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Mutilating Multifocal Osteoarthritis: A Challenging Diagnostic Entity

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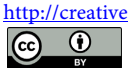

Abstract

Introduction: Multifocal mutilating osteoarthritis or multiple joint osteoarthritis (MJOA), is a rare entity. It is responsible for severe joint destruction, which may mimic neuropathic or metabolic arthropathies. These extreme forms lead to major functional disability and represent a significant diagnostic and therapeutic challenge. **Case Presentation:** We report the case of a 73-year-old patient, a candidate for total hip arthroplasty, referred from the orthopedic surgery department for evaluation of polyarticular involvement. He had experienced mechanical polyarthralgia for ten years, affecting the large joints of both the lower and upper limbs, with progressive worsening over the past year, resulting in to major functional impairment. Clinical examination revealed severe genu varum and bilateral talus valgus deformities, resulting in limping and the need for walking aids. Imaging demonstrated severe and mutilating multifocal osteoarthritis, including Kellgren-Lawrence grade IV knee osteoarthritis, involvement of the ankle, hip osteoarthritis with acetabular protrusion, and eccentric glenohumeral osteoarthritis. The diagnosis of multifocal osteoarthritis (MJOA) was established after an exhaustive diagnostic workup that excluded secondary causes. Given the severity of the polyarticular involvement, prosthetic surgery was considered; however, the patient ultimately did not undergo the procedure, highlighting the functional and therapeutic complexity of this phenotype. **Discussion:** Several secondary arthropathies may mimic MJOA, including neuropathic arthropathies, metabolic disorders, and chronic infections. Clinical and radiological similarities make the diagnosis challenging. This case emphasizes the importance of a rigorous diagnostic approach in order to avoid inappropriate therapeutic management. **Conclusion:** Recognition of these rare forms helps prevent diagnostic delay, improves understanding of the evolutionary spectrum of MJOA, and allows optimization of func-

tional management strategies.

Subject Areas

Medicine, Rheumatology, Osteoarthritis

Keywords

MJOA, Mutilating, Multifocal, Osteoarthritis, Differential Diagnosis

1. Introduction

Osteoarthritis is the most common chronic joint disorder and represents a major cause of functional disability in the elderly population. Although most cases are primary, up to 10% - 15% correspond to secondary osteoarthritis related to neuropathic, metabolic or infectious causes [1]. Neuropathic arthropathies, particularly those associated with diabetic neuropathy, syringomyelia, or tertiary syphilis, lead to rapidly progressive, painless joint destruction with a mutilating radiographic appearance [2]. Metabolic disorders such as chondrocalcinosis, gout, hemochromatosis, or ochronosis may also result in aggressive osteoarthritis [3]-[6].

In the absence of an identifiable secondary cause, multifocal osteoarthritis may be classified as multiple joint osteoarthritis (MJOA). Several definitions have been proposed based on the number and distribution of affected joints, including involvement of at least three joint sites or specific combinations of lower-extremity joints [7] [8]. However, no universally accepted operational definition currently exists. The prevalence of MJOA has been reported to range from 8% to 12% among patients with osteoarthritis, while the severe or mutilating phenotype represents a much smaller proportion. This phenotype is characterized by extensive joint destruction and major functional impairment, with a marked discrepancy between radiographic severity and limited clinical or biological inflammatory expression [7] [8]. These features underscore the importance of early recognition and appropriate management [8]-[10].

2. Case Presentation

We report the case of a 73-year-old man who was a candidate for total hip arthroplasty. He was referred from the orthopedic surgery department for evaluation of polyarticular involvement. He had experienced progressively worsening mechanical arthralgia since 2016, affecting the large joints of both the lower and upper limbs, with a pain visual analog scale (VAS) score of 50 mm.

Functional impairment was severe, requiring the use of a walking aid for the past year. The Lequesne index was 16 for the knee and 15 for the hip, indicating major disability. His walking distance was limited to less than 100 meters.

The patient was retired and had previously performed manual labor involving regular heavy lifting. His medical history included benign prostatic hyperplasia

requiring two surgical procedures and prior lacunar ischemic strokes. There was no history of primary or secondary syphilis, nor of diabetes. He denied any previous or repetitive trauma and had no similar family history. There were no clinical signs suggestive of tuberculosis exposure or active tuberculosis.

On physical examination, the patient appeared in generally good condition but underweight (44 kg for 163 cm in height). A persistent limp was observed during ambulation. Both knees exhibited severe bilateral genu varum, with mild positive patellar tap sign on the left and marked patellofemoral crepitus on the right.

Examination of the ankles revealed asymmetric right anterolateral swelling, absent plantar flexion, dorsiflexion limited to 20˚, and bilateral hindfoot valgus, without local inflammatory signs. Hip examination showed limited internal rotation (10˚ on the left, 0˚ on the right) with preserved flexion. In the upper limbs, both shoulders demonstrated pseudoparalysis.

Neurological examination revealed preserved muscle strength. Patellar reflexes were absent. There were no signs of posterior column syndrome, no impairment of deep sensation, and no Argyll Robertson pupil. Additionally, a firm, well-defined, painless mass was palpable over the left pectoralis major region. It measured approximately 7 cm in diameter and was not associated with overlying cutaneous inflammatory changes.

The patient's radiological assessment revealed a complex pattern combining destructive lesions. Imaging demonstrated left medial femorotibial and bilateral femoropatellar knee osteoarthritis, classified as Kellgren-Lawrence grade 4. There was marked joint space narrowing with large osteophytes and subchondral sclerosis (Figure 1). A moderate to severe genu varum deformity was measured on long-leg alignment radiographs (pangonogram), with mechanical axis deviation of −18˚ on the right and −25˚ on the left.

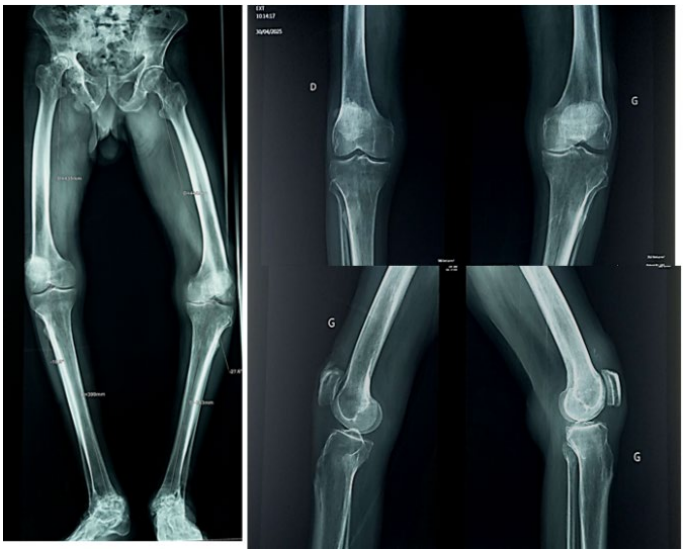

Figure 1. Knee radiographs and long-leg alignment radiograph (pangonogram) demonstrating bilateral genu varum associated with left medial femorotibial and femoropatellar osteoarthritis.

Advanced right hip osteoarthritis was also identified, characterized by a heterogeneous femoral head, cortical erosions, and widening of the acetabular floor. Acetabular protrusion was noted, along with a probable fracture line of the right ischium. These findings were associated with joint effusion and a posterolateral fluid collection within the right gluteal muscle on MRI (Figure 2).

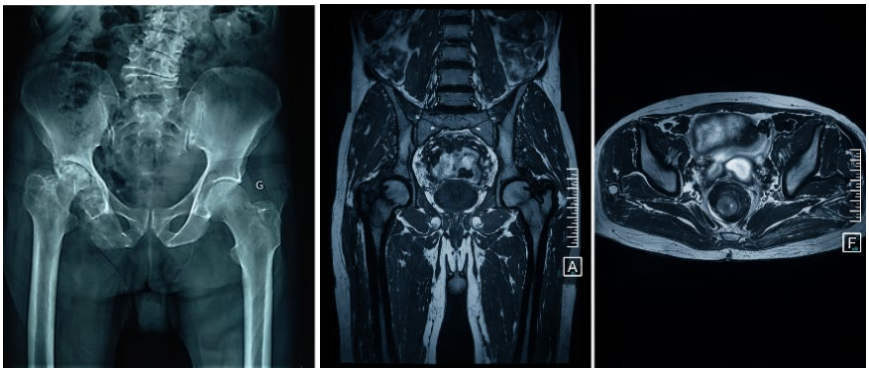

Figure 2. Pelvic radiograph demonstrating secondary hip osteoarthritis with erosions of the femoral head. Magnetic resonance imaging (MRI) revealed hip joint effusion associated with a right gluteal fluid collection.

The ankles exhibited mutilating changes, including bilateral talar osteolysis, large subchondral geodes, and exuberant osteophyte formation, while preserving the talonavicular and Lisfranc joints (Figure 3).

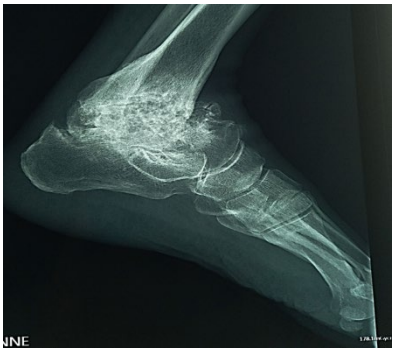

Figure 3. Lateral ankle radiograph demonstrating areas of osteolysis associated with exuberant osteophyte formation.

Standard radiographs revealed bilateral eccentric glenohumeral osteoarthritis and acromiohumeral joint degeneration. Ultrasound demonstrated subacromial-subdeltoid bursitis with marked synovial hypertrophy and small intramuscular collections, including a calcified cyst of the left pectoralis minor measuring 28 × 21 × 69 mm. Spinal imaging showed degenerative lumbar scoliosis and multilevel disc disease, predominantly at the L1 - L2 level.

Laboratory investigations showed fluctuating C-reactive protein (CRP) levels ranging from 4 to 29 mg/L. The erythrocyte sedimentation rate (ESR) was within the normal range at 20 mm/hour. A mild leukocytosis was noted, with a white

blood cell count of 10.86 G/L. Procalcitonin was negative at 0.07 ng/mL. Renal, hepatic, and thyroid function tests were within normal limits.

All these clinical, radiological, and laboratory findings were suggestive of a chronic mutilating polyarthropathy, predominantly affecting the large joints of both the upper and lower limbs, and associated with axial deformities.

Serologic testing for syphilis in the blood was negative. Electroneuromyographic (ENMG) studies revealed multiple mononeuropathy with motor involvement (reduced compound muscle action potentials of the bilateral median nerves and left ulnar nerve in the upper limbs, and of the left common peroneal and posterior tibial nerves in the lower limbs) as well as sensory involvement (right median nerve, left ulnar nerve, and left sural nerve).

Neurogenic arthropathy was initially considered in view of the marked joint destruction and the discrepancy with the reported pain intensity. However, comprehensive investigations, including specialist neurological assessment, concluded that there was no severe central or peripheral neurological involvement capable of explaining the articular lesions.

Radiographic and ultrasonographic imaging showed no evidence of cartilaginous calcification, and ankle involvement was atypical. Serum uric acid was 63.17 mg/L. The phosphocalcium profile showed a corrected serum calcium level of 83.6 mg/L and normal phosphate levels (45 mg/L). Parathyroid hormone (PTH) was elevated at 97.60 ng/L [10 - 65 ng/L]. Renal function was normal. 25-hydroxyvitamin D was markedly decreased at 7 ng/mL [30 - 100 ng/mL]. The elevated PTH was therefore considered secondary to vitamin D deficiency, with no evidence of a clinically significant metabolic bone disorder contributing to the destructive arthropathy.

Synovial fluid analysis (subacromial-subdeltoid bursa aspiration) showed no crystals and a non-inflammatory cell count with white blood cells 160/mm^3. Iron studies did not demonstrate iron overload, with a serum iron level of 0.46 mg/L, ferritin of 218.12 ng/mL, and transferrin saturation coefficient of 15.44%. Additionally, minor salivary gland biopsy showed no amyloid deposits.

Metabolic causes such as chondrocalcinosis or gout, as well as deposition disorders including amyloidosis or hemochromatosis, were suspected due to their potential for destructive joint disease and the unusual involvement of certain joints, particularly the ankles. However, the results of the investigations allowed these etiologies to be excluded.

The tuberculosis workup revealed a positive QuantiFERON-TB Gold test at 2.98 IU/mL. However, GeneXpert testing and cultures from multiple specimens (subacromial-subdeltoid bursal fluid, right gluteal muscle collection, biopsy of the left subpectoral mass, and sputum samples) were all negative. Synovial fluid from the shoulder was non-inflammatory (160 cells/mm^3) and sterile. Histological examination of biopsies from right hip soft tissue lesions and the left pectoralis minor muscle demonstrated nonspecific fibro-inflammatory tissue, without epithelioid or giant-cell granulomas and without caseous necrosis. A small nonspecific

pulmonary micronodule was identified, with no radiological evidence suggestive of pulmonary tuberculosis.

Osteoarticular tuberculosis was considered because of the chronic and destructive nature of the lesions, the presence of periarticular collections, and particularly the epidemiological context, as Morocco is a tuberculosis-endemic country. This situation warranted an exhaustive diagnostic approach, which ultimately allowed us to rule out active osteoarticular tuberculosis.

Following a multidisciplinary case conference involving rheumatologists, radiologists, infectious disease specialists, neurologists, and pathologists, the diagnosis of severe multifocal polyosteoarthritis (MJOA) was established. The diagnosis was based on the chronic course, the mechanical nature of the pain, and the multifocal, mutilating involvement of the hips, knees, ankles, and shoulders. Radiological findings including marked joint space narrowing, exuberant osteophyte formation, and large subchondral geodes were consistent with advanced osteoarthritis, without evidence supporting a secondary etiology.

The main comorbidities identified included isolated hypogammaglobulinemia (6.84 g/L), without Bence Jones proteinuria or monoclonal light chains on serum and urine immunofixation. Imaging revealed a right tonsillar nodular lesion measuring 13 × 13 mm, without associated lymphadenopathy. Incidental findings included moderate aneurysmal dilation of the ascending aorta and aortic arch (37 mm), a 7.7 mm left renal macrocalculus, moderate dilation of the main pancreatic duct (Wirsung duct, 5 mm), and bilateral simple renal cysts.

Given the severity of the polyarticular involvement, the patient's advanced age, and the presence of multiple comorbidities, although prosthetic surgery was initially indicated, it was ultimately not pursued because of the patient's advanced age, extensive bone destruction, multiple comorbidities, and unfavorable overall surgical risk-benefit assessment.

3. Discussion

Osteoarthritis is the most common chronic joint disorder and represents a major cause of functional disability in the elderly population. Although most cases are primary, up to 10% - 15% correspond to secondary osteoarthritis related to neuropathic, metabolic, or infectious causes [1]. The clinical and radiological presentation of our patient, characterized by severe and mutilating polyarticular involvement, required the priority exclusion of the main secondary causes that may mimic multifocal polyosteoarthritis (MJOA).

Neurogenic arthropathies, particularly in the context of Charcot joint disease, syringomyelia, or neurosyphilis, represent a major differential diagnosis. They are characterized by rapidly progressive joint destruction, large subchondral geodes, and frequent clinico-radiological dissociation, often with relative joint indolence [2] [11]. These features have also been described in certain severe forms of MJOA. However, the absence of profound neurological deficits, trophic disturbances, or severe central or peripheral nervous system involvement, together with negative

blood syphilis serology and no prior history of primary syphilis in our patient, made this hypothesis unlikely. The peripheral neuropathy documented on electroneuromyography (ENMG) was considered a potential aggravating factor, may contributing to disease severity and the mutilating pattern through impaired joint protective mechanisms.

Microcrystalline arthropathies, particularly calcium pyrophosphate deposition disease (CPPD) and chronic gout, may also lead to destructive joint lesions, characterized by linear or punctate calcific deposits within hyaline cartilage, asymmetric joint space narrowing, irregular subchondral erosions, and large geodes. These features may mimic an aggressive osteoarthritis phenotype such as MJOA [5]. However, the absence of cartilaginous calcifications, negative crystal analysis in synovial fluid, and a non-suggestive biological profile allowed these diagnoses to be ruled out.

Certain metabolic and deposition disorders, such as hemochromatosis, amyloidosis, and more rarely Wilson's disease, may also cause chronic destructive arthropathy. Radiographically, these conditions are characterized by joint space narrowing, atypical hook-like osteophytes, and subchondral geodes. Joint involvement predominantly affects large joints and the metacarpophalangeal (MCP) joints [4] [6]. Clinical similarities with MJOA include chronicity, multifocal involvement, and the mechanical nature of pain. Nevertheless, the absence of biological, clinical, and histological findings supporting these conditions enabled their exclusion.

Chronic infectious arthritis, particularly osteoarticular tuberculosis, must be systematically considered in the presence of any slowly progressive destructive arthropathy, especially in endemic areas. These conditions may manifest as progressive bone destruction, subchondral geodes, chronic joint effusions, and periarticular collections, potentially mimicking mutilating osteoarthritis [12]. However, the negative microbiological, histological, and molecular investigations, together with the absence of systemic features, allowed this diagnosis of active osteoarticular tuberculosis unlikely in our patient. The positive Quantiferon -TB Gold test was interpreted as reflecting latent tuberculosis infection, which is common in this age group and in endemic regions.

After rigorous exclusion of all these secondary causes, the diagnosis of severe MJOA was retained [8] [13]. In our case, involvement of the hips, knees (Kellgren-Lawrence grade IV), ankles, shoulders, and spine clearly fulfilled the concept of multiple joint osteoarthritis, although no universally accepted diagnostic criteria currently exist for MJOA [7]. The term "mutilating" is used here to describe the unusually severe structural destruction and associated functional impairment rather than as a formally established diagnostic subtype. Several cases have been reported in the literature describing severe multifocal osteoarthritis following a negative etiological workup, notably the case reported by Çelik *et al.*, which closely resembles our patient's presentation [14].

Risk factors associated with severe forms of MJOA are multifactorial. Advanced age and severe axial deformities, such as genu varum, increase mechanical joint

stress and accelerate cartilage degeneration. From a genetic perspective, MJOA and generalized osteoarthritis have a substantial and polygenic genetic component. More than 300 osteoarthritis-associated loci have been identified through large-scale genomic analyses, and several genes, including *GDF5*, *COL11A1*, *NCOA3*, and *COG5*, appear to contribute to susceptibility to severe forms of the disease, irrespective of the specific joint site involved [15] [16]. Our patient had no evident family history. Nevertheless, multifactorial genetic susceptibility likely contributed to his severe, multifocal, and mutilating phenotype, in interaction with mechanical and neurological factors. However, no genetic testing was performed in this case.

Management of severe forms of MJOA requires a multidisciplinary approach, combining pharmacological analgesic strategies (nonsteroidal anti-inflammatory drugs and step II/III analgesics), non-pharmacological interventions (physical therapy, biomechanical optimization, orthoses, and walking aids), as well as nutritional support in underweight or sarcopenic patients. Surgical options (total joint arthroplasty, osteotomies) may be considered for the most disabling joints; however, their indication is often limited by the extent of bone destruction, axial deformities, and bone quality [17].

Given the extensive joint destruction and major functional impairment, a multidisciplinary conservative management strategy was implemented. Pain was managed with paracetamol 1 g three times daily as needed. Symptomatic slow-acting anti-osteoarthritis treatment with chondroitin sulfate (Chondrosulf®) was prescribed at one capsule daily for 3 months, followed by a 1-month therapeutic interval. The patient also underwent rehabilitation and used a tripod walking aid to improve mobility and stability. Nutritional management was considered in view of his low body weight (44 kg for 163 cm). Total hip arthroplasty was initially considered because of the severity of the right hip involvement but was ultimately not performed. The multidisciplinary team considered that the severe knee involvement would limit the functional benefit of hip arthroplasty, while his advanced age and low body weight increased the anesthetic and surgical risk. In addition, the patient preferred to avoid surgery. At the latest follow-up, the patient was bedridden due to a stroke and was therefore unable to undergo a meaningful functional assessment of his osteoarthritis.

Finally, several diagnostic limitations should be acknowledged. No genetic analysis was performed. No bone biopsy was undertaken. Syphilis serology (TPHA and VDRL) was not assessed in the cerebrospinal fluid, given the absence of clinical or biological arguments supporting tabetic arthropathy (arthrotabes).

4. Conclusion

This case illustrates a severe and mutilating form of multifocal osteoarthritis. The final diagnosis of severe MJOA was established after an exhaustive workup excluding neuropathic, metabolic, and infectious causes. These observations highlight the need to develop precise diagnostic criteria to better identify severe forms

of MJOA, to establish dedicated registries, and to conduct longitudinal studies in order to improve understanding of their natural history and optimize management strategies. Finally, this case underscores that, although rare, such forms do exist and warrant heightened clinical and radiological vigilance, supported by close coordination between rheumatology, neurology, radiology, and orthopedic surgery.

Patient Consent

Written informed consent was obtained from the patient for the publication of this case report, including clinical details and any accompanying images.

Author Contributions

Patient management: Ilyass Chergaoui, Anass Kherrab, Mirieme Ghazi; data collection: kaoutar Elaatifi, Maria Adel, Anass Chbihi-Kaddouri; manuscript drafting: kaoutar Elaatifi; critical revision of the manuscript: Imane El Bouchti, Redouane Niamane; supervision and final approval: Redouane Niamane. All authors have read and approved the final version of the manuscript.

Conflicts of Interest

The authors declare no competing interest.

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