

# Evaluating Risk Factors of Glaucoma Following Paediatric Cataract Surgery

Birgitte Haargaard and John Thygesen

Department of Ophthalmology, Copenhagen University Hospital, Glostrup Hospital and Rigshospitalet

## Abstract

The risk of developing glaucoma after paediatric cataract surgery is substantial. A number of risk factors have been associated with the post-operative development of glaucoma, the most important and consistent one being age at the time of surgery. Glaucoma occurs in up to one-third of patients who have had cataract surgery at an early age (<9 months), with a particularly high risk when surgery is performed during the first four weeks of life. The discovery of glaucoma in children <2 years of age often requires additional measurements apart from the intraocular pressure measurement. Young children have elastic eye bulbs, and an increase in axial length, an increase in corneal diameter and/or an increase in excavation of the optic disc may be the only indication of glaucoma. The risk of glaucoma after paediatric cataract surgery continues throughout life, and it is therefore pertinent to perform glaucoma evaluation of these patients during follow-up.

## Keywords

Cataract, children, glaucoma, risk factor, cataract surgery

**Disclosure:** The authors have no conflicts of interest to declare.

**Received:** 10 July 2009 **Accepted:** 25 July 2009 **DOI:** 10.17925/EOR.2009.03.01.27

**Correspondence:** Birgitte Haargaard, Department of Ophthalmology, Copenhagen University Hospital, Glostrup Hospital, Nordre Ringvej 57, DK-2300 Glostrup, Denmark.  
E: birgitte@haargaard.dk or bgd@ssi.dk

Glaucoma is one of the most severe complications after surgery for paediatric cataract, and is reported to occur in up to one-third of these eyes.<sup>1-8</sup> The clinical presentation of glaucoma after paediatric cataract surgery is largely divided into two subtypes: early-onset secondary angle-closure glaucoma and secondary open-angle glaucoma. Secondary angle-closure glaucoma often presents with corneal oedema, distorted pupil, inflammation and increased intraocular pressure (IOP), whereas open-angle glaucoma generally presents with more subtle clinical signs that are undetectable by a hand-held slit-lamp examination at the beginning. After prolonged increased IOP in both types of secondary glaucoma, increased corneal diameter, ruptures of Descemet's membrane and corneal oedema may be seen in patients <2 years of age.

The pathophysiology of angle-closure glaucoma is probably increased inflammation, which may be caused by retained lens material and/or vitreous causing pupillary block. The cause of open-angle glaucoma is not fully understood, other than the preceding cataract surgery. Deferred normal maturation of the trabecular meshwork or chronic inflammation after surgery, perhaps caused by contact with vitreous body substances, or even the post-operative use of steroids, have been suggested as causes.<sup>9</sup> Open-angle glaucoma may occur months after primary surgery, but continues to occur >10 years after surgery,<sup>2,3,8,10-12</sup> highlighting the importance of continuous follow-up.

## Risk Factors

Several risk factors have been suggested to be associated with the post-operative development of glaucoma after paediatric cataract surgery. Paediatric cataract appears most frequently as an isolated disease, but may be associated with other eye malformations and/or systemic

diseases.<sup>13-15</sup> Some ocular anomalies, e.g. aniridia, are associated with both early cataract and an independent risk of glaucoma. Systemic diseases such as Lowe's (oculocerebrorenal) or rubella syndrome are also associated with both cataract and glaucoma development. The presence of other ocular anomalies, such as microcornea, has been found in some studies to increase the risk of glaucoma after cataract surgery.<sup>2-5,16</sup> It has been proposed that microcornea may be associated with a simultaneous malformation of the trabecular meshwork microstructure that increases the risk of glaucoma.<sup>11,17</sup> However, it has been reported that the majority of cases with both microcornea and cataract undergo early cataract surgery (<9 months of age),<sup>5,8</sup> which makes the interpretation of a direct association between microcornea and secondary glaucoma tricky. In fact, age at surgery appeared exclusively important in studies stratifying by age at surgery.<sup>8,18</sup>

Certain cataract types have been suggested to be risk factors for post-operative glaucoma.<sup>4,16,19,20</sup> The focus in the literature has been on nuclear cataracts. Nuclear opacities are with the central position more likely to cause visually significant cataracts, and nuclear cataracts may therefore have earlier lensectomy. Early surgery and/or an associated developmental anomaly of the anterior chamber angle are possible explanations for the increased risk. It is obvious that cataract surgery in itself is the necessary event in developing glaucoma in eyes with no known competing risk factors. Moreover, post-operative complications with retained lens material, pupillary block or vitreous in the trabecular meshwork may increase the risk.<sup>12,17,19</sup> Further surgeries for secondary cataract have been found to increase the glaucoma risk by some authors<sup>5,17</sup> but not by others.<sup>8,20</sup> The theoretical cause of glaucoma being vitreous contact with the trabecular meshwork has drawn

attention to the surgical technique for paediatric cataracts, including posterior capsulorhexis and anterior vitrectomy. This technique was found to be associated with an increased risk of glaucoma in one study;<sup>5</sup> however, as the author argues, posterior capsulorhexis/ anterior vitrectomy was used on almost all patients who were operated on at the high-risk age of <9 months. In another study, the surgical technique was no longer significant when adjusting for age at surgery.<sup>8</sup> The role of primary intraocular lens (IOL) implantation in decreasing<sup>21-23</sup> or increasing<sup>24-26</sup> the risk of glaucoma has been debated. Theories for the former effect are that the IOL may prevent vitreous substances from accessing the trabecular meshwork or that the IOL provides mechanical support mimicking that of the natural lens. However, the seemingly protective role of IOL is more likely to be due to the fact that most children with primary IOL implantation are older at the time of surgery than children left aphakic.<sup>8,18</sup>

The most important risk factor for glaucoma after paediatric cataract surgery is age at surgery.<sup>1,5,8,17,18-20,27-29</sup> In one study, early age at detection was the only significant risk factor for secondary open-angle glaucoma, but the authors argue that this is because of the close correlation between age at detection and age at surgery.<sup>29</sup> The risk of glaucoma is higher among children with an age at surgery below nine to 12 months. The risk is increased seven-fold in children <9 months of age at surgery compared with children who were older at the time of surgery.<sup>8</sup> Cataract surgery should be avoided during the first four weeks of life because of a particularly higher risk in this age group.<sup>27</sup> The risk of glaucoma after cataract surgery continues to be present many years after surgery.<sup>8,27,29</sup>

## Diagnosis of Glaucoma

Open-angle glaucoma in children <4-5 years of age may remain undetected because of difficulties in measuring the IOP in an unco-operative child, but also because glaucoma diagnosis may be difficult in children <2 years of age. In this age group, IOP is not always clearly increased. Indications of glaucoma are an increase in excavation of the optic disc with a thinning of the nerve fibre rim, an increase in corneal diameter with ruptures of Descemet's membrane and/or an increase in axial length.<sup>30</sup> The increase in axial length is particularly characteristic in children <2 years of age because of the elasticity of the young eye bulb. Therefore, it is crucial to measure not only IOP in these children, but also axial length and corneal diameter, as well as to perform ophthalmoscopy at every post-operative examination. Depending on the method of measuring IOP, it is important to realise that the values measured under general

anaesthesia may be erroneously low. Another fallback is the fact that the central corneal thickness may be higher in children having had cataract surgery with or without IOL, which may lead to a measurement of erroneously higher IOPs.<sup>31</sup>

## Conclusion

It is beyond doubt that a child born with dense bilateral congenital cataract requires early surgery to avoid amblyopia, probably before eight to 10 weeks of age.<sup>32-34</sup> However, there is a high risk of post-operative development of glaucoma in children with early surgical intervention, even >10 years after surgery where the risk is up to 30%.<sup>8</sup> The risk is higher among children who are <9 months of age at surgery, with a seven-fold increased risk compared with children who are older at surgery, and the risk is very high during the first four weeks of life. Whether or not the child has a primary IOL or not, they should have a close follow-up for life with the necessary examinations appropriate for age performed at each visit. ■



Birgitte Haargaard is a Resident in the Department of Ophthalmology at Glostrup Hospital at Copenhagen University Hospital. In 2009, she completed the European Society of Ophthalmology European Leadership Programme (SOE EuLDP), a European ophthalmology leadership development programme. Dr Haargaard's main research interests are childhood cataract and glaucoma, and her published work has mainly focused on population-based studies on the epidemiology of

childhood cataract, risk factors for the disease and post-operative complications. She is a Board Member and Scientific Secretary of the Danish Ophthalmological Society and a member (co-opted) of the steering group of the Swedish Registry of Paediatric Cataract Surgery. Dr Haargaard received her MD and PhD, entitled 'Childhood cataract in Denmark: incidence and risk factors', from the University of Copenhagen.



John Thygesen is an Associate Professor in the Department of Ophthalmology at Rigshospitalet at the University Hospital of Copenhagen and Director of Glaucoma and Traumatology Services. He is a Senior Consultant Ophthalmologist at the same hospital. He is an Executive Committee member and member of the Educational Committee of the European Glaucoma Society (EGS), and co-author of the EGS Guidelines 1998, 2003 and 2008. Dr Thygesen is the EGS

representative at the World Glaucoma Association and President of the Danish Glaucoma Society. He is a medical advisor for the Danish Medicines Agency under the auspices of the Ministry of the Interior and Health. He has given 468 presentations at national and international glaucoma and perimetry meetings and authored 158 papers and abstracts on glaucoma, ocular pharmacology, automated perimetry and ocular traumatology. He has moderated 92 national and international meetings. Dr Thygesen is an Associate Editor for *Acta Ophthalmologica* and a reviewer for several international journals.

1. Keech RV, Tongue AC, Scott WE, *Am J Ophthalmol*, 1989;108:136-41.
2. Mills MD, Robb RM, *J Pediatr Ophthalmol Strabismus*, 1994;31:355-60.
3. Miyahara S, Amino K, Tanihara H, *Graefes Arch Clin Exp Ophthalmol*, 2002;240:176-9.
4. Parks MM, Johnson DA, Reed GW, *Ophthalmology*, 1993;100:826-40.
5. Rabiah PK, *Am J Ophthalmol*, 2004;137:30-37.
6. Johnson CP, Keech RV, *J Pediatr Ophthalmol Strabismus*, 1996;33:14-17.
7. Ariturk N, Oge I, Mohajery F, Erkan D, Turkoglu S, *Int Ophthalmol*, 1998;22:175-80.
8. Haargaard B, Ritz C, Oudin A, et al., *Invest Ophthalmol Vis Sci*, 2008;49(5):1791-6.
9. Russell-Eggitt I, *J Cataract Refract Surg*, 1997;23: 664-8.
10. Asrani SG, Wilensky JT, *Ophthalmology*, 1995;102:863-67.
11. Chen TC, Walton DS, Bhatia LS. *Arch Ophthalmol*, 2004;122: 1819-25.
12. Phelps CD, Arafat NI, *Arch Ophthalmol, Invest Ophthalmol Vis Sci*, 2000;41:2108-14.
13. Wirth MG, Russell-Eggitt IM, Craig JE, et al., *Br J Ophthalmol*, 2002;86:782-6.
14. Haargaard B, Wohlfahrt J, Fledelius HC, et al., *Ophthalmology*, 2004;111:2292-8.
15. Watts P, Abdolell M, Levin AV, *J AAPOS*, 2003;7:81-5.
16. Walton DS, *Trans Am Ophthalmol Soc*, 1995;93:403-13.
17. Trivedi RH, Wilson ME Jr, Golub RL, *J AAPOS*, 2006; 10:117-23.
18. Chen TC, Bhatia LS, Halpern EF, Walton DS, *Trans Am Ophthalmol Soc*, 2006;104:241-51.
19. Lundvall A, Zetterstrom C, *Acta Ophthalmol Scand*, 1999;77:677-80.
20. Asrani S, Freedman S, Hasselblad V, et al., *J AAPOS*, 2000;4:33-9.
21. Biglan AW, *J AAPOS*, 2006;10:17-21.
22. Brady KM, Atkinson CS, Kilty LA, Hiles DA, *J Cataract Refract Surg*, 1997;23 (Suppl. 1):669-74.
23. Lambert SR, Buckley EG, Plager DA, et al., *J AAPOS*, 1999;3:344-9.
24. Wilson ME, Peterseim MW, Englert JA, et al., *J AAPOS*, 2001;5:238-45.
25. Plager DA, Yang S, Neely D, et al., *J AAPOS*, 2002;6:9-14.
26. Vishwanath M, Cheong-Leen R, Taylor D, et al., *Br J Ophthalmol*, 2004;88:905-10.
27. Magnusson G, Abrahamsson M, Sjostrand J, *Acta Ophthalmol Scand*, 2000;78:65-70.
28. Chak M, Rahi JS, *Ophthalmology*, 2008;115(6):1013-18.e2.
29. Egbert JE, Kushner BJ, *Arch Ophthalmol*, 1990;108:1257-9.
30. Simon JW, O'Malley MR, Gandham SB, et al., *J AAPOS*, 2005;9:326-9.
31. Gelbart SS, Hoyt CS, Jastrebski G, Marg E. *Am J Ophthalmol*, 1982;93:615-21.
32. Lundvall A, Kugelberg U, *Acta Ophthalmol Scand*, 2002;80:593-7.
33. Lambert SR, Lynn MJ, Reeves R, et al., *J AAPOS*, 2006;10:30-36.