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ORIGINAL ARTICLE

The role of nutritional management in preventing stroke and improving clinical outcomes

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Abstract

Background: Stroke is a leading cause of global morbidity, mortality, and substantial economic burden. Modifiable stroke risk factors, including hypertension, dyslipidemia, obesity, and diabetes mellitus, can be effectively managed through targeted nutritional interventions. Nutritional management also supports neurological recovery and contributes to improved clinical outcomes in stroke survivors. **Methods:** A literature review was conducted using PubMed, MEDLINE, and EMBASE databases from the last ten years, focusing on nutritional management for stroke prevention, clinical outcomes, dietary patterns, and nutrient supplementation during post-stroke recovery. **Results:** Dietary strategies, including Dietary Approaches to Stop Hypertension (DASH), high intake of dietary fiber, and omega-3 fatty acids, significantly reduce stroke risk by improving cardiovascular health and decreasing inflammation. Supplementation with micronutrients such as B vitamins supports neurological and vascular function. Post-stroke nutritional issues such as dysphagia, malnutrition, sarcopenia, and pressure injuries commonly impair patient recovery. Targeted nutritional intervention, particularly adequate protein and branched-chain amino acid (BCAA) supplementation, help preserve muscle mass, enhance functional recovery, and optimize rehabilitation outcomes. **Conclusions:** Nutritional management plays a crucial role in both preventing stroke and improving clinical outcomes following stroke. Implementing effective nutritional strategies, together with patient education on healthy dietary practices, is fundamental for reducing stroke risk, accelerating recovery, and enhancing quality of life.

Keywords: clinical outcomes, nutritional management, nutritional supplementation, rehabilitation, stroke

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Introduction

Stroke remains a significant global health issue with high morbidity and mortality rates. Approximately 15 million new cases occur worldwide annually, making stroke one of the leading causes of disability and death. There are two main types of stroke: ischemic stroke, accounting for around 80–85% of global cases, and hemorrhagic stroke, comprising approximately 15–20%. This condition creates

substantial health and economic burdens for both individuals and healthcare systems.[1]

In Indonesia, stroke prevalence is also concerning. According to the Riset Kesehatan Dasar (RISKESDAS) 2018, the prevalence of stroke reached 10.9%, affecting approximately 713,783 individuals. Men have a higher risk of stroke than women, and individuals older than 75 years show the highest prevalence. Stroke incidence is also higher in urban areas than rural regions. Additionally, stroke ranks third in healthcare expenditures, following cardiovascular disease and cancer, emphasizing the urgent need for effective prevention and management strategies.[2]

Effective stroke prevention and management are essential to reduce incidence rates and long-term consequences.[3] Clinical nutrition interventions have emerged as an important approach for managing major risk factors such as hypertension, diabetes mellitus, and dyslipidemia. Appropriate nutrition not only lowers the risk but also significantly enhances clinical outcomes during post-stroke recovery.[4,5] Siotto et al.,[6] demonstrated that stroke patients with poor nutritional status and sarcopenia experienced worse functional recovery compared to well-nourished patients, highlighting the importance of early sarcopenia detection. Additionally, Sato et al.,[7] found that adequate energy and protein intake during the first week post-stroke was associated with higher discharge-to-home rates and improved activities of daily living (ADL) scores. Irisawa and Mizushima further noted that higher muscle mass and better nutritional status were linked to superior motor function recovery within the initial four weeks of rehabilitation.[8] Optimal nutritional management can minimize complications, such as malnutrition, infections, and inflammation, thereby supporting cognitive and motor recovery and enhancing patient quality of life.[3–5]

This review aims to evaluate the role of nutritional management in stroke prevention and in improving clinical outcomes for stroke patients by reducing complication risks and facilitating recovery. Through this analysis, a better understanding of the benefits of nutritional management in stroke care can be achieved, providing a basis for developing more effective clinical strategies.

Methods

A literature search was conducted in MEDLINE, PubMed, and EMBASE databases covering the last ten years. Keywords used included “medical nutrition in stroke,” “stroke management,” “micronutrient supplementation in stroke,” “nutrient supplementation in stroke,” “protein supplementation in stroke,” “sarcopenia,” “malnutrition,” and “stroke-related complications.” Reference lists from relevant articles were also reviewed to include studies not indexed in the initial search. Only English-language literature relevant to the main topic was included.

Literature Review

The Role of Nutrition in Stroke Prevention

Stroke is closely associated with various metabolic conditions, such as hypertension, diabetes mellitus, obesity, and dyslipidemia, most of which can be modified through dietary interventions. Nutritional management plays a critical role in stroke prevention by addressing these modifiable risk factors. Unhealthy dietary patterns characterized by high consumption of saturated fats, added sugar, and sodium, as well as low intake of dietary fiber and micronutrients, significantly increase stroke risk. Therefore, implementing evidence-based nutritional management, including diets rich in fiber, polyunsaturated fatty acids (PUFA), and micronutrients such as B vitamins is considered a key strategy for reducing stroke risk. [9–14]

DASH Diet

The Dietary Approaches to Stop Hypertension (DASH) diet is a well-established dietary pattern that emphasizes the consumption of fruits, vegetables, whole grains, low-fat or fat-free dairy

products, lean protein sources such as legumes and fish, and healthy plant oils. It is designed to lower blood pressure and reduce cardiovascular risk. The DASH diet, when combined with sodium restriction, typically with a target intake between 1,500 and 2,300 mg/day depending on individual characteristics, has been shown to produce greater reductions in blood pressure than either intervention alone. This effect is largely attributed to reduced sodium-induced fluid retention, improved vascular tone, and enhanced endothelial function.[15]

A systematic meta-analysis conducted by Chiavaroli et al.[14] evaluated nine randomized controlled trials (RCTs) assessing the blood pressure-lowering effects of the DASH diet with sodium restriction. Results indicated that interventions involving direct meal provision were most effective, achieving reductions in systolic blood pressure (SBP) ranging from 7.7 mmHg to 2.4 mmHg and diastolic blood pressure (DBP) ranging from 8.3 mmHg to 0.1 mmHg. For instance, one of the included studies reported that structured meal provision led to an SBP reduction of 7.7 mmHg and DBP reduction of 8.3 mmHg.[14]

Another meta-analysis by Xun et al.[13], which included data from ten RCTs involving 4,667 participants, demonstrated that behavioral interventions aimed at reducing salt consumption significantly lowered blood pressure. The interventions included education on cooking methods designed to reduce salt usage, resulting in an average systolic blood pressure (SBP) reduction of −1.17 mmHg (95% CI: −1.86 to −0.49 mmHg) and diastolic blood pressure (DBP) reduction of −0.58 mmHg (95% CI: −1.07 to −0.08 mmHg). This study highlighted behavioral approaches, especially salt-reduction education in schools and families, as effective strategies for managing blood pressure. Building upon previous research emphasizing the importance of healthy dietary patterns such as the DASH diet, these findings support dietary modification and salt management as essential preventive strategies for hypertension and cardiovascular complications. The authors recommended combining behavioral interventions with additional strategies, such as reformulating food products to contain lower salt levels, to further enhance the effectiveness of salt reduction programs.[13]

Omega-3 Fatty Acid

Omega-3 fatty acids, particularly eicosapentaenoic acid (EPA), play a significant role in stroke prevention through multiple molecular pathways. Eicosapentaenoic acid is incorporated into membrane phospholipids, replacing arachidonic acid and modifying lipid raft composition, ion channel function, and membrane signaling. These alterations contribute to cardiac electrophysiological stability and reduce the risk of atrial arrhythmias associated with cardioembolic stroke. EPA also serves as a precursor for specialized pro-resolving mediators (SPMs), such as resolvins, which actively terminate vascular inflammation. In addition, EPA inhibits thromboxane A_2 production and platelet aggregation, providing antithrombotic effects important for ischemic stroke prevention.[16]

Furthermore, EPA has distinct membrane-stabilizing properties. It reduces membrane oxidative stress, prevents cholesterol crystal formation in atherosclerotic plaques, and preserves endothelial membrane integrity—mechanisms that lower inflammation and plaque vulnerability. These effects, which differ from those of docosahexaenoic acid (DHA), underline the unique vascular protective role of EPA.[17] Collectively, these findings demonstrate the multifaceted actions of EPA in modulating inflammation, thrombosis, and endothelial stability, all of which are crucial in the prevention of stroke.

O'Keefe et al.,[11] reported that individuals with the highest blood EPA levels experienced a 17% reduction in total stroke risk with hazard ratio (HR) 0.83 (95% CI: 0.76–0.91) and an 18% reduction in ischemic stroke risk. EPA, a marine-derived long-chain omega-3 polyunsaturated fatty acid, is primarily obtained from oily cold-water fish such as salmon, mackerel, sardines, and anchovies.[18] In line with these findings, Chen et al.[19] conducted a systematic review and meta-analysis demonstrating that high fish consumption was associated with a significantly

reduced risk of total stroke with relative risk (RR) 0.87 (95% CI: 0.79–0.96), with more pronounced effects observed for ischemic stroke. The greatest protective effect was observed among individuals consuming approximately 2−3 servings (100 g each) of fish per week.[19] This intake provides an average of 250–500 mg EPA and DHA per day, which is linked to a lower incidence of stroke, reinforcing their protective role in cerebrovascular health.[19,20]

Overall, evidence suggests that stroke prevention benefits are more consistently associated with regular fish consumption rather than isolated omega-3 supplementation, likely due to the synergistic interactions among various nutrients present in fish, such as vitamin D, B vitamins, and essential minerals.[19] Thus, increasing fish consumption as part of a balanced diet is recommended for stroke prevention, with the understanding that omega-3 supplementation alone might not yield comparable outcomes.

Dietary Fiber

In addition to omega-3 fatty acids, dietary fiber has been shown to play a significant role in stroke prevention through multiple mechanisms, including weight regulation, serum cholesterol reduction, and attenuation of vascular inflammation. Soluble fiber forms a gel-like substance in the stomach and small intestine, slowing nutrient absorption and delaying gastric emptying, which enhances satiety and facilitates weight control. In the colon, fermentation of fiber by gut microbiota produces short-chain fatty acids (SCFAs) such as acetate, propionate, and butyrate. These compounds inhibit hepatic cholesterol synthesis and subsequently lower serum cholesterol concentrations. SCFAs also stimulate the release of appetite-regulating hormones, including glucagon-like peptide-1 (GLP-1) and peptide YY, which contribute to improved insulin sensitivity and reduced systemic inflammation. Additionally, dietary fiber binds bile acids, reduces their reabsorption, and promotes the use of endogenous cholesterol for new bile acid synthesis, further contributing to decreased blood cholesterol levels.[21,22] The dietary fiber content of various food sources is summarized in **Table 1**.

Table 1. Dietary fiber content of various food items[23]

Source	Dietary fiber (g per 100 g of food item)		
	Total	Insoluble	Soluble
Oats	10.3	6.5	3.8
Rice (uncooked)	1.3	1.0	0.3
Rice (cooked)	0.7	0.7	0.0
Whole wheat	12.6	10.2	2.3
Almonds	11.2	10.1	1.1
Beets	7.8	5.4	2.4
Spinach (raw)	2.6	2.1	0.5
Radish	2.0	1.5	0.5
Tomato (raw)	1.2	0.8	0.4
Eggplant	6.6	5.3	1.3
Cucumber (peeled)	0.6	0.5	0.1
Cauliflower (raw)	1.8	1.1	0.7
Carrot (raw)	2.5	2.3	0.2
Broccoli (raw)	3.3	3.0	0.3
Apple (unpeeled)	2.0	1.8	0.2
Kiwi	3.4	2.6	0.8
Mango	1.8	1.1	0.7
Grapes	1.2	0.7	0.5
Orange	1.8	0.7	1.1
Strawberry	2.2	1.3	0.9
Banana	1.7	1.2	0.5
Pear	3.0	2.0	1.0

g: gram

A systematic review by Alahmari et al.,[22] found that individuals who consumed high-fiber diets, approximately 25 to 38 g per day or 14 g per 1,000 kilocalories, had a relative risk reduction for stroke ranging from 0.83 to 0.93. Greater protective effects were observed with fiber derived from fruits and cereals. Furthermore, Reynolds et al.[24] demonstrated in a meta-analysis of adults with established cardiovascular disease that higher fiber intake, with a median of 20 g/day (IQR 18–22 g/day), was associated with a 25%

reduction in all-cause mortality (RR 0.75; 95% CI: 0.58–0.97), equivalent to 60 fewer deaths per 1,000 individuals.[24]

These results support the recommendation to increase dietary fiber intake as part of a comprehensive nutritional strategy for stroke prevention, particularly ischemic stroke. Further research is needed to elucidate the effects of different fiber types and food sources on stroke risk reduction, which may contribute to more refined dietary guidelines for populations at elevated risk.

B Vitamins

Elevated plasma homocysteine levels are significant contributors to stroke risk, primarily via mechanisms involving vascular injury and chronic inflammation.[25] Mechanistically, hyperhomocysteinaemia impairs endothelial function by reducing nitric oxide (NO) bioavailability. This occurs through the uncoupling of endothelial nitric oxide synthase (eNOS) and increased levels of asymmetric dimethylarginine (ADMA), an endogenous inhibitor of NOS. These alterations promote vascular smooth muscle proliferation, oxidative stress, and disruption of the blood–brain barrier (BBB), contributing to the development of cerebral small vessel disease and lacunar infarction. Hyperhomocysteinaemia also activates proinflammatory signaling and endothelial apoptosis, while genetic variants, particularly the methylenetetrahydrofolate reductase (MTHFR) C677T polymorphism, further exacerbate homocysteine accumulation.[25,26]

The metabolism of homocysteine depends on several B vitamins, including folic acid, vitamin B6 (pyridoxine), and vitamin B12 (cobalamin)—each playing a distinct and complementary role in homocysteine regulation and stroke prevention. Folic acid acts as a methyl donor in the remethylation of homocysteine to methionine, vitamin B12 is an essential cofactor in this reaction, while vitamin B6 is required for the transsulfuration pathway, converting homocysteine to cysteine. Deficiencies in any of these vitamins may result in elevated homocysteine levels and increased vascular risk.[27,28]

Folic Acid

Recent meta-analyses confirm that higher dietary folic acid intake, particularly between 400–600 μg per day, is associated with a reduced risk of incident stroke, with optimal benefit at intakes of approximately 611 μg/day. Randomized controlled trials also demonstrate that folic acid supplementation at doses ranging from 0.4 to 0.8 mg/day is effective for primary stroke prevention in populations without mandatory food fortification. The protective effect of folic acid is primarily attributed to its ability to lower plasma homocysteine, thereby reducing endothelial dysfunction and vascular risk. Thus, folic acid is supported as a safe, cost-effective public health strategy for stroke prevention, especially in regions without food fortification.[28]

Vitamin B6

Vitamin B6 is integral to homocysteine metabolism via the transsulfuration pathway, acting as a cofactor for cystathionine β-synthase. However, evidence from RCTs consistently indicates that vitamin B6 supplementation alone does not significantly reduce the risk of stroke, myocardial infarction, or all-cause mortality, either for primary or secondary prevention. Large studies such as NORVIT have not demonstrated significant benefits for vitamin B6 monotherapy, and meta-analyses also report a lack of protective effect. Consequently, current evidence does not support vitamin B6 supplementation alone for primary stroke prevention in the general population.[29]

Vitamin B12

Vitamin B12 is a key cofactor in the remethylation of homocysteine to methionine and is essential for vascular health. Although clinical trials and meta-analyses have used supplementation doses of 0.4 to 1 mg/day for homocysteine lowering, RCTs show that vitamin B12 supplementation alone does not significantly reduce the incidence of stroke, myocardial infarction, or all-cause mortality, regardless of

whether it is used for primary or secondary prevention. Most observed benefit for stroke prevention has been limited to regimens combining vitamin B12 with folic acid and/or vitamin B6. Therefore, vitamin B12 monotherapy is not currently recommended for stroke prevention in the general population.[29,30]

The clinical significance of these mechanisms is supported by epidemiological and interventional data. A meta-analysis of 16 studies involving over 10,000 individuals found that elevated plasma homocysteine levels were associated with a markedly increased risk of ischemic stroke with Odds Ratio (OR) 2.51 (95% CI: 1.94–3.26), particularly in those with concentrations ≥15 μmol/L.[26] Intervention trials demonstrate that daily supplementation with folic acid 0.5 mg, vitamin B6 10 mg, and vitamin B12 0.4 mg for six weeks can reduce homocysteine concentrations by 25–30%.[27] In the HOPE-2 trial, a regimen of folic acid 2.5 mg, vitamin B6 50 mg, and vitamin B12 1 mg daily reduced stroke risk by 25% (RR 0.75; 95% CI 0.59–0.97) in populations without folic acid fortification.[27] Furthermore, a recent dose–response meta-analysis identified the most effective supplementation ranges as folic acid 0.8–2.5 mg/day, vitamin B6 20–50 mg/day, and vitamin B12 0.5–1 mg/day, associated with a 13–25% reduction in stroke risk.[30] Notably, doses below these ranges were less effective, and higher doses did not confer additional benefit.

Importantly, no clinical trials have shown that vitamin B6 or vitamin B12 supplementation alone—outside of combination with folic acid—significantly reduces stroke risk. The available evidence suggests that the stroke prevention benefits of B vitamins are most pronounced when used as part of a combined regimen, particularly in individuals with elevated homocysteine and low dietary folate intake.

Antioxidant

Antioxidants play a crucial role in stroke prevention by counteracting oxidative stress caused by reactive oxygen species (ROS), which are major contributors to vascular injury and the pathogenesis of stroke.[12] Based on this mechanistic pathway, numerous studies have explored the effects of various antioxidant vitamins, such as beta-carotene, vitamin C, and vitamin E, on stroke prevention. However, not all of these vitamins have demonstrated clear or consistent benefits in reducing stroke risk.

Vitamin E is a major antioxidant that has been extensively evaluated for its role in stroke prevention, primarily due to its ability to counteract oxidative stress caused by ROS—a central factor in vascular injury and stroke pathogenesis.[31,32] Two recent meta-analyses have looked at whether vitamin E can help prevent stroke. Loh et al.[31] analyzed 18 RCTs involving over 148,000 participants, with vitamin E dosages ranging from 50 IU to 800 IU per day, most commonly 400–600 IU daily. Their analysis found that vitamin E, at both low (<300 IU) and high (≥300 IU) doses, did not significantly reduce the overall risk of stroke (RR 0.98; 95% CI 0.93–1.04). There was a modest reduction in ischemic stroke risk (RR 0.92; 95% CI 0.86–0.98), but this was offset by a slight increase in hemorrhagic stroke risk (RR 1.17; 95% CI 1.02–1.34).[31]

Similarly, Maggio et al.[32] reviewed 16 RCTs with vitamin E supplementation doses ranging from 330 IU to 800 IU per day. Their meta-analysis confirmed that vitamin E alone, regardless of the dose, did not significantly reduce the risk of stroke. However, when vitamin E was combined with other antioxidants, such as vitamin C (100–1,000 mg/day), beta-carotene (20–50 mg/day), or selenium (up to 100 mg/day), there was a small but statistically significant reduction in ischemic stroke risk (RR 0.91; 95% CI 0.84–0.99). This benefit, however, was offset by a significant increase in the risk of hemorrhagic stroke (RR 1.22; 95% CI 1.00–1.48)[32]

Given the lack of benefit for total or fatal stroke at all tested doses, and the potential for increased risk of bleeding, routine supplementation with vitamin E, either alone or in combination with other antioxidants, cannot be recommended for stroke prevention. Current evidence supports a cautious approach, and further research is needed to determine if there are

specific patient subgroups who may derive net benefit from antioxidant therapy.

The Role of Nutrition in Improving Clinical Outcomes in Stroke Patients

Malnutrition represents a major clinical challenge in stroke patients and has a significant impact on clinical outcomes. The prevalence of malnutrition among hospitalized stroke patients is estimated at 26% upon admission and increases to 56–62% during hospital stays exceeding three weeks. This condition is often exacerbated by dysphagia which affects up to 60% of stroke patients, particularly during the acute phase. Dysphagia often causes inadequate oral intake and dehydration, thereby significantly increasing the risk of serious complications such as aspiration pneumonia. Additionally, social and psychological factors, including depression, anxiety, and social isolation, further contribute to the deterioration of nutritional status.[33]

Four major nutrition-related complications frequently observed in stroke patients include dysphagia, malnutrition, sarcopenia, and pressure injuries. Malnutrition, characterized by deficiencies in energy, protein, and micronutrients, is associated with increased morbidity, susceptibility to infections, prolonged hospital stays, and higher mortality rates. It also negatively affects neurological recovery and functional outcomes. Dysphagia, if inadequately managed, impairs oral intake, increases the risk of dehydration and aspiration pneumonia, leads to significant weight loss, and ultimately compromises quality of life and increases care burden.[33]

Sarcopenia, defined as the loss of skeletal muscle mass and strength due to prolonged immobility, further impedes rehabilitation, reduces functional capacity, increases the risk of falls, and extends dependency on care. Meanwhile, pressure injuries are often worsened by malnutrition, which delays wound healing, increases the risk of infection, and compromises skin integrity. Adequate nutritional intake—particularly of protein, arginine, vitamin C, and zinc—is essential for supporting wound healing and preventing the development of pressure injuries. Together, these complications underscore the critical importance of timely and sustained nutritional management in the overall management and recovery of stroke patients.[33]

Appropriate nutritional management is essential at all stages of stroke recovery to manage complications and support rehabilitation (**Figure 1**). In the acute phase (≤7 days), nutritional assessment should be conducted within the first 48 hours to prevent rapid nutritional deterioration. Enteral nutrition is recommended for patients with severe dysphagia to ensure adequate energy and protein intake. In the early subacute phase (7 days–3 months), oral intake can be resumed with texture-modified diets based on individual needs, supported by high-protein supplementation enriched with leucine and vitamin D to prevent sarcopenia. During the late subacute (3–6 months) and chronic phases (>6 months), nutritional management focuses on maintaining nutritional status, improving functional capacity, and preventing long-term complications such as pressure injuries.[33]

The role of nutrition in stroke rehabilitation lies in its capacity to support functional recovery and mitigate post-stroke complications. Stroke often leads to long-term neurological deficits, which elevate the risk of malnutrition, sarcopenia, anemia, diabetes mellitus, and osteoporosis. Appropriate nutritional management can improve nutritional status and facilitate the rehabilitation process, with the goal of accelerating recovery and preventing secondary complications. Several studies have demonstrated that targeted nutritional interventions significantly enhance post-stroke recovery. For instance, amino acid supplementation helps prevent post-stroke muscle protein hypercatabolism, increases muscle mass, and improves functional performance during early recovery. Supplementation with micronutrients such as vitamin D and vitamin B12 has been investigated in stroke patients, with some studies reporting improved outcomes. However, the evidence remains variable across different patient populations and study designs.[33,34]

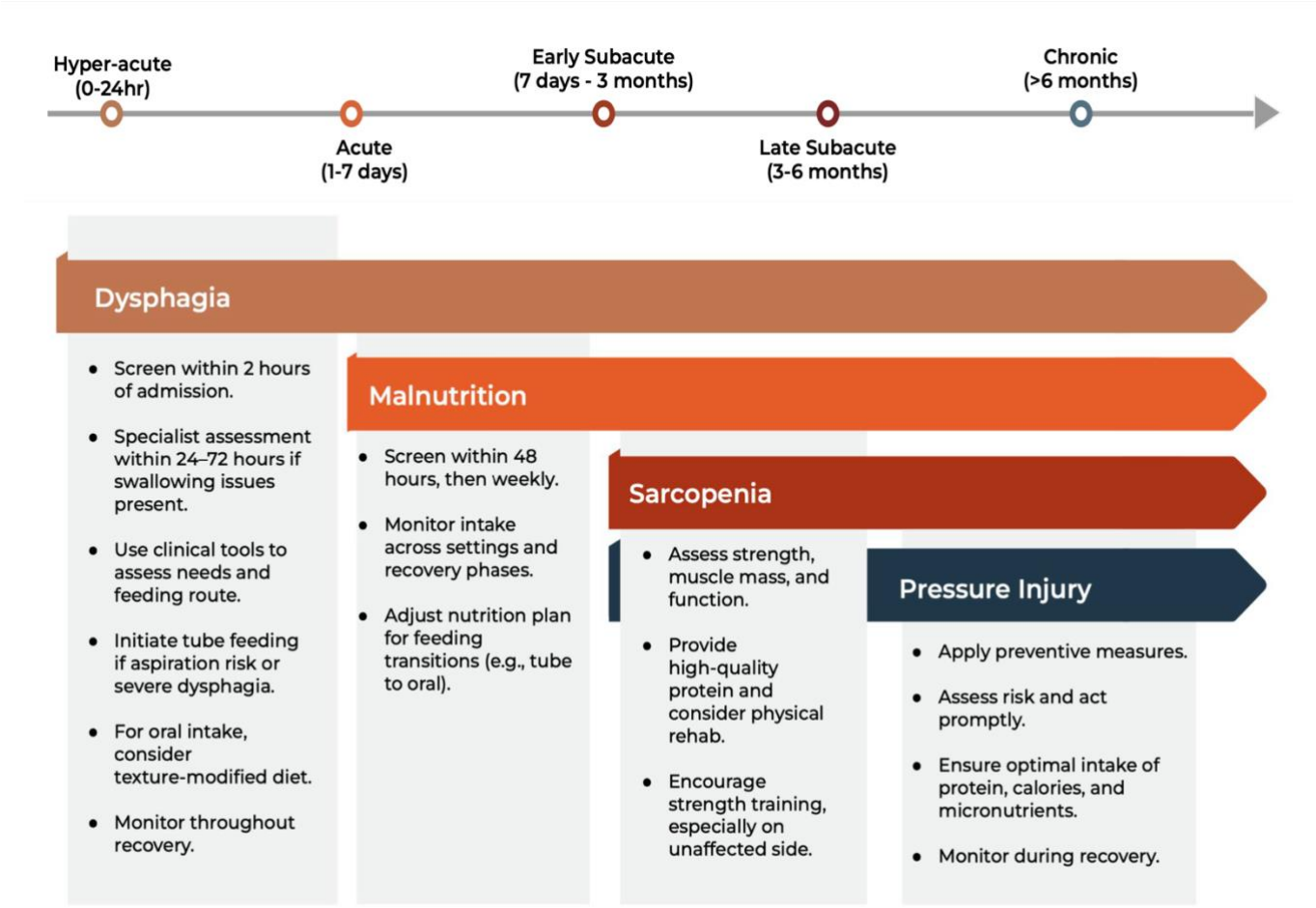

Figure 1. The role of nutrition management across stroke phase[33]

Protein Supplementation

Protein is essential for post-stroke recovery, as it supports the regeneration of neural and muscular tissues that are frequently damaged by stroke. Adequate protein intake promotes tissue repair and functional improvement. Branched-chain amino acids (BCAAs) play a central role in neural repair, modulation of inflammation, and the restoration of motor function. These amino acids serve as precursors for glutamate and stimulate muscle protein synthesis via activation of the mechanistic target of rapamycin (mTOR) pathway, thereby facilitating both neural and muscular recovery.[33–36]

Recent evidence from multiple interventional studies and RCTs, as reviewed by Chen et al.,[37] shows that supplementing 20–33 g of protein per day for 4 to 8 weeks improves neurological recovery, motor function, and physical performance, especially among post-stroke patients with poor nutritional status or sarcopenia. These studies, which included both subacute and chronic stroke populations, reported benefits such as greater reductions in National Institutes of Health Stroke Scale (NIHSS) scores, higher Functional Independence Measure (FIM) gains, improved balance, and increased discharge rates. However, effects on activities of daily living and cognitive outcomes were inconsistent, underscoring the need for personalized nutritional assessment and further high-quality research to determine long-term clinical efficacy.[37]

Branched-Chain Amino Acids

Branched-chain amino acids (BCAAs)—including leucine, isoleucine, and valine—are important for skeletal muscle recovery and may prevent muscle atrophy, a common complication

after stroke. Leucine, in particular, activates the mTOR pathway to stimulate muscle protein synthesis and inhibit degradation.[36] Supplementation with BCAAs can help preserve muscle mass and improve functional outcomes during rehabilitation. In a study by Park et al.,[35] post-stroke patients with sarcopenia who received 30 g of protein daily, including 6 g of BCAAs, for one month demonstrated significant improvements in both skeletal muscle index and handgrip strength compared to controls. Functional measures such as the Korean Mini-Mental Status Examination, Berg Balance Scale, and Functional Ambulation Categories also improved to a greater extent in the BCAA group, alongside a 35% lower incidence of infection. These results suggest that BCAA supplementation, combined with intensive rehabilitation, can help prevent muscle wasting and support recovery after stroke.[35]

Ko and Shin reviewed several RCTs and clinical studies involving adult and elderly post-stroke patients, particularly those who are malnourished or at risk of sarcopenia. Interventions typically included supplementation with 20–23 g of protein or whey protein per day, or 3–3.5 g per day of leucine-enriched essential amino acids, given for 4 to 12 weeks and resulted in significant improvements in muscle mass, handgrip strength, and functional outcomes such as Functional Independence Measure (FIM) and skeletal muscle index, especially in undernourished individuals.[38] These findings indicate that targeted BCAA supplementation may be particularly beneficial for post-stroke patients with sarcopenia or poor nutritional status to enhance rehabilitation outcomes.

Omega-3 Fatty Acid

A systematic review and meta-analysis by Alvarez Campano et al.[39] examined 29 RCTs involving 3999 patients with stroke or transient ischemic attack (TIA), and found that supplementation with marine-derived omega-3 fatty acids (400–3300 mg/day, for 3 months to over 1 year) did not provide significant improvements in functional capacity, dependency, vascular mortality, recurrent stroke, quality of life, or mood compared to placebo.[39]

Consistent with these findings, a randomized controlled trial by Rist et al.[40] in 197 stroke survivors who received omega-3 supplementation (1 g/day of fish oil, containing 460 mg eicosapentaenoic acid [EPA] and 380 mg docosahexaenoic acid [DHA]) prior to stroke, with a median 1.4 years follow-up, showed no significant reduction in risk of post-stroke functional limitations or physical disability. The odds ratios for functional limitation and physical disability on the Nagi, Rosow-Breslau, and Katz ADL scales were 0.55 (95% CI 0.28 to 1.09), 0.56 (0.31 to 1.02), and 0.32 (0.08 to 1.24), respectively; none were statistically significant.[40] Taken together, current evidence does not support the clinical benefit of omega-3 fatty acid supplementation for improving post-stroke functional outcomes or reducing disability and mortality.

Vitamin B12 (Cobalamin)

Recent studies have explored the potential benefits of vitamin B12 supplementation in post-stroke patients. Yuan et al.,[42] investigated the effects of vitamin B12 supplementation in patients with ischemic stroke and H-type hypertension—a subtype of hypertension associated with elevated plasma homocysteine levels. Mecobalamin 500 μg three times daily led to significant reductions in homocysteine, hs-CRP, and carotid plaque thickness at 3 and 6 months (all $p < 0.05$). Functional recovery, assessed by the NIHSS, was also significantly greater in the mecobalamin group than in controls ($p < 0.05$).[42] These findings support the role of mecobalamin in improving neurological recovery and vascular health. When combined with evidence on the benefits of BCAA supplementation, the results reinforce the importance of nutritional management in optimizing post-stroke recovery and patient outcomes.

Vitamin D

Vitamin D supplementation in post-stroke patients has been studied using a wide range of

dosing regimens, including single intramuscular injections of 300,000–600,000 IU, daily oral doses of 600–2,000 IU, weekly doses of 50,000 IU, and monthly doses of 60,000 IU.[41] Several studies have reported statistically significant improvements in motor function (Brunnstrom Recovery Stage), balance (Berg Balance Scale), lower extremity function, mobility (Functional Ambulation Classification), and reductions in stroke impairment scores (Scandinavian Stroke Scale, NIH Stroke Scale). However, the effects on activities of daily living (Barthel Index, Functional Independence Measure) were inconsistent, and the clinical significance of these improvements remains uncertain due to small sample sizes, heterogeneity of study design, and the lack of clear evidence for minimal clinically important differences.[41] Therefore, despite its safety profile, current evidence does not support routine vitamin D supplementation specifically for improving clinical outcomes in stroke rehabilitation, and further large, high-quality trials are needed to determine its true benefit and optimal dosing strategy.

The Role of Nutritional Education in Stroke Management

Nutritional education is a critical component of comprehensive stroke care, particularly in promoting long-term dietary adherence and reducing the risk of recurrent cerebrovascular events. According to the latest American Heart Association (AHA) guidelines, effective dietary strategies should prioritize reducing sodium and saturated fat intake, emphasize the consumption of whole grains, fruits, vegetables, legumes, nuts, and non-tropical vegetable oils, and recommend lean protein sources such as fish and skinless poultry. Limiting sodium to less than 1,500–2,300 mg/day and saturated fat to less than 6% of total energy intake is essential for optimal blood pressure and lipid management.[1] Patient counselling should focus on practical approaches to selecting low-sodium foods, replacing saturated and trans fats with unsaturated fats, and increasing plant-based food consumption. Diets rich in dietary fiber, and micronutrients derived from plant-based foods support vascular endothelial function and overall cerebrovascular health.[43,44]

Family engagement is equally important in reinforcing dietary behavior change. Involving caregivers in nutritional education empowers them to support patients in adhering to therapeutic diets, thereby enhancing compliance, and promoting sustainable behavioral change. Such a family-centered approach has been associated with improved health literacy, self-efficacy, and preparedness for post-stroke care, contributing to secondary prevention and reducing the risk of recurrent stroke.

Conclusion

Nutritional intervention is fundamental to both the prevention and management of stroke. Diets that are rich in dietary fiber, unsaturated fats, and essential micronutrients such as folate, vitamin B6, and vitamin B12 help regulate major vascular risk factors including hypertension, abnormal blood lipids and excess body weight. Omega-3 fatty acids also offer anti-inflammatory and vascular-protective effects, thereby reducing the risk of blood clots and cerebrovascular disease.

After a stroke, nutritional support becomes critical. Adequate intake of calories, protein, and micronutrients helps the brain adapt, reduces systemic inflammation, and supports physical recovery. Common stroke complications such as difficulty swallowing, muscle wasting, and pressure injuries can delay rehabilitation and should be addressed with targeted nutrition interventions. Supplementation with branched-chain amino acids and vitamin B12 (mecobalamin), especially when combined with structured rehabilitation, has been shown to help preserve muscle mass and improve physical function.

In the long term, nutritional management should be integrated into every stage of stroke care. Ongoing nutrition education and the implementation of personalized, evidence-based nutrition plans can optimize recovery, support

daily functioning, and improve overall quality of life for stroke survivors.

Conflict of interest

The authors declare no competing interests.

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