Coll. Antropol. **25** Suppl. (2001) 67–70
UDC 617.731-005.4:616.72-002.77
Original scientific paper

Anterior Ischaemic Optic Neuropathy in Patient with Rheumatoid Arthritis – Case Report

S. Perić[1], B. Cerovski[1] and P. Perić[2]

[1] Clinical Hospital Center Zagreb, Department of Ophthalmology, Zagreb, Croatia
[2] Clinical Hospital Center Zagreb, Department of Rheumatology, Zagreb, Croatia

A B S T R A C T

This case report presents a patient with long-lasting rheumatoid arthritis (RA) of fourth clinical grade, having ocular complications. RA was diagnosed according to current modified ARA criteria from 1987. Upon admission to the Department of Ophthalmology clinical examination revealed anterior ischaemic optic neuropathy (AION), which is not characteristic manifestation of RA in the eye. The occurrence of AION in patients with RA has been explained in literature as a secondary manifestation of hypertension in these patients or, by the presence of other connective tissue disease apart from RA (for example, MCTD – mixed connective tissue disease). Both mentioned causes were excluded in our case, as well as any other condition that could lead to AION. Therefore, we had concluded that AION presented a late complication of RA.

Introduction

Anterior ischaemic optic neuropathy (AION) is caused by luminal narrowing of blood vessels of the optic nerve head, which, consequently, leads to impaired blood flow and various degrees of optic nerve ischemia.

The term AION has been used in literature since 1975, and it has been described in many systemic diseases (for example, in ischemic cardiac diseases, atherosclerosis, cerebrovascular diseases, arterial hypertension and diabetes mellitus), including also some of the autoimmune diseases[1–4]. In AION circulation is compromised, mainly because of the changes in short posterior ciliary arteries, leading to ischaemic necrosis of the optic nerve papilla with the development of papillary edema[5].

AION is not frequently found in patients with rheumatoid arthritis[6]. If it is the case, vasculitis is said to be secondary to concomitant hypertension or a result of

Received for publication June 4, 2001.

connective tissue diseases other than rheumatoid arthritis[6].

Data from literature suggest that retinovasculitis and angiopathy are uncommon findings in active rheumatoid arthritis. Henkind, in these cases, holds that a patient either has concomitant hypertension or some connective tissue disease other than rheumatoid arthritis[6].

On post-mortem examinations approximately 25% of patients with rheumatoid arthritis were found to have vasculitis. Vasculitis is much more common in male patients with RA than in female patients, with duration of more than 10 years and high titer of rheumatoid factor in serum.

Case Report

R.K., a 52-year-old housewife, presented at the Department of Ophthalmology, Clinical Hospital Center Zagreb in 1997. with classical rheumatoid arthritis lasting for 15 years. Several exacerbations of polyarthritis had developed in the last few years of the disease. A month previously she had developed last flare of the disease. She was continuously treated with low doses of corticosteroids (prednisolone), sulfasalazine and metotrexat in doses customary for treatment of RA. She came to our unit suffering from a nonpainful loss of vision in the left eye, which had developed two days previously.

Physical examination showed signs of subacute generalized polyarthritis, especially of hands and feet, with characteristic deformation of almost all joints, typical for the last grade of RA. Examination revealed subcutaneous nodules of knees and elbows with no signs of vasculitis or other extra-articular manifestations of disease.

Findings:

Right eye status: V = 0.8, normal anterior chamber finding. Ophthalmoscopy revealed pale optic nerve papilla with narrowing of the vessels. Left eye status: V = 0.05, normal anterior chamber. Ophthalmoscopy showed pale optic nerve papilla of indistinct margins with small areas of effusion.

Fluorescein angiography- left eye: increased native fluorescence of the optic disc, registered hypoperfusion of the optic nerve. Goldmann visual field – left eye: I_1, I_2, not registered, I_3, I_4 considerably narrowed. Goldmann visual field – right eye: I_1 not registered, I_2, I_3, I_4 concentrically mildly narrowed.

These are the results of tests conduced during the exacerbation of patient's arthritis and while the patient was on Prednisolone tablets 10 mg/day, Sulfasalazine tablets 2 g/day and Metotrexat 7.5 mg/week.

E.S.R. 33 mm/first hour, RBC 4.79×10^{12}/l, Hemoglobin 85 g/l, Leukocytes: 5.9×10^9/l, Platelets: 358×10^9/l. Total protein 72.0 g/l, Serum albumin 34.8 g/l, Waaler-Rose titer 1:512, Latex RF titer 1:320, ANF negative. Immune complexes and complements were normal.

After short-term high-dose steroid therapy polyarthritis rapidly subsided, as well as patient's left eye vision. The Goldmann visual field, on discharge from hospital, was improved and isopters were well recovered. The absence of I_1 and I_2 isopters persisted.

Discussion

Fundus lesions are usually not found in direct relation with rheumatoid arthritis. In cases of vasculitis or retinopathy patients have either concomitant hypertension or some connective tissue disease other than rheumatoid arthritis[6]. Both mentioned causes were excluded in our case, as well as any other condition that could lead to AION.

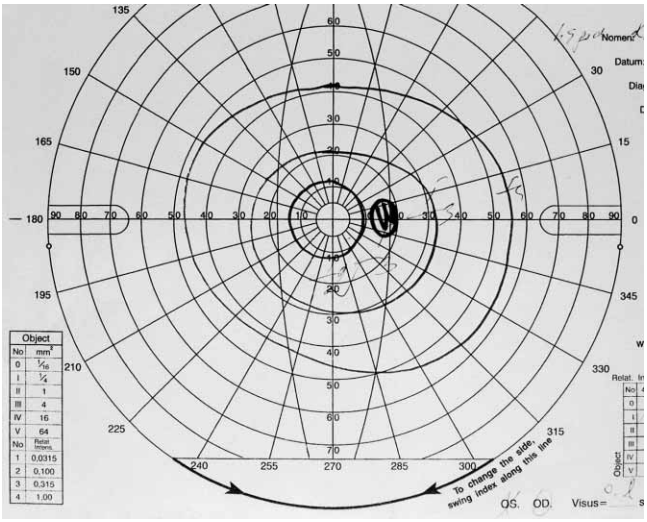

Fig. 1. Right eye – visual field.

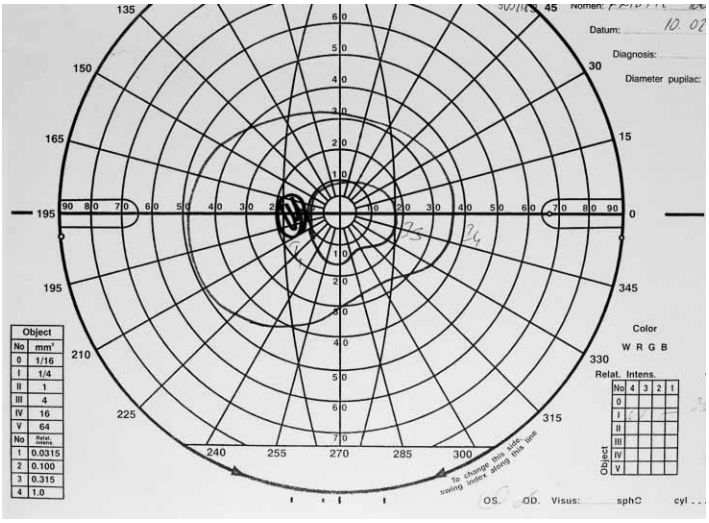

Fig. 2. Left eye – visual field.

Holborow reports on rheumatoid vasculitis closely associated with the presence of circulating IgM rheumatoid factor, but less closely with rheumatoid factor titre[7], although Watson and Hazelman say that arterial involvement occurs only in strongly positive patients[8]. Our patient presented with high Waaler- Rose titer of 1 in 512 and had subcutaneous nodules, which are more common in patients with RA who developed vasculitis.

In literature we didn't find any other case report on the correlation between rheumatoid arthritis and anterior ischaemic optic neuropathy in patients who were normotensive and had neither clinical or immunological manifestation of other connective tissue disease. Therefore, we concluded that AION was a late complication of RA.

REFERENCES

1. HAYREH, S. S.: Anterior ischaemic optic neuropathy. (Springer Verlag, New York, 1975). — 2. PAVAN-LANSTON, D.: Manual of ocular diagnosis and therapy. (Little, Brown and Co., Boston, 1996). — 3. ANDREWS, B. S., J. MCINTOSH, V. PETTS, R. PENNY, Clin. Exp. Immunol., 29 (1977) 23. — 4. CEROVSKI, B., J. ŠIKIĆ, T. VIDOVIĆ, I. EKERT, M. HULJEV, M. TOJAGIĆ, L. BOJIĆ, Neurol. Croat., 46(3–4) (1997) 91. — 5. SPRAUL, C. W., J. AMANN, G. E. LANG, G. K. LANG, Ophtalmologe., 93 (1996) 354. — 6. HENKIND, P., Trans. Ophtal. Soc. UK, 94 (1974) 785. — 7. HOLBOROW, E. J., Trans. Ophtal. Soc. UK., 94 (1974) 712. — 8. WATSON, P. G., B. C. HAZELMAN: The sclera and systemic disorders. (Saunders, London, 1976).

S. Perić

Clinical Hospital Center Zagreb, Department of Ophthalmology, Kišpatićeva 12, 10000 Zagreb, Croatia

PREDNJA ISHEMIČKA NEUROOPTIKOPATIJA U BOLESNIKA S REUMATOIDNIM ARTRITISOM – PRIKAZ SLUČAJA

SAŽETAK

U radu je prikazana bolesnica s dugogodišnjom anamnezom reumatoidnog artritisa (RA) IV završnog, kliničko-radiološkog stupnja s očnim komplikacijama. RA je utvrđen prema postojećim modificiranim ARA kriterijima iz 1987. godine. Tijekom hospitalizacije i opservacije u Klinici za očne bolesti postavljena je dijagnoza prednje ishemičke neurooptikopatije (AION), koja nije karakteristična manifestacija RA na očima.

Pojava AION-a u sklopu RA u literaturi se tumačila kao sekundarna pojava popratne hipertenzije ili prisutnošću neke druge bolesti vezivnog tkiva osim RA (npr. miješane bolesti vezivnog tkiva). Opservacija je isključila postojanje oba entiteta i preostalih mogućih uzroka AION-a. Stoga je pojava AION-a protumačena kao kasna komplikacija RA.