

Transient cortical blindness after coronary artery bypass surgery

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Introduction

Transient cortical blindness (TCB) is an infrequent and intriguing postoperative complication following cardiac surgery. It is characterized by the sudden and temporary loss of vision in both eyes, usually resolving within hours (1). By reporting this rare occurrence of TCB after coronary artery bypass grafting (CABG) surgery, we aim to raise awareness among clinicians and researchers about this potential complication.

Case

A 54-year-old non-diabetic woman complaining of chest pain with a history of myocardial infarction (MI) and percutaneous coronary intervention about 13 years ago was evaluated. Her drug history included nitroglycerin, atorvastatin, and, aspirin. Before the operation, her ejection fraction was 40% and the carotid Doppler showed no abnormality. Also, angiography revealed 80%, 90%, and 60% stenosis in the left anterior descending (LAD), diagonal artery, and right coronary artery respectively. Bypass grafts were implanted as the left internal mammary artery to the LAD artery, the saphenous vein to the obtuse marginal and ramus artery, however, right coronary artery bypass grafting was incapable. During surgery, the blood runoff was good. Additionally, iodinated contrast agent was used and the time of the operation and aortic clamp duration were 128 minutes and 70 minutes respectively.

The patient was asymptomatic after the operation but started to have bilateral blurring of vision three days after the surgery, which worsened drastically over 2 weeks. After this period, she only was able to perceive light bilaterally and her vision improved gradually for 2 months. After the surgery, vital signs were good, had no change of electrocardiogram or enzymes, and cardiac status was stable. The ophthalmology examination detected no causative ocular damage except bilateral papilledema and reduced visual acuity at the level of light perception. So, as the initial management, intravenous hydrocortisone was administrated (100 mg bd for three days). A non-contrast computed tomography (CT)-Scan revealed bilateral subarachnoid hyperdensities in the occipital and parietal lobes. Hence to confirm cerebral hemorrhage, magnetic resonance imaging (MRI) was done which showed no evidence of hemorrhage. The evaluation of related arteries (carotid, vertebral and ophthalmic) was completely normal. Nevertheless, the patient remained under close observation with no additional intervention. Finally, her vision completely recovered after two months and remained with no other complications during the further follow-up.

Discussion

TCB is an uncommon but potentially severe and stress-full complication that has been mainly associated with cerebral and vertebral angiography (2, 3). Reports on TCB following CABG have also existed in the literature since the 1990s (4, 5). The pathophysiology of TCB remains incompletely understood, but several proposed mechanisms may shed light on its occurrence. Hyperosmolar iodinated contrast agents have been

associated with TCB occurrences (1). Contrast agents may induce a disruption of the blood-brain barrier, leading to the infiltration of the contrast in to the brain parenchyma. This localized infiltration, particularly affecting the occipital lobes, may result in an inflammatory response, potentially contributing to the sudden onset of cortical blindness (6).

It is worth noting that not all cases of TCB involve hyperosmolar iodinated contrast agents. Reports have shown that non-ionic contrast agents, which are considered less osmotically active, have also been associated with TCB incidents (7). Although the use of non-ionic and hypoosmolar contrast agents was expected to mitigate the risk of TCB, it is evident that this measure does not provide complete prevention (7). This discrepancy suggests that factors beyond the osmolality of contrast agents may be involved in the development of TCB.

In the presented case, bilateral subarachnoid hyperdensities in the occipital and parietal lobes were observed in the non-contrast CT scan. However, subsequent MRI findings did not show any pathological abnormalities. This inconsistency raises questions about the nature of the observed hyperdensities and their relation to transient cortical blindness. A similar case reported by Zhen-Vin Lee also displayed acute subarachnoid bleeding in both occipital lobes on a CT scan but a normal MRI, further emphasizing the transient nature of these changes and the lack of lasting damage associated with TCB (8). This highlights the importance of MRI in the work-up of these patients, however, the diagnosis of TCB remains to be based on clinical presentation mostly. The prognosis for TCB is generally favorable, with most cases resolving within hours, as observed in this patient (9). In conclusion, clinicians should be vigilant about the possibility of TCB following coronary angiography and CABG procedures. Additionally, patients undergoing these procedures should be informed about the potential risk of TCB, and close monitoring for signs and symptoms of this condition is advised.

Ethical Approval

The patient signed an informed consent form.

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