

Combined Central Retinal Vein and Cilioretinal Artery Occlusion in a Patient Who Underwent Allogeneic Renal Transplantation

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Authors' contributions

This work was carried out in collaboration between all authors. All authors read and approved the final manuscript.

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Case Study

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ABSTRACT

A high incidence of venous thromboembolism has been reported in patients who have had renal transplantation especially within the early postoperative period. Herein, the management of a 31-year-old renal graft recipient with the diagnosis of combined left central retinal vein occlusion (CRVO) and cilioretinal artery occlusion (CRAO) is presented. Although detailed work-up for inherited as well as acquired thrombophilic disorders was performed in our case, none of the definite prothrombotic and predisposing risk factors for venous thromboembolism was identified. The case under review or the index case had a remarkable visual recovery within 3 weeks after the referral, since anterior chamber paracentesis was immediately scheduled after performing ocular massage in order to achieve acute resolution of venous stasis.

Keywords: Central retinal vein occlusion; cilioretinal artery occlusion; complications; renal transplantation.

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1. INTRODUCTION

Combined central retinal vein and cilioretinal artery occlusion was firstly described by Oosterhuis in 1968 [1]. Some eyes with CRVO, at its onset, were observed to have concurrent cilioretinal artery occlusion. Several hypotheses have been put forward to explain the simultaneous development of CRAO and CRVO. Cilioretinal artery occlusion may occur secondary to the reduced micro-circulation associated with the development of CRVO. Since the perfusion pressure in cilioretinal artery is lower than the pressure in central retinal artery, the cilioretinal artery is more prone to be occluded secondary to optic discswelling and/ or reduced microcirculation after the development of CRVO [2-4]. Recently, McLeod [5] speculated that reduced circulation in cilioretinal artery may occur as blood flow gradually shunts and is redirected to the choroidal circulation after the development of CRVO.

2. CASE REPORT

A 31-year-old woman without any history of ocular pathology, was referred to our retinal department with a history of blurred vision in her left eye for 20 hours. She had been using sulfamethoxazole-trimethoprim 2 g q.d., prednisolone 12.5 mg q.d., tacrolimus 3 mg b.i.d. mycophenolate mofetil 1g b.i.d. and acyclovir 1200 mg t.i.d. since she had undergone allogeneic renal transplantation fifty days prior to presentation.

At presentation, her best-corrected visual acuity (BCVA) was counting fingers in the left eye, BCVA was 20/20 in the fellow eye at the time of admission. Intraocular pressure (IOP) and the slit-lamp examination were bilaterally normal. The findings on fundoscopy in the right eye was essentially normal, however the left fundus showed tortuous and dilated retinal veins with scattered retinal hemorrhages in all quadrants of the retina, as well as hemorrhagic optic disc edema (Fig. 1 and Fig. 2). Although retinal whitening in the territory of the cilioretinal artery was noticed, no embolic material was observed in contact fundoscopic examination performed using Goldmann three mirror lens. However, combined left CRVO and cilioretinal artery occlusion were suspected after the initial

ophthalmologic examination, as we could not perform fundus fluorescein angiography (FFA) because of the possibility of renal dysfunction due to fluorescent substance in our case who underwent recent allogeneic renal transplantation. After performing ocular massage for about 15 minutes, patient was hospitalized and anterior chamber paracentesis was immediately scheduled in order to dislodge possible clot. Postoperatively, one course of intravenous mannitol therapy (20%, 250 cc/30 min) combined with oral acetazolamide tablets 250 mg q.i.d. for 3 days. Also, topical ofloxacin 0.3% was given six times daily for 10 days.

Systemic work-up of the patient including cardiovascular evaluation, full hematological screening including complete blood cell count (CBC) and serum homocysteine, anti-cardiolipin antibody, lupus anticoagulant, erythrocyte sedimentation rate levels, as well as serum protein electrophoresis and nephrologic evaluation for acute allograft rejection were performed. Her systolic/ diastolic blood pressure was 140/ 90 mm Hg. Signs of hepatosplenomegaly or lymphadenopathy were searched for. Her detailed laboratory findings were as follows: C-reactive protein (CRP) of 11.5 mg/dl, blood urea nitrogen of 14.0 mmol/dl (6–20), serum creatinine of 0.86 mg/dl (0.7–1.2), plasma potassium of 4.1 meq/l (3.5–5.1), plasma Sodium of 141 meq/l (136–145) and serum albumin of 3.7 gr/dl. Screening of CBC revealed 13300/mm³(4800–10800) of white blood cell (WBC), 215000/mm³(15000–45000) of platelet, 11.8 gr/dl (12–16) of serum hemoglobin and 35.5% of hematocrit. Furthermore, no identifiable risk factors for venous thromboembolism was found.

After the nephrology consultation, we were convinced about the safety of fluorescein for this case; so, we decided to perform angiography at the end of the first week. A patent cilioretinal artery was revealed in FA that was performed at the first week post-anterior chamber paracentesis (Fig. 3). Three weeks after the diagnosis, BCVA improved to 20/20 in the affected eye with the complete regression of scattered retinal hemorrhages and hemorrhagic optic disc edema (Fig. 4). Regular follow-up visits were scheduled at three-month intervals and after nine months of follow-up no recurrence has occurred.



Fig. 1. The left fundus at presentation showing a swollen, haemorrhagic optic disc, mildly dilated and tortuous retinal veins in all four quadrants and scattered perivenous retinal haemorrhages. there is retinalwhitening corresponding to the distribution of a cilioretinal artery

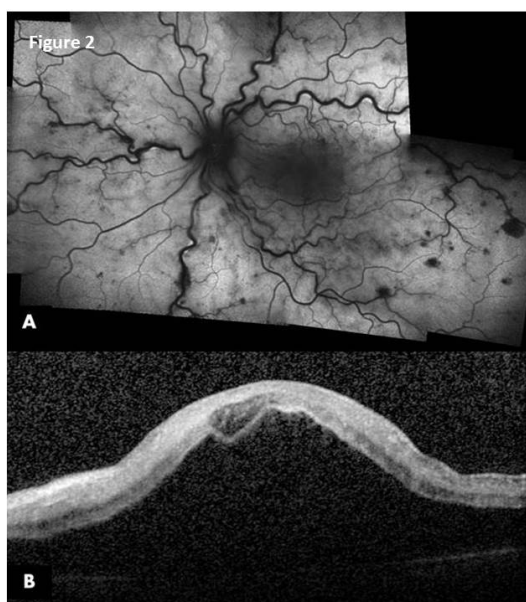


Fig. 2. Autofluorescent image (A) demonstrating tortuous retinal veins, hypoautofluorescence corresponding to scattered haemorrhages, subretinal fluid and the ischemic area. optical coherence tomography (OCT) at baseline (B) shows the presence of subretinal fluid and intra-retinal cystoid spaces within the left macula

3. DISCUSSION

Combined CRVO and cilioretinal artery occlusion has been reported to represent 40% of all cilioretinal artery obstructions [6]. In a large retrospective cohort study conducted by Hayreh et al. [3] the cause of cilioretinal artery impairment was related to non-occlusive

etiology in 32 out of 33 eyes; with combined cilioretinal artery occlusion and non-ischemic CRVO. Although the exact pathogenesis of combined retinal venous and arterial occlusion remains still unknown, Schatz et al. [7] reported the various possible mechanisms especially those affecting retinal microcirculation. The impact of retinal hemodynamics on the

development of combined CRVO and cilioretinal artery occlusion has recently been intensively debated. Since disruption of microcirculation depends on both the arterial and venous pressures, the factors that cause either arterial pressure decrease or venous pressure increase may result in a cessation of retinal blood flow. As sudden rise in intraluminal pressure of retinal capillary network

occurs soon after the development of CRVO, subsequent reduction of the blood flow in the cilioretinal artery may be seen [3]. The duration of the time interval between the cessation of cilioretinal artery blood flow and the re-establishment of central retinal vein-related collateral microcirculation, through its multiple tributaries on the optic disc, affects the severity of such hemodynamic block.

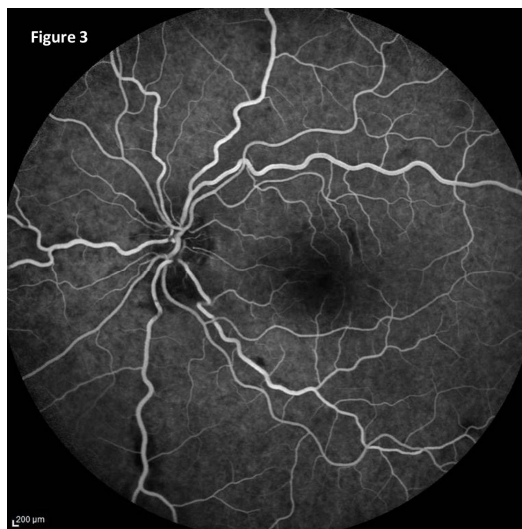


Fig. 3. The early phase of fluorescein angiogram exhibits patent cilioretinal artery at first week after diagnosis

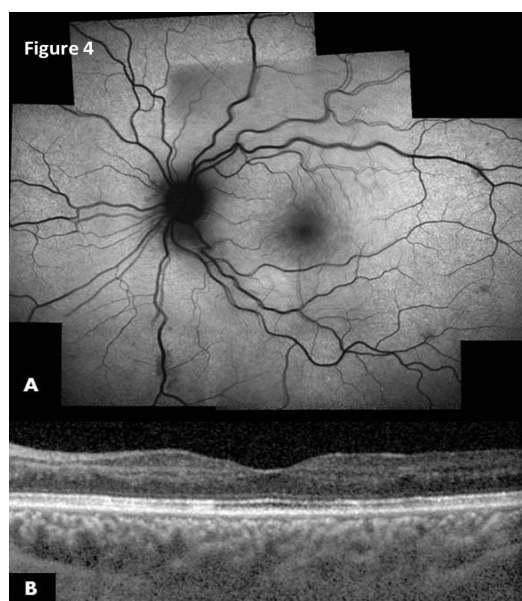


Fig. 4. Autofluorescent (A) and OCT (B) images show improvement of central retinal thickness and complete resolution of neurosensory retinal detachment three weeks after diagnosis

Ischemic CRVO is possibly related to retinal thromboembolism which is more commonly seen in elderly. Patients with non-ischemic CRVO were found to be significantly younger than those with ischemic CRVO or hemi-CRVO [3]. As cilioretinal artery occlusions are also reported to be rare in young patients, any systemic hypercoagulable states should be evaluated in patients aged less than 40 years with a diagnosis of retinal arterial occlusion [8]. Although the presented case was thoroughly investigated for inherited as well as acquired thrombophilic disorders, none of the definite prothrombotic and predisposing risk factors for venous thromboembolism could be identified and a very recent history of renal transplantation was thought to be responsible for such retinal pathology. Although, increased risk for venous thromboembolic events such as pulmonary thromboembolism, deep vein thrombosis and graft thrombosis has been reported after renal transplantation in various studies, [9,10] to the best of our knowledge this is the first reported case of the retinal venous thrombosis developing soon after renal transplantation. In particular, these recipients have an increased risk of venous thromboembolism, possibly due to impaired fibrinolysis and a persistent hypercoagulable state. The incidence of this complication reported in the literature ranges from 0.6 to 25% [11].

Although the visual acuity was very poor at presentation, the patient had marked visual recovery within 3 weeks after the referral. The duration and severity of retinal ischemia directly affect the severity of visual impairment and the recovery of retinal function. Due to the temporary pattern of cilioretinal artery occlusion in eyes with patent parafoveal capillary border, as well as the non-ischemic nature of the concomitant CRVO, an excellent visual prognosis was achieved in the present case. After the complete resolution of the venous stasis, her vision improved to 20/20 without any sequel on ocular examination. As a take home message, especially in young patients, presenting with combined cilioretinal artery occlusion and non-hemorrhagic CRVO, we have to adopt the standard management protocol to optimise visual recovery, even if we have a very poor promise.

4. CONCLUSION

Combined CRVO and cilioretinal artery occlusion are rare in young patients, so systemic

hypercoagulable states and prior surgeries which elevated thromboembolic event risk must be investigated. In case of combined CRVO and cilioretinal artery occlusion, it is extremely important to improve visual acuity. Emerging strategies such as anterior chamber paracentesis is an alternative method of preventing permanent visual loss.

CONSENT

All authors declare that 'written informed consent was obtained from the patient (or other approved parties) for publication of this case report and accompanying images.

ETHICAL APPROVAL

All authors hereby declare that all experiments have been examined and approved by the appropriate ethics committee and have therefore been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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