# Hypertension and its relation to renal function 10 years after pregnancy complicated by pre-eclampsia and pregnancy induced hypertension

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## ABSTRACT

**Objective:** To assess the development of hypertension and its relation to renal function 10 years after pregnancy complicated by pre-eclampsia and pregnancy induced hypertension.

**Methods:** Women with pre-eclampsia (n=47), pregnancy induced hypertension (n=54) or normotensive (n=46) during 1988 were reviewed at King Hussein Medical Center, Amman, Jordan, for the development of hypertension and renal disorder. Their renal function was reviewed by measuring blood levels of urea, uric acid, creatinine, calcium and albumin. Urine was examined for microalbuminuria.

**Results:** Women with pre-eclampsia and pregnancy induced hypertension had a higher risk of developing hypertension 10 years later compared to the control group, (23% for pre-eclampsia, and 39% for pregnancy induced hypertension vs. 3% for control). Albumin corrected calcium levels were significantly higher in patients with history of pre-eclampsia (2.41 mmol/l) and pregnancy induced hypertension (2.42 mmol/l) vs. control (2.33 mmol/l) as well as a significant difference in microalbuminuria levels (23% in pre-eclampsia, and 16% in pregnancy induced hypertension vs. 3% in control). Serum urea, creatinine and uric acid levels were not significantly affected (4.4 mmol/l in pre-eclampsia, 4.7 mmol/l in pregnancy induced hypertension and 4.6 mmol/l in control for urea, 76.0 mmol/l in pre-eclampsia, 74.0 mmol/l in pregnancy induced hypertension and 77.0 mmol/l in control for creatinine and 252.0 mmol/l in pre-eclampsia, 250.0 in pregnancy induced hypertension and 248 mmol/l in control for uric acid).

**Conclusion:** The risk of development of chronic hypertension 10 years after pregnancy complicated by pre-eclampsia and pregnancy induced hypertension is increased and this is closely related to residual renal disorder.

**Keywords:** Renal function, pre-eclampsia, pregnancy induced hypertension.

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It is well known that women with chronic hypertension and renal disease are at particularly high risk of developing pre-eclampsia (PET) and pregnancy induced hypertension (PIH),[1] but whether the opposite is true (i.e. PET and PIH will cause chronic hypertension and renal disorder later in life) is a matter of controversy. Chesley in 1978 claimed that the average risk for development of hypertension later in life, in patients with history of PIH, was 35%.[2] Older studies claimed that there is a higher incidence of chronic hypertension in patients with a history of PET or PIH, but this was not related to impaired renal function.[3] The aim of our study was to assess the development of chronic hypertension 10

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**Table 1** - Age, weight and parity in 1988.

| | Age (Years) | Nulliparity (%) | Weight (Kg) |
|---|---|---|---|
| PET (n = 47) | 27 | 68 | 83 |
| PIH (n = 54) | 32 | 43 | 88 |
| Control (n = 46) | 31 | 45 | 71 |

PET = pre-eclampsia
PIH = pregnancy induced hypertension
(Values are given as mean)

years after pregnancy complicated with PET or PIH and its relation to renal function; and to identify features of renal function that are affected.

Methods. All patients who took part in this study were delivered at King Hussein Medical Centre. Midwives performed uncomplicated deliveries, while the doctor on call delivered all other patients with PET or PIH. All records were retrospectively studied. Patients whose records indicated PET or PIH were asked to return for follow-up. Out of the patients who agreed to return, and after studying their medical records, 2 groups of patients were identified. The first group was the PET group (n= 47) and the second was the PIH group (n= 54). To obtain a control group, a number of women who had uneventful singleton vaginal deliveries during 1988 were invited to take part in this study. Forty-six women agreed to return to act as our control group. None of the women had a history of chronic renal disease, endocrine disease or cardiovascular disease. Blood pressure was recorded in the sitting position after 10 minutes of rest. The women were considered to have chronic hypertension if they were on anti-hypertensive medication or if 2 diastolic blood pressure readings, with 15-minute intervals, were 90 mmHg. Blood samples for urea, uric acid, creatinine, albumin and calcium were drawn. Urine samples were taken for albumin testing. The presence of microalbuminuria (albumin excretion of 20-200 mg/ 24h) was determined by testing 24-hour urine samples.

Results. Table 1 shows the age and weight of all women in 1988, as well as the percentage in each group who were nulliparous. This table shows that nulliparity was more common in the PET group, while heavier women were more common in the PIH group. Table 2 shows the weight of the same women 10 years later. It also shows the relevant biochemical changes (urea, uric acid, creatinine, Ca++, and the percentage who had microalbuminuria). The mean blood pressure readings are indicated for each group. Development of hypertension was significantly higher in the PET and PIH groups compared to the control group. Hypertension was also more common in the PIH group compared to the PET group. Biochemical changes indicate that plasma Ca++ concentrations, corrected for albumin, were significantly higher in patients who developed hypertension compared with the control group. Microalbuminuria was also higher in patients with hypertension compared with the control group, however, other variables (urea, uric acid and creatinine) where not significantly altered.

Discussion. There has been a lot of controversy regarding the development of hypertension in later life, after pregnancy complicated by PIH or PET. Chesley claimed that PIH predisposes to hypertension later in life[2] while Svensson et al and Sibai et al claimed that PET as well as PIH, could also lead to future hypertension,[4,5] and this agreed with our study and with other studies performed by Adams and Mac-Gillivray.[6] Sibai et al claimed that, as development of gestational diabetes could be used as a screening test for future development of diabetes, PET and PIH can be used as a screening test for future development of chronic hypertension.[5]

In our study we have found that a history of PIH or PET increases the risk for microalbuminuria later in life but we could not define whether glomerular lesion was a cause or result of hypertensive disorder in pregnancy. We also could not identify whether hypertension and glomerular damage persisted from the time of pregnancy or appeared later, but in all cases, and since microalbuminuria appeared in both PIH and PET groups, it seems to be a blood pressure dependant factor and not due to any pre-eclampsia related factor.

In conclusion, history of both PIH and PET is associated with a significant risk of developing hypertension later in life. The history of previous

**Table 2** - Clinical data in 1998.

| | Weight (Kg) | Sys/Dias BP (mm Hg) | Hypertension (% of patients) | Urea (mmol/l) | Uric acid (mmol/l) | Creatinine (mmol/l) | $Ca^{+2}$ (mmol/l) | Microalbuminuria (% of patients) |
|---|---|---|---|---|---|---|---|---|
| PET (n = 47) | 75 | 120/85 | 23 | 4.4 | 259 | 76 | 2.41 | 23 |
| PIH (n = 54) | 80 | 125/90 | 39 | 4.7 | 250 | 74 | 2.42 | 16 |
| Control (n = 46) | 65 | 110/72 | 3 | 4.6 | 248 | 77 | 2.33 | 3 |

(Values are given as mean)

PET or PIH should therefore alert the physician to the possibility of persistence of renal damage, which might become obvious later in life. The presence of microalbuminuria in a woman with a previous history of PET or PIH may indicate persistent renal damage from the original insult.

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