Review Article

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Pulmonary Manifestations and Complications of Type 2 Diabetes Mellitus

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Abstract
Type 2 diabetes mellitus (T2DM) is a chronic multisystem disorder traditionally linked to vascular complications; however, the lung is increasingly recognized as an important yet underappreciated target organ. This narrative review summarizes current evidence on the pathophysiology, clinical manifestations, and therapeutic implications of pulmonary involvement in T2DM.

Chronic Hyperglycemia and insulin resistance contribute to lung injury through microangiopathy, endothelial dysfunction, oxidative stress, inflammation, and accumulation of advanced glycation end-products. These mechanisms lead to structural remodeling of the alveolar capillary membrane, reduced lung compliance, and impaired gas exchange. Clinically, patients with T2DM commonly exhibit restrictive lung disease, reduced lung volumes, and decreased diffusing capacity, with an increased risk of interstitial lung disease and pulmonary fibrosis.

Diabetes also predisposes individuals to respiratory infections, as highlighted during the COVID-19 pandemic, where increased ACE2 expression and a pro-fibrotic state contribute to severe outcomes and post-infection fibrosis. Emerging biomarkers such as surfactant protein D and markers of cellular senescence, along with genetic predisposition, may aid early detection.

Certain glucose-lowering therapies, particularly SGLT-2 inhibitors, show potential benefits in respiratory outcomes. Recognizing the "diabetic lung" is essential for comprehensive diabetes care and improving long-term patient outcomes.

Abbreviations
ACE2: Angiotensin-Converting Enzyme 2
AGEs: Advanced Glycation End-products
BMI: Body Mass Index
COPD: Chronic Obstructive Pulmonary Disease
COVID-19: Coronavirus Disease 2019
DLCO: Diffusing Capacity of the Lung for Carbon Monoxide
FEV1: Forced Expiratory Volume in One Second
FVC: Forced Vital Capacity
ILD: Interstitial Lung Disease
NO: Nitric Oxide
PARP: Poly (ADP-ribose) Polymerase
PGI2: Prostacyclin
RLD: Restrictive Lung Disease
SARS-CoV-2: Severe Acute Respiratory Syndrome Coronavirus 2
SASP: Senescence-Associated Secretory Phenotype
SGLT-2: Sodium-Glucose Cotransporter-2
SP-D: Surfactant Protein D
SLE: Systemic Lupus Erythematosus
T2DM: Type 2 Diabetes Mellitus
TGF-β1: Transforming Growth Factor Beta 1
THBS1: Thrombospondin-1
TLC: Total Lung Capacity
TxA2: Thromboxane A2

Introduction

Type 2 diabetes mellitus (T2DM) is a chronic, systemic metabolic disorder characterized by insulin resistance and progressive pancreatic beta-cell failure, leading to chronic hyperglycemia. While the clinical management of T2DM has traditionally focused on classic microvascular complications—such as retinopathy, nephropathy, and neuropathy—and macrovascular diseases like coronary artery disease, emerging evidence suggests that the lung is also a target organ for diabetic damage. The pulmonary system possesses an extensive microvascular bed and a large surface area of connective tissue, both of which are susceptible to the biochemical alterations induced by chronic hyperglycaemia. Recent epidemiological and clinical studies have identified a significant association between T2DM and various respiratory impairments, including restrictive lung disease (RLD), interstitial lung disease (ILD), and increased susceptibility to infectious agents such as SARS-CoV-2. Despite the growing body of evidence, the diabetic lung remains a relatively under-recognized clinical entity in standard endocrine and respiratory practice, necessitating a comprehensive review of the underlying mechanisms and clinical implications [1-4].

The pathophysiology of lung involvement in T2DM is multifactorial, involving a complex interplay of biochemical, structural, and genetic factors. Chronic hyperglycemia leads to the accumulation of advanced glycation end-products (AGEs), which can cross-link collagen and elastin in the pulmonary parenchyma, resulting in reduced lung compliance and impaired gas exchange. Furthermore, the pulmonary vasculature undergoes changes similar to those seen in the kidneys and retina, characterized by basement membrane thickening and endothelial dysfunction. These structural alterations are often accompanied by systemic low-grade inflammation and oxidative stress, which further exacerbate tissue damage and promote fibrotic signaling pathways. Interestingly, recent genetic studies have begun to identify specific polymorphisms, such as those in the MUC5B gene, that may predispose certain diabetic individuals to developing interstitial complications, suggesting that genetic susceptibility plays a crucial role alongside metabolic derangements [1-5].

Clinically, pulmonary dysfunction in T2DM often manifests as a subtle reduction in lung volumes and diffusing capacity for carbon monoxide (DLCO). While these changes may be asymptomatic in the early stages, they can progress to significant restrictive patterns and breathlessness, particularly in patients with long-standing disease or poor glycemic control. The presence of comorbidities such as obesity and systemic lupus erythematosus (SLE) further complicates the respiratory profile of diabetic patients, sometimes presenting paradoxical findings in terms of disease prevalence and severity. Moreover, the COVID-19 pandemic has highlighted the vulnerability of the diabetic population, as T2DM is associated with increased expression of the ACE2 receptor in lung tissue and a higher risk of post-infection pulmonary fibrosis. Understanding these interactions is vital for developing targeted therapeutic strategies, including the potential protective roles of modern glucose-lowering agents like SGLT-2 inhibitors and sulfonylureas in managing comorbid chronic obstructive pulmonary disease (COPD). This review aims to synthesize current knowledge on the pulmonary manifestations of T2DM, exploring the transition from molecular mechanisms to clinical phenotypes and therapeutic management [6-11].

Pathophysiological Mechanisms of Lung Injury in Type 2 Diabetes

Microangiopathy and Endothelial Dysfunction in the Pulmonary Vasculature

The pulmonary circulation is a high-flow, low-pressure system that relies on a delicate balance of vasoactive mediators to maintain optimal gas exchange. In the context of T2DM, this balance is disrupted by chronic hyperglycaemia and insulin resistance, leading to what is termed & diabetic endothelial microangiopathy. Similar to the microvascular changes observed in the retina and renal glomeruli, the pulmonary capillaries in diabetic patients exhibit significant basement membrane thickening. This structural remodelling is driven by the non-enzymatic glycation of proteins and the subsequent formation of advanced glycation end-products (AGEs), which alter the mechanical properties of the vascular wall and impair its permeability. The resulting microangiopathy reduces the effective surface area available for gas exchange and contributes to the decline in diffusing capacity often observed in these patients [9,10].

Endothelial dysfunction in the diabetic lung is further characterized by an imbalance in the production of vasodilators and vasoconstrictors. Specifically, there is a marked reduction in the bioavailability of nitric oxide (NO) and prostacyclin (PGI2), coupled with an overproduction of endothelin-1 (ET-1) and thromboxane A2 (TxA2). This shift promotes a pro-thrombotic and pro-inflammatory environment within the pulmonary vasculature. Additionally, the expression of thrombospondin-1 (THBS1) is often altered in diabetic states, further influencing vascular remodelling and potentially contributing to pulmonary hypertension. These vascular changes are not limited to establish T2DM but can be detected as early as the prediabetic stage, suggesting that even moderate elevations in blood glucose can initiate the cascade of endothelial damage. The cumulative effect of these microvascular alterations is a reduction in pulmonary capillary blood volume and an increase in vascular resistance, which may eventually manifest as clinical respiratory impairment [10].

Molecular Pathways of DNA Damage, Cellular Senescence, and Fibrotic Signaling

At the cellular level, the progression of lung injury in T2DM is closely linked to the accumulation of DNA damage and the induction of cellular senescence. Research using diabetic mouse models (db/db) has demonstrated increased nuclear signals of γH2AX, a marker of double-strand DNA breaks, in lung tissue compared to healthy controls. This DNA damage is often a consequence of chronic oxidative stress and the activation of poly (ADP-ribose) polymerase (PARP) pathways. When DNA repair mechanisms are overwhelmed, cells enter a state of senescence, characterized by a permanent cell-cycle arrest and the acquisition of a senescence-associated secretory phenotype (SASP). The SASP involves the secretion of pro-inflammatory cytokines, such as IL-6, and profibrotic factors like TGF-β1, which drive the remodelling of the lung parenchyma and the deposition of extracellular matrix [3].

The transition from cellular senescence to organ-scale fibrosis is a critical step in the development of restrictive lung disease in diabetic patients. Clinical studies have shown that elevated markers of DNA damage and senescence in the blood and urine are predictive of the progression of restrictive lung patterns over time. Specifically, plasma levels of TGF-β1 and IL-6 correlate with the severity of lung function decline, suggesting that systemic inflammation and fibrotic signalling are key drivers of the diabetic lung phenotype. This cascade of DNA damage, senescence, and SASP creates a self-perpetuating cycle of tissue injury and repair failure. Unlike some other diabetic complications, the progression of these markers is not always strictly tied to HbA1c levels, indicating that metabolic memory or other non-glycaemic factors may play a significant role in sustaining the fibrotic process once it has been initiated [3].

Genetic Determinants and Protein Expression Alterations in Bronchial Epithelium

Recent advancements in genomic sequencing have shed light on the genetic underpinnings of pulmonary complications in T2DM, particularly in cases where interstitial lung disease (ILD) is present. A significant finding in this area is the identification of the MUC5B promoter polymorphism (rs2943512) as a potential risk factor for ILD in diabetic patients [1]. MUC5B is a major gel-forming mucin in the airways, and its dysregulation is known to impair mucociliary clearance and promote alveolar injury. In diabetic pedigrees, the presence of this specific SNP has been associated with an increased susceptibility to ILD. Experimental evidence suggests that high glucose environments significantly up-regulate MUC5B expression in bronchial epithelial cells through the activation of the ERK1/2 signalling pathway, which in turn stimulates the production of pro-inflammatory cytokines like IL-1β and IL-6. This suggests a double hit model where genetic predisposition and metabolic stress converge to promote lung injury [1].

Beyond mucin expression, the bronchial and alveolar epithelium in diabetic patients shows significant alterations in the expression of proteins involved in viral entry and immune response. A notable example is the Angiotensin-Converting Enzyme 2 (ACE2), which serves as the primary receptor for SARS-CoV-2. Studies have shown that while ACE2 mRNA levels may remain stable, ACE2 protein expression is significantly increased in the lung tissue of patients with T2DM. This upregulation occurs in both the alveolar and bronchial epithelium and appears to be independent of other factors such as smoking, BMI, or the use of renin-angiotensin-aldosterone system (RAAS) inhibitors. Such protein-level alterations not only increase the risk of severe viral infections but also reflect a broader state of epithelial remodelling and dysfunction in the diabetic lung. The combination of genetic variants like MUC5B and the altered expression of receptors like ACE2 underscores the complexity of the pulmonary phenotype in T2DM, where the epithelium becomes both a driver and a target of disease [1,2].

Clinical Spectrum of Diabetic Lung Complications

Restrictive Lung Disease and Impairment of Pulmonary Function Parameters

The most common clinical manifestation of pulmonary involvement in type 2 diabetes is a restrictive pattern of lung function impairment. Restrictive lung disease (RLD) in this population is characterized by a reduction in total lung capacity (TLC), forced vital capacity (FVC), and forced expiratory volume in one second (FEV1), while the FEV1/FVC ratio typically remains normal or elevated. Studies have demonstrated that the prevalence of RLD increases significantly with the duration of diabetes and the severity of metabolic derangement. For instance, RLD has been found in approximately 20% of patients with newly diagnosed T2DM and up to 27% of those with long-term disease, compared to much lower rates in non-diabetic individuals. This decline in lung volumes is often accompanied by a reduction in the diffusing capacity of the lungs for carbon monoxide (DLCO), reflecting both structural changes in the alveolar-capillary membrane and microvascular dysfunction [6,9].

The clinical impact of RLD in diabetic patients is primarily manifested as breathlessness or dyspnea on exertion. This symptom is frequently reported by patients with T2DM and is significantly associated with the presence of restrictive patterns on spirometry. Interestingly, the risk of developing RLD is also closely linked to other microvascular complications, particularly diabetic nephropathy. Patients with albuminuria or established nephropathy have a substantially higher risk of RLD, with odds ratios as high as 8.57 compared to non-diabetics. This association suggests that the lung and the kidney may share common pathogenic pathways, such as systemic microangiopathy and basement membrane thickening. Despite its prevalence, RLD in diabetes often goes undiagnosed because its symptoms can be attributed to obesity or cardiovascular deconditioning. However, the consistent finding of reduced lung volumes across various diabetic cohorts emphasizes the need for routine pulmonary function testing in patients presenting with unexplained respiratory symptoms [6,11].

Interstitial Lung Disease and the Progression of Pulmonary Fibrosis

While RLD represents a functional deficit, interstitial lung disease (ILD) and pulmonary fibrosis represent the structural progression of diabetic lung injury. T2DM is increasingly recognized as a significant comorbidity and potential risk factor for the development of fibrosing ILD. Radiographic evidence from multidetector computed tomography (MDCT) in diabetic patients with breathlessness often reveals interstitial changes, including reticular opacities and ground-glass appearances, which correlate with functional impairment in tests like the Six-Minute Walk Test (6MWT). Histological examinations of lung tissue from diabetic subjects have further confirmed the presence of alveolar wall thickening and increased collagen deposition, characteristic of a fibrotic process. This suggests that the diabetic lung is not merely a functional disorder but involves a progressive remodelling of the lung parenchyma [1,6,10].

The relationship between T2DM and ILD is complex and can be influenced by other systemic conditions. For example, in patients with systemic lupus erythematosus (SLE), the presence of T2DM has paradoxically been associated with a lower prevalence of ILD and pulmonary fibrosis in some large-scale database analyses. This finding highlights the potential for unexpected interactions between metabolic and autoimmune pathways, although the underlying mechanisms for this specific protective association remain unclear and require further investigation. Conversely, in the general diabetic population, the risk of fibrosis is generally elevated, driven by oxidative stress, chronic inflammation, and dysregulated fibrotic signalling. The progression of ILD in T2DM is a serious concern, as it can lead to irreversible loss of lung function and increased mortality. Early identification of patients at risk for fibrotic progression, perhaps through the use of genetic markers or specific biomarkers, is essential for timely intervention [1,3,10].

Influence of Glycemic Control and Comorbidities on Respiratory Outcomes

The severity and progression of pulmonary complications in T2DM are heavily influenced by the degree of glycemic control and the presence of various comorbidities. While some studies suggest that markers of DNA damage and lung function decline are not always directly predicted by HbA1c levels, there is a clear association between chronic hyperglycemia and the risk of restrictive lung disease. Elevated blood glucose levels promote the formation of AGEs and trigger inflammatory cascades that damage the pulmonary architecture. Furthermore, the transition from prediabetes to established T2DM is marked by a progressive decline in respiratory parameters, indicating that even early metabolic disturbances contribute to lung impairment. Effective management of blood glucose is therefore a cornerstone of preventing or slowing the progression of diabetic lung disease.

Comorbidities such as obesity and cardiovascular disease further exacerbate the respiratory burden in diabetic patients. Obesity, in particular, imposes mechanical constraints on the chest wall and is associated with systemic inflammation, both of which can worsen restrictive lung patterns. In obese patients with T2DM, the presence of metabolic syndrome components is strongly correlated with impaired FEV1 and FVC. Additionally, the presence of diabetic nephropathy serves as a powerful predictor of pulmonary dysfunction, suggesting that the diabetic lung should be viewed as part of a multi-organ syndrome of microvascular and fibrotic damage. The interplay between these factors necessitates a holistic approach to the management of T2DM, where respiratory health is monitored alongside renal and cardiovascular function. Addressing modifiable risk factors and optimizing metabolic control may help mitigate the long-term impact of T2DM on the respiratory system.

Interaction Between Type 2 Diabetes and Infectious or Inflammatory Airway Diseases

Impact of Diabetes on Coronavirus Disease 2019 Severity and Post-Infection Fibrosis

The COVID-19 pandemic has brought the intersection of T2DM and respiratory health into sharp focus. Patients with T2DM are not only more susceptible to infection with SARS-CoV-2 but also experience significantly higher rates of severe disease, hospitalization, and mortality. One of the key biological drivers of this increased vulnerability is the upregulated expression of the ACE2 receptor in the lung tissue of diabetic patients. As the primary entry point for the virus, higher levels of ACE2 protein in the alveolar and bronchial epithelium may facilitate a higher viral load and more extensive tissue damage. This upregulation appears to be a direct consequence of the diabetic state itself, independent of other common comorbidities or medications, making the diabetic lung a primed environment for severe viral pneumonia.

Beyond the acute phase of infection, T2DM is a major risk factor for the development of post-COVID-19 lung fibrosis. Studies have shown that approximately 19.1% of diabetic patients who survive COVID-19 develop some degree of pulmonary fibrosis. This high rate of fibrotic complications is likely due to the pre-existing pro-fibrotic environment in the diabetic lung, characterized by chronic inflammation and impaired repair mechanisms. However, there is a significant protective effect associated with COVID-19 vaccination in this population. Vaccinated patients with T2DM show a markedly lower risk of both mortality and post-infection lung fibrosis, with odds ratios as low as 0.3 for the latter. This underscores the critical importance of preventative measures, including boosters, in managing the respiratory risks associated with T2DM during and after the pandemic. The long-term monitoring of lung function in diabetic COVID-19 survivors is essential to manage the potential burden of chronic respiratory impairment.

Management of Chronic Obstructive Pulmonary Disease in Diabetic Populations

Chronic obstructive pulmonary disease (COPD) and T2DM frequently coexist, creating a challenging clinical scenario where each condition can exacerbate the other. Patients with both diseases are at a higher risk for acute exacerbations, cardiovascular events, and premature death compared to those with COPD alone. Recent research has focused on how various glucose-lowering therapies might influence respiratory outcomes in this specific population. For instance, the use of sulfonylureas (SU) in patients with T2DM and COPD has been associated with significantly lower risks of all-cause mortality, cardiovascular events, and severe respiratory complications such as the need for invasive mechanical ventilation or the development of bacterial pneumonia. These benefits appear to be duration-dependent, with longer use of sulfonylureas providing greater protection, suggesting that stable glycemic control and perhaps specific anti-inflammatory effects of these drugs are beneficial for COPD patients.

Even more promising are the findings regarding sodium-glucose cotransporter-2 (SGLT-2) inhibitors. In a large population-based cohort study, the use of SGLT-2 inhibitors in patients with T2DM and COPD was linked to a significantly lower risk of a composite primary outcome including hospitalization for COPD, mortality, and the need for ventilator support. Specifically, SGLT-2 inhibitor users had a 36% lower risk of mortality and a 52% lower risk of requiring non-invasive positive-pressure ventilation compared to non-users. These benefits were consistent across various subgroups and showed a dose-response relationship. The potential mechanisms for these protective effects may include the reduction of systemic inflammation, improvement in cardiovascular function, and the promotion of weight loss, all of which are beneficial in the context of COPD. These findings suggest that the choice of antidiabetic medication can have a profound impact on the respiratory prognosis of patients with T2DM and COPD, potentially positioning SGLT-2 inhibitors as a preferred therapy in this group.

Diagnostic Biomarkers for Identifying Pulmonary Involvement in Metabolic Disorders

Given the often subtle and progressive nature of lung involvement in T2DM, there is a pressing need for reliable diagnostic biomarkers that can identify patients at risk for respiratory complications before significant irreversible damage occurs. One such promising biomarker is Serum Surfactant Protein D (SP-D). SP-D is a collectin primarily synthesized by alveolar type II cells and Clara cells, and it plays a crucial role in pulmonary host defense and the regulation of inflammation. In obese patients with T2DM, serum concentrations of SP-D have been found to be significantly higher than in non-diabetic controls. Furthermore, elevated SP-D levels are inversely associated with FEV1 and positively correlated with residual volume, making it a potentially useful marker for detecting early lung impairment. A specific cutoff of 132.3 ng/mL has been proposed to identify patients with a FEV1 <80% of the predicted value, offering a tool for clinicians to screen for those who require more formal pulmonary function testing.

In addition to SP-D, markers of DNA damage and cellular senescence are emerging as valuable tools for monitoring the progression of the diabetic lung. As previously discussed, markers such as γH2AX in white blood cells and urinary p21 excretion are associated with the presence and progression of restrictive lung disease in diabetic cohorts. These biomarkers reflect the underlying molecular pathology of tissue injury and could potentially be used to track the efficacy of therapeutic interventions. Furthermore, the identification of genetic markers, such as the MUC5B rs2943512 polymorphism, provides a way to identify individuals with a high genetic susceptibility to interstitial complications. The integration of these biochemical and genetic markers into clinical practice could facilitate a more personalized approach to the management of T2DM, allowing for early detection and targeted treatment of pulmonary complications. Future research should focus on validating these biomarkers in larger, more diverse populations and exploring their utility in clinical decision-making.

Conclusion and Future Work

The evidence presented in this review underscores the fact that the lung is a significant, albeit often overlooked, target organ for the complications of type 2 diabetes mellitus. From the molecular level—where DNA damage, cellular senescence, and genetic predispositions like the MUC5B polymorphism drive tissue remodelling—to the clinical level, where restrictive lung disease and interstitial changes manifest as breathlessness and reduced functional capacity, the diabetic lung represents a complex and multifaceted clinical entity. The pathophysiology is rooted in a combination of microangiopathy, epithelial dysfunction, and chronic inflammation, all of which are exacerbated by the metabolic stresses of hyperglycaemia and insulin resistance. Furthermore, the heightened expression of the ACE2 receptor in the diabetic lung provides a clear biological explanation for the increased severity of COVID-19 in this population, while the high rate of post-infection fibrosis highlights the long-term respiratory risks faced by diabetic survivors.

Despite these insights, several gaps in our knowledge remain, providing fertile ground for future research. First, while the association between T2DM and restrictive lung patterns is well-established, the long-term natural history of these changes is still not fully understood. Prospective, longitudinal studies are needed to determine the rate of progression from subtle functional impairment to clinically significant interstitial lung disease and to identify the factors that accelerate this transition. Second, the paradoxical findings in certain populations, such as the lower prevalence of ILD in diabetic patients with SLE, suggest that there are complex interactions between metabolic and immune pathways that we have yet to unravel [4]. Investigating these protective mechanisms could yield new therapeutic insights for preventing fibrosis in other contexts.

From a therapeutic perspective, the recent findings regarding the benefits of SGLT-2 inhibitors and sulfonylureas in patients with comorbid COPD are highly encouraging. Future work should include randomized controlled trials specifically designed to evaluate the respiratory outcomes of these and other modern glucose-lowering agents, such as GLP-1 receptor agonists, which also possess potent anti-inflammatory properties. Moreover, the potential for senolytic therapies—drugs that selectively eliminate senescent cells—to treat or prevent diabetic lung fibrosis is an exciting area of investigation that warrants further exploration in both preclinical and clinical settings.

Finally, the clinical management of T2DM must evolve to include routine assessment of respiratory health. The identification of biomarkers like Serum Surfactant Protein D (SP-D) offers a practical starting point for screening at-risk patients in primary care and endocrine clinics. Integrating simple screening tools, such as questionnaires for breathlessness and portable spirometry, into standard diabetic care could lead to earlier diagnosis and intervention. As our understanding of the genetic and molecular drivers of the diabetic lung continues to grow, we move closer to a precision medicine approach where treatments can be tailored to an individual's specific risk profile. In conclusion, recognizing and addressing the pulmonary manifestations of T2DM is essential for reducing the overall morbidity and improving the quality of life for the millions of people living with this systemic disorder [11].

Declarations

Ethics Approval and Consent to Participate

Not applicable. This study is a narrative review based on previously published data and does not involve human participants or primary data collection.

Consent for Publication

Not applicable.

Availability of Data and Materials

Not applicable. No new datasets were generated or analyzed in this study.

Competing Interests

The authors declare that they have no competing interests.

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