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Sjogren's Syndrome: Relationship Between Ocular Findings and Parotid Sialograms

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This study evaluates the incidence and severity of the ocular findings and its correlation with the parotid gland changes, as demonstrated by sialography, in 82 patients with Sjogren's syndrome. It is shown that the ocular component of the syndrome manifests rather autonomously with respect to the accompanying collagen disease and that there seems to be only a tentative trend towards a positive correlation between the ocular and parotid findings. Sjogren's syndrome immunologically is closer to the collagen diseases than to the so-called organ-specific autoimmune diseases.

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Sjogren's syndrome[1] consists of keratoconjunctivitis sicca and xerostomia associated with rheumatoid arthritis or other collagen disease.[2] This study is an attempt to evaluate the incidence and severity of ocular findings in a series of patients with this syndrome and to correlate these findings with parotid changes as demonstrated by sialography.

Materials and Methods

Eighty-two patients, all female, comprise the study group as shown in Table I. All were diagnosed as having Sjogren's syndrome in association with either rheumatoid arthritis, systemic lupus erythematosus, or progressive systemic sclerosis. Also included is a group of patients with keratoconjunctivitis sicca without other known involvement (Table I). All patients underwent a complete ophthalmologic examination including Schirmer's test and corneal examination under the biomicroscope employing cobalt light, after 2% fluorescein staining.[3] Secretory sialography was performed on each patient following the method of Rubin and Holt.[4]

Results

No relationship was found in rheumatoid patients between the degree of involvement, disease activity and lacrimal hyposecretion as measured by the Schirmer's test. Patients with systemic lupus ery-

Table I

CLINICAL GROUPS		NUMBER OF PATIENTS	PERCENTAGE
A (R.A.- classic or definite)	Aa ACTIVE	11	13.4
	Ai INACTIVE	12	14.6
B (R.A.- probable or possible)	Ba ACTIVE	3	3.7
	Bi INACTIVE	18	21.9
C (OTHERS)	C_1 S.L.E.	8	9.7
	C_2 P.S.S.	9	10.9
D (Keratoconjunctivitis sicca)		21	25.6

Table II

HYPOSECRETION	CLINICAL GROUPS						
	A		B		C		D
	Aa	Ai	Ba	Bi	C_1	C_2	
SLIGHT (8-10 mm/5')	0	1	0	1	0	0	0
MODERATE (4-7 mm/5')	2	2	1	4	2	5	6
SEVERE (0-3 mm/5')	9	9	1	13	6	4	15

Table III

SIALOGRAPHIC INVOLVEMENT	CLINICAL GROUPS						
	A		B		C		D
	Aa	Ai	Ba	Bi	C_1	C_2	
NORMAL	1	1	1	5	4	2	12
SLIGHT	2	6	1	2	0	2	4
MODERATE	8	4	0	9	2	5	3
SEVERE	0	1	1	2	2	0	2

SIALOGRAPHIC INVOLVEMENT:
NORMAL: Lack of sialectasia
SLIGHT: Punctate sialectasia
MODERATE: Globular sialectasia
SEVERE: Cavitary sialectasia

thematosus and progressive systemic sclerosis revealed moderate or severe hyposecretion. Approximately 75% of the patients with keratoconjunctivitis sicca alone demonstrated a marked reduction in tear formation (Table II).

Likewise, there was no correlation between clinical severity and activity of the collagen disorders with parotid sialographic changes. More than half of the patients with keratoconjunctivitis sicca without manifestations of collagen disease demonstrated normal sialograms (Table III).

Table IV reveals a lack of close parallelism between sialographic involvement and ocular signs in groups A, B and C. A somewhat better correlation was obtained in group D, where patients with a normal sialogram revealed only conjunctival irritation, whereas those with severe sialographic changes were associated with more advanced ocular changes (filamentary keratitis) (Table III).

Comments

Our findings demonstrate that both ocular and sialographic changes may progress independently of other disease signs in rheumatoid arthritis, systemic lupus erythematosus and progressive systemic sclerosis. The occurrence of sicca manifestation in SLE and PSS, with or without ocular complaints, has not been sufficiently emphasized.[5 – 8]

Table IV
Group A

SIALOGRAPHIC INVOLVEMENT	OCULAR CHANGES		
	SLIGHT	MODERATE	SEVERE
NORMAL	1	1	0
SLIGHT	2	3	3
MODERATE	1	6	5
SEVERE	0	1	0

GROUP A
(R. A .- classic or definite)

OCULAR CHANGES
Slight: Conjunctivitis
Moderate: Punctate Keratitis
Severe: Filamentary Keratitis

Table IV
Group B

SIALOGRAPHIC INVOLVEMENT	OCULAR CHANGES		
	SLIGHT	MODERATE	SEVERE
NORMAL	0	5	1
SLIGHT	0	1	2
MODERATE	2	3	4
SEVERE	1	0	2

GROUP B
(R.A.- probable or possible)

Table IV
Group C

SIALOGRAPHIC INVOLVEMENT	OCULAR CHANGES					
	SLIGHT	MODERATE	SEVERE	SLIGHT	MODERATE	SEVERE
NORMAL	3	1	0	2	0	0
SLIGHT	0	0	0	1	0	1
MODERATE	0	0	2	1	2	2
SEVERE	0	0	2	0	0	0
	GROUP C_1 (S.L.E.)			GROUP C_2 (P.S.S.)		

Table IV
Group D

SIALOGRAPHIC INVOLVEMENT	OCULAR CHANGES		
	SLIGHT	MODERATE	SEVERE
NORMAL	11	1	0
SLIGHT	1	1	2
MODERATE	1	0	2
SEVERE	0	0	2

GROUP D
(Keratoconjunctivitis sicca)

Collagen diseases are characterized by the presence of non-organ specific auto-antibodies in contrast to the immune responses seen in pernicious anemia, Hashimoto's thyroiditis, and idiopathic adrenal insufficiency. Auto-antibodies to thyroid, gastric and adrenal antigens is not greater in patients with Sjogren's syndrome than in controls.[9] Non-organ specific auto-antibodies (anti-ductal) are found more frequently in collagen disorders than in the more specific auto-immune diseases noted here.

It would seem that keratoconjunctivitis sicca more closely parallels collagen disorders in its type of immunologic response than it does diseases in which organ-specific antibodies are found. The reasons for these disparities are unknown. It is possible that in combined lacrimal-parotid disease, there is a common antigen, although either system may respond independently to a more specific antigen.

Conclusions

1. The ocular component in Sjogren's syndrome manifests somewhat autonomously with respect to the accompanying collagen disease.
2. There is no more than a tentative trend towards a positive correlation between ocular findings and parotid changes.
3. There appears to be a group of patients with isolated keratoconjunctivitis sicca who deserve further study.
4. Sjogren's syndrome is related immunologically to the collagen diseases and does not demonstrate the variety of organ-specific auto-antibodies seen in diseases such as pernicious anemia, Hashimoto's thyroiditis, and idiopathic adrenal insufficiency.

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