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SYNDROME OF INADEQUATE SECRETION OF ANTIDIURETIC HORMONE ASSOCIATED TO PULMONARY THROMBOEMBOLISM: A NON-DESCRIBED CAUSE OF HYPONATREMIA IN THE LITERATURE.

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SUMMARY

Syndrome of inadequate secretion of antidiuretic hormone (SIADH) is a condition that can be associated to various sort of lung diseases.

In this report we present a clinical case where a SIADH associated to pulmonary thromboembolism was documented. As long as we know, this association has not been described yet in the literature, and we hypothesized that lung cytokines released due to pulmonary necrosis could be the main stimulus to vasopressin secretion in this entity.

Conclusion: Pulmonary thromboembolism should be incorporated in the list of lung causes of SIADH.

KEY WORDS: Syndrome of Inadequate Secretion of Antidiuretic Hormone (SIADH). Pulmonary Thromboembolism

RESUMEN

El síndrome de secreción inadecuada de hormona antidiurética (SIADH) es una condición que puede asociarse a diversas neumopatías.

En el presente reporte clínico se presenta un caso de SIADH asociado a un tromboembolismo pulmonar. Hasta donde sabemos, la asociación entre TEP y SIADH no había sido reportada aun en la literatura, y presentamos como hipótesis etiológica la liberación de citoquinas inductoras de la secreción de vasopresina, secundaria al daño parenquimatoso pulmonar del TEP.

Conclusion: El tromboembolismo de pulmón podría ser incorporado a las causas pulmonares de SIADH.

PALABRAS CLAVE: Síndrome de Secreción Inadecuada de Hormona Antidiurética. Tromboembolismo Pulmonar

INTRODUCTION

Syndrome of inadequate secretion of antidiuretic hormone (SIADH) is a condition which typically presents hypo-osmolar hyponatremia with normal extracellular liquid and high fractional excretion of urea ($\geq 55\%$) in the absence of hypothyroidism, cortisol deficit, renal failure, and hyponatremia inducing drugs ¹.

Among the causes of SIADH we find those related to various sort of lung diseases, nevertheless no report to date has included hyponatremia associated to pulmonary thromboembolism (PTE) as one of them². Thus, we present the following report where such association was documented

CASE REPORT:

72 year-old-female patient with a history of hypertension treated and normalized with low sodium diet (she had no need neither of diuretics nor other antihypertensive drugs); and multiple myeloma treated with thalidomide. She was hospitalized suffering from a sudden episode of dyspnea and pulmonary infiltration on the X-ray which was interpreted as secondary to PTE. Then this suspicion was corroborated by a pulmonary CT angio scan.

Together with the PTE diagnosis, it was also documented an acute hypo-osmolar hyponatremia in the absence of illnesses or drugs which could potentially cause such dysnatremia. Besides, she had no thoracic pain nor fever and it was not documented neither hypothyroidism, cardiac failure, nor cortisol deficiency in her analysis. Her serum glucose level and blood pressure were in normal range.

The patient was treated with anticoagulation (heparin) and water restriction as measures for her PTE and SIAHD respectively. After initiating that treatment, the patient's natremia was normalized and she was discharged from the hospital (Tables 1 and 2).

Table 1: sequence of the main analysis

(Normal Value)	Natremia (mmol/l) (135-145)	Plasmatic osmolality (mOsm/l) (280-300)	Urinary osmolality (mOsm/l) (50-1200)
Antecedent values	137	282	--
SIHAD	126	252	800
After water restriction	130	267	--
At discharge	136	280	--

Table 2: sequence of the main analysis

(Normal Value)	Hematocrit (%) (39 - 42)	Uremia (mg%) (20-40)	Creatininemia (mg %) (0.8-1.2)	EFU (%) (50)
Antecedent values	36	40	1.5	---
SIHAD	33	20	1.0	90 %
After water restriction	35	43	1.2	23 %
At discharge	36	40	1.7	---

EFU: fractional excretion of urea.

DISCUSSION:

It has already been reported some causes of SIAHD secondary to pulmonary diseases. In these settings, the mechanism proposed as the cause of this phenomenon would be the release of hyponatremia inducing cytokines by the damaged lung parenchyma. Among pulmonary diseases that were described as potential cause of SIHAD it can be found: pneumonia (viral, bacterial, tuberculosis, micotic), lung abscess, asthma, atelectasis, pneumo-thorax and fibrocystic diseases. Regarding pulmonary tumors, such as the small cell carcinoma, they could induce SIHAD by a paraneoplastic mechanism, which consist of an ectopic vasopressin secretion¹⁻³.

Pain can be another cause of vasopressin release⁴ but it was not present in this case report where the only PTE symptom was the sudden appearance of dyspnea.

Concerning the present described hypotranemia (associated to pulmonary thromboembolism), it could be caused by vasopressin secretion inducing cytokines released due to lung necrosis.

CONCLUSION:

Pulmonary thromboembolism could be considered another cause of SIADH secondary to lung damage.

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