International Journal of Science and Research (IJSR)
ISSN: 2319-7064
Impact Factor 2025: 7.089

A Decade in Silence: Incidental Pulmonary Cement Embolism After Post-Traumatic Vertebroplasty with Concurrent Pulmonary Tuberculosis

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Abstract: ***Pulmonary cement embolism (PCE) is a recognized but frequently underdiagnosed complication of percutaneous vertebroplasty (PVP). Most cases are asymptomatic and incidentally detected on imaging. We report a rare case of delayed incidental PCE identified approximately ten years after post-traumatic vertebroplasty in a 50-year-old female undergoing pre-employment medical evaluation. Chest radiography demonstrated linear hyperdense branching opacities within the lungs. HRCT thorax revealed peripheral pulmonary cement emboli involving segmental and subsegmental pulmonary arteries, with cement tracking from the L2 vertebral body through the paravertebral venous plexus and inferior vena cava. Concurrent imaging findings of active pulmonary tuberculosis with fibro-bronchiectatic sequelae were also identified. This case highlights the long-term persistence of PCE, the importance of systematic HRCT interpretation, and the need to consider prior vertebroplasty history when evaluating intravascular high-attenuation pulmonary opacities.***

Keywords: pulmonary cement embolism; percutaneous vertebroplasty; post-traumatic vertebral fracture; road traffic accident; HRCT thorax; pulmonary tuberculosis; PMMA embolism; delayed complication; incidental finding; cement leakage; thoracolumbar fracture; PMMA vertebroplasty complication; thoracic CT imaging

1. Introduction

Percutaneous vertebroplasty (PVP) is a minimally invasive fluoroscopic or CT-guided procedure in which polymethylmethacrylate (PMMA) bone cement is injected into a fractured or structurally compromised vertebral body to restore mechanical stability and provide pain relief [1]. Originally developed for the treatment of vertebral hemangiomas, PVP has since been widely adopted for osteoporotic vertebral compression fractures and, increasingly, for post-traumatic vertebral fractures in younger patients following high-energy mechanisms such as road traffic accidents (RTAs) [2,3].

Post-traumatic vertebral compression fractures- particularly burst fractures or stable wedge fractures of the thoracolumbar junction- are increasingly being managed with PVP or balloon kyphoplasty as an adjunct or alternative to open surgical fixation, especially in patients who are not candidates for major spinal surgery [4]. The biomechanical goals of cement augmentation in the post-traumatic setting are analogous to those in osteoporotic fractures: restoration of vertebral body height, prevention of progressive kyphosis, and alleviation of pain [3,4].

A well-recognized, though often subclinical, complication of PVP is the extravasation of PMMA cement into the paravertebral and epidural venous plexus, with subsequent embolisation into the systemic venous circulation and pulmonary vasculature [5,6]. Pulmonary cement embolism (PCE) has been reported in 3.5–26% of vertebroplasty cases when specifically sought by post-procedural computed tomography (CT), with the majority of cases being clinically silent and escaping clinical detection [7,8]. The characteristic CT appearance of PCE- linear, branching, high-attenuation (>400 HU) structures within the pulmonary arterial tree, often extending from the IVC through the right heart- is pathognomonic and permits definitive radiological diagnosis [9].

What distinguishes the present case from the existing literature is the remarkable temporal gap between the index vertebroplasty (performed in 2015, approximately a decade prior to presentation) and the incidental detection of PCE. This not only raises important questions about the persistence and clinical evolution of cement emboli over time, but also emphasizes that PCE must remain on the differential diagnosis whenever characteristic CT findings are encountered, regardless of how remote the vertebroplasty history may be.

We present this case- unique in its combination of post-traumatic vertebroplasty aetiology, decade-long delayed detection of PCE, and the concurrent radiological diagnosis of active pulmonary tuberculosis- to highlight the critical role of systematic and comprehensive HRCT interpretation, and to advocate for permanent inclusion of vertebroplasty history in the radiological differential for intravascular high-attenuation linear opacities on thoracic CT.

2. Case Presentation

2.1 Clinical History and Background

A 50-year-old female presented to the outpatient department for routine pre-employment medical fitness evaluation. She reported a 15-day history of cough and breathlessness. She explicitly denied chest pain, fever, hemoptysis, and night sweats. There was no history of significant anorexia or weight loss. On direct and detailed history-taking, the patient disclosed that approximately ten years prior (in 2015) she had sustained a road traffic accident (RTA) resulting in a

compression fracture of the L2 vertebral body. She had subsequently undergone percutaneous vertebroplasty with PMMA cement augmentation at an outside tertiary facility. The procedure had been uneventful, with no documented immediate post-procedural complications. She remained ambulatory and largely asymptomatic regarding spinal symptoms after surgery. She had no recollection of having undergone a post-procedural chest radiograph or CT scan following the vertebroplasty in 2015.

General physical examination was unremarkable. Vital signs were stable: blood pressure 118/76 mmHg, heart rate 78 beats/minute, respiratory rate 18 breaths/minute, and SpO2 98% on room air. Chest auscultation showed normal breath sounds bilaterally, with no wheeze, crepitations, or pleural rub. There was no peripheral lymphadenopathy or digital clubbing.

2.2 Initial Imaging: Chest Radiograph

A standard posteroanterior (PA) chest radiograph was obtained as the primary investigation for pre-employment fitness evaluation. The radiograph demonstrated the following salient findings:

- Linear, hyperdense (radio-opaque), branching opacities in the left lung field and the right parahilar region- an atypical pattern that raised the differential of vascular foreign material, endobronchial pathology, or less commonly, parasitic infection.
- Fibrotic changes with mild volume loss in the right upper zone- consistent with sequelae of prior pulmonary infection.

The combination of these findings was considered sufficiently abnormal to warrant further cross-sectional characterization, and the patient was referred for HRCT of the thorax with and without intravenous contrast enhancement.

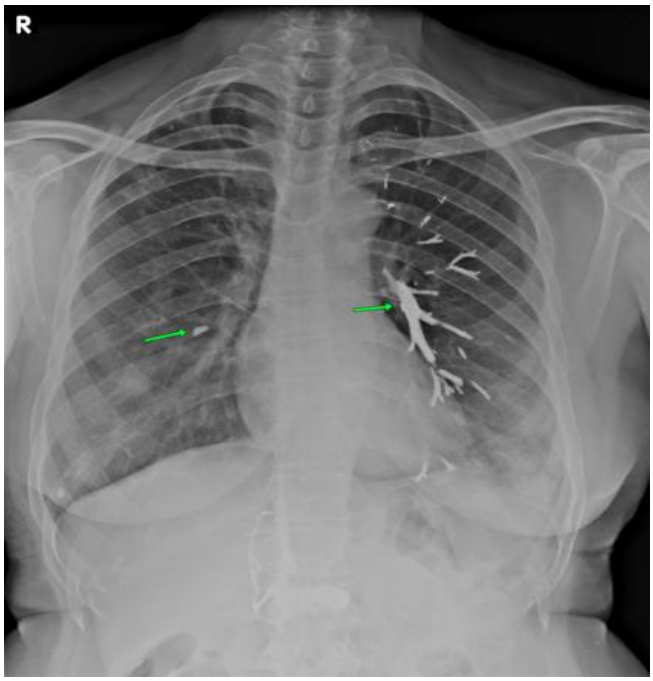

Figure 1: Posteroanterior (PA) chest radiograph. Linear hyperdense branching opacities (radio-opaque) are visible in the left lung field and the right parahilar region, consistent with intravascular high-density foreign material.

2.3 HRCT Thorax: Plain and Contrast-Enhanced Findings

HRCT of the thorax was performed on a multidetector CT scanner using dedicated lung (window width 1500 HU, window level -500 HU) and mediastinal (window width 350 HU, window level 40 HU) window protocols. Both pre-contrast and post-contrast acquisitions were obtained. The findings are described systematically below according to aetiological category for reporting clarity.

2.3.1 Pulmonary Parenchymal Findings: Active Tuberculosis and Sequelae

The following findings were identified within the lung parenchyma on lung window settings:

- Mild fibro-bronchiectatic changes with adjacent pleural and septal thickening involving the apical and posterior segments of the right upper lobe- indicative of fibro-cavitary sequelae of prior granulomatous (tuberculous) infection.
- Multiple centrilobular branching opacities exhibiting a tree-in-bud morphology, with focal coalescence into patchy areas of consolidation, involving the apical segment of the right upper lobe and the superior segment of the right lower lobe- consistent with active endobronchial spread of tuberculosis.
- A few centrilobular branching opacities in the lingular segment of the left upper lobe- likely representing early endobronchial involvement.
- Mild bronchial wall thickening throughout the right lung parenchyma- consistent with infectious bronchiolitis in the context of active tuberculous disease.
- An ill-defined patchy area of consolidation along the adjacent fissure in the superior segment of the right lower lobe, measuring average attenuation values of +30 to +50 HU. The lesion demonstrated spiculated margins, internal calcific foci, and an absence of air bronchogram. On post-contrast series, no significant enhancement was identified- findings collectively most consistent with an old infective fibrotic focus, likely of tuberculous origin.
- A few linear atelectatic bands in the lateral basal segment of the right lower lobe.
- Patchy mosaic attenuation in the bilateral lung parenchyma- most likely representing air trapping secondary to small airway disease or obliterative bronchiolitis. An expiratory HRCT series was recommended for confirmation.

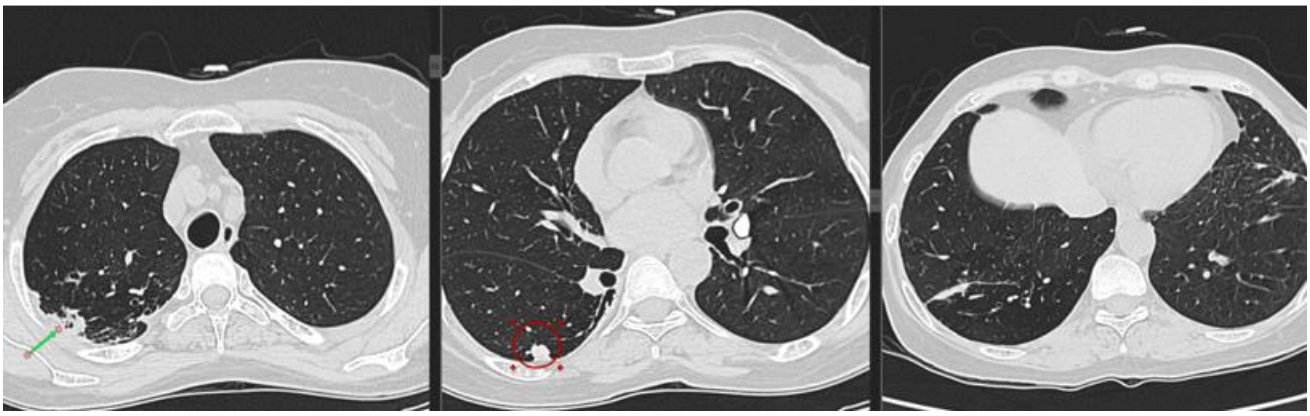

Figure 2(A) **Figure 2(B)** **Figure 2(C)**

Figure 2 (A, B and C): HRCT thorax, lung window, axial sections. (A) Right upper lobe: fibro-bronchiectatic changes and pleural thickening (arrow) with superimposed centrilobular branching tree-in-bud opacities indicating active endobronchial spread of tuberculosis. (B) Superior segment, right lower lobe: coalescent centrilobular opacities forming patchy consolidation (ellipse); an old fibrotic focus with calcific foci and spiculated margins along the fissure. (C) Bilateral lung parenchyma: patchy mosaic attenuation pattern consistent with air trapping from small airway disease.

2.3.2 Vertebral and Vascular Findings: PMMA Cement and Embolic Pathway

The following findings were identified on mediastinal window settings, facilitated by the pre-contrast series to optimally demonstrate the high-attenuation cement:

- Near-total anterior and central wedge compression collapse of the L2 vertebral body with PMMA bone cement in situ - consistent with the history of percutaneous vertebroplasty performed in 2015 following the RTA.
- Linear branching hyperdensities (attenuation +3000 to +3070 HU) within the right paravertebral region- representing cement that extravasated into the paravertebral (Batson's) venous plexus and has remained static within these channels.
- Multiple similar linear branching hyperdensities within the lumen of the inferior vena cava (IVC)- confirming cephalad migration of cement through the ascending lumbar veins into the systemic venous system.
- Linear branching hyperdensities within the segmental and subsegmental pulmonary arteries supplying the left lung parenchyma and the superior segment of the right lower lobe- consistent with and diagnostic of pulmonary cement embolism (PCE). These emboli were peripherally distributed and did not involve the main, lobar, or proximal segmental pulmonary arteries.

Taken in aggregate, these findings delineated the complete anatomical embolic pathway: from the L2 vertebral body cement depot, through extravasation into the right paravertebral venous plexus, cephalad migration via the ascending lumbar veins into the IVC, transit through the right heart, and distribution into the peripheral pulmonary arterial vasculature (Figure 3). No right heart dilatation or paradoxical septal motion was identified. No pleural effusion was present.

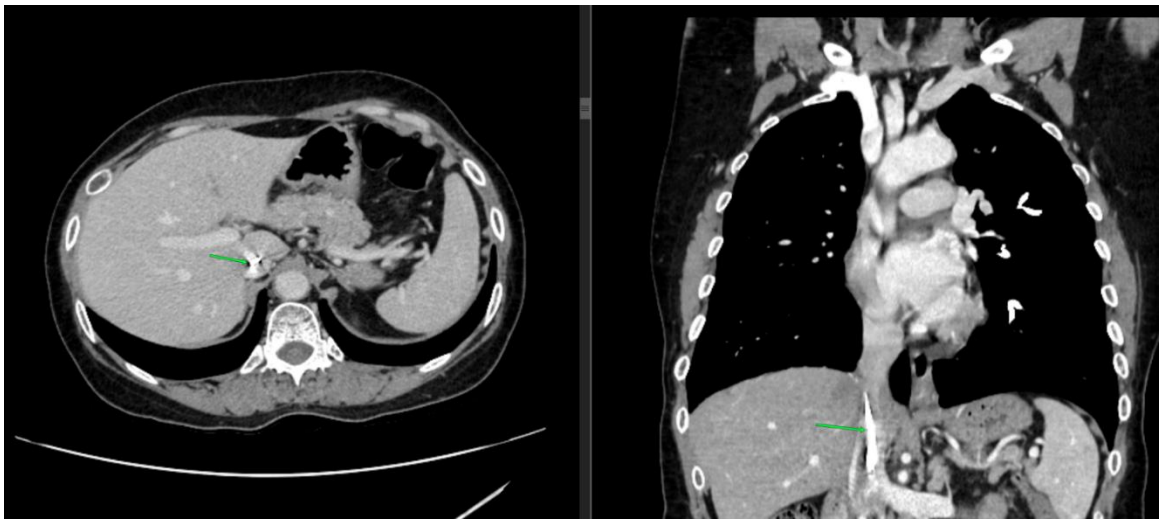

Figure 3: HRCT thorax and upper abdomen, post contrast images (Axial and coronal in venous phase) at the level of the IVC: linear hyperdense cement within the IVC lumen (arrows).

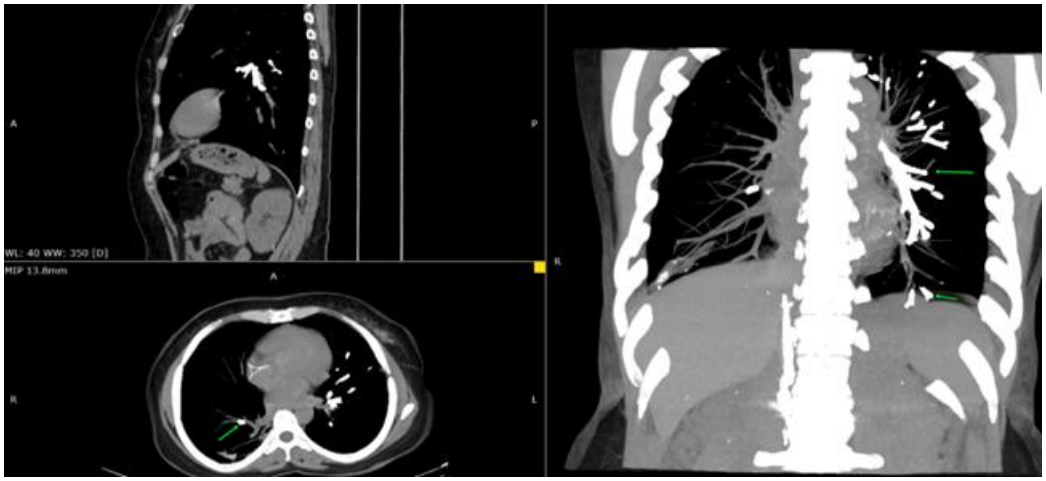

Figure 4: HRCT thorax plain images (sagittal, axial MIP and coronal MIP) at the pulmonary hila: linear branching hyperdensities within segmental and subsegmental pulmonary arteries of the left lung parenchyma and superior segment of right lower lobe (arrows).

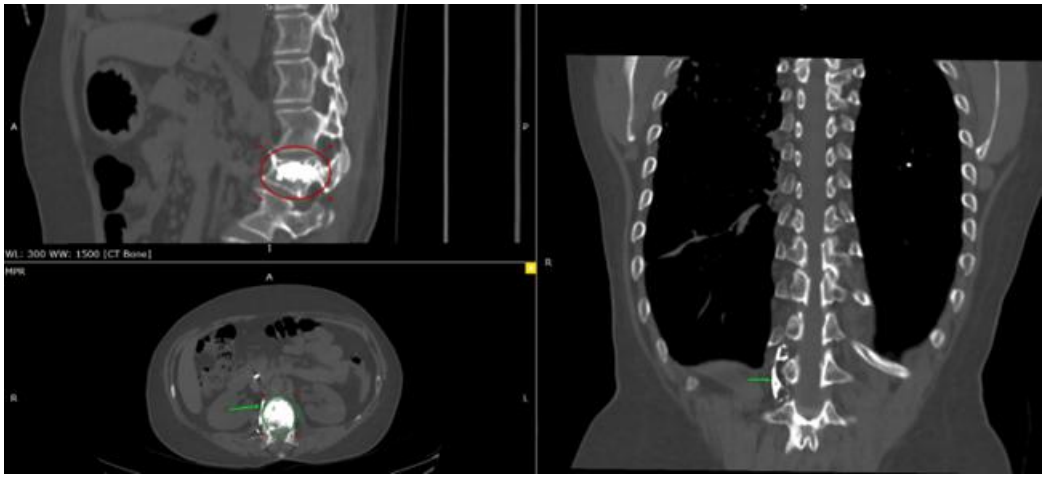

Figure 5: HRCT Thorax bone window (Axial, sagittal and coronal reconstruction) showing L2 vertebral body with PMMA cement in situ (ellipse), post-traumatic wedge collapse, and cement extending into the paravertebral plexus delineating the embolic route (arrows).

2.4 Integrated Radiological Impression

The HRCT findings, considered in totality, were most consistent with the following dual pathology:

- Active pulmonary tuberculosis (Koch's disease) with evidence of active endobronchial spread (tree-in-bud pattern, centrilobular nodules, coalescent consolidation), superimposed on fibro-bronchiectatic and fibrocalcific sequelae of prior pulmonary tuberculosis.
- Incidental pulmonary cement embolism (PCE) as a delayed complication of percutaneous vertebroplasty performed at L2 in 2015 following an RTA, with clear delineation of the full embolic pathway from the vertebral body through the paravertebral venous plexus, IVC, and into the peripheral pulmonary arterial vasculature.

Clinical and laboratory correlation was recommended, including sputum examination for acid-fast bacilli (AFB), GeneXpert MTB/RIF nucleic acid amplification testing, CBNAAT, liver function tests (for anti-tubercular therapy monitoring), and echocardiography to assess right heart function in the context of PCE.

3. Discussion

3.1 Post-Traumatic Vertebroplasty: An Expanding Indication

Percutaneous vertebroplasty was originally described by Galibert and colleagues in 1987 for the treatment of vertebral hemangiomas, and was subsequently extended to osteoporotic vertebral compression fractures — the most common indication globally [1,10]. However, its role in post-traumatic vertebral fractures, particularly stable burst fractures and wedge compression fractures of the thoracolumbar spine following high-energy trauma such as RTAs, has been the subject of increasing interest and clinical adoption over the past two decades [3,4].

Post-traumatic vertebroplasty offers biomechanical advantages including immediate pain relief, restoration of vertebral body height, and prevention of progressive kyphotic deformity, with the added advantage of avoiding the morbidity of open posterior or anterior spinal reconstruction in selected patients [4]. The L1–L2 junction is among the most commonly affected segments in thoracolumbar RTA

injuries, consistent with the level involved in the present case [11].

The risk profile for cement extravasation and PCE in post-traumatic vertebroplasty may differ from that in osteoporotic disease. Post-traumatic fractures frequently involve disruption of the posterior vertebral wall and cortical integrity, increasing the likelihood of cement extravasation into the epidural space or the paravertebral venous plexus [12]. The younger patient age in post-traumatic cases implies greater physiological resilience, potentially explaining the clinically silent course of PCE observed in the present patient over a decade.

3.2 Pulmonary Cement Embolism: Pathophysiology and Radiological Diagnosis

The embolic pathway of PMMA cement from the vertebral body to the pulmonary vasculature is well characterized. Following injection under pressure, liquid or early-polymerizing cement enters the paravertebral venous system (Batson's valveless plexus) and subsequently the ascending lumbar veins, the IVC, the right atrium, the right ventricle, and finally the pulmonary arterial tree [5,6]. The cement polymerizes and solidifies within these channels, producing the characteristic linear branching intravascular opacities seen on CT [9].

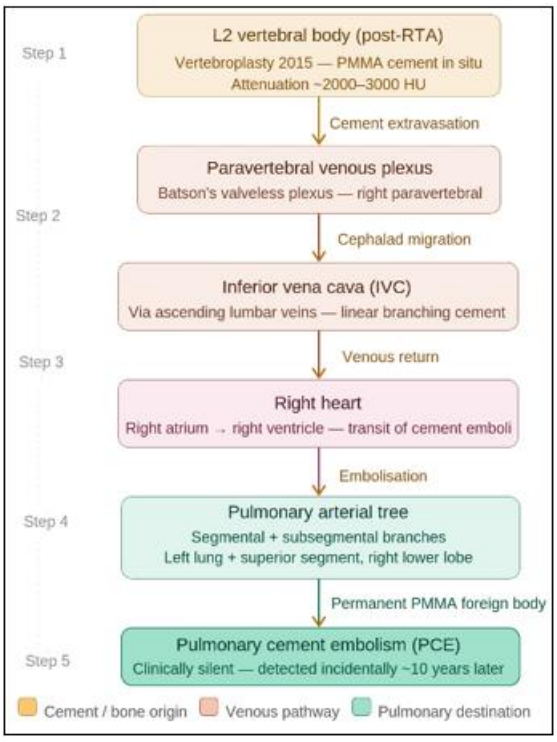

Figure 6: Embolic pathway of PMMA cement following post-traumatic vertebroplasty

The CT diagnosis of PCE is straightforward when actively considered: PMMA cement attenuates at +400 to +1000 HU- substantially higher than any soft tissue, vessel wall, or thrombus- rendering it readily identifiable on both lung and mediastinal window settings [9,13]. The systematic tracing of these hyperdensities from the vertebral body through the IVC to the pulmonary arteries, as demonstrated in the present case, provides definitive confirmation of the embolic pathway.

Reported incidence rates of PCE following vertebroplasty range from 3.5% to 26% when post-procedural CT is performed as a routine investigation, and likely represent a significant underestimate of the true incidence since post-procedural CT is not universally performed [7,8]. The VERTOS II study, which systematically evaluated post-vertebroplasty CT, reported a PCE incidence of 4.6%, with all cases in that series being clinically asymptomatic [8].

3.3 Delayed Detection: A Decade Later

The most distinctive and, to our knowledge, exceptionally rare feature of this case is the interval of approximately ten years between the index vertebroplasty (2015) and the incidental detection of PCE on pre-employment HRCT. The existing literature on PCE predominantly describes detection in the immediate post-procedural period (within hours to days) or on early follow-up CT [14]. Reports of truly delayed detection- years after the procedure- are sparse and typically involve symptomatic presentations rather than incidental discovery [15].

The persistence of PMMA emboli within the pulmonary vasculature for extended periods is physiologically plausible: unlike organic thromboemboli, PMMA does not undergo fibrinolytic dissolution. The cement remains as a permanent intravascular foreign body. Over time, it may become incorporated into the vessel wall, provoke a foreign body giant cell reaction, or serve as a nidus for in situ thrombus formation [16]. The clinical course in patients with peripherally distributed, asymptomatic PCE is generally benign, but long-term sequelae such as pulmonary arterial hypertension have been described in a small number of patients with extensive bilateral embolic burden [17].

The fact that this patient remained asymptomatic with respect to PCE for an entire decade — and was only detected because of an unrelated pre-employment examination- underscores the importance of lifelong vigilance. Any CT scan of the chest, abdomen, or spine in a patient with a history of vertebroplasty should prompt deliberate review of the pulmonary vasculature, IVC, and paravertebral regions for residual or previously undetected cement emboli.

3.4 Concurrent Active Pulmonary Tuberculosis: A Diagnostic Confound

The simultaneous HRCT diagnosis of active pulmonary tuberculosis in this patient introduced considerable diagnostic complexity. The tree-in-bud pattern of centrilobular nodules and branching opacities is one of the most reliable HRCT signatures of active endobronchial spread of tuberculosis and was prominently demonstrated in the right upper lobe and superior segment of the right lower lobe [18]. The fibro-bronchiectatic changes and fibrocalcific nodule with no contrast enhancement confirmed a background of prior, healed tuberculosis- a well-recognized substrate for reactivation disease [18,19].

Crucially, the respiratory symptoms of cough and breathlessness in this patient- the primary presenting complaint- were almost certainly attributable to the active pulmonary tuberculosis rather than to the PCE, which was

clinically silent. This illustrates the potential for anchoring bias during image interpretation: the symptomatic, clinically dominant pathology (tuberculosis) could have drawn all diagnostic attention, leaving the co-existing but asymptomatic PCE undetected. The systematic, structure-by-structure approach to HRCT reporting- encompassing the lung parenchyma, airways, pulmonary vasculature, mediastinum, pleura, chest wall, and visible spine- is the essential safeguard against this diagnostic pitfall.

From a management standpoint, the co-existence of active tuberculosis and PCE creates therapeutic complexity. Standard anti-tubercular therapy (ATT) typically comprises rifampicin, isoniazid, pyrazinamide, and ethambutol in the intensive phase. Anticoagulation- typically recommended for symptomatic or extensive PCE and sometimes for asymptomatic peripheral PCE to prevent in situ thrombosis- carries an increased risk of hemoptysis in the setting of active pulmonary tuberculosis and may interact with rifampicin via cytochrome P450 enzyme induction [20]. Multidisciplinary decision-making involving pulmonology, infectious disease, and hematology teams is therefore essential.

3.5 The Role of Pre-Employment Chest Imaging as an Opportunistic Screen

Pre-employment medical examinations, including chest radiography, remain a standard component of medical fitness certification in many countries, particularly for occupational categories with potential exposure to respiratory hazards. In the present case, the pre-employment chest radiograph served as the initial trigger for further investigation, ultimately leading to the diagnosis of two clinically significant, previously undetected conditions: active pulmonary tuberculosis and PCE.

This case advocates for the recognition of pre-employment chest imaging as an opportunistic, population-level screening opportunity for the early detection of clinically silent but potentially life-threatening conditions- particularly in regions with high tuberculosis burden and a rising prevalence of spinal interventional procedures.

3.6 HRCT as the Definitive Diagnostic Modality

HRCT remains the gold standard for characterization of complex thoracic pathology. In the present case, the multi-window, multi-phase HRCT protocol provided simultaneous and comprehensive assessment of: (a) the lung parenchymal disease (tuberculosis and bronchiolitis on lung window), (b) the high-attenuation cement emboli (on mediastinal and pre-contrast windows), (c) the embolic pathway (mediastinal window with IVC and paravertebral assessment), and (d) the nature of the fibrotic focus (absence of post-contrast enhancement excluding active neoplasm or vascularity).

The additional recommendation for expiratory HRCT to confirm air trapping is consistent with standard practice for cases with suspected small airway disease or obliterative bronchiolitis, which may be subtle or equivocal on inspiratory imaging alone [21].

4. Conclusion

This case demonstrates that pulmonary cement embolism may remain clinically silent for many years after post-traumatic vertebroplasty and may be detected incidentally during imaging performed for unrelated conditions. The coexistence of active pulmonary tuberculosis further increased diagnostic complexity and emphasized the importance of systematic HRCT evaluation. Recognition of characteristic high-attenuation branching intravascular opacities and careful tracing of the embolic pathway are essential for accurate diagnosis. Radiologists and clinicians should remain aware that pulmonary cement embolism can persist indefinitely and retain lifelong diagnostic relevance following vertebroplasty.

Take-Home Message: *In any patient with a history of percutaneous vertebroplasty- whether for osteoporotic or post-traumatic fracture, and irrespective of how remote the procedure — the detection of linear branching high-attenuation opacities on thoracic CT should immediately prompt systematic tracing of the complete embolic pathway and confirmation of pulmonary cement embolism. HRCT, when interpreted comprehensively and with meticulous attention to all anatomical compartments, is capable of simultaneously diagnosing multiple co-existing pathologies and providing a complete, clinically actionable radiological report. Pulmonary cement embolism carries no expiry date: it remains diagnostically relevant for the lifetime of the patient.*

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