

Overview of human nutrition for the general physician

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Abstract

Human nutrition is a poorly understood aspect of medical care. The nutritional state of patients is an essential component of overall treatment yet doctors have a poor understanding of this field of medicine. This review will highlight aspects of identifying malnutrition by nutritional screening and nutritional assessment. Generalised nutritional support and treatment options available in specific disease processes will also be discussed.

Background

Malnutrition is defined as state of nutrition in which there is a deficiency or excess of energy, protein and other nutrients causing measurable adverse effects on tissue/body form, function and clinical outcome. (1) It is recognised that 30% of in-hospital patients are malnourished (undernourished) on admission and the majority of these will lose further weight while in hospital. (2) In this review article the term undernutrition will be used instead of the generalised term malnutrition. Undernutrition develops due to increased losses (vomiting, diarrhoea, malabsorption), decreased intake (anorexia, vomiting, nausea, dysphagia), increased requirements (catabolic state) or a combination of all these processes.

Is undernutrition important?

Consequences of undernutrition include reduced muscle mass, impaired immune function, poor tissue viability, poor clinical outcome and psychosocial effects. (3) Reduced muscle mass decreases cardio-pulmonary function, lean muscle mass and muscle weakness. Impaired immune function increases infection and sepsis risk. Poor tissue viability can cause pressure sores and poor wound healing. Undernutrition can amplify the length of hospital stay. Psychosocial effects include altered mood and poor quality of life. (4) It is therefore essential that undernutrition is properly treated to diminish patient morbidity and mortality.

How to recognise undernutrition:

1. Malnutrition screening tools

Screening tools should be performed easily with low-level staff training. Hospital patients at risk of undernutrition are identified by screening methods such as MUST (Malnutrition Universal Screening Tool) (5), SGA (Subjective Global Assessment) (6) or MNA (Mini Nutritional Assessment – validated for > 65 years) (7). These screening methods usually consider current body mass index (BMI), recent weight loss and

possible future weight loss. MUST is currently well advertised as a nutritional screening tool within UK hospitals. The British Association of Parenteral and Enteral Nutrition have endorsed this 5 step-screening tool. The MUST score (low, moderate, high risk) has been shown to correlate with mortality (low risk group 8% vs. high risk group 32%, $p < 0.01$) and length of hospital stay (low risk group 15 days vs. high risk group 28 days, $p < 0.02$) (8). See Table 1.

Table 1: MUST Screening tool (5)

Step 1: BMI (can use alternatives such as ulna length for height or mid-upper arm circumference (MUAC) as approximation for BMI: MUAC <23 cm = BMI 20kg/m ² , MUAC >32cm = BMI >30 kg/m ²)
Step 2: Percentage weight loss
Step 3: Establish Acute Disease Effect and score
Step 4: Add scores step 1,2,3 together to obtain overall risk of malnutrition
Step 5: Develop care plan

2. Assessment of nutritional status:

If a patient is found to be at risk of undernutrition, following screening, then a formal nutritional assessment ensues. This assessment involves anthropometrics, biochemical testing, clinical methods and dietary history.

- Anthropometrical data: appropriately trained staff can perform weight, height, waist circumference, mid-upper arm circumference and skinfold thickness measurements. Indices can subsequently be calculated. These include percentage weight loss, BMI and waist-hip ratio.
- Biochemical data: information acquired from blood testing includes renal function as a marker of hydration. Also sepsis markers including CRP, ESR and WBC's are valuable surrogate markers for stress response. Albumin is a poor marker of nutritional status. (9)
- Clinical: medical history including past and present is valuable. It is essential to obtain plans regarding fasting for

investigations. Knowledge of current treatment that may cause decreased intake or increased losses is essential.

d) Dietary History: there are assorted techniques of obtaining dietary history. Mostly recall, record diary and food frequency questionnaires methods are utilised.

The overall nutritional assessment entails considering all the information obtained from these different methods. Subsequently a clinical decision is reached regarding the overall nutritional status.

How to assess nutritional requirements:

In-patient energy requirements are calculated using a combination of:

- a) basal metabolic rate equations such as Schofield, Harris Benedict and Ireton Jones
- b) stress factors or weight gain/loss
- c) combined factor for activity level and diet induced thermogenesis.

The basal metabolic rate is typically calculated using the Schofield equation (10). Schofield estimates basal metabolic rate of a healthy individual. An adjustment is then made for stress or weight gain/loss. Stress factors have been published for various clinical conditions including brain injury, infection, pancreatitis and surgery. Finally a combined factor (activity and diet-induced thermogenesis) is added to calculate total energy requirements. This combined factor is adjusted depending on patient mobility. Community patient's energy requirements are calculated using a separate method. Occupational and non-occupational activity is estimated to determine a physical activity level which is multiplied by basal metabolic rate to achieve overall energy requirements.

Nitrogen/protein requirements are estimated using current patient clinical state i.e. hypermetabolic, depleted or normal state. Normal state nitrogen requirements are 0.14-0.20 g/kg/day. Depleted state patients nitrogen requirements are 0.20-0.40 g/kg/day. (1g nitrogen = 6.25g protein)

Methods of treating undernutrition

Following identification of undernutrition a patient's treatment can be instituted. Undernutrition is typically treated using a graded stepwise approach and subsequently monitoring response. If however it appears clinically obvious that the first steps would not be advantageous then treatment can be commenced at a more aggressive phase. Improving energy intake using the following methods can treat undernutrition:

Step 1. Increase frequency and quantity of food intake. Consider nutrient dense foods. Encourage foods that are energy and nutrient dense such as meat, fish, cheese, eggs, dairy produce and snack foods.

Step 2. Increase nourishing drinks including milk based drinks, soups, fruit juices and sugary drinks. Nourishing drinks are

simple to make and can provide high calories in a small quantity.

Step 3. Food fortification. This essentially increases the energy density of foods by adding high-energy components such as addition of cheese, milk powder, cream, jam and butter to other foods.

Step 4. Supplements. These can be milk, yoghurt or juice based. They contain varying calories (1-2kcal/ml) and protein (4-6g protein/100ml). Supplements are very useful at boosting energy intake. Most are nutritionally complete but others contain calories only. Examples include Ensure Plus, Fortisip, Calogen and Calshake.

Step 5. Enteral feeding. Enteral feeding is used to provide either supplementary or complete nutrition to patients that are unable to maintain adequate nutrition by oral route. It is only likely to benefit malnourished patients or those at risk of malnutrition. This includes patients that have had a failed trial of diet modification or supplementary feeds or patients at pulmonary aspiration risk from oral nutrition.

Step 6. Parenteral nutrition. The development of parenteral nutrition in the 1960's meant that feeding was possible even in patients that did not have a functioning gastrointestinal tract. Although it is mentioned in this article as a final step in nutritional support it may also be appropriate to use early depending on clinical scenario.

Enteral versus Parenteral feeding

Enteral feeding produces gastro-intestinal luminal contents that can decrease the possibility of gut atrophy. Maintaining a normal intestinal mucosa reduces the hazard of bacteria and toxins crossing the gastro-intestinal wall and therefore can decrease proinflammatory mediator levels. Compared with parenteral nutrition, enteral feeding attenuates the acute phase response and improves disease severity in acute pancreatitis. (11) Hernandez et al have shown that enteral feeds decrease gut mucosal atrophy in critically ill patients. (12) A meta-analysis has shown that in acute pancreatitis use of EN was associated with a significant reduction in infectious morbidity, hospital length of stay, and a trend toward reduced organ failure when compared with use of parenteral nutrition. (13) However it is important to note that many of the studies involving parenteral nutrition had full dose daily calorie intake whereas enteral feeding studies were less likely to reach estimated energy requirements. This is significant since ill stressed patients should not be given full calorie energy requirements in the first 24-48 hours of commencing feeding. Therefore the parenteral feeding groups were disadvantaged in that the patients were overfed initially and received excess energy calorie intake. National Institute of Clinical Excellence states that parenteral nutrition is only to be used in patients with "inadequate or unsafe oral and/or enteral nutritional intake and a non-functional, inaccessible or perforated (leaking) gastrointestinal tract." (14) Enteral nutrition can be provided by a number of

methods including nasogastric tubes, nasojejunal tubes, gastrostomy tubes (including PEG tubes) and jejunostomy tubes (including PEG-J and D-PEJ). Parenteral nutrition can be given peripherally for approximately 14 days but central access should be used if greater than 14 days.

Refeeding syndrome

Refeeding syndrome is a potentially lethal condition with severe electrolyte and fluid shifts with resulting metabolic disturbances in malnourished patients. (15) It is caused by refeeding in malnourished patients with resulting insulin release and intracellular movement of potassium, phosphate and magnesium and increased thiamine uptake. It is essential that patients are correctly identified (See Table 2) and electrolytes corrected and intravenous Vitamin B and C is given. Feeding should be initiated slowly. Fluid balance and electrolytes should be monitored closely.

Table 2 NICE guidelines: Risk of refeeding syndrome (14)

>1 of BMI <16.5 kg/m ² Weight loss >15% No food intake 10 days Decreased magnesium/phosphate/potassium
>2 of BMI <18.5 kg/m ² Weight loss <10% No food intake 5 days Alcohol abuse, Insulin, Chemotherapy

Cardiovascular disease and nutrition

Research has confirmed link between cardiovascular disease and serum cholesterol. The NHANES II data has shown that patients who died from myocardial infarction had increased cholesterol. (16) Hooper et al confirmed that dietary fat is linked to cardiovascular risk. (17) This group demonstrated that decreasing total dietary fat over 6 month period reduced cardiac events by 16%. The Seven Countries Study illustrated that cardiovascular mortality was linked to saturated fat intake. (18)

There has been recent interest in the role of the Mediterranean diet in the prevention of cardiovascular disease. (See Table 3)

Table 3 Mediterranean Diet Studies

Population Studies CARDIO2000. Panagiotakos et al 2002. (28) Greece. 2000-2002. CVD group versus control group. Intake of Mediterranean diet type foods significantly decreased risk of developing CVD. The daily use of olive oil and consumption of vegetables, legumes, cereals and fish was associated with 23% risk of developing acute coronary syndromes. Trichopoulou. 2005. (29) Studies relationship between Mediterranean diet and survival in 1302 Greek patients with CVD. Patients with higher compliance with Mediterranean diet had lower cardiac mortality.

Martinez-Gonzalez. 2002. (30) Case-control study. Examined the relationship between a Mediterranean diet and risk of first MI. Better compliance with Mediterranean diet lowered risk of MI.

Intervention Studies

Medi-RIVAGE. Dietary intervention Study. (31) 3 month diet. Compared Mediterranean diet and low fat diet. There was greater reduction in cardiovascular risk in Mediterranean diet group compared to low fat group (15% vs 9%).

GISSI-Prevenzione. Barzi et al. (32) Supplement of Vit-E and omega-3 FA's given to survivors of recent MI. Also informed to increase intake of Mediterranean diet foods. Outcomes showed that lower chance of premature death if higher intake of Mediterranean foods.

Lyon Diet Heart Study. (33) Key study in relation to Mediterranean diet and CVD. Secondary prevention trial. Examined the effects of a Mediterranean diet in survivors of first MI. Experimental group **supplied** with margarine with high levels of alpha-linolenic acid (n-3 FA). Mediterranean diet group had lower total mortality (70%).

Patients were previously educated regarding low fat diet benefits but Mediterranean diet appears to have enhanced beneficial effects. There is however some complexity with definition of a Mediterranean diet. The diet is based on dietary patterns of Greece, Crete and Southern Italy in 1960's. This diet was abundant in plant foods, fresh fruit, olive oil as principal fat source (monounsaturated fatty acid), low red meat intake, fish (polyunsaturated fatty acid) and red wine in low to moderate amounts. In respect to dietary fats the Mediterranean diet is low in saturated fat but high in monounsaturated fatty acid and polyunsaturated fatty acid.

Diabetes and nutrition

The diabetic diet is essentially a healthy diet. The total fat intake should be less than 35% with saturated fat less than 10%. Total monounsaturated fatty acid 10-20% and polyunsaturated fatty acid less than 10% which corresponds to oily fish 1-2 per week. Interestingly polyunsaturated fatty acid supplements should be avoided in this group as they have been shown to increase LDL-cholesterol. Diabetic patients should also be encouraged to eat regular starchy carbohydrate, avoid sugar, increase fibre, eat regular meals and snacks and avoid diabetic foods.

Cancer and nutrition

Is there a link between diet and cancer?

The World Cancer Research Fund states that 30% of all cancers could be prevented by a change in diet, increased physical activity and healthy weight. Animal studies and metabolic studies have been performed that reveal evidence linking diet and cancer. An ecological study linking diet and cancer compared risk of cancer against intakes of fat, cereals and vegetables in 39 countries (19). The ATBC was a clinical trial

that related Vitamin A and E to increased lung cancer rates. (20) 2 large cohort studies have been performed in relation to colorectal cancer and dietary fibre. (21, 22). EPIC showed a significant benefit of dietary fibre against colorectal cancer. The EPIC data showed a 40% difference in colorectal cancer rates between the lowest and highest quintile dietary fibre groups. NIH-AARP however showed no benefit when adjusted for multivariate analysis. The American Gastroenterology Association suggest that available evidence from animal, epidemiological and interventional studies does not unequivocally support protective role of fibre against development of CRC. However the whole body of evidence is analysed overall the overall conclusion is that there is an inverse relationship between dietary fibre and CRC.

Cancer cachexia

Cancer cachexia is defined as anorexia, weight loss and muscle wasting, fatigue and weakness in a cancer patient. Cancer instigates an inflammatory response and production of tumour products. This triggers metabolic abnormalities that produce lipolysis, protein loss and anorexia. As with all disease processes the dietary management of cancer cachexia is initiated by a nutritional assessment. Any symptoms including vomiting and nausea should be treated. There has also been some research regarding fish oils and cancer cachexia. One available product contains docosahexaenoic acid, eicosapentaenoic acid and antioxidants. However a Cochrane review has shown no benefit in relation to beneficial effects of fish oils and cancer cachexia. (23)

Critical care

Injury and sepsis cause major disturbance to clinical state. There is rapid weight loss, increased metabolic rate, protein losses and sodium retention. There are also changes in hormone levels including increased insulin, catecholamines, growth hormone, glucagons and cortisol. The energy requirements in critical care can vary greatly. The Ireton-Jones equation is often used to calculate energy requirements in the critical care setting (See Table 4).

Table 4 Immunonutrition: ESPEN guidelines (34)

Surgical patients
Perioperatively for
Major Cancer Neck Surgery
Major Cancer Abdominal Surgery
Critical Care Patients
Elective Upper GIT surgery
Mild Sepsis
Trauma
ARDS (n-3 FA's and antioxidants)
Glutamine (burns and trauma)

The specific immune modulating aspects of nutrients have been widely researched in the critical care setting. This field of immunonutrition has shown disparaging results.

Arginine has been investigated as an immune modulating nutrient. Arginine levels can increase or decrease in relation to clinical state. It produces nitric oxide that can lower blood pressure and has been revealed to have detrimental effects in critically ill patients. (24) Glutamine is another possible immune modulating nutrient. It has lots of metabolic functions. It enhances heat shock protein that protect against sepsis. It is thought to be useful in septic shock. Omega-3 fatty acids (alpha-linolenic acid, docosahexaenoic acid, eicosapentaenoic acid) have also been investigated as an immune modulating nutrient. Omega-3 fatty acids produce less proinflammatory eicosanoids than omega-6 fatty acids. There is some evidence that omega-3 fatty acids decrease duration of hospital stay (25) Antioxidants have also been researched as an immunomodulator. Antioxidant levels are lower in critically ill patients. Vitamins A,C,E and Selenium have been studied. A recent meta-analysis has suggested overall mortality benefit but no septic complications benefit in antioxidant trials. (26)

Gastrointestinal disease and nutrition

Liver disease

The energy requirements in chronic liver disease are dependent on clinical state i.e. compensated or decompensated. Nutritional requirements for compensated liver disease are 25-35 kcal per kg (dry body weight) day and protein 1.2g per day. Nutritional requirements for decompensated liver disease are energy 35-45 kcal per kg (dry body weight) day and protein 1.5grams per day.

Porto-systemic encephalopathy

This disease process is multifactorial and comprises increased ammonia levels, increased aromatic amino acids, decreased branched chain amino acids and alterations of brain neurotransmitters. There is a widely held belief among doctors that protein intake should be restricted but this is mistaken. Protein requirements are approximately 1g/kg/day and should be divided throughout day.

Ascites

Low salt intake (<6g per day) is an essential component of ascites treatment. Advice should include no salt in cooking, no added salt, avoid processed foods, and avoid foods rich in salt.

Inflammatory Bowel Disease

Inflammatory bowel disease patients are often undernourished due to meagre intake (anorexia, vomiting), amplified losses (diarrhoea and malabsorption) and increased demands

(catabolic state). Protein requirements are also increased due to nitrogen losses and catabolic state. It is therefore important that nutritional measures are instituted to improve calorie and protein intake. Interestingly nutrition has been investigated as a treatment for active Crohn's Disease. Elemental diet is used in active paediatric Crohn's Disease more than adult Crohn's Disease to achieve remission. (27). Often this has to be given via naso-gastric tube due to unpalatability. After 4-6 weeks if the patient is in remission foods are introduced slowly over a 3-week period.

Coeliac Disease

Coeliac disease is genetically determined chronic inflammatory disease secondary to gluten (gladin is the alcohol-soluble fraction) that is a component of wheat. In addition the allergy involves similar proteins found in barley, rye and possibly oats. Coeliac patients consequently exclude these dietary sources. Oats can be reintroduced later depending on response. Dietary sources can be obvious or hidden as gluten can be found in numerous manufactured foods. Coeliac patients can however eat natural gluten-free foods or gluten-free proprietary foods (e.g. Schar, Juvela, Dietary Specials, Glutafin).

Irritable Bowel Syndrome

Simple healthy eating advice is suggested. Diet is tailored to either constipation or diarrhoea symptoms. Typically increasing dietary fibre gradually ameliorates constipation symptoms. Soluble fibre appears to offer benefit more than non-soluble fibre. Decreasing dietary fibre intake treats diarrhoea symptoms. There is not enough evidence regarding exclusion diets although some centres do offer exclusion diets.

Renal disease

Acute renal failure and nutrition is divided into non-catabolic and catabolic patients. Non-catabolic patients do not usually have increased energy requirements. Catabolic patients have high protein requirements but no benefit of >0.2 g nitrogen per kg per day. Their energy requirements should be no greater than >20% above resting energy expenditure.

Chronic kidney disease patients have estimated energy requirements of 35 kcal/kg/IBW/day. (IBW = Ideal Body Weight which in the UK this approximates to BMI 23kg/m²). There has been much research regarding protein restriction and possibility of slowing progression of chronic kidney disease. The Northern Italian Co-op study (protein <0.6grams/day) did show possible slower progression of CKD. However it is known that low protein diets have poor compliance and can increase risk of malnutrition. The Renal Association Standards suggest protein intake 0.75g/kg/IBW/day.

Discussing the dietary management of end-stage renal disease, nephrotic syndrome and renal stones is beyond the scope of this article.

Conclusion

This review article has highlighted the importance of undernutrition in patients under our care. There are numerous methods of screening and assessing patients for undernutrition. There is also a stepwise approach to improving calorie intake: improving oral intake by various methods to enteral and parenteral nutrition. Nutrition is an important aspect of treatment of different disease processes that include cardiovascular disease, diabetes, gastrointestinal disease, renal disease, critical care and cancer. This review article will hopefully provide the medical practitioner with improved knowledge that can be translated into improved awareness and treatment of undernutrition.

COMPETING INTERESTS

None Declared

AUTHOR DETAILS

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