iname.com

Received 15 June 2007
Accepted 24 October 2007

Necrotizing fasciitis is a life- and limb-threatening soft tissue infection. Due to its underlying predisposition and rapid progression, treatment should be started quickly using antibiotherapy and surgical intervention. Although necrotizing fasciitis is mainly caused by streptococci and staphylococci, it may also be polymicrobial. Other peptostreptococci have been reported as necrotizing fasciitis agents in the literature, though we encountered no cases of necrotizing fasciitis caused by *Ruminococcus productus*. Here, we describe a case of necrotizing fasciitis caused by *R. productus*, a Gram-positive, obligatory anaerobe.

Introduction

Necrotizing fasciitis is caused by aerobic and anaerobic micro-organisms, and is a life-threatening infection arising mainly with staphylococci and streptococci (Wong *et al.*, 2003). In addition to regional damage in the soft tissues, it may also cause systemic damage and insufficiency in many vital organs. The presence of underlying predisposing factors further increases the scale of this damage. It frequently occurs in the lower extremities, anterior abdominal wall, perineum and surgical wounds, generally following trauma. Skin colour changes, vesicles and significant cutaneous gangrenes arise within a few days (Wong *et al.*, 2003; Hasham *et al.*, 2005). *Ruminococcus productus*, formerly known as *Peptostreptococcus productus*, is an obligatory anaerobic Gram-positive coccoid bacterium (Ezaki *et al.*, 1994). This bacterium is rarely observed as a clinical infection agent, and may colonize the upper respiratory tract, gastrointestinal tract, vagina and skin in humans and animals (Sucu *et al.*, 2006). The cases reported here are patients who suffered from necrotizing fasciitis caused by *R. productus*, an occurrence that to the best of our knowledge has not been reported in the literature before.

Case 1

A 60-year-old male patient presented to our hospital with complaints of fever for the previous 10 days, and inflammation and swelling in the left groin and anterolateral pelvic region. The patient raised livestock and farmed, and his job involved frequent contamination with animal faecal material. He had a 20 year history of diabetes mellitus and using oral antidiabetic drugs. He did not have any trauma history. At physical examination, his general condition was poor, he was conscious, his temperature was 38 °C, his pulse 98 beats min^{-1} and his blood pressure was 110/70 mmHg. Inflammation, swelling and localized skin necroses were present in the anterolateral region of the thigh, the groin and the trochanteric region.

At laboratory examination his white blood cell count was 19 000 cells μl^{-1}, he had 10.2 g haemoglobin dl^{-1}, 137 mg glucose dl^{-1}, 5.9 g total protein dl^{-1} and 0.9 g albumin dl^{-1}. Other biochemical parameters were within normal limits. No findings of osteomyelitis were determined in pelvic and thigh images. After blood cultures (BACTEC 9200; Becton Dickinson) and aerobic and anaerobic cultures had been taken from the wound, the patient was prepared for surgery and the entire pelvic anterolateral trochanteric area was debrided.

Upon microscopic examination of the debridement material, Gram-positive cocci chains and abundant polymorphonuclear leukocytes were observed. The material was cultured in aerobic and anaerobic jars using the BACTEC system (Becton Dickinson). Treatment with 200 mg ciprofloxacin and 600 mg clindamycin was initiated, twice a day and four times a day, respectively.

Enterobacter cloacae and meticillin-resistant coagulase-negative staphylococci growth was found in aerobic cultures taken before debridement, and *R. productus* growth was found in anaerobic culture of the debridement material. Identification was performed using a Sceptor panel (Becton Dickinson). There was no growth in the aerobic culture. Treatment was changed to 500 mg

 47453 © 2008 SGM *Printed in Great Britain*

imipenem four times a day + 1000 mg amikacin once a day + 400 mg teicoplanin once a day. The defective area in the patient, monitored on a daily basis in the postoperative period with wound care and using antibiotherapy, was repaired with a split thickness skin graft taken from the contralateral thigh. The patient was discharged without complication on day 65 after admission.

Case 2

A 63-year-old male patient had inflammation and swelling in the left leg and foot for the previous 10 days. He had a history of a minimal thermal injury from a stove 2 weeks before. This had worsened significantly over the preceding few days, inflammation had advanced and a vesicle with a yellowish appearance had also developed. The patient had a 20 year history of diabetes mellitus and a 10 year history of congestive heart failure. He was in the stock-breeding line of work. His general condition at physical examination was good. Vesicles containing purulent material were present in the oedematous left leg and foot. His temperature was 37 °C, he had 363 mg glucose dl^{-1}, a white blood cell count of 22 500 cells μl^{-1} and 11.8 g haemoglobin dl^{-1}. Other laboratory findings were within normal limits. Upon microscopic examination of material taken from the wound, Gram-positive coccus chains and clusters and abundant polymorphonuclear leukocytes were observed. Aerobic and anaerobic cultures were taken, after which 200 mg ciprofloxacin and 600 mg clindamycin was started, twice a day and four times a day, respectively. Once the appropriate conditions had been established, debridement was performed and the patient followed-up with wound care. Growth in the cultures taken was identified using an automated Sceptor panel (Becton Dickinson). Meticillin-resistant coagulase-negative staphylococci grew in aerobic culture and *R. productus* in anaerobic culture of the debridement material. The treatment was changed to 500 mg imipenem four times a day + 1000 mg amikacin once a day + 400 mg teicoplanin once a day. When the wound floor became suitable 3 weeks later, a repair was performed using a split thickness skin graft. The patient was discharged without complication 42 days after admission.

Discussion

Necrotizing fasciitis has been known for many years, but the term was first employed by Wilson in 1952. Wilson described necrotizing fasciitis as a soft tissue infection spreading along the fascias (Wilson, 1952). Necrotizing fasciitis is a rarely seen but severe infection characterized by rapid progression along the subcutaneous tissues. It has a high mortality rate, ranging between 6 and 76 % (Hasham *et al.*, 2005). Immunosuppression, diabetes, chronic disease, malnutrition, intravenous drug use, alcohol abuse, peripheral vascular disease, renal failure, blunt or penetrating trauma, surgery, burns, muscle injuries and hypertension may be observed as predisposing factors (Hasham *et al.*, 2005; Rieger *et al.*, 2007). Patients' initial symptoms may be swelling, inflammation, skin colour changes and systemic findings, leading to a picture of severe infection (Hasham *et al.*, 2005). Necrotizing fasciitis frequently occurs as a polymicrobial infection involving aerobic and anaerobic bacteria, though a single agent has been determined in 15 % of cases. Polymicrobial agents were determined in both cases presented in this report. Of these, *R. productus* may colonize the upper respiratory tract, gastrointestinal tract, vagina and skin in humans and animals (Sucu *et al.*, 2006). Despite occurring in the intestinal flora, this bacterium seldom appears as an infective agent. The presence of type II diabetes in both our patients and the fact that both raised livestock may be regarded as predisposing factors.

There are few reports in the literature of infections arising through this bacterium; it is rarely isolated in clinical specimens (Sucu *et al.*, 2006; Nakatani *et al.*, 1998; Botha *et al.*, 1993; Sklavounos *et al.*, 1986). Sucu *et al.* (2006) from our hospital reported that they had isolated *R. productus* in cases of liver abscess and infective endocarditis. Nakatani *et al.* (1998) isolated *P. productus* as an agent in epidural abscess, and Botha *et al.* (1993) and Sklavounos *et al.* (1986) in orofacial abscess cases. Bezirtzoglou & Romond (1991) showed that *P. productus* may colonize in the first day of life in ocular conjunctivitis in neonates delivered by Caesarean section.

In conclusion, although ruminococci do not often appear as infective agents, they may appear in the presence of predisposing factors. In such cases developing necrotizing fasciitis, deep tissue cultures should be taken promptly and rarely observed infective agents borne in mind. To the best of our knowledge, at the time of submission of this report, there were no cases in the literature in which *R. productus* was considered as an agent in necrotizing fasciitis.

References

Bezirtzoglou, E. & Romond, C. (1991). Nosocomial infections of ocular conjunctiva in newborns delivered by Cesarian section. *Ophthalmic Res* **23**, 79–83.

Botha, S. J., Senekal, R., Steyn, P. L. & Coetzee, W. J. (1993). Anaerobic bacteria in orofacial abscesses. *J Dent Assoc S Afr* **48**, 445–449.

Ezaki, T., Li, N., Hashimoto, Y., Miura, H. & Yamamoto, H. (1994). 16S ribosomal DNA sequences of anaerobic cocci and proposal of *Ruminococcus hansenii* comb. nov. and *Ruminococcus productus* comb. nov. *Int J Syst Bacteriol* **44**, 130–136.

Hasham, S., Matteucci, P., Stanley, P. R. & Hart, N. B. (2005). Necrotising fasciitis. *BMJ* **330**, 830–833.

Nakatani, S., Hoshi, K., Yuasa, T., Sato, T. & Tauchi, T. (1998). A case report of epidural abscess due to anaerobic bacteria, producing a mass of gas. *Rinsho Shinkeigaku* **38**, 224–227 (in Japanese).

Rieger, U. M., Gugger, C. Y., Farhadi, J., Heider, I., Andresen, R., Pierer, G. & Scheufler, O. (2007). Prognostic factors in necrotizing fasciitis and myositis: analysis of 16 consecutive cases at a single institution in Switzerland. *Ann Plast Surg* **58**, 523–530.

Sklavounos, A., Legakis, N. J., Ioannidou, H. & Patrikiou, A. (1986). Anaerobic bacteria in dentoalveolar abscesses. *Int J Oral Maxillofac Surg* **15**, 288–291.

Sucu, N., Koksal, I., Yilmaz, G., Aydin, K., Caylan, R. & Aktoz-Boz, G. (2006). Liver abscess and infective endocarditis cases caused by *Ruminococcus productus*. *Mikrobiyol Bul* **40**, 389–395.

Wilson, B. (1952). Necrotising fasciitis. *Am Surg* **18**, 416–431.

Wong, C. H., Chang, H. C., Pasupathy, S., Khin, L. W., Tan, J. L. & Low, C. O. (2003). Necrotizing fasciitis: clinical presentation, microbiology, and determinants of mortality. *J Bone Joint Surg Am* **85**, 1454–1460.