Review Article

Nutrition therapy for chronic obstructive pulmonary disease and related nutritional complications*

AMANDA CARLA FERNANDES[1], OLÍVIA MARIA DE PAULA ALVES BEZERRA[2]

ABSTRACT

Chronic obstructive pulmonary disease is characterized by progressive and partially reversible airway obstruction. The innumerable complications that occur during the progression of the disease can affect the nutritional state of patients suffering from this illness. The objective of this study was to present a brief review of the literature regarding the nutrition therapy used in the treatment of chronic obstructive pulmonary disease. To that end, we performed a bibliographic search for related articles published within the last 18 years and indexed for the *Literatura Latinoamericana y del Caribe en Ciencias de la Salud* (LILACS, Latin American and Caribbean Health Sciences Literature) and Medline databases. Malnutrition is associated with a poor prognosis for patients with chronic obstructive pulmonary disease, since it predisposes such patients to infections, as well as reducing respiratory muscle force, exercise tolerance and quality of life. Despite the fact that such malnutrition is extremely common in chronic obstructive pulmonary disease patients, it should be recognized as an independent risk factor, since it can be modified through appropriate and efficacious diet therapy and monitoring. For patients with chronic obstructive pulmonary disease, nutrition therapy is initiated after the evaluation of the nutritional state of the patient, which identifies nutritional risk, thereby allowing the proper level of treatment to be established. In this evaluation, anthropometric and biochemical markers, as well as indicators of dietary consumption and body composition, should be used. The prescribed diet should contain appropriate proportions of macronutrients, micronutrients and immunonutrients in order to regain or maintain the proper nutritional state and to avoid complications. The physical characteristics of the diet should be tailored to the individual needs and tolerances of each patient. In the treatment of patients with chronic obstructive pulmonary disease, individualized nutrition therapy is extremely important and has been shown to be fundamental to improving quality of life.

Keywords: Pulmonary disease, chronic obstructive; Nutrition assessment; Malnutrition; Nutrition therapy

---

* Study carried out at the Escola de Nutrição da Universidade Federal de Ouro Preto (UFOP, Federal University of Ouro Preto School of Nutrition) - Ouro Preto, Brazil.

1. Undergraduate student in Nutrition at the Universidade Federal de Ouro Preto (UFOP, Federal University of Ouro Preto) - Ouro Preto, Brazil

2. PhD in Animal Science and Epidemiology from the Universidade Federal de Minas Gerais (UFMG, Federal University of Minas Gerais). Adjunct Professor in the Clinical Nutrition Department at the Escola de Nutrição da Universidade Federal de Ouro Preto (UFOP, Federal University of Ouro Preto School of Nutrition) - Ouro Preto, Brazil

Correspondence to: Amanda Carla Fernandes. Rua das Andorinhas, 364, Bairro Laranjeiras - CEP: 32666-160, Betim, MG, Brazil. Phone: 55 31 8833-0502. Email: amandanut@gmail.com

Submitted: 05 June 2005. Accepted, after review: 06 March 2006.

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a preventable and treatable disease, caused by partially reversible, and usually progressive, airway obstruction. It is associated with an abnormal pulmonary inflammatory response to the inhalation of noxious particles or gases and is primarily caused by cigarette smoking. As well as affecting the lungs, COPD produces significant systemic consequences, such as a reduction in the body mass index (BMI) and in physical capacity.[1-2] In addition, COPD results in increased circulation of inflammatory mediators and acute-phase proteins.[3]

In general, COPD is diagnosed in patients who are in their 50s or 60s.[4] An earlier onset can occur in individuals exposed to environmental pollution (principally to sulfur dioxide) and to pollutants resulting from occupational hazards such as cadmium, silica, asbestos, and quartz.[5] It also affects those individuals with α-1 antitrypsin deficiency.[6] Deficiency of α-1 antitrypsin, a serum protease-inhibitor protein of inflammatory cells, can result in the proteolytic digestion of the pulmonary parenchyma.[7] Smoking is responsible for over 90% of all cases of COPD, which is one of the leading causes of death worldwide. Due to its progressive and incapacitating nature, COPD has a considerable economic and social impact by decreasing productivity, increasing premature death, causing family indebtedness, and provoking early retirement, as well as generating high health care costs related to treatments and hospitalizations, which are frequent.[8] In 1993, an estimated 23.9 billion dollars were spent to deal with COPD morbidity and mortality in the USA, of which 14.7 billion was directly related to the treatment of the disease, and 9.2 billion was related to indirect morbidity and premature death.[9]

The prevalence and mortality rates are higher in males than in females. However, these rates are likely to remain stable or even decrease among males, especially the younger ones, whereas there is a tendency toward an increase among females.[10] The estimated prevalence of COPD in the population of smokers in Brazil indicates that there are 7.5 million individuals with this disease. This number represents approximately 5% of the Brazilian population.[11]

According to data obtained from the Departamento de Informação and Informática do Sistema Único de Saúde (DATASUS, Unified Health Care System Department of Information and Information Science; accessed on September 2, 2003), the number of hospitalizations for COPD in Brazil between 1998 and 2003 was 1,480,881, the southern region accounting for 42.3% of this total. A study carried out in the city of Pelotas revealed that the prevalence of chronic bronchitis was 12.7% in individuals over 40 years of age.[12] The preliminary data of the Proyecto Latinoamericano de Investigación en Obstrucción Pulmonar (Latin American Project for the Investigation of Pulmonary Obstruction), carried out by the Associação Latino-Americana de Tórax na Cidade de São Paulo (Latin American Thoracic Association in the city of São Paulo), indicate that the prevalence of COPD in the population of individuals aged 40 and above ranges from 6% to 15.8%, which corresponds to 2,800,000 to 6,900,000 individuals affected by the disease.[2] The World Health Organization estimates that, by the year 2020, COPD will rank fifth among the conditions having the greatest impact on society worldwide.[11,13]

The evolution of COPD can bring innumerable complications that affect the nutritional state of the patients, such as malnutrition, which results from decreased food consumption and increased energy expenditure.[6,14]

The objective of this study was to review the literature on COPD, especially emphasizing the nutrition therapy to be introduced. To that end, we performed a bibliographic search of national and international scientific articles published within the past eighteen years and indexed for the Literatura Latinoamericana y del Caribe en Ciencias de la Salud (Latin American and Caribbean Health Sciences Literature) and MEDLINE databases.

MALNUTRITION

The marasmic type of malnutrition is a common finding among patients with COPD, and is a form of adaptation to chronic malnutrition.[15] The prevalence of malnutrition among outpatients ranges from 22% to 24%, and it varies from 34% to 50% in patients hospitalized with COPD.[2] It results in decreased respiratory performance due to the depletion of muscle proteins.[16] In addition, it increases susceptibility to pulmonary infections.

Malnutrition in COPD does not depend on a single mechanism. Various studies suggest that its etiology is multifactorial, and that the two principal

mechanisms involved in its genesis are inadequate ingestion of food and increased energy expenditure.[17]

Various factors, such as difficulties in mastication and swallowing resulting from dyspnea, cough, secretion, and fatigue, can lead to inadequate ingestion of food, and consequently to weight loss, in patients with COPD.[14] A peptic ulcer is a common finding in patients with COPD.[18]

Corticosteroids also have a quite significant negative impact on the nutritional state of these patients, due to appetite loss, bone demineralization, and weakening of the muscle mass.[19-20]

Increased energy expenditure in patients with COPD can be attributed to hypermetabolism subsequent to an increase in respiratory muscle work, which results in greater demand for oxygen. These muscles are subjected to increased demand and present decreased mechanical efficiency.[6,21] It has been shown that, due to the increased respiratory work and increased inflammatory mediators, in addition to the influence of medication, the basal metabolic rate is 15% to 17% higher in patients with COPD.[22]

The increased basal metabolic rate occurs more frequently in patients with severe COPD and can result in weight loss. Intercurrent infections and surgical procedures can lead to anorexia and to greater catabolism, resulting in the loss of muscle mass.

Leptin is a protein synthesized by the adipose tissue that plays an important role in the energy metabolism. This hormone is a signal for cerebral and peripheral tissue alterations, also regulating caloric intake, basal energy expenditure, and body weight. Recent studies suggest that the increased production of inflammatory mediators can alter the leptin metabolism in patients with COPD, thereby contributing to weight loss.[23-24]

Consequences of malnutrition

The impact of malnutrition on the respiratory system is that it decreases lung elasticity and pulmonary function, as well as reducing respiratory muscle mass, force and resistance. Malnutrition also alters pulmonary immune mechanisms and breath control.[14]

In malnourished patients, macronutrient and micronutrient deficiency generate a series of alterations that further increase the severity of COPD. Protein and iron deficiency can result in low hemoglobin levels, with subsequent decreased oxygen transport capacity. Vitamin C deficiency affects collagen synthesis, which is important in the composition of the lung support connective tissue.

At the cellular level, muscle function can be compromised due to low levels of calcium, magnesium, phosphorus, and potassium. The reduced availability of proteins and phospholipids also compromises the function of the surfactant, contributing to the alveolar collapse and consequently increasing respiratory effort.[25] It is also worth mentioning that the mucus in the airways is composed of water, glycoproteins, and electrolytes, and that hypoproteinemia also leads to the development of pulmonary edema, due to the decreased colloidal osmotic pressure, which makes it possible for body fluids to move to the interstitium.[14]

The alterations in the immune system as a consequence of malnutrition leave the patient more susceptible to pulmonary infections. Malnutrition results in the atrophy of the lymphoid tissue, principally affecting cell-mediated immunity. There is a reduction in the number of T-helper lymphocytes (due to reduced interleukin-1 activity) and of the T4/T8 ratio in the bronchoalveolar lavage fluid, together with decreased production of lymphokines and monokines.[26] With renutrition and body weight gain, there is an improvement in the absolute lymphocyte counts and in the responsiveness to antigens in skin tests.[27]

The severe protein-calorie malnutrition also results in alterations in the complement system and increased serum levels of tumor necrosis factor, which can be accompanied by anorexia, muscle degradation, and altered metabolism of lipids by the inhibition of tissue lipoprotein lipase and by the pyrogenic effect.[6,26,28]

It has also been observed that malnutrition leads to the reduction of the tidal volume and of the number of sighs, provoking alveolar collapse and inadequate secretion removal, which, in turn, predispositions the patient to pulmonary infections.[27] The respiratory pressures maximal inspiratory pressure and maximal expiratory pressure reflect the inspiratory and expiratory muscle force, respectively. Weight loss in patients with COPD can result in low maximal inspiratory pressure values due to inspiratory muscle weakness.[29]

The consequences of the morphological, functional, muscular, and pulmonary alterations

due to malnutrition in patients with COPD are as follows: decreased respiratory performance upon exertion; acute respiratory insufficiency; difficulty in weaning from mechanical ventilation, since the respiratory muscles are atrophied during prolonged mechanical ventilation, which requires only passive movements; and greater susceptibility to respiratory infections.[30] COPD significantly affects respiratory resistance and elasticity. Reduced muscle resistance with subsequent predisposition to fatigue is the clearest functional effect of malnutrition on the diaphragm and the respiratory muscles, whose resistance is compromised by the loss of the fast-twitch muscle fibers.[31]

NUTRITION THERAPY IN COPD

The treatment for COPD includes a series of procedures, from prophylactic measures to those specifically related to the correction of the alterations caused by the disease. A comprehensive treatment regimen can relieve the symptoms, reduce the number of hospitalizations, prevent premature death, and grant patients a more active and satisfactory life. Nutrition therapy is quite important in COPD due to its great impact on the morbidity and mortality caused by the disease. The data in the literature show that malnutrition is associated with a high rate of mortality in COPD patients, from 33% at the onset of the weight loss process to as high as 51% after five years.[30] A formal rehabilitation program for patients with COPD, using a team approach, presents a highly efficacious result.[32] Such a program should cover the items discussed below.

Evaluation of the nutritional state in COPD

The objective of the evaluation of the nutritional state of patients with COPD is to identify the organic and metabolic alterations that depend on nutrition or that can be mitigated by adequate dietary treatment.

Various methods can be used in the evaluation of the nutritional state, Such methods include subjective global nutritional evaluation, evaluation of dietary intake, anthropometry, determination of body composition, and biochemical testing. An isolated parameter does not characterize the general nutritional condition of an individual, and it is therefore necessary to use a combination of various nutritional state indicators to increase the diagnostic precision.

Subjective global nutritional evaluation

Nutritional triage is a method of evaluating the nutritional state of hospitalized patients andt can be performed through the protocol of the subjective global nutritional evaluation. The objective of the subjective global nutritional evaluation is to identify the patients who are at nutritional risk and to then establish what level of nutrition assistance should be used (primary, secondary, or tertiary).[33]

This method is quite useful because it makes it possible to adopt measures to prevent the worsening of the nutritional profile of the patient, as well as to avert the development of any type of malnutrition during hospitalization.

Evaluation of dietary consumption

Nutrition anamnesis is an evaluation method that makes it possible to predict or estimate the nutritional state of the patient through a qualitative and quantitative analysis of dietary consumption. It consists in obtaining information on dietary consumption and individual eating habits in order to enable a diagnosis of the past and present status of the patient, as well as to determine dietary habits, practices, intolerance, acceptance, and taboos, thereby allowing the physician/clinician to provide guidelines for the most appropriate diet prescription for each case.[34-35]

The 24-hour recollection method and semi-quantitative survey of the frequency of food consumption can be used in the evaluation of the dietary consumption of the patient with COPD. The 24-hour recollection method provides detailed information on current consumption, as well as on the number and scheduling of the meals on the day prior to the day of the interview. The semi-quantitative survey of the frequency of dietary consumption provides a list of different foods, as well as the frequency of consumption of each one of them.

In one study, two groups were evaluated: one consisting of pulmonary patients; and the other consisting of healthy individuals. The authors applied the 24-hour recollection method and observed that both groups typically presented a diet based on carbohydrates. Therefore, these individuals, especially the lung disease patients, required some guidance as to the importance of consuming only

a moderate amount of foods rich in carbohydrates, giving preference to foods rich in fibers.(36)

Anthropometry

Anthropometry is widely used in the evaluation of the nutritional state due to its easy application, low cost, and noninvasive nature.[6] The anthropometric measures most often used are weight, height, skinfold thickness, and circumferences.

Due to its practicality, the BMI - body weight (kg)/height $(m)^2$ - has been used as a good indicator of the nutritional state. However, this index does not portray individual differences in body composition.

According to the literature, the degree of severity of pulmonary diseases is associated with BMI,[37] a low BMI being related to high mortality risk in patients with severe COPD.

In a study analyzing the factors that influence the quality of life of patients with COPD, it was observed that BMI, independently, has a significant influence on the quality of life of these patients.[38]

The Nutrition Screening Initiative, the American Academy of Family Physicians, and the American Dietetic Association have suggested the following BMI values as cut-off points for patients with COPD: 22-27 kg/m^2 for normal weight; < 22 kg/m^2 for malnutrition; and > 27 kg/m^2 for obesity.[19-20]

In 2004, an index comprising four fundamental aspects of the disease was created. This index was designated the Body mass index, airway Obstruction, Dyspnea, and Exercise capacity (BODE) index. The BODE index is a COPD predictor of mortality, since it combines the various factors that can be indicators of mortality in these patients.[37] The BMI cutoff point used in the BODE index is 21 kg/m^2, since values lower than this have been associated with an increased risk of death.[37,39] Determining body fat reserves in patients with COPD is extremely important, since, without these reserves, the organism begins to mobilize its own protein reserve as an energy source.[6] Measurement of skinfold thickness constitutes a quite convenient method of estimating the body fat reserve.

In patients suffering from chronic diseases, measurement of skinfold thickness can be useful in the evaluation of the long-term changes that occur in the subcutaneous adipose tissue reserves. Through the summation of the triceps, biceps, subscapular and supra-iliac skinfold thicknesses, it is possible to calculate the body composition of an individual.

Half of the body fat is located in the subcutaneous tissue and, with advancing age, the internal fat deposition also increases. Therefore, measurement of skinfold thicknesses is not ideal for the evaluation of elderly patients. However, the measurement of the circumference of the arm satisfactorily reflects the body protein reserve.[21] This information deserves special attention during the evaluation of the nutritional state of elderly patients with COPD.

Muscle mass depletion is the principal factor responsible for the negative effects attributed to malnutrition.[40] The muscle protein reserves are mobilized to meet the demand of the protein synthesis in the patients with chronic diseases, and can result in muscle depletion, which represents a serious problem for patients with COPD.

The arm circumference is an anthropometric parameter of nutritional evaluation and is quite often used to estimate the total skeletal muscle protein. Through measurement of arm circumference and triceps skinfold thickness, it is possible to calculate the muscle circumference and the arm muscle area, low values of which are good indicators of severe depletion of muscle mass and fat reserve.[17]

Bio-impedance

The bio-electrical impedance (bio-impedance) technique is employed to measure the conductive properties of an individual and thereby define the body composition and type, as well as to determine the volume and distribution of fluids and tissues.[41]

The estimation of body composition through the use of bio-impedance has frequently been used, because it is easily applied and is a noninvasive method. Patients with emphysema ('pink puffers') typically present lower percentages of body fat and lower BMIs than do patients with chronic bronchitis ('blue bloaters') and normal individuals.[42-43]

Bio-impedance is a highly precise method of evaluating of the body composition of patients with chronic diseases. However, it presents low sensitivity in predicting alterations in body composition over a short period of time.[44] The literature also shows that, in the evaluation of the body composition of elderly patients with COPD, bio-impedance is preferable to the measurement of skinfold thickness.[6]

Biochemical testing

Biochemical testing is useful in the evaluation of the nutritional state of patients with COPD and

contributes to the monitoring of the nutrition therapy as well.

The creatinine/height index is used in the evaluation of the lean BMI in malnourished patients with COPD.[30]

Creatinine is a metabolite derived from the nonenzymatic and irreversible hydrolysis of creatine and phosphocreatine Creatinine, little of which is absorbed by the organism, is excreted by the kidneys.[45] The size of the muscle protein pool of the patient is directly proportional to the quantity of creatinine excreted, and the expected 24-h excretion is related to the height of the patient.[46] Therefore, the creatinine/height index can be considered an extremely important muscle mass index in the evaluation of the nutritional state of patients with COPD who present muscle mass depletion.

Plasma proteins are transport proteins synthesized by the liver and are known indicators of visceral protein status. Such proteins include albumin, pre-albumin, transferrin, and the retinol transport protein. Determining serum protein levels, used in conjunction with other methods of evaluating the nutritional state, is quite useful in the evaluation of patients with COPD, contributing, principally, to monitoring the nutrition therapy.

The evaluation of immunocompetence is also quite important, due to the evident correlation between nutritional state and immunity. The evaluation of the immune response through the total lymphocyte counts and the hypersensitivity skin test allows the identification of alterations in nutritional state, since there is depletion of humoral and cellular immunity in malnourished patients.[33]

Nutritional practices in COPD

Nutrition that is appropriate in quantitative and qualitative terms is of fundamental importance in the treatment of COPD. The principal objectives of nutrition therapy in COPD are summarized in Chart 1.

According to the results of the evaluation of the nutritional state, patients with COPD can be divided into two groups. The first group comprises the patients who presented high risk of nutritional complications due to the exacerbation of the disease. In this case, the objective of the dietary treatment is to prevent protein-calorie malnutrition, as well as its consequences, by providing adequate nutrition. The second group comprises those patients who presented malnutrition, with or without respiratory insufficiency. For this group, the objective of the dietary treatment is to reverse the malnutrition profile through nutrition in order to ensure the reposition of all the deficient macronutrients and micronutrients.[30]

The reversal of malnutrition in patients with COPD results in improvement of the immune response of the neutrophils and of the complement, thereby strengthening the defense of the organism against infections. Improvement in respiratory muscle function, reversal of the alterations of the ventilatory response, and normalization of the surface forces have also been observed subsequent to the normalization of the rate of phosphatidylcholine synthesis in the pulmonary tissue and in the bronchoalveolar lavage fluid.[30]

Although malnutrition is extremely common in patients with COPD, being an indicator of worse prognosis, it is important to recognize it as an independent risk factor, since it can be potentially modified through appropriate and efficacious dietary treatment.

Calculation of energy requirements

The calculation of the energy requirements of a hospitalized patient with COPD is based on formulas that estimate their energy expenditure. The ideal would be to calculate the energy requirements of the patient through the use of

Chart 1- Objectives of nutrition therapy in COPD

- Providing nutrition that promotes maintenance of respiratory muscle force, mass, and function in order to optimize the global performance status of the patient and satisfy the demands of daily activities.
- Maintaining an adequate reserve of lean body mass and adipose tissue, since patients with COPD present alterations in body composition, manifested by weight loss, and, principally, muscle mass loss.
- Correcting the water imbalance that is common in patients with COPD.
- Controlling the interaction between drugs and nutrients that negatively interfere both in the consumption of food and in the absorption of nutrients.
- Promoting an improvement in the quality of life of the patient.

direct or indirect calorimetry. However, since these tests are not available in most hospitals, it is necessary to use formulas that estimate this information.[30]

Energy requirements can be calculated using the equation devised by Harris-Benedict in 1919. According to Harris-Benedict, the calculation of the basal metabolic rate is based on patient gender, weight (kg), height (cm), and age (years). The calculation of the total energy expenditure is performed by determining the basal metabolic rate, according to Harris-Benedict, and multiplying it by the physical activity factor and the injury factor.[47]

Having determined the caloric needs of the patient with COPD, we proceed to determining the distribution of the nutrients in the diet, so that ingestion is matched to demand.

Distribution of nutrients in the diet

The availability of calories in the form of protein should correspond to 20% of the total energy expenditure of the patient, and the remaining 80% should be distributed in the form of carbohydrates and lipids. Adequate availability of nonprotein calories prevents the ingested protein from being consumed by the organism as a source of energy.[30]

For patients with COPD, a high-protein diet is recommended in order to restore respiratory muscle force and promote improvement in the immune function, and the protein value should be from 1 to 1.5 g/kg of body weight/day. However, an excess of proteins in the diet should be avoided. A diet with overly high protein content can result in increased dyspnea in patients with increased respiratory impulse, as well as in those with marginal respiratory reserve. In addition, diets containing proteins with high concentrations of ramiform amino acids (valine, leucine, and isoleucine) should also be avoided, since they can stimulate the respiratory center, resulting in increased respiratory effort and inducing muscle fatigue.[30]

The recommended quantity of carbohydrates for patients with COPD ranges between 50% and 60% of the total energy expenditure of the patient. An amount of carbohydrates exceeding necessity leads to lipogenesis, resulting in the production of an excess of carbon dioxide, which can also lead to liver steatosis. There is an increase of the respiratory quotient during lipogenesis. Therefore, more carbon dioxide will be produced and, consequently, respiratory frequency will have to be increased to make it possible for the lung to eliminate this excess of carbon dioxide, which can result in respiratory insufficiency.[6,30]

Overnutrition can also result in lipogenesis, causing increased production of carbon dioxide and consequently difficulty in eliminating it. Studies suggest that the total amount of calories provided by the diet influences the production of carbon dioxide more clearly than does the quantity of carbohydrates available.[47]

For some authors,[6] the recommended quantity of lipids for patients with COPD ranges from 25% to 30% of the estimated total energy expenditure. Nevertheless, others[30] recommend that, in the event of respiratory insufficiency, the quantity of lipids used range from 30% to 50% of the patient requirements.

The excessive administration of lipids can be associated with decreased diffusing lung capacity, in addition to hypertriglyceridemia, principally in the presence of hypercatabolism and septicemia. The reduction of carnitine can be responsible for the dysfunction in the lipid metabolism, resulting in increased triglycerides.[30] Studies show that the combination of L-carnitine supplementation and physical training can result in greater exercise tolerance in patients with COPD.[48] Therefore, excessive lipid levels should be avoided.

Smoking is the most significant factor leading to the development of COPD. In emphysema, there is an excess of proteases that cause the destruction of elastin and of the collagen matrix, which sustain the lung architecture. Smoking results in an influx of neutrophils into the lung and in the release of elastase and other proteases. In addition, the oxidants released by the activated inflammatory cells, together with those that are released in cigarette smoke, hinder the action of endogenous antiproteases. Antioxidants typically found in the lower respiratory tract (superoxide dismutase, catalase, and glutathione), together with ceruloplasmin, copper, methionine sulfoxide, retinol, vitamin E, and vitamin C, protect the organism against the effect of these oxidants.[27]

Therefore, it is believed that dietary antioxidants such as vitamin C and retinol[49] can limit the destruction of the lung tissue by proteases and protect the organism against the development of COPD.

Electrolyte deficiencies, such as hypokalemia, hypocalcemia, hypomagnesemia, and

hypophosphatemia, can affect respiratory function in patients with COPD. Therefore, restoring the normal intracellular electrolyte concentrations can improve muscle force. Phosphate deficiency results in the decrease of 2,3-diphosphoglycerate in erythrocytes, resulting in lower oxygen hemoglobin and, consequently, decreased oxygen availability to the tissues, which leads to iatrogenic hypophosphatemic respiratory insufficiency. Therefore, the adequate reposition of inorganic phosphate is necessary in patients with COPD.[22]

Osteoporosis is a common finding in patients with advanced COPD, and it results in increased susceptibility to fractures, which is a debilitating condition for these patients. The use of glucocorticosteroids, vitamin D deficiency, smoking, low BMI, and a sedentary lifestyle are involved in the etiology of osteoporosis. Calcium and vitamin D supplementation in these patients has proven quite efficient, principally when they are under treatment with corticosteroids.[50]

Dietary adaptations in COPD

The consistency of the diet for patients with COPD should be adapted to the physiological conditions of each patient. It should be specifically noted whether the patient presents dental problems, which can affect proper chewing of food, or dyspnea, which impairs the ability to eat. In these cases, it is necessary to implement a mild or soft-food diet.

The diet must be well distributed at intervals, in order to offer the patient between five and six meals, of lesser volume, per day. In patients with COPD, large meals can cause fatigue and anorexia, thereby limiting the consumption of food.

In the vast majority of cases, individuals affected by COPD are elderly. One of the physiological modifications that occur with aging is the decrease of the thickness of the tissue of the mouth and tongue mucosa, whose appearance becomes smoother and thinner. Therefore, there is an increase in the thermal sensitivity in the oral mucosa, which makes these patients more intolerant to foods of extreme temperatures. It is recommended that these patients be served mild temperature food, in order to avoid damages to the oral mucosa.

Xerostomia is a quite common symptom in the elderly, and can also occur as a consequence of the use of some medications (such as antidepressants, antihypertensives, and bronchodilators),[51] resulting in difficulty in chewing, swallowing, and digesting food. Therefore, its presence should also be considered when choosing a more appropriate consistency of food.

Some authors[52] have established guidelines for patients with COPD in order to avoid the reduction in dietary consumption These are synthesized in the adaptation shown in Chart 2.

Nutrition support

In the cases in which conventional nutrition alone cannot supply all of the nutritional requirements of a patient, it is necessary to implement nutrition support, which can be provided through oral, enteral, or parenteral nutrition therapy.

When the gastrointestinal tract can be used without limitations or risks to the patient, preference should be given to the use of oral nutritional supplements, since it is the most physiological path of all.[47] However, when this method is not efficacious, or when the patient presents dysphagia or even refuses oral food ingestion, enteral nutrition therapy should be chosen.[53]

However, when it is impossible to use the gastrointestinal tract, or when enteral nutrition is counter indicated, parenteral nutrition therapy should be implemented. Parenteral nutrition consists of the intravenous infusion of a sterile solution of nutrients, prepared according to the characteristics of each patient, though a venous access, peripheral or central, in order to completely exclude the digestive tract from the process.[54]

The inclusion of immunonutrients in nutrition therapy is necessary due to the hypermetabolic state and pre-existing malnutrition, which produce immunosuppression in the patient with COPD. The use of these immunonutrients is aimed at increasing the production of less potent inflammatory mediators and reducing those that are highly inflammatory, in addition to minimizing the production of free radicals and the modulation of the generalized inflammatory response. Therefore, the patient should be offered a diet enriched with the following: lipids, such as short-chain and Omega-3 fatty acids, as well as gamma linolenic and eicosapentaenoic acids; amino acids, such as glutamine, glycine, cysteine, and arginine; and nucleotides/oligo-elements such as copper, zinc, and selenium.[22,55]

Chart 2 - Nutrition therapy for patients with chronic obstructive pulmonary disease

Symptom Presented	Nutrition Therapy
Anorexia	The diet should be well distributed throughout the day, prioritizing the offer of food that the patient prefers. The food with greater energy density should be ingested first. In order to increase the energy density of the food, butter, margarine, mayonnaise, and cream might be added to the preparation.
Early satiation	The more energetic food should be initially ingested, with preference to colder food, and limiting ingestion of liquids during meals (the ingestion of liquids should occur one hour after the meals).
Dyspnea	Patients should avoid using bronchodilators near mealtimes and should, if indicated, use strategies of secretion release before the meals. Patients should also eat slowly and have food prepared for periods of increased dyspnea.
Flatulence	The ingestion of flatulence-inducing foods (kale, cabbage, broccoli, onion, beans, etc.) should be avoided, as should that of carbonated beverages (soft drinks and sparkling water). The patient should eat slowly and ingest smaller quantities of food more frequently.
Fatigue	It is advisable to rest before meals and choose easy-to-prepare food. In addition, there should always be food already prepared for the periods of increased fatigue.
Intestinal Constipation	The ingestion of more easily chewable fibers should be increased, as well as the ingestion of liquids, in order to improve gastrointestinal motility. The patient should do physical exercises, if tolerated.
Dental Problems	An alteration should be made in the consistency of the diet in order to facilitate chewing and swallowing, and the patient should be referred for dental treatment.

Hydration

The volumes used in the fluid replacement should be calculated individually for each patient, respecting important factors such as age and possible accompanying clinical conditions. Patients with COPD need to be properly hydrated, since they produce a great quantity of mucus. Adequate hydration loosens secretions, since water reduces viscosity, facilitating their expulsion.[19]

FINAL CONSIDERATIONS

The prevalence of COPD has grown rapidly over the last few decades, with considerable economic and social impact. Various studies have demonstrated the importance of nutrition therapy to improving the clinical profiles of these patients.

The treatment for malnutrition and other nutritional complications associated with COPD is fundamental to the evolution of the clinical profile, as well as to the improvement of the quality of life of the patient. Individualized nutrition therapy is quite important and should be instituted as early as possible in order to improve patient nutritional state, immune function, respiratory muscle function, and exercise tolerance.

REFERENCES

1. Celli BR, MacNee W; ATS/ERS Task Force. Standards for the diagnosis and treatment of patients with COPD: a summary of the ATS/ERS position paper. Eur Respir J. 2004;23(6):932-46. Erratum in: Eur Respir J. 2006;27(1):242. Erratum in: Eur Respir J. 2006;27(1):242.
2. Sociedade Brasileira de Pneumologia e Tisiologia. II Consenso Brasileiro de Tuberculose: diretrizes brasileiras para tuberculose 2004. J Pneumol. 2004;30(Supl 1):S1-S42.
3. Wouters EF, Creutzeberg EC, Schols AM. Systemic effects in COPD. Chest 2002;121(5 Suppl):127S-130S.
4. Menezes AMB. Epidemiologia da bronquite crônica e do enfisema (DPOC): até onde sabemos? J Pneumol 1997;23(3):153-7.
5. Mannino DM, Homa DM, Akinbami LJ, Ford ES, Redd SC. Chronic obstructive pulmonary disease surveillance--United States, 1971-2000. MMWR Surveill Summ. 2002;51(6):1-16.
6. Sachs A, Lerario MC. Doenças pulmonares. In: Cuppari L. Guia de nutrição clínica no adulto. São Paulo: Manole; 2002. p. 249-62.
7. Sandford AJ, Weir TD, Pare PD. Genetic risk factors for chronic obstructive pulmonary disease. Eur Respir J. 1997;10 (6):1380-91. Comment in: Eur Respir J. 1998;12(4):998-9.
8. Campos HS. O preço da DPOC. Pulmão RJ. 2003;12(4):5-7.

9. Sullivan SD, Ramsey SD, Lee TA. The economic burden of COPD. Chest. 2000;117(2 Suppl):5S-9S.
10. Campos H. Doença pulmonar obstrutiva crônica. J Bras Med. 1993;64(6):98-114.
11. Pelagrino NRG, Godoy I. Doença pulmonar obstrutiva crônica. Rev Bras Med. 2001;58(1):81-8.
12. Menezes AMB, Victora CG, Rigatto M. Prevalence and risk factors for chronic bronchitis in Pelotas, RS, Brasil: a population-based study. Thorax. 1994;49(12):1217-21.
13. Murray CJ, Lopez AD. Evidence-based health policy lessons from the Global Burden of Disease Study. Science. 1996;274(5288):740-3. Comment in: Science. 1996;274 (5293):1593-4.
14. Mueller D. Terapia clínica nutricional na doença pulmonar. In: Mahan L K, Stump S E. Krause - Alimentos, nutrição e dietoterapia. 10a ed. São Paulo: Roca; 2002. p. 789-805.
15. Waitzberg DL, Gama-Rodrigues J, Correia MITD. Desnutrição hospitalar no Brasil. In: Waitzberg DL. Nutrição oral, enteral e parenteral na prática clínica. 3a ed. São Paulo: Atheneu; 2000. p.385-97.
16. Ferreira IM, Brooks D, Laçasse Y, Goldstein R S. Nutritional support for individuals with COPD: a meta-analysis. Chest. 2000;117(3):672-8.
17. Hugli O, Fitting JW. Alterations in metabolism and body composition in chronic respiratory diseases. In: Wouters EFM. Nutrition and metabolism in chronic respiratory diseases. London: Maney; 2003. p. 11-22; [European Respiratory Monography, 24].
18. Brashear RE, Rhodes ML. Doença pulmonar obstrutiva crônica. Rio de Janeiro: Interamericana; 1981. p.189-98.
19. American Academy of Family Physicians; American Dietetic Association. A physician's guide to nutrition in chronic disease management for older adults [text on the Internet]. Washington, DC: Nutrition Screening Initiative; 2002; [cited 2004 Mar 02]. Available from: http://www.aafp.org/PreBuilt/NSI_CME.pdf
20. Harmon-Weiss S. Chronic obstructive pulmonary disease: nutrition management for older adults [text on the Internet]. Washington, DC: Nutrition Screening Initiative; 2002. [cited 2004 Mar 02]. Available from: www.aafp.org/PreBuilt/NSI_COPD.pdf.
21. Pereira CAC. Nutrição em doença pulmonar obstrutiva crônica. J Pneumol. 1988;14(1): 45-54.
22. Vasconcelos FC, Mota ES, Lopes MFL, Fernández SSL, Medeiros ZL. Terapia nutricional na doença pulmonar obstrutiva crônica associada à desnutrição protéico-calórica: artigo de revisão. Rev Para Med. 2002;16(1):47-52.
23. Takabatake N, Nakamura H, Abe S, Hino T, Saito H, Yuki H, et al. Circulating leptin in patients with chronic obstructive pulmonary disease. Am J Respir Crit Care Med. 1999;159(4 Pt 1):1215-9.
24. Creutzberg E. Leptin in relation to systemic inflammation and regulation of the energy metabolism in chronic obstructive pulmonary disease. In: Wouters EFM. Nutrition and metabolism in chronic respiratory diseases. London: Maney; 2003. p.56-67. [European Respiratory Monography, 24].
25. Jardim JRB, Cendon SP. Reabilitação pulmonar. In: Tarantino AB. Doenças pulmonares. 4a ed. Rio de Janeiro: Guanabara Koogan; 1997. p.627-40.
26. Moreira JC, Waitzberg DL. Conseqüências funcionais da desnutrição. In: Waitzberg DL. Nutrição oral, enteral e parenteral na prática clínica. 3a ed. São Paulo: Atheneu; 2000. p. 399-409.
27. Johnson MM, Chin R, Haponik EF. Nutrição, função respiratória e doença. In: Shils ME, Olson JA, Shike M, Ross AC. Tratado de nutrição moderna na saúde e na doença. 9a ed. São Paulo: Manole; 2002. p.1579-96.
28. Debigare R, Cote CH, Maltais F. Peripheral muscle wasting in chronic obstructive pulmonary disease. Clinical relevance and mechanisms. Am J Respir Crit Care Med. 2001;164(9):1712-7.
29. Barros SEB. Correlação das pressões respiratórias máximas com o estado nutricional de doentes pulmonares. Rev Bras Nutr Clin. 2003;18(3):123-9.
30. Vianna R, Maia F, Waitzberg DL. Insuficiência respiratória. In: Waitzberg DL. Nutrição oral, enteral e parenteral na prática clínica. 3a ed. São Paulo: Atheneu; 2000. p.1199-208.
31. Rosa EA. Dispnéia e achados funcionais em portadores de DPOC nutridos e desnutridos. J Pneumol. 1992;18(3):105-10.
32. Matthay RA, Arroliga AC. Patologias crônicas das vias aéreas. In: Bennett JC; Plum F. Cecil - Tratado de medicina interna. 20a ed. Rio de Janeiro: Guanabara Koogan; 1997. p.421-9.
33. Kamimura MA, Baxmann A, Sampaio LR, Cuppari L. Avaliação nutricional. In: Cuppari L. Guia de nutrição clínica no adulto. São Paulo: Manole; 2002. p.71-109.
34. Vasconcelos FAG. Avaliação nutricional de coletividades. 2a ed. Florianópolis: UFSC; 1995. p.115-26.
35. Hammond K. Avaliação dietética e clínica. In: Mahan LK, Stump SE. Krause - Alimentos, nutrição e dietoterapia. 10a ed. São Paulo: Roca; 2002. p. 341-66.
36. Barros SEB. Correlação das pressões respiratórias máximas com o estado nutricional de doentes pulmonares. Rev Bras Nutr Clin. 2003;18(3):123-9.
37. Celli BR, Cote CG, Marin JM, Casanova C, Montes de Oca M, Mendez RA, et al. The body-mass index, airflow obstruction, dyspnea, and exercise capacity index in chronic obstructive pulmonary disease. N Engl J Med. 2004;350(10):1005-12. Comment in: ACP J Club. 2004;141(2):53. N Engl J Med. 2004;350(10):965-6. N Engl J Med. 2004;350(22):2308-10; author reply 2308-10. N Engl J Med. 2004;350(22):2308-10; author reply 2308-10. N Engl J Med. 2004;350(22):2308-10; author reply 2308-10. N Engl J Med. 2004;350(22):2308-10; author reply 2308-10.
38. Dourado VZ, Antunes LCO, Carvalho LR, Godoy I. Influência de características gerais na qualidade de vida de pacientes com doença pulmonar obstrutiva crônica. J Pneumol. 2004;30(3):207-14.
39. Landbo C, Prescott E, Lange P, Vestbo J, Almdal TP. Prognostic value of nutritional status in chronic obstructive pulmonary disease. Am J Respir Crit Care Med. 1999;160(6):1856-61.
40. Soler-Cataluna JJ, Sanchez-Sanchez L, Martinez-Garcia MA, Sanchez PR, Salcedo E, Navarro M. Mid-arm muscle area is a better predictor of mortality than body mass index in COPD. Chest. 2005;128(4):2108-15.
41. Mendes CCT, Raele R. Avaliação corporal por bioimpedância. Rev Nutr Pauta. 1997;24:12-4.
42. Coppini LZ, Waitzberg DL. Impedância bioelétrica. In: Waitzberg DL. Nutrição oral, enteral e parenteral na prática clínica. 3a ed. São Paulo: Atheneu, 2000. p. 295-304.

43. Wouters EF. Nutrition and metabolism in COPD. Chest. 2000 May;117(5 Suppl 1):274S-80S.
44. Ferreira, IM. Chronic obstructive pulmonary disease and malnutrition: why are we not winning this battle? J Pneumol. 2003;29(2):107-15.
45. Bottoni A, Oliveira GPC, Ferrini MT, Waitzberg DL. Avaliação nutricional: exames laboratoriais. In: Waitzberg DL. Nutrição oral, enteral e parenteral na prática clínica. 3a ed. São Paulo: Atheneu; 2000. p. 279-94.
46. Carlson T. Dados laboratoriais na avaliação nutricional. In: Mahan LK; Stump SE. Krause - Alimentos, nutrição e dietoterapia. 10a ed. São Paulo: Roca; 2002. p. 367-85.
47. Pingleton SK. Enteral nutrition in patients with respiratory disease. Eur Respir J. 1996;9(2):364-70.
48. Silva AB, Di Lorenzo VAP, Jamami M, Sampaio MM, Demonte A, Cardello L, et al. Efeitos da suplementação oral de L-carnitina associado ao treinamento físico na tolerância ao exercício de pacientes com doença pulmonar obstrutiva crônica. J Pneumol. 2003;29(6):379-85.
49. McGowan, SE. Contributions of retinoids to the generation and repair of the pulmonary alveolus. Chest 2002;121(5 Suppl): 206S-208S.
50. Biskobing MD, Diane M. COPD and osteoporosis. Chest. 2002;121(2): 609-20.
51. Di Maria-Ghalili, RA, Amella E. Nutrition in older adults: intervention and assessment can help curb the growing threat of malnutrition. Am J Nurs. 2005;105(3):40-50; quiz 50-1. Comment in: Am J Nurs. 2005;105(8):16; author reply 16.
52. Rogers MR, Donahoe M. Nutrition in pulmonary rehabilitation. In: Fishman AP, editor. Pulmonary rehabilitation. New York: Marcel Dekker; 1996. p. 543-64.
53. Vasconcelos MIL. Nutrição enteral. In: Cuppari L. Guia de nutrição clínica no adulto. São Paulo: Manole; 2002. p.369-90.
54. Monte JCM. Nutrição parenteral. In: Cuppari L. Guia de nutrição clínica no adulto. São Paulo: Manole; 2002. p.391-7.
55. Gadek JE, DeMichele SJ, Karlstad MD, Pacht ER, Donahoe M, Albertson TE, et al. Effect of enteral feeding with eicosapentaenoic acid, gamma-linolenic acid, and antioxidants in patients with acute respiratory distress syndrome. Enteral Nutrition in ARDS Study Group. Crit Care Med. 1999;27(8):1409-20. Comment in: Crit Care Med. 1999;27(8):1646-8.