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# Inflammatory arthritis and malignancy: A consensus report on risk assessment and clinical management based on a systematic review from the Turkish Society of Rheumatology Malignancy Study Group

## Türkiye Romatoloji Derneği - Romatoloji ve Malignite Çalışma Grubu enflamatuvar artrit ve malignite: Sistematik derlemeye dayalı risk değerlendirmesi ve klinik yönetim üzerine fikir birliği raporu

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**Abstract**

Inflammatory arthritis (IA) is associated with an increased risk for certain malignancies, particularly lymphoma, due to underlying chronic inflammation. Conventional and targeted therapies used in IA modulate the immune system, raising concerns about the development of *de novo* malignancies or the progression of pre-existing ones. Managing IA patients with a history of cancer remains one of the most challenging areas for clinicians, and while international guidelines exist, they generally focus on a narrower scope. This report is the first comprehensive consensus report from Türkiye to address the relationship between IA and malignancy across a wide spectrum, including baseline risk, treatment-related risk, management of patients with a history of cancer, treatment during active malignancy, premalignant lesions, and family history. Based on a systematic literature review, this report provides evidence-based, practical

**Özet**

Enflamatuvar artrit (İA), altta yatan kronik enflamasyona bağlı özellikle lenfoma başta olmak üzere bazı malignite türleri için bir risk artışı ile ilişkilidir. İA tedavisinde kullanılan konvansiyonel ve hedefe yönelik tedaviler, immün sistemi modüle etmeleri nedeniyle *de novo* malignite gelişimi veya var olan malignitenin seyri konusunda endişeler barındırmaktadır. Kanser öyküsü olan İA hastalarının yönetimi, hekimler için en zorlayıcı alanlardan biridir ve bu konudaki uluslararası rehberler mevcut olsa da genellikle daha dar bir kapsama odaklanmıştır. İA ve malignite ilişkisini; temel risk, tedaviye bağlı risk, kanser öyküsü olan hasta yönetimi, aktif kanser sırasında tedavi, premalign lezyonlar ve aile öyküsü gibi geniş bir yelpazede ele alan ilk kapsamlı Türkiye fikir birliği raporudur. Sistematik literatür taramasına dayalı olarak, mevcut uluslararası rehberlerin daha dar kapsamda ele aldığı; aktif tedavi altında kanser gelişimi, premalign lezyonlar ve aile öyküsü gibi

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recommendations for specific scenarios frequently encountered in daily practice—such as cancer development during active treatment, premalignant lesions, and family history—which are often narrowly addressed in existing international guidelines. This report will help rheumatologists standardize decision-making processes regarding the coexistence of IA and malignancy, enabling them to take safer clinical steps. By promoting risk individualization and shared decision-making between patients and clinicians, it will strengthen personalized treatment approaches that ensure both effective control of rheumatic disease and oncologic safety.

**Keywords:** Inflammatory arthritis, rheumatoid arthritis, spondyloarthritis, malignancy, cancer, biological therapies, disease modifying anti-rheumatic drugs (DMARDs)

günlük pratikte sık karşılaşılan spesifik senaryolar için kanıta dayalı pratik öneriler sunar. Sistematik literatür taramasına dayalı olarak, mevcut uluslararası rehberlerin daha dar kapsamda ele aldığı; aktif tedavi altında kanser gelişimi, premalign lezyonlar ve aile öyküsü gibi günlük pratikte sık karşılaşılan spesifik senaryolar için kanıta dayalı pratik öneriler sunar. Bu rapor, romatologların İA ve malignite birlikteliğindeki karar verme süreçlerini standartlaştırmasına ve daha güvenli adımlar atmasına yardımcı olacaktır. Risklerin bireyselleştirilmesi ve hasta ile hekim arasında paylaşılan karar verme süreçlerinin teşvik edilmesi yoluyla, hem romatizmal hastalığın etkin kontrolünü hem de onkolojik güvenliği gözeten kişiselleştirilmiş tedavi yaklaşımlarını güçlendirecektir.

**Anahtar Kelimeler:** Enflamatuvar artrit, romatoid artrit, spondiloartrit, malignite, kanser, biyolojik tedaviler, hastalık modifiye edici antiromatizmal ilaçlar (DMARD)

## Introduction

The management of inflammatory arthritis (IA), including rheumatoid arthritis (RA) and spondyloarthritis (SpA), has been revolutionized over the past two decades with the widespread adoption of disease-modifying anti-rheumatic drugs (DMARDs), especially biologic and targeted synthetic DMARDs (b/tsDMARDs). While these therapies significantly improve patients' quality of life by controlling disease activity, their complex relationship with malignancy is a major concern for both clinicians and patients due to their immunomodulatory effects.

This relationship has two key dimensions. The first is the underlying disease itself. It is well-established that chronic inflammation and autoimmune processes are risk factors for certain types of cancer, particularly malignant lymphomas. The second dimension involves the potential risks of the therapies used to suppress this inflammation. Specifically, b/tsDMARDs have raised theoretical concerns regarding the risk of *de novo* malignancy, as they target critical pathways involved in cancer surveillance, such as tumor necrosis factor (TNF), interleukin (IL)-6, and Janus kinase/signal transducer and activator of transcription (JAK/STAT). Elucidating this complex risk profile requires a systematic evaluation of the evidence.

This overall picture raises a series of complex clinical questions in practice. With advancements in cancer treatment and an aging population, the number of IA patients with a history of cancer is steadily increasing. In these patients, balancing the need to treat active rheumatic disease against the concern of cancer recurrence is a primary challenge. Furthermore, there is a significant need for clear, unified guidance on the optimal management of patients who develop malignancy while on active anti-rheumatic therapy, and personalizing treatment choices for individuals with premalignant lesions or a strong family history of cancer.

To address these comprehensive clinical questions and fill existing evidence gaps, a systematic literature review (SLR) was conducted based on pre-defined Population, Intervention, Comparison, Outcome (PICO) questions. The primary aim of this study is to integrate the evidence obtained from the SLR with the clinical experience of expert rheumatologists, presenting an evidence-based consensus report and practical clinical management recommendations on the broad relationship between IA and malignancy. This report systematically addresses risks stemming from the disease itself, the risk profiles of different treatment options, and the management of patients with a history of cancer and other special circumstances.

## Methodology

The objective of this study was to assess the malignancy risk associated with the underlying disease and its treatments in patients with RA and SpA, and also to create an evidence-based consensus report and a set of clinical practice recommendations, particularly for the management of patients with a history of cancer.

### 1. Formation of the Study Group

A task force was established among rheumatologist members of the Turkish Society of Rheumatology (TSR) to achieve the project's objectives. All participants submitted potential conflict of interest declarations before commencing the process.

### 2. Definition of PICO Questions and Systematic Literature Review

In its initial meeting, the task force finalized the research questions in the PICO format, which formed the foundation of the project. The study group was divided into subgroups for each PICO question, and these groups shared the findings of their work with the entire task force in regular meetings.

These questions determined the scope and strategy of the SLR. The core research questions examined were:

• **PICO 1:** In individuals with RA (P), is the overall incidence of malignancy (O) higher compared to the general population (C)?

• **PICO 2:** In individuals with SpA [including ankylosing spondylitis and psoriatic arthritis (PsA) subtypes] (P), is the overall incidence of malignancy (O) higher compared to the general population (C)?

• **PICO 3:** In RA patients (P), how does the use of a specific class of DMARD (I) affect the risk of developing *de novo* malignancy (O) compared to another DMARD class or no treatment (C)?

3a. Conventional synthetic DMARDs (csDMARDs)

3b. TNF inhibitors

3c. Non-TNF biologic DMARDs (bDMARDs)

3d. Targeted synthetic DMARDs (tsDMARDs)

• **PICO 4:** In RA patients with a history of malignancy (P), what is the effect of initiating a specific DMARD class (csDMARD, bDMARD, tsDMARD) (I) on malignancy recurrence or the development of a new primary malignancy (O) compared to patients not receiving active treatment or receiving a different DMARD class (C)?

4a. In those with a history of solid organ tumors

4b. In those with a history of melanoma

4c. In those with a history of non-melanoma skin cancer

4d. In those with a history of lymphoproliferative disease

• **PICO 5:** In RA patients diagnosed with malignancy during active DMARD (cs/b/tsDMARD) therapy (P), what is the effect of different management strategies for the current anti-rheumatic treatment (I) on the patient's rheumatologic and oncologic survival outcomes (O)?

• **PICO 6:** In RA patients with a known premalignant lesion (P), what is the effect of initiating or continuing a specific DMARD therapy (I) on the risk of the lesion's progression to malignancy (O)?

• **PICO 7:** In RA patients with a strong family history of cancer (e.g., in first-degree relatives) (P), is there an evidence-based approach to optimizing treatment selection among different DMARD classes (I), considering the patient's future malignancy risk (O)?

**Data Sources and Search Strategy:** A comprehensive literature search was conducted in major medical databases, including PubMed/MEDLINE, Embase, and the Cochrane Library. A detailed search strategy was developed using keywords and Medical Subject Headings (MeSH) terms specific to each PICO question.

**Study Selection Criteria:**

• **Inclusion Criteria:** Randomized controlled trials, observational cohort studies, case-control studies, meta-analyses, and systematic reviews that answered the defined PICO questions were included. To broaden the scope of evidence, studies on other inflammatory diseases where similar treatment mechanisms are used, such as inflammatory bowel disease (IBD), were also considered.

• **Exclusion Criteria:** Case reports, editorials, expert opinion articles, animal studies, and studies not relevant to the PICO questions were excluded.

The SLR was conducted by the designated subgroups for each PICO question. Data extraction and quality assessment were performed according to a predefined standard protocol. Extracted data included study design, patient population, treatment type, follow-up duration, and outcomes such as cancer incidence, cancer recurrence, hazard ratios, and relative risk.

### 3. Development of Recommendations and Voting

The SLR results were discussed within the respective PICO subgroups and reported in evidence summary tables. Subsequently, the Task Force convened in online meetings to discuss these data holistically. At the end of this process, draft statements were prepared by each PICO group under the headings "General Principles" and "Specific Recommendations" addressing the PICO questions.

**Voting and Consensus:** The drafted recommendations were subjected to an anonymous voting process involving all task force members. Each recommendation was rated on a 6-point Likert scale from 0 (completely disagree) to 5 (completely agree). Recommendations with a mean agreement score of 4.0 or higher were accepted with strong consensus. Items that did not reach sufficient consensus were rediscussed and revoted.

### Final Text Approval

The final recommendation text was first reviewed and approved by all members of the task force and subsequently by the members and executive board of the TSR.

### Recommendations

This section presents the consensus recommendations reached following the SLR and expert opinion, guided by the PICO questions outlined in the methodology.

**Note:** The number in parentheses at the end of each recommendation indicates the mean level of agreement on a 0 (completely disagree) to 5 (completely agree) Likert scale.

**Section 1. Inflammatory Arthritis and Baseline Malignancy Risk (PICO 1&2)**

This section assesses the impact of the underlying inflammatory rheumatic disease itself on the development of malignancy.

**1.1. Rheumatoid Arthritis**

**• 1.1.1. Overall and Hematologic Risk**

An increased risk of cancer development has been identified in RA patients compared to the general population. High and cumulative disease activity is considered the primary driver of the increased risk of lymphoma (4.87).[1-6]

An overall increased risk of hematologic malignancies has been found in RA patients (4.68).[2-5,7-16]

Lymphoma is the hematologic malignancy with the most significantly increased risk in this group (4.75).[2-4,7,9-16]

While there are data suggesting an increased risk of leukemia and multiple myeloma in RA, this evidence is insufficient (4.43).[3,5,7,8]

**• 1.1.2. Solid Organ Risk**

It is difficult to establish increased cumulative risk of solid organ malignancy in RA patients, as an increased risk has been observed for some solid organ cancers, while a decreased risk has been seen for others (4.5).

**- Lung Cancer:** This is the solid organ malignancy with the highest observed risk increase in RA patients (4.68).[2-5,7,17-20] The evidence is insufficient to conclude that RA is an independent risk factor for lung cancer, separate from known factors like interstitial lung disease and smoking (4.5).[17,18]

**- Colorectal Cancer:** The risk of colorectal cancer in RA is lower than in the general population, and this has been suggested to be associated with the frequent use of non-steroidal anti-inflammatory drugs (4.62).[1,5-7,11,16]

**- Other Solid Organs:** RA is not considered a risk factor for gastric, hepatic, biliary tract, pancreatic, or thyroid cancers (4.56).[2,5,7,16,19] It has also not been associated with the development of breast cancer (4.66).[2,5,7,21,22] The risk status for urinary tract malignancies remains uncertain (4.56).[2,5,7,19] Likewise, current data do not provide clear evidence of an increased risk for gynecological malignancies (4.5).[2,4,5,7] Moreover, there are insufficient data to suggest an increased risk of prostate cancer (4.75).[2,5,23]

**• 1.1.3. Skin Cancer Risk:** The risk of malignant melanoma in RA is uncertain (4.5).[2,4,5] The current evidence is insufficient to state that RA is a risk factor for other skin cancers (4.62).[3]

**1.2. Spondyloarthritis**

**• 1.2.1.** While the overall risk of malignancy in SpA patients does not significantly differ from the general population, some studies have reported an increased risk for certain hematologic malignancies, such as lymphoma and multiple myeloma (4.62).[24-35]

**• 1.2.2.** In PsA patients, the overall risk of solid and hematologic malignancies is not increased (4.56).[24,27,36-38] However, some studies have reported an increased risk of non-melanoma skin cancer (NMSC) (4.56).[27,37,39-42]

**• 1.2.3.** Patients with enteropathic arthritis are at an increased risk for gastrointestinal cancers due to the concomitant IBD (4.81).[43-47]

**Section 2. Anti-Rheumatic Therapies and *de novo* Malignancy Risk (PICO 3)**

This section examines the effect of anti-rheumatic treatments on malignancy development.

**• 2.1. Conventional Synthetic DMARDs (csDMARDs):** The use of csDMARDs does not cause increase in malignancy risk in patients with RA (4.81).[48,49] Data on its association with lymphoma risk are conflicting (4.18).[50,51] However, csDMARDs may increase the risk of NMSC (4.43).[52,53]

**• 2.2. Biologic and Targeted Synthetic DMARDs (b/tsDMARDs):**

**- Overall Malignancy Risk:** The relationship between TNF inhibitors and malignancy development is controversial, and recent meta-analyses have not confirmed this link. Data for non-TNF biologics are more limited, but no significant risk increase has been reported (4.5).[2,54-58]

**- JAK Inhibitors:** The ORAL surveillance trial found that tofacitinib use in high-risk RA patients increased the risk of malignancy, particularly lung cancer (4.68).[59] The FDA considers this risk increase as a class effect for other JAK inhibitors. However, other studies comparing bDMARDs with JAK inhibitors have not observed an overall increase in solid organ malignancy risk (4.31).[60,61]

**- Lymphoma Risk:** There is some evidence suggesting that bDMARD use may be associated with a slight increase in lymphoma risk, possibly due to the underlying high disease activity (4.31).[62-65]

**Section 3. Treatment Management in Patients with a History of Malignancy (PICO 4)**

This section addresses the safety of anti-rheumatic treatment options in patients previously diagnosed with cancer.

**• 3.1. History of Solid Organ Tumor:** Current evidence indicates that methotrexate, leflunomide, sulfasalazine, hydroxychloroquine, TNFi, and non-TNFi bDMARDs such as tocilizumab and rituximab do not increase the risk of recurrence (4.75, 4.5, 4.5).[56,66-77] Therefore, patients with a

history of malignancy may be treated in a manner similar to those without such a history, provided that an individualized assessment is conducted and, when necessary, collaboration with oncology is ensured (4.18).[78] For JAK inhibitors, there are insufficient data in this population (4.5).[66,69,74] Although data are limited, given its mechanism of action, abatacept should be used cautiously in situations where no alternative therapeutic options are available.[79]

• **3.2. History of Non-Melanoma Skin Cancer:** Although historic data reports an association of increased *de novo* NMSC risk, current evidence suggests that TNF inhibitors and non-TNF bDMARDs do not increase recurrence risk (4.43, 4.5).[56,71-77,80] JAK inhibitors and abatacept may be used with caution if therapeutic alternatives are unavailable. Data regarding methotrexate and leflunomide are insufficient, whereas sulfasalazine and hydroxychloroquine are considered safe (4.75).[66,78,81] These patients should undergo regular skin examinations every 6-12 months (4.81).[66]

• **3.3. History of Melanoma:** Given melanoma's strong dependence on immune surveillance and the lack of sufficient data, a more cautious approach is warranted in patients with a history of melanoma, who should be considered separately from the general recommendations for TNF inhibitors in patients with a history of solid cancers.[79]

• **3.4. History of Lymphoproliferative Disease:** Rituximab may be the preferred first-line option in this patient group, as it does not increase the risk of recurrence (4.81).[66,68-70,73,76,82] Tocilizumab and abatacept also do not increase recurrence risk and may be preferred over TNF inhibitors (4.4).[66,82-84] There is no evidence that TNF inhibitor use increases the risk (4.43).[72-77] Although data for methotrexate and leflunomide are limited, they do not suggest an increased risk of recurrence (4.68).[66,82,78]

### Section 4. Management of Patients Who Develop Malignancy During Active Treatment (PICO 5)

This section addresses the management of patients diagnosed with malignancy while on active anti-rheumatic therapy.

• **4.1. General Approach:**

- **Malignancy Development on csDMARDs:** Treatment is personalized. Generally, agents with lower immunosuppressive potential, such as hydroxychloroquine and sulfasalazine, are preferred (4.5).[85-90] In cases of lymphoproliferative disease, discontinuation of methotrexate and leflunomide should be considered (4.5).[91,92]

- **Malignancy Development on b/tsDMARDs:** Although the level of evidence is insufficient, the standard approach in routine practice is to discontinue these agents due to the risk of potential drug interactions and toxicity (4.68).[93,94]

• **4.2. RA Management During Active Cancer Treatment:** If RA activity persists, glucocorticoids and/or csDMARDs are the preferred initial choice (4.81, 4.43).[85-90] If these treatments are inadequate, a bDMARD (TNF inhibitor or rituximab) may be initiated on a case-by-case basis in collaboration with an oncologist (4.43).[57,93,95-97]

• **4.3. Special Situation: Immune Checkpoint Inhibitors (ICIs):** ICI therapy can exacerbate RA.[98] If steroids and csDMARDs are insufficient for flares, TNF inhibitors or IL-6 inhibitors may be used.[96,97] Abatacept should be avoided due to concerns that it may negatively affect the anti-tumor response of ICIs.[93]

• **4.4. Palliative Patients:** In palliative patients, quality of life is paramount, and treatment is personalized in collaboration with an oncologist. Methotrexate is not recommended due to its potential hematologic toxicity (4.62).[86]

### Section 5. Treatment Management in Special Situations (PICO 6&7)

This section examines the management of patients with premalignant lesions or a family history of cancer.

• **5.1. Approach in the Presence of Premalignant Lesions (PICO 6):** A strategy of proactive monitoring and early intervention should be adopted for these patients (4.81). Evidence is insufficient to suggest that medications increase the development or progression of lesions (4.56, 4.5, 4.62),[98,99] although it has been reported that TNF inhibitors may increase the risk of *in situ* squamous cell carcinoma (4.25).[100] It is recommended to eliminate the lesion before starting therapy. If this is not possible, options other than JAK inhibitors and abatacept may be prioritized (4.5).[101]

• **5.2. Approach in the Presence of a Family History of Malignancy (PICO 7):** More rigorous surveillance programs are recommended for these patients instead of standard screening programs (4.75). There is no evidence that immunosuppressive therapies further increase this risk (4.56). However, the decision to use bDMARDs and tsDMARDs requires a careful risk-benefit analysis (4.56).

### Discussion

In this study, the task force examined the complex relationship between IA and malignancy through seven main PICO questions, culminating in an evidence-based consensus report. This report offers a comprehensive framework, starting from the baseline malignancy risk in inflammatory arthritis, to the impact of anti-rheumatic

therapies on *de novo* cancer risk, the management of patients with a history of cancer or an active cancer, and special situations such as premalignant lesions and family history.

The core themes of this work include: (a) the necessity of individualizing risk for each patient, (b) the importance of striking a balance between the risks of undertreated rheumatic disease and the potential risks of anti-rheumatic therapy, and (c) the principle that treatment decisions should be made through a process of shared decision-making between the patient and clinician. Furthermore, the recognition that chronic inflammation itself is a risk factor and that effective disease control can mitigate this risk is another crucial element underpinning all recommendations.

The findings from this systematic review have confirmed several key points. Patients with RA have an increased risk of lymphoma, particularly associated with high disease activity. Regarding treatments, csDMARDs and bDMARDs may slightly increase the risk of NMSC. Among the tsDMARDs, the ORAL Surveillance trial highlighted a specific risk increase (lung cancer) with JAK inhibitors in high-risk patients (advanced age, smoking history), emphasizing the need for careful patient selection when using these agents.

These recommendations, although based on the best available evidence, should be interpreted with consideration for several important limitations in the literature. First, the vast majority of available data is derived from RA patients. The data for the SpA and other IA subtypes are more limited. Second, long-term safety data for b/tsDMARDs other than TNF inhibitors remain insufficient. A third and major limitation is that the median follow-up times in studies are often short (e.g., <5 years), which may not be long enough to detect late recurrences of some solid organ cancers. Finally, most studies do not differentiate between a new primary cancer and a recurrence of an existing cancer, and they often do not report oncologic outcomes such as survival. These factors underscore the need for clinician caution, especially concerning newer agents and rare cancer types, and highlight the necessity for more observational studies in this field.

This study provides several important messages for clinical practice. The approach to treatment in patients, with a history of cancer, has shifted away from the previously held, more rigid stance of avoiding all targeted therapies. There is growing evidence that many DMARDs, particularly TNF inhibitors—supported by the largest available dataset—do not increase the risk of cancer recurrence, especially in patients with a history of a solid organ tumor. For patients with a history of lymphoma, the consensus has solidified, indicating that rituximab, which causes B-cell depletion, is a rational choice for both its rheumatologic efficacy and oncologic safety profile. For JAK inhibitors and abatacept, due to a lack of direct evidence and reliance on indirect data, the prevailing cautious approach is to limit their use to situations where other therapeutic alternatives are not available, especially in patients with a history of cancer or premalignant lesions.

The scenario where malignancy develops during active treatment represents one of the areas with the weakest evidence base. In this situation, our recommendation to discontinue b/tsDMARDs is not based on definitive evidence but rather represents a pragmatic, safety-first approach aimed at avoiding potential drug interactions and unforeseen risks. The management of such complex cases requires close collaboration between the rheumatologist and the oncologist.

## Conclusion

In conclusion, this consensus report provides a comprehensive, practical, and evidence-based framework to guide rheumatologists in Türkiye through the complex intersection of IA and malignancy. Its goal is to standardize clinical decision-making, enhance clinical safety, and ultimately achieve the optimal treatment balance for patients.

### Footnotes

### Authorship Contributions

Concept: H.B., G.S., E.T., P.A.D., S.Ç., İ.V., B.Ö.U., D.B.G., R.Y., N.Ş.Y.B., T.K., B.B., İ.E., Ö.D.A., G.G.Ö., C.B., E.D.E., Design: H.B., G.S., E.T., P.A.D., S.Ç., İ.V., B.Ö.U., D.B.G., R.Y., N.Ş.Y.B., T.K., B.B., İ.E., Ö.D.A., G.G.Ö., C.B., E.D.E., Data Collection and Processing: H.B., G.S., E.T., P.A.D., S.Ç., İ.V., B.Ö.U., D.B.G., R.Y., N.Ş.Y.B., T.K., B.B., İ.E., Ö.D.A., G.G.Ö., C.B., E.D.E., Analysis or Interpretation: H.B., G.S., E.T., P.A.D., S.Ç., İ.V., B.Ö.U., D.B.G., R.Y., N.Ş.Y.B., T.K., B.B., İ.E., Ö.D.A., G.G.Ö., C.B., E.D.E., Literature Search: H.B., G.S., E.T., P.A.D., S.Ç., İ.V., B.Ö.U., D.B.G., R.Y., N.Ş.Y.B., T.K., B.B., İ.E., Ö.D.A., G.G.Ö., C.B., E.D.E., Writing: H.B., G.S., E.T., P.A.D., S.Ç., İ.V., B.Ö.U., D.B.G., R.Y., N.Ş.Y.B., T.K., B.B., İ.E., Ö.D.A., G.G.Ö., C.B., E.D.E.

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