

Transient cortical blindness can also be seen in the absence of contrast enhancement at computed tomography scan after coronary angiography

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ABSTRACT

Transient cortical blindness after coronary angiography or angioplasty is a rare complication. Patients with aortocoronary bypass grafts such as internal mammary artery have a separate risk factor probably because of the direct contrast injection to the vertebral artery during the catheterisation of the internal mammary artery ostium. In most cases, computed tomography scan revealed typical, symmetrical contrast enhancement in both occipital lobes. Nevertheless, transient cortical blindness can be seen after coronary angiography without contrast enhancement at computed tomography scan.

KEY WORDS: Internal mammary artery graft, Coronary angiography, Transient cortical blindness.

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INTRODUCTION

The incidence of cerebrovascular complications during coronary angiography is so low.¹ One of them is transient cortical blindness (TCB). TCB is characterised by a loss of visual perception whilst ocular motility and pupillary response are preserved intact.² It was firstly reported in

neurological procedures, and it has rarely been seen after coronary angiography.^{3,4} TCB usually develops during or just after procedure. The pathophysiology is largely controversial. Patients with an coronary artery bypass graft, reduced glomerular filtration rate and undergoing extended procedures are more prone to CB. Although the condition seems too catastrophic, the outcome is generally favorable, with spontaneous return of sight within a few days. Herein, we report our experience of a patient with TCB developed one hour after CAG and resolved spontaneously at 24 hour after the procedure.

CASE REPORT

A 56-year-old ex-smoker man was admitted to the hospital because of typical chest pain. The patient underwent coronary artery bypass grafting (left internal mammary to left anterior descending artery and right internal mammary artery to first obtuse marginal) six years ago and stent implantation to right coronary artery was performed two years ago. An admission 12-lead electrocardiogram (ECG) showed minimal ST depression in inferior leads, and then, patient was transferred to the coronary

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intensive care unit. Elective coronary angiography was performed. CAG revealed significant instant restenosis in RCA. Balloon angioplasty for instant restenosis in RCA was performed successfully. Nevertheless, significant stenosis was still present before the proximal end of the previous stent and a second 4.0x20 mm bare metal stent was deployed successfully. Despite the successful catheterisation of native coronary artery vessels, the catheterization of the left and right IMA was prolonged owing to technical difficulties. After several attempts, we succeed to view the left and right IMA, and they were patent.

During the all procedure, 300 mL of non-ionic low-osmolar contrast agent (Omnipaque™ 350 mg/mL) was used and the patient did not report any complaint. Also, his vital parameters were within normal ranges. Standard biochemical tests including renal function were normal pre- and post catheterization. However, one hour after procedure the patient complained of bilateral complete lack of vision. Examination of neurological system and fundi was normal. Non-contrast cranial CT scan was performed to determine whether a microembolic event or cranial hemorrhage were present or not. The non-contrast cranial CT scan demonstrated no evidence of cerebral haemorrhage or ischaemia. The repeat non-contrast cranial CT scan at 12 hours also showed no evidence of cerebral haemorrhage or ischaemia. Thus, we suggested that TCB occurred probably because of the local disruption of the blood-brain barrier by contrast agent, especially in occipital lobe. However, cranial CT did not demonstrated marked bilateral contrast enhancement in the occipital lobes. Subsequent hydration with normal saline was performed. The patient's vision spontaneously returned at 24 hours after CAG and completely recovered within 48 hours. The patient was discharged at a very good condition. The patient had free of residual blindness under at his 6-month follow-up control.

DISCUSSION

TCB after coronary angiography and angioplasty is not so frequently reported. The estimated incidence after CAG is of 0.05%.^{3,4} It is more common after cerebral and vertebral angiography. In most cases, the loss of vision usually starts during the procedure or in the following 12 hours and progresses rapidly after onset. Also, some symptoms such as severe headache, vomiting, loss of co-ordination, limb weakness, aphasia and confusion can attend.⁵

The exact mechanism of this phenomenon remains unclear. Many explanations have been suggested. These are local disruption of the blood-brain barrier with direct neurotoxicity of the contrast agent, microembolization into the brain, sudden increase in systemic blood pressure or contrast-induced hypotension during the procedure, hypoxia and cerebral oedema.⁶⁻¹¹ Among them, the most likely mechanism is the local disruption of the blood-brain barrier by the contrast agent and an increase in vascular permeability, mostly in the occipital areas.¹² All contrast agents can be associated with this complication and TCB does not seem to be volume dependent.¹³

Patients underwent coronary artery bypass graft operation, especially IMA grafts, have separate risk factor for developing TCB. The direct injection of the contrast agent into the vertebral artery during catheterisation of the IMA ostium and the need for a higher amount of the contrast agent because of technical difficulties can explain the reason for local disruption of the blood-brain barrier in these patients.⁹ Nevertheless, our patient had also left and right IMA grafts, and we also had some technical difficulties during catheterisation of the IMA grafts. The occurrence of cortical blindness seems to be a serious clinical problem (whether transient or permanent). Fortunately, the outcome is generally favorable. The vision usually returns spontaneously within hours to up to five days.⁵ Recurrence following a repeated procedure has never been reported. In most cases, CT scan revealed typical, symmetrical contrast enhancement in both occipital lobes.^{2,5,14} CT findings returns to normal within 48-72 hours when the contrast agent has been excreted. However, there was no marked bilateral contrast enhancement at CT scan in our patient. In this regard, our case report is a worthy one demonstrating that TCB might occur after coronary angiography without contrast enhancement at CT scan. Also, there is no requirement for specific therapy. Pre- and postprocedural *hydration* may have a protective effect from contrast-media-associated toxicity in high-risk patients.

In conclusion, the circumstances leading to TCB should be well-understood and precautions such as the use of low osmolality agents in reduced amounts, hydration pre- and post catheterization in high risk patients should be considered, especially in catheterisation of the IMA grafts. Also, it should be kept in mind that TCB can be seen in the absence of contrast enhancement at CT scan.

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