WebLog Journal of Pulmonology and Respiratory Medicine
Letter to Editor
Published: 15 May, 2026

The Diabetic Lung: Time to Move from Neglect to Recognition

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Respected Editor,

Diabetes mellitus (DM) is universally recognized for its devastating microvascular and macrovascular complications. Yet one organ has been conspicuously absent from both clinical guidelines and everyday practice: the lung. A growing body of evidence compellingly positions the lung as a genuine target organ of chronic hyperglycemia, giving rise to the concept of "diabetic pneumopathy" - a term encompassing restrictive ventilatory impairment, reduced diffusing capacity for carbon monoxide (DLCO), accelerated lung function decline, and, in its most severe form, diabetes-induced pulmonary fibrosis (DiPF) [1]. Despite this accumulating evidence, diabetic pneumopathy remains unrecognized in major clinical guidelines, underdiagnosed in routine practice, and essentially absent from diabetes management protocols. This gap deserves urgent and deliberate attention.

The mechanistic case for the lung as a diabetic target organ is robust. Chronic hyperglycemia drives pulmonary microangiopathy with basement membrane thickening analogous to that seen in glomerular and retinal capillaries [2]. Simultaneously, the accumulation of advanced glycation end products (AGEs) activates RAGE signaling on abundant type I alveolar epithelial cells, unleashing oxidative stress, NF-κB activation, and profibrotic cytokine release [1, 3]. Critically, sustained hyperglycemia impairs DNA repair through a reduced NAD^+/NADH ratio and defective non-homologous end joining, driving alveolar type II cell senescence. These senescent cells adopt a pro-inflammatory senescence-associated secretory phenotype (SASP) - releasing IL-6, TGF-β, and TNF-α - perpetuating fibroblast activation and extracellular matrix accumulation [4]. Experimental proof of causality, not merely association, has been demonstrated: restoring DNA repair pathways reverses pulmonary fibrosis in diabetic animal models [4]. This mechanistic cascade mirrors and parallels the biology of diabetic nephropathy and retinopathy, providing a strong scientific rationale for formal recognition of DiPF as a true diabetic complication.

Clinically, the evidence is equally persuasive. Meta-analyses consistently demonstrate significant reductions in forced vital capacity (FVC), FEV_1, and DLCO in diabetic patients compared with controls - with a uniformly preserved FEV_1/FVC ratio confirming a restrictive, not obstructive, pattern [5]. Longitudinal cohorts including the Fremantle Diabetes Study and the ARIC (Atherosclerosis Risk in Communities) Study document an accelerated annual FVC and FEV_1 decline two to three times faster than in healthy non-smokers, graded by glycemic burden and independently predictive of all-cause mortality [6, 7]. Crucially, impairment is demonstrable even in prediabetes and in children with type 1 DM, establishing that pulmonary dysfunction is an early and progressive feature rather than a late sequela. The odds of restrictive lung disease are approximately four times higher in patients with DM versus controls, and the strongest predictors of pulmonary impairment are the same as for classical microvascular complications: duration of disease, HbA1c, microalbuminuria, retinopathy, and nephropathy.

Diabetic pneumopathy does not exist in isolation. It significantly overlaps with idiopathic pulmonary fibrosis (IPF) - with a diabetes prevalence of nearly 33% in IPF cohorts and a 54% higher incidence of pulmonary fibrosis in diabetic patients [8] - as well as with COPD, asthma, pulmonary hypertension, and heightened susceptibility to infections including tuberculosis. Compounding this, treatments for these comorbidities - particularly corticosteroids - themselves precipitate or worsen glycemic control, creating a clinically challenging bidirectional relationship that is inadequately addressed by current guidelines. Emerging therapeutic directions including SGLT-2 inhibitors, GLP-1 receptor agonists, NAD^+ precursors, senolytic agents, and antifibrotics such as nintedanib and pirfenidone offer promising avenues, but dedicated randomized trials in DiPF are urgently needed before clinical translation [1].

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Received Date: *12 May 2026*
Accepted Date: *13 May 2026*
Published Date: *15 May 2026*

Citation:
Rahul Garg. The Diabetic Lung: Time to Move from Neglect to Recognition. WebLog J Pulmonol Respir Res. wjprm.2026.e1503. https://doi.org/10.5281/zenodo.20230397

The practical implications are clear. Every diabetic patient with progressive exertional dyspnea, a reduced 6-minute walk distance, or unexplained restrictive spirometry should be evaluated for diabetic pneumopathy through comprehensive pulmonary function testing including DLCO, body plethysmography, and HRCT chest when indicated. Conversely, every patient with apparently "idiopathic" pulmonary fibrosis who has longstanding diabetes and albuminuria should prompt consideration of DiPF rather than a purely idiopathic process. Incorporating routine pulmonary screening into diabetes management guidelines - particularly in patients with established microvascular complications - would represent a straightforward and high-yield intervention that currently receives no mention in major guidelines from the American Diabetes Association, the European Association for the Study of Diabetes, or the Global Initiative for Fibrosis.

With 589 million adults living with diabetes worldwide today and projections of 853 million by 2050, even a modest prevalence of diabetic pneumopathy translates to an enormous unrecognized disease burden [9]. The lung has waited long enough. It is time for the clinical and research communities to formally recognize diabetic pneumopathy as a distinct complication of DM, invest in prospective trials, and integrate pulmonary assessment into the standard of diabetes care.

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