International Journal of Contemporary Pediatrics
Nandagopal SS et al. Int J Contemp Pediatr. 2025 Sep;12(9):1571-1573
http://www.ijpediatrics.com
pISSN 2349-3283 | eISSN 2349-3291

DOI: https://dx.doi.org/10.18203/2349-3291.ijcp20252613

Case Report

Sandpaper nails: an uncommon tale of twenty-nail dystrophy in a 5-year-old

Nandagopal S. S.*, Jewel Maria George, Carol Sara Cherian, Jacob Abraham

Department of Paediatrics, Pushpagiri Institute of Medical Sciences and Research Centre, Thiruvalla, Kerala, India

Received: 17 June 2025
Revised: 14 July 2025
Accepted: 21 July 2025

***Correspondence:**
Dr. Nandagopal S. S.,
E-mail: drngs2k15@gmail.com

ABSTRACT

Trachyonychia, also known as twenty-nail dystrophy (TND), is a rare nail matrix disorder in children, often idiopathic but occasionally linked to dermatologic or immunologic conditions. Characterized by brittle, ridged, and roughened nails, it may coexist with systemic or cutaneous manifestations. We present the case of a 5-year-old girl admitted for fever and myositis, with a background of recurrent respiratory tract infections. On examination, she was found to have TND, patchy alopecia, dry skin, and bilateral medial madarosis-features suggestive of atopy. Anthropometric assessment indicated undernutrition. Her immunologic profile revealed significantly reduced serum IgA levels, with other immunoglobulins within normal range. Workup for autoimmune and malabsorptive conditions was negative. Dermatological consultation confirmed trachyonychia, and topical JAK inhibitor (Tofacitinib) was prescribed. Supportive care and close follow-up were advised. This case highlights a rare pediatric presentation of trachyonychia associated with features of atopy and selective IgA deficiency. The findings raise the possibility of an immunologic basis for TND in some children. Awareness of such associations can facilitate early diagnosis, comprehensive evaluation, and individualized management strategies in affected patients.

Keywords: Trachyonychia, Twenty-nail dystrophy, Selective IgA deficiency, Atopy, Pediatric nail disorders

INTRODUCTION

Trachyonychia, often referred to as twenty-nail dystrophy (TND), was first described in the late 1970s in children presenting with dystrophic changes in all fingernails and toenails. Over time, the term "trachyonychia" has become more appropriate, as not all cases involve all 20 nails. It denotes a specific pattern of nail roughness caused by proximal nail matrix disorder.[1] Clinically, the nails appear rough, brittle, and lack their normal shine. In milder forms, the surface shows fine longitudinal ridges, pitting, and a shiny, satin-like texture. More severe cases may exhibit a sandpaper-like appearance with diffuse opacification and a whitish-gray discoloration.[2] Although the condition is frequently idiopathic, familial forms have been documented with autosomal dominant inheritance patterns.[3] Trachyonychia has also been reported in monozygotic twins, highlighting a potential genetic basis.[4] It can present at birth or develop during early childhood, with the most common age of onset being between 3 and 12 years.[5] Both males and females can be affected equally, although trachyonychia associated with alopecia areata may show a slight male predominance. While any number of nails may be involved, the cosmetic impact of the condition is often significant, despite the absence of pain or systemic involvement. This case study describes a 5-year-old female child who presented with progressive roughness and discoloration of all twenty nails, and was subsequently diagnosed with idiopathic trachyonychia; she was managed conservatively with topical therapy and regular follow-up, showing gradual improvement without invasive interventions.

CASE REPORT

A 05-year-old girl presented at our OPD with complaints of fever and myalgia lasting for around 03 days at the time of presentation. Her history was non-contributory to the common causes of pyrexia and she was admitted to ward for a detailed work up. Mother gave history of recurrent respiratory tract infections in her since 03 years of age and history of alopecia which was noted evidently in the child around 02 years back. She was born as a term infant whose pre and perinatal course was unremarkable. There were no histories of jaundice, diabetes, hyper and hypothyroid, and of other skin diseases (porphyria, pellagra, psoriasis, herpes, syphilis, contact irritants). Histories of long-term consumption of drugs (antibiotics or steroids), pulmonary infection, and allergies were denied. There was no history suggestive of alopecia areata, psoriasis, lichen planus or other dermatological disorders in the family. On examination general condition of the child was satisfactory, she was noted to have thin, brittle, rough, non-pitting nails in all fingers and toes, patchy alopecia, dry skin and bilateral medial madarosis. Anthropometric assessment showed she was underweighting with weight falling below the 3rd centile in IAP growth chart. Other growth parameters were normal for age. Developmental assessment done was found to be normal.

Preliminary blood investigations were within normal limits except for raised levels of CRP and CKT. Lepto IgM send was also negative. Thyroid profile and serum vitamin D levels were within normal limits. Considering a history of recurrent respiratory tract infections, atopy and undernutrition, the possibilities of cystic fibrosis, malabsorption syndrome, SLE and primary immunodeficiency syndromes were entertained. A stool fat screening was done which ruled out malabsorption syndromes and cystic fibrosis. ANA ELISA tested negative, excluding the possibility of SLE. An immunoglobulin profile was sent which revealed low serum levels of IgA. An expert dermatology consultation sought and they advised the use of tofacitinib ointment (JAK 2 inhibitor) for a month. Child was managed with supportive measures and was suggested regular follow up for monitoring progression of condition.

Figure 1: Thin brittle trachyonychia.

Table 1: Immunoglobulin profile of the child.

Immunoglobulin	Serum levels	Normal range
Total IgA	0.12 (Low)	0.27-1.90
Total IgG	5.56	5-11.7
Total IgM	0.62	0.5-1.8

Figure 2: Patchy non cicatricial alopecia.

DISCUSSION

The etiology of trachyonychia is heterogeneous, and it may present as an idiopathic disorder or be associated with various dermatoses, particularly alopecia areata, lichen planus, psoriasis, and atopic dermatitis. In children, it is more commonly idiopathic, whereas in adults it often accompanies autoimmune or inflammatory skin disorders. Trachyonychia develops due to an inflammatory process targeting the proximal nail matrix, the site primarily responsible for nail plate formation. This inflammation, often lymphocyte-mediated, leads to structural damage within the matrix epithelium, including intercellular edema (spongiosis) and infiltration of inflammatory cells. Such disturbances impair keratinocyte differentiation and disrupt normal keratin synthesis, resulting in nails that appear coarse, fragile, and ridged. The extent of these changes is influenced by the degree and duration of inflammation-mild inflammation typically produces subtle ridging with a satin sheen, whereas chronic or intense inflammation can lead to markedly rough, discolored, sandpaper-like nails. Histological examination, though infrequently undertaken, may reveal spongiosis, inflammatory infiltrates, and occasionally hypergranulosis, especially in cases linked to underlying skin disorders.[6]

Trachyonychia, has been associated in rare instances with childhood selective immunoglobulin A (IgA) deficiency. A classic case series published in the journal of pediatrics in 1982 reported children with recurrent infections who also exhibited the distinctive nail changes of TND alongside markedly reduced serum IgA levels. This

association suggests that an underlying impairment in mucosal immunity may predispose susceptible children to subclinical or overt inflammation of the nail matrix, disrupting normal nail growth. Although uncommon, such findings highlight the importance of considering immunodeficiency screening-particularly measuring serum IgA-in pediatric patients presenting with unexplained multi-nail dystrophy and a concomitant history of recurrent infections or atopic features.[7]

The condition may present in two clinical variants: a shiny variant with smooth, thin, and opalescent nails, and a rough variant with brittle, thin, and sandpaper-like nails. Histopathological examination is rarely performed due to the invasiveness of nail matrix biopsy, but when undertaken, findings often reflect the underlying inflammatory dermatosis, with spongiotic or lichenoid interface dermatitis being typical.[8]

Management of trachyonychia can be challenging and depends on the severity of symptoms and any underlying disease. It is a self-limiting condition in many children and often warrants only observation unless associated with significant cosmetic concern or underlying dermatoses such as lichen planus or alopecia areata. First-line therapy typically includes topical corticosteroids or calcineurin inhibitors, though their efficacy is limited by poor nail penetration. Systemic agents like retinoids and cyclosporine may be considered in severe, refractory cases, while intralesional corticosteroids can be effective but are painful and challenging in pediatric patients. Emerging reports have highlighted the potential role of topical Janus kinase inhibitors such as tofacitinib, especially in immune-mediated cases, offering a promising and less invasive therapeutic approach. Though nail matrix biopsy remains the gold standard for histologic confirmation, it is not routinely indicated due to the invasive nature and risk of permanent scarring, and should be reserved for atypical presentations or diagnostic uncertainty. Management should be individualized, balancing the risk-benefit ratio while ensuring psychosocial reassurance and periodic monitoring of nail growth and appearance.[9,10] The prognosis of trachyonychia is generally favorable, especially in idiopathic pediatric cases, which often resolve spontaneously over months to years. However, recurrence or chronicity may occur in those with underlying autoimmune dermatoses.

CONCLUSION

This report illustrates a rare pediatric presentation of twenty-nail dystrophy in the setting of selective IgA deficiency and atopic features. The case underscores the need for clinicians to consider underlying immunologic and systemic associations when evaluating nail dystrophies in children. Early recognition and a multidisciplinary approach can aid in appropriate diagnosis and long-term care. Further research into the immunological underpinnings of trachyonychia may improve understanding of its pathogenesis and therapeutic options.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Tosti A, Bardazzi F, Piraccini BM, Fanti PA. Idiopathic trachyonychia (twenty-nail dystrophy): A pathological study of 23 patients. Br J Dermatol. 1994;131(6):866-72.
2. Sehgal VN. Twenty nail dystrophy trachyonychia: an overview. Dermatol. 2007;34(6):361-6.
3. Pavone L, Li Volti S, Guarneri B, Rosa ML, Sorge G, Incorpora G, et al. Hereditary twenty-nail dystrophy in a Sicilian family. J Med Genet. 1982;19(5):337-40.
4. Karakayali G, Lenk N, Gungor E, Gür G, Alli N. Twenty-nail dystrophy in monozygotic twins. J Eur Acad Dermatol Venereol. 1999;12(3):192-3.
5. Dehesa L, Tosti A. Treatment of inflammatory nail disorders. Dermatol Ther. 2012;25(6):525-34.
6. Jacobsen AA, Tosti A. Trachyonychia and twenty-nail dystrophy: A comprehensive review and discussion of diagnostic accuracy. Skin Appendage Disord. 2016;2(1-2):7-13.
7. Leong AB, Gange RW, O'Connor RD. Twenty nail dystrophy (trachyonychia) associated with selective IgA deficiency. J Pediatr. 1982;100(3):418-20.
8. Mahajan R, Kaushik A, De D, Handa S. Pediatric trachyonychia-A retrospective study of 17 cases. Indian J Dermatol. 2021;66(6):689-90.
9. Grover C, Bansal S, Nanda S, Reddy BSN. Trachyonychia: A comprehensive review. Indian J Dermatol Venereol Leprol. 2011;77(5):640-5.
10. Dhayalan A, King BA. Tofacitinib citrate for the treatment of nail dystrophy associated with alopecia universalis. JAMA Dermatol. 2016;152(4):492-3.

Cite this article as: Nandagopal SS, George JM, Cherian CS, Abraham J. Sandpaper nails: an uncommon tale of twenty-nail dystrophy in a 5-year-old. Int J Contemp Pediatr 2025;12:1571-3.