Scalp dermoscopy or trichoscopy is a useful noninvasive adjunct to the pediatric scalp examination that allows the rapid capture of high-resolution images for in vivo evaluation. We describe a novel normal scalp finding in children and document the presence of dermoscopic patterns previously described in adults.

Dirty dots, which present on the scalp as clumped and haphazardly arrayed particulate debris and loose fibers of various colors, were most commonly found in the intermediate age group (83% of those aged 9 months to 10 years; 10 of 12), and not at all in younger or older age groups. Two intermediate-aged subjects reevaluated 3 years after their initial examination demonstrated an improvement with age. The experience of 1 of us (A.T.) suggests that dirty dots are rarely if ever seen on the adult scalp.

Bacterial and fungal cultures from the scalp of 2 subjects with dirty dots were negative. Dirty dots were readily removed by shampoo, but reaccumulated as early as 1 day afterwards. Unlike dirty dots, the yellow scale of seborrheic dermatitis was more common in younger and older children.

We present dirty dots as a novel and unique dermoscopic finding of the normal scalp examination in prepubertal children. Dirty dots likely represent nonmicrobial environmental particles and should not be confused with cadaverized hairs or other abnormal trichoscopic structures. Given the negative correlation between the presence of dirty dots and seborrheic dermatitis in our series, we postulate that the relative decrease in sebaceous activity in prepubertal children may lead to an inability to repel particulate debris from exogenous environmental sources. Future studies to elucidate the potential relationship between dirty dots and sebaceous activity will be of interest.

Correspondence: Dr Fu, Department of Dermatology, University of California, San Francisco, 1701 Divisadero St, San Francisco, CA 94115 (fu@derm.ucsf.edu).

Author Contributions: Study concept and design: Tosti. Acquisition of data: Starace and Tosti. Analysis and interpretation of data: Fu and Tosti.

Financial Disclosure: None reported.

Funding/Support: This work was supported by an educational grant from the Women’s Dermatologic Society Mentorship Program, San Francisco, California (Dr Fu).

Role of the Sponsors: The Women’s Dermatologic Society Mentorship Program had no role in the design or conduct of the study; in the collection, analysis, or interpretation of data; or in the preparation, review, or approval of the manuscript.

between elevated muscle enzyme levels and underlying clinical symptoms of muscle weakness and/or pain or any coexisting confounding medical condition. A focused neuromuscular examination was performed on each patient, evaluating proximal muscle strength, vibratory sensation, and deep-tendon reflex status. Creatine kinase, lactate dehydrogenase, and aldolase levels were measured. Results of muscle enzyme testing were considered abnormal if 1 (or more) of the 3 tested muscle enzymes were considered abnormal. Considering the curvilinear and/or myeloid bodies on skeletal muscle electron microscopy (defined as AMM). However, only 8 of these patients (6.7%) manifested clinical muscle weakness (defined as clinical AMM), which improved in all patients on cessation of antimalarial therapy. Casado et al\(^3\) report the prevalence of AMM (12.6%) and clinical AMM (6.7%) as higher than that previously reported (<2%), and they suggest screening patients without clinical symptoms by measuring muscle enzyme levels on a repeated basis.

One of us (J.P.C.) has used antimalarial agents extensively in the treatment of cutaneous disease over the past 30 years and has not identified a relationship between elevated muscle enzyme levels and underlying clinical AMM or clinical benefit of routine determination of muscle enzyme levels. Casado et al\(^3\) identified 22 of 119 patients receiving long-term treatment with antimalarial agents as having persistent muscle enzyme level elevation (18.5%). The clinical significance of this elevation is not clear because only 6.7% were ultimately determined to have clinical AMM, and all patients improved on cessation of antimalarial therapy. Thus, we designed this prospective study to evaluate the clinical relevance of screening muscle enzyme levels in our dermatologic patients treated with AMM.

Methods. The study was approved by the institutional review board at the University of Louisville, Louisville, Kentucky. We prospectively enrolled study participants who met inclusion criteria during routine follow-up visits. Patients were eligible for inclusion if they had been undergoing antimalarial therapy for at least 3 months at a minimum oral dose of 200 mg of hydroxychloroquine sulfate every other day or 250 mg of chloroquine hydrochloride daily. Patients were excluded if they had any medical condition potentially affecting neuromuscular function or muscle enzyme levels.

After giving written informed consent, patients completed a comprehensive questionnaire targeting symptoms of muscle weakness and/or pain or any coexisting confounding medical condition. A focused neuromuscular examination was performed on each patient, evaluating proximal muscle strength, vibratory sensation, and deep-tendon reflex status. Creatine kinase, lactate dehydrogenase, and aldolase levels were measured. Results of muscle enzyme testing were considered abnormal if 1 (or more) of the 3 tested muscle enzyme levels was elevated.

Results. Over a 10-month study period, we prospectively enrolled 21 patients (20 women and 1 man), mean age, 51.7 years (age range, 35-76 years). Eight patients were treated with chloroquine and 13 patients with hydroxychloroquine. The most common underlying disorder was cutaneous lupus erythematosus. Seven patients (33%) had either symptoms of muscle weakness or abnormal results of neuromuscular examination; 4 of these 7 patients exhibited both weakness and abnormal examination results.

Laboratory evaluation detected abnormal muscle enzyme levels in 4 patients (19%). However, there was no correlation between abnormal muscle enzyme levels and the presence of either muscle symptoms or abnormal neuromuscular examination results. Our patient population and study results are summarized in the Table. The trend showing higher prevalence of muscle enzyme level elevation in chloroquine-treated patients (38%) than in hydroxychloroquine-treated patients (8%) is not statistically significant (P = .25 using the Fisher exact test).

Comment. Our study identified a 19% prevalence of abnormal muscle enzyme levels in dermatologic patients treated with long-term antimalarial regimens, similar to that determined by Casado et al\(^3\) (18.5%). However, in contrast to the 36% correlation between elevated enzyme levels and clinical AMM identified by Casado et al, none of our patients with abnormal muscle enzyme levels experienced clinical myopathy. Thus, our study findings do not support the clinical relevance of muscle enzyme level determination for the identification of clinical AMM. The significance of the observed muscle enzyme level elevations observed in our patients is unknown; they may be incidental findings or may represent subclinical AMM; however, neither invasive testing nor empirical discontinuation of otherwise successful therapy based solely on asymptomatic muscle enzyme level elevations seems warranted.

Of the 5 patients with abnormal physical findings, 1 patient had a history of stroke, thus explaining her unilateral weakness. Three other patients experienced subtle proximal muscle weakness that could potentially be related to antimalarial therapy along with numerous other possible causes. The last patient experienced markedly diminished bilateral patellar deep tendon reflexes that predated antimalarial therapy. While it is difficult to interpret the significance of muscle symptoms and abnormal neuromuscular examinations experienced by some of our patients, we are reassured by the lack of correlation with elevated muscle enzyme levels.

While this prospective study is limited by a small sample size, our findings, combined with prior large retrospective studies failing to identify any correlation between abnormal muscle enzyme levels and clinical AMM, call into question the suitability of using muscle enzyme testing in screening for clinical AMM.\(^1\,\,2\,\,4\) As AMM is typically reversible on discontinuation of antimalarial therapy, routine muscle enzyme level determination and potential subsequent electromyography and/or muscle biopsy do not seem warranted. For these reasons, empirical discontinuation of antimalarial therapy may be the most prudent action for the small percentage of patients who develop clinical AMM. Accordingly, we believe screening for AMM via a thoro-
Table. Summary of Patient Details and Study Findings

<table>
<thead>
<tr>
<th>Patient No./ Sex/Age, y</th>
<th>Antimalarial Medication and Daily Oral Dose, mg</th>
<th>Duration of Therapy, mo</th>
<th>Underlying Disease</th>
<th>Muscle Symptoms</th>
<th>Muscle Weakness</th>
<th>Abnormal Nerve Test Results</th>
<th>Muscle Enzyme Levels</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/F/37</td>
<td>HCQ 200</td>
<td>24</td>
<td>DLE</td>
<td>Absent</td>
<td>No</td>
<td>No</td>
<td>Normal</td>
</tr>
<tr>
<td>2/F/56</td>
<td>HCQ 200</td>
<td>22</td>
<td>DLE</td>
<td>Absent</td>
<td>No</td>
<td>No</td>
<td>Abnormal (CK, 315 U/L)</td>
</tr>
<tr>
<td>3/F/36</td>
<td>HCQ 200</td>
<td>77</td>
<td>DLE</td>
<td>Absent</td>
<td>No</td>
<td>No</td>
<td>Normal</td>
</tr>
<tr>
<td>4/F/65</td>
<td>HCQ 200</td>
<td>44</td>
<td>SGLE</td>
<td>Present</td>
<td>No</td>
<td>No</td>
<td>Normal</td>
</tr>
<tr>
<td>6/F/55</td>
<td>HCQ 200</td>
<td>37</td>
<td>SGLE</td>
<td>Present</td>
<td>Yes (shoulder girdle strength, 4/5; otherwise 5/5)</td>
<td>No</td>
<td>Normal</td>
</tr>
<tr>
<td>7/F/59</td>
<td>HCQ 400</td>
<td>62</td>
<td>DLE</td>
<td>Absent</td>
<td>No</td>
<td>Patellar reflex decreased; otherwise normal; vibratory sensation normal</td>
<td>Normal</td>
</tr>
<tr>
<td>12/F/76</td>
<td>HCQ 100</td>
<td>9</td>
<td>SGLE</td>
<td>Absent</td>
<td>No</td>
<td>No</td>
<td>Normal</td>
</tr>
<tr>
<td>13/F/64</td>
<td>HCQ 400</td>
<td>51</td>
<td>SLE with LE-associated vasculitis</td>
<td>Present</td>
<td>No</td>
<td>Yes (right thigh and right extensor arm, 3/5; patient with history of stroke; otherwise normal)</td>
<td>Normal</td>
</tr>
<tr>
<td>14/F/56</td>
<td>HCQ 400</td>
<td>48</td>
<td>SCLE with Livedo reticularis</td>
<td>Present</td>
<td>No</td>
<td>No</td>
<td>Normal</td>
</tr>
<tr>
<td>15/F/41</td>
<td>HCQ 200</td>
<td>4</td>
<td>SLE</td>
<td>Absent</td>
<td>No</td>
<td>No</td>
<td>Normal</td>
</tr>
<tr>
<td>16/F/57</td>
<td>HCQ 300</td>
<td>76</td>
<td>DLE</td>
<td>Absent</td>
<td>No</td>
<td>No</td>
<td>Normal</td>
</tr>
<tr>
<td>20/F/42</td>
<td>CQ 500</td>
<td>53</td>
<td>Degos disease</td>
<td>Absent</td>
<td>No</td>
<td>No</td>
<td>Normal</td>
</tr>
<tr>
<td>5/F/41</td>
<td>CQ 500</td>
<td>5 (previously taking HCQ for 36 mo)</td>
<td>SCLE and RA</td>
<td>Present</td>
<td>No</td>
<td>Yes (hip flexors, 4/5 bilaterally; otherwise normal)</td>
<td>Normal</td>
</tr>
<tr>
<td>8/F/58</td>
<td>CQ 250</td>
<td>24</td>
<td>SLE with Chilblains and DLE lesions</td>
<td>Absent</td>
<td>No</td>
<td>No</td>
<td>Normal</td>
</tr>
<tr>
<td>9/F/56</td>
<td>CQ 250</td>
<td>3 (previously taking HCQ for 22 mo)</td>
<td>Generalized GA</td>
<td>Absent</td>
<td>No</td>
<td>No</td>
<td>Normal</td>
</tr>
<tr>
<td>10/F/64</td>
<td>CQ 250</td>
<td>4 (previously taking HCQ for 9 mo)</td>
<td>Generalized GA</td>
<td>Absent</td>
<td>No</td>
<td>No</td>
<td>Abnormal (CK, 477 U/L; ALD, 8.0 U/L; LDH, 272 U/L)</td>
</tr>
<tr>
<td>11/M/37</td>
<td>CQ 375</td>
<td>23 (previously taking HCQ for 6 mo)</td>
<td>DLE</td>
<td>Absent</td>
<td>No</td>
<td>No</td>
<td>Normal</td>
</tr>
<tr>
<td>16/F/35</td>
<td>CQ 250</td>
<td>4</td>
<td>DLE</td>
<td>Absent</td>
<td>No</td>
<td>No</td>
<td>Normal</td>
</tr>
<tr>
<td>17/F/52</td>
<td>CQ 250</td>
<td>13</td>
<td>Hypertrophic DLE</td>
<td>Absent</td>
<td>No</td>
<td>No</td>
<td>Normal</td>
</tr>
<tr>
<td>21/F/45</td>
<td>CQ 500</td>
<td>3</td>
<td>DLE</td>
<td>Absent</td>
<td>No</td>
<td>No</td>
<td>Normal</td>
</tr>
</tbody>
</table>

Abbreviations: ALD, aldolase (normal range, 1.2-7.6 U/L); CK, creatine kinase (normal range, 24-173 U/L); CQ, chloroquine hydrochloride; DLE, discoid lupus erythematosus; HCQ, hydroxychloroquine sulfate; GA, granuloma annulare; LDH, lactate dehydrogenase (normal range, 100-250 U/L); LE, lupus erythematosus; SCLE, subacute cutaneous lupus erythematosus; SLE, systemic lupus erythematosus; RA, rheumatoid arthritis.

SI conversion factors: To convert ALD, CK, and LDH to micromoles per liter, multiply by 0.167.

Andrew H. Kalajian, MD
Jeffrey P. Callen, MD

Correspondence: Dr Kalajian, 310 E Broadway, Floor 2A, Louisville, KY 40202 (akalajian@yahoo.com).

Author Contributions: Drs. Kalajian and Callen had full access to all of the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis. Study concept and design: Kalajian and Callen. Acquisition of data: Kalajian and Callen. Analysis and interpretation of data: Kalajian and Callen. Drafting of the manuscript: Kalajian and Callen. Critical revision of the manuscript for important intellectual content: Kalajian and Callen. Administrative, technical, or material support: Kalajian. Study supervision: Callen.

Financial Disclosure: Dr Callen has received hono-

raria from Amgen, Abbott Immunology, Genentech, Centocor, Electrical Optical Sciences, Medicis, and Steifel. He serves on a safety monitoring committee for Genmab. None of these financial relationships are relevant to this manuscript.

Additional Contributions: Andrea Booth-Kalajian provided assistance with study design and data analysis.

Disclaimer: Dr Callen is associate editor of the Archives of Dermatology, but he was not involved in any of the decisions regarding review of the manuscript or its acceptance.


Giant Subcutaneous Tumors on the Thighs

I read with some interest the recent Off-Center Fold “Giant Subcutaneous Tumors on the Thighs” by Bae and colleagues,¹ published in a recent issue of the Archives. Regrettably, Bae and coworkers appear to have missed the clinical and pathologic significance of the giant thigh masses in their patient. Based on the clinical history, the provided clinical photograph, and the photomicrographs, these “tumors” would appear to be indisputable examples of massive localized lymphedema of the morbidly obese, a distinctive pseudoneoplasm first reported by Farshid and Weiss² in The American Journal of Surgical Pathology in 1998 and subsequently reported in a number of additional publications.³⁻¹²

As in the case presented by Bae et al,² massive localized lymphedema is almost always seen in morbidly obese patients, where it presents as giant, pendulous masses of the medial thighs. On gross pathologic examination, massive localized lymphedema displays striking dermal thickening and fibrosis with prominent stromal edema and expansion of the fibrous septa between the individual fat lobules. As the photographs provided by Bae and coworkers nicely illustrate, the microscopic features of massive localized lymphedema include prominent lymphangiectasia and lymphatic proliferation, chronic inflammatory cell aggregates, and myxofibrous expansion of the intralobular septa, with scattered stellate to dendritic fibroblasts and myofibroblasts.⁵ CD3+ expression, a largely nonspecific finding, may be seen in these reactive fibroblasts.

Massive localized lymphedema is thought to be due to local lymphatic obstruction by abundant adipose tissue. Although massive localized lymphedema has typically been thought of as quite rare, we have seen easily 20 or more cases in our soft-tissue pathology consultation practice at Mayo Clinic over the past year or so, likely reflecting the increased prevalence of obesity in the American population. This letter does not permit a lengthy discussion of so-called dendritic fibromyxolipoma, this purported entity is regarded by most soft-tissue experts as simply a myxoid variant of spindle cell lipoma, and is not included as a distinct entity in the most recent World Health Organization classification of soft-tissue tumors.¹³

Andrew L. Folpe, MD

Correspondence: Dr Folpe, College of Medicine, Mayo Clinic, 200 First St SW, Rochester, MN 55905 (Folpe.Andrew@Mayo.edu).

Financial Disclosure: None reported.


VIGNETTES

Trichoscopy Using a Handheld Dermoscope: An In-Office Technique to Diagnose Genetic Disease of the Hair

Over a dozen hair shaft disorders have been described, all diagnosable using light microscopy.¹ Difficulties exist in the placement of hairs onto slides for viewing in that the hairs may shift or fly away. Furthermore, sampling requires cutting short hairs, often in the brows, which is difficult to perform in young children.

Trichoscopy is a technique of examining the hairs using dermoscopy. The technique using videodermoscopy at ×20 to ×70 original magnification has been proven comparable to light microscopy for the diagnosis of hair shaft abnormalities, including those of Netherton syndrome.²³ A single case report of trichoscopy using a handheld camera with dermoscopy attachment has been described,⁴ the findings of which were partially corroborated using videodermoscopy.⁵ We hypothesized that dermoscopy with polarized light could aid in the visualization in vivo and in vitro of hair shaft abnormalities.

Report of Cases. Three patients were examined using the handheld Canon Powershot A630 (Canon, Lake Success, New York) with the Derm foto polarized dermoscopy lens attachment (3Gen, San Juan Capistrano, California). Trichoscopy was performed on patients with known hair shaft abnormalities, previously confirmed by light microscopy of cut hairs. The patients were examined with dermoscopy of hair in vivo and dermoscopy of cut hair samples from the scalp (n = 3) and the brows...