

Clinical and Polysomnographic Phenotypes of Obstructive Sleep Apnea in Non-Obese Versus Obese Patients

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ABSTRACT

Objective: This study aimed to compare clinical and polysomnographic features between obese and non-obese obstructive sleep apnea (OSA) patients.

Material and Methods: This retrospective cohort study included: adult patients having undergone type I polysomnography; from 2017 until 2022. Participants were categorized as: obese (body mass index [BMI] ≥ 30 kg/m²) and non-obese (BMI < 30 kg/m²). Clinical features and sleep study parameters were compared. Correlation analyses were performed to assess any relationships between apnea hypopnea index (AHI) and body weight, BMI and neck circumference (NC). The diagnostic performance of STOP-Bang in obese individuals was additionally investigated.

Results: There were 573 patients; of which 359 (62.7%) were obese and 214 (37.3%) were non-obese. The prevalence of OSA and OSAS were 94.9% and 38.9%, respectively, increasing with BMI. Obese patients were significantly younger and had higher BMI, neck and waist circumferences, STOP-Bang and Epworth sleepiness scale, and more frequent symptoms; such as fatigue and choking. Polysomnographic findings revealed that obese patients had higher AHI, oxygen desaturation index (ODI) and T90, lower minimum SpO₂, and shorter total sleep time. NC showed a modest correlation with AHI ($r=0.442$, $p\text{-value}<0.001$). In obese patients, a STOP-Bang score ≥ 3 yielded 80.3% sensitivity and 62.5% specificity for detecting OSA.

Conclusions: Obesity markedly increases OSA severity, correlating strongly with higher AHI and more pronounced clinical and polysomnographic abnormalities. NC is the predictor of OSA severity, and a STOP-Bang of ≥ 3 has an acceptable performance in identifying OSA in obese patients.

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INTRODUCTION

Obesity is now recognized as one of the most prevalent nutritional disorders globally and represents an increasingly major public health issue. The World Health Organization (WHO) has reported that the worldwide prevalence of obesity, defined as a body mass index (BMI) of ≥ 30 kg/m², was approximately 15.8% in 2022: 13.6% among males and 17.9% among females. In Thailand, the prevalence of obesity has markedly risen from 11.9% in 2016 to 15.4% in 2022: affecting 12.2% of males and 18.4% of females. This reflects a very high obesity burden relative to other ASEAN countries¹. Obesity is a chronic condition linked to numerous comorbidities including; cardiovascular, respiratory, gastrointestinal, musculoskeletal diseases, cancer and mental health disorders. Additionally, obesity significantly impacts both sleep quality and duration. Obstructive sleep apnea (OSA), the most prevalent type of sleep-disordered breathing, affects approximately 9–38% of the adult population^{2,3}. OSA is characterized by repeated episodes of upper airway collapse during sleep, causing intermittent hypoxia, hypercapnia, fragmented sleep and consequent daytime dysfunction. Additionally, it increases the risks of cardiovascular morbidity and mortality^{4–6}. Obesity is a well-established, strong risk factor for OSA due to increased adiposity around the upper airway and thoracic region. This results in airway narrowing and reduced lung volume; thereby, exacerbating OSA severity. Previous studies reported OSA prevalence exceeding 50–98% among obese adults, with severity strongly correlating with increased body weight^{7,8}. Specifically, each 10% increment in weight has been associated with a 32% rise in the apnea-hypopnea index (AHI)⁹. Furthermore, obese patients exhibit prolonged durations of oxygen saturation below 90% and lower nadir oxygen saturation compared to non-obese individuals¹⁰. Research focusing on the clinical and polysomnographic phenotypes of OSA

in obese populations within Thailand still remains limited. Understanding phenotype differences between obese and non-obese Thai patients with OSA has important clinical implications for early screening, perioperative risk assessment, and individualized management strategies; particularly in resource-limited healthcare settings. Hence, this present study aimed to determine the prevalence of OSA among obese patients, and to compare the clinical and polysomnographic characteristics between obese and non-obese patients.

MATERIAL AND METHODS

This retrospective cohort study was conducted at the Sleep Center of Songklanagarind Hospital, Faculty of Medicine, Prince of Songkla University. The study population included: obese patients having undergone polysomnography; from 2017 until 2022. Eligible participants were adults aged 18 years or older having undergone Type I polysomnography. Patients were excluded if their polysomnographic data were considered unreliable: defined as having a total sleep time of less than 2 hours. This study was approved by the Ethics Committee of the (EC 66-283-46-1).

BMI was determined by dividing body weight in kilograms by squared height in meters (kg/m²). Patients were classified into non-obese and obese groups if their BMI was < 30 and ≥ 30 . Neck circumference (NC) was measured at the upper border of the thyroid cartilage, waist circumference (WC) was measured at the level of the umbilicus, while hip circumference (HC) was recorded at the point of maximal buttock width.

All patients underwent a type I in-lab polysomnography, which included an Electroencephalography (EEG), Electromyography (EMG), Electrooculogram (EOG), Electrocardiogram (ECG), oronasal airflow, respiratory movement and oxygen saturation. Apnea was characterized

as a total absence of airflow lasting ≥ 10 seconds; whereas, hypopnea was defined as a $\geq 30\%$ decline in oronasal airflow associated with either a $\geq 3\%$ drop in oxygen saturation, or an arousal. AHI defined as the number of apneas and hypopneas occurring per hour of sleep, was applied to classify OSA severity into normal (< 5 events/hour), mild ($5-15$), moderate ($15-30$) and severe (> 30)¹¹⁻¹³.

Statistical analysis

Baseline characteristics were expressed as numbers with percentages, means with standard deviations, or medians with interquartile ranges. Group comparisons were carried out using chi-square and t-tests. Spearman's correlation analysis was used to identify the correlations between parameters, because several variables showed non-normal distributions. Statistical significance was considered with a p -value < 0.05 . All statistical analysis was performed using R software version 4.5.1. Cases with missing data for specific variables were excluded from the corresponding analyses using a complete-case approach.

RESULTS

A total of 573 patients were enrolled. The prevalence of OSA, using an AHI of ≥ 5 events/hour, was demonstrated as being 94.9% (544/573), of which were: mild OSA (19.5%), moderate OSA (26.7%) and severe OSA (48.7%). The prevalence of obstructive sleep apnea syndrome (OSAS), defined by AHI ≥ 5 events/hour along with excessive daytime sleepiness, which was 38.9%

(223/573). The prevalence of OSA and OSAS increased with BMI (Table 1).

Comparison of clinical characteristics between obese and non-obese patients

Three hundred and fifty-nine patients (62.7%) were obese, whilst 214 patients (37.3%) were nonobese, with a mean BMI of 39.2 ± 8.0 and 27.5 ± 1.6 kg/m², respectively. Obese patients were significantly younger than non-obese patients, with mean ages of 41.7 ± 14.2 and 52.6 ± 15.1 years; respectively (p -value < 0.001). Obese patients had significantly greater weight, BMI, NC, WC, and HC (all p -value < 0.001). They also had significantly higher STOP-Bang and ESS scores (p -value = 0.001 and p -value = 0.009, respectively). Regarding OSA symptoms, daytime fatigue (12.5% vs. 4.7%, p -value = 0.003) and choking during sleep (27.3% vs. 18.7%, p -value = 0.026) were significantly more frequent among obese patients. Other symptoms and comorbidities (e.g., hypertension, diabetes mellitus, heart disease, stroke) were not significantly different between the groups (Table 2).

Comparison of polysomnographic findings between obese and non-obese patients

Obese patients exhibited significantly more severe sleep-disordered breathing. They had shorter total sleep time (4.4 vs. 5.2 hours; p -value < 0.001), a higher hypopnea index (35.0 vs. 19.3; p -value < 0.001), and significantly higher AHI (47.0 vs. 29.0; p -value < 0.001)

Table 1 Prevalence of OSA and OSAS by BMI category

BMI category	Prevalence (%)	
	OSA (definition of AHI ≥ 5)	OSAS (definition of AHI ≥ 5 with excessive daytime sleepiness)
BMI ≥ 25	95.2	39.2
BMI ≥ 30	97.8	43.7
BMI ≥ 40	97.7	44.6
BMI ≥ 50	100.0	47.4

BMI=body mass index; OSA=obstructive sleep apnea; OSAS=obstructive sleep apnea syndrome; AHI=apnea hypopnea index

Table 2 Clinical characteristics between non-obese and obese patients

Factors	BMI <30 (n 214)	BMI ≥30 (n 359)	p-value
Gender, n (%)			<0.001
Male	123 (57.5)	130 (36.2)	
Female	91 (42.5)	229 (63.8)	
Age (years) ^a	52.6 (15.1)	41.7 (14.2)	<0.001
Weight (kg) ^a	74.1 (8.8)	104.3 (25.9)	<0.001
Height (m) ^a	1.6 (0.1)	1.6 (0.1)	0.055
BMI (kg/m ²) ^a	27.5 (1.6)	39.2 (8.0)	<0.001
Neck circumference (cm) ^a	37.8 (3.2)	40.9 (4.1)	<0.001
Waist circumference (cm) ^a	95.7 (7.3)	115.8 (15.6)	<0.001
Hip circumference (cm) ^a	103.2 (5.6)	123.9 (15.8)	<0.001
STOP- Bang score ^b	3 (2,4)	4 (3,5)	0.001
ESS ^b	7.5 (5,10)	8 (5,12)	0.009
Symptoms, n (%)			
Snoring	209 (97.7)	357 (99.4)	0.109
Daytime sleepiness	160 (74.8)	275 (76.6)	0.692
Nonrestorative sleep	107 (50.0)	192 (53.5)	0.471
Neurological impairment	79 (36.9)	144 (40.1)	0.503
Awakening during sleep	65 (30.4)	124 (34.5)	0.350
Choking	40 (18.7)	98 (27.3)	0.026
Morning headache	48 (22.4)	92 (25.6)	0.447
Daytime fatigue	10 (4.7)	45 (12.5)	0.003
Comorbidities, n (%)			
HT	94 (43.9)	166 (46.2)	0.652
DM	34 (15.9)	79 (22.0)	0.095
Heart disease	18 (8.4)	35 (9.8)	0.692
Asthma	24 (11.2)	27 (7.5)	0.177
Stroke	6 (2.8)	7 (1.9)	0.567

^amean (standard deviation); ^bmedian (interquartile range); BMI=body mass index; ESS=Epworth sleepiness scale; HT=hypertension; DM=diabetes mellitus

than non-obese patients. Mild OSA was higher in non-obese patients (36.0% vs 17.8%); whereas, severe OSA was higher in obese patients (56.0 % vs 36.4%). The AHI during REM and NREM sleep as well as in supine and non-supine positions were all significantly higher in the obese group (p-value<0.001). Minimum oxygen saturation was significantly lower (78.5% vs. 85.0%; p-value<0.001); additionally, the oxygen desaturation index (ODI) as well as percentage of sleep time with oxygen saturation <90% (T90) were significantly greater in obese patients (p-value<0.001). However, there were no significant differences in the hypopnea-apnea ratio (HAR) pattern, indicating similar respiratory event profiles in both groups. Sleep architecture

was broadly similar; although, obese patients showed a slightly lower percentage of REM sleep (13.0% vs. 14.4%; p-value=0.046) (Table 3).

Correlations between AHI and body weight, BMI and NC

Body weight and BMI were positively correlated with AHI (rho=0.3935, p-value<0.001 and rho=0.327, p-value<0.001, respectively). (Figure 1, 2) Neck circumference demonstrated a moderate positive correlation with AHI (rho=0.442), indicating a clinically meaningful association between upper airway adiposity and OSA severity (p-value<0.001) (Figure 3).

Table 3 Polysomnographic characteristics between non-obese and obese patients

Parameters	BMI <30 (n 214)	BMI ≥30 (n 359)	p-value
Total sleep time (hours) ^a	5.2 (1.9)	4.4 (2.1)	<0.001
Sleep efficiency (%) ^a	83.1 (55.7)	78.6 (14.3)	0.145
Stage N1 (%) ^a	25.9 (15.6)	27.9 (17.7)	0.168
Stage N2 (%) ^a	48.2 (12.0)	46.5 (13.0)	0.124
Stage N3 (%) ^a	11.7 (9.0)	13.3 (10.8)	0.067
Stage R (%) ^a	14.4 (7.0)	13.0 (9.3)	0.046
Apnea index (events/hour) ^a	9.7 (18.3)	12.5 (22.3)	0.120
Hypopnea index (events/hour) ^a	19.3 (14.5)	35.0 (27.5)	<0.001
AHI (events/hour) ^a	29.0 (23.2)	47.0 (35.9)	<0.001
HAR pattern, n (%)			0.476
Ratio >1	187 (87.4)	322 (89.7)	
Ratio ≤1	27 (12.6)	37 (10.3)	
REM AHI (events/hour) ^a	31.4 (22.4)	53.2 (30.6)	<0.001
NREM AHI (events/hour) ^a	28.1 (24.7)	45.5 (37.9)	<0.001
Supine AHI (events/hour) ^a	36.8 (29.2)	53.9 (38.3)	<0.001
Non-supine AHI (events/hour) ^a	17.7 (19.7)	36.6 (37.3)	<0.001
Minimum SpO2 (%) ^a	85.0 (7.6)	78.5 (11.7)	<0.001
T90 (hours) ^a	4.1 (10.4)	9.4 (17.1)	<0.001
ODI (events/hour) ^a	13.3 (19.9)	31.2 (52.9)	<0.001
OSA severity, n (%)			<0.001
Mild OSA (AHI 5–<15)	77 (36.0)	64 (17.8)	
Moderate OSA (AHI 15–30)	59 (27.6)	94 (26.2)	
Severe OSA (AHI >30)	78 (36.4)	201 (56.0)	

^amean (standard deviation); AHI=apnea–hypopnea index; HAR=hypopnea–apnea ratio; T90=time spent with an oxygen saturation below 90%; ODI=oxygen desaturation index; REM=rapid eye movement; NREM=non rapid eye movement; OSA=obstructive sleep apnea

Diagnostic accuracy of STOP-Bang in identifying OSA in obese patients

In patients with a BMI ≥30, a STOP-Bang score cutoff of ≥3 yielded a sensitivity of 80.3% and specificity of 62.5% in identifying OSA (AHI ≥5). The area under the receiver operating characteristic curve (AUC) was 0.797 (95% CI: 0.658–0.936).

DISCUSSION

This study highlights the differences in clinical presentations and polysomnographic characteristics between obese and non-obese individuals having obstructive sleep apnea (OSA). Consistent with global trends, this study observed a high prevalence of OSA (94.9%) among its study participants; significantly increasing with higher BMI categories and reaching 100% among those with a BMI ≥50 kg/m². This finding aligns with previous studies, which

reported OSA prevalence rates between 50–98% in obese populations; confirming obesity as a major risk factor for OSA development and severity progression^{7,8}.

In our study, obese patients presented at significantly younger ages, contrasting with traditional assumptions that OSA primarily affects older populations. This may reflect recent epidemiological shifts towards increasing obesity rates among younger populations, emphasizing the evolving demographic of OSA patients. Furthermore, obese patients exhibited notably higher anthropometric indices; including neck circumference (NC), waist circumference and BMI, all of which positively correlated with OSA severity. In this study, NC demonstrated the strongest correlation with AHI, suggesting its utility as a key clinical indicator for predicting OSA severity. This finding was consistent with prior literature identifying NC as a stronger predictor of OSA severity compared to other measures^{14–17}.

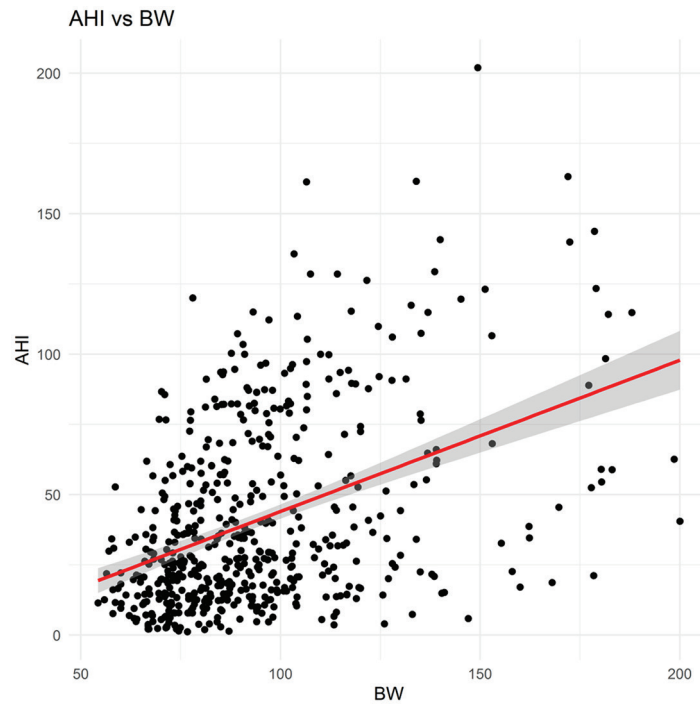


Figure 1 Box plot correlation between the apnea hypopnea index and body weight

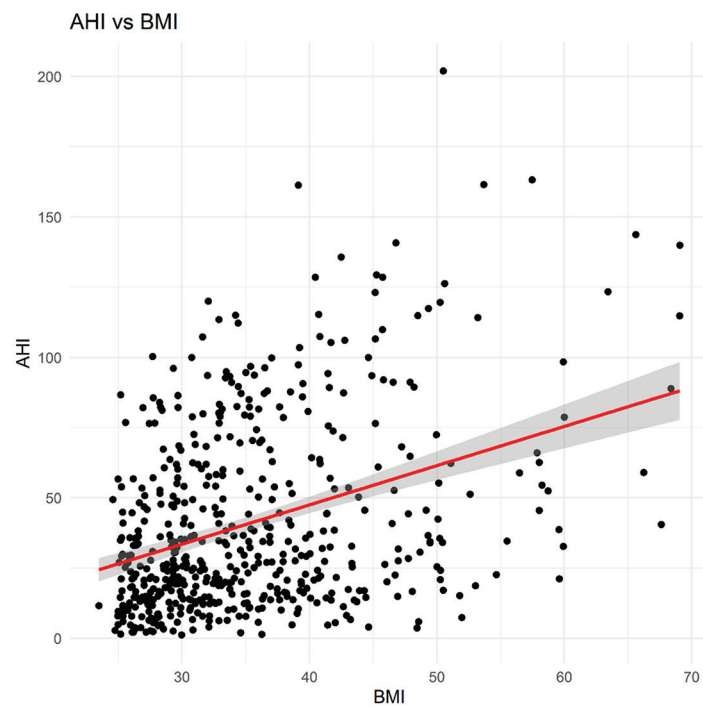


Figure 2 Box plot correlation between the apnea hypopnea index and body mass index

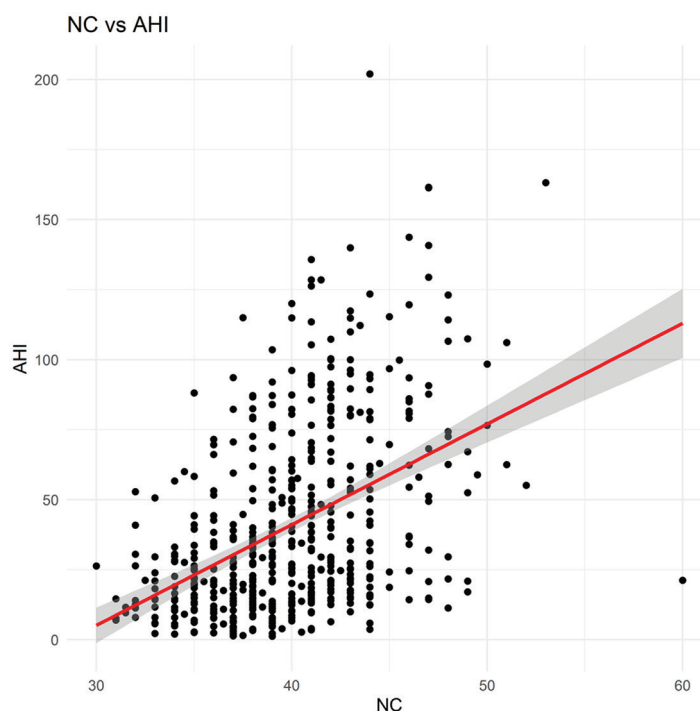


Figure 3 Box plot correlation between the apnea hypopnea index and neck circumference

Clinically, our results indicated that fatigue and choking during sleep were significantly more prevalent in obese individuals. This is consistent with the symptomatology typically reported in severe OSA phenotypes. Previous studies support these clinical differences, underscoring that obese patients often report more pronounced nocturnal symptoms. This may be possibly due to greater upper airway collapsibility and airway narrowing driven by adiposity around the pharyngeal region^{18,19}. Although, other comorbidities; such as hypertension, diabetes mellitus and cardiovascular disease, showed no significant differences between the groups in this study, evidence from larger population studies suggests that these conditions are closely associated with OSA and obesity^{20,21}. The absence of significant differences in our sample might reflect sample size limitations or high risk populations requiring sleep testing.

The polysomnographic data indicated that obese patients experienced significantly greater, severe sleep-

disordered breathing. This was characterized by higher AHI, increased oxygen desaturation and longer durations of hypoxemia. Our findings are consistent with a previous Thai study²³, which also demonstrated significantly higher AHI, ODI, and nocturnal hypoxemia in obese compared with non-obese OSA patients. These similarities confirm that obesity-related OSA phenotypes in Thailand resemble patterns observed in other Asian populations^{18,19,23}. These findings also align with several studies demonstrating that obesity exacerbates respiratory disturbances during sleep; predominantly through increased airway resistance, reduced pulmonary volumes and promoted upper airway collapsibility^{18,19,22-24}. Interestingly, the hypopnea-apnea ratio (HAR) pattern was similar between obese and non-obese groups, which was obstructive hypopnea predominance. This observation warrants further investigation, by potentially exploring mechanisms; including anatomical impairment, low arousal threshold, poor muscle responsiveness and impaired respiratory control, in obese versus non-obese individuals²⁵.

Sleep architecture differences were subtle, with obese patients exhibiting marginally less REM sleep percentage compared to their non-obese counterparts. Reduced REM sleep may reflect sleep fragmentation and increased respiratory instability typical in obese patients with severe OSA. A previous study has similarly documented mild reductions in REM sleep associated with obesity-related respiratory disturbances²⁶. Nonetheless, the clinical implications of these minor changes remain uncertain; highlighting the need for additional research to explore the broader significance of altered sleep architecture in obesity-driven OSA.

Our study further evaluated the diagnostic performance of the STOP-Bang questionnaire, confirming its moderate sensitivity (80.3%) and specificity (62.5%) among obese individuals. This aligns with previous validation studies of STOP-Bang in obese populations, suggesting its clinical utility as an accessible screening tool for identifying OSA in high-risk populations^{15,27,28}. However, its moderate specificity underscores the necessity of confirmatory polysomnography in suspected cases. Collectively, our results support the use of neck circumference in combination with STOP-Bang screening as simple, low-cost tools to prioritize high-risk obese patients for early polysomnography and perioperative risk assessment.

Limitations and strengths

This study has several limitations. Its retrospective, single-center design may introduce selection bias and limit the generalizability of the findings. In addition, the absence of multivariable regression analyses adjusting for important confounders such as age and gender precludes definitive conclusions regarding independent associations, and residual confounding cannot be entirely excluded. Nevertheless, the relatively large sample size and comprehensive polysomnographic assessment provide valuable insights into the relationship between obesity and OSA within the Thai population, helping to address an important gap in regional evidence.

Future directions

Future research should incorporate prospective cohort studies, with multivariable modeling to determine independent predictors of OSA severity and outcomes. Exploration of mechanistic differences between obese and non-obese OSA phenotypes using ventilatory control and upper airway imaging may further refine precision-based management.

CONCLUSION

OSA prevalence increases progressively with increasing BMI, with obese individuals exhibiting markedly more severe sleep-disordered breathing. Neck circumference is the predictor of OSA severity, and a STOP-Bang of ≥ 3 has an acceptable performance in screening OSA in obese patients.

Author contributions

Conceptualization, KT; data collection, KW, KP; formal analysis, KW, KP and KT; writing, and editing of manuscript, KT. All authors have read and agreed to the published version of the manuscript.

Ethical approval

The study was approved by the Institutional Review Board (IRB) of Faculty of Medicine, Prince of Songkla University (EC 66-283-46-1).

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CONFLICT OF INTEREST

There are no potential conflicts of interest to declare.

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