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Prevalence and Risk Factors of Hyperuricemia in Patients with Type 2 Diabetes Mellitus

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Abstract: Objective: To investigate the prevalence and risk factors of hyperuricemia in patients with type 2 diabetes mellitus (T2DM). Methods: A total of 416 hospitalized patients with type 2 diabetes mellitus were divided into two groups according to whether they were complicated with hyperuricemia (HUA), type 2 diabetes mellitus group (T2DM with HUA, n=362) and type 2 diabetes mellitus with HUA group (T2DM with HUA, n=54). General demographic information was collected, present medical history and past medical history were inquired, and clinical biochemical indicators were detected. The prevalence of hyperuricemia in T2DM patients was analyzed. Pearson was used to analyze the correlation between hyperuricemia and other indicators. Binary Logistic regression was used to analyze the influencing factors of hyperuricemia in T2DM patients. Results: HUA54 cases were detected in 416 inpatients with type 2 diabetes mellitus, and the prevalence was 13. 0%. Logistic regression analysis showed that after adjusting for confounding factors, After adjusting for confounding factors, male, age, disease duration, BMI, TG, UREA, sCr and hypertension were risk factors for hyperuricemia in T2DM patients [OR(95%CI) were 1. 746 (1. 097-2. 348), 1. 06 (1. 022-1. 102), 1. 305 (1. 001-1. 605) and 1. 934, respectively] (1. 613-2. 305), 1. 865 (1. 104-2. 419), 6. 247 (2. 016-8. 943), 3. 547 (2. 092-6. 774), 4. 961 (2. 022-9. 618)]. Conclusion: Gender, age, disease duration, BMI, TG and hypertension are associated with hyperuricemia in T2DM patients, which can increase the risk of hyperuricemia in T2DM patients.

Hyperuricemia (HUA) is a disorder of purine metabolism in the body. The prevalence of HUA is increasing year by year, and it tends to be younger. Clinically, in addition to asymptomatic hyperuricemia, hyperuricemia is often accompanied by obesity, hypertension, dyslipidemia, diabetes and other metabolic complications [1]. In addition, long-term hyperuricemia will aggravate the disorder of glucose and lipid metabolism and the chronic persistent damage of multiple organs and tissues [2]. Domestic and foreign epidemiological studies have shown that HUA accounts for a certain proportion in patients with type 2 diabetes. Fan Meijuan etc. [3] The prevalence of hyperuricemia in type 2 diabetes mellitus (T2DM) patients in Tianjin was 14. 6%. Foreign Shiferaw etc. [4] Studies have shown that the prevalence of hyperuricemia and metabolic syndrome (MS) in patients with type 2 diabetes mellitus is as high as 33. 8% and 70. 1%, respectively. Casta [5]Casta et al. also found that serum uric acid levels were higher in people at high risk for diabetes (including first-degree relatives with diabetes and/or impaired glucose tolerance regulation) and patients with diabetes than in non-diabetic people. The main cause of death or disability in patients with diabetes

is its complications. In recent years, a number of observational studies have been conducted[6-7]. The results suggest that T2DM patients with hyperuricemia may aggravate metabolic disorders and accelerate the occurrence and development of diabetes and its chronic complications. Therefore, it is of significant significance to understand the prevalence and related risk factors of hyperuricemia in patients with T2DM, and take targeted interventions to reduce the occurrence of hyperuricemia when necessary. The aim of this study was to investigate the prevalence and related risk factors of HUA in hospitalized patients with T2DM, so as to provide a theoretical basis for early prevention of HUA and reduction of its related complications.

1. Data and Methods

1.1 General Information

A total of 456 patients with T2DM hospitalized in the Department of Endocrinology of our hospital from May 2018 to February 2019 were enrolled in this study, and 416 patients with normal purine diet during hospitalization were selected as the research objects. All patients met the 1999 WHO diagnostic criteria for diabetes mellitus. Exclusion criteria: 1) type 1 diabetes mellitus, gestational diabetes mellitus and secondary diabetes mellitus. 2) Those who had taken drugs that affected uric acid metabolism or clearance, such as pyrazinamide, salicylic acid preparations, glucocorticoids, urate-lowering drugs, cyclosporine, diuretics, angiotensin receptor antagonists, fenofibrate, etc., within 2 weeks, and those who had consumed a high-purine diet within 2 weeks. 3), chronic renal insufficiency, nephrotic syndrome, neoplasms, and hematological diseases. The patients were divided into type 2 diabetes mellitus group (T2DM, n=362) and type 2 diabetes mellitus with hyperuricemia group (T2DM, n=54) according to whether they had hyperuricemia. The study was approved by the ethics committee of the hospital, and written informed consent was obtained from the participants.

1.2 Research Methods

All patients underwent detailed medical history collection, height, weight, blood pressure at admission, duration of diabetes, family history of diabetes, occupation, past medical history, smoking, drinking and other general information, and body mass index (BMI) was calculated. Fasting for 8-10h, venous blood was collected the next day. Serum uric acid (sUA), serum creatinine (sCr), UREA (UREA), fasting blood glucose (FPG), triglyceride (TG), total cholesterol (TC), high density lipoprotein cholesterol (HDL-C) and low density lipoprotein cholesterol (LDL-C) were measured by Hitachi 7600 automatic biochemical analyzer. Serum uric acid (SUA) and serum creatinine were measured by uricase-peroxidase method and sarcosine oxidase method. Glycosylated hemoglobin (HbA1C) was analyzed by high pressure liquid ion-exchange method on Bole (D-10) glycosylated hemoglobin analyzer. All data were recorded.

Diagnostic criteria: 1) Hypertension: SBP≥140mmHg and/or DBPSBP≥90mmHg according to the 1999 WHO/ISH Guidelines for the Prevention and treatment of Hypertension, and/or previous definite diagnosis of hypertension.2) Coronary heart disease: according to the American Heart Association (ACC)/American Heart Association (AIA) criteria, the presence of angina pectoris and/or myocardial infarction indicated by the typical symptoms of angina pectoris and the results of ECG or cardiac stress test, 24-hour holter electrocardiogram, or laboratory examination, coronary heart disease can be suspected. Or coronary heart disease was confirmed by previous coronary angiography.3), lipid metabolism disorders: TG > 1.71mmol/L, TC > 5.70mmol/L, LDL-C > 3.10mmol/ L, HDL-C level decreased, male < 1.10mmol/L, female <1.20mmol/L, or have received lipid-lowering therapy, meeting any of the above criteria. 4) Diabetic peripheral neuropathy (DPN) :

Endocrinology, chairman of China physicians association, and physicians in 2009 chapters of the "guidelines for diabetic peripheral neuropathy and treatment", in the case of rule out other causes, diabetic nerve dysfunction related symptoms or signs, such as the abnormal temperature sense, check dragon silk, foot hypesthesia or disappear, abnormal vibration sense, ankle reflex disappears, there are two or more than two unusual person, DPN can be diagnosed if there are two or more abnormalities. 5) Diabetic kidney disease (DKD): According to the expert consensus on the clinical diagnosis of adult diabetic nephropathy in China formulated by the endocrinology society of Chinese medical association in 2015, chronic kidney disease caused by diabetes, including glomerular filtration rate (GFR), is less than 60mL/ (min·1.73m)^2 or a urinary albumin creatinine rate (ACR) higher than 30mg/g for more than 3 months. 6), Diabetic retinopathy (DR): fundus examination was performed by ophthalmologist, and dilated fundus examination with compound tropine phthalide was performed, and microaneurysms, bleeding spots or bleeding spots, hard or soft exudation, neovascularization and fiber proliferation were diagnosed. 7) Nonalcoholic fatty liver Diseas (NAFLD) : According to the guidelines for the diagnosis and treatment of nonalcoholic fatty liver disease (NAFLD) formulated by the hepatology society of Chinese medical association in 2010, NAFLD can be diagnosed if one of the following items 1-4 and item 5 or 6 are present :(1) no history of alcohol consumption or alcohol consumption equivalent to ethanol <40g/w;(2) excluding viral hepatitis, total parenteral nutrition and other specific diseases that can lead to fatty liver disease;(3) In addition to the primary clinical manifestations, fatigue, abdominal distension, dull pain in the liver area and other symptoms can be accompanied by hepatosplenomegaly;(4) Serum aminotransferase levels were increased, especially ALT levels, often accompanied by elevated levels of γ-glutamyltransferase and TG. (5) The imaging findings of the liver were consistent with the imaging diagnostic criteria of diffuse fatty liver. The ultrasonographic features of the fatty liver were as follows :a Mild fatty liver: fine spot, enhanced near-field echo, slightly attenuated far-field echo, and clear vascular structure;B Moderate fatty liver: fine spot, enhanced anterior-field echo, significant far-field attenuation, unclear vascular structure;C Severe fatty liver: fine spot, significantly enhanced anterior-field echo, significantly attenuated distal field echo, and unrecognizable vascular structure;(6) The liver histological changes were consistent with the pathological diagnostic criteria of fatty liver disease. 8) hyperuricemia: according to the "multidisciplinary expert consensus on the diagnosis and treatment of hyperuricemia related diseases in China" formulated by the expert group on kidney diseases in 2017, fasting blood uric acid level > 420μmol/L (7g/dL).

1.3 Statistical Analysis

Spss21. 0 software was used for statistical analysis. Measurement data with normal distribution were expressed as mean ± standard deviation (x±s). Independent sample t-test was used for comparison between the two groups. Enumeration data were expressed as percentage (%) and $\chi 2$ test was used. Pearson was used to analyze the correlation between hyperuricemia and each index in T2DM patients. Binary Logistic regression analysis was used to analyze the influencing factors of hyperuricemia in T2DM patients, and P <0. 05 (two-sided) was considered statistically significant.

2. Results

2.1 Prevalence of Hyperuricemia in T2DM Patients

The prevalence of hyperuricemia in T2DM patients was 13. 0%, and the prevalence of hyperuricemia in male patients was significantly higher than that in female patients (15. 7% in male and 7. 7% in female, P < 0. 01).

2.2 Comparison of General Data and Biochemical Parameters between the Two Groups

Compared with T2DM patients alone, BMI, SBP, sUA, sCr, TG and UREA were increased in T2DM patients with HUA, and the proportion of smoking and drinking was increased in T2DM patients with HUA. At the same time, the proportion of hypertension, coronary heart disease, lipid metabolism disorder, DKD and DR in T2DM patients with HUA were increased ($P < 0.05$). HbA1C, FPG level decreased ($P < 0.05$). (See Table 1)

Table 1: Comparison of general data and biochemical parameters between the two groups [X ± S, N (%)]

group	The number of casesn	Men N (%)	Age (years old)	Smoking N (%)	Drinking N (%)	professional		T2DM A family history of N (%)
						Farmers N (%)	The farmers N (%)	
T2DM	362	231 (63.8)	57 + 11	103 (28. 5)	106 (29. 3)	78 (21. 5)	284 (78. 5)	225 (62. 2)
T2DM+HUA	54	43 (79.6)#	52 + 11	26 (48. 4)*	29 (54. 0)*	10 (18. 5)	44 (81. 5)	43 (79. 6)*
group	T2DM Duration of disease (years)	hypertension N (%)	Coronary heart disease (CHD) N (%)	Abnormal lipid metabolism N (%)	BMI (kg/m^2)	SBP (mmHg)	DBP (mmHg)	HbA_1C %
T2DM	9.7+ /-6.9	179 (49.5)	23(6.4)	215 (59.4)	25.2+/-3.8	138+20	79 + 11	9.1+/-2.2
T2DM+HUA	8.6+/- 6.7	36 (66.7)#	9 (16.7)#	43 (79.6)#	26.4+/-3.7#	141 + 18*	81 + 15	8. 1 + /- 1.7#
group	FPG mmol/L	sUA Mu mol/L	sCr Mu mol/L	UREA mmol/L	TG mmol/L	TC mmol/L	HDL-C mmol/L	LDL-C mmol/L
T2DM	8.1+/-3.4	291+67	58+19	5.5+/-2.8	2.3+/-1.6	4.5+/-1.1	1.2+/-0.3	2.3+/-0.7
T2DM+HUA	7.7+/-2.5#	491+70#	76+27#	6.6+/-3.3#	4.0+/-2.4#	4.6+/-1.1	1.1+/-0.2	2.2+/-0.9
group	DR n (%)	DKD n (%)	DPN n (%)	Fatty liver disease N (%)				
T2DM	121 (33.4)	68 (18.8)	317(87.6)	206 (56.9)				
T2DM+HUA	20 (37.0)*	12 (22.2)*	47 (87.0)	43 (79.6)#				

VsT2DM +HUA group and T2DM group, $^*P < 0. 05, ^#P < 0. 01$

2.3 Pearson Correlation Analysis was used to Analyze the Correlation between Hyperuricemia and various Indexes in T2DM Patients

Correlation analysis showed that male, age, disease duration, BMI, SBP, TG, UREA, FPG, hypertension, coronary heart disease, lipid metabolism disorder, smoking, drinking, DKD and DR were correlated with hyperuricemia in T2DM patients.

2.4 Binary Logistic Regression Analysis was used to Analyze the Influencing Factors of T2DM Patients with Hyperhua

HUA was taken as the dependent variable, and male, age, disease duration, BMI and HbA were analyzed1C, FPG, SCr, UREA, TG, hypertension, coronary heart disease and family history of diabetes were used as independent variables. Binary Logistic regression analysis showed that, after adjusting for the influence of diabetes course factors, male, age, BMI, TG, UREA, SCr and hypertension were risk factors for hyperuricemia in T2DM patients. Low FPG level and high HbA1C level were protective factors for hyperuricemia in T2DM patients. (See Table 2)

Table 2: Logistic regression analysis of the influencing factors of hyperuricemia in T2DM patients

variable	Beta.	SE	Wald $\chi 2$	P	OR (95% CI)
men	0. 863	1. 166	12. 577	0. 002	1. 746 (1. 097 to 2. 348)
age	0. 060	0. 019	9. 686	P < 0. 001	1. 06 (1. 022 ~ 1. 102)
BMI	0. 627	0. 097	38. 425	P < 0. 001	1. 934 (1. 613-2. 305)
FPG	0. 126	0. 045	9. 204	0. 003	0. 912 (0. 876-1. 014)
HbA1C	0. 381	0. 152	34. 605	P < 0. 001	0. 625 (0. 432-0. 821)
SCr	1. 105	0. 276	7. 596	0. 031	3. 547 (2. 092 to 6. 774)
UREA	0. 367	0. 252	27. 327	P < 0. 001	6. 247 (2. 016 ~ 8. 943)
TG	0. 493	0. 173	6. 853	P < 0. 001	1. 865 (1. 104 ~ 2. 419)
hypertension	1. 602	0. 458	12. 239	P < 0. 001	4. 961 (2. 022 ~ 9. 618)
Coronary heart disease (CHD)	0. 047	0. 522	0. 815	0. 367	1. 602 (0. 576-4. 458)
Family history of diabetes	0. 019	0. 033	0. 317	0. 576	0. 982 (0. 920 ~ 1. 047)

3. Discuss

The results of this study showed that 54 T2DM patients had hyperuricemia, and the prevalence was 13. 0%. Regression analysis showed that hyperuricemia in T2DM patients was positively correlated with age, BMI, TG and hypertension, suggesting that hyperuricemia may be a risk factor for T2DM patients. FPG and HbA1C were negatively correlated with hyperuricemia, which were protective factors for hyperuricemia in T2DM patients.

This observational study showed that the prevalence of hyperuricemia in T2DM patients was 13. 0%, which was higher in males than in females (15. 7%vs7. 7%, P < 0. 01), which was related to Wen Cao[8]The results of these studies were basically consistent with Wen Cao's. B. Afsar[9]. Studies have found that male sUA may be related to testosterone level. Insulin resistance can lead to decreased testosterone level, decreased renal tubular membrane uric acid secretion, decreased renal excretion of uric acid, and increased serum uric acid level. At the same time, Wen Cao[8]Studies have found that testosterone can promote the synthesis of protein and nucleic acid. Therefore, when testosterone decreases, protein synthesis decreases, endogenous purine level increases, and serum uric acid level increases. Xuan - Long YI, etc[10]. It was found that intronic variants of SLC2A9 increased the risk of hyperuricemia in Chinese men with type 2 diabetes mellitus. Further studies are needed to investigate the mechanism and correlation of SLC2A9. In addition, hyperuricemia may be related to the unhealthy lifestyle of men, such as carnivorous diet, drinking and smoking. The results of this study also showed that the proportion of smoking and drinking in T2DM patients with hyperuricemia was significantly higher than that in T2DM patients alone.

A cross-sectional study[11]. The results showed that obesity was closely related to HUA. nan[12]The BMI level of T2DM patients with hyperuricemia was significantly higher than that of T2DM patients alone (P=0. 01). Li[13] We retrospectively analyzed 300 T2DM patients and found that serum uric acid level was positively correlated with BMI (P < 0. 01). Xue[14]Xue et al. came to a similar conclusion. The present study showed that BMI was significantly associated with T2DM with hyperuricemia, and the correlation remained after adjusting for age and gender, which was consistent with the results of other studies. Obesity is associated with HUA. The possible mechanisms are as follows: obese people generally consume more high-calorie and high-purine foods and gradually gain weight;Obese people tend to have insulin resistance, which induces an increase in insulin concentration in the body, resulting in increased renal tubule reabsorption of uric acid, decreased excretion, and elevated blood uric acid.

Fan Meijuan etc. [3]It was found that the risk of hyperuricemia in T2DM patients with HbA1C > 7. 5% was lower than that in T2DM patients with HbA1C ≤7. 5% (P < 0. 01). Baby etc. [15]Studies

have shown that the FPG and HbA1C of T2DM patients with hyperuricemia are lower than those of T2DM patients with hyperuricemia. The possible mechanism may be that T2DM patients with better glycemic control have reduced urine glucose, competitive inhibition of glucose on proximal renal tubule uric acid reabsorption and decreased osmotic diuretic effect, leading to increased uric acid reabsorption and decreased excretion, and then increased sUA. The results of this study were consistent with the results of the two studies. The FPG and HbA1C of T2DM patients with hyperuricemia were lower than those of T2DM patients with hyperuricemia, suggesting that T2DM patients with hyperuricemia had better glycemic control, and the glycemic control was negatively correlated with sUA level. However, this study is a cross-sectional study, and FPG and HbA1C in T2DM patients reflect the blood glucose at admission and blood glucose control in the past 3 months, respectively, which are susceptible to various factors such as diet, hypoglycemic drugs and duration of diabetes, so the relationship between them and hyperuricemia still needs to be confirmed by further studies.

Hyperuricemia in patients with type 2 diabetes mellitus may aggravate the occurrence and development of diabetes and its complications. A retrospective study in Taiwan[16]SUA level was found to be an independent risk factor for DR and DKD in T2DM patients, and the more severe the degree of DR and DKD, the higher the level of sUA, suggesting that sUA level could reflect the severity of microvascular complications in T2DM patients. This study found that the proportion of DR and DKD in T2DM patients with HUA was higher than that in T2DM patients alone, suggesting that the incidence of diabetic microvascular complications increased with the increase of sUA level. However, the microvascular complications of diabetes were not stratified in detail in this study, which needs to be supplemented by further studies. The correlation between hyperuricemia and diabetic peripheral neuropathy remains controversial. Guo Lian[17]It was found that the incidence of DPN gradually increased with the increase of serum uric acid level. The serum uric acid level of diabetic patients with DPN was significantly higher than that of non-DPN patients, and the difference was very significant ($P < 0.01$). However, Guo Meiling et al[15]The study showed that there was no significant correlation between HUA and DPN in T2DM patients. In this study, there was no significant difference in the proportion of diabetic peripheral neuropathy between the two groups ($P > 0.05$), which may be related to the small sample size of this study. The sample size can be increased to confirm the correlation.

This study showed that the SBP level and the proportion of hypertension in T2DM patients with hyperuricemia were higher than those in T2DM patients alone, but there was no significant difference in DBP. Regression analysis suggested that hypertension was associated with a higher risk of hyperuricemia in T2DM patients. The influencing mechanism may be that hypertension can cause glomerular hypoxia, increase of blood lactic acid and competition for uric acid excretion, resulting in decreased renal uric acid excretion. At the same time, the uric acid concentration in patients with hyperuricemia exceeds the saturation state, which is prone to the formation of urate crystals. The deposition of urate crystals in the blood vessel wall of small arteries can damage the intima of arteries, cause the activation of RSA system and aggravate hypertension, causing a vicious cycle[18].

In recent years, a large number of clinical studies and epidemiological data have confirmed that HUA is closely related to the risk of coronary heart disease[19]. A retrospective study of 418 patients with type 2 diabetes mellitus[20]Hyperuricemia was found to significantly increase the risk of coronary heart disease in patients with type 2 diabetes. There have been many clinical studies in China[21-23]It has also been found that the incidence of coronary heart disease in T2DM patients with high uric acid level is higher than that in T2DM patients with normal uric acid level. Its possible mechanism is: the coronary heart disease (CHD) patients with cardiac output volume reduction, easy cause the body oxygen supply is insufficient, thus affecting the renal tubules

microcirculation disorder, cause the kidneys excrete uric acid decreased, cause the sUA levels, at the same time, the body state of HUA established lesions in patients with coronary heart disease has certain inhibitory effect on vascular collateral circulation, thus aggravating coronary heart disease (CHD) patients. But Moriazity[24]. In a follow-up study of 13504 healthy middle-aged adults, there was no significant correlation between SUA level and coronary heart disease. At the same time, Ong[25]It was also concluded that HUA could not predict the occurrence of cardiovascular disease in T2DM patients. This study showed that the proportion of coronary heart disease in T2DM patients with HUA was higher than that in T2DM patients with HUA alone. Correlation analysis showed that coronary heart disease was positively correlated with T2DM patients with HUA, but after adjusting for confounding factors, Logistic regression analysis showed that coronary heart disease was not associated with T2DM patients with HUA. Since this study was a cross-sectional study, the sequence of occurrence of HUA and coronary heart disease could not be determined. Therefore, the causal relationship between the two and related specific mechanisms need to be further confirmed by prospective studies with large samples.

HUA is closely associated with abnormal lipid metabolism, especially with TG. This study showed that the TG level and the proportion of lipid metabolism disorders in T2DM patients with hyperuricemia were significantly higher than those in T2DM patients alone. The results of regression analysis showed that TG was a risk factor for hyperuricemia in T2DM patients, and the risk of hyperuricemia increased 1. 865 times for every 0. 493mmol/L increase in TG ($P < 0. 001$, OR=1.865,95% CI= 1. 104-2.419). The results were similar to those of Fan Meijuan et al[3]. The study was consistent.

In addition, this study showed that the incidence of NAFLD was significantly higher in T2DM patients with hyperuricemia than in T2DM patients alone. This is basically consistent with the results of several domestic studies. Li kwok pan[26]Studies have found that NAFLD is correlated with sUA level in T2DM patients, and high uric acid level may be an independent risk factor for NAFLD. XinGuoQiu[27]Studies have also found that HUA is closely related to the pathogenesis of NAFLD. A study of healthy people undergoing physical examination[28]. It was found that the detection rate of NAFLD increased with the increase of sUA level in both men and women, and HUA was a high risk factor for NAFLD. The specific mechanism of the increased risk of NAFLD in patients with hyperuricemia and T2DM is still unclear. Some researchers believe that high uric acid level can cause IR and the occurrence of metabolic syndrome (MS). Excessive blood lipids accumulate fat in hepatocytes and eventually lead to NAFLD. IR can not only increase uric acid synthesis, but also inhibit uric acid excretion[29]. In this study, the diagnosis of fatty liver was made by B-ultrasound examination, not by liver biopsy, which may have some defects in the sensitivity and specificity of diagnosis.

In addition, this study is an observational study, which has some limitations. The sample size is not large enough, and the prevalence results only represent the patients with type 2 diabetes in our hospital, which is limited to Chengde region, and further increase of the sample size is needed to confirm. In addition, the data in this study are mostly cross-sectional results at the same time point, which cannot determine the occurrence and sequence of HUA and type 2 diabetes-related diseases, and the causal relationship needs to be determined by prospective studies.

In conclusion, the prevalence of hyperuricemia in T2DM patients is high, and it is correlated with male sex, age, BMI, SBP, TG, hypertension, DKD, DR and other indicators. The proportion of DR, DKD, hypertension and coronary heart disease in T2DM patients with hyperuricemia is higher than that in T2DM patients alone. Therefore, for T2DM patients, it is important to not only manage blood glucose, but also control other metabolic indicators, such as sUA, BMI, and blood lipid, which is very important for the prevention and treatment of diabetes complications and delay the progression of diabetes, which should be paid close attention to by clinicians.

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