Oesophageal dysfunction in patients with primary Sjögren’s syndrome

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SUMMARY Oesophageal motility was studied in 22 patients with primary Sjögren’s syndrome and 20 normal volunteers. Oesophageal dysfunction was detected in eight of the 22 patients (36.4%) with primary Sjögren’s syndrome. No abnormalities were detected in the normal subjects. Individual analysis of the oesophageal motility studies showed different patterns of oesophageal dysfunction: aperistalsis (three patients), triphasic tertiary contractions (two patients), frequent non-peristaltic contractions (two patients), and low contractions (one patient). These oesophageal abnormalities did not correlate with the parotid flow rate, the degree of inflammatory infiltrate of the minor salivary glands, the extraglandular manifestations, or the presence of autoantibodies.

Key words: Raynaud’s phenomenon, dysphagia.

Dysphagia is a common complaint of patients with primary Sjögren’s syndrome.1 Despite that, studies of the oesophageal function in these patients are limited. In 1964 Stevens et al. studying oesophageal function in connective tissue disorders had included three patients with Sjögren’s syndrome. In their work, however, it is not clear whether or not the patients with Sjögren’s syndrome had any dysfunction in the oesophageal motility.2 In 1976 Ramirez-Mata et al. observed that nine out of 10 patients with primary Sjögren’s syndrome had a characteristic pattern of abnormal oesophageal motility.3

In the present study we have found that the oesophageal motility is affected in one third of patients with primary Sjögren’s syndrome. The oesophageal dysfunction does not follow any characteristic pattern and does not correlate with clinical, histological, or serological parameters.

Patients and methods

Twenty-two female patients with primary Sjögren’s syndrome were studied. Their ages ranged from 33 to 81 years (average 52.7±12.2 (SD) years). The diagnosis of Sjögren’s syndrome was based on xerostomia (decreased parotid flow rate) and keratoconjunctivitis sicca (punctate corneal ulcers on slit-lamp examination and abnormal Schirmer’s test).4 In all patients the diagnosis was confirmed by lip biopsy.5 None of these patients had other associated autoimmune disease. Eight of the 22 patients with Sjögren’s syndrome had extraglandular manifestations (Raynaud’s phenomenon, lymphadenopathy, splenomegaly, interstitial pneumonitis, vasculitis). The sera of all these patients were tested for rheumatoid factor (RF) titre and antibodies to Ro(SSA) and La(SSB) cellular antigens as previously described.6

Twenty normal volunteers had an oesophageal manometric study. Their ages ranged from 22 to 74 years (average 43.7±15.2 (SD) years).

In all patients and normal volunteers the oesophageal manometric study was done after an overnight fast by the step pull-through method,7 which has been modified by us.

MANOMETRIC PROCEDURE

A solid state catheter with built-in three double pressure sensors was passed into the stomach through the nose. The three pressure sensors were spaced at 5 cm intervals and were oriented radially at angles of 60° to each other. The pressure sensing catheter generates voltages which were transmitted directly to a Beckman R 511 A multichannel ink recorder. Respiration and deglutition were moni-
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Oesophageal peristalsis was elicited by both wet and dry swallows. Wet swallows consisted of 5 ml water at room temperature. With the patient supine the catheter was withdrawn slowly until first the proximal double pressure sensor and next the other two were placed on the lower oesophageal sphincter (LOS). At each station the pressure of LOS was read as the mean amplitude recorded after three wet and three dry swallows (one mean for wet and another for dry swallows). The catheter was next positioned with the distal double pressure sensor 1, 2, and 4 cm above the LOS. The same technique as for LOS pressure was used to study the upper oesophageal sphincter (UOS) pressure. The catheter was next positioned with the proximal double pressure sensor 2 cm above and 2 and 4 cm below the UOS.

The manometric study and the reading of the tracings were carried out by the first author who was unaware of the patient’s diagnosis.

The statistical differences between the groups were evaluated by Student’s t test and χ² test where indicated.

Results

Oesophageal dysfunction was detected in eight of the 22 patients (36.4%) with primary Sjögren’s syndrome, whereas none of the normal volunteers had any abnormality. Individual analysis of the oesophageal motility studies showed different patterns of oesophageal-body motility: (a) aperistalsis of the upper 10 cm in one patient and of the whole oesophagus in one patient; (b) aperistalsis of the whole oesophagus during dry swallows in one patient (Figs 1 and 2); (c) triphasic tertiary contractions of the upper 8–12 cm in two patients (Fig. 3); (d) frequent non-peristaltic contractions of the upper 10 cm in one patient or of the whole oesophagus in one patient (Fig. 4); (e) low contractions of the upper 5–7 cm in one patient (Fig. 5).

We have not observed any abnormality of the upper or lower oesophageal sphincter. More precisely, the mean pressures of UOS and LOS in our normal volunteers were 81±83±14±53 (SD) and

Fig. 1 Manometric tracing of the oesophagus in a patient with primary Sjögren’s syndrome. Aperistalsis was observed in the whole oesophageal body during dry swallows. Each 5 mm in the tracing is equal to 10 mmHg.

Fig. 2 Normal peristalsis in the same patient during wet swallows.
This discrepancy of the incidence of oesophageal dysfunction in Sjögren's patients can be attributed to the small number of patients examined by Ramirez-Mata et al. The lack of a specific pattern of oesophageal involvement in Sjögren's patients is in accordance with recent work which indicates that even patients with scleroderma and myositis do not have specific oesophageal dysfunction patterns, though it is well known that scleroderma affects only smooth muscle.

Our study did not show any correlation of the oesophageal abnormality with the degree of the inflammatory infiltrate of the labial minor salivary glands, the parotid flow rate, the presence of Raynaud's phenomenon or other extraglandular manifestations, or the presence of autoantibodies such as rheumatoid factor and antibodies to Ro (SSA) and La(SSB) cellular antigens.

Why then is oesophageal function impaired in some patients with Sjögren's syndrome? We propose that, firstly, the exocrine glands of the oesophageal body are infiltrated by inflammatory...
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References


Fig. 5 Low contractions in the 7 cm from the upper oesophageal sphincter (UOS). UOS is recorded in the second tracing from the upper end.

cells which directly (cytotoxic action or lymphokines) or indirectly (development of collagen tissue) interfere with the muscular function of the oesopha-
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