Review Article

Crack in 4 Silent Membrane: Angioid Streaks

Swarnima Sonkar*, Sandeep Saxena

Department of Ophthalmology, King George Medical College, Lucknow, Uttar Pradesh, India

Abstract

Angioid streaks are irregular, crack-like abnormalities of Bruch's membrane produced by loss of its normal elastic property and subsequent mineralisation or mechanical fragility. Foveal involvement, choroidal neovascularisation (CNV), subretinal haemorrhage and traumatic choroidal rupture can cause irreversible central visual loss. The most important clinical task is therefore not simply to recognise the streaks, but to identify an associated systemic disorder and detect macular complications at their earliest. Assessment combines dilated fundus examination with colour and red-free photography, fundus autofluorescence (FAF), optical coherence tomography (OCT), fluorescein angiography (FA), and OCT angiography (OCTA).

Keywords: Angioid streaks, Bruch's membrane, Pseudoxanthoma elasticum, Choroidal neovascularization.

Introduction

Angioid streaks are a funduscopic abnormality at the level of Bruch's membrane, where degeneration and mineralisation make the membrane brittle. The resulting breaks look like narrow reddish-brown or dark linear bands radiating from the optic disc. Their vessel-like appearance can be misleading. Multimodal imaging becomes important when the lesions are faint, the macula is involved, or CNV is suspected.[1-5]

Aetiology and Systemic Associations

- Angioid streaks may be idiopathic, but a systemic association should be considered, particularly in a younger patient or when the lesions are bilateral and extensive. Pseudoxanthoma elasticum (PXE) is the best-known association and reflects mineralisation and fragmentation of elastic tissues. Other associations include Paget disease of bone, sickle-cell disease and related haemoglobinopathies, Ehlers–Danlos syndrome and other connective-tissue disorders.[1,3,4]
- PXE: lax or redundant skin, yellowish papules or plaques in flexural areas and vascular manifestations.
- Paget disease of bone: bone pain, deformity, increased alkaline phosphatase or radiologic evidence of abnormal bone remodelling.
- Haemoglobinopathies: consider sickle-cell disease or trait and relevant family history; confirm with haemoglobin analysis when appropriate.
- Connective-tissue disorders: systemic hyperelasticity, joint manifestations or a known inherited syndrome.

Pathophysiologic Considerations

Abnormal Bruch's membrane due to loss of elasticity/ mineralization leading to cracks in Bruch's membrane, secondary RPE/choriocapillaris disturbance causing vulnerability to haemorrhage and fibrovascular ingrowth. CNV can develop through a break and may be the dominant cause of visual loss. Even relatively minor blunt trauma can produce a choroidal rupture with subretinal or submacular haemorrhage because the membrane is mechanically fragile.[2-5]

Clinical Phenotype and Investigations

Most patients are usually asymptomatic until a streak approaches or crosses the fovea, or CNV develops. Sudden vision loss, metamorphopsia or micropsia call for immediate macular assessment. On funduscopic examination, streaks are typically bilateral, irregular and jagged, around a

Address for correspondence: Swarnima Sonkar
Department of Ophthalmology, King George Medical College, Lucknow, Uttar Pradesh, India
E-mail: sonkarswarnima@gmail.com

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Figure 1: Color fundus photograph showing angioid streaks



Figure 2: Red free fundus photograph

peripapillary ring of pigmentary alteration which extends outward. They may cross beneath retinal vessels, which distinguishes them from true vascular structures. A 'peau d'orange' fundus pattern and other PXE-related changes can strengthen the systemic diagnosis.[1-4]

Baseline Ophthalmic Assessment

Best-corrected visual acuity, refraction and colour vision, macular assessment when metamorphopsia is suspected. Slit-lamp examination and dilated fundus examination, intraocular pressure and routine ocular examination.

Multimodal Retinal Imaging

Colour fundus photography remains the starting point, but subtle streaks may be easier to appreciate with red-free, infrared or multicolour imaging (Figures 1 and 2).

Fundus fluorescein angiography (FAF) is particularly useful for mapping the retinal pigment epithelium (RPE) response: areas overlying established streaks often show reduced autofluorescence, while adjacent stressed or hyperpigmented RPE may produce increased signal.[3,6]

Optical coherence tomography (OCT) is essential whenever the macula is symptomatic, or CNV is suspected. It can demonstrate RPE/Bruch's membrane irregularity, focal disruptions, outer retinal loss, subretinal or intraretinal fluid, subretinal hyperreflective material, haemorrhage-related changes and fibrovascular scarring. OCT Angiography (OCTA) can reveal a neovascular network without dye injection and is useful for detection and follow-up of CNV. However, structural OCT and clinical examination remain essential for judging exudative activity (Figure 3).[3,6]

FAF shows characteristic leakage patterns in CNV, in contrast to angioid streaks, which show linear hyperfluorescent tracks. Indocyanine green angiography can provide additional information about deeper choroidal pathology.[3,6]

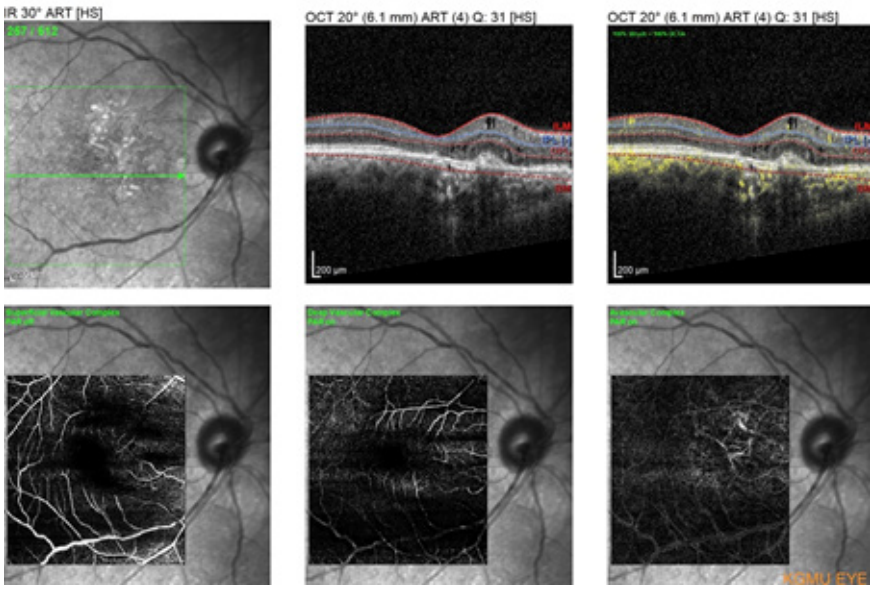


Figure 3: OCT/OCTA demonstrates a neovascular complex with associated structural changes: hard exudates and cystic spaces in the macula and choroidal dots

Systemic Evaluation

Systemic investigations are usually guided by history and examination. When PXE is suspected, dermatological assessment, histopathology, and ABCC6 molecular testing are indicated. The key principle is to identify a systemic disorder that may affect cardiovascular, gastrointestinal, skeletal or dermatologic health as well as the eye.[1,2,4]

Major Vision-threatening Complications

- Choroidal neovascularisation: the most important cause of progressive central visual loss; may present with metamorphopsia, haemorrhage or exudation.[1,2]
- Subretinal/submacular haemorrhage: may occur spontaneously or after minor blunt trauma and can directly damage the fovea.[2,4]
- Foveal involvement by an angioid streak: chronic RPE/choriocapillaris disruption may produce irreversible central dysfunction.[1,2]
- Chorioretinal atrophy and disciform scarring: may develop independently of active CNV and contribute to long-term visual impairment.[2]

Differential Diagnosis

The principal mimics are lacquer cracks of pathologic myopia, traumatic choroidal rupture, inflammatory chorioretinal scars and selected macular/choroidal disorders. Lacquer cracks are usually associated with high myopia and posterior staphyloma; choroidal rupture is linked to trauma and tends to have a different orientation and clinical history. The combination of bilateral peripapillary radiating streaks, peau d'orange changes and systemic features strongly supports angioid streaks. Multimodal imaging helps resolve uncertain cases.[3,5]

Management: Complication-directed Rather Than Streak-directed

No established treatment reverses the underlying Bruch's membrane abnormality. Asymptomatic patients without macular complications generally require observation with periodic visual assessment, fundus documentation and OCT. Patients should be counselled about the potential for haemorrhage after ocular trauma and risk of CNV. Protective eyewear is advisable for activities with a risk of blunt injury.[1-4]

When CNV is present, intravitreal anti-VEGF therapy is the principal contemporary treatment. Treatment decisions should be based on symptoms, OCT evidence of exudation and angiographic/OCTA findings rather than on the presence of streaks alone. Photodynamic therapy or other interventions may have a role in selected cases, but anti-VEGF therapy has become the mainstay for active macular neovascularisation.[1-4]

Conclusion

Angioid streaks are regarded as a clinical marker of a fragile Bruch's membrane rather than an isolated fundus curiosity. Recognition of the characteristic peripapillary radiating pattern on fundus should trigger two parallel questions: 'Is there an associated systemic disorder?' and 'Is the macula already threatened by CNV or haemorrhage?'

A structured multimodal approach—particularly colour photography, FAF, OCT and targeted angiography/OCTA—improves documentation, detects complications early and guides treatment. In ophthalmology practice, the most important intervention is often not treating the streak itself but preventing and promptly treating complications that can convert an asymptomatic finding into permanent central visual loss.

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