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Case Report

Report of Refeeding Syndrome in Chronic Obstructive Pulmonary Disease Elderly Patient

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Refeeding Syndrome (RS) is a life-threatening condition where the body is predisposed to a non-obvious multi-deficiency occurring with minerals shifts between the intra- and extracellular compartments. We used biochemical parameters and Naranjo probability scale. Herein we report the development of RS in a patient with chronic obstructive pulmonary disease, treated with parenteral nutrition (PN). In our patient cholesteric index (with an increase of gamma-GT and bilirubin) was observed, as well as the association between PN and clinical symptoms (Naranjo score 7). PN was stopped, replaced with nasogastric feeding at first and subsequently to a percutaneous endoscopic gastrostomy tube, with a time-related improvement of symptoms. Although PN represents an effective approach in patients that cannot be treated with enteral nutrition its use in elderly patient can be related to the development of adverse metabolic reactions.

Keywords: Refeeding Syndrome, elderly, parenteral nutrition.

INTRODUCTION

During starvation, low levels of circulating insulin are present, leading to an increased breakdown of lipids and proteins, starting the gluconeogenesis mechanism (Parli et al., 2014). The changing of fuel substrates from carbohydrate to fat leads to an increased turnover and depletion of vitamins. Therefore, misleading normal serum levels, in the total amounts of thiamine, magnesium, potassium and phosphorus can be possible. When rapid feeding of patients with pre-existing malnutrition is re-started, the reverse biochemical processes occur, causing biochemical derangements known as Refeeding Syndrome (RS) (Parli et al., 2014). RS is a life-threatening condition, characterized by low-serum electrolyte concentrations, fluid and electrolyte imbalance and disturbance of organ function resulting from over-rapid or unbalanced nutrition support (Walmsley, 2013). The early signs of RS are non-specific and include severely low-serum electrolyte concentrations of serum phosphate, potassium and magnesium which, if untreated, can progress to acute circulatory fluid overload, respiratory compromise and cardiac failure (Weinsier and Krumdieck, 1981; Rio et al., 2013). Herein we report a case of RS in a patient with chronic obstructive pulmonary disease (COPD) related to artificial nutrition support.

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Table 1. Parenteral Nutrition Scheme

Step	Drug
I	250 mL of glucose solution 33% + insulin retard 10 U.I. + 10 mL of amidolite + 0.5 mg of magnesium sulphate + 20 mEq of potassium chloride + 10% of calcium gluconate + 500 mg of C VitaminC with a flow of 30 mL/hour followed by 250 cc of glucose solution 33% + insulin retard 10 U.I. + 10 mL of amidolite + 0.5 mg of magnesium sulphate + 20 mEq of potassium chloride + 10% of calcium gluconate with a flow of 30 mL/hour
II	780 mL of solamin 7.5% at the flow of 32 mL/hour
III	500 mL of lipofundin at the flow of 21 mL/hour

CASE PRESENTATION

An 81-year-old man was admitted to the emergency room for shock and urinary incontinence. He was a heavy smoker (60 packs/years since 71), affected by blood hypertension, type 2 diabetes mellitus, benign prostatic hypertrophy, hiatus hernia, COPD grade IV Gold and type II respiratory failure. He was under long action β2agonist (LABA), long action muscarinic antagonist (LAMA), corticosteroids inhaler (CSI) and long-term oxygen therapy (LTOT). Clinical evaluation, biochemical findings and brain computer tomography led to diagnose a possible transitory ischemic attack. Domiciliary treatment with LABA, LAMA, LTOT and CSI was kept, and cardioaspirin (100 mg) and parenteral nutrition were started (Table 1). Two days later, the patient was re-admitted to the hospital because of worsening of his symptoms. He had uncompensated respiratory acidosis and coma. The patient was quickly intubated and a mechanical invasive ventilation was started. Three days later, his clinical conditions impaired (Body Mass Index, BMI= 15.6; Glasgow Coma Scale, GCS= 14). A ventilation with a continuous positive airway pressure (CPAP setting to 5 cm H2O) + pressure support ventilation (PSV setting to 10 cm H2O, FiO2 28%, rice time 0.3, trigger insp. 3 L/min, trigger esp. 30%, frequency of backup 10 breath/min) using a Esprit ventilator (Respironics®) was started and maintained. Blood gas analysis confirmed the respiratory acidosis with metabolic alkalosis pH 7.46 (normal range: 7.38-7.45) paCO2 48 mmHg (normal range 35-45 mmHg) paO2 100 mmHg (normal range 80-100 mmHg) HCO3 33.4mEq/L (normal range 21-30 mEq/L) base excess 9.4 (normal range –2/+2 mmol/L). In the same time, omeprazole (Mepral®20mg/day) and enoxaparin (Clexane®4000 UI/day) were started. Moreover, piperacillin+tazobactam (Tazocin®4 gr/8hi.v.), nitroglycerin (Venitrin® 20 mg in 250 cc of saline, flow 10mL/houri.v.), fructose diphosphate sodium (Esafosfina® 5 gr/dayi.v.), albumin 20% (10 gr/dayi.v.), sodium ferric gluconate (62.5 mg/day i.v.) and calcium folinate (Calciofolin® 15 mg/day i.v.) were also started. Biochemical parameters revealed the follow serum values for renal condition uric acid 29 (normal range 10-50 mg/dL), creatinine 0.4 (normal range 0.7-1.2 mg/dL), albumin 3.8 (normal range 3.4-4.8 g/dL), total protein 6.1 (normal range 6.3-8.3 g/dL). For hepatic condition: cholesterol 93 (normal range 30-200 mg/dL), triglycerides 30 (normal range30-210 mg/dL), AST14 (normal range <32IU/L), ALT 21 (normal range <31IU/L), alkaline phosphatase 95 (normal range 40 - 150 IU/L), gamma-GT 30 (normal range 5-36 IU/L), bilirubin 0.8 (normal range 0.2-1.1 mg/dL). Minerals: phosphorus 1.7 (normal range 2.7-4.5 mg/dL), iron 74 (normal range 61-157mcg/dL), potassium 3.1 (normal range 3.5 – 5.1 mmol/L), magnesium 2.07 (normal range 1.58-2.55 mg/dL). Echocardiography revealed a mitral and aortic regurgitation with pulmonary hypertension. Chest computer tomography and fiberoptic bronchoscopy revealed the presence of severe lung inflammation related to COPD but failed to document the presence of a disease able to explain the worsening of symptoms. A diagnosis of RS was postulated and few days later clinical chemistry revealed the presence of liver injury (increase in serum gamma-GT from 30 to 70 IU/L and bilirubin from 0.8 to 40mg/dL) with a decrease in hemoglobin (from 11.5 to 8.2 gr/dL) and platelets (from 23.5 to 5.8 x104/uL) see figure 1. Abdominal echography excluded the presence of liver diseases, therefore a diagnosis of parenteral nutrition associated cholestasis (PNAC) and parenteral nutrition associated liver disease (PNALD) was performed. In agreement with our previous papers (Gareri et al., 2008; Caroleo et al., 2015; Gallelli et al., 2003; Gallelli et al., 2009; Gallelli et al., 2010), using the drug probability interaction scale and the Naranjo probability scale we excluded an association between drug treatment and clinical symptoms, while we recorded a probable association between PN and clinical symptoms (score 7). PN was quickly ceased and replaced by nasogastric feeding at first and afterwards (7 days later) to a percutaneous endoscopic gastrostomy (PEG) tube, with a time-related improvement of symptoms. Around 2 months later, laboratory findings revealed a normalization of plasma values and the patient was discharged with OLTL and PEG.

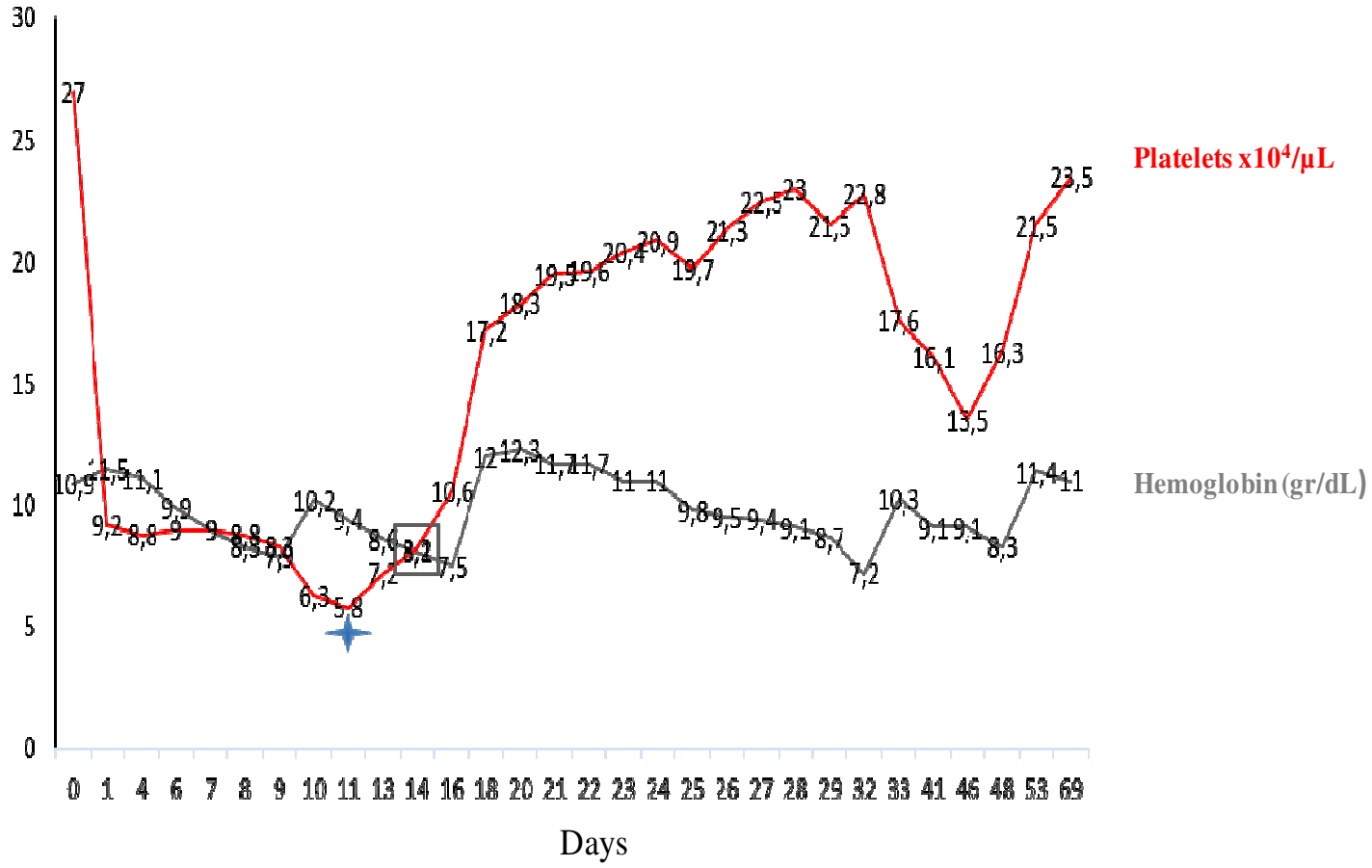

Figure 1. Hemoglobin and platelets blood levels

DISCUSSION

Although PN represents an effective approach in patients that cannot be treated with enteral nutrition (Oshima and Pichard, 2015; Mercadante et al., 2015; Baker et al., 2016) its use can be related to the development of adverse metabolic reactions to rapid refeeding. Herein we describe a patient with COPD, treated with high energy/high protein PN, without the supplementation of potassium, magnesium and phosphate that resulted in RS. In our COPD patient, we detected a poor physical condition (BMI = 15.6) that probably induced glycogenolysis in the liver and muscles and lipolysis in fat tissues. Therefore, the refeeding with high amount of carbohydrates, proteins, lipids, and electrolytes induced an increased release of insulin and a metabolic "switch" to an anabolic phase with an increased consumption by the body of phosphates, potassium and magnesium as we documented through the biochemical laboratory tests. Clinical and radiological examination excluded the presence of systemic diseases able to induce the development of symptoms. Therefore, a diagnosis of "refeeding syndrome" related to the presence of phytosterols in parenteral nutrition was firstly postulated and then confirmed using the Naranjo probability scale (score 7). In conclusion, this case could help to remind that respiratory failure in COPD may be related to refeeding. Therefore, it is necessary in real life a high quality observance of clinical guidelines and hospital protocols that remember an elderly patient is at a higher risk of malnutrition than to a younger one. We invite physicians as well as nurses to mind at artificial nutrition as a drug and suspend it quickly in presence of adverse drug reactions.

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