To the Editor,

Eosinophilic granulomatosis with polyangiitis (EGPA) is a necrotising vasculitis involving the medium-to-small blood vessels. Ophthalmological involvement in EGPA is rare\(^1\). Previous case reports of ocular involvement in EGPA were attributed to retinal vasculitis, retinal artery or vein occlusion\(^2\)–\(^4\), temporal artery involvement\(^5\) or occipital lobe infarction\(^6\).

Blindness caused by anterior ischemic optic neuropathy (AION) was reported in several cases. We report a case of sequential ocular involvement in AION secondary to EGPA and was treated with rituximab.

A 59-year-old Chinese lady presented to our hospital with painless right ocular blindness and progressive weakness of the lower limbs. The condition worsened despite a course of intravenous (IV) methylprednisolone from another institution, a week prior to this admission. The weakness was associated with numbness at glove and stocking distribution. The most significant past medical history was late onset bronchial asthma two years prior to this acute presentation. She also had persistent anaemia due to beta thalassaemia, with haemoglobin ranged between 7-8 g/L.

Her physical examination demonstrated features of mononeuritis multiplex, pale right optic disc with blindness, and vasculitic rash of the lower limbs. The nerve conduction test confirmed asymmetrical mixed sensory and motor axonal polyneuropathy, consistent with clinical findings. Blood investigation revealed a significant eosinophilia, elevated ESR and positive c-ANCA and anti-PR3. There was neither renal involvement nor clinical features to suggest giant cell arteritis. Sural nerve biopsy had shown wallerian degeneration. Fortnightly 500 mg of IV cyclophosphamide was initiated and followed by 40 mg (1 mg/kg) of oral prednisolone.

Unfortunately, the patient developed blurriness of vision on the left eye, which could happen in the arteritic type of AION. Her visual acuity was 6/60 despite on high dose glucocorticoid and after two doses of IV cyclophosphamide. In view of her rapid deterioration while on standard treatment, IV rituximab (1000 mg x 2) was added as a salvage therapy. She was later maintained with azathioprine. Rituximab and azathioprine had provided steroid sparing effect as we were able to taper down her steroid gradually to 7.5 mg without any new symptoms or signs. However, she demonstrated little therapeutic response with marginal improvement of visual acuity of the left eye and a non-reversible blindness on the right. The numbness continued with mild improvement in the motor component of the peripheral nervous system.

**REFERENCES**


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**Rituximab is not useful in bilateral ocular involvement caused by eosinophilic granulomatosis with polyangiitis**

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