fovea, results in loss of stereopsis and sometimes frank diplopia at near; the two eyes cannot fixate on the same near target to achieve binocular single vision.

The adequate performance of a near visual task, such as the reading of a book or the threading of a needle, requires intact convergence. Convergence insufficiency interferes with the performance of such tasks through the disruption of binocular single vision at near. The Titmus stereoacuity test – a book of plates viewed at 40cm distance with polarized glasses – is also a near visual task, allowing quantification of near stereopsis but not distance stereopsis. Because convergence of the eyes is necessary for viewing the Titmus test and other tests of near stereoacuity, CI may interfere significantly with such measurements of stereopsis.

These observations are relevant to the study by Kim et al[1], because CI is significantly more common among PD patients than age-matched controls and correlates with increasing Hoehn and Yahr disease severity[4,5]. In one study, the prevalence of CI among PD patients was 31%, compared to 0% among controls (P<0.001)[5]. Therefore, in the study by Kim et al, it would have been critically important to exclude subjects with CI before comparing the stereoacuity of PD patients to that of controls, especially when studying drug naïve PD patients[1]. It is not clear whether this was done, and therefore the authors' interpretation of their results may be confounded by CI. Because CI is, by definition, a phenomenon that emerges only when viewing a near target, it can easily be overlooked unless specifically sought by: a) examining ocular alignment while the patient views a near target; and b) by measuring the near point of convergence. In fact, ocular alignment and stereopsis may be completely normal when a patient with CI is asked to view a distant target (e.g., a Snellen eye chart or distant fixation light). Although "strabismus" and "ocular motility disturbance" were set as exclusion criteria by Kim et al, none of the seven patients excluded from the study were actually eliminated on these grounds, despite the reported 30% prevalence of CI in PD patients[1].

It would be interesting to repeat the study using a test of distance stereoacuity, which would eliminate altogether the need for intact convergence during stereopsis testing. More robust conclusions about the role of central dopaminergic pathways in stereopsis could then be drawn.

Convergence insufficiency is an underrecognized cause of diplopia and asthenopia in PD patients, often presenting as "difficulty reading" or "tired eyes" when performing near tasks. Symptomatic treatment is easy and generally appreciated by PD patients[5], and it is therefore worthwhile maintaining a high index of suspicion for CI in the PD population. Convergence insufficiency in some PD patients may respond to levodopa[6], but usually CI must be corrected optically using base-in prisms, which are typically either affixed onto or ground into the patient's reading glasses by an optometrist or orthoptist.

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To the Editor

Isolated Recurrent Monocular Vision Loss as a Presentation of Temporal Arteritis

A 73-year-old gentleman was referred to emergency department by his family physician because of a one week history of recurring episodes of monocular vision loss. The episodes were painless and involved the entire visual field of the left eye. Although the episodes had only begun a week ago, they were increasing in frequency and duration. At the time of initial assessment in the emergency department, he estimated approximately eight to ten similar episodes over five to seven days, each lasting anywhere from 10 to 60 seconds in duration before resolving completely without any residual deficits. He denied any other unusual signs, symptoms or focal neurological

deficits during the episodes or in between the episodes. He also denied any associated headache, neck pain, jaw pain or jaw claudication.

He denied any obvious precipitating factors for the onset of episodes. He had no significant medical concerns and was on no medications other than aspirin, which was started earlier that week by his family physician when the episodes started. At that time, a referral to stroke neurology was also made, but the accelerating pattern of the episodes necessitated more urgent assessment and investigation. A full functional inquiry revealed no other symptoms and no systemic symptoms apart from some non-specific and diffuse joint aches for several years.

On examination, the patient was afebrile and blood pressure and heart rate were within normal limits. Head and neck exam was normal and there was no scalp tenderness and no

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abnormalities of the superficial temporal artery noted. Fundoscopy was normal. Cardiac exam revealed normal heart sounds. Neurological examination was completely normal.

Initial investigations revealed a mildly elevated elementary sedentary rate (ESR) of 34 mm/h and a mildly elevated C-reactive protein (CRP) of 48.6 mg/L. Other laboratory investigations were normal. A CT head was performed and was normal as well.

The patient was admitted to the stroke neurology service and an urgent carotid ultrasound was arranged. In the interim, he was loaded with clopidogrel at 300 mg and kept on dual anti-platelet therapy until ultrasound could rule out the possibility of left sided carotid artery disease as the source of his events. Despite the dual antiplatelet therapy, the patient continued to have episodes including a prolonged episode lasting three to five minutes. The carotid ultrasound was completed emergently and showed absolutely no abnormalities within the carotid arteries. The only abnormalities on investigations were the ESR and CRP which rose further to 39 mm/h and 59.4 mg/L respectively, while the episodes continued to occur.

Despite the lack of other classic features, concern over the possibility of temporal arteritis (TA) emerged and the patient was started on intravenous methylprednisolone 1 gram per day and sent for an urgent opthomology consultation and left temporal artery biopsy (Figure). The biopsy confirmed inflammatory changes within the tunica media and adventitia as well as infiltration of all layers of the artery by lymphocytes and plasma cells consistent with a diagnosis of TA.

The patient was maintained intravenous methylprednisolone at 1 gram per day for five days and then transitioned to oral prednisone 60 mg per day. The episodes had stopped completely after the second day of treatment with intravenous methylprednisolone and both the ESR and CRP had decreased
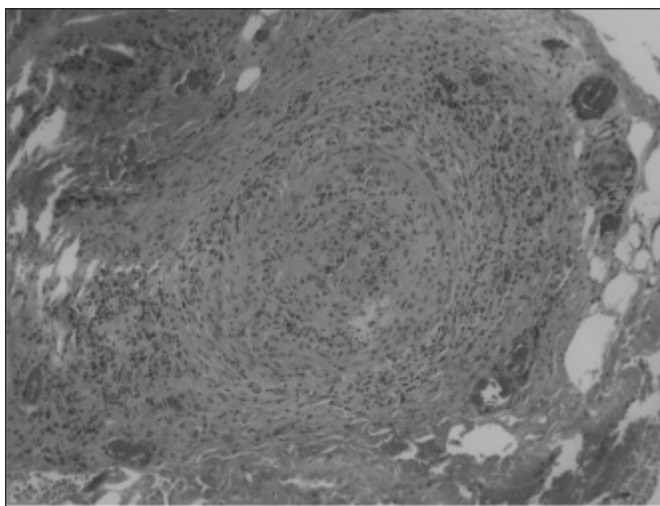

Figure: Biopsy of temporal artery. Inflammatory changes are evident in the tunica media and adventitia of the temporal artery. More extensive inflammation of the surrounding arteries is also present including lymphocyte and plasma cell infiltration of all layers.

Table: Differential diagnosis of TMVL

A) Vascular
1) Arterial
    i) Ischemia/Embolism/Coagulopathy
    ii) Vasospasm/Vasculitis(GCA)
    iii) Retinal Migraine
2) Venous
    i) Chronic Retinal Vein Occlusion(CRVO)
B) Ocular Diseases
1) Anatomic
    i) Anterior Segment
      -Dry Eyes, Hyphema, Keratoconus,
       Acute Closed Angle Glaucoma
    ii) Retinal Detachment
2) Hypoperfusion/Ocular Hypoperfusion
C) Optic Nerve/Disc Disorders
1) Transient Visual Obscuration
    i) Pappilledema
    ii) Optic Disc Drusen
    iii) Congenital optic disc anomalies
    iv) Compressive lesions of the intraorbital optic nerve
2) Demyelinating disease

to normal levels by the last day of intravenous methyl-prednisolone treatment. The oral prednisone was continued at the same dose for two weeks, followed by 10 mg decrements every two weeks until 40 mg per day. The dose was then decreased by 10% every two weeks until a once daily dose of 10 mg and then decreased to 5 mg after two weeks and then discontinued two weeks later. The patient tolerated the titration without any difficulty or recurrence of symptoms. He continues to be followed in the stroke clinic and is maintained only on daily aspirin. Because he has not had any recurrence of symptoms, initiation of long-term immunosuppressive medication was deferred in favour of observation.

DISCUSSION

Transient monocular visual loss (TMVL) is a common presentation encountered by primary care physicians, in the emergency department, and by consulting neurologists. The first challenge in the approach to this presentation is establishing, on history and physical exam, whether the vision loss was in fact monocular versus binocular and the differentiation is crucial for localization and differential diagnosis. (See Table) When visual loss is monocular, it implies pathology anterior to the chiasm, in either the optic nerve, the eye itself or the vasculature to either structure. A thorough ocular exam, including fundoscopy and examination of the anterior chamber, should be conducted to detect primary ocular causes. When primary eye pathology is not suspected or is ruled out, an important consideration is the possibility of an ischemic, particularly embolic etiology. A diagnostic work-up for sources of emboli, namely carotid plaque

or cardiac sources, is imperative. If carotid ultrasound rules out significant ipsilateral carotid artery disease as a potential source for emboli, cardio-embolic source for stroke should be investigated with electrocardiogram and in some circumstances echocardiography and holter monitor.

The urgency in ruling out proximal sources of emboli rests in the potential to prevent vision loss and future stroke from further emboli. Following an event, certain patient groups incur considerable benefit from timely interventions; large prospective studies have shown that early intervention in patients following transient ischemic attack can reduce stroke incidence by up to 80%, and in those with carotid stenosis (70-99%) identified on the side of their symptoms, a recent meta-analysis reported an ARR of 16% with early referral for endarterectomy; the latter highlighted the importance of early intervention, showing the greatest benefit in those treated early compared to those with delayed treatment. Cleary, secondary prevention can be successfully offered to patients identified as having a transient ischemic attack, with the prompt and accurate determination of cause and the aggressive and timely response.

Once an embolic phenomenon has been thoroughly ruled out, other causes of ischemia should be considered, as these too may progress without treatment; the timely pursuit of further investigations remains essential to prevent potentially devastating consequences including permenent visual loss.

Temporal arteritis deserves particular attention in this regard; early clinical suspicion is critical, as features of presentations and histories are highly variable and physical exam is often unremarkable or non-specific; a JAMA review deemed the history a poor tool to rule out the diagnosis, and found the most useful physical exam finding (temporal artery abnormality) to be only 65% sensitive, indicating a substantial number of patients with underlying TA in the absence of these signs.[1] Perhaps more importantly, if instituted in a timely manner, intervention is effective in preventing the progression of permanent visual loss. Classic symptoms of TA include headache, jaw claudication, scalp tenderness, visual disturbances and constitutional symptoms; as a constellation, these features are likely to trigger appropriate clinical suspicion. When visual loss, a common disease manifestation, occurs in isolation, it may present a diagnostic challenge. Although its pathophysiology is not completely elucidated, visual loss is believed to result from inflammatory changes in the walls of affected arteries, intimal thickening, and resulting narrowing of the vessels, predisposing to thrombosis and ischemia downstream of the arterial lesion, which may be transient or permanent. Irreversible blindness results from anterior ischemic optic neuritis. Cohort and population-based studies of patients with biopsy-proven TA have reported TMVL incidences ranging from 15- 50%. Of those who ultimately progress to permanent visual loss, half had experienced episodes of transient visual loss prior to its occurrence.[2]

The American college of rheumatology published diagnostic criteria for TA in 1990, which define a case of TA as having at least three of the following five features: age >/= 50, new headache, ESR >/= 50, temporal artery abnormality on physical exam, and temporal artery biopsy consistent with the diagnosis. Limitations of these criteria when applied in clinical settings have been demonstrated, and their utility is now widely accepted to be confined to research.[3] Experts have recognized the spectrum of presentations and the implications of delaying diagnosis. For example, the British society for rheumatology recently published practice guidelines which urged clinicians to respond to a broad range of suggestive symptoms, even when isolated, with prompt initiation with corticosteroid therapy and TA biopsy; when a negative biopsy is obtained in the setting of ongoing clinical suspicion, a contralateral biopsy should be obtained.[4] In these settings, a response to therapy, namely a resolution of the presenting symptoms with the initiation of therapy, should support suspicion of the diagnosis, while continuing symptoms once therapy has been started strongly suggests an alternative diagnosis.

Review of the literature suggests that prior to the use of corticosteroids, blindness complicated the course of up to 60% of TA patients[2]; with the introduction of these agents as the mainstay of therapy, the incidence has decreased to approximately 20%[2,5]. While it is clear that we have improved outcomes dramatically, the diagnosis and treatment of TA in atypical cases is still likely missed if physicians rely too heavily on classic descriptions of a highly variable disease entity, or good criteria in the wrong context rather than clinical judgement in the right setting. The potential for better outcomes still exists. We propose that patients with recurrent TMVL and a negative work-up for embolic sources be presumed TA until proven otherwise. This should involve urgent temporal artery biopsy, and initiation of steroid therapy while biopsy results are pending. With the availability of effective therapy, increased indices of suspicion and lower thresholds for initiating empiric treatment, the number of cases of TA suffering consequences of vision loss from missed or delayed diagnosis will decrease even further.

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