

Staphylococcus aureus Toxic Shock Syndrome

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Received date: June 24, 2015, Accepted date: June 26, 2015, Published date: June 28, 2015

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Editorial

Toxic shock syndrome (TSS), caused by toxic shock syndrome toxin-1 (TSST-1) and type B enterotoxins produced by *Staphylococcus aureus* [1,2]. The disease (TSS) was initially described in 1978 and came to public attention in 1980 with the occurrence of a series of menstrual-associated cases and at least 50% of non-menstrual cases. TSST-1 is a pyrogenic toxin superantigen produced by *Staphylococcus aureus* [3,4]. TSST-1 super antigen activate a vast number of T cells in a T cell receptor B chain V β -selective manner in direct association with major histocompatibility complex class II molecules on antigen-presenting cells especially V β 2+ T cells [5-7].

TSS symptoms will be manifested within 8-12 hours after infection, the symptoms include fever, low blood pressure, rash, malaise and confusion, diarrhea and inability to maintain proper hemostasis. Severe cases often progress to multiple-organ involvement and desquamation of the skin over the entire body, some cases end in death [8].

Menstrual TSS is usually related to tampon use which increases the vaginal concentration of oxygen that stimulates TSST-1 production by *S. aureus* normally present in the vagina. High-absorbency tampons also sequester magnesium ions, which causes nutrient depletion in the vagina and may simulate late log-phase conditions for resident *S. aureus*, inducing TSST-1 secretion [9]. Menstrual toxic shock syndrome can be prevented by avoiding the use of highly absorbent tampons, changing tampons more frequently and using tampons during menstruation only (not regularly). Non-menstrual TSS was reported in postoperative patients [10], postpartum women [11], patients with skin and bone infections [12], patients with respiratory infections [13], and patients with small burns [14,15].

Treatments of TSS include cleaning of wounds and remove any foreign bodies, beta-lactamase-resistant antistaphylococcal antibiotics should be administered intravenously to patients with staphylococcal infections and IV fluids administration [15]. The usually prescribed antibiotics are nafcillin, oxacillin, and first generation cephalosporin. Recently, researches are directed to development of either monoclonal antibodies against TSST-1 or other peptides to block the ability of bacterial toxins to activate T cells, therefore blocking the toxicity cascade [16]. Diagnosis of toxic shock syndrome can be made by detection of toxin -1 by high performance liquid chromatography [17], flow cytometry [18], reversed passive latex agglutination, enzyme linked immunosorbent assay (ELISA) and immunoblot [19]. Davis and Fuller [20] reported that *S. aureus*, *S. pneumonia*, enterococci, and groups A and B streptococci can be identified in about 2.5 h using a commercially available DNA probe kit that utilizes hybridization protection assay technology [20]. Feng et al., [21] reported that vaccination with plasmid DNA encoding a mutant toxic shock syndrome toxin-1 ameliorates toxin-induced lethal shock in mice. The mice were intranasally immunized with the plasmid DNA (named

pcDNA-mTSST-1) plus a mucosal adjuvant, a non-toxic mutant labile toxin (mLT).

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