CLINICAL CONDITION OF THE FACIAL-JAW AND NECK ABSCESS

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Abstract

In some bacterial infections, the immune system cannot destroy the invading pathogen. In these cases, the invading pathogen is successful in creating a favorable environment for survival and continuation in the host organism. (1) effective violation of the immune response; and (2) protection against immune cells and molecules. S. aureus releases several proteins that contain coagulases and toxins that trigger abscess formation and stagnation. If staphylococcal abscesses are not surgically drained and treated with antibiotics, the spread infection and septicemia will lead to death. In this regard, this document develops a simple mathematical model of abscess formation, which includes characteristics that we consider important for abscess formation. Our goal is to create a mathematical model that reproduces some of the characteristics and behaviors observed in the process of abscess formation. No clinical trials have been conducted to guide the surgeon in the optimal technique of drainage of the Backgroundpilonidal abscess. The purpose of our study was to check whether the location of the incision affects wound healing.The methods of selectronic recordings from the surgical database at our 200-bed District General Hospital were examined from January 2003 to February 2010 for surgical techniques (midline and lateral) for patients with incision and drainage for acute pilonidal abscess. These patients were admitted from the emergency department with a pilonidal abscess, underwent operative drainage and returned for observation. The main outcome measure was the time to heal the injury.Results 243 pilonidal abscess drained, with 134 lateral and 74 middle linear incisions. All patients underwent a simple longitudinal incision. No patient has undergone roofing, marsupialization or closure. 48 patients with midline drainage who returned for observation matched the results of gender, age and microbiology culture with patients who had undergone lateral drainage. Almost all are drained under general anesthesia, on average 1 day after surgery. The total length of the observation was the same in both groups. . Untreated abscesses have been observed for the same

period of time, regardless of the type of incision. Abscesses treated after cutting the middle line lasted about 3 weeks longer than those drained through the lateral incision .

Keywords: Abscess formation, fibrin network, partial differential equation, computational modeling

Objective

This article developed computational models based on differential equations, capable of reproducing some of the features observed in the process of abscess formation. The study consisted of analyzing the spatial-temporal behavior of bacteria, coagulation factors fibrin, toxins and neutrophils. These analyses were important and helped to understand how modeled processes interact, the effects of the inclusion of specific processes, among other factors. This article shows that the use of the G function in conditions of population growth and distribution is one of the characteristics that allow us to reproduce some of the main aspects of the abscess formation process in mathematical models . Another important feature was the formation of the fibrin network. The Fibrin network protected bacteria from the immune response given by neutrophils. The formation of the fibrin network has been modeled taking into account the production of coagulation factors and the interaction of these factors with the bacterial colony. More tests and model improvements may be needed, but this early model was able to replicate some of the features found in the abscess, such as: the formation of a fibrin network around bacterial colonies and the accumulation of necrotic neutrophils and living neutrophils. abscess area. Based on the results of the simulation and the analyzes carried out so far, we believe that the fibrin network is important for the continuation of bacteria in the abscess lesion, along with the mechanisms used to produce toxins that bacteria use to kill neutrophils and to avoid phagocytosis.

MATERIALS & METHODS

$$\partial u \partial t = fg + D \nabla \cdot (g \nabla u)$$

$$u(x,0) = u0, \partial u(.,t) \partial n \rightarrow || \partial \bar{O} = 0, \quad (1)$$

where u is a variable that refers to a given population, the term f is a function that models the growth of u and the term $D\nabla \cdot (g\nabla u)$ models the nonlinear diffusion of u. Function g is equivalent to the g function proposed in (Painter and Sherratt, 2003). This function was originally developed to model the movement of interacting cell populations (Painter and Sherratt, 2003). We extended it to model interactions that also occur in other cellular processes. For example, we use the *g* function to model interactions that occur during bacterial growth or neutrophil migration. The *g* function is used to account for different interaction strengths between the populations and the effects of these in processes of growth, phagocytosis, migration, death and diffusion.

The *g* function is defined as the heaviside function of $\overline{g}$ �‾:

$g(w)=\{\overline{g}(w),\ 0\le\overline{g}(w)\le 1\ 0,\ \ \text{otherwise}.$

Function $\overline{g}(w)$�‾(�) is defined as:

where *w* is a term that models the interactions between distinct populations and *total* is a parameter that denotes the maximum population supported in a discretized region of the domain. In this work, we consider that the value of *total* is constant and is equal to 1 for all discretized regions.

The interactions between the populations can be stimulatory or inhibitory. In this paper, we consider only inhibitory interactions in the *w* term. To illustrate the meaning of *w*, consider, for example, a system with two types of populations: *u* and *v*. The interactions that each population has with the other one are modeled by the *w* term. Therefore, the *w* term is defined for each distinct population in the system. For example, the *w* for the *u* population is defined as:

$wu=wuuu+wvuv$

where w_{uu} *u* is the inhibition that *u* exerts on itself and w_{vu} *v* is the inhibition that *v* exerts on *u*. These inhibitory relations will affect all processes in *u* dynamics. w_{uu} and w_{vu} are constant parameters. We call these parameters "weights" to refer to the fact that they control the strength of the inhibition that one population exerts on the other.

The $\overline{g}$ �‾ function for the *u* population is

$\overline{g}(wu)=1-wu$

$wv=wvvv+wu,vu$

RESULTS & DISCUSSION

In this article, we have built a mathematical model of abscess formation in stages. Interactions between Model components . It should be noted that the intensity of a certain inhibitory relationship depends on the concentration of cellular species that carry out inhibition.

Conclusions

In this article, they developed computational models based on differential equations.They were able to reproduce some of the characteristics observed in the process of abscess formation.The study consisted of analyzing the spatial-temporal behavior of bacteria, coagulation factors, fibrin, toxins and neutrophils. These analyses are important and help to understand how modeled processes interact, the effects of the inclusion of specific processes, among other factors.This article shows that the use of the G function in conditions of population growth and distribution is one of the characteristics that allow us to reproduce some of the main aspects of the abscess formation process in mathematical models . Another important feature was the formation of the fibrin network. The Fibrin network protected bacteria from the immune response given by neutrophils. The formation of the fibrin network has been modeled taking into account the production of coagulation factors and the interaction of these factors with the bacterial colony. More tests and model improvements may be needed, but this early model was able to replicate some of the features found in the abscess,Based on the results of the simulation and the analysis carried out so far, it turns out that the fibrin network is important for the continuation of bacteria in the abscess lesion along with the mechanisms used to produce toxins that bacteria use to kill neutrophils and to avoid phagocytosis.

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