Peripheral Retinal Abnormalities

22

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22.1 Introduction

The major peripheral vitreoretinal and/or chorioretinal changes associated with pathologic myopia are lattice degeneration, white-without-pressure, pigmentary degeneration, paving stone degeneration, retinal holes, retinal tears, and retinal detachment. Each of these entities has a distinct morphology and prevalence varying with age and axial length. They are all prone to progression, although the number and extent of the lattice lesions tend to not progress after the teens. The dynamic interaction between the vitreous and the retina plays an important role in the development, appearance, and progression of these peripheral retinal changes. The combination of abnormal vitreoretinal adhesion, traction by posterior vitreous detachment, and liquefied vitreous gel that can enter the subretinal space through retinal breaks is necessary to produce a rhegmatogenous retinal detachment. High myopes have an increased liquid component of the vitreous gel, associated with reduced viscosity and stability [1] and abnormal vitreoretinal adhesion, whether visible, such as lattice degeneration, or invisible, possibly leading to retinal breaks. They also tend to experience vitreous detachment at a younger age compared to emmetropic subjects [2]. Therefore, high myopes have an increased frequency of rhegmatogenous retinal detachments, often at a younger age.

The recognition of the peripheral retinal changes associated with high myopia through careful ophthalmoscopic examination and possibly additional wide-field retinal imaging is critical because lattice degeneration is frequently associated with retinal breaks and rhegmatogenous retinal detachments while white-without-pressure, paving stone, and pigmentary degeneration are usually benign. Some important peripheral findings are seen in syndromic myopia such as Stickler syndrome.

22.2 Lattice Degeneration

Lattice degeneration is acknowledged to be the most important clinically recognizable vitreoretinal abnormality in pathologic myopia [3]. In 1904, Gonin was the first to describe the histologic appearance of an equatorial lesion consistent with lattice degeneration in an enucleated globe from a patient with retinal detachment [4]. Lattice is known to be closely associated with retinal breaks and therefore to be a potential precursor of rhegmatogenous retinal detachment. Even though this entity has been widely described both clinically and histologically, some aspects of the condition remain controversial especially with regard to its management.

22.2.1 Historical Background

A wide variety of names has been given to lattice degeneration. Gonin gave the first description of lattice degeneration in 1920 and introduced the terms *snail-track degeneration (Schneckenspuren)*, *palissades*, and *état-givre* [3]. In 1930, Vogt provided the first complete clinical description of the disease and demonstrated that the white lines represented blood vessels and were not essential to the diagnosis. His mistaken hypothesis was that the entity corresponded to peripheral *cystoid degeneration* of the retina. These dissimilar designations for lattice degeneration may be confusing but reflect the variety of its clinical appearances.

22.2.2 Clinical Features

The shape, location, and orientation of lattice degeneration are characteristic: they typically appear as sharply demarcated

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oval, round, or linear areas oriented circumferentially and parallel to the ora serrata at or anterior to the equator. Many features can be observed separately or in combination. One or many of the following features can predominate in an individual lesion, explaining the striking differences between one lesion and another [3]. These features are localized round, oval, or linear areas of retinal thinning; pigmentation; glistening whitish-yellow surface flecks; round, oval, or linear white patches; round, oval, or linear red craters; small atrophic round holes in approximately 25%; branching white lines corresponding to retinal vessels with thickened or hyalinized walls; yellow atrophic spots; and sometimes tractional retinal tears at the posterior margins of lesions in the context of vitreous separation (Fig. 22.1). The presence of white lines is not a defining feature [3]. A variable amount of pigment may be seen with these lesions, probably due to retinal pigment epithelial proliferation into the retina, but it is not essential to the diagnosis (Fig. 22.1) [3]. Lattice degeneration is associated with liquefaction of the overlying vitreous gel and firm vitreoretinal adhesion along the edges of the lesion. Vitreous traction on these areas during posterior vitreous detachment is often responsible for retinal tears. The size of the lattice degeneration can vary from a small solitary lesion to extensive lesions covering almost the complete circumference of the peripheral retina [3]. Areas of lattice degeneration are usually multiple, with the average number ranging from a low of two at 60 years and above to a high of 4.5 lesions at ages 20–29 [5]. Given that these lesions do not disappear over time, these variations are probably related to sampling variation.

22.2.3 Prevalence

The prevalence of lattice degeneration has been found to vary from 7.1 [5] to 8% [6] in clinical surveys and was 10.7% [7] in a histologic survey in the general population. No statistically significant difference in prevalence between men and women or between left and right eyes has been reported in the literature. Lattice degeneration is not known to show any racial preference either. Cambiaggi [8] reported palissade degeneration in 4.5% of normal eyes and in 19% of myopic eyes with increased prevalence in those over −8.00 diopter (D). The disease appears to reach its maximum prevalence prior to the age of 10 years [5], but this is subject to small sample size and sampling variation. Bansal and Hubbard [9] evaluated 54 eyes of 30 highly myopic children under the age of 10 years and identified peripheral retinal changes in 33% of eyes, the most common being lattice degeneration in 20% of eyes. Karlin and Curtin showed an increased prevalence of lattice degeneration with increased axial length in adult myopia with an overall prevalence of 6.1% in a series of 1437 myopic eyes [10]. Celorio and Pruett found a prevalence of lattice degeneration of 33% among 218 highly myopic patients (436 eyes) and an inverse relationship between axial length and the prevalence of lattice degeneration [11]. More recently, Lai et al. reported that 13.6% of 337 highly myopic adults in China with a mean axial length of 26.84 mm had lattice degeneration [12]. The discrepancies between these different studies in the prevalence of lattice degeneration may be due to the differences in axial length, refractive error, and age of the evaluated populations.

It is often the bilateral (34 [5], 40 [10], 50 [13], 63% [14]) and the temporal quadrants [10, 13, 15, 16] and the vertical axis are predominantly involved [5, 6].

22.2.4 Clinical Variants

22.2.4.1 Snail-Track Degeneration

Linear nonpigmented lesions that have a glistening, frostlike appearance have been termed *snail-tracks* (Fig. 22.1). They usually have borders that are less discrete than typical lattice degeneration, and the interstitial spaces between the dots have a translucent appearance. The presence of tiny glistening whitish-yellow flecks at their surface is essential to the diagnosis (Fig. 22.1). The lesion reported by Gärtner as *Milky Way-like* or *galaxy-like degeneration* falls within the group of snail-track lesions [3]. These lesions lack both the white lines and pigmentation and are not commonly seen with lattice degeneration [17]. They seem to pose a greater risk of retinal detachment [18]. Therefore, there is controversy as to whether they represent a variant of lattice degeneration or a separate entity. However, *snail-track* degeneration is usually clinically classified as lattice degeneration because they closely resemble the characteristic shape and orientation of lattice lesions [3]. They are located very anteriorly, typically behind the ora serrata. Moreover, the white flecks of the snail-track appearance occur to varying degrees in 80% of lattice lesions [5], and the *snail-track* appearance is frequently combined with other classic features of lattice lesions such as round atrophic holes, horseshoe tears, or a reddish base [3]. Overall, a snail-track appearance may actually represent a variant or an early stage of lattice degeneration [19].

22.2.5 Associations with Hereditary Disorders

Lattice degeneration has been observed in various hereditary disorders such as Ehlers-Danlos syndrome [20], Wagner's hereditary vitreoretinal degeneration [21–24], and Turner's syndrome [25].

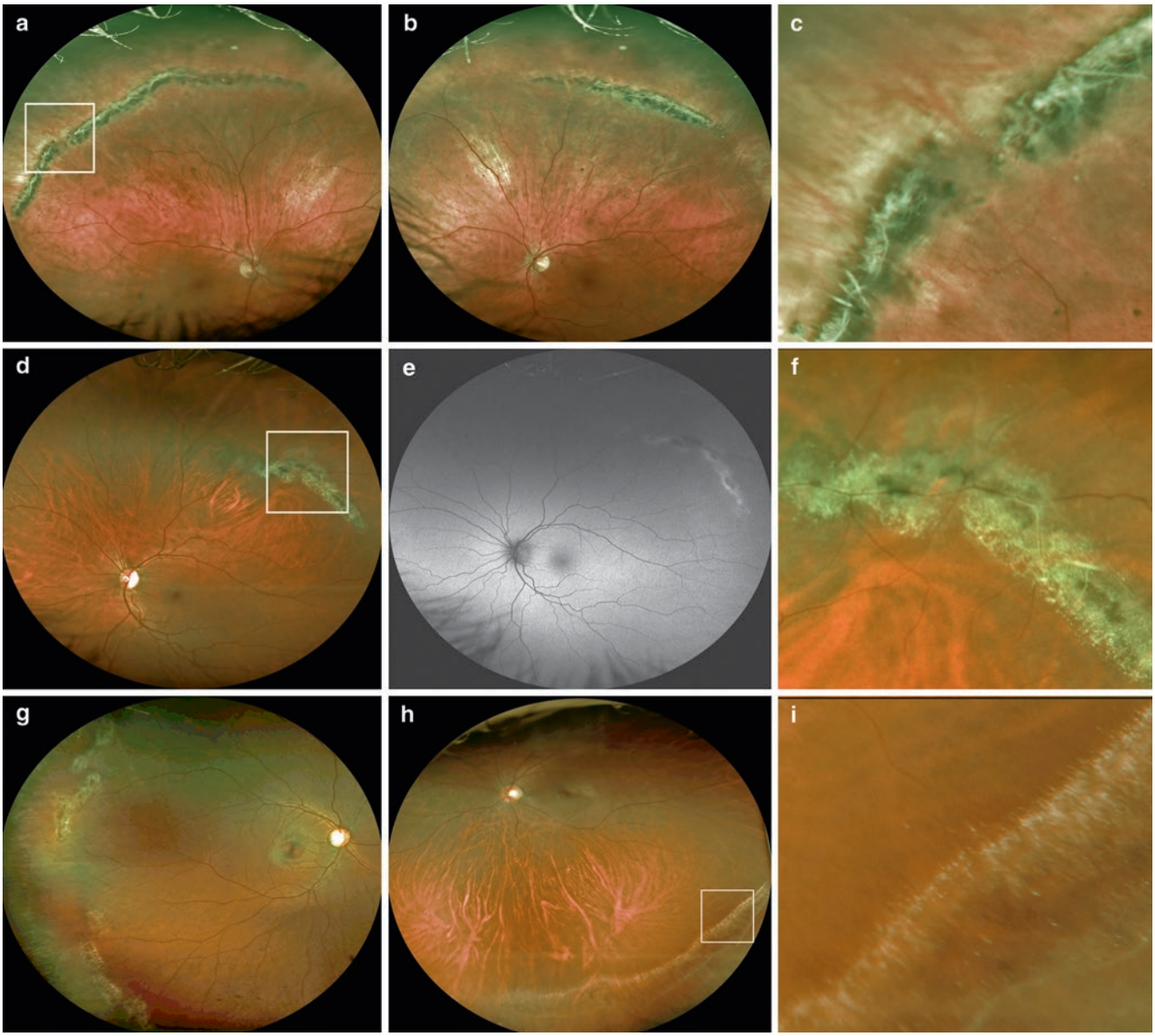

Fig. 22.1 Various clinical appearances of lattice degeneration, all located at the level of or anterior to the equator and oriented circumferentially parallel to the ora serrata, in four highly myopic patients, a 65-year-old male (**a**–**c**), a 29-year-old female (**d**–**f**), a 34-year-old female (**g**), and a 31-year-old female (**h**, **i**), using ultrawide-field imaging. (**a**–**c**) Bilateral lattice degeneration superotemporal with magnification of the area in the *white rectangle* (**c**) showing linear areas of retinal thinning, pigmentation, branching *white lines* corresponding to hyalinized walls of retinal vessels, and areas of whitish atrophy, glistening whitish-yellow surface flecks, and sheathing of the overlying retinal vessels. (**d**–**f**) The lattice degeneration appears hyperautofluorescent on fundus autofluorescence imaging (**e**). Note on magnification of the area in the *white rectangle* (**f**) the glistening whitish-yellow surface flecks, round white patches, moderate pigmentation, and sheathing of the overlying retinal vessels. (**g**) Multiple lattice lesions with white patches, glistening white flecks, and no pigmentation. (**h**, **i**) Lattice degeneration showing a pure snail-track feature with magnification of the area in the white rectangle in (**i**) showing discrete yellow-white glistening flecks on the surface and abrupt borders

22.2.6 Histologic Features

Straatsma et al. [7] evaluated a series of 86 autopsy cases with lattice degeneration. Three constant features were found in all the 286 lesions evaluated: retinal thinning, vitreous liquefaction, and vitreous condensation with exaggerated vitreoretinal adherence at the edges of the lesion. The autopsy reports also mention accumulation of glial proliferation within the lesion. The authors evaluated lattice degeneration with electron microscopy as well, demonstrating retinal thinning, fibrosis of blood vessels, loss of retinal neurons, accumulation of extracellular glial material, and pigment

abnormalities. The retinal thinning and degeneration were more advanced toward the center of the lesions [7]. Electron microscopic analysis revealed focal thinning and intermittent absence of the inner limiting membrane in the center of the lattice degeneration [7]. There was no age-related trend in regard to the size, location, or orientation of lesions, but there was an age-related increase in degree of vitreoretinal attachment, prevalence of pigmentation, white lines, retinal holes, posterior vitreous detachment, and retinal tears. Of interest, it has also been observed histologically that the earliest and most severe degenerative changes occur at the level of the inner retinal layers [7] and that the choroid was most often not involved in lattice lesions [26].

22.2.7 Pathogenesis

The etiology of lattice degeneration is still uncertain. Their bilateral occurrence in patients with unilateral pathologic myopia [27] suggests a hereditary etiology. It is likely that both genetic and environmental factors play important roles in its development. Michaelson had postulated that the primary event in lattice degeneration was at the level of the choroid, leading to a focal loss of perfusion to the outer retina from the choriocapillaris [28], but this hypothesis has not been reinforced by histologic observations which have shown that the choroid was most often not involved in lattice lesions and that the earliest changes were actually at the level of the inner retina. Tolentino et al. hypothesized that lattice degeneration was primarily a vitreous disease with secondary retinal degeneration [29]. A primary retinal vascular etiology leading to retinal ischemia has also been proposed [7].

The pathogenesis may also be due to a developmental abnormality involving primarily the Müller cells resulting in an aplasia or focal defect of the internal limiting membrane [30].

22.2.8 Evolution and Management

Lattice degeneration may not only contain round holes within the confines of the lesion but is also predisposed to developing retinal tears at the posterior margin and ends of the lesion (Fig. 22.2). In one large study, the majority of eyes demonstrating retinal breaks (55%) showed lattice degeneration [31]. These breaks frequently lead to detachment of the retina. Approximately 6–8% of patients in the general population have lattice degeneration [32, 33], and up to 35% of these will have atrophic holes in the lattice [33]. Some of the atrophic holes are associated with subretinal fluid. Any collection of fluid greater than 1 disc diameter is generally considered to be a retinal detachment, although collections of about 1 disc diameter are sometimes called subclinical detachments. Tillery et al. found that 2.8% of all retinal detachments were due to atrophic retinal holes in lattice degeneration [34]. It is therefore not surprising that approximately 20–30% of patients with retinal detachments have lattice degeneration [33]. In a series of 553 consecutive retinal detachments, 29% were due to lattice degeneration. Forty-five percent of these were due to atrophic holes in the lattice degeneration, and 55% were due to tears caused by traction posterior to or at the end of a patch of lattice [32].

Retinal detachment secondary to retinal holes is more commonly seen in young myopes, whereas detachments due to tears tend to occur in older, less myopic patients [32]. The detachments secondary to atrophic holes in lattice degeneration show a more insidious course than those due to traction breaks, which generally occur in the context of a symptomatic posterior vitreous detachment. The formation of demarcation lines is not uncommon in chronic round hole detachments. The risk of detachment in lattice degeneration with round holes was estimated at about 1 in 90 [35], but this risk has to be tempered by the refractive error. Lattice degeneration increases the risk by a factor of 6–7 [36]. Detachment of the retina can therefore be readily appreciated as a frequent sequel to lattice degeneration.

Whether to treat lattice degeneration in adult eyes has previously been a source of controversy. A high proportion of eyes with retinal detachment were found to have lattice lesions: 20 [28], 29 [32], 30 [13, 16], or 38.5% [14], and even 65% [37]. Therefore, treatment of such lesions was generally favored in the 1970s and 1980s. However, eyes with lattice degeneration may experience retinal detachment from tears which are not in an area of lattice in 40% [33]. In a long-term natural history study, Byer showed that in 276 adults (423 eyes) with lattice degeneration followed for an average of 10.8 years, retinal detachment occurred in only 1.08% of patients (0.7% of eyes) [33]. Byer concluded that prophylactic treatment of lattice lesions with or without holes in phakic eyes should be discontinued if the fellow eye had no history of rhegmatogenous retinal detachment [33]. Prophylactic treatment of lattice in fellow eyes of patients with rhegmatogenous retinal detachment has been shown by Folk et al. to reduce the risk of retinal detachment from 5.1 to 1.8% [38]. In a consensus between a panel of vitreoretinal experts based on a review of the literature regarding the prevention of retinal detachment in adults in 2000, there was not sufficient data to support prophylactic treatment of lesions other than symptomatic flap tears [39]. In the same consensus, there was substantial evidence to recommend to "sometimes treat" lattice lesions with or without retinal holes in fellow eyes of patients with a history of retinal detachment in the first eye, to "rarely treat" asymptomatic lattice lesions in aphakic eyes, and not to treat asymptomatic lattice lesions in phakic and myopic eyes [39]. However, treatment of fellow eyes of patients with a history of retinal detachment with

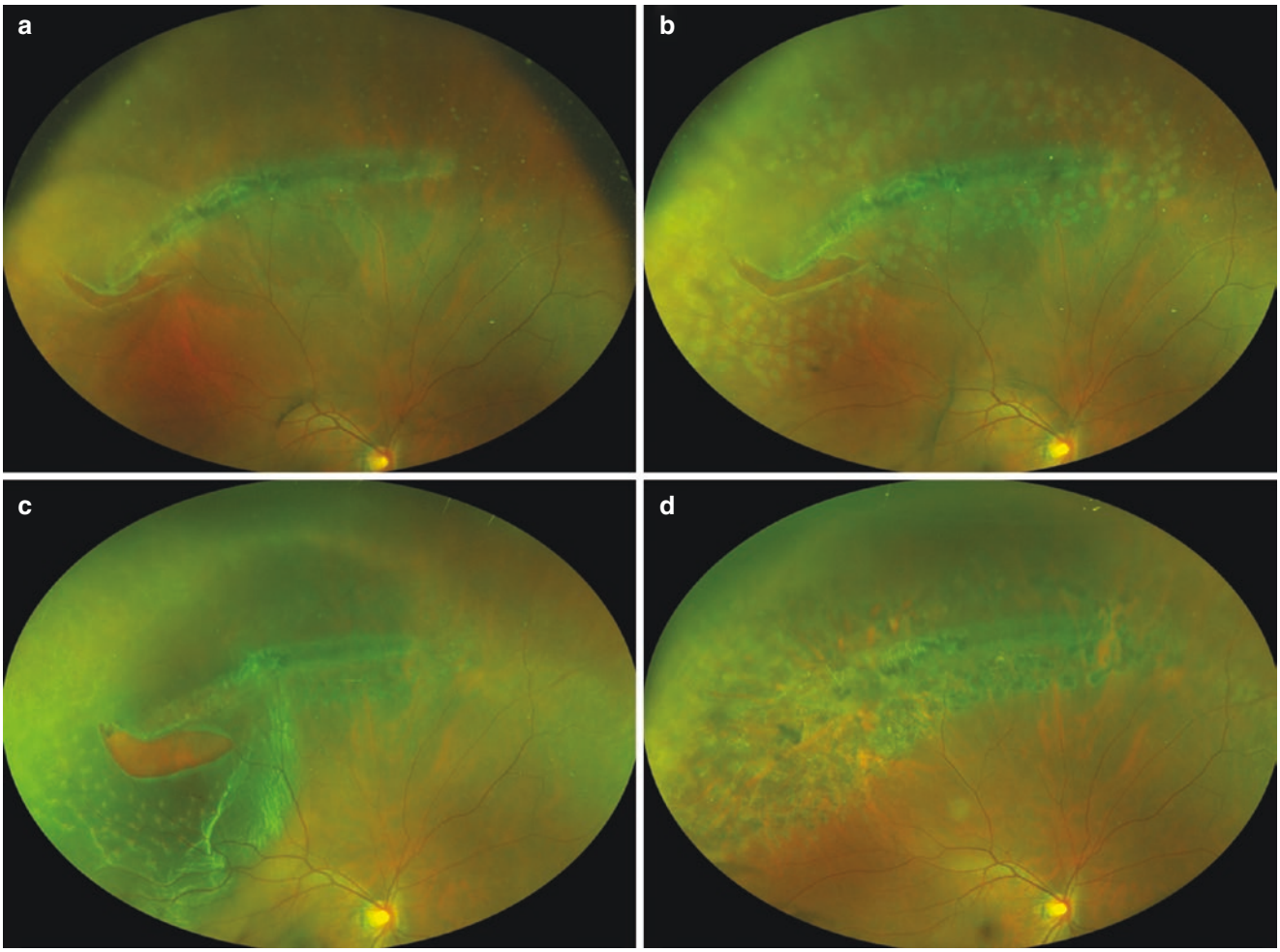

Fig. 22.2 This 42-year-old high myope developed a horseshoe tear at the lateral and posterior edge of a patch of pigmented lattice after vitreous separation (**a**). This tear and the associated lattice were surrounded with retinopexy (**b**), but the subretinal fluid progressed through the laser barrier and involved the macula (**c**). Vitrectomy with removal of subretinal fluid through the preexisting tear and endolaser were performed to reattach the retina (**d**)

lattice degeneration seems to be more beneficial if the eye has less than 6 diopters of myopia, if the lesion is present in less than 6 clock hours, and especially if there is no posterior vitreous separation [38].

22.3 White-Without-Pressure

22.3.1 Clinical Features

Since the first clinical description by Schepens in 1952 [40], the terms white-without-pressure and white-with-pressure refer to opacification of the retina noticed either spontaneously or upon scleral depression. This white to gray opacification partially obscures the normal choroidal vascular color and pattern as if it was visualized through a "semi-translucent veil" [41]. This phenomenon tends to run circumferentially in wide swatches affecting the retina immediately posterior to the ora serrata (Fig. 22.3). The lesions may extend more posteriorly and reach the equator and sometimes even the vascular arcades of the posterior pole [10, 41]. Karlin and Curtin found that the white-without-pressure areas can assume various shapes and locations [10]. White-without-pressure can be diffused with ill-defined margins or may have a very distinct edge with an abrupt transition to a zone of normal choroidal coloration (Fig. 22.3) [41]. It can be flat or have a slightly elevated appearance. They often cover diffusely almost the entire retinal periphery but can also appear as smaller focal patches, with a predilection for the temporal quadrants, particularly the inferior [10]. Similar to lattice degeneration [5], retinoschisis [42], or snowflake degeneration [43], white-without-pressure can also be covered by glistening yellow-white dots and fine lines (Fig. 22.3).

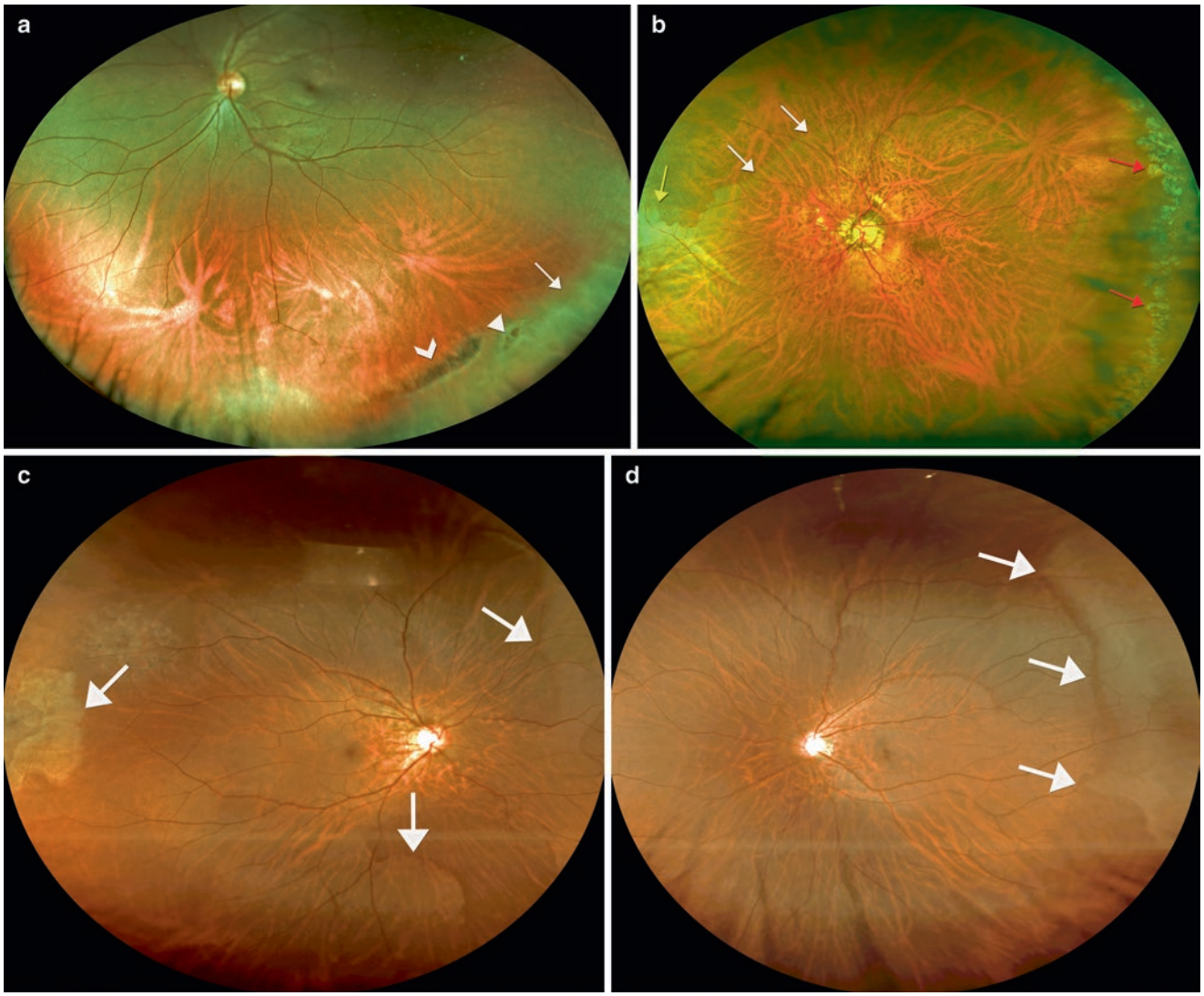

Fig. 22.3 Various shapes and locations of white-without-pressure in three highly myopic males, a 27-year-old (**a**), a 52-year-old (**b**), and a 37-year-old (**c**, **d**), using ultrawide-field multicolor fundus photographs. (**a**) White-without-pressure appears as a focal patch oriented circumferentially, located immediately posterior to the ora serrata in the temporal inferior quadrant (*white arrow*). The lesion is covered by glistening *yellow-white dots* and has relatively ill-defined margins. Note the presence of pigmented lattice degeneration (*arrowhead*) as well as atrophic retinal holes (*white triangles*). (**b**) White-without-pressure appears as a wide area immediately posterior to the ora serrata in the nasal quadrant (*yellow arrow*) and extends more posteriorly to the equator (*white arrows*) with a very distinct edge and an abrupt transition to a zone of normal choroidal coloration. Note the presence of paving stone degeneration in the temporal periphery as well (*red arrows*). (**c**, **d**) Bilateral multiple areas of white-without-pressure appearing as wide swatches oriented circumferentially temporal, inferior, and nasal in the right eye (**c**, *white arrows*) and temporal in the left eye (**d**, *white arrows*)

22.3.2 Prevalence

White-without-pressure has been demonstrated to be significantly more prevalent in young patients [10, 44]. Its prevalence in patients under the age of 20 years is 36% and decreases considerably to 9.5% in patients over the age of 40 [10]. White-without-pressure is also easier to visualize in pigmented individuals [45].

Karlin and Curtin found a tendency for white-without-pressure to affect eyes of individuals 19 years of age or younger and more myopic, with a prevalence reaching 100% in this age group when the axial length was 33 mm or more [10]. Pierro et al. evaluated 513 patients (513 eyes) with an axial length superior to 24 mm and a mean age of 48 years and found a prevalence of white-without-pressure of 22.8%, the second most common peripheral retinal change after paving stone degeneration (27.1%) [44]. They also found that white-without-pressure was significantly more common in younger patients [44]. Bansal and Hubbard evaluated 54 eyes of 30 highly myopic children under the age of 10 years

and identified white-without-pressure in 11% of eyes, the second most common change after lattice degeneration (20%) [9]. Lai et al. found a prevalence of white-without-pressure of 21.1% among 337 highly myopic Chinese subjects with a mean age of 36 years and a mean axial length of 26.84 mm [12]. The most prevalent peripheral retinal change in this population was pigmentary retinal degeneration (37.7%) [12]. Lam et al. evaluated 213 highly myopic Chinese patients (213 eyes) with a mean age of 33.5 years and a mean axial length of 26.69 mm and found a prevalence of white-without-pressure of 31%. The most frequent peripheral retinal change in this population was pigmentary degeneration (51.2%) [46]. This higher prevalence of white-without-pressure in young patients could be explained if the lesion is viewed as an earlier stage of another lesion type [10] or if the lesion can change or fade in time [47].

22.3.3 Pathogenesis and Histology

Since its first description by Schepens in 1952 [40], there have not been many reports about white-without-pressure pathogenesis in the literature. Karlin and Curtin postulated that white-without-pressure may represent advanced retinal cystoid degeneration, flat retinal detachment, or flat retinoschisis [10]. Nagpal and coworkers reported that all their patients had posterior vitreous detachment except in the areas of white-without-pressure, suggesting that white-without-pressure may be due to areas of vitreoretinal traction [47]. White-without-pressure is considered by many to be an advanced form of white-with-pressure. White-with-pressure is commonly found in elderly fundi, almost invariably in areas of lattice degeneration and around small retinal breaks [41]. It is also frequently seen in the attached retina of eyes with partial retinal detachment or in the asymptomatic fellow eye of patients with unilateral rhegmatogenous retinal detachment [41]. However, Fawzi and coworkers demonstrated with multimodal imaging that the fundus color change in white- or dark-without-pressure corresponds to a change in the OCT reflectivity of the outer retina and were unable to demonstrate any associated vitreoretinal interface changes [48].

22.3.4 Evolution and Prognosis

Nagpal and coworkers described the peculiar phenomenon of migratory white-without-pressure areas in nine patients with various hemoglobinopathies [47]. In their series, follow-up examinations revealed a change in configuration of the lesions: some receded while others progressed [47]. The authors also claim that the migratory nature of white-without-pressure was not restricted to hemoglobinopathies. In their series, the white-without-pressure areas were not associated with vascular occlusions or tortuosity or sea fans, nor did fluorescein angiography show any vascular abnormality. The fact that these lesions may change with time is also supported by the dramatic reduction in prevalence between age groups. White-without-pressure is essentially a benign lesion.

22.3.5 Associations and Variants

22.3.5.1 Associations

A number of retinal disorders can give rise to areas of peripheral retinal whitening quite similar to white-without-pressure. Condon and Serjeant in 1972 described peripheral whitening of the retina in 76 patients with sickle cell anemia [49], but this may have been because these patients were pigmented and the prevalence of white-without-pressure in pigmented patients with sickle cell anemia may not be greater than in pigmented patients without sickle cell anemia [48]. These patients had condensation of the overlying vitreous base. Most of the white areas were ill defined, but some were well demarcated and associated with vascular abnormalities. These lesions may have represented white-without-pressure. In 1966, Tasman and coworkers reported that the characteristic appearance of the premature fundus included white-without-pressure [50]. Pars planitis, snowflake degeneration [43], retinoschisis, and flat retinal detachment have also been associated with white-without-pressure.

White-without-pressure is mainly an incidental finding, and its chief importance is that it may be confused with a retinal detachment. Children may need an examination under anesthesia to distinguish between both entities [9, 51].

22.3.5.2 Dark-Without-Pressure

Nagpal and coworkers also described homogeneous, geographical, flat, brown areas surrounded by a halo of pale retina in the fundi of seven Black patients, of whom six had various hemoglobinopathies (Fig. 22.4). These lesions were called "dark-without-pressure fundus lesions" [52]. These lesions were associated with iridescent glistening spots and varied in size, shape, location, and orientation. They could be oriented either radially or circumferentially. Most of these lesions were transient. Fluorescein angiography in these areas did not reveal any vascular abnormality. The authors hypothesized that these dark areas may represent focal and relatively preserved retinal areas surrounded by more diffuse white-without-pressure areas. Like white-without-pressure, these dark lesions can vary in size, shape, and location and can also fade in time. Unlike white-without-pressure, these dark lesions occur usually near the posterior pole or in the midperiphery, and they seem to be unrelated to the status of the vitreous [52]. Indeed, Fawzi and colleagues demonstrated

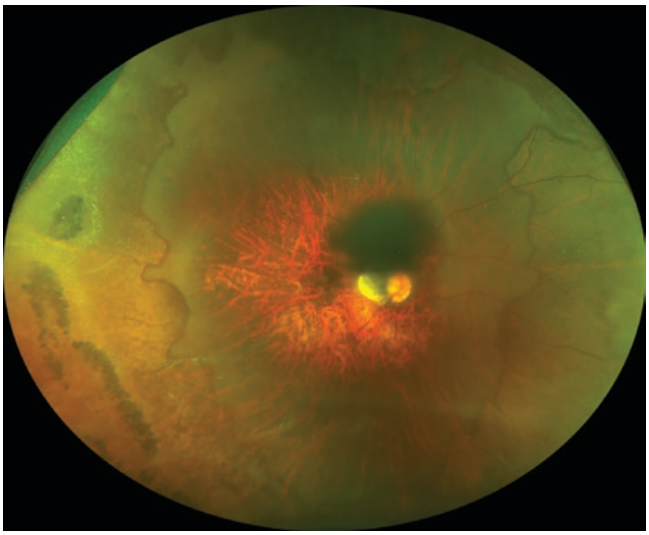

Fig. 22.4 Dark-without-pressure in the right eye of a 25-year-old black female with high myopia and pigmented lattice degeneration. The central dark opacity stems from a posterior subcapsular cataract

the fundus appearance to correlate with changes in outer retinal reflectivity, as in white-without-pressure [48].

22.4 Pigmentary Degeneration

22.4.1 Clinical Features

Pigmentary degeneration corresponds to a variable degree of pigmentation in the extreme periphery. This pigmentation may vary widely from a light dusting of fine particles to large discrete pigment clumps (Fig. 22.5). The temporal quadrants especially the superior are involved preferentially. The posterior margin can extend several disc diameters from the ora serrata and is typically indistinct. The border may be contiguous with a relatively depigmented zone of the fundus. This retinal lesion is typically bilateral.

22.4.2 Prevalence

Pigmentary degeneration affects an increasing proportion of eyes as the axial length and the age increase [10]. No sex preference has been reported in the literature. Karlin found them in only 6% of eyes in young patients and 41% of patients age 40 and above in a series of 1437 predominantly myopic eyes [10]. Two community-based studies reported pigmentary degeneration to be the most frequent peripheral retinal change in highly myopic Chinese patients [12, 46]. Lam et al. found pigmentary degeneration in 51.2% of 213 patients with a mean age of 33.5 years and a mean axial length of 26.69 mm [46], and Lai et al. found it in 37.7% of 337 patients with a mean age of 36 years and a mean axial length of 26.84 mm [12]. In a population of 30 children under the age of 10 years, there was no pigmentary degeneration [9].

22.4.3 Pathogenesis and Evolution

Despite its frequency, this retinal peripheral change is the least studied and therefore least understood. Vascular, inflammatory, and toxic agents may be involved in their development. This higher prevalence of pigmentary degeneration in older patients could be explained if the lesion is viewed as a later stage of another lesion type. For example, it is plausible that lattice degeneration may become increasingly pigmented over time and become indistinguishable from pigmentary degeneration. Pigmentary degeneration of the retina can be associated with both retinal holes and tears [10]. Everett evaluated fellow eyes of patients with retinal detachment and found that 32% of patients with pigmentary degeneration had retinal breaks [53]. Pigmentary degeneration appears to be essentially a benign lesion, and its morbidity however may be attributed to unidentified underlying areas of lattice degeneration prone to formation of retinal breaks.

22.4.4 Differential Diagnosis

Areas of pigmentary changes may appear in the periphery of myopic eyes after a spontaneously flattening retinal detachment. These regions can adopt a variety of appearances to include nummular, bone-spicule, or a densely packed granular hyperpigmentation. Bilateral and extensive areas of pigmentary degeneration in a myopic eye could be leading to an erroneous diagnosis of retinal dystrophy with bone-spicule formation, such as retinitis pigmentosa.

22.5 Paving Stone Degeneration (Cobblestone Degeneration)

22.5.1 Clinical Features

Paving stone degeneration is a distinctive and fairly common disease process found in the evaluation of the peripheral fundus. During the past century, a number of appellations have been applied to this lesion: some of them presume of an etiology such as chorioretinal atrophy [54] or equatorial choroiditis [55], and some are purely descriptive such as punched-out chorioretinal degeneration [53], paving stone degeneration, or cobblestone degeneration [37]. Paving stone degeneration can be used interchangeably with cobblestone degeneration. Paving stone degeneration is the preferred

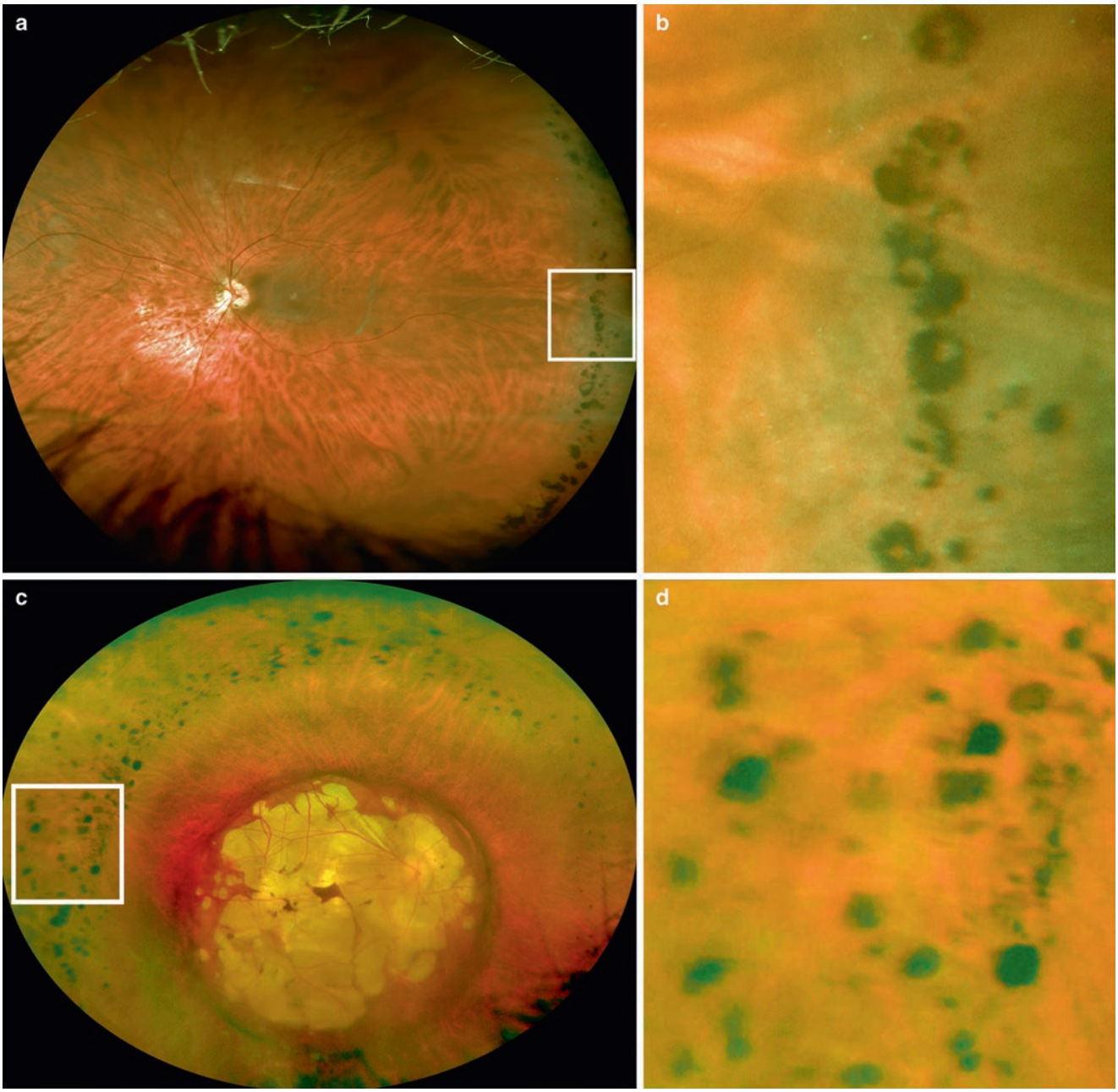

Fig. 22.5 Ultrawide-field fundus multicolor photographs of pigmentary degeneration in two highly myopic patients. (**a**) Pigmentary degeneration in the temporal periphery of a 52-year-old male, with magnification in (**b**) Note the presence of glistening *yellow-white dots* surrounding the pigmentary degeneration. (**c**) Pigmentary degeneration in 360° periphery of a 34 D myopic male, with magnification in (**d**). Note that the areas of pigmentary degeneration are surrounded by relatively depigmented zones of the fundus. (*Bottom image* courtesy of Jerome Sherman)

designation in the United States because cobblestone may suggest the connotation of elevation although these lesions are typically flat or depressed [37]. First described in 1855 by Donders [56], they usually appear as small, sharply demarcated, flat or slightly depressed, rounded, flat yellow to whitish areas of depigmentation and retinal thinning, with subsequent increased visualization of the relatively preserved underlying major choroidal vessels (Fig. 22.6). They often possess pigmented margins. They are usually located one or two disc diameters posterior to the ora serrata and are separated from the ora serrata by a band of intact retinal pigment epithelium. The basic unit can vary in size from 0.1 to 1.5 mm in diameter [55]. They can occur singly or in groups. When in group, they have a tendency to coalesce and form

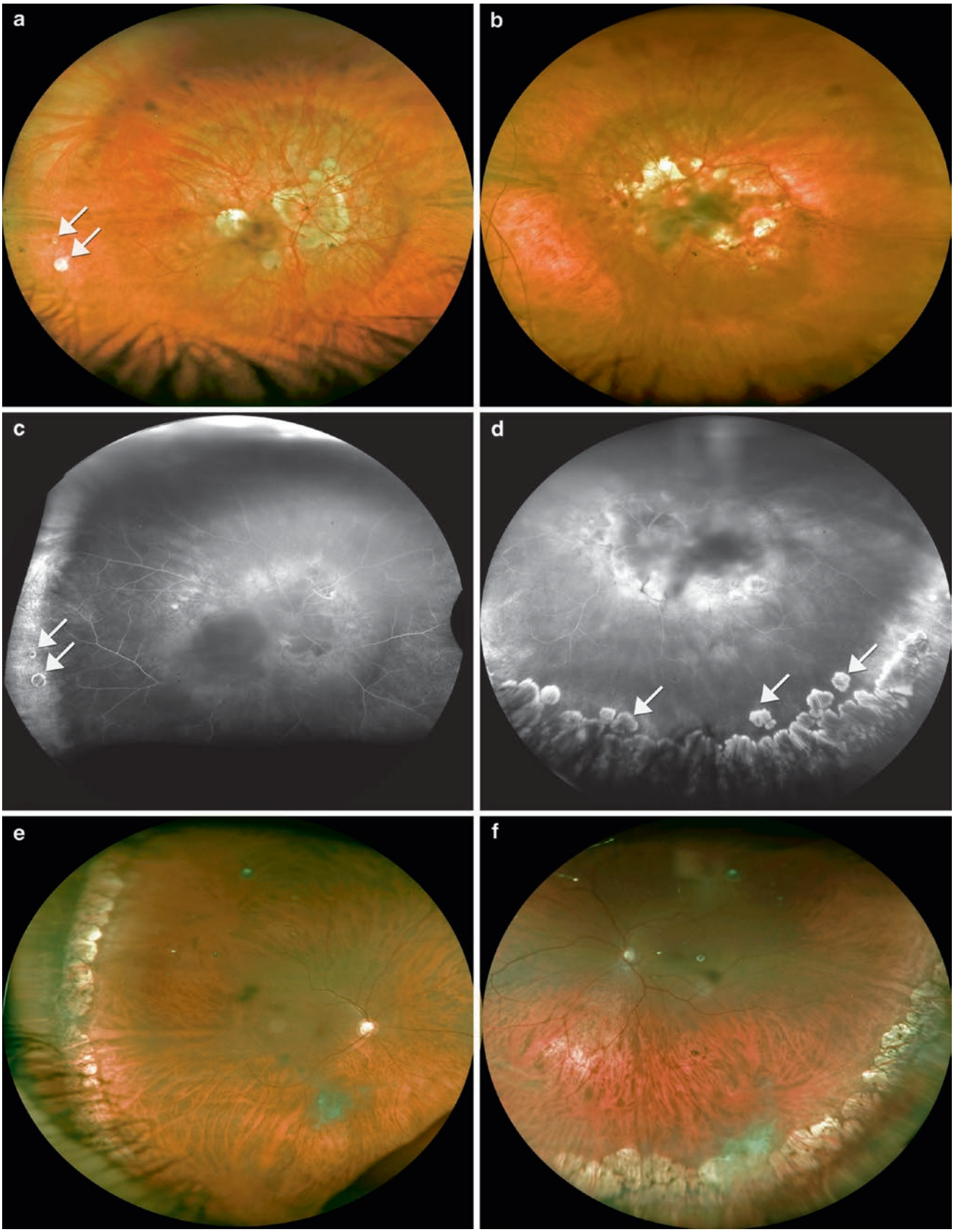
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Fig. 22.6 Ultrawide-field fundus multicolor photographs and fluorescein angiography (late phase) of bilateral paving stone degeneration in a 63-year-old highly myopic female (**a**–**d**) and a 58-year-old highly myopic male (**e**, **f**). (**a**, **c**) In the right eye, the paving stone degeneration appears as two small, sharply demarcated, flat, rounded, whitish areas of depigmentation with subsequent increased visualization of the relatively preserved underlying major choroidal vessels (*white arrows*), located in the temporal periphery approximately two disc diameters posterior to the ora serrata. The lesions appear hyperfluorescent by window defect with increased visualization of the major choroidal vessels through a focal defect of the retinal pigment epithelium. (**b**, **d**) In the left eye, the paving stone degeneration appears as multiple coalescent lesions in the inferior periphery visualized as hyperfluorescent lesions in the fluorescein angiography by window defect as well (*white arrows*). (**e**, **f**) In both eyes, the paving stone degeneration appears as multiple coalescent lesions in the temporal (*right eye*) and inferior (*left eye*) periphery, at the level of and anterior to the equator

bands with scalloped borders (Fig. 22.6). The inferior and temporal retinal quadrants are affected most frequently [10, 55], and the lesion has been reported to be bilateral in 38 [57] to 57% [10].

22.5.2 Prevalence

The prevalence of paving stone degeneration has a clear significant association with increasing age and axial length [10]. In young subjects, Karlin and Curtin found a prevalence of less than 1%, whereas 40% of patients over the age of 40 years were affected [10]. Pierro et al. evaluated 513 patients (513 eyes) with an axial length superior to 24 mm and a mean age of 48 years and found that the most common peripheral degenerative change was paving stone degeneration with a frequency of 27.1% [44]. Lam et al. evaluated a younger population of 213 highly myopic Chinese patients (213 eyes) with a mean age of 33.5 years and a mean axial length of 26.69 mm and found a prevalence of cobblestone degeneration of only 5.2% [46]. Bansal and Hubbard evaluated 54 eyes of 30 highly myopic children under the age of 10 years and did not identify any cobblestone degeneration [9]. At autopsy, 27% of eyes over 20 years demonstrate these changes, and clinically, they are seen in 30% of the general population over the age of 60 years [55]. There does not appear to be a strong gender predilection for these lesions [55] although one report found males to be three times more likely to be affected [54].

22.5.3 Histologic Features and Pathogenesis

This entity is not fully understood. O'Malley et al. evaluated 1223 consecutive eyes obtained from 614 autopsies and found paving stone lesions in 186 eyes from 134 patients. The histologic features of all the lesions evaluated were remarkably similar showing a thinned retina closely applied to Bruch's membrane at an area devoid of RPE [55]. The RPE ended abruptly at the margins of the lesion and appeared normal in the surrounding areas. Hyperpigmented margins corresponded to proliferated RPE. The degree of retinal thinning was poorly correlated to the size of the lesions. The retinal thinning was mainly due to the loss of the rods and cones and outer limiting membrane. The vitreous body was unchanged in the presence of the lesions, and when detached posteriorly, it showed no tendency to remain adherent to the retina at the site of the lesions. At the level of the choroid, the choriocapillaris was the only structure showing significant changes, with thinning, and even occasionally completely absent. The color of paving stone is generally very white due to the absence of underlying choriocapillaris. Brown and Shields showed that paving stone degeneration frequently developed peripheral to choroidal melanomas [58]. They theorized that the melanoma caused a steal syndrome and the paving stone developed secondary to decreased peripheral choroidal blood flow [58].

The pathogenesis of paving stone degeneration is unknown, but O'Malley postulated the likelihood of a vascular etiology due to the topography of the lesions limited to the portions of retina supplied by the choriocapillaris, the histologic appearance of the choriocapillaris altered beneath the lesions, and the absence of gliosis, fibrosis, or inflammatory infiltrate [55]. The anatomy of the choriocapillaris appears consistent with the size and shape of the basic lesion. The mechanical stretching of the highly myopic globe may generate vascular compromise and the development of choroidal ischemic atrophy of the RPE and overlying retina.

22.5.4 Evolution

Paving stone is not significantly associated with retinal breaks. Considering its prevalence and histologic features, a prophylactic treatment of this relatively benign process is not warranted. In fact, Meyer-Schwickerath suggested that treatment applied to these areas may even be detrimental, producing shrinkage of the retina and possibly a retinal break (quoted in [55]).

22.6 Retinal Breaks

What is called a full-thickness break in retinal continuity can be either a retinal hole or retinal tear. These two types of retinal breaks are labeled differently because they have different

morphological characteristics, pathogenic mechanism, and distinct risks for retinal detachment. Retinal holes are related to chorioretinal degenerative changes. Retinal tears result from the traction of adherent vitreous on a weakened retinal area or from zonuloretinal tractions.

22.6.1 Prevalence

The prevalence of retinal breaks has varied among histopathological and clinical studies. These variations may be due to sampling variations since the prevalence of retinal breaks increases with age [59] and axial length [10, 46]. Histopathological studies have reported a prevalence of retinal breaks between 4.8% of cases (2.4% of eyes) [60] and 18.3% of cases (10.6% of eyes) [61]. Byer performed a clinical study on 1700 patients presenting for a complete eye examination and found that 5.8% of cases had one or more retinal breaks, in whom only two patients had symptoms: light flashes or floaters [31]. Lai et al. reported that 6.2% of 337 highly myopic Chinese adults (337 eyes) with a mean axial length of 26.84 mm and mean age of 36 years had retinal breaks [12]. Lam et al. evaluated 213 highly myopic Chinese patients (213 eyes) with a mean age of 33.5 years and a mean axial length of 26.69 mm and found a prevalence of retinal breaks of 7.5%. In this study, the prevalence of retinal breaks was 6.4% in eyes with an axial length inferior to 30 mm and increased to 30% in eyes with an axial length superior to 30 mm [46]. Pierro et al. evaluated 513 patients (513 eyes) with an axial length superior to 24 mm and a mean age of 48 years and found a prevalence of retinal breaks of 12.1% [44]. Bansal and Hubbard evaluated 54 eyes of 30 highly myopic children under the age of 10 years, with a mean age of 6 years and a mean refractive error of −13.88 D, and identified retinal holes in two eyes (3.7% of eyes) and a vitreoretinal tuft in one eye (1.9% of eyes) [9].

22.7 Tractional Retinal Tears

22.7.1 Clinical Features and Classification

Retinal tears can be flap (or arrowhead or horseshoe) tears (64% of all tears) [62] (Figs. 22.7 and 22.8) or operculated tears. They occur suddenly and can be symptomatic but are most commonly asymptomatic [31, 63]. The chief complaints are usually light flashes, floaters, or rarely blurred vision in the visual field corresponding to the quadrant of the retinal tear. The size can vary considerably from less than a quarter of a disc diameter to a giant tear if extending for one or more retinal quadrants. Byer found that 76% of 156 retinal breaks found in 1700 patients evaluated were less than a quarter of a disc diameter in size [31]. Retinal tears have been found to occur preferentially in the upper half and temporal half of the retina in both myopes and nonmyopes [64].

Foos proposed a classification of retinal tears in autopsy eyes based on their relationship to the vitreous base and pathogenic mechanism [62]. This anatomical classification helps to determine the clinical prognosis of retinal tears. The four categories are oral, at the level of the ora serrata; intrabasal, located within the vitreous base; juxtabasal, at the posterior border of the vitreous base; and extrabasal, in the equatorial zone of the peripheral retina posterior to the vitreous base. Retinal tears were postoral in 92% of eyes in Foos' analysis [62]. An oral tear is due to traction of the vitreous base primarily in the posterior direction and is usually associated with trauma and developmental abnormalities. Therefore, they are more common in younger subjects, with a peak of occurrence at the age of 20 years [65]. Oral tears do not have an anterior flap but rather rolling of the posterior flap and are traditionally called dialysis.

Intrabasal tears are due to zonuloretinal traction, resulting from the avulsion of a zonular traction tuft. These tears have been found to represent only 6.1% of all retinal tears in an autopsy study [66]. Most of them are operculated tears [62]. They carry a good prognosis and rarely lead to retinal detachment because the retina surrounding them is not under traction from the vitreous base.

Juxtabasal tears are typically flap tears. They result from an acute change in the vitreous body conformation, typically after a posterior vitreous detachment or a cataract surgery. The traction is exerted from the posterior border of the vitreous base on the anterior margins of the flap tear. The posterior edge of the tear is free of any traction. These tears carry the highest risk of retinal detachment among all postoral retinal tears, and this risk is highest acutely after a posterior vitreous detachment or a cataract surgery and then decreases [65]. Extrabasal tears are usually operculated and are considered relatively benign because they are free of traction at the margins of the tear.

Lattice lesion can lead to a tractional retinal tear at the time of posterior vitreous detachment depending on its precise location toward the vitreous base: intrabasal and extrabasal lesions are less likely than juxtabasal lesions to produce a flap tear [62, 66]. Foos found that 17% of eyes with flap tears had lattice degeneration in an autopsy series of 4812 eyes, but only 20% of these lattice lesions were actually adjacent to the flap tears, suggesting that in eyes with lattice degeneration, there are more widespread vitreoretinal traction and retinal weakness than just limited to the areas of visible lattice [66].

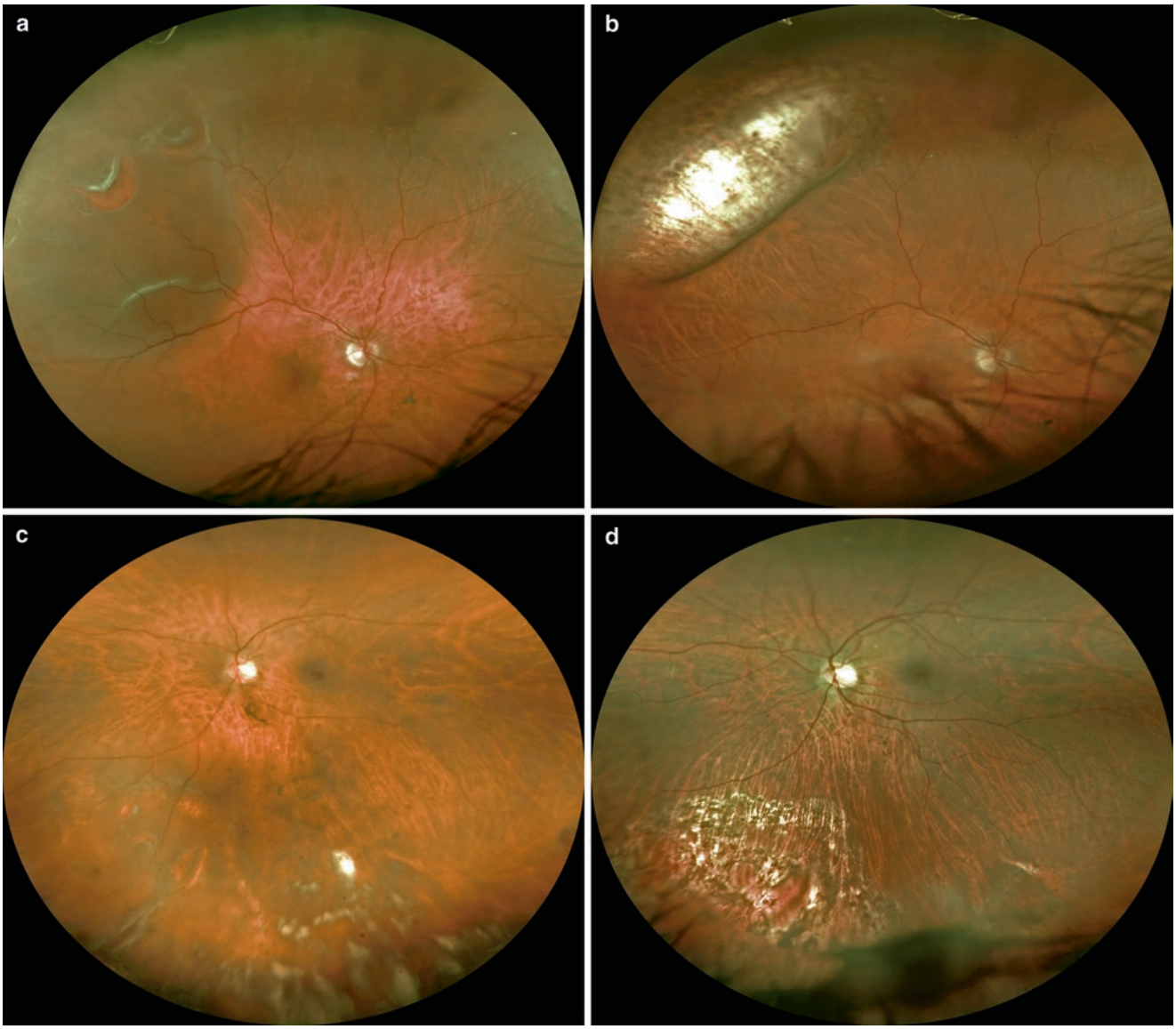

Fig. 22.7 Various clinical features of horseshoe tears in two highly myopic patients, a 58-year-old male (**a**, **b**) and a 41-year-old female (**c**, **d**), using ultrawide-field fundus multicolor photographs. (**a**, **b**) Two horseshoe tears with superotemporal retinal detachment. One month after a radial scleral buckle and cryotherapy, the retina is flat (**b**). (**c**, **d**) Three horseshoe tears inferonasal with retinal detachment despite prophylactic laser therapy (**c**). One month after vitrectomy, gas, and endolaser, the retina is flat (**d**)

22.7.2 Giant Tear

Giant retinal tears are tears of one or more retinal quadrant in the presence of a posterior vitreous detachment [67, 68]. It is a rare condition representing approximately 0.5% of all retinal detachments [69]. It is associated with a bad prognosis due to complicated retinal detachment, a high incidence of proliferative vitreoretinopathy, and therefore a high recurrence rate [70]. The identified predisposing factors are high myopia [70], trauma [71], hereditary vitreoretinopathies such as Stickler syndrome [72], and intraocular surgical procedures [73]. The fellow eyes of patients with giant retinal tears, especially nontraumatic, are at higher risk of developing a giant retinal tear (11.3%) and retinal detachment of any cause (up to 36%) [69, 74]. Therefore, it is recommended to perform 360° laser prophylactic therapy in the fellow eye, even though there is no prospective or case-control study demonstrating the benefit of this procedure [70].

22.7.3 Iatrogenic Retinal Tears

Iatrogenic retinal tears can be induced during pars plana vitrectomy and represent one of the most serious complications

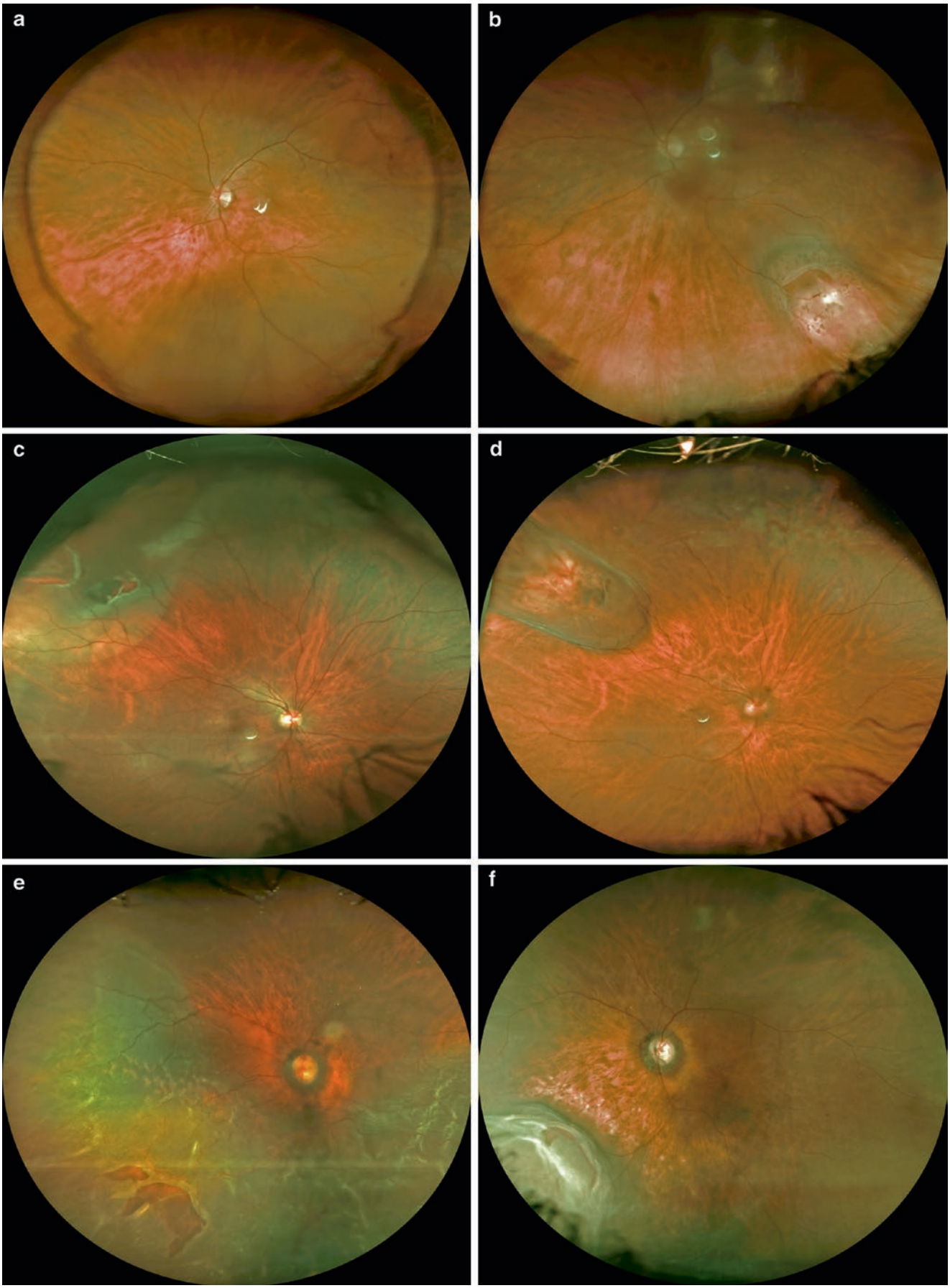
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Fig. 22.8 Three examples of myopic patients with retinal detachment caused by horseshoes tears treated with radial scleral buckles, a 57-year-old male (**a**, **b**), a 55-year-old male (**c**, **d**), and a 63-year-old monocular male (**e**, **f**), illustrated with ultrawide-field fundus multicolor photographs. (**a**, **b**) Inferior retinal detachment caused by a single large horseshoe tear inferotemporal of approximately 2 disc diameters (**a**) and treated successfully with a radial scleral buckle and subsequent thermal laser the day after the surgical procedure, when the retina was reattached (**b**). The patient is pseudophakic and the edges of the implant are visualized in the color photograph (**a**). (**c**, **d**) Superonasal retinal detachment caused by two horseshoe tears radially aligned (**c**) and treated successfully with a radial scleral buckle and cryotherapy (**d**). (**e**, **f**) Inferior macula-off detachment caused by two large horseshoe tears at the level of the equator (**e**) and treated successfully with a large radial scleral buckle and cryotherapy (**f**) without drainage

of elective vitreoretinal surgery. These iatrogenic tears are more common when there is surgical induction of the posterior vitreous detachment [75] and in phakic eyes [76, 77]. There were significantly fewer entry-site breaks associated with transconjunctival 23-gauge vitrectomy compared with 20-gauge conventional vitrectomy [76, 78]. It is possible that the trocars' extension into the eye past the vitreous base enables instruments to pass repeatedly into the eye without engaging vitreous gel and leads to less vitreous traction and fewer iatrogenic breaks [76].

22.8 Atrophic Retinal Holes

Atrophic retinal holes are typically tiny and located near the ends of lattice lesions (Fig. 22.9). They usually occur early in life independent of a detaching posterior vitreous and do not produce symptoms [3]. Foos found that 75% of all round atrophic holes are within lattice lesions in 5600 autopsy eyes [79]. They are usually located at the level of or anterior to the equator. They typically favor the inferior retina but have been found to be more common superiorly in myopes [64].

These atrophic retinal holes may favor the equator due to the vascular anatomy of this region. The equator is a kind of watershed zone where the deep retinal capillary plexus disappears. The uveal circulation may be unable to compensate the blood flow in this region under certain circumstances such as age-related or myopia-related choroidal vascular atrophy [64]. The vascular hypothesis is reinforced by fluorescein angiographic observations that there was no perfusion of the choroid and retina in areas of retinal holes and the retina surrounding them [80]. Since 75% of round holes are in lattice lesions and lattice has been demonstrated to have genetic susceptibility [81], the vascular atrophic hypothesis may not be the only pathogenic mechanism involved though.

Retinal holes are more common than tractional retinal tears but lead to retinal detachment less frequently [3]. Tulloh evaluated 422 patients with primary retinal detachment and found 516 round retinal holes and 222 flap tears (ratio 2.3:1) [64]. Of interest, 65% of patients who develop new retinal holes are under 35 [6], and therefore, there is a decrease in retinal detachments due to round holes with increasing age [3]. Tillery and Lucier found that 2.8% of all primary retinal detachments were due to round holes of lattice lesions [34]. Retinal detachments caused by retinal holes occurred in younger and myopic patients (half under age 30, 75% exceeding −3.00 D) and were inferior, with slow progression and good prognosis [34].

22.9 Risk of Retinal Detachment and Prophylactic Therapy of Retinal Breaks

Identifying the retinal breaks at higher risk of progression to retinal detachment is essential because the prophylactic treatment of these is relatively safe and reduces this risk of retinal detachment significantly. Davis evaluated 213 patients (222 eyes) with one or more retinal breaks without clinical retinal detachment (defined as greater than two disc diameters) and found symptomatic breaks in 39 eyes and asymptomatic breaks in 183 eyes [63]. All the 39 symptomatic breaks were tears (33 flap tears and 6 operculated tears), and 101 out of 183 asymptomatic breaks were tears (71 flap tears and 30 operculated tears) [63]. Davis found that 9 out of 25 patients (36%) with fresh symptomatic flap tears developed a clinical retinal detachment within 6 weeks if left untreated [63]. Colyear and Pischel had found that 11 out of 20 patients (55%) with symptomatic flap tears developed a retinal detachment [82]. Symptomatic flap tears are at high risk of retinal detachment and are the only lesions with a strong evidence-based recommendation for systematic prophylactic therapy [39].

Fresh symptomatic operculated tears are very rare and have been shown to lead to retinal detachment only in one out of six cases in Davis' study [63]. They are usually considered relatively benign, but if the vitreous is adherent to the edge of the operculated tear, it can be considered an equivalent of a flap tear and should therefore be treated [83].

The progression to retinal detachment in phakic eyes with asymptomatic retinal breaks has been found to be very low in multiple studies, whether it is flap tears with less than 10% [63, 84, 85] or retinal holes with less than 5% [6, 63]. The consensus is usually not to treat asymptomatic breaks, except for dialysis or in aphakic, myopic, and fellow eyes of patients who had a retinal detachment [39]. The consensus is to always treat dialysis, whether they are symptomatic or not, with three rows of laser if feasible or cryotherapy [39].

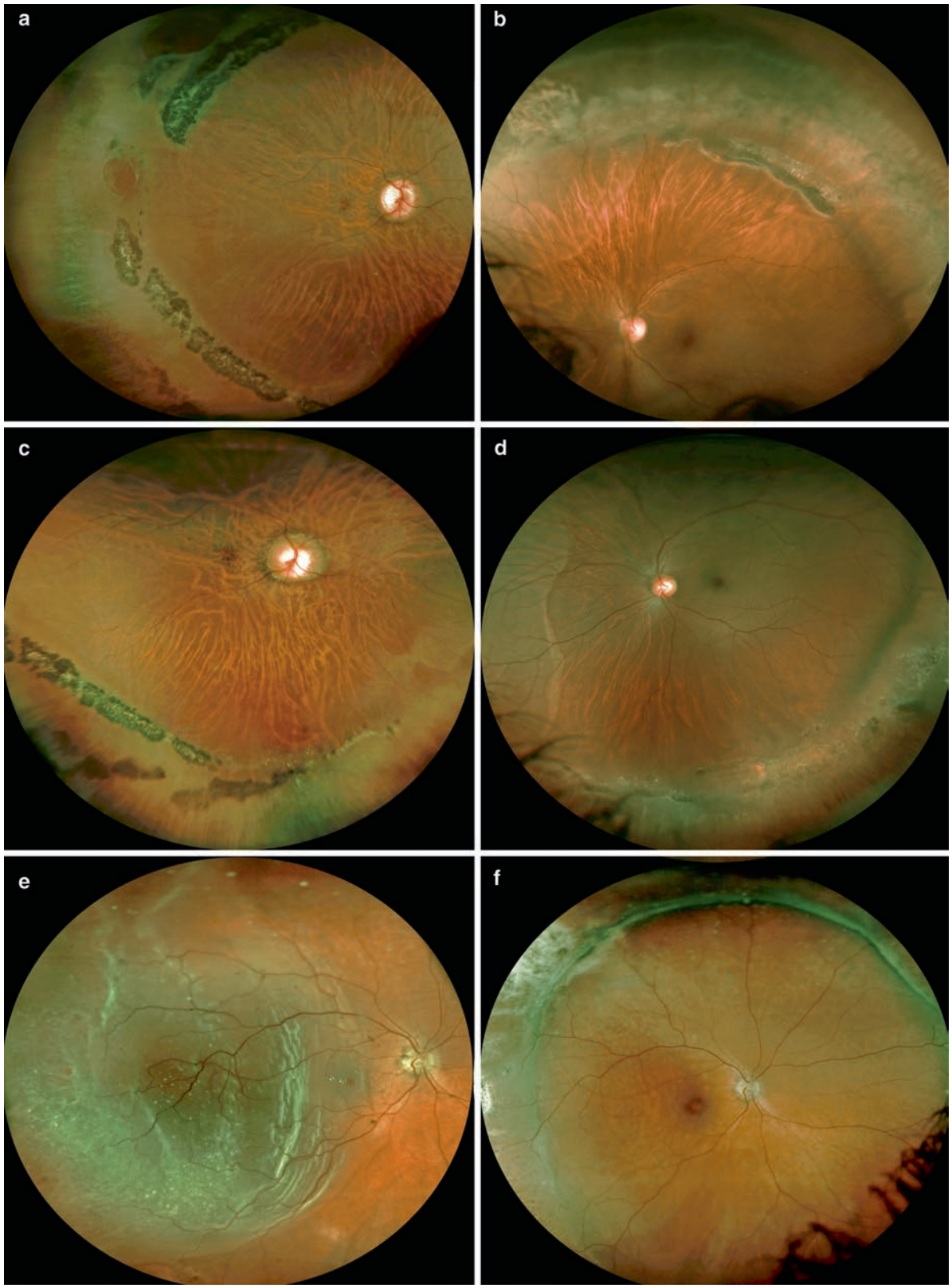

Fig. 22.9 Various clinical features of atrophic retinal holes in two myopic patients, a 39-year-old male (**a**–**d**) and a 29-year-old male (**e**, **f**), using ultrawide-field fundus multicolor photographs. (**a**–**d**) Bilateral multiple atrophic round holes in lattice lesions: most of them are tiny (**b**–**d**) and one is 1 disc diameter (**a**). There was a superior retinal detachment caused by an atrophic retinal hole in lattice degeneration in the left eye (not shown) treated with cryotherapy and encircling band because of the multiplicity of the peripheral atrophic holes in lattice degeneration (**b**, **d**). Note the white-without-pressure that may be confused with a retinal detachment in the temporal midperiphery of the left eye (**d**). (**e**, **f**) Bullous temporal retinal detachment caused by an atrophic round hole (**e**) treated successfully with cryotherapy and encircling band due to the multiplicity of peripheral breaks (not shown) (**f**)

22.9.1 Modulation of the Risk in Myopic, Aphakic, and Fellow Eyes

Börhinger reported that the lifetime risk of retinal detachment was 0.2% for emmetropes and hyperopes and myopes to −1.00 D, 4% for myopes −5 to −9 D, and reached 7% for high myopes greater than −9 D (quoted in [86]). The prevalence of retinal detachment after cataract extraction has been reported to be as high as 6.7% in 136 highly myopic eyes in 1975 in the era of intracapsular extractions and 0.28% according to the same authors in emmetropic eyes [87]. More recently, the risk for postoperative retinal detachment in high myopes has been reported to be 1.5–2.2% in 2356 eyes [88].

Breaks in aphakic eyes have been found to be more prone to progression [63]. Fellow eyes of patients who had a retinal detachment develop a retinal detachment in 5–10% of cases [89–91]. Aphakic fellow eyes have been found to develop retinal detachment in 26% of cases [92]. Therefore, the consensus is to sometimes treat asymptomatic flap tears in myopic eyes, aphakic eyes, and fellow eyes of patients who had a retinal detachment [39].

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