ENDOCRINE SOCIETY

ENDO 2020

MARCH 28–31, 2020 SAN FRANCISCO, CA

GUIDE TO WRITING A CASE REPORT ABSTRACT

SUBMISSION DEADLINE EXTENDED: THURSDAY, NOVEMBER 7, 2019 1:00PM EST US

GUIDELINES FOR CASE REPORT ABSTRACTS

Eligibility

A Case Report abstract should provide valuable teaching points or learning lessons. Case Report abstracts are typically accepted as poster presentations (sometimes as oral presentations). Case Report abstracts that do not provide meaningful teaching points will not be accepted.

Title

The abstract title should emphasize the clinical condition and main teaching point.

Format

Case Report abstracts must be submitted in the following structured format:

- Introduction or Background
- Clinical Case (including diagnostic evaluation, treatment, and follow-up)
- Clinical Lesson(s) or Conclusion(s) (emphasizing the learning point[s] and implications for clinical practice)

Abbreviations

Abbreviations that are familiar to endocrinologists (eg, PCR, GHRH, TSH, etc) may be used without explanation.

Laboratory Values

For laboratory parameters, the units of measurement and normal ranges must be provided.

Statements

Avoid making statements about ongoing studies or pending results.

References

References are not necessary; keep them to a minimum.

EXAMPLE OF AN OUTSTANDING CASE REPORT ABSTRACT

ACTH-Independent Macronodular Adrenal Hyperplasia and Histamine-Induced Cortisol Secretion: In Vivo and In Vitro Studies

Maarten O van Aken, Richard A Feelders, Wouter W de Herder, Alberto M Pereira, Neveen A Hamdy, Steven W Lamberts, Aart Jan van der Lely, Leo J Hofland, Johannes A Romijn
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Background: In several animal species, histamine (H) has been shown to stimulate adrenal steroid secretion. In humans, however, this has not been reported yet.

Clinical Case: A 51-year women showed typical symptoms of Cushing syndrome (CS). Initial tests were consistent with ACTH-independent CS: elevated 24 hr urinary cortisol (449 and 517 nmol/24 hr, n<220 nmol/24 hr), abnormal

1 mg dexamethason overnight test (cortisol after 1 mg dex 620 nmol/l, n<50 nmol/l), elevated midnight serum cortisol (670 nmol/l, n<220 nmol/l), ACTH-concentrations below detection (< 4 ng/l). Abdominal CT-scan showed bilateral macro- nodular adrenal hyperplasia (diameter adrenal glands right 3 cm, left 2 cm). A screening protocol, as proposed by Lacroix et al. (1), revealed no change in cortisol secretion in response to LHRH, TRH, food, posture, metoclopramide, or cisapride.

Unexpectedly, an increased 24 hr urinary N-Methyl-Histamine (N-M-H) excretion was found (2623 and 2720 µmol/mol creat, n < 200 µmol/mol creat). Bone marrow biopsy did not show mastocytosis. Evaluating a possible link between H and cortisol, a single i.v. dose of H1 and H2-antagonists (clemastine 4 mg and ranitidine 300 mg) did not affect serum cortisol concentrations. The patient was treated by laparoscopic biadrenalectomy. Histological examination showed macronodular hyperplasia of the adrenal cortex. Of note, 24 hr urinary N-M-H fell to near normal level (370 µmol/mol creat), suggesting previous H-excess from adrenal origin. In vitro studies: Directly after laparoscopic removal, adrenal cortical tissue was processed to cell suspensions for further testing. H (10-7 M) increased cortisol production (131% of contr), similar to the effect of ACTH (615 pg/ml) (146% of contr). Ranitidine or clemastine completely aborted the H-stimulated cortisol production. In control experiments on hyperplastic adrenal tissue from patients with persistent Cushing disease, H (10-6 M) increased cortisol secretion in 1 of 6 subjects (189% of contr), with unaltered cortisol secretion in the other 5 subjects. Finally, mRNA from H type 1 receptor and H type 2 receptor was detected both in adrenocortical tissue from the patient and in normal or hyperplastic adrenocortical tissue (n=8), using a semi-quantitative PCR-technique.

Conclusion: This is the first case demonstrating the possible role of histamine in ACTH-independent Cushing syndrome with in vitro proof of histamine-induced cortisol secretion.

Reference: (1) Lacroix, A.,Ndiaye, N.,Tremblay, J.,Hamet, P. Ectopic and abnormal hormone receptors in adrenal Cushing's syndrome. Endocr. Rev. 2001;22;75-110.