Case Report

Transient Cortical Blindness Following Intracardiac Repair of Congenital Heart Disease in an 11-year-old Boy

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Abstract

Postoperative blindness (PB) primarily involves reception and conductance parts of the visual pathway due to ischemia following cessation of blood supply, for example, retinal vascular occlusion. Although a rare cause of PB, cortical blindness (CB), which results from ischemia/infarction of visual cortex, has a poor outcome due to its mostly nonreversible nature. Ischemic optic neuropathy is the most common cause of PB following cardiac surgeries. CB following cardiac surgeries involving cardiopulmonary bypass has been rarely reported. Only a few of those articles reported partial or complete reversal of CB. We report an incidence of transient CB in an 11-year-old child who was operated for double chambered right ventricle with ventricular septal defect.

Keywords: *Cardiopulmonary bypass, cortical blindness, microembolization, postoperative blindness*

Introduction

The majority of reported postoperative (nonocular surgery) blindness cases were caused by retinal vascular occlusion or ischemic optic neuropathy.[1] "Cortical blindness" (CB) which is characterized by loss of vision due to transient or permanent dysfunction of the occipital cortex, has been rarely reported as a postoperative complication following cardiac surgery involving cardiopulmonary bypass (CPB). Meticulous vigilance is required to avoid this grave complication during cardiac surgery involving CPB which is often associated with hypotension, hemodilution or microembolization that may cause hypoperfusion of occipital cortex leading to ischemia/infarction. Postoperative blindness (PB) often goes unnoticed in pediatric patients because of their inability to express the event leading to delay in diagnosis. We report a case of transient CB due to areas of infarction in occipital lobe following intracardiac repair of congenital heart defects under CPB.

Case Report

An 11-year-old boy was admitted to our hospital with the complaint of shortness of breath. Two-dimensional echocardiography revealed a large malaligned perimembranous ventricular septal defect (VSD) with bidirectional shunt. A low infundibular and right ventricular outflow tract (RVOT) muscle bundle was detected causing severe RVOT obstruction with a maximum pressure gradient of 64 mmHg. Right coronary cusp prolapse was present causing mild aortic regurgitation. Trivial tricuspid regurgitation was present. The other important findings were: situs solitus, levocardia, normal systemic and pulmonary venous return, good sized and confluent branch pulmonary arteries, intact interatrial septum, atrioventricular-ventriculoarterial concordance, good biventricular function, left arch, absent major coronaries surrounding RVOT, absent coarctation of aorta and absent persistent left-sided superior vena cava (SVC).

The patient was diagnosed with "double chambered right ventricle (RV) with VSD." An intracardiac repair comprising of RVOT bundle resection, pulmonary valvotomy (pulmonary valve was preserved), and "Sauvage Dacron" patch closure of VSD were done under standard general anesthesia technique. Intraoperative course was uneventful. Systemic cooling to 30°C along with del Nido cardioplegia (500 ml) at 3°C were used for myocardial protection. For an institution of CPB 16Fr DLP aortic, 16Fr DLP (SVC) and 18Fr DLP (inferior vena cava) cannulas, Minimax Plus®

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Oxygenator (Medtronic), PerfX® arterial filter with 40 μm Polyester screen filter efficiency (B. L. Lifesciences) were used. Mean perfusion pressure was maintained around 50-60 mm of Hg. Standard de-airing protocol was followed (de-airing through patent foramen ovale and aortic root suction). Total CPB and aortic cross-clamp time were 72 and 43 min. The patient came off CPB with moderate inotropic support (dobutamine at 5 μg/kg/min and adrenalin at 0.05 μg/kg/min). RV/left ventricle pressure ratio was found to be 0.6. Post-CPB heart showed normal sinus rhythm. The patient was shifted to Intensive Therapy Unit after on-table tracheal extubation. However, soon after regaining consciousness, he complained about the total loss of vision (without light perception) in both the eyes. A detailed ophthalmologic examination including direct and indirect ophthalmoscopy could not detect any ocular damage involving both the eyes. After consulting Neurologist and ophthalmologist, plain computed tomography (CT), and magnetic resonance imaging (MRI) of the brain were done on the same day. The CT [Figure 1] did not find any obvious acute focal abnormality. However, MRI brain [Figure 2] at different secretions revealed multiple regions of acute ischemic infarct in bilateral occipitoparietal and right frontal lobes. The T2-weighted image [Figure 3] at the level of the orbits shows normal signal within the optic nerves bilaterally. A diagnosis of CB was made and conservative medical management was adopted. A gradual recovery of vision was noted as on a postoperative day (POD) 1 he was able to identify light in both the eyes and on POD 3 he was able to detect hand movement in both the eyes. On POD 9, he was able to identify faces of his parents and known people. He was discharged from the hospital on POD 10 and was explained about the slow progress of visual recovery.

On follow-up visit after 1 week following discharge from the hospital, he was found to have hazy vision with poor comprehension, and his vision acuity was found to be 6/12 (with 'E' chart) in both the eyes. One week later, on the second follow-up visit, his vision was re-examined for acuity and was found to be 6/9 in both the eyes. However, difficulty in visual comprehension persisted. One month later, on the third follow-up visit, his vision was re-examined which revealed improved vision with the restoration of visual comprehension (6/9 both eyes with English alphabets and N6 in both eyes at 25 cm distance with Hindi alphabets).

Discussion

Although the most common cause of CB is cerebrovascular disease, cardiac surgery, and cerebral angiography are also considered as major causes.[2-6] CB following cardiac surgery may happen due to occipital cortex dysfunction through a variety of mechanisms, including hypoxia/anoxia from hypoperfusion, cerebral hemorrhage, blood, fat, and air embolism or combination of these factors. Cortical penetration of contrast media is the reason behind CB following cerebral or coronary angiography.[7] Despite taking all the precautions which includes maintaining optimum hematocrit and perfusion pressure during CPB period, preventing hypoxia, acidosis,

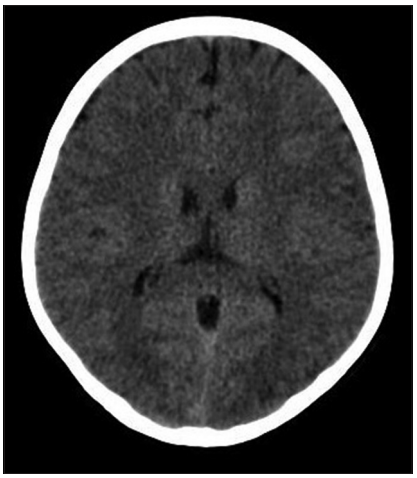

Figure 1: Computed tomography scan of brain showing normal signals of brain parenchyma including occipitopareital region

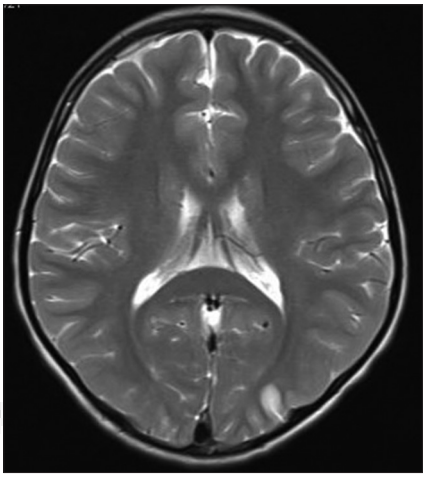

Figure 2: T2-weighted image of the brain shows increased signal within the occipital lobe suggestive of acute infarction

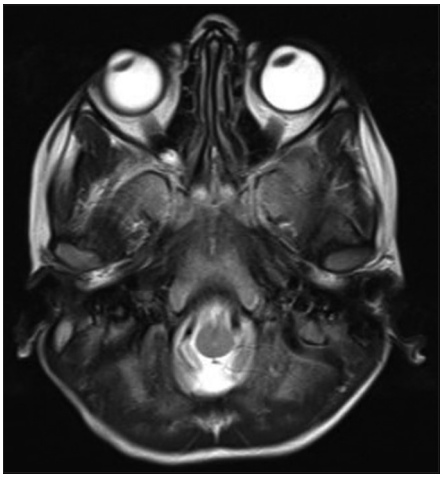

Figure 3: T2-weighted image at the level of the orbits shows normal signal within the optic nerves bilaterally

and hypotension our patient developed multiple infarctions in both occipitoparietal and right frontal lobe which probably indicates the incidence of microemboli shower. Although CPB was the most likely source of microemboli, manipulation of the aorta, cannulation and deairing techniques all are potential microemboli source. Using 40 μ filter or membrane oxygenator for CPB circuit do not give hundred percent protection against microembolization which is also independent of CPB duration.[8-10] The clinical findings after occipital infarction comprise loss of vision, homonymous visual field defect of various configurations, central vision, and color vision.[11]

Following CB the loss of vision may be permanent or transient.[12-15] The recovery of vision if happens may take variable period depending on various factors. Best prognosis has been observed in patients under the age of 40 years, in those without a history of hypertension or diabetes mellitus, and in those without associated cognitive, language, or memory impairments.[2] Good visual recovery has been observed in children where the blindness is associated with cardiac surgery[16] as is in our case. In our case, the patient regained full vision with the restoration of presurgical level for acuity of vision. However, unlike other case reports the recovery of presurgical vision for both distant as well as near took only about 2 months of time.[3]

Postoperative posterior ischemic optic neuropathy (PION) which can result in PB with similar signs and symptoms. However, MRI study including the apparent diffusion coefficient map did not find any features suggestive of ischemia/infarction of posterior segment of optic nerve ruling out PION as a diagnosis.

Therefore, strong suspicion for loss of vision due to cortical blindnessis necessary if any feature of microembolization and/or cerebral ischemia/infarction is found in pediatric patients following cardiac surgeries associated with CPB. Efforts should be made to prevent microembolization.

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Conflicts of interest

There are no conflicts of interest.

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