

# Charcot Knee in Disguise: An Overlooked Neuroarthropathy in an Elderly Patient with Sepsis and Neurologic Decline – A Case Report

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## Abstract

**Background:** Charcot neuroarthropathy is a progressive destructive joint disorder resulting from loss of protective sensation, most frequently associated with diabetes mellitus but rarely affecting the knee. Diagnosis is often delayed because Charcot knee can mimic septic arthritis, particularly in critically ill patients with elevated inflammatory markers. Advanced cases may present with severe instability, pseudoarthrosis, or joint collapse, complicating management.

**Case Report:** An 83-year-old man with hydrocephalus, encephalopathy and Type 2 diabetes mellitus was admitted to the intensive care unit with sepsis. During physiotherapy, he was found to have a painful, immobile and swollen right knee, raising concern for septic arthritis. Aspiration yielded blood-tinged fluid with negative Gram stain and cultures. Radiographs and computed tomography demonstrated a neglected distal femoral fracture with non-union, condylar collapse, pseudoarthrosis, patellar fragmentation, and tibial plateau erosion. Magnetic resonance imaging showed complete cruciate ligament tears, meniscal destruction and medial collateral ligament attenuation. History revealed several years of recurrent falls and 7 months of non-ambulation, consistent with neuropathic joint neglect.

**Results:** Given extensive joint destruction, knee arthrodesis with an Ilizarov fixator was considered the only viable salvage procedure; however, severe comorbidities and high anesthetic risk led to family refusal. The patient was managed conservatively with bracing, analgesia, physiotherapy and pneumatic compression, stabilizing systemically before discharge. Long-term outcomes could not be assessed due to loss to follow-up.

**Conclusion:** This case illustrates an uncommon presentation of Charcot knee arising from combined central and peripheral neurological impairment, emphasizing diagnostic challenges and the necessity of individualized, risk-sensitive management in medically fragile patients.

**Keywords:** Charcot joint, Neuroarthropathy, Knee joint, Diabetes mellitus, Hydrocephalus, Conservative treatment.

## Introduction

Charcot neuroarthropathy is a progressive destructive joint disorder caused by impaired protective sensation, most commonly associated with diabetes mellitus but also seen in central neurologic conditions such as syringomyelia, spinal cord injury and neurosyphilis [1]. Although the foot and ankle are

typical sites, involvement of the knee is rare and often presents late due to minimal pain perception and delayed recognition. Diagnostic uncertainty frequently arises because Charcot knee can mimic septic arthritis, especially in medically complex or critically ill patients with elevated systemic inflammatory markers. Advanced cases may show severe instability, fracture–dislocation patterns or pseudoarthrosis, complicating both diagnosis and management. This report describes an atypical presentation of Charcot knee in an elderly patient with combined central and peripheral neurological compromise, emphasizing the challenges of diagnosis, the influence of comorbidities on treatment decisions and the

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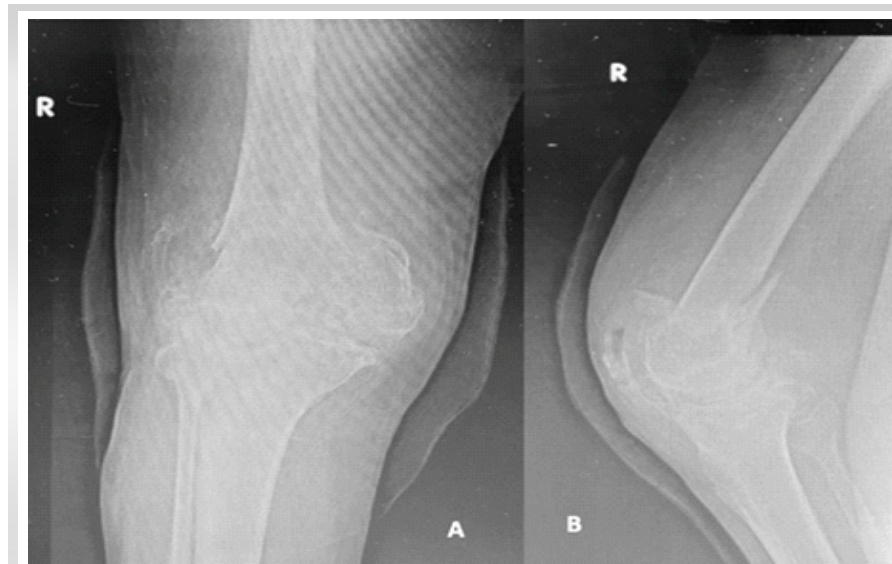
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**Figure 1:** Images (a and b) illustrate right knee radiographs revealing a neglected distal femoral fracture accompanied by severe joint destruction, patellar fragmentation, and prominent peri-articular callus with osteophyte formation.

rationale for conservative management in high-risk individuals.

### Case Report

An 83-year-old man with hydrocephalus, encephalopathy and Type 2 diabetes mellitus was admitted to the medical intensive care unit (ICU) with sepsis secondary to an upper respiratory tract infection and initiated on intravenous antibiotics. During routine bedside physiotherapy, the team noted a painful, immobile right knee with diffuse swelling and mild warmth relative to the contralateral side, prompting orthopedic consultation for suspected septic arthritis.

Diagnostic aspiration yielded 40 mL of blood-tinged fluid, with negative Gram stain and sterile cultures, although systemic inflammatory markers remained elevated due to ongoing sepsis.

Plain radiographs revealed a neglected distal femoral fracture with complete destruction of the joint, lateral femoral condyle erosion, dipping into the tibial plateau leading to bone loss, patellar fragmentation and severe distortion of knee architecture with peri-articular callus and osteophytes (Fig. 1). Computed tomography (CT) confirmed distal femoral non-union lacking callus formation between the fragments but was present distal to the fracture toward tibia causing pathological pseudoarthrosis at fracture site, a fragmented-eroded patella and a

depressed-eroded tibial plateau with peri-articular callus giving an impression of pathological ankylosis (Fig. 2). 3-D reconstruction of the CT images revealed the multiple fragmentations with peri-articular callus, but no consolidation of the fracture site (Fig. 3). Magnetic resonance imaging further demonstrated complete anterior and posterior cruciate ligament ruptures, meniscal destruction and attenuated medial collateral ligament secondary to chronic fragment displacement (Fig. 4).

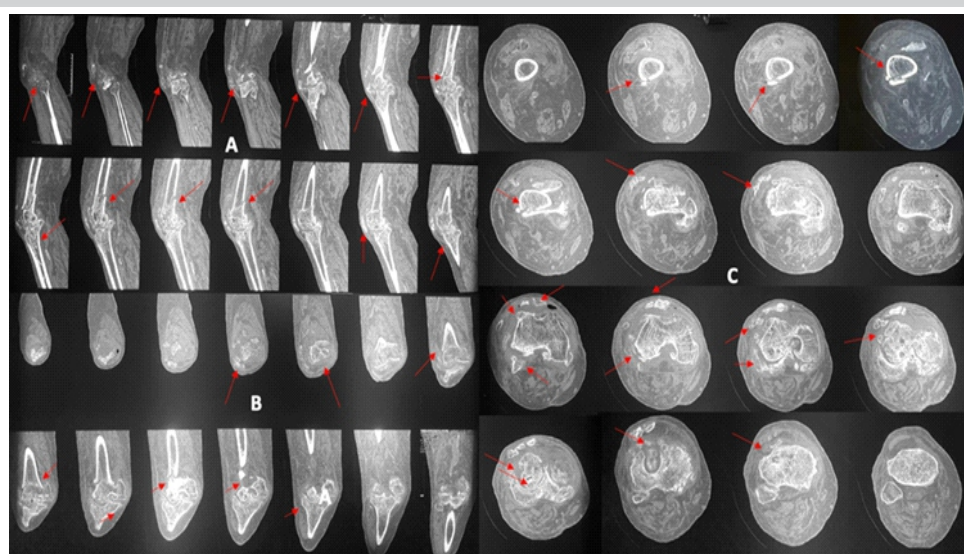
Discussion with the patient's attendant uncovered a 4–5 year history of recurrent falls and approximately 7 months of non-ambulation, consistent with neuropathic neglect of the limb. Given the extensive irreversible joint destruction, chronic instability and compromised soft tissues, knee arthrodesis with an Ilizarov external

fixator was determined to be the only feasible salvage procedure. However, due to significant anesthetic risk from the patient's multiple comorbidities, the family declined surgical intervention.

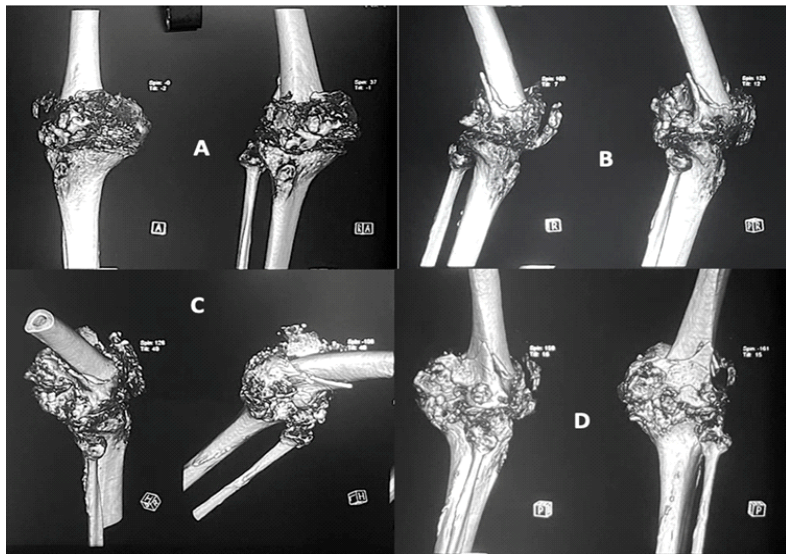
The patient was managed conservatively with a long knee brace, analgesia, ankle range-of-motion exercises and pneumatic compression. He remained under observation for 2 months with gradual improvement in infectious parameters and was subsequently discharged, but was lost to follow-up (Table 1).

### Discussion

Neuropathic arthropathy or Charcot joint results from loss of



**Figure 2:** Images (a, b, c, d) show sagittal, coronal, and axial computed tomography views of the knee, with arrow markers indicating pronounced bony erosions and fragmentation characteristic of advanced neuropathic arthropathy.



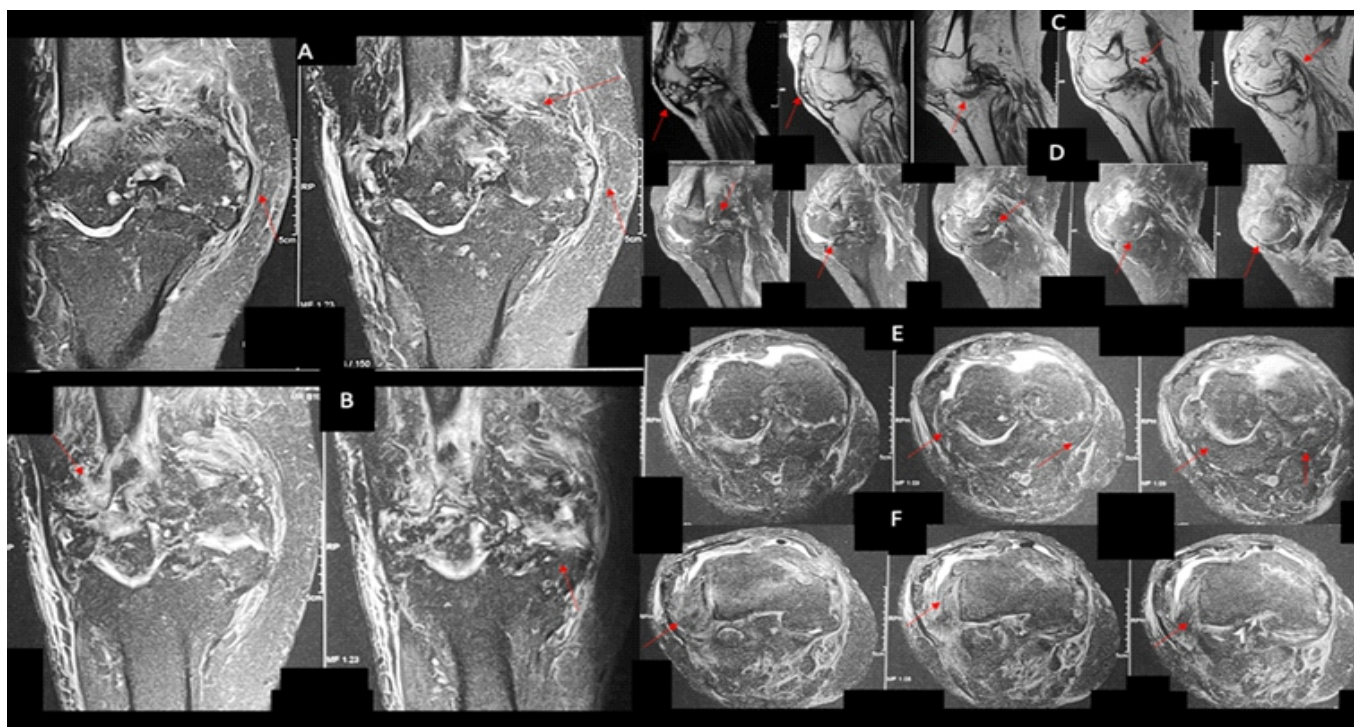
**Figure 3:** Images (a, b, c, d) show 3D-reconstructed views of the knee from multiple orientations, demonstrating the extent of joint destruction.

protective sensation due to neurologic disorders, permitting repetitive unnoticed microtrauma that triggers joint destruction through neurotraumatic (cumulative insensible injury) and neurovascular (hyperemia-driven osteoclastic resorption) mechanisms [1]. Diabetes mellitus causes most cases, followed by syringomyelia, tabes dorsalis (syphilis), alcoholism, spinal cord injury, stroke and congenital

insensitivity [1, 2]. In this patient, hydrocephalus-related encephalopathy and diabetes likely produced central-peripheral sensory deficits, allowing 4–5 years of falls to cause a distal femoral neglected fracture with pseudoarthrosis, akin to rare knee Charcot from neurosyphilis rather than typical diabetic foot predominance.

Neuropathic arthropathy frequently occurs in the context of multisystem comorbidities that accelerate joint destruction and complicate management. Diabetes mellitus contributes through peripheral neuropathy, impaired microvascular perfusion and diminished healing capacity, while central neurologic disorders such as hydrocephalus or encephalopathy impair cognition, balance and protective reflexes, predisposing patients to repetitive unrecognized trauma. Superimposed sepsis further elevates systemic inflammatory markers, obscuring local diagnostic cues and complicating differentiation from

infection-related arthropathies [3]. Advanced age compounds these factors by increasing anesthetic risk, reducing physiological reserve and limiting post-operative rehabilitation potential. In this patient, the convergence of hydrocephalus-related encephalopathy, diabetes and active sepsis created a far more complex biological environment than that seen in typical



**Figure 4:** Images (a and b) show coronal T2-weighted magnetic resonance imaging cuts demonstrating attenuation of the medial collateral ligament caused by displaced bony fragments and medial femoral condyle impingement. Images (c and d) display sagittal T1- and T2-weighted sequences revealing patellar fragmentation, meniscal destruction, absence of both cruciate ligaments, joint space obliteration, and effusion. Images (e and f) present axial T2-weighted images from the distal femoral surface to the tibial plateau, highlighting the extensive anatomical distortion of the knee.

Charcot knee cases, which usually stem from isolated peripheral neuropathy without critical illness.

Charcot arthropathy of the knee typically presents with insidious swelling, warmth, erythema, progressive deformity and mechanical instability, evolving through the classic Eichenholtz stages: Fragmentation (acute hyperemia, osseous fragmentation and joint debris), coalescence (resorption of loose fragments and early structural consolidation) and reconstruction (ankylosis or stabilization through exuberant bony overgrowth) [4]. In non-ambulatory or bedridden patients, however, overt deformity or instability may be less apparent and more subtle indicators, such as physiotherapy-elicited pain, immobility, or an unexplained effusion may serve as the first clinical clues [1, 4]. In this case, the combination of diffuse swelling, mild warmth, a blood-tinged effusion, and radiological evidence of condylar collapse despite 7 months of non-ambulation is most consistent with an advanced coalescence stage, comparable to patterns described in syphilitic and diabetic Charcot knee cohorts [1, 4]. However, the predominance of distal femoral destruction and the patient's demonstrable pain response, despite underlying neuropathy, distinguish this presentation from more typical peripheral neuropathic knee patterns, reflecting superimposed mechanical failure from an unhealed distal femoral fracture.

Neuropathic arthropathy can closely imitate septic arthritis, as both may present with joint effusion, warmth, swelling, and elevated systemic inflammatory markers such as erythrocyte sedimentation rate and C-reactive protein [5, 6, 7]. However, distinguishing features of Charcot arthropathy include sterile synovial aspirates, chronic deformity, and characteristic imaging findings; marked osseous fragmentation, joint disorganization, and collapse without abscess formation or periosteal reaction [5, 6]. Other differential diagnoses include rapidly progressive osteoarthritis, which produces subchondral collapse but generally preserves ligamentous integrity; crystal arthropathies, which are identified by birefringent crystals on aspiration; and amyloid arthropathy, which typically exhibits lower systemic inflammatory activity [6, 7]. None of these entities demonstrates the profound instability, distal femoral non-union, or patellar fragmentation observed in this patient. In the ICU setting, where systemic sepsis confounded interpretation of inflammatory markers, septic arthritis was an appropriate initial concern. However, the combination of

negative Gram stain and cultures, along with radiographs showing lateral condylar cave-in and patellar fusion, provided decisive evidence against acute pyogenic arthritis and instead supported a diagnosis of advanced neuropathic joint destruction.

Early management of Charcot arthropathy prioritizes strict off-loading through total contact casting, bracing, or external fixation to interrupt the destructive cycle during the active phase, supplemented by non-steroidal anti-inflammatory drugs or bisphosphonates to mitigate hyperemia-driven osteolysis [5, 8]. Surgical intervention, most commonly knee arthrodesis using circular external fixation such as an Ilizarov frame, is reserved for end-stage disease (Eichenholtz stage 3) marked by fixed deformity, instability, or non-union [8, 9]. Reported fusion rates range from 85 to 95%, though complication rates remain substantial (20–50%), including pin-tract infections, refractures, delayed union, and soft-tissue problems, particularly in elderly or medically complex patients [8, 9]. In frail individuals with significant comorbidities, conservative treatment is often favored, emphasizing pain control, maintenance of residual range of motion, prevention of venous thromboembolism, and overall functional support rather than operative reconstruction [10]. In this case, the choice of a long knee brace, analgesia, ankle mobilization exercises, and pneumatic compression following medical stabilization represented an appropriate non-operative strategy given the patient's advanced age, encephalopathy, diabetes, and ongoing sepsis, all of which greatly increased anesthetic and perioperative risk. This aligns with outcomes reported in high-risk diabetic Charcot knee cohorts, where non-operative management is frequently selected despite its limitations [10]. Unfortunately, loss to follow-up prevented assessment of long-term stability or functional recovery in this patient.

This case report is limited by the absence of long-term follow-up, which precludes evaluation of the outcomes of conservative management, such as progression to spontaneous ankylosis, maintenance of functional stability, or complications, including skin breakdown from prolonged bracing. In addition, the lack of formal neuropathy assessment (e.g., nerve conduction studies) and precise Eichenholtz staging restricts the ability to definitively characterize disease chronicity and pathophysiology beyond clinical and radiological correlation. Furthermore, the absence of surgical intervention or a

**Table 1: Sequential follow-up of inflammatory and infection markers was performed throughout the 8-week hospitalization**

Weeks parameters	1	2	3	4	5	6	7	8
Erythrocyte sedimentation rate	62	47	44	48	36	35	32	27
C-reactive protein	4.68	3.74	3.63	3.8	3.2	3.2	2.93	2.6
Total leukocyte count	14170	12160	12450	12780	13980	12060	12142	11984

comparative cohort limits the ability to empirically assess the influence of comorbidities on management decisions, rendering such conclusions primarily observational and inferential.

### Conclusion

This case highlights a rare presentation of Charcot neuroarthropathy of the knee in an elderly, medically complex patient, where combined central and peripheral neurological impairment led to progressive, unrecognized joint destruction culminating in distal femoral non-union and severe arthropathy. The overlapping features of systemic sepsis, chronic neuropathy, and advanced structural collapse obscured early

diagnosis, underscoring the importance of maintaining a high index of suspicion for neuropathic arthropathy in atypical joints, particularly when aspirate cultures are sterile, and imaging reveals chronic deformity. Management decisions in such patients must balance the potential benefits of surgical reconstruction against substantial perioperative risks posed by age and comorbidities. In this instance, conservative therapy provided a pragmatic and appropriate alternative. Ultimately, this case reinforces the need for early identification, multidisciplinary assessment, and individualized treatment strategies in Charcot knee, especially in frail or critically ill populations where standard surgical pathways may not be feasible.

**Declaration of patient consent:** The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the Journal. The patient understands that his name and initials will not be published, and due efforts will be made to conceal his identity, but anonymity cannot be guaranteed.

**Conflict of Interest:** NIL; **Source of Support:** NIL

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