Received: 28 August 2023 | Revised: 23 December 2023 | Accepted: 9 January 2024

DOI: 10.1002/ccr3.8448

Clinical Case Reports Open Access WILEY

CASE REPORT

Prolonged generalized osteomalacia associated with a sinonasal cavity phosphaturic mesenchymal tumor: A case report

Mehdi Montazer[1] | **Naser Tayyebi Meibodi**[2] | **Elmira Teymouri**[1] | **Zohreh Mousavi**[3] | **Sedigheh Reisian**[3] | **Motahare Ebrahimnejad**[1]

[1]Department of Pathology, Mashhad University of Medical Sciences, Mashhad, Iran

[2]Cutaneous Leishmaniasis Research Center, Mashhad University of Medical Sciences, Mashhad, Iran

[3]Metabolic Syndrome Research Center, Mashhad University of Medical Sciences, Mashhad, Iran

Correspondence
Motahare Ebrahimnejad, Department of Pathology, Mashhad University of Medical Sciences, Mashhad, Iran.
Email: me_mu9097@yahoo.com

Key Clinical Message

Phosphaturic mesenchymal tumor (PMT) is a rare disorder primarily affecting the extremities. It is notable for its correlation with hypophosphatemic osteomalacia and high FGF23 serum levels, which results in renal phosphate wasting and clinical symptoms associated with low serum phosphorus. We presented a patient with a 5-year history of progressive osteomalacia who recently experienced a major pathological bone fracture. Laboratory findings showed a persistent low serum phosphate, normal calcium, elevated alkaline phosphatase activity, high parathyroid hormone levels, and increased renal excretion of phosphate. According to ultrasonography and nuclear imaging, there was no evidence of parathyroid adenoma. During further diagnostic assessment, a sinonasal cavity tumor was found and resected. Histologically, the tumor was composed of bland spindle cell proliferation in the background of a calcified matrix with foci of osteoid formation, hemangiopericytoma-like (HPC-like) vasculature, and osteoclast-like giant cells. Tumor cells showed variable positivity for SMA, but CD34, S100, CD99, Melan-A, p63, and desmin were all nonreactive. Regarding the clinical context, histological and immunohistological findings, a final diagnosis of tumor-induced osteomalacia (TIO) secondary to a PMT was made. After surgery, laboratory results returned to normal, clinical symptoms disappeared, and the patient did not experience a recurrence during a six-month follow-up.

KEYWORDS

fibroblast growth factor-23, hypophosphatemia, phosphaturic mesenchymal tumor, sinonasal cavity, tumor-induced osteomalacia

1 | INTRODUCTION

In 1947, McCance reported the first case of tumor-induced osteomalacia (TIO) in a 15-year-old girl suffering from weakness, gait disturbance, and hypophosphatemia which was cured after the removal of her right femur tumor.[1]

TIO is a rare paraneoplastic syndrome that is mainly caused by a phosphaturic mesenchymal tumor (PMT)

and is characterized by renal phosphate wasting and hypophosphatemia, which eventually leads to reduced bone mineralization.[2–4] In 2013, the WHO classification of soft tissue and bone tumors described PMT as a new entity for the first time.[5] It is noteworthy that only about 450 PMT instances have been documented to date.[6]

Although the precise etiology of PMT is still unknown, the associated osteomalacia is best attributed to the abnormal secretion of fibroblast growth factor 23 (FGF-23), which in turn causes low serum phosphate levels due to increased renal phosphate clearance and altering vitamin D metabolism.[7,8]

Most PMTs are benign mesenchymal tumors with a bland spindle cell proliferation and complex vasculature. However, PMTs can exhibit diverse morphological characteristics that challenge the diagnostic process, especially in differentiating a PMT from its mesenchymal mimickers.[8,9]

Surgical resection is curative, but the diagnosis is often delayed due to the low prevalence of PMT and ambiguous clinical symptoms.[6,10] Therefore, it is crucial to carefully evaluate the clinical context along with laboratory results and histological findings to arrive at an accurate diagnosis.[9]

We herein report a patient with long-lasting severe and generalized osteomalacia secondary to a sinonasal PMT, which was initially misdiagnosed as a giant cell granuloma.

2 | CASE PRESENTATION

The patient was a 54-year-old woman with a long history of bone pain, refractory hypophosphatemic osteomalacia of unknown etiology, and nasal mass referred to Imam-Reza Hospital, Mashhad, Iran as the main referral hospital for endocrinology and ENT diseases in the east of Iran. The patient had a history of progressive generalized bone pain and muscular weakness over the past 5 years, which has been further deteriorated by a femoral neck fracture 3 months before the referral. Her daily activities were restricted, and she had to use a walker. There was no pertinent medical history except nasal bleeding since 1 year ago, and family history was negative for bone or mineral abnormalities.

Laboratory workup since 2018 showed a constant pattern of low serum phosphate (1.7 mg/dL), high 24-h urine phosphate (1439 mg/24 h), high alkaline phosphatase level (643 U/L), normal serum calcium level (9.7 mg/dL), and normal 25- hydroxyvitamin D level (60 ng/mL). An elevated parathyroid hormone (PTH) level (102.8 pg/mL) was also detected prompting further work-up for hyperparathyroidism. Table 1 shows the details of laboratory tests over time.

Bone mass density (BMD) indicated osteoporosis with a T score of −5.9 in the hip and −2.7 in the lumbar spine. There was no evidence of a parathyroid lesion on the ultrasonography and technetium 99m sestamibi (MIBI) scan, which made primary parathyroid disease unlikely. This consequently limited the differential diagnosis to the potential causes of secondary hyperparathyroidism.

The history of nasal bleeding, exacerbated by a progressive sense of nasal fullness, shifted the focus toward the nasal cavity. A CT scan of the sinonasal area showed an expansile heterogeneous soft tissue mass occupying the right-side meatus with deviating the nasal septum (Figure 1).

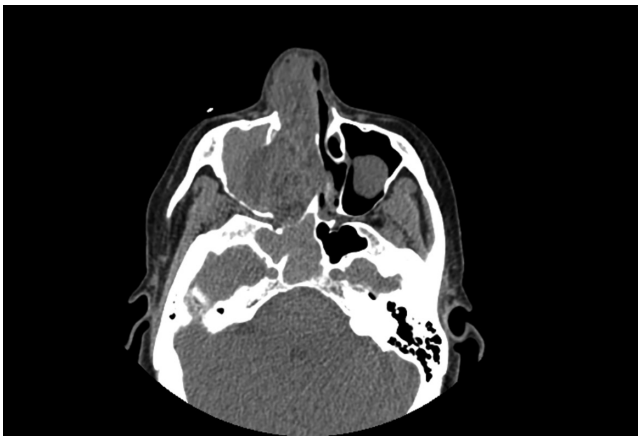

FIGURE 1 Axial CT-scan shows an expansile lesion in the right sinonasal cavity.

TABLE 1 Laboratory workup.

Year						2023		Normal range (unit)
Parameters	**2018**	**2019**	**2020**	**2021**	**2022**	**Pre-operation**	**Post-operation**	
Serum phosphate	2.1 ↓	1.9 ↓	2 ↓	1.3 ↓	1.7 → 1.6 ↓	1.9 ↓	➡4.5	2.6–4.5 mg/dL
Alkaline phosphatase		164 ↑	218 ↑	229 ↑	643 ↑	164 ↑	➡100	30–104 U/L
Phosphate, 24-h urine					1439 ↑			400–1300 mg/24 h
Calcium, total	9	9.1	9.7	9.3	10.1 → 9.5	9.9	➡10.1	8.2–10.7 mg/dL
PTH (CLIA)			58.4	89.3 ↑	102.8 ↑	156 ↑	➡58	15–68.3 pg/mL
25-hydroxy vitamin D	59.4	52.3	48.3	60.1	65.3 → 100	98		30–100 ng/mL

A subsequent biopsy was performed in an outpatient setting 3 weeks before referral, which revealed compact collections of multinucleated giant cells in a vessel-rich background of spindle-cell mesenchymal cells, suggestive of a peripheral giant cell granuloma. The patient was then referred to our hospital because the proposed diagnosis on the biopsy could not explain the patient's illness, and the clinical condition and laboratory tests were unresponsive to phosphate and vitamin D supplementations.

A preliminary diagnosis of TIO was made and the patient underwent complete surgical removal of the nasal cavity mass. The tumor measured 5×4×2 cm with a smooth surface and variable soft to firm consistency. The cut surface had a variegated appearance with foci of gray-yellowish tissue. Histopathological characteristics depicted a bland spindle cell proliferation in a background of HPC-like vasculature, hemorrhage, hemosiderin depositions, areas of fat with some hyalinized thick-walled vessels, and collections of osteoclast-like giant cells with foci of osteoid formation. The mitotic activity was low (1-2/10 HPF), and no pleomorphism or necrosis was identified (Figures 2A–D). Immunohistochemically, the tumor cells showed variable staining for smooth muscle actin (SMA) while they were negative for CD34, S100, HMB-45, Melan-A, CD99, p63, desmin, and SALL4. CD68 was only positive in histiocytic cells and Ki67 showed a low proliferative index (~2%). Figures 3A–D show some immunohistochemical studies. Pathological features were consistent with PMT, and along with the clinical and laboratory findings indicative of a final diagnosis of PMT-induced osteomalacia in the patient.

Following the surgical excision of the tumor, the patient's symptoms gradually improved. Muscle strength increased and the bone pain subsided. Moreover, the patient's laboratory tests, including serum phosphate, ALP, and PTH levels, returned to normal ranges. After 6 months, there was no indication of recurrence of the nasal tumor.

3 | DISCUSSION

Osteomalacia is a metabolic disorder that is characterized by inadequate mineralization of the bones, and it is usually caused by a deficiency of vitamin D.[9,11] However, in rare cases, osteomalacia may occur secondary to other factors such as TIO.[9,11]

TIO is a rare paraneoplastic syndrome primarily caused by a PMT.[12] PMTs predominantly occur in adults without sex preference and are principally found in bone and soft tissue, especially in the lower extremities.[7,10,13] Common clinical characteristics are bone pain, pathologic fractures,

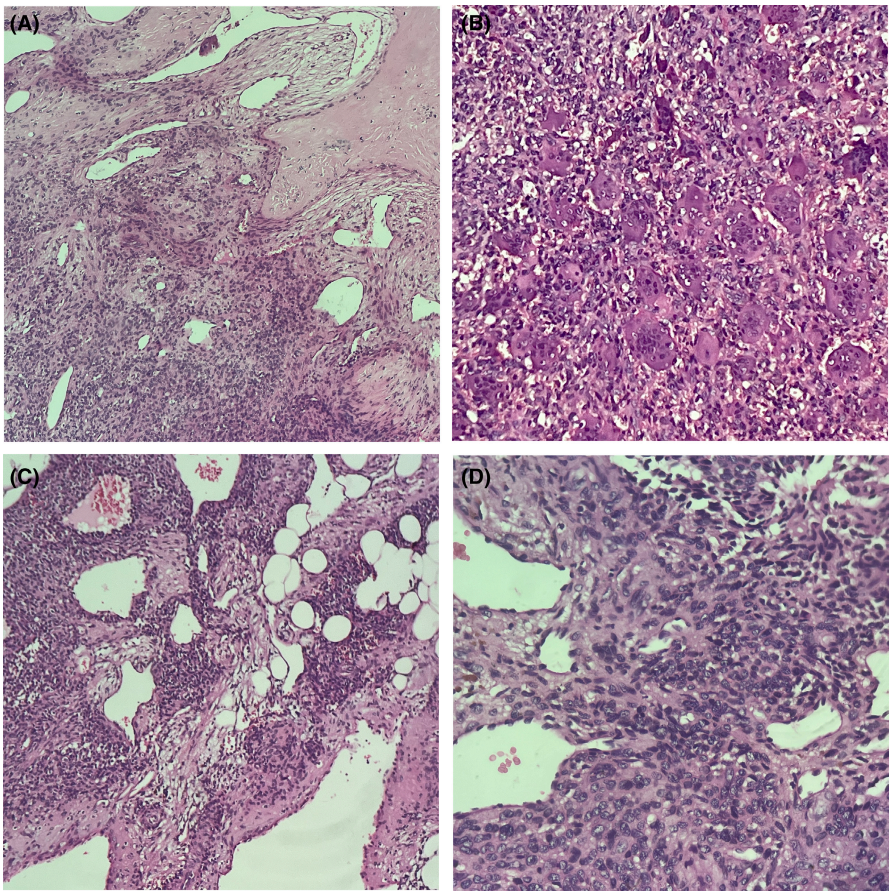

FIGURE 2 Histopathologic findings of PMT. The tumor sections showed the proliferation of bland spindle cells in the background amorphous basophilic matrix and foci of osteoid formation (at the top right) (A, H&E stain, 200×). Numerous osteoclast-like giant cells are intermixed with the spindle component (B, H&E stain, 200×). There is fat tissue with some thick-walled vessels (C, H&E stain, 200×). Prominent hemangiopericytoma-like vessels are present (D, H&E stain, 400×).

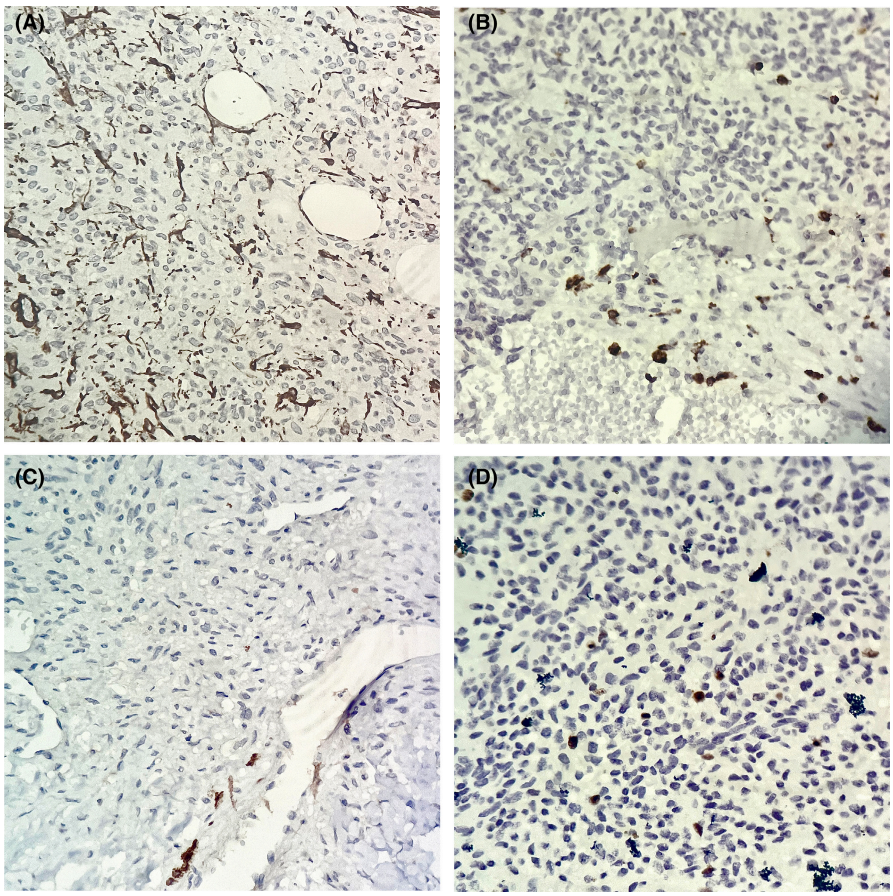

FIGURE 3 Immunohistochemical study showed focal positivity for SMA in some tumoral cells (A, 200×). CD68 was only positive in histiocytic cells (B, 200×). Desmin was negative in tumoral cells (C, 200×). Ki67 was positive in about 2% of tumoral cells (D, 200×).

and musculoskeletal weakness. Such vague medical presentation, difficulty in tumor localization, and nonspecific histologic characterization lead to delayed or even misdiagnosis.[10]

Tumors that cause TIO secrete phosphate-regulating substances called phosphatonins, particularly FGF23, which are believed to play a major role in the development of TIO. Interestingly, the expression of the FGF23 gene is significantly higher in TIO-associated tumors than in normal tissue.[4,9,11,13] FGF23 affects the kidneys by binding to the FGF receptor 1, with the help of the transmembrane co-receptor α-Klotho. The kidneys consequently excrete more phosphate in the urine. Moreover, FGF23 facilitates the resorption of calcium and phosphate from the bones, reduces the absorption of calcium and phosphate in the intestines, and inhibits the production of 1,25 dihydroxycholecalciferol. These actions result in hypophosphatemia, which causes decreased bone mineralization.[9,11,14]

Laboratory findings indicate low levels of serum phosphate, high urinary phosphate excretion, and normal to low levels of 25-dihydroxy vitamin D.[3,12] Furthermore, serum levels of FGF23 help clinicians in diagnosing TIO, localizing the occult source of FGF23 secretion by selective venous sampling, and monitoring recurrences of PMT after surgical excision.[14]

Currently, there is no standardized imaging modality, for the detection and localization of PMTs, and a variety of methods, such as radiography, computed tomography (CT), magnetic resonance imaging (MRI), and octreotide scintigraphy are in use.[12,15]

Some PMTs do not pick up 18F-fluorodeoxyglucose (18F-FDG). Therefore, it has been proposed to replace 18F-FDG-based scans with octreotide-based radiotracers like 68Ga DOTATATE (positron emission tomography (PET)/CT). Nevertheless, this modality is expensive and unavailable in most parts of the world.[4,6,7]

Histologically, a typical PMT demonstrates hypocellular proliferation of bland neoplastic spindle cells growing in a highly vascular, partially calcified (so-called "grungy" calcification), basophilic myxochondroid matrix accompanied by fat tissue and foci rich in osteoclast-like multinucleated giant cells. The HPC-like pattern of the vasculature is also a prominent feature.[2,10,14]

However, PMTs may display remarkable morphologic variations,[2,10] and as in the first biopsy from our patient, an otherwise typical morphology may not be readily perceptible on small biopsies.

There is no specific immunohistochemistry (IHC) marker for PMTs. Therefore, the role of IHC is limited to the exclusion of other possibilities. Vimentin is the only marker that is consistently positive, leading some

authors to consider PMT as a "vimentin-only" neoplasm.[2,16] Otherwise, some markers are variably and weakly reactive, including SMA, CD34, neural, and neuroendocrine proteins (S100 protein, synaptophysin, CD56), ERG, CD68, somatostatin receptor 2A (SSTR2A), and dentin matrix protein-1 (DMP-1).[2,7,8,17,18] Recently, it has been described that PMTs may express SATB2, a regulator of osteogenic differentiation. This correlates with the well-known ability of osteocytes to produce FGF23.[2,17]

IHC testing for FGF23 protein has shown controversial results, mainly due to a lack of specificity.[2,19] On the other hand, some PMTs do not react with anti-FGF23 antibodies, particularly those not associated with the TIO.[2,13] FGF-23 expression level can also be determined using the reverse transcription-polymerase chain reaction (RT-PCR). However, some tumors, such as fibrous dysplasia, chondromyxoid fibroma, and aneurysmal bone cysts, produce low amounts of FGF-23.[9,20]

In molecular genetic studies, the FN1-FGFR1 and FN1-FGF1 fusions are present in approximately 42% and 6% of PMT cases, respectively, according to Lee et al. This finding provides the possibility of a targeted therapy approach in the future.[11,13,20,21]

Expecting the rarity of PMT and its nonspecific histological and immunohistochemical findings, the histopathological diagnosis is usually challenging.[16]

The abundance of multinucleated giant cells may easily deviate the diagnosis to other giant cell-rich mimickers such as the giant cell tumor, benign fibrous histiocytoma, or non-ossifying fibroma of bone, which usually lack the unique matrix of PMT.[2,22] To make it more complex, PMTs may rarely show aneurysmal bone cyst-like (ABC-like) areas and an ABC may have a PMT-like calcifying matrix. The lack of spindle cell proliferation and the presence of USP6 gene rearrangements are in favor of an ABC diagnosis.[2]

As noted earlier, PMT is a highly vascularized neoplasm containing capillary-sized to hyalinized thick-walled vessels and typically HPC-like areas (the so-called staghorn vascular pattern).[2,10,14] Therefore, the distinction between PMT and other HPC-like neoplasms, on top of all a solitary fibrous tumor (SFT), may challenge the diagnosis. On the morphologic ground, SFTs lack the calcified matrix and the giant cell-rich areas of PMTs, and they immunohistochemically show diffuse staining for CD34 and STAT6 proteins. The molecular basis for such diffuse nuclear STAT6 expression is the NAB2::STAT6 gene fusion, which is absent in PMTs.[2,10,20,22,23]

The sinonasal origin of the present tumor poses further difficulties not only for the diagnosis, with several benign and malignant mimickers existing in the area, but also concerning the accessibility to obtain a biopsy and perform a complete curative excision.[24] In the sinonasal cavity, the differential diagnoses include vascular, epithelial, and mesenchymal tumors. Fortunately, a thorough morphologic examination and negative immunostaining for cytokeratins rule out the possibility of most epithelial lesions, including those of salivary gland origin, such as myoepitheliomas.[24]

Glomangiopericytomas of the sinonasal type are distinguished based on the invariable expression of SMA and desmin, along with an abnormal nuclear localization of the beta-catenin protein. Another common vascular neoplasm in the area, nasal angiofibroma, can also make the diagnosis difficult in small biopsies. However, it is more common in males and usually displays a uniform pattern of dilated, thin-walled vessels in a background of collagenous stroma.[2,25]

Some tumors of soft tissue and bone have PMT-like morphological characteristics and express FGF23, but they do not cause TIO symptoms. These tumors are known as "non-phosphaturic" PMTs. There is still debate about whether a true non-phosphaturic PMT exists, especially due to the diverse group of patients. Some such tumors either produce a low level of FGF23 or find a way to neutralize its effects. Other patients do have osteomalacia, but their symptoms are not recognized clinically. Finally, some non-phosphaturic PMT patients have been diagnosed far before the appearance of full-blown phosphate wasting.[2,14,26]

Typically, PMTs are slow-growing benign neoplasms and complete surgical resection is curative.[12] However, local recurrences can occur, and distant metastases, usually to the lungs, are on record.[2,9,16,27,28] Qari et al. reported a case of PMT with multiple local recurrences and eventually pulmonary metastases.[27] Yavropoulou et al. also described a patient who experienced several local recurrences and malignant transformation with lung metastasis 2 years after the first diagnosis of a benign PMT.[16] Malignant transformation typically resembles an undifferentiated pleomorphic sarcoma with high cellularity, pleomorphism, high proliferative activity, and TP53 mutations.[6,16,28,29] Nonetheless, malignant PMTs may sometimes exhibit bland-looking spindle cell lesions.[9,29] The potential for recurrence and metastasis necessitates a complete removal and long-term follow-up, even in PMTs with benign histological features.[27] Symptoms usually resolve a few weeks following a total resection, and the laboratory tests, including FGF23 levels, return to normal ranges.[28] It is important to regularly monitor patients for serum levels of phosphorus, alkaline phosphatase, and FGF23 to detect any potential local tumor recurrences as early as possible.[6,14]

Completely removed benign PMTs are expected to pursue an excellent clinical course and not require any

treatment other than phosphate and active vitamin D supplements. On the other hand, patients with unresectable or incompletely resected tumors may undergo radiation therapy,[4,28] while adjuvant chemoradiation is administrated for malignant PMTs.[22,27] Moreover, an anti-FGF23 monoclonal antibody, called Burosumab, has recently been considered as a potential medical treatment for TIO patients.[6]

4 | CONCLUSION

PMT/TIO is a rare entity that manifests as a paraneoplastic syndrome. Regrettably, symptoms are often nonspecific, the tumor is not easily detectable in routine examinations, and several histological mimics exist, that all may lead to a delayed diagnosis with severe morbidities, such as in our case. The key to a timely diagnosis resides in a careful combination of clinical, laboratory, and histopathological findings.

AUTHOR CONTRIBUTIONS

Mehdi Montazer: Conceptualization; methodology; supervision; writing – review and editing. **Naser Tayyebi Meibodi:** Conceptualization; methodology; supervision; writing – review and editing. **Elmira Teymouri:** Conceptualization; data curation; methodology. **Zohreh Mousavi:** Conceptualization; data curation; methodology. **Sedigheh Reisian:** Conceptualization; data curation; methodology. **Motahare Ebrahimnejad:** Conceptualization; investigation; writing – original draft.

ACKNOWLEDGMENTS

The authors would like to express gratitude toward all who helped in the improvement of this paper.

FUNDING INFORMATION

The authors did not receive any funding for this study.

CONFLICT OF INTEREST STATEMENT

The authors declared no conflicts of interest.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

ETHICS STATEMENT

This study was approved by the institutional review board committee.

CONSENT

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

ORCID

Mehdi Montazer https://orcid.org/0000-0002-7759-3268
Naser Tayyebi Meibodi https://orcid.org/0000-0003-2114-4671
Elmira Teymouri https://orcid.org/0009-0004-9463-9776
Zohreh Mousavi https://orcid.org/0000-0002-2170-9591
Sedigheh Reisian https://orcid.org/0009-0001-9817-912X
Motahare Ebrahimnejad https://orcid.org/0000-0002-0000-4808

REFERENCES

1. Mc CR. Osteomalacia with Looser's nodes (Milkman's syndrome) due to a raised resistance to vitamin D acquired about the age of 15 years. *Q J Med*. 1947;16(1):33-46.
2. Folpe AL. Phosphaturic mesenchymal tumors: a review and update. *Semin Diagn Pathol*. 2019;36(4):260-268.
3. Chong WH, Molinolo AA, Chen CC, Collins MT. Tumor-induced osteomalacia. *Endocr Relat Cancer*. 2011;18(3):R53-R77.
4. Jan de Beur SM. Tumor-induced osteomalacia. *JAMA*. 2005;294(10):1260-1267.
5. Jo VY, Fletcher CD. WHO classification of soft tissue tumours: an update based on the 2013 (4th) edition. *Pathology*. 2014;46(2):95-104.
6. Montanari A, Pirini MG, Lotrecchiano L, Di Prinzio L, Zavatta G. Phosphaturic mesenchymal tumors with or without phosphate metabolism derangements. *Curr Oncol*. 2023;30(8):7478-7488.
7. Liu Y, He H, Zhang C, Zeng H, Tong X, Liu Q. Phosphaturic mesenchymal tumors: rethinking the clinical diagnosis and surgical treatment. *J Clin Med*. 2022;12(1):252-264.
8. Toyosawa S, Tomita Y, Kishino M, et al. Expression of dentin matrix protein 1 in tumors causing oncogenic osteomalacia. *Mod Pathol*. 2004;17(5):573-578.
9. Hudairy RE, Meliti A, Mokhtar G, Alanazi M. Phosphaturic mesenchymal tumor: a case report of a rare entity. *Cureus*. 2022;14(2):e22712.
10. Ledford CK, Zelenski NA, Cardona DM, Brigman BE, Eward WC. The phosphaturic mesenchymal tumor: why is definitive diagnosis and curative surgery often delayed? *Clin Orthop Relat Res*. 2013;471(11):3618-3625.
11. Ghorbani-Aghbolaghi A, Darrow MA, Wang T. Phosphaturic mesenchymal tumor (PMT): exceptionally rare disease, yet crucial not to miss. *Autops Case Rep*. 2017;7(3):32-37.
12. Chan J, James C, Darbandi K, Burch D, Bacaj P, Cheng J. Phosphaturic mesenchymal tumor: a rare cause of severe osteomalacia and debilitation in a 44-year-old man. *AACE Clinical Case Reports*. 2017;3(4):313-316.
13. Lee CH, Su SY, Sittampalam K, et al. Frequent overexpression of klotho in fusion-negative phosphaturic mesenchymal tumors with tumorigenic implications. *Mod Pathol*. 2020;33(5):858-870.
14. Boland JM, Tebben PJ, Folpe AL. Phosphaturic mesenchymal tumors: what an endocrinologist should know. *J Endocrinol Investig*. 2018;41(10):1173-1184.
15. Broski SM, Folpe AL, Wenger DE. Imaging features of phosphaturic mesenchymal tumors. *Skeletal Radiol*. 2019;48(1):119-127.

16. Yavropoulou MP, Poulios C, Foroulis C, et al. Distant lung metastases caused by a histologically benign phosphaturic mesenchymal tumor. *Endocrinol Diabetes Metab Case Rep*. 2018;2018:18-23.
17. Wu H, Bui MM, Zhou L, Li D, Zhang H, Zhong D. Phosphaturic mesenchymal tumor with an admixture of epithelial and mesenchymal elements in the jaws: clinicopathological and immunohistochemical analysis of 22 cases with literature review. *Mod Pathol*. 2019;32(2):189-204.
18. Houang M, Clarkson A, Sioson L, et al. Phosphaturic mesenchymal tumors show positive staining for somatostatin receptor 2A (SSTR2A). *Hum Pathol*. 2013;44(12):2711-2718.
19. Bahrami A, Weiss SW, Montgomery E, et al. RT-PCR analysis for FGF23 using paraffin sections in the diagnosis of phosphaturic mesenchymal tumors with and without known tumor induced osteomalacia. *Am J Surg Pathol*. 2009;33(9):1348-1354.
20. Beygi S, Denio A, Sharma TS. The foot that broke both hips: a case report and literature review of tumor-induced osteomalacia. *Case Rep Rheumatol*. 2017;2017:3191673.
21. Lee JC, Su SY, Changou CA, et al. Characterization of FN1-FGFR1 and novel FN1-FGF1 fusion genes in a large series of phosphaturic mesenchymal tumors. *Mod Pathol*. 2016;29(11):1335-1346.
22. Folpe AL, Fanburg-Smith JC, Billings SD, et al. Most osteomalacia-associated mesenchymal tumors are a single histopathologic entity: an analysis of 32 cases and a comprehensive review of the literature. *Am J Surg Pathol*. 2004;28(1):1-30.
23. Rabie A, Hasan A, Mohammed Y, Abdelmaksoud A, Rabaan AA. Recurrent malignant solitary fibrous tumor of the scalp: a case report and literature review. *J Pathol Transl Med*. 2022;56(2):103-108.
24. Mocanu H, Mocanu AI, Moldovan C, Soare I, Postolache PA, Nechifor A. Rare and unusual benign tumors of the sinonasal tract and pharynx: case series and literature review. *Exp Ther Med*. 2022;23(5):334.
25. Linsey M, Smith W, Yamauchi H, Bernstein L. Nasopharyngeal angiofibroma presenting as adult osteomalacia: case report and review of the literature. *Laryngoscope*. 1983;93(10):1328-1331.
26. Sent-Doux KN, Mackinnon C, Lee JC, Folpe AL, Habeeb O. Phosphaturic mesenchymal tumor without osteomalacia: additional confirmation of the "nonphosphaturic" variant, with emphasis on the roles of FGF23 chromogenic in situ hybridization and FN1-FGFR1 fluorescence in situ hybridization. *Hum Pathol*. 2018;80:94-98.
27. Qari H, Hamao-Sakamoto A, Fuselier C, Cheng YS, Kessler H, Wright J. Phosphaturic mesenchymal tumor: 2 new oral cases and review of 53 cases in the head and neck. *Head Neck Pathol*. 2016;10(2):192-200.
28. Qiu S, Cao LL, Qiu Y, et al. Malignant phosphaturic mesenchymal tumor with pulmonary metastasis: a case report. *Medicine (Baltimore)*. 2017;96(17):e6750.
29. Oyama N, Kojima-Ishii K, Toda N, et al. Malignant transformation of phosphaturic mesenchymal tumor: a case report and literature review. *Clin Pediatr Endocrinol*. 2020;29(2):69-75.

How to cite this article: Montazer M, Meibodi NT, Teymouri E, Mousavi Z, Reisian S, Ebrahimnejad M. Prolonged generalized osteomalacia associated with a sinonasal cavity phosphaturic mesenchymal tumor: A case report. *Clin Case Rep*. 2024;12:e8448. doi:10.1002/ccr3.8448