



PHARMACY CONTINUING EDUCATION FROM WF PROFESSIONAL ASSOCIATES

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"Hiatal Hernia/GERD: Review & Update"

March 2018



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In this lesson we review GERD & hiatal hernia. Specifically, we concentrate upon antacids, and OTC H₂RAs and PPIs. Our goals are: (1) consideration of therapeutic options, (2) comparisons of when to use H₂ blockers versus PPIs, and finally (3) provide updated information that can improve communications with patients.

This lesson is intended for pharmacists & technicians in all practice settings. **The program ID # for this lesson is 707-000-18-003-H01-P for pharmacists, and 707-000-18-003-H01-T for technicians.**

Participants completing this lesson by February 28, 2021 may receive full credit. Release date for this lesson is March 7, 2018.

To obtain continuing education credit for this lesson, you must answer the questions on the quiz (70% correct required), and return the quiz. Should you score less than 70%, you will be asked to repeat the quiz. Computerized records are maintained for each participant.

If you have any comments, suggestions or questions, contact us at the above address, or call 1-847-945-8050. **Please write your name, NABP eProfile (CPE Monitor®) ID Number & birthdate (MM/DD) in the indicated space on the quiz page.**

The objectives of this lesson are such that upon completion participants will be able to:

For Pharmacists:

1. Define hernia & GERD & describe the relationship between the 2 disorders.
2. List the contributing factors for GERD, esophagitis, Barrett's esophagus, esophageal stricture & esophageal adenocarcinoma.
3. Discuss the symptoms of GERD & its complications.
4. Differentiate between the mechanisms of actions between H₂ blockers & PPIs.
5. Compare the therapeutic effects of H₂ blockers & PPIs. Discuss the symptoms of GERD & its complications.

6. Discuss the diagnosis of GERD.

For Technicians:

1. Define hernia & GERD & describe the relationship between the 2 disorders.
2. Discuss the symptoms associated with GERD.
3. Compare the therapeutic effects of H₂ blockers & PPIs.
4. Discuss the diagnosis of GERD.

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INTRODUCTION

The esophagus is a fibromuscular, hollow tube covered on the inside by a mucous membrane and ranges from 8-10 inches long in adults. It connects the pharynx (throat) with the stomach, lies behind the trachea and heart, and passes through the diaphragm. It ends at the upper most region of the stomach. When swallowing, the **upper esophageal sphincter (UES)**, which consists of a muscular ring located below the junction of the pharynx and esophagus, opens to allow the passage of food into the esophagus. The downward movement of the food is facilitated by muscular contraction. Another sphincter called the **lower esophageal sphincter (LES)** is located above the junction of the stomach and lower portion of the esophagus. When the esophagus is at rest, both sphincters contract to prevent the backflow of acidic gastric fluid from the stomach to the esophagus and mouth. During the swallowing process, both sphincters open to allow passage of food to the stomach.

The esophagus is exposed to many disorders, some of which include gastroesophageal reflux, hiatal hernia, esophagitis, esophageal stricture, Barrett's Syndrome and esophageal adenocarcinoma.

GASTROESOPHAGEAL REFLUX DISEASE (GERD)

Gastroesophageal reflux disease (GERD) is a common chronic digestive disorder that affects the LES. When dilated, or if it fails to close tightly, gastric contents are allowed to flow backward (refluxate) through the LES and into the esophagus. The gastric fluid contains hydrochloric acid along with digestive enzymes. The presence of these causes irritation and inflammation of the esophageal lining (mucosa). The frequency of this reflux ranges from occasional to a daily occurrence. This often is called heartburn or acid indigestion. Normally when food is swallowed, the UES will open allowing the food to pass into the esophagus and into the stomach through the LES. Once the food reaches the stomach, the LES will close tightly to prevent the gastric contents from regurgitating (reflux) backward into the esophagus. The severity of GERD depends on the condition of the LES, type and acidity of food taken, and the neutralizing effect of the saliva.

Gerd Contributing Factors

LES Malfunction

Relaxation, stretching or weakening of the LES will contribute to its failure to tightly close. This can allow gastric content to flow backward into the esophagus.

Hiatal Hernia

"Hiatal" is derived from "hiatus", which anatomically means a gap or a hole in a body organ. Hernia is a condition that occurs when an internal organ of a body cavity bulges out as a result of pushing through a weakened area in the muscle or tissue that holds it in place. Hernias occur most commonly in the abdomen, but can develop in the upper thigh, umbilicus (navel, belly button) and groin area. The most common types are inguinal, hiatal, umbilicus and incisional hernias. Inguinal hernia is the most common type and occurs when the intestine forces itself through a weak area or through a tear in the lower abdomen or groin area. The patient may experience mild pain or discomfort, especially when coughing, sneezing, or exercising. It appears as a bulging in the affected area.

Hiatal hernia is a condition that involves the upper part of the stomach. As the esophagus connects the mouth to the stomach, it passes through the chest cavity via an opening known as the esophageal hiatus. Hiatal hernia is a condition that occurs when part of the upper stomach (normally located in the abdominal cavity) pushes up into the chest through the esophageal hiatus and bulges within the chest cavity. When the upper part of the stomach is squeezed upward, part of the gastric contents will pass into the esophagus causing GERD. Usually a small hiatal hernia causes no symptoms, and the patient may be unaware of its presence. Large hiatal hernias can cause pain and allow reflux. Surgery may be needed if medications fail to provide relief. As a group, hernias have potential of cutting off blood supplies to the affected organ as the opening (hiatus) may apply pressure on the organ to become strangulated. In this example, a medical or surgical emergency can occur.

Increased intraabdominal pressure

Pregnancy frequently precipitates heartburn. It has been estimated that 50% to 80% of pregnant women experience GERD, especially after the fourth month. It is believed that the symptoms occur as a result of pressure on the abdomen caused by the fetus. Others have theorized that hormones play a role in the symptoms.

Intake of large and frequent meals, in particular before bedtime and while lying down, can trigger regurgitation of gastric contents. Likewise, wearing tight clothes around the waist can trigger a similar effect.

Alcohol

Beverages containing alcohol act as a gastric stimulant, and can result in an increase in acid secretion. Excessive consumption of alcohol may cause heartburn and contribute to damage to the gastric mucosa. Carbonated beverages, orange juice and other citrus base mixers may also contribute to GERD.

Consumption of certain foods

Spicy or highly acidic foods such as pickles, pepper, lemon, tomato base foods, peppermint, garlic, onion and other spices, may stimulate gastric secretions and/or increase gastric acidity. Caffeinated beverages may stimulate gastric secretion. Meals rich in fat can delay gastric emptying, resulting in food remaining longer in the stomach and more production of acid leading to GERD.

Cigarette smoking

Tobacco products can aggravate GERD due to the nicotine contents, that act as a gastric stimulant and a relaxant to the LES.

Certain prescription and non-prescription drugs

A number of medicinals can contribute to GERD. Drugs like NSAIDs (i.e. aspirin, celecoxib, ibuprofen, indomethacin and naproxen), bone strengthening drugs (such as bisphosphonate, alendronate, ibandronate, and risedronate), iron supplements (such as ferrous sulfate), theophylline, tetracycline, certain benzodiazepines, tricyclic antidepressants, narcotic opioids (such as codeine, hycodan and morphine) all have potential to cause problems. Additionally, antihypertensives, (such as calcium channel blockers---nifedipine, verapamil, or diltiazem), beta blockers (like nadolol), as well as progesterone, quinidine, and Cox-2 inhibitors, can trigger GERD. These drugs act by:

- Weakening or causing relaxation of the LES.
- Triggering or exacerbating already existing GERD.
- Delaying gastric emptying rate.

Stress and depression

Anxiety, stress and other psychological issues increase the vulnerability to GERD.

SYMPTOMS OF GERD

The most common symptom of GERD is the **presence of a burning sensation** sometimes accompanied by pain in the back of the throat and chest. It most often occurs after eating, especially large, fatty meals. The symptoms may worsen when lying down. A transient pain may radiate to the left arm, shoulder and neck. Such symptoms may disrupt sleep. The patient may experience "water-brash," which is the flow of a sour fluid into the mouth due to hypersalivation. Bending over during exercise or any other activity, especially after a meal, may cause flow of gastric fluid into the esophagus. Aspiration refluxate into the airways and bronchioles may trigger coughing and asthma-like episodes due to chemical irritation. It has been reported that GERD is responsible for 50% of cough in non-smokers.

TREATMENT OF GERD

Treatment begins by diagnosis of the cause by a healthcare professional. The main goals that should be attempted to be achieved in the treatment of GERD are to: provide symptomatic relief, enhance healing of damaged mucosa, prevent complications or recurrence of GERD. It is essential to reduce contact between the inflamed mucosa and the acidic refluxate. This may be accomplished by increasing the pH of the gastric fluid to or above 4, reduce production of gastric fluid or modify diet and lifestyle. Using antacids may achieve a better pH. In addition, the use of Histamine-2 receptor antagonists (H2RAs) and Proton Pump Inhibitors (PPIs) are effective in reduction of gastric acid production. Lifestyle changes and diet, along with the use of medications, is to be implemented for long-term treatment.

Antacids

Antacids provide quick, but short-term, relief from occasional heartburn or indigestion. Even though nonprescription antacids are indicated in treating heartburn, manufacturers of these drugs do not list their use for GERD on the label. They are often the first drugs to be used by self-treating patients for treating GERD and heartburn, however. One or more of the following chemicals may be included in antacids: calcium carbonate, magnesium oxide, aluminum hydroxide, and sodium bicarbonate. Alginic acid may be included as a component of antacid products. Alginic acid and alginates create a foamy gel barrier against acidic gastric fluid. Brown algae is a source of alginic acid. That being said, antacids alone are not considered as a treatment of choice for heartburn and GERD.

H2-Receptor Antagonists or blockers (H2RAs)

This group of medications relieve GERD by binding to H2 receptors of parietal cells found in the gastric mucosa and results in reduction of gastric acid production and the risk of damage to the esophageal lining. It appears that such medications have no effect on LES tone. H2RAs usually provide relief within two hours. They are available as nonprescription drugs. This class includes cimetidine, ranitidine, famotidine, and nizatidine. Common side effects include: headache, drowsiness, constipation, nausea and vomiting.

Proton Pump Inhibitors (PPIs)

Proton pump inhibitors are a potent group of drugs that are used to relieve GERD by prolonged reduction of gastric acid secretion. PPIs are considered more potent than H₂RAs. Their mechanism of action is due to irreversible inhibition of the H⁺/K⁺ ATPase in the parietal cells. These epithelial cells secrete HCl and intrinsic factor. They are located in the glands found in the gastric mucosa and possess the ability to secrete HCl by active transport into the stomach. The enzyme hydrogen potassium ATPase is the proton pump of the stomach whose function is to acidify the gastric content and activate the enzyme pepsin. In addition to their use for treating GERD, PPIs can treat gastric and duodenal ulcers as well as other gastric disorders such as stress gastritis, esophagitis and Barrett's syndrome. Some PPIs are available without prescription. This group of medications are considered top sellers in the U.S. Examples include esomeprazole, omeprazole, dexlansoprazole, lansoprazole, rabeprazole, and pantoprazole. Side effects include: diarrhea, nausea, abdominal discomfort, fatigue, flatulence, dizziness and constipation.

Lifestyle and Diet Modification

Control over lifestyle and diet modification are achievable and may result in reduction in gastric acid production. Such measures should accompany the intake of medications. Many patients attempt to self-treat themselves by using nonprescription drugs that, if used properly, may provide short-term relief. Improper use of nonprescription drugs may worsen GERD symptoms or may mask an underlying condition. For example, improper use of sodium bicarbonate may result in acid rebound which increases gastric activity. An important factor that should be recognized is identifying and avoiding the offending foods and harmful everyday habits.

There is no universal GERD diet that works for all sufferers. The following are recommendations:

- 1% or 2% low-fat milk;
- low-fat or fat free yogurt;
- all vegetables except tomatoes;
- fruits such as apples, berries, melons, bananas, pears, but not citrus such as oranges, grapefruits as well as pineapple;
- grains;
- oatmeal;
- whole grain bread;
- potatoes;
- low fat meat such as chicken and turkey without skin;
- decaffeinated beverages and noncarbonated ones;
- most juices except citrus, tomato, and pineapple;
- low-fat soups;
- unsaturated fats from plants like olive oil, sesame, canola, peanuts, peanut butter and nut seeds;
- polyunsaturated fat found soybean, corn, flaxseed and walnut; and,
- fatty fish such as salmon.

Lifestyle modifications should be carefully implemented. Eating small, frequent portions reduces the risk of gastric distention, and avoids weight gain. Wearing loose clothing, sitting up while eating and avoiding reclining after meals for a minimum of two hours is advisable. Since some GERD sufferers may experience nocturnal reflux, the last meal should be a few hours before retiring. Exercises that require bending, especially following eating, should be carefully avoided. Smoking as well as avoiding excessive consumption of alcoholic beverages should be implemented.

ESOPHAGITIS

Esophagitis is an acute or chronic inflammation of the esophagus that may cause damage to the esophageal mucosa due to GERD. Continued irritation of the mucous membrane of the esophagus and exposure to reflux that contains acid and the enzyme pepsin results in a gradual destruction of the epithelial layer and the appearance of micro-ulcers. Deeper ulceration may cause damage to submucosal and muscular tissue and the development of fibrosis leading to esophageal stricture. When these symptoms occur, the condition is termed erosive esophagitis.

Symptoms of esophagitis include abdominal pain, painful swallowing (odynophagia), adherence of food to the esophageal lining, anorexia, nausea, cough, difficulty swallowing (dysphagia), and mouth ulcers. Other than GERD, esophagitis can occur as a result of chronic consumption of alcoholic beverages. Esophagitis can be diagnosed through physical examination, symptomatically, or utilizing diagnostic tests including endoscopy, barium x-ray, and biopsy of esophageal tissue to rule out the presence of bacteria, fungi, virus, allergy or cancer.

Types of Esophagitis

1. Reflux esophagitis that is caused by refluxate from the stomach.
2. Infectious esophagitis that is caused by bacteria, fungi or viruses.
3. Eosinophilic esophagitis which is inflammation due to an increase in the number of the white blood cells, eosinophils, within the lining of the esophagus. The blood cells play an important role in the immune system which is responsible for protecting the body from invasion by microorganisms. Eosinophils, along with mast cells and basophils, are associated in the development of asthma and allergic reactions. This could explain why GERD and esophagitis could trigger asthma-like cough.

Treatment of esophagitis should be based on the causative factors. If it is due to GERD, then a treatment similar to that used for GERD should be utilized. Antibiotics may help if infectious esophagitis is diagnosed. Short-term use of corticosteroids can be effective in reducing inflammation.

ESOPHAGEAL STRICTURE

Esophageal stricture is a complication of GERD and esophagitis. It involves narrowing of the esophagus lumen. Long-term exposure to acidic gastric refluxate results in swelling and damage to the esophageal lining. The healing process can cause formation of scar tissue (fibrosis) which can make the lining of the esophagus tighten and thicken, leading to narrowing of the lumen. Stricture can also form as a result of healing after esophageal surgery and laser therapy. Stricture usually develops at the lower end of the esophagus, but it may extend over the years to the middle or higher portions. The risk factors for esophageal stricture include: chronic GERD, malignancy, birth defects, medications that trigger GERD and infections.

Symptoms

These may include dysphagia, food sticking to the lumen, which is more severe than that encountered in esophagitis, burning pain in the chest, bitter acidic taste in the mouth, weight loss which may be a result of not eating enough to avoid dysphagia, burping and choking more than normal. Esophageal stricture occurs in 7-23% of untreated GERD patients. These account for 70-80% of all cases of esophageal strictures, and are 2-3 times more common in men than women. Patients are usually older and have been experiencing reflux symptoms for a long time. Diagnosis of strictures is similar to that of GERD and esophagitis.

Treatment

Options can be a surgical procedure such as stretching or dilation of the esophageal lumen to normalize the lumen size. An endoscope is inserted via the mouth to reach the esophagus where a small balloon is inflated to stretch the esophagus. Another procedure involves using a stent to widen the narrowed esophageal lumen.

BARRETT'S ESOPHAGUS (BE)

Also called Barrett's Syndrome, this disorder is another complication of GERD. It develops when the esophagus tries to repair itself of damage and inflammation, ulceration, or fibrosis caused by reflux. The repair process involves conversion of the epithelial cells of the esophagus to resemble the cells that make up the intestine. The majority of short term and uncomplicated GERD cases usually do not lead to BE or cell transformation. BE can occur after years of exposure to reflux. About 10% of patients who suffer from chronic GERD develop BE. Individuals who experience acute or occasional GERD do not develop BE (which affects men twice as frequently as women). The age at which BE develops is typically 55 and older.

Diagnosis of BE is similar to that described for esophagitis. However, the biopsy tissue must be examined for the presence of precancerous or cancerous cells. Once confirmation is achieved, it is advisable to have periodic endoscopic tests as well as biopsy.

The main goal of treating BE is to arrest or hinder the process of the cell transformation process. This goal may be achieved by controlling GERD. The treatment for GERD should be followed. Additionally, there are certain treatments that aim at destroying abnormal tissue. These involve:

1. Radio frequency ablation (RFA), which employs radio waves emanated from an endoscope inserted into the esophagus.
2. Photodynamic therapy (PDT) using a laser through an endoscope that destroys abnormal cells.
3. Endoscopic spray (cryotherapy) kills abnormal cells by freezing them with cold nitrogen or carbon dioxide liquid.
4. Endoscopic mucosal resection (EMR) focuses on removal of the abnormal lining.
5. Surgical removal of part or most of the esophagus may be performed in cases of the presence of precancerous or cancerous cells.

There are no specific symptoms related to BE as GERD is the source of the symptoms. As such, BE patients may experience frequent chest pain, difficulty in swallowing, vomiting of blood, and passing tarry stool.

Risk factors include:

1. Chronic and persistent heartburn.
2. Even though BE may occur at any age, older individuals are more prone to the disease.
3. BE is more common among men than women.
4. Whites are more vulnerable to BE than others.
5. Overweight patients are more likely to develop the disease.
6. Smokers are more susceptible than non-smokers.

ESOPHAGEAL ADENOCARCINOMA

This term is derived from "adeno" meaning gland, and "carcinoma," meaning malignant cancer. Thus, esophageal adenocarcinoma means a malignant cancer arising from the esophageal glandular cells present in the lower third of the esophagus and may have transformed to intestinal cells. It originates from the epithelial tissue and is capable of metastasizing. It can affect any part of the body including the esophagus. Metastasis occurs via the lymphatics or blood stream. As indicated earlier, BE can lead to this type of cancer. Occurrence is higher in patients having BE. About 1% of these individuals develop esophageal adenocarcinoma. As late as 2012, esophageal cancer was considered the eighth leading cancer worldwide with 450,000 cases that year. The disease resulted in 400,000 deaths in 2012 compared to 345,000 in 1990. The incidences vary from one country to another. Half of all cases are in China.

Prognosis of the disease is usually poor due to late diagnosis. The tumor results in narrowing of the esophagus and in dysphagia. Weight loss and anorexia occur in order to avoid pain. Other symptoms include: pain around the breastbone, coughing, and hoarseness due to irritation of laryngeal nerves by the tumor. The primary cause is the chronic erosive effect of the acidic digestive refluxate that may trigger transformation of normal esophageal cells to intestinal cells and erosion of the squamous cells.

POTENTIAL DRUG – DRUG INTERACTIONS

Antacids

These are capable of delaying absorption of acidic drugs and result in reduced blood concentration and therapeutic effects. Such drugs include: digoxin, phenytoin and chlorpromazine. On the other hand, absorption of other medications such as pseudoephedrine and levodopa may be increased causing higher blood concentration and emergence of side effects. Magnesium containing antacids may reduce absorption and therapeutic effects of drugs due to their binding capacity with the antacid. Due to its fast and strong reduction of gastric acidity, sodium bicarbonate may ultimately affect the acidity of urine. This applies to basic drugs such as quinidine and amphetamine, and they enhance excretion of acidic drugs such as aspirin. The intake of aluminum hydroxide with raltegravir, dolutegravir and citrates, which may be found in soft drinks, may increase aluminum level in the blood due to an increase in GI absorption of aluminum.

Foods containing calcium, such as milk and dairy products, can cause milk-alkali syndrome when taken with antacids. This occurs as a result of the presence of calcium and absorbable alkali. Continuation of this practice can lead to formation of kidney stones, kidney damage, weight loss, vomiting, constipation and fatigue.

The activity of antacids can be prolonged when taken with food. The intake of citrus juices and products along with aluminum containing antacids can increase absorption of aluminum.

Histamine H2 Receptor Antagonists

Drugs such as cimetidine, famotidine, nizatidine and ranitidine are capable of interacting with a number of medications. Cimetidine can bind to cytochrome P450 (CYP) and as a result interfere with body mechanism for drug metabolism and elimination by the liver. As such, it inhibits the enzymes CYP1A2, CYP2C9, CYP2C19, CYP2D6, CYP2E1, and CYP3A4. Cimetidine may reduce the metabolic pathway of drugs, thereby increasing their blood concentration causing potential for toxicity. Drugs that interact with cimetidine include warfarin, theophylline, phenytoin, lidocaine, quinidine, propranolol, labetalol, methadone, tricyclic antidepressants, metronidazole, certain calcium channel blockers, and alcoholic beverages. Not all H2 Receptor blockers share the same effect as cimetidine. Ranitidine has the same effects, but with lesser intensity. Famotidine has a weak activity for inhibiting CYP and thus the interaction is negligible.

Proton Pump Inhibitors (PPIs)

Drugs such as esomeprazole, lansoprazole, omeprazole, pantoprazole, rebeprazole and dexlansoprazole may cause the following interactions:

- Decreased absorption when given with antacids
- Increased anticholinergic activity
- Antidepressant tricyclics cause increased antidyskinetic (an agent that relieves or prevents dyskinesia which is defined as impairment of voluntary movement) effect.
- Increased effect of carbidopa
- May increase the sedative effect of CNS depressants
- Decreased effect of chlorpromazine
- Decreased effect of dopamine antagonists such as pramipexole, and ropinirole
- Increased blood level of the HIV drugs nelfinavir and atazanavir
- Reduction of the activity of the enzyme that converts clopidogrel to its active form
- Increased blood level of methotrexate

USE OF H2 BLOCKERS VERSUS PPIs

Both groups reduce production of gastric acid, but each has different mechanisms of action. PPIs turn off the proton pump, which is a molecule found in the parietal cells of the gastric glands that cause pumping of hydrochloric acid and intrinsic factor into the stomach. Acid production occurs by an ion exchange. The pump attracts non-acidic potassium ion from the stomach and exchanges it with an acidic hydrogen ion that contributes to the activity of the gastric fluid. PPIs inhibit this exchange activity by the pump, resulting in blocking the acidic hydrogen ion production and subsequent reduction in gastric acidity.

H2 receptor blockers act on the parietal cells in the gastric mucosa which are responsible for acid production. The surface of the parietal cells contains H2 receptors. Stimulation of these receptors results in increased hydrochloric acid production. Histamine is a gastric acid stimulant. Thus, H2 receptor blockers act by binding to those H2 receptors found on the parietal cells surface and subsequently blockade of acidic production.

PPIs have slower onset of action than H2 blockers, but have longer duration of action.

SUMMARY

GERD is a common disorder. It has been estimated that 1/3 of the population in the U.S. has GERD - related symptoms. GERD may be asymptomatic but is usually characterized by a burning sensation behind the breast bone that radiates to the neck and throat and regurgitation of sour tasting fluid in the mouth. These symptoms occur from long exposure of the esophageal lining to acidic gastric reflux. Esophagitis, Barrett's esophagus, esophageal stricture and esophageal adenocarcinoma may occur as a complication of GERD. There are a number of factors that contribute to GERD. Offending food and drinks should be avoided and lifestyle should be modified. In addition, drugs such as antacids, H2 blockers and Proton Pump Inhibitors are effective.

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Please fill out this section as a means of evaluating this lesson. The information will aid us in improving future efforts. Either circle the appropriate evaluation answer, or rate the item from 1 to 7 (1 is the lowest rating; 7 is the highest).

1. Does the program meet the learning objectives?

Define hiatal hernia & GERD	YES NO
List contributing factors for esophageal disorders	YES NO
Discuss symptoms of GERD	YES NO
Differentiate MOAs of PPIs & H2 blockers	YES NO
Compare therapeutic effects of PPIs & H2 blockers	YES NO
Discuss diagnosis of GERD	YES NO

2. Was the program independent & non-commercial? YES NO

3. Relevance of topic

	Low Relevance		Very Relevant
	1 2 3 4 5 6 7		

4. What did you like most about this lesson? _____

5. What did you like least about this lesson? _____

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Please Mark the Correct Answer(s)

- | | |
|---|--|
| <ol style="list-style-type: none"> 1. During swallowing, the UES opens to allow passage of food through the esophagus.
A. True
B. False 2. Which of these is NOT considered a contributing factor associated with GERD?
A. LES malfunction.
B. Smoking.
C. Diet rich in acid.
D. Eating slowly 3. "Hiatus" is a term to describe:
A. Absence of HCl in the stomach.
B. Opening or hole in an organ.
C. Backward moving of food from esophagus.
D. Intestines bulge thru abdominal wall. 4. When part of the upper stomach pushes thru to the chest cavity, the condition is known as:
A. Inguinal hernia.
B. Umbilicus hernia.
C. Hiatal hernia.
D. Incisional hernia. 5. Water-brash is a symptom of GERD describing:
A. Flux of gastric fluid to the esophagus.
B. Flow of sour fluid into the mouth.
C. Damage to esophageal lining.
D. Accumulation of sour fluid in stomach. | <ol style="list-style-type: none"> 6. Which of the following drugs does NOT contribute to GERD?
A. Indomethacin.
B. Vitamin B1.
C. Bisphosphonates.
D. Celecoxib. 7. H2 blockers are considered more potent than PPIs.
A. True
B. False 8. Barrett's esophagus is characterized by:
A. Narrowing of the esophageal lumen.
B. Bleeding.
C. Gastric ulceration.
D. Conversion of the epithelial cells of the esophagus to intestine-like cells. 9. The concurrent intake of PPIs & helminx may cause:
A. Increased blood level of the antiviral drug.
B. Increase in metabolism of the PPI.
C. Decrease renal elimination of the viral drug.
D. Increased activity of the gastric fluid. 10. Dysphagia means:
A. Difficulty in swallowing.
B. Food sticking to esophageal lining.
C. Difficulty in breathing.
D. Loss of appetite. |
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