

It's Going TIBIA Okay:

Octreotide in the Treatment of Lower Extremity Pain Due To Pulmonary Hypertrophic Osteoarthropathy

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INTRODUCTION

Hypertrophic osteoarthropathy (HOA) is a syndrome eponymously known as Bamberger-Marie Syndrome or Pierre Marie-Bamberger syndrome, which is defined as an abnormal periosteal proliferation of tubular (long) bones, clubbing of the digits (Hippocrates fingers) and synovial effusions. Primary HOA is a rare, hereditary condition associated with congenital heart disease, also known as pachydermoperiostosis. Secondary HOA is associated with malignancy, notably non-small cell lung cancer, and is referred to as Hypertrophic Pulmonary Osteoarthropathy (HPOA)¹.

The exact mechanism of HOA is unknown. The most common clinical presentation is characterized by a painful, generalized arthropathy with symmetrical involvement of the ankles, knees, wrists and elbows. Radiographic imaging may show periosteal changes in the long bones. An isotope bone scan typically shows diffuse radiotracer uptake in the long bones in a “railroad track” pattern. HPOA can be differentiated from Rheumatoid arthritis and osteoarthritis as there are no erosions or inflammatory synovitis and no joint space narrowing, respectively².



Figure 1: CT Chest, axial view. Left upper lobe mass



Figure 2: Clubbing of digits



Figure 3: Xray of R lower extremity – AP view



Figure 4: Xray of R lower extremity – Lat view

PATIENT PRESENTATION

A 54-year-old-male with PMH of COPD, current smoker with 40 pack year history and bilateral knee osteoarthritis (OA) presented to the ED after a fall. Of note, patient was in a motorcycle accident in 1985, and had ORIF of his RLE. Upon further questioning, patient stated that he had 3 falls within the past few months, attributed to vertigo and tinnitus—worsened with changes in position. He also reported decreased oral intake secondary to food scarcity. ROS positive for orthopnea, PND, watery diarrhea with intermittent maroon-colored stools for 3 months.

Imaging incidentally found a left lung upper lobe mass. Patient underwent IR guided biopsy which revealed stage III non-small-cell lung adenocarcinoma. He was not a candidate for cardiothoracic surgery and outpatient treatment with chemotherapy was recommended.

Over the course of his hospitalization, patient developed worsening bilateral lower extremity pain. Right knee X-ray imaging showed tri-compartmental OA, intact hardware, and chronic periosteal reaction along femoral shafts and right tibial and fibular shafts which indicated the possibility of hypertrophic osteoarthropathy. On initial evaluation by Physical Therapy, the patient was functioning at a contact guard level for bed mobility and transfers. He was able to ambulate 25ft contact-guard with a rolling walker. Over the course of hospitalization, the patient's functional status declined, requiring moderate assist for bed mobility, and sit-to-stand transfers, and with inability to ambulate secondary to severe pain. Due to impaired mobility and lack of family/social support, the patient was unable to be discharged, and initiation of chemotherapy was delayed. Under the assumption that pain was attributed to OA, decision was made to trial intra-articular corticosteroid injection of the right knee. Patient received minimal relief and remained bedbound. The Palliative care team was consulted for pain management. PM&R resident on a cancer rehabilitation rotation with the palliative team performed the consult. A thorough social and functional history revealed that the patient noted 30+ years of chronic bilateral knee pain usually relieved with Naproxen. He was functioning at a modified independent level at baseline and ambulated with a single-axis cane. His described the pain as a deeper, sharp, aching pain that was different than his typical knee pain.

The decision was made to confirm diagnosis of HPOA with bone scintigraphy. After positive results for HPOA, decision was made to treat his pain with Zoledronic acid 4mg IV. However, the patient had poor dentition with multiple missing teeth, which prompted a Dental consult for evaluation and clearance due to the potential side effect of osteonecrosis of the jaw. Patient had multiple teeth extracted with dental and required at least 2 weeks of recovery prior to initiating bisphosphonate therapy. Due to delay in therapy, and patient with continued knee pain, treatment with Octreotide was started. The patient obtained significant relief of his pain and was functional status improved thereafter. The patient was able to begin chemotherapy and was discharged to a sub-acute rehabilitation facility.

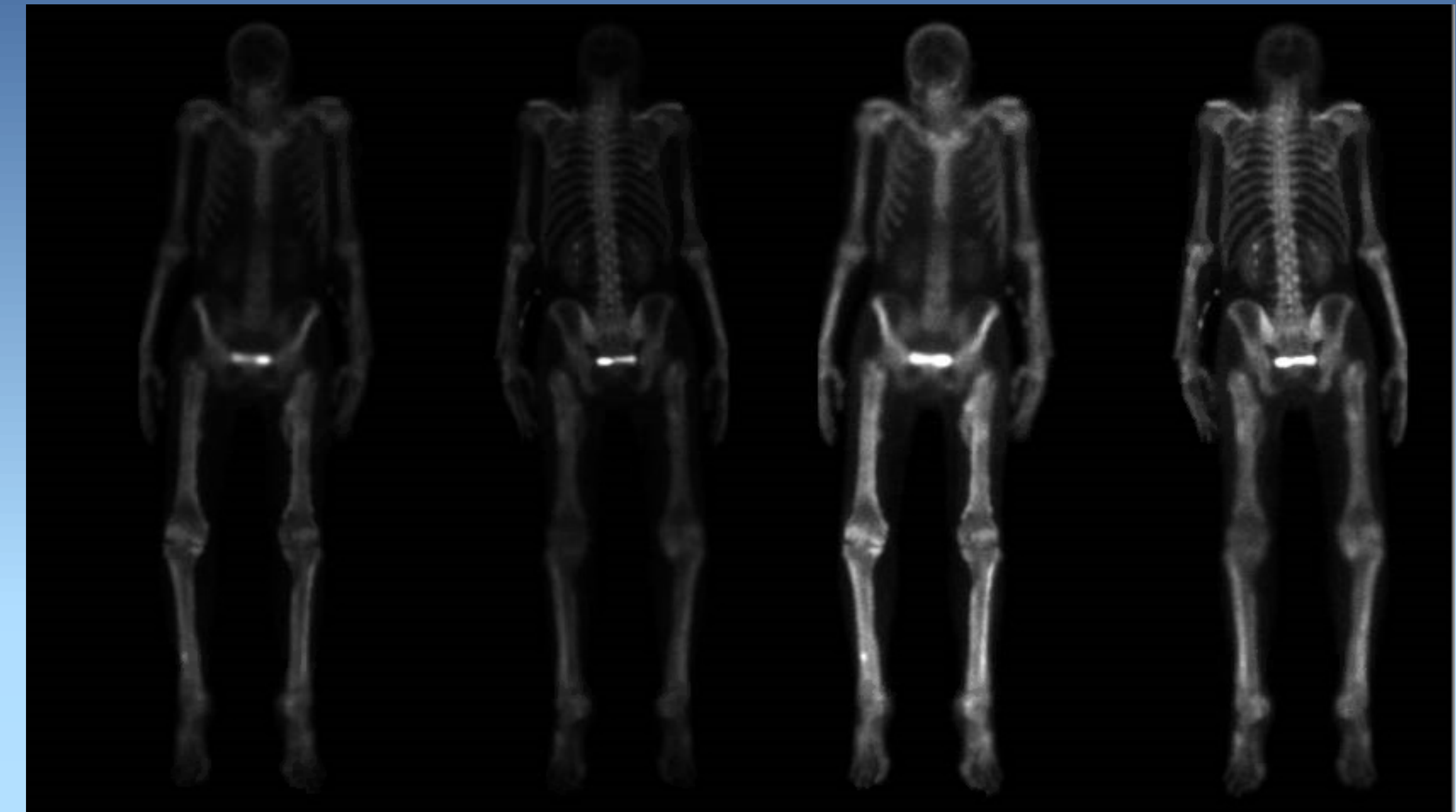


Figure 5: Bone scintigraphy

DISCUSSION

In patients with acute or chronic musculoskeletal pain, with an exacerbation of pain that is out of proportion with typical symptoms, further work up is warranted to elucidate the etiology of pain. In patients with known malignancy—especially of the lung—who present with a triad of digital clubbing, synovial effusions and periostitis, the diagnosis of HPOA should be considered. Diagnosis is confirmed with radiographic imaging including nuclear bone scintigraphy.

Although definitive treatment of the underlying malignancy often results in resolution of PHO, treatment with bisphosphonates or Octreotide can assist with pain management^{3,4}.

CONCLUSION

In this case, due to the patient's existing diagnosis of tri-compartmental OA from his prior trauma, a further work-up of his pain was delayed. Only upon discussing the patient's functional history while fulfilling a pain consult was it elucidated that the patient's pain was new and out-of-proportion with his chronic lower extremity pain.

Due to his deteriorating functional status, there was a significant delay in his disposition and initiation of chemotherapy. A multi-disciplinary, whole-patient approach allowed for the patient's pain to be properly diagnosed, treated and for his chemotherapy to be initiated.

REFERENCES

1. Yap FY, Skalski MR, Patel DB, et al. Hypertrophic Osteoarthropathy: Clinical and Imaging Features. *RadioGraphics*. 2017;37(1):157-195. doi:10.1148/rg.2017160052
2. Kaur H, Muhleman M, Balon HR. Hypertrophic Osteoarthropathy on Bone Scintigraphy. *J Nucl Med Technol*. Jun 2018;46(2):147-148. doi:10.2967/jnmt.117.199315
3. Nguyen S, Hojati M. Review of current therapies for secondary hypertrophic pulmonary osteoarthropathy. *Clin Rheumatol*. 2011 Jan;30(1):7-13. doi: 10.1007/s10067-010-1563-7. Epub 2010 Oct 9. PMID: 20936419.
4. Birch E, Jenkins D, Noble S. Treatment of painful hypertrophic osteoarthropathy associated with non-small cell lung cancer with octreotide: a case report and review of the literature. *BMJ Support Palliat Care*. 2011;1(2):189-192. doi:10.1136/bmjspcare-2011-000052

