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Increased admissions for diabetes mellitus after burn

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ABSTRACT

Background: Currently, limited long-term data on hyperglycaemia and insulin sensitivity in burn patients are available and the data that do exist are primarily related to paediatric severe burns. The aim of this study was to assess if burn is associated with increased post-burn admissions for diabetes mellitus.

Methods: A population-based longitudinal study using linked hospital morbidity and death data from Western Australia was undertaken of all persons hospitalized for a first burn (n = 30,997) in 1980–2012 and a frequency matched non-injury comparison cohort, randomly selected from Western Australia's birth registrations and electoral roll (n = 123,399). Crude admission rates and summed length of stay for diabetes mellitus were calculated. Negative binomial and Cox proportional hazards regression modelling were used to generate incidence rate ratios (IRR) and hazard ratios (HR), respectively.

Results: After adjustment for socio-demographic factors and pre-existing health status, the burn cohort had 2.21 times (95% Confidence Interval (CI): 1.36–1.56) as many admissions and almost three times the number of days in hospital with a diabetes mellitus diagnosis (IRR, 95% CI: 2.94, 2.12–4.09) than the uninjured cohort. Admission rates were significantly elevated for those burned during childhood (<18 years, IRR, 95% CI: 2.65, 1.41–4.97) and adulthood (≥18 years, IRR, 95% CI: 2.12, 1.76–2.55). Incident admissions were significantly elevated in the burn cohort during the first 5 years post-burn when compared with the uninjured (HR, 95% CI: 1.96, 1.46–2.64); no significant difference was found beyond 5 years post-burn (HR, 95% CI: 1.08, 0.82–1.41).

Conclusions: Findings of increased hospital admission rates and prolonged length of hospital stay for diabetes mellitus in the burn cohort provide evidence that burns have longer term effects on blood glucose and insulin regulation after wound healing. The first five years after burn discharge appears to be a critical period with significantly elevated incident admissions for diabetes mellitus during this time. Results would suggest prolonged clinical

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management after discharge and or wound healing to minimise post-burn admissions for diabetes mellitus is required.

1. Introduction

Severe burn characteristically triggers a range of systemic responses that includes inflammatory and immune changes, endocrine responses, stress and hypermetabolism [1,2]. While these responses are experienced trauma, surgical and critically ill patients, the patterns, persistence and severity of these responses are unique to burn [3,4]. After burn, there is a period of increased glycolysis, lipolysis, proteolysis, insulin resistance, liver dysfunction and decreases in lean and total body mass [5–10]. While it was believed that these metabolic alterations would resolve with complete wound healing, research has identified that these responses may last for a prolonged period [1,2,11–13].

Currently, minimal long-term data on hyperglycaemia and insulin sensitivity in burn patients are available and the data that do exist are primarily related to paediatric severe burns. Sustained hypermetabolic alterations after burn, including elevations in serum cytokines, catecholamines and basal energy requirements, have been accompanied by impaired glucose metabolism and insulin sensitivity that have been shown to persist for at least three years after the initial burn [2,12,14,15].

Initial investigations of morbidity after burn using population-based data have identified increased long-term post-burn circulatory [16,17] and musculoskeletal [18,19] morbidity, after both minor and severe burns, as well as increases in incidence of some types of cancers [20,21], suggesting long-term effects of burns on the immune system. Given the limited long-term data available on the persistence of hyperglycaemia, insulin sensitivity and insulin resistance in burn patients, this study aimed to use population-based linked hospital morbidity data to investigate if burn patients have increased diabetes mellitus morbidity in terms of hospital use after discharge for the initial burn.

2. Methods

This study used population-based linked hospital morbidity and death data from the Western Australian Data Linkage System which is a validated record linkage system that routinely links administrative health data for the whole population of Western Australia [22]. The project has approvals from the University of Western Australia and the Western Australian Department of Health, Human Research Ethics Committees. The retrospective cohort study forms part of the Western Australia Population-based Burn Injury Project which supports multiple studies. The project methods have been published previously [16,23,24].

A de-identified extraction of all linked hospital morbidity records from the Hospital Morbidity Data System for all persons admitted to hospital with an index burn in Western Australia for the period 1 January 1980 to 30 June 2012. The index burn was defined as the first hospital admission with a burn in either principal or additional diagnosis data using the International Classification of Diseases (ICD) codes (ICD9-CM 940-949; ICD10-AM T20-T31). A population-based comparison uninjured cohort was randomly selected from the Western Australian Birth Registrations and Electoral Roll and excluded any person with an injury hospitalisation during the study period. The uninjured comparison cohort was frequency-matched on birth year (4:1) and gender of the burns cases for each year from 1980 to 2012.

Hospital and death data were linked to each cohort (burn, non-injury) for the period 1980–2012. Indices of socio-economic disadvantage [25] and geographic remoteness [26] based on national census data were also linked to each cohort. Hospital admissions data included principal and additional diagnoses, age and gender, Indigenous status, admission and discharge dates, burn characteristics (total burns surface area percent (TBSA %), burn depth, site) and residential postcode. Mortality data included the date and cause of death. The principal diagnosis data were used with ICD Chapter 4 codes to identify admissions for diabetes mellitus (ICD10 E10-E14); ICD9 codes were mapped to ICD10 codes [27].

TBSA% was classified by ICD supplementary codes (ICD9 948; ICD10 T31) into three groups: minor burns (TBSA $<$ 20%), severe (TBSA $\geq$ 20%), and unspecified TBSA%. Charlson co-morbidity index (CI) [28] using principal and additional diagnosis data and a 5-year look back period was used to estimate comorbidity [29] and then classified as any comorbidity at baseline (CI = 0; $\geq$1). A variable to identify prior diabetes mellitus admissions was generated. Indices of socio-economic disadvantage were partitioned into quintiles from most to least disadvantaged and geographic remoteness was classified into five categories (major cities, inner regional, outer regional, remote and very remote).

The total number of years a person was at risk (person-years) was estimated from the final discharge date for the burn cases and this date was used for the respective frequency matched non-injury controls. Categorical and non-parametric continuous variables were compared using χ^2 and Kruskal–Wallis tests, respectively; level of statistical significance set at 0.05. Statistical analyses were performed using Stata version 12 (StataCorp. LP, College Station, United States of America).

The total number of admissions and summed length of stay in hospital (days) for diabetes mellitus after burn discharge were used as outcome measures. The admission of the index burn was not included in these outcomes. Analyses were undertaken on the total burn and uninjured cohorts, and subcohorts defined by burn TBSA severity, gender and age ($<$18, $\geq$18 years at study start). Due to non-linearity, age was entered into the final models (total cohorts with combined age)

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