Central retinal vein occlusion in hypertensive patient — a case report

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Abstract

Retinal vein occlusion (RVO) is, beside diabetic retinopathy, the most common retinal vascular disease. RVO is associated with many risk factors, both systemic and ocular. Among the systemic risk factors is hypertension. A 36-year-old man came to the hospital because of impaired vision. Central retinal vein occlusion (CRVO) was diagnosed. The main reason for developing RVO was the untreated hypertension, which was diagnosed in the form of hypertensive crisis. The patient presented numerous additional CRVO risk factors that contributed to the development of the described pathology like: obesity, dyslipidaemia, hyperhomocysteinaemia, renal cancer. The authors suggested active examination for CRVO risk factors regardless of the age of patients.

Key words: hypertension; retinal vein occlusion; risk factor

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Introduction

Retinal vein occlusion (RVO) is, beside diabetic retinopathy, the most common retinal vascular disease [1, 2]. It is the fifth cause of blindness [3].

Two types of RVO are distinguished: central retinal vein occlusion (CRVO) and branch retinal vein occlusion (BRVO) [1, 2]. The central retinal vein occlusion is usually caused by thrombus in the vein near lamina cribrosa [1]. The alterations in the central retinal vein are secondary to sclerotic changes in nearly situated central retinal artery [1]. However, may also be combined with morphologically altered endothelium that resembles arterial endothelium [4]. The CRVO can be divided into two subtypes: non-ischaemic and ischaemic [1, 2]. Ischaemic form is often more severe [1] and less frequent [5].

The central retinal vein occlusion is manifested by a painless vision disorder. The retina's images reveal: dilated and tortuous veins, haemorrhages, oedema and cotton wool spots [1].

There are many risk factors of RVO, among them: hypertension [1, 5, 6], dyslipidaemia [1, 6], malignancies [5], obesity [6], hyperhomocysteinaemia [1, 5], abnormal coagulation [1, 5], genetic predisposition [7].

The two main complications of RVO are macular oedema and retinal ischaemia leading to neovascularization [8].

Case report

A 36-year-old man (BMI = 32.8 kg/m^2), intellectually efficient, non-smoker, with negative family history of cardiovascular and ophthalmic diseases, was admitted to the emergency room because of blindness of the right eye for 3 days and high blood pressure.

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Table I. Laboratory tests

Laboratory test	Test result	Norm
Total cholesterol	5.84 mmol/L	3.60–5.20 mmol/L
High-density lipoprotein	1.25 mmol/L	> 1.00 mmol/L
Low-density lipoprotein	4.62 mmol/L	< 3.60 mmol/L
Triglycerides	1.21 mmol/L	0.30–1.70 mmol/L
Homocysteine	23.3 umol/L	5.0–13.9 umol/L

He suffered from cerebral palsy from birth.

During the hospitalization additional tests showed, further abnormalities, including:

- dyslipidaemia — total cholesterol: 5.84 mmol/L, high-density lipoprotein: 1.25 mmol/L, low--density lipoprotein: 4.62 mmol/L, triglycerides: 23.3 umol/L (Tab. I);
- hyperhomocysteinaemia — 23.3 umol/L;
- mutation of the *MTHFR C677T* gene — heterozygote (genetic marker of the abnormal coagulation);
- high blood pressure:
  - — 230/130 mm Hg as assessed with oBP, at admission,
  - — 24-h ambulatory mean systolic and diastolic blood pressure of 152 and 89 mm Hg, respectively, mean activity time BP: 156/93 mm Hg, mean sleep time BP: 135/75 mm Hg;
- ultrasound of the abdomen: hyperechoic focal lesion (predominantly solid + little fluid) in the middle part of the right kidney;
- computing tomography (CAT scan) of the abdomen (with contrast): a heterodense cystic-solid change of 19 mm diameter in the right kidney; non-uniform contrast enhancement;
- histopathological examination of the renal tumour showed polycystic form of clear cell renal carcinoma;
- fundoscopy: right eye — papilloedema, haemorrhages and blurring of the optic disc and its margins, macular oedema, intraretinal haemorrhages and retinal oedema, dilated and tortuous retinal veins; left eye — single cotton wool spot, no other relevant changes;
- fluorescence angiography (Fig. 1):
  - — right eye: extensive ecchymosis of the blood in the posterior pole and on the periphery, the disc is swollen with blurred boundaries; along the large vascular trunks, around the disc and in the macula, extensive foci of soft exudates; numerous areas of blockade of fluorescence by the blood; interrupted blood flow in many vessels; late stagnation of the dye in the vessels. The assessment of capillary circulation is practically impossible, but the clinical picture speaks for high ischaemia. Central retinal vein occlusion,
  - — left eye — a single cotton-wool spot over the papilla; straightened blood vessels; without leakage or dye stagnation.

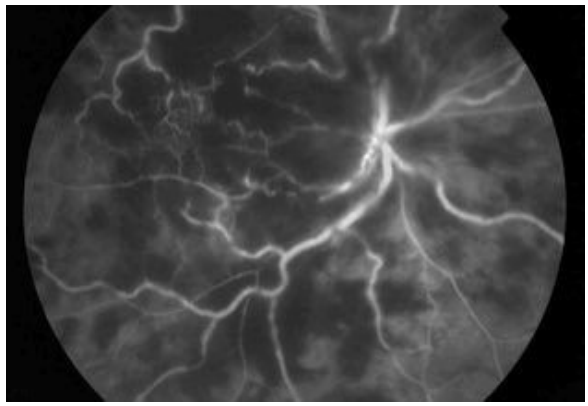

Figure 1. Fluorescence angiography

Discussion

In the presented case we report on severe RVO secondary to hypertensive crisis in a patient with renal tumour.

Increased blood pressure is inherently associated with vascular damage. Mechanical stress caused by shearing forces causes endothelial dysfunction as measured by the reduced bioavailability of the nitric oxide, a potent vasodilatory factor. Concurrently, angiotensin II excess contributes to constriction of the vessels, which further confers risk of hypertension development. The examination of the retinal vessels may also show characteristic changes resulting from long-standing or uncontrolled hypertension. Narrowing of the vessels impairs capillary blood flow, which may lead to RVO, at extremes [9].

Tissue hypoxia promotes vascular endothelial growth factor (VEGF) production and release, which in turn leads to hyperperfusion. The VEGF excess is also claimed to be responsible for the initiation and progression of the oedema and ischaemia in RVO [9, 10].

The reported patient also presented two other common RVO risk factors, i.e.: dyslipidaemia and obesity. Both conditions are associated with insulin resistance and may further contribute to vessel damage. The discussed pathomechanisms contribute to the development of hypertension at an early age [11].

The other factor which possibly added the risk of the development of the RVO in our patient was

a genetic propensity to hyperhomocysteinaemia. This condition is frequently associated with an enhanced resistive index (derived from carotid ultrasound) as well as with damage to the vascular endothelium [12, 13].

Literature also provides reports on the cases of patients in whom RVO had developed in the course of cancers, thus the RVO may be referred to as a paraneoplastic syndrome. However, in the presented case the diagnosed tumour was least likely to play a key role in the RVO development, as it was at an early stage, unlike in the previous reports [14, 15].

Conclusions

The aetiology of CRVO in the presented case was multifactorial; however, hypertension was the most probable triggering factor.

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Conflict of interest

None declared.

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