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Case Report

Refeeding syndrome after radical cystoprostatectomy: A case report

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ABSTRACT

Described as a potentially lethal condition that occurs in undernourished patients, refeeding syndrome (RFD) is a severe electrolyte disturbance that includes low intracellular serum concentrations of phosphor, magnesium and potassium in patients undergoing inappropriate oral or parenteral renutrition. We report a case of RFD in a 50-year-old male patient that occurs 22 days after a radical cystoprostatectomy. The patient was anorexic after the surgery, the body mass index decreased to 12,36 kg/m^2. The concentrations of albumin, magnesium, phosphor, and calcium were low. The Patient was admitted into the intensive care unit for severe cachexia and poor general condition 24 after introduction of parenteral nutrition (1500 Kcal/day). The evolution was lethal with multiple organ failure.

1. Introduction

Nutritional support remains essential for patients undergoing surgical oncology especially in intensive care. Undernutrition can be responsible for the increased patients' morbidity and mortality. Renutrition is also a sensitive time which can be deleterious to undernourished patients.

Renutrition techniques such as enteral or parenteral nutrition have specific complications. The major risk for malnourished patients during parenteral renutrition is metabolic disorders. The clinicopathological features of renutrition were grouped under the term of "refeeding syndrome" (RS), or inappropriate renutrition syndrome (SRI) [1].

The refeeding syndrome (SRI) is a complication occurring in malnourished patients during the reintroduction of enteral or parenteral energy intake. There is no consensual definition of this condition. The diagnosis is often made in an evocative context of biological disorders including plasma concentrations of phosphor, potassium and magnesium, retention of salt and water and oedema. The clinical manifestations are usually severe, sometimes fatal. There are numerous historical references suggesting the occurrence of RFD, particularly during the reintroduction of food after a long period of deprivation [2,3]. Recently, efforts have been risen in the intensive care fields to prevent and manage undernutrition especially in the early postoperative period. This fact has increased the risk of refeeding syndrome in case of inadequate or insufficiently monitored supplementation.

The aim of this case report is to provide readers with a wider understanding of RFD risk factors and to adopt a suitable protocol for correcting undernutrition to prevent this potentially critical situation.

The report has been arranged in line with SCARE guidelines [4].

2. Presentation of the case

A 50-year-old male patient was admitted to our intensive care unit, after undergoing radical cystoprostatectomy, for the management of severe malnutrition. The history of the disease dates to two years. The patient had a long history of tobacco consumption and he was followed for a recurrent bladder tumor. The last endoscopic resection showed a muscle infiltrating urothelial bladder tumor. A thoracoabdominal scan showed no metastasis. The case was discussed in a multidisciplinary concertation meeting. Then, a radical cystoprostatectomy with substitution enterocystoplasty was performed by two experienced onco-urologists via open-laparotomy approach.

No complications were detected at earlier postoperative period. The patient was transferred to the urology department four days after the operation. A parenteral nutrition (25 Kcal/kg) was administrated to the patient and stopped gradually postoperatively after five days. At Urology Department, the patient has become anorexic with dyspeptic disorders. Blood analyses revealed metabolic and ionic disorders with severe hyponatremia. At 22 postoperative days, the patient presented a severe cachexia with poor general condition. Then, he was retransferred

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to the intensive care unit for prompt support. The physical examination showed no hemodynamic, respiratory, or neurological distress. The patient had an important weight loss with body mass index of 13.26 kg/m^2. No other abnormalities were detected. Laboratory tests showed a hyponatremia 117 mmol/l, hypokalaemia 2.7 mmol/l, hypoalbuminemia 19 g/l, uraemia,16.36 mmol/l, Haemoglobin 8.4 g/dl. hypophosphatemia 0.68 mmol/l. A blood gas analysis showed not state alkalosis with a pH 7.44; Bicarbonate 13.6 mmol/l, PaCO2 = 20 mmHg. The patient management was parenteral nutrition by 1500 Kcal/day supplementation and nutritional supplement orally (500 Kcal/day). However, we have noticed a poor clinical outcome by the onset of a progressive respiratory degradation with a severe hypoxemia and tachypnea. The patient was initially put under non-invasive ventilation, then intubated and put under mechanical ventilation after worsening of respiratory symptoms and the onset of a neurological failure. Chest radiography was done showing progressively extensive interstitial syndrome. Hemodynamic and respiratory states have worsening despite the implementation of Vasoactive drugs.

The evolution was fatal with multiple organ failures leading to death 29 days after surgery.

3. Discussion

Refeeding syndrome is defined by clinicobiological disorders that occur during renutrition of undernourished patients with prolonged fasting [5–7]. Too rapid refeeding, bad conducted can lead to hypophosphatemia, hypomagnesemia, hypokalemia, hyperglycemia, decreased B1 vitamin and the collapse of multiple organic functions in particular cardiac, neurological, respiratory, renal, hepatic and muscular failure. A lethal evolution can occur in a context of multiple organ dysfunction syndrome [6,7], metabolism which was initially based on the hydrolysis of carbohydrates. In this context, lipid catabolism and protein catabolism can produce glucose by activating gluconeogenesis. The substrates of this pathway are the gluconeogenic amino acids (alanine, glutamine), glycerol resulting from the complete hydrolysis of triglycerides and lactate. At the same time, reducing energy intake is associated with a decrease in plasma insulin concentration while that glucagon increases, stimulating in turn the lipid catabolism feeding gluconeogenesis. The electrolyte depletion (phosphates, magnesium, potassium) button initially intracellular stocks, which explains the maintenance of normal plasma levels often in malnourished patients. Reintroducing glucoses input causes a shift in insulin secretion and inhibition of glucagon production and is accompanied by an abrupt metabolic transition resulting in the development of carbohydrate metabolism at the expense of fat catabolism. The sudden increase in insulin level triggers the synthesis of glycogen, fat and protein and causes the cellular uptake of potassium, phosphorus, and magnesium whose plasma levels plummet. Clinical symptoms of malnutrition syndrome are the result of electrolyte disturbances [8,9]. Hypophosphatemia is probably the most common biological abnormality, the first term attributed to this syndrome was " hypophosphatemia of refeeding ". Moreover, this term has been proposed to describe the biological consequences of the reintroduction of energy intake after a period of deprivation [10]. Biological abnormalities were associated with high mortality (18.5 versus 4.6%) [11]. Premature beats or ventricular arrhythmias can be triggered by both hypophosphatemia and hypokalaemia. A decreased concentration of ATP in myocytes is associated with muscle weakness that can induce rhabdomyolysis [12]. Involvement of respiratory muscles including the diaphragm can cause acute respiratory failure. In our case, the preoperative nutritional status was mild undernutrition. The patient did not have appropriate preoperative nutritional support. In the postoperative period, we have initially started parenteral feeding for 5 days until the gradual introduction of enteral feeding. We have noticed a remarkable weight loss. The refeeding of an assay in serum phosphorus and magnesium levels was not effective. The renutrition regimen was inappropriate. The nutritional support was higher as 35 kcal/kg per day. In addition, we did not provide microelements and vitamins supplementation. A prompt detection and correction of hypophosphatemia is crucial [13,14]. Then, renutrition should be progressively started with a nutritional support of 10 Kcal/kg per day including trace elements and vitamins supplementation (mainly vitamin B1) [15,16].

4. Conclusion

The RFD is generally underestimated despite its potentially fatal complications during refeeding. This situation is related to a various clinical presentation and heterogeneous biological disturbances. Earlier detection of patients at high risk of developing RFD is the main prevention. The prognosis is directly related to a prompt diagnosis. Subsequently, Prevention by substituting electrolytes and vitamins before initiating adequate support refeeding is crucial. In fact, we have demonstrated through this case the various failures occurring during the nutritional management of our patient. This inappropriate and excessive renutrition has led to a fatal evolution.

The key learning point is to raise consciousness about the harmful effects of inappropriate refeeding in a malnourished patient, especially respiratory and nervous ones. The possible evolution towards a multiple organ dysfunction syndrome illustrates the severity of such complication.

Ethical approval

Nothing to declare.

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Author contributions

M.W wrote the manuscript. H.A and W. Z provided references. I.B and K.M reviewed the manuscript. A.S reviewed and submitted the manuscript. Y.B and A.M contributed to the writing and the reviewing of the manuscript. All authors read and approved the final manuscript.

Consent

Written informed consent was obtained from the patient's guardian for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Declaration of competing interest

The authors declare that there are no conflicts of interest regarding the publication of this article.

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Patient consent

Written informed consent was obtained from the patient's family for publication of this case report. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Trial registry number

1. Name of the registry:
2. Unique Identifying number or registration ID:
3. Hyperlink to your specific registration (must be publicly accessible and will be checked):

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.amsu.2021.102349.

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