Cortical Blindness Following Coronary Angiography

B W K Kwok, T T Lim

ABSTRACT
A 53-year-old man developed transient cortical blindness after coronary angiography, which appears to be an adverse reaction to contrast agent. A possible mechanism of this complication is contrast penetration of the blood-brain barrier with direct neurotoxicity to the occipital cortex.

Keywords: blindness, adverse reaction, contrast media

INTRODUCTION
Contrast induced transient cortical blindness is an uncommon complication after cerebral angiography (1,2). Its occurrence after coronary angiography is even less frequently seen (3-5).

CASE REPORT
A 53-year-old Chinese man underwent coronary and left ventricular angiography after a positive exercise ECG stress test. During the procedure, less than 100 ml of contrast media ioversol (Optiray 350, Mallinckrodt Medical) was given. This was the patient's first exposure to contrast media.

Approximately 30 minutes after the procedure, the patient complained of loss of vision. Neurological examination revealed bilateral lower visual field defects which progressed rapidly to total blindness. Papillary reflexes were normal. Soon after, the patient went into a catatonic state. He was unresponsive to verbal cues and did not exhibit any spontaneous body movements. The initial diagnosis was embolism to the posterior cerebral circulation. However, the full neurological manifestation in this case cannot be explained by embolic phenomenon alone. The patient was treated with antiplatelet therapy and subcutaneous low molecular weight heparin. Both computed tomography (CT) and magnetic resonance imaging (MRI) of the brain did not reveal any abnormality.

Six hours after the angiogram, the patient began regaining his vision and could speak fluently and coherently. Over the next 12 hours, there was complete recovery of his neurological deficits. No repeat CT or MRI scans were done. On review 1 month later, he was noted to be well.

DISCUSSION
The incidence of cortical blindness following cerebral angiography has been reported to be 1-4% (2). Its occurrence after coronary angiography is far less common. Fischer-Williams (3) and Kinn and co-workers (4) reported 1 case in 12,367 coronary procedures (incidence 0.008%) and 3 cases in over 6,000 procedures (0.05%) respectively. Reviewing a 6-year period from our institution, we found 1 case in 6,670 coronary angiographic procedures (0.015%).

The onset of blindness is usually rapidly progressive. Symptoms may begin while the procedure is in progress or develop within 10 minutes after it is completed. Return of vision may begin within the next few hours. Generally by 12 hours, significant recovery would have taken place, although complete recovery may take up to 72 hours.

In the 3 cases described by Kinn (4), the patients did not have any associated neurological deficit. Parry (5) reported loss of right arm co-ordination in his patient. Sticherling (6) reported concomitant confusion and total retrograde amnesia. In addition to cortical blindness, our patient was unresponsive to vocal stimuli and did not exhibit any voluntary movements.

Previously postulated mechanisms included spasm of the posterior cerebral arteries, dissection of the aortic arch, and contrast-induced hypotension (7). Intracranial enhancement by contrast in the occipital region has been shown in some cases when CT was done (4-6). The predilection for this area may be explained by the patient's prolonged supine posture during coronary angiography. The mechanism has been attributed to direct contrast effect following a breakdown in the blood-brain barrier, allowing contrast media to accumulate in the occipital cortex (7). Demirtas (8) has suggested an immunological mechanism, although this seems unlikely. Direct idiosyncratic neurotoxicity still appears the most
plausible explanation, although the exact mechanism remains unknown. Our case supports this last hypothesis as the volume and concentration of contrast given were both very small.

There is limited experience with re-challenge in patients with history of cortical blindness. Rama(9) reported 3 cases who did not develop blindness on rechallenge. Re-exposure appears to be of no increased risk.

In summary, transient cortical blindness is a rare but recognised complication following coronary angiography, with an incidence of < 0.05%. The demonstration of contrast enhancement in the occipital cortex on CT scan confirms the diagnosis in most cases. Patient outcome is excellent, with complete recovery expected within 24 - 48 hours. Experience with re-exposure is limited, but appears to probably carry no increased risk.

REFERENCES