Amelanotic Subungual Melanoma Mimicking Telangiectatic Granuloma: Clinical, Histologic, and Radiologic Correlations

Melanoma amelanótico subungueal simulando granuloma telangiectásico. Correlación clínica, histológica y radiológica

To the Editor:

Subungual melanoma is uncommon and accounts for just 2% to 3% of all cutaneous melanomas. Less than 10% of subungual melanomas are amelanotic and 90% of these occur on the thumb or great toe. They typically appear in the seventh decade of life and are associated with a delayed diagnosis and a poor prognosis.1-4 Amelanotic subungual melanoma generally manifests as a persistent vascular or ulcerated nodule. On ultrasound (US), subungual melanoma appears as a moderately well-defined hypoechogenic area with increased thickness and decreased echogenicity of the nail bed, which frequently erodes into the bone margin of the distal phalanx, viewed as a hypoechogenic structure pushing up and eroding into the bilaminar hypoechogenic structure of the nail plate. Color Doppler US shows marked hypervascularization. Telangiectatic granuloma is the main entity to be considered in the differential diagnosis. This benign acquired vascular tumor is relatively common, and frequently affects the nail, the nail bed, and the periungual tissues.5 On US, subungual telangiectatic granuloma appears as a focal hypoechogenic area with thickening and decreased echogenicity of the nail bed, which pushes up the nail plate but does not erode into the bone margin of the distal phalanx; hypervascularization is also observed.6 Subungual melanoma requires a high index of clinical suspicion given its similarity to other conditions. We report on such a case evaluated at our department.

A 67-year-old man presented with a fast-growing asymptomatic lesion of 1 month’s duration on the nail of his left great toe. He reported bleeding following minor trauma (Fig. 1). Physical examination revealed a friable subungual erythematous nodule with a vascular appearance associated with disruption of the nail plate. Color Doppler US showed increased thickness and a diffuse decrease in echogenicity of the nail bed. Diffuse hypervascularization

Figure 1 Erythematous subungual nodule.

Figure 2 Histology. Ulcerated amelanotic melanoma; Breslow depth, 3 mm.

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of the nail bed was also observed. These findings were consistent with subungual inflammation associated with granulomatous changes predominantly affecting the central zone, suggesting a preliminary diagnosis of telangiectatic granuloma (Fig. 2). The lesion was surgically excised with wide margins, and the histologic evaluation showed nodular, ulcerated malignant melanoma of the nail bed, with a Clark level III, a Breslow depth of 3 mm, and a mitotic rate of 10 mitoses/mm² (Fig. 3).

Amelanotic subungual melanoma generally manifests as a persistent vascular or ulcerated nodule. The differential diagnosis is broad and includes glomus tumor, squamous cell carcinoma, and, most importantly, telangiectatic granuloma. Some of the most useful characteristics that can help to distinguish between these conditions are summarized in Table 1.

Clinically, subungual melanomas have a dull erythematous surface, while the majority of vascular tumors have a bright red surface. They also tend to bleed less and grow slowly, over the course of months or years, unlike telangiectatic melanoma, which typically grows in a matter of weeks. There may be a history of trauma in up to 25% of cases of subungual melanoma. The case of amelanotic subungual melanoma described in this report was difficult to diagnose by US, as it lacked well-defined borders and erosion of the bone margin of the distal phalanx, 2 common features of this tumor. In addition, the ability of US to detect pigment is currently limited.

A strong correlation has been reported between tumor thickness measured by color Doppler US and Breslow depth (histology) in cutaneous melanoma, and US is therefore a useful additional tool for investigating suspected cases of subungual melanoma. Although color Doppler US is used to study a wide variety of nail lesions, it has only been used in isolated cases of amelanotic subungual melanoma and none of the studies have used plain US.

Early studies reported 5-year survival rates of close to 16% for subungual melanoma, and even lower rates were described for patients who had undergone distal amputation at the metacarpophalangeal or metatarsophalangeal levels. Proximal amputation is thus the current treatment of choice.

Mohs micrographic surgery has also been proposed as a potentially interesting treatment for subungual melanoma. The drawback, however, is that it has been used in small series and more cases are needed to determine its effectiveness.

In conclusion, amelanotic subungual melanoma is difficult to diagnose both clinically and sonographically. It is therefore essential to rule out other malignant or benign tumors and to perform an early biopsy. Amelanotic subungual melanoma can mimic subungual telangiectatic granulomas on US, particularly in the absence of erosion of the phalanx or nail plate.

**Conflicts of Interest**

The authors declare that they have no conflicts of interest.

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**References**

Polyarteritis Nodosa with a Systemic Inflammatory Response Pattern: Effectiveness of anti-TNF

Panarteritis nudosa con patrón de respuesta inflamatoria sistémica: respuesta a anti-TNF

To the Editor:

Polyarteritis nodosa (PAN) is a vasculitis that affects small and medium-sized vessels. It is traditionally divided into 2 forms. The cutaneous form is the variant in which the clinical manifestations are limited to the skin, though muscle pain and generalized joint pain may also be reported, and neuritis in areas close to the affected skin. Fever is detected in up to a third of cases. The systemic form affects internal organs, most commonly the kidney, liver, and central nervous system.1

A 50-year-old man was seen in our department for a 5-year history of outbreaks of nodular lesions, initially only affecting the legs, associated with widespread joint pain. In the last year, lesions had also appeared on his arms, and he had developed episodes of fever, weight loss, asthenia, and anorexia. He had been on follow-up at another hospital, where several biopsies had been performed with nonspecific results (a dermal neutrophilic infiltrate, stasis dermatitis, and, in the most recent biopsy, a predominantly lobular panarculitis). He had received no definitive diagnosis. Despite treatment with various drugs, control of his disease had not been achieved; throughout this whole period he had been administered oral corticosteroids, with intermittent courses of colchicine, azathioprine, methotrexate, and ciclosporin. Important blood test results included elevation of the acute phase reactants (AFRs), with elevation of C-reactive protein and erythrocyte sedimentation rate (which had risen to 120 mm/h in recent months). Other additional tests, including autoimmunity studies (antineutrophil antibodies, extractable nuclear antigen, and antineutrophil cytoplasmic antibodies) and serology (HIV and hepatotropic viruses), were normal or negative.

When the patient first came to our hospital, he presented erythematous plaques with peripheral desquamation, mainly on his lower limbs but also on his upper limbs. Nodules were present beneath the plaques. He also presented edema of the dorsum of the left foot and ankle (Fig. 1, A–C). A biopsy was taken, which was consistent with septal panarteritis (Fig. 1D).

Coinciding with tapering of the dose of prednisone, the patient presented a further outbreak at the same sites as the previous outbreak, with purpuric plaques that spread centrifugally, pain, bilateral ankle edema, and fever.

A further biopsy revealed a neutrophilic infiltrate in the small and medium-sized vessels, with fibrinoid necrosis, and a moderate eosinophilic infiltrate (Fig. 2, A and B). Based on these findings, we made a diagnosis of PAN. Given the absence of organ involvement, despite the marked elevation of the AFRs and alteration of the patient’s general state of health, we considered this to be cutaneous PAN with an associated systemic inflammatory response.

After increasing the dose of prednisone to 1 mg/kg/d and controlling the acute outbreak, treatment was started with cyclophosphamide and immunoglobulins. However, this did not enable us to reduce the dose of corticosteroids without renewed deterioration. Treatment was therefore commenced with infliximab at a dose of 5 mg/kg, which led to a very good response after the first infusion, with complete normalization of laboratory parameters. The dose of corticosteroids was progressively reduced until their withdrawal, with no further outbreaks of lesions in a year of follow-up, except for a residual livedo (Fig. 2C); during this period the patient continued treatment with infliximab every 8 weeks.

The typical clinical manifestations of cutaneous PAN are characterized by outbreaks of painful nodular lesions that leave a residual livedo.1 In the first patient series, dating from 1974, the presence of a starburst pattern was described...