GUIDE TO WRITING A CASE REPORT ABSTRACT

LATE-BREAKING ABSTRACT SUBMISSION DEADLINE: WEDNESDAY, FEBRUARY 17, 2016 1:00 PM ET US

Use the following guide and example when submitting a Case Report abstract. Visit endo2016.org for category details.

1. **Eligibility:**
   - Clinical Case Reports provide valuable teaching points or learning lessons
   - Clinical Case Reports will be considered for presentation – usually as a poster presentation
   - Examples of rare cases that do NOT provide additional meaningful teaching points will not be accepted

2. **Title:** The title of the abstract should emphasize the clinical condition and main teaching point.

3. **Format:** Clinical case report abstracts must be submitted in a structured format as follows:
   - Introduction or Background
   - Clinical Case (including diagnostic evaluation, treatment, and follow-up)
   - Clinical Lesson(s) or Conclusion(s)

4. **Conclusion:** The conclusion should emphasize the learning point(s) and implications for clinical practice.

5. **Abbreviations:** Abbreviations that are familiar to endocrinologists may be used without explanation (e.g., PCR, GHRH, TSH, etc.)

6. **Laboratory values:** For laboratory parameters, the units of measurement and normal ranges must be provided.

7. **Statements** about ongoing studies or pending results should be avoided.

8. **References** are not necessary and should be kept to a minimum.

**EXAMPLE OF OUTSTANDING CASE REPORT ABSTRACT**

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

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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 dexamethasone 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 macronodular 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 biadrenallectomy. 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.