ELSEVIER

Burns 31 (2005) 981–985

BURNS

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On the use of prophylactic antibiotics in prevention of toxic shock syndrome

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Accepted 26 June 2005

Abstract

No consensus exists among burn surgeons on the role of prophylactic antibiotics in prevention of toxic shock syndrome (TSS). We recently reported a series of 71 children admitted with burns to our burn unit. By Centres for Disease Control (CDC) criteria, six of these were 'definite' and four 'probable' cases of TSS. Prior to this report, none of our patients were given prophylactic antibiotics. Thereafter, prophylactic therapy was included in the management of children admitted to the burns unit. The aim of this study was to assess whether prophylaxis with a one off single dose of systemic antibiotics prevented the occurrence of TSS.

Data were collected prospectively between 1 January and 31 December 2001, on all children admitted to the burns unit. Out of 50 children admitted to the burns unit, 39 received prophylactic antibiotics in the referring accident and emergency. Two of these became unwell but none fulfilled the CDC criteria. The remaining 11 patients were given antibiotics on admission out of which one child required direct admission to the intensive care unit with a working diagnosis of TSS. Retrospectively, his features did not conform to the CDC criteria. In conclusion, this study suggests that prophylaxis may prevent TSS in children.

Keywords: Toxic shock syndrome; Prophylaxis

1. Introduction

Toxic shock syndrome (TSS) is a rare and life threatening illness. The term 'toxic shock syndrome' was introduced by Todd et al. when they presented a series of seven children who developed a condition characterized by high fever, shock, vomiting, diarrhoea, conjunctival hyperaemia, scarletiniform rash, disseminated intravascular coagulation and multi-system dysfunction [1]. One child died and those who survived developed desquamation of palms and soles during the convalescence period. It was also postulated that the features were due to an exotoxin produced by *Staphylococcus aureus* (*S. aureus*) that was biochemically, pathologically and immunologically distinct from staphylococcal exfoliatin. Similar features were later recognized in patients with several other conditions, more notably in young females using tampons [2]. Thereafter, in 1980, the Centres for Disease Control (CDC) collected the available data and devised six criteria to define TSS (Table 1) [3]. Patients that met four features were thought to be probable cases while patients fulfilling all six criteria were considered definite cases of TSS. Bergdoll et al. and Schlievert et al., simultaneously yet separately, reported the unique protein exotoxin of TSS-associated isolates [4,5]. The former group named their toxin the staphylococcal enterotoxin F (SEF) while the later group called it the pyrogenic exotoxin C (PEC). At a symposium held on TSS in 1984 in Madison, Wisconsin, it was decided in the view of the work done by Bonventre et al. that both of these were identical and renamed the toxin as the toxic shock syndrome toxin-1 or TSST-1 [6].

In 1985, the first series of TSS in burned children, comprising seven patients, was reported by Frame et al. from St. Andrews Hospital, Billericay, UK [7]. Since then it is now established that younger children are more predisposed to acquire this illness as they have low levels of antibodies against the toxin, which only become sufficient to confer adequate immunity later on in childhood

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0305-4179/$30.00
doi:10.1016/j.burns.2005.06.017

[8–10]. Also children that acquire TSS typically have small un-infected burns and develop the symptoms a few days after the burn has occurred [7,11,12].

Current literature suggests that *S. aureus* is the most common microorganism to colonise burn wounds [13,14]. Research has also shown that in majority of cases TSS is caused by *S. aureus*, while other bacteria such as streptococcus and pseudomonas have also been implicated [10,15]. Approximately 20% strains of staphylococci produce TSST-1 [16]. Similarly, some strains of methicillin resistant *S. aureus* (MRSA) can also produce TSST-1. Although TSST-1 is the most prevalent exotoxin associated with TSS, other exotoxins produced by *S. aureus* commonly implicated with staphylococcal food poisoning (enterotoxins A–E) can also cause TSS [17]. Mortality reported from TSS in paediatric burns ranges from 11 to 50% [7,18]. Nevertheless, there remains no agreement among burn surgeons regarding the role of prophylactic antibiotics in the prevention of TSS in children [19,20].

The Royal Belfast Hospital for Sick Children (RBHSC) provides the regional service for thermally injured children for the whole of Northern Ireland. We recently reported a series of 71 children admitted to the unit over a period of 15 months [21]. Out of these, 13 children became acutely unwell. When the CDC criteria were applied on these patients, six were found to be definite cases of TSS while four were probable cases. At that time, prophylactic antibiotics were not given to any of our patients. The results of that study were also presented at the annual meetings of the European Burn Association and the British Burn Association in the year 2000. It was suggested to us at both meetings to give prophylactic antibiotics to all new admissions and study the group prospectively. In the year 2001, a prophylactic antibiotic protocol was introduced in the unit. The objective of the present study was to assess whether the alteration in our practice prevented the occurrence of toxic shock syndrome in children with thermal injuries.

2. Methods

All referring accident and emergency (A&E) departments in the province were instructed at the time of referral, to administer one dose of systemic antibiotics to the child. Data were collected prospectively on all children admitted with burns to the RBHSC between 1 January and 31 December 2001. Data included, age, sex, size of burn as percentage of total body surface area (TBSA, %), type of thermal injury (burn/scald), type of dressing and topical agent applied, antibiotic used for prophylaxis and information related to the case definition of TSS provided by the CDC, i.e. presence of fever, rash, desquamation, hypotension, organ failure and microbiology results (Table 1) [3].

3. Results

In total, 50 patients were admitted to the unit. Out of these, 32 (64%) were between the ages of 1 and 4 years (Fig. 1). There were 31 males and 19 females. The mean size of burn was 5.7% TBSA $\pm$ 5.6 (range = 0.25–24 and median = 4.5). There were 33 children with scalds while 17 sustained burns.

The wound management policy of our unit remained relatively the same as that in our previous study [21]. Paraffin tulle was used as the main dressing for all burns, while facial burns were largely treated with exposure method (Table 2). In partial thickness burns, the paraffin tulle was impregnated with povidone iodine while for full thickness burns a thick layer of 1% silver sulphadiazine cream was used until the burn was excised and grafted (Table 3). These dressings were reenforced with several layers of gauze and cotton wool and secured with crepe bandage. For superficial burns or those treated with exposure method, an ointment containing polymixin B sulphate 10,000 units and bacitracin zinc 500 units/g was applied to keep them moist.

Table 1
Toxic shock syndrome case definition (all, definite and four, probable)

1. Temperature $>$ 39.2 °C
2. Diffuse macular rash
3. Desquamation of skin 1–2 weeks after onset of illness (typically palms and soles)
4. Hypotension (systolic blood pressure $<$ fifth centile by age for children $<$ 16 years)

5. Involvement of three or more of the following organ systems:
   A. Gastrointestinal (vomiting or diarrhoea at onset of illness)
   B. Muscular (severe myalgia or creatine phosphokinase level $>2 \times$ ULN)
   C. Hyperaemia of conjunctivae and mucous membranes of the oropharynx or vagina
   D. Renal (BUN/Cr $>2 \times$ ULN or >5 WBC per high power fields in absence of UTI)
   E. Hepatic (total bilirubin, AST or APT $> 2 \times$ ULN)
   F. Haematological (platelets $< 100 \times 10^{-9}$/l)
   G. Central nervous system (disorientation or alteration in consciousness without focal neurological signs when fever and hypotension are absent)

6. Negative results on the following tests, if obtained:
   A. Blood, throat or CSF cultures
   B. Serological tests for rocky mountain spotted fever, leptospirosis or measles

ULN, upper limit of normal; BUN, blood urea nitrogen; Cr, serum creatinine; UTI, urinary tract infection.