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Stroke after burn: Population data analysis

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ABSTRACT

Purpose of the study: We aimed to describe a population cohort study of burns with the prevalence of stroke, and discuss possible etiologies.

Analytical methods: This study uses data obtained from the Longitudinal Health Insurance Database 2005 (LHID2005). The study cohort consisted of all patients who had diagnoses of burn recorded in the database (ICD-9-CM codes 948) between January 1 2004 and December 31 2008 (N = 1549).

Main findings: The patients with burn and the comparison cohort are 7410 patients, 146 experienced stroke during the follow-up period of up to 5 years. In average, the burn patients suffered from stroke by 1.48 years after burn. The hazard ratio of stroke was 1.74 (95% CI, 1.15–2.63) for patients with TBSA burn <20%, 3.78 (95% CI, 1.39–10.26) for patients with TBSA burn ≥20%. The adjusted hazard ratio of ischemic stroke was 1.63 for patients with TBSA burn <20%, 2.96 for patients with TBSA burn ≥20%, whereas the hazard ratio of hemorrhagic stroke were not significant ($p = 0.231$).

Conclusions: In our study, severe burned patients, more than 60 years of age, had higher risk of stroke in their recovery life. We suggest close follow up for the burn patients in high risk of stroke.

1. Introduction

A burn is not only a localized injury affecting the skin; but also, it involves all systems of the body. Brain is one of the organs that may be influenced by burn. In the 1970s, Antoon, Andreasen, and Sevitt observed that some patients presented with neurological problem after burn, which they termed "burn encephalopathy" [1–3]. At that time, the causes leading to the condition were not clear.

Stroke is regarded as a possible early complication of burn. Winkelman and Galloway [4] reported that eighteen percent of their autopsy cases had cerebral infarcts. In almost half of these patients, the infarcts were caused by septic arterial occlusions or other complications of the burn, particularly the condition of disseminated intravascular coagulation (DIC) and

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septic shock. Another report [5] showed that 0.07% (9/13,468) patients developed stroke after burns, while the median duration between the burn and stroke onset was 33 days (range from 2 to 307 days). The stroke after burn included ischemic infarction (four patients), intracerebral hemorrhage (three), multiple hemorrhagic infarction (one), and subdural hematoma (one). Seven out of the nine patients revealed the presence of septic conditions that occurred subsequent to the burn. Sepsis are the leading cause of the early occurrence of cerebral stroke after burn [6]. However, the incidence of stroke as the late sequela of burn is unknown. Till now, stroke in the recovery phase of burn in population has not been reported in the literature. We describe a population cohort study of burns with the prevalence of stroke, and discuss possible etiologies.

2. Materials and methods

2.1. Study population and study design

This study uses data obtained from the Longitudinal Health Insurance Database 2005 (LHID2005), released by the Taiwan National Health Research Institutes (NHRI). There are currently over 22 million enrollees covered by the program, representing approximately 99% of the island's population. The LHID2005 contains all the medical claims data for one million beneficiaries, randomly sampled from over 22 million enrollees under the National Health Insurance (NHI). There were no statistically significant differences in age, gender, or healthcare costs between the sample group and all enrollees, as reported by Taiwan's NHRI [7].

The study cohort consisted of all patients who had diagnoses of burn recorded in the database (ICD-9-CM codes 948) between January 1 2004 and December 31 2008 ($N = 1549$). Exclusion criteria were as follows: patients with missing variables such as birthday, sex, age or urbanization level were excluded from the study ($N = 10$), as well as those with diagnosis of stroke before that of burn ($N = 47$). The resulting study cohort included 1482 patients with burn.

The controls were retrieved from the remaining patients in the LHID2005 who had been registered between January 1 2004 and December 31 2008. Patients were excluded if they had been diagnosed as having stroke before 2004 or burn. The selected 5928 patients were matched with those in the study cohort (4 control patients per case patient) in terms of age ($\leq$30, 31–40, 41–50, 51–60, 61–70 and $>$70 years), gender.

Stroke as outcome variable included all types of stroke (ICD-9 codes 430–438). Each case was followed up from their entry day to they occurred stroke, and to the end of 2008.

2.2. Baseline variables

Baseline variables, including age, urbanization level, and monthly income, diabetes mellitus (DM; ICD-9-CM codes 250), hypertension (ICD-9-CM codes 401–405), hyperlipidemia (ICD-9-CM codes 272.0–272.4), and coronary heart disease (ICD-9-CM codes 410–414), were obtained for all patients. Urbanization levels in Taiwan were stratified into three levels. As to the urbanization level, all 359 cities/towns in Taiwan are stratified into seven levels according to standards published by the Taiwan NHRI, with 1 referring to the "most urbanized" and 7 referring to the "least urbanized." For our study, levels 1 and 2 were combined into a single group, referred to as urban; levels 3 and 4 were combined into a single group, referred to as suburban; and the remaining three levels (5, 6, and 7) were combined into a single group, thereafter referred to as rural.

2.3. Statistical analysis

Pearson's Chi-squared tests and the Fisher's exact test were used to compare cohort differences for Table 1. We evaluated stroke hazard changes over time at baseline levels of burn using Cox proportional hazards model to examine differences in the risk of stroke between the two cohorts after adjusting for patient's age, DM, hyperlipidemia, hypertension, coronary heart disease and urbanization level. In order to control for age and co-morbidities, we used propensity score to balance age and co-morbidities. Rosenbaum and Rubin [8] proposed propensity score to be the probability of treatment assignment conditional on observed baseline covariates. The propensity score is a balancing score, to take the distribution of observed baseline covariates will be similar between burns and controls. The propensity method has been used in many studies to control confounding factors. Our propensity score was predicted from the baseline variables included age, gender, urbanization level, diabetes mellitus, hypertension, hyperlipidemia and coronary heart diseases. To meet the proportional hazards assumption, each dichotomous variable

Table 1 – Demographic characteristics and comorbid medical disorders for patients with burn and age- and gender-matched comparison control ($N = 7410$).

Baseline variable	Burn patients $n = 1482$ N (%)	Control patients $n = 5928$ N (%)
Age (years)		
≤30	715 (48.2)	2860 (48.2)
31–40	212 (14.3)	848 (14.3)
41–50	244 (16.5)	976 (16.5)
51–60	158 (10.7)	632 (10.7)
61–70	80 (5.4)	320 (5.4)
>70	73 (4.9)	292 (4.9)
Gender		
Male	744 (50.2)	2976 (50.2)
Female	738 (49.8)	2952 (49.8)
Urbanization level		
Urban	899 (60.7)	3491 (58.9)
Suburban	448 (30.2)	1806 (30.5)
Rural	135 (9.1)	631 (10.6)
Diabetes mellitus		
Yes	72 (4.9)	221 (3.7)
No	1410 (95.1)	5707 (96.3)
Hypertension		
Yes	104 (7.0)	436 (7.4)
No	1378 (93.0	5492 (92.6)
Hyperlipidemia		
Yes	63 (4.3)	216 (3.6
No	1419 (95.7)	5712 (96.4)
Coronary heart disease		
Yes	43 (2.9)	155 (2.6)
No	1439 (97.1)	5773 (97.4)