Retinal Thickness

Related terms:

Neovascularization (Pathology), Glaucoma, Retinal, Visual Acuity, Retina Detachment, Macular Edema, Diabetic Retinopathy, Diabetic Macular Edema, Optical Coherence Tomography

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Optic nerve imaging

Larissa Camejo, Robert J Noecker, in Becker-Shaffer's Diagnosis and Therapy of the Glaucomas (Eighth Edition), 2009

Macular analysis

A retinal thickness analysis and a retinal map analysis can be obtained from the macular scan data. The retinal thickness printout provides a cross-sectional image of the retina along a specific axis of scan (indicated in the printout), signal strength, and a thickness chart with background shaded areas representing the normative database. A retinal thickness measurement is also provided. The retinal map analysis also provides a cross-sectional image and includes two maps, one with qualitative and another with quantitative thickness measurements. Measurements for nine macular sectors are shown as well as thickness measurements for the center of the scan and the total macular volume (Fig. 14-14).

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Posterior eye imaging

James S. Wolffsohn PhD PgC PgDipAdvClinOptom, in Ophthalmic Imaging, 2008

Retinal thickness analyser (RTA)

The retinal thickness analyser projects a vertical narrow green helium-neon (543 nm) laser slit beam at an angle on the retina while a CCD camera records the backscattered light. Due to the oblique projection of the beam and the transparency of the retina, the backscattered light returns two peaks corresponding to the vitreoretinal and the chorioretinal interfaces. The calculated algorithm distance between the two light peaks determines the retinal thickness at a given point. A 3 × 3 mm scan consisting of 16 optical cross-sections is acquired within 0.3 seconds. Five such scans are obtained at the macula, three scans at the disc, and additional five scans cover the peripapillary area. During scanning, the RTA acquires a red-free fundus image. Using blood vessels as guidelines, registration software automatically overlays the map on the fundus image, enhancing reproducibility and measurement accuracy (Fig. 5.12). Using edge detection analysis, the topography algorithm identifies the left border of the light, corresponding to the vitreoretinal surface, and calculates the disc topography. In order to obtain quantitative stereometric measurements, the examiner draws a contour line along the disc edge, which is used in follow-up visits to ensure accurate monitoring of subtle changes. Hoffman et al 2005 found a moderate agreement on optic disc parameters between RTA, OCT and HRT II, but there were discrepancies between them. Macular oedema from diabetic retinopathy was more reliably detected with OCT and HRT II than with RTA (Guan et al 2004, Goebel and Franke 2006).

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Nonproliferative Diabetic Retinopathy and Diabetic Macular Edema

Henry E. Wiley, Frederick L. FerrisIII, in Retina (Fifth Edition), 2013

Diurnal variation of DME

On average, retinal thickness in DME decreases slightly during the day, but the proportion of eyes with DME exhibiting clinically meaningful changes is small. In the largest study to date, 156 eyes of 96 participants with center-involved DME on ophthalmoscopy and CSMT of 225 microns or greater were evaluated at six time points between 8am and 4pm using the Stratus OCT.[126] Two scans of adequate quality were obtained at each time point and sent to a reading center. The mean change in relative central subfield thickening, defined to represent the change in excess retinal thickness, was a decrease of 6% (95% CI –9% to –3%) between the 8am and 4pm time points. The mean absolute change was a decrease in 13 μm (95% CI –17 to –8 μm). Three percent (5 of 156) of eyes met a composite endpoint of

25% or greater decrease in relative central subfield thickening and 50 μm or greater decrease in CSMT at two consecutive time points, and 1% (2 of 156) of eyes exhibited increases in both measures by at least these amounts.

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SURGICAL RETINA

In Moorfields Manual of Ophthalmology, 2008

Background

A full-thickness retinal defect centred on the fovea. Commonest in late-middle-aged women. Usually idiopathic but may occur in high myopes, following trauma, or prolonged cystoid macular oedema.

Symptoms

Reduced VA, distortion or incidental finding.

Signs

- *Stage I*: yellow spot with loss of normal foveolar depression.
- *Stage II*: round or curvilear full-thickness retinal defect (<350 μm).
- *Stage III*: full-thickness macular hole (FTMH). A pre-foveal operculum is common.
- *Stage IV*: FTMH with complete posterior vitreous detachment (Fig. 11.9). Associated findings include a grey cuff of subretinal fluid surrounding the hole, fine yellow-white deposits in the base of the hole, underlying RPE atrophy and occasionally retinal detachment in high myopes. VA is typically 6/24–6/60. Approximately 30% also have an epiretinal membranes (p. 536).

History and examination

Record: duration and severity of symptoms, near and distance VA, lens and refractive status, vitreous attachment, stage of hole and check retinal periphery. Perform Watzke-Allen test using a macular lens: shine a narrow slit of light vertically across the hole; ask, 'Is the line of light continuous, narrowed, or broken?' If significantly narrowed (>50%) or broken, the test is positive, suggesting a FTMH rather than pseudo- or lamellar hole. Repeat in the horizontal meridian.

Differential diagnosis

Partial-thickness (lamellar) holes tend not to have cuff of fluid around the hole or RPE changes but they may progress to FTMH. *Pseudohole* an epiretinal membrane or macular cysts create the appearance of a FTMH.

Investigations

Not routinely required.

- *B-scan ultrasonography*: may show posterior vitreous detachment if not visible clinically.
- Fluorescein angiography: RPE atrophy may give central hyperfluorescence with the cuff of subretinal fluid producing a hypofluorescent annulus.
- *Ocular coherence tomography*: if available, may confirm uncertain cases.

Management

- *Casualty*: routine clinic referral.
- *Clinic*: studies suggest stage I holes do not benefit from vitrectomy and gas injection but consider for stage II–IV holes of up to 9–12 months duration. Surgical success probably reduces the longer the hole has been present, but the duration may not be known and surgery is done as the only way to possibly improve vision. To maximize gas tamponade many, but not all, recommend face-down posturing for 50 minutes in every hour, for 7–14 days. Many peel the internal limiting membrane and some use adjuncts such as serum or autologous platelets. Spontaneous hole closure occurs in 12% but VA tends to remain unchanged.

Consent

Stage III and IV: approximately 80% anatomic closure; 70% get 2 line VA improvement, 10% no change, 10% lose VA. Explain the risks of vitrectomy and gas injection (p. 532). Failed primary surgery may warrant re-operation.

Follow–up

- *No surgery*: discharge for annual optometrist review.
- *Postoperative*: G. Chloramphenicol 0.5% q.d.s. 2 weeks; G. Atropine 1% b.d. 2 weeks; G. Dexamethasone 0.1% q.d.s. two weeks, then tail off over three

weeks. Inspect for raised IOP, retinal breaks, visual and anatomic outcome, and cataract, at day 1, 7, 30, and 90, then discharge for annual optometrist review. The risk of contralateral FTMH is approximately 10–20%, but is unlikely if the vitreous is detached. Provide an Amsler grid.

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Retinal Detachment and Allied Diseases

Adam H. Rogers MD, Jay S. Duker MD, in Retina, 2008

Rhegmatogenous Retinal Detachment

Key Facts

- Caused by vitreous traction exerted on a full-thickness retinal defect, allowing liquefied vitreous fluid to enter the subretinal space
- Photopsias and floaters are early symptoms
- Patients may be asymptomatic until the macula is involved
- **Risk factors include:**• myopia • retinal detachment in opposite eye • trauma • lattice degeneration • acute posterior vitreous detachment (PVD) • pseudophakia • aphakia • Nd : YAG laser capsulotomy
- 1 in 10 000 risk

Clinical Findings

• Decreased visual acuity • PVD with liquefaction of vitreous • Pigment or hemorrhage in vitreous cavity • Elevation of the retina, with fluid separating the neurosensory retina from the retinal pigment epithelium • Corrugated appearance to the retina in more acute detachments, smooth elevation with thinning in more chronic detachments • Retinal tears primarily occurring in the vitreous base

Ancillary Testing

- Ultrasound when vitreous hemorrhage prevents direct visualization of retina

Differential Diagnosis

• Retinoschisis • Exudative retinal detachment • Traction retinal detachment • Choroidal neoplasm

Treatment

- The goal of treatment is to seal the retinal break to prevent further fluid accumulation under the retina•Relief of the vitreous traction is usually necessary
- Surgical intervention should be performed promptly to maximize visual recovery
- Chronic, subclinical retinal detachments with a demarcation line in the inferior retina not involving the macula may be observed
- Argon laser photocoagulation may be used to demarcate limited subclinical retinal detachments in superior retina and asymptomatic chronic detachments without demarcation lines in inferior retina
- Pneumatic retinopexy for retinal detachments with one or more breaks within one or two clock hours of one another occurring in the superior eight clock hours of the eye• Positioning for 3–4 days is then required to position the intraocular gas over the retinal tear for tamponade • Rarely, inferior detachments can be treated with pneumatic retinopexy and head-down positioning
- Scleral buckle (segmental or encircling) with cryopexy to retinal tears and gas tamponade with or without external drainage of subretinal fluid
- Pars plana vitrectomy with endolaser surrounding retinal tears and intravitreal gas tamponade, either alone or with an encircling scleral band

Fig. 7.5. (**A**) Inferior rhegmatogenous retinal detachment with subretinal fluid splitting the fovea. (**B**) Optical coherence tomography shows separation of the retinal pigment epithelium from the neurosensory retina.

Fig. 7.6. Laser demarcation of a chronic temporal retinal detachment.

Prognosis

- Visual outcome, regardless of method of treatment, depends primarily on preoperative visual acuity
- **Other factors limiting postoperative visual recovery include:•** formation of proliferative vitreoretinopathy • epiretinal membrane • cystoid macular edema • macular atrophy
- Untreated retinal detachments involving the fovea will lead to diminished vision in all cases
-

- Laser demarcation of subclinical retinal detachments has a high success rate, with few cases requiring more definitive surgical treatment
- Pneumatic retinopexy is successful in 80% or more of all eyes
- Scleral buckle surgery has a nearly 85–90% anatomical success rate with one operation
- Pars plana vitrectomy is successful in >90% of eyes with one operation

Overall, the anatomical success rate for repairing rhegmatogenous retinal detachments is 98%

Fig. 7.7. Chorioretinal scar formation surrounding a retinal tear in an eye 7 days after pneumatic retinopexy.

Fig. 7.8. Peripheral scleral buckle with chorioretinal atrophy 1 year after retinal detachment repair.

Fig. 7.9. A pigmented demarcation line in the inferior temporal quadrant of a patient with a subclinical retinal detachment. The detachment has been present for 9 years without any evidence of progression, and the patient remains asymptomatic.

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Diagnostic Testing

Scott M. Whitcup, in Uveitis (Fourth Edition), 2010

Optical coherence tomography

Optical coherence tomography (OCT) allows the noninvasive assessment of retinal thickness. This technique may be useful in assessing retinal edema in patients with uveitis and response to therapy. In a recent study, Antcliff and associates[54] compared OCT and fluorescein angiography in 58 patients with uveitis and suspected cystoid macular edema (CME). One hundred and eight eyes had similar results by both OCT and fluorescein angiography; 67 eyes had CME and 41 eyes had no CME. In 10 eyes subretinal fluid was detected by OCT but not by fluorescein angiography. Five of these eyes had CME detected by fluorescein angiography but not by OCT. Three other eyes had CME detected by fluorescein angiography but not by OCT. The authors concluded that OCT is as effective as fluorescein angiography for detecting CME but is superior for demonstrating axial distribution of fluid. Similar to fluorescein angiography, the accuracy of the measurements is affected by small pupil size and media opacity.

Since the last edition of this book there have been improvements in OCT technology and a number of new studies assessing the use in uveitis. OCT is useful for detecting macular edema and assessing response to therapy.[55] Reductions in macular thickness in patients with uveitic CME can be detected on OCT within a week of corticosteroid therapy (**Fig. 5-7**). OCT may be useful in detecting retinal pathology in other forms of posterior uveitis.[56] Finally, spectral-domain OCT may provide additional information and be useful in assessing uveitic eyes, especially when hazy media obscures clinical examination.[57]

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Infectious Diseases

Adam H. Rogers MD, Jay S. Duker MD, in Retina, 2008

Cytomegalovirus Retinitis

Key Facts

- Slowly progressive herpes virus affecting the retina • Full-thickness retinal necrosis • Retinitis occurs in immunocompromised patients when the CD4 count is <50 cells/mm3 • No sexual or racial predilection • Unilateral or bilateral • With the introduction of highly active antiretroviral therapy (HAART), the incidence of cytomegalovirus (CMV) retinitis has dramatically decreased • Patients may be asymptomatic or complain of decreased vision, floaters, or visual field defects

Clinical Findings

- Early infection may appear as a white lesion similar to a cotton wool spot occurring anywhere within the retina alone or as multiple foci • Retinitis slowly spreads in a brushfire-like pattern, with a leading edge of active retinitis (retinal whitening) and intraretinal hemorrhage • Pigmentation and atrophy remain in areas of inactive retinitis • Papillitis • Vascular sheathing • Mild or absent vitritis • Rhegmatogenous retinal detachment from multiple small holes in affected retina

Ancillary Testing

- HIV test • CD4 count • Color photographs of affected retina to compare with older pictures to determine if subtle reactivation is present • PCR testing of

aqueous or vitreous samples or retinal biopsy when the diagnosis is in doubt (these techniques are rarely used)

Differential Diagnosis

- Progressive outer retinal necrosis • Acute retinal necrosis • Toxoplasmosis

Treatment

- HAART includes one or more viral protease inhibitors and two or more reverse transcriptase inhibitors•This treatment should be instituted immediately in untreated patients
- In CMV retinitis that threatens the macula or in patients already receiving HAART, treatment with intravitreal or intravenous medications below should be instituted promptly
- **Ganciclovir:**•intravenous ganciclovir 5 mg/kg twice daily for 2 weeks as an induction, followed by 5 mg/kg daily as maintenance therapy; the main systemic side effect is neutropenia • oral ganciclovir 600 mg five times daily as maintenance therapy • intravitreal ganciclovir 200 μg/0.1 mL once or twice weekly • ganciclovir implant releases 1 μg/h for 6–12 months
- **Foscarnet:**•intravenous foscarnet as an induction dose of 60 mg/kg three times daily or 90 mg/kg twice daily for 2 weeks, followed by a maintenance dose of 90–120 mg/kg daily; nephrotoxicity is the main side effect and is offset with adequate intravenous hydration • intravitreal foscarnet 2400 μg/0.1 mL once or twice weekly
- **Cidofovir (intravenous):**•intravenous cidofovir 5 mg/kg weekly for 2 weeks as maintenance dose, followed by 5 mg/kg every 2 weeks as maintenance; side effects include nephrotoxicity (offset with intravenous fluids) • intravitreal cidofovir should be avoided because of the uveitis and hypotony that may form
- Valganciclovir is an oral prodrug of ganciclovir with 10 times the bioavailability and is administered in a dose of 900 mg/day
- Pars plana vitrectomy with silicone oil for rhegmatogenous retinal detachments

Prognosis

- Left untreated, CMV retinitis carries a high risk of permanent vision loss from macular involvement and/or rhegmatogenous retinal detachment
- Treatment with HAART induces immune system recovery•With the implementation of HAART therapy in the 1990s, the incidence of CMV retinitis has dramatically decreased to levels at which the infection is rarely seen

Fig. 13.3. Active CMV retinitis presenting as retinal whitening and hemorrhage in the superior retina outside of the macula.

Fig. 13.4. The inactive CMV retinitis in this HIV-positive female patient shows chorioretinal scarring with pigmentation.

Fig. 13.5. A ganciclovir implant visible through a dilated pupil.

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Optical Coherence Tomography

Carlos Alexandre de Amorim Garcia Filho, ... Philip J. Rosenfeld, in Retina (Fifth Edition), 2013

Nonproliferative diabetic retinopathy and diabetic macular edema

The important role of OCT in DME management involves the evaluation of retinal pathology, including retinal thickness, CME, intraretinal exudates, vitreomacular interface abnormalities, subretinal fluid, and photoreceptor IS/OS junction abnormalities. OCT is also important in monitoring the response to treatment of DME by laser, intravitreal pharmacotherapies, and vitreoretinal surgery.

Determination of macular edema can be difficult with biomicroscopy or color fundus imaging, especially when the edema is mild.[182–184] It has been suggested that OCT measurements may be a more sensitive and reproducible indicator of true change in retinal thickness than color fundus imaging, supporting the use of OCT as the principal method for documenting retinal thickness. However, OCT is less suitable than fundus imaging for documenting the location and severity of other morphologic features of diabetic retinopathy, such as hard exudates, retinal hemorrhages, microaneurysms, and vascular abnormalities. Furthermore, OCT cannot provide information on overall retinopathy severity, for which color photographs remain the gold standard.[185–188]

OCT can be used to distinguish patients with normal retinal contour and thickness despite extensive angiopathy from those with early retinal edema. In general, the DME can be classified into several categories: diffuse retinal thickening, CME, serous retinal detachment or subretinal fluid, and vitreomacular interface abnormality.-[189–191] Diffuse retinal thickening is usually defined as a sponge-like swelling of the retina with a generalized, heterogeneous, mild hyporeflectivity compared with normal retina. CME is characterized by the presence of intraretinal cystoid areas of low reflectivity, which are typically separated by highly reflective septa (Fig. 3.31).

Serous retinal detachment is defined on OCT as a focal elevation of neurosensory retina overlying a hyporeflective, dome-shaped space. The posterior border of the detached retina is usually highly reflective, which helps to differentiate subretinal from intraretinal fluid. Vitreomacular interface abnormalities include the presence of ERMs, VMT, or both. Intraretinal focal hyperreflections that correspond clinically to retinal exudates are a frequent finding in all the patterns described above.

OCT has become widely accepted in monitoring progression and treatment response in patients with DME. Prior to OCT imaging, precision in central retinal thickness monitoring was not possible. The ETDRS provided guidelines for laser management of patients with DME.[192–194] Although OCT was not available for use in this study, quantitative retinal thickness maps can be used to direct laser therapy and may be better than using biomicroscopy alone. In the era of pharmacotherapy, many agents like triamcinolone and anti-VEGF agents (ranibizumab and bevacizumab) have been studied to treat DME. In these studies, OCT played an important role in determining the retinal thickness and the treatment response.[195,196] The treatment response of each OCT pattern of DME has been shown to be different.[197] Patients with diffuse retinal thickening may achieve a greater reduction in retinal thickness and a greater improvement in visual acuity compared with patients exhibiting CME, subretinal fluid, or vitreomacular interface abnormality.[197,198]

Macular traction has become increasingly recognized in patients with DME, especially in eyes with persistent edema after focal laser or pharmacological treatment. These patients often show the clinical appearance of a thick posterior hyaloid with diffuse fluorescein leakage. Recognition of this condition can be difficult using the clinical exam alone. This is readily recognized on OCT imaging as diffuse cystoid retinal thickening, a flat-appearing foveal contour, and a thickened hyperreflective linear vitreoretinal interface. Focal vitreoretinal adhesions that cannot be identified on clinical exam are also often evident on OCT.[199,200] These findings can direct the decision as to whether to proceed with pars plana vitrectomy and membrane peeling.[201]

Furthermore, the improvement in axial resolution with SD-OCT has enhanced the ability to evaluate foveal microstructural abnormalities, including the photoreceptor IS/OS junction, which may reveal damage to macular photoreceptors. Several studies have demonstrated that an intact IS/OS junction is predictive of a better visual acuity in patients after treatment for DME.[202–204]

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Therapeutic monoclonal antibodies and fragments

Ainat Klein MD, Anat Loewenstein MD, in Retinal Pharmacotherapy, 2010

RETINAL TELANGIECTASIAS

Short-term results indicate that inhibition of VEGF by intravitreal bevacizumab is associated with a decrease in retinal thickness and a reduction in angiographic leakage in type 2 idiopathic macular telangiectasia (IMT). Furthermore, it may improve VA in affected patients.[44] It was also reported as a treatment of foveal detachment in idiopathic perifoveal telangiectasia (IPT). The OCT images showed marked reversal of the foveal detachment after the injection, with restoration of foveal depression in all eyes. The vision of all treated eyes improved and remained unchanged up until the last follow-up visit.[45]

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Dietary Wolfberry and Retinal Degeneration

Hua Ji, ... Dingbo Lin, in Handbook of Nutrition, Diet and the Eye, 2014

Protection against Retinal Degeneration in the Early Stage of Diabetes

The present authors and others have reported that changes in the structure and function of the retina such as damage to retinal photoreceptor and inner nuclear layers, loss of RGCs, and altered retinal thickness occur in the early stages of diabetes, before the observation of clinical retinopathy in db/db type 2 diabetic mice.[6,44] The db/db mice fed 1% (kcal) wolfberries did not develop observable retinal structural abnormalities as tested by histologic analysis.[6] Thus, wolfberry and/or its bioactive components, including but not limited to zeaxanthin and lutein, exert neuroprotective qualities via regulation of gene expression, although their antioxidant capabilities cannot be excluded in the diabetic retina.

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