Peripheral Corneal Thinning as an Initial Manifestation of Undiagnosed Rheumatoid Arthritis: Case Report

Tanı Konulmamış Romatoid Artritin İlk Bulgusu Olarak Görülen Periferal Korneal İncelme

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ABSTRACT Periferal corneal thinning may be associated with a variety of autoimmune diseases and in some cases, it may be the first manifestation. Rheumatoid arthritis (RA) is a chronic, systemic inflammatory disorder that may affect many tissues and organs including peripheral cornea. A case of previously undiagnosed (RA) is reported in a 60 year old patient who has peripheral corneal thinning that was detected on routine eye examination. Due to hand joint restrictions and peripheral corneal thinning, the patient was consulted with rheumatology clinic for a newly presented RA. Many patients with ophthalmic involvement in autoimmune disorders experience ocular sequelae during the course of undiagnosed immunological disorders. Ophtalmologists should therefore raise awareness about corneal manifestations of these diseases for early detection and treatment.

Key Words: Cornea; arthritis, rheumatoid; corneal perforation

ÖZET Periferal korneal incelme birçok otoimmün hastalık ile ilgili olarak görülebilmekte ve bazen bu hastalıkların ilk bulgusu olarak ortaya çıkabilmektedir. Romatoid artrit (RA) kronik, sistemik inflamatuar bir hastalık olup periferal kornea dahil birçok doku ve organı etkileyebilmektedir. Rutin göz muayenesi sırasında periferal korneal incelme tespit edilen 60 yaşında bir hastada görülen daha önce tanı konulmamış bir RA vakası bu makalede rapor edilmiştir. Hastanın el eklemlerinde hareket kısıtlılığı olması ve periferal korneal incelme görülmesinden dolayı, hasta tanı konulmamış RA hastalığı şüphesi ile romatoloji kliniğine yönlendirilmiştir. Otoimmün hastalıklarda göz tutulumu olan birçok hasta henüz tanı konulmamış dönemde oküler bulgular gösterebilmektedir. Bu yüzden göz doktorlarının bu hastalıkların erken tanısı ve tedavisi için göz bulguları konusunda dikkatli olmaları gerekmektedir.

Anahtar Kelimeler: Kornea; artrit, romatoid; korneal perforasyon

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Rheumatoid arthritis (RA) is a chronic, systemic inflammatory disorder that may affect many tissues and organs. The pathology of the disease process often leads to the destruction of articular cartilage. Although the cause of RA is unknown, autoimmunity plays a pivotal role in both its chronicity and progression.[1] RA is associated with a number of extra-articular organ manifestations including ocular manifestations such as keratoconjunctivitis sicca, episcleritis, scleritis, corneal changes, and retinal vasculitus.[2-4]

RA can be difficult to diagnose. Many other conditions resemble it and its symptoms can develop insidiously. Blood tests and x-rays may show normal results for months after the onset of joint pain. When RA is clinically suspected, immunological studies are required, such as testing for the presence of rheumatoid factor (RF). During the first year of illness, RF is more likely to be negative with some individuals converting to seropositive status over time. RF is also seen in other illnesses, therefore the test is not very specific. Because of this low specificity, new serological tests have been developed, which test for the presence of the anti-citrullinated protein antibodies or anti-cyclic citrullinated peptide (anti-CCP).[3,5-7]

We report a case of periferal corneal thinning in a diabetes mellitus patient who has a previously undiagnosed RA.

CASE REPORT

Written informed consent was obtained from patient. A 60-year old man with type 2 diabetes mellitus presented with periferal corneal thinning in his last routine examination at ophthalmology clinic. Slit-lamp biomicroscopic examination showed a peripheral corneal thinning in right eye for nearly 360 degrees circumferentially inside the limbus with a well-demarcated central edge (Figure 1, 2). At examination, corrected visual acuities were 10⁄10 in the right eye and 10⁄10 in the left eye. The patient also defined restrictions of movement and limitation of physical activity especially in the mornings at small joints of his hand in detailed medical history. Due to hand joint restrictions and peripheral corneal thinning, the patient was consulted with rheumatology clinic for a newly presented RA. The results of antibody test showed negative RF and positive anti-CCP. The patient was tested for newly presenting RA according to the 2010 American College of Rheumatoloy/Eeuropean League Against Rheumatism classification criteria (4-10 small joint involvement – 3 points, high positive anti-CCP – 3 points, duration of symptoms > 6 weeks – 1 point) and classified as definite RA.[8]

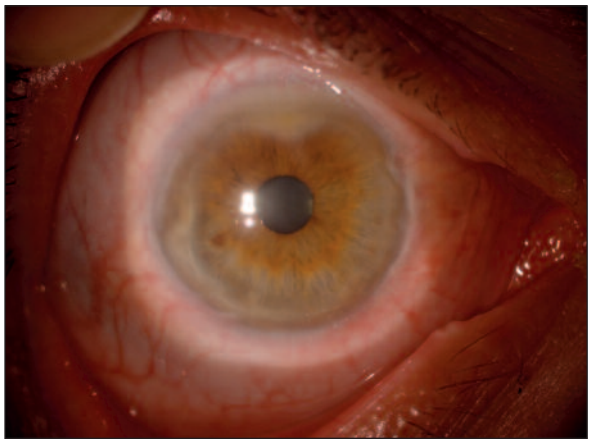

FIGURE 1: Nearly 360° peripheral corneal thinning of right eye.
(See for colored form http://oftalmoloji.turkiyeklinikleri.com/)

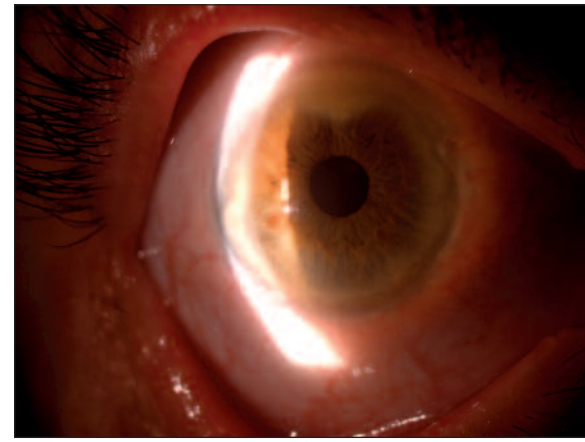

FIGURE 2: Slit lamp photograph of corneal thinning.
(See for colored form http://oftalmoloji.turkiyeklinikleri.com/)

Rheumatologist prescribed methotrexate, at 15 mg/week, plaquenil, at 200 mg/day and folbiol, at 10 mg/week. Due to risk of scleromalacia perforans and not having severe arthralgia corticosteroid treatment not started. We prescribed artificial tears to patient for every 1 to 2 hours while awake to lubricate the eye and continue to routine follow up.

After topical and systemic treatment for six months ophthalmologic examination showed no decline at corneal thinning. Patients complaints about restrictions of movement and limitation of physical activity also decreased during this period.

DISCUSSION

RA is a common disorder that is affecting peripheral cornea. The peripheral cornea has distinct morphologic and immunologic characteristic that predispose it to inflammatory reactions. Unlike the central cornea, the peripheral cornea is closer to the limbal conjunctiva where capillary beds provide both nutrients, and immunocompetent cells.[9]

Many patients with ophthalmic involvement in autoimmune disorders experience ocular sequelae during the course of undiagnosed immunological disorders. Reduced tear secretion, chronic inflammation of the ocular surface, as well as ultrastructural abnormalities are believed to play a major part in decompensating the ocular surface.[10]

The differential diagnosis of corneal thinning and perforation involve disease processes such as infection, inflammation, trauma, and degeneration. Besides RA, any of the collagen vascular diseases, such as Wegener's granulomatosis, systemic lupus erythematosus, polyarteritis nodosa, and relapsing polychondritis, can predispose people to peripheral corneal thinning.[11]

Regardless of the cause of the epithelial defect, if re-epithelialization does not occur, the potential for corneal thinning and perforation exists. Treatment is directed toward healing of the epithelial defect, inhibiting stromal melting, and stimulating stromal fibroplasia. Treatment firstly includes non-preserved artificial tears every 1 to 2 hours while awake to lubricate the eye. A simple and effective measure to promote re-epithelization of the cornea is a pressure bandage. Soft contact lenses used as a bandage are a significant advance in the treatment of corneal thinning. Patients with pain or photophobia might benefit from cycloplegic drops such as 1% atropine twice daily. When other methods have failed mucous membrane graft or conjunctival flap are effective methods for management of corneal thinning.[12]

In the present case periferal corneal thinning detected during routing follow up examination and detailed medical history helped us at diagnosing newly presented RA. RA is typically a progressive illness that has the potential to cause joint destruction and functional disability. Early treatment of RA results in better outcomes, for this reason it is important for disease management to diagnose and treat people with RA as early as possible.[13]

Anti-CCP is a new and important serologic diagnostic marker for the diagnosis of RA and anti-CCP could be detected in almost 70% of the patients with recent-onset RA at baseline visit. Given the presence of autoantibodies can precede the clinical manifestation of RA by many years.[14] In this case although the RF was negative, the anti-CCP result was positive.

Ophtalmologists can encounter patients with peripheral corneal thinning and this symptom may be the first detected manifestation of RA. In patients with periferal corneal thinning if a history of joint pain or syptoms suggesting RA exists, ophtalmologist should consult these patients with rheumatology clinic for early diagnosis and treatment of RA.

REFERENCES

1. Scott DL, Wolfe F, Huizinga TW. Rheumatoid arthritis. Lancet 2010;376(9746):1094-108.
2. Coskun M, Akal A. [Ocular findings in osteoporosis and rheumatismal diseases]. Dicle Med J 2009;36(4):329-32.
3. Başarer T, Yiğitsubay U, Cicik E, Sakallı R. [Ocular complications in rheumatoid arthritis]. Turkiye Klinikleri J Ophthalmol 1998;7(1):42-7.
4. Goronzy JJ, Weyand CM. Developments in the scientific understanding of rheumatoid arthritis. Arthritis Res Ther 2009;11(5):249.
5. Shidara K, Inoue E, Tanaka E, Hoshi D, Seto Y, Nakajima A, et al. Comparison of the second and third generation anti-cyclic citrullinated peptide antibody assays in the diagnosis of Japanese patients with rheumatoid arthritis. Rheumatol Int 2011;31(5):617-22.
6. Heidari B, Abedi H, Firouzjahi A, Heidari P. Diagnostic value of synovial fluid anti-cyclic citrullinated peptide antibody for rheumatoid arthritis. Rheumatol Int 2010;30(11):1465-70.
7. Kaşifoğlu T, Korkmaz C. [Diagnosis and differential diagnosis of rheumatoid arthritis]. Turkiye Klinikleri J Int Med Sci 2006;2(25):35-40.

8. Britsemmer K, Ursum J, Gerritsen M, van Tuyl LH, van Schaardenburg D. Validation of the 2010 ACR/EULAR classification criteria for rheumatoid arthritis: slight improvement over the 1987 ACR criteria. Ann Rheum Dis 2011;70(8):1468-70.
9. Murgova S, Balabanov C. Corneal melting. J Biomed Clin Res 2010;3(1):59-64.
10. Squirrell DM, Winfield J, Amos RS. Peripheral ulcerative keratitis 'corneal melt' and rheumatoid arthritis: a case series. Rheumatology (Oxford) 1999;38(12):1245-8.
11. Pleyer U. Immunobiology of the cornea. In: Pleyer U, Zierhut M, Behrens-Baumann W, eds. Immuno-Ophthalmology. 1st ed. Basel: Karger Pub; 1999. p.167-87.
12. Ma'luf RN, Awwad ST. Mucous membrane graft versus Gunderson conjunctival flap for fitting a scleral shell over a sensitive cornea. Ophthal Plast Reconstr Surg 2005;21(5):356-8.
13. Visser H. Early diagnosis of rheumatoid arthritis. Best Pract Res Clin Rheumatol 2005; 19(1):55-72.
14. Ateş A, Karaaslan Y, Aksaray S. Predictive value of antibodies to cyclic citrullinated peptide in patients with early arthritis. Clin Rheumatol 2007;26(4):499-504.