

Article

Inflammatory and Oxidative Stress Biomarkers in Bronchiectasis: Current Evidence and Future Perspectives—A Review

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Abstract: It is a heterogeneous chronic airway disease with an inflammatory cycle of persistent infection with impaired mucociliary clearance, dysregulated host immunity and structural airway damage. Biomarkers can offer a biological dimension of information that enhances the clinical, microbiology, pulmonary function and radiology information. The aim of this review is to emphasize the current evidence on inflammatory and oxidative stress biomarkers in bronchiectasis, including their biological compartments, association with clinical parameters, potential limitations and uses in precision medicine. The most well-established biological process is the neutrophil dominated inflammation, which is represented by the presence of neutrophil extracellular traps, sputum myeloperoxidase, myeloperoxidase, matrix metalloproteinases and inflammatory cytokines in the sputum [1–4,8–12]. Systemic markers such as C-Reactive protein and fibrinogen could be useful to obtain a readily available measure of disease activity and risk of exacerbation but are not specific [13–16]. Oxidative stress can also be measured, by increases in lipid peroxidation and ROS production and decreases in antioxidant defenses [21,22]. Notably, type 2 inflammatory and eosinophilic endotypes have been identified in a clinically meaningful proportion and neutrophilic markers are not able to represent bronchiectasis [28,29]. New proteomic, microbiome, transcriptomic, and composite biomarker approaches might prove to be more effective for endotyping and treatment selection [30–35]. Further studies are warranted including: longitudinal validation, standard assays, clinically relevant thresholds and combining biomarkers with treatable traits (rather than using laboratory assays alone).

Keywords: bronchiectasis; biomarkers; neutrophilic inflammation; oxidative stress; neutrophil elastase

Citation: Allawi, M. S. Inflammatory and Oxidative Stress Biomarkers in Bronchiectasis: Current Evidence and Future Perspectives—A Review . Central Asian Journal of Medical and Natural Science 2026, 7(4), 567-578

Received: 13th Jun 2026

Revised: 28th Jul 2026

Accepted: 15th Aug 2026

Published: 29th Sep 2026



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1. Introduction

Bronchiectasis is defined as a persistent dilatation of the bronchi which is accompanied by chronic cough, sputum formation, recurrent respiratory infections, and differing amounts of lung function compromise. Current models of the disease have the disease in a “vicious vortex” where impaired mucous clearance allows for the persistence of microorganisms that lead to infection, which in turn leads to inflammation, which leads to progressive airway injury, which leads to more impaired mucous clearance [1], [2], [3], [4]], [5], [6], [7] (Figure 1). These results support this concept, which implies that there is significant variation in clinical symptoms, exacerbation rate, microbiological profile, and functional deterioration rate among patients with CT appearances similar to the one studied. Disease severity is therefore multidimensional, and disease severity tools such as the Bronchiectasis Severity Index (BSI) and FACED score do not only include biological measurements, but also other clinical, functional, microbiological and radiological parameters [1], [2].

This is because conventional clinical parameters are not sufficient to represent disease activity and inflammatory endotype, resulting in the use of biomarkers. The most convincing evidence is that neutrophilic airway inflammation is linked to the level of the enzyme, neutrophil elastase (NE), which has been linked to exacerbation and lung-function decline, and disease severity [8], [9], [10]. However, proteins relevant to the acute phase, cytokines, matrix-degrading enzymes, oxidative stress products, eosinophilic markers and emerging omics-derived signatures might be addressing different facets of inflammation, which is biologically heterogeneous [11], [12], [13], [14], [15], [16]. The objective of this review is to summarize the current knowledge on inflammatory/oxidative stress markers in bronchiectasis and propose areas where these markers can move from descriptive towards clinical application as precision markers.

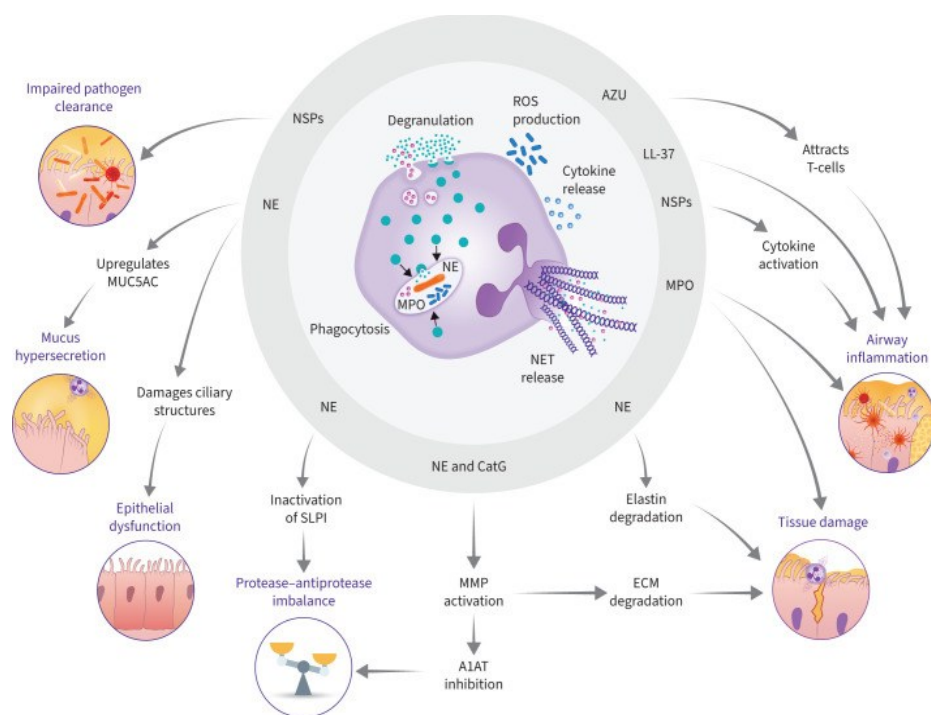


Figure 1. The bronchiectasis “vicious vortex” linking impaired mucociliary clearance, persistent infection, neutrophilic inflammation, oxidative/proteolytic injury, and structural airway damage.

2. Materials and Methods

This study employed a narrative review approach to synthesize the current evidence regarding inflammatory and oxidative stress biomarkers in bronchiectasis. Relevant peer-reviewed literature was evaluated with particular emphasis on studies addressing the biological mechanisms of neutrophilic inflammation, protease–antiprotease imbalance, oxidative stress, airway and systemic inflammatory biomarkers, eosinophilic and mixed inflammatory endotypes, airway microbiology, and emerging multi-omics approaches. The reviewed evidence included observational studies, prospective cohorts, randomized clinical trials, registry-based studies, clinical reviews, and molecular investigations relevant to bronchiectasis. Biomarkers of particular interest included sputum neutrophil elastase, neutrophil extracellular traps, myeloperoxidase, matrix metalloproteinases, inflammatory cytokines, C-reactive protein, fibrinogen, eosinophil-related markers, and oxidative stress indicators such as malondialdehyde, lipid-peroxidation products, reactive oxygen species, glutathione, superoxide dismutase, catalase, and total antioxidant capacity. The evidence was synthesized thematically according to biomarker compartment, underlying biological pathway, association with disease severity and exacerbation, treatment responsiveness, and potential clinical applicability. Particular

attention was also given to methodological limitations, including pre-analytical variability, differences in assay platforms, lack of standardized thresholds, and heterogeneity among patient populations. Because this review was based exclusively on previously published studies and did not involve direct recruitment of human participants or collection of identifiable patient information, ethical approval and informed consent were not required.

3. Results

3.1 Biological Basis of Inflammation and Oxidative Stress

3.1.1 The neutrophil-centered inflammatory response

Clinically significant bronchiectasis is characterized by a predominant neutrophilic inflammation. Microbial stimulation, epithelial signaling and chemokine release leads to the recruitment and activation of neutrophils in the airways. IL-8/CXCL8 is a known neutrophil chemoattractant in bronchiectatic sputum and other mediators such as IL-1 β , IL-6 and TNF- α enhance inflammatory signaling both locally and systemically (Figure 2) [17], [18], [19], [20]. Activated neutrophils produce serine proteases such as NE, cathepsin G and proteinase 3, in addition to the myeloperoxidase and reactive oxygen species. If the activity of protease is greater than that of the antiprotease, then the property of the epithelial structures, extracellular matrix and mucus can be changed [8], [9], [10], [21], [22], [23], [24].

NE is especially relevant in terms of its association with clinical outcomes and inflammation. In prospective cohorts, increased sputum NE activity has been linked to increased bronchiectasis severity scores, exacerbation frequency, radiological extent and accelerated FEV1 loss rates [8]. NE also increases during exacerbations and can decrease after antibiotic therapy, suggesting that it is a dynamic parameter, and not simply a static disease feature [8], [9]. Another way that activated neutrophils play a role in mucous plugging and chronic inflammation is via NET formation. A higher NET burden has been correlated with increased disease severity, and may affect antibiotic responsiveness [11]. The results indicate that the numbers of neutrophils is not enough; the functional state and products of neutrophils may be more informative.

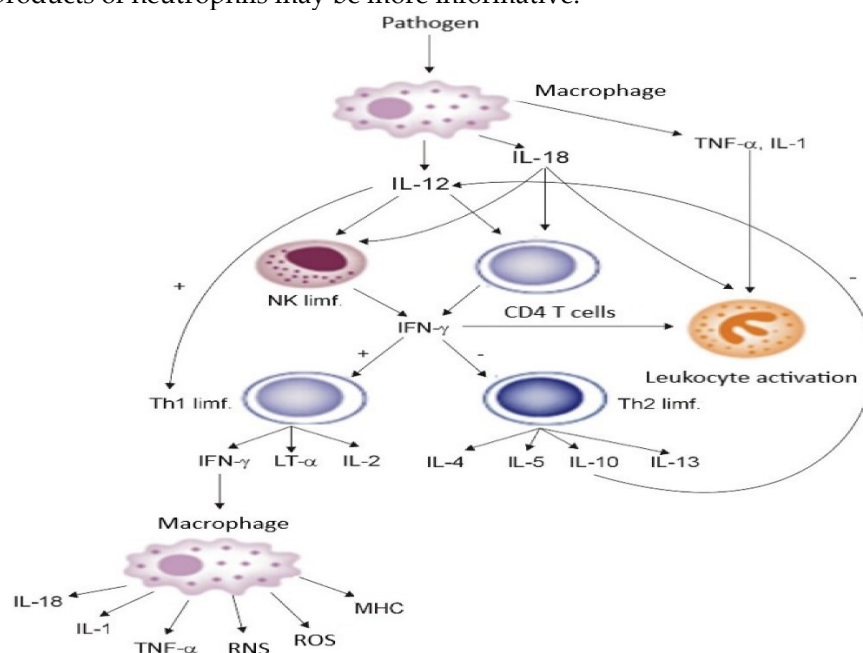


Figure 2. Neutrophil and Systemic inflammatory response

3.1.2 Protease–antiprotease imbalance and airway remodeling

A protease activity is a link between inflammation and permanent damage. NE and other neutrophil serine proteases cleave the ECM proteins and affect the function of

epithelial cells, host defense mechanisms and can increase mucus viscosity. Furthermore, the presence of increased levels of the matrix metalloproteinases (MMP-8 and MMP-9) in the sputum of bronchiectasis patients relates to the radiological severity, lung-function deficits and exacerbation risk of bronchiectasis [23]. Such enzymes are part of a complex protease–antiprotease system that includes endogenous antiproteases, including secretory leukocyte protease inhibitor (SLPI) (Figure 3). The biological consequence is not only a function of the concentration but rather depends on the ratio of proteases to protective mechanisms [25], [26], [27], [28].

This pathway has been confirmed clinically by therapeutic studies. Biological pathways (defined by biomarkers) are important and can be a therapeutic target: In randomized trials, the DPP-1 inhibitor brensocatic led to a reduction in exacerbations and sputum NE activity [29], [30]. The relationship of suppression of the biomarkers with long-term structural preservation is not completely clear, however. Future research is therefore required to evaluate the ability of serial measurements of proteases to predict patients who are most likely to progress and whether changes in protease activity are associated with clinical improvement.

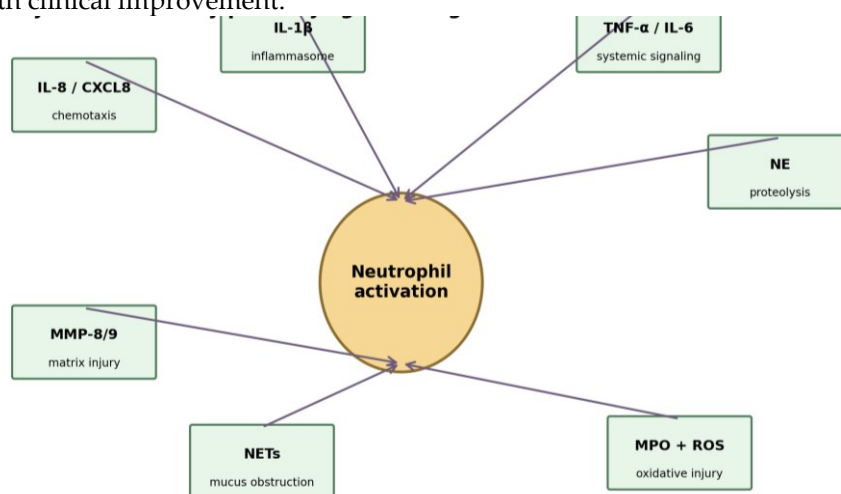


Figure 3. Major inflammatory pathways generating candidate biomarkers in bronchiectasis.

3.1.3. Inflammatory Biomarkers: Current Evidence

3.1.3.1. Airway biomarkers

The concept of airway biomarkers is thus substantially more advantageous because they are measured near the site of disease. The most well-documented candidate and associated with exacerbation, severity, lung-function decline, and microbiological features is sputum NE [8–10]. These are complementary to each other as they give information on extracellular matrix remodeling (ECMR) (MMP-8 and MMP-9) and neutrophil activation and oxidant generation (MPO). Cytokines present in sputum, including IL-1 β , IL-6, IL-8 and TNF- α , can be used to provide information about inflammatory signaling but may be influenced by sampling technique, dilution, the presence of infection and by the method of assay [17], [18], [19], [20].

The neutrophil-derived proteins such as calprotectin are attractive due to their potential for being more stable than some soluble cytokines, however there is a lack of sufficient standardization for clinical thresholds. Recent studies of airway IL-1 β indicate that it is linked to the severity of the disease, characteristics of mucus, and mucociliary function, which is consistent with the idea that biomarkers reflect certain biological processes and not just “inflammation” [19]. The biggest drawback of sputum biomarkers is their practicality, the fact that sputum may not be spontaneously or inducibly obtained in certain patients or that the processing conditions can significantly affect the outcome of the test. Hence, the biomarkers in the airways are of special interest for research

stratification and therapeutic trials, whereas blood-based biomarkers can be used more for routine longitudinal monitoring.

3.1.3.2. Systemic inflammatory biomarkers

Blood biomarkers are desirable because they are easily incorporated into the standard health care system, they are easily replicated, and they are inexpensive. In registry studies, higher levels in baseline C-reactive protein (CRP) have been linked to subsequent severe exacerbations [14], [15] and are a sign of systemic inflammatory activity. Fibrinogen has also been found to have consistent associations with severity of disease, *Pseudomonas aeruginosa* colonisation, and poor health status and risk of exacerbation in the future [13], [16]. However, other circulating cytokines, such as tumour necrosis factor (TNF- α) can be used to differentiate inflammatory phenotypes, but their clinical value is limited because of biological variation and lack of validated decision thresholds [20].

The results of this study suggest systemic biomarkers can be useful, but in a non-specific manner. A raised CRP or fibrinogen level may be a result of other inflammatory factors (not necessarily the activity of bronchiectasis) – these include smoking, obesity, auto-immune disease, and/or activity of another comorbidity. Recent developments have been the use of composite measures (e.g. CRP/albumin-related measures and fibrinogen/FeNO) that combine complementary signals that can provide greater biological resolution than a single measure [15], [16]. However, composite biomarkers need to be externally validated, collected under standard circumstances and have demonstrated to modify clinical decisions, and not simply correlate with severity scores.

3.1.4. Oxidative Stress Biomarkers and Redox Biology

Oxidative stress is emerging as an important element of the biology of bronchiectasis (Figure 4). Respiratory burst (and myeloperoxidase dependent) pathways by activated neutrophils and the overwhelming of epithelial and systemic antioxidant systems during inflammatory and infectious processes contribute to the production of reactive oxygen species. In bronchiectasis adults, a decrease in total antioxidant capacity and superoxide dismutase activity and increase in superoxide and hydrogen peroxide has been shown when compared with healthy adult controls [21]. These findings corroborate a condition of redox imbalance and not just of a single oxidative product.

Oxidative stress biomarkers which are potential candidates are malondialdehyde, lipid-peroxidation and its products, 8-isoprostanes, intracellular reactive oxygen species, glutathione status, superoxide dismutase, catalase and total antioxidant capacity [21], [22]. Exhaled breath condensate (EBC) can be used as a non-invasive method to quantify oxidative products but pre analytical issues and low concentration of analytes make the practical use of EBC in clinical routine more challenging [22]. Glutathione is especially important as it is involved in epithelia antioxidant defense and redox signaling. Oxidative injuring can further increase the harm caused by proteases on epithelial function, affecting the cilia function and integrity of the epithelial barrier, thus establishing a positive feedback loop between infection and inflammation.

Although possible, oxidative stress biomarkers are less clinically advanced than NE. The results obtained from different analytical platforms, different handling of samples, and different definitions of oxidative stress make it difficult to compare the results from different studies. Perhaps the best approach that can be used in the future is to use a redox panel consisting of oxidant products and antioxidant capacity instead of just one oxidative marker.

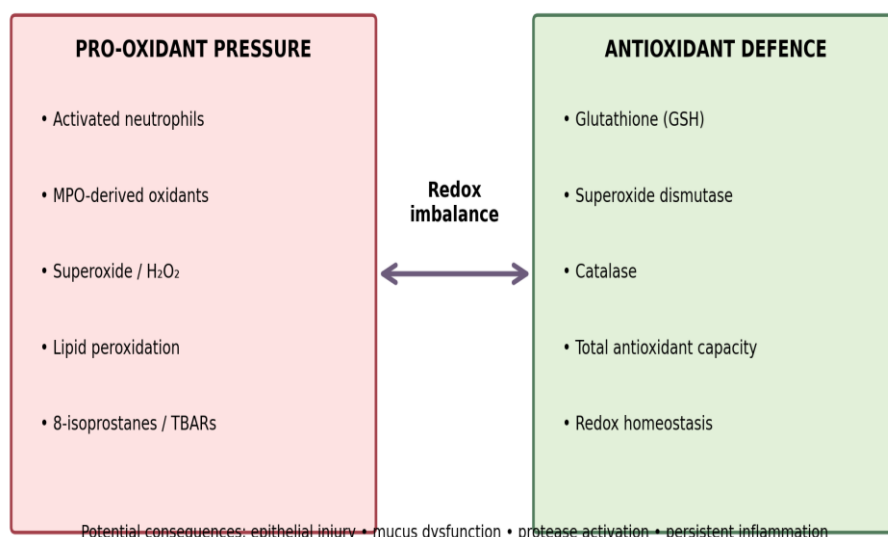


Figure 4. Oxidative stress–antioxidant imbalance in bronchiectasis and its potential consequences for airway injury.

3.1.5. Biomarker Heterogeneity, Endotypes, and Clinical Interpretation

3.1.5.1. Neutrophilic and eosinophilic endotypes

While the most common type is neutrophilic inflammation, there is no single immunopathology of bronchiectasis. Patients with allergic bronchopulmonary aspergillosis (ABPA) and/or other associated diseases, such as asthma, typically have blood or sputum eosinophilia, features of type 2 inflammation, and are a clinically useful subpopulation (Figure 5) [28], [29]. Blood eosinophils that can be accessed, but are not as reflective. The use of FeNO may give additional information about epithelial type 2 signaling, but is influenced by atopy, corticosteroid use, smoking and infection [28], [29]. This is relevant, as a neutrophil-only biomarker panel could underestimate the patients with eosinophilic or mixed inflammation.

Recent research has suggested that adaptive immune pathways are also involved in inflammatory heterogeneity. The balance of T-helper cells/airway microbiome-host immunity, IL-17 signaling [12], [29] may be involved in persistent inflammation. Therefore, biomarkers are not necessarily indicators of disease severity but rather are indicators of endotype. A marker must be “high” but it must also be a marker of a biological pathway and it must be stable over time and be able to predict the response to a treatment.

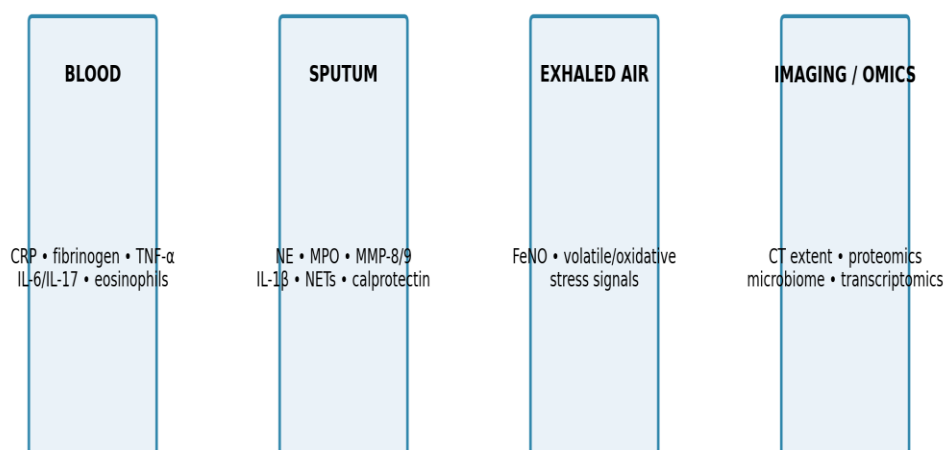


Figure 5. Biomarker compartments and the biological information they may provide in bronchiectasis

3.1.5.2. Microbiology and inflammation as interacting biomarkers

The most clinically important other bacteria associated with bronchiectasis are *P. aeruginosa*, which has been associated with increased disease burden, exacerbations, and increased hospitalization and mortality [25], [26]. Importantly, infection and inflammation are not a completely independent process. Dominance of *P. aeruginosa* is associated with increased neutrophilic activity, a decrease in microbial diversity and an increase in NE activity [10], [27]. This is in line with the concept of host-microbe biomarkers in which microbial composition and inflammatory response are taken into consideration.

More specifically, the concept of 'microbiome' applies, in that only a small proportion of the airway microbiome can be detected by standard culture methods. Studies conducted through sequencing have revealed associations between decreased microbial diversity, pathogens dominance and disrupted microbial networks and inflammatory phenotypes [27][30], [31], [32], [33], [34]. The majority of the available evidence, however, is observational only and correlations are not proof of cause-and-effect. Therefore, the development of biomarkers should focus on an approach of longitudinal sampling to identify microbiome shifts that happen before, after or at the same time that inflammatory activation occurs during exacerbations.

3.1.6. Clinical Utility, Therapeutic Response, and Current Limitations

However, clinical utility is the final measure of the utility of a biomarker not just statistical association (Figure 6). The ideal bronchiectasis biomarker should be analytically reliable, biologically plausible, sensitive to disease change, linked to clinically meaningful outcomes and useful for treatment selection or monitoring [30], [31], [32]. NE is most closely associated to this concept as it has also been linked to disease severity and exacerbation risk, and has been included in clinical trials of targeted neutrophil therapies [8], [35], [36], [37]. In addition to this, markers of oxidative stress are more easily accessed but less specific, while CRP and fibrinogen are mainly research tools [13], [14], [15], [16], [21], [22].

A recent example of therapeutic development that demonstrates the impact of biomarker biology on the clinical approach is the development of Aprepitant, a selective, selective neurokinin 1 receptor antagonist (NK-1). Brensocatib is an inhibitor of the neutrophil-protease pathway, and the WILLOW and ASPEN trials showed that inhibiting activation of neutrophil serine protease reduces exacerbation burden, which is a biological indication of the relevance of the neutrophil-protease pathway [36], [37]. Long-term antibiotic trials also show that the microbiological phenotype has an impact on treatment choices, especially when the infection is chronic with *P. aeruginosa* infection [38], [39], [40]. But response to treatment is seldom governed by one biomarker. History, microbiology, lung function, symptoms, imaging, comorbidities and inflammatory phenotype need to be taken into account.

Pre-analytical variability and a lack of universally agreed standards for thresholds, definition of stable disease and exacerbation, variability in assay platforms and limited representation of diverse populations are all major limitations. Numerous biomarkers are well correlated with the cross-sectional clinical scores, but have limited incremental value above the established clinical scores. Future research studies should thus be designed to determine whether the use of biomarkers to guide treatment decisions is associated with better patient outcomes, fewer exacerbations or identification of patient responders to targeted therapies.

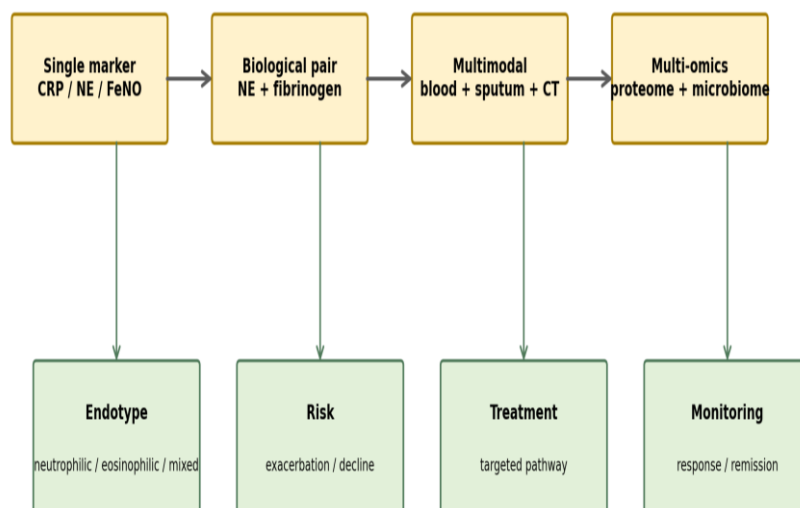


Figure 6. Conceptual transition from single biomarkers to composite and multi-omic precision-medicine signatures.

3.1.7. Future Perspectives

A new era of bronchiectasis biomarker initiatives is likely to progress from individual markers to biological "signatures. Interactions that cannot be represented by a single inflammatory protein can be captured by multi-omics approaches such as the proteomics, microbiome sequencing, transcriptomics and metabolomics [30–35]. Molecular profiling, by proteomic analyses of the bronchoalveolar lavage (BAL) fluid from exacerbations in recent years, has shown coordinated changes in epithelial and epithelial derived pathways related to neutrophil degranulation and in host-defense pathways, supporting the potential of molecular profiling to define exacerbation disease state [34]. The key is creating large panels of molecular information in a small format that can be measured from within the standard clinical laboratory.

It is also necessary to do longitudinal sampling. Biomarkers should be used to identify those that indicate changes that lead to clinical deterioration versus those that only reflect the rise in inflammation when used in stable disease, early exacerbation, treatment, and recovery. International validation is required and validation across ethnicities and aetiologies and with other comorbid conditions. Biomarker trials must be accompanied by standardized sample collection and assay calibration in order to be able to compare thresholds between labs in the future.

A future model would utilize the structural phenotype from the MRI, the clinical history, the microbiology, blood inflammatory markers, the protease activity of the sputum and some of the omics signals. This composite could be used to differentiate neutrophilic and/or eosinophilic and/or mixed endotypes; to provide a risk stratification for exacerbation; and to lead pathway-based treatment. Importantly, health-economic and implementation outcomes should be included in the development of biomarkers; a biomarker that is biologically valid is not very useful if it is not available, is too expensive or does not change clinical practice.

4. Discussion

The evidence reviewed indicates that bronchiectasis is characterized by considerable biological heterogeneity, although neutrophil-dominated airway inflammation remains the most consistently documented inflammatory pattern. Activated neutrophils release neutrophil elastase, myeloperoxidase, reactive oxygen species, and other proteolytic mediators that contribute to epithelial injury, extracellular matrix degradation, mucus dysfunction, and continued recruitment of inflammatory cells. Among these biomarkers, sputum neutrophil elastase appears to have the strongest clinical evidence because

increased activity has been associated with greater disease severity, more frequent exacerbations, more extensive radiological abnormalities, and accelerated decline in lung function. Its dynamic response during exacerbations and after antibiotic therapy further suggests that neutrophil elastase reflects active disease biology rather than merely established structural damage.

The protease–antiprotease imbalance provides an important mechanistic link between persistent inflammation and structural airway damage. Neutrophil elastase and matrix metalloproteinases can degrade extracellular matrix components, alter epithelial function, compromise host defense, and increase mucus viscosity, whereas endogenous antiproteases attempt to counterbalance this activity. Therefore, the biological consequences of proteolysis depend not simply on absolute protease concentrations but on the balance between destructive and protective pathways. The clinical relevance of this mechanism is supported by trials of the DPP-1 inhibitor brensocatib, in which suppression of neutrophil serine protease activation was associated with reductions in exacerbation burden and sputum neutrophil elastase activity. Nevertheless, it remains uncertain whether short-term biomarker suppression translates into long-term preservation of airway structure.

Airway biomarkers provide the advantage of measuring inflammatory processes close to the principal site of disease. In addition to neutrophil elastase, sputum myeloperoxidase, MMP-8, MMP-9, IL-1 β , IL-6, IL-8, TNF- α , and calprotectin may provide complementary information regarding neutrophil activation, tissue remodeling, and inflammatory signaling. However, their clinical implementation is limited by variability in sputum availability, processing procedures, dilution effects, infection status, and assay methodology. In contrast, systemic biomarkers such as CRP and fibrinogen are easier to measure repeatedly in routine practice and have been associated with disease severity and future exacerbation risk. Their major limitation is lack of specificity, because elevated systemic inflammatory markers may also reflect smoking, obesity, autoimmune conditions, infection, or other comorbid diseases.

Oxidative stress represents another important component of bronchiectasis pathobiology. Increased reactive oxygen species production associated with neutrophil respiratory burst and myeloperoxidase activity, together with reduced antioxidant defenses, may amplify epithelial injury and protease-mediated damage. Available evidence demonstrates increased oxidative products together with reductions in antioxidant capacity and superoxide dismutase activity in patients with bronchiectasis. Potential biomarkers include malondialdehyde, 8-isoprostanes, lipid-peroxidation products, intracellular reactive oxygen species, glutathione, catalase, superoxide dismutase, and total antioxidant capacity. However, oxidative stress biomarkers remain less clinically established than neutrophil elastase because differences in analytical platforms, sample handling, and definitions of oxidative stress limit cross-study comparability. This suggests that future assessment may be more informative when based on combined redox panels rather than a single oxidative marker.

Importantly, bronchiectasis cannot be represented exclusively by a neutrophilic inflammatory model. Eosinophilic and type 2 inflammatory endotypes occur in a clinically relevant subgroup, particularly among patients with asthma or allergic bronchopulmonary aspergillosis. Blood and sputum eosinophils and FeNO may provide information about these pathways, although their interpretation may be influenced by corticosteroid exposure, smoking, atopy, and concurrent infection. These findings reinforce the concept that biomarkers may be more useful for identifying biological endotypes than for providing a universal measure of disease severity. A clinically useful biomarker should therefore represent a defined biological pathway, remain sufficiently stable or predictably dynamic over time, and ideally identify patients likely to respond to pathway-specific treatment.

The interaction between microbiology and inflammation further complicates biomarker interpretation. Chronic *Pseudomonas aeruginosa* infection has been associated with greater disease burden, more frequent exacerbations, increased hospitalization and mortality, stronger neutrophilic activity, reduced microbial diversity, and higher

neutrophil elastase activity. However, most microbiome evidence remains observational, and cross-sectional associations cannot establish whether microbial alterations drive inflammatory activation or result from the inflammatory environment. Longitudinal studies are therefore needed to determine whether changes in microbial composition precede, accompany, or follow clinical exacerbations and inflammatory changes.

From a clinical perspective, statistical association alone is insufficient to establish biomarker utility. Biomarkers intended for routine use should be analytically reliable, biologically plausible, responsive to disease change, associated with clinically meaningful outcomes, and capable of improving treatment selection or monitoring. Current evidence suggests that neutrophil elastase comes closest to fulfilling these criteria, whereas CRP, fibrinogen, oxidative stress markers, eosinophilic markers, and other emerging candidates provide complementary but less definitive information. Biomarker interpretation should therefore be integrated with microbiology, lung function, symptoms, radiological findings, comorbidities, and inflammatory phenotype rather than being used in isolation.

Overall, the most promising future direction is the transition from individual biomarkers toward composite and multi-omic signatures. Proteomic, transcriptomic, metabolomic, microbiome, imaging, sputum, blood, and clinical data may collectively provide a more precise characterization of neutrophilic, eosinophilic, or mixed inflammatory endotypes and improve prediction of exacerbation risk and therapeutic response. However, such approaches require longitudinal validation, standardized sample collection and assay calibration, external validation across different populations and etiologies, and evidence that biomarker-guided strategies actually improve patient outcomes. Thus, biomarker development in bronchiectasis should move beyond simple correlation with disease severity toward clinically actionable tools that support risk stratification, treatment selection, and longitudinal monitoring within a precision-medicine framework.

5. Conclusion

Bronchiectasis is a very clinically and molecularly heterogeneous disease and inflammatory and oxidative stress biomarkers provide an important biological window into this disease. Neutrophil elastase is the most well-established airway biomarker, nets, myeloperoxidase, matrix metalloproteinases and inflammatory cytokines provide complementary information regarding neutrophil activation, proteolysis and airway injury. All four markers (crp, fibrinogen and markers of oxidative stress) are systemic markers more practical, and markers of oxidative stress provide more evidence of a biologically plausible redox imbalance, which require further analytical standardization. The identification of eosinophilic and mixed inflammatory endotypes further shows that there is no single biomarker to describe the whole spectrum of the disease. The most promising way to go is integration: the integration of the blood-based biological markers with the markers that are found in the sputum and exhaled air, the microbiology, the image and the multi-omics data. Future studies are needed to focus on longitudinal validation, to set validated thresholds, and to show the clinical utility, especially. Pathway-directed inhibitor of neutrophil proteases—an emerging success of biomarker-driven biology and disease-modifying therapy. Lastly, a validated composite biomarker tool could aid in the assessment of the efficacy of risk stratification, therapeutic selection and monitoring in precision bronchiectasis management.

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