Scientific Journal of Dermatology and Venereology
Vol. 4, No. 1, 2026, pp. 57–81 · e-ISSN 3025-6208 *journal homepage: phlox.or.id/index.php/sjdv*

Case Report

Subacute Cutaneous Lupus Erythematosus in an 11-Year-Old Girl with Childhood-Onset Systemic Lupus Erythematosus: Adult Reference Intervals, Unscored Instruments and the Findings the Record Did Not Contain

Indira Dharmasamitha[1*], Putu Ayu Gadis Laksmitha Ksata Yadnya[2], Reika Ravenski Novsa[2]

[1]*Department of Dermatology and Venereology, Ngoerah Hospital, Denpasar, Indonesia*
[2]*Department of Dermatology and Venereology, Faculty of Medicine, Universitas Udayana, Denpasar, Indonesia*

ARTICLE INFO

Article history:
Received: July 15, 2026
Revised: August 6, 2026
Accepted: August 29, 2026
Available Online: September 1, 2026
Published: September 3, 2026

Keywords:
Childhood-onset systemic lupus erythematosus
Glomerular filtration rate
Nutritional assessment
Photosensitivity
Subacute cutaneous lupus erythematosus

***Corresponding author:**
Indira Dharmasamitha
E-mail: indiradharma@unud.ac.id
ORCID: 0009-0005-1272-7658

DOI:
https://doi.org/10.59345/sjdv.v4i1.338

ABSTRACT

Background: Subacute cutaneous lupus erythematosus is an uncommon cutaneous expression of childhood-onset systemic lupus erythematosus, and paediatric reports usually describe morphology and treatment rather than the measurements on which severity is judged.

Objective: To describe paediatric subacute cutaneous lupus in skin of colour and to recompute, from the record alone, the classification, renal, nutritional and drug-exposure quantities it never derived.

Methods: One child's record over three assessments and 56 days was reviewed retrospectively and reported following the CARE guideline. Values were reproduced as printed; body surface area, estimated glomerular filtration rate, two nutritional indices, transaminase ratios, per-kilogram drug exposures, the 2019 EULAR/ACR score and the cutaneous activity and damage index were computed from them.

Results: An 11-year-old premenarchal Javanese girl presented with a seven-month photodistributed eruption of erythematous and hyperpigmented macules, papules and plaques on the face, neck, thorax, limbs and palms, with fever and weight loss. Antinuclear antibody was 1:10,000 fine-speckled and the erythrocyte sedimentation rate 124 mm/hour; nuchal biopsy showed interface dermatitis with vacuolar degeneration, Civatte bodies and pigment incontinence. A line immunoassay was positive for anti-Sm, anti-SSA/Ro, anti-SSB/La and anti-RNP/Sm. On topical treatment and photoprotection alone the cutaneous activity index fell from 18 to 4 by day 28. A creatinine of 0.84 mg/dL reported as normal corresponds to 69.2 mL/min/1.73 m^2 by the bedside Schwartz equation; the 2019 criteria total 10 points, never computed; the nutritional indices disagreed. No urinalysis, creatine kinase or complement was done.

Conclusion: Paediatric cutaneous lupus should be reported with its instruments scored and its reference intervals age-appropriate.

All authors have reviewed and approved the final version of the manuscript.

1. Introduction

Systemic lupus erythematosus is a multisystem autoimmune disease in which loss of tolerance to nuclear antigens produces autoantibodies, immune complexes and inflammatory tissue injury. When it begins in childhood it is uncommon. A population-based study covering a defined United States region reported an adjusted incidence of 0.7 per 100,000 children overall and 1.2 per 100,000 in girls, with an adjusted point prevalence on 1 January 2015 of 1.1 per 100,000.[1] A national health-insurance analysis in the Republic of Korea reported a higher prevalence of 6.92 per 100,000 persons and an incidence of 2.76 per 100,000 person-years between the ages of 5 and 18 years, over a whole-population denominator rather than a paediatric one, so the two figures are not directly comparable.[2] Cutaneous involvement is among the commonest features at any age of onset: in a single-centre cross-sectional comparison of 415 patients, of whom 79 had childhood-onset disease, the commonest manifestations across the whole cohort were articular in 75.2 %, haematological in 70.1 %, cutaneous in 67.9 %, photosensitivity in 59.3 % and renal in 41.7 %.[3] A multicentre comparison of 174 children with lupus in Türkiye and the United States

found photosensitivity in 45.4 % and 21.2 % of the two cohorts and renal involvement in 41.7 % and 36.4 %.[4]

Subacute cutaneous lupus erythematosus is one of the three classical subtypes of cutaneous lupus. It presents as annular or papulosquamous lesions in photoexposed skin, heals without scarring, leaves dyspigmentation, and is associated with anti-SSA/Ro antibodies, although less uniformly than the textbook description suggests. In a matched case-control study of 64 patients with subacute disease, anti-Ro was present in 64 % of the proton-pump-inhibitor-induced group and in only 46 % of the primary group.[5] A pooled analysis of 29 published cases of checkpoint-inhibitor-induced subacute disease found anti-Ro in 91.7 %, antinuclear antibodies in 75.0 % and anti-La in 50.0 % of those in whom serology was reported, and interface dermatitis in 65.5 % and lymphocytic infiltration in 82.8 % of the 29 biopsies.[6] The subtype is not distributed evenly across the age range: in a cross-sectional study of 291 patients with cutaneous lupus, 79 % were early-onset and 21 % late-onset, and a subtype other than the chronic form carried an odds ratio of 2.18 for late onset.[7] Subacute disease in a child is therefore an uncommon presentation of an uncommon disease, and the published paediatric experience consists largely of individual reports, of which the two paediatric patients treated with anifrolumab described by Shimizu and colleagues are an example.[8]

The pathogenesis of the cutaneous lesion is now understood in some detail. Keratinocytes from patients with lupus are epigenetically primed for ultraviolet injury: in a translational study of cultured cells from six patients, hypomethylation of the Hippo regulator *WWC1*, with a methylation difference of −0.153, was accompanied by significant overexpression of that regulator.[9] Apoptotic keratinocytes translocate Ro/SSA and La/SSB to the cell surface, where circulating autoantibodies can reach them, and the resulting type I interferon response sustains the interface reaction. In a translational study of lesional cutaneous lupus skin, four patients with therapy-refractory acute, subacute, chronic discoid and chilblain disease were successfully treated with a tyrosine kinase 2 inhibitor, which is consistent with, although it does not establish, the interferon pathway lying upstream of the interface reaction.[10] Blockade of the type I interferon receptor reduced the activity score from 40 to 8 in one patient with generalised discoid disease.[11]

What the paediatric literature reports far less often is the arithmetic. A cutaneous lupus case report will usually give the morphology, the biopsy, the antibody panel and the drugs, and will state a severity score as a single number. It will rarely state which items of that score were met, which of the tests that the score presupposes were performed, or what a paediatric laboratory value means once it is converted into the quantity that clinicians actually act on. That gap matters because the instruments used in lupus are additive and item-based: a classification score is a sum over domains, an activity index is a sum over descriptors, and a nutritional classification depends entirely on which index is chosen. A number reported without its constituents cannot be audited by a reader, and a number read against the wrong reference interval is not merely imprecise but can be the opposite of the truth.

Novelty and aim of study. This report describes subacute cutaneous lupus erythematosus in an 11-year-old girl, a presentation for which the published paediatric material is thin, and then does something that the paediatric cutaneous lupus literature has not to our knowledge done with a single case: it reconstructs, from the recorded data alone, the three quantities on which the characterisation of this child as having lupus of unstated classification, unimpaired kidney function and adequate nutrition rested, and shows that two of them were never derived at all and the third was derived and then never reconciled with the competing index that contradicts it. The aim is threefold. First, to describe the clinical, histopathological and serological features of paediatric subacute cutaneous lupus in skin of colour, in which post-inflammatory dyspigmentation dominates the appearance. Second, to demonstrate by explicit item-by-item scoring what the 2019 classification criteria, the cutaneous activity and damage index, two nutritional indices and two paediatric glomerular filtration rate equations say about this child, and where they disagree with what the record concluded. Third, to convert those discrepancies into a numbered minimum data set that any clinician seeing a child with photodistributed cutaneous lupus can apply at the first visit.

2. Methods

2.1. Study design, setting and source material

This is a retrospective single-patient record review, reported in accordance with the CARE guideline for case reports. The source material is the complete clinical record of one child assessed on three occasions at a tertiary referral centre in Bali over 56 days, together with the photographs, the laboratory reports and the histopathology report filed with it. Day 0 is the first specialist assessment on 9 September 2025; the intervals before it are calculated from the seven-month history and the stated month in which each event occurred and therefore carry the uncertainty of a month, while the intervals after it are exact. Institutional approval for the publication of anonymised clinical material and written informed consent from the patient's mother, with the assent of the patient, were obtained as stated in the Declarations.

2.2. Data extracted, and the rule applied to them

Every value reported here is reproduced as the source record printed it, including the reference intervals the laboratory quoted and the rounding it applied. Where the record is internally inconsistent the inconsistency is declared rather than silently corrected, and the eight discrepancies found are set out together in a dedicated section below. A quantity that is absent from the record is reported as never obtained rather than as normal, and the distinction between a finding that was

sought and not found and a finding that was never sought is preserved throughout.

2.3. Quantities recomputed from the recorded data

None of the quantities in this paragraph appears anywhere in the source record; each was computed at manuscript preparation from the recorded weight of 29.8 kg, height of 140.7 cm, mid-upper arm circumference and laboratory values. Body surface area was obtained from the Mosteller formula, the square root of the product of weight in kilograms and height in centimetres divided by 3600. Estimated glomerular filtration rate was derived by two height-based paediatric equations: the bedside Schwartz equation, 0.413 times height in centimetres divided by serum creatinine in mg/dL, and the CKiD U25 equation with the female constant of 37.6 and height in metres. Nutritional state was classified twice, by body mass index-for-age in standard-deviation scores and by mid-upper arm circumference as a percentage of the quoted standard, the percentage-of-standard convention the source record itself applied. Transaminases were expressed as multiples of the upper limit quoted for each, and the aspartate-to-alanine ratio was computed from the same two values. Every prescribed drug was normalised to body weight and, where the convention requires it, to body surface area. The 2019 European Alliance of Associations for Rheumatology and American College of Rheumatology criteria were applied item by item, counting only the highest weighted criterion met within each domain, and the cutaneous lupus erythematosus disease area and severity index was scored separately for activity and for damage at each assessment at which the record contains enough description to score it. Neither estimated filtration rate can be interpreted without knowing whether the creatinine assay was enzymatic or based on the Jaffe reaction, which the record does not state.

2.4. Photographic analysis

Erythema was quantified from the clinical photographs as a within-photograph contrast of the anterior chest, computed as the mean CIELAB a* of the reddest decile of skin pixels minus that of the median decile in the same region of the same photograph. The measure is taken within each image so that illuminant and white balance cancel, because the photographs were not taken against a colour standard and illumination, white balance and camera distance were not standardised between sessions. The images available in the record have a native resolution of approximately 250 by 320 pixels per panel and are reproduced at the largest size that resolution supports.

3. Results

3.1. Presentation and history

An 11-year-old premenarchal Javanese Indonesian girl was referred from a general hospital to the Dermatology and Venereology outpatient clinic of a tertiary referral hospital in Denpasar, Bali, with suspected subacute cutaneous lupus erythematosus. She was first assessed there on 9 September 2025, which is taken throughout as day 0. The complete chronology of the illness, from the first appearance of the eruption to the final recorded assessment, is illustrated in Figure 1. Her mother described erythematous patches that had appeared on the face seven months earlier and had subsequently spread to the postauricular skin, the posterior neck and the palms. Their appearance had been accompanied by intermittent fever, after which the patches had darkened to a brownish-black. Taking the stated seven-month history from the date of presentation places the onset around February 2025, an interval of 212 days. The lesions were intermittently pruritic and stinging but did not disturb sleep or daily activity. The child had noticed that her skin had become more sensitive to sunlight and that the eruption worsened during outdoor activity. In the weeks before referral she had become weak, tired easily and had lost weight. Joint pain, cough, rhinorrhoea, nausea, vomiting and hair loss were all specifically denied at this visit. Oral and fluid intake had fallen; bowel and bladder function were normal.

She had never had a similar illness. There was no personal or family history of systemic or autoimmune disease and no drug or food allergy in the patient or in any family member. She had been given an unnamed topical cream at a community health centre and, since May 2025, a compounded cream prescribed by a dermatovenereologist; neither the child nor her mother knew the name or composition of either preparation, so that about 131 of the 212 days before referral, and an unrecorded period before that, were spent applying topical agents that cannot now be identified. Because the record gives only the month, the 131 days carry the uncertainty of a month in either direction. She was a schoolgirl who had taken part in an outdoor marching band until June 2025, when she stopped. She was the second of three children and lived with her parents. She bathed twice daily with a mild soap.

3.2. Physical and dermatological examination

She appeared mildly unwell but was fully alert, with a Glasgow Coma Scale score of E4V5M6, a pulse of 98 beats per minute, a respiratory rate of 20 breaths per minute, an axillary temperature of 36.8 °C and an oxygen saturation of 99 % breathing room air. She weighed 29.8 kg and measured 140.7 cm, giving a body mass index of 15.05 kg/m^2, recorded in the notes as 15.1 kg/m^2. Body mass index-for-age lay between −2 and −1 standard deviations, described in the record as a normal nutritional state with a risk of undernutrition, and height-for-age lay between −1 and 0 standard deviations. Mid-upper arm circumference was 18.0 cm against a quoted standard of 22.4 cm for age and sex. The head was normocephalic. There was no conjunctival hyperaemia, conjunctival pallor or scleral icterus, no pharyngeal hyperaemia, tonsils were graded T1 on each side and there was no cervical lymphadenopathy. The first and second heart sounds were regular and single without a murmur, breath sounds were vesicular without rhonchi or wheeze, the abdomen was soft with normal bowel sounds and no

organomegaly, and the limbs were warm without oedema.

Dermatological examination of the face, the anterior and posterior thorax and both upper limbs showed multiple well-defined erythematous-hyperpigmented macules and patches of geographic outline measuring from 0.2 × 0.4 cm to 1 × 1.4 cm in a discrete, localised distribution, together with well-defined erythematous-hyperpigmented papules and plaques of the same size range, several of them confluent and some eroded. The distribution and morphology at this first assessment are shown in Figure 2. The most infiltrated lesions were the violaceous, scaly, partly confluent plaques at the nape and upper back, seen in Figure 2e and Figure 2f. Palmar involvement, an unusual site for subacute cutaneous lupus, is illustrated in Figure 2g, and crusted eroded papules on the extensor forearms in Figure 2h. In this Fitzpatrick type IV to V skin the dominant impression was of hyperpigmentation rather than erythema. Dermoscopy was not performed at any visit; in 120 discoid lupus erythematosus lesions from 110 patients of Fitzpatrick types IV to V, follicular keratotic plugs were seen dermoscopically in 73 lesions (60.8 %) and perifollicular whitish haloes in 65 (54.1 %), features of structure rather than of colour, which is why dermoscopy is often more informative than inspection in this skin type.[12]

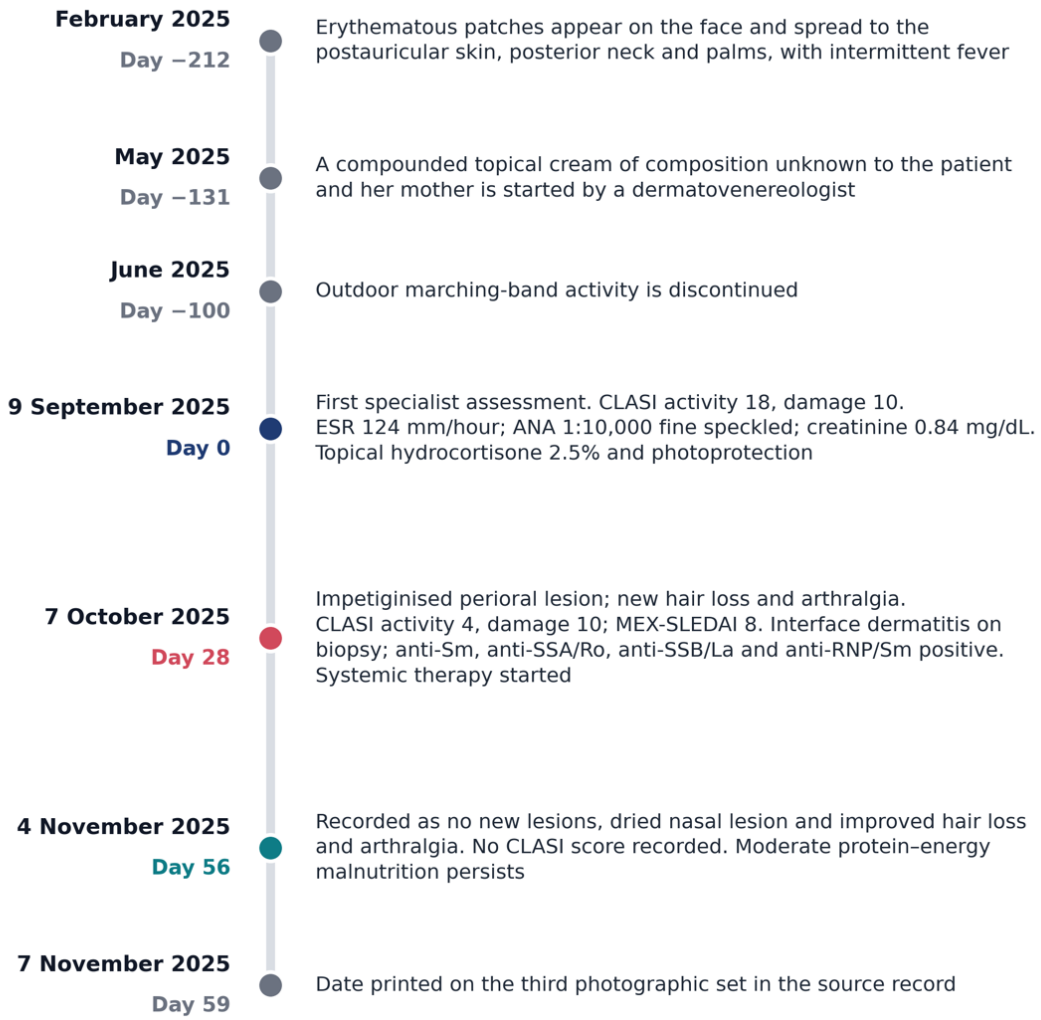

Figure 1. Chronology of the illness. Day 0 is the first specialist assessment on 9 September 2025. The three intervals before it are calculated from the seven-month history and the stated month in which each event occurred, so they carry the uncertainty of a month; the intervals after day 0 are exact. The record labels the third assessment day 56 but dates the photographs taken at it 7 November 2025, which is day 59; both are shown.

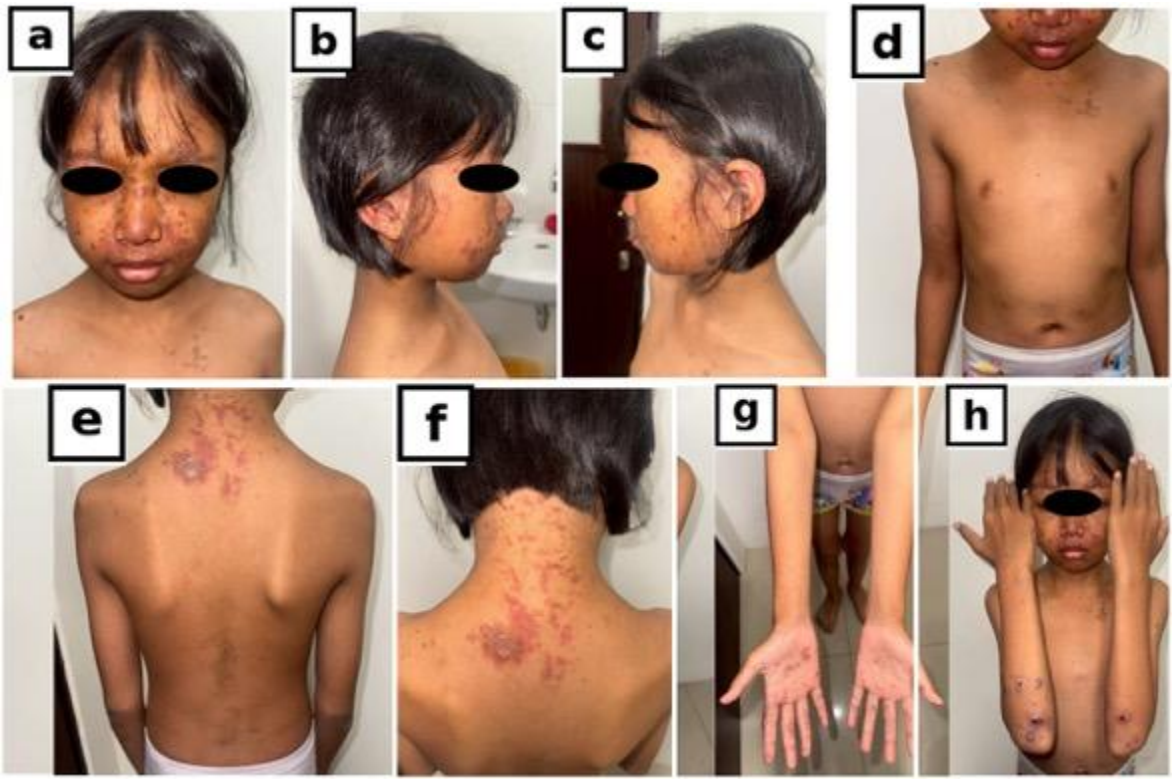

Figure 2. Cutaneous findings at first presentation, 9 September 2025. (a) Frontal view of the face showing confluent erythematous-hyperpigmented macules and patches over the malar skin, nose, perioral region and chin. (b, c) Right and left lateral views showing involvement of the preauricular and postauricular skin and the lateral neck. (d) Anterior thorax with discrete macules over the photoexposed V area. (e) Posterior thorax and (f) posterior neck showing violaceous, scaly, partly confluent plaques at the nape, the site later biopsied. (g) Palmar surfaces with erythematous macules and eroded papules. (h) Extensor surfaces of both forearms with crusted, partly eroded papules. The eyes are masked in every panel of this and of every other figure in which they appear.

3.3. Laboratory investigations at presentation

Table 1 presents the complete blood count, biochemistry and serology obtained on the day of the first assessment, together with the reference intervals against which each was reported and the quantities that can be derived from them. The white cell count was 5.83 × 10^3/µL with a normal differential; the absolute neutrophil and lymphocyte counts of 3.16 and 2.30 × 10^3/µL are internally consistent with the percentages, and the five differential percentages sum to 100.00 %, and the five absolute counts sum exactly to the white cell count. The reproduction is exact for the lymphocyte, monocyte, eosinophil and basophil counts; the product of the white cell count and the neutrophil percentage is 3.15 against a printed 3.16, a difference in the second decimal that is analyser rounding rather than error. The red cell indices are likewise internally consistent: haematocrit divided by red cell count, multiplied by ten, gives a mean corpuscular volume of 92.63 fL against the reported 92.60 fL, and haemoglobin divided by haematocrit, multiplied by one hundred, gives a mean corpuscular haemoglobin concentration of 33.12 g/dL against the reported 33.10 g/dL. There was a normocytic anaemia with a haemoglobin of 10.40 g/dL, a haematocrit of 31.40 % and a red cell count of 3.39 × 10^6/µL, with a normal red cell distribution width of 13.60 %. The platelet count was normal at 241 × 10^3/µL with a mean platelet volume of 10.1 fL, marginally above the quoted upper limit of 10.0 fL. The erythrocyte sedimentation rate was 124.0 mm/hour, 4.13 times the quoted upper limit of 30 mm/hour.

Aspartate aminotransferase was 94 U/L against an upper limit of 34 U/L and alanine aminotransferase was 57 U/L against an upper limit of 55 U/L, so that the two enzymes were 2.76 and 1.04 times their respective limits and the aspartate-to-alanine ratio was 1.65. Blood urea nitrogen was 7.7 mg/dL, serum creatinine 0.84 mg/dL and random blood glucose 81 mg/dL; all three were reported as within the normal range. Indirect immunofluorescence for antinuclear antibodies gave a fine-speckled pattern at a titre of 1:10,000. Alkaline phosphatase, bilirubin, serum albumin, complement C3 and C4, anti-double-stranded DNA antibody, creatine kinase, lactate dehydrogenase, reticulocyte count, haptoglobin, direct antiglobulin test, urine microscopy and the urine protein-to-creatinine ratio were not requested at this or at any later visit, and are tabulated in Table 1 as absent rather than as normal. Table 1 also carries a second grey row for the tests that were performed but whose results the record states only in words. It should be said at once that three of the intervals in Table 1 are themselves adult ones: the haemoglobin, haematocrit and erythrocyte count are quoted against adult female limits, so the anaemia described below is graded on the same kind of reference that section 4.6 criticises for the creatinine.

Table 1. Laboratory results at first presentation on 9 September 2025, and the quantities derived from them. Values outside the quoted interval are shown in bold red; a grey cell marks a result for which the laboratory quoted no age-appropriate interval.

Investigation	Result	Quoted reference interval
Complete blood count		
Leukocytes (WBC)	5.83 ×10³/µL	4.1 – 11 ×10³/µL
Neutrophils (NE%)	54.1 %	47 – 80 %
Lymphocytes (LY%)	39.5 %	13 – 40 %
Monocytes (MO%)	6 %	2 – 11 %
Eosinophils (EO%)	0.2 %	0 – 5 %
Basophils (BA%)	0.2 %	0 – 2 %
Neutrophils, absolute (NE#)	3.16 ×10³/µL	2.5 – 7.5 ×10³/µL
Lymphocytes, absolute (LY#)	2.3 ×10³/µL	1 – 4 ×10³/µL
Monocytes, absolute (MO#)	0.35 ×10³/µL	0.1 – 1.2 ×10³/µL
Eosinophils, absolute (EO#)	0.01 ×10³/µL	0 – 0.5 ×10³/µL
Basophils, absolute (BA#)	0.01 ×10³/µL	0 – 0.1 ×10³/µL
Erythrocytes (RBC)	**3.39 ×10⁶/µL**	4 – 5.2 ×10⁶/µL
Haemoglobin (HGB)	**10.4 g/dL**	12 – 16 g/dL
Haematocrit (HCT)	**31.4 %**	36 – 46 %
MCV	92.6 fL	80 – 100 fL
MCH	30.7 pg	26 – 34 pg
MCHC	33.1 g/dL	31 – 36 g/dL
RDW	13.6 %	11.6 – 14.8 %
Platelets (PLT)	241 ×10³/µL	150 – 440 ×10³/µL
Mean platelet volume (MPV)	**10.1 fL**	6.8 – 10 fL
Inflammatory markers, chemistry and serology		
Erythrocyte sedimentation rate	**124 mm/hour**	< 30 mm/hour
Neutrophil-to-lymphocyte ratio	1.37	< 3.13
AST (SGOT)	**94 U/L**	< 34 U/L
ALT (SGPT)	**57 U/L**	< 55 U/L
Blood urea nitrogen	7.7 mg/dL	7 – 18 mg/dL
Serum creatinine	*0.84 mg/dL*	*No paediatric interval quoted*
Random blood glucose	81 mg/dL	70 – 140 mg/dL
Antinuclear antibody, indirect immunofluorescence	**1:10,000, fine speckled**	Negative at 1:80
Quantities derived from the values above		
Body mass index (29.8 kg; 140.7 cm)	15.05 kg/m²	Recorded in the source as 15.1 kg/m²
Mid-upper arm circumference as a percentage of the quoted standard (18.0 cm; 22.4 cm)	**80.4 %**	> 85 % of standard
Body surface area, Mosteller formula	1.079 m²	—
Estimated glomerular filtration rate, bedside Schwartz equation	**69.2 mL/min/1.73 m²**	> 90 mL/min/1.73 m²
Estimated glomerular filtration rate, CKiD U25 sex-dependent equation	**63.0 mL/min/1.73 m²**	> 90 mL/min/1.73 m²
Aspartate aminotransferase to alanine aminotransferase ratio	**1.65**	< 1 in most hepatocellular injury
Aspartate aminotransferase as a multiple of the upper limit of normal	**2.76 ×**	≤ 1 ×
Erythrocyte sedimentation rate as a multiple of the upper limit of normal	**4.13 ×**	≤ 1 ×
Interval from stated symptom onset to first specialist assessment	212 days	—
Urinalysis, urine protein-to-creatinine ratio, complement C3 and C4, anti-double-stranded DNA antibody, creatine kinase, lactate dehydrogenase, reticulocyte count, direct antiglobulin test, haptoglobin, serum albumin, alkaline phosphatase, total bilirubin	*Not performed at any visit*	—
Serum 25-hydroxyvitamin D; and at day 28 the urea, creatinine, sodium, haemoglobin, eosinophil count and estimated glomerular filtration rate	*Performed, but no value is recorded*	—

Notes: *Derived quantities were computed at manuscript preparation from the recorded weight, height, mid-upper arm circumference and laboratory values; none appears in the source record. The bedside Schwartz equation is 0.413 × height in centimetres ÷ serum creatinine in mg/dL. The CKiD U25 sex-dependent equation uses a constant of 37.6 with height in metres. Neither estimate can be interpreted without knowing whether the creatinine assay was enzymatic or based on the Jaffe reaction, which the record does not state. The seven day-28 abnormalities named in section 3.5, of which the vitamin D deficiency is one, are recorded in the source only as words, with no numerical values, and therefore cannot be tabulated. Three intervals in this table are adult ones: the haemoglobin, haematocrit and erythrocyte rows are quoted against adult female limits, so the anaemia graded here is graded on the same kind of reference the creatinine row is criticised for. The neutrophil-to-lymphocyte ratio and its cut-off of 3.13 are reproduced as the source record printed them. The 90 mL/min/1.73 m² comparator is the conventional staging boundary below which a filtration rate is called reduced, not a measured paediatric normal range; the aspartate-to-alanine comparator is clinical convention rather than a quoted study value; and the 85 % mid-upper-arm-circumference band is the percentage-of-standard convention the source record itself applied.*

3.4. Initial diagnosis and management

On the history, examination and initial investigations the department recorded a working diagnosis of suspected subacute cutaneous lupus erythematosus, with discoid lupus erythematosus, mixed connective tissue disease and photocontact dermatitis as differential diagnoses. The Cutaneous Lupus Erythematosus Disease Area and Severity Index, which scores erythema and scale or hypertrophy as activity and dyspigmentation and scarring as damage, and reports the two as separate totals,[13] gave a total activity score of 18 and a total damage score of 10. She was given cetirizine 10 mg orally once daily as required for pruritus and hydrocortisone 2.5 % cream twice daily to the erythematous lesions, with the back deliberately left untreated because a biopsy was planned. The whole sequence of assessments, from the first appearance of the eruption to the last entry, is set out in Figure 1. She was referred to the paediatric allergy and immunology division because of the raised transaminases and the antinuclear antibody result, and was counselled to use a sunscreen with a sun protection factor of 30 and to avoid sun exposure.

3.5. Day 28: flare of the perioral skin, histopathology and extended serology

At review on 7 October 2025, day 28, the facial and back lesions had become thinner and more uniformly hyperpigmented, but a new erythematous, pustular and crusted lesion had appeared beneath the nose. She now reported hair loss and pain in both elbows and both knees, both of which had been specifically denied at the first visit, together with continuing intermittent pruritus, which was present at day 0 and for which she was already taking an antihistamine. Examination showed hyperpigmented macules and patches together with erythematous-hyperpigmented papules and plaques on the face, the anterior and posterior thorax and both upper limbs, with pustules and yellowish crusts in the orbicularis oris region. The activity score had fallen to 4 while the damage score remained at 10, and a Mexican Systemic Lupus Erythematosus Disease Activity Index of 8 was recorded, described as moderate activity. Repeat investigation was reported as showing mild anaemia, eosinophilia, raised blood urea nitrogen, raised creatinine, a reduced estimated glomerular filtration rate, mild hyponatraemia and vitamin D deficiency; no numerical value accompanies any of these seven abnormalities in the record.

Histopathological examination of the nuchal plaque is set out in Figure 3. The specimen comprised epidermis, dermis, adnexal structures and subcutaneous adipose tissue, with effacement of the rete ridges, as seen in the whole-mount view in Figure 3b. At the dermoepidermal junction there was moderate lymphocytic infiltration with vacuolar degeneration of the basal layer and Civatte bodies, shown in Figure 3c and Figure 3e. A superficial perivascular lymphocytic infiltrate and pigment incontinence were present in the papillary dermis, illustrated in Figure 3d, with melanophages visible at high power in Figure 3f. The appearances were reported as consistent with lupus erythematosus. A line immunoassay for extractable nuclear antigens was positive for anti-Sm, anti-SSA/Ro, anti-SSB/La and anti-RNP/Sm. Gram staining of the eroded perioral lesion showed Gram-positive cocci and leucocytes, and the lesion was treated as a secondary bacterial infection; no culture or susceptibility testing was requested.

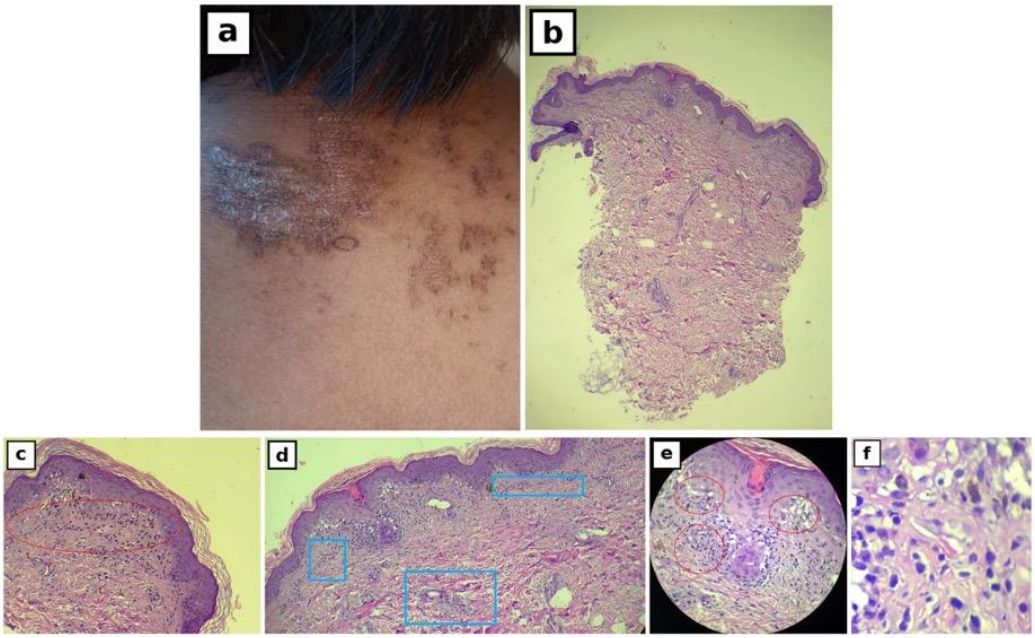

Figure 3. Biopsy site and histopathology of the nuchal plaque. (a) The posterior neck lesion from which the specimen was taken; the source record does not date this photograph. (b) Whole-mount view showing epidermis, dermis, adnexal structures and subcutaneous adipose tissue, with effacement of the rete ridges (haematoxylin and eosin). (c) Interface dermatitis with vacuolar degeneration of the basal layer, outlined. (d) Superficial perivascular lymphocytic infiltrate and pigment incontinence, boxed. (e) Vacuolar change and Civatte bodies at the dermoepidermal junction, circled. (f) Melanophages in the superficial dermis at high power. Two further fields supplied with the record, of 193 by 121 and 479 by 222 pixels, were omitted because at any printed size they add nothing that panels (e) and (f) do not show more legibly; the panels reproduced here carry between 286 and 386 source pixels per printed inch. No magnification bar was present on any field, so magnifications are not stated.

The final diagnoses recorded were subacute cutaneous lupus erythematosus associated with systemic lupus erythematosus, vitamin D deficiency, moderate protein-energy malnutrition and secondary bacterial infection. Treatment was extended to topical hydrocortisone 2.5 %, topical sodium fusidate 2 %, urea 10 % cream and continued photoprotection, together with oral methylprednisolone tapering from 32 mg to 4 mg daily, mycophenolate sodium 900 mg daily, hydroxychloroquine 150 mg daily, cholecalciferol 5,000 IU daily, calcium carbonate 500 mg three times daily, folic acid 1 mg daily and nutritional therapy.

3.6. The final assessment

At the third and last assessment recorded, described in the notes as day 56, no new lesions were seen, the lesion beneath the nose had dried, the earlier lesions had become predominantly hyperpigmented, and the hair loss and arthralgia had improved. Moderate protein-energy malnutrition was recorded as persisting. No anthropometric measurement, no laboratory value and no activity or damage score accompanies this entry. The photographs taken at this visit are dated 7 November 2025, which is day 59 rather than day 56; both labels are reproduced in Figure 1 rather than reconciled. Figure 4 places the same four regions side by side at all three assessments, and it is the visual record on which the statement that no new lesions appeared has to be judged.

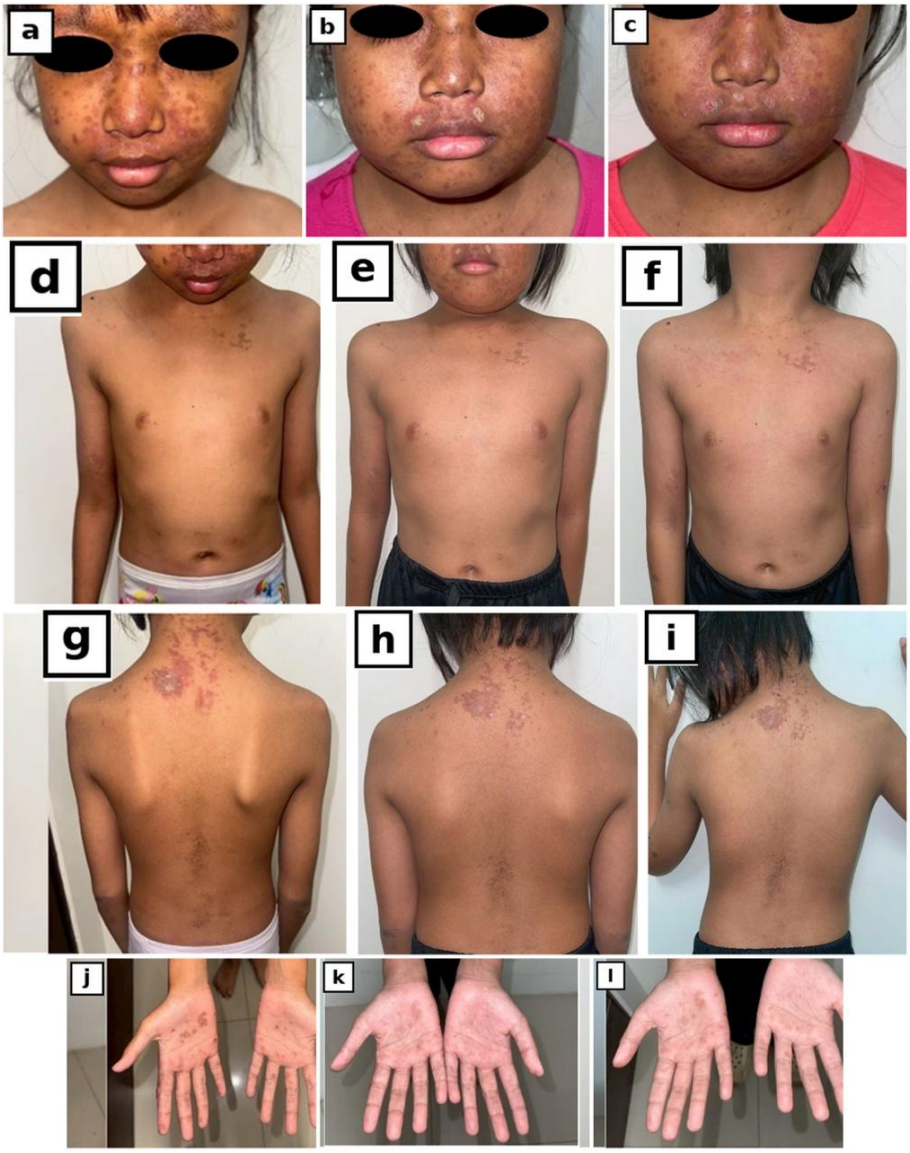

Figure 4. The same four regions at the three assessments: day 0 (9 September 2025) in the left column, day 28 (7 October 2025) in the middle column and the final assessment in the right column. (a--c) Perioral and mid-facial skin: dense hyperpigmentation at day 0; two honey-coloured crusts on the upper lip at day 28, the impetiginised lesion from which Gram-positive cocci were reported; erythematous papules with resolution of the crusts at the final assessment. (d--f) Anterior thorax: discrete brown macules at day 0 and day 28, and a confluent erythematous patch across the photoexposed V area at the final assessment. (g--i) Posterior neck and upper back: the nuchal plaque flattens progressively and develops a pale centre whose nature, dyspigmentation or early atrophy, cannot be settled from a photograph and was never examined. (j--l) Palms: eroded and crusted papules at day 0 resolving to residual macules. Illumination, white balance and camera distance were not standardised between sessions, which is why the erythema comparison in Figure 5E is made within each photograph rather than between photographs.

4. Discussion

4.1. What this case is, and how long it took to become one

The diagnosis reached in this child is correct. A photodistributed, non-scarring eruption that heals with dyspigmentation, a high-titre fine-speckled antinuclear antibody, anti-SSA/Ro and anti-SSB/La positivity and a biopsy showing interface dermatitis with vacuolar degeneration, Civatte bodies and pigment incontinence is subacute cutaneous lupus erythematosus until proved otherwise. The purpose of the sections that follow is not to dispute that diagnosis but to examine the three quantities that were used to characterise its severity, because each of them can be recomputed from the record and each returns a different answer from the one the record reached. Those recomputations are collected in Figure 5. Figure 5B begins with a quantity simpler than any of the three, the interval that preceded them all, which Figure 1 also marks on the clinical timeline.

The interval from the onset the mother describes to the first specialist assessment is 212 days. Of that interval, 131 days were spent applying a compounded topical preparation whose composition neither the child nor her mother could name, prescribed by a dermatovenereologist to whom the diagnosis of lupus had evidently not yet occurred. The specialist follow-up that the record contains is 56 days by the narrative label and 59 days by the date on the last photographs. This child was therefore undiagnosed for close to four times as long as the specialist follow-up the record contains, as Figure 5B sets out. That interval is not a neutral administrative fact. In a longitudinal cohort of 286 patients with isolated cutaneous lupus, progression to systemic disease occurred in 4.8 % of the hydroxychloroquine group against 27 % of the group given topical corticosteroids or calcineurin inhibitors alone, and early initiation of hydroxychloroquine was associated with an 87 % reduction in the hazard of progression.[14] In childhood-onset systemic disease a related quantity has been measured, although it is time spent in high disease activity after diagnosis rather than time spent before it: in a cohort of 196 children, the mean time to damage was 5.0 years in those who spent at least 5 % of follow-up in a high-activity state against 12.5 years in those who did not.[15] Damage in childhood lupus accrues early, and it accrues fastest at the beginning: in a prospective cohort of 50 children, damage occurred in 34 % and, of those, 64.7 % had accrued it within one year of diagnosis.[16] In a multicentre cohort of 670 children with a median disease duration of only 2.8 years, 29.9 % already had at least one item of irreversible damage.[17] A more recent single-centre cohort of 102 children found accrued damage in 33.3 %, with mucocutaneous damage in 5.9 %.[18] The seven months this child spent without a diagnosis are the single most modifiable variable in her whole record, and they were spent, in part, under specialist dermatological care.

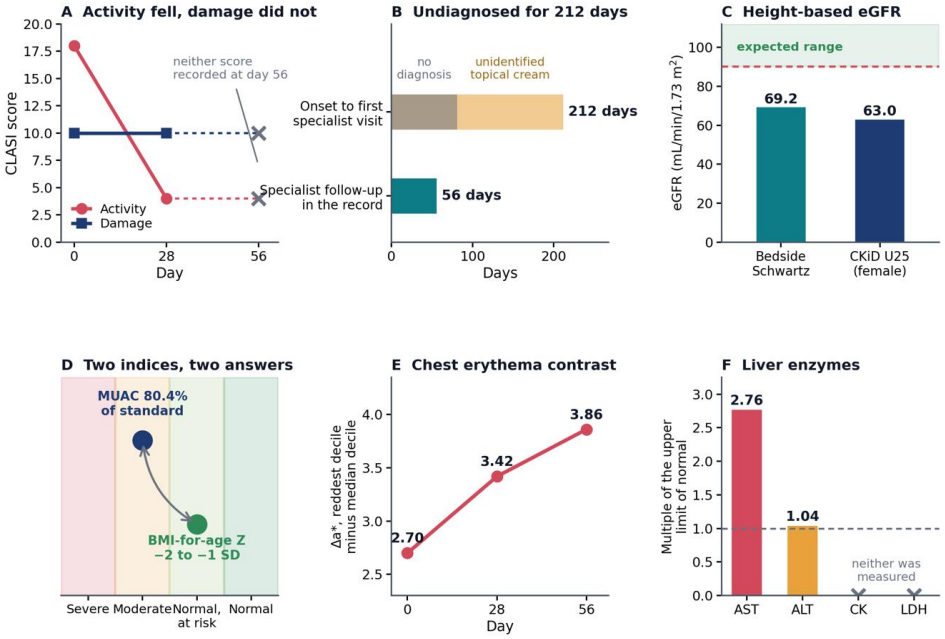

Figure 5. Quantities reconstructed from the record. (A) The CLASI activity score fell from 18 to 4 between day 0 and day 28, on an oral antihistamine, topical corticosteroid and photoprotection alone, while the damage score stayed at 10; neither component was recorded at the final assessment, marked by crosses. (B) The interval from stated symptom onset to the first specialist assessment, 212 days, of which 131 days were spent applying a compounded topical preparation whose composition neither the patient nor her mother could name, against the 56 days of specialist follow-up the record contains. (C) The recorded serum creatinine of 0.84 mg/dL converted by two height-based equations. (D) The two nutritional indices recorded on the same day place the child in different categories. (E) Within-photograph erythema contrast of the anterior chest, computed as the mean CIELAB a* of the reddest decile of skin pixels minus that of the median decile in the same region of the same photograph; the measure is taken within each image so that illuminant and white balance cancel. (F) Aspartate and alanine aminotransferase as multiples of their upper limits of normal; creatine kinase and lactate dehydrogenase are drawn as no bar at all because they were never measured, and a cross on the baseline marks each of them.

4.2. Establishing the subtype

Two features anchor the diagnosis of the subacute subtype rather than its alternatives. The first is the absence of recorded scarring. No lesion in this child healed with a scar, and the lesions that resolved did so with dyspigmentation, as the serial photographs in Figure 4 show; no atrophic scar, follicular plug or cicatricial alopecia was recorded at any visit. That statement needs one qualification, which is made here rather than left for a reader to find. The nuchal plaque, the most infiltrated lesion in the whole record and the one that was biopsied, develops a pale central area in the final photographs, seen in Figure 4i. Whether that is dyspigmentation alone or early atrophy cannot be settled from a photograph, and it was never examined for atrophy at any visit. Since the absence of atrophic scarring is the principal discriminator from discoid lupus, that is not a trivial omission, and the differential-diagnosis matrix below carries it as a grey qualification of the scarring row. The second is the histopathology. Interface dermatitis with vacuolar degeneration of the basal layer, Civatte bodies, a superficial perivascular lymphocytic infiltrate and pigment incontinence is the expected pattern, and it is not a universal one: in the pooled series of 29 patients with drug-induced subacute disease, biopsies showed interface dermatitis in 65.5 % and lymphocytic infiltration in 82.8 %, so roughly a third of biopsies do not show the defining change.[6] The pattern is also not unique. Vacuolar interface dermatitis was present in 46.5 % of 43 patients with clinically amyopathic dermatomyositis, together with mucin in 69.8 %, telangiectasia in 65.1 % and a lymphocytic infiltrate in 48.8 %.[19] Histology in this setting supports a clinical diagnosis; it does not make one on its own. No heliotrope discolouration and no Gottron papules are recorded. Proximal muscle strength, on the other hand, was never tested: the recorded examination covers consciousness, vital signs, head, conjunctivae, pharynx, tonsils, lymph nodes, heart, lungs, abdomen and limb warmth, and contains no strength testing, while the history records that she had become weak and tired easily. A myopathic component was therefore not excluded so much as not looked for, a point taken up in section 4.8. Within that histopathology, the vacuolar change shown in Figure 3c and the melanophages in Figure 3f carry most of the weight.

The distribution deserves a separate remark. Subacute cutaneous lupus is classically described on the upper back, shoulders, neck and extensor arms with relative sparing of the face, and this child had extensive facial involvement together with palmar lesions, illustrated in Figure 2a and Figure 2g. Palmar involvement is not part of the classical description. Its presence here does not overturn the diagnosis, which rests on morphology, histology, serology and course, but it is worth recording because the classical distribution is what a clinician searches for and this child would not have matched it. The distribution row of the differential-diagnosis matrix below makes that comparison explicit.

4.3. Does she classify as having systemic lupus erythematosus? The score nobody computed

The record moves from suspected subacute cutaneous lupus at day 0 to subacute cutaneous lupus associated with systemic lupus erythematosus at day 28, and states a Mexican Systemic Lupus Erythematosus Disease Activity Index of 8. It never states a classification score. Table 2 applies the 2019 European Alliance of Associations for Rheumatology and American College of Rheumatology criteria to this child item by item.[20] She satisfies the entry criterion with an antinuclear antibody titre of 1:10,000, far above the required 1:80. She scores four points in the mucocutaneous domain for subacute cutaneous lupus and six points in the SLE-specific antibody domain for anti-Sm. The two points for non-scarring alopecia do not add, because within each domain only the highest weighted criterion counts, and four already exceeds two. The constitutional domain is the interesting one. The mother reported intermittent fever from the onset of the eruption, and the criterion is worth two points; but the criterion is defined by a temperature above 38.3 °C, and the only temperature recorded anywhere in this file is the 36.8 °C measured at day 0. Applying to fever the same standard this manuscript applies to the joint domain, it is not scoreable. The strict total is therefore 10 points, exactly at the threshold, on two domains alone; accepting the reported fever would give 12. The weight attached to each criterion, and the basis on which it was or was not met, is tabulated in Table 2.

Two things follow from that arithmetic, and they point in opposite directions. The first is reassuring: the classification is robust. Every criterion in Table 2 that is marked grey was either never tested or never documented in the detail its definition requires, and every one of them could only have added points. Had complement been low, had a urinalysis shown proteinuria, had a direct antiglobulin test been positive, the total would have risen. Nothing that was omitted could have brought her below the threshold. The threshold itself is well validated in children: in a multicentre study of 1,045 children comprising cases and disease controls, the 2019 criteria showed a sensitivity of 95.3 % and a specificity of 97.8 % at an optimal total score threshold of 10.[21] In a retrospective comparison of 442 patients with lupus and 428 controls, of whom 221 and 214 respectively were children, the 2019 criteria were the most sensitive of three criteria sets in the childhood-onset subgroup at 98.2 %, with a specificity of 93.5 % in that subgroup.[22] The second implication is less comfortable. A score of 10 points assembled from two domains, in a child whose renal, complement and antiphospholipid domains were never sampled at all and whose haematological domain was sampled only for the two cytopenias, is a floor and not a description. It tells us that she has lupus. It tells us almost nothing about how much lupus she has.

Table 2. The 2019 European Alliance of Associations for Rheumatology and American College of Rheumatology classification criteria for systemic lupus erythematosus, applied item by item to this patient. Criteria that were met are shown in bold red; grey marks a criterion that could not be evaluated because the test was never performed or because the clinical detail its definition requires was never recorded. Where a single row combines criteria of differing weight, only the highest of them is printed in the weight column.

Domain	Criterion	Weight	Status	Basis in this record
Entry criterion	ANA at a titre of ≥1:80 on HEp-2 cells	Entry	**Met**	Fine-speckled pattern at 1:10,000
Constitutional	Fever	2	*Not scoreable*	Intermittent fever reported by the mother from onset; the criterion is defined by a temperature above 38.3 °C and no febrile reading is recorded. The only measured temperature in the file is 36.8 °C at day 0
Haematological	Leukopenia	3	Not met	WBC 5.83 ×10³/μL
Haematological	Thrombocytopenia	4	Not met	Platelets 241 ×10³/μL
Haematological	Autoimmune haemolysis	4	*Never tested*	No direct antiglobulin test, reticulocyte count, lactate dehydrogenase or haptoglobin
Neuropsychiatric	Delirium, psychosis or seizure	5	Not met	None documented
Mucocutaneous	Non-scarring alopecia	2	**Met**	Hair loss reported at day 28; not additive within the domain
Mucocutaneous	Oral ulcers	2	Not met	None documented
Mucocutaneous	Subacute cutaneous or discoid lupus erythematosus	4	**Met**	Clinical morphology plus interface dermatitis on biopsy
Mucocutaneous	Acute cutaneous lupus erythematosus	6	Not met	No malar rash of acute-cutaneous type; the confluent malar lesions are of subacute morphology and are scored in the row above
Serosal	Pleural or pericardial effusion; acute pericarditis	6	Not met	No effusion; cardiorespiratory examination normal
Musculoskeletal	Joint involvement	6	*Not scoreable*	Arthralgia of elbows and knees without documented synovitis, tenderness in ≥2 joints or ≥30 minutes of morning stiffness
Renal	Proteinuria >0.5 g/24 h	4	*Never tested*	No urinalysis at any visit
Renal	Class II or V lupus nephritis	8	*Never tested*	No kidney biopsy
Renal	Class III or IV lupus nephritis	10	*Never tested*	No kidney biopsy
Antiphospholipid antibodies	Anticardiolipin, anti-β2GP1 or lupus anticoagulant	2	*Never tested*	Not requested
Complement proteins	Low C3 or low C4; low C3 and low C4	4	*Never tested*	C3 and C4 never measured
SLE-specific antibodies	Anti-dsDNA or anti-Sm	6	**Met**	Anti-Sm positive on line immunoassay; anti-dsDNA never measured
Total scored, strict reading	**Highest weighted criterion in each domain that is met**	**10**	**≥ 10 classifies**	**Mucocutaneous 4 + SLE-specific antibodies 6**
Total if the reported fever is accepted	**As above, plus the constitutional domain**	**12**	**≥ 10 classifies**	**Mucocutaneous 4 + SLE-specific antibodies 6 + constitutional 2**

Notes: *Within each domain only the highest weighted criterion that is met contributes to the total, so the two points for non-scarring alopecia do not add to the four points for subacute cutaneous lupus erythematosus. The strict total is therefore ten, exactly at the threshold, on the mucocutaneous and SLE-specific antibody domains alone; accepting the reported intermittent fever as a constitutional criterion would give twelve. Every criterion marked grey could only have raised the total, and none could have lowered it. The haematological domain is the one grey area that was partly sampled: leukopenia and thrombocytopenia were measured and were absent, and only autoimmune haemolysis was never tested. Delirium, psychosis and seizure carry two, three and five points respectively, and a pleural or pericardial effusion five against six for acute pericarditis; the highest of each is shown.*

4.4. The differential diagnosis, and a premise the record contradicts

Table 3 sets three of the four diagnoses that were entertained, together with acute cutaneous lupus erythematosus, which was not entertained, against the findings in this patient; the fourth, mixed connective tissue disease, cannot be separated at the bedside and is dealt with on its own below. Acute cutaneous lupus is excluded by the absence of a malar rash, of a generalised morbilliform eruption and of a severe systemic flare. Discoid lupus is excluded by the absence of recorded follicular plugging, atrophic scarring and cicatricial alopecia at any visit, with the qualification made above and carried in the scarring row of Table 3, and by the widespread symmetrical distribution seen in Figure 2, which is more characteristic of the subacute form; the scarring row of Table 3 is the single most useful

discriminator between the two. Photoallergic contact dermatitis is excluded by the absence of any identified photosensitiser, by a lesion pattern that is annular and papulosquamous rather than eczematous, and by the persistence of the lesions with dyspigmentation rather than their resolution on withdrawal. It has to be said that this exclusion is weaker than it looks, because the child had been applying an unidentified compounded cream for 131 days, and a photopatch test was never performed. The exclusion is nonetheless secure on the histology, which shows interface dermatitis rather than spongiosis. The reported histopathology describes interface change, vacuolar degeneration, Civatte bodies and pigment incontinence and describes no spongiosis; the fields reproduced in Figure 3 show none either, although five published fields cannot speak for a whole specimen. Table 3 places that distinction in the row that separates the two diagnoses.

Table 3. Bedside discrimination of subacute cutaneous lupus erythematosus from the three alternatives that can be separated at the bedside, with the finding in this case set against each. Mixed connective tissue disease, which cannot be separated this way, is treated separately in Table 4. Red marks a feature that is present and discriminating in this patient; grey marks one that could not be assessed because the examination or the test it depends on was never done.

Feature	SCLE	ACLE	DLE	PACD	This patient
Distribution	Photoexposed upper back, shoulders, neck, extensor arms; face less often	Malar and generalised	Head, neck, ears; localised	Sites of applied photosensitiser, photoexposed	**Face, postauricular skin, nape, upper back, extensor arms, palms**
Morphology	Annular or papulosquamous, non-indurated	Confluent oedematous erythema	Indurated plaques with follicular plugging	Eczematous, papulovesicular	**Erythematous-hyperpigmented macules, patches, papules and plaques, some confluent, some eroded**
Scarring	Absent	Absent	Characteristic, with atrophy	Absent	**No scar recorded at any visit** *(the pale centre of the nuchal plaque was photographed but never examined for atrophy)*
Dyspigmentation	Common and long-lasting	Transient	Central hypopigmentation with a hyperpigmented rim	Post-inflammatory only	**Prominent and persistent**
Photosensitivity	Marked	Marked	Variable	By definition	**Reported, with worsening on outdoor activity**
Histopathology	Interface dermatitis, vacuolar change, superficial perivascular infiltrate	Sparse interface change	Dense periadnexal infiltrate, follicular plugging, basement membrane thickening	Spongiotic dermatitis	**Interface dermatitis with vacuolar degeneration, Civatte bodies, superficial perivascular infiltrate, pigment incontinence**
Serology	Anti-SSA/Ro in about half of primary and about two thirds of drug-induced cases	Anti-double-stranded DNA	Frequently negative	No specific autoantibody	**Anti-Sm, anti-SSA/Ro, anti-SSB/La and anti-RNP/Sm all positive**
Systemic features	Mild; a minority meet criteria for systemic disease	Part of a systemic flare	Usually skin-limited	None	**Fever, fatigue, weight loss, arthralgia; no Raynaud phenomenon and no sclerodactyly; proximal strength never tested**
Diagnostic test not performed here	—	—	—	Photopatch testing	*Never requested*
Verdict in this patient	**Supported**	Not supported	Not supported	Not supported	—

Notes: *SCLE, subacute cutaneous lupus erythematosus; ACLE, acute cutaneous lupus erythematosus; DLE, discoid lupus erythematosus; PACD, photoallergic contact dermatitis. The scarring row is the single most useful discriminator between the subacute and the discoid form. The exclusion of photoallergic contact dermatitis is the weakest of the three, because the child had applied an unidentified compounded preparation for 131 days and photopatch testing was never performed; it rests on the histology, which shows interface dermatitis rather than spongiosis.*

Mixed connective tissue disease requires a longer answer, because the reason the record gives for excluding it is contradicted by the record itself. Table 4 sets out, requirement by requirement, what that diagnosis asks for, what this record contains and what was never tested. The manuscript from which this case is drawn states that specific anti-U1 ribonucleoprotein antibody testing had not been performed or was not reported as positive. The line immunoassay obtained at day 28 was in fact reported as positive in the anti-RNP/Sm band, which is the band that carries the U1 small nuclear ribonucleoprotein complex. Two things follow. The first is that the record contradicts its own premise: an assay result bearing on U1 ribonucleoprotein was obtained and was reported positive. The second is that a combined RNP/Sm band on a qualitative line immunoassay cannot say which of the two antigens the serum binds, and no quantitative anti-U1RNP assay was ever requested, so the antibody status by which mixed connective tissue disease is defined was never resolved either way. If serology had been the only argument the exclusion would fail. It is not

the only argument, and the diagnosis is nonetheless correctly excluded, for two reasons that the record contains but did not use.

The first reason is the simultaneous positivity for anti-Sm. In an inception cohort of 228 patients with newly diagnosed lupus, anti-double-stranded DNA, anti-Sm, anti-U1RNP, anti-Ro and anti-La were positive in 50.0 %, 30.7 %, 42.5 %, 54.8 % and 22.4 % respectively, so each is common, although that study does not cross-tabulate them and marginal frequencies cannot establish how often the two coexist.[23] A cluster analysis of 278 patients separated the two serologies by phenotype: the most severe phenotype carried anti-Sm in 34.4 % with renal involvement in 62.2 %, whereas the cluster in which half the patients met the definition of mixed connective tissue disease carried anti-U1-RNP 70 kDa antibodies in 87.5 %.[24] Those are marginal frequencies within clusters rather than a cross-tabulation, so they show that the two specificities load onto different phenotypes rather than that they never coexist. In mixed connective tissue disease anti-U1RNP is the dominant, usually isolated, high-titre specificity; anti-Sm is not a feature of it. The second reason is that mixed connective tissue disease is a clinical diagnosis with formal criteria, and this child meets none of their clinical requirements. In a multicentre case-control cohort of 47 children with juvenile-onset mixed connective tissue disease, 44 of 47, or 93.6 %, fulfilled either the Sharp or the Kasukawa criteria, and none met any other criteria set without fulfilling one of those two.[25] This child had no Raynaud phenomenon, no swollen hands and no sclerodactyly, and neither dysphagia nor a nailfold abnormality is recorded. Two of those absences are weaker than the others: nailfold capillaroscopy was never performed, and proximal muscle strength, as section 4.2 notes, was never tested. The point generalises beyond this case: an antibody result excludes a diagnosis only when the diagnosis is defined by that antibody alone, and mixed connective tissue disease is not. Table 4 therefore lists the clinical features of that diagnosis, not only its serology, and records against each what this file actually contains.

That distinction has a measurable cost when it is got wrong. In a registry-based validation of the 2019 criteria in 105 children with childhood-onset lupus against disease controls, sensitivity was 97.1 % and specificity was 94.7 % against juvenile dermatomyositis and 92.6 % against paediatric Sjögren disease, but only 55.6 % against mixed connective tissue disease, a figure that rests on five of only nine such controls.[26] The estimate is fragile, but it points in the direction one would expect: the classification criteria are weakest precisely at the boundary this child sits on, which is why the discriminating features are tabulated in Table 3 and in Table 4 rather than left to the score to carry. Scoring at the threshold does not by itself exclude mixed connective tissue disease; the clinical examination does, and that examination has to be written down. Three of the seven requirements detailed in Table 4 could not be assessed at all from what this record contains.

Table 4. Mixed connective tissue disease: what the diagnosis requires, what this record contains, and what was never tested. Red marks an entry that bears directly on the exclusion; grey marks a requirement that could not be assessed because the examination or the test it depends on was never performed.

Requirement	What the diagnosis requires	What this record contains	Status here
Dominant antibody	High-titre anti-U1RNP, usually the isolated dominant specificity	A qualitative line immunoassay reported positive in the combined anti-RNP/Sm band at day 28. No quantitative anti-U1RNP assay was ever requested, and a combined band cannot say which of the two antigens the serum binds.	**Never resolved**
Competing antibody	Anti-Sm is not a feature of the disease	Anti-Sm reported positive on the same immunoassay at day 28.	**Against the diagnosis**
Raynaud phenomenon	Present in almost every patient	Not recorded at any of the three assessments.	**Against**
Swollen hands or sclerodactyly	Characteristic	Not recorded at any of the three assessments.	**Against**
Myositis or proximal weakness	Common	No weakness recorded. Creatine kinase and lactate dehydrogenase were never measured, so a subclinical myositis cannot be excluded.	*Not assessable*
Oesophageal involvement	Common	No symptom recorded and no study performed.	*Not assessable*
Nailfold capillaroscopy	Abnormal in most patients	Never performed.	*Not assessable*
The reason the source record gives	—	That specific anti-U1RNP testing had not been performed or was not reported as positive.	**Contradicted by the anti-RNP/Sm result in the same file**
Verdict in this patient	—	Correctly excluded, but on the simultaneous anti-Sm positivity and on the absence of every clinical requirement, not on the serological reason the record gives.	**Excluded**

Notes: *The exclusion of mixed connective tissue disease in this patient does not rest on the absence of anti-U1RNP, which the line immunoassay in fact reported as positive in the anti-RNP/Sm band. It rests on the simultaneous positivity for anti-Sm, which is not a feature of mixed connective tissue disease, and on the absence of every clinical feature that the Sharp and Kasukawa criteria require. Three of the seven requirements above could not be assessed at all from what the record contains.*

4.5. What the activity instruments say, and what they were never asked

The cutaneous index was recorded twice and the systemic index once. Figure 5A plots them. The activity score fell from 18 to 4 between day 0 and day 28, a fall of 14 points or 77.8 %. That is a large and unambiguous response. An anchor-based analysis of 126 patients found that in those with an initial activity score of 8 or more, a fall of 42.1 % or at least 7 points predicted meaningful improvement in the emotions subscale of a quality-of-life instrument and a fall of 31.0 % or at least 5 points predicted it in the symptoms subscale.[27] This child cleared the first threshold by a factor of about two and the second by a factor of between two and three. The index is also responsive in the subacute subtype specifically: in a randomised placebo-controlled trial of 288 patients, 91.7 % of those with subacute disease on the highest dose achieved at least a 50 % reduction in the activity score at week 24 against 52.9 % on placebo, although the same trial found no significant difference in the overall population, 55.6 % against 44.6 %, nor in the stratum with a baseline activity score of 8 or more, 66.7 % against 50.0 %, which is one of the two strata this child belongs to; she is also in the subacute stratum, in which the difference was significant.[28]

The damage score is a different quantity and behaves differently. It counts dyspigmentation and scarring, which reverse slowly if at all, and it did not move: 10 at day 0 and 10 at day 28. That is not a treatment failure but a property of the instrument, and it is reproduced in larger series. In a multicentre registry of 443 patients treated with belimumab, activity scores fell significantly in acute and subacute cutaneous lupus at six months while damage scores remained stable over 36 months.[29] In 15 patients treated with type I interferon receptor blockade, the median activity score fell from 16 to 1 while the median damage score fell only from 5 to 4.[30] A damage score of 10 in an 11-year-old at her first specialist visit is therefore the most consequential of the irreversible numbers in this record, because it is the one that treatment is least able to take back. It was accrued during the 212 days before anyone made the diagnosis.

Neither component was recorded at the final assessment. The crosses in Figure 5A mark that absence. This matters more than it may appear, because the claim of improvement at that visit rests on the phrase \"no new lesions\", which is not an item of the cutaneous index, and on the statement that the score fell by at least four points between the first visit and the last. That statement is arithmetically true of the interval between day 0 and day 28, for which two scores exist; it cannot be verified for the final visit, for which none does. A composite index that is written down once and then replaced by a qualitative phrase has stopped being a measurement.

The systemic index invites the same question in a sharper form. A Mexican Systemic Lupus Erythematosus Disease Activity Index of 8 was recorded at day 28 and described as moderate activity. The value is plausible; in a cross-sectional study of 52 patients with early cutaneous involvement, the median score on the same index was 8 with an interquartile range of 4.5 to 11, while the median cutaneous activity score in that cohort was 3 with an interquartile range of 1 to 5, against this child's 18. The two instruments plainly measure different things.[31] But the record does not say which descriptors produced the 8. That is not a stylistic omission. The index is a weighted sum of clinical descriptors that includes a renal descriptor, a myositis descriptor and an arthritis descriptor. In this child the renal descriptor could not be scored, because no urine was examined at any visit and the day-28 urea and creatinine were recorded only as words. The myositis descriptor could not be evaluated at all, because creatine kinase was never measured and muscle strength was never tested. The arthritis descriptor rests on a record of pain in the elbows and knees with no documented swelling, tenderness in a stated number of joints or morning stiffness. Whatever combination produced the 8, at least three of its candidate constituents depended on an observation or a measurement that the record does not contain. For comparison, a national registry of 1,346 patients defined active disease as a Systemic Lupus Erythematosus Disease Activity Index above 4 with prednisone above 10 mg daily and immunosuppressive drugs, low disease activity as a score of 2 or less off both, and a modified low-activity state as a score of 4 or less with no major organ activity, no new features and prednisone of 10 mg daily or less.[32] Those bands belong to the Systemic Lupus Erythematosus Disease Activity Index, and the score recorded here is the Mexican index, a different instrument with a different item set and a different maximum. A number that cannot be decomposed, on a scale those bands were not drawn for, cannot be compared with them at all.

4.6. A creatinine of 0.84 mg/dL, and the reference interval it was read against

The single most consequential misreading in this record is also the quietest. The serum creatinine at day 0 was 0.84 mg/dL and was reported as within the normal range. The reference interval against which it was judged is an adult one; the record quotes no paediatric interval for it, which is why that cell is shaded grey in Table 1. In an 11-year-old girl of 140.7 cm, the quantity a clinician acts on is not the creatinine but the filtration rate it implies. The bedside Schwartz equation, derived in 349 children with chronic kidney disease and expressed as 0.413 times height in centimetres divided by serum creatinine, gives 69.2 mL/min/1.73 m^2.[33] The CKiD U25 equations, developed from 2,655 observations in 928 participants with iohexol-measured filtration, use sex-specific constants of 41.8 for males and 37.6 for females with height in metres; applying the female constant gives 63.0 mL/min/1.73 m^2.[34] Figure 5C shows both estimates against the 90 mL/min/1.73 m^2 line. Neither reaches it.

Three caveats belong immediately after that calculation rather than after the conclusion drawn from it. The first is the constant. The CKiD U25 equations are age- as well as sex-dependent, and the same publication

reports that the female constant ranges from 33.1 to 41.4 across childhood, so 37.6 is an anchor value rather than the value for an 11-year-old and 63.0 should be read as an approximation lying within a band.[34] The second is assay calibration. The height-based equations were derived on enzymatic, isotope-dilution-mass-spectrometry-traceable creatinine, and the record does not state which assay produced the 0.84 mg/dL, which is why the assay method appears in the minimum data set below and why the creatinine row of Table 1 carries a grey reference cell rather than a numerical one. A study of 513 healthy African schoolchildren with enzymatically measured creatinine found that 93.4 % had normalised values within the 0.67 to 1.33 interval derived in children of European descent, and showed that the bedside Schwartz equation itself carries significant age and sex dependency.[35] The third caveat is precision. In an agreement study of 120 samples from 23 paediatric kidney transplant recipients, the bedside Schwartz equation achieved a P30 of 89.2 % against 93.3 % for the CKiD U25 equation and 96.7 % for a full-age-spectrum equation, differences the authors describe as comparable performance.[36]

None of those three caveats runs in the reassuring direction. Where the bias of creatinine-based estimation has been measured against a reference method it has been upward: in 105 critically ill neonates and children studied against iohexol-measured filtration, the estimating equations gave between 60 and 71 against a measured 41 mL/min/1.73 m^2.[37] That is an intensive-care population and the magnitude cannot be carried across to a stable outpatient, but it is the opposite of a reason to treat 69.2 as generous. A cross-sectional study of 52 children after liver transplantation found that the Schwartz formula classified one patient as having chronic kidney disease where cystatin C and full-age-spectrum equations classified four, and that up to 42 % of the children without chronic kidney disease had at least one positive urinary marker of injury.[38] Both populations are unlike this child; what they establish is the direction in which a creatinine-based estimate errs, not its size.

The generalisable point is not about this child but about how the number was presented. In a survey of 121 physicians shown identical paediatric kidney data presented in different ways, giving the estimated filtration rate rather than the creatinine with anthropometric data more than quadrupled the proportion who assessed kidney function correctly, and 38.8 % assessed it correctly from the estimated rate while misinterpreting the equivalent creatinine.[39] That is precisely the error in this record, made by clinicians who would not have made it had the laboratory issued the derived value. Paediatric reference intervals are not a courtesy. Reference-interval work in healthy children finds age partitions to be statistically required even for analytes with no obvious developmental physiology: for serum neurofilament light a partition falls at age 10, and the upper limit of the interval for children aged 1 to under 10 years, 20.6 pg/mL, is 2.8 times that for those aged 10 to under 19 years, 7.44 pg/mL.[40] Reading a child against an adult interval is not conservative. It is wrong in a direction that reassures. It is also, in this table, not confined to the creatinine: the haemoglobin, haematocrit and erythrocyte count in Table 1 are quoted against adult female limits, so the anaemia discussed in section 4.9 is graded on exactly the kind of reference this section is criticising.

4.7. The test that was never done, and the conclusion drawn without it

At day 28 the record notes raised blood urea nitrogen and creatinine and a reduced estimated glomerular filtration rate, without values. The manuscript nonetheless concludes that the child had moderately active disease with predominantly mucocutaneous manifestations and without renal, serosal or other major organ involvement. That is a negative claim, and the test capable of refuting it was never performed. No urinalysis, no urine microscopy and no urine protein-to-creatinine ratio appears anywhere in the record. The omission is the single most consequential one in this whole file.

Renal involvement in childhood-onset lupus is frequent and, when it comes, it comes early. Its frequency in an unselected paediatric cohort is given above: 41.7 % and 36.4 % in the two arms of the multicentre comparison cited in the Introduction.[4] Its timing has been measured in cohorts assembled for the purpose: among 53 children with juvenile lupus nephritis, more than 80 % developed it within one year of the onset of lupus and more than half, 29 children, had it at the diagnosis of lupus.[41] In a cohort of 220 children with lupus nephritis, proteinuria was the commonest manifestation at 81.36 %, and class IV was the commonest grade among those biopsied at 33.33 %.[42] The detecting test is neither invasive nor expensive: in a method-comparison study of 356 children, the spot urine protein-to-creatinine ratio identified nephrotic-range proteinuria with a sensitivity of 89.9 % and a specificity of 92.2 % against a 24-hour collection.[43] That accuracy was measured at the nephrotic threshold, several times above the 0.5 g per 24 hours that the classification criteria use, so it establishes that the test works rather than that it works at the threshold that matters here. In the framework of Table 2, a result above 0.5 g per 24 hours would have added four points and moved the renal domain from grey to scored.

The correct statement, on this record, is not that there was no renal involvement. It is that renal involvement was not looked for at the first visit, that the estimated filtration rate implied by the first creatinine was already below 90 mL/min/1.73 m^2, and that by day 28 the record itself describes the creatinine as rising and the filtration rate as falling without recording either. A claim that a finding is absent is only as strong as the search that was made for it.

4.8. An aspartate-predominant transaminase elevation with four candidate sources

Aspartate aminotransferase was 2.76 times its upper limit while alanine aminotransferase was 1.04 times its own, giving a ratio of 1.65. Figure 5F shows the two enzymes against their limits. The record interprets this

as mild to moderate hepatic dysfunction and refers the child to paediatric immunology. Hepatic involvement is a reasonable first thought: in a prospective study of 135 patients with lupus, 20.7 % had liver abnormalities, ten had transaminases above twice the upper limit, three of those were attributable to antituberculous therapy and seven, or 5.2 % of the cohort, were classified as lupus hepatitis after no other cause could be found.[44] True overlap with autoimmune hepatitis is rarer still: among 805 hospitalised patients with lupus, five, or 0.6 %, had coexisting autoimmune hepatitis.[45]

The pattern here, however, points away from the liver. Alanine aminotransferase is the more liver-specific of the two enzymes, and in most hepatocellular injury it exceeds aspartate aminotransferase. When the ratio inverts, the differential widens to include muscle and red cells. Muscle is directly relevant, because the activity index recorded in this child contains a myositis descriptor, and myositis in lupus is identified by measuring creatine kinase: in a prospective cohort of 560 patients with no history of myositis at baseline, followed for an average of 8.5 years, five new cases were identified, an incidence of 1.05 per 1,000 person-years.[46] That is a low rate, and it is an argument for measuring the enzyme in the patients whose presentation raises the question rather than in every patient; this child, with weakness in the history, an aspartate-predominant elevation and a myositis descriptor inside the activity score she was given, is one of them. Red cells are relevant because this child was anaemic and haemolysis releases aspartate aminotransferase. Neither creatine kinase nor lactate dehydrogenase was measured, which is why Figure 5F draws no bar for them at all rather than a bar of zero: no measurement was taken, and a bar of any height would assert a value that does not exist. Alkaline phosphatase and bilirubin were also never measured, so the elevation cannot even be classified as hepatocellular or cholestatic. A fourth candidate is drug-induced injury from the compounded preparation the child had been applying for about 131 days, which cannot be excluded for a product nobody can name. Table 1 lists all four of those absent measurements in a single grey row, and the minimum data set below gives the moment at which each should have been obtained.

4.9. An anaemia that was never characterised

The haemoglobin of 10.40 g/dL with a mean corpuscular volume of 92.60 fL and a red cell distribution width of 13.60 % describes a normocytic, non-anisocytic anaemia, graded, as section 4.6 notes, against an adult female interval. In the presence of an erythrocyte sedimentation rate 4.13 times its upper limit, anaemia of chronic disease is the likeliest explanation. It is not the only one, and in juvenile lupus the alternatives are equally common: in a retrospective cohort of juvenile lupus, anaemia was the commonest cytopenia at 60 %, and anaemia of chronic disease and autoimmune haemolytic anaemia were each responsible in 23 % of patients.[47] The two are not reliably separated without a reticulocyte count, a direct antiglobulin test, a haptoglobin and a lactate dehydrogenase. In an ambispective cohort of 46 children with haemolytic anaemia and a positive direct antiglobulin test, lupus was the commonest trigger, accounting for 13 cases, with a mixed immunophenotype in 27 children (59 %), warm type in 12 (26 %) and cold agglutinin type in 7 (15 %).[48] No reticulocyte count, direct antiglobulin test, haptoglobin or lactate dehydrogenase was obtained. Had haemolysis been present it would have contributed four points to the haematological domain of Table 2, would have explained part of the aspartate aminotransferase elevation discussed above, and would have changed the treatment. Instead the anaemia was treated, if it was treated at all, with folic acid 1 mg daily and no baseline haematinic assessment. The red cell indices that define the anaemia are set out in Table 1, and the tests that would have characterised it are items 4 and 5 of the minimum data set.

4.10. Two nutritional indices measured on the same day, and two different answers

On 9 September 2025 two nutritional assessments were made on the same child on the same day. Body mass index-for-age lay between −2 and −1 standard deviations, which the record correctly describes as a normal nutritional state with a risk of undernutrition. Mid-upper arm circumference was 18.0 cm against a quoted standard of 22.4 cm, which is 80.4 % of that standard and falls in the 75 to 85 % band that the percentage-of-standard convention, the convention the source record itself used, calls moderate protein-energy malnutrition. Figure 5D shows the two results side by side. The record described the first as a normal nutritional state with a risk of undernutrition, made a diagnosis of moderate protein-energy malnutrition on the second, and never reconciled the two. The arithmetic also deserves a note: 18.0 divided by 22.4 is 80.3571 %, which rounds to 80.4 % rather than to the 80.3 % that appears in the source record.

The disagreement is not an artefact of this child. It is a well-documented property of the two indices. In 124 children with coeliac disease assessed by both, 35.5 % were malnourished by body mass index Z score and 48.4 % by mid-upper arm circumference Z score, the body mass index Z score failed to identify chronic malnutrition in 70.9 %, and agreement between the two was weak with a kappa of 0.300.[49] In 160 children with cancer admitted to intensive care, malnutrition prevalence was 21.3 % by body mass index-for-age and 34.4 % by mid-upper arm circumference-for-age.[50] The discordance runs in the other direction too: validated against total body water by deuterium dilution in 206 adolescent girls, body mass index-for-age had a sensitivity of only 14.5 % for high body fatness whereas a mid-upper arm circumference of 22.6 cm reached 96.9 %, that figure being an obesity threshold rather than a thinness one.[51] Reference data in centimetres exist for this age range: a multicentric study of 6,680 healthy Indian schoolchildren aged 5 to 17 years defined thinness at a Z score of −0.7, which is the 25th centile, with a rounded cut-off of 18.5 cm for both sexes between 10 and 14 years,[52] and a school-based study of

913 Ethiopian adolescent girls identified cut-offs of 20.1 cm for thinness and 19.7 cm for severe thinness with a sensitivity of 92.1 % for moderate thinness.[53] The two cut-offs differ by 1.6 cm and were derived in different populations, neither of them Indonesian, and no Indonesian reference was applied. What can be said is that this child's 18.0 cm falls below both of them.

The indices disagree because they weight different tissues. Handgrip strength correlated with mid-arm *muscle* circumference at r = 0.743 independently of age and sex in 63 children with cancer,[54] although that is a derived index requiring a triceps skinfold, and no skinfold was measured here, so the correlation supports the principle rather than this child's 18.0 cm directly. A low arm measurement also predicts outcome: among 111 hospitalised children with cancer, those below the fifth centile had almost 2.73 times the chance of a prolonged stay.[55]

The practical consequence for this child arrives at day 28, when systemic glucocorticoids begin. Glucocorticoids raise body mass index while slowing growth. In a two-centre cohort of 126 patients with paediatric lupus assessed with a glucocorticoid toxicity index, increased body mass index was among the commonest toxicities at 21 %, while decreased growth velocity was observed in 18 %.[56] In a case-control study of 155 children with juvenile idiopathic arthritis, 25.81 % had growth retardation, glucocorticoid use was more frequent among the affected children at 65.00 % against 34.78 %, and it was identified as an independent risk factor in multivariable analysis.[57] A rising body mass index in a child on steroids is therefore compatible with falling lean mass, and body mass index stops being informative at exactly the moment treatment starts. That the arm circumference should be preferred in that situation follows from the tissue each index weights rather than from any study cited here, and is offered as an inference. The arm circumference was measured once, on day 0, and never again. Undernutrition in paediatric autoimmune disease is not benign: in a prospective cohort of 107 children with juvenile idiopathic arthritis, 20.6 % were stunted, 22.4 % underweight and 25.2 % had a low body mass index, and underweight children had longer disease duration and more damage.[58]

4.11. A vitamin D deficiency recorded as a word

Vitamin D deficiency appears in the day-28 entry and in the final diagnosis, and cholecalciferol 5,000 IU daily was prescribed, which is 168 IU/kg/day in a 29.8 kg child. No serum 25-hydroxyvitamin D concentration appears anywhere. The prevalence data make the diagnosis entirely plausible: in a retrospective cohort of 192 children with newly diagnosed lupus of mean age 12.1 years, using a threshold below 20 ng/mL, the median concentration was 15 ng/mL and deficiency was present in 126 patients, or 65.6 %, with a higher prevalence in those with moderate and high disease activity.[59] In a case-control study of 168 children with initial-onset lupus and 109 controls, concentrations were 18.63 against 25.53 ng/mL, and activity scores were significantly higher in the insufficient and deficient groups, with medians of 14.5 and 14.0 against 9.0.[60] But a diagnosis recorded without its measurement cannot be graded, and a supplement given without a baseline cannot be evaluated. The one interventional record here used 8,000 IU daily for eight weeks in deficiency, or for four weeks in insufficiency, followed by 2,000 IU daily as maintenance in 31 patients, and reported improvement in the systemic activity index at 12 months and a significant fall in the anti-double-stranded DNA titre.[61] Choosing between that schedule and the 5,000 IU given here requires the number that is missing. Table 5 sets out the dose actually given alongside that comparator.

4.12. Treatment checked against the child rather than against the adult

Table 5 recalculates every drug in the record against the recorded weight of 29.8 kg, the height of 140.7 cm and a Mosteller body surface area of 1.079 m^2.[62] Most of the regimen survives that check comfortably. Methylprednisolone at 32 mg daily is 1.07 mg/kg/day, a prednisone equivalent of 1.34 mg/kg/day, and the recorded taper to 4 mg daily brings the methylprednisolone to 0.13 mg/kg/day, a prednisone equivalent of 0.17 mg/kg/day. That taper is worth achieving: in a two-centre cohort of 257 young people with childhood-onset lupus, each additional 1 mg/day of prednisone carried a hazard ratio of 1.1 for hospitalised infection, and 5 % had at least one hospitalised infection within a year of starting a disease-modifying drug.[63] Mycophenolate sodium 900 mg daily is 834 mg/m^2/day, equivalent to 1250 mg/day of mycophenolate mofetil or 1158 mg/m^2/day, which sits within the range used in paediatric lupus. The evidence supporting that agent in children is now strong: in a multicentre randomised non-inferiority trial of 107 children aged 5 to 17 years with proliferative lupus nephritis, the total remission rate was 92 % with mycophenolate mofetil against 89 % with intravenous cyclophosphamide.[64] A prospective registry cohort of 71 children reported complete remission in 35 % at 6 months and 58 % at 24 months, with 51 % still on corticosteroids at 24 months and 56 % experiencing at least one corticosteroid side effect.[65] Exposure in children is driven mainly by body size: a population pharmacokinetic study of 51 patients yielding 146 area-under-the-curve values found a mean of 31.05 µg × hour/mL and a mean clearance of 11.10 L/hour.[66]

One dose does not survive the check. Hydroxychloroquine 150 mg daily in a child of 29.8 kg is 5.03 mg/kg/day of actual body weight, which is above the 5.0 mg/kg/day threshold, by 0.03 mg/kg/day. The margin is small and the consequence is not immediate, but the threshold is where it is for a measured reason. In a retrospective cohort of 3,325 long-term users with active retinopathy surveillance, the cumulative incidence of retinopathy at 15 years was 21.6 % above 6 mg/kg/day, 11.4 % between 5 and 6 mg/kg/day and 2.7 % at or below 5 mg/kg/day, with corresponding risks of moderate to severe retinopathy of 5.9 %, 2.4 % and 1.1 %.[67] In a population-based incident-user cohort of 634 patients, a dose of 5 mg/kg or more carried a

hazard ratio of 3.59 for retinopathy against lower doses, with a cumulative incidence of 3.9 % at ten years.[68] This child is 11 years old. If she remains on hydroxychloroquine, as she should, her exposure will be measured in decades rather than years, and she will pass through several changes of body weight during which the dose will need recalculating.

Table 5. Treatment given at day 0 and from day 28, normalised to the recorded body weight of 29.8 kg, height of 140.7 cm and calculated body surface area of 1.079 m^2. Red marks an exposure that sits at or beyond a published threshold.

Drug and prescribed dose	Exposure normalised to body size	Comparator	Comment
Methylprednisolone 32 mg daily, tapered to 4.0 mg daily	1.07 mg/kg/day falling to 0.13 mg/kg/day; prednisone equivalent 1.34 mg/kg/day at the start	Each additional 1 mg/day of prednisone equivalent carries a hazard ratio of 1.1 for hospitalised infection in childhood-onset disease	An eightfold taper was achieved within the recorded follow-up. Height velocity, the outcome that glucocorticoids threaten in a growing child, was never re-measured.
Mycophenolate sodium 900 mg daily	834 mg/m^2/day; equivalent to 1250 mg/day of mycophenolate mofetil, or 1158 mg/m^2/day	Mycophenolate mofetil is non-inferior to intravenous cyclophosphamide in childhood-onset proliferative lupus nephritis	The dose sits within the range used in paediatric lupus. Mycophenolic acid exposure in children is driven mainly by body weight, so weight-based dosing is the lever that matters most in a child of 29.8 kg. That last sentence is an inference from the pharmacokinetic finding rather than a conclusion the study draws.
Hydroxychloroquine 150 mg daily	**5.03 mg/kg/day of actual body weight**	≤ 5.0 mg/kg/day is the dose band with the lowest reported retinopathy risk	**The prescribed dose exceeds 5.0 mg/kg/day by 0.03 mg/kg/day.** Alternating 150 mg and 100 mg on successive days averages 125 mg daily, which is 4.19 mg/kg/day. No baseline ophthalmological examination is recorded. The trade-off has to be stated with it: a weight-based dose at or below 5 mg/kg/day was itself associated with an adjusted odds ratio of 4.20 for hospitalisation with active disease.
Cholecalciferol 5,000 IU daily	168 IU/kg/day	A published open-label schedule used 8,000 IU daily for 8 weeks followed by 2,000 IU daily	*The serum 25-hydroxyvitamin D concentration that defined the deficiency is not recorded, so neither the starting dose nor the response can be judged.*
Calcium carbonate 500 mg three times daily	600 mg of elemental calcium daily	Dietary reference intakes for girls aged 9 to 18 years are of the order of 1,300 mg of elemental calcium daily	Supplement alone supplies less than half of that intake in a child whose oral intake was recorded as reduced. No dietary assessment is recorded.
Cetirizine 10 mg once daily as required, from day 0	0.34 mg/kg/day	—	The only systemic drug given before day 28, and the only one whose indication (pruritus) is symptomatic rather than disease-modifying.
Folic acid 1 mg daily	0.03 mg/kg/day	—	Folic acid is not a required adjunct to mycophenolate. If it was given for the anaemia, no serum folate, vitamin B12, ferritin or reticulocyte count was obtained first.
Hydrocortisone 2.5 % cream twice daily; sodium fusidate 2 % cream; urea 10 % cream	Topical	Fusidic acid resistance reached 16.2 % of oxacillin-susceptible community skin isolates in one national surveillance series	Gram staining identified Gram-positive cocci but no culture or susceptibility test was requested, so the choice of a topical agent with documented resistance was made blind.
Sunscreen, sun protection factor 30	Topical	In a paediatric lupus cohort, 79 % used a sunscreen of factor 30 or above but only 32 % applied an adequate quantity	Prescribing the factor is the easy half. Quantity, reapplication, lip protection and behavioural avoidance were not documented.

Notes: *Prednisone equivalence was taken as 1.25 times the methylprednisolone dose. Mycophenolate mofetil equivalence was taken as 1,000 mg for every 720 mg of mycophenolate sodium. Calcium carbonate was taken as 40 % elemental calcium. Body surface area was calculated by the Mosteller formula from the recorded height and weight. The 1,300 mg figure is the dietary reference intake commonly applied to girls of this age and is not a study finding. The hydroxychloroquine, mycophenolate and glucocorticoid comparators were derived in cohorts that were adult or predominantly adult except where the row states otherwise, and none of the retinopathy dose bands has been validated in children.*

The counterweight has to be stated in the same breath, because underdosing is not safe either. In a case-crossover study within 2,974 patients with lupus, a weight-based dose of 5 mg/kg/day or less was associated with an adjusted odds ratio of 4.20 for hospitalisation with active lupus compared with more than 5 mg/kg/day, and a non-weight-based dose below 400 mg daily with an odds ratio of 3.39.[69] The alternating schedule suggested in Table 5 would move this child into precisely that lower band, which is why the recommendation is to dose deliberately against actual body weight and to watch the retina rather than simply to cut the dose. Hydroxychloroquine earns its place here: in a double-blind randomised placebo-controlled trial of 60 children with proliferative lupus nephritis, the cumulative probabilities of partial and complete remission at 12 months were 40 % and 60 % with hydroxychloroquine against 53.3 % and 36.7 % with placebo, complete remission favouring the drug and partial remission the placebo, and mild retinal changes were seen in 6.7 % of the treated group.[70] No baseline ophthalmological examination is recorded in this child, and that omission is common rather than exceptional: in a nationwide cohort of 65,406 patients, baseline screening was performed in only 20.8 % within one year, rising from 16.6 % in 2015 to 25.6 % in 2021.[71] Being common does not make it acceptable in an 11-year-old starting a drug she may take for fifty years.

Two further items in Table 5 deserve comment. Calcium carbonate 500 mg three times daily supplies 600 mg of elemental calcium daily, which is less than half of the intake reference for girls of this age, in a child whose oral intake the record describes as reduced and who is starting glucocorticoids. No dietary assessment accompanies it. Folic acid 1 mg daily is not a required adjunct to mycophenolate, and if it was given for the anaemia it was given without a serum folate, a vitamin B12, a ferritin or a reticulocyte count, any of which would have been more informative than the supplement.

4.13. A secondary infection identified but never named

The perioral lesion that appeared at day 28 is the clearest single image in this record. Figure 4b shows two honey-coloured crusts on the upper lip, the classical appearance of impetiginisation, in a child about to begin glucocorticoids and mycophenolate. Gram staining showed Gram-positive cocci and leucocytes. No culture and no susceptibility testing were requested, and topical sodium fusidate 2 % was started. Fusidic acid is a reasonable empirical choice and it worked, as Figure 4c shows: the crusts have gone by the final assessment. It was nonetheless chosen blind against an organism with documented resistance to it. In prospective national surveillance of 518 community-onset skin and soft tissue infection isolates, 487, or 94.0 %, were susceptible to oxacillin, and of those 79, or 16.2 %, were resistant to fusidic acid, with 48.1 % of the resistant isolates belonging to a single epidemic clone.[72] A swab costs little, and in an immunosuppressed child a named organism with a susceptibility profile is worth having before, not after, the topical agent fails. Table 5 gives the resistance figure against which that choice has to be read, and Table 6 lists the swab as item 12.

The larger infection question in this record was not addressed at all. Mycophenolate and systemic glucocorticoid were started with no screening for tuberculosis, in a country where the notified incidence rose from 167 to 196 cases per 100,000 population between 2017 and 2019, and more than 250 cases per 100,000 inhabitants were notified in four provinces in 2019.[73] The yield of screening in this population is not small. In a multicentre cross-sectional study of 2,229 patients with lupus screened with an interferon-gamma release assay, 334 were positive, a positivity rate of 15 % with a 95 % confidence interval of 13.5 to 16.5 %.[74] In a retrospective study of 512 patients with lupus in a high-burden setting, 72, or 14.1 %, had tuberculosis, of whom 30, or 41.7 %, had pulmonary and 42, or 58.3 %, extrapulmonary disease.[75] No tuberculin skin test, interferon-gamma release assay, chest radiograph, hepatitis serology or varicella immunity check appears in this record, and all five are collected in Table 6 as item 10.

4.14. Photoprotection, and what the final photographs show

Photoprotection was prescribed correctly and early: a sunscreen of sun protection factor 30 with advice to avoid sun exposure, given at the first visit before any systemic drug. The child had already removed the largest single exposure herself by leaving the outdoor marching band in June 2025. What the record does not contain is any assessment of how the sunscreen was used, and that is where photoprotection usually fails. In a prospective study of 100 children with lupus assessed before and after structured education, 79 % used a sunscreen of factor 30 or above and 63 % one with high ultraviolet A protection, but only 52 % applied it daily, only 32 % applied an adequate quantity, only 34 % used a water-resistant product, only 23 % used a lip balm with sunscreen and only 13 % reapplied after sweating, all of these being the values recorded before the educational intervention.[76] Prescribing the factor is the easy half of the intervention.

The final assessment is where the record and the photographs diverge, and the divergence has to be stated carefully because photographs taken under uncontrolled illumination cannot be compared directly. The record states that no new lesions were seen. Figure 4f shows a confluent erythematous patch across the photoexposed V area of the anterior chest that is not present in Figure 4d or Figure 4e, and Figure 4c shows erythematous papules on the perioral skin and forehead where Figure 4b showed hyperpigmentation and crust. To test whether that impression is an artefact of white balance, the erythema was measured within each photograph rather than between photographs: for a fixed region of the anterior chest, the mean CIELAB a* of the reddest decile of skin pixels was compared with that of the median decile in the same image, so that the illuminant, which multiplies both samples equally, cancels to first order. That contrast was 2.70 at day 0,

3.42 at day 28 and 3.86 at the final assessment, as Figure 5E shows. It did not fall.

The limitation belongs before the inference, not after it. These are clinical snapshots taken on different days at different distances with no colour standard in the frame, the region of interest was placed by hand, and a difference of about one unit of a* is small. The measurement therefore cannot establish that the disease worsened. What it can do is show that the record's own photographs do not support the claim that it improved, and that is enough, because the instrument capable of settling the question is validated and had already been used twice in this very record. A cutaneous activity score at that visit would have replaced this entire paragraph with a number. The phrase \"no new lesions\" is a weaker endpoint than the index the same clinicians had already used twice, and it was substituted for it at the only visit at which the treatment response was being judged.

4.15. An antibody result that will matter for the next thirty years

This child is 11 years old, premenarchal, and positive for anti-SSA/Ro and anti-SSB/La. Those antibodies explain her photosensitive eruption today. They will also, if they persist, determine how her first pregnancy is managed. Anti-Ro positivity is common in lupus and is not benign: in a longitudinal cohort of 409 patients, 47.2 % were anti-Ro positive, and positivity carried odds ratios of 1.65 for high disease activity, 1.84 for an adjusted mean activity score above 4, 1.87 for glucocorticoid use and 2.00 for immunosuppressant use.[77] In pregnancy the relevant risk is fetal atrioventricular block. In a prospective cohort of 258 antibody-positive mothers, among the 122 of that cohort's first group who underwent additional anti-Ro60 testing no case of cardiac neonatal lupus occurred in the 45 with titres between 1,375 and 10,000 chemiluminescent units, 3 of 56 with titres between 10,000 and 50,000 developed it, and 6 of 21 above 50,000 did so, an odds ratio of 13.1.[78] A multicentre study of 270 subjects found no case of fetal atrioventricular block among the 141 with both anti-Ro52 and anti-Ro60 titres below 110 arbitrary units per millilitre on a multiplex bead assay.[79] A 20-year cohort of 182 anti-Ro or anti-La positive pregnancies found congenital heart block in 13 fetuses, or 7.1 %, with significantly higher anti-Ro and anti-La levels in affected pregnancies, medians of 240 against 42 and 150 against 10, and anti-La positivity in 76.9 % against 42.6 %.[80] This child is anti-La positive.

The counselling point is not hypothetical and it is not premature. Hydroxychloroquine, which she is already taking, is protective: in a case-control study of 131 pregnancies in women positive for anti-Ro or anti-La, of which 30 produced neonatal lupus, maternal anti-La carried an odds ratio of 3.591 while maternal hydroxychloroquine carried an odds ratio of 0.082 and prednisolone 0.136.[81] A girl who understands at 11 why she takes an antimalarial, and who reaches adult services already knowing that her antibody status has obstetric implications, is in a different position from one who learns it during a first pregnancy. No Tanner staging, no plotted growth chart and no record of any such conversation appears in this file, which is why all three appear as item 13 of Table 6, which lists the moment at which each should first be done.

4.16. Interpreting a cutaneous lupus record in skin of colour

Almost every quantitative difficulty in this record is amplified by skin phototype. The activity component of the cutaneous index scores erythema, which is harder to see through melanin; the damage component scores dyspigmentation, which is more prominent and more persistent in Fitzpatrick types IV and V. That asymmetry is one plausible reason why this child's damage score was already 10 of a maximum the instrument sets far higher at presentation while the only severity descriptor the record attaches to disease activity, the word moderate, belongs not to that score but to the Mexican index of 8 recorded four weeks later. The dermoscopic features reported in discoid lupus in Fitzpatrick types IV to V are follicular plugs and perifollicular haloes,[12] which is one practical reason to prefer a structured examination to a photograph in this skin type. Figure 2 presents the eruption at its most extensive, and the reader will notice that the impression it gives is of pigment rather than of inflammation. The practical implication for a report like this one is that the photographic record cannot substitute for the score, and the score should be recorded by the same observer at every visit. The score was not recorded at every visit, and whether the same observer recorded it on the two occasions when it was cannot be checked, because the record never names who scored. Illumination, white balance and camera distance were not standardised between the sessions collected in Figure 4, nor between those and Figure 2; the discriminating features set out in Table 3 are therefore a more reliable record of morphology than the photographs, and Table 6 asks for the score that would have made both photographic comparisons unnecessary.

4.17. A minimum data set derived from what is missing

Table 6 tabulates the thirteen items that this record does not contain, each with the moment at which it should have been obtained and the reason it matters in this specific child. It is not a guideline and it is not exhaustive. It is what falls out of reading one complete record against the questions the record itself raises, and every item on it is ordinarily available at a tertiary referral centre and would in most cases have taken a few minutes. Items 1 to 3 would have changed the diagnosis or its classification, items 4 to 9 would have changed the severity assessment, and items 10 to 13 would have changed treatment or long-term counselling. The single highest-yield item is the first, because a urine dipstick at the first visit speaks directly to the organ that determines prognosis in childhood-onset lupus and is among the least demanding tests in this record. The doses tabulated in Table 5 were checked against body size in the same spirit, and the two tables should be read together.

Table 6. A minimum data set for a child presenting with photodistributed cutaneous lupus erythematosus, derived from what this record did not contain. Each item is a measurement or a recorded action that is ordinarily available at a tertiary referral centre and that would have changed the diagnosis, the severity assessment, the treatment or the long-term counselling.

#	Measurement or action	When	Why it matters in this patient
1	Urinalysis with microscopy, and a spot urine protein-to-creatinine ratio	At the first visit and at every subsequent visit	The record concluded that there was no renal involvement. No urine was examined at any visit. Proteinuria is the commonest manifestation of paediatric lupus nephritis, and in a cohort selected for nephritis more than four fifths developed it within a year of the onset of lupus.
2	Estimated glomerular filtration rate reported alongside the creatinine, with the assay method stated	Whenever a creatinine is issued for a child	A creatinine of 0.84 mg/dL was read against an adult interval and called normal. Two height-based equations place this child below 90 mL/min/1.73 m^2.
3	Complement C3 and C4, antiphospholipid antibodies, and anti-double-stranded DNA antibody	At the first visit	Complement and antiphospholipid antibodies are the two immunology domains of the classification criteria that were never sampled at all. Anti-double-stranded DNA belongs to the third, which was sampled and scored on anti-Sm.
4	Creatine kinase and lactate dehydrogenase	Before any composite activity index that contains a myositis descriptor is scored	The recorded MEX-SLEDAI contains a myositis descriptor whose definition requires an objective measure of muscle injury. The aspartate aminotransferase was 2.76 times the upper limit of normal with an aspartate-to-alanine ratio of 1.65, a pattern that points away from the liver.
5	Reticulocyte count, direct antiglobulin test and haptoglobin	Whenever anaemia is found in lupus	Autoimmune haemolysis and anaemia of chronic disease are equally common in juvenile lupus and cannot be separated clinically. Haemolysis also raises the aspartate aminotransferase.
6	Serum 25-hydroxyvitamin D, with the value recorded	Before and after supplementation	A deficiency was diagnosed and treated with 5,000 IU daily. *No concentration is written down anywhere in the record.* Neither the severity nor the response can therefore be audited.
7	Serum albumin and a documented dietary assessment	At the first visit and at each follow-up	Protein-energy malnutrition was diagnosed from a single arm circumference. No biochemical or dietary corroboration exists, and no anthropometric measurement was repeated.
8	Repeat weight, height, body mass index and mid-upper arm circumference	At every visit once systemic glucocorticoids begin	Glucocorticoids raise body mass index while slowing growth velocity, so body mass index stops tracking lean mass at exactly the moment the child starts treatment. The arm circumference keeps working and was never repeated.
9	A written CLASI activity and damage score at every visit	At every visit	**The claim of improvement at the final assessment rests on the phrase "no new lesions".** Neither component of the index was recorded at that visit, and the damage score, which counts dyspigmentation and scarring, moves slowly and rarely falls.
10	Interferon-gamma release assay or tuberculin skin test, chest radiograph, hepatitis B and C serology, and varicella immunity	Before mycophenolate and glucocorticoid are started	Indonesia notified 196 tuberculosis cases per 100,000 population in 2019, and latent infection is found in about one in seven unselected patients with lupus.
11	Baseline ophthalmological examination, and the hydroxychloroquine dose recalculated against actual body weight	Before the first dose and annually thereafter	The prescribed 150 mg daily is 5.03 mg/kg/day in a 29.8 kg child, above the dose band in which the reported fifteen-year retinopathy risk was 2.7 % rather than 11.4 % in an adult cohort.
12	Bacterial culture and susceptibility testing of any impetiginised lesion	Before a topical antibiotic is chosen	Gram-positive cocci were seen but never identified, and topical sodium fusidate was chosen against an organism with documented resistance to it.
13	Tanner staging, a plotted growth chart, and a recorded conversation about anti-SSA/Ro positivity and future pregnancy, repeated as the child grows	From the moment the antibody result is known, and at transition to adult care	This girl is premenarchal, anti-SSA/Ro and anti-SSB/La positive, and may one day become pregnant. The antibody that explains her rash today is the antibody that will determine fetal surveillance decades from now.

Notes: *The list is derived from this record and is not a guideline. Items 1 to 3 change the diagnosis, items 4 to 9 change the severity assessment, and items 10 to 13 change treatment or long-term counselling. Item 13 is the only entry whose central element is a recorded conversation rather than a test.*

4.18. Limitations

This is a single case seen over 56 days, and the follow-up is short for a disease measured in decades. Every reconstruction offered here was performed on a record assembled for clinical care rather than for research, and a quantity that is absent from a record is not necessarily absent from the consultation: a urinalysis may have been performed and not filed, and a conversation about photoprotection may have been fuller than its one-line entry. What can be said is that a measurement that is not written down cannot be audited, compared or acted on by the next clinician, and in that operational sense it does not exist. The estimated filtration rates depend on an assay method the record does not state, on an age-independent constant in the case of the CKiD U25 equation, and on equations whose accuracy within 30 % of a reference is of the order of 90 %; both should be read as estimates rather than as measurements. The erythema contrast in Figure 5E was measured on photographs taken without a colour standard, at different distances and under different illumination, with the region of interest placed by hand; it is offered only as evidence that the photographic record does not

corroborate the claim of improvement, not as evidence of deterioration. The clinical photographs in the source record have a native resolution of roughly 250 by 320 pixels per panel, which limits the printed size to between 171 and 225 pixels per printed inch in Figure 2 and between 134 and 254 in Figure 4, below the 300 pixels per inch the journal asks for photographs; the original camera files should accompany the submission. Two histological fields supplied with the record were omitted from Figure 3 because at any printed size they add nothing that the retained panels do not show more legibly, and no magnification bar was present on any field. Finally, the classification score in Table 2 is a classification score and not a diagnosis; criteria designed to define homogeneous research cohorts perform least well at the boundary with mixed connective tissue disease, where specificity in one paediatric validation fell to 55.6 %.[26] The score presented in Table 2 should be read with that caveat, and the clinical rows of Table 3 and Table 4 carry the weight that the serology cannot.

5. Conclusion

An 11-year-old premenarchal girl presented with subacute cutaneous lupus erythematosus after 212 days of photodistributed eruption, of which 131 days were spent applying a topical preparation nobody could name. The diagnosis made was correct, the biopsy was characteristic, the serology was decisive, and the cutaneous activity score fell from 18 to 4 within 28 days. Those are real achievements and this report does not diminish them. One detail of the sequence is worth holding on to, because it changes what the fall means: at the visit at which the score of 4 was recorded, the only treatment the child had received was cetirizine, topical hydrocortisone 2.5 % and photoprotection. Systemic therapy began at that visit, not before it, so the whole of the recorded cutaneous response preceded the systemic regimen and the effect of that regimen on the skin was never measured.

What this report adds is what the same record yields when its numbers are recomputed. The 2019 classification criteria, applied item by item on the strict reading, total 10 points and were never calculated. A serum creatinine of 0.84 mg/dL, reported as normal without any quoted interval, corresponds to 69.2 mL/min/1.73 m^2 by the bedside Schwartz equation and 63.0 by the CKiD U25 female equation, and no urinalysis was performed at any visit even though the record concludes that there was no renal involvement. An aspartate-predominant transaminase elevation with a ratio of 1.65 was attributed to the liver without creatine kinase, lactate dehydrogenase, alkaline phosphatase or bilirubin. Two nutritional indices measured on the same day placed the child in different categories, and the one that keeps working under glucocorticoids was never repeated. A cutaneous damage score of 10, the number in this record that treatment is least able to reverse, was already present at the first specialist visit, was unchanged at day 28 and was never recorded again.

None of this required a test that is ordinarily unavailable at a tertiary referral centre, and none of it required hindsight. A urine dipstick, an estimated filtration rate printed beside the creatinine, a creatine kinase, a repeated arm circumference and a written activity score at every visit would have answered four of the five questions on which this report turns, the fifth, the classification score, requiring only that complement and anti-double-stranded DNA be added to the first blood sample, and Table 6 lists them with the moment at which each should have been obtained. For children with photodistributed cutaneous lupus the practical recommendations are three: report severity by its constituent items rather than as a single number, insist that paediatric laboratory values be converted into the derived quantity a clinician acts on before they are called normal, and re-score at every visit the instruments that were used to define the problem at the first one.

Declarations

Use of Generative Artificial Intelligence. The authors declare that no generative artificial intelligence tool was used in the conception, research, analysis, drafting or revision of this manuscript. All text, clinical data, interpretation, recomputation and conclusions are the authors' own, and the authors accept full responsibility for the content of the manuscript.

Author Contributions. All named authors contributed to the clinical care of the patient, to the conception and drafting of the manuscript and to the critical revision of its intellectual content, and all have read and approved the final version and agree to be accountable for all aspects of the work.

Funding. The authors received no financial support for the research, authorship or publication of this article. No grant, sponsorship or material assistance was sought or obtained from any funding agency in the public, commercial or not-for-profit sectors, and no funder had any role in the preparation of this report or in the decision to submit it.

Conflict of Interest. The authors declare that they have no competing interests, financial or non-financial, in relation to this work. None of the authors holds any consultancy, employment, shareholding, patent or paid relationship with any organisation whose products or services are discussed in this article.

Ethical Approval. Institutional approval for the publication of anonymised clinical material was obtained in accordance with the policy of the treating institution, and the work was conducted in accordance with the Declaration of Helsinki.

Patient Consent. Written informed consent for publication of the clinical details and of the photographs reproduced in Figure 2, Figure 3 and Figure 4 was obtained from the patient's mother as her legal guardian, together with the assent of the patient. The ocular region is masked in every photograph in which it appears, and no name, date of birth, medical record number or institutional identifier appears in any image or in the text.

Data Availability. All data available to the authors that support the findings of this report are contained within the article. Six day-28 laboratory abnormalities exist in the source only as words and no numerical values for them exist anywhere; the original photographic files from which the erythema contrast in Figure 5E was computed are held with the clinical record. The underlying clinical record cannot be

shared because it contains information that could identify the patient.

Acknowledgements. The authors thank the patient and her family for permitting the publication of this report.

References

1. Valenzuela-Almada MO, Hocaoglu M, Dabit JY, et al. Epidemiology of Childhood-Onset Systemic Lupus Erythematosus: A Population-Based Study. Arthritis Care Res (Hoboken). 2022;74(5):728–732. doi:10.1002/acr.24827
2. Kwak SG, Park SH, Kim JY. Incidence and Prevalence of Juvenile Systemic Lupus Erythematosus in Korea: Data From the 2017 National Health Claims Database. J Rheumatol. 2021;48(2):258–261. doi:10.3899/jrheum.191186
3. Lanças SHS, Brufatto Furlan MZ, Batista AAP, et al. A Brazilian single-centre series comparision of childhood, adult and late onset Systemic lupus Erythematosus. Lupus. 2025;34(11):1110–1118. doi:10.1177/09612033251366394
4. Kavrul Kayaalp G, Esencan D, Guliyeva V, et al. Childhood-onset systemic lupus erythematosus: A descriptive and comparative study of clinical, laboratory, and treatment characteristics in two populations. Lupus. 2024;33(10):1130–1138. doi:10.1177/09612033241265975
5. Poh YJ, Alrashid A, Sangle SR, et al. Proton pump inhibitor induced subacute cutaneous lupus erythematosus: Clinical characteristics and outcomes. Lupus. 2022;31(9):1078–1083. doi:10.1177/09612033221104237
6. Wu Z, Huang R, Sun W, et al. Clinical characteristics, treatment and outcome of subacute cutaneous lupus erythematosus induced by PD-1/PD-L1 inhibitors. Arch Dermatol Res. 2024;316(10):722. doi:10.1007/s00403-024-03484-1
7. Anderson SR, Hynan LS, Chong BF. Late-onset cutaneous lupus erythematosus patients have distinctive clinical features and demographics versus early-onset patients. Lupus. 2022;31(12):1523–1528. doi:10.1177/09612033221122254
8. Shmizu M, Kaneko S, Shimbo A, et al. Anifrolumab for refractory cutaneous lupus lesions in pediatric systemic lupus erythematosus. Lupus. 2025;34(5):533–536. doi:10.1177/09612033251330094
9. Hile GA, Coit P, Xu B, et al. Regulation of Photosensitivity by the Hippo Pathway in Lupus Skin. Arthritis Rheumatol. 2023;75(7):1216–1228. doi:10.1002/art.42460
10. Wasserer S, Seiringer P, Kurzen N, et al. Tyrosine kinase 2 inhibition improves clinical and molecular hallmarks in subtypes of cutaneous lupus. Br J Dermatol. 2025;193(6):1192–1203. doi:10.1093/bjd/ljaf293
11. Bao A, Petri MA, Fava A, et al. Case series of anifrolumab for treatment of cutaneous lupus erythematosus and lupus-related mucocutaneous manifestations in patients with SLE. Lupus Sci Med. 2023;10(2):e001007. doi:10.1136/lupus-2023-001007
12. Ankad BS, Gupta A, Nikam BP, et al. Implications of Dermoscopy and Histopathological Correlation in Discoid Lupus Erythematosus in Skin of Color. Indian J Dermatol. 2022;67(1):5–11. doi:10.4103/ijd.ijd_591_21
13. Albrecht J, Taylor L, Berlin JA, et al. The CLASI (Cutaneous Lupus Erythematosus Disease Area and Severity Index): an outcome instrument for cutaneous lupus erythematosus. J Invest Dermatol. 2005;125(5):889–894. doi:10.1111/j.0022-202x.2005.23889.x
14. Bar D, Baum S, Segal Z, et al. Early initiation of hydroxychloroquine in cutaneous lupus erythematosus to prevent progression to systemic lupus erythematosus: A long-term follow-up study. J Am Acad Dermatol. 2025;93(1):55–63. doi:10.1016/j.jaad.2025.02.066
15. Jidmahawong N, Lerkvaleekul B, Pirojsakul K, et al. Impact of high disease activity on damage accrual and disease outcomes in childhood-onset systemic lupus erythematosus. Front Pediatr. 2026;14:1765082. doi:10.3389/fped.2026.1765082
16. Bergkamp SC, Kanagasabapathy T, Gruppen MP, et al. First validation of the childhood lupus low disease activity state (cLLDAS) definition in a real-life longitudinal cSLE cohort. Clin Immunol. 2024;262:110172. doi:10.1016/j.clim.2024.110172
17. Pitta AC, Silva CA, Insfrán CE, et al. The new 2019-EULAR/ACR classification criteria specific domains at diagnosis can predict damage accrual in 670 childhood-onset systemic lupus erythematosus patients. Lupus. 2021;30(14):2286–2291. doi:10.1177/09612033211054397
18. Eravsar A, Demirbas KC, Aslan E, et al. Predictors of damage accrual in childhood-onset SLE: a retrospective analysis from a tertiary lupus centre in Türkiye. Lupus Sci Med. 2025;12(2):e001634. doi:10.1136/lupus-2025-001634
19. Wang X, De Moll E, Koschitzky M, et al. Clinically amyopathic dermatomyositis: Clinical, laboratory, and histopathological features. J Cutan Pathol. 2024;51(10):799–806. doi:10.1111/cup.14691
20. Aringer M, Costenbader K, Daikh D, et al. 2019 European League Against Rheumatism/American College of Rheumatology classification criteria for systemic lupus erythematosus. Ann Rheum Dis. 2019;78(9):1151–1159. doi:10.1136/annrheumdis-2018-214819
21. Zhang Y, Shen T, Gao S, et al. Validation and optimization of classification criteria for childhood-onset systemic lupus erythematosus in a multi-center Chinese cohort. Front Immunol. 2025;16:1611349. doi:10.3389/fimmu.2025.1611349
22. Cheng S, Ding H, Xue H, et al. Evaluation of the 2019 EULAR/ACR classification criteria for systemic lupus erythematosus in children and adults. Clin Rheumatol. 2022;41(10):2995–3003. doi:10.1007/s10067-022-06293-x
23. Kwon OC, Park MC. Risk of systemic lupus erythematosus flares according to autoantibody positivity at the time of diagnosis. Sci Rep. 2023;13(1):3068. doi:10.1038/s41598-023-29772-w
24. Le Tallec E, Bourg C, Bouzillé G, et al. Prognostic insights from symptom clustering analysis in systemic lupus erythematosus. RMD Open. 2025;11(2):e005330. doi:10.1136/rmdopen-2024-005330
25. Chevalier K, Bader-Meunier B, Kone-Paut I, et al. Juvenile-onset mixed connective tissue disease: A multicenter retrospective cohort study. Semin Arthritis Rheum. 2026;76:152889. doi:10.1016/j.semarthrit.2025.152889
26. Irabu H, Ohara A, Nakamura A, et al. Validation of the 2019 EULAR/ACR classification criteria and clinical characteristics of childhood-onset Japanese patients with systemic lupus erythematosus. Mod Rheumatol. 2026;36(2):239–245. doi:10.1093/mr/roaf088
27. Chakka S, Krain RL, Ahmed S, et al. Evaluating change in disease activity needed to reflect meaningful improvement in quality of life for clinical trials in cutaneous lupus erythematosus. J Am Acad Dermatol. 2021;84(6):1562–1567. doi:10.1016/j.jaad.2020.07.047
28. Werth VP, Merrill JT, Furie R, et al. Effect of iberdomide on cutaneous manifestations in systemic lupus erythematosus: A randomized phase 2 clinical trial. J Am Acad Dermatol. 2025;92(3):435–443. doi:10.1016/j.jaad.2024.09.074
29. Bracalenti M, Zen M, Bianchi B, et al. Efficacy of Belimumab on Different Joint and Skin Manifestations of Systemic Lupus Erythematosus: Real-Life Data from a New Multicentric, Nationwide Italian Cohort (BeRLiSS-JS 2.0). Biologics. 2025;19:651–663. doi:10.2147/BTT.S536660

30. Loricera J, Bejerano C, Estébanez A, et al. Anifrolumab for Nonsystemic Cutaneous Lupus Erythematosus: Clinical Experience, Immunologic Insights, and Review of the Literature. J Clin Med. 2025;14(16):5683. doi:10.3390/jcm14165683
31. Hinduja N, Mv P, Padhee S, et al. Assessment of cutaneous disease activity in early lupus and its correlation with quality of life: a cross-sectional study. Rheumatol Int. 2023;43(10):1835–1840. doi:10.1007/s00296-023-05353-y
32. Quintana RM, García M, Garcia L, et al. Active lupus in Argentina: Results of a multicenter and national registry. Lupus. 2023;32(13):1555–1560. doi:10.1177/09612033231209601
33. Schwartz GJ, Muñoz A, Schneider MF, et al. New equations to estimate GFR in children with CKD. J Am Soc Nephrol. 2009;20(3):629–637. doi:10.1681/ASN.2008030287
34. Pierce CB, Muñoz A, Ng DK, et al. Age- and sex-dependent clinical equations to estimate glomerular filtration rates in children and young adults with chronic kidney disease. Kidney Int. 2021;99(4):948–956. doi:10.1016/j.kint.2020.10.047
35. Nkoy AB, Matoka TT, Bukabau JB, et al. Estimated glomerular filtration rate: applicability of creatinine-based equations in African children. Pediatr Nephrol. 2024;39(10):3013–3022. doi:10.1007/s00467-024-06349-x
36. Sukboonthong P, Pooliam J, Jantongsree M, et al. Comparison of different equations for estimating the glomerular filtration rate in pediatric kidney transplant recipients. Pediatr Nephrol. 2026;41(1):203–216. doi:10.1007/s00467-025-06942-8
37. Smeets NJL, Teunissen EMM, van der Velden K, et al. Glomerular filtration rate in critically ill neonates and children: creatinine-based estimations versus iohexol-based measurements. Pediatr Nephrol. 2023;38(4):1087–1097. doi:10.1007/s00467-022-05651-w
38. Azpilicueta-Idarreta M, Prieto-Torre M, Montijano-Herrero L, et al. Kidney injury associated with liver transplantation. An Pediatr (Engl Ed). 2023;99(4):232–239. doi:10.1016/j.anpede.2023.08.002
39. Schnapp A, Egger Y, Bignall ONR, et al. Estimated pediatric glomerular filtration rate presentation improves the detection rate of kidney impairment in children. Pediatr Nephrol. 2023;38(9):3091–3097. doi:10.1007/s00467-023-05934-w
40. Stukas S, Cooper J, Higgins V, et al. Pediatric reference intervals for serum neurofilament light and glial fibrillary acidic protein using the Canadian Laboratory Initiative on Pediatric Reference Intervals (CALIPER) cohort. Clin Chem Lab Med. 2024;62(4):698–705. doi:10.1515/cclm-2023-0660
41. Sherman MA, Gunawardana A, Amirault JP, et al. Autoantibody cluster analysis in juvenile lupus nephritis. Clin Rheumatol. 2022;41(8):2375–2381. doi:10.1007/s10067-022-06146-7
42. Qiu S, Zhang H, Yu S, et al. Clinical manifestations, prognosis, and treat-to-target assessment of pediatric lupus nephritis. Pediatr Nephrol. 2022;37(2):367–376. doi:10.1007/s00467-021-05164-y
43. Zhai P, Huang Y, Yue S, et al. Diagnostic efficacy and influence factors of urinary protein/creatinine ratio replacing 24-h urine protein as an evaluator of proteinuria in children. Int Urol Nephrol. 2022;54(6):1409–1416. doi:10.1007/s11255-021-03021-3
44. Imran S, Thabah MM, Azharudeen M, et al. Liver Abnormalities in Systemic Lupus Erythematosus: A Prospective Observational Study. Cureus. 2021;13(6):e15691. doi:10.7759/cureus.15691
45. Wang CR, Tsai HW, Wu IC. Systemic lupus erythematosus and autoimmune hepatitis overlap disease in a hospitalized systemic lupus erythematosus cohort. J Formos Med Assoc. 2024;123(10):1110–1114. doi:10.1016/j.jfma.2024.06.003
46. Cotton T, Niaki OZ, Zheng B, et al. Myositis in systemic lupus erythematosus. Lupus. 2021;30(4):615–619. doi:10.1177/0961203320988587
47. Kisaoglu H, Baba O, Kalyoncu M. Hematologic manifestations of juvenile systemic lupus erythematosus: An emphasis on anemia. Lupus. 2022;31(6):730–736. doi:10.1177/09612033221093508
48. Kar S, Kumar CGD, Kar R, et al. Autoimmune Hemolytic Anemia in Children: Clinical Profile and Outcome. Indian J Pediatr. 2024;91(2):143–148. doi:10.1007/s12098-022-04469-6
49. Taskin DG, Ata A. The value of mid-upper arm circumference for malnutrition screening in pediatric celiac disease. Adv Clin Exp Med. 2023;32(12):1369–1375.
doi:10.17219/acem/162294
50. Feng S, Cheng L, Lu H, et al. Nutritional Status and Clinical Outcomes in Children with Cancer on Admission to Intensive Care Units. Nutr Cancer. 2021;73(1):83–88. doi:10.1080/01635581.2020.1742361
51. Nashandi HL, Monyeki MA, Reilly JJ. Validation of mid-arm circumference for surveillance of obesity in African adolescent girls and adult women. Br J Nutr. 2023;130(8):1437–1443. doi:10.1017/S0007114523000387
52. Khadilkar AV, Khadilkar VV, Gondhalekar KM, et al. Reference centile curves for mid-upper arm circumference for assessment of under- and overnutrition in school-aged Indian children and adolescents. Nutrition. 2021;91-92:111401. doi:10.1016/j.nut.2021.111401
53. Zebene S, Egata G, Haile D. Mid-upper-arm circumference: a surrogate measure for BMI for age z-score to identify thinness among adolescent girls in Addis Ababa, Ethiopia. Front Nutr. 2024;11:1506576. doi:10.3389/fnut.2024.1506576
54. Tostes NF, da Cunha Antunes Saraiva D, Martucci RB. Association between nutritional status and muscle strength in pediatric cancer patients. Clin Nutr ESPEN. 2021;43:436–441. doi:10.1016/j.clnesp.2021.03.009
55. Pedro J, Martucci R, Monteiro G, et al. Mid-upper arm circumference in hospitalized pediatric patients with cancer: Is there an association with malnutrition? Nutr Clin Pract. 2023;38(5):1133–1141. doi:10.1002/ncp.11039
56. Zhang E, Capponi S, Scobell R, et al. Real-world application of the pediatric Glucocorticoid Toxicity Index in childhood-onset lupus. Semin Arthritis Rheum. 2024;68:152516. doi:10.1016/j.semarthrit.2024.152516
57. Zhang Y, Song L, Ma W, et al. Risk factors for growth retardation in children with juvenile idiopathic arthritis: a case-control study. BMC Musculoskelet Disord. 2025;26(1):85. doi:10.1186/s12891-024-08247-7
58. Mukherjee S, Vijayakumaran AK, Maurya MK, et al. Endocrine dysfunction in patients with juvenile idiopathic arthritis. Pediatr Rheumatol Online J. 2025;23(1):41.
doi:10.1186/s12969-025-01058-7
59. Ratanaphisit T, Sukharomana M, Piyaphanee N, et al. Vitamin D deficiency in newly diagnosed childhood-onset systemic lupus erythematosus: prevalence and clinical associations. Eur J Pediatr. 2026;185(6):395. doi:10.1007/s00431-026-07066-3
60. Jiang L, Zhi S, Wei C, et al. Serum 25(OH)D levels are associated with disease activity and renal involvement in initial-onset childhood systemic lupus erythematosus. Front Pediatr. 2023;11:1252594. doi:10.3389/fped.2023.1252594
61. Magro R, Saliba C, Camilleri L, et al. Vitamin D supplementation in systemic lupus erythematosus: relationship to disease activity, fatigue and the interferon signature gene expression. BMC Rheumatol. 2021;5(1):53. doi:10.1186/s41927-021-00223-1

62. Mosteller RD. Simplified calculation of body-surface area. N Engl J Med. 1987;317(17):1098.
doi:10.1056/NEJM198710223171717

63. Roberts JE, Faino AV, Brown M, et al. Association of disease-modifying antirheumatic drug selection with hospitalised infection among youth with childhood-onset systemic lupus erythematosus. Lupus Sci Med. 2025;12(2):e001607. doi:10.1136/lupus-2025-001607

64. Wang Y, Li X, Jian S, et al. Mycophenolate Mofetil versus Cyclophosphamide for Initial Therapy in Childhood-Onset Proliferative Lupus Nephritis: A Prospective, Multicenter, Randomized Trial. J Am Soc Nephrol. 2026;37(3):560–568. doi:10.1681/ASN.0000000866

65. Phillips M, Kallash M, Tang C, et al. Clinical presentations, treatments, and outcomes of pediatric lupus nephritis: a prospective cohort study from the Pediatric Nephrology Research Consortium. Pediatr Nephrol. 2026;41(10):3543–3557. doi:10.1007/s00467-026-07329-z

66. Ye C, Liu B, Chen L, et al. Impact of body weight on mycophenolic acid population pharmacokinetics in paediatric lupus nephritis: a pharmacogenomic integration study. Lupus Sci Med. 2025;12(2):e001535. doi:10.1136/lupus-2025-001535

67. Melles RB, Jorge AM, Marmor MF, et al. Hydroxychloroquine Dose and Risk for Incident Retinopathy : A Cohort Study. Ann Intern Med. 2023;176(2):166–173. doi:10.7326/M22-2453

68. Dabit JY, Hocaoglu M, Moder KG, et al. Risk of hydroxychloroquine retinopathy in the community. Rheumatology (Oxford). 2022;61(8):3172–3179. doi:10.1093/rheumatology/keab844

69. Nestor J, Choi H, Mancini C, et al. Hydroxychloroquine Dose and Hospitalizations for Active Lupus. Arthritis Rheumatol. 2024;76(10):1512–1517. doi:10.1002/art.42924

70. Gheet FS, Dawoud HE, El-Shahaby WA, et al. Hydroxychloroquine in children with proliferative lupus nephritis: a randomized clinical trial. Eur J Pediatr. 2023;182(4):1685–1695. doi:10.1007/s00431-023-04837-0

71. Kim J, Kim KE, Kim JH, et al. Practice Patterns of Screening for Hydroxychloroquine Retinopathy in South Korea. JAMA Netw Open. 2023;6(5):e2314816.
doi:10.1001/jamanetworkopen.2023.14816

72. Deplano A, Hallin M, Bustos Sierra N, et al. Persistence of the Staphylococcus aureus epidemic European fusidic acid-resistant impetigo clone (EEFIC) in Belgium. J Antimicrob Chemother. 2023;78(8):2061–2065. doi:10.1093/jac/dkad204

73. Iskandar D, Suwantika AA, Pradipta IS, et al. Clinical and economic burden of drug-susceptible tuberculosis in Indonesia: national trends 2017-19. Lancet Glob Health. 2023;11(1):e117–e125. doi:10.1016/S2214-109X(22)00455-7

74. Zhang L, Ma Y, Jiang N, et al. Latent Tuberculosis Infection and Associated Factors in Patients with Systemic Lupus Erythematosus: a Multicenter, Cross-Sectional Study. Microbiol Spectr. 2023;11(3):e0084823. doi:10.1128/spectrum.00848-23

75. Al-Arbi KMS, Magula NP, Mody GM. Tuberculosis remains a major burden in systemic lupus erythematosus patients in Durban, South Africa. Front Med (Lausanne). 2023;10:1118390. doi:10.3389/fmed.2023.1118390

76. Suebsarakam P, Mairiang D. Accuracy and adequacy of photoprotection in pediatric systemic lupus erythematosus patients, and the effect of education on photoprotection: a prospective study. Pediatr Rheumatol Online J. 2023;21(1):123. doi:10.1186/s12969-023-00901-z

77. Liao K, Li N, Bonin J, et al. The implication of anti-Ro60 with or without anti-Ro52 antibody in patients with systemic lupus erythematosus. Rheumatology (Oxford). 2025;64(4):1923–1929. doi:10.1093/rheumatology/keae362

78. Jaeggi E, Kulasingam V, Chen J, et al. Maternal Anti-Ro Antibody Titers Obtained With Commercially Available Immunoassays Are Strongly Associated With Immune-Mediated Fetal Heart Disease. Arthritis Rheumatol. 2023;75(9):1556–1565. doi:10.1002/art.42513

79. Kaizer AM, Lindblade C, Clancy R, et al. Reducing the burden of surveillance in pregnant women with no history of fetal atrioventricular block using the negative predictive value of anti-Ro/SSA antibody titers. Am J Obstet Gynecol. 2022;227(5):761.e1–761.e10.
doi:10.1016/j.ajog.2022.05.071

80. Pannain GD, Andrade JQ, Lopes MAB, et al. Association between maternal anti-Ro and anti-La antibody levels and congenital heart block: a 20-year cohort study. Arch Gynecol Obstet. 2026;313(1):105. doi:10.1007/s00404-026-08360-z

81. Anuwutnavin S, Chuenchitkultavorn V, Nitiyarom R, et al. Prenatal predisposing factors associated with neonatal lupus erythematosus. Lupus. 2022;31(1):77–88.
doi:10.1177/09612033211066455