A Practical Approach to the Diagnosis and Treatment of Vitiligo in Children

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Vitiligo is a chronic, acquired depigmenting condition of the skin, and sometimes of the mucosa, that results in the selective loss of melanocytes.1 Vitiligo has been reported since ancient times and a description of it exists in the Latin medical classic De Medicina of Celsus during the second century.2, 3 The name might come from the Latin “vitium,” meaning defect or blemish.4 Although vitiligo is not a rare disease, it has been until recently an orphan disease in terms of drug development and a neglected one in the field of dermatology. Moreover, patients often complain that physicians are unaware of treatment options and that most of these physicians consider vitiligo as a “cosmetic disease.”5 Despite the lack of demonstrative physical symptoms accompanying the disease as compared with other common chronic dermatological disorders, such as eczema and psoriasis, vitiligo causes emotional distress and impairs patient and parental quality of life in childhood.

EPIDEMIOLOGY OF PEDIATRIC VITILIGO

The prevalence of vitiligo worldwide ranges from 0% to 2.16% of the population,6 with approximately one-third to one-half of all cases having onset in childhood.7 There appear to be 2 subsets of patients with vitiligo: those with early onset (12 years of age or younger) who have more halo nevi, Koebner phenomenon (KP) (lesional development in response to trauma), family history, segmental disease and atopy, and those with late onset who have more acrofacial lesions and thyroid disease (in those older than 12 years).7–9 High estimates of prevalence in pediatric vitiligo are noted when analyzing groups of pediatric patients attending a dermatology clinic. For instance, in Nepal and India, 2.0% and 2.6%, respectively, of children attending a dermatology clinic were diagnosed with vitiligo.10 In general population-based studies, vitiligo occurs in fewer than a half percent of the population of children. In a large Chinese population-based study in

which the prevalence overall was 0.56% of the population, a prevalence of 0.1% in the 0 to 9 years age group and 0.36% by 10 to 19 years of age was recorded.11 Similarly a Taiwanese study showed 0.09% of children had vitiligo12 and a Danish population-based study showed 0.09% and 0.15% prevalence for 0 to 9 and 10 to 20 years age groups, respectively.13 In the Sinai desert, a cohort of children younger than 18 years showed a prevalence of 0.18%.14

Disease onset increases through the first 2 decades of life. Disease is quite uncommon in children younger than 2 years as opposed to congenital disorders of pigmentation, such as nevus depigmentosus.15 Onset before age 2 years represents 11% of pediatric-onset cases, 28% of cases start between 2 and 5 years, 40% of cases begin between 5 and 10 years, and 21% between 10 and 18 years, demonstrating that median age of onset is between 5 and 10 years of age.16 In a cohort of Indian children, 56.7% of pediatric cases were noted in childhood between 8 and 12 years.17 Early-onset disease (before age 12 years) represented 35.2% of cases in an Indian cohort.17 Children younger than 20 years represent 35.5% of patients in a Nigerian cohort18 and age of onset before 20 years of age was noted in 47.8% of patients in a Gujarati cohort.19 Mean age of onset can be as low as 6.9 years in Indian children 12 years and younger17 and as high as 17.4 years in an unselected Saudi Arabian mixed pediatric and adult population.20 Family history appears to be associated with earlier age of onset.21

The prevalence of vitiligo by gender is usually close to if not equal, with some studies supporting female predilection in the youngest age groups. Vora et al19 described a Gujarati cohort of 1100 patients of all ages in which 57.3% were female individuals and 42.7% were male individuals. In a cohort of 268 Indian children 12 years and younger, 56.7% were girls (n = 152) and 43.3% were boys (n = 116).17 Similarly, in a Greek population, two-thirds of the children ages 0 to 10 years were girls, whereas by late adulthood, incidence was similar between the genders.22

The breakdown of vitiligo types in a pediatric population of patients with vitiligo varies by population reviewed. In a case series of 119 pediatric patients with vitiligo, 34% had generalized disease, 13% acrofacial, 3% mucosal, 29% segmental, and 21% undetermined. Lower estimates of segmental disease include 17.6% of cases in children who were 12 years or younger in an Indian cohort.17

**Clinical Features**

Recently, an international panel of experts has proposed consensus definitions of vitiligo.23 Overall, vitiligo can be divided into segmental vitiligo (SV) and vitiligo/nonsegmental vitiligo (NSV), which encompasses rare forms of vitiligo. Typical vitiligo lesions can be defined as whitish, nonscaly macules that have usually distinct margins. When first seeing a patient, it is of prime importance to differentiate between these 2 forms, as these are quite different in terms of prognosis, evolution, and response to treatment patterns.

**Nonsegmental Vitiligo**

NSV is the most common variant of vitiligo, and accounts for almost 80% of all cases. NSV is characterized by asymptomatic, well-circumscribed, milky-white macules involving multiple parts of the body, usually in a symmetrical pattern (Fig 1). The disease can start at any site of the body, but the fingers, hands, and face are frequently the initial sites. Within NSV, several subphenotypes have been well described, including acrofacial, mucosal, generalized, universal, mixed, and rare forms. Of note, overlaps between these forms may exist; for example, NSV may initially have an acrofacial pattern, with a later evolvement to the generalized form. Interestingly, a recent study based on latent class analysis has distinguished 2 types of vitiligo with probable different pathophysiological pathways.8
Acrofacial vitiligo is not very common in children. In this form, areas involved are often limited to the face, hands, feet, and orifices. The form may later evolve to typical generalized vitiligo.

Vitiligo universalis is a widespread form of the disease that is generally seen in adults, although cases in children are reported. The term “universalis” refers to the almost virtually universal depigmentation (>60% to 90% of the body surface area) (Fig 2). Hairs may be partially spared. In general, vitiligo universalis is the result of generalized vitiligo that gradually progresses to nearly complete depigmentation of the skin.

Mucosal vitiligo states for oral and/or genital mucosae involvement in vitiligo as part of generalized vitiligo or as an isolated condition. When limited to mucosa, differential diagnosis should include lichen sclerosus. Moreover, the coexistence of both conditions has also been reported.

Mixed vitiligo refers to the concomitance of SV and NSV in a single patient. Criteria proposed for mixed vitiligo are detailed elsewhere.

Rare Forms

Several forms may fit into the spectrum of rare vitiligo and all should be considered as NSV forms. Punctate vitiligo was first described by Falabella et al and refers to pea-sized depigmented macules that may involve any area of the body.

Vitiligo minor is rarely reported in children. In this rare form, hypopigmented macules are distributed mainly on the face, and the back has been recently reported. Another striking rare form is follicular vitiligo, which has the particularity to primarily involve the melanocyte follicular reservoir with whitening of most of the body hairs and rare depigmented macules.

Segmental Vitiligo

SV accounts for 10% to 15% of all types of vitiligo. SV is defined as a unilateral and segmental or band-shaped distribution (asymmetric vitiligo) (Fig 3). Generally, 1 unique segment is involved in SV, but 2 or more segments with ipsi- or contralateral distribution have been described. In this type of vitiligo, the early involvement of the follicular melanocyte reservoir is common and the disease rapidly stabilizes over a few months. Epidemiologic data also show an earlier age of onset. Finally, SV should be differentiated from focal vitiligo in which a unique small lesion without a clear segmental distribution pattern is described.
SV occurs focally, based on localized susceptibility to disease and is not associated with autoimmune phenomena unless nonsegmental disease or a generalized autoimmune condition such as alopecia areata occurs concurrently.31, 32 The leading theory is that generalized vitiligo is a multifactorial, polygenic autoimmune disorder that occurs in only a minority of genetically susceptible individuals and is therefore believed to have a strong component of environmental triggering. Other theories of vitiligo development include biochemical defects in the tetrahydrobiopterin pathway/oxidative damage, adhesion defects, and neural induction;

### Pathophysiology and Genetic Determinants

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In a survey of 2624 primarily White probands, frequency was 6.1% in siblings, and concordance only 23% in identical twins. Early-onset disease was associated with more family members, suggesting that this subgroup carries more genetic susceptibility determinants.35 Associated autoimmune diseases in family members in this cohort were vitiligo itself, autoimmune thyroid disease (particularly hypothyroidism), pernicious anemia, Addison disease, systemic lupus erythematosus, and inflammatory bowel disease. Other studies have demonstrated personal and familial association of vitiligo with canity, 21 atopic dermatitis, 36, 37 rheumatoid arthritis, types 1 and 2 diabetes mellitus, alopecia areata, psoriasis, chronic urticaria, lichen sclerosus et atrophicus, Celiac disease, systemic lupus erythematosus, and sarcoidosis.38 Extensive disease and increasing years with disease are the factors most associated with vitiligo-associated autoimmune disease.

At this time, there is no genetic test for vitiligo. The genetics of vitiligo are complex, as there are multiple determinants found in most genome-wide association studies. Consistent association with genes such as DDR1, XBPI, NLRP1, PTPN22 and COMT has been noted. Other studies have noted association with ACE, AIRE, CD4, COX2, ESR1, EDN1, FAS, FOXD3, FOXP3, IL1-RN, IL-10, MBL2, MC1R, MYG1, Nrj2, PDGFRA, PRO2268, SDF, SCGF, TXNDC5, UVRAG, and VDR genes.39 These genes can be broken down into 6 categories of genes: (1) pigmentation gene polymorphisms believed to create increased risk of autoimmune attack and susceptibility to damage: TYR, TRP 1 and 2, OCA2 and its transcription down regulator HERC2, MC1R, and DDR1, which affects melanocyte cellular adhesion; (2) MHC loci (eg, HLA-A*02:01, HLA-DR4, and HLA-DR7 alleles) and XBPI, which regulates MHC expression; (3) B- and T-cell developmental genes, which promote activity and/or repression promoting immune response against melanocytes (eg, CTLA4, BACH2, CD44, IKZF4, LNK); (4) genes involved in innate immunity (eg, NLRP-1, formerly NALP-1); (5) apoptosis determinants (CASP7); and (6) polymorphisms in genes that regulate antiinflammatory activity (eg, glutathione S transferase, vitamin D receptor).

### Diagnosis and Differential Diagnosis

The diagnosis of vitiligo is generally made clinically through the appearance of reduced or lost pigmentation of the skin in a typical distribution, including periorificial, segmental, lips and tips of the fingers, toes and/or penis, flexural surfaces, and frictional areas, such as waistbands.

Clues to the presence of generalized vitiligo include multiple halo nevi, poliosis (loss of pigmentation in the hairs), canity, family history of vitiligo, and lesions in sites of trauma, so-called KP.42, 43 SV is often corroborated by the presence of linear lesions, broadly in the Lines of Blaschko or on the face in typical segments.44 Colocalization of poliosis is common, whereas alopecia areata overlap may be noted as well. In vitiligo, biopsy will demonstrate in the center of a lesion the loss of melanocytes (pigment cells) with...
special stains of the epidermis. The border of a lesion may be noted visually to be inflamed (inflammatory vitiligo), and a notable inflammatory infiltrate of CD 4+ and CD 8+ T lymphocytes may be seen on biopsy of an “active” border, although the inflammation may be clinically absent.

The differential diagnosis of vitiligo is broad and includes inflammatory, postinflammatory, neoplastic, and primary pigmentary genetic disorders (Table 1).

The first step is to determine whether the lesion is inherited or not. Indeed, if the lesion is present at birth, inherited or genetically induced hypomelanoses should be ruled out. This is quite easy in patients with dark phototype. However, in patients with fair skin complexion, hypopigmented patches are usually revealed after the first sun exposure due to tanning of normal surrounding skin, sometime in the first 2 years of life. In this case, family history, ethnic background/consanguinity history, and a detailed family tree are of prime importance. Several genetic diseases may be misdiagnosed as vitiligo, but the most frequent are piebaldism and tuberous sclerosis. In piebaldism, the combination of white forelock, anterior body midline depigmentation, and bilateral shin depigmentation is the hallmark of the disease. Differential diagnosis with tuberous sclerosis might be trickier in case of ash-leaf hypopigmented spots (Fig 4A) without seizures or other usually later cutaneous symptoms, such as shagreen patches, or angiofibromas.

In noninherited lesions, which are the most common differential diagnoses in children with vitiligo, pityriasis versicolor (Fig 4B) and postinflammatory hypomelanoses (hypopigmentation) should be ruled out. A Wood lamp can be used to highlight lesions of vitiligo for confirmation distinguishing vitiligo from pityriasis alba (Fig 4C); on the other hand, one of the neoplastic hypomelanoses to exclude is mycosis fungoides (Fig 4D), and that may highlight on Wood lamp examination.45,46 Therefore, biopsy may be required in atypical cases and where mycosis fungoides is suspected; in that setting, T cell gene rearrangement studies on the biopsy may be helpful.

For segmental vitiligo, naevus depigmentosus (Fig 4E) is the most common lesion in the differential diagnosis. Naevus depigmentosus is usually congenital and stable in size, growing in proportion to the child’s growth. The lesion generally holds a normal or subnormal number of melanocytes with a reduced production of melanin pigment. Where biopsy is needed, the inclusion of a specimen of normal skin for comparison may be needed for definitive reading.

The psychological impact of vitiligo is profound in childhood. Associated negative experiences can include fear of being questioned about one’s appearance, teasing and bullying, anxiety over the potential for disease spread, interference with emotional maturation, depression, and interference with socialization (eg, sexual debut). Of the 25% of children with vitiligo evaluated for psychological issues, 60% reported psychological problems in a cohort of 119 Brazilian children.16 A recent review article of quality-of-life issues in childhood skin disease stated: “In general, patients with vitiligo experience low self-esteem, social stigmatization, shame, avoidance of intimacy, anxiety, depression, adjustment disorder, fear, suicidal ideation, and other psychiatric morbidity.”47 Recent studies have suggested that childhood vitiligo impacts quality of life similarly to psoriasis, as children enter adulthood.

An Internet-based survey of vitiligo probands ages 0 to 17 years and their families showed that pediatric vitiligo was associated with increasing

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**TABLE 1 Common Mimics of Vitiligo in Childhood**

<table>
<thead>
<tr>
<th>Differential diagnosis with nonsegmental vitiligo</th>
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</thead>
<tbody>
<tr>
<td>Congenital depigmentation (often with appearance by 2 y of age)</td>
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<tr>
<td>Albinism</td>
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<tr>
<td>Piebaldism (white forelock + ventral depigmentation)</td>
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<tr>
<td>Waardenburg syndrome</td>
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<tr>
<td>Tuberous sclerosis (leaf-ash depigmentation) (Fig 4A)</td>
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**Postinflammatory**

| Chemical depigmentation |
| Psoriasis |
| Atopic dermatitis |
| Lichen sclerosis et atrophicus |
| Morphea |
| Pityriasis alba (Fig 4B) |
| Pityriasis rosea |
| Sarcoidosis |
| Seborrhoeic dermatitis |
| Postinflammatory hypopigmentation |

**Infectious**

| Progressive macular hypomelanosis |
| Tinea versicolor (Fig 4C) |
| Tinea incognito |
| Neoplastic |
| Mycosis fungoides (Fig 4D) |

**Differential diagnosis with segmental vitiligo**

| Naevus depigmentosus/hypochromic nevus (Fig 4E) |

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* Associations between vitiligo and other inflammatory dermatoses may exist. In particular association of vitiligo and atopic dermatitis is frequently reported.
disturbances in quality of life with advancing age. When asked whether they were bothered by their vitiligo, only 4.1% of teenagers 15 to 17 years were not bothered by their disease versus 45.6% of children ages 0 to 6 years and 50% of children 7 to 14 years. In particular, facial and leg involvements were most distressing to the patient. Facial and arm lesions were most associated with teasing and bullying. Similarly, Bilgiç et al demonstrated in a cohort of 41 children ages 8 to 18 years versus controls that vitiligo had a negative impact on the quality of life. Head and neck in boys and genitalia and legs in girls were correlated with negative impact on quality of life. Camouflage of visible facial lesions has been shown to improve quality of life in children with vitiligo. Depression and anxiety have been reported in 26% and 42% of parents/caregivers of children with pediatric vitiligo.

**NATURAL COURSE OF THE DISEASE AND TREATMENT OPTIONS**

**Assessment of the Overall Involvement, Pace of Pigmentary Loss, and Sites and Types of Disease**

At first sight, defining the type of vitiligo that should be treated is of prime importance, as SV and NSV have different course, prognosis, and treatment options. Once the type of vitiligo has been clearly defined, the construction of a therapeutic and management plan may be started. Physicians should thoroughly examine their patient by using natural light and a Wood lamp for assessing disease extent. For that purpose, the use of the Vitiligo European Task Force questionnaire, which summarizes the results of the personal and family history of the patient along with clinical examination items, may be helpful. Skin phototype, disease duration, and extent and activity of the disease are important elements for guiding therapeutic management. In addition, the analysis of KP (ie, development of lesions at friction and traumatized sites) can reflect disease activity and is of particular interest for the prevention of relapses. Indeed, there is clinical evidence that in vitiligo, repeated trauma areas related to daily life habits (eg, hygiene or clothing) are more susceptible to KP, and a scoring of the probability of KP has been proposed. Finally, there is now strong evidence for the association of NSV with other autoimmune diseases, including autoimmune thyroid diseases, atopic dermatitis, diabetes type 1, alopecia areata, rheumatoid arthritis, ulcerative colitis, and pernicious anemia, as well as some less common autoimmune diseases.

**Natural Course of Disease if Treatment Not Rendered**

SV has a very well-defined course and is not associated with an autoimmune diathesis. The disease usually spreads over the involved segment over a 3- to 24-month period. KP, when present, is limited to the affected area. Early involvement of the hair follicular reservoir is the rule, and careful examination of the hairs in the affected area often reveals whitening.

**FIGURE 4**

One characteristic feature in SV is that once repigmentation has occurred, relapse is rare.

On the other hand, the course of NSV is unpredictable. The natural history of the disease tends to be cyclic and includes a silent phase, during which melanocyte destruction is minimal, and a so-called acceleration phase with rapid disease progression over a few weeks or months followed by a stabilization phase. Whitening of the hairs may appear later in the course of the disease. Repigmentation may occur without any therapeutic intervention or after sun exposure.

The number of lifetime cycles and triggering factors are different in each patient and yet still to be clearly elucidated. Epidemiologic studies support the role of stress episodes as a trigger of disease and/or relapse. An overall therapeutic algorithm for the treatment of vitiligo is given in Fig 5.

**Medical Workup**

The care of SV does not require medical workup, unless NSV or another systemic autoimmune disease is noted concurrently. The workup of children with NSV includes a complete blood count, metabolic profile, thyroid function screen (thyrotropin) and antibodies (anti-thyroid peroxidase and anti-thyroglobulin) to identify early cases of thyroid disease requiring closer follow-up, as well as 25 (OH) vitamin D, which when low may identify a subset of patients with greater propensity for secondary autoimmunity. Anti nuclear antibody (ANA) screening is recommended before phototherapy. Other screening can be performed based on the presence of signs and symptoms of other autoimmune diatheses (eg, polyuria, polydipsia, gum pigmentation, joint pain).
Topical Therapies

There is no approved or labeled treatment of vitiligo. Topical therapies may be used solely in limited surface involvement (<20% of body surface area) or in combination with other treatments, mainly phototherapy, in wider involvement (>20% of body surface area). There are 3 main classes of topical drugs that are used in vitiligo: topical steroids, topical calcineurin inhibitors, and topical vitamin D. The site of involvement should be taken into account for the choice between these different treatments. Hence, the European Dermatology Forum consensus group has recently published guidelines favoring the use of topical calcineurin inhibitors as a first-line option for face and neck on a bidual basis, as these have fewer side effects in these particular areas.53

Midpotency topical corticosteroids may be used for the rest of the body on a daily basis and in a sequential discontinuous scheme (eg, 1 week treatment and 1 week off for 6 months) to prevent local side effects (ie, skin atrophy, telangiectasia, hypertrichosis, acneiform eruptions, and striae). The use of topical vitamin D derivatives should be in combination with topical steroids.

Mineral Complex Cream

Mineral complex creams have been developed and tested in a broad age group of patients largely adjunctive to phototherapy. The principle of therapy has been the reduction of oxidative pigment cell damage that is felt to occur through tetrahydrobiopterin pathway breakdown. Variable results have been seen, with some centers reporting good outcomes with proprietary products. The initial clinical report54 described 33 patients ages 4 to 68 years. Pseudocatalase and calcium topically, combined with narrowband UVB or climatotherapy at the Dead Sea, has been described to be effective at repigmentation of facial and dorsal hand vitiligo. Not all mineral complexes benefit patients with vitiligo. For example, topical co-Q10 application is not advised because of potential triggering vitiligo through a putative mechanism of peroxide generation.55 Therefore, mineral complex cream is generally used adjunctively and should be used only when the product efficacy can be confirmed.

Sun Protection

Sun protection is generally advised in all patients, but is needed more so for areas of depigmentation. As a result, any sun exposure that is not sought for purposes of repigmentation should be paired with sun protection, including age-appropriate sun block or sunscreen, hats, sunglasses, and clothing.

Oral Vitamins and Supplements

There have been a few studies that suggest vitamin supplementation can enhance vitiligo outcomes. First, vitamin deficiencies have been noted in patients with vitiligo, including vitamin D, which has been linked to comorbid autoimmunity,56 and B-complex vitamins, including folate and B12.57 Hyperhomocysteinemia, which can be associated with vitamin B deficiencies, has also been linked to vitiligo.58,59 Low-dose vitamin supplementation, such as 400 IU Vitamin D3 daily, is common in childhood, but high-dose vitamin supplementation in childhood has limited data supporting its usage for childhood vitiligo, and in fact there are few safety data for usage of herbal remedies, such as gingko in childhood. One clinical trial that has looked at safety and efficacy of the amino acid phenylalanine in high dosage for vitiligo (a precursor to melanin) showed modest benefit in repigmentation.60,61

Minipulse Oral Steroids

Steroid pulse therapy refers to the intermittent administration of suprapharmacological doses of steroids. This method is weighted to reduce the side effects of steroids. No randomized placebo-controlled clinical trial has yet confirmed the interest of low-dose oral minipulse (OMP) steroids in vitiligo/NSV. However, several retrospective studies have underlined the interest of OMP of low doses of betamethasone or dexamethasone for 3 to 6 months in rapidly evolving vitiligo with the principal aim to halt disease progression.62,63 In addition, in a recent retrospective study, the early use of short-term systemic steroids in combination with targeted phototherapy and topical tacrolimus has shown to be effective in repigmenting SV.63 Although uncommon, side effects such as weight gain and acneiform eruptions have been described with the use of OMP.

Phototherapy

A variety of phototherapy modalities exist that have been shown to be beneficial in pediatric vitiligo. Generalized phototherapy is often performed in extensive disease and in disease that is spreading rapidly. Psoralens and UVA (PUVA) has been historically used in vitiligo with good benefit, but there is difficulty with nausea, compliance of eyewear, office visits, and many side effects including phototoxic reactions.64 Therefore, PUVA has been largely replaced by narrowband UVB (NB UVB). Furthermore, in head-to-head study, there has been demonstrable increased repigmentation that was not significant over PUVA.65 In children, NB UVB has become the therapy of choice and can produce 2 types of benefits: (1) repigmentation, and (2) stabilization, the latter being an important way to gain control over widespread disease. Njoo et al demonstrated >75% repigmentation in 53% of children.

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Cosmetics

Cosmetic camouflage, ranging from self-tanners to clothing alterations to concealers including stage-type make-up, have been used to reduce the clinical appearance of disease.\(^7^7\) Cosmetic camouflage make-up can be color matched to the skin, and children/parents can learn how to apply these on a daily basis or before major events to improve overall quality of life.\(^5^0\)

Surgical Grafting

Autologous grafting should be reserved to stable vitiligo lesions (ie, lesions with no progression for at least 1 year). The best indication for grafting is stable SV. Different grafting techniques have been described, including punch grafting, split-thickness skin graft, and the most recent is melanocyte transfer grafting. The main side effect of punch grafting is a cobblestoning effect.\(^7^8\) All these techniques remain painful, with potential scarring and/or mottled pigmentation side effects on the recipient area and possible KP on the donor site. Grafting techniques have shown great results, although there is a concern about the long-term maintenance of these results in vitiligo/NSV.\(^7^9\)

Clinical Consultation and Comanagement

Comanagement of vitiligo with the following practitioner types may be advisable at times. These include endocrinology (eg, thyroid management), rheumatology (eg, ANA-positive photosensitivity), nutrition (eg, known vitamin deficiencies), psychology/psychiatry (eg, anxiety), developmental specialists (eg, school-based difficulties arising from vitiligo), hematology (eg, pernicious anemia), gastroenterology (eg, suspected ulcerative colitis), and pediatrics (eg, for coordination of care).

Furthermore, patients and parents may benefit from referral to a support group whether online or in person. The following is a list of some support sites for patients with vitiligo worldwide:

1. Vitiligosupport.org
3. www.mynvfi.org

Conclusions

Vitiligo is a frequent cause of consultation and pediatricians and general physicians play a central role in its management. Indeed, as the disease should be referred early to potentialize treatment outcomes, they are at the forefront for referring patients to dermatologists and managing further treatment follow-up. Moreover, once the diagnosis has been confirmed by a dermatologist, early recognition of new flare-ups by pediatricians and general physicians will allow rapid therapeutic intervention to prevent the wide spread of the disease. Although there is no approved drug for the treatment of vitiligo, there is now an arsenal of therapeutic options that have proven efficacy in the management of the disease. Parents should be advised that the treatment is often long term and requires their adherence. The association of vitiligo with other autoimmune diseases should prompt physicians to carefully seek any autoimmune/autoinflammatory-associated disease. Finally, as the disease is disfiguring, one should not neglect its potential psychological impact, especially if the onset occurs during adolescence.

### ABBREVIATIONS

- ANA: antinuclear antibody
- KP: Koebner phenomenon
- NB UVB: narrowband UVB
- NSV: nonsegmental vitiligo
- OMP: oral minipulse
- PUVA: psoralens and UVA
- SV: segmental vitiligo
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