Nigerian Journal of Ophthalmology, Vol. 6, No. 1, August 1998: 23–25.

Screening for Retinopathy of Prematurity in Ibadan

***A.M. BAIYEROJU-AGBEJA, S. I. OMOKHODION**

From University College Hospital, Ibadan

SUMMARY

Between January to June 1995, a prospective study was carried out to determine the incidence and severity of retinopathy of prematurity (ROP) in newborn infants of less than 1,500g birthweight or less than 31 weeks gestational age. 18 babies were eligible for inclusion in this study and only one developed ROP, a Stage 1, Zone III disease i.e. 5.5% of babies at risk. In spite of the low incidence compared with other studies[1], it is advisable to continue the collaboration between opthalmologists and neonatologists to prevent babies at risk from developing untreatable ROP, thereby increasing the number of blind children in our society.

KEY WORDS: *Retinopathy of prematurity, gestational age, infant, premature, hyperoxia.*

INTRODUCTION

Retinopathy of prematurity (ROP) is attracting renewed interest from opthalmologists and neonatologists because of the improved survival of very low birthweight premature infants, some of whom develop blinding disease[1].

By the mid 1950's it had been generally agreed by Patz[2], Kinsey[3], Ashton[4], and Guy[5] that this disease entity previously called "Retrolental fibroplasia" was a totally preventable disease, especially when the liberal use of oxygen was restricted. This was followed by a decrease in the number of ROP cases[6] but an associated increase in perinatal mortality rate was noted[7].

In the mid 1970's, an increase in cases of ROP was noted[8]. By the time pathological retinal changes are evident to the neonatologist performing external examination of the eye and direct ophthalmoscopy, the disease is usually very advanced[1]. This led to the recruitment of ophthalmologists who were experienced in the technique of indirect ophthalmoscopy with scleral depression to be able to detect the disease at an early stage.

The best way of detecting ROP at the earliest possible stage is to actively screen all neonates at risk. No study has ever been published about ROP in Nigeria and this was what prompted this study from the Special Care Baby Unit of this tertiary institution. The study was designed to determine the incidence and severity of ROP in all infants considered to be at risk i.e. infants born at less than 31 weeks gestation or less than 1,500g birthweight[1,8], and to investigate the association between a range of clinical features and the development of ROP. This was a pilot study to pave the way for a more extensive study.

MATERIALS AND METHODS

Over a 6 month period (January– June) 1995), all babies born in the University College Hospital, Ibadan, who fell into the 2 categories of less than 31 weeks gestation or less than 1,500g birthweight were recruited in the study. Since University College Hospital houses the only Special Baby Care Unit with incubators in Oyo State, other babies born in the state and referred for admission into the unit, who fell into the two categories were also recruited. A protocol was designed for each patient, to obtain gestational age, birthweight, sex, duration of oxygen administration, intercurrent illness such as severe infections and a full ophthalmological examination. The ophthalmic examination was first performed soon after birth, followed by a weekly examination until discharge, if the baby was stable clinically. Both pupils were dilated with Gutt. Cyclopentolate 0.5% and Gutt. Phenylephrine 0.25% 30 and 60 minutes prior to examination.

Examinations were performed by a single

*Correspondence

ophthalmologist using a binocular indirect ophthalmoscope, 20 diopter hand lens and a neonatal eye speculum. Scleral indentation was only used in selected cases.

Retinal changes were recorded in accordance with the International Classification Scheme[9,10] i.e. Stage I: the presence of a demarcation line, Stage II: ridge formation; Stage III: the presence of a ridge with extraretinal fibrovascular proliferation; Stage IV: subtotal retinal detachment; Stage V: total retinal detachment. The disease location on the retina, zones I–III and extent in clock hours were also noted. Zone 1 being the innermost zone with its centre at the optic disc. Retinal changes before the development of the demarcation line were documented but not classified as ROP. Vitreous haze was also noted.

RESULTS

Within the 6 months period, January – June 1995, 18 babies were found to be at risk of developing ROP, out of a total of 102 babies admitted to the special care baby unit i.e. 17.6%. All 18 babies were either less than 1,500g birthweight or less than 31 weeks gestation.

There were 10 males and 8 females, a male: female ratio of 1: 1.25. The birthweights ranged from 870 - 1,500g and gestational age ranged from 26–31 weeks, 3 babies under 1,000g weight and 7 under 28 weeks gestational age. The 3 babies under 1000g weight were all under 28 weeks gestation. Duration of oxygen was found to be between 4–51 days, although almost all the babies who had less than 14 days oxygen died within 14 days after birth while on oxygen.

Out of the 18 babies, 12 died, 11 of whom died within 18 days after birth and only one died at 41 days after birth (after recurrent apnoeic attacks). Only one case of ROP was found in the babies i.e. 5.5%, a baby of 30 weeks gestational age. This was found at 7 days after birth and the baby died the next day from suspected septicaemia. The stage of ROP was Stage 1, Zone III, 4 clock hours in both eyes. Other vitreous and retinal changes were noted in some of the other babies. There was vitreous haze in 10 babies, 3 of which were so bad, no further view of the retina could be obtained in spite of the brightest illumination of the binocular indirect ophthalmoscope. 9 babies had thin retinal vessels, 8 of which had no peripheral vascularisation. On subsequent examinations, all these features disappeared with good peripheral vasculirisation appearing, no ROP developed in these eyes. Generalised septicaemia was confirmed in 6 babies while 10 babies were suspected of having septicaemia, 8 of whom died before blood cultures could confirm septicaemia.

DISCUSSION

The incidence of retinopathy of prematurity (ROP) is estimated to range from 3–5%[11,12] to 35%[13].

Only one study has been published in Africa which revealed an incidence of 10.6% in South Africa of children blind from ROP[14]. This is the only country in Africa where blindness from ROP has been reported.

In the present study, the incidence of the disease in infants at risk was found to be 5.5%, the lower end of the estimated range. The disparity in reported incidence from different units reflects a difference in the mortality rate as well as the survival rates in these units. The survival rate of 85% of all infants less than 1,500g birthweight was found in the study by Prendiville in England[8], comparing favourably with other European studies of 69%[11] unlike our study where the survival rate was found to be much lower, 33.3%. It is worth mentioning here that the major factor affecting the survival of low birthweight infants in our unit is lack of adequate facilities.

Only 3 out of 6 incubators are presently functioning and recurrent episodes of lack of oxygen for use in the special care baby unit is a major problem. While the study was underway in 1995, a severe shortage of oxygen occurred for 3 days during which 4 at-risk babies died.

The only baby who developed ROP, had a stage I, Zone III disease by 7 days after birth could not be re-examined for progression or regression as she died the day after.

Vitreous haze was found in 10 babies. This is normal in very immature babies before the media clears sufficiently to allow a good view of the periphery[1]. Another normal developmental feature found in 8 of our babies is the absence of peripheral vasculirization which later became vascularised as the babies matured[15].

Septicaemia is a well recognised association of ROP[8,16] which was confirmed in 6 infants and suspected in 10 others, a total of 16 infants (88.9% of infants at risk). 8 of the 10 with suspected septicaemia died while 3 of the confirmed group died. Septicaemia, even though a risk factor, its finding alone does not lead to retinopathy of prematurity in a baby at risk, it does so in association with hyperoxia, ges-

tational age and acidosis[8].

In conclusion, although a low incidence of ROP was found in the present pilot study, we advocate a larger multicentric study for an extended period of time, probably 5 years, to study the real incidence and severity of the disease in our babies at risk and to investigate the associations between a range of clinical features and the development of ROP.

A study of the incidence of ROP in our blind schools will also give a good idea of the morbidity caused by the disease in the preterm, low birthweight babies who survived.

REFERENCES

1. Standard KP, Mushin AS, Gamsu HR(1989) Screening for Retinopathy of prematurity in a regional neonatal intensive care unit. Eye **3:** 371–378.

2. Patz A, Hoeck LE, de la Cruze E (1952) Studies of the effect of high oxygen administration in retinolental fibroplasia I. Nursery observations. *Am J Ophthalmol* **35:** 1248–1253.

3. Kinsey VE. (1956) Retrolental fibroplasia. Cooperative study of retrolental fibroplasia and the use of oxygen. *Arch Ophthalmol* **56:** 481–543.

4. Ashton N, Ward B, Serpell G.(1954) Effect of oxygen on developing retinal vessels with particular reference to the problem of retrolental fibroplasia. *Br J Ophthalmol* **38:** 397–432.

5. Guy LP, Lanman JT, Dancis J (1956) The possibility of total elimination of retrolental fibropolasia by oxygen restriction. Paediatrics **17:** 247–249.

6. McDonald AD. (1962) Neurological and Ophthalmic disorders in children of very low birthweight. *Br Med J* **1:** 895–900.

7. Cross KW. (1973) The cost of preventing retrolental fibroplasia. *Lancet* **2:** 954–956.

8. Prendiville A, Schulenburg WE. (1988) Clinical factors associated with retinopathy of prematurity. *Arch Dis Childhood* **63:** 522–527.

9. Committee for the classification of ROP. (1984) An international classification of ROP. Arch Ophthalmol **102:** 1130–1134.

10. Committee for the classification of ROP. (1984) An international classification of ROP. *Paediatrics* **74:**127–133.

11. Campbell PB, Bull MJ, Ellis FD, Bryson CQ, Lemons JA, Schreiner RL. (1983) Incidence of the retinopathy of prematurity in a tertiary newborn intensive care unit. *Arch Ophthalmol* **10:** 1868–1688.

12. Yu VYH, Hookham DM, Nave JRM. (1982) Retrolental fibroplasia controlled study of 4 years experience in a neonatal intensive care unit. Arch Dis Child **57:** 247–252.

13. Glass P, Avery GB, Subramanian S. (1986) Effect of bright light in the hospital nursery on the incidence of retinopathy of prematurity *N Eng J Med* **313:** 401–404.

14. O'Sullivan J, Gilbert C, Foster A. The causes of childhood blindness in South Africa. *S Afr Med J.* In press.

15. Kumar H, Singha U. (1997) Retinopathy of prematurity: Clinical aspects. Comm Eye Health **10**(22): 19–22.

16. Gunn TR, Easdown J, Outerbridge EW, Arandja JV. (1980) Risk factors in retrolental fibroplasia. *Paediatrics* **65:** 1096–1100.