Infective Cataract (TORCH)

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Certain viral and parasitic infections occurring during embryogenesis may predispose to the development of congenital cataract [1, 2]. TORCH group of infections like Toxoplasma, Rubella, Cytomegalovirus (CMV), Herpes Simplex virus (HSV), and Others including syphilis have been associated with congenital cataract formation especially in developing countries [3–5]. The pathogen enters the fetus through the placenta of the infected mother during pregnancy [6]. Following infection, the immune systems produces a series of antibodies in the fetus. Antibodies are transferred to the developing fetus through the placenta from the infected mother and remains for a specific period of time following birth [3]. TORCH infections can be detected by immunoglobulin (Ig) M and Ig G titers in serum [3-5, 7-9]. Mahalakshmi and coworkers have described the presence of IgM antibodies in congenital cataract with infective etiology [3]. They have reported CMV Ig M in 17.8% and rubella virus Ig M in 8.4% among 593 infants with congenital cataract. Various studies have reported Ig M for

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Professor Ophthalmology, Dr. RP Centre for Ophthalmic Sciences, All India Institute of Medical Sciences, New Delhi, India HSV and Toxoplasma with congenital cataract [4, 5, 7–9]. There can be an overlap in manifestation of these infections. TORCH group of pathogens may additionally affect the ectodermal tissues [10], from which the lens is also derived. Other ocular manifestations of TORCH infections in addition to childhood cataract include microphthalmos, keratitis, glaucoma, iris atrophy, iridocyclitis, optic neuritis, retinitis, and chorioretinitis [11].

14.1 Congenital Toxoplasmosis

Congenital Toxoplasmosis has wide-ranging clinical manifestations from being completely asymptomatic at birth to severe neurological and ocular disease. The majority of the infants have no apparent clinical manifestations at birth. These may be identified during routine new born examination and maternal screening [12]

Systemic features includes [12]

- Neurological features such as micro or macrocephaly, seizures, nystagmus, hydrocephalus, cerebral calcifications, meningoencephalitis
- Small for gestational age
- Hepatosplenomegaly
- Generalized lymphadenopathy
- Jaundice
- Thrombocytopenia, anemia, petechiae
- Maculopapular rash

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Ophthalmic features includes

- Chorioretinitis
- · Microphthalmos
- Retinochoroiditis
- Strabismus
- Iridocyclitis
- Cataract
- Glaucoma

Congenital toxoplasmosis is a reason for substantial visual loss and neurologic morbidity in children. Even if treatment is initiated at birth, it may not successfully prevent ocular damage [13-16]. The most common ocular manifestation of congenital toxoplasmosis is chorioretinitis (upto 92%), which is then followed by microphthalmos, squint, iridocyclitis, cataract and glaucoma [17–20]. Congential cataract is featured more commonly in congenital rubella, chorioretinitis is more commonly associated with congenital toxoplasmosis (Fig. 14.1). The pathogenesis of cataract in congenital toxoplasmosis remains unknown. Generally, the retina and choroid are affected first. It is then followed by involvement of iris leading to iridocyclitis and cataract, which can develop as secondary complication of retinochoroiditis. The incidence of cataract was 11.6% in the National Collaborative Chicago-based Congenital Toxoplasmosis Study (NCCCTS), typically occurring in those with the more extreme disease [15]. These infections can affect any part of the lens and cataract can vary in severity from partial to total. Most frequently noted cataract variety is posterior subcapsular; but, nuclear, anterior subcapsular, and anterior polar cataracts have also been reported [16].

Test for diagnosis of toxoplasmosis [12]

- Serological test
- Presence of Toxoplasma specific Ig M and/or Ig A antibodies 10 days after birth.
- Persistent or increasing IgG titer without treatment in infants at or beyond 1 year of age
- Positive PCR for T. gondii DNA or positive Toxoplasma IgM or Ig A antibodies in the CSF
- Positive IgG is indicative of prior or current maternal infection. Repeat IgG testing is done at every 4–6 weeks until complete disappearance in case of clinical suspicion of toxoplasmosis with negative IgM and IgA antibodies.



Fig. 14.1 (a, b) Clinical picture of right and left eye fundus showing typical punched out macular scar suggestive of healed congenital toxoplasma infection

- Negative IgM and IgA antibodies do not exclude the infection. There's a delay within the production of antibodies in the newborn, if the mother is affected later in her pregnancy. When an infection is suspected, the antibodies should be repeated every 2–4 weeks till at least 3 months of age.
- Imaging
- The main findings on computed tomography (CT) of the head include diffuse intracranial periventricular calcifications, brain atrophy or hydrocephalus
- CSF examination—CSF composition is usually abnormal with high protein levels (over 1 g/dl) and mild elevation of WBC with monocyte predominance.
- Hemogram—A complete blood count may demonstrate anemia or thrombocytopenia.

14.2 Congenital Cytomegalovirus (CMV) Infection

CMV is the reason for most common intrauterine infection, varying from 0.5 to 2.4% of live births [21]. However, it is usually subclinical.

14.2.1 Clinical Manifestation [22]

Systemic features

- Neurological features includes intracranial calcification, brain atrophy, ventriculomegaly, microcephaly, seizures, and hypotonia
- Hearing loss
- Hepatosplenomegaly
- Jaundice
- Petechiae
- Ocular features includes chorioretinitis, microphthalmos, microcornea, cataract, optic nerve hypoplasia, disc anomaly, keratitis, and glaucoma

Like congenital toxoplasmosis, chorioretinitis is the most common ophthalmic manifestation seen [11, 23–25].

Test for diagnosis of congenital cytomegalovirus infection includes:

- 1. Isolation of virus from urine
- 2. Identification of CMV-DNA by polymerase chain reaction (PCR) in urine, blood (including dried blood spots (DBS)), saliva, and cerebrospinal fluid sampled before 3 weeks of age
- 3. Detection of antigen or CMV-IgM in blood

Detection of IgM antibodies before 2–3 weeks of age in a new born is indicative of a congenital infection.

14.3 Herpes Simplex Virus (HSV) Infection

HSV strains are of two types—HSV-1 and HSV-2. HSV-1 is the oral strain and HSV-2 is genital strain. HSV-1 causes mouth lesions, eye infections, and encephalitis, while HSV-2 causes genital infection. The HSV-2 IS transmitted venereally [26, 27]. Multiple studies have reported the association of congenital cataracts with HSV infection [4, 28, 29]. Both HSV 1 and 2 were implicated in neonatal infections and more often causes Conjunctivitis, keratitis, iridocyclitis, iris atrophy, cataract, posterior synechiae, Retinitis, chorioretinitis, chorioretinal scarring, optic atrophy, optic neuritis and microphthalmos [11]. Common morphology reported in literature in Herpes congenital cataract is anterior linear cortical cataract and fluid clefts.

Diagnosis of congenital Herpes.

Serological diagnoses can be made with detection of Ig M antibody in infant blood.

14.4 Congenital Rubella Syndrome

Gregg in 1941, during epidemic of rubella in Australia, put forward that Rubella infection can be a cause of congenital cataract [30]. He had given idea of maternally transmitted infections causing ocular and systemic disease in children and it remains a milestone in ophthalmology and epidemiology. Rubella is still an important cause of congenital cataract in developing countries, despite being eradicated from developed countries [3, 31, 32].

14.4.1 Clinical Presentation

Congenital rubella syndrome (CRS) involves multiple systems of patients. Congenital heart disease, deafness, and cataract is the classical triad seen in patients with congenital rubella syndrome [23]. World health organization (WHO) has given the clinical criteria for diagnosing CRS [33] (Table 14.1). Cardiovascular abnormalities seen in this syndrome include patent ductus arteriosus (PDA) (most common), atrial septal defect, pulmonary stenosis, and ventricular septal defect [34]. Neurological abnormalities consist of microcephaly, hearing disorder, developmental delay, mental retardation, seizure disorder, and speech abnormality [34, 35] (Fig. 14.2).

Ocular features are the following, and the presence of these features would raise a suspicion of CRS [35–37]:

- Microphthalmos (Fig. 14.3).
- Opacification or cloudiness of cornea may be present (Fig. 14.4).
- Abnormal iris pattern like atrophy of iris, iritis, iridocyclitis, or iris hypoplasia may be seen. In most of the patients, pupils are nondilating (Fig. 14.5).
- Lens may be cataractous which include total, nuclear, or membranous type. Sometimes, peripheral clear area around central cataract may be seen (Fig. 14.6).
- Anterior chamber angle abnormalities and glaucoma may be present.

Infection only	Suspected	Probable	Confirmed
Only laboratory	Patient does not meet the	Patient does not have the	Patient with at least one
evidence of infection	criteria for a probable or	laboratory confirmation but	of the symptoms
demonstrated by-	confirmed case but who has	has at least two of the	clinically consistent with
Isolation of rubella	one or more of the following	following finding:	congenital rubella
virus	finding:	Cataracts	syndrome, and
OR	Cataracts	Congenital glaucoma	laboratory evidence of
Detection of	Congenital glaucoma	Congenital heart disease	congenital rubella
rubella-specific IgM	Congenital heart disease	(PDA or PS)	infection demonstrated
antibody	(PDA or PS)	Hearing impairment	by:
OR	Hearing impairment	Pigmentary retinopathy	Isolation of virus
Persistence of	Pigmentary retinopathy	OR	OR
rubella antibody at	Purpura	Patient does not have the	Detection of rubella
a higher level and	Hepatosplenomagaly	laboratory confirmation but	specific IgM
for a longer period	Jaundice	has at least one or more of	OR
of time than	Microcephaly	the following finding:	Persistence of rubella
expected from	Developmental delay	Cataracts	antibody at a higher
passive transfer of	Meningoencephalitis	Congenital glaucoma	level and for a longer
maternal antibody	Radiolucent bone disease	Congenital heart disease	period of time than
OR		(PDA or PS)	expected from passive
A specimen that is		Hearing impairment	transfer of maternal
PCR positive for		Pigmentary retinopathy	antibody
rubella virus		AND	OR
		one or more of the following:	A specimen that is
		Purpura	PCR positive for
		Hepatosplenomagaly	rubella virus
		Jaundice	
		Microcephaly	
		Developmental delay	
		Meningoencephalitis	
		Radiolucent bone disease	

Table 14.1 WHO criteria for diagnosis of CRS

• Retina may show features of pigmentary retinopathy typically salt and pepper type



Fig 14.2 Showing 6-month-old child with microcephaly with CRS

(Fig. 14.7). Maculopathy may be associated with it. In vitro studies have observed that rubella virus infection of RPE can hamper the phagocytosis function by RPE cells [38]. Subretinal neovascularization may be seen in rare cases [20].

- There might be presence of squint or nystagmus.
- Optic atrophy, optic neuritis, lens absorption, keratoconus, corneal hydrops, or even phthisis bulbi may often be associated [20]

14.5 Management

General principles of management remain similar to normal pediatric cataract surgery. Here we discuss about the management specifically related to cataract with infective etiology.



Fig. 14.3 (a, c) Two-month-old infant with clinical picture showing microphthalmos, small pupil, atrophic iris and cataract of right and left eye, respectively, (b, d)

showing microphthalmos, microcornea, shallow anterior chamber, and membranous cataract on ultrasound biomicroscopy of the same patient



Fig. 14.4 Hazy cornea, atrophic iris, and cataract in infant with CRS

14.5.1 Preoperative Evaluation

It starts with a good history taking, also including various investigations to confirm the diagnosis and prognosticating the disease. It is equally important to emphasize the need for counseling the parents to relieve their anxiety and fear related to the condition in their child.

- 1. History—a thorough history to rule out the presence of fever with rash in antenatal period should be taken.
- Investigation—TORCH profile done in cases with bilateral congenital cataract where there is high clinical suspicion based on ocular or systemic findings.

In children infected with rubella, the specific IgM is present up to 3 months of age in all confirmed cases. It is present in 86% at the age of 3–6 months, 62% at 6 months to 1 year, and 42% at 12–18 months, rarely above 18 months. Maternally transferred rubella specific IgG disappears by around 6 months of age. A persistent level of High IgG or Rubella specific IgG during the age of 1–2 years is an indication to congenital infection by this organism. Antibody levels are higher after congenital infection than after vaccination [37]. In addition, due to multisystem involvement seen in these conditions an appropriate systemic evaluation has to be undertaken in these patients.

Cardiology evaluation may reveal the presence of patent ductus arteriosus, pulmonary stenosis, ventricular septal defect, and atrial septal defect and any of these if seen have to be tackled accordingly. Neurology evaluation is done in cases with seizure and gross developmental delay. ENT evaluation should be done to rule out sensorineural hearing loss.

3. Parent counseling-It is particularly important in these cases as there is a higher rate of both intraoperative and postoperative surgical complications as compared to normal pediatric cataract surgery with normal axial length. Parents should be explained that in eyes with microphthalmos and or microcornea due to TORCH infection there is a chance of increased tissue manipulation during surgery. This in turn leads to increase in the postoperative complications including anterior chamber inflammation, posterior synechiae, glaucoma, and posterior capsular opacification (PCO). Regarding IOL, parents need to be counseled in cases where no IOL has been inserted regarding visual rehabilitation with aphakic glasses or contact lenses.

14.5.2 Intraoperative

- Starts with an examination under anesthesia to measure and note the various parameters like corneal diameter, keratometry and axial length. IOL may not be implanted if horizontal corneal diameter is less than 10 mm and axial length is less than 17 mm or at least two standard deviations below the mean for age [39].
- Anterior segment examination: Gonioscopy is performed in each patient suspected of having shallow angle with Swan Jacob goiniolens to observe for crowding of angle and also to compare the anterior chamber angles between the two eyes. Ultrasound biomi-



Fig. 14.5 CRS with iris abnormality. (a) Patchy iris atrophy with non-dilating pupil with membraneous cataract. (b) Loss of normal iris pattern with diffuse iris atrophy

croscopy (UBM) may also be done to evaluate the angle, anterior chamber depth, lens thickness, bag to bag diameter, measurement of natural lens and also to know status of posterior capsule of lens.

 Incision: In eyes with microcornea and microphthalmos—length of clear corneal incision made is kept smaller or scleral entry to anterior chamber can be done to minimize corneal trauma and postoperative decompenalong with total congenital cataract with plaque over anterior capsule (c) Iris atrophy with posterior synechiae with membranous calcified cataract

sation. Two such incisions should be made, 180° apart in nasal and temporal quadrants. The corneal incision must be meticulously built in order to prevent iris prolapse and maintain a stable anterior chamber intra- and postoperatively. In microcornea, IOL is not implanted so it averts the need for a 2.2/2.8 mm superior incision.

4. Anterior chamber depth: In microphthalmos, anterior chamber is on shallower side, so the



Fig. 14.6 Different type of cataract in CRS. (a) Total cataract, (b) nuclear cataract, (c) absorbed cataract, (d) membranous cataract

distance between the lens and corneal endothelium is reduced hence restricting the space for maneuvering during surgery. Arshinoff's soft-shell method may be useful in such cases. First cohesive ophthalmic viscosurgical device (OVD) is injected into the center of the anterior chamber in order to inflate the anterior chamber and then a dispersive OVD is injected on top to protect the corneal endothelium [40]. 5. Management of a small pupil: In Cataract due to TORCH infection particularly rubella, children may have poorly or non-dilating pupils and/or posterior synechiae. If adequate pupil dilation is not achieved after intracameral injection of mydriatics and release of posterior synechiae, cohesive OVDs may be used to facilitate mydriasis [41]. If pupil is still not dilating with cohesive OVDs, adequate size pupilloplasty



Fig. 14.7 Retinal changes in CRS. (a) Retcam picture showing mild salt and pepper retinopathy at macula. (b) Chorioretinal atrophy with Salt and pepper retinopathy.

should be done with vitrectomy cutter. This will also reduce future incidence of VAO.

6. Continuous curvilinear capsulorrhexis (CCC): Anterior continuous curvilinear capsulorhexis in microphthalmic eyes is little challenging and complicated because of shallow anterior chamber, elevated intraocular pressure and extreme elasticity of the anterior capsule in all pediatric cataract [42]. Staining of anterior capsule of lens with trypan blue dye is done under air this is preferred method. This is followed by expanding the anterior chamber by cohesive OVD till the anterior capsule becomes flat

and this enables a sufficient operating space and also counteracts the elevated posterior pressure. In some cases, cataract is membranous with fibrosed and fused anterior capsule. In such cases, incision is made on the fibrosed capsule with MVR blade and rhexis is completed 360° with the help of microvitreoretinal scissor (Fig. 14.8).

system with anterior route showing rubella pigmentary

retinopathy (c) and maculopathy (d)

 Hydrodissection and lens aspiration: Mild hydrodissection in all quadrants aids in lowering surgical time period and improving the cortical clean up [43]. Lens matter can be aspirated by either Coaxial or bimanual methods. Bimanual method of lens matter



Fig. 14.8 Intraoperative picture of a patient with CRS with absorbed cataract with fibrosis of capsule (**a**) Incision with MVR is made in the fused anterior and posterior capsule. (**b**) Central 4 mm opening is made with microinci-

sion. (c) Anterior vitrectomy is done after making opening, 360 support is present for IOL insertion in sulcus

aspiration is preferred as it helps in complete removal of lens matter including subincisional one. It additionally provides improve chamber stability and more meticulous lens aspiration.

 Posterior capsulorhexis and anterior vitrectomy: Pediatric cataract with TORCH infection has high incidence of VAO formation compared to normal [44]. Posterior capsulorhexis with or without anterior vitrectomy has been used to lessen VAO formation since 1980s [45]. This is standard method of prevention of VAO formation in pediatric cataract surgery and it is advocated by many previous studies [46–49]. So, we suggest posterior capsulorhexis in pediatric patients up to 8 years of age and anterior vitrectomy up to 6 years of age specially in cataract with TORCH infection [39, 50]. Procedure has been discussed in detail in Chap. 3.

9. IOL implantation: IOL should not be implanted in eyes with severe microcornea

or microphthalmos. If the Axial Length is >17 mm and white to white distance is >10 mm then IOL can be implanted to reduce the postoperative refractive error and amblyopia [39]. Peripheral iridectomy should be done if IOL is not implanted.

10. Viscoelastic removal and wound suturing: All Viscoelastic substance should be removed from the chamber at the end of surgery. In most of the cases high molecular weight OVD is used, so aspiration is generally easy and fast. In the end main incision which is used for IOL implantation must be sutured with 10-0 nylon suture to prevent wound leakage and thus minimize the risk of hypotony. Side incision is closed with wound hydration and if in cases side wound is leaking then wound must be secured with 10-0 nylon suture.

14.6 Complication

- Corneal injury: Risk of corneal injury is more in patients with microphthalmic eye because of the shallow anterior chamber which reduces the distance between the instruments and the cornea, making the eye more susceptible to corneal endothelial injury.
- 2. Posterior synechiae: Posterior synechiae is one of the important postoperative complications of pediatric cataract extraction with infective etiology. In microphthalmos with cataract the incidence of posterior synechiae formation after surgery reported is as much as 35.7–59.5% [51, 52]. The cause of posterior synechiae formation is intense postoperative anterior chamber inflammation. This may be due to release of rubella virus from intralenticular space and thus causes severe inflammation either by direct viral insults or by delayed hypersensitivity reaction. Live rubella virus has been isolated from the lens till 3 years of age [44]. Good postoperative inflammatory control with mydriatics like atropine 1% and intensive topical steroids minimize the formation posterior synechiae.

- Glaucoma: Pediatric cataract with infective etiology with microphthalmos has a crowding of anterior chamber angle and thus is susceptible to postoperative glaucoma. Incidence of secondary glaucoma in cataract surgery with microphalmos is 30.9–43.2% of patients [51, 52]. Management includes inclusion of a peripheral iridectomy during cataract surgery and adequate anterior vitrectomy and meticulous control of inflammation in postoperative period.
- 4. Visual axis opacification: Children have high incidence of visual axis opacification because of younger age and of course in this case incidence increases due to infective etiology of cataract. VAO after cataract surgery in microphthalmos range from 16.6 to 24.3% [51, 52].

14.7 Visual Rehabilitation

It is suggested that in eyes with microphthalmia, IOL implantation should be deferred to lessen the risks of complications such as posterior synechiae and glaucoma [51, 53]. The general management for aphakia after cataract extraction is wearing spectacles or contact lenses and follow up at regular interval. Most important is counseling of parents to encourage the use of glasses. Refraction should be done at regular interval. Parents must ensure that child is wearing glass with latest refractive power. In unilateral aphakia amblyopia therapy should be given. Alternate to spectacles is use of contact lens. In future, Secondary IOL implantation may be planned on the basis of growth of the aphakic eye.

Spectacles: It is the most important form of refractive correction in patients with binocular aphakia specially in developing countries. Advantages of glasses include affordability, safe, easy to wear compared to contact lens, can be replaced readily and monofocal and bifocal design are available.

Disadvantages are:

Cosmetic blemish and cumbersome to wear— Aphakic glasses are thick and heavy, which causes cosmetic blemish. In infant and younger kids, the nose bridge and ears are not fully developed, which makes it tough to achieve a solid suit for the spectacles.

Others problems are image magnification, restricted field of vision, colored vision, spherical aberration, prismatic aberration

Contacts lens—Contact lens is one of the best devices to visually rehabilitate the children with aphakia specially in unilateral cases.

Advantages are less magnification of image, elimination of aberration, wider and better field of view and cosmetically more acceptable.

Disadvantages are compliance in young children is poor, costly, allergic hypersensitivity to lens some may develop corneal infection.

14.8 Conclusion

Cataract with infective etiology is usually bilateral and the children often have systemic association. Most of the patients have other ocular morbidity besides having cataract. So, doing surgery in these cases is challenging. IOL implantation should be delayed in microphthalmos in view of many postoperative complications. Postoperative complications like VAO, posterior synechiae, secondary glaucoma is more in this type of cataract. These complications can be minimized with proper preoperative planning, meticulous surgery and judicious control of inflammation in postoperative period. Most of the patients can achieve good vision with proper management.

References

- Matoba A. Ocular viral infections. Pediatr Infect Dis. 1984;3:358–68.
- Cotlier E. Congenital varicella cataract. Am J Ophthalmol. 1978;86:627–9.
- Mahalakshmi B, Therese KL, Devipriya U, Pushpalatha V, Margarita S, Madhavan HN. Infectious aetiology of congenital cataract based on TORCHES screening in a tertiary eye hospital in Chennai, Tamil Nadu, India. Indian J Med Res. 2010;131:559–64.

- Raghu H, Subhan S, Jose RJ, Gangopadhyay N, Bhende J, Sharma S. Herpes simplex virus-1associated congenital cataract. Am J Ophthalmol. 2004;138:313–4.
- Madhavan HN. Laboratory investigations on viral and chlamydia trachomatis infections of the eye: Sankara Nethralaya experiences. Indian J Ophthalmol. 1999;47:241–6.
- Li Z, Yan C, Liu P, Yan R, Feng Z. Prevalence of serum antibodies to TORCH among women before pregnancy or in the early period of pregnancy in Beijing. Clin Chim Acta. 2009;403:212–5.
- Mahalakshmi B, Therese KL, Shyamala G, Devipriya U, Madhavan HN. Toxoplasma gondii detection by nested polymerase chain reaction in lens aspirate and peripheral blood leukocyte in congenital cataract patients - the first report from a tertiary eye hospital in India. Curr Eye Res. 2007;32:653–7.
- Shyamala G, Sowmya P, Madhavan HN, Malathi J. Relative efficiency of polymerase chain reaction and enzyme-linked immunosorbent assay in determination of viral etiology in congenital cataract in infants. J Postgrad Med. 2008;54:17–20.
- Lu B, Yang Y. Detection of TORCH pathogens in children with congenital cataracts. Exp Ther Med. 2016;12(2):1159–64.
- Krummenacher C, Baribaud F, Ponce de Leon M, Baribaud I, Whitbeck JC, Xu R, Cohen GH, Eisenberg RJ. Comparative usage of herpesvirus entry mediator A and nectin-1 by laboratory strains and clinical isolates of herpes simplex virus. Virology. 2004;322:286–99.
- Mets MB. Eye manifestations of intrauterine infections. Ophthalmol Clin North Am. 2001;14:521–31.
- Kota AS, Shabbir N. Congenital toxoplasmosis. [Updated 2019 Sep 22]. In: StatPearls [Internet]. Treasure Island: StatPearls Publishing; 2020. https:// www.ncbi.nlm.nih.gov/books/NBK545228/
- McLeod R, Boyer K, Karrison T, Kasza K, Swisher C, Roizen N, et al. Outcome of treatment for congenital toxoplasmosis, 1981-2004: The National Collaborative Chicago-Based, Congenital Toxoplasmosis Study. Clin Infect Dis. 2006;42:1383–94.
- McAuley J, Boyer K, Patel D, Mets M, Swisher C, Roizen N, et al. Early and longitudinal evaluations of treated infants and children and untreated historical patients with congenital toxoplasmosis. The Chicago Collaborative Treatment Trial. Clin Infect Dis. 1994;18:38–72.
- Mets M, Holfels E, Boyer KM, Swisher C, Roizen N, Stein L, et al. Eye manifestations of congenital toxoplasmosis. Am J Ophthalmol. 1996;122:309–24.
- Vutova K, Peicheva Z, Popova A, Markova V, Mincheva N, Todorov T. Congenital toxoplasmosis: eye manifestations in infants and children. Ann Trop Paediatr. 2002;22:213–8.
- Vutova K, Peicheva Z, Popova A, Markova V, Mincheva N, Todorov T. Congenital toxoplasmosis:

eye manifestations in infants and children. Ann Trop Paediatr. 2002;22:213–8.

- Datta S, Banerjee DP. Chorioretinitis in congenital toxoplasmosis. Indian Pediatr. 2003;40:790–1.
- Mittal V, Bhatia R, Singh VK, Sengal S. Low incidence of congenital toxoplasmosis in Indian children. J Trop Pediatr. 1995;41:62–3.
- O'Neill JF. The ocular manifestations of congenital infection: a study of the early effect and long-term outcome of maternally transmitted rubella and toxoplasmosis. Trans Am Ophthalmol Soc. 1998;96:813–79.
- Dobbins J, Stewart J, Demmler G. Surveillance of congenital cytomegalovirus disease, 1990-1991: Collaborating Registry Group. MMWR Morb Mortal Wkly Rep. 1992;41:35–9.
- Malm G, Engman M-L. Congenital cytomegalovirus infections. Semin Fetal Neonatal Med. 2007;12(3):154–9. https://doi.org/10.1016/j. siny.2007.01.012.
- Boniuk I. The cytomegalovirus and the eye. Int Ophthalmol Clin. 1972;12:169–90.
- Tarkkanen A, Merenmies L, Holmstrom T. Ocular involvement in congenital cytomegalic inclusion disease. J Pediatr Ophthalmol. 1972;9:82–6.
- Hittner HM, Desmond MM, Montgomery JR. Optic nerve manifestations of human congenital cytomegalovirus infection. Am J Ophthalmol. 1976;81:661–5.
- Feigin R, Cherry J. Textbook of pediatric infectious disease, vol. 1. 4th ed. Philadelphia: WB Saunders; 1998.
- Whitley RJ, Kimberlin DW, Roizman B. Herpes simplex viruses. Clin Infect Dis. 1998;26:541–55.
- Madhavan HN. Laboratory investigations on viral and chlamydia trachomatis infections of the eye: Sankara Nethralaya experiences. Indian J Ophthalmol. 1999;47:241–6.
- 29. Shyamala G, Sowmya P, Madhavan HN, Malathi J. Relative efficiency of polymerase chain reaction and enzyme-linked immunosorbant assay in determination of viral etiology in congenital cataract in infants. J Postgrad Med. 2008;54:17–20.
- Gregg NM. Congenital cataract following German measles in the mother. Trans Ophthalmol Soc Aust. 1941;3:35–46.
- 31. Papania MJ, Wallace GS, Rota PA, Icenogle JP, Fiebelkorn AP, Armstrong GL, Reef SE, Redd SB, Abernathy ES, Barskey AE, Hao L, McLean HQ, Rota JS, Bellini WJ, Seward JF. Elimination of endemic measles, rubella, and congenital rubella syndrome from the Western hemisphere; the US experience. JAMA Pediatr. 2014;168:148–55.
- Sheridan E, Aitken C, Jeffries D, Hird M, Thayalasekaran P. Congenital rubella syndrome: a risk in immigrant populations. Lancet. 2002;359:674–5.
- 33. World Health Organization. Surveillance guidelines for measles, rubella and congenital rubella syndrome in the WHO European Region. http://www.euro.who. int/__data/assets/pdf_file/0018/79020/e93035-2013. pdf?ua=1.

- Cooper LZ, Ziring PR, Ockerse AB, Fedun BA, Kiely B, Krugman S. Rubella clinical manifestations and management. Am J Dis Child. 1969;118:18–9.
- Vijayalakshmi P, Kakkar G, Samprathi A, et al. Ocular manifestations of congenital rubella syndrome in a developing country. Indian J Ophthalmol. 2002;50:307–11.
- 36. Vijayalakshmi P, Rajasundari TA, Prasad NM, et al. Prevalence of eye signs in congenital rubella syndrome in South India: a role for population screening. Br J Ophthalmol. 2007;91:1467–70.
- Boger WP III, Petersen RA, Robb RM. Spontaneous absorption of the lens in the congenital rubella syndrome. Arch Ophthalmol. 1981;99:433–4.
- Williams LL, Lew HM, Davidorf FH, et al. Altered membrane fatty acids of cultured human retinal pigment epithelium persistently infected with rubella virus may affect secondary cellular function. Arch Virol. 1994;134:379–92.
- Khokhar SK, Pillay G, Dhull C, Agarwal E, Mahabir M, Aggarwal P. Pediatric cataract. Indian J Ophthalmol. 2017;65(12):1340–9.
- Arshinoff SA. Dispersive-cohesive viscoelastic soft shell technique. J Cataract Refract Surg. 1999;25(2):167–73.
- Jhanji V, Sharma N, Vajpayee RB. Management of intraoperative miosis during pediatric cataract surgery using healon 5. Middle East Afr J Ophthalmol. 2011;18(1):55–7.
- Yu SY, Lee JH, Chang BL. Surgical management of congenital cataract associated with severe microphthalmos. J Cataract Refract Surg. 2000;26(8):1219–24.
- Vasavada AR, Trivedi RH, Apple DJ, et al. Randomized, clinical trial of multiquadrant hydrodissection in pediatric cataract surgery. Am J Ophthalmol. 2003;135:84–8.
- Cordes FC. Surgery of congenital cataracts. Am J Ophthal. 1948;31:1073.
- Parks MM. Management of the posterior capsule in congenital cataracts. J Pediatr Ophthalmol Strabismus. 1984;21:114–7.
- BenEzra D, Cohen E. Posterior capsulectomy in pediatric cataract surgery the necessity of a choice. Ophthalmology. 1997;104:2168–74.
- Vasavada A, Desai J. Primary posterior capsulorhexis with and without anterior vitrectomy in congenital cataracts. J Cataract Refract Surg. 1997;23(Suppl 1):645–51.
- Alexandrakis G, Peterseim MM, Wilson ME. Clinical outcomes of pars plana capsulotomy with anterior vitrectomy in pediatric cataract surgery. J AAPOS. 2002;6:163–7.
- 49. Guo S, Wagner RS, Caputo A. Management of the anterior and posterior lens capsules and vitreous in pediatric cataract surgery. J Pediatr Ophthalmol Strabismus. 2004;41:330–7.
- Lin AA, Buckley EG. Update on pediatric cataract surgery and intraocular lens implantation. Curr Opin Ophthalmol. 2010;21:55–9.

- 51. Vasavada VA, Dixit NV, Ravat FA, Praveen MR, Shah SK, Vasavada V, Vasavada AR, Trivedi RH. Intraoperative performance and postoperative outcomes of cataract surgery in infant eyes with microphthalmos. J Cataract Refract Surg. 2009;35:519–28.
- 52. Shah SK, Praveen MR, Vasavada AR, Vasavada VA, Carelli R, Trivedi RH, Rasoebala V. Long-term longitudinal assessment of postoperative outcomes

after congenital cataract surgery in children with congenital rubella syndrome. J Cataract Refract Surg. 2014;40(12):2091–8. https://doi.org/10.1016/j. jcrs.2014.04.028.

53. Yu YS, Kim SJ, Choung HK. Posterior chamber intraocular lens implantation in pediatric cataract with microcornea and/or microphthalmos. Korean J Ophthalmol. 2006;20(3):151–5.