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Surgical site infection after central venous catheter-related infection in cardiac surgery. Analysis of a cohort of 7557 patients

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SUMMARY

The aim of this study was to establish the relationship between the occurrence of a surgical site infection (SSI) and the presence of a central venous catheter-related infection (CVCRI). The Department of Thoracic and Cardiovascular Surgery, University Hospital, Rouen, has carried out a prospective epidemiological survey of all nosocomial infections (pneumonia, SSI and CVCRI) since 1997. The study group included all consecutive patients who underwent cardiac surgery over a 10-year period from 1997 to 2007. A nested case–control study was conducted to identify the risk factors for SSI after CVCRI. Cases were patients with SSI after CVCRI and controls were randomized from patients who presented with CVCRI not followed by SSI. In total, 7557 patients were included and 133 SSIs (1.7%) were identified. The rate of superficial SSI was 0.7% [95% confidence interval (CI): 0.5–0.9] and of mediastinitis was 1.0% (95% CI: 0.8–1.2). Among the 133 cases of SSI, 12 (9.0%; 95% CI: 5.0–14.8) occurred after a CVCRI with identical micro-organisms. CVCRI [adjusted odds ratio (aOR): 5.2; 95% CI: 3.2–8.5], coronary artery bypass grafting (aOR: 2.9; 95% CI: 1.6–5.2), and obesity (aOR: 11.4; 95% CI: 1.0–130.1) were independent factors associated with SSI. The new finding of this study is that patients with CVCRI were 5.2 times more likely to develop SSI compared to patients without CVCRI.

Introduction

Surgical site infection (SSI) is a serious complication after cardiac surgery and can prolong hospitalization by more than three weeks.[1,2] SSIs are associated with increased hospital costs, and significant hospital mortality, ranging from 0 to 25%.[1,4–8] Incidence of SSI after cardiac surgery ranges from 0.55 to 9.7%.[2–5,7–11]

A number of risk factors for SSI after cardiac surgery have been identified in the literature including obesity, diabetes, use of bilateral mammary artery grafts, prolonged length of stay in the intensive care unit, re-exploration for bleeding, smoking, and chronic obstructive pulmonary disease (COPD).[2–5,7–15]

It is well known that coincident remote site infections increase the risk of SSI.[16,17] In adult cardiac surgery, Abboud *et al.* showed that infection at another site was a risk factor for SSI, but without specifically targeting central venous catheter-related infection (CVCRI).[3] Patients undergoing cardiac surgery appear to be at increased risk of nosocomial infections due to the use of invasive devices [central venous catheter (CVC), Swan–Ganz, or intra-aortic balloon counterpulsation].[18,19]

Some studies have investigated the association of SSI with the presence of a CVC. In adult cardiac surgery, Rosmarakis *et al.* identified the duration of CVC placement as a risk factor for nosocomial infection.[20] In vascular surgery, Rebollo *et al.* found the presence of a CVC to be a risk factor for nosocomial infection (SSI, pneumonia, urinary tract infection, bacteraemia).[21] In paediatric cardiac surgery, Ben-Ami *et al.* found CVC dwell time to be a risk factor for sternal wound infection.[22]

Sandoe *et al.* used risk of surgical site contamination by catheter-related bacteraemia to justify a study comparing different

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antibiotic protocols, but they failed to provide convincing evidence of any assosciation.[23] Colonized intravascular catheters are the most commonly identified source of nosocomial endocarditis.[24] However, to our knowledge no study has addressed the risk of SSI after CVCRI in patients undergoing cardiac surgery.

The aims of this study were to determine the proportion of SSIs that could be attributed to CVCRI and to identify the risk factors associated with SSI after CVCRI.

Methods

Setting

University Hospital Charles Nicolle is a 2500 bed tertiary-care teaching hospital in Rouen, France, which has 107,000 admissions per year. The Department of Thoracic and Cardiovascular Surgery (DTCS) is a 37-bed ward including an intensive care unit (ICU), employs five surgeons and 10 anaesthetists and performs 800 open surgical procedures each year. The patients are admitted to hospital the night before their scheduled surgery. Each surgical patient had a CVC sited prior to surgery. As a consequence, there are no patients in our cohort without a CVC.

Surveillance

An in-house prospective surveillance programme identifying nosocomial infection including SSI, CVCRI and pneumonia has included each consecutive patient undergoing surgery in the DTCS since October 1997. The diagnosis of mediastinitis and nosocomial pneumonia was based on Centers for Disease Control and Prevention (CDC) definitions.[16,25] After removal, all catheter tips were cultured. Quantitative catheter culture was performed according to the Brun–Buisson modified Cleri technique.[26] Results were expressed as the number of colony-forming units (cfu)/mL. CVCRIs were classified into three categories according to the definition of the French Society of Anaesthesia and Intensive Care.[27] Colonization was defined as a quantitative CVC culture $\geq 10^3$ cfu/mL without clinical evidence of infection. CVC infection was defined as quantitative CVC culture $\geq 10^3$ cfu/mL and pus at the insertion site or clinical signs improving within 48 h after catheter removal. Catheter-related bloodstream infection was defined as bloodstream infection occurring 48 h before or after catheter removal and positive culture with the same micro-organism of either (i) quantitative CVC culture $\geq 10^3$ cfu/mL; (ii) positive culture from pus from insertion site; (iii) quantitative blood culture ratio CVC blood sample: peripheral blood sample >5; or (iv) differential time to positivity of blood cultures: CVC blood sample culture positive ≥2 h before peripheral blood culture (blood samples drawn at the same time).

For each patient, a questionnaire was completed including the following data: age, sex, date of admission and discharge. An infection control practitioner and a hospital epidemiologist were involved in the surveillance programme. Microbiology laboratory results were reviewed, and patients' records were discussed weekly to identify possible cases of nosocomial infection. Nosocomial pneumonia and CVCRI identified by epidemiological surveillance were validated weekly by anaesthetists and SSI was validated by the surgeon. Every three months the results of the surveillance programme were communicated to the cardiac surgery department staff. The results included the description of the patient population in terms of age, sex, urgent or non-urgent surgery, type of surgery [valve, coronary artery bypass grafting (CABG), valve and CABG], type of SSI (superficial or deep), type of CVCRI (colonization, CVC infection, catheter-related bloodstream infection), or nosocomial pneumonia, and micro-organisms involved.

Design

We retrospectively analysed a cohort of patients who had undergone cardiac surgery since 1997. A nested case–control study was conducted to identify the risk factors for SSI after CVCRI. Cases were patients with SSI following CVCRI with the same micro-organism, occurring ≤30 days after surgery or one year if a prosthetic valve was left in place. The identical nature of the micro-organism involved in the SSI and CVCRI was not determined by typing but by comparing antibiotic susceptibility. Controls were randomized among patients who presented with CVCRI not followed by SSI. Controls were matched to cases according to five criteria: sex, age (±5 years), year of surgery (±2 years), type of surgery (CABG, valve, or CABG + valve) and type of CVCRI.

Data collection

The following data were recorded retrospectively from hospital records: (i) preoperative clinical data: obesity [defined as body mass index (BMI) ≥ 30 kg/m^2], diabetes, COPD, smoking; (ii) intra-operative data: number of mammary grafts, use of introducer and/or Swan–Ganz catheter; (iii) postoperative data: use of inotropic drugs, re-exploration for bleeding, renal insufficiency, duration of mechanical ventilation, blood transfusion, parenteral nutrition, duration of CVC; (iv) CVCRI data: identity of micro-organism, date of CVCRI, antibiotic used, route of antibiotic administration, duration of treatment, delay between positive bacteriological results and start of antibiotic therapy; (v) catheter-related bloodstream infection: time between discovery of a positive blood culture and removal of the CVC; (vi) SSI data: identity of the micro-organism and date of occurrence.

Management of CVC colonization was according to institutional protocol: colonization was only treated in patients undergoing surgery for valve replacement; patients undergoing surgery for CABG were monitored but not routinely treated. The institutional protocol for prophylactic antibiotics before surgery included a second-generation cephalosporin (cefazolin), 2 g preoperatively followed by a further injection of 1 g after 4 h.

Statistical analysis

Analysis of qualitative data was performed using frequency distributions. The chi-squared test or Fisher's exact test were used for comparisons. Quantitative variables were summarized by means and medians and compared using Student's *t*-test. Logistic

Table I
Main characteristics of the 7557 patients and factors associated with surgical site infection: univariate analysis and logistic regression ($N = 7557$) (1997–2007)

	SSI$^+$	SSI$^-$	Univariate analysis	Multivariate analysis
	($n = 133$)	($n = 7424$)	*P*-value	aOR (95% CI)
Mean age, years (SD)	65.8 (10.2)	65.1 (13.8)	NS	
Sex ratio (M:F)	3	2.5	NS	
Emergency (%)	30.2	15.4	<0.001	1.3 (0.7–2.5)
Type of surgery				
CABG (%)	68.0	46.2	<0.001	2.9 (1.6–5.2)
Valve (%)	20.0	44.4		
Valve + CABG (%)	12.0	9.4		
Nosocomial pneumonia (%)	7.6	7.5	NS	
CVCRI (%)	24.8	10.4	<0.001	5.2 (3.2–8.5)

SSI, surgical site infection; aOR, adjusted odds ratio; CI, confidence interval; CABG, coronary artery bypass graft; CVCRI, central venous catheter-related infection; NS, non-significant.