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Case report

Primary intraosseous Rosai Dorfman Disease: Case series with review of literature

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Keywords:

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ABSTRACT

Rosai-Dorfman disease is a rare form of non-Langerhans cell histiocytosis of uncertain etiology, typically presenting as nodal lesions, with osseous involvement being uncommon. We retrospectively analyzed four intraosseous Rosai-Dorfman disease cases among 1,064 bone biopsies received between April 2023 and October 2024. All participants were young adults (mean age: 24 years), predominantly female, with solitary lytic lesions, most commonly located in the femur and tibia. Histology revealed histiocytes amidst lymphoplasmacytic infiltrates, neutrophils, and marrow fibrosis; emperipolesis was inconspicuous in two cases. Immunohistochemistry showed S100, CD68, CD163, and Cyclin D1 positivity; CD1a was negative. All patients underwent curettage (with or without bone grafting) and remained disease-free at follow-up. Rosai-Dorfman disease should be considered in young adults with lytic bone lesions; diagnosis requires careful histological and immunohistochemical evaluation.

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INTRODUCTION

Rosai-Dorfman disease (RDD) is a rare histiocytic disorder of unknown etiology, typically affecting lymph nodes, particularly in the neck.[1] Extranodal involvement occurs in 33–43% of cases, with bone affected in <10%.[2] RDD can present at any age with variable patterns—isolated nodal, combined nodal-extranodal, or purely extranodal without lymphadenopathy.[3] Diagnosis in bone and soft tissue is challenging due to its rarity, nonspecific imaging, and histology, often lacking prominent emperipolesis.[2] Small biopsies further complicate recognition. Given the limited literature on its clinicopathological spectrum, our study aims

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to expand current knowledge. We retrospectively analyzed four intraosseous RDD cases among 1,064 bone biopsies at Ganga Medical Center and Hospital, Mettupalayam Road, Coimbatore, Tamil Nadu, India, received between April 2023 and October 2024. Clinical and imaging data were retrieved from records; histopathology and immunohistochemistry slides were reviewed. Treatment and follow-up outcomes were also evaluated. This study highlights the diagnostic challenges and emphasizes the need for a high degree of clinical suspicion and thorough histopathological evaluation to differentiate RDD from its mimics.

CASE SERIES:

CASE- 1: A 24-year female presented with a 2-month history of dull, progressively worsening left leg pain, localized, with severe tenderness to the mid-diaphysis of the tibia. Systemic review and history were negative for fever or trauma, though inflammatory markers (ESR, CRP) were mildly elevated. The anteroposterior and lateral radiographs of the left lower limb demonstrated an expansile, well-circumscribed lytic lesion situated at the intramedullary aspect in the mid-diaphysis of the left tibia. MRI revealed a well-defined, mildly expansile lytic intramedullary lesion (4.5×1.4 ×1.3cm) with endosteal scalloping, cortical thinning, and irregular periosteal reaction, raising differentials of Langerhans cell histiocytosis or round cell tumors such as lymphoma or Ewing sarcoma. C-arm–guided needle biopsy of the lesion was performed. Histopathological examination revealed a heterogeneous infiltrate of histiocytes, plasma cells, lymphocytes, and neutrophils. Some histiocytes showed features of emperipolesis. No granulomas or atypical cells were observed. Based on histomorphology, a histiocytic lesion, probably Langerhans cell histiocytosis (LCH) or RDD, was considered. However, acute and chronic osteomyelitis were also considered as differentials. Immunohistochemical analysis revealed diffuse CD163 and patchy S100 positivity in the histiocytes, with negative CD1a expression, thus excluding LCH. Following discussion in a multidisciplinary tumor board, the patient underwent intralesional extended curettage. Histopathological analysis of the curetted specimen revealed a florid, heterogeneous infiltrate comprising sheets of histiocytes along with numerous plasma cells, lymphocytes, and neutrophilic microabscesses. Emperipolesis was well evident, with histiocytes containing engulfed neutrophils, lymphocytes, and plasma cells. Special stains, PAS stain, and Ziehl-Neelsen stain were negative for fungus and acid-fast bacilli, respectively. The microbial culture was negative for any organisms. These histomorphological findings, along with negative culture results, supported the diagnosis of Rosai-Dorfman disease. PET-CT was performed to look for any lymph node enlargement, as RDD is primarily a nodal disease. However, no abnormal findings were detected. At 2 years and six months follow-up, the patient remained symptom-free and ambulatory.

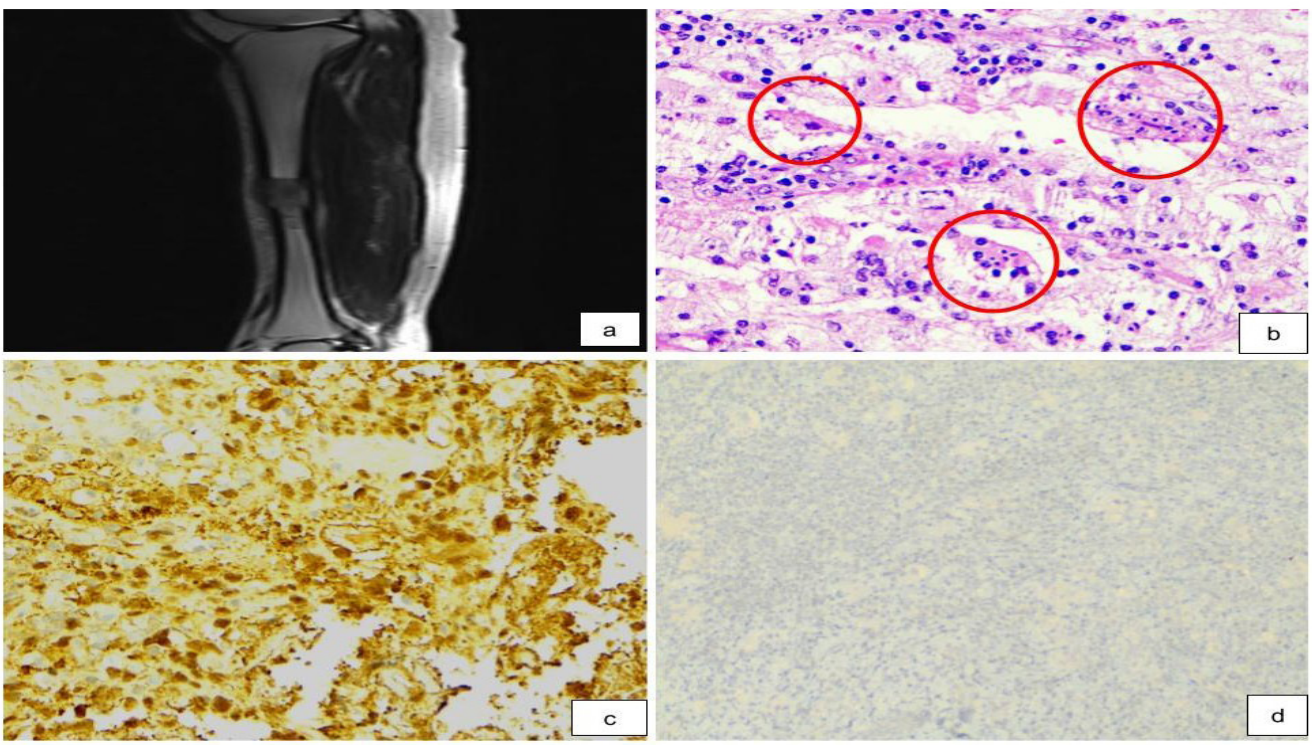

Figure 1: *(a) MRI Left Leg showing well-defined, mildly expansile lytic intramedullary lesion in mid-diaphysis of tibia; (b) Emperipolesis with mixed inflammation (HE stain; 20X); (c) S100 positivity(IHC x 40X) ; (d) Negative for CD1a (IHC; 10X)*

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CASE-2: A 22-year female presented with a 3-month history of gradually progressive right hip pain and a limp. Laboratory evaluation revealed mild leukocytosis and elevated inflammatory markers. The anteroposterior radiograph of the pelvis demonstrated an aggressive, expansile lytic lesion of size 5.5 x 5 x 4 cm involving the head and neck of the right femur. Pelvic MRI demonstrated an aggressive moth-eaten pattern of bone destruction involving the entire femoral head and neck, with cortical breach and minimal soft tissue extension, raising strong suspicion for aggressive malignancy like chondrosarcoma or round cell tumor. A biopsy of the femoral head and neck was performed. Histopathological examination revealed lamellar bone with intervening marrow spaces infiltrated by sheets of foamy histiocytes, plasma cells, and lymphocytes, along with focal fibrosis. Crushed cells and a few binucleated plasma cells were also present. Based on histomorphological findings, hematolymphoid lesions, probably a histiocytic lesion or lymphoma, were considered with no evidence of chondroid or pleomorphic cells. Other differentials included plasma cell neoplasm and IgG4 disease. Immunohistochemistry showed that histiocytes expressed S-100, CD163, and CD68, while CD1a was negative. CD20 highlighted the scattered lymphocytes. Due to persistent clinical suspicion

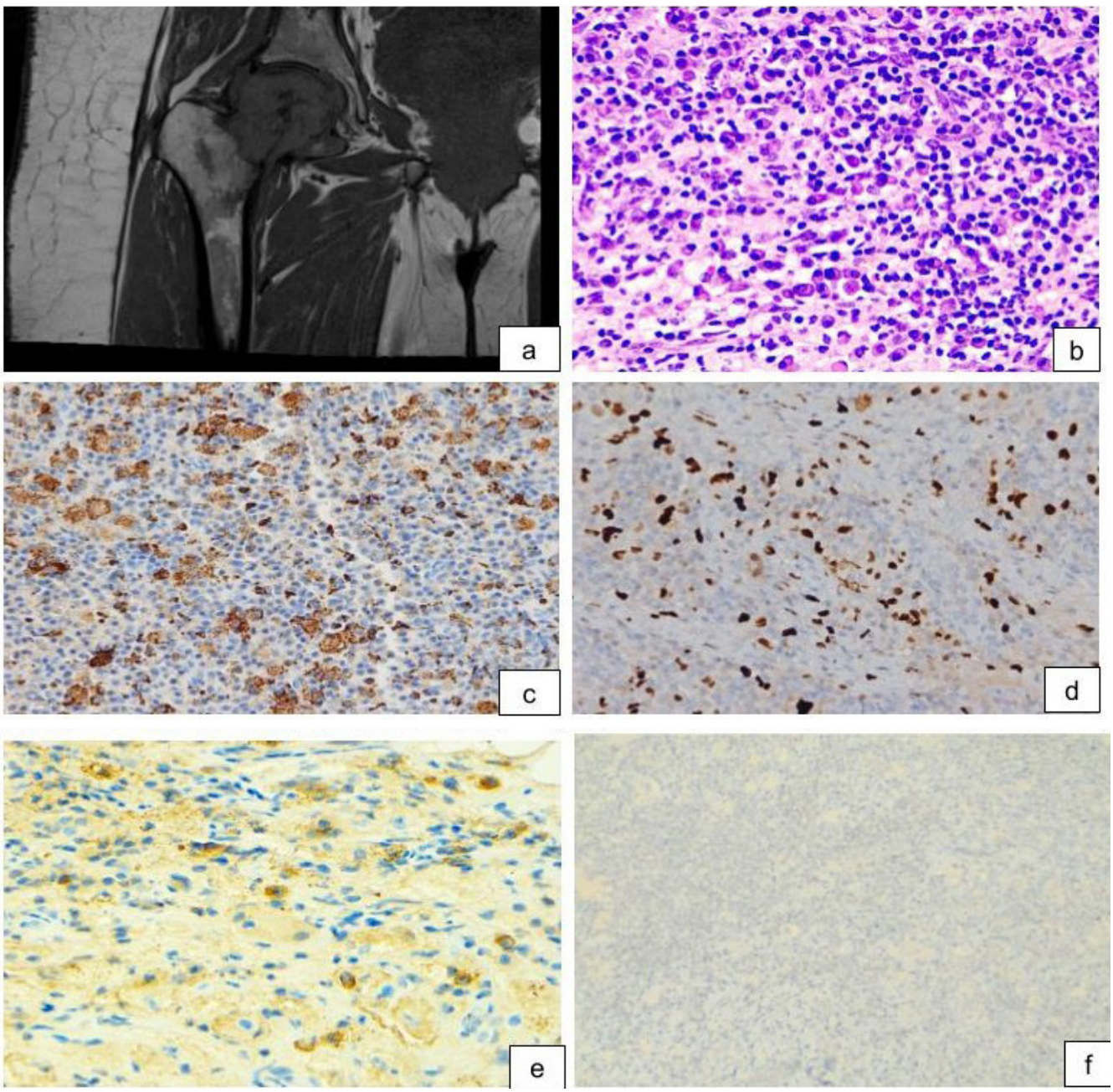

Figure 2: *a) MRI Pelvis: Osteolytic lesion involving the head and neck of the right femur; (b) Mixed inflammation (HE stain; 40X); (c) Histiocytes showing CD68 cytoplasmic positivity (IHC; 20X);(d) Cells showing cyclin D1 nuclear positivity (IHC; 20X);(e) No increase in IgG4-positive cell (IHC; 20X); (f) Lesional cells were negative for LCA (IHC; 10X).*

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of malignancy, a repeat CT-guided biopsy was performed. Histopathology revealed a linear core of bone infiltrated by atypical medium to large cells with oval to polygonal shapes, moderate eosinophilic to foamy cytoplasm, irregular nuclear membranes, vesicular nuclei, and occasional nuclear indentations and grooves. Numerous mature plasma cells and lymphocytes were present. Occasional histiocytes demonstrating inconspicuous emperipolesis were evident on diligent search. Mitoses were sparse, and necrosis was absent. Immunohistochemistry revealed diffuse nuclear and cytoplasmic positivity for S-100, CD68, and CD163 in the large cells, confirming a histiocytic lineage. The large cells were negative for LCA, CD99, synaptophysin, CD1a, and PanCK, thus ruling out lymphoma, small round cell tumor, neuroendocrine tumor, LCH, and metastatic carcinoma, respectively. Plasma cells were polyclonal (expressing both kappa and lambda light chains), and there was no increase in IgG4-positive cells, excluding plasma cell neoplasm and IgG4-related disease. The Ki-67 proliferation index was 5–7%. Based on the above IHC findings with inconspicuous emperipolesis, RDD was strongly suspected, and Cyclin D1 was performed to support the diagnosis. Given the atypical location and presentation, further investigations like biopsy of inguinal/pelvic lymph nodes (as identified on PET-CT) and comprehensive molecular workup for diagnostic confirmation were recommended. A biopsy from the inguinal lymph node was done, which was found to be reactive. Hence, a final diagnosis of Primary RDD of right proximal femur was made. The patient was treated with an intralesional corticosteroid injection and remains symptom-free and ambulatory at one year and five months post-operative follow-up, with no clinical or radiological evidence of local recurrence or development of new nodal or extranodal lesions.

CASE 3: A 26-year female presented with a 6-month history of progressively worsening pain and swelling over the left ankle. X-ray of left ankle showed an expansile lytic lesion in

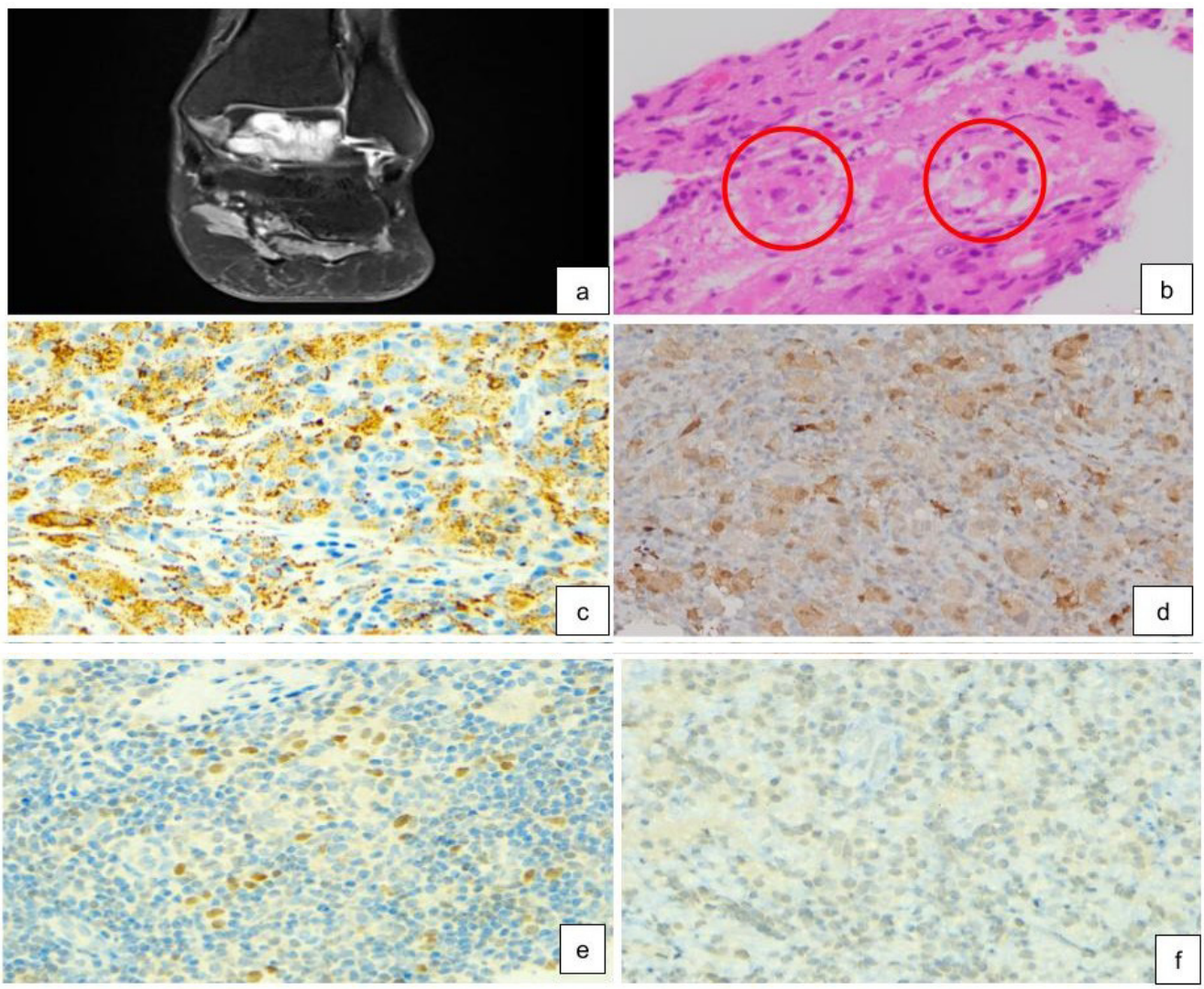

Figure 3: *Figure 3:(a) MRI Left Ankle showing lytic lesion involving the entire posterior third of the talus from talar dome to the posterior subtalar sub-articular surface; (b)Mixed inflammation with scattered emperipolesis (H&E stain; 40X); (c) Cells showing CD68 cytoplasmic positivity (IHC; 20X); (d) S100 cytoplasmic and nuclear positivity (IHC; 20X); e) Occasional cells show OCT-2 nuclear positivity (IHC; 20X); (f) Negative for CD1a (IHC; 10X)*

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the talus, and MRI revealed an expansile lytic lesion of size 3 x 2x 1.4 cm occupying the dome and posterior subarticular surface of the left talus, without intra-articular extension. Routine blood work, including inflammatory markers, was within normal limits. Based on the clinical and radiological findings, the patient underwent extended intralesional curettage with allograft placement in the left talus. Histopathological analysis of the curetted specimen showed fragments of lamellar bone interspersed with marrow tissue containing dense aggregates of chronic inflammatory cells, mainly lymphocytes and plasma cells. Multiple foci revealed sheets of foamy histiocytes and scattered large histiocytic cells with abundant eosinophilic cytoplasm, indistinct cell borders, and hyperchromatic nuclei. Some cells were binucleated with vesicular nuclei and prominent nucleoli. Notably, several histiocytes demonstrated emperipolesis—containing intracytoplasmic neutrophils and plasma cells. Immunohistochemistry showed that the histiocytes were positive for S100, CD68, and Cyclin D1. Occasional cells were positive for OCT2, which further supported the diagnosis in challenging cases. They were also negative for CD1a, thus ruling out LCH. IgG and IgG4 staining revealed no increase in IgG4-positive plasma cells, thus ruling out IgG4 disease. Further evaluation was performed to assess for lymph nodal or other site involvement, but no abnormal findings were identified. The patient was symptom-free and ambulatory at one year and four months postoperative follow-up.

CASE 4: A 24-year male presented with intermittent left knee pain for one week, exacerbated by sitting, with a history of trauma one year back. Local examination revealed mild bony tenderness. The anteroposterior and lateral radiographs of the left knee demonstrated a lytic lesion in the lateral femoral condyle of the left femur. MRI and CT showed an ill-defined, altered signal intensity area with an eccentric lucent lytic lesion of 4x3x1 cm in the posterolateral aspect of the lateral femoral condyle, associated with significant cortical thinning and post-contrast enhancement. Radiological differentials included Langerhans cell histiocytosis, chondroid tumor, and giant cell tumor (GCT). Considering the patient's age and imaging findings, a C-arm guided biopsy of the lesion was performed. Histopathological examination revealed multiple tissue fragments with areas of hemorrhage, degenerated bone, and fibrocollagenous stroma infiltrated by sheets of inflammatory cells, including histiocytes, plasma cells, lymphocytes, and neutrophils. The histiocytes had abundant eosinophilic cytoplasm, ill-defined borders, and round hyperchromatic nuclei. Emperipolesis—the presence of lymphocytes and plasma cells within the cytoplasm of histiocytes was observed. No atypical or foreign cells were identified. Immunohistochemical staining demonstrated positivity for CD163 and S100 in the histiocytes, with CD1a negativity. Occasional cells exhibited weak nuclear Cyclin D1 positivity. These findings were consistent with Rosai-Dorfman disease. A thorough evaluation was conducted for lymph nodes and other lesions, but no significant findings were detected. The patient was symptom-free and ambulatory at one year and one month postoperatively.

Table 1: Summary of individual case

Case	Age	Gender	Presenting complaints	Site	CT/ MRI Findings	Histopathology findings	IHC	Treatment offered	Outcome & follow-up
Patient 1	24 yrs	Female	Dull aching pain – 2 months	Left mid-diaphysis of tibia	Expansile lytic intramedullary soft tissue lesion with cortical thinning and irregular periosteal reaction. Size: 4.5 × 1.4 × 1.3 cm Imaging differential: LCH, lymphoma, Ewing sarcoma	Heterogeneous infiltrate of histiocytes, plasma cells, lymphocytes, neutrophilc microabscesses. Emperipolesis and fibrosis present. Suggestive of LCH /RDD or acute and chronic osteomyelitis. (Special stains and culture- Negative)	CD163+ S100+ CD1a-	Curettage with bone graft	Recovered well; symptom-free at 2 yrs and 6 months follow-up.
Patient 2	22 yrs	Female	Right hip pain – 3 months, limping	Right proximal femur	Moth-eaten pattern of bone destruction involving entire femoral head with cortical breach. Size: 5.5 × 5 × 4 cm Imaging differential: Aggressive neoplasm-chondrosarcoma, round cell tumor	Histiocytes,plasma cells, lymphocytes,few RS-like cells. Inconspicuousemperipolesis. Suggestive of histiocytic lesion, lymphoma, plasma cell neoplasm, IgG4-related disease.	CD163+ CD68+ S100+ CyclinD1+ CD1a- LCA- Polyclonal plasma cells. No increase in IgG4-positive cells.	Curettage	Recovered well; symptom-free at 1 yr 5 months postoperatively.

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Case	Age	Gender	Presenting complaints	Site	CT/ MRI Findings	Histopathology findings	IHC	Treatment offered	Outcome & follow-up
Patient 3	26 yrs	Female	Pain & swelling – 6 months	Left talus	Lesion seen on talar dome and posterior subarticular surface. Size: 3 × 2 × 1.4 cm. Imaging differential: LCH.	Histiocytes,plasma cells, lymphocytes, microabscesses. Emperipolesis, fibrosis, and perivascularplasma cell infiltrate present.	CD68+ S100+ Cyclin D1+ Oct-2+ CD1a- No increase in IgG4-positive cells	Extended curettage and bone grafting	Recovered well; symptom-free at 1 yr 4months postoperatively.
Patient 4	24 yrs	Male	Left knee pain – 1 week	Left lateral femoral condyle	Ill-defined signal changes and eccentric lytic lucent areas. Size: 4 × 3 × 1 cm Imaging differential: LCH, chondroid tumor, GCT	Histiocytes,plasma cells, lymphocytes, neutrophil microabscesses. Emperipolesis present.	CD163+ S100+ CyclinD1+ CD1a-	Curettage	Recovered well; symptom-free at 1yr 1 month postoperatively.

DISCUSSION

RDD is a rare disorder of uncertain pathogenesis characterized by proliferation of large histiocytes with intact intracytoplasmic leukocytes (emperipolesis) and a mixed inflammatory infiltrate.[1] It is now classified as a distinct subtype—"R group"—among histiocytic disorders.[2] The Histiocyte Society categorizes RDD into sporadic (non-cutaneous), familial, and cutaneous forms.[4] Originally described in children and young adults with painless, bilateral cervical lymphadenopathy,[4] RDD is now recognized in extranodal sites, including skin, soft tissues, bones, CNS, and viscera.[5]

Etiology remains uncertain, though MAPK pathway mutations and cyclin D1 activation have been implicated.[5] Solitary bone lesions without nodal disease are rare, with ~100 cases reported (Table 2). Our cohort (3 females, 1 male) mirrors the female predominance reported by Demicco et al. in 15 cases.[6] Similar trends were seen in other studies, though small series have shown male predominance.[5,7] Our patients (mean age: 24 years) presented with pain, as consistently noted in the literature. Commonly involved bones include tibia, femur, and fibula;[6,7] our cases involved femur (2), tibia (1), and talus (1). Talus involvement is exceedingly rare, with some cases linked to autoimmune RDD with arthritis.[8]

Table 2: Clinico-radiological characteristics of published cases on Rosai-Dorfman Disease (RDD) of bone

	Present study	Demicco et al.[6]	Dong et al.[7]	Weng et al.[5]	Other case reports[9,12,13]
Total no. of patients	4	15	14	4	1,2,1
Study duration (years)	1.5	-	10	10	-
Mean age	24	27	31	25	299; 76, 2012; 4813
M:F ratio	1:3	7:8	8:6	3:1	F9; 1:1; M13
Most common site	Femur	Proximal tibia	Tibia	Skull and vertebra	Medial femoral condyle9; Proximal and distal radius12; Temporal bone13
Average size	4 cm	-	-	-	-
Common clinical symptom	Pain	Pain	Pain	Pain	Pain and swelling (9); Pain (12); Otalgia and vertigo (13)
Average duration of onset	2.81 months			2.75 months	2 month; 2yr, 1yr (12); 18 month
Radiological diagnosis	LCH, high-grade chondroid tumor, GCT, round cell tumor	Metastatic disease, osteomyelitis, EG, GCT, lymphoma, fibrous dysplasia, CMF, enchondroma, TB, Ewing sarcoma	Lytic lesion with sclerotic margins	Osteomyelitis, LCH, Erdheim–Chester, lymphoma, plasma cell myeloma, metastases	GCT, LCH, lymphoma (9); Metastases, myeloma (12)
Multifocal lesions	NIL	Two patients (Tibia and humerus; Femur and humerus)	One patient (Vertebrae and nasal cavity)	NIL	NIL

GCT = Giant Cell Tumor; LCH = Langerhans Cell Histiocytosis; EG = Eosinophilic Granuloma; CMF = Chondromyxoid Fibroma; TB = Tuberculosis.

TABLE 3: Comparative Analysis of Published Cases on Rosai-Dorfman Disease (RDD) of Bone-

	OUR STUDY	Demicco et al.[6]	Dong et al.[7]	Weng et al.[5]	OTHER CASE REPORTS.[9,12,13]
Histopathological features	Mixed inflammation, emperipolesis, neutrophilic microabscess, fibrosis	Mixed inflammation, emperipolesis, neutrophilic microabscess, sclerosis	Mixed inflammation, emperipolesis, vasculitis, increased plasma cells	Mixed inflammation, emperipolesis, fibrosis, increased plasma cells	Florid histiocytosis exhibiting emperipolesis(9,12); lymphoplasmacytosis with emperipolesis(13)
IHC	S100+, CD 68+, CD163+, CyclinD1+	S100+ CD68+ CD1a-	S100+, CD68+, CD163+, CyclinD1+ CD 1a CD 207+ in one case	S100+, CD68+, OCT2+, CyclinD1+, Langerin-, CD1a-; IgG4/IgG > 40% (one case).	S100+, CD68+ CD1a-
Treatment	Curettage & Surgical resection	Curettage	Surgical resection	Surgical resection	Curettage (9,12); Cortical mastoidectomy(13)
Follow-up	Disease free (1yr- 2yr 6 months)	12 patients followed: 5 had recurrence or new lesions (2+ months); others disease-free (10–106 months)	12 followed: 3 recurrences; others disease-free (9–49 months)	Disease free (2-65 months)	Disease-free 6 wks–1 yr (9); 1–8 wks (12); 1 wk–6 months (13)

GCT = Giant Cell Tumor; LCH = Langerhans Cell Histiocytosis; EG = Eosinophilic Granuloma; CMF = Chondromyxoid Fibroma; TB = Tuberculosis.

Radiologically, all lesions were lytic, consistent with prior reports.[7] Differential diagnoses included LCH, chondroid tumors, giant cell tumors, and round cell tumors. Imaging features often mimic osteomyelitis, Erdheim-Chester disease (ECD), lymphoma, plasma cell myeloma, fibrous dysplasia, and Ewing sarcoma.[5,6] Diagnosis relied on histopathology and IHC, as inflammation and fibrosis may obscure emperipolesis, sometimes necessitating repeat biopsy. One case had neutrophilic microabscesses; another revealed emperipolesis only on a second biopsy. Histologic findings include trabecular bone disruption, histiocytic proliferation, fibrosis, plasma cell infiltrates, and sometimes vasculitis or necrotic foci.[6,7]

IHC showed S100 and CD163 positivity with CD1a negativity, confirming RDD and excluding LCH. CyclinD1 was focally positive in some cases, as seen in previous series. One reported case had CD1a and CD207 co-positivity, indicating possible RDD-LCH overlap.[7]

All patients underwent surgical curettage with bone grafting; one also received intralesional steroids. No recurrences or systemic involvement were observed. Some series reported recurrence or multi-organ disease.[7] Our findings highlight the diagnostic challenge of isolated skeletal RDD, particularly in unusual sites like the talus. Radiologic resemblance to neoplastic lesions mandates histopathological confirmation.[9] Emperipolesis and IHC profile (S100+/CD1a-) are diagnostic hallmarks.

Differentiation from LCH, ECD, osteomyelitis, and IgG4 related disease is crucial due to differing management and prognosis.[5] LCH affects all ages but is more common in children, with lytic bone lesions and possible systemic symptoms.[6] Histology reveals Langerhans-type cells with nuclear grooves and eosinophilic infiltrates. IHC shows CD1a+, Langerin+, and S100+ cells; CD68 is variable.[9] BRAF V600E mutation is seen in 50–60% of cases, rare in RDD.[1]

ECD, a non-Langerhans histiocytosis in middle-aged adults, presents with bilateral osteosclerosis, systemic symptoms, and foamy histiocytes. S100 and CD1a are negative; CD68 and CD163 are positive. BRAF V600E mutations are found in ~50%.[10]

Osteomyelitis presents with pain, fever, and radiologic features like periosteal reaction and cortical destruction. Acute forms show neutrophilic infiltrates; chronic cases show fibrosis and sequestra. No emperipolesis or histiocytic proliferation is seen.[1,5]

IgG4-related disease is a fibroinflammatory condition involving multiple organs, rarely the bone.[11] Histology shows dense plasma cell infiltrates, storiform fibrosis, and obliterative phlebitis. IgG4+ plasma cells are abundant, with an IgG4/IgG ratio >40%. S100 is negative; emperipolesis is absent.[5] Some RDD cases may show IgG4+ cells despite normal serum IgG4.[5] Comprehensive clinicopathologic correlation is needed to differentiate IgG4-RD from autoimmune-associated RDD.

Fibrosis in bone lesions may mimic malignancy or metastases. Hence, IHC in an appropriate clinical context is essential for accurate diagnosis.[1]

CONCLUSION

Intraosseous RDD poses diagnostic challenges due to subtle histologic features and overlap with other entities. Key distinguishing features include younger age, lytic lesions, emperipolesis, and S100 positivity. Careful evaluation for

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emperipolesis and awareness of RDD's diagnostic pitfalls are essential for accurate diagnosis and management, underscoring the need for multidisciplinary vigilance in histiocytic bone lesions.

Conflicts of interest: None

REFERENCES

1. Garcia RA, DiCarlo EF. Rosai-Dorfman disease of bone and soft tissue. Arch Pathol Lab Med. 2022;146(1):40–6.DOI: https://aplm.kglmeridian.com/view/journals/arpa/146/1/article-p40.xml
2. Adam R, Harsovescu T, Tudorache S, et al. Primary bone lesions in Rosai-Dorfman disease, a rare case and diagnostic challenge: case report and literature review. Diagnostics (Basel). 2022;12(4):783. DOI: https://pubmed.ncbi.nlm.nih.gov/35453831
3. Mosheimer BA, Oppl B, Zandieh S, et al. Bone Involvement in Rosai-Dorfman Disease (RDD): a Case Report and Systematic Literature Review. Curr Rheumatol Rep. 2017;19(5):29. DOI: https://pmc.ncbi.nlm.nih.gov/articles/PMC5388731
4. Natkunam Y, Rossi S, Demicco EG, Zelger BWH, Wenig BM, Idbaih A. Rosai-Dorfman disease. In: WHO Classification of Tumours Editorial Board, editors. Soft Tissue and Bone Tumours. 5th ed. Lyon, France: International Agency for Research on Cancer; 2020.660–2.
5. Weng X, Yang Y, Zhang M, et al. Primary intraosseous Rosai-Dorfman disease: an analysis of clinicopathologic characteristics, molecular genetics, and prognostic features. Front Oncol. 2022;12:950114. DOI: https://pmc.ncbi.nlm.nih.gov/articles/PMC9520307
6. Demicco EG, Rosenberg AE, Björnsson J, Rybak LD, Unni KK, Nielsen GP. Primary Rosai-Dorfman disease of bone: a clinicopathologic study of 15 cases. Am J Surg Pathol. 2010;34(9):1324–33. DOI: https://journals.lww.com/ajsp/abstract/2010/09000/primary_rosai_dorfman_disease_of_bone__a.12.aspx
7. Dong RF, Gong LH, Zhang W, et al. [Primary intraosseous Rosai-Dorfman disease: A clinicopathological analysis of fourteen cases]. Zhonghua Bing Li Xue Za Zhi. 2020;49(10):904–9. DOI: https://rs.yiigle.com/cmaid/1234142
8. Gupta S, Finzel KC, Grubber BL. Rosai-Dorfman disease masquerading as chronic ankle arthritis: a case report and review of the literature. Rheumatology (Oxford). 2004;43(6):811–2.DOI: https://academic.oup.com/rheumatology/article-abstract/43/6/811/2891250?redirectedFrom=fulltext
9. Rahman E, Sugath S, John N. A rare case of Rosai-Dorfman disease presenting as a solitary lesion of the distal femur: a case report. J Bone Soft Tissue Tumors. 2022;8(1):7–10. DOI: https://journals.indexcopernicus.com/api/file/viewByFileId/1774471
10. Goyal G, Ravindran A, Young JR, et al. Clinicopathological features, treatment approaches, and outcomes in Rosai-Dorfman disease. Haematologica. 2020;105(2):348–57. DOI: https://pmc.ncbi.nlm.nih.gov/articles/PMC7012468
11. Detlefsen S. IgG4-related disease: a systemic condition with characteristic microscopic features. Histol Histopathol. 2013;28(5):565-84. DOI: https://www.hh.um.es/pdf/Vol_28/28_5/Detlefsen-28-565-584-2013.pdf
12. Ross AB, Davis KW, Buehler D, Chan BY. Primary Rosai-Dorfman disease of bone: a report of two cases. Case Rep Radiol. 2019;2019:1720131. DOI: https://onlinelibrary.wiley.com/doi/10.1155/2019/1720131
13. Koonar E, Ramazani F, Hyrcza M, Chau J. Temporal bone manifestation of primary extranodal Rosai-Dorfman disease: a case report. J Med Case Rep. 2023;17(1):280. DOI: https://link.springer.com/article/10.1186/s13256-023-03790-8