# Choroidal neovascularization

Choroidal neovascularization is considered to cause almost 90% of the cases of severe visual loss related to AMD[(50)].

CNV is usually studied by fluorescein angiography and OCT to assess the extent, location and nature of the lesion[(51)].

Fluorescein angiography shows changes in retinal vascularization, but does not reveal how deeply RPE is affected.

FAF imaging shows RPE damage, with the advantage that is a non invasive test, less time consuming than angiography.

Several studies have reported that CNV may show irregular FAF alternating areas of increased, normal and decreased fluorescence intensity[(20,34-37)]. (Fig. 14).

Areas of abnormal FAF extend beyond the edge of the angiographically defined lesion.

As in other exudative retinal diseases such as central serous chorioretinopathy, areas of increased FAF next to CNV are frequently found inferior to the leaking areas in fluorescein angiography.

The hypothesis was that they might represent areas with subretinal fluid and that their location was influenced by gravity. Other fluids may typically decrease FAF, as occurs with haemorrhages and exudates. Decreased FAF is caused by blocked fluorescence.

It is usually necessary to compare the results of FAF with colour photographs.

Recent research has examined early CNV in FAF[(36, 52, 53)], reporting that early CNV lesions tend to show normal FAF in areas that were hyperfluorescent in fluorescein angiography, whereas eyes with a history of one month or more since CNV was diagnosed, showed decreased FAF in areas of previous fluorescein leakage[(52)].

These data suggest that RPE affected by the CNV may still be viable in the early stages of the disease (Fig. 15).

These studies have also reported that areas with previously high levels of FAF may show decreased FAF six months later[(36)].

These changes may be secondary to photoreceptor loss, RPE atrophy, replacement of normal phenotypes of RPE cells with scar, and increased melanin deposition.

These findings may have therapeutic implications and clarify long-term visual prognosis.

For example, a person with an active CNV on fluorescein angiography, and normal FAF, may show a much better outcome than another with an abnormal basal FAF.

Data comparing FAF findings in occult and classic CNV are limited.

Spital et al. reported that classic CNV usually shows more focal areas of decreased FAF than occult CNV(34).

These findings have been confirmed by McBain et al.(54) who guessed that low FAF at the site of the CNV are related to blocked fluorescence induced by the presence of CNV in the subretinal space, rather than to severe damage to the RPE.

A more recent study did not find significant differences in FAF patterns in early classic and occult CNV secondary to AMD(53) (Fig. 16).

A continuous preserved autofluorescence pattern was observed in the central macula in most of the cases.

These findings suggest that neovascular complexes, regardless if classic or occult, would be external to the RPE in most cases.

Additional studies with a higher number of patients and longer monitoring are required to verify with these changes in patients with CNV (Fig. 17).

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