

## Transient cortical blindness after coronary angiography and graft study

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### ABSTRACT

Transient cortical blindness is extremely rare but usually reversible complication of coronary angiography. We report a case of transient cortical blindness in a 63 year old male patient one hour after native coronary and bypass graft angiography through the right femoral access. A non-contrast enhanced CT and MRI brain scans showed multiple asymmetrical lesions in brain parenchyma in the distribution of posterior cerebral circulation predominantly in the cerebellum and left occipital lobe. Direct contrast neurotoxicity to the sensitive occipital cerebral lobes seemed to be the possible cause but the exact mechanism of cerebral injury remains unknown.

**KEY WORDS:** Coronary bypass graft angiography, Cortical blindness, Contrast neurotoxicity.

Pak J Med Sci July - September 2011 Vol. 27 No. 4 917-919

### How to cite this article:

Sadiq MA, Azman W, Han CK, Unduk AW, Yaakob Z, Abidin IZ. Transient cortical blindness after coronary angiography and graft study. Pak J Med Sci 2011;27(4):917-919

### INTRODUCTION

Although transient cortical blindness is a known complication of cerebral and vertebral angiography with occurrence rate as high as 1% to 4%, it is very rare after angiography of coronary arteries and bypass grafts.<sup>1-4</sup> Transient cortical blindness following coronary angiography was first reported in 1970 and review of literature revealed only 23 such cases despite the widespread and frequent use of coronary angiography worldwide.

The National Institute of Health in America and British Cardiac Society have reported the incidence of transient cortical blindness in diagnostic coronary angiography as 0.03% and 0.06% respectively.<sup>5</sup> This is the first report of transient cortical blindness post coronary angiography or of graft study in our centre since the start of coronary angiography in 1982.

### CASE REPORT

A 63-year old man with diabetes and hypertension was admitted to cardiology ward for non ST Elevation Myocardial Infarction (NSTEMI). He had history of ischemic heart disease for 12 years and in 1996 underwent Coronary Artery Bypass Grafts (CABG) surgery with left internal mammary artery (LIMA) to left anterior descending artery and sequential radial artery graft to distal right coronary and posterolateral branch of left circumflex arteries. A selective native coronary angiogram with 6F left and right Judkins catheters was performed via the right femoral artery without difficulty. Sequential arterial graft was identified, selectively catheterized and showed no obstruction. LIMA to LAD was blocked. A total of 160 ml of non-ionic, water soluble, contrast medium (Iopamiro 370, Bracco S.p.A, Feretino, A)

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\* Received for Publication: March 4, 2011

\* Accepted: June 26, 2011

Italy) was used. Procedure was completed without any immediate complication.

One hour later, patient complained of sudden onset of headache and severe blurring of vision in both eyes followed by loss of vision. His blood pressure was 104/70mmHg with pulse of 86 beats per minute at regular intervals. Neurological examination did not show any cranial, sensory and motor nerves abnormality. Both of his pupils were equal and reactive to light. There was a partial ptosis noted on left eye however there was no obvious ophthalmoplegia and fatigability noted. Further history from wife revealed that the left eye ptosis was present two years prior to current event. Visual field examination revealed bilateral homonymous hemianopia. Fundoscopy examination also yielded normal finding. A diagnosis of transient cortical blindness was made.

A plain cranial computed tomographic (CT) done six hours after the procedure showed multiple ill defined hypodense lesions in brain parenchyma in the distribution of posterior cerebral circulation with lesions predominantly in the cerebellum and left occipital lobe. An MRI performed 24 hours later showed high intensity signals in both cerebellar regions and left occipital hemisphere suggestive of recent infarcts. Twenty four hours after the procedure, patient vision gradually improved and complete recovery was noted after 96 hours. He was discharged five days after angiogram with no residual blindness.

## DISCUSSION

The diagnosis of transient cortical blindness remains mainly clinical in patients presenting with rapidly progressive loss of vision or severe blurring of vision during or after coronary angiography. Cortical blindness, like in our patient, usually is not associated with any neurological deficit but there are reports of associated neurological deficit.<sup>3</sup> The CT scans findings vary from no abnormality to contrast enhancement in occipital lobes to multiple and multilevel hypodense lesions in brain parenchyma.<sup>2,6,7</sup>

The exact mechanism of cerebral injury causing transient cortical blindness remains unknown. Disruption of blood brain barrier with direct contrast neurotoxicity has been postulated as potential mechanism in many reports.<sup>8,9</sup> A relationship between cortical blindness and hypertensive encephalopathy, also termed as posterior reversible leucoencephalopathy syndrome, especially in patients with history of hypertension has been

suggested.<sup>2,10</sup> Hypertensive encephalopathy results from abrupt increase in blood pressure during or after angiography which exceeds the auto regulatory capacity of cerebral vessels producing regions of vasodilatation and vasoconstriction with breakdown of blood brain barrier and focal transudation of fluid.<sup>11</sup> The CT and MRI scans of these patients have shown symmetrically distributed areas of vasogenic edema predominantly within the distribution of posterior cerebellar circulation with complete clearance of edema within the next 24-48 hours in follow-up CT scans.<sup>2,5,6</sup>

Although our patient had a long history of hypertension but no acute rise was documented during or after the procedure. CT and MRI scans showed multiple hypodense lesions in both cerebellar regions and left occipital lobe suggesting recent infarcts. Similar multiple hypodense lesions in right occipital lobe, thalamus and left cerebellar hemisphere regions have been reported by Till et al on CT and MRI scans of their patient who developed transient cortical blindness without any abrupt rise in blood pressure following vertebral angiography.<sup>7</sup> Cortical involvement without subcortical white matter involvement is not normally associated with hypertensive encephalopathy; therefore, pathophysiological mechanisms of cortical blindness and hypertensive encephalopathy may not be related.<sup>12</sup> The exact explanation of multiple hypodense lesions in both cerebellar and left occipital hemisphere in our patient is likely to be of complex etiology. Cerebral microembolization have been detected by MRI diffusion-weighted imaging sequences in up to 5% of patients after left heart catheterization mostly via right upper limb arterial access, however, its clinical relevance remains unknown. Patients with silent cerebral infarcts remain asymptomatic and such events are not associated with transient cortical blindness.<sup>13,14</sup>

The highest incidence of transient cortical blindness has been reported after selective vertebral angiography and risk is higher with non-ionic contrast agent.<sup>8,15</sup> Most cases of transient cortical blindness have been reported after angiography of bypass grafts. As the internal mammary artery and vertebrate artery arise in close proximity, it is possible that in our patient excess volume of contrast travelled via the left vertebral artery to posterior cerebral circulation. Direct contrast neurotoxicity to the sensitive occipital cerebral lobes appears to be most possible explanation in our patient, although the exact mechanisms remain unknown.

In conclusion transient cortical blindness following coronary angiography is extremely rare but fully

reversible complication with gradual return of vision in an average of three days without any residual blindness and neurological deficit. CT scans are frequently used to confirm the diagnosis. MRI scans have consistently shown high intensity signals in brain parenchyma and can confirm the diagnosis where CT scans appear normal. There is no experience of re-challenge for graft study in patients with history of cortical blindness but it appeared to be safe for native coronary angiography in patients with history of cortical blindness.<sup>16</sup> Precautionary measures like decreasing the volume and different type of contrast may be considered for LIMA to LAD study in patients with history of cortical blindness.

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