Chapter 21: Glaucoma

PATIENT STORY

A 50-year-old black man was noted to have a large cup-to-disc ratio during a funduscopic examination by his primary care provider (**Figure 20-1**). The patient reported no visual complaints. Further evaluation revealed elevated intraocular pressure and early visual field defects. He was started on medication to lower his intraocular pressure. He remained asymptomatic, and his visual field defects did not progress for the next several years.

INTRODUCTION

Glaucoma is a leading cause of blindness in the United States and globally. Open-angle glaucoma is an acquired loss of retinal ganglion cells characterized by either normal or increased intraocular pressure (IOP), a large cup-to-disc ratio, and visual field defects. Open-angle glaucoma is treated by reducing IOP, most commonly with eye drops. Angle-closure glaucoma, which is much less common, is an acute increase in IOP from a mechanical obstruction that must be treated emergently to preserve vision.

EPIDEMIOLOGY

- In 2013, an estimated 65 million people worldwide had glaucoma.[1]
- The prevalence of glaucoma in the global population between ages 40 and 80 is 3.54%; Africa has the highest prevalence of open-angle glaucoma (4.2%); Asia has the highest prevalence of angle-closure glaucoma.[1]
- The incidence of primary open-angle glaucoma was 8.3 per 100,000 population in people older than 40 years in a Minnesota population study.[2]
- According to a population-based study, a family history of glaucoma increased the risk of having glaucoma (odds ratio [OR] = 3.08).[3]

ETIOLOGY AND PATHOPHYSIOLOGY

- Glaucoma pathophysiology is incompletely understood, but the level of IOP correlates with the acquired loss of retinal ganglion cells and resulting irreversible vision loss.[4]
- The ciliary body secretes aqueous humor, which drains through the trabecular meshwork and/or the uveoscleral outflow pathway; the balance between secretion and outflow determines IOP.[4]
    - In open-angle glaucoma, resistance in the trabecular meshwork impedes outflow
    - In angle-closure glaucoma, the drainage pathways is obstructed
- High systemic blood pressure affects ocular circulation and is associated with glaucoma progression.[5]
- IOP can cause mechanical stress on the posterior structures of the eye, with consequent damage to optic nerve fibers.[4]

- Optic nerve atrophy is seen as optic disc cupping and irreversible visual field loss. Compare **Figures 21-1** and **21-2** to see the difference between abnormal (**Figure 21-1**) and normal (**Figure 21-2**) optic disc cupping.

FIGURE 21-1

A 50-year-old man with glaucoma has an increased optic cup-to-disc ratio of 0.8. Median cup-to-disc ratio is 0.2 to 0.3, but varies considerably among individuals. (*Reproduced with permission from Paul D. Comeau*.)

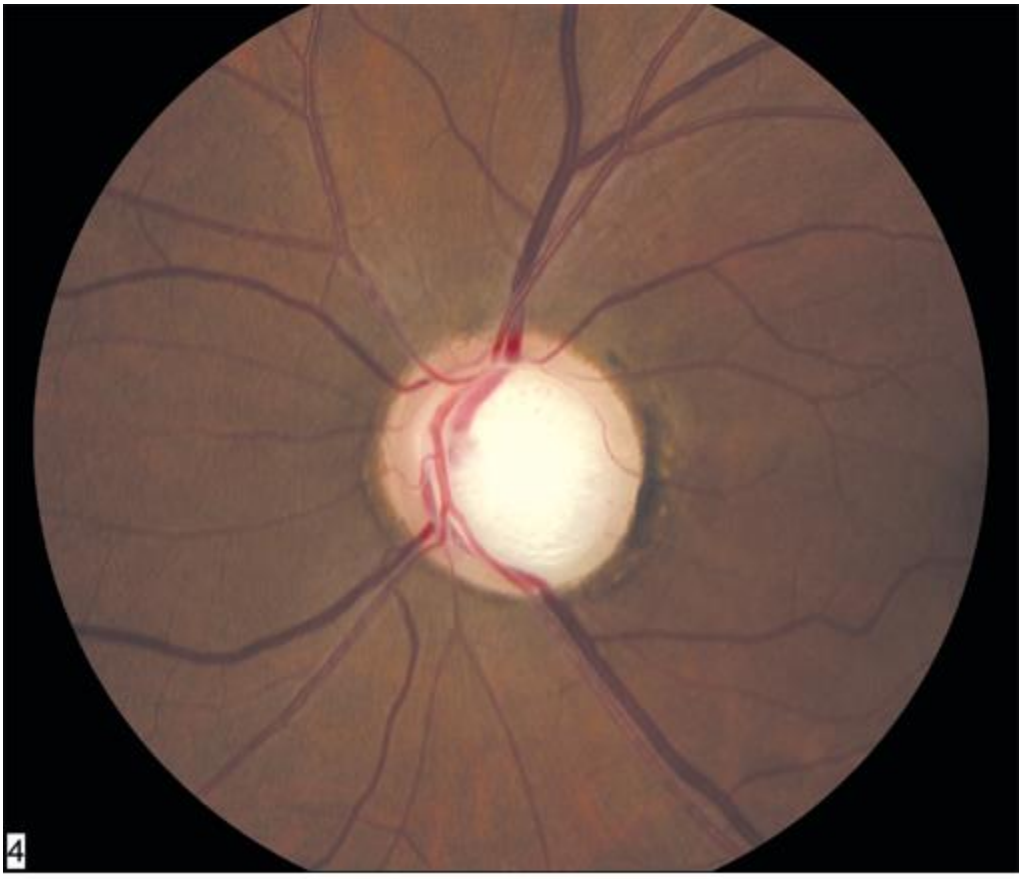
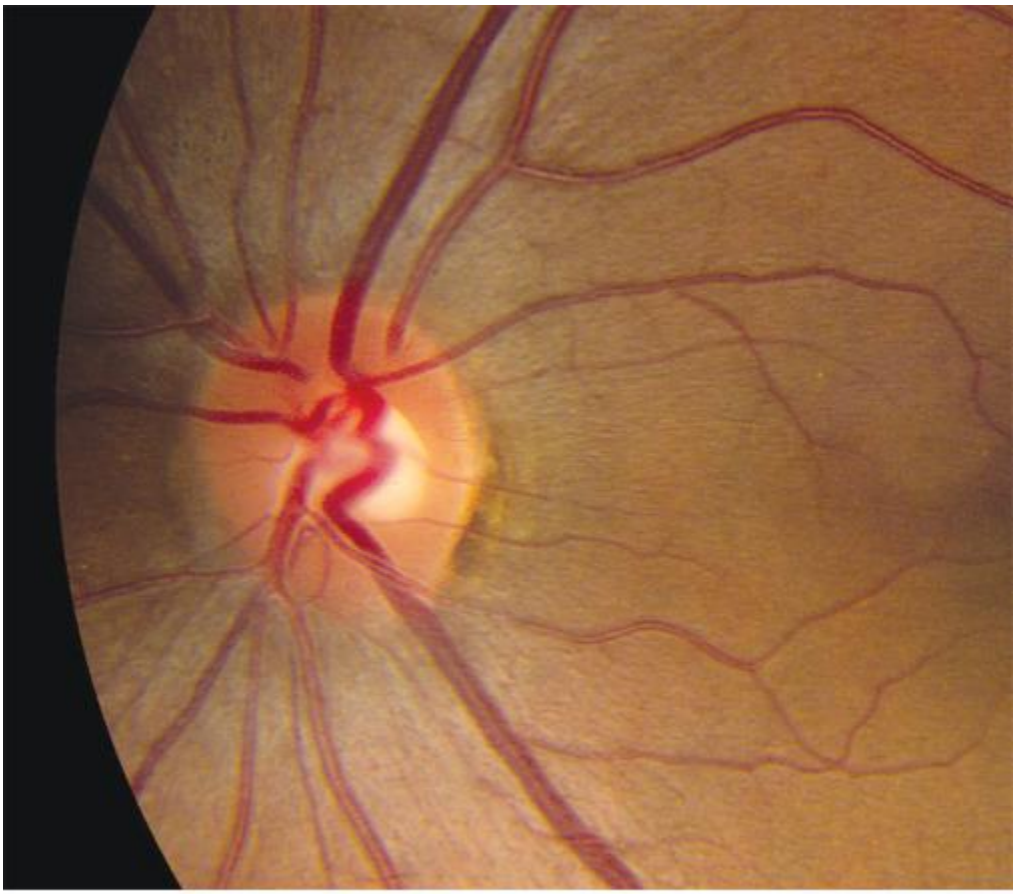


FIGURE 21-2

Normal eye with a normal cup-to-disc ratio of 0.4. A cup-to-disc ratio of more than 0.5 requires further evaluation. (*Reproduced with permission from Paul D. Comeau*.)

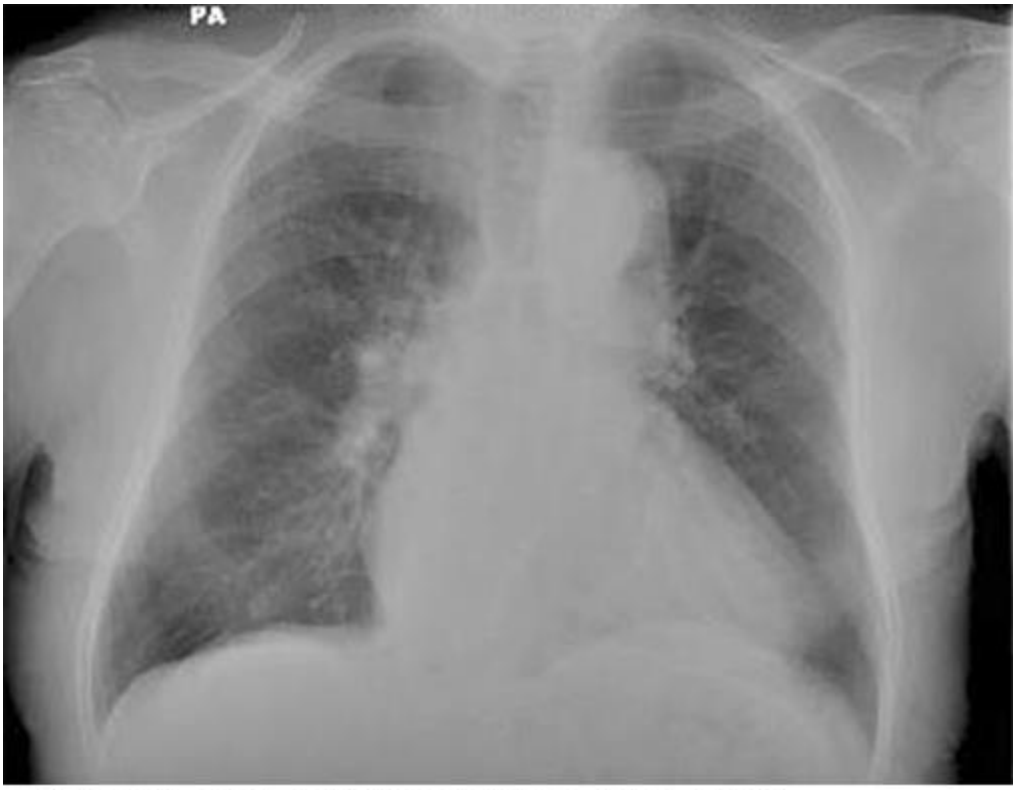


Chapter 50: Heart Failure

A 60-year-old man presents to the emergency department with exertional shortness of breath increasing in severity over the past several days, along with paroxysmal nocturnal dyspnea and orthopnea. He does not have a history of heart failure or previous myocardial infarction. On examination, it was found that he had a third heart sound and an elevated jugular venous pressure. His chest radiograph showed cardiomegaly (**Figure 50-1**) and his B-type natriuretic peptide (BNP) was elevated at 600 pg/mL. He was diagnosed with heart failure, evaluated for underlying causes including coronary artery disease, and treated initially with an angiotensin-converting enzyme inhibitor (ACEI) and a loop diuretic. Later, he will be started on a β-blocker and an aldosterone receptor antagonist.

FIGURE 50-1

Cardiomegaly demonstrated in a posteroanterior (PA) view. The widest part of the heart is greater than 50% of the diameter of the chest. (*Reproduced with permission from Heidi Chumley, MD.*)



INTRODUCTION

Heart failure (HF) is common and increases with age. HF is a clinical syndrome that has multiple etiologies, all of which lead to a decrease in heart pumping capacity. There are two main types of heart failure: heart failure with reduced ejection fraction (HFrEF), also referred to as systolic HF, and heart failure with preserved ejection fraction (HFpEF) or diastolic HF.[1] ACEIs and β-blockers with or without aldosterone antagonists and angiotensin II blockers are the main pharmacologic therapies.

SYNONYMS

Congestive heart failure (CHF), systolic or diastolic dysfunction.

EPIDEMIOLOGY

- The prevalence of HF in the community increases with age: 0.7% (45 to 54 years); 1.3% (55 to 64 years); 1.5% (65 to 74 years); and 8.4% (75 years or older).[2]
- In the United States, the prevalence of HF is more than 5.8 million and the annual incidence is approximately 550,000.[3]
- There are race-related differences in the risk of HF, with the prevalence among black men and women about twice that of whites.[4]
- More than 40% of patients in the community with HF have an ejection fraction greater than 50%.[5]
- At age 40 years, the lifetime risk for HF is 21.0% (95% confidence interval [CI] 18.7% to 23.2%) for men and 20.3% (95% CI 18.2% to 22.5%) for women.[5]
- Survival rate is 50% at 5 years after diagnosis.[1]

ETIOLOGY AND PATHOPHYSIOLOGY

- The most common cause of heart failure is ischemia, but heart pumping capacity can decline from several causes (i.e., myocardial infarction or ischemia, hypertension, valvular dysfunction, cardiomyopathy, or infections such as endocarditis or myocarditis).
- Cardiac dysfunction activates the adrenergic and renin-angiotensin-aldosterone systems.
- These systems provide short-term compensation, but chronic activation leads to myocardial remodeling and eventually worsening of cardiac function.
- Norepinephrine, angiotensin II, aldosterone, and tissue necrosis factor ...

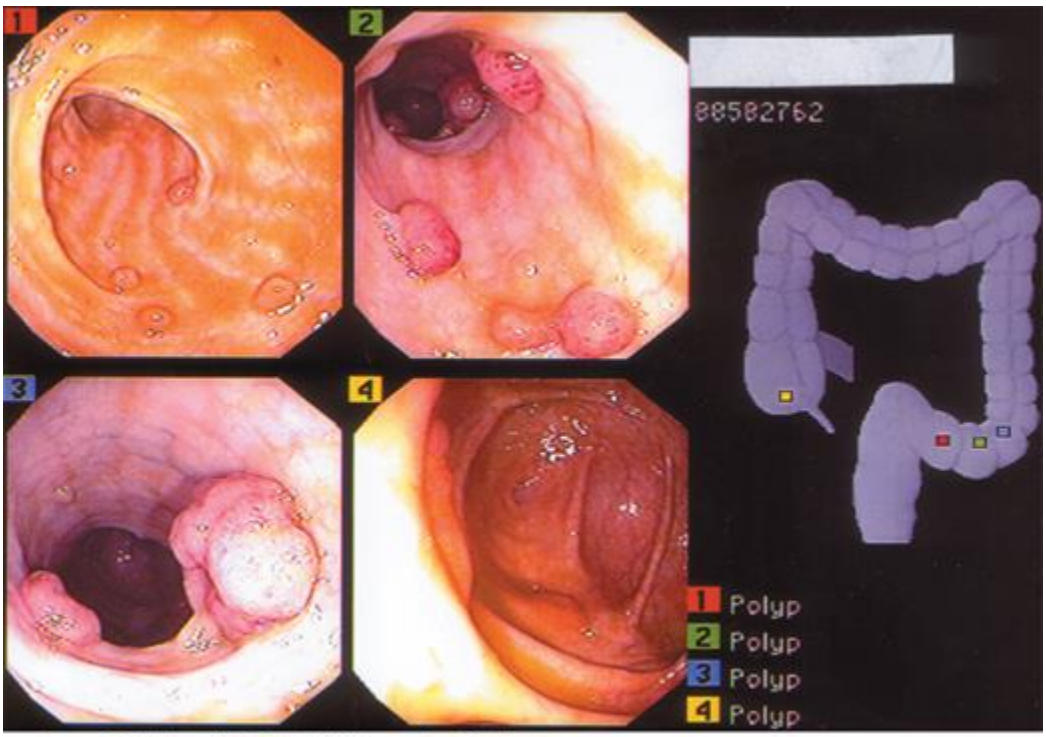
Chapter 65: Colon Polyps

PATIENT STORY

A 62-year-old woman presents to her physician for routine annual examination. She has no known family history of colon disease and is asymptomatic. Stool cards and flexible sigmoidoscopy were recommended and on flexible sigmoidoscopy a 2.4-cm polyp was noted at 35 cm. A colonoscopy was performed and additional polyps were identified in the descending colon and cecum (**Figure 65-1**).

FIGURE 65-1

Colon polyps seen on colonoscopy. (*Reproduced with permission from Michael Harper, MD.*)



INTRODUCTION

Colon polyps are growths that arise from the epithelial cells lining the colon.

EPIDEMIOLOGY

- More than 30% of middle-aged and elderly patients are found to have adenomatous polyps on screening and based on autopsy surveys; fewer than 1% will become malignant.[1] The lifetime risk of colon cancer is 4.4%.[2]

- In the first round of screening in the Bowel Cancer Screening Programme in England, of over 1 million subjects aged 60–69 years, 2.5% of men and 1.5% of women had an abnormal test. About 40% underwent subsequent colonoscopy revealing higher risk adenomas (n = 6543) and cancer (n = 1772) in 43% and 11.6% of men and 29% and 7.8% of women investigated.[3]
- Patients with an adenomatous polyp have a 30% to 50% risk for developing another adenoma and are at higher risk for colon cancer.[1] This risk is greatest in the first 4 years after diagnosis of the first polyp, and greater if a villous adenoma or more than 3 polyps were found.
- Familial adenomatous polyposis of the colon is a rare autosomal dominant disorder associated with a deletion in the long arm of chromosome 5. Thousands of adenomatous polyps appear in the large colon, generally by age 25 years, and colorectal cancer develops in almost all these patients by age 40 years.[1] Other hereditary polyposis syndromes include Gardner syndrome, Turcot syndrome, Peutz-Jeghers syndrome, and MYH-associated polyposis, familial juvenile polyposis.[1]

ETIOLOGY AND PATHOPHYSIOLOGY

- There are several types of colon polyps, including:
    - Hyperplastic polyps—These contain increased numbers of glandular cells with decreased cytoplasmic mucus and an absence of nuclear hyperchromatism, stratification, or atypia. They are thought to be benign, with the exception of those associated with hyperplastic polyposis syndrome (a familial disorder with multiple [>30] hyperplastic polyps proximal to the sigmoid colon with 2 or more >10 mm).[4] The percentage of polyps reported to be in this category ranges from 12% to 90%.[4,5]
    - Adenomatous polyps—These may be tubular, villous (papillary), or tubulovillous; villous polyps are most likely to become malignant.[1] In a case series of 582 patients who had a polyp removed, 81% were adenomatous, including 65.0% that were tubular, 25.8% tubulovillous, 7.2% villous adenomas, and 0.5% mixed adenomatous hyperplastic polyps; 12 (1.4%) were invasive carcinomas.[5]...

Chapter 111: Dermoscopy

INTRODUCTION

The dermatoscope is to the skin as the otoscope is to the ear. Once you have examined the ear with an otoscope, it is hard to imagine examining the ear without one. The same is true for the dermatoscope and the skin. The dermatoscope allows you to see into the skin and make diagnoses with greater accuracy and confidence. The dermatoscope was originally used and studied to better diagnose skin cancers, but its use has been expanded into all aspects of dermatology including the diagnosis of scabies, alopecia, nail disorders, and inflammatory skin diseases.

SYNONYMS

The scope is called either the dermatoscope or dermoscope. The process of using the dermatoscope is interchangeably called dermoscopy or dermatoscopy.

DERMOSCOPY

Dermoscopy is a technique that allows clinicians to evaluate subsurface structures within the skin with an instrument called a dermatoscope (**Figure 111-1**). Most of the light shining onto the skin is scattered at the air-skin interface, resulting in back-scattered light (glare). The dermatoscope is a handheld device consisting of a 10× magnification lens and a transilluminating polarized or non-polarized light source designed to diminish surface glare to see into the deeper layers of the skin.

FIGURE 111-1

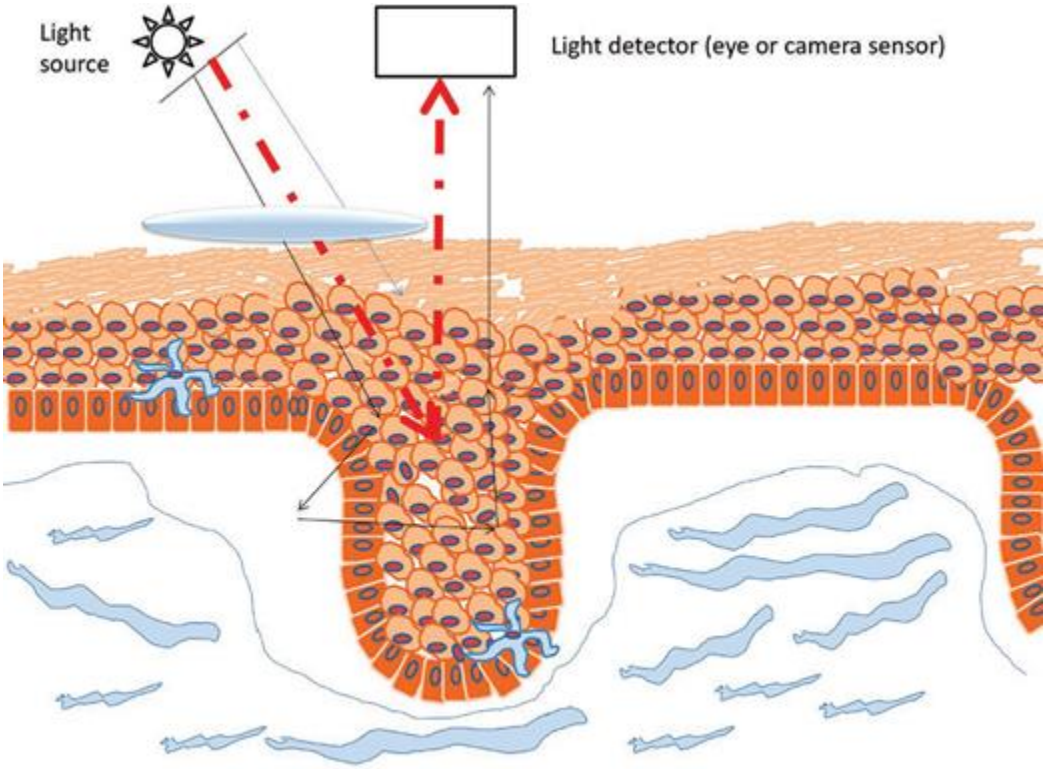
Various dermatoscopes. (*Reproduced with permission from Richard P. Usatine, MD.*)

NON-POLARIZED DERMOSCOPY (NPD)

Non-polarized dermatoscopes (NPDs) eliminate the air-skin interface with the glass faceplate of the dermatoscope and immersion fluid. The following liquids work well: ultrasound gel, mineral oil, and alcohol. Because of the lack of air bubbles and the clarity of the image, 70% alcohol is the preferred immersion liquid. For certain locations such as the nail, ultrasound gel is preferred, as it will not flow off the nail plate surface.[1] NPDs primarily allow for the observation of structures located between the stratum corneum and superficial papillary dermis, with the superficial structures being more conspicuous (**Figure 111-2**).

FIGURE 111-2

Non-polarized dermoscopy. Light is transmitted via the dermatoscope—light source (*blue*, *black*, and *red arrows*). The *red arrow* represents the superficial penetrating light, the main source of contrast using non-polarized dermoscopy, which undergoes minimal scattering events. The *black arrow* represents the deep penetrating light, contributing a small fraction of the back-reflected light due to multiple scattering events. The *blue arrow* represents the surface glare, eliminated by the use of the immersion fluid.



POLARIZED DERMOSCOPY (PD)

Polarized dermatoscopes (PDs) are similar to NPDs with the main difference between the two being that PD uses two polarized filters to achieve cross-polarization, which eliminates the glare off the skin.[2] One advantage of PDs is that they do not require direct contact with ...