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REVIEW ARTICLE

Malnutrition in Stroke Patients: Risk Factors, Assessment, and Management

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Introduction

Stroke is the fourth leading cause of death in the USA [1] and one of the major causes of disability generating a massive economic burden [2]. Ischemic strokes account for 65–85% of stroke patients in the Western world, and the rest are hemorrhagic strokes which are more disabling [3]. Only 10–20% of hemorrhagic stroke patients will recover functional independence [4]. In order to improve neurological and cognitive functions of stroke patients, numerous rehabilitation interventions are implemented, including nutritional interventions, in attempt to overcome the metabolic consequences of stroke [5, 6].

Even though malnutrition in stroke patients is underrecognized and undertreated, its prevalence on admission is estimated to be around 20% [7, 8]. However, the prevalence of malnutrition after acute stroke varies widely ranging between 6.1 and 62% [9, 10]. This wide range has been attributed to different timing of assessment, patients' characteristics, and most importantly, nutrition assessment methods [10]. Malnutrition before and after acute stroke is responsible for extended hospital stay, poorer functional outcome, and increased mortality rates at 3–6 months after stroke [11–13]. The metabolic requirements and the resting energy expenditures (REEs) depend on the type of stroke with subarachnoid hemorrhage (SAH) requiring the most caloric intake when compared to ischemic strokes and intracerebral hemorrhage (ICH). As a result, the hasty identification of malnutrition using body mass index (BMI) or anthropometric measures or laboratory parameters after the acute event is fundamental to avoid poor outcomes [10, 14, 15]. The type of feeding depends on the swallowing status of the stroke patient; if dysphagia is present, enteral nutrition (EN) through nasogastric tube (NGT) or percutaneous endoscopic gastrostomy/jejunostomy (PEG/J) is a preferred intervention to oral feeding [14]. Although the exact day of initiation of feeding after an acute stroke event remains debatable, it is preferable to start feeding after the clinical stabilization of patients in order to reduce complication rates and improve overall recovery [16–18].

The aim of this review article is to discuss the risk factors for malnutrition in stroke patients and its assessment, the metabolic requirements for each type of stroke, and the importance of early feeding using the appropriate feeding method. We reviewed all English papers on risk factors, assessment, and management of malnutrition in stroke patients using Google Scholar and PubMed. Relevant studies are included in this review.

Risk Factors for Malnutrition in Stroke Patients

Elderly, women, preexisting malnutrition, poor family or nursing care, presence of malignancy, delayed rehabilitation, and history of severe alcoholism have been associated with

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Table 1 Risk factors for malnutrition in stroke patients

History	Physical examination
Elderly	Reduced level of consciousness
Women	Reduced mobility
Poor family or nursing care, preexisting malnutrition	Facial or arm weakness
PMH of hypertension, diabetes mellitus, severe alcoholism, malignancy, chronic diseases, and polypharmacy	Swallowing difficulties
Previous stroke	Xerostomia
Presence of dysphagia	Poor oral hygiene
Cognitive impairments, visual, language, and speech deficits	
Depression and antidepressants	

PMH Past medical history

malnutrition and dehydration (Table 1) [11, 19–21]. On admission, polypharmacy, eating difficulties, chronic diseases, functional disabilities, and high National Institutes of Health Stroke Scale (NIHSS) are associated with high risk of malnutrition in the elderly [11, 22]. Diabetes mellitus, hypertension, and stroke history increased the risk of malnutrition on admission by 58 and 71%, respectively [9, 14]. Interestingly, micronutrients deficiency such as antioxidants (vitamin A, C, E, zinc), B vitamins, vitamin D was also associated with an increased risk of cognitive impairment and stroke in the elderly [5, 7].

The presence of dysphagia is a major risk factor for developing malnutrition in stroke patients [11]. In the acute stage of stroke, dysphagia occurs in 30–50% of the patients and leads to a 12-fold increase in developing aspiration pneumonia and subsequent malnutrition [16, 23, 24]. Patients without dysphagia may still suffer from malnutrition when they are not well fed, particularly of protein [10, 20]. Moreover, the presence of cognitive impairments, visual, language, and speech deficits can hinder effective communication about food preference and satiety leading to malnutrition [14].

It is quite common that stroke patients suffer from depression and taking into consideration the side effects of the prescribed antidepressants such as xerostomia can help reduce feeding difficulties [14]. It is also possible that stroke patients may experience fatigue while eating leading to premature suspension of feeding [19].

Other important factors that should not be overlooked when assessing the risk of developing malnutrition in stroke patients include reduced level of consciousness, reduced mobility, facial or arm weakness, and poor oral hygiene [25]. Type and severity of stroke are considered major risk factors for malnutrition, especially that subarachnoid hemorrhage produce a hypercatabolic state in the body [26, 27]. In contrast, the location of the stroke, paresis of the dominant arm, education, and socioeconomic status were not significantly related to malnutrition [9, 14].

Assessment of the Nutritional Status in Stroke Patients

The assessment of the nutritional status in stroke patients is often challenging because of lack of a universally accepted definition of malnutrition and a gold standard for nutritional status assessment [28]. After stroke, elevation of plasma catecholamines, glucagon, cortisol, interleukin-6, interleukin-1RA, and acute phase proteins results in alteration of the metabolic requirements [29]. Use of barbiturates or induced hypothermia to lower intracranial pressure (ICP) also leads to a decrease in caloric demands [30].

Assessment of the nutritional status should always start by obtaining a thorough nutritional history that includes food intake, recent weight history, and the risk factors discussed in the previous section. When a stroke patient cannot provide a history because of limited cognitive function, history should be taken from family members or caregivers [14]. Table 2 summarizes the important elements of the assessment of the nutritional status in stroke patients.

Estimating BMI from simple measurement of weight and height in stroke patients is not always practical, especially in immobile patients [15]. Specialized equipment such as weighing scales or beds which accommodate wheelchairs can be used in assessing the weight of immobile patients [15]. Using more complex anthropometric measures such as triceps skin-fold thickness (TFT) and mid-arm muscle circumference (MAMC) is attainable and requires the use of a measure tape and trained personnel [14]. Unfortunately, BMI, TFT, and MAMC have low sensitivity and specificity [31]. Davalos and colleagues [32] assessed protein–energy status by TFT, MAMC, and serum albumin level; protein–energy malnutrition was defined as one abnormal finding of the three used parameters. However, authors noted that TFT has low sensitivity and intraobserver variability and therefore cannot be used dependably.

Table 2 Assessment of the nutritional status in stroke patients

Thorough history
BMI, TFT, MAMC
Laboratory parameters: lymphocyte count, serum albumin, pre-albumin, transferrin, CRP
Hydration status: hematocrit, BUN/creatinine ratio (>15:1)
? A, B, C, E vitamins
NST: MUST, MNA-LF, MNA-SF, SGA, NRS 2002

BMI Body mass index, *MAMC* Mid-Arm Muscle Circumference, *MNA-LF* Mini-Nutritional Assessment Long Form, *MNA-SF* Mini-Nutritional Assessment Short Form, *MUST* Malnutrition Universal Screening Tool, *NRS 2002* Nutritional Risk Screening 2002, *NST* Nutrition Screening Tool, *SGA* Subjective Global Assessment, *TFT* triceps skin-fold thickness

Laboratory parameters such as total lymphocyte count, serum protein, albumin, pre-albumin, and transferrin are readily available; however, their values can be affected by the presence of inflammation [12]. In the absence of infection and inflammation, serum albumin level can give a fair estimate of the nutritional status [14]. In acute settings, pre-albumin, transferrin, and C-reactive protein (CRP) are used to monitor changes in inflammation [14]. Moreover, CRP has been found to predict vasospasms and long-term outcome in SAH patients [33, 34].

High-plasma total homocysteine (tHcy) has been associated with cognitive impairment in patients with previous strokes or transient ischemic attacks [35]. Several studies assessed the effect of B vitamins on the outcome of stroke patients, and the results have been controversial. Hankey et al. [35] did not find an improved cognitive outcome when supplementing stroke patients with daily folic acid, vitamin B6, and vitamin B12, even though tHcy significantly decreased. On the other hand, serum levels of vitamin A, C, and E were found to be low in acute stroke patients [5]. Reduced levels of these vitamins were associated with functional decline, larger cerebral infarctions, and higher mortality rates most likely due to increased oxidative stress in the acute period [5].

Over the past years, many studies attempted to validate a nutrition screening tool (NST) in stroke patients. In 2003, the Malnutrition Universal Screening Tool (MUST), which includes an assessment of BMI, percentage of weight loss in the previous 3–6 months, and the effect of acute illness on dietary intake, was investigated for use in any patient [36] and was accepted for use in acute stroke patients [37, 38]. According to several studies [11, 39, 40], being at high risk of malnutrition, as assessed by MUST, is a significant independent predictor of mortality, length of hospitalization, and hospitalization costs at 6 months post-stroke. Other NSTs are readily available to use such as Nutritional Risk Screening 2002 (NRS 2002), Mini-Nutritional Assessment, and Subjective Global Assessment [41].

Clinicians must be aware of the limitations of each parameter and should keep in mind that using a combination of these parameters helps understand the nutritional status of each patient [42]. According to the current guidelines, screening stroke patients on admission and periodically thereafter is strongly encouraged [16, 37, 38].

Energy Expenditure in Stroke Patients

A main concern in stroke patients is the accurate calculation of REE in order to ensure adequate feeding and avoid a negative energy balance. A negative energy balance is particularly undesired in severe SAH patients (Hunt and Hess grades III, IV, and V) because of the dreaded infectious complications and poorer outcomes [26, 43]. A summary of the major studies measuring REE and its significance are found in Table 3.

Indirect calorimetry (IC), when available, is considered the gold standard for assessing REE and the caloric requirements for stroke patients. IC is a noninvasive, reproducible, and quantitative tool for measuring energy expenditure at the bedside [44]. IC estimates cellular energy metabolism by quantitatively measuring carbon dioxide output and oxygen consumption [45]. Many studies tried to reproduce the accuracy of IC by using relationships including the Harris–Benedict equation (HBE) and metabolism nomograms, but the results were equivocal. HBE estimates the basal energy expenditure (BEE) for men and women: for men, BEE (kcal/day) $= 66.5 + 13.75 \times W + 5.005 \times H - 6.775 \times A$; for women, BEE (kcal/day) $= 655.1 + 9.563 \times W + 1.85 \times H - 4.676 \times A$, where W is weight in kilograms, H is height in centimeters, and A is patient age in years [46].

Ischemic stroke patients have been found to have low–normal REE in most of the studies [29, 30, 47]. Leone and Pencharz [48] suggested that a lower REE in chronic stroke patients, who are dependent on tube feeding, is partially explained by paresis and hypothesized that decreased sympathetic nervous system activity and age-related organ atrophy are other possible explanations [49]. Bardutzky et al. [30] concluded that REE is low in stroke patients who are sedated and receiving mechanical ventilation because of deep sedation and added that HBE can be used

Table 3 Summary of studies reporting energy expenditures in stroke patients

Population	Resting energy expenditure (kcal/d]	Comments	References
Ischemic stroke on TF;	1300	Lower REE in stroke patients partially explained by organ atrophy in elderly, decreased sympathetic activity, and paresis	[48]
Healthy control	1514		
Sedated ischemic stroke on mechanical ventilation;	1603	No significant difference between the two groups ($p = 0.9$)	[30]
Sedated SICH on mechanical ventilation	1628	Sedation could lower energy expenditure	
Ischemic and hemorrhagic stroke	Day 21 1521	REE = 110% BEE	[29]
Healthy control	Day 90 1663	No significant difference in energy expenditure between ischemic and hemorrhagic strokes	
	Average REE 1665		
Acute ischemic stroke	First measurement (24–72 h) 1252	REE not elevated probably because of change in muscular tone and decreased physical activity	[47]
	Second measurement (10–14 days) 1219		
Critically ill SICH	Average REE 1878.9	REE = 117.5% BEE	[51]
		More energy requirement probably because of higher body temperature and slight elevation of serum cortisol levels	
Non-traumatic IH	1810	Both groups are hypermetabolic with no significant difference ($p = 0.25$)	[52]
sTBI	2238		
Mechanically ventilated TBI on TPN	473–2172	REE = 120% BEE for TBI; REE = 140% BEE for IH	[53]
Mechanically ventilated IH on TPN	552–1591	Deeper coma patients had lower REE	
SAH underwent surgical clipping (mild group: grades I and II HH)	Day 4 151% BEE = 1795	BEE mild = 1189	[43]
		BEE severe = 1270	
SAH underwent surgical clipping (severe group: grades III, IV, V)	Day 10 172% BEE = 2045	SAH managed by surgery provokes high stress response with increased catabolism	
	Day 4 146% BEE = 1854.2		
	Day 10 198% BEE = 2514		
Poor-grade SAH (HH)	REE = 1960	REE = 110–130% BEE	[26]
	Caloric intake = 980	Negative energy balance associated with infectious complications (UTI, bloodstream infections, and pneumonia) and medical complications (anemia, hyperglycemia, and fever)	
SAH	Day 14 REE = 1679	Underfeeding, inflammation, and high HH are associated with HAI	[27]
	Caloric intake = 846		

BEE basal energy expenditure, *HAI* hospital-acquired infections, *HH* Hunt and Hess, *IH* intracranial hemorrhage, *REE* resting energy expenditure, *SAH* subarachnoid hemorrhage, *SICH* spontaneous intracerebral hemorrhage, *sTBI* severe traumatic brain injury, *TF* Tube feeding, *TPN* Total parenteral nutrition, *UTI* urinary tract infections

accurately to estimate REE. To the contrary, Finestone et al. [29] proposed to use a caloric intake of 110% of estimated BEE in stroke patients. Gariballa et al. [50] found that acute ischemic stroke patients without swallowing difficulties are malnourished and emphasized the importance of using oral nutritional support (ONS) and enteral sip feeding in improving outcomes and decreasing mortality after 3 months.

The metabolic profile in patients with ICH and intraventricular hemorrhage (IVH) patients is not well established. Although some studies suggested that spontaneous ICH patients are not hypermetabolic [30], other studies concluded that ICH and IVH patients are hypermetabolic and require monitoring using IC to avoid undernutrition or over-nutrition [51–53]. SAH patients are—with possible association between cerebrovascular vasospasm and increased catabolic

state, and that HBE can estimate caloric needs with a 10–30% correction factor [26, 43, 52, 53]. In a larger-scale study on 229 SAH patients, malnutrition- and inflammation-mediated protein catabolism has been strongly associated with hospital-acquired infections. Pneumonia was the most common infection (33%) followed by urinary tract infections (21%) [27]. CRP-transthyretin ratio (TTR)—was implemented by Badjatia and colleagues to assess inflammation-mediated protein–energy malnutrition and 3-month outcome [27]. They reported a new relationship between elevated CRP: TTR (high CRP and low TTR), higher Hunt and Hess scores, and delayed cerebral ischemia, which correlated with a poorer long-term outcome after SAH [27]. High CRP levels in the acute setting of SAH were also linked to poor long-term outcomes [27, 33, 54].

Adverse Outcomes Related to Undernutrition in Stroke Patients

Undernutrition in ischemic and hemorrhagic stroke patients has been strongly associated with negative clinical outcomes. On the cellular level, protein and energy malnutrition on admission has been found to impair the recovery of hippocampal fibers from ischemic brain injury by altering the expression of trkB and GAP-43 proteins [55]. In acute ischemic stroke patients, malnutrition on admission has been strongly associated with poor 1-month and 3-months outcome [6, 32, 50, 56].

Malnourished ischemic and hemorrhagic stroke patients suffer from higher rates of pressure ulcers, respiratory, and urinary tract infections [26, 27, 57]. The Feed or Ordinary Diet (FOOD) trial collaboration [13] followed 2955 stroke patients out of which 279 were malnourished. Of those 21% developed pneumonia, 23% developed other infections, 4% pressure sores, and 4% gastrointestinal hemorrhage. All these complications were statistically significant compared to normal or overweight patients. Moreover, the malnourished population had high mortality (37%) compared to patients (21%) with normal nutritional status [13].

Malnutrition has been associated with an increase in dependency, duration of hospitalization and rehabilitation, and mortality rate [13, 58]. Moreover, around 40% of stroke patients, especially dysphagic patients, are at risk of becoming malnourished in rehabilitation centers [59, 60]. Weight loss in stroke patients is correlated with difficulties in regaining physical function in the long term [61]. Therefore, well-adjusted nutritional supplementation and maintenance of body weight are essential in these patients to accelerate their recovery [60–62].

Nutritional Support

After acute stroke, oxidative stress suppresses protein synthesis, resulting in impairment in brain recovery [5, 63]. Nutritional support has been proven to strongly enhance the physical and mental functioning of stroke patients [64] by preserving the muscle and fat masses, shortening hospitalization stay, and improving functional outcome [5].

Screening for Dysphagia

All stroke patients should have a clinical bedside screening for dysphagia by a trained personnel or speech-language pathologist (SLP) shortly after presentation to hospital (Table 4) [14]. It has been established that a formal and systematic dysphagia screening results in fewer rates of pneumonia and mortality [65].

Despite this evidence, Water-Swallowing-Test accuracy has been questioned and was found to have a sensitivity below 80% in detecting aspiration when compared to videofluoroscopic swallowing study (VFSS) and fiberoptic endoscopic evaluation (FEES) [66, 67]. On the other hand, the Multiple-Consistency-Test has been found to have 100% sensitivity and 50% specificity when compared to FEES [68]. This low specificity may result in more restricted diet and nasogastric tube insertions [68]. Finally, the Swallowing-Provocation-Test (SPT) has a sensitivity of 74.1% and a specificity of 100% in detecting aspiration when compared to FEES [69]. Hence, SPT cannot be used alone as a screening tool but more as a complementary tool [16].

All stroke patients failing a dysphagia screening test should be further assessed using VFSS or FEES by a trained personnel or SLP [16]. Furthermore, due to the insensitivity of bedside screening tests, all patients presenting with severe neurological deficits, facial palsy, aphasia, or marked dysarthria should be further evaluated even if their initial screening tests were normal [70, 71].

VFSS or modified barium swallow test, the gold standard test for assessment of dysphagia, requires the use of non-ionic, non-irritating contrast agents allowing dynamic visualization of oral, pharyngeal, and esophageal phases of swallowing [72]. VFSS allows defining the defected phase, grading of penetration of contrast, and aspiration using the rating scale of Rosenbek et al. [73]. On the other hand, FEES allows visualization of the pharynx using a nasolaryngoscope [74]. FEES can be used by bedside in severely uncooperative and handicapped patients, does not require radiation exposure, and allows detection of residues [75].

Early dysphagia screening and swallowing rehabilitation have been recognized in helping in regaining swallowing functions [76]. Moreover, spontaneous improvement in

Table 4 Screening tests for dysphagia in stroke patients

Test	Description	Comments
Water-Swallowing-Test (WST) [16]	According to SIGN guidelines, 50-ml WST is recommended for use Positive test: clinical signs of aspiration	Even if the patient passes this test and feeding is initiated, monitoring for coughing and chest infection is recommended
Multiple-Consistency-Test (aka. Gugging Swallowing Screen GUSS) [68]	Stepwise procedure for grading of dysphagia using separate non-fluid and fluid textures	Allows grading into one of four categories: severe, moderate, mild, or no dysphagia Each category has its special diet and further recommended strategies
Swallowing-Provocation-Test (SPT) [16]	Examination of the involuntary swallowing reflex by bolus injection of 0.4 ml of distilled water through a small nasal catheter into the oropharynx Normal test: time from water injection to reflex is below or equal to 3 s	It is a moderately sensitive and highly specific test

dysphagia is expected during the first 2 weeks after stroke, especially with supratentorial lesions [77]; however, stroke recurrences are common occurring in 5–10% of patients in the first weeks necessitating a routine dysphagia screening in acute stroke patients [16].

Feeding Strategies

According to the second part of FOOD trial, tube feeding, when indicated, has demonstrated a decrease in mortality in dysphagic stroke patients, especially if started within 7 days after the event [17, 18]. In mechanically ventilated patients, early tube feeding is beneficial and is preferred over parenteral nutrition (PN) [16]. Patients with severe dysphagia are at high risk of aspiration pneumonia and malnutrition; however, tube feeding does not prevent aspiration pneumonia nor does it increase its occurrence [17, 78]. Therefore, the indication of tube feeding in severe dysphagic patients is tailored to prevent malnutrition and improve prognosis [50, 79].

When prolonged severe dysphagia is expected (more than 7 days), tube feeding is preferred to be initiated within the first week and preferably within 72 h [16]. It is not advisable to start feeding on the first day of stroke because many patients' condition is still vague and some may require mechanical ventilation [16]. The FOOD trials [17, 18] showed that patients who received tube feeding within 7 days [either NGT or PEG] had a reduction in mortality rate by 5.8%, which was not significant ($p = 0.09$). Feeding tubes are inserted preferably in gastric position as there is no sufficient evidence or statistical significance to suggest an increase in aspiration pneumonia when compared to duodenal or jejunal feeding tubes [80–82]. Loeb and colleagues [83] and Kostadima et al. [84] did not find a significant difference between feeding into the small bowel versus nasogastric feeding regarding aspiration pneumonia, nutritional intake, and tube displacement. Therefore, the suggested use of post-pyloric tube feeding is considered on a case-to-case basis where there is suspicion of upper gastrointestinal dysfunction or delayed gastric emptying despite the use of prokinetic agents [82, 85].

When oral food intake is challenging during the acute phase of stroke, smallest size NGT (8-Fr, 10-Fr, 12-Fr) is the preferred method of enteral feeding [14]. Patients in the intensive care unit can have elevated ICP, which can delay gastric emptying and thus hindering a successful NGT feeding [14]. When enteral feeding is expected more than 28 days, PEG should be placed after 14–28 days in a stable clinical phase [16]. Likewise, mechanically ventilated patients should have a PEG placed at an earlier stage [16]. PEG feeding within 24 h in mechanically ventilated stroke patients produced better outcomes than NGT and decreased the incidence of ventilator-associated pneumonia [84]. Before insertion of PEG, severity of stroke, unfavorable prognosis, and ethical considerations should be intensively considered [86].

Dysphagia due to ischemic stroke resolved in 73–86% of the patients within 7–14 days and in a large proportion of patients within 3 months [87]; therefore, it is preferable to start with a less invasive feeding method than PEG. Norton et al. [88] compared NGT and PEG feeding and found that the PEG group had better nutritional status, shorter hospital stay, and less mortality after 6 weeks of interventions [88]. On the other hand, FOOD study did not find significant difference between NGT group and PEG group regarding outcomes after 6 months [17, 18]. Dislodgement of NGT can be managed by nasal loops, which have been demonstrated to be safe, effective, and well tolerated in stroke patients without a difference in outcome after 3 months [89].

In order to avoid aspiration pneumonia, continuous application of feeding in addition to frequent clinical examinations, monitoring of residual volumes, and elevation of the head of the bed are indicated in patients with

history of gastroesophageal reflux (GERD), concurrent signs of GERD, and jejunal or duodenal tubes [90]. If there are no risk factors, intermittent bolus application (6 times daily) for, respectively, 1 h is safe [16].

NGT feeding was not found to interfere with swallowing training and rehabilitation, and dysphagia therapy should be started as early as possible [91]. It is also preferable that conscious dysphagic stroke patients have oral feeding according to the severity and kind of dysphagia [92]. Oral hygiene is a main concern in dysphagic patients as the bacteria in the saliva is responsible for aspiration pneumonia [93] suggesting a strict oral hygiene in such patients [94].

PN is indicated when EN is not feasible or contraindicated [16]. Moreover, if caloric requirements or sufficient hydration cannot be met in well-nourished patients, supplemental PN is recommended [95].

ONS, when tolerated, is found to reduce morbidity and improve survival in malnourished elderly stroke patients [10]. Patients supplemented with ONS had a significantly improved caloric intake when compared to patients receiving hospital food only (1807 ± 318 kcal/d vs. 1084 ± 343 kcal/d, $p < 0.0001$), thus decreasing medical complications and decreasing mortality [50]. This was also supported by the FOOD trial [96], which showed a reduction in pressure sores and an improvement of outcome in malnourished stroke patients.

Nutritional Considerations

Enteral tube feeding formulas are well tolerated in stroke patients. The selection formula is usually 1–1.5 kcal/ml, polymeric, rich in protein, and sometimes supplemented with elemental nutrients [14]. Fiber-containing formulas are reserved for rehabilitation settings and are avoided in the acute settings when pressors are used. Medications should also be taken into consideration as some of them have nutritional impact (i.e., propofol gives 1.1 kcal/ml as fat, barbiturates cause a decrease in caloric requirements, narcotic agents cause constipation, and sorbitol causes diarrhea) [14].

Dysphagia diets were developed as National Dysphagia Diet (NDD) by the American Dietetic Association. NDD is divided into three levels with level 1 NDD being pureed (spoon-thick), level 2 NDD being mechanically altered (nectar-thick), and level 3 NDD being dysphagia advanced (honey-thick) [14].

Malnutrition in Stroke Patients and Current Guidelines

Current clinical guidelines and recommendations either partially discuss [97–99] or do not discuss at all nutritional support in stroke patients [100]. The recently published guidelines by the Society of Critical Care Medicine and American Society for Parenteral and Enteral Nutrition [100] did not discuss nutritional support in critically ill stroke patients. The guidelines did recommend to: (1) screen all critically ill patients for malnutrition, (2) start EN within 24–48 h or when hemodynamically stable, and (3) use post-pyloric feeding if high risk of aspiration. The European Society of Intensive Care Medicine supports the use of early EN to improve outcomes and mortality in all critically ill patients including ischemic and hemorrhagic stroke patients [98]. However, their guidelines do not openly discuss the indications of post-pyloric feeding and PEG tube insertions in stroke patients. The guidelines for the acute management of SAH [99], acute ischemic stroke [101], and spontaneous intracerebral hemorrhage [102] from American Heart Association/American Stroke Association do not address nutrition at all. In the guidelines of management of large hemispheric infarctions, Torbey and colleagues [97] recommended to screen for dysphagia after weaning from sedation and ventilation, use early NGT if dysphagia is present, and discuss PEG tube insertion with family if high NIHSS and dysphagia persist. The clinical guidelines for nutrition in stroke patients by Wirth and colleagues [16] reviewed the screening methods for dysphagia and aspiration pneumonia and different feeding strategies. However, the guidelines failed to include recommendations to assess energy requirements for the different types of strokes.

Conclusion and Recommendations

Stroke is a devastating event, leading to high morbidity and mortality. Malnutrition is prevalent in stroke patients and its early recognition significantly affects the outcomes. All stroke patients should be assessed thoroughly for malnutrition by checking their nutritional status and risk factors. IC helps in understanding the energy expenditure in stroke patients, and when it is not accessible, metabolism equations can fairly estimate the metabolic requirements in stroke patients. Dysphagia is quite common in stroke patients and has major impacts on short-term and long-term outcomes. If a stroke patient cannot tolerate oral feeding, tube feeding via NGT or PEG is beneficial and improves long-term outcomes. Despite these facts, more studies are required to clarify the optimal timing and method of nutrition. Moreover, large studies are required to assess the energy requirements in all stroke patients as previous studies are not conclusive. Future guidelines should identify a unified nutrition screening method, assess and evaluate reliable methods for energy needs, clarify timing of initiation of feeding, and specify when to use intragastric versus small bowel feeding. Quality of life is affected by

enteral feeding, and ethical issues should be given intensive considerations, especially in patients with poor outcomes.

Based on the current literature available, we can make the following recommendations for nutrition in stroke patients:

- Assess risk factors for malnutrition in stroke patients by performing a thorough history and physical examination. Nutritional screening using risk scores can also be implemented (NRS 2002, MUST, etc.).
- Assess for dysphagia and aspiration pneumonia.
- Standard protocols to assess energy requirements, initiation, and maintenance of nutrition should be made for the different types of strokes.
- EN is preferred over PN. EN should be initiated once the patients are hemodynamically stable.
- The preferred EN method is feeding via NGT. Post-pyloric feeding is decided on case-to-case basis to decrease risk of aspiration pneumonia.
- Additionally, to decrease risk of aspiration pneumonia, elevation of the head of the bed by 30–45° and use prokinetic medications are recommended.
- PEG tube feeding can be discussed with families when long-term tube feeding is provisioned.
- Multidisciplinary approach is strongly recommended.

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