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Early coagulopathy of major burns

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ABSTRACT

Introduction and aims: The pathophysiology and time-course of coagulopathy post major burns are inadequately understood. The aims of this study were to review the incidence of acute coagulopathy post major burns, potential contributing factors associated with this coagulopathy and outcome of patients who developed early coagulopathy.

Methods: A retrospective review of all patients with major burns (≥20% total body surface area (TBSA)) presenting to a tertiary burns referral centre was conducted. Data on demographic, injury characteristics and fluid resuscitation practices were recorded and tested for association with coagulopathy (INR > 1.5 or aPTT > 60 s) at hospital presentation and within 24 h of burns injury. Mortality, intensive care unit (ICU) admission, mechanical ventilation and blood and blood product usage were primary endpoints.

Results: There were 99 patients who met the inclusion criteria with 36 (16) %TBSA burns. Coagulopathy was present in only three patients on presentation, but 37 (37%) patients developed early onset (within 24 h of injury) coagulopathy. Early onset coagulopathy was independently associated with %TBSA burnt ($p < 0.001$) and volume of fluid administered ($p = 0.005$). Early onset coagulopathy was associated with higher volumes of blood and blood product administration, ICU admission and prolonged mechanical ventilation.

Conclusions: Post major burns, a very low proportion of patients presented with coagulopathy, but a substantial proportion of patients developed coagulopathy within 24 h. This and the association of coagulopathy with the volume of fluid resuscitation suggest dilution as a major cause of the early coagulopathy of major burns.

Introduction

Coagulopathy associated with burn injury was well recognised as early as in the 1970s and noted to cause "cut tissues to ooze incessantly over time, resulting in transfusion of bank blood which may contribute to thrombocytopenia resulting in more bleeding and therefore a vicious cycle was established".[1] Forty years on, the exact pathophysiology behind this coagulopathy remains elusive. It has been shown early on that the coagulopathy observed in burn patients shows a high fibrinogen turnover, indicating the presence of a hypercoagulable state.[2,3] This model of consumptive coagulopathy has been challenged by a remarkably low incidence of disseminated intravascular coagulation (DIC) among thermally injured patients.[4] Regardless of pathophysiology, coagulopathy has been repeatedly identified as a risk factor for increased morbidity and mortality in the early post burn period as well as in later clinical course.[5]

Along with its pathophysiology, the exact time course of coagulopathy in thermally injured patients remains relatively unknown. In extensive coagulation analysis on a small sample of burns patients, delayed coagulation abnormalities were observed, possibly secondary to a hypercoagulable state.[6] However, coagulopathy in blunt and penetrating trauma has been observed to be present in the pre-hospital phase, raising the possibility of a cytokine mediated activation of the protein C pathway.[7] As this coagulopathy remains difficult to measure, early management has relied on recognition of massive haemorrhage and resulting massive blood transfusions to manage the acute coagulopathy of trauma through formulaic transfusion of blood products.

Burns injuries are different in rarely requiring massive blood transfusions in the acute phase. Early coagulopathy of major burns may therefore remain untreated till recognised through pathology

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testing, which can take up to an hour post presentation to hospital. After a normal initial coagulation profile, coagulopathy may develop secondary to consumption, dilution or delayed fibrinolysis. Even when recognised, there are no guidelines for the management of coagulopathy in major burns. The aims of this study were to review the incidence of acute coagulopathy post major burns, potential contributing factors associated with this coagulopathy and associations with outcome.

Methods

Setting

The Victorian Adult Burns Service (VABS) at The Alfred Hospital is the state-wide provider of burns care for all adults with complex or major burn injuries serving a population of 5.5 million in South-eastern Australia.

Patients

All patients with a body surface area burnt of greater than or equal to 20% and admitted to the VABS between 01 July 1999 and 30 June 2009 were included in the study. Patients initially received and treated at other hospitals and transferred were excluded. Patients with other tissue conditions admitted under the VABS such as toxic epidermal necrolysis were also excluded. Patients managed palliatively in the first 24 h, those who self-presented and those with incomplete information were also excluded.

Study design

A retrospective explicit chart review of patient records was conducted by two operators. Patient demographics, burn type and extent, vital signs, pathology results, associated injuries, fluid administration, surgical management and outcomes were documented. Vital signs, renal function as measured by urine output, serum urea and creatinine levels, and respiratory function measured by the A-a gradient were end-points measured on presentation, discharge from ED and at 24 h. The time to presentation to the Emergency & Trauma Centre was calculated from the "estimated time of incident" according to Ambulance records. The weight was as estimated by the Emergency & Trauma Centre staff and the percentage of body surface area burnt was as estimated and documented by the admitting Burns Registrar. The fluid administration rate (per % TBSA burns/weight in kg) was used for analyses of fluid resuscitation. This distinguished the fluid administration rate from being a surrogate marker of burns severity or patient body weight, i.e. whether 2 mL/%TBSA/kg or 4 mL/%TBSA/kg was administered was assumed to be independent of burns severity or body habitus. The volume of fluid transfused did not include blood or blood products.

"Acute" coagulopathy was defined as an INR of >1.5 or an aPTT of >60 s recorded at presentation. This was consistent with recent studies on acute traumatic coagulopathy.[8] "Early-onset" was defined as coagulopathy in the first 24 h post burns injury. On arrival to the ED, patients were assessed for inhalation burns by direct visualisation of the airway. Patients suspected to have inhalation burns, but not requiring intubation underwent nasal endoscopy. All intubated patients underwent bronchoscopy to confirm inhalation burns. Inhalation burns were coded to be present regardless of severity. All patients presenting to the ED had their coagulation profile measured on presentation. All included patients had further measurements as determined by clinical need. Patients who did not have both an initial and 24 h measurement of coagulation profile were deemed to have missing data and excluded from the study.

Analysis

Data were stored and collated using Microsoft Excel and analysed using Stata version 11 (Statacorp, TX, USA). Normally distributed continuous data were reported as mean (with standard deviations), whereas skewed or ordinal data were reported as medians (with interquartile ranges). The *t*-test was used to test for a statistically significant difference between two mean values, while the chi-squared test, with the Yates continuity correction, was used to test for a statistically significant difference between two proportions. A p value of <0.05 was considered statistically significant.

Using univariate logistic regression, the crude association between early onset coagulopathy and the selected series of variables was tested (Tables 1 and 2). Those variables with a significant univariate association with early onset coagulopathy were subsequently tested for an independent association with the same outcome using multivariable logistic regression. For the final multivariable logistic regression model, predictor variables showing an independent association with early onset coagulopathy were preserved. Given the total sample size of 99 patients and the proportion of coagulopathy of 37%, the maximum number of co-variates was determined to be 4.[9]

This study was approved by The Alfred Hospital Research and Ethics Committee.

Results

Over the study period, 163 patients with TBSA burnt of $\geq$20% presented to the VABS. Of these, 42 patients had incomplete data

Table 1
Results of test for association with coagulopathy.

Variable	Coagulopathy (SD)	No coagulopathy (SD)	p-Value for test of univariate association
n	37	62	–
Age (years)	42.8 (15.8)	42.7 (16.6)	1.0
Body weight (kg)	77.1 (12.8)	80.4 (15.8)	0.3
% TBSA burnt	45 (20)	31 (11)	<0.001**
Fluid rate (mL/%TBSA/kg)	5.7 (1.9)	4.6 (1.9)	0.01**
Temperature (°C; presentation)	35.5 (1.2)	36.2 (0.9)	0.004*
Temperature (°C; ED discharge)	35.6 (1.2)	36.3 (1.0)	0.02*
Temperature (°C; 24 h)	37.1 (1.2)	37.3 (0.9)	0.4
A-a gradient	95.8 (70.1)	103.1 (61.7)	0.7
Surgery in 24 h	25 (68%)	30 (48%)	0.1
Urine output in ED (mL/h)	95.6 (83.7)	124.1 (64.3)	0.4
Urine output in 24 h (mL/h)	112.9 (48.3)	125.9 (82.2)	0.5

TBSA: Total body surface area; ED: Emergency Department.
* Statistically significant association on univariate analysis.
** Statistically significant association on multivariable analysis.