The presence of pus indicates bacteria at the site!

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ABSTRACT

The presence of pus necessarily suggests bacterial contamination caused by staphylococcus and streptococcus. Interaction of neutrophils with these bacteria represents the mechanism of formation of pus in the human body. The presence of these bacteria can be analyzed and questioned as follows: 1. Were the bacteria already present prior to the surgical procedure? 2. Was the material previously contaminated by bacteria? 3. Was there any failure in the aseptic procedure? 4. Was there lack of oral hygiene in the postoperative phase? If the pus is formed, staphylococcus and streptococcus are present.

Keywords: Abscess. Bacteria. Pus. Contamination.

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The human body has ten trillion cells, but it also has ten times more bacteria (a hundred trillion). This impressive datum allows us to highlight the importance of the microbiota with which we have a close relationship. The presence of bacteria in our lives is essential to stimulate our defense mechanism.[3,4,21,22] Initially, they are known as animalcules and share the environment with fungi, viruses and parasite.

The skin and mucosae, natural integument, act in our defense mechanism as a physical, chemical and biological barrier that prevents microorganisms from entering the internal environment. Connective tissue represents the internal environment and comprises the oral submucosa, the dermis and bone tissue.

When the skin is cut, lacerated, subject to surgery, puncture or other methods that cause loss of skin or mucosa continuity, the integuments are transposed, overcome or ignored and the bacteria found in the microbiota enter the underlying connective tissue. In the event of loss of skin or mucosa continuity, bacteria will inevitably enter the internal environment due to the large amount of opportunities that are offered.

After all, we have a close relationship, we share and we biologically balance ourselves with a great amount of bacteria present in our inner and outer surfaces. The human body is prepared for the bacteria that occasionally enter its internal environment, which occurs practically every day.

When bacteria enter the connective tissues, inflammation is immediately triggered, and the blood vessels allow many plasmatic substances, the exudate, as well as many leukocyte cells, known as the infiltrate, to go to the affected area (Fig 1).

Blood vessels exude plasmatic components

Antibodies or immunoglobulins are among the substances that immediately flow out the blood vessels after bacteria enter the organism. These products, along with other plasmatic components such as proteins and enzymes that altogether are known as the complement system, react in a specific manner and provoke mechanisms that destroy the bacteria comprising human microbiota.

How does the organism previously know that these specific bacteria will enter the internal environment? Every day, these bacteria enter or try to enter our organism. Every day, our immune system is stimulated to produce antibodies and other products specifically aimed at destroying them. This is the reason why the blood plasma has high levels of specific antibodies that fight against the bacteria present in the microbiota, of which the most predominant are the staphylococcus and streptococcus.

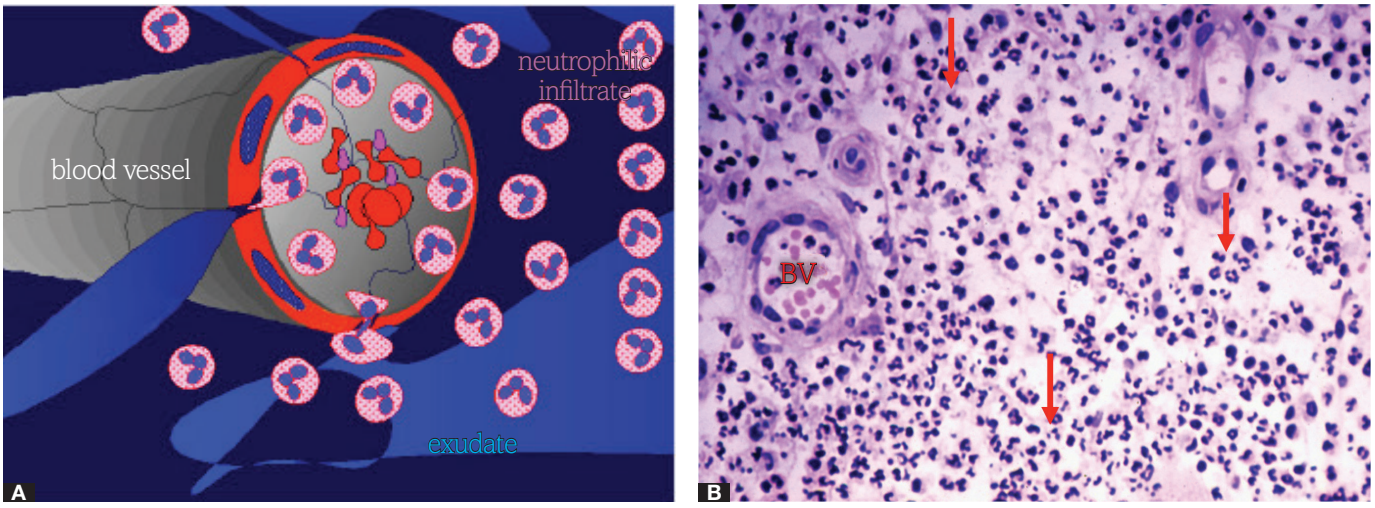

Figure 1. The neutrophils reach the attacked site through the vascular walls by means of leukodiapedesis (bent arrow in **A**) and predominate for 48-72h (smaller arrows in **B**). After 90 minutes, they interact with the bacteria and destroy them while releasing their lysosomal components. Should there be no bacteria at site, the neutrophils migrate or disappear by apoptosis and the site moves on to repair (**B** = H.E.; 160X).

The arrival of neutrophils and the formation of pus

Ninety minutes after any attack is caused to the connective tissues, for instance, when bacteria enter it, the first inflammatory cells that arrive in a considerable large number — almost exclusively within the next 48-72 h — are the neutrophils. The inflammatory, immune or defense cells are the leukocytes that continuously circulate in the blood (Fig 1).

The neutrophils are the most numerous leukocytes present in the blood (50-60%) and are specialized in phagocytizing bacteria, especially staphylococcus and streptococcus. Their function of interacting, phagocytizing and destroying these types of bacteria is related to the presence of many granules full of enzymes and other cytoplasmic powerful products (Fig 2). Other types of bacteria, such as tuberculosis and leprosy bacilli, do not interact with neutrophils. In short, neutrophils interact almost exclusively with staphylococcus and streptococcus.

The neutrophils are programmed to interact with staphylococcus and streptococcus, given that the latter are the most predominant microorganisms of our microbiota, in inner and outer surfaces, including the mouth.

When microbiota bacteria are able to cross or enter through open skin and mucosa barriers, antibodies, proteins of the complement system and other enzymes and products are immediately ready to inhibit, destroy and neutralize them. The neutrophils arrive ninety minutes later to phagocytize and completely eliminate them.

When neutrophils interact with bacteria, before closing the "clasp" they build around the microorganism to phagocytize it, the cytoplasm releases the content of its granules, the proteolytic degrading enzyme-based lysosomes as well as bactericidal substances such as hydrogen peroxide and chlorine solutions (Fig 2).

Neutrophil products also degrade collagen and other components of the extracellular matrix of the connective tissue. Degraded bacteria, dissolved components of connective tissue as well as dead neutrophils join the inflammatory exudate which, at this point, is no longer a serous fluid, but a yellow and viscous fluid (Fig 4), thus originating purulent exudate, also known as pus.

Neutrophils that cannot destroy the bacteria they have phagocytized burst and release a massive amount of degrading enzymes, a phenomenon known as cytolysis (Fig 3).

Neutrophils are also known as pyocytes or pus cells, whereas staphylococcus and streptococcus are also known as pyogenic bacteria or pus producers. The cluster of pus and neutrophils is generally known as abscess.

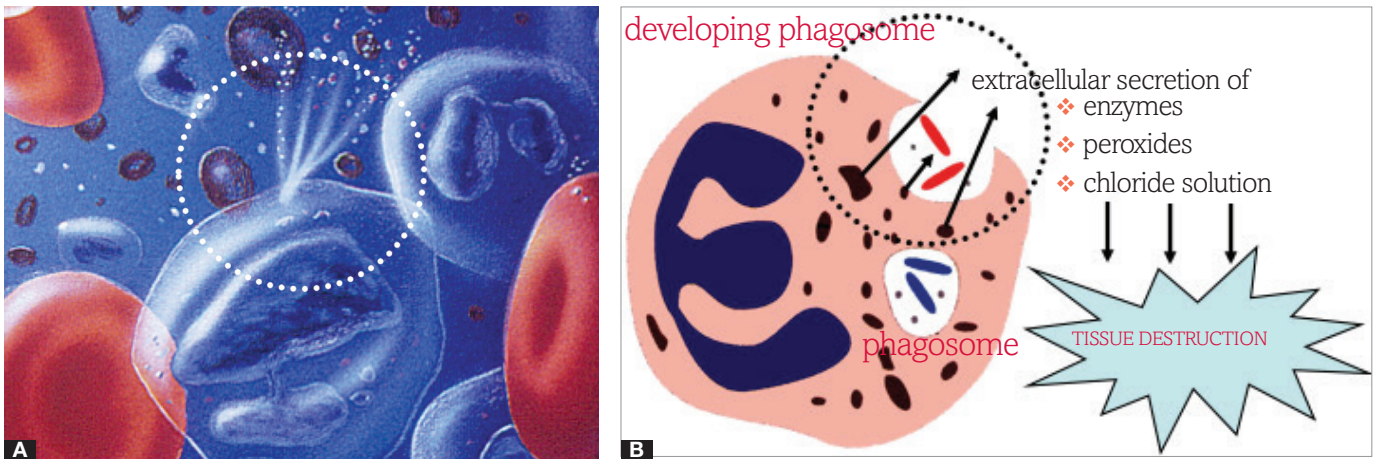

Figure 2. Phagocytosis of neutrophils is characterized by enzyme regurgitation (circles). Lysosomes release their powerful content into the phagocytic vacuole (arrows) before the latter is closed and, as a consequence, into the tissues. Depending on the amount of bacteria, tissue destruction occurs at different levels where the neutrophils are phagocytizing.

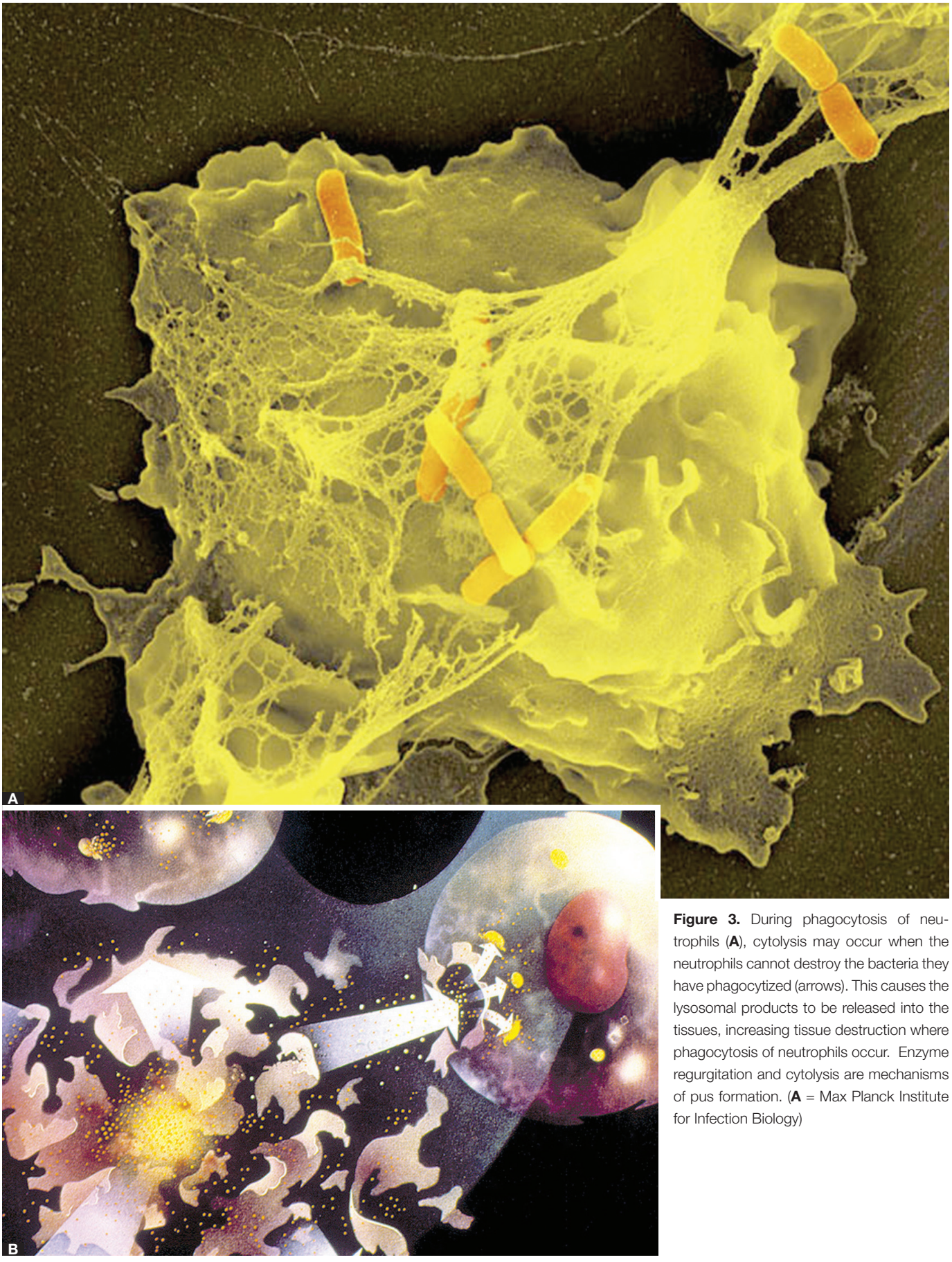

Figure 3. During phagocytosis of neutrophils (**A**), cytolysis may occur when the neutrophils cannot destroy the bacteria they have phagocytized (arrows). This causes the lysosomal products to be released into the tissues, increasing tissue destruction where phagocytosis of neutrophils occur. Enzyme regurgitation and cytolysis are mechanisms of pus formation. (**A** = Max Planck Institute for Infection Biology)

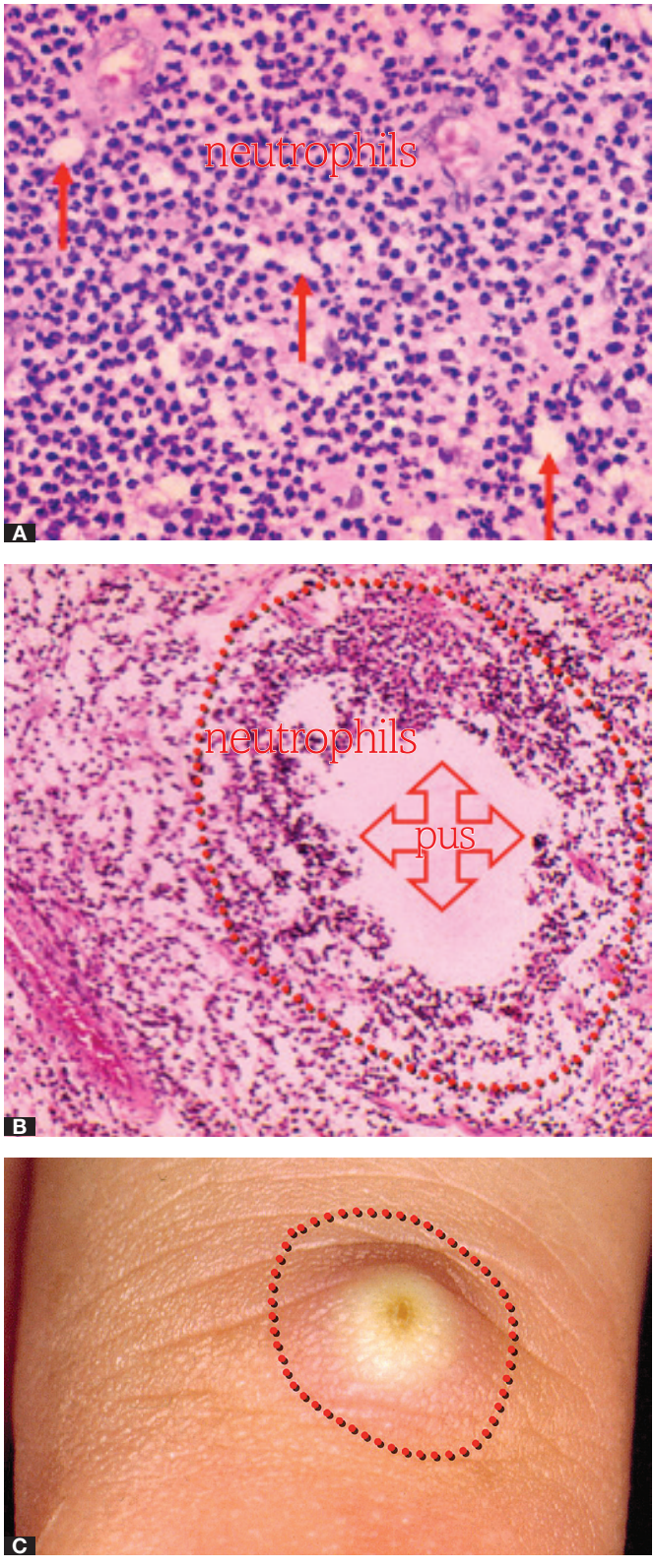

Figure 4. The extracellular matrix is filled with inflammatory exudate in the affected area and as enzyme regurgitation and cytolysis occur, the exudate ceases to be a serous fluid to become the yellow and viscous fluid that characterizes the formation of pus in microabscess (arrows in **A**). The microabscess gradually coalesce, forming clusters that characterize the abscesses (longer arrow and circles in **B** and **C**).

Little amount of bacteria forms no pus!

The destructive phenomena of bacteria-neutrophils interaction will occur regardless of the number of bacteria that enter a certain area of the connective tissue. However, should there only be a small amount of bacteria, the formation of pus will not be seen, i.e., the initially serous fluid does not become a yellow and viscous fluid, even though the phenomena occur likewise.

In other words: the amount of bacteria that enter the tissues is also important to clinically determine whether or not pus will be formed.

In general, a small amount of bacteria enters the surgical site, particularly in cases of parendodontic, periodontal and implant placement surgeries as well as in direct pulp procedures. However, because they are in small amounts, the antibodies, proteins and neutrophils are able to quickly destroy them, thus avoiding the formation of pus. Likewise, in endodontic procedures, bacteria can reach the stump and the apical periodontal ligament, but if in small amounts, they will not cause the formation of pus.

Asepsis represents a set of procedures aimed at preventing bacteria from entering sites where they had not existed before. To this end, sterilization, disinfection and antisepsis procedures are carried out. Nevertheless, it is quite impossible to prevent bacteria from entering the oral environment, especially into connective tissue exposed by endodontic or surgical procedures. No problems occur in the majority of cases because, when proper aseptic care is taken, only a small number of bacteria reach the surgical wound, even in cases of staphylococcus and streptococcus.

Should there be formation of pus, bacteria are present!

Regardless of where, the formation of pus has a well-established meaning: presence of staphylococcus and streptococcus. It may occur around a suture wire, in the alveolus, after extraction, in the gingiva after surgery or around a dental implant. Some rare cases involving chemical products, as it is the case of turpentine, may lead to the formation of pus when the

products are inserted in large amounts in the connective tissue. Nevertheless, this practice only happens in experimental procedures.

The main and almost exclusive cause of pus formation is the interaction between bacteria and neutrophils. The presence of pus does not suggest rejection, immune rejection, bad material quality, improper composition of filling cement, bad suture wire, occlusal overload or trauma.

Solid/particulate material usually placed within tissues does not induce the formation of pus. Metals and alloys, surgical and filling cements, resin, polymers and other solid material alone do not lead to the formation of pus, unless they have been contaminated with bacteria. Staphylococcus and streptococcus contamination is what induces the formation of pus. The presence of these bacteria can be analyzed and questioned as follows:

- Were the bacteria already present prior to the surgical procedure?
- Was the material previously contaminated by bacteria?
- Was there any failure in the aseptic procedure?
- Was there lack of oral hygiene in the postoperative phase?

No matter the explanation, if pus is formed, staphylococcus and streptococcus are present.

Final considerations

The presence of pus necessarily suggests bacterial contamination caused by staphylococcus and streptococcus. The neutrophil-bacteria interaction is the main and most important mechanism of pus formation in the human body. Additionally, it may be an important point for clinical thinking: how, when and why these bacteria reach the spot.

I have been questioned and induced to answer the following question:[7] What does the presence of pus around osseointegrated implants mean? This question is naturally extended to other clinical conditions, especially with regard to Endodontics: in clinical practice, the formation of pus only and necessarily occurs when bacteria are present!

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