Myxoedema and Raynaud's phenomenon

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Abstract. Cold hands is a common complaint in myxoedema and were reported by 15 of 17 patients before treatment of myxoedema with L-thyroxine. An increased cold sensitivity of the digital arteries could be demonstrated during finger cooling in about half of the patients and Raynaud's phenomenon was verified in the 4 patients as digital arterial closure. The cold sensitivity decreased significantly after treatment with L-thyroxine. The increased cold sensitivity in untreated myxoedema is probably due to an increased sympathetic discharge to the hands that might disclose a predisposition for Raynaud's phenomenon.

Patients with myxoedema have cold, dry and pale hands. This might be explained by the lowered basal metabolic rate and the decrease in the cardiac output (Graettinger et al. 1958). Measurement of the noradrenaline concentration in venous plasma has shown a threefold increase in myxoedema which has been taken to indicate an increased sympathetic discharge to the skin (Christensen 1973). We asked a consecutive group of patients with myxoedema about hand symptoms in cold and performed an objective test of digital cold sensitivity in order to demonstrate the frequency of Raynaud's phenomenon. The patients were investigated before and during treatment with L-thyroxine.

Patients and Methods

Seventeen patients with classical myxoedema were referred to investigation after having given an informed consent. There were 14 females and 3 men as it appears from Table 1. Ten normal subjects participated voluntarily as control subjects. Their age and sex distribution was comparable to the patient group, none had metabolic disorders or complaints from the hands.

A medical interview and a cold provocation test was performed in the morning in the outpatient clinic. The patients were dressed in their normal indoor clothing and their body was not cooled. They were recumbent for 30 min before the cold provocation started. This test was performed with a blood pressure cuff placed on the midphalanx of the left hand (Nielsen 1978). The midphalanx was cooled with the cuff to 30, 15 and 10°C during ischaemia, and the response to cooling to 15 or 10°C was recorded as the decrease in the indirectly measured finger systolic blood pressure. The response expressed in per cent of the pressure measured at 30°C indicates a change in the re-opening pressure of the digital arteries in the finger. This technique also allowed us to measure the finger systolic pressure where a decrease can be taken as either vasospasm or rather severe oblitative arterial disease (Dige-Petersen et al. 1977). The measurements were repeated after L-thyroxine treatment for a mean period of 9 months.

The variation in the cold provocation test is about 20 per cent (Nielsen 1978). The standard technique implicates body cooling to enhance the sympathetic nervous discharge to the hands. This was not performed in untreated myxoedema, but in the 10 control subjects and in 10 outclinic-patients treated for myxoedema. The
Table 1.
Clinical data in 17 patients with myxoedema.

<table>
<thead>
<tr>
<th>Group</th>
<th>No.</th>
<th>Sex</th>
<th>Age (range) years</th>
<th>Cold hands No.</th>
<th>Raynaud's phenomenon No.</th>
<th>Serum thyroxine µg/100 ml</th>
<th>Serum triiodothyronine µg/100 ml</th>
<th>Triiodothyronine binding</th>
<th>Serum thyrotrophin µU/ml</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myxoedema untreated</td>
<td>17</td>
<td>14:3</td>
<td>59 (26–76)</td>
<td>15</td>
<td>4</td>
<td>2.7 (1.4)</td>
<td>48 (31)</td>
<td>0.7 (0.1)</td>
<td>41 (20)</td>
</tr>
<tr>
<td>L-thyroxine</td>
<td>2</td>
<td>1</td>
<td>55</td>
<td>2</td>
<td>1</td>
<td>8.7 (1.7)</td>
<td>117 (24)</td>
<td>1.1 (0.1)</td>
<td>1.8 (2.8)</td>
</tr>
<tr>
<td>Normals*</td>
<td>10</td>
<td>8:2</td>
<td>55 (25–70)</td>
<td>2</td>
<td>0</td>
<td>4.3–10</td>
<td>106–182</td>
<td>0.8–1.2</td>
<td>&lt; 3.0</td>
</tr>
</tbody>
</table>

* The thyroid parameters are normal values from the laboratory.

response to finger cooling in these groups were not different neither before nor after body cooling for 15 min with a blanket perfused with water at 10°C. Statistical evaluation was performed with non-parametric tests.

Results

The mean values and standard deviations for arm systolic blood pressure, finger systolic blood pressure and pulse rate in the normal group and the patients with myxoedema are given in Table 2. The pulse rate showed a slight, but significant increase during treatment with L-thyroxine. The pressure gradient from arm to finger did not change, and the gradient was not different from the normal subjects.

The response of finger arteries at 10°C was abnormal in 8 untreated patients, and 4 with complaints of Raynaud's phenomenon showed complete digital arterial closure (Fig. 1). During treatment the cold response improved significantly ($P < 0.02$, Wilcoxon test of paired differences), and only one patient showed complete closure of the arteries during cooling.

Discussion

The frequency of cold hands in untreated myxoedema was 88% (64–99%) which is higher than in comparable normal groups (Graettinger et al. 1958). The appearance of cold hands can be explained from the decrease in heat production causing peripheral vasoconstriction to preserve body temperature. The blood flow in the extremities of patients with myxoedema is decreased (Stewart & Evans 1942), as is the skin temperature. These symptoms improve during treatment. The

<table>
<thead>
<tr>
<th>Group</th>
<th>Arm systolic pressure mmHg</th>
<th>Finger systolic pressure mmHg</th>
<th>Pressure* index</th>
<th>Pulse rate beats/min</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myxoedema untreated</td>
<td>132 (14.3)</td>
<td>123 (16.8)</td>
<td>0.93 (0.09)</td>
<td>60 (8.2)</td>
</tr>
<tr>
<td>L-thyroxine</td>
<td>135 (20.6)</td>
<td>121 (22.2)</td>
<td>0.90 (0.08)</td>
<td>63 (8.4)</td>
</tr>
<tr>
<td>Normals</td>
<td>122 (7.2)</td>
<td>101 (11.4)</td>
<td>0.96 (0.06)</td>
<td>70 (6.3)</td>
</tr>
</tbody>
</table>

* Ratio finger: arm systolic pressure.
increased noradrenaline concentration found in patients with myxoedema (Christensen 1973), could therefore be explained by an increased sympathetic discharge to the skin vessels. The cardiac response to sympathetic stimulation is normal in patients with myxoedema as is the increase in noradrenaline concentration and pulse rate during shift from supine to standing position (McDevitt et al. 1978; Christensen 1972). This indicates that the sensitivity of the cardiovascular system to noradrenaline is normal in untreated myxoedema (Landsberg 1977). It is therefore interesting that Raynaud’s phenomenon appears in 24% (7–50%) of patients with myxoedema. The frequency is not higher than could be expected from a young female population (Nielsen 1978). The improvement during L-thyroxine treatment might indicate that the increased sympathetic discharge elicits the phenomenon in predisposed females. An increased stiffness of the artery or intimal thickening in myxoedema is not likely to cause the phenomenon as the finger systolic blood pressure was normal and the arm to finger systolic pressure gradient did not change during treatment (Dige-Petersen et al. 1977). It has been proposed to treat patients with Raynaud’s phenomenon with thyroxine and reserpine to increase the heat production and to alleviate the sympathetic tone in these patients (Peacock 1960). From our findings with treatment of myxoedema such an attempt might appear promising, but controlled blind studies have to be performed.

References


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