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Melorheostosis with Giant Extraosseous Ossification of the Fibula Mimicking Malignancy: A Case Report

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ABSTRACT

Melorheostosis is a rare sclerosing bone dysplasia characterized by segmental cortical hyperostosis, classically presenting with a "dripping candle-wax" appearance. We report a 52-year-old woman with a three-year progressive mass of the left lower leg associated with pain, numbness, and previous ulceration. A prior core biopsy suggested osteochondroma. However, radiographs demonstrated characteristic flowing cortical hyperostosis of the fibula with extensive adjacent extraosseous ossification, while cross-sectional imaging showed a mineralized mass without destructive features. Histopathology revealed woven and sclerotic bone trabeculae with osteoblastic rimming and vascular fibrous stroma without evidence of malignancy. The diagnosis of melorheostosis was established through clinicoradiological and histopathological correlation. The extensive extraosseous component obscured the typical presentation and mimicked a neoplastic process, highlighting the potential limitations of isolated biopsy interpretation. This case emphasizes the importance of recognizing imaging-pathology discordance and integrating clinical course, characteristic radiographic findings, and histopathology to avoid misdiagnosis and unnecessary oncological treatment.

***Keywords*:** melorheostosis; extraosseous ossification; cortical hyperostosis; diagnostic challenge; osteochondroma

INTRODUCTION

Melorheostosis is a rare, sporadic sclerosing bone dysplasia characterized by excessive bone formation and irregular cortical hyperostosis. First described by Léri and Joanny in 1922 as hyperostose en coulée, its classical radiographic appearance resembles "dripping candle wax" flowing along one side of a long bone [1,2]. The estimated prevalence is approximately one case per million population, with no consistent sex predilection [3]. Melorheostosis predominantly affects the appendicular skeleton and usually has a segmental, asymmetric distribution. It may involve a single bone, multiple bones within one extremity, or contiguous bones across a joint [2,3]. Somatic mosaic activating MAP2K1 variants, which dysregulate RAS–MAPK signaling, have been identified in a substantial proportion of lesions with the classical radiographic phenotype [4,5].

Clinical manifestations vary with lesion location, extent, and activity. Patients may

present with pain, swelling, joint stiffness, restricted motion, contracture, deformity, or limb-length discrepancy [3,6–8]. Progressive osseous or periarticular overgrowth may also cause peripheral nerve compression and sensory disturbance [9]. Melorheostosis is principally a radiologic diagnosis [8,10]. Radiographs demonstrate the characteristic cortical pattern, computed tomography best delineates cortical architecture and mineralization, and magnetic resonance imaging complements these studies by assessing marrow, muscle, neurovascular structures, and non-mineralized soft tissue.

Mineralization may extend into adjacent ligaments, tendons, fascia, joint capsules, and muscles. Although extraosseous involvement is increasingly recognized, most reported lesions are focal or periarticular; a giant multicompartmental soft-tissue mass remains uncommon [8,10–12]. When the extraosseous component dominates, the lesion may resemble heterotopic ossification, osteochondroma, or a surface or extraskeletal bone-forming neoplasm [13,14]. Diagnosis is further complicated by heterogeneous, non-specific histology that may include woven and lamellar bone, osteoid, osteoblastic activity, fibrosis, vascular proliferation, and osteocartilaginous tissue [5,15]. A limited biopsy may therefore be unrepresentative when interpreted without whole-lesion imaging [13,15].

We report melorheostosis in a 52-year-old woman presenting with an approximately 30-cm multicompartmental extraosseous ossified mass surrounding the fibula. The lesion's extensive blastic appearance raised concern for a bone- or soft-tissue-forming neoplasm, whereas a previous biopsy was reported as osteochondroma. This case highlights the diagnostic value of integrating the clinical course, characteristic fibular cortical pattern, non-destructive cross-sectional imaging, operative anatomy, and histopathology.

CASE PRESENTATION

A 52-year-old woman with type 2 diabetes mellitus presented with a mass along the proximal anterolateral left lower leg that had enlarged progressively over three years from an initially marble-sized nodule. She reported no preceding trauma and no similar lesion elsewhere. During the year before presentation, she developed intermittent dull pain along the lateral leg and numbness radiating to the dorsum of the foot as the mass enlarged. Six months before presentation, she reported a febrile episode associated with ulceration over the mass and underwent wound debridement at another hospital. One month before presentation, a biopsy performed elsewhere was reported as osteochondroma. She denied current fever, appetite loss, nausea, or vomiting, and reported no family history of bone tumor.

Examination showed a firm, fixed swelling extending along the anterolateral left leg, with an overlying ulcerated scar at the site of previous treatment (Figure 1). There was localized tenderness without venous engorgement, diffuse erythema, or distal edema. Distal perfusion and motor function were preserved; the patient reported intermittent altered sensation over the dorsum of the foot. Knee and ankle ranges of motion were clinically preserved.

Anteroposterior and lateral radiographs demonstrated extensive irregular longitudinal cortical hyperostosis along the fibula with a large adjacent ossified soft-tissue component (Figure 2). There was no acute fracture, aggressive cortical destruction, or aggressive periosteal reaction.

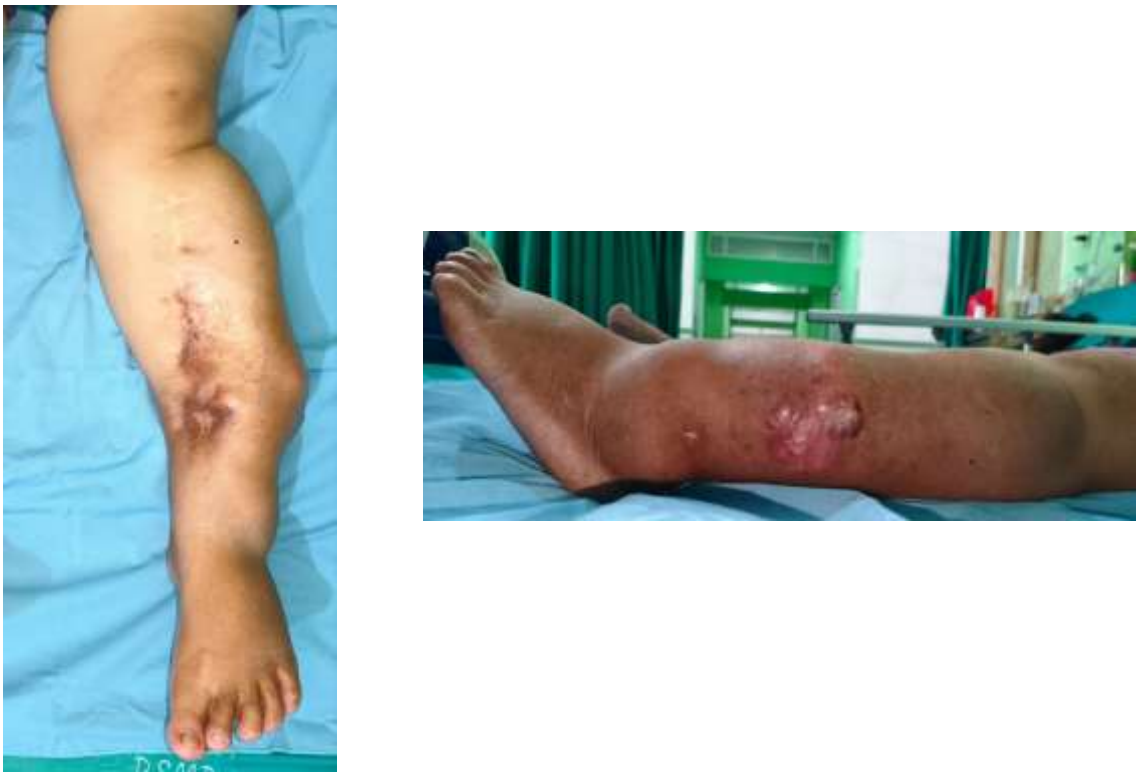

Figure 1. Preoperative clinical photographs showing a long anterolateral left-leg mass with overlying ulceration and scars from previous treatment.

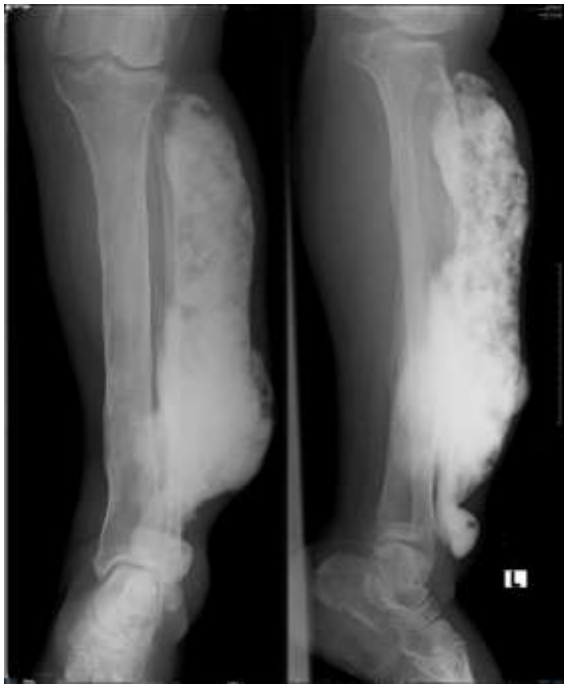

Figure 2. Anteroposterior and lateral radiographs of the left leg showing longitudinal flowing cortical hyperostosis of the fibula and an extensive adjacent ossified soft-tissue mass without aggressive osseous destruction.

Magnetic resonance imaging of the left leg demonstrated an approximately 30-cm ill-defined multicompartmental soft-tissue lesion with irregular margins and multiple mineralized foci (Figure 3). The lesion was heterogeneous and extended through the anterior, lateral, and posterior muscular compartments, involving the regions of the

tibialis anterior and posterior, peroneal muscles, soleus, and gastrocnemius. Fibular periosteal and cortical thickening was present. The tibia and fibula remained aligned, with no aggressive cortical destruction, medullary replacement, or pathological marrow signal. The MRI report did not describe vascular invasion, and the lesion showed no appreciable post-contrast enhancement. The integrated imaging impression favored melorheostosis with extensive extraosseous involvement.

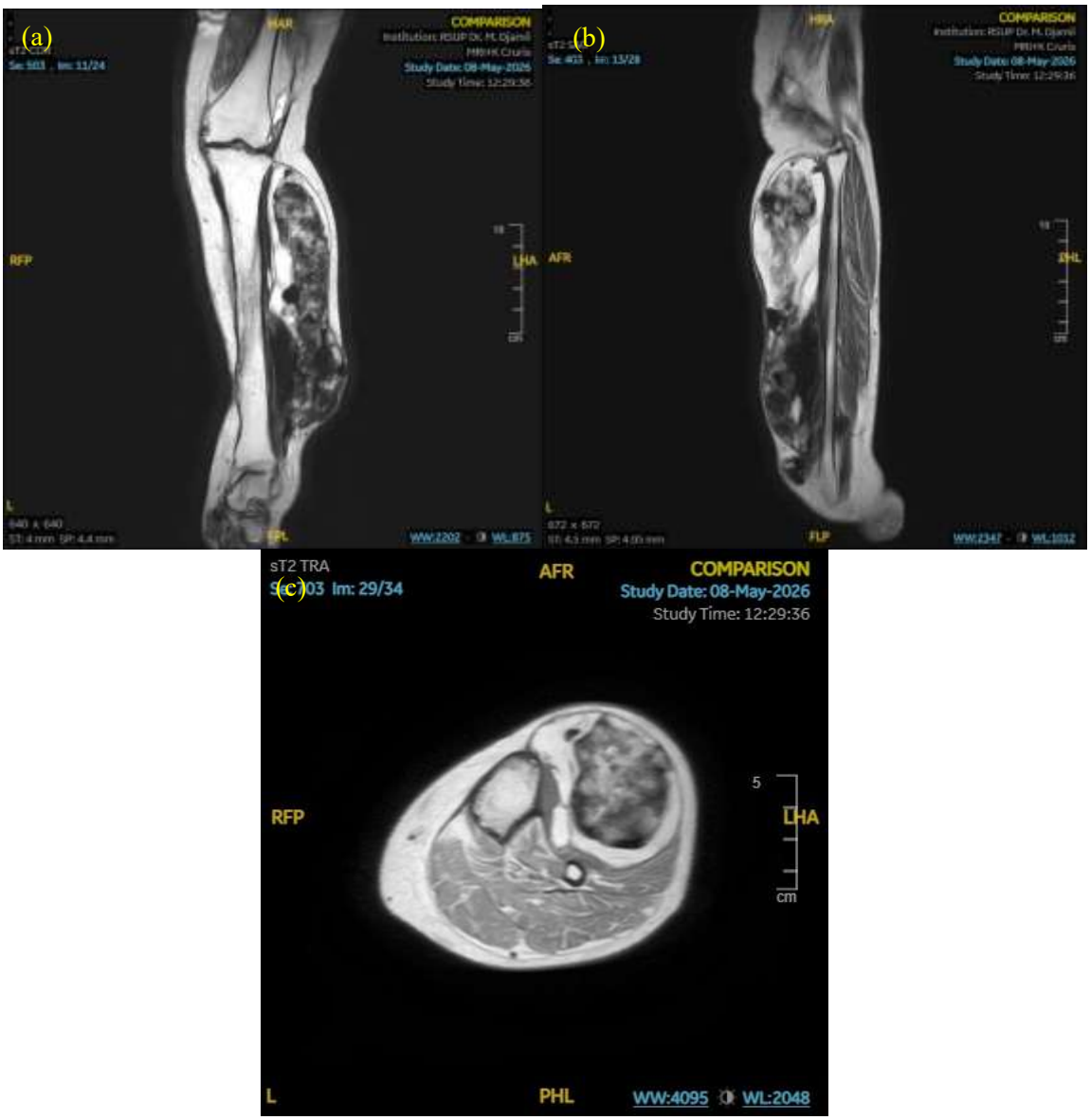

Figure 3. Coronal (a), sagittal (b), and axial (c) magnetic resonance images showing a large multicompartmental extraosseous lesion surrounding the fibula, with mineralized foci and no medullary replacement.

Complete blood count, lactate dehydrogenase, serum calcium, and alkaline phosphatase were within the laboratory reference ranges. Erythrocyte sedimentation rate and random blood glucose were elevated.

Because of progressive symptoms, skin compromise, substantial mechanical burden, and persistent diagnostic uncertainty, an extensile en bloc excision was performed. A longitudinal lazy-S incision incorporated elliptical excision of the ulcerated skin and previous biopsy tract (Figure 4). The

predominantly extraosseous ossified lesion was dissected from the surrounding compartments (Figure 5). The operative specimen measured 33 × 12 × 10 cm (Figure 6). The superficial peroneal nerve was inseparable from the mass and was therefore excised en bloc with the lesion. At gross pathology, the principal specimen labeled as a left crural bone tumor measured 30 × 12 × 6 cm and comprised a firm tan-yellow portion measuring 8 × 7 × 1.5 cm and a hard osseous portion measuring 22 × 9 × 7 cm. A separately submitted tan-yellow soft-tissue specimen measured 9 × 5 × 1 cm. Immediate postoperative radiographs were obtained (Figure 8).

On histopathologic review showed trabeculae of woven bone transitioning to sclerotic bone, with osteoblastic rimming and intervening connective-tissue stroma containing variably sized hyperemic vessels (Figure 7). The separately submitted soft-tissue specimen comprised lobulated nodules of adipocytes separated by fibrous septa and skeletal muscle and surrounded by a thin fibrous capsule, consistent with intramuscular lipoma. The pathology report concluded that the osseous findings were compatible with melorheostosis in the appropriate clinicoradiologic context, with a separate intramuscular lipoma.

Immediate postoperative radiographs demonstrated substantial removal of the extraosseous ossified mass with residual fibular cortical hyperostosis (Figure 8). Long-term wound status, sensory findings, ankle-eversion strength, functional outcome, and radiographic follow-up data were not available at the time of manuscript preparation.

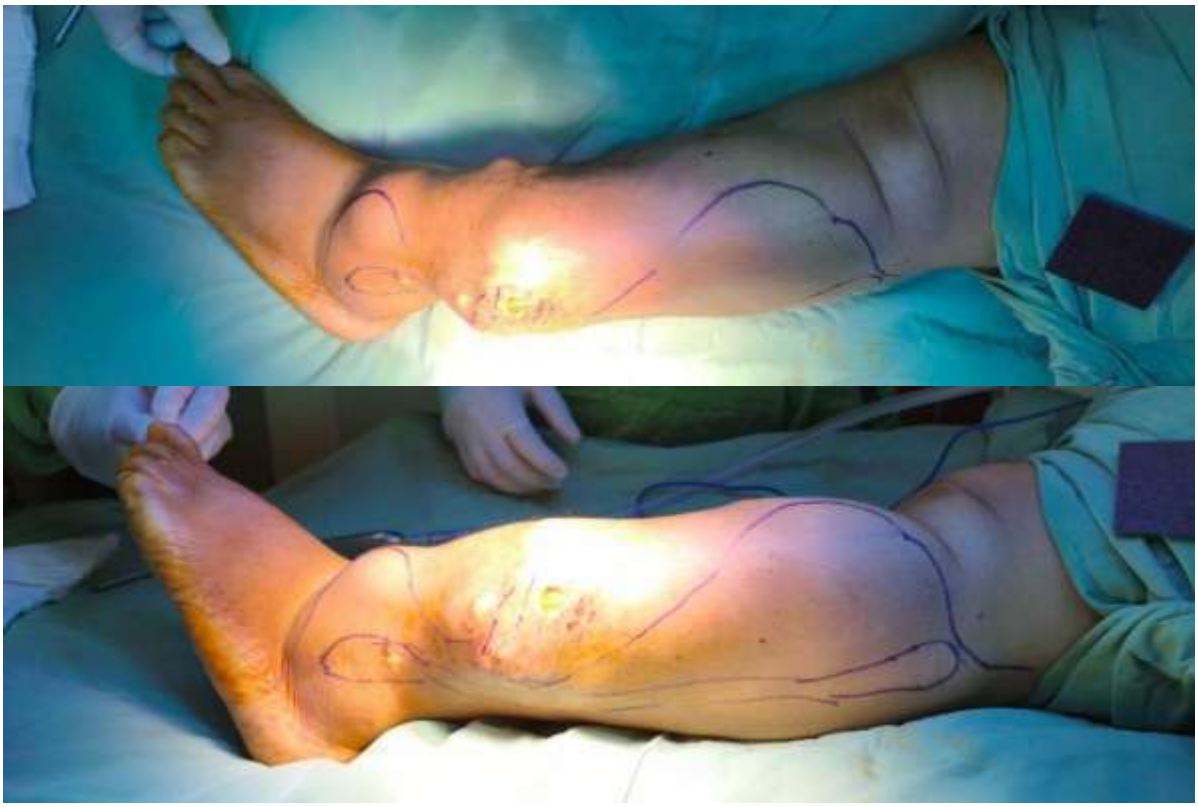

Figure 4. Preoperative lazy-S incision design shown in two projections, incorporating the ulcerated skin and previous biopsy tract.

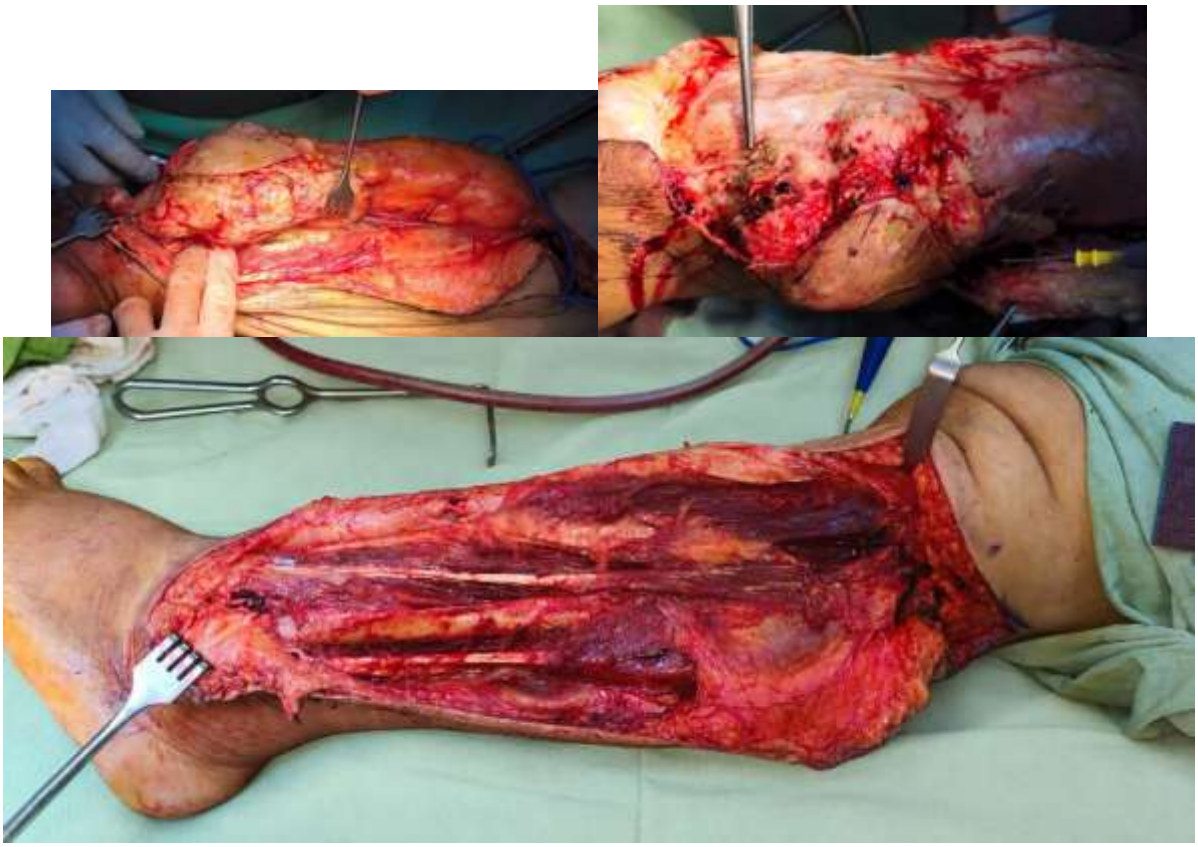

Figure 5. Intraoperative findings: exposure of the ossified lesion (upper left); the superficial peroneal nerve coursing within and inseparable from the mass (upper right); and the operative field after en bloc excision (lower).

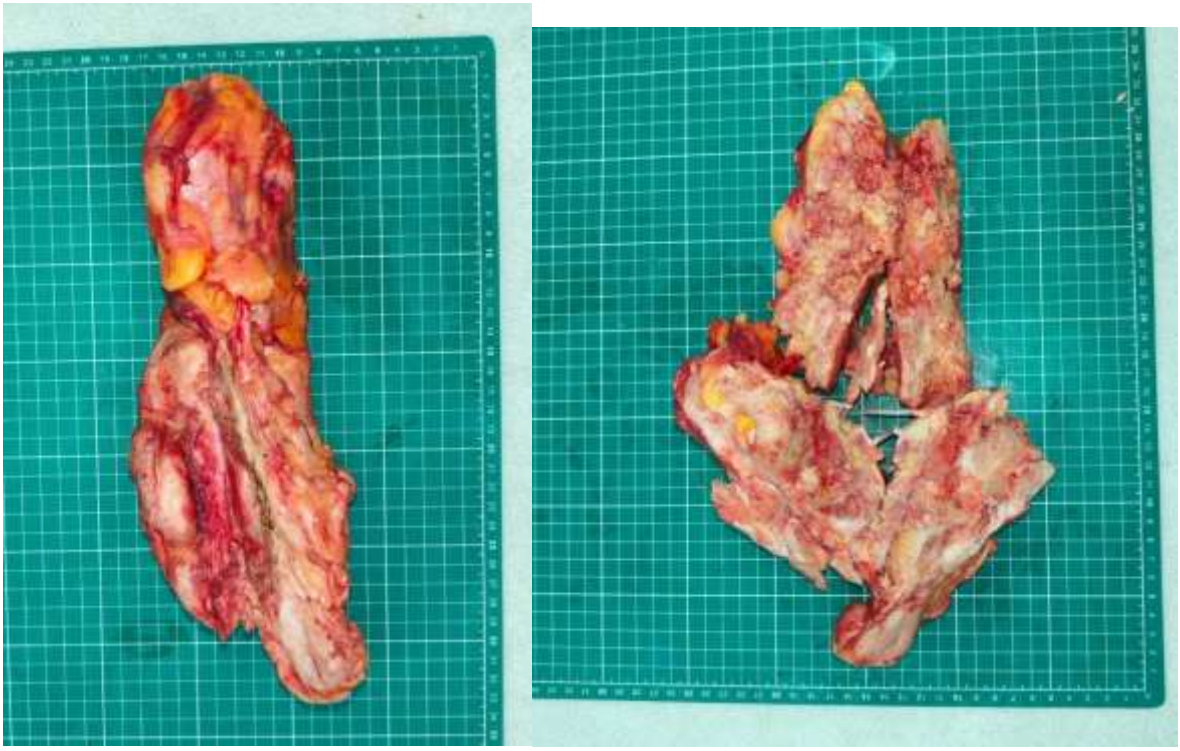

Figure 6. Gross resection specimen measuring 33 × 12 × 10 cm, shown intact and sectioned.

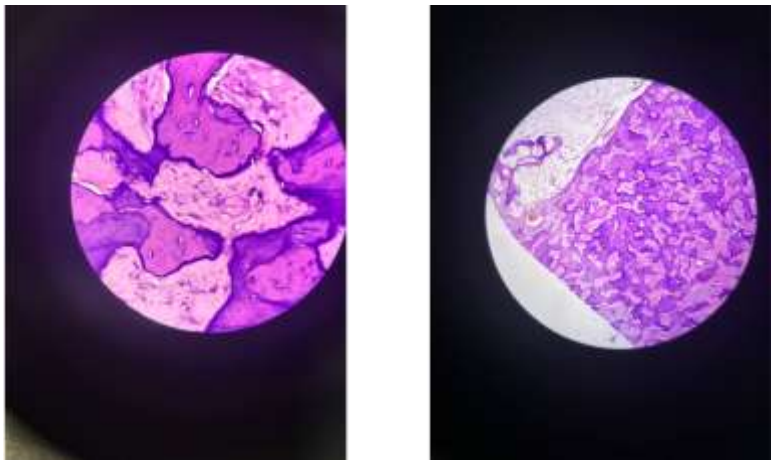

Figure 7. Representative histopathological sections. The left and right panels show woven-bone trabeculae transitioning to sclerotic bone, with osteoblastic rimming and intervening hyperemic vascular connective-tissue stroma, compatible with melorheostosis in the clinicoradiologic context.

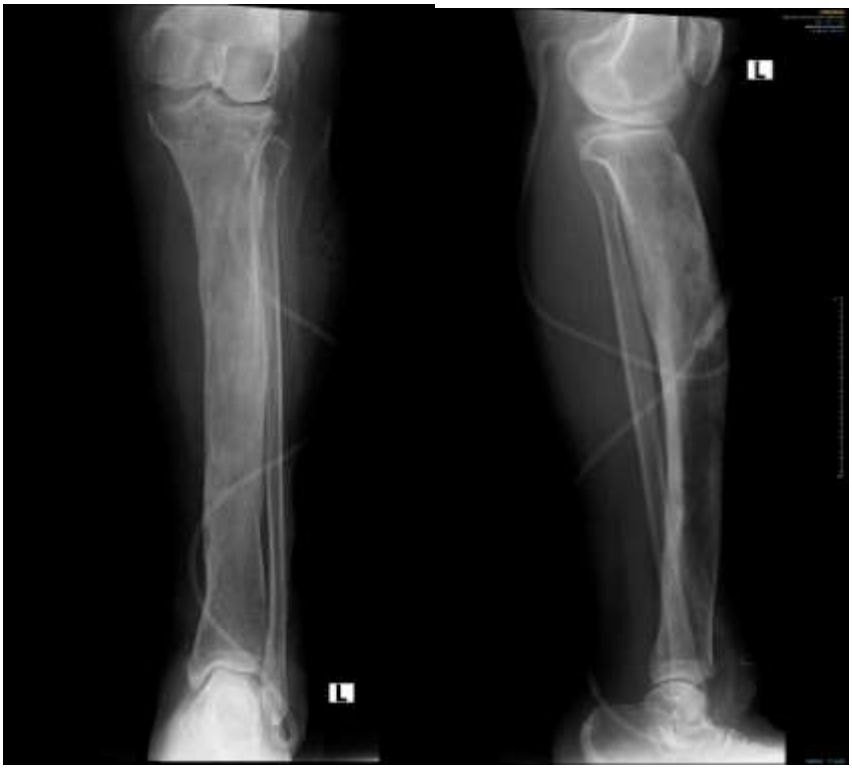

Figure 8. Immediate postoperative anteroposterior and lateral radiographs showing substantial removal of the extraosseous ossified component and residual fibular cortical hyperostosis.

DISCUSSION

This case demonstrates an unusual form of melorheostosis in which a giant extraosseous ossified mass obscured the more disease-specific finding of longitudinal flowing fibular cortical hyperostosis. Progressive enlargement, deep fixation, pain, sensory symptoms, multicompartmental extension, and skin ulceration appropriately raised concern for a bone- or cartilage-forming neoplasm. However, the three-year course and non-destructive imaging indicated relatively indolent behavior, and the diagnosis ultimately depended on clinicoradiologic–pathologic correlation rather than any single finding [3,6–9].

Radiography provided the principal diagnostic clue. The irregular longitudinal hyperostosis along the fibula resembled the classic dripping-candle-wax phenotype [2,3].

Cross-sectional imaging showed extensive contiguous periosteal and soft-tissue ossification but no aggressive cortical destruction, medullary replacement, pathological marrow signal, or appreciable enhancement. These features reduced the likelihood of an aggressive malignant process, although imaging alone cannot exclude every low-grade surface or cartilage-forming tumor [10,13].

The previous biopsy diagnosis of osteochondroma created radiologic–pathologic discordance. Conventional osteochondroma is defined by continuity of both cortex and medullary cavity with the parent bone [16]. That architecture was not demonstrated here, and osteochondroma did not explain the segmental flowing fibular hyperostosis. Sampling of a mature osseous or osteocartilaginous portion of a heterogeneous lesion is a plausible explanation, but it remains inferential without side-by-side review of the original biopsy and resection material.

This sampling limitation is biologically plausible because melorheostosis has heterogeneous and non-specific histology. Reported specimens contain variable combinations of woven and lamellar bone, dense sclerosis, osteoid, osteoblastic rimming, fibrocartilage, fibrous tissue, and increased vascularity [5,15]. Lemontzis similarly reported extraosseous melorheostosis mimicking a cartilaginous tumor, in which the overall cortical morphology and non-aggressive imaging were decisive [13]. In the present case, the osseous specimen showed active osteogenesis compatible with that spectrum, but histology alone could not establish melorheostosis. The separately submitted intramuscular lipoma was histologically distinct and should not be conflated with the osteogenic component.

The main imaging differentials for an ossified surface or soft-tissue mass included heterotopic ossification and surface bone-forming tumors. Heterotopic ossification generally develops a zonal maturation pattern and is often associated with trauma, surgery, or neurologic injury; the absence of such a history and the presence of characteristic fibular cortical flow argued against it [14]. Surface osteosarcoma or chondrosarcoma would be favored by destructive cortical or marrow invasion and corresponding malignant histology, which were not demonstrated in the available radiologic and pathologic descriptions [16].

The extraosseous component is not incompatible with melorheostosis. Mineralization and fibrosis may involve muscle, fascia, tendons, ligaments, and periarticular tissues [10–13]. The patient's paresthesia over the dorsum of the foot was anatomically concordant with the intraoperative finding that the superficial peroneal nerve was inseparable from the mass, supporting chronic neural entrapment or encasement. A previous melorheostosis report described common peroneal nerve compression at the fibular tunnel, whereas the present case involved the superficial peroneal nerve [6,9].

Somatic activating MAP2K1 variants have been associated with the classic candle-wax phenotype, increased osteoid formation, and extraosseous mineralization [4,5]. This case is phenotypically compatible with that subgroup; however, molecular data were unavailable and genotype cannot be inferred from imaging or histology.

Surgical excision was reasonable because the lesion caused progressive pain, ulceration, sensory symptoms, substantial mechanical burden, and persistent diagnostic uncertainty. Recurrence or progression of soft-tissue components has been described, so long-term clinical and radiographic surveillance is warranted [8,17]. When the superficial peroneal nerve is involved, preoperative neural mapping and postoperative documentation of dorsal-foot sensation and ankle-eversion strength are important because nerve excision may produce clinically meaningful sensory and motor morbidity [18].

This report has several limitations. Although the resection specimens underwent documented histopathologic review, the original biopsy slides were not documented as having undergone side-by-side subspecialty review with the resection material; the level and length of superficial peroneal nerve excision were unavailable; molecular testing was unavailable; and longitudinal postoperative sensory, motor, functional, and radiographic outcomes were not available. These limitations restrict conclusions regarding the mechanism of biopsy discordance, genotype, operative morbidity, and recurrence. Nevertheless, the case provides a clear diagnostic lesson: a limited biopsy should be reconsidered when it does not explain the characteristic architecture and biological behavior of the entire lesion.

CONCLUSION

Melorheostosis should be considered when a slowly progressive limb mass with extensive soft-tissue ossification coexists with segmental flowing cortical hyperostosis. In this case, characteristic fibular radiography, non-aggressive magnetic resonance findings, and compatible but non-specific osteogenic histology outweighed a discordant limited-biopsy diagnosis of osteochondroma. Whole-lesion radiologic–pathologic correlation is essential, and symptomatic lesions with skin or neural involvement may require limb-preserving excision and long-term surveillance.

Declarations by Authors

Consent for publication: Written informed consent was obtained from the patient for publication of this case report and all accompanying clinical, radiologic, operative, and gross-specimen images.

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REFERENCES

1. Léri A, Joanny J. Une affection non décrite des os: hyperostose “en coulée” sur toute la longueur d’un membre ou “mélorhéostose”. Bull Mem Soc Med Hop Paris. 1922; 46:1141–1145.
2. Freyschmidt J. Melorheostosis: a review of 23 cases. Eur Radiol. 2001;11(3):474–479. doi:10.1007/s003300000562.
3. Kotwal A, Clarke BL. Melorheostosis: a rare sclerosing bone dysplasia. Curr Osteoporos Rep. 2017;15(4):335–342. doi:10.1007/s11914-017-0375-y.
4. Kang H, Jha S, Deng Z, Fratzl-Zelman N, Cabral WA, Ivovic A, et al. Somatic activating mutations in MAP2K1 cause melorheostosis. Nat Commun. 2018;9(1):1390. doi:10.1038/s41467-018-03720-z.
5. Jha S, Fratzl-Zelman N, Roschger P, Papadakis GZ, Cowen EW, Kang H, et al. Distinct clinical and pathological features of melorheostosis associated with somatic MAP2K1 mutations. J Bone Miner Res. 2019;34(1):145–156. doi:10.1002/jbmr.3577.
6. Jha S, Cowen EW, Lehky TJ, Alter K, Flynn L, Reynolds JC, et al. Clinical evaluation of melorheostosis in the context of a natural history clinical study. JBMR Plus. 2019;3(8):e10214. doi:10.1002/jbm4.10214.
7. Smith GC, Pingree MJ, Freeman LA, Matsumoto JM, Howe BM, Kannas SN, et al. Melorheostosis: a retrospective clinical analysis of 24 patients at the Mayo Clinic. PM R. 2017;9(3):283–288. doi: 10.1016/j.pmrj.2016.07.530.
8. Bhattacharyya T. Unraveling melorheostosis: insights into clinical features, diagnosis, and treatment. JBMR Plus. 2025;9(2): ziae163. doi:10.1093/jbmrpl/ziae163.
9. Puffer RC, Sabbag OD, Logli AL, Spinner RJ, Rose PS. Melorheostosis causing compression of common peroneal nerve at fibular tunnel. World Neurosurg. 2019; 128:1–3. doi: 10.1016/j.wneu.2019.04.208.
10. Hurley-Novatny A, Karantanas AH, Papadakis GZ, Bhattacharyya T, Jha S. Cross-sectional imaging useful in melorheostosis. JBMR Plus. 2021;5(4): e10472. doi:10.1002/jbm4.10472.
11. Gachhayat A, Mishra D, Gulia A, Nanda SN, Preethiv R, Mohanty SA. A rare case of

melorheostosis presenting with extra-osseous lesions around the knee: case report. J Orthop Case Rep. 2023;13(5):39–43. doi:10.13107/jocr. 2023.v13.i05.3638.
12. Papadakis GZ, Jha S, Bhattacharyya T, Millo C, Tu TW, Bagci U, et al. 18F-NaF PET/CT in extensive melorheostosis of the axial and appendicular skeleton with soft-tissue involvement. Clin Nucl Med. 2017;42(7):537–539. doi:10.1097/RLU.0000000000001647.
13. Lemontzis O. Melorheostosis with extraosseous extension mimicking a cartilaginous tumor: a case report. J Belg Soc Radiol. 2025;109(1):52. doi:10.5334/jbsr.4139.
14. Mujtaba B, Taher A, Fiala MJ, Nassar S, Madewell JE, Hanafy AK, et al. Heterotopic ossification: radiological and pathological review. Radiol Oncol. 2019;53(3):275–284. doi:10.2478/raon-2019-0039.
15. Fick CN, Fratzl-Zelman N, Roschger P, Klaushofer K, Jha S, Marini JC, et al. Melorheostosis: a clinical, pathologic, and radiologic case series. Am J Surg Pathol. 2019;43(11):1554–1559. doi:10.1097/PAS.0000000000001310.
16. WHO Classification of Tumours Editorial Board. Soft tissue and bone tumours. 5th ed. Vol. 3. Lyon: International Agency for Research on Cancer; 2020.
17. Hasegawa S, Kanda S, Imada H, Yamaguchi T, Akiyama T. Melorheostosis with recurrent soft-tissue components: a histologically confirmed case. Skeletal Radiol. 2017;46(3):399–404. doi:10.1007/s00256-016-2562-9.
18. Fortier LM, Markel M, Thomas BG, Sherman WF, Thomas BH, Kaye AD. An update on peroneal nerve entrapment and neuropathy. Orthop Rev (Pavia). 2021;13(2):24937. doi:10.52965/001c.24937.

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