Lichenoid Chronic Graft-vs-Host Disease Following Blaschko Lines

Enfermedad del injerto contra el huésped crónica liqueenoide con patrón blaschkoide

Graft-vs-host disease (GVHD) is a clinical syndrome that occurs when immunocompetent donor cells attack various host tissues, with the skin, gastrointestinal tract, and liver being the main target organs. The onset of skin involvement in chronic GVHD is usually more than 100 days posttransplant. Signs basically consist of lichenoid eruptions and sclerodermoid manifestations, although many other patterns have been described. We report a new case of lichenoid chronic GVHD following Blaschko lines.

A 16-year-old boy was seen for a pruritic, linear rash on his left upper limb and the trunk that had appeared 2 weeks earlier. The patient had previously been diagnosed with a blastic plasmacytoid dendritic cell neoplasm, which was in complete remission following chemotherapy and an allogeneic bone marrow transplant with complete hematopoietic chimerism. He also had a history of acute cutaneous and intestinal GVHD. At the time of consultation, 20 months after the transplant, the patient was on low doses of methylprednisolone and cyclosporin and reported no history of herpes zoster.

Physical examination revealed 2 linear eruptions on the left upper limb—1 posteroexternal—extending from the shoulder to the distal part of the dorsum of the first, third, fourth, and fifth fingers, respectively, of the left hand. The patient also had 3 similar S-shaped lesions on the left hemithorax that followed Blaschko lines (Fig. 1, A-C). The lesions consisted of flat, erythematous-violaceous papules measuring 1 to 3 mm that tended to coalesce. The physical examination was otherwise unremarkable.

Biopsy of a papule revealed features suggesting lichenoid dermatitis (Fig. 2). The patient was diagnosed with lichenoid chronic GVHD following Blaschko lines and, because no extracutaneous involvement was present, high-potency topical corticosteroids were prescribed. The lesions resolved after 1.5 months, leaving a slight hyperpigmentation.

Given the presence of an acquired linear lichenoid eruption along Blaschko lines, we considered the following differential diagnoses: linear lichen planus, lichen striatus, inflammatory linear verrucous epidermal nevus, linear porokeratosis, and linear psoriasis. Nevertheless, given the patient’s history of allogeneic bone marrow transplantation, localized lichenoid chronic GVHD was the first option.

Chronic cutaneous GVHD can follow 2 main patterns: lichenoid eruptions and sclerodermoid manifestations. Other, less frequent manifestations include xerosis, poikiloderma, keratosis pilaris, lichenification, psoriasisform lesions, palmoplantar eczema, erythrophora, exfoliative dermatitis, and manifestations that mimic other annular dermatoses such as pityriasis rosea, centrifugal annular erythema, erythema multiforme, and subacute lupus. Most patients present generalized lesions but localized linear lesions, both lichenoid and sclerodermoid, have also been reported. Some lesions follow Blaschko lines, whereas others—with or without a history of herpes zoster in the same area—follow a metameric pattern.

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## Table 1: Characteristics of Published Cases of Linear Lichenoid Graft-vs-Host Disease.

<table>
<thead>
<tr>
<th>Case</th>
<th>Age, y</th>
<th>Underlying Disease</th>
<th>Interval Between Transplant and Clinical Presentation, mo</th>
<th>History of GVHD</th>
<th>History of Herpes Zoster</th>
<th>Site of Skin Lesions</th>
<th>Treatment Prescribed</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>16</td>
<td>Metachromatic leukodystrophy</td>
<td>6.5</td>
<td>Acute</td>
<td>Yes (same metamere), negative PCR for VZV</td>
<td>Scalp and right lateral cervical region</td>
<td>Topical corticosteroids</td>
<td>Linear lichenoid chronic GVHD</td>
</tr>
<tr>
<td>2</td>
<td>19</td>
<td>Aplastic anemia</td>
<td>17</td>
<td>No</td>
<td>Yes (same metamere and other ipsilateral metameres)</td>
<td>Trunk and left upper limb</td>
<td>Topical tacrolimus</td>
<td>Lichen striatus</td>
</tr>
<tr>
<td>3</td>
<td>23</td>
<td>Chronic myeloid leukemia</td>
<td>8</td>
<td>No</td>
<td>Yes (same metamere)</td>
<td>Right hemithorax (T5-T6)</td>
<td>NR</td>
<td>Lichenoid chronic GVHD</td>
</tr>
<tr>
<td>4</td>
<td>47</td>
<td>Chronic myeloid leukemia</td>
<td>10</td>
<td>Yes</td>
<td>Yes (same metamere)</td>
<td>Cervical region (C3-C4), right upper limb and hemithorax</td>
<td>NR</td>
<td>Lichenoid chronic GVHD</td>
</tr>
<tr>
<td>5</td>
<td>38</td>
<td>Chronic myeloid leukemia</td>
<td>33</td>
<td>Acute</td>
<td>Yes (different metamere), negative PCR for VZV</td>
<td>Left upper limb, oral reticulated lichenoid eruption</td>
<td>Topical corticosteroids</td>
<td>Linear lichenoid chronic GVHD</td>
</tr>
<tr>
<td>6</td>
<td>12</td>
<td>Acute lymphocytic leukemia</td>
<td>17</td>
<td>Acute</td>
<td>Yes (different metamere)</td>
<td>Trunk and right lower limb</td>
<td>Methylprednisolone</td>
<td>Linear lichenoid chronic GVHD</td>
</tr>
<tr>
<td>7</td>
<td>15</td>
<td>Aplastic anemia</td>
<td>18</td>
<td>No</td>
<td>Yes (different metamere)</td>
<td>Trunk and right upper limb</td>
<td>Topical corticosteroids Psoralen-UV-A therapy</td>
<td>Linear lichenoid chronic GVHD</td>
</tr>
<tr>
<td>8</td>
<td>40</td>
<td>Myelodysplastic syndrome</td>
<td>16.6</td>
<td>Acute and chronic</td>
<td>No (herpes zoster developed in a different metamere after GVHD)</td>
<td>Right lower limb</td>
<td>Topical corticosteroids</td>
<td>Linear lichenoid chronic GVHD</td>
</tr>
<tr>
<td>9</td>
<td>26</td>
<td>Acute promyelocytic leukemia</td>
<td>6</td>
<td>No</td>
<td>No</td>
<td>Trunk and right upper and lower limbs</td>
<td>Topical corticosteroids</td>
<td>Linear lichenoid chronic GVHD</td>
</tr>
<tr>
<td>10</td>
<td>50</td>
<td>Anaplastic large-cell lymphoma</td>
<td>14</td>
<td>Acute</td>
<td>No</td>
<td>Right lower limb (coinciding with the appearance of skin lesions associated with anaplastic large-cell lymphoma on the right buttock)</td>
<td>Topical corticosteroids NR</td>
<td>Superimposed linear lichenoid chronic GVHD</td>
</tr>
<tr>
<td>11</td>
<td>44</td>
<td>Acute myeloid leukemia</td>
<td>8.3</td>
<td>No</td>
<td>No</td>
<td>Right lower limb</td>
<td>Topical corticosteroids</td>
<td>Linear lichenoid chronic GVHD along Blaschko lines</td>
</tr>
<tr>
<td>12</td>
<td>16</td>
<td>Blastic plasmacytoid dendritic cell neoplasm</td>
<td>20</td>
<td>Acute</td>
<td>No</td>
<td>Trunk and left upper limb</td>
<td>Topical corticosteroids</td>
<td>Linear lichenoid chronic GVHD along Blaschko lines</td>
</tr>
</tbody>
</table>

Abbreviations: GVHD, graft-vs-host-disease; NR, not reported; PCR, polymerase chain reaction; VZV, varicella zoster virus.
CASE 6
reactivation,

patterns
underlying
distributed
(in
Figure
prednisolone
(hematoxylin-eosin,
third,
patients,
We
Several
host’s
donor
infection.
Several
immunocompetent
lesions------may
2
1,3---10
hyperplasia,
This
clinically
reactivation
somatic
(lichenoid
GVHD---whether
defeated
lesions
advanced
with
the
immunologic
characteristics
of
the
erup
tocyt
ces
become
altered,
causing
them
to
be
attacked
by
donor
lymphocytes
(isotopic
response,
in
which
a
dermatosis
occurs
at
the
site
of
a
different
healed
disease).
There
have
also
been
cases
of
sclerodermoid
GVHD
involving
lesions
that
affect
previously
irradiated
or
injured
zones.
In
such
cases,
the
pathophysiological
mechanism
would
be
the
underlying
disease
(a
Köb-
ner
isomorphic
response,
in
which
new
lesions
appear
in
previously
damaged
areas).
These
2
mechanisms
can
overlap.1,3,4,7-11

Differential
diagnosis
between
linear
lichenoid
GVHD
and
ordinary
linear
lichen
planus
is
practically
impos-
able.
In
linear
lichenoid
GVHD,
the
papules
are
less
angular,
less
well-defined,
and
can
be
associated
with
other
manifestations
of
acute
sclerodermoid
GVHD
or
poikiloderma.
Histologically,
both
processes
are
characterized
by
features
of
lichenoid
dermatitis,
although
in
GVHD
the
lymphohisti-
cytic
infiltrate
is
more
dispersed
and
follows
a
perivascular
and
periadnexal
distribution.1,5

In
our
patient’s
case---following
an
allogeneic
bone
mar-
row
transplantation---the
clinical
and
histologic
findings
support
a
diagnosis
of
lichenoid
chronic
GVHD
distributed
along
Blaschko
lines.
In
a
patient
with
complete
hematopo-
ietic
chimerism,
this
dermatosis
would
have
been
caused
by
the
immunocompetent
donor
cells’
reaction
to
the
host
and
therefore
satisfies
the
immunologic
criteria
required
for
a
diagnosis
of
GVHD.
The
fact
that
the
disease
exclusively
affected
the
skin
along
Blaschko
lines
is
explained
by
the
presence
of
a
latent
somatic
mosaicism
in
the
host,
which
was
unmasked
by
the
donor
cells.

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Dermatomal lichenoid chronic graft-vs-host disease following

Figure 1 A, Linear lichenoid eruption following the Blaschko lines on the left upper limb. B, Linear lichenoid eruption on the first,
third, fourth, and fifth fingers of the left hand. C, S-shaped linear lichenoid eruption on the left hemithorax.

Figure 2 Lichenoid dermatitis: orthokeratotic hyperkeratosis
with
a
scaly
appearance,
focal
hypergranulosis,
slight
irregular
epidermal
hyperplasia,
blurring
of
the
dermal-epidermal
junction,
basal
layer
vacuolization,
and
necrotic
keratinocytes
(hematoxylin-eosin,
original
magnification
×200).

We
found
11
reported
cases
of
lichenoid
chronic
GVHD
(Table
1).
More
than
half
of
these
patients
had
a
history
of
acute
GVHD.
A
prior
history
of
herpes
zoster
was
present
in
7
patients,
4
of
whom
developed
lichenoid
papules
in
the
same
metameres
that
had
been
affected
by
the
viral
infection.
Topical
corticosteroids
were
prescribed
to
6
patients,
topical
tacrolimus
to
1
patient,
oral
methyl-
prednisolone
to
1
patient,
and
psoralen-UV-A
therapy
to
1
patient.1,3-10

Several
hypotheses
have
been
proposed
to
explain
the
pathophysiology
of
this
unusual
pattern.
Lesions
dis-

Figure 1 A, Linear lichenoid eruption following the Blaschko lines on the left upper limb. B, Linear lichenoid eruption on the first, third, fourth, and fifth fingers of the left hand. C, S-shaped linear lichenoid eruption on the left hemithorax.
Contact Allergy to Octocrylene in Children: A Report of 2 Cases

Dos casos de alergia de contacto a octocrileno en niños

To the Editor:

The incidence of allergic and photoallergic reactions to sunscreen has increased in recent years due to the widespread use of UV filters. We report 2 pediatric cases of contact allergy to octocrylene; while known in adults, this reaction has not been previously reported in children in Spain.1,2

Patient 1

A 4-year-old girl was referred to our department as she had developed a skin rash on sun-exposed areas the previous summer. The physical examination at the time showed a skin rash consisting of erythematous micropapules on the face and limbs. While wearing sunscreen (Crema Solar Pediátrica Carrefour), the child had been exposed to the sun for 4 hours before the rash appeared. The distribution of the rash coincided with the areas where the sun cream had been applied. The patient had no history of atopic dermatitis or of the use of any medications or topical anti-inflammatory creams. We performed patch and photopatch tests with the Marti-Tor UV filter series and with the sunscreen product used by the patient. Positive results (++) were seen at 96 hours for the sun cream (Crema Solar Pediátrica Carrefour) and for octocrylene 10% in petrolatum in the photopatch tests. The diagnosis was photoallergic dermatitis to octocrylene.

Patient 2

A 5-year-old girl was referred to our department following 2 episodes of acute eczema in sun-exposed areas that had been protected with 2 different sunscreens (Isdin Extrem Pediatrics 50+ and Anthelios Dermopédiatriques). The patient had no history of atopic dermatitis or of the use of medications or topical anti-inflammatory creams. Patch and photopatch tests were performed using the Marti-Tor UV filter series and the sun creams the patient had used. Positive results (++) were seen at 48 and 96 hours for octocrylene 10% in petrolatum and for 1 of the sunscreens, Isdin Extrem Pediatrics 50+, which contained octocrylene 9%. The diagnosis was allergic contact dermatitis to octocrylene.

Greater awareness of the harmful effects of the sun and public health messages have led to a progressive increase in the use of sunscreens. UV filters are now found not only in sunscreens but also in a wide range of skincare and cosmetic products.3,4 At the same time, however, there has also been an increase in the incidence of sensitization and photosensitization to these filters. UV filters have traditionally been classified as physical or chemical, and chemical filters are more frequently associated with skin allergy.

Octocrylene is an organic compound belonging to the cinnamate family. It is a relatively new filter, capable of absorbing both UV-B and UV-A rays. When used in isolation, its sun protection abilities are poor and it is therefore generally combined with other UV filters to offer a higher sun protection factor and a more stable product that is easier to apply and more water-resistant.4

Octocrylene has considerable allergenic potential and can induce serious contact eczema, even through passive transfer.5 In some series, it has been found to be the main

References


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