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Open Excision and Neurolysis for Calcific Tendinitis of the Proximal Hamstring With Sciatic-Distribution Neuropathic Symptoms: A Case Report

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Abstract

Calcific tendinitis is the most common form of hydroxyapatite deposition disease and classically involves the rotator cuff, while involvement of the proximal hamstring origin is rare. We describe a 62-year-old White female with a body mass index of 19 kg/m^2 who presented with a four-year history of left posterior hip pain and paresthesias in the distribution of the sciatic nerve that persisted despite physical therapy, nonsteroidal anti-inflammatory drugs, and muscle relaxants. Radiographs and magnetic resonance imaging demonstrated an approximately 2 cm calcific deposit at the conjoint hamstring origin adjacent to the sciatic nerve, with perineural edema but without displacement or deformation of the nerve. She underwent open excision of the deposit with neurolysis of the sciatic and posterior femoral cutaneous nerves, including release of overlying fibrous tissue and of fibrous bands constricting the posterior femoral cutaneous nerve. Histopathology showed calcification within fibroconnective tissue without atypia, and cultures showed no growth. At eight months, her sciatic-distribution paresthesias had improved, and she had full strength on provocative hamstring testing, although she reported persistent subjective weakness and a discrete area of decreased sensation adjacent to the incision. Calcific tendinitis of the proximal hamstring should be considered in patients with posterior hip pain and sciatic-distribution neuropathic symptoms, and surgical excision with neurolysis may be considered when conservative treatment fails.

Categories: Orthopedics
Keywords: calcific tendinitis, hamstring tendinopathy, hydroxyapatite deposition disease, proximal hamstring, sciatic nerve compression

Introduction

Hydroxyapatite crystal deposition disease (HADD) refers to the deposition of hydroxyapatite crystals in and around the joint space [1,2]. Calcific tendinitis, characterized by calcium crystal deposition within tendons, is the most prevalent form of HADD, affecting approximately 3% of adults [1,3]. The shoulder, specifically the supraspinatus tendon, is most frequently involved, followed by the longus colli, Achilles tendon, and proximal rectus femoris [3]. Calcific tendinitis is thought to progress through precalcific, calcific, and postcalcific stages, described further in the discussion, with the resorptive phase of the calcific stage accounting for the acute pain flares characteristic of the condition. Involvement of the proximal hamstring origin at the ischial tuberosity is rare and has been described only sporadically, but it should be considered in patients presenting with posterior hip pain [3].

The conjoint tendon of the semitendinosus and long head of the biceps femoris and the semimembranosus tendon originate from the ischial tuberosity, and the sciatic nerve courses immediately lateral to this origin, accompanied by the posterior femoral cutaneous nerve [4]. Due to their anatomic proximity, a mass or inflammatory process at the hamstring origin may irritate the adjacent nerves and produce neuropathic symptoms in addition to signs of tendinopathy. While this is a recognized complication of proximal hamstring tendinopathy (PHT), it is more commonly associated with tendinosis and fibrosis than with calcific deposition [4]. Calcific tendinitis is distinguished from PHT with tendinosis by discrete amorphous mineralization on imaging, including radiographs, ultrasound, and magnetic resonance imaging (MRI), and by a clinical course that may include acute inflammatory flares superimposed on chronic pain [2,3].

First-line treatment typically involves conservative measures, including physical therapy and nonsteroidal anti-inflammatory drugs (NSAIDs). In refractory cases of calcific tendinitis and PHT, surgical excision of calcific deposits and release of adjacent neural structures may be warranted [3-6]. We report a case of calcific tendinitis of the proximal hamstring in a 62-year-old female with posterior hip pain and sciatic-distribution neuropathic symptoms treated with surgical excision of the calcific deposit and neurolysis of the sciatic and posterior femoral cutaneous nerves. Endoscopic excision of proximal hamstring calcific deposits encountered during repair of partial-thickness tendon tears was recently described [7]. However, to our knowledge, calcific tendinitis at this site presenting with sciatic-distribution neuropathic symptoms and

treated with open excision and neurolysis has not been reported.

Case Presentation

A 62-year-old White female with a body mass index of 19 kg/m ^{2} and a past medical history notable only for hypothyroidism presented with a four-year history of left posterior hip pain exacerbated by sitting, standing on her left leg, and navigating stairs. She described pain localized to the left ischial tuberosity with occasional paresthesias in the distribution of the sciatic nerve. Her pain had increased over the preceding two to three months despite eight weeks of formal physical therapy, NSAIDs, and muscle relaxants. Given her slim habitus, a mass was palpable over the left ischial tuberosity.

On examination, she had hamstring tightness and a straight leg raise limited to 45 degrees bilaterally. Because this finding was symmetric, it was attributed to generalized hamstring tightness rather than unilateral nerve root tension. There was tenderness to palpation over the left greater trochanter and left ischial tuberosity. Greater trochanteric tenderness was attributed to concurrent greater trochanteric bursitis. She had significant pain with resisted hamstring activation on the left but full hamstring strength. Hip flexion was 110 degrees, external rotation was 45 degrees, and internal rotation was 15 degrees. Flexion, adduction, and internal rotation (FADIR) and flexion, abduction, and external rotation (FABER) tests were negative. She had no pain with resisted hip flexion, her gait was normal, and she was neurovascularly intact in the L2 to S1 distributions.

Focal ischial tuberosity tenderness, pain with resisted hamstring activation, negative FADIR and FABER testing, normal gait, and an intact distal neurovascular examination localized her symptoms to the proximal hamstring origin and made intra-articular hip pathology and ischiofemoral impingement less likely. Lumbar radiculopathy and deep gluteal syndrome, including piriformis syndrome, were also considered. Dedicated lumbar spine imaging and electrodiagnostic studies were not obtained, as these diagnoses were considered less likely based on her presentation and clinical exam. Proximal hamstring tendinopathy with tendinosis was considered less likely given the discrete mineralized deposit on imaging, and neoplasm was addressed by tissue diagnosis.

Radiographs of the pelvis and left hip demonstrated maintained joint spaces and an approximately 2 cm focus of calcification at the hamstring origin adjacent to the ischial tuberosity (Figure *1*). MRI of the hip without contrast revealed a 1.8 cm ovoid focus of low signal intensity within the left conjoint hamstring tendon at its origin with surrounding edema, consistent with a calcific mass (Figure *2*). The deposit lay adjacent to the sciatic nerve without significant displacement or deformation of the nerve. There was high signal intensity surrounding the nerve consistent with perineural edema and inflammation, which was felt to account for the patient's neuropathic symptoms.

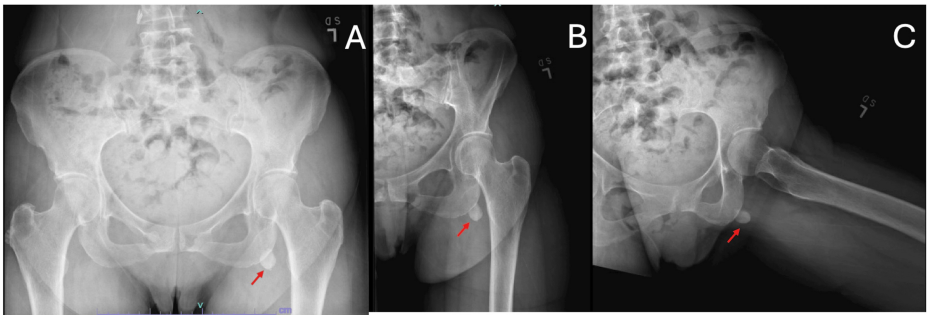


FIGURE 1: Preoperative radiographs of the pelvis and left hip.

Anteroposterior pelvis (A), anteroposterior hip (B), and frog-leg lateral (C) radiographs. Arrows indicate an approximately 2 cm ovoid calcific deposit at the left hamstring origin adjacent to the ischial tuberosity.

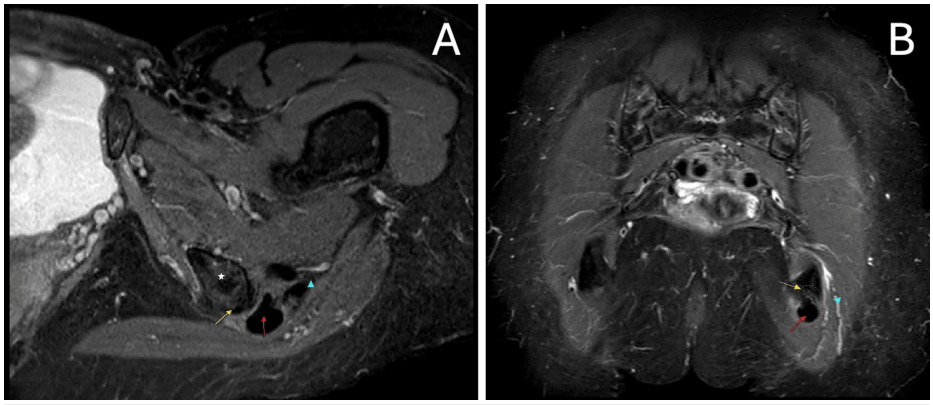


FIGURE 2: Axial (A) and coronal (B) T2 magnetic resonance imaging (MRI).

T2 MRI demonstrates an approximately 1.8 cm calcified mass (red arrow) at the origin of the left hamstring conjoint tendon (yellow arrow), adjacent to the sciatic nerve (blue arrowhead), which is surrounded by perineural edema without displacement. The left ischial tuberosity is marked with an asterisk (A).

Management options discussed with the patient included continued physical therapy, anti-inflammatory medication, image-guided corticosteroid injection, extracorporeal shockwave therapy, ultrasound-guided percutaneous lavage, and surgical excision. Risks of surgery were reviewed, including the possible need for hamstring tendon repair, recurrence of calcification, and persistent sciatic-distribution pain. Ultrasound-guided percutaneous lavage was not pursued because of the depth of the deposit beneath the gluteal soft tissue envelope and its proximity to the sciatic nerve. Corticosteroid injection and extracorporeal shockwave therapy were considered less likely to provide durable relief of a four-year, treatment-refractory deposit with associated neuropathic symptoms. In addition, the presence of a discrete calcified mass at the ischial tuberosity warranted tissue diagnosis, and the procedure was therefore planned jointly with orthopedic oncology. Given her persistent symptoms despite conservative measures, the patient elected to proceed with surgery.

Surgery was performed jointly by an orthopedic sports medicine surgeon and an orthopedic oncology surgeon. Under general anesthesia, the patient was positioned prone with a bump beneath the operative extremity to relieve tension on the hamstring and sciatic nerve. A transverse incision was made along the gluteal crease, centered over the palpable mass. Flaps were elevated superficial to the gluteal fascia, the fascia was incised, and blunt dissection at the inferior border of the gluteus maximus defined a plane of entry to the hamstring origin. The mass was identified overlying the conjoint tendon at its origin and was ballotable beneath the paratenon.

Before the mass was explored, the posterior femoral cutaneous nerve, including a small cutaneous branch, and the sciatic nerve were identified laterally and protected. Fibrous tissue overlying both nerves was dissected to free them and confirm that neither was under tension, and several fibrous bands constricting the posterior femoral cutaneous nerve were released. After hemostasis was achieved with bipolar electrocautery, the sciatic nerve was gently retracted laterally to allow safe access to the lesion.

Incision of the mass released a cottage cheese-like material, which was sent for pathology and culture. The calcific material extended into the adjacent ischiogluteal bursa, which was opened, and abundant calcified particulate matter was released and excised piecemeal with curettes and a rongeur. The aggregate specimen measured approximately 3.0 × 1.5 × 0.5 cm and included the surrounding bursal and fibroconnective tissue, which accounts for its larger dimensions relative to the mineralized focus measured on radiographs and MRI. The hamstring origin was taut and in complete continuity without tearing or degenerative change, and repair was not required. On MRI, the deposit was intratendinous, within the conjoint tendon at its origin; at surgery, it lay beneath the paratenon, with calcific material extending into the adjacent ischiogluteal bursa. Intraoperative radiographs confirmed excision of the calcific deposit, and the wound was closed in a layered fashion.

The patient recovered from anesthesia without complication and was discharged home after standard postoperative observation. Indomethacin 25 mg three times daily for two weeks, with sucralfate for gastric protection, was prescribed as prophylaxis against heterotopic ossification. As no tendon repair was performed, she was permitted to bear weight as tolerated immediately, and formal physical therapy began with gentle range of motion two weeks after surgery and progressed to gentle strengthening at three weeks, with no restrictions on range of motion.

Histopathology demonstrated fibroconnective tissue with calcification and no atypia, compatible with calcific tendinitis and without evidence of neoplasm (Figure 3). Cultures showed no growth. Crystal analysis was not performed; hydroxyapatite crystals are not birefringent and are not detected by the polarized light microscopy routinely used to identify monosodium urate and calcium pyrophosphate. The diagnosis therefore rested on the clinical presentation, preoperative imaging, operative findings, and histopathology.

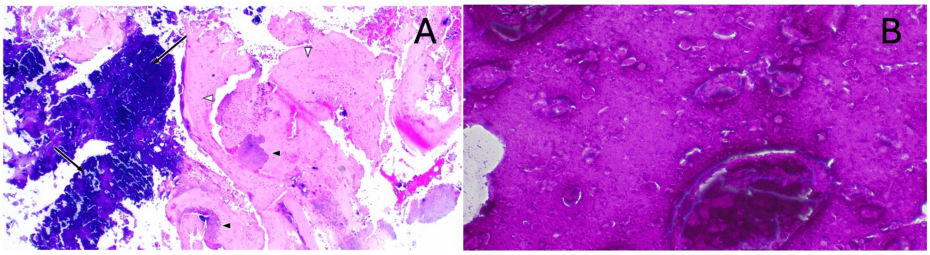


FIGURE 3: Histopathological examination of the excised specimen.

Hematoxylin and eosin stain, low power (A) and high power (B), showing fibroconnective tissue with calcification. Black arrows point to amorphous, deeply basophilic calcific deposits. White arrowheads point to the surrounding eosinophilic fibrocollagenous matrix. Black arrowheads point to small foci of bone within the matrix.

At four weeks, she had tenderness over the proximal hamstring and mild pain on provocative hamstring testing with 5/5 strength. Her preoperative sciatic-distribution neuropathic symptoms had improved. She also reported a new, discrete area of decreased sensation immediately distal to the incision that had not been present before surgery.

At eight months, her preoperative sciatic-distribution paresthesias had continued to improve. Pain at the distal aspect of the incision was improving. She was still attending physical therapy and reported subjective weakness of the left lower extremity compared with the contralateral side, despite 5/5 strength on provocative hamstring testing. The area of decreased sensation adjacent to the incision was unchanged from the four-week examination and remained distinct in distribution and character from her preoperative sciatic-distribution paresthesias.

Discussion

This case highlights an unusual presentation of calcific tendinitis involving the proximal hamstring with sciatic-distribution neuropathic symptoms. Several systemic conditions, including systemic sclerosis and chronic kidney disease, can lead to abnormal calcium deposition in soft tissues through dystrophic or metastatic calcification [8,9]. Apart from hypothyroidism, our patient had no disorders predisposing her to calcinosis. A clinical association between thyroid disorders and calcific tendinopathy of the rotator cuff has been reported, although the mechanism is undefined and its relevance to deposits outside the rotator cuff is less clear [10]. Notably, both patients in a recent report of proximal hamstring calcific tendinopathy also had thyroid disease [7]. Calcific tendinitis most frequently affects the shoulder, and recognition of atypical sites such as the proximal hamstring is important when evaluating proximal hamstring pain.

The location of the deposit at the tendon origin is consistent with calcific tendinitis, typically an intratendinous process arising from fibrocartilaginous metaplasia within the tendon substance [6]. MRI localized the deposit within the conjoint tendon at its origin, and at surgery it lay beneath the paratenon with the tendon fibers intact. Extension of calcific material into the adjacent ischiogluteal bursa does not alter this interpretation, because intratendinous deposits can rupture into neighboring bursae during the resorptive phase, as is well described for the subacromial bursa in the shoulder [2,5].

Calcific tendinitis around the hip has previously been described, but involvement of the sciatic nerve is rare. Singh and Yip described calcific tendinosis of the gluteus maximus presenting with pain radiating to the buttock, lateral hip, posterior thigh, and lateral calf, initially suspected to be a lumbar disc herniation [11]. Imaging revealed calcification inferior to the greater trochanter with edema around the sciatic nerve, and symptoms resolved with physical therapy and oral corticosteroids [11]. Chua and Shah described calcific deposits in the proximal piriformis adjacent to the sciatic nerve in a patient with sciatic-distribution tenderness and hip pain with flexion and internal rotation consistent with piriformis syndrome, which improved with corticosteroid injection and shockwave therapy [1]. Neither report demonstrated direct mass effect on the sciatic nerve; perineural edema and inflammation were considered the source of symptoms [1,11]. In the present case, MRI likewise showed perineural edema without displacement of the nerve, but at surgery, fibrous tissue overlying the sciatic and posterior femoral cutaneous nerves was released, including fibrous bands constricting the posterior femoral cutaneous nerve. Elkousy et al. recently described two women with calcific tendinopathy of the proximal hamstring origin that was not identified on MRI and was found during endoscopic repair of partial-thickness tendon tears; neither presented with sciatic-distribution

symptoms, and both improved after endoscopic debridement and suture anchor repair [7]. These cases are summarized in Table *1*. The present case differs in that the deposit was identified on preoperative imaging, the tendon was intact, she had sciatic-distribution neuropathic symptoms, and treatment consisted of open excision with neurolysis after conservative treatment failed.

Study	Patient, deposit characteristics, and presentation	Treatment	Outcome
Singh and Yip, 2015 [11]	Middle-aged male; deposit in the gluteus maximus tendon, inferior to the greater trochanter; pain radiating to the buttock, lateral hip, posterior thigh, and lateral calf, with perineural edema near the sciatic nerve	Physical therapy and oral corticosteroids	Gradual resolution; return to full activity
Chua and Shah, 2022 [1]	63-year-old female; 9 mm deposit in the proximal piriformis, adjacent to the sciatic nerve; sciatic-distribution tenderness and hip pain with flexion and internal rotation consistent with piriformis syndrome	Corticosteroid injection and extracorporeal shockwave therapy	Significant symptom improvement
Elkousy et al., 2026 [7]	54-year-old female (case 1) and 72-year-old female (case 2); deposits at the semimembranosus insertion (case 1) and conjoint tendon origin (case 2) with partial-thickness tears, size not reported and not seen on MRI; posterior hip and thigh pain with sitting and walking, without sciatic-distribution symptoms	Endoscopic debridement and suture anchor repair	Pain relief by 10 days with return of function at 3 months (case 1); VAS 74 to 0 at 12 months (case 2)
Present case	62-year-old female; 2 cm deposit at the conjoint hamstring origin at the ischial tuberosity; four-year posterior hip pain with sciatic-distribution paresthesias, perineural edema without displacement on MRI, and fibrous bands constricting the posterior femoral cutaneous nerve at surgery	Open excision with neurolysis of the sciatic and posterior femoral cutaneous nerves	Paresthesias improved and full hamstring strength at eight months; persistent subjective weakness and incisional sensory deficit

TABLE 1: Reported cases of hydroxyapatite deposition disease at the proximal hamstring origin or adjacent to the sciatic nerve around the hip.

Summary of reported cases of hydroxyapatite deposition disease involving the proximal hamstring origin or periarticular soft tissues adjacent to the sciatic nerve, including presentation, treatment, and outcomes.

VAS: visual analog scale.

A calcified mass at the hamstring origin also has a relevant differential diagnosis. Warburton et al. reported a 73-year-old woman with hip and gluteal pain and a lobulated mass at the hamstring origin with erosion of the adjacent ischial tuberosity, initially diagnosed radiographically as calcific tendinitis, which proved on biopsy to be a calcified chondroid mesenchymal neoplasm [12]. As the imaging features of these entities overlap, tissue diagnosis is valuable when a discrete calcified mass is excised at this location, and multidisciplinary management with orthopedic oncology is reasonable. Computed tomography can further characterize the mineralization pattern when radiographs and MRI are equivocal, although it added little in this case once MRI had defined the lesion [1].

The pathophysiology of calcific tendinitis remains unclear, but Uhthoff and Loehr proposed a reactive, cell-mediated process divided into precalcific, calcific, and postcalcific stages [6]. In the precalcific stage, fibrocartilaginous transformation with tenocyte metaplasia into chondrocytes occurs along with increased proteoglycan production. The calcific stage has three phases. In the formative phase, calcium crystals deposit in vesicles and coalesce into larger calcifications. During the resting phase, fibrocartilaginous tissue borders the calcification without inflammation. The resorptive phase involves infiltration of mesenchymal cells, leukocytes, lymphocytes, and giant cells that form a calcium granuloma surrounded by capillaries and vascular channels; removal of calcium in this phase is associated with acute pain, and the deposits have a toothpaste-like consistency. In the postcalcific stage, new collagen forms along with granulation tissue, increased fibroblast activity, and capillary formation [6]. The cottage cheese-like material and the escalation of symptoms in the months before surgery suggest that our patient presented in the resorptive phase, and such an acute-on-chronic pain pattern may help distinguish calcific deposition from tendinosis.

Conservative management, including physical therapy and NSAIDs, is the first-line treatment for calcific tendinitis [2,3,13]. Extracorporeal shockwave therapy and ultrasound-guided percutaneous lavage are minimally invasive options for refractory cases. Shockwave therapy delivers high-energy acoustic waves to the affected tendon to fragment the calcified material, whereas lavage uses one or two needles to irrigate,

fragment, and aspirate the deposit [13]. Surgery is indicated when conservative management fails, or symptoms progress [2,3,6,13], and may be performed through open or arthroscopic techniques to remove the deposit and repair the tendon if needed, followed by lavage to clear residual calcium [13]. This algorithm derives largely from experience with the rotator cuff, and the depth of an ischial deposit limits the practicality of percutaneous lavage at this site. Shockwave therapy was considered unlikely to provide durable relief after four years of refractory symptoms, and when a discrete calcified mass is present, the need for tissue diagnosis is itself an indication for open excision [12]. Elkousy et al. favor an endoscopic approach at this site, in part because it may lower the risk of posterior femoral cutaneous nerve injury [7]; an open approach was chosen here to allow neurolysis of the sciatic and posterior femoral cutaneous nerves and full exposure of a deposit adjacent to the sciatic nerve for tissue diagnosis.

Following excision, indomethacin was given empirically for its anti-inflammatory effect and to reduce the risk of heterotopic ossification after dissection at the ischial tuberosity, extrapolating from its use after hip surgery. Heterotopic ossification is a process distinct from recurrent hydroxyapatite deposition, and no controlled evidence supports this practice after excision of calcific tendinitis. Although her preoperative sciatic-distribution paresthesias improved, the persistent area of decreased sensation distal to the incision is most consistent with injury to a cutaneous branch of the posterior femoral cutaneous nerve during its dissection and release, a recognized risk of posterior approaches to the ischial tuberosity. Her subjective weakness despite full strength on examination likewise indicates that her symptoms improved but did not fully resolve, and patients considering this procedure should be counseled accordingly.

This report is limited by its retrospective, single-case design, which precluded validated pain and functional outcome measures; by the absence of documented reflex testing, lumbar spine imaging, electrodiagnostic studies, and crystallographic confirmation of hydroxyapatite; and by eight months of follow-up, which leaves the long-term risk of recurrence at this site unknown.

Conclusions

Calcific tendinitis of the proximal hamstring should be considered in patients presenting with posterior hip pain and sciatic-distribution neuropathic symptoms. When proximal hamstring pain does not improve with conservative management, radiographs and MRI can identify calcific deposits and define their relationship to the sciatic nerve; computed tomography adds little once MRI has established the diagnosis but may be useful when MRI is unavailable or contraindicated. In this patient, surgical excision of the deposit with neurolysis of the sciatic and posterior femoral cutaneous nerves improved her preoperative neuropathic symptoms, with residual subjective weakness and a persistent incisional sensory deficit at eight months. Whether this approach generalizes to other patients cannot be established from a single case, but awareness of calcific tendinitis at uncommon sites can support timely diagnosis and targeted treatment.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Acquisition, analysis, or interpretation of data: Olivia R. Perrotta, Ariel Aminov, Ashley E. Rosenberg

Drafting of the manuscript: Olivia R. Perrotta, Ariel Aminov

Critical review of the manuscript for important intellectual content: Ashley E. Rosenberg, Rachel Talley-Bruns

Concept and design: Rachel Talley-Bruns

Supervision: Rachel Talley-Bruns

Disclosures

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