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EDİTÖRE MEKTUP / LETTER TO THE EDITOR

Elevated human chorionic gonadotropin levels in a boy with chronic kidney disease

Kronik böbrek hastalığı olan erkek çocukta artmış insan koryonik gonadotropin seviyeleri

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To the Editor,

Human chorionic gonadotropin (HCG) is an important tumor marker in the diagnosis and monitoring of treatment response in childhood cancers such as germ cell tumors and liver tumors. The effect of chronic kidney disease (CKD) on increased HCG is not a well-researched issue. HCG elevation of unknown cause not only causes diagnostic confusion, but also causes economic loss by necessitating many unnecessary examinations. In the current report a boy with elevated HCG levels with chronic kidney disease is presented. An informed consent was taken from his parents for publication of this report.

A 10-year-old boy, who was being followed up for CKD, was seen in the pediatric oncology department, where he was consulted for increased HCG. Despite aggressive research, the high HCG levels could not be explained. On physical examination, the patient's weight was 29 kg (25-50 percentile), height 132 cm (25-50 percentile), body temperature 36°C, pulse 105 /min., respiration 21/min. and blood pressure was 90/60 mmHg. His general condition was good, he was conscious and cooperative, and his systemic examination was normal. From laboratory tests, BUN 55 mg/dL, creatinine 2.99 mg/dL, Na 136 mEq/L, K 4.2 mEq/L, albumin 4.1 g/dL, Ca 9.8 mg/dL, P 5.5 mg/dL, LDH 224 U/L, uric acid 5.5 mg/dL, Hb 10.2 g/dL, leukocyte 10400/mm^3, platelet 3260000/mm^3 were found. In the complete urinalysis, the density was 1006, pH 6, protein negative and microscopic evaluation was normal. HCG values measured at 3-month intervals were 7.52, 5.79, 8.81, 12.71 and 10.35 mIU/ml (0-0.5). AFP was within normal limits. Total testosteron was 0.025 ng/ml (2.41-8.27), LH 17.81 mIU/ml (1.24-8.62) and FSH 78.43 mIU/ml (1.27-19.26). There were no pathological findings except bilateral hypoplasic kidneys in USG and CT.

The role of CKD as a cause of elevated HCG levels has not been well researched. It is well known that one third of the HCG produced is excreted and a part of it is metabolized by the kidney[1]. There was no organomegaly or mass finding in the physical examination and radiological imaging of our patient. Only bilateral hypoplasic kidney was observed. Laboratory examinations revealed low testosterone levels and an increase in LH and FSH in addition to increased HCG. Since the increase in HCG is not a finding that can be explained by the tumor, it was thought that the patient's pre-existing chronic disease might be responsible for this situation. Therefore, the presence of CKD in our patient may have served to increase HCG levels. It is not known why some CKD patients have high HCG and others have normal levels. The hypothesis that severe kidney disease as in

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the presented case has an effect on HCG remains to be proven. Hypogonadism is a clinical condition often described in individuals with chronic kidney disease[2]. Decreased testosterone concentration with increased levels of gonadotropins, including LH and FSH, has been attributed to a wide variety of factors. Under these circumstances pituitary overproduction of HCG in parallel with LH and FSH also seems reasonable. Elevation of some tumor markers like CEA, HCG, CA 19-9, and CA 15-3 was reported in patients with CKD without a malignant disease [3]. Elevation in serum tumor markers may be due to the decrease in the excretion of these markers because of the decreased glomerular filtration rate in CKD patients. Therefore, caution is required in interpreting serum tumor markers as representative of underlying malignancy in patients with CKD.

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