Review

Dysphagia and other manifestations of oesophageal involvement in the musculoskeletal diseases

N. J. Sheehan

Oesophageal dysfunction can occur in a variety of musculoskeletal conditions, most notably the autoimmune connective tissue diseases. However, correlation between oesophageal symptoms and investigations is frequently poor. It is often uncertain whether symptoms are caused by the underlying disease or by its treatment. As the primary disease process may not be responsive to treatment, correctable iatrogenic causes should always be sought.

Key words: Dysphagia, Oesophageal function, Connective tissue diseases, Cervical spine, Treatment.

Introduction

Oesophageal dysfunction can occur in a wide range of musculoskeletal conditions. Symptoms include dysphagia, odynophagia and heartburn.

The oesophagus connects the pharynx opposite the sixth cervical vertebra to the cardia, slightly to the left of the eleventh thoracic vertebra. It is fixed above by the cricoid cartilage and below by the diaphragm, the upper end being closed by the pharyngo-oesophageal sphincter and the lower end by the lower oesophageal sphincter (LOS). The muscular coat has outer longitudinal and inner circular layers with Auerbach’s plexus in between. Both muscle layers are striated in the upper portion of the oesophagus.

At the initiation of swallowing, food is moved voluntarily from the mouth to the pharynx, triggering a primary peristaltic wave in the pharynx and causing the upper oesophageal sphincter (UOS) to relax, allowing the food to enter the oesophagus. Direct stimulation of the oesophagus incites secondary peristalsis which propels the contents forwards to the stomach, usually assisted by gravity. The LOS also relaxes at the onset of swallowing.

Dysphagia can be divided into upper and lower types depending on whether the delay occurs in the pharynx or upper oesophagus or, alternatively, in the body of the oesophagus or at the oesophago–gastric junction. Most cases of high dysphagia are due to a failure of pharyngeal contraction or cricopharyngeal relaxation, or both, whilst low dysphagia is more often caused by obstruction. Dysphagia for both solids and liquids is more likely to be due to an oesophageal motility disorder than to mechanical obstruction.

Involvement of the oesophagus in musculoskeletal diseases

Although dysfunction of the oesophagus is common in autoimmune and inflammatory rheumatic diseases, it is usually asymptomatic. Nevertheless, significant swallowing difficulties can occur and occasionally dysphagia is severe. Many different mechanisms can contribute to dysphagia in the musculoskeletal diseases (Table 1). However, the cause of oesophageal symptoms is often obscure as they frequently correlate poorly with the findings of investigations.

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Submitted 11 October 2007; revised version accepted 11 January 2008.

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Investigation of oesophageal dysfunction

Because of its accessibility, the oesophagus is the easiest part of the gastrointestinal (GI) tract to investigate. Oesophageal manometry is highly sensitive and specific for the detection of oesophageal dysmotility. It provides quantitative pressure data and is capable of detecting early changes such as minor reductions in the amplitude of peristaltic waves and reduced LOS pressure [1]. Although less sensitive, videofluoroscopy with barium swallow is also a useful technique for identifying abnormal oesophageal peristalsis [2]. Barium studies alone are of limited use in detecting motility abnormalities but may be the most sensitive tests for the detection of oesophageal strictures or dilatation [3]. Endoscopy is the preferred investigation for oesophagitis and the presence of Barrett’s metaplasia, whilst 24-h ambulatory pH testing is highly sensitive for the diagnosis of gastro-oesophageal reflux disease (GERD) [3]. Oesophageal scintigraphy or radionuclide scanning specifically demonstrates gastro–oesophageal reflux or pulmonary aspiration but lacks sensitivity [3]; it correlates with reduced amplitude of peristalsis or aperistalsis and low LOS pressure [4].

Autoimmune diseases

Scleroderma

Amongst the musculoskeletal diseases, dysphagia is best known as a complication of scleroderma, in which it is an eponymous feature of CREST syndrome. Although scleroderma can affect any part of the GI tract, the oesophagus is involved most often, with oesophageal manifestations occurring in up to 90% of patients [3]. Dysphagia is only rarely a presenting complaint of scleroderma and symptoms occur as the oesophagus becomes more severely involved. When the oesophagus is affected, usually the disease process is diffuse with involvement of multiple levels of the GI tract. Replacement of the smooth muscle layers of the oesophagus by fibrous tissue results in diminished peristalsis, the extent of the resulting hypomobility varying from occasional uncoordinated contractions to complete paralysis [5]. The motility disturbance characteristically involves the body of the oesophagus and the LOS [1]. UOS impairment is less common than in the other connective tissue diseases [1]. The velocity of peristaltic contractions in the proximal oesophagus is increased in scleroderma [6], possibly as a compensatory response to the dysmotility below.

Abnormalities of the LOS are severe in 30% of patients [1]. Incompetence of the LOS permits gastro-oesophageal reflux and several other mechanisms also contribute to the development of GERD. Reflux may be exacerbated by hiatus hernia which occurs in over 90% of patients due to dilatation and shortening of the
oesophagus [5], whilst impaired or absent oesophageal peristalsis prevents clearance of refluxed acid back into the stomach [3]. Oesophagitis occurs in one-third of the patients and its incidence approaches 100% in patients with severe cutaneous involvement [7]. Poor emptying of the oesophagus, immunosuppressive therapy and acid suppression predispose to Candida oesophagitis [4]. Chronic acid reflux may result in complications such as stricture formation, Barrett’s metaplasia and carcinoma [3]. The frequency of strictures ranges from 2% to 48% in different series, with an overall average of ~9% [8] (Fig. 1). Barrett’s metaplasia affects one-third of patients with scleroderma creating an increased risk of adenocarcinoma [9]. A case of oesophageal–atrial fistula has been described in CREST syndrome, secondary to perforation of an ulcer in Barrett’s oesophagus into the left atrium [10]. Upper GI haemorrhage has been reported from oesophageal ulcers and oesophageal telangiectasia [10]. However, extensive oesophageal damage, including severe oesophageal reflux, may be present in progressive systemic sclerosis in the absence of oesophageal symptoms [11]. Other factors that may contribute to dysphagia in scleroderma include impaired mandibular motion and mastication due to atrophy and fibrosis of the perioral skin, and reduced acid-neutralizing capacity due to secondary Sjögren’s syndrome [12]. An involuntary sound, dubbed the scleroderma bark, has been attributed to expulsion of swallowed air from the stomach back into the oesophagus [13].

**Eosinophilic fasciitis**

A similar pattern of oesophageal dysfunction as it occurs in systemic sclerosis can also occur in eosinophilic fasciitis [14].

**Sjögren’s syndrome**

Dysphagia occurs in three-quarters of patients with Sjögren’s syndrome [15, 16] and may be related to a combination of lack of saliva and oesophageal dysmotility. As well as reducing lubrication and hence prolonging pharyngeal transit time, absence of saliva predisposes to dental caries and to oral Candida, both of which may impair mastication [17]; it also diminishes the acid clearance capacity of the oesophagus [18].

Defective peristalsis has been demonstrated in one-third or more of patients with primary Sjögren’s syndrome [16, 19]. Decreased or absent contractility has been shown in the upper third of the oesophagus [20], and UOS impairment may be more severe than in the other connective tissue diseases [1]. Other patterns of oesophageal dysfunction have also been described [19], including achalasia [21]. Oesophageal webs are found in ~10% of patients [15].

Oesophageal symptoms do not correlate well with manometry or salivary secretion [16, 18, 22] and other unknown factors may be instrumental in the dysphagia of Sjögren’s syndrome. It has been postulated that the discrepancy between subjective swallowing difficulties and the lack of objective signs of pharyngeal and oesophageal dysmotility as assessed by videoradiography may be related to impaired parasympathetic function [23].

**Systemic lupus erythematosus**

Dysphagia occurs in up to 13% and heartburn in up to 50% of patients with SLE [24]. Oesophagitis with ulceration has been observed in 3–5% and oesophageal perforation may rarely occur. Reduced oesophageal motility has been demonstrated in up to 72% of patients with SLE [25] and, although the motility disorder is mostly mild, aperistalsis has been reported in 10–25% [26, 27].

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**Table 1. Causes of dysphagia in the musculoskeletal diseases**

<table>
<thead>
<tr>
<th>Lesion</th>
<th>Disorder</th>
<th>Mechanism</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diseases of the mouth and tongue</td>
<td>Xerostomia</td>
<td>Loss of lubrication, dental caries</td>
<td>Sjögren’s syndrome, Behçet’s disease, SLE</td>
</tr>
<tr>
<td></td>
<td>Ulceration</td>
<td>Painful mastication</td>
<td>Immunosuppression with corticosteroids, cytotoxic agents</td>
</tr>
<tr>
<td></td>
<td>Candidiasis</td>
<td>Painful mastication</td>
<td>RA</td>
</tr>
<tr>
<td>Joint disease</td>
<td>Temporomandibular joint arthritis</td>
<td>Painful mastication</td>
<td>Polymyositis, inclusion body myositis</td>
</tr>
<tr>
<td>Inflammatory myopathy</td>
<td>Cricopharyngeal muscle spasm</td>
<td>Impaired initiation of glutation</td>
<td>Medication, vasculitis</td>
</tr>
<tr>
<td>Mucosal lesions of the oesophagus</td>
<td>Oesophagitis, ulceration, stricture, web, vasculitis</td>
<td>Hypo- or aperistalsis</td>
<td>Systemic sclerosis, other connective tissue diseases</td>
</tr>
<tr>
<td>Oesophageal motility disorders</td>
<td>Achalasia, pseudo-achalasia, fibrosis</td>
<td>External compression of oesophagus</td>
<td>Spondylosis, DISH</td>
</tr>
<tr>
<td>Cervical spine disease</td>
<td>Osteophytes</td>
<td>External compression of oesophagus</td>
<td>Ankylosing spondylitis</td>
</tr>
<tr>
<td></td>
<td>Syndesmophytes, infection</td>
<td></td>
<td>Pre-vertebral abscess</td>
</tr>
<tr>
<td>Mediastinal disease</td>
<td>Lymphadenopathy</td>
<td></td>
<td>Sarcoïdosis, lymphoma complicating autoimmune disease</td>
</tr>
<tr>
<td>Orthoses</td>
<td>Cervical collar</td>
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</table>

**FIG. 1.** Barium swallow examination in patient with scleroderma, showing long distal oesophageal peptic stricture (large arrow) and mucosal ulceration (small arrow). (Image kindly provided by Dr Hany El-Madbouh).
As in Sjögren’s syndrome, the upper one-third of the oesophagus is mainly affected [28], whilst the LOS is relatively spared compared with the other autoimmune rheumatic diseases [1]. The most specific disorder is an isolated abnormal peristalsis of the body of the oesophagus [1].

The aetiopathological process underlying oesophageal dysmotility in patients with SLE is uncertain but inflammation of the oesophageal muscles or vasculitic damage to the Auerbach plexus has been postulated [25].

Whilst dysphagia is typically related to GERD or stricture, swallowing difficulties may be exacerbated by recurrent mouth ulcers in ~30% of patients and Sjögren’s syndrome in ~20%. Dysphagia can also have an infective cause, such as Candida albicans, especially in patients on immunosuppressive therapy [29].

There has been a single report of dysphagia due to epidermolyisis bullosa acquisita involving the oesophagus [30].

Dysphagia and GERD correlate poorly with oesophageal manometric abnormalities [24].

Anti-phospholipid antibody syndrome, which usually occurs in association with SLE but can also exist in a primary form, has been linked to a number of GI manifestations, including a case of oesophageal necrosis with perforation due to vascular thrombosis [31].

**Idiopathic inflammatory myopathies**

The GI manifestations of the idiopathic inflammatory myopathies include uncoordinated swallowing, uncoordinated oesophageal peristalsis and hiatus hernia with reflux and stricture formation [32].

Dysphagia occurs in 8–30% of patients with inflammatory myopathy and it is a presenting symptom in one-fifth of these, the incidence being highest in those with inclusion body myositis [33, 34]. It is associated with more severe disease and a poor prognosis. Abnormalities of oesophageal motility are common and involve both the upper and the lower oesophagus [32]. Where there is weakness of the tongue and loss of muscle tone in the upper part of the oesophagus, swallowing may become impossible in the recumbent position owing to elimination of the effect of gravity [35].

Impairment of the UOS is more frequent than in scleroderma [1]. In contrast to the lack of association between inflammation and dysmotility of the upper and lower oesophagus, cricopharyngeal muscle dysfunction, which can cause a distinctive dysphagia characterized by a sensation of food sticking in the back of the throat or coughing with swallowing, correlates with general disease activity [36]. Distal oesophageal abnormalities occur in the absence of proximal oesophageal skeletal muscle dysfunction in 70% of patients [32]. Whilst the dysmotility of the distal oesophagus is functionally similar to that of scleroderma, muscle atrophy and fibrosis may not be found, suggesting that other factors may contribute to the abnormality in polymyositis and dermatomyositis. The degree of distal oesophageal involvement appears to correlate with the duration of myositis [32].

Weakness of smooth muscle and degeneration of the striated muscle may lead to the formation of oesophageal diverticulae [35], whilst decreased oesophageal motility and LOS pressure may predispose to GERD.

Patients treated with steroids have an increased risk of oropharyngeal and oesophageal candidiasis and there is also an increased risk of oesophagitis secondary to herpes simplex and CMV infections [17].

The possibility of a co-existent malignant tumour obstructing the oesophagus should be borne in mind, particularly in dermatomyositis [37, 38].

Vasculitis can cause mucosal ulceration and even intestinal perforation. This is more common in childhood dermatomyositis in which these features have been described throughout the GI tract from the oesophagus to the large intestine [39].

Dysphagia occurs in 40% to over 80% of cases of inclusion body myositis in which it may occasionally be the presenting manifestation [40, 41]. Dysphagia is most refractory in patients with this type of myopathy [34] and, when progressive, it is indicative of a poor prognosis [41]. However, as with polymyositis and dermatomyositis, some cases are due to cricopharyngeal dysfunction and respond to myotomy [40].

**Mixed connective tissue disease**

Diagnostic criteria for mixed connective tissue disease [42] include oesophageal hypomobility (or dilatation of the oesophagus). Oesophageal involvement is found in up to 85% of patients, with dysphagia (38%) and heartburn (48%) being the commonest GI symptoms [43, 44].

The pattern of oesophageal dysfunction is similar to that seen in systemic sclerosis. However, as well as diminished distal peristalsis and reduced LOS competency, UOS hypotension is also common [43].

**Rheumatoid arthritis**

In a survey of digestive symptoms in connective tissue diseases, one-third of the patients with RA reported dysphagia [45].

Disorders of oesophageal motility are found in 30% of cases or more of RA [1, 46]. Although less frequent than in systemic sclerosis [1], low-amplitude peristaltic waves in the lower two-thirds of the oesophagus and reduced LOS pressure have also been shown in RA in which they similarly predispose to GERD [47]. In addition, decreased peristaltic pressure has been demonstrated in the proximal part of the oesophagus, indicating dysfunction of the striated muscles [48]. As with the other connective tissue diseases, there is no correlation between dysphagia and oesophageal manometry results [48].

The evidence is conflicting whether dysphagia is related to disease severity or duration in RA [47, 48].

Sicca syndrome and temporomandibular joint involvement may make chewing and swallowing difficult. Dysphagia has also been reported as a complication of laryngeal involvement with synovitis and nodules in RA [49], and due to Plummer–Vinson syndrome in patients with RA and iron deficiency anaemia [50, 51]. There has been a reported case of perforation of the oesophagus in RA occurring in association with achalasia, where a penetrating stasis ulcer led to an oesophagomediastinal sinus and mediastinal abscess [52].

However, the most common GI problems encountered in patients with RA are due to drug therapy with NSAIDs, glucocorticoids and DMARDs.

In Felty’s syndrome, nodular hyperplasia of the liver can give rise to portal hypertension and potentially catastrophic bleeding from oesophageal varices [53, 54].

The prevalence of oesophageal dysmotility in the connective tissue diseases is summarized in Table 2. Figure 2 shows the main sites of the motility disorder in the respective diseases.

**Vasculitis**

Oesophageal involvement in vasculitis is largely overlooked. However, vasculitis can affect vessels of any type in any organ and GI involvement occurs in many vasculitic diseases [56].

**Behçet’s disease**

Although GI involvement occurs in 3–26% of patients [57] and oesophageal manometry is abnormal in a third of cases [58], oesophageal symptoms are reported by a minority of individuals with Behçet’s disease. Vasculitis is observed rarely and varices, ulceration or perforations of the oesophagus are also rare complications [56, 59, 60].
Sarcoidosis can affect the oesophagus in different ways. Stenosis of the distal oesophagus due to direct granulomatous involvement [69, 70], or extrinsic compression by enlarged hilar and mediastinal lymph nodes, may both cause dysphagia. Sarcoideal infiltration of the distal oesophagus can give rise to achalasia [71], and granulomatous myositis of the criopharyngeal muscle causing dysphagia has also been reported [72]. Barrett’s oesophagus can also occur in sarcoidosis [73].

**Amyloidosis**

Although oesophageal deposits of amyloid are usually due to primary amyloidosis, dysphagia caused by pseudoachalasia due to secondary amyloidosis complicating RA has been described [74].

**Inflammatory bowel disease**

Ulcerative colitis and Crohn’s disease are strongly associated with spondyloarthropathy with 15–20% of patients with inflammatory bowel disease having a characteristic peripheral arthritis, 4–18% bilateral sacroilitis and 3–6% spondylitis [75].

Reports of the frequency of oesophagitis in ulcerative colitis have been contradictory but one endoscopy study found erosions of the abdominal oesophagus in one in seven patients [76, 77].

Crohn’s disease of the oesophagus is rare but it may include inflammation, ulceration, strictures and fistula formation [78].

**Disorders of the cervical spine**

Because of the close relationship of the oesophagus to the cervical spine, spinal disorders in the neck can interfere with oesophageal function. Extrinsic compression by large anterior osteophytes may occur in cervical spondylosis and especially in diffuse idiopathic skeletal hyperostosis (DISH, Forestier’s disease) [79–81] (Fig. 3). In one series, dysphagia was found in 17–28% of cases of DISH [82]. The obstruction occurs most commonly at the C5/6 vertebral level and less commonly at C4/5, C2/3 and C3/4 [79]. Thoracic osteophytes rarely cause dysphagia as the thoracic portion of the oesophagus is relatively mobile and is displaced rather than compressed by the hyperostoses [83].

As well as direct mechanical compression, other postulated mechanisms for dysphagia include periesophageal fibrosis and paralytic ileus, fixation of the oesophagus by fibrosis and adhesions, cricopharyngeal muscle spasm, impaired epiglottic motility and distortion of the larynx or laryngeal cartilages [81]. Irritation of the cranial or sympathetic nerves has also been suggested [84].

Ossification of the annulus fibrosus and longitudinal ligament in ankylosing spondylitis can also compress the oesophagus causing dysphagia [85]. Infective causes include cervical osteomyelitis with a pre-vertebral abscess [86]. A tight-fitting cervical collar prescribed for neck pain may cause dysphagia by restricting laryngeal movement during swallowing [87].

**Complications of anti-rheumatic therapy**

Often, it is impossible to know whether upper GI dysfunction is a systemic manifestation of the disease in question or due to its treatment. Many drugs used to treat musculoskeletal conditions can affect swallowing in various ways.

Gold compounds (intramuscular and oral), penicillamine, sulfasalazine, methotrexate and other cytotoxic drugs can cause stomatitis and oral ulcers. Similarly, NSAIDs, corticosteroids and bisphosphonates can cause oesophagitis and oesophageal ulceration. Alendronate sodium can cause injury both by a toxic effect of the drug and by physical irritation of the mucosa by the pill [88]. Whereas ‘pill oesophagitis’ may cause identical symptoms to GERD with retrosternal chest pain and possibly dysphagia, odynophagia is usually the dominant complaint [3]. Steroid therapy and other immunosuppressive agents may also impair deglutition by predisposing to candidiasis of the upper GI tract. Gold-induced enterocolitis has been found histopathologically to involve the oesophagus as well as the stomach and small bowel [89].

**TABLE 2. Prevalence of oesophageal dysmotility in the connective tissue diseases**

<table>
<thead>
<tr>
<th>Disease</th>
<th>Oesophageal dysmotility (%) [Refs]</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systemic sclerosis</td>
<td>75–92 [1, 3]</td>
</tr>
<tr>
<td>SJögren’s syndrome</td>
<td>30–100 [1, 16, 19, 20]</td>
</tr>
<tr>
<td>SLE</td>
<td>63–72 [1, 26]</td>
</tr>
<tr>
<td>Idiopathic inflammatory myopathy</td>
<td>50–69 [1, 55]</td>
</tr>
<tr>
<td>Mixed connective tissue disease</td>
<td>88 [1]</td>
</tr>
<tr>
<td>RA</td>
<td>30–58 [1, 46]</td>
</tr>
</tbody>
</table>

**Other systemic vasculitides**

Involvement of the GI tract is uncommon in WG but odynophagia due to oesophageal vasculitis with ulceration has been reported [61]. GI involvement is frequent in Churg–Strauss syndrome but eosinophilic vasculitis-related oesophagitis is very rare [62].

Dieulafoy’s lesion, an abnormal submucosal artery characterized by massive GI bleeding, has been described in the oesophagus as a complication of Takayasu’s arteritis [63].

The GI complications of Henoch–Schoénlein purpura include oesophageal strictures [17, 64] and carcinoma of the oesophagus [65].

**Inherited disorders of connective tissue**

Defective collagen synthesis may give rise to megaesophagus in Marfan’s and Ehlers–Danlos syndromes. Achalasia has been described in Marfan’s syndrome [66], whilst spontaneous rupture of the oesophagus has been reported in Ehlers–Danlos syndrome [67, 68].

**Other systemic rheumatic diseases**

Sarcoidosis

Sarcoidosis can affect the oesophagus in different ways. Stenosis of the distal oesophagus due to direct granulomatous involvement [69, 70], or extrinsic compression by enlarged hilar and mediastinal lymph nodes, may both cause dysphagia. Sarcoideal infiltration of the distal oesophagus can give rise to achalasia [71], and granulomatous myositis of the criopharyngeal muscle causing dysphagia has also been reported [72]. Barrett’s oesophagus can also occur in sarcoidosis [73].
In SLE, in addition to standard measures for oesophageal peptic or drug-induced lesions, treatment of the lupus disease process may be of benefit if the oesophageal lesions are proven on biopsy to be vasculitic in origin [24].

Corticosteroid therapy may improve oesophageal dysfunction in mixed connective tissue disease [43] but it is rarely needed.

Plasma exchange improves dysphagia in patients with acute polymyositis and dermatomyositis [98]. Intravenous immunoglobulin (IVIG) can be efficacious for refractory inflammatory myopathy, with prolonged remission occurring in 50% of successfully treated patients with polymyositis following discontinuation of therapy [99]. It is more effective when used as second-line therapy [100]. Administration of IVIG, with or without corticosteroids, may also be highly effective for severe dysphagia in inclusion body myositis [101].

Obstructive causes of dysphagia may require surgery. Cricopharyngeal myotomy is the most beneficial intervention for dysphagia in idiopathic inflammatory myopathy [34] but dilatation may be attempted if surgery is contraindicated [36]. Injection of botulinum toxin A into cricopharyngeus may also obviate the need for surgical myotomy [102].

Oesophageal lesions in Behcet’s disease may respond to prednisolone [60] but the recurrence rate after both medical and surgical treatment is high [59]. Oesophageal involvement in WG responds to standard therapy for the disease [61].

Mild Crohn’s disease of the oesophagus should be treated with acid suppression and a short course of corticosteroids whilst moderate-to-severe or resistant disease requires a longer course of steroid therapy and possible immunosuppressive therapy with azathioprine or 6-mercaptopurine. Infliximab or other TNF-α antagonists can be considered for difficult cases with the aim of preventing strictures and fistula formation [103]. Crohn’s strictures of the oesophagus can be improved by balloon dilatation and injection of a long-acting steroid but surgery may be required for severe refractory symptoms [78, 103]. Pneumatic bag dilatation may also be successful in pseudoachalasia due to amyloid infiltration of the oesophagus [104].

Infiltration of botulinum toxin may provide rapid symptom relief in achalasia secondary to sarcoidosis, pending definitive treatment with steroid therapy or surgery [71].

Dysphagia due to DISH is relieved by surgical cervical decompression [79] but conservative treatment with sedation, anti-inflammatory analgesics, antibiotics, corticosteroids, muscle relaxants and a soft diet may be adequate in milder cases [80, 83, 84]. Surgery is indicated for persistent dysphagia, pain and weight loss where medical management has failed [83].

In extreme cases, severe dysphagia can lead to protein–calorie malnutrition and the need for nutritional support. Enteral feeding via a nasogastric tube or percutaneous endoscopic gastrostomy (PEG) may be necessary. Nasogastric tube feeding should be used only for short-term nutritional repletion as it exacerbates gastro-oesophageal reflux and the tube can also cause pharyngeal oesophageal ulceration and oesophageal stricture [105]. PEG feeding is superior for longer term use with regard to tolerance, complications and weight gain [105].

Optimal management of dysphagia leading to an improved quality of life may be best achieved through a multi-disciplinary strategy that includes physical therapy and psychological counseling [106].

Conclusion

Oesophageal dysfunction is a common feature of musculoskeletal conditions but fortunately serious dysphagia is rare. As the underlying disease process is not always responsive to treatment, correctable iatrogenic causes should be borne in mind.

Management of dysphagia may benefit from a multidisciplinary approach.
**Rheumatology key messages**

- Esophageal dysfunction is common in connective tissue diseases, but is frequently asymptomatic.
- Dysphagia can occur in a wide range of musculoskeletal conditions.
- Esophageal symptoms may be due to the underlying disease, its treatment or both.

**Acknowledgements**

The author is grateful to Dr Mike Dronfield, consultant gastroenterologist, for commenting on the manuscript.

**Disclosure statement:** The author has declared no conflicts of interest.

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