

Case Report

Transient cortical blindness after coronary angiography: a case report and literature review

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Abstract

Transient cortical blindness after coronary angiography is a rarely encountered, rapidly progressive complication with complete return of vision within hours to up to five days. Patients at risk include those undergoing coronary artery bypass graft study and those with renal failure. Although, the exact mechanism is not clear, the most likely explanation is a breakdown of blood brain barrier with direct neurotoxicity of the contrast media as most reported patients underwent coronary bypass graft study. We report a case of transient cortical blindness following diagnostic coronary angiography in a 39-year old patient with spontaneous recovery of vision within 1 hour.

Keywords: Cortical blindness, transient, coronary angiography.

Introduction

The incidence of cerebrovascular complications in diagnostic cardiac catheterizations is low and usually includes transient ischaemic attacks, strokes and amaurosis fugax.¹ Transient cortical blindness after coronary angiography was first reported in 1970.¹ It is a rare complication, considering the widespread and frequent use of coronary angiography world wide. Less than twenty cases have so far been reported in English literature and the majority of these involved coronary artery bypass graft study.^{2,3} This has led to the hypothesis that it is the breaching

of the blood-brain barrier by the radiographic dye that may lead to this rare outcome. We report a case of transient cortical blindness following diagnostic coronary angiography in a patient with hypertrophic cardiomyopathy with spontaneous rapid recovery. We believe this is the first report of such case from Pakistan.

Case Report

A 39 years old man underwent coronary angiography due to symptoms of unstable angina. He was a diagnosed case of hypertrophic cardiomyopathy (HCM), dyslipidaemia and had family history of premature coronary artery disease. The angiography was done via right femoral artery using 6 French arterial sheath. Judkins catheters (JL4+JR4) were used to cannulate the coronary arteries. Left ventriculogram was not done in view of findings in 2D echo. His coronary angiography showed severe three vessel coronary artery disease. The procedure lasted 15 minutes during which the patient remained haemodynamically stable. Around 80 ml of non-ionic, hypo-osmolar contrast medium "Iopamiro-370" (Iopamidol) was used. This was patient's first exposure to any contrast medium.

Just after angiography, while still on the catheterization table, the patient complained of blurring of vision which within 15 minutes progressed to complete loss of vision in both eyes. He was awake, alert and oriented. Immediate neurology consult was sought. Examination by the neurologist revealed pupils of 2.5 mm, equal, symmetric

and reactive. He had normal eye movements and normal fundi. Vision was bilaterally limited to bare perception of light. Other cranial nerves were normal. Motor and sensory examination was unremarkable. Reflexes were normal and toes were bilaterally down going. The initial diagnosis was top of the basilar artery syndrome, caused by thromboembolism of posterior cerebral circulation. His immediate CT scan of brain (without contrast) showed residual contrast from recent angiography. It showed no features of haemorrhage or infarct. The patient was treated with anti-platelet therapy and intravenous heparin. Emergent cerebral (vertebral) angiogram was done to evaluate for thromboembolism and to decide about the need of thrombolytic therapy. It showed normal basilar artery and branches, with no occlusion, cut-off or vessel narrowing. While diagnostic work-up was ongoing, the patient started regaining his vision and within 1 hour of onset of symptoms, he recovered his eye sight completely. There was no residual visual deficit.

Discussion

Transient cortical blindness is a rare but recognized complication of coronary angiography. It may start during the procedure or immediately afterwards and return of vision has been reported within minutes up to 5 days.² Our patient regained his vision approximately 60 minutes after the angiography with no residual visual impairment or neurological deficit. The differential diagnosis of the visual impairment after cardiac catheterization includes thromboembolism (posterior cerebral artery/top of the basilar artery syndrome), vasospasm of the posterior cerebral arteries, contrast-induced hypotension, hysterical blindness and contrast-induced cortical blindness.²

Several theories have been proposed regarding the possible pathophysiology of contrast induced transient cortical blindness. Most widely accepted amongst them is that of the direct neurotoxic effect of the contrast agent on the blood-brain barrier in the occipital lobe.¹ CT brain in some of these cases has revealed intracranial enhancement by contrast in the occipital region. It may be due to prolonged supine posture during coronary angiography. The posterior cerebral circulation is known to be more susceptible to such injury and this may relate to differences in sympathetic innervations.^{4,5} Demirtas et al suggested an immunological mechanism,⁶ while according to Kwok et al direct idiosyncratic neurotoxicity is the most plausible explanation, although the exact mechanism remains elusive.⁷

Risk groups include patients with impaired renal function leading to decreased clearance of the contrast agent, and patients with LIMA bypass.² The risk increases when hyperosmolar iodinated contrast agents are used, however, it can also occur with newer hypo-osmolar and non-ionic

radiographic contrast media, and this potential complication is indicated in the product information.³ Hypertrophic cardiomyopathy (HCM) is associated with a diffuse coronary microvascular dysfunction which is in line with the autopsy evidence of widespread remodeling of the intramural coronary arteries.⁸ To our knowledge, previously no case has been documented with HCM and it needs to be studied if this microvascular dysfunction is systemic or limited to coronary arteries only.

Yazici et al have compiled a total of 18 published case reports of transient cortical blindness including 7 after native coronary vessels and 11 after coronary artery bypass graft study.³ Franzl² presented the clinical data of the 15 reported cases at Europrc 2006 and showed male predominance with the mean age of 54 ± 7.6 years, our patient being the youngest (39 years). More than half (9 patients) in his data set had known systemic hypertension. Six had angiography of left internal mammary artery grafts. There was one patient with impairment of renal function. Nine patients had history of previous contrast exposure. Volume of contrast injected ranged from 75 ml to 400 ml. Different patients took different time to recover their normal vision, ranging from 15 min to 5 days. Our patient, a normotensive, with normal renal functions and no previous exposure to contrast medium was injected 100 ml of a non-ionic, hypo-osmolar contrast medium Iopamidol. He recovered his normal vision in 60 minutes.

The clinical dilemma in this case is regarding re-injection of contrast medium; especially if coronary angioplasty is needed. The only evidence supporting re-injection of contrast medium in such patients comes from a case series of three patients where re-challenge did not produce recurrence.⁹

Our patient also had no recurrence of visual symptoms with re-exposure during cerebral angiogram. Nevertheless minimizing the amount of dye used and pretreatment with corticosteroids is advised before re-exposure.²

Conclusion

Transient cortical blindness is a rarely encountered, rapidly progressive complication following coronary angiography. It may even start during the procedure with return of vision within hours but may take up to 5 days for complete recovery. Although, exact mechanism is not known, a breakdown of the blood-brain barrier with direct neurotoxicity of the contrast media seems to be the cause. Reassurance to the patient is very important as despite its devastating nature, the deficit resolves completely in all patients as long as the etiology is contrast induced cortical blindness. Cerebral embolism and haemorrhage have to be excluded by CT scan or MRI studies before making the

diagnosis of contrast-induced visual loss.

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