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Causes of Sudden Dimness of Vision: A Retrospective Study at the University Teaching Hospital, Awka, Nigeria

Bernard Chukwunonyerem Ochiogu [a], Akunne Ijeoma Apakama [b], Anthonia Chinyelu Udeaja [c], Oluwafumi Adebimpe Ijeoma Otuka [d] and Arinze Anthony Onwuegbuna [b*]

[a] Eye unit, Department of Surgery Chukwuemeka Odimegwu Ojukwu University Teaching Hospital Awka, Nigeria.
[b] Department of Ophthalmology, Nnamdi Azikiwe University Teaching Hospital, Nnewi, Nigeria.
[c] Department of Ophthalmology, Chukwuemeka Odumegwu Ojukwu University, Awka Campus, Anambra State, Nigeria.
[d] Ophthalmology Unit, Department of Sugery, Abia State University, Uturu, Abia State, Nigeria.

Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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ABSTRACT

Aim: To determine the causes of sudden dimness of vision at the Chukwuemeka Odumegwu Ojukwu University Teaching Hospital. Awka, Nigeria.
Methods: This is a retrospective hospital-based survey carried out at the Chukwuemeka Odumegwu Ojukwu University Teaching Hospital Awka, Nigeria. The case notes of new patients seen at the Eye Unit of the hospital from January 2017 to December 2021 were examined. Those with a history of sudden dimness of vision were identified and relevant data were extracted and analyzed using descriptive statistics.

**Corresponding author: E-mail: aa.onwuegbuna@gmail.com, aa.onwuegbuna@unizik.edu.ng;*

Results: A total of 3755 new patients were seen during the study period, of which 191 (5.1%) presented with sudden dimness of vision. Three people had bilateral lesions making a total of 194 involved eyes. Of the 191 patients, 101(52.9%) were male while 90 (47.1%) were female (M: F ratio 1.1:1). The age range was 1 year to 84years, and the mean and median age were 40.2years and 34years respectively while the bimodal age distributions of 30years and 60years (7patients each) was noted. The age range of 21-30years presented more cases of sudden vision loss at 37 (19.4%).

Generally, ocular injuries were responsible for more sudden dimness of vision 102 (53.4%), with contusion being the highest 31 (16.2%) of the traumatic diagnosis. Non-traumatic causes were responsible for 89 (46.6%) sudden dimness of vision. Forty-one eyes (21.1%) had visual acuity of 6/9 – 6/18, 85 (44%) had visual acuity of 6/18-3/60, while 68 (35.8%) presented with visual acuity of <3/60.

Conclusion: Sudden dimness of vision is a serious concern to the affected and relations. The causes are divers and may be traumatic or non-traumatic, with the traumatic type in the majority. Traumatic types of sudden dimness of vision are more common in younger age groups than older adults, and males sustain more traumatic sudden dimness of vision than in female folks. Avoiding trauma-risky tasks, applying safety measures, and adequately managing existing systemic morbidity may mitigate the trend.

Keywords: Causes; sudden; dimness; vision; Awka; Nigeria.

1. INTRODUCTION

Vision dimness is sudden if it develops within a few minutes to a few days [1] Hornby [2] defined sudden as happening or done quickly and unexpectedly. Sudden dimness of vision can affect one or both eyes and all or part of a field of vision [3]. Vision dimness is a decreased ability to see to the degree that causes problems not fixable by usual means, such as spectacles or contact lenses [4,5]. It is also a decrease in vision to the degree that causes concern to the affected or the relations. Sudden dimness of vision has three general causes: clouding of usually transparent eye structures, abnormalities of the retina and nerves that carry visual signals from the eye to the brain (the optic nerve and visual pathway) [3]. Acute dimness of vision is a frightening experience for the patient and has the potential for long-term consequences [6]. It is vital to distinguish between actual sudden loss of vision and sudden realization of vision loss which may be partial or total, temporary or permanent depending on the cause [7].

Vision dimness significantly impacts the lives of those who experience it as well as their families, friends and society [8,9]. Complete loss or deterioration of existing eyesight can be frightening, and overwhelming leaving those affected to wonder about their ability to maintain independence, pay for needed medical care, retain employment and provide for themselves and their families [8,10]. The health consequences associated with vision loss extends well beyond the eye and visual system. In general, vision loss can affect one's quality of life (QOL), independence and mobility. It has been linked to falls, injury and worsened status in mental health, cognition, social function, employment and educational attainment domains [8,10]. There is total or near total loss of instrumental activities of daily living (IADL). Acute visual failure may be a presenting symptom of ocular stroke, and ocular strokes are due to central retinal artery occlusion, branch retinal artery occlusion or anterior ischaemic optic neuropathy, which is the result of infarction of the optic nerve head [11,12].

Amaurosis fugax, a subjective phenomenon caused by a transient and temporary ceasing of retina blood flow, has been associated with temporary and monocular blindness lasting a few seconds to a few minutes [11,12] Wray [11] has classified amaurosis fugax into four types viz embolic, hypoperfusion, angiospasm and idiopathic mechanism. From the above, acute or sudden dimness of vision, permanent or temporary, could be a harbinger of cardiovascular or other systemic problems. Transient monocular blindness (type I or II) is a premonitory symptom suggesting an embolic cause or temporal arteritis [13] In patients under the age of 40years, the heart is the leading source of emboli [14,15] because of rheumatic valvular disease, bacterial endocarditis or cardiac myxoma [16] In older people, the source of the embolus may be cardiac [17] or intra-arterial from atheromatous ulcerations of the aorta or the ipsilateral internal carotid artery.

Ocular trauma is a known cause of temporary and permanent vision loss through varied mechanisms and circumstances [6,9,18-21] Compression of the globe may be self-inflicted in cases involving heavy alcohol use with or without drug consumption, followed by stupor, resulting in sudden dimness of vision [22].

Both central (Ischaemic and non-ischaemic) and branch retinal vein occlusions cause sudden dimness of vision [12]. Some systemic diseases like hypertension, diabetes, and sickle cell disease, in the course of their progression, can cause sudden visual loss through many mechanisms, especially when poorly controlled [12,23]. Age related macular degeneration, especially the wet type and macular hole formation are known to cause sudden and profound dimness of vision most commonly in the elderly folks [12,24]. Intraocular inflammations, like uveitis, endophthalmitis and/or panophthalmitis, are known causes of sudden and profound dimness of vision.

Discrete areas of monocular vision loss may represent intraocular lesions like vitreous/retinal haemorrhage or retinal detachment, while monocular vision loss respecting the horizontal meridian may result from vascular lesions of the optic disc or retinal circulation [25] Artery occlusions are more rapid in their onset than vein occlusions. The occlusion site determines the scotoma's extent; a central retinal vessel occlusion results in global monocular vision loss, while a branch retinal vessel occlusion causes a segmental scotoma. Sudden dimness of vision associated with headaches, Jaw claudication, scalp tenderness, unexplained weight loss, night sweats, diplopia or temporal artery tenderness is strongly suggestive of giant cell arteritis. It should be considered in any patient over the age of 50 years with sudden onset of vision loss or diplopia [26] Accidental or intentional ingestion of toxic agents like methanol can lead to methanol toxicity with resultant sudden blindness [27]. The oxidation of the methanol in the body results in toxic agents like formic acid and formaldehyde, which cause oedema and degeneration of the ganglion cells. Acute microbial infection of the eye can also result in sudden and permanent vision loss.

2. MATERIALS AND METHODS

This was a five-year retrospective hospital-based survey from January 2017 to December 2021. However, the study was carried out at the Chukwuemeka Odumegwu Ojukwu University Teaching Hospital, Awka, Anambra State, Nigeria.

The case notes of all the new patients seen at the Eye Unit of the hospital within the study period were examined. Those with a history of sudden dimness of vision (dimness of vision within one month in a previously normal eye) were further reviewed. Information on biodata (age, sex, occupation and clinical data, which included visual acuity at presentation, chief complaints, duration of the complaints, diagnosis, and eventual visual acuity at the last follow-up visit) were recorded on a standard proforma. The data were analyzed using descriptive statistics.

3. RESULTS

Of the 3755 new patients, 191 (5.1%) presented with a history of sudden dimness of vision. Out of the 191 patients, 101 (52.9%) were males while 90 (47.1%) were females (M: F ratio 1.1:1). The age range was 1 year to 84years, the mean age was 40.2years and the mean, the standard deviation was ±18.7, while the median was 34 years. There were bimodal age distributions of 30 and 60 years (7 patients each).

The age range 21-30 years presented with more cases of sudden visual loss 37 (19.4%), followed closely by 51-60 years age range 34 (17.8%) and 31-40 years 33 (17.3%)- Table 1.

Ocular injuries, which comprised contusions, 31 (16.2%) cases of hyphema, 15 (7.9%), open globe injuries, 12 (6.3%), and traumatic cataracts, 9 (4.7%) cases were noted. Ulcerative keratitis 17 (8.9%), which was partly traumatic and non-traumatic, uveitis 9 (4.7%), chemical burns 5 (2.6%), vitreous haemorrhage 4(2.1%), traumatic aphakia 1(0.5%) and couched eye 3 (1.6%) were also observed.

The non-traumatic causes of sudden dimness of vision identified include herpes zoster ophthalmicus 13 (6.8%) cases, retinal vein occlusion 12 (6.3%), optic neuritis 11 (5.8%), diabetic refractive changes 11 (5.8%) and uveitis 8 (4.2%) which consists of anterior, posterior and pan uveitis. Others were non-ulcerative keratitis 7 (3.7%) and macular hole 5 (2.6%). The ulcerative keratitis, orbital cellulitis and panophthalmitis had 4 (2.1%) cases each. One (0.5%) case each was observed for central retinal artery occlusion, endophthalmitis and diabetic retinopathy.

Contusional eye injuries 31 (16.2%) were the most specific diagnosis causing sudden dimness of vision, followed by ulcerative keratitis and uveitis 17 (8.9%). Central retinal artery occlusion, traumatic aphakia, endophthalmitis and diabetic retinopathy, which accounted for 1 (0.5%) case each, were the least common Table 2.

Traumatic causes of sudden dimness of vision were more frequent in males than females and in the younger age group than the older age group. The non-traumatic diagnosis exhibited the opposite trend of traumatic causes regarding sex and age distribution. Tables 3 and 4.

Some patients recovered good vision, some had moderate vision, while others permanently lost vision in the affected eye. Generally, trauma cases presented earlier than non-trauma cases.

Table 1. Age and sex distribution of 191 patients

Age Range	Sex		Total	Percentage
	Male	Female		
0-10	5	2	7	3.7
11-20	12	7	19	9.9
21-30	20	17	37	19.4
31-40	18	15	33	17.3
41-50	15	13	28	14.7
51-60	18	16	34	17.8
61-70	10	16	26	13.6
71-80	2	4	6	3.1
≥81	1	0	1	0.5
Total	101 (52.9%)	90 (47.1%)	191 (100%)	100

Table 2. Diagnosis, frequency and sex distribution

Diagnosis	Male	Female	Total	Percentage
Contusional injuries	16	15	31	16.2
Ulcerative keratitis	14	3	17	8.9
Uveitis	10	7	17	8.9
Hyphema	10	5	15	7.9
Herpes zoster ophthalmicus	5	8	13	6.8
Open globe injury	7	5	12	6.3
Retinal vein occlusion	5	7	12	6.3
Optic neuritis	5	6	11	5.8
Diabetic refractive change	4	7	11	5.8
Traumatic cateract	6	3	9	4.7
Non-ulcerative keratitis	4	3	7	3.7
Macular hole	0	5	5	2.6
Chemical burns	3	2	5	2.6
Vitreous haemorrhage	3	1	4	2.1
Orbital cellulitis	1	3	4	2.1
Panophthalmitis	1	3	4	2.1
Retinal detachment (RD)	1	2	3	1.6
Couched eye	1	2	3	1.6
Angle closure glaucoma	1	1	2	1.0
Hypertensive retinopathy	2	0	2	1.0
Central retinal artery occlusion	0	1	1	0.5
Traumatic aphakia	1	0	1	0.5
Endophthalmitis	0	1	1	0.5
Diabetic retinopathy	1	0	1	0.5
Total	101	90	191	100

Table 3. Traumatic causes of sudden dimness of vision

Diagnosis	No	Percentage
Contusional injuries	31	16.2
Hyphema	15	7.9
Ulcerative keratitis	13	6.8
Open globe injury	12	6.3
Uveitis	9	4.7
Traumatic cataract	9	4.7
Chemical burns	5	2.6
Vitreous hemorrhage	4	2.1
Couched eye	3	1.6
Traumatic aphakia	1	0.5
Total	102	53.4

Table 4. Non-Traumatic causes of sudden dimness of vision

Diagnosis	No	Percentage
Herpes zoster ophthalmicus	13	6.8
Retinal vein occlusion	12	6.3
Optic neuritis	11	5.8
Diabetic refractive changes	11	5.8
Uveitis	8	4.2
Non ulcerative keratitis	7	3.7
Macular hole	5	2.6
Ulcerative keratitis	4	2.1
Orbital cellulitis	4	2.1
Panophthalmitis	4	2.1
Retinal detachment (RD)	3	1.6
Angle closure glaucoma	2	1
Hypertensive retinopathy	2	1
Central retional artery occlusion	1	0.5
Endophthalmitis	1	0.5
Diabetic retinopathy	1	0.5
Total	89	46.6

4. DISCUSSION

Whether bilateral, unilateral, partial, complete, temporary or permanent, vision loss is always a scary experience for the victim [3,6]. Sudden dimness of vision may create a worst-case scenario for the affected as they may not have envisioned the situation. The prevalence of sudden dimness of vision in this survey was 5.1%. However, there is no available literature to compare. This study showed marginally more males (52.9%) had sudden dimness of vision than females (47.1%). This preponderance of males could be due to the contribution of trauma to the sudden dimness of vision in this survey, which has been collaborated by other studies [28,29]. And because men tend to perform more artisan/risky tasks compared to females, these put the former at a greater risk for ocular trauma [28].

The age range 21-30years presented with more cases of sudden dimness of vision (19.4%) in this study, which were majorly due to ocular injuries. The increased frequency of ocular injuries among this age group and its attendant contribution to sudden dimness of vision and ocular morbidity has been reported by other researchers [18,28,30-33]. This has been attributed to increased activities among this age group. Generally, traumatic causes of sudden dimness of vision were commoner in the males and younger age groups than in the females and geriatric age groups in this review. This agrees with the report of Ochiogu et al. [18] Decreasing activity, change of lifestyle, and occupational

pattern with advancing age have been suggested as the reason [18]. However, the causes of non-traumatic sudden dimness of vision were seen more in the older age group with the marginal disparity in frequency seen in males and females in this study. These include herpes zoster ophthalmicus (6.8%), retinal vascular occlusions (6.3%), optic neuritis (5.8%), diabetic refractive changes (5.8%), uveitis (4.2%), keratitis (ulcerative and non ulcerative) (5.8%) and macular hole (2.6%). The 5 (2.6%) patients with macular holes in this study were all females with post-menopausal status. Another study [12] corroborated that senile or idiopathic macular hole is more common (83%) in females aged 60-80years than males and typically comes with vision around 6/60 level.

Herpes zoster ophthalmicus, an infection caused by the human herpes virus, the same virus that causes chickenpox, was observed to cause significant visual loss (6.8%) in this study. Wiafe [34] also reported the association of herpes zoster ophthalmicus and vision loss through different mechanisms and that increasing age is one of the predisposing factors to the development of herpes zoster ophthalmicus, probably due to immune down regulation. Retinal vascular occlusion (6.8%) was noted as a cause of sudden dimness of vision among the elderly in this study than in the young. Kharana [12] had previously reported an increased incidence of central retinal vein occlusion in the elderly. This association between central vein occlusion and old age may be due to pressure on the vein by the atherosclerotic retinal artery, where the two share a common adventitia [12]. Of the (6.8%) retinal vascular occlusion, 1 (0.5%) was due to the central artery, while [12] (6.3%) was due to central retinal vein occlusion. The central retinal vein occlusion was observed to be more commoner than the former in this study, and this was similar to the findings of Nwosu [35] Artery occlusions are more rapid in their onset than vein occlusions.7Thrombo-embolic and vascular disorders have also been adduced as one of the causes of central retinal artery occlusions with diabetes, hypertension and giant cell arthritis as predisposing factors [11,36]. Majority of the subjects in this review with central retinal vein occlusion were in their sixties and seventies, thus similar with the findings of Khurana.12 Central retinal artery occlusion and retinal vascular disease have also been associated with elevated levels of antiphospholipid antibodies and systemic lupus erythematosus [37-42]. Though systemic lupus erythematosus is commoner in people of African and Asian descent, its thrombotic complications are more common in caucasian patients [43].

Diabetes mellitus, another non-traumatic cause of sudden dimness of vision, was identified in this study 12(6.3%). Frequently, newly developed diabetics are present in the eye clinic first because of diabetic refractive changes. Many authors [44-50] have reported refractive changes in association with diabetes. Those refractive changes may be myopic or hyperopic shifts depending on the mechanism involved. In the present review, most of the refractive changes were of myopic shift 7(3.7%), while 4 (2.1%) were hyperopic shifts. One (0.5%) was a case of diabetic retinopathy. While the myopic shift findings in this study are in agreement with the findings of those authors [44-48], the later (hyperopic) shift aligns with the reports of Furushima and colleague [49] and Satio et al. [50].

Optic neuritis was noted to cause sudden dimness of vision in this study, with a prevalence of 0.3%. Osaguona and colleagues [51] as well as other authors reported sudden dimness of vision [52,53]. In Benin, however, Osaguona et al. [51]. reported a prevalence of 0.13%. This difference could result from differences in study duration and population size of the two studies. Both studies agreed that more females than males were affected. Keratitis (both ulcerative and non-ulcerative) was found to cause significant vision loss in this study and agrees with other studies, [54,55] this is because the cornea has been reported as the most effective refractive medium in the eye and pathologies affecting the cornea usually have a significant impact on vision [56] Trauma was the cause of ulcerative keratitis in this study corroborating the findings of earlier authors [18,54,55] Non-ulcerative keratitis was also noted to be a cause of dimness of vision in the present review and was a majorly non-traumatic cause. Uveitis 17(8.9%) was cited as a cause of acute sudden dimness of vision, but other authors [57] have reported that uveitis is a significant cause of visual loss in both developed and developing nations of the world and that it accounts for 25% of legal blindness in the developing world [58-62]. Varied aetiologies have been proposed by some authors [18,63]. However, another author, [64], has reported that despite a great deal of experimental research and many sophisticated methods of investigations, the aetiology and immunology of uveitis still need to be

Table 5. Diagnosis and age distribution

Diagnosis	0-10 years	11-20 years	21-30 years	31-40 years	41-50 years	51-60 years	61-70 years	71-80 years	≥81 Years	Total	Percentage
Contusional injuries	3	4	9	4	4	3	3	1	0	31	16.2
Ulcerative keratitis	0	3	2	3	3	5	1	0	0	17	8.9
Uveitis	0	4	3	1	1	2	5	1	0	17	8.9
Hyphema	0	3	4	3	3	1	1	0	0	15	7.9
Herpes zoster ophthalmicus	0	0	3	2	4	3	1	0	0	13	6.8
open globe injury	4	1	1	3	3	0	0	0	0	12	6.3
Retina vein occlusion	0	0	1	1	0	3	5	2	0	12	6.3
Optic neuritis	0	0	3	1	0	4	2	1	0	11	5.8
Diabetic refractive changes	0	0	3	3	3	2	0	0	0	11	5.8
Traumatic cataract	0	2	3	2	1	1	0	0	0	9	4.7
Non. ulcerative keratitis	0	0	3	1	1	1	1	0	0	7	8.7
Macular hole	0	0	0	0	0	2	3	0	0	5	2.6
Chemical burns	0	0	2	1	1	1	0	0	0	5	2.6
Vitreous haemorrhage	0	0	0	2	1	1	0	0	0	4	2.1
Orbital cellutitis	0	2	0	2	0		0	0	0	4	2.1
Panophthalmitis	0	0	0		1	1	1	1	0	4	2.1
Retinal detachment	0	0	0	1	0	1	1	0	0	3	1.6
Couched eye	0	0	0		0	2	1	0	0	3	1.6
Angle closure glaucoma	0	0	0	1	1	0	0	0	0	2	1.0
Hypertensive retinopathy	0	0	0	0	1	0	0	0	1	2	1.0
Central retinal artery occlusion	0	0	0	1	0	0	0	0	0	1	0.5
Traumatic aphakia	0	0	0	0	0	0	1	0	0	1	0.5
Endoplthalmitis	0	0	0	1	0	0	0	0	0	1	0.5
Diabetic retionpathy	0	0	0	0	0	1	0	0	0	1	0.5
Total number	7	19	37	33	28	34	26	6	1	191	100
Total %	3.7	9.9	19.4	17.3	14.7	17.8	13.6	3.1	0.3		100

Table 6. Visual acuity (VA) at presentation and last follow up visit

WHO Category	Presenting visual acuity	Last follow up visual acuity
WHO category Normal vision	Presenting visual acuity Injured Eye	Last follow up VA Injured eye
6/6-6/18	41 (21. 1%)	69 (35.6%)
Impaired vision 6/18-3/60	85 (44%)	98 (50.5%)
Blind < 3/60	68 (35.8%)	27 (13.9%)
	194 (100%)	194 (100%)

Three people had bilateral lesions

understood. So the causes of many clinical conditions are disputed. Though allergic uveitis is the most familiar occurrence in clinical practice, the complex subject of immune-linked inflammation of uveal tissue is still not clearly understood.64 Ochiogu and colleague [65] reported that inappropriate application of topical steroid eye drops has been linked to symptoms and signs that resemble acute anterior uveitis.

In the present review, anterior uveitis 8 (4.2%) was the commonest, followed by posterior uveitis 6 (3.1%) and panuveitis 3 (1.6%). When screened, two cases of panuveitis patients were positive for the human immune deficiency virus. Ajayi and colleagues [63]. in Ekiti reported anterior uveitis 109 (63.7%) as the commonest type of uveitis, which aligns with the present study. However, they noted that panuveitis 38(22.2%) was more commoner than posterior uveitis 20 (11.7%). Generalized uveitis has been associated with human immune deficiency virus seropositivity and acquired immune deficiency syndrome (HIV/AIDS) [66]. Ajayi and colleagues [63] reported that 3(1.8%) patients were HIV positive but did not categorize their uveitis type. More importantly, the Ajayi [63] study was purely on uveitis as opposed to the present study. Nwosu [67] in Onitsha reported that 4% of HIV/AIDS-positive patients had uveitis.

Orbital cellulitis, an acute infection of soft tissues of the orbit behind the orbital septum [68], was noted as a cause of sudden dimness of vision in this study. Other authors [69-71] had also documented orbital cellulitis as a cause of ocular morbidity and vision loss in their separate studies. In this review, 4 (2.1%) cases were noted, of which one was male while three were female. Previous researchers [69-71] had reported a preponderance of one sex or the other, but no sex predilection has been reported. Upper respiratory tract infection and sinusitis were noted as predisposing factors by these authors [69-71] and this agrees with this study. However, traumatic causes were also reported by Uhumwangho and colleagues [70].

Khurana, [12] Nwosu [35] and Nwosu et al. [72] had reported the causes of visual challenges and emergencies as panophthalmitis, traumatic hyphema, endophthalmitis, acute angle closure glaucoma and others. Panophthalmitis, 4(2.1%), retinal detachment 3(1.6%), angle closure glaucoma 2(1%) and endophthalmitis 1(0.5%) were all found to be causes of sudden vision loss. Hypertensive retinopathy 2 (1%) was noted as a cause of sudden vision loss, and the patients never knew that they were living with the pathology before the presentation. Many patients engaged in self-medication due to self-choice and advice from friends and relatives before coming to the hospital [73,74]. However, trauma cases generally visited the hospital earlier than non-trauma cases. Sudden vision loss of any cause can cause psychological problems and absenteeism from work or school.

5. CONCLUSION

Sudden dimness of vision or deterioration is alarming to the affected. The causes are many and may be traumatic or non-traumatic causes. However, traumatic causes are in the majority, and younger people encounter more trauma than older ones. People should be educated on the need to avoid general and ocular trauma. The application of safety measures in workplaces should be stressed. Regular screening for those with systemic diseases should be encouraged to detect early signs of non-traumatic vision loss to necessitate the prompt application of intervention measures.

CONSENT

As per international standards or university standards, patient(s) written consent has been collected and preserved by the author(s).

ETHICAL APPROVAL

Ethical approval was sought and granted by the ethical committee of the Chukwuemeka Odumegwu Ojukwu University Teaching Hospital.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Trobe JD. The physicians guide to eye care 2nded. The foundation of the American Academy of ophthalmology, San Fransasco; 2001.
2. Hornby AS. Oxford advanced learner's dictionary of current enghooh. In Wehmeier S. ed. 6th ed. Oxford university Press; 2000.
3. Brady JC. Acute Vision loss; 2015. Available:www msd Manuals.com
4. Whttps://en. M. Wikipedia org. A. ab.cd changes in the Definition of Blindness. (PDF) from original; 2015. [Accessed on 23 may 2015]
5. Aabcdefgh. Blindnoss and vision Imaparment. Archieved from the original 3 on 29, Aprial 2015; 2011. [Accessed on 23 may 2015]
6. Borooah S, Dhillon A, Dhillon B. Gradual loss of vision in adults. BMJ 2015;350: 2093.
7. Goold L, Durkin S, Crompton J. Sudden loss of vision: History and examination. Australian family physician. 2009;38(10): 764-768.
8. National Academies of Sciences, Engineering and Medicines. Making eye health a population health imperative: Vision for tomorrow. Washington DC: The national Academies press; 2016. Available:https://doi.org/10.17226/23471.
9. Ochiogu BC, Ughachukwu PO. Airbag induced ocular injuries. A short Report: orient Journal of Surgical Sciences. 2020;1(1):25-28.
10. Sandford SJ. Introduction. In; Sandford SJ ed Eye Diseases in Hot climate 3rd ed. Torino: Butterwort Heinenuann. 1997;1-11.
11. Wray SH. Acute visual failure, In Hyghes RAC (ed) Neurological Emergencies. London WC IH 9JR, BMJ Publishing groups, BMA House tavistock Square. 1994;316-333.
12. Khurana Ak. Diseases of Retina. In; khurana Ak ed Comprehensive ophthalmology 5th ed. New dechi: New Age International LTD. 2012;263-306.
13. Wray SH. Extracramal Internal carotid artery disease. In Bernstein EF ed. Amaurosis fugax. New York: springer-verlag. 1988:72-80.
14. Cogan DG, Wray SH. Vascular occlusion in the eyes from cardiac myxoma. AMJ ophthalmol. 1975;80:396-403.
15. Appen RF, Wary SH, Cogan DG. Central retinal artery occlusion. AMJ Ophthalmol 1975;79:374.
16. Burde RM. Amaurosis Fugax, an Oveview J. clinNeuro-Ophthalmol. 1989;9(3):185-9.
17. Zimmerman LE. Embolism of central retinal artery; secondary to myocardial infarction with mural thrombosis. Arch Ophthalmol. 1965;73:822.
18. Ochiogu BC, Udeaja AC. Incidence and pattern of ocular injuries at the Chukwuemeka Odumegoru Ojukwu University teaching Hospital Awkq, Nigeria. Orient Journal of Surgical Sciences. 2021;2:26-34.
19. Kim JM, Kim KO, Kim YO, Chio GT. A case of airbag associated severe ocular injury. Korean J. Ophthalmol 2004;18(1): 84-88.
20. Onyekwe LO. Spectrum of Eye Injuries in Children in Guinness Eye Hospital Onitsha. The Nigerian Journal of Surgical Research. 2001;3:3-4:126-132.
21. John DL. Beare: Eye injuries from assault with chemicals. British Journal of Ophthalmology. 1990;74:514-518.
22. Jayam AN, Hass WK, Carr RE, Kumar AJ. Saturday night retinopathy. J Neurol. Sci. 1974;22:413.
23. Kansiki JJ. Retinal vascular disorders. In, kansiki JJ ed. Clinical ophthalmology 3rd ed. Butterworth – Heinemann. 1997;343-379.
24. Kansiki JJ. Degenerations and dystrophies of the retina. In Kansiki JJ ed. Clinical ophthalmology a systematic approach 3rd ed. Butterworth-Heinemann 1997;381-425.
25. Kansiki JJ. Clinical ophthalmology: A systematic approach. 6th ed. Edinburgh: New York: Butterworth-Heinemann/ Elsevier; 2007.
26. Danesh-Meyer HV, Savino PJ. Giant cell arteritis. Curropinophthalmol. 2007;18:443-9.
27. Cursiefen C, Bergua A. Acute bilateral blindness caused by accidental methanol

intoxication during fire “eating”. Br J Ophthalmol. 2002;86:1064-5.
28. Ngozika Esther Ezinne et al. ocular injuries among Adults in Owerri Municipal, Imo State, Nigeria ARC journal of Ophthalmology. 2018;3(1):4-9.
29. Negrel AD, Thylefors B. The global impact of eye injuries. Ophthalmol Epidemol. 1998;5:143-169.
30. Omolase CO, Omolade EO, Ogunleye OT, Omolase BO, Ihemedu CO, Adosun OA. Pattern of ocular injuries in Owo, Nigeria. J. Ophthalmol Vis Res. 2011;6(2):114-8.
31. Addisu Z. Pattern of ocular trauma seen in Grorbet Hospital, Butajira Central Ethiopia. Ethiop J Health Dev. 2011;25(2):150-155.
32. Rafindadi AL, Pam VA, Chinda D, Mohammed-Ajeigbe FA. Orbital and ocular Trauma at Ahmadu Bello University Teaching Hospital, Shika-Zaria. A retrospective review. Ann Nigeria Med. 2013;7:20-23.
33. Mela EK, Mantzouranis GA, Giokoumis AP, Blastsios G,Andrkopouelos GK, Gartagonis SP. Ocular Trauma in a Greek population. Review of 899 cases resulting in Hospitalization. Ophthalmic Epidemiol. 2005;12:185-90.
34. Wiafe B. Herpes Zoster Ophthalmucos in HIV/AIDS. Community Eye Health Journal. 2003;16(47):35-36.
35. Nwosu SNN. Ophthalmic Emergencies, In; Nwosu SNN (ed) NAUTH Emergency care Handbook. Nimo, Anambra State. Rex Charles and Patrick Limited. Book Smith House Harmony Palace Box. 2014;575:70-113.
36. Nwosu SNN. Ophthalmic Surgical Priorities in a new teaching hospital in Nigeria. Niger. Med J. 1994;26(1):26-6.
37. Englert H, Hawks CH, Boey ML et al. Dego’s disease: Association with anticardiolipin antibodies and the Lupus anticoagulant. BMJ. 1984;289:576.
38. Glueck HI, Kant KS, Weiss MA et al. Lupus erythematosus: Relation to the presence of circulatory anticoagulant. Arch intern. Med. 1985;145:389-95.
39. Shalev Y, Green L, Pollack A, et al. Myocardial Infarction with central retinal artery occlusion in a patient with antinuclear antibody-negative systemic lupus erythematosus. Arthritis Rheum. 1985;28:1185-7.
40. Jonas J, Kolbe K, Volcker HE, et al. Central retinal artery occlusion in sneddon’s disease: association with antiphospholipid antibodies. AMJ ophthalmol. 1986;102:37-40.
41. Pulido JS, Ward LM, Fishman GA, et al. Antiphospholipid antibodies associated with retinal vascular disease. Retina. 1987;7:215-18.
42. Silverman M, Lubeck MJ, Briney WG. Central retinal vein occlusion complicating systemic Lupus erythematoses. Arthritis Rheum. 1978;21:839-43.
43. Tikly M, Navera SV. Lupus in the developing world- is it any different? Best Practice and research. 2008;22(4):643-655.
44. Fledelius HC. Is myopia getting more frequent?. A cross-sectional study of 1416 Danes aged 16years and above. Actaophthalmol (copenh). 1983;61:545-559.
45. FledeliusHC Myopia and diabetes mellitus with special reference to adult onset myopia. Actaophthalmol (copenh). 1986; 64-33-38.
46. WU SY, YOO YJ, Nemesure B, Hennis A, Leske MC. Nine year refractive changes in Barbados Eye studies. Invest ophthalmolvissci. 2005;46(11):4032-4039.
47. Tarczy Harnock K, Ying-Lai M. Vermia B and the Los Angeles Latino Eye study group. Myopic refractive error in adult latino. The loss Angeles Latino Eye study. Invest ophthalmolvissci. 2006;47:1845-1852.
48. Duke-Elder S. Changes in refraction in diabetes mellitus. Br J ophthalmol. 1925;9:167-187.
49. Furushima M, Imaizumi M, Nakatsuka K. Changes in refraction caused by induction of acute hyperglycaemia in healthy volunteers. JpnOphthalmol 1999;43:398-403.
50. Satio Y, Ohmi G, Kinashito S, Nakamura V, ogowa K, Harino S, Okada M. Transient hyperopia with lens swelling at initial therapy in diabetes. Br J ophthalmol 1993;77:145-148.
51. Osaguona VB, Orosaye DA. Clinical profile of optic neuritis in Nigeria. Experience at the University of Benin Teaching Hospital Nigeria. Niger J. Ophthalmol 2020;28:76-80.
52. Miller NR, Newman NJ, Biousse V, Kerrison JB. ed. Walsh and Hoyt’s clinical Neuro-ophthalmology, 6th ed. Philadephia; USA Lipincott Williams and Wilkins. 2005;293-341.

53. Shams PN, Plant GT. Optic neuritis: a review. Int. Ms J. 2009;16:82-89.
54. Nwosu SNN, Onyekwe LO. Corneal ulcer at a Nigerian eye hospital. Niger. J. Surg. Res. 2003;5:152-9.
55. Eze CN, Ogbonnaya CE, Okoye O, Ezeanosike E, Ginger-Eke H, Arinze OC. Microbial keratitis- A review of epidemiology pathogenesis, ocular manifestations and management Niger J. Ophthalmol. 2018;26:13-23.
56. Whitcher JP, Srinivasan M, Upadhyay MP. Corncal blindness. A global perspective. Bull World Health Organ. 2001;79:204-21.
57. GameiroFllho AR, Albuquerque Afd, Martins DGds et al. Epidemiological analysis of cases of uveitis in a tertiary Hospital. Revista Brasileira de aftalmologia. 2017;76:181-185.
58. Abdullaal MR, Abiad BH, Hamam RN. Uveitis in the aging eye: Incidence, patterns and differential diagnosis. Journal of Ophthalmology; 2015.
59. Zheng Y, Zhang L-x, Meng Q-L et al., Clinal patterns and characteristics of uveitis in a secondary hospital in Southern China. Int, J. Ophthalmol. 2015;8:337-341.
60. Nizamuddin SH, Bawazeer Am. Causes of uveitis in a tertiary center in Western Saudi Arabia, Saudi Medical Journal. 2013;34: 379-387.
61. Suttorp-Schultern M, Rothova A. The possible impact of uveitis in blindness. A literature survey: The british Journal of ophthalmology. 1996;80:844-848.
62. London NJ, Rathinam SR. Cunningham ET. The epidemiology of uveitis in developing countries. Int Ophthalmolclin. 2010;50:1-17.
63. Ajayi 1A, Omotoye OJ, Adeleke FO. Epidemiology of uveitis in a Nigerian Tertiary Eye care centre. J. Ophthalmic Res Ocular care. 2019;3(1):50-54.
64. Khurana AK. Disease of the uveal tract. In: KhuranaAK ed. Comprehensive Ophthalmogy 5thed. New Delh: New Age International Ltd. 2012;141-176.
65. Ochiogu BC, Udeaja AC, Ughachukwu PO. Acute red eye following the use of steroid eye drops by patients seen at the Eye Clinic, of Chukwuemeka Odumegwu Ojukwu University Teaching Hospital Amaku-Awka, South Eastern Nigeria case series. Magna Scientia Advanced Research and review. 2021;02(01):001-007.
66. Sandford SJ. Diseases of the uvea. In; SandfordSJ.ed. Eye Diseases in Hot climate 3rd ed. Torino: Butterworth-Heinemann. 1997;170-184.
67. Nwosu NN. HIV/AIDS in Ophthalmic patients. The Guinness Eye centre Onitsha experience. Niger Postgraduate Med. J. 2008;15(1):24-7.
68. Khurana AK. Diseases of orbit. In; Khurana AK ed. Comprehensive Ophthalmology 5thed. New Delh: new Age International Ltd. 2012;403-427.
69. Balogun GA, Balogun MM, Adekoya BJ. Orbital Cellulitis. Clinical course and management challenges, the Lagos State University Teaching Hospital experience. Nig QJ Hosp. Med. 2012;22(4):231.
70. Uhumwangho OM, Kayoma DH; current trends in treatment outcomes of orbital cellulitis in a Tertiary Hospital in Southern Nigeria. Niger J Surg. 2016;22(2):107-110.
71. Bekibele CO, Onabanjo AO. Orbital cellulitis. A review of 21 cases from Ibadan Nigeria. Int J clin 2003;57:14-6.
72. Nwosu SN, Nubia CA, Akudinobi CU, Okpala NE, Apakama AI. Incidence and pattern of Ophthalmic Emergencies in Onitsha Nigeria. Niger J. Ophthalmol. 2019;27-32.
73. Okosa MC, Uwakwe R, Apakama AI, Onwuegbuna AA, Uzozie CC, Amobi MBC, Uzuke CA. Psychological Effects of Eye Diseases: A Tertiary Eye Center Study. Journal of Psychiatry and Psychiatric Disorders. 2021;5:128-139.
74. Ochiogu BC, Onwuegbuna AA, Apakama AI. Reasons patients seek ophthalmic medical certificates/ Reports at the Chukwuemeka Odumegwu Ojukwu University Teaching Hospital Amaku, Awka, Nigeria. Asian Journal of Medicine and Health. 2023;21(10):217-225.

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