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Transient Cortical Blindness Following Coronary Angiography

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Abstract

Coronary angiography (CA) became the golden tool for coronary artery disease (CAD) management. Transient cortical blindness following coronary angiography (TCBCA) is a rare complication of CA, in comparison with the other common complications. The neurotoxic effect of contrast medium is the possible cause of this clinical phenomenon, by disrupting blood brain barriers (BBB). TCBCA usually occurs during or immediately after CA. The clinical picture includes loss of vision that progressively resolves within hours or days. Ophthalmologic examination reveals normal findings, while computed tomography scan (CT scan) may show contrast accumulation in the occipital area. No current clinical protocols to manage this condition, except of excluding other possible causes of blindness after CA. Further well-organized studies are recommended to understand this clinical phenomenon, and more effort is needed to set a variety of interventional strategies regarding TCBCA management.

Keywords: Coronary angiography; Arrhythmia; Myocardial infarction; Nephropathy; Neurotoxic effect

Introduction

Coronary angiography (CA) is the headmost clinical tool for both diagnosis and intervention of coronary artery disease (CAD), and therefore, CA procedures occupy a huge space in modern healthcare practices. At the same time, the recurrent complications which accompany CA are extensively investigated. Such these complications are allergy to contrast, infection, nephropathy, embolism, local vascular injury, great vessel dissection, arrhythmia, myocardial infarction, and even death [1].

Transient cortical blindness following coronary angiography (TCBCA) is scarcely documented in the literature, and poorly studied. TCBCA is rare and less frequent; however, it has a devastating effect on patients who have already finished their CA. In this paper, a thorough investigation of this phenomenon and a comparison review of the available published literatures that specifically discuss TCBCA.

CA is defined as "the radiographic visualization of the coronary arteries after direct opacification with contrast media" [2]. While cortical blindness is defined as visual impairment or loss, despite normal ophthalmologic examination findings regarding eye structures, pupillary characteristics and responses [3].

Early recognition of TCBCA was in 1970 by Fischer-Williams and his colleagues. Fischer-Williams and co-workers published their paper in effort to explain the differences between the hysterical blindness that rarely follows CA patients, and the TCBCA, which was unusually encountered in the same patients [4] .

In fact, the incidence of TCBCA is not well known. However, one study suspects that up to 0.45% of all CA patients develop TCBCA [5]. Furthermore, the majority of the published literatures are case-report papers, rather, surveillance or national survey studies.

Many hypotheses tried to explain the pathophysiological process that explains the development of TCBCA. Contrast medium which is used in CA procedure has a neurotoxic effect on blood-brain barriers (BBB), especially on the occipital cortex [6], which has a selective vulnerability for contrast medium [7], and that leads to reversible visual impairment or loss. Computed tomography scan (CT scan) for TCBCA patient shows accumulation of contrast medium in this anatomical area [8]. Keeping in the mind the nature of CA procedure that requests the patients to stay at supine position during the procedure and the few hours follow [9], which supports the previous hypothesis.

Using an ionic contrast or contrast with high osmolarity in CA increases the risk of developing TCBCA [10], but still, few cases have been reported with non-ionic or low osmolarity contrast [11]. Moreover, volume of the contrast agent may play a significant role in the disease severity, particularly, when the volume of the introduced medium is more than 80 ml [12].

Schwartz et al. suggest the hypertensive encephalopathy as possible explanation of TCBCA. Hypertensive encephalopathy is a sudden increase of blood pressure during or after the CA, that interrupts the autoregulation of cerebral arteries, especially the occipital area, which in turn forms vasodilation and local congestion, then rapidly, leads to disruption of BBB and leak of fluid to the occipital lobe, and finally forming edema that reaches to cortical surface [13].

The immunological mechanism is another proposed explanation of how TCBCA develops [14]. The endothelins, specifically, ET-1, ET-2 and ET-3 increase the brain endothelial cells permeability, and the consequent development of cerebral edema [15]. Endothelins are strong vasoconstrictors and are released from the endothelial cells, as well as other organs, including the brain, lungs and kidney. It is believed that these chemical substances are released in some pathological circumstances also, such as developing sensitivity to contrast agent during CA procedure.

Typically, symptoms of TCBCA start during CA, or the few hours after the procedure, and last for 48 hours [16], but it may rarely extend

for five days [17]. Clinical picture improves as BBB restore its protective function. Patients start to return normal visual function gradually; first light perception followed by color vision, and eventually full sight and pre TCBCA vision status [18].

Vulnerable patients such those with impaired renal function have a higher risk for developing TCBCA, because of the decreased ability of kidney to clear the contrast medium [17]. Other risk factors include conditions with BBB disruption, as seen in eclampsia and using of immunosuppressive substances [18].

Alp et al. stated that neurological examination reveals normal findings, particularly, cranial nerve, sensory and motor function [19]. In the same study by Alb and co-workers, they found normal extra ocular movements and pupillary reflexes when applying ophthalmological examination. On the other hand, positive findings include visual field defect bilaterally, that sooner became total blindness, and hypertensive retinopathy when fundi examination was carried out. In conclusion, bilateral cortical blindness is the main clinical picture, rather than, central neurological deficit.

CT scan and magnetic resonance image (MRI), in addition to the routine neurological and ophthalmological examinations are useful to confirm TCBCA. Indeed, these usual diagnostic techniques are essential, and used usually to exclude other possible causes of visual impairment after CA. Other possible causes of the blindness after CA include hysterical blindness, hypotension, embolism, or cerebral arteries vasospasm [17]. Considering these differential diagnoses is crucial before giving final decision about the disease.

Restoring normal vision is the ultimate goal for TCBCA management, and current therapy tends to exclude major cerebral focal injury, as the worst possible etiology for developing blindness after CA procedure. Meanwhile, no specific treatment is tested or recommended for this type of blindness, but fast resolution of visual impairment is an agreed outcome. The available recommendations suggest maintaining normal blood pressure and enhancing diuresis to accelerate contrast medium clearance [20].

Fortunately, TCBCA has a favorable prognosis, and most patients restore their normal vision after 24-48 hours of incidence [6]. Although, the unpredicted and disabling nature of this condition should be the counted one, rather than how minor is the percentage of TCBCA incidence among CA patients.

Review of Literature

Current knowledge regarding TCBCA is scarce despite numbers of exploring efforts that have been made to understand the possible factors that sit behind this phenomenon. In addition, modern healthcare practices do not provide a clear intervention to manage such complication, or formulate a well-planned protocol to prevent or even minimize the occurrence of TCBCAC.

The majority of recent studies about TCBCA address the temporary and reversibility nature of this clinical phenomenon [20-23].

Giving the surprising and non-predicting effect of TCBCA on CA patients, this clinical problem holds a devastating impact on both patients and healthcare providers. TCBCA may develop immediately as the patient still on the catheterization table, and may remain as less as only one hour [21]. But in the other hand, patients with renal failure are susceptible for longer period of TCBCA because of their decreased clearance of contrast material, and this period of vision disability may extend to five days [17].

In the same study, Frantz observed 12 cases of left internal mammary artery (LIMA) bypass graft surgery that have developed transient cortical blindness after operation. Frantz's explanation of this clinical event is the possible pass of contrast medium during LIMA bypass graft surgery to left vertebral artery, because of its proximity to left subclavian artery, and then to the posterior circulation.

Yazici et al. have claimed that the type and amount of administered contrast may play a significant role in the development and severity of TCBCA, particularly, hyper-osmolar or ionic contrast medium which poses a greater risk of BBB disruption, in contrast with hypo-osmolar or non-ionic contrast medium. Usual amount of contrast in this procedure ranges from 80 ml to 400 ml, however, the same patients in Yazici's study have received smaller amount of contrast (75 ml), and even though, they developed TCBCA [18].

Further discussion about this phenomenon is built to exclude other possible causes of blindness after coronary angiography, which addresses more clinical challenges in diagnosing and managing this phenomenon. TCBCA has the clinical picture of post CA embolism, and therefore, careful radiology examination as CT scan is necessary [21]. CT scan procedure can provide the clinical judgment about whither there is a contrast enhancement in the occipital lobes and the antecedent cause of TCBCA, or cerebral embolism [19].

Other researchers have reported transient cortical blindness in different procedures; few cases have been documented after vertebral angiography [24]. Patients who undergo CT with contrast [25], spinal surgery [26], and cerebral angiography [27] have a comparable incidences of and similar manifestation.

Conclusion

This clinical focus paper about TCBCA demonstrates the remarkable gap in current clinical knowledge and practice regarding this rare phenomenon. More research efforts are on demand to achieve better understanding of this clinical complication, and therefore, healthcare providers will not rely only on limited experiences and recommendations. Moreover, proper protocols and guidelines for patients who undergo CA can minimize occurrence of TCBCA, or even prepare patients and healthcare providers for such unpredictable and devastating vision disability. Finally, nurses who take the role of first line contact with CA patients should have a clear assessment, interventional and teaching skills for patients who may develop TCBCA.

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