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Serum oncostatin M is a potential biomarker of disease activity and infliximab response in inflammatory bowel disease

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Abstract

Crohn's disease (CD) and ulcerative colitis (UC) are two types of inflammatory bowel diseases (IBD) diagnosed by chronic inflammation of the gastrointestinal system. Despite being the gold standard for assessing the therapeutic response to biological medicines like infliximab and disease activity in IBD patients, endoscopy's widespread use is limited by its time-consuming, expensive, and intrusive nature. This prospective case-control study was performed at Ain Shams University School of Medicine Hospital to examine the clinical utility of serum oncostatin M (OSM) as a biomarker for disease activity and response to infliximab in Egyptian IBD patients. It included 72 IBD patients (19 CD, 53 UC) and 29 controls. Patients were divided into three groups to investigate the connection between disease activity and OSM levels. To analyze the connection between OSM expression and clinical response, 36 IBD patients (22 with UC and 14 with CD) receiving infliximab maintenance were enrolled. All patients were subjected to comprehensive medical history, clinical evaluation, endoscopies, and detection of serum OSM levels. Of the 36 IBD patients, 18 patients responded to infliximab treatment, while the other 18 patients did not. The results demonstrated that, in comparison to controls, patients with IBD had higher levels of serum OSM expression. Serum OSM levels in IBD patients showed a positive association with disease activity. Individuals with moderate-to-severe UC and active CD had considerably elevated levels compared to those in remission. In conclusion, serum OSM showed as a promising biomarker for managing individuals with IBD, it was substantially expressed and positively connected with the severity of the disease. Infliximab non-response was linked to elevated OSM levels.

Keywords: Serum Oncostatin M, Infliximab Response, Inflammatory Bowel Disease.

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Introduction

Inflammatory Bowel Disease (IBD) is a persistent, episodic inflammatory condition that includes Crohn's disease (CD) and ulcerative colitis (UC). Endoscopy is the most reliable method for diagnosing and classifying IBD. Nevertheless, it is time-consuming, expensive,

uncomfortable, and intrusive for patients, rendering it impractical to monitor disease activity regularly.[1]

Infliximab is considered the first-line biological agent for IBD patients, however, it was found to increase the likelihood of severe infections because of its influence on immunosuppression,[2] anti- tumor necrosis factor (anti-TNF) therapies may cause a primary nonresponse in as many as 40% of IBD patients.[3] There is currently no perfect non-invasive biomarker to assess infliximab effectiveness in patients with IBD.

Oncostatin M (OSM), an interleukin 6 (IL-6) cytokine, was identified as a target for IBD diagnosis and treatment.[4] OSM overexpression was demonstrated to impair tight junction expression and induce epithelial-mesenchymal transition, hence jeopardizing the integrity of intestinal barrier function.[5]

In several studies,[6-9] when compared to the mucosa of apparently healthy people, OSM was the most transcriptionally elevated cytokine in CD patients' inflammatory intestinal mucosa and one of the most up-regulated in UC patients.

Moreover, the researchers provided clear evidence that raises important issues about down-regulated or over-expressed OSM and over-expressed oncostatin M receptor (OSMR) at the intestinal mucosa, indicating that the OSM-OSMR has a specific role for IBD. OSM and OSMR expression was markedly increased before treatment and was strongly correlated with the level of mucosal irritation.[6]

This study aimed to examine the clinical application of serum OSM as a potential disease activity biomarker and treatment response to infliximab in Egyptian patients with IBD.

Materials and Methods

This observational case-control study included IBD patients, with definite diagnosis, recruited from the Ain Shams University School of Medicine Hospital, during the period from March 2023 to March 2024. They were diagnosed based on imaging, endoscopy, histology, laboratory testing, and clinical complaints.

The study included 101 Egyptian subjects (≥ 18 years of age), who were divided into 3 groups: Group I; included 37 IBD patients in activity, group II; included 35 IBD patients in remission. Group III included 29 apparently healthy individuals as a control group with cross-matched age and sex.

Serum samples were collected from patients either before or during infliximab administration (the times of infliximab injection ≥ 4) in Ain Shams University Hospital.

IBD patients were further categorized according to their disease to patients with UC (n=53) and patients with CD (n=19). Comparisons between groups were based on laboratory results, serum OSM levels, endoscopic scores, and treatment regimens, all documented according to clinical status and investigations.

The association between OSM expression and clinical response was investigated in 36 IBD patients treated with infliximab (22 UC and 14 CD).

Excluded patients included those who were pregnant, had unfinished clinical records, isolated upper GIT lesions, had a history of cancer or tumors, had autoimmune illnesses, or declined to participate in the study.

All individuals underwent a complete history, clinical examination, and routine laboratory tests; these data were obtained from the hospital records. All patients underwent endoscopy, and histopathology was performed on all endoscopic biopsies.

The partial Mayo score was utilized to evaluate UC patients, and the Harvey-Bradshaw Index (HBI) was used to assess patients with CD. Accordingly, clinical activity was estimated by these activity scores as follows: moderate-to-severe disease (HBI > 7, partial Mayo score (pMS) > 4), mild disease (5 ≤ HBI ≤ 7, 2 ≤ pMS ≤ 4), and remission (HBI < 5, pMS < 2).

The simple endoscopic score (SES) for Crohn's disease (SES-CD) was employed to evaluate endoscopic activity in individuals with the condition, considering intestinal ulcers, narrowings, diseased surfaces, and ulcerated

surfaces. The Mayo endoscopic sub-score (MES) was employed to evaluate endoscopic activity in patients with UC based on ulcers, bleeding, and vascular patterns. SES-CD < 2 or MES ≤ 1 were used to describe mucosal healing.

Treatment regimens for all patients were recorded along with their clinical status; serum OSM level was measured for all participants utilizing commercial enzyme-linked immunosorbent assay (ELISA) kits (Cat. No. E1663Hu, produced in 16/2/2023, from Bioassay Technology Laboratory, China).

Serum OSM quantification

Plasma was prepared by centrifuging of blood samples at 800–1200 xg for 20 minutes at room temperature and then the plasma was kept frozen at -80°C until used for OSM analysis.

Before usage, the unutilized strips were stored at temperatures ranging from 2 to 8°C. The ELISA experiment was carried out at room temperature. A volume of 50 µl of standard solution was added to the designated standard wells. No additional antibody was introduced into these wells as the standard solution already contained biotinylated antibody. For the test samples, 40 µl of sample solution was dispensed into the sample wells, followed by 10 µl of anti-OSM antibody, and subsequently 50 µl of streptavidin–HRP conjugate. Plates were sealed and incubated for 60 min at 37 °C.

Following incubation, the plates were washed five times with wash buffer, ensuring each wash included a 30–60 s soak. Excess liquid was removed, and the plates were blotted with absorbent material. Subsequently, 50 µl of substrate solution A and 50 µl of substrate solution B were added to each well, and plates were incubated for 10 min at 37 °C in the dark. The enzymatic reaction was terminated by adding 50 µl of stop solution to each well, resulting in a color change from blue to yellow.

Optical density (OD) was measured at 450 nm using a microplate reader (Thermo Scientific, USA) within 10 min of adding the stop solution.

Statistical Analysis

Data were collected, revised, coded and entered to the Statistical Package for Social Science (IBM SPSS) (IBM Corp. Released 2020. IBM SPSS Statistics for Windows, Version 27.0. Armonk, NY: IBM Corp). The quantitative data were presented as mean, standard deviations and ranges when parametric and median, inter-quartile range (IQR) when data found non-parametric. Also qualitative variables were presented as number and percentages. The comparison between groups regarding qualitative data was done by using Chi-square test and/or Fisher exact test when the expected count in any cell found less than 5. The comparison between two independent groups with quantitative data and parametric distribution was done by using Independent t-test while with non parametric distribution were done by using Mann-Whitney test. The comparison between more than two groups regarding quantitative data and parametric distribution was done by using One Way ANOVA test followed by post hoc analysis using LSD test while with non parametric distribution was done by using Kruskall-Wallis test followed by post hoc analysis using Mann-Whitney test. Spearman correlation coefficients were used to assess the correlation between two quantitative parameters in the same group. Receiver operating characteristic curve (ROC) was used to assess the best cut off point with its sensitivity, specificity, positive predictive value, negative predictive value and area under curve (AUC) of the studied marker. The confidence interval was set to 95% and the margin of error accepted was set to 5%. So, the *p*-value was considered significant *p*-value < 0.05.

Results

Regarding demographic characteristics, the study included 33 (32.7%) males and 68 females (67.3%) with ages ranged from 18 to 64 (34.04 ± 10.0 years). Patients and controls were matched for their age and sex; however, patients with CD showed statistically significant male predominance (n=11, 57.9%) in comparison to UC patients (n=16, 30.2%) and the control group (n=6, 20.7%) (*p*=0.023).

The participants included 37 IBD patients in activity, 35 in remission groups, and 29 in the control group. IBD patients included UC (n=53) and CD (n=19). IBD patients on infliximab maintenance were 36 (22 with UC and 14 with CD). Of these, 18 individuals responded to infliximab, and the other 18 individuals did not.

CD patients were classified according to SES-CD to remission [9 (47.4%)], mild [7 (36.8%)], and moderate [3 (15.8%)], while the UC group was classified according to the Mayo score 0 [8 (15.1%)], score 1 [13 (24.5%)], score 2 [16 (30.2%)] and score 3 [16 (30.2%)].

The percentage of active patients in the UC group was 31 (58.5%), statistically substantially greater than in the CD group [6 (31.6%)], (p=0.044).

Serum oncostatin M (OSM) in the study groups

IBD patients showed a statistically significant rise in the median OSM level. (CD patients and UC patients) [2617.5 (1086.5 - 3440) ng/l] than controls [255 (218 - 639) ng/l] (p <0.001), and there was no substantial difference between the Crohn's and ulcerative groups. In addition, patients in the disease activity group had considerably higher median OSM than those in remission group, as did patients in remission group compared to the control group (p<0.001).

Regarding disease activity

According to our findings, the degree of IBD activity and OSM levels were significantly correlated, as determined by clinical and endoscopic severity scores. The level of OSM was significantly higher in mild and moderate SES-CD than in patients in remission, (p =0.003).

There was a statistically significant rise in the median OSM in patients in activity [3337 (2689 - 3780)] ng/l than patients in remission [1073 (519 - 2120)] ng/l and an increase in the median OSM in patients in remission than in the control group [255 (218 - 639)] ng/l (p <0.001). Also, there was a statistically significant rise in the median OSM level in the Crohn's group [1600 (873 - 3142)] ng/l and ulcerative group [2667 (1200 - 3521)] ng/l than in the control group [255 (218 - 639)] ng/l (p <0.001) (Table 1).

Table 1. Comparison of Oncostatin M (OSM) levels in the groups under the study.

Category	Disease Activity Status			Patient Groups		
	Remission	Activity	Control	Crohn's group	Ulcerative group	Control
	No.= 35	No.= 37	No.= 29	No.= 19	No.= 53	No.= 29
OSM Median (IQR)	1073 (519 - 2120)	3337 (2689 - 3780)	255 (218 - 639)	1600 (873 - 3142)	2667 (1200 - 3521)	255 (218 – 639)
OSM Range(ng/l)	230 – 4638	1742 – 4800	179 – 802	230 – 4638	257 – 4800	179 – 802
p-value	<0.0001			<0.0001		

IQR = Interquartile Range; ng/l = nanograms per liter; No. = Number of patients. $p \leq 0.05$ is significant.

In addition, we found that severity assessments based on clinical severity scores were strongly correlated with OSM levels. A statistically significant positive correlation was found between OSM, PMS score (p < 0.0001), and HBI score (p= 0.027). This suggested that the severity of the disease relates to an increase in OSM.

OSM levels and hemoglobin levels showed a statistically significant negative link, while OSM levels and total leukocyte count (TLC), platelets, erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP) showed a statistically significant positive correlation. However, there was no significant correlation with the other studied parameters (Table 2).

Collectively, the median OSM level was significantly higher in the mild [3104 (1600 – 4185)] ng/l and moderate [3324 (2089 – 3337)] ng/l SES- CD than in patients in remission [873 (257 – 1100)] ng/l (p =0.003). Also, the median OSM level was significantly higher in patients with Mayo score 3 [3600 (3181 – 4334.5)] ng/l than with Mayo score 2 [2694 (2476 – 3440)] ng/l and also higher in patients with Mayo score 2 than in patients with Mayo score 1 [904 (680 – 2635)] ng/l and in patients with Mayo score 0 [805 (435 – 1660)] ng/l (p <0.001). The median OSM was also considerably higher in patients who did not respond to infliximab. [2924 (2587 – 3780)] ng/l than in patients responded to infliximab [897 (375 – 1200)] ng/l (p <0.001) (Table 3).

Table 2. Correlation between Oncostatin M (OSM) level and the other studied parameters.

	OSM	
	r	*p*-value
Age	0.046	NS
TLC	0.407	<0.0001
HB	-0.523	<0.0001
Plt	0.313	0.007
AST	0.004	NS
ALT	0.000	NS
Bil	-0.027	NS
Alb	-0.177	NS
Creat	0.075	NS
ESR	0.545	<0.0001
CRP	0.411	<0.0001
HBI score	0.505	0.027
PMS score	0.796	<0.0001

TLC=Total leukocyte count; HB = Hemoglobin; Plt = Platelets; AST = Aspartate aminotransferase; ALT = Alanine aminotransferase; Bil = Bilirubin; Alb = Albumin; Creat = Creatinine; ESR = Erythrocyte sedimentation rate; CRP = C-reactive protein; HBI score = Harvey–Bradshaw Index score; PMS score = Partial Mayo Score. Notes: r = Correlation coefficient. p > 0.05 is not significant (NS).

Table 3. Relation between Oncostatin M (OSM) and the other studied parameters.

Other parameters		OSM		*p*-value
		Median (IQR)	Range	
Gender	Female	2699 (960 – 3564)	257 – 4800	•NS
	Male	2468 (1100 – 3324)	230 – 4800	
SES CD	Remission	873 (257 – 1100) [b]	230 – 2740	≠0.003
	Mild	3104 (1600 – 4185) [a]	1500 – 4638	
	Moderate	3324 (2089 – 3337) [a]	2089 – 3337	
Mayo score	Mayo 0	805 (435 – 1660) [c]	257 – 2587	≠<0.0001
	Mayo 1	904 (680 – 2635) [c]	359 – 4297	
	Mayo 2	2694 (2476 – 3440) [b]	1203 – 4800	
	Mayo 3	3600 (3181 – 4334.5) [a]	2387 – 4800	
Response to Infliximab N=36	No	2924 (2587 – 3780)	1742 – 4638	•<0.0001
	Yes	897 (375 – 1200)	230 – 1600	

IQR = Interquartile range; SES CD = Simple Endoscopic Score for Crohn's Disease. Superscripts with different letters (a, b, c) indicate significant pairwise differences between groups. p > 0.05 is not significant (NS).
•: Mann-Whitney test; ≠: Kruskal-Wallis test followed by post hoc analysis using Mann-Whitney test.

According to the receiver operating characteristic (ROC) curve (Figure 1) analysis, the optimal cut-off point between the patient and control groups for OSM levels was > 802, with a sensitivity of 83.33%, specificity of 100.0%, at an area under the curve of 0.929.

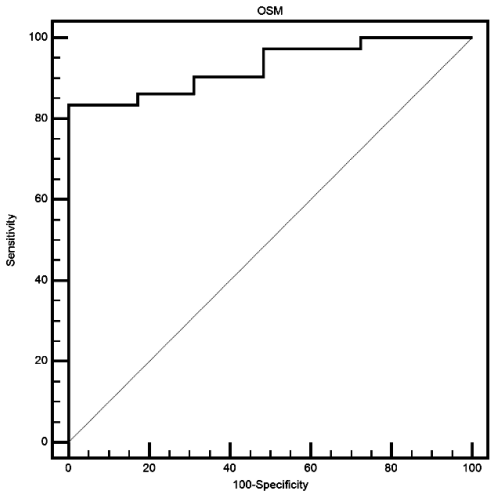

Figure 1. Receiver operating characteristic (ROC) curve for Oncostatin M (OSM) level to differentiate between the patients' group and the control group.

ROC curve demonstrated that the optimal cut-off point between patients in remission and those in activity in terms of OSM level was found to be > 2120 ng/l, with a sensitivity of 94.59%, specificity of 77.14%, at an area under curve (AUC) of 0.898 (Figure 2).

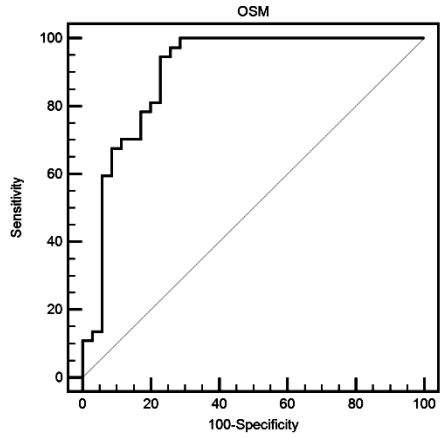

Figure 2. Receiver operating characteristic curve (ROC) for Oncostatin M (OSM) level to differentiate between remission and activity groups.

UC and CD groups did not vary significantly in their reaction to infliximab (p = 0.171); serum OSM expression was greater in clinical non-responders than in responders (p <0.001).

The ROC curve identifies the optimal cut-off point for distinguishing clinical non-responders from clinical responders to infliximab in terms of OSM level > 1600 ng/l with a sensitivity of 100%, specificity of 100.0%, at an AUC of 1 (Figure 3).

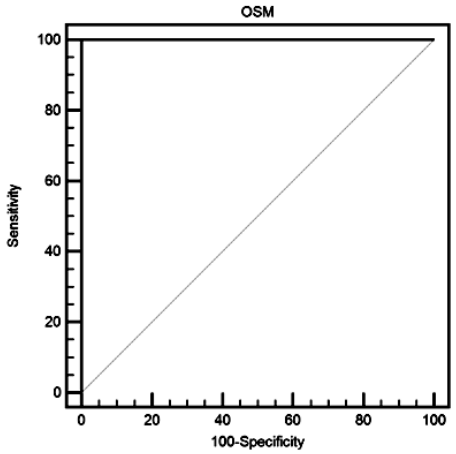

Figure 3. Receiver operating characteristic curve (ROC) curve analysis for Oncostatin M (OSM) level to differentiate between responders and non-responders on infliximab.

Discussion

This study investigated the connection between serum OSM levels and endoscopic findings and clinical activity in IBD. As a supplementary objective, serum OSM was also used to assess infliximab effectiveness.

A typical biomarker usually used in place of endoscopic activity is fecal calprotectin. However, it is challenging to collect stool samples, and there is a lot of variation in sample testing at different times throughout the day. Fecal calprotectin is present in several intestinal inflammatory diseases, such as celiac disease and infectious enteritis, although it is not a diagnostic marker for IBD. For accurate disease surveillance, a novel biomarker is thus required.[7] OSM is viewed as a multifunctional cytokine produced from various sources of activated T cells, monocytes, macrophages, and neutrophils. It is thought to be pro-

inflammatory because it helps stimulate the recruitment of leukocytes.[8] Furthermore, it may stimulate SERPIN family members, increase cell proliferation, and decrease apoptosis to support signal transducer and activator of transcription 3 (STAT3), dependent intestinal epithelial regeneration. The expression in intestinal tissues aligned with our results that blood OSM levels were elevated in IBD patients and had a significant correlation with both clinical and endoscopic activity. Serum OSM has shown exceptional efficacy in assessing mucosal healing and quantifying intestinal inflammation. Consequently, serum OSM may be a valuable biomarker for endoscopic activity in IBD.[6]

In our investigation, serum (OSM) levels differed significantly between patients and controls. Although no significant difference was observed between CD and UC in patients with IBD, which exhibited significantly higher serum OSM levels compared to control individuals ($p < 0.001$). These findings highlight the potential diagnostic value of OSM in active IBD and contribute meaningfully to the existing literature.

Various studies supported this outcome; they examined tissue samples from intestinal lesions of patients with active IBD and discovered high levels of OSM and its receptor.[6-9] The ROC analysis based on mucosal oncostatin revealed a noteworthy distinction between IBD patients and controls.[9] Further evidence is that serum OSM levels in IBD patients and healthy control people differed significantly.[10] According to our research, OSM levels did not significantly differ between patients with UC and CD. Nonetheless, the UC patients had considerably greater fecal OSM levels than those with CD ($p = 0.132$).[10]

Our investigation used endoscopic severity scores to determine the substantial correlation between OSM levels and severity scores. IBD patients lacking mucosal repair had greater levels of OSM. As a result, OSM can serve as a marker for endoscopic activity. Patients with proctitis, the left-distal group, and those with pancolitis had significantly different ulcerative colitis endoscopic index of severity (UCEIS) defined symptom severity ($p < 0.027$). The degree of symptoms as reported by UCEIS and the OSM level differed significantly. This finding is supported by another study that investigated the link between OSM, UC, and disease severity.[11] The blood samples from UC patients in activity exhibited higher levels of OSM gene expression than those in remission. These findings are consistent with those reported by Zhou et al., 2019.[12]

The intestinal stromal cells of people with IBD create OSM, which triggers a potent inflammatory response by generating chemokines that draw T lymphocytes and phagocytic cells. Oncostatin's genetic antagonism was shown to alleviate intestinal inflammation in mice with infliximab resistance. Such findings explain the robust relationship seen in this research between OSM levels and severity ratings derived from an endoscopic severity evaluation.[6]

Our findings were largely consistent with those of Cao et al.,[13] as serum OSM showed positive correlations with key inflammatory markers including WBC, ESR, CRP, and platelets, and a negative correlation with hemoglobin. These associations reinforce the concept that OSM reflects the underlying inflammatory burden in IBD. However, in contrast to their study, which demonstrated a significant negative relationship between OSM and serum albumin, we did not observe any meaningful correlation with albumin in our study. This discrepancy may indicate variability across populations or methodological differences, but overall, both studies support the potential role of OSM as a biomarker of disease activity.

In our study, OSM showed higher discriminative accuracy in UC than CD (AUC =0.949 vs. 0.873), while another study reported the reverse trend, with CD performing better than UC (AUC =0.943 vs. 0.824).[13] Similarly, although remission in our patients was best identified at a threshold around 802 ng/L with high specificity, the previous report described slightly different remission cut-offs (=103 pg/ml for UC and = 98.9 pg/ml for CD), each associated with good sensitivity and specificity. Taken together, these results highlight that while absolute values vary across studies, both consistently support the role of OSM as a biomarker for disease activity and remission in IBD.

In terms of using OSM levels to predict treatment response, in this study, we observed that OSM levels in infliximab responders and non-responders varied significantly. Serum OSM expression was noticeably higher in clinical non-responders than in responders. (p< 0.001). Resistance to infliximab treatment was indicated by higher OSM levels. Also, oncostatin can predict responsiveness to infliximab treatment in IBD patients.[13] Individuals with CD may exhibit clinical non-response if their baseline serum OSM levels were high according to Bertani et al., 2020.[14]

The OSM expression may be indicative of how well treatment with infliximab would perform. It further states that in a study conducted on children who had CD, a significantly strong correlation between high levels of OSM and resistance to infliximab was found according to West et al., 2021.[6] The sole trial reported that in a small number of patients with severe UC, mucosal OSM did not predict infliximab response.

In conclusion, serum OSM was shown to be a reliable biomarker for the treatment of IBD. Also, it has a high correlation with disease activity and was considerably expressed in individuals with IBD. Non-responsiveness to infliximab was correlated with increased levels of OSM.

Author Contributions

All authors contributed to the study's conception and design. Writing and preparation of original draft: TGM and HAE; Writing, review and editing: AMFM and CRL; Conceptualization: MMS and AME; Methodology: CRL, AMFM and MMS; Formal analysis and investigation: HAE and CRL; Resources: MMS and AME; Supervision: TGM, MMS and all authors commented on previous versions of the manuscript. All authors agree with the manuscript and declare that the content has not been published elsewhere.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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Ethical approval

The study protocol was reviewed and approved by the Research Ethical Committee of the Faculty of Medicine Ain Shams University (Assurance FMASU MD, No.64/2023).

Informed consent

Each subject who participated in the study provided written informed consent before being enrolled in the research study.

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