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Fulminant *Staphylococcus lugdunensis* Septicaemia Following a Pelvic Varicella-zoster Virus Infection in an Immune-deficient Patient: A Case Report

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Abstract
Introduction: The deadly threat of systemic infections with coagulase negative *Staphylococcus lugdunensis* despite an appropriate antibiotic therapy has only recently been recognized. The predominant infectious focus observed so far is left-sided native heart valve endocarditis, but bone and soft tissue infections, septicaemia and vascular catheter-related bloodstream infections have also been reported. We present a patient with a fatal *Staphylococcus lugdunensis* septicaemia following zoster bacterial superinfection of the pelvic region.
Case presentation: A 71-year old male diagnosed with IgG kappa plasmocytoma presented with a conspicuous weight loss, a hypercalcaemic crisis and acute renal failure. After initiation of haemodialysis treatment his condition improved rapidly. However, he developed a varicella-zoster virus infection of the twelfth thoracic dermatome requiring intravenous acyclovir treatment. Four days later the patient presented with a fulminant septicaemia. Despite an early intravenous antibiotic therapy with ciprofloxacin, piperacillin/combactam and vancomycin the patient died within 48 hours, shortly before the infective isolate was identified as *Staphylococcus lugdunensis* by polymerase chain reaction.
Conclusion: Despite *S. lugdunensis* belonging to the family of coagulase-negative staphylococci with an usually low virulence, infections with *S. lugdunensis* may be associated with an aggressive course and high mortality. This is the first report on a *Staphylococcus lugdunensis* septicaemia following a zoster bacterial superinfection of the pelvic region.

Introduction

Staphylococcus lugdunensis was first described in 1988 [1]. It belongs to the family of coagulase-negative staphylococci (CoNS), which are skin commensals and historically regarded as pathogens of low virulence. Despite their resistance to a large number of antibiotics, infections with CoNS clinically manifest as less severe, subacute diseases with a low mortality rate [2]. In contrast to other CoNS, *S. lugdunensis* infections exhibit a particular aggressive course associated with a high mortality. Thus *S. lugdunensis*, although a CoNS, should be regarded as equivalent to *S. aureus* when considering the pathogenic potential. [3]. *S. lugdunensis* is a member of the normal skin flora and colonizes predominantly the perineal region, especially the inguinal folds [4-6]. Infectious isolates are mostly derived from skin- and less frequently from vascular-related infections. This pathogen occurs in patients with underlying diseases, especially immunosuppressed patients [7-11], but has also been described in otherwise healthy people [5, 12, 13]. Due to the susceptibility of *S. lugdunensis* to a large number of antimicrobial agents, these infections are often underestimated in their severity by physicans – explaining why *S. lugdunensis* is also called 'a wolf in sheep's clothing' [13].

Here, we report a case of fulminant and fatal septicaemia with *S. lugdunensis* following a pelvic varicella-zoster virus infection in an immunodeficient patient. Despite early and appropriate antibiotic treatment, the patient died within 48 hours.

Case Presentation

A 71 year-old male was diagnosed with IgG kappa plasmocytoma 15 months earlier, exhibiting an extensive bone marrow infiltration of more than 60%. A renal involvement was suspected by a proteinuria of 1.8 g/die, including a Bence-Jones proteinuria, and a chronic renal insufficiency stage 3 without further evaluation of the underlying renal disease. After implantation of a central port-catheter system he received a chemotherapeutic regime consisting of vincristine, doxorubicin and dexamethasone for the next four months. His excellent physical constitution allowed the subsequent autologous bone marrow transplantation eight months later. As renal insufficiency progressed, an autologous arteriovenous fistula was created pre-emptively. Six months later he presented with a conspicuous weight loss, a hypercalcaemic crisis and acute renal failure. On clinical examination the patient was afebrile. The cardiac auscultation revealed a pansystolic murmur, first described three years before, suggesting a mitral valve insufficiency. His initial labo-

ratory panel showed a pronounced hypercalcaemia of 3.8 mmol/l (reference range: 2.0 - 2.6 mmol/l), a serum phosphate level of 16 mg/dl (reference range: 2.6 - 4.5 mg/dl), a serum creatinine of 7.9 mg/dl (reference range: 0.5 - 1.1 mg/dl) and a C-reactive protein (CRP) of 6.1 mg/dl (reference range: <0.5 mg/dl). Haemodialysis treatment was initiated, followed by ibandronate administration. Serum calcium levels rapidly decreased and the overall condition of the patient improved. However, he developed a varicella-zoster virus infection of the left twelfth thoracic dermatome (Th12) requiring intravenous treatment with acyclovir. The diagnosis was based on the typical clinical findings, including limitation of the skin lesions to the left dermatome Th12, and was confirmed by the dermatology department. The zoster lesions were superficial with small vesicles and bullae, but without haemorrhagic lesions, crusts or any signs of bacterial superinfection. Four days later the patient developed a temperature of 39.5 °C and chills. The laboratory findings at this time included a normal leukocyte count, a CRP level of 13.2 mg/dl, which further increased on the following day and persisted over 20 mg/dl. Urine examination, an abdominal ultrasound examination, a computed tomography of chest and cranium as well as a three phase bone scan could not reveal the infectious focus. On clinical examination there was no change of the known cardiac murmur and no signs of septic embolization or skin lesions other than his zoster lesions. The latter by now included haemorrhagic vesicle extending to the dermis, disseminated pustules as well as lesions covered by crusts, but no signs of an erysipel or abscess. The central port and the haemodialysis fistula showed no clinical signs of infection. Four sets of aerobic and anaerobic blood cultures were taken from each the peripheral veins as well as from the central port, which was used for the first time since approximately 6 months to draw the blood cultures. An intravenous antibiotic therapy was started with piperacillin/combactam (3 x 2.5 g/day), ciprofloxacin (2 x 250 mg/day) and vancomycin (initial dose 1 g). All blood cultures showed rapid bacterial growth within few hours. There was no apparent difference in time to positivity between peripheral vein and central port drawn blood cultures. Microscopy revealed a gram-positive, but coagulase-negative staphylococcus. Despite early antibiotic treatment, the patient's condition rapidly deteriorated, impeding the realisation of a transoesophageal echocardiogram to exclude endocarditis. The patient died 48 hours after onset of clinical symptoms. Unfortunately, an autopsy was strictly refused by the patient's relatives. The antibiotic susceptibility pattern arrived on the day of death, 48 hours after the beginning of antibiotic treatment. Except for a resistance towards penicillin, the isolated microorganism was sensitive to all antimicrobial agents tested (isoxazolyl penicillin, piperacillin/tacobactam, cefazolin, cefuroxim, imipenem, erythromycin, clindamycin, gentamycin, cotrimoxazol, doxycyclin, ciprofloxacin, levofloxacin, fosfomycin, fusidic acid, linezolid, vancomycin, rifampicin) including all three antibiotics primarily administered. At the same time, the isolated CoNS was identified as *S. lugdunensis* by polymerase chain reaction.

Discussion and Review of the Literature

Staphylococcus lugdunensis belongs to the family of CoNS and is frequently found as skin commensal of the perineal region and inguinal folds [4-6]. Yet it cannot be considered a typical member of the CoNS family as it increasingly has become known for nosocomial and community-acquired infections with a rather aggressive course and a high mortality rate, mirroring the virulent coagulase-positive *Staphylococcus aureus*. Most frequently, *S. lugdunensis* isolates were detected with left-sided native valve endocarditis, less frequent prosthetic valve endocarditis, pacemaker-related endocarditis or myocarditis [10, 14-19]. In addition, skin and soft tissue infections have been described [5], rarely blood stream infections including sepsis or septic shock [20], bone and joint infections [8, 21, 22], central nervous system infections [23], peritonitis [24, 25], urinary tract [26] or ocular infections [27]. Despite its apparent virulence, only case reports or small patient studies have been published since 1989. Only recently publications on fatal infections emerge and highlight the pathological potential of this atypical CoNS [3]. The low incidence in the past might be explained by the fact that most microbiology laboratories do not routinely identify CoNS isolates to the species level. Yet case of severe sepsis caused by an CoNS with an unusually sensitive antibiotic susceptibility pattern the suspicion for *S. lugdunensis* should be raised.

S. lugdunensis is a gram-positive, catalase-positive coccus, occurring singly, in pairs or short chains with varying colony morphology and pigmentation [1]. Despite the lack of secreted coagulase, some *S. lugdunensis* strains express a membrane-bound coagulase, leading to a positive clumping test, which usually indicates *S. aureus* [28]. In addition, a fibrinogen binding surface protein (Fbl) with considerable similarity to clumping factor A (ClfA) of *S. aureus* is expressed, which promotes bacterial adhesion to immobilized fibrinogen in animal models of endocarditis [29]. Some isolates demonstrate hemolysis on blood agar when streaked in proximity with beta-hemolytic staphylococcal strains [30, 31]. Other identified virulence factors of *S. lugdunensis* include the production of an esterase and a lipase as well as the ability to bind extracellular matrix proteins, such as collagen type I and IV, thrombospondin, plasminogen, fibronectin, vitronectin, laminin and human IgG [32, 33]. *S. lugdunensis* produces tannase, which degrades bacteriostatic and toxic tannins secreted by many microorganisms of the gastrointestinal tract [34]. The accessory gene regulator (agr) system, which has been identified in different staphylococcus strains as regulators of virulence factors, is also detectable in *S. lugdunensis*, but its impact on the reported virulence still needs to be elucidated [35]. Thus, the CoNS-associated ability of *S. lugdunensis* to colonize and form biofilms on the surfaces of medical devices combined with virulence factors, which are more reminiscent of *S. aureus*, highlight the pathogenic potential of this organism.

S. ludgunensis can be identified by conventional biological methods such as pyrrolidonyl arylamidase (PYR) activity, the presence of ornithine decarboxylase and a correct interpretation of the oxcillin mini-

mal inhibitory concentration (MIC) test [28]. The introduction of a characteristic *Eikenella corrodens*-like odor on Colombia sheep blood agar combined with colony pleomorphism and prominent ß-hemolysis after 2 days of incubation, confirmed by API-ID-32 Staph to further analyze CoNS, led to an 11-fold increase in detection of *S. lugdunensis* isolates in skin and soft tissue infections in a Danish community [36]. These traditional microbiologic tests are effective, but timely for an accurate identification of this aggressive CoNS. In case of a fulminant septic course the diagnosis comes too late to influence outcome. Alternatively *S. lugdunensis* can be identified by molecular methods such as an analysis of ribosomal ribonucleic acid (rRNA) restriction fragment length polymorphism [28], real-time polymerase chain reaction (PCR) assays which amplify the 16S rRNA sequence [37] or blot hybridization of a 600-base-pair sequence of the heat shock protein gene (hsp60) [38]. In recent publications conventional [39] as well as quantitative multiplex real-time PCR assays [40] identify *S. lugdunensis* by amplification of the fbl gene, which encodes a fibrinogen-binding protein, or the *tanA* gene [41] for tannase.

S. lugdunensis isolates exhibit susceptibility towards a large number of antibiotics in MIC tests, often including penicillin, macrolides, cephalosporins, β-lactams, aminoglycosides, chinolons, rifampicin, fosfomycin, glycopeptides, sulphonamides, chloramphenicol and others [42, 43]. Only few isolates with β-lactamase activity or oxacillin-resistance [3] have been described. Nevertheless, our patient died despite an early and appropriate antibiotic treatment with vancomycin, piperacillin/combactam and ciprofloxacin. How can such a virulence be explained despite a high susceptibility to most antibiotics? With regard to vancomycin, Frank et al. described a vancomycin tolerance of 93 % of clinical *S. lugdunensis* isolates despite susceptibility in the in vitro testing, as defined by a minimal bactericidal concentration (MBC)/MIC ratio of ≥32 [42]. This tolerance to vancomycin and teicoplanin was verified by time-kill curve methodology [43]. In contrast to glycopeptides, linezolid was successfully employed in the recent past [11, 42]. Thus, if a *S. lugdunensis* infection is suspected, a combined antimicrobial therapy should be considered which includes linezolid. It is absolutely critical to identify *S. lugdunensis* as fast as possible by applying the new PCR-based methods described above in contrast to much more time-requiring microbiological tests.

In our patient the source of the *S. lugdunensis* infection can only be a matter of speculation, as a postmortem autopsy was denied. An endocarditis without any embolic signs or alteration of the heart murmur seems unlikely. Furthermore, the rapid course, mirroring a septic shock, argues against an endocarditis. *S. lugdunensis* has been reported to be responsible for vascular catheter-related bloodstream infections (CRBSI) [20]. Yet in our patient the central catheter initially did not raise any suspicion of infection and was used for the first time in six months to draw the blood cultures. In addition, both diagnostic criteria to confirm a CRBSI diagnosis, as defined by the Infectious Diseases Society of America [45], are not fulfilled: the ratio of positive blood cultures drawn through the port and through a peripheral vein was 1:1 and not as suggested for CRBSI 5:1 or above. The second criterion of differential time to positivity, which means blood cultures drawn from a central venous catheter to become positive two hours earlier than simultaneously drawn blood cultures from a peripheral vein, was equally not met, arguing against a CRBSI in our case. There are several factors indicating that the herpetic lesions are the most probable site of bacterial entrance. *S. lugdunensis* is capable and known to cause soft tissue infections and abscesses [5]. The zoster infection preceded the fatal septicaemia by 4-6 days. The herpetic lesions localized at the pelvic girdle region, known to host *S. lugdunensis* as skin commensal. They showed multiple breaks in the skin surface, allowing the entrance of bacteria. Most importantly, the herpetic lesions included disseminated pustules as sign for a bacterial superinfection.

There are striking clinical similarities to a recently reported concomitant *S. lugdunensis* and cytomegalovirus infection in an immune deficient patient with scleroderma [10], which is suggestive for a common infectious pathway. Both patients were immunosuppressed, featured a co-infection with *S. lugdunensis* and a herpes virus, showed a systemic inflammatory response syndrome (SIRS) and rapidly died despite an appropriate antibiotic therapy. Bacterial superinfections of zoster lesions and secondary bacterial infections following respiratory viral infection are common, but the phenomenon of "lethal synergism" is an intriguing new pathogenic mechanism and was first proposed by Beadling and Slifka in 2004 [46]. Different murine models were established to further analyze this mechanism and suggest several factors to cause the high and rapid lethality: a thousandfold increased bacterial titer in solid organs during an acute viral challenge, an increased inflammatory cytokine production or a reduced antibacterial granulocyte defence as a consequence of the innate antiviral immune response [46-48]. Given the aggravating value of coinfections between influenza virus and *S. aureus*, it is tempting to speculate that a similar situation exists in coinfections with *S. lugdunensis* and herpes viruses.

Conclusion

Here we report a case of fulminant and fatal septicaemia with *S. lugdunensis*, following a pelvic varicella-zoster virus infection in an immune-deficient patient. The most likely bacterial entry site was a bacterial superinfection of herpes zoster lesions of dermatome Th12. Despite *S. lugdunensis* belonging to the group of CoNS with a usually low virulence, infections with *S. lugdunensis* have recently been associated with an aggressive course and high mortality. Therefore early identification and begin of an appropriate antibiotic therapy which includes linezolid seem crucial for the patient's survival. To allow a true judgement on incidence estimation of *S. lugdunensis* infections as well as their clinical course, CoNS in infective isolates should always be characterized to the underlying species, especially when the antibiotic susceptibility pattern reveals a rather unusual susceptibility to most

antimicrobial agents. Furthermore, in case of a suspected *S. lugdunensis* infection, vancomycin should not be considered as the antimicrobial agent of choice with regard to the reported in vivo tolerance of *S. lugdunensis* to glycopeptides.

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