

Typical pathological processes in oral cavity

Disorders of odontogenesis

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Typical pathological process: definition, signs, examples

 The stereotype reactions of the body to various external and internal stimuli. Their appearance causes a cascade of reactions, which are aimed at maintaining the constancy of the internal environment of the body.

The main characteristics of the typical pathological process

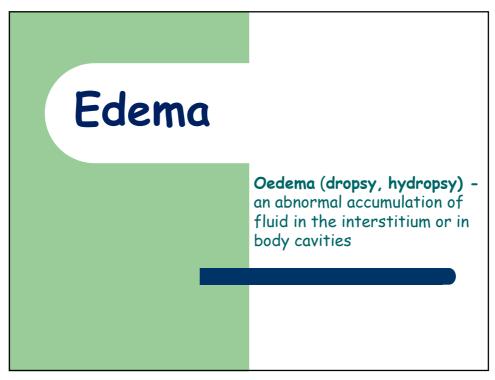
- Stereotype. The presence of the features of a typical process, regardless of the cause of its appearance and localization.
- Versatility. Typical pathological process can be in the composition of different nosological units.
- Polyetiologic. The etiologic factor of the disease performs only a starting role and is not permanent.
- Autochthonism. The process's ability to develop independently, even when the etiologic factor ceases to function.
- Equifinality. Different ways to implement the pathological process, which lead to the same development and resolution.

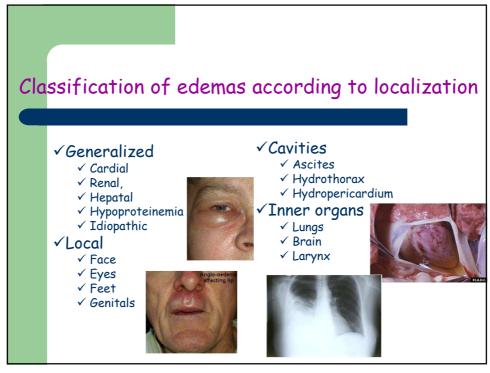
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- Hypoxia
- Fever
- Pain
- Disorders of consciousness
- Edema
- Thrombosis
- Tumors
-



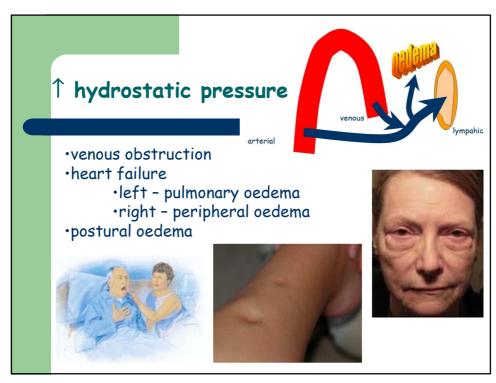


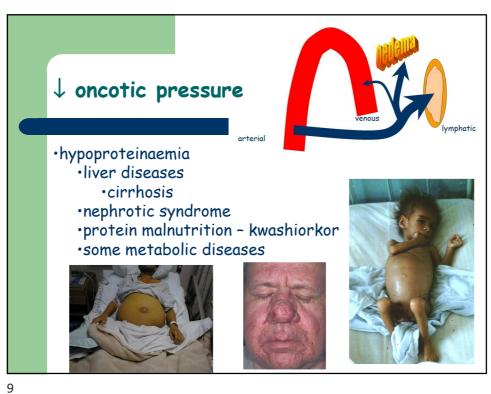


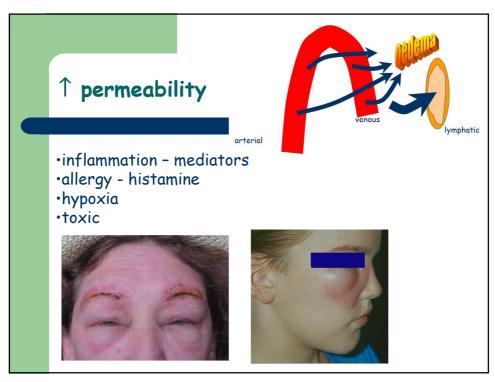
Classification of edemas according to etiology

- √Edemas caused by increased hydrostatic pressure
- √Edemas caused by decreased oncotic pressure
- ✓ Edemas caused by increased permeability
- ✓ Lymphedemas

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Angioedema

Quincke's edema Angioneurotic edema



- repetitive episodes of swelling face, lips, tongue, limbs, and genitals
- edema of the gastrointestinal mucosa severe abdominal pain; in the upper respiratory tract
- edema of larynx life-threatening

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Classification

Acquired angioedema

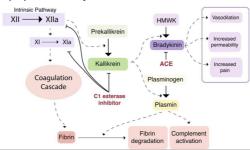
- Immunologic allergy
- Nonimmunologic medication (ACE inhibitors)
- Idiopathic

Hereditary angioedema - AD

- Types I mutation in the SERPING1 gene diminished levels of the C1-esterase inhibitor protein
- Types II mutation in the SERPING1 gene dysfunctional forms of the same protein
- Type III mutation in the F12 gene coagulation protein factor XII
- Abnormal activation of the complement system, activation of the contact pathway by the initial generation of kallikrein and/or clotting factor XII by damaged endothelial cells - production of bradykinin increased vascular permeability and vasodilation - swelling.

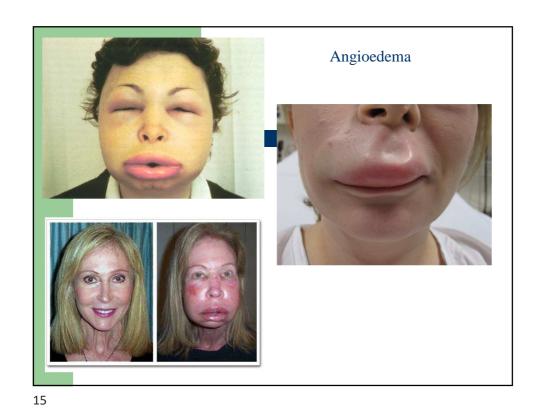
C1 esterase inhibitor

- A protease inhibitor inhibition of the complement system, also inhibits proteases of the fibrinolytic, clotting, and kinin pathways.
- In hereditary angioedema deficiency of C1-inhibitor permits plasma kallikrein activation, which leads to the production of the vasoactive peptide bradykinin → edema.



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Lymphatic obstruction

lymphoedema (woody oedema)

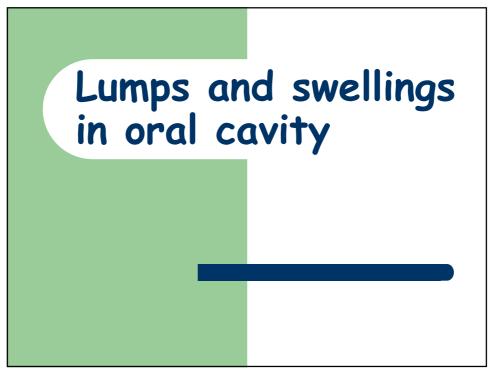
·lymfatic obstruction

·parasites (filariasis)

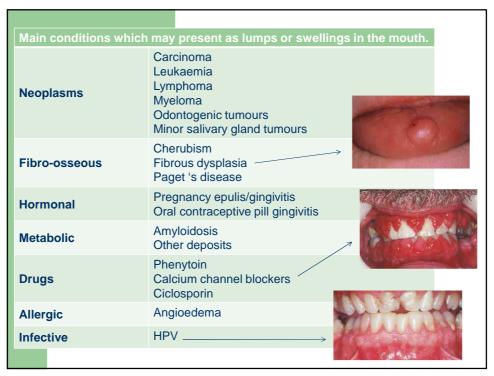
·cancer of lymph nodes

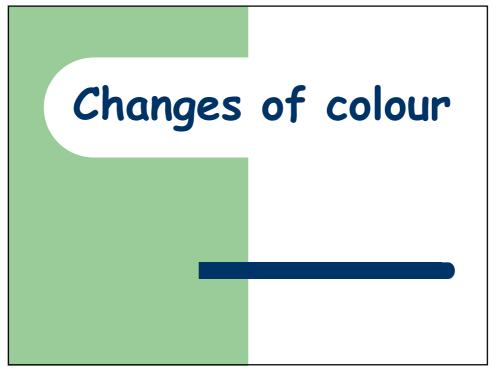
·surgery or radiation therapy (breast cancer)

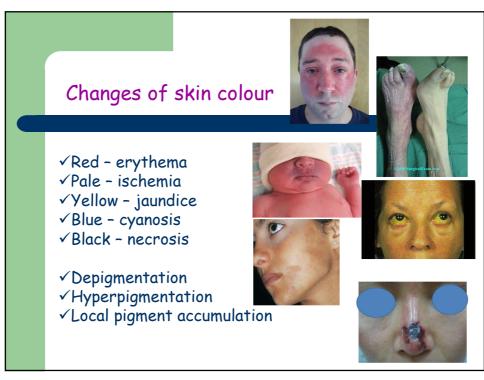
·inflammatory changes - lymphangitis



Main conditions which	may present as lumps or swelling	s in the mouth.	
Normal Anatomy	Parotid papillae Lingual papillae		
Developmental	Unerupted teeth Developmental cysts Haemangioma Lymphangioma Maxillary and mandibular tori		
Inflammatory	Cellulitis Sialadenitis Pyogenic granuloma Chronic granulomatous disorders - Orofacial granulomatosis - Crohn's disease - Sarcoidosis Insect bites		
Traumatic	Denture-induced hyperplasia Epulis Fibro-epithelial polyp Haematoma Mucocele Surgical emphysema		







Erythema

- > Hyperemia UV radiation, ↑ temperature, inflammation
- > Flushing fever, anger, alcohol...





Alcohol intolerance

• East Asia - China, Taiwan, Japan, Korea

Cause

- Alcohol dehydrogenase variant 1C ADH1C
 Higher production of acetaldehyde
- Deficiency of aldehyde dehydrogenase 2

Signs:

- Anafylaktická reakcia
- Flushing of the face
 - It starts a few minutes after drinking alcohol, peaks in 30-40 minutes, disappears after 1-2 hours.
 - A little alcohol is enough, but the intensity also depends on the dose
- Hives, red itchy rashes
- Stuffy nose, runny nose as in rhinitis
- Hypotension
- Nausea, vomiting, diarrhea
- Headaches, anxiety, drowsiness
- Anaphylactic reaction





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Pallor

- > Anemia
- > Emotional shock, panic attack, stress
- > Cold environement, frostbite
- > Fever
- > Leukemia
- > Medications (amphetamines, ethanol, cannabis)
- > Heart disease
- > Hypotension
- > Peripheral vascular disease
- > Sleep deprivation





Jaundice



- Hyperbilirubinemia
 - Prehepatic hemolytic anemia
 - Hepatic liver diseases, virus infection, alcohol, drugs...
 - Posthepatic posthepatic obstruction









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Cyanosis

- Blue colour of the skin and musoca
- Cyanosis = reduced hemoglobin > 50 g/l
- The skin or musoca cyanosis (blue colour) depends on amount of reduced hemoglobin in capillaries - not in arteries or in veins
- In anemia cyanosis is not present
- In polyglobulia can be



Classification of cyanosis

Hemoglobin cyanosis

- reduced hemoglobin > 50 g/l
- Central and periferal cyanosis

Hemiglobin cyanosis

-abnormal hemoglobins (methemoglobin, sulfhemoglobin)

Methemoglobin cyanosis

- -Fe3+ (hemiglobin)
- -metHb 1,5% of total Hb
- clinical signs > 35% of total hemoglobin
- -inborn (defects of methemoglobin reductese, G-6-PD)
- -acquired (drugs-antipyretics, lidokain, nitrites)

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Central cyanosis

Hemoglobin cyanosis

- > total, arterial, anoxic
- > visible: mucosa, lips, tongue, nail beds, ears
- > arterial blood is not enough saturated bu oxygen
- > ↑ reduced Hb

Inborn errors with right-left shunt

-Fallot tetralogy, Eisenmenger sy., Ebstein sy.

Lung diseases

- -obstructive lung diseases
- -pulmonary edema, pulomonary embolia

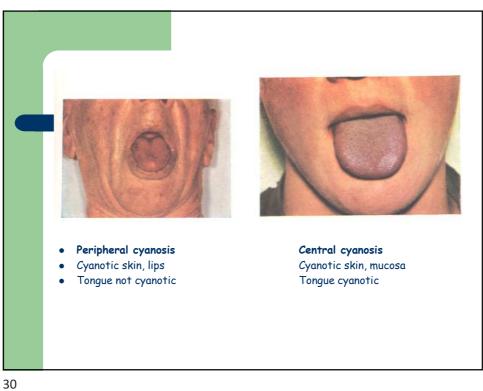




Peripheral cyanosis

- > acral, venous, stagnation
- > normal saturation of arterial lood
- \succ stagnation \uparrow oxygen extraction
- ↑ reduced Hb
- > tongue pink





Pseudocyanosis

Chemical compounds accumulated in the skin

Argyria (argyrosis)

- Gray-violet colour of skin
- Silver intoxication (antiobacterial creams)



- Arsenic melanosis





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Local pigment accumulation

Alkaptonuria

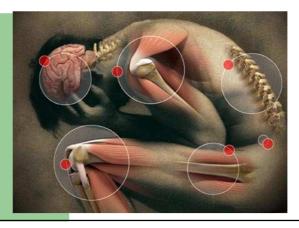
ochronosis – accumulation of the ochronotic pigment in:

- joints ochronotic artropathy
- endocardium
- renal and prostatic stones
- other (eyes, teeth, CNS)





Pain



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Definition

Pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage

(IASP – The International Association for the Study of Pain, 1979)

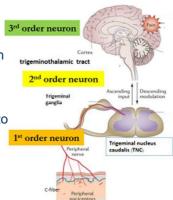
- damage (danger) signal
- indication and localization of damage
- prevention of more serious damage

Pain in orofacial region - oral cavity (teeth, gingiva, oral mucosa), face, jaw bone, temporomandibular joint

Orofacial Pain Pathway

Orofacial pain pathways includes primary afferent neurons, trigeminal ganglion, brainstem nociceptive neurons, and higher brain function regulating orofacial nociception.

- Nociceptors in the orofacial region
- 1st order neurons in the trigeminal nerve
- 2nd order neurons in the trigeminal nucleus caudalis located in the brainstem
- 3rd order neurons in the thalamus via the ventral trigeminothalamic tract
- The descending pathway sends signals to the trigeminal nucleus caudalis – serotonin, norepinephrine and opioid peptides are produced - this process leads to pain reduction



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Classification of orofacial pain

		Acute orofacial pain	Chronic orofacial pain
D	Ouration	Onset	Sustained, persistent >3 months in humans
C	Cause	Inflammation or injury of tissue	Inflammation, nerve damage
	Physiologic esponse	Increased blood pressure, tachycardia via sympathetic response	Adaptation behaviors or psychological responses such as depression and anxiety
tł	examples in the orofacial trea	Dental pain: pulpitis Mucogingival pain	Neuropathic pain: trigeminal neuralgia, peripheral trigeminal nerve injury, postherpetic neuralgia Chronic inflammatory pain: chronic pulpitis and apical lesions, temporomandibular disorder pain Neurovascular pain: migraines, tension-type headaches

Classification of orofacial pain

	Nociceptive orofacial pain	Inflammatory orofacial pain	Neuropathic pain		
Causes and mechanism of pain pathway	Noxious stimulation at the peripheral nerve and transmitted by normal components of the sensory trigeminal nerve	Strong noxious stimulus causes lesions in the tissue leading to local inflammation responses and increased inflammatory mediators	Caused by nerve damage or injury and increased peripheral sensitization, structure change by increased sodium activation, calcium activity of nerves leading to ectopic discharges, and glia cell activation		
Stimulation	Response to noxious stimulus	Response to noxious stimulus	Response to non-noxious and noxious stimulation Spontaneous pain without stimulation occurred in damaged nerves		
Example	E.g. response to hot drink	Pulp necrosis Temporomandibular joint	Peripheral trigeminal nerve injury - facial trauma accident		

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Pain in oral cavity



"Toothache" is pain typically around a tooth, teeth or jaws.

Causes

- dental problems dental cavity, a cracked or fractured tooth, an exposed tooth root, or gum disease.
- diseases of the jaw joint (temporomandibular joint)
- spasms of the muscles
- cancers
- teeth implants

The severity of a toothache can range from chronic and mild to sharp and excruciating. It can be a dull ache or intense.

The pain may be aggravated by chewing or by foods and liquids which are cold or hot, sweet.



Heart pain can radiate to the jaw and teeth

Sometimes, pain in oral cavity may be caused by a problem not originating from a tooth or the jaw at all.

Pain around the teeth and the jaws can be symptoms

- of diseases of the heart (angina pectoris, myocardal infarction)
- ears (inner or external ear infections)
- sinuses (sinusitis)
- neuralgias and other nerve ailments

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Some problems that cause oral pain

Sensitivity to hot or cold foods without recent dental work.

- loose filling,
- decay,
- fracture in the tooth,
- minimal gum recession which exposes small areas of the root surface.

Prolonged sensitivity to hot or cold foods without recent dental work.

- pulp irreversibly damaged by deep decay,
- crack/fracture,
- periodontal disease or trauma.

Sensitivity to hot or cold foods after recent dental treatment.

• inflammation of the pulp, inside the tooth, causing temporary sensitivity.

Some problems that cause oral pain

Dull ache near a tooth and/or biting sensitivity <u>after recent dental</u> treatment

inflammation

Sharp pain when biting down on food

- loose filling
- decay
- cracked or split tooth
- cuspal fracture
- · vertical root fracture

Constant and severe pain with pressure, swelling of the gum, and sensitivity to touch

absces

A tooth hurts after taping on it with finger from the side.

• inflammation of periodontal ligament

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Some problems that cause oral pain

Dull ache and pressure in upper teeth and jaw

- sinus problems (sinusitis)
- grinding of teeth (bruxism)

Chronic pain in head, neck, or ear

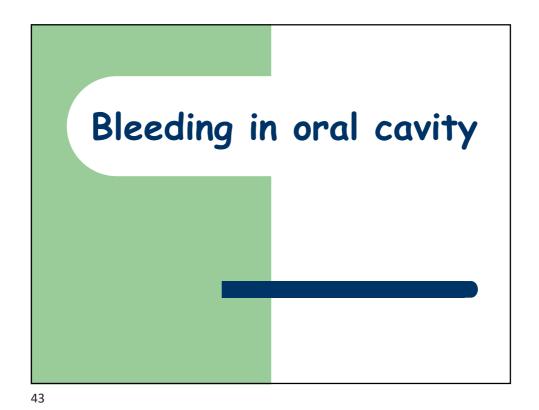
• sometimes pulp-damaged teeth cause pain in other parts of the head and neck, but other dental or medical problems may be responsible.

Touching a specific spot in or near mouth triggers a sharp, jabbing pain lasting a few seconds. Sometimes talking may also cause this to occur.

trigeminal neuralgia

Clicking or pop is heard when opening mouth. Opening/closing of mouth may be painful

• temporomandibular dysfunction



Causes of bleeding in oral cavity

Gingivitis

• Edema, pain, bleeding

Periodontitis

- Inflammation of gums, jawbone and supportive tissues
- Bleeding, inflammation, pain, loss of teeth.

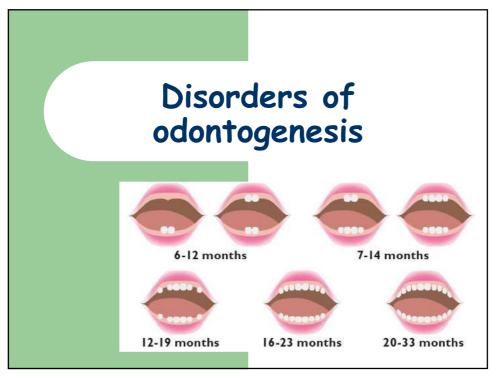
Vitamin deficiencies

- Vitamin C
- Vitamin E

Other

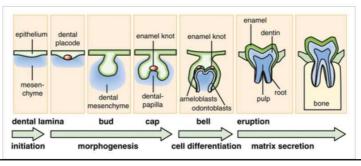
- Leukemia
- Hemophilia
- Chronic irritation, injury
- Surgery
- Pregnancy hormonal changes

S Of Teeth.



Teeth development

 The process of development of teeth is a very complex process resulting from interactions between the ectoderm of the oral cavity, which gives rise to cells that produce enamel, and the neural crest ectomesenchyme which gives rise to the tooth structures other than enamel.



There are four stages in the development of the tooth germ: 1. Bud stage 2. Cap stage 3. Early bell stage 4. Late bell stage STAGE/TIME SPAN DESCRIPTION Initiation stage/ Ectoderm lining stomodeum gives rise to oral epithelium and sixth to seventh week then to dental lamina; adjacent to deeper ectomesenchyme, which is influenced by the neural crest cells. Both tissue types are separated by a basement membrane Bud stage/ Growth of dental lamina into bud shape that penetrates eighth week growing ectomesenchyme Cap stage/ Formation of tooth germ as enamel organ forms into cap ninth to tenth week shape that surrounds inside mass of dental papilla, with an outside mass of dental sac, both from the ectomesenchyme. Bell stage/ Differentiation of enamel organ into bell shape with four cell eleventh to twelfth week types and dental papilla into two cell types Apposition stage/ Dental tissue types secreted in successive layers as matrix varies per tooth Maturation stage/ Dental tissue types fully mineralize to mature form

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varies per tooth

Disorders of tooth development and eruption

- Anodontia
 - Absence of teeth Hypodontia
 - Oligodontia





• Hyperdontia - supernumerary teeth







Disorders of tooth development and eruption Abnormalities of size and form Fusion of teeth Dens evaginatus Dens in dente Enamel pearls Macrodontia Microdontia Peg-shaped (conical) teeth Supernumerary roots Taurodontism

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Disorders of tooth development and eruption Mottled teeth Dental fluorosis Disturbances of tooth formation Aplasia and hypoplasia of cementum Enamel hypoplasia Hypocalcification of teeth Hereditary disturbances in tooth structure Amelogenesis imperfecta Dentinogenesis imperfecta Odontogenesis imperfecta

Disorders of tooth development and eruption

- Disturbances in tooth eruption
 - late
 - obstructed
 - premature

