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Hypertensive Crisis-A Serious Problem in Medical Practice

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Abstract

There has recently been a considerable increase in interest in hypertensive crisis-a life-threatening condition. Recent publications indicate that hypertensive crisis is a problem not only in general medicine but also in ophthalmology. Visual disturbances may be the initial symptoms of severe hypertension and can reflect severe systemic changes. It seems appropriate to conduct further studies on the pathogenesis of vascular hypertensive changes, and particularly any associated inflammatory reactions. It also seems justified to introduce screening for hypertensive changes on the eye fundus photographs in emergency departments.

Keywords: Hypertensive crisis; Hypertensive urgency; Hypertensive emergency; Hypertensive choroidopathy; Hypertensive retinopathy

Hypertensive Crisis

Hypertensive crisis is a life-threatening condition in which the diastolic pressure usually exceeds 120 mmHg [1,2]. The condition may occur as a hypertensive emergency with target organ damage (brain, heart, kidneys and eyes) or it may occur without target organ damage (hypertensive urgency) [3,4]. Treatment involves lowering blood pressure with antihypertensive agents [3,4].

Arriozola-Rodríguez and others presented a twenty-three-year-old patient with sudden bilateral vision loss and hypertensive retinopathy. The patient was diagnosed with hypertensive crisis (his blood pressure was 220/140 mmHg) secondary to chronic renal disease and underwent renal transplantation from a relative [5]. This case was similar to ours of a twenty-five-year-old patient who presented with sudden bilateral reduction in vision associated with chronic kidney failure [1]. This patient was also qualified for a kidney transplant. Aarriozola-Rodríguez and others emphasized that changes in the retinal vasculature may be similar to those in other organs [5,6]. The vascular changes within the kidneys, heart, and retina may have a similar pathogenesis that involves disturbances in endothelial function leading to circulatory dysfunction and reduction of vascular reactivity [5,7]. In our paper, we cited Kovach who described an eighteen-year-old female treated for renal disease secondary to focal segmental glomerulonephritis who developed sudden bilateral visual loss [8]. Kovach reported that glomerulonephritis may be an autoimmune disease although the fundi showed only hypertensive changes and absence of any inflammatory signs [1,8]. However, it should be underlined that clinicopathologic correlations between tissue findings in various organs, including the eyes and kidneys, have already been well-documented. D'Souza and Short claim that the association between high blood pressure and renal and retinal dysfunction is well recognized [9,10]. Both the glomeruli and retina include tiny nets of capillaries. Izzedine and others state that several factors, such as the epithelial growth factor or integrins, which were observed in mouse ocular and renal organogenesis, may later be proven to be important in the development of human eyes and kidneys [11]. Consequently, the authors propose a clinical diagnostic approach of oculorenal syndromes with their genetic links [11].

Hypertensive retinopathy initially shows segmental or generalized retinal arterial narrowing, which if severe may lead to the total closure of secondary arteries and micro-infarcts manifest "cotton wool spots". Other consequences of arterial hypertension are vascular leakage leading to retinal edema, "flame-shaped hemorrhages," and rarely a "macular star" consisting of radiating linear hard intraretinal exudates [12]. Hard exudates and hemorrhages are the result of increased vascular permeability, which may be related to loss of pericytes [7]. Hypertensive retinal endothelial changes may be related to an inflammatory process. This is confirmed by Grunwald and others [7]. Coban and others also suggest a relationship between an inflammatory process and hypertensive retinopathy [13]. Although the pathogenesis of hypertensive retinopathy remains unclear, Coban and others showed a relationship between hypertensive retinopathy and serum levels of C-reactive protein, which in turn may be related to low-grade systemic inflammation [13]. Arterial sclerosis characterized by "arterio-venous nicking" as described by Gunn, "silver wire arterioles," and "copper wire arterioles" are signs of chronic hypertension. Optic disc edema is the result of ischemic blockage of axoplasmic transport at the level of the lamina cribrosa and is commonly associated with malignant hypertension [8]. The hypertensive lesions in the eye fundus can be documented as a picture taken with special fundus cameras, even without mydriasis [14]. In our opinion, fundus screening for the presence of hypertensive changes in patients admitted to emergency departments may be a helpful diagnostic tool in some cases. Correctly diagnosed hypertensive changes in the eye fundus may have prognostic value and help to explain the course of systemic hypertension [15]. It has recently become possible to photograph the eye fundus not only in ophthalmology rooms thanks to the increasing availability of portable cameras or properly adjusted mobile phones [14,16]. Images can be transmitted for analysis by way of telemedicine.

Another study by Stacey and others describes a forty-three-year-old patient with a three-week history of decreasing vision associated with a hypertensive crisis [15]. The authors indicate that choroidopathy is a rare manifestation of hypertension, which occurs mainly in young patients due to the sudden elevation of blood pressure. It is believed

that this phenomenon is due to the higher flexibility and susceptibility of "young" blood vessels [15]. Thus, hypertensive choroidopathy typically occurs in young patients with hypertensive crises, in pre-eclampsia and eclampsia, in pheochromocytoma, in renal disorders [1,12,17-20]. Choroidopathy involves disturbances at the level of the choriocapillaries, which leads to ischemic necrosis of the choroid and the retinal pigment epithelium. "Elschnig spots" (yellow spots with dark centres), which represent foci of infarction, and "Sigerist streaks" characterized by linear spots along the choroidal vessels, which represent areas of fibrinoid necrosis. Local retinal pigment epithelial detachment (PED) and exudative retinal detachment may occur secondary to hypertensive choroidopathy [1,5,15,21,22]. Stacey and others also emphasize the value of fundus screening for the presence of hypertensive changes in patients admitted to emergency departments with concomitant visual symptoms and elevated blood pressure [15].

The publication by Abbassi and others [23] is a good example of fundus findings in hypertensive crisis. The authors present the fundus picture of a twenty-year-old patient suffering from acute hypertension, renal failure, and congestive heart failure. The fundus showed signs of retinopathy, choroidopathy, and optic disc edema.

In conclusion, the growing interest in hypertensive crisis indicates the seriousness of this life-threatening condition. It is necessary to conduct further research on the pathogenesis of vascular changes in systemic hypertension, especially inflammatory reactions. It also seems necessary to introduce screening for fundus hypertensive changes using the fundus photographs in emergency departments which may be a new, helpful diagnostic tool in difficult cases. The hypertensive changes found on fundoscopy may correlate with the vascular abnormalities in other organs. Fundus cameras could help general practitioners search for vascular changes in other organs and to establish a definitive diagnosis.

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