Development of Hypertensive Manifestations after the Disappearance of Hypertension

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The accelerated ("malignant") phase of hypertensive vascular disease is more common in men; sympathectomy may modify some of its signs and symptoms even in those cases in which hypertension is not modified. Six instances are reported here with in which patients in the accelerated phase have developed retinopathy and papilledema during a sustained period of normal blood pressure brought on by a vascular accident or sympathectomy. Therefore factors other than the intensity of the hypertension may be involved in the pathogenesis of this phase.

In hypertensive patients, events such as myocardial infarction, cerebral vascular accidents or bilateral thoracolumbar sympathectomy may be followed by periods of normal blood pressure for weeks or even months. During such a period of reduced blood pressure, occlusive phenomena, changes in heart size, or alterations in renal function may occur, yet the underlying mechanisms cannot be interpreted with certainty; they may result from the pre-existing organic disease or be the sequel to a drop in arterial tension or to differences in cardiac function. On the other hand, progression of retinal lesions during this period of sustained normal blood pressure may throw some light on the processes involved—at least on those pertaining to the production of retinopathy.

In six instances (three after a myocardial infarction, one after a cerebral thrombosis, two after sympathectomy) papilledema and retinopathy have been observed to appear during a normotensive period when they had been absent prior to the hypertensive phase. Examination of the ocular fundi was made through dilated pupils by a single observer, with at least three observations before the episode, and one observation two or more weeks after, during which interval no hemorrhages, exudates or papilledema were visualized. All of these patients had documented hypertensive vascular disease with subsequent proteinuria; none of them had evident complicating or intracranial disease; on clinical grounds they were all in the early accelerated phase of their disease; and, following these observations, two have died with confirmation of the diagnosis of the accelerated phase with necrotizing arteriolitis.

A case report will serve as an illustrative example:

Eight years ago, J. B., a 43 year old Negro janitor, came to the hospital complaining of frequent occipital headaches. His blood pressure at that time was recorded as 176/110. Three urinalyses were negative. There was no evidence of intracranial or endocrine disease, nor was there evidence of coarctation of the aorta. Four years later an intravenous pyelogram disclosed no abnormalities, and one year before the present observations a benzodione test disclosed a normal, mild pressor response. Throughout the eight-year period of observation, repeated blood pressure readings were recorded and varied from 170/100 to 226/126. The eyegrounds showed mild, then moderate arteriosclerotic vessel changes only, but for several years urinalyses disclosed a mild proteinuria together with hyaline casts and occasional red blood cells. After eight years of known hypertensive disease and without antecedent cardiac signs or symptoms, he developed the characteristic pain pattern of a myocardial infarction and was admitted to the medical wards of the Presbyterian Hospital. The diagnosis was confirmed by the appearance of fever, leukocytosis, elevation of erythrocyte sedimentation rate, and typical serial electrocardiographic changes consistent with a posterior wall infarction. The patient's course was complicated by a day of mild shock, and the disappearance of hypertension throughout a hospital stay of 38 days. Daily casual blood pressure readings during the first 10 days and measurements twice...

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weekly thereafter, varied from an initial 96/50 to a maximum value of 138/86. There were no signs of congestive failure, no arrhythmias, and no signs or symptoms of encephalopathy or cerebral vascular disease. The cardiac silhouette was moderately enlarged by X-ray examination and three urinalyses in the course of this admission disclosed a constant 2-plus proteinuria with specific gravities never above 1.016. The blood urea nitrogen concentration was 18 mg. per 100 cc.

On admission the eyegrounds revealed only moderate arteriosclerotic changes in the retinal vessels, and the funduscope picture was similar 11 and 19 days after the date of admission. On the twenty-fourth day of his hospital stay (blood pressure 126/80), bilateral retinal hemorrhages and blurring of the nasal disc margins were noted for the first time, progressing to the point of definite papilledema within a space of one week. Although complaining of blurred vision, he refused further assistance, was discharged still with a normal blood pressure, and died eight months later of congestive failure and uremia following admission to another hospital.

Autopsy, including examination of the brain, disclosed cardiac hypertrophy, a healed myocardial infarction, extensive necrotizing arteriolitis, nephrosclerosis, and no signs of intracranial disease.

**Discussion**

It has been suggested that the mechanism of the accelerated ("malignant") phase of hypertensive vascular disease is closely related to blood pressure levels, particularly to increases above some critical level for each patient, and that neuroretinal edema is due to raised intracranial pressure, a consequence of the high level of diastolic arterial pressure.1 There is no doubt that levels of the arterial tension are generally higher in those in this stage of the disease than in those in less advanced stages, and that sodium-restricted regimens, drugs or surgical procedures which lower the blood pressure are associated usually with regression of retinopathy. However, the accelerated phase may develop at times with only slight to moderate increases in blood pressure. Furthermore, cerebrospinal fluid pressures within the normal range have been encountered in some hypertensive patients with papilledema and retinopathy in this clinic, and consequently the appearance of papilledema and retinopathy in the absence of hypertension in six instances observed by us poses further questions regarding the mechanisms involved.

If a complication, for example, papilledema, can develop weeks after the disappearance of hypertension, at least in the accelerated phase, consideration must be given to possible causative factors other than those related only to arteriolar vasoconstriction. Thus it would appear that the blood pressure lowering effects of agents and procedures employed in the management of hypertensive disease may be only a partial reason for their usefulness. At any rate, the rare but definite appearance of retinopathy and papilledema, after a hypertensive patient has lost his hypertension, indicates that high diastolic values are not solely responsible for their development. These observations support the view that at least some manifestations of the accelerated phase of hypertensive vascular disease may be related to something more than the intensity of the hypertension.

**Conclusions**

1. Six instances have been encountered in which patients with the accelerated ("malignant") phase of hypertensive vascular disease have developed retinopathy and papilledema during a sustained period of normal blood pressure.

2. Factors other than the intensity of the hypertension may be involved in the pathogenesis of the accelerated phase.

**SUMARIO ESPAÑOL**

La fase acelerada ("maligna") de la enfermedad hipertensiva vascular es más común en los varones; la simpatectomía puede modificar algunos de los hallazgos y síntomas aún en los casos en los cuales la hipertensión no sea modificada. Seis casos en la fase acelerada se informan, en los cuales se desarrolló retinopatía y papiledema durante un período sostenido de presión arterial normal causado por accidente vascular o simpatectomía. De manera que hay otros factores además de la intensidad de la hipertensión que pueden estar relacionados a la patogénesis de esta fase.

**REFERENCE**

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