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Causes of upper gastrointestinal tract obstruction

Przyczyny niedrożności górnego odcinka przewodu pokarmowego

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Abstract

Obstruction of the upper gastrointestinal tract, caused by blocked passage in the oesophagus, stomach or duodenum, is an important clinical and diagnostic problem in gastroenterological practice. The typical symptoms are dysphagia, postprandial vomiting, epigastric pain and weight loss. Post-inflammatory oesophageal lesions associated with reflux oesophagitis are the most common cause of obstruction. Other common causes include foreign bodies, neoplasms, chemical burns of the oesophagus and radiation-induced stenosis. In more than 2/3 cases, foreign bodies are localised in the proximal part of the oesophagus, but anatomical abnormalities, such as a Schatzki ring or post-inflammatory stenosis, increase the risk of food bolus impaction in the distal part of the oesophagus. Radiotherapy of head and neck tumours may cause stenosis, which affects more than 7% of patients treated this way. For the stomach and duodenum, 50–80% of obstruction cases are associated with neoplastic processes, with gastric cancer and pancreatic adenocarcinoma accounting for 35% and 15–25% of these cases, respectively. Mild causes of peripyloric obstruction include gastric and duodenal peptic ulcer, peritoneal adhesions, gastric polyps and Crohn's disease. Symptoms of temporary pylorus obstruction can be caused by large, gastric hyperplastic pedunculated polyps. Therapeutic endoscopy is the most commonly used method for upper gastrointestinal tract obstruction. Depending on the cause, it involves foreign body removal, balloon enteroscopy, stenting with self-expanding metallic stents, and, in the case of treatment failure, surgical resection or palliative gastrojejunostomy.

Keywords: upper gastrointestinal tract, oesophageal obstruction, gastric outlet obstruction, duodenal obstruction

Streszczenie

Niedrożność górnego odcinka przewodu pokarmowego spowodowana zaburzeniem pasażu w obrębie przełyku, żołądka lub dwunastnicy stanowi istotny problem kliniczny i diagnostyczny w praktyce gastroenterologicznej. Typowymi objawami są dysfagia, nudności, wymioty poposiłkowe, ból nadbrzusza i utrata masy ciała. Najczęstszą przyczyną niedrożności są zmiany pozapalne przełyku w przebiegu zapalenia refluksowego. Innymi częstymi przyczynami są ciała obce, nowotwory, chemiczne oparzenia przełyku oraz zwężenia indukowane radioterapią. Ciała obce w ponad 2/3 przypadków lokalizują się w proksymalnym odcinku przełyku, ale anatomiczne nieprawidłowości w postaci pierścienia Schatzkiego lub zwężeń pozapalnych zwiększają ryzyko utknięcia kęsa pokarmowego w odcinku dystalnym. Radioterapia guzów głowy i szyi może skutkować wystąpieniem zwężeń, obecnych u ponad 7% pacjentów poddanych tej formie leczenia. W obrębie żołądka i dwunastnicy 50–80% przypadków niedrożności związanych jest z procesem nowotworowym, przy czym 35% tych przypadków stanowi rak żołądka, a 15–25% gruczolakorak trzustki. Do łagodnych przyczyn niedrożności okołoodźwiernikowej należą: wrzód trawienny żołądka i dwunastnicy, zrosty otrzewnowe, polipy żołądka i choroba Leśniowskiego–Crohna. Duże uszypułowane polipy hiperplastyczne żołądka mogą powodować objawy przemijającej niedrożności odźwiernika. Najczęściej stosowaną metodą leczenia niedrożności górnego odcinka przewodu pokarmowego jest leczenie endoskopowe, w zależności od przyczyny polegające na usunięciu ciała obcego, poszerzaniu balonowym, protezowaniu z wykorzystaniem samorozprężalnych metalowych stentów, a w razie jego nieskuteczności wykonuje się resekcyjne zabiegi operacyjne lub paliatywną gastrojejunostomię.

Słowa kluczowe: górny odcinek przewodu pokarmowego, niedrożność przełyku, niedrożność żołądka, niedrożność dwunastnicy

INTRODUCTION

Typical symptoms of upper gastrointestinal (GI) tract obstruction depend on the cause and location of the blockage. They include epigastric pain, nausea, postprandial vomiting, early satiety, weight loss, malnutrition, dysphagia and chest pain[1]. Dysphagia and upper GI tract obstruction are an indication for urgent esophagogastroduodenoscopy (EGD), which, in most cases, also allows for a therapeutic intervention. Most often, upper GI obstruction is caused by mild strictures, for example secondary to reflux oesophagitis or peptic ulcer disease of the stomach and duodenum, and cancer[2–4]. Due to the wide range of possible causes and non-specific symptoms, upper GI obstruction is an important problem and a gastrointestinal diagnostic challenge. This paper summarises possible, often unknown, causes of upper gastrointestinal stricture and obstruction.

OESOPHAGUS

Oesophageal obstruction is caused by critical narrowing and total blockage of the oesophagus[2]. In most cases, the obstruction develops slowly, leading to increasingly severe dysphagia, which initially causes difficulty swallowing solid foods, though it can occur suddenly after a large food bolus impaction[5]. The symptoms of oesophageal obstruction include dysphagia, odynophagia, heartburn, vomiting, epigastric pain, cough and pharyngeal pain in the case of proximal oesophageal obstruction[1,5–9]. Mild and malignant causes of oesophageal strictures are distinguished. The vast majority occur in chronic reflux disease. Other causes are squamous cell carcinoma and adenocarcinoma, foreign body impaction, oesophageal achalasia, food in the Zenker's diverticulum, burns with caustic substances, radiation-induced stricture or a Schatzki ring[1,2,5–9].

Reflux oesophagitis

Reflux disease is responsible for 70–90% of mild oesophageal strictures, mostly located in its distal part[2,10,11]. Such narrowing occurs as a result of inflammation and fibrotic processes during prolonged exposure to gastric acid. In recent years, these rates have decreased due to the common use of proton pump inhibitors. Macroscopically, strictures in reflux disease appear smooth and concentric, and they are usually 1–4 cm long. They occur in 7–23% of patients with reflux oesophagitis. Strictures secondary to reflux oesophagitis are treated with endoscopic pneumatic balloon dilation. Mechanic Savary–Gilliard dilators in combination with high-dose proton pump inhibitors are also used. After endoscopic therapy, patients should continue treatment with drugs that suppress gastric acid secretion to prevent relapse[10,11].

Oesophageal cancer

A total of 1,400 new cases of oesophageal cancer are reported every year in Poland. Men, usually older than 40 years of age, are mostly affected (80%). Precancerous states of squamous cell carcinoma include caustic substance burns or achalasia, while Barrett's oesophagus is the main risk factor of adenocarcinoma. Dysphagia of increasing severity occurs in 75% of advanced cancer cases. Obstruction in oesophageal cancer is a late symptom, usually occurring in the advanced stage of the disease. Polypoid forms account for around 60% of squamous cell carcinomas. Surgical procedure is a preferred radical treatment method; however, in palliative therapy of dysphagia and oesophageal obstruction, prosthetics of the narrowed segment using self-expandable metallic stents (SEMS) plays the most important role[12,13].

Foreign bodies

Oesophageal foreign bodies are mostly found in patients with psychiatric disorders or older adults with dementia[1,12]. They are, after gastrointestinal bleeding and biliary diseases, the third most common indication for therapeutic endoscopic surgery[1]. In more than 2/3 cases, obstruction occurs in the proximal part of the oesophagus; endoscopic treatment is necessary in 10–20% of cases[1]. Aiolfi et al. found in their meta-analysis that about 43% of cases are caused by sharp-edged objects[1]. About 3.4% of patients needed surgery due to oesophageal perforation, fistula or unsuccessful endoscopic treatment. The presence of a sharp-edged foreign body in the oesophagus for more than 24 hours is an independent risk factor for complications in patients aged ≥60 years[12]. The most common symptoms include retrosternal pain, dysphagia, odynophagia and pharyngeal pain[1,12]. Gastroscopy with removal of foreign body is the primary treatment method. It is often performed using a plastic protection tube (overtube). Delayed endoscopic treatment is associated with higher rates of complications. Another type of foreign bodies that cause temporary oesophageal obstruction are pieces of food (mostly meat such as beef or poultry), which account for more than 1/3 of foreign bodies found in the oesophagus[1]. In the vast majority of cases, no endoscopic treatment is needed[1,5]. In patients with a Schatzki ring or stricture due to reflux oesophagitis, obstruction occurs mostly in the distal part of the oesophagus. Longstreth et al. found anatomical abnormalities in 88% of patients with oesophageal food impaction[5].

Oesophageal achalasia

Oesophageal achalasia is a motor oesophageal disease of unknown aetiology, in which impaired relaxation of the lower oesophageal sphincter (LES) occurs as a result of damage and reduction in the number of postganglionic neurons of

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the myenteric plexus. The lack of primary peristalsis wave in oesophagus is also found in this disease. Oesophageal achalasia most commonly occurs in people between 30 and 60 years of age. It manifests with growing dysphagia, which initially causes difficulty swallowing solid foods, chronic cough and choking. Untreated achalasia increases the risk of oesophageal squamous-cell carcinoma. Contrast-enhanced oesophageal radiography, endoscopy and oesophageal manometry are used in the diagnosis. Treatment includes, among others, pharmacotherapy, i.e. medications to reduce LES pressure, for example nifedipine or isosorbide dinitrate; however, it is not very effective in most cases. The wide range of invasive treatment approaches involves endoscopic oesophageal dilatation, peroral endoscopic myotomy (POEM) and surgical cardiomyotomy[8,9].

Zenker's diverticulum

Pharyngeal pouch (Zenker's diverticulum) develops as a result of cricopharyngeus muscle dysfunction (CMD) and appears as a bulging of the weakened posterior pharyngeal wall and the oesophagus in the area of the upper oesophageal sphincter. Zenker's diverticulum is most frequently found in people 70–80 years of age. Small diverticula are usually asymptomatic. However, with an increasing size of the diverticulum, there is more accumulation of food residues. These residues exert extrinsic compression on the oesophagus, causing dysphagia. Zenker's diverticulum may lead not only lead to oesophageal obstruction, but also perforation or recurrent aspiration pneumonia. Symptomatic Zenker's diverticulum requires endoscopic treatment (diverticulomy or septotomy)[13,14].

Chemical burns of the oesophagus

Oesophageal stricture is one of the most common complications of chemical burn of the oesophagus[15,16]. Children who ingested a caustic substance, such as chemical detergent used to clean the kitchen and the bathroom, account for 80% of patients with oesophageal burns[16]. Oesophageal stricture is the consequence of second and third degree burns. About 70% of patients with second-degree burns and over 90% of patients with third-degree burns develop stricture, most often at 8 weeks after the incident; however, the inflammatory fibrotic processes start at about 3 weeks. Therefore, early diagnostic endoscopy is of key importance[15]. It is recommended to perform gastroscopy in the first 12–48 hours after chemical injury, though it can be safely postponed up to 96 hours. Endoscopy is repeated after 2–3 weeks to detect possible oesophageal narrowing. The treatment involves repeated dilatation of the oesophagus with mechanical or pneumatic dilators, which is shown to be effective in about 1/3 of patients. In the case of failure, it is necessary to perform gastrostomy or partial resection of the oesophagus with reconstruction, with the use of a fragment of the large intestine. After chemical burns of the oesophagus, the risk of developing squamous cell carcinoma increases significantly, which after about 40 years is approximately 1,000 times higher than in the general population. Therefore, endoscopic surveillance is necessary.

In about 5% of cases of oesophageal stricture due to chemical burns in children pyloric obstruction also occurs. This mostly affects the prepyloric area due to prolonged mucosal contact with caustic substances, caused by pyloric contraction[17].

Radiotherapy-induced oesophageal stenosis

Radiotherapy is a commonly used adjuvant treatment method for oesophageal, head, neck, breast or lung cancer. Oesophageal stenosis, with more than half of cases among patients after radiation of head and neck, is one of important late complications of radiotherapy[18]. It occurs usually 3–8 months after irradiation (median of 6 months), usually in the proximal part of the oesophagus[19]. Wang et al. estimated in their meta-analysis that the general risk of oesophageal narrowing during radiotherapy of head and neck carcinoma is 7.2%, while the risk of narrowing with classical radiotherapy and intensity-modulated radiation therapy is 5.7% and 16.7%, respectively[20]. The risk of stricture increases with radiation dosage, reaching 15% in patients treated with 60 Gy[19]. Treatment options for radiotherapy-induced stricture include surgical resection or endoscopic balloon dilation, which is increasingly used, although relapses occur and further procedures are needed in 40% of patients. In a study conducted by Park et al. more than 3 dilation procedures were needed to achieve long-term clinical outcome. The ultimate success rate of endoscopic dilatation is over 85%. If this method is ineffective, an oesophageal prosthesis is used or, if this is not possible, a gastrostomy is carried out[19].

Schatzki ring

Schatzki ring is a concentric, symmetrical, inflexible oesophageal narrowing at the level of the transition zone from stratified epithelium to glandular epithelium (Z line). The ring is usually 2–3 mm thick. The main symptom is dysphagia, usually causing difficulty swallowing solid foods. Schatzki ring is found in 10–15% of patients undergoing gastroscopy for dysphagia. Dysphagia occurs when oesophageal narrowing of 13 mm or less in diameter is present. Full obstruction of the oesophagus is possible also as a result of food bolus impaction. Symptomatic Schatzki ring requires treatment, which involves endoscopic dilatation with Maloney or Savary–Gilliard dilators[21].

STOMACH AND DUODENUM

Gastric outlet obstruction (GOO) is a syndrome characterised by epigastric pain, postprandial vomiting, early satiety

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and weight loss. These symptoms are caused by mechanic or functional obstruction of the distal part of the stomach, pyloric canal or duodenum. The incidence of GOO is not precisely known. From 50% to 80% of all GOO cases are associated with pyloric malignancy. Mild causes include peptic ulcer, polyps, abdominal tuberculosis, Crohn's disease, bezoars, annular pancreas and pancreatitis[3].

Gastric and pancreatic cancer

Gastric cancer accounts for about 35% of GOO cases, while pancreatic adenocarcinoma invading the duodenum or the stomach is responsible for 15–25% of cases[3]. In a prospective research by Meinke et al., 30% of patients with adenocarcinoma of the head of pancreas who were under palliative prosthetic treatment of the biliary tract developed pyloric obstruction[22]. Less common causes of obstruction include carcinomas of the duodenal bulb, gastric lymphoma or metastatic tumours[3].

Treatment approaches for pyloric obstruction include radical surgery and, in unresectable cases, surgical gastrojejunostomy and endoscopic insertion of protheses[23]. In a prospective study by Fiori et al., endoscopic stenting with metallic self-expanding stents contributed to earlier resumption of oral food intake and shorter hospital stay compared with surgical treatment; however, patients treated with prosthetics were more likely to develop a relapse of obstruction. There were no differences between the two methods in the incidence of late relapse of obstruction or in patient survival[24].

Adenocarcinoma of the ampulla of Vater

Duodenal obstruction may develop secondary to a neoplastic process in the course of adenocarcinoma of the greater duodenal papilla (ampulla of Vater). Adenocarcinoma of the ampulla of Vater mainly affects people over 60 years of age. It may invade the digestive tract or spread along the bile duct. It is usually diagnosed at a relatively early stage due to cholestatic jaundice. Diagnosis at an early, non-advanced stage makes duodenal obstruction a rare finding in this type of cancer. Early forms of adenocarcinoma of the ampulla of Vater may be treated endoscopically (ampullectomy). Surgical treatment includes duodenotomy with tumour removal and, in advanced cases, Whipple pancreatoduodenectomy. Palliative treatment consists of endoscopic stenting of duodenal and bile duct stenosis[25].

Peptic ulcer disease of the stomach and duodenum

In addition to bleeding and perforation, obstruction may also be a complication of peptic ulcer disease of the stomach and duodenum[3]. Of the mild causes of mechanic gastric obstruction, ulcers account for approximately 90% of cases[4]. Due to the common use of proton pump inhibitors, the rates of this disorder are decreasing. Typical symptoms are nausea, vomiting and epigastric pain. The treatment of obstruction as a complication of peptic ulcer disease, in addition to pharmacotherapy including eradication of *H. pylori* infection and the use of proton pump inhibitors, consists of endoscopic balloon dilatation of the stenotic site or surgical treatment (rarely used nowadays)[3]. Relapses are less common after successful eradication of *H. pylori*[26]. Clinical improvement after endoscopic balloon dilation is estimated at about 70%[27]. In the absence of *H. pylori* infection, the rate of endoscopic failures is higher. Therefore, surgical vagotomy may be considered in patients with pyloric obstruction due to peptic ulcer disease without *H. pylori* infection and poorly responsive to balloon dilatation[26].

Duodenal adhesions

More than 90% of adhesions are the consequence of surgical procedures, mainly laparotomy, less often laparoscopy. Adhesions may be also caused by previous inflammations and be a radiotherapy complication[28]. They account for 65% of mechanic obstructions in the small intestine[28]. Hyvärinen et al., in their retrospective research, observed the presence of adhesions in the duodenal area in almost 25% of patients after conventional cholecystectomy[29]. After this procedure, advanced adhesions connecting fragments of the distal loops of the small intestine to the duodenum, forming a tight knot in the site after gallbladder resection, were found in 9% of cases and adhesions causing gastrointestinal symptoms in 34% of patients[30]. Cholecystectomy affects the frequency of duodenal adhesions. Polymeneas et al. showed that adhesions after laparoscopic cholecystectomy occurred much less often than in case of standard method, affecting 20% and 100% of patients, respectively[31].

Gastric polyps

Gastric polyps are a rare cause of pyloric obstruction. The vast majority of polyps are silent; however, they may, in certain cases, cause gastrointestinal bleeding, or, even less often, GI obstruction[32]. The symptoms of upper GI obstruction may be transient because of its partial mobility. Strangulated polyps of the prepyloric area may lead to transient pyloric canal obstruction[33]. Large hyperplastic polyps may cause symptoms of obstruction, the severity of which increases over time[34].

Crohn's disease

Crohn's disease (CD) can affect any part of the GI tract, although lesions are less frequent in the upper part. CD limited to the stomach and duodenum is found in 0.5–4% of patients, but most patients have concomitant lesions in the distal ileum[35]. Crohn's disease isolated to the stomach usually causes non-specific symptoms, such as nausea, epigastric pain, vomiting and weight loss. Pyloric obstruction or

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duodenal stricture are very unlikely to be the first symptoms of the disease[36]. Proton pump inhibitors, glucocorticoids, biological therapy and endoscopic balloon dilation are used in the treatment[35].

Abdominal tuberculosis

Abdominal tuberculosis accounts for 1–3% of all cases of this disease and 5–17% cases of non-pulmonary tuberculosis[37]. Rao et al. showed that gastric and duodenal lesions are found in 0.5% of cases, while isolated gastric tuberculosis is even less common[38]. Such tuberculosis symptoms include vomiting, epigastric pain, loss of appetite, upper GI bleeding and, often, fever[37]. Pyloric obstruction is one of the most common complications of abdominal tuberculosis, occurring in 61% of patients with this organ location in a study by Rao et al.[38]. Obstruction can be secondary to duodenal or antral involvement, or may be related to compression by enlarged lymph nodes. Treatment involves the use of antimicrobial drugs and, if no improvement is noted, gastrojejunostomy with vagotomy is used[38].

Pancreatitis

Chronic and, less commonly, acute pancreatitis can cause changes in the duodenum and lead to duodenal obstruction, which develops in 0.8–4% of cases. Obstruction is caused by inflammation of the anterior surface of the pancreas, which results in fibrosis of the adjacent tissues, duodenum, jejunum and transverse colon. Patients with duodenal obstruction in chronic pancreatitis often present with coexisting biliary strictures. Duodenal obstruction may also be caused by pancreatic necrosis and large pseudopancreatic cysts[39].

Groove pancreatitis

Groove pancreatitis is a type of chronic pancreatitis located near the duodenum, the head of pancreas and the common bile duct. Potential causes include smoking, long-term alcohol intake and a history of gastrectomy. Groove pancreatitis is characterised by non-specific symptoms, such as epigastric pain, postprandial nausea, vomiting and weight loss. Due to the similarity of clinical and radiological symptoms to those of carcinoma of the pancreatic head, the diagnosis of groove pancreatitis remains a significant clinical challenge. Exceptionally, groove pancreatitis can cause duodenal stenosis and obstruction[40]. Treatment includes prevention, surgical or endoscopic procedures. More than a half of cases of groove pancreatitis are treated surgically, with pancreatoduodenectomy remaining the most common treatment method[41].

Annular pancreas

Annular pancreas is a rare congenital cause of pyloric obstruction, characterised by the presence of a band of tissue around the descending part of the duodenum. It is silent in most cases. Symptoms concern 1/3 of patients and usually occur in people from 30 to 60 years of age. Sandrasegaran et al. showed that 40% of patients had symptoms of pyloric obstruction, such as epigastric pain, postprandial fullness, vomiting, and severe or chronic pancreatitis. Annular pancreas can also cause bile duct obstruction. An incomplete tissue ring around the duodenum is also a risk factor for pyloric obstruction[42]. If obstruction symptoms occur, surgical treatment is advised. It involves gastrojejunostomy or duodenojejunostomy. Resection of the annular pancreatic tissue is less likely to lead to symptom resolution[43].

Bezoars

Gastric bezoars are masses of foreign bodies caused by accumulation of undigested food residues. They are usually found accidentally, during less than 0.5% of gastroscopy procedures[44]. Phytobezoars, created from undigested vegetable matter, are the most common form. However, bezoars can also form from ingested hair or medicines. They are most often found in patients with impaired gastric motility due to resection surgeries or pyloroplasty with vagotomy[45]. In most cases, they are asymptomatic, occasionally causing epigastric pain, nausea, vomiting, early satiety and weight loss. Sometimes they may cause pyloric obstruction. In most cases, bezoars can be removed with endoscopic methods[46].

Conflict of interest

The authors do not declare any financial or personal relationships with other persons or organisations that could adversely affect the content of the publication and they claim rights to this publication.

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