

## Cortical Blindness Following Coronary Angiography

The article by Kwok and Lim<sup>(1)</sup> has stimulated my interest. The authors assume that the transient cortical blindness developed in this 53-year-old man, after coronary angiography, was due to a direct idiosyncratic neurotoxic reaction to contrast agent.

However, I feel that there were a few pointers to suspect a transient cerebral embolic event in the distribution of the upper basilar artery in this patient. Bilateral altitudinal visual field defect progressing to total blindness, preservation of pupillary light reflexes, "catatonic state" with unresponsiveness to verbal cues and total inability to exhibit any spontaneous body movement - all suggested a basilar artery syndrome with bilateral posterior cerebral artery occlusion giving rise to total cerebral or cortical blindness. It appears that this patient possibly was either in a transient 'akinetic mute' or 'locked-in plus' state due to ischaemia of the brain stem, thus manifesting as "catatonia", as described by the authors.

Cortical blindness consists of complete loss of visual function including perception of light and dark and loss of reflex lid closure to menace, but with retention of reflex constriction of the pupils to illumination and convergence. Bilateral posterior cerebral artery (PCA) embolisation or thrombosis usually gives rise to total cerebral or cortical blindness<sup>(2,3)</sup>. A combination of brain stem and occipital lobe signs, as described in this patient, is eminently suggestive of a thromboembolic event in the distribution of basilar and PCAs. These posterior circulation syndromes can be due to (i) embolism from the heart, or (ii) thrombotic occlusion or ischaemia due to low flow complicating atheroma of the vertebrobasilar system.

The authors appropriately mentioned that the spasm of posterior cerebral arteries, dissection of the aortic arch, contrast-induced hypotension etc. can precipitate ischaemic cerebrovascular events during cerebral or coronary angiographic procedures. But, they conclude with the hypothesis that their patient plausibly had a direct idiosyncratic neurotoxicity. This possibility of direct toxicity to occipital cortex was unlikely because (i) he had a combination of brain stem and occipital lobe findings, as described earlier; (ii) patient was treated with antiplatelet therapy and low molecular weight heparin which would have counteracted the embolic phenomenon (iii) a negative computed tomography (CT) or magnetic resonance imaging (MRI) of the brain does not exclude an infarction in the first few hours (iv) transient ischaemic attacks usually improve within a few hours, as in this case.

Bilateral occipital lesions can cause altitudinal visual field defects, since the termination of the optic radiation is topographically arranged. The lower retinal fibres terminate in the cortex below the calcarine fissure and hence, superior quadrantic defects are found with inferior lesions and vice versa<sup>(4)</sup>. In most cases of cerebral or cortical blindness, the period of total amaurosis is brief, followed by progressive recovery of visual function<sup>(5)</sup>, as demonstrated in this patient. The majority of these patients show signs of mental status changes indicative of a diffuse or bilateral dysfunction necessary to cause cortical blindness. As a result, it may be often difficult to ascertain the degree of visual impairment<sup>(6)</sup>. It may be emphasised here that, when clinically confronted with altitudinal defects in the fields of vision (homonymous altitudinal hemianopias), one has to look for bilateral lesions of the occipital lobe and none of them are limited strictly to bilateral superior or inferior quadrants. The most common cause still remains to be a vascular disease affecting the vertebrobasilar system<sup>(6)</sup>.

The case reported by Kwok and Lim underscores the importance of exercising great caution, while performing diagnostic or interventional endovascular procedures so that thromboembolic complications can be avoided or minimised. This message becomes more relevant towards the new millennium when diagnostic and interventional vascular procedures are becoming more and more popular. Angiographic procedures can potentially dislodge atherosclerotic plaques distally, thus precipitating a transient ischaemic attack (TIA) or stroke. Hence, I would like to add that, in the elderly, prior to performing a coronary or carotid angiographic procedure, it is better to screen the functional status of intracranial vessels non-invasively with carotid duplex scan and transcranial doppler studies.

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