level and severe constipation of the colon,^{1,2} even if other segments of GI are known to be affected in subclinical form.

Objectives Few studies have assessed the morphological features of the stomach in SSc. A 52 years old female with limited SSc from 1979, suffering from dysphagia, pyrosis, epigastric pain, gastro-esophageal reflux, in 1998, underwent to distal esophagectomy for Barrett's oesophagus and, in 1999, to total gastrectomy with Roux y esophago-jejunal anastomosis. The stomach appeared very small, shrunken, its wall being stiff and increased in consistence. Samples were taken to study the ultrastructural modifications of the gastric wall and, in particular, the muscle coat.

Methods Samples of the gastric anterior wall from fundus, corpus and antrum, along the greater curvature, were obtained. The specimens were fixed in 4% glutaraldehyde in 0,1 M phosphate buffer, postfixed in 1% osmium tetroxide in 0,1M phosphate buffer and routinely processed for transmission electron microscopic observation. The ultrathin sections (1 µm) were stained with uranyl acetate and alkaline bismuth subnitrate and observed under Jeol 1010 electron microscope.

Results Wide focal areas of fibrosis, characterised by collagen fibres depositions, surrounding smooth muscle cells (SMC) were found. Many SMC were hypercontracted and/or showed a severe disarrangement of the myofilaments. Often, the dense bodies and the dense bands were thickened. Some SMC showed cytoplasmic vacuolisation and swollen mitochondria. Nerve bundles containing many axons were close to vessels and SMC. The axoplasm of nerve fibres were oedematous and poor in neurotubules and filaments, but the nerve endings close to SMC showed normal morphology, with the presence of synaptic vesicles, whose number was seldom decreased. Blood vessels were lined by intact endothelial cells, while the basement membrane appeared or thickened and laminated. The lumen was partially or completely occluded by erythrocytes and neutrophils. Inflammatory infiltrate as well, and many mast cells rich in granules or partially degranulated, were close to vessels and SMC.

Conclusion In the muscle coat of stomach focal fibrosis, severe ultrastructural modification of SCM and mild nerve fibres involvement are evident, without signs of endothelial damage.

REFERENCES

- 1 Russell LM, et al. Ultrastructure of the esophagus in scleroderma. *Arthritis Rheum.* 1982;**25**:1117-2
- 2 Malandrini A, et al. Autonomic nervous system and smooth muscle cell involvement in systemic sclerosis: ultrastructural study of three cases. *J Rheumatol.* 2000;**27**:1203-6

FRI0199 EVALUATION OF QUALITY OF LIFE IN PATIENTS WITH SYSTEMIC SCLEROSIS BY THE SF-36 QUESTIONNAIRE

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10.1136/annrheumdis-2001.280

Background

Objectives To evaluate the quality of life of patients affected by systemic sclerosis (SSc) through the application of SF-36 questionnaire and to correlate the results with the disability index of HAQ (DI) and the systemic involvement.

Methods We studied 100 (3 M, 97 F) patients affected by SSc (mean age 60 years, range 39-83, mean duration of disease 6 years, range 1-34). The organic involvement was evaluated by skin score, chest HRCT, electrocardiography according to Holter, Doppler-echocardiography and oesofagogram.

Results In the Table 1 are shown the results of the mean values of the 8 question groups in the control population and in the patients. The 8 question groups are: PA = physical activity, RP = role of physical activity, BP = bodily pain, GH = general health, VT = vitality, SA = social activities, EP = emotional problems, MH = mental health.

A very good correlation was found between the DI and all the scores of SF-36. The skin involvement showed a statistically significant correlation with both the DI ($r = 0.3304$; $p = 0.0008$) and the PA score of the SF-36 ($r = 0.2635$; $p = 0.0081$). No other statistically significant correlation was found between any of the SF-36 dimensions and involvement of a specific organ.

Abstract FRI0199 Table 1

	PA	RP	BP	GH	VT	SA	EP	MH
Mean score of the control population	84.46	78.21	73.67	65.22	61.89	77.43	76.16	66.59
Mean score of the patients	61.35	39.05	50.46	40.92	50.81	67.55	49.96	55.72
Difference%	27.36	50.07	31.51	37.26	17.90	12.76	34.15	16.32

Conclusion SF-36 showed to be a valid instrument to evaluate the quality of life and the disability of patients with SSc and it seems to correlate with extent of skin involvement.

REFERENCES

- 1 Apolone G, et al. Questionario sullo stato di salute SF-36; manuale d'uso e guida all'interpretazione dei risultati. Guerini e Associati. Milano, 1997
- 2 Poole JL, Steen VD. *Arthritis Care Res.* 1991;**4**:27-31
- 3 Ruof J, et al. *Rheumatology* 1999;**38**:535-42
- 4 Silman A, et al. *J Rheumatol.* 1998;**25**:79-83
- 5 Steen VD, Medsger TA. *Arthritis Rheum.* 1997;**40**:1984-91
- 6 Stewart AL, et al. *J Am Med Ass.* 1989;**262**:907-13
- 7 Stewart AL, et al. *Med Care* 1988;**26**:724-35
- 8 Ware JE, et al. *Med Care* 1992;**30**:473-83

FRI0200 ELECTROCARDIOGRAPHIC ALTERATIONS IN SYSTEMIC SCLEROSIS AND IN RHEUMATOID ARTHRITIS

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10.1136/annrheumdis-2001.281

Background Myocardial involvement is a common finding in patients with systemic sclerosis. It is also present in rheumatoid arthritis, but very rarely. Different alterations can be revealed by an electrocardiographic examination.

Objectives Aim of this study was to evaluate and to compare the frequency and type of electrocardiographic alteration in patients with systemic sclerosis and rheumatoid arthritis.

Methods In the study group of 40 pts with SSc (38 female, 2 male mean age $52,85 \pm 12,44$) and 22 pts with RA (20 female, 2 male; mean age $51,45 \pm 11,41$) clinical, laboratory and ECG examination were done. All the patients with SSc were divided into two subgroup: with diffuse (dSSc) and localised (lSSc) SSc. The electrocardiographic data of each of these subgroup were compared. The existence of the next ECG abnormalities were investigated: ventricular premature beats (VPB), supraventricular premature beats (SVPB), arrhythmia absoluta, left bundle branch block (LBBB), left anterior fascicular block (LAFB),

complete right bundle branch block (CRBBB), atrioventricular block (AV block), infarct or pseudoinfarct, ST depression.

Results Electrocardiographic alterations were found in 65% of SSc patients, but only in 18,19% of RA patients. The difference was significant ($p < 0,001$) Table 1. In the dSSc group, ECG alterations were found in 6 pts (15%) - not significant compared to the lSSc group (5 pts, 12,5%). $p = 0,220$. ST depression was found in 2 pts (5%) with dSSc, but only in 1 pts (4,5%) with RA. Infarct or pseudoinfarct were found in 9 pts (22,5%) with SSc (6 in dSSc, 3 in lSSc).

Abstract FRI0200 Table 1

	RA	%	SSc	%
VPB	3	13,64	10	25
SVPB	1	4,55	4	10
Ar. Abs.	-	-	3	7,5
LBBB	-	-	1	2,5
LAFB	-	-	2	5,0
CRBBB	-	-	3	7,5
AV Block	-	-	3	7,5

Electrocardiographic alterations in patients with SSc and RA.

Conclusion The results of our study showed a significant prevalence of ECG alterations in SSc, but low prevalence in RA. Differences between a presence of alterations in two subgroup (dSSc, lSSc) of SSc is not significant ($p = 0,220$).

FRI0201 CIRCULATING PROCOLLAGEN III AMINOPROPEPTIDE IN SYSTEMIC SCLEROSIS: RELATIONSHIP TO A RECENTLY VALIDATED DISEASE ACTIVITY INDEX

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10.1136/annrheumdis-2001.282

Background Systemic sclerosis (SSc) is characterised by excessive deposition of extracellular matrix components in the skin and in involved internal organs. Serum levels of procollagen III aminopropeptide (PIIINP), an index of collagen synthesis, have been reported as a possible marker of disease activity in SSc.¹

Objectives To study the relationship between a recently developed index of disease activity (2, 3) and serum concentrations of PIIINP.

Methods Nineteen SSc patients (17 F, 2 M; age ranging from 24 to 76, median 55.5 years; 13 lcSSc, 6 dcSSc; disease duration ranging from 2 to 34, median 12 years) consecutively admitted to 3 Italian tertiary centres and 19 sex and age-matched healthy controls were investigated, for serum levels of PIIINP, by radioimmunoassay.

For each SSc patient a recently validated activity index^{2,3} was calculated.

Results Increased serum concentrations of PIIINP were found in SSc compared with controls (4.59 ± 1.84 vs. 3.05 ± 0.85 micrograms/L; $p = 0.002$). Age, sex, subset and disease duration did not influence PIIINP serum levels.

The activity scores calculated in SSc patients ranged from 0.0 to 7.0, median 2.0. No significant correlations were registered between the PIIINP levels and the calculated activity index ($\rho = 0.04$; $p > 0.05$).

Conclusion This study demonstrates that elevated PIIINP serum levels found in SSc, even reflecting fibroblast activation, cannot be used as a surrogate for the measurement of disease activity as evaluated by a recently proposed index.

REFERENCES

- 1 Stevens, et al. *Br J Rheumatol*. 1992;**31**:683-89
- 2 Valentini, et al. *Arthritis Rheum*. 2000;**43**:S9-S392
- 3 Valentini, et al. *Ann Rheum Dis*. 2001, in press

FRI0202 LOW-DOSE ORAL CYCLOPHOSPHAMIDE IN THE TREATMENT OF "ACTIVE" SYSTEMIC SCLEROSIS: AN OPEN PROSPECTIVE 2-YEAR STUDY

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10.1136/annrheumdis-2001.283

Background Previous studies have reported cyclophosphamide (CYC) to be active in the treatment of fibrosing alveolitis in its early, inflammatory stages. Akesson *et al.*¹ demonstrated, in CYC treated SSc patients with alveolitis, an effect on inflammation parameters and skin thickening.

Objectives To test, by a prospective 2-years trial, the efficacy and safety of low-dose oral CYC in the treatment of "active" SSc whatever the disease manifestation underlying activity.

Methods SSc patients consecutively admitted were divided into 2 groups: group I with active disease (i.e. patients who had a new disease manifestation and/or a deterioration of a previously present one in the six months preceding the enrollment or who presented laboratory alterations indicating immune-inflammatory activity such as hypergammaglobulinemia and/or hypocomplementemia) and group II with inactive disease (i.e. patients with neither feature). Group I patients were treated with oral CYC (50 mg/daily) in addition to the previously administered symptomatic treatment; group II patients were only treated with symptomatic therapy. The following parameters (total skin score; ulcer healing; FVC; DLCO; EF; gammaglobulin levels; complement levels) and the severity scales recently proposed by Medsger *et al.*² were carried out at 12 and 24 months.

Results Out of the 45 recruited patients (42 F, 3M; aged 30 to 72 years, median age 49; 34 with lcSSc, 11 with dcSSc; disease duration ranging from 1 to 34, median 16 years) 22 were included in group I and 23 in group II. The study was discontinued for lack of efficacy in 5 out of the 19 group I patients and 8 out of the 19 group II patients who had at least the 12-months assessment ($p > 0.05$). No significant difference was detected between the 2 groups in any clinical, laboratory and instrumental parameters assessed, including Medsger's severity scales. C3 and C4 significantly increased in the group I patients after 12 and 24 months; ESR decreased in group I patients only after 12 months.

Conclusion Our results suggest that CYC might be able to contrast the evolution of disease in a significant percentage of SSc active patients. The role as a disease-modifying drug of SSc as a whole must be further investigated.

REFERENCES

- 1 Akesson, et al. *Arthritis Rheum*. 1994;**37**:729-35
- 2 Medsger TA, et al. *J Rheumatol*. 1999;**26**:2159-67