Turkish Journal of Cerebrovascular Diseases 2022; 28(3): 128-140 Türk Beyin Damar Hastalıkları Dergisi 2022; 28(3): 128-140
Turk J Cereb Vasc Dis
doi: 10.5505/tbdhd.2022.15013

REVIEW **DERLEME**

NUTRITION IN ACUTE STROKE: PROBLEMS AND SOLUTIONS

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ABSTRACT

The major consequences of acute stroke are malnutrition, dysphagia and muscle loss. Nutritional treatment should be included in the management protocols of acute ischemic and hemorrhagic stroke as early as possible. Malnutrition prevalence is not rare among acute stroke patients at the time of admission, however it is more common after hospitalisation in neurointensive care, stroke units or inpatient neurology wards together with noticeable muscle loss. Altered mental status and swallowing disturbance may lead to nutritional deficiencies, dehydration, aspiration and pneumonia. In fact, oral and pharyngeal phases of swallow and laryngeal elevation is disturbed after stroke. Dysphagia is caused by lesions in medulla, pons, parietal operculum, insula, anterior and superior temporal regions, precentral and postcentral gyrus, cingulate cortex. Dysphagia, loss of cough reflex, vallecular pooling, swallowing apraxia and aspiration are especially more common in right hemispheric strokes. All stroke patients should be examined for the presence of dysphagia with bedside tests within 24 hours of hospitalization and instrumental assessment later on. Enteral nutrition should be applied, if needed. It prevents the development of malnutrition and muscle loss. Patients who are fed properly have low rates of complications during the hospital stay, and better functional recovery in long term. Neurologists who take care of hospitalized stroked patients, should be able to plan enteral nutrition and master the main principles of the treatment. Stroke related muscle loss differs from sarcopenia in terms of physiopathology and diagnostic work-up. Quantitative demonstration of muscle mass with BIA, DEXA, CT, MR or ultrasonography is crucial for diagnosis. For today, the novel methods for prevention and management of stroke related muscle loss are active exercises and nutrition.
Keywords: Stroke, malnutrition, dysphagia, enteral feeding.

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Please cite this article as following: Güngör L. Nutrition in acute stroke: Problems and solutions. Turkish Journal of Cerebrovascular Diseases 2022; 28(3): 128-140. doi: 10.5505/tbdhd.2022.15013

AKUT İNMEDE NÜTRİSYON: SORUNLAR VE ÇÖZÜM YOLLARI

ÖZ

Akut inmeden sonra ortaya çıkan başlıca nütrisyonel sorunlar malnütrisyon, disfaji ve kas kaybıdır. Nütrisyonel tedavi, akut iskemik ve hemorajik inme tedavi basamakları arasında mümkün olduğunca erken yer almalıdır. Malnütrisyon akut inme sırasında nadir değildir, ama daha sık karşılaşılan, inme hastasında nöroloji yoğun bakım, inme ünitesi ve hastane yatışı sırasında hızla gelişen malnütrisyon ve kas kaybıdır. Bilinç etkilenmesi ve yutma bozukluğu beslenme yetersizliğine, dehidratasyona, aspirasyon ve pnömoniye yol açar. İnmeden sonra yutmanın oral ve farengeal fazları ile larengeal elevasyon bozulur. Medulla oblongata, pons, pariyetal operkül, insula, anterior ve superior temporal alanlar, presentral ve postsentral gyrus, singulat korteks lezyonlarında disfaji bildirilmiştir. Özellikle sağ hemisfer lezyonlarında disfaji, yutma apraksisi, öksürük refleksi kaybı, valleküler göllenme ve aspirasyon daha sık olur. Her inme hastasının yatışının ilk 24 saatinde yutma fonksiyonu açısından yatak başı testler ve ardından enstrümental yöntemlerle değerlendirilmesi gerekir. Gerek varsa enteral nütrisyon, hem malnütrisyon gelişimini hem de kas kaybını önlemek için faydalıdır. İyi beslenen hastalarda hastane yatış boyunca komplikasyon oranları azalmakta, uzun dönemde fonksiyonel iyileşme daha fazla olmaktadır. İnme hastalarını yatırarak takip eden her nöroloji uzmanı enteral nütrisyonu uygun şekilde planlayabilmeli, temel prensiplerine hakim olmalıdır. İnme ile ilişkili kas kaybı hem fizyopatoloji hem de tanı basamakları yönünden sarkopeniden farklılıklar içerir. İnme hastasında BİA, DEXA, ultrasonografi, BT, MR ile ekstremite ve gövde kaslarının kantitatif olarak gösterilmesi tanıda kritiktir. Bugün için inme ile ilişkili kas kaybını önlemenin ve tedavi etmenin yegane yöntemi aktif egzersiz ve nütrisyondur.
Anahtar Sözcükler: İnme, malnütrisyon, disfaji, enteral nütrisyon.

Introduction

Approximately 125,000 people suffer a stroke annually in Turkey, and 82,000 of these are ischemic strokes (1). While surgical treatment can be lifesaving in some patients with intracerebral hemorrhages, the focus is on intravenous tissue-type plasminogen activator infusion in the first 4.5 hours or recanalization treatment with mechanical thrombectomy in the first 6 hours in an acute ischemic stroke. Treatment for the preservation of the lesion and the vulnerable tissue around it in ischemic or hemorrhagic strokes, whether recanalization treatment or surgical intervention is performed or not, includes good oxygenation, fluid replacement, optimization of blood pressure and blood sugar, prevention of fever, and correction of intracranial pressure elevation, if any. After ensuring cerebral hemodynamics, possible complications of stroke such as infection and seizures are handled. Early rehabilitation and secondary prophylaxis method should be determined to prevent early recurrence by determining the etiologic subtype of stroke (2,3). The patient's nutritional and swallowing status must be evaluated, and an appropriate nutritional strategy must be developed, while carrying out all these intensive and complex treatments. In stroke patients, nutritional assessment and management will lessen nutritional complications and aid in functional recovery.

Acute Stroke and Malnutrition

16-25% of stroke patients are malnourished at presentation, and the malnutrition rate increases to 60% as the length of hospital stay is prolonged (4-6). Stroke-induced systemic stress reaction, loss of consciousness, and dysphagia are direct risk factors for malnutrition which is also common in stroke patients. Those with pre-existing chronic diseases, those who have had a previous stroke, those who use multiple drugs, and the elderly are more likely to be malnourished at the time of stroke presentation (4). In contrast, patients who were obese and had a body mass index (BMI) of 30 kg/m^2 or higher had lower mortality rates both during hospitalization and 1.5 years after a stroke. Although the underlying mechanism —known as the obesity paradox— cannot be fully understood, it is important to avoid trying to make the patients gain weight (7).

Every stroke patient should be evaluated for the presence and risk of malnutrition after hospitalization. For nutritional assessment, first, anthropometric values should be measured. These include measurements of body weight, height, triceps skinfold thickness, arm circumference, waist circumference, and calf circumference. Height divided by kilograms squared yields the BMI (kg/m^2). It is a crucial indicator for the malnutrition diagnosis (8).

Triceps skin thickness is measured on the

back of the arm with a caliper held at right angles with the finger. One third of the fat in the body is located under the skin. Triceps skin thickness gives quick information about body fat ratio and lean body mass. It is estimated that the fat ratio is 20% when it is 25 mm in men and 30% when it is 18 mm in women (9). Global Leadership Initiative on Malnutrition (GLIM) criteria defined in 2019 are used for malnutrition assessment and diagnosis (Figure 1) (8).

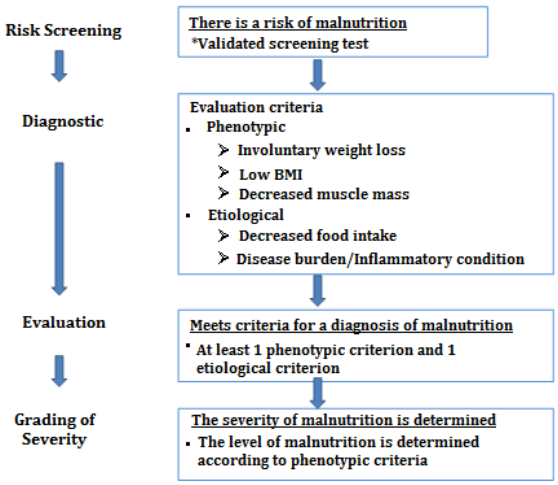

Figure 1. GLIM diagnostic algorithm for malnutrition (8).

According to the consensus criteria, first, a validated screening test should be used to determine whether there is a risk of malnutrition. As malnutrition screening tests, Nutrition Risk Screen-2002 (NRS-2002), Mini Nutrition Assessment-Short Form (MNA-SF), Malnutrition Screening Tool (MST), Geriatric Nutritional Risk Index (GNRI), Malnutrition Universal Screening Test (MUST), and Short Nutrition Assessment Questionnaire (SNAQ) are used. A patient who has been identified as at risk needs to be phenotypically diagnosed. This is based on a BMI of 20 or lowers for people under the age of 70 and below 22 for people over the age of 70, or on losing more than 5% of body weight in the last six months or more than 10% in more than six months. Muscle mass loss, if present, is also considered a phenotypic criterion. Malnutrition is defined as the presence of at least one of these criteria as well as an etiological criterion. There must be a systemic, gastrointestinal, or inflammatory disease that can cause malnutrition or a reduction in food intake. Decreased food intake is defined as less than 50% of the required intake in the last week or reduced food intake for more than 2 weeks, or impaired digestion or absorption of food due to a chronic gastrointestinal disorder (8).

In stroke patients, there is no biomarker of malnutrition that can be measured in the blood. Although serum total protein, albumin, and prealbumin levels can provide a general consideration, those are not reliable markers as they can be affected by various medical conditions. Monitoring serum B12, folic acid, and vitamin D levels in stroke patients and correcting any deficiencies may provide clinical benefits (10).

If a stroke patient is malnourished at the beginning or develops malnutrition during hospitalization, the outcome worsens. Malnutrition increases mortality, lengthens hospitalization, increases infection, and limits functional improvement in skills such as standing, using hands, and walking in stroke patients (11-14).

Acute Stroke and Dysphagia

Dysphagia is detected in 8-80% of patients with acute stroke, depending on the method of evaluation (15,16). Dysphagia increases the risk of aspiration, pneumonia, dehydration, and malnutrition. Post-stroke dysphagia resolves spontaneously in most patients. While dysphagia improves in half of the cases within the first two weeks, this rate rises to 85% after one month. At the end of one year, only 2% of patients have persistent dysphagia (17).

The development of dysphagia after stroke is highly correlated with infarct localization. Lateral medullary infarcts involving the nucleus tractus solitarius, the sensory central pattern generator of swallowing, and the nucleus ambiguous, the motor pattern generator, cause prolonged dysphagia. Contrary to popular belief, dysphagia is also common in supratentorial lesions. While the oral phase is mostly disrupted in the left hemisphere lesions, the pharyngeal phase is mostly disrupted in the right hemispheric lesions, penetration and aspiration occur. Dysphagia is common in bilateral external capsule, right insula, superior temporal gyrus, supramarginal gyrus, temporal plane, and postcentral gyrus lesions. The cough reflex is impaired and oropharyngeal residue remains in lesions of the right limbic lobe, left

parietal sensory areas, and right parietal operculum. Penetration and aspiration are seen in right anterior temporal region and right postcentral gyrus infarcts. The cough reflex is weakened in lesions of the paralimbic region; somatosensory association areas, anterior cingulate cortex, and left parietal operculum. Lesions involving the precentral gyrus, frontal areas, and thalamus are more likely to cause pneumonia. Vallecular pooling and prepriform residues are still present in pons lesions. Cortical small infarcts in the irrigation area of the middle cerebral artery can cause swallowing apraxia without significant motor loss (18-21). Sarcopenic dysphagia should be suspected in patients whose dysphagia does not improve for a long time. Sarcopenic dysphagia refers to the further deterioration of swallowing function due to sarcopenia that develops in the swallowing muscles as well as the whole-body muscles. In these patients, swallowing function is restored when sarcopenia is corrected (22).

After cerebrovascular stabilization, every stroke patient should be evaluated for dysphagia before receiving oral medication and nutrition (23). For this, modified Mann Assessment of Swallowing Ability (mMASA) (24), Toronto bedside swallowing wound test (25), Barnes-Jewish Hospital stroke dysphagia screening test (26), Gag swallowing screening (27), and emergency physicians dysphagia screening (28) tests can be used. The first three were validated in Turkish. These tests include examination of the tongue and pharyngeal muscles, cough and pharyngeal reflex, observation of swallowing water and foods of different consistencies. The most used is the bedside water swallow test. The 50 cc or 3 ounce (90 ml) water swallow tests are most commonly used (29). 5 ml of water is first drunk in a spoon or glass by the patient who can communicate and sit upright. If the patient experiences coughing, bruising, gagging, or a wet sound after swallowing, as well as choking or wheezing while swallowing, the patient is considered unable to swallow safely. The patient, who is seen to swallow 5 cc of water comfortably three times, is given 50 cc of water in a glass this time. With the observation of the same findings, it is decided that the patient swallows safely. Observing the oxygen saturation remains above 92% with a peripheral probe during the test increases the reliability of the test. Patients who cannot safely complete these steps are not given oral food or medication, nasogastric tube feeding is initiated, and advanced swallowing evaluation is performed as soon as possible (30).

Oropharyngeal dysphagia after stroke is characterized primarily by the inability to swallow liquids; however, the patient can swallow thick or solid foods which are determined by volume-viscosity tests. It is done by evaluating aspiration findings such as coughing and making a wet sound while swallowing foods of various consistencies such as buttermilk, nectar, syrup, yogurt, and pudding, which start with water and gradually increase in consistency, in a glass and with a spoon (31).

Instrumental advanced swallowing evaluation shows which stage of swallowing is impaired and the severity of dysphagia in a stroke patient. It is useful to see whether aspiration can be prevented with special postural arrangements or maneuvers, whether swallowing can be corrected, and whether safe swallowing can be achieved with different consistencies. It helps to determine which rehabilitation method and which exercise will be preferred. Fiberoptic Endoscopic Evaluation of Swallow (FEES) and video fluoroscopy (VFS) are the two most widely used advanced swallowing evaluation methods (18). FEES is based on direct observation of findings such as aspiration, pharyngeal pooling and residue, which occur when the patient uses a flexible short endoscope to reach the oropharynx and swallows liquids and foods of varying consistency coloured with food colouring. All stages of swallowing are monitored simultaneously inside through imaging of the patient sitting upright in a fluoroscopy device and drinking water mixed with contrast material, during VFS. While the benefits of FEES include the ability to perform it at the bedside, the ability to repeat it at short intervals and the lack of radiation, the disadvantage is that it requires experienced staff, and the moment of full swallowing cannot be visualised directly. The advantage of VFS is that it can show all phases of swallowing, including the oral phase, upper esophageal sphincter function, and the disadvantage is that the patient must go to the scope room and includes radiation (18).

Three methods can be used in the treatment of dysphagia after stroke: restorative methods, compensatory techniques and adaptive methods.

The aim of restorative methods is to provide maximal improvement in impaired swallowing function. Compensatory treatments are based on altering the physiology of swallowing without correcting the underlying neuromuscular dysfunction. In adaptive methods, an adaptation to swallowing disorder is tried to be achieved with external support, namely enteral or parenteral nutrition and diet modification (32).

Restorative techniques include stimulation methods with tongue, lip, palate, neck, jaw, larynx, and pharynx exercises that will strengthen different swallowing muscles. Shaker exercises are a 60-second standing movement by flexing the neck while lying down. It strengthens the suprahyoid muscles, facilitates the opening of the upper esophageal sphincter, prevents aspiration, and facilitates swallowing. In the Masako maneuver, the patient does swallow exercises by biting his/her tongue. Each time, an attempt is made to remove more of the tongue. Cheek puffing and holding the tongue in and out of the mouth left and right, up, and down for 6 seconds are examples of oropharyngeal muscle training. These movements are performed 4 times a day with 15 repetitions each time. By strengthening the tongue, soft palate, and pharyngeal muscles, oropharyngeal exercises increase the pushing power of the bolus, strengthen the oral phase, accelerate the pharyngeal phase, and reduce the amount of pharyngeal residue and aspiration (33). Biofeedback systems can be combined with jaw opening exercises performed with elastic bands attached to the head under the chin. Flow spirometry or special instruments designed for this purpose are used to perform respiratory muscle exercises. Expiratory or inhalation exercises help strengthen the cough reflex and reduce the risk of pulmonary complications (34,35).

Postural adjustments such as elevated head posture, turning the head towards the hemiparetic side, and chin tuck during swallowing passively provide closure of the epiglottis and prevent aspiration when the patient is unable to elevate the laryngeal. Swallowing maneuvers are also aimed at pushing the bolus towards the esophagus and closing the epiglottis without aspiration. During supraglottic swallowing, the patient holds his/her breathe and coughs slightly after swallowing to clear his throat. It prevents aspiration. The patient is asked to hold his/her breath and swallow by pushing during super-supraglottic swallowing which enhances laryngeal movement. Stroke patients may struggle with forced swallowing maneuver in which the patient is asked to swallow while the muscles of the jaw, pharynx, palate, and neck are contracted. It allows the tongue to push the bolus, reduces pharyngeal residue and aspiration. In the Mendelsohn maneuver, the patient is asked to swallow after lifting the larynx upwards with his/her hand. This maneuver also increases the swallowing coordination (36).

Neuromuscular stimulation can be done simply by thermal or tactile stimulation into the mouth, palate and oropharynx. Transcranial magnetic stimulation (TMS) is performed by repeating a 5Hz stimulus given to the cortical region of the pharyngeal muscles for 10 seconds 10 times at 1-minute intervals. It reduces the penetration and aspiration severity (37). Transcranial direct current stimulation (tDCS) uses an electrical stimulation of 1.0-1.6 mAmp on the scalp in the intact or infarcted hemisphere to stimulate the swallowing muscles (38). Neuromuscular electrical stimulation (NMES) is the application of bidirectional current between 0-15 mV with a wavelength of 700 ms for 30 minutes with two electrodes placed under the thyroid cartilage and under the hyoid. It doubles the effect of swallowing exercises when continued for 4 weeks, 5 days a week (39). All these applications can be performed in conjunction with the stroke neurologist and speech and language therapist. Examinations and VFS or FEES should be used to monitor the effect of exercise, postural regulation, and stimulation treatments on swallowing.

Stroke Related Muscle Loss

Sarcopenia was first described as muscle wasting and functional impairment that occurs with age in the geriatric patient population (40). The European Working Group on Sarcopenia (EWGSOP) defines it as a syndrome characterized by a progressive and generalized decrease in muscle mass and strength, which can result in negative consequences such as physical disability, decreased quality of life, and death (41). Muscle wasting that occurs naturally with age is referred to as primary sarcopenia, whereas muscle loss that occurs as a result of surgery, inflammatory, oncological, or other systemic diseases is referred

to as secondary sarcopenia. One of the diseases that contribute to the development of sarcopenia is stroke. Sarcopenia is diagnosed by demonstrating loss of muscle strength or function, as well as muscle wasting, using ultrasound, magnetic resonance, or computed tomography (CT), or by demonstrating a decrease in lean body mass using dual-energy X-ray absorptiometry (DEXA) or bioelectrical impedance analysis (42,43). In recent years, it has been reported that measuring the volume of both temporal muscles from cranial CT sections can be used to show whole body muscle mass and muscle loss (42,44). The titin N-fragment in urine and the serum C-terminal agarin fragment are related to muscle wasting after stroke (42). It is not possible to clearly distinguish whether the loss of muscle function and decrease in muscle mass are caused by sarcopenia or damage to the brain in a stroke patient. Therefore, the term "stroke-associated muscle wasting" is preferred to "sarcopenia" to express loss of skeletal muscle function and volume that cannot be explained by upper motor neuron damage in stroke patients (45, 46).

It is now known that muscle loss, which can be quantified, begins in the early post-stroke period (1-3 weeks) (47-51). During the rehabilitation process, the rate of stroke-related muscle wasting increases, especially in patients who are immobile and undernourished. A meta-analysis of about 1700 patients found that stroke-related muscle wasting was 50% at the end of the first month and 34% at the end of the sixth month (52). Stroke-related muscle loss occurs not only in the paretic side but also in the non-paretic extremity (46,53-55). Extremity paralysis is the most common finding in stroke patients that determines functional loss. When muscle loss is added functional capacity decreases over time, and the ability to walk, use hands, and carry out daily activities independently deteriorates (56-58). For this reason, the neurologist who treats and follows the stroke patient should know the ways to prevent the development of sarcopenia and to diagnose and correct it, if any.

The pathophysiology of muscle wasting following a stroke is quite complicated. The main causes are immobility and a failure to meet daily protein and calorie requirements. Muscle wasting is exacerbated by spasticity, disuse atrophy, denervation, and sympathetic activation. Muscle catabolism is exacerbated by systemic and local inflammation in muscle tissue that occurs as a result of a stroke (47,59). Histopathologically, primary sarcopenia seen in old age differs from muscle wasting after stroke. Myosin heavy chain fibers in stroke patients transform into fast-twitch type IIa and type IIx slow-twitch muscle fibers, the opposite of the slow-twitch type I fibers seen in primary sarcopenia (45,60).

Two methods that have been shown to be effective so far for the treatment of sarcopenia are physiotherapy and an appropriate nutritional therapy (61,62). Meeting daily protein and calorie needs is very critical for the prevention and correction of muscle wasting development. Although many different drugs and nutraceuticals have been tried in the treatment of sarcopenia, none have been routinely used in daily practive (59). Evidence shows that the essential amino acids leucine, hydroxymethyl butyrate, and vitamin D have a positive effect (63-65). Active and aerobic exercises, such as elastic band resistance and stretching exercises, maximum voluntary isometric contraction, leg pressing with knee extension, progressive high-speed exercise against low resistance, and weightlifting, are the most effective treatment. Exercise reduces inflammation in the muscle, maintains mitochondrial homeostasis, and stimulates angiogenesis and myogenesis (66,67).

Nutritional Therapy in Acute Stroke Patient

In all stroke patients who are unconscious and cannot swallow safely or have a communication deficit in the bedside water swallow test, enteral nutrition should be started via a feeding tube within the first 24-48 hours (18,30,68). 30% of acute stroke patients require tube feeding. Early enteral nutrition shortens the duration of intensive care, and hospital stays and decreases pneumonia, complications and mortality rates (10,69,70). Stroke patients can consume an average of 60% of the calories they require while hospitalized. This deficit is lower in patients who receive enteral nutrition (7). Enteral tube feeding not only meets protein, calorie, and micronutrient needs in dysphagic stroke patients, but it also aids in dysphagia recovery and reduces aspiration (71).

The resting energy needs of stroke patients are not different from those of other patients in the acute and subacute periods. Patients with left hemisphere lesions have a slightly lower calorie

requirement. Caloric requirements increase by 25% in patients receiving active rehabilitation in the chronic period. Subarachnoid and intraparenchymal hemorrhage increase daily caloric requirement by 12-14%. Additional infection in the acute period increases caloric requirements (7,72).

Indirect calorimetry, which is based on the principle of measuring the amount of CO_2 in the patient's breath, is the gold standard technique for determining the daily calorie requirement. VO_2 measured from the pulmonary arterial catheter or VCO_2 (carbon dioxide production) measured from the ventilator expiratory tube can be used to calculate the energy consumption of ICU patients who do not have a calorimetry device (73). Predictive formulas based on different parameters such as age, height, body weight, BMI, and lean body mass can be used instead of calorimetry. These formulas have a 70% compatibility rate with the resting energy consumption calculated by indirect calorimetry. It is the Harris-Benedict formula with the highest correlation and the most used (74). In the original Harris-Benedict calculation, for men 66.5+(13.76 x weight kg)+(5.003 x height cm)-(6,755 x years of age), and for women 66.5+(9.563 x weight kg)+(1.85 x height cm)-(4,676 years of age) formula is used. In general, the resting energy expenditure is supposed to be 20-25 kcal/kg in patients who cannot get out of bed daily, and as 25-35 kcal/kg in mobilized patients. It should be noted that in cases where basal metabolism and need increase, such as advanged age, multiple co-morbidities, active exercise, systemic infection, metabolic stress, and pressure ulcers, basal energy consumption will increase. The calorie calculation can be increased to 25-35 kcal/kg/day depending on the need (10,73). If indirect calorimetry is not used, it is recommended to give 70% of the calculated calories in the first week and increase this dose at the end of the third day in acute stroke patients who require to be admitted to the intensive care unit. Beginning in the second week, 80-100% of the target calories can be consumed. If indirect calorimetry is used for energy consumption, hypocaloric feeding (70% of need) is given for the first three days, then isocaloric feeding (73).

The daily amount of protein to be given to patients should be 1.0-1.5 g/kg. When protein is given at a dose of 1.2-1.7 g/kg/day by enteral tube, the mortality rate in the sixth month decreases significantly in stroke patients who need to be admitted to the intensive care unit (75).If there is muscle wasting, pressure sore, or infection, higher protein values can be given (2.0 gr/kg/day) (68,73). When calculating the dose of enteral products, the amount of water taken should be kept in mind. The fluid requirement per day is 30-35 ml/kg. It should be considered that 80-85% of the enteral nutrition products are water.

The calorie and protein needed per kilogram should be determined in these calculations, and the patient's actual weight should be measured with a scale. Stroke patients who are unable to stand are weighed using special patient beds or patient lifts that can measure weight. The calculations take into account the ideal body weight rather than the current weight. Ideal body weight is the weight that should be corrected for height and gender and is calculated with the formula [0.91 x (actual height cm–152.4)]+50 for men and [0.91 x (actual height cm-152.4)]+45.5 for women. When current body weight is used in obese patients (BMI>30), patients are given too many calories; therefore, using body weight adjusted for ideal body weight and BMI provides a more accurate calorie prediction. Adjusted body weight is calculated with the formula [(actual body weight–ideal body weight) x 0.33]+ ideal body weight [73]. In low-weight patients (BMI <20 for those under 70 years of age, BMI<22 for those over 70 years of age for Europe and America, BMI<18.5 for those below 70 years of age, BMI<20 for those over 70 years of age) for Asian population if the ideal body weight is used, the patient suddenly consumes an excessive number of calories. Therefore, it is recommended to choose the actual body weight rather than the ideal body weight (8).

It is advised to start nutrition with standard enteral formulas. Maltodextrin and corn syrup are carbohydrate sources in standard products, casein, lactalbumin, whey protein, or soy protein are protein sources, and canola, soy, or safflower are fat sources. Carbohydrates provide approximately 50% of the energy in these products, fats 30%, and protein 15%. 500 cc of product typically contains 500 kcal and 20 g of protein. Its osmolarity ranges from 180 to 330 mOsm. High energy products are used to provide more calories in a small amount of space. The fat content has been increased. A 500-cc product typically contains 750 kcal of energy. Protein content in high protein products is obtained from different sources such as whey

protein, casein, lactalbumin and it has been increased to approximately 40-50 g per 500 cc. In multifiber products, the ratio of soluble (pectin, gum, benefiber, inulin, maltodextrin) and insoluble (cellulose, hemicellulose, lignin) fiber was increased to 7.5-15 g. The daily fiber requirement is 15-30 gr. In diabetic products, the fat rate was increased, the carbohydrate source was reduced, and the amount of fiber was increased compared to standard products. It facilitates regulation by preventing sudden blood sugar spikes and providing more stable sugar levels (68,76).

After calculating the patient's target calorie, protein, and water intake, the appropriate enteral product is infused at a rate of 20-40 ml/hour. If no signs of gastrointestinal intolerance are present, such as nausea, vomiting, increased gastric residue, diarrhea, hemodynamic instability, hyperglycemia, electrolyte disturbances, or hypercapnia, the dose can be increased by 20-30 ml every 4-12 hours. The amount of enteral product that provides the target calorie and protein values is expected to be reached after 2-3 days. During this period, there is no need to administer parenteral amino acids and lipid solutions (30,68). There is no need for routine gastric residue follow-up in every patient fed by the nasogastric route. It can only be examined in patients with gastrointestinal intolerance (nausea, vomiting, abdominal distension, decreased bowel sounds) (77). Trophic feeding is an infusion of 10-20 cc/hour to feed the intestinal wall. This dose, which cannot meet the patient's calorie, water, and protein requirements, protects the intestinal epithelium, stimulates local secretion, maintains immune function, protects epithelial tight junctions, and prevents bacterial translocation (73). Continuous infusion for 24 hours is recommended for patients hospitalized in the hospital and intensive care unit to reduce the workforce of allied health personnel, the number of set manipulations and the rate of infection, and to prevent gastric residue, nausea, vomiting, and diarrhea (73). In patients who are discharged home, intermittent infusion or bolus applications are started, which will allow the mobilization of the patient, taking into account the social status (68). In bolus application, the amount of product given at once should not exceed 200 cc. (10). While the patients are hospitalized, their tolerance and efficacy to the product or application method to be used must be monitored.

In enteral nutrition,the first-choice access route is the nasogastric feeding tube. For gastric access, thinner diameter (12 French) silicone or polyurethane feeding tubes should be used, not thick (14-16F) nasogastric tubes made of plastic. Nasogastric tubes are only used in emergencies, for gastric drainage or lavage. Thin feeding tubes can be left unworn for 4-6 weeks, but they clog faster. Post pyloric feeding is initiated in patients with increased intra-abdominal pressure, increased gastric residue, recurrent aspirations, prolonged mechanical ventilation, or delayed gastric emptying though prokinetic agents. There are two alternatives for post pyloric nutrition: nasoduodenal and nasojejunal access (73). Nasoduodenal feeding tubes are thinner (8-10F). It progresses gradually through with the use of prokinetic agents. Nasojejunal feeding tubes have curved ends and are 10-30 cm longer. If a nasoduodenal or nasojejunal tube is inserted, bolus feeding is not possible. Direct X-ray or examination of the acidity of the aspirated contents (gastric fluid) in nasogastric tubes confirms tube location.

Nausea and vomiting are the most common complications during enteral tube feeding in stroke patients. If nausea and vomiting develop, the infusion rate is slowed, and gastric emptying is accelerated by decreasing the fat and fiber content. It should be ensured that the product is not given cold; the ideal is to infuse at room temperature. Opioids and anticholinergic drugs that can cause nausea and vomiting should be avoided. Antiemetics and prokinetic agents can be used. Intravenous erythromycin, the well-known prokinetic agent, is not available in Turkey. The use of methchloropropamide in stroke patients with brain damage and domperidone in patients with concomitant cardiac problems should be done with caution (10,68).

More than three watery stools per day and more than 200 g/day are considered diarrhea. Diarrhea is typically caused by changes in intestinal flora, antibiotic use, gastrointestinal infection, high osmolarity, and the use of cold products. The product can be administered at room temperature, and fiber products with lower osmolarity can be substituted. Sorbitol, magnesium, laxatives, antibiotics, proton pump inhibitors, hypoglycemic drugs, and nonsteroidal anti-inflammatory drugs may cause of diarrhea. *Clostridium difficile enterocolitis* should be

excluded in cases that do not improve. Testing for type A and B toxin with enzyme immunoassay in stool is the fastest diagnostic method. In patients who develop constipation, drugs that may cause this should be examined, phenytoin, iron preparations, narcotic analgesics, calcium channel blockers may cause constipation. Constipation can be treated by increasing fiber intake, mobilisation, and staying hydrated. The possibility of intestinal ischemia and obstruction from insoluble fibers, particularly when used in high doses, should not be overlooked (10,68).

During enteral nutrition, metabolic disturbances can occur. Continuous infusion and diabetic products with higher fiber content and lower carbohydrate content can be used to treat hyperglycemia. The goal is to keep blood sugar levels between 140 and 180 mg/dl. Hypokalemia may develop in patients with diarrhea and hypokalemia, hypophosphatemia and hypomagnesemia can be seen in refeeding syndrome (68).

The most common mechanical complication is tube occlusion. Causes include insufficient water during tube administration, the administration of drugs such as inappropriate syrup and capsules, the use of products with a high fiber content, and frequent gastric residue follow-up. It can be tried to open with 5 ml of warm water, carbonated water and a mixture of crushed pancreatic enzymes or soda. Nasogastric tube may have local effects such as rhinitis, epistaxis, local pressure sore, nasopharyngeal edema and pharyngitis, esophageal erosion and esophagitis (68). Oral care should be given with chlorhexidine every 6 hours in every patient fed with an enteral nasogastric tube, this will reduce pneumonia and other local infections. Unnecessary use of proton pump inhibitors increases the risk of aspiration pneumonia by destroying gastric acidity (10).

Keeping the nasogastric feeding tube longer than 4 weeks is not recommended because of these complications. Patients who need enteral nutrition and oropharyngeal dysphagia for more than 4 weeks should receive gastrostomy and enteral nutrition should be continued. Gastrostomy can be opened by percutaneous endoscopic (PEG), surgical and radiologically guided (RIG-radiologic inserted gastrostomy). PEG can be opened earlier in patients whose dysphagia is predicted to last longer than 4 weeks. When the patient is able to swallow safely, closing the gastrostomy is a simple and quick procedure (79). Percutaneous endoscopy jejunostomy (PEJ) may be preferred if administration of nutritional product to the stomach causes a high risk of aspiration (10).

In enterally fed patients, the number of oral drugs should be reduced as much as possible. It is not advised to administer medications in syrup, capsule, or granule form via nasogastric tubes. Granules can be given if absolutely necessary. Before administering oral medications via enteral feeding tubes, they should be thoroughly beaten and crushed.It is given with a 50-gauge syringe after being suspended in 30 ml of water. The tube should be cleaned with at least 20 ml of water before and after the drug. Feeding is halted 30 minutes before the drug and then resumed 30 minutes later. A daily-dose drug reduces reaching the target calorie by 12-17% in a patient receiving continuous enteral infusion, while two-dose drugs reduce it by 25-30%. The drugs are definitely not given with the medical nutrition product or added to the product bag. It should be kept in mind that the dose of the oral drug may decrease during crushing, dilution, and injection. Absorption of oral drugs may be poor due to gastrointestinal vasoconstriction in patients receiving vasoactive drugs in the intensive care unit. Immediate-release drugs that require gastric acid to dissolve cannot be absorbed if given post-pylorically. Drugs in high concentrations cannot be tolerated in the intestines. Warfarin must be administered specifically to the stomach. When beta blockers and calcium channel blockers are administered to the stomach, their absorption is reduced because they have a very high first pass effect in the liver. When sustained-release tablets are crushed, these properties are lost, and high doses of the drug can be rapidly absorbed and cause toxic effects. In addition, these forms can quickly clump together and clog tubes when mixed with water. For this reason, it is not recommended to crush slow-release preparations through feeding tubes (80,81).

Aspirin can be given to a patient who has had an ischemic stroke by crushing it through a tube; its bioavailability is high. When it is pounded into the stomach, it does not cause any additional complications. Clopidogrel's bioavailability is increased, and effective blood concentrations are reached quickly when administered via tube. Warfarin adheres to the wall of the tube, only 35%

of the administered dose reaches the stomach. After adjusting the dose according to the prothrombin time, the tube is no longer needed, and if the drug is taken orally, the INR value will increase rapidly, a new dose adjustment is necessary. Apixaban, rivaroxaban, and edoxaban are direct oral anticoagulants that can be administered to the stomach via tube. The administration of dabigatran capsules via a feeding tube is not recommended. Since edoxaban does not adhere to the tube wall, its bioavailability remains unchanged. Apixaban should be reconstituted with 60 mL of 5% dextrose. As rivaroxaban is a drug that is better absorbed when taken with food, starting the enteral product infusion immediately after preparing the solution with 50 ml of water improves its bioavailability (81,82).

Atenalol and amiadaron do not provide effective plasma concentrations when administered via a tube. Quinapril preparations contain magnesium bicarbonate, and the drug breaks down when crushed. Amlodipine tablets are rapidly denatured after crushing and reconstituting, so they should be given promptly.

Oral supplementation should not be started on a regular basis in stroke patients. The Feed Or Ordinary Diet (FOOD) study, which was the first and is still the most extensive research on this subject, found no difference in functional capacity between those who received oral supplements and those who received a standard diet at the end of six months (83). Subsequent studies with fewer patients found that adding oral supplementation to stroke patients with low weight or malnutrition may have a positive effect on long-term functional improvement, quality of life, and independent ability to carry out daily activities (7,10).

Total parenteral nutrition (TPN) in the stroke patient is used only when enteral nutrition is contraindicated. In these patients, 3-7 days are waited to start TPN. TPN can be started earlier in patients with severe malnutrition and who cannot be fed enterally. Complementary TPN can be given after 7-10 days in patients who are in the intensive care unit and cannot meet 60% of their calorie and protein targets with enteral nutrition (73). Contraindications to enteral nutrition are very limited (Table). Enteral nutrition in a hypotensive patient with low cardiac output may trigger intestinal ischemia if intestinal perfusion is impaired. However, this is a very rare case. Enteral nutrition should be delayed in patients who need continuous, increasing doses and multiple vasopressors, whose mean blood pressure is not increased above 60 mmHg, and whose lactate levels increase. Prone position, recent inactive peptic ulcer bleeding, hemodynamic shock corrected with vasoactive drugs, presence of aspiration and pneumonia, pancreatitis, intubation and mechanical ventilation are not contraindications of enteral feeding (68,73).

Table. Enteral nutrition contraindications.

Non-functional gastrointestinal tract
High-output gastrointestinal fistulas
Intestinal obstruction
Prolonged mechanical or paralytic ileus
Acute mesenteric ischemia
Active upper gastrointestinal bleeding
Abdominal compartment syndrome
Failure to provide enteral access
Intolerance of enteral nutrition (recurrent vomiting, gastric residue >500 ml/6 hours)
Statement of not accepting aggressive treatment in the last days of patient's life

TPN can be used as single component vials, combining amino acids, carbohydrates, and lipids, as two-in-one (amino acid+lipid) or all-in-one (amino acid+lipid+dextrose) preparations.In some hospitals, mixer systems (compounders) prepare the specific needs of each patient from various preparations. TPN application is a time-consuming and expensive procedure. Products above 700 mOsm can be delivered via a peripheral vein, whereas nutritional products above 900 mOsm require a central catheter (84-86).

Conclusion

Nutrition should be a basic part of treatment for patients who have had an acute ischemic or hemorrhagic stroke. Each patient's nutritional status should be questioned, examined, and, if necessary, laboratory studies should be performed. Silent dysphagia can be detected by bedside testing, FEES, and VFS. Nutritional therapy improves patients' functional recovery and significantly reduces complications and length of hospital stay. Muscle wasting caused by a stroke initiates early and is not uncommon. It is preventable with the right nutritional approach. Its treatment requires a high protein diet combined with active physiotherapy exercises.

In the patient who cannot swallow, enteral nutrition is started with special feeding tubes prepared for this task. When the basic calorie requirement is calculated as 20-25 kcal/kg/day and the protein requirement as 1.0-1.5 g/kg/day, the needs of the patients will be met to a large extent. Higher calorie and protein values can be targeted in patients who require intensive care, as well as those suffering from multiple diseases, infections, pressure sores, and muscle wasting. In the hospital, enteral tube feeding is administered as a continuous infusion, while at home; intermittent infusion or bolus applications are used. Enteral nutrition complications are very rare and easily corrected. However, PEG should be opened in patients in whom enteral nutrition will last longer than 4 weeks. TPN is rarely preferred in patients who cannot tolerate enteral nutrition or in the presence of a contraindicated situation. Proper nutritional support will improve stroke patients' recovery (Figure 2).

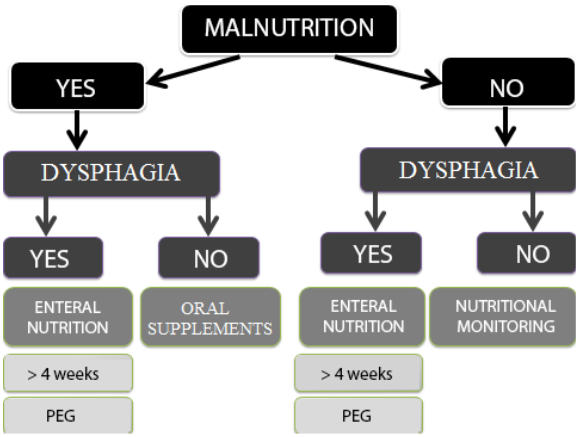

Figure 2. Nutritional algorithm in the acute stroke patient.

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Ethics

Ethics Committee Approval: There is no need to ethical approvel because of the article is a review.

Copyright Transfer Form: Copyright Transfer Form was signed by the author.

Authorship Contributions: Surgical and Medical Practices: LG. Concept: LG. Design: LG. Data Collection or Processing: LG. Analysis or Interpretation: LG. Literature Search: LG. Writing: LG.

Peer-review: Internally peer-reviewed.

Conflict of Interest: No conflict of interest was declared by the author.

Financial Disclosure: The author declared that this study received no financial support.