Pathology of gastric adenocarcinoma

Gastric adenocarcinoma is the most common neoplasii (http://lekarske.slovniky.cz/pojem/neoplasie) gastric wall (up to 90% of all gastric cancers). The initial symptoms are shared with chronic gastritis, i.e. dyspepsia, dysphagia and nausea. As a result, similar symptoms are behind the late diagnosis, when the tumor is detected only in advanced stages.

Morphology

Gastric carcinomas are classified according to their **location** in the stomach and according to their characteristic **morphology**. It most commonly affects the antrum. A small curvature is affected more often than a large one. It is formed in the middle part of the mucosa (necks of glands) and spreads to the sides, to the surface or to the depth.

Lauren's morphological classification

- **Diffuse:** When the growth pattern is infiltrative and causes a desmoplastic reaction in the wall that is harder and resembles a leather bottle (odb. **Linitis plastica**). Cells are discohesive (loss of intercellular adhesive and communication links between epithelial cells). The cells contain mucin vacuoles in the cytoplasm, which push the nucleus to the periphery. These cells are called signet-ring cells. It occurs in younger individuals, has no precursor lesions and is more prognostically unfavorable. [1]
- **Intestinal:** Consists of a bulkier mass composed of glandular structures similar to those in adenocarcinoma of the esophagus and intestine. The meat grows exophytically or as a meat with an ulcerative surface. The cells contain apically deposited mucinous vacuoles. It occurs in old age and has precursor lesions (atrophic gastritis, dysplasia, adenoma). [1]

File:Signet Ring Cells (2202231656) .jpg
Diffusion type - annular cells

Histological classification according to WHO

1. adenocarcinoma (95%) - papillary, tubular, mucinous, from cells in the shape of a sealing ring;
2. adenosquamous or squamous cell carcinoma;
3. undifferentiated carcinoma.

- **According to the depth of the invasion, there are types:**

1. **Early cancer** - in the mucosa or submucosa, but does not grow into the external muscularis, it can metastasize.
    1. Small mucosal type - in the mucosa, only shallowly invading the submucosa, diameter less than 4 cm.
    2. Superficially spreading - penetrates into the submucosa in the mucosa or only in foci, diameter over 4 cm.
    3. Pen - penetrating into the submucosa, diameter less than 4 cm, the muscularis interna can completely destroy (type Pen A) or preserves it and penetrates it in the form of fenestrations (Pen B).
2. *'Advanced cancer'* - grows into the lamina muscularis externa or deeper.

TNM classification

- T1 - bounded on the mucosa and submucosa;
- T2 - penetrates through the muscularis propria;
- T3 - penetrates serosis;
- T4 - invasion of surrounding structures;

- N1 - 1–6 regional nodes (perigastric, hepatoduodenal);
- N2 - 7–15 regional nodes;
- N3 - more than 15 regional nodes;

- M1 - presence of distant metastases (irregular nodes - supraclavicular, liver, lungs, bones, brain...).

Macroscopy

- Localization in the pylor and antrum (50%), in the cardia and fornix (25%) and in the body of the stomach (25%).
- The Bormann classification *is used in the evaluation of endoscopic findings:*

1. **polyposis type** - best prognosis, rarest;
2. **ulceriform type** (ca planum, ca patanaeforme) - sharply demarcated;
3. **ulcerative infiltrating type** - blurred edges;
4. **type diffusely infiltrating** (ca diffusum, ca gelatinosum) - the most common.

Spread of gastric cancer

1. **Continuous** - per continuation to surrounding organs (pancreas, liver, bile ducts, oesophagus, duodenum, colon), when the wall is perforated, it enters the peritoneal cavity, where it spreads further by implantation.
2. **Discontinuous**:
    1. lymphogenically into the gastric and mediastinal nodes (up to the deep cervical nodes - Virchow's nodes), into the lungs;
    2. hematogenously to liver, bone (osteolysis - pathological fractures), ovary - Krukenberg's tumor;
    3. implantation after peritoneum (carcinomatosis with hemorrhagic ascites, metastases in oment).

Pathogenesis of carcinogenesis

Mutations

Diffuse Ac:

A germline or acquired somatic mutation for a gene encoding a protein E-cadherin (CDH1) that contributes to intercellular epithelial adhesion (50% of familial diffuse tumors of the stomach).

Intestinal Ac

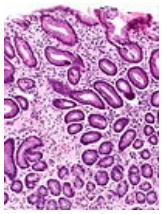
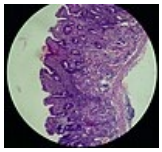
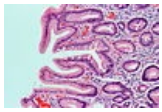
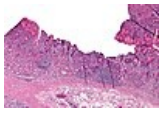
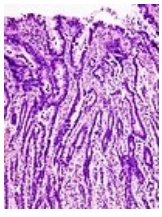
Patients with FAP have an increased risk of developing intestinal gastric adenocarcinoma for many reasons. Mutation of β-catenin, a protein that coordinates the expression of individual adhesive proteins and at the same time the subsequent proper involvement.

Sporadic Ac:

At the same time, in patients with H.pylori infection, mutations in genes encoding Toll-like receptors (TLR4), il-8 and il-10] increase the risk of gastric cancer. , IL-1β and p53. Mutations in these proteins are present in most sporadic gastric adenocarcinomas. These mutations and gene damage occur in chronic gastritis by a hitherto unknown mechanism.

Pathogenesis of the intestinal type

Pelayo Correa showed that the pathogenesis of intestinal-type gastric cancer has relatively distinct stages that can be demonstrated histologically. The development ranges from a histologically completely normal gastric wall through acute inflammation, chronic inflammation, atrophic gastritis, intestinal metaplasia and dysplasia to adenocarcinoma with possible metastatic dissemination. [2]

Acute gastritis	manifested by infiltration of the mucosa and submucosa by lymphocytes, polymorphonuclear foci accompanied by mild mucosal defects and edema can be observed.	
Chronic gastritis	Long-term active inflammation with significant mucosal defects, dilatation of glands and mineralization.	
Atrophic gastritis	Chronic inflammation with focal fibrosis and complete loss of parietal and stem cells.	
Intestinal metaplasia	Gastric epithelial cells elongate and mucins are produced.	
Dysplasia	In the early stages, it can be confused with reactive epithelial changes (acute gastritis, postoperative healing).	
Gastric cancer	Characterized by invasion below the basement membrane.	

Infection

H. pylori

Chronic gastritis, most commonly associated with H. pylori, induces development and progression to the tumor. Stimulated genes increase the expression of proinflammatory proteins (IL-1β, TNF, IL-8 and 10).

> 🔎 *For more information see Role of Helicobacter pylori in the pathogenesis of gastric cancer.*

EBV

Clonal episomes of Epstein-Barr virus (EBV) were found in 10% of adenocarcinomas. This suggests that this percentage of tumours was preceded by EBV infection. Both asymptomatic and infection with an infectious mononucleosis. The mechanism is not yet known, but it is known that there is no TP53 mutation in EBV-associated adenocarcinomas, so apparently the molecular pathogenesis differs from the usual pathogenesis stimulated by H. pylori.

Dietary and socio-economic factors

Dietary factors have not yet been fully explored, however, it is thought that some foods such as cold cuts, abundantly salty delicacies, red meat and generally processed meals increase the risk of gastric adenocarcinoma. All this, mainly due to the contained nitrates and nitrogen compounds, which are used for flavoring, food preservation, can be converted into carcinogens by bacteria (**H. pylori**). A higher incidence was also observed in smokers and obese people. The exact mechanisms are not known, but this is thought to be due to the higher incidence of GERD (Gastroesophageal Reflux Disease) in these groups.

Clinical correlations

The intestinal type of cancer predominates in areas with a higher risk of developing tumors from dysplasia, through adenoma to adenocarcinoma. The median age of the patient is 55 years, male (2: 1 MF-ratio). The above does not apply to the diffusion above. It has a uniform incidence across genders, ages and regions. The prognosis is determined by the depth of the invasion in the wall and the presence of metastases, local invasion. Metastatic spread takes place through the blood and lymphatic routes. Blood most often to the liver, lungs and bones. Lymph especially to the Virchow's node. The local invasion can penetrate the duodenum, pancreas and retroperitoneum, where it can further spread to the ovaries (Krukenberg's tumor), umbilical nodes. Surgical resection is preferred for treatment, if performed and the tumor has not metastasized, then 90% of patients will survive 5 years. In advanced conditions, when resection is no longer possible or the tumor has already metastasized, chemotherapy is used, but even so, only about 20% of patients survive 5 years.

Links

Related Articles

- Stomach tumors
- Stomach cancer
- Stomach

External links

- Stomach cancer - wikipedia

Source

1.

   „ *{{{1}}}“

2. {{#switch: article |book = *Incomplete publication citation.* 1988. pp. 3554-3560. Also available from <https://www.ncbi.nlm.nih.gov/pubmed/3288329>. |collection = *Incomplete citation of contribution in proceedings.* . 1988. pp. 3554-3560. Also available from <https://www.ncbi.nlm.nih.gov/pubmed/3288329>. {{ #if: |978-80-7262-438-6} } |article = *Incomplete article citation.* . A Human Model of Gastric Carcinogenesis. *Cancer Res* [online]. 1988, year 1988, well. 48, pp. 3554-3560, also available from <https://www.ncbi.nlm.nih.gov/pubmed/3288329>. ISSN 1538-7445. |web = *Incomplete site citation.* . ©1988. <https://www.ncbi.nlm.nih.gov/pubmed/3288329>. |cd = *Incomplete carrier citation.* . ©1988. |db = *Incomplete database citation.* ©1988. <https://www.ncbi.nlm.nih.gov/pubmed/3288329>. |corporate_literature = *Incomplete citation of company literature.* . 1988. Also available from <https://www.ncbi.nlm.nih.gov/pubmed/3288329>. legislative_document = *Incomplete citation of legislative document.* 1988. s. 3554-3560. Also available from URL <https://www.ncbi.nlm.nih.gov/pubmed/3288329>.

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