

# Overflow of filling material: Is it good or bad?

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doi: <http://dx.doi.org/10.1590/2178-3713.4.2.018-025.end>

## ABSTRACT

In case of overflow of filling material with controlled or absent microbiota, periodontal ligament and periapical bone reconstruction do not occur immediately around fragments. These fragments are considered as foreign bodies for inducing inflammatory, yet non-immune response. Macrophages and fibroblasts enclose fragments and form foreign body periapical granuloma. Dental granuloma is known as “periapical granuloma” of immunogenic and inflammatory nature. Although foreign body periapical granuloma does not produce further systemic results and have a low risk of anachoresis, it does not restore normality of periapical tissues after endodontic treatment. Should material be calcium

hydroxide-based, macrophages phagocytize, migrate and remove it. As a result, foreign body periapical granuloma disappears within a few months. However, should filling material be resin or glass ionomer-based, foreign body periapical granuloma remains at the site for an indefinite period of time. Preventing overflow would be ideal; however, should it be unavoidable, it would be best if material easily removed by macrophages were used so as to ensure restoration of normality even though, from a systemic and clinical point of view, foreign body periapical granuloma does not cause relevant clinical problems.

**Keywords:** Overflow. Periapical granuloma. Foreign body. Periapical periodontitis. Filling procedure. Filling cement.

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Submitted: April 15, 2014. Revised and accepted: April 17, 2014.

**How to cite this article:** Consolaro A, Bittencourt G. Overflow of filling material: Is it good or bad? *Dental Press Endod.* 2014 May-Aug;4(2):18-25. DOI: <http://dx.doi.org/10.1590/2178-3713.4.2.018-025.end>

» The authors report no commercial, proprietary or financial interest in the products or companies described in this article.

» Patients displayed in this article previously approved the use of their facial and intraoral photographs.

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## Introduction

All offending agents in contact with tissue release protein as a result of connective tissue cells, fibers and extracellular matrix components breakage; thereby triggering inflammation. Every inflammatory process is unspecific at first. In other words, the same phenomena occur for all types of offending agents.<sup>1</sup>

Free proteins immediately degranulate millions of mast cells loaded with histamines into connective tissues, whether fibrous, bony, adipose or medullary. Histamine induces the first vascular alterations upon arrival of plasmatic substances and blood or inflammatory cells: the exudate and, later on, inflammatory infiltrate form.

Millions of neutrophils are the first cells to reach the site. Their function is to phagocytize bacteria, especially *staphylococcus* and *streptococcus*. Should there be interaction with these bacteria, neutrophils expel proteolytic enzymes and burst by cytolysis, in which case they might not change the characteristics of the exudate. However, should neutrophils intensely interact with these bacteria, they render serous exudate into purulent exudate, thereby resulting in focal accumulation of pus. In other words, abscess, known under different names according to the site of occurrence, is established. For instance, in the apex, it is known as dentoalveolar abscess; whereas in the pulp it is known as purulent acute pulpitis. Pus formation implies these morphotypes of bacteria: *staphylococcus* and *streptococcus*.

Pus formation might persist and increasingly accumulate, resulting in a dramatic clinical presentation in terms of symptoms. Furthermore, it might induce subclinical presentation as a result of ongoing pus drainage by surface fistulas, via canal or periodontal ligament. While *staphylococcus* and *streptococcus* bacteria remain on the site, neutrophils persist for an indefinite period of time, continuously renewing themselves despite having a short life span of 10 to 24h.

When the offending agent represented by mixed microbiota consistently injures the tissues, low intensity neutrophils remain on the site interacting with bacteria that reach the tissues. Nevertheless, after a few days or months, mononuclear leukocytes (macrophages, lymphocytes and plasma cells) accumulate around the site of microbial penetration and neutrophils accumulation forming a second de-

fense barrier: Should bacteria escape from neutrophils, they are countered or eliminated along with their byproducts by potent macrophages aided by substances as those released by lymphocytes and plasma cells, particularly cytokines and antibodies.

This cluster of mononuclear leukocytes, especially macrophages, is known as granuloma. At the periapical region, it is known as periapical granuloma. Lesion may remain for months or years. Since its cells participate in immunologic response, chronic inflammatory response and immunologic response simultaneously occur at the site of periapical granuloma. Immunologic response is always stimulated by offending agents of protein composition, as it is the case of bacterial components and byproducts.

## After root canal filling

Offending agents do not always comprise bacteria and their byproducts. Cuts, perforation and other mechanical actions performed on tissues also trigger the same inflammatory response with exudate and neutrophils reaching the injured site. This is the case of biopulpectomy. Without bacteria, neutrophils remain on the site for a few hours and soon migrate or disappear by apoptosis. Within 48 to 72h, neutrophils are gradually replaced by macrophages, lymphocytes and plasma cells so as to fulfill the following:

- 1) Clean the site by eliminating cell debris and, as a result, promote repair; as it occurs in periodontal stump or apical periodontal ligament after biopulpectomy.

- 2) Interact with offending agents not destroyed or controlled by neutrophils. For instance, bacteria and bacterial byproducts forming granuloma. The latter is used to identify accumulation or cluster of macrophages with any degree of organization, associated or not with lymphocytes and plasma cells.

We will always consider the hypothesis that the endodontist was able to destroy or control microbiota in root canal and mineralized structures of dental tissue.

A few hours after root canal filling, neutrophils migrate or disappear by apoptosis, and macrophages reach the site associated or not with lymphocytes and plasma cells. The macrophages begin cleaning cell, tissue and bacterial debris while releasing mediators that stimulate angiogenesis in neighboring tissues.

The vessels form a vascular network through which young and proliferating cells migrate to form a set of vessels and cells known as granulation tissue. This tissue evolved to periodontal ligament and neoformed bone, restoring periapical structural normality (Fig 1).

### What happens when filling material overflows?

The principles of endodontic treatment consist in destroying or controlling microbiota from root canal and mineralized tooth structures. Repair phenomena prevail after biopulpectomy and necropulpectomy with or without periapical lesion, given that microbiota is controlled or eliminated.

Filling material may occasionally overflow and remain out of the mineralized tooth structure. Irregular fragments of filling cement may remain in the

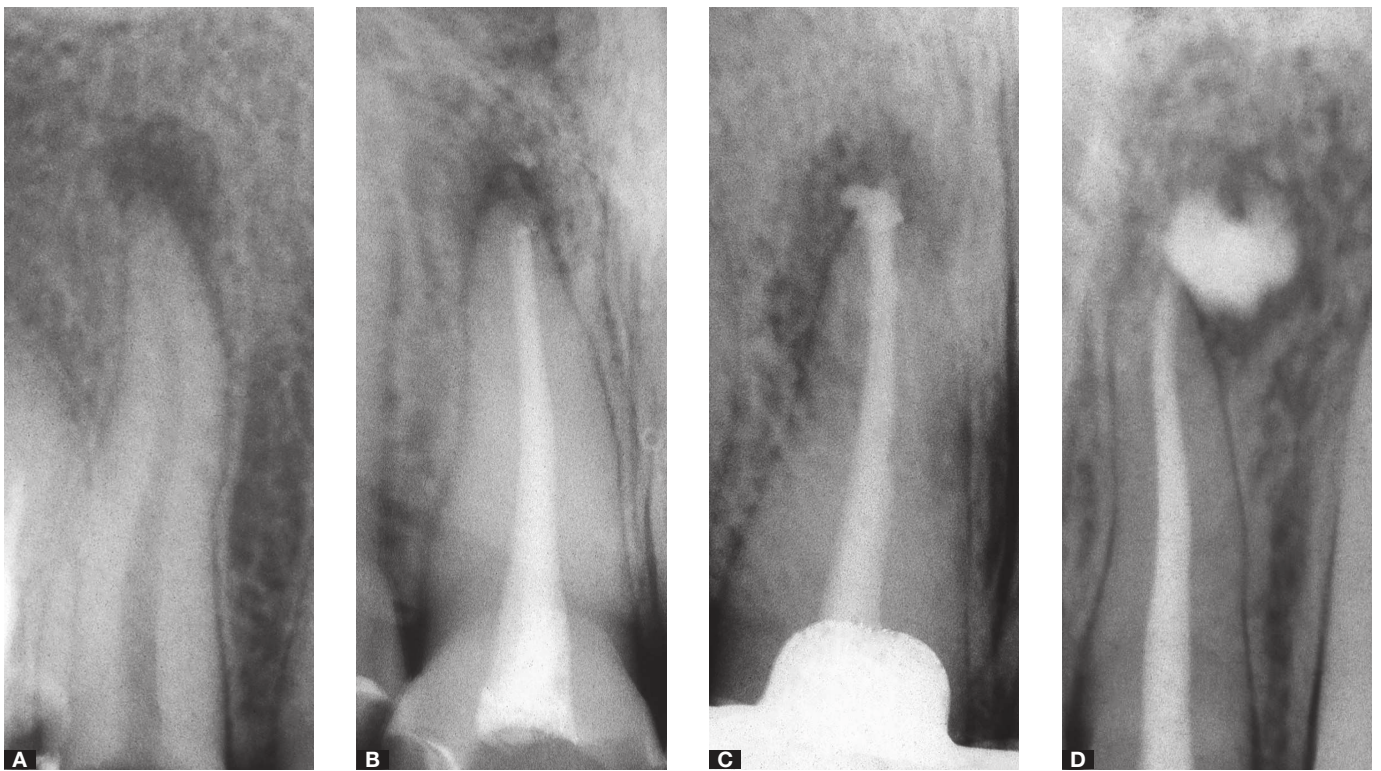
periodontal ligament. Since periodontal ligament is extremely thin (from 0.2 to 0.4 mm), irregular fragments of filling cement also remain in neighboring periapical bone spaces (Figs 2 to 6). In these cases, two different conditions may occur:

1) Overflow after biopulpectomy — Should overflow of filling cement occur after biopulpectomy, material will induce primary inflammation around itself, apical periodontal ligament and other periapical tissues. Mast cells will be degranulated, and exudate as well as inflammatory infiltrate will form. Within the first 24–48h, neutrophils prevail. However, should they not find any bacteria to interact with, they migrate or disappear by apoptosis.

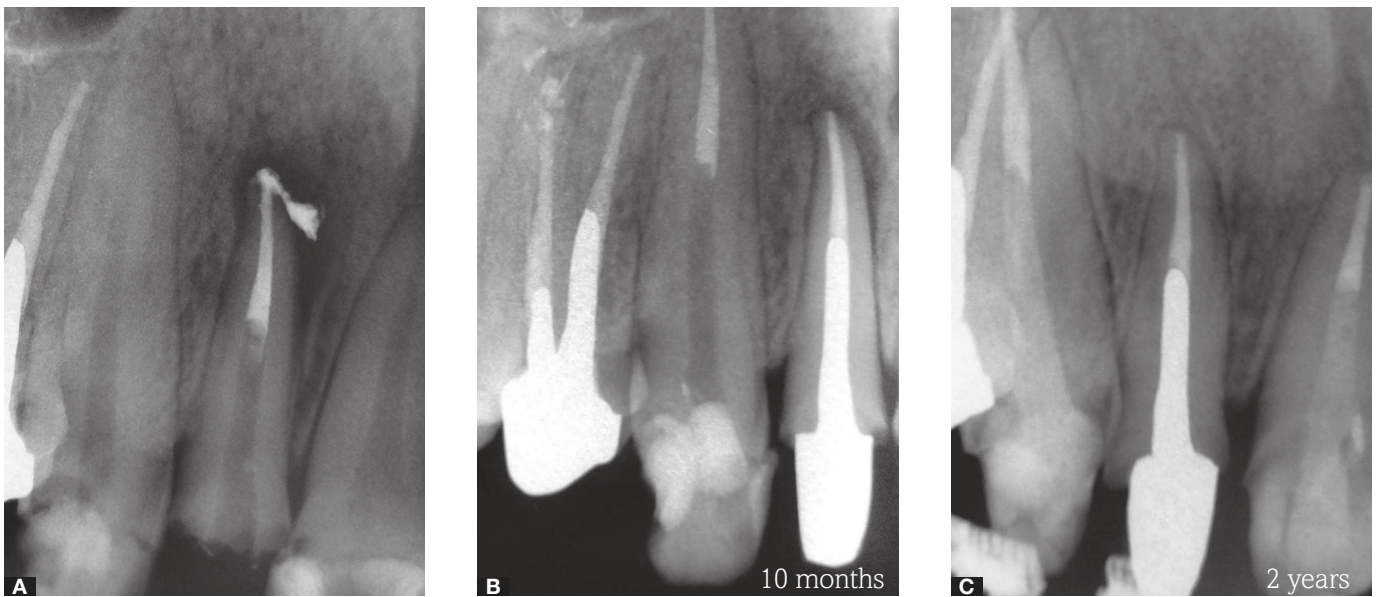
After 8 to 12 hours, macrophages reach the site where they prevail for 48 to 72 hours. To trigger tissue repair, macrophages start cleaning the site and



**Figure 1.** A few months after endodontic treatment without microorganisms or foreign bodies, periodontal tissues are restored to normality as revealed by microscopy: FM = filling material, C = new cementum formed over material, PL = periodontal ligament, AB = alveolar bone and D = dentin (HE, 25X) (Source: Leonardo,<sup>2</sup> 1992).



**Figure 2.** Regardless of the amount of filling material, should overflow occur with or without controlled microbiota, periapical lesion will evolve to repair except for its surrounding areas. Transient or permanent foreign body periapical granuloma will form depending on the physicochemical characteristics of the cement which determines whether granuloma is resorbable.



**Figure 3.** Root canal filling with calcium hydroxide-based cement overflow. After 10 months, material overflow was no longer observed. After 2 years, periodontal tissues were completely restored to normality as revealed by imaging exams.

try to phagocytize the filling material in order to make room for granulation tissue and tissue neoformation. Even the smallest fragments of overflow filling material are too big for macrophages; for this reason, these cells assemble around them. Such accumulation of macrophages is what constitutes a granuloma, and does not establish immunologic response (Figs 4, 5 and 6).

Immunologic response is triggered by protein agents such as microorganisms, transplants and others. Manufacturers of root canal filling material are particularly concerned about not adding any protein components in their composition. Thus, when filling cements, gutta-percha, suture wires, metallic and plastic material, and resins contact any type of tissue, they promote nonspecific inflammation (early and late) given that they do not promote immunologic or immunogenic response.

Particulate material inducing inflammatory response, but not inducing immunogenic response is known as foreign body. Granuloma embracing this type of material consists of macrophages in its interface with neighboring tissues, such as fibroblasts and collagen fibers. For this reason, this type of granuloma is known as foreign body-type or non-immunogenic granuloma.

In the apical region, this type of granuloma is known as foreign body periapical granuloma. It differs from periapical granuloma because the latter is induced by microbiota of high immunogenic capacity. In other words, periapical granuloma is an inflammatory and immunogenic response of the organism against the ongoing entry of bacteria and byproducts.

2) **Overflow after necropulpectomy** — Should overflow occur after necropulpectomy, with or without periapical lesion, fragments of filling material will randomly stand in the middle of the pre-existing inflammatory periapical lesion. In either chronic dentoalveolar abscess or periapical granuloma, neutrophils migrate or disappear as microbiota is absent. Macrophages enclose the fragments of filling material and organize themselves into foreign body-type granuloma. The other areas will evolve to periodontal ligament and bone repair; however, macrophages will remain around fragments of filling cement as foreign body periapical granuloma for an indefinite period of time (Figs 2 and 3).

## **Foreign body periapical granuloma: Clinical, imaging and microscopic views.**

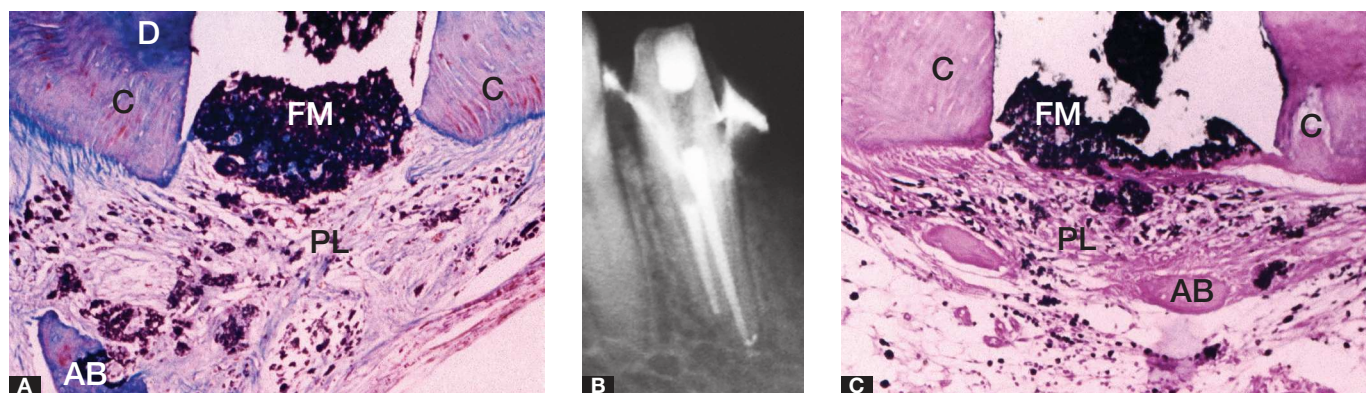
» **Clinical view** — Patients carriers of foreign body periapical granuloma occasionally and most of times rarely report twinges of pain. This may be due to the fact that teeth enter and leave the socket while performing ordinary movement. While chewing, one may intrude a tooth more than normal and, as a result, compress the ligament with fragments of filling cement pressing against the periapical bone and stimulating one of the neural threads of that area. Importantly, this situation is rare and does not justify case retreatment, but is used to understand and explain why it occurred.

» **Imaging view** — Within a few weeks or months, two different evolving situations may occur (Figs 2 and 3) depending on the type of filling material:

1) **Resin/glass ionomer-based material**: An irregular discrete radiolucent halo is established around the material. It accounts for the space surrounding the material and occupied by macrophages, fibroblasts and collagen fibers of the foreign body periapical granuloma. Remaining spaces and characteristics of the lesion are gradually repaired, as microbiota has been controlled and/or eliminated.

This situation remains for an indefinite period of time with material enclosed by a radiolucent halo on one side. Should it be resin or glass ionomer-based, its enzymes do not break the fragments of glass and/or resin because macrophages are not able to phagocytize or “reabsorb” this type of material (Fig 2). Macrophages remain around the material for an indefinite period of time. Similarly, when gutta-percha cones overfill, the same type of reaction is established around them.

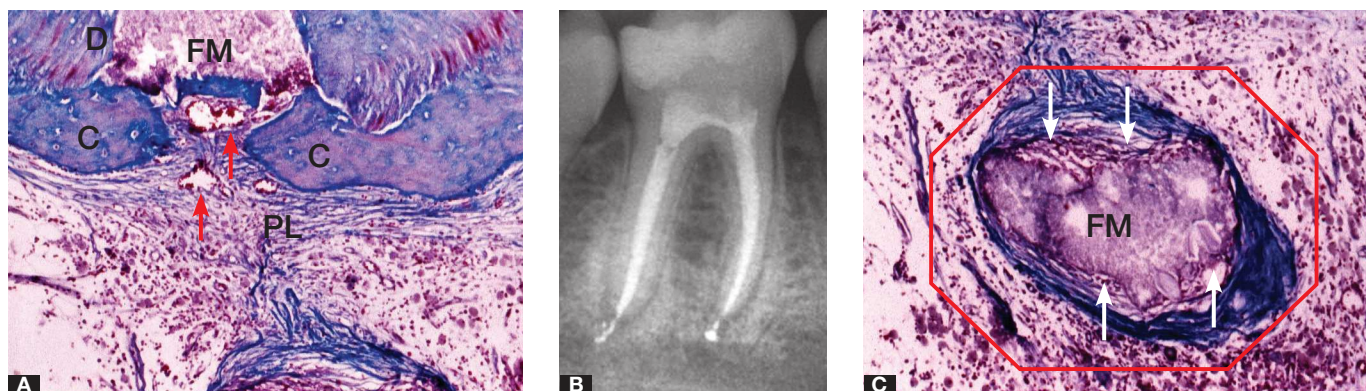
2) **Calcium hydroxide-based material**: Should material be calcium hydroxide-based or derived from other product phagocytatable or resorbable by macrophages, imaging exams will slowly and progressively reveal decreased filling material overflow (Fig 3). Mononuclear or multinucleated macrophages gradually remove microparticles from the main fragment body interiorizing them. Nevertheless, they are oftentimes not capable of dissolving the microparticles and, for this reason, carry them further away until they are eliminated via urine, feces, sweat and



**Figure 4.** Root canal filling with calcium hydroxide-based cement overflow. As in **B**, filling material overflow in the apical region is gradually phagocitized by macrophages — cells are evinced (black) as their cytoplasm is loaded with fragments of filling material. AB = alveolar bone, C = cement, FM = filling material (TM and HE, 25X) (**A** and **C**, Source: Leonardo,<sup>2</sup> 1992).



**Figure 5.** As in **B**, root canal filling with material overflow induces formation of foreign body periapical granuloma in the periodontal ligament (PL) and periapical tissues (between lines) in the apical region. The clusters of macrophages are evinced (black) as the cells cytoplasm is loaded with fragments of filling material. Neighboring vessels may have increased wall permeability (arrow). AB = alveolar bone, C = cement, FM = filling material (HE, 25X) (**A** and **C**, Source: Leonardo,<sup>2</sup> 1992).



**Figure 6.** Despite cement overflow, apical tissues are reorganized and promote, as in **B**, formation of foreign body periapical granuloma in the periodontal ligament (PL) and periapical tissues (between lines) in the apical region, sometimes enclosed by a fibrous capsule (blue). The clusters of macrophages are not so evinced (white), since filling material does not appear as black particles within the cell cytoplasm. Neighboring vessels may have increased wall permeability (red). C = cement, FM = filling material, D = dentine (TM, 25X) (**A** and **C**, Source: Leonardo,<sup>2</sup> 1992).

other secretions. After one or two years, depending on the size and amount of overflowed material, periapical imaging exams may not reveal foreign body periapical granuloma (Fig 3).

» Microscopic view — In the previous situations, the following is observed:

1) Foreign body periapical granuloma induced by resin or glass ionomer-based material: Mononuclear or multinucleated macrophages enclose and interact with the surface of resin or glass ionomer-based material and remain as so for an indefinite period of time. Around them, fibroblasts and delicate collagen fibers enclose the entire process. Fragments of filling material are not observed within the cytoplasm of macrophages or at neighboring macrophages.

2) Foreign body periapical granuloma induced by calcium hydroxide-based material: Mononuclear or multinucleated macrophages have numerous phagocytized particles (Figs 4, 5 and 6) which are transposed to other sites. Importantly, macrophages are characterized by a “walking” behavior when loaded with phagocytized material and without stimuli to remain around it. Phagocytosing macrophages’ stimuli of remaining at site generally originates from lymphocytes absent in foreign body-type or non-immunogenic granuloma.

## **Foreign body periapical granuloma: Long-term effects on the organism**

From a systemic standpoint, foreign body-type granuloma does not produce further consequences; however, one should not overlook the possibility of anachoresis.

A granuloma is synonym of chronic inflammation nourished by neighboring vessels of increased permeability (Fig 5, 6). When in the blood, bacteria may settle at previously injured or inflamed sites, thereby triggering a phenomenon known as anachoresis.

Rarely and of difficult detection, anachoresis may evolve to foreign body periapical granuloma. This possibility should not be dismissed, at least hypothetically.

Daily activities such as tooth brushing, showering, hair combing, chewing and crying end up carrying microorganisms into connective tissues by breaking the epithelium, followed by entry of microorganisms in the blood via broken vessels. Transient bacteremia occurs during any type of dental procedure, even those consisting of placement of orthodontic accessories only.

In other words, every day we have brief moments of bacteremia. It lasts for a short period of time due to a number of leukocytes and substances of our immune system present in the blood. Once bacteria reach the vessels, they have difficulty in achieving tissues because the openings of endothelial walls are smaller than the bacteria diameter. Nevertheless, in inflamed or continuously injured areas (for instance, in foreign body periapical granuloma), these bacteria occasionally leave the permeable vessels and settle at the material hollow, thereby promoting potential relapses by hematogenous route.

## Final considerations

Should there be overflow of filling material — in the event of biopulpectomy or necropulpectomy with or without lesion in which microbiota has been controlled and eliminated — periapical tissue repair with reconstruction of periodontal ligament and periapical bone occur in all areas, except for those around material fragments considered as foreign body for inducing inflammatory, but not immunologic response.

Macrophages and fibroblasts enclose fragments and form foreign body periapical granuloma. Dental granuloma is known as “periapical granuloma” of immunogenic and inflammatory nature.

Although foreign body periapical granuloma does not produce further systemic results and have a low risk of anachoresis, it does not restore normality of periapical tissues after endodontic treatment. Should material be calcium hydroxide-based, macrophages phagocytize, migrate and remove it. As a result, foreign body periapical granuloma disappears within a few months. However, should filling material be resin or glass ionomer-based, foreign body periapical granuloma remains at the site for an indefinite period of time, since macrophages do not degrade it.

Preventing overflow would be ideal; however, should it be unavoidable, it would be best if material easily removed by macrophages were used so as to ensure restoration of normality even though, from a systemic and clinical point of view, foreign body periapical granuloma does not cause relevant clinical problems.

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