CASE REPORT

Transient cortical blindness: A benign but devastating complication after coronary angiography and graft study

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Abstract

Transient cortical blindness after coronary angiography and bypass graft is a very rare complication. In this report we present the case of a 63-year-old man who developed transient cortical blindness within 30 minutes of coronary angioplasty and graft study, but subsequently recovered within 72 hours without any neurological deficit. A plain computed tomography brain scan showed bilateral symmetrical subarachnoid hyperdensities in the posterior cerebral circulation area suspicious of subarachnoid bleed. However, magnetic resonance imaging and magnetic resonance angiography scans were normal. Excess contrast volume causing direct neurotoxicity seems to be the most probable cause, but the exact mechanism is unclear.

Keywords: Coronary angioplasty, Bypass graft angiography, Transient cortical blindness, Contrast neurotoxicity.

Introduction

Transient cortical blindness is a very rare but benign, and devastating complication after coronary angiography and bypass graft study. It was first reported in 1970.[1] It is an uncommon complication with occurrence rates of 0.3% to 1.0% in cerebral and vertebral angiography, but when hyperosmolar iodinated contrast agents are used, the rates can be as high as 4%.[2-4]

The incidence of cerebrovascular complications in diagnostic cardiac catheterisation and coronary angiography from the National Institute of Health in America and British Cardiac Society have been reported as 0.03% and 0.06% respectively.[5] Though transient cortical blindness was first reported in 1970, to our knowledge after literature review, so far only 36 cases have been reported despite coronary angiography being the main tool for interventional cardiologists worldwide, and from our centre ever since the start of coronary intervention in 1982,this is the second case report of transient cortical blindness post-coronary angiography and bypass graft study.

Case Report

A 63-year-old man with hypertension, hyperlipidaemia, non-insulin-dependent diabetes mellitus was admitted electively for coronary angiogram and bypass graft study for recurrent angina. He had history of coronary artery bypass graft (CABG) surgery done in 2012 with three saphenous vein grafts(SVG) and left internal mammary artery graft (LIMA).Angiography of native coronary artery was performed without any difficulty via the right femoral artery approach, using 6F right and left diagnostic Judkins catheters. However, the SVG using Judkins right (JR) number 3.5 and 4 were unable to locate, so a 6F pigtail catheter with injector was used, and it noted that none of the SVG was patent. Engagement of LIMA was a bit difficult with JR 3.5, 6F diagnostic catheter resulting in selective

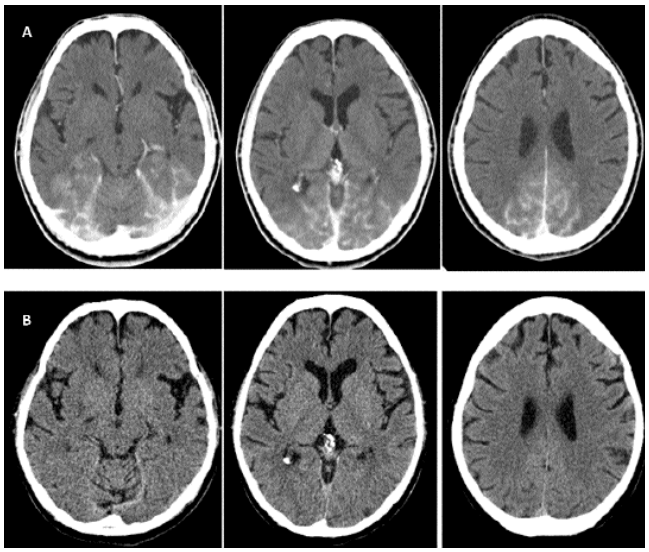

Figure: (A) Cranial computed tomographic scan done within 30 minutes after coronary angioglasty showed marked bilateral symmetrical subarachnoid hyperdensities in the posterior cerebral circulation area. (B) A repeat CT scan done after complete recovery of vision (72 hours) showed complete resolution of hyperdensities in the same vascular territory.

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engagement of left vertebral artery for a couple of times. Eventually LIMA was engaged selectively with 6F LIMA catheter which showed no obstruction. Angioplasty of short chronic total occlusion (CTO) occlusion of native left circumflex artery was performed without difficulty and throughout the procedure there was no complication. A total of 250mL of non-ionic, water-soluble contrast (Iopamiro 370,Bracco S.p.A, Ferentino, Italy) was used.

Upon transferring the patient to the recovery room, within 30 minutes of the procedure, the patient experienced blurring of vision and rapidly deteriorated to complete blindness. His blood pressure was 138/82mmHg with pulse rate of 78 beats per minute with regular rhythm, and there were no associated symptoms. His higher mental function was normal, pupils were equal and reactive to light bilaterally, extra-ocular range of movements were normal, no nystagmus, no papilloedema or retinal changes, and he had no other neurological deficit. The clinical findings were consistent with cortical blindness. Differential diagnosis such as embolic stroke, dissection of the aortic arch and its branches, spasm of cerebral vessels and in-situ thrombosis of cerebral vessels were considered.

His activated clotting time (ACT) was 217 seconds. Bedsides, echocardiography showed normal left ventricular ejection fraction (LVEF) and no clots. A non-contrast enhanced computed tomography (CT) scan of brain was done immediately after the deterioration of his vision showed bilateral symmetrical subarachnoid hyperdensities in the posterior cerebral circulation area, possibly a bleed (Figure-1A). In this case, bilateral subarachnoid bleed was unlikely, so a full sequence magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA) scans were performed immediately after CT brain and approximately one hour after the procedure. They showed no evidence of intracranial bleed or acute infarct. Twenty four hours post-procedure, the patient's vision gradually improved and fully recovered with no residual blindness within 72 hours. A repeat CT scan was completely normal (Figure-1B). A diagnosis of transient cortical blindness was made and he was discharged after 96 hours. His follow-up at two weeks post-procedure, he had no complaints with his vision or angina.

Discussion

Transient cortical blindness, first reported in 1970 following coronary angiography, is an uncommon complication of cerebral and vertebral angiography. The exact mechanism of cerebral injury remains unclear and diagnosis remains mainly clinical in patients presenting with rapid deterioration of vision during or after coronary angiography.[1-5] Unlike in our patient, there are reports of associated neurological deficit and CT scan findings vary from normal to contrast enhancement in occipital lobes and multiple hypodense lesions in brain.[4,5-8]

The definite cause of cerebral injury causing cortical blindness remains questionable. There are a number of possible explanations, and it has been reported that cerebral injury may be related to contrast-induced hypotension during the procedure, to the hypertensive vascular changes or to the type and the amount of contrast used.[4] Disruption of blood brain barrier due to direct neurotoxic effect of contrast media has been postulated in several reports.[2,9]

Hinchey et al. described the relationship between cortical blindness and hypertensive encephalopathy, also known as posterior reversible leucoencephalopathy syndrome, affected mainly patients with underlying hypertension, immunosuppression and renal impairment.[10] Hypertensive encephalopathy is a result of sudden increase in blood pressure during or after the angiography which disrupts the autoregulatory mechanism of cerebral arteries, causing regions of vasoconstriction and vasodilatation with breakdown of blood brain barrier and focal transudation of fluid.[11] Imaging studies in these patients have shown bilateral symmetrical vasogenic oedema in occipital lobes and often extending into cortical surface and follow-up imaging studies in the next 24-48 hours have confirmed complete resolution of oedema.[6,7,12]

Even though our patient was hypertensive for many years, it was well-controlled and it was documented there was no rise or drop in blood pressure during or after the procedure. The CT findings were similar to imaging in hypertensive encephalopathy, but they were against the diagnosis as our patient's blood pressure remained within normal range throughout and after the procedure and, most importantly, his MRI and MRA were normal. He was not immunosuppressed nor had renal impairment.

The disruption of blood brain barrier due to hypertonicity or to the volume of the contrast is still unclear. Several investigators have reported using 80-400mL of contrast.[1-4] The newer non-ionic agents are supposed to be less toxic compared to the ionic agents, yet a few cases of transient cortical blindness have been reported with the use of non-ionic agents and most of them during angiography or bypass grafts.[4,12] The highest incidence has been reported after selective vertebral angiography.[9,13] In our patient, the use of 250mL of contrast and difficulty in engaging LIMA as the internal

mammary artery and vertebral artery arose from close vicinity. It is likely that the excess volume of contrast might have reached the posterior circulation, causing direct neurotoxicity. However, the exact mechanism is unknown. The prolonged supine posture might also play a key role in enhancement of contrast to occipital region.[6]

Finally, Rama et al. reported a case series of three patients where re-challenge did not produce recurrence during native coronary angiography,[14] but there is no experience of re-challenge in graft study.

Conclusion

Transient cortical blindness is an uncommon complication of a common procedure which is completely reversible within 72-96 hours without any residual neurological deficit. CT scan, MRI and neurological consultation are indicated to confirm the diagnosis. It may be a devastating complication for the patient, the family and to the medical staff involved. Reassurance and a good rapport with the patient and family members are critically important.

Reference

1. Fischer Williams M, Gottschalk PG, Browell JN. Transient cortical blindness. An unusual complication of coronary angiography. Neurology 1970; 20: 353-5.
2. Horwitz NH, Wener L. Temporary cortical blindness following angiography. J Neurosurg 1974; 40: 583-6.
3. Studdard WE, Davis DO, Young SW. Cortical blindness after cerebral angiography. Case report. J Neurosurg 1981; 54: 240-4.
4. Kinn RM, Breisblatt WM. Cortical blindness after coronary angiography: a rare but reversible complication. Cathet Cardiovasc Diagn 1991; 22: 177-9.
5. 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: 917-9.
6. Yazici M, Ozhan H, Kinay O , Kilicaslan B, Karaca M, Cece H, et al. Transient Cortical Blindness after cardiac catheterisation with lobitridol. Tex Heart Inst J 2007; 34: 373-5.
7. Parry R, Rees JR, Wilde P. Transient cortical blindness after coronary angiography. Br Heart J 1993; 70: 563-4.
8. Till V, Koprivsek K, Stojanovic S, Avramov P, Vulekovic P. Transient cortical blindness following vertebral angiography in a young adult with cerebellar haemangioblastoma. Pediatric Radiology 2009; 39: 1223-6.
9. Lantos G. Cortical blindness due to osmotic disruption of blood brain barrier by angiographic contrast material: CT and MRI studies. Neurology 1989; 39: 567-71.
10. Hinchey J, Chaves C, Appignani B. A reversible posterior leucoencephalopathy syndrome. N Engl J Med 1996; 334: 494-500.
11. Schwart RB, Jones KM, Kalina P, Bajakian RL, Mantello MT, Garada B, et al. Hypertensive encephalopathy: Findings on CT, MR imaging, and SPECT imaging in 14 cases. Am J Roentogenol 1992; 159: 379-83.
12. Lim KK, Radford DJ. Transient cortical blindness related to coronary angiography and graft study. Med J Aust 2002; 177: 43-4.
13. Kermode AG, Chakera T, Mastaglia FL. Low-osmolar and non-ionic x-ray contrast media and cortical blindness. Clin Exp Neurol 1992; 29: 272-6.
14. Rama BN, Pagano TV, Delcore M, Knobel KR, Lee J. Cortical blindness after cardiac catheterization: Effect of re-challenge with dye. Cathet Cardiovasc Diagn 1993; 28: 149-51.