MALIGNANT HYPERTENSION IN THE NATAL AFRICAN AND INDIAN

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Volhard and Fahr (1914 - 19) defined malignant hypertension as (1) fixed elevated systolic and diastolic blood pressures, (2) eye grounds revealing papilloedema, spastic arteriolitis, haemorrhages, and exudates, and (3) rapidly progressive course towards a fatal outcome, and usually ending in uraemic coma. In this paper an attempt has been made to study malignant hypertension as we see it at the Hypertension Clinic from clinical material in the wards and from postmortem studies. Material for this study was obtained at the King Edward VIII Hospital, Durban, from 32 cases studied at the Hypertension Clinic, 53 cases admitted to a ward, and 34 postmortem examinations.

Incidence

Hypertension appears to be a common disease among Africans and Indians in this area. Over 600 cases of hypertension have been seen in the Hypertension Clinic in the past 3 years, and out of the first 450 of these cases 32 suffered from malignant hypertension, a proportion of 7%. This is higher than that reported by Kincaid-Smith *et al.*[1] (1%) and by Perera[2] (5%).

In this series of 450 cases of all types of hypertension, males and females were equally affected (230 males, 220 females), but malignant hypertension was much commoner in males (25 males, 7 females), suggesting that hypertension is more severe in the male. The average age of the 32 cases was 44 years, with a minimum of 31 and a maximum of 79. A family history could only be obtained from Indian patients, 4 out of 16 reporting hypertension in relatives.

Mode of Presentation

Of 32 cases of malignant hypertension studied at the Clinic, headache was by far the commonest symptom, being present in 22 cases (70%). Blurring of vision was present in 14 cases (45%); on this account a patient may first seek advice from an ophthalmologist. When first seen, one patient had acute pulmonary oedema, 4 nocturia, and 4 others hypertensive encephalopathy. Loss of weight, which occurred in 20% of patients in the series reported by Milliez *et al.*,[3] was not seen.

The mode of onset of the malignant phase of hypertension may be quite sudden, as was observed in 3 of our patients. Thus one patient was quite well until he felt faint while working and started to vomit. He was brought to hospital and found to have malignant hypertension. Another patient had severe leg pains while working, lost consciousness, and was admitted to hospital with malignant hypertension.

Blood Pressure

Among the 32 cases analysed at the Clinic, the average systolic blood pressure (BP) was 240 mm.Hg (range 175 - 280), while the average diastolic pressure was 147 mm.Hg (range 120 - 180).

Renal Insufficiency

Some authors consider renal insufficiency an important and constant sign of malignancy,[4] but its diagnostic value is held to be of secondary importance by others[5] because renal failure is not always present in malignant hypertension. Out of 24 cases of renal insufficiency reviewed at the Clinic, 14 (60%) had an initial blood urea below 40 mg.%, 5 (20%) between 40 and 100 mg.% and 5 (20%) over 100 mg.%. Untreated, there was a progressive rise in the blood urea.

Albuminuria is a frequent feature: 75% initially showed albuminuria (in 20% it was gross); 10% later developed albuminuria, and 15% had no albuminuria. There was a good correlation between the level of albumin in the urine and the blood pressure. If the diastolic BP was maintained below 100 mm.Hg the albumin gradually disappeared, and when therapy was stopped the albumin reappeared. One can use this as a guide to the recognition of therapeutic response and of tolerance, and whether or not patients are taking treatment.

Clinical Course

In one case there was a regression in the malignant hypertension. This patient first presented in June 1960 with typical features—BP 240/160, gross albuminuria, grade-4 retinopathy and congestive cardiac failure. She was treated with reserpine and an oral diuretic combined with macamylamine, 15 mg. daily. There was improvement over a period of 8 months, and today she is normotensive and out of failure, her fundi are normal, and she is controlled on reserpine and an oral diuretic. One may ask in this case if the vigorous use of hypotensive agents did not re-set the baroceptors of the carotid sinus at a lower threshold. Milliez *et al.*[6] have described one case of spontaneous regression of papilloedema, and Keith and Wagener[7] 15 cases.

In one medical unit, 53 cases of malignant hypertension admitted between 1955 and 1962 were treated on various hypotensive agents such as pentolinium, hexamethonium bromide, mecamylamine, guanethidine and hydralazine. It was found that of 44 patients whose urea was over 50 mg.%, 34 died during their first admission to hospital. Hypotensive agents were used in 41 of the over-50 mg. cases and in only one of them did the urea not rise. The remainder showed a progressive rise and 32 died of uraemia. Cerebrovascular episodes occurred in 4 cases, and 7 had associated congestive cardiac failure. It would seem that in malignant hypertension hypotensive agents are most effective when the urea is under 50 mg.%. Bjork *et al.*[8] found that in treated cases of malignant hypertension with remaining renal function at about half the normal or more as measured by the endogenous creatinine clearance, 82% survived 1 year, and after 5 years 64% were still alive.

The average period of regression of papilloedema observed in 14 patients after the use of hypotensive agents was 3 months, with a minimum of 1 month and a maximum of 7 months.

The following incidental findings were noted: (*a*) 50% of those questioned had a history of a large intake of salt; (*b*) all the African and Indian patients had lived in Durban for 10 years or more; (*c*) only one patient (an Indian) had glycosuria.

At the Hypertension Clinic, the minimum follow-up period was 1 month and the maximum 45 months, with an average of 10 months; 17 patients had a good follow-up of 6 months or more.

Guanethidine was used for 17 patients, and 50% of these remained overweight in spite of the use of the diuretic. Tolerance developed in 57% of the cases. Other side-effects were tiredness in 3, and giddiness, mainly in the mornings and on walking, in 8. The dosage of guanethidine varied from 10 to 150 mg. daily, with an average of 70 mg. daily.

Mecamylamine was used for 5 patients, in one of whom tolerance developed. One of them developed severe parasympathetic side-effects and 2 complained of giddiness. The dosage varied from 7·5 mg. to 50 mg. daily, with an average of 22·5 mg. daily.

Necropsy Findings

Among 34 necropsies performed on patients with malignant hypertension between 1959 and 1961, in 2 cases (6%) chronic pyelonephritis was present. This figure is in contrast to that of Somers,[9] in Uganda, who found that about half the patients with hypertension suffered from renal hypertension, usually with chronic pyelonephritis. Other estimates of the proportion of malignant hypertension due to chronic pyelonephritis are those of Weiss and Parker[10] (15 - 20%), Kincaid-Smith *et al.*[1] (21%), and Heptinstall[11] (16%). Using as an index the presence of bacteria and pus cells in the urine, we had considered 8 of these 34 patients with malignant hypertension to have had pyelonephritis, but in 6 of the 8 this diagnosis was disproved at necropsy.

Summary and Conclusions

The salient features of malignant hypertension among Africans and Indians as seen at the King Edward VIII Hospital, Durban, in necropsy material, among patients admitted to a medical unit, and in others attending the Hypertension Clinic, are reviewed.

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