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CUSHING SYNDROME AS A PRESENTATION OF PARANEOPLASTIC SYNDROME

Medicine

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ABSTRACT

We are presenting an interesting case of 48-year-old male who presented with fatigue ,proximal muscle weakness ,myalgia ,polyuria ,polydipsia since 1 month. He had no known comorbidities, no significant drug history or medical/surgical history, neither alcoholic nor other addictions. On examination his blood pressure was 160/100mm of Hg and rest examination was normal. On evaluation his potassium was 1.5, sugars were 280mg/dl, arterial blood gas was suggestive of metalbolic alkalosis (PH 7.59, bicarbonate 37.6mmol/lit) and serum calcium was 6.8mg/dl. Patient was receiving 100 to 120 mEq of KCl every day still his potassium was around 2.5 mEq.

KEYWORDS

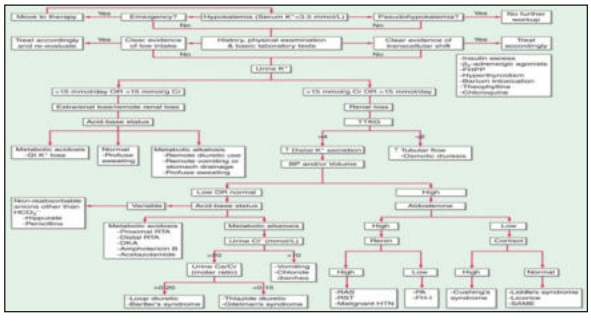
SERUM ELECTROLYTES over course in the hospital

	15/2/21	17/2/21	18/2/21	21/2/21	23/2/21	26/2/21	27/2/21	4/3/21	9/3/21	16/3/2
Ng	142	141	139	141	143	140	144	139	137	138
K	1.5	1.8	1.4	1.8	1.5	2.6	1.5	2.6	5.5	4.4
Cl	95	100	97	96	96	101	97	100	103	104

NOTE- 100 to 120 milliequivalents of potassium was given every day

His urine potassium was 14 mmol/day which was again not very conclusive. TTKG couldn't be calculated as patient's urine osmolality was 270 mosm/kg (less than 300). But no extrarenal or remote cause of potassium loss was established.

For raised sugars we started him giving insulin and his insulin requirement was increasing. He was receiving gliclazide 240mg, metformin 2g and 80 units of insulin per day, still his sugar control was poor. His glycated hemoglobin was 8% which was contradictory.

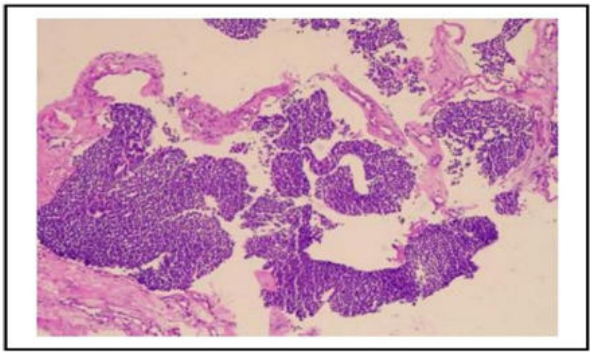


In view of uncontrolled diabetes, decreased plasma renin activity along with hypokalemia, metabolic alkalosis, raised blood pressure Cushing's was suspected. Serum Cortisol 8 AM turned out 43.7microgram/dl (5 to 23), serum cortisol 4pm- 49.2 microgram/dl (3 to16),serum ACTH – 206 Pg/ml (7.2 – 63.6)which were significantly high. MRI brain was done which showed normal pituitary fossa. Serum prolactin levels were normal. Suspicion of ACTH secreting paraneoplastic tumour was raised. Contrast enhanced CT scan of abdomen showed variable sized hypo dense heterogeneously enhancing soft tissue lesions in liver, could be of neoplastic etiology likely metastasis. PET CT showed multiple hepatic lesions in right lobe of live, probably multifocal hepatocellular carcinoma. There was no abnormal hypermetabolism elsewhere in the body in PET scan. As no hyper metabolism was found in PET except liver, primary hepatic neuroendocrine tumour was suspected. Alpha- fetoprotein, ca19-9 and serum CEA was sent which turned to be insignificant.

Liver biopsy with immunohistochemistry suggested neuroendocrine origin of the tumour which was further confirmed significantly elevated 24 hour urine chromogranin levels (1502 ng/ml).

Diagnosiss:

ACTH SECRETING PRIMARY HEPATIC NEUROENDOCRINE TUMOR (PHNET) was established.



Similar cases of PHNET are published in international journals but probably first of this kind in India.