

# PERIODONTAL DISEASES & CARDIOVASCULAR SYSTEM

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# The Link Between Heart & Gum Diseases

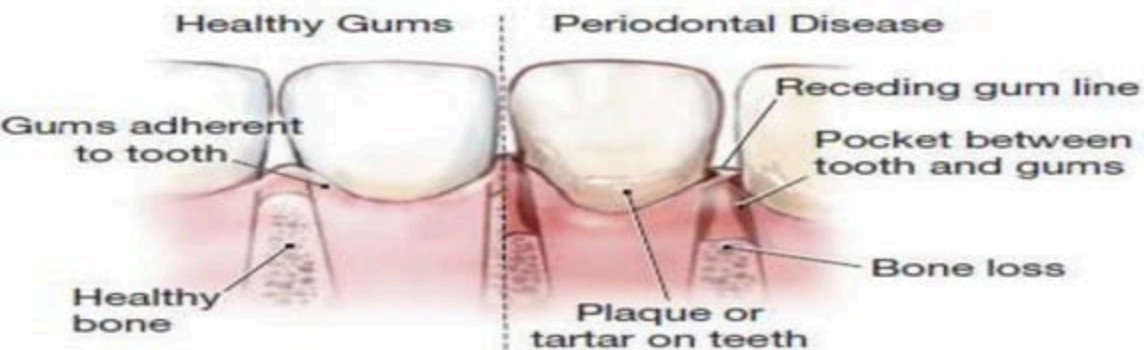
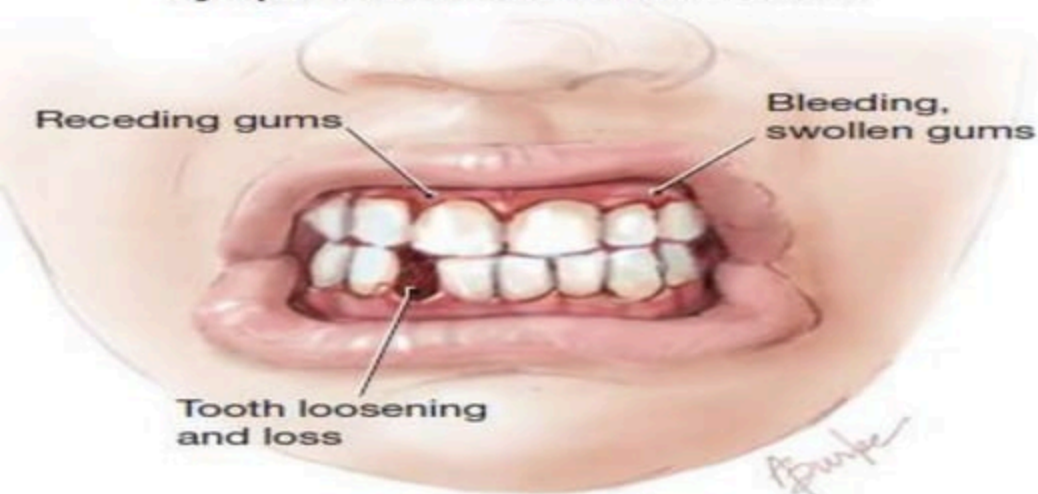


# INTRODUCTION

- **Periodontal disease** is an infectious disease but environmental, physical, social & host stresses may affect & modify disease expression.
- Evidences also show the converse sides of relationship between systemic health & oral health i.e., the potential effect of periodontal disease on a wide range of organ system.



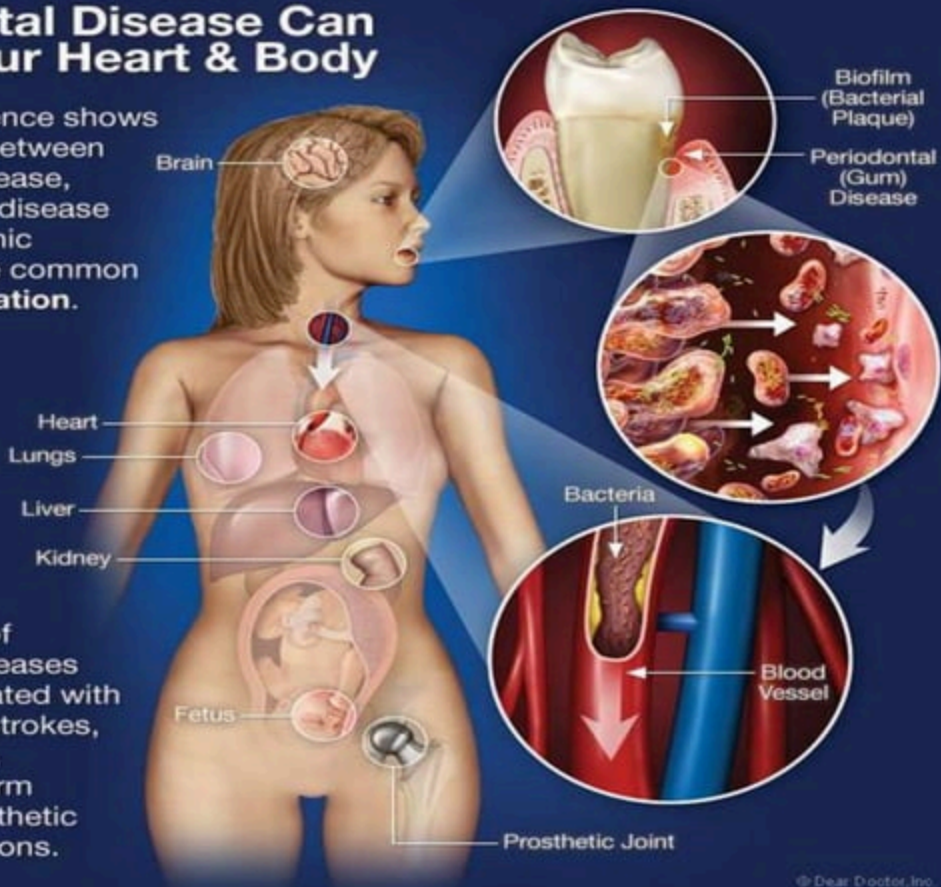
## Symptoms of Periodontal Disease



# Periodontal Disease Can Affect Your Heart & Body

Emerging evidence shows a relationship between periodontal disease, cardiovascular disease and other chronic diseases — the common link is **inflammation**.

The presence of periodontal diseases may be associated with heart attacks, strokes, kidney disease, diabetes, preterm births and prosthetic joint complications.

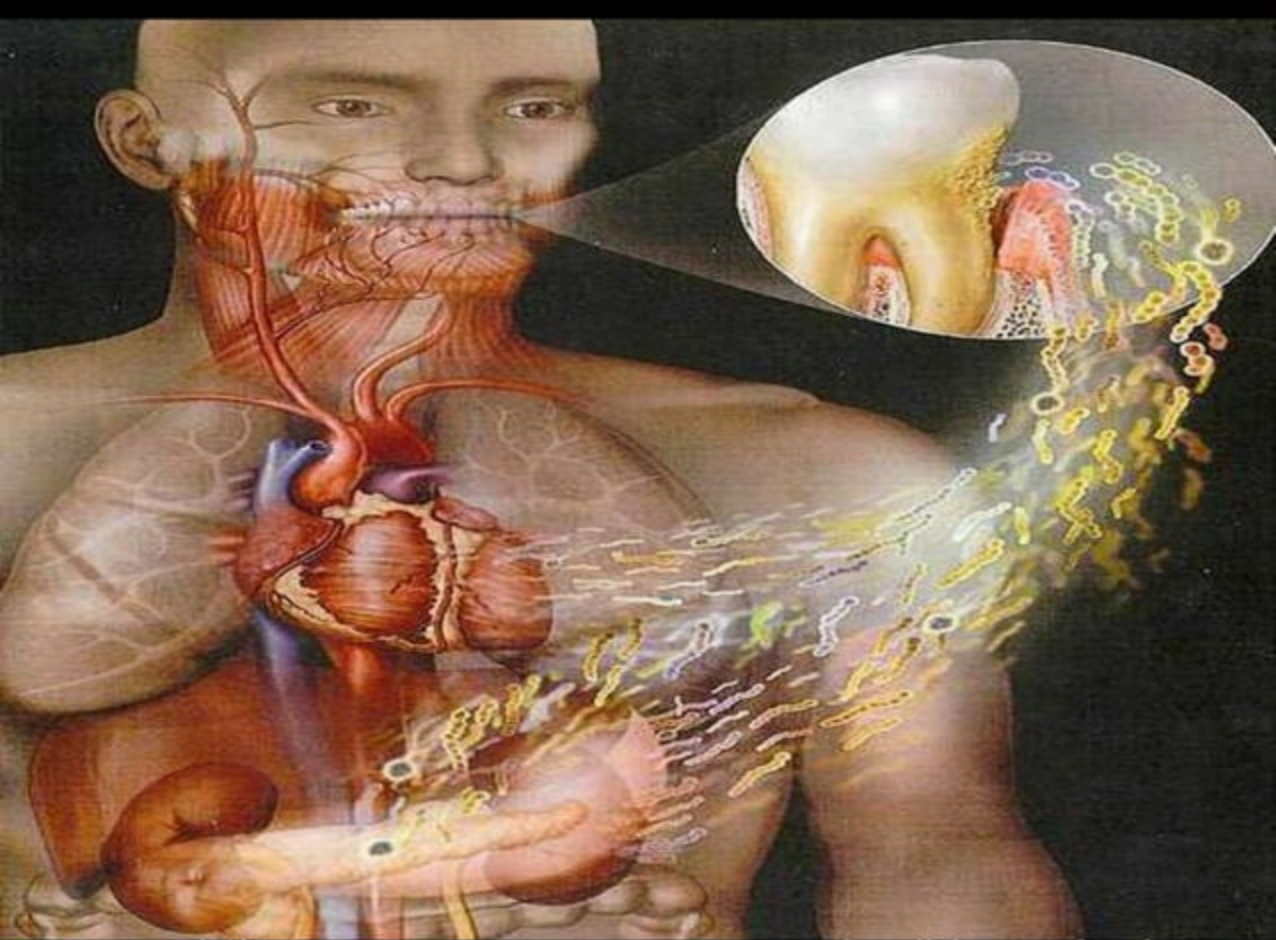




# **Periodontal Disease & Coronary Heart disease/Atherosclerosis**

- To further explore the periodontal disease & CHD/atherosclerosis association, investigators have studied specific disorders & medical outcomes to determine their relationship to periodontal status.
- In cross sectional studies of patients with acute myocardial infection compared with age & gender matched control patients, MI patients had significantly worse dental health than did controls.





- This association between poor dental health and myocardial infarction was independent of known risk factors for heart diseases such as age, cholesterol level, hypertension, diabetes & smoking.
- There may be a greater risk for CHD related events such as MI when periodontitis affects a greater number of teeth in mouth as compared to subjects having periodontitis of fewer



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## Dental Exposures

Poor Oral Hygiene → Periodontal/Caries → Dental Procedures → Tooth Loss  
Other Oral Infections

### *Noncausal Confounding Pathway*

#### Common Risk Factors:

Genetic Predisposition

Age

Smoking

Diabetes

Socioeconomic Status

Stress, Obesity, Diet

Physical Activity

Access/Use of Dental Care

Health Awareness/Behavior

Bacteremia

Psychosocial  
Factors

Diet Nutrients  
Weight Change

Inflammation/Vascular Injury

### *Potential CVD Outcomes*

#### Atherosclerotic

Coronary Heart Disease

Ischemic Stroke

Peripheral Vascular Disease

#### Other

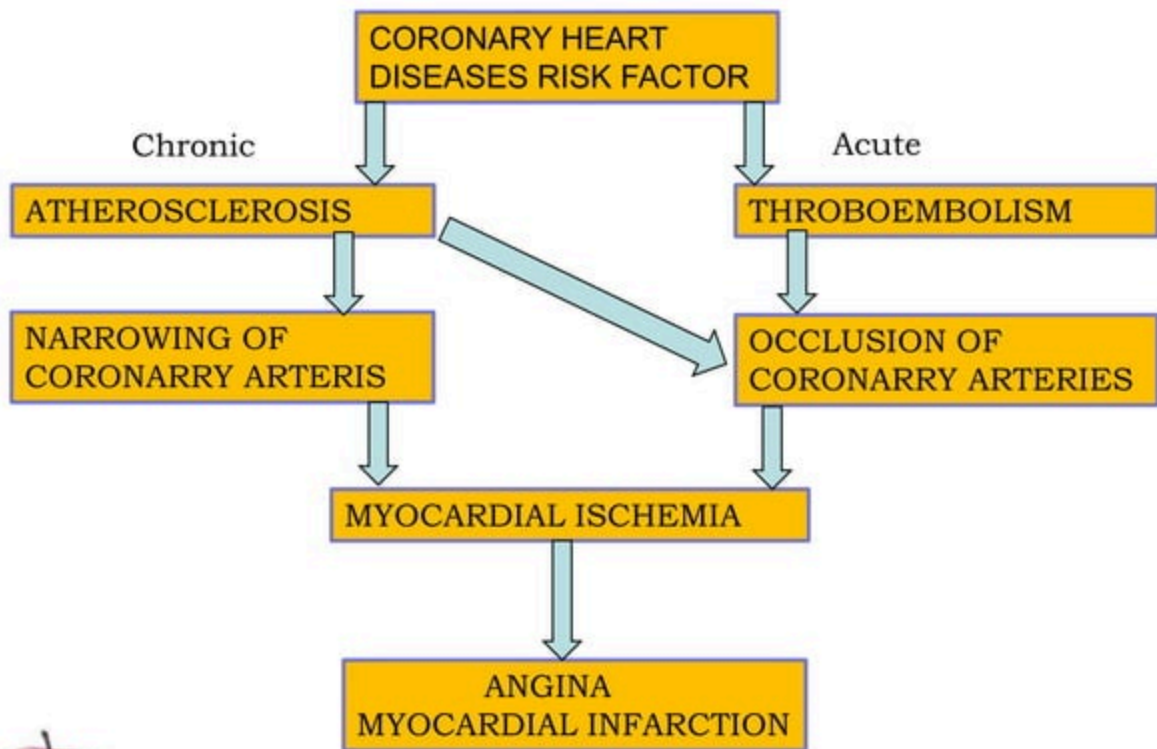
Hypertension

Hemorrhagic Stroke

Congestive Heart Failure

Rheumatic Heart Disease

CVD Mortality



# ISCHEMIC HEART DISEASE

- IHD is associated with the process of atherogenesis and thrombogenesis.
- Increased viscosity of blood may promote major ischemic heart disease and cerebrovascular accident by increasing risk of thrombus formation.



Systemic/ periodontal infection

↑  
Plasma fibrinogen  
Plasma lipoproteins  
White blood cell count

↑  
Fibrinogen  
Von willebrand factor



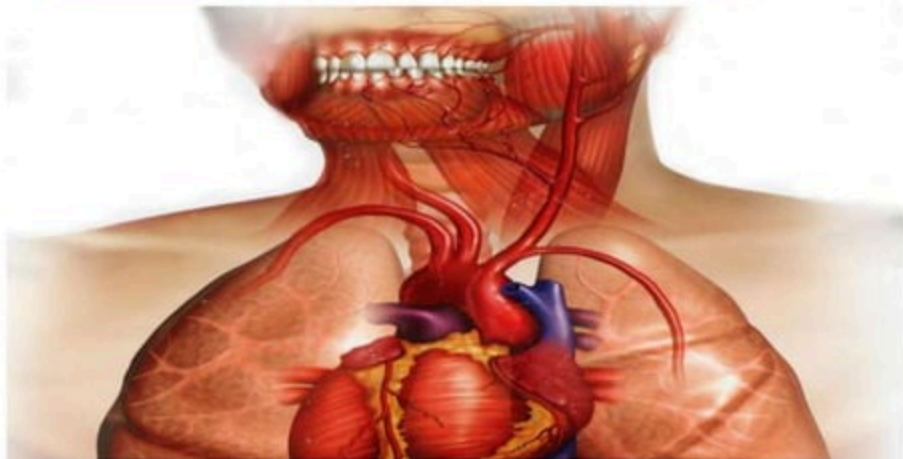




Blood viscosity



Ischemic heart disease



# THROMBOGENESIS

- Platelet aggregation plays a major role in thrombogenesis, & most cases of acute MI are precipitated by thromboembolism.
- Oral organisms may be involved in coronary thrombogenesis.
- Platelet selectively bind some strains of *Streptococci sanguis*, a common component of supragingival plaque & *Porphyromonas gingivalis*, a pathogen closely related with periodontitis.



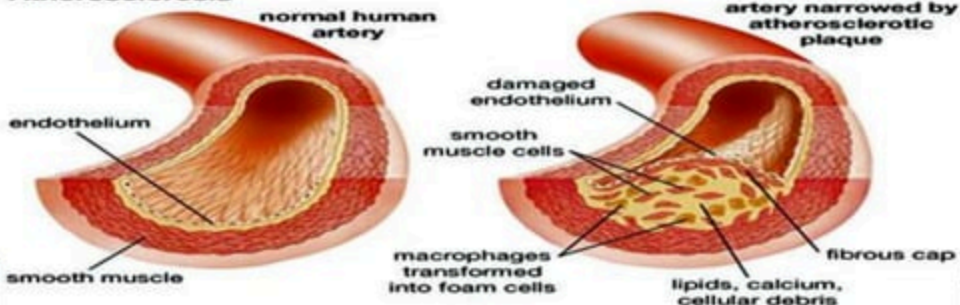
- Aggregation of platelets is induced by the platelet aggregation associated protein (PAAP) expressed on some strains of these bacteria.
- It results in formation of thromboemboli and resultant cardiac and pulmonary changes.



# ATHEROSCLEROSIS

- Atherosclerosis is a local thickening of arterial intima, the innermost layer lining the vessel lumen, and the media, the thick layer under the intima consisting of smooth muscle, collagen & elastic fibres.

**Atherosclerosis**

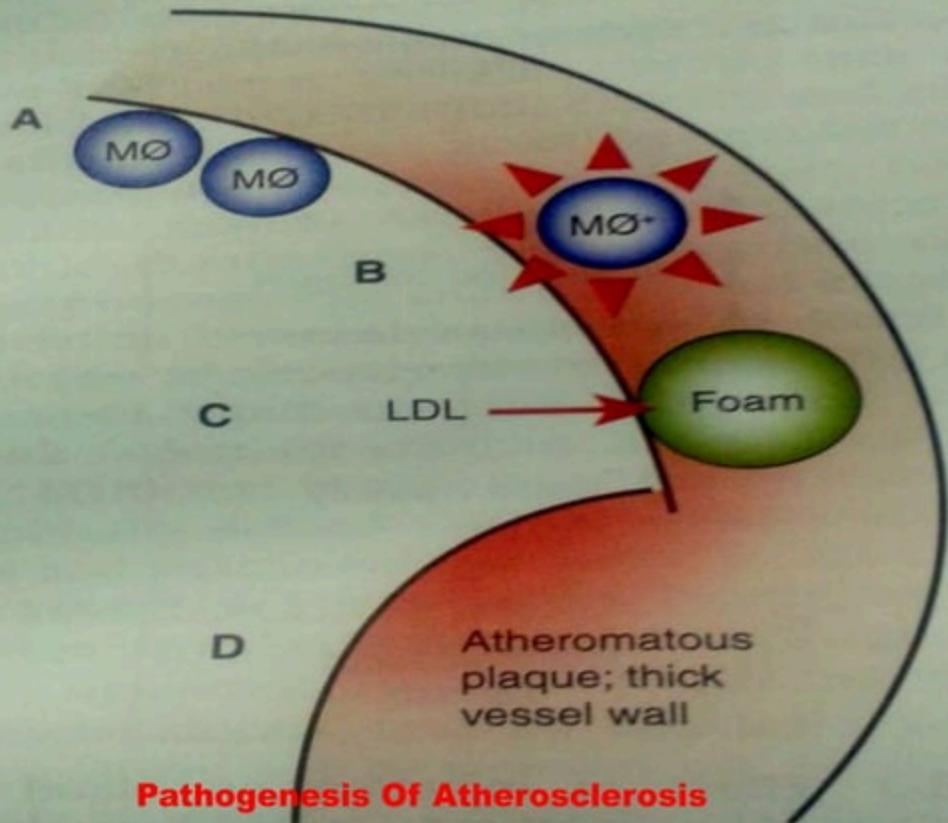




# Pathogenesis

- First, circulating monocytes adhere to the vascular endothelium, mediated by various adhesion molecules including intercellular adhesion molecule-1 (ICAM-1), endothelial leukocyte adhesion molecule-1 (ELAM-1) & vascular adhesion molecule-1 (VCAM-1).
- After binding of the monocytes, they penetrate the endothelium & migrate under the arterial intima.





- Monocytes now ingest the circulating low density lipoprotein (LDL), & form foam cells which are characteristics of atheromatous plaques.
- Proinflammatory cytokines, such as interleukin-1, tumor necrosis factor & prostaglandin E-2 are produced, which propagate the atheromatous lesion.



- Further there is smooth muscle and collagen proliferation within the media, thickening the arterial wall. Atheromatous plaque formation & thickening of vessel wall narrow the lumen & decrease the blood flow.
- Arterial thrombosis occurs after an atheromatous plaque ruptures.





## **Role of periodontal infection in myocardial infarction**

- In several studies of atheromas obtained during endarterectomy, more than half of the lesions contained periodontal pathogens.
- Periodontal diseases result in chronic system exposure to products of these bacteria.
- Low level bacterimia may initiate host responses that alter the coagulability, endothelial & vessel wall integrity, platelet function, resultin in thromboembolic events.



- There is wide variation in host response to bacterial challenge.
- Patients with exuberant inflammatory response have a hyper inflammatory monocyte/ macrophage phenotype.
- Here monocytes secrete increased levels of proinflammatory mediators in response to bacterial LPS compared to normal monocyte phenotype.



- The monocyte cell line is intimately involved in pathogenesis of both periodontal disease and atherosclerosis.
- Diet induced increased LDL level may increase secretion of destructive & inflammatory cytokines by monocytes.
- The presence of an M $\phi$  phenotype may place patient at risk of both CHD & periodontitis.



- Also, detection of systemic inflammatory markers play an important role in risk assesment for vascular events such as MI & cerebral infarction.
- Acute phase protein such as C- reactive protein (CRP) & fibrinogen are produced in liver in response to inflammatory or infectious stimuli & act as inflammatory markers.
- CRP induces monocytes to stimulate coagulation pathway & increases blood coagulability.





- Recent efforts have focused on periodontitis as a potential trigger for systemic inflammation.
- Serum CRP & fibrinogen levels are often elevated in subjects with periodontitis compared with non periodontitis subjects & may act as intermediary steps in the pathway from periodontal infection to cardiovascular disease.



**THANK YOU**

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