



ISCHAEMIC OPTIC NEUROPATHY IN RHEUMATOID ARTHRITIS (CASE REPORT)

W. A. Sultan

Physical Medicine and Rehabilitation Dept. Mansoura University

F.E Bruckner

Rheumatology Department, St. George's Hospital, London.

M. B. R Mathalone

Ophthalm. Dept., St. Stephen's Hospital, London.

T. E Gamal

Ophthalm. Dept, Mansoura University

Follow this and additional works at: <https://mmj.mans.edu.eg/home>

Recommended Citation

sultan, W. A.; Bruckner, F.E; Mathalone, M. B. R; and Gamal, T. E (1990) "ISCHAEMIC OPTIC NEUROPATHY IN RHEUMATOID ARTHRITIS (CASE REPORT)," *Mansoura Medical Journal*: Vol. 19 : Iss. 1 , Article 17.
Available at: <https://doi.org/10.21608/mjmu.1990.138853>

This Original Study is brought to you for free and open access by Mansoura Medical Journal. It has been accepted for inclusion in Mansoura Medical Journal by an authorized editor of Mansoura Medical Journal. For more information, please contact mmj@mans.edu.eg.

ISCHAEMIC OPTIC NEUROPATHY IN RHEUMATOID ARTHRITIS (CASE REPORT)

By

sultan, W. A. ¹; Bruckner, F.E.²; Mathalone , M. B. R³
and Gamal, T. E.⁴

From

¹ Physical Medicine and Rehabilitation Dept. Mansoura Univ.

² Rheumatology Department, St. George's Hospital, London.

³ Ophth. Dept., St. Stephen's Hospital, London.

⁴ Ophth. Dept., Mansoura University.

* Received For Publication : 2 / 4 / 1990

Although generalised vasculitis is well recognised in rheumatoid arthritis, ischaemic optic neuropathy secondary to this appears to be quite rare . We describe a patient with this condition.

Case Report :

Mrs. L. B. was a 58 years old housewife with a 5 years history of arthritis and early morning stiffness of 3 - 4 hours . In March 1982 she was found to have active polyarthritis affecting mainly the hands, right wrist and right shoulder , and rheumatoid oedema of the feet. Her blood pressure was 140/80. ESR was 65 mm in the first hour.

Rheumatoid factor was positive in a titre of 1 : 80.

X-rays of the hands and feet showed periarticular osteoporosis and

marginal erosions. A diagnosis of rheumatoid arthritis was made and she was treated with ketoprofen 100 mg twice daily. In May 1982 she suffered a severe exacerbation of her arthritis and at the same time developed sudden impairment of vision in the right eye so that she was able only to count fingers in the upper field. On general examination there were no nail fold lesions, skin rash, episcleritis or sub cutaneous nodules.

Examination of the eyes : On the right side , the disc was not swollen but it was pale with very narrow vessels (Fig. 1) and there was an afferent pupil defect.

Examination of the asymptomatic left eye showed a normal disc (Fig. 2) and an appearance like a venous infarct in the lower temporal quadrant

(Fig. 3). There was no uveitis, episcleritis, or scleritis, and tear production was only slightly diminished in either eye. The discs appeared normal on fluorescein angiography and there was no leakage of dye or hypofluorescence. A diagnosis of right ischaemic optic neuropathy was made.

She was treated with prednisolone 30 mg daily, azathioprine 100 mg daily and continued on ketoprofen.

During the next two months the patient's vision in the right eye improved to 6/60 and there was moderate improvement of her joints. As the dose of prednisolone was reduced, however, the patient's general condition deteriorated with increasing pain and swelling in most joints and ESR rising to 95 mm in 1 hour. In January 1983, azathioprine was stopped and she was started on D-Penicillamine which was slowly increased to 500 mg daily. Her arthritis gradually settled over the next 12 months. There was, however, little further change of vision in the right eye and the pupil defect remained. Other causes of ischaemic optic neuropathy were excluded by negative tests for antinuclear antibodies to rule out systemic lupus erythematosus, normal blood glucose to exclude diabetes and normal blood urea and serum creatinine to exclude renal disease. There was no clinical evidence of polyarteritis nodosa, scleroderma or dermatomyositis and temporal arteritis were normal. She remained normotensive.

There have been two previous reports in the English medical literature of fundus lesions in rheumatoid arthritis due to ischaemic retinopathy. The first case reported by Meyer et al. in 1978 was of a 67 years old woman who had seropositive erosive rheumatoid arthritis. She developed bilateral impairment of vision and pericarditis during a flare up of her arthritis. Fundal examination showed scattered pericapillary cotton wool spots but surprisingly the optic discs and retinal vessels were normal on fluorescein angiography. More recently Crompton et al. (1980) reported the second case in a seropositive rheumatoid female patient aged 68 who became blind in the left eye from ischaemic papillitis, and two weeks later developed a transient field defect in the other eye. The patient died two months later from a myocardial infarction. Fluorescein angiography of the blind eye showed a pale swollen optic disc with haemorrhage and irregular narrowing of the arterioles. Post mortem exami-

nation showed necrotising and lymphocytic vasculitis of posterior ciliary arteries in both eyes, loss of ganglion cells in the left retina and atrophy of the left optic nerve. There is a single report of central retinal vein thrombosis in a male patient aged 41 who had seronegative rheumatoid arthritis, Cushing's disease and IgA deficiency Andrews et al. (1977). In contrast to the rarity of this finding in the English literature, a Russian study Tupikin and krikanov (1972) used ophthalmoscopy and fluorescein angiography of the eye to examine 70 patients with rheumatoid arthritis. The arthritis was active in 53 patients and quiescent in the remaining 17. They found that "retinovasculitis and angiopathy were common with rheumatoid arthritis in the active phase", but they did not give the exact figures.

Our patient's visual loss was due to an ischaemic optic neuropathy. Although this is quite unusual, fluorescein angiography was normal in the affected eye, as in Meyer's visual loss was due to an ischaemic optic neuropathy. Although this is quite unusual, fluorescein angiography was normal in the affected eye, as in Meyer's et al., (1978) case. It is likely that the acute episode was over by the time the patient was seen, so that the disc swell-

ing and leakage of dye had subsided. Though there were no other stigmata of a generalised rheumatoid vasculitis, this was nevertheless the most probable underlying condition, in view of the concomitant exacerbation of her arthritis, the rheumatoid oedema, seropositive erosive disease and the absence of any other recognised cause.

Eye vasculitis occurs in other connective tissue diseases. In systemic lupus erythematosus the incidence of fundus lesions is reported to be relatively common (Lanham et al., 1982). Retinal microvascular abnormalities can be shown in apparently normal fundi using fluorescein angiography. Edmonds et al. (1975) thought that this technique might be important in assessing disease activity in patients with suspected cerebral lupus. In scleroderma, the generalised vascular abnormality is suggested as a primary factor in the pathogenesis of the disease Grannan and Forrester (1977) attribute ischaemic papillitis to this vasculitis. However, hypertension may be a contributory cause (Ashton et al., 1968). Ischaemic fundus lesions are found in polyarteritis nodosa (Manjani 1967), giant cell (temporal) arteritis, and dermatomyositis (Calamia and Hunder, 1980).

The eye is commonly affected in

It seems likely that, in generalised rheumatoid vasculitis, the fundus is affected more frequently than is usually thought. As loss of vision is irreversible in this condition, it should be actively sought in those patients at risk.

SUMMARY

Fundus lesions in rheumatoid arthritis due to vasculitis affecting the eye are rare. Our patient was a 58 years old female who presented with seropositive, erosive rheumatoid arthritis. During a period of exacerbation of her arthritis, she developed impairment of vision in the right eye due to ischaemic optic neuropathy and the most likely cause of this was rheumatoid vasculitis. The disc was pale with narrow vessels. She was treated with corticosteroid and immunosuppressive drugs but there was very little improvement of her vision. Other causes such as malignant hypertension, diabetes mellitus, systemic lupus erythematosus, polyarteritis nodosa, progressive systemic sclerosis, dermatomyositis and temporal arteritis were excluded.

General speaking rheumatoid vasculitis is of two types: one shows fibrinoid necrosis of the vessel wall and the other simple arteritis with cellular infiltration within the vessel wall but without necrosis Schmid et al. (1961) have proved in this case to be the possible cause of optic atrophy.

Rheumatoid vasculitis is frequently associated with the evidence a high concentration of circulating immune complexes which may have a fundamental role in the pathogenesis of vasculitis (Zvaifler, 1973).

It has been reported that rheumatoid vasculitis plays a key role in the pathogenesis of the rheumatoid node, nail fold lesions, neuropathy Chamberlain and Bruckner, (1970) and scleritis and episcleritis Jayson & Jones (1971).

(Hazleman and Watson, 1977).

or in association with scleritis ulceration or keratitis may occur alone associated. On the other hand, corneal uveitis and conjunctivitis are rarely associated. While main ocular complications are keratoconjunctivitis sicca and scleritis, while adult rheumatoid arthritis and the two

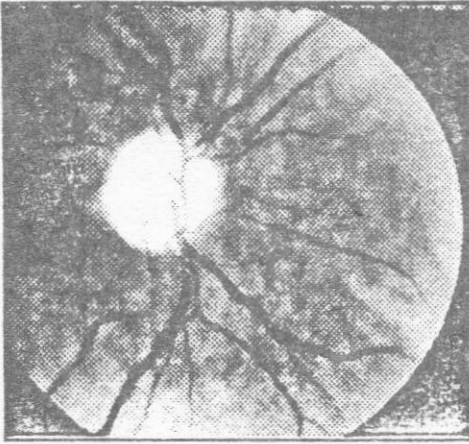


Fig. 1. Rt. optic atrophy (pale disc, narrow vessels).

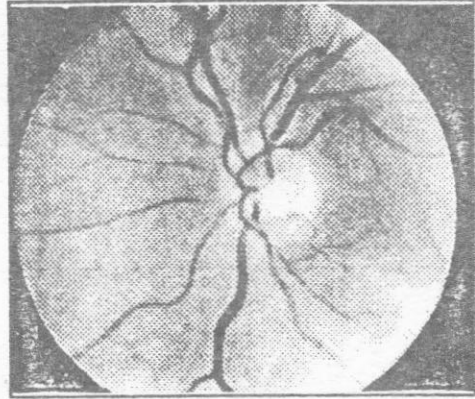


Fig. 2. Lt. normal disc.

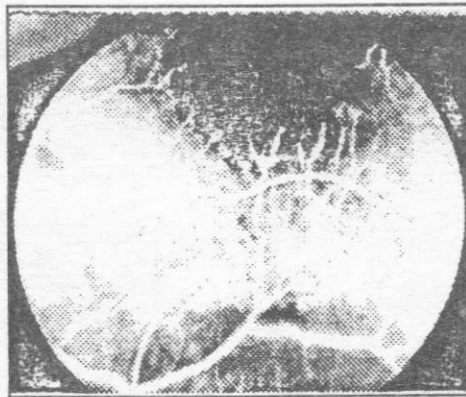


Fig. 3. Lt. lower temp. venous infarct.

REFERENCE

- Grannan DM and Forrester J. Ann. Rheum. Dis., 1977: 36 : 152.
- Ashton N, Coomes EN, Garner A, and Oliver DO. J. Path. Bacter., 1968 : 96 : 259.
- Manjiani MR, Brit. J. Ophthal., 1967: 51 : 696.
- Calamia KT, and Huder GG. Clinis in Rheumatic Diseases, 1980 : 6; 2; 389.
- Hazleman BL and Watson PG. Clin. Rheumatic Dis., 1977 : 313; 501.
- Chamberlain MA, and Bruckner FE. Rheum. Dis., 1970 : 6090.
- Jayson MIV and Jones DEF. Ann. Rheum. Dis., 1971 : 30 : 343.
- Zvaifler NJ. Advances in Immunology, 1973 : 16 : 265.
- Schmid FR, Copper N , Ziff M and MeEwen C. Am. J. Mad., 1961 : 30. 543.
- Edmonds JP, Bruneau C. and Hughes GRV. Ann. Rheum. Dis., 1975 : 34 : 473
- Lanham JC, Barrie T, Kohner EM and Hughes GRV. Ann. Rheum. Dis., 1982 : 41 : 12 : 37
- Tupikin CVand Krikanov VP. Voprosy. Revmatizma. 1972 : 12 : 37
- Andrews BC, McIntosh J, Pett V and Penny R. Clinical and Experimental Immunology, 1977 : 29 : 23.
- Crompton JI, Lye P, Begg MW. Australian. J. Ophthal. 1980 : 8 : 219;
- Meyer E, Scharf J, Miller B et al. Ann. Ophthal. 1978 : 10: 1583,

الملخص العربى

تندر أصابة قاع العين فى التهاب المفاصل الروماتويدى بسبب التهاب الاوعية الدموية بالعين.

كانت هذه الحالة لمريضه تبلغ من العمر ٥٨ عاما تشكو من روماتويد تأكلى بالمفاصل مع نتائج إيجابية لتحليل عينات الدم.

وفى فترة من تهيج التهاب المفاصل إشتكت المريضة من ضعف فى أبصار عينها اليمنى بسبب التهاب بالعصب البصرى نتيجة عجز الدورة الدموية به وكان السبب المقبول لهذا هو التهاب روماتويدى بالاعوية الدموية .

كان القرص البصرى باهتا مع ضيق الاوعية الدموية ، وقد عولجت المريضة بمشتقات الكورتيزون والأدوية المهيطة للمناعة ، ولكن لم يحدث التحسن طفيف فى الأبصار .

وقد عملت لها فحوص معملية كثيرة أثبتت عدم وجود أى من الأمراض الأتية :
ارتفاع ضغط الدم - مرض البول السكرى - مرض الذئبة الحمراء - الألتهاب الشريانى المتعدد
العقدى - التصلب العضوى المتزايد - الالتهاب الجلدى العضى ، والتهاب الشريان المعبدى .

