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RETINOPATHY OF PREMATURITY: AN OVERVIEW

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Abstract

Retinopathy of prematurity (ROP) is a disorder characterized by rapid growth of retinal blood vessels in the premature infant. Fetus, retinal vascularization begins at 4 months and progresses until completion at 9 months or shortly after birth. The premature infant is born with incomplete retinal vascularization, yet new vessels continue to grow between the vascularized and non -vascularized retina.

Retinopathy of prematurity (ROP) is a disorder of premature low birth weight infants featuring abnormal proliferation of the developing blood vessels at the junction of vascular and the avascular retina. Chances are 16-17% for all premature infants 68% ROP in birth weight < 1250g, 98% among those having birth weight < 750g. Risk factors associated will be Low birthweight, Early gestational age, Sepsis, Hypothermia, Hypoxia, Oxyhemoglobin dissociation curve changes that occur when adult blood is transfused to the premature infant, Duration/concentration of supplemental oxygen.

Keywords:

Retinopathy of Prematurity, Disorder, Premature, low birth weight, Retina, Sepsis, Hypothermia, Hypoxia, Oxyhemoglobin

Causes

- Acute and chronic effects of oxygen toxicity on the developing blood vessels of the premature infant's retina.
- Progressive vascular growth of retina
- Eventual blindness if not treated

Classification

Stage I demarcation line:

- Mildly abnormal blood vessel growth.
- Many children who develop stage I improve with no treatment and eventually develop normal vision.
- The disease resolves on its own without further progression**.**

Stage II ridge:

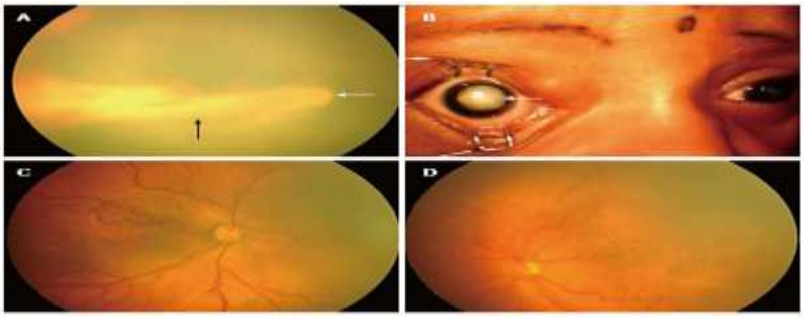
- Moderately abnormal blood vessel growth.
- Many children who develop stage II improve with no treatment and eventually develop normal vision.
- The disease resolves on its own without further progression**.**

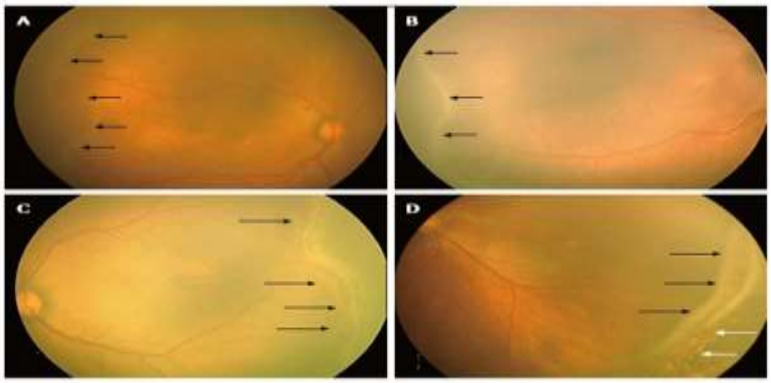
Stage III ridge with extra retinal fibro vascular proliferation:

- Severely abnormal blood vessel growth.
- The abnormal blood vessels grow toward the centre of the eye instead of following their normal growth pattern along the surface of the retina.
- Some infants who develop stage III improve with no treatment and eventually develop normal vision.
- However, when infants have a certain degree of Stage III and "plus disease" develops, treatment is considered.
- **Plus, disease** means that the blood vessels of the retina have become enlarged and twisted, indicating a worsening of the disease. Treatment at this point has a good chance of preventing retinal detachment**.**
- **Pre-plus disease:** It is defined as posterior pole vascular dilation and tortuosity which is more than normal but less than plus disease

Stage IV — **Partially detached retina**. Traction from the scar produced by bleeding, abnormal vessels pulls the retina away from the wall of the eye.

Stage V — **Completely detached retina** and the end stage of the disease. If the eye is left alone at this stage, the baby can have severe visual impairment and even blindness.





The first blood supply to the inner retina appears in the form of "spindle cells "from the adventitia of the hyaloids artery at about 16 weeks of gestation. Spindle cells canalize and metamorphose into mature vessels. and reach the nasal ora serrata by around 36 weeks and temporal by around 39-41weeks.

Two theories are described:

1) **The classical theory- proposed by arthon and patz-**

• according to this theory supplemental oxygen was considered main causative factor.

• Elevated po2 if sustained leads to permanent vasoconstriction.

• Neonate is subjected to room temperature, endothelial proliferation from nearby capillaries takes place leading to neovascularization which may reach to vitreous producing fibrosis causing vitreous traction and finally retinal detachment.

2) **Spindle cell theory**

This theory which was proposed by Kretzer et al

• postulates the induction of retinal and vitriol neovascularization by spindle cell insult • After birth the spindle cells are exposed to hypertoxic environment because of increased oxygen diffusion through this retina from choroidal vasculature which leads to formation of gap junctions.

• Oxygen free radical, a cytotoxic agent attacks compromised spindle cells, which has efficient anti-oxidative defence mechanism.

• This abnormal spindle cells stop migration and canalization. The damaged spindle cells secrete angiogenic factors that trigger neovascular response.

Features are graded in stages of severity depending on the retinal signs and the zone of retina involved.

Zone 1 is a small area at the heart of the retina (surrounding the central visual area, including the optic nerve)

Zone 2 covers the middle of the retina; and

Zone 3 runs along the retina's outer edge.

The lower the zone number, the more serious the ROP.

Children at risk should be screened periodically. The procedure is performed at NICU by pediatric ophthalmologist, under the supervision of neonatologist so that complication can be handled. The pupil is dilated with a mixture of phenyl purine 2.5% and tropicamide 0.4 % or cyclopentolate 0.5% instilled 3 times at 10min intervals before the scheduled examination. Topical anesthetic and lid speculum should be used to reduce discomfort. Indirect ophthalmoscopy is performed with 20D / 30D lens using fresh sterile instruments. Scleral depression is done to stabilize the eye, rotate it, indent it. Follow-up examinations should be recommended by the examining ophthalmologist based on retinal findings classified according to the international classification.

a) 1-Week or Less Follow-up: Immature vascularization: zone I—no ROP, immature retina extends into posterior zone II, near the boundary of zone I, stage 1 or 2 ROP in zone I, stage 3 ROP in zone II, the presence or suspected presence of aggressive posterior ROP.
b) 1- to 2-Week Follow-up: Immature vascularization; posterior zone II, stage 2 ROP in zone II, unequivocally regressing ROP in zone I.
c) 2-Week Follow-up: Stage 1 ROP in zone II, immature vascularization: zone II—no ROP, unequivocally regressing ROP in zone II.
d) 2- to 3-Week Follow-up: Stage 1 or 2 ROP in zone III, regressing ROP in zone III.

Prevent preterm birth. Provide early screening and detection in infants born at <30 weeks of gestation and weight <1500 g (3.3 pounds). Decrease exposure to bright, direct lighting; although exposure to bright light has not been proven to contribute to retinopathy of prematurity, such exposure is undesirable from neurobehavioral developmental perspective. Use supplemental oxygen judiciously and monitor oxygen blood levels carefully; prevent wide fluctuations in oxygen blood levels (hyperoxemia and hypoxemia). The administration of an anti-vascular endothelial growth factor drug bevacizumab. Which arrests the proliferation of vessels and prevents retinal detachment commonly seen in retinopathy of prematurity. Laser surgery: This is done most often for ROP. Small laser beams scar the peripheral retina. This procedure (also called laser therapy or photocoagulation) lasts about 30–45 minutes for each eye. In cry therapy, physicians use an instrument that generates freezing temperatures to briefly touch spots on the surface of the eye that overlie the periphery of the retina. Both laser treatment and cryotherapy destroy the peripheral areas of the retina, slowing or reversing the abnormal growth of blood vessels. Both are performed only on infants with advanced ROP.

Scleral buckle

- This involves placing a silicone band around the eye and tightening it.
- This keeps the vitreous gel from pulling on the scar tissue and allows the retina to flatten back down onto the wall of the eye.
- Sclera buckles are usually performed on infants with stage IV or V.

Vitrectomy

- Vitrectomy involves removing the vitreous and replacing it with a saline solution.
- After the vitreous has been removed, the scar tissue on the retina can be peeled back or cut away, allowing the retina to relax and lay back down against the eye wall.

Conclusion

Retinopathy of prematurity is an important cause of ocular morbidity and blindness in children. With better understanding of the pathogenesis, screening and treatment guidelines have changed over time and are unit specific.

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