Pictorial essay

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The "sunken sphinx head" sign: a novel ultrasound marker of dorsal hood lesions

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Abstract

Post-traumatic lesions of the dorsal metacarpophalangeal joint are typically described in the context of sagittal band injury. However, certain deep-layer disruptions may occur independently of sagittal band involvement. This report introduces a distinct lesion pattern, termed the dorsal radial burst, characterized by a longitudinal split in the deep fibrocartilaginous component of the dorsal hood (the dorsal triangular fibrocartilage), with preservation of the overlying sagittal band. Three patients with persistent dorsal metacarpophalangeal pain, swelling, and extension weakness following closed trauma demonstrated consistent sonographic findings. High-frequency dynamic ultrasound revealed a focal longitudinal defect in the dorsal triangular fibrocartilage, accompanied by localized extensor tendon herniation into the defect but without tendon (sub)luxation. An overview of the layered anatomy of the dorsal hood is provided to clarify the imaging findings and the underlying mechanism.

Introduction

The dorsal triangular fibrocartilage (TFC) of the metacarpophalangeal (MCP) joint is a key stabilizing component of the dorsal hood complex, yet it remains underrecognized in the imaging literature[1–3]. This study reviews the layered anatomy and imaging features of the dorsal hood, focusing on the deep fibrocartilaginous layer, designated as the dorsal TFC to distinguish it from the triangular fibrocartilage complex (TFCC) of the wrist. The dorsal hood comprises the dorsal TFC and the overlying sagittal bands (SB), forming a retinacular–capsular structure that maintains extensor tendon alignment during motion[1–3]. Although SB injuries are well described as a cause of extensor tendon subluxation[4–9], isolated lesions of the dorsal TFC may produce dorsal MCP pain or extension weakness without instability and can easily be overlooked[1–3,10,11]. This work reviews the imaging anatomy of the dorsal hood using high-frequency dynamic ultrasound[2,3,10,12] and describes a newly recognized injury pattern of the dorsal TFC, termed the dorsal radial burst lesion, which is identified by the new "sunken sphinx head" sign. Recognition of this entity broadens the diagnostic understanding of dorsal hood trauma and supports a layered, anatomy-based ultrasound approach to dorsal MCP assessment[1–3,10].

Anatomy of the dorsal hood

The dorsal hood of the MCP joint is a multilayered retinacular–capsular structure that stabilizes the extensor mechanism and coordinates tendon motion during finger flexion and extension[1–3] (Fig. 1). Its deepest component is a distinct fibrocartilaginous plate, referred to here as the dorsal TFC. This structure appears triangular in the longitudinal plane and crescent-shaped in the axial plane[1–3]. By creating a dorsal sulcus and elevating the extensor tendon from the articular surface, the dorsal TFC provides a low-friction gliding bed and contributes to dorsal load distribution across the MCP joint[1–3]. Superficial to the dorsal TFC, the SB form a dynamic fibro-osseous sling that centers the extensor tendon and resists radial–ulnar translation. The radial SB is the primary stabilizer; isolated radial

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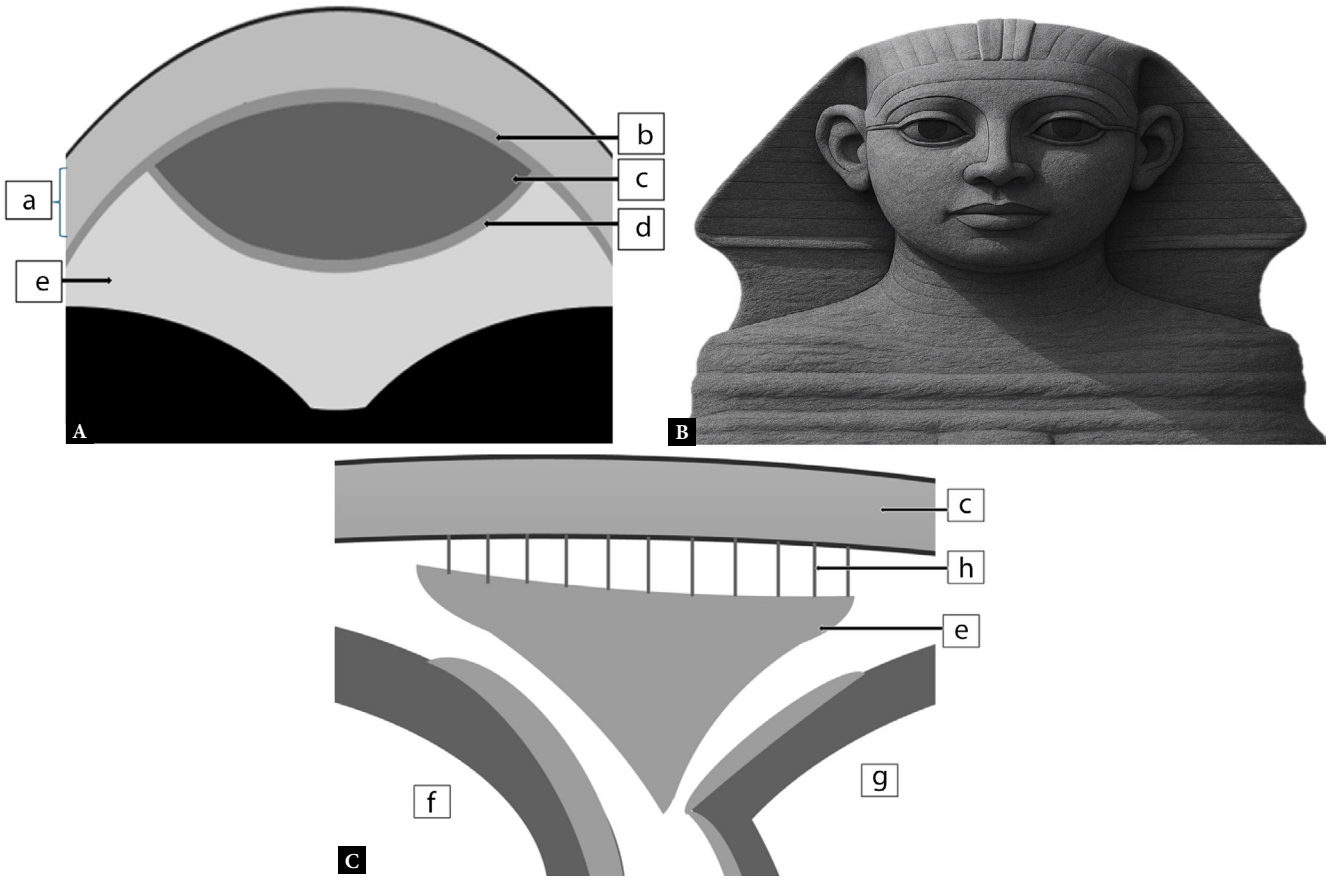


Fig. 1. *Schematic representation of the dorsal hood.* **A.** *Axial layered anatomy showing the superficial subcutaneous tissue (a); the extensor tendon (c) encircled by the superficial (b) and deep (d) sagittal bands (SB); and the dorsal triangular fibrocartilage (TFC) (e) forming a gliding platform for the tendon and bands.* **B.** *Conceptual "sphinx" view, with the extensor tendon and SB forming the "head" and the dorsal TFC representing the "shoulders," illustrating normal spatial relationships.* **C.** *Sagittal layered view at the metacarpophalangeal joint showing the extensor tendon (c) passing over the metacarpal head (f) and the base of the proximal phalanx (g). The dorsal TFC (e) forms a triangular gliding bed for the tendon and is connected to the extensor tendon and SB via loose connective tissue, the intertendinous fascia of Landsmeer (h).*

disruption consistently results in ulnar tendon subluxation during MCP flexion, whereas isolated ulnar injury rarely causes persistent instability[4–9]. A thin layer of loose connective tissue, known as the intertendinous fascia of Landsmeer, bridges the dorsal TFC and the SB–tendon complex. This compliant interface preserves continuity of the extensor apparatus while permitting differential glide[1–3].

Disruption of this fascial layer may contribute to extensor dysfunction even in the absence of overt tendon instability[1–3,10]. Although combined SB and dorsal TFC injury produces obvious dynamic subluxation, isolated dorsal TFC lesions may present primarily with dorsal MCP pain and subtle extensor-mechanism symptoms. Recognition of this deep-layer injury pattern underscores the importance of layered anatomical analysis and targeted imaging when evaluating dorsal hood pathology[1–3,10,11].

Ultrasound anatomy

High-frequency ultrasound allows detailed evaluation of the dorsal hood, which consists of three layers: the superficial subcutaneous layer, the intermediate extensor mechanism (including the SB), and the deep dorsal TFC[2,3,10,12] (Fig. 2).

In the short-axis plane, the SB appear as thin hyperechoic retinacular structures located superficial and deep to the extensor tendon[1–3,7,9]. Because of their oblique radial and ulnar fiber orientation, anisotropy can result in apparent hypoechogenicity; adjustment of probe angulation allows reliable differentiation of the superficial and deep SB layers[7,9,10]. The dorsal TFC is visualized deep to the extensor tendon as a homogeneous hyperechoic plate conforming to the dorsal curvature of the metacarpal head and the base of the proximal phalanx[1–3]. Proximally, it creates a dorsal sulcus that elevates and stabilizes the extensor tendon, forming a fibrocartilaginous gliding platform[1–3]. Distally, the structure tapers and assumes a crescent configuration as the dorsal sulcus flattens toward the proximal phalanx[1–3]. Dynamic examination is essential for assessing functional integrity[10,12]. Active flexion, resisted extension, clenched-fist positioning, and gentle radial–ulnar deviation facilitate evaluation of tendon alignment and help uncover subtle instability or subluxation patterns[10,12]. When the dorsal hood is intact, a consistent layered relationship is maintained, with the extensor tendon resting on the dorsal TFC over the articular cartilage throughout MCP flexion and extension[1–3].

A pattern-recognition approach assists interpretation[1–3,10,12]. On axial imaging, the normal configuration resembles a "sphinx": the

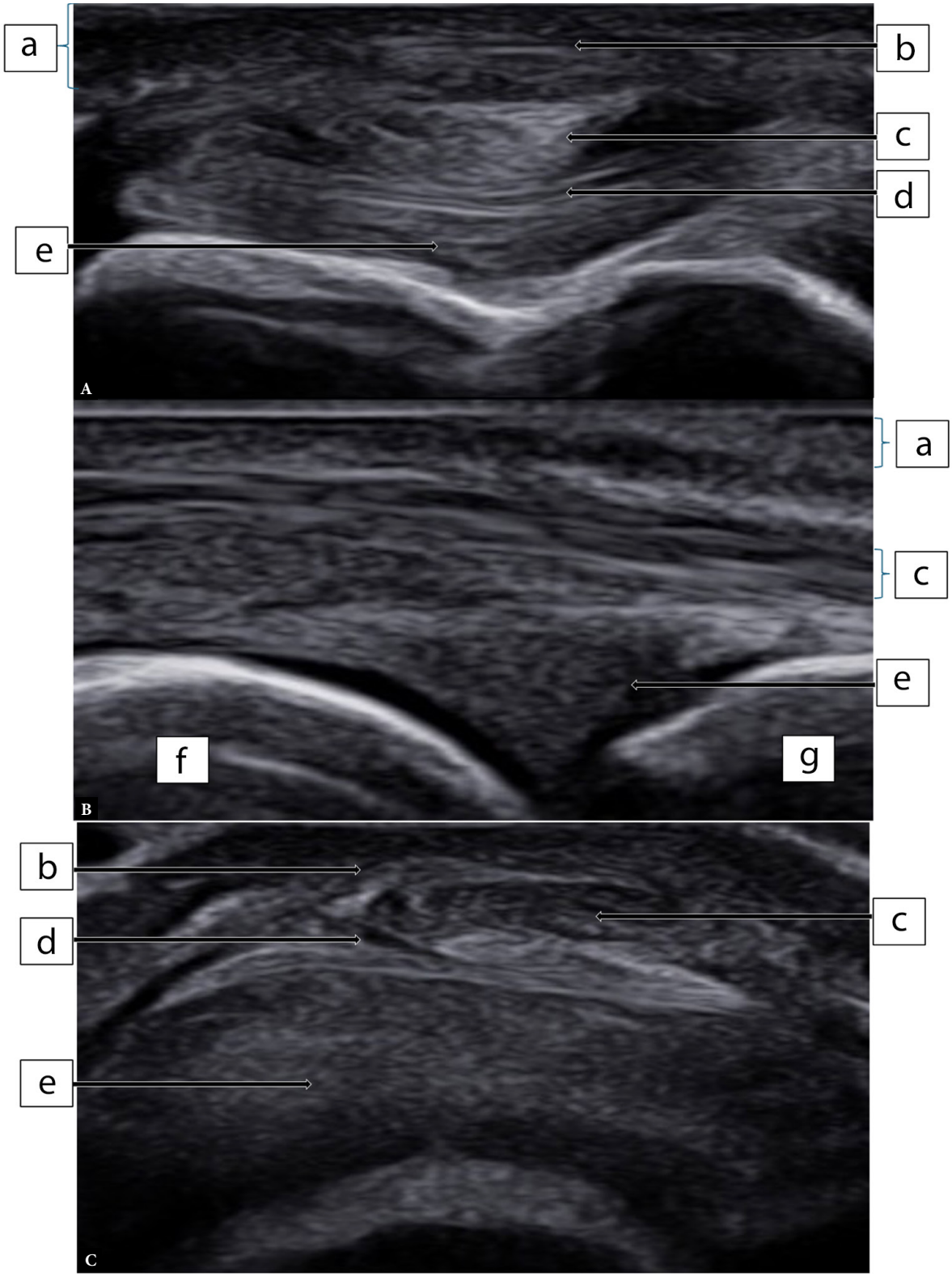


Fig. 2. *Normal dorsal hood anatomy on ultrasound.* **A.** *Axial image obtained proximally at the metacarpal head demonstrates the dorsal sulcus created by the dorsal triangular fibrocartilage (TFC) (e), elevating and stabilizing the extensor tendon (c).* **B.** *Long-axis image of the metacarpophalangeal joint shows the curved contour of the dorsal TFC (e) conforming to the dorsal surfaces of the metacarpal head (f) and the base of the proximal phalanx (g).* **C.** *Distal axial view reveals tapering and a crescent configuration of the dorsal TFC (e) as the sulcus flattens toward the base of the proximal phalanx. The layered configuration is depicted: superficial subcutaneous layer (a); intermediate extensor tendon (c) with superficial (b) and deep (d) sagittal bands forming the dynamic sling; and deep dorsal TFC (e). Bony landmarks include the metacarpal head (f) proximally and the base of the proximal phalanx (g) distally.*

extensor tendon and adjacent SB form the "head," while the dorsal TFC represents the "shoulders." This model aids differentiation of SB pathology from deep dorsal TFC defects, which may produce dorsal MCP pain and extensor-mechanism symptoms in the absence of dynamic instability[1–3,10,12].

Case reports of dorsal TFC injuries

We present a descriptive case series of three patients with (1) closed trauma to the MCP joint, (2) imaging evidence of dorsal hood abnormality with preserved SB, and (3) clinical findings of localized dorsal MCP pain and/or mechanical symptoms.

All patients underwent high-frequency dynamic ultrasound using a high-frequency linear transducer. Two cases were examined with a Canon Aplio i700 (Canon Medical Systems, Ōtawara, Tochigi, Japan) using a 24 MHz probe, and the third case was examined with a Samsung RS85 Prestige (Samsung Medison, Seoul, Republic of Korea) using a high-frequency hockey-stick probe. All cases were reviewed by highly experienced musculoskeletal radiologists, each with 30 years of experience.

Case 1

A 52-year-old right-handed man presented after a fall with trauma to the MCP joint. He reported focal pain at the dorsal aspect of the third MCP joint. Physical examination showed mild swelling and discomfort over the joint without overt instability, but with a mild extension deficit. High-resolution ultrasound demonstrated a longitudinal defect within the dorsal TFC, consistent with a fibrocartilaginous tear (Fig. 3). Herniation of the extensor tendon into the defect was seen, creating a characteristic appearance on ultrasound. The SB were intact, and dynamic assessment showed no (sub)luxation of the extensor tendon.

Case 2

A 25-year-old amateur boxer developed sudden pain over the dorsal aspect of the right third MCP joint after impact with a clenched fist during sparring. Ultrasound showed a longitudinal tear within the deep layer of the dorsal hood (Fig. 4). The superficial and intermediate layers, including both SB, were preserved.

Case 3

A 30-year-old man presented with persistent pain and weakness of the second digit following blunt trauma. Ultrasound showed intact superficial and intermediate layers, including both SB. Herniation of the extensor tendon into a longitudinal gap of the dorsal TFC was observed, without tendon luxation (Fig. 5).

Discussion

We propose evaluating dorsal hood pathology using a layer-based approach: first assessing the dorsal TFC, then characterizing SB involvement (superficial versus deep fibers), and finally integrating dynamic extensor tendon behavior[1–3,7–10,12–14]. This approach distinguishes three principal patterns: (1) intact dorsal TFC with isolated SB sprain or tear (Fig. 6); (2) isolated longitudinal dorsal TFC defect, designated as the dorsal radial burst (Fig. 7); and (3) combined dorsal TFC and SB disruption (Fig. 8).

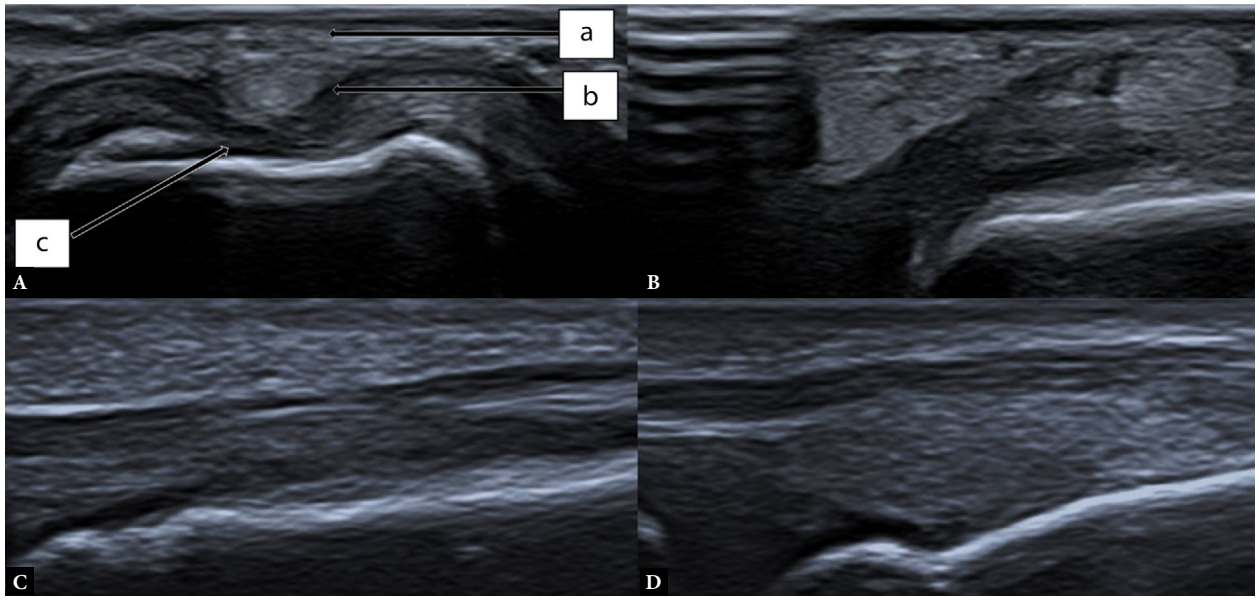


Fig. 3. *Ultrasound images of case 1.* **A.** *Pathologic axial image of the dorsal hood demonstrating volume loss of the dorsal triangular fibrocartilage (TFC) and herniation of the extensor tendon into the defect (c). The sagittal bands remain intact (a and b), producing the "sunken sphinx head" appearance.* **B.** *Normal axial image of the contralateral, non-traumatic side for comparison, demonstrating normal dorsal hood anatomy with preserved thickness of the dorsal TFC supporting the extensor tendon in its anatomic position without tendon herniation.* **C.** *Pathologic sagittal image demonstrating the extensor tendon running directly against the bone due to the absence of the underlying dorsal TFC.* **D.** *Normal sagittal image for comparison, showing the layer of dorsal TFC that separates the bone from the tendon.*

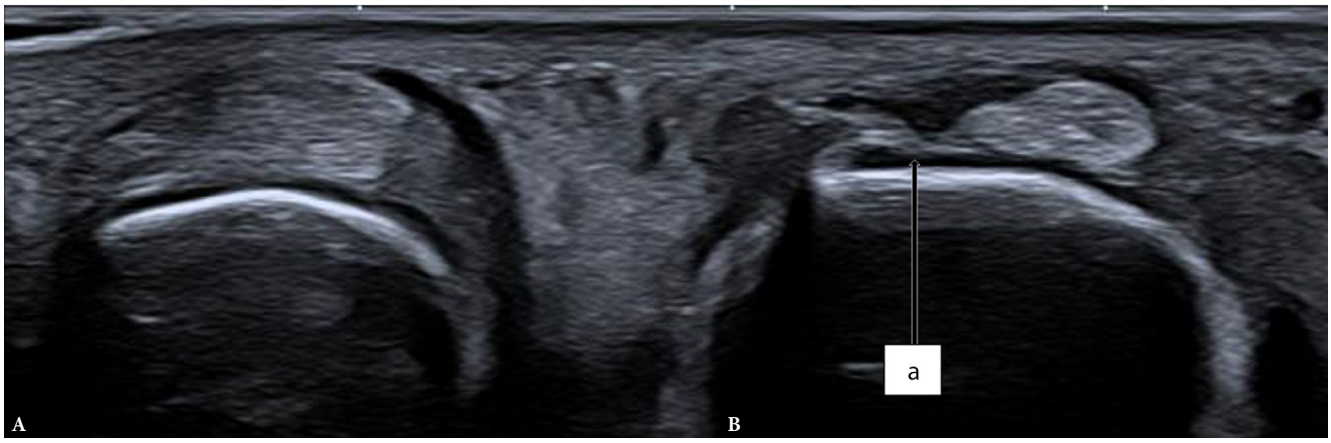


Fig. 4. *Ultrasound images of case 2.* **A.** *Axial view of the dorsal metacarpophalangeal joints of the third digit (***B***) and fourth digit (***A***).* **A.** *Intact dorsal hood is shown with normal superficial, intermediate, and deep layers (reference).* **B.** *Superficial layer is intact and the sagittal bands remain continuous, but a clear defect is present in the deep layer of the dorsal hood, with a tear in the dorsal triangular fibrocartilage (a). This produces the "sunken sphinx head" appearance. Mild contralateral eccentricity of the tendon is visible within the defect, though without frank subluxation or dislocation.*

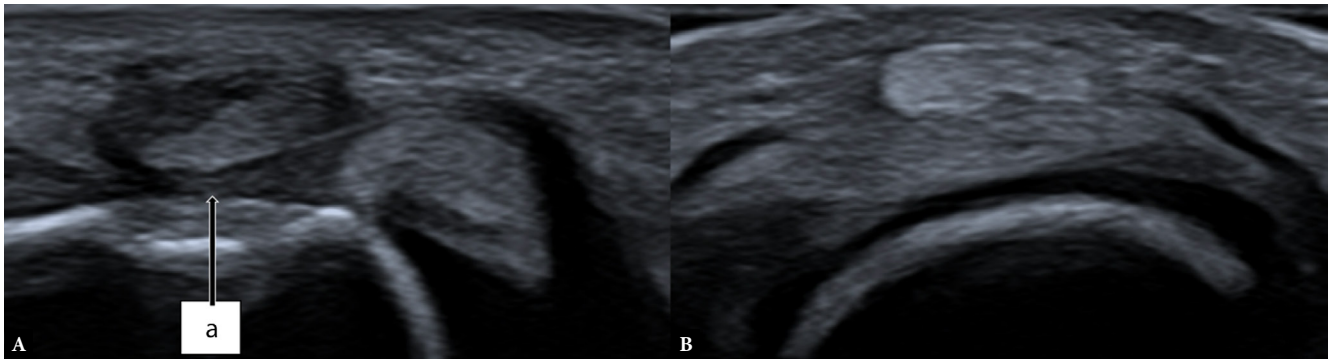


Fig. 5. *Ultrasound images of case 3.* **A.** *Axial view of the dorsal aspect of the second digit with herniation of the extrinsic extensor tendon into a longitudinal defect of the dorsal triangular fibrocartilage (TFC) (a). The extensor tendon courses along the bone, with loss of the intervening dorsal TFC layer, again creating the "sunken sphinx head" appearance.* **B.** *A normal reference image of the contralateral side.*



Fig. 6. *Schematic representation of the dorsal hood with a sagittal band lesion, associated herniation of subcutaneous tissue into the defect, and contralateral (sub)luxation of the tendon. The dorsal triangular fibrocartilage is spared.*

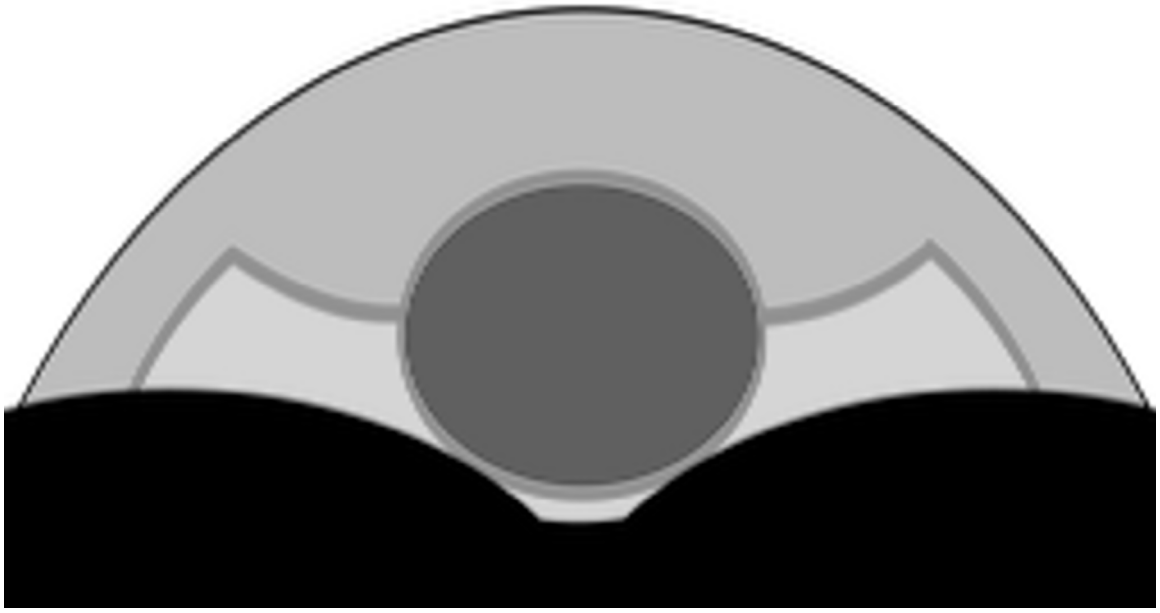


Fig. 7. *Schematic representation of the dorsal hood with a lesion confined to the dorsal triangular fibrocartilage layer (radial burst lesion). Herniation of the intact sagittal bands and extensor tendon into the defect produces the "sunken sphinx head" appearance.*

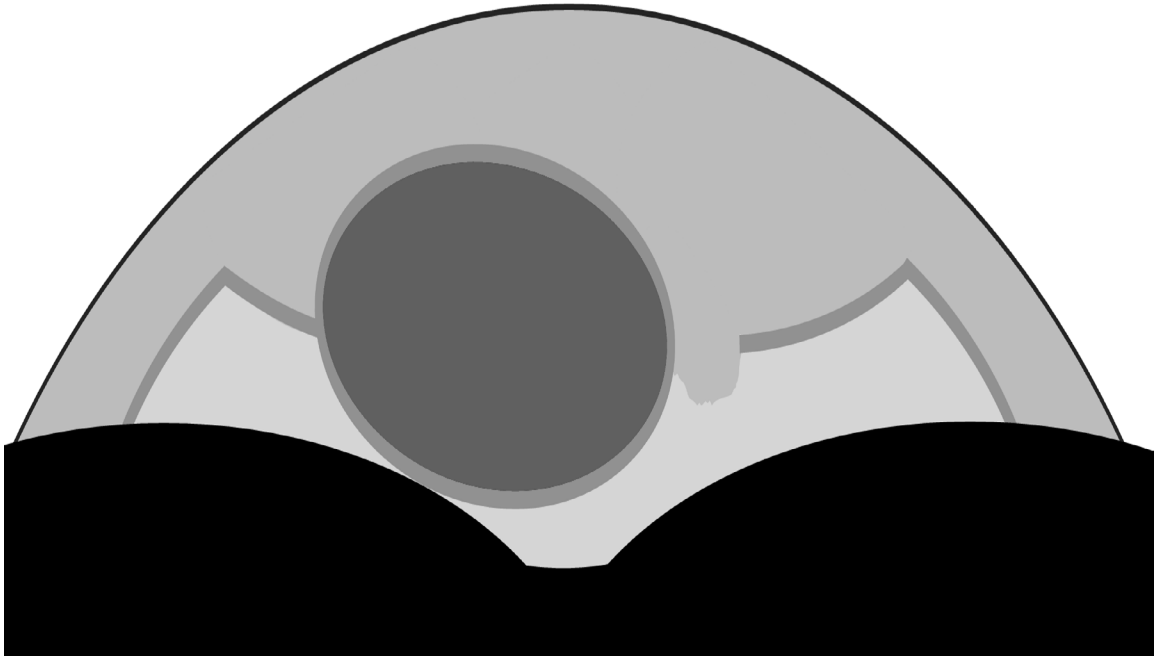


Fig. 8. *Schematic representation of the dorsal hood with combined sagittal bands (SB) and dorsal triangular fibrocartilage (TFC) disruption. The SB defect permits herniation of subcutaneous tissue and contributes to (sub)luxation of the extensor tendon, while the additional dorsal TFC rupture results in central loss of the dorsal supporting plateau, corresponding to the "sunken sphinx head" configuration.*

The term "dorsal radial burst" is proposed because the lesion resembles a radial tear of the knee meniscus: the fibrocartilaginous layer splits perpendicular to its fibers, creating a gap that interrupts structural continuity. Just as radial meniscal tears impair hoop-stress transmission and weaken joint stabilization, a dorsal radial burst disrupts the mechanical role of the dorsal TFC in distributing dorsal forces and maintaining extensor tendon alignment at the MCP joint. Dynamic extensor instability (subluxation or dislocation) primarily reflects SB failure. In contrast, isolated dorsal TFC lesions generally produce dorsal MCP pain while maintaining tendon centering during motion[1–3,10–12]. In a dorsal radial burst, the extensor tendon does not subluxate when the SB remain intact. Ultrasound typically demonstrates a linear split of the dorsal TFC with concordant focal tenderness[1–3,10,12].

A separate superficial-layer entity to recognize is adventitial bursitis, an acquired post-traumatic friction bursa superficial to the dorsal hood (Fig. 9)[10,12]. This presents with focal swelling and tenderness while preserving dynamic tendon stability and demonstrating intact SB and dorsal TFC[10,12].

Existing grading systems primarily characterize instability or gross anatomic involvement. The Rayan–Murray system describes three stability grades but does not define the depth of SB injury or the condition of the dorsal TFC[4]. Lee et al. distinguish superficial from deep SB tears, which correlates approximately with low-grade versus high-grade dynamic instability[7]. However, neither system fully addresses layered involvement nor consistently integrates dynamic behavior, both of which are increasingly recognized as critical determinants of prognosis and operative technique[8,13,14].

A layer-based approach that evaluates the subcutaneous plane, the extensor tendon with associated SB, and the dorsal TFC, combined with dynamic assessment, enables precise lesion mapping and correlates with surgical repair strategies[1–3,7–10,12–14]. Dynamic ultrasound, which best detects subtle or positional instability, can therefore be structured to first evaluate dorsal TFC integrity, then define SB-layer disruption, identify associated adventitial bursitis, and finally grade tendon tracking[1–3,7–10,12–14] (Tab. 1). Such a structured approach may help refine treatment selection and improve return-to-sport outcomes[8,13,14].

Conclusion

We describe a distinct pattern of dorsal hood injury characterized by a longitudinal dorsal TFC defect through which the extensor tendon herniates without loss of centering, designated as the dorsal radial burst lesion. This deep-layer lesion produces pain and focal tenderness while preserving SB-mediated stability, separating it conceptually and diagnostically from SB-driven extensor instability.

We further introduce the "sunken sphinx head" sign as an ultrasound marker of this lesion. On axial imaging, the extensor tendon and intact SB form the "head," while the torn dorsal TFC forms depressed "shoulders," creating a sunken variant of the classic sphinx appearance. Sagittal imaging demonstrates effacement of the dorsal TFC plane, resulting in closer contact of the tendon with the dorsal

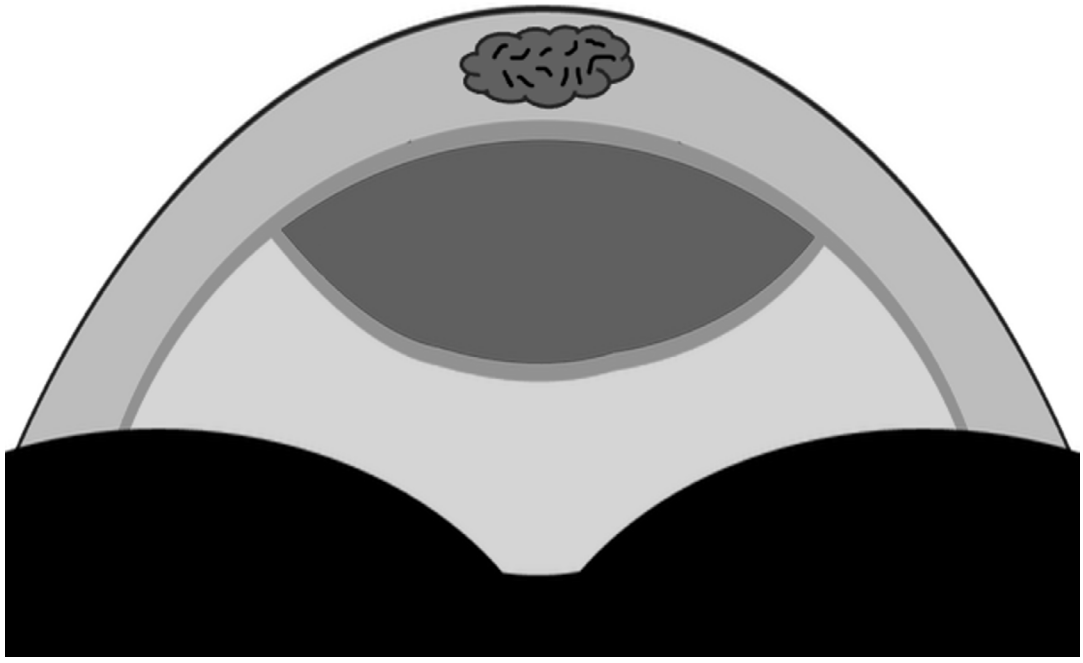


Fig. 9. *Schematic representation of the dorsal hood with a lesion confined to the subcutaneous layer (adventitial bursitis), causing focal swelling and tenderness while preserving structural continuity of the sagittal bands and dorsal triangular fibrocartilage.*

Tab. 1. Proposed classification of dorsal hood injuries by layer, lesion type, and dynamic behavior

Layer	Lesion type	Dynamic behavior and clinical presentation
Superficial	Adventitial bursitis	Normal tendon position; mild swelling
Intermediate	Radial sagittal band rupture	Possible tendon (sub)luxation (radial → ulnar)
Intermediate	Ulnar sagittal band rupture	Rare tendon subluxation (ulnar → radial)
Deep (dorsal triangular fibrocartilage)	Dorsal radial burst lesion	Stable tendon position; mechanical symptoms

bone cortex. This sign localizes the lesion to the deep dorsal TFC and distinguishes it from both SB-driven subluxation and isolated superficial adventitial bursitis.

Our findings support a layer-based ultrasound approach: first evaluate deep dorsal TFC integrity, then analyze SB continuity, and finally assess dynamic tendon tracking. Incorporating this systematic approach improves detection of deep-layer pathology, avoids misclassification as subtle instability, and provides a reproducible framework for reporting dorsal hood injuries. Recognition of the dorsal radial burst lesion and the "sunken sphinx head" sign refines sonographic analysis of the dorsal MCP joint and broadens the diagnostic spectrum of dorsal hood pathology.

Conflict of interest

The authors do not report any financial or personal connections with other persons or organizations which might negatively affect the contents of this publication and/or claim authorship rights to this publication.

Author contributions

Original concept of the study: CS, TJ. Writing of the manuscript: CS, TJ, FV. Analysis and interpretation of data: CS, TJ, KV, SM, FV. Final approval of the manuscript: CS, TJ, FV. Collection, recording and/or compilation of data: CS, TJ, KV, SM. Critical review of the manuscript: CS, TJ, PVH, FV.

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