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Abstract: Recent studies have shown that obesity is not a homogeneous condition and that there is a subgroup of people with obesity, but without metabolic disturbance. This phenotype of obesity is called "metabolically healthy obesity" (MHO) [1]. More and more data are appearing in the scientific literature, indicating that quantitative and qualitative changes in the gut microbiota (GM) can be a trigger in the development of obesity and metabolic disorders [2]. In order to study the role of GM in the development of various types of obesity, 37 patients were examined, divided into 3 groups: group 1 (n = 11) - healthy people without obesity and overweight (control), group 2 (n = 13) - patients with MHO, group 3 (n = 13) - patients with metabolically unhealthy obesity (MUHO). The basic metabolic parameters were determined for all of them and a quantitative assessment of the condition of the GM was performed using the Real-time PCR method. Results: 1. In people from the control group (group 1) in the GM, compared with formal normative indicators, the number of Lactobacillus spp., Bifidobacterium spp., B. thetaiotaomicron was reduced ($p<0.05$) and indicators of the total bacterial mass and Enterobacter spp./Citrobacter spp. were increased ($p<0.05$). 2. In subjects with MHO (group 2), GM changes similar to group 1 were observed. However, in comparison to group 1, Klebsiella spp. and Proteus spp. were recorded in feces in quantities exceeding the formal regulatory. In patients with MUHO (group 3), in addition to changes detected in group 2, C. difficile was found in feces, as well as a significant ($p <0.05$) decrease in F. prausnitzii and an increase ($p <0.05$) in the detection frequency of banal E.coli, as well as the more diverse composition of the microbiota. Thus, the data obtained as a result of a pilot study certainly indicate changes in the GM in people with different phenotypes of obesity and in healthy ones. Further study of the GM in patients with various types of obesity, but in a larger groups, is required.
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Neuroendocrinology and Pituitary

CASE REPORTS IN CLASSICAL AND UNUSUAL CAUSES OF HYPOPITUITARISM II

Adenocarcinoma Pituitary Metastasis with Suprasellar Extension

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Background
Metastasis to the pituitary gland (MP) is an infrequent clinical problem, however, during the last few decades, MPs is increasing in frequency, due both to the improved cancer survival rates and availability of better imaging techniques. Breast cancer, lung cancer and lymphoma are the most common primary sites. MP mimics pituitary adenoma making the diagnosis difficult, especially when clinical evidence of the primary malignancy is absent. We report a case of histologically confirmed pituitary metastasis (adenocarcinoma) from carcinoma of unknown primary, leading to and presenting as panhypopituitarism.
Case details
54 year male HBV carrier, asthmatic, non smoker, non alcoholic was seen elsewhere with complaints of increased frequency of micturition associated with nocturia, increased thirst, asthenia and weight loss of 05 kg since 06 months. He also had loss of libido and erectile dysfunction. Patient never experienced headache, vomiting or visual disturbances. There was no personal or family history of malignancy. On physical examination skin was pale, atrophic with fine wrinkles around the eyes. He had BP of 96/60mmHg and PR 100/min but system examination was otherwise unremarkable. Biochemical evaluation confirmed diabetes insipidus, secondary hypothyroidism, adrenal insufficiency and hypogonadism. MRI Brain showed a mass like thickening (11.6 x 11 x 16mm) of the infundibulum and posterior portion of the pituitary gland with upward displacement of optic chiasm. Workup for granulomatous conditions (sarcoidosis/ disseminated Koch's) was negative. He was initiated on desmopressin, thyroxine, hydrocortisone, testosterone and managed elsewhere as hypophysitis.
At presentation to us after 3 months of treatment, visual field examination showed decrease in peripheral vision involving right superior temporal quadrant and an attempt was made to delineate the size and etiology of the lesion. Repeat imaging revealed increase in size of the lesion to 12 x 15 x 18mm. He underwent TSS and the lesion was biopsied. Histopathology showed infiltrative adenocarcinoma with CK20 +, CK7 -, and GATA3 + on IHC. Post procedure WB-PET CT, showed metabolically active residual tumor in suprasellar region and negative for metabolically active disease in other areas. Upper GI Endoscopy, Colonoscopy and Bronchoscopy were also normal. Currently he is on adjuvant radiation therapy for residual suprasellar lesion.
Conclusion
Pituitary metastasis may be difficult to differentiate from other lesions in the sellar/parasellar region, presentation as DI and thickened stalk confounded the diagnosis, given that it is a feature of the commoner lesion- hypophysitis. This case reports the rare occurrence of CK20 positive, CK7 negative adenocarcinoma metastasis to pituitary and no evidence of primary.