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Long term cardiovascular impacts after burn and non-burn trauma: A comparative population-based study

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ABSTRACT

Objective: To compare post-injury cardiovascular disease (CVD) hospital admissions experienced by burn patients with non-burn trauma patients and people with no record of injury, adjusting for socio-demographic, health and injury factors.

Methods: Linked hospital and death data were analysed for a cohort of burn patients (n=30,997) hospitalised in Western Australia during the period 1980–2012 and age and gender frequency matched comparison cohorts (non-burn trauma: n=28,647; non-injured: n=123,399). The number and length of hospital stay for CVD admissions were used as outcome measures. Multivariate negative binomial regression was used to derive adjusted incidence rate ratios (IRR) and 95% confidence intervals (95%CI). Multivariate Cox regression models and hazard ratios (HR) were used to examine first time post-injury CVD admissions.

Results: The burn cohort had a higher rate of CVD (combined) admissions (IRR, 95%CI: 1.16: 1.08–1.24) and spent longer in hospital (IRR, 95%CI: 1.37, 1.13–1.66) than the non-burn trauma cohort. Both the burn cohort (IRR, 95%CI: 1.50, 1.40–1.60) and the non-burn trauma cohort (IRR, 95%CI: 1.29, 1.21–1.37) had higher adjusted rates of post-injury CVD admissions compared with the non-injured cohort. The burn cohort (HR, 95%CI: 2.27, 1.70–3.02) and non-burn trauma cohort (HR, 95%CI: 2.19, 1.66–2.87) experienced significantly elevated first time CVD admissions during the first 6 months after injury, decreasing in magnitude from 6 months to 5 years after injury (HR, 95%CI: burn vs. non-injured; 1.31, 1.16–1.48; non-burn trauma vs. non-injured; 1.16, 1.03–1.31); no significant difference in incident admission rates was found beyond 5 years (HR, 95%CI: burn vs. non-injured; 0.99, 0.92–1.07; non-burn trauma vs. non-injured; 1.00, 0.93–1.07).

Conclusions: Burn and non-burn trauma patients experience elevated rates of post-injury CVD admissions for a prolonged period after the initial injury and are particularly at increased risk of incident CVD admissions during the first 5-years after the injury event. Detailed clinical data are required to help understand the underlying pathogenic pathways triggered by burn

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and non-burn trauma. This study identified treatment needs for injury patients, burn and non-burn, for a prolonged period after discharge.

1. Introduction

Traumatic injury triggers a range of host responses including inflammatory, immune and neuroendocrine [1–3]. The impact of injury on sympathetic activity has long been recognised and it is clear that in the acute phase after injury sympathetic changes are critical to survival [4]. However, it is the long-term sustained activation that has the potential to negatively affect health outcomes. This long-term activation has been demonstrated to persist for up to 3 years in paediatric patients with severe burns [5,6]. However there remains a paucity of long-term health data of patients that suffer an injury.

Our previous investigations of post-burn cardiovascular morbidity using linked health data of paediatric [7] and adult [8] patients found increased post-burn hospital use for cardiovascular diseases (CVD) when compared with non-injured people. In addition, research into the long-term effects of non-severe burns that incorporated animal, clinical and population-based investigations also identified cardiac changes and cardiovascular pathology [9]. These results suggest that burns are a component cause of post-discharge cardiovascular morbidity.

Cardiovascular responses are observed immediately after a traumatic insult; however, the pattern of response appears to depend on the type of injury, and responses may be different, for example, if penetrating vascular trauma, burns or fracture are compared [1]. In addition, recent animal research reported that the immune response to skin trauma is dependent on the type of injury, with different innate and adaptive immune responses triggered by burns and excision injury [10].

Given that inflammatory, immune, endocrine and stress responses may vary according to the type of injury, differences in subsequent post-injury cardiovascular health may also occur. The objective of this study was to use linked hospital data to examine the risk of post-injury cardiovascular morbidity experienced by burn and non-burn trauma patients compared with non-injured people, adjusting for socio-demographic, pre-existing health factors and injury severity.

2. Methods

This retrospective population-based longitudinal project was approved by the Human Research Ethics Committees of the Western Australian Department of Health and the University of Western Australia, and forms part of the Western Australian Population-based Burn Injury Project [11].

We analysed de-identified linked health data from the Western Australian Death Registry and the Hospital Morbidity Data System (public and private hospital admissions data) supplied by the Western Australian Data Linkage System (WADLS) [12]. The WADLS is a population-based linkage system that links health data for the entire population of Western Australia. In Australia all people have access to medical care and acute care is provided in either private or public hospitals, where public hospital treatment is free for public patients.

The study included three cohorts: (i) Burn cohort: all persons hospitalised for a first or index burn injury between 1980 and 2012; (ii) Non-burn trauma cohort: A random sample of persons hospitalised with a first (index) non-burn trauma, age and gender frequency-matched from the same Statistical Local Area (SLA) as the index burn case (~1:1) for each year. This trauma comparison cohort excluded those with burns; effects of foreign bodies entering through orifices; injuries to nerves and spinal cord; poisoning; toxic effects of nonmedical substances (e.g. alcohol); and complications of surgical and medical care; and, (iii) No injury cohort: Cohort of individuals randomly selected from the Western Australia Birth Registrations and Electoral Roll who did not have a record of injury admission during the study period. This cohort was frequency-matched on age, gender from the same SLA as the index burn case (~4:1) for the study period.

Indices of social disadvantage (Socio-economic Indices for Areas (SEIFA) [13]) and remoteness (Accessibility Remoteness Index of Australia (ARIA+) [14]) were supplied for the burn, non-burn trauma and non-injured cohorts. Mortality data included date and cause of death. Admissions for diseases of the circulatory system were identified and classified using principal diagnosis data with International Classification of Diseases and Health Related Problems (ICD) codes (ICD9 390-459; ICD10 I00-I99, G45).

Age was classified into 5-year age groups with adults 85 years and older included in one group. The Charlson Comorbidity Index (CCI) [15] was used to identify comorbidities using a 5-year look-back period in the hospital files [16]; the variable "any comorbidity" (0: CCI=0; 1: CCI≥1) was included in the analyses. The CCI was used as an indicator of chronic health status at baseline and accounted for a range of chronic diseases including acute myocardial infarction, heart failure, peripheral vascular disease, stroke, lung diseases, diabetes, chronic gastrointestinal disease, cancer, dementia and rheumatic diseases.

Diagnoses codes (ICD9 740-759; ICD10 Q00-G99) were used to identify record of congenital anomaly (yes/no). A record of ventilation during the index admission was defined by procedure codes (ICD10-AM: 92038-00, 92040-00, 92051-00, 13882-00, 13882-01, 13882-02) and used as proxy indicator of injury undergoing surgery at index admission. Record of circulatory disease admission in year prior to study start was identified using (ICD9 390-459; ICD10 I00-I99, G45).

A variable was generated to classify injury type (0 no injury; 1 non-burn trauma; 2 burn injury). Depth of burn (full thickness, partial thickness, erythema (first degree) and unspecified) and total body surface area percent burned (TBSA %) were classified using ICD codes. TBSA was classified minor (<20%), severe (≥20%) and unspecified (if supplementary ICD

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