CLINICAL DIAGNOSIS

Catherine Chiarelli, OD, FAAO
Vision Institute of Canada

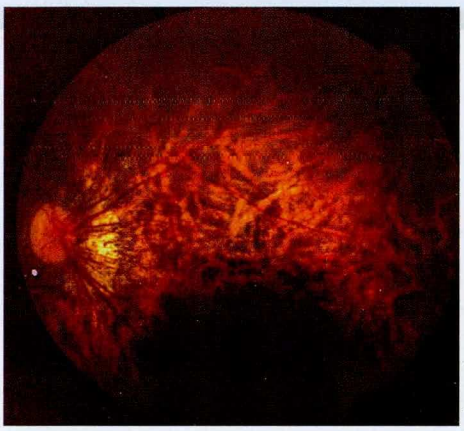

photo 1

A 12-year-old male presents for vision examination, along with teachers and classmates from the local school for deaf children. An initiative to ensure that the students receive regular vision care was undertaken by the school, in response to concerns about undetected and untreated vision problems in this at-risk population. The child's teachers have observed that his left eye turns out, and that he displays difficulty with glare. He himself reports that vision in the left eye is poor, and that people must stand on his right side in order for him to communicate with them by sign language. His kindergarten teacher remembers that he was supposed to wear a contact lens in one eye, however this was seldom done. The child reports that he has glasses at home, but that he never wears them. No further information regarding the cause of deafness, health status, visual status or ongoing vision care was available, in part due to language barriers between the school and the family.

Unaided visual acuity in the right eye is 10/10- at distance and at near. Acuity in the left eye cannot be determined at distance but is quantified as 20/400-20/500 at near. A large angle constant left exotropia is evident. No stereoacuity is appreciated. Cycloplegic refraction reveals: Right eye –0.75-0.50x180, Left eye +7.50-2.50x180. Anterior segment examination reveals aphakia in the left eye. Applanation tonometry cannot be completed due to patient apprehension, however intraocular pressures are equal by palpation. Dilated fundus examination is unremarkable in the right eye. Posterior pole of the left eye is shown in Photo 1. Non-specific pigmentary degeneration is observed peripherally in the left eye.

What condition is evident in the left eye?
What ocular history is assumed?
What treatment is advised?
What is the long-term prognosis?

(see page 119)

DIAGNOSTIC clinique
diagnositic CLINIQUE

Un garçon de 12 ans se présente pour un examen visuel, accompagné d'enseignants et d'élèves de l'école locale pour enfants sourds. L'école, préoccupée par les problèmes de la vue non détectés et non traités chez cette population à risque, a mis sur pied cette initiative pour que les élèves reçoivent des soins de la vue réguliers. Les enseignants de l'enfant ont observé un strabisme à l'œil gauche et un problème d'éblouissement. L'enfant lui-même affirme que sa vision de l'œil gauche est pauvre et que les gens doivent se placer à sa droite afin qu'il puisse communiquer avec eux par signes. Son enseignante de maternelle se rappelle qu'il devait porter une lentille de contact sur un œil, ce qu'il faisait rarement. L'enfant dit avoir des lunettes à la maison mais ne jamais les porter. Aucun renseignement supplémentaire au sujet de la cause de la surdité, de l'état de santé, de l'état visuel ou des soins de la vue en cours, en partie à cause des obstacles de langue entre l'école et la famille.

L'acuité visuelle sans aide est 10/10- de loin et de près pour l'œil droit. On ne peut préciser l'acuité visuelle de loin pour l'œil gauche mais elle s'établit à 20/400-20/500 de près. Une exotropie continuelle à grand angle à l'œil gauche est évidente. On ne dénote aucune acuité stéréoscopique. La réfraction cycloplégique révèle : œil droit –0,75-0,50x180, œil gauche +7,50-2,50x180. L'examen du segment antérieur révèle une aphakie à l'œil gauche. On ne peut effectuer la tonométrie par aplanation en raison des craintes du patient, toutefois les pressions intra-oculaires par palpation sont égales. L'examen du fond de l'œil dilaté ne révèle rien de spécial à l'œil droit. La photo 1 indique le pôle postérieur de l'œil gauche. On observe une dégénérescence pigmentaire non spécifique à la périphérie de l'œil gauche.

Quel état est évident à l'œil gauche?
Quels seraient les antécédents oculaires?
Quel est le traitement approprié?
Quel est le pronostic à long terme?

(voir la page 121)

Pathological Myopia

from page 102

Moderate myopic retinal degeneration is evident in the left eye. This is consistent with the level of refractive error predicted by the residual aphakic refraction. Myopia greater than 6D is considered to be pathological. It is caused by elongation of the globe, with characteristic fundus changes. Within the posterior pole, a temporal peripapillary atrophic crescent is seen and the retinal pigment epithelium is stretched and atrophic, causing increased visualization of choroidal vessels. Posterior staphyloma often forms, causing ectasia and outpouching of the retina, with associated pigmentary changes and variable loss of retinal function. Peripherally, there is a higher incidence of lattice degeneration, pavingstone degeneration, vitreo-retinal degeneration and retinal holes in pathological myopia. In this case, non-specific peripheral retinal degeneration is observed. There also is a higher occurrence of nuclear and posterior subcapsular cataracts in high myopia, and an increased risk of glaucoma.

The ocular history in this case is unconfirmed. It is assumed that the lens of the left eye was removed surgically, perhaps to remove a congenital cataract and/or to reduce the degree of anisometropia. Such treatment might permit refractive correction with spectacles and thereby improve the potential for visual development in the left eye after poor tolerance and compliance with contact lens correction. Unfortunately, any spectacle treatment plan was not followed through, and deep amblyopia has resulted. At this time, the prognosis for amblyopia therapy is quite poor, considering the constant exotropia, anisometropia, severely reduced visual acuity and the child's age.

A strategy to maximize remaining visual function is crucial to this hearing-impaired child. Full prescription polycarbonate spectacles were prescribed for constant wear, for eye protection. The importance of eye safety was explained to the child, with emphasis on the implications of being deaf *and* blind, should any injury damage the right eye. The responsibility for wearing protective glasses was assigned to the child, as he is old enough to comprehend the situation.

Compensatory strategies for home and school also were discussed:

1. *visual materials for learning and communication are to be placed on the right side*
2. *seat assignment in the classroom is to be on the left side of the room*
3. *interaction with people for lip-reading and sign language is to be on the right side*
4. *classroom work is to be modified for reduced copying and scanning from blackboard to desk*
5. *extreme caution and safety goggles are to be used for sports activities; contact sports are to be avoided*
6. *orientation and mobility training are to be considered, to teach compensatory safety skills in view of reduced depth perception and functional peripheral vision, so that greater independence may be permitted*
7. *an education and counselling session with the child, his parents and his teachers is to be held so that all parties understand the importance of these strategies*

The prognosis to maintain good vision in the right eye over time is favourable, provided that protection of this eye is maintained. The left eye, however, remains at risk for myopia- and aphakia-related complications, including subretinal hemorrhage, laquer cracks through the RPE, choroidal neovascularization, peripheral retinal breaks, retinal detachment and glaucoma. Regular examination, every 6-12 months, is recommended.

This hearing-impaired child's overall visual function is compromised, due to the visual impairment in the left eye. This will have significant implications in the development of communication, cognitive and learning skills throughout his life. Intervention to address these issues should have been initiated in infancy, or immediately upon diagnosis of the hearing impairment.

Pr Visudyne*
verteporfin for injection

PRESCRIBING INFORMATION (September 2004)

PrVisudyne* Verteporfin for Injection for Intravenous Use

PHOTOSENSITIZING AGENT FOR AGE-RELATED MACULAR DEGENERATION, PATHOLOGIC MYOPIA AND PRESUMED OCULAR HISTOPLASMOSIS

VISUDYNE* (verteporfin) is a drug to be used in Visudyne* Therapy. Visudyne* Therapy is a two-stage process requiring administration of both verteporfin for injection and nonthermal red light.

CAUTION: Visudyne* Therapy should only be used by physicians trained in the treatment of age-related macular degeneration and pathologic myopia using photodynamic therapy with verteporfin for injection and specified lasers. Following VISUDYNE* injection, residual photosensitivity for 48 hours or more may result in erythema and blistering of the skin when exposed to sunlight or brightly focused indoor light.

INDICATIONS AND CLINICAL USE Visudyne* Therapy is indicated for the treatment of age-related macular degeneration, pathologic myopia and presumed ocular histoplasmosis in patients with predominantly classic subfoveal choroidal neovascularization.

CONTRAINDICATIONS VISUDYNE* (verteporfin) is contraindicated for patients with porphyria or a known hypersensitivity to any component of this preparation, and in patients with severe hepatic impairment.

WARNINGS Following injection with VISUDYNE* (verteporfin), care should be taken to avoid exposure of skin or eyes to direct sunlight or bright indoor light for 2 days. In the event of extravasation during infusion, the extravasation area must be thoroughly protected from direct light until the swelling and discoloration have faded in order to prevent the occurrence of a local burn which could be severe. If emergency surgery is necessary within 48 hours after treatment, as much of the internal tissue as possible should be protected from intense light. Patients who experience severe decrease of vision of 4 lines or more within 1 week after treatment should not be retreated, at least until their vision completely recovers to pretreatment levels and the potential benefits and risks of subsequent treatment are carefully considered by the treating physician.

Caution should be exercised when Visudyne* Treatment under general anesthesia is considered (See PRECAUTIONS).

Use of incompatible lasers that do not provide the required characteristics of light for the photoactivation of VISUDYNE* could result in incomplete treatment due to partial photoactivation of VISUDYNE*, overtreatment due to overactivation of VISUDYNE*, or damage to surrounding normal tissue.

Pregnancy TERATOGENIC EFFECTS There are no adequate and well-controlled studies in pregnant women. VISUDYNE* should be used during pregnancy only if the benefit justifies the potential risk to the fetus. Rat fetuses of dams administered verteporfin for injection intravenously at ≥10 mg/kg/day during organogenesis (approximately 40-fold the human exposure at 6 mg/m^2 based on AUC_{inf} in female rats) exhibit an increase in the incidence of anophthalmia/microphthalmia. Rat fetuses of dams administered 25 mg/kg/day (approximately 125-fold the human exposure at 6 mg/m^2 based on AUC_{inf} in female rats) had an increased incidence of wavy ribs and fetal alterations. In pregnant rabbits, a decrease in body weight gain and food consumption was observed in animals that received verteporfin for injection intravenously at 10 mg/kg/day during organogenesis. The no observed adverse effect level (NOAEL) for maternal toxicity was 3 mg/kg/day (approximately 7-fold the human exposure at 6 g/m^2 based on body surface area). There were no teratogenic effects observed in rabbits at doses up to 10 mg/kg/day.

Nursing Mothers Verteporfin and its diacid metabolite have been found in the breast milk of one woman after a 6 mg/m^2 infusion. The verteporfin breast milk levels were up to 66% of the corresponding plasma levels. Verteporfin was undetectable after 12 hours. The diacid metabolite had lower peak concentrations but persisted up to at least 48 hours. Because the effects of verteporfin and its metabolite on neonates are unknown, either nursing should be interrupted or treatment postponed, taking into account the risks of delayed treatment to the mother. Women should not nurse for 96 hours after Visudyne* Therapy.

Pediatric Use Safety and effectiveness in pediatric patients have not been established.

PRECAUTIONS

General Extravasation of VISUDYNE*, especially if the affected area is exposed to light, can cause severe pain, inflammation, swelling or discoloration at the injection site. The relief of pain may require analgesic treatment.

Standard precautions should be taken during infusion of VISUDYNE* (verteporfin) to avoid extravasation. Examples of standard precautions include, but are not limited to:

- A free-flowing intravenous (IV) line should be established before starting VISUDYNE* infusion and the line should be carefully monitored.
- Due to the possible fragility of vein walls of some elderly patients, it is strongly recommended that the largest arm vein possible, preferably antecubital, be used for injection.
- Small veins in the back of the hand should be avoided.

If extravasation does occur, the infusion should be stopped immediately. The extravasation area must be thoroughly protected from direct light until the swelling and discoloration have faded in order to prevent the occurrence of a local burn which could be severe. Cold compresses should be applied to the injection site (see Warnings).

Visudyne* Therapy should be considered carefully in patients with moderate hepatic impairment or biliary obstruction since there is no clinical experience with verteporfin in such patients.

Chest pain, vaso-vagal reactions and hypersensitivity reactions, which on rare occasion can be severe, have been reported. Both vaso-vagal and hypersensitivity reactions are associated with general symptoms such as syncope, sweating, dizziness, rash, dyspnea, flushing, and changes in blood pressure and heart rate.

There is no clinical data related to the use of VISUDYNE* in anesthetized patients. At a >10-fold higher dose given by bolus injection to sedated or anesthetized pigs, verteporfin caused severe hemodynamic effects, including death, probably as a result of complement activation. These effects were diminished or abolished by pretreatment with antihistamine and they were not seen in conscious non-sedated pigs or in any other species, whether conscious or under general anesthesia. Caution should be exercised when Visudyne* Treatment under general anesthesia is considered (see WARNINGS).

VISUDYNE* at >5 times the expected maximum plasma concentration in treated patients caused a low level of complement activation in human blood in vitro. VISUDYNE* resulted in a concentration-dependent increase in complement activation in human blood in vitro. At 10 µg/ml (approximately 5 times the expected plasma concentration in human patients), there was mild to moderate complement activation. At ≥ 100 µg/mL, there was significant complement activation. Signs (chest pain, syncope, dyspnea, and flushing) consistent with complement activation have been observed in < 1% of patients administered VISUDYNE*. Patients should be supervised during VISUDYNE* infusion.

Photosensitivity Patients who receive VISUDYNE will become temporarily photosensitive for 2 days after the infusion. During that period, patients should avoid exposure of unprotected skin, eyes or other body organs to direct sunlight or bright indoor light. This includes, but is not limited to, tanning salons, bright halogen lighting and high power lighting used in surgical operating rooms or dental offices (see Warnings). Prolonged exposure to light from light emitting medical devices such as pulse oximeters should also be avoided for 48 hours following VISUDYNE* administration. If treated patients must go outdoors in daylight during the first 2 days after treatment, they should protect all parts of their skin and their eyes by wearing protective clothing and dark sunglasses. UV sunscreens are not effective in protecting against photosensitivity reactions because photoactivation of the residual drug in the skin can be caused by visible light. Patients should not stay in the dark and should be encouraged to expose their skin to ambient indoor light, as it will help inactivate the drug in the skin through a process called photobleaching.

Drug Interactions Drug interaction studies in humans have not been conducted with VISUDYNE*. Verteporfin is rapidly eliminated by the liver, mainly as unchanged drug. Metabolism is limited and occurs by liver and plasma esterases. Microsomal cytochrome P450 does not appear to play a role in verteporfin metabolism. Based on the mechanism of action of verteporfin, many drugs used concomitantly could influence the effect of Visudyne Therapy. Possible examples include the following. Calcium channel blockers, polymyxin B or radiation therapy could enhance the rate of VISUDYNE* uptake by the vascular endothelium. Other photosensitizing agents (e.g., tetracyclines, sulfonamides, phenothiazines, sulfonylurea hypoglycemic agents, thiazide diuretics and griseofulvin) could increase the potential for skin photosensitivity reactions. Compounds that quench active oxygen species or scavenge radicals, such as dimethyl sulfoxide, b-carotene, ethanol, formate and mannitol, would be expected to decrease VISUDYNE* activity. Drugs that decrease clotting, vasoconstriction or platelet aggregation, e.g., thromboxane A_2 inhibitors, could also decrease the efficacy of Visudyne Therapy.

Carcinogenesis, Mutagenesis, Impairment of Fertility No studies have been conducted to evaluate the carcinogenic potential of verteporfin. Verteporfin was not mutagenic, in the absence or presence of light, when studied in microbial mutagenicity, unscheduled DNA synthesis, mammalian point mutation, chromosome aberration, and mouse micronucleus assays.

Photodynamic therapy (PDT) as a class has been reported to result in DNA damage including DNA strand breaks, alkali-labile sites, DNA degradation, and DNA-protein cross links which may result in chromosomal aberrations, sister chromatid exchanges (SCE), and mutations. In addition, other photodynamic therapeutic agents have been shown to increase the incidence of SCE in Chinese hamster ovary (CHO) cells irradiated with visible light and in Chinese hamster lung fibroblasts irradiated with near UV light, increase mutations and DNA-protein cross-linking in mouse L5178 cells, and increase DNA-strand breaks in malignant human cervical carcinoma cells, but not in normal cells. Verteporfin was not evaluated in these latter systems. It is not known how the potential for DNA damage with PDT agents translates into human risk.

No effect on male or female reproduction has been observed in rats following intravenous administration of verteporfin for injection up to 10 mg/kg/day (approximately 60- and 40-fold human exposure at 6 mg/m^2 based on AUC_{inf} in male and female rats, respectively). Males were dosed 28 days prior to and during mating until necropsy (approximately 60 days). Females were dosed for 14 days prior to and during mating until Gestation Day 7.

Geriatric Use Approximately 90% of the patients treated with VISUDYNE* in the clinical efficacy trials were over the age of 65. A reduced treatment effect was seen with increasing age.

Fluorescein Angiography Standard precautions for fluorescein angiography should be observed. Certain medical conditions (such as pregnancy or allergy to fluorescein) may make the injection of fluorescein dye for a particular patient inadvisable in the opinion of the ophthalmologist. Approximately 1/225,000 patients may experience a severe reaction resulting in a heart attack, stroke, or death. Most reactions are mild, such as temporary nausea or vomiting in a few patients and a rash, hives, or wheezing in about 1%.

Effects on ability to drive and use machines Following Visudyne* Therapy, patients may develop transient visual disturbances such as abnormal vision, vision decrease, or visual field defects that may interfere with their ability to drive or use machines. Patients should be advised to not drive or use machines as long as these symptoms persist.

ADVERSE REACTIONS In randomized clinical trials in choroidal neovascularization, mainly in patients with age-related macular degeneration (AMD), the most frequently reported adverse events to VISUDYNE* (verteporfin) are injection site reactions (including pain, edema, inflammation, extravasation, rashes, and less commonly, hemorrhage and discoloration) and visual disturbances (including blurred vision, flashes of light, decreased visual acuity and visual field defects such as grey or dark haloes, scotoma and black spots). These events occurred in approximately 10-30% of patients. The following events, listed by Body System, occurred in 1-10% of patients:

Ocular Treatment Site: Blepharitis, cataracts, conjunctivitis/conjunctival injection, dry eyes, ocular itching, severe vision decrease with or without subretinal or vitreous hemorrhage

Body as a Whole:	Asthenia, infusion related pain primarily presenting as back pain, fever, flu syndrome, photosensitivity reactions.
Cardiovascular:	Atrial fibrillation, hypertension, peripheral vascular disorder, varicose veins
Dermatologic:	Eczema
Digestive:	Constipation, nausea
Hemic and Lymphatic:	Anemia, white blood cell count decreased, white blood cell count increased
Hepatic:	Elevated liver function tests
Metabolic/Nutritional:	Albuminuria, creatinine increased
Musculoskeletal:	Arthralgia, arthrosis, myasthenia
Nervous System:	Hypesthesia, sleep disorder, vertigo
Respiratory:	Cough, pharyngitis, pneumonia
Special Senses:	Cataracts, decreased hearing, diplopia, lacrimation disorder
Urogenital:	Prostatic disorder

Severe vision decrease, equivalent of 4 lines or more, within 7 days has been reported in 1-4% of patients. At least partial recovery of vision, defined as more than one line improvement of vision following the event, occurred in most patients (approximately 75% of patients).

Photosensitivity reactions usually occurred in the form of skin sunburn following exposure to sunlight during the first 2 days after treatment usually within 24 hours of VISUDYNE* infusion. The higher incidence of back pain in the VISUDYNE* group occurred primarily during infusion and was not associated with any evidence of hemolysis or allergic reaction and usually resolved by the end of the infusion.

The following adverse events have occurred either at low incidence (<1%) during clinical trials or have been reported during the use of VISUDYNE* in clinical practice where these events were reported voluntarily from a population of unknown size and hence the frequency of occurrence cannot be determined precisely. They have been chosen for inclusion based on factors such as seriousness, frequency of reporting, possible causal connection to VISUDYNE*, or a combination of these factors:

Ocular Treatment Site: Retinal detachment (nonrhegmatogenous), retinal or choroidal vessel nonperfusion, severe vision decrease with retinal hemorrhage.

Nonocular Events: Chest and back pain (which may radiate to other areas including but not limited to pelvis, shoulder, girdle or rib cage) and other musculoskeletal pain during infusion.

Vaso-vagal and hypersensitivity reactions can occur, which on rare occasions can be severe. General symptoms can include headache, malaise, syncope, sweating, dizziness, rash, urticaria, pruritus, dyspnea, flushing and changes in blood pressure or heart rate.

Adverse reactions reported in treated eyes in patients with pathologic myopia or presumed ocular histoplasmosis were similar to those reported in AMD patients.

SYMPTOMS AND TREATMENT OF OVERDOSAGE Overdose of drug and/or light in the treated eye may result in nonperfusion of normal retinal vessels with the possibility of severe decrease in vision that could be permanent. An overdose of drug will also result in the prolongation of the period during which the patient remains photosensitive to bright light. In such cases, it is recommended to extend the photosensitivity precautions for a time proportional to the overdose.

DOSAGE AND ADMINISTRATION A course of Visudyne* Therapy is a two-step process requiring administration of both drug and light. The first step is the intravenous infusion of VISUDYNE* (verteporfin). The second step is the activation of VISUDYNE* with light from a nonthermal diode laser. The physician should re-evaluate the patient every 3 months and if choroidal neovascular leakage is detected on fluorescein angiography, therapy should be repeated.

Lesion Size Determination The greatest linear dimension (GLD) of the lesion is estimated by fluorescein angiography and color fundus photography. All classic and occult CNV, blood and/or blocked fluorescence, and any serous detachments of the retinal pigment epithelium should be included for this measurement. Fundus cameras with magnification within the range of 2.4-2.6X are recommended. The GLD of the lesion on the fluorescein angiogram must be corrected for the magnification of the fundus camera to obtain the GLD of the lesion on the retina.

Spot Size Determination The treatment spot size should be 1000 microns larger than the GLD of the lesion on the retina to allow a 500 micron border, ensuring full coverage of the lesion. The maximum spot size used in the clinical trials was 6400 microns. The nasal edge of the treatment spot must be positioned at least 200 microns from the temporal edge of the optic disc, even if this will result in lack of photoactivation of CNV within 200 microns of the optic nerve. For treatment of lesions that are larger than the maximum treatment spot size, apply the light to the greatest possible area of active lesion.

VISUDYNE* Administration VISUDYNE* should be reconstituted according to the directions given under PHARMACEUTICAL INFORMATION, Reconstitution. The volume of reconstituted VISUDYNE* required to achieve the desired dose of 6 mg/m^2 body surface area is withdrawn from the vial and diluted with 5% Dextrose for Injection to a total infusion volume of 30 mL. The full infusion volume is administered intravenously over 10 minutes at a rate of 3 mL/minute, using an appropriate syringe pump and in-line filter. The clinical studies were conducted using a standard infusion line filter of 1.2 microns. Precautions should be taken to prevent extravasation at the injection site. If extravasation occurs, protect the site from light (see Precautions).

Light Administration Initiate 689 nm wavelength laser light delivery to the patient 15 minutes after the start of the 10-minute infusion with VISUDYNE*. Photoactivation of VISUDYNE* is controlled by the total light dose delivered. In the treatment of choroidal neovascularization, the recommended light dose is 50 J/cm^2 of neovascular lesion administered at an intensity of 600 mW/cm^2 . This dose is administered over 83 seconds. Light dose, light intensity, ophthalmic lens magnification factor and zoom lens setting are important parameters for the appropriate delivery of light to the predetermined treatment spot. Follow the laser system manuals for procedure set up and operation. The laser system must be acceptable for the delivery of a stable power output at a wavelength of 689±3 nm. Light is delivered to the retina as a single circular spot via a fiber optic and a slit lamp, using a suitable ophthalmic magnification lens. The following laser systems have been tested for compatibility with VISUDYNE* and are acceptable for the delivery of a stable power output at a wavelength of 689±3 nm:

Lumenis Opal Photoactivator laser console and modified LaserLink adapter, Manufactured by Lumenis, Inc., Santa Clara, CA

Zeiss VISULAS 690s laser and VISULINK PDT adapter, Manufactured by Carl Zeiss, Inc., Thornwood, NY.

Concurrent Bilateral Treatment The controlled trials only allowed treatment of one eye per patient. In patients who present with eligible lesions in both eyes, physicians should evaluate the potential benefits and risks of treating both eyes concurrently. If the patient has already received previous Visudyne* Therapy in one eye with an acceptable safety profile, both eyes can be treated concurrently after a single administration of VISUDYNE*. The more aggressive lesion should be treated first, at 15 minutes after the start of infusion. Immediately at the end of light application to the first eye, the laser settings should be adjusted to introduce the treatment parameters for the second eye, with the same light dose and intensity as for the first eye, starting no later than 20 minutes from the start of infusion. In patients who present for the first time with eligible lesions in both eyes without prior Visudyne* Therapy, it is prudent to treat only one eye (the most aggressive lesion) at the first course. One week after the first course, if no significant safety issues were identified, the second eye can be treated using the same treatment regimen after a second VISUDYNE* infusion. Approximately 3 months later, both eyes can be evaluated and concurrent treatment following a new VISUDYNE* infusion can be started if both lesions still show evidence of leakage.

AVAILABILITY OF DOSAGE FORMS VISUDYNE* (verteporfin) is supplied in a single-use glass vial with a gray bromobutyl stopper and aluminium flip-off cap. It contains a lyophilized cake with 15 mg verteporfin. The product is intended for intravenous injection only.

Product monograph available upon request. September 2004.

QLT Inc. Vancouver Canada V5T 4T5

Co-developed and distributed by:

NOVARTIS OPHTHALMICS

Novartis Ophthalmics, Novartis Pharmaceuticals Canada Inc. Mississauga, ON L5N 2X7

* Visudyne is a registered trademark

References:

1. Treatment of Age-Related Macular Degeneration With Photodynamic Therapy (TAP) Study Group. Photodynamic Therapy of Subfoveal Choroidal Neovascularization in Age-Related Macular Degeneration with Verteporfin. TAP Report 2. Arch Ophthalmol 2001;119:198-207

PAAB

VIS-051114

The need for deaf children to undergo early vision examination is widely recognized. The incidence of vision problems in deaf children is at least 2-3 times greater than in hearing children. The literature reports that up to 43% have significant ocular and/or vision problems, including refractive error, strabismus, amblyopia or retinopathy associated with systemic conditions. Unfortunately, there is no established standard of care to provide routine vision care to all deaf children, either through medical facilities or schools. As a result, vision problems may go undetected and untreated for many years.

In Toronto, a partnership has been established between the Vision Institute of Canada, the Toronto District School Board and a local school for the deaf. The '*Eyes to the Future*' committee ensures that all children undergo a comprehensive vision examination, with treatment and follow-up as needed. To date, 25% of children have been newly diagnosed with vision problems through the program. Diagnoses include refractive errors, strabismus, psychogenic visual impairment, accommodative esotropia, retinopathy and one case of Usher's Syndrome. Overall, approximately 43% of children have a significant vision problem requiring treatment.

Optometrists in all communities are encouraged to promote their services to schools and caregivers for deaf and hard-of-hearing students. The service is greatly needed in many cases, and it is a uniquely rewarding experience.

Myopie pathologique

from page 103

La dégénérescence rétinienne myopique modérée évidente à l'œil gauche est conforme au niveau d'erreur de réfraction prévu par la réfraction aphake résiduelle. Une myopie au-delà de 6D est considérée comme pathologique. Elle est causée par l'allongement du globe oculaire et elle présente des modifications caractéristiques du fond de l'œil. Au pôle postérieur, on voit un croissant atrophié péri-papillaire temporal, et l'épithélium pigmentaire rétinien est étiré et atrophié, causant une visualisation accrue des vaisseaux choroïdiens. Il se forme souvent un staphylome postérieur, causant une ectasie et une enflure de la rétine, avec modifications pigmentaires associées et perte variable de la fonction rétinienne. En périphérie, la myopie pathologique comporte une plus grande incidence de dégénérescence palissadique, de dégénérescence maculaire, de dégénérescence vitréo-rétinienne et de trous rétiniens. Dans ce cas-ci, on observe une dégénérescence rétinienne périphérique non spécifique. Il y a également beaucoup plus de cataractes sous-capsulaires postérieures et nucléaires dans les cas de myopie avancée, et un risque accru de glaucome.

Les antécédents oculaires dans ce cas-ci ne sont pas confirmés. On présume que le cristallin de l'oeil gauche a été retiré chirugicalement, peut-être pour enlever une cataracte congénitale et/ou réduire le degré d'anisométropie. Un tel traitement pourrait permettre une correction de la réfraction au moyen de lunettes et, par conséquent, améliorer le développement visuel de l'œil gauche à la suite de la faible tolérance et de la négligence entourant la correction par lentilles de contact. Malheureusement, il n'y a eu aucun traitement avec des lunettes et une grave amblyopie a suivi. À ce moment-ci, le pronostic d'un traitement de l'amblyopie est très peu reluisant, si l'on tient compte de l'exotropie continuelle, de l'anisométropie, de l'acuité visuelle gravement réduite et de l'âge de l'enfant.

Il est de la plus haute importance d'optimiser la fonction visuelle restante chez cet enfant malentendant. On a prescrit des lunettes en polycarbonate à port permanent pour protéger l'œil. On a expliqué à l'enfant l'importance de protéger l'œil et les conséquences d'une cécité *et* d'une surdité à la suite d'une blessure à l'œil droit. On a attribué à l'enfant la responsabilité de porter ses lunettes de protection, car il est suffisamment âgé pour comprendre la situation.

On a aussi discuté de stratégies compensatoires pour l'école et la maison :

1. *placer le matériel visuel d'apprentissage et de communication à la droite de l'enfant*
2. *asseoir l'enfant du côté gauche de la classe*
3. *les intervenants pour la lecture labiale et le langage gestuel devraient être placés à la droite de l'enfant*

4. *diminuer en classe le travail de copie et de visualisation au tableau*
5. *extrême prudence et lunettes de sécurité pour les activités sportives; éviter les sports de contact*
6. *rééducation de l'orientation et de la mobilité pour donner à l'enfant des capacités compensatoires sécuritaires en raison de sa perception de la profondeur et de sa vision périphérique fonctionnelle réduites, ce qui lui permettrait une plus grande autonomie*
7. *tenir une rencontre d'aide et de formation avec l'enfant, les parents et les enseignants afin que toutes les parties comprennent l'importance de ces stratégies*

Le pronostic de maintenir une bonne vision de l'œil droit au fil du temps est favorable, à condition de protéger cet œil. Toutefois, l'œil gauche demeure à risque d'être affecté par des problèmes liés à la myopie et à l'aphakie, comme une hémorragie sous-rétinienne, des bris en laque de l'EPR, l'apparition d'une néovascularisation choroïdienne, des altérations rétiniennes périphériques, un décollement de rétine et un glaucome. On recommande un examen régulier tous les 6 à 12 mois.

Comme la fonction visuelle globale de cet enfant malentendant est compromise en raison du handicap visuel de l'œil gauche, elle aura des incidences significatives sur le développement des habiletés de communication, cognitives et d'apprentissage tout au long de sa vie. On aurait dû intervenir dès l'enfance pour répondre à ces problèmes ou immédiatement après le diagnostic du handicap auditif.

On reconnaît généralement la nécessité pour les enfants malentendants de subir un examen visuel précoce. L'incidence de problèmes visuels chez les enfants malentendants est au moins deux à trois fois plus élevée que chez les enfants sans problème d'audition. Selon la documentation, jusqu'à 43 % d'entre eux ont des problèmes oculaires et/ou visuels majeurs, comme l'erreur de réfraction, le strabisme, l'amblyopie ou la rétinopathie associées à des états systémiques. Malheureusement, on ne trouve aucune norme de soins prévoyant des soins de la vue réguliers à tous les enfants malentendants par l'entremise d'institutions médicales ou scolaires. Voilà pourquoi de nombreux problèmes visuels peuvent demeurer non détectés et non traités pendant une longue période.

À Toronto, on a établi un partenariat entre le Vision Institute of Canada, le Toronto District School Board et une école locale pour sourds. Le Comité « *Eyes to the Future* » fait en sorte que tous les enfants reçoivent un examen de la vue complet avec traitement et suivi au besoin. Grâce au programme, on a récemment diagnostiqué des problèmes de la vue chez 25 % des enfants. Les diagnostics incluent : erreur de réfraction, strabisme, handicap visuel psychogène, ésotropie accomodative, rétinopathie et un cas de syndrome de Usher. Globalement, environ 43 % des enfants ont un problème de vision important nécessitant un traitement.

On invite les optométristes de toutes les collectivités à offrir leurs services aux écoles et aux aidants travaillant auprès d'élèves sourds et malentendants. Ce service est urgent dans beaucoup de cas et représente une expérience très enrichissante.

References

Atebara NH. Miscellaneous Abnormalities of the Fundus in Duane's Foundations of Clinical Ophthalmology. Lippincott Williams & Wilkins. Philadelphia. 2001. Volume 3, Chapter 36, p. 4-8.

Cheng KP, Biglan AW. Pediatric Cataract Surgery in Duane's Foundations of Clinical Ophthalmology. Lippincott Williams & Wilkins. Philadelphia. 2001. Volume 6, Chapter 100, p. 1-10.

Datiles MB, Magno BV. Cataract: Clinical Types in Duane's Foundations of Clinical Ophthalmology. Lippincott Williams & Wilkins. Philadelphia. 1994. Volume 1, Chapter 73, p. 11-15.

Guy R, Nicholson J, et. al. A clinical evaluation of ophthalmic assessment in children with sensori-neural deafness. Child Care Health Dev. Sep; 29(5): 377-84. 2003.

Layden WE. Cataracts and Glaucoma in Duane's Foundations of Clinical Ophthalmology. Lippincott Williams & Wilkins. Philadelphia. 1994. Volume 3, Chapter 55, p. 5-6.

Nikolopoulos TP, Loumi D, et. al. Evidence-based overview of ophthalmic disorders in deaf children: a literature update. Otol Neurol Feb: 27(2 Suppl 1):S1-24. 2006.

Woodruff ME. Differential effects of various causes of deafness on the eyes, refractive errors and vision of children. Am J Physiol Opt Aug; 63(8):668-75. 1986.