Recurrent and new primary cancer risk in adult patients with idiopathic inflammatory myositis and a history of malignancy

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Abstract

Objective

In adult patients with idiopathic inflammatory myopathies (IIM) and a history of cancer, the risk of subsequent cancer is unclear. We describe our centre's experience with recurrent and new primary cancer development after IIM symptom onset.

Methods

A retrospective cohort study was conducted at The Johns Hopkins Myositis Center, analysing adult IIM patients enrolled from 2003 to 2024 with a documented cancer history. Patients were categorised into three groups: 1. remained cancer-free, 2. had cancer recurrence, or 3. developed a new primary cancer.

Results

Among 2,476 IIM patients, 280 (11%) had a cancer history, with 39 (14%) diagnosed within the 3 years prior to IIM symptom onset. Of these, 29 (74%) remained cancer-free after symptom onset over a median follow-up period of 5.5 (IQR 6.7) years. Six patients (16%) experienced cancer recurrence, and four (10%) developed a new primary cancer after IIM symptom onset. The median time from index cancer to recurrence was 2.4 (IQR 1.6) years, whereas the time from index cancer to new primary was 1.4 (IQR 1.0) years. Of the ten patients who developed either recurrence or a new primary cancer after IIM symptom onset, all were diagnosed within the first three years after IIM symptom onset.

Conclusion

In our tertiary referral centre, approximately one quarter of adult IIM patients who had a cancer diagnosis in the three years before IIM onset went on to develop an additional malignancy, either a recurrence or a new primary.

Key words

myositis, dermatomyositis, autoantibodies, autoimmune diseases

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Introduction

The link between specific subgroups of idiopathic inflammatory myopathy (IIM) and cancer has been well-documented, with numerous studies highlighting this association (1-3). Specifically, patients with dermatomyositis (DM) exhibit a four-fold increased risk of cancer compared to the general population, particularly within the first three years post-diagnosis (1, 2). Additionally, certain autoantibody profiles, such as anti-TIF1γ antibodies, are associated with significantly elevated cancer risk, underscoring the relevance of autoantibody screening in clinical practice (1, 3, 4). Recent international guidelines recommend stratified cancer screening approaches for IIM patients, emphasising intensified surveillance within the initial three-year period post-diagnosis, particularly in patients with high-risk autoantibodies (3). Yet, a gap remains in understanding the risk for adult IIM patients with a prior cancer history, namely, their likelihood of developing a new primary cancer (*i.e.* a second primary) or experiencing a recurrence of a previous cancer. To address this gap, we investigated the frequency of new or recurrent cancers in IIM patients over long-term follow-up at a large tertiary care referral centre.

Methods

This was a retrospective cohort study conducted at The Johns Hopkins Myositis Centre. Data were collected from patients who enrolled in our myositis centre between 2003 to 2024 and met the following criteria:

- A diagnosis of IIM defined as any of the following: (i) probable or definite IIM according to ACR/EULAR 2017 Criteria (5), (ii) immune-mediated necrotising myopathy (IMNM) according to the 2003 European Neuromuscular Centre Criteria (6), (iii) classic dermatomyositis (DM) rash (Gottron's/heliotrope) and consistent histopathology on skin biopsy, and (iv) anti-synthetase syndrome (ASyS) defined as the presence of an ASyS autoantibody with one of the following features: an inflammatory myopathy, interstitial lung disease (ILD), inflammatory arthritis, Raynaud's syndrome, fever, or mechanic's hands (7). Patients with clear diagnoses of muscular dystrophy, metabolic or mitochondrial myopathy, or inclusion body myositis were excluded, even if they technically met criteria for polymyositis (PM). Patients with myositis occurring in the setting of another rheumatic disease (*i.e.* overlap myositis) were included, and their symptom onset was defined by the onset of myositis.
- Age ≥18 at IIM symptom onset.
- Documented history of cancer.

Every patient was contacted by research staff by either phone or online survey to update their cancer status using a systematic script as previously described (1). If patients could not be reached, two sources were required to consider a patient "cancer-free". Cancer site, grade, and stage (per the American Joint Committee on Cancer, AJCC) details were obtained directly from pathology reports and oncology notes for 80% of patients with a cancer history. For the remaining 20%, this information was extracted from rheumatologist or primary care physician records. Premalignant lesions and non-melanoma skin cancers were not counted towards malignancy and were excluded from the analysis. Autoantibody profiling was performed using a combination of line blot, ELISA, and immunoprecipitation as previously described (1). Patients were divided into three groups as follows:

Group 1: patients who remained cancer free after IIM symptom onset;
Group 2: patients who had recurrence of prior cancer after IIM symptom onset;
Group 3: patients who had a new primary cancer after IIM symptom onset.

Cancer recurrence was determined based on physician documentation indicating the return of the original cancer following a period of remission. The primary analysis was performed on the group of patients who had cancer diagnosed within 3 years prior to IIM symptom onset (from -3 years to time 0, with time 0 being the date of IIM symptom onset). A secondary analysis was performed on the group of patients who had a history of cancer at any time prior to IIM symptom onset.

Cancer locations were classified according to their anatomical location in the analysis to facilitate developing cancer screening/surveillance strategies. Specifically, cancers of the orbit, larynx, nasopharynx, oropharynx, salivary gland, tongue, and tonsil were binned as 'Head and Neck'. Cancers of the oesophagus and stomach were binned as 'Upper GI'. Cancers of the gallbladder, kidney, pelvis, liver and pancreas were binned as 'Abdominal'. Cancers of the small intestine, colon, rectum and anus were binned as 'Lower GI'. All blood cancers were binned together as 'Liquid Tumour', and all other anatomical locations not mentioned were binned together as 'Miscellaneous'.

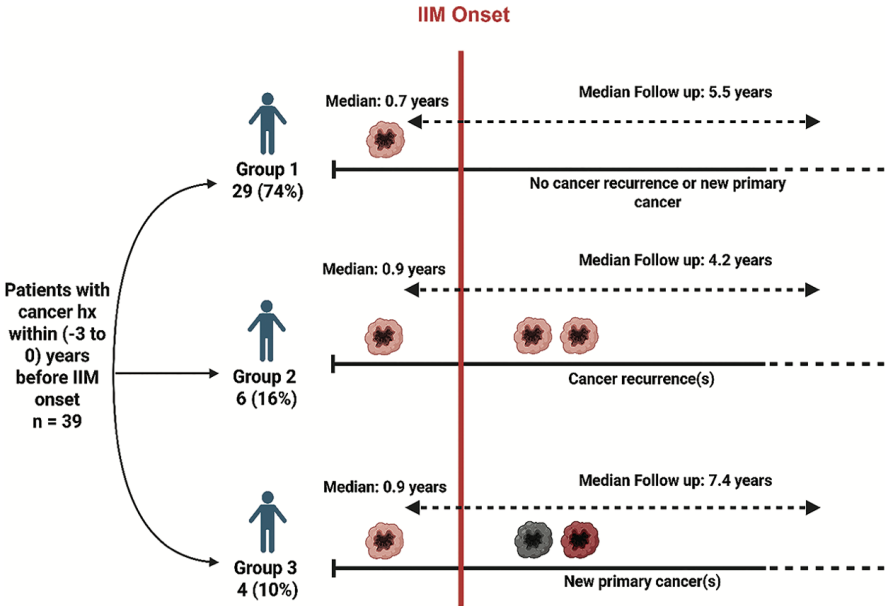

Fig. 1. Adult IIM patients with a history of cancer diagnosed within the 3 years prior to IIM symptom onset.Patients can have one of three outcomes: Group 1: no cancer recurrence or new primary; Group 2: cancer recurrence; Group 3: new primary cancer.

Results

Out of a total of 2,476 adult IIM patients, 280 (11%) patients had a confirmed history of cancer, 39 (14%) of which were diagnosed within the 3 years prior to IIM symptom onset. Of these 39, the majority were female (59%), white (82%), and had the IIM subtype of DM (62%).

Of 39 adult IIM patients with a history of cancer within the 3 years prior to IIM symptom onset, 29 (74%) remained cancer free after IIM symptom onset over a median follow-up period of 5.5 (IQR 6.7) years (Group 1), with a median time of 0.7 (IQR 1.3) years between their index cancer and IIM symptom onset. Six (16%) patients developed recurrence of their original cancer after IIM symptom onset over a median follow-up period of 4.2 (IQR 2.3) years (Group 2), with a median time of 0.9 (IQR 1.2) years between their index cancer and symptom onset, and four (10%) developed a new primary cancer after IIM symptom onset over a follow-up period of 7.4 (IQR 6.9) years (Group 3), with a median time of 0.9 (IQR 1.5) years between their index cancer and IIM symptom onset (Fig. 1). The median time from index cancer to recurrence was 2.4 (IQR 1.6) years, whereas the time from index cancer to new primary was 1.4 (IQR 1.0) years (Table I).

Of 39 adult IIM patients with a history of cancer within the 3 years prior to IIM symptom onset, 14 (36%) died during follow-up. Mortality varied significantly across outcome groups: 8/29 (28%) patients who remained cancer-free after IIM symptom onset died, compared to 5/6 (83%) patients with cancer recurrence and 1/4 (25%) with a new primary cancer. Using a competing-risk analysis that accounted for death as a competing event, the cumulative incidence of recurrence was 21.4% (95% CI: 0.0–44.3%) at 5 years and 35.7% (95% CI: 7.9–63.6%) at 10 years. For new primary cancer, both the 5-year and 10-year cumulative incidence was 7.1% (95% CI: 0.0–21.4%).

No significant differences were observed in sex, race, or IIM type when comparing across groups (Table I). With regards to autoantibodies, the median interval from IIM symptom onset to antibody testing was 1.8 (IQR 4.7) years. No significant associations were found between autoantibody status and cancer outcomes. However, 38% (5/13) of patients positive for anti-TIF1γ antibodies developed a recurrence or new primary cancer after IIM symptom onset during follow-up, compared with 0% (0/10) of anti-NXP2-positive and 10% (1/10) of anti-SAE-positive patients.

Of the 6 patients who had recurrence of their index cancer (Group 2), 2/6 were ovarian cancers. Of the 4 patients who developed a new primary cancer after IIM symptom onset (Group 3), all were diagnosed within the first three years from IIM symptom onset. The new primary cancers included breast, melanoma, lung and thyroid (Fig. 2). Tumour-type distributions for recurrences and new primary cancers in the primary analysis are shown in (Supplementary Fig. S1A and S2A).

Given prior research suggesting an association between anti-CCAR1 and anti-SP4 antibodies and reduced cancer risk among DM patients, we examined the association between these autoantibodies and recurrence/new primary cancer development (8, 9). Anti-CCAR1 antibodies were not present in any patients in the recurrence group, 25% (1 of 4 patients) in the new primary cancer group, and zero patients in the cancer-free group. Similarly, anti-SP4 antibodies were not present in any patients in the recurrence group, present in 25% (1 of 4 patients) in the new primary group, and zero patients in the no new cancer group.

For the secondary analysis, we included patients with cancer diagnosed at any time before IIM symptom onset. A total of 121 adult IIM patients met this criterion; most were female (68%), white (83%), and had the DM subtype (56%). 95 (78%) remained cancer-free after IIM onset over a median follow-up period of 5.9 (IQR 7.5) years (Group 1),

Table I. Demographic summary for IIM patients with a history of cancer within -3 to 0 years of IIM symptom onset.

	Patients who had a history of cancer from -3 to 0 years of IIM onset					
	Total	Patients without recurrence or primary after IIM onset	Patients with Multiple Cancer (either recurrence or new primary) after IIM onset		*p*-value (single *vs.* recurrence)	*p*-value (single *vs.* new primary)
			Recurrence	New Primary		
Cancer	39 (100%)	29 (74%)	6 (16%)	4v (10%)		
Median number of cancers (range)	1 (1-3)	1	2 (2-3)	2 (2-3)		
Median time between cancers (range, years)	1.5 (IQR: 1.4)	NA	2.4 (IQR: 1.6)	1.4 (IQR: 1.0)		
Median time between index cancer and symptom onset (years)	0.7 (IQR: 1.3)	0.7 (IQR: 1.3)	0.9 (IQR: 1.2)	0.9 (IQR: 1.5)	0.662	0.890
Median follow-up duration since symptom onset (years)	5.0 (IQR: 6.6)	5.5 (IQR: 6.7)	4.2 (IQR: 2.3)	7.4 (IQR: 6.9)	0.710	0.457
Median age at IIM onset (median)	56.9	56.9	53.5	64.3	0.512	0.457
Number of deceased patients	14 (100%)	8 (57%)	5 (36%)	1 (7%)	0.019	1.000
Sex						
Male	16 (100%)	14 (88%)	1 (6%)	1 (6%)	0.207	0.607
Female	23 (100%)	15 (65%)	5 (22%)	3 (13%)		
Race						
Asian	4 (100&)	4 (100%)	0 (0%)	0 (0%)	0.079	1.000
Black	1 (100%)	0 (0%)	1 (100%)	0 (0%)		
White	32 (100%)	25 (78%)	3 (9%)	4 (12%)		
Other/Unknown	2 (100%)	0 (0%)	2 (100%)	0 (0%)		
IIM type						
Dermatomyositis	24 (100%)	17 (71%)	3 (12%)	4 (17%)	1.000	0.185
Polymyositis	15 (100%)	12 (80&)	3 (20%)	0 (0%)		
ASyS	3 (100%)	1 (33%)	1 (33%)	1 (33%)		
IMNM	6 (100%)	6 (100%)	0 (0%)	0 (0%)		
Overlap Myositis	3 (100%)	2 (67%)	1 (33%)	0 (0%)		
Autoantibody						
Anti-TIF1-γ (ELISA)	13 (100%)	8 (62%)	3 (23%)	2 (15%)	0.352	0.567
Anti-TIF1-γ alone (Anti-CCAR1 and Anti-SP4 neg)	8 (100%)	3 (38%)	3 (38%)	1 (24%)	0.049	0.099
Anti-TIF-γ (Anti-CCAR1 OR Anti-SP4)	1 (100%)	0	0	1 (100%)	1.000	0.121
Anti-TIF-γ (Anti-CCAR1 and Anti-SP4)	1 (100%)	0	0	1 (100%)	1.000	0.121
Anti-Mi2 (Line Blot/ELISA)	3 (100%)	3 (100%)	0 (0%)	0 (0%)	1.000	1.000
Anti-HMGCR (ELISA)	3 (100%)	3 (100%)	0 ´0~`	0 (0%)	1.000	1.000
Anti-PL7 (Line Blot)	2 (100%)	2 (100%)	0 (0%)	(0%)	1.000	1.000
Anti-SAE (Line Blot)	2 (100%)	1 (50%)	0 (0%)	1 (50%)	1.000	0.231
Anti-SRP (Line Blot)	2 (100%)	1 (50%)	0 (0%)	1 (50%)	1.000	0.231
Anti-Jo1 (Line Blot)	1 (100%)	0 (0%)	1 (100%)	0 (0%)	0.171	1.000
Anti-Ku (Line Blot)	1 (100%)	1 (100%)	0 (0%)	0 (0%)	1.000	1.000
Anti-PL12 (Line Blot)	1 (100%)	0 (0%)	1 (100%)	0 (0%)	0.171	1.000
Anti-NXP2 (IVTT IP)	1 (100%)	1 (100%)	0 (0%)	0 (0%)	1.000	1.000
Anti-PM/Scl (Line Blot)	0 (100%)	0	0	0	1.000	1.000
Anti-EJ (Line Blot)	0 (100%)	0	0	0	1.000	1.000
Anti-MDA5 (Line Blot)	0 (100%)	0	0	0	1.000	1.000
Anti-OJ (Line Blot)	0 (100%)	0	0	0	1.000	1.000
Cancer site						
Breast	12 (100%)	9 (75%)	1 (8%)	2 (17%)	0.649	0586
Melanoma	5 (100%)	3 (60%)	0 (0%)	2 (40%)	1.000	0.099
Abdominal	4 (100%)	3 (75%)	1 (25%)	0 (0%)	0.546	1.000
Thyroid	3 (100%)	1 (33%)	0 (0%)	2 (67%)	1.000	0.033
Lung	3 (100%)	1 (33%)	1 (33%)	1 (33%)	0.318	0.231
Ovary	2 (100%)	0(0%)	2 (100%)	(0%)	0.025	1.000

with a median time of 7.1 (IQR 11.5) years between index cancer and IIM symptom onset. Thirteen (11%) patients developed recurrence of their original cancer over a median follow-up period of 6.0 (IQR 2.9) years (Group 2), with a median time of 4.4 (IQR 9.6) years between index cancer and IIM symptom onset, and thirteen (11%) developed a new primary cancer over a median follow-up period of 6.4 (IQR 1.9) years (Group 3), with a median time of 5.0 (IQR 16.0) years between index cancer and IIM symptom onset (Suppl. Fig. S3). The median time from index cancer to recurrence was 4.8 (IQR 8.4) years, whereas the time from index cancer to new primary was 7.9 (IQR 15.7) years (Suppl. Table S1). Tumour-type distributions and timing (≤3 years *vs.* >3 years after IIM onset) for first recurrences and new primaries are shown in (Suppl. Fig. S1B and S2B]. The most common new primary cancers included breast, melanoma and thyroid, and the most common cancer that recurred was breast cancer (Suppl. Fig. S4 and S5). There were no significant differences in demographic characteristics between the groups (Suppl. Table S1). When examining autoantibody status, of the 23 IIM patients positive for anti-TIF1γ antibodies, 5 (22%) experienced recurrent cancer, and 3 (13%) were diagnosed with a new primary cancer after symptom onset.

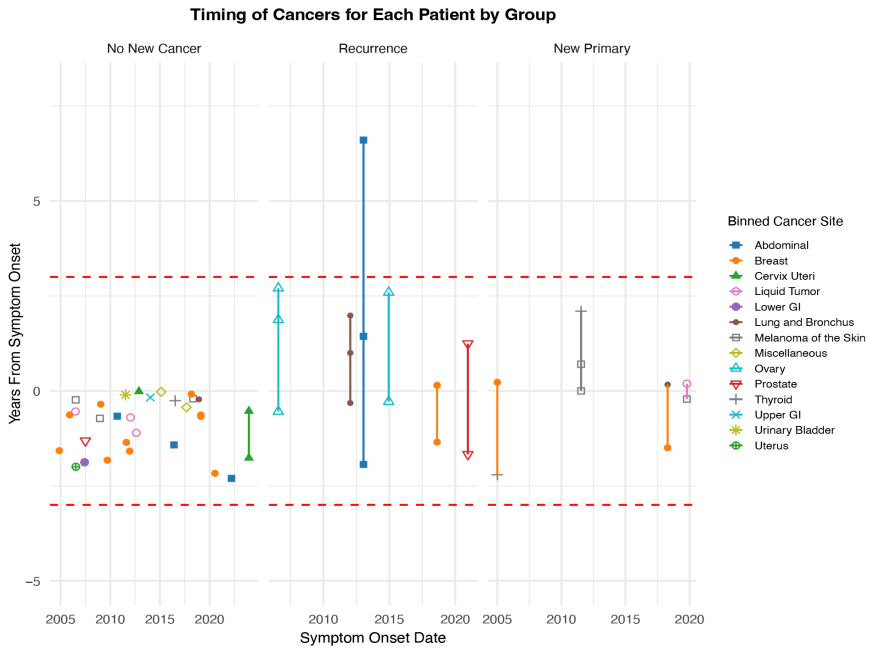

Fig. 2. Timing of cancers relative to symptom onset date in patients diagnosed with cancer within 3 years prior to IIM symptom onset (-3 to 0 years) (primary analysis). Patients are grouped based on cancer outcome: no new cancer, recurrence of prior cancer, and development of new primary cancer after IIM symptom onset. Red dashed lines indicate a period of 3 years before and after IIM symptom onset (-3 to +3 years).

Discussion

Our findings highlight the need for continued oncologic surveillance in adult IIM patients with a prior cancer history. Among those diagnosed with cancer within 3 years before IIM onset, 26% developed another malignancy after IIM, 16% as recurrences and 10% as new primaries, underscoring persistent cancer risk in this high-risk subset. Notably, all recurrences and new primaries were diagnosed within 3 years of IIM onset.

Among patients with a history of cancer within 3 years of IIM onset, breast cancer was the most common malignancy occurring after IIM symptom onset (2 of 10 cancers in the primary analysis; 8 of 36 in the secondary analysis). The second most common cancer in our cohort was melanoma skin cancer. This observation is consistent with the findings by Luo *et al*., highlighting melanoma and other epithelial sites after IIM (10). This data may suggest the need for heightened vigilance for such cancers in the post-IIM period, even among patients already diagnosed with cancer within the three years preceding IIM onset.

Consistent with prior literature, a strikingly high number of patients who had a history of cancer within 3 years of symptom onset died over follow-up. Of the six patients who developed recurrence, only one patient (who had ovarian cancer recurrence) was alive as of the censoring date for our study. The high fatality we observed after recurrence aligns with findings from Che *et al*. (11). Although they did not distinguish recurrent cancers from new primaries, their data similarly demonstrate that once any cancer occurs after IIM onset, mortality increases sharply. This elevated mortality could also be exacerbated by immunosuppressive therapies commonly used in IIM management, which may impair anti-tumour immunity and increase susceptibility to recurrence. Future studies quantifying treatment exposure as an effect modifier are warranted.

In our cohort, of the 47 patients who were diagnosed with breast cancer prior to IIM onset, 6 (13%) experienced a recurrence. In contrast to breast cancer patients without IIM where the highest window of recurrence is within the first 5 years (12), in our IIM cohort, the median time from initial breast cancer diagnosis to recurrence was 11.4 years, with a range extending over two decades, emphasising the prolonged risk window in this patient population. Although cancer risk in IIM is classically clustered within ±3 years of IIM onset, recent work also documents "long interval" cancers, especially in anti TIF1γ-positive DM, suggesting that a subset remains at risk beyond 3 years and may benefit from individualised surveillance (13).

Among the 39 patients diagnosed with cancer within 3 years prior to IIM onset, 13 (33%) were positive for anti-TIF1γ antibodies. Of these, 5/13 (38%) developed either recurrent or new primary cancers. Specifically, recurrence rates were higher among TIF1γ-positive patients compared to TIF1γ-negative patients (23% *vs*. 11%), although this difference did not reach statistical significance. While the underlying mechanism awaits investigation, several possibilities exist: (i) an anti-TIF1γ immune response may serve as a marker of a cancer-permissive state, (ii) patients with anti-TIF1γ positivity may receive more potent immunosuppressive therapy, thereby increasing cancer susceptibility, or (iii) these patients may inherently have cancer types or stages associated with a higher likelihood of recurrence. Emerging evidence suggests that the co-presence of other autoantibodies, such as anti-CCAR1 and anti-SP4, might modulate cancer risk among DM patients, potentially lowering malignancy incidence and recurrence rates (8, 9). However, anti-CCAR1 and anti-SP4 positivity rates did not significantly differ across different cancer outcome groups, possibly due to small numbers. Future studies in larger patient populations will be necessary to examine the association between these two autoantibodies and risk of cancer recurrence and the development of new primary cancers.

Our study has several limitations. Given our tertiary referral centre population, the generalisability of our findings may be limited. As a retrospective study, the availability of clinical details was limited, particularly regarding treatment refractoriness which was not systematically collected. This limitation affects our ability to evaluate whether treat-

ment intensity influenced malignancy risk. Additionally, data on family history of cancer, genetic predisposition, and lifestyle factors such as smoking and alcohol use were not systematically recorded. These variables may significantly impact cancer risk and should be assessed in future prospective studies to better delineate their role in this population. In addition, we lacked a general population reference (patients with cancer without IIM) for rates of recurrence and new primary cancer, as the Surveillance, Epidemiology, and End Results Program (SEER Registry) does not report these data. Lastly, we did not have access to linked external cancer registries with our electronic health record system and relied solely on clinical documentation, potentially resulting in incomplete ascertainment of cancer outcomes. However, all patients were contacted directly to determine cancer status.

Despite these limitations, our findings underscore the need for ongoing cancer surveillance in IIM patients with a cancer diagnosis in the three years preceding IIM onset. Our data support an individualised surveillance approach that is intensified during the first three years after IIM onset and may even extend beyond this period, especially for cancers such as breast cancer and for patients with high-risk serologic profiles, including anti-TIF1γ.

Competing interests

J.J. Paik has received research funding and clinical trial support from EMD Serono, Priovant, Astrazeneca, ArgenX, in accordance with institutional conflict-of-interest policies.

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The other authors have declared no competing interests.

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