Optometric Management of a Third Nerve Palsy in a Prison Setting

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Abstract

Third nerve palsy (TNP) is a neuro-ophthalmic condition that is likely to be encountered by optometrists in various clinical settings. It affects extraocular muscles and may impair eyelid and pupil functions to various degrees. Its aetiology in adults is often linked to compressive lesions of the central nervous system or to vascular ischemic causes from systemic disease such as diabetes and hypertension. This case report presents the management of a TNP in a multidisciplinary Canadian prison setting, where the patient population is often underserved. Current practices for neuroimaging cases of TNP in a conventional clinical setting are presented. The challenges and advantages of management of TNP in a prison setting are discussed, highlighting the particularities of caring for the vulnerable patient population in the prison setting.

KEY WORDS:
Third nerve palsy, prison, interdisciplinary care, neuroimaging, underserved population

INTRODUCTION

Third cranial nerve palsy affects extraocular muscles, the eyelid and the pupil, and is encountered by many optometrists. This case highlights the particularities of its management in a multidisciplinary prison setting.

CASE REPORT

A 60-year-old Caucasian male presented to the optometry service of a Canadian medium-security penitentiary after being referred by the prison's mental health facility physician for "reduced and/or double vision".

Upon questioning, the patient mainly complained of blurry vision since having lost his spectacles. He denied the presence of diplopia, pain, headaches and ocular trauma. The patient's responses were somewhat limited by his mental health condition and a speech impediment. He had undergone cataract surgery (2009) and usually wore spectacles to correct compound hyperopic astigmatism (2011). Binocular vision and ocular health were otherwise unremarkable.

His current medical history included type-2 diabetes (1999, recent HbA1c 10.1%), obesity, hypertension, hypercholesterolemia, benign prostatic hyperplasia, diabetic neuropathy, sleep apnoea, stuttering, anaemia, slight intellectual disability and paranoid schizophrenia. He was being treated with gabapentin, olanzapine, valproate, atorvastatin, ezetimibe, furosemide, irbesartan, nifedipine, insulin, ferrous sulfate and terazosin. Neither the family medical history nor the family ocular history were available.

External exam revealed complete ptosis OD, which had not been mentioned by either the patient or a physician. Uncorrected distance visual acuities were OD 6/21 (holding eyelid) and OS 6/12, with inconclusive pinhole testing. Manifest distance refraction showed a stable compound hyperopic prescription, which corrected visual acuities to

OD 6/6 (holding eyelids and OS 6/7.5. Cover test was inconclusive (comprehension/fixation difficult), but ocular alignment by Krimsky reflexes showed a 30 prism dioptre exotropia and 10 prism dioptre hypotropia OD. In the right eye, adduction, depression and elevation showed grade -4 limitations, with full motility OS in all directions. Pupils were equal and reactive to light and accommodation, without afferent pupillary defect. Anterior segment examination revealed complete ptosis OD, but was otherwise unremarkable. Intraocular pressure was 14 mmHg OU. The left intraocular lens had slight posterior capsule opacification. Dilated fundus examination was unremarkable, with no evidence of diabetic retinopathy. Automated visual field testing was not available, and confrontation visual fields were unreliable.

This clinical portrait suggested a complete, isolated, pupil-sparing TNP, supported by the following: unilateral, painless, atraumatic, recent-onset exodeviation and hypodeviation, complete ptosis, age above 50, normal pupillary function and normal function of the fourth and sixth cranial nerves. Diabetes mellitus was considered to be the most likely cause, given the patient's glycaemic profile and well-controlled systemic hypertension. The prison physician was consulted, which prompted a referral for neuroimaging to rule out compressive aetiologies. For the following week, the nursing staff was advised to monitor the patient daily for late-onset right pupil dilation. A follow-up optometry appointment was scheduled three weeks later. New spectacles were ordered, but no diplopia management was deemed necessary from the complete ptosis. The patient was counselled on the importance of glycaemic control and the effects of diabetic eye disease. The prison nutritionist was solicited to reinforce dietary recommendations.

At the follow-up exam, the patient was more alert, which facilitated history-taking. Upon questioning, he denied diplopia and pain, and seemed content with the new correction. Neuroimaging results were not yet available. The patient record showed no changes in pupil function. Corrected visual acuities were OD 6/6 and OS 6/7.5. External exam showed no ptosis. The patient's eyes were now aligned in primary gaze, with a 30 prism dioptre exophoria at distance and near, normal sensory fusion (red filter) and stereopsis. Extraocular motilities were full in all directions. Pupillary reflexes were normal, without anisocoria. A follow-up examination was planned for two months later. The patient was advised to alert the nursing staff of any diplopia, or any changes in vision or pupil/eyelid appearance.

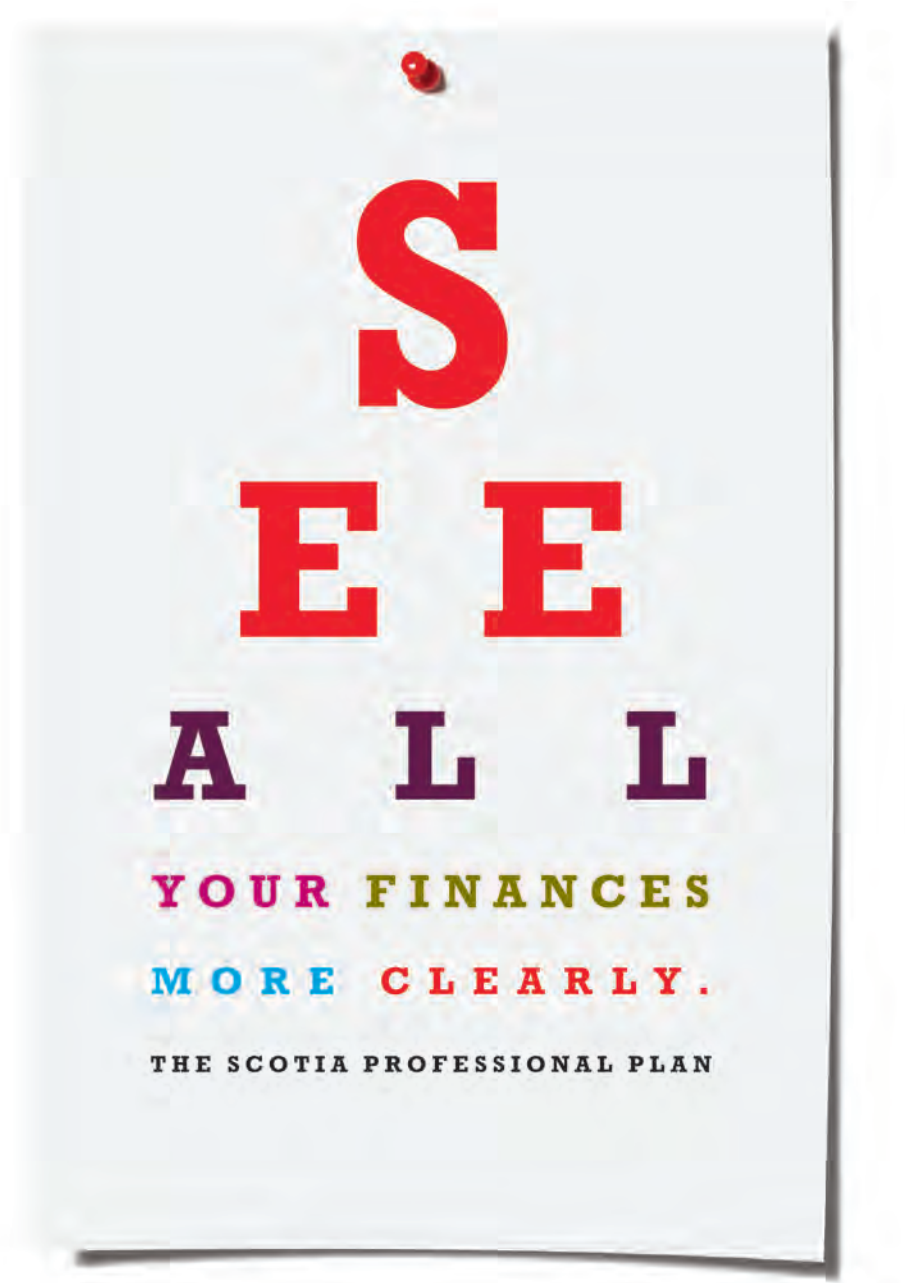
At the two-month follow-up exam, the patient reported no change in his condition. Corrected visual acuities were stable and an external exam was normal. The patient was now orthophoric at distance and near. Extraocular motilities were full in all directions and pupil reflexes were normal. The radiology report included a computed tomography (CT) scan of the head and orbits, with contrast, showing no intracranial lesions or significant anomalies. The patient was educated on his apparent complete recovery and advised to return in two years, in accordance with the Correctional Service of Canada (CSC) policy on the frequency of eye examinations.

DISCUSSION

Clinical presentation and pathophysiology

The third cranial nerve innervates the superior, inferior and medial recti muscles, as well as the inferior oblique and levator palpebrae. Its external and dorsolateral fibres control accommodation and miosis. As such, TNP usually presents with ipsilateral ptosis, hypodeviation with concurrent exodeviation, diplopia, a possible fixed and mydriatic pupil, and blurry vision.[1,2] Complete TNP includes the inability to elevate, depress or adduct the affected eye, along with a complete ptosis. Pupil involvement depends on the aetiology. Partial or incomplete TNP will restrict extraocular motilities to various degrees, impairing one or more extraocular muscles, and cause variable pupil involvement. Partial palsies most often affect either the inferior branch of the nerve (medial rectus, inferior rectus and inferior oblique) or its superior branch (superior rectus and levator).[1,3] Most cases of TNP are unilateral – bilateral cases occur only with central oculomotor nucleus involvement.

Principal aetiologies for TNP include vascular or ischemic causes (diabetes, hypertension, atherosclerotic disease), compression (aneurysm, tumour), trauma and congenital.[1,3,4] Factors that determine the aetiology include pupil involvement, pain, vascular risk factors and age. Mydriasis will typically indicate a compressive lesion.[1,3] As this nerve travels from the midbrain to the orbit through the subarachnoid space, it passes next to the Circle of Willis, close to the posterior communicating artery, where an aneurysm may compress the nerve's external pupillary fibres, resulting in mydriasis and loss of pupil function. Compressive palsies often present with pain or headache.[3,4] Ischemic causes, conversely, will typically only affect internal third nerve fibres, resulting in a painless, pupil-sparing presentation.[4,5] In patients above age 50, TNP is more likely ischemic in nature, where the *vasa nervorum* may suffer ischemia, affecting the main internal fibres that it supplies. Cases under age 50 are more likely to have compressive aetiologies.[1] TNP may be combined with fourth, fifth and/or sixth nerves palsies with lesions affecting the cavernous sinus.[6]



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Differential diagnosis

In this case, differential diagnoses included myasthenia gravis, partial TNP and a pre-existing or decompensated deviation with concurrent ptosis. The latter was excluded based on previous records that showed normal ocular alignment and eyelid function. A partial palsy was excluded based on complete impairment of the eyelid and extraocular muscles. Lastly, ocular myasthenia gravis may present with diplopia and involve extraocular and/or levator muscles. However, muscular imbalances will typically increase with muscle fatigue. Here, it was excluded, since ptosis was full and constant, with both adduction and elevation completely impaired.

Case management

The recovery period for TNP from suspected vascular causes is typically between three and 12 months.[1,3] Its management involves controlling diplopia symptoms, monitoring for pupil involvement, neuroimaging and targeting the underlying systemic condition.[3,4] Diplopia management involves patching or using a frosted lens or Fresnel prisms to minimize symptoms.[3,4] While Fresnel prisms are inexpensive and simple, ordering a frosted lens requires more expense and delays for a likely transient solution. In this case, although history-taking was unreliable, complete ptosis eliminated potential complaints of diplopia, and when ptosis had receded, the patient had also recovered sensory fusion.

A pupil-sparing, presumed ischemic TNP should be closely monitored for pupil involvement during the first week, as some compressive causes initially present with normal pupil function.[1,3,6] Here, this was facilitated by the fact that the nursing staff was present on a daily basis.

Role of neuroimaging

The evidence based on neuroimaging for presumed ischemic TNP in patients over age 50 has evolved over time and is still debated.[7,8] The main issue regards the diagnostic value of neuroimaging to rule out aneurysm or other compressive lesions in such cases. Current practices in ophthalmology have evolved from careful observation (3 to 6 months) to performing magnetic resonance imaging (MRI) and angiography (MRA) for most patients with TNP, to rule out compressive lesions more effectively.[6,9–11] Despite a current lack of high-level evidence (systematic review, meta-analyses or evidence-based clinical guidelines) regarding this matter, prospective studies[10,11] have shown that, in between 14% and 16.5% of patients over age 50 with presumed TNP from vascular aetiologies, TNP was actually due to other causes (compressive lesions, aneurysms, demyelinating disease, etc.), and neuroimaging (CT or most recently MRI and MRA) led to an earlier appropriate management of the underlying cause. However, the low cost-benefit ratio of MRI imaging for all patients can be a limiting factor. Thus, some clinicians may use CT scan and

computed tomography angiography (CTA), which are less costly than MRI but may still detect vascular and compressive lesions.[12] In the present case, the physician's confidence in the likelihood of an ischemic cause lead to CT imaging, likely motivated by cost considerations in a publicly funded context. Careful practitioners should therefore consider ordering MRI (with MRA) for all TNP patients, even when an ischemic aetiology is presumed, as well as for cases resulting from suspected compressive or traumatic causes (pupil involvement [anisocoria greater than 2mm], incomplete motility deficit or under age 50).[9–11]

Implications of optometric practice in a prison setting

Optometry in a prison environment is unfamiliar to many practitioners. In Canada, the CSC contracts optometrists as part of their duty of care to offenders, to maintain ocular health and facilitate daily tasks, education and rehabilitation programs and ultimately social reinsertion. This practice has distinct challenges and advantages.

The prison population is underserved, with a significant burden of disease. High levels of systemic disease and risk factors (diabetes, hypertension, hypercholesterolemia, drug and alcohol use, tuberculosis, hepatitis C, etc.) lead to important amounts of diabetic complications, optic neuropathies, cataracts, retinopathies, etc. Referrals to external specialties (e.g. ophthalmology, neuroimaging) may be subject to limitations (availability of transport, security escorts, etc.). Many offenders are not keen to undertake external trips, since transport conditions involve restraints and uncomfortable vehicles. Since an offender may decline medical care (barring specific exceptions) and outside referrals, disease may progress, potentially increasing morbidity. Due to limited budgetary allowances, an institution of 400 to 500 offenders may have access to one day of optometry clinic per month. The number of patients seen in a day (approximately 6 to 14) is in inverse proportion to the security level of an institution. These factors lead to waiting times of 3-12 months for optometric services.

CSC policies for offenders' access to optometric care are outlined in the CSC's National Essential Health Services Framework.[13] Currently, offenders are allowed one eye examination every 2 years, and spectacle replacement (single vision or bifocal lenses) paid by the CSC every 3 years. Eye examination or follow-up visits can be more frequent upon request by the institution's physician or optometrist, as in this case. Due to the great demand and infrequent supply of optometric services, follow-up of certain conditions is more difficult and results in delaying the monitoring of certain conditions, such as, in this case, the patient's opacification of the left posterior capsule.

The CSC provides examination rooms and ophthalmic equipment. Although equipment rooms are meant to enable an optometrist to perform complete eye examinations, in the author's experience equipment is not distributed equally across institutions. Although basic requirements are usually present (ophthalmic chair, phoropter, autorefractor, slit lamp [with fundus and gonioscopy lenses], tonometer, handheld diagnostic set, trial lens set, binocular vision tests, binocular indirect ophthalmoscope, etc.), imaging equipment (fundus photography and optical coherence tomography [OCT]) is conspicuously absent from institutions. Few institutions have automated visual fields. Although not possible in this case, a visual field assessment would have been beneficial, since a compressive lesion along the visual pathway may be highlighted by various types of scotomata. Requests for additional equipment are possible and may be granted, but the decision rests with the CSC authorities according to budgetary allowances and other competing health specialities. If needed, external referrals may be made to local optometric or ophthalmological practices, although these are subject to the same limitations regarding the availability of transport and security escorts.

The clinical environment of a prison also brings distinct advantages to the optometric clinician. The patient's optometric record is part of the offender's complete health record, which includes medical, pharmaceutical, dental, psychosocial, psychiatric and nutritional charts. This provides privileged access to complementary information and test results that greatly contribute to quality optometric care. Furthermore, the health staff of the facilities consists of nurses, who have enlarged roles similar to nurse practitioners, and who are present every day and often overnight. They are key collaborators in delivering comprehensive care to the patients. The medical doctor at each facility, as well as other professionals (pharmacists, nutritionists), is also accessible for consultation. This accessibility is well illustrated in this case, since co-management by the optometrist and the MD led to neuroimaging, the permanence of the nursing staff allowed daily monitoring of pupil function and many professionals were involved in attempting to improve the patient's diabetes control. ●

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