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КАФЕДРА ПОЛИКЛИНИЧЕСКОЙ ТЕРАПИИ

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ДИСПЕПСИЯ В АМБУЛАТОРНОЙ ПРАКТИКЕ

DYSPEPSIA IN OUTPATIENT PRACTICE

Учебно-методическое пособие

Минск БГМУ 2024

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ABBREVIATIONS

CCK — Cholecystokinin
CG — Chronic gastritis
EPS — epigastric pain syndrome
FD — Functional dyspepsia
GI — gastrointestinal
GIP — Gastric inhibitory peptide
GIT — The gastrointestinal tract
H2RA — histamine receptor 2 antagonists
NSAID — Nonsteroidal anti-inflammatory drugs
OLGA — Operative Link on Gastritis Assessment
PDS — postprandial distress syndrome
PPIs — proton pump inhibitors
TCAs — tricyclic antidepressants
VIP — The vasoactive intestinal polypeptide

INTRODUCTION

Dyspepsia is one of the most frequently encountered diseases symptoms in outpatient practice. Establishing the dyspepsia variant and recognizing the cause at an outpatient appointment is a difficult clinical task, which is complicated by the limited time of the initial admission and the diseases wide range, among which a differential diagnosis should be carried out.

GASTROINTESTINAL SYSTEM: BASIC OF ANATOMY AND PHYSIOLOGY

The relevant anatomy knowledge is of vital importance for understanding and managing gastrointestinal (GI) disease. The basic anatomy review is illustrated in fig. 1–3. It may be a good idea to return to this chapter from time to time while reading.

The gastrointestinal tract (GIT) consists of a hollow muscular tube starting from the oral cavity, where food enters the mouth, continuing through the pharynx, esophagus, stomach, and intestines to the rectum and anus, where food is expelled.

There are various accessory organs that assist the tract by secreting enzymes to help break down food into its component nutrients. Thus the salivary glands, liver, pancreas, and gall bladder have important functions in the digestive system. Food is propelled along the length of the GIT by the muscular walls peristaltic movements.

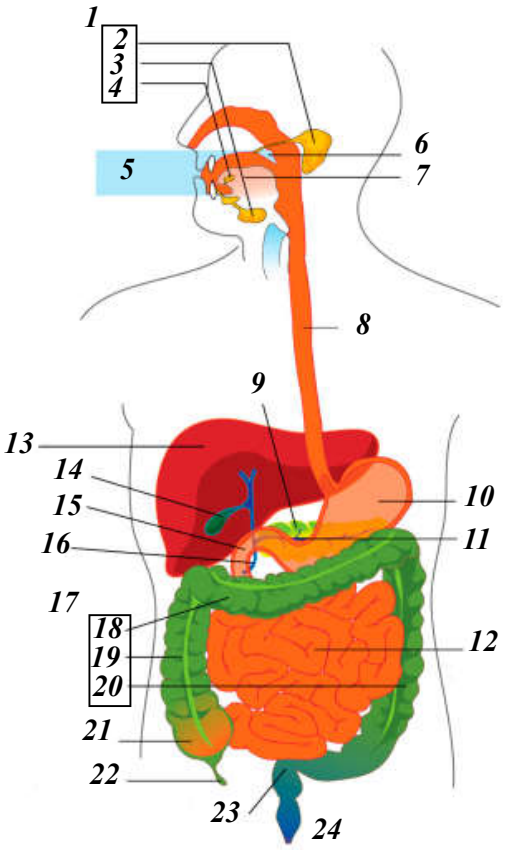
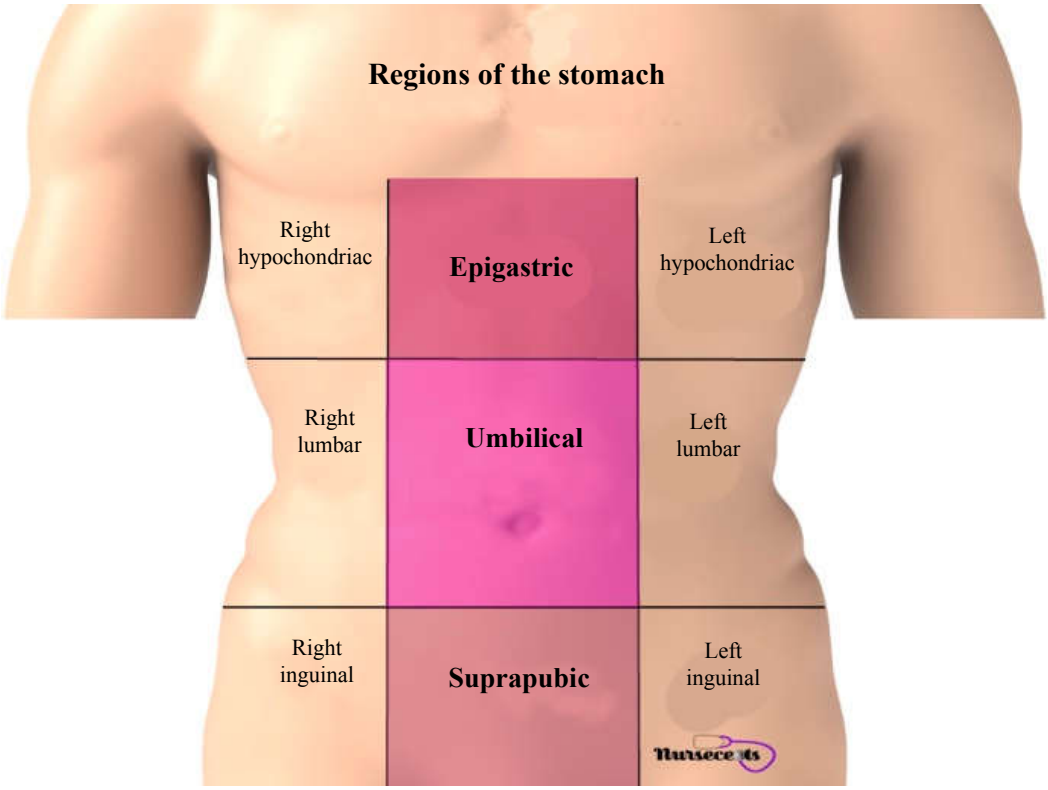


Fig. 1. Upper and lower gastrointestinal tract:
1 — salivare glands: *2* — parotid, *3* — submandibular, *4* — sublingual; *5* — oral cavity; *6* — pharynx; *7* — tongue; *8* — esophagus; *9* — pancreas; *10* — stomach; *11* — pancreatic duct; *12* — lleum (small intestine); *13* — liver; *14* — gallbladder; *15* — duodenum; *16* — common bile duct; *17* — colon: *18* — transverse colon, *19* — ascending colon, *20* — descending colon; *21* — cecum; *22* — appendix; *23* — rectum; *24* — anus

Fig. 2. The Bristol Stool Chart (https://en.wikipedia.org/wiki/Bristol_stool_scale)



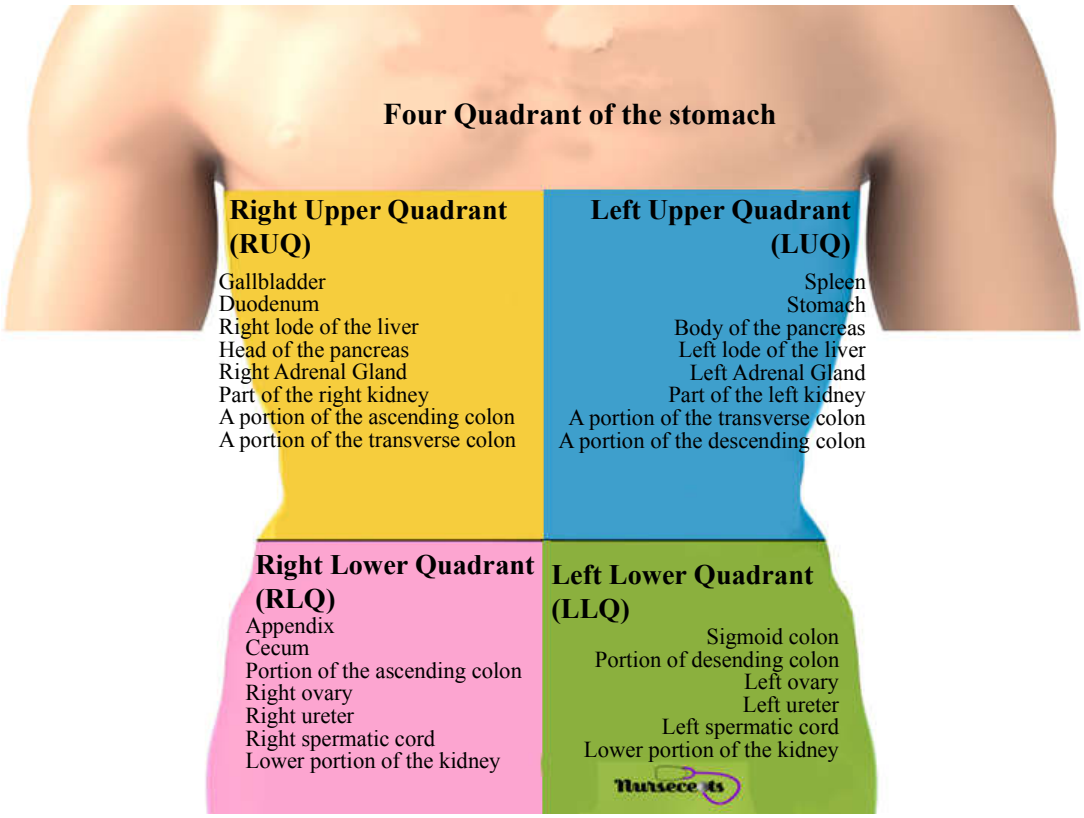


Fig. 3. The abdomen Landmarks (https://nursecepts.com)

The gastrointestinal tract primary purpose is to break food down into nutrients, which can be absorbed into the body to provide energy. First food must be ingested into the mouth to be mechanically processed and moistened. Secondly, digestion occurs mainly in the stomach and small intestine where proteins, fats and carbohydrates are chemically broken down into their basic building blocks. Smaller molecules are then absorbed across the small intestine epithelium and subsequently enter the circulation. The large intestine plays a key role in reabsorbing water excess. Finally, undigested material and secreted waste products are excreted from the body via defecation (feces passing).

In the case of GI disease or disorders, these gastrointestinal tract functions are not achieved successfully. Patients may develop symptoms of nausea, vomiting, diarrhea, malabsorption, constipation or obstruction. Gastrointestinal problems are very common and most people will have experienced some of the mentioned above symptoms several times throughout their lives.

The gastrointestinal tract is divided into upper and lower ones by the Treitz ligament.

The upper gastrointestinal tract consists of the esophagus, stomach and duodenum. The exact demarcation between upper and lower ones can vary. Upon gross dissection, the duodenum may appear to be a unified organ, but it is often divided into two parts based upon function, arterial supply or embryology.

The lower gastrointestinal tract includes most of the small intestine and all of the large intestine. According to some sources, it also includes the anus.

This drawing shows the small intestine position in the gastrointestinal tract. The small intestine is shown surrounded by the colon, on the left, the rectum and anus underneath, the cecum and appendix on its right and the stomach above it.

Key Points:

– digestive system two important functions are digestion and absorption;

– the nutrients that come from food are derived from proteins, fats, carbohydrates, vitamins and minerals. These complex macromolecules must be broken down and absorbed in the GI tract;

– mechanical digestion starts in the mouth with the food physical processing by the teeth and continues in the stomach;

– chemical digestion starts with the enzymes release in saliva and continues in the stomach and intestines;

– during absorption, the nutrients that come from food pass through the small intestine wall and into the bloodstream.

Key Terms:

– *mastication*: The mechanical breakdown process by the teeth; also known as chewing.

– *bolus*: Moistened and mechanically manipulated food.

– *mechanical digestion*: The food breaking down into digestible chunks, normally using the teeth.

– *chemical digestion*: The process that involves the enzymes action to break down food into components that can be absorbed by the small intestine.

– *gastrointestinal tract*: This tract consists of the stomach and intestine, and sometimes includes all the structures from the mouth to the anus. The digestive system is a broader term that includes other structures, including the digestion accessory organs, such as the liver, gallbladder and pancreas.

GIT FUNCTIONS

Let's dwell in more details on the functions of the digestive system each section.

The Oral Cavity Functions to Provide:

– food material sensory analysis before swallowing;

– mechanical processing via the teeth, tongue and palatal surfaces action;

– lubrication by mixing food material with mucus and salivary gland secretion;

– carbohydrates and lipids limited digestion.

Starting with the ***oral mucosa***, which is lined by both keratinized (seen in the tongue and hard palate superior surface) and nonkeratinized squamous epithelial cells (seen in cheeks, lips and the tongue inferior surface), these cells are not known to absorb molecules except for the mucosa inferior to the tongue.

The ***tongue*** functions include mechanical processing by compression, abrasion and distortion; manipulation to assist in chewing and prepare material for swallowing; sensory analysis by touch, temperature, and taste receptors; and mucins and lingual lipase secretion. The lingual lipase has a broad pH and breaks down lipids (mainly triglycerides). The pH of 3.5 to 6 allows lingual lipase to work even in the stomach acid environment.

Within the oral cavity, there are three pairs of the ***salivary glands***. The first pair is the parotid salivary glands located inferior to the zygomatic arch and posterolateral to the mandible. The parotid glands produce serous secretions containing the salivary amylase large amount, which breaks down carbohydrate complexes. Next are the sublingual salivary glands located on the mouth floor. The sublingual glands produce a mucous secretion that serves as both a buffer and lubricant. The third is the submandibular salivary glands, located at the mouth floor within the mandibular groove. They function by secreting the buffers mixture, glycoproteins called mucins and salivary amylase.

Altogether these ***glands*** produce 1.0 to 1.5 liters of saliva each day. Close to 99.4 % of the saliva produced is water and the remaining 0.6 % consists of

electrolytes, buffers, glycoproteins (mucins), antibodies, enzymes and waste products. They function for the mouth lubrication to prevent friction between the oral cavity mucosa and food material; moisten the food material for easy swallowing process; and initiation of lipid and carbohydrate complex digestion.

The teeth provide the food materials mechanical breakdown; for instance, the meat and plant fibers connective tissue in vegetables. This process also saturates the salivary secretions and enzymes within the food material for better digestion.

The ***pharynx*** serves as a food material passageway to the esophagus although it also has a respiratory function for air movement into the lung. During swallowing, the nasopharynx and larynx closure occurs to maintain the food proper direction. This process is achieved by cranial nerves IX and X. From the pharynx, food material goes to the esophagus.

The ***esophagus*** primary function is to empty food materials into the stomach via its longitudinal and circular muscle contraction waves known as peristalsis. The esophagus upper one-third is predominantly skeletal muscle. The middle one-third is both the skeletal and smooth muscle mixture. The lower one-third is mainly smooth muscle. However, during the deglutition act, the buccal phase is the voluntary phase only where one can still control the swallowing process. The skeletal muscles found in the pharynx and upper esophagus are all under the swallow reflex control; hence the swallowing pharyngeal and esophageal phases are under involuntary control help with afferent and efferent fibers of glossopharyngeal and vagus nerves. The esophagus smooth muscles are arranged in a circular and longitudinal fashion and aid in peristaltic movement during swallowing.

Once the food material arrives in the ***stomach***, it can be temporarily stored and mechanically and chemically broken down by the stomach acids and enzymes actions. The intrinsic factor secretion produced by the stomach helps with the vitamin B_{12} proper absorption. The stomach ability to store food stems from its compliance and ability to change size. On average, the stomach lesser curvature has a 10 cm length approximately, and the larger curvature has a 40 cm length approximately. The stomach typically spans from vertebrae T7 and L3, giving it the ultimate ability to hold on to the food large amount.

The stomach function in breaking down food materials mechanically is due to its sophisticated muscular dimensions. The stomach has 3 muscular layers: inner oblique layer, middle circular layer and external longitudinal layer. These 3 stomach muscular layers contraction and relaxation assist in the mixing and churning activities essential in the chyme formation. Then the food material chemical breakdown in the stomach is propagated by the gastric glands produced majorly by the parietal cells, the chief cells, G-cells, the foveolar cells and the mucous neck cells. The parietal cells secrete intrinsic factors and hydrochloric acid. The intrinsic factor produced is essential in the vitamin B_{12} absorption. It binds to B_{12}, allowing for proper absorption at the small intestine ileum. The hydrochloric acid produced

by the parietal cell keeps the stomach pH between 1.5 to 2.0. The stomach acidity brought on by hydrochloric acid destroys most of the microorganisms ingested with food, denatures protein and breaks down plant cell walls, and is essential for the pepsin activation and function, a protein-digesting enzyme secreted by chief cells. The chief cells produce a zymogen called pepsinogen, which gets activated at pH between 1.5 to 2 to become pepsin. Pepsin is a protein-digesting enzyme. The foveolar cells and mucous neck cells produce mucous, which protects the gastric epithelium from acidic corrosion. The G cells are abundant within the stomach pyloric section. They produce gastrin which stimulates secretions from the parietal and chief cells. Within the stomach pyloric section, D cells produce somatostatin, which inhibits gastrin release.

The ***small intestine*** is the next location where digestion takes place. But unlike the stomach, which has minor absorptive properties, 90 % of food absorption occurs in the small intestine. The small intestine has three segments: the duodenum, the jejunum and the ileum. The duodenum receives chyme from the stomach as well as digestive material from the pancreas and the liver. The jejunum is where the bulk of chemical digestion and absorption occur. The ileum also has digestion and absorption functions. The ileum is the small intestine last segment and has the ileocecal valve, a sphincter that controls the material flow from the ileum to the large intestine cecum. The small intestine mucosa has villi, and each villus has multiple microvilli; thereby increasing the surface area exponentially for optimal absorption. There are capillaries extensive networks within the villi that carry absorbed nutrients to the hepatic portal circulation. Also, the lymphatic capillaries vast quantity called lacteals help in the chylomicron transportation to the venous circulation.

The intestine has both endocrine and exocrine glands that produce hormones, enzymes and alkaline mucinous material. The hormones released by the small intestine include:

– gastrin produced by G-cells in the upper small intestine (but mostly found in the stomach);

– cholecystokinin (CCK) produced by I-cells in the upper small intestine;

– secretin produced by the S-cells in the upper small intestine in response to decreased upper intestine pH;

– gastric inhibitory peptide (GIP) produced by K-cells in the upper small intestine in response to fat, amino acids, and glucose;

– pro-glucagon produced by the L-cells in the distal ileum and colon in response to glucose and fat;

– somatostatin is produced by D-cells in the small intestine, including the stomach and pancreas;

– the vasoactive intestinal polypeptide (VIP) produced by parasympathetic ganglia in the small intestine in response to distention;

– motilin produced by M-cells in the upper small intestine.

The enzymes produced by the small intestine include lipase for fats digestion, peptidase for peptide breakdown, sucrase, maltase and lactase for sucrose, maltose, and lactose breakdown, respectively. Then there are the Brunner glands mostly found in the duodenum that produce bicarbonate for acid neutralization.

Within the duodenum, accessory digestive organs such as the liver and pancreas release digestive secretions. The ***liver*** is the largest internal organ and gland in the human body. It has numerous functions, but as the digestive system accessory organ, it produces bile which emulsifies fats and various lipids for optimal digestion. Bile produced in the liver is stored in the gallbladder. The gallbladder contracts to release bile into the duodenum when fat-containing food is present. The pancreas also has exocrine glands that are essential for the food digestion process. The pancreas exocrine glands produce multiple enzyme precursors and enzymes, which include trypsinogen, chymotrypsinogen and procarboxypeptidase, which are activated by enteropeptidase in the small intestine; active alpha-amylase; lipases and colipase, which act on triglycerides and phospholipids; and several other enzymes like ribonuclease, elastase, and collagenase.

The unabsorbed and undigested food material progresses to the ***large intestine***. At this point, it is called feces. The large intestine is about 6 feet long and starts with the cecum, ascending colon, transverse colon, descending colon and sigmoid colon. The large intestine absorbs water and electrolytes. Also, due to the trillions of microbes that live in the large intestine, these organisms can break down the undigested food material. In addition, nutrients such as vitamin K are produced and absorbed in the large intestine. The large intestine peristaltic action moves the feces into the rectum. In the **rectum**, stretch receptors signal for the defecation process to start, which includes a reflexive relaxation of the internal anal sphincter smooth muscle and conscious relaxation of the external anal sphincter skeletal muscle.

GIT CLINICAL EXAMINATION

The gastrointestinal system diseases can be one of many causes, affecting anywhere from the mouth to the anal canal. Within the esophagus there is a wide range of pathologies: scleroderma, esophageal dysmotility, esophageal strictures, esophagitis, achalasia and esophageal varices; these diseases can affect the food movement into the stomach. Further along the gastrointestinal tract, gastritis involves the stomach inflammation. This condition can vary, depending on the symptoms duration. Gastritis may have an acute onset caused by NSAIDs or mucosal ischemia. Gastritis chronic causes are typically due to Helicobacter pylori or autoimmune disease. One of the autoimmune disease such cause is pernicious anemia, a condition preventing the intrinsic factors proper formation to vitamin

B_{12}, a nutrient vital in physiologic processes such as DNA/RNA synthesis and hematopoiesis, and neurologic function. Vitamin B_{12} deficiency can also be attributed to the dietary intake lack, as the nutrient must be acquired through animal products or supplemented food sources.

The small and large bowel diseases include celiac disease, tropical sprue, Whipple disease, Crohn's disease, and ulcerative colitis, which impact the food material digestion and absorption. In addition to pathologic conditions, congenital diseases such as Hirschprung, biliary atresia, intestinal atresia, the intestine malrotation and pyloric stenosis occur during infancy. They may be life-threatening as adequate nutrients cannot be absorbed.

Within the gastrointestinal tract accessory organs, there are hereditary hyperbilirubinemia disorders such as Gilbert syndrome, Dublin-Johnson syndrome and Crigler-Najjar syndrome. The commonality among these conditions is the normal processes impairment that allow proper uptake, conjugation and bilirubin waste products excretion to take place. Other accessory organ pathologies include hemochromatosis, Wilson's disease, biliary tract diseases and pancreatitis. The gallbladder diseases prevent proper storage of bile from the liver, leading to malabsorption in the gut. Examples of these conditions include cholelithiasis, choledocholithiasis and cholecystitis.

All of these diseases warrant proper work-up starting with a thorough history and physical exam. Obtaining a present illness history is essential to the gastrointestinal system disease diagnosis and questions clarification regarding the pain location and duration, radiation or changes in intensity, precipitating factors, associated symptoms such as fever, chills, nausea, vomiting, changes in bowel habitus and stool color. Inquiries on any illness previous episodes or related illnesses and previous surgeries, medication lists and allergies are crucial.

A proper and thorough physical examination is imperative in working up gastrointestinal system diseases. All the abdomen four quadrants must be inspected to appreciate the general abdominal contour. Proper inspection allows for identifying any surgical scars, bulges, hemangiomas or dilated veins of the caput medusae when present. Patients may be asked to cough in order to be checked for abdominal herniation.

After inspection, auscultation is performed to detect any abnormal bowel sounds, such as rubs and bruits. It is necessary to consider the different abdominal organs anatomical location, as it determines the sounds heard and the pathologies correlated. For example, auscultating the right upper quadrant checks for liver rubs and bowel sounds while listening to the left upper quadrant examines rubs or bruits within the splenic region. Pitch, intensity and duration of the sounds should also be appreciated during auscultation.

The abdomen palpation starts at the right upper quadrant to outline the liver size and detect the tenderness signs. The left upper quadrant, periumbilical, left and

right lower quadrants are subsequently palpated to identify any unusual masses or discomfort signs. The liver and spleen are solid organs that, when percussed, elicit a dull sound. Percussing the abdomen in the areas overlying these organs serves the the liver and spleen size assessing purpose, in addition to determining whether tenderness is present. The abdomen percussion can also identify any abnormal gas collection or ascites. The principle behind this technique is to compare the sounds elicited over a particular area with the normal, expected findings.

The rectal exam includes the anal area thorough inspection to identify any skin lesions, scars, fistula tracts or external hemorrhoids. The anal wall careful palpation may help identify any hypertrophic papillae, inflamed crypts, strictures and abnormal sphincter tone that might affect the stool normal passage.

Dyspepsia is any chronic or recurrent discomfort in the epigastric area described as bloatedness, fullness, gnawing or burning continuously or intermittently. About 40 % of the adult population may suffer from dyspeptic symptoms, but most of them are un-investigated because only about 2 % consult their physician.

Symptoms often come and go, rather than being constant and are particularly worse after eating large meals, eating too quickly and if food is eaten shortly before going to bed. The pain from the upper abdomen may spread up to the chest center (behind the breastbone), into the neck or through to the back.

There are some tips for the patient with gastrointestinal complaints physical examination below. By following these recommendations you can determine the dyspepsia cause more easily.

Tip 1. Gather information on the main complaints or symptoms

There are a variety of the upper and lower gastrointestinal complaints or symptoms. Gathering health information about the patient's chief complaints and symptoms will help narrow the gastrointestinal system diagnosis.

Some main gastrointestinal system complaints include nausea, vomiting, loss of appetite, change in bowel habits, diarrhea and constipation.

***Nausea and vomiting*.** Nausea is a sensation that comes in waves in the throat back, the abdomen or the epigastric region that causes the patient to have the urge to vomit.

Vomiting is the stomach forceful voluntary or involuntary emptying. This is a means of the gastrointestinal tract getting rid of the irritation. Sensory fibers stimulate the vomiting center. The abdominal muscles contract, the lower esophageal sphincter opens and the stomach contents are regurgitated out.

Ask the patient the following questions about nausea:

Have you ever felt sick to the stomach?

Have you ever felt sick to the stomach and without vomiting?

How often do you feel sick to the stomach?

Have you ever had periods of retching?

Ask the patient the following questions about vomiting:

Have you ever vomited?

How often do you vomit?

How much vomiting do you at one time? Try help the patient to count.

Do you have pain when vomiting?

Is the vomiting related to meals?

Does the vomiting have smell?

What is the vomiting color?*

Does the vomiting contain blood?

Do you have any vomiting other symptoms?

Is there anything that alleviates nausea? Vomiting?

Is there anything that makes nausea or vomiting become worst?

Change in bowel habit. Questions about bowel habits provide information about the bowel functioning. Assess elimination patterns and any changes in the stool characteristics.

Ask the patient the following questions about bowel habits:

When was your last bowel movement?

How frequently do you have a bowel movement?

What is the bowel movement consistency?

Do you have any diarrhea or constipation?

Have you had any change in bowel habits?

Do you have any problems having a bowel movement?

Do you have to use laxatives?

Can you describe the stool?

What is the stool color?**

Are the stools dark, maroon-colored or black and tarry?

Diarrhea. Diarrhea is the loose, watery, frequent stools passage. This occurs when the small intestine water contents go through the large intestine too fast.

There is not enough time for the water or electrolytes to be absorbed. It can cause problems if the diarrhea is prolonged.

Ask the patient the following questions about diarrhea:

Do you have diarrhea frequently?

How often do you have diarrhea?

How much do you have at one time?

Does any pain accompany diarrhea?

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*Hematemesis is red bloody or coffee-ground emesis. Blackish vomit that looks like coffee ground indicates blood.

**Melena is black tarry stools. Hematochezia is red or maroon-colored stools.

What does the stool look like? Is it oily, frothy, contains pus or mucus?

Does the stool have an unusually foul smell?

Does the stool float?

Do you have excessive gas with diarrhea?

Constipation

Constipation is difficulty passing stools or the infrequent passage of hard stools. Sometimes the intestine contents move through the large intestine too slow.

Too much water is reabsorbed what makes it hard for the feces to continue movement through the system. And, the longer the feces stay in the larger intestine the more water is reabsorbed. This can become a vicious circle.

Ask the patient the following question about constipation:

How often do you have bowel movements?

How often do you have bowel movements normally?

Are the bowel movements painful?

Do you have to strain with the bowel movement?

Do you feel like you do not completely empty your rectum?

Can you describe the stools?

What color are the stools?

Do you frequently use a laxative?

The Bristol Stool Chart is a tool used to assess human stool samples based on its shape and how formed or lose it is. The scale was created in 1997 by the healthcare providers' team at the British Royal Infirmary in Bristol, England.

Tip 2. Gather information about abdomen pain

Abdominal pain is the gastrointestinal system main complaint or symptom. Abdominal pain can be a benign condition or it can be life-threatening.

There are several types of the abdominal pain. These pain types include visceral pain, parietal pain and referred pain.

Visceral pain. Visceral pain is burning, aching, cramping and gnawing pain. It can have additional symptoms such as nausea, vomiting and restlessness.

Visceral pain originates from the intestine or another hollow organ. Also, the pain can arise from the biliary tree. Whenever these organs are stretched beyond capacity the patient will experience visceral pain.

Parietal pain. Parietal pain is usually more severe than visceral pain. This pain type is constant aching pain. It is usually localized over the organs causing the pain.

If a patient moves or coughs, the pain becomes worst. These patients like to remain very motionless.

Referred pain. Referred pain is the pain that is not felt at the origination site. This pain type can be felt in the back, over the chest or at other sites.

As abdominal pain becomes greater, the pain appears to radiate or travel to another site. This pain type can be deep or superficial.

Ask the following questions to gather more information about abdominal pain.

Find out if the pain is acute or chronic.

When did the pain start?

Did the pain start suddenly or gradually?

How long have you experienced this pain?

Does the pain last long or come and go?

Are there certain times when you feel the pain?

Describe the pain. Is the pain aching, gnawing, cramping, burning or stabbing?

Point to where you feel the pain.

Does it move or travel?

Is there anything that aggravates the pain?

What do you do to make the pain feel better?

Is the pain affected by meals, alcohol or medication?

On the 0–10 scale, how severe is the pain (fig. 4)?

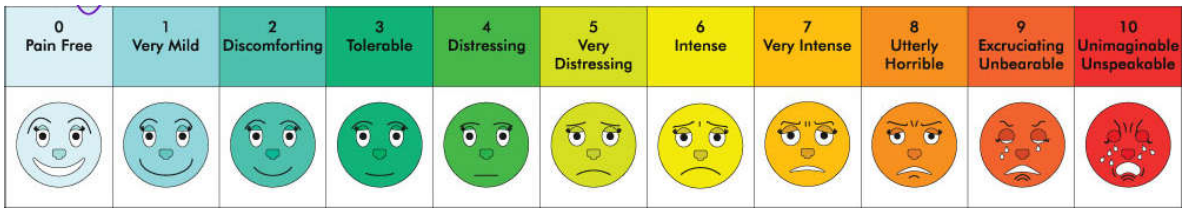

Fig. 4. Pain Scale

Tip 3. Inspection the abdomen

When assessing the abdomen, remember that palpation and percussion are contraindicated in a patient that you suspect of having the abdominal aortic aneurysm, appendicitis diagnosis and some other conditions. Always check for contraindications before beginning the abdominal examination.

To inspect the abdomen:

The patients should be in a lying position with their head on a pillow.

Ask the patient to take off the clothing or lift the gown.

Alternative positions are lying with knees bent on a pillow or a side-lying position.

Standing on the right side is a good position to begin. It will help you proceed to the rest of the evaluation methods.

Keep your eyes at the abdominal area level to begin the inspection.

On inspection look at the abdominal surface.

Inspect the skin. The skin should be smooth and even.

Check the skin for any rashes, scars, lesions.

Inspect the abdominal contour.

Is the abdomen flat, rounded, scaphoid or protuberant?

Check the abdomen for any bulges or masses.

Inspect the abdomen for symmetry. The abdomen should be symmetrical bilaterally.

Inspect the umbilicus.

Look for any abdominal movements.

Also, check for any surgical incisions.

Check for any types of equipment such as G-tube, drains, etc.

Peristalsis is not normally visible but, can be visible with intestinal obstruction.

Check for pulsations. Pulsations can sometimes be visualized with the abdominal aneurysm.

The flat abdomen is commonly evident in a person of normal weight. There is a straight line from the costal margin to the symphysis pubis.

The rounded abdomen has a convex shape. It usually indicates additional fat around the abdominal area. The rounded abdomen is normal in pregnant women and toddlers.

The scaphoid abdomen has a concave shape. From the side, the abdomen looks sunken. This shape is usually evident in patients who are extremely thin.

The protuberant abdomen is evident in patients who are obese or have ascites. The abdomen is extremely rounded. This shape is usual for women who are pregnant but sometimes may be observed in men with ascites.

Tips 4. Auscultate the abdomen

Bowel sounds are auscultated to check for bowel motility.

Auscultation should take place prior to percussion and palpation as they can change the bowel sounds frequency.

To auscultate the abdomen:

Use the stethoscope membrane.

Apply light pressure.

Auscultate the abdomen in all four quadrants.

Listen for bowel sounds, noting the characteristics and frequency.

Listen for bruits or any vascular sounds.

Types of Bowel Sounds

Hyperactive bowel sounds are high-pitched and loud. They are caused by increased gastric motility.

Borborygmus is a type of hyperactive bowel sound. It usually occurs when your stomach growls.

Hypoactive or absent bowel sounds are a decreased gastric motility sign.

Tip 5. Percussion of the abdomen

Percussion of the abdomen is used to evaluate the gas amount in the abdomen.

Also, it can be used to identify organs and masses, e.g. it can help estimate the liver or spleen size.

To percuss the abdomen:

Percuss the abdomen in all four quadrants.

Evaluate the tympany and dullness areas.

Tympany is usually heard over a gas-filled area.

Dullness is heard over solid masses or organs.

Tip 6. The abdominal palpation

Always ask the patients if they have any pain areas before you begin palpation. Palpate the painful areas last.

The abdominal palpation is used to evaluate the abdominal organs size and location. Also, you can use palpation to evaluate tenderness. When palpating the abdomen, begin with light palpation. Light palpation is helpful on tenderness evaluation. Perform deep palpation to check the abdominal masses.

To palpate the abdomen lightly:

With the fingers together, place the hand flat on the abdomen.

Lightly palpate the abdomen using a dipping motion.

Raise the hand off the skin while moving from one place to another.

Palpate all four quadrants.

Check for tenderness.

Palpate for any superficial organs or masses.

Notice if the patient is guarding while palpating.

To palpate the abdomen deeply:

Use the hand palmer side.

Palpate all four quadrants.

Eveluate for the masses presence, noting the location, size, and shape.

Check for tenderness.

To palpate the liver:

Stand on the patient's right side.

Place your left hand behind the patient around the 11th or 12th rib.

Make the patient relax his back on your hand.

Press your left hand forward as the patient relaxes in it. It pushes the liver forward and makes it easier to palpate with your right hand.

Place your right hand on the patient's abdomen right side.

Place your fingertips at the costal margin lower border.

Press gently inward and upward on the abdomen.

Ask the patient to take a deep breath so you can feel the liver borders as it moves under your fingers.

Ask the patient if he has any tenderness.

Try the Hooking method:

This method is useful if you are unable to palpate the liver using the method above.

Stand on the bed right side facing the bed foot.

Use both hands and place them side by side at the costal margin.

Press in and up toward the patient's head with your finger. It is as if you are trying to hook your hands under the ribs.

Ask the patient to take a deep breath.

You should be able to feel the liver with both hands.

To palpate the spleen:

The spleen is not usually palpable except when it is enlarged.

To palpate the spleen you can stand on the bed right side and reach over the patient.

Place your left hand under the patient's left side around the 11th or 12th rib.

Ask the patient to relax his back on your hand.

Lift the patient's ribcage with your left hand.

Palpate the spleen using the right hand fingertips.

Place your hand just below the costal margin.

Push your right hand inward and upward.

Ask the patient to take a deep breath and see if you can feel the spleen with your fingertips.

Ask the patient if he has any tenderness.

Check for rebound tenderness- Rebound tenderness is present when there is pain with the hand withdrawal. The pain is caused by the peritoneum movement.

(Blumberg's Symptom)

Once the area is mapped using palpation, press down with the fingers slowly.

Next, withdraw your fingers quickly.

Observe the patient for the pain signs.

Ask the patient if it hurts more when you press down or when you release.

Ask the patient to point out where it hurts.

Rovsing's Symptom. The Rovsing's symptom is positive when pain is felt in the right lower quadrant when pressure is applied to the abdomen left lower quadrant. Referred rebound tenderness is when the patient has pain in the right side when pressure is applied to the abdomen left side and withdrawn quickly.

Both of the signs indicate appendicitis.

Psoas Symptom. The psoas symptom checks the iliopsoas muscle for irritation.

Place the patient in a supine position.

To perform the psoas symptom, place your hand on the patient's thigh just above the right knee.

Ask the patient to raise his right leg against your hand.

Push against when the patient is trying to raise his leg.

This test is positive if the patient feels pain.

The pain is usually present in the right lower quadrant.

Obturator Symptom. This test is performed to evaluate the obturator muscle for irritation.

Place the patient in a supine position.

Bend the patient's right leg at the hip while the knee is bent at a 90-degree angle.

Take the patient by the right knee and ankle and turn the leg internally at the hip.

It stretches the obturator muscle.

If the patient has pain in the hypogastric or suprapubic area, it is a positive obturator sign.

Cutaneous hyperesthesia. This test is done in the lower abdomen.

Take a piece of skin between your fingers without squeezing.

If the patient has pain, it is an appendicitis indication.

Murphy's Symptom. The Murphy's symptom is found in patients that have an inflamed gallbladder.

Place your fingers under the liver border.

Ask the patient to take a deep breath.

If the patient experiences pain and stops the respiration midway, this is a positive Murphy's symptom.

Pain localization. **The pain is localized below the waist (abdomen)**:

– there are possible urinary system diseases in men, so you must monitor the urination and urine.

– in women there may be the urinary system disease, pregnancy, painful menstruation, internal genitals inflammation.

Pain over the pubis (lower abdomen, «sore abdomen») in women — pathological processes in the bladder, uterus, and appendages, may indicate any problems with the reproductive system. Pelvic pain, occuring each month before menstruation, can indicate endometriosis — a condition in which tissue from the uterus particles moves through the fallopian tubes; get on the ovaries, pelvis, bladder, and other organs. Soreness in the lower abdomen may indicate pelvic inflammatory disease (the uterus, ovaries or fallopian tubes infection). In women of childbearing age, ectopic pregnancy can also cause acute or sharp stabbing pain in the peritoneum.

The pain is localized in the stomach projection: the esophagus and stomach diseases. In myocardial infarction, pneumonia and pyelonephritis may be similar

localization. Soreness in the umbilical region is observed in the small intestine diseases. Acute pain at the abdomen top may be due to perforated ulcer and duodenal ulcer.

Pain in the right iliac region (about the ilium right-wing) — the appendix inflammation in the cecum.

Abdominal pain started in the lower back and moved into the groin: possible pathologies urinary system, urolithiasis. Sudden pain in the lumbar — renal colic.

Abdominal pain is spread into the **right hypochondrium**, can radiate to the right shoulder blade. In this case there may be diagnosed the liver, biliary tract or gall bladder diseases. The urine and feces color must be watched.

The pain occurrence in the upper right abdomen usually confirms biliary colic, which usually causes gallstones in the bile ducts, preventing the bile free outflow from the liver and gall bladder.

Pain in the **left lower abdomen** can be a symptom of diverticulitis. Diverticulitis occurs when the colon walls produce a small spherical capsule called diverticula, which subsequently becomes infected and inflamed. Fever, nausea, vomiting, chills, cramps and constipation are among other diverticulitis symptoms.

Pain in the hernia is a strangulated hernia sign. There has been an increase in seal herniation. The skin over the hernia is often bluish in color.

Girdle pain in the epigastric area radiating to the shoulder blades is typical for acute pancreatitis. The pain is accompanied by nausea and vomiting. The patient usually lies motionless on his side. Belly is swollen and tense.

DYSPEPTIC SYNDROME

Dyspeptic syndrome is a collective term used to describe a variety of the digestive tract motor dysfunction: stomach, intestine, bile ducts (symptoms, pathogenesis).

There is the diseases range associated with these symptoms and patients' survey on an outpatient basis. Dyspepsia, depending on the affected organ, is divided into gastric, intestinal, hepatoduodenal, pancreatogenic (fig. 5).

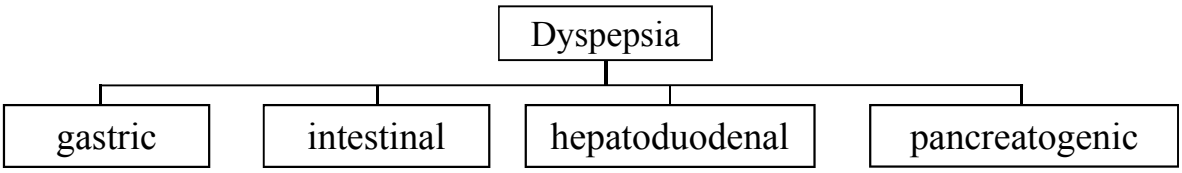

Fig. 5. Types of dyspepsia

Gastric dyspepsia is caused by insufficient or excessive secretion of hydrochloric acid, as well as overly fast or dramatically slow gastric emptying (fig. 6). The symptoms of gastric dyspepsia:

1. **Eructation** — a sudden, involuntary release of stomach contents into the mouth.

2. **Heartburn** — burning sensation along the esophagus or in the epigastric area due to the gastric contents hit into the esophagus. It is often the increased acidity manifestation in the stomach and cardiac sphincter insufficiency.

3. **Nausea** — painful pressure in the epigastric region, accompanied by unpleasant sensation in the mouth and hypersalivation.

4. **Vomiting** — a complex reflex act caused by the vomiting center irritation. So, in this case involuntary gastric contents jerky ejection through the mouth occurs.

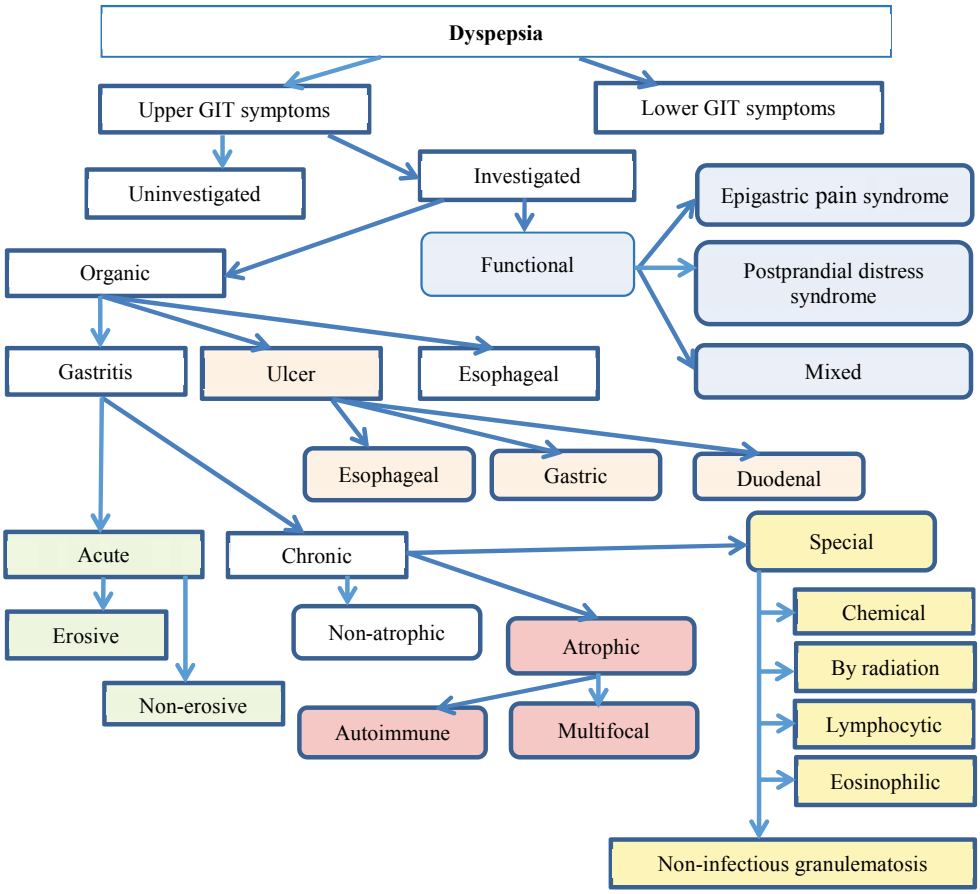

Fig. 6. Classification of dyspepsia

Gastric indigestion often manifests itself in such diseases as chronic gastritis, duodenitis, gastric ulcer and duodenal ulcer.

Intestinal dyspepsia — the food digestion process violation in the intestine as a result of the small intestine, pancreas, liver enzyme deficiency or because of the

food accelerated movement through the intestines. It can lead to dysbacteriosis or the intestine morphology changing.

Types of intestinal dyspepsia: *flatulence*, *rumbling*, *defecation disorder* (diarrhea, constipation and their sequencing).

Types of diarrhea: enteral, colitis.

Enteral diarrhea is observed in the small intestine pathology. A relatively small number of bowel movements (4–6 times a day), malabsorption during defecation and polyfecalia may be noticed.

Colitis diarrhea is observed in the colon pathology. It is characterized by frequent bowel movements (more than 10 times a day), scanty stool, tenesmus, pain during bowel movements, spitting rectal symptom.

Dyspepsia is a very broad concept that includes many other symptoms. There are some classifications to be discussed below.

GASTRIC DYSPEPSIA
CHRONIC GASTRITIS — DA42/ K29 (ICD-11/ICD-10)

Chronic gastritis (CG) is a gastric mucosa chronic inflammation, combined with the epithelial cells renewal impairment with atrophy outcome and disturbances in the secretory, motor and endocrine functions. So, first of all, CG — is a morphological concept.

CG — a collective concept that brings together different etiology and pathogenesis of inflammatory or disregenerative (focal or diffuse) lesions of the stomach mucous membranes and submucosa with symptoms of progressive atrophy, functional and structural adjustment with different clinical manifestations. All this makes the gastritis diagnosis is very difficult, especially at the disease process early stages. Gastric dyspepsia syndrome, combining such clinical symptoms as epigastric pain, loss of appetite, indigestion, nausea and vomiting is observed in many stomach diseases.

The most common cause of chronic gastritis is Helicobacter pylori infection. The source of Helicobacter pylori infection is an infected person and pets. The main route of infection contact is household (oral-oral or fecal-oral).

Risk factors

Factors that increase your risk of gastritis include:

Bacterial infection. Although infection with Helicobacter pylori is among the most common worldwide human infections, only some people with the infection develop gastritis or other upper gastrointestinal disorders. Vulnerability

to the bacterium could be inherited or caused by lifestyle choices, such as smoking and diet.

Regular use of pain relievers. Such common pain relievers as aspirin, ibuprofen and naproxen can cause both acute and chronic gastritis. Using these pain relievers regularly or taking too much may reduce a key substance that helps preserve the stomach protective lining.

Older age. Older adults have an increased risk of gastritis because the stomach lining tends to get thin with age. So, the elderly are more likely to have H. pylori infection or autoimmune disorders than the young ones.

Excessive alcohol use. Alcohol can irritate and erode the stomach lining, what makes your stomach more sensitive to digestive juices. Excessive alcohol use is more likely to cause acute gastritis.

Stress. Severe stress of major surgery, injury, burns or severe infections can cause acute gastritis.

Own body attacking cells in the stomach. This type of gastritis is called autoimmune gastritis. It occurs when the body attacks the cells that make up the stomach lining. This reaction can wear away at the stomach protective barrier.

Autoimmune gastritis is more common in people with other autoimmune disorders, including Hashimoto's disease and type 1 diabetes. Autoimmune gastritis can also be associated with vitamin B_{12} deficiency.

Less Common Causes:

– cocaine addiction;
– bile reflux;
– constipation (the colon constant straining and dryness leads to infection and inflammation);
– crohn's disease;
– poisons consumption and other caustic or corrosive chemical substances;
– sarcoidosis;
– radiation therapy;
– chemotherapy drugs;
– iron and potassium supplements;
– stress as a result of major surgery or trauma or other illness;
– infections caused by:
  • viruses such as HSV, cytomegalovirus (mostly occured in immunocom promised individuals);
  • parasites;
  • fungi.

Primary Prevention. Effective measures for the gastritis primary prevention include:

– avoiding long term or extended use of medications such as NSAIDs (e. g. aspirin, naproxen, ibuprofen);

– abstaining from excessive alcohol consumption is recommended;
– smoking cessation;
– decreasing consumption of caffeine or acidic beverages excessive amounts;
– avoiding spicy foods;
– abstaining from illicit drugs such as cocaine;
– avoiding or reducing stress which may trigger the excessive gastric acid secretion;
– inculcating healthy eating habits, exercising regularly, and maintaining healthy body weight may help in avoiding gastritis;
– hand washing (antibacterial soaps)
– maintain proper hygiene (hand sanitizers, antiseptic washes)
– avoid close contact with infected family members (e. g., kissing, sharing eating utensils and drinking glasses);
– H. pylori eradication is the choice treatment for patients with peptic ulcer disease and low-grade MALT lymphoma;
– test and treat strategy is recommended to prevent peptic ulcer disease in nsaid users and for patients with non-ulcer dyspepsia.

Secondary prevention

Secondary prevention strategies for gastritis following h. pylori infection to prevent recurrence of peptic ulcer disease and gastric cancer include:
– use of antibiotics to prevent infection recurrence;
– post-treatment confirmation of H. pylori eradication after treatment using diagnostic tests.

Classification of gastritis. The Updated Sydney System establishes the gastritis classification and grading which underlines the significance of combining topographical, morphological, and etiological information to help come to a clinical diagnosis (table 1).

Table 1

Classification of Gastritis: Updated Sydney System

Type of Gastritis		Etiology	Gastritis synonyms
Non-atrophic		*Helicobacter pylori* Other factors	Superficial Diffuse antral gastritis (DAG) Chronic antral gastritis (CAG) Interstitial - follicular Hypersecretory Type B
Atrophic	**Autoimmune**	Autoimmunity	Type A Diffuse corporal Pernicious anemia-associated
	Multifocal atrophic	*Helicobacter pylori*	Type B, type AB
		Dietary	Environmental
		Environmental factors	Metaplastic

End of the table 1

Type of Gastritis		Etiology	Gastritis synonyms
Special forms	**Chemical**	Chemical irritation	Reactive
		Bile	Reflux
		NSAIDs	NSAID
		Other agents	Type C
	Radiation	Radiation injury	
	Lymphocytic	Idiopathic? Immune mechanisms	Varioliform (endoscopic)
		Gluten	Celiac disease-associated
		Drug (ticlopidine)	
		Helicobacter pylori	
	Noninfectious granulomatous	Crohn's disease	
		Sarcoidosis	
		Granulomatosis with polyangiitis and other vasculitides	
		Foreign substances	
		Idiopathic	Isolated granulomatous
	Eosinophilic	Food sensitivity	Allergic
		Other allergies	
	Other infectious gastritides	Bacteria (other than *Helicobacter pylori*)	Phlegmonous
		Viruses Fungi Parasites	

In clinical practice, gastritis staging is made using the OLGA (Operative Link on Gastritis Assessment) staging system for reporting gastric histology. Gastritis staging combines the atrophy score which is determined by biopsy and the atrophy topography which is determined by directed biopsy mapping (table 2).

Table 2

The OLGA staging system

Atrophy Score		Corpus			
		No Atrophy (Score: 0)	**Mild Atrophy (Score: 1)**	**Moderate Atrophy (Score: 2)**	**Severe Atrophy (Score: 3)**
ANTRUM	**No Atrophy (Score: 0) (including incisura angularis)**	STAGE 0	STAGE I	STAGE II	STAGE II
	Mild Atrophy (Score: 1) (including incisura angularis)	STAGE I	STAGE I	STAGE II	STAGE III
	Moderate Atrophy (Score: 2) (including incisura angularis)	STAGE II	STAGE II	STAGE III	STAGE IV
	Severe Atrophy (Score: 3) (including incisura angularis)	STAGE III	STAGE III	STAGE IV	STAGE IV

The disease severity and activity can be estimated according to the Sydney classification criteria which are presented in the following table 3.

Table 3

Sydney system for Chronic Gastritis grading

Feature	Definition	Grading guidelines
Chronic inflammation	Increased lymphocytes and plasma cells in lamina propria	Mild, moderate or severe increase in density
Activity	Neutrophilic infiltrates of the lamina propria, pits or surface epithelium	Mild: less than one-third of pits and surface infiltrated Moderate: one-third to two-thirds Severe: more than two-thirds
Atrophy	Loss of specialized glands from either antrum or corpus	Mild, moderate, or severe loss
Helicobacter pylori	*H. pylori* density	Mild colonization: scattered organisms covering less than one-third of the surface Moderate colonization: intermediate numbers Severe colonization: large clusters or a continuous layer over two-thirds of surface
Intestinal Metaplasia	Intestinal metaplasia of the epithelium	Mild: less than one-third of mucosa involved Moderate: one-third to two-thirds Severe: more than two-thirds

In medical practice some other classifications are widely used:

Localization:

– limited (antral, fundic);

– common (diffuse).

Depending on the stomach acid-forming function:

– with encreased and preserved secretion;

– the secretory failure (moderate to severe, including achlorhydria).

By phases:

– aggravation.

– remission.

It's necessary to indicate the classification used in the 10th or 11th international classification of diseases (ICD) according to which the main diagnoses are encrypted in clinical practice (tabl. 4).

Gastritis (gastroduodenitis) is treated with the etiology, endoscopic and histopathological changes, and the process severity. Predominant gastritis is associated with Helicobacter pylori (HP) (85 %), atrophic, usually autoimmune (5 %), often manifested B12-deficiency anemia. Allocated gastritis is associated with taking drugs, granulomatous, eosinophilic.

The most common reason for gastritis development is associated with Helicobacter presence in the body. This bacterium must be studied in more details.

Table 4

Diseases of stomach and duodenum by ICD

ICD-10	ICD-11
K29.0 Acute gastritis	DA42 Gastritis
K29.2 Alcoholic gastritis	DA42.0 Autoimmune gastritis
K29.3 Chronic superficial gastritis	DA42.1 Helicobacter pylori induced gastritis
K29.4 Chronic atrophic gastritis	DA42.2 Eosinophilic gastritis
K29.5 Unspecified chronic gastritis	DA42.3 Lymphocytic gastritis
K29.6 Other gastritis	DA42.4 Allergic gastritis
K29.8 Duodenitis	DA42.5 Gastritis due to duodenogastric reflux
K29.9 Gastroduodenitis, unspecified	DA42.6 Menetrier disease
	DA42.7 Gastritis of unknown aetiology with specific endoscopic or pathological features
	DA42.8 Gastritis due to external causes
	DA42.9 Gastric phlegmon
	DA42.Y Other specified gastritis
	DA42.Z Gastritis, unspecified
	DA51 Duodenitis

Helicobacter pylori is a common gastric pathogen that causes gastritis, peptic ulcer disease, gastric adenocarcinoma and low-grade gastric lymphoma. Infection may be asymptomatic or result in dyspepsia varying degrees. Diagnosis is by the urea breath test, stool antigen test and testing of endoscopic biopsy samples. The treatment is a proton pump inhibitor plus two antibiotics.

H. pylori is a spiral-shaped, gram-negative organism that has adapted to thrive in acid. The organism has been cultured from stool, saliva and dental plaque, which suggests oral-oral or fecal-oral transmission.

Pathophysiology of H. pylori Infection. Effects of H. pylori infection vary depending on the location within the stomach.

Antral-predominant infection results in increased gastrin production, probably via local impairment of somatostatin release. The acid resultant hypersecretion predisposes to prepyloric and duodenal ulcer.

The body-predominant infection leads to gastric atrophy and decreased acid production, possibly via increased local production of interleukin-1 beta. Patients with body-predominant infection are predisposed to gastric ulcer and gastric adenocarcinoma.

Some patients have mixed infections of both antrum and body with varying clinical effects. Many patients with H. pylori infection have no noticeable clinical effects.

Ammonia produced by H. pylori enables the organism to survive in the stomach acidic environment and may erode the mucus barrier. Cytotoxins and mucolytic enzymes (e. g., bacterial protease, lipase) produced by H. pylori may play a role in mucosal damage and subsequent ulcerogenesis.

Infected people are 3 to 6 times more likely to develop stomach cancer. H. pylori infection is associated with intestinal-type adenocarcinoma of the gastric body and antrum but not the gastric cardia cancer. Other associated cancers include gastric lymphoma and mucosa-associated lymphoid tissue (MALT) lymphoma, the monoclonal restricted B-cell tumor.

The scheme (fig. 7) on natural course and progression of Helicobacter pylori gastritis from the non-atrophic form to gastric malignancy (sc. «Correa cascade»). Several potentially pathogenetic factors and mechanisms, linked with carcinogenesis, play a role and are triggered stepwise during the course and progression of the cascade.

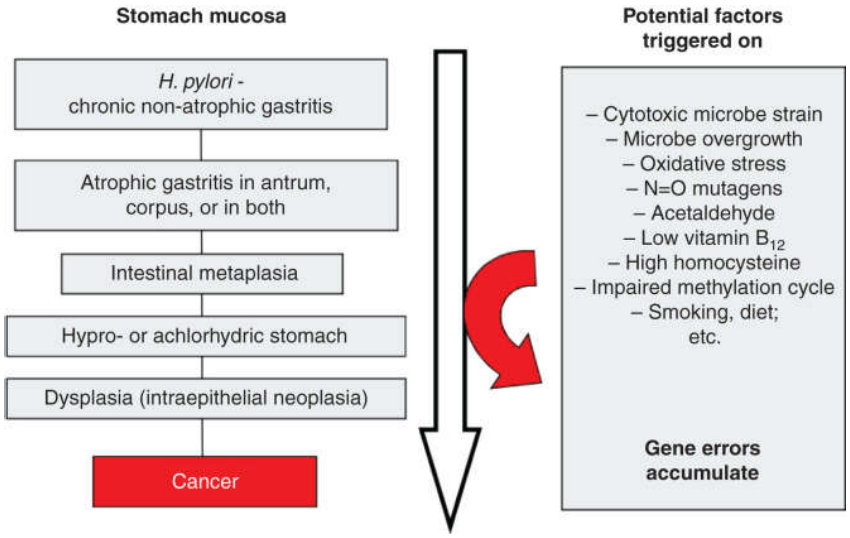

Fig. 7. «Correa cascade» Scand J. Gastroenterol. 2015

H. PYLORI INFECTION DIAGNOSIS

Screening of asymptomatic patients is not warranted. Tests are done during evaluation for peptic ulcer and gastritis. Post treatment testing is typically done to confirm the organism eradication.

Noninvasive tests. Laboratory and office-based serologic assays for antibodies to H. pylori have a sensitivity and specificity of > 85 % and previously were considered the noninvasive tests of choice for initial documentation of H. pylori infection. However, as the infection prevalence has been declined, the percentage of false-positive results with serologic assays has increased significantly, making these tests too unreliable in the most countries and regions. As a result, urea breath testing and stool antigen testing are preferred for initial diagnosis. Qualitative assays remain positive for up to 3 years after successful treatment and because quantitative antibody levels do not decline significantly for 6 to 12 months after treatment, serologic assays are not usually used to assess cure.

Urea breath tests (UBT) use an oral dose of 13C- or 14C-labeled urea. In an infected patient, the organism metabolizes the urea and liberates labeled CO_2, which is exhaled and can be quantified in breath samples taken 20 to 30 minutes after the urea ingestion. Sensitivity and specificity are > 95 %. Urea breath tests are well suited for confirming the organism eradication after therapy. False-negative results are possible with recent antibiotic use or concomitant proton pump inhibitor therapy; therefore, follow-up testing should be delayed ≥ 4 weeks after antibiotic therapy and 1 week after proton pump inhibitor therapy. H2 blockers do not affect the test.

Stool antigen assays have a sensitivity and specificity similar to that of urea breath tests, particularly for initial diagnosis.

Invasive tests. Endoscopy is used to obtain mucosal biopsy samples for a rapid urease test (RUT) or histologic staining. Bacterial culture is of limited use because of the organism fastidious nature. **Endoscopy is not recommended itself for the diagnosis of H. pylori**; noninvasive tests are preferred unless endoscopy is indicated for other reasons.

The RUT, in which the bacterial urease presence in the biopsy sample causes a color change on the special medium, is the choice diagnostic method on tissue samples. Histologic staining of biopsy samples should be done for patients with negative RUT results but suspicious clinical findings, recent antibiotic use, or treatment with proton pump inhibitors. RUT and histologic staining each have a sensitivity and specificity of > 90 %.

Antibody testing is inexpensive and widely available but the poor positive predictive value (PPV) in populations with the low prevalence of H. pylori infection limits its usefulness in clinical practice.

The UBT and fecal antigen tests provide reliable means of identifying active H. pylori infection before the antibiotic therapy.

The UBT is the most reliable non-endoscopic test to document the eradication of H. pylori infection.

The monoclonal fecal antigen test provides another non-endoscopic means of setting H. pylori cure after antibiotic treatment.

Testing to prove H. pylori eradication appears to be most accurate if performed at least 4 weeks after the antibiotic therapy completion. Gastritis diagnosis is based on the clinical picture comprehensive assessment and the laboratory and instrumental studies results. Gastroscopy with biopsies of the stomach antrum and body mucous membrane is crucial. The gastroscopy results only reflect the pathological process location and extent. The gastric secretion biopsy and study of can finally verify chronic gastritis and its form.

Autoimmune gastritis confirmation is the antibodies detection to parietal cells and intrinsic factor.

Treatment

Gastritis treatment depends on the specific cause. Acute gastritis caused by nonsteroidal anti-inflammatory drugs or alcohol may be relieved by stopping those substances usage.

Foods and other substances that should be avoided to reduce or prevent gastritis symptoms include alcohol, spicy, fatty and fried foods. Moreover, anything that might be toxic or irritating to the stomach should also be avoided (for example cigarette smoking, acidic drinks like coffee, garlic powder, chili powder, peppers and tomato products).

Medications used to treat gastritis include. Antibiotic medications to kill H. pylori:

1. **Medications that block acid production and promote healing**. Proton pump inhibitors reduce acid by blocking the cells parts action that produce acid. These drugs include the prescribed and over-the-counter medications omeprazole, lansoprazole, rabeprazole, esomeprazole, dexlansoprazole and pantoprazole.

2. Medications to reduce acid production. Acid blockers — also called histamine (H-2) blockers — reduce the acid amount released into the digestive tract, which relieves gastritis pain and encourages healing. Acid blockers include famotidine, cimetidine and nizatidine.

3. Antacids that neutralize stomach acid. Antacids neutralize existing stomach acid and can provide rapid pain relief. Side effects can include constipation or diarrhea, depending on the main ingredients.

Patients with complications (e. g., ulcer, cancer) should have the organism eradicated. H. pylori eradication can even cure some MALT lymphoma cases (but not other infection-related cancers). Asymptomatic infection treatment has been controversial, but the H. pylori role recognition in cancer has led to the recommendation for treatment. Vaccines, both preventive and therapeutic (i. e., as an adjunct to the infected patients treatment), are under development.

H. pylori eradication requires multidrug therapy, typically antibiotics plus acid suppressants. Proton pump inhibitors suppress H. pylori, and the increased gastric pH accompanying their use can enhance tissue concentration and antimicrobials efficacy, creating the hostile environment for H. pylori.

Empirical antimicrobial therapy is not recommended.

H. pylori testing in all patients with dyspepsia.

If positive for H pylori, eradication therapy recommended.

First-line eradication therapy: Standard triple therapy (i. e., amoxicillin, clarithromycin, proton-pump inhibitor [PPI]).

First-line therapy if high clarithromycin resistance detected: Ten-day sequential therapy with four drugs (i. e., amoxicillin, clarithromycin, metronidazole, PPI).

Second-line therapy if first-line failed: Ten-day levofloxacin-amoxicillin triple therapy.

Start standard triple therapy after 72-96 hours of intravenous PPI, for a 14-day duration.

Quadruple therapy is the best initial therapy in areas where the clarithromycin resistance rate is > 15%. In quadruple therapy, the following oral drugs are given for 14 days:

1. A proton pump inhibitor (PPI): lansoprazole 30 mg 2 times a day; omeprazole 20 mg 2 times a day; pantoprazole 40 mg 2 times a day; rabeprazole 20 mg 2 times a day; or esomeprazole 40 mg once a day;
2. Bismuth subsalicylate (524 mg 4 times a day);
3. Metronidazole 250 mg 4 times a day;
4. Tetracycline 500 mg 4 times a day.

Triple therapy was the most frequently prescribed scheme for H. pylori infection. However, in many world regions, the clarithromycin resistance rate has been increasing and triple therapy failure is likely increasingly. Thus, this scheme is not recommended for initial therapy unless ≥ 85 % of H. pylori local strains are known to be susceptible or the scheme is known to be still clinically effective in the local area.

The following oral drugs are given for 10 to 14 days:

1. A proton pump inhibitor (PPI);
2. Amoxicillin (1 g 2 times a day) or metronidazole 250 mg 4 times a day;
3. Clarithromycin (500 mg 2 times a day).

For multidrug-resistance strains of H. pylori, triple therapy with a proton pump inhibitor, rifabutin and amoxicillin appears to be effective.

Infected patients with duodenal or gastric ulcer require the acid suppression continuation for at least 4 weeks. Eradication may be confirmed by the urea breath test, stool antigen test or upper endoscopy done ≥ 4 weeks after therapy completion. The eradication confirmation is reasonable in all treated patients but is mandatory in patients who have H. pylori infection serious manifestations (e. g., bleeding ulcer). The recurrent bleeding ulcer is likely if the infection is not eradicated.

If either quadruple or triple therapy fails to eradicate H. pylori, treatment is repeated. If two courses are unsuccessful, some authorities recommend endoscopy to obtain cultures for sensitivity testing. If bismuth quadruple therapy fails, clinicians should engage in the shared decision-making discussion with patients to determine whether they should receive levofloxacin triple therapy (with amoxicillin), rifabutin triple therapy or alternate bismuth-containing therapy.

Autoimmune (atrophic) gastritis with megaloblastic anemia is confirmed by bone marrow examination.

Drug treatment includes vitamin B_{12} (or cyanocobalamin) 1000 mg intramuscularly for 6 days, then — in the same dose 1 day a week for a month, and in the subsequent long-term (life) 1 every 2 months. You can assign replacement therapy achlorhydria, enzyme preparations and preparations of nicotinic acid.

Symptomatic treatment with the following drug combinations:

– *in non-ulcer dyspepsia* — gastrocepin to 25–50 mg 2 times a day + Maalox (Gastal, Aluminium phosphalugel, remagel, etc.), 2 tablets or 15 ml (sachet) 3 times a day 1 hour after eating;

– in case of *hypomotoric dyspepsia* — domperidone (Motilium) or cisapride (koordinax) 10 mg 3–4 times before meals + Maalox (or other antacids) to 2 tablets or 15 ml (sachet) three times a day an hour after taking food.

Indications for hospitalization. Indications for planned hospitalization:

– expressed worsening;
– the disease severe form;
– uneffective outpatient treatment;
– the need for hospital examination to differential diagnosis;
– the need for surgical treatment. Indications for emergency admissions:
– bleeding.

Length of hospitalization is usually 10 days. But it may be reduced considering the disease morphological manifestations etiology and severity. Most treatment is carried on the outpatient basis.

PEPTIC AND DUODENAL ULCER DA60-62/ K25 (ICD-11/ICD-10)

Peptic ulcer disease (PUD) is a break in the the stomach inner lining, the small intestine first part or sometimes the lower esophagus. An ulcer in the stomach is called the gastric ulcer, while one in the intestines first part is the duodenal ulcer. The most common symptoms of the duodenal ulcer are waking at night with upper abdominal pain and upper abdominal pain that increases with eating. With the gastric ulcer, the pain may worsen with eating. The pain is often described as a burning or dull ache. Other symptoms include belching, vomiting, weight loss or poor appetite.

About a third of older people have no symptoms. Complications may include bleeding, perforation and the stomach blockage. Bleeding occurs in as many as 15 % of cases.

Common causes include the bacteria Helicobacter pylori and non-steroidal anti-inflammatory drugs (NSAIDs). Other less common causes include tobacco smoking, stress as a result of other serious health conditions, Behçet's disease, Zollinger–Ellison syndrome, Crohn's disease and liver cirrhosis. Older people are more sensitive to the NSAIDs ulcer-causing effects. The diagnosis is typically suspected due to the presenting symptoms with confirmation by either endoscopy

or barium swallow. H. pylori can be diagnosed by testing the blood for antibodies, the urea breath test, testing the stool for the bacteria signs or stomach biopsy (for more details look at the gastritis topic).

Etiology. Peptic ulcer disease (PUD) has various causes; however, Helicobacter pylori-associated PUD and NSAID-associated PUD account for the disease etiology majority (fig. 8).

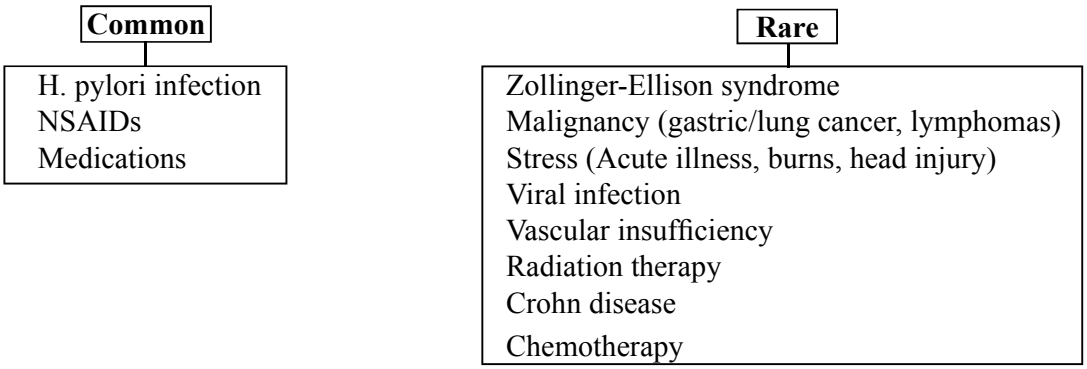

Fig. 8. Causes of peptic ulcer disease

Helicobacter Pylori-Associated PUD. H. pylorus is a gram-negative bacillus that is found within the gastric epithelial cells. This bacterium is responsible for 90 % of duodenal ulcers and 50 % to 80 % of gastric ulcers. The organism has the virulence factors wide spectrum allowing it to adhere to and inflame the gastric mucosa. This results in hypochlorhydria or achlorhydria, leading to gastric ulceration.

Virulence Factors of Helicobacter Pylori. Urease: The urease secretion breaks down urea into ammonia and protects the organism by neutralizing the acidic gastric environment.

Toxins: CagA/VacA is associated with stomach mucosal inflammation and host tissue damage.

Flagella: Provides motility and allows movement toward the gastric epithelium.

NSAID-associated PUD. NSAIDs use is the second most common cause of PUD after H. pylori infection. The prostaglandin secretion normally protects the gastric mucosa. NSAIDs block prostaglandin synthesis by inhibiting the COX-1 enzyme, resulting in decreased gastric mucus and bicarbonate production and the decrease in mucosal blood flow.

NSAIDs are more commonly responsible for gastric ulcers than duodenal ulcers.

NSAIDs cause ulcers by the following mechanism:

- inhibits systemic prostaglandins production;
- decreases blood flow;
- decreases mucus production;
- inhibits leucocyte adhesion.

Apart from NSAIDs, corticosteroids, bisphosphonates, potassium chloride and fluorouracil have been implicated in the PUD etiology.

Smoking also appears to play a role in duodenal ulcers, but the correlation is not linear. Alcohol can irritate the gastric mucosa and induce acidity.

Classification. Peptic ulcer disease may be classified according to location into two subtypes:

– gastric ulcers;

– duodenal ulcers.

Gastric ulcer is further divided on the location and endoscopic findings basis.

Johnson classification. Gastric ulcer is further classified into 3 subtypes depending on their location:

Type 1: Ulcer present at the stomach body without involving duodenum, pylorus or prepyloric region and not associated with the gastric acid hypersecretion.

Type 2: Ulcer present at the stomach body combined with duodenum and associated with gastric acid hypersecretion.

Type 3: Ulcer close to pylorus and associated with gastric acid hypersecretion.

Sakita classification. Gastric ulcer classification by Sakita's using endoscopic staging system into three stages: active, healing and scarring.

Active stage:

A1 — Surrounding mucosa is found to be edematously swollen and there is no regenerating epithelium observed on endoscopy.

A2 — Surrounding mucosa is less edematous, a small amount of regenerating epithelium is observed at the ulcer margin. A red halo in the marginal zone, a white slough circle and converging mucosal folds at the ulcer margin are frequently observed.

Healing stage:

H1 — The white coating is becoming thin and the regenerating epithelium is extending into the ulcer base. The gradient between the ulcer margin and the ulcer floor is becoming flat.

The ulcer crater is still evident and the ulcer margin is sharp. The mucosal defect diameter is about one-half to two thirds that of A1.

H2 — The defect is smaller than in H1 and the regenerating epithelium covers most of the ulcer floor. The white coating area is about a quarter to one-third that of A1.

Scarring stage:

S1 — The regenerating epithelium completely covers the ulcer floor. The white coating has disappeared. Initially, the regenerating region is markedly red, but upon close observation, many capillaries can be observed and is called «red scar».

S2 — In several months to few years, the redness is reduced to the surrounding mucosa color and is called «white scar».

Complications. Acute complications.

Bleeding is the most common complication. Sudden large bleeding can be life-threatening. Bleeding occurs when the ulcer erodes one of the blood vessels around the stomach.

Perforation is most commonly occurring at the duodenal bulb (62 %), followed by the pyloric region (20 %) and the gastric body (18 %). Erosion of the gastro-intestinal wall by the ulcer leads to the stomach or intestinal content spillage into the abdominal cavity leading to fatal consequences.

Perforation at the stomach anterior surface leads to acute peritonitis, initially chemical and later bacterial peritonitis. Often, the first symptom is sudden intense abdominal pain.

Posterior wall perforation leads to **pancreatitis**, pain in this situation often radiates to the back.

Penetration is also the perforation form when the ulcer continues into adjacent organs such as the liver and pancreas.

Obstruction. Occurs as the result of stricture and scarring narrowing in the duodenum leading to gastric outlet obstruction.

Chronic complications:

- Chronic Helicobacter pylori infection leads to;
- Gastric cancer;
- MALT lymphoma;
- Iron deficiency anemia;
- Idiopathic thrombocytopenic purpura.

Prognosis. Prognosis is good if the Helicobacter pylori eradication therapy is taken. The recurrence rate of patients with peptic ulcer disease is less than 20 %.

Diagnosis. The PUD diagnosis requires history taking, physical examination, and invasive/non-invasive medical tests. The careful history should be obtained and noted for any complications presence. The patient reporting of epigastric abdominal pain, early satiety and fullness follows the PUD meal raise suspicion. The gastric ulcers pain increases 2 to 3 hours after a meal and may result in weight loss, whereas the duodenal ulcers pain decreases with a meal which can result in weight gain. Any patient presenting with anemia, melena, hematemesis or weight loss should be further investigated for the PUD complications, predominantly bleeding, perforation or cancer. The physical exam may reveal epigastric abdominal tenderness and anemia signs.

History and Physical Examination. Signs and symptoms of peptic ulcer disease may vary depending on the disease location and the patient's age. Gastric and duodenal ulcers can be differentiated from their symptoms timing in relation to meals. Nocturnal pain is common with duodenal ulcers. Those with gastric outlet obstruction commonly report the abdomen bloating and fullness history.

Common signs and symptoms include:

- epigastric abdominal pain;
- bloating;
- abdominal fullness;
- nausea and vomiting;
- weight loss/weight gain;
- hematemesis;
- melena.

Warning symptoms or alarm symptoms that should prompt urgent referral include:

- unintentional weight loss;
- progressive dysphagia;
- overt gastrointestinal bleeding;
- iron deficiency anemia;
- recurrent emesis;
- family history of upper gastrointestinal malignancy;

Investigations. Esophagogastroduodenoscopy (EGD): Gold standard and most accurate diagnostic test with sensitivity and specificity up to 90 % in diagnosing gastric and duodenal ulcers. The American Society of Gastrointestinal Endoscopy has published guidelines on the endoscopy role in patients presenting with upper abdominal pain or dyspeptic symptoms suggestive of PUD. Patients over 50 years of age and new dyspeptic symptoms onset should get evaluated by the EGD. Anyone with the presence of disturbing symptoms should undergo EGD irrespective of age.

Barium swallow: It is indicated when EGD is contraindicated.

Complete blood work, liver function and amylase and lipase levels.

Serum gastric is ordered if Zollinger Ellison syndrome is suspected.

Helicobacter pylori testing.

The abdomen computerized tomography with contrast is of limited value in the PUD diagnosis itself but is helpful in the complications diagnosis like the perforation and gastric outlet obstruction.

Differential Diagnosis. The following conditions can present with symptoms similar to peptic ulcer disease and it is important to be familiar with their clinical presentation in order to make the correct diagnosis.

Gastritis — the gastric mucosa inflammatory process from immune-mediated or infectious etiology presenting with upper abdominal pain and nausea. Clinical presentation is very similar to that of peptic ulcer disease.

Gastroesophageal reflux disease (GERD) — patients usually describe a burning sensation in the epigastrium and lower retrosternal area, excessive salivation or food material intermittent regurgitation.

Gastric cancer — apart from abdominal pain, patients usually describe disturbing symptoms like weight loss, melena, recurrent vomiting or malignancy evidence elsewhere in case of metastasis.

Pancreatitis — epigastric or right upper quadrant pain that is more persistent and severe, worse in the supine position, and patients usually have the alcoholism or gallstones history. Elevated serum amylase and lipase are useful in the diagnosis.

Biliary colic — intermittent, severe deep pain in the right upper quadrant or epigastrium precipitated by fatty meals.

Cholecystitis — right upper quadrant or epigastric pain that usually lasts for hours and is exacerbated by fatty meals and associated with nausea and vomiting. Fever, tachycardia, positive Murphy sign, leukocytosis and abnormal liver functions help further distinguish this from biliary colic.

These are some potentially life-threatening conditions that can also have similar presentations.

Myocardial infarction — especially in the inferior wall and right ventricular involvement, sometimes patients can present with epigastric pain with nausea and vomiting. Other symptoms presence like dizziness, breath shortness and abnormal vital signs in a high-risk patient should worn the clinician to look for this.

Mesenteric ischemia — while acute mesenteric ischemia presents with severe, acute onset abdominal pain; the chronic variant usually presents with ongoing post-prandial epigastric pain and can be mistaken for peptic ulcer disease. Older age, risk factors presence for atherosclerosis and weight loss should prompt a workup for the same.

Mesenteric vasculitis — unexplained abdominal symptoms with or without lower gastrointestinal bleeding in a patient with other features from underlying systemic vasculitis should raise the mesenteric vasculitis suspicion.

Treatment. Antisecretory drugs used for peptic ulcer disease (PUD) include H2-receptor antagonists and the proton pump inhibitor. PPIs have largely replaced H2 receptor blockers due to their superior healing and efficacy. PPIs block acid production in the stomach, providing the symptoms relief and promote healing. Treatment may be incorporated with calcium supplements as the PPIs long-term use can increase the bone fractures risk. NSAIDs induced PUD can be treated by stopping the NSAIDs use or switching to the lower dose. Corticosteroids, bisphosphonates and anticoagulants should also be discontinued if possible. First-line treatment for H. pylori-induced PUD is the triple scheme comprising two antibiotics and the proton pump inhibitor. Pantoprazole, clarithromycin and metronidazole or amoxicillin are used for 7 to 14 days. Antibiotics and PPIs work synergistically to eradicate H. pylori. The antibiotic selected should take into consideration the antibiotic resistance presence in the environment. If first-line therapy fails, quadruple therapy with bismuth and different antibiotics is used.

Refractory Disease and Surgical Treatment. Surgical treatment is indicated if the patient is unresponsive to medical treatment, noncompliant or at complications high risk. The refractory peptic ulcer is one over 5 mm in diameter that does not heal despite 8–12 weeks of PPI therapy. The common causes are persistent H. pylori infection, continued the NSAIDs use or significant comorbidities that impair ulcer healing or other conditions like gastrinoma or gastric cancer. If the ulcer persists despite addressing the above risk factors, patients can be candidates for surgical treatment. Surgical options include vagotomy or partial gastrectomy.

FUNCTIONAL DYSPEPSIA DD90.3/ K30 (ICD-11/ICD-10)

Functional dyspepsia is one of the most prevalent functional gastrointestinal disorders. Functional dyspepsia comprises three subtypes with presumed different pathophysiology and etiology: postprandial distress syndrome (PDS), epigastric pain syndrome (EPS) and the subtype with overlapping PDS and EPS features.

Functional dyspepsia symptoms can be caused by disturbed gastric motility (for example, inadequate fundic accommodation or delayed gastric emptying), gastric sensation (for example, sensations associated with hypersensitivity to gas and bloating) or gastric and duodenal inflammation. A genetic predisposition is probable but less evident than in other functional gastrointestinal disorders, such as irritable bowel syndrome (IBS). Psychiatric comorbidity and psychopathological state and trait characteristics could also play a part, although they are not specific to functional dyspepsia and are less pronounced than in IBS. Possible differential diagnoses include Helicobacter pylori infection and peptic ulceration. Pharmacological therapy is mostly based on the functional dyspepsia subtype, such as prokinetic and fundus-relaxing drugs for PDS and acid-suppressive drugs for EPS, whereas centrally active neuromodulators and herbal drugs play a minor part. Psychotherapy is effective only in the patients' small subset, whereas the life quality can be severely affected in nearly all patients.

Rome IV diagnostic criteria for functional dyspepsia subtypes (2016) (*https://theromefoundation.org/rome-iv/rome-iv-criteria)

FUNCTIONAL DYSPEPSIA

One or more of the following:
- bothersome postprandial fullness;
- bothersome early satiation;
- bothersome epigastric pain;

– bothersome epigastric burning;

– no structural disease evidence (including upper endoscopy) that is likely to explain the symptoms;

– criteria fulfilled for the last 3 months with symptom onset at least 6 months prior to diagnosis.

Postprandial Distress Syndrome (PDS). Must include one or both of the following at least 3 days a week for the last 3 months with symptom onset at least 6 months prior to diagnosis:

1. Bothersome postprandial fullness (i. e., severe enough to impact on usual activities)
2. Bothersome early satiation (i. e., severe enough to prevent finishing a regular size meal)

No evidence of organic, systemic or metabolic disease that is likely to explain the symptom on routine investigations (including upper endoscopy)

Supportive criteria:

1. Postprandial epigastric pain or burning, epigastric bloating, excessive belching and nausea can also be present
2. Vomiting warrants consideration of another disorder
3. Heartburn is not a dyspeptic symptom but may often co-exist
4. Symptoms that are relieved by the feces or gas evacuation should not be considered generally as the dyspepsia part
5. Other individual digestive symptoms or symptoms groups (e.g., from GERD and IBS) may co-exist with PDS

Epigastric Pain Syndrome (EPS). Must include one or both of the following symptoms at least 1 day a week for the last 3 months with symptom onset at least 6 months prior to diagnosis:

1. Bothersome epigastric pain (i. e., severe enough to impact on usual activities)
2. Bothersome epigastric burning (i. e., severe enough to impact on usual activities)

No organic, systemic or metabolic disease evidence that is likely to explain the symptoms on routine investigations (including upper endoscopy).

Supportive criteria:

1. Pain may be induced by meal ingestion, relieved by meal ingestion or may occur while fasting.
2. Postprandial epigastric bloating, belching and nausea can also be present.
3. Persistent vomiting likely suggests another disorder.
4. Heartburn is not a dyspeptic symptom but may often co-exist.
5. The pain does not fulfill biliary pain criteria.

6. Symptoms that are relieved by the feces or gas evacuation should not be considered generally as dyspepsia part.

7. Other digestive symptoms (such as from GERD and IBS) may co-exist with EPS.

The dyspepsia etiology has been poorly defined; however, numerous pathophysiologic mechanisms, most of which are directed at gastroduodenal pathways, have been proposed to explain the disorder. Many mechanisms are currently being investigated as the functional dyspepsia symptoms potential causes. Given the potentially unidentified etiologies number for dyspepsia symptoms and the word association functional with the organic cause lack for symptoms, we cautiously use the term functional dyspepsia to describe dyspepsia symptoms without an identified organic etiology.

By definition, functional dyspepsia is diagnosed in the organic etiology absence for the dyspepsia symptoms. As outlined previously, patients with functional dyspepsia report the symptoms range that can vary greatly in severity, and symptoms are not the reliable way to differentiate organic from functional dyspepsia. Thus, the evaluation goal is to rule out organic etiologies for the patient's symptoms. Evaluation is based on patient age, presence of disturbing features, symptoms severity, malignancy risk and physical examination findings. Esophagogastroduodenoscopy (EGD) is recommended in patients 60 years of age or older or in any patient with more than 1 disturbing feature, the rapidly progressive disturbing feature, clinically significant weight loss (typically > 5 % of baseline body weight), or overt gastrointestinal bleeding. EGD with gastric biopsies is recommended in any patient 60 years of age or older with dyspepsia due to the cancer increased risk in this age group. In order to ensure the H. pylori infection detection, gastric biopsies should be obtained from the lesser antrum curvature, the antrum greater curvature, the body lesser curvature, the body greater curvature, and incisura angularis. 24 Duodenal biopsies should be obtained in immunosuppressed patients, particularly bone marrow transplant patients, to exclude graft vs host disease or infection. Additionally, given increasing evidence of duodenal pathology driving functional dyspepsia symptoms is the argument, which can be made to obtain duodenal biopsies in all patients undergoing EGD for the dyspepsia evaluation.

A typical history of long-standing troublesome early satiety and postprandial fullness is sufficient to make the clinical diagnosis and commence treatment, but often gastroscopy is required. Any of the following **red flag symptoms** should prompt endoscopy:

- new onset in older age;
- unintended weight loss;
- vomiting;
- bleeding;

– iron deficiency anaemia;
– family history of upper gastrointestinal cancer;
– progressive dysphagia or odynophagia.

Patients under 60 years of age without disturbing features should undergo H. pylori testing via stool antigen testing or urea breath test, followed by treatment and eradication confirmation if testing is positive for active infection. It is important to ensure that patients undergoing testing do not take the proton pump inhibitor (PPI) for 4 weeks prior to testing, as PPI use can cause the false-negative test result.

EGD performed for dyspepsia evaluation identifies peptic ulcer disease in approximately 10 % of cases, erosive esophagitis in 6 % and gastroesophageal malignancy in less than 1 %. Thus, EGD evaluation is unrevealing in a majority of cases. If evaluation via EGD is nondiagnostic, further evaluation should be based on the patient's symptoms severity and risk factors. Gastric emptying testing should be considered in patients with prominent nausea or vomiting, particularly if gastroparesis risk factors are present (e. g., diabetes, connective tissue disorder evidence or more diffuse gastrointestinal dysmotility evidence). Celiac disease should be ruled out in patients with dyspepsia, via either duodenal biopsies or anti-tissue transglutaminase immunoglobulin A antibody serology testing. Medications should also be reviewed, as dyspepsia symptoms can be induced by nonsteroidal anti-inflammatory drugs; bisphosphonate; or antibiotic, neuropsychiatric, antidiabetic or anti-hypertensive medication use. The patient's diet should also be reviewed for potential triggers, including alcohol or caffeine use.

Despite the increasing number of patients undergoing bariatric surgery, there is limited evidence regarding the dyspepsia prevalence or the dyspepsia symptoms recommended diagnostic evaluation in these patients. Bariatric surgery is intended to limit oral intake by the dyspepsia inducing symptoms such as early satiety and postprandial fullness. However, there is the dyspepsia symptoms growing awareness in these patients' population that can occur many years after the initial surgery and in the normal EGD setting. In patients with complaints that exceed expected postsurgical symptoms, EGD with gastric pouch and jejunal biopsies is reasonable to rule out H. pylori infection, marginal ulcers or stricture formation.

Treatment. There are many treatment options available for functional dyspepsia, with some being more effective than others (table 5). Many patients will respond to non-pharmacological management and drug therapy should be reserved for refractory cases.

Making a firm diagnosis even in the endoscopy absence is sound medical practice and probably therapeutic. Functional dyspepsia is common and impacts on the life quality, but the good news is that there is no associated increased mortality. Reassurance, explanation and advice to reduce stress should be routine. Depression should be excluded by asking simple screening questions.

Table 5

Usefulness of therapies for functional dyspepsia

Therapy	Functional dyspepsia subtypes	
	Epigastric pain syndrome	**Postprandial distress syndrome**
Reassurance	+	+
Diet	+	+
Acid suppression	++	+
Prokinetics	+	++
Fundic relaxors	-	+
Tricyclic antidepressants	++	+
Rifaximin	+	+
Psychological therapy	+	+

– not useful, + limited evidence of efficacy, ++ efficacious

Diet. Traditionally eating smaller regular low-fat meals is the advice offered, as the stomach and duodenum can process these more easily (high fat intake slows gastric emptying) and gastric distension is minimised. Wheat may induce typical dyspepsia symptoms. Eliminating it may provide relief in some patients although strong empirical evidence is lacking. Other triggers have been identified, including fatty, fried or spicy foods and carbonated drinks, and avoiding these may be of benefit.

Acid suppression. Reducing the acid bathing amount the duodenum may be helpful. Proton pump inhibitors are superior to placebo in functional dyspepsia. However, they have risks with the long-term use. The majority of patients do not respond to this therapy, and it is the most useful in those with epigastric pain. The alternative is H2 receptor antagonist therapy, which is also superior to placebo. Some patients find this helpful even if proton pump inhibitors have failed. Antacids and sucralfate are not efficacious.

Prokinetics. Domperidone is sometimes prescribed but the evidence for efficacy in functional dyspepsia is very limited. Cisapride has a better evidence base. Both of these drugs prolong the QT interval and must be used with caution. ECG monitoring is recommended. Prokinetics help postprandial distress more than pain. Metoclopramide should be avoided unless nausea is a serious issue as irreversible tardive dyskinesia is the concern. For nausea in such cases the 5HT3 antagonist (ondansetron) is preferred.

Fundic relaxors. Fundic relaxors can be considered for people unresponsive to prokinetics. Cisapride relaxes the gastric fundus, but alternative options include the anti-anxiety drug buspirone or Iberogast.

Table 6

Prokinetic Drugs Used in the Functional Dyspepsia Treatment

Drug	Mechanism of action	Dose	Special comments	Side effects
Levosulpiride	Dopamine D2 receptor antagonist, 5-HT4 receptor agonist, weak 5-HT3 receptor antagonist	25 mg tid	Limited to short duration use for avoiding side effect	Menstrual abnormalities and galactorrhea, drug induced parkinsonism
Metoclo-pramide	Dopamine D2 receptor antagonist, 5-HT4 receptor agonist	5–10 mg tid (max. 30 mg per day)	Limited to only 5 day- use/ treatment, maximal dose: 0.5 mg/kg per day (both adult and child)	Extrapyramidal symptom, gynecomastia, galactorrhea, menstrual irregularities
Domperidone	Dopamine D2 receptor antagonist	10 mg tid (max. 30 mg in a day)	Limited to one-week use/ treatment	Gynecomastia, galactorrhea, menstrual irregularities
Itopride	Dopamine D2 receptor antagonist, inhibition of acetylcholinesterase	50 mg tid		Rash, diarrhea, giddiness
Mosapride	5-HT4 receptor agonist	5 mg tid, 15 mg qd	Sustained-release mosapride (once daily) is available	
Acotiamide	M1 and M2 mus-carinic receptors antagonist	100 mg tid		Headache, diarrhea

*5-HT, 5-hydroxytryptamine; tid, 3 times a day; qd, once a day.

Antidepressants. Low-dose tricyclic antidepressants are superior to placebo for functional dyspepsia, but they are probably most helpful for those with epigastric pain. Consider amitriptyline 10–25 mg at night increasing to 50 mg if tolerated after 2–4 weeks. Some people may need doses up to 100 mg. These doses may be associated with adverse effects, especially in older patients.

Selective serotonin reuptake inhibitors and selective noradrenaline reuptake inhibitors are reported to be no better than placebo. Mirtazepine may have some efficacy particularly if nausea is associated.

Psychological therapy. Evidence for psychological therapy in functional dyspepsia is limited. However, for patients with the strong psychological component, offering cognitive behavioural therapy is reasonable (fig. 9).

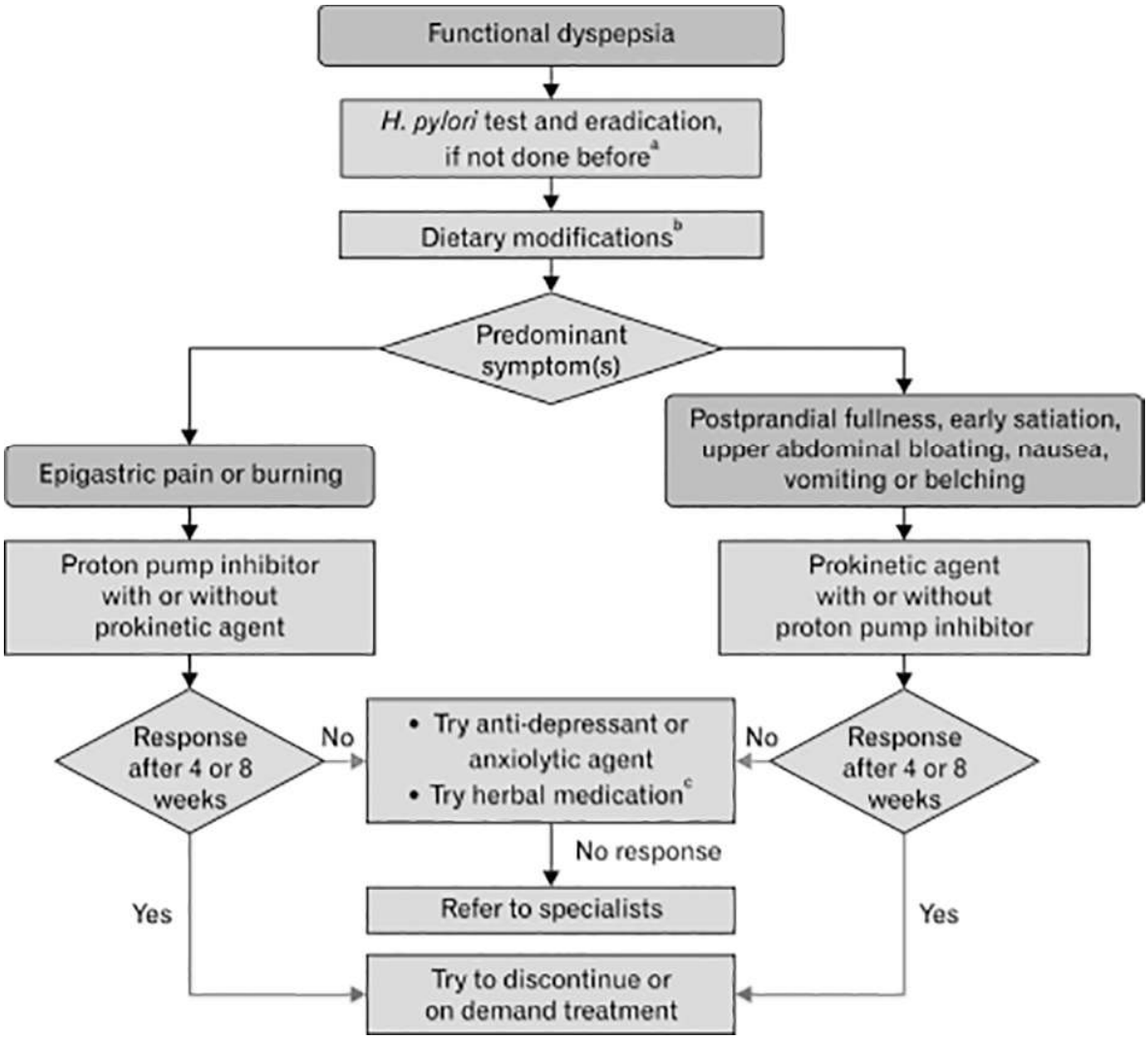

Fig. 9. Functional dyspepsia treatment algorithm

Functional dyspepsia is common and the diagnosis can be made clinically in the red flags absence. Concerning features on history or physical examination should prompt referral to the gastroenterologist for the gastroscopy consideration. Although symptoms can be significantly troublesome or disabling, there is no long-term effect on mortality. Multiple pharmacological and non-pharmacological therapies are available for patients with functional dyspepsia, giving clinicians several options for managing patients with this condition (fig. 10).

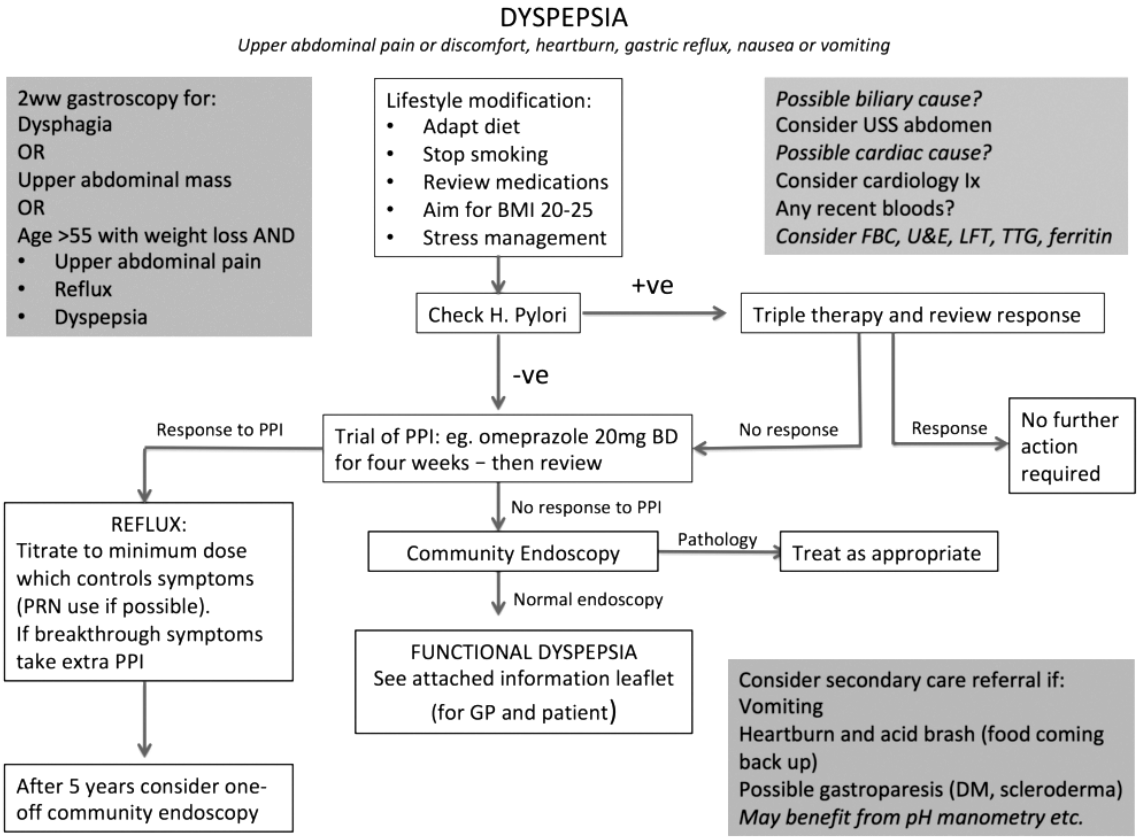

Fig. 10. Action' algorithm for dyspepsia

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