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Case Report

Acute diplopia associated with systemic hypertension - A case Report

***[1]Augustus Onyeka Ezenwankwo and [2]Bernadine N Ekpenyong**

[1]Benita Eye clinic, Calabar, Cross River State, Nigeria
[2]Department of Public Health, University of Calabar, Cross River state, Nigeria

*Corresponding author E-mail: onyeka.ezenwankwo@gmail.com, benyital2001@yahoo.com Phone: +234-818-309-6264

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Abstract

Background: Microvascular ocular cranial nerve palsy can result from diabetes and hypertension and may be increasing with the increase in the incidence of diabetes and hypertension in Nigeria. Objective: This study provides clinical considerations for the neuro-ophthalmic evaluation of diplopic patients with presumed microvascular ocular cranial nerve palsy. Method: A case report is presented of a forty two (42) years old man recently managed for hypertensive emergency and was referred to our clinic following sudden onset of diplopia. A review of the neuro-ophthalmic evaluation of microvascular ocular cranial nerve palsy is presented. Result: Examination of the patient revealed a right acute esotropia. No other neuro-ophthalmic sign was found. Pupils and visual fields were normal. Resolution of diplopia was gradual and complete three weeks post presentation. A diagnosis of a presumptive microvascular right abducens nerve palsy associated with systemic hypertension was made. Literature reveals microvascular ocular cranial nerve palsy to be the most common cause of acute diplopia especially in the older age group. Conclusion: Microvascular diseases can result in acute diplopia. However, because certain sinister pathology such as intracranial neoplasm and aneurysm can present with acute diplopia, neuro-ophthalmic evaluation for red flags such as the presence of multiple palsies, bilateral palsies, pupil involvement, less than forty (40) years, non resolving diplopia and onset of new symptoms/signs should prompt consideration for referral for extensive neuro-diagnostic investigation.

Keywords: Diplopia, cranial nerve palsy, diabetes, hypertension, microvasculature

INTRODUCTION

Acute diplopia represents the onset of diplopia in patients with previously normal binocular vision. Binocular diplopia results from the misalignment of the visual axis of the both eyes. Pelak (2004), identified seven (7) mechanisms involved in the causes of binocular diplopia to include; orbital disorders, extraocular muscle restriction, extraocular muscle weakness, neuromuscular junction dysfunction, palsies of third, fourth, or sixth nerve cranial nerve (CN), brain stem injury to CN nuclei and supranuclear injury (pathways to and between CN nuclei). Ocular CN palsy represents the most common mechanism of acquired binocular diplopia (Nolan, 1968; Morris, 1991; Tiffin et al., 1996; Comer et al., 2007), and accounts for more than 80% of all cases of binocular diplopia in patients 40years and older (Comer et al., 2007). Microvascular neuropathy is presumed to cause ocular CN palsy and accounts for between 59%-86% of all cases of diplopia with a cranio-neuropathic mechanism (Comer et al., 2007; Rush et al., 1981; Chou et al., 2004; Tamhanker et al., 2013).

The association between vascular diseases and ocular CN palsy dates back to the first reported case in 1866 of a patient with CN VI palsy and co-existing diabetes in the absence of any other neuro-ophthalmic or somatic signs and symptoms (Dreyfus et al., 1957).The vasculopathic mechanism which was thought to be responsible was for many decades considered to be largely speculative. Studies abound in literature explaining the role of microvascular diseases in the pathogenesis of ocular CN palsy. Dreyfus et al. (1957), found evidence of CN III infarction in a patient with CN III

palsy but without any evidence of an occluded vessel, but he however postulated that an occluded vessel must have been responsible for the infarction. Asbury et al. (1970), on the other hand reported an 88year old woman with CN III palsy with thickening and hyalination of arterioles and capillaries but without any evidence of an infarction. These two studies seem to complement each other and provide evidence explaining the pathogenesis of CN palsies in patients with vascular diseases. However, because each of these reports are isolated and inconclusive, the diagnosis of microvascular ocular CN palsy is considered presumptive and is based on the presence of co-existing CN palsy and any vascular disease, and the absence of any other neuro-ophthalmic or somatic signs/symptoms.

Microvascular cranial neuropathy typically involves CN III, IV and VI either in isolation or very infrequently as multiple CN palsies. CN VI is the most affected in all cases of isolated microvascular neuropathy, and represents more than 50% of all cases (Tiffin et al., 1996, Comer et al., 2007, Saleh et al., 1999). This is probably due to the long peripheral course of the nerve making it more susceptible to various forms of assaults. The ratio of the involvement between CN III and CN IV is inconsistent and varies across reports.

The prevalence of vascular diseases in ocular CN palsy varies across reports. Most hospital studies reported a higher prevalence of hypertension than diabetes in patients with ocular CN palsy (Murchison et al., 2011; Brinar et al., 2007; Comer et al., 2007), but the study by Saleh et al. (1999), which considered only microvascular CN palsy reported a higher prevalence for diabetes. However, because hospital based studies were inconsistent in the reported prevalence for diabetes and hypertension, Patel et al. (2005) used a population based case control study to determine the magnitude of any pre-existing diabetes and hypertension with isolated CN VI palsy. They found there is a 6-fold increase in odds of having diabetes in cases of CN VI palsy over control, 8-fold increase in odds in cases of diabetes and hypertension co-existing and about 1.62 increase in odds of having hypertension. They concluded that the much cited association of hypertension and CN VI palsy may be coincidental.

World over, diabetes, hypertension and dyslipidemia are major public health concerns. Recent reports on the prevalence of these non-communicable diseases in Nigeria suggest that diabetes, hypertension and dyslipidemia may be approaching epidemic levels. In a review of the trend of non-communicable diseases in Nigeria, Maiyaki et al. (2014), reported a rise in the prevalence of diabetes from 2% in 1992 to about 4.04% in 2011. The prevalence of hypertension was reported to have risen from a prevalence of about 8.6% in the 70s to a pooled prevalence of about 22.5% (2000-2011) (Ogah et al., 2012). Also, in a national review to assess the prevalence of dyslipidemia, dyslipidemia which was reported to have a low prevalence in Nigeria, now have a prevalence of about 60% in 2012 and is similar in all geopolitical zones in Nigeria (Oguejiofor et al., 2012). With the incidence of cranio-neuropathy increasing with the increase in the incidence of these diseases, the eye care provider is expected to witness an increase in the number of patients presenting with sudden onset of diplopia. However, because one cannot expect all cases of non traumatic acute diplopia to be due to microvascular neuropathic mechanism, the clinician must consider the possibility of any sinister pathology such as an intracranial tumour, aneurysm, or other central nervous system pathology or somatic disease, especially as intracranial tumours affecting the visual system had been reported as a major source of malpractice claim involving optometrists in the United Kingdom (Classe, 1993). This study presents a case report and a review of the neuro-ophthalmic consideration for evaluation of cases of sudden non traumatic binocular diplopia.

CASE REPORT

A forty two (42) years old male was referred to our clinic following the sudden onset of diplopia while under admittance and management for hypertensive emergency. Patient reported onset of diplopia for two days that subsequently became constant. Patient reported diplopia to disappear on closing an eye. On further questioning he revealed images to be laterally displaced. Patient history was unremarkable and had never had an eye examination.

Assessment/First visit

Entry VA:	6m	0.4m
OD	6/6	N18
OS	6/6	N18

External Examination: external ocular tissues appeared normal

Cover Test: esotropia @ distance and near

Red Lens: revealed diplopia greater at distance than at near and also greater when OD is abducted than in the primary position. Magnitude of deviation as measured with the red lens using a phoropter:

Esotropia @far = 15^{Δ}
Esotropia @near = 9^{Δ}
Ophthalmoscopy: fundus appeared normal
Subjective refraction:
OU: +0.50 6/5 Add 1.25 N5
Tonometry: NCT @ 3:00pm
OD: 10mmHg
OS: 06mmHg
Blood Pressure: mercury sphygmomanometer
200/120 mmHg
Diagnosis: - Presumptive right CN VI palsy associated with systemic hypertension
- Presbyopia

Management:

-patient was reassured that diplopia will resolve as blood pressure is controlled
-patient was asked to return to his physician since blood pressure is still high despite treatment.
-asked to return to clinic in 1wk.

Follow up visit: 1 week

Patient no longer experiences diplopia @ near but only @ far and on looking to the right
VA: same as on first visit
Blood Pressure: 155/100 mmHg
Red Lens: - esotropia @ far = 7^{Δ}
-esophoria @ near =2^{Δ}
Management: -tab forever vision i bd x 1/12
- to return to clinic in two weeks

Follow up visit: 2 weeks

VA: same as on first visit
Patient no longer experiences diplopia.
Blood pressure: 140/90 mmHg
Red lens: -phoria @ far =ortho
-esophoria @ near = 2^{Δ}
Management:-glasses was dispensed
- Patient told to continue with blood pressure control.

DISCUSSION

Diplopia is a common presenting ophthalmic symptom, and presents a diagnostic dilemma because of the many possible etiologies of acute onset of diplopia. First and foremost, one most determine if the diplopia is monocular or binocular. Binocular diplopia is defined as diplopia that resolves when either eye is closed. Our patient reported resolution of diplopia if he closes either eye. If the patient does not, asking the patient in the consulting room to occlude an eye would reveal if a diplopia is monocular, in which case would cause little worry as the etiology is usually due to an ocular aberration (Pelak, 2004). If it is binocular, further investigation would be required.

When a diplopia has been established to be binocular, the next question to answer is to determine the underlying mechanism of the deviation; is the deviation due to an ocular nerve paralysis (incomitant) or not (comitant)? Simply asking the patient if diplopia gets worse at looking at certain direction(s) could give a clue. But to be certain, we used a red lens on the right eye to dissociate the eye and asked the patient to compare the separation of both images at different directions of gaze. Patient reported the worsening of diplopia on dextroversion (OD abduction), and at far than at near, revealing the incomitancy of the deviation. A comitant deviation in most (but not all) cases would suggest a benign etiology such as accommodative esotropia or decompensated heterophoria. For very rare cases where a comitant deviation may be associated with a neurological disease, the paper by Hoyt et al. (1995) on “Acute onset concomitant esotropia: when is it a sign of a serious neurological disease?” is a classic reference material. If however,

the deviation was established to be incomitant, as observed in this case, an evaluation to determine if the cause of CN palsy is microvascular in which case only close observation may be required or is due to other possible sinister pathology.

Although our patient had isolated CN palsy, occasionally, patients may present with multiple CN palsies. Palsies of all or any two of the three nerves (CN III, IV and VI) can occur at a time, but simultaneous palsies of CN III and CN VI seems to be the most common presentation. Most cases of multiple palsies may have accompanying visual loss/ field defect and usually suggest other possible intracranial or systemic pathology (Saleh et al., 1999). In Tiffin et al. (1996), report of eight (8) patients with non-traumatic multiple palsies, 2 = herpes zoster ophthalmicus (HZO), 2 = brain stem cerebrovascular accidents (CVA), 2 = metastasis, 1 = intravenous aneurysm and 1 = C-C fistula. All patients also had remarkable clinical symptoms/signs. In Nolan (1968), report, in all 6 patients with aneurysm, all but one presented with multiple palsies, while of 3 with CVA, one presented with multiple palsies.

Pupillary function in our case was normal. Pupillary involvement is usually not associated with CN VI and CN IV palsies. Pupil sparing, usually in cases with third nerve palsies, is long considered a safety net in the neuro-ophthalmic evaluation of all patients with ocular CN palsy. Pupil involvement may present as anisocoria, blown out (dilated) pupil or a non-reactive pupil, classical Argyll-Robertson pupil (Whitsell, 1962). Microvascular third nerve neuropathy may or may not be associated with pupil abnormalities. Pupil abnormality occurs in about 25% of diabetic neuropathy. However, aneurysm of the posterior communicating artery (PCA) is the most common cause of CN III palsy associated with pupil involvement and is present in 95% of cases of PCA aneurysm (Trimble et al., 1979). Clinicians must consider the possibility of an aneurysm in all cases of CN III palsies with pupil involvement and should consider referral for imaging studies.

Acute CN palsies can occur at any age group. The consideration for microvascular neuropathy should however begin from about age forty (40) and older as the prevalence of the vascular diseases is very low in those forty (40) years and younger. Brinar et al. (2007), had suggested, that based on etiologies, patients with ocular CN palsies can be divided into three age groups;

- Children – where most causes are either postviral, tumour or trauma
- Young adults aged 20-50 – where the pathology is most difficult to predict
- Patients older than 50 – where microvascular diseases are most common

This suggestion provides a reliable age specific consideration for the causes of acute CN palsy.

Rarely will there be bilateral involvement of a cranial nerve (usually CN VI). Some authors have recommended neuro-diagnostic investigation in all cases of bilateral cranial nerve involvement. Brinar et al. (2007), reported causes in bilateral involvements were aneurysm, cardiovascular-related accidents and arteriovernous malformations, tumours, encephalitis, vasculitis and meningitis. On the contrary, Tiffin et al. (1996), found all cases of bilateral CN VI palsies occurring in patients without prior diagnosis or other signs to be benign, despite a high rate of investigation and reported the rate of recovery for this subgroup to be similar to the rest of the groups. Considering that many isolated case reports of bilateral involvement are due to other etiologies (non microvascular), it may be useful to consider bilateral involvement as a red flag for neuro-diagnostic evaluation.

When isolated CN palsies presents with other clinical signs and symptoms, other etiologies should be considered. Such signs/symptoms may be present at presentation or appear subsequently during observation for recovery. Common symptoms include; severe or persistent pain, headache, claudication, unilateral vesicles, general body weakness, facial palsy, thyroid disease, stroke, and papilloedema (Nolan, 1968; Morris et al., 1991; Tiffin et al., 1996; Saleh et al., 1999).

Mean time for the resolution of microvascular palsy is three (3) months (Nolan, 1968; Comer et al., 2007; King et al., 1995). Diplopia resulting from other etiologies usually take longer time to recover and also tend to reoccur (King et al. 1995). This makes non recovering palsy (>3months) and reoccurrence an indication for neuro-diagnostic investigation. Literature consistently reports CN palsies to spontaneously resolve, but was not so with our case as resolution was gradual with the patient reporting resolution of diplopic symptom at near (one week) before at distance and on dextroversion (three weeks).

CONCLUSION

Many patients presenting with acute non traumatic binocular diplopia, especially in the older than forty (40) years age group will have the microvascular neuropathic mechanism at play in the pathogenesis of the diplopia. Diplopia from vascular diseases will increase as the prevalence of vascular diseases increases in Nigeria. Close observation alone is initially appropriate for patients with neurologically isolated sixth, fourth and pupil sparing third nerve palsies in the presence of vascular diseases such as diabetes, hypertension and dyslipidemia. However the presence of red flags for any sinister pathology should prompt referral for neuro-diagnostic investigation.

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