



Article

# Microbiota of the Upper Respiratory Tract in Patients With Severe Bronchial Asthma

Satlikov Rashid Karimovich<sup>\*1</sup>, Aripova Tamara Uktamovna<sup>2</sup>, Abdullayev Ravshanbek Babajonovich<sup>3</sup>, Ziyadullayev Shuhrat Khudoyberganovich<sup>4</sup>

1,3. Urgench State Medical Institute

2,4. Institute of Immunology and Human Genomics, Academy of Sciences of The Republic of Uzbekistan

\* Correspondence: [rashidsatlikov7@gmail.com](mailto:rashidsatlikov7@gmail.com)

**Abstract:** A total of 26 severe bronchial asthma patients were studied (9 men – 34.6%, 17 women – 65.4%; mean age  $\pm$  years,  $41.9 \pm 15.8$ ). Disease severity was classified according to GINA-2024. Aerobic bacteria were isolated in 83–85% of nasopharyngeal specimens. The identified isolates were *Staphylococcus epidermidis* (57.7%), *Staphy aureus* (30.8%) and *Staphy haemolyticus* (3.8%) in addition to *Candida* spp. (11.5%). For *S. aureus*, 60% was MRSA resistant to  $\beta$ -lactams; macrolides and tetracyclines while all strains were susceptible to aminoglycosides and co-trimoxazole. *Candida* spp. isolates were highly resistant to azole antifungals. These results show that bacteria frequently staphylococci, resistant ones included are part of the flora of severe asthma and suggest that surveillance for microbiological data combined with anti-infective therapy based on solid evidence are warranted.

**Keywords:** Bronchial Asthma, Nasopharyngeal Microbiota, *Staphylococcus* *Aureus*, *Staphylococcus* *Epidermidis*, Antimicrobial Resistance, Colonization

## 1. Introduction

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Asthma is a chronic condition characterized by inflammation in the airways, with various cells and elements involved in its pathophysiology. Around 5-10% of asthma patients experience severe forms of the disease that remain difficult to manage, despite the use of high-dose inhaled corticosteroids (ICS) combined with long-acting beta2-agonists[1-2]. These individuals often face frequent exacerbations, ongoing symptoms, and a significant impact from the side effects of their medications. Although systemic glucocorticosteroids (GCS) continue to be commonly used in treating severe asthma, they come with notable side effects, including weight gain, secondary diabetes, osteoporosis, hypertension, cataracts, adrenal suppression, and psychological issues. Short-term use of systemic GCS can also pose risks such as sleep disturbances, infections, and thromboembolic events[3-4]. Therefore, identifying additional factors that influence disease progression is of paramount importance.

The importance of the respiratory microbiome in asthma pathogenesis has recently been acknowledged. It has been demonstrated that the microbial dysbiosis of asthmatic airways is significantly different from non-asthmatics indicating variation in terms of microbial diversity and local immunomodulation between the two phenotypes [5-6]. The concept of “infectious asthma” was first described decades ago, based on the clinical

response to macrolides, like triacetyloleandomycin [7]. Subsequent studies have revealed increased rates of *Mycoplasma pneumoniae* and *Chlamydophila pneumoniae* in airway samples from asthmatics, and response to appropriate antibacterial therapy [8-9]. It seems that the gut and airway microbiota can both impact immune system maturation, as well as disease severity, implying that microbial dysbiosis might contribute to different asthma phenotypes and responses to treatment.

The objective of the present article was to assess microbial populations in upper respiratory tracts from patients with severe bronchial asthma as well as bacteriological and phage-typing characteristics of isolated microorganisms[10-11].

## 2. Materials and Methods

The study involved 26 patients with diagnosed severe bronchial asthma. Twenty-six patients (65.4%) were women and nine (34.6%) men, with an age range of 18–72 years (mean  $41.9 \pm 15.8$ ). The study population consisted of 88.50% adults and 11.50% children. Severity and control of disease were measured based on GINA-2024 classification criteria and validated questionnaires that is, ACT and ACQ-7. None of the subjects had received antibacterial or antifungal medication in the 2 months prior to inclusion [12-13]. All persons gave their informed consent, and the protocol was approved by the local ethics committee of Institute of Immunology and Human genomics 12which is a part of academy of sciences Uzbekistan13.

Nasopharyngeal samples were taken using sterile swabs and transported in specific transport media. Blood, yolk-salt, Endo and Sabouraud agars were inoculated in the studies. The plates were incubated at  $+36^{\circ}\text{C}$  according to the standard procedure for 24–48 h, then kept and further observed without post-mortem treatment up to five days. Colony formation could generally be seen on days 2–4. Species identification was performed with MALDI-TOF MS (Microflex Lt/Sh, Bruker Daltonics) following isolation of colonies. A score of  $\geq 2.0$  was defined as a reliable identification. The in vitro antibacterial and antifungal sensitivity pattern was assessed by EUCAST-2025 guidelines. The data were analyzed using Statistica 10.0. Values are given as  $M \pm m$ . The differences were considered significant at  $p < 0.05$ .

## 3. Results

Microbial growth on blood agar and YSA occurred in 83–85% of samples. No gram-negative bacteria were detected on Endo agar. *Candida* spp. colonies appeared in three cases.

### Microbiological findings

**Table 1.** The most frequently isolated organisms.

Microorganism	Number of patients	Percentage (%)	Note
<i>Staphylococcus epidermidis</i>	15	57,7%	Commensal flora
<i>Staphylococcus aureus</i>	8	30,8%	60% MRSA
<i>Staphylococcus haemolyticus</i>	1	3,8%	Rare isolate
<i>Candida</i> spp.	3	11,5%	Found together with <i>S. aureus</i>
Unidentified strains	4	7,7%	Low biomass

Table 1 is Approximately one-third of patients were carriers of *S. aureus*, consistent with published data [14]. The distribution was similar across age and sex groups.

Most isolates were resistant to benzylpenicillin (100%) and cefoxitin (up to 67%), confirming MRSA status. Resistance to macrolides (erythromycin) and tetracyclines was also frequent. Susceptibility was highest to aminoglycosides, co-trimoxazole, rifampicin, and doxycycline. All fungal isolates were resistant to nystatin, fluconazole, ketoconazole, and amphotericin B. Only itraconazole showed partial effectiveness (67% susceptibility).

#### 4. Discussion

The present study indicates that patients with severe bronchial asthma frequently carry coagulase-negative staphylococci, mainly *S. epidermidis*, which are otherwise mucosal commensals. Their high level could imply a compensatory colonization or low competition with the pathogenic ones. The sizeable number of *S. aureus* carriers, including those colonized by MRSA is of special clinical interest. Empirical therapy for resistant strains is also problematic in patients who are on glucocorticoid treatment more frequently. The identification of *Candida* spp. in some cases might simply represent secondary changes in the microbiota that were accelerated because of previous antibiotic treatment or decreased local immune protection. These findings highlight the necessity of a focused microbiological vigilance, especially in high-risk groups with chronic inflammatory airway disease[15].

#### 5. Conclusion

The upper respiratory tract in patients with severe bronchial asthma is predominantly colonised by *Staphylococcus epidermidis* and *Staphylococcus aureus*. Approximately 60% of *S. aureus* strains are methicillin-resistant and multidrug-resistant, although susceptibility is high to aminoglycosides, rifampicin, doxycycline, and co-trimoxazole. Combined colonization with *Candida* spp. may require additional clinical evaluation. Antibacterial activity in bronchial secretions of controls and patients with severe asthma is suppressed: the role of systemic glucocorticoid treatment.

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