patients on MFP are inaccurate indices of systemic PG and/or GC deficiency or excess. However, the potential impact of MFP use on other serum steroid assays has not been widely studied. We report the case of a 55 yr old man on treatment with MFP for adrenal CS found to have striking artefactual changes in serum androgen and estrogen levels. Case Summary: A 55 yr old African American man was referred with poorly controlled type 2 diabetes, hypertension, hyperlipidemia, obesity and untreated hypogonadism. He had a 3.2cm left adrenal incidentaloma associated with adrenal CS and he chose medical management rather than surgery. Upon starting MFP at 300mg QD serum total estrogen (by radio-immunoassay; RIA) and estradiol (by chemiluminescence immunomassay; CIA) were markedly elevated while serum total, free, bioavailable testosterone and dihydrotestosterone were all markedly reduced. His HBA1c, weight and energy levels improved on MFP despite these findings. The serum steroid levels normalized to pre-treatment levels after stopping MFP for ~ 4 weeks but the changes recurred after restarting therapy. After MFP dose escalation to 300mg BID the serum steroid levels normalized after stopping MFP for ~ 6 weeks. The artefactual low testosterone levels also occurred with measurement by equilibrium dialysis but “normal accurate” results were obtained when measured by liquid chromatography-Tandem mass spectrometry (LC-TMS). He remains on MFP 300mg BID without need for androgen repletion. Discussion: With increased use of MFP for CS, indices for tracking its clinical and biochemical effects assume great importance. There are few reports of the possible effects of MFP on estrogen and testosterone serum assays despite its touted low cross reactivity with sex steroids. Our case suggests that the significance, extent and prevalence of artefactual changes on serum sex steroid assays may be underestimated and under-appreciated. Conclusions: Our case of wide disparities in serum estrogen and androgen measures in a patient on MFP indicates that caution needs to be exercised in the interpretation of such results in patients on current MFP therapy. Our clinical observations suggest depending on the dose that a wash out period of 4–6 weeks is required to ensure accurate measures. Studies to ascertain the prevalence of this artefactual effect are needed and it appears testosterone measurement by LC-TMS obviates the testosterone assay artefact.

Adrenal

ADRENAL CASE REPORTS

Mixed Corticomedullary Tumors of the Adrenal Gland Harboring Both Medullary and Cortical Properties
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Adrenal cortex and medulla are derived from mesoderm and ectoderm, respectively. Mixed corticomedullary tumors (MCMTs), comprising an intimately admixed population of both adrenal cortical cells and pheochromocytes in a single adrenal tumor, are extremely rare and its pathogenesis has remained unknown. Here, we report a case of MCMT whose cells co-expressed cortical and medullary antigens in the same tumor cells. Case description: A 40-year-old woman was referred to our hospital for investigating Takotsubo cardiomyopathy following resection of uterine fibroids. An abdominal CT scan depicted a 24 mm tumor on her left adrenal gland. Her basal serum ACTH, cortisol levels and urinary cortisol were 13.8 pg/mL, 9.5 μg/dL, and 26.5 μg/day respectively. The cortisol level was normally suppressed by an administration of 1 mg dexamethasone (1.4 μg/dL). Plasma renin activity, aldosterone levels and urinary aldosterone were 15.0 ng/mL/h, 122 pg/mL, and 5.0 μg/day, respectively (with administration history of azosemide). On the other hand, her plasma adrenaline and noradrenaline levels were elevated as high as 177 pg/mL and 536 pg/mL, and urinary metanephrine and normetanephrine were 2.12 μg/day and 1.10 μg/day. A 123I-metaiodobenzylguanidine scan revealed high uptake in the tumor. After adequate adrenergic α-receptor blockade, left adrenalectomy was performed. Her postoperative endocrine and clinical findings were normalized without any further complications. Pathology: Immunohistochemistry (IHC) revealed the presence of MCMT. Cells morphologically consistent with pheochromocytoma and adrenocortical cells were confirmed by immunostaining of chromogranin A and SF-1, respectively. Chromogranin A-positive medullary-derived and SF-1-positive cortical-derived tumor cells were intermixed in the chimeric fashion. In addition, some tumor cells were positive for both proteins, indicating hybrid nature of the cells. Tumor cells of cortical origin expressed CYP11B1, 3β-HSD, p450c21, and p450c17, but not CYP11B2. Non neoplastic adrenal cortex were atrophic, whereas the glomerulosa was hyperplastic positive for CYP11B2, consistent with diffuse hyperplasia and adrenal medullar unremarkable. Conclusions: The adrenal tumor was clinically diagnosed as pheochromocytoma, but the pathological findings did reveal cortisol production in the tumor and aldosterone overproduction in the accompanying cortex. This is the first case of MCMT co-expressing adrenal medullary and cortical antigens in the same tumor cells as hybrid cells.