Chapter 1

Introductory Chapter: Surgical Infections

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1. Introduction

Surgical infections are infectious diseases that can be treated with surgical procedures or occur in the surgical site. Synthetically, these are a localized, closed infectious disease. In the first group, the autonomous infectious pathologies of single organs or closed sites, as abscesses, appendicitis, cholecystitis, colonic diverticulitis, etc., are included. However, in the other group, there are the surgical site infections, surgical wound infections, etc. It is important to remind that the post-surgical infections can develop as local disease or as general disease with startup of systemic inflammatory response syndrome (SIRS) and then sepsis, severe sepsis, and septic shock. The surgical site and wound infections can come from the external environment or from an endogenous contamination. The infections from external environment, hospital ward, operating room, and surgical equipment, are controlled and resolved by sterilization procedures. Very crucial is the role of perioperative nurses, which should control and save the sterile techniques, detect the occurred breaks, and communicate actively to all team members. Less easily the contaminations from endogenous infective agents, pathologic aerobes and anaerobes, that are present, as commensals, in the digestive, biliary and urinary tract, airways, etc., can be controlled. In the perspective of infective risk, surgical procedures have been subdivided into four types. This classification enables proper risk stratification of occurrence of infective complications [1]. Obviously, this risk is not only connected with the environmental or endogenous sources of contamination involved during surgical procedures, but is conditioned by the general conditions of patients, characteristics of occurred intraoperative contamination, etc. [2]. The surgical procedures are classified as following: class I clean intervention—during these procedures, there is no opening of the lumen of intestinal, urinary, respiratory, genital tract; also there is no treatment for inflamed tissues or septic outbreaks. Among these interventions, there are abdominal parietal hernias, thyroid and breast surgery, exploratory abdominal surgery, etc. Class II clean-contaminated intervention—in this class, interventions during which the opening of digestive, urinary, and respiratory tract is scheduled, with checked normal situation and without uncommon contamination are collected. We

can list in this the following: group biliary tract, urinary tract, gynecological surgery, appendectomy, etc. Class III contaminated intervention—this section encompasses the procedures with prolonged opening of digestive, biliary, and urinary tract, especially with major leak of intestinal or biliary content. Also there is the presence and treatment of inflamed sites. Usually large bowel surgery should be inserted in this class. Class IV dirty-infected intervention—this includes all surgical procedures for acute peritonitis, with septic collections, pus, and fecal contamination: ultimately all the pathologies with severe septic contamination to be treated by surgery [3].

2. Pathogenesis

In the clinical scenario of the surgical infections, some factors are in evidence: infectious agents, the patient's immune defenses, and finally, but most important, the physiopathological characteristics of the site of infections, for example, the type of site perfusion. The infections agents are bacteria, virus, fungi, etc. Their involvement can develop as contamination from outside the body or with the assumption of active pathogenetic function of endogenous infective agents and development of disease. The list of possible infectious agents is very long and varied. Aerobic bacteria are steadily on the skin. *Staphylococcus aureus* is always present in wounds infections. *Streptococcus* penetrates usually in the skin's lesions, fractures, and interests connective tissue [4], following which the bacteria invasion of the connective tissue develops a complex vascular, lymphatic, and local tissue reaction which is defined as inflammation. This is a basic reaction to injury is caused by a foreign and deleterious agent and is intended to locate and destroy it. When inflammation is caused by viable agent (bacteria, virus, etc.), it may be considered as the physical basis of infectious process. The morphological picture of inflammation can suggest that inflammation is a relatively static phenomenon. The viable agents in contact with the tissue will cause an inflammatory reaction of various degree of severity, from hyperemia to serious suppurative process. The first step of inflammation process is the alteration of local fluid exchange by an increase of capillary permeability. There is an immunological action of inflammation process, with the purpose to limit the bacteria, firstly by the initial increase of capillary permeability: with the increased passage of plasma protein, there is the accumulation of fibrinogen in the site of lesion, the formation of fibrous network, and occlusion of draining lymphatics by trombi. In this way, the site of inflammation is confined and limited. The celerity and effectiveness of the process of boundary are very important in the control of diffusion of the pathological microorganisms [5]. The staphylococcus is a very damaging agent but in turn causes rapid local fixation and poor dissemination. On the contrary the hemolytic *Streptococcus*, with a local bland action, is consequently more invasive. The role of inflammation in immunity is a control in bacterial invasiveness. Anaerobic microorganisms are more frequently identified in surgical infections. The important pathologic anaerobes with clinical role are *Clostridium*, *Bacteroides*, *Fusobacterium*, *Peptostreptococcus*. All these bacteria are commensals and therefore the origin of anaerobic infections is endogenous; especially in the colon, the anaerobic flora is largely prevalent. We have to add, for its great diffusion, also the *Escherichia coli*, which is anaerobic/aerobic. In fact, *Escherichia coli*, an enteric microorganism, and other enterococci are often detected together with anaerobes in the surgical infections. The most frequent anaerobic surgical infections are the complications of

abdominal surgery, as wound infections after large bowel and gynecological surgery, and intra-abdominal septic collections especially caused by anastomotic leakage. The characteristics of anaerobic infections are the presence of putrid exudate, feculent odor, and gas production [6]. In the immunosuppressed patients, the role of opportunistic bacteria Pseudomonas and Serratia is preeminent, which are external surface contaminants, but usually nonpathogenic. A particular problem is the possible peripheral dissemination of bacteria in case of contaminated wounds. *Streptococcus* bacteria release around the infected site speedily. On the contrary, *Escherichia coli* and *Staphylococcus* are more slower. In this septic scenario, the surgical action of the debridement in the infected wounds is in evidence. With debridement, all devitalized tissues from the site are removed. This action is important because the phagocytic activity of neutrophils in the site of inflammation is more efficacious in reducing the bacteria dissemination if the devitalized tissues have been removed [7]. Also fungi, yeasts, and parasites (*Echinococcus, Amoeba*) can cause infestations; sometime, these develop in the septic collections, abscesses, which require the surgical procedures. The tuberculous infections, usually treated with pharmacological therapy, may be treated by surgery in cases of drug-resistant forms, sequelae of pulmonary tuberculosis, pulmonary aspergilloma, nonfunctioning tuberculous kidney, etc.

3. Host's defense mechanisms

The autonomous infectious diseases of single organs and the surgical site infections, surgical wound infections, in each class of risk, are affected in their clinical evolution by some predisposing conditions. Defective or missing control of external contaminations and imperfect check of intraoperative contaminations have been previously considered. In this scenario, the state and the condition of host's defense mechanisms are certainly crucial. The control of environmental source of contamination can be obtained by strict observance of sterilization procedures of ward, operating room, surgical equipment and devices, etc. To avoid or minimize the risk of intraoperative contaminations, it is decisive to follow the specified well-known procedures for each surgical intervention. More complex is the evaluation of host's defense mechanisms. The first obstacle to infections is the integrity of anatomical barriers: the skin and mucous membranes. Beside anatomical barriers, the immunity system is at the heart of defense mechanisms. Usually, the immunity in the scientific treaties is subdivided into nonspecific and specific immunity. The nonspecific immunity is based on the phagocytic activity of reticuloendothelial system which encompasses distributed phagocytes in the various organs: circulating monocytes and macrophages, polymorphonuclear granulocytes, neutrophils, connective tissue and mucosa mast cells, Kupffer cells in the liver, etc. The phagocytes incorporate pathogenic microorganisms, foreign materials, and cellular debris and destroy them. The macrophages also transport the antigen to the lymph nodes where this stimulates the lymphocytes. The antibodies, secreted by B lymphocytes and bound to particles, favor the recognition of the latter by phagocytes. These accessory cells play a predominant role in killing parasites and in controlling inflammatory processes. The mast cells and basophils contain various molecules which are mediators of inflammation. Consequently, they are very important in the correlations between immune responses and inflammatory reactions [8]. The specific immunity

synthetically develops through some phases: the exposition to an antigen (foreign body with antigenic capacity, such as bacteria, viruses, etc.) and afterward recognition and processing of antigen by macrophages, entered into action of T and B lymphocytes, and subsequently, synthesis of specific antibody. The impairment, also in a specific phase, of this multifactorial process causes the global alteration of the immunity functions, the immunodeficiency, which can concur in increased severity of surgical infections. The lymphocytes play a central role in the control of immune response. They specifically recognize antigens distinguishing them from the body's own components. There are two lines of lymphocytes: B cells that produce antibodies and T cells that have various functions—assist B cells in the production of antibodies, recognize and destroy infected virus cells, activate phagocytes for the destruction of pathogens, and check the level and quality of the immune response [9]. Synthetically the inflammation is the response of a tissue to a damage and is meant to bring serum molecules and immune system cells to the damaged site. The flogosis encompasses local increase in blood perfusion and vasodilation, increased capillary permeability, and cells migration from blood vessels to tissues. In this process, some phases develop, as vasodilation, tissue oozing, exudation, marginalization, diapedesis, and chemotaxis; the latter can be defined as movement of cells in response to chemoattractive molecules. These acute phase proteins are various: PCR, interferons, interleukin, etc. Then, phagocytosis follows: the cells incorporate particles and microorganisms. The phases of phagocytosis are the following: adhesion to phagocytes of particles through nonspecific receptors or through opsonization by antibodies and/or complement and adhesion by receptors for Fc, C3b, and C3bi; then, phagosome formation, fusion of lysosome (damage and digestion), and release of microbial products [10].

4. Systemic inflammatory response syndrome: clinical evolution of sepsis

Sepsis is a clinical syndrome initiated by immune system and coagulation, caused by the presence of bacterial or viral infection. Severe sepsis can be defined as organ dysfunction or tissue hypoperfusion due to sepsis, requiring intensive therapy. Septic shock is a severe condition characterized by hemodynamic instability, hypotension, and despite the adequate infusion of fluids, as evolution of organs dysfunction and sepsis. Most frequent causes of severe sepsis are pulmonary infections, intestinal perforations, and urinary and skin infections. Severe sepsis requires the diagnostic quick recognition and starting treatment in the early stages. It can be briefly stated that sepsis and its worsening evolutions are the result of systemic inflammatory response, resulting from an infection (systemic inflammatory response syndrome, SIRS). The diagnosis of sepsis requires at least two of the following signs: body temperature more than 38°C or less than 36°C, heart rate more than 90 bm, breath frequency more than 20 bm, and white blood cells more than 12,000 or less than 4000. Keep in mind that SIRS can be triggered not only by infection but also by numerous other factors as trauma, burns, pancreatitis, etc. However, not all infections cause sepsis, which is conditioned by the body's inflammatory response. In fact there is different degree of inflammatory response to infection and therefore it is necessary to distinguish the infection accompanied by physiological response of

the organism, such as fever for small localized infections, from infections accompanied by an abnormal and exaggerated, therefore negative, inflammatory systemic response (SIRS), with start of organ dysfunction [11]. The sequential-sepsis-related organ failure assessment (SOFA) score allows to quantitatively evaluate organ damage. The assessment of the progressive alterations of the clinical function indexes of six organ system allows to evaluate, in the evolution of the sepsis, four severity classes. The score SOFA is based on the assessment of the function of each organ system by means of the appropriate measuring medium: Respiratory, PaO_2/FiO_2, mmHg-coagulation, platelets, $\times$10,000/mm—liver, bilirubin, mg/dL—cardiovascular, mean arterial pressure (MAP) + amine support—central nervous system, Glasgow Coma Scale—renal, creatinine, mg/dL + urine output, mL/d. A score from 0 (normal condition) to 4 for increasing severity is assigned to each of the indices that report the functional condition of the six organ system. The baseline score is 0; the score 2 indicates already organ dysfunction, with 10% mortality; higher scores indicate serious functional impairment. In clinical practice, the quickSOFA has been proposed; it allows to more easily identify the patients at risk of developing septic status. The quickSOFA regards only three parameters of immediate clinical finding: Tachypnea—breath frequency more than 22 bm arterial pressure—less than 100 mmHg Glasgow Coma Scale—less than 15 (it ranges from 3 to 15; higher score indicates better neuro condition) [12]. It is necessary to briefly clarify the mechanism and timing of systemic inflammatory syndrome which from a simple localized infection can lead to the septic state. In this process, bacteria and endotoxins play the role of the triggers of the onset of systemic inflammation and sepsis, which is conditioned by the response of organism, as the trigger causes for the evolution and severity of subsequent events [13]. The cascade of inflammation mediators is activated and the inflammatory response is amplified. The systemic inflammation evolves in three steps. The first step is the trigger of inflammation, characterized by the intracellular activation of trypsinogen to trypsin, by zymogen-lysosomal granule. In the second step, the systemic inflammatory response develops; some phenomena follow: activated digestive enzymes in the blood circulation are present, chemokines in the secretory vesicles released by damaged or infected cells chemoattract inflammatory cells, and neutrophils-macrophages release cytokines. Therefore they develop: local inflammatory response with increased vascular permeability, hemorrhage, necrosis and systemic inflammatory response with the development of SIRS, MODS, toxic phase. Finally in the third step, there is the systemic infections response with the compensatory anti-inflammatory response syndrome (CARS), which is a complex and not a well-defined plan of immunologic responses to severe sepsis [13]. High mortality of septic shock is linked to multi-organ dysfunction, lung, kidney, liver, digestive system, heart, brain, and vascular system. The beginning of multi-organ dysfunction is very variable and unpredictable. In fact, it can be precipitated or slow and sneaky. The number of organs involved in the dysfunctional process and the time of the dysfunction condition the prognosis that progressively worsens with the increase of involved organs: if the involved organs are two, the mortality reaches 32%; it rises to 67% for three organs and finally to 90% in case of four or more organs. The mortality index rises significantly due to prolongation of dysfunction over 24–48 h. The evolution of systemic inflammatory syndrome, which underlies the multiple organ dysfunction syndrome (MODS) is favored by impaired general conditions of patients: old age, immune impairment, and active comorbidities as cardiovascular, renal, hepatic, metabolic pathologies [11].

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