A Rare Neurologic Complication of Coronary Artery Bypass Graft Surgery: Occipital Infarction with Binocular Amaurosis Fugax

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ABSTRACT

Herein, we report a major but transient neurologic complication occurring early after coronary artery bypass grafting with cardiopulmonary bypass. In June 2010, a 63-year-old male patient with a history of unstable angina pectoris and severe coronary stenosis was admitted for surgery. He also had a past medical history of hypertension, hyperlipidemia and smoking for 38 years. Preoperative neurological examination was normal with no visual disturbance or orbital motor dysfunction. Physical examination of other organ systems were also normal. During the early postoperative hours, the patient was clinically stable. Direct and consensual light reflexes were normal bilaterally. Following extubation, bilateral total loss of vision developed. Computed tomography of the brain at postoperative day 1 revealed a cerebral infarction with surrounding tissue edema in the occipital lobes. Following the anti-edematous treatment, a gradual improvement of vision occurred in both eyes. Despite the dramatic improvement in visual functions, a computed tomography of the brain at postoperative day 12 showed the persistence of initial radiological findings, suggesting amaurosis fugax, which is generally defined as a transient monocular visual loss and blindness. Binocular amaurosis fugax associated with bilateral total blindness due to postoperative ischemic optic neuropathy has also been described, with an incidence of less than 0.5%. Some other studies have reported a prevalence rate of 1.3-2% among patients undergoing open-heart surgery. In any case, bilateral amaurosis fugax developing as a transient postoperative neurological complication after coronary artery bypass grafting represents a rare condition. Radiological imaging studies may sometimes fail to detect the condition and complete restoration of visual functions may be achieved by appropriate anti-edema therapy.

Key Words: Amaurosis fugax; postoperative complications; coronary artery bypass

Koroner Arter Greft Baypas Cerrahisine Bağlı Nadir Bir Nörolojik Komplikasyon: Oksipital İnfarkt Sonrası Binoküler Amarozis Fugaks

ÖZET

Yazımızda koroner arter baypas cerrahisinin erken döneminde görülebilen, geçici olan, majör bir nörolojik komplikasyonu sunmaktayız. Ciddi koroner arter lezyonları olan 63 yaşındaki bir hasta cerrahi tedavi için kliniğimize yatırıldı. Ameliyat öncesi değerlendirmede hastanın yüksek tansiyon, hiperlipidemi ve sigara öyküsü mevcut idi. Hastada nörolojik bir patoloji ve görme kusuru yoktu. Diğer fizik muayene bulguları olağandı. Ameliyat sonrası erken dönemde hasta entübe iken durumu stabil seyretti. Puppileride ışık refleksine cevap veriyordu. Ekstübasyondan hemen sonra hasta göremediğinden bahsediyor, kör olduğunu söylüyordu. Bunun üzerine hastaya beyin tomografisi çekildi ve oksipital lobda enfarkt alanı ve çevresinde ödem saptandı. Hastaya antiödem tedavisi başlandıktan kısa süre sonra hastanın görme fonksiyonları düzeldi. Postop 12. günde çekilen kontrol tomografide, hastanın görme fonksiyonunun çabucak düzelmiş olmasına rağmen, radiyolojik patolojik bulguların devam ettiği görüldü. Meydana gelen durum amarozis fugaks olarak değerlendirildi. Amarozis fugaks, ipsilateral gözde geçici monooküler körlük olup, binoküler bilateral körlüğe sebep olan amarozis fugaks postoperatif iskemik optik nöropatide oluşur. Çok nadir görülüp insidansı %0.5 den bile azdır. Açık kalp cerrahisi sonrası görülme oranı %1.3-2'dir. Bize göre açık kalp cerrahisi sonrası bilateral amarozis fugaks görülmesi çok nadir olan, majör fakat geçici bir nörolojik komplikasyondur. Olgumuzda olduğu gibi radiyolojik bulgular her zaman hastanın klinik gidişatını açıklamayabilir. Uygun antiödem tedavi ile görme fonksiyonlarının düzelmesi sağlanarak, radiyolojik bulguların daha geç dönemde düzelmesi beklenebilir.

Anahtar Kelimeler: Amarozis fugaks; postoperatif komplikasyon; koroner baypas

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INTRODUCTION

Herein, we report a major but transient neurologic complication occurring early after coronary artery bypass grafting (CABG) with cardiopulmonary bypass (CPB).

CASE REPORT

In June 2010, a 63-year-old male patient with a history of unstable angina pectoris and severe coronary stenosis was admitted for surgery. Also, he had hypertension, hyperlipidemia as well as a 38-year history of smoking. He was on treatment with acetylsalicylic acid 300 mg/day, omeprazole 30 mg/day, atorvastatin 20 mg/day, and metoprolol 100 mg/day.

Preoperatively he had an arterial blood pressure of 130/80 mmHg and heart rate of 90 bpm with normal sinus rhythm. Auscultation of the carotid arteries, lungs, and heart were normal with no murmurs or crackles. No neurological abnormality, visual disorder, or dysfunction of the orbital muscles were observed. Other organ system examinations were unremarkable.

The chest radiography was normal with no carotid and/or aortic calcification (Figure 1, 2).

Preoperative transthoracic echocardiogram did not suggest any leaflet pathology and the left ventricular ejection fraction was 60% without ventricular motion abnormality.

Coronary angiography showed severe stenosis requiring surgery in the left anterior descending artery, the circumflex coronary artery and the right coronary artery (Figure 3, 4).

A CABG was performed. Routine surgical maneuvers of aorto-caval cannulation and cross clamp were applied with cold cardioplegic arrest. Three reversed autogenous saphenous vein grafts were anastomosed to the right coronary artery, second obtuse branches of the circumflex artery and to diagonal artery distally. Left mammarian artery was anastomosed to the left anterior descending artery. The aortic cross clamp time was 75 minutes and CPB time was 110 minutes. Proximal anastomosis was performed with a single application of aortic side clamp. Operation ended with a safe closure.

In the early post-operative hours, the patient was clinically stable, with bilaterally normal direct and consensual light reflexes.

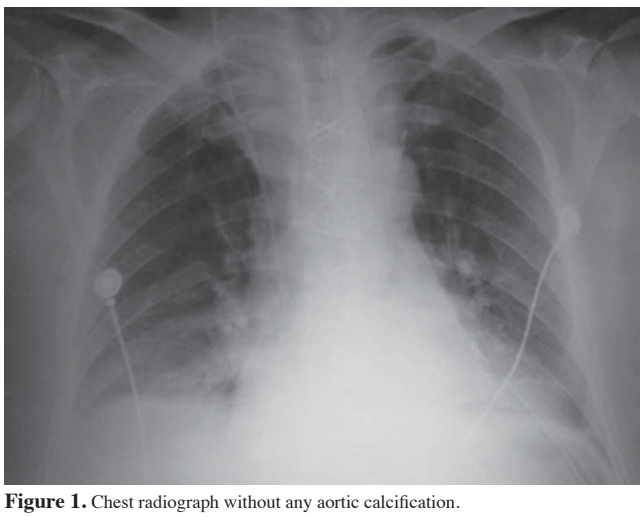

Figure 1. Chest radiograph without any aortic calcification.

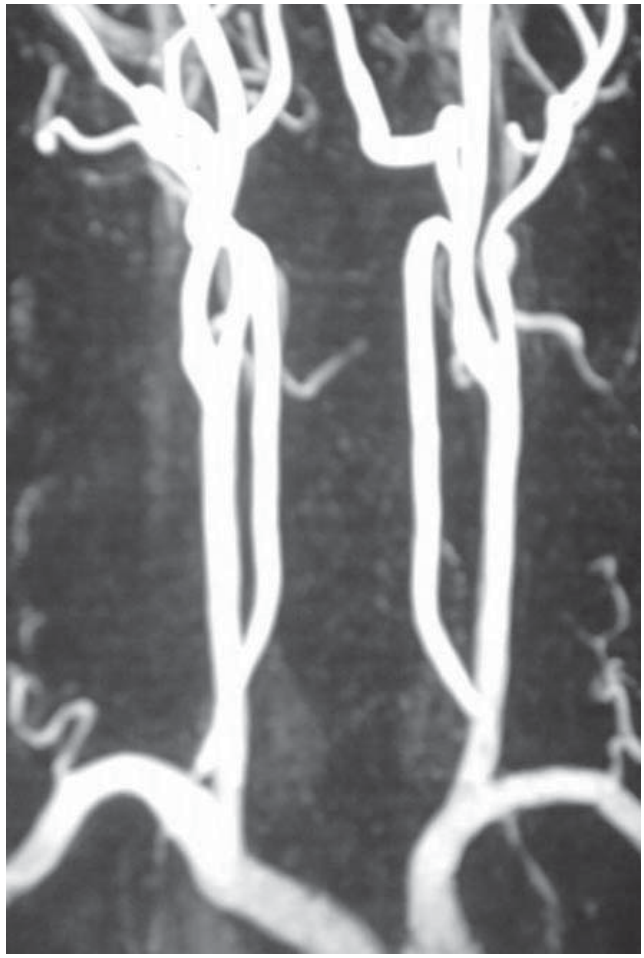

Figure 2. Preoperative MRI of carotid system.

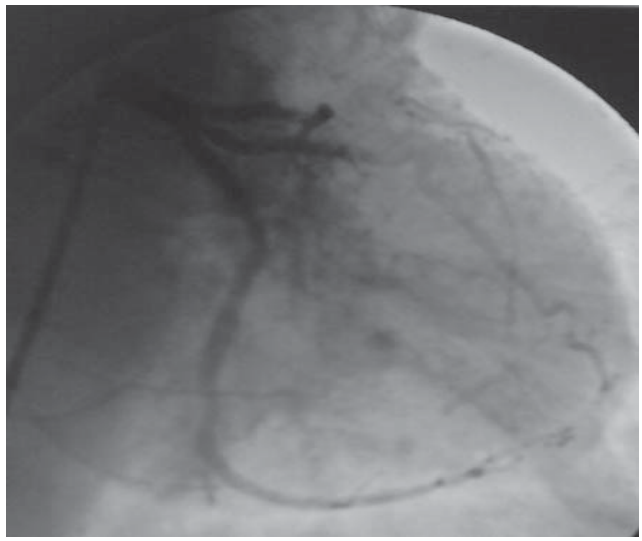

Figure 3. Preoperative left system coronary angiography with severe coronary stenosis.

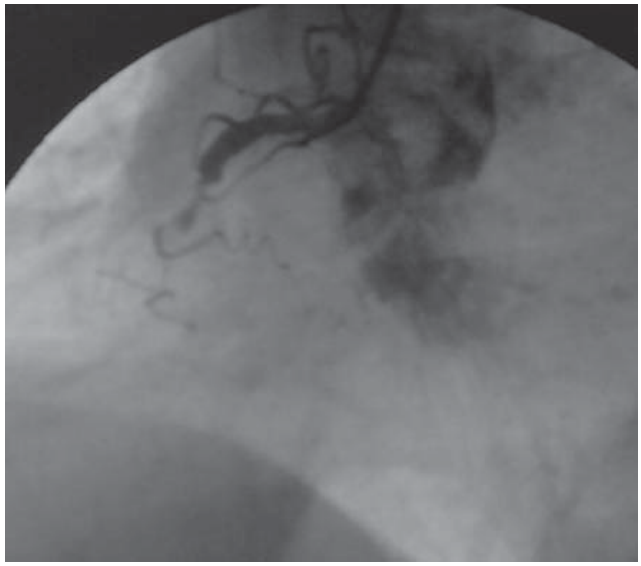

Figure 4. Preoperative right system coronary angiography with severe coronary stenosis.

However, following extubation, bilateral total visual loss developed.

Computed tomography of the brain at postoperative day 1 revealed a cerebral infarction with surrounding tissue edema in the occipital lobes (Figure 5). Following the anti-edematous treatment, a gradual improvement of vision occurred in both eyes. Despite the dramatic improvement in visual functions, a computed tomography of the brain at postoperative day 12 showed the persistence of initial radiological findings (Figure 6).

Patient was discharged at post-operative day 15 with normal neurological and visual functions.

DISCUSSION

CABG is one of the most widely performed surgical procedures, with two major surgical strategies: CPB or on-pump surgery.

CABG with CPB generally involves a number of procedures such as median sternotomy, pericardiectomy, heparinization, aorto-caval cannulation and cardioplegia infusion to achieve cardiac arrest. CPB is associated with an induction of the inflammatory cascade through a complex interaction between multiple factors. During the cardiac arrest, tissue perfusion is maintained by extracorporeal circulation from a roller pump where the mean arterial blood pressure is kept around 60 mmHg to 70 mmHg. The use of of cannulas and cross clamps always hold the potential to mobilize calcifications from the vascular wall which may be a source of embolus[1].

On the other hand, on-pump surgery without the use of cardiac arrest and cannulations may offer certain advantages over CPB, since it does not involve cross-clamping and cardiac arrest as well as the absence of inflammatory cascade induced by CPB. Perfusion pressure is also higher during surgery and cerebral hypo-perfusion with micro and/or macro thromboembolisms originating from calcified debris from the vascular wall is a less frequent occurrence[2].

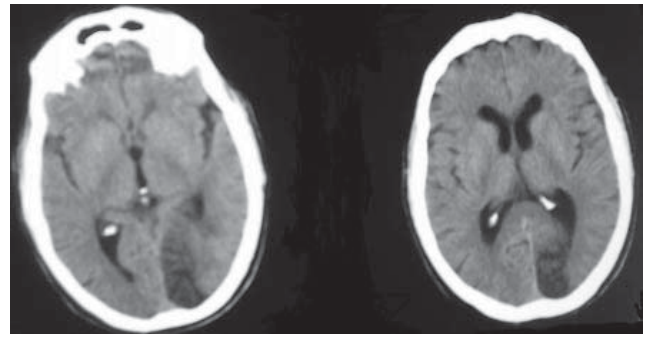

Figure 5. Computed brain tomography with occipital infarct at the first postoperative day.

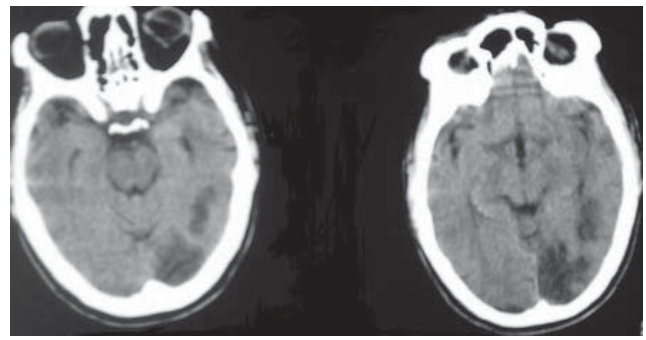

Figure 6. Computed brain tomography with persisting occipital infarct at the postoperative day of twelve.

On the other hand, CABG with or without CPB is a complex surgical procedure that can be complicated by a variety of postoperative factors.

Also, perioperative complications that may involve any organ system are among the most dreaded consequences of both procedures[1].

Neurological complications that may occur following CABG may include peripheral nervous injury, neurocognitive dysfunction, encephalopathy, coma, stroke and brain dead. Of these, postoperative stroke and brain dead represent rare complications, while peripheral nervous injury and neurocognitive dysfunction are more common. Risk factors for neurologic complications after CABG include advanced age, diabetes mellitus, history of central and peripheral neurologic disease, peripheral arterial stenosis including carotid stenosis, renal failure, reduced left ventricular ejection fraction, re-do CABG, severe aortic calcifications and emergency surgery[1,2].

Postoperative peripheral nervous injury have been reported to occur in 10 to 25% of the patients and exhibit a wide variation in severity from unspecific shoulder and back pain to radiculoplexopathies complicated with motor deficiency of upper extremities.

Although most of these events are transient in nature, even a minor peripheral nervous injury after CABG may severely impair the patients' quality of life during hospitalization[1,3].

Compared to peripheral nervous injury, postoperative neurocognitive dysfunction occurs much more frequently among CABG patients in their routine admission to cardiovascular surgery intensive care units. Again, neurocognitive dysfunction

may vary from mild orientation/cooperation disorders to delirium and encephalopathy. The reported overall prevalence ranges between 5% and 50%. The highest incidence of neurocognitive dysfunction after CABG is observed among the elderly, with a reported incidence between 30% and 70% in different studies. Thus, neurocognitive dysfunction is not a rare phenomenon in this patient population and its well established causes include cerebral micro-embolus due to aortic manipulations during cross clamp and/or cannulations and cerebral hypo-perfusion during the prolonged CPB[4]. Some authors have claimed that a dramatic reduction in the severity of neurocognitive dysfunction after CABG may be achieved through avoidance from CPB.

Neurocognitive dysfunction doubtlessly reduces patients' compliance to treatment and prolongs the duration of stay in the intensive care unit and/or in the hospital, with consequent increase in the cost of treatment[3].

Surgical technique and instruments, which have been used during open-heart surgery, progressed since the beginning of first described series of 1960s. Stroke, however, stayed as a main source of postoperative mortality and morbidity for open-heart surgery.

Recent reports suggest that stroke occurs in 1 to 8% of patients undergoing CABG[5], and in our experience the incidence of stroke in this patient group is around 3%. As compared to procedures involving simultaneous valvular surgery, CABG alone is associated with a much lower incidence of stroke. Stroke and atrial fibrillation after surgery present a collaboration. Coma is a rare but terminal neurologic complication of CABG indicating the presence of lesions in the brain stem and/or wide bihemispheric lesions. Overall survival in patients developing coma after CABG is extremely poor[5].

Amaurosis fugax is defined as a transient monocular visual loss and blindness and has five main types: idiopathic, embolic, hemodynamic, neurologic and ocular[6,7]. Embolic and hemodynamic amaurosis fugax is associated with systemic conditions such as systemic lupus erythematosus, periarteritis nodosa, thrombocytosis, atherosclerosis of ophthalmic artery and carotid system, cardiac embolism, temporary vasospasm and surgery. The latter type also includes cases occurring after CABG with CPB[8]. Binocular amaurosis fugax associated with bilateral total blindness has also been described, with an incidence of less than 0.5%[9]. However, some other studies have reported a prevalence rate of 1.3-2% among patients undergoing open-heart surgery[10]. In our experience, postoperative total visual loss occurs in less than 1% of the patients. Visual defects and/or visual hallucinations varying in severity are more frequently observable as a part of the wide-spectrum of mild neurological conditions after CABG[11].

Obviously, bilateral amourosis fugax with resolves within 24 hours is a rare condition[10-13].

In our case, a dramatic recovery of visual functions was observed after the initial total loss of visual function.

However, despite clinical improvement of the neurological condition, radiologic findings suggestive of ischemia persisted, leading to a discordance between radiological and clinical status.

Effective management of postoperative amaurosis fugax requires quick determination of the cause and administration of the appropriate treatment. The reported sensitivity and specificity of computed tomography for this condition is 89% and 100%, respectively. A higher sensitivity of magnetic resonance imaging (MRI) has been reported in some cases due to surgical liga-clips and sternal closure material, although MRI is not suitable during the early postoperative period after CABG[14].

CONCLUSION

Bilateral amaurosis fugax, a transient postoperative neurologic complication after coronary artery bypass graft operations is a rare entity. Radiologic evidence may not always reflect the clinical status of the patients. Total clinical recovery with the return of visual functions can be achieved with appropriate anti-edema therapy.

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