Dansk Endodontiforening

Hindsgavl Slot d. 8. -9. januar 2016

Biofilms in endodontic infections

Domenico Ricucci MD, DDS


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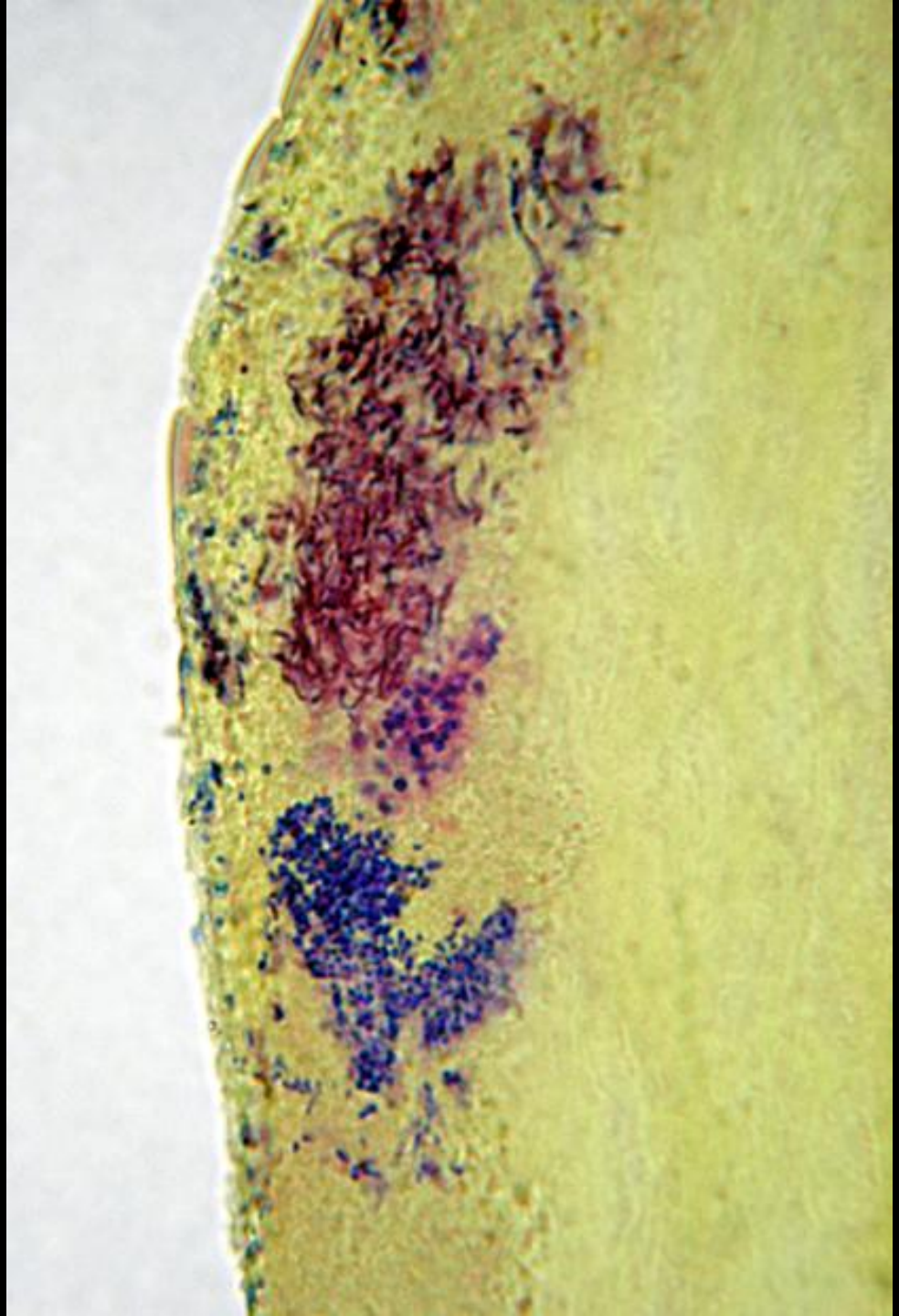
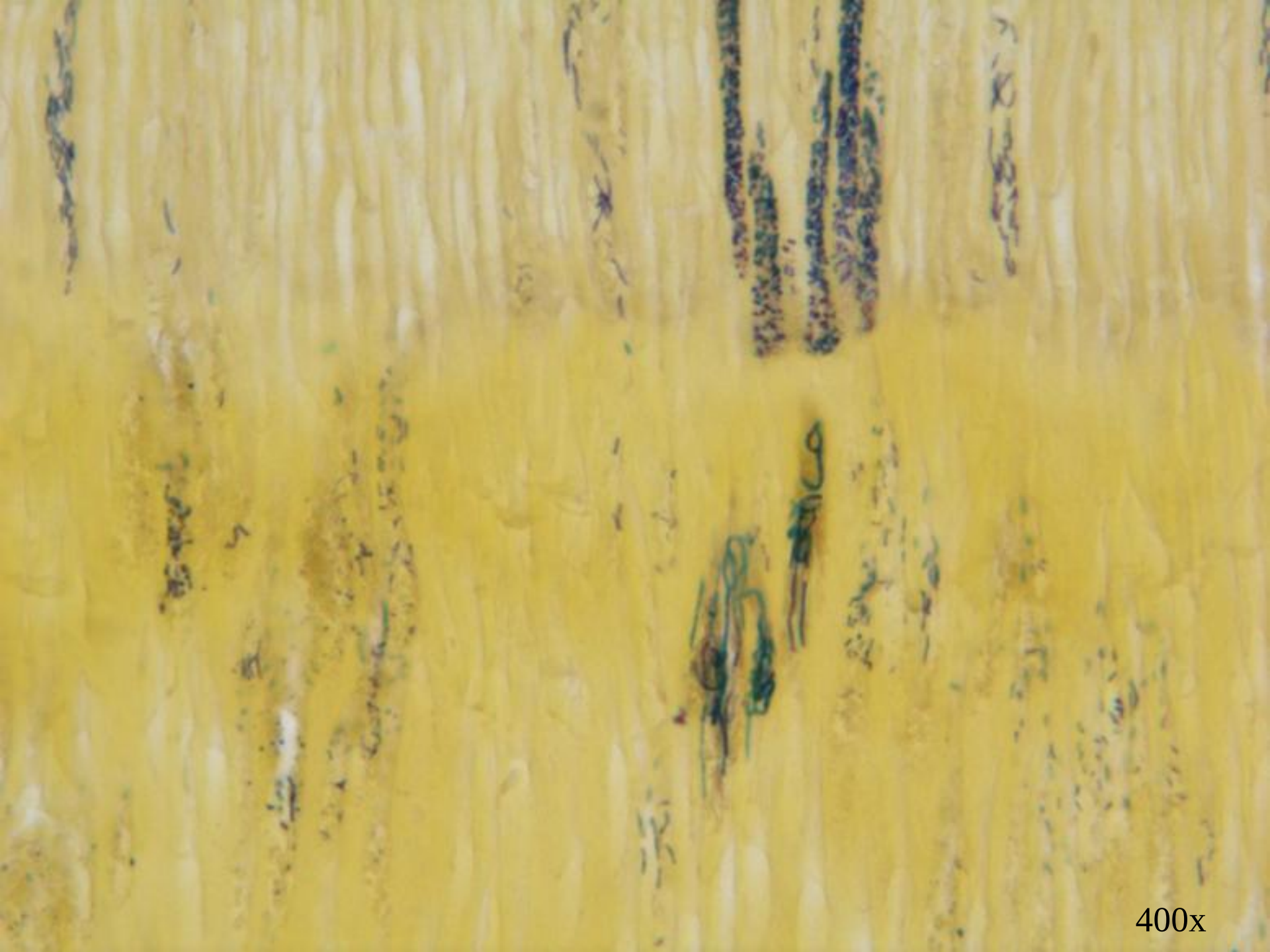
Taylor RD.
Modification of the Brown and Brenn Gram stain for the differential staining of gram-positive and gram-negative bacteria in tissue sections.
Am J Clin Pathol 1966;46:472-6.





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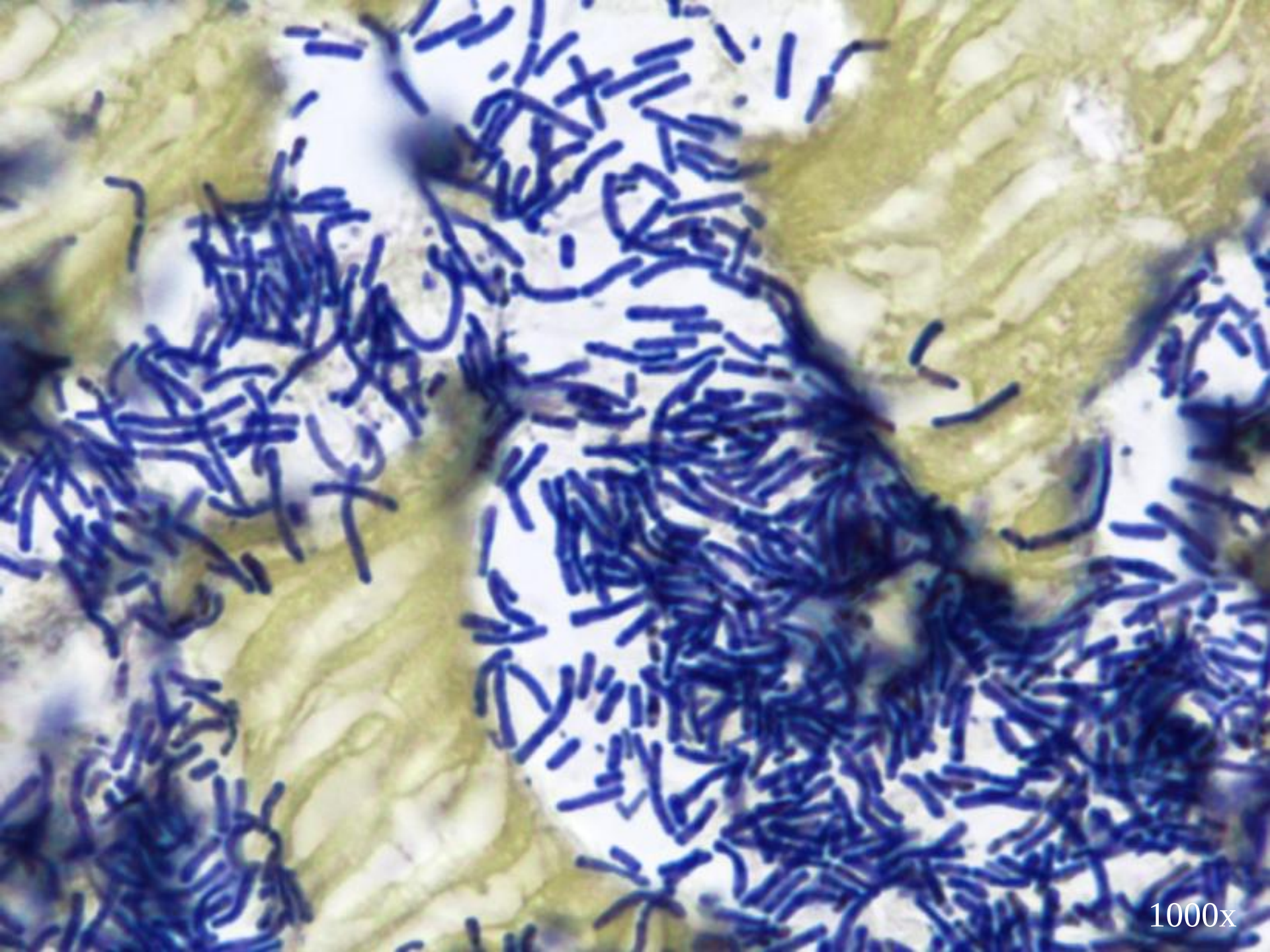
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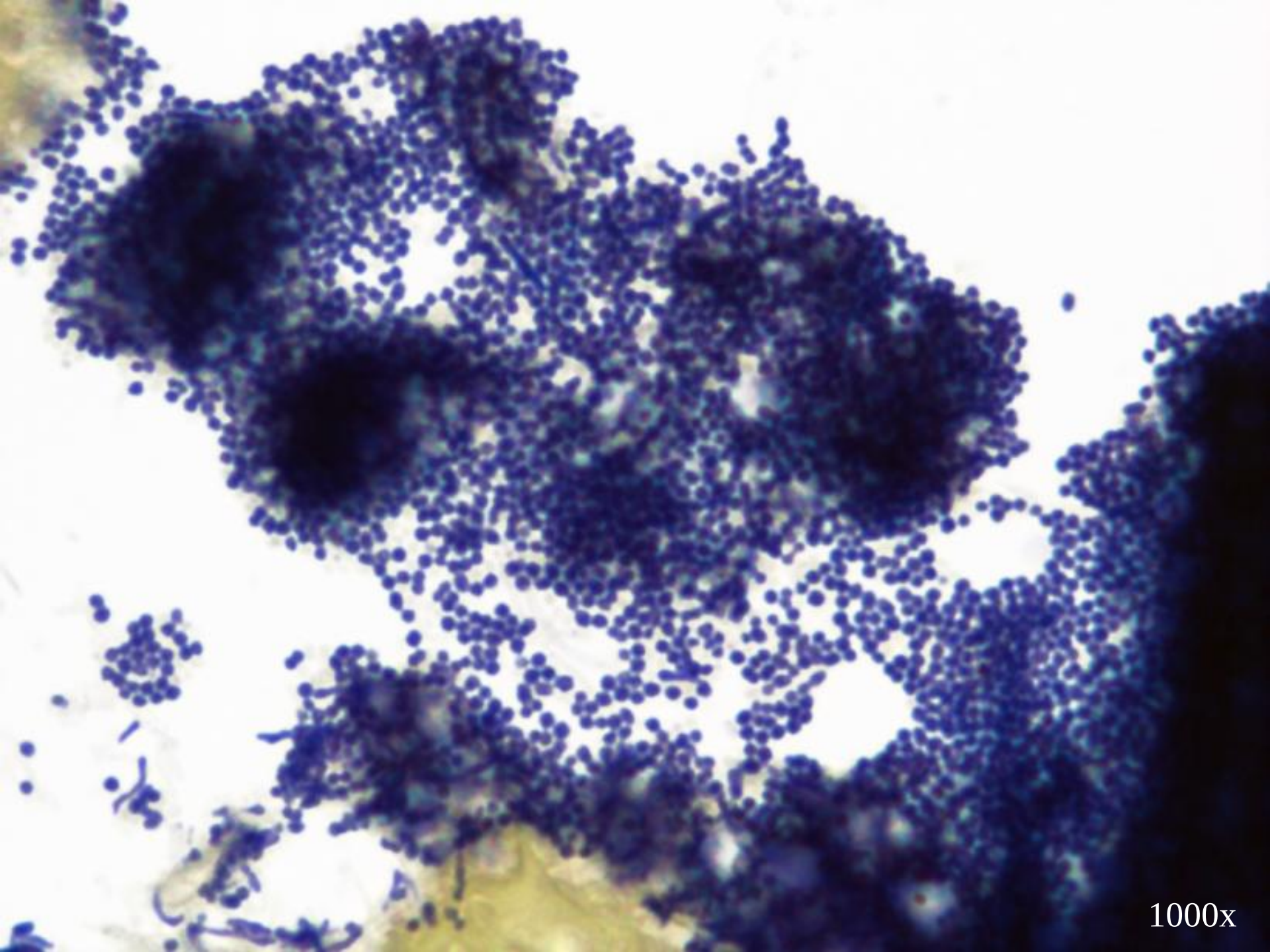
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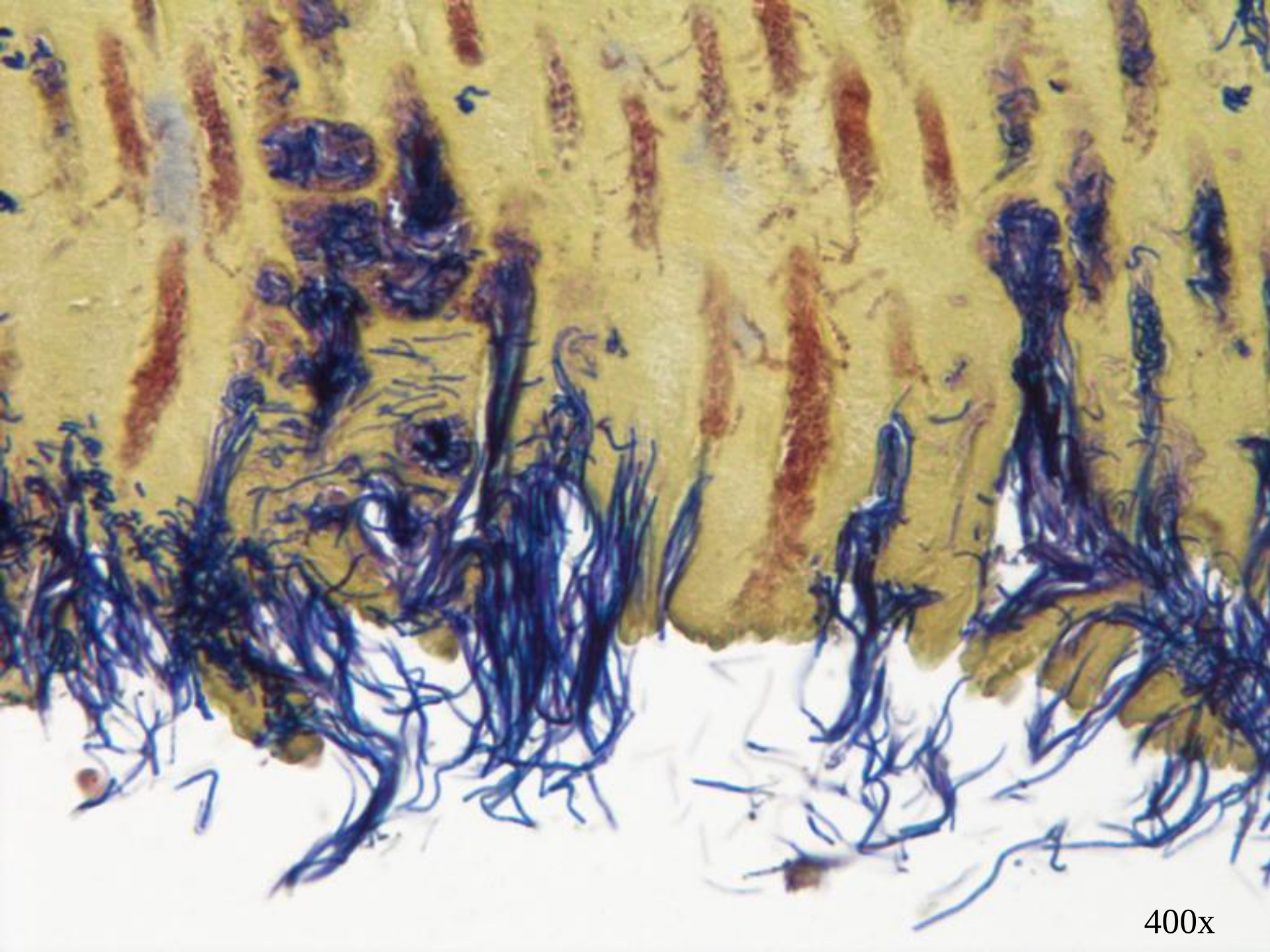
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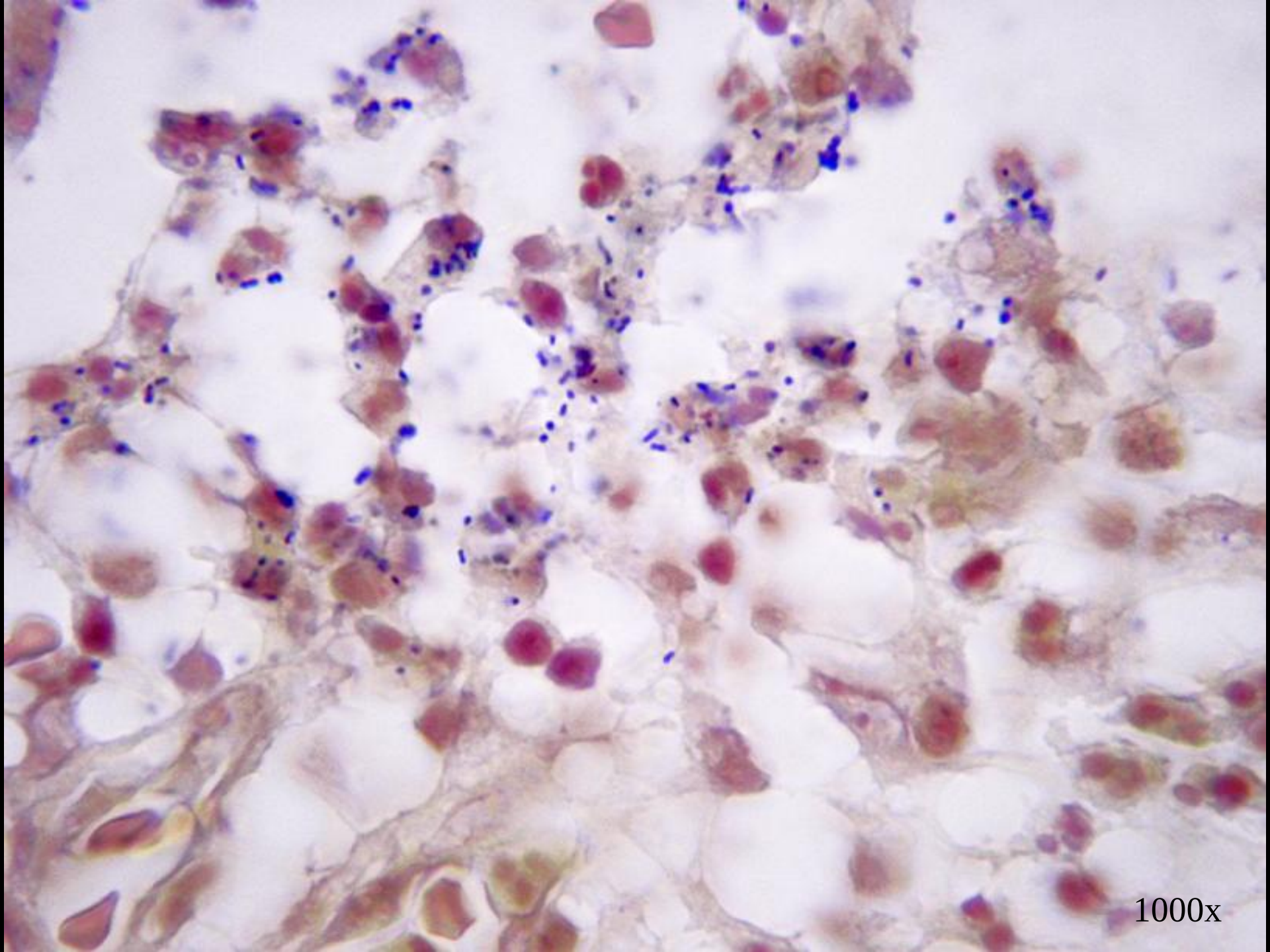
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JOURNAL OF ENDODONTICS

Printed in U.S.A.
VOL. 13, NO. 1, JANUARY 1987

Light and Electron Microscopic Studies of Root Canal Flora and Periapical Lesions

P. N. Ramachandran Nair, BVSc, DVM

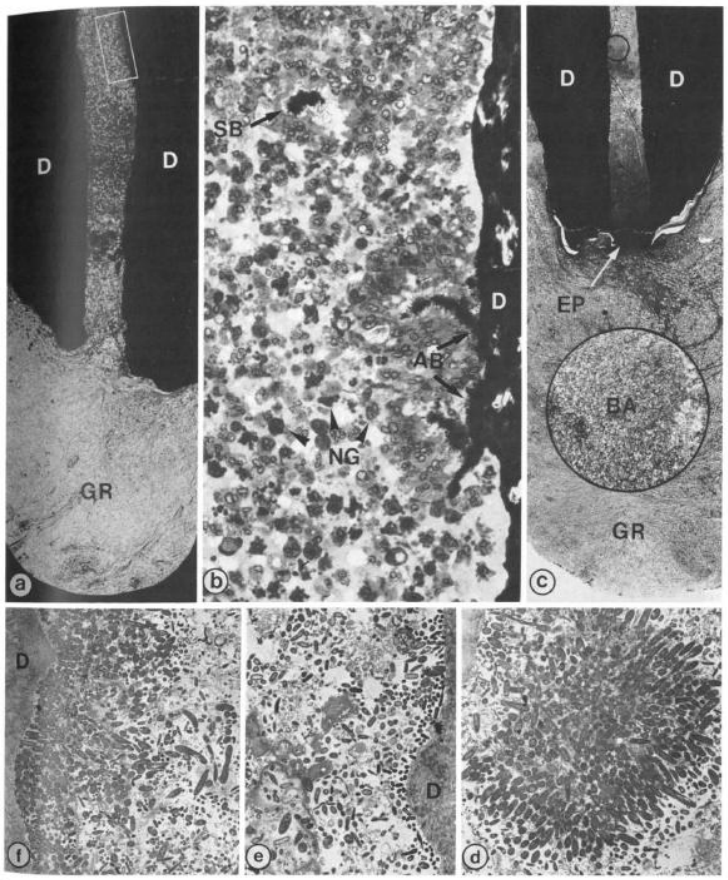
This study elaborates on (a) the structure of the root canal flora in periapically affected human teeth; (b) the relationship of flora to the dentinal wall of the root canal; (c) morphological aspects of interaction among diverse groups of bacteria; and (d) the dynamic nature of inflammatory host response at the periapex. Spontaneous human periapical lesions (30 granulomas and 1 radicular cyst) obtained by tooth extraction were processed for light and electron microscopy. All 31 teeth showed bacteria in their root canals. The flora consisted of cocci, rods, filamentous organisms, and spirochetes. The rods often revealed Gram-negative walls. The bacterial flora formed clusters of "self-aggregating" colonies of one distinct type or "coaggregating" communities of several types. Electron microscopically a condensed bacterial layer could be identified on the dentinal wall of the root canal which when light microscopically visible, gave the palisade structure of bacterial plaques adhering to tooth surfaces. The endodontic flora was separated from the rest of the lesion by a dense wall of neutrophils (PMN's) or by an "epithelial plug" at the apical foramen. Five specimens revealed both intra- and extracellular bacteria within the body of the lesions. The membrane-delimited phagosomes containing bacteria showed interaction between phagocytic cells and bacteria. The presence of the "immunocoating" on the surface of certain phagocytosed bacteria strongly suggests that they were not contaminants but actual tissue invaders against which the host mounted a specific immune response at the periapex.

The primary etiological agent of all periapical lesions has long been considered to be bacterial. Nevertheless, attempts (1–4) to identify organisms which are significant for the pathogenesis of periapical lesions have largely failed due to the enormous problem of bacterial contamination or to inadequacy in microbial culture techniques (5). In spite of this, samples of granulomas gained after tooth extraction or removed by flap surgery have been studied both bacteriologically and histologically (1, 2, 4, 6–11). In view of the obvious contamination problem, only bacteria found within intact lesions with firm attachment to the root apex, particularly those found within phagocytic cells, were considered genuine findings. With the exception of one investigation (9), those studies (1, 6, 8, 10, 11) tend to suggest that only a small fraction (5–15%) of periapical lesions, especially those with acute inflammation may show the presence of bacteria within. Consequentiy, periapical lesions were considered to be caused not necessarily by microorganisms alone but rather by other primary and independent cofactors such as decomposition products of necrotic pulp tissue (12) or root canal fillings. Efforts have also been made to implicate immunopathological mechanisms in the initiation of these lesions (13, 14).

Investigations using refined microbiological techniques (15–19) resulted in repeated reporting of certain groups of bacteria, particularly some obligate and facultative anaerobes in combination with less fastidious Gram-positive organisms. Carefully designed pathobiological experiments using those organisms or their products (20–27) in animal models tend to suggest high endodontopathic potential of the mixed root canal infections and their probable involvement in the pathogenesis of periapical lesions. Nevertheless, modern histopathological studies of intact periapical lesions with firm attachment to the root apex are extremely rare. Except for a couple of publications (28, 29), transmission electron microscopic studies of the root canal and periapical flora do not seem to exist. The purpose of this communication is to study the endodontic and periapical flora of diseased human teeth using correlated light and transmission electron microscopy.

MATERIALS AND METHODS

The tissue specimens (31 lesions) formed part of a collection of endodontically untreated and periapically affected human teeth obtained by extraction. All 31 teeth were profoundly carious. The pulps were nonvital and the periapical regions were consistently radiolucent. Five teeth were symptomatic (pain) and were clinically diagnosed as presenting acute periapical inflammation. The other teeth were asymptomatic and



“Dense aggregates of bacteria sticking to the dentin wall”

“Microbial biofilms are populations of microorganisms that are concentrated at an interface and typically surrounded by an extracellular polymeric substance matrix”.

Hall-Stoodley L, Costerton JW, Stoodley P. Bacterial biofilms: from the natural environment to infectious diseases. Nat Rev Microbiol 2004; 2: 95-108.

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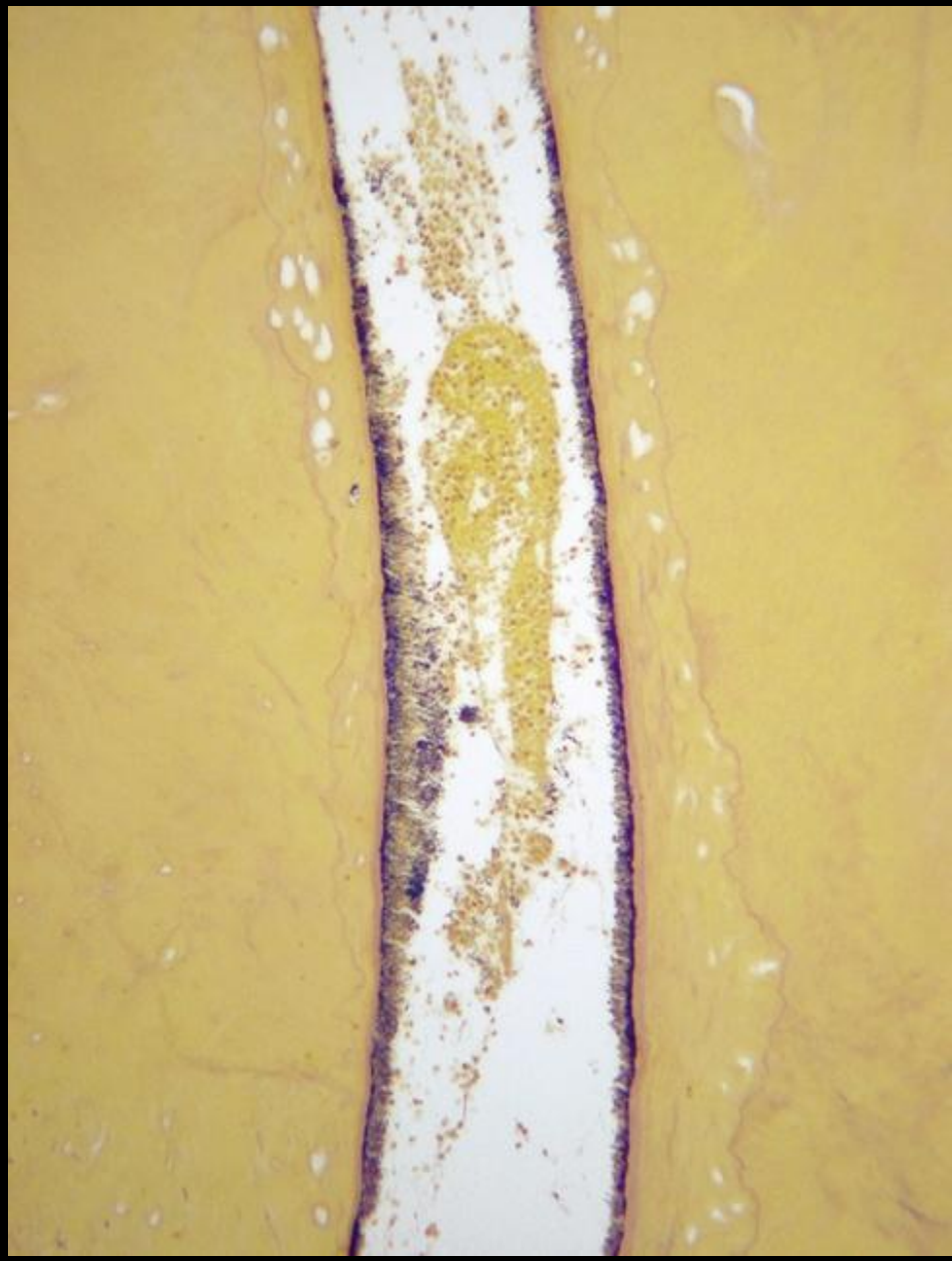

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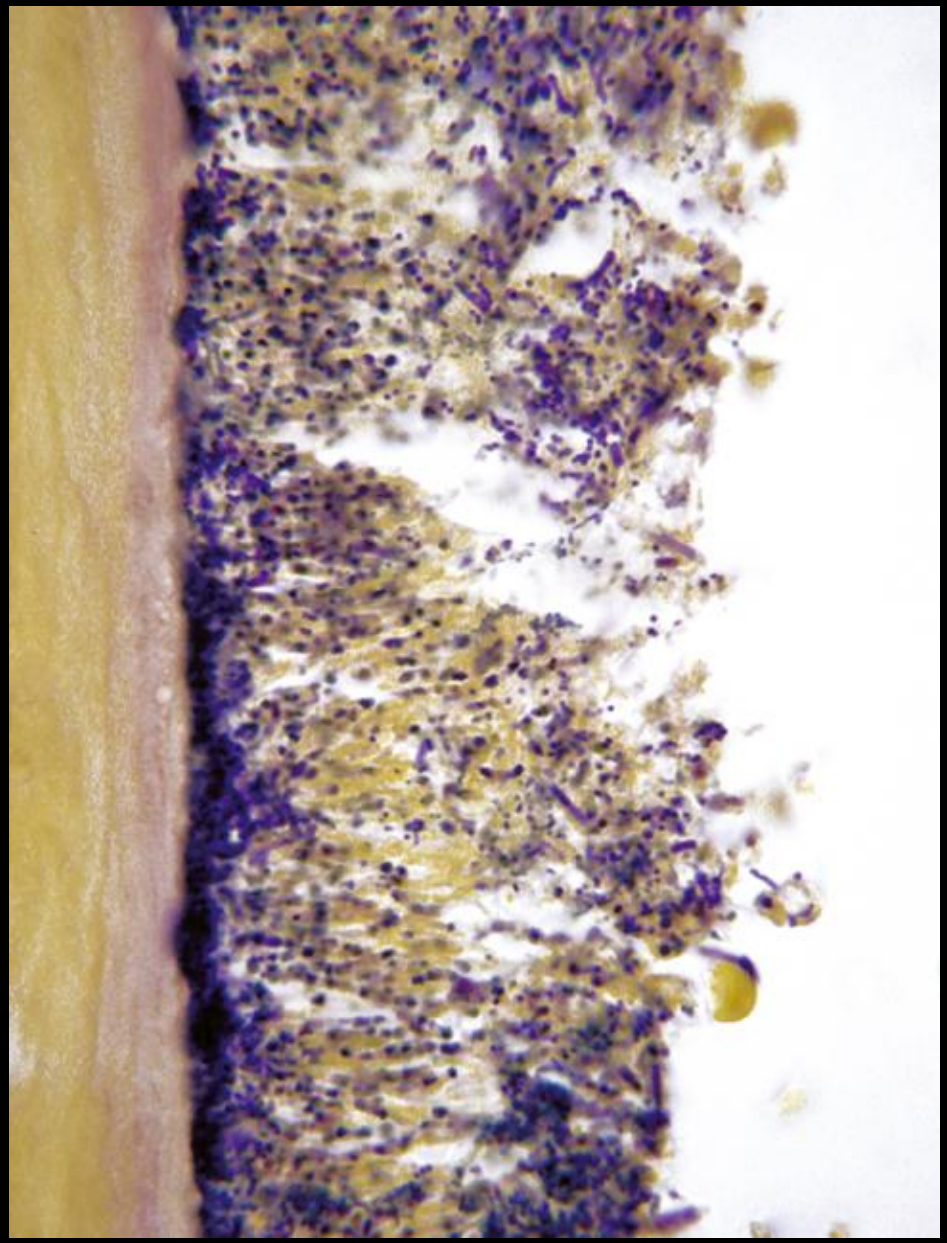

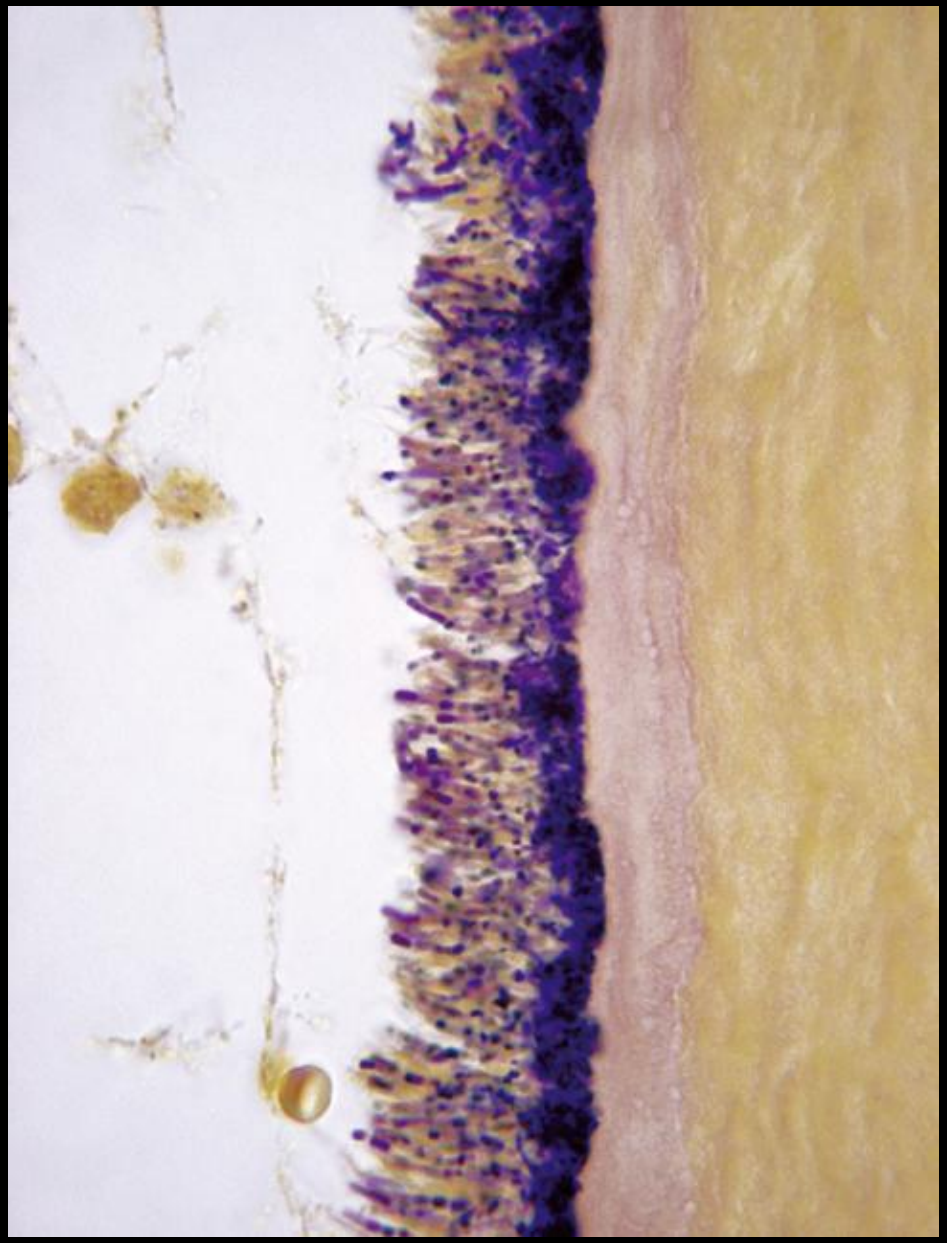

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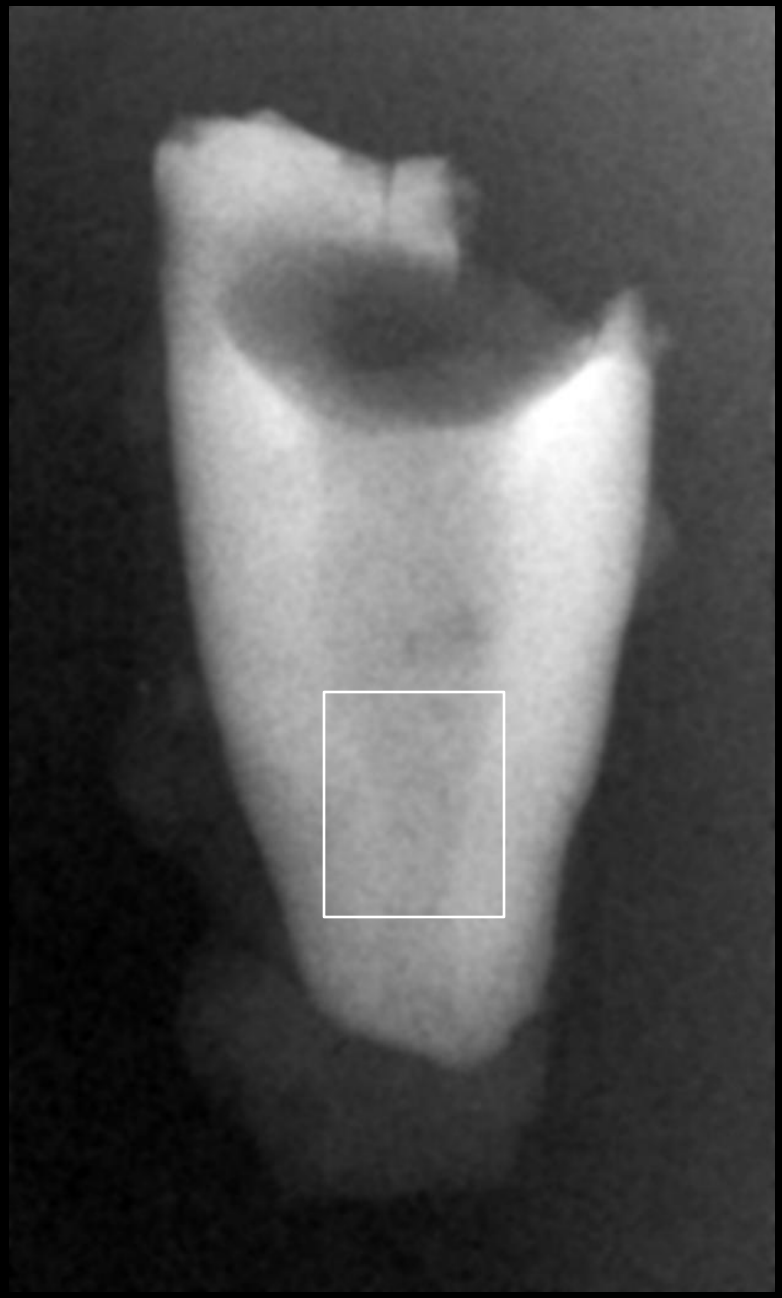

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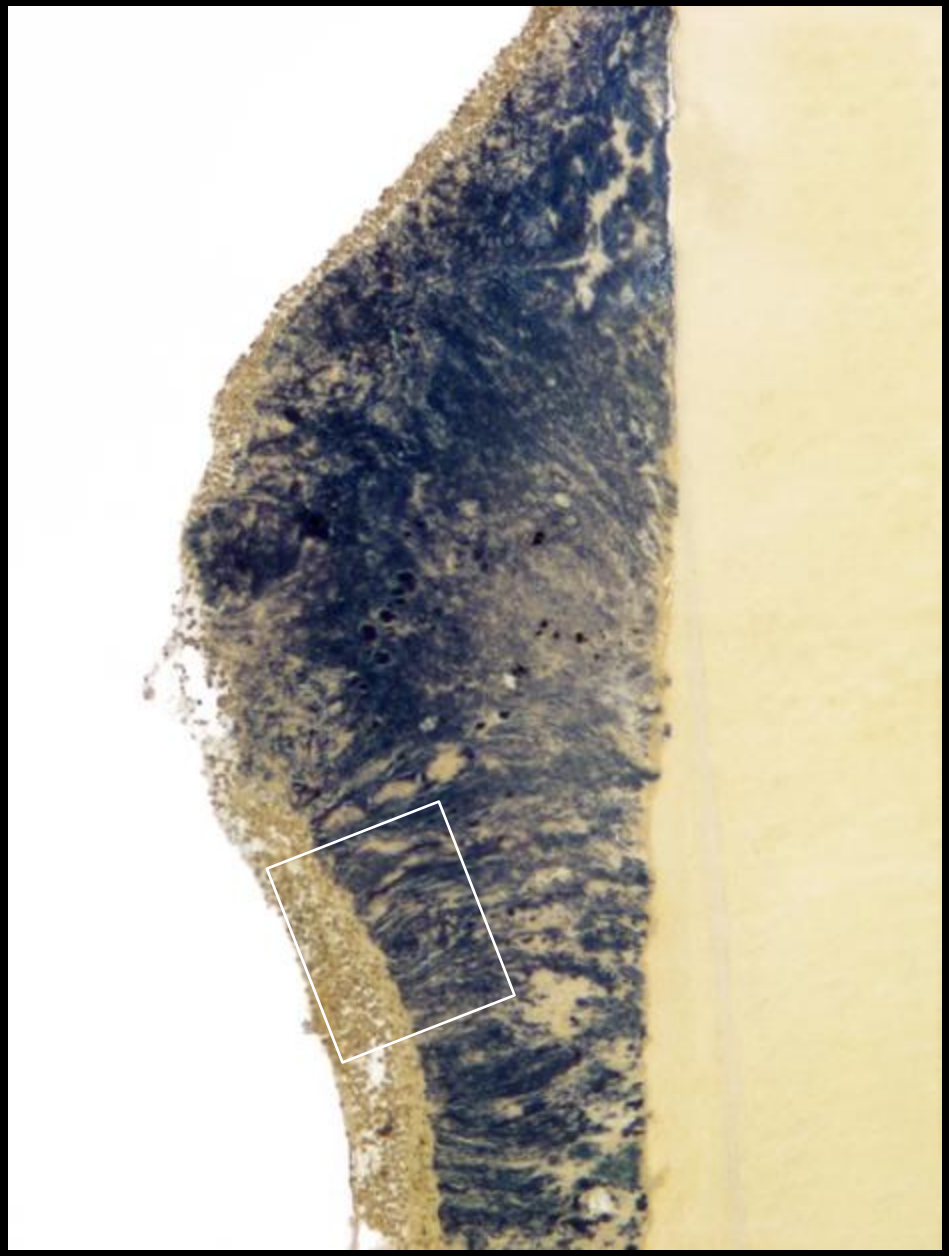

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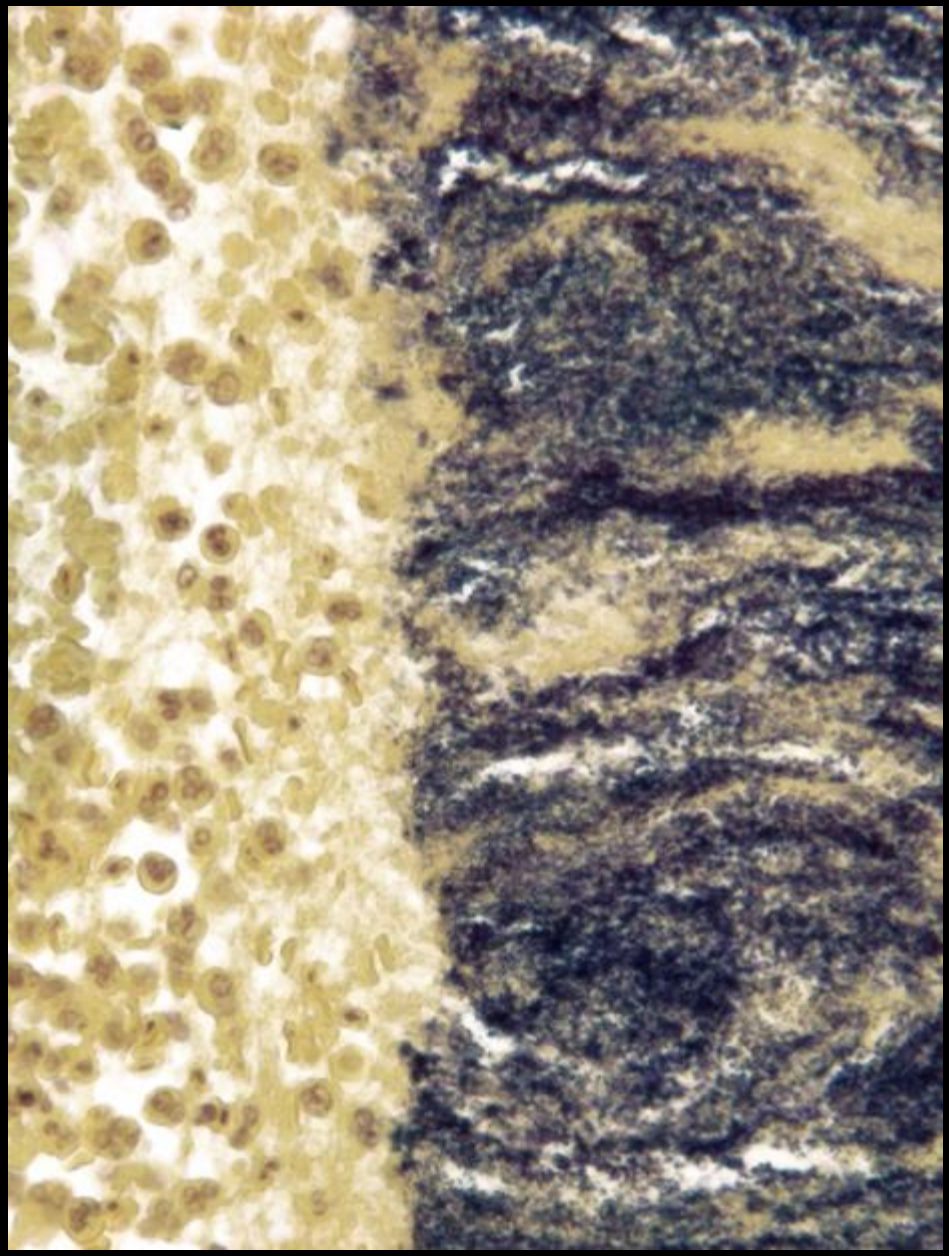

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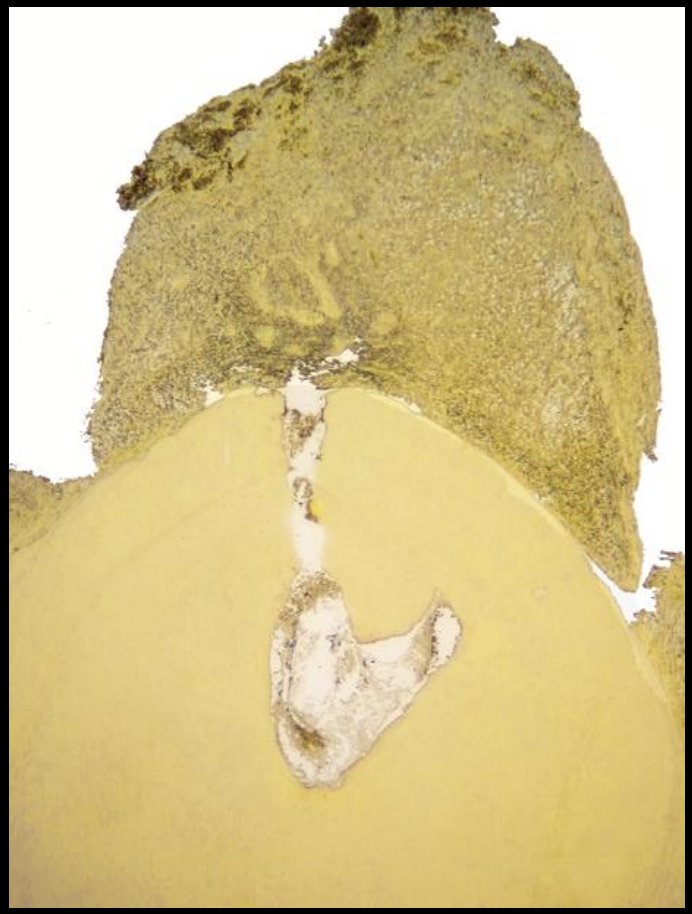
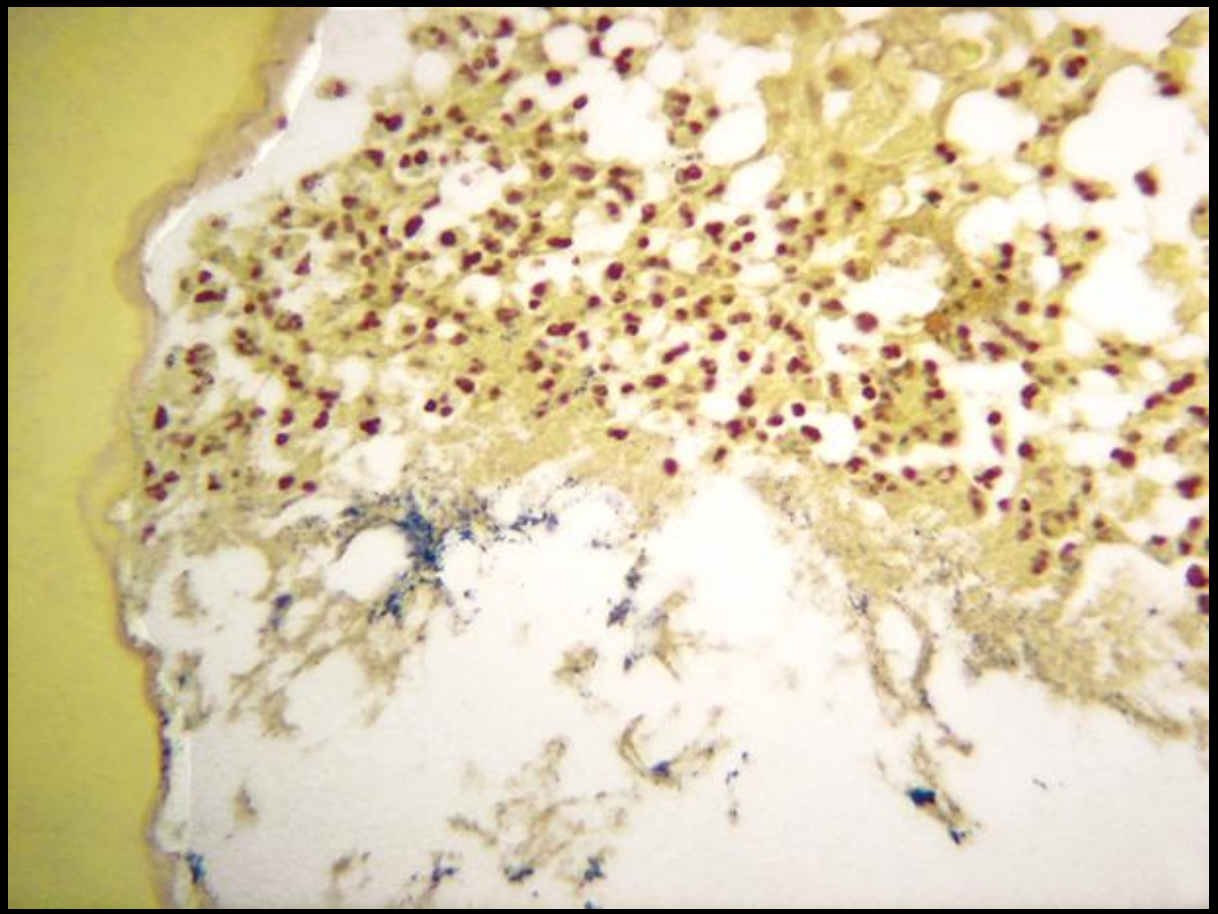
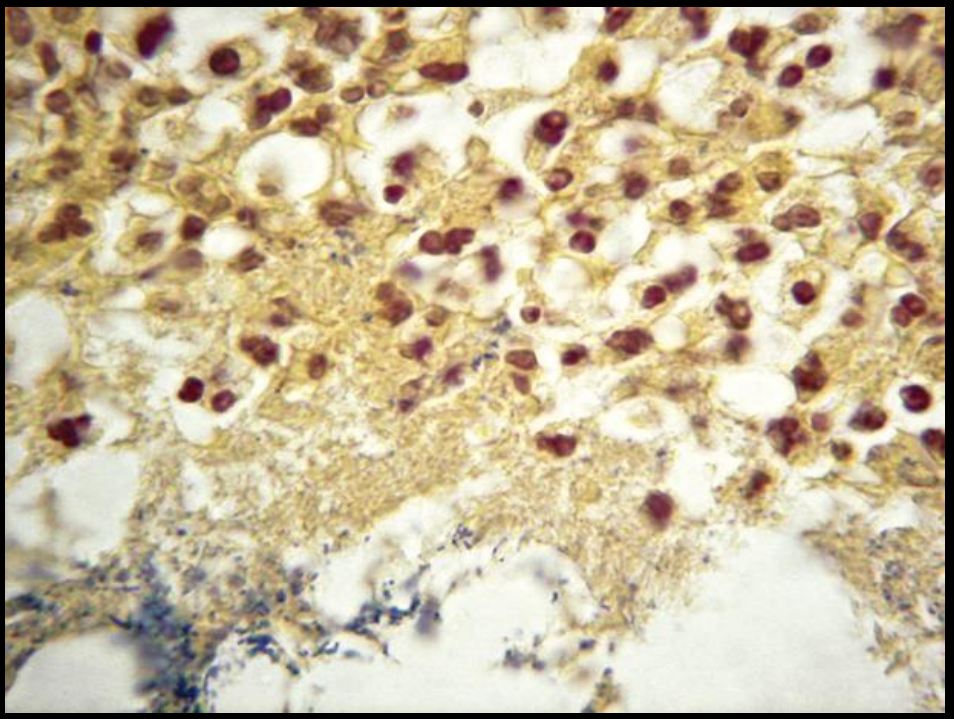
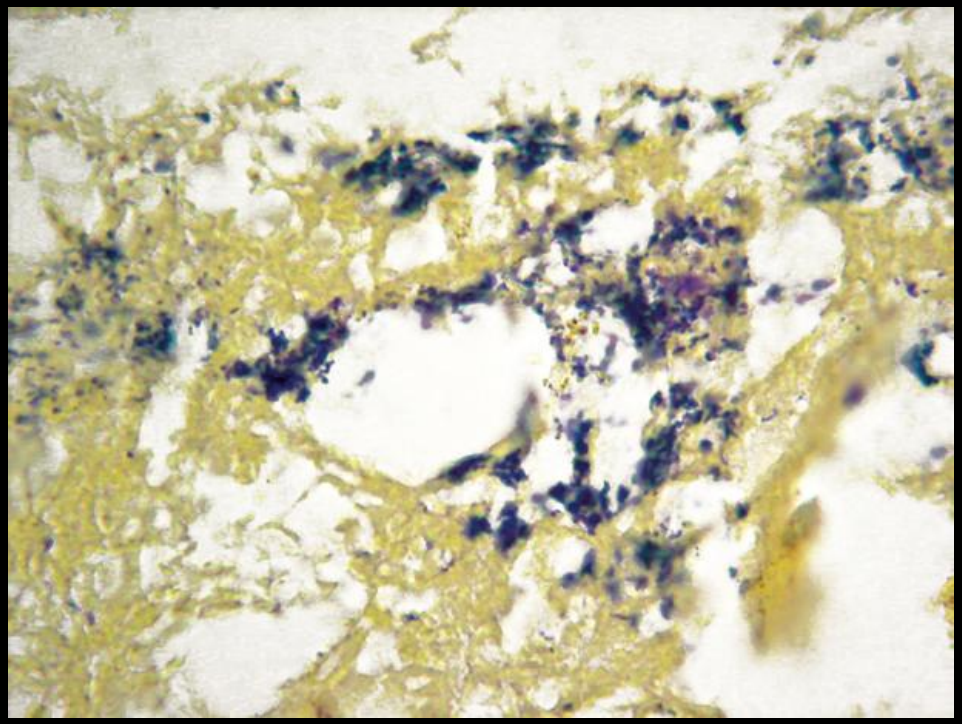




Bacteria are also seen in the lumen of the main canal, ramifications, and isthmuses as ***flocs*** and ***planktonic cells***, either intermixed with necrotic pulp tissue or possibly suspended in a fluid phase





Bacterial flocs in clinical specimens may originate from the growth of cell aggregates/co-aggregates in a fluid or they may have detached from biofilms. Flocs may exhibit many of the same characteristics as biofilms, and are sometimes regarded as “planktonic biofilms”.

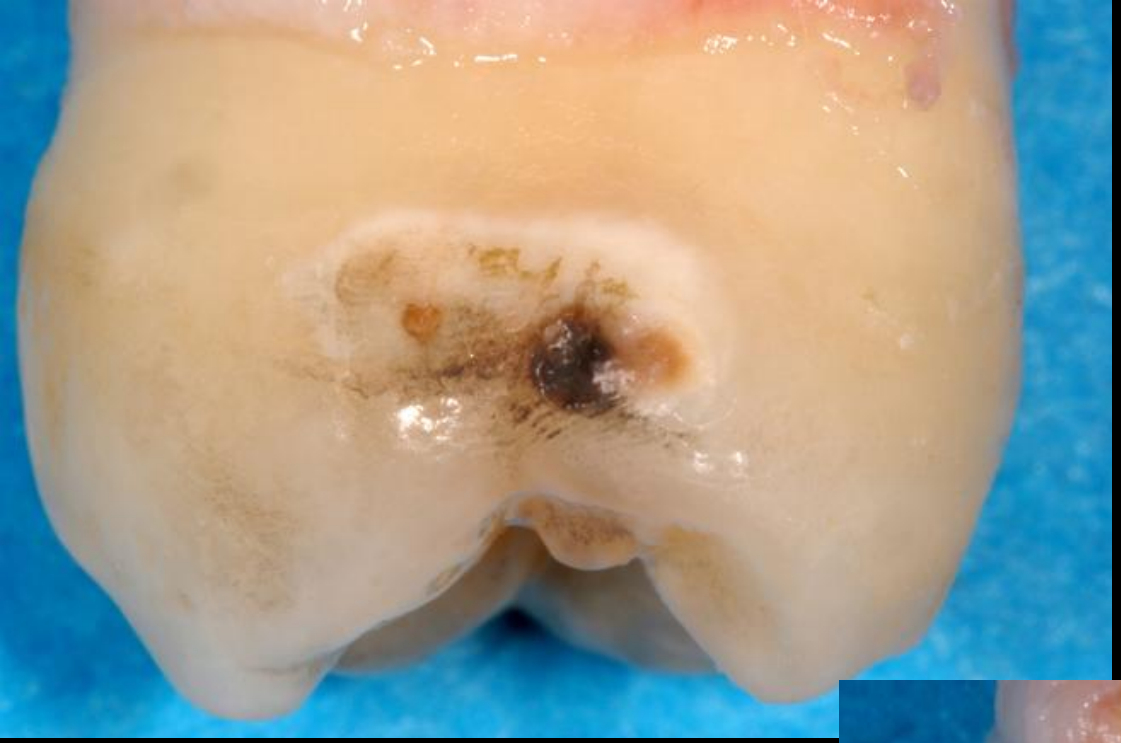
Siqueira JF, Rôças IN, Ricucci D. Biofilms in endodontic infection. Endodontic Topics 2012; 22:33-49.

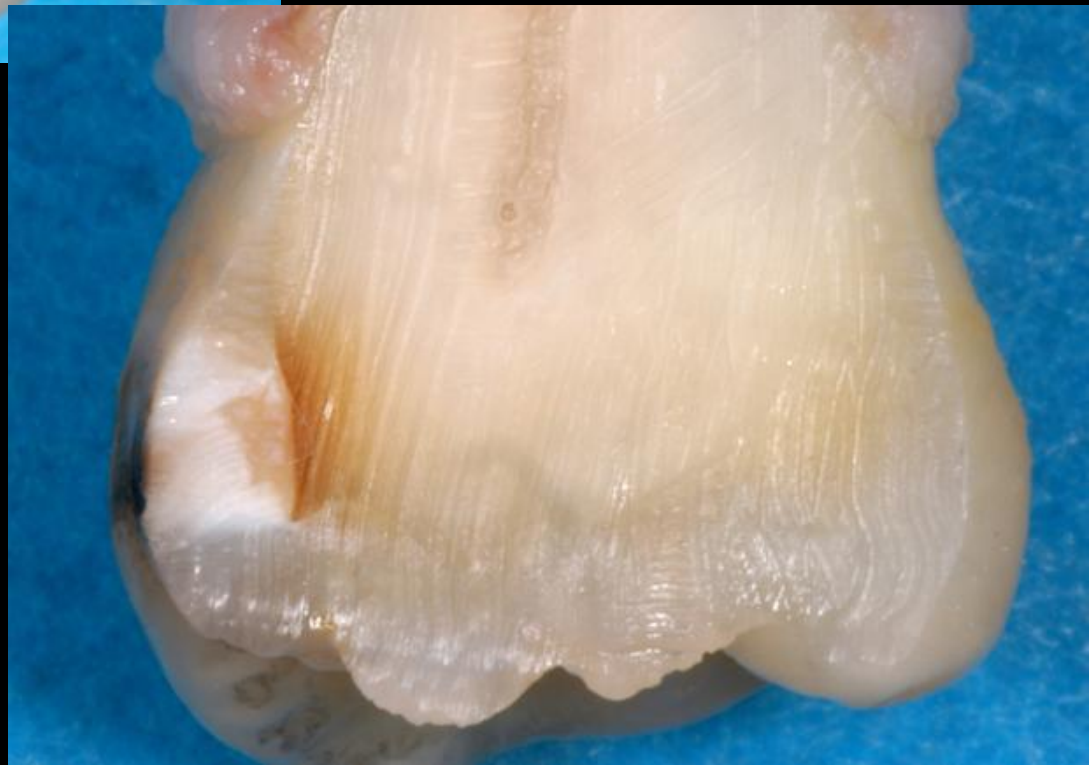
The biofilm community lifestyle provides microorganisms with a series of advantages and skills that are not observed for individual cells living in a free-floating (planktonic) state:

- **Establishment of a broader habitat range for growth;**
- **Increased metabolic diversity and efficiency;**
- **Protection against competing microorganisms, host defenses, antimicrobial agents, and environmental stress;**
- **Enhanced pathogenicity.**

ETIOLOGY OF PULPAL DISEASE

NATURAL	CARIES
	PERIODONTAL DISEASE
	PERIODONTAL DISEASE / CARIES
	ATTRITION
	PHYSICAL TRAUMA
	NEOPLASM
IATROGENIC	CAVITY PREPARATION WET / DRY
	MEDICAMENTS
	LINERS
	BASES
	TEMPORARY DRESSINGS
	IMPRESSION MATERIALS
	RESTORATIVE SYSTEMS





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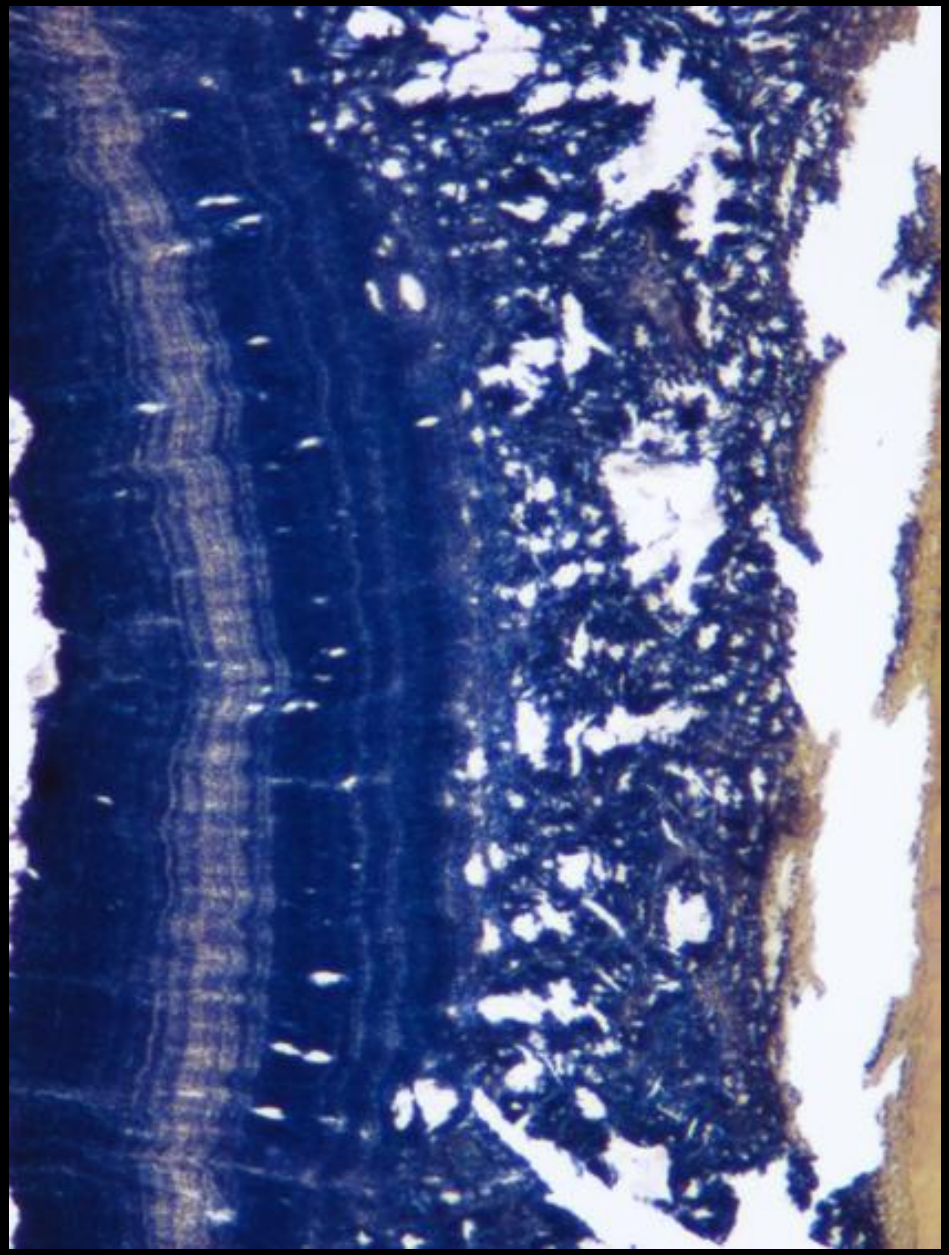

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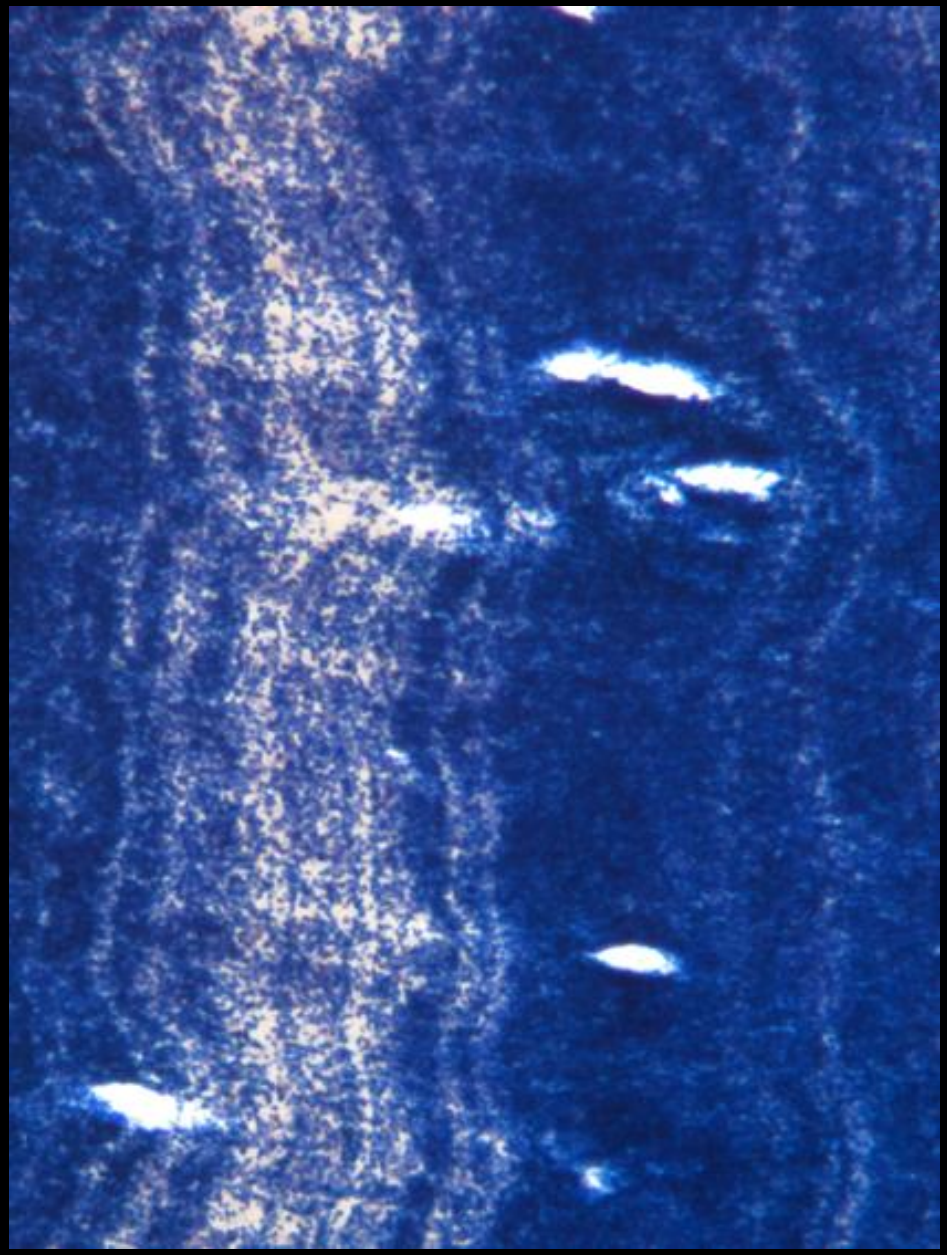

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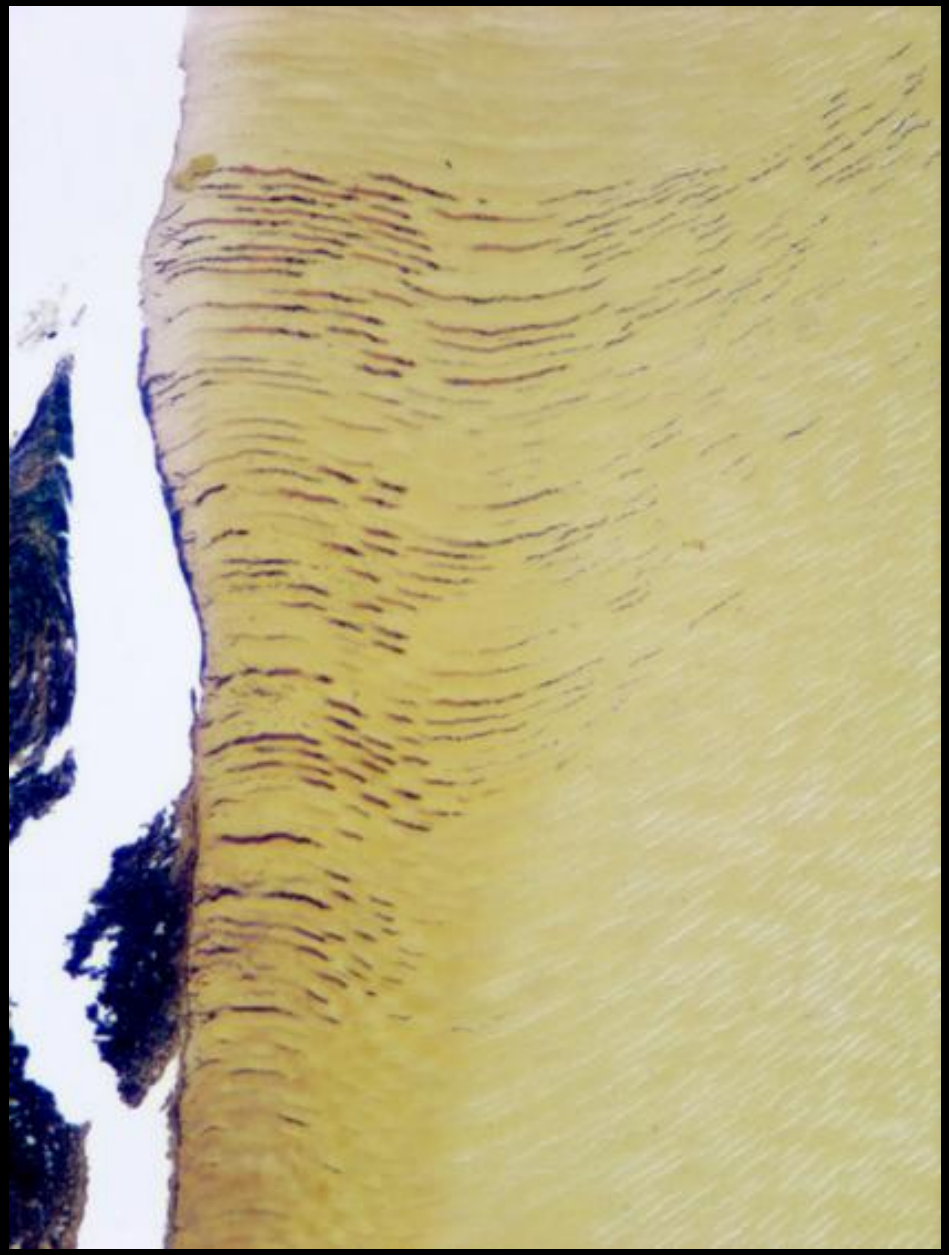

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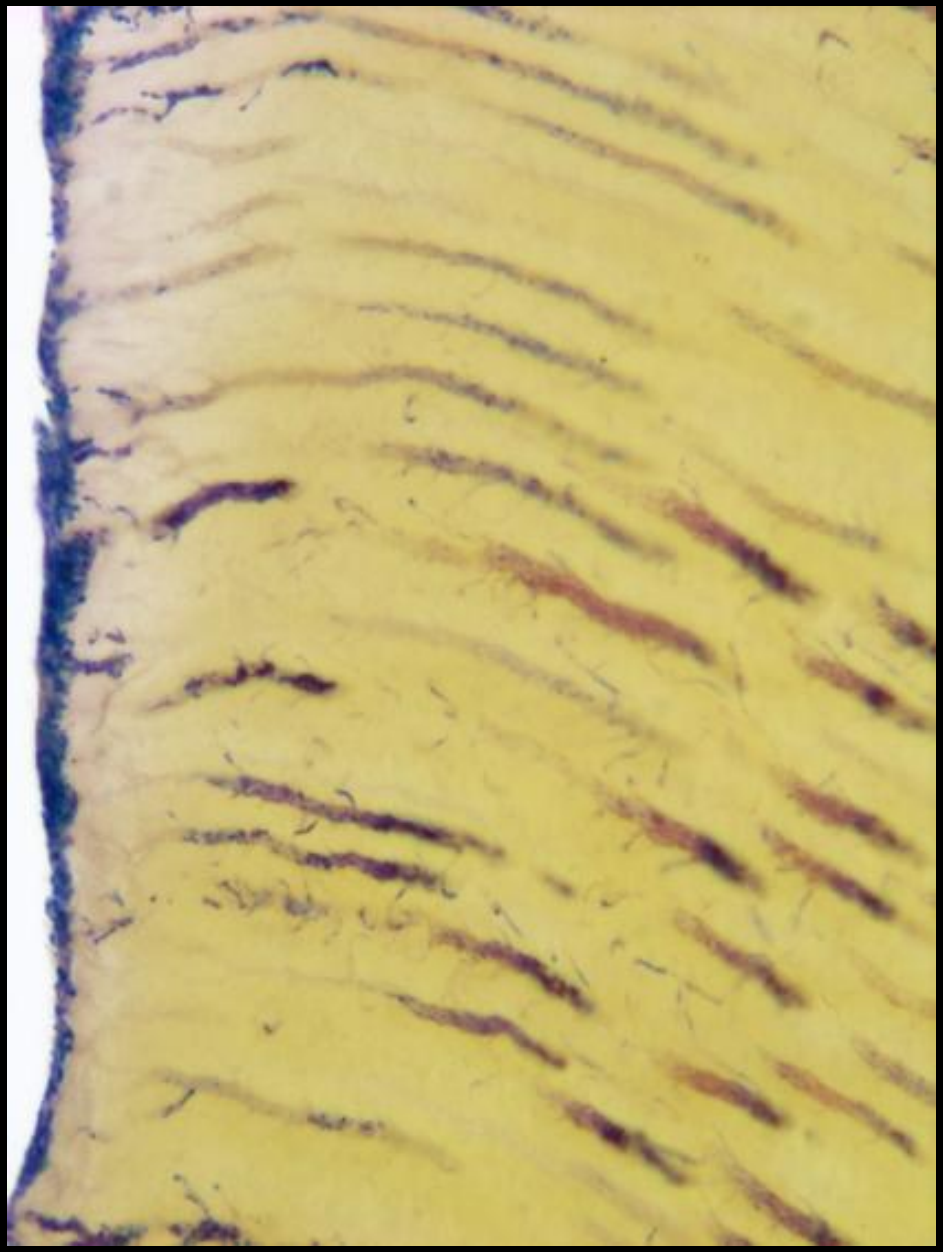

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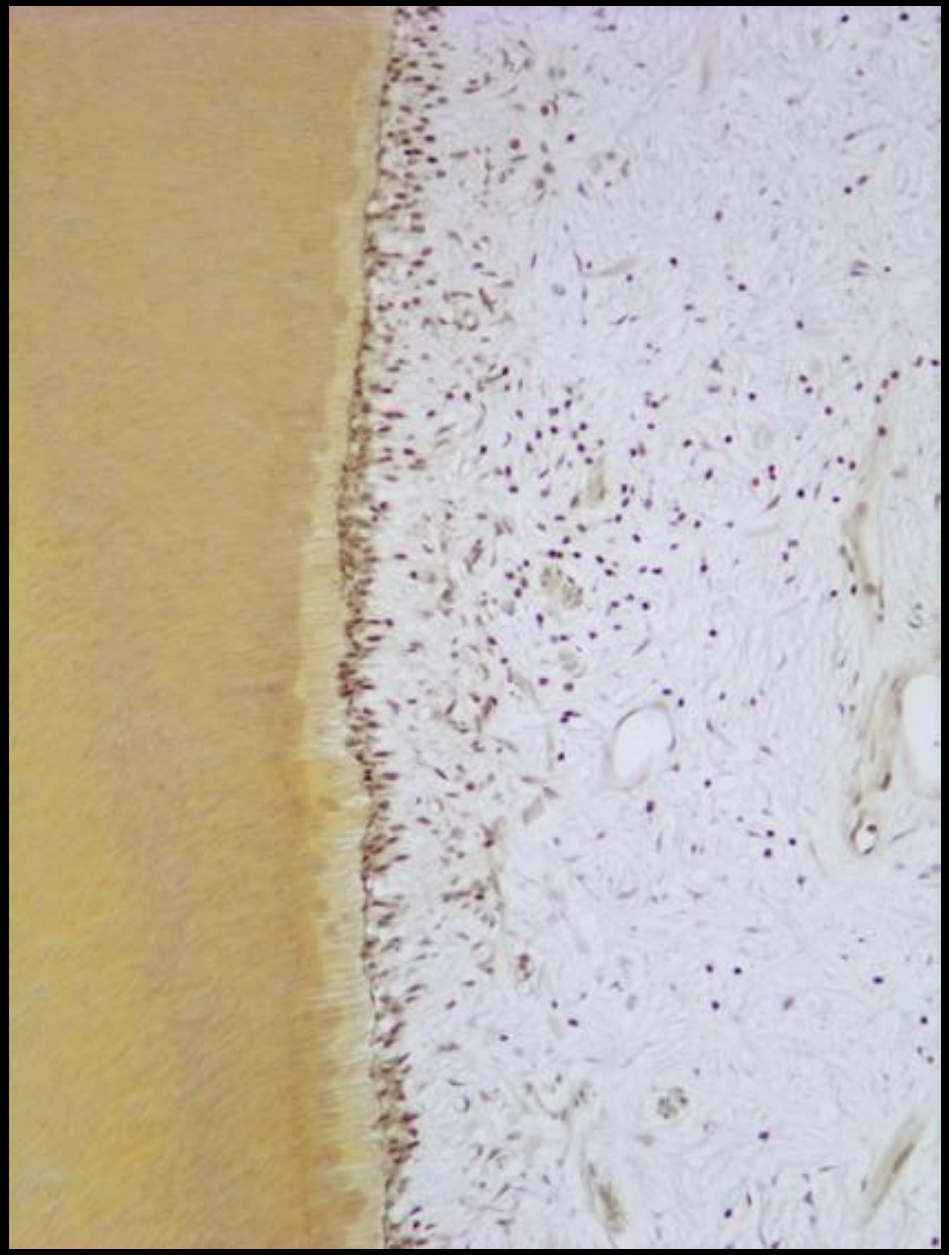

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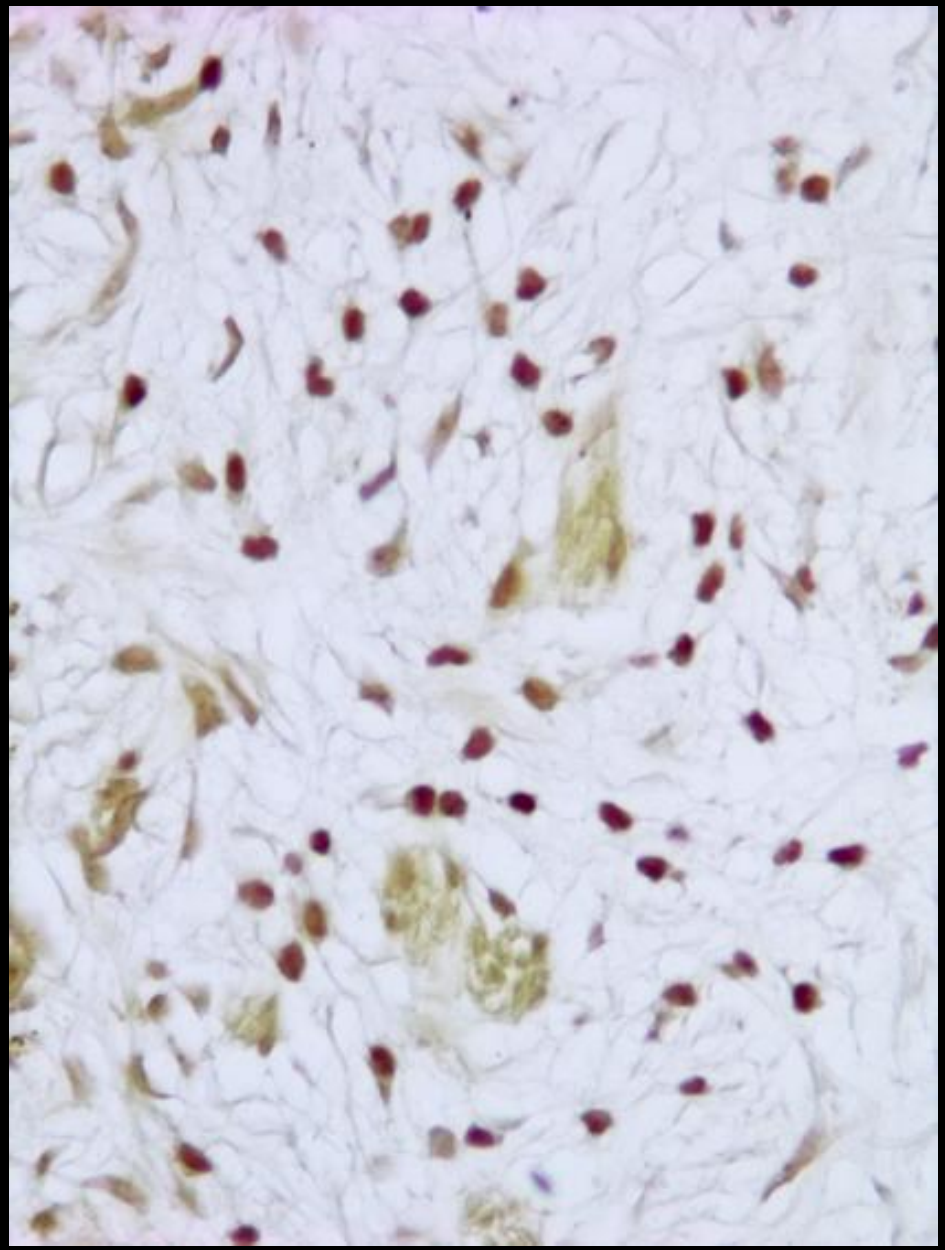

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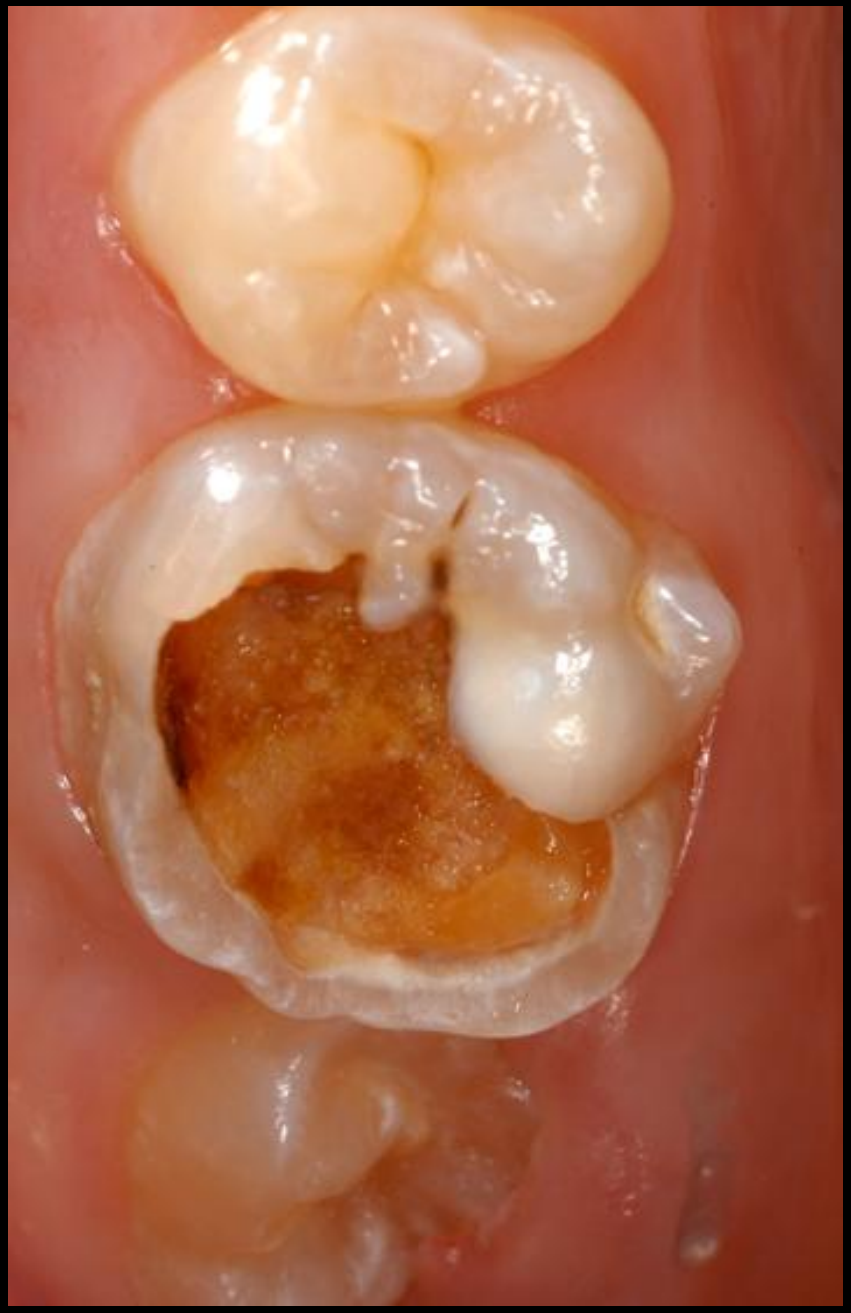
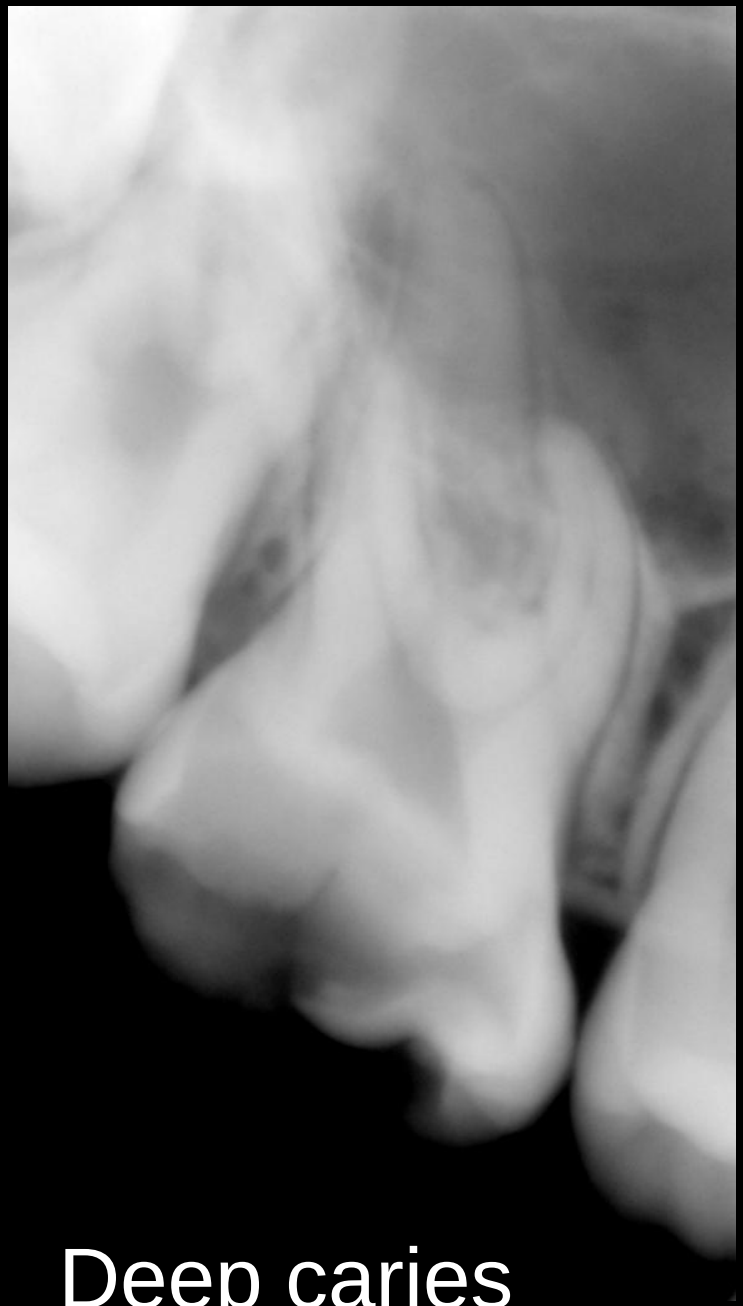
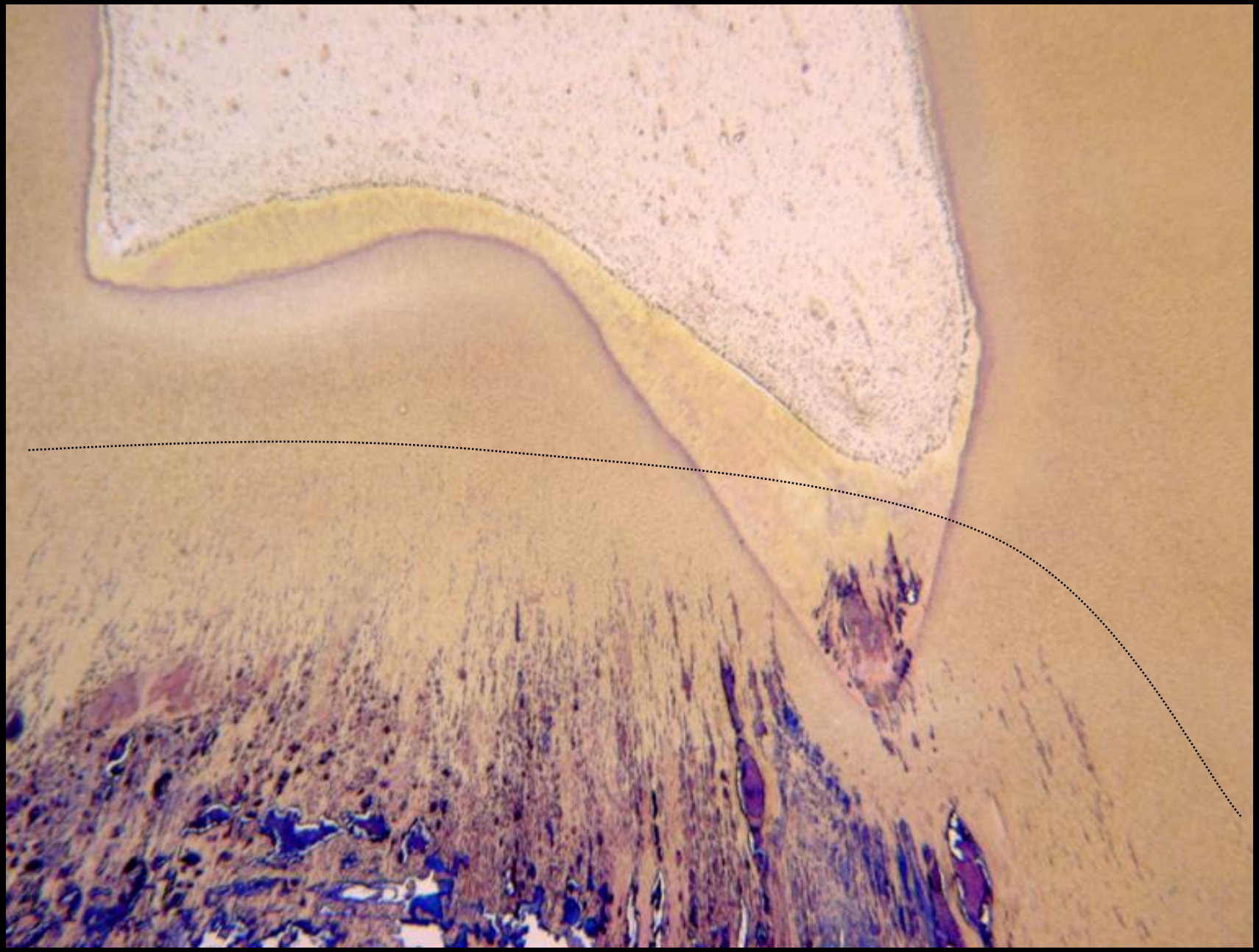
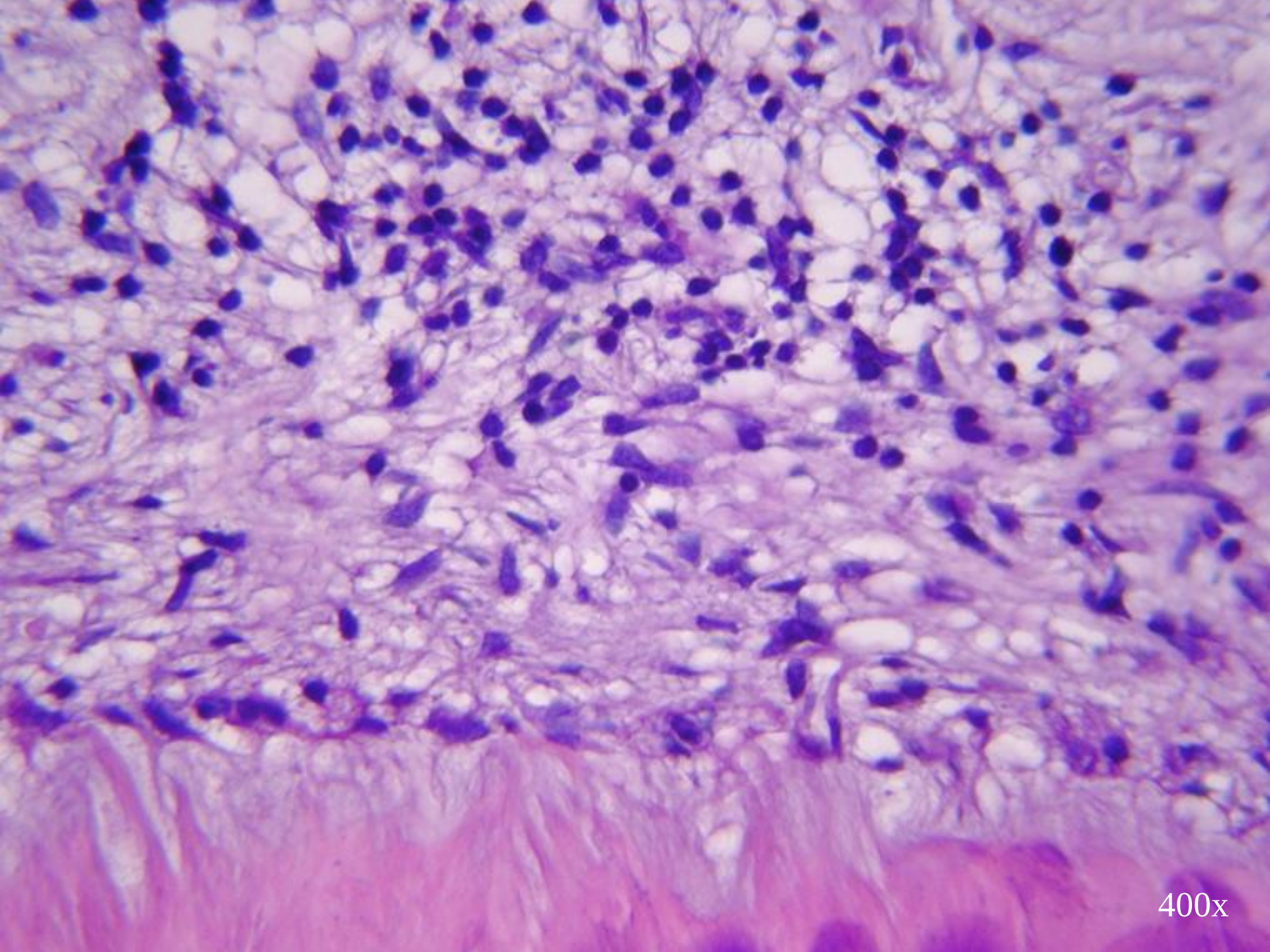




Deep caries



Reversible pulp inflammation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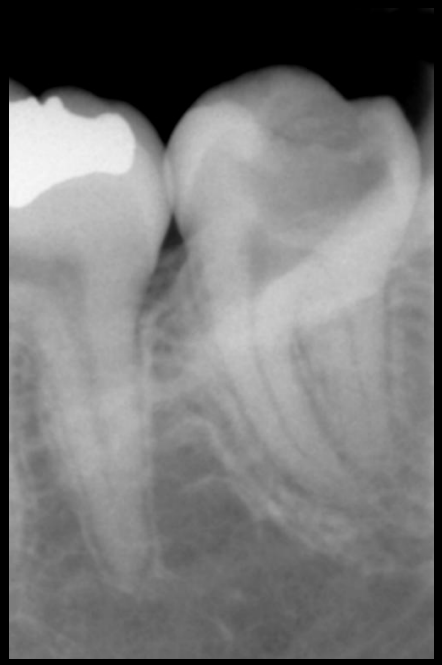
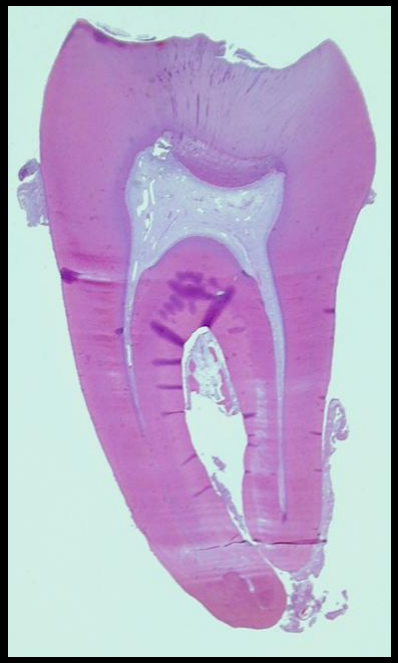
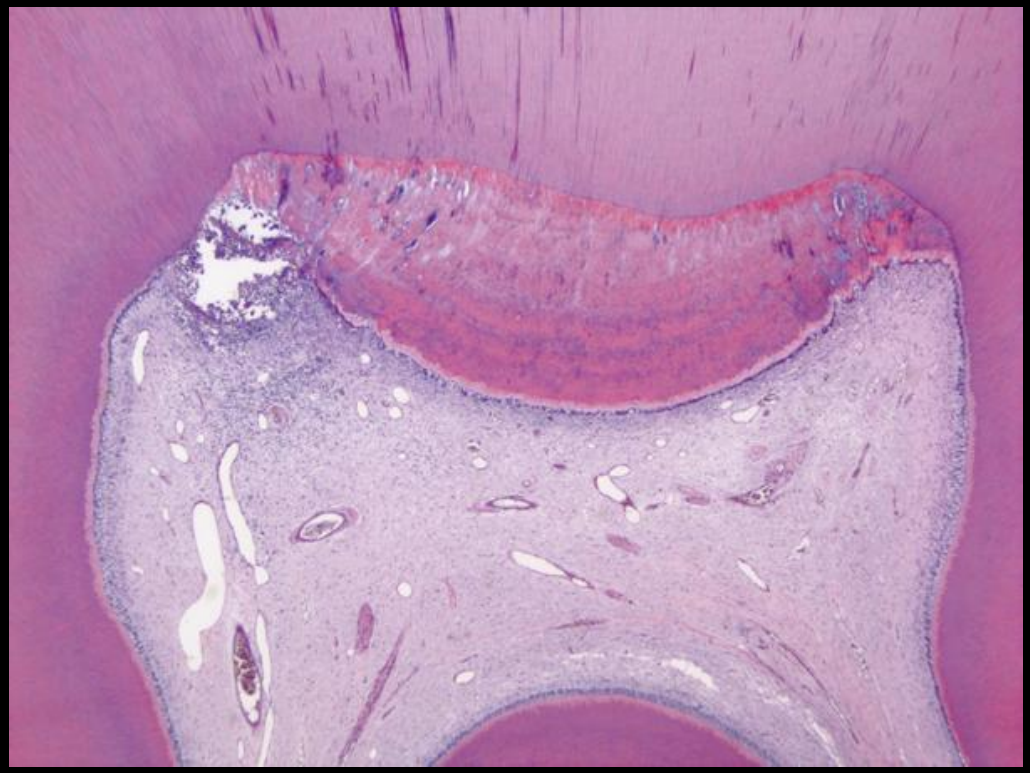
Pulp penetration by caries

Pulp penetration by caries







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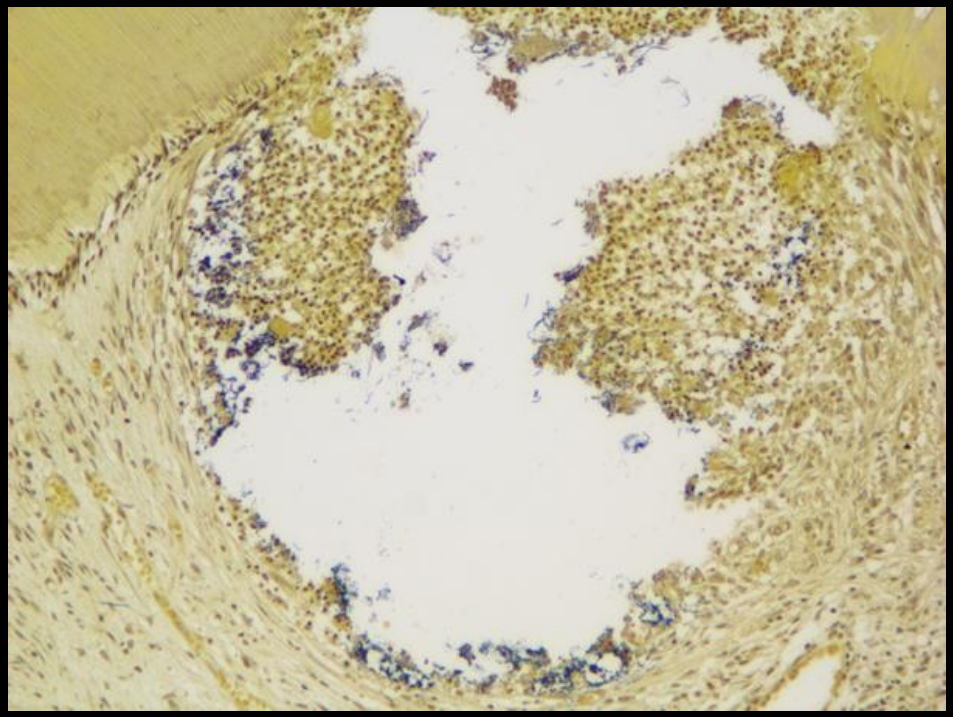

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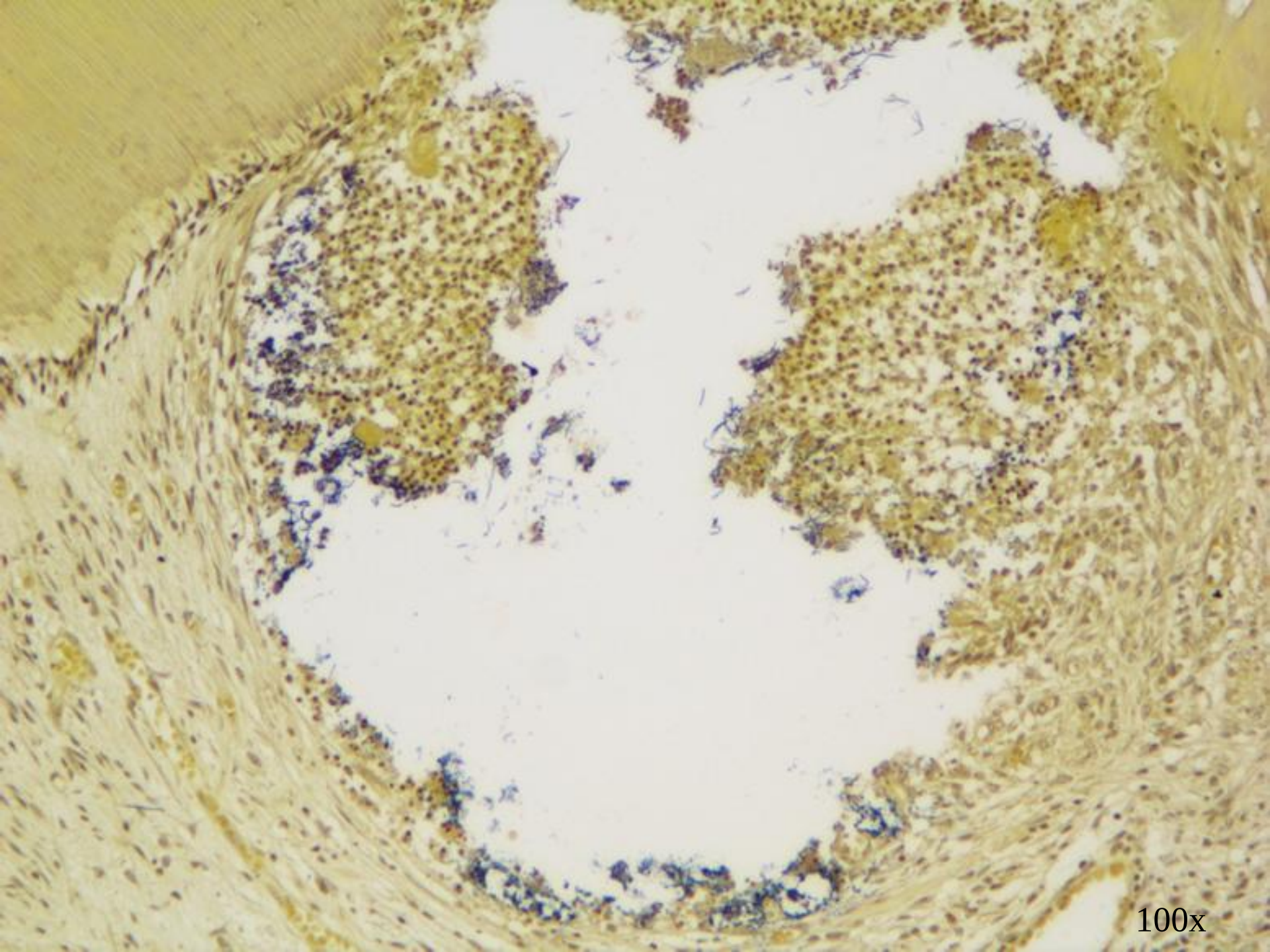
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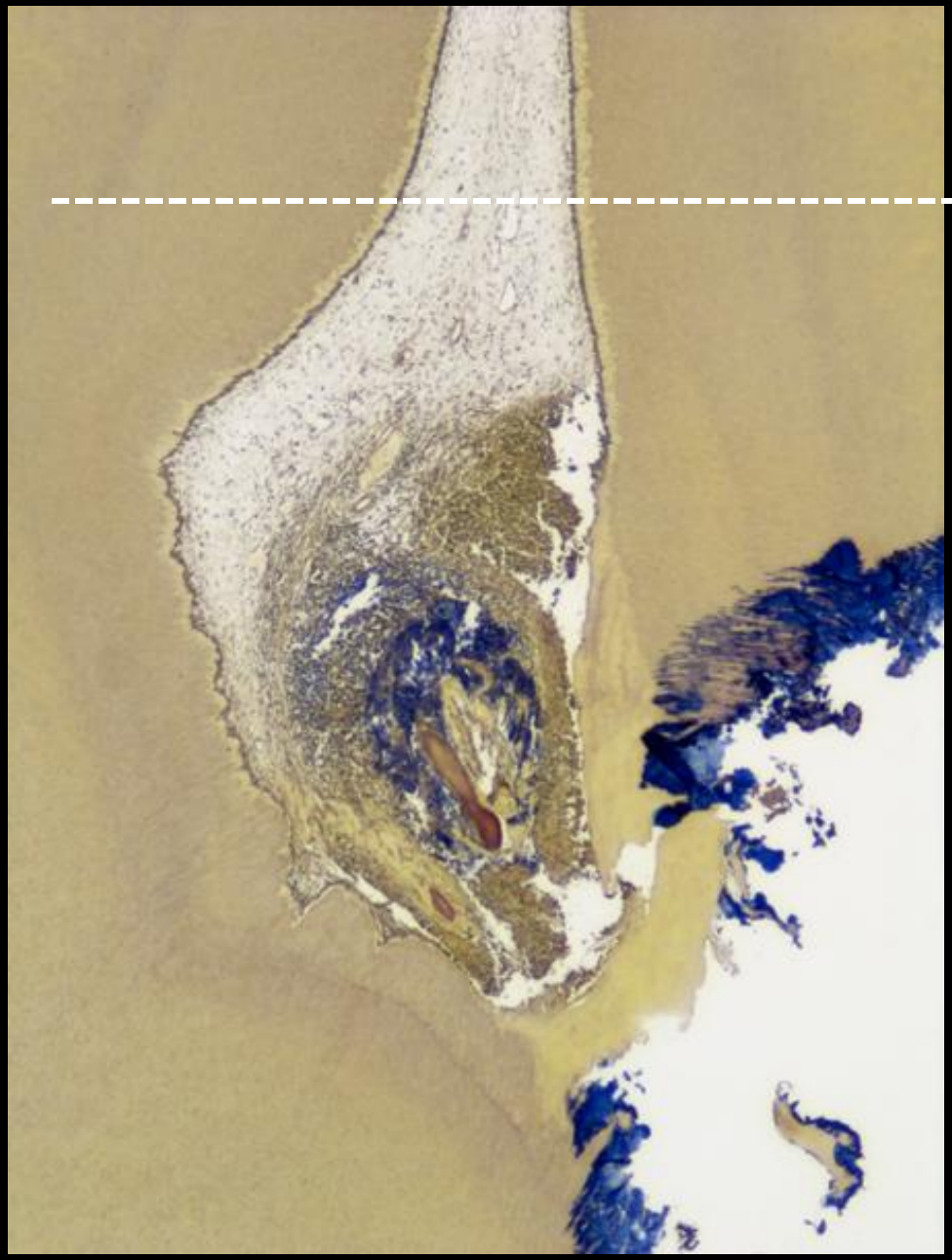
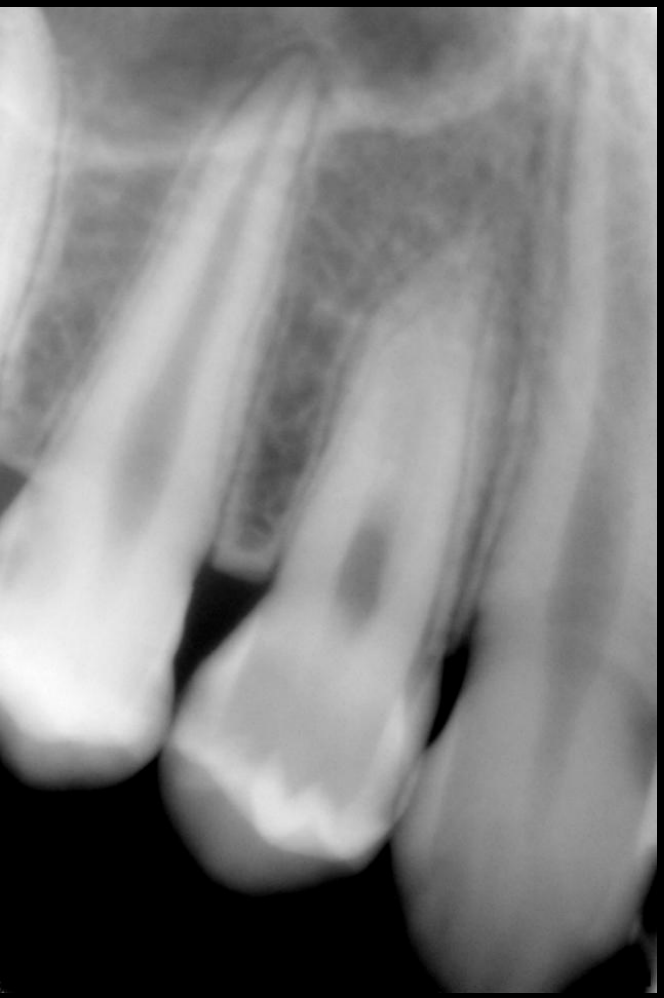
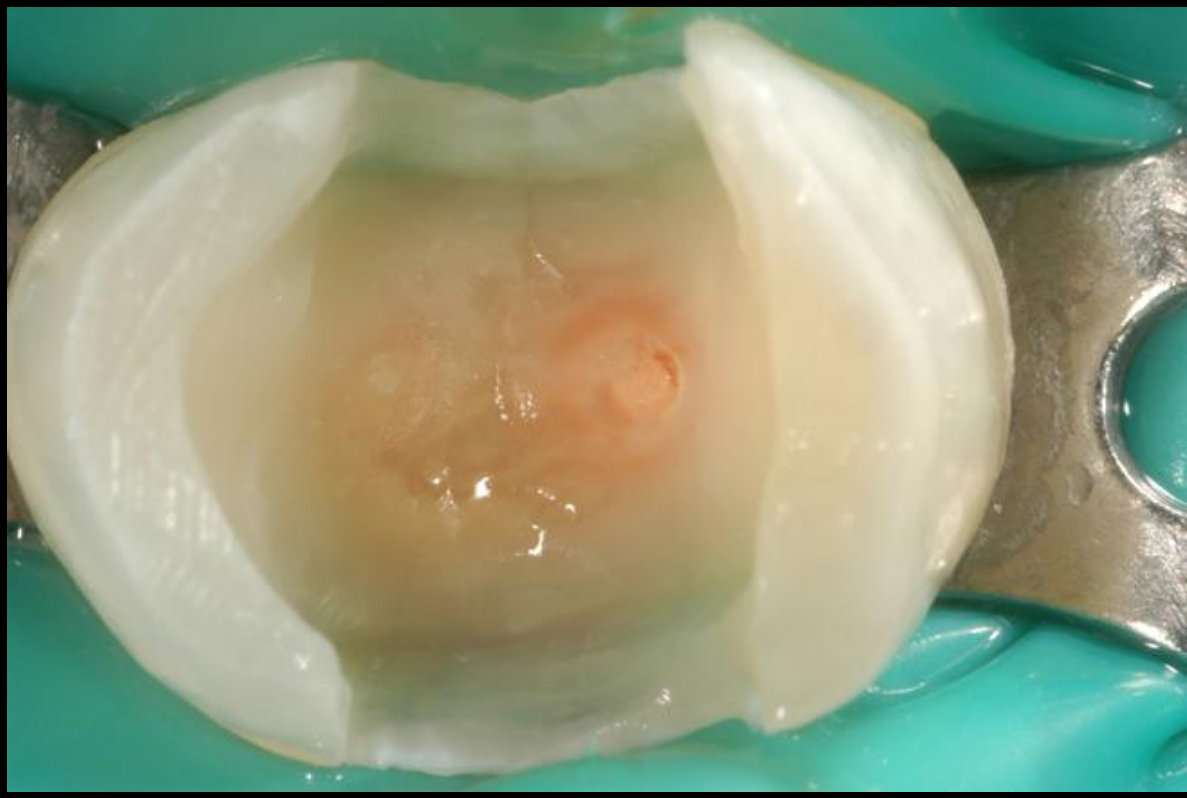
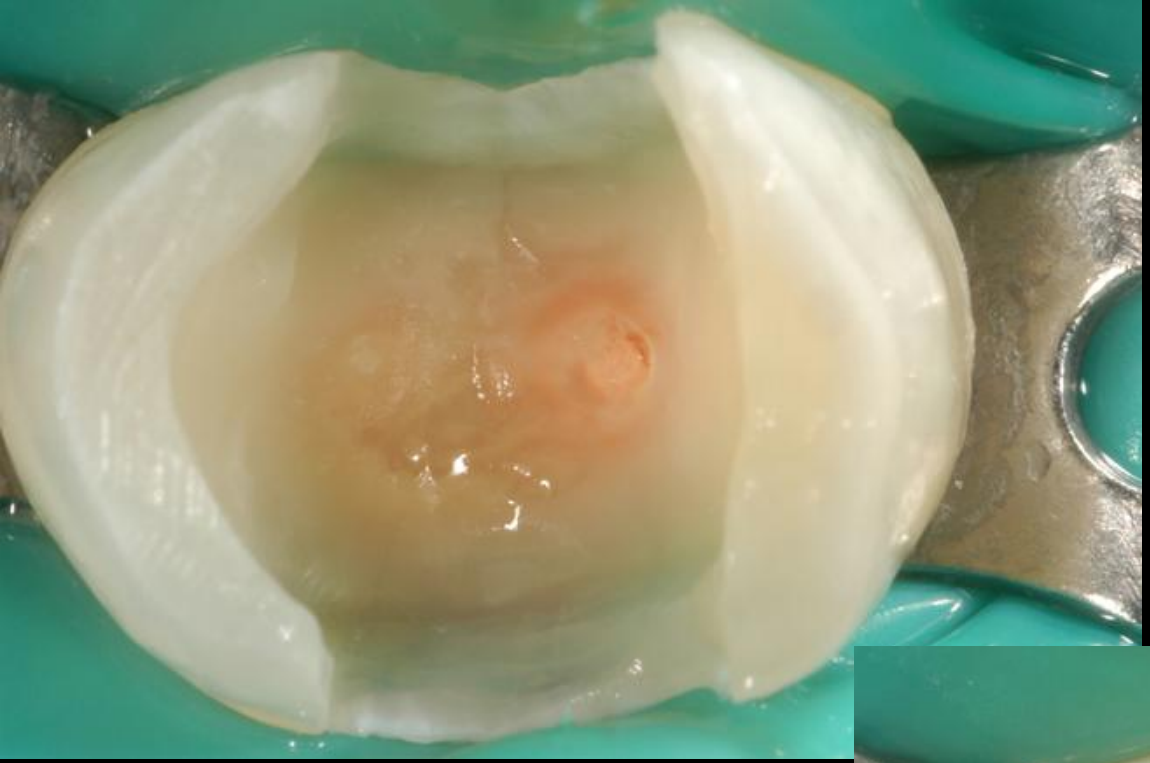
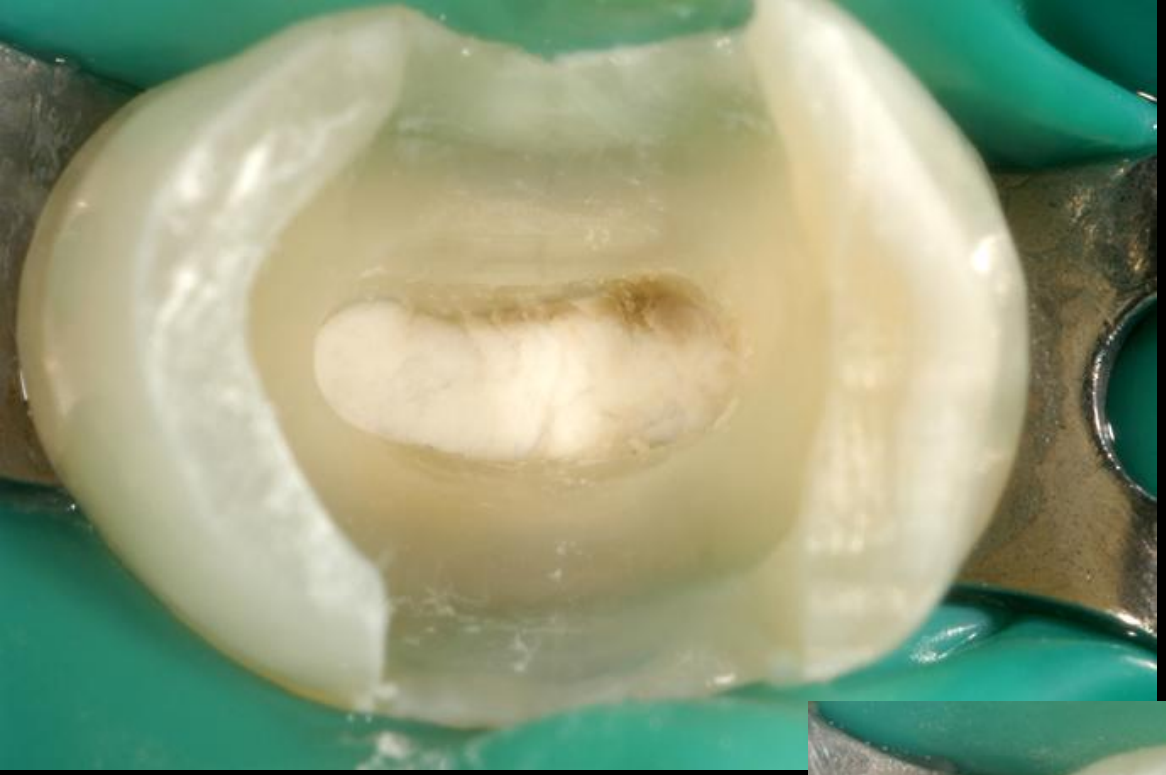


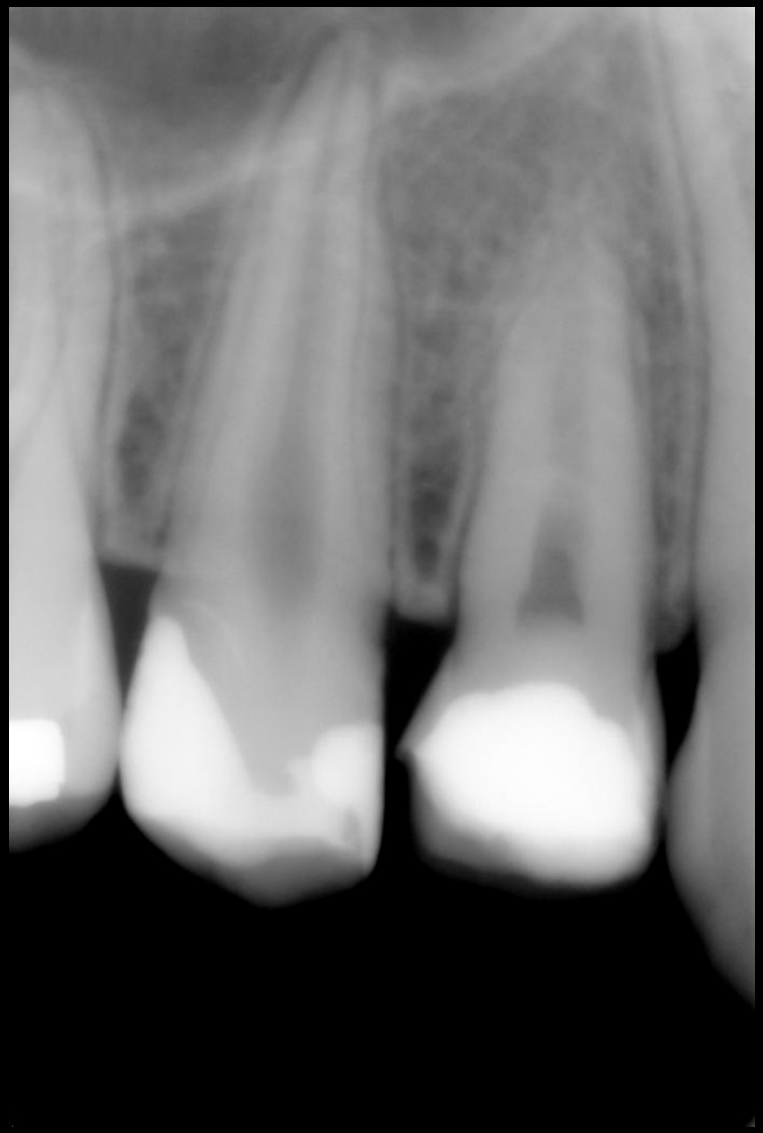
Pulpotomy???

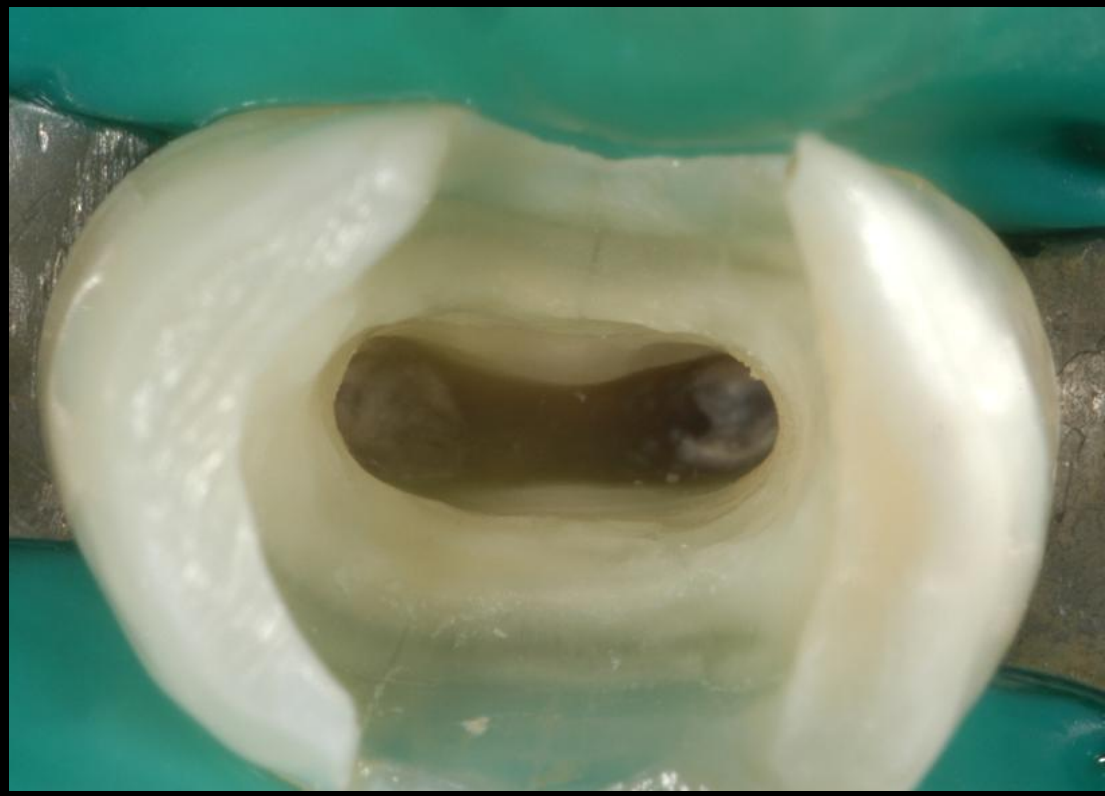




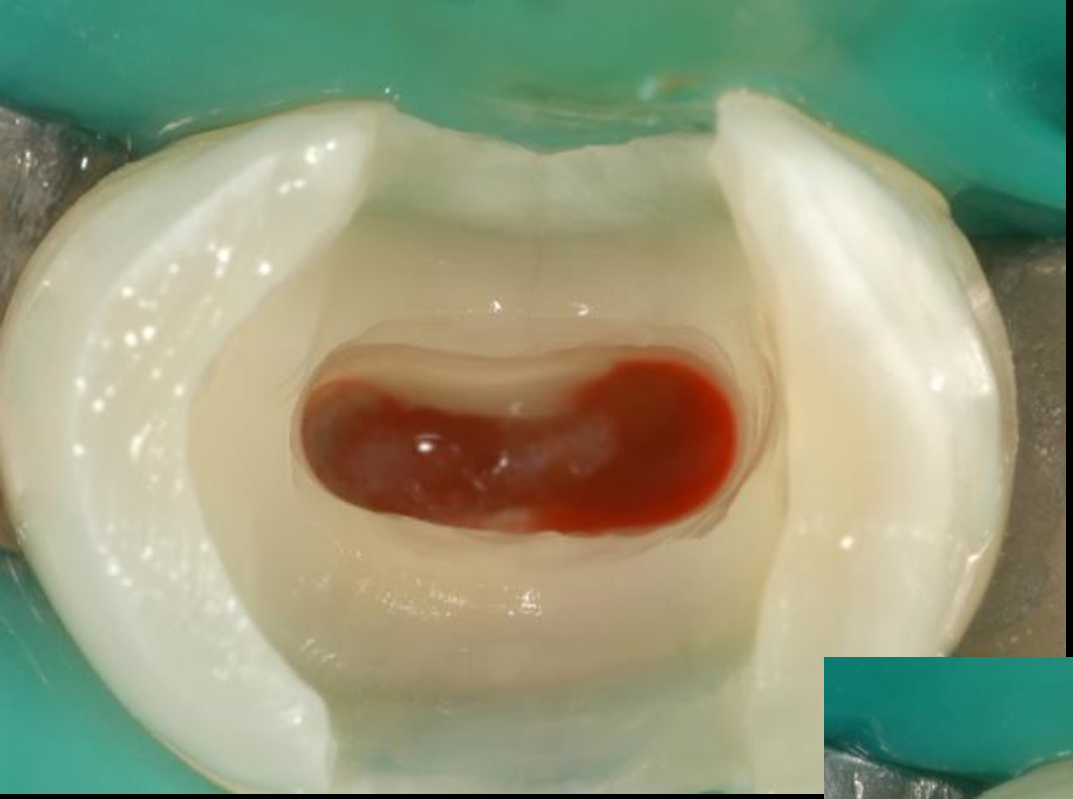


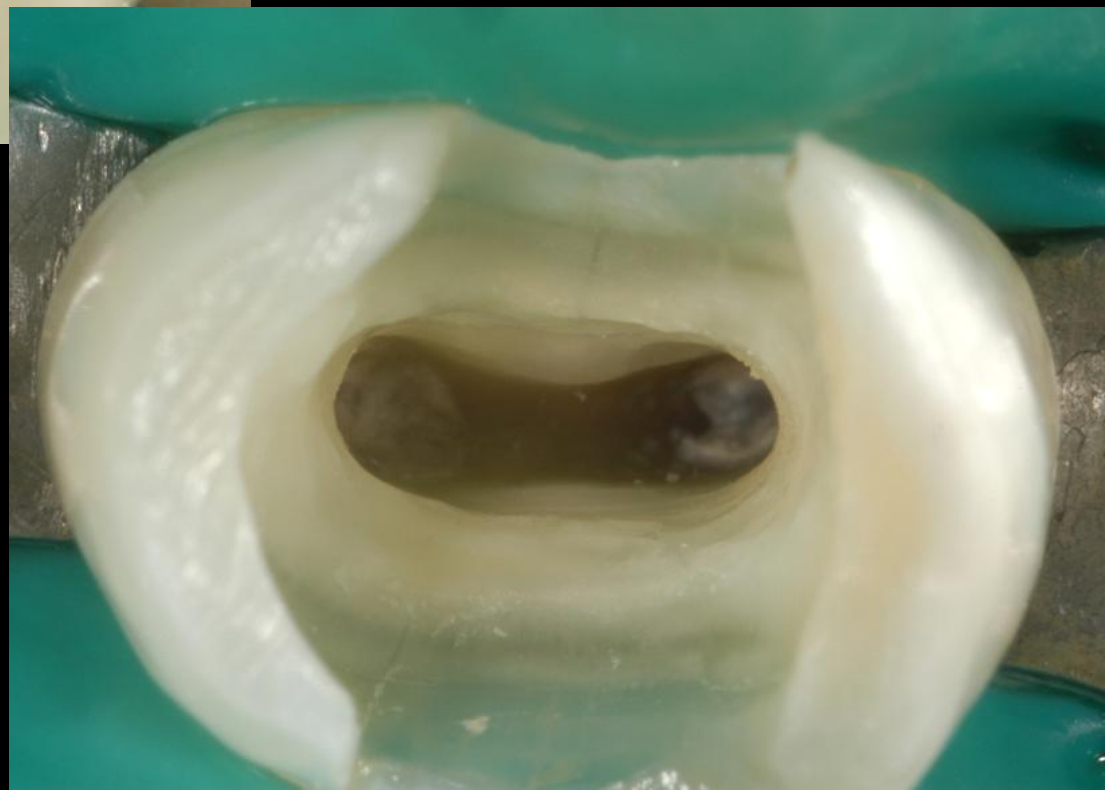






98 days





98 days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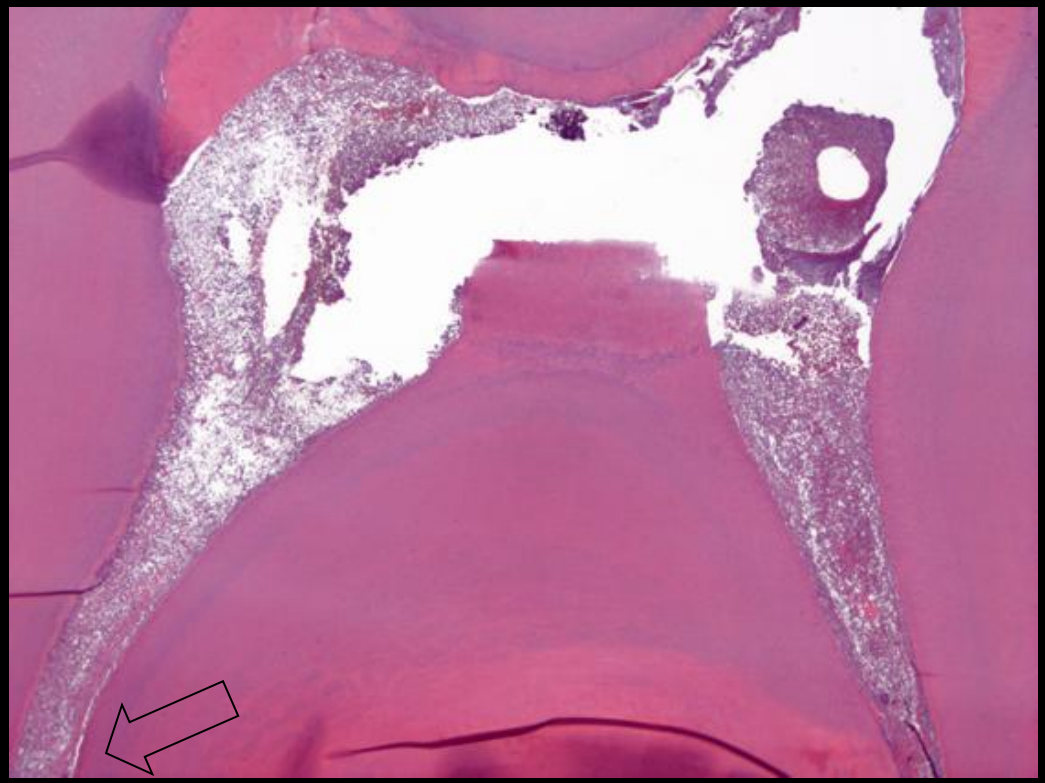

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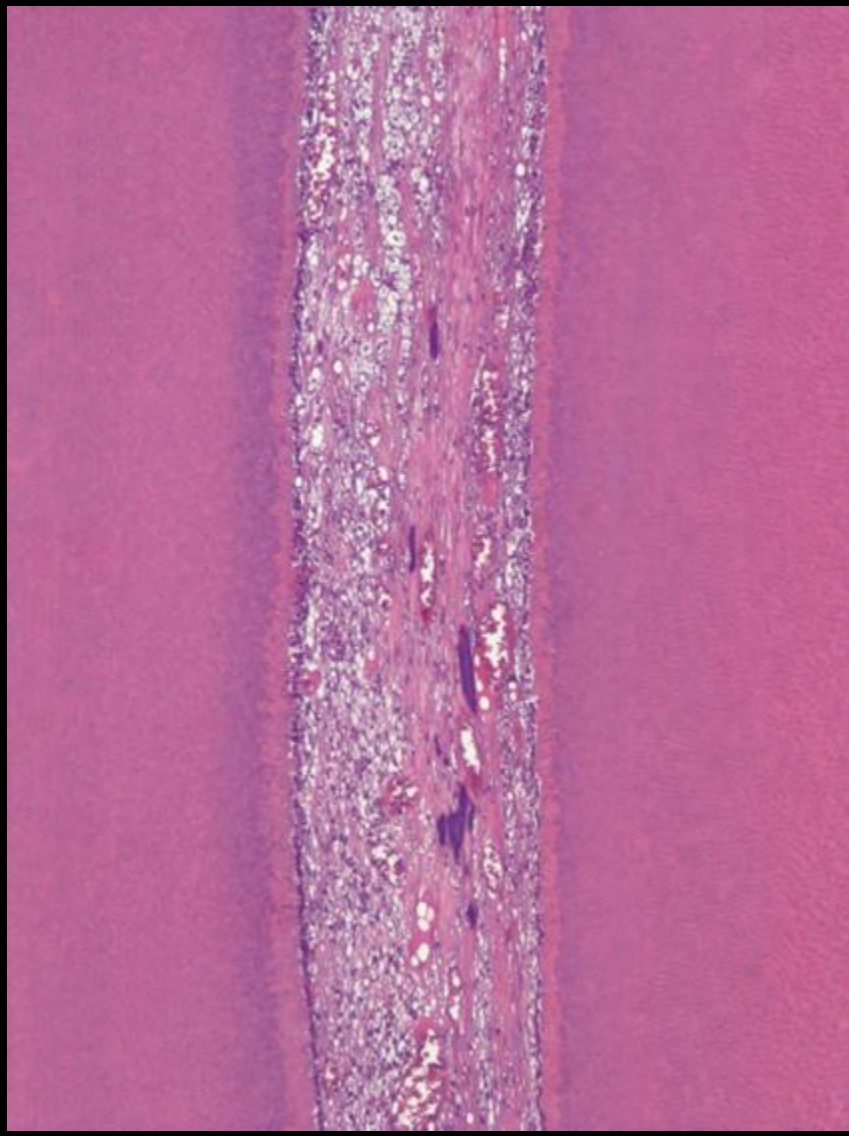

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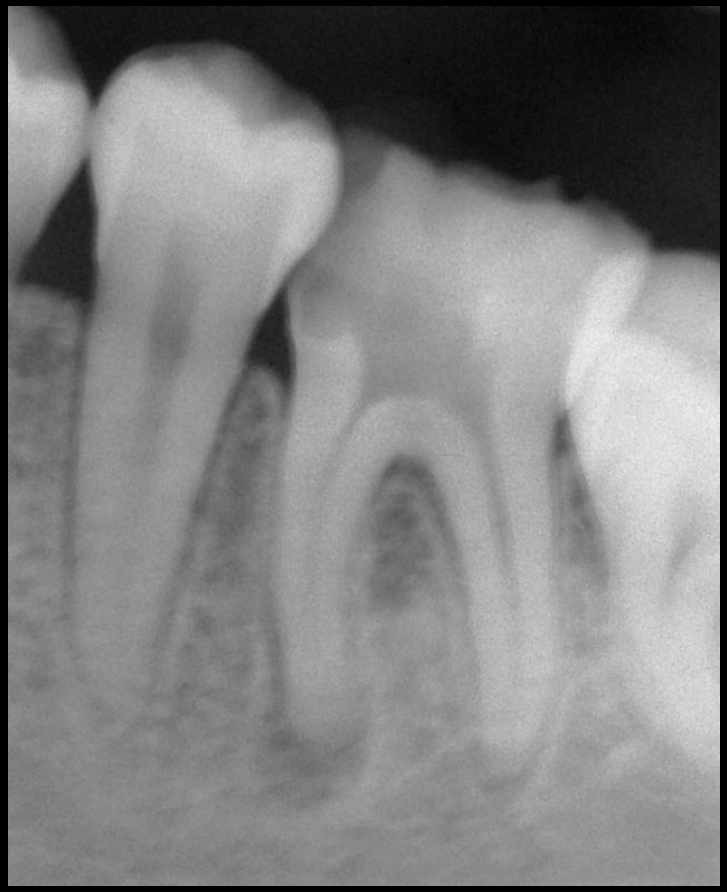
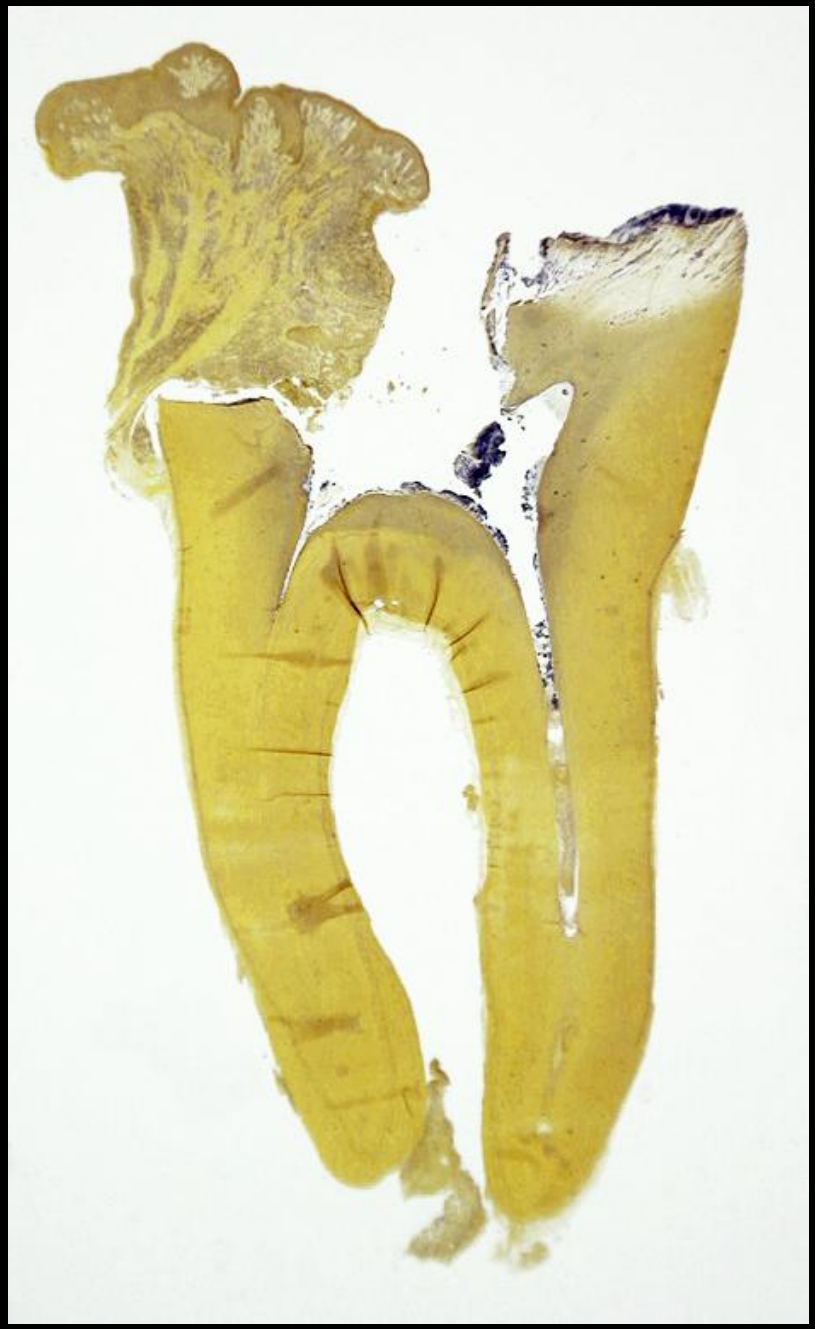
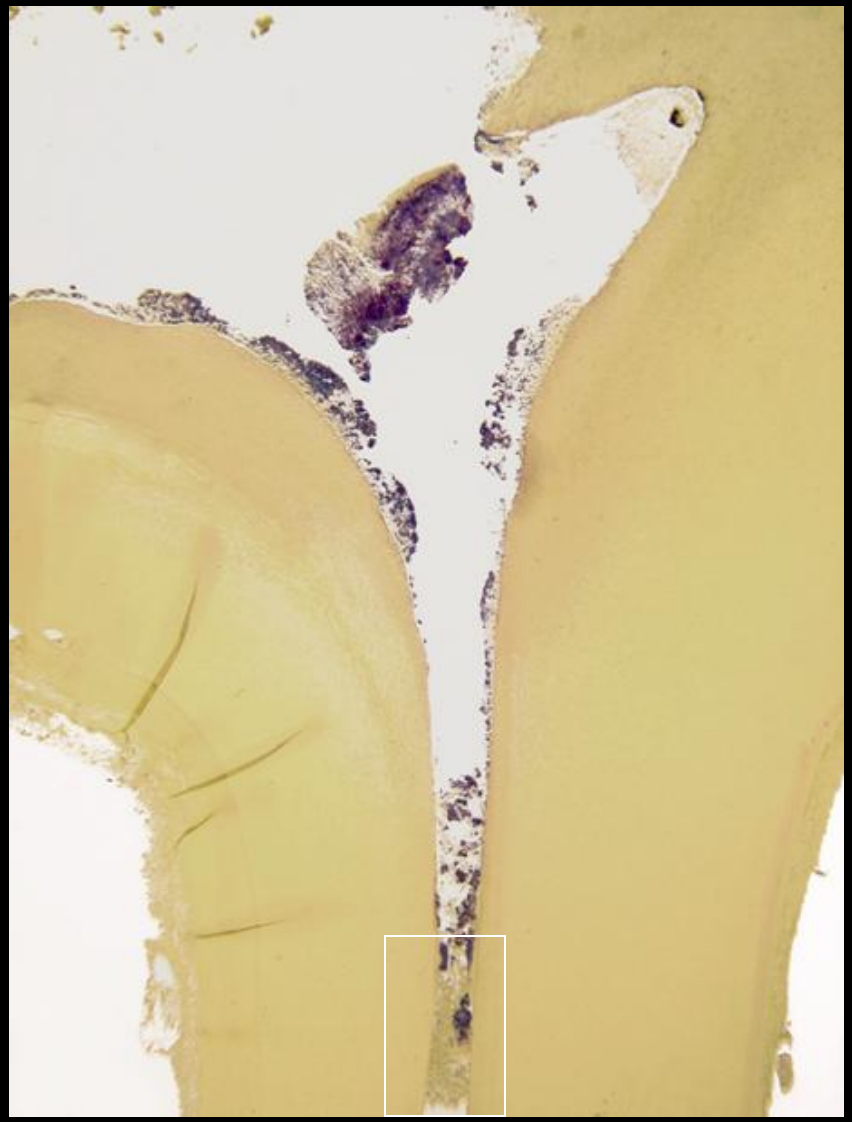
Periapical lesion formation







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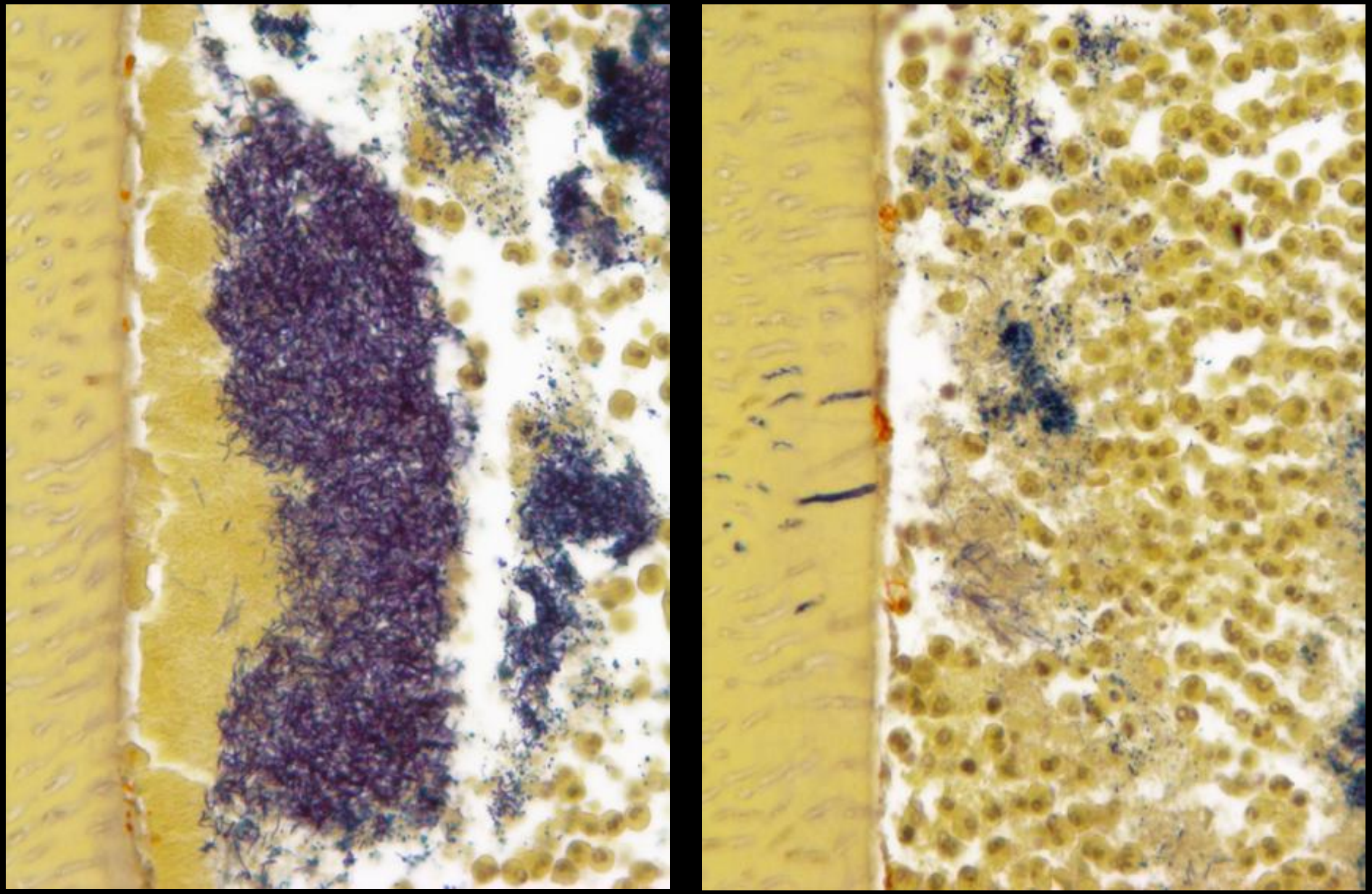

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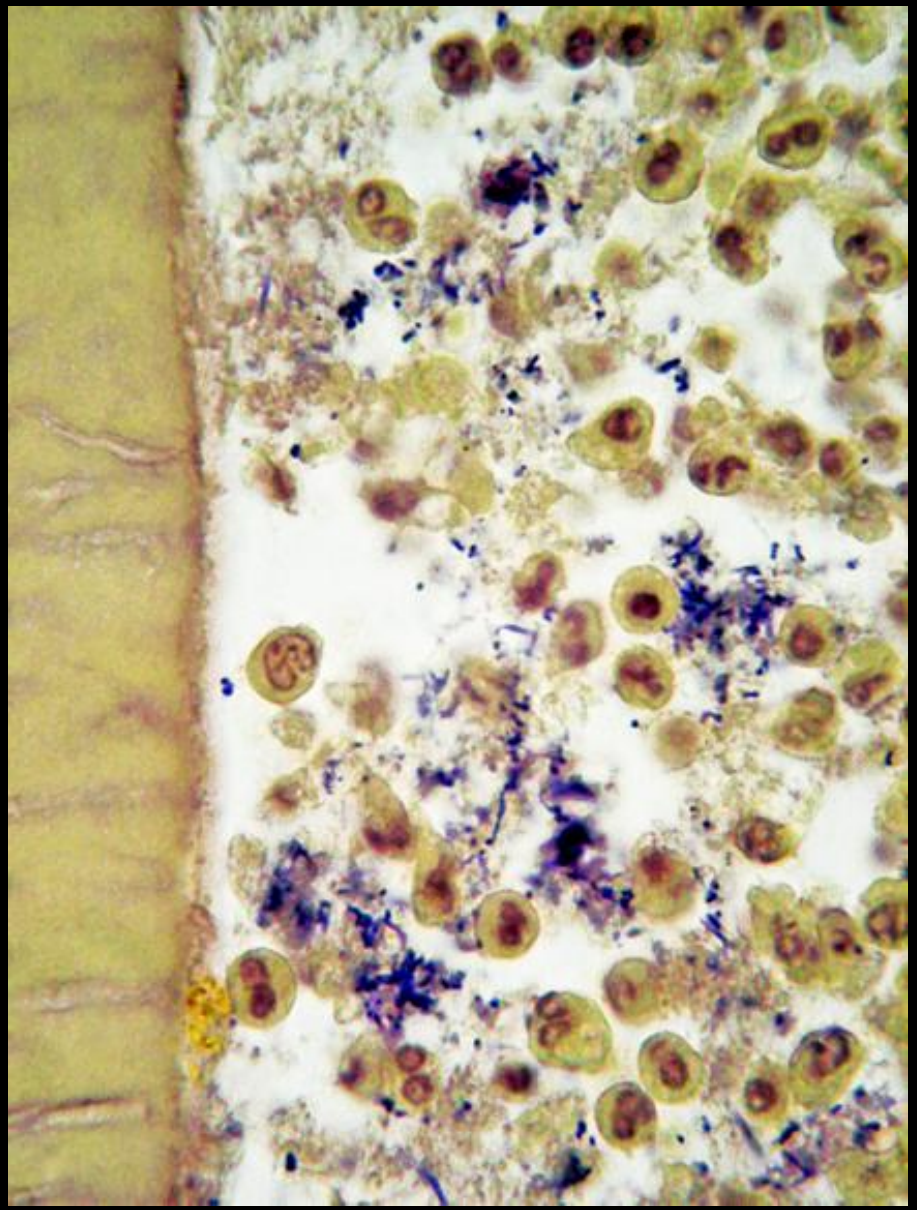

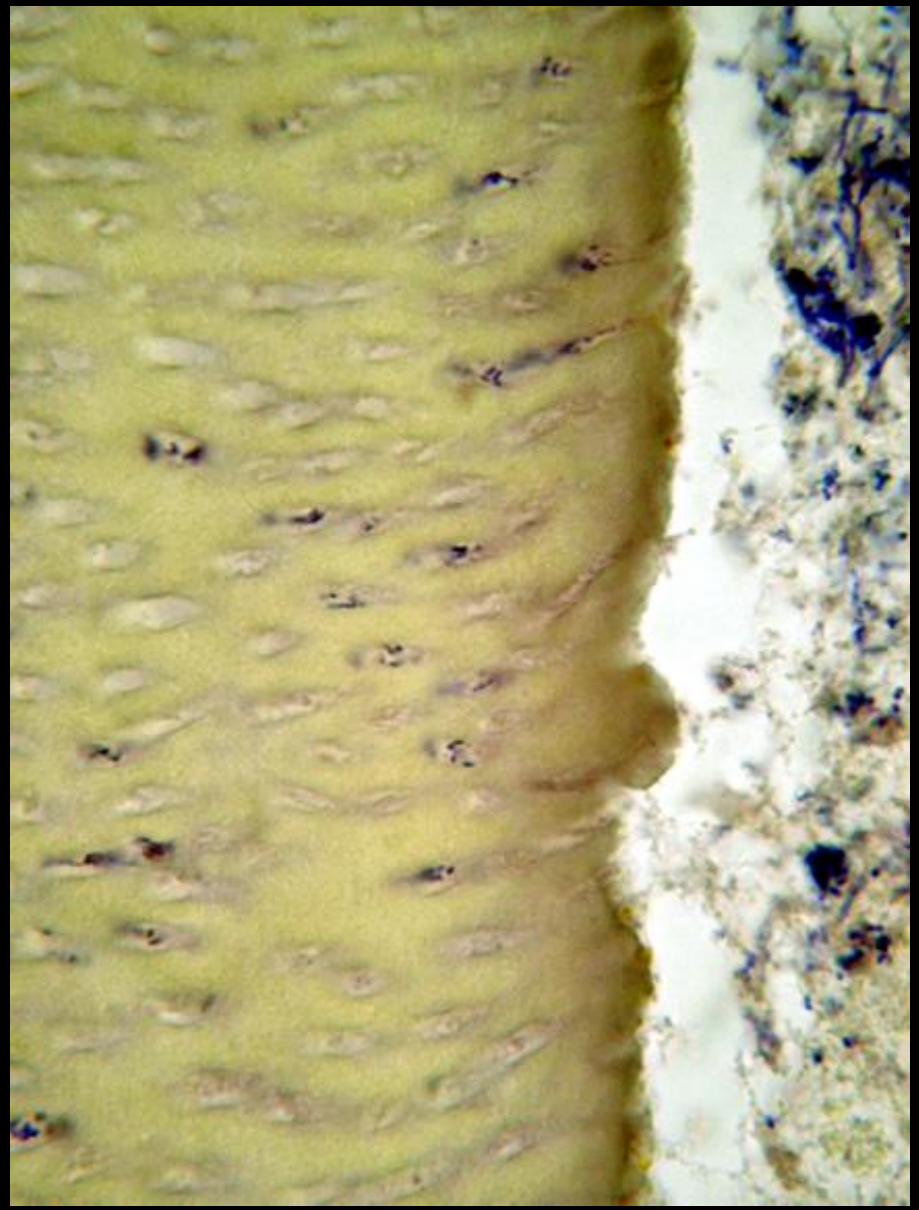

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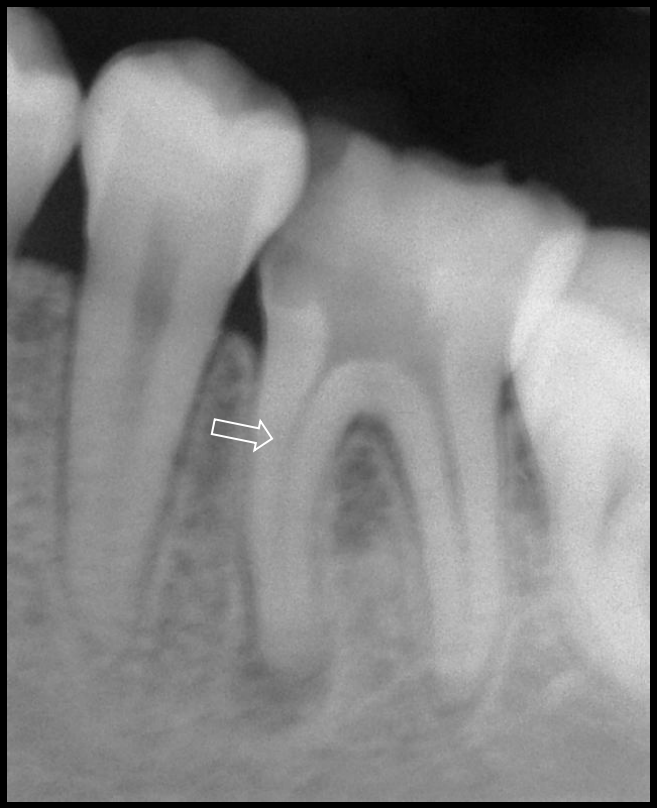

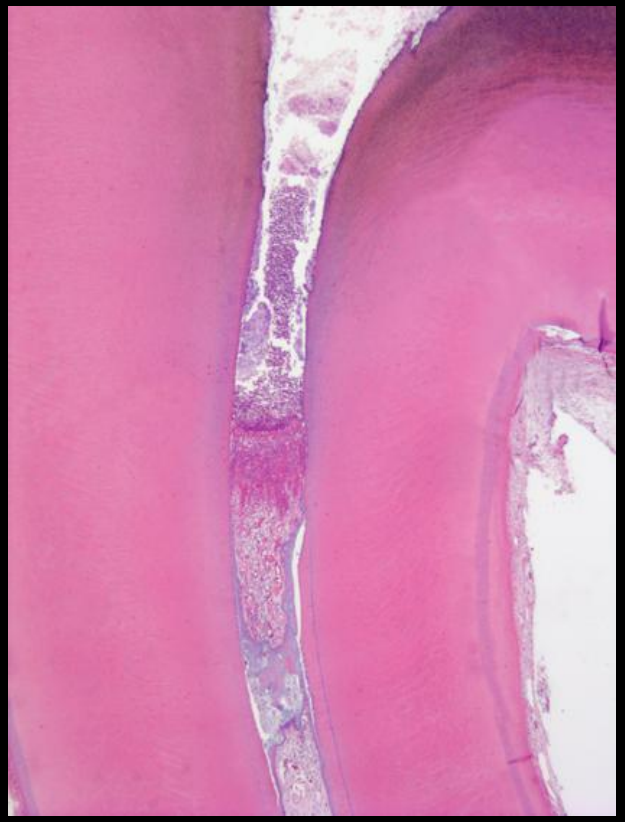

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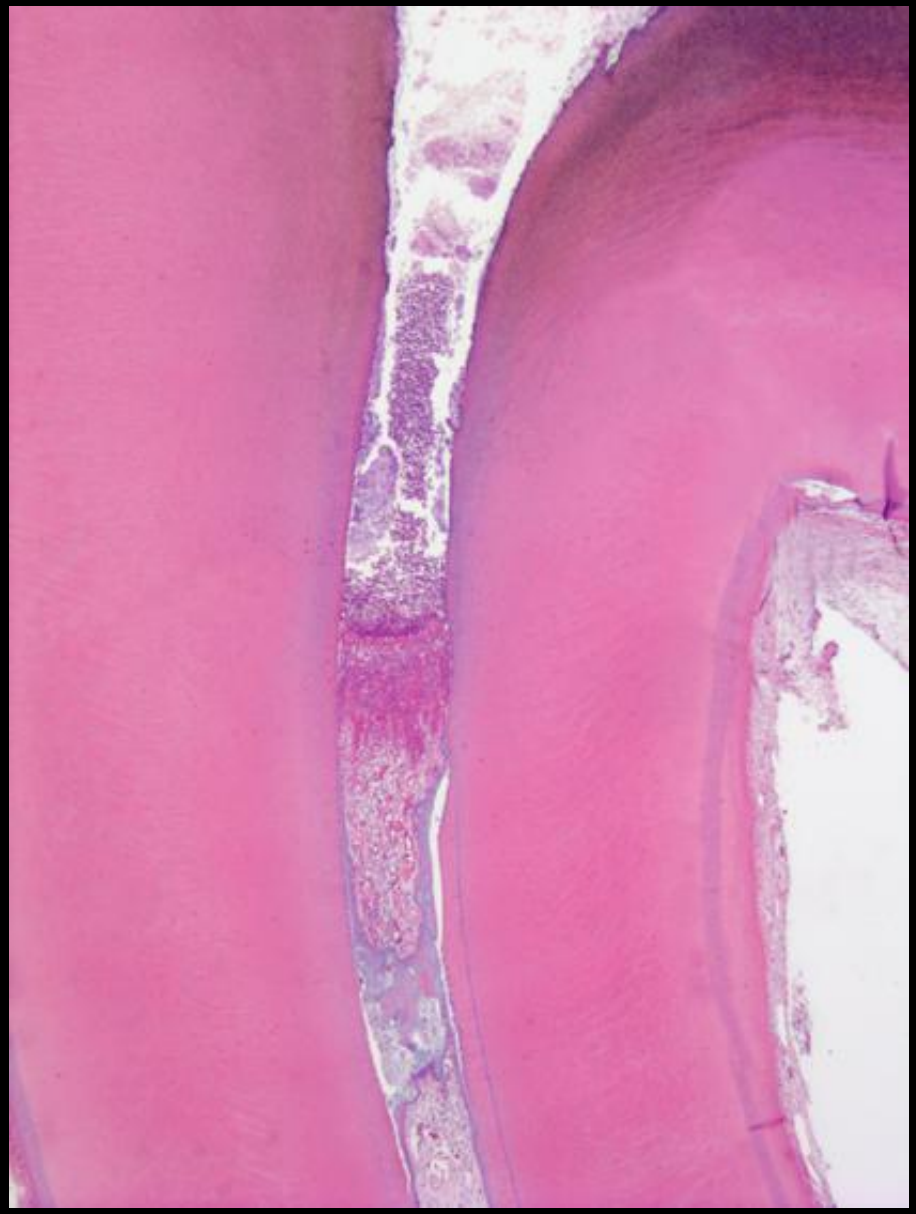

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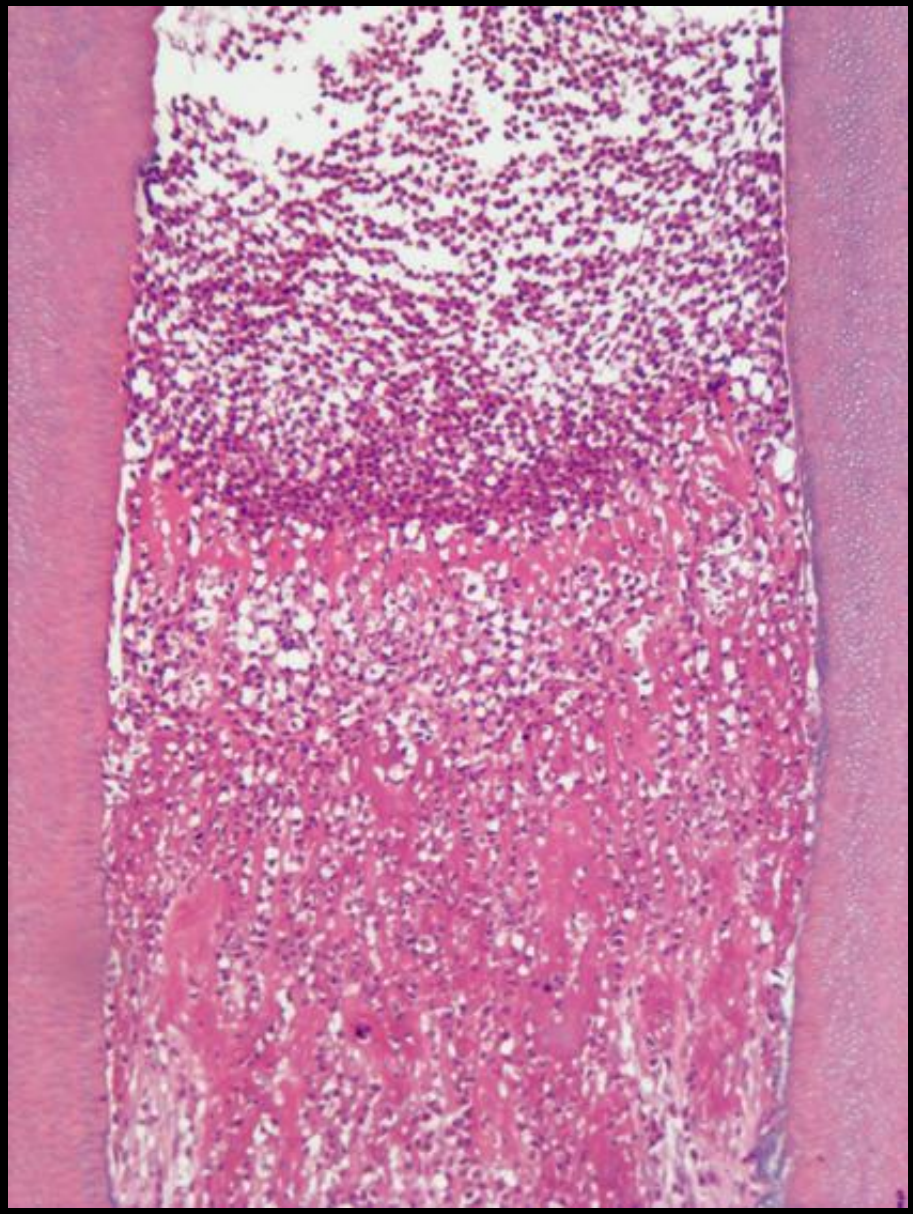

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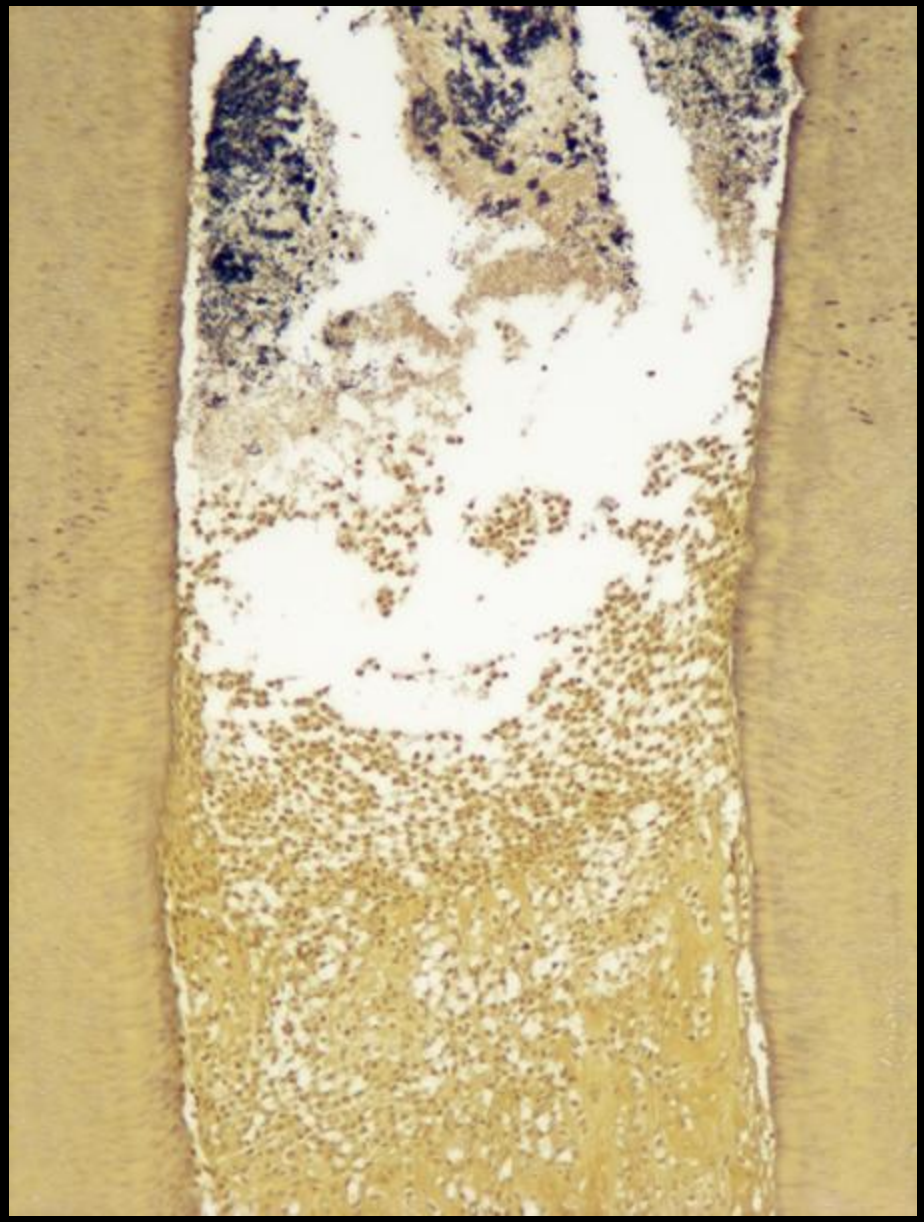

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Morphological aspects of the biofilm

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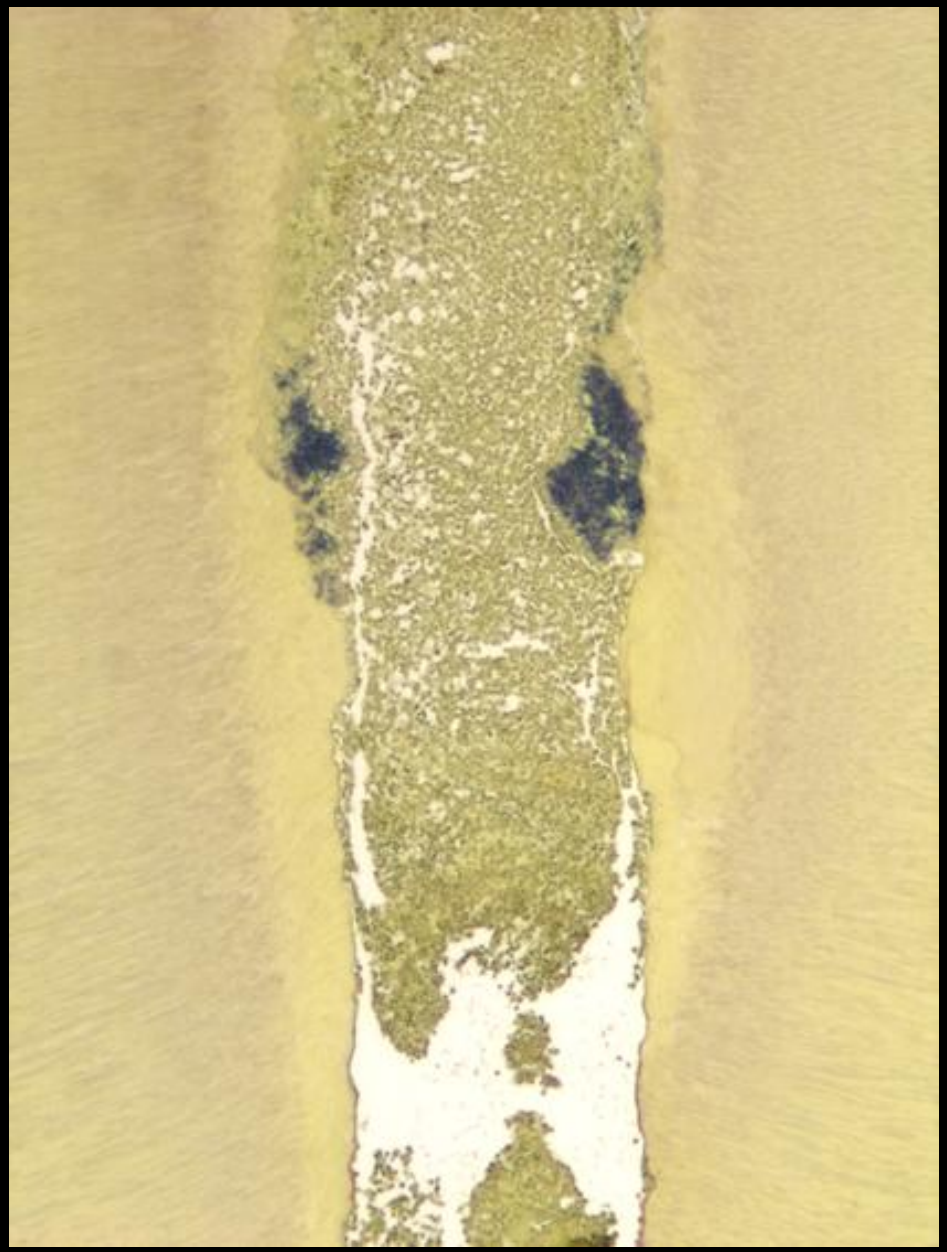

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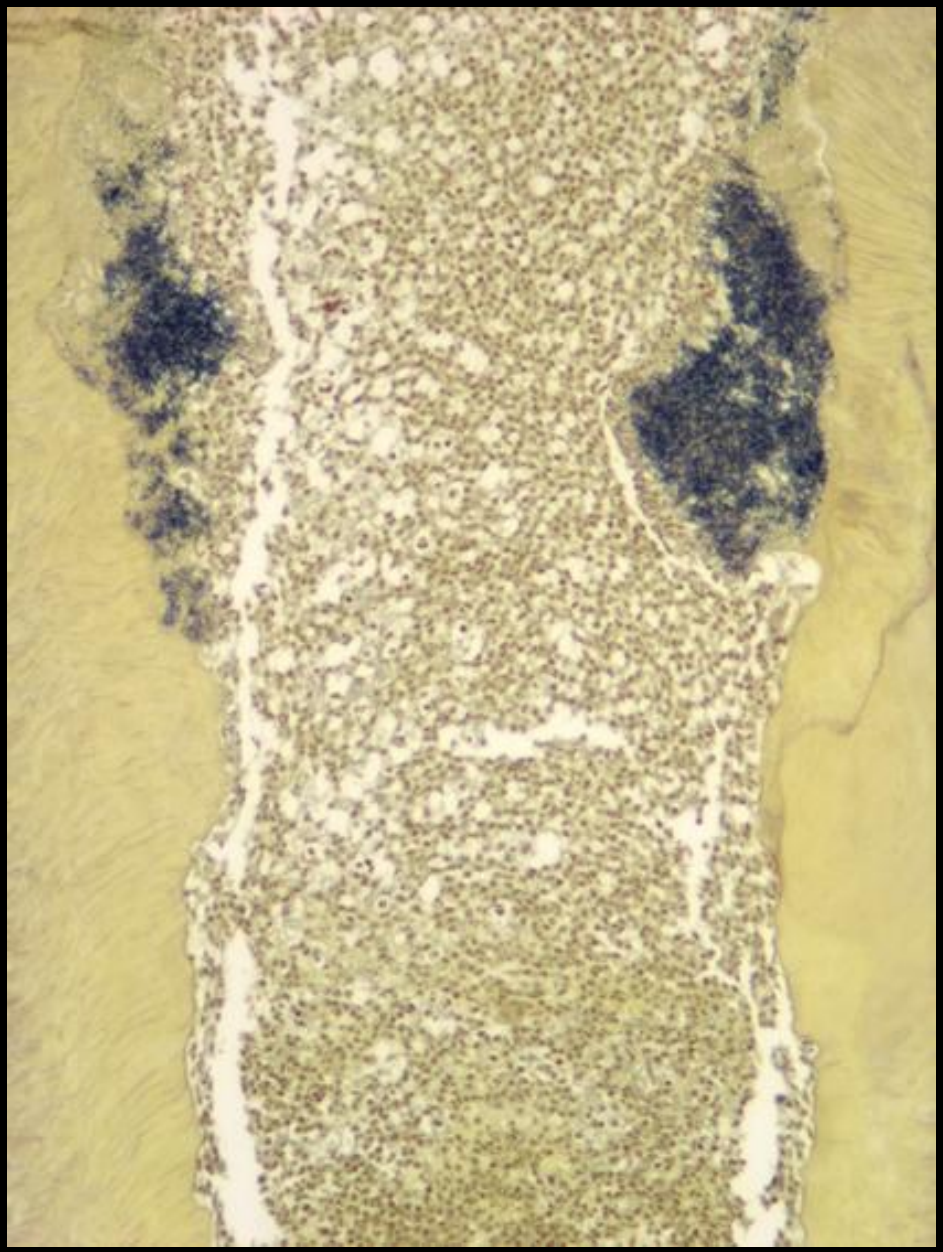

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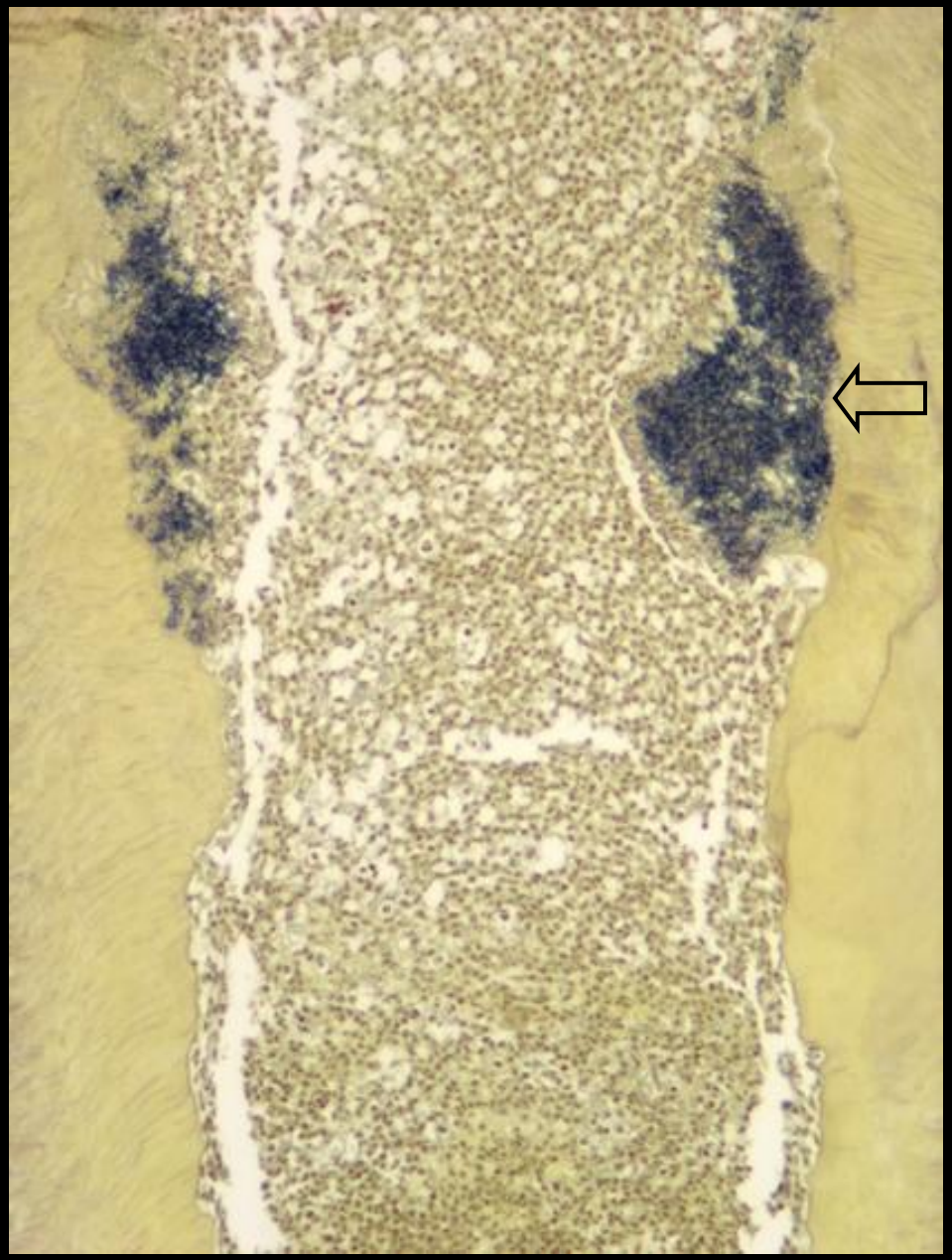

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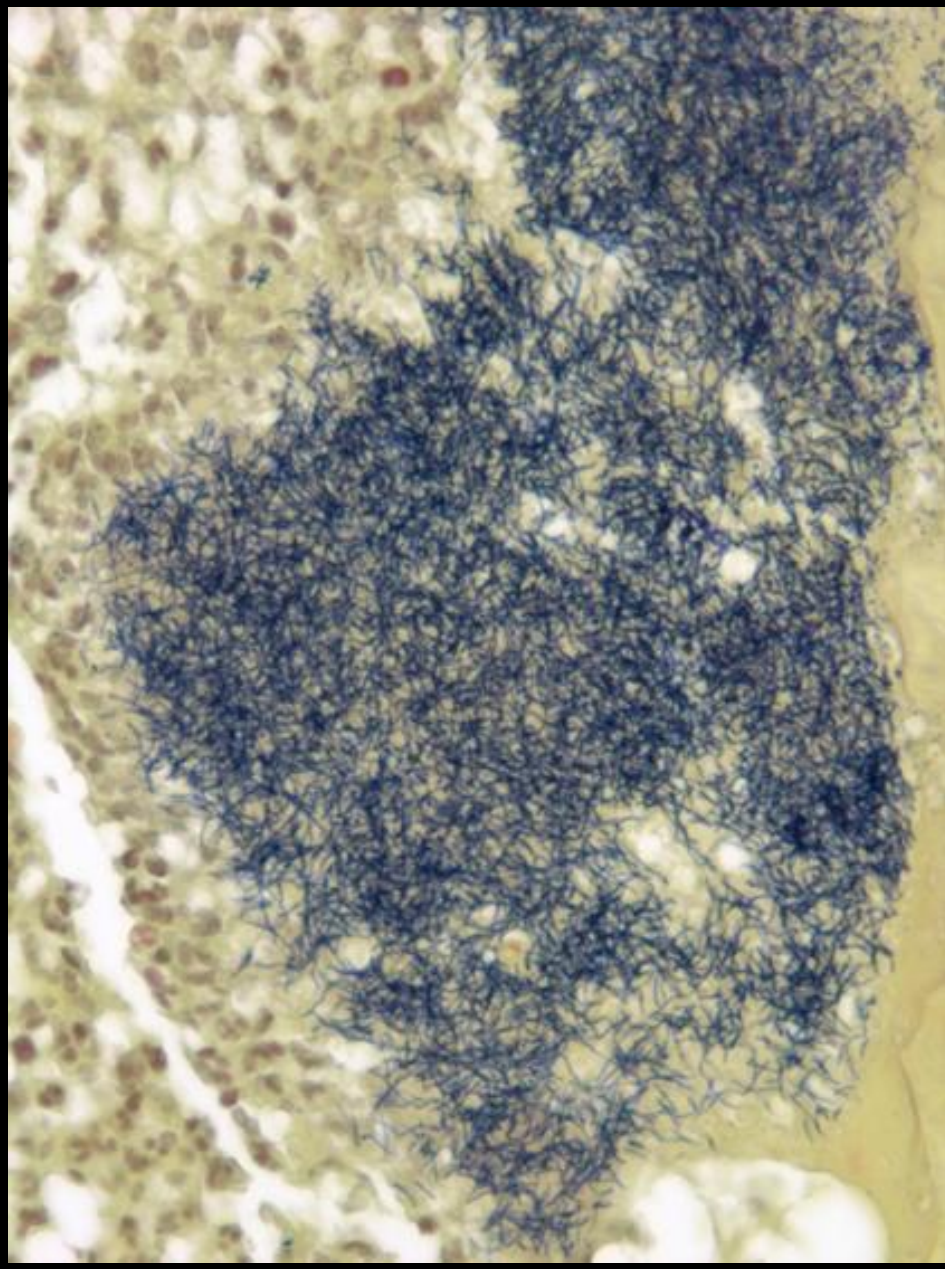

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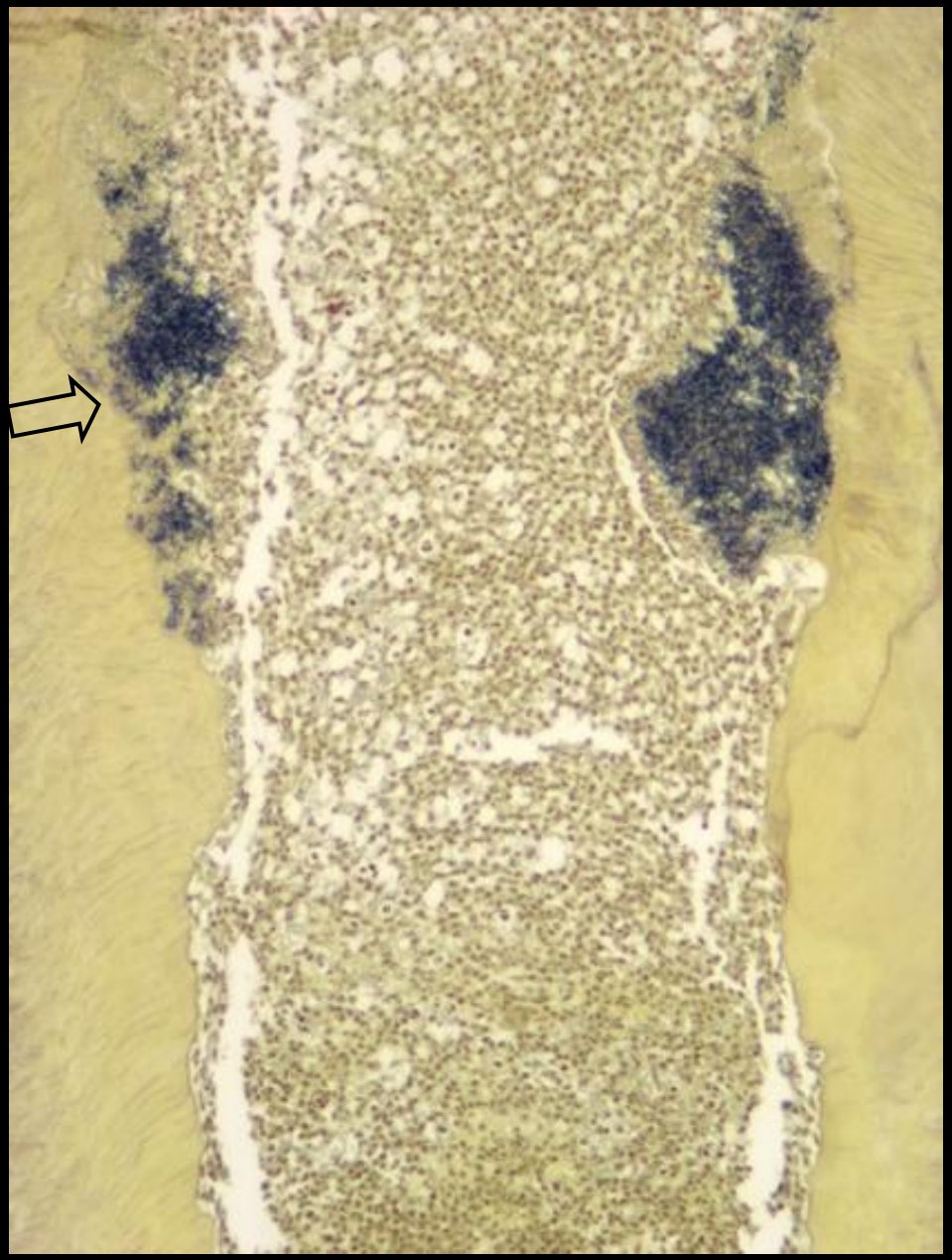

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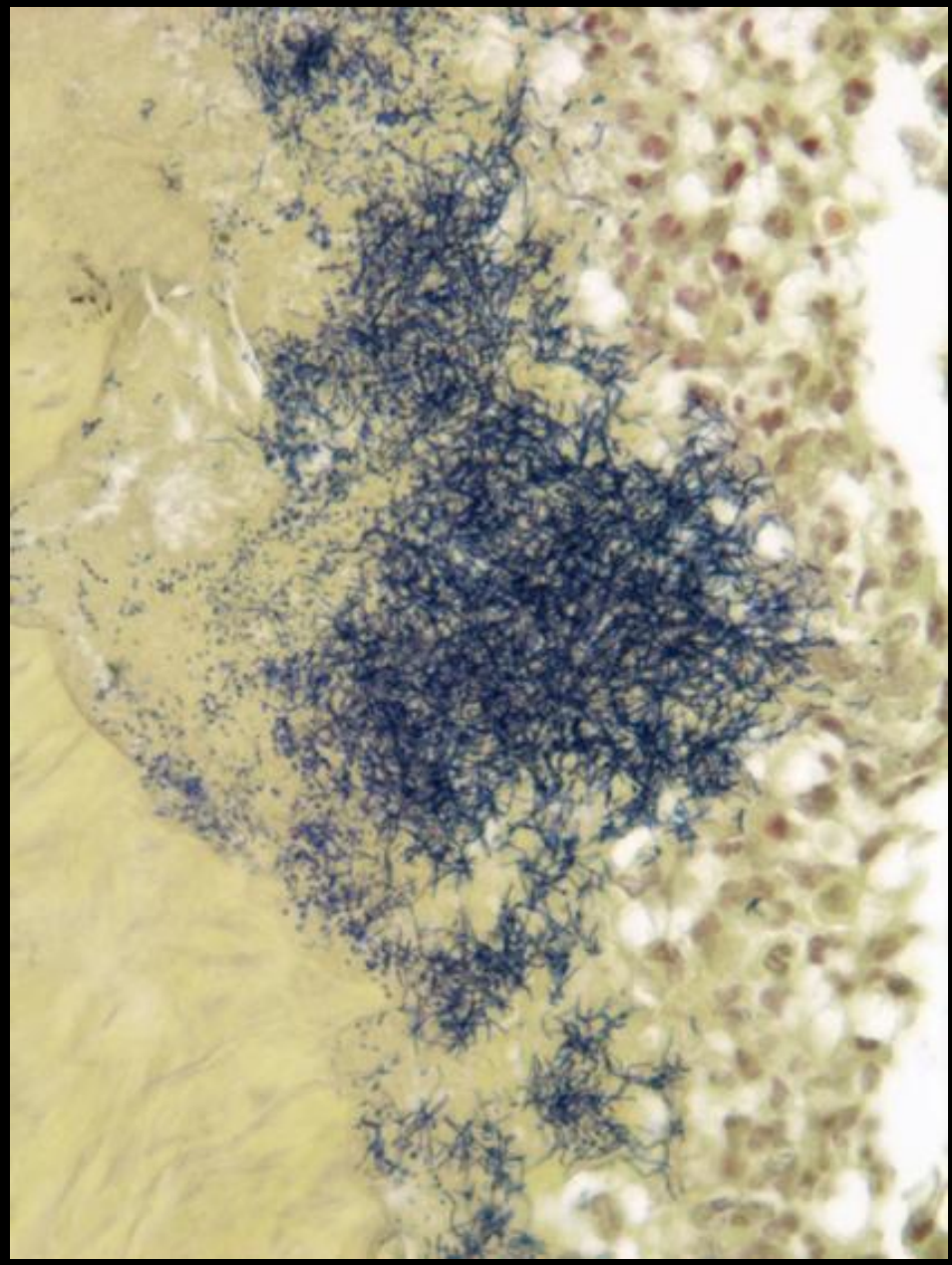

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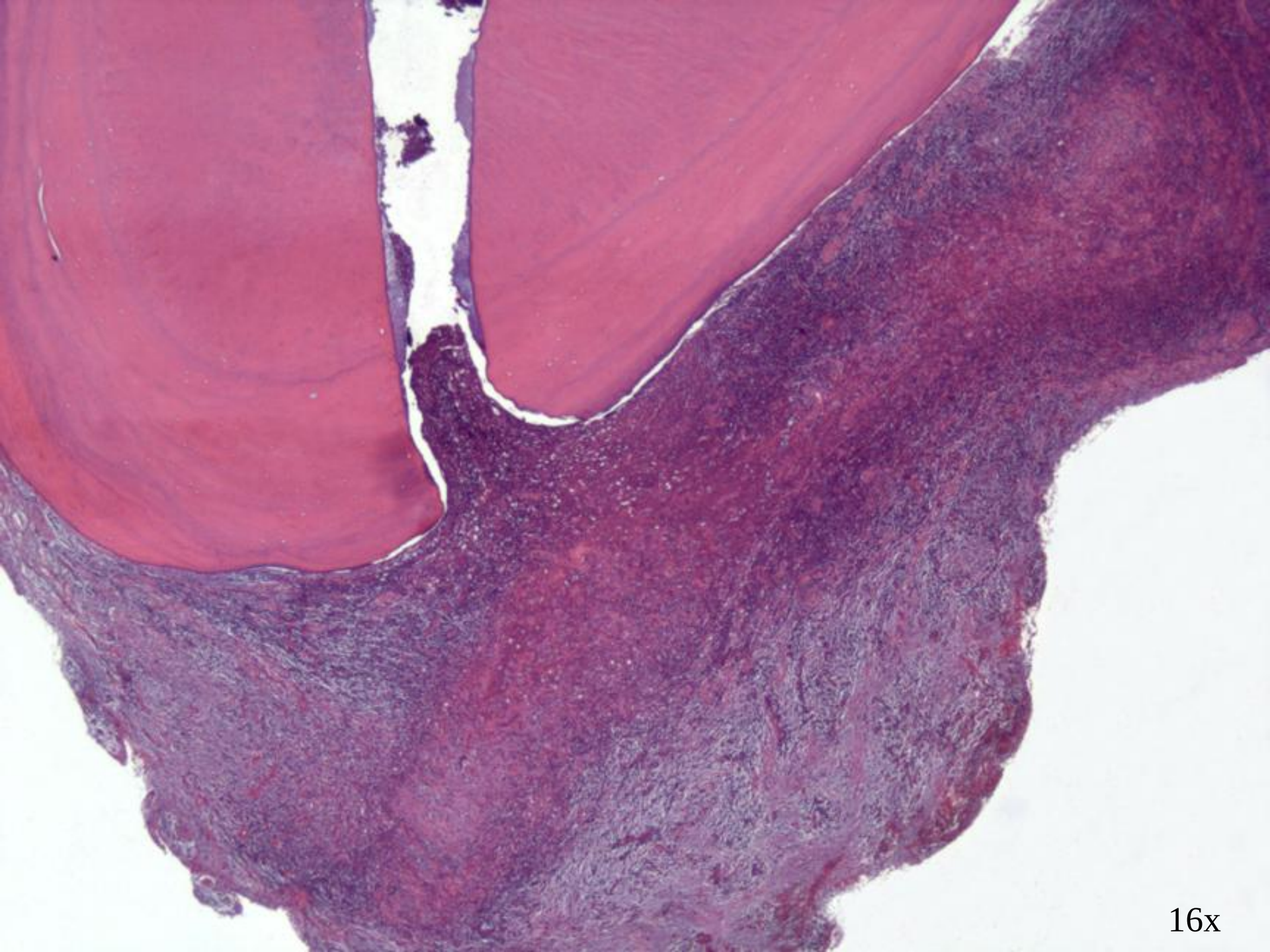
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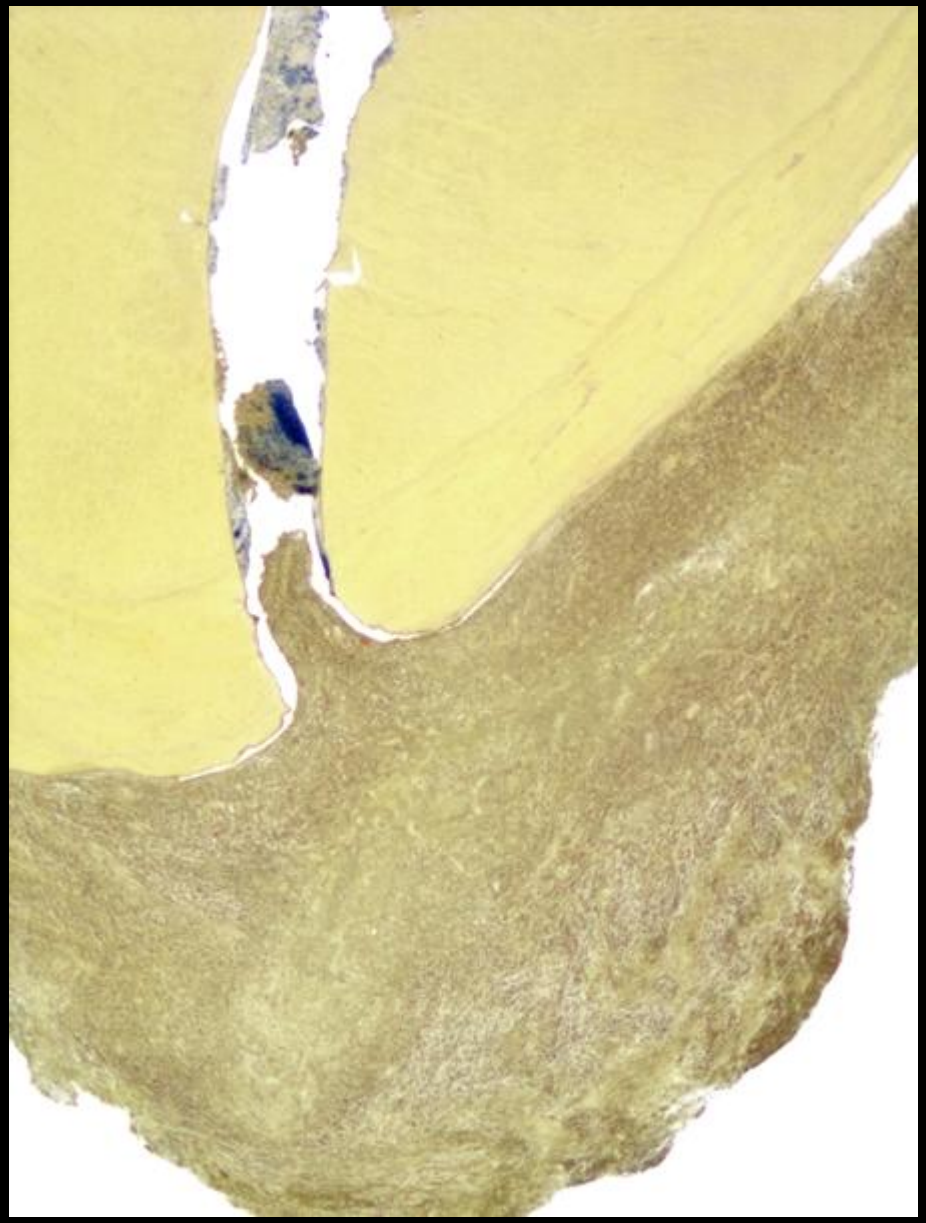

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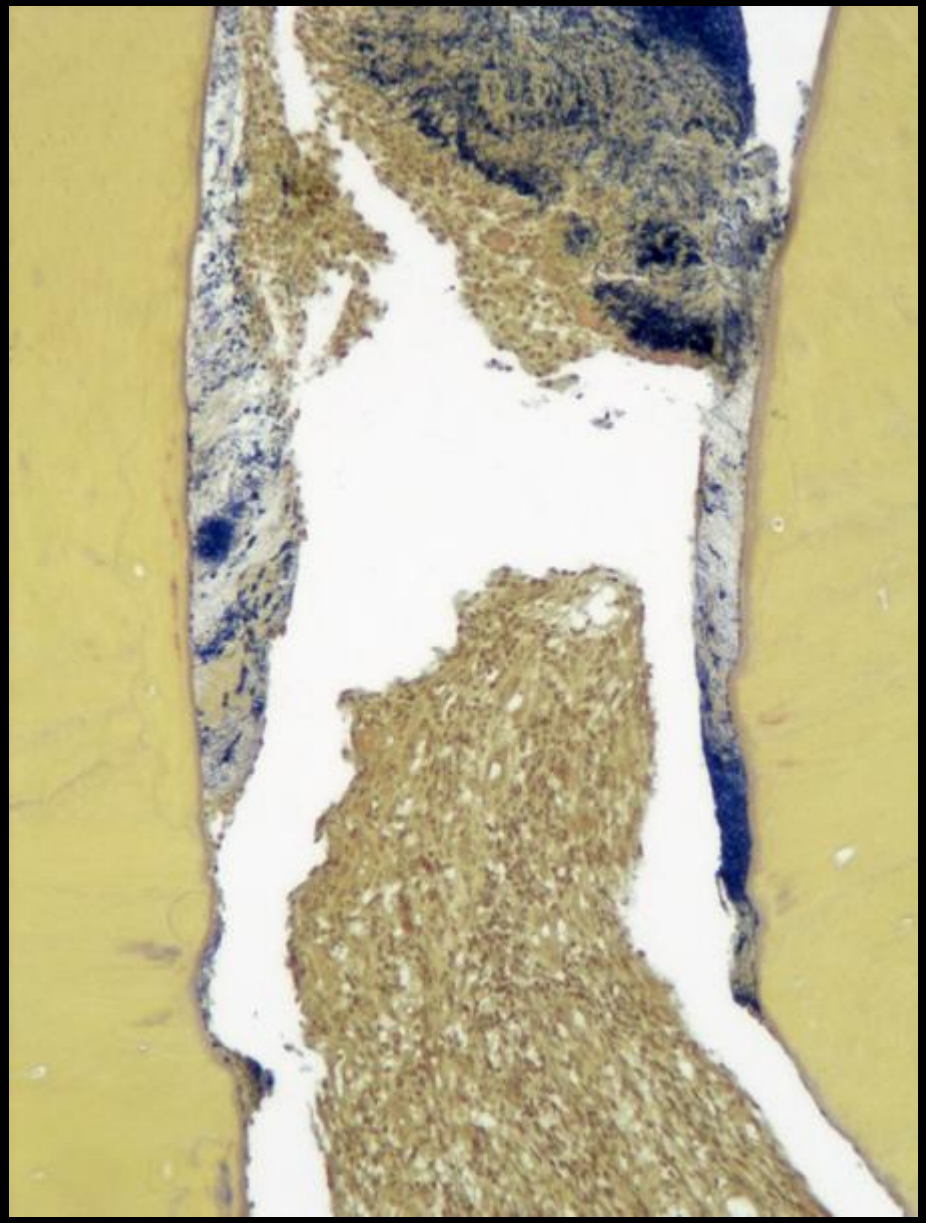

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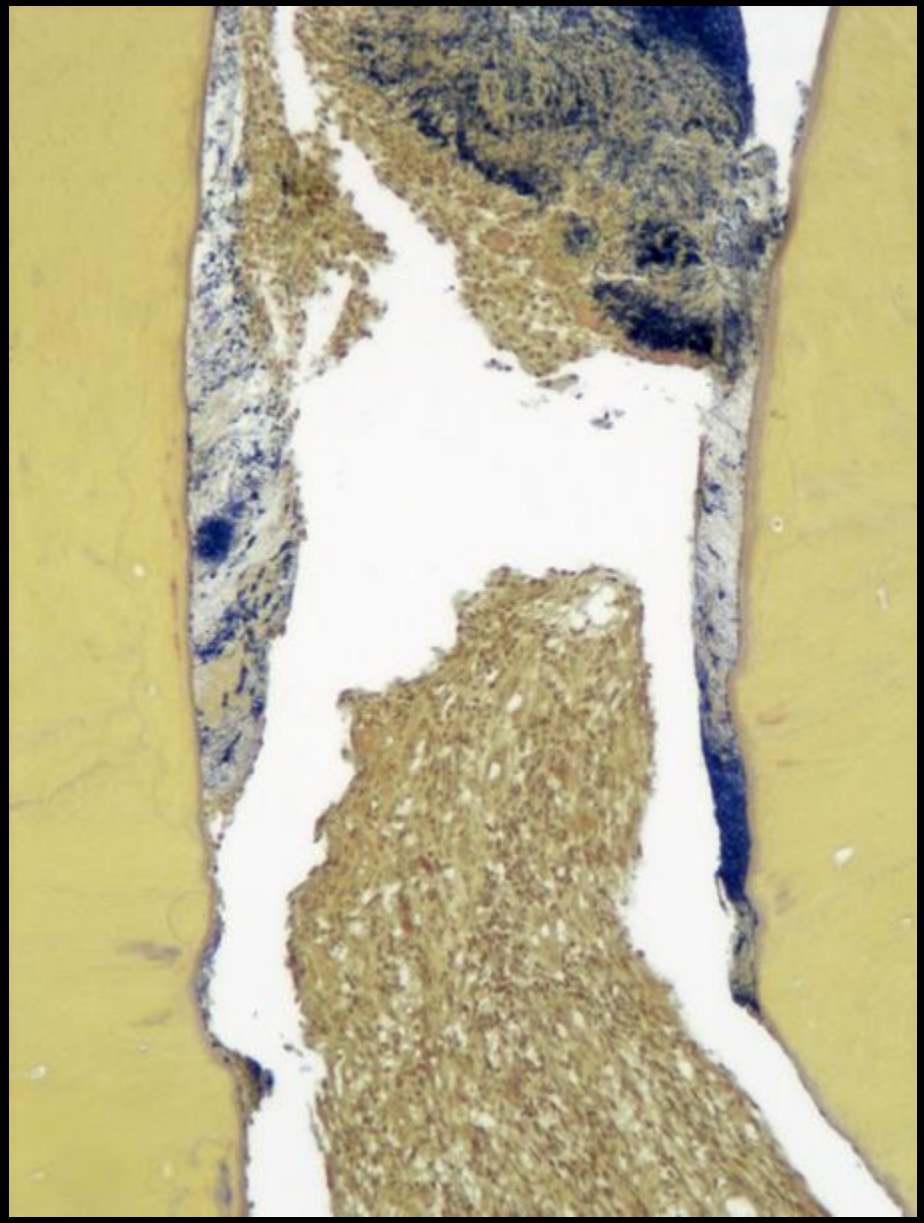

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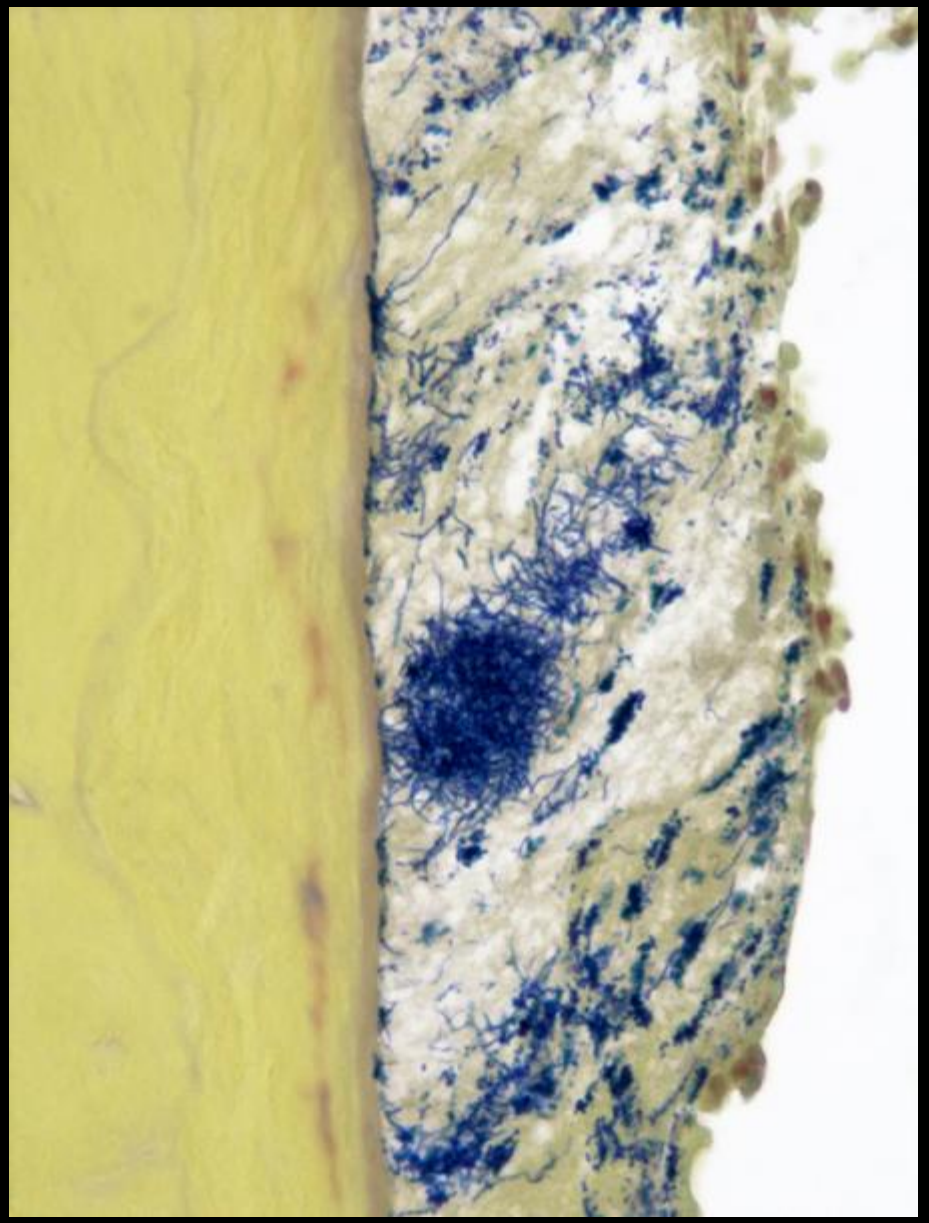

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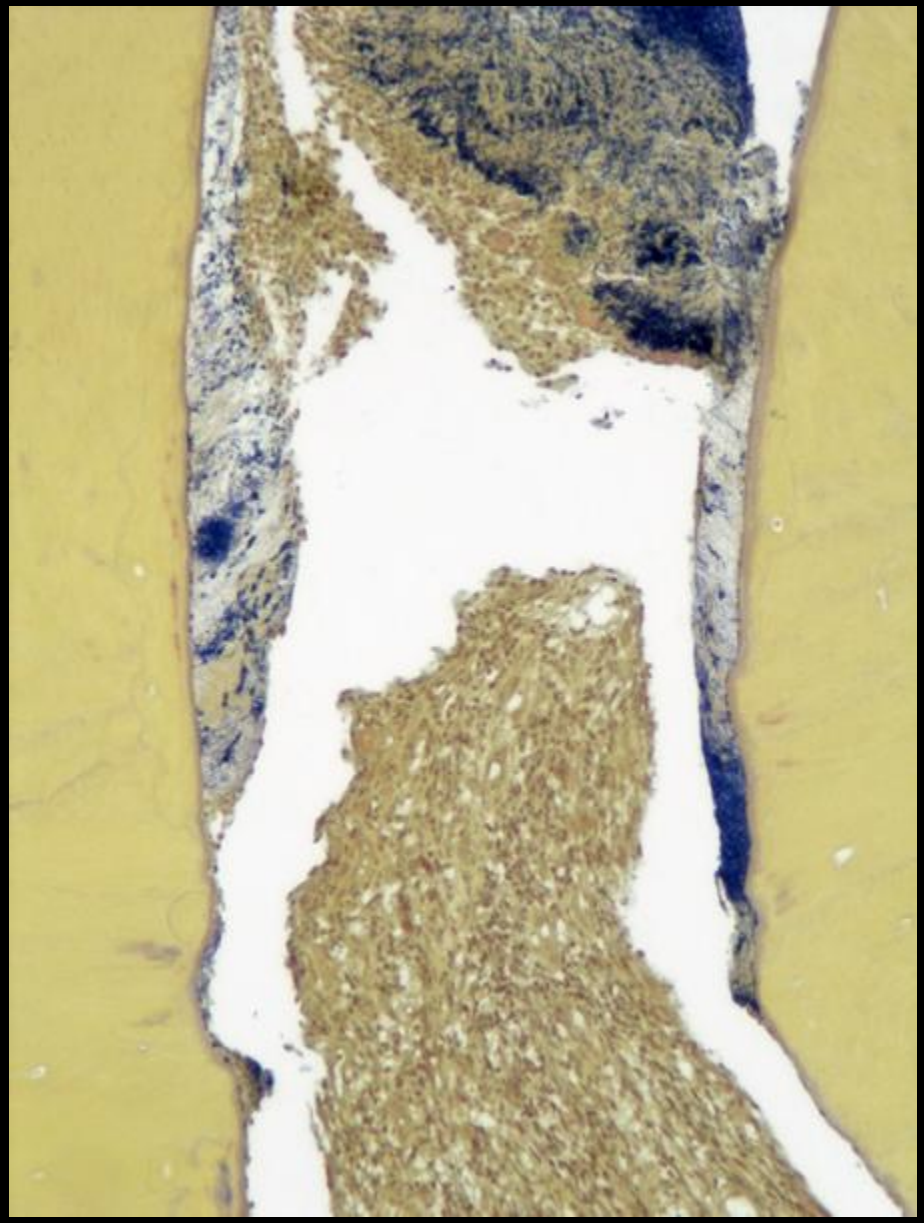

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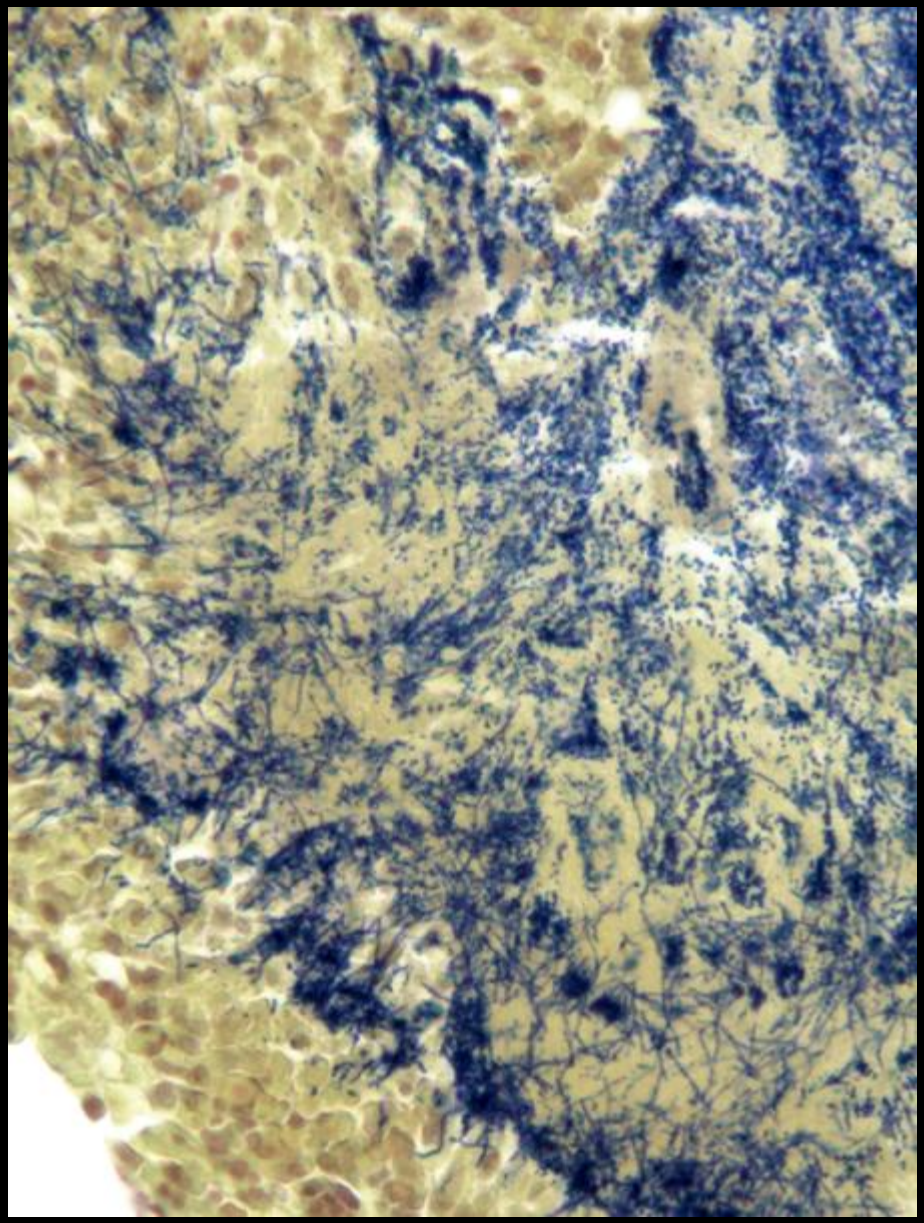

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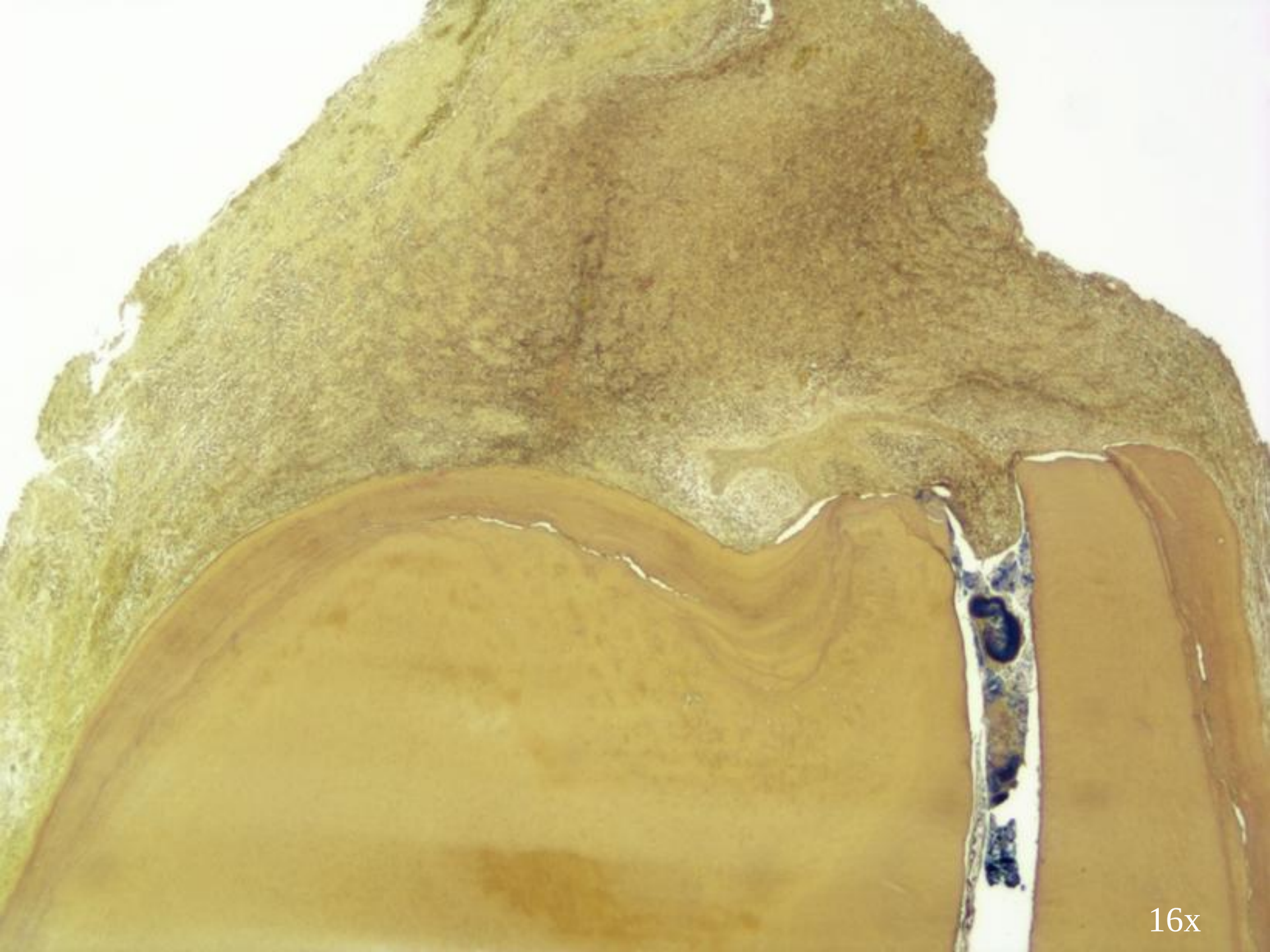
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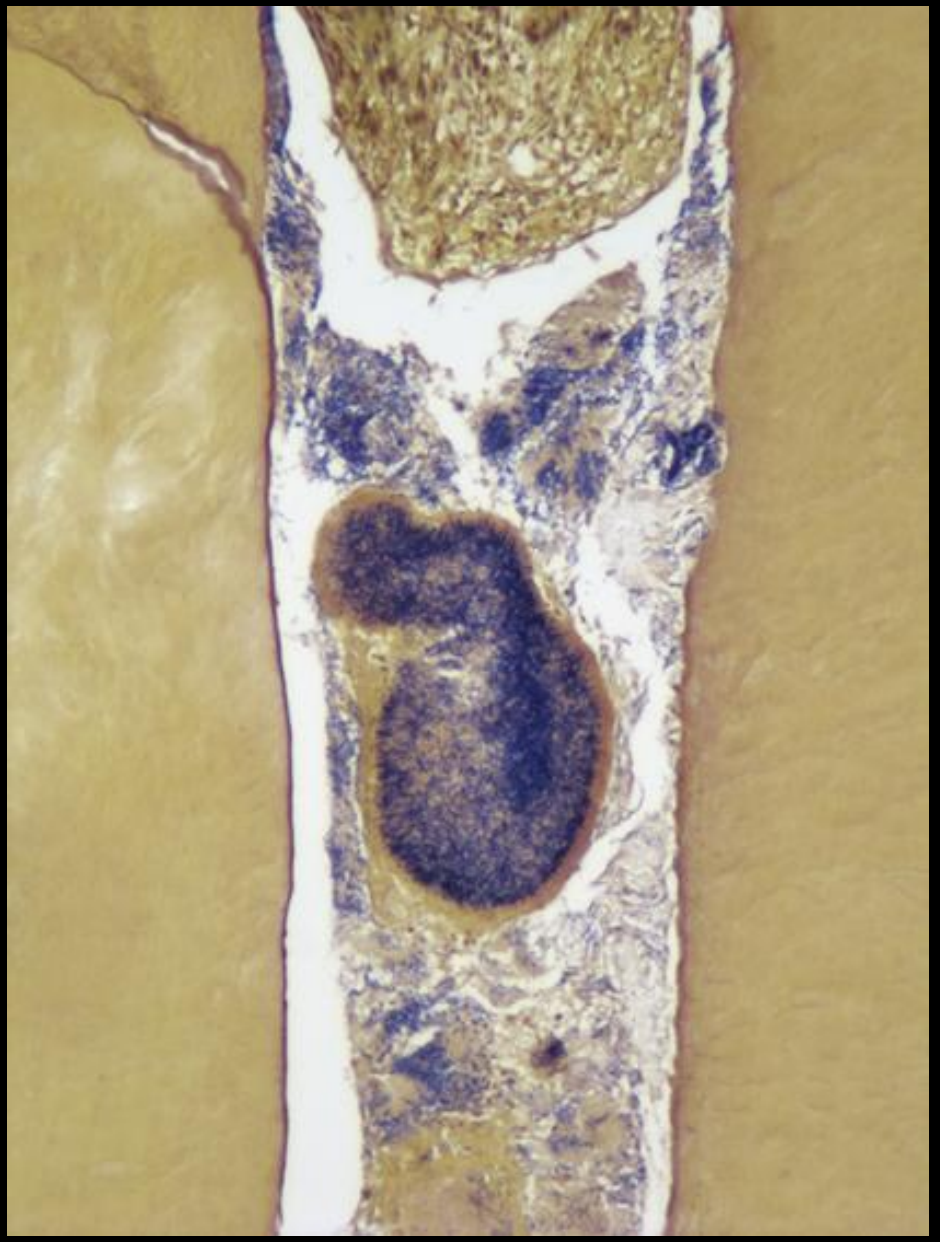

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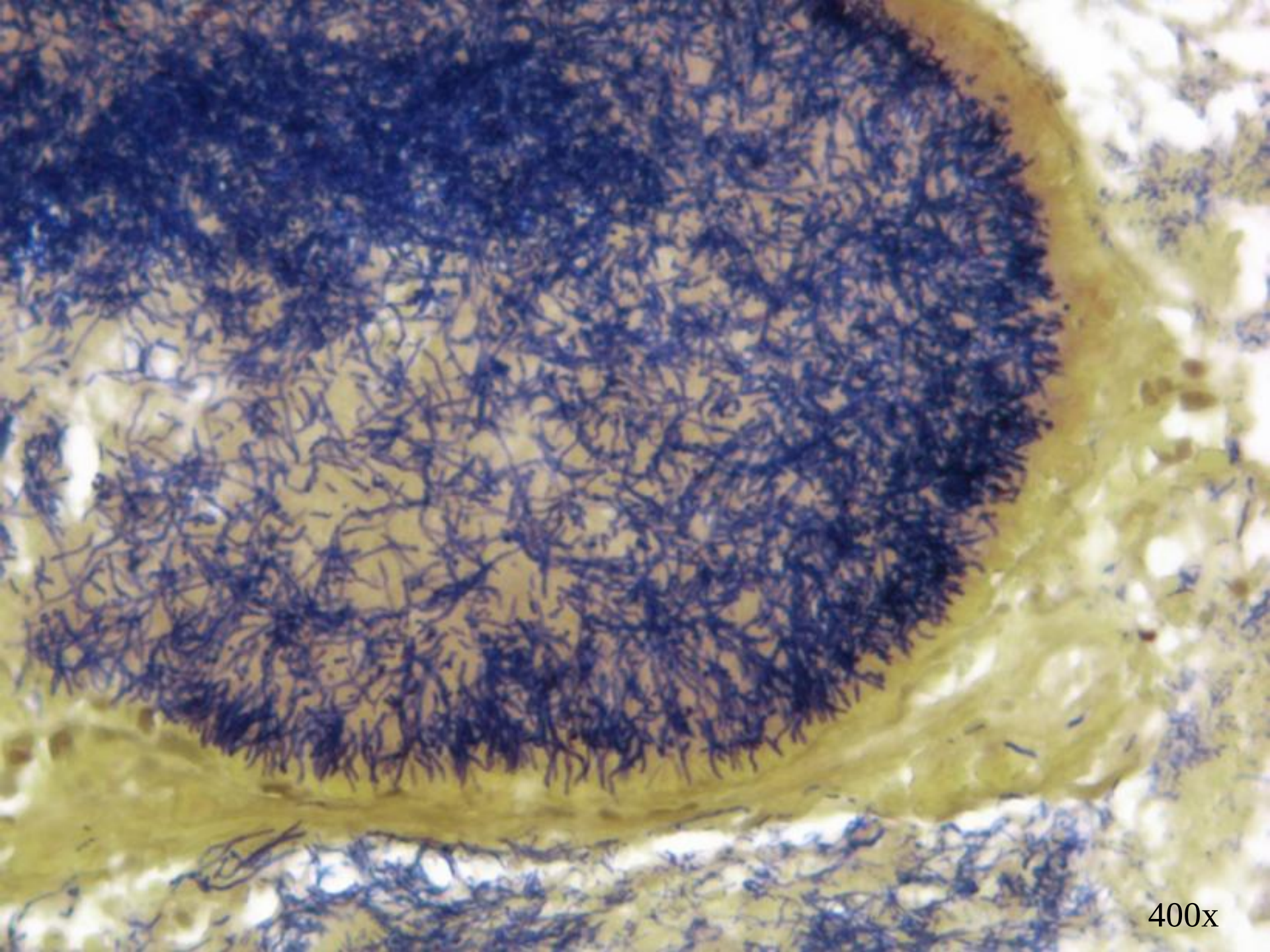
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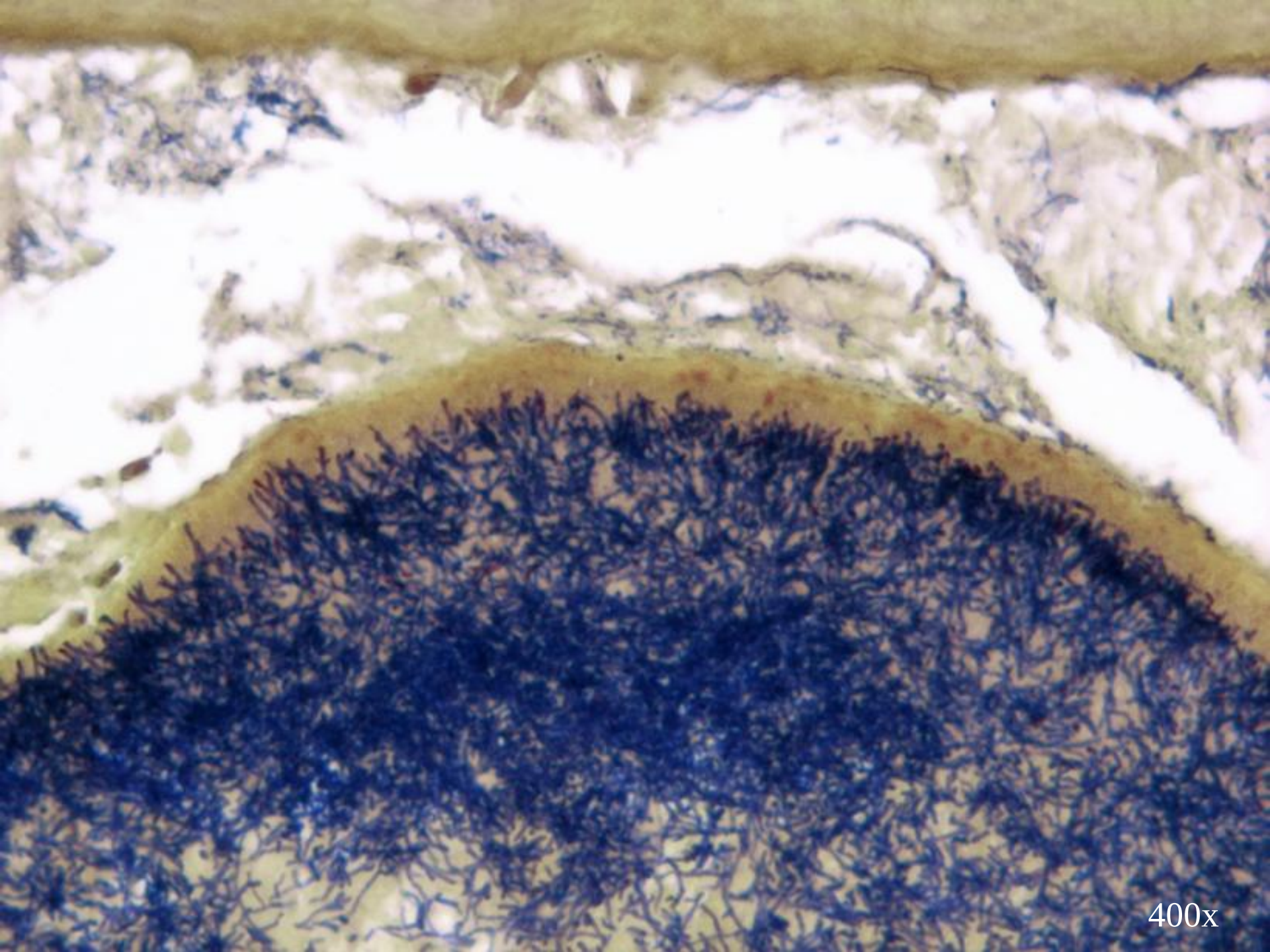
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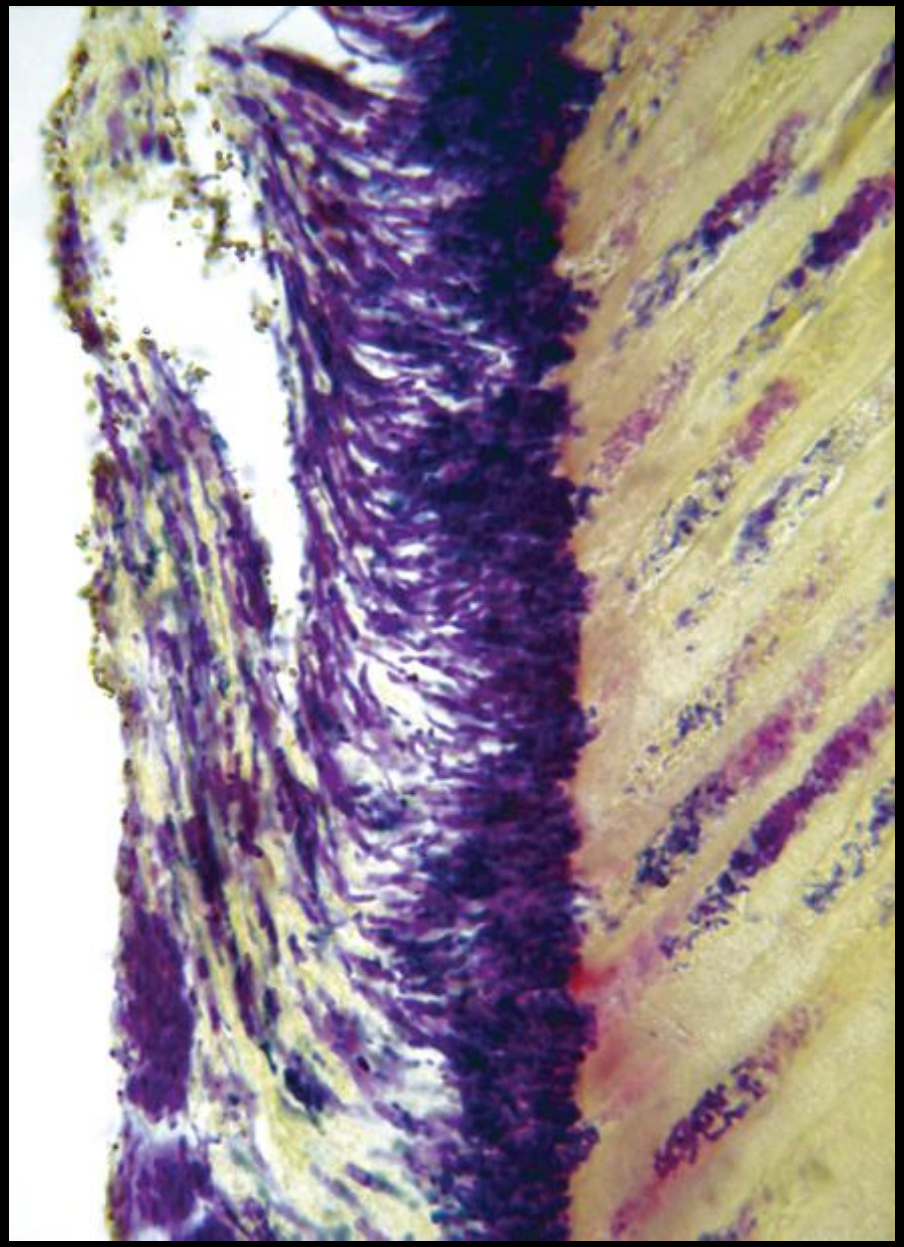

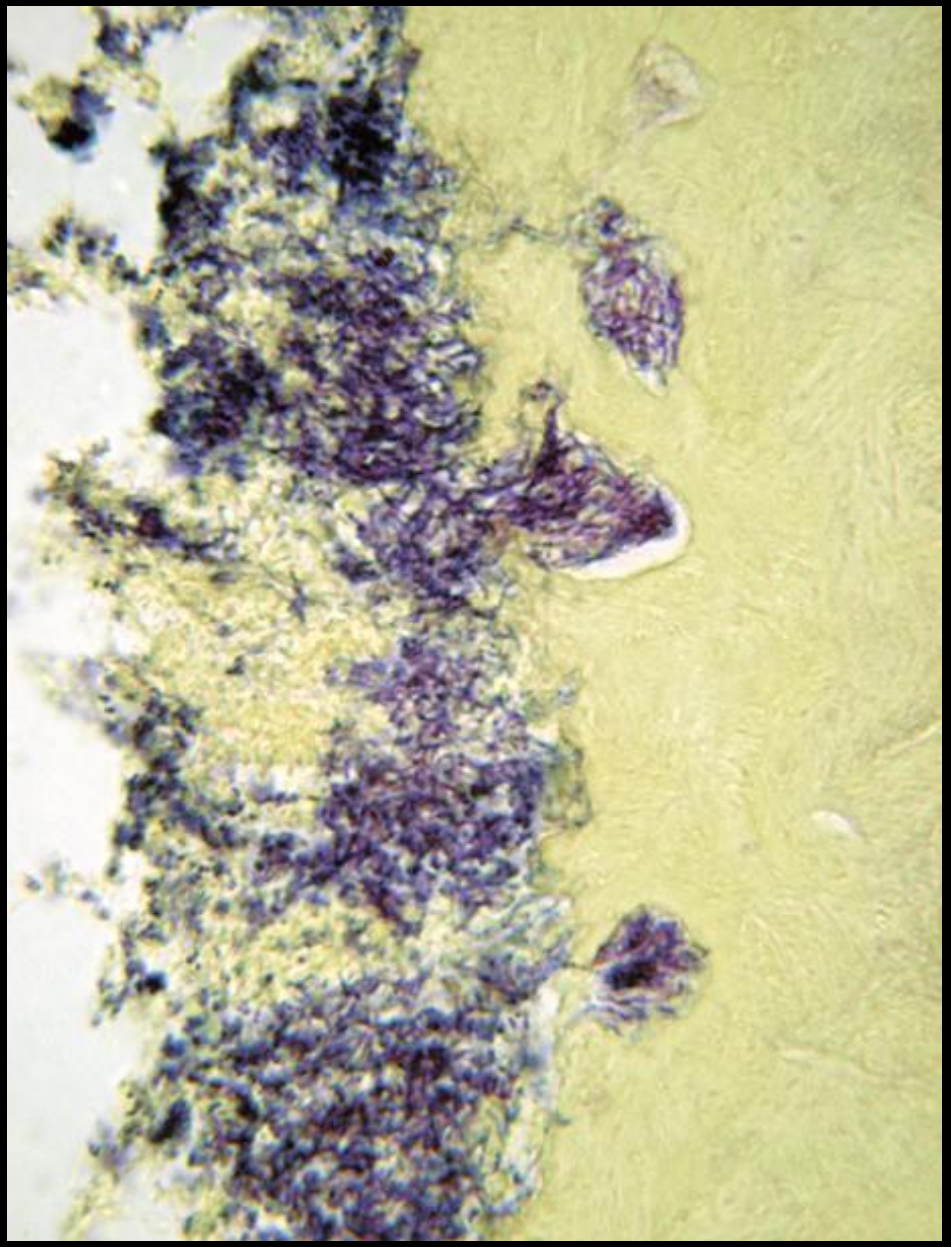

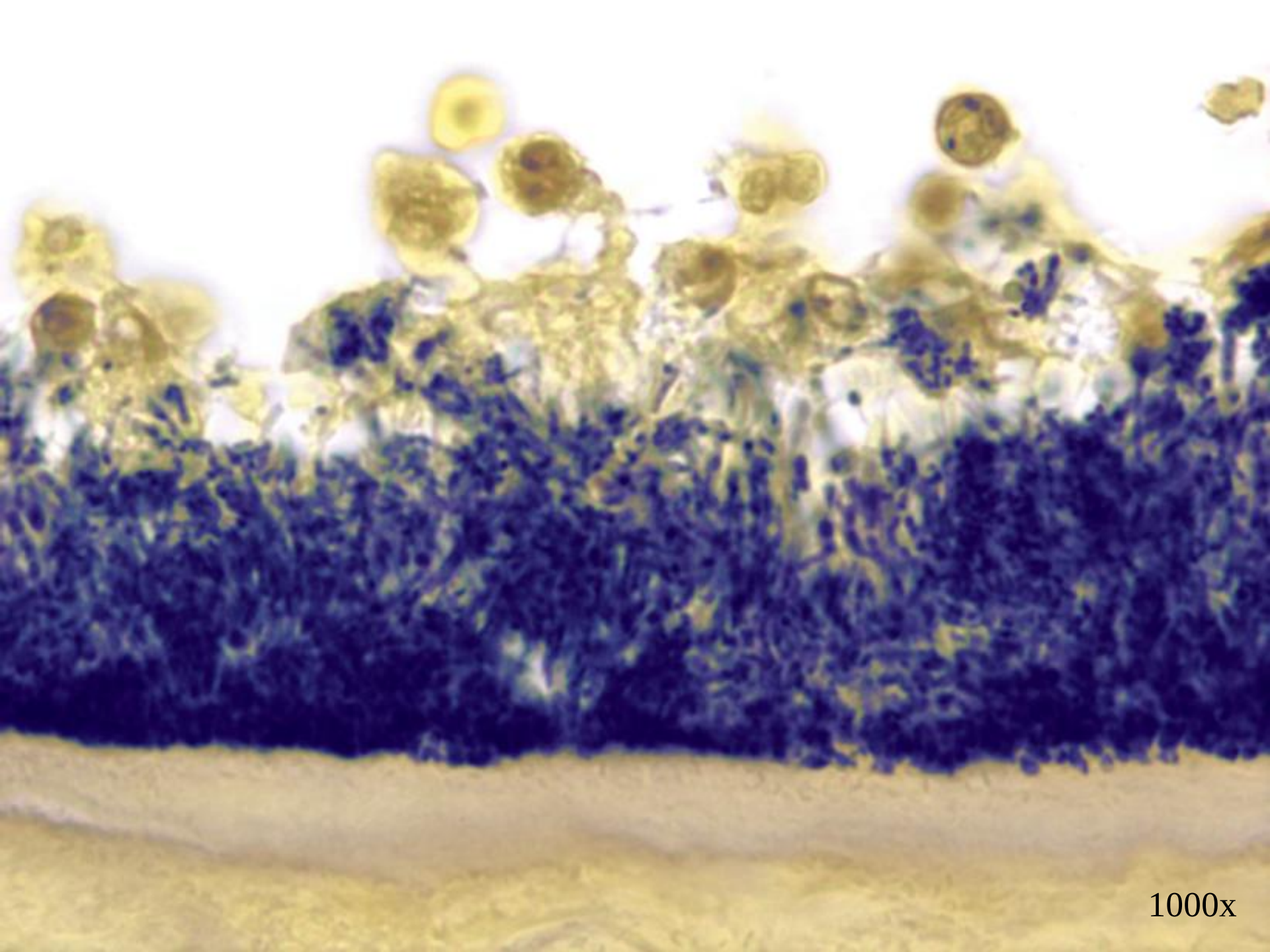
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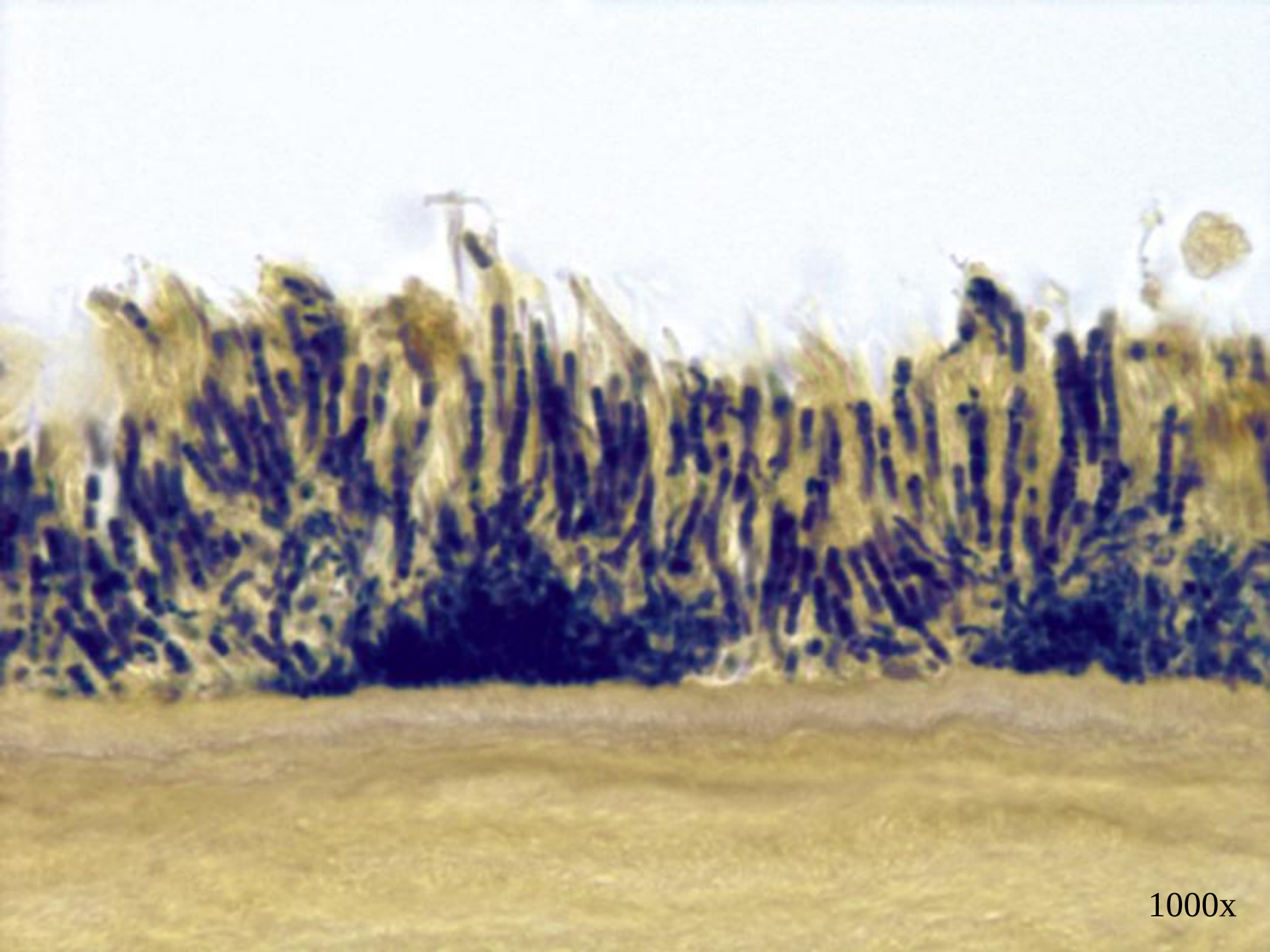
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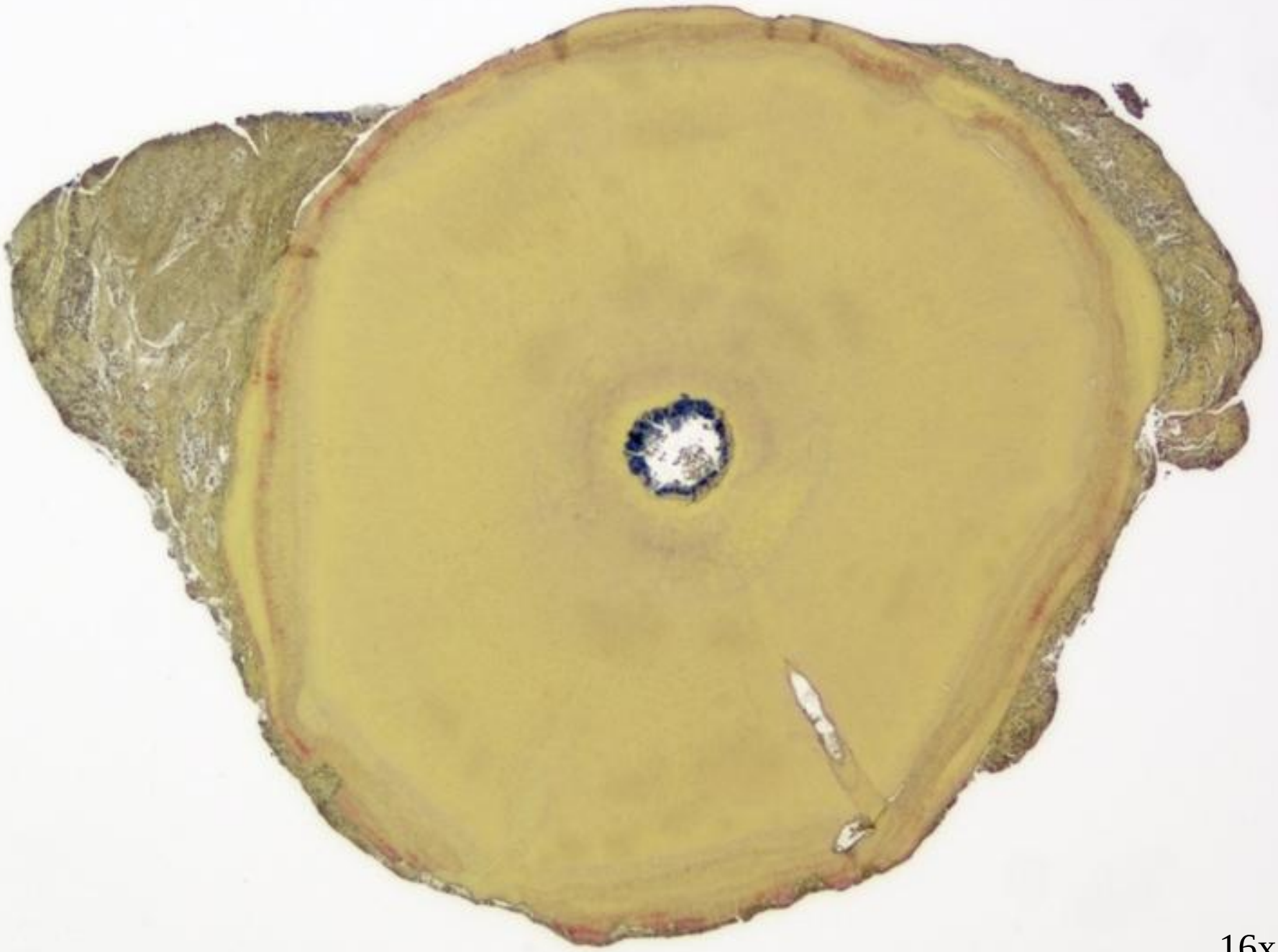

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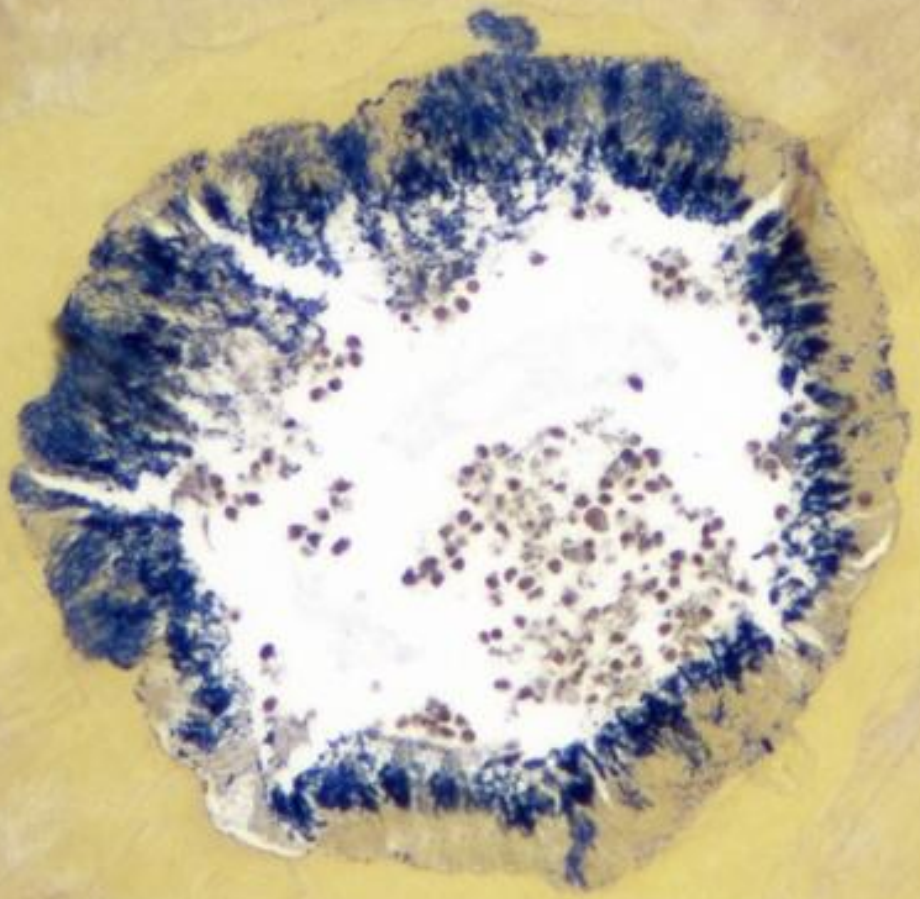

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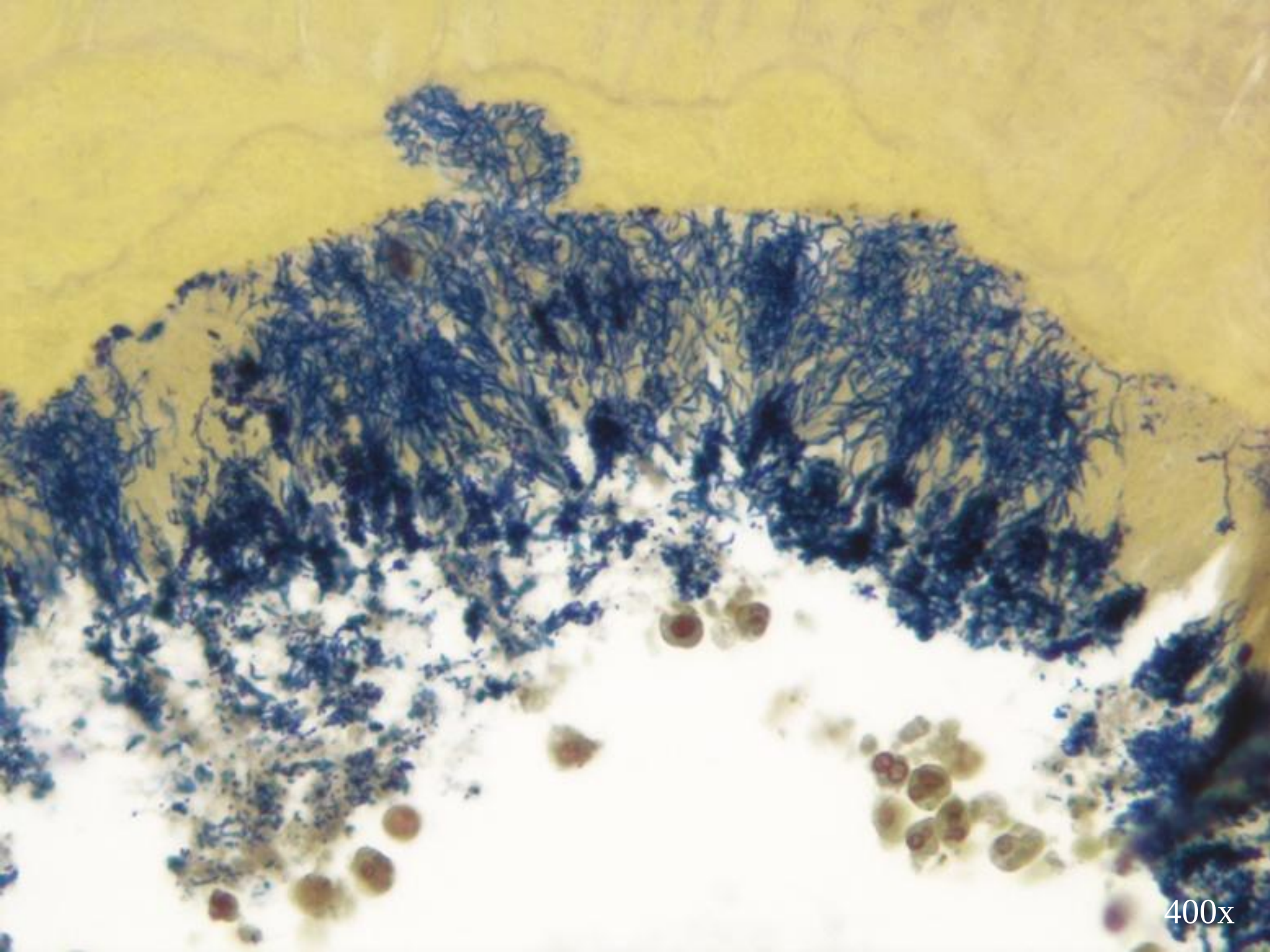
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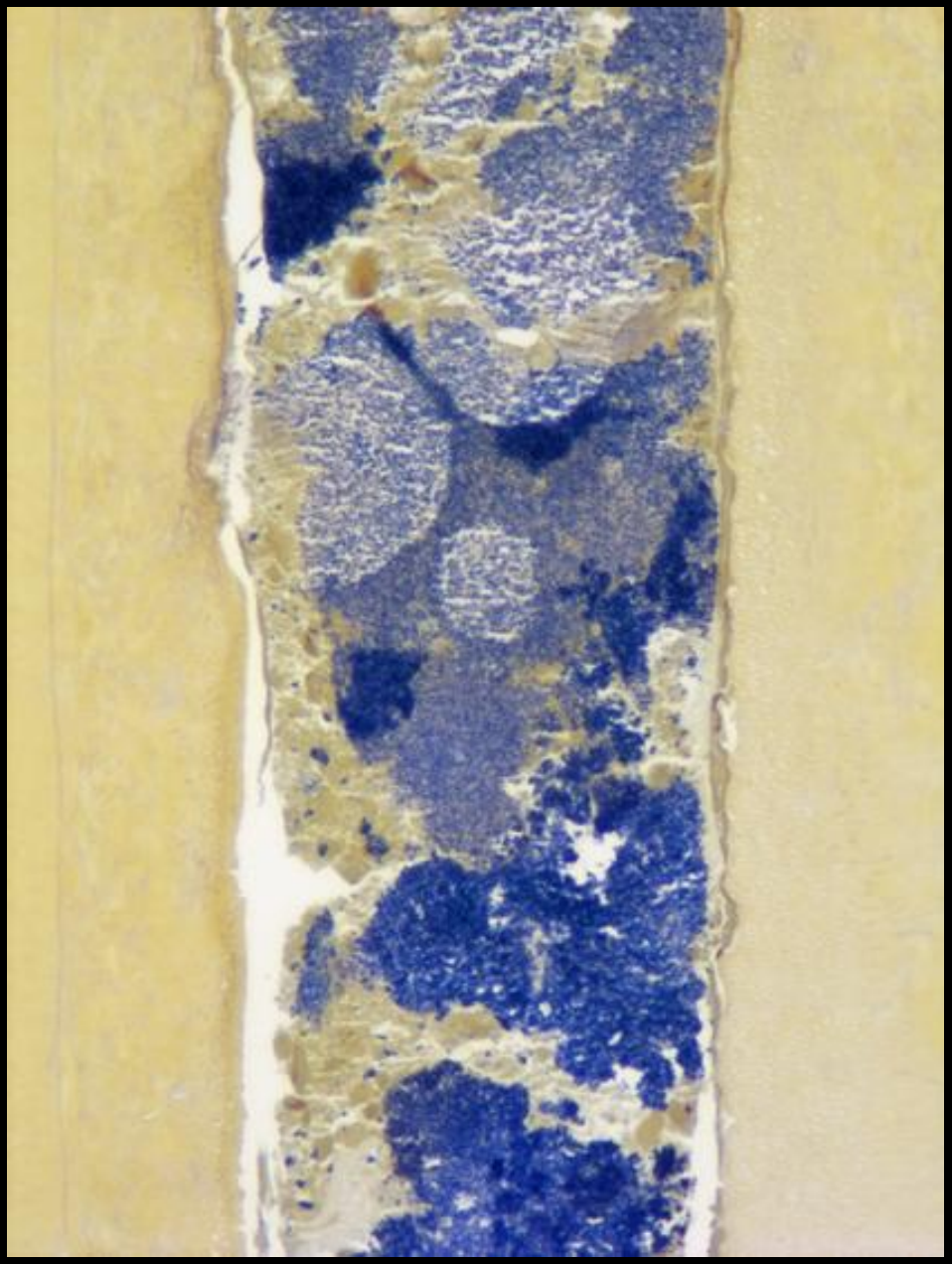

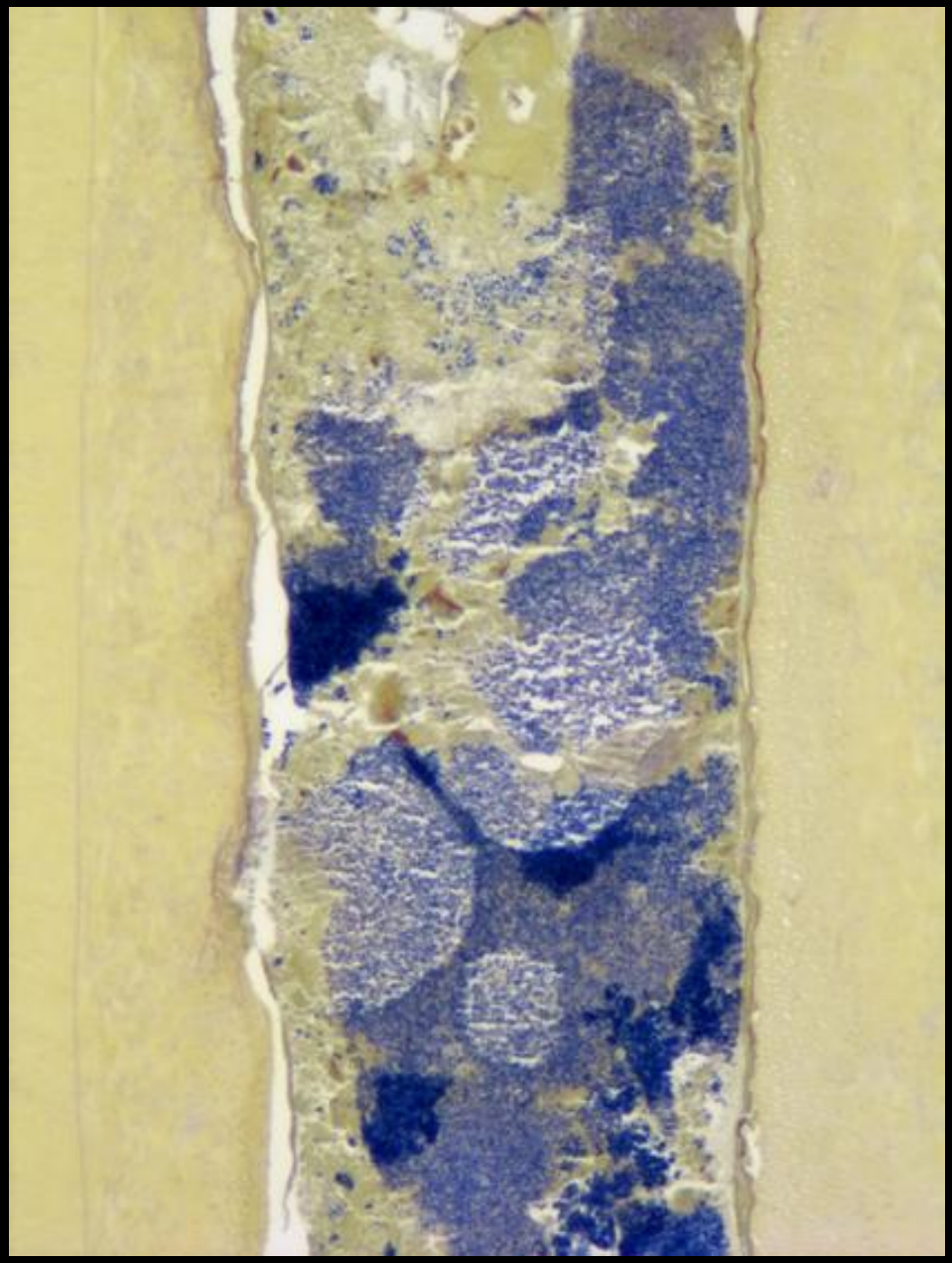

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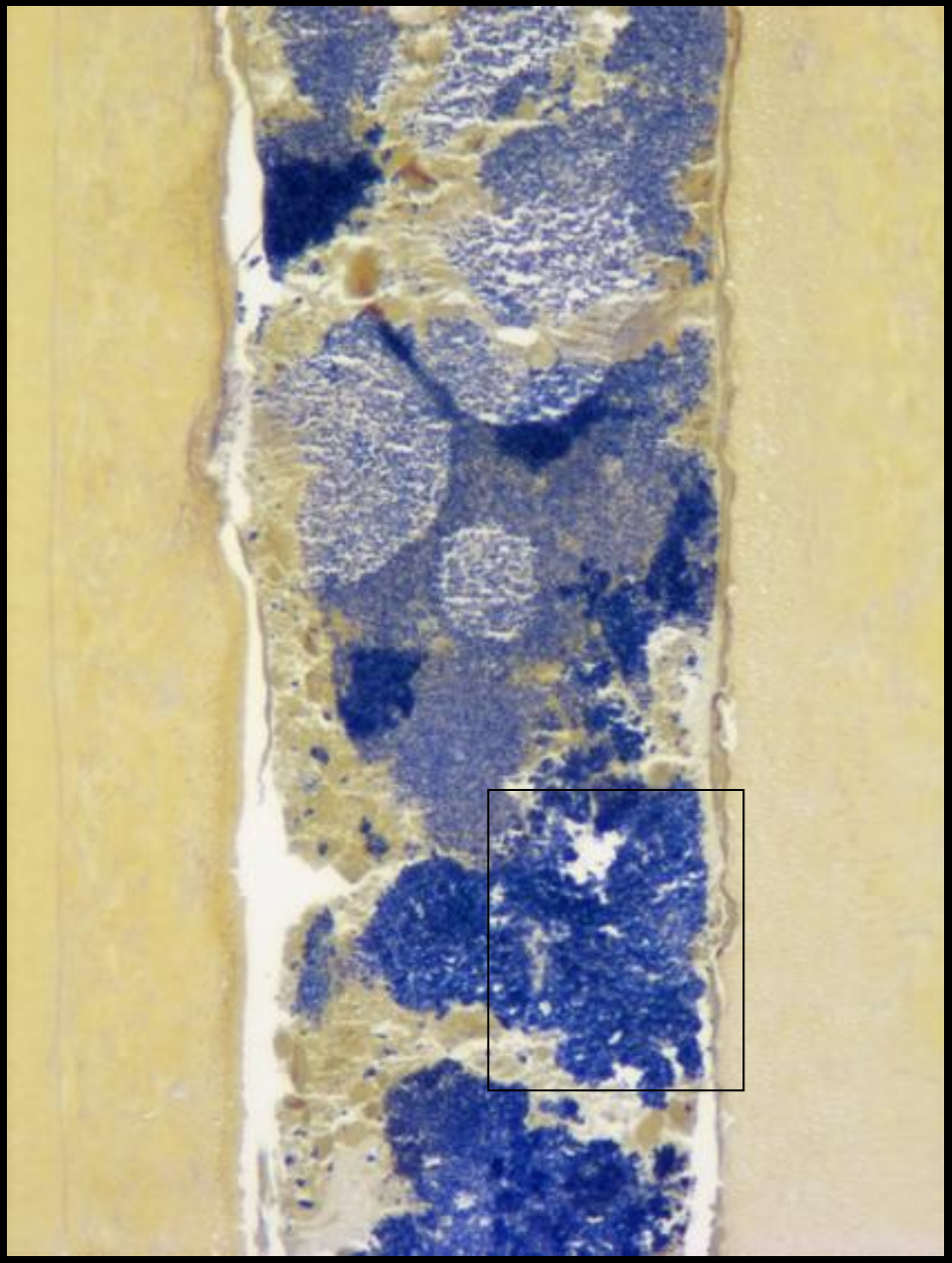

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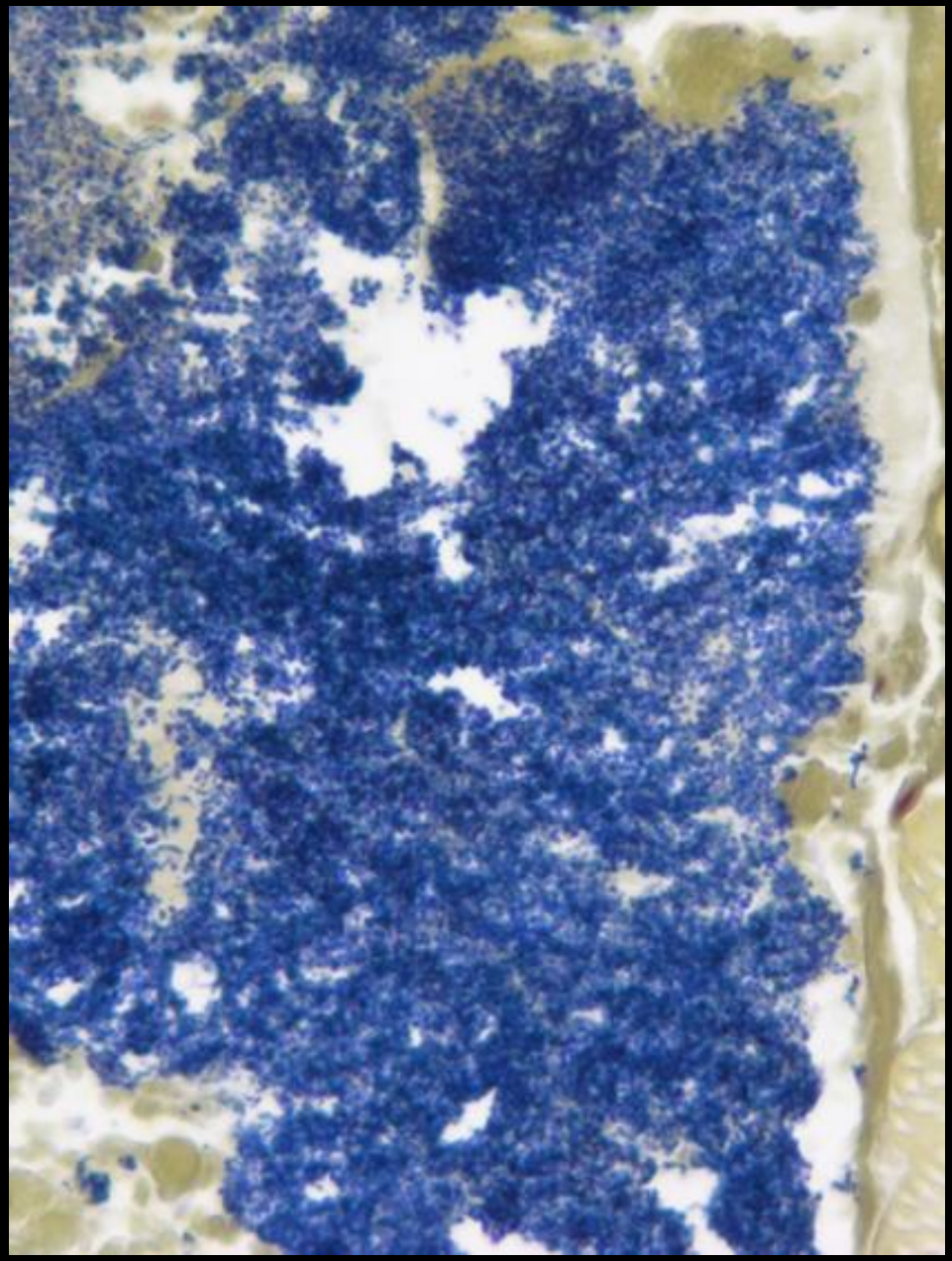

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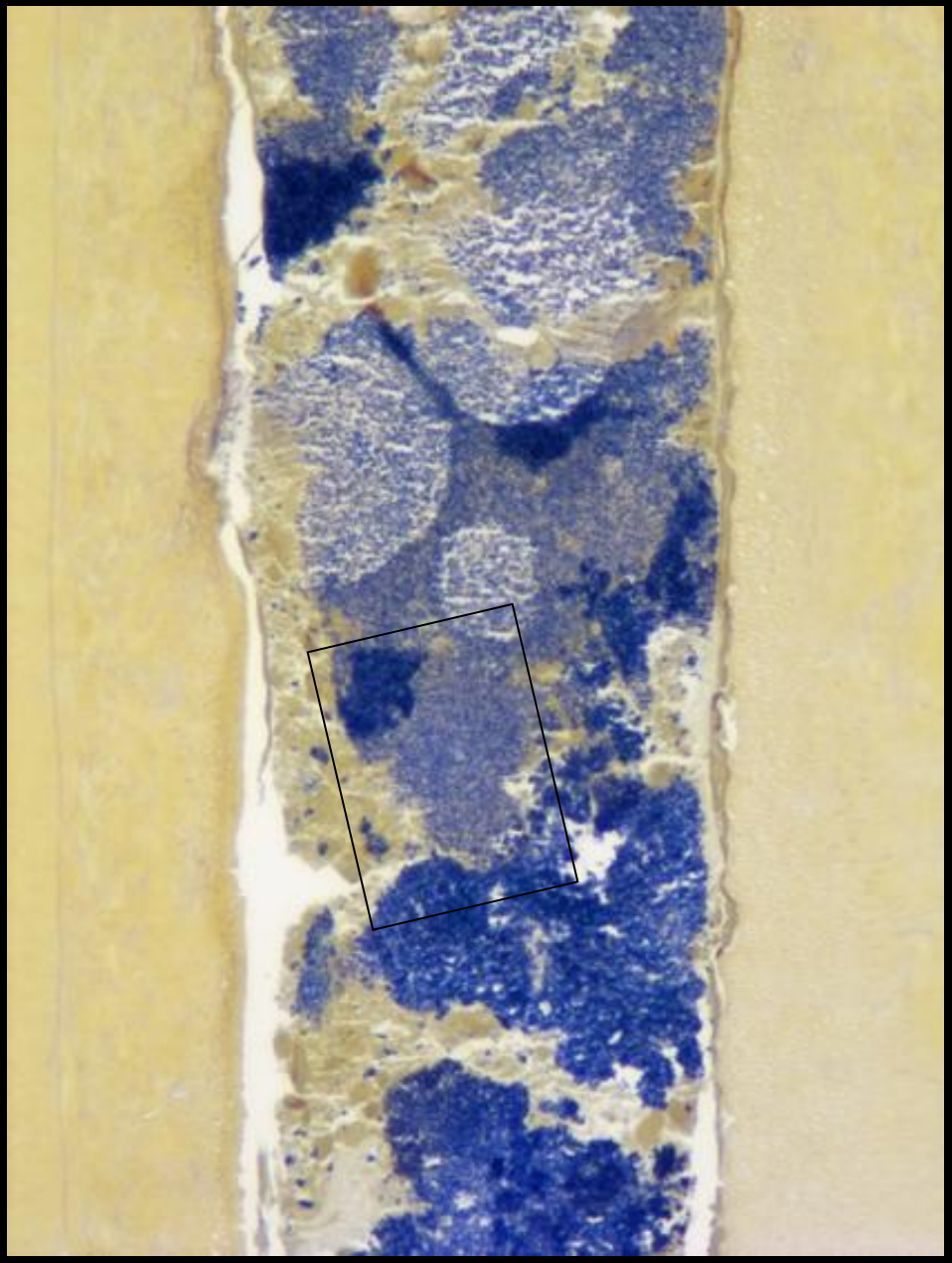

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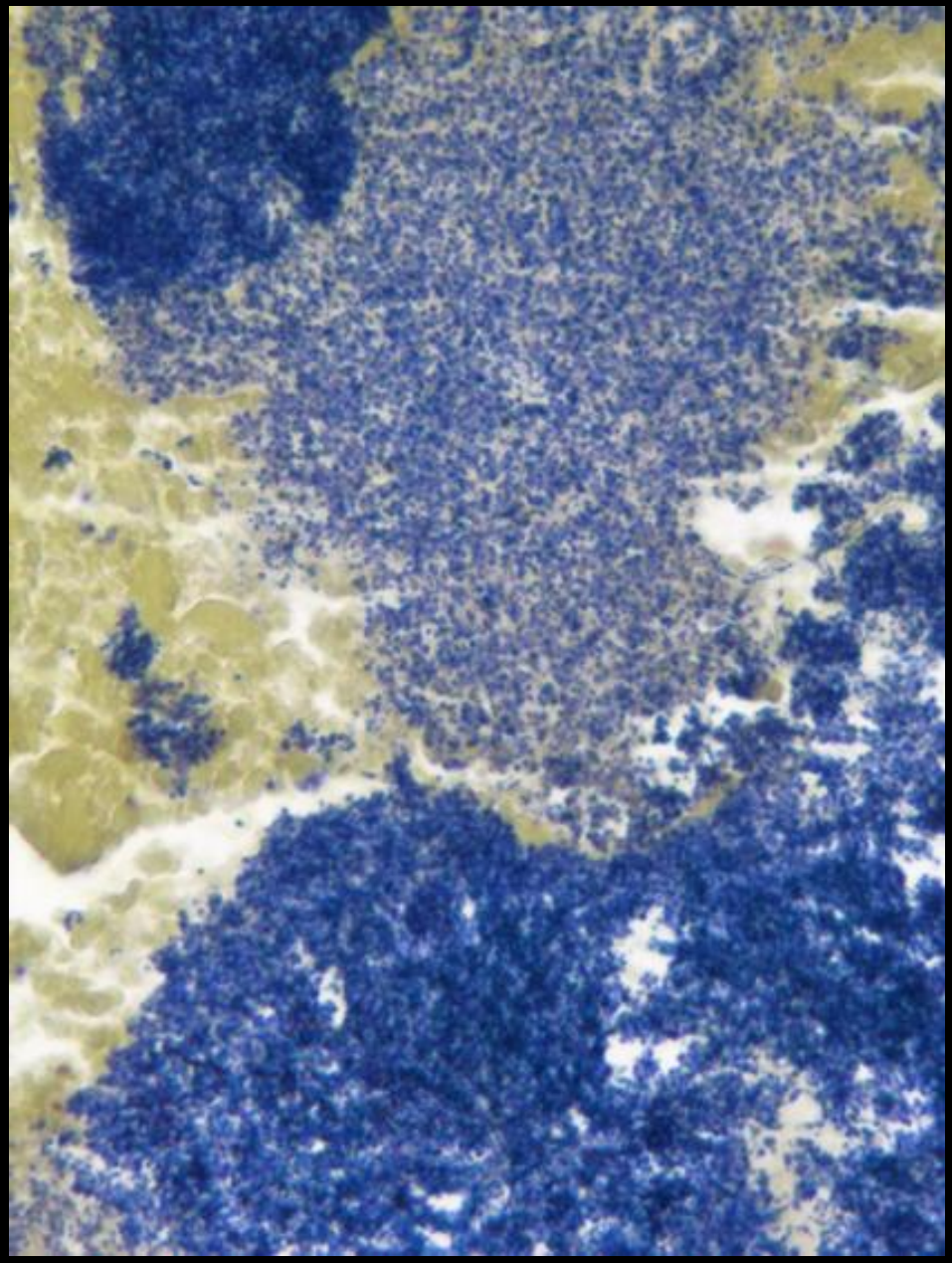

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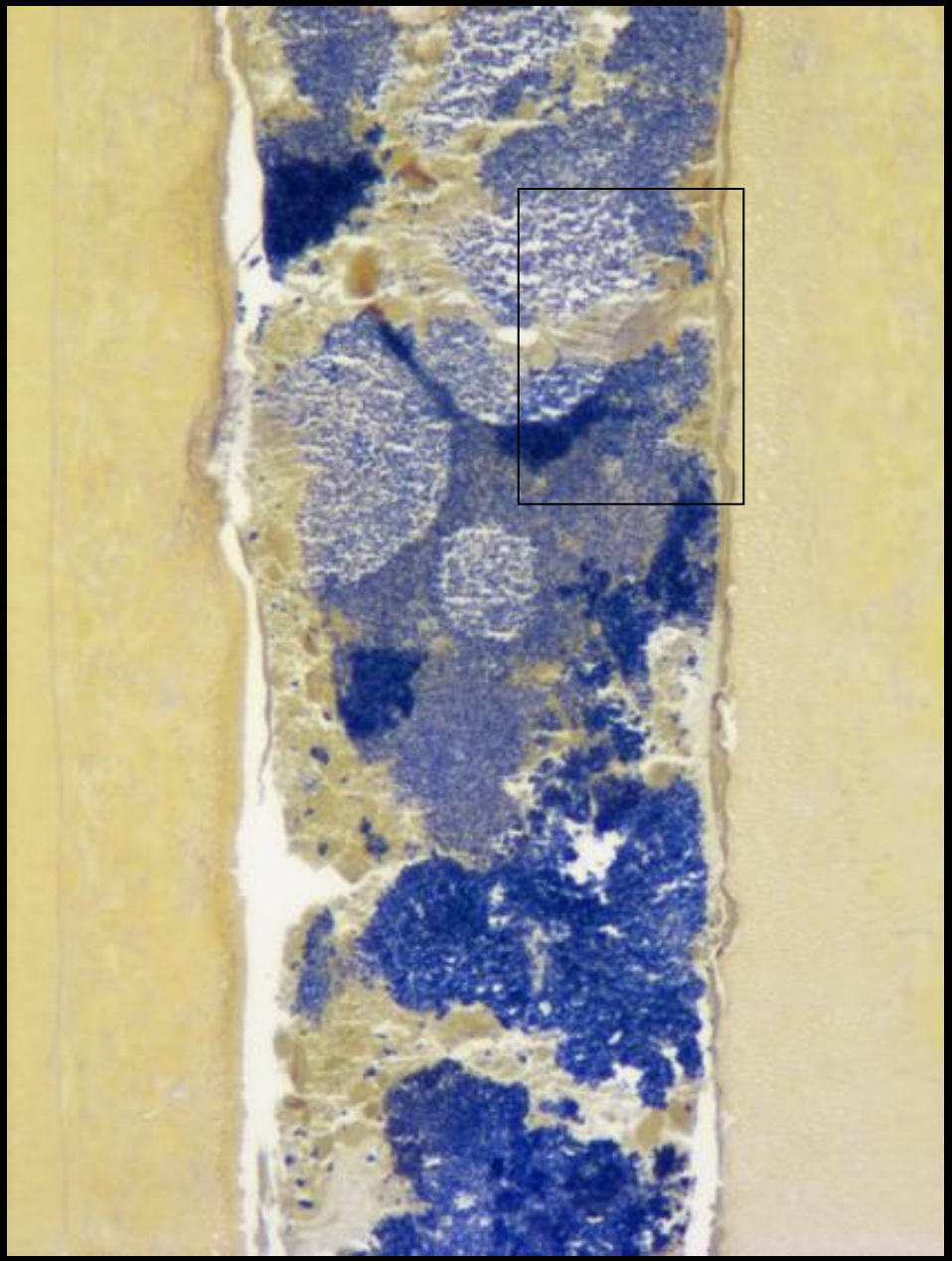

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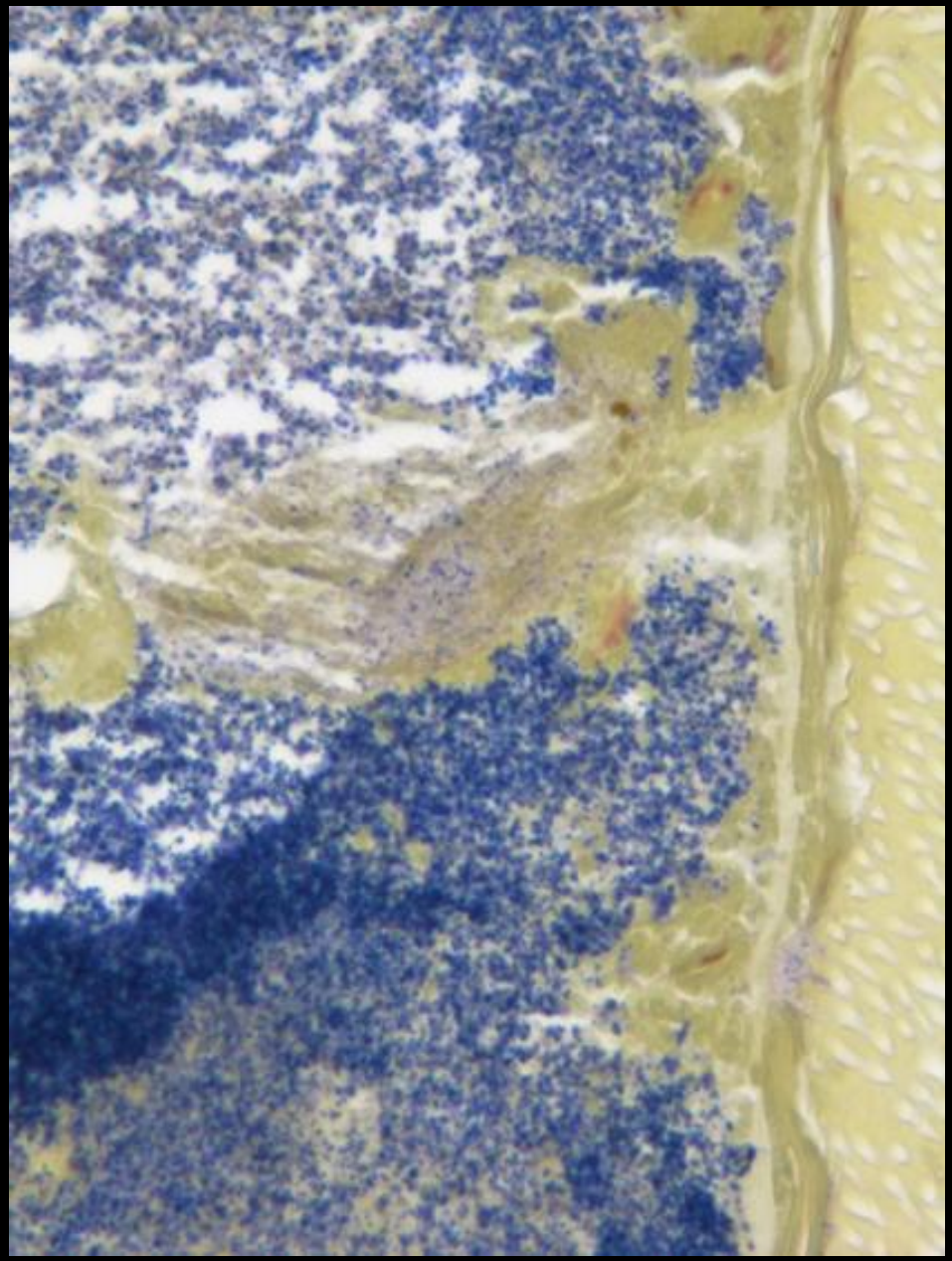

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Biofilms and Apical Periodontitis: Study of Prevalence and Association with Clinical and Histopathologic Findings

Domenico Ricucci, MD, DDS, and José F. Siqueira, Jr., DDS, MSc, PhD†*

Abstract

Introduction: This study evaluated the prevalence of bacterial biofilms in untreated and treated root canals of teeth evincing apical periodontitis. The associations of biofilms with clinical conditions, radiographic size, and the histopathologic type of apical periodontitis were also investigated. **Methods:** The material comprised biopsy specimens from 106 (64 untreated and 42 treated) roots of teeth with apical periodontitis. Specimens were obtained by apical surgery or extraction and were processed for histopathologic and histobacteriologic techniques. **Results:** Bacteria were found in all but one specimen. Overall, intraradicular biofilm arrangements were observed in the apical segment of 77% of the root canals (untreated canals: 80%; treated canals: 74%). Biofilms were also seen covering the walls of ramifications and isthmuses. Bacterial biofilms were visualized in 62% and 82% of the root canals of teeth with small and large radiographic lesions, respectively. All canals with very large lesions harbored intraradicular biofilms. Biofilms were significantly associated with epithelialized lesions (cysts and epithelialized granulomas or abscesses) ($p < 0.001$). The overall prevalence of biofilms in cysts, abscesses, and granulomas was 95%, 83%, and 69.5%, respectively. No correlation was found between biofilms and clinical symptoms or sinus tract presence ($p > 0.05$). Extraradicular biofilms were observed in only 6% of the cases. **Conclusions:** The overall findings are consistent with acceptable criteria to include apical periodontitis in the set of biofilm-induced diseases. Biofilm morphologic structure varied from case to case and no unique pattern for endodontic infections was identified. Biofilms are more likely to be present in association with longstanding pathologic processes, including large lesions and cysts. (*J Endod 2010;36:1277–1288*)

Key Words

Apical periodontitis, bacterial biofilm, endodontic infection, endodontic treatment

In their natural habitats, microorganisms almost invariably live as members of metabolically integrated communities usually attached to surfaces to form biofilms (1). The biofilm community lifestyle provides microorganisms with a series of advantages and skills that are not observed for individual cells living in a free-floating (planktonic) state including establishment of a broader habitat range for growth; increased metabolic diversity and efficiency; protection against competing microorganisms, host defenses, antimicrobial agents, and environmental stress; and enhanced pathogenicity (2). The study of microbial biofilms assumes a great importance in different sectors of industrial, environmental, and medical microbiology. In medical microbiology, biofilms have been increasingly studied and estimates indicate that biofilm infections comprise 65% to 80% of the human infections in the developed world (3). As for the oral cavity, caries, gingivitis, and marginal periodontitis are examples of diseases caused by bacterial biofilms in the form of supragingival or subgingival dental plaque.

Mounting evidence indicates that apical periodontitis is also a biofilm-induced disease (4–6). *In situ* investigations using optical and/or electron microscopy have allowed observations of bacteria colonizing the root canal system in primary or persistent/secondary infections as sessile biofilms covering the dentinal walls (7–12). Apical ramifications, lateral canals, and isthmuses connecting main root canals have all been shown to harbor bacterial cells, which are also frequently organized in biofilm-like structures (13–15). In addition, biofilms adhered to the apical root surface (extraradicular biofilms) have been reported and regarded as a possible cause of posttreatment apical periodontitis (16, 17).

Although the concept of apical periodontitis as a biofilm-induced disease has been built upon these observations, the prevalence of biofilms and their association with clinical and histopathologic findings have not yet been reported. Before this information becomes available, it may seem somewhat imprecise to generalize and categorize apical periodontitis as a biofilm-induced disease. The purpose of the present study was twofold: (1) evaluate the prevalence of intraradicular and extraradicular bacterial biofilms in untreated and treated root canals of human teeth evincing apical periodontitis through a histobacteriologic approach and (2) look for associations of biofilms with some clinical conditions, radiographic size, and the histopathologic type of apical periodontitis lesions.

Materials and Methods

Clinical Specimens

The material for this study consisted of sequential biopsies of roots or root tips together with surrounding apical periodontitis lesions. Specimens were part of the histologic collection of one of the authors (DR). The material comprised 106 roots from 100 human teeth. Of these, 58 were teeth with untreated root canals (6 incisors, 3 canines, 18 premolars, and 31 molars) from 52 patients (25 females, 27 males) aged 18 to 75 years (mean, 42 years). In total, 64 roots from untreated teeth were available, of which 59 were extracted with apical periodontitis lesions attached while in the other

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0099-2399/$0 - see front matter

doi:10.1016/j.joen.2010.04.007

Methods

The material comprised biopsy specimens from 106 (64 untreated and 42 treated) roots of teeth with apical periodontitis.

Overall, intraradicular biofilm arrangements were observed in the apical segment of 77% of the root canals.

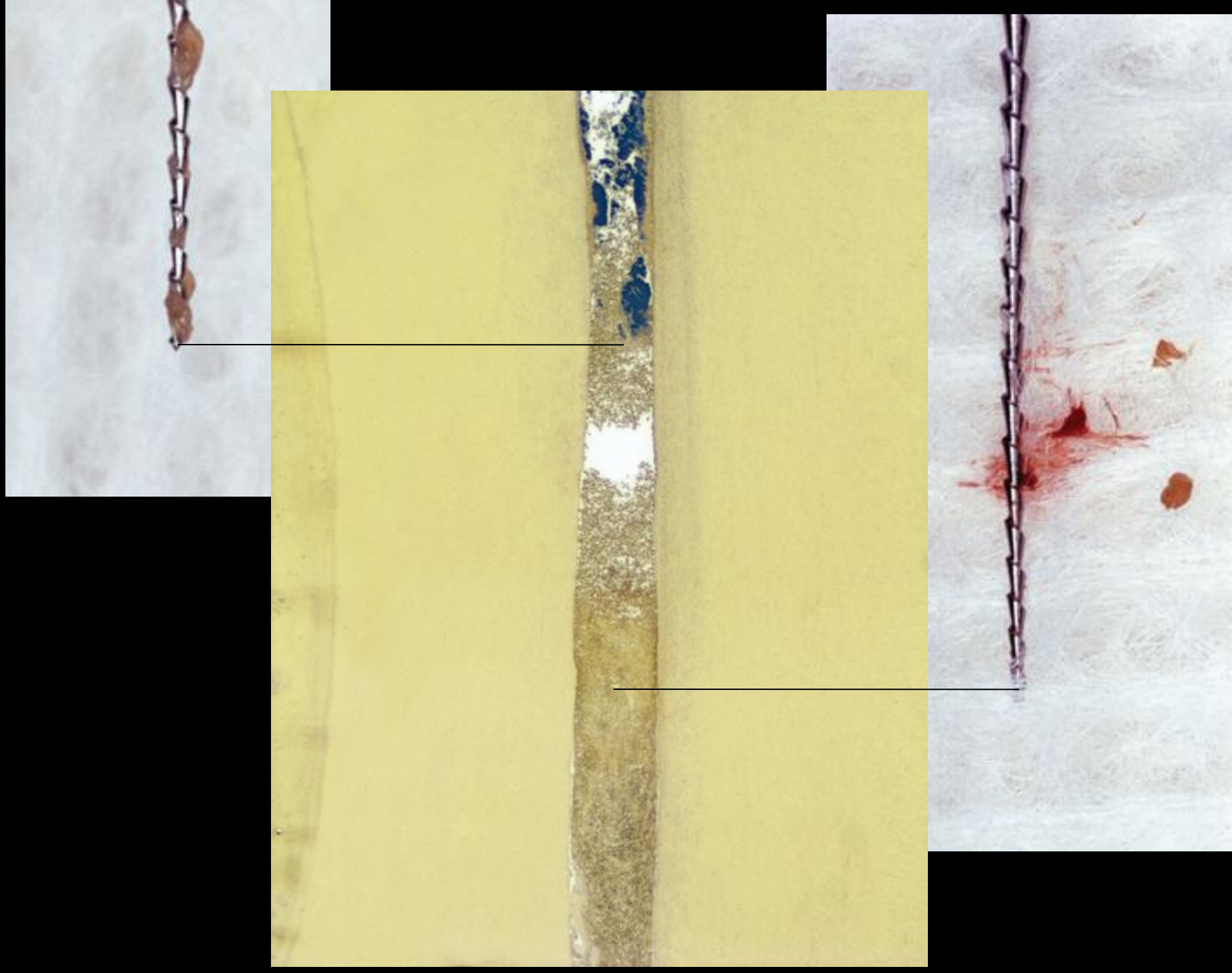
Conclusions

The overall findings are consistent with acceptable criteria to include apical periodontitis in the set of biofilm induced diseases. Biofilm morphologic structure varied from case to case and no unique pattern for endodontic infections was identified. Biofilms are more likely to be present in association with longstanding pathologic processes, including large lesions and cysts.

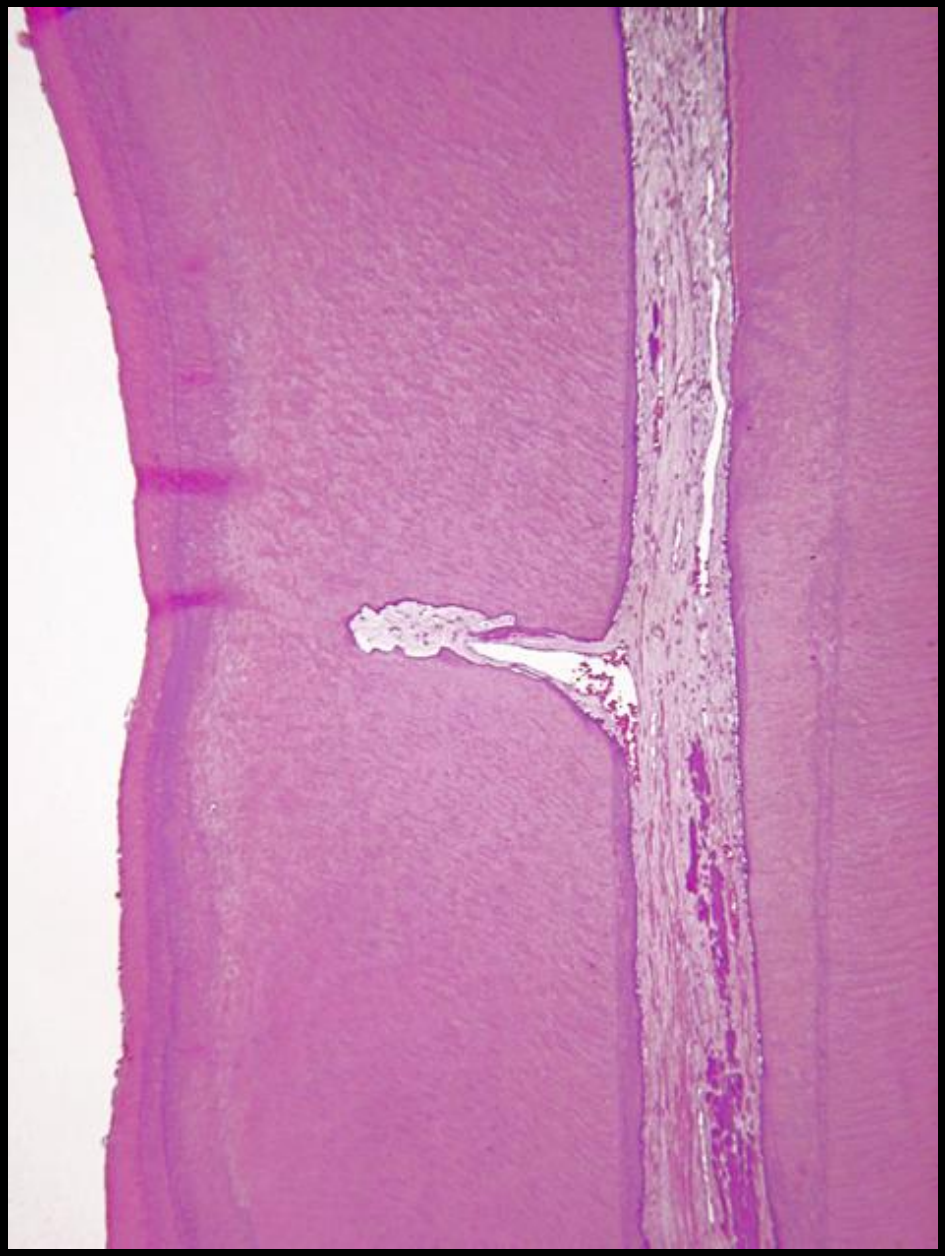
Bacterial biofilms were visualized in 62% and 82% of the root canals of teeth with small and large radiographic lesions, respectively.

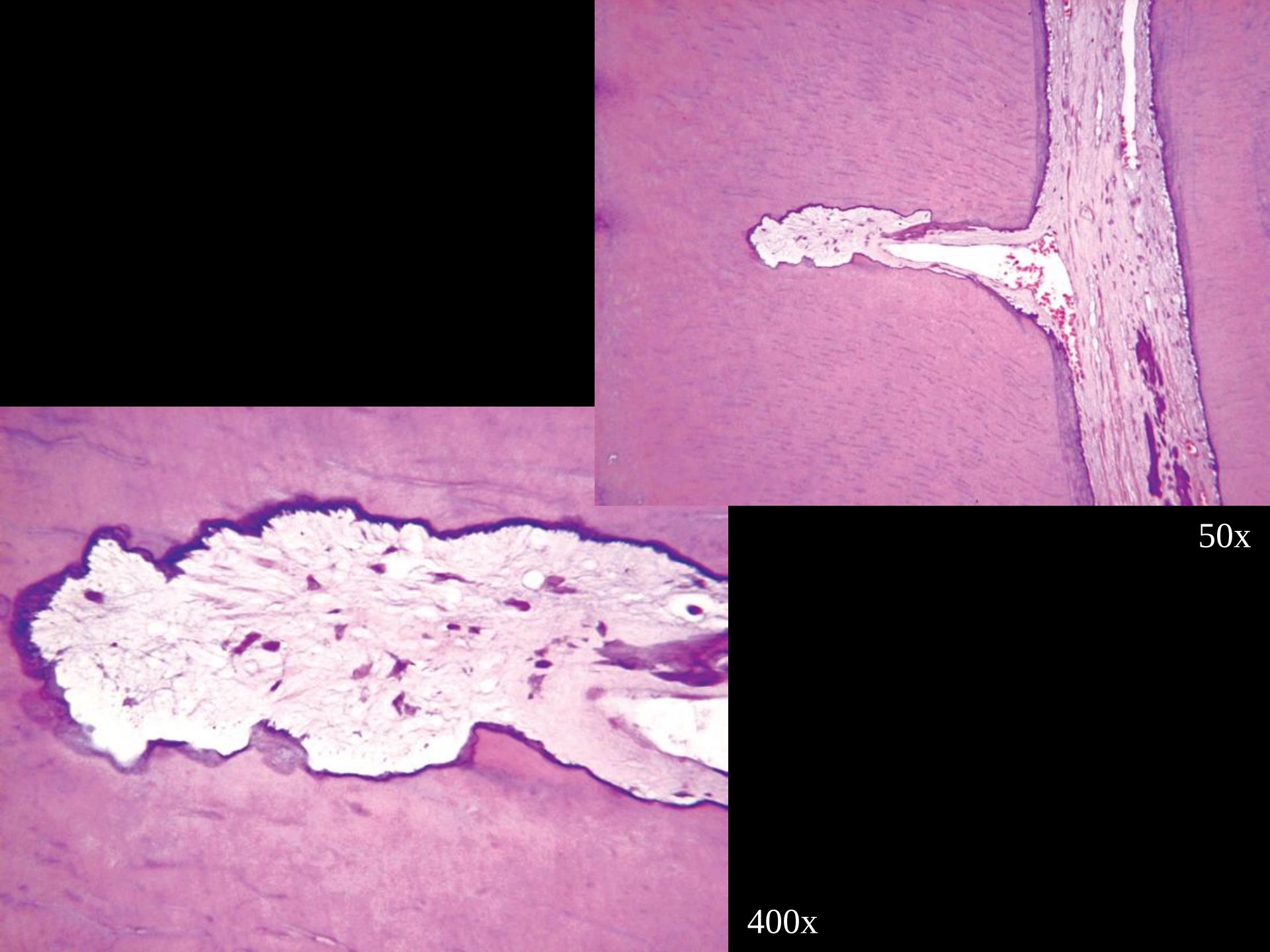
All canals with very large lesions harbored intraradicular biofilms. Biofilms were significantly associated with epithelialized lesions (cysts and epithelialized granulomas or abscesses) ($p < 0.001$). The overall prevalence of biofilms in cysts, abscesses, and granulomas was 95%, 83%, and 69.5%, respectively. No correlation was found between biofilms and clinical symptoms or sinus tract presence ($p > 0.05$).

Extraradicular biofilms were observed in only 6% of the cases.



Lateral canals & apical ramifications

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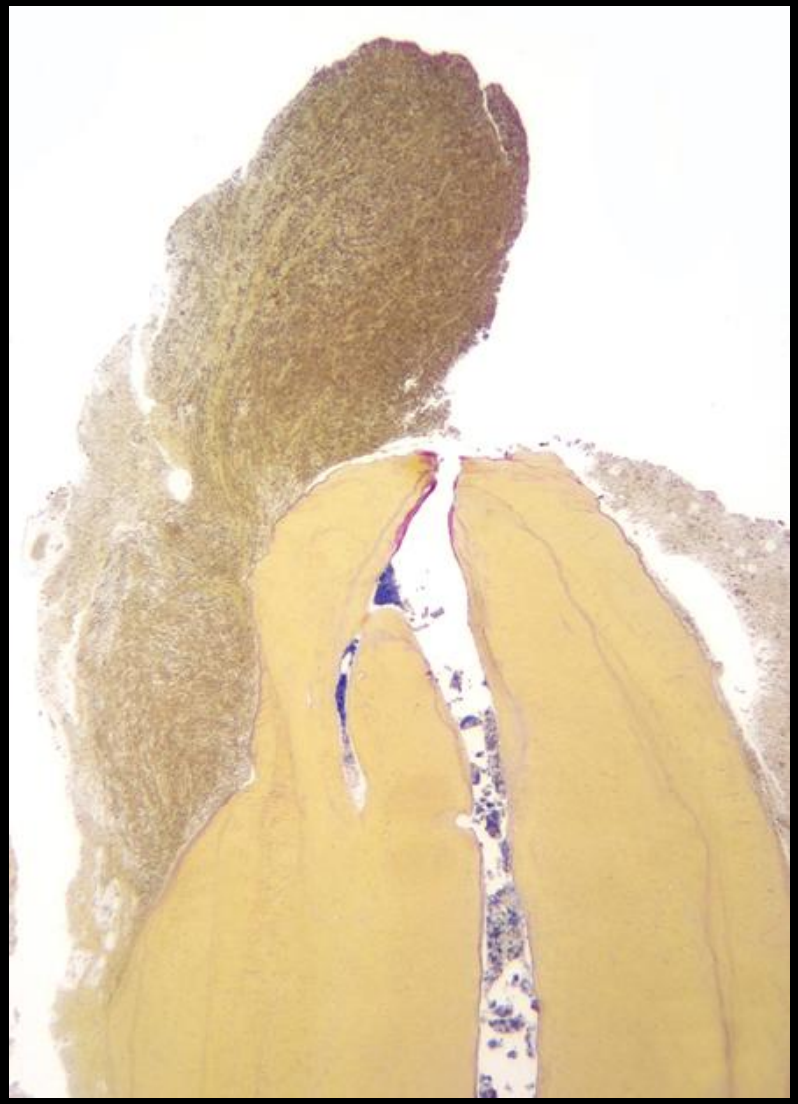

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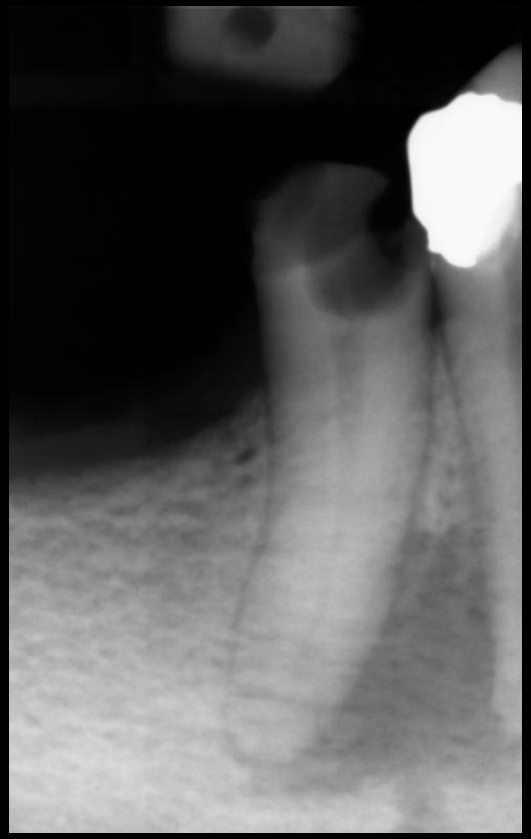

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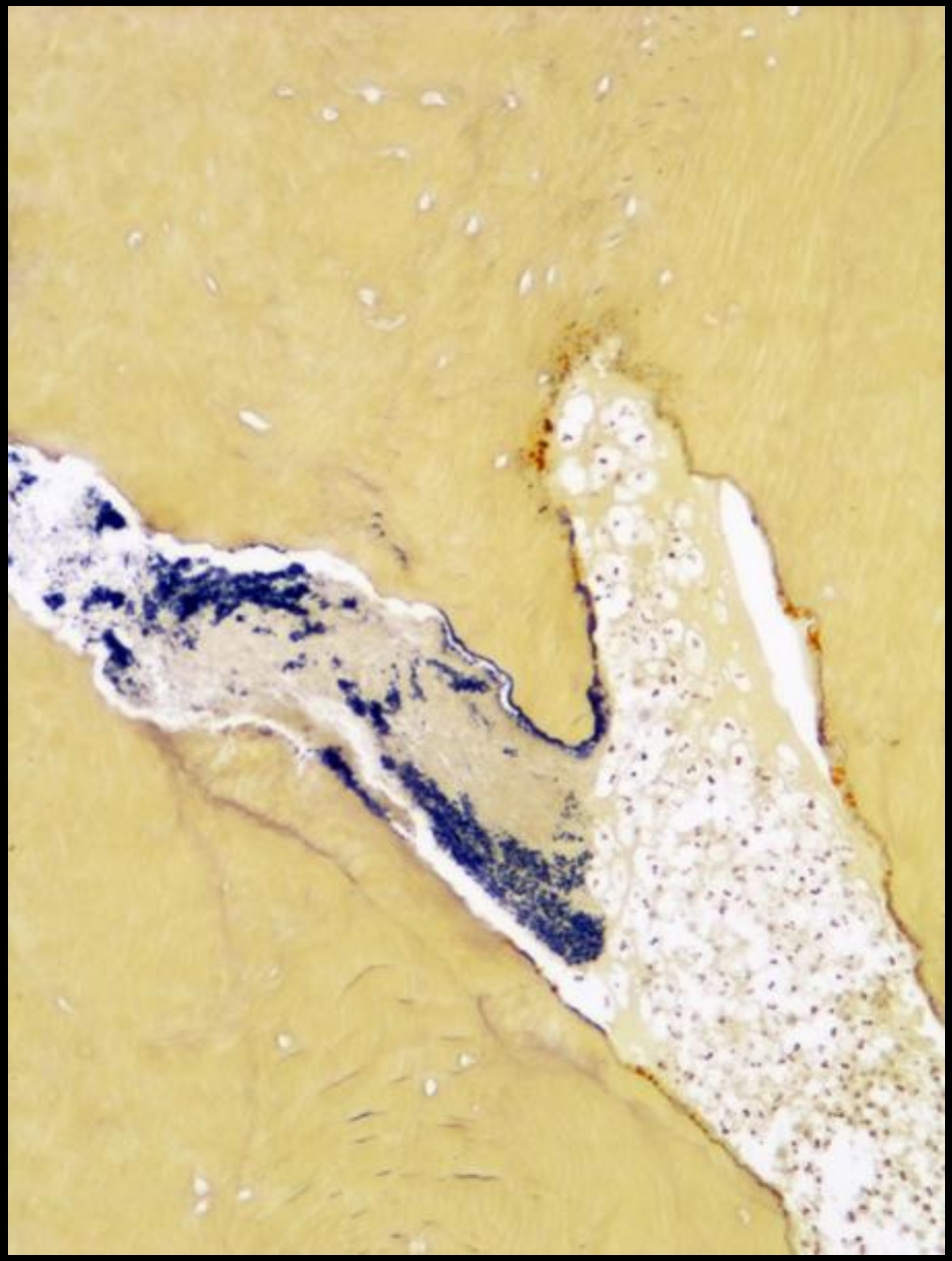

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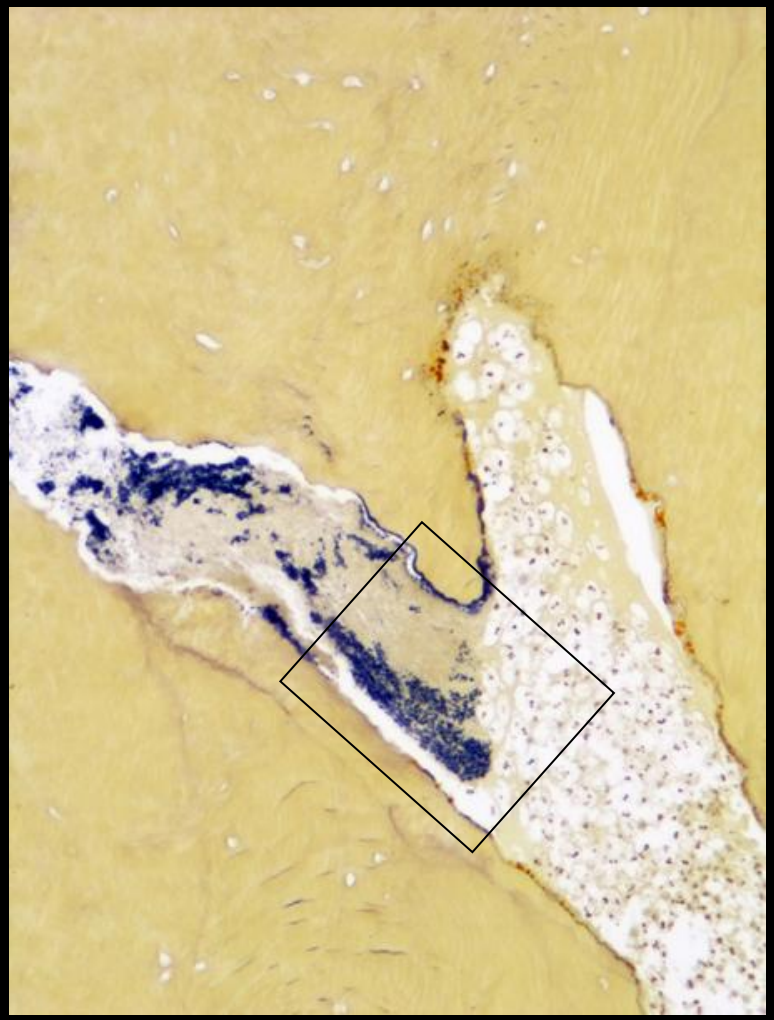

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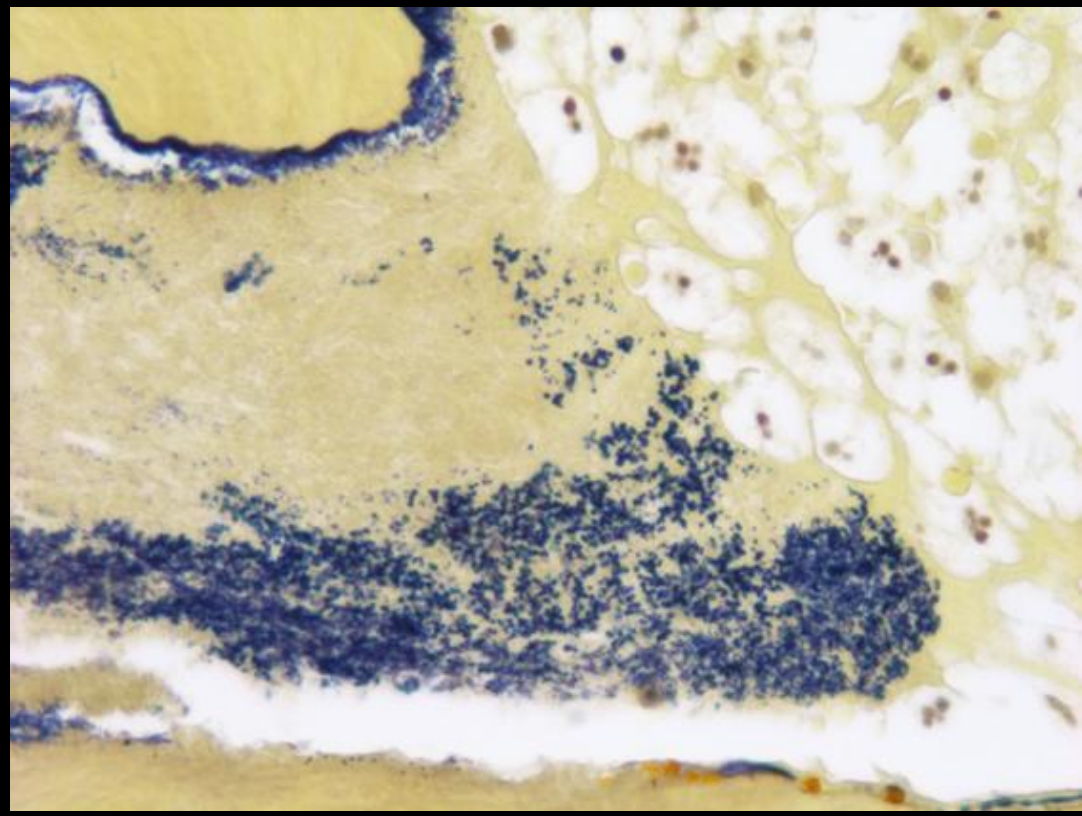

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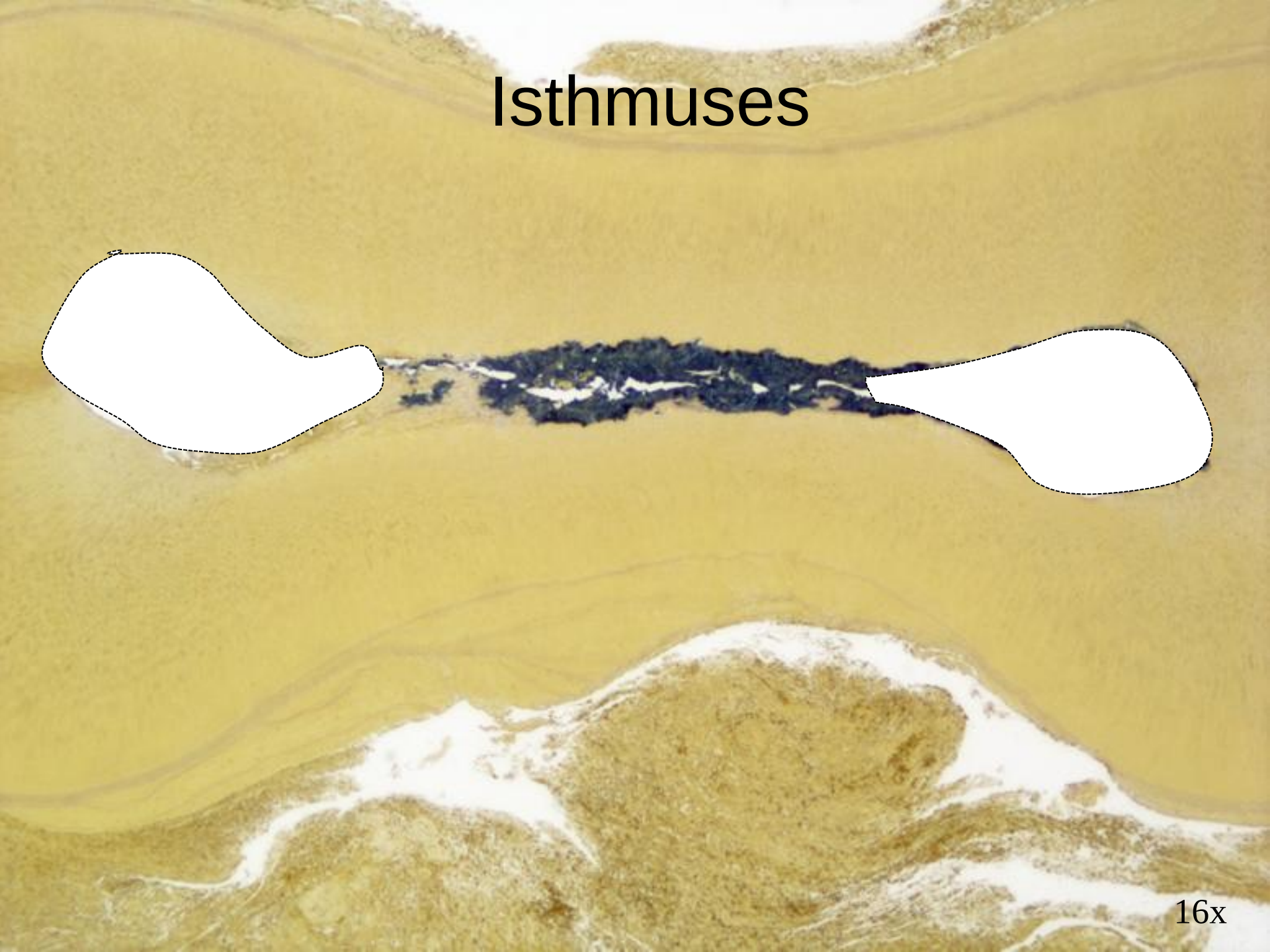
Isthmuses



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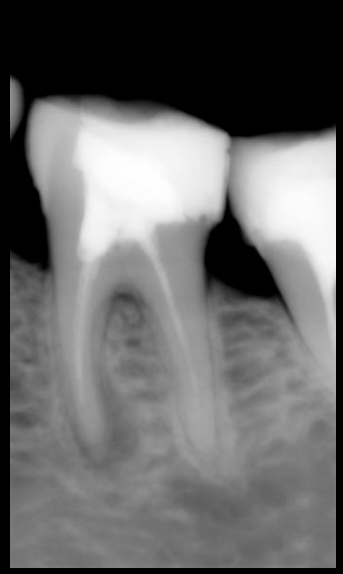
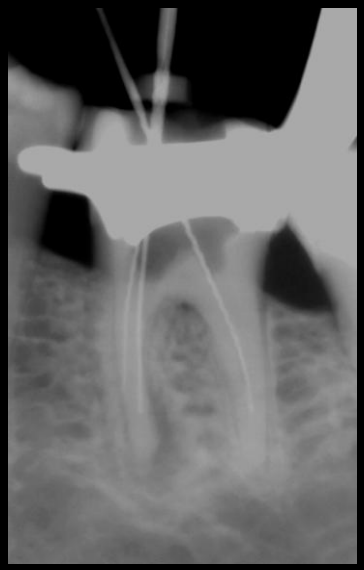
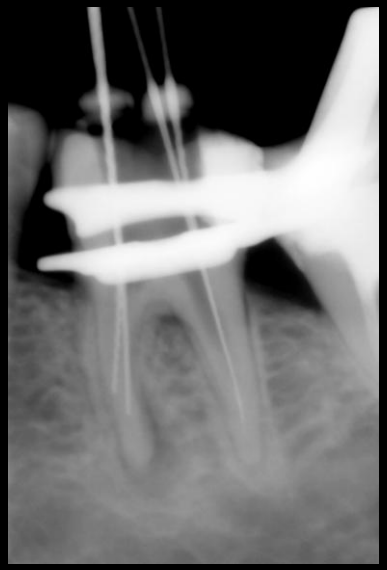
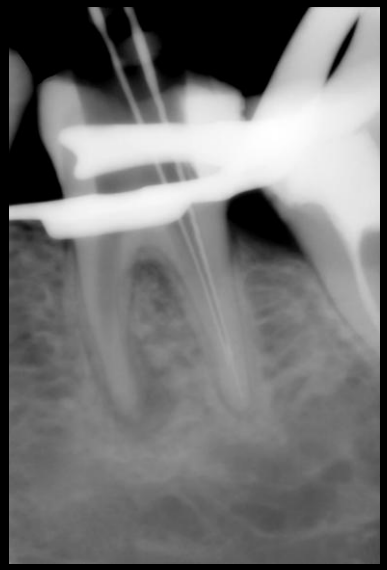
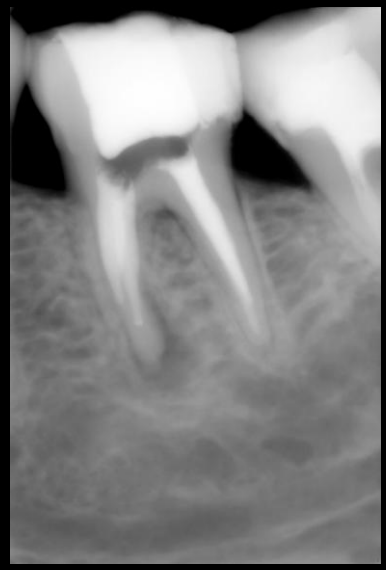
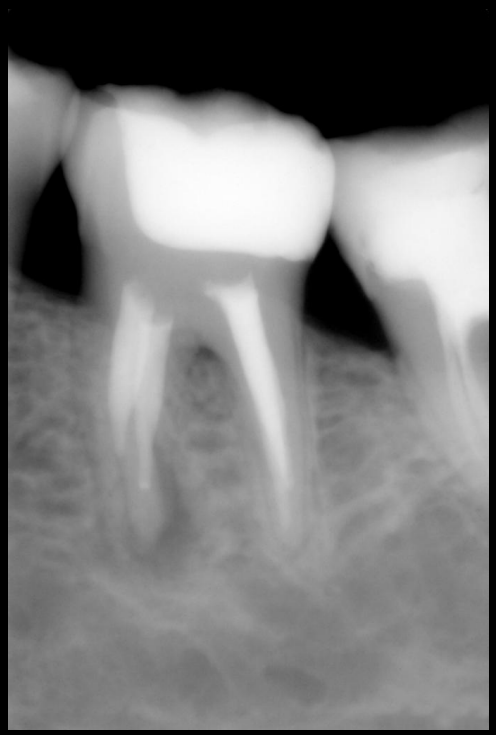
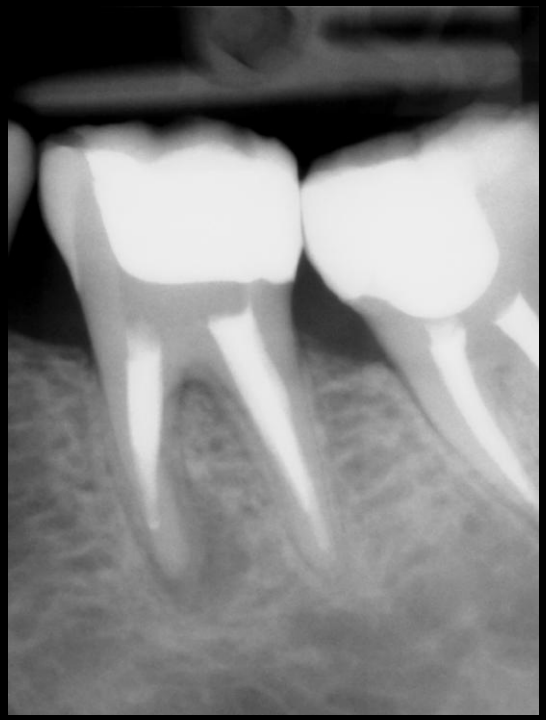

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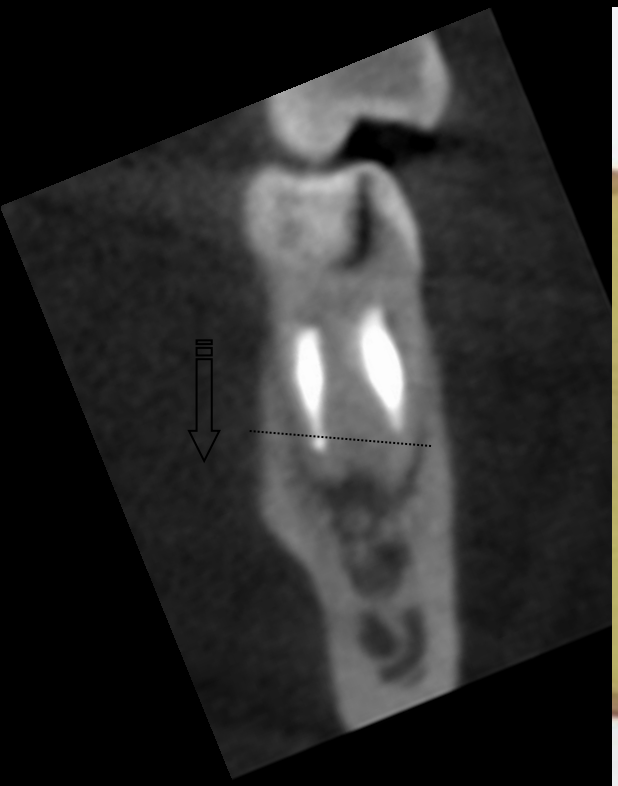

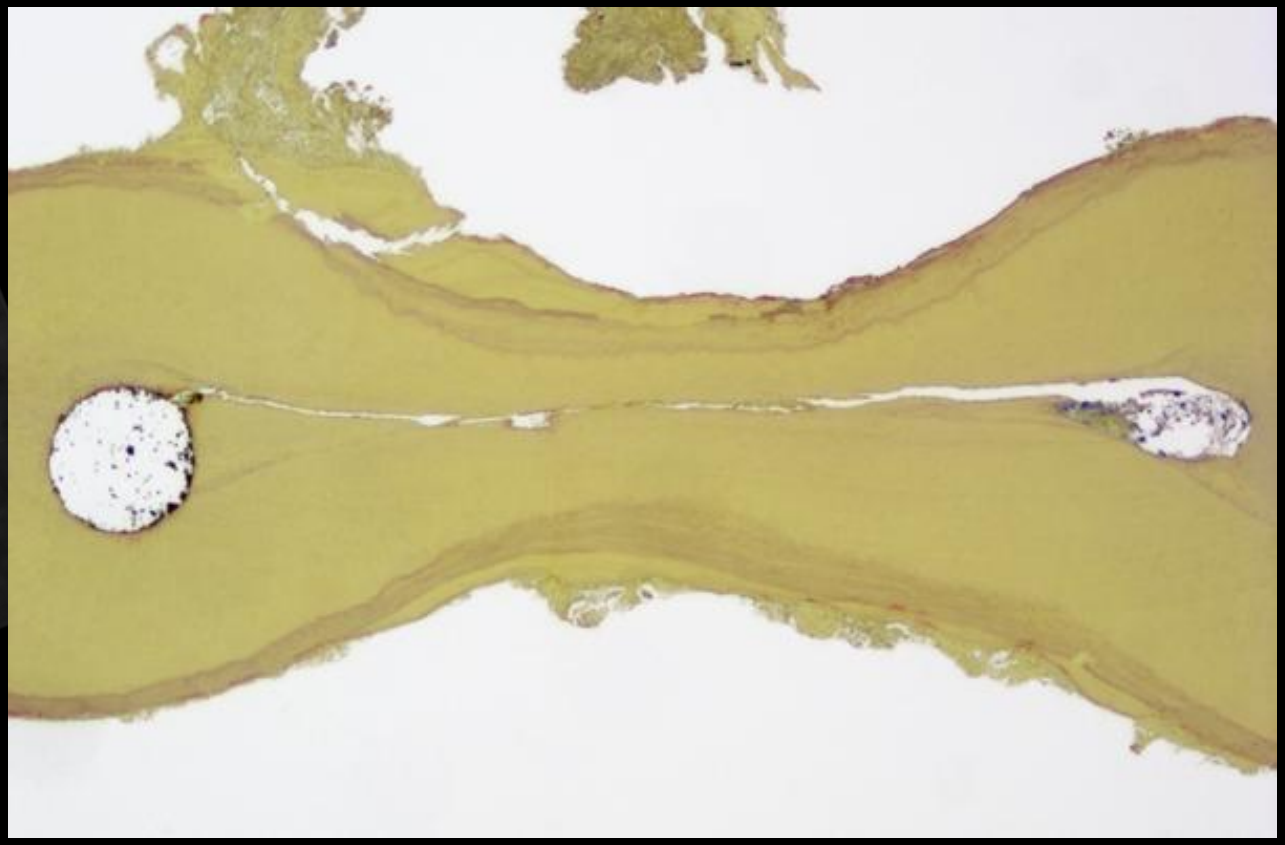

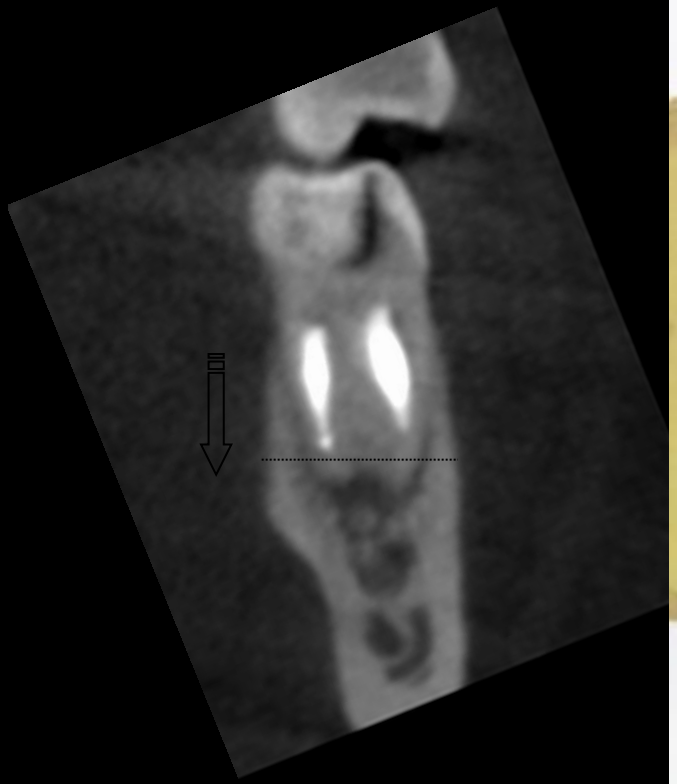

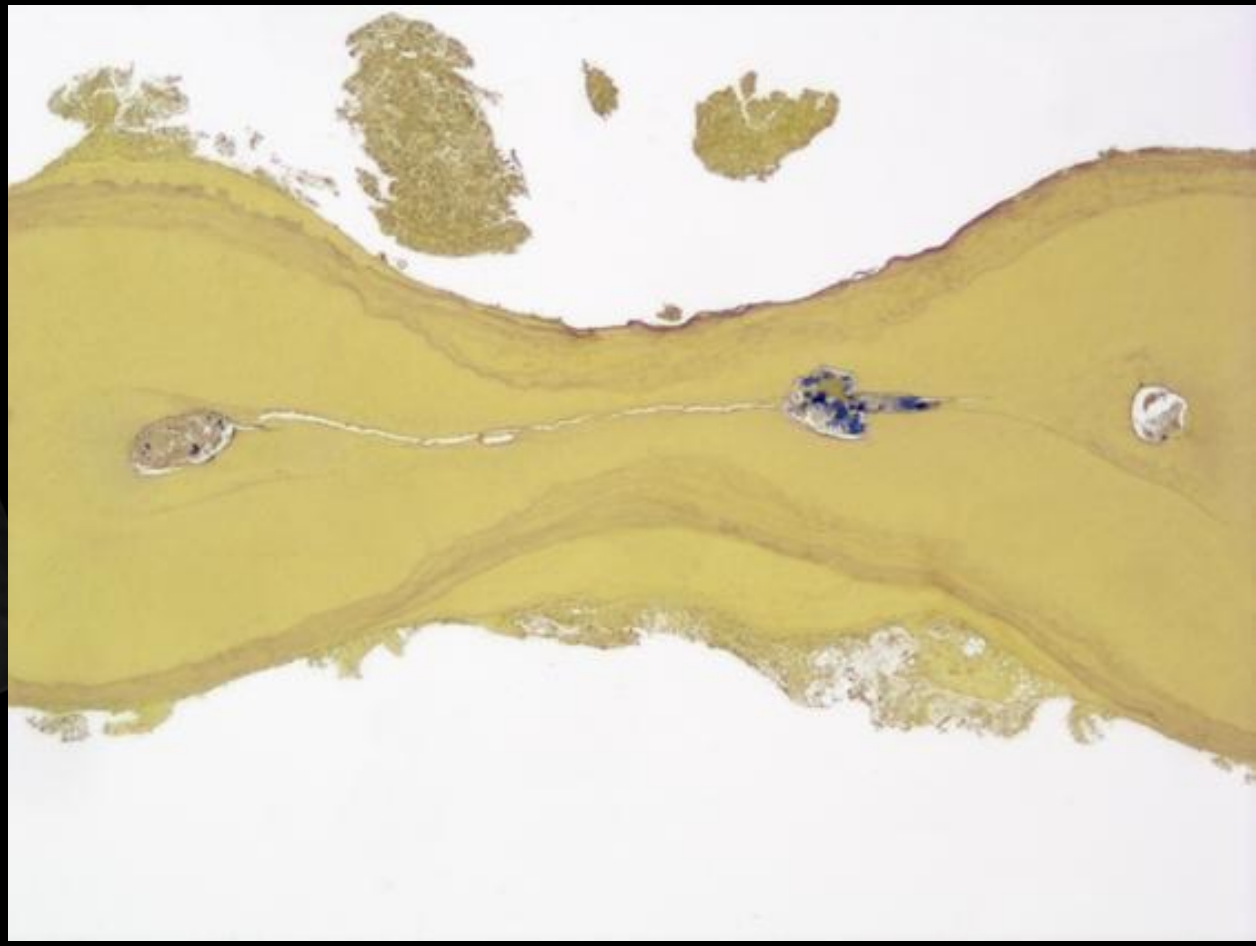

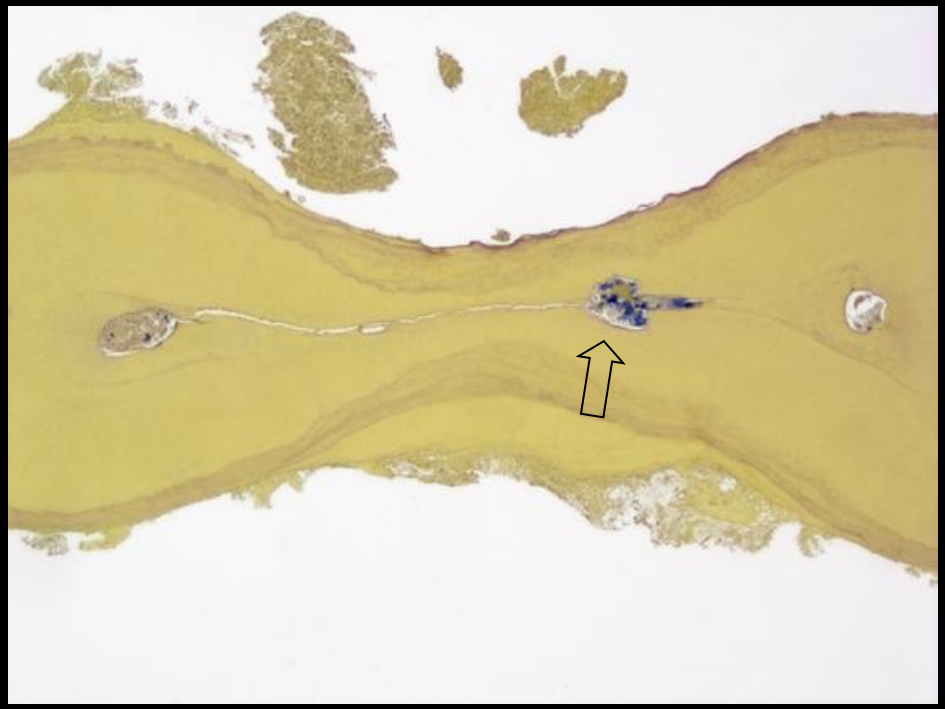

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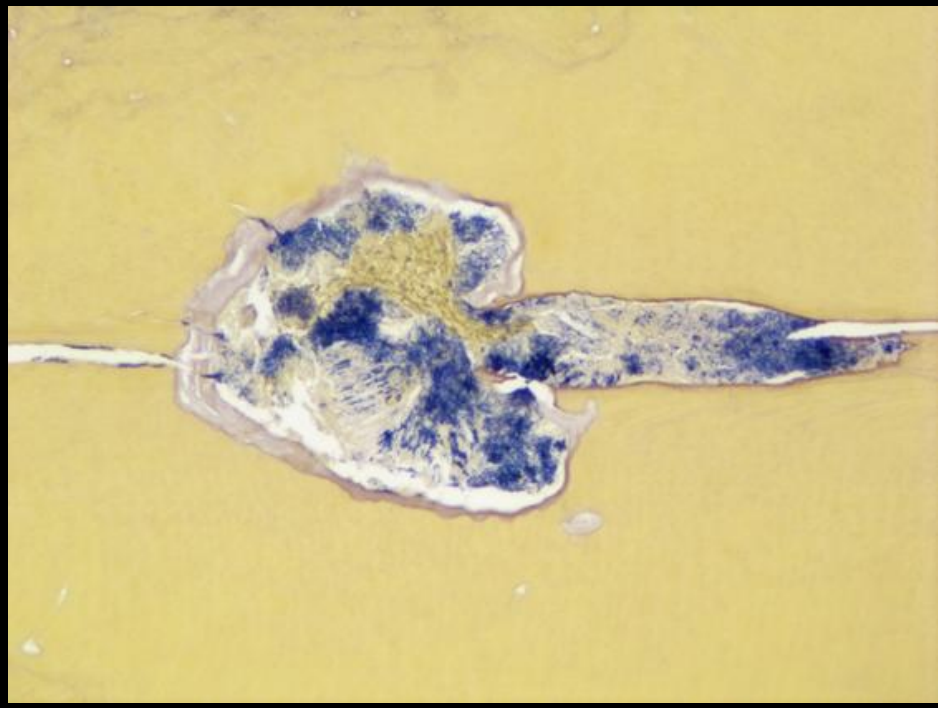

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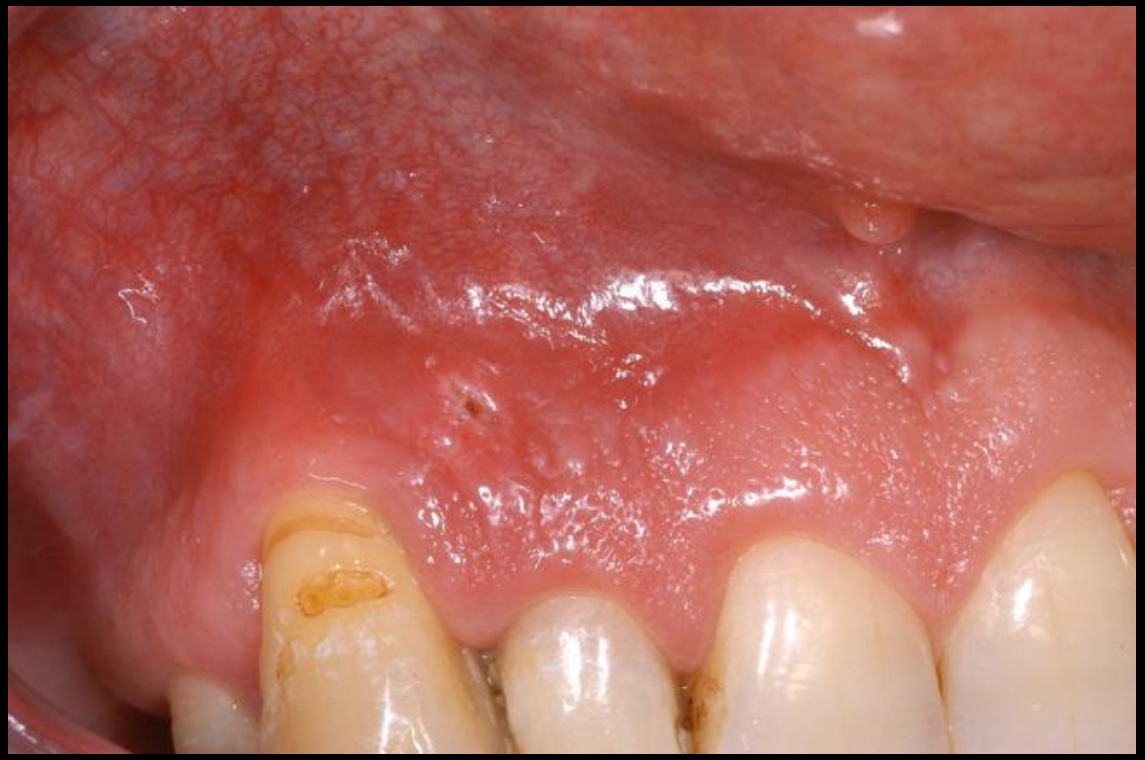
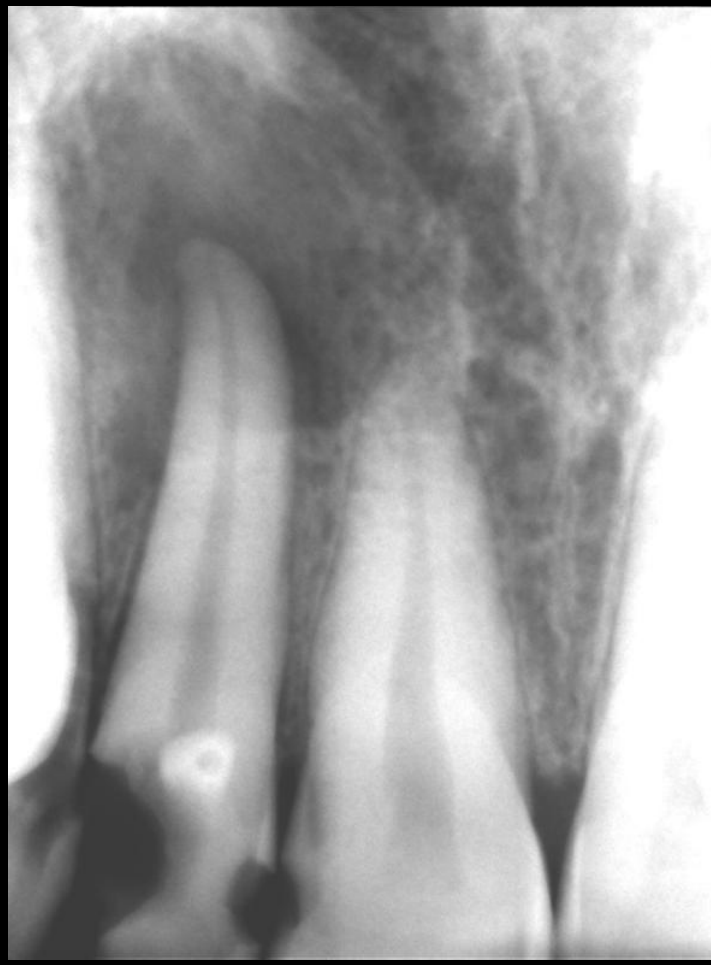
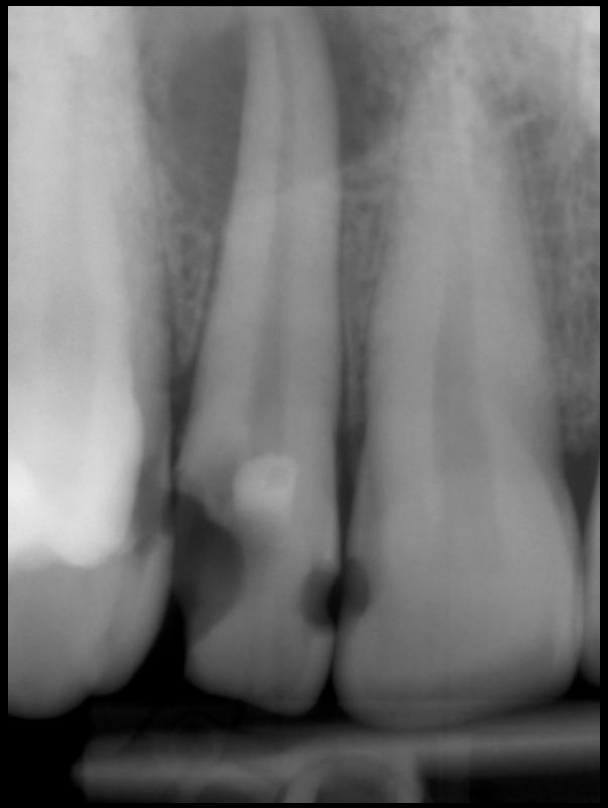
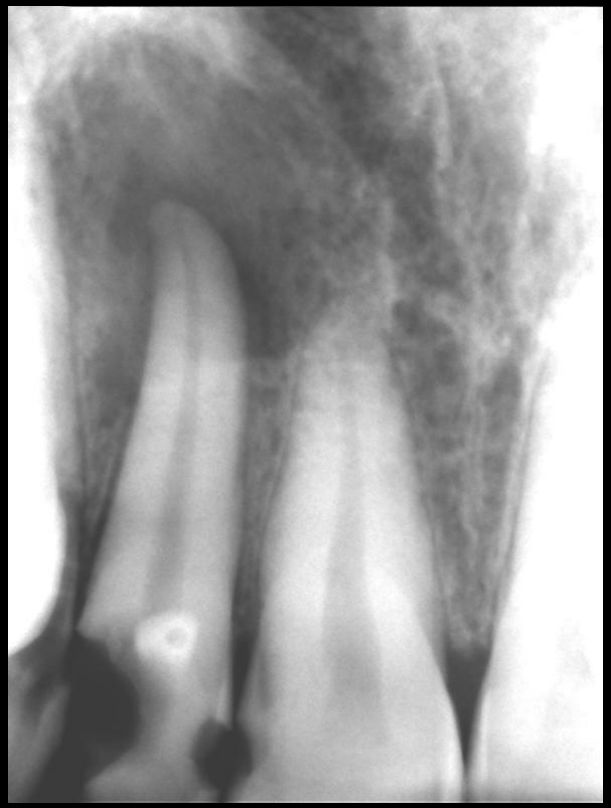
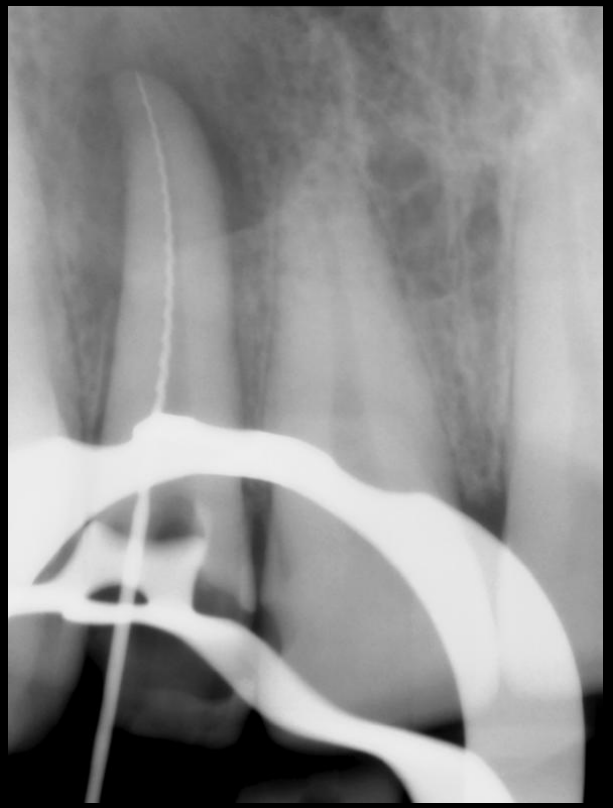
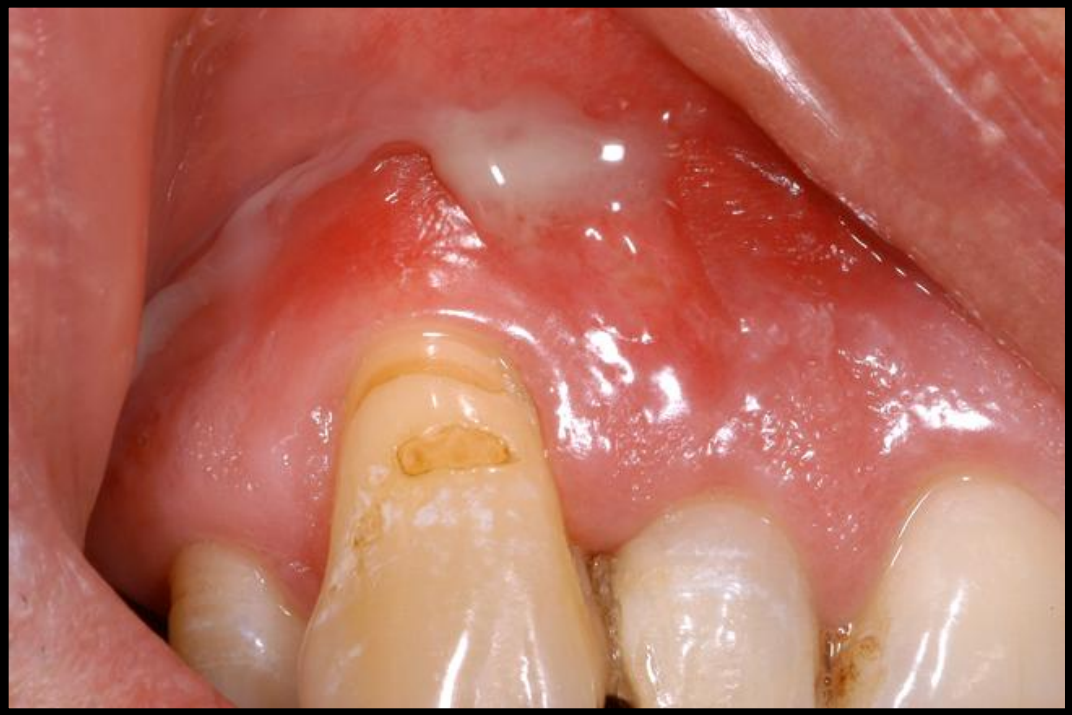
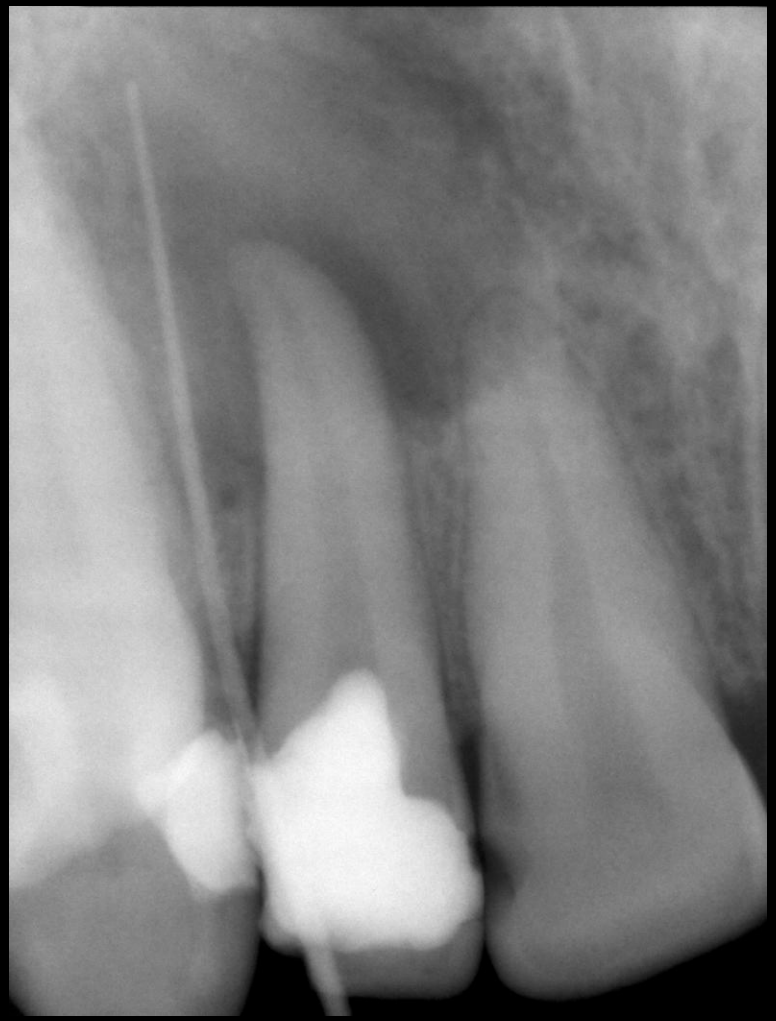


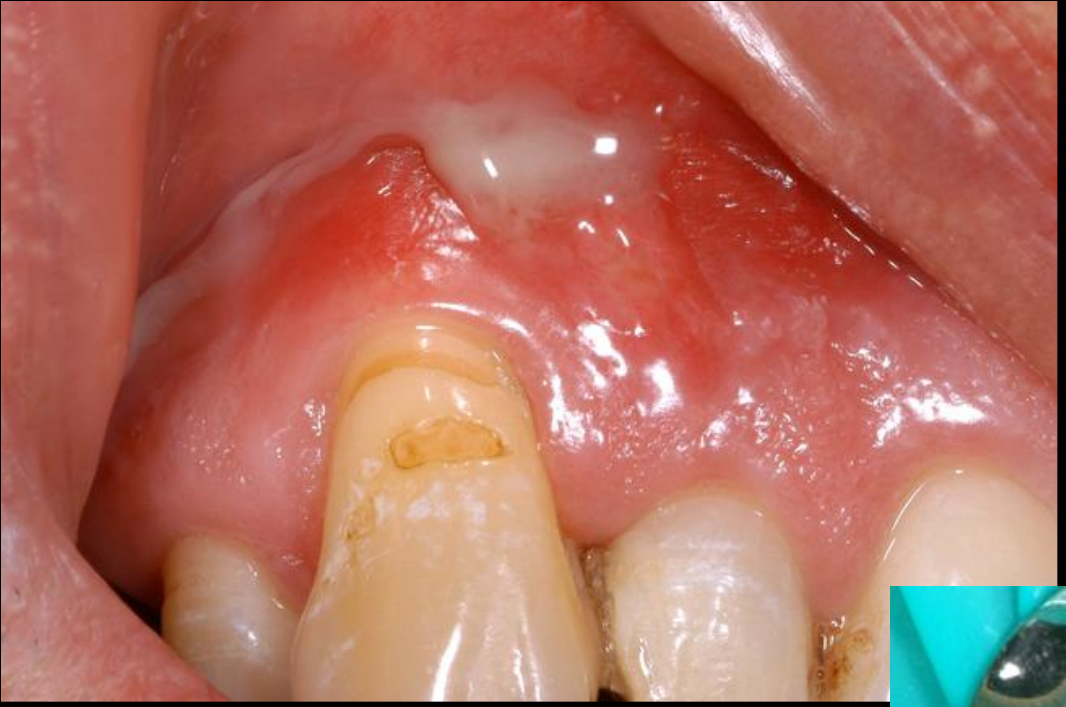


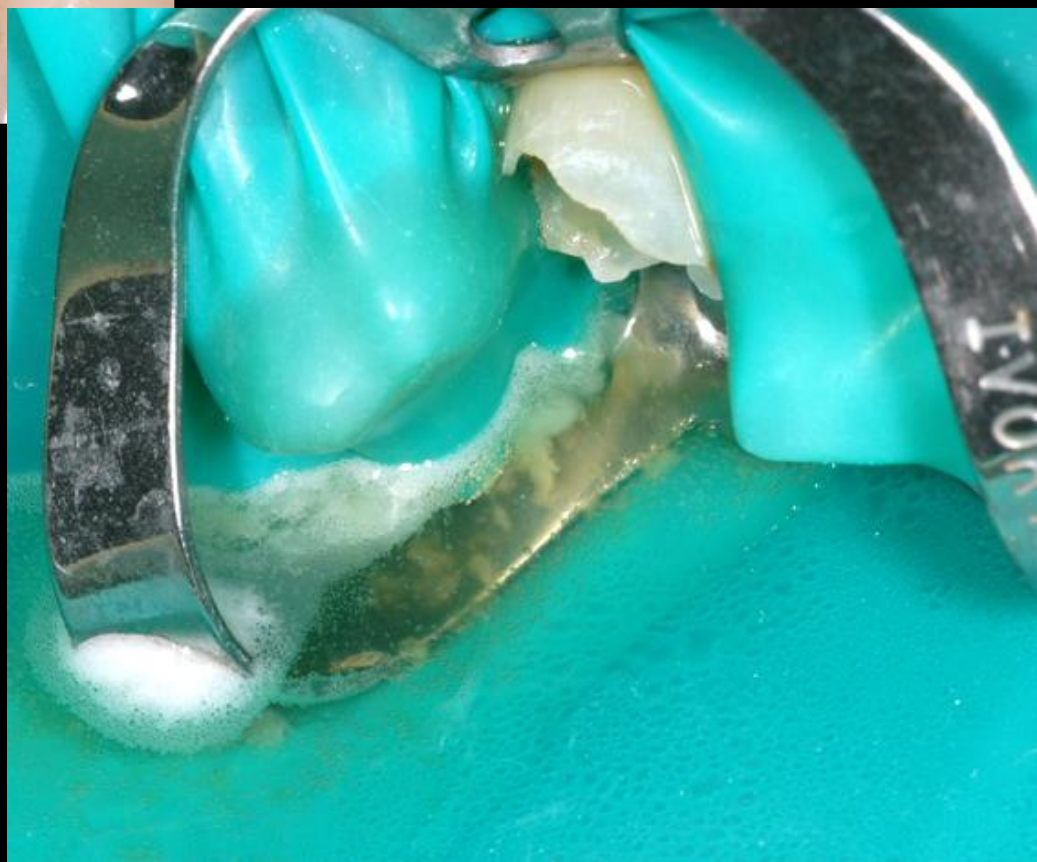


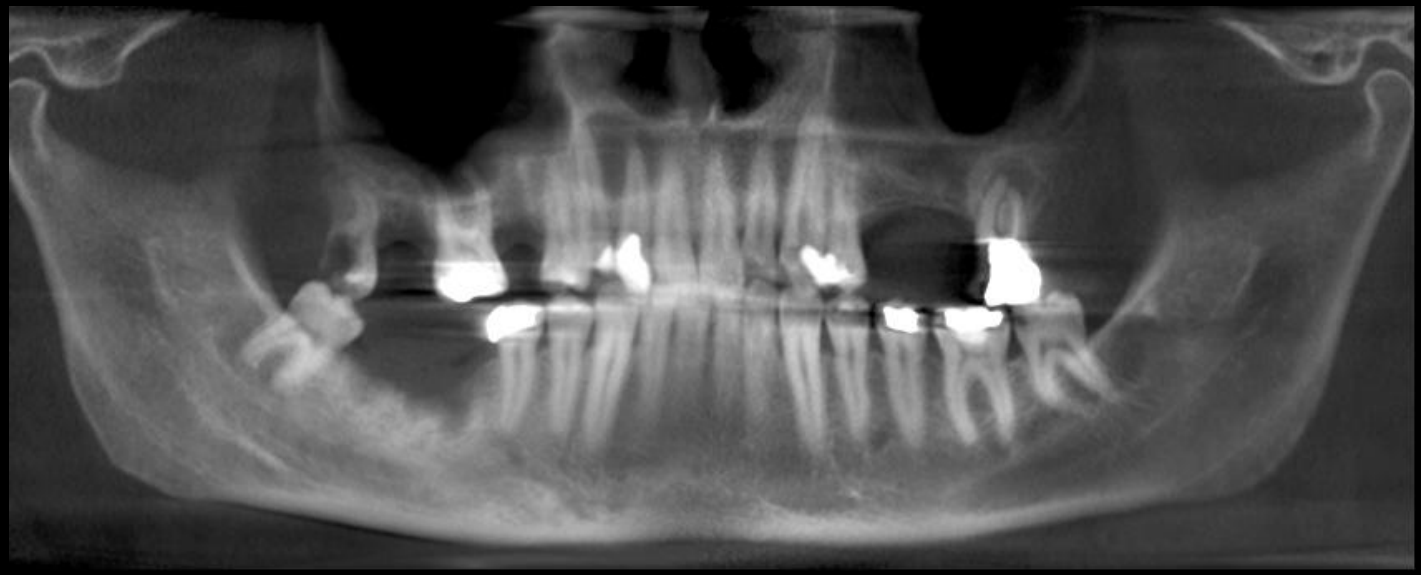


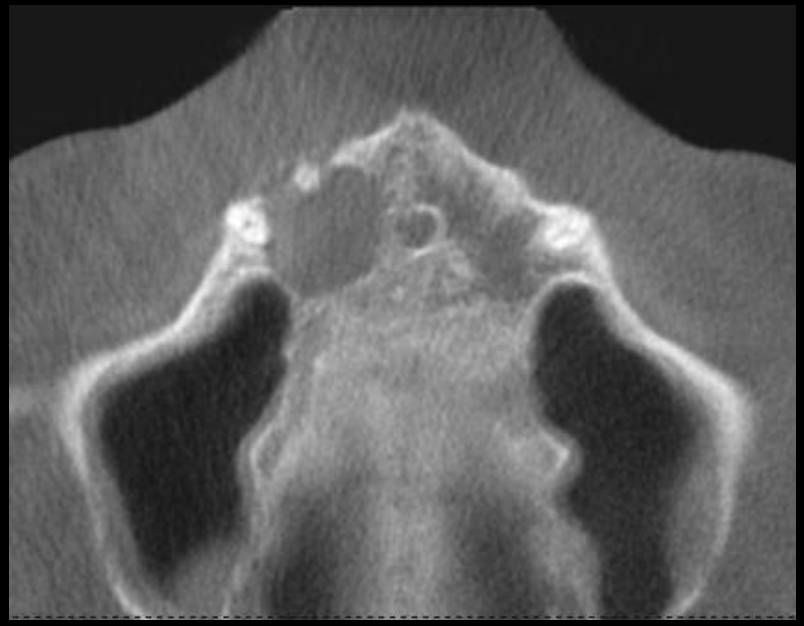
$Ca(OH)_2$ for 106 days

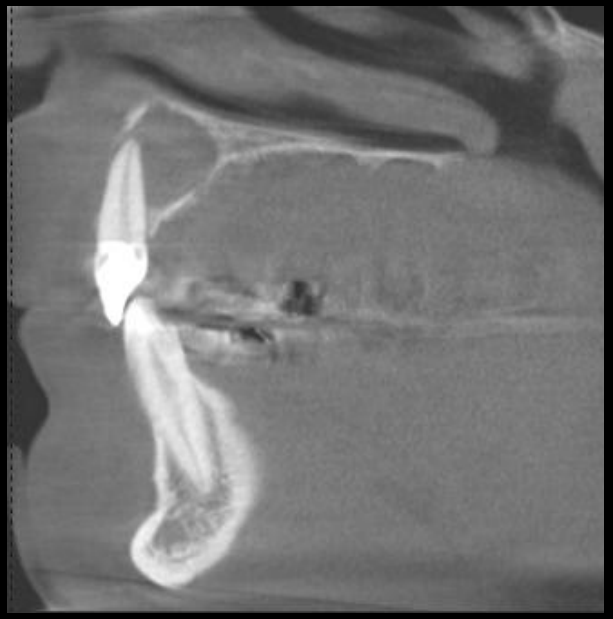


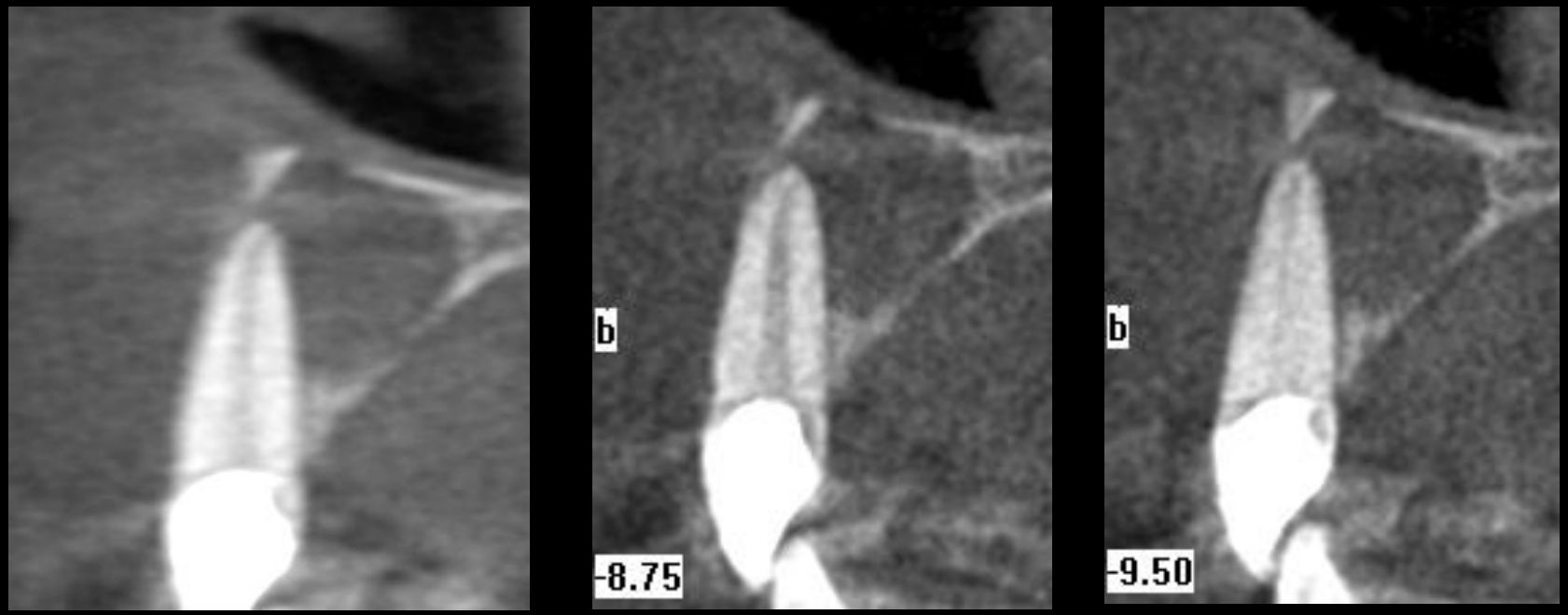
$Ca(OH)_2$ for 106 days







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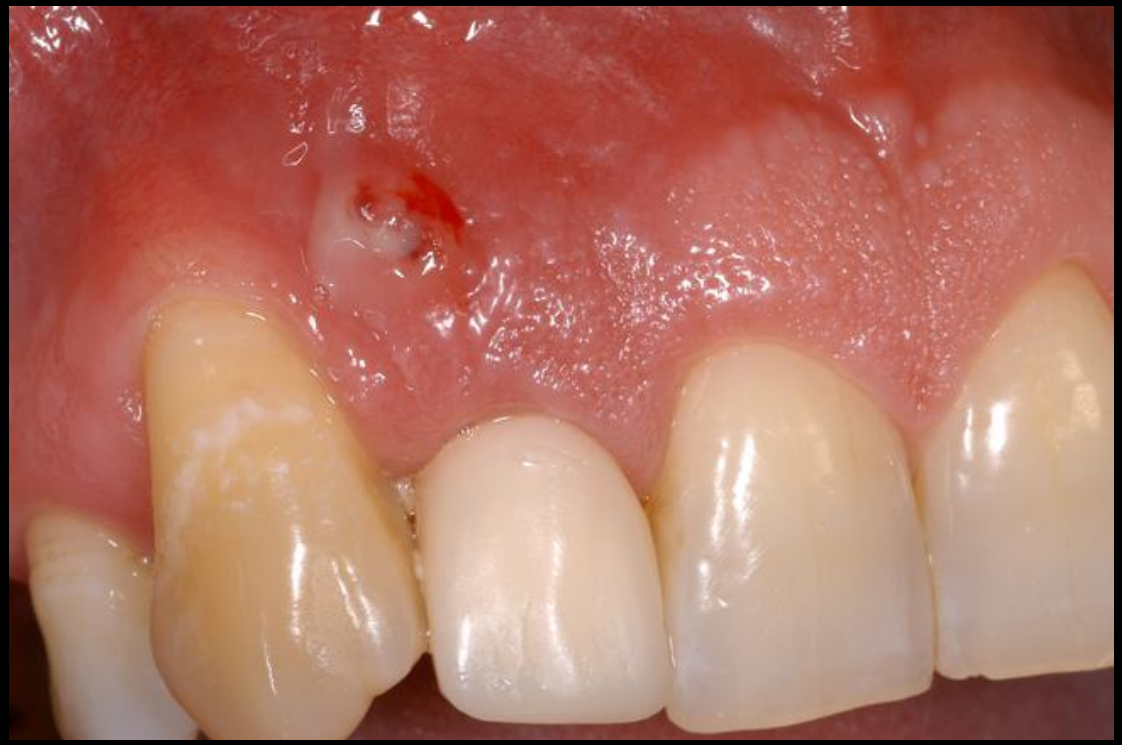
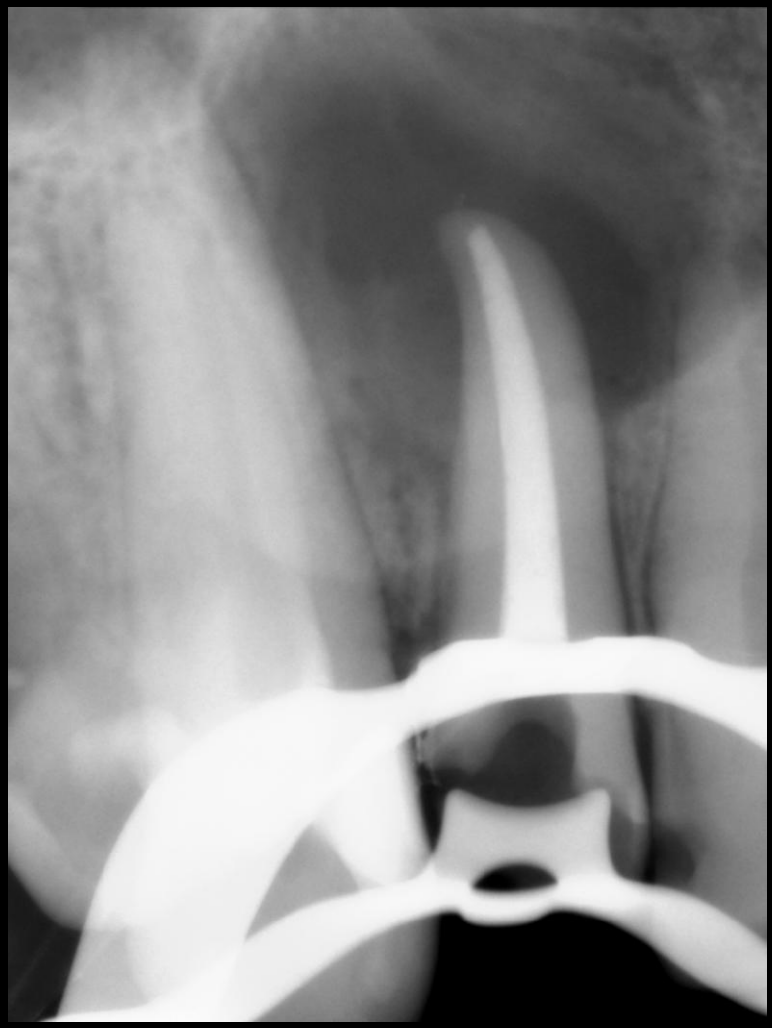
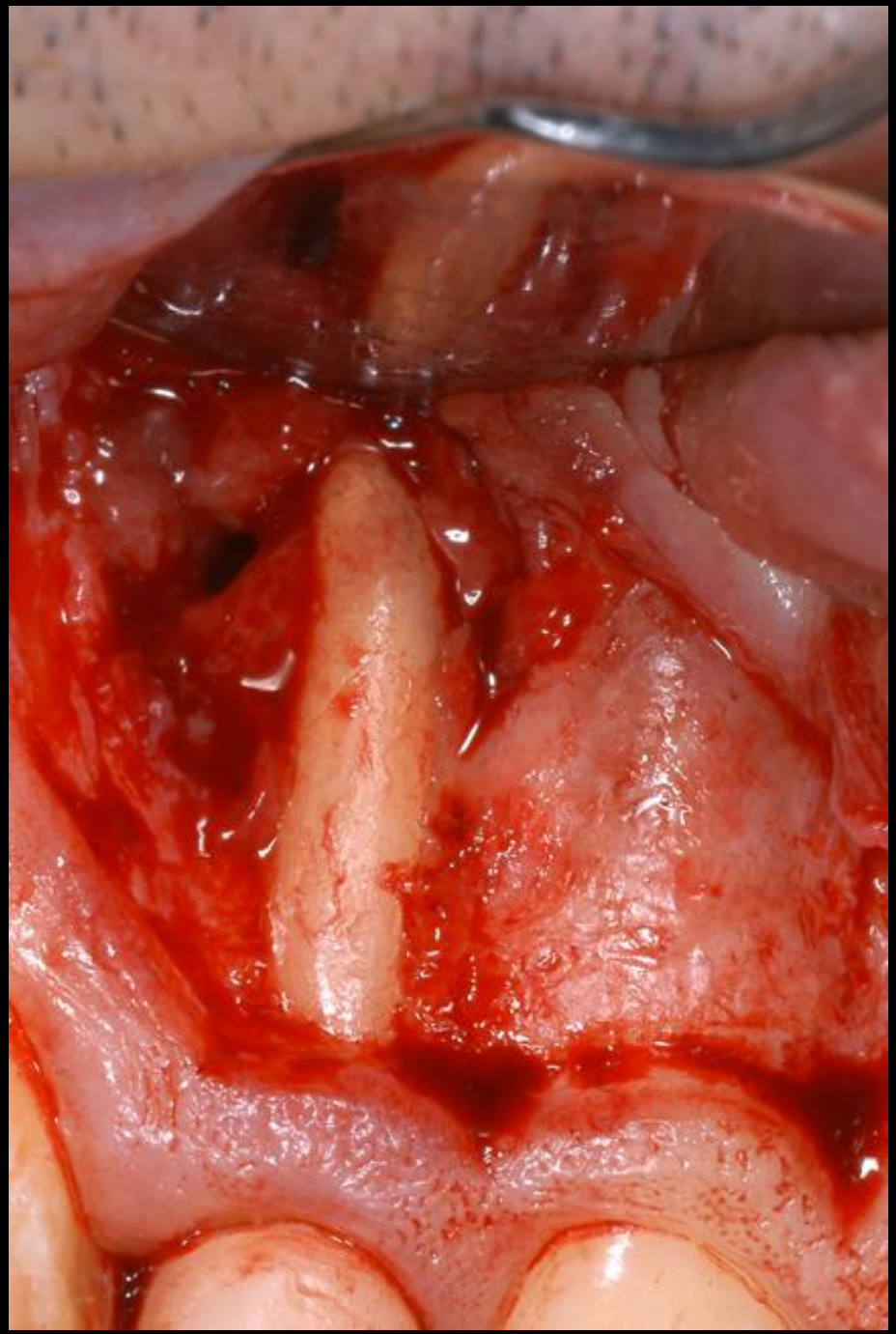
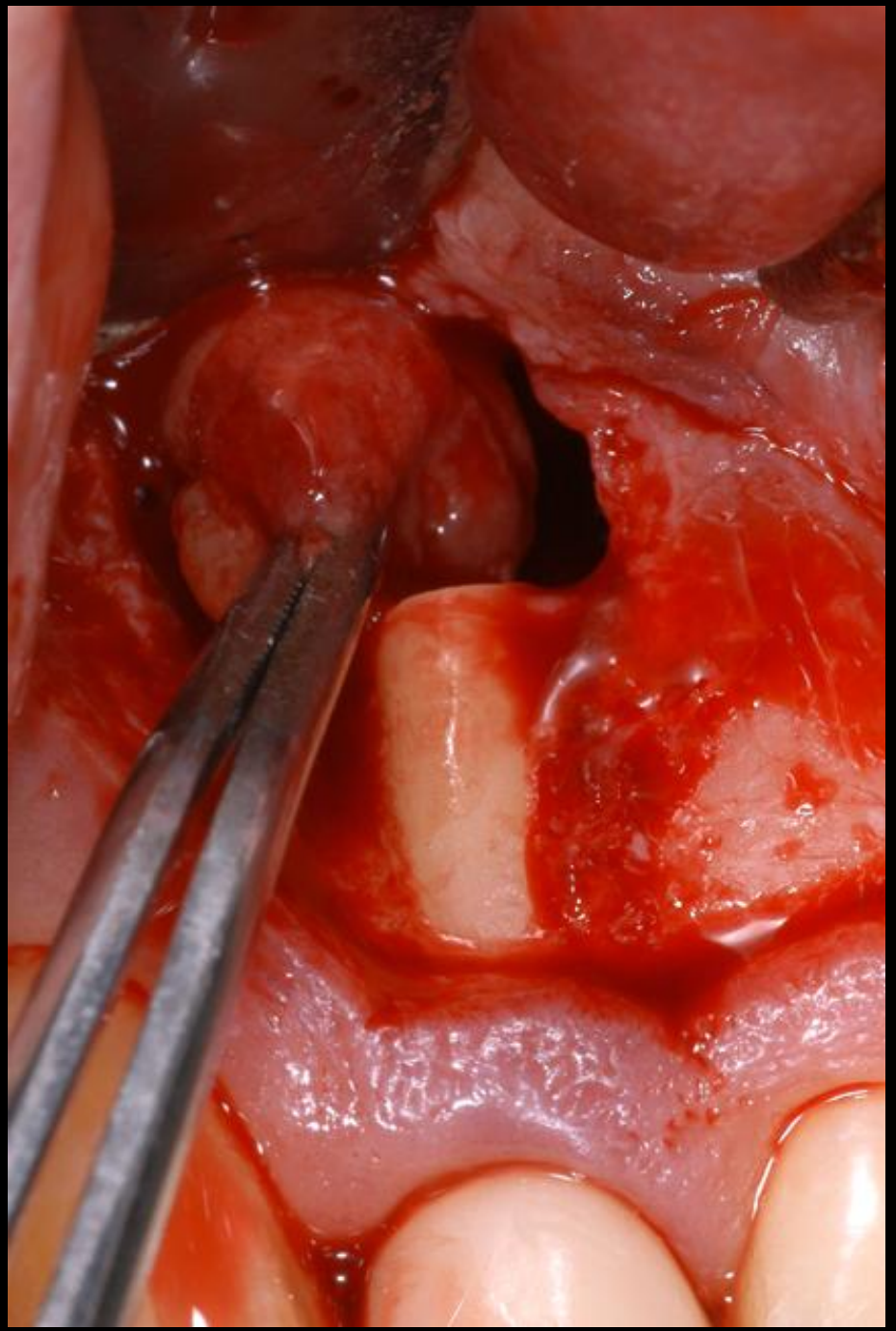
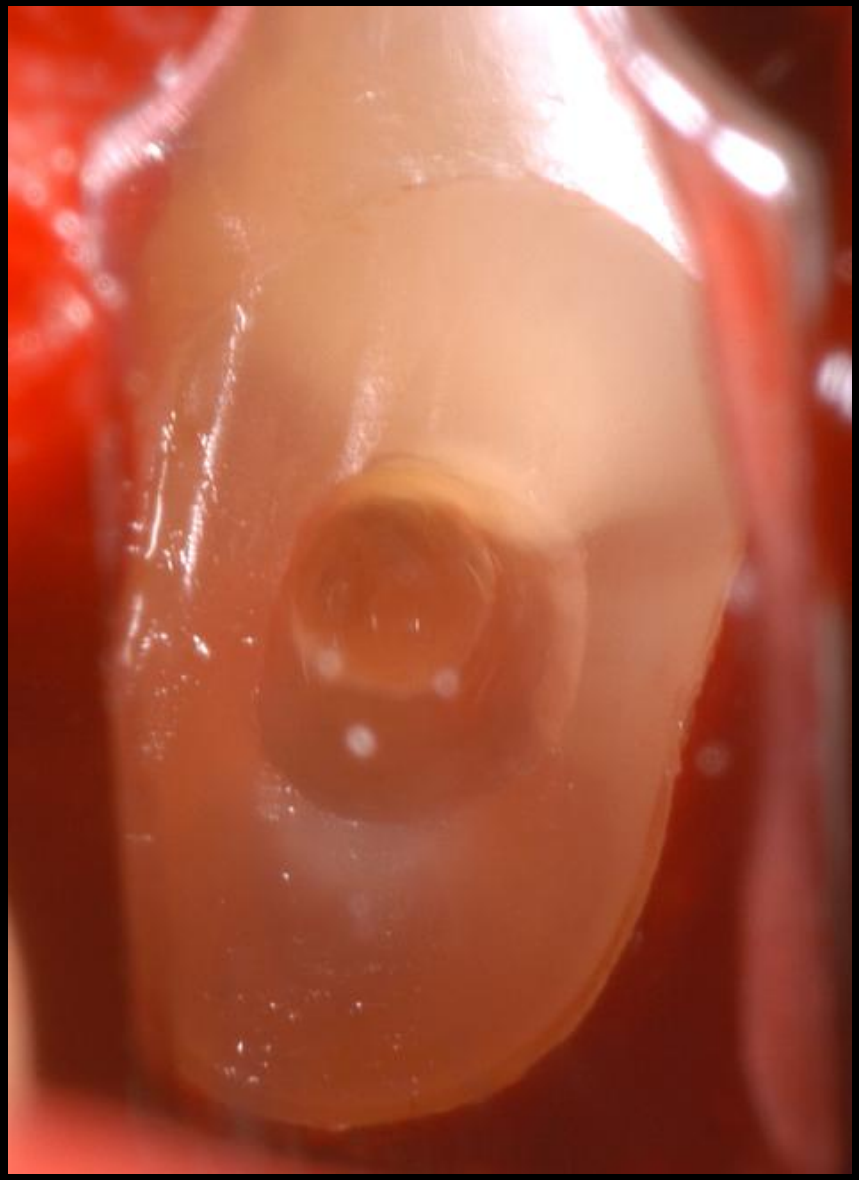
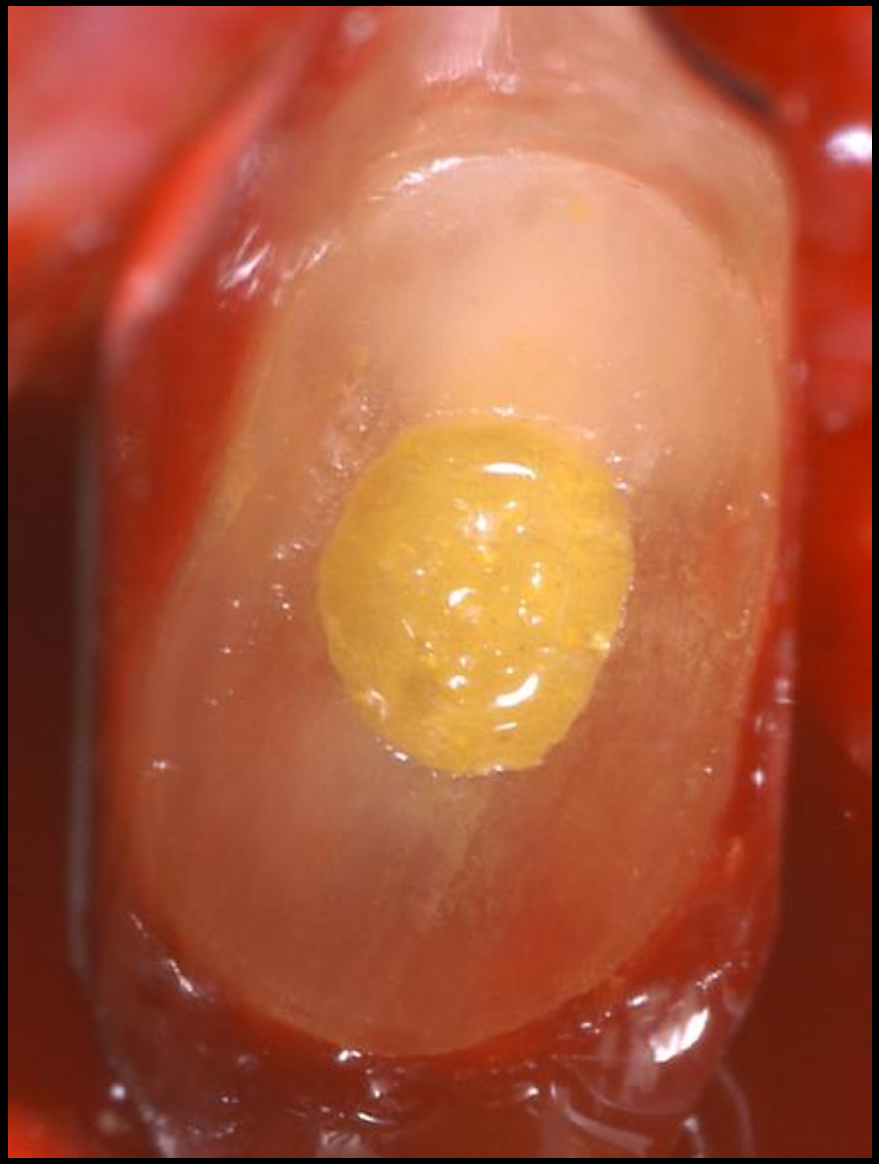


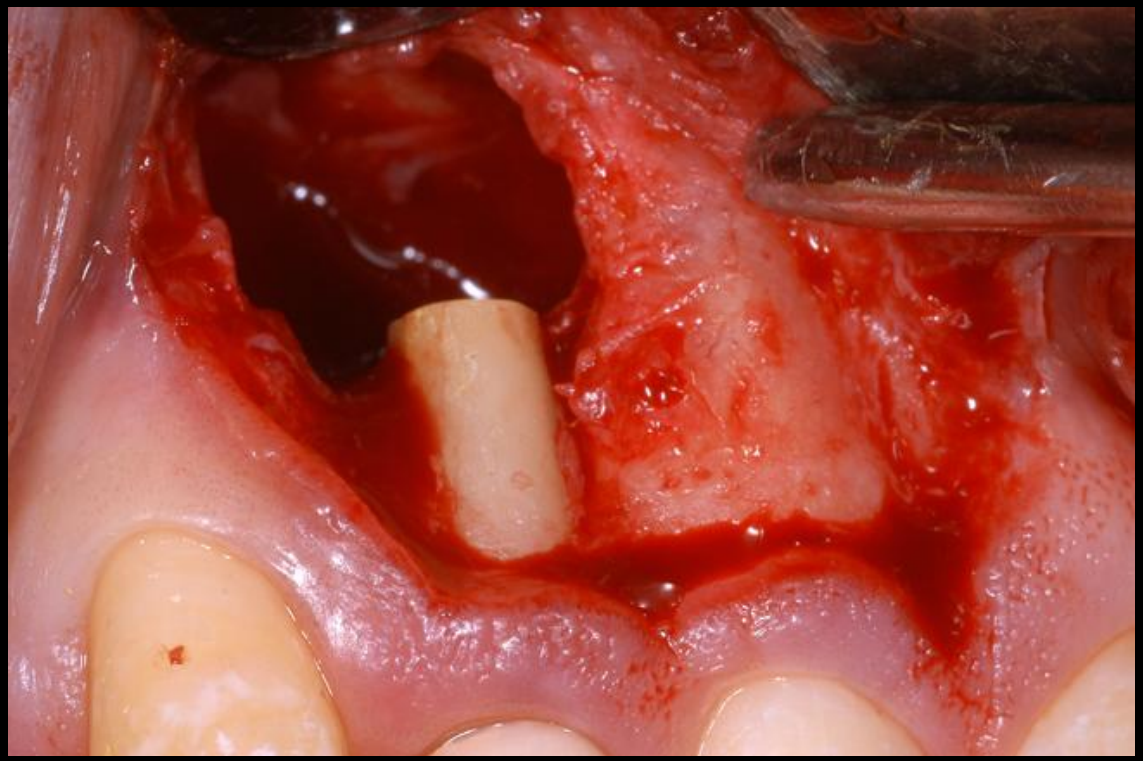


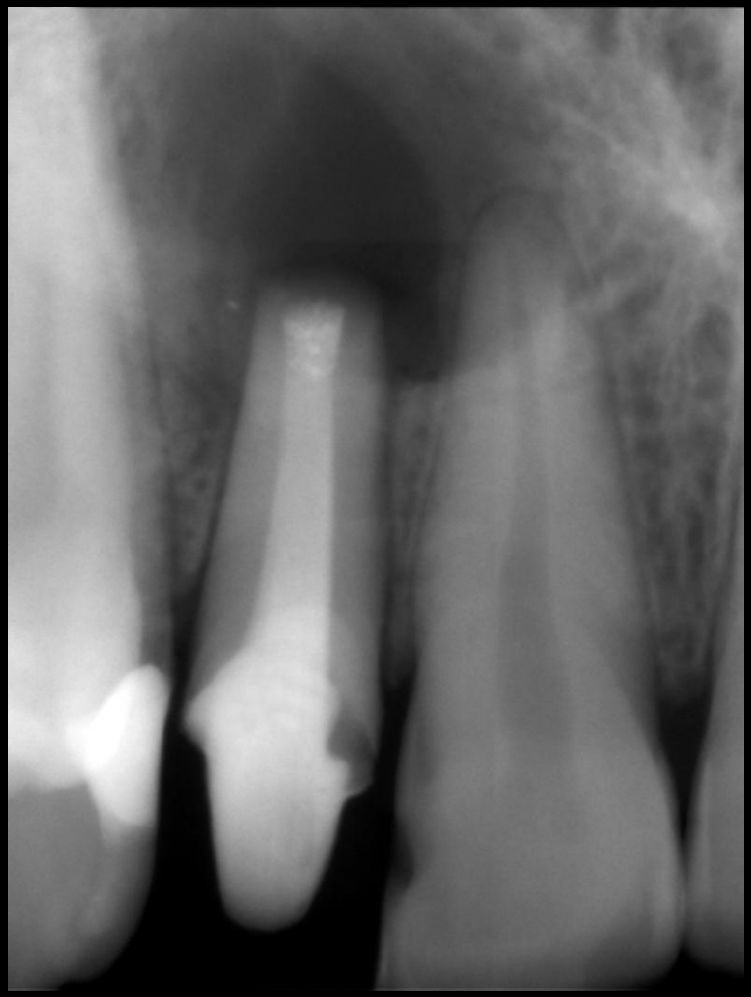
$Ca(OH)_2$ for 106 days

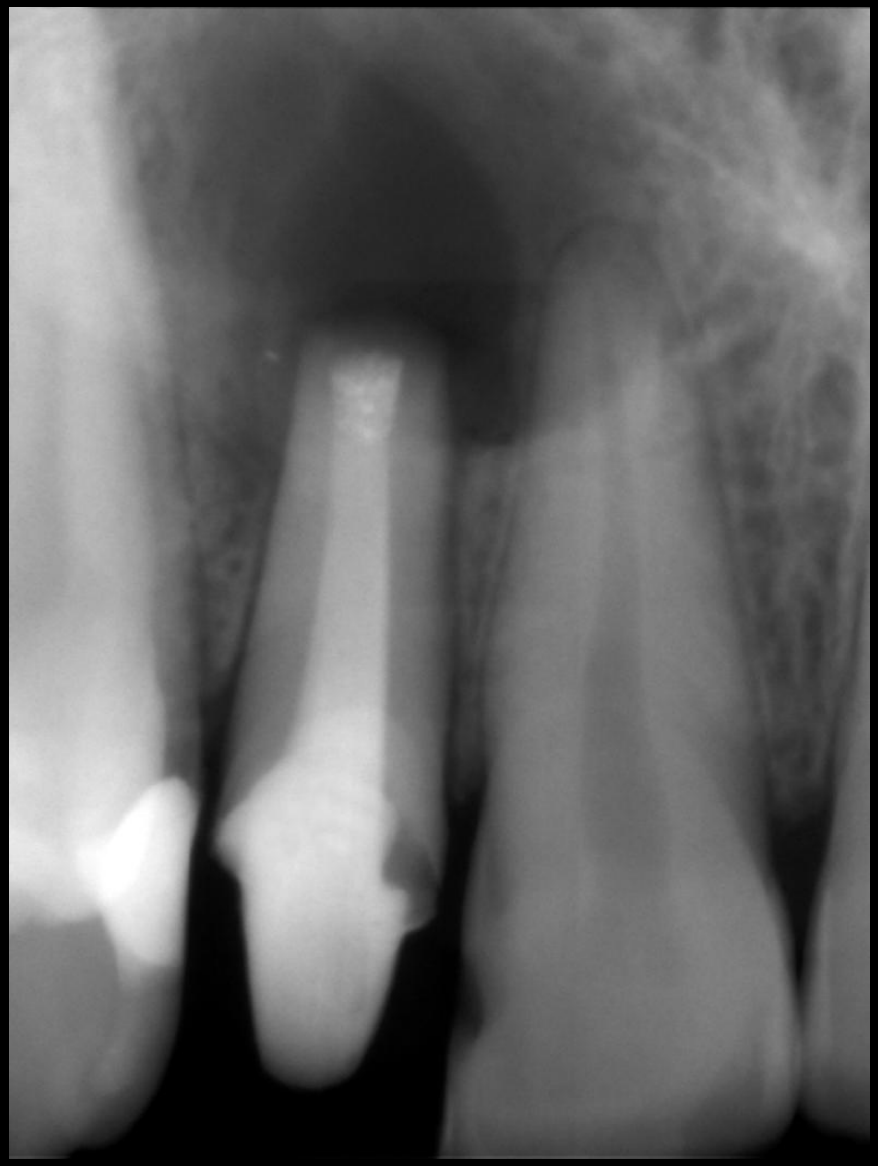



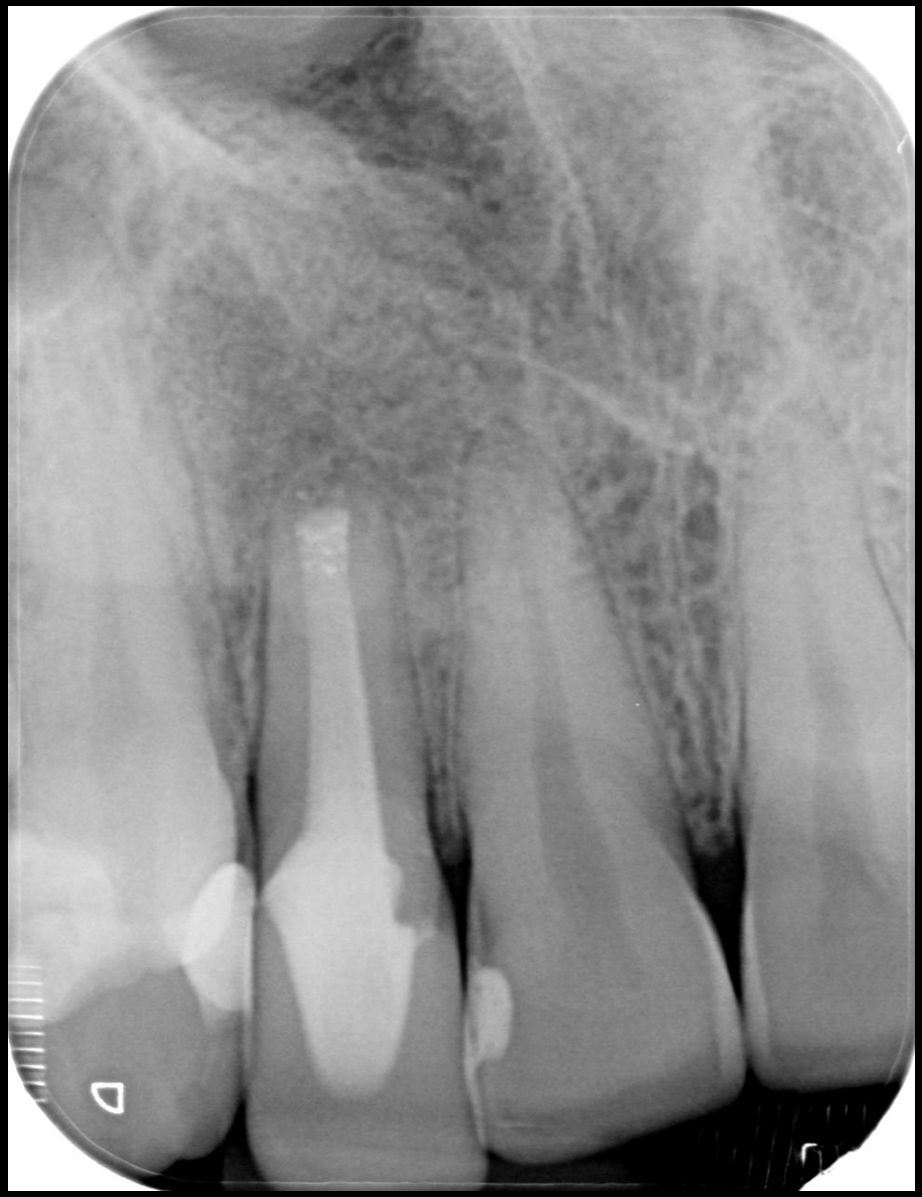












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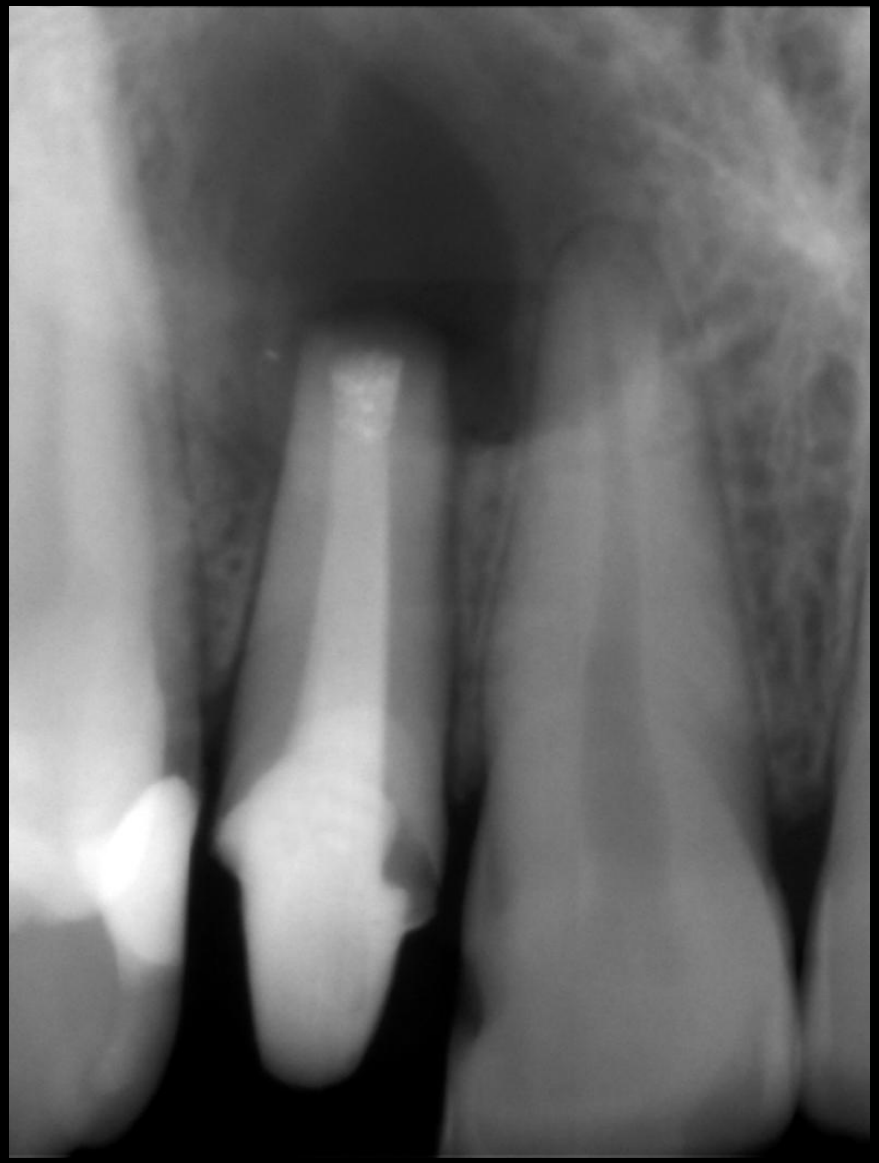

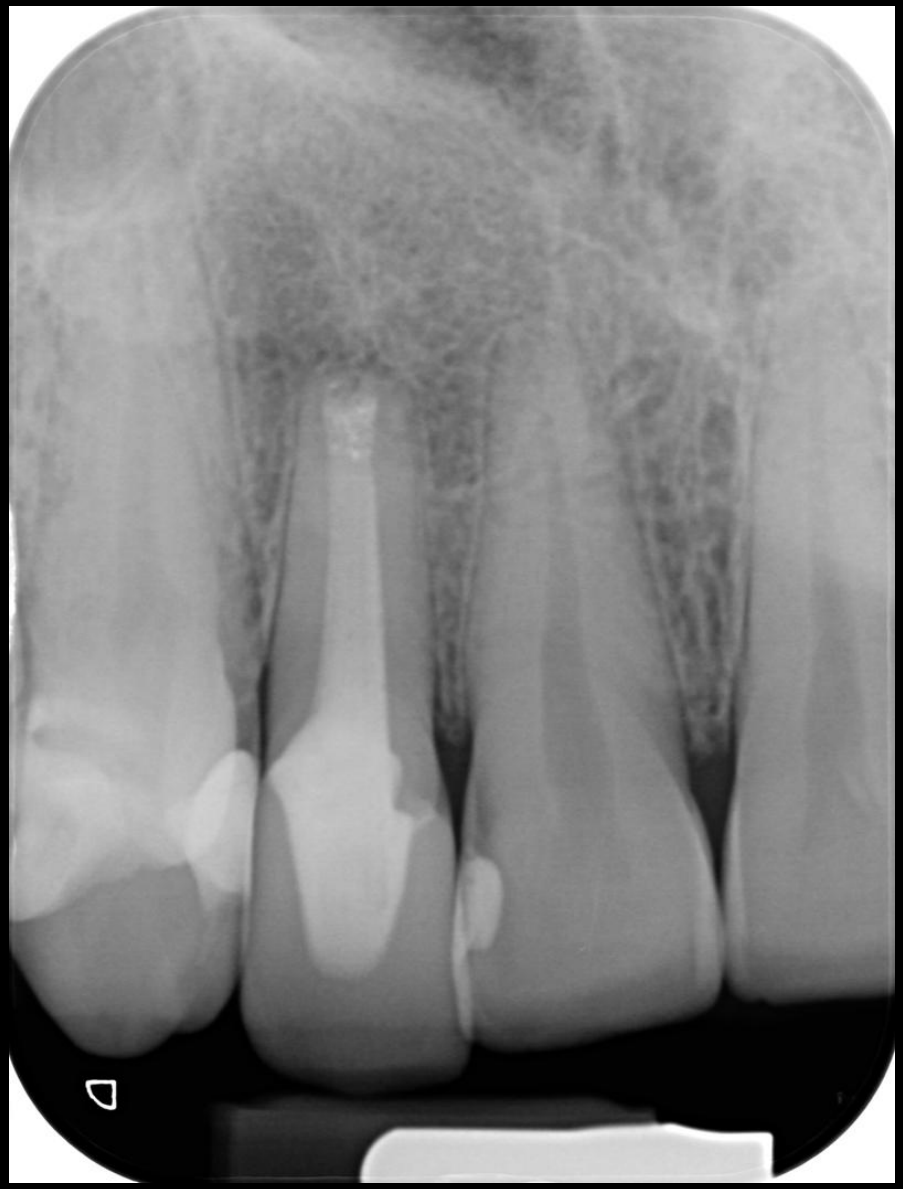

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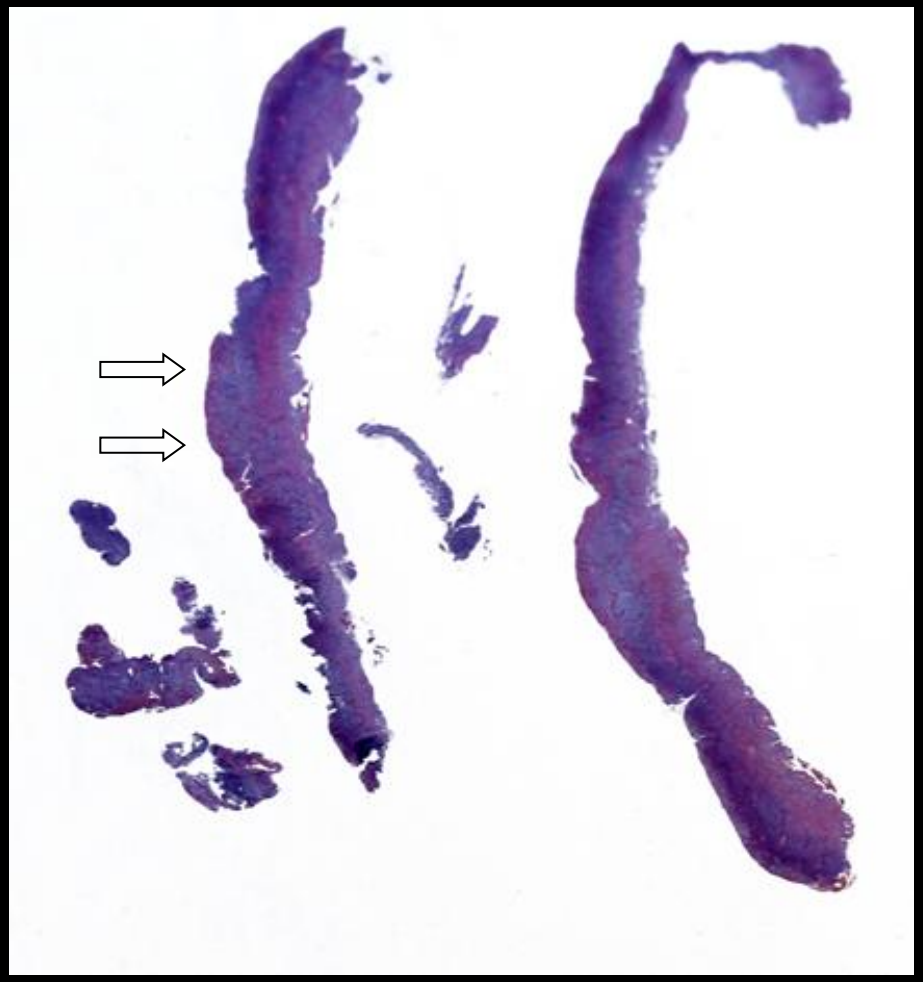

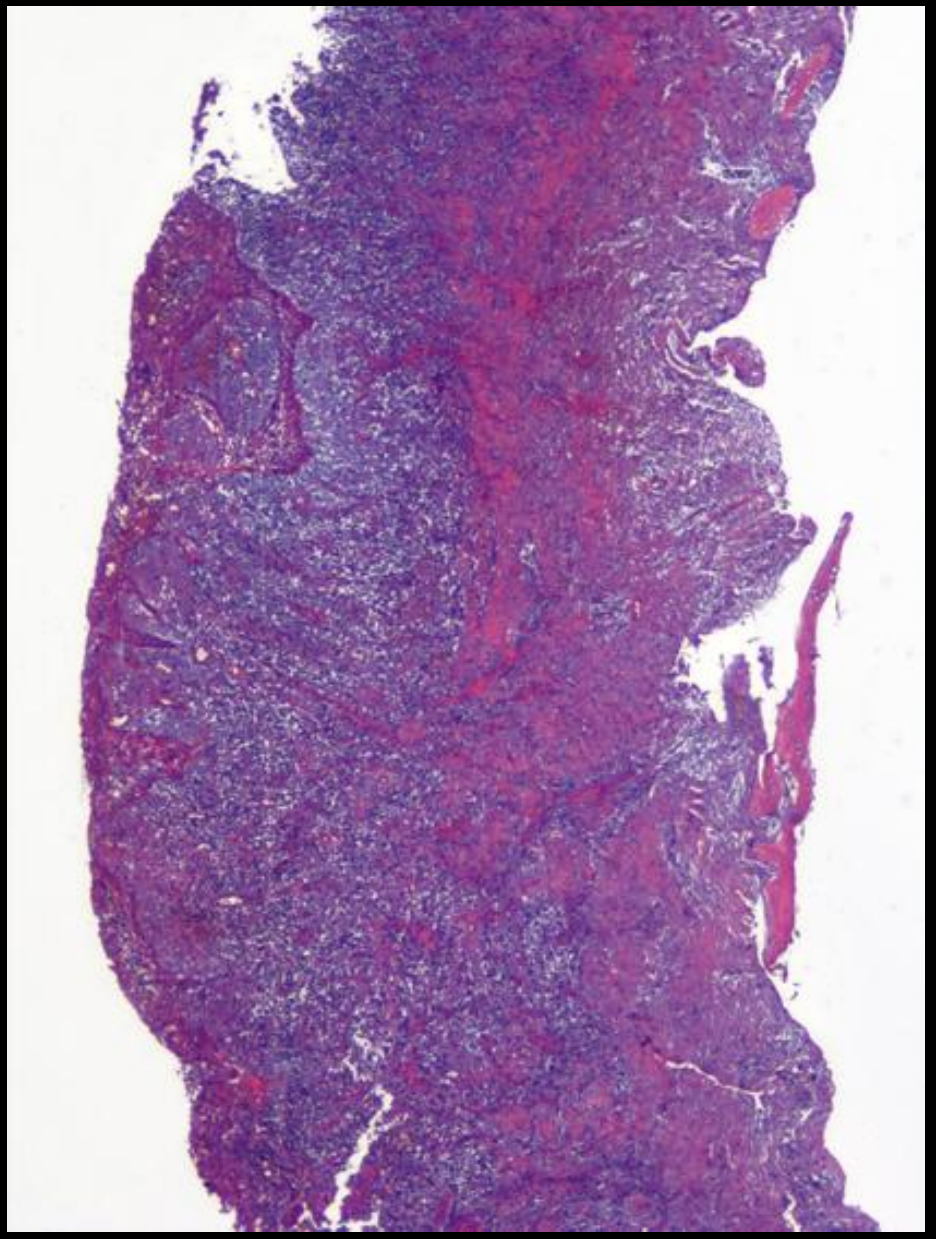

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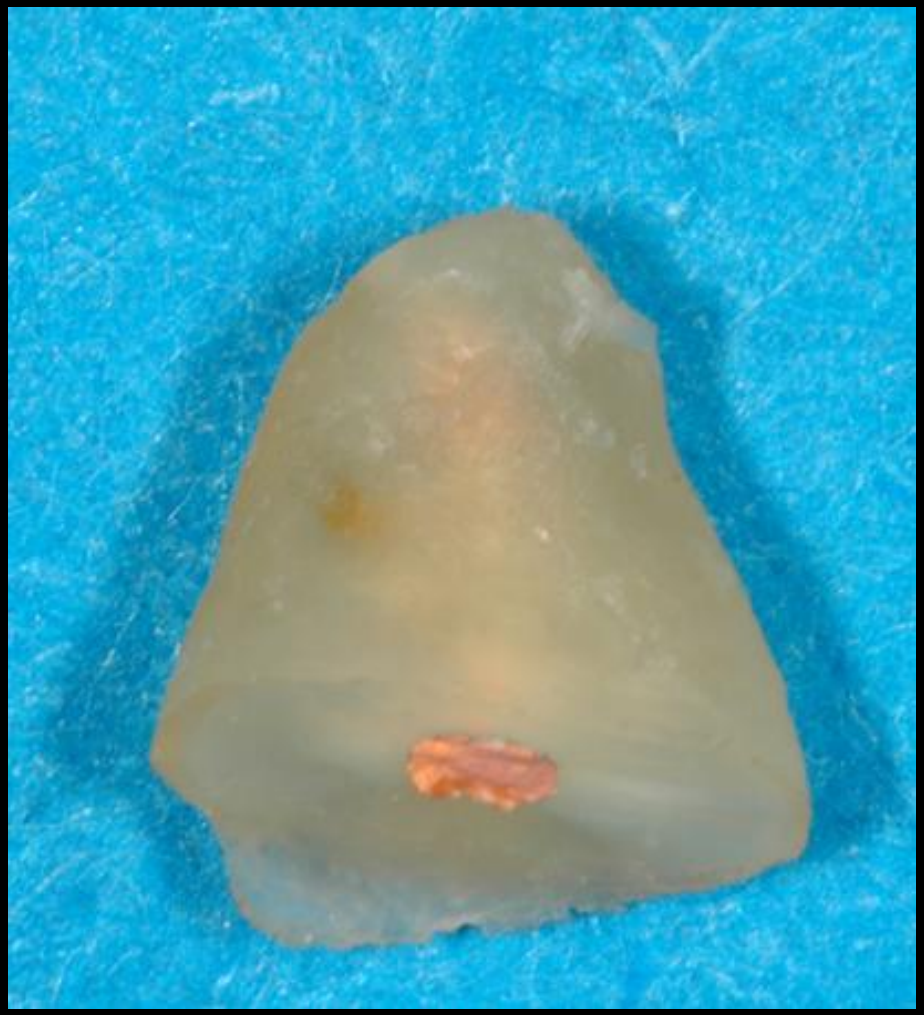

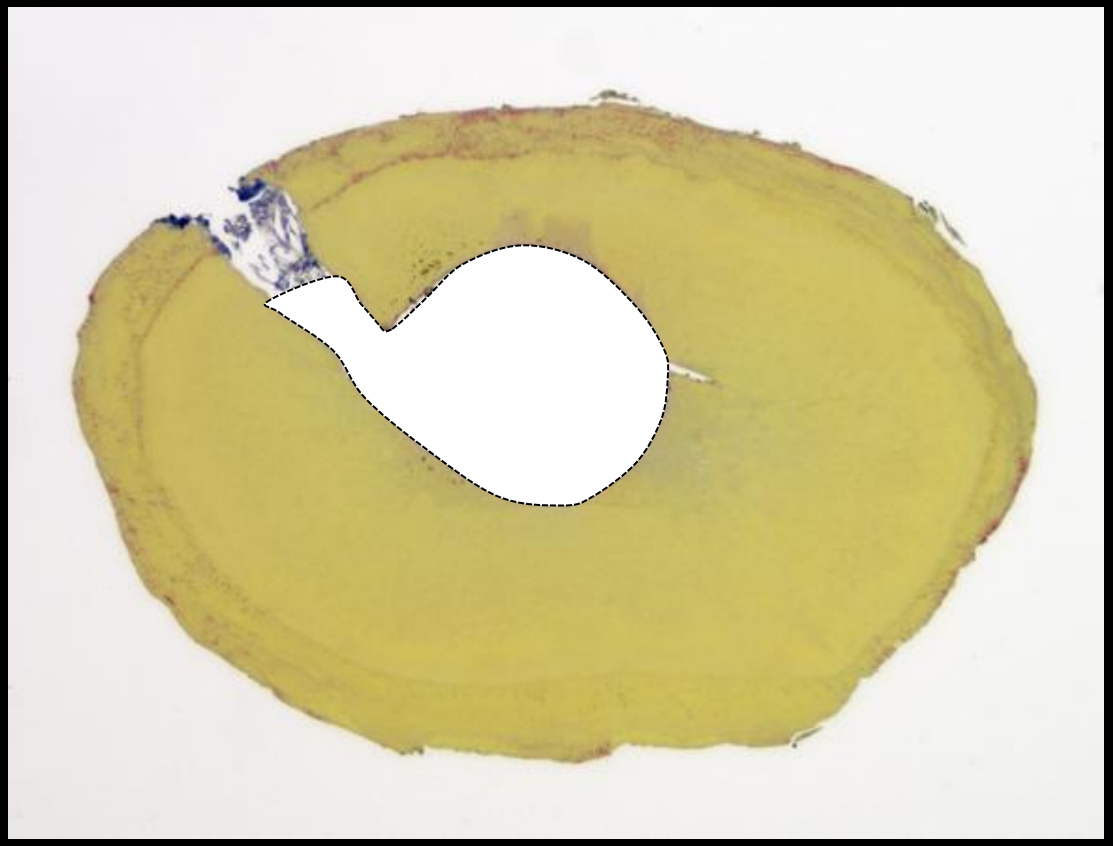

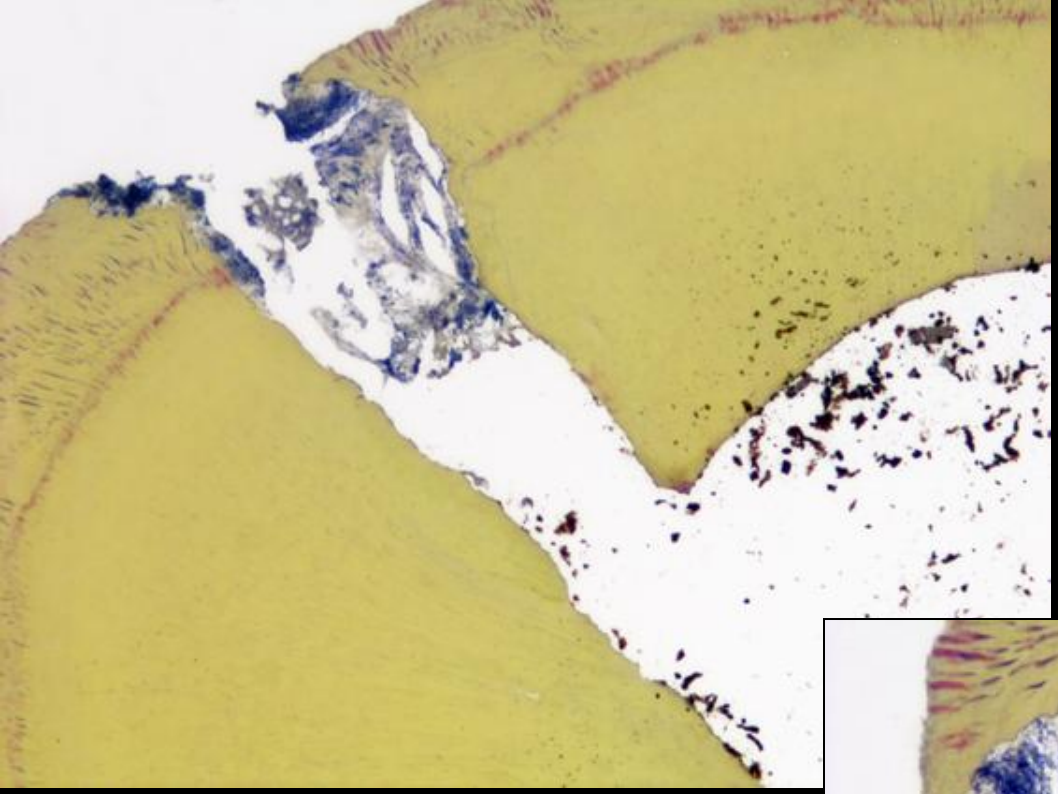

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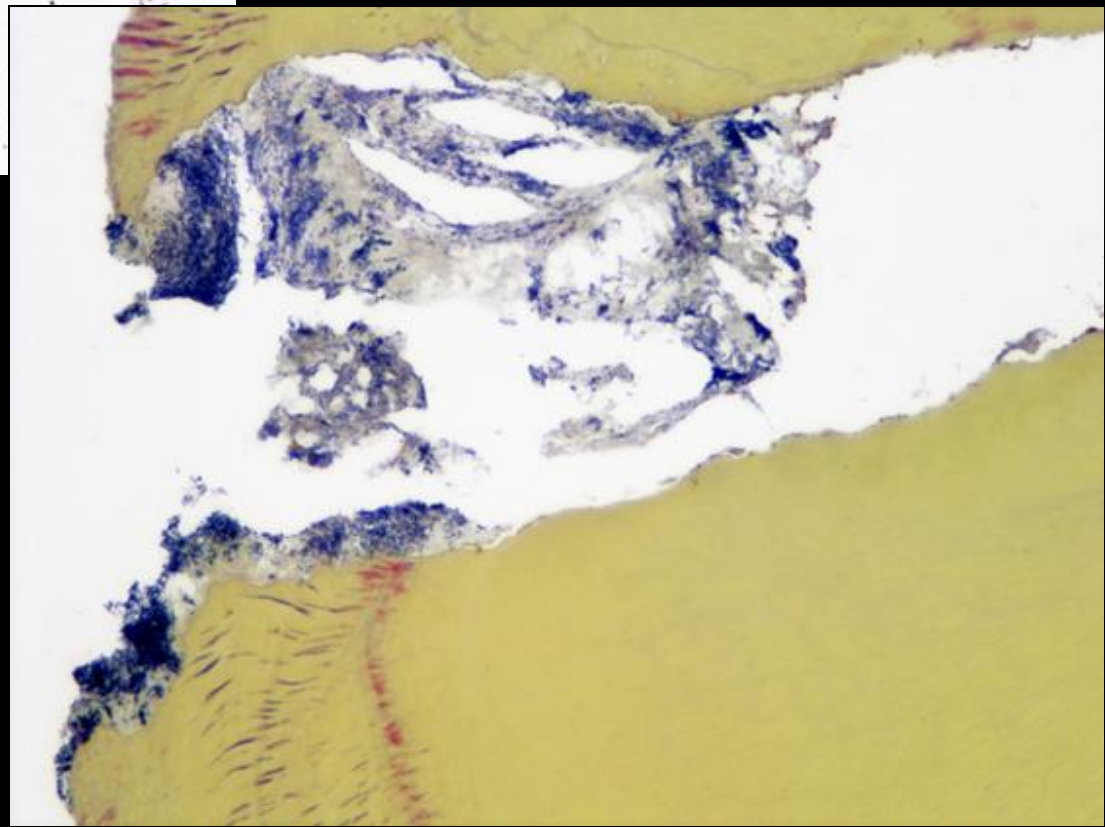

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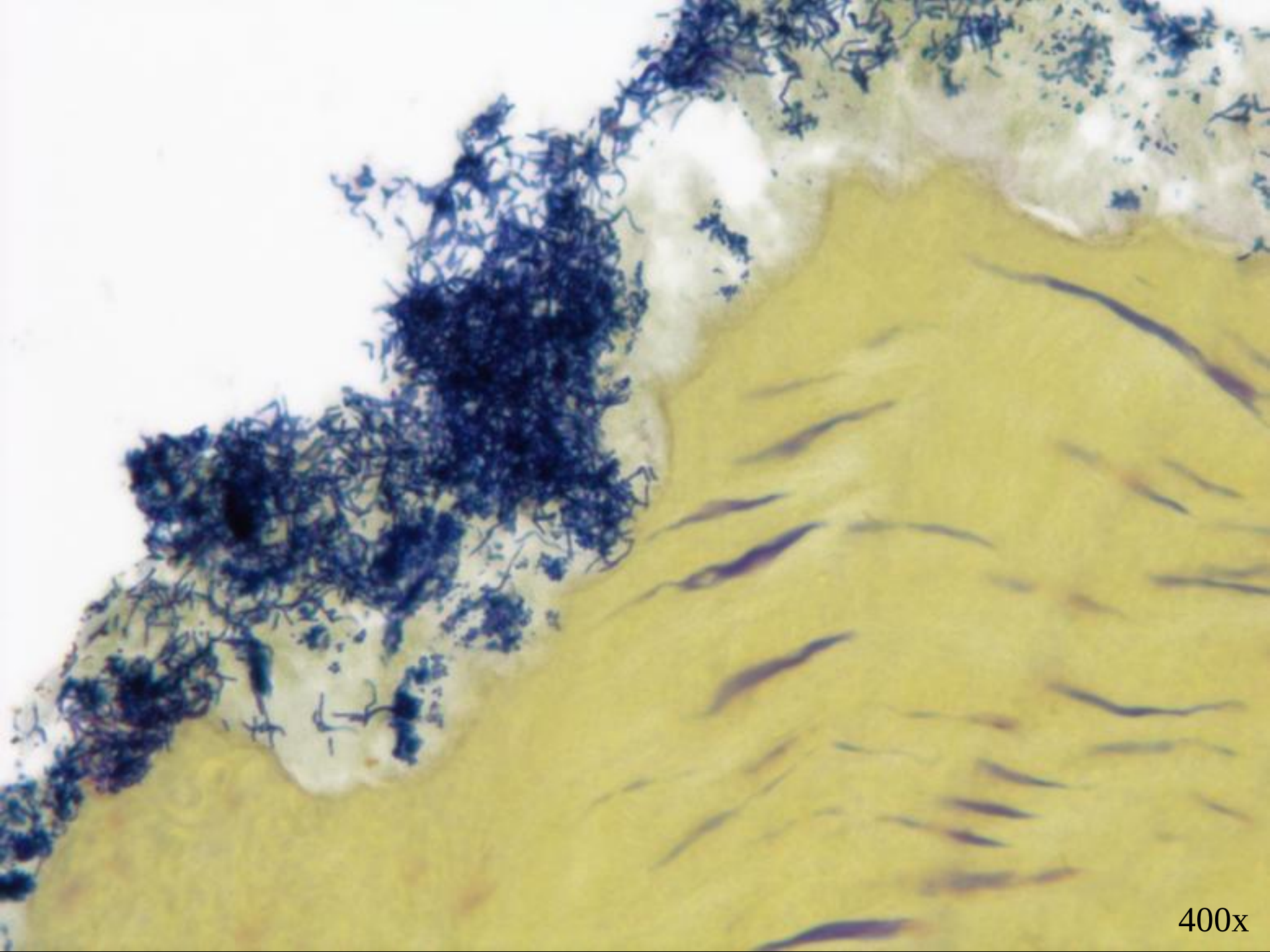
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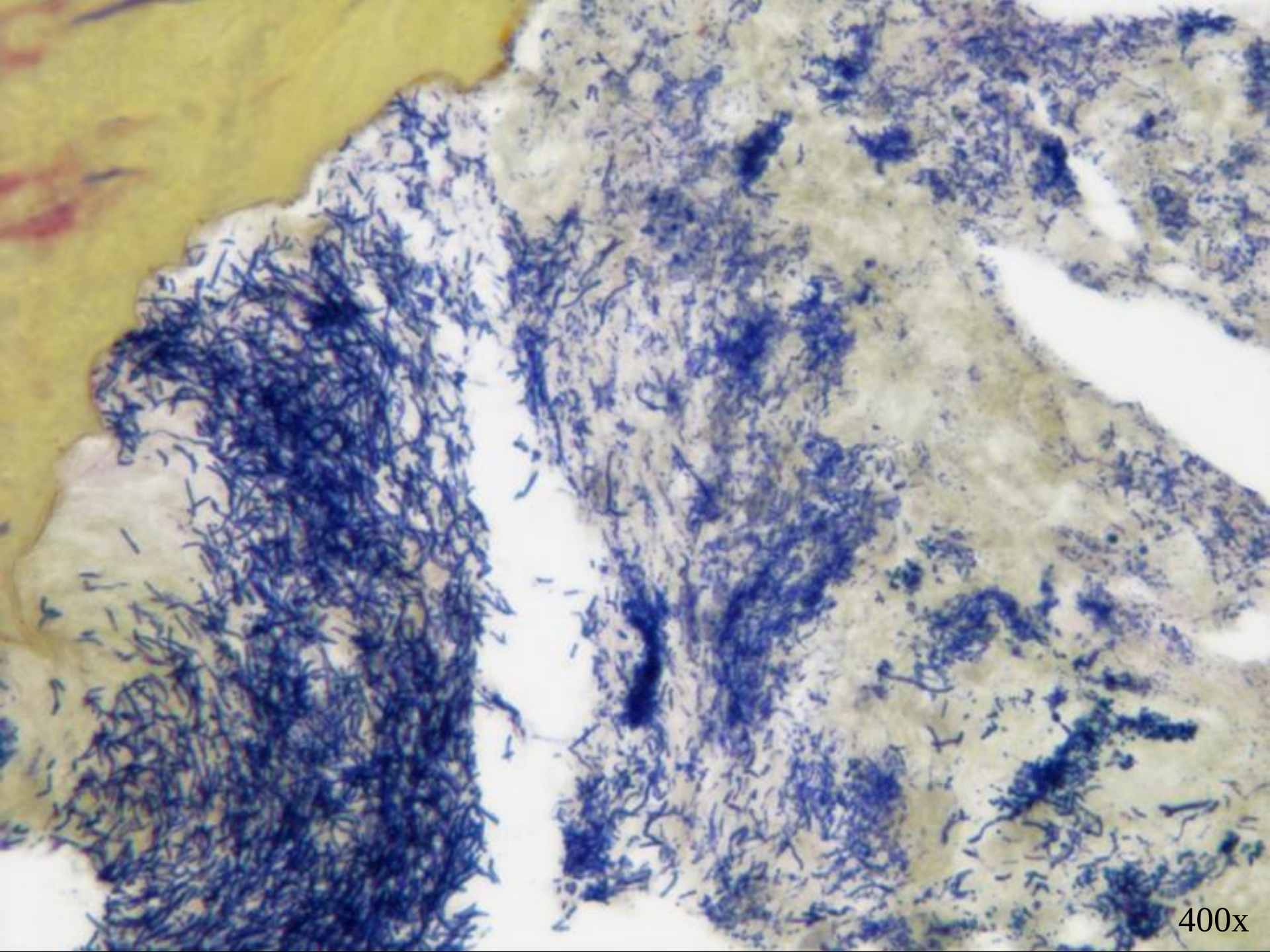
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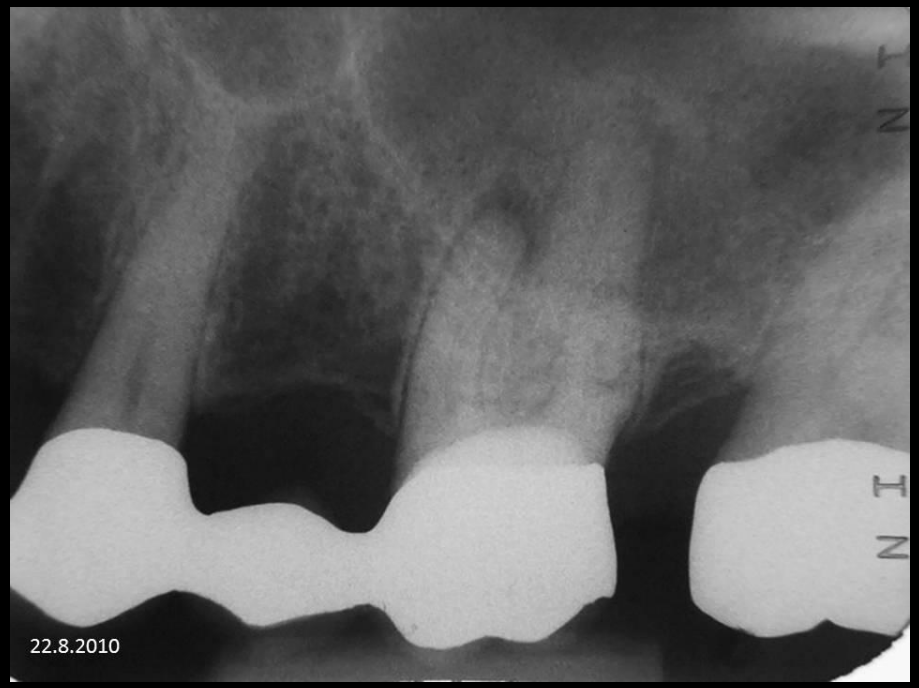
Ricucci D, Loghin S, Siqueira JF Jr.

Exuberant infection in a lateral canal as the cause of short-term endodontic treatment failure: report of a case.

Journal of Endodontics 2013; 39:712-18.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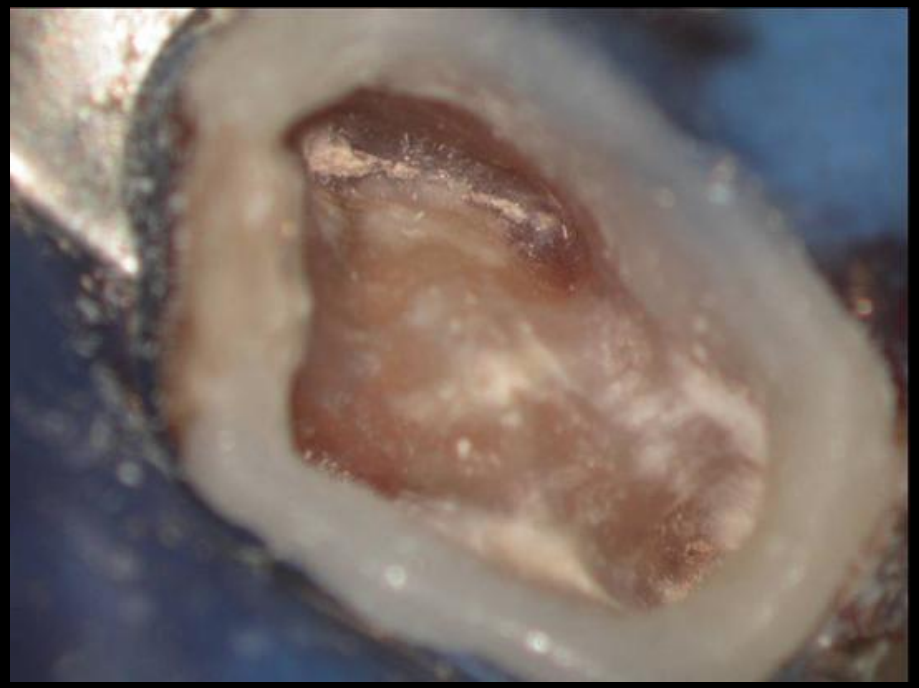
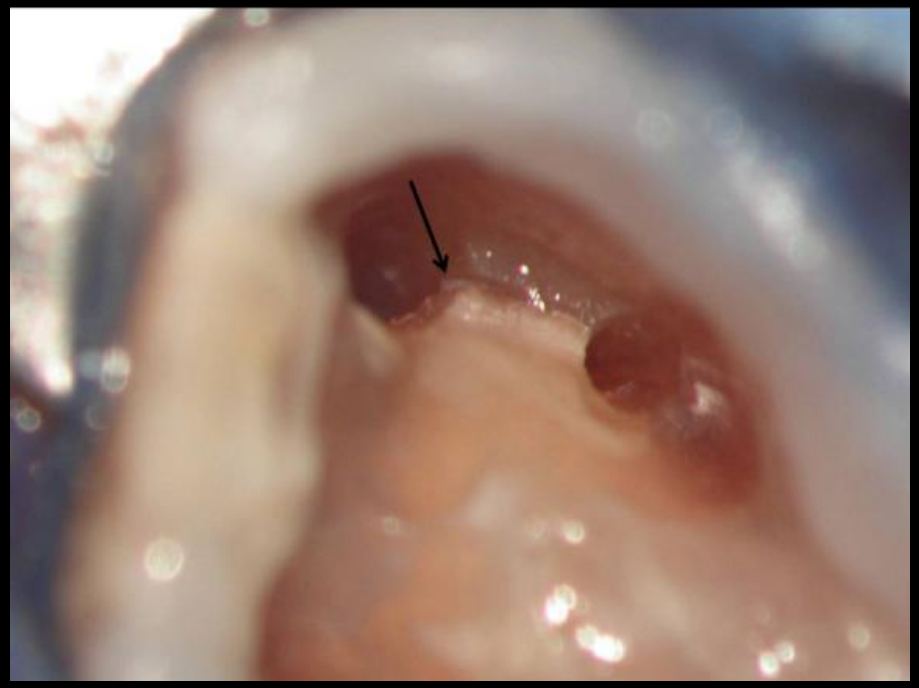
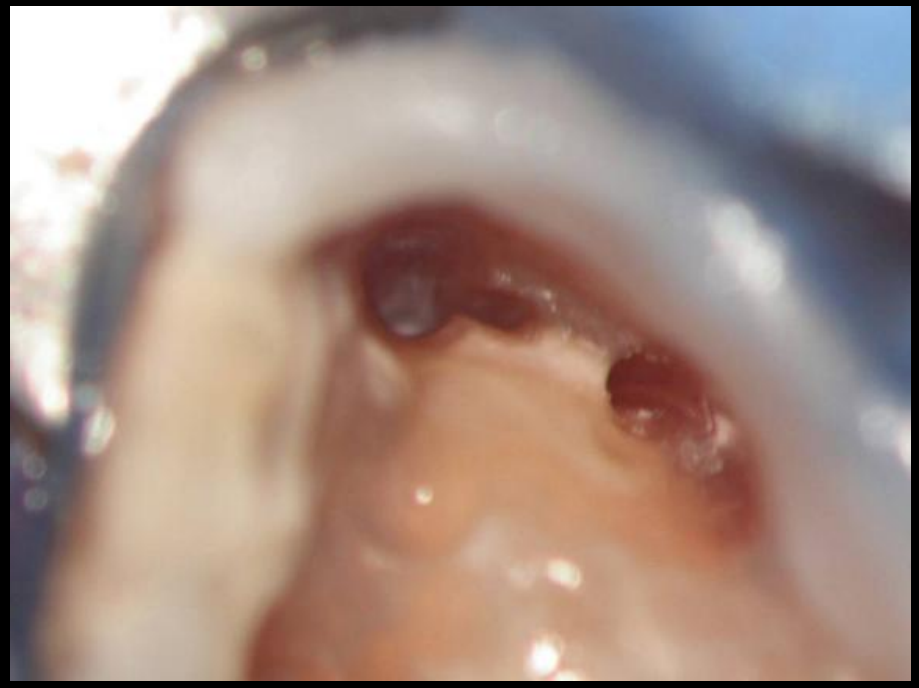
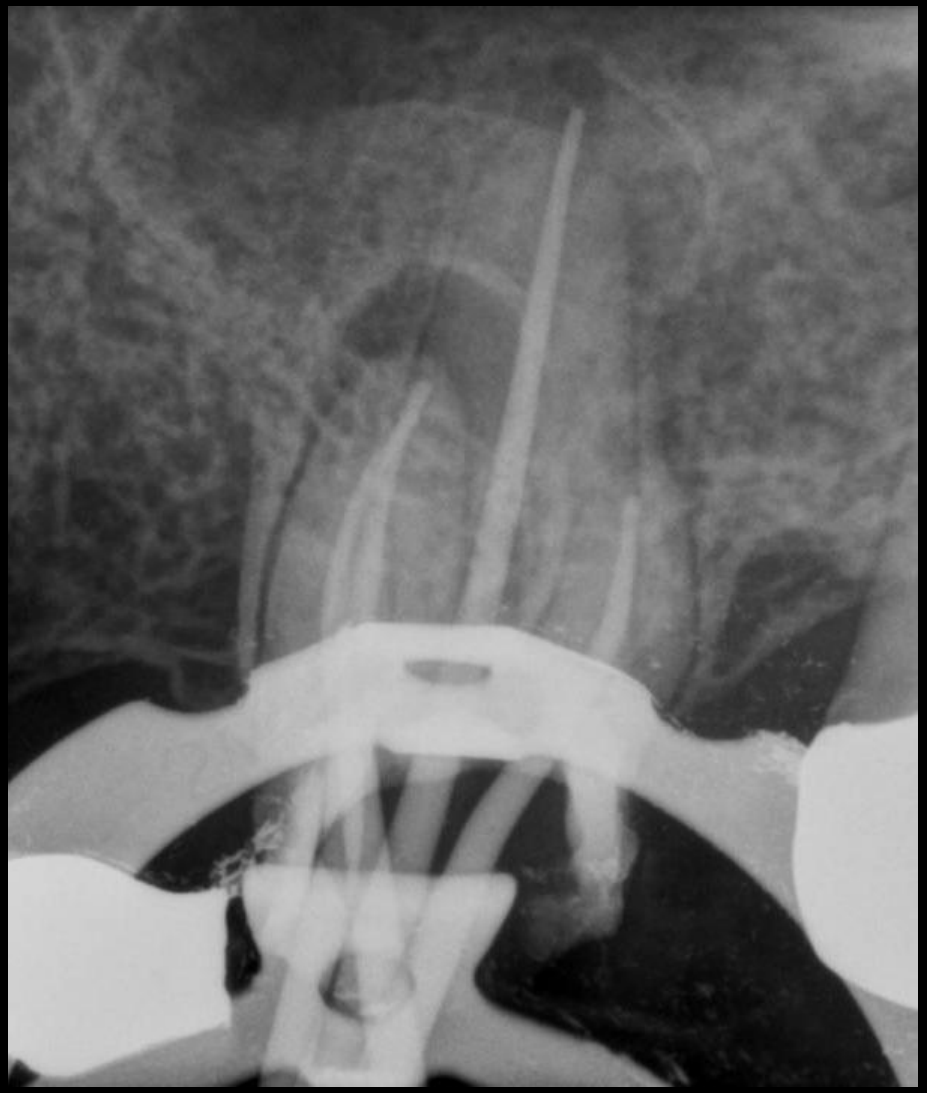
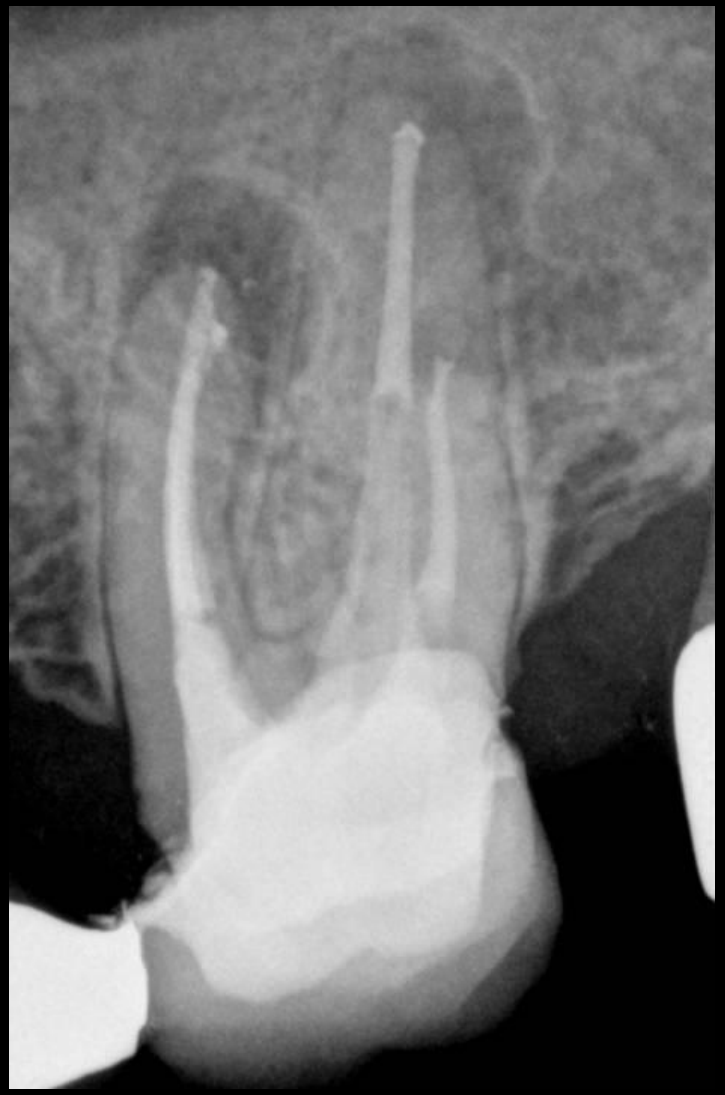










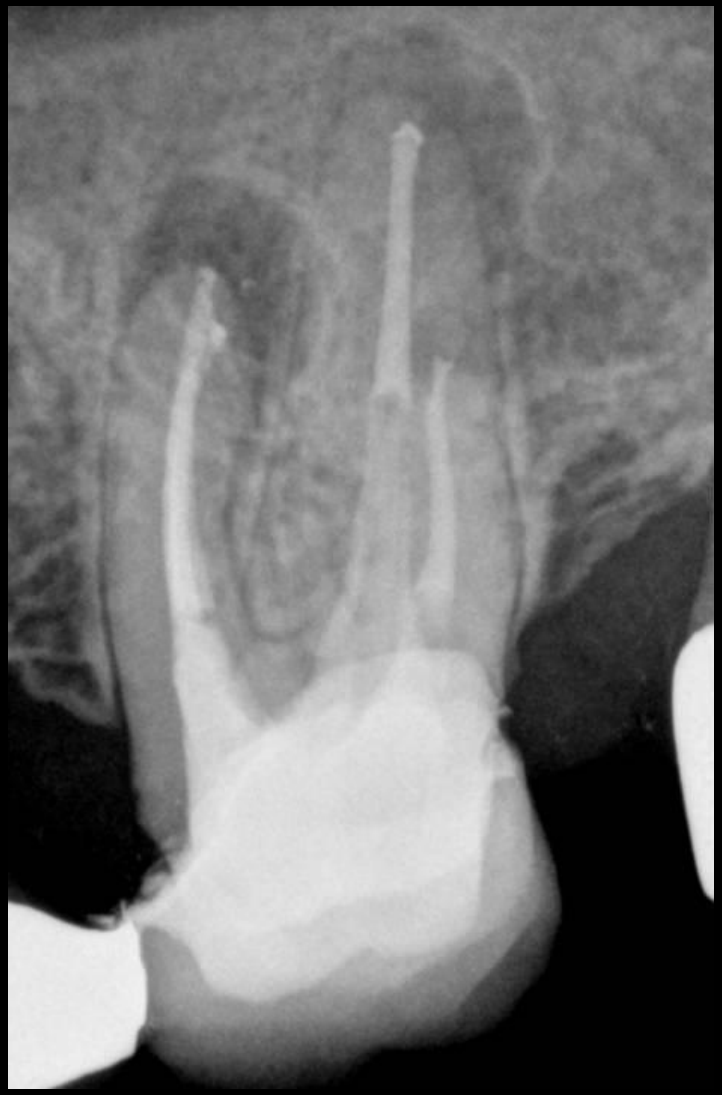
• The root canals were prepared with rotary nickel-titanium (NiTi) files (ProFile in combination with GTX and FlexMaster).

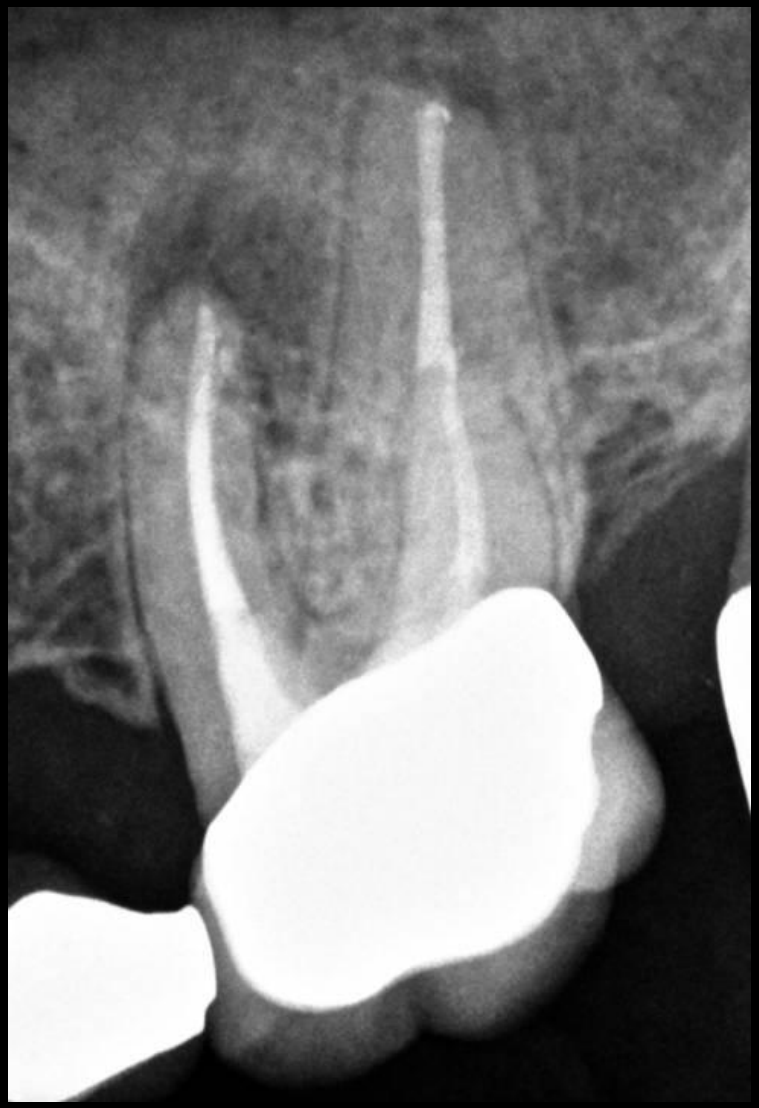
• A #08 K-file was used to assure patency of the apical foramen by taking it 1 mm beyond the WL.

• Irrigation with 5% NaOCl.

• Final irrigation with 10% citric acid to remove the smear layer, followed by a final rinse with 2% chlorhexidine. These solutions were activated with ultrasonics for about 20 seconds each.

• The root canals were filled with gutta-percha and 2Seal using the Schilder´s vertical compaction technique.





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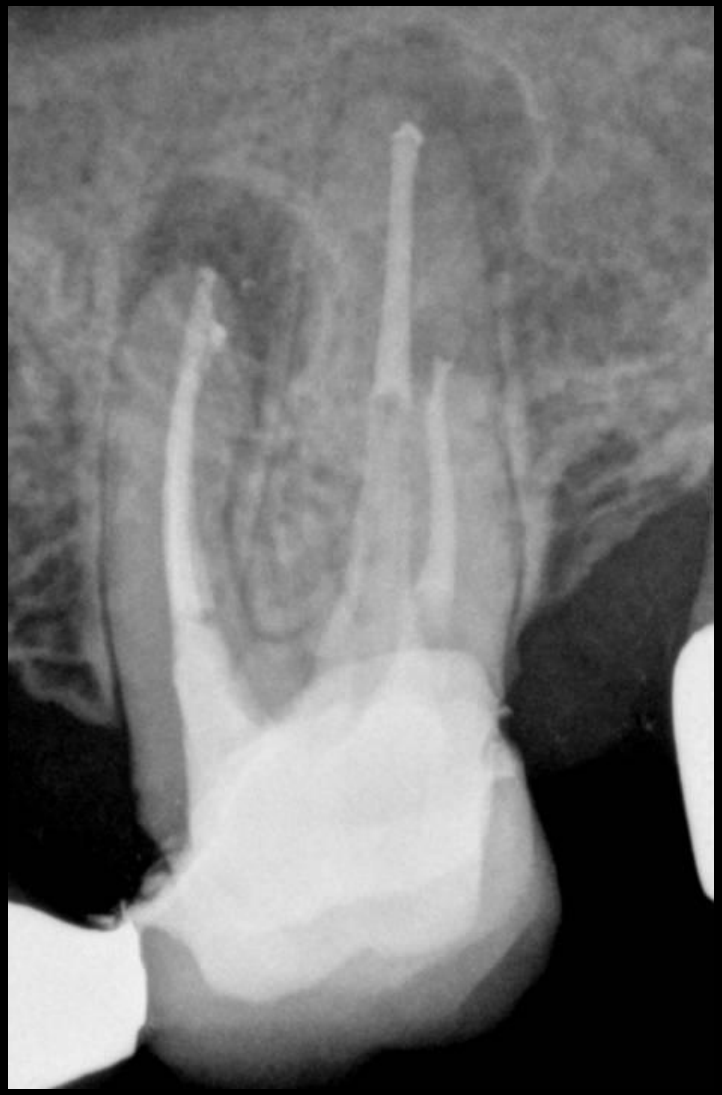

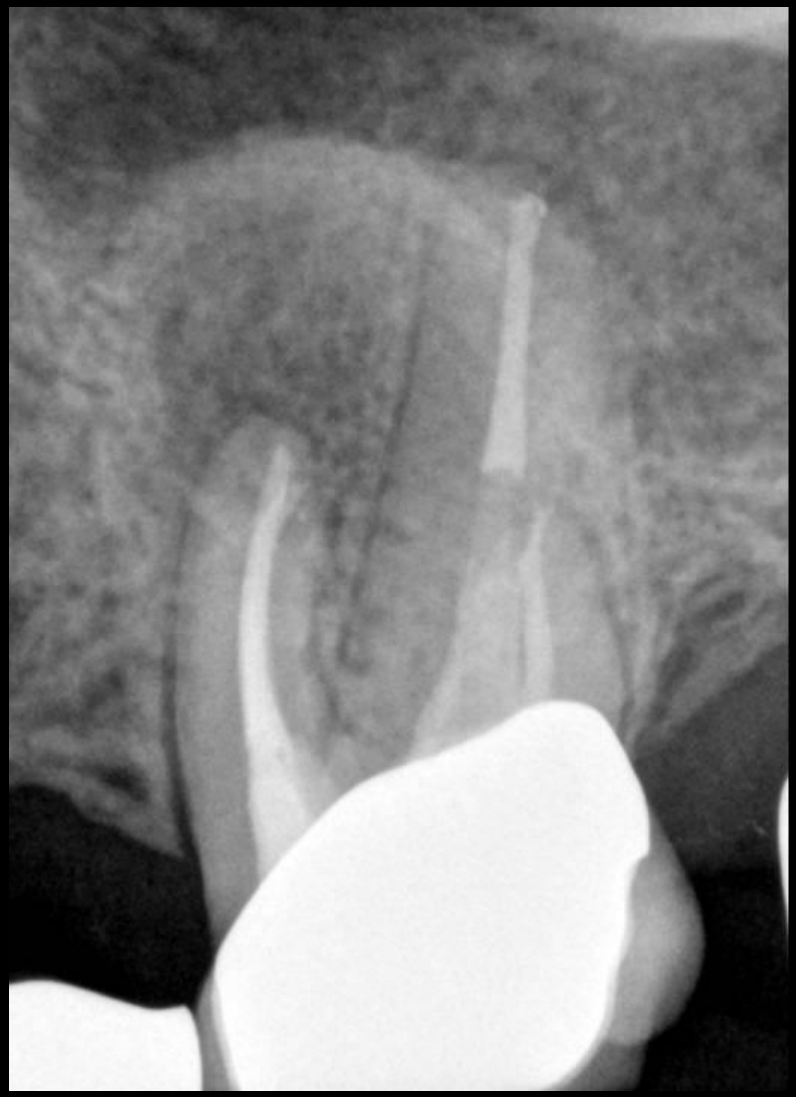

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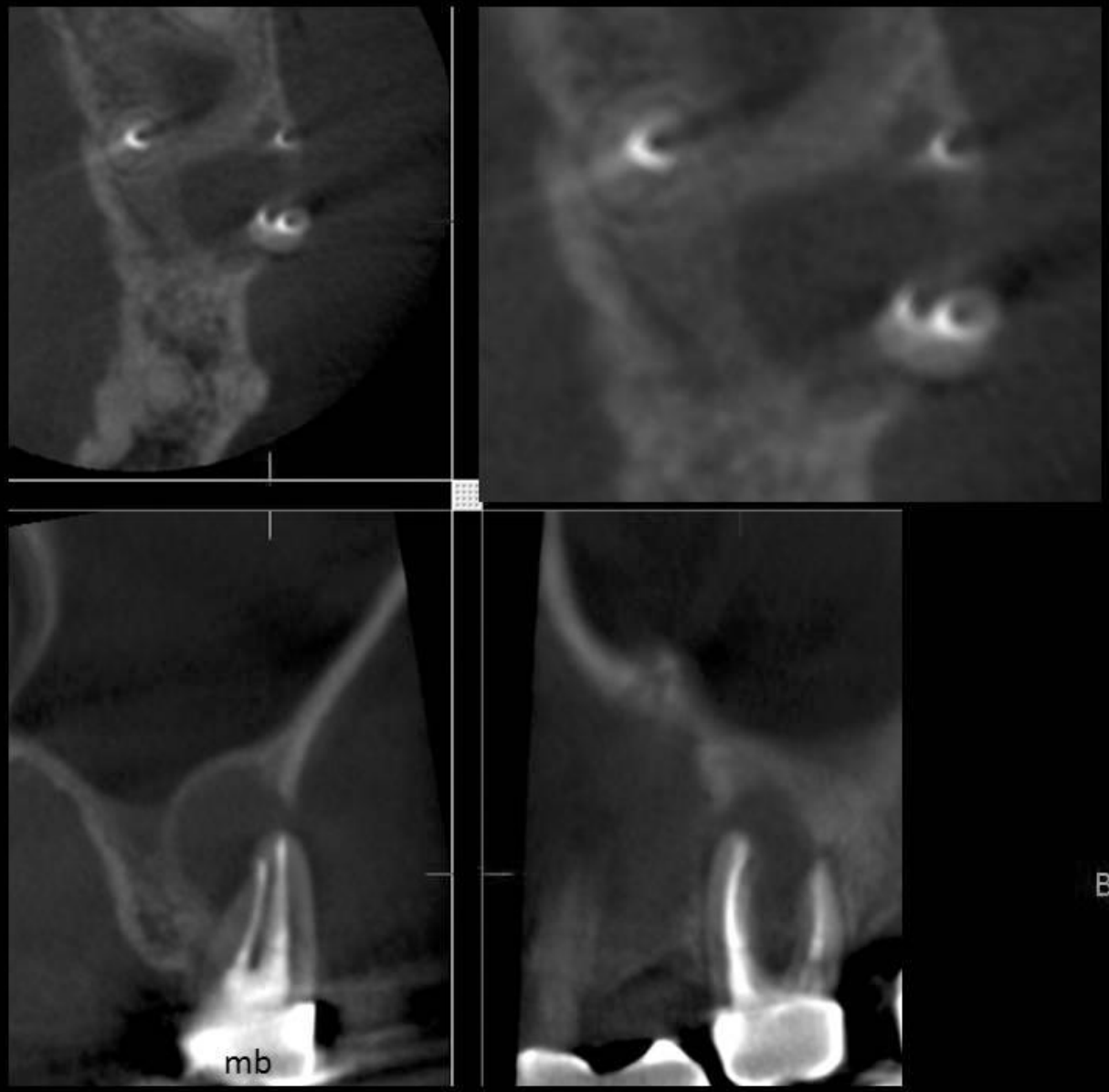

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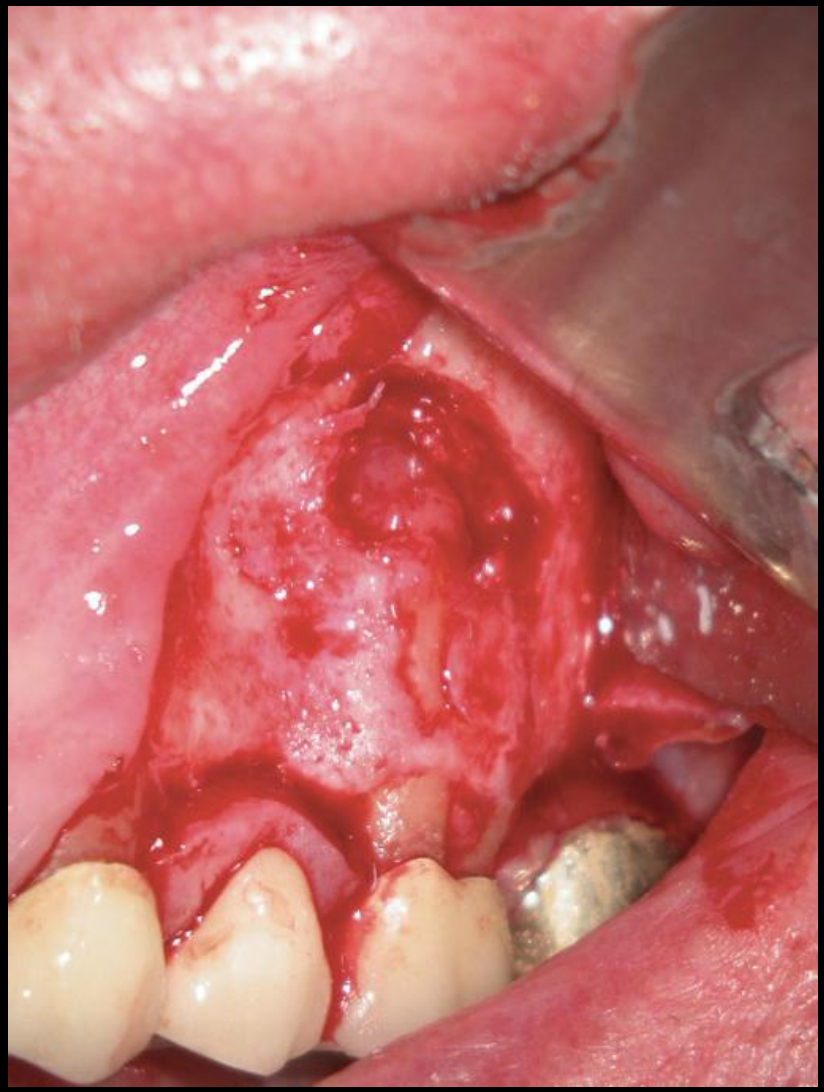

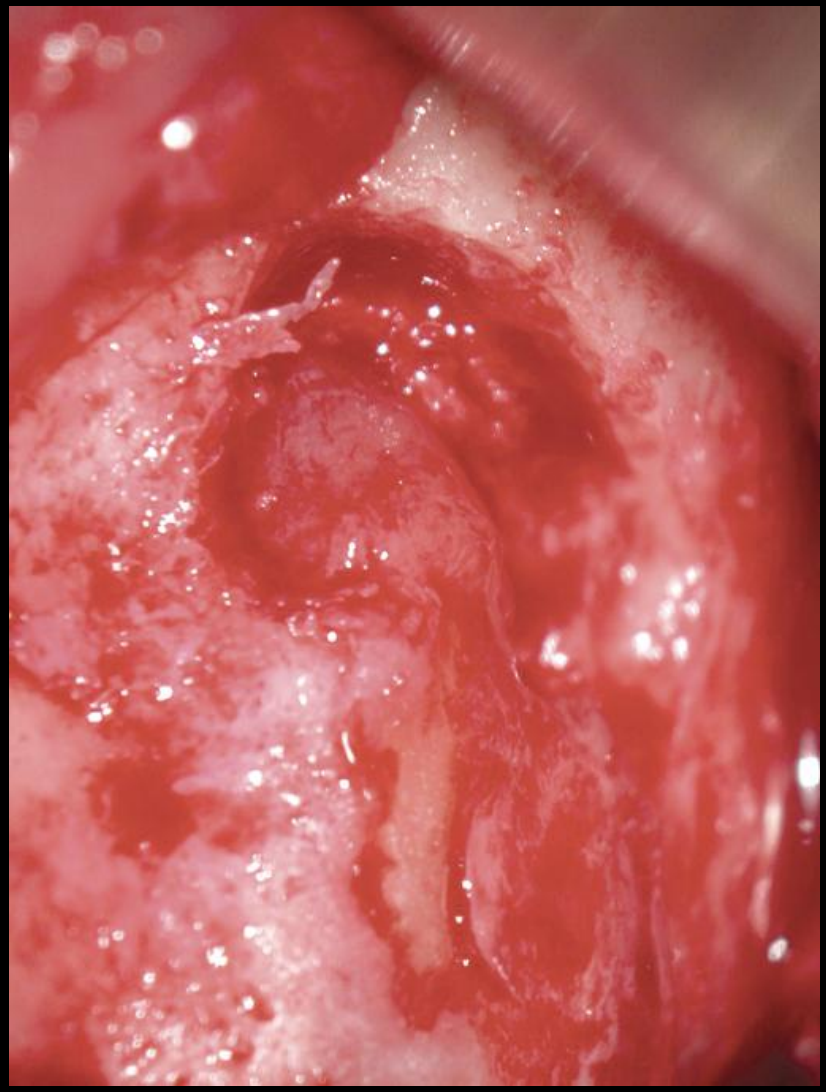

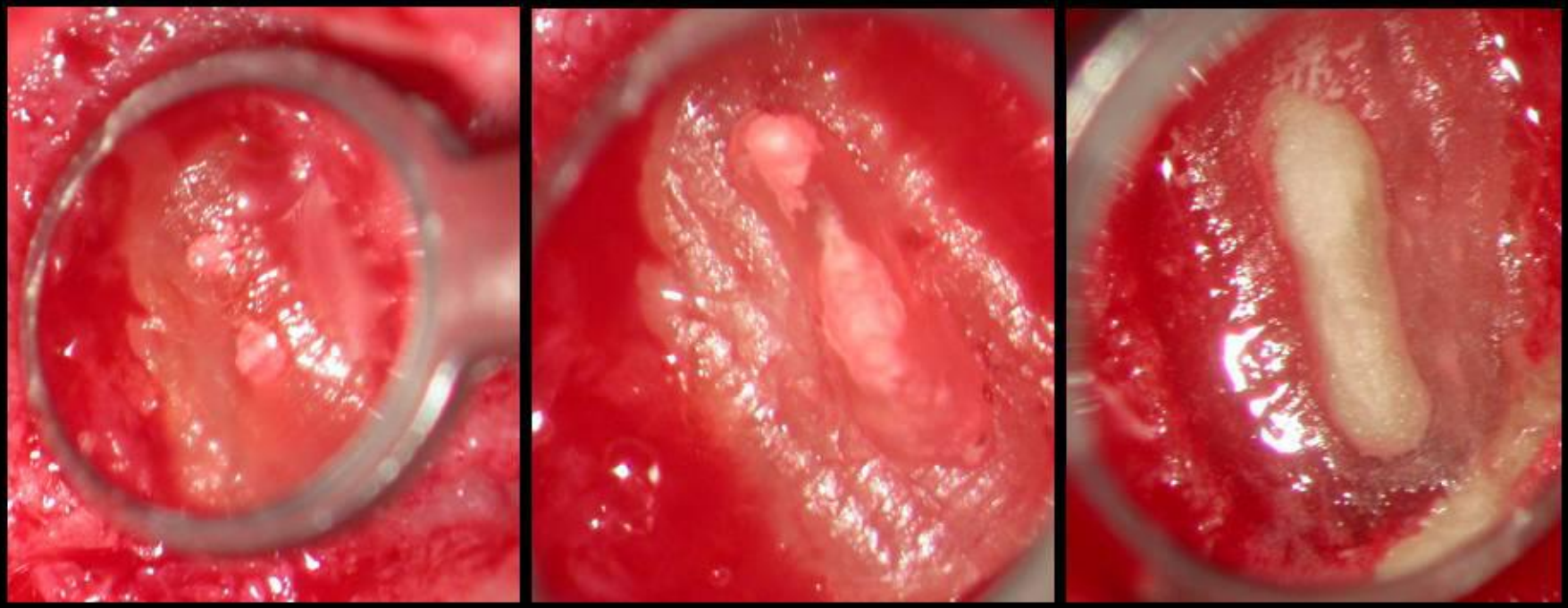

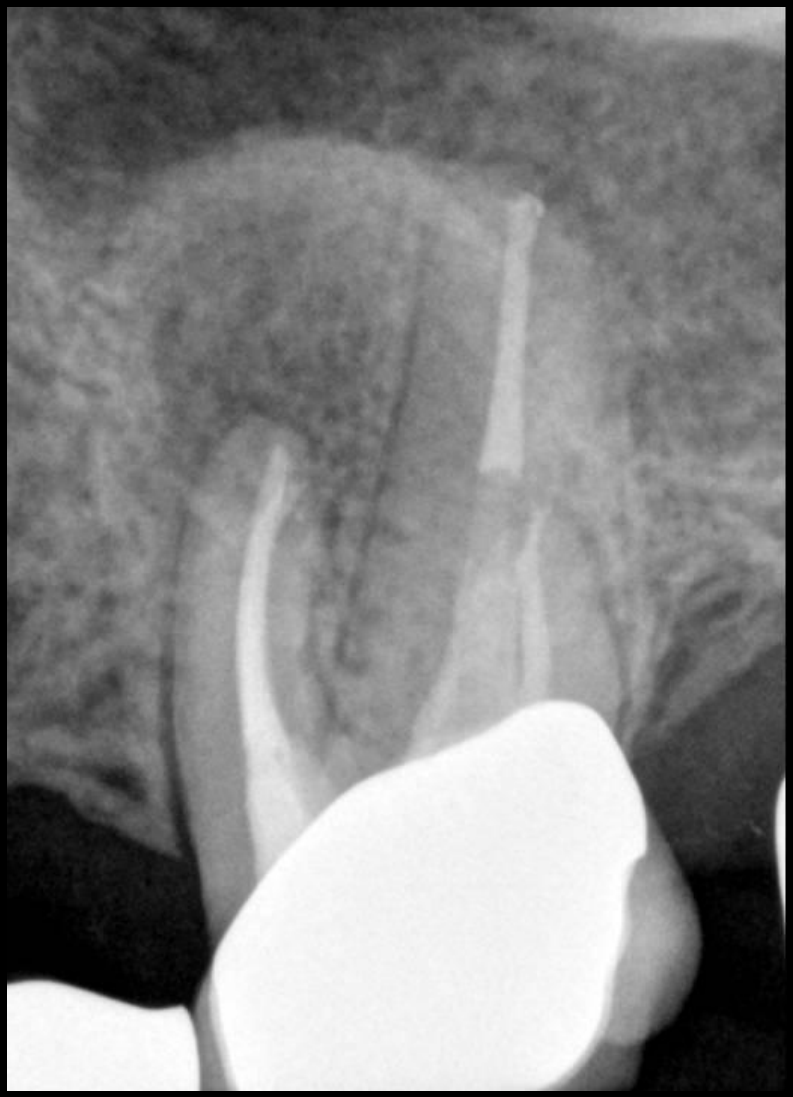

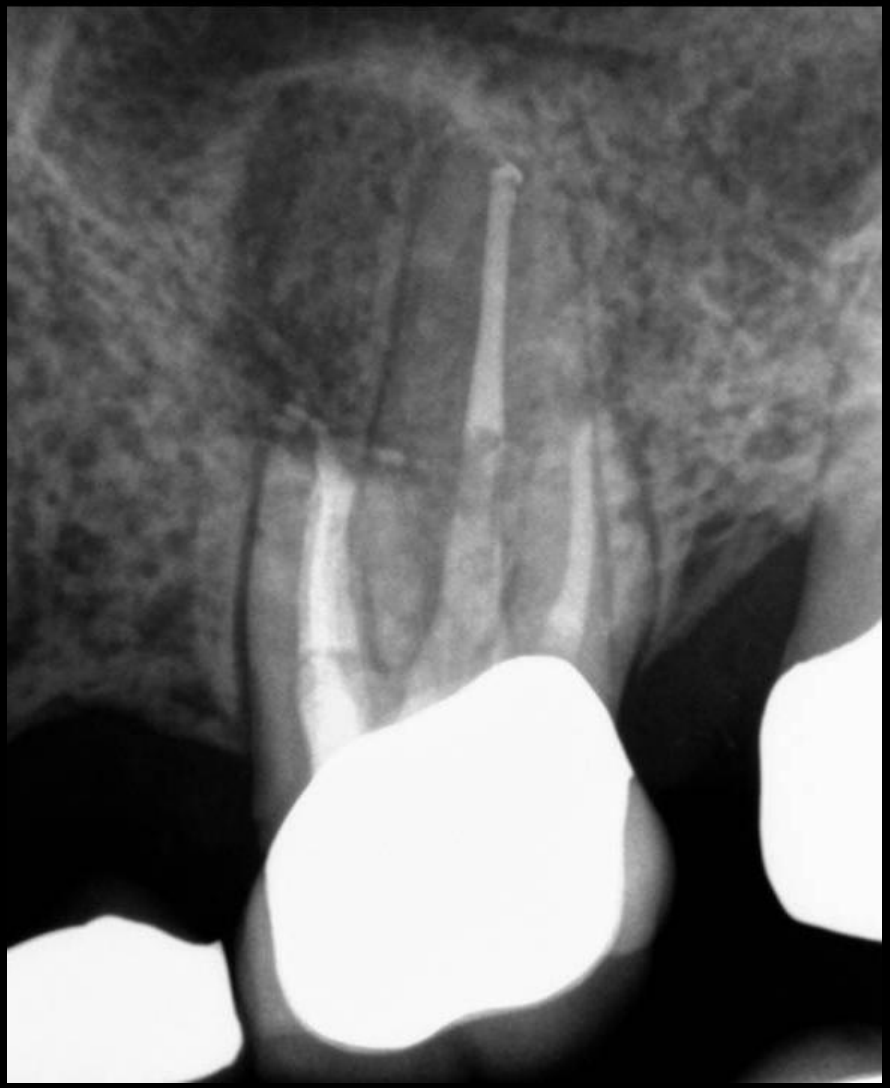

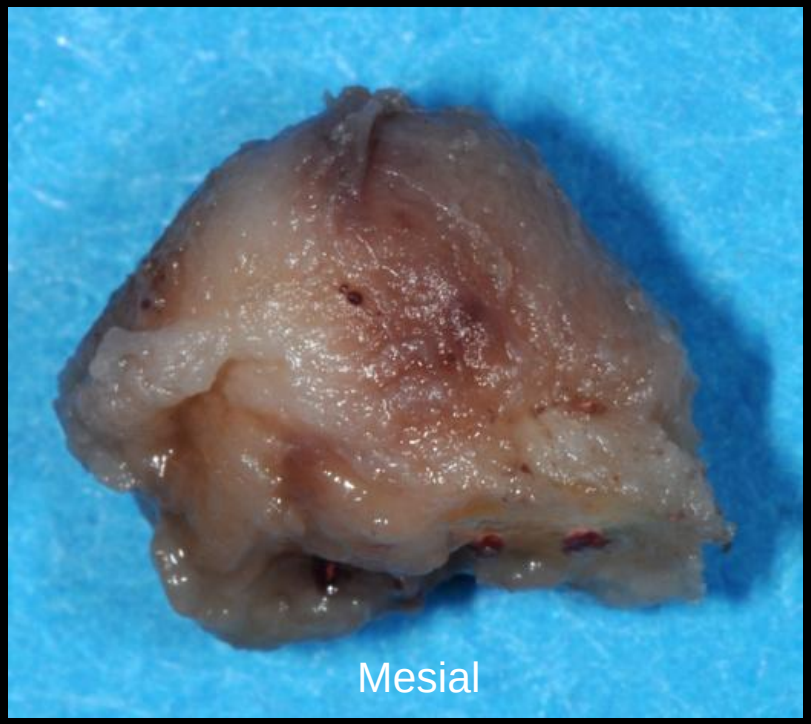
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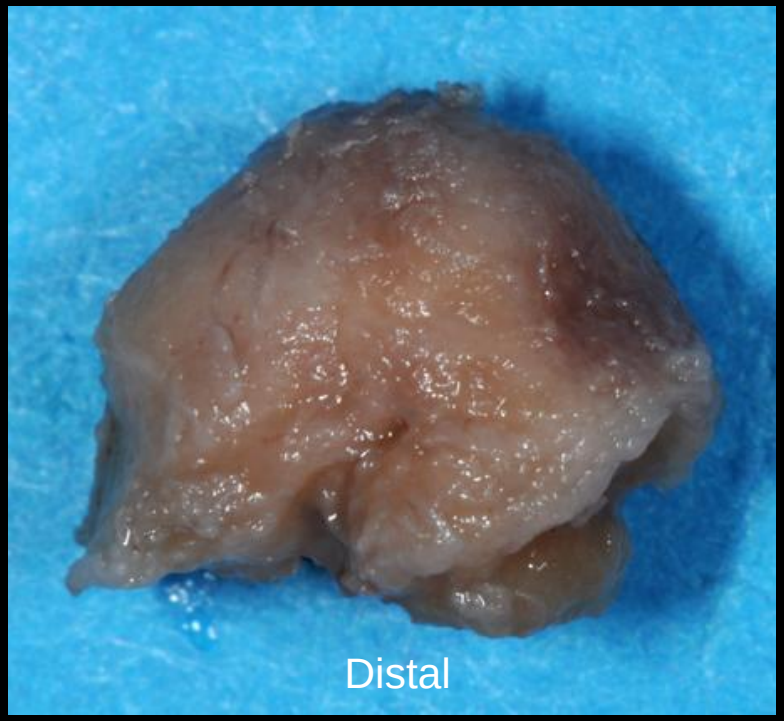
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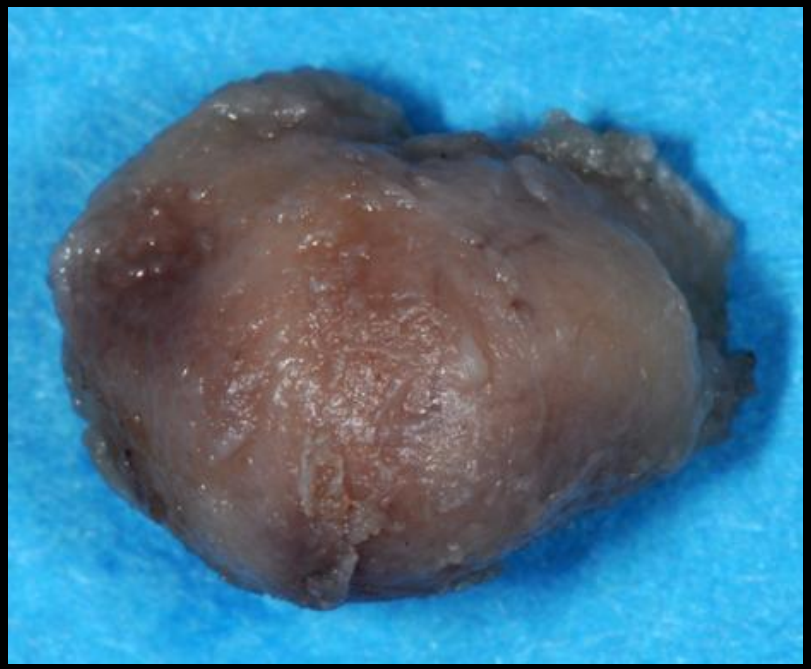

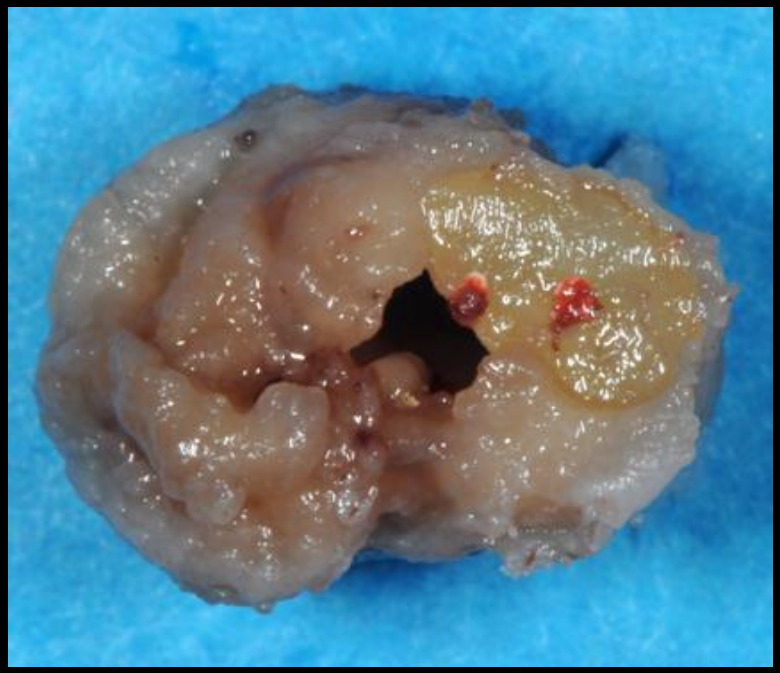

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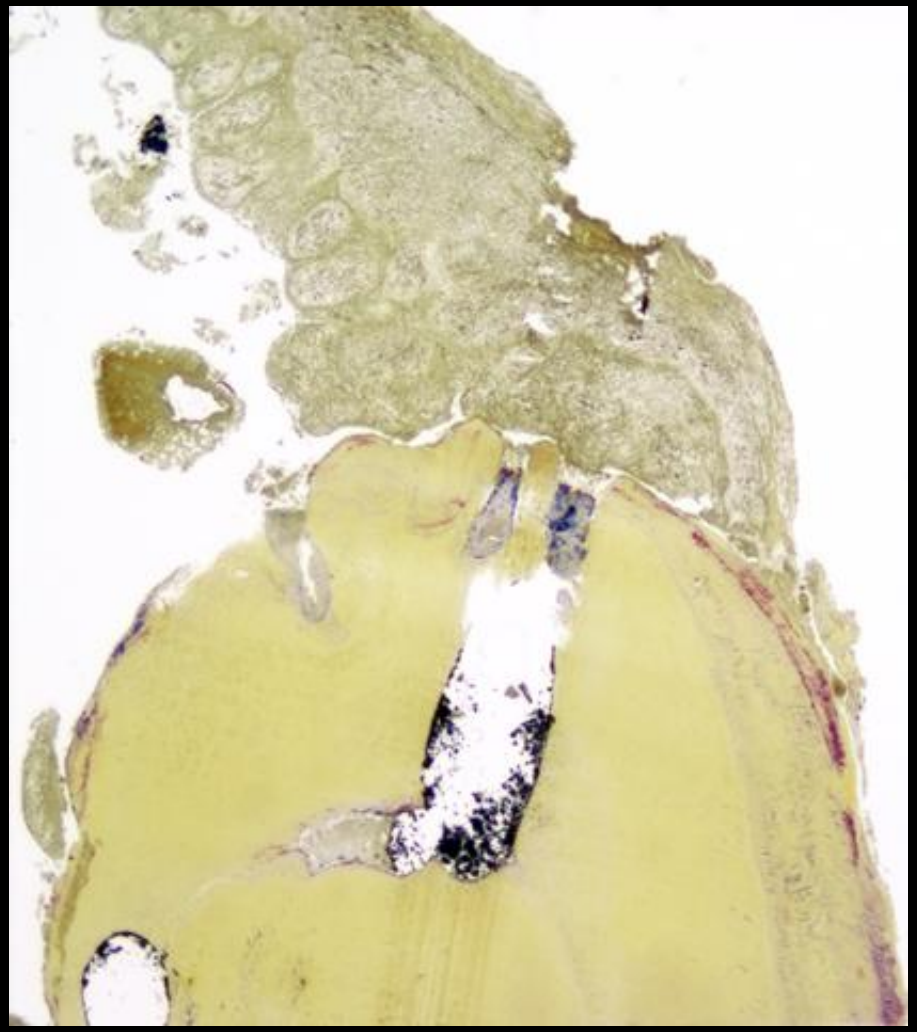

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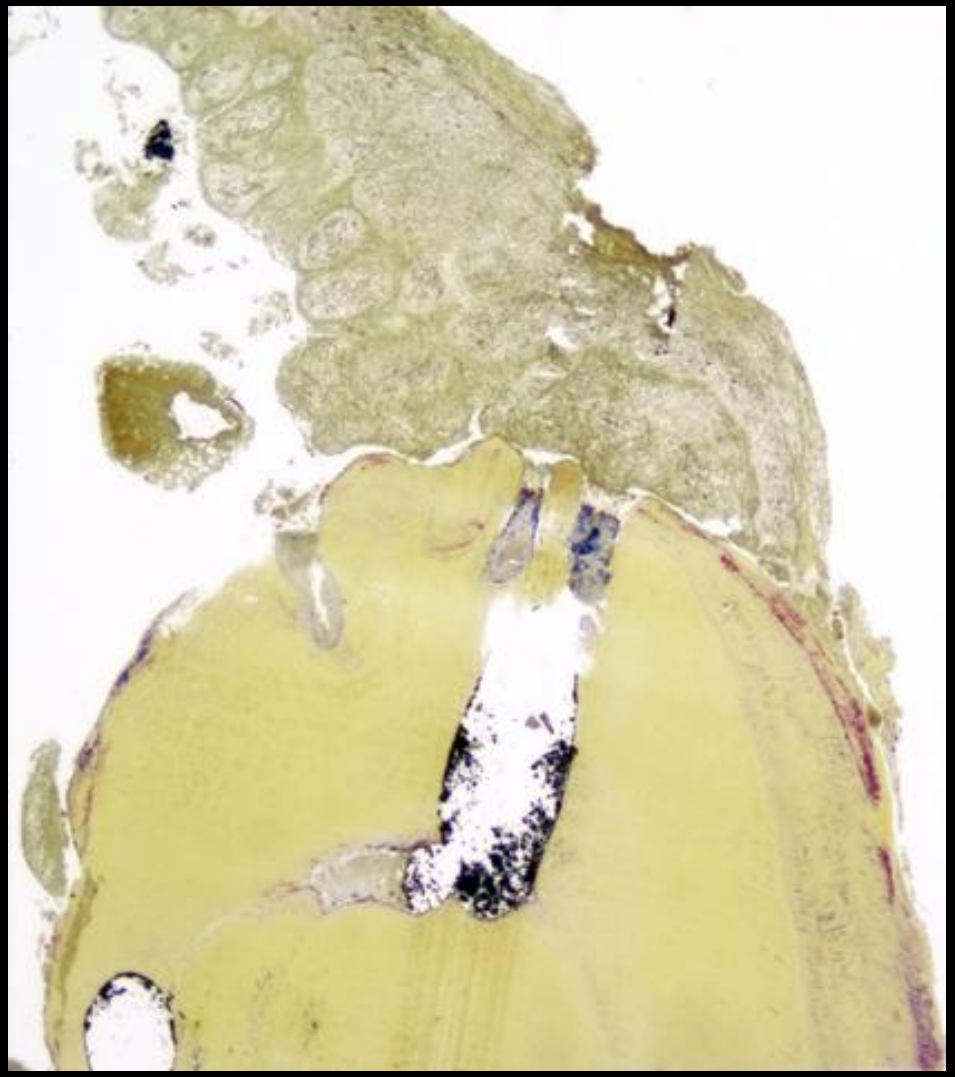

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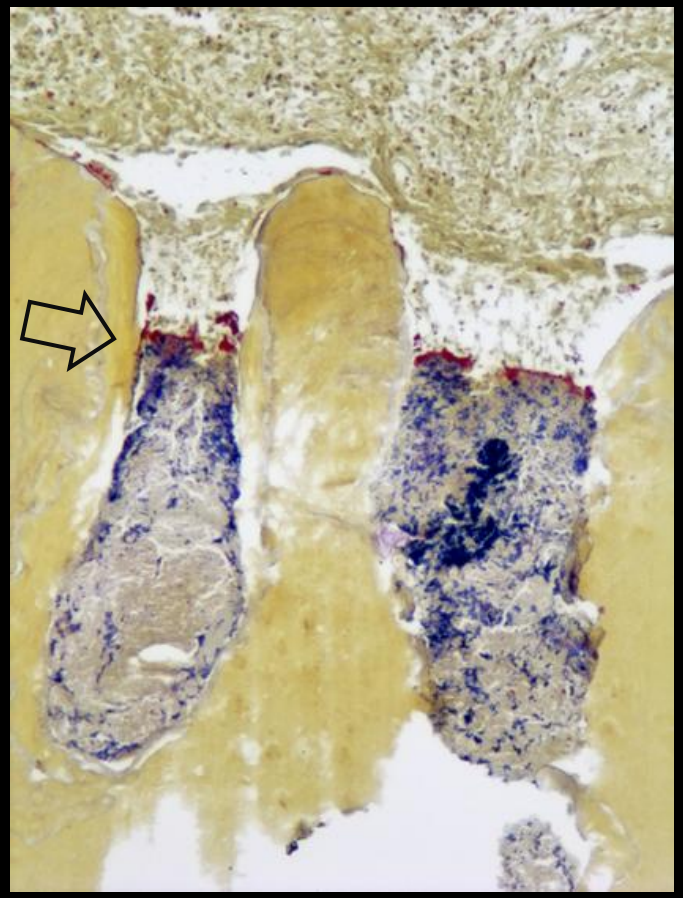

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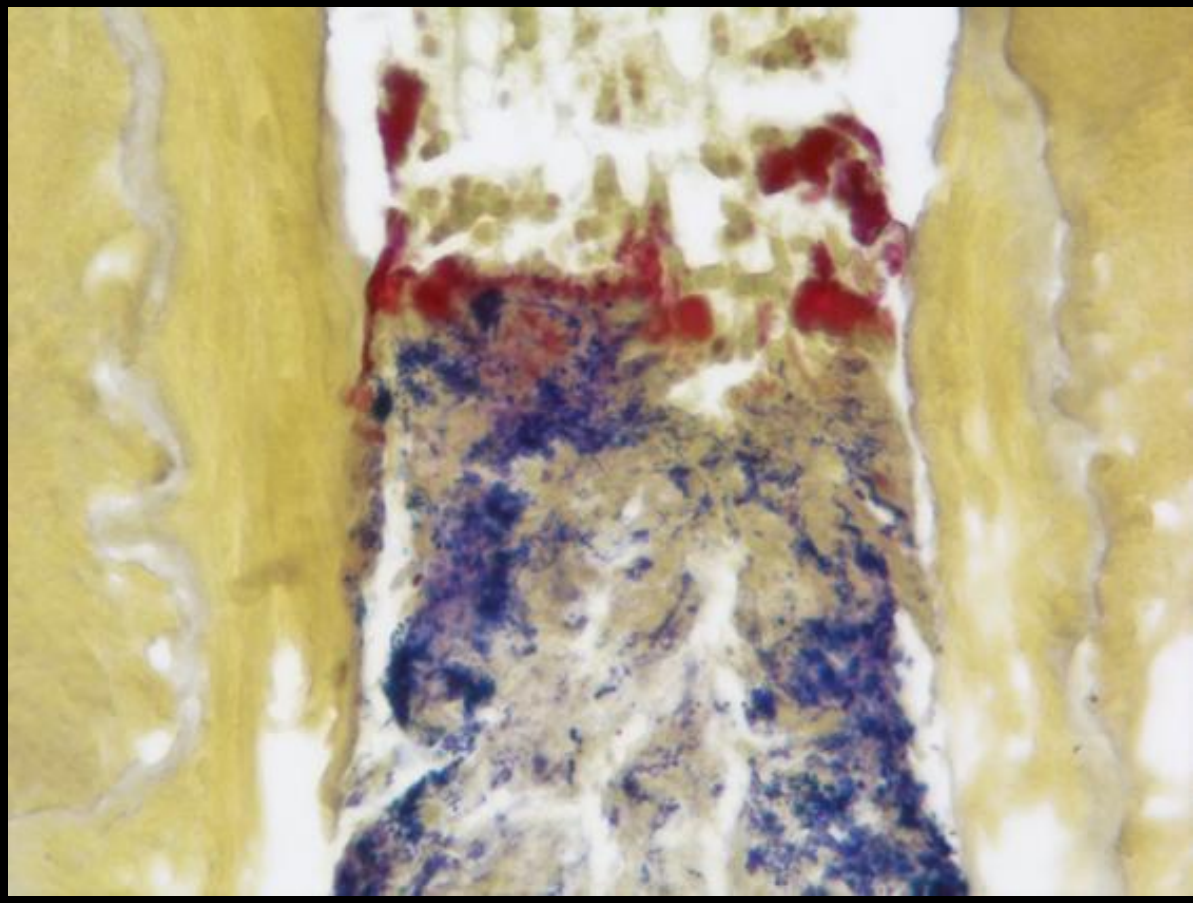

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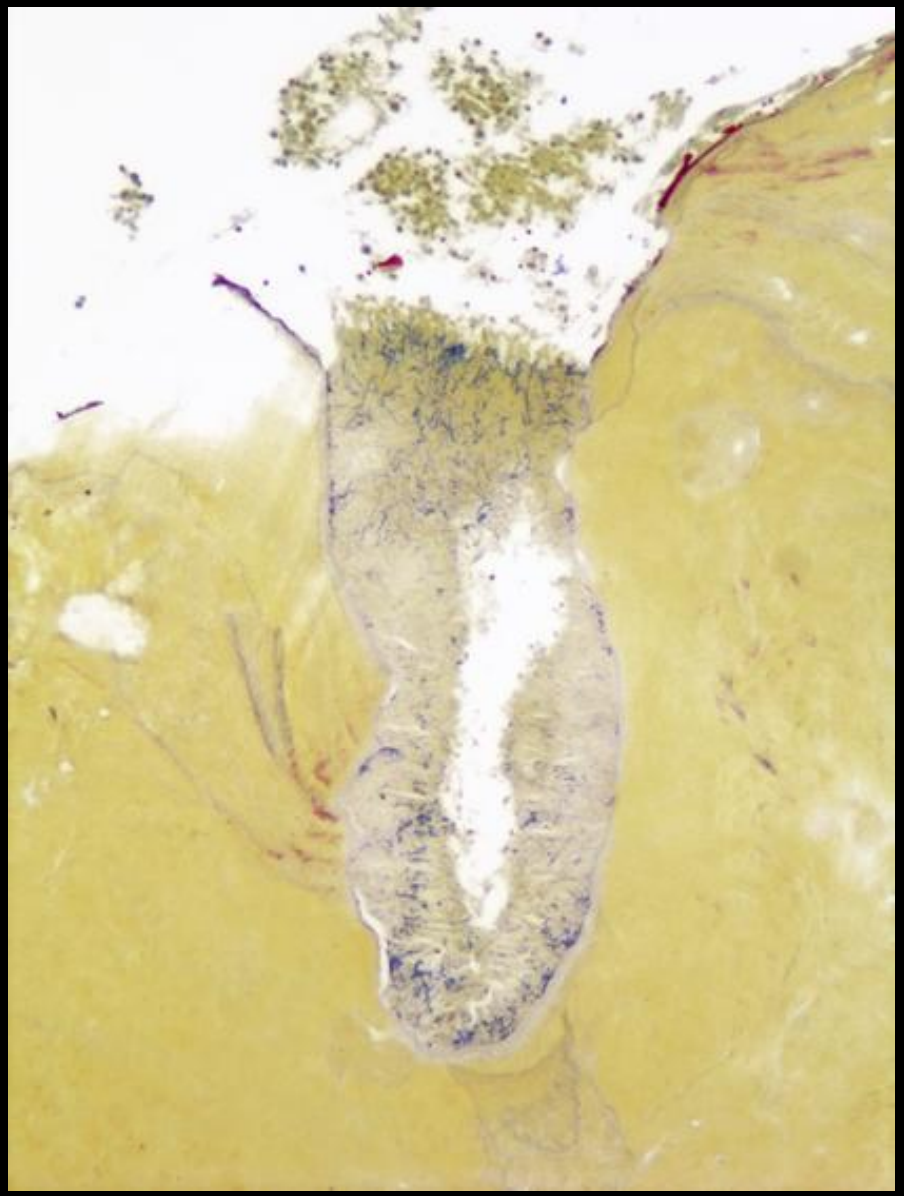

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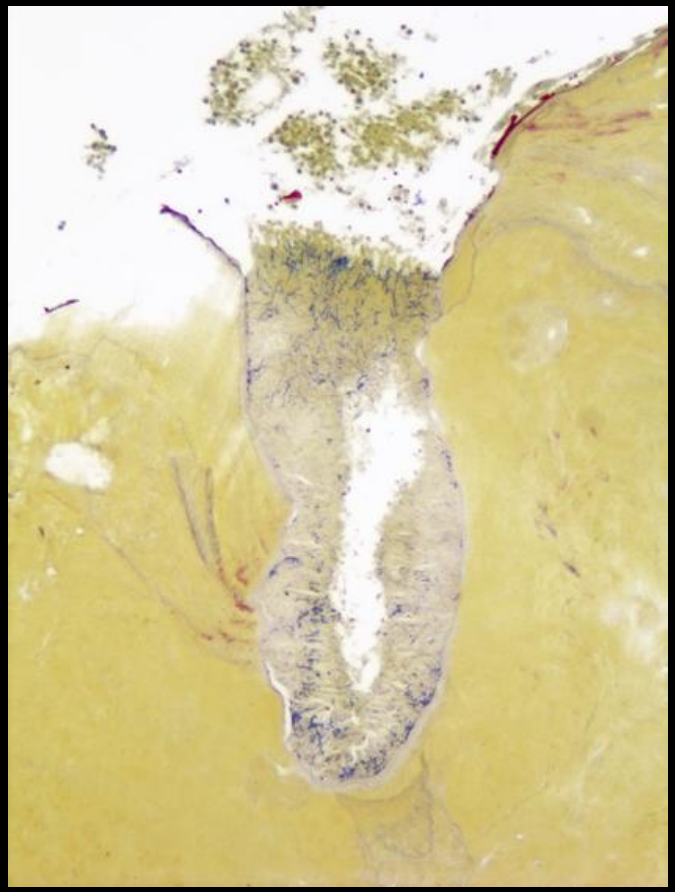

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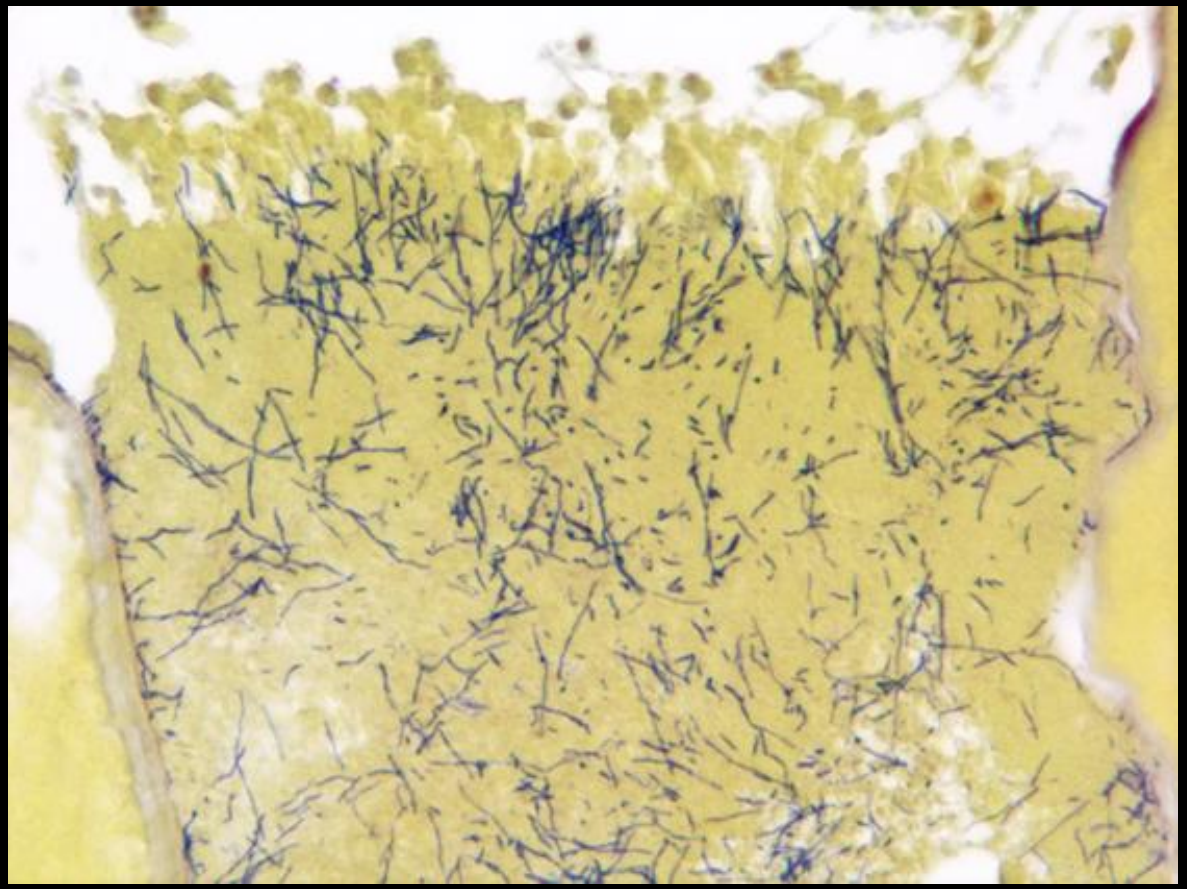

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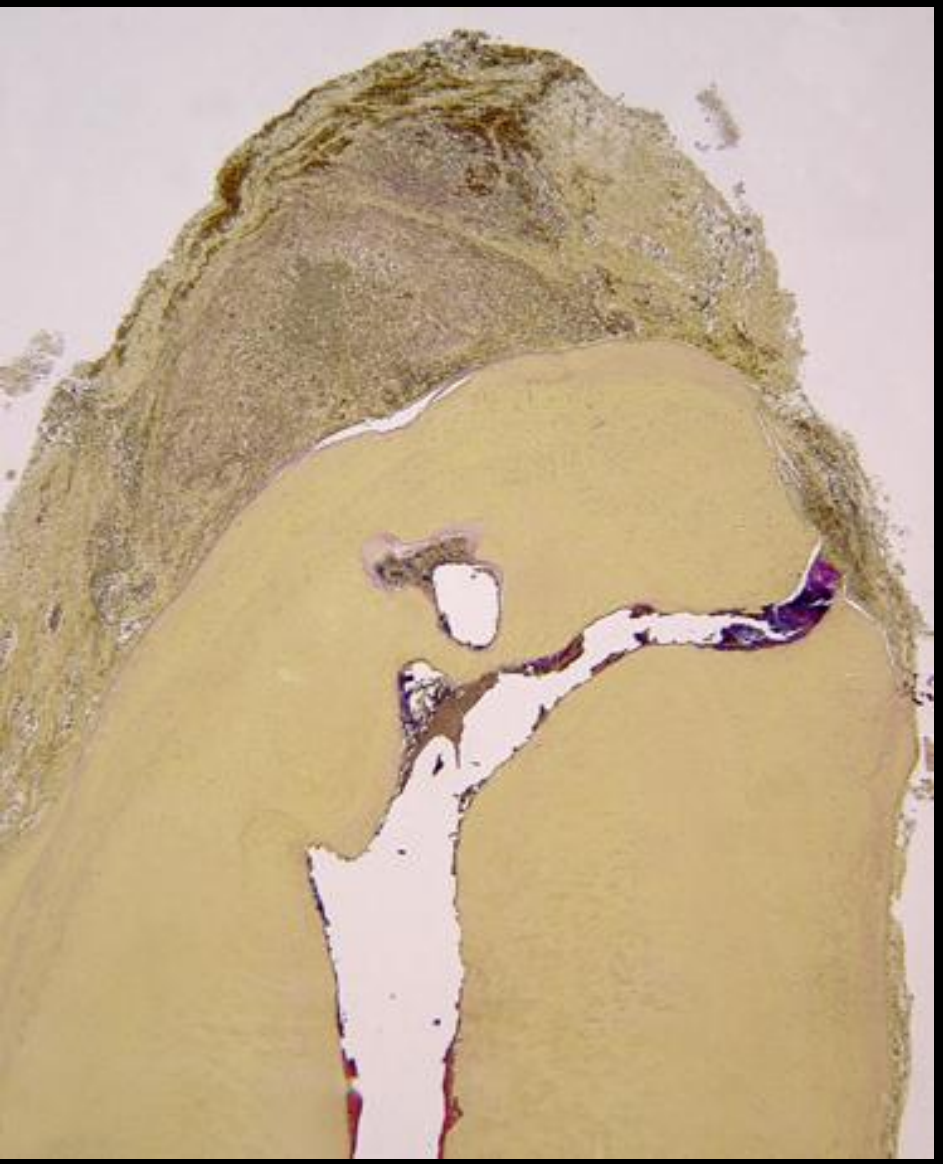
Arnold M, Ricucci D, Siqueira JF Jr

Infection in a complex network of apical ramifications as the cause of persistent apical periodontitis: a case report

Journal of Endodontics 2013; 39:1179-84.

Extension of the intracanal biofilm to the external root surface



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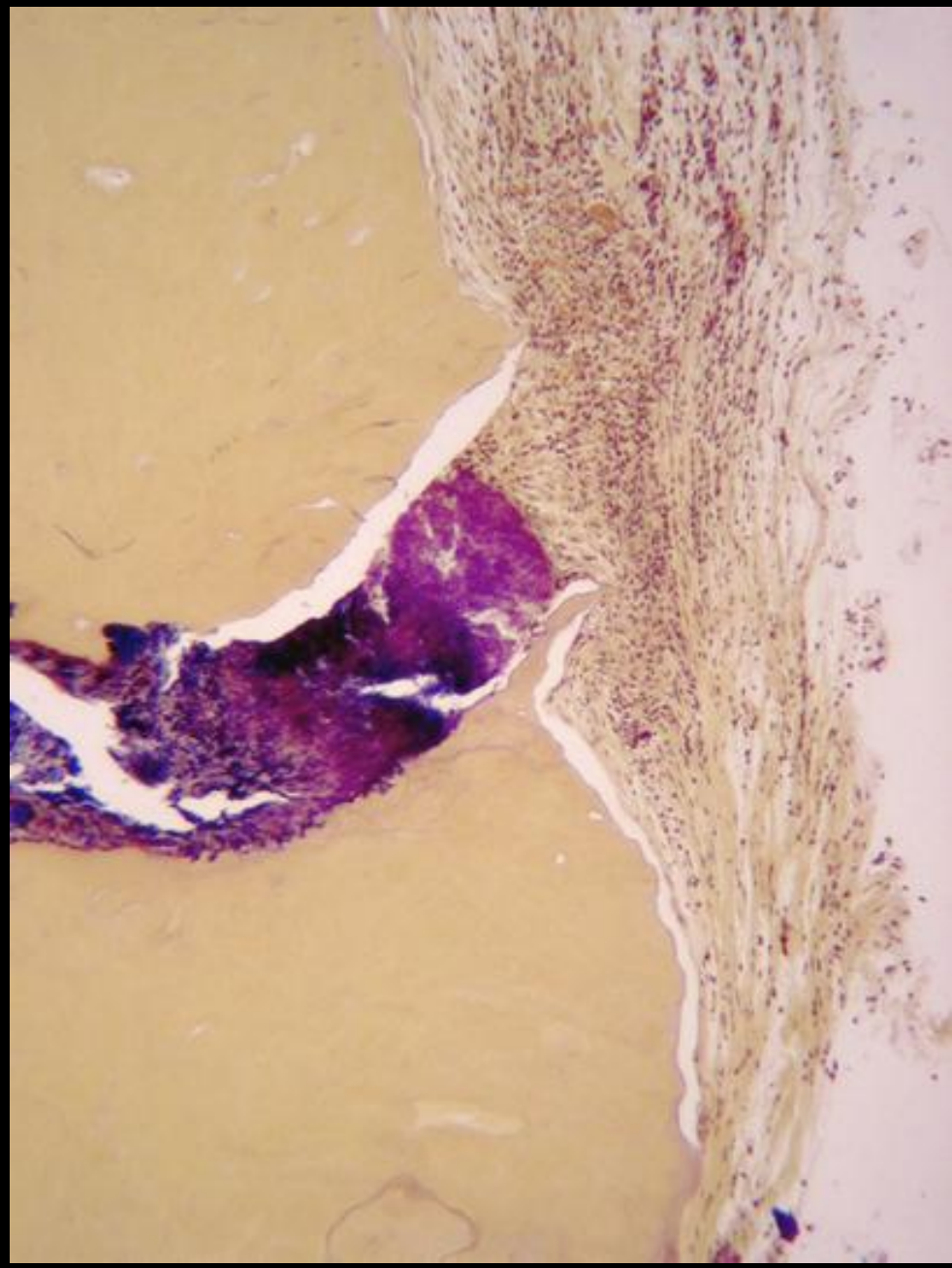

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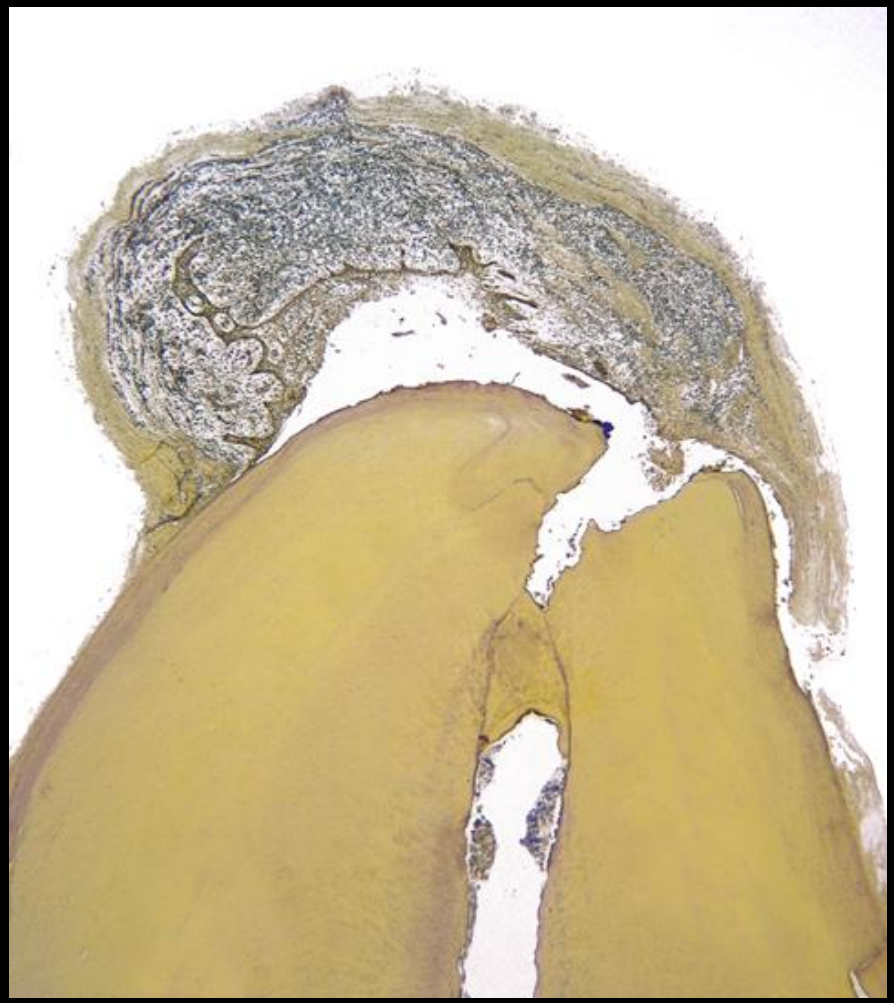

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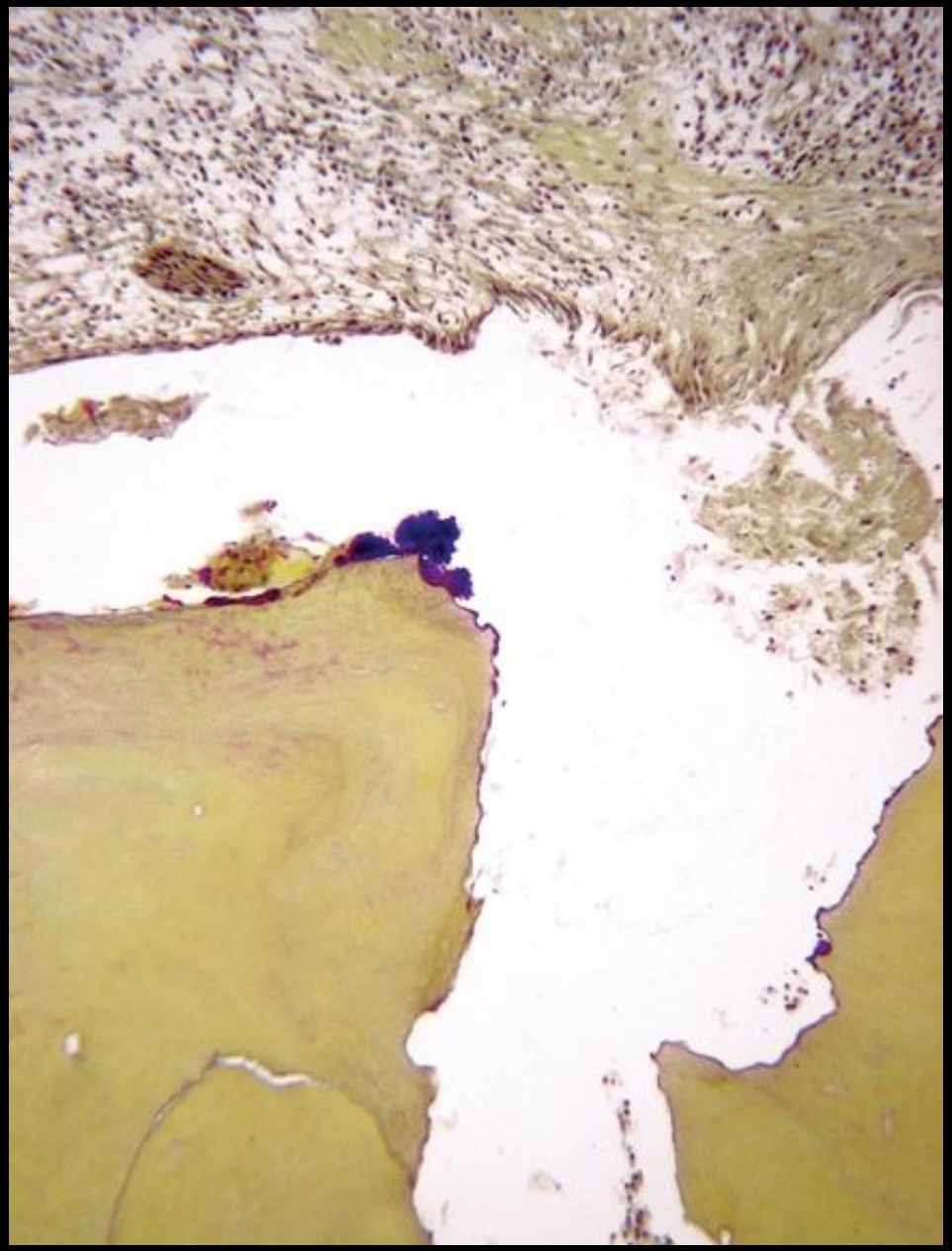

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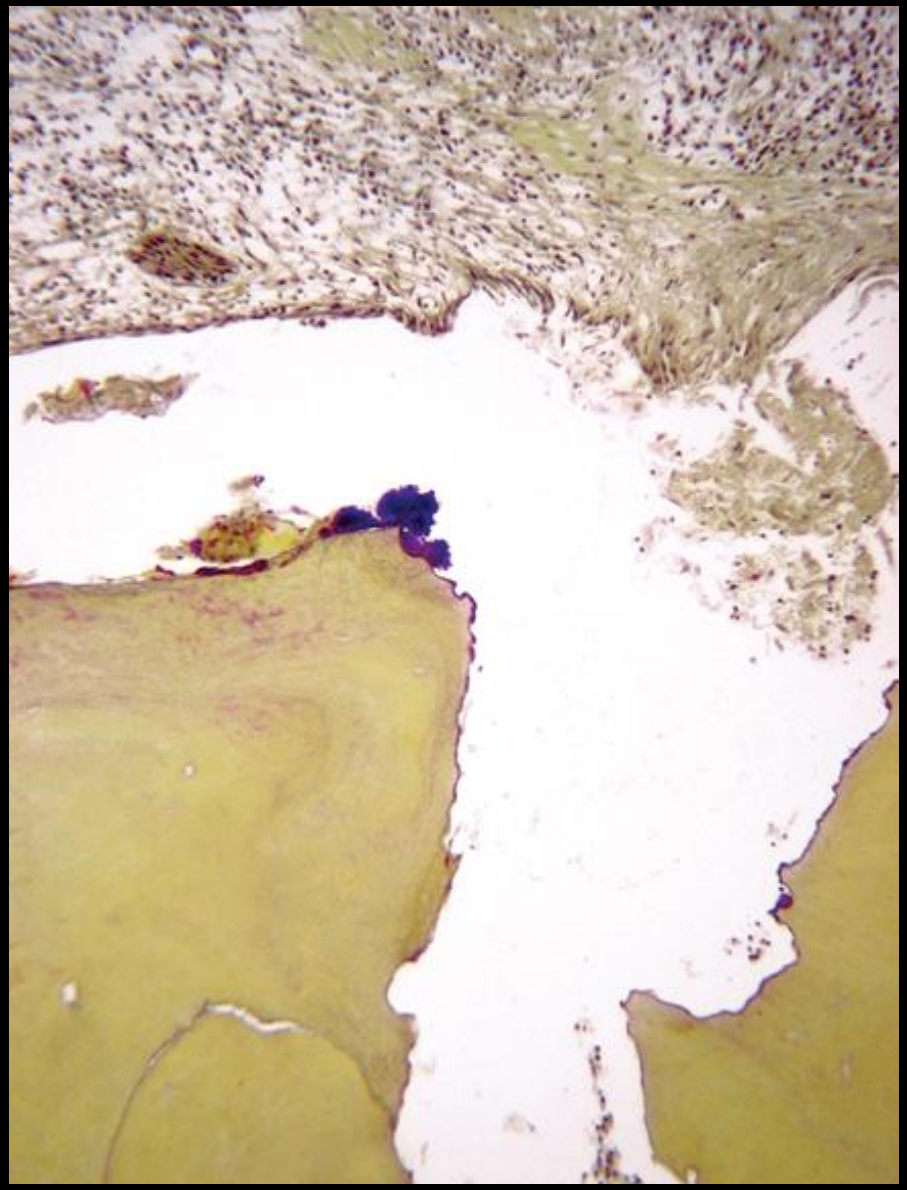

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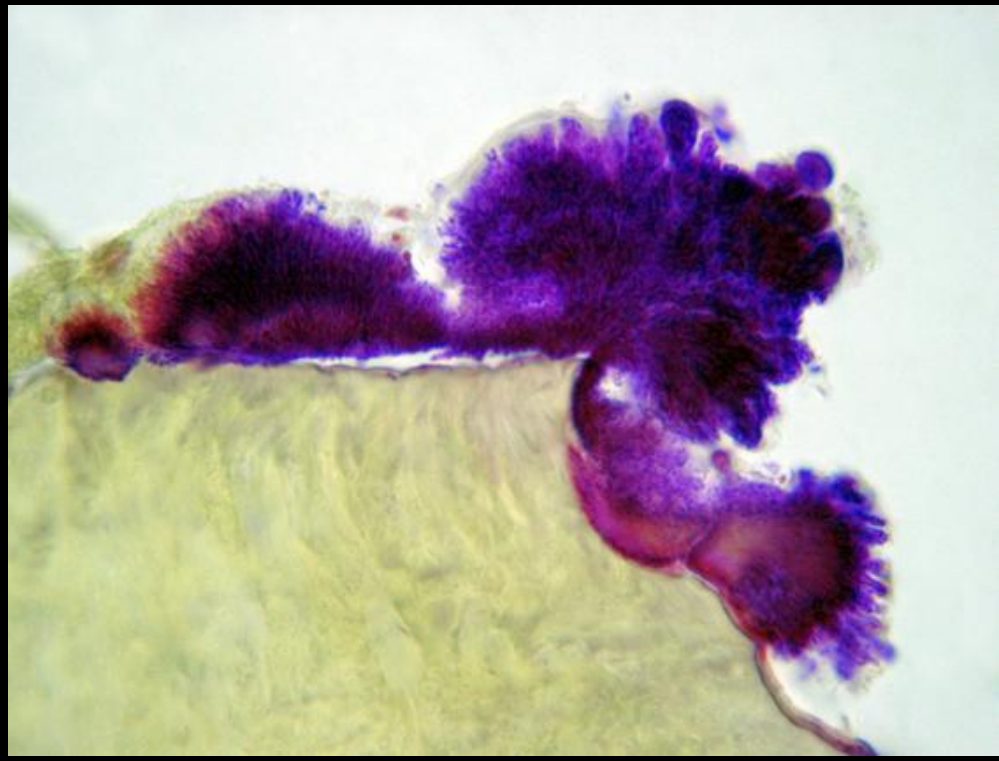

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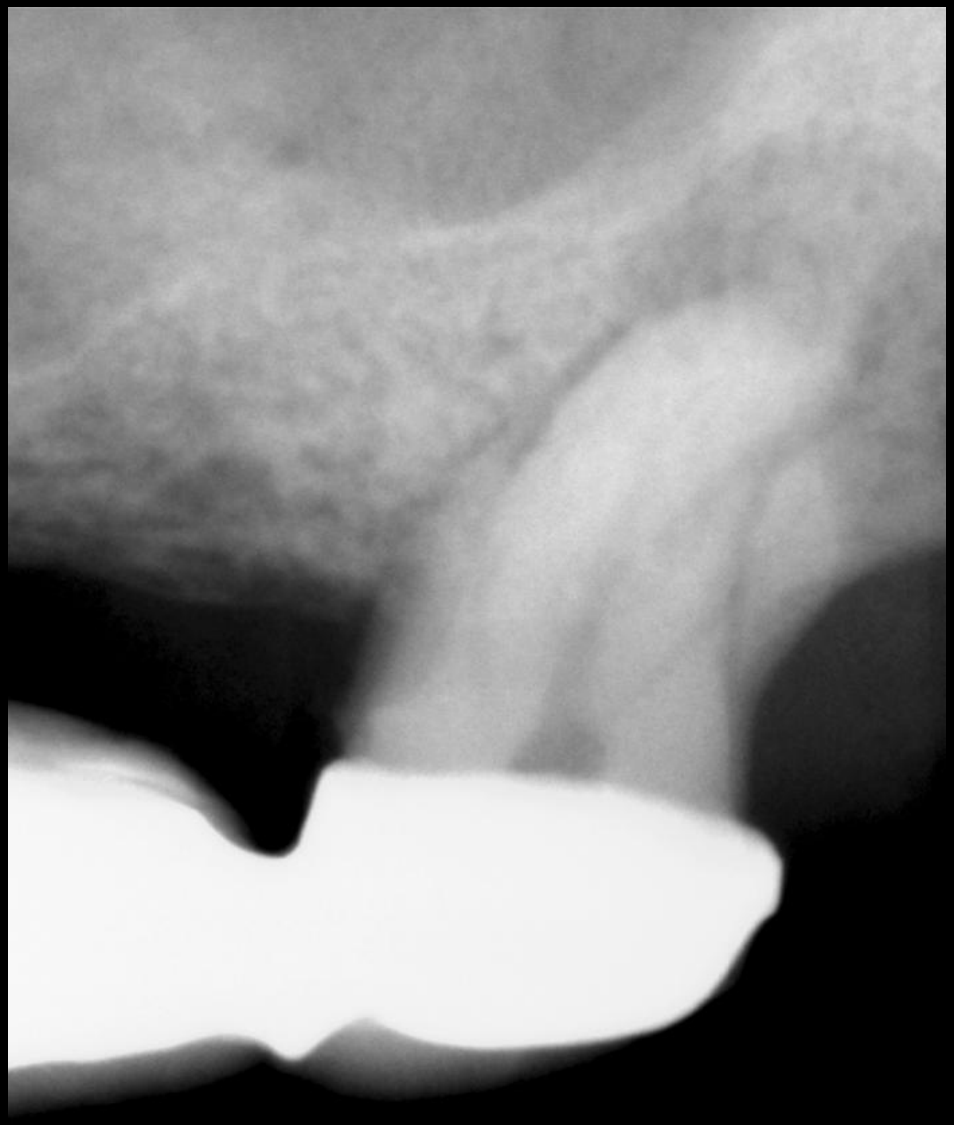

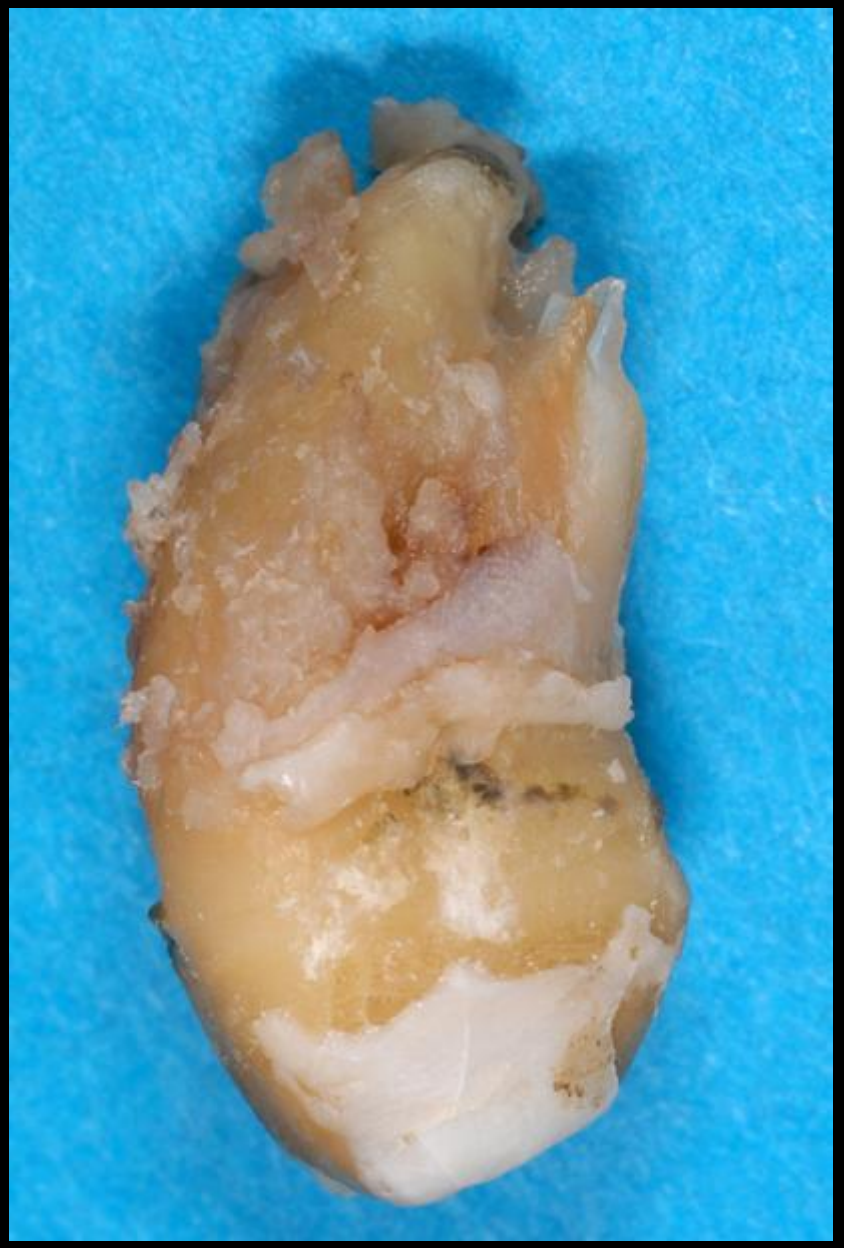

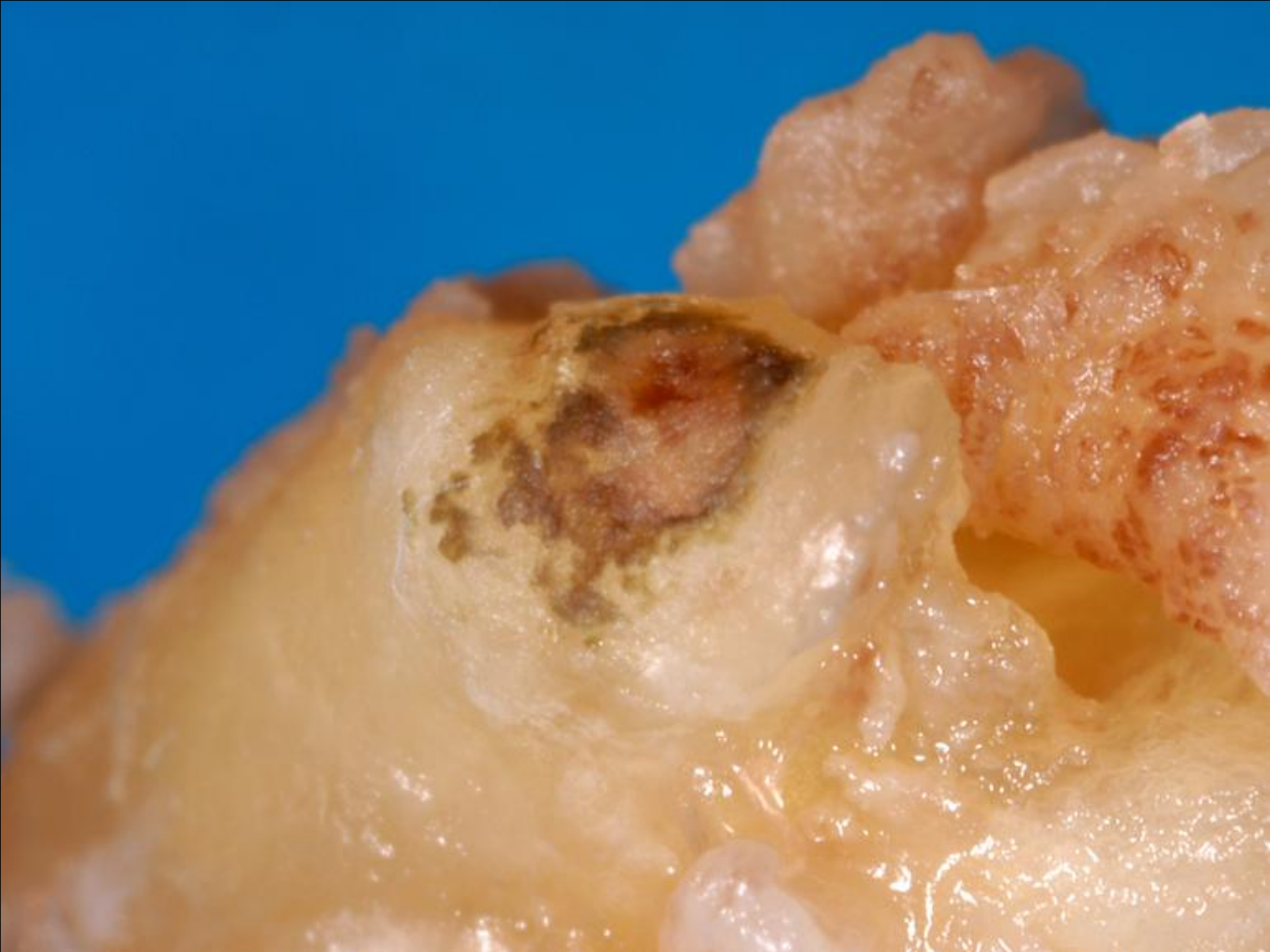

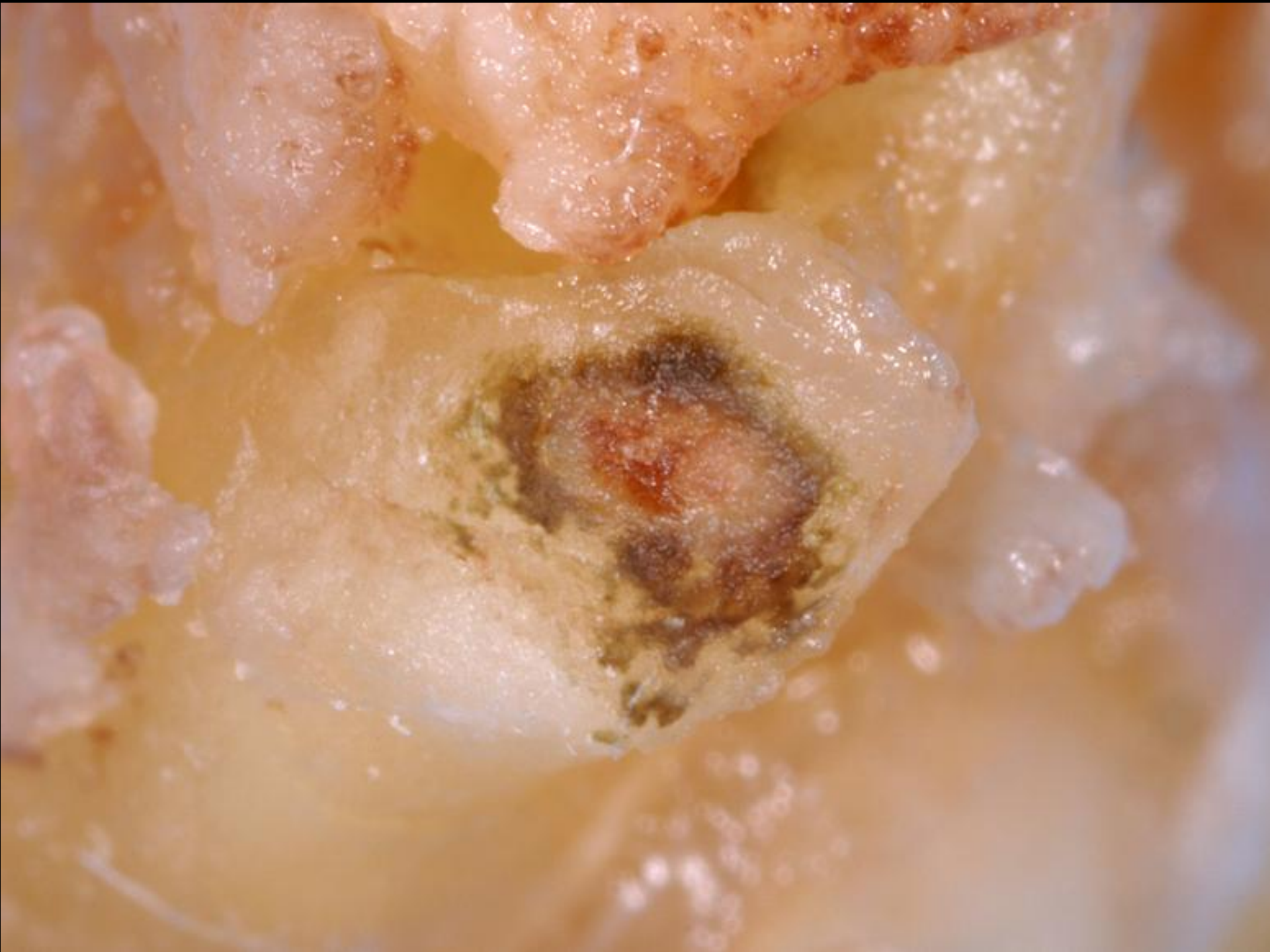

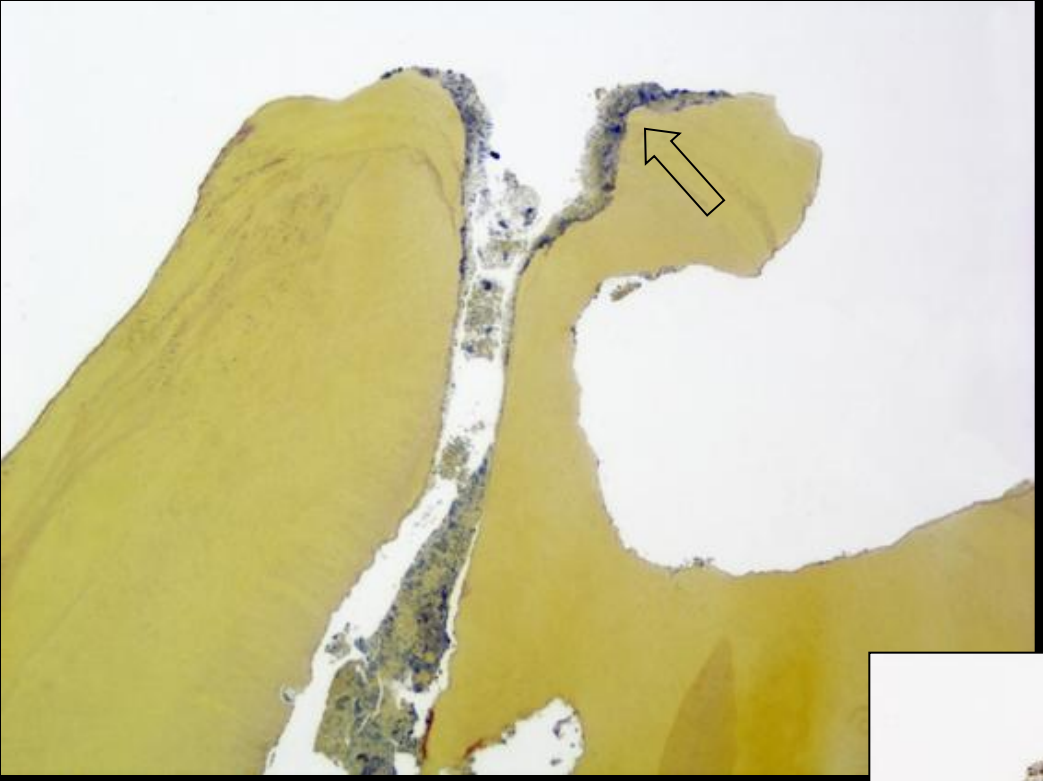

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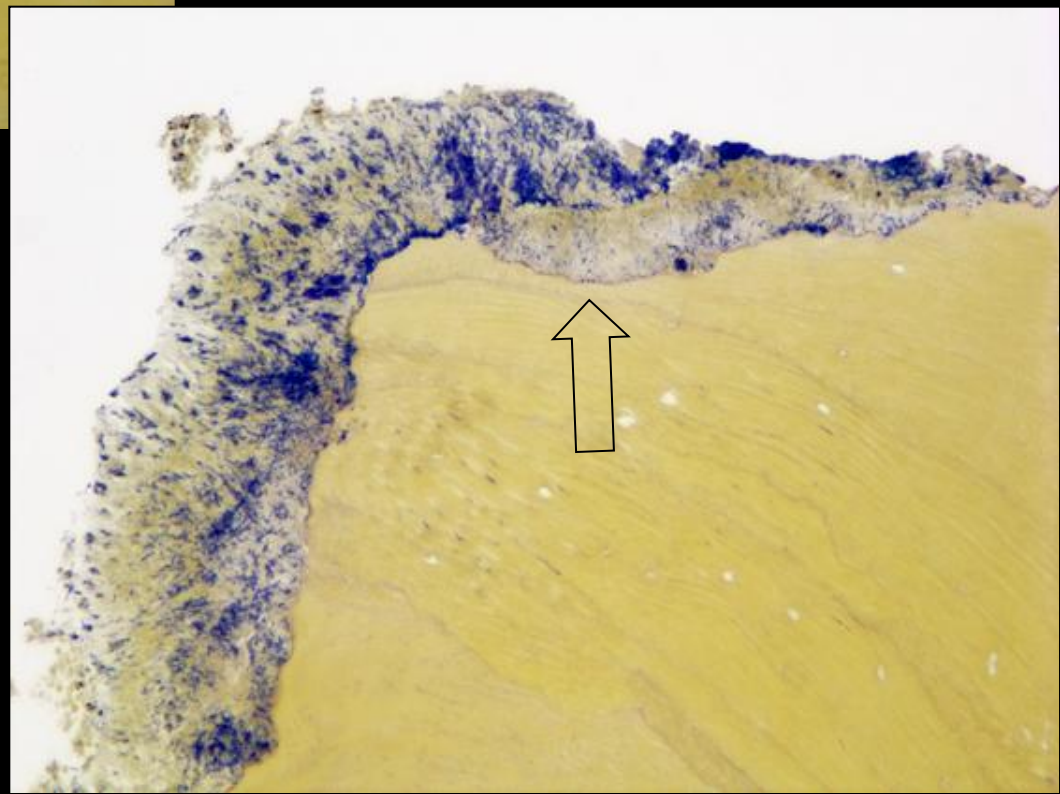

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CASE REPORT

Calculus-like deposit on the apical external root surface of teeth with post-treatment apical periodontitis: report of two cases

D. Ricucci[1], M. Martorano[2], A. L. Bate[3] & E. A. Pascon[4]

[1]Private Practice, Rome, Italy; [2]Private Practice, Sala Consilina, Italy; [3]Private Practice, Cuneo, Italy; and [4]Department of Endodontics, Faculty of Dentistry, University of Toronto, Toronto, Ontario, Canada

Abstract

Ricucci D, Martorano M, Bate AL, Pascon EA. Calculus-like deposit on the apical external root surface of teeth with post-treatment apical periodontitis: report of two cases. *International Endodontic Journal*, **38**, 262–271, 2005.

Aim To report two cases in which calculus-like material was found on external root surfaces of (i) an extracted root and (ii) an apicected part of a root, both of which were removed due to post-treatment refractory apical periodontitis.

Summary In each case, there was a fistulous tract, which did not heal after conventional root canal treatment. The first case did not heal even after apical surgery, and subsequent tooth extraction revealed calculus-like material on a root surface of complex anatomy. The second case showed radiographic signs of healing after apicectomy. Histology of the apical biopsy revealed a calculus-like material on the external surface of the root apex. It is suggested that the presence of calculus on the root surfaces of teeth with periapical lesions may contribute towards the aetiology of failure.

Key learning points

- Biofilm on the external root surface has been implicated in the failure of apical periodontitis to heal, despite adequate root canal treatment.
- Calculus-like material was found, in two cases, on the root surface of teeth with post-treatment apical periodontitis, where the only communication externally was a sinus tract.

Keywords: apical periodontitis, calculus, endodontic failure, extraradicular infection.

Received 26 July 2004; accepted 1 December 2004

Introduction

The major aetiological factor for the development and the maintenance of apical periodontitis is the colonization of microorganisms (bacteria and fungi) in the root canal

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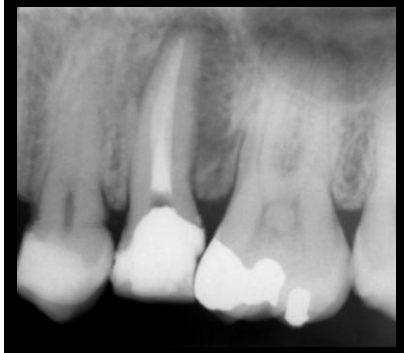

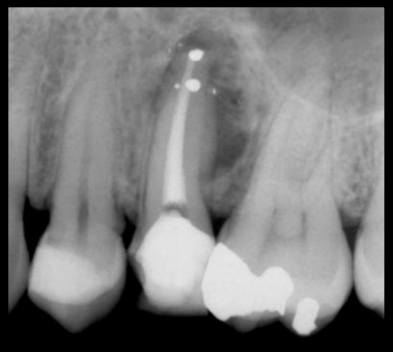

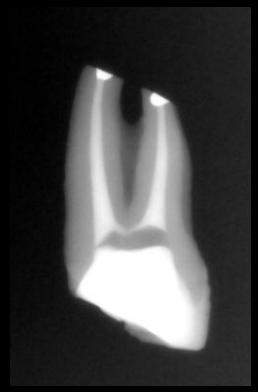

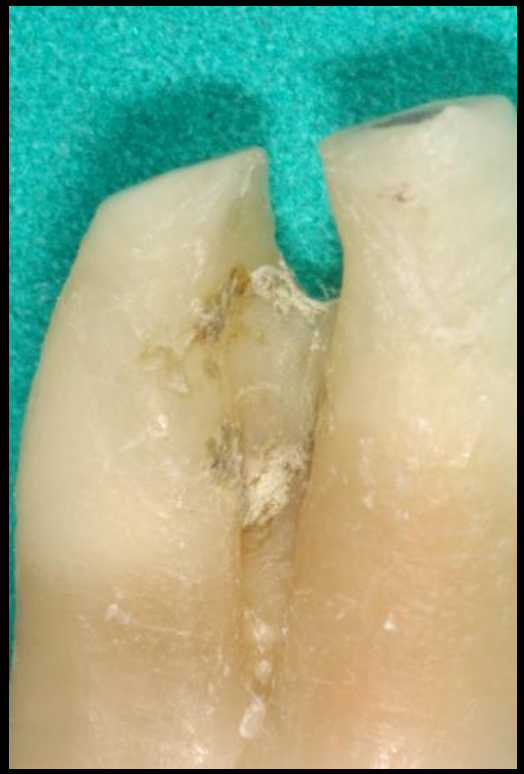

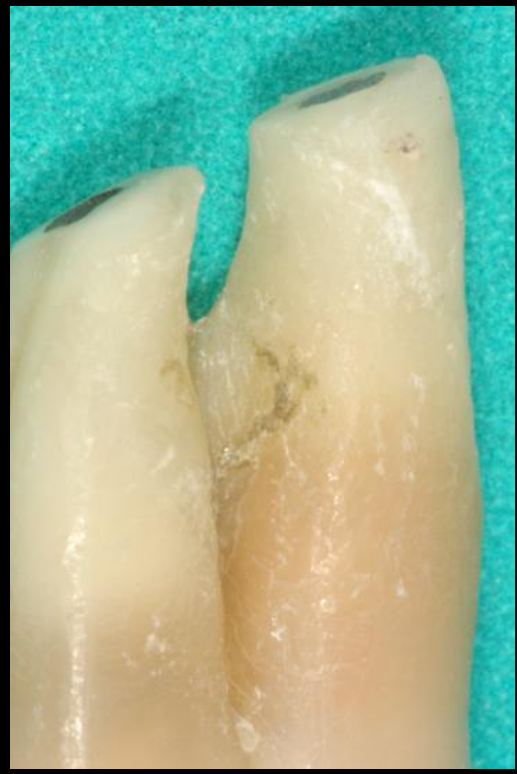

Extraradicular Infection as the Cause of Persistent Symptoms: A Case Series

Domenico Ricucci, MD, DDS, José F. Siqueira, Jr, DDS, MSc, PhD,† Weber S.P. Lopes, DDS, MSc, PhD,‡ Adalberto R. Vieira, DDS, MSc,‡ and Isabela N. Rôças, DDS, MSc, PhD†*

Abstract

Introduction: This article describes 3 cases that presented persistent symptoms after appropriate endodontic treatment. Histopathologic and histobacteriologic investigation were conducted for determination of the cause. **Methods:** Three cases are reported that presented with persistent symptoms after endodontic retreatment (cases 1 and 2) or treatment (case 3). Periapical surgery was indicated and performed in these cases. The biopsy specimens, consisting of root apices and the apical periodontitis lesions, were subjected to histopathologic and histobacteriologic analyses. **Results:** Case 1 was an apical cyst with necrotic debris, heavily colonized by ramifying bacteria, in the lumen. No bacteria were found in the apical root canal system. Case 2 was a granuloma displaying numerous bacterial aggregations through the inflammatory tissue. Infection was also present in the dentinal tubules at the apical root canal. Case 3 was a cyst with bacterial colonies floating in its lumen; bacterial biofilms were also seen on the external apical root surface, filling a large lateral canal and other apical ramifications, and between layers of cementum detached from the root surface. No bacteria were detected in the main root canal. **Conclusions:** Different forms of extraradicular infection were associated with symptoms in these cases, leading to short-term endodontic failure only solved by periapical surgery. (*J Endod 2015;41:265–273*)

Key Words

Endodontic retreatment, extraradicular infection, post-treatment apical periodontitis, treatment outcome

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0099-2399/$ - see front matter

http://dx.doi.org/10.1016/j.joen.2014.08.020

Post-treatment apical periodontitis is usually caused by persistent or secondary intraradicular infections (1, 2). It has also been suggested that infection located beyond the confines of the root canal system, either in the form of a biofilm attached to the external root surface (3, 4) or as cohesive colonies present within the mass of the inflammatory lesion (5, 6), may be responsible for post-treatment disease in some cases. One of the most debatable issues in the field of endodontic microbiology is whether or not infection can establish itself outside the canal system (except for abscess cases) and as such be the independent cause of post-treatment apical periodontitis.

Culture-dependent (7–11) and culture-independent studies (12–16) have reported the extraradicular occurrence of a complex microbiota associated with apical periodontitis lesions that have not responded favorably to the root canal treatment. One important discussion on this topic refers to whether contamination can be effectively ruled out during surgical sampling of apical periodontitis lesions for microbiological analysis. Bacteria located in the very apical part of the canal may be displaced into the biopsy specimen during surgical procedures and be regarded as "extraradicular" bacteria by culture and molecular studies (17). Most previous studies have not evaluated the bacteriologic conditions of the apical part of the root canal, making it difficult to ascertain whether the extraradicular infection was dependent on or independent of an intraradicular infection (18). As a consequence, there is no sufficient evidence supporting that an extraradicular infection can exist as a self-sustained process independent of the intraradicular infection (19). In a histologic study, Ricucci et al (20) evaluated several root canal–treated teeth with apical periodontitis and found no case of independent extraradicular infection. In the few instances that bacteria were observed outside the root canal system, a concomitant intraradicular infection was present.

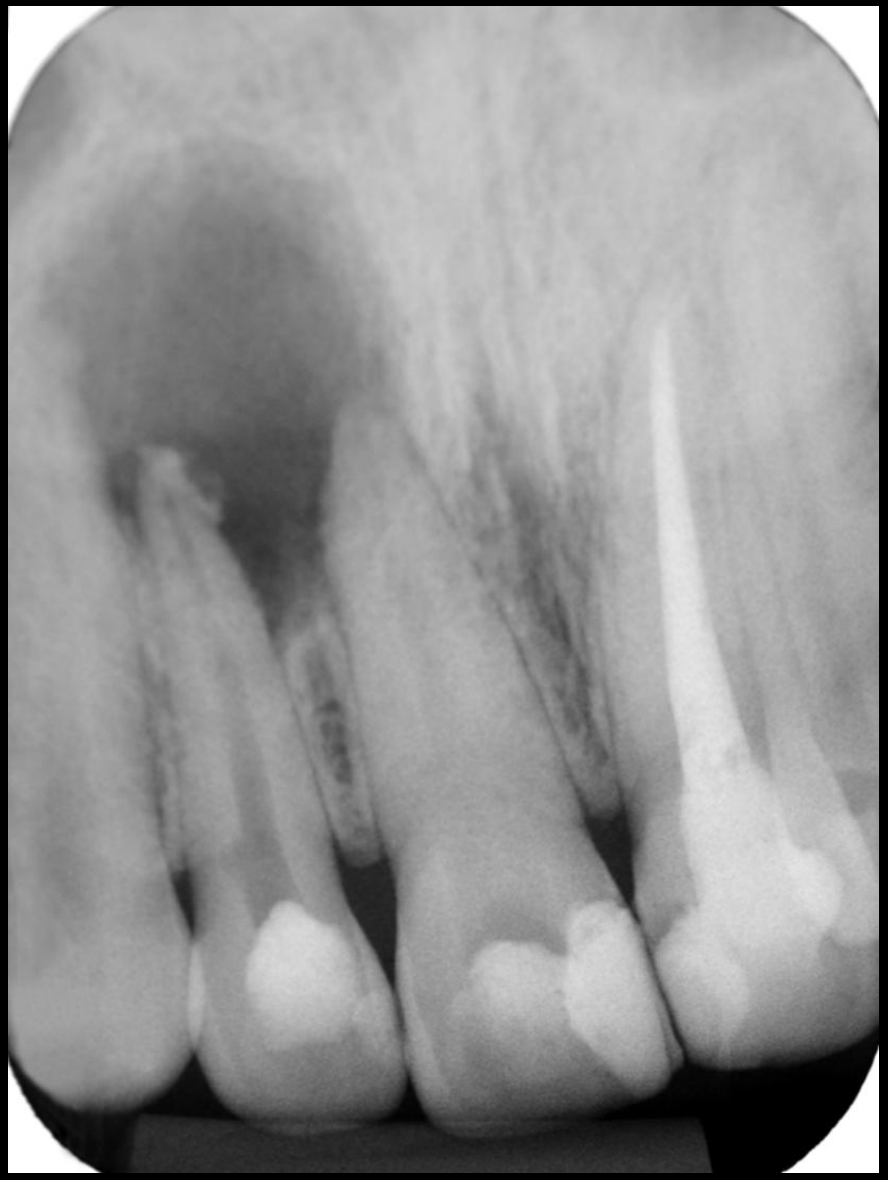
Histologic analysis of block specimens composed of the lesion attached to the root apex in their original spatial relationship can circumvent most of the shortcomings of previous studies because it may permit one to distinguish infection from contamination, detect artifactual bacterial displacement into the lesion, and reveal the microbiological conditions of the apical part of the root canal. This article is intended to contribute to the knowledge of the causes of endodontic treatment failure by reporting 3 cases of post-treatment apical periodontitis showing persisting symptoms associated with different types of extraradicular infection.

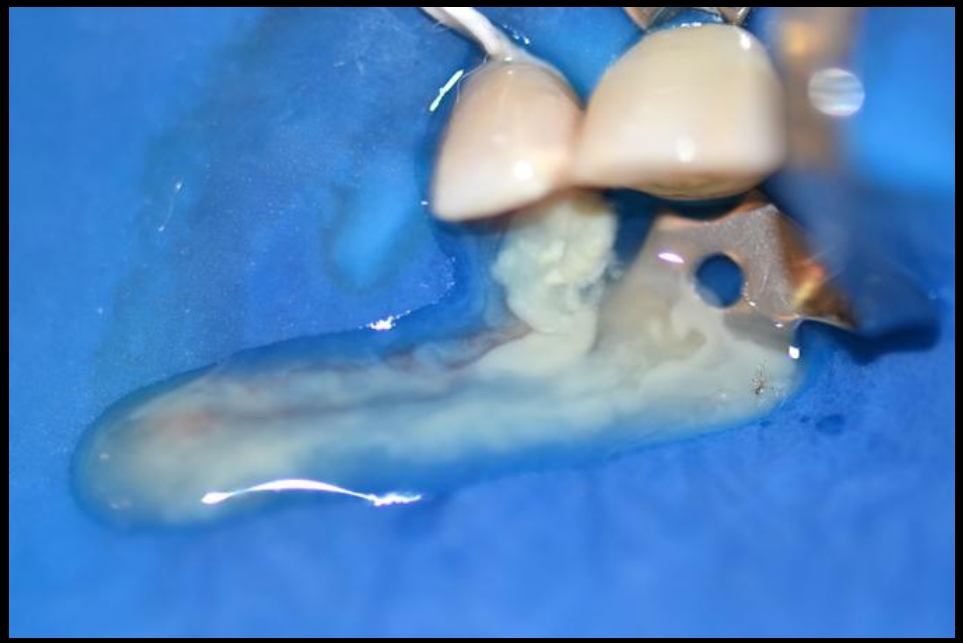
Case Series

Case 1

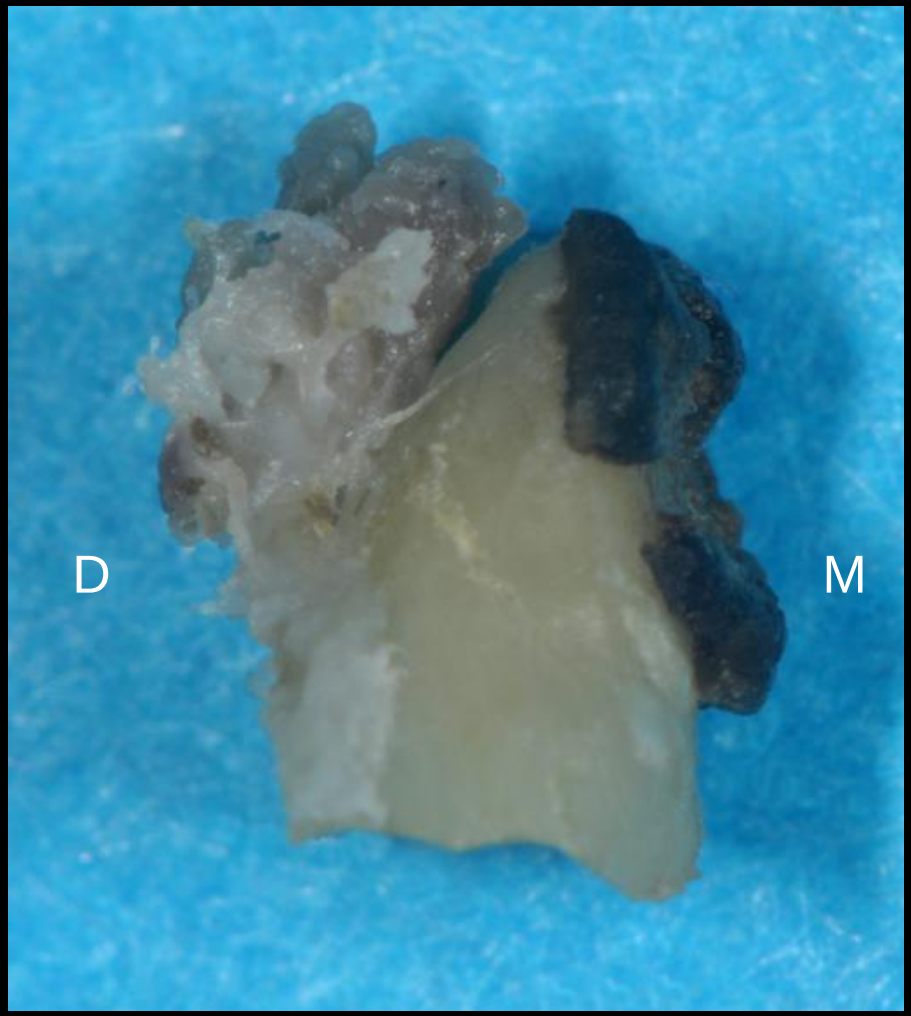
This case relates to a 35-year-old man with a history of repeated abscesses in the anterior mandible with severe pain and swelling. The medical history was non-contributory. His general dentist had initiated treatment of the 2 mandibular central incisors, which had necrotic pulps and were associated with a large periapical radiolucency. The canals (1 per tooth) were instrumented and medicated with an iodoform-based paste. Clinical symptoms did not recede even after 2 sessions of instrumentation and intracanal medication with calcium oxide. At this point (6 months after beginning of the treatment), the lesion had increased in size (Fig. 1A), and the patient was then referred to an endodontist.

At the first visit with the endodontist, signs of severe attrition were noted for all the anterior teeth. The patient denied any acute traumatic event. The access cavities on both





Ricucci D, Candeiro GTM, Bugea C, Siqueira JF Jr. Complex apical intraradicular infection and extraradicular mineralized biofilms as the cause of wet canals and treatment failure: report of two cases. J Endod 2016. In press.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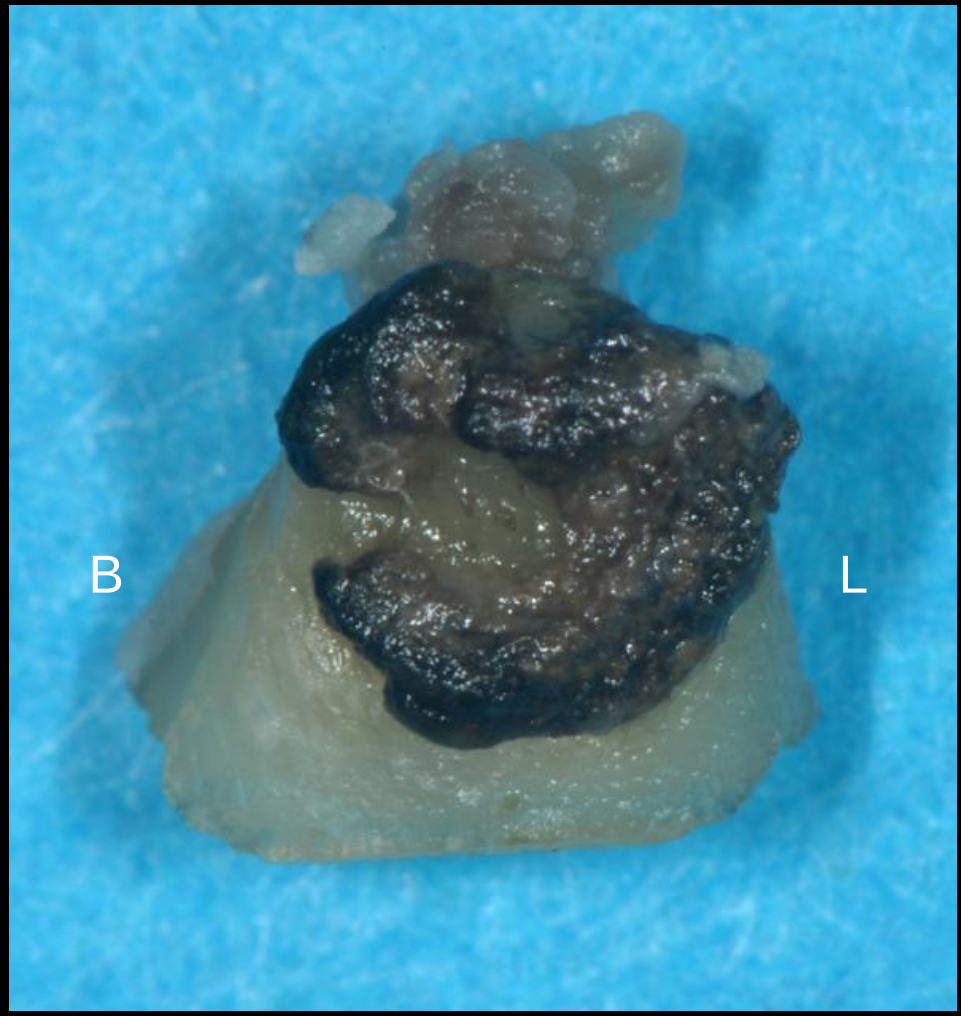
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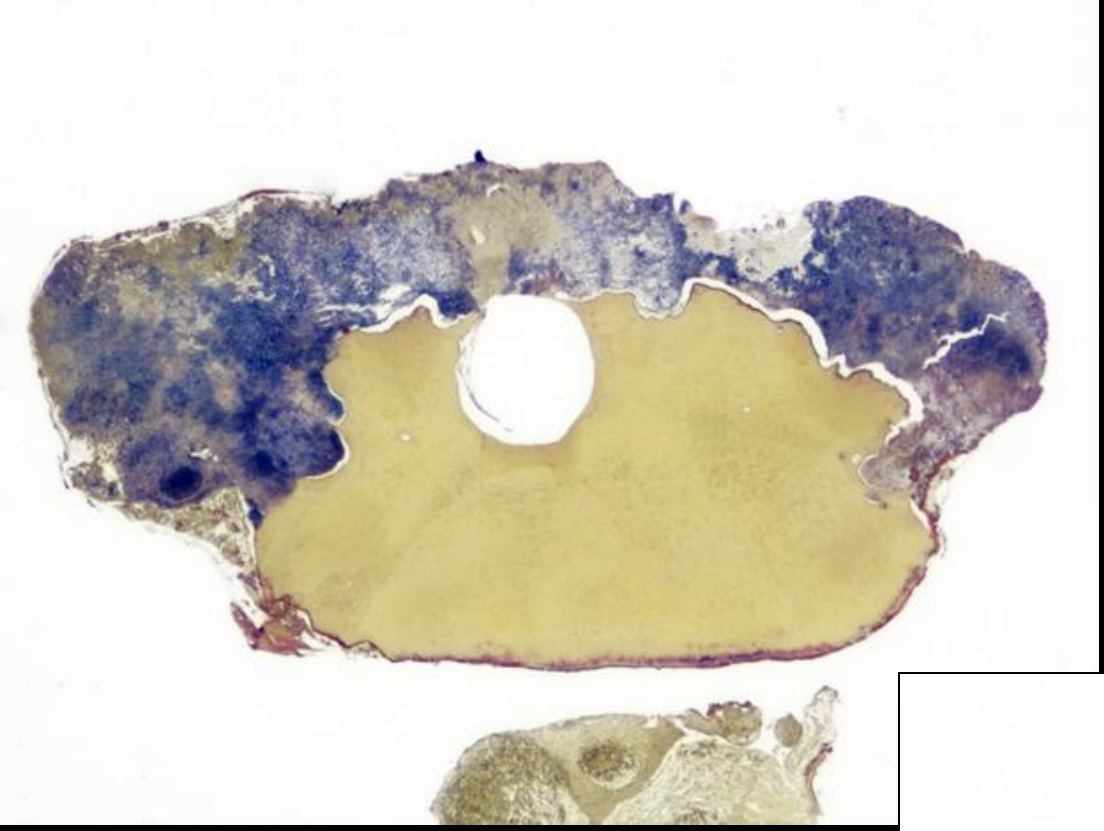

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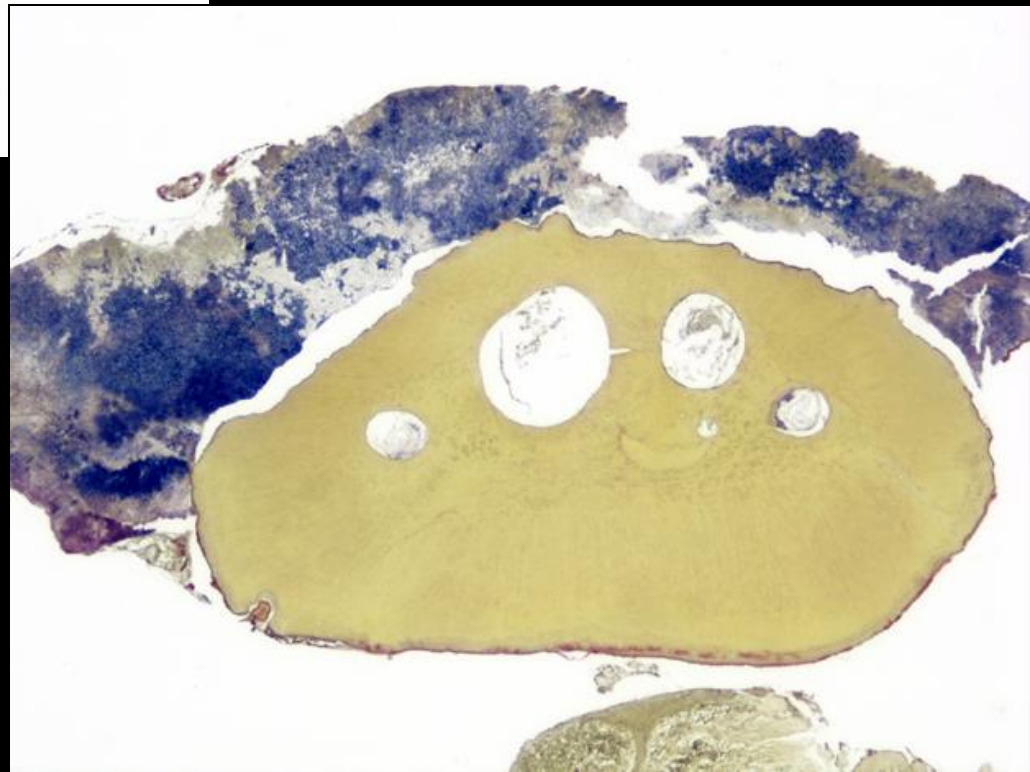

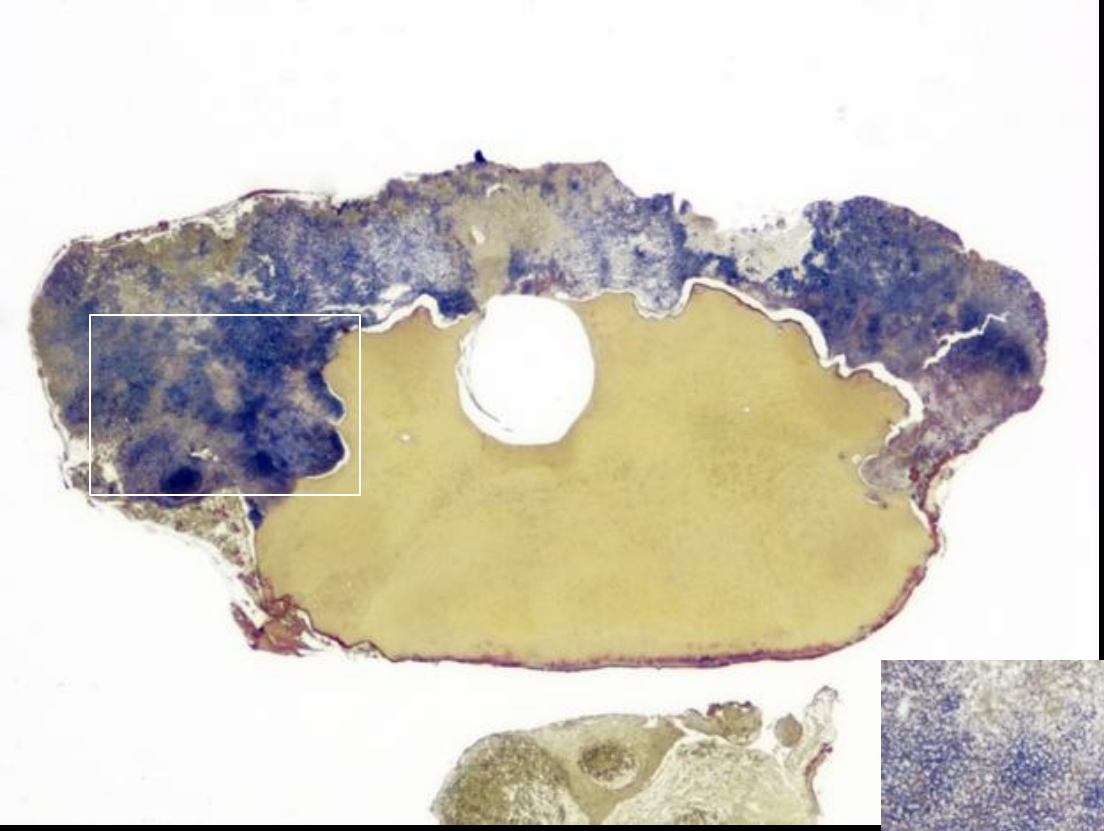

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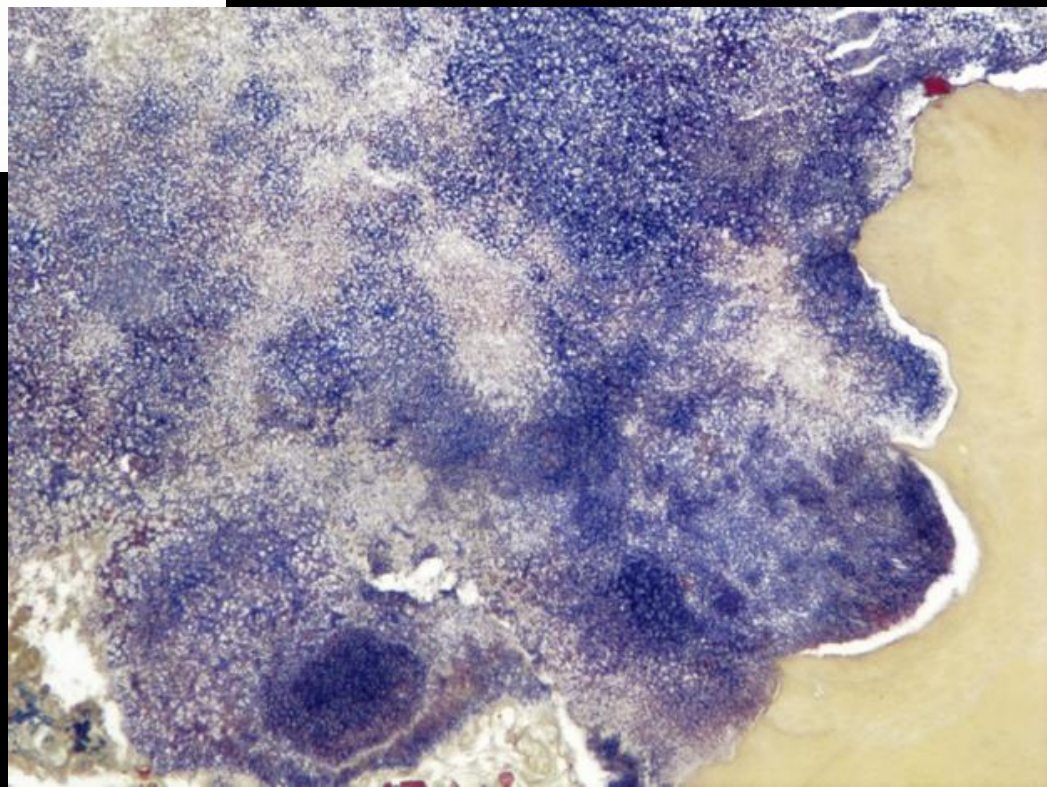

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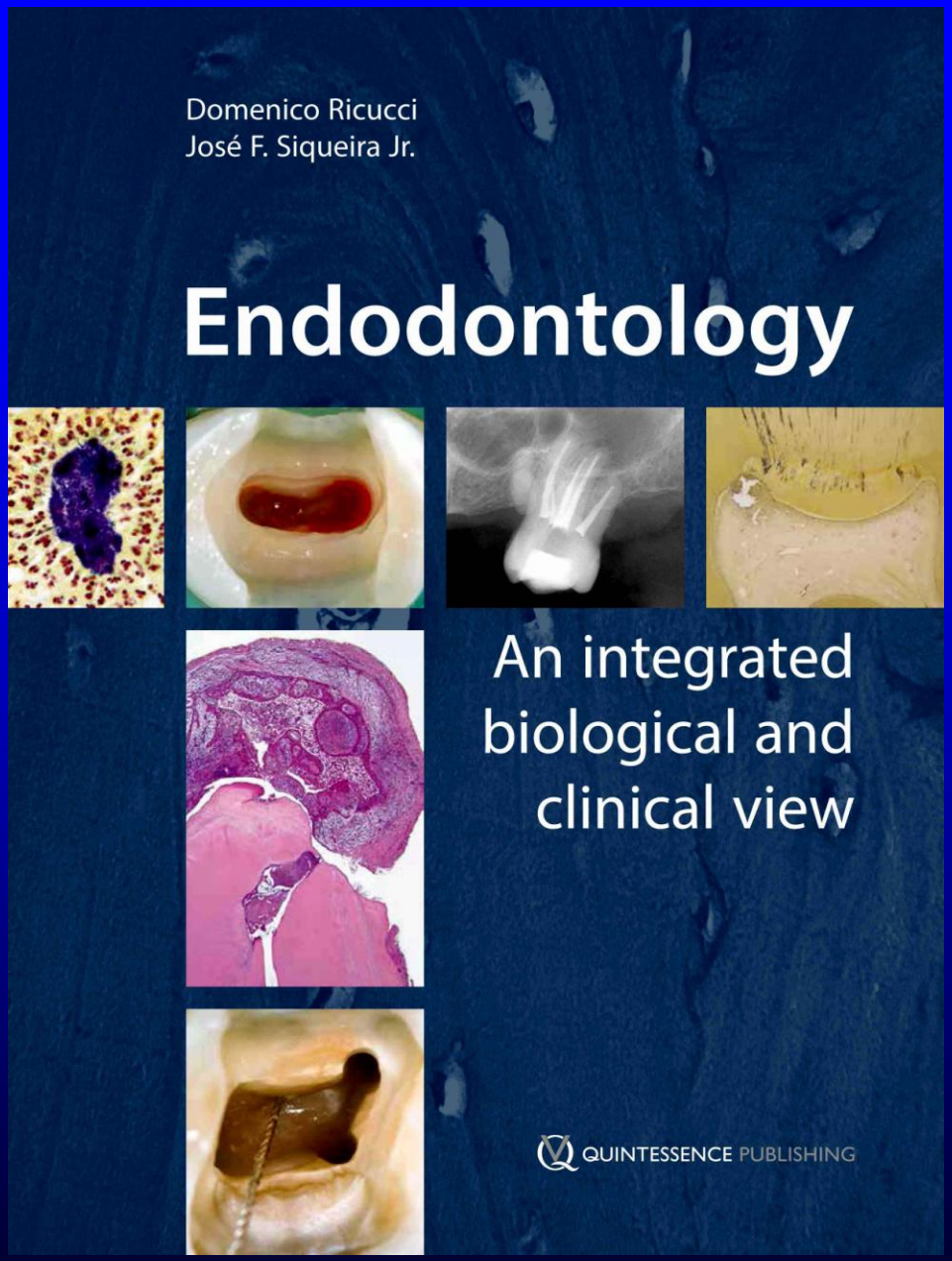

Thank you

for your attention

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