Sweat gland function in Sjögren’s syndrome

Sir, Although it is generally agreed that the skin dryness associated with Sjögren’s syndrome (SS) is caused by decreased secretion from chronically inflamed sweat glands,1 sweat output in patients with SS has not been fully investigated. In the only study specifically to address this issue Bloch et al2 reported normal sweat production and a normal sweat sodium content in each of the seven patients they tested with pilocarpine iontophoresis. However, in presenting their findings, these authors neither described their study design nor recorded the ages and symptomatology of their study population.

To investigate further whether diminished sweat output is indeed a feature of SS and to assess whether alterations in sweat sodium concentrations are an additional feature of the condition we studied 12 patients (nine women, three men) with SS of one to 13 years’ duration. Their ages ranged from 28 to 65 years. Of the 12, three had the sicca complex alone, while the remaining nine had associated connective tissue disease (rheumatoid arthritis in three and SLE in six). Eight of the 12 reported skin dryness as a specific, troublesome symptom of their disease.

Since sweat output is known to diminish with advancing age and to differ between the sexes,3 we matched each patient for age and sex with a normal control. Each subject in the study was referred for pilocarpine iontophoresis, which was carried out according to a standardised method recommended by Schwartz et al.4 After applying a 3 × 3 cm square of filter paper of known weight to the iontophoresed area for one hour the paper square was reweighed. The difference between these two weights provided a measure of sweat production, expressed in milligrams. Sweat sodium concentrations were then calculated.4

By means of a two tailed unpaired t test at the 0·05 significance level we were unable to show a statistically significant difference between the mean sweat weights of the group with SS and their matched controls (Table 1). Moreover, the mean sweat sodium concentrations for both patients and controls fell within the normal range (<65 mmol/l) and there was no statistically significant difference between the two groups. When we compared the eight patients who specifically complained of skin dryness with the four who did not there was again no detectable difference in either sweat weights or sodium concentrations.

Although our results suggest that there is neither a measurable diminution in sweat output nor a change in sweat sodium concentration in patients with SS, it is possible that we have missed a statistically significant difference between patients and controls because of our small sample size.5 It is also possible that our method of pilocarpine iontophoresis, although standardised and universally accepted for use in adults with cystic fibrosis, may be insensitive to small changes in the sweat secretions and sodium concentrations of patients with SS. Nonetheless, our findings do seem to cast some doubt on the generally

Table 1 Sweat weights and sweat sodium concentrations in 12 patients with Sjögren’s syndrome and their age and sex matched controls

<table>
<thead>
<tr>
<th></th>
<th>Mean (standard deviation)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Patients</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sweat weight (mg)</td>
<td>0·33 (0·12)</td>
<td></td>
</tr>
<tr>
<td>Sweat sodium</td>
<td></td>
<td></td>
</tr>
<tr>
<td>concentration (mmol/l)</td>
<td>48·71 (17·10)</td>
<td></td>
</tr>
<tr>
<td><strong>Controls</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sweat weight (mg)</td>
<td>0·30 (0·14)</td>
<td>NS*</td>
</tr>
<tr>
<td>Sweat sodium</td>
<td></td>
<td></td>
</tr>
<tr>
<td>concentration (mmol/l)</td>
<td>61·61 (14·11)</td>
<td>NS*</td>
</tr>
</tbody>
</table>

*Not significant at 0·05 significance level.
Tissue typing in brucellosis

Sir, We read with interest the article by Dawes and Ghosh on tissue typing in brucellosis.1 The lack of association between reactive arthritis due to acute B abortus infection and HLA antigens, reported by them, confirms our observations in patients infected by B melitensis.2 In order to define the genetics of this condition we have recently performed HLA typing in families with two or more members affected by brucellosis3 and in patients with brucellar spondylitis.4 None of these studies showed linkage between brucellosis caused by B melitensis, with or without arthritis, and the HLA system.

The increased frequency of A2 in brucellosis patients, observed by Dawes and Ghosh1, and of B27 in brucellar spondylitis, reported by Hodinka et al.,5 are in contrast with our findings but might be explained by the different antigenic structures of B abortus and B melitensis6 and/or the genetic background of the populations studied.

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References

Book reviews


This book is the fifth in a series aimed at satisfying a need perceived by the editors for a series of textbooks devoted to subspecialties within dermatology, and is aimed ambitiously both at the clinician and at the researcher.

The book is undoubtedly comprehensive, although I found the order in which the sections appear a little baffling: I had always been taught to think of the epidemiology of a disease before the clinical features, but perhaps that is a personal quirk. As such, the information contained within the book is potentially invaluable—the problem is finding it. I tested the index by listing 10 items I felt to be relevant: streptococcus, arthritis mutilans, sarcoiditiis, geographic tongue, and alopecia as ‘clinical’ topics, and phosphodiesterase, calmodulin, T lymphocytes, extracorporeal photochemotherapy, and epidermal differentiation as ‘research’ topics. Only two of these items are listed in the index at all! This makes it impossible to recommend the book as a reference source, which is unfortunate because reading through it is interesting, rewarding, and at times provocative.

As always with edited collections of contributions from many authors the standard varies. It seems odd that references are listed in at least three different styles, and it is very rare that any reference later than 1983 is found. I suspect the book has been overlong in gestation. In general