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Drug-induced oesophageal injury: case reports and literature review

Polekowe uszkodzenia przełyku – opis przypadków i przegląd literatury

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Abstract

Drug-induced oesophageal injury (pill-induced oesophagitis) is a relatively rare but clinically significant complication of pharmacotherapy. The pathogenesis of oesophageal mucosal injury is associated with direct and prolonged contact of medication with the mucosal surface, leading to local irritation, ulceration, and necrosis. The most commonly implicated agents include antibiotics (particularly tetracyclines), non-steroidal anti-inflammatory drugs, bisphosphonates, and potassium preparations. Clinical manifestations are non-specific (typically retrosternal pain, odynophagia, and dysphagia), which may delay accurate diagnosis. Drawing on two clinical cases with differing courses, this paper reviews the recent literature, highlighting the importance of early diagnosis and appropriate therapeutic management. Improved patient education regarding the correct use of medication, together with increased physicians' awareness of the risk of drug-induced oesophageal injury, may reduce its incidence, facilitate earlier diagnosis, and limit the risk of severe clinical complications.

Keywords: drug-induced oesophageal injury, pill-induced oesophageal stricture, oesophageal ulceration

Streszczenie

Polekowe uszkodzenie przełyku stanowi stosunkowo rzadkie, lecz istotne klinicznie powikłanie farmakoterapii. Patogeneza uszkodzenia błony śluzowej przełyku związana jest z bezpośrednim i przedłużonym kontaktem substancji leczniczej z błoną śluzową prowadzącym do miejscowego działania drażniącego, owrzodzeń i martwicy. Najczęściej odpowiedzialne są antybiotyki (zwłaszcza tetracykliny), niesteroidowe leki przeciwzapalne, bisfosfoniany oraz preparaty potasu. Objawy kliniczne są niespecyficzne (ból zamostkowy, odynofagia i dysfagia), co może utrudniać właściwe rozpoznanie. W artykule na kanwie dwóch przypadków klinicznych o różnym przebiegu przedstawiono przegląd najnowszej literatury przedmiotu podkreślającej istotne znaczenie wczesnego rozpoznania i podjęcia właściwego postępowania terapeutycznego. Konieczność edukacji pacjentów w zakresie prawidłowego przyjmowania leków oraz zwiększenie świadomości lekarzy dotyczącej ryzyka wystąpienia polekowych uszkodzeń przełyku może zmniejszyć częstość ich występowania, przyczynić się do wcześniejszego rozpoznania oraz ograniczyć ryzyko ciężkich powikłań klinicznych.

Słowa kluczowe: polekowe uszkodzenie przełyku, polekowe zwężenie przełyku, owrzodzenie przełyku

INTRODUCTION

Drug-induced oesophageal injury is a rare but potentially severe complication of oral pharmacotherapy. Pill-induced esophagitis was first described by Pemberton in 1970 in a patient following oral potassium chloride administration[1]. The true incidence remains unknown and is likely underestimated, as available data are derived mainly from case reports and observational studies; consequently, some diagnostic and therapeutic recommendations are based on clinical experience. At the same time, symptoms such as dysphagia or retrosternal pain are often non-specific and may be attributed to other conditions, including gastro-oesophageal reflux disease[2,3].

Medications most commonly associated with oesophageal injury include tetracyclines, non-steroidal anti-inflammatory drugs, bisphosphonates, and agents used in cardiovascular diseases (potassium chloride, quinidine). Diagnosis is usually established based on a detailed medical history, clinical correlation, and endoscopic findings, which may reveal erosions, ulcerations, and, in severe cases, mucosal necrosis of the oesophagus[3–6].

The aim of this study is to present two clinical cases with different clinical courses and to review the current state of knowledge regarding the pathogenesis, clinical presentation, diagnostic methods, and management of drug-induced oesophageal injury.

METHODS OF LITERATURE REVIEW

A literature search was conducted using the PubMed database. The following English-language search terms were used: "pill-induced esophagitis", "drug-induced esophageal injury", "pill esophagitis", "drug-induced esophagitis", and "medication-related esophageal injury".

CASE 1

A 73-year-old male was admitted to the emergency department due to persistent vomiting lasting several days and severe epigastric pain. His medical history was significant for multiple comorbidities, including type 2 diabetes mellitus, heart failure, chronic kidney disease, epilepsy, and osteoarthritis, for which he had been regularly taking non-steroidal anti-inflammatory drugs (ibuprofen, diclofenac) for several months. Physical examination revealed epigastric tenderness without peritoneal signs, hypotension (90/70 mm Hg), and tachycardia of up to 110 bpm.

Computed tomography of the chest and abdomen revealed thickening of the oesophageal, duodenal, and jejunal walls, suggestive of an inflammatory process. The patient was admitted to the cardiology department because of cardiorespiratory decompensation and persistent vomiting. Urgent upper gastrointestinal endoscopy demonstrated extensive necrosis of the oesophageal mucosa involving its entire length ("black oesophagus"), together with gastric and duodenal ulcerations.

Intensive medical management was initiated (nil *per os*, fluid resuscitation, broad-spectrum antibiotics, and proton pump inhibitor therapy), resulting in significant clinical improvement. Despite this, follow-up endoscopic examinations revealed non-healing mucosal ulcerations with progressive luminal narrowing, eventually leading to complete oesophageal obstruction. During the subsequent course of treatment, the patient underwent five endoscopic dilatations of the oesophageal strictures. As no clinical improvement was achieved, surgical intervention was indicated (Figs. 1–3).

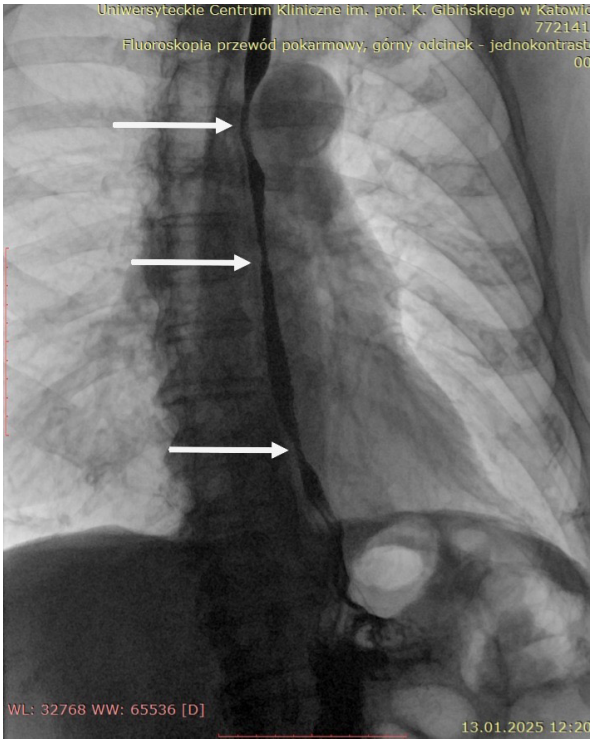

Fig. 1. Fluoroscopic image of oesophageal stricture (white arrows)

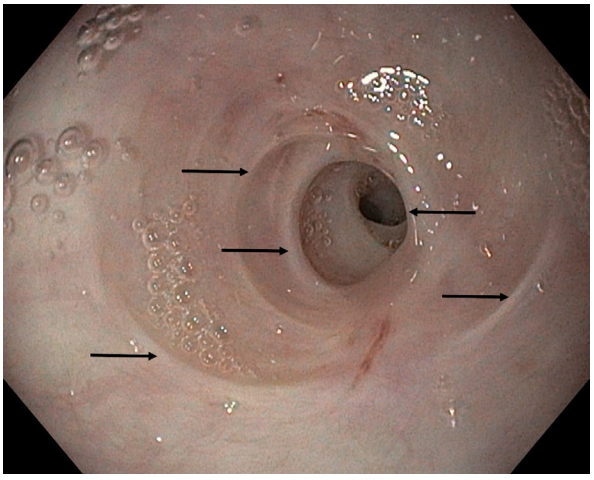

Fig. 2. Multilevel cicatricial oesophageal strictures (black arrows) secondary to healed ulcerations

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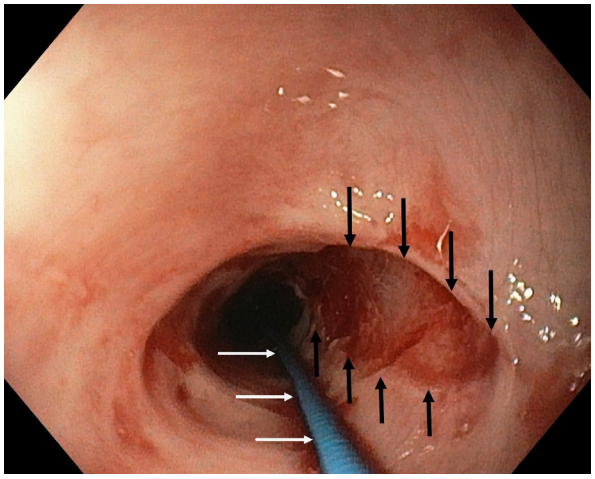

Fig. 3. Extensive disruption of the oesophageal stricture ring (black arrows) following dilatation using the bougienage technique. The white arrows indicate the atraumatic guidewire over which a Savary-Gilliard dilator is advanced

CASE 2

A 39-year-old woman was referred to hospital due to severe chest pain persisting for several hours. The symptoms began during the night after taking an evening dose of clindamycin prescribed for a periodontal abscess. Initially, the pain was burning and intermittent; subsequently, the patient developed vomiting of food contents mixed with blood, and the symptoms eventually progressed to constant, severe pain precluding swallowing.

At the first medical facility, laboratory tests, including a complete blood count and C-reactive protein (CRP) measurement, were within normal limits. Treatment with a proton pump inhibitor (PPI) was initiated, and clindamycin was replaced with metronidazole. Due to worsening symptoms, the patient subsequently presented to another hospital, where normocytic anaemia and a mildly elevated CRP level were found.

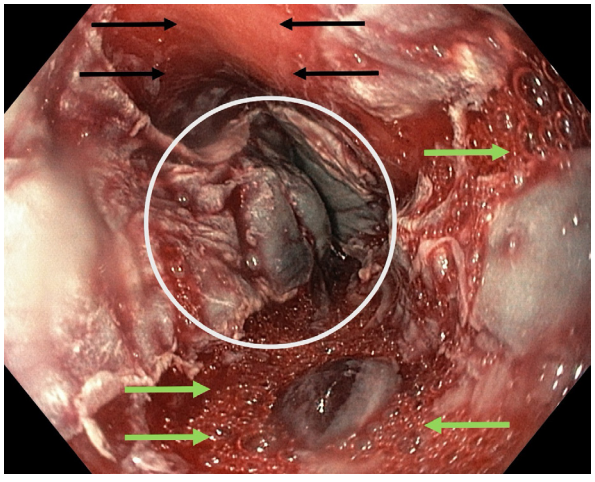

Fig. 4. Extensive necrosis of the oesophageal mucosa following clindamycin ingestion (white circle). Black arrows indicate the exposed muscular layer of the oesophageal wall. Green arrows indicate active bleeding from the mucosal surface

Urgent upper gastrointestinal endoscopy revealed extensive oesophageal mucosal necrosis extending from 25 to 38 cm from the incisors, with exposure of the muscular layer and spontaneous bleeding (Fig. 4). Additionally, at approximately 34 cm from the incisors, a suspected developing perforation was observed; however, this finding was not confirmed on contrast imaging. On the second day of hospitalisation, computed tomography of the chest and mediastinum was performed, which excluded perforation but demonstrated reactive changes in the mediastinal fat adjacent to the oesophagus.

Conservative treatment was initiated, including cessation of oral intake, continuous intravenous PPI infusion, empirical intravenous antibiotic therapy (metronidazole, piperacillin/tazobactam), thromboprophylaxis with low-molecular-weight heparin, and – from the second day of hospitalisation onward – parenteral nutrition.

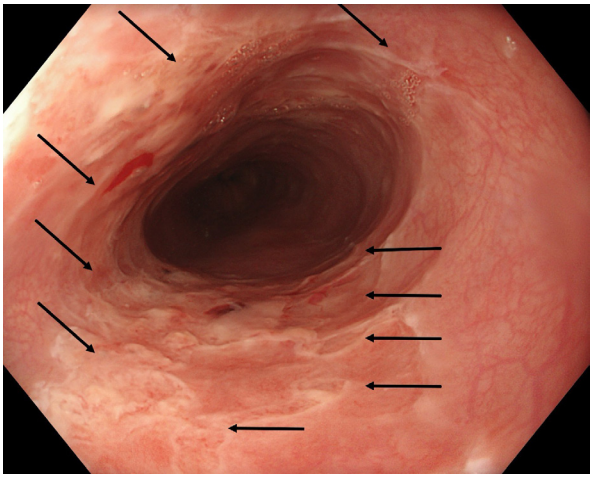

Fig. 5. Patient from Fig. 4, on day 12 following clindamycin exposure. Black arrows indicate healing oesophageal mucosa with scarring

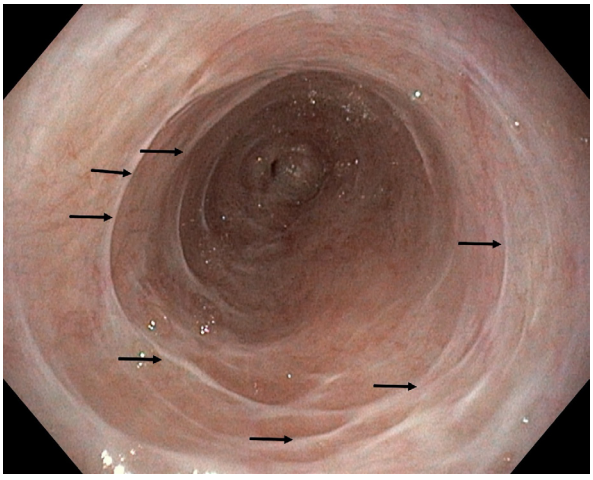

Fig. 6. Multilevel cicatricial oesophageal strictures (black arrows) secondary to healed ulcerations

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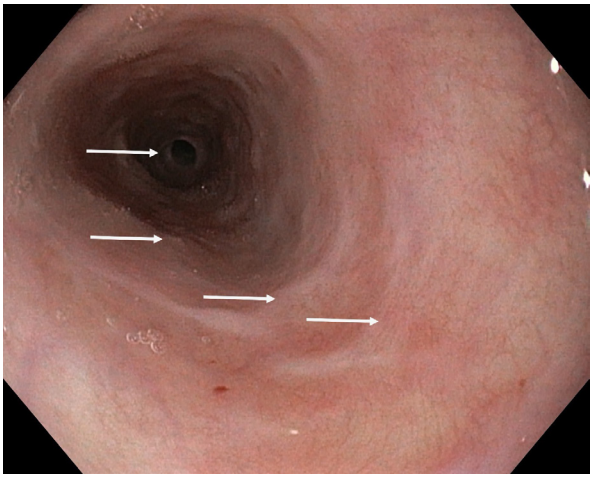

Fig. 7. Multilevel subtle cicatricial oesophageal strictures (white arrows) secondary to healed ulcerations

Follow-up upper gastrointestinal endoscopy performed on day 12 of hospitalisation demonstrated, at 31–35 cm from the incisors, two shallow healing ulcers partially covered with epithelium, fragile and prone to bleeding upon contact (Fig. 5). A subsequent endoscopic examination conducted two months later revealed only subtle post-ulcerative scarring, without evidence of oesophageal stricture (Figs. 6 and 7).

EPIDEMIOLOGY

Accurate epidemiological data on drug-induced oesophageal injury are difficult to obtain, as the condition is rare and its symptoms are often mild, leading to underdiagnosis[4,7]. The condition occurs most commonly in middle-aged individuals (approximately 40–45 years old), more frequently in women, who account for 60–70% of patients. In the paediatric population, drug-induced oesophageal injury is significantly less common, and sex-related differences are less pronounced[4,7]. Elderly individuals are particularly susceptible because of the higher prevalence of oesophageal motility disorders, reduced salivary secretion, and more frequent use of medications associated with an increased risk of oesophageal injury[4].

AETIOLOGY

To date, more than 100 medicinal substances have been reported to cause drug-induced oesophageal injury[8]. The most common causative agents are antibiotics – particularly doxycycline and other tetracyclines – and non-steroidal anti-inflammatory drugs. Less frequently, bisphosphonates, potassium preparations, iron salts, ascorbic acid, certain chemotherapeutic agents, warfarin, and paracetamol are implicated[3–6].

PATHOPHYSIOLOGY

Drug-induced oesophageal injury develops as a result of direct and prolonged contact between a medication and the oesophageal mucosa, leading to local irritation and impairment of cytoprotective mechanisms[9]. Both the pharmacological properties of the drug and mechanical factors promoting its retention within the oesophageal play a significant role. Lodgement of a tablet or capsule at a physiological or pathological narrowing – particularly when medication is taken with insufficient fluid or immediately before bedtime – increases the risk of local irritation and epithelial necrosis.

A common mechanism underlying most cases involves the synergistic effects of chemical and mechanical factors, resulting in oesophageal mucosal damage of varying severity, ranging from superficial erosions, through deep ulcerations with extensive necrosis, to late complications such as scarring and strictures[5,6,9].

Another important element of pathogenesis is the formation of an adhesive layer of drug residues on the mucosal surface, the so-called "medication biofilm", which prolongs epithelial exposure to the irritant and promotes progression of injury. Histopathological findings may demonstrate features of acute injury and inflammation consistent with the local effect of the drug[5,8].

Antibiotics from the tetracycline group (doxycycline, tetracycline, minocycline), warfarin, and iron preparations are among the agents with particularly strong irritant potential. Their mechanism of injury primarily involves local pH reduction and, in the case of iron preparations, additional oxidative stress and necrotic reactions, leading to deep ulcerations. Brownish discolouration of the mucosa at the site of contact is a characteristic endoscopic finding associated with their use[5,10].

Non-steroidal anti-inflammatory drugs damage the oesophagus both through a direct irritative effect and via inhibition of prostaglandin synthesis, which plays a key role in maintaining the integrity of the cytoprotective barrier. This results in impaired protective mechanisms, exacerbation of reflux oesophagitis, and ulcer formation, typically in the distal oesophagus[5].

Bisphosphonates (e.g. alendronate, risedronate) are characterised by particularly strong irritant properties, meaning that even brief contact with the mucosa may lead to deep ulceration. The risk increases significantly with improper administration, such as swallowing the tablet in a supine position or without adequate fluid intake[11,12].

The mechanism of oesophageal injury caused by potassium chloride and certain cardiovascular drugs (e.g. quinidine) is primarily related to high osmolarity and direct irritant effects, with lesions most commonly located in the mid-oesophagus[5].

CLINICAL PRESENTATION

Typical symptoms of drug-induced oesophageal injury include retrosternal pain, pain on swallowing (odynophagia), swallowing disorders (dysphagia), and heartburn. Less common manifestations include epigastric pain,

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haematemesis, low-grade fever or mild fever, and weight loss. Symptom onset usually occurs from a few hours up to 10–30 days after drug ingestion[2,3,10]. In most cases, however, the condition is self-limiting and resolves completely, which may hinder accurate diagnosis[3–5,13].

DIAGNOSIS

The diagnosis of drug-induced oesophageal injury is most often based on symptom assessment, a detailed medication history, and endoscopic findings. Differential diagnosis should primarily include oesophageal foreign bodies and oesophageal malignancy[5]. Endoscopic lesions are typically located in the mid-oesophagus, with erosions and ulcerations being the most common findings[4,13]. A characteristic feature is the presence of so-called "kissing ulcers", i.e. paired ulcers located on opposing walls of the oesophagus[4,5].

Most cases of drug-induced oesophageal injury are self-limiting and resolve without permanent sequelae[3–6]. Due to the limited number of prospective studies, symptomatic treatment is largely based on observational data and clinical practice. The cornerstone of management is immediate discontinuation of the offending drug and initiation of supportive therapy[5,6,9]. Depending on severity, treatment includes PPIs, antacids, and mucosal protective agents such as sucralfate. In selected cases, topical lidocaine may be used for pain relief. Avoidance of irritating foods – particularly hot, cold, or acidic substances – may help reduce symptom severity[4,6,14]. Antibiotic therapy is not routinely indicated but may be warranted in the following situations:

- oesophageal perforation;
- mediastinitis;
- signs of systemic infection (fever, sepsis);
- secondary infection of ulcerations;
- severe immunosuppression (e.g. neutropenia, post-transplant status, advanced chemotherapy).

In such cases, antibiotic therapy is either targeted or empirical broad-spectrum therapy and is directed at treating complications rather than the drug-induced injury itself.

A key component of effective management is patient education on proper medication administration. This includes avoiding the supine position when taking medications, swallowing them with an adequate amount of water, and ensuring that they are taken at least 30 minutes before bedtime[5,6,9].

CLINICAL SIGNIFICANCE

Although relatively rare, drug-induced oesophageal injury has important clinical implications due to the risk of complications and its impact on patients' quality of life. Typical symptoms – such as retrosternal pain, odynophagia, or dysphagia – may be misinterpreted as manifestations of ischaemic heart disease, gastro-oesophageal reflux disease, or upper gastrointestinal infections[14]. Limited awareness of this condition among physicians may lead to diagnostic delays, excessive testing, or unnecessary invasive procedures. In some cases, untreated or chronic drug-induced oesophageal injury may result in serious complications, including extensive ulcerations, strictures, and even perforation requiring advanced endoscopic or surgical management[4].

MANAGEMENT OF COMPLICATIONS OF DRUG-INDUCED OESOPHAGEAL INJURY

Drug-induced oesophageal injury most commonly leads to short fibrotic strictures, which are typically treated with endoscopic dilatation using either balloon dilators or rigid bougies (bougienage)[5,6,15]. Both methods have comparable efficacy and safety profiles in simple strictures, and the choice largely depends on the endoscopist's experience and equipment availability[15]. During a single dilatation session, no more than three sequential dilatations are recommended, regardless of the technique used. In most patients, adequate oesophageal lumen expansion is achieved after 3–5 sessions[15,16]. In clinical practice, a target oesophageal diameter of ≥16 mm is generally pursued, as this reduces the number of required sessions and prolongs dysphagia remission, while maintaining a very low rate of severe complications such as perforation, haemorrhage, pneumothorax, or sepsis. In population-based analyses from the United States, the complication rate of endoscopic dilatation was approximately 0.2%, with a 30-day mortality below 0.01%[16,17].

Adjunctive therapy for oesophageal strictures includes local steroid injections – most commonly triamcinolone – administered submucosally into the dilated segment. This approach has been shown to reduce stricture recurrence and the need for repeated endoscopic interventions, particularly in strictures following endoscopic submucosal dissection[18–20]. Although data specific to drug-induced strictures are limited, the antifibrotic mechanism suggests potential benefit in this patient group, improving dilatation outcomes and prolonging symptom-free intervals.

Surgical intervention should be considered in cases of refractory strictures or long-segment involvement. Indications also include inability to maintain adequate oral nutrition, suspicion of malignant transformation, or unclear aetiology of the stricture[15,21].

Surgical treatment typically involves resection of the affected oesophageal segment with restoration of gastrointestinal continuity via oesophagogastric anastomosis. The most common complications include anastomotic leakage (approximately 8–15%) and anastomotic stricture (approximately 10–20%). The 30-day postoperative mortality remains relatively high at approximately 2–4%[22–26].

Alternatively, colonic interposition or, less commonly, small bowel interposition may be used, although these approaches are associated with a higher risk of perioperative complications, including mortality[21,27]. Long-term overall survival following surgery is approximately 54% at 3 years and 42% at 5 years, with acceptable quality of life in most patients[22,27].

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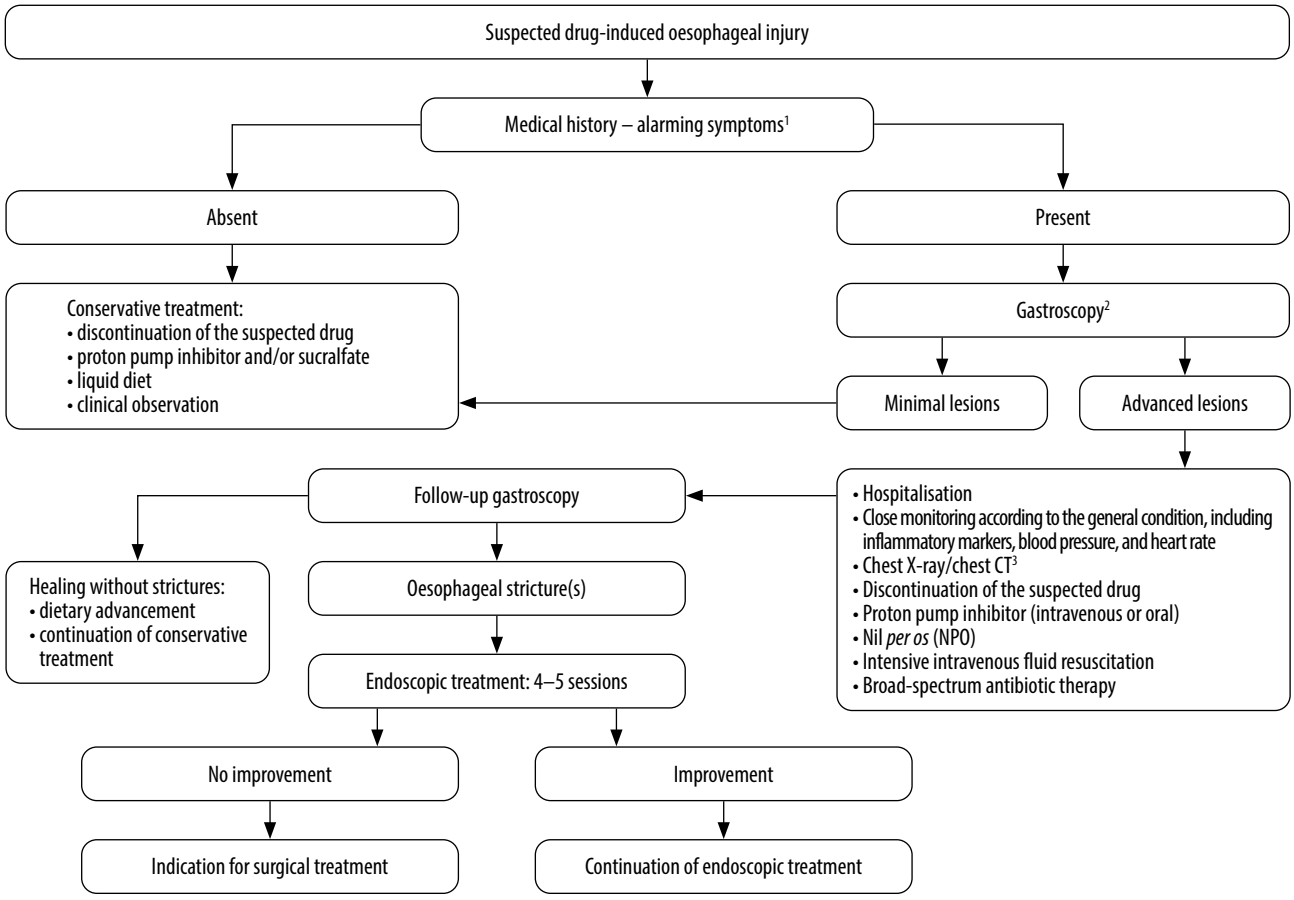

[1] Bleeding, dysphagia, suspected perforation.
[2] Assessment of lesion extent; exclusion of other causes (foreign body, malignancy, infections); presence of erosions, "kissing ulcers"; in severe cases, extensive mucosal necrosis.
[3] X-ray/CT – radiography/computed tomography.

Fig. 8. Management of drug-induced oesophageal injury[5,6,15]

The prognosis of benign inflammatory oesophageal strictures, including those caused by medications, is generally favourable when managed with a strategy of gradual, repeated dilatations to a diameter of at least 16–18 mm. This approach reduces the need for reintervention, while surgical treatment remains a rescue option reserved for a small, carefully selected group of patients[15–17]. It should be noted that most survival data after oesophagectomy are derived from patients operated on for oesophageal cancer and may not fully reflect outcomes in other patient populations.
The management algorithm for drug-induced oesophageal injury is presented in Fig. 8.

CONCLUSIONS

Drug-induced oesophageal injury, although a rare complication of pharmacotherapy, represents a significant clinical problem due to the risk of serious outcomes such as strictures and perforation. Early diagnosis is crucial for effective management and relies on careful medication history and endoscopic evaluation.
The cornerstone of therapy is discontinuation of the causative agent, symptomatic treatment, and modification of medication-taking practices. In cases of complications, minimally invasive approaches – particularly endoscopic dilatation – are the mainstay of treatment, while surgical intervention remains reserved for selected patients in whom other strategies have failed.
Analysis of the presented cases highlights the need for heightened awareness of drug-induced oesophageal injury among both physicians and patients. Patient education on proper medication administration and early recognition of symptoms suggestive of oesophageal injury may significantly reduce the risk of complications.

Conflict of interest

The authors declare no financial or personal relationships with other individuals or organisations that could adversely affect the content of this publication or claim rights to this publication.

Authors' contributions

Original concept of study; critical review of manuscript; final approval of manuscript: PW. Collection, recording and/or compilation of data; analysis and interpretation of data: KTT, WWM. Writing of manuscript: KZT, WWM, KDK.

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