

# Gastritis – medical condition essay sample

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Gastritis is a medical condition characterized by the inflammation or erosion of the lining of the stomach. It is characterized by gastric mucosal damage represented by inflammation processes, degenerative metaplasia, allergic processes.

Gastritis comes in two forms and they are:

\* Acute Gastritis

It involves the superficial erosion of the gastric mucosa. With acute gastritis, it is self-limiting. Regeneration of the mucosa occurs within 24 to 72 hours.

\* Chronic Gastritis

With chronic gastritis there is prolonged and repeated irritation of the mucosa. It results in progressive, irreversible atrophy of the gastric mucosa and glands. It occurs in three forms; \* Superficial gastritis: It causes

reddened oedematous mucosa with haemorrhage and small erosion. \* Atrophic gastritis: This occurs in all three layers of the stomach and is

characterized by a decreased number of parietal and chief cells. \* Hypertrophic gastritis: Causes a dull and nodular mucosa with irregular

thickening rugae. There are two main classification; \* TYPE A (Fundal): It results from parietal cell changes leading to atrophy and cellular infiltration. It may be triggered by psycho-emotional stresses. \* TYPE B (Antral): It occurs

in the antrum and is usually due to degenerative changes and colonization of the mucosa by bacteria. Example is the *Helicobacter pylori*.

With chronic gastritis the causes are the same as the acute form. When acute gastritis is not treated, it progresses into the chronic form.

## Etiology

**Acute Gastritis:** This is mostly caused by excessive alcohol consumption and smoking especially on an empty stomach. Potassium and iron supplements, chronic ingestion of irritating food or allergic foods (example mushroom, hot spices, shellfish etc), ingestion of corrosive poison like lead, mercury are also associated with gastritis. Prolonged use of non-steroidal anti-inflammatory drugs (NSAIDs) such as aspirin also causes acute gastritis.

Gastritis may also develop after major surgery, traumatic injury, burns, or severe infections. Gastritis usually occurs in those who have had weight loss surgery resulting in the reconstruction of the digestive tract.

**Chronic Gastritis:** This is caused by infection most especially *Helicobacter pylori*; endotoxins released from infecting bacteria such as streptococci, staphylococci; certain diseases such as Crohn's disease; pernicious anemia; chronic bile reflux; stress; excessive radiation use; chemotherapy and some autoimmune disorders.

Mucous gland metaplasia could also cause this form of Gastritis.

## Pathophysiology

The stomach's rich blood supply, mucous layer, and acidic environment provide a formidable barrier to localized infections, and the digestive substances it secretes normally HCL and pepsin. The pathogenesis of gastritis however, is multifactorial and results from an imbalance of the aggressive gastric luminal factors, acid and pepsin, and defensive mucosal barrier functions of the mucus and bicarbonate. Infections of a type of bacteria called *Helicobacter pylori* colonize the deep layers of gastric mucosa

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and weaken its defense system by reducing the thickness of the mucosal layer and diminishing mucosal blood flow. This results in the development of gastritis in infected individuals.

Mucous gland metaplasia (a reversible replacement of differentiated cells) occurs in the setting of severe damage of the gastric glands, which then waste away (atrophic gastritis). Intestinal metaplasia (complete or incomplete) typically begins in response to chronic mucosal injury in the antrum, and may extend to the body of the stomach. Gastric mucosa cells change to resemble intestinal mucosa and may even assume absorptive characteristics. In complete intestinal metaplasia, gastric mucosa is completely transformed into small-bowel mucosa both histologically and functionally, with the ability to absorb nutrients and secrete peptides. Whereas in incomplete intestinal metaplasia, the epithelium assumes a histologic appearance closer to that of the large intestine and frequently exhibits dysplasia thus reducing the thickness of the stomach lining with increased gastric secretion leading to gastritis.

In some cases, bile, normally used to aid digestion in the small intestine enters through the pyloric valve of the stomach if it has been removed during surgery or does not work properly leading to gastritis.

NSAIDs inhibit cyclooxygenase-1 (an enzyme responsible for the biosynthesis of eicosanoids in the stomach) which increases the possibility of peptic ulcers forming. NSAIDs such as aspirin, naproxen reduce the level of mucosal prostaglandin -a substance that protects the stomach lining. These drugs

when used in a short period of time are not typically dangerous however regular use lead to gastritis by weakening the mucosal barrier.

Alcohol consumption erodes the mucosal lining of the stomach by stimulating hydrochloric acid secretion.

Ingestion of strong acid and alkali such as lye (potassium hydroxide) irritates and corrodes the lining of the stomach.

### Clinical Manifestations

Many people with gastritis may experience no symptoms at all, however epigastric pain is the most common symptom. The pain may be dull, vague, burning, aching, gnawing, sore or sharp and is usually located in the upper central portion of the abdomen but may occur anywhere from the upper left portion of the abdomen around to the back.

Other signs and symptoms include \* Nausea \* Vomiting (if present, may be clear, green or yellow, blood-streaked, or completely bloody, depending on the severity of the stomach inflammation) \* Belching (if present, usually does not relieve the pain much) \* Bloating

\* Feeling full after only a few bites of food

\* Loss of appetite

\* Unexplained weight loss

\* Nausea or recurrent upset stomach

\* Abdominal bloating

\* Indigestion

\* Hiccups and Heartburn

- \* Loss of appetite
- \* Black, tarry stools
- \* Erosion of the mucosa may cause bleeding
- \* Anaemia

## DIAGNOSTIC MEASURES

Diagnosing gastritis and its root cause begins with taking a thorough Personal and Family Medical History, including symptoms, completing a Physical Examination and Laboratory Evaluation.

### \* Physical Examination

\* Vital signs: The single most important aspect of the initial physical examination is determining the patient's hemodynamic stability. Unstable patients should be managed as trauma patients. Placement of a nasogastric (NG) tube is considered the " fifth vital sign" in patients with hemorrhagic gastritis.

\* Focused Physical Examination: After ensuring hemodynamic stability, the initial physical examination should eliminate a nasal or oropharyngeal source of bleeding. Examine the skin and abdomen carefully for clues to an underlying cause. A rectal examination is mandatory. \* Skin examination. Ecchymoses, petechiae, and varices should be noted. Conjunctival pallor is a sign of chronic anemia. Numerous mucosal telangiectasias can point to an underlying vascular abnormality. \* Abdominal examination. Look for stigmata of chronic liver disease (hepatosplenomegaly, spider angiomas, ascites, palmar erythema, caput medusae, gynecomastia, and testicular atrophy).

\* Laboratory Evaluation

\* Basic laboratory studies: This should include a complete blood count with particular attention to the hematocrit, coagulation studies [prothrombin time (PT) and partial thromboplastin time (PTT)], liver function tests (LFTs), serum chemistries (blood urea nitrogen is elevated disproportionately to creatinine in patients with blood loss), electrocardiogram (ECG), and NG aspirate analysis. Acutely, the hematocrit is a poor indicator of blood loss; however, serial hematocrits can be useful in assessing ongoing blood loss. A prolonged PT or PTT suggests an underlying coagulopathy. Elevated LFTs suggest underlying liver disease.

\* Endoscopy plays a central role in the diagnosis and management of hemorrhagic gastritis. Fiberoptic endoscopy is 90% accurate in pinpointing the source of bleeding. In addition, the endoscope can also be used to deliver therapy directly.

\* Angiography can also identify the source of bleeding. It is not as sensitive as nuclear scanning, requiring a blood loss of more than 0.5 ml/minute.

\* Gastric biopsy to confirm the diagnosis and rule out cancer of the stomach.

\* X-ray - using Barium meal or swallow to help locate the area of the stomach affected.

\* Lab analysis of vomitus or stool may detect occult blood if the patient is bleeding.

\* Test for the presence of H. pylori infection in the blood of patient.

\* ECG is important, especially in elderly patients, to search for evidence of cardiac ischemia.

\* Finally, the NG aspirate is essential, if the aspirate is bright red, or “ coffee grounds” in appearance, an upper Gastro Intestinal source is likely. MEDICAL

#### TREATMENT

The treatment of hemorrhagic gastritis will depend on its cause. For most types of gastritis, reduction of stomach acid by medication is often helpful. Beyond that, a specific diagnosis needs to be made. Antibiotics are used for infection. Elimination of aspirin, NSAIDs or alcohol is indicated when one of these is the problem. \* Non-pharmacology therapy: modification of risk factors

\* Avoid smoking; tobacco smoking increases the risk of gastritis, decreases healing rate and increase the frequency of recurrence. \* Avoid Non-steroidal anti-inflammatory drugs (NSAIDs) and alcohol. \* Avoid foods that cause symptoms.

\* Acute general therapy:

\* Eradication of *Helicobacter pylori* when present; this can be accomplished with various regimens: \* Classic therapy: requires the use of Omeprazole Metronidazole, Clarithromycin or Amoxicillin for 7-10 days. \* Dual therapy: requires the use of Omeprazole and a single antibiotic, amoxicillin or Clarithromycin.

\* Patients testing negative for *H pylori* should be treated with antisecretory agents: \* Histamine- 2 receptor antagonists: These block the action of



histamine on the parietal cell by antagonizing H<sub>2</sub> receptors. Examples are Cimetidine, Ranitidine, Famotidine and Nizatidine are all effective; they are usually given in split doses or at night time.

\* Proton pump inhibitors: Esomeprazole, Omeprazole, Lansoprazole, Pantoprazole, or Rabeprazole can also induce rapid healing; they are usually given 30 minutes before meals.

\* Antacids and Chelates are also effective agents for the treatment and relief of pains as a result of gastritis. Antacids help to neutralize stomach acid and can provide fast pain relief while Chelates are designed to help protect the mucosa by stimulating the secretion of bicarbonates, and increasing the synthesis of protective prostagladins.

\* Misoprostol therapy (100µg qid with food, increased to 200µg qid if well tolerated) should be considered for the prevention of NSAID-induced gastritis in all patients receiving long-term NSAID therapy; it however contraindicated in women of childbearing age because of its abortifacient properties; treatment with high-dose H<sub>2</sub> blockers or PPIs also reduces the incidence of gastritis in patients with arthritis who are receiving long-term NSAID therapy.

\* Vitamin B 12 shots for gastritis due to pernicious anemia

#### NURSING CARE MANAGEMENT

\* Psychological Care

\* Reassure patient and relatives that he is in the hands of competent staff and everything will be done to ensure speedy recovery and that the disease can be managed. \* Reassure patient of pain management, that the patient

will be well managed when in pain. \* Assess his perception of the treatment and diagnostic regimen and its outcome, identify his concerns and misconceptions and then provide appropriate solution. The level of the patient's fear may be assessed by observing his behaviors that may give a direct or indirect indication of expression of fears. \* Relieve anxiety by providing information about the disease condition and answering of patient's questions tactfully.

\* Physical Assessment/Observation

\* Continuously observe patient's vital signs (temperature, pulse, respiration, and blood pressure) and record them accurately taking into consideration any abnormality. \* The patient should be weighed to provide a base-line data for future progress or deterioration in client's condition. \* Monitor intravenous infusions and maintain intake and output chart if patient is on intravenous infusions. If patient is on infusion, observe intravenous infusion for patency, flow rate, dislodgement. Also observe infusion site for signs of infection. \* Observe patient's vomitus and stool for occult blood. \* Observe patient for signs of dehydration (example dry and cracked skin and lips) and palor. \* Rest and Sleep

\* The nurse should ensure rest and sleep by providing a comfortable bed free from creases, and ensure good ventilation. \* Maintain a quiet environment and regulate the number of visitors to preserve the patient from unduly disturbances. \* Serve patient with warm beverages

\* Advice patient to undertake relaxation therapy.

### \* Personal Hygiene

Assist patient to ensure personal hygiene is maintained where necessary as in the following:

- \* Bathing and grooming

- \* Oral care

- \* Care of hands and feet

- \* Change patient soiled linen or clothes

- \* Hand washing especially before and after eating, using the offered bed pans, urinal or visiting the sluice room.

### \* Diet

- \* Patient is put on Nil per OS to help rest the stomach for 6 to 12 hours.

When symptoms subside ice chips followed by clear liquid are offered.

Frequent small bland meals should also be introduced as soon as possible.

Afterwards patient can be given well balanced diet. \* Meal should be taken at regular intervals

- \* Acidic, spicy or fried foods should be avoided.

### \* Medication

- \* Nurse should ensure that the Patient's drugs were served whilst taking into consideration the 7Rs: the right patient, the right time, the right date, the right drug, the right dosage, the right of patient to refuse medication, and the right route of administration. \* The effects of the drug should be observed after administration and observation dully recorded.

### \* Education

\* Educate patient on the disease condition, possible causes, complications, importance of treatment and prevention of disease. \* Educate patient on the need for follow up and continuity of care. \* Patient should be advised to steer clear of indulging in habits or ingesting substances that could trigger the disease. \* Educate patient to stick to medication therapy and report peculiar symptoms early.

### PREVENTION

\* Hand washing with soap and running water especially before meals and eating well cooked meals could aid in protection from infection. \* Avoiding caffeinated beverages, smoking and alcohol. \* Engaging in stress management techniques.

\* Educate patient to undertake deep breathing exercises to aid relaxation. \* There is the need to engage in exercise to improve circulation \* Avoiding the abuse of NSAIDs drugs

### COMPLICATIONS

\* Severe forms of hemorrhagic gastritis can lead to peritonitis. \* If left untreated for a long time can also lead to perforation of the stomach walls. \* There may be shock since there is bleeding.

\* There may also be gastric cancer in severe, chronic and untreated forms of hemorrhagic gastritis. \* There may be anaemia as a result of the bleeding.

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