CLINICAL VIGNETTE

A Case of Idiopathic Onychomadesis

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Case

A 36-year-old male with a past medical history of major depressive disorder, polysubstance abuse (alcohol and cocaine), G6PD deficiency, and latent TB presented to the emergency department for evaluation of fingernail loss. Two weeks prior to presentation, he noticed blood under the nails of his right hand 2nd and 3rd digit and left hand 3rd digit. The nails lifted and eventually came off. He reported that his fingers were edematous with intermittent paresthesias. He denied any trauma to the fingers and otherwise reported to be in his normal state of health. He was currently using tretinoin cream for acne and recently resumed taking sertraline.

He was comfortable with unremarkable vital signs. Right hand was remarkable for exposed nailbeds on the second and third digits. His left hand was remarkable for an exposed nailbed on his third digit. His right second finger nailbed had an ill-defined dark red discoloration. The periungal areas had no edema or erythema. There was no evidence of skin breaks in his bilateral hands. Capillary refill was less than two seconds on all digits.

Labs

Complete blood count and basic metabolic panel were within normal limits. Autoimmune labs were drawn in the emergency department including: ESR, CRP, ANA, and cyroglobulins.

Clinical Course

The case was discussed with rheumatology and dermatology. Assessment by both services concluded the patient's symptoms were not consistent with Raynaud's disease or scleroderma and would require a more extensive outpatient work up. The patient was discharged from the emergency department with close outpatient follow up with rheumatology and dermatology. He was instructed to discontinue all medications.

On outpatient follow up the patient had new nail growth suggesting intact nail plates. His autoimmune laboratory workup was negative. Therefore, consultants felt that this was medication induced or idiopathic onychomadesis. Follow up with the patient two years after presentation revealed that he has not had a recurrence of onychomadesis.

Discussion

The diagnosis of onychomadesis is rare and will not usually be made at the index emergency department or clinic visit. However, it is important for primary care and emergency physicians to familiarize themselves with nail disorders as they may provide a window into underlying undiagnosed pathology. Traditional clinical training may not focus on thorough examination of nails. Knowledge of common nail disorders can be a handy bedside diagnostic tool.

History should focus on the age at onset, possible inciting events two or three months prior to presentation, social history, and known systemic or dermatologic conditions. The age at onset may help differentiate between benign and malignant conditions. For example, a patient with nail discoloration (i.e., band of melanonychia or dark pigmentation) present at birth or childhood suggests a benign process, whereas onset in adulthood may need to rule out malignancy. History of possible inciting events two or three months prior to presentation is important as nails grow slowly and damage to the nail matrix may not be apparent initially. Social history that focuses on work, hobbies, and hygiene habits is also important in ruling out common nail issues.

Pathophysiology

The nail matrix produces the cells that eventually become the nail plate. The dorsal aspect of the nail plate is created by the proximal matrix while the ventral aspect of the nail plate is created by the distal matrix.[1] Disturbances in the dorsal and ventral nail growth results in deformities in the nail. The deformity can range from slight indentation (Beau's lines) to onychomadesis. Onychomadesis occurs when the nail plate separates from the matrix with persistent attachment of the nail to the nail bed. Often the nail will shed completely. Complete replacement of the nail can take approximately 6 months in the fingers and 12 months in the toes.[1]

Onychomadesis has been associated with infection, autoimmune disease, critical illness, and medications. The exact mechanism of nail matrix arrest in the setting of infection, systemic disease, or drug exposure is unknown. In medication induced onychomadesis, acute toxicity to the nail epithelia or inhibition of cellular proliferation have been described as possible mechanisms.[2]

Autoimmune diseases associated with onychomadesis includes alopecia areata and pemphigus vulgaris.[3] Onychomadesis is the second most common nail finding in pemphigus vulgaris.[4] Severe Guillain-Barré syndrome, major depressive disorder, Stephen-Johnsons Syndrome, meningitis and other major medical illnesses have been associated with onychomadesis. In pediatric patients, hand-foot-mouth disease (HFMD), which is commonly caused by Coxsackie, has been well documented as a cause of onychomadesis.[2]

Most reports of medication induced onychomadesis are anecdotal. Chemotherapeutic agents, such as such as capecitabine, doxorubicin with cytosine arabinoside, and etoposide, are the most common cause of medication induced onychomadesis.[5,6] Onychomadesis has also been reported to be caused by antiepileptics medications, specifically carbamazepine and valproic acid.[7,8] Other case reports linked onychomadesis to azithromycin, retinoids, lead and lithium.[9]

Idiopathic cases of onychomadesis are rare. These cases can be classified as familial or sporadic. In familial idiopathic cases of onychomadesis an autosomal dominant pattern has been observed.[2]

Treatment

Elimination of the underlying cause of onychomadesis typically leads to the eventual regrowth of the nails. Nail specific treatments of onychomadesis include treatment of the nail with 40% urea under occlusion, and topical halcinonide under occlusion.[10,11]

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