ORIGINAL ARTICLE

Factors associated with methicillin-resistant coagulase-negative staphylococci as causing organisms in deep sternal wound infections after cardiac surgery

R. Sommerstein[1], **P. Kohler**[1], **M. J. Wilhelm**[2], **S. P. Kuster**[1] **and H. Sax**[1]

1) *Division of Infectious Diseases and Hospital Epidemiology and* 2) *Division of Cardiac Surgery, University Hospital Zurich, Zurich, Switzerland*

Abstract

Established preoperative antibiotic prophylaxis in cardiac surgery is ineffective against methicillin-resistant coagulase-negative staphylococci (CoNS). This case–control study aimed to determine factors predicting deep sternal wound infections due to methicillin-resistant CoNS. All cardiac surgery patients undergoing sternotomy between June 2009 and March 2013 prospectively documented in a Swiss tertiary care center were included. Among 1999 patients, 82 (4.1%) developed deep sternal wound infection. CoNS were causal in 36 (44%) patients, with 25/36 (69%) being methicillin resistant. Early reintervention for noninfectious causes (odds ratio (OR) 4.3; 95% confidence interval (CI) 1.9–9.5) was associated with methicillin-resistant CoNS deep sternal wound infection. Among CoNS deep sternal wound infection, perioperative antimicrobial therapy (p 0.002), early reintervention for noninfectious causes (OR 7.9; 95% CI 0.9–71.1) and time between surgery and diagnosis of infection over 21 days (OR 10.8; 95% CI 1.2–97.8) were associated with methicillin resistance. These findings may help to better tailor preoperative antimicrobial prophylaxis.

Keywords: Cardiac surgery, coagulase-negative staphylococci, methicillin resistance, sternotomy, surgical site infection
Original Submission: 11 February 2015; **Accepted:** 5 April 2015
Article published online: 20 April 2015

Preliminary results from this study were presented 28–29 August 2014 at the Joint Annual Meeting of the Swiss Society for Infectious Diseases, Aarau, Switzerland, and 16–18 November 2014 at the Healthcare Infection Society International Conference, Lyon, France.
Corresponding author: R. Sommerstein, Department of Infectious Diseases, University Hospital Bern, 3010 Bern, Switzerland
E-mail: rami.sommerstein@insel.ch
S.P. Kuster and H. Sax contributed equally to this article, and both should be considered senior author.

Introduction

Surgical site infections (SSI) in cardiac surgery with midline sternotomy remain a major challenge. Reported incidence varies between 2% and 14% for all SSI and between 2% to 4% for deep sternal wound infections. Deep sternal wound infections have a substantial impact on morbidity and mortality and are associated with considerable costs [1–8]. Several risk factors for deep sternal wound infection have been described, either related to comorbidities (e.g. diabetes and obesity) or interventions (e.g. duration of surgery and reintervention) [1,9–12].

The most common etiologic pathogens are *Staphylococcus aureus,* coagulase-negative staphylococci (CoNS) and Gram-negative bacteria [4,7,13–15]. In one study, infection control interventions have been successful in reducing the rate of sternal SSI caused by *S. aureus,* while the incidence of deep sternal wound infection attributable to CoNS has not declined in parallel [7].

Known risk factors for sternal SSI due to CoNS are length of hospital stay before surgical intervention, duration of surgery and reintervention at the same surgical site [15]. Intraoperative, presumably airborne, transmission of CoNS from healthcare workers to patients has been shown to occur [16,17]. In addition, there is evidence that CoNS from clinical specimens are closely related to one another at a hospital, interhospital or even interregional level [18,19].

http://dx.doi.org/10.1016/j.nmni.2015.04.003

Factors associated with methicillin resistance in CoNS causing deep sternal wound infection in patients with heart surgery involving sternotomy have not been defined yet. Understanding these factors is important for infection control and antibiotic prophylaxis policy, as routine prophylaxis recommended by official guidelines does not cover SSI caused by methicillin-resistant bacteria [20]. Furthermore, empirical antimicrobial treatment in the case of an infection could be more targeted in cases with presence of known risk factors. We aimed to address this question by analysing a large prospective cohort of cardiac surgery patients with midline sternotomy from a major Swiss university-affiliated tertiary care center.

Methods

Setting

The University Hospital Zurich is a tertiary care center with 942 beds. In 2012, 1166 patients underwent cardiac surgery, including coronary artery bypass graft procedures, valve repairs and replacements, heart transplantation and placement of implantable cardiovascular devices, e.g. aortic grafts and ventricular assist devices. Cefuroxime 1.5 g provided intravenously with repetition every 8 hours in case of prolonged surgery is the standard antibiotic prophylaxis for initial surgery and reintervention.

Surveillance of SSI

All consecutive patients undergoing heart surgery at the University Hospital Zurich are included prospectively in the surveillance protocol of the Swissnoso Surgical Site Infection Surveillance Module (http://www.swissnoso.ch/), which is based on the US Centers for Disease Control and Prevention definitions [21]. As a modification to the latter, sternal osteitis is considered a deep incisional SSI even in the absence of concomitant mediastinitis (http://www.swiss-noso.ch/wp-content/uploads/2009/05/2_-D-30-09-2013_D_Teilnehmerhandbuch_VERSION-UPDATE_SEPTEMBER-20132.pdf).

Importantly, the protocol includes rigorous postdischarge surveillance. All patients are contacted by phone 1 month after the surgical intervention, or after 1 year if an implant, i.e. sternal plate, wire cerclage, valve repair or replacement, was involved. If suspicion for SSI arises, the case is further discussed with the treating physician.

The data collected for each patient included the following: sex, age, days between admission and surgery, timing of preoperative antibiotic prophylaxis, body mass index (BMI), wound contamination class, American Society of Anesthesiologists (ASA) score, intervention exceeding 75th percentile of duration cut point [22] and reintervention. National Nosocomial Infections Surveillance scores were calculated [22]. In case of SSI, the depth of the infection, i.e. superficial incisional, deep incisional or organ/space, and the causing organisms were recorded.

Case definition

All consecutive patients undergoing coronary artery bypass graft procedures, valve repairs and replacements, or placement of implantable cardiovascular devices, e.g. aortic grafts, ventricular assist devices, between 1 June 2009 and 30 September 2013 were included, with the exception of minimally invasive interventions. SSI surveillance was suspended between 1 January 2011 and 31 March 2011.

Deep sternal wound infection was defined according to the Swissnoso Surgical Site Infection Surveillance Module definitions (http://www.swiss-noso.ch/wp-content/uploads/2009/05/2_-D-30-09-2013_D_Teilnehmerhandbuch_VERSION-UPDATE_SEPTEMBER-20132.pdf). In brief, deep sternal wound infection had to become manifest within 1 year after surgery and had either to be confirmed by a treating physician *or* to be present with purulent drainage of the deep incisional compartment or spontaneous wound dehiscence together with pain, redness or temperature >38°C *or* a positive bacterial culture from a deep tissue sample. Mediastinitis was deep sternal wound infection with cultural, histologic or macroscopic evidence of mediastinal involvement. Postoperative endocarditis was not considered a deep sternal wound infection.

Besides the clinical criteria, CoNS etiology of deep sternal wound infection required either a monoculture of CoNS from at least one deep tissue sample. In polymicrobial deep sternal wound infection, CoNS was only considered when additional pathogens were *Propionibacterium acnes* or appeared in follow-up cultures only. Antibiotic susceptibility was reported for the first CoNS isolated from the wound. Susceptibility testing was done on Müller-Hinton agar (Becton-Dickinson, Franklin Lakes, NJ, USA) using MacFarland 0.5 from overnight cultures followed by incubation at 35°C for 16 to 18 hours. The disc diffusion method according to Kirby-Bauer was applied for susceptibility testing (http://www.eucast.org/antimicrobial_susceptibility_testing/disk_diffusion_methodology/) using discs from i2a (Montpellier, France). Inhibition zone diameters were determined and recorded in the automated Sirweb/Sirscan system (i2a) and interpreted according to EUCAST 1.3 guidelines from the European Committee on Antimicrobial Susceptibility Testing using their breakpoint tables for interpretation of minimum inhibitory concentrations and zone diameters, versions 1.3 and 2.0 (http://www.eucast.org/antimicrobial_susceptibility_testing/previous_versions_of_tables/).