

When Joint Pain and Clubbing Speaks: Pulmonary Scar Carcinoma Heralded by Hypertrophic Pulmonary Osteoarthropathy

Richmond Ronald Gomes^{1*}, Jannatul Moua Efa², Nafisa Rahman Bushra³ and Samina Yasmeen Bari⁴

¹Professor and Head, Medicine, Ad-din Women's Medical College Hospital, Dhaka, Bangladesh

²Clinical Assistant, Medicine, Ad-din Women's Medical College Hospital, Dhaka, Bangladesh

³Medical Officer, Medicine, Ad-din Women's Medical College Hospital, Dhaka, Bangladesh

⁴Consultant, Respiratory Medicine, Barnsley Hospital, Yorkshire, UK

***Corresponding Author:** Richmond Ronald Gomes, Professor and Head, Medicine, Ad-din Women's Medical College Hospital, Dhaka Bangladesh.

Received: June 01, 2026; **Published:** September 29, 2026

Abstract

Scar carcinoma is an uncommon form of bronchogenic carcinoma arising in areas of prior pulmonary fibrosis or scarring and may rarely present with hypertrophic pulmonary osteoarthropathy (HPOA), a paraneoplastic syndrome characterized by digital clubbing, bilateral, symmetrical periostitis of long bones and noninflammatory arthritic pain and swelling. We report the case of a 70-year-old male smoker with the history of treatment completed tuberculosis who presented with chronic cough, progressive dyspnea, painful swelling of the small joints of both hands, wrists and ankles, and digital clubbing. Radiography and computed tomography of the chest demonstrated a spiculated mass arising within a fibrotic scar in the left mid lung. Histopathological examination following CT guided FNAC confirmed adenocarcinoma of the lung associated with scar tissue. No distant metastases were identified. This case highlights the importance of considering underlying lung malignancy in patients presenting with HPOA and clubbing. Early recognition of scar carcinoma is essential because delayed diagnosis may occur when symptoms are attributed solely to chronic pulmonary fibrosis or inflammatory disease. Awareness of this rare association can facilitate timely investigation, accurate staging, and improved multidisciplinary management in high-risk patients with unexplained musculoskeletal manifestations and clubbing symptoms.

Keywords: Hypertrophic Pulmonary Osteoarthropathy (HPOA); Hypertrophic Osteoarthropathy (HOA); Platelet-Derived Growth Factor (PDGF); Vascular Endothelial Growth Factor (VEGF)

Introduction

Lung cancer is the leading cause of cancer-related mortality worldwide. Although lung cancer is predominantly seen in smokers, never-smokers (individuals who have smoked less than 100 cigarettes in their lifetime) account for 20% of cases globally [1]. Adenocarcinoma is the most common histologic type among both groups [2]. Although cigarette smoking is, by far, the biggest risk factor for developing lung cancer, age, occupational exposures, environmental pollution, race, gender, and preexisting lung disease are all important contributors [3]. There is an etiologic relationship between lung scarring and the development of pulmonary carcinoma [4]. In this article, we present a case of lung adenocarcinoma that originated from a post tubercular scar.

Citation: Richmond Ronald Gomes., *et al.* "When Joint Pain and Clubbing Speaks: Pulmonary Scar Carcinoma Heralded by Hypertrophic Pulmonary Osteoarthropathy". *EC Pulmonology and Respiratory Medicine* 15.10 (2026): 01-06.

Hypertrophic Pulmonary Osteoarthropathy (HPO) or Pierre-Marie-Bamberger syndrome is a subcategory of hypertrophic osteoarthropathy (HOA). HOA is characterized by clubbed fingers, periostitis of long bones and arthritic changes [5]. The primary form of HPO is hereditary and predominantly seen in males. Secondary HPO is an arthropathy that results from a pulmonary disease [5]. The most common, however, is that of intrathoracic neoplasms (lung cancer, pleural fibroma, mesothelioma) which accounts for approximately 90% of HPO cases and of those 80% are lung cancer [5]. Although HPO is most commonly seen in lung neoplasms, the overall incidence of HPO in lung cancer patients is very low. According to one study, less than 1% of lung cancer patients develop HPO as a paraneoplastic syndrome while historical studies have published anywhere from 0.2-17% [5]. The demographics of the lung cancer patients that most commonly develop HPO are males, heavy smokers and advanced disease [5].

In such conditions platelet precursors through arteriovenous shunting may circumvent the pulmonary circulation, avoiding damage to these cells. In such instances, the unfragmented megakaryocytes are believed to enhance the production of platelet-derived growth factor (PDGF) and vascular endothelial growth factor (VEGF) by endothelial cells [6,7]. This, in turn, leads to new blood vessel formation and endothelial cell growth, clinically presented as the characteristic fingers and toes clubbing. Chronic inflammation and tissue damage associated with these conditions likely contribute to the development of HPO.

Case Report

A 70 years old farmer from rural Bangladesh, not known to have hypertension and diabetes presented to our medicine department with the complaints of pain and swelling in both wrists, elbows, ankles and knees for last 6 months. Pain persisted throughout the day without significant inactivity stiffness. Spine and small joints of hands and feet were not involved. There were no features of enthesitis. He also had chronic daily cough and shortness of breath on exertion for the same duration. But he denied any chest pain or hemoptysis. On query, he had lost five kg of his weight in the given period with poor appetite. He had history of pulmonary tuberculosis eight years back and completed treatment. On examination, he was cachectic with BMI 16.4 kg/m², mildly anemic with no palpable lymph nodes. Digital clubbing was present in both hands and feet (Figure 1 and 2). Grade 2 tenderness was present in all of his wrists, elbows, ankles and knees with restricted both active and passive movement. All other systemic examination including respiratory system examination were unremarkable.



Figure 1 and 2: Showing clubbing in both feet and hand respectively.

Investigation showed normocytic normochromic anemia with hemoglobin 9.2 gm/gl. Renal and liver function tests and serum uric acid were normal. Anti-CCP came negative. CRP was raised to 179.2 mg/dl.

Sputum for Gene x-pert and malignant cell was negative. Chest x ray showed ill defined opacity in left mid zone (Figure 3). X ray hand showed new bone formation at wrist (Figure 4).

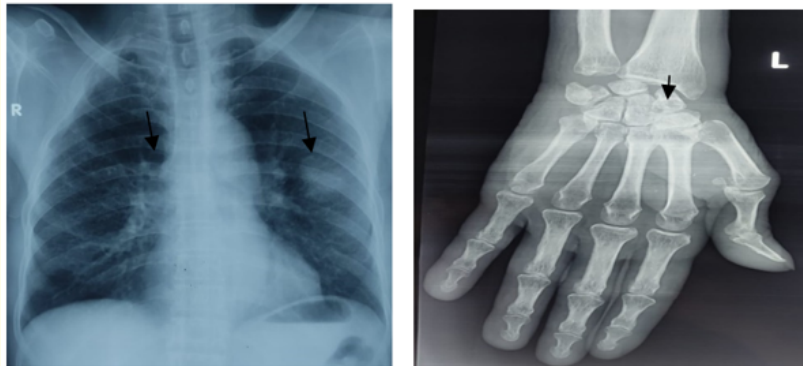


Figure 3 and 4: Chest x ray PA view showing ill defined opacity in left mid zone (Black arrow) and X ray right hand showing new bone formation at wrist (Black arrow).

CT chest was done which revealed a 1.7×1.1 cm opacity in the left lower lobe with interlobular septal thickening, prominent interstitial infiltrates within the left lung, and paratracheal lymphadenopathy (Figure 5). CT guided FNAC was done and histopathology revealed adenocarcinoma (Figure 6). Patient refused for immunohistochemistry and further investigations for staging. He was referred to national institute of cancer diseases and hospital for further multidisciplinary management.

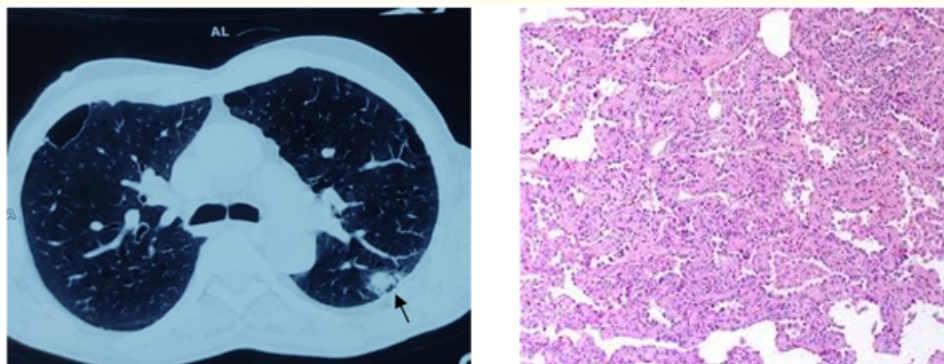


Figure 5 and 6: CT chest showing opacity in left lower lobe (black arrow) and histology from pulmonary lesion revealed adenocarcinoma.

Discussion

There are two forms of hypertrophic osteoarthropathy (HOA), the secondary form as presented in this case study and a primary, idiopathic form of HOA also termed pachydermoperiostosis was first described in 1889. The primary form of this condition is much less

common, consisting of only 3 - 5% of all HOA cases and presenting around the time of puberty as the result of an inherited autosomal dominant trait without underlying disease [8]. HPOA is a condition which is characterized by hypertrophic osteoarthropathy (HOA) secondary to underlying pulmonary disease [9]. The majority of cases of HPOA arise secondary to paraneoplastic syndromes, with studies showing this occurring at a rate of over 80% [10,11]. Of these cases, adenocarcinoma is the most common form of malignancy with small cell carcinoma being the least common [12]. Chronic infections have also been associated with HOA, such as cystic fibrosis, subacute bacterial endocarditis, syphilis, acquired immunodeficiency syndrome, pulmonary tuberculosis.

Lung scar carcinoma (LSC) was first described in 1939 by Friedrich as a form of lung cancer that originates from peripheral scars in the lung. These, in turn, may arise from infection, injury, intrinsic pulmonary disease, or recurrent episodes of tumor necrosis and healing [13]. The most common etiologic factor for the development of LSC is scarring secondary to tuberculosis, but it is also known to occur in the setting of pneumonia, pulmonary abscess, bronchiectasis, and pulmonary infarction [14]. The pathogenesis involves production of acute-phase reactants during the inflammatory response, which leads to recruitment of leukocytes. These activated cells produce reactive oxygen species (ROS) that mediate mutagenic changes in deoxyribonucleic acid (DNA) and damage proteins involved in the maintenance of genomic stability [15,16]. Chronic inflammation promotes persistent DNA damage and eventual activation of oncogenes with subsequent neoplastic transformation. Inflammatory mediators such as tumor necrosis factor (TNF), transforming growth factor (TGF), and interleukins 1, 4, 6, and 13 cause angiogenesis and fibrosis [15,17] and are also implicated in tumorigenesis. Notably, the Prostate, Lung, Colorectal, and Ovarian (PLCO) Cancer Screening Trial has demonstrated a two-fold increased risk of lung cancer associated with the detection of pulmonary scarring on chest X-ray [18].

LSC is most commonly subpleural adenocarcinoma with no evidence of bronchial origin and is characterized histologically by contiguity with dense, hyalinized scar tissue that itself does not comprise any tumor cells [13]. It is hypothesized that impedance of lymphatic drainage by scar tissue can lead to accumulation of malignant cells with subsequent accelerated vascular and/or lymphatic spread of LSC, as was observed in our patient [19]. LSC therefore carries a dismal prognosis, with a five-year survival rate of only 5%, as opposed to 22% for non-LSC adenocarcinoma and 28% for non-LSC adenosquamous cell carcinoma [20]. It is, therefore, important to diagnose and treat this disease early. Because of the physical overlap that exists between LSC and scar tissue on CT imaging, the former is often misdiagnosed as an old fibrotic lesion in the absence of sequential imaging, particularly in younger patients. However, radiographic changes in scar tissue, such as expansion, vacuolation, vessel convergence within the region of fibrosis, and emergence of well-defined surrounding ground-glass opacities and spiculations, strongly support the presence of LSC. Vacuolation and ground-glass opacities are especially useful in differentiating malignant from benign lesions. Ground glass opacities surrounding areas of scar tissue are considered to be reliable markers of early-stage LSC but tend to consolidate over time and so run the risk of being missed if not detected early enough [21].

The cause of HPOA remains poorly understood, but is thought to be due to the production of growth factors, such as growth hormone, oestrogen, adrenocorticotrophin or vascular endothelial growth factor (VEGF) by the lung lesion itself, or failure of the normal removal of these factors by pulmonary arteriovenous shunting [5,22,23]. Shunting is thought to be dominant factor in non-cancerous lesions but may also play a role in lung cancers due to intratumor shunting. There is growing evidence that VEGF may play a central role in HPOA. Firstly, it is a powerful endothelial cell-stimulator factor and VEGF receptors are found in subperiosteal bone cells. Secondly, elevated levels have been found in many diseases associated with HPOA and it is known to be produced by many tumors as well as in response to hypoxia. Finally, VEGF is a platelet derived factor and platelet dysfunction in arteriovenous shunting is thought to be involved in the pathogenesis of HPOA [24].

Diaphyseal and metaphyseal periostitis is the radiological hallmark of HPOA on plain film, causing periosteal thickening and new bone growth [25]. Bone scintigraphy is the investigation of choice, revealing changes earlier and with a higher sensitivity than plain radiography [22,26]. Single-photon emission computed tomography may increase the confidence of diagnosis [27].

Treatment of the lung condition is hypothesized to exert its effect by the reduction in the number of cells producing endogenous growth factor, although the degree of response to treatment is sometimes incomplete or absent. There is evidence that cisplatin and other chemotherapy agents such as doxorubicin directly reduce VEGF transcription in both hypoxic situations and at baseline levels suggesting an alternative pathway for the action of chemotherapy on HPOA [28]. In contrast, docetaxel has not been shown to have any action on VEGF transcription [28] and the failure to provide symptomatic relief in this case may be a reflection of the lack of action on transcription in addition to failure to reduce tumor bulk.

Conclusion

Our case serves to highlight the link between pulmonary scarring and carcinogenesis, as well as to demonstrate the relentless course of lung scar carcinoma, which necessitates timely diagnosis. The USPSTF (US Preventive Service Task Force) recommends annual screening for lung cancer with a low-dose CT scan of chest in adults aged 55 to 80 who have a 30 pack-year smoking history. However, no such recommendations exist for individuals with a known history of lung scarring. Our case reinforces the need for periodic CT imaging of the chest in this group of individuals who are at increased risk of this form of malignancy and underscores the need for evidence to determine the survival benefit of such a measure.

Conflict of Interest

None declared.

Bibliography

1. DM Parkin, *et al.* "Global cancer statistics, 2002". *CA: a Cancer Journal for Clinicians* 55.2 (2005): 74-108.
2. S Couraud, *et al.* "Lung cancer in never smokers - a review". *European Journal of Cancer* 48.9 (2012): 1299-1311.
3. P de Groot and RF Munden. "Lung cancer epidemiology, risk factors, and prevention". *Radiologic Clinics of North America* 50.5 (2012): 863-876.
4. RK Bobba, *et al.* "Scar carcinoma of the lung: a historical perspective". *Clinical Lung Cancer* 12.3 (2011): 148-154.
5. Ito T, *et al.* "Hypertrophic pulmonary osteoarthropathy as a paraneoplastic manifestation of lung cancer". *Journal of Thoracic Oncology* 5.7 (2010): 976-980.
6. Nahar I, *et al.* "Secondary hypertrophic osteoarthropathy: new insights on pathogenesis and management". *Gulf Journal of Oncology* 1.1 (2007): 71-76.
7. Ntaios G, *et al.* "Hypertrophic pulmonary osteoarthropathy secondary to bronchial adenocarcinoma and coexisting pulmonary tuberculosis: a case report". *Cases Journal* 1 (2008): 221.
8. Castori M, *et al.* "Pachydermoperiostosis: an update". *Clinical Genetics* 68.6 (2005): 477-486.
9. Pourmorteza M, *et al.* "Hypertrophic pulmonary osteoarthropathy: a rare but treatable condition in palliative medicine". *Journal of Pain and Symptom Management* 50.2 (2015): 263-267.
10. Jajic Z, *et al.* "Primary hypertrophic osteoarthropathy: clinical, radiologic, and scintigraphic characteristics". *Archives of Medical Research* 32.2 (2001): 136-142.
11. Yao Q, *et al.* "Periostitis and hypertrophic pulmonary osteoarthropathy: report of 2 cases and review of the literature". *Seminars in Arthritis and Rheumatism* 38.6 (2009): 458-466.

12. Poant_a L., *et al.* "Paraneoplastic hypertrophic osteoarthropathy: evaluation at 25 years after pneumectomy". *Polskie Archiwum Medycyny Wewnetrznej* 119.9 (2009): 603-606.
13. RK Bobba., *et al.* "Scar carcinoma of the lung: a historical perspective". *Clinical Lung Cancer* 12.3 (2011): 148-154.
14. H Yokoo and EE Suckow. "Peripheral lung cancers arising in scars". *Cancer* 14.6 (1961): 1205-1215.
15. F Balkwill and A Mantovani. "Inflammation and cancer: back to Virchow?". *The Lancet* 357.9255 (2001): 539- 545.
16. M Jaiswal., *et al.* "Inflammatory cytokines induce DNA damage and inhibit DNA repair in cholangiocarcinoma cells by a nitric oxide dependent mechanism". *Cancer Research* 60.1 (2000): 184-190.
17. K Dheda., *et al.* "Lung remodeling in pulmonary tuberculosis". *The Journal of Infectious Diseases* 192.7 (2005): 1201-1209.
18. Y-Y Yu., *et al.* "Lung cancer risk following detection of pulmonary scarring by chest radiography in the prostate, lung, colorectal, and ovarian cancer screening trial". *Archives of Internal Medicine* 168.21 (2008): 2326-2332.
19. R Carroll. "The influence of lung scars on primary lung cancer". *The Journal of Pathology and Bacteriology* 83.1 (1962): 293-297.
20. LJ Freant., *et al.* "Scar carcinoma of the lung. Fact or fantasy?". *The Annals of Thoracic Surgery* 17.6 (1974): 531-537.
21. F Gao., *et al.* "CT features of lung scar cancer". *Journal of Thoracic Disease* 7.3 (2015): 273-280.
22. Uchisako H., *et al.* "Bone scintigraphy in growth hormone-secreting pulmonary cancer and hypertrophic osteoarthropathy". *Journal of Nuclear Medicine* 36.5 (1995): 822-825.
23. Marino WD., *et al.* "A 49-year-old smoker with a lung mass and diffuse bone pain". *Chest* 139.2 (2011): 460-463.
24. Olan F., *et al.* "Circulating vascular endothelial growth factor concentrations in a case of pulmonary hypertrophic osteoarthropathy. Correlation with disease activity". *Journal of Rheumatology* 31.3 (2004): 614-616.
25. Sato K., *et al.* "[Pulmonary hypertrophic osteoarthropathy associated with primary lung cancer]". *Nihon Kokyuki Gakkai Zasshi* 38.1 (2000): 73-77.
26. Morgan B., *et al.* "Hypertrophic osteoarthropathy in staging skeletal scintigraphy for lung cancer". *Clinical Radiology* 51.10 (1996): 694-697.
27. Lin Y., *et al.* "The usefulness of bone scintigraphy in the evaluation of hypertrophic osteoarthropathy after chemotherapy: a case report and literature review". *Annals of Nuclear Medicine and Science* 23.1 (2010): 47-51.
28. Duyndam MC., *et al.* "Cisplatin and doxorubicin repress Vascular Endothelial Growth Factor expression and differentially down-regulate Hypoxia-inducible Factor I activity in human ovarian cancer cells". *Biochemical Pharmacology* 74.2 (2007): 191-201.

Volume 15 Issue 10 October 2026

©All rights reserved by Richmond Ronald Gomes., *et al.*