

Gastro-intestinal disorders

1. Which are the most common causes of chronic gastritis?

1. Toxic substances
2. Chronic stress
3. Alimentary factors
4. Endogenous noxious stimuli
5. Genetic factors

2. Chronic atrophic gastritis is a prerequisite for:

1. Ulcer disease and gastric carcinoma
2. Pyloric stenosis.
3. B12 / folic acid / Fe-deficient anemias
4. Perforation of the stomach.
5. 1, 3.
6. 1, 2, 4.

3. Pathogenetic factors for chronic atrophic gastritis are:

1. The genetic inferiority of the gastric mucosa.
2. Autoimmune destruction of the epithelium.
3. Infectious and hypoxic lesions of the gastric mucosa
4. Neural reflex / stress influences.
5. 1, 2, 3.
6. 1, 2, 3, 4.

4. Duodenal reflux primarily leads to:

1. Total gastritis
2. Fundus gastritis
3. Prepyloric and antral gastritis
4. Diffuse mucosal hyperplasia
5. Gastric carcinoma.

5. Which component of the duodenal contents entering the stomach causes the most severe changes to gastric mucosa:

1. Pancreatic enzymes
2. The bicarbonates of the pancreas.
3. Bile salts and acids.
4. Lysolecithin
5. 3, 4.
6. 1, 2, 4.

6. The late development of the vitamin B12 / folic acid deficiency anemia in chronic atrophic gastritis is associated with:

1. Achlorhydria.
2. Preserved synthesis of vit. B12 by the chief cells
3. Persistent protection of vit. B12.
4. Significant hepatic reserve of vit. B12.
5. Slow metabolism of vit. B12 in the body

7. The main pathogenic unit for the formation of ulcer defect is:

1. Impaired gastric motility.
2. Recurrent duodenal refluxes.
3. Mandatory bacterial presence in the gastric juice.
4. Dominant peptic activity of the gastric juice.
5. Existing imbalance in gastrin / hydrochloric acid relationship

8. The most significant pathogenetic unit in duodenal ulcer is:

1. Increased acid/peptic activity of gastric juice.
2. Impaired secretion/evacuation kinetics of the stomach
3. Reduced resistance of duodenal mucosa
4. Delayed intestinal peristalsis.
5. Deficiency of gastro-duodenal hormones

9. Which bacterial colonization proven in the stomach is essential for the emergence of gastritis and peptic ulcer:

1. Staphylococcus aureus.
2. Escherichia Coli.
3. Streptococcus pyogenes
4. Helicobacter pylori.
5. Rickettsia conori

10. What is the central unit in the pathogenesis of ulcer on the body of the stomach:

1. Hyperfunction of the parietal cells of the stomach.
2. Duodenal reflux.
3. Reduced resistance of the gastric mucosa
4. Presence in the mucosa of Staphylococcus aureus
5. Lost neuro-humoral regulation of secretion.

11. Which of the mechanisms participate in the pathogenesis of duodenal ulcer:

1. Increased acid/peptic activity of gastric juice.
2. Impaired secretory-evacuation kinetics of the stomach

3. *Helicobacter pylori*.
4. The reduced resistance of the duodenal mucosa.
5. 1, 2, 3.
6. 1, 2, 3, 4.

12. Main stimuli for increased acid-peptic activity of the gastric juice are:

1. Increased tone of the vagal nerve
2. Hypergastrinaemia.
3. Increased histamine secretion
4. Increased pepsinogen secretion
5. 1, 2, 3.
6. 2, 3, 4.

13. Which factor does not take part in the alkalization of gastric contents:

1. Bicarbonates in the pancreatic juice.
2. Swallowed saliva
3. Acid-stimulated duodenal motility
4. Duodenal and biliary secretion.
5. Diffusion of H^+ ions across the intestinal wall

14. Mucus protects duodenal mucosa by:

1. Its gel-form creating mechanical barrier
2. Alkalizing effect.
3. Presence of glycoproteins A and B
4. Presence of pepsinogen
5. 1, 2, 3.
6. 1, 2, 3, 4.

15. The resistance of duodenal mucosa is primarily due to:

1. Mucus.
2. Glycoproteins A and B.
3. Enzyme inhibitors
4. Prostaglandins of group E.
5. *Helicobacter pylori*.

16. The ulcer of the body of the stomach occurs with:

1. Hyperchlorhydria.
2. Hypo- or normochlorhydria.

3. Low basal secretion
4. Low maximum secretion.
5. 2, 3, 4.
6. 1, 2, 4.

17. Duodenal ulcer occurs with:

1. Hyperchlorhydria.
2. Increased basal and maximal secretion.
3. Hypochlorhydria.
4. 1, 2.
5. 2, 3.

18. The development of acute pancreatitis is a result of:

1. Spontaneous apoptosis of acinar cells
2. Chronic dystrophic process
3. The process of self-digestion of the gland
4. Mandatory bacterial infection
5. Complication due to damage of the endocrine function of the pancreas

19. Important factors for the development of pancreatitis are:

1. Alcohol abuse.
2. Deficiency of antiproteases.
3. Gallstones.
4. Carbohydrate overload
5. 1, 2, 3.
6. 1, 2, 3, 4.

20. The edematous phase of acute pancreatitis is associated with increased blood level of:

1. Pancreatic amylase.
2. Salivary amylase
3. Lipase
4. Carboxypeptidase
5. Elastase

21. Alcohol triggers the pathogenetic chain of events leading to acute pancreatitis by:

1. Stimulation of exogenous secretion of the pancreas.
2. Spasm of the sphincter of Oddi.
3. Direct activation of the pancreatic lipase.

4. Blocking the activity α 1-antitrypsin, and α 2-macroglobulins.
5. 1, 2.
6. 1, 2, 3, 4.

22. Obstructive ileus is usually due to:

1. Complication after abdominal surgery.
2. Severe contusion to the abdominal wall.
3. Spasm of the intestinal musculature.
4. Tumors of the colon.
5. Mesenteric thrombosis.

23. In case of high intestinal obstruction the following signs dominate:

1. Water and electrolyte disturbances.
2. Intoxication.
3. Disorders in fat metabolism
4. Arterial hypertension
5. Chronic constipation.

24. The classification of intestinal obstruction does not include:

1. Mechanical ileus
2. Dynamic ileus
3. Restrictive ileus
4. Paralytic ileus
5. Strangulation ileus