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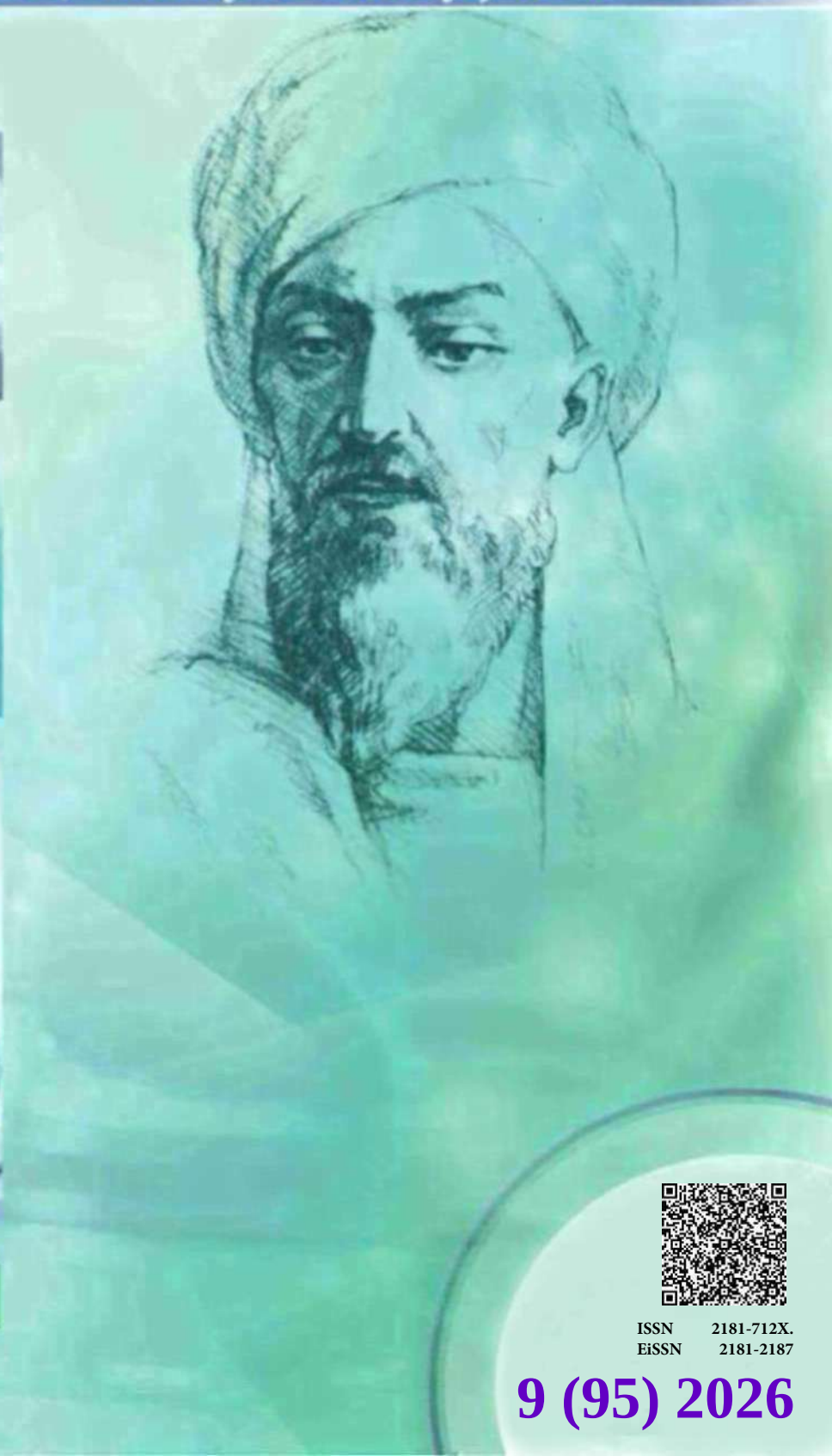


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ТИББИЁТДА ЯНГИ КУН НОВЫЙ ДЕНЬ В МЕДИЦИНЕ NEW DAY IN MEDICINE

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Научно-реферативный,
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INTEGRATIVE MSCT–MICROBIOTA PHENOTYPES OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE: A CROSS-SECTIONAL STUDY

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✓ Resume

Background: Chronic obstructive pulmonary disease (COPD) is structurally and microbiologically heterogeneous, but multislice computed tomography (MSCT) findings are rarely integrated with oropharyngeal and intestinal microbiota data in the same patients. **Objective:** To derive integrative MSCT-microbiota phenotypes of COPD and characterise their structural, functional, inflammatory, microbiological and psycho-neurovegetative profiles. **Methods:** In this single-centre cross-sectional study, 180 patients with COPD and 30 controls underwent MSCT, spirometry, oropharyngeal and stool culture with MALDI-TOF identification, measurement of CRP, IL-6 and TNF-alpha, heart-rate variability (HRV), and the MoCA, HADS and SGRQ instruments. Patients were assigned to one mutually exclusive MSCT-microbiota phenotype using a hierarchical rule prioritising bronchiectasis and resistant flora. **Results:** Four phenotypes were identified: inflammatory-dysbiotic (37.8%, n=68), bronchitic-colonisation (35.6%, n=64), emphysematous-dysbiotic (13.9%, n=25) and bronchiectatic-resistant (12.8%, n=23). The bronchiectatic-resistant phenotype had the lowest FEV1 (23%), the highest CRP (103 mg/L), the lowest SDNN (37.1±11.2 ms) and the lowest MoCA score (19.4±3.3), and by definition carried bronchiectasis in 100% of cases (all $p < 0.001-0.05$ across phenotypes). Combined oropharyngeal-intestinal colonisation was concentrated in the bronchitic-colonisation phenotype (75.0% of its patients) and was absent by definition from the inflammatory-dysbiotic phenotype. **Conclusion:** Integrating MSCT with dual-compartment microbiota data identifies four clinically distinct COPD phenotypes with different dominant pathogenetic mechanisms, which may help target antimicrobial stewardship and probiotic correction.

Keywords: chronic obstructive pulmonary disease; multislice computed tomography; phenotype; oropharyngeal microbiota; intestinal dysbiosis; bronchiectasis.

ИНТЕГРАТИВНАЯ МСКТ–ДИАГНОСТИКА ФЕНОТИПОВ МИКРОБИОТЫ ПРИ ХРОНИЧЕСКОЙ ОБСТРУКТИВНОЙ БОЛЕЗНИ ЛЕГКИХ: ПЕРЕКРЕСТНОЕ ИССЛЕДОВАНИЕ

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✓ Резюме

Хроническая обструктивная болезнь легких (ХОБЛ) структурно и микробиологически неоднородна, но результаты мультиспиральной компьютерной томографии (МСКТ) редко

сопоставляются с данными орофарингеальной и кишечной микробиоты у одних и тех же пациентов. Цель: получить интегративные фенотипы ХОБЛ с помощью МСКТ-микробиоты и охарактеризовать их структурные, функциональные, воспалительные, микробиологические и психонейровегетативные профили. Методы: В этом одноцентровом перекрестном исследовании 180 пациентам с ХОБЛ и 30 пациентам контрольной группы были проведены МСКТ, спирометрия, посев ротоглотки и кала с определением MALDI-TOF, измерение СРБ, IL-6 и TNF-альфа, вариабельности сердечного ритма (BCP), а также MoCA, HADS и Инструменты SGRQ. Пациенты были отнесены к одному взаимоисключающему фенотипу МСКТ-микробиоты с использованием иерархического правила, определяющего приоритетность бронхоэктазов и резистентной флоры. Результаты: Были выявлены четыре фенотипа: воспалительно-дисбиотический (37,8%, n=68), бронхиально-колонизационный (35,6%, n=64). Сочетанная орофарингеально-кишечная колонизация была сосредоточена в фенотипе бронхиальной колонизации (у 75,0% пациентов) и по определению отсутствовала в фенотипе воспалительно-дисбиотическом. Заключение: Интеграция МСКТ с данными двухкомпонентной микробиоты позволяет выявить четыре клинически различных фенотипа ХОБЛ с различными доминирующими патогенетическими механизмами, которые могут помочь в целенаправленном лечении антимикробными препаратами и коррекции пробиотиками.

Ключевые слова: хроническая обструктивная болезнь легких; мультиспиральная компьютерная томография; фенотип; микробиота ротоглотки; дисбактериоз кишечника; бронхоэктатическая болезнь.

SURUNKALI OBSTRUKTIV O'PKA KASALLIGINING INTEGRAL MSCT-MIKROBIOTA FENOTIPLARI: KESMA TADQIQOT

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✓ Rezyume

Fon: surunkali obstruktiv o'pka kasalligi (KOA) tizimli va mikrobiologik jihatdan heterojen, ammo multislice kompyuter tomografiyasi (MSCT) topilmalari kamdan-kam hollarda bir xil bemorlarda orofaringeal va ichak mikrobiota ma'lumotlari bilan birlashtiriladi. **Maqsad:** KOAHNING integral MSCT-mikrobiota fenotiplarini olish va ularning tarkibiy, funktsional, yallig'lanish, mikrobiologik va psixo-neyrovegetativ profillarini tavsiflash. **Usullari:** bu yagona-markazi ko'ndalang kesimi ishda, KOAH va 180 elementlari bilan 30 bemorlar MALDI-TOF aniqlash, CRP, IL-6 va Tnf-alfa o'lchash bilan MSCT, spirometriya, orofaringeal va axlat madaniyati o'tdi, yurak-darajasi o'zgaruvchanlik (HRV), va MoCA, HADS va SGRQ asboblari. Bemorlar bronxoektaziya va chidamli floraga ustuvor bo'lgan ierarxik qoidadan foydalangan holda bir-birini istisno qiladigan MSCT-mikrobiota fenotipiga tayinlangan. **Natijalar:** to'rtta fenotip aniqlandi: yallig'lanish-disbiyotik (37,8%, n=68), bronxitik-kolonizatsiya (35,6%, n=64),

Kombinatsiyalangan orofaringeal-ichak kolonizatsiyasi bronxitik-kolonizatsiya fenotipida (uning bemorlarining 75,0%) to'plangan va yallig'lanish-disbiyotik fenotipning ta'rifi bo'yicha yo'q edi. **Xulosa:** MSCT-ni ikki bo'linmali mikrobiota ma'lumotlari bilan birlashtirish turli xil dominant patogenetik mexanizmlarga ega to'rtta klinik jihatdan alohida KOAH fenotiplarini aniqlaydi, bu antimikrobiyal boshqaruv va Probiyotik tuzatishga yordam beradi.

Kalit so'zlar: surunkali obstruktiv o'pka kasalligi; multislisli kompyuter tomografiyasi; fenotip; orofaringeal mikrobiota; ichak disbiyozi; bronxoektaziya.

Relevance

Chronic obstructive pulmonary disease (COPD) is increasingly recognised as a heterogeneous, systemic disease rather than a single entity defined solely by airflow limitation. Structural imaging by multislice computed tomography (MSCT) distinguishes emphysematous, bronchitic, mixed and bronchiectatic patterns, while a growing body of evidence implicates dysbiosis of the oropharyngeal and intestinal microbiota, mediated through the gut-lung axis, in disease progression and systemic inflammation [1,2]. Most published work addresses these dimensions separately: imaging phenotype studies rarely include culture-based microbiota data, and microbiota studies in COPD, including our own previous single-compartment reports, rarely incorporate a structural correlate [3].

Combining structural, microbiological, inflammatory and psycho-neurovegetative data in the same patients may allow the derivation of integrative phenotypes that better capture the dominant pathogenetic mechanism operating in an individual patient, and may therefore be more informative for targeted therapy than either domain alone. We hypothesised that four clinically distinct integrative phenotypes could be derived from MSCT pattern, dual-compartment (oropharyngeal and intestinal) colonisation status, and antibiotic-resistance profile.

The aim of this study was to derive integrative MSCT-microbiota phenotypes of COPD and to characterise their structural, functional, inflammatory, microbiological and psycho-neurovegetative profiles in a well-characterised single-centre cohort.

Materials and methods

Design, setting and participants

This was a single-centre, cross-sectional observational study conducted at the Republican Specialised Scientific-Practical Medical Centre of Therapy and Medical Rehabilitation, Tashkent, Uzbekistan. The study enrolled 180 patients with physician- and spirometry-confirmed COPD (post-bronchodilator FEV1/FVC <0.70) and 30 controls without chronic bronchopulmonary disease, matched for age, sex and smoking status. Patients with active tuberculosis, pulmonary malignancy, severe immunosuppression, decompensated comorbidity, or systemic antibiotic, probiotic, prebiotic or synbiotic use within the preceding 4 weeks were excluded. For analysis, COPD patients were grouped as GOLD I-II (n=81) and GOLD III-IV (n=99).

The study protocol was approved by the Institutional Review Board/Ethics Committee of Tashkent Pediatric Medical Institute, now Tashkent State Medical University following institutional reorganisation (Approval No. 14, 25 January 2025). Written informed consent was obtained from all participants.

MSCT assessment

All COPD patients underwent MSCT of the chest. Patients were classified into one of three mutually exclusive primary patterns — emphysematous, bronchitic or mixed — with bronchiectasis recorded separately as an overlapping structural feature that could accompany any primary pattern.

Microbiological and inflammatory assessment

Oropharyngeal swabs and stool specimens were cultured quantitatively; isolates were identified by MALDI-TOF mass spectrometry (Bruker Biotyper). Opportunistic/pathogenic oropharyngeal and intestinal flora were defined according to standard laboratory reference ranges, and an intestinal dysbiosis index was calculated. Antimicrobial susceptibility was determined by disk diffusion and interpreted by EUCAST criteria; isolates non-susceptible to at least one agent in three or more antimicrobial classes were classified as multidrug-resistant (MDR). CRP, IL-6 and TNF-alpha were measured by standard laboratory assays.

Autonomic, cognitive and quality-of-life assessment

Heart-rate variability (HRV; SDNN, RMSSD, LF/HF) was derived from 10-minute resting recordings. Cognitive status was assessed with the Montreal Cognitive Assessment (MoCA), anxiety and depression with the Hospital Anxiety and Depression Scale (HADS), and health-related quality of life with the St George's Respiratory Questionnaire (SGRQ).

Phenotype derivation

Each patient was assigned to exactly one integrative phenotype using a hierarchical, MSCT-microbiota-based rule. Patients with bronchiectasis and resistant flora were assigned to the bronchiectatic-resistant phenotype irrespective of other findings, reflecting the greatest structural and

microbiological burden. Remaining patients were classified as emphysematous-dysbiotic (emphysema without bronchiectasis plus intestinal dysbiosis), bronchitic-colonisation (bronchitic/mixed MSCT pattern plus oropharyngeal opportunistic flora, with or without intestinal dysbiosis), or inflammatory-dysbiotic (bronchitic/mixed MSCT pattern plus intestinal dysbiosis, without oropharyngeal opportunistic flora).

Statistical analysis

Continuous variables were tested for normality (Shapiro-Wilk) and expressed as mean $\pm$ SD or median [IQR] as appropriate; categorical variables were expressed as n (%). Comparisons across the four phenotypes used one-way ANOVA or the Kruskal-Wallis test for continuous variables and the chi-squared or Fisher exact test for categorical variables. A two-sided $p < 0.05$ was considered statistically significant.

Results and discussions

Phenotype distribution

Four mutually exclusive integrative MSCT-microbiota phenotypes were identified among the 180 COPD patients: inflammatory-dysbiotic (37.8%, $n=68$), bronchitic-colonisation (35.6%, $n=64$), emphysematous-dysbiotic (13.9%, $n=25$) and bronchiectatic-resistant (12.8%, $n=23$) (Table 1). Combined oropharyngeal-intestinal colonisation was present overall in 62/180 patients (34.4%) and was concentrated in the bronchitic-colonisation phenotype (48/64, 75.0%); by definition it was absent from the inflammatory-dysbiotic phenotype.

Table 1. Criteria for and distribution of integrative COPD phenotypes

Phenotype	n	%	Assignment criteria	Combined oropharyngeal + intestinal colonisation
Bronchitic-colonisation	64	35.6	Bronchitic/mixed MSCT + oropharyngeal opportunistic flora ($\pm$ intestinal dysbiosis)	48 (75.0%)
Inflammatory-dysbiotic	68	37.8	Bronchitic/mixed MSCT + intestinal dysbiosis, oropharynx without opportunistic flora	0 (0%)
Emphysematous-dysbiotic	25	13.9	Emphysema without bronchiectasis + intestinal dysbiosis	5 (20.0%)
Bronchiectatic-resistant	23	12.8	Any MSCT pattern with bronchiectasis (highest priority) + resistant flora	9 (39.1%)

Data are n (%). Assignment was hierarchical and mutually exclusive (see Methods).

MSCT features by phenotype

Emphysema was most frequent in the emphysematous-dysbiotic phenotype (90.5% of its patients), bronchial-wall thickening and peribronchial change predominated in the bronchitic-colonisation and bronchiectatic-resistant phenotypes, and bronchiectasis was present in 100% of the bronchiectatic-resistant phenotype by definition (Table 2).

Table 2. Distribution of MSCT features across integrative phenotypes (% of patients within phenotype)

CT feature	Emphysematous-dysbiotic	Bronchitic-colonisation	Bronchiectatic-resistant	Inflammatory-dysbiotic
Emphysema	90.5	31.4	52.8	24.7
Bronchial wall thickening	28.6	82.4	76.4	35.1
Bronchiectasis	4.8	18.6	100.0	7.0
Bullous changes	61.9	17.6	38.9	14.0
Peribronchial changes	23.8	78.4	83.3	29.8

Clinical severity, inflammation and dysbiosis pattern

The bronchiectatic-resistant phenotype had the lowest FEV1 (23% predicted) and the highest CRP (103 mg/L) of the four phenotypes (both $p < 0.001$, Kruskal-Wallis). The dominant dysbiosis pattern also differed by phenotype: the inflammatory-dysbiotic phenotype showed the highest rate of reduced *Lactobacillus* (76%), the emphysematous-dysbiotic phenotype the highest rate of reduced *Bifidobacterium* (72%), and the bronchiectatic-resistant phenotype the highest rate of Enterobacteriaceae overgrowth (30%) (Table 3). Autonomic, cognitive and quality-of-life profile

HRV, mood, cognitive and quality-of-life measures also differed significantly by phenotype (Table 4). The bronchiectatic-resistant phenotype had the lowest SDNN (37.1 ± 6.9 ms), the lowest RMSSD (39.7 ± 8.2 ms) and the lowest MoCA score (19.4 ± 3.3) of the four groups ($p < 0.001$ - 0.001). The emphysematous-dysbiotic phenotype showed the highest anxiety and depression scores (HADS-A 11.4 ± 3.5 ; HADS-D 15.2 ± 3.6). SGRQ scores trended toward a difference between phenotypes but did not reach statistical significance ($p = 0.082$).

Table 3. Clinical severity, inflammation and gut dysbiosis pattern by integrative phenotype

Parameter	Bronchitic-col.	Inflamm.-dysb.	Emphys.-dysb.	Bronchiectatic-res.	p
FEV1, %	50	57	44	23	<0.001
CRP, mg/L	45	32	56	103	<0.001
<i>Lactobacillus</i> below reference, %	62	76	52	52	<0.05
<i>Bifidobacterium</i> below reference, %	69	66	72	52	<0.05
Enterobacteriaceae overgrowth, %	9	16	12	30	<0.01

Kruskal-Wallis test across the four phenotypes.

Table 4. Heart-rate variability, HADS, MoCA and SGRQ by integrative phenotype

Parameter	Emphys.-dysb. (n=25)	Bronchitic-col. (n=64)	Bronchiectatic-res. (n=23)	Inflamm.-dysb. (n=68)	p
SDNN, ms	46.3 ± 11.2	48.0 ± 14.0	37.1 ± 6.9	52.5 ± 13.8	<0.001
RMSSD, ms	48.8 ± 12.8	45.4 ± 9.0	39.7 ± 8.2	50.0 ± 11.1	<0.001
LF/HF	3.1 ± 1.9	2.5 ± 2.0	2.5 ± 1.6	2.2 ± 1.9	<0.05
HADS-A	11.4 ± 3.5	9.7 ± 4.6	9.7 ± 2.1	8.8 ± 4.4	<0.05
HADS-D	15.2 ± 3.6	13.0 ± 5.3	14.0 ± 2.4	12.0 ± 5.2	<0.05
MoCA, points	23.1 ± 3.8	21.8 ± 4.1	19.4 ± 3.3	22.9 ± 3.6	0.001
SGRQ, points	40.4 ± 14.8	34.5 ± 16.1	27.4 ± 13.2	35.4 ± 16.5	0.082

This study derived four integrative MSCT-microbiota phenotypes of COPD from structural, dual-compartment microbiological, inflammatory and psycho-neurovegetative data collected in the same 180 patients. Each phenotype was anchored in a distinct dominant mechanism: bronchial colonisation with oropharyngeal opportunists in the bronchitic-colonisation phenotype, isolated intestinal dysbiosis without oropharyngeal involvement in the inflammatory-dysbiotic phenotype, emphysematous remodelling with intestinal dysbiosis in the emphysematous-dysbiotic phenotype, and a combination of bronchiectasis with resistant flora in the bronchiectatic-resistant phenotype.

The bronchiectatic-resistant phenotype, although the smallest group (12.8%), carried the greatest structural, functional, inflammatory and autonomic burden, consistent with the concept that bronchiectasis creates a mucosal niche favouring persistence of resistant opportunistic flora and chronic antigenic stimulation [2,4]. The concentration of combined oropharyngeal-intestinal colonisation within the bronchitic-colonisation phenotype (75.0%) suggests that bronchial-wall remodelling and impaired mucociliary clearance may facilitate simultaneous derangement of both microbial compartments, consistent with a shared upper-airway-gut axis rather than independent single-organ processes [1,3].

The differing dominant dysbiosis pattern across phenotypes — *Lactobacillus* depletion in the inflammatory-dysbiotic phenotype, *Bifidobacterium* depletion in the emphysematous-dysbiotic

phenotype, and Enterobacteriaceae overgrowth in the bronchiectatic-resistant phenotype — argues against a single, uniform dysbiosis signature in COPD and instead supports phenotype-specific microbial mechanisms, which may have implications for targeted probiotic selection.

Strengths and limitations

Strengths include the simultaneous acquisition of MSCT, dual-compartment culture-based microbiota data, inflammatory markers, HRV and validated psychometric instruments in a well-defined single-centre cohort. Limitations include the cross-sectional design, which precludes causal inference about phenotype stability or transition over time; the single-centre setting; the reliance on culture-based rather than sequencing-based microbiota characterisation; and the hierarchical assignment rule, which by construction prevents overlap between phenotypes and may somewhat understate the true prevalence of combined structural-microbial abnormality. External validation in an independent cohort is required.

Conclusion

Integrating MSCT pattern with oropharyngeal and intestinal microbiota data identifies four integrative phenotypes of COPD with distinct structural, inflammatory, dysbiosis and psycho-neurovegetative profiles. The bronchiectatic-resistant phenotype represents the most severe combination of structural damage, systemic inflammation, autonomic dysfunction and antimicrobial resistance risk, and may warrant the most intensive microbiological surveillance and targeted correction.

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Conflict of interest

The authors declare no conflicts of interest.

Data availability

The anonymised dataset is available from the corresponding author on reasonable request, subject to institutional and ethical restrictions.

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