Neonatal and Infantile Erythrodermas
A Retrospective Study of 51 Patients
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Objective: To determine the frequency of the various underlying causes of erythroderma in newborns or infants, as well as which clinical or laboratory findings were relevant for the etiological diagnosis.

Patients: Fifty-one patients who presented with exfoliative erythroderma during their first year of life were included in this retrospective study.

Setting: Department of Pediatric Dermatology at a university hospital.

Results: On average, the etiological diagnosis was established 11 months after the onset of erythroderma. The underlying causes observed included immunodeficiency (30%), simple or complex ichthyosis (24%), Netherton syndrome (18%), and eczematous or papulosquamous dermatitis (20%). Five patients (10%) had erythroderma of unknown origin. The following parameters were of value in determining the underlying cause of erythroderma: congenital onset, skin induration and the presence of large scaling plaques, alopecia with or without hair dysplasia, evolution, response to topical corticosteroid therapy, presence of infections, and failure to thrive. Histological analysis confirmed the diagnosis in only 19 (45%) of 42 cases. However, it proved of great value for the detection of significant lymphocyte infiltration or keratinocyte necrosis indicating a diagnosis of Omenn syndrome or immunodeficiency. The prognosis was poor in this series: the mortality rate was 16%, and severe dermatosis persisted in 29 (67%) of the survivors.

Conclusions: The etiological diagnosis of neonatal erythroderma is difficult to make; some clinical features may be helpful, but no one feature is characteristic of a cause. An immunodeficiency must be suspected in cases of severe erythroderma with skin induration, severe alopecia, failure to thrive, infectious complications, or evocative histological findings. The prognosis is poor, with a high rate of mortality in immunodeficiency disorders and severe chronic disease in Netherton syndrome and psoriasis.

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N E O N A T A L and infantile erythroderma is rare, but its frequency is unknown. Numerous underlying causes have been reported in the literature. The etiological diagnosis of erythroderma is frequently difficult to establish, and is usually delayed, owing to the poor specificity of clinical and histological signs. The problems involved in the diagnosis and treatment of erythroderma are specific to the neonatal period.

We carried out a retrospective study of 51 pediatric cases of congenital erythroderma, or erythroderma presenting during the first year of life. We did not include types of erythrodermas involving blister formation.

The aim of the study was to determine the frequency of the various underlying causes and to analyze retrospectively the clinical and laboratory data of these patients. Identification of the underlying disease is necessary to provide optimum care.

RESULTS

UNDERLYING CAUSES AND CLINICAL DATA

In all cases, there was a significant delay in diagnosis, which was made an average 11 months after the onset of erythroderma. The underlying causes are listed in Table 1. Erythroderma began at an average age of 7 weeks. Congenital erythroderma was observed in 16 cases (32%); they were all ichthyosis, except for 1 case, which was an Omenn syndrome. Six cases of ichthyosis were preceded by a colloidion baby syndrome and were grouped with the cases of congenital erythroderma. In the other cases, there was a dis-
PATIENTS AND METHODS

Fifty-one patients (27 girls and 24 boys) who were hospitalized at the Hôpital Necker–Enfants Malades, Paris, France, and who presented with exfoliative erythroderma during their first year of life were included in this retrospective study. The mean follow-up period was 66 months. Clinical data were recorded and analyzed according to underlying cause.

A skin biopsy specimen was obtained from 42 patients during the erythrodermic stage. All hematotoxin-eosin–stained sections were examined by the same 3 investigators (A.P., C.B., and S.F.) to determine the relevance of the histological findings in relation to the final etiological diagnosis and its reproducibility. Thirteen biopsy specimens were processed for immunophenotyping.

Routine biochemical tests were performed at admission, including measurement of electrolyte and serum IgG levels and an eosinophil count. Immunological tests, which included determination of immunoglobulin levels and lymphocyte typing and proliferation studies, were performed in cases of suspected immunodeficiency and in infants with persistent erythroderma associated with skin infiltration, infectious complications, or severe failure to thrive. Additional specialized tests, including fatty acid and amino acid chromatography and enzyme assays of biotin, zinc, and steroid sulfatase and of the fatty alcohol oxidoreductase in cultivated fibroblasts, were performed in infants who presented with ichthyosiform erythroderma with alopecia and neurological signs.

The treatment of erythroderma was combined with nutritional therapy involving the correction of energy intake and maintenance of fluid and electrolyte balance. Infectious complications were avoided by the use of topical antiseptics, and samples were obtained regularly for microbiological analysis. Systemic antibiotic therapy was prescribed in cases of septicaemia or deep visceral infection. Emollients were used in all cases, and topical corticosteroid therapy was administered to all patients with pruritic erythroderma. Other treatments, including antifungals, retinoids, cyclosporin, bone marrow transplants, and systemic corticosteroid therapy or chemotherapy, were used in some cases.

ease-free interval after birth. The cases of erythroderma that were not congenital occurred relatively early, before the age of 4 months and at an average age of 9 weeks (Table 1).

Parental inbreeding was observed in 17 cases (34%) of erythroderma, including 3 (25%) of 12 cases of ichthyosis, 8 (88%) of 9 cases of Netherton syndrome, and 5 (33%) of 15 cases of primary immunodeficiency. Sibling mortality and a family history of erythroderma occurred in 4 (57%) of 7 cases of Omenn syndrome and in 6 (66%) of 9 cases of Netherton syndrome. A family history of atopy occurred in 15 cases (30%); it was associated with atopic dermatitis in 4 (66%) of 6 cases (66%), with Netherton syndrome in 3 (33%) of 9 cases, with seborrhic dermatitis in 1 (50%) of 2 cases, with Omenn syndrome in 3 (43%) of 7 cases, with the 1 case of secretory IgA deficiency, and with 3 of the 5 cases with no diagnosis.

All patients had generalized erythema with desquamation; 20 patients (40%) had no other specific cutaneous signs. Pruritus was observed in 31 cases (62%): it was severe and constant in all cases of atopic dermatitis and immunodeficiency, and it was moderate to severe in 8 of 9 of Netherton syndrome. Clinical signs, such as skin infiltration, alopecia, and complications, are listed in Table 2 and Table 3. A skin induration was observed in 10 of the 12 cases of immunodeficiency, in 2 of the 6 cases of atopic dermatitis, and in 2 of the 4 cases of neuroichthyosis.

In 5 cases, no definitive diagnosis was established despite a long period of follow-up (range, 1-13 years) and repeated testing. These 5 patients presented with severe pruritic erythroderma with marked infiltration of the skin, as well as hair and nail abnormalities. Erythroderma began at a mean age of 3½ months and was associated with a dramatic failure to thrive (SD, −4 to −5), with widespread lymphadenopathy and hepatosplenomegaly in 3 cases and severe systemic infectious complications in all cases.

LABORATORY DATA

Apart from nonspecific inflammatory markers and a persistently high leukocyte count, the main laboratory abnormalities were secondary electrolyte disorders, with severe hypernatremia (sodium, >160 mmol/L) without complications in 9 cases, or with major hypoalbuminemia (<30 g/L) in 3 cases (Table 3). The eosinophil count was high (>0.5 × 10^9/L) in 28 cases (55%), and the IgE level was elevated in 22 cases (59%) (Table 3). Laboratory tests of cellular and humoral immune functions were carried out in 35 patients (68%), demonstrating abnormalities in 18 cases. An immunodeficiency disease was
identified in 15 cases, including 7 cases of Omenn syndrome, 1 case of severe combined immunodeficiency, 5 cases of graft-vs-host disease, 1 case of IgA deficiency, and 1 case of Wiskott-Aldrich syndrome (Table 1). In 3 cases of atopic dermatitis, moderate abnormalities were noted: 1 demonstrated transient lymphopenia with a reversed CD4/CD8 ratio, and the other 2 showed low levels of IgA and IgG, respectively. After several immune function tests, the results of which were considered normal, 1 of the 5 patients with no diagnosis developed severe CD3 lymphopenia (CD3, 10%) and 1 developed CD8 lymphopenia (CD8, 4%). Reduction of the activity of the fatty oxidoreductase enzyme was found in 2 patients, both of whom had a typical evolution of Sjögren-Larsson syndrome with spastic paraplegia. No other metabolic abnormalities were noted.

HISTOLOGICAL ANALYSIS

In more than half (n=28 [55%]) of the cases, the histological findings were not specific, and showed psoriasiform epidermal hyperplasia, with parakeratosis, dilatation of the capillaries of the superficial dermis, and various perivascular inflammatory infiltrates. These findings were also demonstrated in patients with Netherton syndrome, congenital ichthyosiform erythroderma, atopic dermatitis, psoriasis, and seborrheic dermatitis, and in 2 of the 5 patients with no diagnosis.

Table 2. Clinical Features in 51 Cases of Erythroderma

<table>
<thead>
<tr>
<th>Underlying Cause</th>
<th>Skin Induration</th>
<th>Severe Alopecia</th>
<th>Alopecia of Eyelashes and Eyebrows</th>
<th>Response to Topical Corticosteroids</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. of Patients</td>
<td>2/6</td>
<td>3/6</td>
<td>6/6</td>
</tr>
<tr>
<td>Atopic dermatitis</td>
<td></td>
<td></td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>Seborrheic dermatitis</td>
<td></td>
<td></td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>Psoriasis</td>
<td></td>
<td></td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>Ichthyosis</td>
<td></td>
<td></td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>Netherton syndrome</td>
<td></td>
<td></td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>NeuroIchthyosis</td>
<td></td>
<td></td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>Omenn syndrome</td>
<td></td>
<td></td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>SCID</td>
<td></td>
<td></td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>Maternal-fetal GVHD</td>
<td></td>
<td></td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>Other types of GVHD</td>
<td></td>
<td></td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>Wiskott-Aldrich syndrome</td>
<td></td>
<td></td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>Secretory IgA deficiency</td>
<td></td>
<td></td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>Unclassifiable</td>
<td></td>
<td></td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>34</td>
<td>46</td>
<td>24</td>
</tr>
</tbody>
</table>

*SCID indicates severe combined immunodeficiency; GVHD, graft-vs-host disease; and NS, not specified.
†With trichorrhesis invaginata by microscopic examination under polarized light.

Table 3. Complications and Laboratory Test Abnormalities in 51 Cases of Erythroderma*

<table>
<thead>
<tr>
<th>Underlying Cause</th>
<th>Severe Dehydration</th>
<th>Eosinophil Count &gt;0.5 × 10^6/L</th>
<th>High IgE (Range, IU/L)</th>
<th>Systemic Infections (Type of Infection)</th>
<th>Failure to Thrive</th>
<th>Chronic Diarrhea</th>
<th>Death</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atopic dermatitis</td>
<td>0/6</td>
<td>4/6 (1.6-25.0)</td>
<td>3/6 (1130-1530)</td>
<td>2/6</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Seborrheic dermatitis</td>
<td>0/5</td>
<td>2/2 (0.7-1.0)</td>
<td>1/2 (250)</td>
<td>1/2</td>
<td>2/2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Psoriasis</td>
<td>1/8</td>
<td>0/8</td>
<td>NS</td>
<td>1/8</td>
<td>3/8</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Ichthyosis</td>
<td>3/9</td>
<td>8/9 (0.5-4.0)</td>
<td>7/9 (66-10 400)</td>
<td>5/9</td>
<td>7/9†</td>
<td>1/9</td>
<td>0</td>
</tr>
<tr>
<td>Netherton syndrome</td>
<td>0/4</td>
<td>1/4</td>
<td>0/4</td>
<td>1/4</td>
<td>4/4†</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>NeuroIchthyosis</td>
<td>4/7</td>
<td>6/7 (2.3-16.5)</td>
<td>5/7 (860-38 300</td>
<td>6/7 (2 PCT)</td>
<td>7/7†</td>
<td>3/7</td>
<td>2/7</td>
</tr>
<tr>
<td>Omenn syndrome</td>
<td>0/1</td>
<td>0/1</td>
<td>NS</td>
<td>1/1 (CMV)</td>
<td>1/1</td>
<td>1/1</td>
<td>1/1</td>
</tr>
<tr>
<td>SCID</td>
<td>0/1</td>
<td>0/1</td>
<td>NS</td>
<td>3/4 (1 PCT, 2 CMV)</td>
<td>3/4</td>
<td>2/4</td>
<td>3/4</td>
</tr>
<tr>
<td>Maternal-fetal GVHD</td>
<td>0/1</td>
<td>1/1 (6.9)</td>
<td>NS</td>
<td>1/1 (CMV)</td>
<td>1/1</td>
<td>1/1</td>
<td>1/1</td>
</tr>
<tr>
<td>Other types of GVHD</td>
<td>0/4</td>
<td>2/4 (6.9-9.8)</td>
<td>NS</td>
<td>3/4 (1 PCT, 2 CMV)</td>
<td>3/4</td>
<td>2/4</td>
<td>3/4</td>
</tr>
<tr>
<td>Wiskott-Aldrich syndrome</td>
<td>0/1</td>
<td>1/1 (2.5)</td>
<td>1/1 (6000)</td>
<td>0/1</td>
<td>1/1</td>
<td>1/1</td>
<td>0</td>
</tr>
<tr>
<td>Secretory IgA deficiency</td>
<td>0/1</td>
<td>1/1 (16.0)</td>
<td>1/1 (5000)</td>
<td>1/1</td>
<td>1/1†</td>
<td>1/1</td>
<td>0</td>
</tr>
<tr>
<td>Unclassifiable</td>
<td>1/5</td>
<td>1/5 (3.5)</td>
<td>4/5 (145-25 680)</td>
<td>5/5</td>
<td>5/5†</td>
<td>0</td>
<td>2/5</td>
</tr>
<tr>
<td>Total</td>
<td>18</td>
<td>15</td>
<td>24</td>
<td>30</td>
<td>16</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*SCID indicates severe combined immunodeficiency; GVHD, graft-vs-host disease; NS, not specified; PCT, Pneumocystis carinii pneumonia; and CMV, cytomegalovirus.
†More than −3 SDs.
Histological examinations that led to the diagnosis of ichthyosis in 6 of 12 cases (except Netherton syndrome) demonstrated significant orthokeratosis with corneocytes arranged in a laminated and compact fashion, striking hypergranulosis, and epidermal hyperplasia, with no or few infiltrates of inflammatory cells. Significant dermal and epidermal lymphocytic infiltration and keratinocyte necrosis with satellite lymphocytes (Figure 1) were noted in 6 (80%) of the 8 skin biopsy specimens from patients with Omenn syndrome and graft-vs-host disease. Eosinophilic infiltrates were less frequent. Three of the 5 cases without diagnosis demonstrated a histological pattern similar to that of Omenn syndrome, with marked lymphocytic infiltration and keratinocytic necrosis.

EVOLUTION AND TREATMENT

The mortality rate in this study was 16% (Table 3). Eight patients died of complications resulting from either the erythroderma or their underlying disease at a mean age of 7 months. After a follow-up of 5.5 years, the skin prognosis was poor in 37 patients (73%): 10 (20%) had persisting erythroderma despite various treatments (2 with psoriasis, 5 with Netherton syndrome, 1 with Omenn syndrome, and 2 unclassifiable cases); 19 (37%) had a chronic condition that persisted for a mean of 7 months. After a follow-up of 5.5 years, the skin prognosis was poor in 37 patients (73%): 10 (20%) had persisting erythroderma despite various treatments (2 with psoriasis, 5 with Netherton syndrome, 1 with Omenn syndrome, and 2 unclassifiable cases); 19 (37%) had a chronic condition that persisted for a mean of 7 months.

COMMENT

Although numerous studies of adult erythroderma have been published in the literature, few have been published regarding infant erythroderma. Since the data reported in the literature are frequently found in isolated case reports, the frequency of erythroderma in newborns is unknown. Underlying causes are distinctive in this age group, and, unlike those found in adults, lymphoma and toxic causes have not been reported (to our knowledge). The most frequent underlying causes in our study were immunodeficiency (30%) and Netherton syndrome (18%). Atopic dermatitis and seborrheic dermatitis were less frequent, with 6 (12%) and 2 (4%) cases, respectively. The high frequency of immunodeficiency is probably attributable to the recruitment of this patient population to the specialized Pediatric Department at Necker Hospital.

In 5 cases, no firm diagnosis had been made; however, immunodeficiency was strongly suspected, because these cases of erythroderma were severe, were only moderately improved by topical corticosteroids, and were associated with skin infiltration, failure to thrive, and serious infections. Three of them had histological features suggestive of an immunodeficiency. These findings may result from immune system abnormalities that have not yet been characterized. Such cases have been described in the literature, and some of them were former classified as Leiner disease.18-22 This term is no longer appropriate because it probably refers to a heterogeneous group of disorders.

The causes of erythroderma in infants are often difficult to determine, which explains the long delay (average, 11 months) in diagnosis that occurred in our study. Analysis of the patient's history and presenting clinical signs, combined with the results of laboratory tests, is helpful in reaching a diagnosis.

The underlying causes could be classified according to whether the onset was congenital (32%) or non-congenital. Congenital onset is indicative of ichthyosis or immunodeficiency. However, the concept of the disease-free interval used in the literature to classify different types of erythroderma does not allow a formal distinction to be made between these diagnoses (Table 1).

The diagnosis of ichthyosis is easy in cases that present with thick scales and massive and compact orthokeratosis. Difficulties arise in cases that are associated with other symptoms, suggesting a diagnosis of complex ichthyosis. In 4 cases, ichthyosis was associated with neurological or ocular symptoms, which were related to KID (keratitis, ichthyosis, and deafness) syndrome in 1 case and Szögren-Larsson syndrome, which was confirmed by the low level of the enzyme fatty alcohol oxidoreductase, in 2 cases. The diagnosis of complex ichthyosis such as is seen in Netherton syndrome is difficult, with a delay in diagnosis of 11 months in some cases (Figure 2). In 5 cases (59%), neither erythroderma nor a keratinization disorder was present at birth. The characteristic hair dysplasia, ie, trichorhexis invaginata, revealed by systematic routine light microscopy of hair, as well as by examination of eyebrows and eyelashes, was noted only after an average age of 10 months, except in 2 cases, in which the diagnosis was made during the first month. Thus, repeated light microscopic examinations of the hair are necessary to confirm a suspected diagnosis of Netherton syndrome.

Erythrodermic psoriasis and congenital ichthyosiform erythroderma are often indistinguishable during the first months of life. Evolution with a typical psoriasis re-
lapse after 9 months in 1 case and identification of specific histological features at 20 months of age in another case confirmed these clinical diagnoses.

In early (first weeks of life) or congenital erythroderma, an immunodeficiency, such as Omenn syndrome, was strongly suspected in cases in which the presenting features included pruritic erythroderma combined with skin induration, resulting in exaggerated skin folds; nonlysplastic alopecia of hair, eyelashes, and eyebrows; widespread lymphadenopathy; and association with profound and deep systemic infection caused by Candida organisms or rare or opportunistic microorganisms. The skin infiltration was strongly indicative of Omenn syndrome or immunodeficiency, although true atopic dermatitis with marked lichenification can have a similar appearance (Figure 3).

In fact, erythroderma may be the first clinical manifestation of an immunodeficiency syndrome. Each dermatological characteristic is not specific and can be observed with other disorders. An association with diarrhea and profound failure to thrive, which are not observed in atopic dermatitis or seborrheic dermatitis, should also suggest the diagnosis. Histological findings in these cases are helpful. Keratinocytic necrosis, satellite cell lymphocytes, and significant lymphocytic or eosinophilic infiltration are highly suggestive of an immunodeficiency syndrome.

Alopecia was observed in 23 cases (46%) and may be a complication of any severe type of erythroderma. Association with alopecia of the eyebrows and eyelashes was strongly indicative of a more complex disorder, eg, immunodeficiency. Such alopecia was present in 4 of 8 cases of Omenn syndrome or severe combined immunodeficiency and in 3 of 5 cases of suspected immunodeficiency, but not in cases of atopic dermatitis. Erythroderma with alopecia may also be observed in association with metabolic disorders, such as biotin deficiency and citrullinemia.23-25

The histological findings agreed with the final diagnosis in only 45% of cases, a percentage that is similar to those reported in other studies of adult erythroderma.26,27 Histological examination can provide the diagnosis of graft-vs-host disease or Omenn syndrome in cases of significant lymphocytic infiltration and keratinocytic necrosis with satellite lymphocytes.

The total IgE level and eosinophil counts, usually considered to be markers of atopy, were of no predictive value in the diagnosis of atopic dermatitis because they were neither constant nor specific. There was no relationship between the levels of IgE or the eosinophil count and the clinical severity or the etiological diagnosis of the erythroderma (Table 3).

The main complications in our series were hypernatremic dehydration, infections, and failure to thrive, which were attributable to the severity of skin disease and the underlying disease (Table 3). As previously reported in the literature, we observed severe hypernatremia in one third of the patients with Netherton syndrome; however, in our series, severe hypernatremia was also frequent in the patients with Omenn syndrome (n=4/7) (Table 3). Cutaneous infections caused by Staphylococcus, Streptococcus, or, more rarely, Gram-negative bacilli were constant and resulted in septicemic infection in 27 (53%) of the 51 patients. Therefore, repeated bacteriological investigations and prophylactic topical antiseptic treatment were justified. Only immunodeficient patients had deep visceral or opportunistic infections and cutaneous fungal infections (Candida).

Failure to thrive affected 35 (69%) of the 51 patients with erythroderma in our study. It was profound (more than −3 SDs) in Netherton syndrome, similar to previous reports in the literature,28 as well as in all cases of primary immunodeficiency and all unclassifiable cases. It was moderate (less than −2 SDs) in simple ichthyosis and absent in atopic dermatitis, contrary to reports in the literature.29 The loss of weight observed with erythroderma appears to be multifactorial.

Erythroderma is a potentially life-threatening condition in infants, with risk of septicemic infections, hypernatremic dehydration, hypoalbuminemia, and hyperpyrexia. The management of erythroderma, regardless of the underlying disease, includes the correction of caloric and protein intake, proper balance of oral and parenteral fluids and electrolytes, and prevention and treatment of infections.

Given the high prevalence of immunodeficiency in our study, immune investigations (such as tests that measure immunoglobulin levels and lymphocyte counts) should be considered in cases of neonatal erythroderma, particularly when 1 or more of the following fea-

Figure 2. Exfoliative erythroderma in a neonate with Netherton syndrome. In the first weeks of life, the erythroderma is not specific. Often, the typical circumflex linear ichthyosis appears several weeks later.

Figure 3. Generalized scaling erythroderma with infiltrated thick folds and alopecia of the eyelashes in a neonate with Omenn syndrome.
tures are present: skin infiltration; severe alopecia without dysplasia of hair or eyelashes; severity and persistence of the condition; resistance to standard topical treatment; histological findings that show marked inflammatory infiltration and keratinocytic necrosis; severe failure to thrive; chronic diarrhea; and severe infectious complications.

Although the treatment of erythroderma is often limited to local care with topical corticosteroids or emollients, whose efficacy varies depending on the underlying cause (eg, frequent corticosteroid resistance is seen in immunodeficiency), certain cases of immunodeficiency may require bone marrow transplantation. This procedure is certainly a major intervention, but in the present series, it did lead to recovery in 3 cases of Omenn syndrome and 1 case of severe combined immunodeficiency.

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REFERENCES