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CASE REPORT

Prune Belly Syndrome - A Rare Presentation in Ghanaian Neonate

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Prune Belly Syndrome (PBS) is a rare congenital anomaly characterized by deficiency or absence of abdominal muscles, cryptorchidism and severe urinary tract abnormalities. Although it is thought to be more common in people of African descent in the USA, there are few reports of the syndrome from countries in Africa including Nigeria and Rwanda. Prenatal diagnosis through ultrasonography where the cardinal signs of hydronephrosis, bilateral hydroureters, megacystis and oligohydramnios are detected is increasingly becoming the norm. However, in resource limited settings where prenatal ultrasound services are not readily accessible or available, late postnatal presentations with pulmonary hypoplasia are encountered. This study reports of a neonate who presented with difficulty in breathing and wrinkled abdomen to a tertiary center in the Northern region of Ghana.

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Keywords*:* Prune Belly Syndrome, Ghanaian neonate, abdominal wall muscle deficiency

INTRODUCTION

Prune belly syndrome (PBS), also known as Eagle-Barrett syndrome or the Triad syndrome, is a rare congenital anomaly characterized by congenital abdominal wall muscle deficiency, cryptorchidism and severe urinary tract abnormalities (Hassett *et al.*, 2012). The syndrome is sometimes termed pseudo-prune belly syndrome when it occurs in females, in view of the fact that by definition, affected females cannot have the complete triad and the urologic manifestation may be less severe (Fotter *et al.*, 2001; Kristoff *et al.*, 2012). The syndrome was first reported in the 19th century but only got its current name in the early part of the 20th century (Osler, 1901). The incidence has been reported to range between 1/29000 and 1/40000 in different studies (Woods and Brandon, 2007). About 95% of patients with PBS are male, making the syndrome an almost exclusively male disorder (Woods and Brandon, 2007; Ademola *et al.*, 2012) with the incidence being quadrupled in twins compared to single deliveries (Balaji *et al.*, 2000). PBS is thought to be more common in African-American/Afro-Caribbean population than Caucasians in the USA although there are very rare reports of the syndrome from Nigeria (Okeniyi, 2005; Ademola *et al.*, 2012). The exact etiology of the syndrome is not well defined but there are a number of theories to explain its etiopathogenesis. The mesodermal developmental defect during early pregnancy and proximal urethral obstruction are the two main theories advanced (Straub and Spranger, 1981; Moerman *et al.*, 1984) but none of these theories fully explains the syndrome.

The most common mode of diagnosis is an obstetric ultrasound scan (USG) in the second trimester of pregnancy. Features that may point to the syndrome include hydronephrosis, bilateral hydroureters, megacystis and oligohydramnios (Hoshino *et al.*, 1998; Hassett *et al.*, 2012). The initial postnatal management depends on the presentation and whether or not there is pulmonary involvement, but all surviving patients will eventually require a multi-disciplinary approach in long term management. Prognosis depends on severity of renal impairment and degree of pulmonary involvement but these have significantly improved over the years

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(Woods and Brandon, 2007). About 1/3 of all cases surviving beyond the neonatal period will need dialysis and subsequent renal transplant due to chronic renal failure (Crompton *et al.*, 1994) making renal and urinary tract care a central part of the management of these patients. Furthermore, patients may benefit from cosmetic surgery to reduce the redundant, floppy skin over the abdomen (Kristoff *et al.*, 2012).

In order to create awareness of this rare syndrome and to sensitize medical practitioners and sonographers of the importance of early referral for expert review when abnormal findings are suspected on obstetric USG, a case report of a day old neonate who was presented to the Neonatal Intensive Care Unit (NICU) of the Tamale Teaching Hospital after delivery at home due to difficulty in breathing and abnormally-appearing abdomen was reviewed.

CASE REPORT

A day old male baby who was delivered at home by spontaneous vaginal delivery (SVD) at term was brought to the NICU of the Tamale Teaching Hospital by his parents on the same day because of difficulty in breathing and abnormally-appearing abdomen. The weight at presentation was 3.2 kg and according to the mother the baby cried immediately after birth although the baby had not been fed prior to presentation at the hospital.

The mother was 30 years old, (gravida 4 para 3), with regular antenatal clinic attendance but none of the routine laboratory investigations (e.g blood group, HBsAg, G6PD, retroviral screen and VDRL) was carried out during the pregnancy. A single obstetric ultrasound scan performed in the second trimester of pregnancy at a radio-diagnostic center reported an enlarged yolk sac with sufficient liquor.

At the initial physical examination on presentation to the NICU, the weight was 3.2 kg, axillar temperature was 37.3^{o}C and SpO_2 was 37% in room air with central cyanosis. There was flaring of the alae nasi and lower chest in-drawing. Air entry was bilaterally reduced on auscultation of the chest. No cardiac murmurs were heard. The abdomen was soft, bulging to the flanks and wrinkled (Figure 1) with visible peristalsis. The baby had normal external male genitalia with bilateral undescended testes.

The patient was diagnosed as having PBS with severe respiratory distress, probably secondary to pulmonary hypoplasia. His airways were immediately suctioned to clear it of secretions and he was placed on a bubble C-PAP (Pressure of 6 cm H_2O) with which the SpO_2 rose to a maximum of 88%. The patient was also started on parenteral ampicillin (50 mg/kg, twice daily), gentamycin (3.5 mg/kg daily), intravenous fluids (1/5 normal saline) and a statum dose of 1 mg vitamin K administered intramuscularly. Eight hours into admission in the hospital, the baby's condition began to deteriorate and the SpO_2 dropped to less than 50% with bradycardia on the bubble C-PAP. Cardiopulmonary resuscitation was initiated immediately but was not successful and the baby passed away after 9 hours on admission.

DISCUSSION

PBS is a rare congenital disorder characterized by abdominal wall muscle defects, cryptorchidism and urinary tract anomalies. The condition is a predominantly male disorder as seen in the present case and others (Ademola *et al.*, 2012). It is thought to be more common in the African-American population in the USA but there has been very few reports in literature from Africa (Ademola *et al.*, 2012). The condition is commonly diagnosed through obstetric ultrasound during the second trimester of pregnancy and in most resource-limited settings this procedure is not generally available or readily accessible. Indeed, very little is known about the condition and this coupled with the fact of non-availability or ready accessibility of diagnostic tools may lead to misdiagnosis hence the resultantly low number of cases recorded in Africa. It could also be due to the negative socio-cultural beliefs that perceive babies with congenital anomalies to be bad omen to the family, precluding families from bringing them to health facilities (kotei, 1990; Okeniyi, 2005). An extensive search through available literature has yielded no published reports of PBS in Ghana and as such makes this case a novelty. Prenatal USG is by far the most common diagnostic method for this

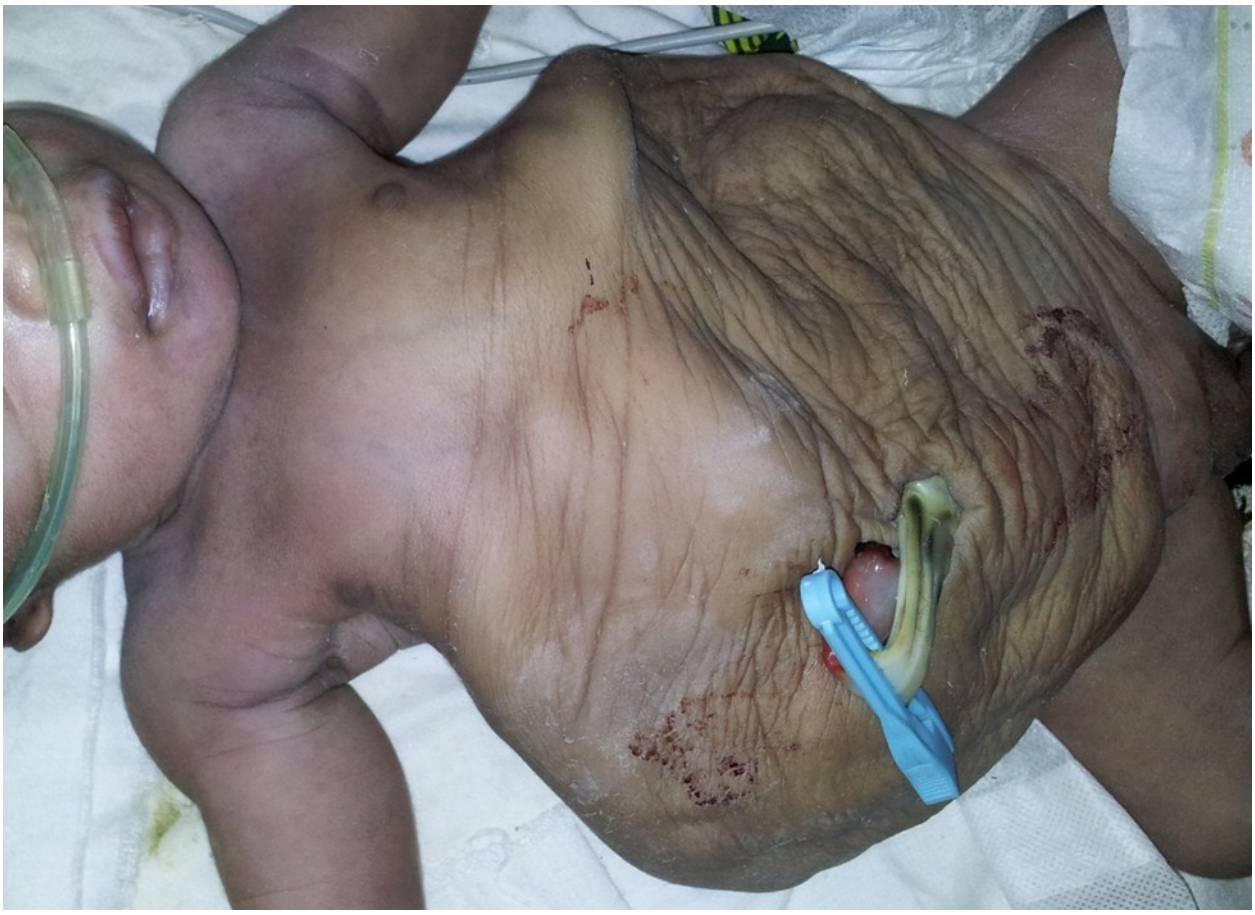

Figure 1: Wrinkled abdominal wall resembling a dry prune in a patient with PBS

complex syndrome and can detect the presence of the syndrome as early as 12-14 weeks of gestation (Hoshino *et al.*, 1998; Papantoniou *et al.*, 2010). In formulating a diagnosis though, other causes of lower urinary tract obstruction which may lead to distended bladder, megaureters and hydronephrosis should be ruled out. With respect to the case under review, the scan was carried out in a peripheral diagnostic center by a technician sonographer who made a record of and reported an enlarged yolk sac but the pregnant woman was not referred for a more detailed scan by an obstetrician until delivery. Termination of the pregnancy may be offered if diagnosis is made before viability (Hoshino *et al.*, 1998; Agarwal, 2005; Papantoniou *et al.*, 2010) and rightly so, in this case,the prenatal diagnosis could have helped in taking a decision regarding termination of the pregnancy.

Pulmonary hypoplasia, associated with approximately 60% of all cases, is the most common respiratory condition encountered in PBS (Hassett *et al.*, 2012; Tonni *et al.*, 2013). It is thought to be secondary to oligohydramnios which was one of the features of this syndrome (Ome *et al.*, 2013). Although the only obstetric USG performed during the second trimester in this case did not report oligohydramnios, the patient presented hours after delivery with severe respiratory distress, central cyanosis and very low oxygen saturation in room air. Majority of newborns with PBS who have pulmonary hypoplasia would die within the first week of life and in conformity to this fact, the patient in the present case died 9 hours into admission due to severe respiratory distress.

In Prune Belly syndrome, the presence of pulmonary hypoplasia in addition to severe renal dysfunction is a predictor of very high mortality in early days after birth (Woods and Brandon, 2007; Hassett *et al.*, 2012; Tonni *et al.*, 2013). Home delivery, inadequate management and delayed presentation as was done in this case might have contributed significantly to the high mortality.

CONCLUSION

Prenatal diagnosis through USG is the contemporary practice and needs to be enhanced particularly in resource-limited settings in order to increase availability and ready accessibility. Sonographers should be encouraged to refer suspicious cases for more detailed scans and expert opinions. In cases where early diagnosis is made in gestation, termination of pregnancy could be offered. Furthermore, early detection and management of pulmonary hypoplasia which is one of the predictors of early and high mortality in Prune Belly Syndrome is very vital to survival.

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COMPETING INTERESTS

The authors declare that they have no competing interests.

REFERENCE

Ademola A., Ofovwe G. and Ibadin M. (2012) Familial prune belly syndrome in a Nigerian family. *Saudi J Kidney Dis Transpl* 23, 338-342.

Agarwal R. (2005) prenatal diagnosis of abdominal wall defects. *indian journal of radiological imaging* 15, 361-372.

Balaji K., Patil A., Townes P., Primack W. and Skare J. (2000) Concordant prune belly syndrome in monozygotic twins. *urology* 55.

Crompton C.H., Balfe J.W. and Khoury A. (1994) Peritoneal dialisis in the prune belly syndrome. *peritoneal dialisis international* 14, 17-21.

Fotter R., Rascher W., Rupprecht T. and RÃsch W. (2001) Prune Belly Syndrome. In *Pediatric Uroradiology*, pp. 177-184: Springer Berlin Heidelberg.

Hassett S., Smith G.H. and Holland A.J. (2012) Prune belly syndrome. *Pediatr Surg Int* 28, 219-228.

Hoshino T., Ihara Y., Shirane H. and Ota T. (1998) Prenatal diagnosis of prune belly syndrome at 12 weeks of pregnancy: case report and review of the literature. *Ultrasound Obstet Gynecol* 12, 362-366.

kotei D.A. (1990) belated diagnosis of congenital anomalies. *Pediatr Surg Int* 5, 412-415.

Kristoff V., Gregory V.E., Veerle D.B. and Paul W. (2012) The prune belly syndrome: report of a rare case and review of literature. *European Journal of Plastic Surgery* 35, 241-243.

Moerman P., Fryns J.P., Goddeeris P. and Lauweryns J.M. (1984) Pathogenesis of the Prune-Belly Syndrome: A Functional Urethral Obstruction Caused by Prostatic Hypoplasia. *PEDIATRICS* 73, 470-475.

Okeniyi J.A., Ogunlesi T.A, Dedeke I.O, Oyelami O.A and Oyedeji G.A (2005) Prune belly syndrome in a nigerian child. *internet journal of pediatrics and neonatology* 5.

Ome M., Wangnapi R., Hamura N., Umbers A.J., Siba P., Laman M., Bolnga J., Rogerson S. and Unger H.W. (2013) A case of ultrasound-guided prenatal diagnosis of prune belly syndrome in Papua New Guinea - implications for management. *BMC Pediatr* 13, 70.

Osler W. (1901) congenital absence of abdominal wall muscles with distended and hypertrophied bladder. *bulletin of john hopkins hospital* 12.

Papantoniou N., Papoutsis D., Daskalakis G., Chatzipapas I., Sindos M., Papaspyrou I., Mesogitis S. and Antsaklis A. (2010) Prenatal diagnosis of prune-belly syndrome at 13 weeks of gestation: case report and review of literature. *J Matern Fetal Neonatal Med* 23, 1263-1267.

Straub E. and Spranger J. (1981) Etiology and path-

ogenesis of the prune belly syndrome. *kidney international* 20, 695-699.

Tonni G., Ida V., Alessandro V. and Bonasoni M.P. (2013) Prune-belly syndrome: case series and review of the literature regarding early prenatal diagnosis, epidemiology, genetic factors, treatment, and prognosis. *Fetal Pediatr Pathol* 31, 13-24.

Woods A.G. and Brandon D.H. (2007) Prune belly syndrome. A focused physical assessment. *Adv Neonatal Care* 7, 132-143; quiz 144-135.

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ORIGINAL ARTICLE

Preliminary Phytochemical Screening and *In vitro* Antioxidant Properties of *Trichilia monadelpha* (Thonn.) J. J. de Wilde (*Meliaceae*)

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The study evaluated the antioxidant potential and phytochemical constituents in the stem bark extracts of *Trichilia monadelpha* (Thonn) JJ De Wilde (Family: Meliaceae). Petroleum ether (PEE), ethyl acetate (EthE) and ethanol extracts (EAE) of the stem bark of *T. monadelpha* were screened for the presence of phytochemicals. The *in vitro* antioxidant potential of the extracts were also determined using the reducing power and 2, 2-diphenyl-1-picryl-hydrazyl radical (DPPH) scavenging tests respectively. Total phenol content of the extracts was also estimated. Phytochemical analysis revealed the presence of important secondary metabolites. Alkaloids, terpenoids, phytosterols, reducing sugars and coumarins were present in PEE. EAE had tannins, alkaloids, terpenoids, phytosterols, reducing sugars, flavonoids, cardiac glycosides, anthraquinones and saponins whiles EthE contained tannins, alkaloids, reducing sugars, cardiac glycosides, anthraquinones, terpenoids and phytosterols. Total phenol contents were estimated to be 7.51±0.87 mg tannic acid equivalent/g of petroleum ether extract, 34.14±0.78 mg tannic acid equivalent/g of ethyl acetate extract and 119.30±3.20 mg tannic acid equivalent/g of hydroethanol extract. The extracts showed a concentration-dependent reduction of Fe^{3+} to Fe^{2+} in the reducing power test as well as concentration-dependent DPPH radical scavenging. Of the three extracts, EAE had the most antioxidant activity. Findings of this study suggests that the stem bark of *Trichilia monadelpha* may be a good source of natural antioxidants and might be useful in treating the diseases associated with oxidative stress.

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Keywords*:* Phytochemicals, total phenol content, reducing power, DPPH, antioxidant, free radicals.

INTRODUCTION

Free radicals and reactive oxygen species have received a lot of attention especially in experimental or clinical medicine and biology because of their role in the aetiology of various chronic and degenerative diseases, including aging, coronary heart disease, inflammation, stroke, diabetes mellitus and cancer (Halliwell, 2011; Halliwell, 2012; Halliwell *et al.*, 1992). The damaging effects of reactive oxygen species (e.g. singlet oxygen, superoxide, peroxyl radicals, hydroxyl radicals and peroxynitrite) on cells has been shown to be abrogated by plants with antioxidant compounds (Dasgupta *et al.*, 2007). Plants are endowed with antioxidant and free radical scavenging molecules including vitamins, terpenoids, phenolic acids, tannins, flavonoids, coumarins, and other secondary metabolites. The search for compounds, that can protect the human body from oxidative damage and retard the progress of many chronic diseases, has therefore greatly focused on plant sources as they produce significant amount of antioxidants and represent a potential source of new compounds with antioxidant activity.

Trichilia monadelpha (Thonn) JJ De Wilde (Family: Meliaceae), known locally as Otanduro (Twi) or Tenuba (Nzema), is a tree that grows 12-20 m high and establishes itself well in the lowland high forest and evergreen semi-deciduous secondary jungles, often near river banks (Abbiw, 1990). Preparations

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(decoctions, infusions and tinctures) of the stem bark of the plant have been used in Ghanaian traditional medicine to treat pain, psychoses, epilepsy and inflammation for many years and their efficacies are widely acclaimed in different communities in Ghana (Abbiw, 1990; Dokosi, 1998). Pharmacological studies have revealed that the ethanolic stem bark extract of *T. monadelpha* has anti-trypanosomal and antiplasmodial activities (Kamanzi Atindehou *et al.*, 2004). Also, various stem bark extracts of the plant have been shown to have anti-inflammatory (Ainooson *et al.*, 2012) and analgesic (Woode *et al.*, 2012) properties. In the present study, the phytochemical constituents and the *in vitro* antioxidant and free radical scavenging potential of stem bark extracts of *Trichilia monadelpha* are determined.

MATERIALS AND METHODS

Chemicals

Acetic anhydride, ammonia, chloroform, Dragendorff's reagent, ethanol, ferric chloride, gelatin, hydrochloric acid, lead acetate, magnesium metal strips, methanol, *n*-propyl gallate, potassium ferricyanide, sodium chloride, sodium carbonate, sulphuric acid, sodium hydroxide and tannic acid were obtained from British Drug House (BDH) Ltd (Poole, England) while2, 2-diphenyl-1-picrylhydrazyl (DPPH), trichloroacetic acid (TCA), Folin-Ciocalteau reagent and Wagner's reagent were purchased from Sigma-Aldrich Inc. (St. Louis, MO, USA). All chemicals were of highest purity (≥ 99.0%).

Plant collection

The stem bark of *Trichilia monadelpha* was obtained from Bomaa (7°05'06.82" N; 2°10'01.63" W) in the Tano North District of the Brong Ahafo Region of Ghana between August and October, 2010. The leaves of the plant were authenticated by Dr. Kofi Annan of the Department of Herbal Medicine, Faculty of Pharmacy and Pharmaceutical Sciences, College of Health Sciences, Kwame Nkrumah University of Science and Technology (KNUST), Kumasi, Ghana. A voucher specimen was kept in the Faculty of Pharmacy Herbarium (No. FP/079/10).

Preparation of stem bark extracts

The plant bark was chopped into pieces, sun dried for fourteen days and pulverized into fine powder. The powdered plant bark was serially extracted with 40-60ºC petroleum ether, ethyl acetate and 70% ethanol over a 24-hour period using the Soxhlet apparatus. The extracts obtained were labelled as follows: EAE (ethanol extract), PEE (petroleum ether extract) and EthE (ethyl acetate extract). The resulting extracts were concentrated under reduced pressure at 40-60˚C to a dark brown syrupy mass in a rotary evaporator. The syrupy mass was further dried using water bath and kept in a desiccator. The final yields were 9.6 % (EAE), 0.9 % (PEE), and 0.7 % (EthE).

Phytochemical analysis

Phytochemical analysis of the extracts was performed according to standard methods (Kokate, 2005; Tiwari *et al.*, 2011; Trease *et al.*, 1989).

Test for Tannins and Phenolic compounds

About 0.5 g of each of the plant extract was boiled with 25 ml of water for 5 minutes. It was then cooled, filtered and the volume adjusted to 25 ml.

Lead acetate test: To 1 ml aliquot of each of the extracts, 10 ml of water and 5 drops of 1% lead acetate solution was added. The formation of white precipitate indicated the presence of tannins (Kokate, 2005).

Ferric chloride test: To 1 ml aliquot of each of the extracts 3-4 drops of neutral 5% ferric chloride solution was added. Formation of dark green colour indicated the presence of phenols (Kokate, 2005).

Gelatin test: To about 1 g of each of the extracts, 1% gelatin solution containing sodium chloride was added. Formation of white precipitate indicated the presence of tannins (Tiwari *et al.*, 2011).

Test for Alkaloids

Five (5) grams of each of the extracts was stirred with 5 ml of 1% aqueous hydrochloric acid (HCl) on water bath and then filtered. Of the filtrates, 1 ml of each extract filtrates were taken into test tubes to be tested for the presence of alkaloids.

Dragendroff's test: To 1 ml of each of the extracts, 1 ml of Dragendroff's reagent (potassium bismuth

iodide solution) was added. An orange-red precipitate indicated the presence of alkaloids.
Wagner's test: To 1 ml of each of the extracts, 2 ml of Wagner's reagent (iodine in potassium iodide) was added. A reddish brown coloured precipitate indicated the presence of alkaloids.

Test for carbohydrates
One gram of each of the extracts were dissolved individually in 5 ml distilled water and filtered. The filtrates were used to test for the presence of carbohydrates (Tiwari *et al.*, 2011).
Benedict's test: 1 ml of each of the filtrates were added to 5 ml Benedict's reagent and heated gently for 2 minutes and cooled. Orange red precipitate indicated the presence of reducing sugars.
Fehling's Test: 1 ml of each of the filtrates was hydrolyzed with dilute HCl, neutralized with alkali and heated with Fehling's A & B solutions. Formation of red precipitate indicated the presence of reducing sugars.

Test for Phytosterols
Salkowski's test: One gram of each of the extracts were dissolved in 10 ml of chloroform and filtered. The filtrates were treated with few (3-4) drops of concentrated sulphuric acid, shaken and allowed to stand. Appearance of golden yellow colour indicated the presence of triterpenes (Tiwari *et al.*, 2011).
Libermann-Burchard's test: One gram of each of the extracts was dissolved in few drops of chloroform, 3 ml of glacial acetic acid and 3 ml of acetic anhydride were added. This solution was warmed and cooled under running tap water. Few drops of concentrated sulphuric acid were added along the side of the test tubes. The appearance of a reddish violet colour at the junction of the two layers and a bluish green colour in the acetic acid layer indicates the presence of unsaturated sterols and/or triterpenes (Wall *et al.*, 1954).

Test for Flavonoids
Shinoda's test: About 1 g of each of the extracts was further dissolved with 5 ml of ethanol (98 %). To this was added a small piece of magnesium foil metal, this was followed by drop wise addition of concentrated hydrochloric acid. Intense cherry red colour indicated the presence of flavonones. Orange red colour indicated the presence of flavonols (Brain *et al.*, 1975).
Lead acetate test: Few drops of lead acetate solution were added to each of the extracts in test tubes. Formation of yellow coloured precipitate indicated the presence of flavonoids (Tiwari *et al.*, 2011).

Test for Coumarins
In a test tube, 1 g of each of the extracts were placed and covered with filter paper moistened with dilute sodium hydroxide (NaOH), then heated on water bath for a few minutes. The filter paper was examined under UV light, yellow fluorescence indicated the presence of coumarins (El-Tawil, 1983).

Test for Glycosides
Extracts were hydrolyzed with dilute HCl, and then subjected to test for glycosides.
Keller-Killiani's test: One ml of each of the extracts was mixed with 5 ml of 70% alcohol for 2 minutes. This was filtered and to the filtrates was added 10 ml of water and 0.5 ml of lead acetate. This was filtered and the filtrate was shaken with 5 ml of chloroform. The chloroform layers were separated in a porcelain dish and the solvent removed by evaporation. This was cooled and dissolved in 3 ml glacial acid containing 2 drops of 5 % ferric chloride solution. The solution was carefully transferred to the surface of 2 ml concentrated sulphuric acid. A reddish brown layer formed at the junction of the two liquids and the upper layer which slowly became bluish green and darkening with standing indicated the presence of cardiac glycosides (Harborne, 1998).
Borntrager's test: Few drops of dilute sulphuric acid were added to 1 ml of each of the extracts. This was boiled and filtered. The filtrate was extracted with chloroform. The chloroform layer was treated with 1 ml of ammonia. The formation of red colour on the ammoniacal layer showed the presence of anthraquinone glycosides (Harbourne, 1984; Sofowora, 1993).

Test for Saponins
Froth Test: Extracts (1 g) were diluted with distilled water to 20 ml and this was shaken in a gradu-

ated cylinder for 15 minutes. Formation of 1 cm layer of foam indicated the presence of saponins (Tiwari *et al*., 2011).

Foam Test: The extract (0.5 g portions) was shaken with 2 ml of water. Foam produced which persisted for ten minutes indicated the presence of saponins (Trease *et al*., 1983).

In vitro anti-oxidant assay

Total Phenolic Content

The total soluble phenolic content of the three extracts (0.3-1 mg ml^{-1}) were quantified using the Folin -Ciocalteu's phenol reagent (Singleton and Rossi, 1965) with tannic acid (0.01-0.1 mg ml^{-1}) as standard. The extracts (1 ml) were added to 1 ml Folin-Ciocalteu's reagent (diluted tenfold in distilled water) in separate test tubes. The content of each test tube was mixed and allowed to stand for five minutes at 25°C in an incubator. One millilitre (1 ml) of 2 % sodium carbonate solution (Na_2CO_3) was added to the mixture. This was allowed to stand for 2 hours at 25°C in an incubator and centrifuged at 1000 ×*g* for 10 minutes to get a clear solution. The absorbance of the supernatant was then determined at 760 nm using UV mini-1240 single beam spectrophotometer (Shimadzu Scientific Instruments, Kyoto, Japan). Distilled water (1 ml) was added to 1 ml Folin-Ciocalteu's reagent (diluted ten-fold in distilled water) processed in the same way as done for the test samples and used as blank. All measurements were done in triplicates. The total phenolics were expressed as milligrams per milliliter of tannic acid equivalents (TAEs) through the calibration curve with tannic acid.

Reducing power

The reducing power of the three extracts (0.1-3 mg ml^{-1}) was determined according to the method of Oyaizu (1986), with tannic acid (0.1-3 mg ml^{-1}) as a reference antioxidant. The reference antioxidant/ extract (1 ml) was mixed with 2.5 ml of 0.2 M sodium phosphate buffer (pH 6.6) and 2.5 ml of 1 % potassium ferricyanide solution ($K_3Fe[CN]_6$) in a test tube. The mixture was incubated at 50°C for 20 minutes. Following this, 1.5 ml of 10% trichloroacetic acid solution (TCA) was added to the incubated mixture, and centrifuged at 865 ×*g* for 10 min. The supernatant (2.5 ml) was then mixed with 2.5 ml distilled water and 0.5 ml of 0.1 % ferric chloride solution in a test tube. The absorbance was measured at 700 nm using UV mini-1240 single beam spectrophotometer (Shimadzu Scientific Instruments, Kyoto, Japan). The blank was prepared by adding distilled water (1 ml) to 2.5 ml sodium phosphate buffer and 2.5 ml 1% potassium ferricyanide ($K_3Fe[CN]_6$) in a test tube. Three replicates were used. Results were then expressed as percentages of blank and presented as concentration-absorbance curves.

DPPH Scavenging Activity

The scavenging of the stable 2, 2-diphenyl-1-picrylhydrazil (DPPH) radical is a widely used method to evaluate the free radical scavenging ability of various samples, including plant extracts (Chang *et al*., 2002). The experiment was carried out as described in literature (Blois, 1958) with a few modifications. The extracts (0.1-3 mg ml^{-1} in methanol) were compared to *n*-propyl gallate (0.01-0.3 mg ml^{-1} in methanol) as standard free radical scavenger. The extracts (1 ml) were added to 3 ml methanolic solution of DPPH (20 mgl^{-1}) in a test tube. The reaction mixture was kept at 25°C for 1 h in an orbital shaker (BoroLabs, Aldermaston Berkshire, UK). The absorbance of the residual DPPH was determined at 517 nm in UV mini-1240 Single beam Spectrophotometer (Shimadzu Scientific Instruments, Kyoto, Japan). Methanol (99.8%, 1 ml) was added to 3 ml DPPH solution, incubated at 25°C for 1 h and used as control.. Methanol (99.8%) was used as blank. Each experiment was carried out in triplicates. The percentage radical scavenging capacity was determined using the following formula:

$$\% \text{ DPPH Scavenging} = [(A_0-A_s)/A_0] \times 100$$

where A_0 is the absorbance of control (DPPH in methanol), and A_s is the absorbance of tested samples.

A graph was plotted with concentration along *X*-axis and % DPPH scavenging along *Y*-axis, and IC_{50} value was calculated. IC_{50} value signifies the concentration of tested samples that scavenges 50% of the DPPH radical.

Data Analysis
All experiments were conducted in triplicates, and the data are expressed as Mean ± S.E.M. Concentration responsible for 50% of the maximal effect (EC_{50}/IC_{50}) was determined by using an iterative computer least squares method, with the following non-linear regression (three-parameter logistic) equation

$$Y = \frac{a + (b - a)}{\left(1 + 10^{(LogED_{50} - X)}\right)}$$

Where, *X* is the logarithm of dose and *Y* is the response. *Y* starts at *a* (the bottom) and goes to *b* (the top) with a sigmoid shape.The fitted midpoints (ED_{50}s) of the curves were compared statistically using *F* test (Motulsky & Christopulos., 2003).GraphPad Prism for Windows version 5.0 (GraphPad Software, San Diego, CA, USA) was used for data analysis and EC_{50}/IC_{50}determinations. $P<0.05$ was considered statistically significant.

RESULTS

Phytochemical Analysis

Table 1 shows the phytochemical constituents of the various extracts of *T. monadelpha*. Alkaloids, terpenoids, phytosterols, reducing sugars and coumarins were present in PEE. EAE had tannins, alkaloids, terpenoids, phytosterols, reducing sugars, flavonoids, cardiac glycosides, anthraquinones and saponins whiles EthE contained tannins, alkaloids, reducing sugars, cardiac glycosides, anthraquinones, terpenoids and phytosterols.

Total Phenolic Content

Results of the assay of total phenolics in the extracts are shown in Table 2. There was a concentration-dependent increase in the total phenolics in all the extracts when expressed as tannic acid equivalents. Total phenolics were estimated to be 7.51 ± 0.87 mg tannic acid equivalent/g of petroleum ether extract, 34.14 ± 0.78 mg tannic acid equivalent/g of ethyl acetate extract and 119.30 ± 3.20 mg tannic acid equivalent/g of hydroethanol extract. EAE had the highest phenolic content and PEE the least (Figure 1).

Table 1: Phytochemical constituents of stem bark extract of *T. monadelpha*

TESTS	PEE	EthE	EAE
Tannins and Phenolic compounds			
Lead acetate test	-	+	++
$FeCl_3$ test	-	-	++
Gelatin test	-	+	++
Alkaloids			
Dragendroff's test	+	+	+
Wagner's test	+	+	+
Phytosterols/ triterpenoids			
Salkowski's test	++	++	+
Libermann-Burchard's test	++	++	+
Carbohydrates			
Benedict's test	+	+	+
Fehling's test	+	+	+
Flavonoids			
Shinoda's test	-	-	++
Lead acetate test	-	-	++
Coumarins			
Test for coumarins	+	-	-
Cardiac glycosides			
Keller-Killiani's test	-	+	++
Anthraquinones			
Borntrager's test	-	+	++
Saponins			
Frost test	-	-	++
Foam test	-	-	++

–: Not detected, +: Present in low concentration, ++: Present in moderate concentration.

Reducing power

EAE, EthE and tannic acid exhibited significant concentration-dependent reducing activity with EC_{50} values (in mg ml^{-1}) of 0.87±0.11, 13.63±0.38 and 1.04±0.26 respectively (Figure 2; Table 3). PEE, however, showed very weak reducing power (EC_{50}: 81.06±4.35; Figure 2, Table 3).

DPPH scavenging activity

The DPPH scavenging ability of the extracts are shown in figure 3. All extracts exhibited concentra-

Table 2: Total phenol content of the extracts of *T. monadelpha,* expressed as milligram tannic acid equivalent per gram of extract.

Extract	Total Phenol Content (mg TAE / g of extract)
PEE	7.51 ± 0.87
EthE	34.14 ± 0.78
EAE	119.30 ± 3.20

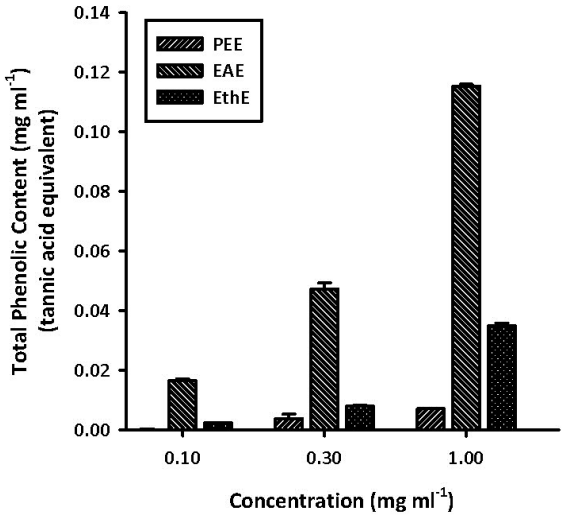

Figure 1: Total phenols (expressed as tannic acid equivalents) present in various concentrations of PEE (0.1-1 mg ml^{-1}), EAE (0.1-1 mg ml^{-1}) and EthE (0.1-1 mg ml^{-1}). Each column represents mean ± S.E.M. (n= 3).

Table 3: EC_{50} values for extracts of *T. monadelpha* and tannic acid in the reducing power assay.

Extract/ standard	Reducing Power	$F_{1,46}$	P value
PEE	81.06 ± 4.35***	317.10	<0.0001
EthE	13.63 ± 0.38***	300.20	<0.0001
EAE	0.87 ± 0.11***	81.21	<0.0001
Tannic acid	1.04 ± 0.26	-	

Values are EC_{50}± S.E.M ***P<0.001 compared to EC_{50} of tannic acid.

Table 4: IC_{50} values for extracts of *T. monadelpha* and *n*-propyl gallate in the DPPH assay.

Extract/ standard	DPPH scavenging	$F_{1,28}$	P value
PEE	0.24 ± 0.04***	50.21	<0.0001
EthE	0.08 ± 0.01***	12.07	0.0017
EAE	0.04 ± 0.04^{ns}	1.69	0.2047
n-propyl gallate	0.02 ± 0.01	-	

Values are IC_{50}± S.E.M ***P<0.0001; ^{ns}P>0.05 compared to IC_{50} of propyl gallate.

tion-dependent scavenging activity in a similar manner to the reference antioxidant, n-propyl gallate (figure 3). The IC_{50} correlates directly with the effectiveness of the substrate/extract to scavenge the DPPH radical. The IC_{50} values (in mg ml^{-1}) obtained (table 4) suggests PEE has the least ability to scavenge free radicals compared to *n*-propyl gallate.

DISCUSSION

Preliminary phytochemical analysis of the various extracts of *T. monadelpha* demonstrated the presence of saponins, tannins, alkaloids, cardiac glycosides, anthraquinones, reducing sugars, flavonoids, coumarins, triterpenoids and steroidal compounds. Of these, PEE indicated the presence of alkaloids, sterols, triterpenoids, reducing sugars and coumarins. EthE contained reducing sugars, sterols, triterpenoids, tannins, alkaloids, cardiac glycosides and anthraquinones while EAE indicated the presence of all the compounds listed except coumarins. The results obtained confirm earlier reports of some of the phytochemical constituents found in extracts of *T. Monadelpha* (Ainooson *et al.*, 2012; Woode *et al.*, 2012). This study, however, reports for the first time, the presence of coumarins in PEE; cardiac glycosides and anthraquinones in EAE and EthE.

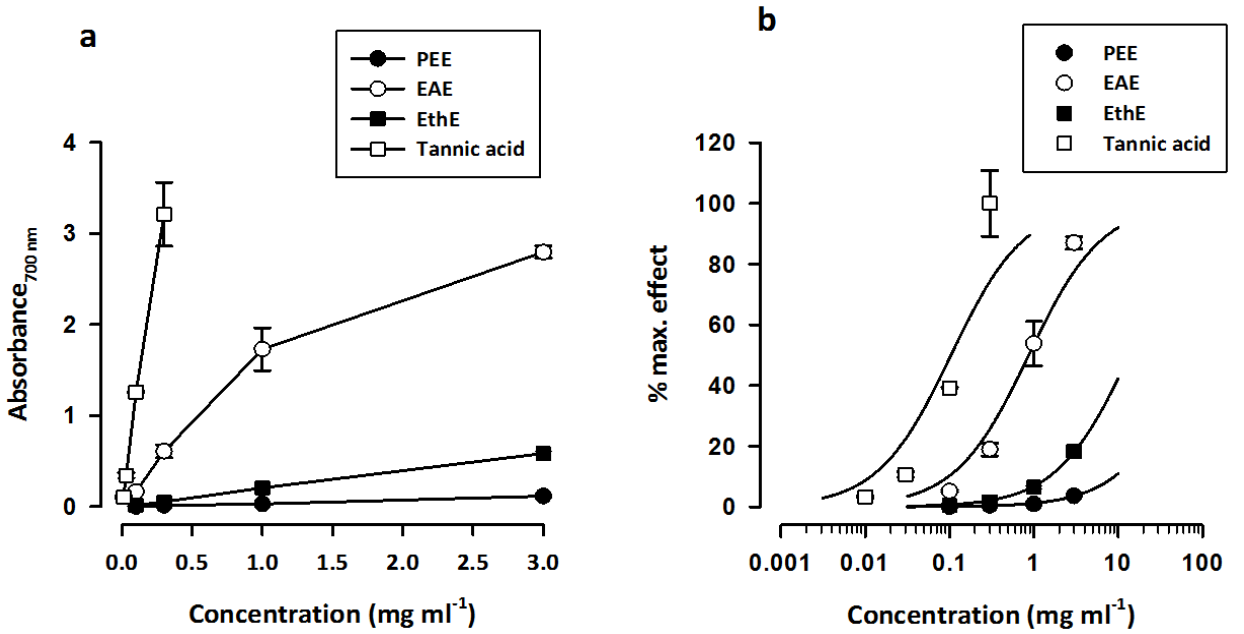

Figure 2: Change in (a) Absorbance and (b) maximal reducing power of the three extracts (0.1-3 mg ml^{-1}) of *Trichilia monadelpha* compared to tannic acid (0.1-3 mg ml^{-1}). Each point represents mean ± S.E.M. (n= 3).

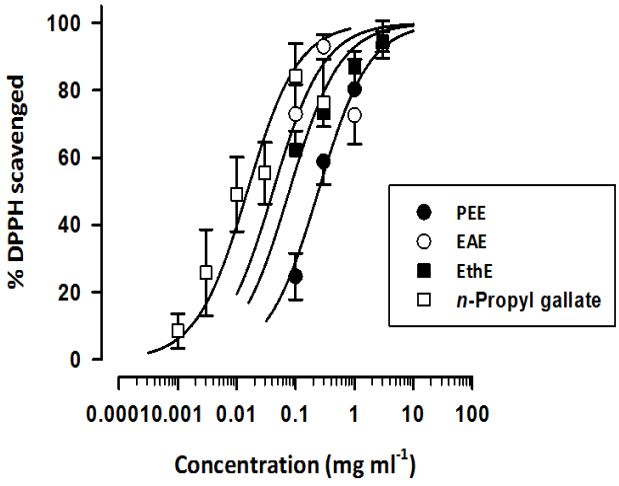

Figure 3: Free radical scavenging ability of the extracts, PEE, EthE, EAE (0.1-3 mg ml^{-1}) compared to *n*-propyl gallate (0.01-0.3 mg ml^{-1}) in the DPPH radical assay. Each point represents the mean ± S.E.M (n = 3).

The phytochemical constituents detected in the three extracts of *T. monadelpha* could contribute to the traditional therapeutic use of the whole stem bark. Phenolic compounds are one of the largest and most ubiquitous groups of plant metabolites and possess diverse biological properties including anti-apotosis, anti-aging, anti-carcinogen, anti-inflammation, anti-atherosclerosis, cardiovascular protection, inhibition of angiogenesis and cell proliferation as well as the improvement of endothelial function (Han *et al.*, 2007). Tannins have also received immense attention in many fields especially in the fields of nutrition, health and medicine due to their physiological activities (e.g. antioxidant, antimicrobial and anti-inflammatory activity)(Mota *et al.*, 1985; Lin *et al.*, 2001; Buzzini *et al.*, 2008; Koleckar *et al.*, 2008). Flavonoids, a group of polyphenolics, are free radical scavengers, super antioxidants which have anti-inflammatory activity, prevent oxidative cell damage through their water soluble property and also possess strong anti-cancer activity (Gurib-Fakim, 2006; Salah *et al.*, 1995). Coumarins are potential antioxidants, according to stud-

ies (Tseng, 1991; Kostova, 2006; Kostova *et al.,* 2011), with the ability of scavenging free radicals and chelating metal ions. Triterpenoids possess analgesic and anti-inflammatory properties (Savithramma *et al.*, 2012). More research is required to determine the specific roles of these phytochemical constituents present in *Trichilia monadelpha*.

The reducing power and DPPH scavenging tests conducted in this study sought to establish the *in vitro* antioxidant properties of the plant extracts. The detection of phenolic compounds, particularly in EAE and EthE, strongly suggests possible antioxidant activity of the extracts. Phenolic antioxidants are potent free radical terminators (Shahidi *et al.*, 1992). The high potential of phenolic compounds to scavenge radicals may be explained by their phenolic hydroxyl groups (Sawa *et al.*, 1999).

Reducing power assay is a convenient and rapid screening method for measuring the antioxidant potential (Meir *et al.*, 1995) of a substance. From the results, there was significant, concentration-dependent Fe^{3+} reducing activity by EAE and EthE compared with tannic acid. The findings further affirm the antioxidant activity of the extracts.

The DPPH test is widely used as measure for the electron donation capacity of the antioxidant under the assay conditions. DPPH (2, 2-diphenyl-1-picryl hydrazyl) is a stable free radical that accepts an electron or hydrogen radical to become a stable diamagnetic molecule. The DPPH radical scavenging ability of EAE was significantly as potent as that of the standard, *n*-propyl gallate, but PEE and EthE were less effective in scavenging the DPPH radical. The DPPH scavenging ability of EAE shows the extract could serve as a free radical inhibitor or scavenger.

From the activities shown by the extracts, particularly EAE, in the antioxidant tests conducted, it is clear that extracts have antioxidant activity. The mechanism of antioxidant activity may be due to the reduction of free radicals as well as scavenging of reactive oxygen species and other free radicals. EAE had the highest amounts of phenolics and hence it is not surprising that it also exhibited most reducing power and free radical scavenging ability. The antioxidant activities observed in this study could account, partly, for the anti-inflammatory effect observed in an earlier study on *T. monadelpha* (Ainooson *et al.*, 2012) since a large pool of evidence implicates free radicals in the process of inflammation (Closa *et al.*, 2004; Conner *et al.*, 1996; Reuter *et al.*, 2010).Further studies are required to clarify the *in vivo* potential of *T. monadelpha* in the management of human diseases resulting from oxidative stress.

CONCLUSION

The current study has shown that the petroleum ether, ethyl acetate and ethanol extracts of the stem bark of *Trichlia monadelpha* containsaponins, tannins, alkaloids, cardiac glycosides, anthraquinones, reducing sugars, flavonoids, coumarins, triterpenoids and steroidal compounds. The extracts also possess antioxidant and radical scavenging properties *in vitro*.

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COMPETING INTERESTS

The authors declare that they have no competing interests.

REFERENCES

Abbiw DK (1990). *Useful Plants of Ghana*. edn. Intermediate Technology Publications and Royal Botanic Gardens Kew.

Ainooson GK, Owusu G, Woode E, Ansah C, Annan K (2012). Trichilia monadelpha Bark Extracts Inhibit Carrageenan-Induced Foot-Oedema in the 7-Day Old Chick and the Oedema Associated with Adjuvant-Induced Arthritis in Rats. *Afr. J. Tradit. Complement. Altern. Med.***9**(1)**:** 8-16.

Blois MS (1958). Antioxidant determinations by the use of a stable free radical. *Nature***181:** 1199-1200.

Brain KR, Turner TD (1975). *The practical evaluation*

of phytochemicals. . Wright Science Technical: Bristol.

Buzzini P, Arapitsas P, Goretti M, Branda E, Turchetti B, Pinelli P, Ieri F., Romani A. (2008). Antimicrobial and antiviral activity of hydrolysable tannins. *Mini Rev. Med. Chem.***8**(12)**:** 1179-1187.

Chang C-C, Yang M-H, Wen H-M, Chern J-C (2002). Estimation of total flavonoid content in propolis by two complementary colorimetric methods. *J. Food Drug Analysis***10**(3)**:** 178-182.

Closa D, Folch-Puy E (2004). Oxygen free radicals and the systemic inflammatory response. *IUBMB Life***56**(4)**:** 185-191.

Conner EM, Grisham MB (1996). Inflammation, free radicals, and antioxidants. *Nutrition***12**(4)**:** 274-277.

Dasgupta N, De B (2007). Antioxidant activity of some leafy vegetables of India: A comparative study. *Food Chemistry***101**(2)**:** 471-474.

Dokosi OB (1998). *Herbs Of Ghana*. 1 edn. Ghana Universities Press: Accra.

El-Tawil BDH (1983). Chemical constituents of indigenous plants used in Native Medicines of Saudi Arabia, II. *Arab Gulf J Sci Res***A1**(12)**:** 395 -410.

Gurib-Fakim A (2006). Medicinal plants: traditions of yesterday and drugs of tomorrow. *Mol. Aspects Med.***27**(1)**:** 1-93.

Halliwell B (2011). Free radicals and antioxidants - quo vadis? *Trends Pharmacol. Sci.***32**(3)**:** 125-130.

Halliwell B (2012). Free radicals and antioxidants: updating a personal view. *Nutr. Rev.***70**(5)**:** 257 -265.

Halliwell B, Gutteridge JM, Cross CE (1992). Free radicals, antioxidants, and human disease: where are we now? *J. Lab. Clin. Med.***119**(6)**:** 598-620.

Han X, Shen T, Lou H (2007). Dietary Polyphenols and Their Biological Significance. *Int. J. Mol. Sci.***8**(9)**:** 950-988.

Harborne JB (1998). *Phytochemical Methods—A Guide to Modern Techniques of Plant Analysis*. 1st edn: Chapman and Hall; London, UK.

Harbourne JB (1984). Phytochemical Methods: A Guide to Modern Technique of Plant Analysis, 2nd ed. Chapman & Hall, London. 282.

Kamanzi Atindehou K, Schmid C, Brun R, Kone MW, Traore D (2004). Antitrypanosomal and antiplasmodial activity of medicinal plants from Cote d'Ivoire. *J. Ethnopharmacol.***90**(2-3)**:** 221-227.

Kokate CK (2005). *A Textbook for Practical Pharmacognosy*. 5th Ed edn.

Koleckar V, Kubikova K, Rehakova Z, Kuca K, Jun D, Jahodar L, Opletal, L. (2008). Condensed and hydrolysable tannins as antioxidants influencing the health. *Mini Rev. Med. Chem.***8**(5)**:** 436-447.

Kostova I, Bhatia S, Grigorov P, Balkansky S, Parmar VS, Prasad AK, Saso L. (2011). Coumarins as antioxidants. *Curr Med Chem*,*18*(25), 3929-3951.

Kostova I. (2006). Synthetic and natural coumarins as antioxidants. *Mini Rev Med Chem*, *6*(4), 365 -374.

Lin CC, Hsu YF, Lin TC (2001). Antioxidant and free radical scavenging effects of the tannins of Terminalia catappa L. *Anticancer Res.***21** (1A)**:** 237-243.

Mota ML, Thomas G, Barbosa Filho JM (1985). Anti-inflammatory actions of tannins isolated from the bark of Anacardium occidentale L. *J. Ethnopharmacol.***13**(3)**:** 289-300.

Meir S, Kanner J, Akiri B, Hadas SP (1995). Determination and involvement of aqueous reducing compounds in oxidative defense systems of various senescing leaves. *J. Agric.Food Chem.***43:** 1813.

Oyaizu M (1986). Studies on products of browning reaction: Antioxidative activities of products of browning reaction prepared from glucosamine. *Jpn. J. Nutr.* **44:** 307–315

Reuter S, Gupta SC, Chaturvedi MM, Aggarwal BB (2010). Oxidative stress, inflammation, and cancer: how are they linked? *Free Radic. Biol. Med.***49**(11)**:** 1603-1616.

Salah N, Miller NJ, Paganga G, Tijburg L, Bolwell GP, Rice-Evans C (1995). Polyphenolic flavanols as scavengers of aqueous phase radicals and as chain-breaking antioxidants. *Arch.Biochem. Biophys.***322**(2)**:** 339-346.

Savithramma N, M. LR, Ankanna S (2012). Preliminary Phytochemical Analysis of Traditionally used Medicinal Plants. *Res. J. Pharm. Bol. Chem. Sci.***3**(1)**:** 308-314.

Sawa T, Nakao M, Akaike T, Ono K, Maeda H (1999). Alkylperoxyl radical-scavenging activity of various flavonoids and other phenolic compounds: implications for the anti-tumor-promoter effect of vegetables. *J. Agric. Food Chem.***47**(2)**:** 397-402.

Shahidi F, Wanasundara PK (1992). Phenolic antioxidants. *Crit. Rev. Food Sci. Nutr.***32**(1)**:** 67-103.

Singleton V.L., Rossi JA (1965). Colorimetry of total phenolics with phosphomolybdic-phosphotungstic acid reagents. *Am. J. Enol. Vitic.***16:** 144-158.

Sofowora A (1993). Phytochemical screening of medicinal plants and traditional medicine in Africa 2nd Edition Spectrum Books Limited, Nigeria;. 150-156.

Tiwari P, Kumar B, Kaur M, Kaur G, Kaur H (2011). Phytochemical screening and extraction: A review. *Internationale Pharmaceutica Sciencia***1**(1)**:** 98-106.

Trease GE, Evans WC (1989). *Textbook of Pharmacognosy*. 12th ed. edn: Balliere; Tindall, London, UK

Tseng A (1991). *Proc. Am. Assoc. Cancer Res***32:** 2257.

Woode E, Amoh-Barimah AK, Abotsi WK, Ainooson GK, Owusu G (2012). Analgesic effects of stem bark extracts of Trichilia monadelpha (Thonn.) JJ De Wilde. *Indian J.Pharmacol.***44**(6)**:** 765-773.

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ORIGINAL ARTICLE

Visual Impairment and Ocular Findings among Deaf and Hearing Impaired School Children in Central Region, Ghana

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The scourge of global blindness continues to be a concern for eye care professionals, International Non-governmental Development Organizations (INGDOs) and eye care workers. While emphasis has been placed on how to address this scourge in the general population, not much is being done among special needs group such as the deaf and hearing impaired. The study was conducted to investigate the prevalence of visual impairment and ocular findings among hearing impaired children in a school for the deaf in the Cape Coast Municipality of Ghana. A cross-sectional descriptive study design was undertaken amongst children in the school for the deaf who had been previously diagnosed of hearing impairment or deafness. A total of 243 children underwent comprehensive eye examination in the school with prior approval from the school board. The mean age of the 243 children examined was 15.9 ± 4.0 years with a range of 9 – 27 years. Fourteen children (5.8%) had moderate visual impairment (WHO grade 1 visual impairment i.e. VA < 6/18 to 6/60) in the right eye, while 15 (6.2%) had moderate visual impairment in the left eye. Refractive error was present in 75 (31.9%) of the children with astigmatism being the commonest form of refractive error. Anterior segment abnormalities were present in 27 (11.1%) while posterior segment abnormalities were present in 25 (10.3%). The overall prevalence of visual impairment was 5.8% among hearing impaired school children in the Central Region of Ghana. There were ocular abnormalities that were previously undiagnosed among the studied population. There is the need for regular eye examination for children diagnosed of hearing impairment.

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Keywords*:* Disability, eye, ear, refractive error, ocular deviation .

INTRODUCTION

Studies have indicated that the prevalence of ocular abnormalities among the deaf and hearing impaired is higher than the general population of comparable age group (Nikolopoulos *et al.,* 2006). This association has been suggested to be due to the close anatomical relationship of the retina and cochlea which develops from the same embryonic layer (Abah *et al.,* 2011). Of all the sense organs, visual and auditory inputs are responsible for 95% of information acquisition (Fillman *et al.,* 1987). It is also generally claimed that visual input accounts for 75% of information acquisition. Existing co-morbidity of hearing and visual impairment in children predisposes them to many challenges including difficulties in communication, learning and social interaction. Suchman (1967) reported that hearing and visually impaired children are significantly more debilitated, less cooperative, less able to lip read and less capable of manual tasks compared to hearing impaired children with normal vision.

In Ghana, there is a high prevalence of hearing impairment. Recent studies have reported a prevalence of 16 per 1000 persons (Amedofu *et al.,* 1997; Amedofu *et al.,* 2005). Notwithstanding this however, there is a paucity of published data on the prevalence of visual impairment and visual abnormalities among the hearing impaired. The aim of this study was to investigate the prevalence of visual impair-

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ment and ocular abnormalities among hearing impaired children in a school for the deaf in Ghana.

MATERIALS AND METHODS

Study site and participants

A cross-sectional descriptive study was conducted to determine the prevalence of visual impairment and ocular abnormalities among deaf and hearing impaired children in a school for the deaf in the Cape Coast Municipality of Ghana from February to April, 2011. The school is the only school for the deaf in Central Region of Ghana. Before admission, each child is examined by an otolaryngologist to certify their status as either being deaf or hearing impaired. Thus, the basis for considering a study participant as being deaf or hearing impaired was based on the status established upon being admitted into the school. None of the children had had a previous eye examination. Every child present in the school during the eye examination visits was eligible to participate in the study. The school is residential with every child living within the premises.

Eye examinations

All eye examinations were carried out by qualified and registered optometrists. Every participant underwent a comprehensive eye examination comprising presenting visual acuity testing using the Snellen Tumbling E chart, near point of convergence test (NPC), cover test, external and internal ocular health examination using a penlight and direct ophthalmoscope respectively as well as both objective and subjective non-cycloplegic refraction. Communication during the examination process was facilitated by a designated teacher from the school who also had responsibility of reporting the response of each child during the examination.

Visual impairment classification

The World Health Organization category of visual impairment was used to specify the category of visual impairment among the children (WHO, 2005). Refractive error was specified as follows: myopia was defined as spherical power of $\geq$ - 0.50D, hyperopia $\geq$ + 2.00D and astigmatism $\geq$ - 0.50D. Emmetropia was defined as spherical correction of $<$ - 0.50D and $<$ + 2.00D and cylindrical correction of $<$ - 0.50D. Ocular deviation of $\geq 10^{\Delta}$ was considered as significant.

Ethical clearance

The protocol for the study was approved by the research committee of the Department of Optometry, University of Cape Coast. Administrative approval was also obtained from the Metropolitan Education Office as well as the head teacher of the school. Given the challenge of securing parental consent as per the ages of the participants, the head teacher further granted institutional clearance. Notwithstanding the institutional clearance, participants were required to give assent to be examined for the study or opt out if they so whish. The purpose of the eye examination was explained to every child and the study was conducted in accordance with the Declaration of Helsinki (WHO, 2007).

Statistical analysis

The data on eye examination was entered electronically using Microsoft Excel 2007. Data analysis was done using Statistical Package for Social Sciences (SPSS 17) (IBM Boston, USA). For qualitative variables, frequencies, percentage proportion and their 95% confidence intervals were computed. Quantitative variables were expressed as means $\pm$ standard deviation.

RESULTS

Study Participants

There were a total of 243 children in the school at the time of the study comprising 141 (58%) males and 102 (48%) females who were enrolled into the study. The mean age of the participants was 15.9$\pm$4.0, (95% CI = 15.4 – 16.4) years with age range of 9 – 27 years. The mean age of the male participants was 16.3$\pm$4.3 years while the mean age of the females was 15.3$\pm$3.6 years. There was no significant difference between the mean ages of male and female participants ($p = 0.068$). One hundred and eighty-eight (77.4%) of the subjects were aged between 11 – 20 years (Table 1).

Table 1: Age and sex distribution of study participants

Parameters	Gender		Total
	Male (n = 141)	Female (n = 102)	(n = 243)
Age (yrs)	16.3 ± 4.3	15.3 ± 3.6	15.9 ± 4.0
Age grp			
6 – 10	15 (10.6)	10 (9.8)	25 (10.3)
11 – 15	48 (34.0)	42 (41.2)	90 (37.0)
16 – 20	56 (39.7)	42 (41.1)	98 (40.3)
21 – 25	20 (14.2)	7 (6.9)	27 (11.1)
26 – 30	2 (1.4)	1 (1.0)	3 (1.2)

Data are presented as mean ± SD, absolute counts and proportions.

Table 2: Visual acuity distribution among study participants (n = 243)

Visual acuity	Right Eye (%)	Left Eye (%)
≥ 6/6	191 (78.6)	189 (77.8)
6/9	19 (7.8)	20 (8.2)
6/12	6 (2.5)	6 (2.5)
6/18	3 (1.2)	4 (1.6)
6/24	3 (1.2)	2 (0.8)
6/36	5 (2.0)	10 (4.1)
6/60	6 (2.5)	3 (1.2)
HM & LP	1 (0.4)	1 (0.4)
Undetermined	9 (3.7)	8 (3.3)

HM = hand movement, LP = light perception

Visual Acuity for distance

The distribution of visual acuity presentations are is shown in Table 2. One hundred and ninety-one (78.6%) had normal vision (VA≥6/6) in the right eye and 189 (77.8%) had normal vision in the left eye. Another 28 (11.5%) and 30 (12.3%) had mild visual impairment in the right and left eye respectively.

Fifteen (4.9%) participants had severe visual impairment in the right eye while 16 (6.6%) had severe visual impairment in the left eye (Table 3). Thus using WHO classification, 219 (90.1%, 95% CI = 85.9 – 93.4) had WHO grade 0 visual impairment (i.e VA ≤ 6/18). Another 14 (5.8%; 95% CI = 3.3 – 9.3) and 15 (6.2%; 95% CI =3.6 – 9.8) had moderate visual impairment (WHO grade 1 visual impairment i.e. VA < 6/18 to 6/60) (WHO, 2005) in the right and left eye respectively. One participant had vision of 'hand movement' and another one had 'light perception' in one eye. The vision in the other eye was 6/5 and 6/4 respectively. This could not be categorized as visually impaired using the WHO classification which considers vision in the worse eye. Visual acuity could not be determined in right eyes of 9 (3.7%) and left eyes of 8 (3.3%) participants. This was usually due to the fact that the children could not respond to the examination routine.

Refractive Error

Refraction results were unreliable in 8 (3.3%) of the subjects and therefore were not included in the analysis. The results from the right eye were used to identify the refractive status of each participant as described by Dandona, *et al*, (2002). One hundred and sixty (68.1%) participants had emmetropia while 75 (31.9%) had various forms of refractive error which comprised of astigmatism, present in 57 (76.0%) participants, myopia, 13 (17.3%) and

Table 3: Distribution of visual impairment categories among study participants (n = 243)

Type of impairment	Right eye (%)	Left eye (%)
Normal (VA≥6/6)	191 (78.6)	189 (77.8)
Mild visual impairment (VA < 6/6 to ≥6/18)	28 (11.5)	30 (12.3)
Severe visual impairment (VA <6/18 to light perception)	15 (4.9)	16 (6.6)
Visual acuity could not be determined	9 (3.7)	8 (3.3)

hyperopia 5 (6.7%) (Figure 1). Thus the prevalence of astigmatism, myopia and hyperopia among the participants was 24.3%, 5.5% and 2.1% respectively. There were 47 (62.7%) males and 28 (37.3%) females with refractive error. Whether a participant had a refractive error was independent of the sex of the participants ($\chi^2 = 1.59$, $p = 0.207$).

Binocular vision

The cover test performed at 40 cm revealed that 50 (20.6%) participants had various forms of ocular deviations including exophoria, exotropia, esophoria

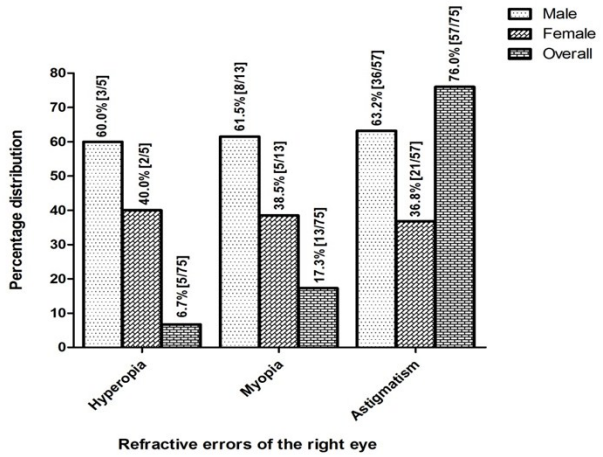

Figure 1: Distribution of refractive errors of the right eye among the participants

and esotropia while 184 (75.7%) had no deviation. Cover test could not be performed in 9 (3.7%) of the participants mainly because they were unable to maintain fixation at the fixation target. The results of the cover test are presented in Figure 2. Exophoria was the commonest ocular deviation being present in 39 participants out of the 50 who had ocular deviation (78%). Respectively, esophoria and exotropia were present in 8% (4/50) of the participants while esotropia was present in 6% (3/50) of the study participants.

Near point of convergence (NPC) could not be determined in 29 participants because they could not maintain fixation and respond to the test procedure. The mean NPC (break/recovery) was 5.6/7.4 ± 3.1/3.5 cm) with range of 1/1 to 25/28. Out of the 214 in which NPC was determined, 26 (12.1%) participants had values ≥ 8 cm for break while 21 (9.8%) had recovery values ≥ 11 cm. This proportion could estimate the proportion of participants who have convergence insufficiency.

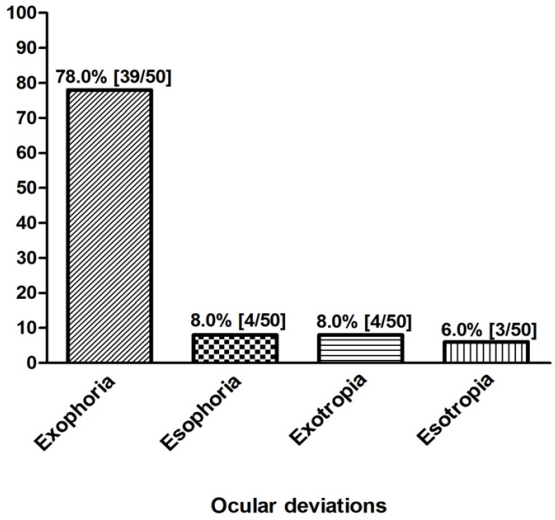

Figure 2: Distribution of ocular deviation among the participants

Ocular health

Ocular health was assessed in all 243 participants with some participants having a co-existence of anterior and posterior segment abnormalities. Anterior segment anomalies were found in 27 (11.1%) of the participants while posterior segment anomalies were found in 25 (10.3%). Table 4 shows the anterior and posterior segment anomalies found in the study.

DISCUSSION

The present study was conducted as an initial exploratory investigation of the ocular findings among the deaf and hearing impaired children in the Cape Coast Municipality of Ghana. Previous studies have reported the frequency of visual impairment and ocular abnormalities among hearing impaired subjects (Regenbogen, 1985; Fillman *et al.,* 1987; Nicoll and House, 1988; Armitage *et al.,* 1995; Mafong *et al.,* 2002; Hanioðlu-Kargý *et al.,* 2003; Nikolopulous *et al.,* 2006; Gogate *et al.,* 2009; Osaiyuwu and Ebeigbe, 2009; Bist *et al.,* 2011). Intepretative analy-

Table 4: Anterior and posterior segment anomalies among participants (N = 243)

Parameters	n (%)
Anterior Segment Abnormality	
Corneal abnormalities	5 (2.1)
Conjunctivitis	6 (2.5)
Lid	7 (2.9)
Nystagmus	1 (0.4)
Dacryocystitis	1 (0.4)
Heterochromia iridis	1 (0.4)
Posterior Segment Abnormality	
Lens	2 (0.8)
Choroid/retinal	13 (5.3)
Retinitis pigmentosa	3 (1.2)
Vitreous	2 (0.8)
Optic nerve/disc	4 (1.6)
Phthisis bulbi	1 (0.4)

sis from results of this study should therefore be done cautiously as a result of the considerable challenge presented in comparative analysis to studies conducted elsewhere due largely to variations in the criteria for reporting findings with typical examples being the cut-off points for visual acuity and the variations in ocular conditions presented.

In Nigeria, the frequency of visual disorders among the hearing impaired has been reported to range between 20.9 – 73.3% (Osaiyuwu and Ebeigbe, 2009; Abah *et al.,* 2011). Some of these studies have also indicated that the frequency of ocular abnormalities are higher among hearing impaired subjects compared to normal hearing subjects in the general population with similar age groups (Gogate *et al.,* 2009; Abah *et al.,* 2011). Recommendations for regular screening for the presence of visual disorders among the hearing impaired have been advanced by several authors so that appropriate remedial measures are taken to address the challenges faced by this cohort of subjects.

Some studies have reported on ocular abnormality findings among hearing impaired children without reporting the level of visual functioning at least as measured by visual acuity (Mafong *et al.,* 2002; Hanioðlu-Kargý *et al.,* 2003; Gogate *et al.,* 2009; Osaiyuwu and Ebeigbe, 2009; Abah *et al.,* 2011). From this study, 10.3% of the children for whom visual acuity was measured had a VA <6/9 with a further 7.3% having category I and II visual impairment. This is more than the 6.4% found by Nicoll and House (1988) in a study conducted in Western Australia but lower than 25.3% reported by Armitage *et al.,* (1995) in the UK. Armitage *et al.,* (1995) in their study had reiterated the fact that a deaf child is dependent on vision for communication and learning. There is no doubt therefore from this study that the estimated visual impairment prevalence rate of 7.3% may lead to this cohort of study participants having certain challenges in acquisition of communication skills.

Refractive error was present in 30.9% of the children for whom refractive error was determined. This is comparable to the estimated 28.9% and 29.8% reported by Armitage *et al.,* (1995) and Hanioðlu-Kargý *et al.,* (2003) in their respective studies. It was much higher compared to values reported by other authors: 7.9% (Abah *et al.,* 2011); 18.5% (Gogate *et al.,* 2009); 16.5% (Bist *et al.,* 2011) and much lower compared to the 73.3% reported by Osaiyuwu and Ebeigbe (2009). In terms of the commonest form of refractive errors, astigmatism was the most prevalent refractive error in this study. While this is consistent with the report of Hanioðlu -Kargý *et al.,* (2003), it was different from other reports. For example, hyperopia was the commonest refractive error reported by Abah *et al.,* (2011) and Mafong *et al.,* (2002) whereas myopia was the commonest refractive error reported by Gogate *et al.,* (2009), Bist *et al.,* (2011) as well as Osaiyuwu and Ebeigbe (2009). Nicoll and House (1988) reported an equal prevalence for myopia and hyperopia. The reason for this variation could be attributed to the differences in the definition and cut off point used by different authors in specifying the types of refractive errors.

A comparison of the prevalence of visual impairment and refractive error in the present study to two earlier studies on refractive error among school children without hearing impairment in Centreal

Region of Ghana by Ovenseri-Ogbomo and Omuemu (2010) and Ovenseri-Ogbomo and Assien (2010) shows that the 10.3% proportion of children with VA< 6/9 in this present study was significantly more than the 2.1% and 4.5% respective prevalence rates reported for school children without hearing impairment. The proportion of refractive error in this study 31.9% was also significantly more than the 25.6% and 13.3% reported by Ovenseri-Ogbomo and Omuemu (2010) and Ovenseri-Ogbomo and Assien (2010) respectively for school children without hearing impairment. This difference could even be more marked when one considers the fact that in the present study, cycloplegic refraction was not incorporated among the list of ocular problems as was the case in earlier studies conducted by Ovenseri-Ogbomo and Omuemu's (2010) or the use of higer cut-off point of +2.00D for hyperopia compared to +0.75D used by Ovenseri-Ogbomo and Assien (2010). Given that these studies were from children without hearing impairment within the same geographical and socio-cultural background, it might be inferred that children with hearing impairment have significantly higher proportion of visual impairment and refractive error compared to those without hearing impairment which finding is consistent with that of Brinks *et al.,* (2001) and Rogers *et al.,* (1988).

The ocular deviation proportion of 20.6% estimated in this study is greater than the 16.5% reported for Ghanaian school children without hearing impairment (Ovenseri-Ogbomo and Assien, 2010). In earlier studies conducted by Ovenseri-Ogbomo and Assien (2010) in children without hearing impairment and this study, exophoria happenened to be the commonest observed ocular deviation. Furthermore, the esophoria proportion of 8.0% estimated from this study is greater than the 2.0% estimated from the study conducted in children without hearing impairment by Ovenseri-Ogbomo and Assien (2010). This study recorded a squint prevalence of 2.5% in the children and this rate is higher than the 1.3% reported by Gogate *et al.,* (2009) for hearing impaired children in India.

The study noted the presence of previously undetected ocular abnormalities among the participants and this underscores the need for regular and more periodic scheduled eye examination for these groups of people as related by Gogate *et al.,*(2009) in their study. The occurrence of ocular abnormalities estimated in this study might have been underreported considering the fact that eye examinations in the school were conducted with direct ophthalmoscope which might have limitations in the diagnosis of some subtle ocular presentation that could have been uncovered with more sophisticated diagnostic procedure.

CONCLUSION

There are high proportions of refractive errors and other ocular abnormalities among the hearing impaired and deaf population of school going age. Given the high proportion of refractive error in this group, it is highly recommended that alongside otolaryngologic examination, children diagnosed of hearing impairment should have scheduled eye examination before placement in the school and during the duration of the schooling period. The relevant authorities should also ensure periodic reexamination of these children to detect any new ocular abnormality and evaluate on-going pathology. This high proportion of retinal/choroidal abnormalities could warrant a detailed retinal evaluation by experienced eye care practitioners. The detection of ocular abnormalities and taking prompt action will have a significant impact in the social interaction of the hearing impaired child.

COMPETING INTERESTS

The authors declare that they have no competing interests.

REFERENCES

Abah ER, Oladigbolu KK, Samaila E, Ahmed AO, Abubakar TH. (2011). Ophthalmologic abnormalities among deaf students in Kaduna, Northern Nigeria. *Ann Afr Med.* 10:29 - 33.

Amedofu Gk, Brobby GW, Baubent JO, Ocansey G, Asante P, Asibey O. (1997). Congenital non-syndromal deafness in Adamarobe, an isolated Ghanaian village: prevalence, incidence and audiometric characteristics of

deafness in the village. *Hereditary Deafness Newsletter.* 13:12 - 19.

Amedofu KG, Ocansey G, Antwi B. (2005). Characteristics of hearing impairment among patients in Ghana. *Afr J Health Sci.*12:87 – 93.

Armitage IM, Burke JP, Buffin JT. (1995). Visual impairment in severe and profound sensorineural deafness. *Arch Dis Child.* 73:53 - 53.

Bist J, Adhikari P,Sharma AK. (2011). Ocular morbidity in hearing impaired schoolchildren. *Child: Care, Health Dev.* 37(3):394 - 397.

Brinks M, Murphey W, Cardwell W, Otos M, Weleber R. (2001). Ophthalmologic screenings of deaf students in Oregon. *J Pediatr Ophthalmol Strab.* 38(1):11 - 15.

Dandona R, Dandona L, Srinivas M et al. (2002). Refractive error in children in a rural population in India. *Invest Ophthalmol Vis Sci.* 43 (3):615 - 622.

Fillman RD, Leguire LE, Rogers GL, Bremer DL Fellows RR. (1987). Screening for vision problems, including Usher's syndrom, among hearing impired students. *Am Ann Deaf.* 132:194 - 198

Gogate P, Rishikeshi N, Mehata R, Renade S, Kharat J, Desphande M. (2009). Visual impairment in the hearing impaired students. *Indian J Ophthalmol.* 57:451 - 453.

Hanioðlu-Kargý Þ, Köksal M, Tomaç S, Uðurbaþ SH, Alpay A. (2003). Ophthalmologic abnormalities in children from a Turkish school for the deaf. *Turk J Pediatr.* 45:39 - 42.

Mafong DD, Pletcher SD, Hoyt C, Lalwani AK. (2002). Ocular findings in children with congenital sensorineural hearing loss. *Arch Otolaryngol Head Neck Surg.* 128:1303 – 1306

Nikolopoulos TP, Lioumi D, Stamataki S, O'Donoghue GM, Guest M, Hall A-M. (2006). Evidence-based overview of ophthalmic disorders in deaf children: A literature update. *Otol Neurotol.* 27:S1 - S24.

Nicoll AM, House P. (1988). Ocular abnormalities in deaf children: A discussion of deafness and retinal pigment changes. *Austr N Z J Ophthalmol.* 16:205 208.

Osaiyuwu AB, Ebeigbe JA. (2009). Prevalence of visual impairement in deaf children in Benin City. *J Nig Opt Assoc.* 15:20 - 23.

Ovenseri-Ogbomo GO, Assien R. (2010). Refractive error in school children in Agona Swedru, Ghana. *S Afr Optom.* 69(2):86 - 92.

Ovenseri-Ogbomo GO, Omuemu VO. (2010). Prevalence of refractive error among school children in the Cape Coast Municipality, Ghana. *Clin Optom.* 2:59 – 66.

Regenbogen L, Godel V. (1985). Ocular deficiencies in deaf children. *J Peadiatr Ophthalmol Strab.* 41:231 - 233.

Rogers GL, Fillman RD, Bremer DL, Leguire LE. (1988). Screening of school-age hearing-impaired children. *J Pediatr Ophthalmol Strab.* 25(5):230 - 232.

Suchman RG. (1967). Visual impairment among deaf children. *Arch Ophthalmol.* 77:18 - 21.

WHO. (2005). International statistical Classification of Diseases and Related Health Problems. 2nd Edition (10th Revision). Geneva. World Health Organization.

WHO. (2007). World Medical Association Declaration of Helsinki: Ethical principles for medical research involving human subjects. *Bull World Health Org.* 79(4):373 – 374.

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