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Traumatic lacerations: what are the risks for infection and has the `golden period' of laceration care disappeared?

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Abstract

Objective—To determine risk factors associated with infection and traumatic lacerations and to see if a relationship exists between infection and time to wound closure after injury.

Methods—Consecutive patients presenting with traumatic lacerations at three diverse emergency departments were prospectively enrolled and 27 variables were collected at the time of treatment. Patients were followed for 30 days to determine the development of a wound infection and desire for scar revision.

Results—2663 patients completed follow-up and 69 (2.6%, 95% CI 2.0% to 3.3%) developed infection. Infected wounds were more likely to receive a worse cosmetic rating and more likely to be considered for scar revision (RR 2.6, 95% CI 1.7 to 3.9). People with diabetes (RR 2.70, 95% CI 1.1 to 6.5), lower extremity lacerations (RR 4.1, 95% CI 2.5 to 6.8), contaminated lacerations (RR 2.0, 95% CI 1.2 to 3.4) and lacerations greater than 5 cm (RR 2.9, 95% CI 1.6 to 5.2) were more likely to develop an infection. There were no differences in the infection rates for lacerations closed before 3% (95% CI 2.3% to 3.8%) or after 1.2% (95% CI 0.03% to 6.4%) 12 h.

Conclusions—Diabetes, wound contamination, length greater than 5 cm and location on the lower extremity are important risk factors for wound infection. Time from injury to wound closure is not as important as previously thought. Improvements in irrigation and decontamination over the past 30 years may have led to this change in outcome.

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Competing interests None.

Ethics approval Each institutional review board of the three participating hospitals approved the study without the need for written informed consent.

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INTRODUCTION

Background/importance

Traumatic wounds are one of the most common reasons people seek medical care in the USA. More than 6 million lacerations are treated each year in emergency departments (ED) at an estimated cost of US$3 billion, with complications from these wounds resulting in significant additional costs.[1]

The infection rate of lacerations treated in ED is likely to be between 2% and 5%.[2, 3] This small rate of infection makes it common practice not routinely to treat traumatic lacerations with prophylactic antibiotics.[4–7] Cost models have suggested that it is only cost effective to treat wounds at high risk when there is a greater than 5% chance of infection.[8] However, there are no clear guidelines on which wounds are `high risk'. Wounds that become infected are also likely to require closer follow-up and potential scar revision.

Goals of this investigation

In this multicentre prospective cohort study we sought to determine the infection rate of lacerations treated in ED and whether a subset of high-risk wounds could be identified and considered as candidates for prophylactic antibiotics and or closer follow-up. Finally, we also carried out a detailed investigation of the relationship between infection and time to repair after injury in order to determine whether prolonged time from injury to closure is truly a risk for increased infection.[9, 10]

METHODS

Study design

This was a multicentre prospective cohort study of patients with lacerations who were treated in three different ED. Each institutional review board of the three participating hospitals approved the study without the need for written informed consent.

Patients and setting

Patients with lacerations presenting from 1 February 2008 to 1 September 2009 to the ED of the three participating hospitals were considered. The participating hospitals were a level 1 trauma centre, a community non-teaching hospital and a city teaching hospital. Any laceration resulting from a human or animal bite was excluded.

Data collection

A structured data form was designed before the study and reviewed with the treating physicians. The form was implemented as part of the documentation requirements at the institutions to ensure all forms were completed on all patients. The data form collected 27 specific patient, laceration and treatment variables and was completed by the treating physician as part of the documentation of the patient's ED visit. Subjective variables were given explicit definitions for the physicians at the time of treatment. For example, with regard to contamination; wounds were classified on the data collection sheet as `clean' (less than 6 h old and occurred in a clean environment or with a clean instrument), or contamination was characterised as `moderate' (wound >6 h, non-clean environment or instrument), or `heavy' (greater than 12 h and/or grossly contaminated and/or with foreign body present). In two hospitals the data from the form was exported from the electronic medical record into a database for follow-up, and in one of the hospitals data were manually transcribed from a paper record.

Outcomes

Thirty days after their initial treatment, patients were contacted by phone and a structured interview was conducted to determine whether the patient had had an infection. Patients were considered to have an infection if they were seen by a physician for a wound infection and treated with oral and/or intravenous antibiotics. Patients were also asked their opinion of the cosmetic outcome of their wound. They did this by rating it on a 100-point scale, with 0 being the worst scar and 100 being the best possible scar. They were also asked if they would consider having their scar revised.

Statistical analysis

We performed univariate analysis on patient, laceration and treatment variables using parametric and non-parametric techniques as appropriate. We report selected risk ratios (RR) and 95% CI. When appropriate we also combined related variables and dichotomised variables to determine significance cut points, and considered them for entry into a direct multivariate logistic regression model based on univariate statistical significance. When variables were combined or clinically related we used the most significant variable for entry. The Hosmer–Lemeshow goodness of fit test was used to determine the model fit. Sixty outcomes would allow sufficient power to consider up to four variables in our multivariate model.[11] Assuming a baseline infection rate of 2–2.5% we estimated that we would need to enrol and complete follow-up on 2500–3000 lacerations. Data were analysed using Stata V. 12.

RESULTS

Data collection forms were completed on 3957 patients with lacerations. The study enrolment rates were 110 patients per month at the trauma centre, 57 patients per month at the community hospital and 58 patients per month at the urban teaching hospital. Infection rates did not differ between centres. Complete outcome assessment was obtained on 2663 patients of whom 69 (2.6%, 95% CI 2.0% to 3.3%) had an infection (table 1).

Wounds that became infected were more likely to receive a worse cosmetic rating at 30-day phone follow-up (70 vs 87 on a 100-point scale, difference 17, 95% CI 12 to 21). Patients with infection were also more likely to consider scar revision (24.6% vs 9.6%, RR 2.6, 95% CI 1.7 to 3.9). There was no statistically significant association of infection with age, sex, race, or tetanus status. Diabetes was recorded in 75 patients and five (6.7%) of them developed infections, compared with a 2.5% infection rate in patients without diabetes (RR 2.70, 95% CI 1.1 to 6.5). Only six patients reported diabetes with complications and two (33%) of them developed infection (RR 13.1, 95% CI 4.1 to 41.5). The length of laceration was greater for patients who developed infections (table 2).

Of 195 patients with lacerations greater than 5 cm long, 13 (6.7%) developed infections (RR 2.9, 95% CI 1.6 to 5.2). Infection rates were not different between sharp mechanisms versus blunt/crush mechanisms. Lacerations located on the head or neck were less likely to become infected compared with lacerations on the torso or an extremity, especially a lower extremity in which the infection rate was 7.6%. Contamination or tissue trauma requiring debridement was also associated with an increased risk of infection. Consistent with their longer lacerations, patients who developed wound infections also had longer closure times and greater suture numbers (table 3).

Overall, 64.3% of lacerations were closed with sutures and 23.8% with tissue adhesive (Dermabond), sterile strips (Steri-strips), or both. The remaining lacerations were either scalp lacerations closed with staples (7.4%) or lacerations that were not closed at all (4.4%). Stapled scalp lacerations had a low infection rate of 1% (2/198, 95% CI 0.1% to 3.6%).

Wounds closed with tissue adhesive, sterile strips, or both had an infection rate of 2.0% (95% CI 1.0% to 3.5%), while sutured lacerations had an infection rate of 3.0% (95% CI 2.2% to 3.9%). Sutured lacerations that included deep stitches (15.4% of all sutured lacerations) had a similar infection rate as sutured lacerations that did not include deep stitches.

Two thousand three hundred and forty-two patients with complete follow-up had documented time of injury. The average time from injury tended to be shorter in patients with infection (table 4). Only 85 patients presented 12 or more hours after injury, and only one of them (1.2%) developed an infection. However, 15.3% (13 of 85) were treated without initial closure compared with only 3.6% of wounds less than 12 h old ($p<0.0001$). Details of the 13 lacerations not closed are included in table 4. Furthermore, lacerations closed after 12 h were more likely to be on the extremities, and there were no differences in the rates of prophylactic antibiotic use, type of repair, sex of the patient, length of the wound or cosmetic outcome.

After combining and determining cut points for variables with univariate significance, we were able to develop a model considering diabetes, laceration length greater than 5 cm, location and contamination demonstrating them as independent significant risk factors for infection. The model and associated OR are described in table 5.

DISCUSSION

In this prospective multicentre study we established that a history of diabetes, wound contamination, length greater than 5 cm and location are important risk factors for wound infection. Patients with these risk factors are `high risk' in that their risk is greater than 5%, and according to costs models should be considered for prophylactic antibiotics and/or closer follow-up. Surprisingly, we found that no true `golden period' of laceration repair exists. In particular, we found no association between the time from injury to wound closure and the development of infection, nor did we find any difference in the type or method of closure and infection.

In a single site study of all traumatic wounds including bite wounds, Hollander *et al*[12] described a higher infection rate of 3.5% and found that age, diabetes, wound size, contamination with foreign material and a non-head or neck location are important risk factors for infection. The study did not perform a detailed analysis of time from injury, and the rate of follow-up was unclear. They used a combination of direct and phone follow-up and only 46% of patients completed direct follow-up compared with close to 70% in our study. We believe our study is the largest multicentre prospective cohort study of consecutive lacerations and we ensured all data were collected at the time of treatment in a similar manner by using the same structured data form at all sites. We completed our study by doing phone follow-up in a quality assurance manner, and we were able to conduct the study without written informed consent and maximise our follow-up. To be categorised as having a wound infection, the patient had to report being treated by a physician with systemic antibiotics for a wound infection. Physician determination of infection drives treatment costs and is also a Centers for Disease Control and Prevention criterion for a surgical site wound infection.[13] Finally, we considered lacerations treated at three different institutions (level 1 trauma centre, academic city teaching hospital and suburban community hospital) and found no differences in infection rates between our sites, and have no reason to believe our study is not generalisable to all ED within developed countries.

Prophylactic antibiotics are controversial and are likely to have a place in the management of high-risk lacerations.[14, 15] In one large study with high baseline infection rates there

appeared to be a benefit to prophylactic antibiotic use.[16] Numerous small studies with a low baseline risk of infection have found no benefit to treating `routine' lacerations with prophylactic antibiotics.[4–6, 17–21] Despite being underpowered these studies all show a trend towards some minimal benefit with antibiotic prophylaxis.[6, 22] A cost model showed that an absolute reduction of 2–3% in infection rates is likely to be cost effective when considering prophylactic antibiotics to prevent wound infections.[8] It appears reasonable to assume that prophylactic antibiotics could provide this modest decrease in infection and be of benefit in `high-risk' lacerations.[8] In this study we identified factors associated with increased risk resulting in a 5–10% risk probably justifying consideration of prophylactic antibiotics or at least closer follow-up.

We found that the time between injury and closure had no association with infection. Most wounds require primary closure, which results in more rapid healing, less patient discomfort and better cosmetic outcomes than secondary closure. Contaminated wound studies were the basis for the determination that bacterial colonisation and the time interval a laceration is opened before closure is directly related to infection.[23, 24] This resulted in the `golden period' for safe laceration closure, which is variable and dependent on site.[25] However, the clinical literature on this is old and controversial.[26] A study from 1980 on prophylactic antibiotics for hand lacerations found a difference in infection rates of 7% versus 20% for lacerations closed before and after 4 h.[27] That older study did not outline decontamination methods and the infection rates earlier than 4 h are very high compared to other studies. A 1990 paediatric study found no difference in paediatric lacerations closed before and after 6 h.[28] In a study of wounds presenting in a delayed fashion to a Jamaican hospital, Berk and colleagues[9] found facial lacerations healed without increased infection regardless of the time of closure while non-facial lacerations did best if closed 19 h before injury. The subgroups and numbers in that study were very small. Our study has a comparatively large number of lacerations older than 12 h on a variety of sites, and we found that the concept of a `golden period' no longer exists. We believe it has probably been eliminated by improved wound decontamination that routinely occurs more frequently and with better equipment than may have occurred 25–30 years ago.[7, 10, 26, 29–31]

CONCLUSIONS

Our study identified high-risk wounds that may benefit from prophylactic antibiotics and/or better follow-up. We also believe this study should change the practice of those who do not close wounds because of a delay in the time of presentation.

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Table 1

Demographic characteristics

	Infection (N=69)	No infection (N=2594)	Infection rate 2.6% (95% CI 2.0% to 3.3%)	p Value
Age in years (SD)	36.6 (25.2)	34.1 (25.0)		0.42
Sex				0.36
Male	55%	61%	2.4%	
Female	45%	39%	2.9%	
Race				0.77
White	62%	59%	2.7%	
Asian/Pacific islander	13%	13%	2.7%	
African American	4.3%	4.2%	2.7%	
Diabetes				
Yes	5 (7.2%)	70 (2.7%)	6.7%	0.04
			RR 2.7, 95% CI 1.1 to 6.5	
With complications	2 (2.9%)	4 (0.2%)	33.3%	0.009
			RR 13.1, 95% CI 4.1 to 41.5	
Would consider scar revision	17 (24.6%)	250 (9.6%)		<0.001
	(RR 2.6, 95% CI 1.7 to 3.9)			
Cosmetic rating (SD)*	70.2 (29.8)	86.8 (17.5)		<0.0001
	Difference 17, 95% CI 12 to 21			

RR, risk ratio.

*Self-rated by patient on 100-point scale during 30-day phone interview.

Table 2

Wound characteristics

	Infection (N=69)	No infection (N=2594)	Infection rate (2.6%)	p Value
Hours from injury to presentation (SD)	2.4 (1.9)	3.0 (4.9)		0.39
	Difference −0.6, 95% CI −1.8 to 0.6			
Length in cm (SD)	3.5 (3.2)	2.5 (2.2)		0.005
	Difference 1.0, 95% CI 0.5 to 1.5			
Length >5 cm				0.001
Yes	13	182	6.7%	
No	56	2396	2.3%	
			RR 2.9, 95% CI 1.6 to 5.2	
Shape				0.03
Linear	36	1657	2.1%	
Irregular/stellate	31	834	3.6%	
Mechanism				0.085
Sharp, incised	29	1180	2.4%	
Blunt, crush	26	1121	2.3%	
Shattered glass	3	98	3.0%	
Other/unknown	11	195	5.3%	
Location				<0.001
Scalp	5	340	1.4%	
Face	9	783	1.1%	
Ear	0	15	0.0%	
Lips	2	91	2.2%	
Arm	6	134	4.3%	
Leg	17	174	8.9%	
Hand	17	581	2.8%	
Foot	4	82	4.7%	
Other/unknown	9	394	2.2%	
Location on lower extremity				<0.0001
Yes	21	256	7.6%	
No	43	2279	1.9%	
			RR 4.1, 95% CI 2.5 to 6.8	
Contamination				0.01
Contaminated	18	378	4.5%	
Clean	51	2179	2.3%	
			RR 2.0, 95% CI 1.2 to 3.4	
Tissue trauma (requiring debridement)				0.04
Yes	19	453	4.0%	

	Infection (N=69)	No infection (N=2594)	Infection rate (2.6%)	p Value
No	44	1866	2.3%	
			RR 1.7, 95%	
			CI 1.03 to 3.0	

Table 3

Treatment characteristics

	Infection (N=69)	No infection (N=2594)	Infection rate (2.6%)	p Value
Mean time of wound closure (minutes)				
For sutured wounds only	24.6	18.1		0.04
				0.48
Repair type				
Simple	49	1987	2.4%	
Intermediate	8	226	3.4%	
Complex	3	117	2.5%	
Closure method				
				0.3
0 None	3	114	2.6%	
1 Sutures	51	1593	3.1%	
2 Tissue adhesive	6	359	1.6%	
3 Steri-strips	4	190	2.1%	
4 Staples	2	196	1.0%	
5 Adhesive + strips	3	73	3.9%	
6 Sutures + (strips or adhesive)	0	69	0.0%	
				0.26
Non-suture closure (2+3+5)	13	622	2.0%	
Sutured (1+6)	51	1662	3.0%	
				N/A
Deep closure	8	255	3.0%	
No deep closure	43	1407	3.0%	
No of sutures per wound				
	7.1	5.5		0.024
Sutured wounds				
≥6 h post-injury	5	130	3.7%	
<6 h post-injury	43	1400	3.0%	

Table 4

Lacerations repaired before and after 12 h from injury

	Injury <12 h (N=2176)	Injury >12 h (N=72)	p Value
Age, years	34	41	0.02
Male sex	61%	58%	0.42
Infection (95% CI)	2.9% (2.3% to 3.8%)	1.4% (0.3% to 6.4%)	0.75
Hours from injury (median IQR)	2 (1–3)	16 (12–24)	
Length of wound (mean cm)	2.6	2.4	0.31
Location			<0.001
Head and neck	55%	31%	
Extremity/torso	45%	69%	
Repair type			0.96
Simple	82%	86%	
Intermediate	9%	4%	
Complex	4%	4%	
Other	5%	6%	
Prophylactic antibiotics	2.2%	2.9%	0.93
Cosmetic outcome 100 mm VAS	86	85	0.67
Considering scar revision	10%	5%	0.40

13/85 Wounds left open after 12 h (72 closed and analysed) 81/2257 (2176 closed and analysed). Four of 13 include delayed primary closure left open up to 96 h before closure. Nine left open.

VAS, visual analogue scale.

Table 5

Multivariate model for predictors of infection

	OR	95% CI for OR
Non-head and neck location	2.5	1.4 to 4.5
Diabetes	3.1	1.2 to 8.0
>5 cm in length	2.4	1.4 to 4.0
Heavy or moderate contamination	1.9	1.04 to 3.3

Hosmer and Lemeshow test p=0.19.