The oral component of Sjögren’s syndrome

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This study reports the results of an interdisciplinary approach to the diagnosis of Sjögren’s syndrome in 100 patients. Ocular and systemic diagnoses and oral features from the history and physical examination are correlated with measurements of stimulated parotid flow rate and labial salivary gland histopathology. A new diagnostic criterion is introduced whereby labial salivary gland focus scores are used to establish the presence of the oral component of Sjögren’s syndrome in place of the subjective evaluation of xerostomia. The differential diagnosis of the oral clinical features of Sjögren’s syndrome and the clinical management of the oral component of this disease are discussed.

Sjögren’s syndrome is a chronic inflammatory disease with multisystem involvement and diverse features. It is characterized by a clinical triad that includes dry eyes (keratoconjunctivitis sicca), dry mouth (xerostomia), and an associated connective-tissue disease. The usual criteria for the diagnosis of this syndrome are the presence of at least two of the three components of this triad.1:2 Objective diagnostic criteria are available and are routinely applied to evaluate eye and connective-tissue disease components of Sjögren’s syndrome.

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(SS), but diagnosis of the oral component is usually based on subjective criteria, such as the presence of xerostomia or the absence of pooled saliva in the floor of the mouth. The purpose of this prospective study was to apply selected objective oral criteria to patients suspected of having SS and to use these in establishing the diagnosis.

**SJÖGREN'S SYNDROME**

Approximately half of the patients with SS present a combination of dry eyes and dry mouth, without another connective-tissue disease (the “sicca complex”). The other half of the patients with SS have at least one of these sicca features associated with another connective-tissue disease, usually rheumatoid arthritis. Less commonly, systemic lupus erythematosus, scleroderma, polymyositis, or polyarteritis is present as the connective-tissue component of SS.

The characteristic histopathologic feature of SS is lymphoid cell infiltration and proliferation, potentially involving any organ system but most commonly affecting the lacrimal and salivary glands. In most patients this lymphoproliferation remains in a chronic benign state, but a progression to lymphoid neoplasm has been reported. The serum often shows hypergammaglobulinemia and autoantibodies, such as rheumatoid factor, antinuclear factor, and antissalivary duct antibody.

The principal oral component of SS is reduced production of saliva, resulting in chronic xerostomia. Enlargement of the parotid glands has been reported in approximately 30 per cent of two large groups of patients with SS.

**MATERIALS AND METHODS**

**Patients**

This article reports the results of evaluation of eighty-two female and eighteen male patients suspected of having Sjögren’s syndrome. Their ages ranged from 16 to 77 years, with a mean of 51 years. The patients were referred to an interdepartmental clinic at the University of California at San Francisco, by dentists, internists, and ophthalmologists in both private and institutional practices.

**Systemic evaluation**

Each patient had a complete medical history and physical examination. Complete blood count, determination of erythrocyte sedimentation rate, quantitative immunoglobulins, antinuclear antibody, anti-DNA antibody, rheumatoid factor, and hepatic and renal function studies were performed on all patients. A diagnosis of rheumatoid arthritis (RA) or systemic lupus erythematosus was based on established diagnostic criteria. Results of these evaluations will be presented in a subsequent report.

**Ophthalmologic evaluation**

The presence or absence of keratoconjunctivitis sicca (KCS) was established after a complete ophthalmologic evaluation, including slit lamp examination,
Oral component of Sjögren's syndrome

Schirmer test, Rose Bengal and fluorescein staining of the cornea and conjunctiva, and tear lysozyme determination. The signs considered to be diagnostic of KCS were diffuse punctate or blotchy epithelial staining in association with less than 10 mm. of wetting of the Schirmer strip in 5 minutes.

**Oral evaluation**

In the history, particular attention was paid to the following: dysphagia; glandular swelling; current medications, particularly those known to cause reduced salivary secretion; and changes in voice, dietary habits, and the senses of taste and smell.

The oral physical evaluation noted enlargement of major salivary glands, mucosal changes, and dental status. After the history and examination, stimulated parotid flow rate (PFR) was measured and labial salivary gland (LSG) biopsy was performed.

**Parotid flow rate**

Parotid saliva was collected by means of Carlson-Crittenden cups as modified by Sproles and Schaeffer. The patients were placed in a slightly reclined sitting position, and the collecting cups were placed bilaterally over the opening of Stenson's ducts. Saliva was collected over a 10-minute period, with stimulation applied for 5 seconds every 30 seconds for the full period. The gustatory stimulant used was a 1:1 dilution of commercial lemon juice (Real lemon, Borden, Inc.) applied with a cotton-tipped applicator to the lateral borders of the tongue. All collections were made on nonfasting subjects between 9:00 and 11:00 A.M. The lower limit of the normal range was considered to be 5 ml. per gland per 10 minutes, and all rates are expressed in these units. Standard deviations were not calculated for these values because the results of measurements of salivary flow on large groups of normal subjects have shown that they do not follow gaussian distribution.

**Labial salivary gland biopsy**

Biopsies were performed and assessed semiquantitatively on all patients in the series by means of techniques described previously. The results are expressed as the focus score, which is the number of foci of lymphoid cells per 4 square millimeters of gland section. If the criteria of Waterhouse and Doniach are applied, a focus is an aggregate of fifty or more lymphocytes, histiocytes, and plasma cells.

**Scintigraphy**

Sequential salivary scintigraphy with sodium pertechnetate Tc99m was performed on forty-eight patients. General statistical correlation was observed between scintigraphy scores and both PFR and LSG focus scores. However, there were many poorly correlated individual cases. The method was a less sensitive indicator in mild cases than were PFR or LSG biopsy focus scores. These data will be reported in a subsequent paper.
RESULTS

In the 100 patients examined, the stimulated parotid flow rate varied from 0 to 14 ml. per gland per 10 minutes, with a mean of 2.5 ml. For the majority of cases, reduced flow rate reflected the severity of the patient's complaint of dryness. LSG focus scores varied from 0 to 12. A score of 10 was the highest that could be accurately counted, but for the purpose of numerical analysis, a focus score of 12 was arbitrarily assigned to those specimens in which lymphoid infiltrates had become confluent. To rule out effects of obstruction or acute inflammation, areas of salivary gland which showed ductal dilation or infiltration by polymorphonuclear leukocytes were excluded in determination of the focus scores. Specimens from patients with chronic granulomatous diseases, leukemia, amyloidosis, lymphoma, a history of therapeutic radiation involving the biopsy site, or the presence of marked mucosal inflammation overlying the biopsy site were excluded from analysis. In our experience, these conditions may result in chronic inflammatory changes of labial salivary glands.

The patients were grouped according to the final ocular and systemic diagnoses and the results of the LSG biopsy and stimulated PFR. Six groups were established as follows:

**Group I: Sjögren's syndrome-sicca complex.** Included were forty patients who had KCS, and LSG biopsy scores greater than 1. None had evidence of another connective-tissue disease.

**Group II: Sjögren's syndrome with connective-tissue disease (not RA).** Included were eleven patients who had a connective-tissue disease (other than RA) and at least one of the sicca components. Ten patients had a LSG biopsy score greater than 1, and seven had KCS. Five of them had systemic lupus erythematosus, three had scleroderma, two had polymyositis, and one had mixed connective-tissue disease.

**Group III: Sjögren's syndrome with rheumatoid arthritis.** Included were fourteen patients who had RA and at least one of the sicca components. Eleven patients had a LSG biopsy score greater than 1, and eleven had KCS.

**Group IV: Possible Sjögren's syndrome.** Included were nineteen patients who did not fulfill the criteria for the diagnosis of SS, but presented evidence suggestive of that disease. Thirteen of these patients had KCS, with or without a reduced PFR but a LSG biopsy score of less than 1. Three patients had a connective-tissue disease and reduced PFR (0.6 to 1.8 ml.) but had no KCS and negative LSG biopsy findings. Three patients had LSG biopsy scores greater than 1 (1.1 to 2.5) and reduced PFR (0.8 to 1.6 ml.) but did not have KCS or a connective-tissue diseases.

**Group V: Connective-tissue disease only.** Included were six patients with rheumatoid arthritis, two with systemic lupus erythematosus, and one with scleroderma. None of them had KCS or positive LSG histology, and their PFRs were within normal limits (except one patient whose PFR was 3 ml.)

**Group VI: Miscellaneous systemic diseases with salivary involvement.** Included were six patients who presented initially with signs or symptoms suggestive of the sicca features of SS. The final diagnoses included sarcoidosis (two patients), malignant lymphoma, chronic myelogenous leukemia, amyloidosis, and
Table I. Clinical findings from 100 patients suspected of having Sjögren's syndrome

<table>
<thead>
<tr>
<th>Clinical groups</th>
<th>I</th>
<th>II</th>
<th>III</th>
<th>IV</th>
<th>V</th>
<th>VI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex (F/M)</td>
<td>40/0</td>
<td>11/0</td>
<td>6/8</td>
<td>15/4</td>
<td>7</td>
<td>3/3</td>
</tr>
<tr>
<td>Age (mean)</td>
<td>54.1</td>
<td>39.4</td>
<td>57.7</td>
<td>47.6</td>
<td>55.0</td>
<td>44.3</td>
</tr>
<tr>
<td>Age (range)</td>
<td>16-75</td>
<td>22-56</td>
<td>35-76</td>
<td>24-77</td>
<td>35-73</td>
<td>18-68</td>
</tr>
<tr>
<td>KCS present</td>
<td>40</td>
<td>7</td>
<td>11</td>
<td>13</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>RA present</td>
<td>0</td>
<td>0</td>
<td>14</td>
<td>2</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td>Other CTD*</td>
<td>0</td>
<td>11</td>
<td>0</td>
<td>1</td>
<td>4</td>
<td>0</td>
</tr>
</tbody>
</table>

*Diagnosis of other connective-tissue disease.

Table II. Parotid flow rate and labial biopsy scores from 100 patients suspected of having Sjögren's syndrome

<table>
<thead>
<tr>
<th>Clinical groups</th>
<th>I</th>
<th>II</th>
<th>III</th>
<th>IV</th>
<th>V</th>
<th>VI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean parotid flow rate (range)</td>
<td>1.2 (0.6-6)</td>
<td>3.5 (0.9-9)</td>
<td>3.6 (0.1-13)</td>
<td>2.7 (0.6-7.1)</td>
<td>0.5 (3.0-14)</td>
<td>0.3 (0.1-5)</td>
</tr>
<tr>
<td>Mean LSG biopsy score (range)</td>
<td>7.0 (1.7-12)</td>
<td>5.1 (0.9-12)</td>
<td>4.6 (0.1-10)</td>
<td>0.5 (0.1-2.5)</td>
<td>0.2 (0.0-5)</td>
<td>0.2 (0.0-5)</td>
</tr>
<tr>
<td>&quot;Positive&quot; biopsy*</td>
<td>0/40</td>
<td>10/11</td>
<td>11/14</td>
<td>3/19</td>
<td>0/10</td>
<td></td>
</tr>
</tbody>
</table>

*Patients with LSG focus scores > 1.
†Excluded from scoring. See results.

sideroblastic anemia associated with hemochromatosis (one patient each). The absence of KCS, characteristic infiltrative changes in the LSG biopsy, or systemic features of connective-tissue disease ruled out SS.

Immunosuppressive therapy with cytotoxic or corticosteroid drugs may affect the LSG histology or PFR in patients with SS. Use of these agents in the present groups was as follows: One patient in Group I was receiving 15 mg. of prednisone per day for biliary cirrhosis. Five patients in Group II were taking between 5 and 25 mg. of prednisone per day. Six patients in Group III were taking 2 to 40 mg. of prednisone per day, and one was receiving 50 mg. of cyclophosphamide per day. Three patients in Group V with RA were taking between 10 and 15 mg. of prednisone per day.

Table I summarizes sex, age at the time of evaluation, presence of connective-tissue disease, and eye involvement in the six groups. Eighty-eight per cent of the patients with definite SS (Groups I, II, and III) were females. All of the male patients with SS had RA as a component of their disease. Patients with definite SS had a mean age of 52.4 years, ranging from 16 to 76.

Table II lists the PFR and LSG biopsy focus scores of the six groups. The groups are arranged in order of decreasing LSG focus scores, with Group I being the highest and Group V the lowest. There was a general correlation within each group between the reduction of the PFR and the focus score.

Table III summarizes the oral symptoms and signs of the six groups. The early age of onset seen in Group II correlates with the generally earlier onset of systemic lupus erythematosus and scleroderma, as compared to the sicca complex
Table III. Oral features in 100 patients suspected of having Sjögren's syndrome

<table>
<thead>
<tr>
<th>Clinical groups</th>
<th>I</th>
<th>II</th>
<th>III</th>
<th>IV</th>
<th>V</th>
<th>VI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean duration of oral symptoms</td>
<td>4.5</td>
<td>4.3</td>
<td>2.9</td>
<td>4.5</td>
<td>0.6</td>
<td>0.7</td>
</tr>
<tr>
<td>Mean &quot;onset&quot; age*</td>
<td>49.6</td>
<td>35.1</td>
<td>54.8</td>
<td>43.1</td>
<td>52.4</td>
<td>43.6</td>
</tr>
<tr>
<td>Patients without oral symptoms</td>
<td>4(10%)</td>
<td>0(0%)</td>
<td>3(21%)</td>
<td>5(26%)</td>
<td>6(60%)</td>
<td>0(0%)</td>
</tr>
<tr>
<td>Salivary gland enlargement</td>
<td>17(43%)</td>
<td>5(45%)</td>
<td>2(14%)</td>
<td>5(26%)</td>
<td>0(0%)</td>
<td>2(33%)</td>
</tr>
<tr>
<td>Mucosal changes on dorsum of tongue</td>
<td>21(55%)</td>
<td>3(27%)</td>
<td>6(43%)</td>
<td>3(16%)</td>
<td>0(0%)</td>
<td>3(50%)</td>
</tr>
<tr>
<td>Cervical or incisal caries</td>
<td>17(63%)</td>
<td>5(56%)</td>
<td>4(36%)</td>
<td>3(19%)</td>
<td>0(0%)</td>
<td>2(50%)</td>
</tr>
<tr>
<td>Patients wearing complete dentures</td>
<td>13(33%)</td>
<td>2(18%)</td>
<td>3(21%)</td>
<td>3(16%)</td>
<td>4(40%)</td>
<td>1(17%)</td>
</tr>
</tbody>
</table>

*Mean age minus mean duration.
†Papillary atrophy, fissuring, erythema.
‡Percentage of patients with remaining natural teeth.

and RA. Eighty-two per cent of the patients in the series had a chief complaint of an oral problem that had been present for an average of 3.8 years prior to diagnosis. The eighteen patients without oral symptoms had a significantly higher mean PFR (5.6 ml., ranging from 1.4 to 14 ml.) than that of the total group. The most common specific oral complaints included: insufficient saliva, food sticking in the mouth, difficulty swallowing, inability to eat dry foods, need to wash down food with fluids, and avoidance of dry foods. Other patients experienced hoarseness or deepening of the voice, an altered sense of taste or smell, and an increase in dental caries. The latter followed the onset of their oral symptoms by 2 years or more. The intensity and diversity of symptoms tended to correlate with the reduction in PFR. Many of the patients with SS described a cyclical course for their oral symptoms, with periods of progression and remission taking place.

Major salivary gland enlargement was present in twenty-four (37 per cent) of the patients with SS: nineteen, parotid; one, submandibular; and four, both parotid and submandibular. Salivary gland enlargement occurred more frequently in Groups I and II than in Group III. Forty-eight per cent of the patients with definite SS described episodes of recurrent enlargement in the region of the parotid or submandibular glands. These episodes of swelling lasted from a few weeks to many months. Chronic suppurative parotitis was present in only two patients, both in Group I. Patients presenting with mucosal changes on the dorsal aspect of the tongue, including papillary atrophy, fissuring, and erythema, were seen in all groups except Group V. The mean PFR of this group of thirty-six patients was 0.9 ml., ranging from 0 to 5.5 ml. Dental caries, located predominantly in the cervical or incisal regions, was present in thirty-one of the patients (43 per cent of those with remaining natural teeth). The mean PFR of these patients was 1.1 ml., ranging from 0 to 5.3 ml.

We modified the usual diagnostic criteria for SS in this study. Subjective xerostomia was not accepted as one of the diagnostic triad for SS. Instead, the
oral component of SS was considered to be present when the LSG biopsy specimens had (after appropriate exclusions) a focus score greater than 1. Seven patients in Groups I and III had “positive” LSG biopsies but did not have symptoms of xerostomia (Table III). However, their mean LSG biopsy focus score was 3.2, ranging from 2 to 7, and they all had KCS, or KCS and RA, to complete the criteria for SS. The combined oral, ophthalmologic, and systemic evaluations clearly indicated the presence of SS in these seven patients, yet the diagnosis could only be made through the use of our new criteria.

Sixteen patients (thirteen in Group IV, and three in Group V) complained of a “dry mouth” and also had KCS or a connective-tissue disease. By the subjective xerostomia criterion these patients would have had a diagnosis of SS, but by our criteria, they are considered to have only “possible SS” or connective-tissue disease without SS. In these cases the histologic criterion is more exclusive than the subjective one. However, the combined oral, ophthalmologic, and systemic evaluations for these sixteen patients provided only equivocal evidence to support the diagnosis of SS for the Group IV patients, and ruled out the diagnosis in Group V.

The patients in Group IV, with possible SS, have less severe symptoms and signs than those patients in Groups I, II, and III. Although an attractive hypothesis is that these patients represent cases of early SS, this is not supported by the duration of their oral symptoms (Table III). It is possible that these patients will develop the features of definite SS at some time in the future, but they may simply have a milder form, or they may not have SS at all.

**DISCUSSION**

**Clinical features and differential diagnosis**

Most of the patients with SS complain of symptoms related to xerostomia. These may be the first symptoms that the patient experiences, and, if appropriately investigated, may lead to the diagnosis of the condition. Presenting complaints, although important, should not be considered the sole criteria for the presence of the oral component of SS. Patients may have no complaint even after direct questioning yet have a clearly abnormal PFR and LSG biopsy. Conversely, patients may complain of oral dryness when their oral examination, PFR, and LSG biopsy specimens are within normal limits.

The wide range of PFRs seen in each group of patients (Table II) reflects the wide range of flow rates in normal individuals. A PFR above the low range of “normal” for patients with SS may have several explanations. A patient with early or mild SS may have a significant reduction in total saliva production and still have a PFR greater than 5 ml. The submandibular glands are responsible for the largest proportion of total production of saliva. Impairment of submandibular function may be greater than that of parotid, however, and not be reflected by the PFR. Qualitative changes have been described in the saliva of patients with SS, and these may have a role in the patient’s sensation of oral dryness. Finally, a patient’s evaluation may take place at a time when his symptoms are in a cycle of relative remission.

A differential diagnosis should always be considered when there are oral
findings suggestive of SS. Chronic xerostomia can result from the use of parasympatholytic drugs, radiation therapy to the head and neck, diabetes mellitus, chronic granulomatous disease, severe malnutrition, and leukemia.15 The oral mucosal changes and the distribution of dental caries reported in this series are characteristics of chronic xerostomia,16 not necessarily of SS. Enlargement of the parotid glands, common in SS, may also be caused by acute sialadenitis, ductal obstruction, virus infections, chronic granulomatous diseases, allergic reactions, diabetes mellitus, alcoholism with liver disease, hyperlipoproteinaemia, lymphoma, and solitary neoplasms.4

Role of histopathology in the diagnosis of Sjögren's syndrome

A major component of Sjögren's syndrome is salivary gland involvement with characteristic histopathologic changes.1-4 Therefore, salivary gland biopsy should play an important role in the diagnosis of this disease. Parotid gland biopsy has been performed on many patients with SS who have parotid enlargement, in order to provide histologic confirmation of the diagnosis. However, when one considers the difficulty and hazards of major salivary gland biopsy, the need for an alternate source of tissue is apparent. Involvement of minor salivary glands in postmortem SS was first described by Sjögren himself,22 but it was not until 1966, that buccal23 and palatal24 glands were biopsied in patients with SS. Several studies have applied qualitative25-27 or semiquantitative28,29 histologic grading systems to series of accessory salivary gland specimens. Several reports on series of postmortem labial salivary gland specimens have shown that, after certain conditions have been excluded, focal lymphocytic sialadenitis does not occur normally.30 All the studies report a high incidence of focal lymphocytic sialadenitis in those patients with SS. However, none used salivary histology in establishing the presence of the oral components of SS. Four studies also reported focal salivary gland infiltrates in connective-tissue diseases other than SS.25,26,28,29 Two of these reported LSG focus scores for groups of ten patients with RA. Chisholm and Mason28 reported a mean focus score of 0.4, with no individual score greater than 1. Davies and associates29 found a mean focus of 1.5, but their range was not reported. Data from the other studies cannot be compared because qualitative histologic criteria were used.25,26 The oral component of SS was excluded from these four groups of patients with connective-tissue disease by subjective criteria only.

Since SS is part of an apparent spectrum of connective-tissue diseases,2-4 it is not unreasonable to expect an overlapping in the clinical features of the various separate diseases. When these conditions are better understood, we may not be able to categorize them separately as we do now. Until then, however, objective criteria are necessary to make the most accurate diagnosis possible in these patients and to form a basis for communication in clinical studies of the connective-tissue diseases. To this end, we believe that a focus score determination from LSG tissue is preferable to subjective evaluation of xerostomia as a diagnostic criterion for the oral component of SS.

The LSG biopsy can also be useful in the diagnosis of other disease pro-
cesses. Three of the Group VI patients had no diagnosis at the time at which they were evaluated for SS. The histopathologic features of their LSG specimens contributed to final diagnoses of sarcoidosis, amyloidosis, and hemochromatosis by demonstrating, respectively, noncaseating granuloma, Congo red staining for amyloid with characteristic birefringence, and intraglandular deposition of iron.

**Clinical management**

The early diagnosis of SS may relieve a patient's anxiety about the source of his diverse symptoms, encourage application of therapeutic measures early enough to prevent dental destruction and corneal scarring, and provide a basis for the diagnosis of systemic complications and associated connective-tissue diseases. A young patient presenting with SS should be carefully followed for possible development of a connective-tissue disease, particularly systemic lupus erythematosus.

Often the most distressing symptoms experienced by patients with SS are those associated with their chronic xerostomia. The clinical management of these problems consists principally of symptomatic relief and prevention of dental destruction. Pilocarpine hydrochloride administered orally to some of our patients with SS led to transient improvement of their mouth symptoms; however, the majority of them complained of unacceptable side effects from the drug before symptomatic relief was achieved. Sialogogues, such as sour, sugarless candies, provided symptomatic relief fairly consistently. Frequent sips of water were helpful, but patients were advised that it is neither necessary nor helpful to swallow all the water. Many patients initially complained of urinary frequency and loss of sleep from nocturia. These symptoms were caused by the ingestion of large quantities of water and were relieved by having the patients rinse with, rather than drink, the water. The use of complete dentures can be both difficult and uncomfortable in the presence of chronic xerostomia. Some patients were able to wear their complete dentures more comfortably and effectively by using 2 per cent methyl cellulose or glycerine as an oral lubricant.

The rapid destruction of natural teeth in patients with chronic xerostomia can be eliminated or greatly reduced by a program of plaque control, daily oral fluoride rinses, and frequent follow-up, including dental hygiene, plaque control reinforcement, and restoration of carious lesions.

Oral candidiasis commonly occurs with chronic xerostomia. Its usual symptom is a burning sensation involving portions or all of the oral mucosa. It commonly occurs in patients with removable dental appliances. Colonies of these organisms may be apparent on the mucosa as white-creamy plaques, but mucosal erythema may be the only sign. An effective therapy is the use of nystatin vaginal tablets (100,000 units) which are dissolved slowly in the mouth, two to four times a day for a minimum of 10 days.

**CONCLUSION**

There are no established objective criteria for determining the presence of the oral component of SS. It is our opinion that subjectively evaluated "xero-
stomia" is not sufficient evidence of the presence of salivary gland involvement in SS. Evaluation of patients suspected of having SS should include standardized estimation of salivary flow rate and an accessory salivary gland biopsy with determination of a semiquantitative focus score. This will provide objective confirmation for the presence of the oral component of this disease.

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REFERENCES


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