PREGNANCY INDUCED CUSHING'S SYNDROME

CE Andreescu[1], LJ Hofland[1], J Hofland[1], LHJ Looijenga[2], WW de Herder[1] and RA Feelders[1]

1 Department of Internal Medicine, Division of Endocrinology, Erasmus MC - University Medical Center, Rotterdam, The Netherlands,
2 Department of Pathology, Erasmus MC - University Medical Center, Rotterdam, The Netherlands.

INTRODUCTION

Misdiagnosis of Cushing syndrome (CS) is common because of the overlapping features of fatigue, weight gain, striae and emotional changes that occur during normal pregnancy.

The clinical presentation together with laboratory and imaging findings help to make a diagnosis. However, changes in maternal hormones and their binding proteins complicate assessment of the normal level of glucocorticoid hormones during gestation.

CS during pregnancy is attributable most frequently to an adrenal adenoma and to a lesser degree to adrenocorticotropic hormone hypersecretion from a pituitary adenoma. Furthermore aberrant expression of various hormone receptors in the adrenal glands have been suggested to be involved in the pathogenesis of this condition, in particular the luteinizing hormone receptor.

METHODS

We investigated and treated three pregnant women with ACTH-independent Cushing's syndrome and an adrenal tumor.

After uncomplicated delivery patient 1 underwent in vivo testing for aberrant hormone receptor expression by the adenoma.

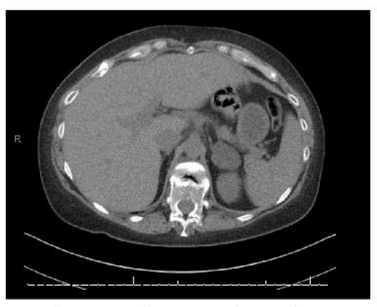
Adrenal tumor tissue of patient 1 and 2 were staind immunohistochemical for LH receptors.

RESULTS

Patient 1	Diurnal cortisol rhythm (nmol/l)	Salivary cortisol (nmol/l)	UFC (nmol/24h)	1 Mg-DST (nmol/l)	ACTH (pmol/l)
During pregnancy	690 - 1070 - 1030	Not measured	1380	360	< 1.1
After delivery	320 - 596 - 280	18 - 12 - 47 - 64	582	210	< 1.1

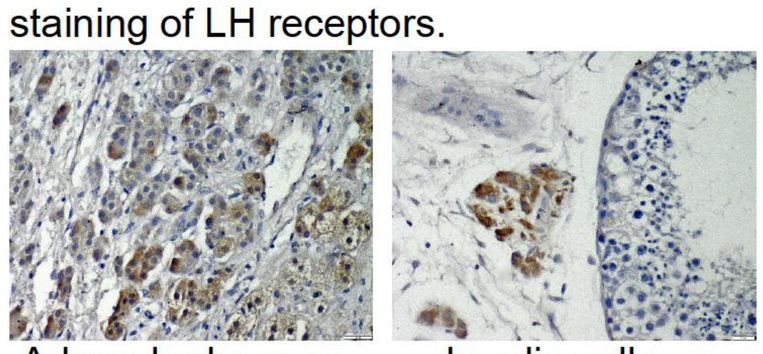
Cortisol response was found after administration of:
- LHRH (211%) en hCG (155%),

- ACTH (213%),
- TRH (133%),
- Standard mixed meal (392% and 140%),
- Glucagon (347%),
- Vasopressine (198%).



CT scan of patient 1 - adrenal adenoma

Adrenal tumor tissue of patient 1 and 2 showed positive immunohistochemical staining of LH receptors.



Adrenal adenoma Leydig cells

CONCLUSIONS

Considering the cortisol responses to LHRH and hCG, and the development of CS during pregnancy in these patients, it is likely that ACTH-independent hypercortisolism was induced by the pregnancy-associated rise in hCG levels that activated aberrantly expressed LH receptors in the adrenal adenoma.

Remarkably adrenal adenomas may simultaneously express multiple aberrant receptors and individual ligands may play a role in the regulation of cortisol production responsible for pregnancy induced CS.

References

Feelders et al., JCEM, 88(1):230-237

Lacroix, et al., Endocrine Review, 22(1):75-110

Lindsay et al., JCEM, 90(5):3077-3083

Mazzuco et al., JCEM, 91(1):196-203

17 ECE 37--EP

Adrenal

Corina Andreescu

DOI: 10.3252/pso.eu.17ece.2015

Poster presented at: ECE2015 16-20 May 2015 DUBLIN, IRELAND 17th EUROPEAN CONGRESS OF ENDOCRINOLOGY