Esophagus

Contents:

Comments on embryology of esophagus

Esophagus develops form the part of the ventral gut caudally from the pharynx after the separation from the laryngotracheal tube. Embryonic esophagus is short at first and caudally continues as a base of the stomach. In the 2nd month it grows faster, lengthens and drags entoderm into its interior. Esophagus rotates because of stomach rotation (asymmetric course of n.X). Epithelium forms from entoderm, it proliferates and can fill all the interior of the esophagus. Later, a star-shaped lumen forms. Fibrous tissue and muscles form from mesenchyme. Striated muscles in upper third of esophagus form from mesenchyme of the branchial arches, smooth muscles in lower 2/3 of esophagus form from splanchnopleuric mesenchyme.

Clinical anatomy and physiology of the esophagus

Clinical anatomy of esophagus

The esophageal wall has 4 layers: mucosa, submucous tissue, muscles and adventitia

Mucosa forms the longitudinal plicae and is formed by spinocellular epithelium

Muscles have 2 layers: inner circular and external longitudinal. Above the upper sphincter (Kilian´s) forms m.constricotr pharyngis weak spot, where Zenker´s diverticulum can develop.

The esophageal wall is 2-5 mm thick. Esophagus is 25-30 cm long in adults. Its beginning is on the level of annular cartilage and C6. Esophagus runs in front of the spine. It enters the

stomach on the level of Th 11. Kilian´s sphincter and cardia have their own closing mechanisms with constant tonus, which prevents intrusion of the air into the stomach and reflux of gastric fluids.

Esophageal strictures:

- **upper** – pars fundiformis of the pharyngeal sphincter and Kilian´s sphincter. The distance from the lower incisive teeth is 7 cm in sucklings, 12 cm in 10-year old children and 16 cm in adults.

- **middle** – caused by pressure of the aortal arch and left main bronchus

- **lower –** at the end of the esophagus and it is caused by cardia and diaphragm crossing. The distance from the incisive teeth is 21 cm in sucklings, 27 cm in 10-year old children and 40 cm in adults.

Topography of esophagus

- **neck part:** C6 – Th1, ventrally connected with the larynx and the trachea, laterally there are lobes of the thyroid gland and the great neck vessels and nerves, dorsally there is the neck spine.

- **thoracic part:** it is the longest. It is in the mediastinum – Th1-Th7-8. In ventral mediastinum there is the trachea with bifurcation, heart and great vessels, in dorsal mediastinum there are nn.vagi, vv.azygos and hemiazygos, sympaticus and ductus thoracicus. The relation of the esophagus to the vessels, aorta, pericardium and pleura is different according to the part of the esophagus so in case of perforation the symptoms are very variable.

- **Abdominal part:** it is the shortest. Hiatus esophageus is round and is on the level of Th 9-11

Innervation of esophagus

- nn.recurrentis in the neck part

- branches of neck and thoracic sympaticus and nn.vagi in thoracic and abdominal part. N.X causes dilatation and sympaticus constriction of the esophageal openings. Caudal part of the esophagus and cardia have cholinergic and adrenergic receptors which control the opening and the closing of the cardia.

Clinical physiology of esophagus

- food intake – food is moved by the swallowing reflex into the esophagus. The esophageal wall is very elastic and food movement is maintained by active contractions of muscles.

Examination methods of esophagus

Imaging methods

X-ray:

- normal picture is used for diagnostics of foreign bodies
- contrast examination: swallowing act with a barium paste, iodine contrast fluids if we suspect a perforation
- CT and MRI in case of expansive lesions

Esophagoscopy: endoscopic examination with rigid or flexible optics

- rigid: especially for extraction of foreign bodies
- flexible especially for diagnostics

pH-metry:

- by sound for diagnostics of GERD

Congenital defects of esophagus

Aplasia, strictures

They are caused by esophageal recanalization failure during the development. In this case newborn cannot swallow neither saliva nor food. Vomiting is present immediately after the birth. Prognosis depends on other associated anomalies. Esophageal strictures cause dysphagia after the change to solid food. Dysphagia and regurgitation need careful examination – suspicion of combined anomalies.

Diagnostics: X-ray possibly with contrast medium (iodine), CT, MR, esophagoscopy, bronchoscopy.

Tracheoesophageal fistula (TEF)

Etiology: according to Denker it is caused by failure in the necking process and in forming the transesophageal septum.

Classification according to Vogt and Haight:

- Atresia without TEF
- Atresia with TEF in proximal segment
- Atresia with TEF in distal segment
- Atresia with TEF in both segments

- TEF without atresia

Symptoms: The most frequent type (88%) is esophageal atresia with fistula to trachea in the distal segment. For atresia with fistula, typical are: the presence of spumous phlegm in the mouth and the nose which persist even after sucking off, vomit is without hydrochloric acid, seizures of dyspnea and cyanosis, asphyxia during feeding. Sometimes notable meteorism is present. In obstetric history, there can be hydramnion. In the most frequent type, aeriform filling of the stomach and the guts is present.

Diagnostics: X-ray possibly with a contrast medium (iodine), CT, MR, esophagoscopy, bronchoscopy.

Therapy: surgery

Congenital anomalies of great vessels

They can cause esophageal patency defects as well. Most frequent are:

- Dysphagia lusoria – caused by abnormally situated a.sublclavia dextra
- Double aortal arch
- Right sided aortal arch

Diagnostics: cardiological examination

Hiatal hernia

Probably, it is esophageal development defect, developed around the 4^{th} embryonic week. Esophagus has an insufficient length and fixation on the hiatus oesophagicus, which leads to the hiatal hernia.

Achalasia

Definition: it is a syndrome of non-organic cardia obstruction combined with great esophageal hypertrophy and dilatation.

Etiology: it is either improper development or atrophy of plexus Auerbachi in muscles.

Symptoms: Parasympathic filament dysfunction leads to the prevalence of sympathicus and sphincter contracture of cardia follows. It is presented either from the birth or later, after the change to solid food – vomiting after several bites, or later vomiting even predigested food. Stridor is caused by pressure on bronchus.

Diagnostics: X-ray examination of esophageal passage (saccifrom esofageal dilatation), esophagoscopy – if organic changes are not found, neurogenic cause of dysphagia is suspected.

Therapy: dilatation, cardiomyotomy according to Heller

Double esophagus

It is a rare congenital defect, when esophagus is divided by a longitudinal septum. The doubling does not have to go all the way through the esophagus, causing troubles and generally it is found by chance.

Therapy: surgery

Brachyesophagus

Short swallow. A part of the stomach lies above the diaphragm so the stomach is held in the diaphragm. Sometimes it does not have to cause troubles, but usually it causes vomiting after every meal. Vomiting stops in the upright body position.

Diagnostics: X-ray examination with a contrast medium

Therapy: surgery

Inflammations of esophagus

Acute non-specific esophagitis

Pathogenesis:

- Catarrhal inflammation of esophagus can develop during quinsy, esophageal, stomach or lung inflammation. The inflammation can spread to the esophagus per continuitatem or via blood. Scarlet fever can be accompanied with an ulcerous esophageal inflammation. The upper third of the esophagus can be afflicted by pseudomembranes during diphtheria. Acute esophagitis can be caused by GERD, irritating food or after surgery in anesthesia as a result of postoperative vomiting.

- Acute suppurating inflammation is localized around decayed tumors and specific inflammations. Injury with foreign body or esophagoscopic tube can cause this inflammation as well. It can be unconfined (flegmona), or limited (abscess of esophageal wall or periesophageal space)

Symptoms: dysphagia, odynophagia, sense of burning and chest pain

Complications: mediastinitis

Mycotic esophagitis

Pathogenesis: primary mycotic esophagitis is very rare. Yeast growth is usually caused by another condition: malignant tumor, food stagnation in esophagus, ATB or corticosteroids administration.

Etiology: the most frequent is Candida albicans, less frequent Candida tropicalis, pseudotropicalis, Krusei.

Symptoms: chest pain and burning, worsening during swallowing, dryness in the neck and dysphagia. Suddenly arisen problems are typical in patients with grave disease.

Esophagoscopy:

- catarrhal form: mucosa is red, dry and fragile
- coating form – mucosa is red, dry, hypertrophic, here and there with white coats
- pseudotumorous form
- „cotton-wool" esophagus – interior is stuffed with white mycotic bunches

Therapy: 5-10% kalium iodatum, gentian violet, antimycotics. We must be in search of basic disease, correct esophageal passage, if possible discontinue ATB and corticosteroids.

Specific esophagitis

They are very rare.

- Tuberculosis spreads in esophagus per continuitatem from mediastinal lymphatic nodes. Possible is an infection via swallowed sputum or via blood.

Symptoms: dysphagia, odynophagia, fever, malnutrition. Patients have spontaneous pain worsening during idly swallowing.

Diagnostics: esophagoscopy – ulcer, granulations, infiltration, fistula or tuberculoma, after heal are present scars.

Therapy: antituberculotic drugs

- Syphilis of esophagus – during the 3rd stage we can see gumma. Dysphagia is present, but not very serious

Stagnation esophagitis

Pathogenesis: food stagnate above cicatricial and functional strictures, interior of esophagus gets wider. Sometimes cystic esophagitis develops which is caused by an inflammatory infiltration of mucosal and submucosal lymphatic nodes and by the retention in slime glands with cyst forming.

Symptoms: chest pain and burning, dysphagia related to primary disease

Esophagoscopy: mucosal hypertrophy, bleeding, ulcers or grey-whitish leucoplakias, irregularly granular surface of red hypertrophic mucosa

Therapy: it should be focused on the primary disease, a temporary relief can be achieved by sucking off the stagnating food, in acute exacerbation local antibiotics is needed.

Gastroesophageal reflux disease - GERD

Pathogenesis: If the closure mechanism coordination of the lower third of the esophagus is damaged (after surgery, by hiatal hernia, etc.), gastric juices reflux to the esophagus is present.

Classification (according to Škeřík):

- catarrhal esophagitis – mucosa in the lower third of the esophagus is congested and swollen
- hypertrophic esophagitis – changes are primary in the lower third of the esophagus, but they can afflict the whole esophagus
- ulcerous esophagitis – in the lower third of the esophagus are areal ulcers with low granulations and fibrin coating
- fibrostenosing esophagitis – concentric strictures develop because of scars formation as a result of inflammation

Symptoms: burning, eructation, sometimes even gastric juices regurgitation, worsening in a forward bend or in a lying position. During swallowing there can be present a painful chest oppression, sometimes even spontaneous chest and epigastric pain.

Diagnostics: esophagoscopy, pH-metry

Therapy: causal, diet, antacids. In fibrostenosing form probe dilatation is indicated.

Peptic esophageal ulcer

Some consider it a single nosologic unit, others consider it a quantitative change of ulcerous reflux esophagitis. It is located in the lower third of the esophagus, more often in men. Symptoms are more expressive than in reflux esophagitis, there can be serious hemorrhages and even a perforation of the ulcer.

Therapy: the same as in case of the gastric ulcer

Sideropenic dysphagia

This disease is in literature known under the following names: syndrome Gougerot-Sjógren, sy Plummer-Vinson, sy Kelly-Patterson, glossopharyngopathia sideropenica, epitheloxerosis. Women are almost exclusively afflicted.

Symptoms: dysphagia, glosodynia, mucosal atrophy of the tongue and the pharynx, stomatitis angularis, small mouth, dry skin, hair loss, defect nails, teeth loss, fluor albus, achylia resistant on histamine, hypochromic anemia.

Esophagoscopy: atrophic, smoothed, glossy mucosa, white leucoplakias, semilunar folds usually from ventral side of esophagus (on X-ray looks like plain notches in a contrast filling).

Therapy: vitamins B, iron by anemia, hydrochloric acid and pepsin by achlorhydria. In case of semilunar folds use spasmolytics and dilatation

Tumors of esophagus

From Children ORL

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Benign tumors of esophagus

Benign tumors of the esophagus grow from mucosa or the esophageal wall, they can be located even outside the wall and cause oppression from the outside.

Polyps: they are located most frequently in the upper part of the esophagus or in the hypopharynx, usually in men. They have a gray or red color, consistency is semi-solid, the shape is long and thin.

Cysts: they are multiple, of inflammatory origin or congenital cyst localized usually in the lower part of the esophagus between mucosa and muscles.

Lipoma, fibrolipoma, fibroma: they are usually pedunculated so that during vomiting, the tumor can be vomited out of the esophagus to the mouth.

Angioma: it is often a source of bleeding.

Papilloma: it is usually around the esophageal sphincters, it has a typical look – a pale surface, a typical shape.

Adenoma: it is very rare, usually in the distal part of the esophagus and around the cardia

Myoma: rhabdomyomas are very rare, but leiomyoma are very frequent, they are found in more than half the cases of benign tumors in the esophagus. They bulge to the interior of the esophagus and are covered with normal mucosa. However, circular strictures of esophagus are also known, as well as multiple myomas and diffuse myomatosis of esophagus.

Symptoms: they depend on the size, shape and localization of tumor

- small tumors do not have to cause any problems, they can be the cause of spasms, transient dysphagia, a feeling of oppression or the presence of a foreign body
- larger tumors are accompanied with bigger problems with swallowing, regurgitation or bleeding if a surface necrosis is present.
- Pedunculated tumors and polyps cause swallowing problems, and in case of displacement to the larynx they cause dyspnea and even suffocation.

Therapy: smaller and pedunculated tumors can be removed with endoscopic nippers, loop, cauterization, laser or cryosurgically. Larger tumors must be removed from external access.

Malignant tumors of esophagus

Carcinoma is the most frequent malignant tumor in the esophagus. Sarcoma and other malignant tumors are rare. Esophageal cancer is relatively common, in statistics of malignant tumor it is on 4th or 5th place. The incidence vary depending on the area. More frequently they are present in men (4-11:1). Carcinoma is more frequent in the upper two thirds of the esophagus, adenocarcinoma in the lower third. Carcinoma is often the result of burning of the esophagus.

Classification (according to esophagoscopic findings):

- exophytic form (nodulated shape, here and there with necrosis)
- infiltrative form (the wall of esophagus is infiltrated, has a limited movement, mucosa is usually not afflicted)
- ulcerous form

Symptoms: they depend on the localization, size and growth of tumor. Tumors with exophytic growth cause symptoms earlier. Problems in esophageal tumors are worsened by transitory spasms of the esophagus around the tumor, but even by reflex spasms on further

places. Paralysis of n. laryngeus reccurens occur sometimes, which results in hoarseness. In later stages esophagotracheal or esophagobronchial fistula can develop.

- Tumors in the area of the upper esophageal sphincter cause at first paresthesia in the neck, later cause dysphagia, pain, weight loss, foetor ex ore, increased salivation. In later stages aspiration of food and fluids can be present.

- Tumors in the pectoral part of the esophagus cause at first painless dysphagia which is getting worse, patients have to drink during eating and finally regurgitation of food during eating occurs.

- Tumors in cardia cause dyspeptic problems, vomiting, weight loss, epigastric pain, dysphagia.

Therapy: surgery, actinotherapy. Palliative procedures are used for maintaining passage thorough esophagus – artificial endoprosthesis implantation, argon or neodym laser.

Injuries of esophagus

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Burning of esophagus

Etiology:

- Alkali: receives free proton (H+) – causes majority of esophageal injuries. It causes colliquative necrosis. Typical are lyes (NaOH, KOH, Ca(OH)2), ammoniac, silicates and carbonates – contained in cleaning agents

- Acids: release proton, cause about 15% of burning injuries, especially in suicide. They are contained especially in WC and pool cleaning agents (HCl, H2SO4, H3PO4, HNO3). They cause coagulation necrosis, which limits farther penetration and damage of muscular layer. Esophagus has slightly alcalic pH and its epithelium is partially protected against acids.

- Disc batteries: usually contain concentrated solution of NaOH or KOH and cause mucosal damage in 1 hour and penetration in 4 hours (damage by alkali, pressure and electricity)

- Thermal damage: hot fluids (warmed up in microwave oven), tomatoes, pizza, can cause swelling of larynx and dyspnea, esophageal injury is rare

- Drug burning: tetracycline or NSAIDs can cause hemorrhages and strictures

Pathogenesis: critical pH causing esophageal ulcers is 12.5 in alkali and 2.0 in acids. Tissue damage depends on concentration and amount of the given substance. Solid substances are

commonly spit up so that damage to the esophagus is rare. Besides local changes, the organism can be influenced generally – intoxication, shock (there is no publication about death caused by systemic toxicity of alkali). Esophagus is damaged most frequently in physiological strictures, stomach is damaged in about 80% of cases.

Classification:

- 1. degree – erythema and mucosal swelling
- 2. degree – submucosal damage, ulcer
- 3. degree – muscle layer damage – perforation

Symptoms: red mucosa of mouth and pharynx, odynophagia, dysphagia, increased salivation, nausea, vomiting. Chest and abdomen pain can signal a possible perforation. Larynx swelling is uncommon (dyspnea can develop in case of larynx entrance burning). Absence of damage in oral cavity does not exclude heavy distal damage.

Course of disease:

- Acute phase: damage of superficial epithelium with possible deeper extension and venous thrombosis. Polynuclears and bacteria infiltrate mucosa in 48 hours. Mucosa is red or cyanotic.
- Reparative phase: after 5 days in average – granulations are forming at the edge of the ulcer, deposits of collagen and fibroblasts are present
- Cicatrical phase: after 2-3 weeks. If circular damage is present, patient is threatened by strictures

Diagnostics: flexible nasopharyngolaryngoscopy, blood examination, chest X-ray . Esophagosopy after 12-48 hours after injury. More serious burning in oral cavity does not mean heavy esophageal damage and vice versa. Consultation with toxicological center.

Therapy:

- Urgent care: neutralization and diluting, in case of the 3rd degree (perforation) surgery is necessary – laparotomy, gastrectomy, esophagectomy. Esophagoscopy and extraction in case of battery in esophagus.
- Intermediate care: Prednisone 1mg/kg/day (must not be given in case of perforation), intravenous ATB, antireflux therapy. Nasogastric probe for 6 weeks in case of circular damage of the 2nd degree or perforation.
    - 1st degree: no farther therapy is needed (a small risk of esophageal stricture), after 3 weeks passage with barium
    - 2nd degree: Prednisone for 4 weeks, ATB for 2 weeks, H2 blockers 2-4 weeks, after 3 weeks passage with barium

- Later care: strictures dilatation if found in anesthesia. This is performed several times a week by anterograde or retrograde way from gastrostomy (the second is safer)

Complications:

- Early: perforation and mediastinitis
- Later: cicatrical strictures of esophagus, malignant tumors as a result of burning (spinocellular carcinoma)

Foreign body in esophagus

Definition: swallowing of a larger foreign body (food is less often than non-food) and embedding in physiological esophageal stricture. According to the duration of the foreign body in the esophagus we divide them into acute and chronic.

Classification:

- organic: coins, pins, toys, batteries
- inorganic: food (bones, gristles, meat …)

Etiopathogenesis: small children put inside their mouth many different things, parts of toys. During game, running, laughing or fright the foreign body can fall inside the esophagus from the mouth. Foreign bodies can be from metal, plastic or they are a part of food – bones, stones. Foreign bodies can have sharp or round edges. The majority of foreign bodies stay stuck in Kilian´s sphincter – the first physiological stricture of the esophagus.

Esophageal varices

Varices are in 90% localized in the lower third of the esophagus, they are rarely in the upper third, exceptionally in the middle third.

Pathogenesis: venous plexuses are subepithelially and submucously in the esophageal wall. Periesophageal varices are found near the fibrous capsula of the esophagus. If blood flow through vena portae is impaired (hepatic cirrhosis or stenosis outside liver), blood has to flow through collateral veins in the esophageal wall which are wider and varicously changed. Very rare are idiopathic varices without portal hypertension or varices in the upper part of the esophagus in obstruction of v. cava by tumors in the mediastinum.

Symptoms: bleeding in almost 70%. If it is small and repeating it causes melena and anemia, if it is larger it causes vomiting of blood. In vomits there are no gastric juices. The patient can vomit swallowed blood as well. Esophageal varices can cause little dysphagic problems.

Diagnostics: On X-ray with a contrast we can find at first irregularity of the lower third of esophagus, later on longitudinal or circular bright areas are present in contrast fluid. If esophagoscopy is performed we can find subepithelial varices (thin, blue strips or nodes) or submucous varices (thick, strips or nodes in same color as esophagus)

Therapy: the primary disease has to be cured. We must premeatus or stop the bleeding from the damaged varix. To stop the bleeding hemostyptics are used as well as Sengstaken-Blakemoore balloon sound. We can sclerotize varices by an injection of sclerotizing fluid or by laser coagulation (argon or neodym laser)

Differential diagnostics of swallowing problems

Definition:

- dysphagia: impaired swallowing
- odynophagia: pain during swallowing

Classification:

- diseases of esophagus and pharynx
- diseases outside the swallowing pathways, which cause pressure from the outside
- neurologic disorder

Swallowing problems are commonly a first sign of organic damage to the esophagus. At first the patient is not able to swallow solid food. In later stages it is impossible to swallow spoon food or even liquids. These symptoms are typical for far gone tumors or achalasia. Next group of disease with odynophagia or dysphagia are patients with foreign body in swallowing pathways. Swallowing problems accompanied with vomiting of indigested food are signs of esophageal diverticulum.

Long lasting swallowing problems lead to significant weight loss, fatigue and nutrition problems. Swallowing problem with an obstruction can be accompanied with laryngeal dysphagia when liquid food runs into airways and cause aspiration bronchopneumonia.

Comments on embryology of external neck

On both sides of pharynx, there are branchial evaginations and branchial arches.

Branchial arches consist of mesenchyme, from the outside they are covered with ectoderm, from inside with entoderm.

- I. branchial arch (mandibular): forms malleolus, incus, ligametum mallei anterior, ligamentum sphenomandibulare, mandible, m.temporalis, m.masseter, m.Pterygoideus medialis and lateralis, m.mylohyoideus, m.tensor tympani, m.tensor veli palatini, m.digastricus (ventral part) – those muscles are innervated by n.mandibularis (3.branch of n.V), as well as skin of mandible and frontal 2/3 of tongue.
- II.branchial arch (hyoid): forms stapes, processus styloideus ossis temporalis, lig. stylohyoideum, upper part and cornu minus ossis hyoidei, m. stapedius, m.

stylohyoideus, m. digastricum (dorsal part), mimic muscles (m.buccinator, m. auricularis, m. orbicularis oris, m. orbicularis oculi, m. occipitofrontalis) – innervation from n. VII.

- III. branchial arch: lower part of body and cornu minus os hyoideum, m. stylopharyngeus, m. constrictor pharyngis superior – innervation of muscles and tongue´s root – n.IX.

- IV. + VI. (or + V.rudimental) branchial arch: cartilages of larynx (cartilago thyroidea, c. cricoidea, c. arytenoidea, c. corniculata, c. cuneiformis), pharyngeal and laryngeal muscles are innervated from n.X (n.laryngeus superior and inferior)

Branchial evaginations:

- I.branchial evagination (pharyngeal): lies between I.and II.branchial arch. Ventral part obliterates, dorsal part dilates into recessus tubotympanicus. It is located between Meckel´s and Reichert´s cartilage. Eardrum, middle ear cavity and hearing tube is formed from recessus tubotympanicus

- II.branchial evagination: form fossae tonsillares and tonsillae palatinae

- III.branchial evagination: medial part forms ductus thymopharyngicus, which later perishes. Lateral part forms thymus and glandula parathyreoidea inferior

- IV.branchial evagination: medial part forms ductus pharyngobranchialis, which later perishes. Lateral part forms ultimobranchial body and glandula parathyreoidea. The base of the upper parathyroid gland descends more slowly than the base of the inferior parathyroid gland from 3.branchial evagination.

- V.branchial evagination: is rudimental, forms ultimobranchial body.

Thyroid gland is unpaired organ communicating with pharyngeal cavity by ductus thyreoglossus. There is tuberculum impair in the opening. The base of the thyroid gland relatively descends into the frontal part of the neck because of embryo growth. Connection with tongue is getting thin to ductus thyreoglossus and enters lingual surface in the form of foramen caecum. Ductus thyreoglossus usually totally perishes; if some remnants remain, they change into small accessory thyroid glands. Its lower part forms lobus pyramidalis of the thyroid gland.