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Research Article

Nasal Carriage of Methicillin-Resistant *Staphylococcus aureus* in Prediabetes and Type 2 Diabetes Mellitus

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ABSTRACT

Aim: *Staphylococcus aureus (S. aureus)* nasal colonization, especially with methicillin-resistant *S. aureus* (MRSA), is a significant risk factor for infection and transmission. While diabetes mellitus (DM) is a recognized risk factor, its association with prediabetes (preDM) is less apparent. This study investigated MRSA nasal carriage in DM and preDM and its relation to metabolic and anthropometric parameters.

Material and Methods: This cross-sectional study included 300 participants: 100 with type 2 DM, 100 with preDM and 100 non-diabetic controls attending General Internal Medicine clinics (May–August 2025). PreDM and DM were defined by American Diabetes Association (ADA) criteria and individuals with recent antibiotic use, acute infection, immunosuppression, dialysis or malignancy were excluded. Nasal swabs were cultured on blood agar, identified by MALDI-TOF and tested for methicillin resistance using oxacillin MIC per Clinical and Laboratory Standards Institute (CLSI M100 35th ed., 2025) standards. Clinical and laboratory parameters were assessed using nonparametric statistical tests, with significance set at $p < 0.05$.

Results: MRSA nasal carriage was higher in DM patients than controls (8% vs 1%, p=0.035). DM was associated with greater waist circumference and poorer glycemic profiles. MRSA carriage showed weak positive correlations with body weight, fasting glucose, HbA1c and triglycerides, and a weak negative correlation with albumin. Prediabetes showed a nonsignificant increase compared with controls.

Conclusion: Type 2 DM was associated with increased MRSA nasal carriage, whereas prediabetes was not; intermediate rates in prediabetes do not support routine MRSA screening in this group. These findings support a selective, risk-based approach rather than routine MRSA screening in prediabetic populations.

Keywords: *Staphylococcus aureus*, methicillin-resistant *Staphylococcus aureus*, nasal carriage, prediabetes, diabetes mellitus.

INTRODUCTION

Staphylococcus aureus (S. aureus) is a Gram-positive bacterium characterized by numerous virulence factors that are responsible for infections ranging from superficial skin conditions to severe systemic illnesses, including bacteraemia, endocarditis and pneumonia, by enabling tissue adhesion, invasion and evasion of the host immune response (1,2). In addition to its pathogenic potential, *S. aureus* is a frequent colonizer of the anterior nares, and a substantial proportion of the population exhibits asymptomatic nasal carriage (3,4). Methicillin-resistant *S. aureus* (MRSA) infections are linked to increased morbidity, mortality, prolonged hospitalization and higher healthcare costs (5). In line with CLSI interpretive criteria, an oxacillin MIC of ≥4 µg/mL was considered methicillin resistance in *S. aureus* (6). Diabetes mellitus is a recognized risk factor for MRSA colonization (7).

Prediabetes (PreDM), defined as impaired glucose metabolism not yet meeting the threshold for diabetes mellitus (DM), is associated with immune dysfunction and reduced host defence mechanisms (8). Impaired leukocyte activity, microvascular alterations, and disruption of epithelial barrier integrity may facilitate bacterial adhesion and colonization. Both prediabetic and diabetic individuals with disturbed glucose metabolism have been shown to exhibit significant immune system dysfunctions (9,10). Despite these well-documented pathophysiological alterations, existing studies on nasal colonization with *S. aureus* and MRSA have focused almost exclusively on patients with overt diabetes mellitus. Prediabetic individuals are either excluded from the analysis or not evaluated as a distinct risk group, leaving a critical gap in our understanding of whether early metabolic dysregulation is sufficient to influence bacterial colonization. Consequently, the prevalence and metabolic correlates of nasal *S. aureus* and MRSA carriage in prediabetes remain largely undefined. The present study was designed to specifically address this gap by evaluating MRSA nasal carriage in prediabetic individuals and examining its association with metabolic and anthropometric parameters. From a clinical perspective, it remains unclear whether prediabetic individuals constitute a population for whom MRSA nasal screening would be meaningful or necessary. While routine MRSA surveillance is not currently recommended for prediabetes in existing guidelines, the absence of evidence does not equate to evidence of absence. Clarifying whether early metabolic dysregulation is associated with a clinically relevant increase in MRSA colonization is essential for determining the potential value of screening strategies in this growing population.

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MATERIAL AND METHODS

The cross-sectional study was initiated after receiving approval from the Ethics Committee of the Institute of Health Sciences Research at Sivas Cumhuriyet University on April 24, 2025, under committee number 2025-04/105. The study included patients diagnosed with DM, prediabetic patients and healthy volunteers who visited the General Internal Medicine outpatient clinics for other reasons between May 2025 and August 2025. A total of 300 participants were enrolled, comprising 100 patients with type 2 DM, 100 patients with preDM and 100 non-diabetic individuals as controls. Definitions followed the American Diabetes Association (ADA) Standards of Care and were based on fasting blood glucose (FBG) and glycated hemoglobin (HbA1c) values. Prediabetes was defined as FBG 100–125 mg/dL and/or HbA1c 5.7–6.4%, and diabetes as FBG ≥126 mg/dL and/or HbA1c ≥6.5% (11). Patients with a history of antibiotic use within the previous four weeks, those with acute infection, individuals receiving immunosuppressive therapy, patients undergoing hemodialysis and those diagnosed with malignancy were excluded from the study.

Sample Collection: Nasal specimens were collected bilaterally from the anterior nares of each participant using sterile, cotton-tipped swabs. Samples were processed immediately in the microbiology laboratory. Each specimen was directly inoculated onto blood agar. Blood agar culture was subjected to MALDI-TOF mass spectrometry for species confirmation.

Identification of MRSA: Colony morphology was used to identify the *S. aureus* isolates and standard microbiological techniques were used for confirmation. In accordance with the Clinical and Laboratory Standards Institute (CLSI) criteria, methicillin resistance was identified by oxacillin minimum inhibitory concentration (MIC) testing.

Identification of MRSA: Suspected *S. aureus* colonies were identified on blood agar and species confirmation was performed by MALDI-TOF mass spectrometry. Methicillin resistance was determined by oxacillin MIC testing using the broth microdilution method in cation-adjusted Mueller–Hinton broth. Briefly, a 0.5 McFarland suspension was prepared from overnight growth and diluted to yield a final inoculum of approximately 5×10^5 CFU/mL in each well of a 96-well microtiter plate containing serial twofold dilutions of oxacillin. Plates were incubated at 35±1°C and read at 24 hours; the MIC was defined as the lowest oxacillin concentration with no visible growth. Interpretation followed CLSI M100 (35th edition, 2025): oxacillin MIC ≤2 µg/mL was considered susceptible, and ≥4 µg/mL was considered resistant (MRSA) (6). Quality control was performed using *S. aureus* ATCC 29213 (methicillin-susceptible) and *S. aureus* ATCC 43300 (MRSA) in accordance with CLSI recommendations (6). Cefoxitin testing was not used because the primary method for determining methicillin resistance in this study was the oxacillin MIC.

Data Collection: Anthropometric data (height, weight, body mass index [BMI], waist circumference), clinical variables (duration of diabetes mellitus, insulin use, hospitalization history) and demographic characteristics (age, sex) were all documented. At the time of registration, laboratory information was gathered from hospital records, including complete blood count, lipid profile, liver and kidney function tests, electrolytes, micronutrients and thyroid parameters.

Statistical Analysis

The study sample size was established through an a priori power analysis performed using G*Power software (version 3.1.9.4), based on a one-way analysis of variance (ANOVA; fixed-effects, omnibus). The calculation assumed an effect size (Cohen's f) of 0.229, a type I error rate (α) of 0.05 and a desired statistical power (1−β) of 0.95. Assuming equal group sizes, the total required sample size was estimated at 300 participants. All statistical analyses were carried out using the IBM SPSS Statistics version 23.0 (IBM Corp., Armonk, NY, USA). The distribution of continuous variables was assessed using the Shapiro–Wilk and Kolmogorov–Smirnov tests. For variables that did not follow a normal distribution, results were summarized as medians with interquartile ranges (IQR). Group comparisons for non-normally distributed variables were conducted using the Kruskal–Wallis test or the Mann–Whitney U test, as appropriate. Categorical variables were expressed as frequencies (n) and percentages (%) and compared using the chi-square test or Fisher's exact test. For 2×3 contingency tables with smaller than expected cell counts, a Monte Carlo simulation was applied to improve the accuracy of chi-square analyses. Associations between variables were examined using Spearman's rho correlation analysis. All statistical tests were two-sided, and a p-value < 0.05 was considered statistically significant.

RESULTS

A total of 300 patients were included in the study: 100 with DM, 100 with preDM and 100 in the control group. The demographic, anthropometric and MRSA carriage characteristics of the study groups are provided in Table 1. The median age of patients in the DM group was 61 (54–65) years, while it was 47 (43–59) and 40 (35–48) years in the preDM and control groups, respectively. There was a statistically significant age difference between the groups ($p < 0.001$). In terms of anthropometric measurements, the median height of the DM group was 160 (157–170) cm, while it was 163 (163–175) cm and 165 (160–175) cm in the preDM and control groups, respectively. Patients in the DM group were significantly shorter than those in the other groups ($p < 0.001$). Body weight ($p = 0.685$) and body mass index (BMI) ($p = 0.107$) did not significantly differ across groups. In terms of waist circumference, the median waist circumference of patients in the DM group was 100 (89–110) cm, 90 (90–91) cm in the preDM group and 87 (80–90) cm in the control group; waist circumference was significantly higher in the DM group ($p < 0.001$). MRSA carriage differed significantly between the DM group and the control group, with eight patients in the DM group and one in the control group ($p = 0.035$).

Table 1. Demographic, anthropometric and MRSA carriage characteristics of the study groups

	Diabetes (n=100)	Prediabetes (n=100)	Control (n=100)	p
Age (years)	61 (54-65)	47 (43-59)	40 (35-48)	<0.001*
Male gender, n (%)	40 (40%)	33 (33%)	46 (46%)	0.171
Duration of diabetes (years)	10 (5-12)	-	-	-
Height (cm)	160 (157-170)	163 (163-175)	165 (160-175)	0.001*
Weight (kg)	80 (72-88)	79 (79-86)	75 (70-83)	0.685
Body mass index (kg/m²)	31 (26-35)	29 (28-31)	27 (27-27)	0.107
Waist circumference (cm)	100 (89-110)	90 (90-91)	87 (80-90)	<0.001*
MRSA carriage, n (%)	8 (8%)	3 (3%)	1 (1%)	0.049*

Data are presented as median (interquartile range) or number (%). *MRSA: methicillin-resistant Staphylococcus aureus. p* values < 0.05 were considered statistically significant.

Three patients in the preDM group were colonized with MRSA, but there was no statistically significant difference between them and the DM group (p = 0.621).

Key metabolic laboratory parameters are summarized in Table 2. As expected, HbA1c and fasting blood glucose were highest in the DM group, intermediate in the preDM group and lowest in controls (both p < 0.001). Triglyceride (TG) levels were higher in DM than in the other groups, whereas low-density lipoprotein cholesterol (LDL-C) and total cholesterol were highest in preDM (all p ≤ 0.001). high-density lipoprotein-cholesterol (HDL-C) was lowest in DM (p = 0.001). Serum albumin differed across groups (p < 0.001).

Table 2. Metabolic laboratory parameters of the study groups

	Diabetes (n=100)	Prediabetes (n=100)	Control (n=100)	p
HbA1c (%)	7.5 (6.8-8.8)	6.1 (5.7-6.2)	5.4 (5.2-5.5)	<0.001*
FBG (mg/dl)	143 (118-183)	93 (89-100)	60 (84-97)	<0.001*
TG (mg/dl)	137 (94-192)	118 (66-149)	94 (70-122)	<0.001*
LDL-C (mg/dl)	109 (94-137)	129 (116-140)	110 (93-124)	<0.001*
HDL-C (mg/dl)	44 (38-51)	48 (47-53)	49 (42-54)	0.001*
TC (mg/dl)	179 (156-206)	193 (177-207)	174 (154-192)	<0.001*
Albumin (g/L)	43 (41-45)	46 (43-46)	44 (43-46)	<0.001*

Data are presented as median (interquartile range). HbA1c: hemoglobin A1C; FBG: fasting blood glucose; TG: triglyceride; LDL-C: low-density lipoprotein-cholesterol; HDL-C: high-density lipoprotein-cholesterol; TC: total cholesterol. p values < 0.05 were considered statistically significant.

As shown in Table 3, Spearman's correlation analysis demonstrated that MRSA carriage was weakly positively correlated with body weight (ρ = 0.163, p = 0.005), BMI (ρ = 0.219, p < 0.001), waist circumference (ρ = 0.195, p = 0.001), fasting blood glucose (ρ = 0.125, p = 0.030), HbA1c (ρ = 0.140, p = 0.015) and triglyceride levels (ρ = 0.139, p = 0.017). Conversely, serum albumin levels showed a weak negative correlation with MRSA carriage (ρ = –0.114, p = 0.049). No significant correlations were observed with age, DM duration, renal function markers, lipid subfractions (except TG) or medication use.

Table 3. Correlation between MRSA nasal carriage and clinical, anthropometric and metabolic variables

	Spearman's rho (ρ)	p
Age (years)	0.028	0.628
Weight (kg)	0.163	0.005*
Body mass index (kg/m²)	0.219	<0.001*
Waist circumference (cm)	0.195	0.001*
FBG (mg/dl)	0.125	0.030*
HbA1c (%)	0.140	0.015*
TG (mg/dl)	0.139	0.017*
Albumin (g/L)	-0.114	0.049*

Spearman's rho (ρ) correlation coefficients are shown. FBG: fasting blood glucose; HbA1c: hemoglobin A1C; TG: triglyceride. p values < 0.05 were considered statistically significant.

DISCUSSION

Several factors contribute to the increased likelihood of *S. aureus* colonization among individuals with DM. The disease suppresses the immune system at both cellular and humoral levels. Hyperglycemia attenuates inflammatory responses to microbial infections by disrupting the expression of inflammatory genes in immune cells (12,13). Chemotaxis, phagocytosis and microbicidal activities of polymorphonuclear leukocytes are impaired (14). Microvascular damage caused by hyperglycemia disrupts epithelial integrity in the nasal mucosa, facilitating the attachment of biofilm-forming *S. aureus* to the mucosa via adhesion molecules (15). In a mouse study, MRSA infection in diabetic mice was found to be more severe than in non-diabetic mice, due to increased endothelial damage, coagulation and inflammation (16).

Insulin resistance prevents the aerobic use of intracellular glucose in leukocytes. The oxidative capacity of lymphocytes is impaired by low intracellular glucose levels in both prediabetic and diabetic patients (10). Accordingly, these findings suggest that glucose metabolism disorders at prediabetic levels are also susceptible to *S. aureus* and MRSA colonization and infection due to impaired immune function. In a cohort study evaluating the relationship between infection-related mortality and fasting blood glucose, HbA1c and insulin levels, 666,888 Korean patients were followed for 15.8 years. The survey indicated that both diabetics and non-diabetics with insulin resistance had a significantly higher risk of infection-related mortality (17). These data suggest that MRSA-associated infections are as severe in the early stages of DM and even in preDM as in overt DM.

Our findings are consistent with the broader literature indicating higher MRSA colonization among patients with diabetes compared with non-diabetic individuals. A meta-analysis reported a modestly increased MRSA colonization rate in diabetic patients and an overall MRSA prevalence around 9% in that population (18), supporting the notion that diabetes is associated with an elevated colonization burden at the population level. However, reported prevalence varies substantially across studies, likely reflecting differences in clinical setting (community vs hospital-associated), intensity of healthcare contact, recent antibiotic exposure, and regional epidemiology. Several studies have documented particularly high MRSA carriage among patients with diabetic foot ulcers, emphasizing a clinically vulnerable subgroup in which chronic wounds, frequent medical visits, and repeated antibiotic courses may increase the opportunity for MRSA acquisition and persistence (19). Similarly, in a Palestinian cohort of diabetic patients scheduled for surgery, MRSA nasal carriage was 9.7%, exceeding rates reported in some other settings; contextual factors such as crowding, healthcare access, and local infection-control practices may contribute to geographic variability (20,21). In the present study, MRSA point prevalence was significantly higher in type 2 DM, whereas the preDM group showed an intermediate prevalence that was not statistically significant compared with controls. This gradient is biologically plausible and may indicate that dysglycemia relates to colonization risk in a severity-dependent manner, but it should be interpreted cautiously given the cross-sectional design and single-time sampling; confirmation in larger cohorts and with repeated swabbing would strengthen inference.

Curiously, prediabetic patients demonstrated metabolic alterations such as higher LDL-C, total cholesterol, uric acid and lymphocyte counts compared to both diabetic and control groups. These results suggest that metabolic and inflammatory alterations may emerge early in the course of glucose dysregulation, even before the onset of overt DM. Also, there has recently been an emphasis on the concept that preDM is not a metabolically “silent” state but rather a condition characterized by systemic disturbances that may facilitate bacterial colonization (22).

The correlation analysis provides further insights into potential mechanisms. MRSA carriage was positively correlated with obesity-related measures (body weight, BMI and waist circumference), hyperglycemia (fasting blood glucose and HbA1c) and hypertriglyceridemia. Supporting our findings, a clinical study evaluating nasal swabs from 2,169 women and 1,709 men aged 30 or older, found that obesity was a risk factor for MRSA carriage in both sexes, independent of DM. In women, each 2.5 kg/m² increase in BMI was associated with a 7% higher rate of *S. aureus* nasal colonization (23). Another study found that high HbA1c levels were associated with MRSA carriage among patients with diabetic foot ulcers; specifically, patients with high HbA1c had a higher MRSA carriage rate. High HbA1c levels may adversely affect immune function, thereby increasing the risk of MRSA carriage (24). In line with this observation, metabolic dysregulation -particularly obesity-related parameters, hyperglycemia, and hypertriglyceridemia- has been associated with an elevated risk of MRSA nasal carriage.

Serum albumin was inversely correlated with MRSA carriage. In a study of 303 surgical patients, hypoalbuminemia was more strongly correlated with *S. aureus* carriage than high HbA1C and smoking (25). A meta-analysis of dialysis patients revealed that MRSA infection was linked to low albumin levels (26). The negative correlation with albumin likely reflects the influence of poor nutritional or inflammatory status on susceptibility to colonization.

In a study aimed at determining the rates and risk factors for nasal carriage of *S. aureus* among diabetic and non-diabetic patients, three groups were formed. The first consisted of 68 diabetic patients on insulin therapy; the second, 80 diabetic patients on oral antidiabetic drugs; and the third, 150 non-diabetic patients. *S. aureus* nasal carriage rates were 35.3%, 13.8% and 10.7%, respectively. Regarding these rates, no significant difference was found between diabetics and non-diabetics using oral antidiabetic drugs (OADs). In contrast, a significant difference was found between non-diabetic and insulin-using diabetic participants. The study found that insulin use, hospitalization within the last six months, diabetes for more than six years, a fasting glucose level greater than 111 mg/dL and antibiotic use within the previous six months, were risk factors for *S. aureus* nasal carriage (27). We also evaluated the relationship between DM duration, metformin and other oral antidiabetic drug use and insulin use and MRSA carriage, but we did not find a statistically significant correlation.

While increased *S. aureus* carriage has been reported in diabetes, the relationship between prediabetes and MRSA colonization remains less clear. Our study specifically

evaluated MRSA nasal point prevalence across the spectrum of glycemic dysregulation. Nasal MRSA carriage is a known risk factor for subsequent MRSA infection, and decolonization strategies (e.g., topical mupirocin with chlorhexidine bathing) can reduce the risk of infection in selected high-risk settings (28,29). In this cohort, type 2 DM was associated with significantly higher MRSA carriage, whereas the preDM group did not differ significantly from controls, supporting a targeted, risk-based approach to screening and prevention rather than universal surveillance in prediabetes.

Limitations: First, the cross-sectional design precludes causal inference regarding the association between glycemic status and MRSA carriage. Second, because colonization phenotypes (persistent vs intermittent carriage) require repeated sampling over time, our single nasal swab per participant reflects point prevalence and does not allow classification of persistent or intermittent carriage. Third, the study was single-center, which may limit generalizability to other settings. Fourth, only anterior nasal swabs were obtained; MRSA colonization at additional sites (e.g., throat or skin) may have been missed, potentially underestimating overall carriage. Finally, the significant age difference between groups is a potential confounder; given the limited number of MRSA-positive cases, we did not perform multivariable modeling to avoid overfitting, and residual confounding cannot be excluded.

CONCLUSION

Type 2 DM was associated with increased MRSA nasal carriage compared with controls. Although the preDM group showed an intermediate carriage rate, the difference versus controls was not statistically significant. MRSA carriage correlated positively with obesity-related measures, hyperglycemia and hypertriglyceridemia, and inversely with serum albumin, suggesting that metabolic and nutritional/inflammatory status may influence colonization risk. These findings support selective, risk-based screening and prevention strategies and highlight the need for larger prospective studies with repeated sampling to clarify the clinical relevance of MRSA carriage in prediabetes.

DECLARATIONS

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