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Hemihypertrophy, Bilateral Wilms' Tumours and Clear-cell Adenocarcinoma of the Uterine Cervix in a Young Girl

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THE ASSOCIATION of congenital hemihypertrophy with tumours of embryonal origin like nephroblastomastosis and Wilms' tumours or with adrenocortical tumours is well known [1, 2]. We report another association in a young girl who had hemihypertrophy and developed Wilms' tumour of both kidneys and clear-cell adenocarcinoma of the uterine cervix.

This 4-year-old Caucasian girl was the offspring of a normal pregnancy with no recorded drug exposure. She was referred to us for left hemihypertrophy first diagnosed at the age of 6 months.

A screening ultrasonography of the kidneys revealed a right mass measuring 21 mm in diameter which was confirmed by CAT scan. The child then underwent a right radical nephrectomy. Pathological examination showed a classical Wilms' tumour. Postoperative chemotherapy (vincristine 1.5 mg/m^2 and D actinomycin 0.75 mg/m^2 at intervals of 3 weeks for 6 months) was given and the child was then closely followed-up by chest X-ray and renal ultrasonography. 54 months after surgery, she had one episode of macroscopic haematuria. Renal ultrasonography revealed a solid tumour of the upper pole of the left kidney. Chest X-ray, liver ultrasonography and bone nuclear scan were normal. Pre-operative chemotherapy combining vincristine and D actinomycin was administered during 4 weeks. The tumour shrank dramatically in size and the child underwent tumorectomy. Examination of pathological specimen revealed a classical triphasic nephroblastoma. Karyotype of the resected tumour showed monosomy 22. Post-operative chemotherapy combined vincristine, D actinomycin and epiadriamycin for a total of 27 weeks. The patient remained free of symptoms for 25 months. She then presented with vaginal bleeding. Ultrasonography and CAT scan of the pelvis revealed a mass developing from the cervix and measuring 3.5 cm in diameter. There was no abnormal node in the pelvis. Resection of the cervical tumour was then performed by the vagina. Pathological examination showed a clear-cell adenocarcinoma of the cervix. The child then underwent conisation with extended lymph node resection but there was no residual tumour and the removed nodes were normal.

The patient has been closely followed-up. There is no evidence of disease 14 months after the last operation.

The association of hemihypertrophy with nephroblastomatosis and Wilms' tumour is well recognised but not with clear-cell adenocarcinoma. Hemihypertrophy is congenital and since nephroblastomatosis, Wilms' tumour [3] and clear-cell adenocarcinoma of the cervix [4] have embryonal origins, one might imagine a common origin to explain their association. It is of interest to note that there was no history of maternal ingestion of stilbestrol or related oestrogens during pregnancy. Furthermore, karyotype on the second Wilms' tumour showed monosomy 22. This chromosome abnormality has never been described in Wilms' tumour. It would have been interesting to have the karyotype of the other Wilms' tumour and of the clear-cell adenocarcinoma and the molecular genetics on these three tumours since several studies indicate the involvement of two distinct regions of chromosome 11p (11p13 and 11p15) [5, 6] and of one locus of chromosome 16q [7] in the development of Wilms' tumours.

1. Benson PF, Vulliamy DG, Taubman JO. Congenital hemihypertrophy and malignancy. *Lancet* 1936, **1**, 468–469.
2. Fraumeni JF Jr, Miller RW. Adrenocortical neoplasms with hemihypertrophy, brain tumours and other disorders. *J Pediatr* 1967, **70**, 129–138.
3. Bolande RP. Congenital and infantile neoplasms of the kidney. *Lancet* 1974, **2**, 1497–1499.
4. Kurman RJ, Scully RE. Clear cell carcinomas of the endometrium. *Cancer* 1976, **37**, 872–882.
5. Koufos A, Hansen MF, Copeland NG, Jenkis NA, Lampkin BC, Cavenee WK. Loss of heterozygosity in three embryonal tumours suggests a common pathogenetic mechanism. *Nature* 1985, **316**, 330–334.
6. Reeve AE, Sin SA, Raizis AM, Feinberg AP. Loss of allelic heterozygosity at a second locus on chromosome 11 in sporadic Wilms tumour cells. *Mol Cell Biol* 1989, **9**, 1799–1803.
7. Maw MA, Millow LJ, Eccles MR, *et al.* Chromosome 16q allele loss in Wilms' tumour. *Cancer Res* (in press).

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Triple Malignant Neoplasms in a Patient with Adult T-Cell Leukaemia

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WE HAVE previously reported multiple primary neoplasms in patients with adult T-cell leukaemia (ATL) [1]. We here report a patient who developed four malignancies including ATL.

In 1984, the patient underwent a gastrectomy for gastric cancer (tubular adenocarcinoma). In 1985 a colonectomy was performed for colon cancer (well-differentiated adenocarcinoma). He was subsequently investigated for leukocytosis (white blood cell count 23 800/μl). By two-colour fluorescence, monoclonal proliferation of CD4$^+$, CD29$^+$, CD45RA$^-$ leu-

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