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Last Days Stage or Something Else?

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ABSTRACT

Keywords: Myxedema; Endocrinology; Bradycardia

Introduction

Clinical History

A 94-year-old woman, currently institutionalized in socio-sanitary residence, dependent for basic activities of daily living, came into Urgencies department. As past medical history, we could find mild cognive disorder, hypertension, auricular fibrillation, chronic heart failure and chronic kidney failure. Actually, her treatment is bisoprolol, folic acid, digoxin, furosemide, formoterol/budesonide, forum, mirtazapine, sertraline, transdermic fentanyl, omeprazole and prednisone.

Current Disease

The patient is admitted from her residency due to worsening overall conditions, resting dyspnea, saturation at 90% with oxygen therapy and supraclavicular retraction the past two days and severe worsening the last two hours.

Clinical Examination

Blood pressure: 107/60mmHg, Heart rate: 30bpm, Temperature: 36.2ºC, Saturation 92%, Glasgow 15/15. Conscious, collaborative and alert, disoriented in time and space. Well hydrated, nourished and perfused. Supraclavicular retraction.

a) Cardiac Auscultation: Arrhythmic, no auscultation murmurs or extra tones.

b) Pulmonary Auscultation: Decreased vesicular murmur, fine binasal crackles.

c) Abdomen: Soft and depressible, not painful on palpation. I do not feel masses or mealies. No signs of peritoneal irritation. Negative dorsal cust-percussion.

d) Lower Extremities: Edema II/IV up to knees, peripheral pulses preserved, no signs of deep vein thrombosis. Pressure ulcer on the right heel.

Objective Tests

a) EKG: Atrial fibrillation with bradycardia at 32lpm, left axis.

b) Chest X-Ray: Cotton infiltrates in the right base.

c) Blood Test: Hemoglobin 11.3, MCV 101, Cr 1.41 similar to previous, CRP 2, proBNP 11000, rest anodyne.

Method and Evolution

Given the clinical situation of a patient with intense asthenia and bradycardia of 30 bpm, we started a slow perfusion of Isoprenaline, getting the rate back to 60-70 bpm. When the pump was finished, the patient became extremely bradycardic again, we observed atrial fibrillation with slow ventricular response at 29 bpm, so we decided to talk to her relative to explain the ominous prognosis we suspected. At the same time, we considered the reason for the symptoms, due to the lack of specificity of the results at the complementary tests obtained. Because of that, we extended the analytical tests with a complete ferric profile, requesting vitamin B12 and folic acid, that were normal, in addition to the thyroid profile, which showed TSH 94, T4 0.27 and T3 1.43. The patient is kept under observation, administered intensive serum therapy to promote the elimination of Bisoprolol and Digoxin from the blood circulation, adding also to treatment Levothyroxine and Hydrocortisone, managing to maintain the frequency around 60-70lpm and improving asthenia, so we admit the patient to Internal Medicine with continuous monitoring. On the ward, following this line of treatment initiated in the Emergency Department, with the support of Endocrinology, we achieved a progressive decrease in TSH and normalization of T4L, presenting in the last analysis TSH 4.4µUI/ml and T4L 1.25ng/dl on admission.

Conclusion

In the case of clinical instability in multipath logical patients, it is important to rule out the pharmacological cause in the first place and to consider less frequent alternative diagnoses. When we find highly complex patients whose treatment is rejected by the ICU, if we can offer them therapeutic alternatives we should initiate them, always making it clear to the patient and family members, if possible, that in the face of adversity they would not be candidates for aggressive measures, but that we would try to use the best possible individualized therapy. Support diagnosis and treatment of patients in other services, in order to seek a comprehensive approach to patients and directed to each case.

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