

IMAGE OF THE MOMENT

Gawn G. McIlwaine*, **James H. Vallance*** and **Christian J. Lueck†**

*Princess Alexandra Eye Pavilion, Chalmers Street, Edinburgh UK; †The Canberra Hospital, P.O. Box 11, ACT, 2606, Australia; Email: Christian.Lueck@hotmail.com
Practical Neurology, 2003, **3**, 178–183

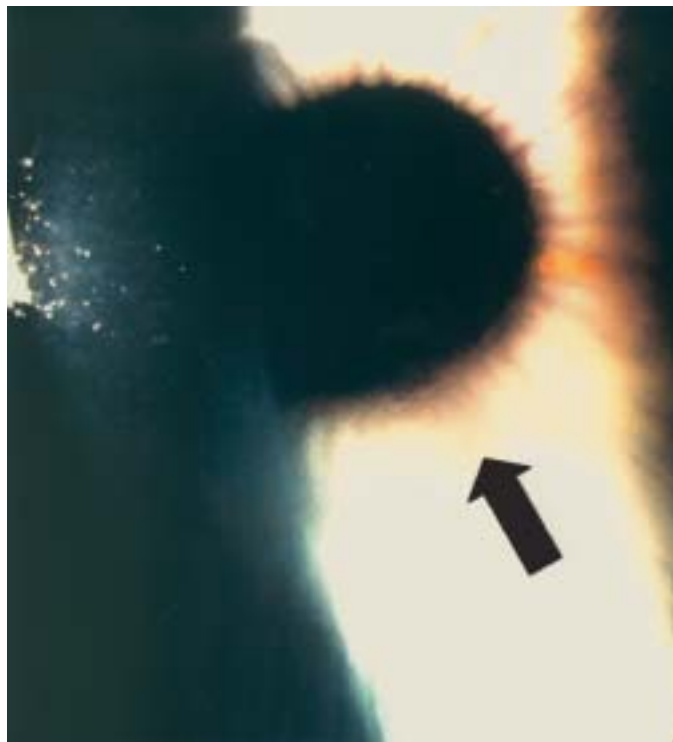


Figure 1 Fuchs' endothelial dystrophy. Slit lamp photograph showing corneal endothelial guttatae (arrow) visualized against the iris as background.

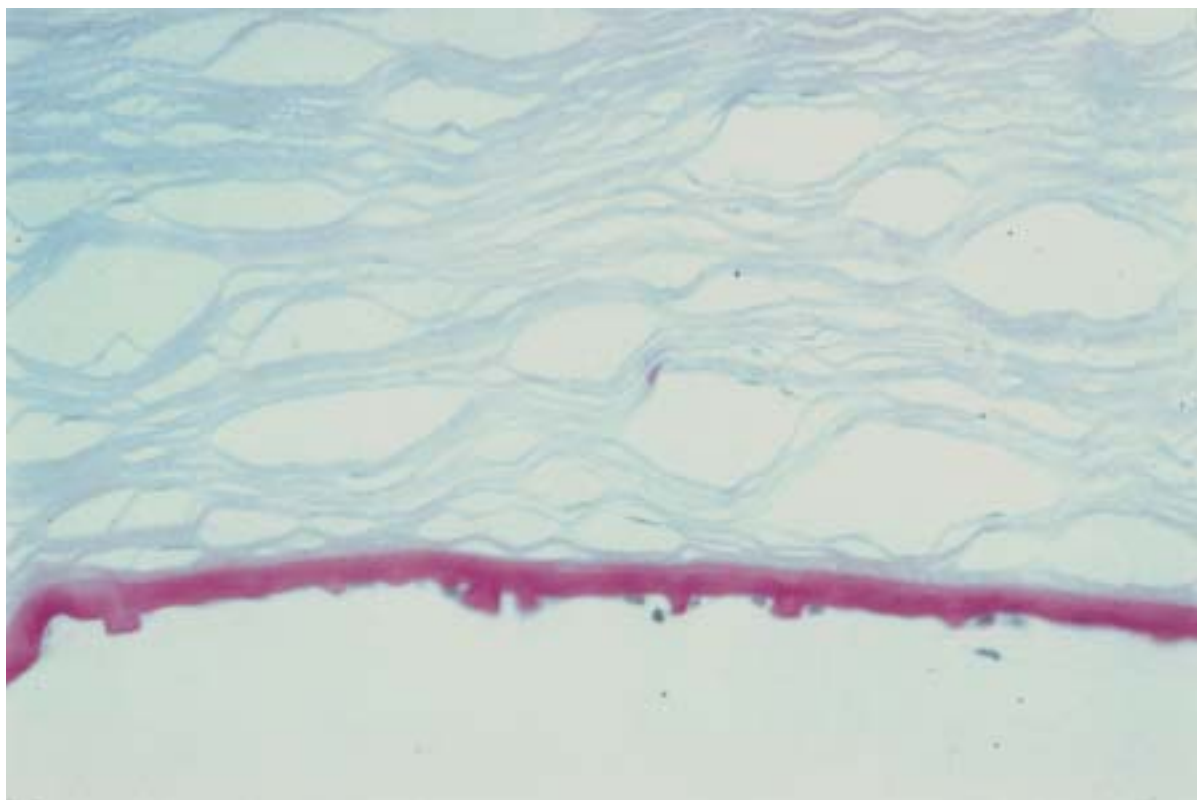


Figure 2 Fuchs' endothelial dystrophy. Histological section of the posterior cornea (haematoxylin and eosin) demonstrating guttatae and sparse endothelial cells (James Ironside, Department of Neuropathology, Western General Hospital).

Transient or intermittent visual loss

The neurologist will be quite familiar with common causes of transient or intermittent visual loss such as migraine, ischaemic amaurosis fugax and optic neuritis. However, if there is any doubt about the diagnosis, and the patient hasn't already been seen by an ophthalmologist, then he or she should be referred to make sure the eye itself is normal. This is because confusion can occasionally arise when the visual loss is actually ocular in origin, as in the following examples.

OBSCURATIONS DUE TO PAPILLOEDEMA

Patients with papilloedema of any cause can develop transient visual disturbance, often brought on by change in posture such as bending over. The visual loss is typically 'grey' rather than 'black', bilateral rather than monocular, and lasts for a matter of seconds, as opposed to the total visual loss in ischaemic and monocular amaurosis fugax, which typically lasts a minute or two.

FUCHS' CORNEAL ENDOTHELIAL DYSTROPHY (CORNEAL GUTTATAE)

(See Figs 1 and 2.) The function of the corneal endothelium is to pump fluid out of the cornea, thereby keeping it relatively dehydrated. Abnormal excrescences of Descemet's membrane (the basement membrane of the corneal endothelial cells) are called corneal guttatae; the endothelial cells are then deficient or malfunctioning, and patients present with blurred vision. In the early stages this tends to occur in the morning, and gradually resolves during the day as the cornea clears due to the evaporation of tears. Symptoms usually occur in the seventh to eighth decade. Similar symptoms can occur due to endothelial cell loss as a consequence of intraocular surgery.

ANGLE CLOSURE GLAUCOMA

(See Figs 3 and 4.) This typically presents in people who are hypermetropic or long-sighted. They are usually over the age of 50 years, and

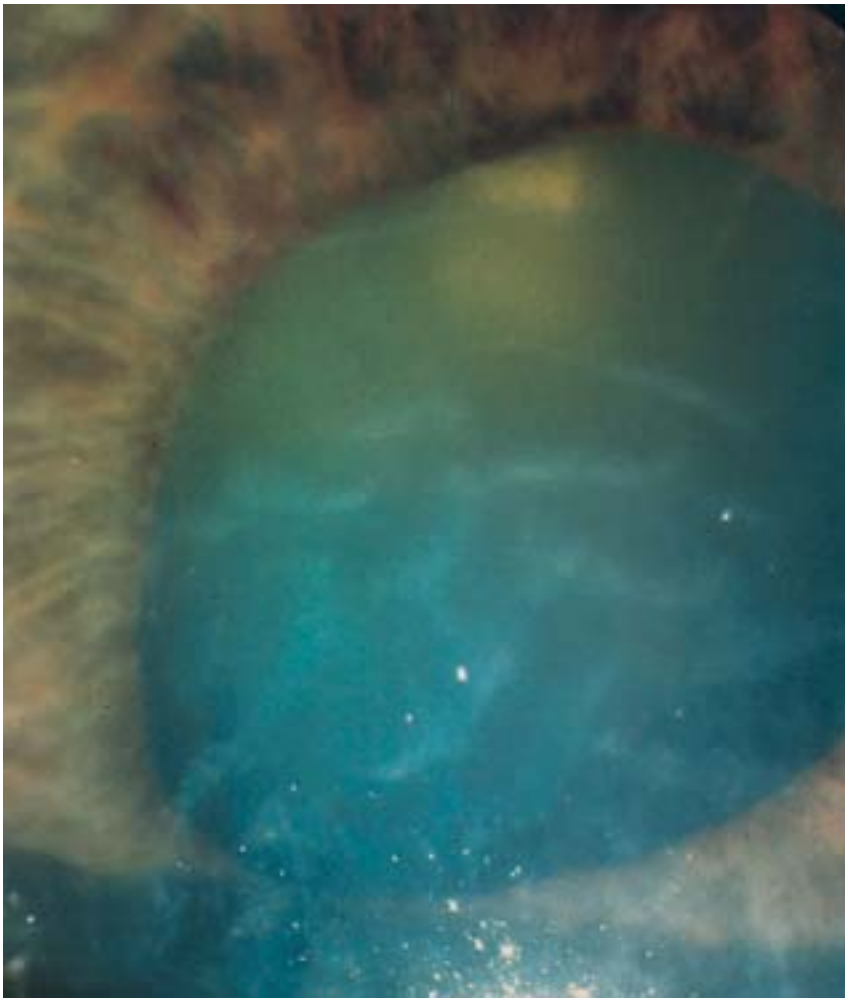


Figure 3 Acute angle closure glaucoma with corneal oedema (the circular green area) and fixed dilated pupil (green – brown) around the edge.

attacks tend to occur in dimly lit conditions. Patients can occasionally present with intermittent visual loss which may be associated with haloes around lights. This is typically associated with pain in one or both eyes.

ANTERIOR ISCHAEMIC OPTIC NEUROPATHY, SECONDARY TO GIANT CELL ARTERITIS

(See Figs 5 and 6.) These patients often give a history of intermittent monocular visual loss before they go on to develop permanent visual loss from anterior ischaemic optic neuropathy. A low threshold of suspicion should be maintained in anyone aged over fifty.

if there is any doubt
about the diagnosis,
and the patient hasn't
already been seen by an
ophthalmologist, then he
or she should be referred
to make sure the eye itself
is normal



Figure 4 Hypermetropic glasses demonstrating magnification.



Figure 5 Prominent, tortuous superficial temporal artery in giant cell arteritis (provided by the late Professor WB Matthews).

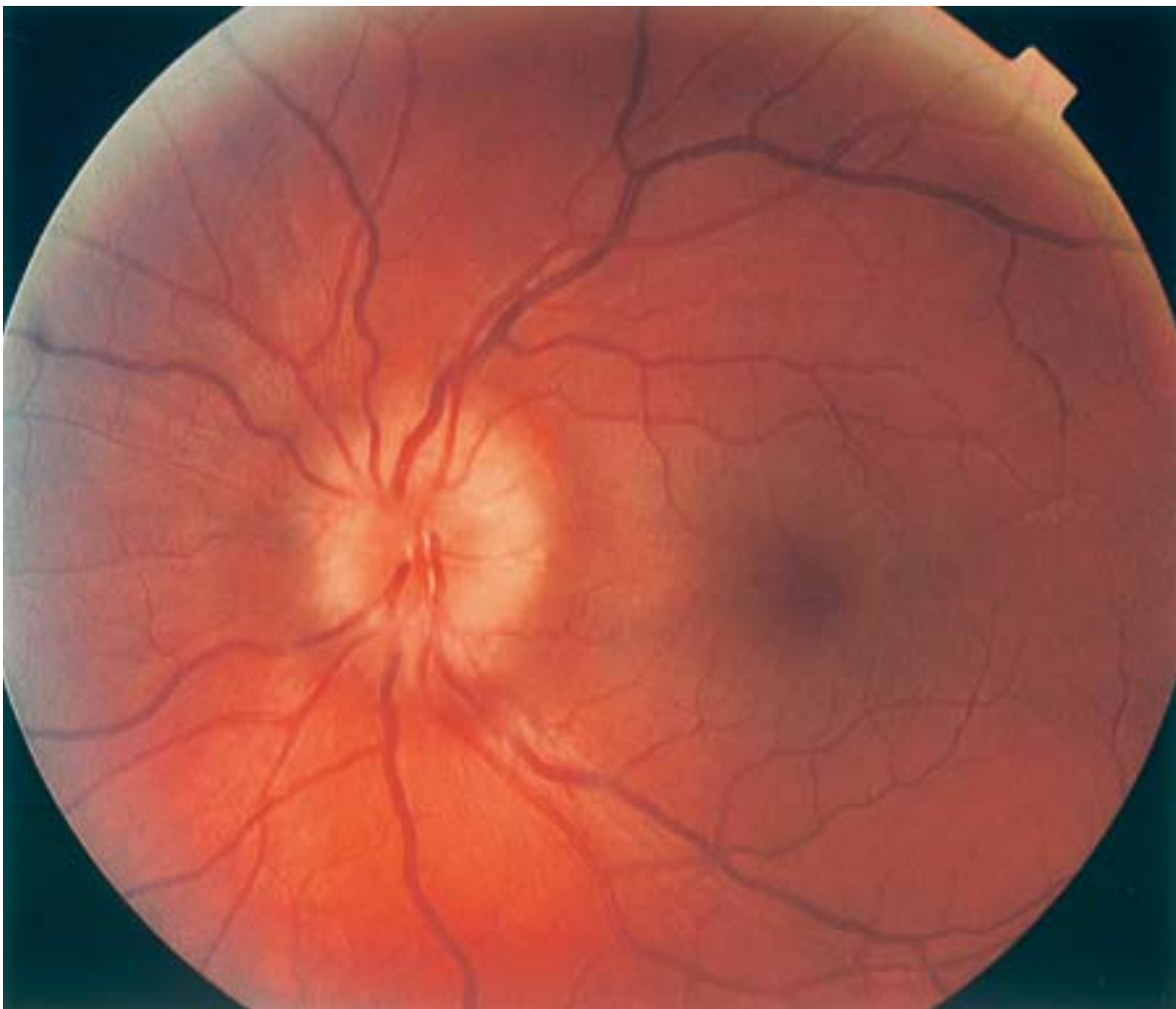


Figure 6 Pale, swollen optic disc in anterior ischaemic optic neuropathy.

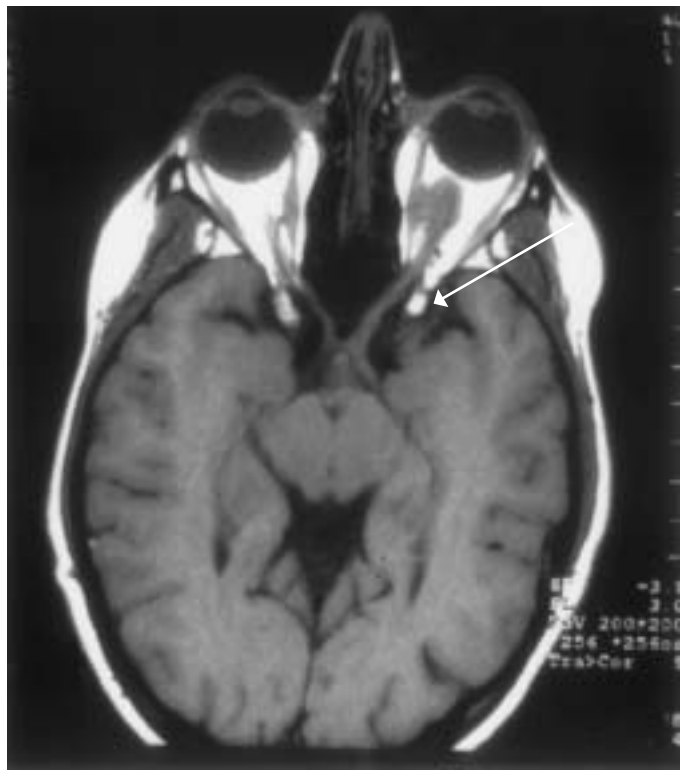


Figure 7 MRI scan of a patient with gaze-evoked monocular visual loss due to an optic nerve sheath meningioma showing as a fusiform enlargement of the left intraorbital optic nerve (arrow).

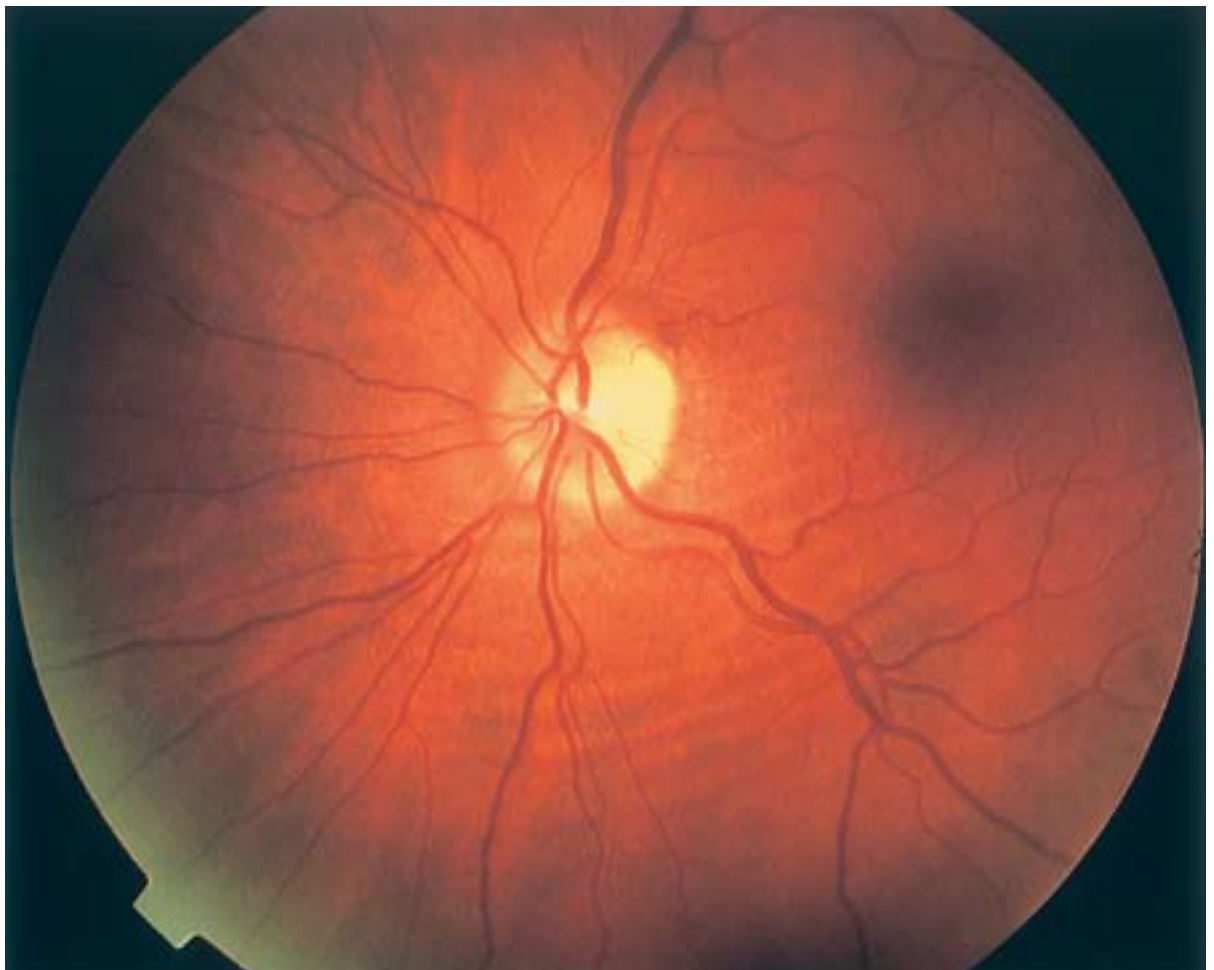


Figure 8 Choroidal folds inferior to the disc caused by an orbital mass.

ORBITAL TUMOURS

(See Figs 7 and 8.) Patients with even relatively small orbital tumours of any sort can suffer from gaze-evoked visual loss. This manifests as blurred or complete visual loss in the affected eye, particularly in abduction. The easiest way to confirm this clinically is to check the Snellen acuity with the patient looking straight ahead, and then again with the patient turned so that the affected eye is in abduction.

CENTRAL SEROUS RETINOPATHY

(See Fig. 9.) This typically affects young men in stressful occupations. Central vision becomes blurred with micropsia (small image due to separation of the photoreceptors) and distortion over a period of hours to days, and it lasts for

days to weeks. It can therefore easily be confused with optic neuritis. The clue to the diagnosis is that the retina is not normal on fundoscopy. There is leakage of fluid through the retinal pigment epithelium elevating the retina, but this may require pupillary dilatation to be visible.

PTOSIS

It is always worth remembering that intermittent ptosis (e.g. myasthenia gravis) can cause transient monocular visual loss.

ACKNOWLEDGEMENTS

We would like to thank Stuart Gairns, Department of Photography, Princess Alexandra Eye Pavilion, for his help in preparing the photographs.

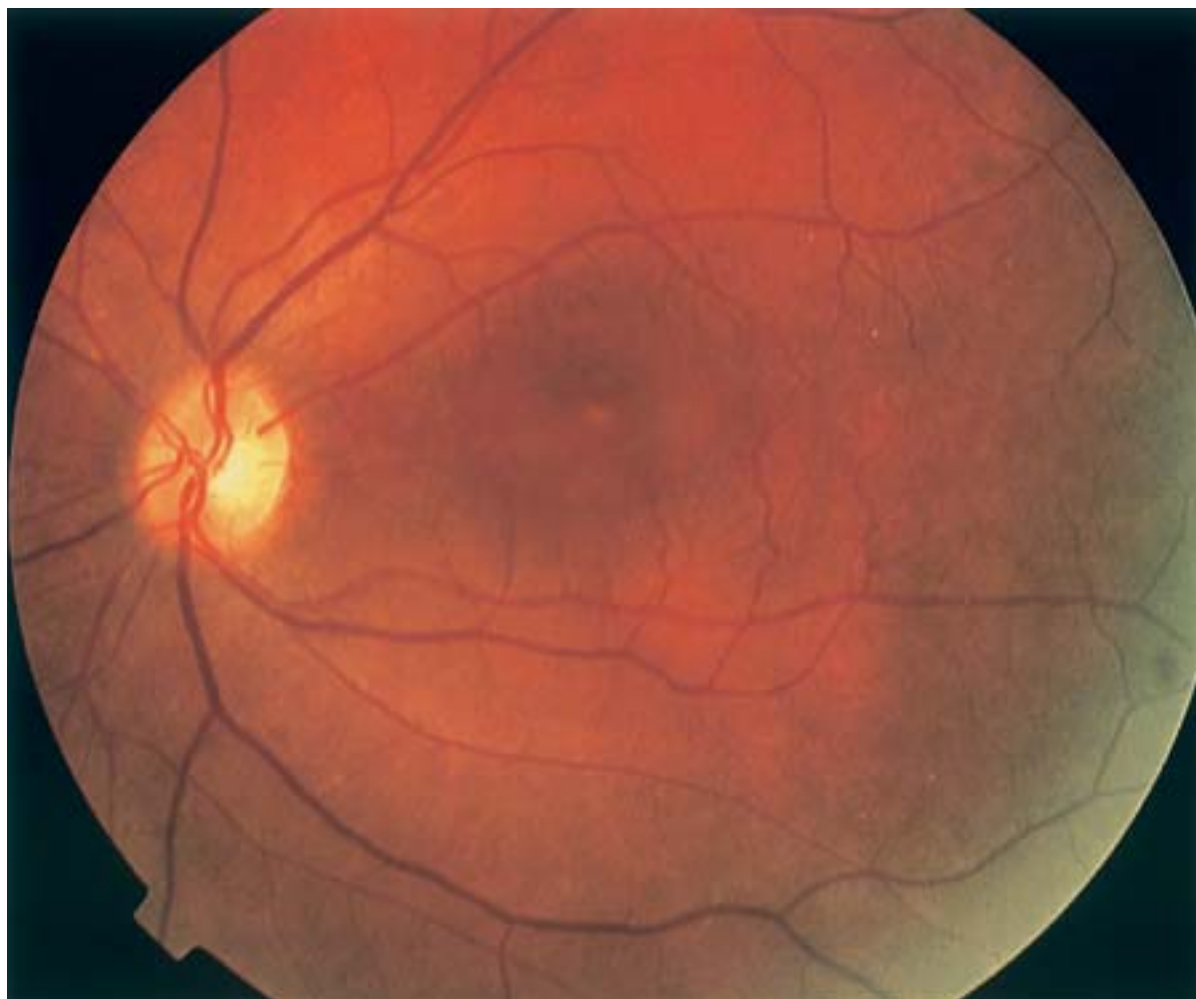


Figure 9 Central serous retinopathy. Oval collection of subretinal fluid and yellow spot seen at the macula.