Gastritis (Stomach Inflammation) — Complication and Special Forms

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Increased production of stomach acid or damage of the gastric mucosal barrier may irritate the sensitive gastric mucosa or stomach lining to the degree that gastritis occurs. Gastritis may develop over a longer period of time, but may also occur suddenly and have many causes. As gastritis is one of the most common stomach diseases, this article will provide you with an overview of the various types of gastritis and also address very rare forms of this disease.

Acute Gastritis

Etiology of acute gastritis

Various endogenous and exogenous factors may lead to acute gastritis.
hypertension, for instance, may cause gastritis. Stress, however, be it psychological or physical in nature, is also considered one of the main triggers of acute gastritis. This includes burns, shock, traumas, after surgery or stress in competitive athletes which is commonly referred to as “runner’s stomach”.

Exogenous causes may be chemical/toxic in nature, such as excessive alcohol use, alimentary excesses, severe smoking, as well as pharmacological therapy with nonsteroidal anti-rheumatic drugs (i.e. ASS), corticosteroids and cytostatic drugs. It may also have infectious causes, especially pathogens like Helicobacter pylori, Candida albicans, the cytomegalovirus, herpes viruses or salmonella. In patients undergoing radiation, acute gastritis may develop as comorbidity.

The so-called stress ulcer is a form of acute erosive gastritis. It develops as a result of decreased mucosal blood flow, excessive stomach acid secretion and, at the same time, reduced alkaline protection of the stomach in stressful situations.

**Note:** Stress ulcers are a common occurrence in patients undergoing intensive medical treatment!

Symptoms of acute gastritis

Symptoms include epigastric pain, abdominal fullness, bloating, nausea and even vomiting.
Diagnosis

With regard to the patient’s medical history, the aforementioned symptoms will be mentioned. During the patient’s physical examination, he will experience epigastric pain when pressure is applied to that area. In order to rule out gastroduodenal ulcer disease, it is necessary to perform a **gastroscopy and histology**.

In endoscopic imaging, acute gastritis will present as edematous gastric mucosa and leukocytes infiltrating the mucosa and possibly superficial mucosa defects. In cases of more severe defects meaning erosions, this is referred to as **erosive gastritis**. One of the most feared complications of erosive gastritis is bleeding.

Treating acute gastritis

To eliminate exogenous toxic substances is the most important measure when treating acute gastritis. Fasting and bed rest may be a reasonable approach as well, followed by a light diet – coffee, alcohol and other irritants to the mucosa must be avoided by the patient under any circumstances. Taking proton pump inhibitors until the symptoms subside is an option. Furthermore, antiemetic therapy for the treatment of nausea and spasmolytic medications are available as well. Herbal products made from chamomile, yarrow, licorice root, balm, etc. may also promote healing.

Acute gastritis, without complications, should heal on its own within a few days, which differs from the chronic form of gastritis.

Chronic Gastritis

In cases of chronic gastritis, it is possible that there are no clinical symptoms. Frequently, symptoms such as upper abdominal pain, bloating and halitosis are rather non-specific.

Classifying chronic gastritis according to the ABC classification

**A gastritis = Autoimmune**

This type of chronic gastritis is found in the gastric corpus and cardia and shows the formation of autoantibodies to gastric parietal cells and intrinsic factor:

- Anticanalicular parietal cell antibodies (PCA)
- H+/K+-ATPase antibodies
Intrinsic factor antibodies (IFA)

The consequences are low or no acid output, hypergastrinemia and pernicious anemia. The causes of type A gastritis have not yet been conclusively determined. In a portion of these cases, the infection is thought to have been caused by Helicobacter pylori.

It is possible that autoimmune gastritis is linked to other autoimmune diseases such as Hashimoto's thyroiditis, Morbus Addison or type I diabetes. A portion of Helicobacter pylori-positive type A gastritis may benefit from eradication therapy and be cured that way. Due to the increased risk of carcinoma, patients with type A gastritis should undergo annual gastroscopy screening.

5% of all cases of chronic gastritis have autoimmune causes.

B gastritis = Bacterial by H. pylori

80% of all cases of chronic gastritis are caused by an infection. The most common cause of type B gastritis is the urea-producing bacteria Helicobacter pylori, which colonizes a large part of the population. The bacteria are spread via oral and oral-fecal transmission and cause an infection at the antrum that ascends from there.

This results in gastric atrophy and finally hypochlorhydria. (It does, however, never lead to low or no acid output as can be observed with type A Gastritis!) In more than 90% of cases, so-called triple therapy consisting of two antibiotics and one proton pump inhibitor will achieve complete elimination of the bacteria.

C gastritis = Chemical

15% of all cases of chronic gastritis are chemical/toxic in nature.

NSAR drugs such as diclofenac or ibuprofen and reflux of bile, i.e. following gastrectomy, may cause type C gastritis, predominantly affecting the antrum. Eliminating toxic substances and administering proton pump inhibitors result in the inflammation subsiding. In cases of type C gastritis, there is the possibility, however, that complications such as ulcers and stomach bleeding may occur if the disease is not treated.
Diagnosing chronic gastritis

The diagnosis of chronic gastritis is confirmed by performing gastroscopy screening, which may show changes such as erythematous mucosa and possibly erosions. Here, histological samples are taken from the antrum and the corpus. In order to rule out an infection with Helicobacter pylori bacteria, the Helicobacter urease test (HUT) is performed.

Furthermore, there are non-invasive test methods such as the breath test and stool or serum antigen testing for Helicobacter pylori available as well. Gastroscopy and testing for HP bacteria should be repeated six to eight weeks after therapy with antibiotics has been concluded, in order to ensure therapy success. Type A gastritis is diagnosed by the presence of antibodies to parietal cells and intrinsic factor; in some cases serum vitamin B12 levels may be low as well.

Complications of chronic gastritis

Type A Gastritis:

- Atrophic gastritis
Pernicious anemia

Gastric carcinoma

Type B gastritis caused by Helicobacter pylori:

- Formation of antibodies: PCA and H+/K+-ATPase antibodies
- Developing type A gastritis
- Ventricular and duodenal ulcer (ulcus ventriculi et duodeni)
- Gastric carcinoma and B cell and gastric MALT lymphoma occur more frequently

Very rare complications are:

- Idiopathic thrombozytopenic purpura
- Idiopathic chronic urticaria
- Guillain-Barré syndrome
- Iron deficiency anemia
- Worsening of hepatic encephalopathy
- Gastrointestinal bleeding
- Intestinal metaplasia
- Peptic ulcer

Type C gastritis:

- Ulcers
- Gastrointestinal bleeding

Special Forms of Gastritis

The following lists very rare special forms of chronic gastritis.
Lymphocytic gastritis

Lymphocytic gastritis is an extremely rare disease of unclear genesis. It is mentioned in connection with celiac disease in children, but also in the context of an infection with Helicobacter pylori. In these cases, lymphocytic gastritis may either be healed with HP eradication therapy or an appropriate gluten-free diet.

Lymphocytic gastritis is diagnosed via gastroscopy that may show a wide range of findings, from normal findings to erosions, or giant fold gastritis within the corpus and fundus. The diagnosis of lymphocytic gastritis is histologically confirmed if the intraepithelial lymphocyte count is elevated > 25/100.

The so-called giant fold gastritis or Ménétrier disease (Morbus Ménétrier) is considered to be a complication of lymphocytic gastritis. There is also the possibility of degenerative malignancies which is why annual follow-up endoscopies are recommended. Severe forms of progression with dysplasia may necessitate a gastrectomy.

Giant fold gastritis: Ménétrier disease (Morbus Ménétrier)

Giant fold gastritis is a form of gastritis of unknown cause. Helicobacter pylori is again suspected to be a triggering factor.

The name of this disease is telling: The mucosal folds become enlarged, the chief and parietal cells degenerate resulting in hypoplasia and little or no acid output; this leads to excessive mucus production. In most cases, giant fold gastritis remains without symptoms. Diarrhea and intestinal protein loss with anemia and edema are rare occurrences. It is usually discovered by accident during an endoscopic examination during which the folds remain stable, even with maximum air insufflation. The folds may be removed via gastric resection.

Eosinophilic gastroenteritis

Eosinophilic gastritis is caused by an allergic reaction or parasitic infection. Symptoms include nausea, vomiting, abdominal pain and diarrhea. In rare instances, malabsorption and steatorrhea have been reported.

In a large part of the cases, eosinophilia and elevated serum IgE are present. During the histopathological examination, eosinophilic infiltration, crypt hyperplasia, villous atrophy and ulcerations are noticeable. Dietary measures and the administration of glucocorticoids, possibly supported by low-dose maintenance therapy, will bring the progression of the disease under control.

Gastritis in the context of Crohn’s disease

During the progression of Crohn’s, the stomach may become affected. The causes for this are unknown.

Other Causes of Gastritis

Granulomatous diseases such as tuberculosis and sarcoidosis may cause gastritis as well.
Review Question

The answers are below the references.

1. **Gastritis may lead to stomach ulcers. Which statement is true?**

   A. Only type A gastritis leads to stomach ulcers.
   B. Gastric ulcers are an exclusive complication of type B gastritis.
   C. All forms of gastritis may result in the formation of ulcers.
   D. In the cases of gastritis type A and B, the formation of ulcers is possible.
   E. In the cases of gastritis type B and C, the formation of ulcers is possible.

2. **Which statement regarding gastritis is not true?**

   A. Pernicious anemia is a complication of type A gastritis.
   B. The formation of PCA and H+/K+-ATPase antibodies may be a complication of type B gastritis caused by Helicobacter pylori.
   C. Complications of gastritis caused by NSAR drugs are ulcers and gastrointestinal bleeding.
   D. In 5% of cases, type B gastritis, caused by HP bacteria, develops into autoimmune gastritis.
   E. Giant fold gastritis is a common complication of type A gastritis

3. **The Helicobacter pylori bacterium is the cause of different stomach diseases. Which of the following statements is not true?**

   A. Helicobacter pylori is associated with type B gastritis.
   B. Helicobacter pylori may be the cause of giant fold gastritis.
   C. Type B gastritis, caused by HP bacteria, increases the risk of gastric cancer or MALT lymphoma.
   D. The most common complication of gastritis, caused by Helicobacter pylori, is gastroduodenal ulcer disease.
   E. Gastritis caused by Helicobacter pylori leads to achlorhydria.

References


**Correct answers:** 1E, 2E, 3E

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