250-1100), LH 8.2 MIU/ml (NL= 1.2-8.6), FSH 4.3 MIU/ml (NL=1.3-19.3), TSH 17.8 uIU/ml (NL= 0.45-5.33), free T4 0.62 ng/dl (NL= 0.58-1.64), free T3 3.05 pg/ml (NL=2.5-3.9), TPO antibody (AB) negative, IGF-1 41 ng/ml (NL=53-331), AM cortisol 8.2 ug/dl (NL=6.7-22.6), ACTH 99 pg/ml (NL=6-50), aldosterone 3 ng/dl (NL ≤28), renin activity 2.78 ng/ml/h (NL=0.25-5.82), 21-hydroxylase AB negative, 17-hydroxyprogesterone 54 ng/dl (NL=42-196), DHEA-S 88 mcg/dl (NL=106-464)]. ACTH stimulation test (250 mcg) was performed with basal cortisol 8.0 ug/dl (NL= 5.0- 21.0), basal ACTH 93 pg/ml (NL=6-50), 30min cortisol 9.7 ug/dl (NL=13.0-30.0), 60min cortisol 10.2 ug/dl (NL=14.0-36.0). Pt was diagnosed with primary AI and started on hydrocortisone. CT abdomen and pelvis with contrast showed normal adrenal glands.
Discussion: Physicians should be cognizant of a unifying diagnosis in a syndrome of such a broad presentation. Endocrinopathies vary in extent of dysfunction and may fluctuate during the course of POEMS syndrome. Multidisciplinary management as well as regular reassessment of endocrine function is integral in managing this complex disease.

Thyroid

BENIGN THYROID DISEASE AND HEALTH DISPARITIES IN THYROID II

Tyrosine Kinase Inhibitors Induced Thyroid Dysfunction: An Experience from a Tertiary Care Hospital

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SUN-414

Tyrosine kinase inhibitors (TKI) belong to a new class of molecular multi-targeted anticancer therapy which targets different growth factor receptors and hence attenuates cancer cell survival and growth. TKI-induced thyroid dysfunction is recognized as a common adverse effect of treatment., but the onset of thyroid dysfunction is variable. This study analysed correlation between initiation of TKIs and the onset of thyroid dysfunction in non-thyroid cancers patients without any background thyroid dysfunction.
METHODS:
This was a retrospective cohort study to evaluate thyroid dysfunction in adult patients (n=227, M:F=153:74) with non-thyroidal cancers treated with TKIs. Patients having pre-existing thyroid disease including euthyroid goitres were excluded. Demographic, clinical, and cancer treatment data were collected. Thyroid function tests (TFTs) were done prior to initiation, at 2 months, 6 months and at 1 year. TFTs were classified as euthyroid (thyrotropin [TSH] normal), subclinical (SCH; TSH 5-10 mIU/L, or higher TSH if free thyroxine normal), or overt hypothyroidism (OH; TSH >10 mIU/L, low free thyroxine, or requiring replacement).
RESULTS:
Of the 227 patients in the study, OH occurred in 57 patients (25.1%)(M:F = 19:38) and SCH occurred in 89 patients (39.2%) (M:F=39:50) with TKI therapy at the end of 12 months. 37 patients (M:F=13:24) developed OH in first 6 months after initiation of TKIs. Female patients were more likely to have OH in the first 6-month period following TKIs irrespective of type of TKI or the cancers. SCH was also more common after 2 months in female patients (n=23) (M:F=6:17) but the conversion of SCH to OH was more common in male patients at the end of 12 months. The symptoms were variable and most the patients did have any thyroid specific symptoms. After adjustment for age, sex, cancer type, cancer stage, performance status, and type of TKI, OH remained significantly associated with survival at 1-year (hazard ratio=0.461; $p<0.0001$), whereas SCH did not (hazard ratio=0.591; $p=0.165$). Analysis of hypothyroid patients (SCH and OH) with TSH >5 and <10 mIU/L stratified by hormone replacement status showed improved survival associated with hormone replacement, although 1 year follow-up is too short to comment on overall survival rates.
CONCLUSIONS:
New onset hypothyroidism, both OH and SCH is common in non-thyroidal cancer patients treated with TKI. SCH is more common after 2 months and OH after 6 months following TKI initiation. Female sex is more predisposed to develop thyroid dysfunction irrespective of underlying cancer or type of TKI used but male patients progressed to OH at the end of 12 months.

Adrenal

ADRENAL CASE REPORTS I

Right Adrenal Mass: An Unusual Presentation

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SAT-183

Background: Adrenal masses may be incidentally found on imaging done for other reasons. The prevalence is 4.4% and up to 10% in older patients. Malignancy is an uncommon cause in patients without a known diagnosis of cancer. The frequency of primary adrenal carcinoma in patients with adrenal incidentalomas is approximately 2.0 to 5.0%; another 0.7 to 2.5% have non-adrenal metastases to the adrenal gland.
Clinical Case: 54-year-old man with Hepatitis C, prior alcohol abuse, and cirrhosis was found to have an increase in the alpha-fetoprotein (AFP) level from normal to 244 ng/ml (nl<15.1) over a 6-month period. Liver MRI was consistent with a cirrhotic liver without focal enhancing lesions and showed a new indeterminate 7.6 cm right retroperitoneal lesion arising from the adrenal gland compared to a prior CT of the abdomen a year early. Further imaging confirmed a 9.6 x 9 x 7.6 cm heterogeneously enhancing right adrenal lesion with a necrotic center, concerning for a primary malignancy; up to 11.1cm a month later. Patient referred to Endocrine for further evaluation. There were no symptoms suggestive of Cushing's, pheochromocytoma or primary hyperaldosteronism. On exam there were no hypertension, dorsal fat pad, supraclavicular fullness, skin thinning or purplish striae. Biochemical workup was consistent with a non-functioning adrenal mass. DHEA-S was 11 (38-313

mcg/dl). CT-guided core needle biopsy of right adrenal gland was consistent with metastatic hepatocellular carcinoma. CT pelvis with contrast re-demonstrated the right adrenal mass now measuring 11.4 x 10 x 10 cm with new enlarged retrocaval lymph node and no focal arterially enhancing lesions. During embolization of adrenal lesion/hepatic angiogram, multiple liver lesions not previously identified were reported with the largest of 2.9cm size and enhancing lesions in the sacrum and bilateral iliac bones; decrease in size of the necrotic right adrenal mass measuring 8.2 x 9.1 x 9.1 cm 1-month post-embolization. Patient following with Oncology.
Conclusion:Unilateral isolated adrenal metastasis from occult hepatocellular carcinoma (HCC) is extremely rare. Adrenal gland is the second most common site of hematogenous spread from HCC after the lung and has been found in up to 8.4% of cases at autopsy. In our case, the adrenal metastasis was the first clinical presentation of HCC with no evident hepatic lesion until 9 months of adrenal finding; few cases have been reported. Fine needle aspiration/needle biopsy of suspected malignancy is useful to detect primary tumor in case of metastatic disease that is silent at this stage. Adrenal metastasis in HCC are seldom treated by surgery as by the time of diagnosis the tumor is usually far advanced and/or patients are poor surgical candidates. This case highlights the importance of suspecting underlying HCC in isolated adrenal mass in a patient with high risk factors.

Reproductive Endocrinology

MALE REPRODUCTIVE HEALTH - FROM HORMONES TO GAMETES

The Short-Term Effect of Multiple Kinase Inhibitor (Lenvatinib) on Spermatogenesis in Mice

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SAT-039

Lenvatinib, a multi-kinase inhibitor, is used in the treatment of solid malignancies. Lenvatinib belongs to a family of tyrosine kinase inhibitors and targets VEGF receptors 1-3, FGF receptors 1-4, PDGF receptor alpha, RET and KIT. However, it is not known whether Lenvatinib like other chemotherapeutic drugs affects spermatogenesis. The objective of this study was to examine whether Lenvatinib induces damage to spermatogenesis in mice. Twenty adult mice (C57BL/6) were randomly divided into 2 groups to receive daily gavage of either water (as control) or Lenvatinib (10 mg/kg) for 6 weeks. All mice were euthanized at the end of the study. We identified that Lenvatinib significantly ($p<0.05$) decreased testis weight (TW: 91.75±1.49mg) compared to control mice (TW: 111.9±3.07mg). This difference in testis weight however, became non-significant after correcting for body weight. The cauda epididymal sperm count was significantly ($p<0.01$) decreased in the Lenvatinib treated (0.82±0.04 million/mg cauda) as compared to control (1.26±0.07 million/mg cauda) mice. There were no differences in plasma testosterone concentrations between Lenvatinib treated (29.76±7.67ng/dl) and control (31.72±6.89ng/dl) mice. Lenvatinib did not induce notable morphological changes in testicular histology. We conclude that 6 weeks of Lenvatinib treatment had minimal effect if any on mouse spermatogenesis. The long-term treatment effect of Lenvatinib on spermatogenesis remains to be determined.

Adrenal

ADRENAL - TUMORS

New Data on High Prevalence and Time of Occurrence of New Onset Hypothyroidism Associated with Mitotane Therapy in a Cohort of Adrenocortical Cancer

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SAT-158

Context. Mitotane is a steroidogenesis inhibitor and an adrenocorticolytic drug used to treat adrenocortical cancer (ACC). Central hypothyroidism is recognized in mitotane-treated patients and recent data suggested that mitotane could have an inhibitory effect on TSH-secreting cells in the pituitary gland. Moreover, mitotane may lead to induction of thyroid hormone metabolism. Clinical data on hypothyroidism related to mitotane such as prevalence and time of occurrence was described in a limited number of patients.
Objective. To better characterize clinically secondary hypothyroidism in patients with ACC treated with mitotane therapy.
Methods. We reviewed retrospectively paper charts and electronic records from patients with histologically confirmed diagnosis of ACC evaluated at our center from 1995-2019. We analysed the pattern of TSH and thyroid function, but also mitotane timing and levels at baseline and during treatment of patients under mitotane therapy. Thyroid hormone assessment including TSH, FT4 and FT3 was performed at least every 3 months during follow-up.
Results. Our cohort of 104 patients with ACC includes 84 patients that received mitotane therapy. Among them, thyroid function data was incomplete for 39 cases. Complete data was retrieved from 45 patients. Ten out of 45 (22.2%) patients were already known for primary hypothyroidism and were receiving L-T4 replacement before the initiation of mitotane. Two of 45 (4.4%) patients maintained a normal thyroid function during complete follow up (4.5 years) and 33/45 (73.3%) had new onset hypothyroidism requiring levothyroxine treatment. Of these 33 patients, 22 were females and 11 were males, ranging from 22-74 yo with a median of 46 yo. The number of patients with ENSAT stage I, II, III and IV of disease were 1, 8, 11 and 13 respectively. Thyroid profiles were compatible with central hypothyroidism (low T4 with low or inappropriately normal TSH) in 22/33 patients (66.7%). Interestingly, 6/33 patients (18.2%) developed a TSH elevation with a normal lower-limit or low T4 level. The timeline distribution of the occurrence of hypothyroidism was 21.2% (n:7) at <3 months, 15.2% (n:5) between 3-6 months, 21.2% (n:7) between