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ORIGINAL RESEARCH

Factors contributing to death of major trauma victims with haemorrhage: A retrospective case–control study

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Abstract

Objective: To identify factors associated with death secondary to haemorrhage following major trauma.
Methods: A retrospective case–control study was conducted on data from adult major trauma patients attending Christchurch Hospital ED between 1 June 2016 and 1 June 2020. Cases (those who died due to haemorrhage or multiple organ failure [MOF]), were matched to controls (those who survived) in a 1:5 ratio from the Canterbury District Health Board major trauma database. A multivariate analysis was used to identify potential risk factors for death due to haemorrhage.
Results: One thousand, five hundred and forty major trauma patients were admitted to Christchurch Hospital or died in ED during the study period. Of them, 140 (9.1%) died from any cause, most attributed to a central nervous system cause of death; 19 (1.2%) died from haemorrhage or MOF. After controlling for age and injury severity, having a lower temperature on arrival in ED was a significant modifiable risk factor for death. Additionally, intubation prior to hospital, increased base deficit, lower initial haemoglobin and lower Glasgow Coma Scale were risk factors associated with death.
Conclusions: The present study reaffirms previous literature that lower body temperature on presentation to hospital is a significant potentially modifiable variable in predicting death following major trauma. Further studies should investigate whether all pre-hospital services have key performance indicators (KPIs) for temperature management, and causes for failure to reach these. Our findings should promote development and tracking of such KPIs where they do not already exist.

Key words: *death, emergency departments, haemorrhage, temperature, trauma.*

Key findings

- 1.2% of major trauma patients in this tertiary hospital died from haemorrhage or multi-organ failure.
- Intubation prior to hospital, increased base deficit, lower initial haemoglobin and lower Glasgow Coma Scale were associated with death.
- Lower body temperature on presentation to hospital is associated with death in trauma patients with major haemorrhage, which may be potentially modifiable.

Introduction

Trauma is a major cause of morbidity and mortality worldwide.[1] In New Zealand (NZ), major trauma is defined as individuals with an Injury Severity Score (ISS) >12 or death due to trauma.[2] The ISS is an anatomical scoring system that provides an overall score for patients with multiple injuries, the highest score being 75.[3] Each year in Canterbury (a large region in NZ), approximately five individuals die due to haemorrhage or a secondary cause such as multiple organ failure (MOF), after major trauma. It is not clear why these individuals die, when others with a similar degree of injury survive.

Previous literature has identified several potential patient-related, hospital-related and trauma-related factors that may be associated with death secondary to haemorrhage. These include patient use of anticoagulant medication, results of early blood tests, time to operative management and use of tranexamic acid.[4–8] Importantly, major trauma deaths may not necessarily be directly due to haemorrhage, but rather as a result of consequences of massive blood loss which can lead to

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MOF.[9] In these cases, death would be expected to be delayed, occurring once individuals have been admitted to hospital or are in intensive care.

This project aimed to identify any patient- or process-related factors that may be associated with death secondary to haemorrhage or MOF following major trauma at a single tertiary hospital in NZ.

Methods

Study design

A retrospective case–control study was conducted on data collected from adult patients who were admitted to Christchurch Hospital or died in Christchurch Hospital ED after major trauma. The STROBE guidelines for reporting observational studies were adhered to.[10]

Setting

Christchurch Hospital is a tertiary level hospital in the South Island of NZ, which covers a region with a population of approximately 550 000. The Christchurch Hospital ED is the only major acute referral centre, and only major trauma centre in the region. With over 110 000 ED attendances and more than 400 major trauma admissions per year, Christchurch Hospital is one of the 10 busiest trauma centres in Australasia.[11]

Participants

Patients with major trauma (ISS ≥13, or death due to trauma) aged 18 years or over, recorded in the Canterbury District Health Board (CDHB) Major Trauma database between 1 June 2016 and 1 June 2020 were considered eligible to be included in the study. Those with an isolated head injury, death prior to arrival in Christchurch Hospital ED, or those who attended another hospital prior to attending Christchurch Hospital ED were excluded. 'Cases' were defined as those who met the inclusion criteria and died in hospital of major haemorrhage or MOF.

Controls were defined as those major trauma patients who suffered injuries with the potential for major haemorrhage but who survived. Patients were included in the list of potential controls if they were noted to have any of: haemothorax or lung laceration, medium or large vessel injury, abdominal organ injury, pelvic ring fracture, femoral fracture, or haemorrhage with blood loss ≥20%. Any patient without any of these injuries were excluded. Controls were randomly selected in a 5:1 ratio to cases from the finalised potential control list using Microsoft Excel,[12] matched by year of injury.

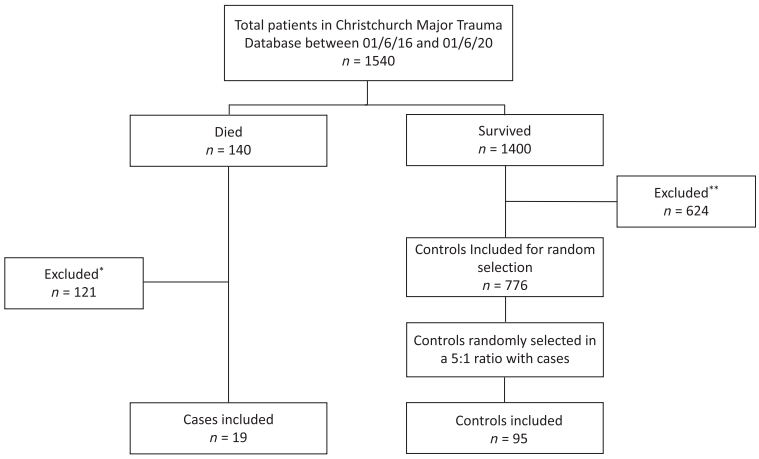

Figure 1. *Case and control selection flow diagram. *Cause of death not haemorrhage or multiple organ failure, or Christchurch Hospital ED not primary treatment site, or age <18 years. **Did not meet injury criteria, or Christchurch Hospital ED not primary treatment site, or age <18 years.*

TABLE 1. *Basic demographics and Injury Severity Score of cases and controls*

Variable	Cases ($n = 19$) n (%) or mean (standard deviation)	Controls ($n = 95$) n (%) or mean (standard deviation)	Univariate *P*-value
Age			
18–64	10 (52.6%)	77 (81.1%)	0.015†
65+	9 (47.4%)	18 (18.9%)	
Sex			
Female	8 (42.1%)	26 (27.4%)	0.200
Male	11 (57.9%)	69 (72.6%)	
Ethnicity			
NZ European	15 (78.9%)	62 (65.3%)	0.813‡
Māori	0 (0.0%)	6 (6.3%)	
Asian	1 (5.3%)	10 (10.5%)	
Pacific peoples	0 (0.0%)	1 (1.1%)	
Other	3 (15.8%)	16 (16.8%)	
Injury Severity Score	38.37 (21.85)	18.14 (6.16)	0.001§

†Calculated using Fisher's exact test. ‡Calculated using Fisher–Freeman–Halton exact test. §Levene's Test for Equality of Variances <0.05 – equal variances not assumed.

TABLE 2. *Categorical variables*

Variable	Cases ($n = 19$) n (%)	Controls ($n = 95$) n (%)	Univariate P-value	Multivariate OR and 95% CI†
Primary injury cause				
Vehicle-related	12 (63.2%)	55 (57.9%)	0.227‡	
Fall	5 (26.3%)	20 (21.1%)		
Sport	0 (0.0%)	12 (12.6%)		
Assault	2 (10.5%)	3 (3.2%)		
Other	0 (0.0%)	5 (5.3%)		
Accidental *versus* inflicted				
Accidental	16 (84.2%)	89 (93.7%)	0.171§	
Inflicted	3 (15.8%)	6 (6.3%)		
Primary injury type				
Blunt	17 (89.5%)	92 (96.8%)	0.193§	
Penetrating	2 (10.5%)	3 (3.2%)		
Mode of transport from scene				
Helicopter ambulance	7 (36.8%)	16 (16.8%)	0.126‡	
Road ambulance	11 (57.9%)	71 (74.7%)		
Police/prison vehicle/private vehicle/taxi/walk in	1 (5.3%)	8 (8.4%)		
ED arrival time				
0800–1559	10 (52.6%)	42 (44.2%)	0.469	
1600–2359	8 (42.1%)	38 (40.0%)		
0000–0759	1 (5.3%)	15 (15.8%)		
Trauma team activation				
No	4 (21.1%)	40 (42.1%)	0.085	1.238 (0.239–6.410)
Yes	15 (78.9%)	55 (57.9%)		
Post-ED destination				
Death in ED	7 (36.8%)	0 (0.0%)	<0.001‡	
HDU	2 (10.5%)	42 (44.2%)		
ICU	8 (42.1%)	11 (11.6%)		
Operating room	0 (0.0%)	1 (1.1%)		
Ward	2 (10.5%)	39 (41.1%)		
Home	0 (0.0%)	2 (2.1%)		
Triage status ED				
Triage 1	14 (73.7%)	0 (0.0%)	<0.001‡	
Triage 2	1 (5.3%)	28 (29.5%)		
Triage 3	2 (10.5%)	33 (34.7%)		
Triage 4	2 (10.5%)	29 (30.5%)		
Triage 5	0 (0.0%)	5 (5.3%)		
Intubation location				
Not intubated	6 (31.6%)	87 (91.6%)	<0.001‡	1 (reference)
Hospital	7 (36.8%)	5 (5.3%)		0.076 (0.009–0.654)
Pre-hospital	6 (31.6%)	3 (3.2%)		0.029 (0.003–0.334)

(*Continues*)

TABLE 2. *Continued*

Variable	Cases ($n = 19$) n (%)	Controls ($n = 95$) n (%)	Univariate P-value	Multivariate OR and 95% CI†
Blood ethanol level				
0	8 (42.1%)	24 (25.3%)	0.248	
>0	2 (10.5%)	7 (7.4%)		
Not taken	9 (47.4%)	64 (67.4%)		
MTP activated				
No	9 (47.4%)	93 (97.9%)	<0.001§	0.039 (0.004–0.420)
Yes	10 (52.6%)	2 (2.1%)		
Anticoagulant therapy				
No	19 (100%)	94 (98.9%)	1.000§	
Warfarin	0 (0.0%)	1 (1.1%)		
Reversed		1 (100%)		
Antiplatelet therapy				
No	15 (78.9%)	87 (91.6%)	0.113§	
Yes	4 (21.1%)	8 (8.4%)		
First dose TXA given				
No	8 (42.1%)	76 (80.0%)	0.001	0.896 (0.185–4.337)
Yes	11 (57.9%)	19 (20.0%)		
TXA infusion given				
No	18 (94.7%)	92 (96.8%)	0.523§	
Yes	1 (5.3%)	3 (3.2%)		
Minimum two doses TXA given				
No	16 (84.2%)	92 (96.8%)	0.057§	2.041 (0.131–32.258)
Yes	3 (15.8%)	3 (3.2%)		

†Note that all multivariate ORs control for age (coded) and Injury Severity Score. ORs significantly more than 1 indicate greater likelihood of survival and ORs less than 1 indicate greater likelihood of death after controlling for age and Injury Severity Score. ‡Calculated using Fisher–Freeman–Halton exact test. §Calculated using Fisher's exact test. CI, confidence interval; HDU, high dependency unit; MTP, massive transfusion protocol; OR, odds ratio; TXA, tranexamic acid.

Data sources

Patients were identified from the Christchurch data in the NZ National Trauma Registry (NTR). Data were extracted from the NTR as well as individual patient electronic and paper medical records. Variables included patient demographics, mechanism of injury and injuries sustained, and in-hospital clinical observations, investigations and management.

Statistical methods

Using Statistical Package for Social Sciences,[13] univariate analysis was carried out for those variables identified as most clinically relevant. For the continuous variables, t-tests for independent means were used. For the categorical variables, chi-squared tests were performed, with Fisher's exact tests being used where appropriate (i.e. when more than 20% of cells had expected frequencies <5). Those with a P-value of less than 0.1 in univariate analyses were included in a multivariate analysis, controlling for age and ISS. This yielded odds ratios (ORs) and 95% confidence intervals (CIs) for both continuous and categorical variables.

Ethical approval

Approval for the study was granted by the University of Otago Ethics Committee (HD20/105) and the CDHB (RO #20238). Māori consultation was undertaken with both the University of Otago, Christchurch, and CDHB Te Komiti Whakarite.

Results

Figure 1 shows the selection process of cases and controls.

Baseline demographic data of the two groups are shown in Table 1. Those who died were more likely to

be aged 65 years and older ($P = 0.015$). Sex ($P = 0.200$) and ethnicity ($P = 0.813$) were similar between the two groups. ISS was seen to be markedly different between the two groups, hence the reason for including this in the multivariate analysis.

Table 2 shows the difference between the two groups for data collected on categorical variables. Accidental injury accounted for the majority of injuries in both the cases (84.2%) and controls (93.7%), however a larger proportion of non-survivors sustained inflicted injury (15.8%) compared to the survivors (6.3%). For non-survivors, a larger proportion of injuries were penetrating (10.5%) compared to the survivors (3.2%). However, the absolute numbers were similar ($n = 2$ and $n = 3$, respectively).

Among non-survivors, 68.4% were intubated at some point, compared to 8.5% of the survivors. Intubation location was significantly different between the case and control groups ($P < 0.001$). For 97.9% of the survivors, the massive transfusion protocol (MTP) was not activated, *versus* for 52.6% of the non-survivors ($P < 0.001$). There was a significant difference between the cases and controls with regards to whether they received an initial dose of tranexamic acid (TXA) ($P = 0.001$). For non-survivors, 57.9% received an initial dose of TXA while only 20% of survivors received this. For non-survivors, 94.7% did not receive a TXA infusion (second dose of TXA). Likewise, 96.8% of survivors did not receive the same. Only 15.8% of non-survivors and 3.2% of survivors received a minimum of two doses of intravenous TXA.

After performing a multivariate analysis controlling for age and ISS, a higher odds of death was found for those who were intubated, and this was greater if they were intubated before arriving at hospital.

TABLE 3. *Continuous variables*

Variable	Cases		Controls		Univariate *P*-value	Multivariate OR and 95% CI†
	n	Mean (standard deviation)	*n*	Mean (standard deviation)		
Time periods						
Time from injury to ED arrival (min)	19	126.26 (120.26)	91	138.85 (173.13)	0.764	N/A
Total time in ED (min)	19	124.16 (149.75)	95	315.06 (137.26)	<0.001	1.01 (1.003–1.018)
Time from ED arrival to index CT scan (min)	11	121.55 (70.63)	87	160.24 (162.25)	0.438	N/A
Total hospital days	19	9.05 (14.70)	95	9.15 (9.14)	0.979‡	N/A
Total ICU days	19	5.21 (12.07)	95	1.41 (4.99)	0.193‡	N/A
Admission to death (hours)	19	205.50 (357.25)	0	N/A	N/A	N/A
Observations						
Estimated MAP in ED (mmHg)	14	74.31 (24.92)	95	94.99 (16.96)	<0.001	1.036 (0.989–1.086)
Initial temperature value in ED (°C)	14	35.25 (1.02)	89	36.36 (0.81)	<0.001	3.354 (1.249–9.004)
Pulse rate in ED (bpm)	18	85.61 (26.08)	95	83.27 (20.41)	0.671	N/A
Initial GCS total in ED	18	7.78 (5.71)	95	14.49 (1.66)	<0.001‡	1.676 (1.232–2.279)
Investigations						
Initial base deficit (mmol/L)	16	−11.37 (10.39)	43	−1.02 (4.13)	0.001‡	1.192 (1.015–1.400)
Initial INR	15	1.37 (0.33)	65	1.09 (0.27)	0.007‡	0.328 (0.040–2.694)
Initial Hb in ED (g/L)	17	108.76 (23.52)	95	137.63 (17.77)	<0.001	1.047 (1.008–1.088)
Interventions						
Litres of crystalloid given in first 24 h post-injury	19	1.86 (2.12)	95	1.72 (2.13)	0.792	N/A

†Note that all multivariate ORs control for age (coded) and Injury Severity Score except for Injury Severity Score which only controls for age. ORs significantly more than 1 indicate greater likelihood of survival and ORs less than 1 indicate greater likelihood of death after controlling for age and Injury Severity Score. ‡Levene's Test for Equality of Variances <0.05 – equal variances not assumed. CI, confidence interval; CT, computed tomography; GCS, Glasgow Coma Scale; ICU, intensive care unit; INR, international normalized ratio; MAP, mean arterial pressure; OR, odds ratio.

Additionally, after controlling for age and ISS, there was a higher odds of death if the MTP was activated (OR 0.04, 95% CI 0.004–0.4). There was no significant difference in odds of death if the trauma team was activated, if a first dose of TXA was given, or if a minimum of two doses TXA were given.

Table 3 shows the difference between the two groups for continuous variables. Non-survivors spent a significantly shorter time in ED than survivors ($P < 0.001$), and this remained significant when controlled for age and ISS. Non-survivors had a lower temperature ($P < 0.001$), base deficit ($P < 0.001$), haemoglobin ($P < 0.001$) and Glasgow Coma Scale (GCS) ($P < 0.001$) on arrival than survivors, and these remained significant after controlling for age and ISS. In univariate analysis, estimated mean arterial pressure was lower in non-survivors ($P < 0.001$) and international normalized ratio was higher ($P = 0.007$), however, these were not significant after controlling for age and ISS.

After controlling for age and ISS, longer time in ED, higher arrival temperature, haemoglobin, and GCS, and lower base deficit were associated with a greater odds of survival.

Using both Pearson ($r = -0.05$, $P = 0.61$) and Spearman (rho = 0.074, $P = 0.47$) correlation coefficients, there was no association between temperature and time elapsed from injury to the ED arrival. Additionally, when the four seasons were compared using 1 way ANOVA, there was no difference in initial temperature based on season of injury ($P = 0.52$).

Figure 2 shows the distribution of time from admission to death. There is a peak of deaths within the first 24 h of injury, and then a second smaller peak at greater than 20 days after injury. For those who died within 1 day of injury, 7 of 10 (70%) were those who died of haemorrhage. For those who died after 20 days post injury, 3 of 3 (100%) died of MOF. For those who died between 1 and 20 days from injury, 5 of 6 (83%) died of MOF, and 17% died of haemorrhage. Appendix S1 details location and cause of death of the individuals studied.

Discussion

When reflecting on potential factors that contribute to death in a setting of major trauma, it is important to note that some factors, such as those pertaining to the patient or injury mechanism, are not easily modifiable. In contrast, others are more easily modifiable, and it is these factors that can be targeted to improve outcomes.

After controlling for age and ISS, the present study found that temperature on arrival in ED was a significant and potentially modifiable variable in predicting death. Other significant variables in the present study included intubation location, base deficit, haemoglobin and GCS.

The 'triad of death' has widely been discussed in trauma literature and is described as the combination of hypothermia, acidosis and coagulopathy. These factors are known to form a vicious cycle consequently increasing trauma mortality.[14–16] The present study's findings regarding temperature and base deficit reiterate the importance of optimising these factors when able. The present study showed that those who died from major trauma had significantly lower body temperature on arrival in ED compared to those who survived. This suggests that in the prehospital setting, there may be an opportunity to intervene and provide methods to maintain body temperature shortly after trauma, which may improve outcomes. It is, however, important to appreciate the challenge in maintaining body temperature in a prehospital setting, particularly when the primary focus may be on haemostatic control of major wounds, or initial airway management.[17] With this in mind, the environmental temperature is an important factor influencing body temperature. However, we found no difference between the groups' arrival temperature based on season of injury, despite a similar time from injury to the ED arrival between the groups. This suggests the trauma and haemorrhage, as well as subsequent interventions prior to hospital arrival, may contribute to the hypothermia as much or more than the environmental exposure. However, this was a small study, with some temperature data missing, hence this finding may be due to insufficient statistical power.

In keeping with previous literature, the present study found that a greater base deficit was associated with increased mortality in trauma patients. In bleeding patients, hypothermia, acidosis/base deficit and hypocalcaemia affect haemostasis which in turn leads

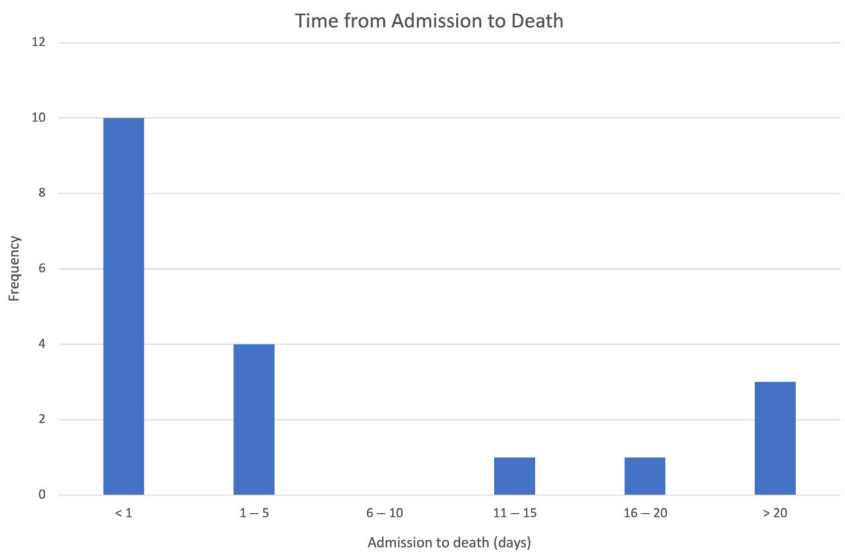

Figure 2. *Time from admission to death. The number of people (frequency) is shown on the* y*-axis, and time from admission to death (days) is shown on the* x*-axis.*

to an increase in mortality. A 2017 study by Ross *et al.* found that pH was the greatest predictor of mortality in trauma as those with severe acidosis (pH <7.0) were six times more likely to die compared to those with pH 7.0 or greater.[18] Another 2020 study by Corwin *et al.* found that trauma patients who died had a base deficit of >−8. They also showed that mortality was significantly higher in those with pH <7.2 while mortality in patients with pH 7.2–7.35 and patients with pH >7.35 was comparable.[19] Given the above findings, it would be appropriate to conclude that correction of acid–base disturbance should be a vital component of trauma management. That said, an exact cut-off for pH for improvement of trauma mortality is still unknown, therefore further studies could consider focusing on this aspect.

While we did not find that administration of TXA (either a single dose or multiple doses) was a significant variable predicting death in our study, it was interesting to note that most patients did not receive two doses in the recommended fashion (initial IV bolus and then infusion). Previous data from the CRASH-2 trial published by Roberts *et al.* showed that receiving an initial intravenous bolus of 1 g TXA, followed by infusion over 8 h reduced mortality in major trauma, if administered within 3 h of injury.[20] One possible reason why the proportion of non-survivors receiving a TXA infusion is low is that seven people (36.8%) died in ED, and six of these were within 1 h, precluding sufficient time to administer the infusion.

As the present study investigated death due to both haemorrhage and MOF, we expected there would be some people who died shortly after injury (those with haemorrhage), and some who died much later after injury (those with MOF). Trunkey's 1983 classification of immediate, early, and late trauma deaths describes a 'trimodal' distribution of deaths. This described immediate death due to unsurvivable injuries within minutes, early death due to severe injuries within hours, and late death due to MOF and sepsis within weeks.[21] A 2010 study by Gunst *et al.* argued that in areas with well-established trauma systems the original 'trimodal' distribution has shifted to a 'bimodal' distribution, with peaks in the immediate and early stages, and marked reduction in late deaths.[22] Likewise, a NZ-based 2004 study by Pang *et al.* showed that there was no evidence of a trimodal distribution of trauma deaths, and that most deaths (80.6%) occurred in a prehospital setting with a gradual decrease thereafter.[23] These findings differ from our findings of a bimodal distribution with peaks in the early (<1 day) and late (>20 days) stages.

Limitations

The retrospective and observational nature of the present study incurs standard limitations in data collection and interpretation. There are sufficient major trauma cases within the studied period in Christchurch to allow for the primary statistical analysis, however the number of cases in some of the subgroup analyses is small which may limit power. There may have been omissions in documentation for certain variables, for example whether TXA was given, temperature and other physical recordings. In addition, the omission of certain documentation meant that certain subgroup analyses had low numbers, which may have impacted power. A further limitation is that the cause of death documented was subjective rather than established by autopsy, meaning what was recorded may not be the true cause of death.

Conclusions

In summary, our study emphasises that lower body temperature on arrival to the ED is a significant potentially modifiable variable associated with death due to haemorrhage or MOF following major trauma. We are aware that key performance indicators (KPIs) exist in pre-hospital settings to optimise body temperature. We propose further studies to determine whether all pre-hospital services have such KPIs, and to ascertain causes for failure to reach these. In instances where such KPIs do not yet exist, we feel our findings are important to promote development and tracking of KPIs for temperature management, in order to reduce mortality.

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Competing interests

None declared.

Data availability statement

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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Supporting information

Additional supporting information may be found in the online version of this article at the publisher's web site:

Appendix S1. Location and cause of death.