ORDINE PROVINCIALE
DEI MEDICI CHIRURGHI
E DEGLI ODONTOIATRI DI
BOLOGNA

Provider Nazionale n. 3744

CUORE E PARODONTOPATIE

LA SALUTE ORALE ALLUNGA LA VITA

Mercoledì 18 febbraio 2026
ore 20:00 – 22:30

Sala Conferenze, Ordine Provinciale dei Medici-Chirurghi e degli Odontoiatri di Bologna
Via Giovanna Zaccherini Alvisi n. 4 – Bologna

Responsabile Scientifico: Maurizio Baroni

19.45-20.00	*Registrazione partecipanti*
20.00-20.10	*Presentazione del Convegno* **Maurizio Baroni** *Moderano* **Carlo Prati e Giuseppe Trisolino**
20.10-20.30	*La malattia parodontale e le Linee Guida Intersocietarie* **Marco Montevecchi**
20.30-20.50	*Aspetti clinici, terapeutici e patogenetici della gengivite e parodontite* **Claudio Cesari**
20.50-21.10	*Malattie cardiovascolari e parodontopatie: quale associazione* **Maurizio Baroni**
21.10-21.30	*Ipertensione arteriosa e parodontite: approccio clinico* **Renzo Roncuzzi**
21.30-22.00	*Discussione*
22.00-22.30	*Questionario ECM e qualità percepita*

Destinatari dell'attività formativa: Medici-Chirurghi (Medico Generico e tutte le discipline) - Odontoiatri
Obiettivo formativo: contenuti tecnico-professionali (conoscenze e competenze) specifici di ciascuna professione, di ciascuna specializzazione e di ciascuna attività ultraspecialistica, ivi incluse le malattie rare e la medicina di genere (18) - Crediti ECM attribuiti: n. 2

Segue Faculty

Partecipazione gratuita previa iscrizione telematica sul sito www.odmbologna.it
Apertura iscrizioni il 29/01/2026 ore 11:00 - Chiusura iscrizioni il 16/02/2026
Segreteria organizzativa: ecm@odmbologna.it

ASPETTI CLINICI, TERAPEUTICI E PATOGENETICI DELLA GENGIVITE E DELLA PARODONTITE :

la salute orale allunga la vita

Dr. **Claudio Cesari, DDS, MSC (perio), MSC (osa), PhD student**

Adjunct Professor in Periodontology
Dept. Periodontology, Prof. Roberto Rotundo
University Vita e Salute SAN RAFFAELE, MILAN
Rettore Prof. Enrico Gherlone

Head of Periodontal UNIT
OSPEDALE SAN ROCCO, Ome (BS)
Dept. Oral and Maxillo-Facial,
Prof. Giorgio Gastaldi

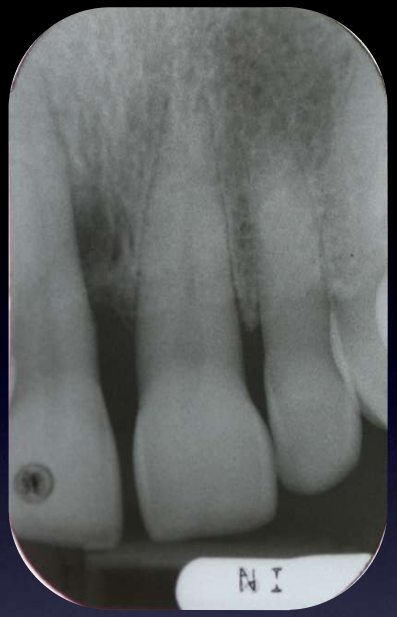
Ordinary member of SIdP since1997

DentalSchool UniSR

UNIVERSITA' VITA E SALUTE
SAN RAFFAELE (MILANO)

Corso di Laurea Magistrale
in Igiene Dentale
Perio UNIT PROF. ROTUNDO

PROFESSOR
ENRICO GHERLONE



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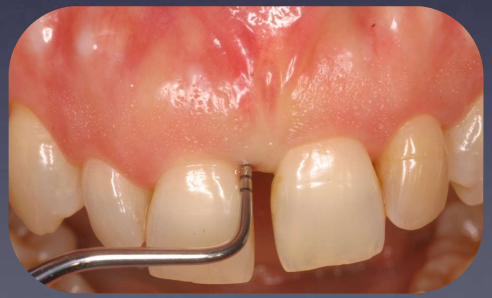
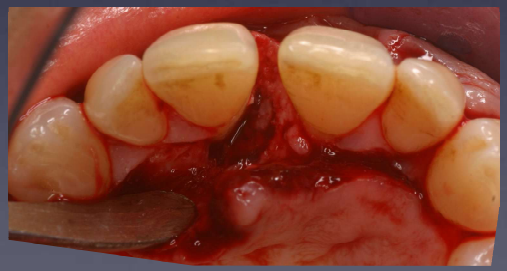
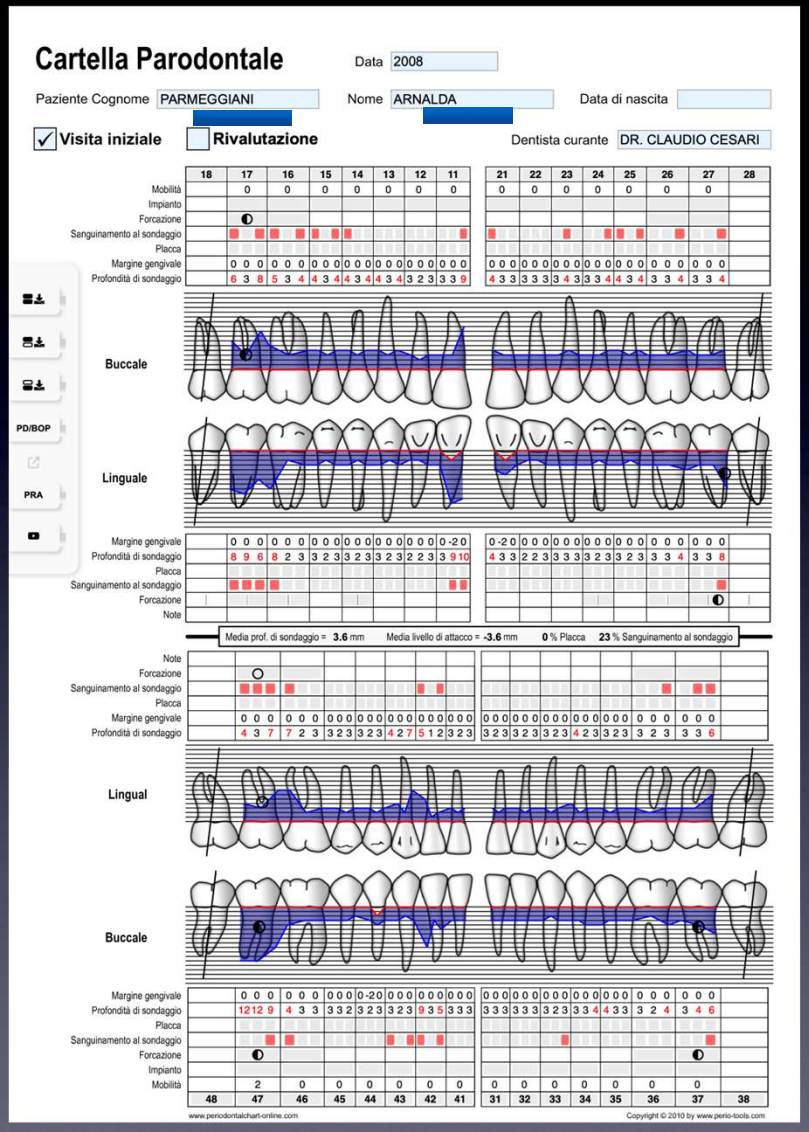
Perio Charting







PAZIENTE AFFETTO DA MALATTIE CV:

1- **Pericolosità di diverse manovre di igiene orale professionale, nonché terapie chirurgiche semplici o complesse in pazienti portatori di protesi valvolare, by-pass aorto-coronarici e con valvulopatie.**

2- Influenza dei diversi farmaci antiaggreganti, anticoagulanti e numerosi altri sui tessuti gengivali e parodontali nelle manovre odontoiatriche generiche o che prevedano profuso sanguinamento (STEP 2-STEP3). Il tessuto connettivo attorno agli elementi dentari ed agli impianti é infatti riccamente vascolarizzato.

3- **Rischio elevato di incidenti o complicazioni per il paziente nell'uso di anestetici ed altri farmaci a base di vasocostrittori nel caso di terapie su cardiopatici o pazienti con insufficienze organiche.**

Recommendations for antibiotic prophylaxis in patients with cardiovascular diseases undergoing oro-dental procedures at increased risk for IE (1)

Recommendations	Class	Level
General prevention measures are recommended in individuals at high and intermediate risk for IE.	I	C
Antibiotic prophylaxis is recommended in patients with previous IE.	I	B
Antibiotic prophylaxis is recommended in patients with surgically implanted prosthetic valves and with any material used for surgical cardiac valve repair.	I	C
Antibiotic prophylaxis is recommended in patients with transcatheter implanted aortic and pulmonary valvular prostheses.	I	C
Antibiotic prophylaxis is recommended in patients with untreated cyanotic CHD, and patients treated with surgery or transcatheter procedures with post-operative palliative shunts, conduits, or other prostheses. After surgical repair, in the absence of residual defects or valve prostheses, antibiotic prophylaxis is recommended only for the first 6 months after the procedure.	I	C

www.escardio.org/guidelines

2023 ESC Guidelines for the management of endocarditis (European Heart Journal; 2023 – doi: 10.1093/eurheartj/ehad193)

Recommendations for antibiotic prophylaxis in patients with cardiovascular diseases undergoing oro-dental procedures at increased risk for IE (2)

Recommendations	Class	Level
Antibiotic prophylaxis is recommended in patients with ventricular assist devices.	I	C
Antibiotic prophylaxis should be considered in patients with transcatheter mitral and tricuspid valve repair.	IIa	C
Antibiotic prophylaxis may be considered in recipients of heart transplant.	IIb	C
Antibiotic prophylaxis is not recommended in other patients at low risk for IE.	III	C

www.escardio.org/guidelines

2023 ESC Guidelines for the management of endocarditis (European Heart Journal; 2023 – doi: 10.1093/eurheartj/ehad193)

gentile concessione DR. BARONI MAURIZIO

INTRODUZIONE

LE MALATTIE PARODONTALI ASSOCIATE A PLACCA

La malattie parodontali sono patologie che interessano le strutture di supporto dei denti **(Fig. 1)**.
Vengono comunemente distinte in gengivite e parodontite.

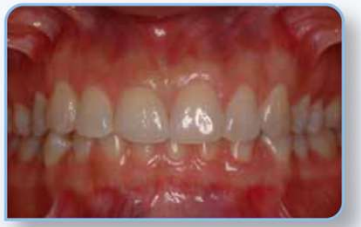


FIG. 1

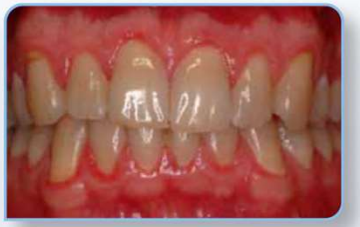


FIG. 2

La *gengivite* interessa la gengiva marginale ed è caratterizzata da arrossamento del margine gengivale, edema, sanguinamento al sondaggio e, talvolta, ipertrofia gengivale **(Fig. 2)**. È completamente reversibile e può precedere una parodontite. Secondo la nuova classificazione delle malattie parodontali del 2017, un paziente soffre di gengivite quando più del 10% dei siti presenta sanguinamento al sondaggio. Se la percentuale di siti è tra il 10% ed il 30% si parlerà di gengivite localizzata, altrimenti sarà una gengivite generalizzata.

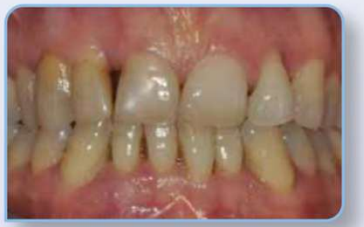


FIG. 3

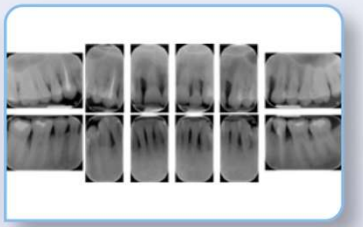


FIG. 4

La *parodontite* è una patologia caratterizzata dalla distruzione dell'apparato di supporto dei denti. Clinicamente si manifesta con perdita di attacco e di osso, formazione di tasche e talvolta formazione di recessioni **(Figg. 3 - 6)**.
Il segno patognomonico di parodontite è rappresentato dalla perdita di attacco.
La distruzione dei tessuti di sostegno dei denti causata da una parodontite è irreversibile.

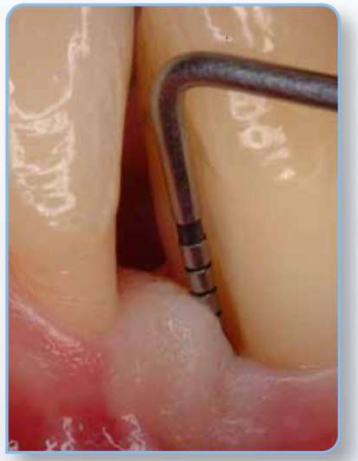


FIG. 5

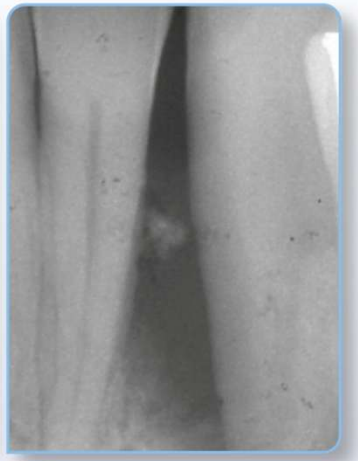
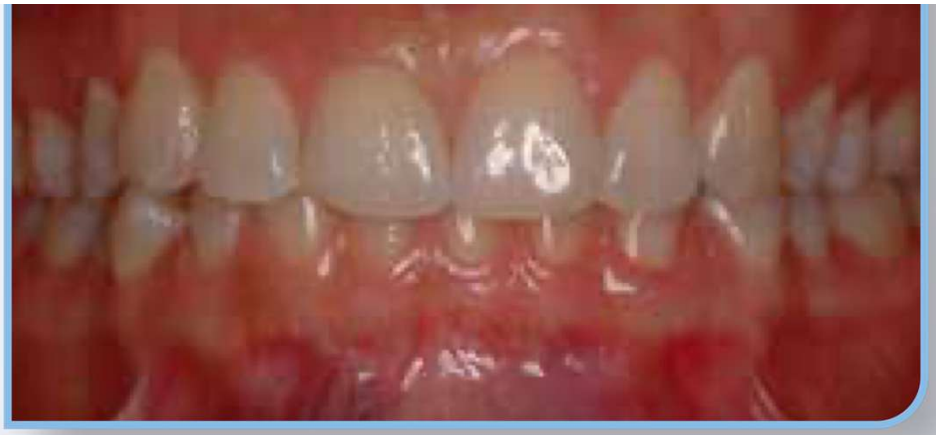


FIG. 6

Le parodontite, secondo la Federazione Europea di Parodontologia (EFP), come riportato nella classificazione del 2017, può essere classificata a seconda del grado di severità, di complessità, di distruzione e di rapidità di progressione. La classificazione prevede 4 stadi (stage), da 1 a 4 e 3 gradi (grade), da 1 a 3. L´estensione della malattia sarà generalizzata se colpisce più del 30% dei siti, oppure localizzata se meno o incisivi-molari se solo questi elementi sono coinvolti.



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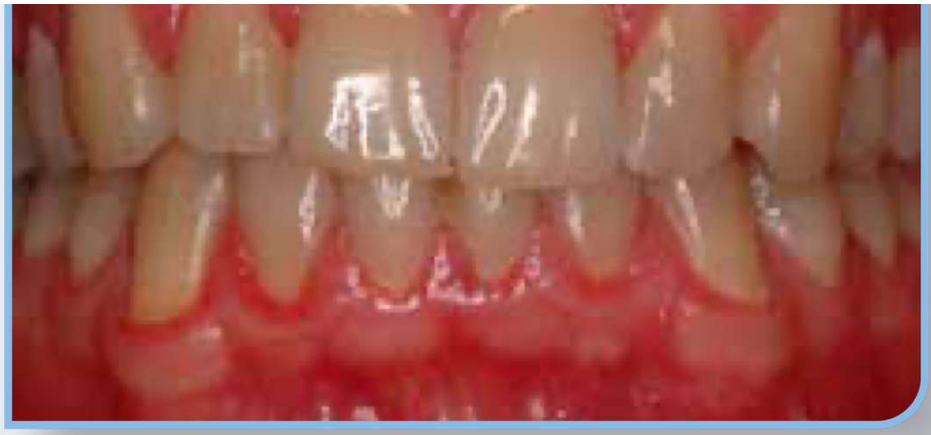

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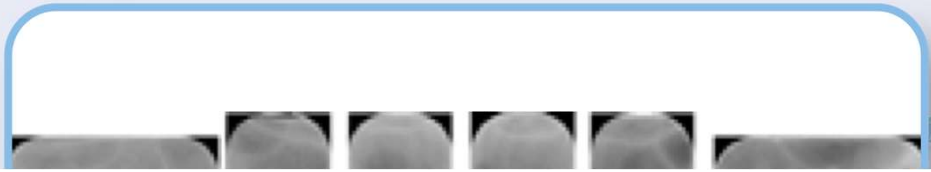


perplexity

Farmaci Coinvolti

I farmaci cardiovascolari più associati all'ipertrofia gengivale (o sovracrescita gengivale indotta da farmaci, DIGO) sono i calcio-antagonisti come nifedipina, amlodipina e diltiazem, usati per ipertensione e angina. La nifedipina è il più frequente, con prevalenza fino al 38% in alcuni pazienti, mentre amlodipina e diltiazem sono meno comuni (1-6%). Fattori di rischio includono sesso maschile, età giovane, infiammazione preesistente e placca batterica.[1] [2] [3] [4] [5] [6]

Meccanismo Patogenetico

Questi farmaci bloccano i canali del calcio voltaggio-dipendenti nelle cellule gengivali, riducendo l'ingresso intracellulare di Ca2+ e inibendo la produzione di collagenasi (enzimi che degradano il collagene). Ne risulta un accumulo di collagene extracellulare, aumento di glicosaminoglicani e proliferazione di fibroblasti gengivali, con infiltrati infiammatori (linfociti e plasmacellule). L'infiammazione da placca batterica aggrava il processo, favorendo la risposta citochinica pro-fibrotica; una teoria unificante coinvolge anche la ridotta captazione di acido folico nei fibroblasti.[2] [7] [6] [1]

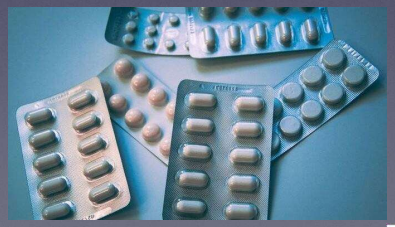
Gestione Clinica

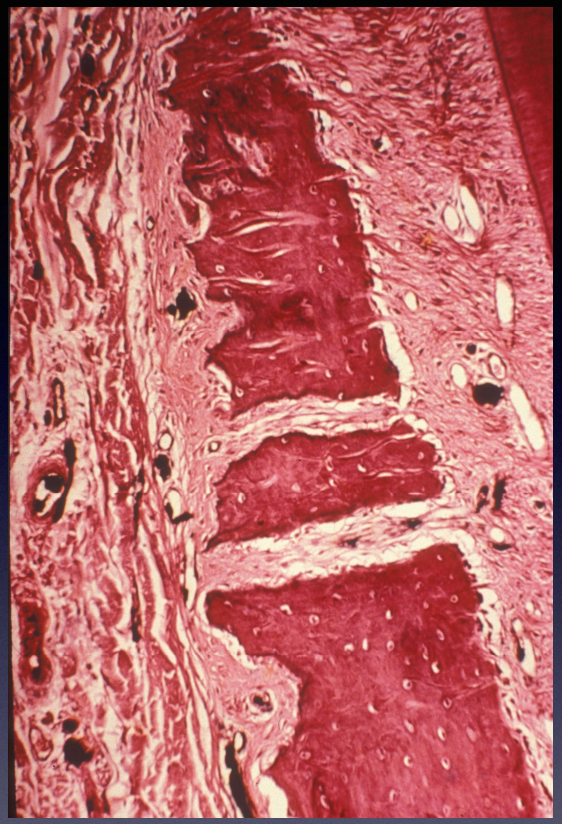
La risoluzione avviene spesso sospendendo o sostituendo il farmaco (es. con olmesartan), combinata con igiene orale professionale e chirurgia gengivale se necessario. Un approccio multidisciplinare tra cardiologo e odontoiatra è essenziale per bilanciare rischi cardiovascolari e orali.[3] [8] [1]

1. https://www.farmacovigilanzasardegna.it/2025/11/28/ipertrofia-gengivale-indotta-da-antagonisti-del-calcio-un-caso-clinico-e-una-revisione-della-letteratura/
2. https://pmc.ncbi.nlm.nih.gov/articles/PMC8069521/
3. https://www.dottnet.it/articolo/27232/dalla-pillola-agli-antipertensivi-alcuni-farmaci-causano-gengiviti
4. https://pmc.ncbi.nlm.nih.gov/articles/PMC8099224/
5. https://www.msdmanuals.com/it/professionale/disturbi-dei-denti/patologie-parodontali/iperplasia-gengivale
6. https://pmc.ncbi.nlm.nih.gov/articles/PMC4520117/
7. https://pmc.ncbi.nlm.nih.gov/articles/PMC5241888/
8. https://www.gengive.org/2020/10/03/sovracrescita-gengivale-indotta-da-farmaci/
9. https://www.ildentistamoderno.com/lipertrofia-gengivale-indotta-da-farmaci-revisione-della-letteratura/
10. https://onlinelibrary.wiley.com/doi/10.1111/j.1600-051X.1996.tb02072.x

GENGIVITE IN PAZIENTE IN TERAPIA PER MALATTIE CV:

Influenza dei diversi farmaci antiaggreganti, anticoagulanti e numerosi altri, che vengono utilizzati in terapia per le numerose patologie CV, causano enormi conseguenze a carico dei tessuti gengivali e parodontali.





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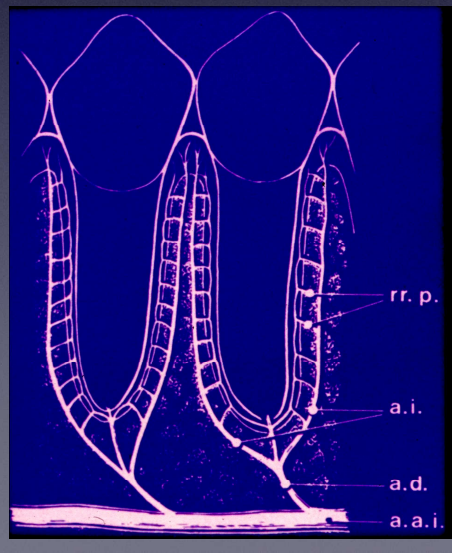
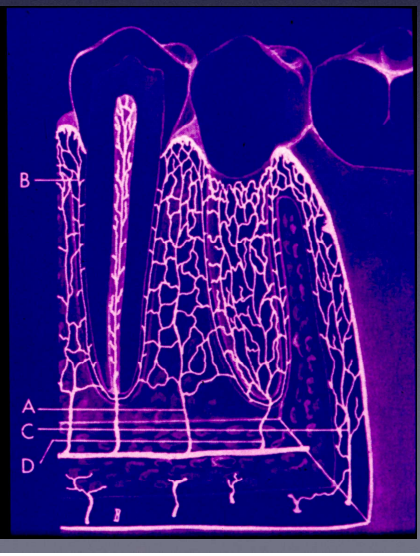
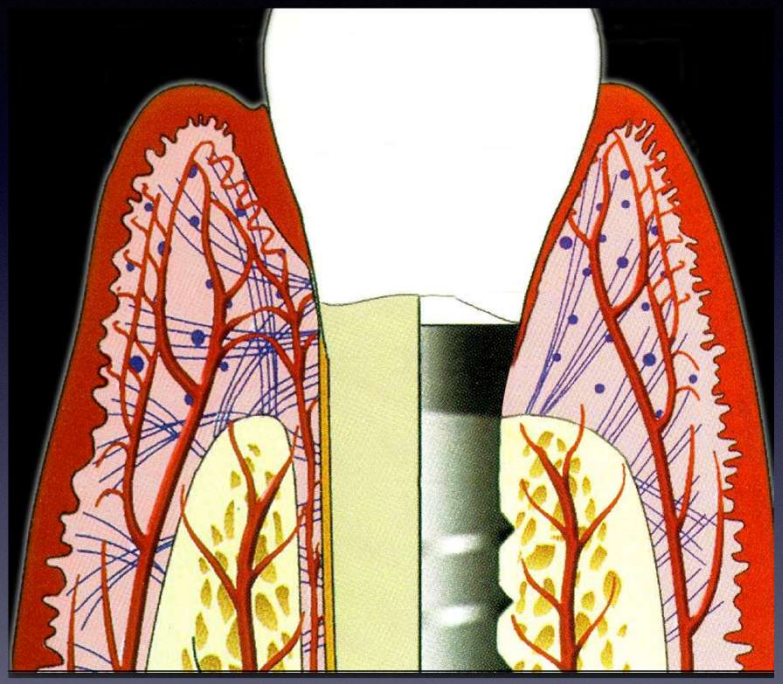




GENGIVITE IN PAZIENTE IN TERAPIA PER MALATTIE CV:

I tessuti connettivale ed epiteliali, infatti, sono particolarmente densi di connettivo e riccamente vascolarizzati sia attorno agli elementi dentari (affetti da GENGIVITE o PARODONTITE), sia attorno agli impianti dentari (MUCOSITE o PERI-IMPLANTITE).

Pertanto, nelle manovre odontoiatriche generiche o parodontali chirurgiche e non (STEP 2- STEP3) c'é un grosso pericolo di forte sanguinamento-emorragia, oltre che una notevole ed intensa diffusione per via ematica "diretta ed indiretta" sia di fattori infiammatori che di Microbioma orale e parodontale.

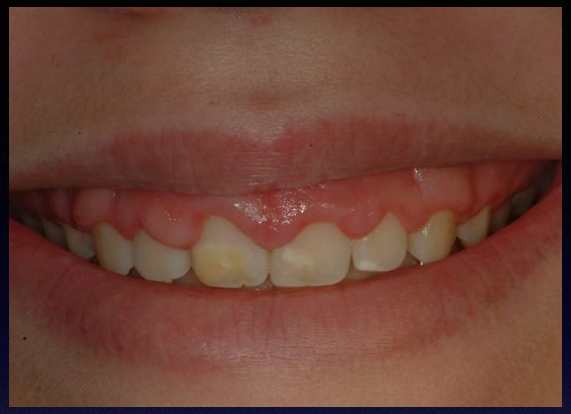


GENGIVITE IN PAZIENTE AFFETTO DA MALATTIE CV:

Influenza dei diversi farmaci antiaggreganti, anticoagulanti e numerosi altri, che vengono utilizzati in terapia per le numerose patologie CV, causano enormi conseguenze a carico dei tessuti gengivali e parodontali.

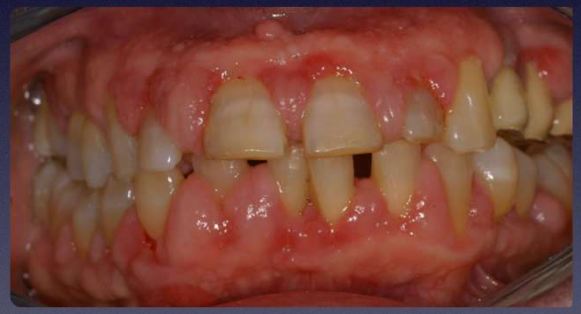
Pertanto, nelle manovre odontoiatriche generiche o che prevedano un "profuso sanguinamento" (STEP 2- STEP3) é necessario conoscerne gli effetti e le interazioni.

Il tessuto connettivale ed epiteliale, che sono particolarmente densi di connettivo e riccamente vascolarizzati sia attorno agli elementi dentari affetti da GENGIVITE o PARODONTITE, sia attorno agli impianti dentari se affetti da MUCOSITE o PERI-IMPLANTITE, hanno notevole pericolo di forte sanguinamento-emorragia, oltre che un ruolo importante nella diffusione per via ematica "diretta ed indiretta" di fattori infiammatori e Microbioma orale parodontale e non.



fenitoina

ciclosporina



beta_bloccanti

Ca+ antagonisti

f(x): ANTIANGINOSI (vasodilatatori), IPOTENSIVI, ANTIARITMICI, BETA-BLOCCANTI, CALCIO-antagonisti

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OPINION REVIEW

Drug-induced gingival overgrowth in cardiovascular patients

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Author contributions: Bajkovec L made contributions to the conception and design of the study, drafted and revised the manuscript critically; Mrzljak A, Alajbeg I and Likic R collected data, drafted and wrote the manuscript; all authors read and approved the final manuscript.

Conflict-of-interest statement: The authors declare no conflict of interest.

Manuscript source: Invited manuscript

Specialty type: Dentistry, oral

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Abstract

Drug-induced gingival overgrowth (DIGO) is a pathological growth of gingival tissue, primarily associated with calcium channel blockers and immunosuppressants. Consequently, it is mainly seen in cardiovascular and transplanted patients. Nifedipine remains the main calcium channel blocker related to the development of this unpleasant side-effect. As for immunosuppress-ants, cyclosporin is the leading causative agent, whereas other drugs from this drug-group, including tacrolimus, have better safety profiles. Accumulated collagen with inflammatory infiltrates is the histological hallmark of this condition. Several factors are involved in the pathogenesis and can increase the risk, such as male gender, younger age, pre-existing periodontal inflammation, and concomitant use of other DIGO-inducing medications. Patients with DIGO may experience severe discomfort, trouble with speech and mastication, pain, and teeth loss, aside from cosmetic implications. Furthermore, these patients also have an increased risk for cardiovascular diseases. The interdisciplinary approach and cooperation with dental care experts are necessary for patient management. Treatment includes discontinuing the drug and switching to one with a better profile, improving oral hygiene, and surgical removal of enlarged tissue. Recognizing the potential of commonly used medications to cause DIGO and its effect on patients' health is necessary for early detection and adequate management of this complication.

Key Words: Drug-induced gingival overgrowth; Calcium channel blocker; Nifedipine;

Figure 1 A complete response of a severe drug-induced gingival overgrowth case following seven weeks after amlodipine removal and six consecutive tooth scaling and cleaning treatments. A: Amlodipine induced gingival overgrowth successfully treated by vigorous weekly plaque control; B: Calculus removal during seven weeks following the drug withdrawal and substitution by angiotensin-converting enzyme inhibitor (courtesy of Prof. Vlaho Brailo).

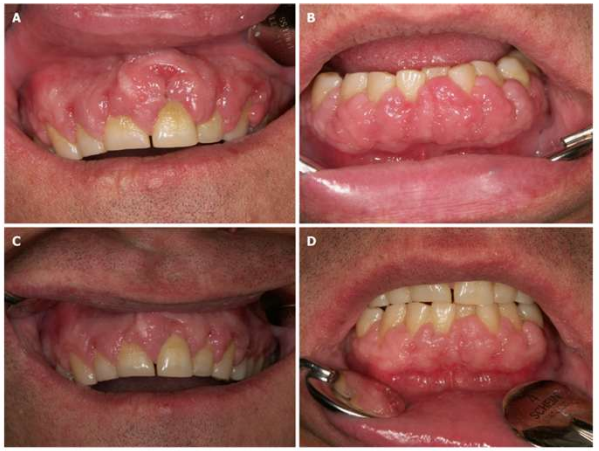


Figure 2 An improvement in a heart transplant patient, whose medication included both cyclosporin and amlodipine, four weeks following professional teeth cleaning and switching to tacrolimus and alternative antihypertensive drug. A and B: Gingival overgrowth in a heart transplant patient receiving both cyclosporin and amlodipine; C and D: An improvement is observed following conservative periodontal treatment and four weeks of switching to tacrolimus and angiotensin-converting enzyme inhibitor.

managed and resolved with discontinuation of the inducing drugs[1].

f(x): ANTIANGINOSI (vasodilatatori),

IPOTENSIVI,

ANTIARITMICI,

BETA-BLOCCANTI,

ALFA-BLOCCANTI

CALCIO-antagonisti

PARODONTITE :

La *parodontite* è una patologia caratterizzata dalla distruzione dell'apparato di supporto dei denti. Clinicamente si manifesta con perdita di attacco e di osso, formazione di tasche e talvolta formazione di recessioni **(Figg. 3 - 6)**.
Il segno patognomonico di parodontite è rappresentato dalla perdita di attacco.
La distruzione dei tessuti di sostegno dei denti causata da una parodontite è irreversibile.

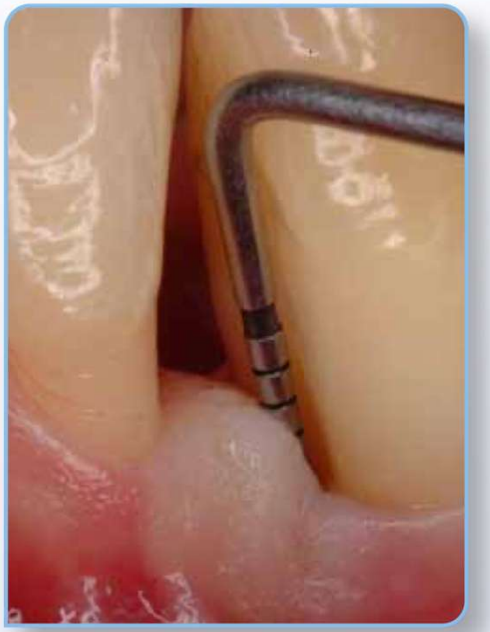


FIG. 5

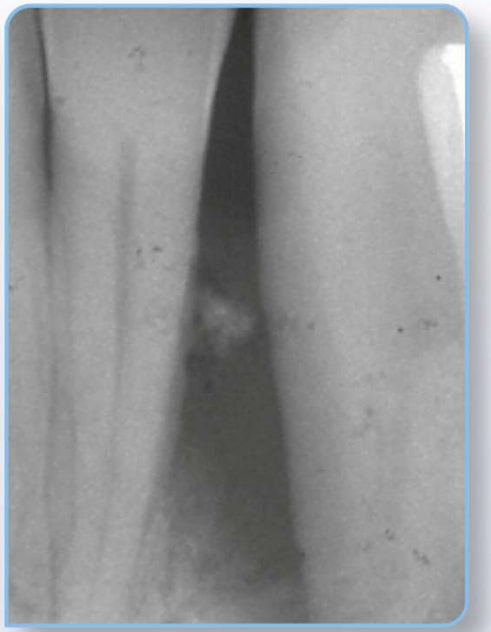


FIG. 6

Le parodontite, secondo la Federazione Europea di Parodontologia (EFP), come riportato nella classificazione del 2017, può essere classificata a seconda del grado di severità, di complessità, di distruzione e di rapidità di progressione. La classificazione prevede 4 stadi (stage), da 1 a 4 e 3 gradi (grade), da 1 a 3. L´estensione della malattia sarà generalizzata se colpisce più del 30% dei siti, oppure localizzata se meno o incisivi-molari se solo questi elementi sono coinvolti.

PARODONTITE :

FISIOPATOLOGIA:

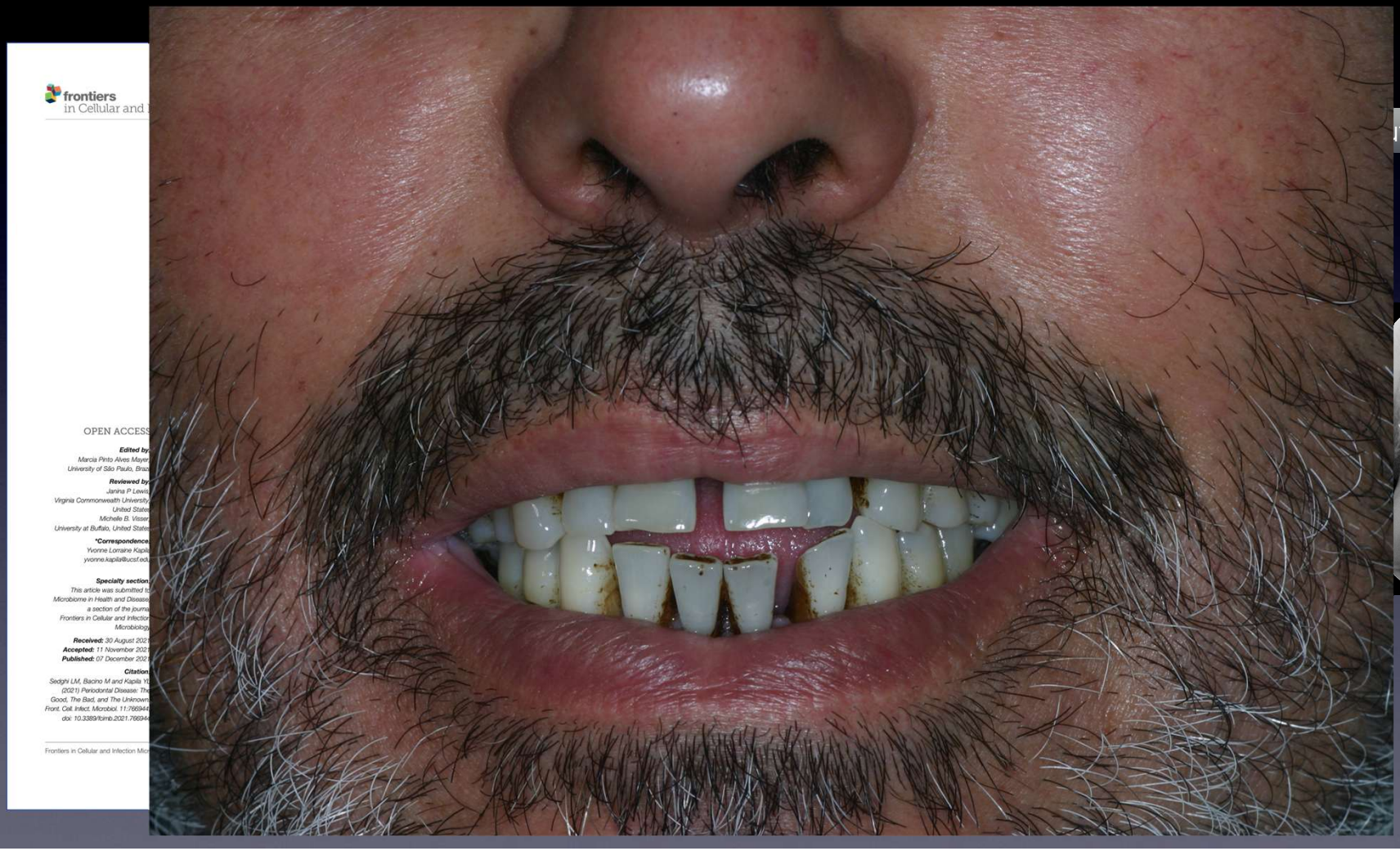
La parodontite è una malattia infiammatoria multifattoriale cronica associata a una disbiosi del biofilm che costituisce la placca dentale.

ASPETTI CLINICI:

Clinicamente le sue caratteristiche principali includono la perdita del tessuto di supporto parodontale, che si manifesta attraverso la perdita di attacco clinico (CAL), la presenza di tasche parodontali e di sanguinamento gengivale (Papapanou, et al. 2018), con il "successivo" riassorbimento di osso alveolare (verificata radiograficamente).
Se non trattata, può portare alla perdita dei denti, sebbene sia prevenibile e curabile nella maggior parte dei casi.

IMPORTANZA:
La parodontite è un grave problema di salute pubblica a causa della sua elevata prevalenza e, poiché può portare alla perdita e alla disabilità dei denti, influisce negativamente sulla funzione masticatoria e sull'estetica, è fonte di disuguaglianza sociale e compromette significativamente la qualità della vita. La parodontite è responsabiledi una parte sostanziale dell'edentulismo e della disfunzione masticatoria, ha un impatto negativo sulla salute generale e comporta costi significativi per cure odontoiatriche (Tonetti, et al. 2017).

DISBIOSYS - ENVIRONMENT



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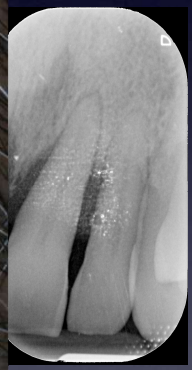

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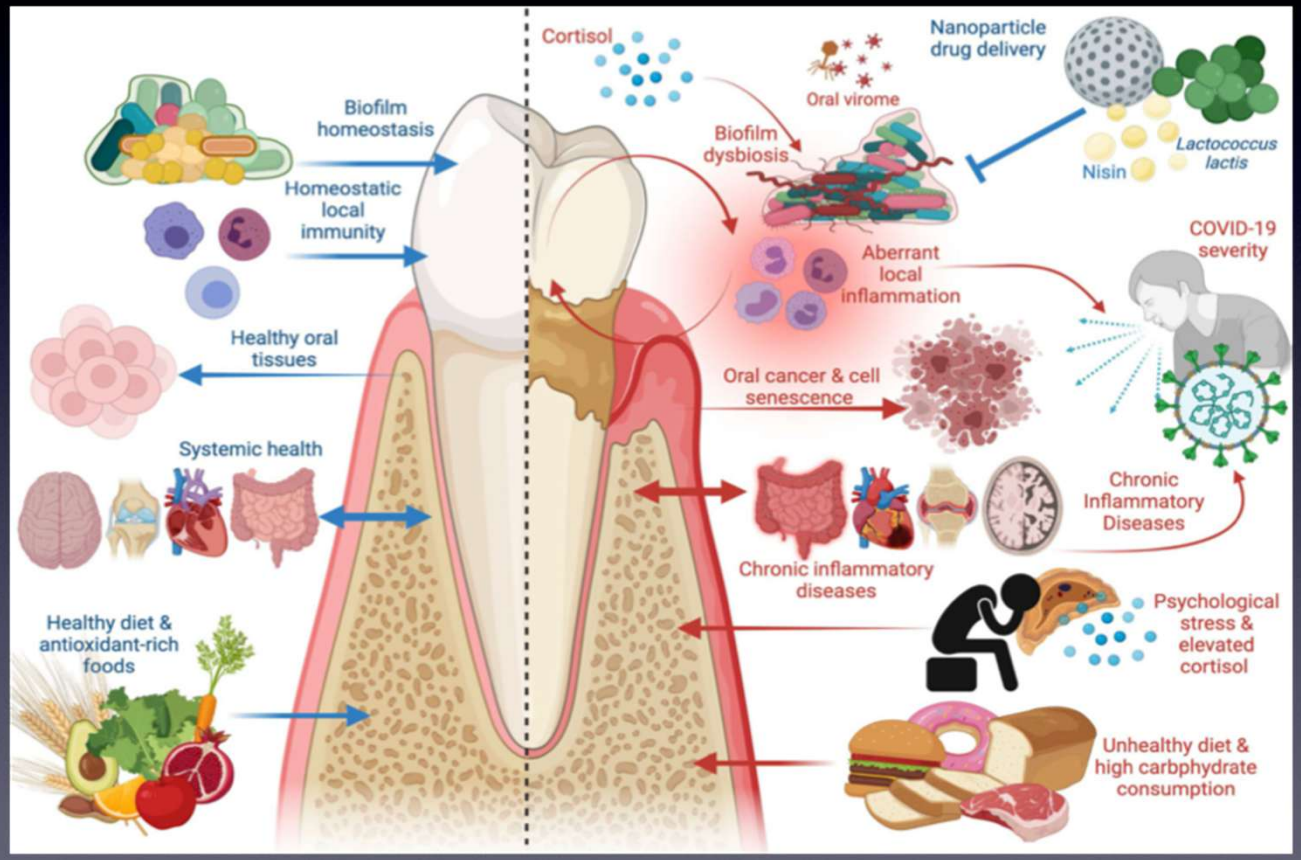
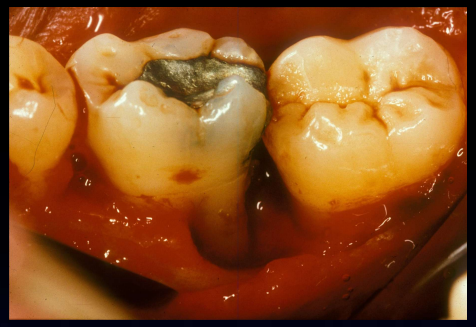
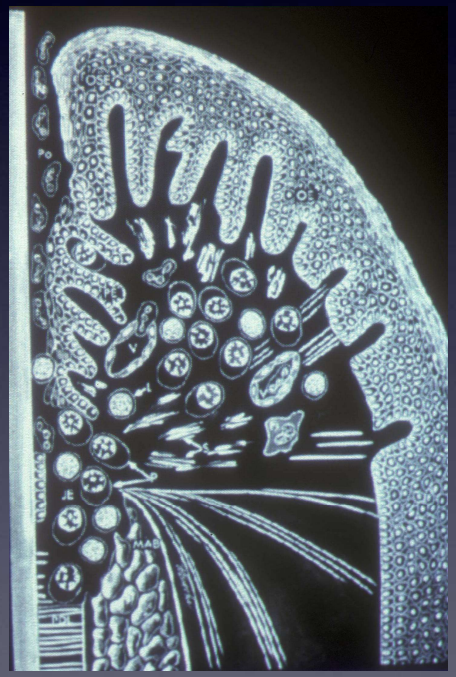
57 systemic diseases correlated







DISBIOSYS - ENVIRONMENT

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REVIEW
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Periodontal Disease: The Good, The Bad, and The Unknown

*Lea M. Sedghi[1,2], Margot Bacino[1,2] and Yvonne Lorraine Kapila[1,2,3]**

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Periodontal disease is classically characterized by progressive destruction of the soft and hard tissues of the periodontal complex, mediated by an interplay between dysbiotic microbial communities and aberrant immune responses within gingival and periodontal tissues. Putative periodontal pathogens are enriched as the resident oral microbiota becomes dysbiotic and inflammatory responses evoke tissue destruction, thus inducing an unremitting positive feedback loop of proteolysis, inflammation, and enrichment for periodontal pathogens. Keystone microbial pathogens and sustained gingival inflammation are critical to periodontal disease progression. However, recent studies have revealed the importance of previously unidentified microbes involved in disease progression, including various viruses, phages and bacterial species. Moreover, newly identified immunological and genetic mechanisms, as well as environmental host factors, including diet and lifestyle, have been discerned in recent years as further contributory factors in periodontitis. These factors have collectively expanded the established narrative of periodontal disease progression. In line with this, new ideologies related to maintaining periodontal health and treating existing disease have been explored, such as the application of oral probiotics, to limit and attenuate disease progression. The role of systemic host pathologies, such as autoimmune disorders and diabetes, in periodontal disease pathogenesis has been well noted. Recent studies have additionally identified the reciprocated importance of periodontal disease in potentiating systemi[illegible]s at distal sites, such as in Alzheimer's disease, inflammatory bowel disea[illegible]nd oral cancer, further highlighting the importance of the oral cavity in systemic hea[illegible]we review long-standing knowledge of periodontal disease progression while [illegible]vel research concepts that have broadened our understanding of periodontal hea[illegible]d disease. Further, we delve into innovative hypotheses that may [illegible]ve to a[illegible]s significant gaps in the foundational knowledge of periodontal disease.

Keywords: dysbiosis, host response, oral microbiome, periodontal pathobionts, oral-systemic association

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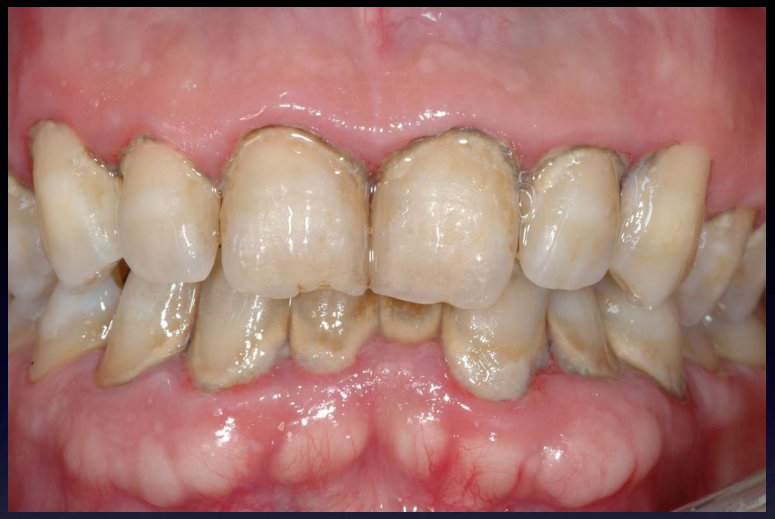

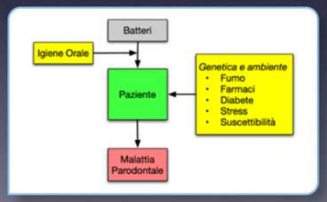

Attualmente la parodontite è la principale causa di edentulismo parziale e totale in Italia. In molti casi la parodontite non viene diagnosticata o solo molto tardivamente. La man[illegible] pre[illegible] anca[illegible] gra[illegible] la progres[illegible] distruzione c[illegible] sosteg[illegible] dei den[illegible] con [illegible] segue la compro[illegible]ione della fun[illegible]e mas[illegible]oria e c[illegible] aspetto e[illegible]co [illegible] non perdo degli el[illegible]
Inoltre c[illegible] sappiamo ch[illegible]ute può ra[illegible]esentare [illegible] pot[illegible]ile fatt[illegible] di rischi[illegible]er alcune pato[illegible]e sistemiche, qual[illegible]tteriemie[illegible]doc[illegible]ti acute [illegible] acute[illegible]atologie cardio[illegible]colari, infezioni di p[illegible]si sostitu[illegible], dia[illegible]e malat[illegible] respirato[illegible] al [illegible]o di nascita di bam[illegible] preterm[illegible] so[illegible] possono essere alla base di fenomeni ascessuali cerebrali e di altre patologie "focali".
Queste conoscenze sottolineano l'inderogabile necessità di diagnosticare precocemente la parodontite per instaurare le corrette procedure preventive e/o terapeutiche.
La parodontite in fatti può essere prevenuta e curata. La prevenzione e la terapia sono molto efficaci nella maggior parte dei casi.

SUSCEPTIBILITY

TABELLA 7. Criteri per la definizione degli stadi di parodontite. (da Tonetti, et al. 2018).

STADIO DI PARODONTITE		STADIO I	STADIO II	STADIO III	STADIO IV
Gravità	**CAL interdentale nel sito di massima perdita di attacco**	Da 1 a 2 mm	Da 3 a 4 mm	≥5 mm	≥5 mm
	Perdita ossea radiografica	Terzo coronale (<15%)	Terzo coronale (da15% a 33%)	Estesa al terzo medio o apicale della radice	Estesa al terzo medio o apicale della radice
	Perdita di denti	Nessuna perdita di denti dovuta alla parodontite		Perdita di denti dovuta alla parodontite ≤4 denti	Perdita di denti dovuta alla parodontite di ≥5 denti
Complessità	Locale			Oltre alla complessità dello Stadio II:	Oltre alla complessità dello Stadio III:
		Massima profondità di sondaggio ≤4 mm	Massima profondità di sondaggio ≤5 mm	profondità di sondaggio ≥6 mm	Necessità di riabilitazione complessa dovuta a:
		Perdita ossea prevalentemente orizzontale	Perdita ossea prevalentemente orizzontale	Perdita ossea verticale ≥3 mm	Disfunzione masticatoria Trauma occlusale secondario (mobilità dentale di grado ≥2) Gravi difetti delle selle Collasso del morso, spostamenti, sventagliamento. Meno di 20 denti rimasti (10 paia di denti contrapposti)
				Interessamento delle forcazioni di Classe II o III	
				Moderati difetti delle selle	
Estensione e distribuzione	**Aggiungere allo stadio come descrizione**	Per ogni fase, descrivere l'estensione come localizzata (> 30% dei denti coinvolti), generalizzata o rapporto incisivo/molare			

DIAGNOSI

Estensione e distribuzione	**Aggiungere allo stadio come descrizione**	Per ogni fase, descrivere l'estensione come localizzata (> 30% dei denti coinvolti), generalizzata o rapporto incisivo/molare

Lo stadio iniziale dovrebbe essere determinato usando il CAL; se non disponibile, utilizzare l'RBL. Le informazioni sulla perdita dei denti che possono essere attribuite principalmente alla parodontite – se disponibili – possono modificare la definizione di stadio. Questo è il caso anche in assenza di fattori di complessità. I fattori di complessità possono spostare lo stadio a un livello superiore, per esempio la classe di interessamento delle forcazioni II o III sposterebbe la classificazione a uno Stadio III o IV indipendentemente dal CAL. La distinzione tra Stadio III e Stadio IV si basa principalmente su fattori di complessità. Per esempio, un livello elevato di mobilità dei denti e/o collasso posteriore del morso indicherebbe una diagnosi di Stadio IV. Per ogni dato caso possono essere presenti solo alcuni, non tutti, i fattori di complessità, tuttavia, in generale, è sufficiente un solo fattore di complessità per spostare la diagnosi a uno stadio superiore. Va sottolineato che queste definizioni di casi sono linee guida che dovrebbero essere applicate usando un sano giudizio clinico per arrivare alla diagnosi clinica più appropriata.

Per i pazienti post-trattamento CAL e RBL sono ancora i principali fattori determinanti di stadio. Se il trattamento elimina un fattore di complessità in grado di spostare lo stadio, lo stadio non dovrebbe essere retrocesso a uno stadio inferiore poiché il fattore di complessità dello stadio originale dovrebbe essere sempre tenuto presente nella gestione della fase di mantenimento.

CAL – perdita di attacco clinico; RBL – perdita ossea radiografica.

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DIAGNOSI

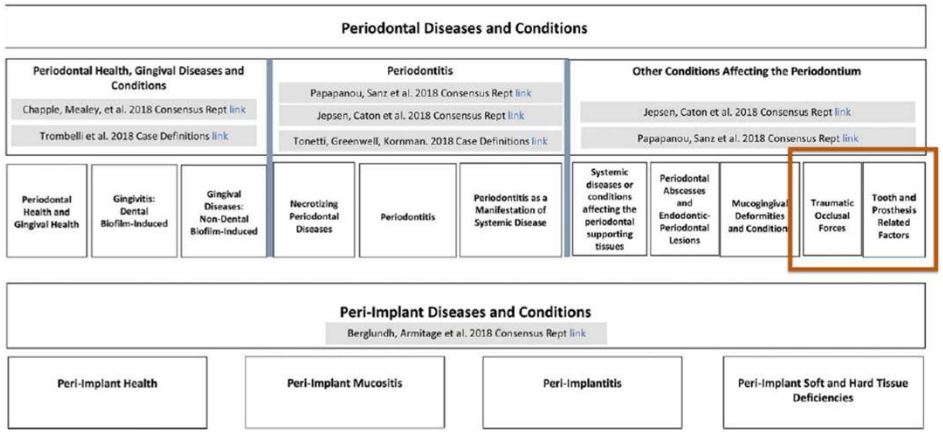

TABELLA 8. Criteri per la definizione dei gradi di parodontite. (da Tonetti, et al. 2018)

Gradi di Parodontite – Si veda testo e Appendice A (nell'online *Journal of Clinical Periodontology*) per spiegazione

GRADO DI PARODONTITE			GRADO A: BASSO TASSO DI PROGRESSIONE	GRADO B: MODERATO TASSO DI PROGRESSIONE	GRADO C: RAPIDO TASSO DI PROGRESSIONE
Criteri Primari	Diretta evidenza di progressione	Dati longitudinali (perdita radiografica di CAL)	Evidenza di nessuna perdita in 5 anni	<2 mm in 5 anni	≥2 mm in 5 anni
	Indiretta evidenza di progressione	% perdita ossea/età	<0.25	0.25 to 1.0	>1.0
		Fenotipo del caso	Grandi depositi di biofilm accompagnati da bassi livelli di distruzione ossea	Distruzione ossea commisurata ai depositi di biofilm	La distruzione supera le attese formulate in base ai depositi di biofilm; specifici schemi clinici indicativi di periodi di rapida progressione e/o malattia a esordio precoce (per es. rapporto incisivo/molare; mancanza di risposta attesa alle terapie standard di controllo batterico)
Fattori di modificazione del Grado	Fattori di rischio	Fumatore	Non – fumatore	Fumatore <10 sigarette/giorno	Fumatore ≥10 sigarette/giorno
		Diabete	Normoglicemia/no diagnosi di diabete	HbA1c <7.0% in pazienti diabetici	HbA1c ≥7.0% in pazienti diabetici
Rischio di impatto sistemico della parodontite*	Carico infiammatorio	Alta sensibilità CRP (hsCRP)	<1 mg/L	Da 1 a 3 mg/L	>3 mg/L
Biomarkers	Indicatori di CAL/perdita ossea	Saliva, fluido crevicolare gengivale, siero	?	?	?

Il grado dovrebbe essere usato come indicatore del tasso di progressione della parodontite. I criteri principali sono l'evidenza diretta o indiretta della progressione. Se disponibili, vengono utilizzate prove dirette; in loro assenza viene effettuata una stima indiretta utilizzando la perdita ossea in funzione dell'età a carico dente più colpito o della presentazione del caso (perdita radiografica di osso espressa come percentuale della lunghezza della radice divisa per l'età del soggetto, RBL/età). Il clinico dovrebbe inizialmente presumere un Grado B di malattia e poi cercare prove specifiche per passare al Grado A o al Grado C, secondo il caso. Una volta stabilita la graduazione in base all'evidenza della progressione, questa può essere modificata in base alla presenza di fattori di rischio.

TABELLA 7. Criteri per la definizione degli stadi di parodontite. (da Tonetti, et al. 2018).

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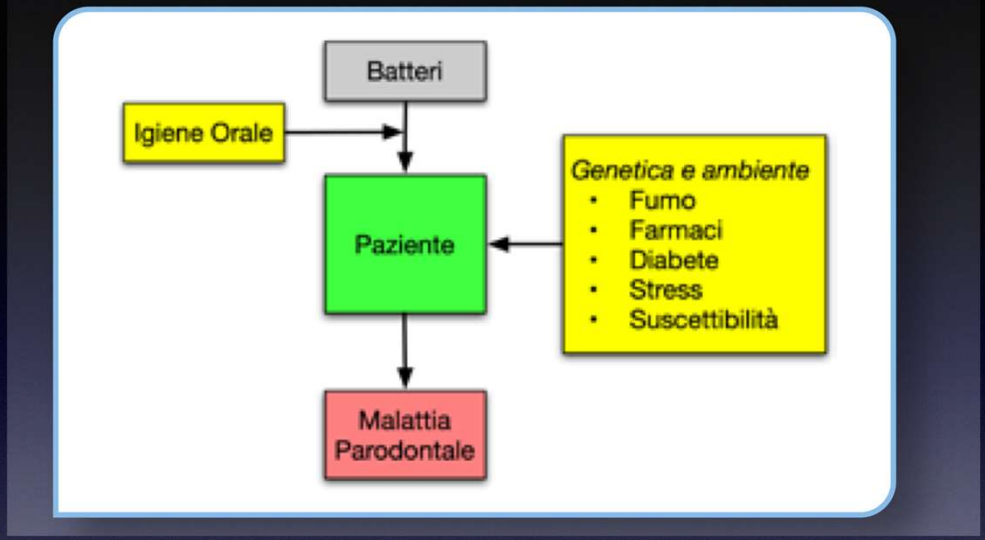
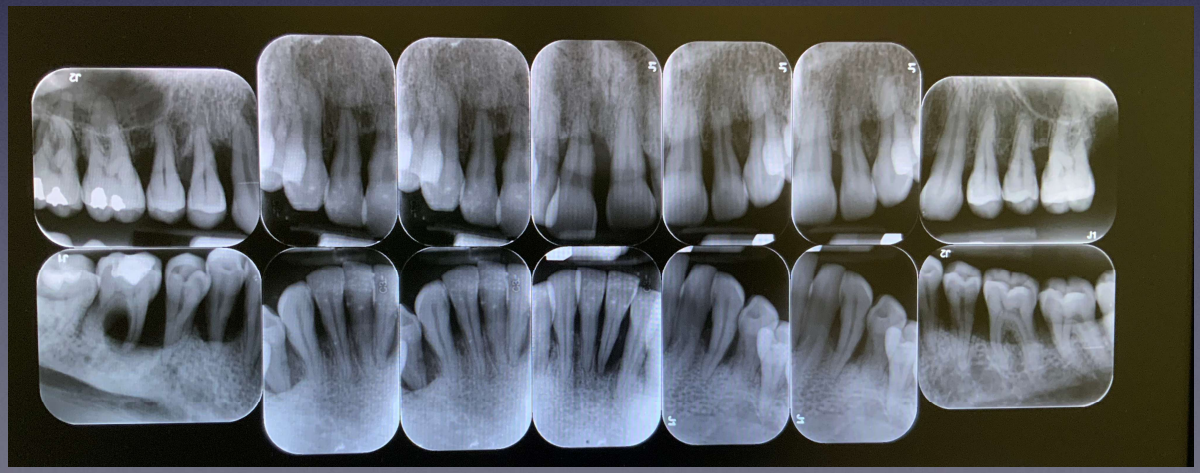
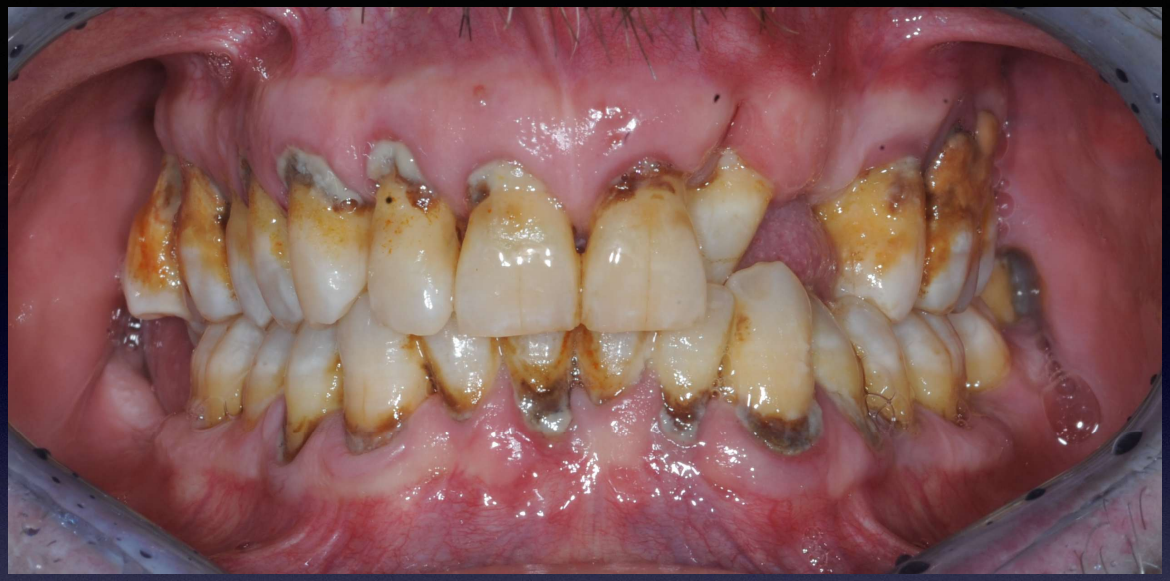
		Massima profondità di sondaggio ≤4 mm	Massima profondità di sondaggio ≤5 mm	profondità di sondaggio ≥6 mm	Necessità di riabilitazione complessa dovuta a:
		Perdita ossea prevalentemente orizzontale	Perdita ossea prevalentemente orizzontale	Perdita ossea verticale ≥3 mm	Disfunzione masticatoria Trauma occlusale secondario (mobilità dentale di grado ≥2) Gravi difetti delle selle Collasso del morso, spostamenti, sventagliamento. Meno di 20 denti rimasti (10 paia di denti contrapposti)
				Interessamento delle forcazioni di Classe II o III	
				Moderati difetti delle selle	
Estensione e distribuzione	**Aggiungere allo stadio come descrizione**	Per ogni fase, descrivere l'estensione come localizzata (> 30% dei denti coinvolti), generalizzata o rapporto incisivo/molare			





PARODONTITE :



DEFINIZIONE
La parodontite è caratterizzata da una progressiva distruzione dell'apparato di sostegno del dente. Le sue caratteristiche principali includono la perdita del tessuto di supporto parodontale che si manifesta attraverso la perdita di attacco clinico (CAL) e la perdita di osso alveolare (verificata radiograficamente), la presenza di tasche parodontali e di sanguinamento gengivale (Papapanou, et al. 2018).
Se non trattata, può portare alla perdita dei denti, sebbene sia prevenibile e curabile nella maggior parte dei casi.

IMPORTANZA
La parodontite è un grave problema di salute pubblica a causa della sua elevata prevalenza e, poiché può portare alla perdita e alla disabilità dei denti, influisce negativamente sulla funzione masticatoria e sull'estetica, è fonte di disuguaglianza sociale e compromette significativamente la qualità della vita. La parodontite è responsabiledi una parte sostanziale dell'edentulismo e della disfunzione masticatoria, ha un impatto negativo sulla salute generale e comporta costi significativi per cure odontoiatriche (Tonetti, et al. 2017).
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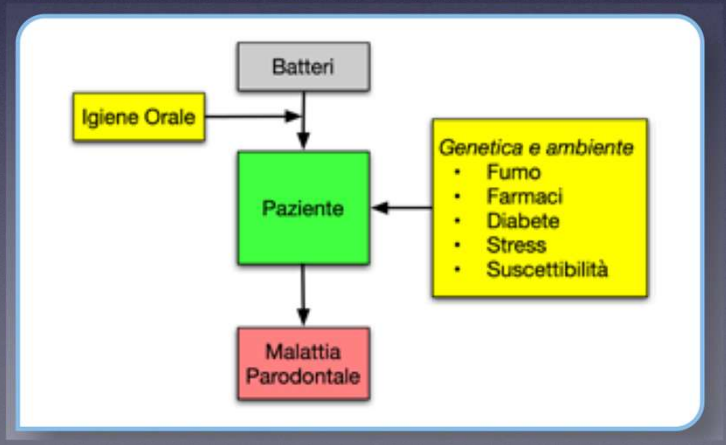

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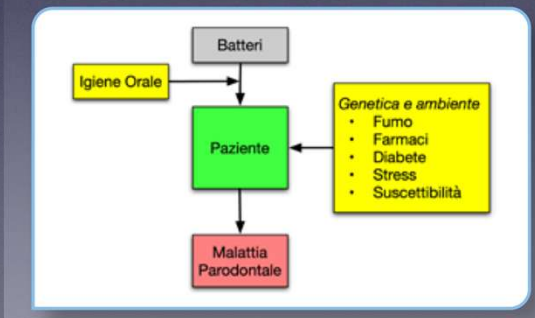

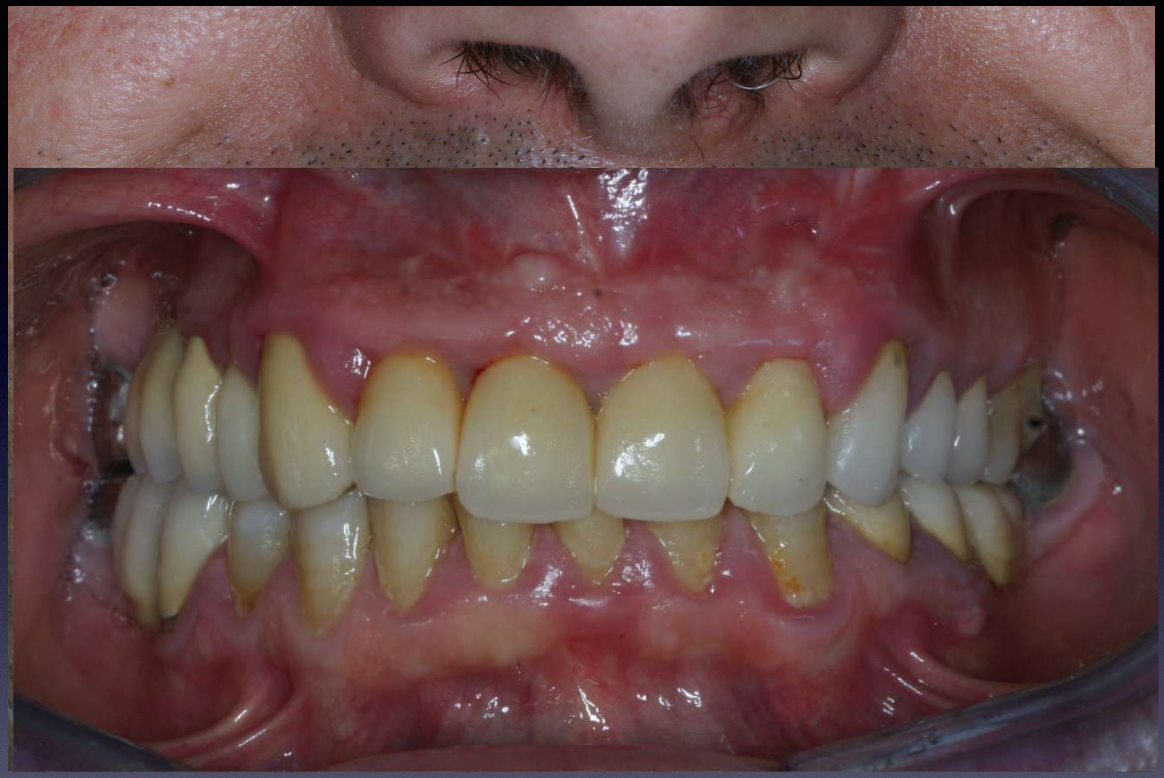

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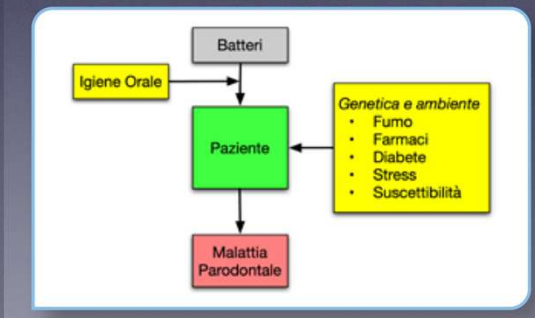
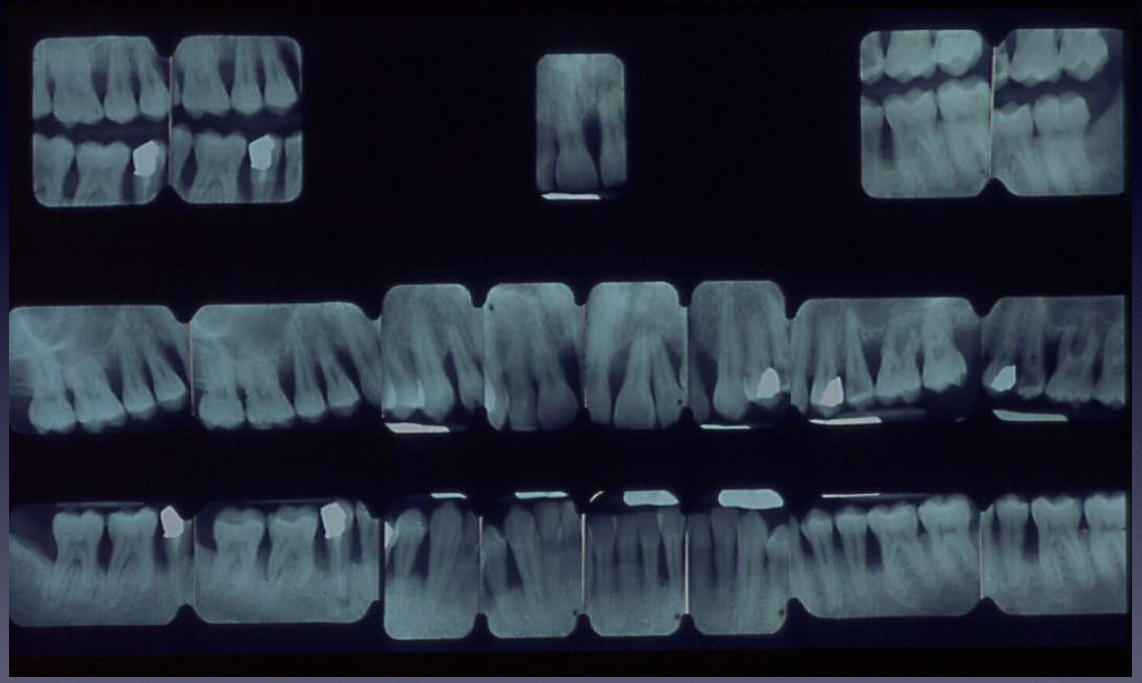
La parodontite è una malattia infiammatoria multifattoriale cronica associata a una disbiosi del biofilm che costituisce la placca dentale.





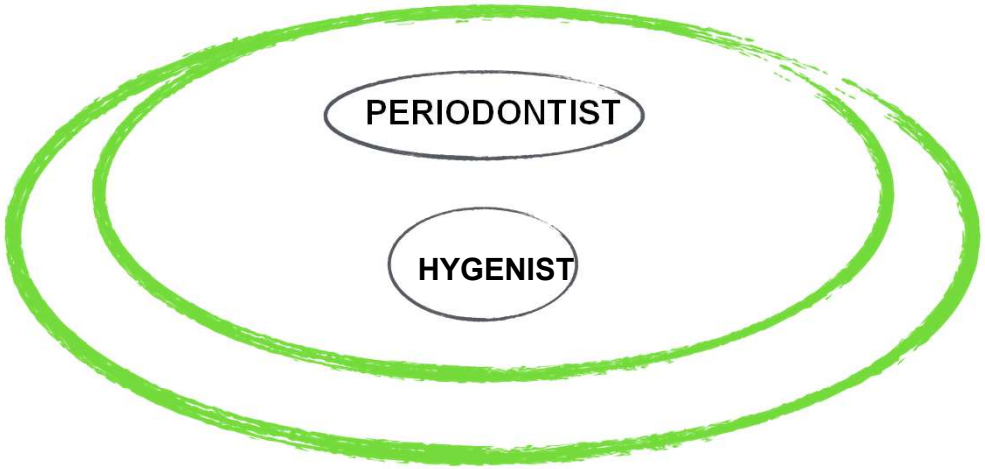
PARODONTITE :

LA TERAPIA PARODONTALE STEP BY STEP

La recente pubblicazione delle linee guida sul trattamento delle Parodontiti Stadio I-III e Stadio IV da parte della Federazione Europea di Parodontologia (EFP) [16,17] ha delineato con estrema chiarezza **il percorso terapeutico da seguire per trattare efficacemente i pazienti affetti da parodontite**, attraverso una sequenza precisa di Step procedurali di ingravescente complessità, in funzione dello stadio della malattia. Tali step sono per praticità schematizzati come segue:

PERIODONTAL THERAPY……

	PROCEDURE	OBJ	M/M
STEP 4	**T P S** **supportive periodontal therapy**	MAINTENANCE of Periodontal STABILITY without: **PPD>4mm**. & **IP** and **BoP** < 10-15%	**Each patient has an INDIVIDUALIZED PROFILE-RISK for presenting PD again, so the FREQUENCY & Procedures has to fit the biological, social, psychological and systemic profile of the SINGLE PT.**



Sanz M et al. Treatment of stage I-III periodontitis - The EFP S3-level clinical guideline. *J Clin Periodontology 2020*.

Therapeutic phases – EFP Guidelines

STEP 2

Use of adjunctive host-modulating agents (local or systemic) to subgingival instrumentation

Open recommendation

Chlorhexidine mouth rinses for a limited period of time may be considered as adjuncts to subgingival instrumentation.

Locally administered sustained-release chlorhexidine may be considered as an adjunct to subgingival instrumentation.

Specific **locally administered sustained-release antibiotics** may be considered as an adjunct to subgingival instrumentation.

PAPERS

Association between dental health and acute myocardial infarction

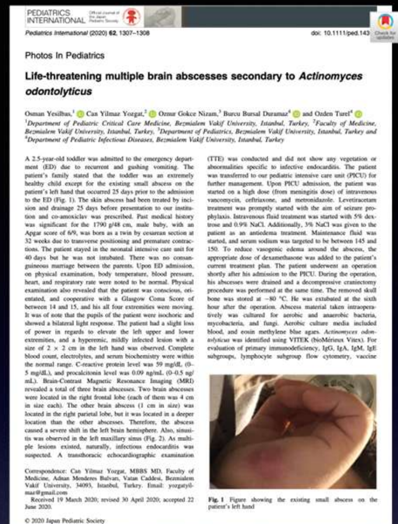
Kimmo J Mattila, Markku S Nieminen, Ville V Valtonen, Vesa P Rasi, Y Antero Kesäniemi, Seija L Syrjälä, Peter S Jungell, Martti Isoluoma, Katariina Hietaniemi, Matti J Jokinen, Jussi K Huttunen

Abstract

Introduction

Subjects and methods

BMJ VOLUME 298 25 MARCH 1989



PEDIATRICS INTERNATIONAL

Pediatrics International (2020) 62, 1307–1308

Photos In Pediatrics

Life-threatening multiple brain abscesses secondary to *Actinomyces odontolyticus*

Fig. 1 Figure showing the existing small abscess on the patient's left hand.

© 2020 Japan Pediatric Society

Case Report

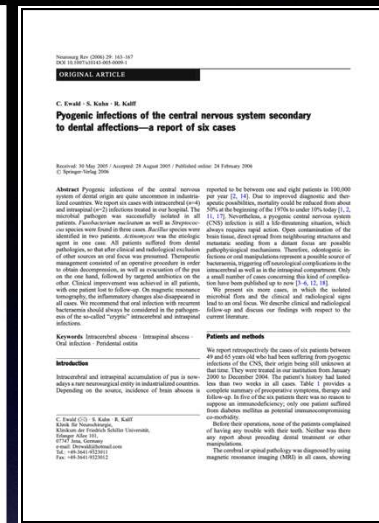
Characterization of *Streptococcus constellatus* Strains Recovered From a Brain Abscess and Periodontal Pockets in an Immunocompromised Patient

Rafael Marques da Silva, Dominique A. Caugant, Roger Josefsen, Leif Tronstad, and Ingar Olsen

KEY WORDS

Abscess, brain/etiology; bacteria, anaerobic; periodontitis; *Streptococcus constellatus*.

CASE DESCRIPTION



ORIGINAL ARTICLE

C. Ewald · S. Kuhn · R. Kalff

Pyogenic infections of the central nervous system secondary to dental affections—a report of six cases

Introduction

Patients and methods

Maria Antonietta Cassini
Francesco Dominici
Ciro Quaranta

Possibile relazione tra parodontite e ipercolesterolemia

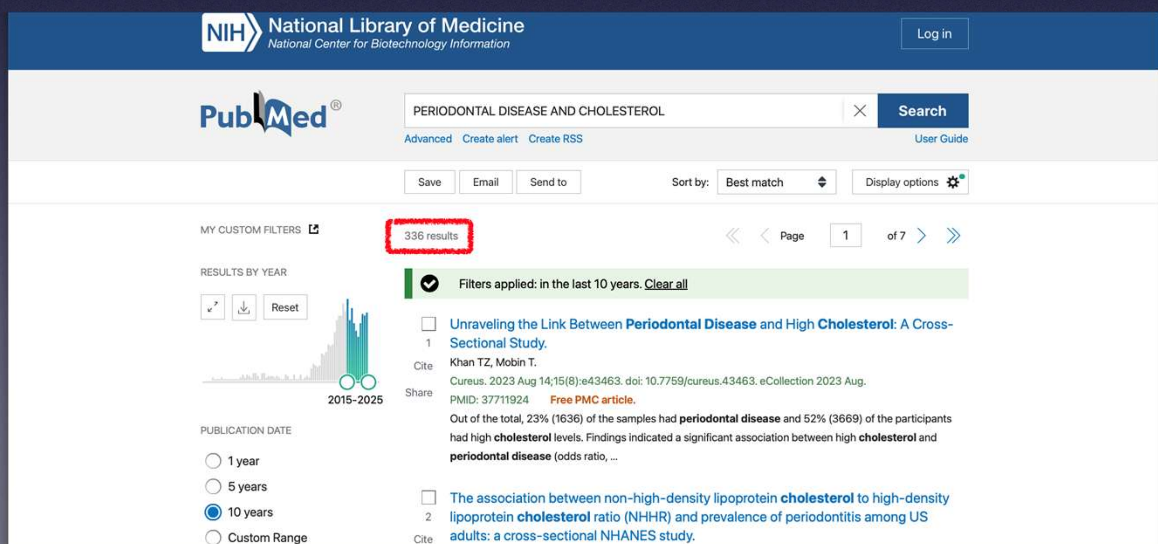
Possible relationship between periodontitis and hypercholesterolemia

RIASSUNTO

PAROLE CHIAVE: parodontite, ipercolesterolemia, colesterolo totale, LDL, HDL, patologie cardiovascolari.

ABSTRACT

KEY WORDS: periodontitis, hypercholesterolemia, total cholesterol, LDL, HDL, cardiovascular disease.



2016

OPEN ACCESS

High-risk periodontal pathogens contribute to the pathogenesis of atherosclerosis

Bradley Field Bale,[1] Amy Lynn Doneen,[1] David John Vigerust[2]

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ABSTRACT
Periodontal disease (PD) is generated by microorganisms. These microbes can enter the general circulation causing a bacteraemia. The result can be adverse systemic effects, which could promote conditions such as cardiovascular disease. Level A evidence supports that PD is independently associated with arterial disease. PD is a common chronic condition affecting the majority of Americans 30 years of age and older. Atherosclerosis remains the largest cause of death and disability. Studies indicate that the adverse cardiovascular effects from PD are due to a few putative or high-risk bacteria: *Aggregatibacter actinomycetemcomitans*, *Porphyromonas gingivalis*, *Tannerella forsythia*, *Treponema denticola* or *Fusobacterium nucleatum*. There are three accepted essential elements in the pathogenesis of atherosclerosis: lipoprotein serum concentration, endothelial permeability and binding of lipoproteins in the arterial intima. There is scientific evidence that PD caused by the high-risk pathogens can influence the pathogenesis triad in an adverse manner. With this appreciation, it is reasonable to state PD, due to high-risk pathogens, is a contributory cause of atherosclerosis. Distinguishing this type of PD as causal provides a significant opportunity to reduce arterial disease.

BACKGROUND

Bacteraemia with germs from the oral cavity was well documented in a publication in 1954. The landmark study indicated systemic spread of oral microbes occurs frequently. The per cent incidence found was 40% with periodontal cleaning, 35% with dental extractions, 24% with brushing and up to 17% with mastication.[1] The spread of these oral bacteria throughout the body happens quickly. This results in acute and chronic inflammation, which can be pathological. High-risk periodontal pathogens include *Aggregatibacter actinomycetemcomitans* (*Aa*), *Porphyromonas gingivalis* (*Pg*), *Tannerella forsythia* (*Tf*), *Treponema denticola* (*Td*) and *Fusobacterium nucleatum* (*Fn*). They are prevalent in periodontitis. These germs enter the systemic circulation directly, and they also produce endotoxins such as lipopolysaccharides (LPS). These endotoxins generate inflammatory cytokines, upregulate endothelial adhesion molecules and induce a prothrombotic environment. These actions can favour the formation of arterial disease and can enhance the risk of an atherothrombotic event.[2] Using DNA to identify putative oral bacteria, several studies have documented their presence within atheroma. In 2009, 44 patients underwent coronary endarterectomies. Thirty-nine of the subjects had periodontal disease (PD). Thirty-six of the specimens from patients with PD were positive for oral pathogens. The most common were *Pg* and *Aa*. Sixty-four per cent of those atheromas had two or more pathogens. Only one of the atheroma from a patient without PD demonstrated any oral pathogens.[3] In 2011, 42 carotid endarterectomy specimens were analysed for oral pathogen DNA. Every atheroma had at least one pathogen, and many had multiple pathogens. Again, the most common bacteria were *Pg* and *Aa*.[4] Oral pathogens create bacteraemia, and those bacteria, especially the high-risk microbes, are frequently associated with atherosclerotic lesions.

The American Heart Association (AHA) stated after an extensive review of the literature that PD was independently associated with arteriosclerotic vascular disease (ASVD). This relationship was demonstrated with level A evidence. They discussed in their statement several plausible mechanisms by which PD could be associated with arterial disease. One explanation involves systemic inflammation, which can occur with periodontitis. This has been documented by increased levels of biomarkers such as high-sensitivity C-reactive protein, tumour necrosis factor-α (TNF-α) and interleukin 6. PD has been associated with the stimulation of the innate immune system via toll-like receptors (TLRs). TLRs can trigger the activation of nuclear factor κβ (NF-κB), which can create increased levels of adhesion molecules stimulating endothelial dysfunction as well as increased inflammatory cytokines. The possibility of LPSs and heat-shock proteins coming from the PD pathogens causing an autoimmune-type reaction from T and B cells was mentioned. Another potential mechanism discussed was direct arterial damage from the bacteria in the blood stream. They acknowledged that there are studies showing PD therapy has improved biomarkers of systemic inflammation and even surrogate indicators of subclinical arterial disease. However, due to the fact that there is no definite evidence to support the claim that treating PD decreases cardiovascular (CV) events, the AHA went on to comment that PD could not be considered causal of ASVD.[5] From a clinical perspective, there is a significant difference between being associated with versus being causal of a disease. Optimal management of an associated condition may not impact the end disease, whereas such management of a causal condition would have a favourable effect on the end disease. Since the AHA generated their statement about the relationship of PD to ASVD, new evidence has emerged which argues PD caused by the high-risk pathogens, *Aa*, *Pg*, *Tf*, *Td* or *Fn*, can enhance elements of the atherosclerosis pathogenesis triad. Due to this scientific knowledge, PD caused by these putative bacteria can be considered causal of ASVD. This is a significant clinical distinction, which means PD due to these organisms should be optimally treated to mitigate the risk of ASVD.

Recent publications indicate that the formation of atherosclerosis is dependent upon three essentials: serum lipoprotein concentration, endothelial permeability and lipoprotein binding in the intima.[6] If a medical condition can influence all of these critical elements in a manner favouring the pathogenesis of atherosclerosis, it can be concluded to be a contributory cause of ASVD. A contributory cause does not require that all those who possess the contributory cause experience the disease, nor does it require that all those who are free of the contributory cause be free of the disease. It also means the contributory cause may not be necessary to experience the disease.[7] PD is independently associated with ASVD; substantial evidence and mechanistic understanding now exist that strongly suggest PD, due to the above high-risk pathogens, is a contributory cause of ASVD. Herein, we present a summary of this evidence and understanding. Based on these findings, PD resulting from high-risk pathogens should be considered causal of ASVD.

PATHOGENESIS, PART 1: SERUM LIPOPROTEIN CONCENTRATION

Atherogenic lipoprotein particles are heterogeneous, containing various amounts of cholesterol. Each particle has an apolipoprotein B (ApoB) on its surface. Wilkins *et al*[8] recently demonstrated that ApoB concentration in the serum is more predictive of the pathogenesis of ASVD than serum cholesterol concentration. In addition to this study, previous studies have also shown the best lipoprotein predictor of cardiovascular disease risk is ApoB.[8–10] There is a direct positive and independent association with ApoB concentration and small, dense low-density lipoprotein (sd-LDL) concentrations.[11] Therefore, an essential element in the pathogenesis of ASVD, namely ApoB concentration, is increased with elevating levels of sd-LDL. Secretory phospholipase A2 (sPLA2) modifies LDL into sd-LDL.[12] The activity of sPLA2 has been shown to increase in subjects with high-risk periodontal pathogens *Aa*, *Pg*, *Tf* and *Td*. The higher the burden of these pathogens, the greater the activity of sPLA2.[13] Generalised aggressive PD is associated with the putative pathogens *Pg* and *Aa*.[14] It is demonstrated that patients with generalised aggressive periodontitis have twice the concentration of sd-LDL compared with subjects without PD.[15] This essentially means the concentration of ApoB is doubled. PD due to high-risk pathogens can increase the plasma concentration of ApoB, which will promote the pathogenesis of ASVD.

PATHOGENESIS, PART 2: ENDOTHELIAL PERMEABILITY

Formation of an atheroma requires monocytes along with lipoproteins to penetrate the endothelium. It is the accumulation of lipoproteins in the intima that creates the core of the atheromatous lesion. The permeability of the endothelium is a critical factor in the pathogenesis of ASVD.[16] Dysfunctional endothelium becomes more permeable. It is proposed that endothelial dysfunction is the initial biological malady leading to ASVD.[17] PD can generate endothelial dysfunction by several mechanisms. The high-risk gram-negative pathogens (*Aa*, *Pg*, *Tf*, *Td*) generate LPSs, which trigger the innate immune system. LPSs stimulate TLRs, which are ubiquitous and present in endothelial cells. TLR signalling activates the genetic transcription factor NF-κB. This in turn leads to elevated levels of endothelial cellular adhesion molecules and TNF-α.[18] Cellular adhesion molecules capture monocytes and increase the permeability of the endothelium.[19] TNF-α is known to induce increased endothelial permeability via its effect on tight junction proteins.[20] Chronic periodontal infections with high-risk pathogens can lead to increased permeability of the endothelium via the innate immune system.

Several other mechanisms exist by which PD can generate dysfunctional and permeable endothelium. Endothelial integrity depends on its endogenous capacity for repair. Persistent exposure to inflammation leads to senescence of endothelial cells and their detachment, with consequent increased permeability. This adverse injury can be repaired by replication of adjacent endothelial cells or by formation of new mature endothelial cells from endothelial progenitor cells.[21] The high-risk PD pathogen (*Aa*) produces a toxic protein known as leukotoxin (LtxA). This toxin has been shown to generate endothelial apoptosis along with impaired proliferation and increase in endothelial adhesion molecules.[22] These adverse effects of LtxA will lead to increased penetrability of the endothelium.

PD can impair the structural coupling of endothelial cells. Cadherin proteins lock endothelial cells tightly together. Any disruption of endothelial cadherin will result in endothelial dysfunction and enhanced permeability.[23] The PD pathogen *Fn* possesses a surface adhesion molecule called FadA. There are two forms of FadA. One is a non-secreted pre-FadA consisting of 129 amino acids and the other is a secreted mature FadA (mFadA) consisting of 111 amino acids. Pre-FadA is anchored in the *Fn* membrane, and mFadA is exposed on the surface of the bacteria. Together, they form a high molecular weight complex that can attach to endothelial cadherin, which causes a relocation of cadherin away from the cell–cell junctions. This results in an endothelium so permeable that even bacteria can pass.[24] High-risk PD pathogens can increase endothelial permeability via multiple mechanisms.

PATHOGENESIS, PART 3: LIPOPROTEIN BINDING IN INTIMA

The third requirement for the pathogenesis of ASVD is binding of the lipoproteins in the intimal layer of the arterial wall. Perfusion studies with labelled lipoproteins show most lipoproteins that penetrate the endothelium diffuse through all the layers and efflux on the adventitial side. The numbers of retained lipoproteins in the intima are several orders of magnitude less than the number of particles that diffuse through the arterial wall.[25] Therefore, retention of lipoproteins in the intima is a vital step in the pathogenesis of ASVD. The type of lipoprotein captured in the wall is not as important as the quantity of lipoproteins retained. Binding occurs via electrostatic forces to proteoglycans found in the extracellular matrix (ECM) of the intima. Proteoglycans have negatively charged areas, which bind to positively charged areas of amino acids on ApoB. There are two sites of attachment on ApoB-100, with one of those exposed only in sd-LDL particles. There is one site of attachment on ApoB-48.[26]

Smooth muscle cells (SMCs) have two phenotypes. One is the contractile phenotype found in the medial layer of the arterial wall. The other is the synthetic phenotype that migrates from the medial layer to the intimal layer. Synthetic SMCs create a proteoglycan-enriched ECM in the intima.[27] Studies demonstrate that synthetic SMCs are the first intimal cells present in locations destined to develop ASVD.[28] Microbial infection is known to stimulate genetic transcription of contractile SMCs to synthetic SMCs.[29] PD due to the high-risk pathogen *Pg* has been shown to influence the levels of angiopoietin 1 (Angpt1) and angiopoietin 2 (Angpt2) in SMCs. *P. gingivitis* creates higher levels of Angpt2, which stimulates a genetic transformation of SMCs to the synthetic phenotype. Zhang *et al*[30] concluded this is another mechanism by which periodontitis is associated with ASVD. PD has the potential to increase the binding of lipoproteins in the intima by enhancing the quantity of proteoglycans in intimal ECM.

DISCUSSION

PD due to high-risk pathogens may facilitate the three critical steps in the pathogenesis of ASVD (figure 1). Therefore, it is reasonable that PD, due to the high-risk pathogens (*Aa*, *Pg*, *Td*, *Tf*, *Fn*), be considered causal of ASVD on clinical grounds. Since ASVD is a complex multifactorial disease process, PD due to high-risk pathogens is a contributory cause. This means such PD is neither required nor sufficient for the pathogenesis of ASVD.[7] It is necessary to elevate the distinction of PD to causal, as opposed to simply associated, for clinical management purposes. Causal classification requires therapy to mitigate the risk of its effect. In this case, it means PD due to these high-risk microbes must be treated effectively to reduce the risk of ASVD.

As is often the case in science, a new realisation creates a list of unsolved issues. In this case, one obvious question is how to objectively identify the pathogens. The definition of PD must include a diagnosis of the specific underlying pathogens causing the PD. There are numerous studies that have demonstrated the CV risk from PD is driven by the pathogen burden as opposed to only clinical exam findings such as pocket depth, bleeding on probing and bone loss.[31–33] The clinical oral health examination is important and must remain an important component of the diagnosis. However, from a CV standpoint, there must be an objective assessment of high-risk pathogen burden as part of the definition of PD. There are several DNA-based oral pathogen tests available to assess high-risk pathogens. However, many dentists are not familiar with these tests, and there is a substantial cost involved. Understanding that high-risk PD pathogens are causal of ASVD places an onus upon our dental colleagues to continue their efforts to develop affordable, reproducible, objective testing for high-risk PD pathogens.

An additional substantial issue following the recognition of PD due to high-risk pathogens as causal of ASVD is: how is it successfully managed? There is sparse data addressing the most effective way to manage such disease. One randomised prospective study of 101 patients with PD tested the effectiveness of scaling and root planning (SRP) alone versus SRP plus systemic antibiotics. There were two antibiotic arms: metronidazole (MTZ) 400 mg three times per day or MTZ+ amoxicillin (AMX) (500 mg three times per day) for 14 days. In addition, half of the patients in each of the three arms of therapy rinsed

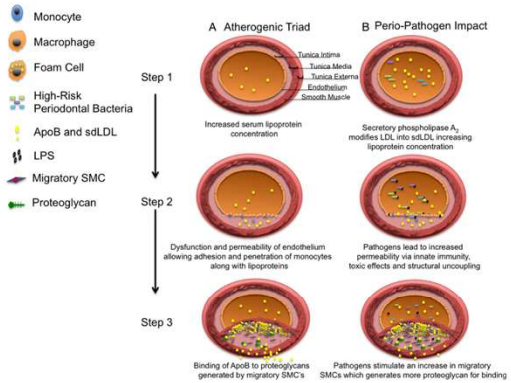

Figure 1 Atherogenic triad in the presence and absence of high-risk periodontal pathogens. Column A: Illustration of the atherogenic triad. (1) Lipoprotein concentration in the serum, with each lipoprotein containing ApoB. (2) Dysfunction and permeability of the endothelium which allow monocytes to adhere to the endothelium and to penetrate into the intima along with lipoproteins. (3) Binding of ApoB to proteoglycans derived from migratory SMCs, as well as conversion of monocytes to macrophages, which become foam cells. Column B: Illustration of how high-risk periodontal pathogens adversely enhance each of the three elements causing greater atherogenesis. (1) In the presence of high-risk pathogens, the concentration of ApoB is increased. (2) In the presence of high-risk pathogens, the endothelial dysfunction and permeability are enhanced by LPS. (3) In the presence of high-risk pathogens, the binding of lipoproteins is enhanced by increased migratory SMCs, which enrich the intima with proteoglycans. ApoB, apolipoprotein B; LDL, low-density lipoprotein; LPS, lipopolysaccharides; sdLDL, small dense LDL; SMCs, smooth muscle cells.

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ASVD remains the number one cause of mortality and morbidity in this country.[40] Current literature supports that it is more cost-effective to prevent ASVD than to treat its complications.[41] Arterial disease has a common core cause, which is inflammation.[42] There are a multitude of conditions that can generate arterial inflammation. The list includes: lipids, smoking, hypertension, insulin resistance, vitamin D deficiency, obstructive sleep apnoea, obesity, diet, physical inactivity, psychosocial issues, oral health issues, systemic inflammatory conditions such as rheumatoid arthritis, lupus, genetic influences, and systemic infectious disease and gut dysbiosis.[43–57] We now have evidence that PD can cause systemic inflammation and can also adversely affect the three essential mechanisms in the pathogenesis of ASVD. It is necessary to classify PD due to high-risk pathogens as a cause of ASVD. This type of PD is a medical condition with a dental solution. At present, our dental colleagues have not elucidated a definitive way in which to

concluded this is another mechanism by which periodontitis is associated with ASVD. PD has the potential to increase the binding of lipoproteins in the intima by enhancing the quantity of proteoglycans in intimal ECM.

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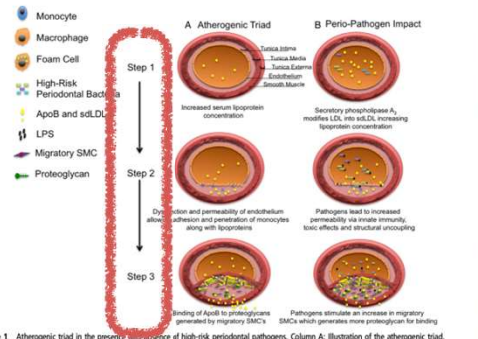

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ABSTRACT
Periodontal disease (PD) is generated by microorganisms. These microbes can enter the general circulation causing a bacteraemia. The result can be adverse systemic effects, which could promote conditions such as cardiovascular disease. Level A evidence supports that PD is independently associated with arterial disease. PD is a common chronic condition affecting the majority of Americans 30 years of age and older. Atherosclerosis remains the largest cause of death and disability. Studies indicate that the adverse cardiovascular effects from PD are due to a few putative or high-risk bacteria: *Aggregatibacter actinomycetemcomitans*, *Porphyromonas gingivalis*, *Tannerella forsythia*, *Treponema denticola* or *Fusobacterium nucleatum*. There are three accepted essential elements in the pathogenesis of atherosclerosis: lipoprotein serum concentration, endothelial permeability and binding of lipoproteins in the arterial intima. There is scientific evidence that PD caused by the high-risk pathogens can influence the pathogenesis triad in an adverse manner. With this appreciation, it is reasonable to state PD, due to high-risk pathogens, is a contributory cause of atherosclerosis. Distinguishing this type of PD as causal provides a significant opportunity to reduce arterial disease.

BACKGROUND
Bacteraemia with germs from the oral cavity was well documented in a publication in 1954. The landmark study indicated systemic spread of oral microbes occurs frequently. The per cent incidence found was 40% with periodontal cleaning, 35% with dental extractions, 24% with brushing and up to 17% with mastication.[1] The spread of these oral bacteria throughout the body happens quickly. This results in acute and chronic inflammation, which can be pathological. High-risk periodontal pathogens include *Aggregatibacter actinomycetemcomitans* (*Aa*), *Porphyromonas gingivalis* (*Pg*), *Tannerella forsythia* (*Tf*), *Treponema denticola* (*Td*) and *Fusobacterium nucleatum* (*Fn*). They are prevalent in periodontitis. These germs enter the systemic circulation directly, and they also produce endotoxins such as lipopolysaccharides (LPS). These endotoxins generate inflammatory cytokines, upregulate endothelial adhesion molecules and induce a prothrombotic environment. These actions can favour the formation of arterial disease and can enhance the risk of an atherothrombotic event.[2] Using DNA to identify putative oral bacteria, several studies have documented their presence within atheroma. In 2009, 44 patients underwent coronary endarterectomies. Thirty-nine of the subjects had periodontal disease (PD). Thirty-six of the specimens from patients with PD were positive for oral pathogens. The most common were *Pg* and *Aa*. Sixty-f cent of those atheromas had two or more gens. Only one of the atheroma from a without PD demonstrated any oral pathog 2011, 42 carotid endarterectomy specime analysed for oral pathogen DNA. Every at had at least one pathogen, and many had pathogens. Again, the most common bacte *Pg* and *Aa*.[4] Oral pathogens create bacteraem those bacteria, especially the high-risk micro frequently associated with atherosclerotic lesi

The American Heart Association (AHA after an extensive review of the literature was independently associated with arterio vascular disease (ASVD). This relationsh demonstrated with level A evidence. They d in their statement several plausible mechan which PD could be associated with arterial One explanation involves systemic inflam which can occur with periodontitis. This h documented by increased levels of biomark as high-sensitivity C-reactive protein, necrosis factor-α (TNF-α) and interleukin has been associated with the stimulation innate immune system via toll-like r (TLRs). TLRs can trigger the activation of factor κβ (NF-κB), which can create ir levels of adhesion molecules stimulating end dysfunction as well as increased inflammato kines. The possibility of LPSs and heat-sho teins coming from the PD pathogens cau autoimmune-type reaction from T and B c mentioned. Another potential mechanism d was direct arterial damage from the bacteri blood stream. They acknowledged that th studies showing PD therapy has improved kers of systemic inflammation and even s indicators of subclinical arterial disease. H due to the fact that there is no definite e to support the claim that treating PD d cardiovascular (CV) events, the AHA wen comment that PD could not be considere of ASVD.[5] From a clinical perspective, th significant difference between being associat versus being causal of a disease. Optimal ment of an associated condition may not the end disease, whereas such manageme causal condition would have a favourable e the end disease. Since the AHA generate statement about the relationship of PD to new evidence has emerged which argues PD by the high-risk pathogens, *Aa*, *Pg*, *Tf*, *Td* can enhance elements of the atherosclerosis genesis triad. Due to this scientific knowle caused by these putative bacteria can be cor causal of ASVD. This is a significant

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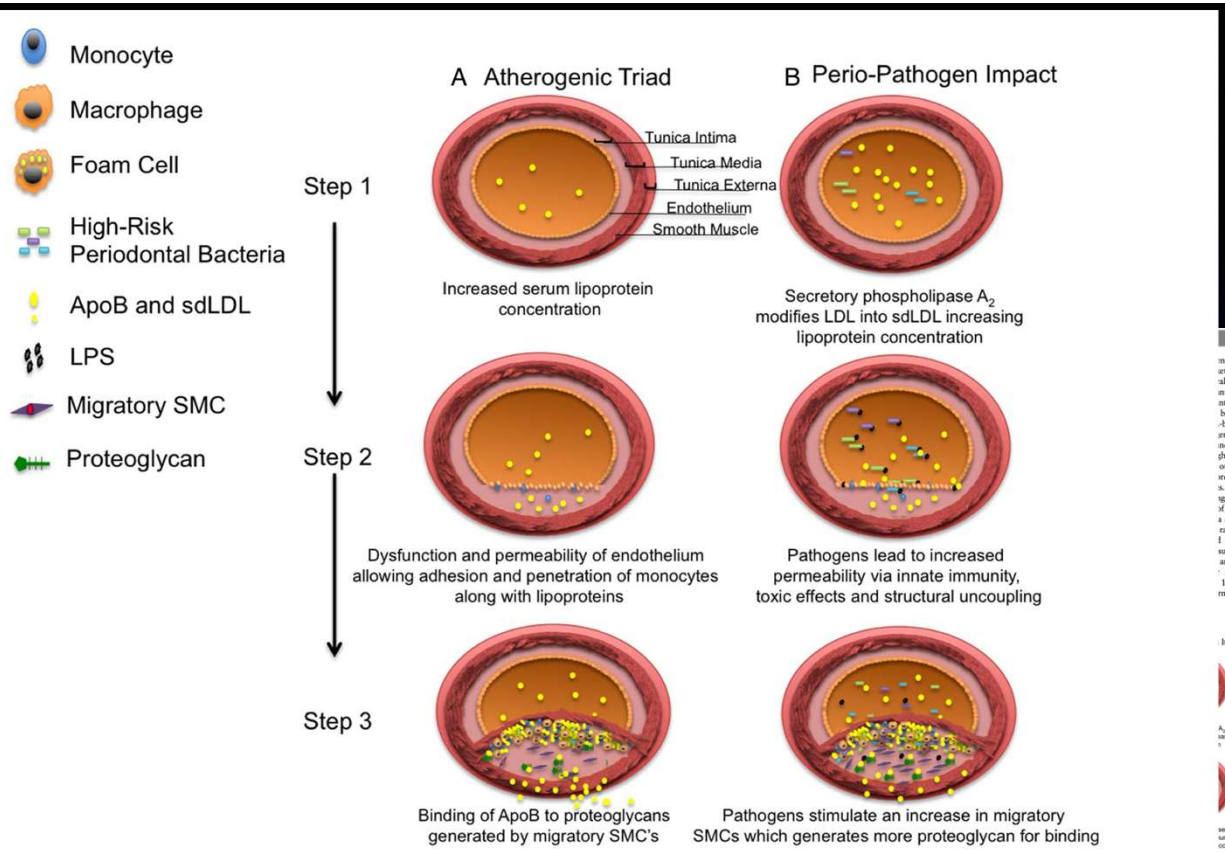

Figure 1 Atherogenic triad in the presence and absence of high-risk periodontal pathogens. Column A: Illustration of the atherogenic triad. (1) Lipoprotein concentration in the serum, with each lipoprotein containing ApoB. (2) Dysfunction and permeability of the endothelium which allow monocytes to adhere to the endothelium and to penetrate into the intima along with lipoproteins. (3) Binding of ApoB to proteoglycans derived from migratory SMCs, as well as conversion of monocytes to macrophages, which become foam cells. Column B: Illustration of how high-risk periodontal pathogens adversely enhance each of the three elements causing greater atherogenesis. (1) In the presence of high-risk pathogens, the concentration of ApoB is increased. (2) In the presence of high-risk pathogens, the endothelial dysfunction and permeability are enhanced by LPS. (3) In the presence of high-risk pathogens, the binding of lipoproteins is enhanced by increased migratory SMCs, which enrich the intima with proteoglycans. ApoB, apolipoprotein B; LDL, low-density lipoprotein; LPS, lipopolysaccharides; sdLDL, small dense LDL; SMCs, smooth muscle cells.

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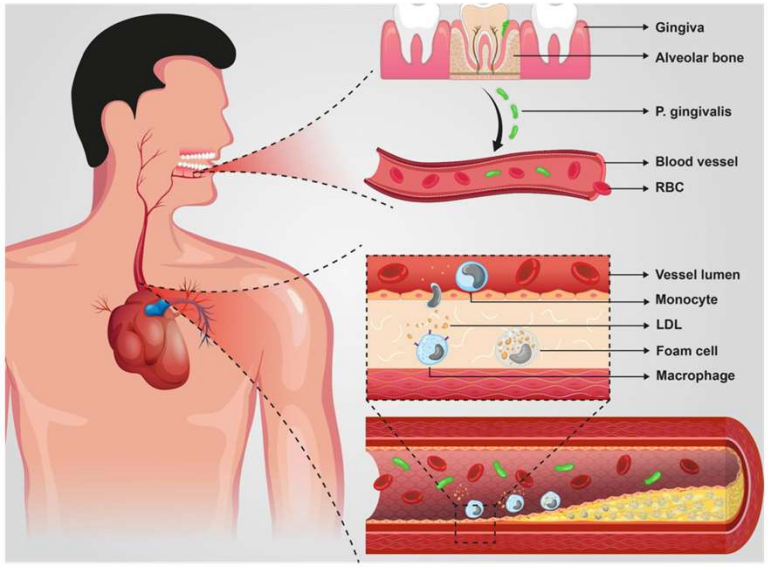

Fig. 2 Illustrates the process by which *P. gingivalis* can play a role in foam cell formation. The infection caused by periodontopathogens, especially *P. gingivalis* destructs periodontal tissue. The progression of this disease leads to the infiltration of *P. gingivalis* into blood vessels which by entering into the blood vessels intima, it can affect the macrophages in that layer and induce LDL endocytosis. This process eventually leads to foam cell formation

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BMC Oral Health

RESEARCH Open Access

A systematic review of the impact of *Porphyromonas gingivalis* on foam cell formation: Implications for the role of periodontitis in atherosclerosis

Saeed Afzoon[1†], Mohammad Amin Amiri[1*†], Mostafa Mohebbi[1], Shahram Hamedani[2] and Nima Farshidfar[3*]

Abstract

Background The current literature suggests the significant role of foam cells in the initiation of atherosclerosis through the formation of a necrotic core in atherosclerotic plaques. Moreover, an important periodontal pathogen called *Porphyromonas gingivalis (P. gingivalis)* is indicated to play a significant role in this regard. Thus, the aim of this systematic review was to comprehensively study the pathways by which *P. gingivalis* as a prominent bacterial species in periodontal disease, can induce foam cells that would initiate the process of atherosclerosis formation.

Methods An electronic search was undertaken in three databases (Pubmed, Scopus, and Web of Science) to identify the studies published from January 2000 until March 2023. The risk of bias in each study was also assessed using the QUIN risk of bias assessment tool.

Results After the completion of the screening process, 11 in-vitro studies met the inclusion criteria and were included for further assessments. Nine of these studies represented a medium risk of bias, while the other two had a high risk of bias. All of the studies have reported that *P. gingivalis* can significantly induce foam cell formation by infecting the macrophages and induction of oxidized low-density lipoprotein (oxLDL) uptake. This process is activated through various mediators and pathways. The most important factors in this regard are the lipopolysaccharide of *P. gingivalis* and its outer membrane vesicles, as well as the changes in the expression rate of transmembrane lipid transportation channels, including transient receptor potential channel of the vanilloid subfamily 4 (TRPV4), lysosomal integral protein 2 (LIMP2), CD36, etc. The identified molecular pathways involved in this process include but are not limited to NF-κB, ERK1/2, p65.

Conclusion Based on the results of this study, it can be concluded that *P. gingivalis* can effectively promote foam cell formation through various pathogenic elements and this bacterial species can affect the expression rate of various

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of bacteria. Shaik–Dasthagirisaheb et al. [50], have shown that the higher the MOI of *P. gingivalis*, the higher rate of foam cell formation. In addition, in another study by Shaik–Dasthagirisaheb et al., it was reported that *P. gingivalis* and *Chlamydia pneumoniae* (*C. pneumonia*) can induce foam cell formation in bone marrow–derived macrophages (BMDMs) in MOI of 100 and 10, respectively [43]. The exposure of these pathogens to LDL–treated BMDM elevated the tumor necrosis factor-α (TNF–α), IL–6, and IL–1β (this factor was exclusively enhanced by *C. pneumonia*) [43]. Similarly, as mentioned in the previous study, LDL downregulates cytokines secretion [50]. The analysis of the cytokine release profile indicated that the cytokine response is not identical for all the pathogens that can cause foam cell formation [43].

Another mechanism concerning the possible effect of *P. gingivalis* on foam cell formation and atherosclerosis is through the suppression of heme oxygenase–1 (HO–1) [47]. This enzyme plays an imperative role in the prevention of vascular inflammation through anti–oxidant, anti–inflammatory, anti–proliferative, anti–apoptotic, and immunomodulatory effects which have shown athero–protective effects [70]. According to Li et al. [47], the HO–1 knockdown results in CD36 and ABCA1 downregulation, and activation of c-Jun-AP/1. In other words, inhibition of HO-1 exacerbates the effect of *P. gingivalis* LPS and aggravates the intracellular lipid content [47].

Aside from the mechanisms mentioned above, another pathway that the authors of this review believe to play a role in this regard is the metabolic changes in macrophages due to *P. gingivalis* infection [71, 72]. *P. gingivalis* can increase the activity of the lactic acid cycle while decreasing oxidative phosphorylation [71, 72]. This process can enhance cellular lactic acid storage, decline mitochondrial oxygen usage, and increase the load of ROS [71, 72]. The enhancement of cellular ROS in macrophages results in a higher rate of lipid oxidation and oxLDL production [71, 72].

This systematic review highlights the importance of periodontal pathogens, especially *P. gingivalis*, in the progression of atherosclerosis which can have significant clinical implications in the long term. Concerning the level of risk of bias, the studies have shown moderate to high levels. Therefore, for future studies in this field, we suggest further well-designed in-vitro studies with low risk of bias and equal in-vitro settings. This would aid future systematic reviews to be able to estimate a predictable correlation between the infection of *P. gingivalis* and the rate of foam cell formation. Moreover, grey literature was not assessed in the current systematic review. Thus, we recommend adding the grey literature in the search strategy of future systematic reviews to provide comprehensive data for screening.

Moreover, according to the compiled outcomes of all the included studies it might be possible to suggest that periodontal treatment procedures could avoid the process of foam cell formation in the arterial intima layer by minimizing the population of *P. gingivalis* in the subgingival plaque area [9, 73]. In this regard, the adjunct application of inflammation-modulatory agents, including nutraceutical agents could also be a treatment option to lessen the severity of the periodontal disease as well [74–77]. In this regard, we strongly recommend further in-vitro, in-vivo, and clinical experiments to assess the reliability of our hypothesis. It is also important to note that the connection of periodontal diseases with atherosclerosis cannot be solely explained based on the effect of *P. gingivalis* on foam cell formation. When interpreting these results, one should be aware that the connection between periodontitis and atherosclerosis is reported to be mediated through various mechanisms, including bacterial species, miRNAs, and so on. Concerning the types of periodontal microbiota *P. gingivalis* is indicated to be the most significant one [73, 78]. On the other hand, the release of certain types of miRNAs into the gingival crevicular fluid can lead to higher susceptibility to cardiovascular diseases by altering gene expression in cardiovascular tissues [79]. Although foam cell formation is central to atherosclerosis, it doesn't involve the whole mechanism of pathogenesis and development of atherosclerosis [80]. Therefore, we can conclude that *P. gingivalis* could contribute to the process of foam cell formation and this periodontal pathogen may enhance the likelihood of developing atherosclerosis.

Conclusion

Our study has shed light on the mechanisms by which *P. gingivalis* can promote the process of foam cell formation. Based on the gathered evidence, *P. gingivalis* affects the macrophages' environment, their gene expression patterns, and cellular mechanisms through which macrophages enhance their lipid uptake and transform into foam cells. The changes in the environment include the effect of *P. gingivalis* on endothelial cells to gather more monocytes to the site and changes in the mechanical and biological properties of the ECM. Moreover, the changes in the gene expression patterns in macrophages can outweigh the equilibrium of lipid transportation into more lipid influx and less lipid efflux. Besides, *P. gingivalis* leads the process of foam cell formation through various cellular mechanisms, including pro-inflammatory cytokines secretion, modification of LDL and HDL, ignition of various cellular signaling pathways, and cell receptor activities. All these processes are ascribed to four marked characteristics in *P. gingivalis*, including MOI of *P. gingivalis*, *P. gingivalis* LPS, *P. gingivalis* major fimbria, and *P. gingivalis* OMV which have demonstrated significant

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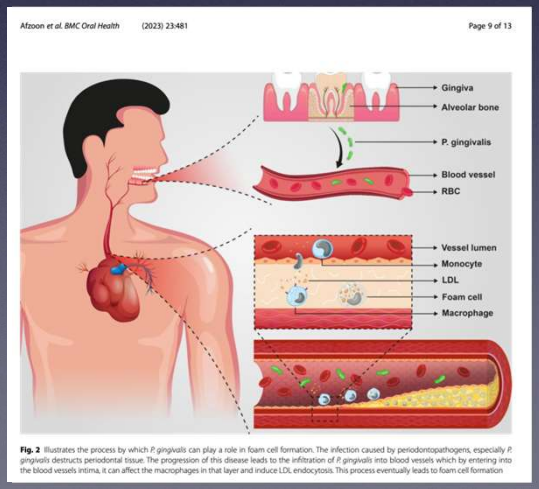
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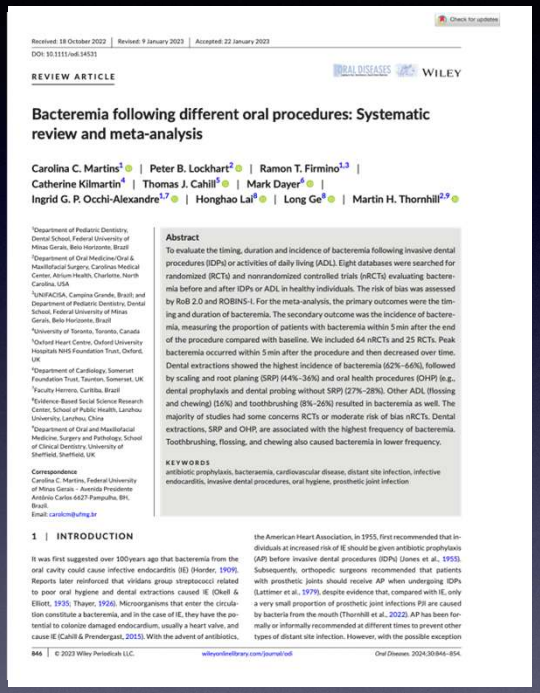
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REVIEW ARTICLE

ORAL DISEASES WILEY

Bacteremia following different oral procedures: Systematic review and meta-analysis

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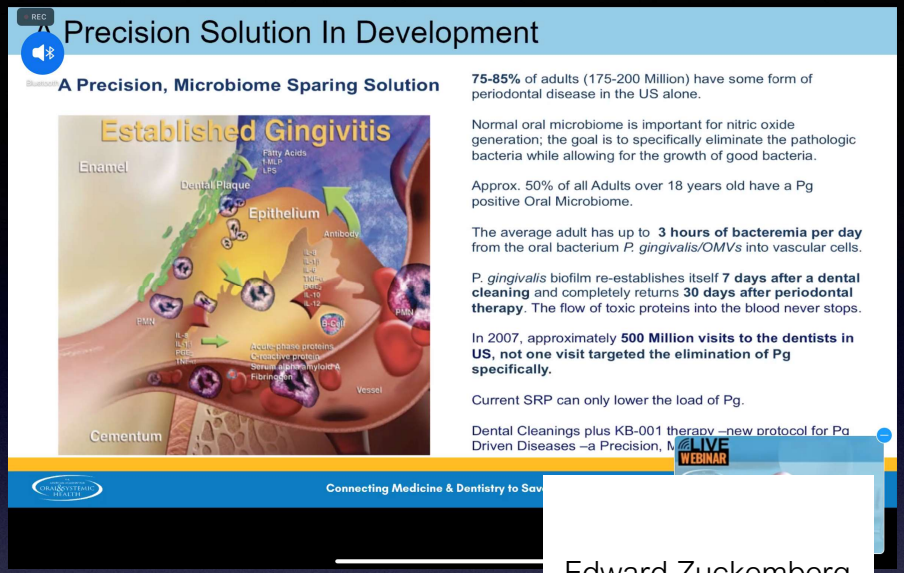
Abstract

To evaluate the timing, duration and incidence of bacteremia following invasive dental procedures (IDPs) or activities of daily living (ADL). Eight databases were searched for randomized (RCTs) and nonrandomized controlled trials (nRCTs) evaluating bacteremia before and after IDPs or ADL in healthy individuals. The risk of bias was assessed by RoB 2.0 and ROBINS-I. For the meta-analysis, the primary outcomes were the timing and duration of bacteremia. The secondary outcome was the incidence of bacteremia, measuring the proportion of patients with bacteremia within 5 min after the end of the procedure compared with baseline. We included 64 nRCTs and 25 RCTs. Peak bacteremia occurred within 5 min after the procedure and then decreased over time. Dental extractions showed the highest incidence of bacteremia (62%–66%), followed by scaling and root planing (SRP) (44%–36%) and oral health procedures (OHP) (e.g., dental prophylaxis and dental probing without SRP) (27%–28%). Other ADL (flossing and chewing) (16%) and toothbrushing (8%–26%) resulted in bacteremia as well. The majority of studies had some concerns RCTs or moderate risk of bias nRCTs. Dental extractions, SRP and OHP, are associated with the highest frequency of bacteremia. Toothbrushing, flossing, and chewing also caused bacteremia in lower frequency.

KEYWORDS
antibiotic prophylaxis, bacteraemia, cardiovascular disease, distant site infection, infective endocarditis, invasive dental procedures, oral hygiene, prosthetic joint infection

1 | INTRODUCTION

It was first suggested over 100 years ago that bacteremia from the oral cavity could cause infective endocarditis (IE) (Horder, 1909). Reports later reinforced that viridans group streptococci related to poor oral hygiene and dental extractions caused IE (Okell & Elliott, 1935; Thayer, 1926). Microorganisms that enter the circulation constitute a bacteremia, and in the case of IE, they have the potential to colonize damaged endocardium, usually a heart valve, and cause IE (Cahill & Prendergast, 2015). With the advent of antibiotics, the American Heart Association, in 1955, first recommended that individuals at increased risk of IE should be given antibiotic prophylaxis (AP) before invasive dental procedures (IDPs) (Jones et al., 1955). Subsequently, orthopedic surgeons recommended that patients with prosthetic joints should receive AP when undergoing IDPs (Lattimer et al., 1979), despite evidence that, compared with IE, only a very small proportion of prosthetic joint infections PJI are caused by bacteria from the mouth (Thornhill et al., 2022). AP has been formally or informally recommended at different times to prevent other types of distant site infection. However, with the possible exception

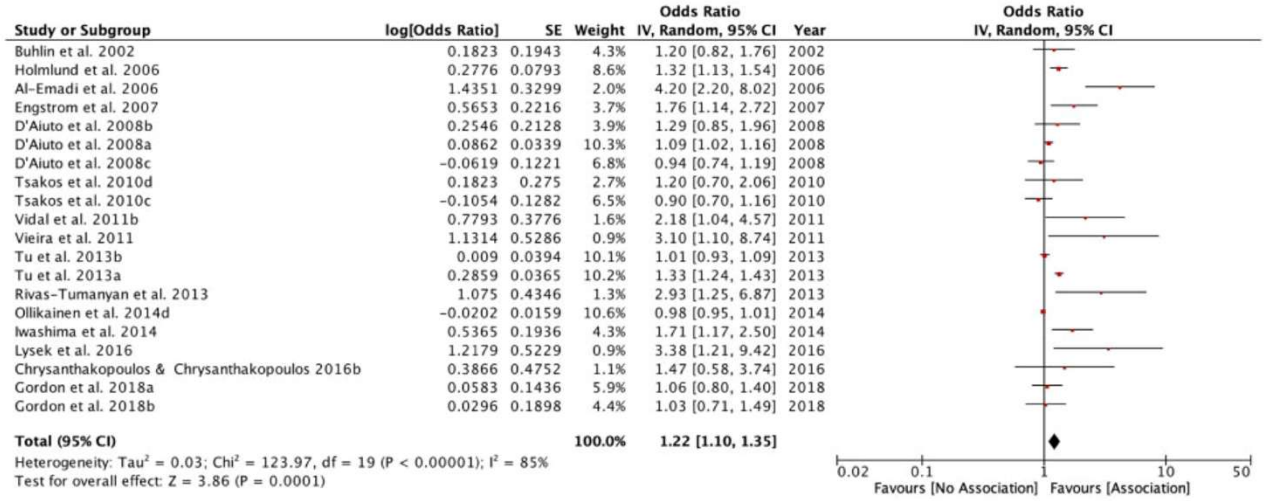


American Academy for ORAL & SYSTEMIC HEALTH

2023

Periodontitis is associated with hypertension: a systematic review and meta-analysis

Eva Muñoz Aguilera [1,2], Jean Suvan[1], Jacopo Buti [1], Marta Czesnikiewicz-Guzik[3,4,5,6], Aline Barbosa Ribeiro [3,4,7], Marco Orlandi[1], Tomasz J. Guzik[3,4,5,6], Aroon D. Hingorani[8], Jose Nart [2], and Francesco D'Aiuto [1]*

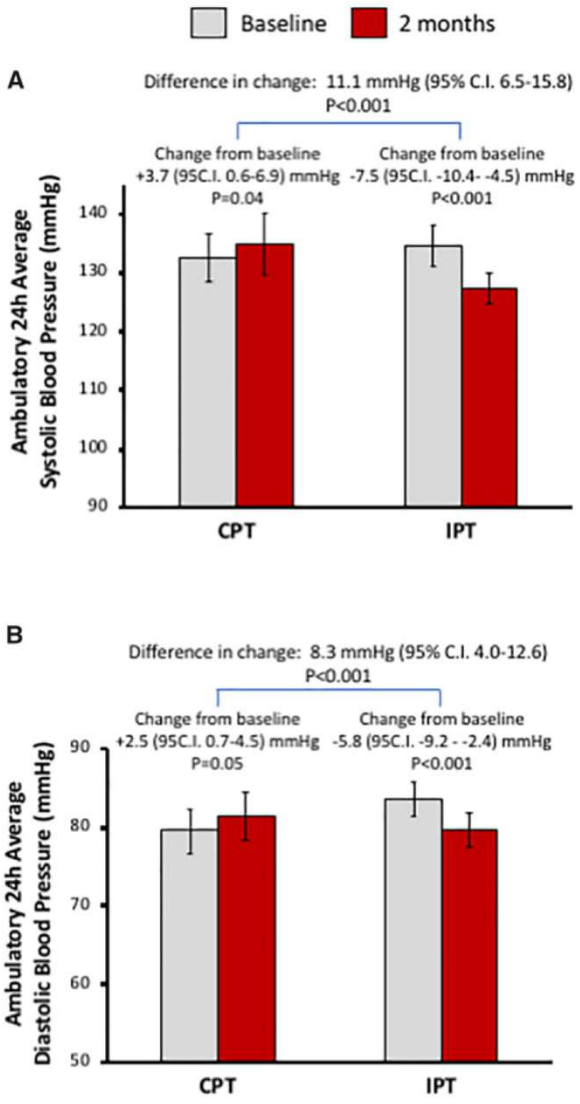


Study or Subgroup	log[Odds Ratio]	SE	Weight	Odds Ratio IV, Random, 95% CI	Year
Buhlin et al. 2002	0.1823	0.1943	4.3%	1.20 [0.82, 1.76]	2002
Holmlund et al. 2006	0.2776	0.0793	8.6%	1.32 [1.13, 1.54]	2006
Al-Emadi et al. 2006	1.4351	0.3299	2.0%	4.20 [2.20, 8.02]	2006
Engstrom et al. 2007	0.5653	0.2216	3.7%	1.76 [1.14, 2.72]	2007
D'Aiuto et al. 2008b	0.2546	0.2128	3.9%	1.29 [0.85, 1.96]	2008
D'Aiuto et al. 2008a	0.0862	0.0339	10.3%	1.09 [1.02, 1.16]	2008
D'Aiuto et al. 2008c	-0.0619	0.1221	6.8%	0.94 [0.74, 1.19]	2008
Tsakos et al. 2010d	0.1823	0.275	2.7%	1.20 [0.70, 2.06]	2010
Tsakos et al. 2010c	-0.1054	0.1282	6.5%	0.90 [0.70, 1.16]	2010
Vidal et al. 2011b	0.7793	0.3776	1.6%	2.18 [1.04, 4.57]	2011
Vieira et al. 2011	1.1314	0.5286	0.9%	3.10 [1.10, 8.74]	2011
Tu et al. 2013b	0.009	0.0394	10.1%	1.01 [0.93, 1.09]	2013
Tu et al. 2013a	0.2859	0.0365	10.2%	1.33 [1.24, 1.43]	2013
Rivas-Tumanyan et al. 2013	1.075	0.4346	1.3%	2.93 [1.25, 6.87]	2013
Ollikainen et al. 2014d	-0.0202	0.0159	10.6%	0.98 [0.95, 1.01]	2014
Iwashima et al. 2014	0.5365	0.1936	4.3%	1.71 [1.17, 2.50]	2014
Lysek et al. 2016	1.2179	0.5229	0.9%	3.38 [1.21, 9.42]	2016
Chrysanthakopoulos & Chrysanthakopoulos 2016b	0.3866	0.4752	1.1%	1.47 [0.58, 3.74]	2016
Gordon et al. 2018a	0.0583	0.1436	5.9%	1.06 [0.80, 1.40]	2018
Gordon et al. 2018b	0.0296	0.1898	4.4%	1.03 [0.71, 1.49]	2018
Total (95% CI)			**100.0%**	**1.22 [1.10, 1.35]**	

Heterogeneity: $Tau^2 = 0.03$; $Chi^2 = 123.97$, df = 19 ($P < 0.00001$); $I^2 = 85\%$
Test for overall effect: Z = 3.86 (P = 0.0001)

Figure 2 Association between periodontitis (moderate to severe combined diagnosis) and hypertension (cross-sectional and case–control studies). Summary Forrest plot for odds ratio of hypertension in relation to periodontitis diagnosis in cross-sectional and case–control studies (moderate to severe combined diagnosis). The random effects model was used and the relative size of the data markers indicates the weight of the sample size from each study. CI, confidence interval; IV, inverse variance; SE, standard error.

UN TRATTAMENTO PERIODONTALE INTENSIVO (IPT) CAUSA UN CALO DI 7.5 MMHG DELLA PRESSIONE ARTERIOSA SISTOLICA MEDIA ED UN CALO DI 5.8 MMHG DELLA PRESSIONE ARTERIOSA DIASTOLICA MEDIA



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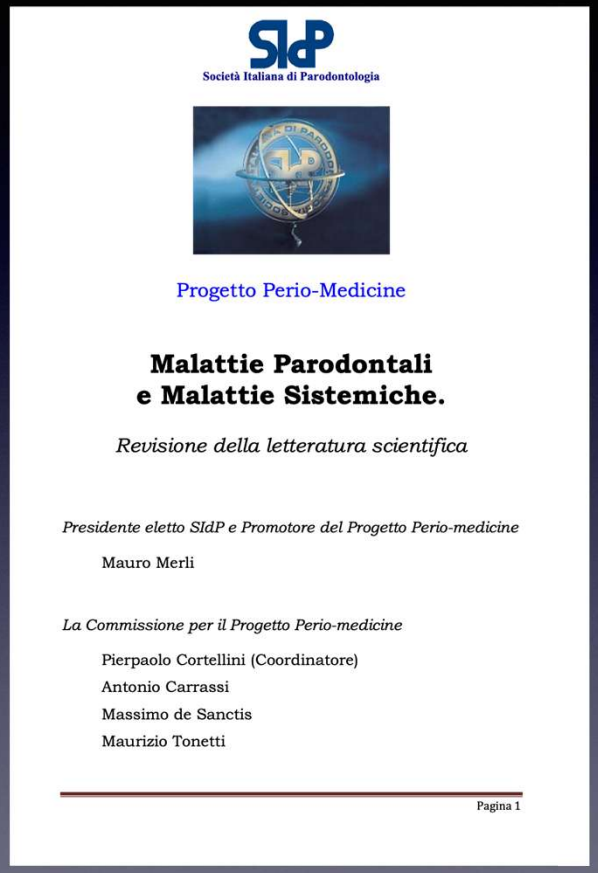

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Progetto Perio-Medicine

Malattie Parodontali e Malattie Sistemiche.

Revisione della letteratura scientifica

Presidente eletto SIdP e Promotore del Progetto Perio-medicine

Mauro Merli

La Commissione per il Progetto Perio-medicine

Pierpaolo Cortellini (Coordinatore)
Antonio Carrassi
Massimo de Sanctis
Maurizio Tonetti

Pagina 1

Studi su marker sierologici

La proteina serica c-reattiva ad alta sensitività (hsC-rp) è un marker non specifico dell'infiammazione.

Livelli sierici di hsC-rp di 42.0 mg/l sono indicatori di infiammazione ed indicano un rischio elevato di malattia cardiovascolare insieme ad altri fattori di rischio (43).

Valori elevati di proteina C-reativa sono stati anche associati con altre malattie(44,45).

Studi recenti hanno suggerito che la parodontite può indurre elevati livelli sierici di protineaC-reattiva (46,47,48,49). Inoltre, la presenza di patogeni parodontali è stata associata con livelli elevati di hsCrp (47,50). In un articolo recente, (51) è stato evidenziato che la concentrazione serica di hsC-rp ed il numero di linfociti nel siero sono associati con la sindrome coronarica acuta.Valori elevati di hsCrp sierici sono associati con parodontite definita utilizzando parametri radiografici in soggetti senza evidenza di malattia cardiovascolare.

D'Aiuto et al (52) ha evidenziato che la riduzione dei livelli plasmatici di proteina C-reattiva sei mesi dopo terapia attiva parodontale erano associati significativamente al numero di denti estratti.

Lo stesso gruppo ha recentemente riportato dati a sei mesi sull'effetto di una terapia parodontale standard vs terapia aggressiva (53).

In confronto con la situazione di base il gruppo con terapia intensiva mostrava una significativa riduzione nei valori quali conta dei linfociti,livelli di CRP,IL-6,colesterolo,LDL e pressione sistolica, mentre nel gruppo sottoposto a terapia standard si rilevava un incremento delle HDL.

In un altro studio (54), pazienti con almeno due denti con sondaggi superiori a 6mm mostravano una significativa riduzione nei livelli di CRP dopo l'estrazione di tutti i denti. D'altra parte non sono stati riportati effetti significativi sulla proteina C–reattiva sierica come risultato della terapia parodontale (55).

Tre lavori (56,57,58) hanno valutato l'effetto della terapia parodontale sulla disfunzione endoteliale ,questa è caratterizzata da una ridotta capacità di vasodilatazione della vascolatura periferica.Questa disfunzione è stata associata ad un aumentato rischio di arterosclerosi nei pazienti sani.

La terapia parodontale non chirurgica e la full mouth disinfection hanno dimostrato di produrre un effetto positivo sulla capacità vasodilatatoria mediata dall'endotelio della vascolatura periferica.

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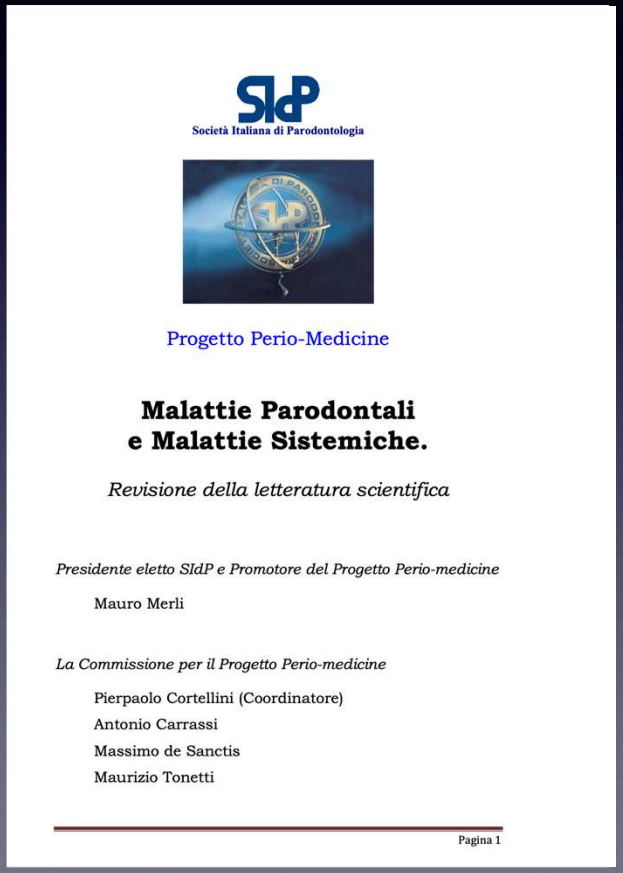

Società Italiana di Parodontologia

Progetto Perio-Medicine

Malattie Parodontali
e Malattie Sistemiche.

Revisione della letteratura scientifica

Presidente eletto SIdP e Promotore del Progetto Perio-medicine

Mauro Merli

La Commissione per il Progetto Perio-medicine

Pierpaolo Cortellini (Coordinatore)
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Maurizio Tonetti

Pagina 1

I meccanismi che vengono indicati come più probabili per spiegare l'"effetto della parodontite sulla genesi dell'"aterosclerosi sono:

Quelli diretti che prevedono la partecipazione di batteri patogeni parodontali nella genesi della pacca ateromatosa , dovuta probabilmente alla translocazione dei patogeni parodontali dal cavo orale al sistema circolatorio.

Quelli indiretti, che indicano invece nella grande produzione locale di mediatori dell'"infiammazione nella lesione parodontale che potrebbero entrare in circolo, la causa dei danni vascolari a distanza. In effetti nel siero dei pazienti parodontali si rilevano elevati livelli serici di citochine pro-infiammatorie e questi mediatori sono considerati markers della malattia cardiovascolare.

Un'"altra strada possibile, potrebbe essere relativa alla stimolazione di una risposta autoimmunitaria a causa dell'"elevata somiglianza tra peptidi antigeni di origine batterica e proteine umane.

Le evidenze epidemiologiche che si sono accumulate negli anni inoltre, indicano che i markers clinici, microbiologici e sierologici dell'"infezione parodontale sono associati con le malattie cardiovascolari sia nelle nelle forme clinicamente evidenti che in quelle subcliniche .

2021

Documento dedotto dal Consensus intersocietario tra:

SOCIETÀ ITALIANA DI CARDIOLOGIA

A.N.M.C.O. ASSOCIAZIONE NAZIONALE MEDICI CARDIOLOGIA OSPEDALIERI

SOCIETA' ITALIANA DI CHIRURGIA CARDIACA SINCE 1967

AIE ACCADEMIA ITALIANA ENDODONZIA

SIE Società Italiana di Endodonzia

SIdP Società Italiana di Parodontologia e Implantologia

Lo screening dentale preoperatorio nei pazienti in attesa di interventi chirurgici cardiovascolari elettivi: presentazione del documento di consenso intersocietario SIC-ANMCO-SICCH-AIE-SIE-SidP

Elisabetta Cotti[1], Francesco Cairo[2], Pier Paolo Bassareo[3], Federica Fonzar[4], Mauro Venturi[5], Luca Landi[6], Alessandro Parolari[7], Vittorio Franco[8], Cristiano Fabiani[9], Fabio Barili[10], Andrea Di Lenarda[11], Michele Massimo Gulizia[12], Mauro Bonizzi[13], Guglielmo Campus[14], Francesco Musumeci[15], Giuseppe Mercuro[15]

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[3]University College of Dublin, Mater Misericordiae University Hospital, Dublino, Irlanda
[4]Medico Chirurgo, Libero Professionista, Udine
[5]Medico Chirurgo, Libero Professionista, Bologna
[6]Medico Chirurgo, Libero Professionista, Verona e Roma
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[8]Medico Chirurgo, Libero Professionista, Roma
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[10]S.C. Cardiovascolare e Medicina dello Sport, Ospedale Maggiore di Trieste, ASUI Trieste
[11]U.O.C. Cardiologia, Ospedale Garibaldi-Nesima, Azienda di Rilievo Nazionale e Alta Specializzazione "Garibaldi", Catania
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Lo **screening dentale** preoperatorio nei pazienti in attesa di **interventi chirurgici cardiovascolari elettivi**

Punti chiave

- È fondamentale conoscere la prognosi complessiva del soggetto al fine di valutare il rischio per il paziente e la conseguente fattibilità del trattamento odontoiatrico.
- Il piano di trattamento odontoiatrico è subordinato al tempo a disposizione prima dell'intervento cardiovascolare.
- In caso di intervento cardiovascolare non urgente, il chirurgo dovrebbe inviare il paziente per lo screening odontoiatrico in concomitanza alla programmazione dell'intervento.
- Nel caso in cui non vi sia tempo per la terapia odontoiatrica, o questa sia sconsigliata per i fattori di rischio del paziente, è necessario che l'odontoiatra comunichi al chirurgo la presenza e la sede di eventuali foci orali.
- La terapia parodontale non chirurgica per il controllo dell'infezione orale è indicata anche quando il tempo a disposizione prima dell'intervento è inferiore a 6 mesi, se le condizioni generali del paziente lo consentono.
- La terapia endodontica è indicata per la risoluzione della sintomatologia anche quando il tempo a disposizione prima dell'intervento sia inferiore a 6 mesi, mentre la valutazione della guarigione dell'infezione periradicolare richiede un tempo più lungo.

Considerazioni conclusive

Gli autori auspicano che i contenuti di questa brochure, quale sintesi del più ampio e citato Documento di Consenso Intersocietario, vengano adottati dai Clinici e dagli Odontoiatri per la realizzazione di percorsi diagnostico-terapeutici intraospedalieri congiunti tra Cardiologi, Cardiochirurghi e Odontoiatri, finalizzati alla minimizzazione del rischio nel paziente con indicazione a intervento in elezione sul sistema cardiovascolare.

Referenze

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DentalSchool UniSR

2021

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Punti chiave

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SIE Società Italiana di Endodonzia

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PAZIENTI IPERTESI A RISCHIO DI PARODONTITE

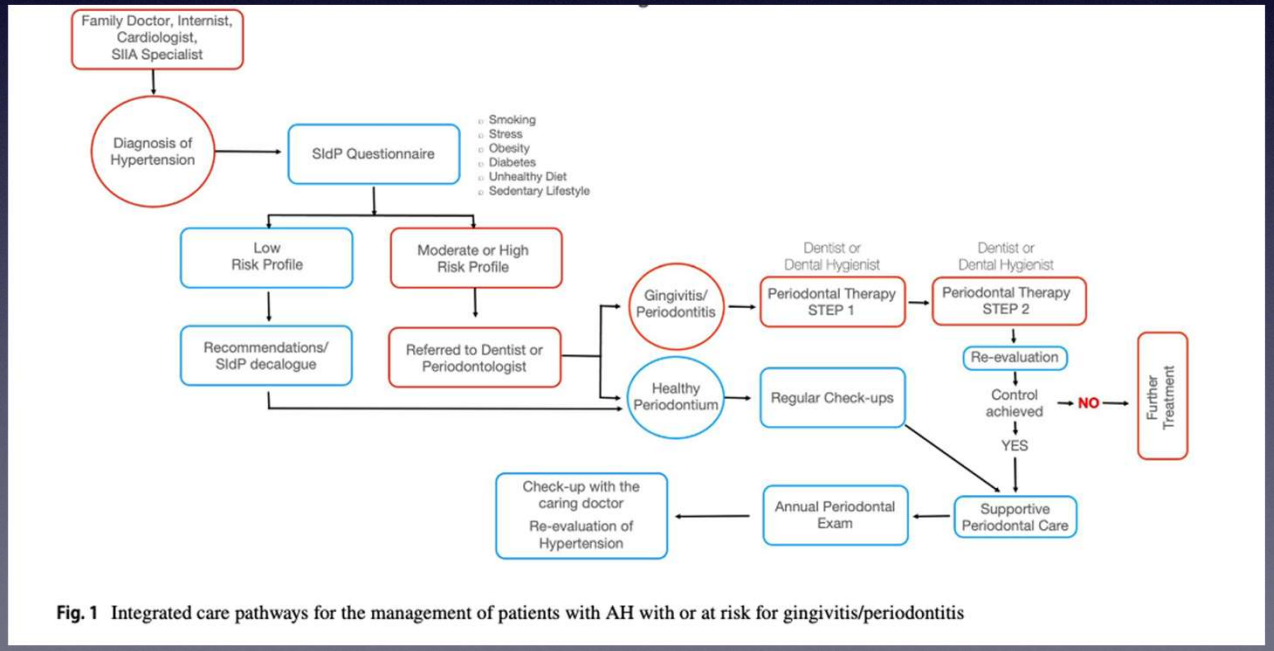
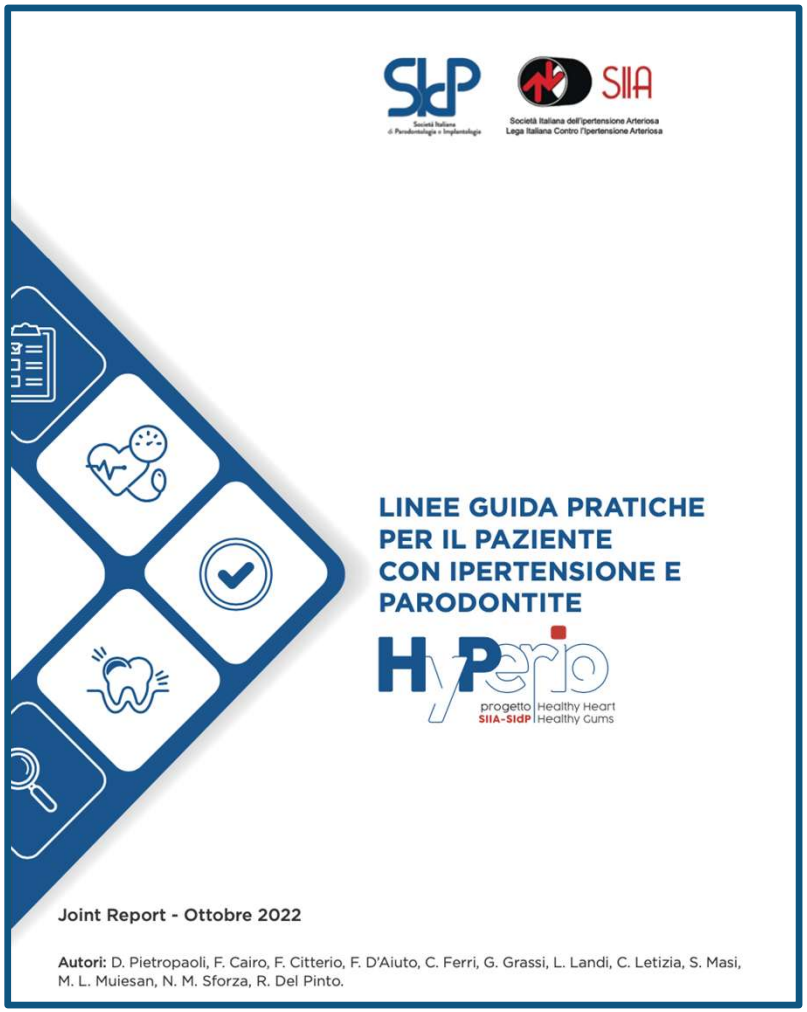
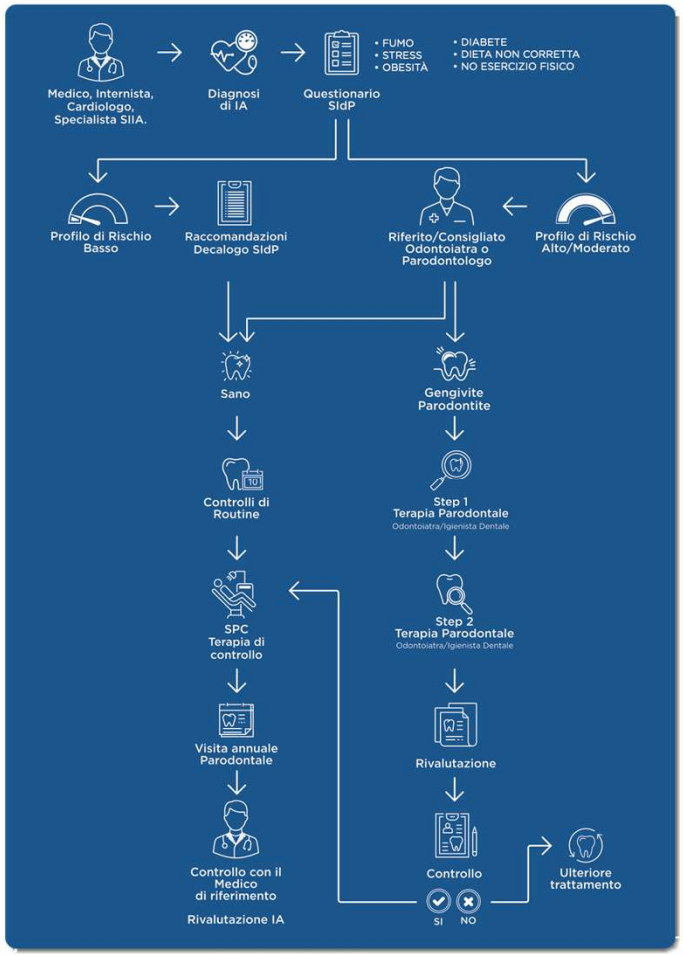


Fig. 1 Integrated care pathways for the management of patients with AH with or at risk for gingivitis/periodontitis

APPROCCIO CLINICO INTEGRATO

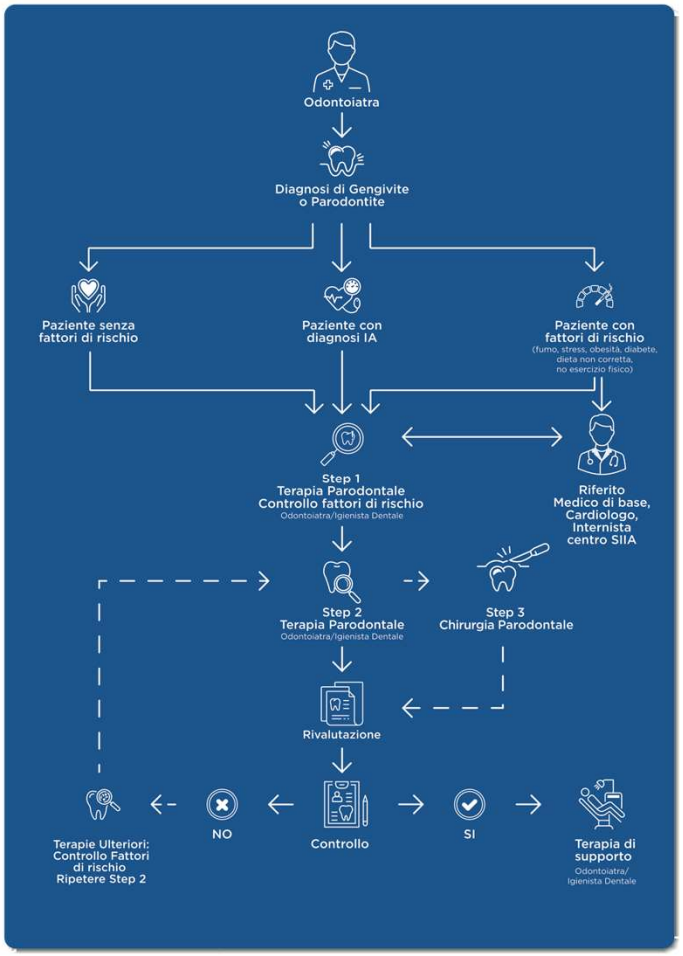


PDTA
PAZIENTE CON IPERTENSIONE ARTERIOSA (IA)
CON/O A RISCHIO DI GENGIVITE O PARODONTITE



PER MAGGIORI INFO: www.gengive.org

PDTA
PAZIENTE CON GENGIVITE/PARODONTITE
IPERTESO O A RISCHIO IPERTENSIONE (IA)



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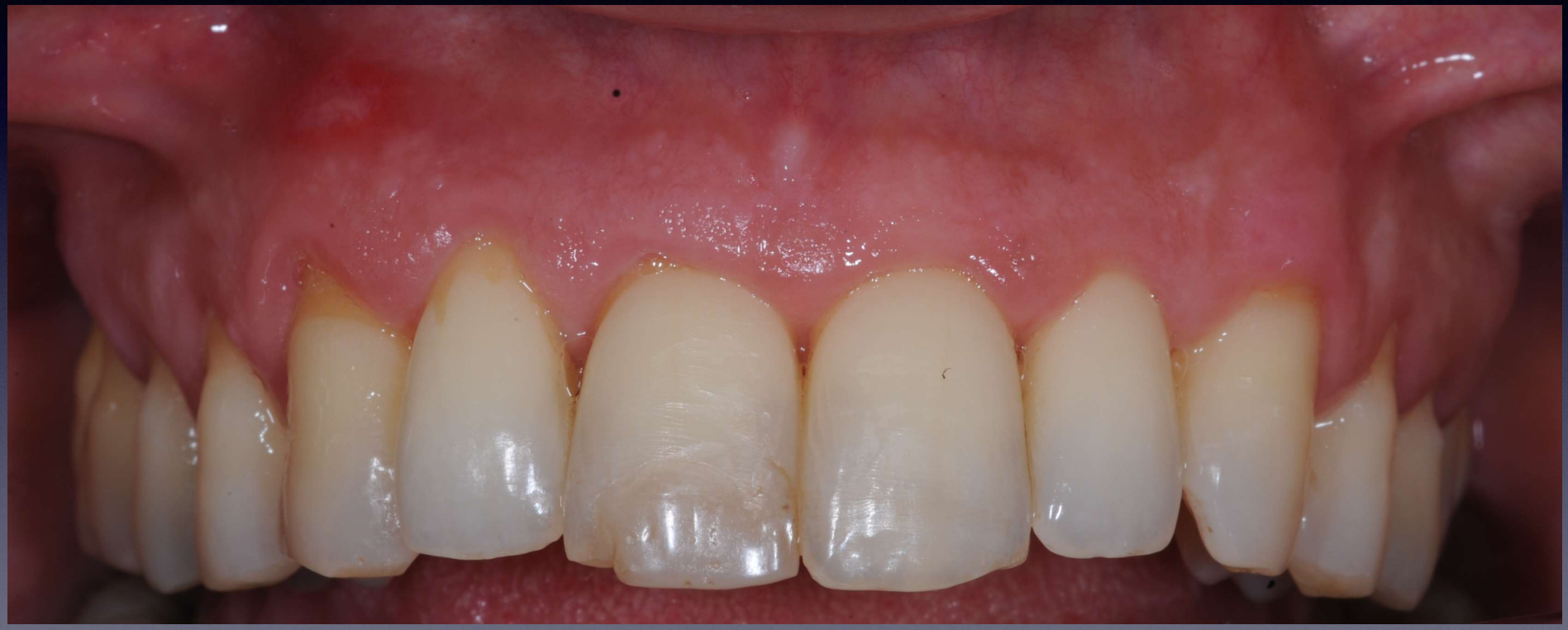

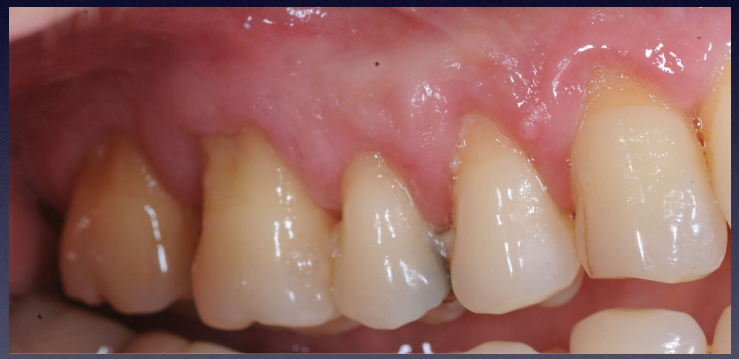

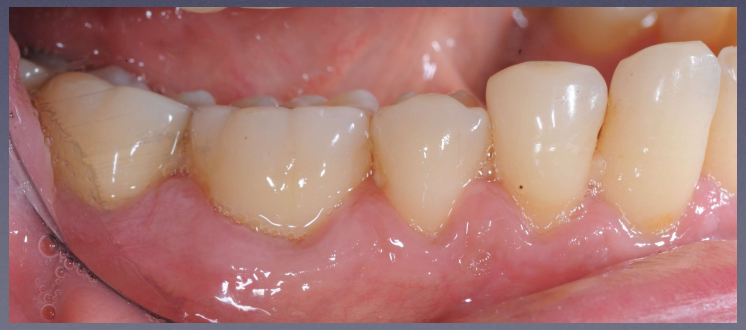

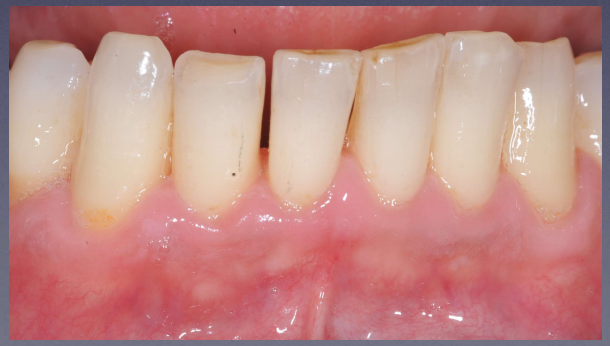

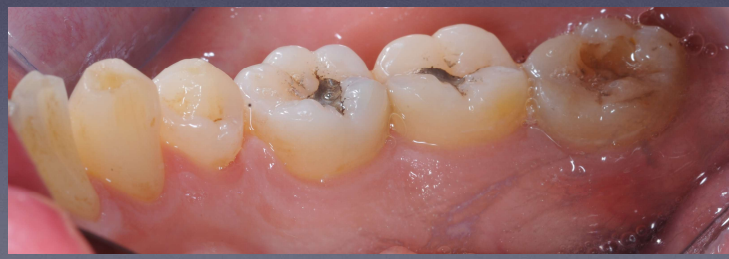

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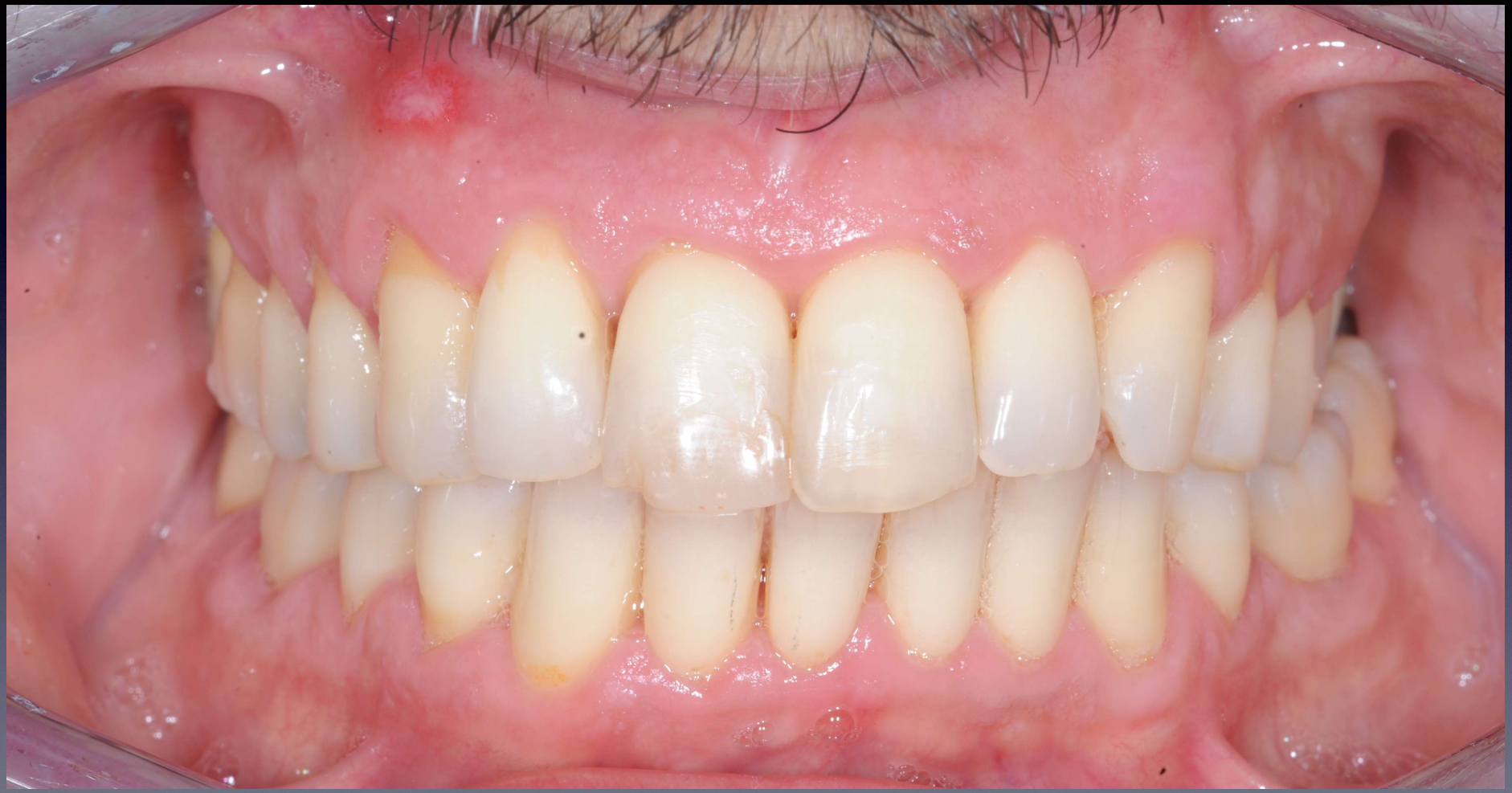
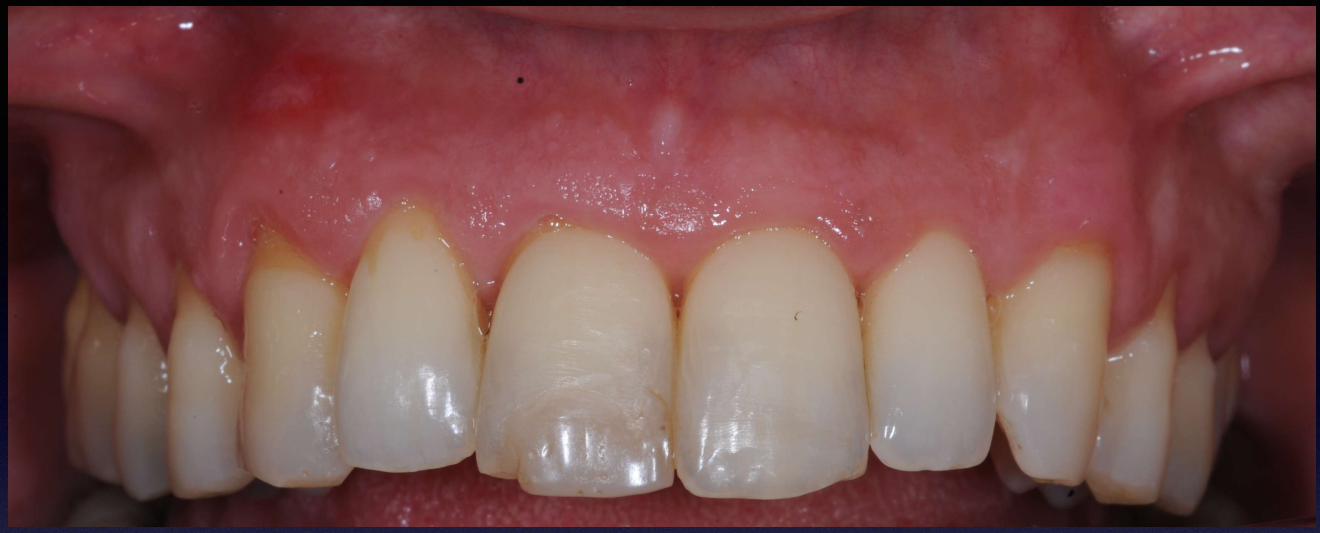
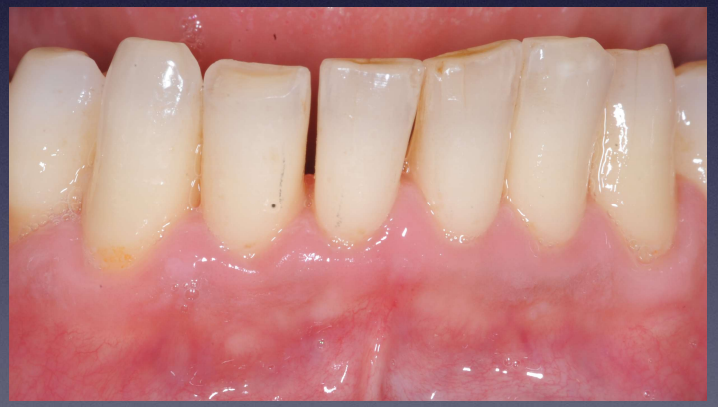
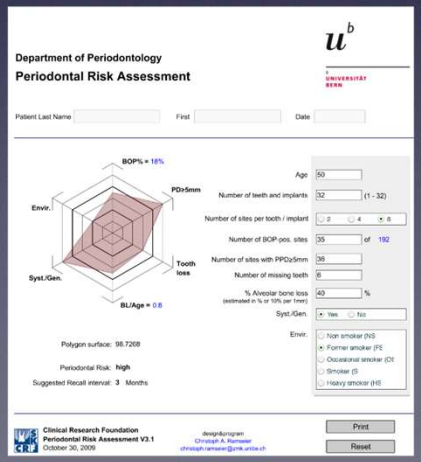
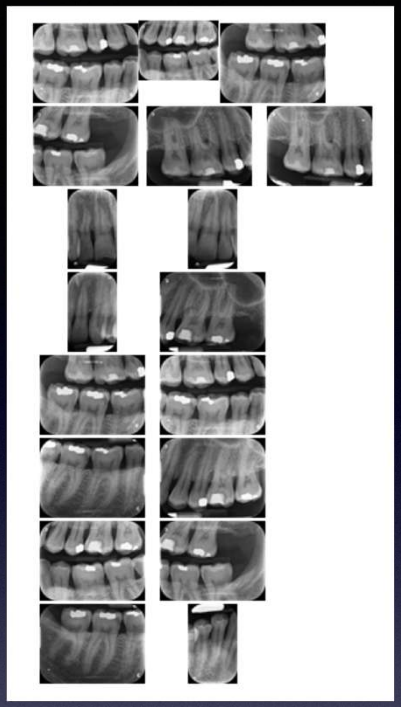
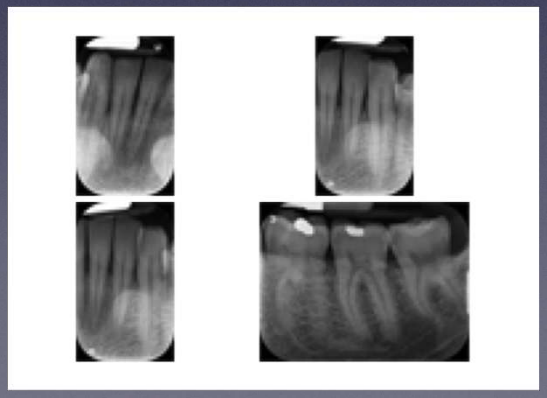


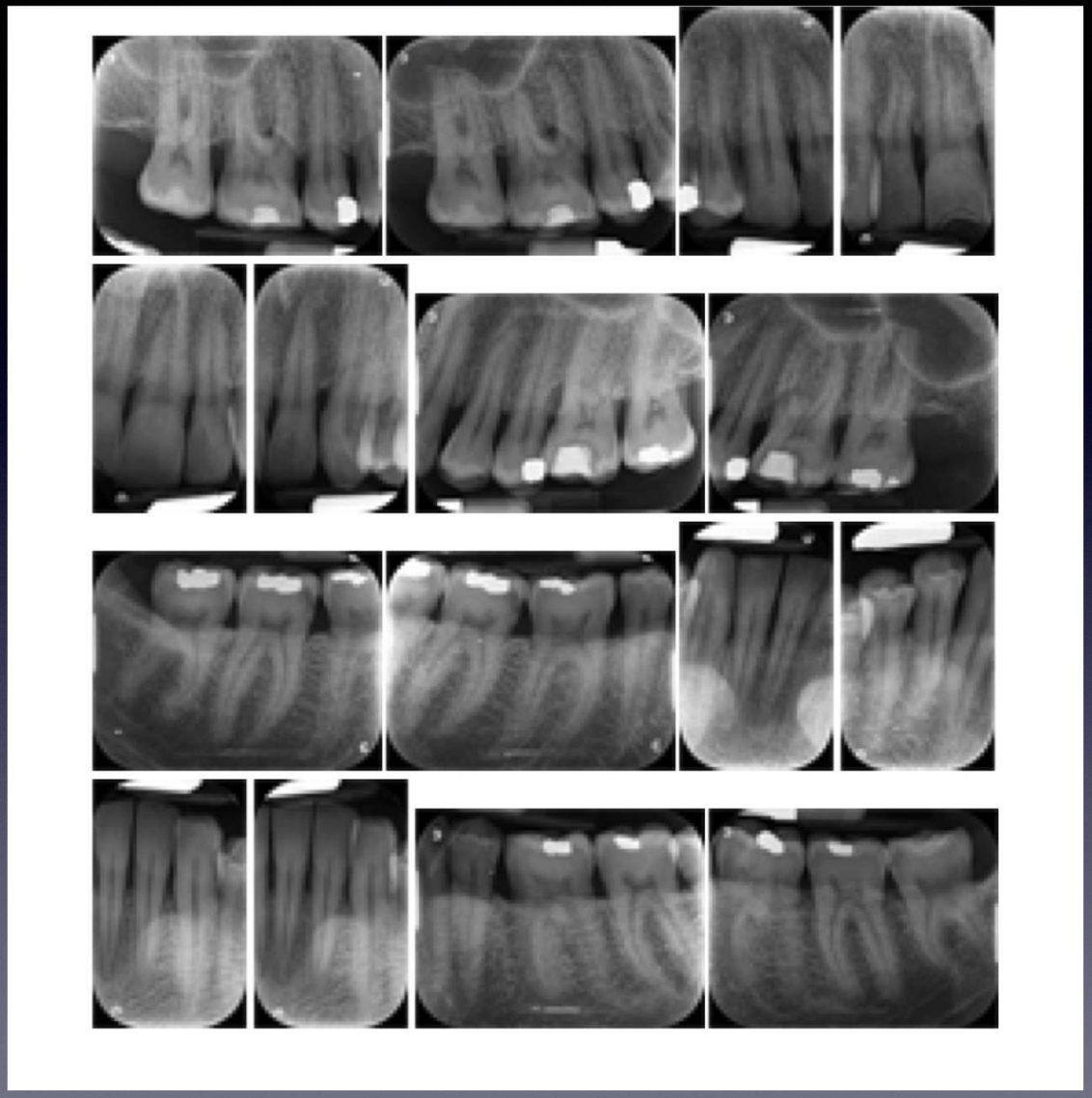


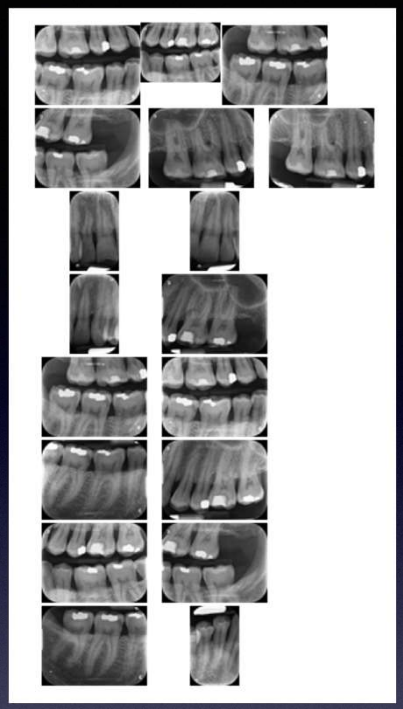


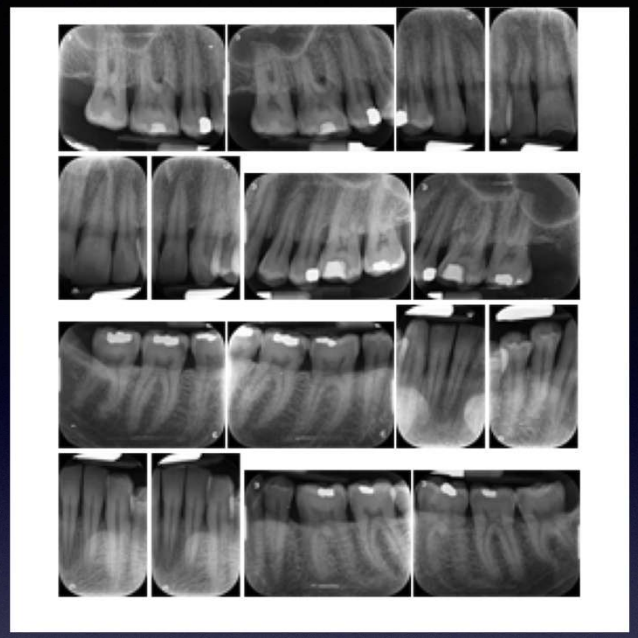

Department of Periodontology
Periodontal Risk Assessment
UNIVERSITÄT BERN
Patient Last Name
First
Date
BOP% = 18%
PD≥5mm
Envir.
Tooth loss
Syst./Gen.
BL/Age = 0.8
Age 50
Number of teeth and implants 32 (1 - 32)
Number of sites per tooth / implant 2 4 6
Number of BOP pos. sites 35 of 192
Number of sites with PPD≥5mm 36
Number of missing teeth 6
% Alveolar bone loss 40 %
(estimated in % or 10% per 1mm)
Syst./Gen. Yes No
Envir.
Non smoker (NS
Former smoker (FS
Occasional smoker (OS
Smoker (S
Heavy smoker (HS
Polygon surface: 98.7268
Periodontal Risk: high
Suggested Recall interval: 3 Months
Clinical Research Foundation
Periodontal Risk Assessment V3.1
October 30, 2009
design&program
Christoph A. Ramseier
christoph.ramseier@zmk.unibe.ch
Print
Reset

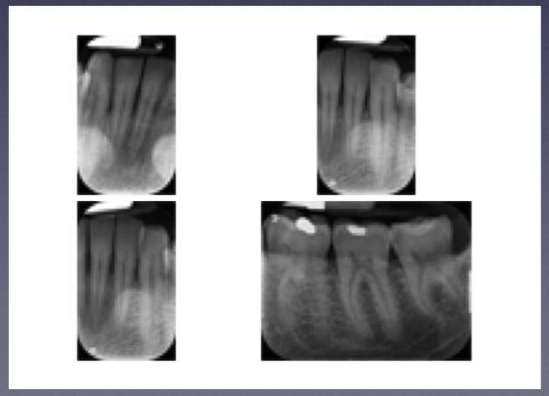












Reduction in Probing Pocket Depth (PPD) measured in millimeter.

Absence of residual Pocket:

PPD<4mm.

PPD of 5mm. NOT BLEEDING

Should be achieved.

ORDINE PROVINCIALE
DEI MEDICI CHIRURGHI
E DEGLI ODONTOIATRI DI
BOLOGNA

Provider Nazionale n. 3744

CUORE E PARODONTOPATIE

LA SALUTE ORALE ALLUNGA LA VITA

Mercoledì 18 febbraio 2026
ore 20:00 – 22:30

Sala Conferenze, Ordine Provinciale dei Medici-Chirurghi e degli Odontoiatri di Bologna
Via Giovanna Zaccherini Alvisi n. 4 – Bologna

Responsabile Scientifico: Maurizio Baroni

19.45-20.00	*Registrazione partecipanti*
20.00-20.10	*Presentazione del Convegno* **Maurizio Baroni** *Moderano* **Carlo Prati e Giuseppe Trisolino**
20.10-20.30	*La malattia parodontale e le Linee Guida Intersocietarie* **Marco Montevecchi**
20.30-20.50	*Aspetti clinici, terapeutici e patogenetici della gengivite e parodontite* **Claudio Cesari**
20.50-21.10	*Malattie cardiovascolari e parodontopatie: quale associazione* **Maurizio Baroni**
21.10-21.30	*Ipertensione arteriosa e parodontite: approccio clinico* **Renzo Roncuzzi**
21.30-22.00	*Discussione*
22.00-22.30	*Questionario ECM e qualità percepita*

Destinatari dell'attività formativa: Medici-Chirurghi (Medico Generico e tutte le discipline) - Odontoiatri
Obiettivo formativo: contenuti tecnico-professionali (conoscenze e competenze) specifici di ciascuna professione, di ciascuna specializzazione e di ciascuna attività ultraspecialistica, ivi incluse le malattie rare e la medicina di genere (18) - Crediti ECM attribuiti: n. 2

Segue Faculty

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Apertura iscrizioni il 29/01/2026 ore 11:00 - Chiusura iscrizioni il 16/02/2026
Segreteria organizzativa: ecm@odmbologna.it

Thank You for Your kind attention

Dr. **Claudio Cesari**

Adjunct Professor in Periodontology
Dept. Periodontology, Prof. Roberto Rotundo
University Vita e Salute SAN RAFFAELE, MILAN

Head of Periodontal UNIT
OSPEDALE SAN ROCCO, Ome (BS)
Dept. Oral and Maxillo-Facial,
Prof. Giorgio Gastaldi

c.cesari1@docenti.unisr.it