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Characterization Of The Gender Dimorphism Following Severe Thermal Injury

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Abstract

Background—Gender based outcome differences have been previously studied following thermal injury with a higher risk of mortality being demonstrated in females. This is opposite to what has been found following traumatic injury. Little is known regarding the mechanisms and time course of these gender outcome differences post burn injury.

Methods—A secondary analysis was performed utilizing data from a prospective observational study designed to characterize the genetic and inflammatory response following significant thermal injury (2003-2010). Clinical outcomes were compared across gender (female vs. male) and the independent risks associated with gender were determined utilizing logistic regression analysis after controlling for important confounders. Stratified analysis across age and burn severity was performed while Cox-hazard survival curves were constructed to determine the time course of any gender differences found.

Results—Over the time period of the study, 548 patients met inclusion criteria for the cohort study. Males and Females were similar age, TBSA%, inhalation injury and APACHE score. Regression analysis revealed female gender was independently associated with over a 2-fold higher mortality after controlling for important confounders. (OR 2.2, p=0.049, 95% C.I. 1.01-4.8) The higher independent mortality risk for females was exaggerated and remained significant only in PEDIATRIC patients and demonstrated a dose response relationship with increasing burn size (%TBSA). Survival analysis demonstrated early separation of female and male curves and a greater independent risk of Multiple Organ Failure was demonstrated in the PEDAITRIC cohort.

Conclusions—The current results suggest that gender based outcome differences may be different following thermal injury as compared traumatic injury and that the gender dimorphism may be exaggerated in patients with higher burn size and in those in the pediatric age group, with female gender being associated with poor outcome. These gender based mortality differences occur early and may be a result of a higher risk of organ failure and early differences in the inflammatory response following burn injury. Further investigation is required to thoroughly characterize the mechanisms responsible for these divergent outcomes.

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INTRODUCTION

Burn injury represents a significant proportion of the accidental injury in the United States, with the majority of patients surviving the initial burn insult due to improvements in transport, early resuscitation, and the critical care.[1,2] However, a significant number of patients that survive initially ultimately succumb to their injury or suffer significant morbidity because of the development of nosocomial infection, multisystem organ failure and sepsis.[3] Studies have increasingly sought to better evaluate the risk factors for this delayed morbidity and mortality. There is increasing evidence that gender based outcome differences exist following thermal injury.[4-6] Female gender has been demonstrated to be associated with worse outcome following burn injury and this gender dimorphism may be different from the response following other non-thermal injuries.[7-13] A greater understanding of the mechanisms and pathways responsible for these gender based divergent outcomes following burn injury has the potential to result in novel interventions that can improve outcome and reduce the morbidity associated with this significant public health problem.

We sought to better characterize the time course and potential mechanisms responsible for the gender dimorphism after thermal injury using a multicenter cohort of severely injured burn patients for which post-injury care was relatively controlled. We hypothesized that females would have a worse outcome and that these differences would be most apparent in women of reproductive age.

METHODS

Data were obtained from the Inflammation and the Host Response to Injury Large Scale Collaborative Program (www.gluegrant.org), supported by the National Institute of General Medical Sciences (NIGMS), which was a multicenter prospective cohort study designed to characterize the genomic and proteomic response following burn injury.[14] Burn patients admitted to one of six institutions (one pediatric center and 5 adult centers) over an 8 year period (2003-2010) were included in the current analysis. Inclusion criteria for the overall cohort study included: burn size ≥ 20% TBSA (> 40% TBSA for children) that required surgical treatment and arrival to an enrolling burn center within 96 hours of injury. Exclusion criteria consisted of: age > 90 years, chemical or deep electrical burns, significant associated traumatic injuries (ISS >24) preexisting severe cardiac dysfunction (< 20% ejection fraction), glucocorticoid treatment, malignancy and prior bilateral lower-extremity amputations.[14,15] Clinical data were entered and stored in TrialDb, a web-based data collection platform, by trained research nurses.[16] Integrity of the data was maintained through ongoing curation and external data review by an independent chart abstractor. Standard operating procedures were developed and implemented across all participating centers to minimize variation in post-burn care including: goal directed resuscitation, glycemic control, burn wound management, perioperative antibiotic prophylaxis, nutrition and enteral feeding, management of central venous catheters and blood stream infections, acute lung injury, ventilator associated pneumonia, diagnosis of inhalation injury and venous thromboembolism prophylaxis.[15]

Outcomes of primary interest were in-hospital mortality, nosocomial infection (NI) and the development of Multiple Organ Failure (MOF). While patients were admitted to the ICU, Denver post injury multiple organ failure scores were determined daily for pulmonary, renal, hepatic and cardiac organ systems.[17] The diagnosis of MOF required a maximum Denver post injury multiple organ failure score > 3 beyond 48 hours from burn injury.[18] All nosocomial infectious complications were monitored for, recorded (infection type, culture specimen source, and bacteriology) with the diagnosis of NI requiring specific clinical criteria along with positive culture evidence. Diagnosis of a ventilator associated pneumonia required a quantitative culture threshold of $\geq 10^4$ CFU/ml for bronchoalveolar lavage specimens. Diagnosis of catheter-related blood stream infections required positive peripheral cultures with the identical organism obtained from either a postivie semiquantitative culture (>15 CFU/segment), or positive quantitative culture ($>10^3$ CFU/segment) from a catheter segment specimen.

For the current secondary analysis and due to the spectrum of age and burn severity across the enrolled patients, the cohort was also further stratified by age of the patient (PEDIATRIC 1-14 years, YOUNG 15-50 years, and OLD > 50 years) and burn size (LOW- ≥20% and ≤ 40% TBSA, MODERATE- >40% and ≤ 60% TBSA, HIGH- >60% TBSA) Demographics, burn injury characteristics, resuscitation requirements, and outcomes were compared across gender (Females vs. Males) in univariate analysis and across the age stratified groups. Multivariate logistic regression analysis was then utilized to determine the independent outcome risk differences across gender (Female vs. Male) after controlling for important confounders for the overall cohort and when stratified by age and burn size. Finally, to characterize the time course of any gender based outcome differences, Cox-hazard regression analysis using the same model covariates was employed. Covariates used in the final regression models in addition to gender, included age, burn mechanism, burn size (TBSA%), presenting base deficit, APACHE II score, presence of inhalation injury (yes/no), Glasgow Coma Scale (GCS), body mass index, and presence of pre-existing comorbidities (yes/no).

Data analysis was conducted using SPSS version 20 (Chicago, IL). For univariate analyses Chi-square tests were used to compare categorical variables, and Mann-Whitney tests were used to compare continuous variables. Continuous data are presented as median (interquartile range [IQR]) or mean ± standard deviation unless noted. A p-value of ≤ 0.05 was considered significant. The institutional review board of each participating center approved the cohort study, while the institutional review board at the University of Pittsburgh Medical Center approved this current secondary analysis.

RESULTS

Over the time period of the study, 548 patients met inclusion criteria and constituted the study cohort. Mean burn %TBSA for the cohort was 48±20% with an overall mortality of 15%. Over 26% of patients suffered > 60% TBSA and an additional 33% having between 40% and 60% TBSA. Just over 46% of patients suffered from inhalation injury and the incidence of multiple organ failure, nosocomial infection was 27%, 69%, respectively. The age of the cohort spanned pediatric ages thru the elderly population. (**Figure 1.)** The study

cohort was 72% male. Univariate analysis was performed across gender for the entire cohort and males and females were found to be similar in demographics, burn size, shock severity, preexisting comorbidities and hospital and ICU requirements. Females did have a significantly lower nadir Mean Arterial Pressure over the first 24 hours post-injury and a trend towards lower 24 hour crystalloid resuscitation and more commonly suffered scald burns as compared to flame or flash burn mechanism (**Table.1**) This unadjusted comparison across gender also revealed no clinical differences in the incidence of multiple organ failure or infectious complications but a higher mortality was found for females which did not reach statistical significance.

Comparison of burn injury characteristics across the different stratified age groups (PEDIATRIC 1-14 years, YOUNG 15-50 Years, OLD > 50 years) demonstrated greater burn size and a higher percentage of 3rd degree burns, lower APACHE scores, a worse presenting base deficit and, as expected, a lower volume of resuscitation in the first 24 hours in the PEDIATRIC group. Importantly, despite higher burn severity, PEDIATRIC patients had a significantly lower mortality rate. (**Table 2**.)

Logistic regression was first utilized to determine the independent risks of poor outcome associated with gender for the overall cohort. Our regression model was an excellent predictor of mortality with an area under the curve (AUC) of 0.90 via receiver operating characteristic. Logistic regression analysis revealed that female gender was independently associated with over a 2-fold higher risk of mortality (OR 2.2, p=0.049, 95% C.I. 1.01-4.8). When the regression model was stratified by burn size (LOW-20% and ≤ 40%, MODERATE- >40% and ≤ 60% TBSA, HIGH- >60% TBSA), a dose response relationship was demonstrated and female gender was again associated with a significantly exaggerated (over 4-fold greater) independent risk of mortality in the HIGH burn size group. (**Figure 2**.) Interestingly, when the cohort was stratified by burn depth (% of 3rd degree burns) no significant dose response relationship was found. When the regression model was stratified by age (PEDIATRIC 1-14 years, YOUNG 15-50 Years, OLD > 50 years) the significantly higher mortality risk for females remained significant and exaggerated only in the PEDIATRIC group (OR 12.3, p=0.030, 95% C.I. 1.2-118.5). (**Figure 3**.) To determine if these gender based outcome differences varied across burn centers in the cohort we tested for interaction between gender and burn center site. This interaction term was not significant (p=0.89) suggesting these gender based outcome differences were consistent across all burn centers.

Cox hazard regression analysis was then utilized to further assess the timing of the mortality outcome differences across gender in the different age groups while controlling for important confounders. After the incorporation of time into the model, PEDIATRIC (age 1-14 years) female patients continued to demonstrate over a 10-fold higher independent risk of mortality (HR 10.4, p=0.005, 95% C.I. 2.0-53) with the survival curves demonstrating an early separation soon after admission. (**Figure 4**.) No clinical or significant differences were found for those patients in the YOUNG or OLD age groups.

Finally, to characterize the potential mechanisms responsible for these gender based mortality risk differences, both the development of multiple organ failure (MOF) and

nosocomial infection (NI) were also analyzed using the same logistic regression models for the overall cohort and for the stratified groups. No significant independent differences across gender were found for the development of nosocomial infection, overall or in the stratified groups. With MOF as the model outcome variable, in PEDIATRIC patients with MODERATE or HIGH burn size (> 40% TBSA), female gender was significantly associated with over a 4-fold greater independent risk of MOF (OR 4.1, p=0.027, 95%C.I. 1.2-14.3)

DISCUSSION

Insight into those pre-injury factors that can provide a beneficial or protective effect has the potential to promote novel interventions or mechanistic understanding that results in improved outcomes following injury. An increasing pool of both basic science and clinical literature has documented outcome differences attributable to gender (female vs. male) following traumatic and thermal injury.[4,5,7,8,19-25] Interestingly, the majority of this literature suggests that female gender may be protective following traumatic injury and associated with detrimental outcome following burn injury, relative to their similarly injured and burned male counterparts. Both sex hormones and genetic differences have been postulated to be responsible such outcome differences, with the specific mechanisms responsible remaining controversial and incompletely characterized clinically.[4,7,8,23,26] The results of the current study suggest that the gender dimorphism following significant burn injury may be most exaggerated in patients with higher burn size (TBSA%) and in those in the pediatric age group, with female gender being associated with poor outcome. These gender based mortality differences occur early and may be a result of a higher risk of organ failure and early differences in the inflammatory response following burn injury.

These results add to prior literature which have also demonstrated worse outcome for females following burn injury and provides further insight into the mechanisms that may be responsible. Previous large retrospective studies following burn injury in adults have demonstrated a similar higher mortality risk for females. The age groups where these gender differences were strongest were within the 10 years and 70 years age groups with smaller retrospective studies suggesting a more narrow age range (30-60 years).[4-6] Prior evaluation of gender based outcome differences following pediatric burn injury has been less consistent with some suggesting female pediatric burn patients have an attenuated inflammatory and metabolic response leading to shorter hospital utilization, while others have demonstrated no gender based divergent outcomes.[27-29] Basic science literature both in traumatic and thermal injury has provided a strong argument for a sex hormome mechanism, however, translation of these mechanistic results into the clinical arena have yet to materialize.

The current results differ from prior studies by demonstrating a robust gender dimorphism primarily in the pediatric age group when the effect of sex hormones should be primarily absent. Additionally, secondary to the robust nature of the dataset utilized for the study, this pediatric gender dimorphism for mortality was demonstrated to be most prominent early following burn injury and resulted in part from a higher independent risk of multiple organ failure in females. The question remains why the current study differs from prior literature. The dataset was a prospective cohort with attempts to control post burn care using standard

operating procedures. It may be that the homogeneity of the care allowed these differences to be demonstrated in a relatively small cohort of patients. The results also demonstrated a dose-dependent effect in the strength of the gender dimorphism with burn size (TBSA%). The greater risk of multiple organ failure in females and the early separation of the survival curves suggest that differences in the early inflammatory response may be responsible. It may be that the competence of the immune response in the pediatric group may in part play a role and result in these gender based outcome difference following burn injury.

This study has several limitations. The current study is a secondary analysis of the Host Response to Injury burn cohort and was not designed to address the specific questions in this analysis. Although the current dataset was collected prospectively and standardized protocols were in place, there is likely variation in care practices across burn centers that could impact our results. We attempted to control for important differences in burn injury severity and other risk factors for poor outcome and investigated any potential bias across different burn centers. Despite our regression models having high predictive characteristics, there remains the possibility that we were unable to control for important confounders. Enrolling centers for the cohort study included multiple adult burn centers and a pediatric burn center. The immune response following burn injury across different age groups may be variable and may bias the current results. Importantly, the inclusion for the cohort study for pediatric patients was a burn size greater than 40%. Although we attempted to adjust for this fact in the age stratified analysis, this different inclusion criterion may be responsible in part for the results found in this group. The relatively small sample size of the cohort, as compared to prior literature, and the spectrum of ages may also limit the applicability of the current conclusions formulated. Finally, despite attempts at controlling confounders in the model, there were differences in burn injury mechanism across the gender groups overall which may play a role in our results and conclusions. Importantly, when we focused our comparison on burn mechanism in the pediatric age group alone, these differences in burn mechanism did not reach statistical significance (p=0.136).

In conclusion, the current results suggest that gender based outcome differences may be different following thermal injury as compared traumatic injury and that the gender dimorphism may be exaggerated in patients with higher burn size and in those in the pediatric age group, with female gender being associated with poor outcome. These gender based mortality differences occur early and may be a result of a higher risk of organ failure and early differences in the inflammatory response following burn injury. Further investigation is required to thoroughly characterize the mechanisms responsible for these divergent outcomes so that the morbidity and mortality associated with burn injury can ultimately be reduced.

Acknowledgments

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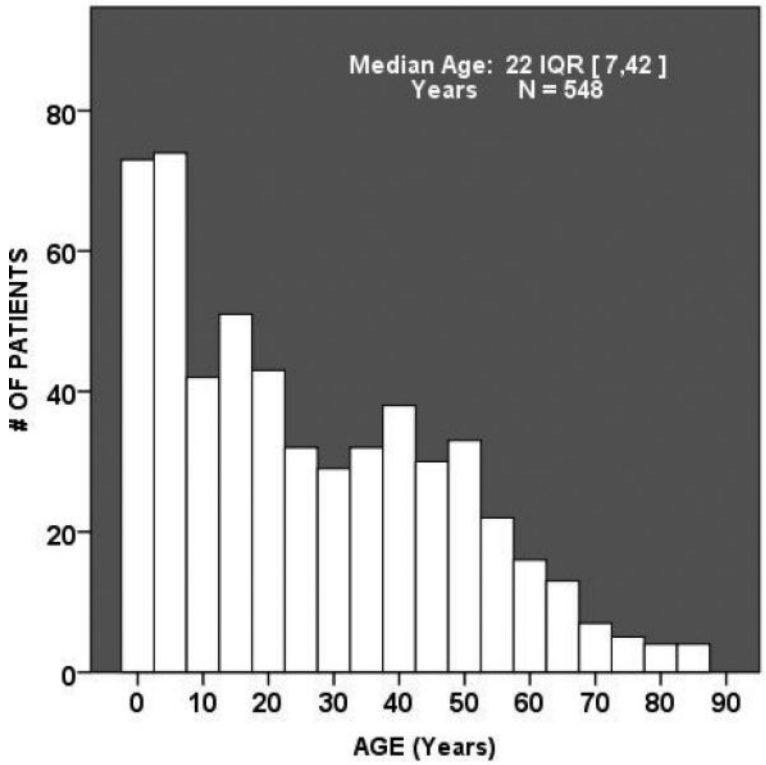

Figure 1.
Histogram demonstrating age of study cohort

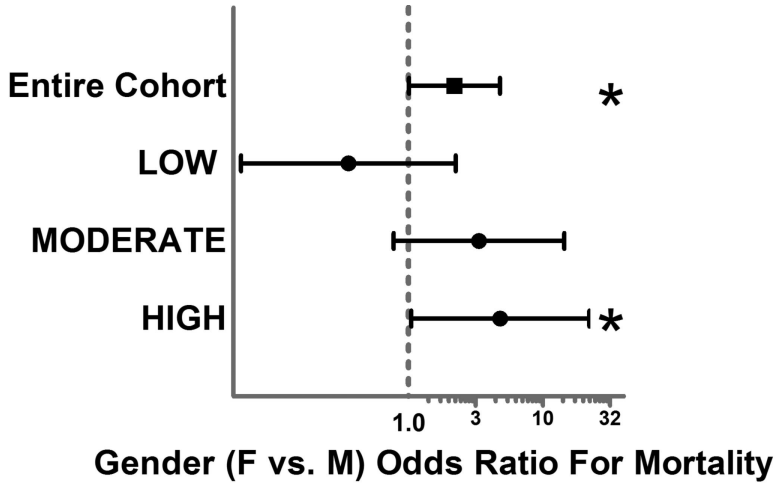

Figure 2.

Forest-Plot depicting the independent gender (female vs. male) odds ratio for mortality stratified by burn size (LOW-20% and ≤ 40%, MODERATE- >40% and ≤ 60% TBSA, HIGH- >60% TBSA).

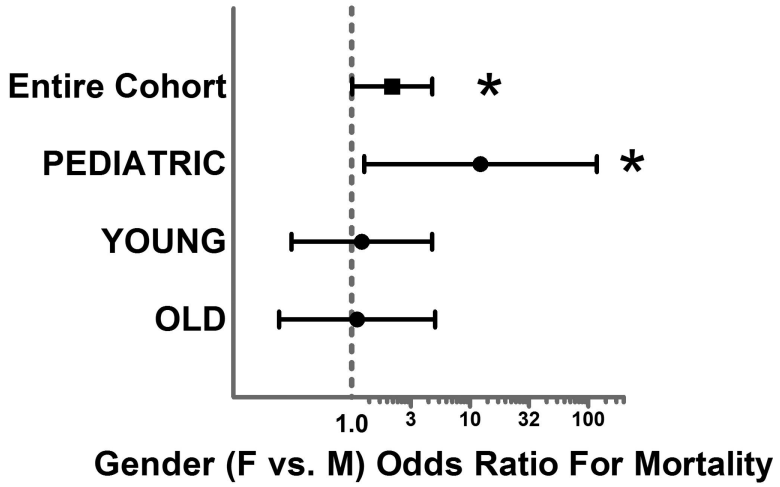

Figure 3.

Forest-Plot depicting the independent gender (female vs. male) odds ratio for mortality stratified by age group (PEDIATRIC 1-14 years, YOUNG 15-50 Years, OLD > 50 years).

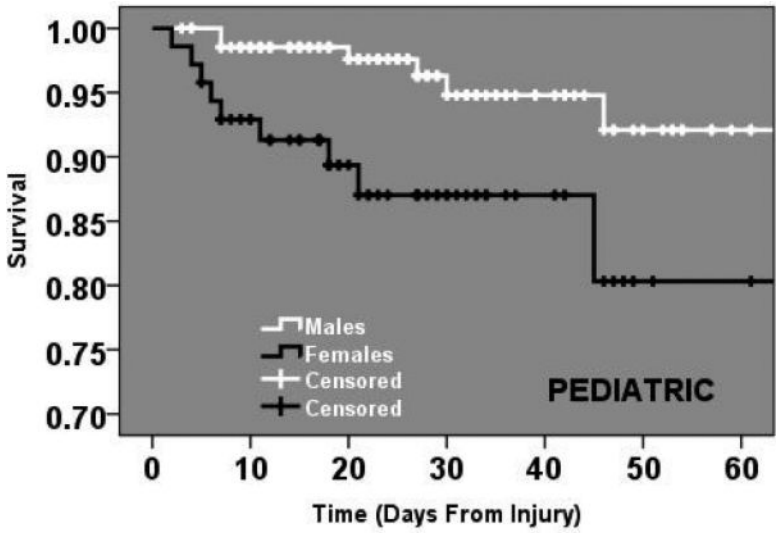

Figure 4.
Cox-hazard survival curves comparing male and female PEDIATRIC patients.

Table 1

Demographic and burn injury characteristic comparison across gender.

	Females (n=155)	Males (n=393)	p-value
Age	24.7±23	26.7±20	0.073
Burn Size (TBSA%)	48.0±19	48.8±20	0.686
Burn Mechanism			
Flame	77.4%	81.2%	
Flash	1.3%	5.9%	0.010*
Scald	18.1%	9.2%	
Other	3.2%	3.8%	
Inhalational Injury (%)	46.1%	47.1%	0.826
Body Mass Index (kg/m^2)	24.2±9	24.0±7	0.347
Presenting Base Deficit (meq/L)	−6.3±5	−5.5±5	0.141
Lowest GCS (first 24hrs)	9.8±5	10.2±5	0.386
Lowest MAP (first 24hrs, mmHg)	66±15	69±17	0.034*
APACHE II score	10.2±5	9.8±5	0.418
First 24hr crystalloid (Liters)	11.9±9	14.2±10	0.064
2nd Degree Burns (%)	18.1%	19.8%	0.228
3rd Degree Burns (%)	40.4%	38.3%	0.329
Pre-Admission Comorbidities (%)	58.1%	52.9%	0.277
Pre-Admission Medications (%)	65.8 %	70.4%	0.294
Hospital Length of Stay (days)	37.7±33	40.1±35	0.379
ICU Days	322±32	34.6±35	0.371
Unadjusted Mortality (%)	18.7%	13.3%	0.106
Unadjusted Rate of MOF (%)	27.3%	27.1%	0.969
Unadjusted Rate of NI (%)	71.4%	69.1%	0.599

Table 2

Burn characteristics compared across age groups (PEDIATRIC 1-14 years, YOUNG 15-50 years, OLD > 50).

	PEDIATRIC (n=210)	YOUNG (n=254)	OLD (n=84)	p-value
Burn Size (TBSA%)	58.3±17	43.8±19	38.7±17	<0.001
3rd Degree Burns (%)	47.8±24	34.2±21	29.4±18	<0.001
APACHE II score	15.5±9	19.2±9	23.1±8	<0.001
Presenting Base Deficit (meq/L)	−6.5±−9	−5.3±5	−4.5±4	0.003
First 24hr crystalloid (Liters)	4.4±3	16.9±10	15.4±8	<0.001
Mortality (%)	8.1%	13.0%	36.9%	<0.001