“Sudden Painless Visual Loss”
In Acute Medicine - Not “Giant Cell Arteritis”, What Is It?

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Introduction
In Acute Medicine, ischemic optic neuropathy is the commonest cause of sudden painless visual loss. It is categorized as anterior (AION, affecting the optic disc, swollen disc) versus posterior (PION, affecting retrobulbar optic nerve, no changes on fundoscopy) and as arteritic (GCA) versus nonarteritic (cardio-embolic). Non-arteritic AION is by far the commonest type.

Case Report
A 67 year old gentleman with a background of asthma, presented with sudden visual loss in his left eye. He had been having a constellation of symptoms for the last 3 months: proximal muscle pains, paraesthesias in both hands, loss of manual dexterity, night sweats and weight loss. On examination he was cachectic and pyrexial (39.3°C). Temporal arteries were pulsatile and nontender. Mild weakness of hip flexors and small muscles of the hands were elicited. Left eye examination revealed no perception of light, relative afferent pupillary defect and fundoscopy showed swollen optic disc (Figure 1). Bloods showed marked eosinophilia, raised inflammatory markers, positive p-ANCA. CT head demonstrated fluid in paranasal sinuses.

Discussion
Churg Strauss Syndrome (CSS) or allergic granulomatosis is a systemic necrotising vasculitis characterized by peripheral neuropathy, pulmonary involvement, paranasal sinus abnormalities and eosinophilia. CSS is associated with a number of ocular manifestations including granulomatous conjunctivitis, retinal vascular occlusions, cranial neuropathy and ischemic optic neuropathy.1 So far, 5 cases of Churg Strauss Syndrome have been reported who have developed visual loss due to ischemic optic neuropathy. Another case of asymptomatic AION has been reported. It is concluded that immunosuppressant therapy may halt or reverse the visual loss in CSS associated with optic neuropathy.1 Our patient was commenced on intravenous methyl prednisolone and cyclophosphamide. At 3 months, his vision had minimally improved to PL (perception of Light) and he developed optic atrophy (Figure 2) but all his other symptoms had improved remarkably.

Conclusion
In summary, our patient fulfilled the clinical criteria of CSS2 (Table 1), based on presence of eosinophilia, asthma, polyneuropathy and paranasal sinus abnormality. Prompt diagnosis and initiation of the treatment is vital to avoid complications.

References

Table 1

<table>
<thead>
<tr>
<th>Criteria for diagnosis of Rheumatology (4 out of 6 criteria)</th>
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<tbody>
<tr>
<td>Asthma</td>
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<tr>
<td>Eosinophilia &gt;10%</td>
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<tr>
<td>Mononeuropathy or Polyneuropathy</td>
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<tr>
<td>Non-fixed pulmonary infiltrates</td>
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<td>Paranasal sinus abnormalities</td>
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<td>Presence of extravascular eosinophils on biopsy</td>
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</tbody>
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Figure 1

Figure 2