**Olgu Sunumu** / Case Report
Aegean J Med Sci 2019;3:137-139

# A Psoriatic Arthritis Patient Presenting With Carotid Artery Thrombosis

Karotid Arter Trombozu ile Prezente olan Bir Psöriatik Artrit Olgusu

Efdal Akkaya[1], Feyza Nur Çetinkaya[2], Gülin Morkavuk[1], Alev Leventoğlu[1]

1 Neurology Department, Ufuk University, Medicine Faculty, Ankara/Turkey

2 Physical Medicine & Rehabilitation Department, Ahi Evran University, Medicine Faculty, Kırıkkale/Turkey

## ÖZET

*Psöriatik artrit, psöriazisli hastaların yaklaşık %14-30'unda görülebilen inflamatuvar bir artrit çeşididir. Bu inflamasyon, vasküler hasara yol açarak serebrovasküler hastalık ile sonuçlanabilir. Bu makalede psöriatik artritli, karotid arter trombozu ile prezente olan 40 yaşındaki bir erkek olgu sunulmuştur.*

*Anahtar Kelimeler: psöriatik artrit, inme, serebrovasküler hastalık*

## ABSTRACT

*Psoriatic arthritis is an inflammatory arthritis that can be observed in %14-30 of patients with psoriasis. So that this inflammation could also be responsible for vascular damage which could end up with cerebrovascular diseases. We report a 40 year-old patient with carotid artery thrombosis who had been diagnosed with psoriatic arthritis..*

*Keywords: psoriatic arthritis, stroke, cerebrovascular disease*

**Introduction**

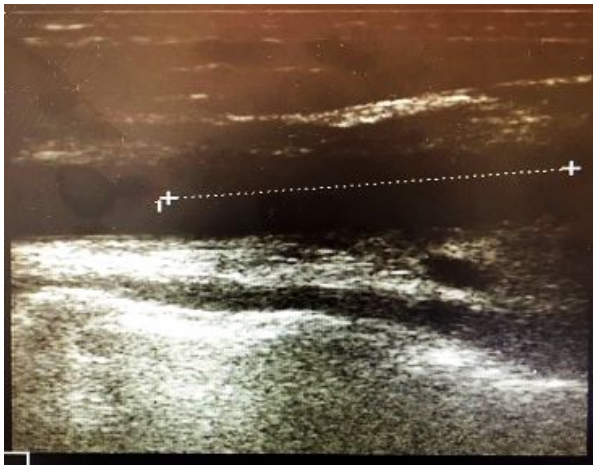
Psoriasis is an immune-mediated systemic inflammatory disease that affects approximately %2-3 of the society. Psoriatic arthritis (PsA) is an inflammatory arthritis that can be observed in %14-30 of patients with psoriasis and causes significant joint damage and disability and is a well-known comorbidity of psoriasis . In patients with PsA, the risk of cardiovascular and cerebrovascular events and the incidence increase compared to the normal population. Although stroke is rare in early ages, the risk of stroke in early ages has increased in rheumatologic diseases, and psoriatic arthritis is one of the rare causes of this condition (1-2)...

**Case**

Our patient was 40-year-old man with a speech disorder and numbness in the left half of the body lasting for 2-3 days. He had the diagnosis of psoriasis for 17 years and PsA for 7 years. Although he had been recommended methotrexate, then salazopyrin; the patient was not taking medication for six years. In physical examination, there were erythematous and scaly plaques around both knees, elbows. Neurological examination revealed no abnormal findings other than subjective sensory complaints. The full blood count, liver and kidney function tests were normal. Hepatitis and vasculitic markers (ANA, ANCA, anti-ds-DNA, anti SS-A and B, Scl-70, Jo-1 antibodies), thrombophilia panel (Factor V Leiden, MTHFR, Protrombin gene mutations), lupus anticoagulant were negative; protein C and S, antithrombin III were in normal ranges. Sedimentation rate was 40 mm/h (0,01-15 mm/h), CRP 15,3 mg/L (0,01-5 mg/L) and HLA B27 was negative. Cardiac consultation and examination also done. Nothing abnormal were found in echocardiography and rhythm Holter tests. Intramural thrombus reaching a height of 3.5 mm in the crescent-shaped lumen, surrounding the lumen in the medial wall, in the 3 cm vessel segment extending 1 cm proximal to the carotid bulbus in the central part of the left common carotid artery was found in the carotid-vertebral artery Doppler USG (Figure I) and thrombotic changes in the millimetric lumen at the left internal carotid artery dissociation site level were found in the neck CT angiography. Contrast-enhanced brain MRI was normal. The patient was initially treated with low -molecular-weight heparin (LMWH) and subsequent warfarin due to the carotid artery thrombus.

**Yazışma Adresi/Address for Correspondence:** Efdal Akkaya, MD, Ufuk Üniversitesi Tıp Fakültesi Hastanesi, Nöroloji ABD, Mevlana Bulvarı (Konya Yolu) No: 86-88 Balgat, Ankara/Türkiye
**E-Posta/E-Mail:** efdalakkaya@hotmail.com ‖ Tel: +90 553 778 5530
**Received/Geliş Tarihi:** 26 May 2019 ‖ **Accepted/Kabul Tarihi:** 18 Eki 2019

***Figure 1.*** *Thrombus material around the carotid artery lumen is seen in Doppler USG.*



**Discussion**

PsA is an inflammatory arthritis associated with psoriasis and observed in %23-30 of patients with psoriasis (3). The pathology in PsA is synovial cell hyperplasia, increased vascularization, and inflammatory cell migration. The characteristic feature of PsA is the presence of asymmetric arthritis. Distal interphalangeal (DIP) joint involvement distinguish it from other inflammatory arthritis (4, 5). In our patient, polyarthritis involving the DIP joints and asymmetric sacroiliac joint involvement were also present during the initial diagnostic period. There is an increased risk of metabolic syndrome, cardiovascular and cerebrovascular diseases (CVD) in PsA compared to the normal population. The risk of hypertension, obesity, hyperlipidemia, type 2 diabetes mellitus (DM) and cardiovascular diseases was found to be significantly higher in patients with psoriatic arthritis compared to patients with only psoriasis (6). The presence of arthritis is associated with higher inflammatory activity and increased disease severity in patients with psoriasis. This inflammatory process is the cause of the onset of endothelial damage. Inflammation plays a role at all stages of atherosclerosis, starting from fatty streaking to plaque formation. Inflammation markers (C-reactive protein- CRP, TNF-alpha) in the plasma are associated with stroke . The presence of antiphospholipid antibody also increases the risk of thrombus . In our patient, erythrocyte sedimentation rate (ESR) and CRP were high, but antiphospholipid antibody screening was negative. Endothelial damage results in cerebrovascular and cardiovascular disease. Ischemic, hemorrhagic or silent vascular damage (white matter hyperintensities) is observed in many rheumatic diseases as well as in PsA patients, its prevalence has increased compared to the normal population, and it has been associated with high inflammatory activity (2,7) This inflammatory activity leads to endothelial damage and constitutes the first step in the development of CVD and atherosclerosis. The risk of increased cardiovascular complication in PsA is associated with the increased intima-media thickness, which is regarded as the beginning of subclinical atherosclerosis. In their study carried out on carotid artery intima-media thickening, carotid total plaque area (TPA) that can be used as a prognostic marker for stroke and vascular events, Lihiedr et al. found that sedimentation rate and PsA duration in patients with PsA were associated with TPA.

We considered our patient's complaints as somatic problems because of normal MRI findings and the side discordance of body and carotid thrombosis. Besides normal brain imaging, the carotid thrombus was founded incidentally as an important cerebrovascular complication of that systemic inflamatory disease. Hyperlipidemia, diabetes mellitus, hypercoagulability syndromes, trauma, aging, smoking or any other factor that affects the vessel walls like systemic inflamatory diseases could cause carotid thrombosis. In treatment anticoagulation is preferable; in some cases surgical procedures could be performed (8).

Because of our patient's young age, we couldn't find any other reason for thrombosis formation: no cardiac reasons, no smoking history, no family history of CVD or cardiovascular disease, no hyperlipidemia and no hypertension. So we considered the reason for that CVD as PsA after other causes have been ruled out. Carotid thrombosis may cause distal embolization and ischemic attack if left untreated. So that we used low -molecular-weight heparin (LMWH) and subsequent warfarin due to the carotid artery thrombus. During hospitalization period, our patient's complaints regressed. We also talked with psychiatry specialist but they didn't offer any medication for somatic signs.

Antirheumatic treatment, changes in lifestyle and the reduction of risk factors are effective in decreasing the risk of CVD. It was observed that the methotrexate used in patients with PsA decreased the incidence of severe vascular events due to its anti-inflammatory and

antithrombotic effects. Non-steroidal anti-inflammatory drugs are frequently used because of their symptomatic benefits, but they do not have therapeutic features. Methotrexate, sulfasalazine, leflunomide, and antimalarials of disease modifying agents (DMARD) can be used alone or in combination. In patients without response to DMARD treatment, TNF-alpha blockers in biological agents, etanercept, infliximab, and adalimumab are among the treatment options (9-11). In our patient, we also started methotrexate 15 mg/week and folbiol 5 mg/ day as disease modifying drug at the beginning...

**Conclusion**

In conclusion, systemic inflammation in the pathogenesis of PsA can cause many inflammatory comorbidities like CVD. Therefore, the presence of systemic effects, the formation of other comorbid conditions and their treatment should be done early and effectively in following PsA patients. It should be taken into account that systemic treatments such as methotrexate and TNF-alpha inhibitors may decrease cardiovascular and cerebrovascular risk factors by reducing inflammation, especially in patients with severe disease activity.

Yazarlar arasında çıkar çatışması yoktur.

The author declares no conflict of interest.

Finansal Destek: yoktur / Funding : none

*doi: https://doi.org/10.33713/egetbd.570346*

**References**

**1.** Takeshita J, Grewal S, Langan SM, Mehta NN, Ogdie A, Van Voorhees AS, et al. Psoriasis and comorbid diseases: Epidemiology. J Am Acad Dermatol. 2017 Mar;76(3):377-390.

**2.** Wiseman SJ, Ralston SH, Wardlaw JM. Cerebrovascular Disease in Rheumatic Diseases: A Systematic Review and Meta-Analysis.Stroke. 2016 Apr;47(4):943-50.

**3.** Gottlieb A, Korman NJ, Gordon KB, Feldman SR, Lebwohl M, Koo JY, et al. Guidelines of care for the management of psoriasis and psoriatic arthritis: Section 2. Psoriatic arthritis: overview and guidelines of care for treatment with an emphasis on the biologis. J Am Acad Dermatol 2008; 58:

851-64.

**4.** Gladman DD, Anhorn KAB, Schachter RK, Mervart H. HLA antigens in psoriatic arthritis.

J Rheumatol 1986;13:586-2.

**5.** Bruce IN. Psoriatic arthritis: clinical features. In: Hochberg MC, Silman AJ, Somolen JS, Weinblat ME, Weisman MH, eds. Rheumatology. 3.ed. Mosby: Elsevier Limited; 2003. p. 1241-52.

**6.** Janıce A. Husted, Arane Thavaneswaran, Vınod Chandran, Lıhı Eder, Cheryl F. Rosen, Rıchard J. Cook, et al. Cardiovascular and Other Comorbidities in Patients With Psoriatic Arthritis: A Comparison With Patients With Psoriasis.

**7.** Wiseman S, Marlborough F, Doubal F, Webb DJ, Wardlaw J. Blood markers of coagulation, fibrinolysis, endothelial dysfunction and inflammation in lacunar stroke versus non-lacunar stroke and non-stroke: systematic review and meta-analysis. Cerebrovasc Dis. 2014;37:64–75.

**8.** Buscaglia LC, Macbeth A. Bilateral carotid artery thrombosis in a young man. J Vasc Surg. 1993 Apr;17(4):790-3.

**9.** Lihi Eder, Ying Wu, Vinod Chandran, Richard Cook, Dafna D Gladman. İncidence and predictors for cardiovascular events in patients with psoriatic athritis. Ann Rheum Dis. 2016 Sep;75(9):1680-6.

**10.** Lihi Eder, Jai Jayakar, Sutha Shanmugarajah, Arane Thavaneswaran, Daniel Pereira, Vinod Chandran, et al. The burden of carotid artery plaques is higher in patients with psoriatic arthritis compared with those with psoriasis alone. Ann Rheum Dis. 2013 May;72(5):715-20.

**11.** Yi-Ying Chin, Hsin-Su Yu, Wan-Chen Li, Ying-Chin Ko,Gwo-Shing Chen, Ching-Shuang Wu, et al. Arthritis as an important determinant for psoriatic patients to develop severe vascular events in Taiwan: a nation-wide study. J Eur Acad Dermatol Venereol. 2013 Oct;27(10):1262-8..