ORIGINAL ARTICLE

Ocular Motor Cranial Nerve Palsy as an Indicator of Neglected Systemic Disease in Nigeria: Perspective from a Neuro-Ophthalmology Clinic

Olufunmilola A. Ogun^{a,b}, Olalekan O. Aremu^b, and Ayotunde I. Ajaiyeoba^{a,b}

^aDepartment of Ophthalmology, College of Medicine, University of Ibadan, Ibadan, Nigeria; ^bDepartment of Ophthalmology, University College Hospital, Ibadan, Nigeria

ABSTRACT

The aim of this article from Nigeria is to draw attention to public health issues in medical care using ocular motor cranial nerve palsy (OMCNP) presenting to a neuro-ophthalmology clinic as a case study. All patients presenting with OMCNP between November 2007 and October 2016 were retrospectively reviewed. Demographic details as well as information regarding the clinical presentation, clinical course, investigation, and treatment outcomes were extracted from case records. Phone calls were made to the contact numbers of patients who had been lost to follow-up. Data were analysed using SPSS version 22 (IBM, Corp. Armonk, NY, USA). Cranial nerve palsies other than oculomotor, trochlear, and abducens palsies were excluded. Fifty-nine patients, comprising 28 males and 31 females, were identified. Age range was 1–84 years (median 43.0 ± 19.7 years). Fifty-one cases (93.2%) were mono neuropathies, while four (6.8%) affected multiple nerves. Oculomotor nerve palsy was most common, accounting for 57.6% of cases. Microvascular angiopathy was the commonest identifiable cause (25.6%). Aetiology was not identified in 16 cases. There were three (5.1%) deaths. Undiagnosed systemic disease appears to be a major risk factor for this patient group in this African setting. Patient investigation was problematic. Poor patient compliance and follow-up resulted in preventable deaths. Neuro-ophthalmologists practicing in low resource settings should be aware of these risks.

ARTICLE HISTORY

Received 26 August 2018
Revised 14 November 2018
Accepted 30 December 2018

KEYWORDS

Ocular motor cranial nerve palsy; oculomotor nerve; abducens nerve; trochlear nerve; microvascular ischaemia; hypertension; healthcare financing

Background

Although relatively uncommon in general ophthalmology clinics, ocular motor cranial nerve palsies (OMCNPs) are a common presentation in neuro-ophthalmology clinics worldwide.¹ The aetiology of OMCNP depends on the characteristics of the population in question. In addition, clinical presentation depends on the cranial nerve(s) (CNs) involved. Causes of OMCNP include trauma, microvascular ischaemia, nerve compression, inflammation, and demyelination.² Less frequently, congenital CN palsies or agenesis are seen.³ Prompt diagnosis and multidisciplinary care is a key component of assuring a favourable outcome of management. Microvascular OMCNPs often resolve within weeks to months spontaneously, and management consists solely of adequate control of risk factors such as hypertension, diabetes mellitus, dyslipidaemia, alcohol, and smoking.

The current retrospective analysis was conducted to provide data on the common causes of OMCNP within an indigenous African population and to draw attention to public health issues in neuro-ophthalmologic services within the Nigerian context.

Methods

All patients presenting with OMCNP between November 2007 and March 2018 were retrospectively reviewed. Demographic details as well as information regarding the clinical presentation, clinical course, investigation, and treatment outcomes were extracted from case records. Telephone calls were made to contact those patients who had been lost to follow-up. Data were analysed using SPSS version 22 (IBM, Corp. Armonk; NY, USA). CNs palsies other than oculomotor, trochlear, and abducens palsy were excluded. This

study was conducted in accordance with the tenets of the declaration of Helsinki.

Results

A total of 59 patients comprising 28 males and 31 females were seen. Age range was 1–84 years with a median age of 43 years.

Clinical presentation

The most common presentation was headache (25.6%), other presenting complaints included double vision (16.3%), blurred vision (18.6%), and a history of trauma (20.9%). The time lag between first symptoms and date of presentation ranged from 3 days to 10.4 weeks (540 days) with a median of 30 days. The most common time lag between first symptoms and presentation to the neuro-ophthalmology clinic was 14 days (16% of cases). The commonest medical comorbidity found among patients from the history was hypertension in 16.7% of cases. Other comorbidities were diabetes mellitus (2.4%), dyslipidaemia (2.4%), and combined hypertension and diabetes (4.8%). About 31% did not have a significant past medical history. The vast majority of cases were single OMCNPs (93.2%) while only four patients (6.8%) had OMCNP involving multiple CNs. The oculomotor nerve was the most common nerve affected, identified in 34 patients (57.6%). Abducens nerve palsy was identified in 18 cases (30.5%) while trochlear nerve palsy was the least common affecting only 3 (5.1%) patients. Distribution of the OMCNP is shown in Table 1.

Table 1. Distribution of ocular motor cranial nerve palsies in 59 cases.

Cranial nerve involved	No. of cases	Per cent (%)
Oculomotor (CN III)	34	57.6
Abducens (CN VI)	18	30.5
Trochlear (CN IV)	3	5.1
Multiple	4	6.8
Total	59	100.0

The most common identifiable cause of OMCNP was microvascular ischaemia (25.6%). Road traffic accidents, RTA (23.3%), and other forms of trauma (7.0%) also constituted a significant non-medical cause of OMCNP. Space-occupying lesions, SOLs (14.0%), aneurysms (9.3%), infective causes such as meningitis and encephalitis (7.0%), and pseudotumor cerebri (2.3%) were other identified causes. Sixteen (27.1%) missing records could not be analysed for information on aetiology.

Oculomotor nerve (cranial nerve (CN) III)

Causes of oculomotor palsy were microvascular ischaemia (30.8%), SOL (26.9%), RTA (19.2%), aneurysm (15.4%), others (7.6%). Almost two-thirds of the cases of oculomotor nerve palsy (20; 58.8%) were pupil-involved, 12 (35.3%) were pupil-sparing and there was no documentation regarding the pupillary reaction in 2 (5.9%) cases. Pupil involvement in oculomotor nerve palsy was more often associated with the presence of a SOL, trauma, or aneurysm when compared with microvascular ischaemia ($p < 0.0001$) as shown in Table 2.

Abducens nerve (CN VI)

Abducens nerve palsy was found in 30.5% of our patients. Aetiology could not be ascertained in 57.1% of these cases although identifiable risk factors included trauma (14.3%), meningitis (14.3%), pseudotumor cerebri (7.1%), and microvascular ischaemia (7.1%).

Trochlear nerve (CN IV)

Trochlear nerve palsy was found in only three patients (5.1%). Aetiology was unclear in one patient while the other two cases were related to head trauma sustained in RTA.

Table 2. Aetiology and pupillomotor involvement in 32 out of 34 patients with oculomotor (CN III) nerve palsy.

Aetiology of CN III palsy	SOL	Trauma	Aneurysm	Microvascular ischaemia	*Unclear	Total
Pupil involvement	7	6	4	1	2	20
Pupil-sparing	0	1	0	7	4	12
Total	7	7	4	8	6	32

* Denotes a peculiarity of cases in the context of our environment. Aetiology of oculomotor nerve palsy in these cases was unclear based on evidence in the clinical records found. In a number of these cases, further investigation may also have been hindered by the patients' failure to return for follow-up consultations.

Neuroimaging and aetiology

Eighteen patients obtained underwent neuroimaging (30.5%) and cranial computerized tomographic imaging (CT) was the commonest procedure performed in eight (29.6%) of these cases. Magnetic resonance imaging (MRI) was performed in 5 (18.5%) out of 18 cases, while 3 (11.1%) patients each had a combination of either CT followed by MRI or CT followed by MRA. Intracranial SOLs were diagnosed in six (14.0%) cases.

Among the 18 patients who had neuroimaging, intracranial aneurysm was confirmed in only 2 (11.1%) cases. One aneurysm involved the posterior communicating artery, while one patient had aneurysmal dilatations involving the M1 segment of the middle cerebral artery (MCA) and the posterior cerebral artery (PCA), respectively. Both cases of radiologically confirmed intracranial aneurysm (two patients) had presented with headache and pupil-involving oculomotor nerve palsy. One patient (case number 2, with MCA and PCA dilatations) resolved spontaneously but showed persistent signs of aberrant regeneration. Two patients who performed only skull radiographs

were excluded from analysis. Radiological findings are shown in Figure 1.

Risk factors

Blood pressure

Blood pressure (BP) was recorded at presentation for 34 cases (57.6%) and systolic blood pressure (SBP) was >130 mmHg in 16.9% (10 cases) while diastolic BP (DBP) was >80 mmHg in 25.4% (15 cases). When compared across the different OMCNPs, the mean SBP and DBP were highest in patients with oculomotor nerve paresis compared with other OMCNPs with a mean SBP of $139 (\pm 32.4)$ mmHg and DBP of $90 (\pm 17.4)$ mmHg, respectively. The differences were however not statistically significant; SBP ($p = 0.12$) and DBP ($p = 0.08$). Table 3 shows mean SBP and DBP in the different OMCNPs.

Alcohol and smoking

There was no history of significant alcohol or tobacco consumption among all 59 patients.

Figure 1. Radiological findings in 18 patients who had neuroimaging.

Table 3. Mean systolic and diastolic blood pressures (in mmHg) measured in 34 patients with different OMCNPs at presentation.

Type of OMCNP	No. of patients	Mean SBP $\pm$ SD	Range (SBP)	Mean DBP $\pm$ SD	Range (DBP)
Oculomotor	21	139 ± 32.4	100–200	90 ± 17.4	60–120
Trochlear	2	120 ± 14.1	110–130	75 ± 7.1	70–80
Abducens	8	113 ± 10.4	100–130	74 ± 7.4	60–80
Multiple	3	120 ± 17.3	110–140	80 ± 10.0	70–90
Total	34	130 ± 28.8		84 ± 15.9	

Diabetes mellitus

Uncontrolled diabetes mellitus was diagnosed in only three cases. There were no cases of OMCNP diagnosed with controlled pre-existing diabetes mellitus.

Dyslipidaemia

Hypercholesterolaemia and plasma hyperlipidaemia were newly diagnosed in one patient, while investigating the aetiology of OMCNP. Other patients with these risk factors were already on lipid lowering medications at the time of presentation.

Management

Patients with medical comorbidities such as hypertension, diabetes, and dyslipidaemia were referred to the Medical Outpatient department and were managed in conjunction with physicians. Four patients with intracranial SOLs were referred to the neurosurgical clinic. There was a high attrition rate with 41 patients (69.5%) lost to follow-up after the first or second visit and generally within the first 3 months.

Outcome and follow-up

Clinical improvement was observed in 10 (16.9%) patients, who are still attending follow-up clinics. One patient (1.7%) was discharged against medical advice following admission to the trauma ward for repair of a zygomatic fracture. Although the majority

did not return for follow-up and as such improvement or deterioration could not be ascertained from clinic records, telephone calls were made to defaulting patients. Follow-up phone calls revealed that 12 patients (20.3%) had defaulted having experienced improvement in their symptoms, 11 patients (18.6%) did not experience any improvement, while 3 patients (5.1%) had died. Information on outcome could not be obtained on the remaining 22 (37.3%) patients who did not respond to the phone calls. [Figure 2](#) shows the distribution of treatment outcomes.

Mortality

Three patients suffered fatal outcomes: two of these three patients had presented with extremely elevated SBP and DBP (SBP >200 mmHg, DBP >110 mmHg) but absconded following referral to the medical emergency room. The first case fatality (53 year old Female) presented with severely elevated blood pressure and blood glucose but did not accept emergency medical treatment. She reportedly died at home several weeks later. The second case (43 year old Female) presented with severe hypertension and pupil-involving left oculomotor nerve palsy, she did not obtain neuroimaging or emergency medical care, and she reportedly died within 2 weeks of presentation. The third patient (42 year old Female) had presented with a 2-week history of headache and right-sided oculomotor nerve palsy with pupil involvement. She was being investigated for a suspected

Figure 2. Outcomes in 59 patients with OMCNP.

intracranial aneurysm, when she reportedly died at home, suddenly, 4 weeks after her initial eye clinic visit. None of the three cases had a post-mortem examination. In all cases, there was documented evidence of financial constraints hindering investigation and treatment.

Discussion

The distribution and pattern of OMCNP in Ibadan, Nigeria, does not appear to be very different from the pattern described in similar reports from other parts of the world.^{4–14} However, the clinical course, management constraints, and subsequent fatal outcomes are peculiar to the setting. This is an issue of public-health significance, which may reflect the impact of socio-economic factors on health-seeking behaviour of Nigerians.

Distribution of OMCNP

In a review of 4,278 patients at the Mayo clinic, abducens palsy was the commonest OMCNP described in more than one-third of cases.¹⁰ However, aetiology was unclear in over 50% of cases.¹⁰ More recently, a review of 31 consecutive cases of OMCNP in Kinshasa, Democratic Republic of Congo, documented similar proportions of abducens (38.7%) and oculomotor (35.4%) nerve palsies.⁶ Sixty-three per cent of the cases of oculomotor nerve palsy were pupil-sparing (63.2%) suggestive of microvascular ischaemia.⁶ Furthermore, systemic hypertension accounted for 25.8%, otorhinolaryngological causes accounted for 19.7%, and craniofacial trauma constituted 12.9%. Although all 31 patients in this study performed CT scan imaging, 54.8% of the scans did not demonstrate any abnormality.⁶

On the other hand, Berlit,⁴ in a retrospective study of 412 patients with OMCNP in Heidelberg, Germany, identified oculomotor nerve palsy as the commonest OMCNP, accounting for 41.8% of cases.⁴ Systemic vascular disease, such as hypertension and diabetes, was also the most frequent associated possible cause of OMCNP⁴ as documented in our study. Conversely, a review of 91 patients in Nepal documented abducens palsy as the commonest OMCNP in 53.85% of cases, with oculomotor palsy accounting for less than 25%.¹ In this study, aetiology of OMCNP was not identified

in almost one-third of cases (31.8%). However, the most common identifiable cause was also microvascular disease (26.3%).¹

In common with Berlit,⁴ we observed that oculomotor nerve palsy (57.6%) was the most prevalent OMCNP in Ibadan, presenting almost twice as frequently as abducens palsy (30.5%). On the contrary, majority of other studies had reported abducens nerve as the most prevalent OMCNP.^{1,2,6–8,10–13} Furthermore, a 3-year retrospective review from Port Harcourt, Nigeria, reported an equal frequency of oculomotor and abducens palsy.¹⁵

Common aetiologies of OMCNP in our study were microvascular ischaemia (25.6%), RTA (23.3%), and SOLs (14%). However, aetiology in 27.1% was unclear. Similarly, Tiffin was unable to identify an aetiology in 35% of 165 cases of OMCNP in Dundee.¹⁴

Aetiology

Microvascular ischaemia involving either the oculomotor or abducens nerve is responsible for most isolated OMCNP in patients aged >50 years, when idiopathic cases are excluded.¹¹

Autopsy findings¹⁶ suggest that microvascular ischaemia results from extensive myelin loss and fragmentation of the nerve sheath associated with thickening and hyalinization of the blood vessels of the vasa nervorum rather than vaso-occlusion.⁸

Clinical presentation: Pupil involvement in oculomotor palsy

Pupil involvement in our study was most commonly associated with head trauma and SOL and very rarely with microvascular ischaemia. Although the usefulness of pupilomotor involvement in CN III palsy has been shown to be limited to complete CN III palsy with 'pupil-sparing'. In which case, aetiology may be assumed to be microvascular ischaemia without further investigation.¹⁷ There was pupil-sparing in 87.5% of the microvascular oculomotor nerve palsies in this study, though one patient had pupil involvement associated with microvascular oculomotor palsy. However, in a prospective study of isolated acute ocular motor neuropathy, 7 out of 57 (29%) patients

were reported with microvascular third nerve palsy associated with pupil involvement.¹⁸

Fourth nerve palsy was uncommon (6%) in our study also reported by Richards¹¹ and others.^{1,2,6,12} While a majority of the studies reported decompensated congenital CN IV palsy as the commonest aetiology, other studies excluded congenital cases altogether. Like other studies, the commonest cause of CN IV palsy was trauma in our study.¹ Though microvascular CN IV palsy has been reported in some studies,^{10,12,14,17} aneurysm and malignancy were rare.^{10,14}

Notwithstanding, combined microvascular ischaemia of the oculomotor and trochlear nerves are of diagnostic significance, as this localizes the lesion to the cavernous sinus, specifically affecting their shared blood supply from the inferolateral trunk of the internal carotid artery.¹⁷

Sixth nerve palsy in our study was associated with head trauma, intracranial SOL and CNS infections. However, aetiology of abducens palsy was not identified in 57.1% of our patients. Similarly, a study in Nepal did not identify the cause of abducens palsy in 56.8% of cases.¹⁹

Hypertension, a major risk factor for microvascular ischaemia

Poorly controlled hypertension, as well as combined hypertension and diabetes, were the commonest microvascular risk factors in our study, while hypercholesterolaemia was uncommon. It has been established that Africans are highly susceptible to hypertension and suffer its vascular complications more frequently.^{20,21} The prevalence of hypertension in Nigerian adults ranges from 2.1% to 47.2% depending on the criteria used for selection. Moreover, the prevalence is higher in urban compared with rural settings.²² Therefore, relatively lower numbers of patients with diabetes and hypercholesterolaemia may reflect the lower prevalence of these comorbidities in the Nigerian population as compared with hypertension.^{23,24} Similar findings were also reported in Port Harcourt, South-East Nigeria.¹⁵ **Table 3** shows the systemic and DBP profiles of 34 patients presenting with ocular motor CN paresis in this study.

Patient management and treatment outcomes

Majority of patients presented late and only 16% presented within the first 2 weeks of symptoms. Moreover, the majority defaulted after their first clinic visit while less than half completed their laboratory or radiological investigations. While it is generally believed that OMCNP, related to microvascular ischaemia, resolves spontaneously within an average of 3–5 months,^{25,26} investigation and treatment of the microvascular risk factors is necessary to prevent another episode, which could be more severe, even fatal.

Case fatality

There were at least three deaths, in this study, due to lack of access to emergency medical care, as a result of public service user fees. Records show that two of these cases had presented with extremely high blood pressures but did not accept emergency medical treatment due to financial constraints. Although no autopsy was performed, the time interval between presentation and demise in at least one case (patient who died within 2 weeks of presentation) suggests that the hypertension may have contributed to this death.

Impact of public service user fees on utilization of health services

Healthcare is neither free nor is it significantly subsidized for the majority of Nigerians, due to cutbacks in Government health funding over the last three-and-a-half decades. In reality, patients must meet health-care costs through direct and immediate 'out-of-pocket spending'. Occasionally, indigent patients may be able to benefit from limited 'fee waivers' or special concessions drawn from individual Hospital initiatives, at the point-of-care, such as an 'indigent patient fund' provided by our Hospital management. However, due to the downturn in Nigeria's economy, pressure on these limited funds is great. Moreover, many patients present late to the hospital when they are severely compromised and then fail to return for clinic appointments or discharge themselves against medical advice, when they cannot afford care. There is also a tendency for patients to defer emergency life-saving medical care when they are asymptomatic or

assume they can seek or find a cheaper alternative, even when the situation is grave. This tendency may be borne out of a genuine lack of resources but also lack of education. The case of the patients in this study, with life-threatening hypertension, who failed to accept treatment at the emergency room as well as the patient who discharged against medical advice are such examples.

Public service user fees were introduced into the Nigerian healthcare system in 1998 as a result of the Bamako initiative.²⁷ Because of the need to account for and recover costs, many emergency medications and consumables are no longer freely stock-piled in the hospital emergency rooms or wards and the process for payment and collection is sometimes laborious. This may discourage patients and their relatives who were used to the previous system of free-medical care when there was an abundance of resources.²⁸ The process of accountability is indeed important to keep the system afloat; however, both the public and politicians need to be enlightened about the direct impact of increasing healthcare costs worldwide. Less than 5% of Nigerians have health insurance coverage²⁹ and this is minimal at best. Moreover, expensive radiological examinations such as neuroimaging are not covered.^{29,30,31} As a result of high cost, obtaining necessary neuroimaging is difficult in practice. This impacts on clinical diagnostic accuracy, management, and outcome.

Conclusion

OMCNP appears to be an important manifestation of undiagnosed, poorly controlled, or neglected systemic microvascular disease in this Nigerian clinic. It is possible that poverty or ignorance may play a role in the apparent neglect of the systemic microvascular conditions in these patients.

Isolated ischaemic OMCNP most often spontaneously recovers. Nevertheless, in the presence of multiple OMCNP or persistent isolated OMCNP, neuroimaging is useful in identifying the likely aetiology. However, obtaining neuroimaging is challenging in this low resource setting. Therefore, the neuro-ophthalmologist should have a high index of suspicion for neglected underlying systemic disease when assessing patients with isolated OMCNP in a low resource

setting and exclude these conditions before recommending expensive imaging.

The fatal consequences of prohibitive public service user fees in the face of medical emergencies underscore the need to re-evaluate public health system financing in Nigeria and similar developing countries.

Limitations

The numbers in this study are small. In addition, many patients had constraints in performing different investigations, which reflect the clinical challenges encountered in practice in this low resource setting where 90% of healthcare costs are borne directly by the patients as out-of-pocket payments.

References

1. Sitaula S, Sharma A, Shrestha GG, Gajurel BP, Shrestha G. Clinical manifestation of ocular motor nerve palsies in a tertiary eye hospital of Kathmandu, Nepal. *J Inst Med*. 2014;36:72–77.
2. Rowe F. UK VIS research group. Prevalence of ocular motor cranial nerve palsy and associations following stroke. *Eye (Lond)*. 2011;25(7):881–887. doi:10.1038/eye.2011.78.
3. Lyons CJ, Godoy F, Al Qahtani E. Cranial nerve palsies in childhood. *Eye (Lond)*. 2015;29(2):246–251. doi:10.1038/eye.2014.292.
4. Berlit P. Isolated and combined pareses of cranial nerves III, IV and VI. A retrospective study of 412 patients. *J Neurol Sci*. 1991;103:10–15.
5. Galtrey CM, Schon F, Nitkunan A. Microvascular non-arteritic ocular motor nerve palsies-what we know and how should we treat? *Neuroophthalmology*. 2015;39(1):1–11. doi:10.3109/01658107.2014.963252.
6. Mwanza J, Ngweme G, Kayembe D. Ocular motor nerve palsy: A clinical and etiological study. *Indian J Ophthalmol*. 2006;54(3):173–175. doi:10.4103/0301-4738.27068.
7. Khadse R, Pawar N, Padmavathy SR, Ramakrishnan MR. Clinical profile of ocular motor nerve palsies at a tertiary centre in southern India. *EC Ophthalmol*. 2017;6:89–94.
8. Kung NH, Van Stavern GP. Isolated ocular motor nerve palsies. *Semin Neurol*. 2015;35(5):539–548. doi:10.1055/s-0035-1563568.
9. Murchison AP, Gilbert ME, Savino PJ. Neuroimaging and acute ocular motor mononeuropathies: a prospective study. *Arch Ophthalmol*. 2011;129(3):301–305. doi:10.1001/archophthalmol.2011.25.
10. Park UC, Kim SJ, Hwang JM, Yu YS. Clinical features and natural history of acquired third, fourth, and sixth cranial nerve palsy. *Eye (Lond)*. 2008;22(5):691–696. doi:10.1038/sj.eye.6702720.

11. Richards BW, Jones FR Jr., Younge BR. Causes and prognosis in 4,278 cases of paralysis of the oculomotor, trochlear, and abducens cranial nerves. *Am J Ophthalmol.* 1992;113:489–496.
12. Surachatkumtonekul T, Soontrapa P, Kampanartsanyakorn S, Dulayajinda D. Causes and treatment outcomes of third, fourth and sixth cranial nerve palsy. *J Med Assoc Thai.* 2012;95:S96–S101.
13. Tamhankar MA, Bioussé V, Ying GS, et al. Isolated third, fourth, and sixth cranial nerve palsies from presumed microvascular versus other causes: a prospective study. *Ophthalmology.* 2013;120(11):2264–2269. doi:10.1016/j.opthta.2013.04.009.
14. Tiffin PAC, Macewen CJ, Craig EA, Clayton G. Acquired palsy of the oculomotor, trochlear and abducens nerves. *Eye.* 1996;10(Pt 3):377–384. doi:10.1038/eye.1996.77.
15. Pedro-Egbe CN, Fiebai B, Awoyesuku EA. A 3-year review of cranial nerve palsies from the University of Port Harcourt Teaching Hospital Eye Clinic, Nigeria. *Middle East Afr J Ophthalmol.* 2014;21(2):170–174. doi:10.4103/0974-9233.129770.
16. Dreyfus PM, Hakim S, Adams RD. Diabetic ophthalmoplegia; report of case, with postmortem study and comments on vascular supply of human oculomotor nerve. *AMA Arch Neurol Psychiatry.* 1957;77:337–349.
17. Sharpe JA, Wong AM, Fouladvand M. Ocular motor nerve palsies: implications for diagnosis and mechanisms of repair. *Prog Brain Res.* 2008;171:59–66. doi:10.1016/S0079-6123(08)00609-2.
18. Chou KL, Galetta SL, Liu GT, Volpe NJ, Bennett JL, Asbury AK, Balcer LJ. Acute ocular motor mononeuropathies: prospective study of the roles of neuroimaging and clinical assessment. *J Neurol Sci.* 2004;219(1):35–39. doi:10.1016/j.jns.2003.12.003.
19. Shakya S, Argrawal JP, Ray P. Profile of isolated sixth cranial nerve palsy: a hospital based study. *J Neurosci.* 2004;1:32–35.
20. Ajayi I, Sowemimo I, Akpa O, Ossai N. Prevalence of hypertension and associated factors among residents of Ibadan-north local government area of Nigeria. *Niger J Cardiol.* 2016;13(1):67–75. doi:10.4103/0189-7969.165168.
21. Howard G, Lackland DT, Kleindorfer DO, Kissela BM, Moy CS, Judd SE, Safford MM, Cushman M, Glasser SP, Howard VJ. Racial differences in the impact of elevated systolic blood pressure on stroke risk. *JAMA Intern Med.* 2013;173(1):46–51. doi:10.1001/2013.jamainternmed.857.
22. Akinlua JT, Meakin R, Umar AM, Freemantle N. Current prevalence pattern of hypertension in Nigeria: A systematic review. *PLoS ONE.* 2015;10(10):e0140021. doi:10.1371/journal.pone.0140021 (Electronic).
23. Oguoma VM, Nwose EU, Skinner TC, Digban KA, Onyia IC, Richards RS. Prevalence of cardiovascular disease risk factors among a Nigerian adult population: relationship with income level and accessibility to CVD risks screening. *BMC Public Health.* 2015;15:397. doi:10.1186/s12889-015-1709-2.
24. Isara AR, Okundia PO. The burden of hypertension and diabetes mellitus in rural communities in southern Nigeria. *Pan Afr Med J.* 2015;20:103. doi:10.11604/pamj.2015.20.103.5619.
25. Capó H, Warren F, Kupersmith MJ. Evolution of oculomotor nerve palsies. *J Clin Neuroophthalmol.* Mar 1992;12(1):21–25.
26. Comer RM, Dawson E, Plant G, Acheson JF, Lee JP. Causes and outcomes for patients presenting with diplopia to an eye casualty department. *Eye.* 2007;21:413–418. doi:10.1038/sj.eye.6702415.
27. Olakunde B. Public health care financing in Nigeria: which way forward? *Ann Niger Med.* 2012;6(1):4–10. doi:10.4103/0331-3131.100199.
28. Lagarde M. The impact of user fees on health service utilization in low- and middle-income countries: how strong is the evidence? *Bull World Health Organ.* 2008;86:839–848.
29. Awosusi A, Folaranmi T, Yates R. Nigeria's new government and public financing for universal health coverage. *Lancet Global Health.* 2015;3(9):e514–e515. doi:10.1016/S2214-109X(15)00088-1.
30. Ogun OA, Adediran OA. Non-glaucomatous optic neuropathy in Ibadan: extrapolations to healthcare funding in Nigeria. *Ann Ibd Pg Med.* 2014;12:103–108.
31. National Health Insurance Scheme. National health insurance scheme: scope of coverage. 2018. <https://www.nhis.gov.ng>. Accessed July 5, 2018.