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Original Article

Physiology Section

Effect of Exercise on ECG Components and their Correlation with Body Mass Index among Young Asymptomatic Obese and Non-obese Individuals: A Cross-sectional Study

SUJOY MUKHERJEE[1], GOUTAM BANERJEE[2], SANHITA MUKHERJEE[3]

ABSTRACT

Introduction: The worldwide higher prevalence of obesity is currently one of the most important reasons for the occurrence of several health disorders, along with an increased risk of mortality and morbidity. However, the magnitude of the effect of sub-maximal aerobic exercise on cardiac autonomic and electrical function among Indian obese individuals has yet to be established.

Aim: To evaluate the effect of exercise on ECG components and their correlation with Body Mass Index (BMI) among young asymptomatic obese and non-obese individuals, in order to assess the risk for developing Cardiovascular Diseases (CVD) in the future.

Materials and Methods: An observational cross-sectional study was conducted at the Autonomic Research Laboratory of the Physiology Department at RG Kar Medical College and Hospital, Kolkata, between January 2017 to December 2017. Two hundred healthy medicos were divided into two groups: Obese (BMI ≥25 kg/m²; n=88) and non-obese (BMI ≥18.5 to ≤23 kg/m²; n=112). Resting Blood Pressure (BP) was manually recorded, followed by the recording of long lead-II ECG in the supine posture both at rest and ten minutes after submaximal (50% VO_2max) aerobic exercise on a treadmill using the modified Bruce protocol and following the Astrand-Rhyming nomogram. The HR, QT interval, and RR interval were recorded from the lead-II ECG. The measured QT interval was corrected using Bazett's formula (QTC=QT/√RR). The results were analysed using both unpaired and paired t-tests and the Pearson correlation coefficient in Graphpad-Quickcalc software.

Results: Significant prolongation of the QTc interval both at rest (p-value: 0.019) and after exercise (p-value: 0.044), along with delayed Heart Rate Recovery (HRR) (p-value: 0.001), were observed among the obese compared to the non-obese population. Additionally, a significantly positive linear correlation was observed between BMI and ECG components at rest (BMI-RHR: r:+0.293; p: 0.005 and BMI-QTc Interval: r:+0.226; p-value: 0.034) and during the recovery state (BMI-HRR: r:+0.42; p-value: 0.00004 and BMI-QTc Interval: r:+0.365; p-value: 0.0004), only among the obese.

Conclusion: At rest, obese individuals had an elevated QTc, which could be due to alterations in cardiac autonomic function, along with a significant positive correlation between BMI and RHR, indicating lower aerobic fitness in this population. Prolongation of the QTc interval, delayed decrease in HR, and a significant positive correlation with BMI after exercise in obese individuals indicated myocardial repolarisation instability, autonomic dysfunction, and an increased risk of out-of-hospital sudden cardiac events in these individuals.

Keywords: Obese, QTc interval, Submaximal exercise

INTRODUCTION

In the post-Coronavirus Disease (COVID) era, obesity is becoming another pandemic worldwide, and the issue of obesity need to be focused on in current times across the world [1]. Increased BMI, an important parameter for measuring Generalised Obesity (GO), is markedly correlated with several health diseases as well as Sudden Cardiac Death (SCD) [2]. The association of these clinical conditions might be due to cardiac autonomic dysfunction [3]. However, the mechanisms behind this autonomic impairment due to weight gain have not been clearly understood. Additionally, reduced Resting Heart Rate (RHR) denotes strong aerobic fitness with better myocardial function, whereas a higher RHR could be due to reduced left ventricular musculature with attenuated resting systolic volume, resulting in a lower aerobic fitness level [4]. Furthermore, a low decline or impairment in HRR after exercise could be due to attenuated parasympathetic reactivation [5]. Therefore, these individuals would be markedly susceptible to mortality and morbidity [6].

Moreover, the risk of CVDs, including SCD, becomes more prevalent when obesity is correlated with a prolonged corrected-QT (QTc) Interval [7]. Thus, highly informative non-invasive ECG parameters such as HR and QTc might be used to identify autonomic impairment among obese individuals, if any, and to predict susceptibility to an increased risk of out-of-hospital sudden cardiac events during the recovery period after exercise. To the best of the authors' knowledge, there is limited information available regarding the association between BMI and ECG parameters among young Indian asymptomatic obese and non-obese adults, particularly during the recovery phase after aerobic exercise. This present study would probably be the first one reported from the eastern part of this country in this regard. Moreover, there is also a paucity of data available regarding potential characteristic alterations in both electrophysiological milieu and Heart Rate (HR), particularly during the recovery state, among apparently healthy obese subjects.

Therefore, the present study aimed to evaluate the effect of exercise on ECG components and their correlation with BMI among young asymptomatic obese and non-obese individuals to assess the risk for developing CVDs in the future. The primary objective was to compare the changes of ECG parameters like HR and QTc among obese and non-obese subjects, both at rest and during the recovery state after a single spell of submaximal aerobic exercise. The secondary objective was to find the correlation between BMI and these two ECG variables, both at rest and during the recovery phase, in this study.

MATERIALS AND METHODS

An observational and cross-sectional study was conducted at the Autonomic Function Research Laboratory of the Physiology Department at RG Kar Medical College and Hospital, Kolkata, between January 2017 to December 2017, after obtaining proper ethical permission from the Institutional Ethics Committee, RG Kar Medical College and Hospital, Kolkata, West Bengal, India, for this research work. A total of 200 young, healthy, normotensive MBBS students, including both genders (M:F=1:1 ratio), aged 18-25 years, with and without a family history of hypertension, were selected.

Sample size calculation: According to the ICMR-INDIAB pilot study report, the maximum upper limit of the prevalence of GO in this country was 31.3% [8]. Keeping this finding in mind, the authors used the following simple formula to calculate the adequate sample size for this study: $n=Z^2\ p(1-p)/d^2$ {where n is the sample size, Z is the statistic corresponding to the Confidence Level (CL), p is the expected prevalence, and d is the allowable error or precision value expressed as a proportion of P, ranging from 5% to 20%} [9].

As this present study was a preliminary investigation to be reported from the eastern part of this country, the authors used the maximum range of precision within the span of error tolerance (at 95% CL) and estimated a corrected final sample size (n) of 200 for this study to maintain homogeneity in the study population. To maintain the ethnicity of the study subjects, it was ensured that all participants resided in the eastern part of India and were chosen by a simple random sampling technique from the medics of this college, following certain inclusion and exclusion criteria as mentioned below. Written consent was obtained from every individual prior to the study.

Inclusion criteria: The total of 200 subjects were included and divided into two groups based on BMI, following the latest revised guidelines for the Asian-Indian population to categorise obesity [10,11]: a) Obese (n=88); and b) non-obese (n=112). The study subjects were included based on the following criteria: Firstly, young, healthy, asymptomatic, normotensive individuals (BP ≤140/90 mmHg as per Joint National Committee (JNC) 8 guidelines) [12], who were either obese (BMI ≥25 kg/m^2) or non-obese with a normal BMI (BMI: ≥18.5 to ≤23 kg/m^2), MBBS students of different semesters (age group: 18-25 years, as there is the lowest occurrence of any kind of cardiac abnormality in this age group particularly [13]) in this institution. Secondly, female subjects who had no menstrual abnormalities for the last three consecutive months before this study were included. Thirdly, all individuals were non-smokers, non-alcoholic, and non-athletic.

Exclusion criteria: Subjects who were smokers, addicts with regular drug intake causing autonomic dysfunction, those suffering from acute illness within the last three weeks before the study, and students with a personal history of hypertension, diabetes, CVDs, endocrine disorders, and autoimmune diseases were excluded from this research work. Trained endurance athletes were also not included in this study setting. To maintain proper inter-group homogeneity among the study subjects, overweight individuals (BMI: >23 to 24.99 kg/m^2), underweight individuals (BMI <18.5 kg/m^2), and females with abnormal menstrual cycles were also not allowed to participate in this research work.

Procedure

Firstly, all the subjects were thoroughly explained in detail about the study procedure. After obtaining anthropometric measurements such as height, weight, and BMI, the subjects were asked to take a 30-minute compulsory rest in a supine posture while being awake in the quiet room of the experimental laboratory. Manual Blood Pressure measurements were then taken three times using an aneroid sphygmomanometer at 2-minute intervals, and the average value was recorded as the baseline Blood Pressure (BP) reading for each subject and documented in the case sheet.

Thereafter, a long lead-II ECG was recorded using a Physiograph Polyrite-D instrument with bio-amplifiers (four channels, Model No.: RMS/ENISO 13485:2003, Recorders and Medicare Systems, RMS, Chandigarh) and accessories for five minutes in a supine posture.

Next, all the subjects performed a single short spell of submaximal (50% of VO_2max) aerobic exercise on a treadmill machine according to the modified Black-Bruce protocol [14]. The exercise was continued until males reached a Pulse Rate (PR) of 128 beats/min and females reached a PR of 138 beats/min, as shown on the pulse oximeter. Achieving these PRs for both genders allowed for obtaining a submaximal workload (50% of VO_2max) according to the Astrand and Rhyming nomogram [15,16]. To achieve homogeneous groups of power values, the exercise was carried out for an additional five minutes with the same workload.

After exercise cessation, the individual was allowed to rest in a supine posture and the study procedure was completed by recording ECG again during the last five minutes of the 15-minute recovery period. The QT interval and RR interval were automatically obtained from the built-in software of the lead II ECG in the Polyrite-D instrument. The measured QT interval was then corrected for HR by the ECG software machine automatically using the formula of Luo S et al., ($QTc=QT/\sqrt{RR}$) [17].

STATISTICAL ANALYSIS

After completing the data acquisition, paired t-tests, unpaired t-tests, and Pearson correlation coefficient were used for statistical analysis using Microsoft Excel 2007 and GraphPad QuickCalc software (California, USA). A p-value of less than 0.05 was considered statistically significant for this study. Comparison was done in the anthropometric parameters, baseline haemodynamic status, and ECG components (HR and QTc only) and for intra-group analysis, RHR and HRR were considered. Correlation was established between BMI and ECG parameters.

RESULTS

The present study showed a significant difference in weight and BMI between age- and height-matched obese and non-obese populations. Furthermore, obese individuals had significantly higher mean values of resting SBP, DBP, and increased corrected QT interval (QTc) compared to non-obese subjects. However, RHR was not significantly higher in the obese group compared to the other population [Table/Fig-1]. Moreover, obese subjects had a higher mean value of HRR and a prolonged QTc interval compared to non-obese individuals [Table/Fig-2].

Variables	Obese (n=88)	Non-obese (n=112)	t value	p-value
Age (years) (Mean±SD)	18.954±1.212	19.125±0.659	1.268	0.206
Weight (kg) (Mean±SD)	72.522±9.756	54.16±7.965	14.651	**<0.0001***
Height (mt) (Mean±SD)	1.641±0.067	1.623±0.091	1.559	0.12
BMI (kg/mt^2) (Mean±SD)	26.874±2.817	20.349±1.745	20.092	**<0.0001***
Resting Heart Rate [RHR] (beats/min) (Mean±SD)	83.568±13.87	80.607±10.318	1.73	0.085
Basal SBP (mm of Hg) (Mean±SD)	123.636±10.927	113.5±16.202	5.036	**<0.0001***
Basal DBP (mm of Hg) (Mean±SD)	79.454±7.579	72.892±9.664	5.229	**<0.0001***
QTc Interval (Mean ±SD) (Rest)	288.936±92.161	259.041±86.842	2.352	**0.019***

[Table/Fig-1]: Comparison of anthropometric parameters, baseline haemodynamic status and ECG components between the study groups.
*Unpaired t-test was used for analysis of the date in this table

Variables	Obese (n=88)	Non-obese (n=112)	p-value
Heart Rate Recovery [HRR] (beats/min) (Mean±SD)	86.82±13.56	81.27±9.98	**0.001***
Recovery QTc Interval (Mean±SD)	291.58±92.668	263.648±99.824	**0.044***

[Table/Fig-2]: Comparison of ECG components at postexercise recovery state between the study groups.
*Unpaired t-test was used for analysis of the date in this table

Further analysis showed a delayed recovery of HR among obese individuals after exercise, whereas no significant difference in HR was observed during that period among non-obese subjects [Table/Fig-3]. In addition, the present study observed a significant moderately positive linear correlation between the obesity index and ECG components (HR and QTc) both at rest and during the recovery phase after exercise among obese individuals [Table/Fig-4-8].

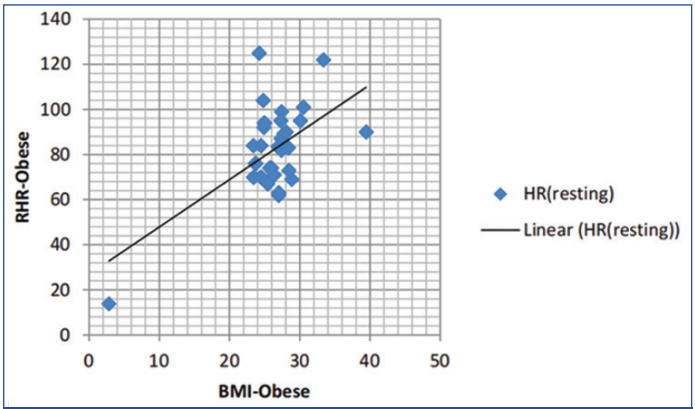
Lastly, no significant linear correlation was found between the obesity index and ECG components (HR and QTc) both at rest and during the recovery phase after exercise among non-obese subjects [Table/Fig-9].

Study group	Resting Heart Rate (RHR) (beats/min) (Mean±SD)	Heart Rate Recovery (HRR) (beats/min) (Mean±SD)	p-value
Obese (n=88)	83.568±13.87	86.82±13.56	**0.0001***
Non-obese (n=112)	80.607±10.318	81.27±9.98	0.076

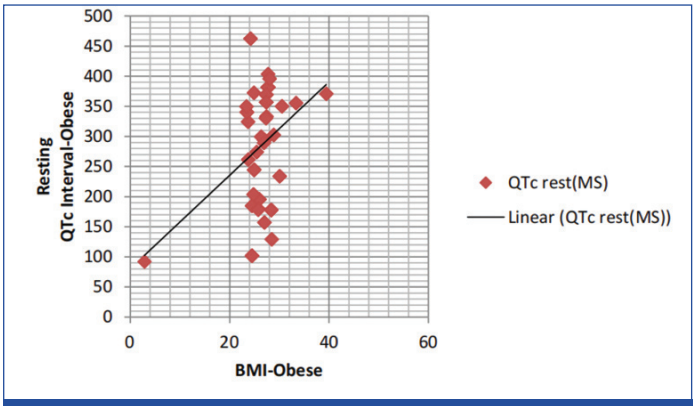
[Table/Fig-3]: Comparison of heart rate between rest and recovery phase among study groups.
*Paired t-test was used for analysis of the date in this table

Variables	r-value	p-value
BMI-RHR	+0.293	**0.005***
BMI-QTc Interval (Rest)	+0.226	**0.034***
BMI-HRR	+0.42	**0.00004***
BMI-QTc Interval (Recovery)	+0.365	**0.0004***

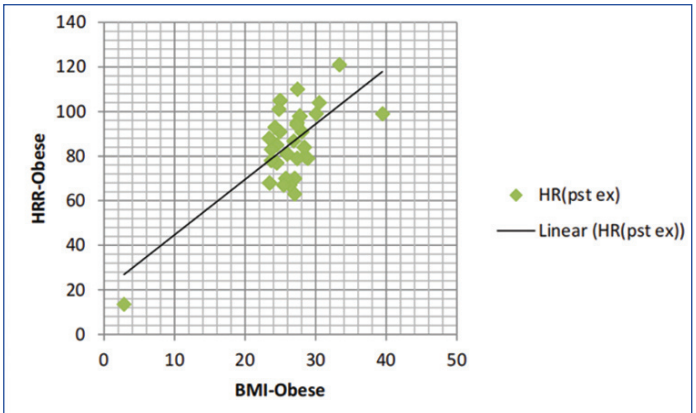
[Table/Fig-4]: Correlation between BMI and ECG components in obese (n=88) group.
*Pearson's correlation coefficient test was applied in this table



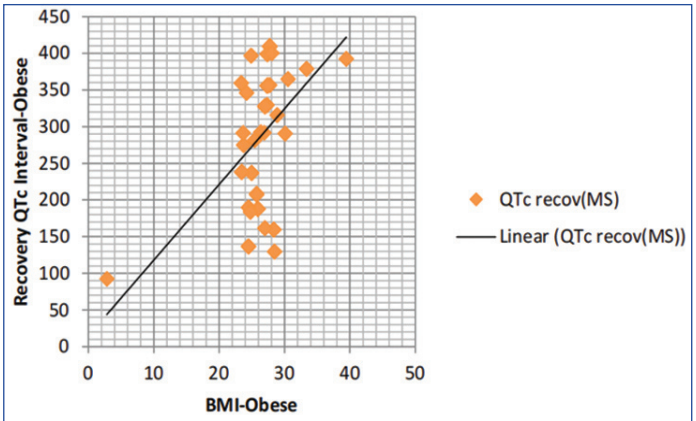
[Table/Fig-5]: Positive linear correlation between BMI and RHR in obese subjects (in Scattered Diagram).



[Table/Fig-6]: Positive linear correlation between BMI and resting QTc interval in obese subjects (in Scattered Diagram).



[Table/Fig-7]: Positive linear correlation between BMI and HRR in obese subjects (in Scattered Diagram).



[Table/Fig-8]: Positive linear correlation between BMI and recovery QTc interval in obese subjects (in Scattered diagram).

Study variables	r-value	p-value
BMI-RHR	-0.004	0.966
BMI-QTc Interval (Rest)	+0.204	0.056
BMI-HRR	+0.017	0.858
BMI-QTc Interval (Recovery)	+0.102	0.284

[Table/Fig-9]: Correlation between "BMI" And ECG components in non-obese (n=112) groups.

DISCUSSION

This study documented a significant prolongation of the QTc interval both at rest and after exercise, along with delayed HRR among obese individuals compared to the non-obese population. Additionally, a significantly linear positive association was observed between BMI and ECG components such as HR and QTc interval, both at rest and during the recovery state, only among the obese subjects.

Baseline haemodynamic variables showed significantly higher values in obese individuals, which was in line with the findings of Kumar T et al., [18]. This may possibly be due to sympathetic hyperactivity and insulin resistance related to excess body fat, as well as the secretion of angiotensinogen, which provokes the formation of angiotensin II and further increases sympathetic outflow [19,20].

Furthermore, a direct relationship was observed between BMI and HR at rest among obese individuals only, which was similar to the findings of a study conducted in the southern region of this country [21]. This strongly suggests lower aerobic fitness in obese subjects [4]. On the other hand, previous cross-sectional and prospective studies have shown a direct or indirect relation between obesity and impaired HRR after exercise, which may be attributed to cardiovagal dysregulation, both in the presence and absence of CVD risk factors [22,23]. These findings also support the suggestion of vagal impairment in the obese population in the present study. The delayed rate of decrease in HR in these individuals may indicate poor aerobic capacity [24]. In a comparative study, researchers also

demonstrated that obesity itself affects HRR, with obese subjects consistently experiencing delayed HRR regardless of their fitness level [25]. It is a well-known fact that practicing aerobic exercise can help maintain sympatho-vagal balance and peak oxygen consumption [26]. Therefore, the significant difference in HR during the recovery period between the study groups may not only indicate fitness-related impairment in autonomic function among obese individuals but also suggest adrenergic overdrive, which could contribute to the development of ischemia, Acute Coronary Syndromes (ACS), fatal or non-fatal arrhythmias, or even heart failure in this population in the future. Furthermore, increased HR after exercise could lead to the development of atherosclerotic lesions in blood vessels due to mechanical exertional effects on the cardiac vasculature, resulting in increased shear stress and impaired arterial compliance [27,28].

Additionally, QTc-related changes were observed among the study groups, and a significant positive correlation was noted between QTc and BMI in obese individuals. These findings were in line with the findings of Seyfeli E et al., [29] and Ravikumar V et al., [30]. Furthermore, Nie J et al., demonstrated the prolongation of the QTc interval in obese adults during recovery from brief high-intensity intermittent exercise [31]. In contrast to the present findings, Girola A et al., previously documented no significant difference in QT interval and QT dispersion values in their comparative study among patients with uncomplicated obesity and controls [32]. However, due to the inadequacy of data regarding the correlation between QTc and BMI during the recovery period after variable intensity of aerobic exercise, it is yet to be established. Therefore, the present study's findings could provide valuable inputs for future investigators in this regard.

As the QTc interval mainly represents ventricular repolarisation in a normal heart [33], the tendency for prolongation of QTc, as well as a positive correlation with BMI among obese individuals, could possibly indicate increased repolarisation instability in the myocardium. Therefore, despite having QTc values well below the maximum upper limit, these apparently healthy normotensive young obese individuals could have an increased propensity for ventricular arrhythmia and sudden cardiac events. A possible molecular mechanism could be related to the modulation of ion channel function within the myocardium [34]. Additionally, it is known that autonomic dysfunction can affect cardiac electrophysiology by altering the duration of the QT interval [35]. Therefore, it is understandable to speculate that the risk of adverse cardiovascular events is higher due to the possibility of prolongation of the QTc interval in this present study, which could be attributed to Sympathovagal Imbalance (SVI) and attenuated parasympathetic activity during the recovery period after exercise.

At this point in the study, the authors would like to highlight the clinical implications of this present study. The most important implication is the prevention of potential disasters by assessing cardiac electrical function within physiological limits in response to a single bout of sub-maximal aerobic exercise. This would facilitate targeted prognostic ECG testing by primary care physicians among asymptomatic but potentially "at-risk" obese individuals in this post-COVID phase, thereby improving the chances of survival in this population. Further investigation is needed to examine the consequences of aerobic and anaerobic Moderate-Intensity Exercise (MIE) on QTc length and dynamics among trained and untrained obese individuals, as well as in other clinical settings such as myocardial infarction or adverse drug effects where long QT can be observed.

In the present study, the authors applied maximum allowable range of error, to estimate the sample size, hence the strength of this study was its methodological rigour in the training protocol for the groups, and the homogeneity of the study population provided greater assurance of the accuracy of the findings. Furthermore, the findings, particularly 10 minutes after sub-maximal aerobic exercise in the study population, can be considered unique, interesting, and helpful data for future researchers to explore further in similar study settings.

Limitation(s)

This section of the article mainly focuses on some major limitations that also provide guidance for further research. Firstly, due to the cross-sectional study design, it was difficult for the authors to establish causality and temporality among variables. Secondly, the authors did not classify the obesity of the study subjects as central or visceral, which would have allowed for further correlation between obesity and myocardial functionality. Moreover, the measurement of neurohumoral parameters was beyond the scope of this present study design.

As the current study population was restricted to young asymptomatic participants with and without obesity from eastern India, the generalisability of the data is limited. Results may vary in other ethnicities with larger sample sizes and different exercise loads. The BP was not measured during the recovery period.

CONCLUSION(S)

Obese individuals had elevated resting BP and QTc, which could be due to alterations in cardiac autonomic function with SVI. The positive correlation between resting BMI and HR suggests lower aerobic fitness in obese individuals. The significant positive correlation between BMI and ECG parameters, as well as the prolongation of the QTc interval and delayed rate of decrease in HR after exercise in obese individuals, indicate repolarisation inhomogeneity of the myocardium and reduced vagal influence on HR. These factors increase the risk of out-of-hospital sudden cardiac events in apparently healthy individuals without structural heart disease. Moreover, HRR can be a helpful marker for physicians to monitor increased response to steady dynamic exercise in their routine clinical practice for this population. Additionally, recording QTc before and after sub-maximal steady treadmill exercise should be considered as a non-invasive, inexpensive, and reproducible index for risk stratification in asymptomatic young obese individuals.

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PARTICULARS OF CONTRIBUTORS:
1. Assistant Professor, Department of Physiology, Rampurhat Government Medical College and Hospital, Rampurhat, West Bengal, India.
2. Professor and Head, Department of Physiology, Sarat Chandra Chattopadhyay Government Medical College and Hospital, Uluberia, West Bengal, India.
3. Professor and Head, Department of Physiology, Rampurhat Government Medical College and Hospital, Rampurhat, West Bengal, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Sujoy Mukherjee,
"Matridham"; c/o. Mrityunjoy Mukherjee, At-Kendua, P.O. + P.S.-Suri, District-Birbhum, Suri-731101, West Bengal, India.
E-mail: dr.sujoymukherjee@rediffmail.com

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