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Nursing Care Plans for the Dehydration and Malnutrition-An Updated Review and Future Trends for Controlling Gastrointestinal Disorders.

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Abstract:

Background: Dehydration and malnutrition are significant contributors to adverse health outcomes, particularly in patients with gastrointestinal disorders. Malnutrition, defined by the European Society of Clinical Nutrition and Metabolism (ESPEN), encompasses nutrient deficiencies that impair physical and mental functions, exacerbated by gastrointestinal diseases that hinder nutrient absorption and elevate metabolic demands.

Aim: This updated review explores nursing care strategies for managing dehydration and malnutrition, focusing on gastrointestinal disorders, and highlights future trends for improving outcomes.

Methods: The review synthesizes data from historical and contemporary studies on malnutrition and dehydration, including research on starvation, disease-induced undernutrition, and micronutrient

deficiencies in populations with intestinal failure. Key areas include clinical impacts, survival thresholds, and the physiological implications of undernutrition.

Results: Findings reveal that undernutrition results in functional impairments, micronutrient deficiencies, and increased morbidity. Gastrointestinal disorders exacerbate malnutrition through malabsorption and inflammation, with specific deficiencies in vitamins A, D, E, and minerals like iron, zinc, and magnesium. Historical starvation studies demonstrate a survival threshold of 35% body weight loss, beyond which mortality sharply increases. Effective nursing care requires individualized plans addressing nutritional assessments, hydration status, and micronutrient supplementation.

Conclusion: The review underscores the importance of integrating advanced nutritional support, patient-centered care plans, and early interventions to manage dehydration and malnutrition. Emerging trends, such as leveraging technological tools for monitoring nutritional status and individualized supplementation, promise to enhance outcomes in patients with gastrointestinal disorders.

Keywords: dehydration, malnutrition, gastrointestinal disorders, nursing care plans, nutritional support, micronutrient deficiencies

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Introduction:

The European Society of Enteral and Parenteral Nutrition (ESPEN), also referred to as the European Society of Clinical Nutrition and Metabolism, has characterized undernutrition and malnutrition as equivalent concepts, denoting "a condition arising from insufficient intake or absorption of nutrients, resulting in changes in body composition (reduced fat-free mass) and body cell mass, which subsequently hinder physical and mental functions, along with clinical outcomes in disease" [1]. Gastrointestinal diseases lead to undernutrition by diminishing appetite, oral intake, digestion, and/or absorption, while simultaneously elevating metabolic demands. Extensive research in the twentieth century on both normal and obese individuals undergoing prolonged starvation, populations suffering from famine, and patients with disease-related undernutrition has deepened our comprehension of the relationship between weight loss, particularly the reduction of body cell mass, and the ensuing deterioration in mental and physical function. The relationship between structural alterations and functional decline is not always linear [6], as certain functions may begin to decrease early in the starvation process, whereas others remain comparatively preserved until later stages. In specific cases, a threshold may be present, beyond which function is predominantly maintained, but deteriorates swiftly beneath it [7]. The rate of tissue depletion and the subject's initial nutritional condition are critical elements in this process [8]. The consequences of complete starving may vary from those of partial starvation, and prolonged low intake may result in adaptive mechanisms, as evidenced in lean subsistence farmers in Asia. The interaction of energy and protein deficits, the emergence of singular or multiple mineral, trace element, and vitamin deficiencies, and the existence of underlying disorders can all affect the consequences of partial starvation. This review largely examines the ramifications of disease-induced undernutrition linked to intestine failure, substantiated by pertinent findings from starvation research. A substantial amount of data regarding undernutrition is derived from pediatric reports in poor nations and studies involving adolescents with anorexia nervosa. The chapter offers a summary

of the ramifications of dehydration, a condition familiar to doctors, although for which published information is scarce.

Survival:

In the early twentieth century, hunger strikes—a protest tactic that dates back to the Roman era—attracted a lot of public attention thanks to the British suffragettes. Famous for his fasting, Mahatma Gandhi fasted at least 14 times, never going over 21 days. The 30 Irish Republican Army (IRA) hunger strikers in Northern Ireland were a striking example of complete deprivation in previously healthy people [8]. They lost 38% of their body weight over the course of 60–70 days, and one-third of them died. According to this scenario, normally nourished humans may withstand a loss of up to 35% of their body weight, or roughly 60 days of total hunger, if there is no illness present. After this, mortality rises dramatically. This threshold is greatly lowered in patients who have previously lost weight or who have underlying medical disorders like sepsis or cancer. The cumulative effects of disease and famine are shown by the fact that one hunger striker who was shot only lived for forty-five days. Before undergoing surgery for peptic ulcers, people who lost more than 20% of their body weight had a 33% mortality rate, while those who lost less than 20% had a 4% mortality rate, as shown by Studley (1936) [9]. Due to their growth and developmental requirements, high metabolic rate in relation to body size, and higher surface-to-volume ratio during infancy, children are especially vulnerable. A newborn who is totally malnourished might not live for more than a few days. Pelletier et al.'s study on Asian children who were undernourished found that their chances of dying from undernutrition and disease increased by more than 7% for every percentage point of weight loss [10]. Additionally, when a child's weight dropped below 80% of what was expected for their age, the risk of morbidity rose tenfold.

Reports of obese adults surviving 130–382 days of hunger [3] demonstrate the protective impact of obesity. Larger energy stores and reduced nitrogen loss in relation to lean mass both contribute to this protection, suggesting a metabolic control mechanism. It is yet unknown, though, if obesity provides a comparable level of protection when there is a coexisting illness. Our knowledge of famine survival is further enhanced by the comprehensive research done in 1942 by Jewish doctors in the Warsaw ghetto [4]. Many residents had lost 20–50% of their pre-war body weight and had been living on fewer than 800 kcal per day for months. As an example, a woman who was 1.52 meters tall and weighed 24 kilograms had a body mass index (BMI) of 10.3 kg/m^2. The majority passed away within three weeks, and the majority were afflicted by concurrent infections. Some, meanwhile, displayed differences in their capacity to withstand famine. According to a review, the lower BMI limit for survival is roughly 12 kg/m^2 for males and 10 kg/m^2 for women [11]. The ramifications of these discoveries for clinical practice are significant. For nutritional support, the MEED guidelines advise admitting patients with a BMI of less than 13 kg/m^2 [12]. Referrals for nutritional support are frequently made for patients who have lost 20–35% of their body weight because their survival and function are at risk. One may also contend that parenteral nourishment is just as life-saving as dialysis in cases of renal failure or ventilation in cases of respiratory failure when it is necessary for 60 days or longer due to persistent gastrointestinal dysfunction. The time frame, not the principle, is what separates these scenarios.

Undernutrition in Intestinal Failure

In the context of intestinal failure (IF), undernutrition is categorized as disease-related malnutrition. This condition can result from a variety of factors, such as malabsorption, significant gastrointestinal losses, repeated surgeries, immobilization, sepsis, or inflammation, in addition to decreased food intake or appetite. All of these elements work together to cause body mass loss. This kind of undernutrition is classified as "disease-related malnutrition with inflammation" by the European Society for Clinical Nutrition and Metabolism (ESPEN) [1]. Cachexia is frequently linked to a particular subtype, known as "chronic disease-related malnutrition with inflammation," which includes that observed in inflammatory bowel disorders [1]. In this case, there is significant muscle atrophy linked to the protracted low-grade inflammatory response (with CRP values ≤40 mg/L). Effective management of malnutrition in IF requires a thorough nutritional evaluation that considers the degree and source of inflammation. Even with a sufficient calorie intake, nutritional status may not improve in sepsis or inflammatory conditions. While people with moderate (CRP 10–100 mg/L) or low (CRP <10 mg/L) inflammation responded better to nutritional interventions, those with CRP levels below 100 mg/L did not significantly improve in 30-day mortality or functional outcomes despite nutritional support, according to a study involving 1,950 medical patients [13]. According to the chapter on "Acute Surgical Intestinal Failure," this insight is a critical component of the SNAP (Sepsis, Nutrition, Anatomy, Plan) paradigm for managing intestinal failure. Sepsis and Enterocutaneous Fistula(s)." Patients with IF are more susceptible to various micronutrient deficiencies because of malabsorption and significant gastrointestinal losses. It is anticipated that adults would experience these impairments, which have been well-documented in pediatric populations. For example, even though the majority of children (101 out of 138) received micronutrient supplementation, a study looking at pediatric patients with long-term IF who switched from parenteral nutrition (PN) to oral feeding found a high rate of vitamin, mineral, and trace element deficits. This emphasizes how difficult it is to control the micronutrient requirements of this patient group [14]. Patients with high-output fistulas and stomas are especially vulnerable to deficiencies in zinc and copper, which are eliminated in bile. Because copper is a positive acute-phase reactant (bound to ceruloplasmin) and zinc is a negative acute-phase reactant (bound to albumin), care should be used when interpreting their levels in critically ill patients [15].

Consequences of Undernutrition

Historical data from 1945 in Western Holland shows that undernutrition can lead to a variety of physical and metabolic problems. Backache, leg pain, tingling in the extremities, diarrhea, and frequent nocturnal urination were among the symptoms that patients experienced. Additional symptoms included cyanosis in the limbs, hunger edema, superficial skin hemorrhages, and, in rare cases, toe gangrene. Bradycardia (heart rate of 40 beats per minute),

hypotension (systolic blood pressure of 80 mmHg), and low body temperature (down to 35°C) were also noted. Reflexes were unaffected, but a red, sensitive tongue tip was typical.

Anemia (hemoglobin of 110 g/L) and leucopenia were common, although laboratory testing showed no albumin, acetone, or glucose in the urine. Patients with chronic undernutrition were also shown to have osteoporosis and kyphoscoliosis [16].

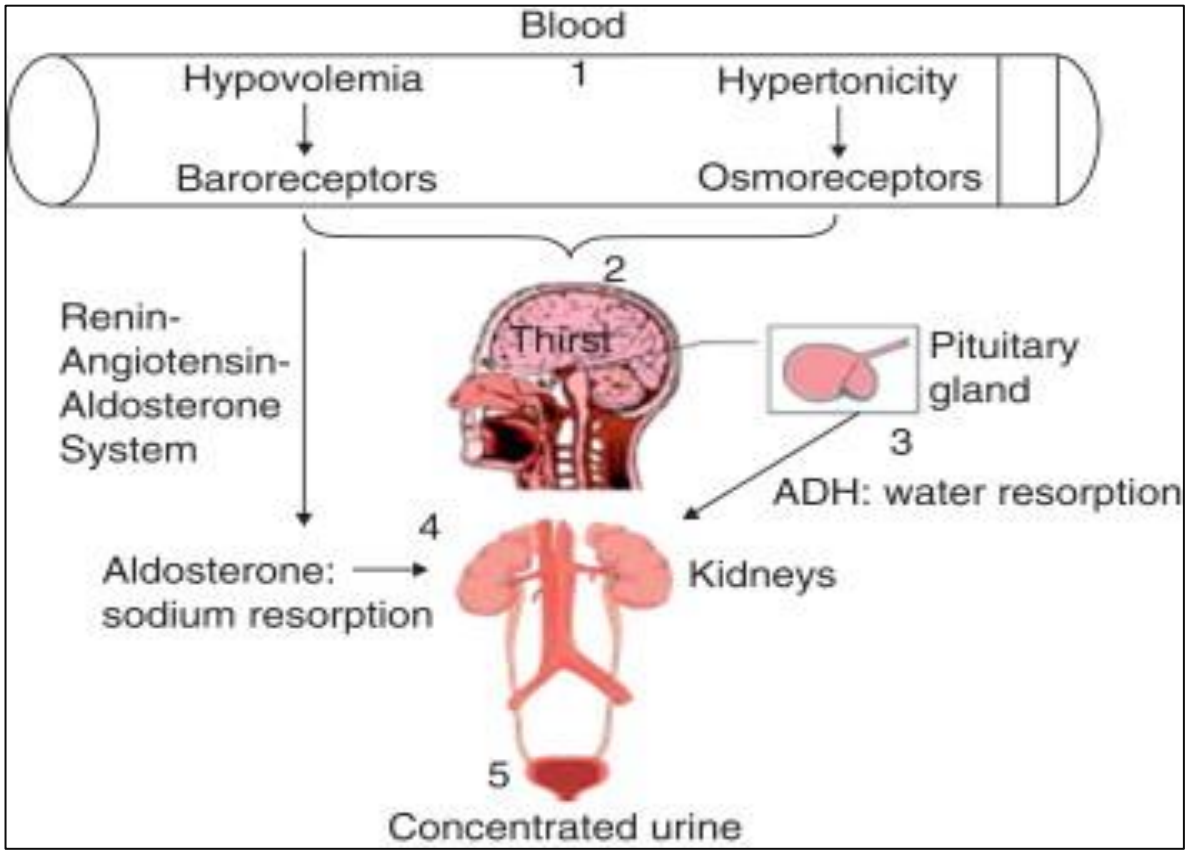

Figure 1: Pathophysiology of Dehydration.

Hunger strikers also report that they frequently feel lightheaded and dizzy, and they learn to get up slowly, so they don't pass out. Even brief durations of fasting usually result in bradycardia and a decrease in blood pressure. By day twenty or so of fasting, orthostatic hypotension can be crippled. Weakness, dizziness, and cold intolerance are typical symptoms. Tri-iodothyronine is quickly transformed into an inactive metabolite during fasting, lowering effective thyroid activity and causing weakness and cold sensitivity, whereas thyroxine levels stay constant. About 75% of hunger strikers report experiencing abdominal pain, which is another common symptom [17, 18, 19]. Undernourished hospital patients need high-dependency care because they usually have longer hospital stays and greater rates of morbidity and death than people with identical clinical conditions but normal body weight [20,21,22,23,24].

Micronutrient Deficiencies in Undernourished Populations

The following data summarizes the incidence of specific micronutrient deficiencies observed in patients with undernutrition, with a notable prevalence of iron deficiency anemia and deficiencies in various vitamins and minerals:

- **Vitamin A**: 19.1% (p-value = 0.046)
- **Vitamin B12**: 12.4% (p-value = 0.41)

- **Vitamin D**: 30.1% (p-value = 0.56)
- **Vitamin E**: 6.2% (p-value <0.001)
- **Folate**: 0% (p-value = 1)
- **Copper**: 7.7% (p-value = 0.14)
- **Iron**: 61% (p-value = 0.003)
- **Selenium**: 4.3% (p-value = 0.26)
- **Magnesium**: 10.9% (p-value = 0.71)
- **Phosphorus**: 8.8% (p-value = 0.88)
- **Zinc**: 22.9% (p-value = 0.73)
- **Anaemia**: 43.2% (p-value <0.001)
- **Iron Deficiency Anaemia**: 29% (p-value = 0.21)

These deficiencies contribute to a variety of adverse outcomes, including psychological, neurological, muscular, and gastrointestinal dysfunction, as well as increased risk of infections, sepsis, and multi-organ failure. Additionally, undernutrition can affect cardiac function (leading to bradycardia, low blood pressure, and even sudden death), liver function (resulting in steatosis and reduced synthesis of important proteins), and the endocrine system (causing delayed puberty, infertility, and osteoporosis). Poor thermoregulation, impaired wound healing, and a weakened immune system are also prominent consequences of severe undernutrition.

Biochemical Considerations:

Hepatic gluconeogenesis maintains blood glucose levels for the brain, red blood cells, and renal medulla when complicated starvation is not present. Adipose tissue, muscles, some brain regions, and the adrenal cortex switch from using glucose as an energy source to using fatty acids and ketones produced by lipolysis. While glutamine provides intestinal and lymphoid cells with an energy substrate, muscles supply alanine, glycine, and lactate for hepatic gluconeogenesis. Stores of glycogen are quickly depleted (between 6 hours and 3 days). Insulin levels fall and glucagon levels rise when there are no carbs available for energy. Like GLP-1, elevated plasma glucagon levels encourage natriuresis, which aids in weight reduction. Energy is mostly obtained from lipid sources through ketones starting on day three. However, this process causes the sodium/potassium pump to malfunction, which lowers the levels of magnesium, potassium, and phosphate in the body. Lipolysis is compromised in sepsis, which exacerbates the breakdown of muscle proteins. Even though protein catabolism is initially restricted (making up around 10% of the energy source), it eventually leads to significant protein breakdown, including in heart muscle, especially once fat reserves are depleted [19].

Psychological and Neurological Effects

Apathy/Depression

In their research, Warsaw medics emphasized the psychological effects of famine as experienced by the ancient Egyptians [4]. "The prevalence of depression, even in young people, is the most striking psychiatric finding," they said. Complete indifference, disinterest, faulty reasoning, and even incoherence are present. In his introduction to the Biology of Human Starvation [2], Sir Jack Drummond examined the psychological effects of the Dutch famine of 1945, highlighting two key findings: the relative ease of the nutritional and biochemical problems and the important psychological elements of inanition. The unresponsiveness and uncooperative behavior of persons accepting charity was one unsettling psychological symptom of famine that surfaced in Western Europe. Once daily caloric intake surpassed 1500–1800 kcal, this behavior subsided. Drummond highlighted the psychological effects of food scarcity in his description of Western Holland. These effects were correlated with calorie intake levels and ranged from mild irritability linked to a 15-20% energy deficit to apathy and the loss of higher human traits in severe hunger.

A reduction of up to 23% of body weight led to a 30% rise in depression scores during Ancel Keys' research of semi-starvation among young male volunteers over a 24-week period [2, 25]. This increase was gradually reversed during the re-feeding phase. It took up to four months for psychological health to recover. Similar trends were noted among undernourished populations in Africa and the Indian subcontinent, in war prisoners, and during the Russian famine of 1918–1921. It is important to understand that people with disease-induced undernutrition frequently exhibit social discomfort, sadness, and apathy. Once the right nutritional support is given, these issues usually go away. Furthermore, undernutrition may make the weariness brought on by illness worse. Short-term memory is frequently hampered, and sleep length rises, even if intellectual performance usually stays constant [2, 11, 26]. Restoring normal oral intake requires breaking the vicious cycle of undernutrition, which results in decreased appetite. Voluntary oral intake increases during the day after 1-2 weeks of nocturnal nasogastric tube feeding, and appetite can be recovered [26]. Patients with inflammatory bowel illness are most affected by this impact. Nutritional supplementation may be discontinued after appetite reaches a certain weight and becomes self-sustaining. It's interesting to note that patients having upper gastrointestinal surgery, like esophagectomy, may have appetite suppression comparable to that following bariatric surgery. This is probably because satiety hormones are released more readily [27].

Skeletal Muscle Function:

Muscle waste results from protein and calorie deprivation, especially the loss of Type II fast-twitch fibers, which are prevalent in the diaphragm and other respiratory muscles [28, 29, 30, 31, 32, 33]. The ensuing respiratory dysfunction increases the risk of respiratory infections, including tuberculosis, and the reduction in movement increases the risk of pressure sores, pulmonary emboli, and deep vein thrombosis [33, 34, 35, 36, 37].

Ten patients with a range of gastrointestinal illnesses had their muscular function evaluated by Lopez et al. [28], who found that undernutrition resulted in increased muscle fatigue as well as changed patterns of muscle contraction and relaxation. Supplementing nutrients rectified these deficiencies. In five morbidly obese females following a 400 kcal/day carbohydrate diet, Russell et al. [29, 30] noted comparable declines in muscle function,

including notable decreases in muscle enzyme levels and Type II fiber atrophy. Brough et al. [31] compared sepsis, surgery, and undernutrition and discovered that sepsis caused aberrant muscle function that could be distinguished from dietary alterations. But after receiving parenteral nourishment, muscular function quickly improved before body composition or plasma protein levels changed. In addition, Hill's research on malnourished inflammatory bowel disease patients showed a 20% decrease in total body protein with no impacts on respiratory muscle power. However, muscle function drastically declined after protein loss exceeded this level. Both grip strength and peak expiratory flow rate increased from 65-75% of normal within 5 days of nutritional supplementation. The recovery process was more protracted in the weeks that followed as body composition returned to normal. This demonstrates a two-stage reaction to refeeding: a more delayed restoration of body cell mass after an initial, quick improvement in cellular function. Muscle function can be seriously hampered by nutritional shortages in minerals and electrolytes, especially in patients with gastrointestinal conditions. For example, magnesium or calcium deficits can impact peristalsis and raise neuromuscular excitability, which may result in seizures; hypokalemia can cause muscle weakness or paralysis; and a selenium deficiency can influence both cardiac and skeletal muscle function.

Respiratory, Cardiovascular, and Gastrointestinal:

The main way that undernutrition affects respiratory function is by negatively affecting respiratory muscles, which affect ventilation and the capacity to cough up secretions [38,39,40,41,42,43,44]. Undernutrition also makes it more difficult to wean off of mechanical breathing [43] and reduces the respiratory response to hypoxia [45,46]. On the other hand, it has been demonstrated that dietary supplementation improves ventilatory function and speeds up the weaning process. The increased likelihood of respiratory difficulties after surgery, trauma, or acute sickness is one of the main effects of undernutrition; bronchopneumonia is usually the result of gradual starvation [16].

A failure to fulfill the increased respiratory demands imposed by excessive caloric intake, especially from carbohydrates, is a result of weakening respiratory muscles. When total calorie intakes surpass 40 kcal/kg/day or when carbohydrate loads surpass 5 mg/kg/min, these patients may develop dyspnea because of the resulting increase in carbon dioxide generation and oxygen use [47,48]. Studies on advanced starvation, like those done in Warsaw [4], show that famine is linked to lower systolic and diastolic blood pressures in the sympathetic nervous system and cardiovascular system, both at rest and in response to activity. Bennett et al.'s study [49] showed that when healthy males were hungry for 48 hours while taking salt supplements, their supine blood pressure decreased slightly. When they stood up, their systolic blood pressure dropped by 15 mmHg, which was not the case for non-deprived controls. Heart rate, blood pressure, and cardiac muscle mass all decrease with starvation; although the decrease in cardiac muscle is not as severe as that in skeletal muscle, it can nonetheless put a person at risk for heart failure [50,51]. Furthermore, the risk of ventricular arrhythmias is increased by a prolonged QT interval, which is the function that recovers the slowest with refeeding [52]. Sudden death is more likely to occur in patients with bradycardia below 40 bpm, a prolonged QT interval, or a postural drop in blood pressure of more than 20 mmHg [12]. Undernourished

people have a markedly lower resting metabolic expenditure, which is further influenced by the compromised function of the sympathetic nervous system, a major regulator of metabolic rate. When evaluating the dietary requirements of patients suffering from nutritional depletion, this needs to be carefully considered.

Depending on its severity, undernutrition can have a complex effect on the gastrointestinal system. For example, the Warsaw studies [4] discovered that those who lost over 30% of their body weight had problems secreting stomach acid. Moreover, villous atrophy, compromised small bowel mucosal function, and elevated intestinal permeability can arise from inadequate luminal nutrition during parenteral feeding, whether in healthy people or those suffering from undernutrition [53,54,55,56]. Such an atrophy may contribute to endotoxemia, immunological impairment, and even multi-organ failure by causing bacterial translocation and mucosal barrier dysfunction [57]. Antibiotics have been linked to significant improvements in recovery and mortality rates when added to the treatment regimen for children suffering from severe acute malnutrition [58]. Diarrhea can also come from negative effects on the colon, which hinder the reabsorption of water and electrolytes [59,60]. Additionally, pathogenic bacteria may migrate into the small intestine, causing changes in the intestinal microbiota that worsen malabsorption [61]. When oral or enteral intake is not available, or when some parts of the colon are skipped, these gastrointestinal alterations are also noticeable during parenteral feeding. These alterations can be undone and adaption accelerated by reintroducing food orally or enterally, especially in the ileum of people with short bowel syndrome. Both the small and large intestinal mucosa may develop and operate better when certain substrates, such soluble fiber and prebiotics like fructo-oligosaccharides, encourage the synthesis of short-chain fatty acids. The protective function of the gut-associated lymphatic tissue may be further enhanced by glutamine, a necessary fuel for the mucosal epithelium.

Undernourished adults and newborns both exhibit markedly compromised pancreatic function. According to research by Winter et al. [62,63,64], pancreatic protein synthesis and enzyme production were reduced before therapy in patients with a mean BMI of 13.6 kg/m² before treatment and 16.5 kg/m² following nutritional support. This resulted in the loss of 70% of pancreatic and stomach secretions, which hindered the absorption of lipids and xylose. After refeeding, these metrics stabilized. Additionally, it was shown that before therapy, there was a decrease in pancreatic amylase and trypsin production, as well as gastric acid secretion. Although it might be difficult to differentiate from environmental enteric dysfunction (EED), sometimes known as "tropical sprue," malnutrition-related enteropathy is a well-known illness. Villous atrophy, crypt hyperplasia, tight junction loss, and inflammatory cell infiltration into the intestinal mucosa are the hallmarks of this illness [65]. This is especially crucial when evaluating small bowel biopsies since, in cases of acute malnutrition, they can be utilized to identify the presence of enteric myopathy or neuropathy. Gastric emptying for both liquid and solid meals is slowed in undernourished people, especially in those with anorexia nervosa [66,67]. Gastric dilatation, peptic ulcers, acid reflux, and colonic atony, which results in constipation, are further gastrointestinal problems that have been documented.

Furthermore, compared to those who are well-nourished, patients with Crohn's disease who are malnourished see a worse course of their illness. It's interesting to note that weight

gain and nutritional support have been demonstrated to lower inflammation and enhance disease outcomes in these patients [68]. Additionally, there are notable alterations in the microbiota of malnourished children, including a decrease in diversity and a rise in the relative abundance of pathogenic taxa from the *phylum Proteobacteria, including Enterobacter, Escherichia, Klebsiella, and Shigella. At the same time, beneficial genera such as Bifidobacterium, Butyrivibrio, Faecalibacterium, Lactobacillus, and Roseburia* have shown a decline in relative abundance [69].

Immunological Function

A number of immunological functions, including cell-mediated immunity, are severely compromised by undernutrition, which increases vulnerability to infections. This phenomenon has been extensively studied in adult populations [4, 70,71,72,73], children [74,75], and the elderly [76]. On the other hand, nutritional assistance has continuously demonstrated advantages in lowering the rate of infections, including postoperative infections [74,75,76,77,78,79,80]. Immune responses are primarily mediated by the gastrointestinal tract and gut-associated lymphatic tissue. By providing these immune tissues with vital nutrients, luminal nutrients, such as glutamine, can have positive effects. But the connection between immunological competence and nutritional status is complex and can change depending on the clinical setting and the particular dietary deficits at play. Immune function may be negatively impacted by mineral and micronutrient deficits in addition to protein-energy deprivation. For example, the significance of zinc in preserving cell-mediated immunity was highlighted by Good et al. [73]. When severe protein-energy malnutrition occurs, immune competence—which is frequently measured by delayed cutaneous hypersensitivity (DCH)—is noticeably compromised. It is crucial to understand that this test can be influenced by a number of illnesses [53] and drugs, which makes it an unreliable indicator of malnutrition in critically ill patients. Infections (bacterial, viral, and granulomatous), diseases like cirrhosis, hepatitis, uremia, trauma, burns, and hemorrhage, as well as the use of steroids, immunosuppressants, cimetidine, warfarin, and possibly aspirin, as well as general anesthesia and surgery, are among the factors that non-specifically change DCH. Due to the possibility of several factors influencing its interpretation, DCH is frequently unreliable for evaluating nutritional status in critically ill patients. Meakins et al. [54] illustrated the intricacy of immune function in malnourished individuals by showing that even basic therapies, like abscess drainage, can correct immunological deficits. As a result, immunity cannot be readily evaluated in clinical settings and is not a reliable sign of protein-energy deficiency [55].

Thermoregulation

The Warsaw experiments showed that malnourished people had lower-than-normal body temperatures and did not develop fever responses to diseases like TB and typhoid [3]. In clinical settings, similar thermoregulatory problems are seen in the aged and undernourished. According to Bastow et al. [81], patients who were properly nourished had core temperatures above 36 °C, but older women with shattered femurs and severe undernutrition had temperatures below this range. Fellows et al. [82] observed normal vasoconstriction but no increase in thermogenesis in undernourished individuals exposed to cold stimuli, suggesting compromised thermoregulatory responses. On the other hand, research by Mansell et al. [83]

and others has demonstrated that the thermogenic response is recovered when body composition is returned to normal with nutritional support. This implies that, although through different processes, both temporary hunger and weight reduction affect thermoregulation. Increased pain, restlessness, and irritability are linked to core temperatures in people between 34 and 36 °C. When the temperature drops further to 33–34 °C, patients experience disorientation, fatigue, and a changed sense of temperature; they frequently take off their clothes even if they feel cold. Coma may develop at temperatures between 26 and 32 °C, and death is likely below 26 °C, occasionally with cardiac fibrillation prior [12]. Because their temperature frequently stays below normal, undernourished individuals are more susceptible to hypothermia, which makes it more difficult to diagnose sepsis or fever.

Wound Healing

As an adaptive survival strategy, the metabolic response to injury mobilizes substrates and energy to fulfill the demands of sickness. When this reaction goes too far, though, it may endanger life. The mobilization of muscle nitrogen to meet metabolic demands is a crucial component of this response, and rats fed a low-protein diet have impaired muscle nitrogen mobilization [85]. Wound healing is frequently compromised in undernourished people [86,87,88,89,90,91,92]. Human wound healing is negatively impacted by inadequate preoperative nutrition, as Windsor et al. [86] showed. Haydock and Hill [87] also found that even slight undernutrition hinders healing in postoperative patients. Additionally, patients who are malnourished are more likely to get pressure sores, which take longer to heal [88]. Wound healing has been demonstrated to be accelerated by nutritional assistance; this process requires a sufficient intake of vital minerals and micronutrients, including vitamins A, B, C, D, E, and K. Because zinc deficiency has been associated with poor wound healing, which can be reversed by taking zinc supplements, zinc, a cofactor for collagen synthesis, is especially important [89, 90, 91].

Endocrine and Bone Health

A BMI of less than 17 kg/m2 is indicative of anorexia nervosa, which frequently results in amenorrhea, hypothalamic dysfunction, and subsequent effects on the pituitary and gonads. Patients who have cachexia as a result of gastrointestinal disorders also exhibit similar endocrine abnormalities. Decreased bone density and diminished muscular mass, strength, and energy are caused by low levels of sex and growth hormones combined with high amounts of cortisol and glucagon. In one case study, a young guy with Crohn's disease had low serum testosterone levels and bone density three standard deviations below the average for his age; both returned to normal following two years of proper nutritional therapy. Bone structure is also adversely affected by protein-energy deficiency, with osteoporosis more common in those who are undernourished. Moreover, osteomalacia and vitamin D insufficiency can result from the malabsorption of fat-soluble vitamins.

Growth and Development

Childhood undernutrition significantly diminishes growth velocity, although nutritional interventions can restore it to some extent. However, extended periods of undernutrition result in irreversible limitations in achieving genetic height potential, even with subsequent optimal

nutrition [93, 94]. Furthermore, puberty is frequently delayed due to the impaired functioning of the pituitary-gonadal axis, a phenomenon commonly observed among adolescents suffering from severe gastrointestinal disorders and malabsorption.

Fluid and Electrolytes

There are significant effects on fluid and electrolyte homeostasis from the interaction between hunger and refeeding. Cell membrane pumps, whose activity is insulin-dependent and accounts for around one-third of resting energy expenditure, control intracellular concentrations of vital ions like sodium and potassium [95]. Even though plasma levels may seem normal during hunger, the decreased action of these pumps causes a considerable depletion of intracellular ions, such as potassium, magnesium, and phosphate. The significant loss during famine is highlighted by the fact that 98% of the body's potassium is found intracellularly [96,97]. Oedema may result in following refeeding, which is partially explained by the reactivation of membrane pumps that control the release of water and sodium from cells. Insulin activity and potassium deficit may worsen the ensuing incapacity to eliminate this burden, hence compromising renal function [99]. Strict limitations on salt and fluid intake are necessary to reduce the dangers of fluid overload and cardiac problems, especially in malnourished patients with cardiac atrophy, because sodium excretion significantly decreases with refeeding [100–102]. As the sodium load and renal arterial constriction brought on by hyperchloremia increase the risk of fluid retention and oedema, particularly bowel oedema that can hinder the healing of anastomoses and fistulae, medications or fluids containing 0.9% saline should be taken with caution [103–105]. High gastrointestinal losses of fluids and electrolytes, such as sodium, potassium, chloride, and magnesium, are frequently seen in patients with intestinal failure (IF). Therefore, it is essential to carefully assess fluid, and electrolyte needs every day in order to guarantee proper maintenance and replacement therapy [106]. In critically unwell IF patients with resistant oedema, uncommon disorders like beriberi may need to be considered. It is important to aim for a slightly negative sodium and fluid balance as well as sufficient potassium supply. Even if plasma levels seem acceptable, appropriate potassium supplementation is necessary because whole-body potassium depletion hinders sodium excretion since there is not enough potassium for renal exchange mechanisms [106,107]. It is essential to accurately record fluid balance in order to account for its effects on body weight and nutritional status, including gastrointestinal losses.

Albumin

Low serum albumin has historically been mistakenly thought to be a sign of starvation. Although malnourished hospitalized patients often develop oedema, hypoalbuminemia is not a direct result of malnutrition or protein deficiencies. This idea is further refuted by data from anorexics, who frequently have low body mass indexes but have normal albumin levels free of oedema [108–111]. A complicated interaction between synthesis, breakdown, body losses, and distribution throughout intravascular and extravascular compartments is reflected in serum albumin concentration. About one-third of albumin, a highly water-soluble protein, is found in the intravascular compartment, whereas the remaining two-thirds are found in the extracellular

region [112]. To ensure equilibration within tissues over a period of 7–10 days, the plasma albumin pool exchanges with extravascular albumin on a daily basis. The decreased polysomal aggregation necessary for protein synthesis causes a significant decrease in hepatic albumin synthesis within 24 hours of fasting [114]. However, under brief dietary restriction, compensatory mechanisms such decreased degradation, extravascular albumin mobilization, and intravascular water contraction frequently avert notable changes in plasma albumin levels [115,116]. Clinical and experimental research have shown that plasma albumin content is not significantly affected by prolonged protein-calorie deficiency. In hospitalized patients, hypoalbuminemia is mostly caused by factors including inflammation, gastrointestinal or renal losses, and increased vascular permeability [118–120]. Albumin losses to the extravascular compartment are greatly increased by illnesses like septic shock and cancer cachexia. According to studies, excessive saline infusion resulted in sustained decreases in serum albumin, inflammatory stress and improper intravenous saline treatment can also cause rapid drops in plasma albumin [123]. Serum albumin correlates inversely with acute-phase reactants like C-reactive protein (CRP) and is a good measure of illness severity while being a poor nutritional marker [124]. Furthermore, poor outcomes in situations like enterocutaneous fistulae and higher mortality in patients with intestinal failure have been associated with low serum albumin levels [125]. Additionally, it is a useful indicator for determining sodium and fluid overload [126].

Nursing Case of Intestinal Failure, Malnutrition, and Dehydration:

Intestinal failure (IF) is a complex clinical condition characterized by the inability of the gastrointestinal (GI) tract to digest and absorb nutrients, water, and electrolytes adequately to maintain normal growth, health, and body composition. It is frequently associated with malnutrition and dehydration, which exacerbate the condition's severity and complexity. This case study examines the nursing management of a patient with IF complicated by malnutrition and dehydration, emphasizing the roles of assessment, intervention, and monitoring.

Case Presentation

A 52-year-old male patient presented to the emergency department with a history of chronic diarrhea, abdominal pain, and unintentional weight loss of 15 kg over the past three months. The patient had a medical history of Crohn’s disease, which had led to multiple bowel resections, leaving him with a significantly reduced absorptive surface area. Laboratory investigations revealed severe electrolyte imbalances, hypoalbuminemia, and elevated serum creatinine, indicating dehydration and renal dysfunction. Physical examination showed generalized muscle wasting, dry mucous membranes, and orthostatic hypotension. The patient was diagnosed with short bowel syndrome (SBS), a type of intestinal failure, accompanied by protein-energy malnutrition and dehydration.

Nursing Assessment

The primary nursing priority in this case involved a comprehensive assessment. This included evaluating the patient's hydration status through clinical signs such as skin turgor, blood pressure, heart rate, and urine output, as well as laboratory indicators like serum sodium, potassium, and creatinine levels. Nutritional status was assessed using anthropometric

measurements, dietary history, and biochemical markers such as serum albumin and prealbumin levels. The patient's GI function was evaluated by documenting stool output, frequency, and consistency to determine fluid and nutrient losses. Psychosocial factors were also considered, as chronic illnesses like IF can lead to significant emotional distress and reduced quality of life. This was assessed through patient interviews and validated tools to identify potential anxiety, depression, or social support deficits.

Nursing Interventions

The nursing care plan focused on stabilizing the patient's hydration, correcting malnutrition, and managing IF-related complications.

1. **Hydration Management**: Initial interventions included intravenous (IV) rehydration with balanced crystalloid solutions to correct dehydration and electrolyte imbalances. Hypokalemia and hypomagnesemia were addressed through IV supplementation, with close monitoring to prevent refeeding syndrome, a potentially fatal complication in malnourished patients undergoing nutritional rehabilitation. Oral rehydration therapy was introduced gradually, tailored to the patient's absorptive capacity and sodium requirements. Strict input-output monitoring was essential to assess fluid balance and guide therapy adjustments.
2. **Nutritional Support**: Nutritional rehabilitation was a cornerstone of management. Enteral nutrition was prioritized to preserve gut integrity, using polymeric or semi-elemental formulas based on the patient's tolerance. For severe malabsorption, parenteral nutrition (PN) was initiated to meet the patient's caloric, protein, and micronutrient needs. The PN regimen was individualized, ensuring adequate provision of essential amino acids, lipids, vitamins, and trace elements. Nurses played a critical role in administering and monitoring PN to prevent complications such as catheter-related infections or metabolic disturbances.
3. **Pharmacological Management**: Pharmacological interventions included anti-motility agents like loperamide to reduce diarrhea and optimize nutrient absorption. Proton pump inhibitors were prescribed to decrease gastric acid secretion, minimizing the risk of gastric hypersecretion common in SBS. Nurses ensured proper medication administration, monitored therapeutic effects, and educated the patient on adherence and potential side effects.
4. **Skin Integrity and Wound Care**: Chronic diarrhea increased the risk of perianal skin breakdown. Nurses implemented a skin care regimen, including barrier creams and frequent cleansing, to prevent and manage irritation. Education on maintaining hygiene and reporting changes in skin integrity was also provided.
5. **Psychosocial Support**: The patient was referred to a multidisciplinary team that included dietitians, social workers, and psychologists. Nursing interventions focused on active listening and providing emotional support to alleviate the psychological burden of living with IF. Education sessions addressed the patient's concerns, promoting self-management skills and adherence to the care plan.

Monitoring and Evaluation

Continuous monitoring was vital to evaluate the effectiveness of interventions and detect complications early. Daily assessments included monitoring vital signs, fluid balance,

and stool output. Regular laboratory tests were conducted to track trends in electrolytes, renal function, and nutritional markers. Improvements in hydration were evidenced by increased urine output and normalized blood pressure. Nutritional progress was measured through weight stabilization, increased serum albumin levels, and improved muscle mass. The patient's tolerance to enteral nutrition was assessed by monitoring signs of GI distress, such as nausea or abdominal cramping. Gradual weaning from PN to oral or enteral nutrition was planned based on the patient's clinical response and GI adaptation.

Nursing Implications

This case highlights the pivotal role of nursing in managing IF complicated by malnutrition and dehydration. Nurses are central to providing holistic care, ensuring that physical, nutritional, and psychosocial needs are met. Their responsibilities include performing detailed assessments, implementing evidence-based interventions, and facilitating patient education to promote long-term self-management. The multidisciplinary approach in IF management emphasizes the collaborative efforts required to address this complex condition. Nurses serve as patient advocates, coordinating care and ensuring adherence to best practices. Additionally, their role in monitoring complications such as refeeding syndrome, catheter-related bloodstream infections, and metabolic derangements underscores their critical contribution to patient safety. Intestinal failure, particularly when compounded by malnutrition and dehydration, poses significant challenges in clinical management. This case underscores the importance of comprehensive nursing care, which integrates hydration, nutritional support, pharmacological management, and psychosocial interventions. Through meticulous assessment, timely interventions, and ongoing evaluation, nurses can improve patient outcomes and quality of life. By fostering a collaborative, patient-centered approach, they play a vital role in managing the complexities of IF and supporting the patient's journey toward recovery.

Conclusion:

Dehydration and malnutrition significantly impact health, particularly among patients with gastrointestinal disorders, where impaired nutrient absorption and elevated metabolic demands contribute to worsening outcomes. This review highlights the critical role of comprehensive nursing care plans in addressing these challenges. Historical data, such as starvation studies, illustrate the functional and survival implications of significant body weight loss. For instance, a 35% reduction in body weight marks a critical threshold beyond which mortality risk dramatically escalates. Intestinal failure, characterized by malabsorption, inflammation, and micronutrient deficits, exemplifies the complexity of managing malnutrition in clinical settings. Common deficiencies in vitamins (A, D, E) and minerals (iron, zinc) underline the need for precise nutritional assessments and tailored interventions. Strategies to address these include individualized hydration protocols, targeted nutritional supplementation, and continuous monitoring of biochemical markers. Nursing care plans play an essential role in mitigating the effects of undernutrition. Effective strategies involve early identification of at-risk patients, incorporating advanced diagnostic tools, and multidisciplinary approaches to manage both acute and chronic cases. Evidence suggests that addressing micronutrient deficiencies alongside macronutrient needs enhances recovery and reduces morbidity, particularly in vulnerable populations such as infants and those with chronic conditions like

inflammatory bowel disease. Future trends include the integration of technology, such as digital health platforms and wearable devices, to enable real-time monitoring of hydration and nutritional status. Additionally, innovations in personalized medicine offer promise in tailoring interventions to individual patient needs, optimizing care delivery. In conclusion, addressing dehydration and malnutrition requires a holistic approach combining clinical expertise, technological advancements, and patient-centered care. By adopting evidence-based strategies, nursing professionals can significantly improve outcomes, reduce hospital stays, and enhance the quality of life for patients with gastrointestinal disorders.

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الرعاية التمريضية للجفاف وسوء التغذية - مراجعة محدثة وتوجهات مستقبلية للسيطرة على الاضطرابات المعوية

الملخص:

الخلفية :يُعتبر الجفاف وسوء التغذية من العوامل الرئيسية المساهمة في النتائج الصحية السلبية، خصوصًا لدى المرضى الذين يعانون من اضطرابات معوية. يُعرّف سوء التغذية، وفقًا للجمعية الأوروبية للتغذية الإكلينيكية والأيض (ESPEN)، بنقص المغذيات الذي يضعف الوظائف الجسدية والعقلية، ويزداد سوءًا بسبب الأمراض المعوية التي تعوق امتصاص المغذيات وتزيد من المتطلبات الأيضية.

الهدف :تستعرض هذه المراجعة المحدثة استراتيجيات الرعاية التمريضية لإدارة الجفاف وسوء التغذية مع التركيز على الاضطرابات المعوية، وتسلط الضوء على التوجهات المستقبلية لتحسين النتائج.

الطرق :تُركّز المراجعة على دمج البيانات من الدراسات التاريخية والمعاصرة حول سوء التغذية والجفاف، بما في ذلك الأبحاث المتعلقة بالمجاعة وسوء التغذية الناتج عن الأمراض ونقص المغذيات الدقيقة لدى الأشخاص الذين يعانون من فشل معوي. تشمل المحاور الرئيسية التأثيرات السريرية، وحدود البقاء على قيد الحياة، والآثار الفسيولوجية لسوء التغذية.

النتائج :تكشف النتائج أن سوء التغذية يؤدي إلى ضعف وظيفي، ونقص المغذيات الدقيقة، وزيادة معدلات المرض. تُفاقم الاضطرابات المعوية سوء التغذية من خلال سوء الامتصاص والالتهاب، مع نقص محدد في الفيتامينات A و Dو E، ومعادن مثل الحديد والزنك والمغنيسيوم. تُظهر الدراسات التاريخية حول المجاعة أن فقدان 35% من وزن الجسم يمثل حداً

لبقاء الإنسان، حيث تزيد معدلات الوفيات بشكل حاد بعده. تتطلب الرعاية التمريضية الفعالة خططًا فردية تشمل تقييم التغذية، وحالة الترطيب، ودعم المكملات الغذائية.

الخلاصة :تؤكد المراجعة على أهمية دمج الدعم الغذائي المتقدم وخطط الرعاية المتمركزة حول المريض والتدخلات المبكرة لإدارة الجفاف وسوء التغذية. تُبشّر التوجهات الناشئة، مثل الاستفادة من الأدوات التقنية لمراقبة الحالة الغذائية والمكملات الفردية، بتحسين النتائج لدى مرضى الاضطرابات المعوية.

الكلمات المفتاحية :الجفاف، سوء التغذية، الاضطرابات المعوية، خطط الرعاية التمريضية، الدعم الغذائي، نقص المغذيات الدقيقة.