ZEBRAS OF MEDICINE

OESOPHAGEAL DYSPHAGIA: HOW TO DIFFERENTIATE BETWEEN ACHALASIA AND GASTRO-OESOPHAGEAL REFLUX DISEASE

Jamie Hulzebos[1]

[1]Master Student Medicine, Radboud university medical center, Nijmegen, the Netherlands

Abstract

Review

BACKGROUND: Gastric acid reflux is a normal physiologic process. It involves into gastro-oesophageal reflux disease (GERD) when the patient has unpleasant symptoms or mucosal damage of the oesophagus. Achalasia is a rare motor disorder of the distal oesophagus and lower oesophageal sphincter (LES). Achalasia is often not recognised by clinicians or confused with peptic strictures, a complication of GERD.
OBJECTIVE: This article aims to provide insight into the different forms of dysphagia, in particular, to differentiate between achalasia and GERD.
RESULTS: Achalasia and GERD are both causes of oesophageal dysphagia. Patients suffering from GERD as well as patients suffering from achalasia, show symptoms of dysphagia, heartburn and regurgitation. However, patients with GERD experience dysphagia only for solid foods. Patients with achalasia experience progressive dysphagia for solids and liquids. GERD can best be diagnosed by clinical symptoms, though an endoscopy is helpful if peptic strictures are suspected. The best diagnostic tools for achalasia are a barium swallow test and oesophageal manometry.
CONCLUSION: Differentiation between achalasia and GERD can be done by anamnesis and diagnostic testing. Patients with GERD typically complain about postprandial and nocturnal regurgitation. Patients with achalasia complain about regurgitation of undigested food, and dysphagia for solids and liquids. Diagnostic testing includes endoscopy, barium swallow tests and oesophageal manometry.

KEYWORDS: oesophageal dysphagia, GERD, achalasia

Introduction

Dysphagia is the medical term for difficulty in swallowing. It can be classified into oropharyngeal and oesophageal dysphagia. Patients with oropharyngeal dysphagia have difficulty initiating a swallow, and this could lead to regurgitation and aspiration. Patients with oesophageal dysphagia have the unpleasant sensation of food and or liquids being obstructed between mouth and stomach resulting in pain at the suprasternal notch or behind the sternum [1]. Gastro-oesophageal reflux disease (GERD) is common in the Western population. It causes heartburn and regurgitation. Heartburn is a burning sensation in the retrosternal area. Achalasia is a rare disorder in which there is a loss of normal peristalsis in the distal oesophagus and disfunction of the lower oesophageal sphincter (LES). The LES cannot relax properly, causing food particles to remain in the oesophagus. Often achalasia is initially not recognised by healthcare professionals, as the symptoms can be similar to other disorders of the digestive tract, such as gastro-oesophageal reflux disease (GERD) [2]. Achalasia and GERD are both forms of oesophageal dysphagia. Differentiation between achalasia and GERD might be a clinical challenge [1,3]. The aim of this article is to give insight into the different forms of dysphagia, in particular, to differentiate between achalasia and GERD.

Oropharyngeal and oesophageal dysphagia

To differentiate between achalasia and GERD, we must first differentiate between oropharyngeal and oesophageal dysphagia. Taking an accurate clinical history is key in differentiating between both types of dysphagia. Oropharyngeal dysphagia is also called transfer dysphagia and arises from the oral cavity, pharynx, upper oesophagus or upper oesophageal sphincter (UES). Patients with oropharyngeal dysphagia complain about repetitive swallowing, nasal regurgitation, coughing, nasal speech, drooling, choking, halitosis and/or recurrent pneumonias [1]. Oropharyngeal dysphagia is often present in neurological patients. Therefore, it is helpful to include neurological symptoms and family history in your anamnesis.

Patients with oesophageal dysphagia have different symptoms. They experience discomfort a few seconds after initiating a swallow, and often locate their pain distal to the suprasternal notch. This type of dysphagia can be caused by both liquids and solids. Patients may have a history of heartburn, scleroderma, congenital oesophageal webs and rings or radiation therapy [1]. Once again, an accurate clinical history can give direction to oropharyngeal or oesophageal dysphagia, but diagnostic tests are needed most of the time. Different causes of oropharyngeal and oesophageal dysphagia can be found in Table 1 and 2 of the appendix on *www.ramsresearch.nl*.

Gastro-oesophageal reflux disease and peptic strictures

Gastric acid reflux into the oesophagus is a normal physiologic process. Gastro-oesophageal reflux becomes GERD when it causes unpleasant symptoms or mucosal injury to the oesophagus [4]. GERD is common in the overall population with a prevalence of 10-20% in western countries, and 5% in Asia [5]. GERD is frequently diagnosed in adults from Western countries, whereby smoking and obesity seem to increase the risk for developing GERD [6]. Heartburn and regurgitation are characteristic symptoms of GERD. Complications include reflux oesophagitis, ulceration, peptic strictures, Barrett's oesophagus and adenocarcinoma of the oesophagus [4]. Peptic strictures are a result of healed erosive oesophagitis lesions. Collagen is deposited during this healing process, and the collagen fibers may contract and narrow the oesophageal lumen. These peptic strictures lead to progressive dysphagia for solid food only. Peptic strictures occur in up to 10% of patients with GERD, but this number decreases with proton-pump inhibitor (PPI) use [1]. GERD can be diagnosed based on the symptoms described above. To diagnose peptic strictures, an endoscopy is needed and a histological biopsy can be

performed if a malignancy is suspected [1]. Treatment of benign peptic strictures comprises of dilation by a mechanical or balloon dilator. Short-term outcomes are good, longer-term outcomes are best when a luminal diameter greater than 12mm is achieved by dilation [7]. Patients with peptic strictures should be treated with a PPI, to prevent more damage to the oesophagus. Apart from surgical and pharmacotherapeutic management, lifestyle and dietary modification should be recommended [8]. Weight loss is recommended for overweight patients and the use of tobacco and alcohol should be diminished. Selective elimination of dietary triggers, such as fatty or spicy foods, can be useful in some patients. Elevation of the head during the night is useful in patients with nocturnal symptoms [6]. These lifestyle modifications are recommended for all patients with GERD. An overview is given in Table 1.

Achalasia

Achalasia is a primary oesophageal motor disorder caused by a lack of myenteric neurons that coordinate oesophageal peristalsis and LES relaxation. However, it remains unclear what causes this lack. It is a rare disorder, with a prevalence of 11 per 100.000 adults. Incidence increases with age, with a mean age of 53 years at diagnosis [9]. Some patients have symptoms for years before achalasia is confirmed. Symptoms are dysphagia, regurgitation of undigested food, respiratory symptoms such as a nocturnal cough, aspiration and pneumonia, chest pain and weight loss [2,3]. Dysphagia after meals and heartburn can lead to misdiagnosis as GERD. However, achalasia leads to progressive dysphagia for both solids and liquids. Moreover, regurgitation caused by achalasia is unresponsive to adequate use of PPI, as the problem is located in the oesophagus instead of the stomach [3]. Achalasia can be treated with pneumatic dilation or laparoscopic surgical myotomy [10]. These are preferred options for initial treatment. Botulinum toxin therapy is recommended only for patients who cannot or do not want to undergo surgery [11]. Oral pharmacologic therapy with calcium channel blockers and long-acting nitrates is an option when other treatment options fail. Short-term efficacy is excellent, but the effectiveness of mentioned treatments decreases with time. The risk of oesophageal squamous cell carcinoma and adenocarcinoma is higher in patients with achalasia, although recent guidelines do not recommend routine endoscopic screening for these patients [2]. An overview is given in Table 1.

Diagnostic tests

GERD can be diagnosed by clinical symptoms only. However, an endoscopy is helpful to exclude peptic strictures or malignancies. A barium swallow test can be done when a motility problem is suspected [12,13]. During a barium swallow test a film is made, while the patient swallows a liquid containing barium sulfate. Barium sulfate lights up on X-ray, so anatomical or motility abnormalities can be seen clearly (Figure 1). A barium swallow test can be used to support, but not confirm, the diagnosis of achalasia [14].

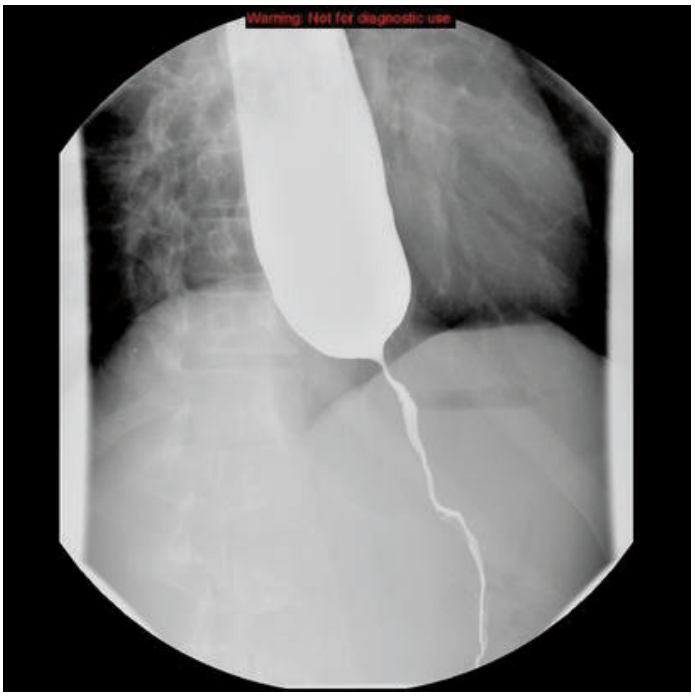

Figure 1: *X-ray of a patient with achalasia during a barium swallow test. A wide, atone oesophagus and a thin gastro-oesophageal junction (Bird's beak) is seen. Image from https://pediatricimaging.wikispaces.com/Achalasia, reuse under CC BY-NC-ND 3.0.*

The best test for confirmation of achalasia is an oesophageal manometry [15]. During manometry, a thin pressure-sensitive tube is inserted in the nose of the patient through the oesophagus and into the stomach. The patient is asked to swallow repeatedly a small fixed amount of water. This test evaluates the pressure in the oesophagus. Findings of aperistalsis and incomplete LES relaxation without a mechanical obstruction present, confirm the diagnosis of achalasia [2]. However, even manometry does not have a sensitivity of 100% [15].

Conclusion

Differentiation between achalasia and GERD can be done by anamnesis and diagnostic testing. Both groups of patients complain about heartburn and regurgitation. Patients with GERD typically complain about postprandial and nocturnal regurgitation. Patients with achalasia complain about regurgitation of undigested food, and dysphagia for solids and liquids. Endoscopy is helpful if GERD with peptic strictures is suspected. A barium swallow test and oesophageal manometry can be used to

Table 1: *Overview of gastro-oesophageal reflux disease versus achalasia.*

	GERD	Achalasia
Pathofysiology	Gastric acid reflux into the stomach, sometimes complicated by peptic strictures	Primary oesophageal motor disorder, lack of myenteric neurons
Prevalence	10-20% in the Western population, 5% in Asia	0,01% of adults in the world population
Dysphagia	For solid food when peptic strictures are present	Progressive for solids and liquids
Other clinical symptoms	Heartburn, postprandial and nocturnal regurgitation	Heartburn, regurgitation of undigested food, nocturnal cough, aspiration
Diagnostic tests	Diagnosis based on clinical symptoms Endoscopy if peptic strictures are suspected	Barium swallow test Oesophageal manometry

confirm the diagnosis of achalasia. On top of that, symptoms of GERD will diminish with the use of a PPI, while symptoms of achalasia do not.

Acknowledgements

I would like to thank dr. T.M. Bisseling, Radboud university medical center, from the Departement of Gastroenterology, Hepatology and Intestinal failure, Nijmegen, the Netherlands for proofreading this article.

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CORRECT ANSWERS TO THE EXAM QUESTIONS

Answer question 1:
C. Within hours.

A patient with an acute asthma attack is treated with inhalers to deliver medication straight into the lungs. This medication is aimed at providing immediate relief by reducing inflammation and/or opening up the airways. In addition to this, prednisolone is administered intravenously. Prednisolone is a corticosteroid drug that can reduce the inflammatory response for a longer period of time. It does so by acting on the glucocorticoid receptor. This is a nuclear receptor, which means that prednisolone alters gene transcription. So, even though prednisolone has a lipophilic structure that allows for easy and rapid passage through the cell membrane, it takes a while for the effects of prednisolone to occur.

During the exam, 35% of the participants answered this question correctly.

Answer question 2:
D. Secretin.

Secretin secretion from the duodenum can inhibit the release of gastrin from the pyloric antrum. Gastrin is a peptide hormone that stimulates the secretion of gastric acid by the parietal cells of the stomach and is able to increase antral muscle mobility.
Acetylcholine is a neurotransmitter in the autonomic nervous system and has a muscle-activating function. Bombesin stimulates gastrin release and thereby stimulates the secretion of gastric acid. Histamine is involved in local immune responses but is also located in the enterochromaffin-like cells within the gastric glands. Histamine release is halted when the pH of the stomach starts to decrease.

During the exam, 55% of the participants answered this question correctly.

The exam questions can be found back on page 13 in this journal.