Clinical Commentary

Fluorescein angiography in equine ophthalmology

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The retinal vasculature of the horse is classified as paurangiotic with 50–80, small diameter retinal arterioles and venules arising from the edge of the optic disc. These retinal vessels then extend only a short distance into the retinal tissue (Brooks and Matthews 2007) (Fig 1). The retina ventral to the optic disc does not receive any blood from this small retinal circulation. The equine retina thus depends primarily on the choroidal circulation for its blood supply. Small dots distributed in a uniform pattern throughout the tapetal fundus are called stars of Winslow and represent end-on views of large choroidal capillaries penetrating the tapetum to form the choriocapillaris layer adjacent to the retina.

Fluorescein angiography (FA) is a commonly utilised ophthalmic diagnostic technique to study microcirculatory abnormalities of the choroid, retina and optic nerve in humans and small animals (Kallberg 2007). It permits analysis of the retinal and choroidal blood vessels, their permeability, ocular blood flow transit times, and the integrity of the retinal pigment epithelium (RPE). It has never been used clinically to a high degree in veterinary medicine despite many attempts due to the cumbersome and often expensive FA equipment, and more importantly due to the lack of significance of FA findings to the treatment and prognostication of choroidal, retinal and optic nerve diseases of animals. Slater et al. (1995) used FA to study the peripapillary, small, circular chorioretinal scars with a hyperpigmented centre and a depigmented peripheral ring of horses with chorioretinitis caused by equine herpesvirus-1 (EHV-1). Dan Wolf studied techniques for using FA in horses at the University of Florida in the mid-1980s (Fig 2).

Sodium fluorescein is a weak acid that maximally fluoresces in response to light at a wavelength of 490 nm, emitting light with a peak wavelength of 520 nm. Once injected i.v., sodium fluorescein appears in the eye within 20–25 s in the horse. After entering the choriocapillaris, unbound fluorescein readily passes into the choroidal interstitial tissue via large choroidal vascular endothelial fenestrae. Movement of dye into the retina is prevented by the tight junctions between cells of the RPE. Fluorescein entering the retinal vasculature is retained within the lumen of these vessels by tight junctions and the lack of fenestrae between the retinal vascular endothelial cells. Breakdown of either the RPE or retinal vascular barrier allows fluorescein to enter the retina or choroid (Kallberg 2007).

Fluorescein angiography is best performed with special fundus cameras developed for this purpose. A blue excitation filter in the light pathway induces maximal fluorescence, and
a yellow filter in the optical pathway provides maximal contrast (Kallberg 2007). Fluorescein (10%, 2 mg/kg bwt) is administered rapidly via the jugular veins in the horse and photography is initiated. Phases of the FA in the holangiotic retinas of man and dogs include prefilling, choroidal, retinal arteriolar, retinal capillary, retinal venous and recirculation (Kallberg 2007). With optimal fluorescein doses, a second retinal arteriolar phase (i.e. recirculation phase) can also be observed. The FA phases in the horse are not so distinct (Pachten et al. 2007). Sodium fluorescein is rapidly eliminated via the kidneys and stains the urine.

Abnormalities of the fluorescein angiogram are described as either hyper- or hypofluorescent. Hyperfluorescence occurs with leakage of fluorescein into the vitreous or the subretinal space, staining of tissue from leaky retinal or choroidal blood vessels, RPE defects (i.e. increased choroidal visibility), and increased accumulation of fluorescein associated with tumours, active haemorrhages and neovascularisation or aneurysms. Hypofluorescence results from vascular vessel attenuation or occlusion, and from visual obstructions to the fluorescence from retinal pigment, haemorrhage, oedema and exudates. Autofluorescence occurs normally in the optic disc and in the tapetal region of the horse fundus (Kallberg 2007).

There is a suggestion of a microcirculatory basis for many retinal and optic nerve lesions of horses, and FA using SDO could provide needed information on their pathogenesis and suggest an effective treatment. Chorioretinitis manifests in equine eyes as focal ‘bullet hole lesions,’ diffuse chorioretinal lesions, nontapetal ‘horizontal band’ lesions and peripapillary chorioretinitis (Brooks and Matthews 2007). ‘Bullet hole’ chorioretinitis is very common clinically and may be seen as white, focal or multifocal, circular scars ventral to the optic disc. ‘Bullet-hole’ chorioretinitis typically leaves a scar consisting of a hyperpigmented centre surrounded by a white, depigmented zone. EHV-1-mediated infarction of the choroid results in RPE depigmentation at the periphery of the infarct and RPE hypertrophy at the central region to cause this condition (Slater et al. 1992, 1995; Allen et al. 2004). Acute active chorioretinitis lesions demonstrate a focus of grey-white retinal oedema (Slater et al. 1992) but must not be commonly noticed clinically as they are not overtly painful, and are not documented by any photographs being present in the scientific literature.

Fig 3: ‘Horizontal band’ retinopathy in a blind horse. Depigmentation below the optic disc is present (courtesy of Dr Adolfo Guandalini, Rome, Italy).

Testing with FA could benefit the following equine eye diseases as we know very little about the pathogenesis of these eye diseases of horses and thus do not have effective therapies to treat them (Brooks and Matthews 2007): the blinding ‘horizontal band’ chorioretinitis of horses (Fig 3); trauma to the occipital region of the equine skull to cause an optic nerve stretching injury and subsequent atrophy (Fig 4); equine glaucoma; amaurotic blindness of horses; ischaemic optic neuropathy from ligation of the internal carotid, external carotid and greater palatine arteries to prevent epistaxis from guttural pouch mycosis; equine proliferative optic neuropathy; and pigmented choroidal lesions (Fig 5).

The development of the scanning digital ophthalmoscope (SDO) utilised by Pachten et al. (2008) in the accompanying
paper may be an important step in utilising FA clinically in large animals with posterior segment disease. In this paper the FA function of an SDO unit was used to examine the microcirculation of the retina and optic nerve of 2 blind horses. Optic nerve atrophy with reduced optic nerve head blood perfusion, and chorioretinal scarring and optic nerve atrophy with fluorescein dye leakage were diagnosed in the 2 horses respectively.

References


