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Hypertension in black South Africans — new perspectives on old material

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Summary

Autopsy material was examined from cases diagnosed as malignant nephrosclerosis in the years 1956 - 1961, a period when adequate antihypertensive therapy had not yet become available, a second group of malignant nephrosclerosis from the years 1970 - 1980, an era during which effective antihypertensive therapy was available, and a third group of essential benign nephrosclerosis, once more from the early pretreatment period (1956 - 1961). The observations suggest that malignant and benign hypertension may be two different diseases. Further studies will be pursued to assess whether hypertensive renal changes seen in this study are a spectrum of one disease extending from malignant nephrosclerosis to novo presenting with acute renal failure or chronic renal failure to benign hypertensive nephrosclerosis.

Many studies have shown that hypertension in the black population of southern Africa is common and severe. It occurs at a younger age than in whites and behaves in an explosive manner, with death occurring mainly from cerebral haemorrhage, uraemia or congestive cardiac failure. It has been demonstrated that hypertension is not a major problem in the rural black but becomes prevalent in the urban environment. Several studies have shown that the vast majority of black South African hypertensives have essential hypertension and that a considerable proportion of the latter develop the malignant phase of the disease.

Methods

The material studied was derived from the autopsy records of the South African Institute for Medical Research's histopathology laboratory at Baragwanath Hospital. Several categories of records were examined and three groups emerged: (i) autopsy cases diagnosed as malignant nephrosclerosis in the years 1956 - 1961, a period when adequate antihypertensive therapy had not yet become available; (ii) cases labelled 'malignant nephrosclerosis' from the years 1970 - 1980, i.e. an era during which effective antihypertensive therapy was available; and (iii) cases labelled 'essential benign nephrosclerosis' from a period when effective hypertensive therapy had not yet become available (1956 - 1961).
Results

In the first group of malignant nephrosclerosis (1956 - 1961) the mean age was 46 years, the mean systolic blood pressure 230 mmHg and the mean diastolic blood pressure 145 mmHg. Death in the great majority of cases had been due to cerebrovascular accident and/or ureaemia. The kidneys (combined weight 250 g) showed fibrointimal necrosis in the majority of cases and characteristic myxoid myo-intimal hyperplasia of the interlobular arteries in every instance. Histopathological findings suggested an acute onset of malignant hypertension with few concomitant features of benign nephrosclerosis. We consider that these changes had occurred de novo over a relatively short period of time. An analysis of the subjects with malignant nephrosclerosis during the treatment era (1970 - 1980) showed that death had now generally been due to ureaemia or congestive cardiac failure. Fibrointimal necrosis was less frequent in this series, but the characteristic onion-skin intimal proliferation of the interlobular arteries was again present in every instance. The combined kidney weight was 205 g. The third group studied consisted of subjects who had died of essential benign nephrosclerosis during a period when effective antihypertensive therapy had not become available (1956 - 1961). Here the mean age was 10 years older than the groups with malignant nephrosclerosis de novo, and the majority had died of a cerebrovascular accident. Fibrointimal necrosis was not found in this group and histologically the kidneys (combined weight 255 g) were characterised by marked fibrous thickening of the walls of all the renal blood vessels.

Discussion

These studies show that, although fibrointimal necrosis in malignant hypertension may not be as frequent as it is among whites, it is still seen in a significant proportion of cases. This is contrary to the claims of several American authors. The hallmark of malignant nephrosclerosis in black subjects is myxoid myo-intimal hyperplasia of the interlobular arteries. These observations suggest that there may be a broad spectrum of malignant nephrosclerosis ranging from an acute de novo presentation to benign nephrosclerosis with superimposed malignant change. The studies confirm that there are some clinical and pathological differences between black and white hypertensives. A recent local study showed that hypertensive causes of death were approximately four times more common in all age groups and in both sexes among blacks than among whites.

The question that arises from these observations is whether malignant hypertension simply arises from neglected benign hypertension or is aetiologically different from benign hypertension. The findings suggest that the two diseases may indeed be different. Recent data from the South African Dialysis and Transplantation Registry confirm that hypertension is the commonest cause of end-stage renal failure in South Africa, particularly among blacks.

The findings also indicate a changing mortality pattern of malignant hypertension from the pretreatment to the treatment era. The accent in the former group is on death from ureaemia, whereas congestive cardiac failure is more prominent in the later series. In addition, fibrointimal necrosis, seen frequently in the earlier group, is rarer in the later group. The characteristic myxoid myo-intimal hyperplasia is present in both groups.

There are clinical and pathological differences between malignant and benign hypertension in the pretreatment era. Pathologically, the latter group is characterised by marked fibrous thickening of all the blood vessels of the kidney but no fibrointimal necrosis or myxoid myo-intimal hyperplasia of the interlobular arteries. In malignant nephrosclerosis de novo, changes in the coronary arteries are minimal and consist of mild fibrous intimal thickening, whereas in malignant nephrosclerosis that develops after a period of benign hypertension the coronary arteries show varying degrees of atherosclerosis.

The causation of fibrointimal necrosis in malignant hypertension is uncertain. It would seem to be related to the severity and rapidity of the hypertension, since it occurred less frequently in the treatment era. Is the myxoid myo-intimal hyperplasia — the hallmark of malignant nephrosclerosis — a product or a cause of the hypertension? The question is relevant because in systemic sclerosis similar changes may be seen in the kidney in the absence of hypertension. Are the hypertensive renal changes seen in this study a spectrum of one disease extending from malignant nephrosclerosis de novo presenting with acute renal failure or chronic renal failure to benign nephrosclerosis?

Future studies

Future investigations should include a study of the distribution of renin and angiotensin II in kidney material with an assessment of the presence of immunoglobulins in the myxoid material of the interlobular arteries. The vasculature of the kidney should be studied to assess the changes that occur with the treatment of malignant nephrosclerosis and how this differs from the vascular lesions seen in benign nephrosclerosis. The smooth-muscle cell is primarily involved in the intimal hyperplasia of the interlobular arteries, and attempts should be made to culture this cell and subject it to various stimuli in an attempt to produce myxoid material.

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