Retinopathy of Prematurity

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KEYWORDS

• Retinopathy of prematurity (ROP) • Retrolental fibroplasia
• Vascular endothelial growth factor inhibitors • Prematurity • Low birth weight

KEY POINTS

- Supplemental oxygen, birth weight, and gestational age are the major risk factors for the development of retinopathy of prematurity (ROP).
- Premature infants at or less than 1500 g or 30 weeks born in the United States should be screened for ROP.
- Current treatment of threshold or type I ROP is laser photocoagulation of the peripheral avascular retina.
- Vascular endothelial growth factor inhibitors such as bevacizumab are the newest treatment option, but more research into dosage, safety, and long-term outcomes must be performed.
- All former premature children are at risk for high refractive error, amblyopia, and strabismus.

INTRODUCTION

Retinopathy of prematurity (ROP) is a potentially blinding retinal vascular disease that occurs in very low birth weight (VLBW) (<1500 g) premature infants. Originally called retrolental fibroplasia (RLF), the disease was first described in 1942.[1] In 1950, RLF was responsible for 21.5% to 41.7% of all childhood blindness.[2] High supplemental oxygen and lower birth weight were discovered to be the major risk factors in RLF.[3] Nevertheless, even with monitoring oxygen saturations in the 1980s, 5% of infants with ROP became totally blind.[4] Screening for ROP, close monitoring of prethreshold disease, treatment of threshold disease with laser photoablation along with prevention of ROP and prematurity have become the foundations for preventing blindness.

EXTENT OF THE PROBLEM

According to the 2010 US census, the preterm birth rate (<37 weeks) was 11.99% of all births and the low birth weight rate (<2500 g) comprised 8.15% of all births.[5]

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VLBW (<1500 g) birth rate was 1.45% and was unchanged compared with the previous year census. In 2008, 24% of VLBW infants died within the first year of life.[5] From 1997 to 2005, 0.17% of total newborns born in the United States had some form of ROP.[6] It is the third leading cause of childhood blindness in the United States (14%).[7] In highly developed countries such as the United States, only the smallest, youngest babies are likely to have ROP, because of improved control of risk factors and more aggressive management of unstable infants. By comparison, in Latin America and Eastern Europe, there are higher rates of severe ROP and blindness, and the disease occurs in older and larger infants compared with the United States.[8] In poorly developed nations, preterm infants do not survive long enough to develop ROP.[8] In 2010, it was estimated that globally 184,700 premature infants would develop ROP, and of those, 20,000 would become blind or severely visually impaired.[9]

CAUSE/CONTRIBUTORY OR RISK FACTORS

Risk factors:

- Birth weight[10–15]
- Gestational age[10–15]
- Poor weight gain[11,16]
- Low cortisol concentration[17]
- Dopamine-resistant hypotension[17]
- White race[10,13,14,18]
- Birth at an outlying hospital[13]
- Low insulinlike growth factor binding protein 3,[19] low insulin growth factor,[16,20,21] and low urine vascular endothelial growth factor (VEGF)[22]
- Hyperglycemia[12,16]
- Insulin treatment[16]
- Corticosteroid treatment[12,16,23]
- Insufficient intake of docosahexaenoic acid[16]

Associated conditions:

- Respiratory conditions such as bronchopulmonary dysplasia[6,12,15]
- Fetal hemorrhage[6]
- Intraventricular hemorrhage[6,12,15]
- Blood transfusion[6]
- Sepsis[12,15]
- Respiratory tract colonization with *Ureaplasma urealyticum*[24]
- Patent ductus arteriosis[12,15]

Protective conditions:

- Hypoxia[6]
- Necrotizing enterocolitis[6]
- Hemolytic disease[6]
- Breast milk[25]
- Improved nutrition (lipids and total calories)[26]
- Maternal preeclampsia[27]

SEQUELAE

Infants with regressed or treated ROP along with premature infants who never developed ROP should be appropriately referred to a pediatric ophthalmologist at a young