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Case Report

Legacy Exposure and Regulatory Gaps: Advanced Lung Adenocarcinoma in a Lifelong Non-Smoker following Prolonged Occupational Asbestos Exposure

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Abstract: Asbestos exposure remains a significant occupational health hazard, particularly in developing countries where its use persists in industrial applications. This case report describes a 61-year-old lifelong non-smoker diagnosed with advanced lung adenocarcinoma (T2N3M1a, Stage IV) with a substantial history of occupational etiology. The patient presented with persistent hoarseness initially misdiagnosed as upper respiratory tract infection, leading to delayed definitive evaluation. A detailed occupational history revealed approximately four decades of cumulative exposure to asbestos during roles involving boiler maintenance and insulation handling in a palm oil mill. Notably, early employment years lacked essential safety measures, including personal protective equipment, engineering controls, and exposure monitoring. Radiological investigations, including contrast-enhanced computed tomography and PET-CT, confirmed metastatic disease, while histopathology established lung adenocarcinoma. Despite advanced malignancy, carcinoembryonic antigen (CEA) levels were not elevated, highlighting the recognized limitations of CEA as a standalone biomarker. Potential mechanisms reported in the literature include tumor heterogeneity and variation in CEACAM5 expression; however, these mechanisms were not evaluated in this patient and therefore remain speculative in the context of this case. Critically, this case illustrates systemic gaps in occupational health governance. The patient's exposure began prior to the implementation of Malaysia's Occupational Safety and Health Act 1994 and continued under suboptimal enforcement conditions, reflecting limitations in regulatory oversight and the absence of retrospective protection for legacy-exposed workers. The lack of structured medical surveillance further contributed to delayed detection of malignancy. This case emphasizes the need for strengthened enforcement, establishment of exposure registries, and implementation of long-term surveillance programs for high-risk occupational cohorts. It also highlights the importance of integrating occupational history into routine clinical assessment. Aligning with Sustainable Development Goals 3, 8, and 12, this report advocates for a shift towards preventive occupational health strategies to reduce the burden of asbestos-related diseases and improve worker safety globally.

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1. Introduction

Asbestos is a group of naturally occurring fibrous silicate minerals historically valued for their heat resistance, tensile strength, and insulating properties [1]. Among its forms, chrysotile (white asbestos) has been the most widely used, particularly in boiler insulation, construction materials, and industrial applications [1]. However, its extensive use has resulted in significant occupational health consequences, with overwhelming evidence now establishing asbestos as a critical hazard in workplace environments [2].

Exposure occurs through inhalation of airborne fibers released in the air when the product contains asbestos being disturbed during the handling, or deterioration of asbestos-containing materials. These microscopic fibers can penetrate deep into the lung parenchyma, where they persist and induce chronic inflammation, progressive fibrosis (asbestosis), and malignant transformation [3]. The International Agency for Research on Cancer classifies all forms of asbestos, including chrysotile, as Group 1 carcinogens, with established links to lung cancer, malignant mesothelioma, and other malignancies [1,4]. Asbestos management in occupational settings in Malaysia is regulated under the Occupational Safety and Health (Use and Standards of Exposure of Chemicals Hazardous to Health) 2000 regulation, which fixes the eight-hour time-weighted average for all types of asbestos except crocidolite at 0.1 f/mL [1]. Crocidolite is banned in Malaysia under Occupational Safety and Health (Prohibition of Use of Substance) Order 1999 except for research or analytical purposes [5].

A hallmark of asbestos-related diseases is their prolonged latency period, often spanning 15 to 30 years, while some articles determine 20 to 30 years [1,3], which complicates early diagnosis and obscures the occupational origin of disease. High-risk groups include workers in construction, manufacturing, and shipbuilding, particularly in settings where historical exposure controls and health surveillance were insufficient [1]. This case report highlights a clinically significant presentation of asbestos-related disease, illustrating the long-term consequences of occupational exposure which result in Asbestos-related lung adenocarcinoma which is a predominant subtype of non-small cell lung cancer. In Malaysia, Department of Occupational Safety and Health (DOSH) statistics recorded only 9 cases of occupational cancer from 2021 to 2024, suggesting that occupational malignancies may remain substantially under-recognized and under-reported despite the presence of established workplace carcinogenic exposures [6]. No specific data on asbestos-related registry is available in the national DOSH occupational disease and cancer registries. Another study highlighted limitations in the documentation and surveillance of occupational cancers arising from the lack of integrated electronic medical databases in Malaysia. Reliance on manual records restricted data integration and systematic analysis, potentially contributing to incomplete case ascertainment and limitations in national occupational cancer statistics [7].

The novelty of this case does not lie in establishing asbestos as a cause of lung cancer or in introducing a new diagnostic modality, as these are already well established. Rather, the approach integrates retrospective occupational exposure reconstruction, clinical and pathological assessment, and analysis of the historical regulatory context to examine how a long-latency occupational malignancy may remain disconnected from occupational health surveillance for decades. Using this integrated approach, the case identifies a practical gap concerning legacy-exposed workers whose substantial historical carcinogenic exposures may no longer be captured by conventional surveillance systems once exposure or employment has ceased. The study consequently proposes a life-course legacy-exposure pathway linking retrospective exposure identification, longitudinal exposure documentation, risk stratification, long-term medical surveillance, clinical occupational history taking, and occupational disease notification. The proposed pathway is intended as a conceptual and practice-oriented response to the gap illustrated by this case, in addition to the existing medical surveillance programs [1,8,9].

2. Case Report

2.1. Retrospective Occupational Asbestos Exposure Assessment

A 61-year-old male who is a lifelong non-smoker with no history of secondary smoker exposure and no known family history of malignancy, thereby reducing the likelihood of conventional risk factors contributing to the disease. He initially presented with a one-month history of hoarseness of voice with no other symptoms and visited primary care clinics and emergency departments to be treated as laryngitis and upper respiratory tract infections. Subsequently, a visit to an occupational health clinic prompted further clinical evaluation and subsequent diagnosis across multiple tertiary healthcare facilities. No history of significant weight loss.

The patient commenced employment in the palm oil mill in 1985 and had approximately four decades of potential occupational asbestos exposure through successive roles as a fitter, boiler engine driver, maintenance supervisor, steam engineer and subsequently mill manager. During the operational and maintenance-related phases of employment, he was directly involved in repair, removal, handling and disturbance of asbestos-containing boiler insulation (Figure 1). Based on the patient's occupational history, these exposure-generating activities occurred on an approximately weekly basis during relevant work periods. Exposure was assessed semi-quantitatively according to duration, frequency, nature of exposure-generating tasks, proximity to the source, and historical control measures. The potential for exposure was considered greatest during the earlier fitter, boiler operation and maintenance-related roles because these involved direct disturbance of insulation material. Respiratory protective equipment, local exhaust ventilation, quantitative exposure monitoring and structured occupational health surveillance were reportedly absent during the earlier employment period until 1995, when the Occupational Safety and Health Act 1994 was enforced. On this basis, the exposure history was classified semi-quantitatively (Table 1) as prolonged and recurrent occupational exposure, involving approximately weekly exposure-generating activities with periods of potentially substantial direct exposure during disturbance of asbestos-containing boiler insulation. Nevertheless, historical personal air-monitoring measurements and task-specific airborne fiber concentrations were unavailable; therefore, quantitative exposure intensity and cumulative exposure expressed as fiber/mL-years could not be reconstructed. The existence of other types of asbestos, especially crocidolite, at work during early years of the career could not be ruled out due to the unavailability of the information and safety data sheets at the industry.



Figure 1. Used insulator material. Photograph courtesy of a retired worker with permission for publication.

Table 1. Semiquantitative exposure assessment.

Parameter	Evidence in This Case
Exposure commencement	Approximately 1985
Duration	Approximately 40 years
Frequency	Approximately weekly during work periods involving maintenance activities
Exposure source	Boiler insulation material
Exposure-generating activities	Repair, removal, handling and disturbance of insulation
Directness	Direct during fitter/boiler/maintenance-related roles
Early PPE	Reportedly absent until 1995
Early engineering controls	Reportedly absent
Historical air monitoring	Unavailable
Quantitative fibre concentration	Unavailable
Cumulative fibre/mL-years	Cannot be calculated
SDS evidence	Asbestos, Chrysotile; CAS 12001-29-5; >99.99%
2026 laboratory evidence	Sample of Boiler Insulator: asbestos YES; chrysotile YES
Other fibres in Sample of Boiler Insulator	No Actinolite, amosite, anthophyllite, crocidolite, or tremolite detected
Laboratory method	NIOSH 9002
Overall assessment	Prolonged, recurrent, approximately weekly potential occupational exposure

2.2. Assessment of Occupational Attribution

The potential contribution of occupational asbestos exposure to the patient's lung adenocarcinoma was evaluated with reference to the Helsinki Criteria for the diagnosis and attribution of asbestos-related diseases and the subsequent 2014 consensus update [10,11]. Although asbestos bodies are not identified, the existence of chrysotile asbestos bodies is rare and leads to false-negative diagnoses [11]. Several features support the plausibility of an occupational contribution, including an approximately four-decade occupational history, recurrent weekly involvement in asbestos-insulation-disturbing activities, inadequate historical exposure controls, and an approximately 40-year interval between initial exposure and diagnosis. However, definitive individual attribution is limited by the retrospective nature of the exposure assessment. Historical airborne fiber measurements and quantitative cumulative exposure data were unavailable, precluding reconstruction of cumulative exposure in fiber/mL-years. Objective measures of pulmonary asbestos fiber or asbestos-body burden were also unavailable. Consequently, the quantitative exposure criteria within the Helsinki framework could not be confirmed in this patient. The available evidence therefore supports the plausibility of a substantial occupational asbestos contribution to the development of lung adenocarcinoma but is insufficient to establish definitive individual causal attribution.

Chrysotile was identified as the principal asbestos type based on both documentary and laboratory evidence. The available SDS identifies the material as "Asbestos, Chrysotile" (CAS No. 12001-29-5), with the composition specified as >99.99% chrysotile asbestos [12]. To provide additional objective confirmation, solid material samples representative of the insulation material were submitted for accredited laboratory analysis in 2026. Analysis using NIOSH Method 9002 detected asbestos in one of the submitted samples from the insulators, with chrysotile specifically identified, while actinolite, amosite, anthophyllite, crocidolite, and tremolite were not detected [13]. These findings provide contemporary documentation and analytical evidence supporting the presence of chrysotile in the sampled material. The SDS additionally classifies chrysotile as Carcinogenicity Category 1A, and its toxicological section identifies it as a Group 1 carcinogen under IARC [4]. However, because the laboratory analysis was undertaken in 2026 rather than during the historical exposure period, the findings cannot establish that the composition of asbestos-containing materials remained unchanged throughout the patient's approximately four decades of employment. Accordingly, historical co-exposure to other asbestos fiber types cannot be completely excluded.

2.3. Clinical Examination

The patient was alert and oriented, with stable vital signs. However, the presence of hoarseness raised clinical suspicion of possible recurrent laryngeal nerve involvement, a known complication in advanced thoracic malignancies due to tumor compression or mediastinal lymphadenopathy. Respiratory system examination demonstrated reduced air entry over the upper left lung, with symmetric chest expansion. Auscultation revealed diminished breath sounds with crackles over the upper left lung, suggestive of underlying parenchymal involvement. There were no clinical features suggestive of chronic obstructive pulmonary disease, which is consistent with the patient's lifelong non-smoking status. Left supraclavicular and cervical lymphadenopathy had been detected. The patient shows no other significant signs and symptoms on abdominal, cardiovascular, or neurological examination.

2.4. Blood Investigation

Blood investigations were conducted to evaluate the patient's baseline health status and support clinical management. Routine hematological parameters, including complete blood count (CBC), revealed a normal hemoglobin level of 15 g/dL with mild elevations in the neutrophil count (8.26×10^9/L) and white blood cell count of 11.16×10^9/L. No other significant hematological abnormalities were detected [14–16]. Renal and liver function parameters were within the normal ranges [16]. The carcinoembryonic antigen (CEA) level was not elevated (2.4 ng/mL) [17]. C-reactive protein (CRP), CA-125, CA 19-9, and CA 15-3 levels were also within their respective reference ranges [16,18–20].

2.5. Diagnostic Examination

The diagnostic evaluation of this patient was comprehensive and involved multi-modality investigations to confirm the presence, stage, and extent of lung malignancy. Radiological imaging formed the cornerstone of diagnosis. Initial chest imaging, followed by contrast-enhanced computed tomography (CECT) of the thorax, revealed a left lung mass (3.1 cm × 3.7 cm × 4.0 cm) with small lung nodules (0.3 cm) scattered in bilateral lungs.

Bilateral cervical nodes, necrotic left suprascapular nodes, multiple mediastinal nodes with largest of left paratracheal extending to subaortic region measuring 2.5 cm × 4 cm identified. In addition to that, right hilar nodes identified measuring 1.6 cm × 1.6 cm. Mediastinal necrotic nodes with left vocal cord palsy suggestive of mediastinal metastasis.

Staging was further supported by positron emission tomography-computed tomography (PET-CT) to assess metabolic activity and detect distant metastases, which subsequently indicated metastasis (M1a), aligning with the final classification of Stage IV (T2N3M1a). The PET scan identified primary left upper lobe lung malignancy with bilateral intrapulmonary metastasis with extensive bilateral mediastinal nodal involvement. The right vocal cord reflects a compensatory mechanism for contralateral left vocal cord palsy. Figures 2–5 illustrate the findings.



Figure 2. Coronal fused Fluorodeoxyglucose (FDG) PET/CT image demonstrating abnormal hypermetabolic uptake within the thoracic region acquired using a Biograph mCT PET/CT scanner (Siemens Healthineers, Erlangen, Germany).

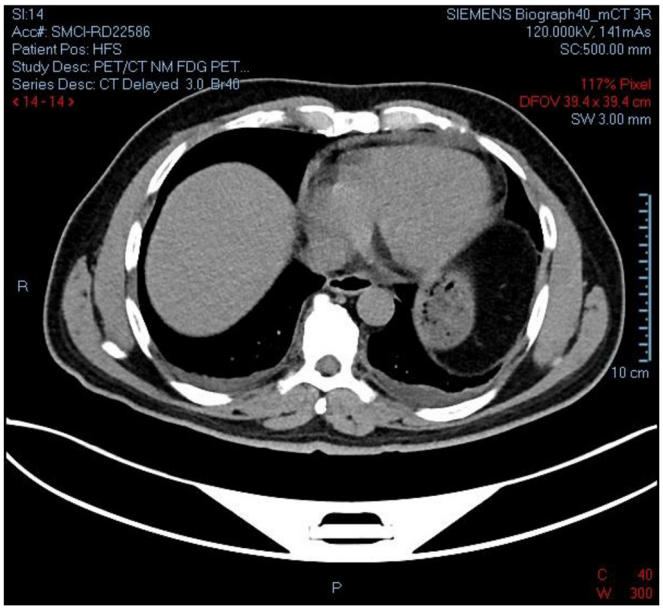


Figure 3. Thoracic PET/CT delayed-phase axial image acquired using a Biograph mCT PET/CT scanner (Siemens Healthineers, Erlangen, Germany).

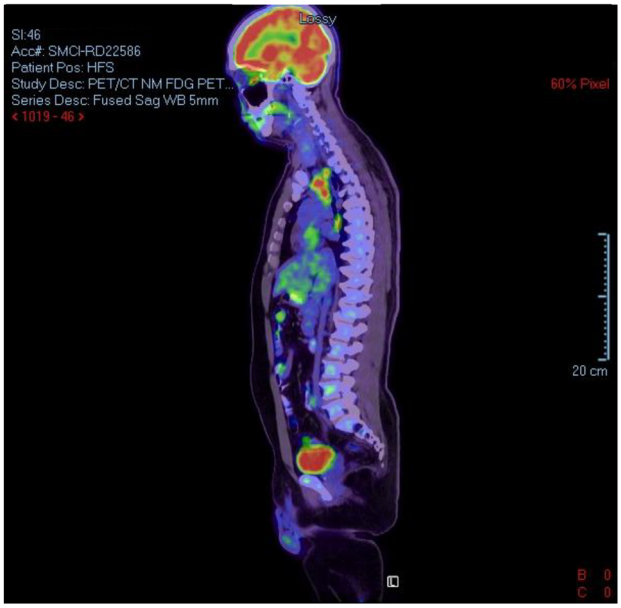


Figure 4. Sagittal fused whole-body FDG PET/CT image demonstrating focal hypermetabolic thoracic activity acquired using a Biograph mCT PET/CT scanner (Siemens Healthineers, Erlangen, Germany).

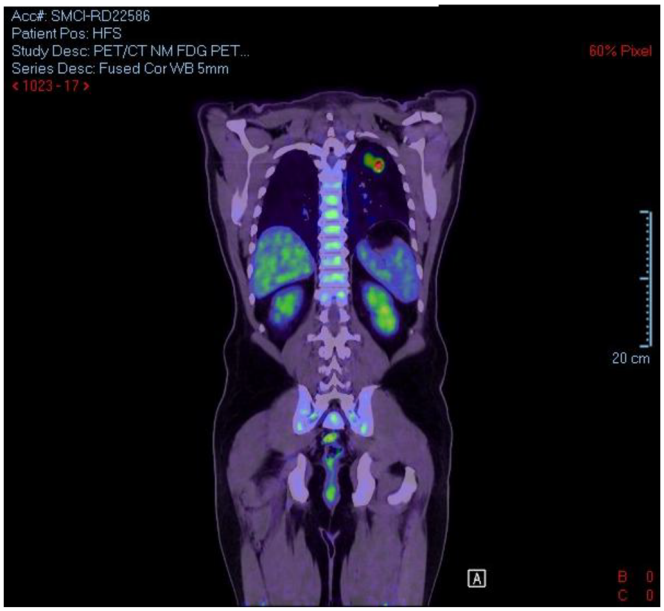


Figure 5. Coronal fused whole-body FDG PET/CT image demonstrating focal hypermetabolic lesion within the upper thoracic region acquired using a Biograph mCT PET/CT scanner (Siemens Healthineers, Erlangen, Germany).

2.6. Histological Examination

Definitive diagnosis was established through histopathological examination of biopsy specimens, confirming invasive non-mucinous lung adenocarcinoma. The sample identified complex glandular structures, cribriform architecture and solid nests with moderate nuclear pleomorphism, hyperchromatic & conspicuous nuclei with pale eosinophilic to focally vacuolated cytoplasm. No asbestos bodies were identified.

Immunohistochemical examination of the left upper lobe biopsy tissue sample demonstrated positive Thyroid transcription factor-1, TTF-1 and negative p40 expression, supporting the diagnosis of pulmonary adenocarcinoma [21]. Anaplastic lymphoma kinase (ALK) protein expression assessed by immunohistochemistry using a monoclonal antibody was negative [22]. However, subsequent molecular testing using next-generation sequencing (NGS) method detected the echinoderm microtubule-associated protein-like 4-anaplastic lymphoma kinase (EML4-ALK) fusion/rearrangement [23]. EGFR testing using the NGS method was negative, while the programmed cell death-ligand 1 (PD-L1) expression assessed using the 22C3 assay showed a tumor proportion score (TPS) of 48% [24,25]. Thus, the apparently discordant ALK findings represent results obtained using different testing modalities: negative ALK protein expression by monoclonal antibodies and detection of an ALK molecular alteration by NGS.

The clinical findings, although not highly specific, were consistent with advanced pulmonary malignancy and, when interpreted alongside imaging and histopathological results, supported the diagnosis of lung adenocarcinoma with a substantial suspicion of occupational etiology [26,27]. A confirmed diagnosis of advanced lung adenocarcinoma (T2N3M1a, Stage IV), in whom a significant occupational etiology is suspected.

3. Discussion

3.1. Biomarker Limitations: The Paradox of Normal CEA in Advanced Disease

The absence of elevated carcinoembryonic antigen (CEA) levels in this patient, despite advanced-stage lung adenocarcinoma, warrants critical appraisal given the widespread use of CEA as a tumor biomarker. Although CEA is frequently utilized for disease monitoring, its sensitivity and specificity in lung adenocarcinoma remain heterogeneous and context-dependent [28,29]. Evidence suggests that CEA expression is not universally elevated across all tumor phenotypes and may vary according to molecular characteristics, tumor differentiation, and biological behavior [28,29]. In addition, the level of CEA depends on health status, smoking, and disease progression and is not a definitive marker of any cancer site [29]. The plausible factors are due to tumor heterogeneity and differential expression of the CEACAM5 gene, which encodes CEA. Recent evidence indicates that lung adenocarcinoma cells undergoing epithelial-mesenchymal transition (EMT) demonstrate reduced CEACAM5 expression, leading to lower circulating CEA levels [28]. This phenomenon is particularly relevant in advanced or treatment-resistant tumors, where cytokine signaling and epigenetic modulation suppress CEA expression. Consequently, tumors with aggressive or dedifferentiated features may not secrete significant amounts of CEA, despite substantial tumor burden [28]. CEA demonstrates variable prognostic and monitoring utilities depending on treatment modality and tumor biology. Studies have shown that baseline CEA is not consistently associated with survival outcomes in all patient groups, particularly those not driven by EGFR-related pathways or those exhibiting alternative oncogenic mechanisms, despite some studies suggesting CEA as a prognostic factor for treatment [28]. This emphasizes that CEA is not a universal biomarker and may lack sensitivity in certain molecular subtypes of lung adenocarcinoma [28,29]. Additionally, the temporal dynamics of CEA expression must be considered. CEA levels may fluctuate during disease progression and can remain within normal ranges even in the presence of radiological progression, reflecting biomarker "non-secretor" status. This is supported by clinical observations where tumor progression occurred without a corresponding rise in CEA levels, highlighting its limited reliability as a standalone indicator [29].

Although reduced CEACAM5 expression and EMT have been proposed as potential mechanisms underlying low circulating CEA levels in some lung adenocarcinomas, these mechanisms cannot be established in the present patient. CEACAM5 expression and EMT-related molecular markers were not assessed in this case. Therefore, the normal serum CEA level (2.4 ng/mL) should be interpreted as illustrating the recognized variability and limited sensitivity of CEA in lung adenocarcinoma rather than as evidence of a specific underlying molecular phenotype. The mechanisms discussed above remain plausible hypotheses based on the existing literature and should not be regarded as patient-specific biological explanations. This case consequently reinforces the importance of integrating serum biomarkers with clinical, radiological, and histopathological findings rather than relying on CEA as a standalone indicator of disease burden.

3.2. Evolving Regulatory Framework and the Persistent Legacy-Exposure Gap

The regulatory context of this case must be interpreted across different periods of Malaysia's occupational safety and health framework development. The patient's occupational exposure began in the mid-1980s, preceding the Occupational Safety and Health Act 1994 (OSH 1994), and subsequently continued during periods in which the Act and its subsidiary regulations were applicable. Furthermore, even in the post-1994 era, enforcement challenges persist. OSH 1994 adopts a self-regulatory framework, placing responsibility on employers to identify hazards, implement control measures, and ensure worker safety [30,31]. However, industries such as palm oil mills and boiler maintenance often operate in semi-regulated environments where compliance may be inconsistent due to limited inspections, inadequate training, and weak safety culture [32]. The continued handling of asbestos-containing materials without proper controls indicates deficiencies in hazard communication, including the use of SDS, and failure to implement chemical health risk assessments and mitigations as required under subsidiary regulations [8,9,33]. Consequently, workers exposed to asbestos prior to implementation remain vulnerable, with no structured requirement for retrospective exposure assessment, medical surveillance, or risk mitigation. This creates a cohort of "legacy-exposed" workers whose occupational risks were neither adequately controlled nor subsequently addressed. In the current case, prolonged exposure without engineering controls or respiratory protection likely resulted in cumulative fiber inhalation, consistent with the latency and severity of the diagnosed

malignancy. Therefore, the case reflects not only historical limitations preceding the establishment of the modern occupational safety and health framework but also the challenges associated with managing the long-term consequences of exposures accumulated across different regulatory periods.

Importantly, Malaysia's occupational safety and health framework has since evolved substantially. The Occupational Safety and Health (Amendment) Act 2022 (Act A1648), which came into force on 1 June 2024, strengthened the existing framework through several important provisions, including broader application of the Act, explicit requirements relating to occupational safety and health risk assessment, additional workplace safety and health responsibilities, and substantially enhanced penalties for non-compliance [34,35]. These amendments represent important progress towards a more preventive and risk-based occupational safety and health system. The revised framework is particularly relevant to the prevention of future hazardous exposures because stronger risk assessment obligations may facilitate earlier identification and control of occupational carcinogens [36]. However, the present case highlights a different challenge that cannot be addressed solely through stronger management of current exposure: the long-term health consequences of hazardous exposures that occurred decades earlier. Workers exposed before contemporary regulatory and surveillance requirements were established may have incomplete exposure records, may no longer work for the original employer, and may remain outside workplace-based surveillance despite continuing to carry an elevated risk of long-latency occupational disease.

Accordingly, this case should not be interpreted as suggesting that the current Malaysian occupational safety and health framework remains unchanged from the Occupational Safety and Health Act 1994. Rather, it demonstrates a persistent legacy-exposure gap despite subsequent regulatory advancement. The Occupational Safety and Health (Amendment) Act 2022 strengthens prospective prevention and risk management, while historical exposure requires a complementary retrospective occupational health approach. Therefore, the researchers propose that the contemporary regulatory framework be complemented by mechanisms for identifying and following workers with significant historical exposure to recognized occupational carcinogens. Such an approach could include retrospective exposure identification, longitudinal exposure registries, risk stratification, long-term medical surveillance, systematic occupational history taking during cancer assessment, and appropriate occupational disease notification. This would bridge the gap between strengthened prospective prevention under the current regulatory framework and the continuing health consequences of occupational exposures that occurred under earlier regulatory conditions.

3.3. Legacy-Exposed Workers and the Imperative for Structured Medical Surveillance: Addressing Long-Term Occupational Cancer Risk

A critical implication arising from this case is the need to formally recognize and manage a "legacy-exposed cohort" of workers who experienced significant occupational hazards prior to, or during the early implementation of, regulatory frameworks such as the Occupational Safety and Health Act 1994 [1]. The absence of adequate exposure control measures through structured risk assessments and risk dissolution measures has likely resulted in a population of workers with cumulative, undocumented exposure to asbestos. Given the well-established latency period of asbestos-related malignancies, failure to identify and monitor this cohort represents a missed opportunity for early detection and secondary prevention [10].

From a policy and occupational health perspective, there is a compelling need for systematic cohort identification through employer records, industry registries, and collaboration with regulatory bodies. The existing medical surveillance program stipulated by DOSH is not adequate in view of not including historical workers who had been exposed to asbestos [1,8]. Workers with historical exposure, particularly in high-risk sectors such as boiler maintenance and palm oil processing, should be stratified based on duration, intensity, and nature of exposure in Malaysia. This risk-based classification would enable targeted medical surveillance, aligning with international best practices for carcinogen-exposed populations [37–39]. This program will complement the enforcement of asbestos-related exposure management by the Act.

A structured medical surveillance program should be implemented, incorporating baseline and periodic clinical evaluation, pulmonary function testing, and imaging modalities such as low-dose computed tomography (LDCT), which has demonstrated utility in early lung cancer detection among high-risk groups [10,40–42]. Biomarker monitoring, although limited in sensitivity, may be used adjunctively, while recognizing its constraints as highlighted in this case [28,43–45]. Importantly, surveillance programs must be longitudinal, extending over decades, to account for the prolonged latency of asbestos-related diseases [1,46–47]. Beyond surveillance, continuity of care is essential. Workers identified within this cohort should be enrolled in dedicated occupational health follow-up systems through a formal registry that ensures timely referral, diagnostic evaluation, and intervention. This includes integration with national cancer screening initiatives and multidisciplinary care

pathways. Additionally, health education and risk communication should be prioritized to enhance awareness of symptom recognition and encourage early healthcare-seeking behaviors.

Critically, such initiatives should not be viewed as discretionary but as an ethical and regulatory responsibility. The principle of "duty of care" extends beyond active employment and must encompass long-term health outcomes associated with occupational exposure [1,48]. Without proactive identification and surveillance of this at-risk cohort, the burden of disease will continue to manifest silently, often at advanced stages, as demonstrated in this case [1,10]. From a policy standpoint, amendments to existing legislation on respective regulations on chemical health hazard management should be considered to address legacy exposures, including provisions for retrospective risk assessment, compulsory employer reporting of historical exposures, and extended employer liability for long-term health outcomes. This approach not only strengthens occupational health governance but also aligns with preventive medicine principles aimed at reducing morbidity and mortality from avoidable workplace hazards.

3.4. Advancement beyond Current Research

Previous research has established the causal relationship between occupational asbestos exposure and long-latency respiratory malignancies, and several international studies have demonstrated the value of exposure registries and longitudinal surveillance of asbestos-exposed workers [47,49]. For example, the Ontario Asbestos Workers Register has enabled long-term linkage of documented occupational exposure with subsequent asbestos-related diseases, including lung cancer, mesothelioma, asbestosis, and pulmonary fibrosis [49]. A similar program has been done in Italy through risk-based epidemiological asbestos-exposed workers identification, where workers have been enrolled in a systemic surveillance program [47]. Ironically, surveillance programmes involving formerly asbestos-exposed workers have demonstrated that asbestos-related cancer risks remain clinically relevant decades after occupational exposure had ceased. Therefore, the present case does not propose exposure registries or long-term surveillance as entirely new concepts. Rather, its contribution lies in demonstrating their relevance to an apparent gap in the Malaysian occupational health context. The patient developed advanced lung adenocarcinoma approximately four decades after the beginning of occupational asbestos exposure, with inadequate exposure control, exposure documentation, and structured medical surveillance during the earlier period of employment. By the time malignancy was diagnosed, the historical occupational exposure and the clinical disease were separated by several decades.

This case therefore advances the current evidence by highlighting the need to bridge historical occupational exposure and contemporary clinical diagnosis within the Malaysian occupational health system. Specifically, researchers propose that workers with substantial historical exposure to recognized occupational carcinogens should remain identifiable as a legacy-exposed population even after exposure or employment has ceased. Historical exposure identification should be linked to longitudinal exposure records, risk-based medical surveillance, systematic occupational history taking when malignancy is diagnosed, and appropriate occupational disease notification. Accordingly, the contribution of this case is not the demonstration that asbestos causes lung cancer, which is already well established, but the identification of a practical translation gap: workers with historical carcinogenic exposure may become disconnected from occupational health surveillance during the prolonged interval between exposure and disease manifestation. Addressing this gap may improve recognition and notification of long-latency occupational cancers and provide a basis for developing a Malaysian legacy-exposure surveillance pathway.

4. Conclusions

This case demonstrates the severe long-term consequences of occupational asbestos exposure and highlights how inadequate enforcement, poor early risk management, and absence of medical surveillance may result in advanced malignancy decades later. In a lifelong non-smoker, the diagnosis of lung cancer occurred in a worker with a substantial history of occupational asbestos exposure. The case also reflects broader systemic weaknesses in occupational health governance, emphasizing the urgent need for preventive strategies focused on early hazard identification, strict regulatory enforcement, and long-term monitoring of exposed workers. Strengthening regulatory enforcement, addressing legacy exposures, and implementing sustained medical surveillance are critical.

Based on this case, we propose the development of a national risk-based registry and long-term medical surveillance framework for workers with significant historical exposure to asbestos and other occupational carcinogens. The proposed framework comprises retrospective identification of exposed workers, establishment of a longitudinal exposure registry, risk stratification according to exposure characteristics, long-term medical surveillance, timely investigation of suspected malignancy, and appropriate occupational disease notification.

This approach is particularly relevant for long-latency occupational cancers, for which the absence of active workplace exposure at the time of diagnosis may otherwise obscure the occupational origin of disease. Such an approach may also address an important diagnostic gap. When cancer develops decades after exposure, the occupational relationship may be overlooked unless a detailed occupational history is actively incorporated into clinical assessment. The normal CEA level in this patient further demonstrates that occupational cancer recognition cannot depend on an isolated biomarker and requires integration of exposure history with clinical, radiological, and histopathological findings. Thus, the key lesson from this case extends beyond asbestos exposure itself: occupational responsibility for carcinogenic exposure should not end when workplace exposure ends.

This case serves as a compelling reminder that occupational health risks, if unaddressed, transcend time and continue to manifest as preventable disease burdens despite the fact that crocidolite has been prohibited except for research and analytical purposes in Malaysia for the past 30 years. Strengthening enforcement, addressing legacy exposures, and embedding sustainable occupational health practices are imperative to safeguard future generations of workers and to achieve broader global health and development accomplishment. The occupational health implications of this case are most directly aligned with Sustainable Development Goal (SDG) 3 (Good Health and Well-being), particularly through the prevention and early detection of occupational disease, and SDG 8 (Decent Work and Economic Growth), through the promotion of safe and secure working environments [50].

Author Contributions

A.K.R.: Conceptualization, methodology, investigation, formal analysis, data curation, project administration, visualization, writing, original draft preparation, writing, review and editing, and correspondence. R.B.I.: Supervision, methodology, validation, formal analysis, writing, review and editing. A.N.A.: Supervision, methodology, validation, formal analysis, writing, review and editing. R.K.S.: Investigation, methodology, validation, writing, review and editing. M.D.S.: Investigation, data curation, visualization, validation, writing, review and editing. I.H.B.Z.: Investigation, data curation, validation, methodology, writing, review and editing. All authors have read and agreed to the published version of the manuscript.

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Institutional Review Board Statement

For the case report, written informed consent for publication was obtained from the patient. The study was conducted according to the guidelines of the Declaration of Helsinki and registered with National Medical Research Registry (NMRR), Ministry of Health of Malaysia with the ID number of RSCH ID-26-07274-KLP. Ethical review and approval were waved for this study, due to single-patient case report in accordance with the NIH Guidelines for Conducting Research in Ministry of Health Institutions & Facilities, 3rd Edition (2021), which identifies case reports or studies with informed consent from patients are among the research activities that do not require Medical Research and Ethics Committee (MREC), Ministry of Health Malaysia ethical approval. Written informed consent was obtained from the patient for the publication of the clinical information presented in this report. All potentially identifying information was omitted or appropriately anonymized to safeguard the patient's privacy and confidentiality.

Informed Consent Statement

Written informed consent was attained from the patient for the publication of the case.

Data Availability Statement

The data supporting the findings of this study are available from the corresponding author upon reasonable request. All datasets have been anonymized to maintain participant confidentiality, and access may be restricted due to institutional, ethical, and privacy considerations.

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Conflicts of Interest

The authors declare no conflict of interest.

Use of AI and AI-Assisted Technologies

During the preparation of this work, the authors used ChatGPT to improve the language and correct typos and grammatical errors. After using this tool, the authors reviewed and edited the content as needed and took full responsibility for the content of the published article.

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