Metastatic Germ Cell Tumor Presenting with Renovascular Hypertension

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Dear Sir,

Renovascular hypertension (RVHT) is encountered not infrequently after combination chemotherapy of testicular cancers, as cisplatin-based regimens could induce renovascular lesions leading to systemic hypertension [1]; however, a pretreatment-settled RVHT due to the primary involvement of renal vasculature is very uncommon [2] and not reported secondary to a metastatic germ cell neoplasm. A 21-year-old man had been diagnosed to have primary mixed germ cell tumor of testis after right inguinal orchiectomy, but he had refused subsequent evaluation and therapy. On readmission after 7 months, physical examination revealed a high blood pressure and a solid nontender mass beneath the left costovertebral angle. Metastatic workup disclosed a tumoral lesion surrounding left renal artery and vein (fig. 1). Urinalysis and laboratory examinations, including serum electrolyte levels and renal functions were normal except for a normochromic normocytic anemia (hemoglobin 5.41 mmol/l, hematocrit 0.31) and high lactate dehydrogenase levels (9.65 μ kat/l). Beta human chorionic gonadotropin, plasma renin activity, and active and total renin levels were also found to be elevated (fig. 2). PEB chemotherapy consisting of cisplatin, etoposide, and bleomycin was started. Although initially a favorable response was obtained with regard to blood pressure and tumor burden, he developed progressive renal failure after successive cycles of PEB (fig. 2), and he died after a stroke leading to respiratory arrest. No autopsy was allowed.

As there was no evidence of vasculitis, coagulopathy, cardiac lesion, or a history of radiotherapy, a vaso-occlusive theory or radiation-induced nephritis does not hold true for RVHT. The acceptable response to PEB therapy on the basis of a decline in blood

Fig. 1. Computed tomography scan of the upper abdomen demonstrating a hypodense mass with irregular contours, extending from the renal hilum to the aortic wall. The mass is almost completely occupying the renal hilum and involving renal vascular pedicle and left psoas muscle. Pulsatility and resistivity indices of the involved kidney were lower after Doppler ultrasonography. Hydronephrosis was not present, and there were no additional lesions in contralateral kidney, liver, spleen, adrenal gland, and lymph nodes. Note the absence of a

discernible interface between the tumoral mass and the surrounding renal vessels. Selective renal angiography with sampling for renin measurements could not be performed due to the probability of improper sampling or nonvisualization of renal artery and vein.

therapy combined with radiotherapy would provide better and prolonged responses as advocated by several authors and also observed in this case. It might be wrong to draw definite conclusions as the institution of a suitable therapy concerning the intricate pre- or posttreatment presentations of this unique neoplasm awaits more detailed and controlled studies.

pressure, lowered renin and beta human chorionic gonadotropin levels, and the reduction in tumor mass suggest the diagnosis of RVHT due to involvement of renal vasculature. Unusual RVHT prior to treatment with potential nephrotoxic and vascular drawbacks of chemotherapy [3] might be challenging; however, early and prompt debulking of the tumoral mass by surgery or chemo-

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Months 0

10-

110 o Systolic BP (mm Hg) A Diastolic BP (mm Hg)

D PRA (ng/ml/h) A β -hCG μ U/t

Fig. 2. Chronological plot of the patient's blood pressure levels (a), serum plasma re-nin activity (PRA) and beta human cho-ronic gonadotropin (β -hCG) levels (b), renal function (c), and mass size (d) against consecutive cycles of PEB chemotherapy and antihypertensive medications. a A decline of 45 ± 5.7 mm Hg was obtained in mean blood pressure levels. b The patient was uneventful with resolved hypertension, the pararenal mass reduced to 50% of its initial size, and the PRA levels within normal ranges at the end of the 3rd PEB cycle. c The renal function began to deteriorate after four cycles of PEB. Despite the supportive attempts, throughout the therapeutic cycles, the creatinine clearance was lowered to 2.2 ml/s. Serum blood urea nitrogen (31 mmol/l) and creatinine (0.42 mmol/l) levels were elevated with concurrent low K (0.4 mmol/l), Ca (1.7), and Mg (0.5 mmol/l) levels.

○ Creatinine clearance (ml/s)

D K (mmol/l)

A Creatinine (mmol/l $\times 10$)

Months 0

9x8 7x8 6x6 5x3 3x3

Months 0

L

PEB 1 PEB 2a PEB 3b PEB 4C

Enalapril

Nisoldipine plus atenolol

○ Tumor size (cm)

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