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Giant Calcific Insertional Achilles Tendinopathy Associated With Diabetes Mellitus: A Clinical and Surgical Case Report

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Case Report

Keywords: Calcific Insertional Achilles Tendinopathy, Calcific Insertional Tendinopathy, Diabetes Mellitus, Calcification, Achilles Tendon, Metabolic Diseases

Posted Date: November 3rd, 2026

DOI: https://doi.org/10.21203/rs.3.rs-10996187/v1

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Additional Declarations: No competing interests reported.

Abstract

INTRODUCTION: Calcific Insertional Achilles Tendinopathy (CIAT) is a rare tendon pathology characterized by calcific deposition at the attachment site of the Achilles tendon to the calcaneus (enthesis). Diabetes Mellitus (DM), one of the common metabolic diseases, adversely affects tendon structure and biomechanics through various pathways, such as increased oxidative stress and the accumulation of advanced glycation end products.

CASE: We report the case of a 47-year-old male patient with DM, treated with a DPP-4 inhibitor and metformin, who presented with a long-standing history of atraumatic heel pain and swelling. Clinical examination revealed that the pain and swelling were localized to the calcaneal enthesis of the Achilles tendon and increased with passive dorsiflexion. Radiological imaging demonstrated giant calcified mass measuring approximately 5 × 3 × 1.5 cm at the calcaneal enthesis of the Achilles tendon. During surgical intervention, the distal one-third of the Achilles tendon was incised using a posterior midline longitudinal approach, followed by excision of the calcified tissue. As approximately 75% of the Achilles tendon insertion was observed to be preserved, the preoperatively planned V-Y lengthening technique was abandoned. Repair was performed by re-fixating the tendon tissue, which had been incised and elevated from the calcaneus, to the calcaneus using a knotted metallic anchor. Histopathological examination revealed osteochondromatous and tendinoligamentous tissues; no findings suggestive of malignancy were detected. At a 7-month postoperative follow-up, the patient showed significant clinical improvement in pain, function, and range of motion.

CONCLUSION: This case highlights a clinically rare giant-sized CIAT and its possible association with the adverse effects of DM on tendon biomechanics. Even in the presence of giant CIAT, in cases where the insertion is largely preserved, anchor-supported partial repair may be a feasible surgical option instead of extensive reconstruction.

INTRODUCTION

Achilles tendinopathy (AT) is a common and multifactorial degenerative condition affecting the Achilles tendon [1]. Degenerative changes at the calcaneal enthesis of the Achilles tendon may progress to an advanced stage, leading to fibrocartilaginous transformation and pathological calcification [2].

Calcific Insertional Achilles Tendinopathy (CIAT), first described by Clain and Baxter in 1992, is a specific form of insertion tendinopathy characterized by pathological calcium deposition at the calcaneal enthesis of the Achilles tendon [3]. Although CIAT traditionally considered a pathology related to excessive tendon loading, current studies indicate that metabolic factors also play a critical role in disease pathogenesis [1, 4].

Diabetes mellitus (DM) is a common metabolic disease known to induce degenerative changes in tendon tissue [5]. The accumulation of advanced glycation end products (AGEs) secondary to hyperglycemia in DM leads to abnormal cross-linking in collagen fibers and disruption of extracellular matrix (ECM) organization [6]. These changes reduce tendon elasticity and suppress regenerative capacity. In addition, the resulting tenocyte dysfunction and impaired healing responses may disrupt tendon homeostasis and predispose the tendon to degeneration and calcification [7]. In the literature, CIAT has been reported to be significantly more frequent in the presence of DM, that most follow an asymptomatic course, and that their frequency increases with age [8].

Conservative treatment remains the initial therapeutic paradigm and includes activity modification, orthotic applications, and physical therapy. In cases that do not respond to conservative treatment, surgical excision of calcified/ossified tissues is the most common treatment approach [9, 2].

Although evidence regarding the relationship between DM and CIAT is increasing, the literature reporting giant-sized CIAT cases associated with DM, as in our case, and addressing their surgical management remains quite limited. In this case report, we aimed to contribute to the existing literature regarding the possible metabolic basis and surgical management of this rare condition by presenting the clinical, radiological, and surgical features of giant-sized CIAT associated with DM.

CASE PRESENTATION

A 47-year-old male patient presented with progressive pain and swelling, present for approximately 3 months localized to the insertional region of the left Achilles tendon, increasing with walking and stair climbing, and developing without a history of trauma. His medical history revealed that he had been diagnosed with DM 4 years earlier and had been receiving antidiabetic treatment (DPP-4 inhibitor and metformin) for 4 years accordingly. In addition, persistently elevated HbA1c levels had been documented over the preceding two years, indicating poor glycemic control.

On physical examination, skin integrity over the left leg and ankle was preserved, and no erythema or ulceration was observed. On palpation, marked tenderness and a palpable hard mass were detected in the insertional region of the left Achilles tendon. Local skin temperature was normal. On evaluation of ankle range of motion, passive dorsiflexion was measured as 0° and plantar flexion as 40°, whereas active range of motion was limited to 0° of dorsiflexion and 30° of plantar flexion. The passive stretch test caused an increase in pain, whereas the Thompson test was evaluated as negative. Distal neurovascular examination was normal.

Diagnostic imaging included radiographs, computed tomography (CT) and magnetic resonance imaging (MRI). Marked thickening of the Achilles tendon together with peritendinous edema was observed. In addition, a solitary calcified mass measuring approximately 5 × 3 × 1.5 cm, accompanied by edema, was detected in the insertional region of the Achilles tendon. The findings were considered consistent with intratendinous calcification localized to the insertional region of the Achilles tendon (Fig-1).

In the preoperative functional assessment, the AOFAS (American Orthopaedic Foot and Ankle Society) score was 26, the Visual Analog Scale (VAS) score was 8, and the VISA-A score was 29. Ankle range of motion was severely restricted to 0°–30°.

Surgical treatment was planned due to the size of the calcific lesion, the lack of response to conservative treatment, and the long-standing persistence of symptoms. Preoperative evaluation suggested that the patient should undergo V-Y Achilles lengthening due to the possibility of a tendon defect occurring after excision of calcified tissue. The operation was performed after preparation of the sterile surgical field, under block anesthesia, with the patient in the prone position.

A posterior midline longitudinal incision was made to include the calcaneal insertion of the Achilles tendon, and the tendon was exposed. Subsequently, the distal one-third of the tendon was longitudinally incised. During exploration, calcified osseous tissue was identified in the calcaneal insertional region of the Achilles tendon (Fig-2). The pathological tissue was excised using an osteotome under C-arm fluoroscopic guidance and was sent for histopathological examination.

Intraoperative evaluation showed that approximately 75% of the Achilles tendon insertion was preserved. Therefore, the preoperatively planned V-Y lengthening technique was not required. Using a knotted metallic

anchor, the intact portion of the tendon was re-fixated to the calcaneus, and partial repair was performed (Fig-3). After hemostasis was achieved, the surgical field was anatomically closed and wrapped with an elastic bandage. In the postoperative period, the ankle was immobilized at approximately 20° of plantar flexion, and a gradual weight-bearing protocol was implemented. Controlled range-of-motion exercises were initiated during the rehabilitation process.

Histopathological examination of the excised calcified lesion demonstrated osteochondromatous and tendinoligamentous tissues; no findings suggestive of malignancy were detected.

The patient showed significant improvement after the operation. The patient continues to show significant clinical improvement at 7 months postoperatively: the AOFAS score increased from 26 to 83, the VAS score decreased from 8 to 2, and the VISA-A score increased from 29 to 92. A significant improvement in ankle range of motion was achieved.

DISCUSSION

The presented case makes two important contributions to the CIAT literature. First, it draws attention to the strong relationship between DM, one of the common metabolic diseases, and tendinopathies, and presents a giant-sized CIAT case that became symptomatic at a late stage. Secondly, it demonstrates an important surgical approach to the management of this rare case with a partial repair approach that prioritizes tendon preservation, as the preoperatively planned V-Y lengthening technique was not deemed necessary during intraoperative evaluation.

CIAT is a multifactorial form of insertional tendinopathy originating from the tendon–bone junction, in which intrinsic and extrinsic factors act together [10, 11]. It has been reported that patients with type 2 DM have a fourfold higher risk of developing tendinopathy and up to a fivefold higher risk of tendon tear or rupture compared with non-diabetic individuals [12].

In addition, it is known that the frequency of CIAT increases with age and is significantly higher in patients with DM [8].

DM has been associated with impaired tissue function, increased susceptibility to tendon injuries, and decreased healing capacity. It has been suggested that this pathology arises as a result of the adverse effect on the relationship among ECM proteins, collagen fibers, and tenocytes due to the increase in advanced glycation end products (AGEs) under hyperglycemic conditions [13, 14]. However, although the epidemiological relationship between DM and CIAT has been supported, the pathophysiology between the two conditions is still not fully understood [15, 8].

Considering the progressive course of CIAT, it has been reported that early stages may follow an asymptomatic course or present with low symptom severity, whereas clinical manifestations often become apparent during advanced degenerative processes [16, 17, 3]. In our case, despite the presence of a giant calcified lesion in a patient with DM, symptoms became apparent only in the last few months, suggesting that the pathological process may have remained minimally symptomatic for a prolonged period. However, it should be noted that no definitive conclusion regarding the temporal progression of the lesion can be drawn from a single case.

Patients with CIAT present to the clinic with complaints of progressive pain, swelling, and tenderness, especially in the insertion region of the tendon. For physical examination, tests such as passive stretching tests that provoke

pain can be performed [18].

Lateral and axial radiographic evaluations are generally the first and appropriate imaging techniques for detecting Achilles insertion tendinopathies. MRI can provide prognostic information that can guide the management of CIAT at different stages [19]. Tendon parenchymal changes consistent with tendinopathy observed on MRI have generally been associated with poor response to conservative treatment. Surgical intervention is recommended in patients with severe tendon degeneration confirmed by MRI, failed conservative treatment, or advanced tendinopathy [20].

Although plain radiographs are effective in demonstrating calcific/ossified lesions in the insertional region, CT and MRI provide more detailed information regarding the size and morphology of the lesion, the degree of tendon degeneration, and its relationship with surrounding soft tissues [9, 18]. In particular, preoperative MRI findings indicating tendon degeneration may be associated with poor response to conservative treatment and may provide guidance in surgical planning [19, 20]. In the present case, the giant calcified mass in the insertional region of the Achilles tendon was clearly demonstrated by CT and MRI, and these findings played a decisive role in planning surgical treatment.

Lateral radiographs demonstrated a solitary calcified lesion measuring approximately 5 × 3 × 1.5 cm at the calcaneal insertion of the left Achilles tendon. CT revealed marked thickening of the Achilles tendon, a hyperdense lesion consistent with calcification, and intratendinous edema, without associated fracture or abnormal joint alignment. MRI demonstrated signal alterations and surrounding soft tissue edema at the level of the calcified lesion, while calcaneal bone signals were preserved.

The principal aims of surgical treatment are debridement of calcific tissue, excision of insertional osteophytes and bony prominences, and restoration of tendon–bone continuity. Although several surgical techniques have been described for CIAT, no clear superiority of one technique over another has been established in the literature [21]. The posterior midline longitudinal approach is frequently preferred because it provides direct access to centrally located lesions, adequate exposure, controlled debridement, and safe protection of the sural nerve [22–24]. V-Y lengthening is a reconstructive tendon lengthening technique commonly used to bridge the tendon gap created in chronic Achilles tendon ruptures or shortened tendons, thereby restoring tendon continuity [25]. In the present case, the posterior midline longitudinal approach was preferred, and V-Y lengthening was planned preoperatively as a reconstructive option.

Surgery was performed under femoral and popliteal block anesthesia with the patient in the prone position. An 8-cm posterior midline longitudinal incision was made to expose the distal insertional region of the Achilles tendon. Intraoperative evaluation demonstrated that the calcified mass could be excised while preserving tendon fibers and that approximately 75% of the tendon insertion remained intact. Therefore, the preoperatively planned V-Y lengthening procedure was abandoned in favor of a tendon-preserving primary repair strategy. The calcified mass was excised using an osteotome and mallet while preserving the tendon fibers, and complete excision was confirmed fluoroscopically. Small superficial tendon tears were repaired using non-absorbable sutures. The remaining intact tendon tissue was reinserted anatomically to the calcaneus using a single anchor screw. Excised intratendinous calcified-ossified tissue samples were sent for histopathological examination.

Histopathological examination of the calcified lesion revealed osteochondromatous and tendinoligamentous tissues, with no evidence of malignancy. These findings were consistent with calcific changes developing on the

basis of a chronic degenerative process, and the possible contribution of DM to this process may be considered in light of the current literature [26, 27].

Although DM has frequently been associated with Achilles tendinopathy in the literature, data regarding its relationship with CIAT, the calcific insertional form, and its surgical management remain very limited. The rarity of DM-associated CIAT cases in the literature suggests that clinical awareness should be increased in diabetic patients presenting with symptoms of tendon pathology. These findings suggest that early diagnosis and adequate metabolic control in diabetic patients may limit the development and progression of CIAT and thereby reduce the need for aggressive treatment. Although the pathophysiology between DM and CIAT has not been fully elucidated, epidemiological studies clearly demonstrate a correlation between the two conditions. Further research investigating the relationship between these two conditions is needed to elucidate the specific pathways and interactions that contribute to CIAT development in DM.

CONCLUSION

CIAT is a rare but clinically important form of tendinopathy that requires careful consideration in terms of diagnosis and treatment, particularly in patients with DM. This case demonstrates the successful clinical and surgical management of a rare giant-sized CIAT associated with DM in the absence of a history of trauma. Although an epidemiological association between DM and CIAT has been reported, the underlying pathophysiological mechanisms remain incompletely understood. A better understanding of diabetes-related tendon pathology may improve clinical awareness, facilitate earlier diagnosis, and help reduce the need for more aggressive surgical interventions in diabetic patients.

Abbreviations

CT
Computerized tomography
DM
Diabetes Mellitus
MRI
Magnetic resonance imaging
CIAT
Calcific insertional Achilles tendinopathy
AOFAS
American orthopaedic foot and ankle society
VAS
Visual analog scale
VISA-A
Victorian institute of sport assessment-achilles

Declarations

Acknowledgements Not applicable.

Authors' contributions Conception and design of the study: M H B. Acquisition and analysis of data: M H B, M E K Interpretation of data: M H B, I U. Manuscript writing: M H B; M E K assisted in drafting parts of the manuscript. Critical revision of the manuscript for important intellectual content: M E K, I U. M H B contributed to the literature review and collation of supplementary materials.

Funding No funding was obtained for this study.

Data availability No datasets were generated or analysed during the current study.

Ethics approval and consent to participate

The study was performed in accordance with the Declaration of Helsinki.

Consent for publication

Written informed consent for the publication of this case report and accompanying images was obtained from the patient.

Clinical trial registration
Not applicable.

Competing interests

The authors declare no competing interests.

References

1. Traweger A, Scott A, Kjaer M, Wezenbeek E, Scattone Silva R, Kennedy JG, et al. Achilles tendinopathy. Nat Rev Dis Primers. 2025;11:20.
2. Su L, Arshi A, Beck JJ. Extensive atraumatic heterotopic ossification of the Achilles tendon in an adolescent with metabolic syndrome: A case report. JBJS Case Connect. 2020;10(3):e0394.
3. Uhthoff HK, Loehr JW. Calcific tendinopathy of the rotator cuff: pathogenesis, diagnosis, and management. J Am Acad Orthop Surg. 1997;5(4):183–91.
4. Ackermann PW, Hart DA. General overview and summary of concepts regarding tendon disease topics addressed related to metabolic disorders. Adv Exp Med Biol. 2016;920:293–8.
5. Giha HA, Sater MS, Alamin OAO. Diabetes mellitus tendino-myopathy: epidemiology, clinical features, diagnosis and management of an overlooked diabetic complication. Acta Diabetol. 2022;59(7):871–83.
6. Lee JM, Veres SP. Advanced glycation end-product cross-linking inhibits biomechanical plasticity and characteristic failure morphology of native tendon. J Appl Physiol (1985). 2019;126(4):832–41. 10.1152/japplphysiol.00430.2018.
7. Vaidya R, Lake SP, Zellers JA. Effect of diabetes on tendon structure and function: not limited to collagen crosslinking. J Diabetes Sci Technol. 2023;17(1):89–98.
8. Giai Via A, Oliva F, Padulo J, Oliva G, Maffulli N. Insertional calcific tendinopathy of the Achilles tendon and dysmetabolic diseases: an epidemiological survey. Clin J Sport Med. 2022;32(1):e68–73.
9. Wiegerinck JI, Kerkhoffs GM, van Sterkenburg MN, Sierevelt IN, van Dijk CN. Treatment for insertional Achilles tendinopathy: a systematic review. Knee Surg Sports Traumatol Arthrosc. 2013;21(6):1345–55.

10. Giai Via A, Papa G, Oliva F, et al. Tendinopathy Curr Phys Med Rehabil Rep. 2016;4:50–5.
11. Waldecker U, Hofmann G, Drewitz S. Epidemiologic investigation of 1394 feet: coincidence of hindfoot malalignment and Achilles tendon disorders. Foot Ankle Surg. 2012;18:119–23.
12. Nichols AEC, Oh I, Loiselle AE. The Effects of Type II Diabetes Mellitus on Tendon Homeostasis and Healing. J Orthop Res. 2020;38(1):13–22.
13. Snedeker JG, Gautieri A. The role of collagen crosslinks in aging and diabetes: the good, the bad and the ugly. Muscles Ligaments Tendons J. 2014;4:303–8.
14. Xu J, Wang J, Ji Y, et al. The impact of diabetes mellitus on tendon pathology: a review. Front Pharmacol. 2024;15:1491633.
15. Xu K, Zhang L, Ren Z, Wang T, Zhang Y, Zhao X, et al. Evaluating the role of type 2 diabetes mellitus in rotator cuff tendinopathy: development and analysis of a novel rat model. Front Endocrinol. 2022;13:1042878.
16. Kaux JF, Forthomme B, Goff CL, Crielaard JM, Croisier JL. Current opinions on tendinopathy. J Sports Sci Med. 2011;10(2):238–53.
17. Paavola M, Kannus P, Järvinen TAH, Khan K, Józsa L, Järvinen M. Achilles tendinopathy. J Bone Joint Surg Am. 2002;84(11):2062–76.
18. Martin RL, Chimenti R, Cuddeford T, Houck J, Matheson JW, McDonough CM, et al. Achilles pain, stiffness, and muscle power deficits: midportion Achilles tendinopathy revision 2018. J Orthop Sports Phys Ther. 2018;48(5):A1–38.
19. Khan KM, Forster BB, Robinson J, Cheong Y, Louis L, Maclean L, et al. Are ultrasound and magnetic resonance imaging of value in assessment of Achilles tendon disorders? A two year prospective study. Br J Sports Med. 2003;37(2):149–53.
20. Nicholson CW, Berlet GC, Lee TH. Prediction of the success of nonoperative treatment of insertional Achilles tendinosis based on MRI. Foot Ankle Int. 2007;28(4):472–7.
21. Lugani G, Santandrea A, Mercurio D, Puddu L, Silvestri J, Cortese F. Treatment of Achilles insertional tendinopathy: our surgical procedure and medium-term results. Acta Biomed. 2023;94(2):e2023053.
22. Gaston TE, Daniel JN. Achilles insertional tendinopathy: is there a gold standard? Arch Bone Jt Surg. 2021;9(1):5–8.
23. Johnson KW, Zalavras C, Thordarson DB. Surgical management of insertional calcific Achilles tendinosis with a central tendon splitting approach. Foot Ankle Int. 2006;27(4):245–50.
24. McGarvey WC, Palumbo RC, Baxter DE, Leibman BD. Insertional Achilles tendinosis: surgical treatment through a central tendon splitting approach. Foot Ankle Int. 2002;23(1):19–25.
25. Kacprzak B, Kisielewska A, Miler A, Hagner-Derengowska M, Kitschke E, Zukow W. Evaluation of the effectiveness of V-Y plasty of frozen Achilles tendon rupture, with immediate follow-up weight-bearing and rehabilitation: case analysis. J Educ Health Sport. 2024;60:51779.
26. Batista F, Nery C, Pinzur M, Monteiro AC, de Souza EF, Felippe FHZ, et al. Achilles tendinopathy in diabetes mellitus. Foot Ankle Int. 2008;29:498–501.
27. Li Y, Fessel G, Georgiadis M, Snedeker JG. Advanced glycation end-products diminish tendon collagen fiber sliding. Matrix Biol. 2013;32:169–77.

Figures



Figure 1

Preoperative Imagıng.

A) CT scan demonstrating calcific deposits within the Achilles tendon

B) T2-weighted MRI

C) Lateral X-ray, demonstrating the calsific lesion's length and thickness

D) CT scan, demonstrating the calsific lesion's width

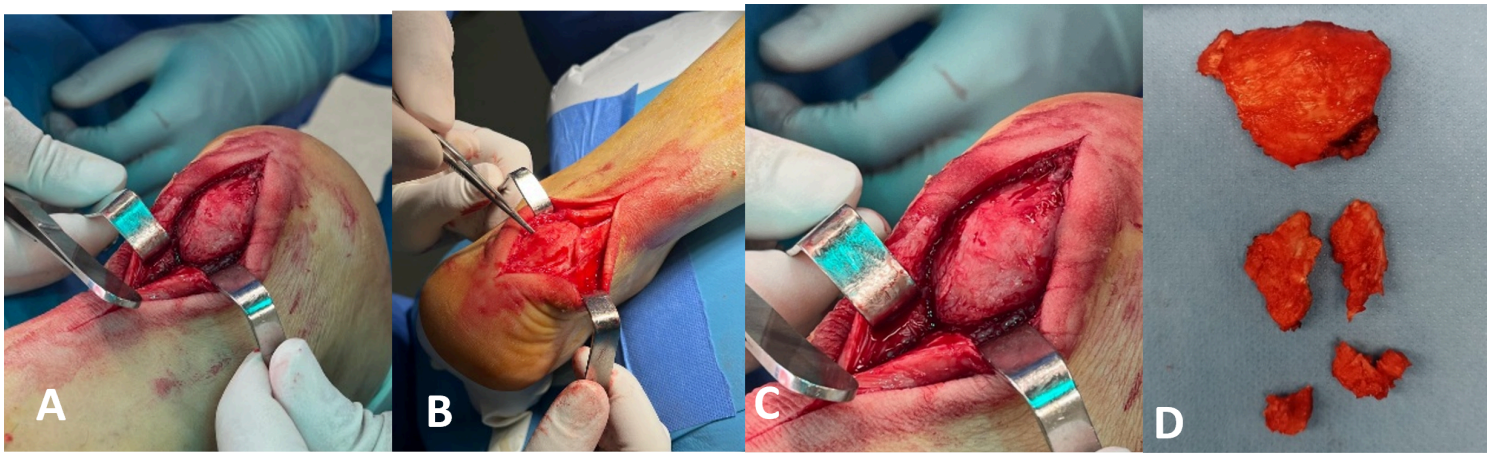


Figure 2

INTRAOPERATIVE IMAGING

A) Exposure of the giant calcific lesion through a posteromedial incision B) Insizion of Achilles Tendon C) Exposure of the giant calcific lesion, different angle D) Resected calcific lesion at the Achilles tendon insertion site



Figure 3

POSTOPERATIVE IMAGING

A) The Achilles tendon was reattached using suture anchors, No calcific lesion remained

Supplementary Files

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