durante la salida de la misma. En segundo lugar, por reflujo de sangre u otros fluidos a través del trayecto de la aguja, llevándose consigo algunas de estas células tumorales hacia la superficie de la piel. Y, por último, debido a un aumento súbito de la presión intratumoral, como suele ocurrir en el desarrollo de la radiofrecuencia[4].

Dado que muchos de estos pacientes se encuentran en un estado terminal cuando las lesiones aparecen, se suele optar por una actitud conservadora en la mayoría de los casos. Sin embargo, la resección quirúrgica se ha llevado a cabo en algunas ocasiones. Cuando esto no es posible ya sea por enfermedad avanzada, *performance status* bajo u otras circunstancias, la radioterapia parece ser una alternativa razonable[2].

Como conclusión, la siembra tumoral a través del trayecto de la aguja en el transcurso de procedimientos percutáneos, si bien sucede excepcionalmente, representa la forma más frecuentemente implicada en la producción de metástasis cutánea en el caso del hepatocarcinoma.

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http://dx.doi.org/10.1016/j.ad.2014.10.014

Dermoscopic characterization of longitudinal melanocytic lesions on acral skin

Caracterización dermatoscópica de lesiones melanocíticas longitudinales sobre la piel acral

Acquired melanocytic nevi usually exhibit an oval or round shape on all body skin surfaces. Although this morphology is also the most frequent among nevi localized on the sole of the foot, we have observed a group of lesions in this location with a marked disproportion between width and length. This particular appearance has not been previously described, and it could be mistaken for malignancy. We present a series of 9 patients with elongated plantar nevi and discuss their main clinical, dermoscopic and histopathological features. Interestingly, all the lesions presented with a linear appearance, with a length greater than 7 mm. Although this measurement has been considered highly suggestive of suspicious malignant lesions in the literature, we have only found one melanoma among our cases. We would like to highlight the importance of this particular presentation of acral melanocytic nevus to avoid an incorrect diagnosis of malignancy.

We describe the clinical and dermoscopic characteristics of nine patients with a clinical diagnosis of melanocytic sole nevi who presented between 2007 and 2013 at the Pigmented Lesion Unit of the Dermatology Department of the General Hospital of Alicante (Fig. 1). The dermoscopic evaluation was performed by DermLite Foto™ (3Gen, LLC, Dana Point, CA, U.S.A.) mounted on a digital camera (Canon G9 and G12™) and with a digital videodermoscope (MoleMax II™). Melanocytic lesions with suspicious clinical or dermoscopic features were excised and histopathologically evaluated and cases without evidence of malignancy underwent videodermoscopic follow-up. Characteristics of the cases are summarized in Table 1.

The cases included 4 men and 5 women, ranging in age from 10 to 49 years old (median age 21) and all were Caucasian. The median length nevus was 17 mm, while the median width was 3.1 mm. Dermoscopy showed a combination of various types of acral benign dermoscopic patterns (parallel furrow and typical fibrillar patterns) in 8 out of the 9 nevi (Table 1). These melanocytic nevi without dermoscopic signs of malignancy were all closely monitored (every 6 months) by videodermoscopy without evidence of neoplastic transformation. Melanoma *in situ* was detected in one out of ten patients, whose lesion had shown an atypical dermoscopic parallel ridge pattern (Fig. 2). An analysis of the recorded images permitted us to observe a particular disproportion between length and width in these plantar skin nevi. Despite their large size (diameter > 7 mm is considered a dermoscopic criterion of suspicion of malignancy) and asymmetry, we have observed that most nevi with this morphology are benign lesions.

The clinical morphology and histology of melanocytic nevi are conditioned by the anatomical location of the lesions. The pressure supported by plantar skin determines the expression of particular clinical, dermoscopic and histological features, making the diagnosis of plantar melanocytic lesions sometimes difficult in these areas.[1] In the difficult clinical dermoscopic evaluation of pigmented lesions on acral skin, the most useful feature to rule out a melanoma is

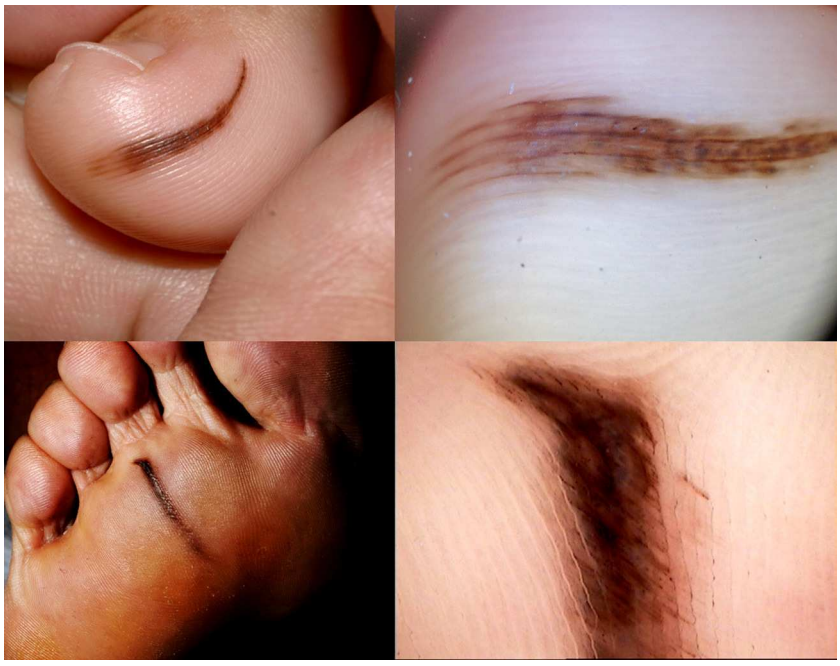

Figure 1 Clinical (A, B) and dermoscopic findings (C, D) of 2 of the 9 longitudinal nevi on the sole.

the absence of a parallel ridge pattern.[2–4] However, size has been also reported in the literature as a crucially important factor for the correct diagnosis of melanoma with nevoid features on the plantar area. Saida et al. studied 140 melanocytic lesions and only a few benign acquired melanocytic nevi on the sole were more than 7 mm in maximum diameter and none exceeded 9 mm.[5] There is widespread agreement that size of acral lesions is an important clinical criterion when considering malignancy, and nevi with diameters over 7 mm are at least suspicious.[5,6] It is important to note that five of our cases presented congenital nevi. Congenital lesions usually show a larger diameter

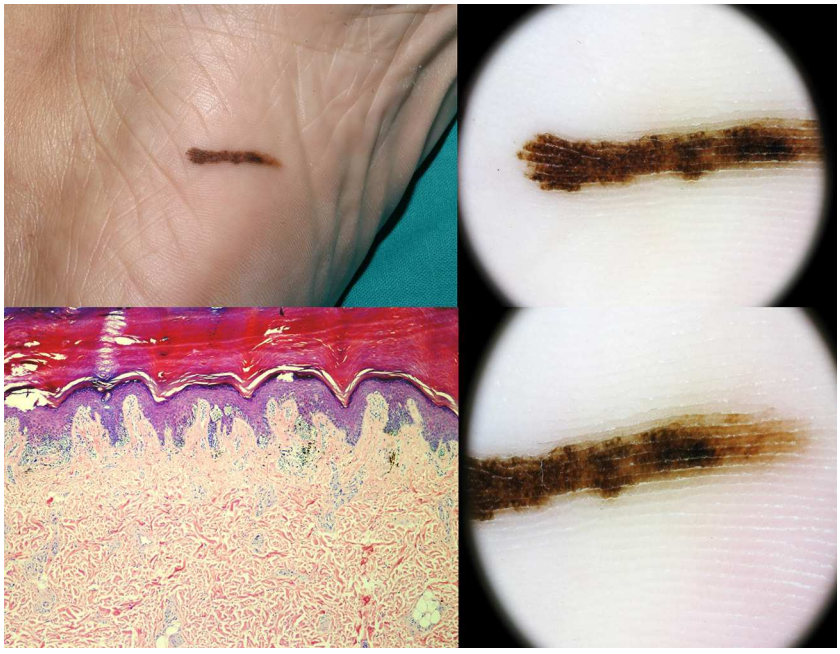

Figure 2 (A) One melanoma *in situ* was found among the 9 reported lineal nevi. (B) Histological study of the lesion was consistent with melanoma *in situ* (H–E × 10). (C, D) The lesion showed a characteristic dermoscopic parallel ridge pattern.

Table 1 Characteristics of the cases of the nine elongated nevi on the sole.

Age (years)/sex	Congenital/Adquired	Location	Dimensions (lengh × width)	Dermoscopy acral pattern	Histology	Time of follow-up
43/woman	Congenital	Forefoot, right sole	10 × 4 mm	Fibrillar pattern	Intradermal melanocytic nevus	–
47/man	Adquired	Forefoot, right sole	28 × 3 mm	Parallel furrow pattern + fibrillar pattern	No: follow up	28 months
24/woman	Adquired	First finger, right foot	17 × 3 mm	Lattice-like pattern	Combined melanocytic nevi	–
13/woman	Congenital	First finger, right foot	10 × 3 mm	Parallel furrow pattern	No: follow up	50 months
49/woman	Adquired	Midfoot	20 × 2 mm	Parallel ridge pattern	*In situ* melanoma	–
40/woman	Adquired	Forefoot, right sole	14 × 3 mm	Fibrillar pattern	No: follow up	55 months
10/man	Congenital	Midfoot, left sole	16 × 2 mm	Parallel furrow pattern + lattice-like pattern + broad, bluish-gray structureless	No: follow up	68 months
11/man	Congenital	Fourth finger, right foot	18 × 2 mm	Parallel furrow pattern + lattice-like pattern + broad, bluish-gray structureless	Combined melanocytic nevi	–
8/man	Congenital	Third finger, right foot	14 × 4 mm	Parallel furrow pattern + lattice-like pattern + broad, bluish-gray structureless	Combined melanocytic nevi	–

and are diverse in size, and they show more heterogeneous color and shape than acquired melanocytic nevi. In addition, acral nevi have characteristic dermoscopic features such as a globulostreak pattern, a homogeneous pattern, and a nontypical pattern.[7]

In conclusion, we have identified a subgroup of patients who presented with pigmented lesions located on the soles with a marked disproportion between length and width, with a diameter greater than 7 mm. We do not know the pathogenesis of the particular morphology of the nevi in our series, but we hypothesize that the distribution of melanocytes following the lines of Blaschko on the acral skin could be responsible for this particular shape. These pigmented nevi showed variable dermoscopic findings, without any particular location on the sole. Although this larger size is regarded as a suspicious finding in itself, we suggest that nevi with this particular morphology are benign lesions in most cases when associated with typical dermoscopic criteria. We recommend the early extirpation of all suspicious nevi presenting a parallel ridge pattern of any size and morphology, and a close follow-up of elongated lesions over 7 mm in their maximum diameter with benign parallel furrow dermoscopic pattern (and its variants), until these particular melanocytic lesions have been better characterized.

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http://dx.doi.org/10.1016/j.ad.2014.10.015

Idiopathic Acquired Leukonychia Totalis of the Fingernails in a Child Treated Successfully with Zinc and Amino Acid Supplementation

Leuconiquia totalis idiopática, adquirida de las uñas de las manos tratada con éxito mediante suplementos de zinc y aminoácidos

Leukonychia is whitening of the nail plate. In 1919, Mees first described leukonychia in arsenic intoxication.[1] Baran classified leukonychia into true, apparent, and pseudo-leukonychia. True leukonychia can be acquired or inherited, and based on the distribution of white blotches, can be further subclassified as leukonychia punctata, leukonychia striata, leukonychia partialis, or leukonychia totalis.[2] There are very few reported cases of idiopathic acquired leukonychia totalis[3] and we report the present case to demonstrate this uncommon clinical entity in a 10-year-old boy and his response to micronutrient supplementation.

A 10-year-old boy presented at our clinic with porcelain white finger nails that he had had since 4 years of age. Leukonychia totalis and leukonychia striata were seen on the fingernails, with leukonychia partialis in both the thumbnails (Fig. 1). A detailed history demonstrated progression from leukonychia partialis to striata and totalis over the years. The strength of the nail plate was normal. Pressure over the nail plate caused no fading of the leukonychia, suggesting nail matrix origin and ruling out Muehrcke's lines (apparent leukonychia striata), a common clinical differential diagnosis.[3] His hair, eyes, teeth, and the remainder of the cutaneous examination were normal. There was no clinical evidence of malnutrition, alopecia areata, psoriasis, or lichen planus. The patient gave no history of preceding illness, surgery, trauma, chemical exposure and drug intake, including herbal medicines. The patient was born out of a nonconsanguinous marriage and there was no family history of leukonychia. Repeated cultures and microscopic examination of the nail clippings with 10% potassium hydroxide were negative. All the routine investigations, including serum proteins, liver function tests, serum calcium, and zinc levels were within normal limits. With the aforementioned clinical and laboratory assessment a diagnosis of idiopathic leukonychia partialis to totalis of the fingernails was established. The patient's parents did not consent to a nail biopsy.

The boy was started orally on Zinc sulfate 137.5 mg (containing 50 mg elemental zinc) capsule once daily, along with a single daily capsule of 8 essential amino acids and vitamins A, B complex, C, D, and E (containing 18.3 mg L-leucine, 5.9 mg L-isoleucine, 25 mg L-lysine hydrochloride, 5 mg L-phenylalanine, 4.2 mg L-threonine, 6.7 mg L-valine, 5 mg L-tryptophan, 18.4 mg DL-methionine, 2500 IU vit A, 200 IU vit D, 5 mg vit B1, 3 mg vit B2, 25 mg vit B3, 5 mg vit B5, 1.5 mg vit B6, 2.5 mg vit B12, 0.75 mg folic acid, 40 mg vit C, and 7.5 IU vit E). Two monthly follow-ups showed serial improvement at each visit, and complete resolution of the leukonychia was observed at the end of 7 months (Fig. 2). The treatment was continued for 3 months after resolution of the leukonychia, and there was no relapse in the 6 months after treatment was discontinued. The patient is presently monitored.

Leukonychia is the most common chromatic abnormality of the nail; however the physiologic mechanisms causing it are not entirely clear and Newton's theorem (i.e. a surface appears white when it reflects the radiation of visible light), has been proposed to explain leukonychia. In true leukonychia there is abnormal matrix keratinization, with persistent parakeratosis and keratohyalin granules in the nail plate, which might play a role in the modification of the light reflection by the ungual plates.[3] Baran classified leukonychia into the following 3 primary types: (1) true leukonychia, where in the nail plate involvement originates in the matrix; (2) apparent leukonychia, in which the pathology lies in the subungual tissue; and (3) pseudo-leukonychia, which is due to keratin granulations, as seen in superficial white onychomycosis.[2] Inherited leukonychia can be presented as an isolated condition or as one of the several other reported syndromes. There is an autosomal, dominantly inherited syndrome in which leukonychia occurs in combination with kidney stones and sebaceous cysts, with sensory-neural deafness and knuckle pads, which is known as the Bart-Pumphrey syndrome.[3] Popular lay media claim that the etiology of leukonychia is due to calcium and/or zinc deficiency. However, no studies are available in the scientific literature to support or refute these claims.[5] Acquired leukonychia has been reported due to trauma, drugs such as chemotherapeutic agents (e.g., anthracyclines, cyclophosphamide, vincristine, cyclosporine, fluorouracil, and methotrexate), and systemic or local infections (e.g., typhoid fever, hepatic cirrhosis, ulcerative colitis, leprosy, and recently due to selenium deficiency in Crohn's disease).[3,5,6] In children, increased requirements for macro- and micronutrients