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RESEARCH ARTICLE

CASE REPORT: CENTRAL RETINAL VEIN OCCLUSION IN ADULT POLYCYSTIC KIDNEY DISEASE

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ABSTRACT

Central retinal vein occlusion (CRVO) is one of the major causes of severe vision impairment and blindness. The prevalence of retinal artery or vein occlusion in the general population increases with age. ADPKD patients are at risk for developing central retinal vascular occlusions at a younger age. Here we report a case of ADPKD with central retinal vein occlusion.

INTRODUCTION

Central retinal vein occlusion (CRVO) is one of the major causes of severe vision impairment and blindness. [1]The prevalence of retinal artery or vein occlusion in the general population increases with age (Lorentzen, 1969). The prevalence of central retinal vein occlusion (CRVO) is 0.1 to 0.7 percent in population-based studies (Mohamed, 2007). CRVO occurs at 0.7% in persons between 49 and 60 years of age. The prevalence progressively increases to 4.6% in persons older than 80 years (Mitchell, 1996). ADPKD patients are at risk for developing central retinal vascular occlusions at a younger age (Gabow, 1984). Adult polycystic kidney disease (ADPKD) is a systemic disease with renal and extra renal, cystic and non-cystic manifestations. The cystic disease involves kidney, liver, pancreas, seminal vesicles and meninges (Torres, 1985). The risk factors for developing CRVO include hypertension, diabetes, hyperlipidaemia, cigarette smoking, hyperviscosity or hypercoagulable states and hyperhomocysteinaemia (Colucciello, 2005).

CASE REPORT

A 53-year-old male presented with a sudden loss of vision in his left eye for last 3 days. The vision loss occurred without any associated symptoms or notable preceding event.He had no prior vision problem.

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On physical examination, blood pressure was 158/88 mmHg. Visual acuity in left eye was counting finger at 1 feet; the right eye was unaffected (OD: 20/20). Pupil examination showed relative afferent pupillary defect in left eye.Intraocular pressures were 12 mm of Hg in right and 18 mm of Hg in left eye.Extraocular movements were normal. Slit examination showed normal anterior segments. Ophthalmoscopic examination of the left eye revealed pale disc, multiple areas of retinal haemorrhages with venous dilatations adjacent to the disc and cotton wool patches (Figure 1).These findings were consistent with combined central retinal vein occlusion (CRVO). Basic investigations were done which revealed azotemia (S.creatinine 2.2 mg/dl). Rest of the baseline investigations were normal. USG Kidneyswere done which showed multiple renal cysts with mild raised cortical echogenicity consistent with signs of polycystic kidney disease. Case was referred to medicine department for same. Diagnosis of adult polycystic kidney disease was made. He was prescribed amlodipine and chlorothiazide for better control of hypertension. He was advised intravitreal injections of bevacizumab and was put on topical antiglaucoma medications. Apart from a diagnosis of ADPKD, the only predisposing condition was hypertension. We propose that retinal vascular occlusion may be a manifestation of the vasculopathy associated with ADPKD (Autosomal dominant polycystic kidney disease).

DISCUSSSION

CRVO has two types: a) Nonischemic (70%): which is characterized by vision that is better than 20/200, 16%

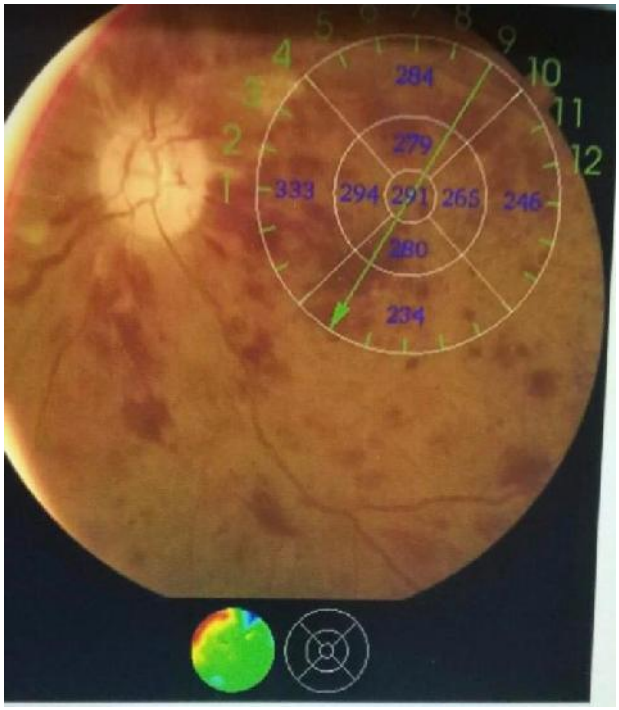

Figure 1. Fundus photograph showing pale disc, multiple retinal hemorrhages and cotton wool spots

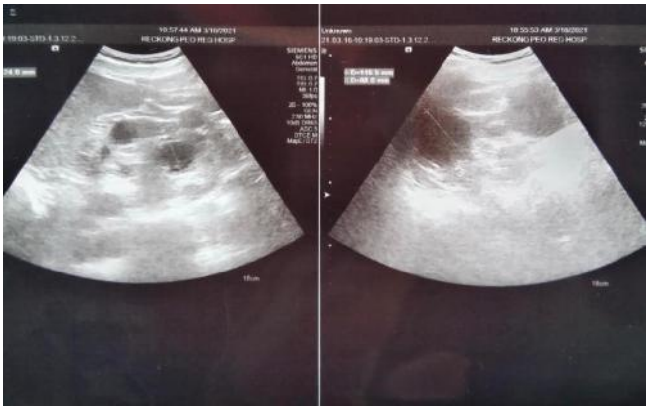

Figure 2. USG Kidneys showing multiple cysts with mild raised cortical echogenicity

progress to nonperfused; 50% resolve completely without treatment; defined as <10 disk diameter (DD) of capillary nonperfusion. b)Ischemic (30%): which is defined as more than 10 DD of nonperfusion; patients are usually older and have worse vision; 60% develop iris NV; up to 33% develop neovascular glaucoma; 10% are combined with branch retinal arterial occlusion (usually cilioretinal artery due to low perfusion pressure of choroidal system) (Freidman, 2005). Ophthalmoscopic examination typically shows retinal haemorrhages with venous dilatation and cotton wool spots for CRVO; and retinal/macular oedema. Long-term complications include retinal neovascularization, macular detachment with vitreous haemorrhage and neovascular glaucoma. The final visual acuity depends on the visual acuity at presentation, the duration of visual impairment and the severity of the subsequent complications (Augsburger, 1980 ; Group, 1997). Qi Qian et al reported occurrence of central retinal vascular occlusions in eight patients with ADPKD at a median age of 51.2 years, contrasted with an approximately 63–75 years in the general population (Lorentzen, 1969 ; Qian, 2007). Retinal vascular occlusion is another manifestation of the systemic vasculopathy in ADPKD. Increased awareness of this manifestation could lead to an earlier diagnosis and treatment before irreversible vision loss develops (Qian, 2007).

Conclusion

When a patient less than 50 years of age presents with central retinal vein occlusion, other causes such as ADPKD should be specifically considered. Detailed history taking, supportive examinations and imaging should be performed. Thus thorough examination helps in early diagnosis and prompt treatment may prevent progress of disease and loss of vision.

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REFERENCES

Augsburger JJ, Magargal LE. 1980. Visual prognosis following treatment of acute central retinal artery obstruction, *Br J Ophthalmol*, vol. 64 (pg. 913-917).

Colucciello M. 2005. Retinal vascular disease in hypertension. Risk factor modification optimizes vision outcomes, *Postgrad Med.*, vol. 117 (pg. 33-38)(pg. 41-42).

Freidman N, Kaiser P, Trattler W. 2005. Central retinal vein occlusion. Review of ophthalmology. 329. Chapter 11.

Gabow PA, Ikle DW, Holmes JH. 1984. Polycystic kidney disease: prospective analysis of nonazotemic patients and family members, *Ann Intern Med.*, vol. 101 (pg. 238-247).

Group TCVOSNatural history and clinical management of central retinal vein occlusion. The Central Vein Occlusion Study Group, Arch Ophthalmol, 1997, vol. 115 (pg. 486-491).

Lorentzen SE. 1969. Occlusion of the central retinal artery. A follow-up, Acta Ophthalmol (Copenh), vol. 47 (pg. 690-703).

Mitchell P, Smith W, Chang A. 1996. Prevalence and associations of retinal vein occlusion in Australia. *The Blue Mountains Eye Study, Arch Ophthalmol*, vol. 114 (pg. 1243-1247).

Mohamed Q, McIntosh RL, Saw SM, Wong, TY. 2007. Interventions for central retinal vein Occlusion: an evidence-based systematic review. *Ophthalmology*. 114:507. j.Ophtha.2006.11.011.

Qian Q,Younge B R, Torres VE.1. 2007. Retinal arterial and venous occlusions in patients with ADPKD. Nephrology Dialysis Transplantation, Volume 22, Issue 6, June, Pages 1769–1771.

The Central Vein Occlusion Study Group Natural history and clinical management of central retinal vein occlusion. Arch Ophthalmol. 1997;115:486–491.

Torres V, Holley K, Offord K. Grantham J, Gardner K. 1985. Epidemiology, Problems in Diagnosis and Management of Polycystic Kidney Disease, Kansas City PKR Foundation (pg. 49-69).

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