Table I. Population and results

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Age (y)</th>
<th>Sex</th>
<th>Phototype</th>
<th>MED mg/cm²</th>
<th>Duration of disease (y)</th>
<th>Past treatments (%)</th>
<th>Type of AA</th>
<th>Onset of hair regrowth</th>
<th>Total of sessions</th>
<th>Cumulative doses</th>
<th>Tolerance</th>
<th>Hair regrowth 3 months after end of treatment</th>
<th>Assessment by the patient</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>32</td>
<td>F</td>
<td>IV</td>
<td>400</td>
<td>1</td>
<td>Intralesional steroid (100%)</td>
<td>AAP</td>
<td>Session no. 4</td>
<td>18</td>
<td>5.2 J/cm²</td>
<td>10/10</td>
<td>5</td>
<td>Excellent</td>
</tr>
<tr>
<td>2</td>
<td>38</td>
<td>F</td>
<td>IV</td>
<td>300</td>
<td>11</td>
<td>Minoxidil 5% (0%); intralesional steroid (0%); UVB (15%)</td>
<td>AAP</td>
<td>Session no. 6</td>
<td>24</td>
<td>7.5 J/cm²</td>
<td>10/10</td>
<td>3</td>
<td>Moderate</td>
</tr>
<tr>
<td>3</td>
<td>12</td>
<td>M</td>
<td>III</td>
<td>220</td>
<td>3</td>
<td>Minoxidil 5% (0%)</td>
<td>AAP</td>
<td>Session no. 7</td>
<td>24</td>
<td>4.8 J/cm²</td>
<td>10/10</td>
<td>4</td>
<td>Excellent</td>
</tr>
<tr>
<td>4</td>
<td>38</td>
<td>F</td>
<td>III</td>
<td>220</td>
<td>10</td>
<td>None</td>
<td>AAU</td>
<td>0</td>
<td>24</td>
<td>6.1 J/cm²</td>
<td>10/10</td>
<td>0</td>
<td>Poor</td>
</tr>
<tr>
<td>5</td>
<td>42</td>
<td>F</td>
<td>III</td>
<td>400</td>
<td>2</td>
<td>Topical steroid (0%); minoxidil 5% (0%); systemic steroid (0%)</td>
<td>AAU</td>
<td>0</td>
<td>24</td>
<td>15.5 J/cm²</td>
<td>10/10</td>
<td>0</td>
<td>Poor</td>
</tr>
<tr>
<td>6</td>
<td>56</td>
<td>W</td>
<td>III</td>
<td>550</td>
<td>4</td>
<td>Minoxidil 5% (0%); systemic steroid (0%)</td>
<td>AAT</td>
<td>0</td>
<td>24</td>
<td>10.8 J/cm²</td>
<td>9/10</td>
<td>0</td>
<td>Poor</td>
</tr>
<tr>
<td>7</td>
<td>45</td>
<td>F</td>
<td>III</td>
<td>400</td>
<td>3</td>
<td>Topical steroid (0%); intralesional steroid (0%)</td>
<td>AAU</td>
<td>0</td>
<td>24</td>
<td>9.5 J/cm²</td>
<td>9/10</td>
<td>0</td>
<td>Poor</td>
</tr>
<tr>
<td>8</td>
<td>47</td>
<td>F</td>
<td>II</td>
<td>400</td>
<td>5</td>
<td>Minoxidil 5% (0%); systemic steroid (0%); Psoralen-UVA (0%)</td>
<td>AAP</td>
<td>Session no. 15</td>
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<td>16.8 J/cm²</td>
<td>10/10</td>
<td>4</td>
<td>Excellent</td>
</tr>
<tr>
<td>9</td>
<td>29</td>
<td>M</td>
<td>III</td>
<td>300</td>
<td>1</td>
<td>Minoxidil 5% (0%); systemic steroid (0%)</td>
<td>AAP</td>
<td>Session no. 6</td>
<td>12</td>
<td>3.9 J/cm²</td>
<td>10/10</td>
<td>5</td>
<td>Excellent</td>
</tr>
</tbody>
</table>

AA, Alopecia areata; F, female; M, male; MED, minimal erythema dose.

Hair regrowth: 0 = no hair regrowth, 1 = hair regrowth 1% to 24%, 2 = hair regrowth 25% to 49%, 3 = hair regrowth 50% to 74%, 4 = hair regrowth 75% to 99%, and 5 = complete hair regrowth.


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Amyopathic dermatomyositis presenting during pregnancy

To the Editor: Amyopathic dermatomyositis (ADM) presents with pathognomonic cutaneous manifestation of dermatomyositis (DM) but without associated skeletal muscle involvement. ADM associated with pregnancy has not been described.

A 34-year-old woman in the third month of pregnancy presented with facial erythema and pruritic papules on the dorsal hands, elbows, and knees. The rash had appeared at 4 weeks’ gestation. She had not taken any medicine before the rash appeared. She did not complain of muscle pain or weakness. In her first pregnancy, she delivered a healthy baby and had had no cutaneous complications. There was no personal or family history of connective tissue disease.

Examination revealed discrete pruritic red-purple papules over the bony prominences. Periungual telangiectasia was also observed. She had exudative erythema bridging the nose (Fig 1). A skin biopsy specimen from her elbow was compatible with DM (Fig 2). Results of complete blood counts, blood biochemistry analysis, and urinalysis were within normal limits. Although antinuclear antibody titer (1:40) was positive in a nucleolar pattern, other autoantibodies were negative. Based on these findings, this patient was diagnosed with ADM. Potent topical steroids were prescribed but the eruption did not improve. She delivered a healthy
newborn at term and the rash began to disappear immediately after delivery. Complete resolution came 2 weeks later.

Reports of DM/polymyositis- (PM) complicated pregnancy are rare. Some authors suggest that the outlook for the fetus is unfavorable when DM is first diagnosed during pregnancy.1 Others consider that fetal prognosis parallels the activity of maternal disease.2 Recent cases have tended to show a good outcome, probably because treatment with oral corticosteroids was introduced. In our case, we administered steroid ointment only; however, both mother and fetus were doing well in spite of ADM, which occurred at 4 weeks of gestation. Although the prognosis of ADM in comparison to DM/PM has not been well clarified, our case suggests that ADM may be a milder subtype of DM/PM.

Various factors have been considered as triggers for development of DM/PM during pregnancy; for example, exposure of the mother to fetal antigens,3 changes in maternal hormonal status,4 and the reactivation of certain viruses by pregnancy.5 Recently it has been proposed that microchimerism may contribute to the pathogenesis of autoimmune diseases. Microchimerism is defined by the presence within an individual of a low level of cells derived from a different individual. The main, natural source of microchimerism is pregnancy. Bidirectional cell trafficking between a mother and fetus during pregnancy has become an accepted fact. Microchimeric cells are reacting toward the host in the manner of chronic graft-versus-host disease and may

**Fig 1.** Discrete pruritic red-purple papules, known as Gottorin’s papules, were seen on the elbow (a) and knuckles (b). Exudative erythema was found bridging the nose (c).

**Fig 2.** Hyperkeratosis and slight atrophy of the epidermis along with focal vacuolization were observed at the dermo-epidermal junction. The upper dermis showed mononuclear inflammatory infiltration around the vessels. (Hematoxylin-eosin stain; original magnification: ×100.)
lead to the development of autoimmune disease. Some investigators propose that cutaneous disorders occurring during pregnancy are associated with microchimerism. Although we did not perform typing of human lymphocyte antigens, the hypothesis that microchimerism played an important part in our case is very plausible.

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Luxation of the eyeball

To the Editor: We would like to report an unusual and memorable event in a recent patient with sarcoidosis.

A 45-year-old African American man with an established diagnosis of sarcoidosis was sent for evaluation of his skin findings. When I (N. A. L.) examined the patient, he was noted to have multiple red-brown plaques on his scalp and on the posterior part of his neck. While retracting his upper eyelid to check the lacrimal gland in his left eye, his entire globe immediately protruded beyond the eyelids which closed behind the eye.

With the patient in obvious pain, I instructed him to lie down and firmly pushed the globe back into the socket. Other than the patient experiencing pain that lasted for the entire day, he had no other sequelae.

Over the past 100 years, fewer than 30 cases of spontaneous luxation of the globe have been reported in the literature. Most of the cases have occurred during lid manipulation during an ophthalmologic examination. The luxation occurs when the equator of the globe protrudes beyond the retracted lids. This situation has also been reported in a patient attempting to place a contact lens in his eye. In most cases, the eyeball can be pushed back into the socket by the physician pressing on the sclera, although some cases may require sedation.

Other than pain, there is usually no damage to the eye in patients who experience luxation. Corneal abrasions and exposure keratitis can occur if the eyeball remains outside of the socket for an extended period of time.

Subluxation (luxation) of the globe usually occurs in patients with a combination of an anterior situated globe, shallow orbital sockets, and somewhat lax extraocular muscles. Risk factors include exophthalmos, which can be secondary to space-occupying orbital pathology, such as excessive orbital fat, sarcoidosis, Graves’ disease, or orbital tumors. In patients who experience repeated episodes of subluxation, surgery may be necessary in order to prevent recurrences.

In conclusion, use caution when examining lacrimal glands in patients with proptosis, unless you are willing to experience an eye-opening event.

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