Differential diagnosis

Differential diagnosis of PCV with central serous chorioretinopathy, AMD with CNV, inflammatory conditions and tumors is not always easy and needs ICG for a clear differentiation.

Polypoidal choroidal vasculopathy is a primary cause of macular serous retinal detachment without hemorrhage in patients over 50 years of age in Asian population[(32)].

Since clinical and fluorescein angiographic findings are often indistinguishable among central serous chorioretinopathy, PCV, and occult choroidal neovascularization, indocyanine green angiography might help to establish a more definitive diagnosis[(16,33)].

Central serous chorioretinopathy shows staining or late leakage but not an abnormal choroidal vascular network neither polyps.

The differential diagnosis becomes more challenging when lipid exudation and small PED are associated. ICG may be helpful differentiating PED from polypoidal lesions.

Small PED from central serous chorioretinopathy become hypofluorescent in late phases ICG and hyperfluorescent in late phases fluorescein angiography.

In contrast, polypoidal lesions are usually hyperfluorescent in late phases ICG because of its vascular nature[(16)].

PCV represents a subtype of CNV in AMD[(4,10,17,18,19)].

However some features distinguish PCV from other subtypes of CNV: eyes with PCV are characterized by a higher incidence of neurosensory detachments, greater neurosensory detachment height, and less intraretinal oedema than eyes with occult or predominantly classic CNV[(34)].

Non polypoidal lesions in exudative AMD patients tend to produce small calibre vessels that are associated with grayish membranes not easily observed clinically, in contrast with the redish-orange lesions clinically observed in PCV and corresponding to vascular saccular polypoid lesions[(18,34,35,36)].

Stromal choroidal fibrosis is common in predominantly classic and occult lesions but is quite rare in PCV.

PED associated with CNV in AMD has a poor prognosis whilst PED in PCV lesions virtually never forms fibrotic scars[(16,18,29)].

The natural evolution of CNV in AMD eyes to fibrosis and disciform scar is not observed in PCV eyes.

Tumoral lesions like choroidal circumscribed hemangioma, renal cell carcinoma or metastasis from carcinoid syndrome may also be confused with PCV[(29)]. Again ICG is essential for differentiation.

Choroidal hemangiomas show, in general, a rapid filling of dye in very early phases and a washout in late phases.

ICG characteristic lesions of PCV are not observed in choroidal or metastatic tumors and ultrasound is also effective for characterization of the tumoral mass.

Inflammatory lesions like, posterior scleritis, multifocal choroiditis, panuveitis, acute posterior multifocal placoid pigment epitheliopathy, Harada disease, sympathetic uveitis, birdshot chorioretinopathy may also be confused with PCV.

PCV does not course with anterior or posterior uveitis neither with pain or staining of the optic disc in fluorescein angiography[(4,16,18,29)].

Lipid deposition, often observed in PCV is not commonly seen in inflammatory conditions.

Scleral or choroidal thickening and liquid in the subtenon space have never been described in PCV eyes[(18,29)].

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