About the Book

Therapeutic Nutrition: A Comprehensive Guide for Healthy Lifestyle" is a book that delves into the principles of nutrition therapy and its role in promoting health and managing various medical conditions. It typically covers topics such as the relationship between diet and health, nutritional requirements for different age groups, and specific dietary interventions for chronic diseases like diabetes, cardiovascular diseases, and gastrointestinal disorders. The book often includes: Detailed explanations of nutrients and their functions in the body. Guidelines for creating balanced and therapeutic meal plans. Case studies and practical applications of nutrition therapy. Discussions on the impact of lifestyle choices on health and disease prevention. This comprehensive approach aims to provide readers, including healthcare professionals and individuals interested in health and wellness, with the knowledge and tools needed to improve dietary habits and overall health.

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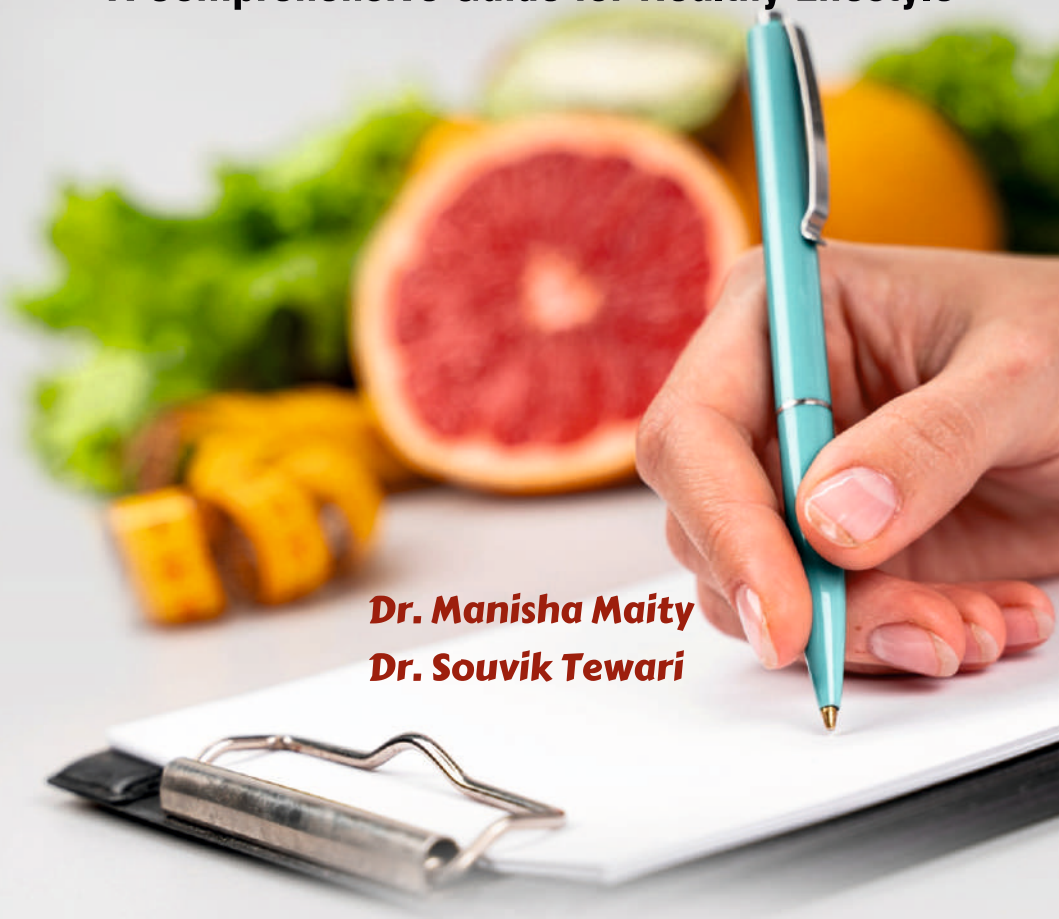
A Comprehensive Guide for Healthy Lifestyle



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THERAPEUTIC NUTRITION

A Comprehensive Guide for Healthy Lifestyle

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Contents

Preface

In recent years, the significance of nutrition in maintaining health and preventing disease has gained unprecedented attention. As our understanding of the intricate relationship between diet and health evolves, it becomes increasingly clear that therapeutic nutrition is not merely about consuming food to sustain life, but about harnessing the power of nutrients to promote healing, enhance well-being, and prevent chronic illnesses. This book, "Therapeutic Nutrition**:** A Comprehensive Guide for a Healthy Lifestyle, " aims to provide a thorough exploration of the principles and practices of therapeutic nutrition. It is designed for healthcare professionals, nutritionists, dietitians, and anyone with an interest in improving their health through dietary interventions. Throughout this book, we delve into various aspects of therapeutic nutrition, including the latest scientific research on the role of nutrients in health and disease, practical guidelines for dietary planning, and case studies illustrating the real-world application of nutritional therapies. Each chapter is crafted to build on the previous one, creating a cohesive and comprehensive guide that is both informative and practical.

Thank you for joining us on this journey towards a healthier, more vibrant life.

Sincerely,

[Dr. Manisha Maity]

Chapter - 1
Introduction to Therapeutic Diet

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Chapter - 1

Introduction to Therapeutic Diet

Shreyasi Das

1. Introduction

A balanced diet is necessary for the human body to function normally in both health and illness. A person's requirement for their food may change in conditions where their health is damaged, both in terms of quantity and quality (Gopalan *et al*., 2004; Therapeutic Diet 2018). The recommendation of fluid-only meals is a typical aspect of hospital care; it is frequently given after gastrointestinal surgery, in cases of severe gastrointestinal sickness, or to patients who are incapable of chewing or swallowing solid food (NSW Agency for Clinical Innovation. Therapeutic diet). Free fluid diets usually come after clear fluid diets. These drinks are prescribed with the understanding that they are typically well absorbed, leave little to no intestinal residue, and lessen the digestive system's excretions (Hancock *et al*., 2002).

The free fluid diet can be less restricted than the clear fluid diet, allowing a wider variety of beverages without added fibre (NSW Agency for Clinical Innovation. Therapeutic diet).

Diets consisting solely of fluids are typically deficient in all macro- and micronutrients. As a result, it is advised that you utilise them for no more than three days (Hancock *et al*., 2002).

Malnutrition has been linked to a longer hospital stay and also increased risk of death and morbidity in hospitals (Gout *et al*., 2009; Barker *et al*., 2011).

It has been said that dietary interventions for hospitalised patients improve clinical results in a number of sectors, such as orthopaedic and cancer patients (Isenring *et al*., 2004; Ligthart-Melis *et al*., 2013; Wong *et al*., 2004). Patients' intakes of protein and energy can be increased through nutrition supplements, personalised dietary counselling, or a combination of the two. This can impact the patients' overall nutritional well-being (Milne *et al*., 2009; Baldwin *et al*., 2011). Dietitians play an important role in ensuring patients in hospitals receive the nutrients they require. One of the main responsibilities of hospital-based dietitians is to prescribe enteral and parenteral nutrition, which

can give patients extra energy and protein, especially if they are following restrictive diets like fluid-only regimens (Ferguson *et al*., 1999).

1.1. Diabetes Mellitus

- One of the main focus for diabetes is to avoid both macrovascular and microvascular problems. This includes instructing patients on helping oneself lifestyle changes, like selecting meals, modifying portion sizes, and switching things around when creating a diet plan.
- Ensuring an overall well-being suitable for age and gender, together with dietary advice that consider religious and cultural diversity, is essential to managing the dual downstream concerns of dysglycemia and dyslipidemia.
- Exchange and portion lists must take into account ethnogeographical variances in the availability of macronutrient groups with easily exchangeable amounts of calories, protein, fat, and carbohydrates.
- In order to help diabetics "live with the disease" with the fewest consequences possible, health care practitioners need to shift their perspective to include the socioeconomic status of their patients (Garg *et al*., 2018; Ghosh *et al*., 2018; Singh *et al*., 2017; Pal *et al*., 2010).

1.2. Wilson Disease

- In Wilson's disease, local obstructions in particular organs, such as liver cirrhosis, are insufficient to regulate copper deposition in the eyes, the brain, kidneys, and liver.
- The two most important therapies are zinc supplementation in the diet to decrease copper absorption from the gut and medication to help eliminate excess copper in urine.
- Researchers believed that restricting consumption of food groups high in copper (liver and shellfish excluded) might not be necessary to minimise problems with optimal medication adherence (Russel *et al*., 2018).

1.3. Inflammatory Bowel Disease

Patients with celiac disease (CD) who have a lifetime intolerance to gluten must refrain from eating anything that contains gluten as well as naturally gluten-free but potentially contaminated items.

This comprehensive dietary approach aims to prevent symptom aggravation and minimise both immediate and delayed consequences.

1) To begin with, cutting out gluten restricts meal options and exchanges, resulting in an imbalanced intake of both micro and macro nutrients that could cause deficiencies that are either clinical or subclinical. Based on comprehensive data on gluten-containing foods, we require a patient-tailored dietetic regimen that strictly adheres to the qualitative as well as quantitative composition of diets.
2) Second, a long trouble-free life and total remission of mucosal damage may not be achieved by following a gluten-free diet.
3) Thirdly, a new treatment strategy recommended avoiding both gluten and wheat as well as fermented monosaccharides, disaccharides, oligosaccharides, and polyols—sugar alcohol and short-chain carbohydrates —in cases where Celiac disease and Irritable Bowel Syndrome exist with similar symptoms (Taus *et al*., 2016).

In order to break free from the glass ceiling of rigid beliefs and acknowledge their poor understanding of both the science and the art of nutrition and diets based on scant translational research, healthcare providers must first transform themselves. Then and only then we will be able to guarantee that every medical practitioner will go through the food chart and its implementation in any sickness. To do this, we need to increase the practice of medical schools to teach undergraduate and graduate students about diet and nutrition, emphasising the importance of a therapeutic diet. Family medicine specialists, in particular, require particular attention as they work to promote primary care at the community level (Pal *et al*., 2014; Pal *et al*., 2016).Furthermore, the focus of our national strategy ought to have been on educating the public about the significance of diets for all-encompassing health care—preventive, promotive, and curative—from conception to death (Sharma *et al*., 2018).

To emphasise the significance of diet as a crucial element of recovery, we must begin with likes and dislikes, family customs, religious views, and other limitations. This can be done by identifying the requirements of patients and facilitating a wide-ranging conversation among the community. This will assist with the obstacles associated with implementing the meal plan's timing and manner with the individual and their family, develop self-sufficiency in eating, provide accessible equipment, and create the ideal environment.

Clinical practice guidelines are required, along with a thorough diet plan tailored to each patient's age, gender, and level of morbidity, as well as daily updates from the care seekers to provide them with personalised guidance (Pal, 2017).

We must take use of our system of healthcare delivery to spread our expertise among the impoverished in both rural and urban areas (Saxena *et al*., 2015).

Despite advancements in healthcare, there are high-quality research on the short, medium, and long-term effects of therapeutic diets in nutrition (Pal *et al*., 2015; Pal *et al*., 2015).

The most popular eating patterns in Western nations are the plant-based diets (PBD), low-carb and low-fat meals, the Mediterranean-style diet (MD), and the Dietary Strategies to Lower Hypertension (D'Almeida *et al*., 2018; Vogt *et al*., 1999; Anand, 2015; Di Daniele *et al*., 2017; Andreoli *et al*., 2008).

1.4. Plant-based diet (PBD)

- PBD drawing support from the scientific community as well as the general public, and is currently one of the most popular dietary regimens in Western countries. In fact, there seems to be a considerable spread of PBDs due to the increasing number of people utilising them (Dedehayir *et al*., 2019).
- There are different types of PBD diets; these includes diets that prohibit eating fish and meat but allow the consumption of dairy products, eggs, and milk; other diets prohibit consuming any food that comes from animals (Rocha *et al*., 2019; Clarys *et al*., 2014).
- The main problem with these diet plans is the potential for developing nutritional deficiencies are calcium, iron, zinc, vitamin B12, omega-3 fatty acids, iodine, and vitamin D.
- PBDs, on the other hand, are abundant in fruits and vegetables and are distinguished by a high concentration of fibre, antioxidants, phytochemicals, and omega-6 fatty acids (Hever *et al*., 2017).
- PBDs can be suitable for all age group, which includes pregnant and nursing women, if they are well-balanced (Melina *et al*., 2016). For moral, practical, or financial reasons, those who decide to make a PBD have a common in the surroundings (Hopwood *et al*., 2020).

1.5. The ketogenic diet, or KDjjkhjhjj

- The ketogenic diet (KD) is considered as non-pharmacological therapy option for DRE, particularly in children who are surgically unreachable (Bough, 2008; Gonzalez *et al*., 2015).
- It is a regular calorie-regime that is high in fat, moderate in carbohydrates, and provides enough protein. Since hunger has been

shown to reduce the seizures, this diet for therapy has been utilised to mimic the metabolic characteristics of fasting in DRE patients (Augustin *et al*., 2018).

1.5.1. Ketogenic diet types: The following kinds of KDs have been linked to the treatment of epilepsy**:**

1) Classic-ketogenic diet or KD

- Classic KD is the most widely utilised diet for therapy in DRE treatment; it was initially reported by Wilder in 1921 (Wilder, 1921).
- The main fat in this diet comes from long chain triglycerides (LCT), which are derived from popular foods.
- Generally, the proportion of fat to carbs plus protein is 4**:**1. The ratio could be reduced to 3**:**1 for children in need of a higher protein diet for growth.
- Eighty to ninety percent of calories are made up of fat (Kossoff *et al*., 2014; Hartman *et al*., 2007).
- The target patients' initial hospital stay may be helpful for starting and adjusting to the diet. It is preferable for a professional dietician to calculate the diet in order to teach the child and their family how to stick to it (Pfeifer *et al*., 2008).

2) Medium Chain Triglyceride diet

- The MCTD is originated in the 1950s and additionally serves to treat DRE among children with positive outcomes.
- This diet is said to be more palatable. MCTD generates more ketones compared to an LCT-based diet because it mostly contains octanoic (C8) and decanoic (C10) fatty acids.
- Portal blood allows for more effective absorption and delivery of the substance to the liver. Because of MCTD's strong potential for ketogenesis, eating less total fat and more carbohydrates and protein is the result. Because of this, MCTD is more kid-friendly and appetising than traditional KD.
- As multiple studies have shown, the efficiency that comes from MCTD is excellent and equivalent to that of the standard KD (Liu *et al*., 2013; Ymc, 2008; Neal *et al*., 2009).

3) Modified Atkins diet

- The MAD was introduced in a 2003 series of cases by Kossoff *et al*.

which dealt with the advantages of a less restrictive diet that was started while the patient was still in the hospital, did not require an initial fast, and did not have any restrictions on calories, protein, or water.

- This kind of KD is meant primarily for children whose guardians or doctors are unable to adhere to the standard KD, or for people who have behavioural concerns. It is less restricted and more pleasant. MAD gets about 65% of its calories from fat (Kossoff *et al*., 2008; Auvin, 2016).

4) Low Glycemic Index treatment

- In 2005, it was presented as a viable dietary solution for managing DRE (Pfeifer *et al*., 2005). The severe carbohydrate limitation of each of the KDs is relaxed with this dietary regimen.
- It raise blood sugar levels just slightly—can be eaten alongside high carbohydrate foods such as bread, rice, potatoes and bagels.
- An indicator of this food's tendency is to raise blood glucose levels and its glycemic index (Pfeifer *et al*., 2008; Muzykewicz *et al*., 2009).
- When comparing the recommended amount of a certain food to the equivalent quantity of reference glucose, the total area during the blood glucose response curve can be used to determine the GI of that particular food(Pfeifer *et al*., 2008)

1.6. Diets Related to Crohn's disease (CD)

- Any section of the gastrointestinal tract, from the mouth to the anus, can be affected by this chronic, progressive, and fatal granulomatous inflammatory bowel disease; however, the colon, perianal region, and end of the ileocaecal region are typically affected (Mills *et al*., 2011; Farraye *et al*., 2017; Nikolaus *et al*., 2007).
- Arthritis, eating disorders, uveitis, and rashes on the skin are the primary extra-intestinal signs, whereas bloody mucopurulent diarrhoea, abdominal discomfort, nausea, emesis, loss of weight, the perineal pain, and urge to defecate are among the digestive symptoms.
- Patients with CD commonly go through phases of clinical remission and relapse (Mills *et al*., 2011; Farraye *et al*., 2017; Nikolaus *et al*., 2007).

- A blend of biochemical, histological, clinical, endoscopic, and imagistic techniques. There is no gold standard for the diagnosis of Crohn's disease, yet criteria are used anyway.
- The following CD diagnostic standards have been set by the World Health Organisation**:** fissures and fistulas, as evidenced by clinical exam, radiologic studies, biopsy results, and removed specimens; transmural inflammatory processes, as demonstrated by radiologic studies, an endoscopy and eliminated specimens; inconsistent or divided lesions, as well as a cobblestone appearances or over time ulcer, as seen on radiologic studies, removed samples, and endoscopy; noncaseating granulomas, as evidenced by specimens that were excised and biopsy results; perianal issues following a medical evaluation (Bernstein *et al*., 2016; Travis *et al*., 2006; Loftus *et al*., 2004; Lovasz *et al*., 2013; Molodecky *et al*., 2012).
- However, after the first year of diagnosis, 15% of people with CD are subsequently diagnosed with ulcerative colitis (UC) (Travis *et al*., 2006; Loftus *et al*., 2004; Lovasz *et al*., 2013).

1.7. Low Fodmap Diet

- Patients with irritable bowel syndrome (IBS) frequently follow a low-"fermentable oligo-, di-, mono saccharides and polyols" (FODMAP) diet because, by restricting foods high in fructose, lactose, fructans, galactans, and polyols, it may lessen symptoms of cramping, diarrhoea, and bloating (Gibson *et al*., 2010).
- FODMAPs, also known as osmotic short-chain carbohydrates, are osmotic carbohydrates that absorb water which are also absorbed in the small intestine before being digested by bacteria in both the proximal and the distal large intestine.
- Gas is produced during this cycle, which may assist in partly clarifying the bloating and flatulence (Gibson *et al*., 2010; Iacovou *et al*., 2015; Staudacher *et al*., 2011).
- In a typical diet, 15–30 g of FODMAP should be consumed daily. Although the Low- FODMAP diet (LFD) was once known as "Avoidance Diet, "
- Current research also views it as a diagnostic tool for a number of gastrointestinal and non-gastrointestinal conditions (Gibson *et al*., 2010; Iacovou *et al*., 2015; Staudacher *et al*., 2011).
- Due to their strong osmotic forces, FODMAPs force the water into

the intestinal lumen. FODMAPs are rapidly and readily broken down by gut microbiota in the colon, which is followed by fermentation.

- This fermentation process produces more gas, which causes distention in the abdomen and diffuse pain in the abdomen(Barrett *et al*., 2010; Bellini *et al*., 2020; Gershon *et al*., 2007; Murray *et al*., 2014)

1.8. The Mediterranean diet (MD)

- It is defined as an elevated intake of foods based on plants, unprocessed cereals, vegetables, fruits, olive oil and legumes as the primary fat source.
- It also includes modest intake of animal items that aren't produced from fish, moderate to high consumption of dairy products (mostly in the form of of cheeses and yoghurt), and moderate to high consumption of fish (Khalili *et al*., 2020; Papada *et al*., 2020; Chicco *et al*., 2020).
- The diets of the Mediterranean countries differed according to many aspects such as location, economy, history, or religion; yet, it is believed that these diets are only slight variations of the MD diet (Khalili *et al*., 2020; Papada *et al*., 2020; Chicco *et al*., 2020).

1.9. The low-lactose diet (LLD)

- It is commonly used as a treatment for patients with long history of insufficient lactose digestion-related symptoms such as gas, bloating, diffuse abdominal discomfort, vomiting, and diarrhoea which do not respond well to conventional forms of therapy (Gudmand-Hoyer *et al*., 1970; Capristo *et al*., 2000; Southworth *et al*., 2018; Herfarth *et al*., 2014; Aziz *et al*., 2015).

In India, it can be difficult to find qualified dieticians and experienced healthcare professionals nearby to meet the specialised needs for particular conditions. In hospital settings, attending physicians regularly recommend therapeutic diets, which are developed by dieticians and challenging to get in home environments. Therapeutic diets are modified based on the needs for different macro- and micronutrients, taking into account the caloric requirements.

Anorexia, frailty, loneliness, self-pity, and many other confusing elements, such as cooking techniques, religious views, traditions, and customs of dietary habits, are among several possible causes for an imbalance in appetite; nonetheless, personal preferences are the most important component

to consider. To accept the sick person's behaviour and persuade them to follow the advised, unhealthy diet, we must have empathy and compassion (Gopalan *et al*., 2004; Therapeutic Diet 2018).

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Chapter - 2
Fever and Gout: Distinguishing Discomforts

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Chapter - 2

Fever and Gout: Distinguishing Discomforts

Moumita Dev

Introduction

Gout, a painful inflammatory arthritis caused by urate crystal deposition in joints, is a prevalent condition. While the cardinal symptom of gout is a sudden and severe inflammatory attack in a single joint, fever can be a confounding factor. This paper explores the relationship between gout and fever, examining potential causes for fever during a gout attack, diagnostic challenges, and treatment considerations. We delve into the inflammatory processes of gout and the immune response's role in triggering fever. Additionally, the paper discusses differential diagnoses that may mimic gout with fever and the importance of accurate diagnosis for optimal treatment. Finally, we explore the management of both gout flares and fever associated with gout, with a focus on anti-inflammatory medications and urate-lowering therapies.

Gout, characterized by recurrent episodes of acute inflammatory arthritis, affects millions worldwide. The culprit behind these painful attacks is the buildup of uric acid crystals in joints, primarily the first metatarsophalangeal joint (big toe) but potentially affecting other joints as well. While the hallmark symptom of gout is severe joint pain and inflammation, the presence of fever can add complexity to the diagnosis and management.

Gout arises from an imbalance in uric acid metabolism. Uric acid is a byproduct of purine breakdown in the body. Normally, the kidneys efficiently excrete uric acid in urine. However, in individuals with gout, uric acid levels become elevated (hyperuricemia). This hyperuricemia can lead to the formation of sharp, needle-like urate crystals within joints.

The presence of urate crystals triggers an inflammatory response mediated by white blood cells called neutrophils. Neutrophils engulf the crystals, releasing various inflammatory mediators such as cytokines and interleukin-1β (IL-1β). These mediators cause swelling, redness, pain, and warmth in the affected joint – the classic symptoms of a gout attack [Kelley & Firestein, 2017].

Gout and fever are distinct conditions, but both are associated with an increase in body temperature. Gout is a specific inflammatory condition that affects joints, while fever is an essential part of the immune response. The purpose of this paper is to examine fever and gout from a mechanism, clinical presentation, and differentiation strategy perspective.

Fever: The Body's Defense Mechanism

Physiology of Fever

Generally, the hypothalamus acts as the body's thermostat, maintaining a tightly regulated core temperature. The hypothalamus is stimulated to raise the body's set point temperature when pyrogens, such as bacterial toxins or inflammatory cytokines, are released by immune cells during an infection [Dinarello, 2004].Immune cells are responsible for meditating this process by producing prostaglandin E2 (PGE2) which acts on the hypothalamus [Yu & Krueger, 2018].A physiological response is triggered, including**:**

- **Shivering:** Involuntary muscle contractions generate heat.
- **Increased metabolic rate:** The body burns fuel at a faster pace to produce heat.
- **Vasoconstriction:** Blood vessels in the skin constrict, reducing heat loss.

Benefits of Fever

As part of the body's immune response, fever creates an environment that is unfavorable to pathogens**:**

- **Inhibition of bacterial growth:** An elevated temperature can hinder the replication of bacteria, slowing their spread [Dinarello, 2004].
- **Enhanced immune cell activity:** Phagocytes, white blood cells that engulf and destroy pathogens, become more efficient at higher temperatures [Yu & Krueger, 2018].
- **Increased production of immune factors:** Fever stimulates the production of antibodies and other immune molecules that help fight infection [Yu & Krueger, 2018].

Clinical Significance of Fever Patterns

Fever patterns can help determine what is causing the disease

- **Sustained fever:** An elevated body temperature marked by a persistent rise might indicate an infection caused by bacteria, virus, or inflammation.

- **Spiking fever:** Periodic, rapid rises in temperature followed by dips can be associated with infections like malaria or sepsis [Frier, 2016].
- **Pel-Ebstein fever:** This biphasic pattern, with two distinct peaks, is characteristic of certain lymphomas or Hodgkin's disease [Kelley *et al*., 2014].

Management of Fever

While fever signifies the body's defense mechanism, it can cause discomfort. The primary aim in managing fever is to ensure patient comfort and prevent complications like dehydration or seizures, particularly in children.

Common management strategies include

- **Antipyretics:** Medications like acetaminophen or ibuprofen help lower body temperature.
- **Hydration:** Encouraging fluid intake is crucial to prevent dehydration.
- **Cooling measures:** Lukewarm baths or cooling cloths can help dissipate heat.

Gout: A Painful Joint Inflammation

Pathophysiology of Gout

Gout is a recurrent inflammatory arthritis characterized by sudden and severe pain, swelling, redness, and tenderness in joints, most commonly affecting the big toe. The culprit behind this condition is uric acid, a by product of purine metabolism.

Two key factors contribute to the development of gout:

- **Hyperuricemia:** High blood levels of uric acid. Increased purine intake, decreased kidney excretion of uric acid, or the combination of both can cause this [Khanna *et al*., 2020].
- **Urate crystal formation:** Urate crystal formation occurs when uric acid levels exceed its solubility limit, which leads to crystallization and a subsequent deposit within joints. The crystals cause an inflammatory response, resulting in gout symptoms.

Clinical Presentation of Gout

Gout attacks usually occur at night and cause excruciating pain, swelling, redness, warmth, and tenderness in the affected joint, which is usually the big toe (metatarsophalangeal joint).It can also affect joints such as ankles, knees,

wrists, and elbows. A typical attack lasts between three and seven days and may resolve spontaneously. If recurrent gout attacks are not managed properly, they can become more severe and frequent, potentially resulting in joint damage and deformities.

Management of Gout

The management of gout aims to

- **Modulate hyperuricemia:** Allopurinol and febuxostat reduce the production of uric acid. Additionally, uricosurics can be used to increase kidney excretion of uric acid.
- **Manage acute gout attacks:** Nonsteroidal anti-inflammatory drugs (NSAIDs), colchicine, or corticosteroids can be used to reduce inflammation and pain during acute attacks.
- **Prevent future attacks:** Lifestyle modifications, such as dietary changes to reduce purine intake and maintaining a healthy weight, combined with long-term uric acid-lowering medications, are crucial to preventing recurrent attacks and joint damage.

Potential Mechanisms of Fever in Gout

While fever is not a typical symptom of gout, it can occur in some cases. Several potential mechanisms may explain this phenomenon**:**

- **Intense Inflammatory Response:** The surge of inflammatory mediators released by neutrophils during a gout attack can trigger the body's thermostat, leading to a rise in core temperature and a fever [Neogi, 2011].
- **Secondary Infection:** Sometimes, the inflamed joint during a gout attack can become secondarily infected with bacteria. This infection can then lead to fever, chills, and other systemic symptoms [Khanna *et al*., 2012].
- **Underlying Autoimmune Disease:** Certain autoimmune diseases can cause both gout and a predisposition to fever. For instance, some patients with psoriatic arthritis (a type of inflammatory arthritis) may have elevated uric acid levels and experience gout flares with fever [Taylor *et al*., 2006].

Diagnostic Challenges: Differentiating Gout with Fever

The presence of fever in conjunction with joint pain can make diagnosing gout more challenging. Several other conditions can present with similar symptoms, requiring meticulous evaluation**:**

- **Septic Arthritis:** Bacterial or other infections within the joint can mimic gout with severe pain, swelling, and fever. However, septic arthritis tends to affect a wider range of joints and may present with additional symptoms like redness and warmth around the entire joint, unlike the localized nature of gout pain [Khanna *et al*., 2012].
- **Pseudogout:** This form of arthritis arises from the deposition of calcium pyrophosphate dihydrate (CPPD) crystals in joints. Like gout, pseudogout can cause sudden joint pain and swelling, but fever is less common [McCarty, 1992].
- **Inflammatory Bowel Disease (IBD):** Active IBD can sometimes cause a type of arthritis called peripheral arthritis. This arthritis can present with joint pain and fever, making differentiation from gout crucial [Loftus Jr., 2009].

Risk Factors and Complications: A Deeper Dive

Expanding on risk factors and complications of gout and fever individually, and their potential interaction, can provide a more comprehensive picture**:**

- **Risk Factors for Fever in Gout:** Discuss the potential influence of age, comorbidities like diabetes, and previous gout attacks with fever on the likelihood of recurrent fever.
- **Complications of Untreated Gout:** Elaborate on the long-term consequences of untreated gout, including joint damage, tophi formation, and kidney complications.
- **Complications of Untreated Fever:** Discuss the potential risks associated with persistent or high fever, such as dehydration, seizures, and organ damage.

Treatment Strategies for Gout and Fever

Management of fever and gout involves addressing both

- **Controlling the Gout Flare:** Controlling the gout**:** Medications that reduce inflammation are the most effective way to manage acute flares of gout. Inflammation, pain, and swelling can be reduced with these**:**
    - **Nonsteroidal Anti-inflammatory Drugs (NSAIDs):** These are usually the first line of treatment for gout flare-ups. Among them are ibuprofen, naproxen, and celecoxib.
    - **Corticosteroid:** A severe gout attack can be treated quickly with injected or oral corticosteroids to reduce inflammation and pain.

- **Colchicine:** This medication can shorten flares, especially if taken early in the flare. The medication is not suitable for everyone due to potential side effects. [Neogi *et al*., 2010].

- **Management of Fever:** Fever treatment depends on the underlying cause. Anti-inflammatory medications used to control the flare of gout may also help reduce fever if the fever is caused by an intense inflammatory response. The infection and fever will need to be treated with antibiotics if a secondary infection is suspected.
- **Long-Term Management: Preventing Future Flares and Fevers:** To prevent future flares and to possibly reduce the risk of fever associated with taste attacks, long-term urate-lowering therapy is recommended once a taste diagnosis has been confirmed. The effects of medications like allopurinol and febuxostat include decreased production or increased excretion of uric acid, reducing the risk of crystal formation and subsequent attacks.

Additional Considerations for Patients with Fever and Gout

- **Hydration:** The key to surviving a gout attack with fever is to maintain adequate hydration. By staying hydrated, uric acid can be flushed out and kidney stones can be prevented.
- **Rest and Joint Protection:** Resting the affected joint and applying ice packs can help reduce inflammation and pain.
- **Dietary Modifications:** Modifications in the diet can help lower uric acid levels in the long run by following a gout-friendly diet that limits purine intake. The treatment of acute gout flares with fever requires more than dietary changes.

Conclusion

Gout and fever are two distinct medical conditions. Fever is not a typical symptom of gout, but its presence can make diagnosis more difficult. It is crucial to differentiate between a gout flare and other potential causes of fever in gout patients with a comprehensive medical history, physical examination, and appropriate diagnostic tests. Gout can be effectively managed with medications, supportive measures, and long-term preventive strategies with an accurate diagnosis. In this way, the patient will be more comfortable and complications will be reduced.

Future Directions

In order to minimize the occurrence of fever during gout attacks, research

into gout continues to explore novel treatment options. The following areas are promising**:**

- **Development of Novel Urate-Lowering Therapies:** New medications with improved efficacy and fewer side effects are being investigated.
- **Targeting Specific Inflammatory Pathways:** Exploring the role of specific inflammatory pathways in gout may lead to more precise treatment strategies.
- **Gut Microbiome and Gout:** Investigating the link between gut microbiome and uric acid levels may potentially lead to dietary or probiotic interventions.

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Chapter - 3
Renal Diseases and Dietary Modifications

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Chapter - 3

Renal Diseases and Dietary Modifications

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Introduction

Acute kidney injury (AKI), chronic kidney disease (CKD), and end-stage renal disease (ESRD) are among the ailments that fall within the category of renal diseases (Hsu and Hsu, 2016). Acute Kidney Injury is a dangerous illness that needs to be treated right away (Levey and James, 2017). Chronic kidney disease is often brought on by diabetes, hypertension, glomerulonephritis, and polycystic kidney disease, and it is often accompanied by a progressive loss of kidney function over a long period of time (Pyram *et al*., 2012). End-Stage Renal Disease is the final stage of Chronic Kidney Disease where the kidneys no longer function adequately to meet the body's needs, necessitating dialysis or a kidney transplant (Webster *et al*., 2017). Dietary changes are essential for the treatment of renal illnesses because they can reduce the rate of disease progression, control symptoms, and enhance patient outcomes (Tewari, 2019). Effective patient management requires a thorough understanding of the pathophysiology of renal disorders as well as customised dietary plans. This chapter explores the dietary strategies employed to manage these conditions.

1. Pathophysiology of Renal Diseases

1.1. Acute Kidney Injury (AKI): Causes and Symptoms

The primary symptom of AKI is an abrupt reduction in kidney function, which causes waste products to build up and electrolyte abnormalities. Nephrotoxins, sepsis, and ischemia are common causes. If treated quickly, AKI can be cured; however, severe instances can lead to CKD (Ronco *et al*., 2019).

1.1.1. Causes of Acute Kidney Injury

Acute Kidney Injury (AKI) happens when the kidneys' capacity to remove waste from the blood is abruptly compromised. This may result in an unsafe accumulation of waste materials as well as an electrolyte and fluid imbalance. Prerenal, intrinsic, and postrenal are the three primary categories of causes of AKI (Kellum *et al*., 2021).

Prerenal Causes (Conditions that reduce blood flow to the kidneys)**:**

- **Dehydration:** Severe loss of fluids from vomiting, diarrhoea, or inadequate fluid intake.
- **Blood Loss:** Significant bleeding, leading to low blood volume.
- **Heart Failure:** Reduced cardiac output affecting blood flow to the kidneys.
- **Septic Shock:** Severe infection causing systemic vasodilation and hypotension.
- **Severe Burns:** Fluid loss and increased metabolic demand impacting renal perfusion (Gloviczki *et al*., 2010).

Intrinsic Causes (Direct damage to the kidneys)

- **Acute Tubular Necrosis (ATN):** Caused by ischemia or nephrotoxins, leading to damage to the renal tubules.
- **Glomerulonephritis:** Inflammation of the glomeruli, often due to autoimmune diseases or infections.
- **Acute Interstitial Nephritis:** Inflammation of the kidney interstitium, often triggered by drugs or infections.
- **Vascular Diseases:** Conditions such as vasculitis or malignant hypertension causing damage to kidney blood vessels.
- **Toxins:** Exposure to nephrotoxic substances like certain antibiotics, radiographic contrast media, or heavy metals.

Postrenal Causes (Obstruction of urine flow)

- **Urinary Tract Obstruction:** Blockages such as kidney stones, tumors, or strictures in the ureters, bladder, or urethra.
- **Prostatic Hyperplasia:** Enlarged prostate causing obstruction of the urinary outflow in men.
- **Neurogenic Bladder:** Nerve damage leading to bladder dysfunction and urinary retention (Madersbacher *et al*., 2012).

1.1.2. Symptoms of Acute Kidney Injury (AKI)

The symptoms of AKI can vary depending on the severity and underlying cause, but common signs and symptoms include**:**

- **Reduced Urine Output:** A significant decrease in the amount of urine produced, although occasionally urine output may remain normal (Friedrich *et al*., 2005).

- **Fluid Retention:** Swelling in the legs, ankles, or feet due to fluid build-up.
- **Fatigue:** Feeling excessively tired or weak due to toxin build-up in the blood.
- **Shortness of Breath:** Fluid accumulation in the lungs can cause difficulty breathing.
- **Confusion:** Mental confusion or decreased alertness as a result of the build-up of waste products and electrolyte imbalances.
- **Nausea and Vomiting:** Gastrointestinal symptoms can occur due to toxin build-up.
- **Chest Pain or Pressure:** This can occur if fluid builds up around the heart.
- **Severe Cases:** Symptoms may progress to seizures or coma if the condition is not treated promptly.

Acute Kidney Injury is a dangerous illness that needs to be treated right away (Lewington *et al*., 2013). For an early diagnosis and successful treatment, it is essential to understand the causes and symptoms of AKI (Endre and Westhuyzen, 2008). Seek medical attention right away if you encounter any of the aforementioned symptoms, particularly if there is a known risk factor or underlying ailment (Doran *et al*., 2023). Prompt intervention can enhance results and lower the chance of chronic kidney injury (Ladin *et al*., 2023).

1.2. Chronic Kidney Disease (CKD): Causes and Symptoms

Gradual loss of kidney function over months or years is known as chronic kidney disease (CKD), and it is frequently brought on by diabetes, hypertension, glomerulonephritis, and polycystic kidney disease (Akinbodewa, 2023). Based on the glomerular filtration rate (GFR), there are five stages of chronic kidney disease (CKD) (Gama *et al*., 2023). Cardiovascular disease risk rises with the progression of chronic kidney disease (CKD), and complications such anaemia, mineral and bone abnormalities, and acidosis occur (Elendu *et al*., 2023).

1.2.1. Causes of Chronic Kidney Disease (CKD)

Chronic Kidney Disease (CKD) is a long-term condition where the kidneys gradually lose function over time (Webster *et al*., 2017). This progressive loss of function can eventually lead to end-stage renal disease (ESRD), requiring dialysis or a kidney transplant. The primary causes of CKD include**:**

Diabetes Mellitus

- **Type 1 and Type 2 Diabetes:** High blood sugar levels over time can damage the blood vessels in the kidneys, leading to diabetic nephropathy.

Hypertension (High Blood Pressure)

- **Chronic High Blood Pressure:** Can cause damage to the blood vessels and filtering units in the kidneys, impairing their ability to function properly (Appel *et al*., 2010).

Glomerulonephritis

- **Inflammation of the Glomeruli:** Can be caused by infections, autoimmune diseases, or other inflammatory conditions, leading to kidney damage (Webster *et al*., 2017).

Polycystic Kidney Disease (PKD)

- **Genetic Disorder:** Characterized by the growth of numerous cysts in the kidneys, which can interfere with normal kidney function.

Chronic Obstructive Uropathy

- **Long-term Blockages:** Conditions such as kidney stones, enlarged prostate, or tumors can obstruct urine flow and lead to kidney damage over time (Truong *et al*., 2011).

Recurrent Urinary Tract Infections (UTIs)

- **Frequent Infections:** Can cause scarring and damage to the kidneys, especially if untreated or improperly managed.

Autoimmune Diseases

- **Lupus Nephritis:** Lupus and other autoimmune conditions can lead to chronic inflammation and damage to the kidneys.

Medications and Toxins

- **Nephrotoxic Drugs:** Long-term use of certain medications, such as nonsteroidal anti-inflammatory drugs (NSAIDs), some antibiotics, and chemotherapy agents, can damage the kidneys (Clavé *et al*., 2019).
- **Environmental Toxins:** Exposure to heavy metals and other toxic substances can also contribute to CKD (Jalili *et al*., 2021).

Genetic Predisposition

- **Family History:** A family history of kidney disease can increase the risk of developing CKD (Webster *et al*., 2017).

Other Medical Conditions

- **Heart Disease:** Cardiovascular conditions can impair kidney function.
- **Obesity:** Increases the risk of conditions like diabetes and hypertension, which are major risk factors for CKD.

1.2.2. Symptoms of Chronic Kidney Disease (CKD)

CKD often progresses slowly and may not cause noticeable symptoms until the disease is advanced (Levey and Coresh, 2012). Common signs and symptoms include**:**

- **Fatigue:** Persistent tiredness and lack of energy due to the buildup of waste products in the blood.
- **Swelling (Edema):** Fluid retention leading to swelling in the legs, ankles, feet, and sometimes the face and hands.

Changes in Urination

- **Frequency:** Increased or decreased urination, especially at night (nocturia).
- **Appearance:** Urine may be foamy, dark, or contain blood.
- **Volume:** Decrease in urine output.
- **Shortness of Breath:** Fluid build-up in the lungs or anemia caused by reduced erythropoietin production can cause difficulty breathing.
- **High Blood Pressure:** Poorly controlled hypertension can be both a cause and a symptom of CKD.
- **Nausea and Vomiting:** Accumulation of waste products can lead to gastrointestinal symptoms.
- **Loss of Appetite:** Reduced desire to eat, which can lead to weight loss.
- **Metallic Taste in Mouth:** Uremia can cause a bad taste in the mouth and bad breath.
- **Muscle Cramps and Twitching:** Electrolyte imbalances, particularly low calcium and high phosphate levels.
- **Itching:** Accumulation of waste products in the blood can cause severe itching.

- **Cognitive Changes:** Difficulty concentrating, memory problems, or confusion due to toxin build-up.
- **Chest Pain:** If fluid builds up around the lining of the heart (pericarditis).

In order to minimise complications and decrease the advancement of Chronic Kidney Disease, early detection and management are crucial (El Nahas and Bello, 2005). This is a serious and progressive condition. Recognising the symptoms and comprehending the causes are essential for prompt response. Chronic kidney disease (CKD) can be avoided or postponed by closely monitoring and treating underlying medical problems like diabetes and hypertension. For an accurate diagnosis and course of treatment, speak with a healthcare provider if you exhibit any CKD symptoms (Ladin *et al*., 2018).

1.3. End-Stage Renal Disease (ESRD): Causes and Symptoms

When kidney function falls below 15% of normal, the condition is known as end-stage renal disease, or ESRD. Dialysis or kidney transplants are necessary for ESRD patients to survive. It takes careful dietary management to avoid complications in the complicated management of end-stage renal disease (ESRD) (Murtagh *et al*., 2007).

1.3.1. Causes of End-Stage Renal Disease (ESRD)

End-Stage Renal Disease (ESRD) is the final stage of chronic kidney disease (CKD) where the kidneys no longer function adequately to meet the body's needs, necessitating dialysis or a kidney transplant. ESRD is typically the result of progressive damage from various underlying conditions**:** (Shayamsunder *et al*., 2005)

Diabetes Mellitus

- **Diabetic Nephropathy:** Persistent high blood sugar levels cause long-term damage to the kidney's filtering units (Raptis and Viberti, 2001).

Hypertension (High Blood Pressure)

- **Hypertensive Nephropathy:** Chronic high blood pressure damages the kidney's blood vessels, reducing their ability to filter blood effectively (Seccia *et al*., 2017).

Chronic Glomerulonephritis

- **Inflammation of the Glomeruli:** Long-standing inflammation due

to infections, autoimmune diseases, or other conditions progressively damages the kidney's filtering system (Moeller *et al*., 2004).

Polycystic Kidney Disease (PKD)

- **Genetic Disorder:** Numerous cysts form in the kidneys, gradually impairing their function over time.

Chronic Obstructive Uropathy

- **Long-term Obstructions:** Conditions like kidney stones, tumors, or an enlarged prostate can cause chronic blockages, leading to kidney damage.

Chronic Pyelonephritis

- **Recurrent Kidney Infections:** Persistent infections cause scarring and deterioration of kidney tissue (Mitra and Alangaden, 2011).

Autoimmune Diseases

- **Lupus Nephritis and Other Conditions:** Autoimmune diseases can cause chronic inflammation and damage to kidney tissue.

Medications and Toxins

- **Nephrotoxic Drugs:** Long-term use of certain medications (e.g., NSAIDs, some antibiotics, chemotherapy agents) and exposure to environmental toxins can lead to chronic kidney damage.

Interstitial Nephritis

- **Chronic Inflammation:** Persistent inflammation of the kidney interstitium, often due to long-term use of medications or chronic infections.

Other Conditions

- **Obesity, Heart Disease:** Conditions that increase the risk of hypertension and diabetes can indirectly contribute to the progression to ESRD.
- **Genetic Factors:** Family history of kidney disease increases the risk.

1.3.2. Symptoms of End-Stage Renal Disease (ESRD)

As ESRD progresses, the symptoms become more severe and systemic, reflecting the kidneys' inability to filter waste products and balance fluids and electrolytes (Murtagh *et al*., 2007)

- **Severe Fatigue:** Extreme tiredness due to anemia and the build-up of waste products.

- **Persistent Swelling (Edema):** Pronounced swelling in the legs, ankles, feet, and sometimes the face and hands due to fluid retention.
- **Decreased or No Urine Output:** Significant reduction in urine production or complete cessation of urine output.
- **Shortness of Breath:** Difficulty breathing due to fluid build-up in the lungs (pulmonary edema) or anemia.
- **Severe Nausea and Vomiting:** Intense gastrointestinal symptoms due to toxin accumulation.
- **Loss of Appetite and Weight Loss:** Decreased desire to eat and unintended weight loss.
- **Metallic Taste in Mouth:** Uremia causes a persistent bad taste and bad breath.
- **Severe Muscle Cramps and Twitching:** Electrolyte imbalances, particularly due to high potassium levels.
- **Severe Itching (Pruritus):** Persistent and severe itching due to the accumulation of waste products in the blood.
- **Confusion and Difficulty Concentrating:** Mental confusion, difficulty concentrating, and memory problems due to toxin build-up.
- **Chest Pain or Pressure:** Pain or pressure in the chest if fluid builds up around the heart (pericarditis).
- **Bone Pain and Fractures:** Weakened bones due to imbalances in calcium and phosphate levels.
- **High Blood Pressure:** Elevated blood pressure that may be difficult to control.
- **Sleep Disturbances:** Insomnia, restless legs syndrome, and other sleep issues.

The most advanced stage of chronic kidney disease, known as end-stage renal disease (ESRD), is marked by the kidneys' incapacity to perform their normal functions (Kher, 2002). Long-term damage from illnesses such diabetes, hypertension, glomerulonephritis, and polycystic kidney disease frequently leads to the progression to end-stage renal disease (ESRD) (Zhong *et al*., 2017) Severe and systemic symptoms of end-stage renal disease (ESRD) impairs several bodily systems and have a major negative influence on quality of life. Preventing or postponing the development of end-stage renal disease (ESRD) requires early identification and treatment of underlying problems.

Treatment options for ESRD patients include kidney transplantation or dialysis.

2. Dietary Modifications in Renal Diseases

2.1. Protein Management

Protein intake must be carefully managed in renal disease to balance the need for protein synthesis and the burden of nitrogenous waste (Ko and Kalantar-Zadeh, 2021). Protein requirements may increase in Acute Kidney Disease due to catabolism, especially in critically ill patients (Sabatino *et al.*, 2017). However, excessive protein can exacerbate uremia. A low-protein diet (0.6-0.8 g/kg body weight) is recommended to reduce the production of uremic toxins and slow disease progression. In stages 4 and 5 Chronic Kidney Disease, protein intake may be further restricted. Dialysis patients often require higher protein intake (1.2-1.5 g/kg body weight) in E End-Stage Renal Disease (ESRD) to compensate for protein losses during treatment (Heng and Cano, 2010).

2.2. Sodium Restriction

Restricting sodium consumption can help control hypertension and fluid retention, two major problems associated with renal impairment. In AKD, sodium restriction aids in the management of hypertension and edoema. For people with CKD, a daily salt intake of no more than 2, 300 mg is advised. When a patient has End-Stage Renal Disease (ESRD), their sodium intake should be customised based on their dialysis schedule and remaining renal function (Ellison, 2017).

2.3. Potassium Management

Since hyperkalemia is a serious consequence of renal illness, maintaining a balance of potassium is essential. When AKD is present, potassium intake needs to be regularly monitored and adjusted in accordance with serum levels. In CKD, potassium restriction (2, 000–3, 000 mg/day) is recommended to avoid hyperkalemia. Patients on dialysis must restrict their potassium intake since their kidneys are not able to properly eliminate excess potassium (Cupisti *et al.*, 2018).

2.4. Phosphorus and Calcium Control

Mineral and bone abnormalities associated with renal illness are partly caused by calcium and phosphorus imbalances. In AKD, phosphorus levels need to be tracked and managed, frequently with the aid of phosphate binders. Phosphorus restriction (800–1, 000 mg/day) and phosphate binders are two strategies used to control hyperphosphatemia in CKD patients. A balanced

calcium intake is necessary to prevent hypercalcemia. Along with dietary modifications and prescriptions specific to the patient's dialysis treatment plan, phosphorus restriction and calcium management are essential (Ash *et al*., 2014).

2.5. Fluid Management

Maintaining fluid balance is crucial to avoiding dehydration and overhydration. In AKD, fluid intake needs to be carefully controlled; it should frequently meet production plus insensible losses. A patient's specific fluid intake should be determined by their level of edoema and urine production. In order to avoid fluid overload, fluid intake is usually limited, with limitations depending on the kind of dialysis (Zoccali *et al*., 2017).

2.6. Micronutrient Considerations

Renal losses and dietary restrictions might result in vitamin and mineral deficiencies. Water-Soluble Vitamins**:** Due to losses during treatment, patients with renal illness, especially those receiving dialysis, may need to take supplements of water-soluble vitamins (e.g., B vitamins, vitamin C). Fat-Soluble Vitamins**:** Because of the possibility of toxicity and insufficiency, careful observation and supplementation of vitamins A, D, E, and K are required (Nowak *et al*., 2022).

2.7. Special Diets

Renal Diet**:** A specially designed diet with an emphasis on controlling the intake of liquids, protein, sodium, potassium, and phosphorus. Mediterranean Diet**:** New research indicates that CKD patients may benefit from a diet modelled after the Mediterranean, as it may lower cardiovascular risk and inflammation (Chauveau *et al*., 2018).

2.8. Practical Considerations and Patient Education

Effective dietary management in renal disease requires collaboration between healthcare providers and patients. Key strategies include**:**

- Individualized Nutrition Plans**:** Personalized dietary recommendations based on the stage of renal disease, comorbid conditions, and patient preferences.
- Patient Education**:** Empowering patients with knowledge about food choices, label reading, and meal planning.
- Monitoring and Follow-up**:** Regular assessment of nutritional status, laboratory parameters, and dietary adherence.

3. Conclusion

Dietary changes are essential for the treatment of renal illnesses because

they can reduce the rate of disease progression, control symptoms, and enhance patient outcomes. Effective patient management requires a thorough understanding of the pathophysiology of renal disorders as well as customised dietary plans. In order to achieve the best outcomes, patients and healthcare providers must work together.

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Chapter - 4
Diagnosis, Dietary Treatment and Prevention of Nutritional Anaemia

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Chapter - 4

Diagnosis, Dietary Treatment and Prevention of Nutritional Anaemia

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Introduction

Anaemia, derived from the ancient Greek term ἀναιμία, anaimia, which means "lack of blood," is characterised by a decrease in the total amount of red blood cells, or haemoglobin (Johnson-Wimbley and Graham, 2011). This results in an inadequate supply of oxygen to fulfil the body's physiological requirements and can be brought on by a variety of factors, such as dietary inadequacies. While there are other conditions that can cause anaemia, such as deficiencies in folate, vitamin B12 and A, chronic inflammation, parasite infections, and genetic abnormalities, iron deficiency is thought to be the most common cause globally (WHO, 2018).

Anaemia is a condition in which the body's need for oxygen is not met by the red blood cells' (RBCs) ability to carry it. Red blood cells carry carbon dioxide from the tissues to the lungs and oxygen from the lungs to the tissues. This is accomplished by the usage of haemoglobin (Hb), a tetramer protein consisting of both haem and globin. Anaemia impairs the body's ability to exchange gases because fewer red blood cells can deliver carbon dioxide and oxygen. Anaemia can result from reduced red cell production, increased red cell lysis, or blood loss, among other events. Iron is needed for haemoglobin synthesis.

The most common cause of anaemia worldwide is believed to be iron deficiency. Nevertheless, anaemia can also result from other nutritional deficiencies (like those pertaining to folate, vitamin B12, and vitamin A), acute and chronic inflammation, parasitic infections, and hereditary or acquired disorders that impact haemoglobin synthesis, red blood cell production, or red blood cell survival. Children with iron deficiency anaemia have delayed cognitive and motor development, while adults with iron deficiency anaemia have reduced work capability (Figure 1.1). Early life and infancy are the most affected. Iron deficiency anaemia during pregnancy can cause preterm birth, low birth weight (LBW) new-borns, and perinatal loss.

The body's immunological response is also negatively impacted by iron deficient anaemia.

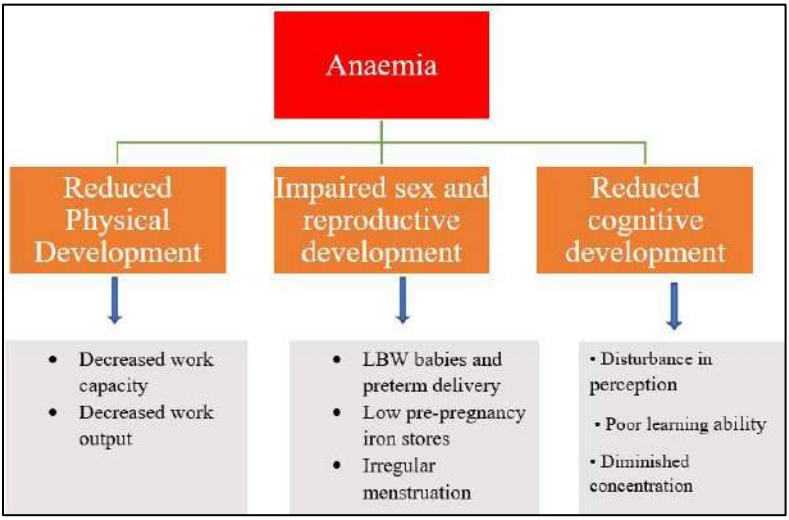

Fig 1.1: Effect of Anaemia

Prevalence of Anaemia

The prevalence of anaemia is estimated to be 25% globally based on data from almost half of the world's population in the WHO Global Database on Anaemia for 1993–2005. According to McLean *et al.* (2009), anaemia is believed to be 9% prevalent in highly developed countries but 43% common in less developed ones. An estimated 293 million preschool-aged children, 56 million pregnant women, and 468 million non-pregnant women worldwide are affected by anaemia.

According to McLean *et al.* (2009), the most vulnerable groups are children and women who are of reproductive age. The prevalence of anaemia is estimated to be 47% in children under five, 42% in pregnant women, and 30% in non-pregnant women between the ages of 15 and 49 worldwide. Most the world's anaemia cases are in India, with about 85% of cases in high-risk categories coming from Africa and Asia. (Table 1).

Table 1: Prevalence of anaemia in India and neighbouring countries

Country	Proportion of population with anaemia (Hb)	Public health problem
Bangladesh	47	Severe
Bhutan	80.6	Severe
India	74.3	Severe

Nepal	78	Severe
Pakistan	50.9	Severe
Sri Lanka	29.9	Moderate

According to WHO (2004), anaemia is thought to be a contributing factor in around 115, 000 maternal and 591, 000 perinatal deaths worldwide each year. Analysing global prevalence data reveals that poor socioeconomic groups are disproportionately home to anaemia cases, and there is a clear correlation between maternal anaemia and child anaemia.

Prevalence of Anaemia in India

India is among the nations where anaemia is most common worldwide, anaemia affects approximately 58% of expectant mothers, and it is thought to be the primary factor in 20–40% of maternal fatalities. About 80% of South Asian maternal fatalities from anaemia are attributable to India (Ezzati *et al.*, 2002).

The main cause of nutritional anaemia, which is a major public health concern in India and is particularly common among the most vulnerable populations, such as pregnant women (nearly 58%), non-pregnant, non-lactating women (50%) adolescent girls (15–19 years old), adolescent boys (30%), and children under three (about 80%), is iron deficiency, according to data from the National Family Health Survey-3 (NFHS-3). (Table 2) (NNMBS, 2006).s

Table 2: Prevalence of anaemia among different age groups According to the NNMBS, 2006

Age groups	Prevalence of anaemia (%)
6–35 months Children	79
6–59 months Children	69.5
15–49 years aged All women	55.3
15–49 years aged Ever married women	56
Pregnant women (15–49 years)	58.7
Lactating women (15–49 years)	63.2
Adolescent Girls 12–14 years	68.6
Adolescent Girls 15–17 years	69.7
Adolescent Girls 15–19 years	55.8

Types of Anaemia

A disorder known as anaemia occurs when the body lacks sufficient amounts of healthy red blood cells. Your body's tissues receive oxygen from

red blood cells. Anaemia comes in a variety of forms, each having a unique etiology.

Here are some of the most common types of anaemia

Iron-deficiency anaemia: Iron-deficiency anaemia is the most common kind of anaemia. Its underlying cause is an iron shortage. Only iron can produce haemoglobin, the protein in red blood cells that carries oxygen. An inadequate diet, certain medical conditions, or blood loss can all lead to iron-deficiency anaemia. The most common kind of nutritional anaemia, iron-deficiency anaemia (IDA), is estimated to account for over 50% of all cases of anaemia by the World Health Organization (2015). It arises from a prolonged negative iron balance. When haemoglobin is below the normal range (Hb 12.0 mg/dL or a hemocrit below 36%), it is a severe stage of iron deficiency.

Iron deficiency anaemia is a severe health concern that affects people in both developed and developing countries. It can seriously hinder social and economic progress. It is more prevalent in women who are reproductive and in young children, however it can occur at any stage of life. WHO (2002) states that iron deficiency anaemia (IDA) is one of the primary causes of anaemia burden globally.

Adolescents around the world are dealing with a number of severe dietary issues that have an impact on both their adult livelihood and growth and development. Understanding the necessary consumption and bioavailability of iron during adolescence is crucial in explaining why certain individuals are susceptible to developing iron deficiency anaemia. A condition known as anaemia occurs when the body does not produce enough red blood cells to satisfy its needs. It can cause a girl to lose concentration at work and in the school, and it poses a major risk to her future ability to become a safe mother. Adequate nutrition, which includes consuming enough iron, is crucial for the growth and development of teenagers (WHO 2008).

Vitamin deficiency anaemia: Vitamin B12 or folate insufficiency can result in vitamin deficiency anaemia. These vitamins are necessary for the production of red blood cells. Vitamin B12 deficiency-related anaemia is more frequent in older adults and people with digestive problems. Anaemia caused by a folate deficiency is more likely in pregnant women and those with low fruit, vegetable, and whole grain intake.

There are two main types of vitamin deficiency anaemia

- **Vitamin B12 deficiency anaemia:** This kind of anaemia is brought

on by insufficient vitamin B12. Meat, chicken, fish, eggs, and dairy products are examples of animal products that contain vitamin B12. This kind of anaemia can occur in those who strictly adhere to a vegetarian or vegan diet and do not take vitamin B12 supplements.

- **Folate deficiency anaemia:** This type of anaemia is caused by a lack of folate, also known as folic acid. Folate is found in leafy green vegetables, fruits, and fortified grains. Pregnant women and women who are breastfeeding need more folate than usual.

Aplastic anaemia is a rare condition that occurs when your bone marrow doesn't make enough red blood cells. The cause of aplastic anaemia is often unknown, but it can be caused by certain medications, infections, or autoimmune diseases.

The exact cause of aplastic anaemia is often unknown (idiopathic). However, several factors can damage stem cells in the bone marrow, leading to the condition**:**

- **Viral infections:** Hepatitis, Epstein-Barr virus (EBV), parvovirus B19
- **Autoimmune diseases:** Lupus, Sjögren's syndrome
- **Exposure to certain toxins:** Heavy metals, pesticides, some medications (chloramphenicol)
- **Radiation therapy**
- **In some cases, inherited genetic conditions**

Haemolytic anaemia is a group of disorders that cause red blood cells to break down faster than they can be made. Haemolytic anaemia can be inherited or acquired. Inherited haemolytic anaemias are caused by genes that you inherit from your parents. Acquired haemolytic anaemias can be caused by infections, autoimmune diseases, or certain medications.

Haemolytic anaemia can be caused by inherited or acquired conditions.

- **Inherited haemolytic anaemia:** These are genetic conditions passed down from parents. Examples include sickle cell disease and thalassemia.
- **Acquired haemolytic anaemia:** These develop later in life due to various factors like**:**
  - Autoimmune problems**:** Your immune system attacks your own red blood cells.

- Medications or toxins**:** Certain medications or exposure to toxins can damage red blood cells.
- Infections**:** Some infections can destroy red blood cells.
- Blood transfusions**:** In rare cases, a mismatch in blood types during a transfusion can destroy red blood cells.

Sickle cell anaemia is an inherited blood disorder that causes red blood cells to be sickle-shaped. Sickle-shaped cells can block blood flow and cause pain, tissue damage, and other health problems.

- Normally, red blood cells are round and flexible, allowing them to navigate your blood vessels easily.
- In sickle cell anaemia, a genetic mutation alters haemoglobin, the protein in red blood cells responsible for oxygen transport.
- This abnormal haemoglobin causes red blood cells to become stiff and sickle-shaped, like crescent moons.

Thalassemia is a group of inherited blood disorders that cause the body to make less haemoglobin or abnormal haemoglobin. Thalassemia can range from a mild to a severe condition.

Causes of Anaemia

The main causes of iron deficiency anaemia in India include poverty, illiteracy, ignorance, and a lack of understanding of the condition, dietary needs, and the nutritional worth of various plants (Park, 2015). Inadequate consumption of iron is a common cause of iron-deficiency anaemia (IDA). Anaemia due to iron shortage does not appear right away. Rather, an individual develops an iron shortage over time, starting with iron depletion, a condition in which the body's iron stores decrease but the iron in red blood cells does not. Iron depletion leads to iron deficiency, which in turn causes IDA if left untreated. Iron deficiency anaemia is the result of our bodies not containing enough iron. A poor diet, blood loss, or an inability to absorb enough iron from food are the usual causes of low iron levels.

Blood loss

An iron deficiency results from blood loss. Iron-deficiency anaemia is the result of inadequate iron storage, which leaves the body unable to compensate for iron loss. Women who have low iron levels may have bleeding fibroids in the uterus or blood loss from protracted or heavy menstrual cycles. Another factor contributing to women's low iron levels is blood loss after childbirth. Anaemia caused by iron deficiency can also result from internal hemorrhage,

or bleeding within the body. Blood loss of this kind might happen gradually and isn't always noticeable. Bleeding ulcers, colon polyps, and colon cancer are a few reasons for internal bleeding. Aspirin or other painkillers on a regular basis, without the use of NSAIDs (such ibuprofen and naproxen), urinary tract bleeding.

Poor diet

Meat, chicken, fish, eggs, and iron-fortified foods (i.e., foods with additional iron) are the finest sources of iron. A person is more prone to develop iron-deficiency anaemia if they don't consume these items on a regular basis or if they don't take an iron supplement. Iron deficiency anaemia is caused by a diet low in iron enhancers, such as meals high in vitamin C like citrus fruits. If the appropriate foods are consumed, vegetarian diets can supply adequate iron. For instance, dark green leafy vegetables like spinach and others, certain beans, dried fruits, and breads and cereals with added iron are good non-meat sources of iron.

Inability to absorb enough iron

Our bodies might not be able to absorb iron from our diet, even if there is enough of it there. This could be the result of gastrointestinal surgery or disorders like celiac disease or Cohn's disease that affect the intestines. Medication used to lower stomach acid can also affect how well iron is absorbed.

Risk Factors for Anaemia

Approximately 22, 000 of the more than one lakh women who die in India due to pregnancy-related causes do so from nutritional anaemia, making anaemia the fifth most common cause of maternal fatalities. Mother fatalities are caused by severe anaemia in 20.3% of cases. Compared to non-anaemic women, anaemic women have a five to ten-fold increased chance of dying from infection and haemorrhage. Women who suffer from anaemia also put their unborn children at risk for intrauterine development retardation, low birth weight, which leads to perineal death, and irreversible brain damage. During the preschool years, when growth is rapid, anaemia is more prone to develop. period of adolescence marked by fast growth and iron loss during menstruation. During pregnancy, the foetus and the mother's tissues expand quickly.

Deficiency of Micronutrients in India

Worldwide health issues resulting in a decline in life quality and a more dire outlook for individuals with chronic illnesses are iron deficiency and iron

deficiency anaemia. Inflammation-related enhanced loss as well as impaired intestinal absorption and transport of iron reserves are typically the combined causes of iron shortage and anaemia. The primary line of treatment is oral iron; however, intolerance frequently makes it difficult. Patients with gastrointestinal disorders and chronic inflammation are best treated with intravenous iron, which is both safe and effective. Normalizing haemoglobin concentration and restoring iron reserves are the two main objectives of treatment. For patients who have chronic inflammation and/or iron loss, it is crucial to receive follow-up care to make sure these goals are achieved and to prevent anaemia from reoccurring (Dahlerup *et al*., 2015).

The body stores iron mostly as hemosiderin and ferritin. Iron is necessary to make haemoglobin. In the liver, spleen, and bone marrow, iron is retained as ferritin and hemosiderin to a degree of around 30%. Anaemia from iron shortage does not appear right away. Rather, an individual develops an iron shortage over time, starting with iron depletion, a condition in which the body's iron stores decrease but the iron content of red blood cells stays unchanged. Iron shortage finally results in iron deficiency anaemia if iron depletion is not treated (Camaschella, 2015).

The synthesis of myoglobin, cytochrome, cytochrome-oxidize, peroxidase, and catalase, among other molecules, depends on iron. The body contains approximately 4 grams of iron overall. The body should have between 65% and 68% of its iron in haemoglobin, 4% in myoglobin in muscle, 1% in different hem compounds involved in intracellular oxidation, 0.1% in transferrin plasma, and 25% to 30% stored in ferritin in the reticuloendothelial system and liver (Sembulingam, 2015).

Iron insufficiency in school-age children is mostly caused by a diet lacking in nutrients, iron malabsorption, increased iron requirements during growth periods, menstrual blood loss, hookworm infestation, malaria, and other infectious disorders (WHO, 2016a). The main causes of the rising incidence of anaemia are poor diet quality and reduced dietary iron bioavailability. Compared to non-haem iron-containing food items, animal food products with haem iron have improved absorption rates of 20%–30%. The existence of boosting or inhibitory variables affects the bioavailability of non-haem iron. Meat (haem iron) and vitamin C are the primary factors that increase the absorption of non-haem iron. The following are examples of inhibitors**:** polyphenols (tea, coffee, chocolate, some spices, and vegetables), phytates (nuts, bran and oat products, whole-wheat and brown flour), calcium (milk products), and phosphorus (Walczyk *et al*., 2014).

The cells lining the gastrointestinal tract take in iron from meals, which is essential for many bodily processes. Only a small portion of the iron that is consumed is absorbed by the body. After digestion, iron in food is changed into the ferrous state, and only this type of iron is absorbed. In healthy individuals, the rate of dietary iron absorption is approximately 10%, whereas in those with iron deficiency anaemia, the rate of absorption is maximum (2% - 30%). Iron absorption requires bile. Both the upper jejunum and the duodenum are where iron absorption happens. Ferric iron is created when the enzyme ferroxidase oxidizes ferrous iron after absorption. Ferric iron then interacts with the mucosa-resident protein apoferritin to generate ferritin. The breakdown and reconstitution of apoferritin occurs during iron absorption. Iron absorption cannot occur further because of the high concentration of ferritin in the cell (De Gruchy‘s, 2010).

Despite particular initiatives including promoting the consumption of iron-rich foods through dietary changes, nutritional education, treatment and prevention of parasitic infections, and weekly iron supplementation, the impact of anaemia among adolescent girls remains a global public health concern (Getachew Mengistu, 2018).

The absorption of non-hem iron will be enhanced by vitamin C and meat proteins. Non-haem iron absorption can be slowed down by tannins (found in tea), calcium, polyphenols, and phytates (found in whole grains and legumes). Soybeans include some proteins that prevent the absorption of nonheme iron. When daily iron intake is lower than recommended, when iron losses are high (as they can be with heavy menstrual losses), when iron requirements are high (as they are during pregnancy), and when only vegetarian non-haem sources of iron are consumed, it is crucial to include foods that improve non-haem iron absorption (Rani, 2010).

Adolescence, along with other susceptible age groups including infancy and early childhood, is more likely to experience iron deficiency anaemia because of a combination of fast physical growth and girls' menstrual iron losses (Jadhav, 2016).

According to the National Family Health Survey (NFHS) (2011), approximately 20%-40% of maternal deaths in India are due to anaemia. In case of Indian scenario, India contributed to about 50% of global maternal deaths due to anaemia.

According to the World Health Organization (2015), iron-deficiency anaemia (IDA) accounts for almost 50% of all cases of anaemia and is the most prevalent kind of nutritional anaemia. It arises from a prolonged negative

iron balance. When haemoglobin is below the normal range (Hb 12.0 mg/dL or a hemocrit below 36%), it is a severe stage of iron deficiency.

Anaemia has negative repercussions on the body and society, including reduced physical and cognitive function, higher morbidity from infectious diseases, low birth weight, early delivery, and increased morbidity from infectious diseases (Hassan *et al*., 2016).

Anaemia also impairs one's ability to work physically. A lack of iron causes skeletal muscles' ability to use oxygen for respiration to decrease along with the iron-containing enzymes in the mitochondrial respiratory chain. An increased vulnerability to early exhaustion is linked to this decrease in aerobic metabolism. Consequently, it reduces work performance over extended periods of time as well as during intensive, brief exercise (Srivastava, 2016). According to a number of studies, children who weren't anaemic did better on the physical performance exam than children who were anaemic. Consequently, anaemia negatively impacts school-age children's ability to perform physical labour, particularly as they are entering the pubertal stage of development (Deli *et al*., 2013).

The most common nutritional issue in the world is anaemia, which is mostly brought on by an iron shortage. The years between 10 and 19 years old make up adolescence, a stage in human growth and development that comes after childhood but before adulthood. They will reach 25% of adult height and 50% of adult weight at this time. Adolescents have higher nutritional needs due to their rapid growth, which also increases their risk of anaemia. In particular, girls' iron requirements significantly increase with an increase in lean body mass, an increase in total blood volume, and the commencement of menstruation, making them more vulnerable to anaemia (UNICEF, 2011).

Initiative to Control Malnutrition

National Nutrition Mission the Government of India launched National Nutrition Mission (renamed as Poshan Abhiyaan) in March 2018.

- Its objectives are to decrease low birth weight, undernutrition, and anaemia (in young children, women, and adolescent girls) by 2%, 3%, and 2% annually, respectively. Moreover, it seeks to decrease stunning, a measure of malnutrition that is defined as a height that is noticeably below the norm for age, by 2% annually, bringing the population's percentage of stunted children down to 25% by 2022.
- The goal of the strategy is to map different programs that combat hunger and establish a framework for convergence on ICT for real-

time monitoring. The NITI Aayog's National Nutrition Strategy supports the National Nutrition Mission, which aims to achieve "Kuposhan Mukt Bharat," or malnutrition-free India, by 2022.

Diagnosis

Although there are other methods for diagnosing anaemia, a complete blood count (CBC) test is the most often used technique. This exam evaluates several elements, such as**:** haematocrit levels are determined by dividing the volume of red blood cells (RBCs) by the total volume of blood.

Table 3: Haemoglobin levels to diagnose anaemia (g/dl) According to WHO

Age Group	Anaemia			
	No Anaemia	**Mild**	**Moderate**	**Severe**
Children 6–59 months of age	≥11	10–10.9	7–9.9	<7
Children 5–11 years of age	≥11.5	11–11.4	8–10.9	<8
Children 12–14 years of age	≥12	11–11.9	8–10.9	<8
Non-pregnant women (15 years of age and above)	≥12	11–11.9	8–10.9	<8
Pregnant women	≥11	10–10.9	7–9.9	<7
Men	≥13	11–12.9	8–10.9	<8

Red Blood Cell count

A person's general health can be inferred from a CBC. It can also assist a physician in determining whether to perform an examination for underlying diseases like renal disease or leukemia. If RBC, haemoglobin, and hematocrit levels fall below the typical range, a person likely has some form of anaemia. However, it is possible for a healthy person’s levels to fall outside this range. A CBC is not conclusive, but it is a helpful starting point for a doctor to make an accurate diagnosis.

Nutrition Guidelines

It is necessary to consume a well-balanced diet that includes enough calories, high-quality protein, and foods high in iron, folic acid, vitamin "B12, " and vitamin "C."

- To promote improved absorption and digestion, eat frequently and in small portions.
- Eat more foods high in protein. Vegetarians can acquire their protein from milk, paneer, cheese, soy nuggets, and curds (yoghurt). In addition to the foods listed above, non-vegetarians can acquire protein from eggs and lean meats like chicken and fish.

- Eat meals high in vitamins and minerals.

Foods High in Iron

Cereals and millets: Bajra, ragi, whole wheat flour, puffed rice and rice flakes.

Legumes: lentils, cowpeas, rajmah, horse gram, moth beans, and bengal gram dal.

Not vegetarian: Mutton, chicken, egg yolk, and liver of chicken.

Leafy green vegetables such as drumstick leaves, onion stalks, gogu, methi, curry powder, and mint chutney should be consumed on a daily basis.

Nuts and oil seeds: melon seeds, coconut dry, almonds and pista, flax seeds, chia seeds, groundnuts, and flax seeds.

Dried fruits: Apricot, black dates, and raisins.

Consume foods high in vitamin C, such as amla, guava, berries, melon, mango, pineapple, citrus fruits, lime juice, etc., with your meals to improve the absorption of iron from these iron-rich foods even more.

It is advised to use double-fortified salt (iron + iodine).

Avoid the following

- Coffee or tea should not be consumed with meals. Take it an hour before dining or just after.
- Avoid eating too many calcium-rich foods, such as milk, cheese, paneer, etc., with a meal high in iron. The absorption of iron is also impacted by a diet high in fiber.
- Steer clear of processed and refined foods. Eat fewer noodles, polished rice, pasta, and other ready-to-eat items.
- Fluids**:** minimum intake of 2½ -3 litres / day

Iron Deficiency Anaemia Sample 1-Day Menu

Menu Time	**Menu**
Breakfast	1 serving citrus fruit 3/4 cup raisin bran cereal 1 egg 1 slice whole-wheat toast 1 tsp margarine
Lunch	3 oz tuna fish 2 slices whole-grain bread 1/2 cup carrots 1 medium apple 1 tsp mayonnaise

	1/2 cup low-fat or nonfat milk
Afternoon Snacks	1 cup nonfat yogurt 1/2 cup grapes 1 pear
Evening Meal	3 oz chicken 1 medium baked potato, with skin 1.5 cups mixed salad 1 tsp olive oil and vinegar dressing 1/2 cup low-fat or nonfat milk

Iron Deficiency Anaemia Vegan Sample 1-Day Menu

Menu Time	**Menu**
Breakfast	1 cup fortified bran cereal with raisins 1 cup soymilk fortified with calcium, vitamin B12, and vitamin D ½ cup strawberries
Morning Snacks	2 tablespoons walnuts
Lunch	1 cup kidney beans ½ cup sweet potatoes, mashed ½ tablespoon margarine, soft, tub 1 cup raw broccoli 1 orange
Afternoon Snacks	3 apricots, dried 11 almonds
Evening Meal	Stir-fry made with**:** 1 cup tofu 1 cup brown rice 1 cup spinach, cooked ¼ cup coconut milk 1 tablespoon peanut butter

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27. Ashley Mauldin, MSN, APRN, FNP-BC. Editors**:** Alyssa Haag, Emily Miao, PharmD. Illustrator**:** Jillian Dunbar. Copyeditor**:** Sadia Zaman, MBBS, BSc. Modified**:** 31 Jan 2022 Pernicious Anaemia**:**What It Is, Causes, Signs, Symptoms, and More.

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Chapter - 5
Obesity and Related Complications

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Chapter - 5

Obesity and Related Complications

Moumita Das

Introduction

Obesity is a complex metabolic disorder mainly caused by an imbalance between energy intake and expenditure. It can result from the interaction of numerous aspects such as genetic, dietary, lifestyle, physiological, and also environmental factors (Karri *et al*., 2019). According to World Health Organisation, currently more than 1.9 billion adults are overweight, of these over 650 million are obese worldwide. Overweight and obesity condition are linked to more death rates over the world than underweight. A rise in sedentary lifestyle and diet modification majorly provoked a progressive increase in the incidence of obesity and its associated metabolic complications. In the global scenario, three countries USA, China and India have captured approximately more than 50% of obese individuals.

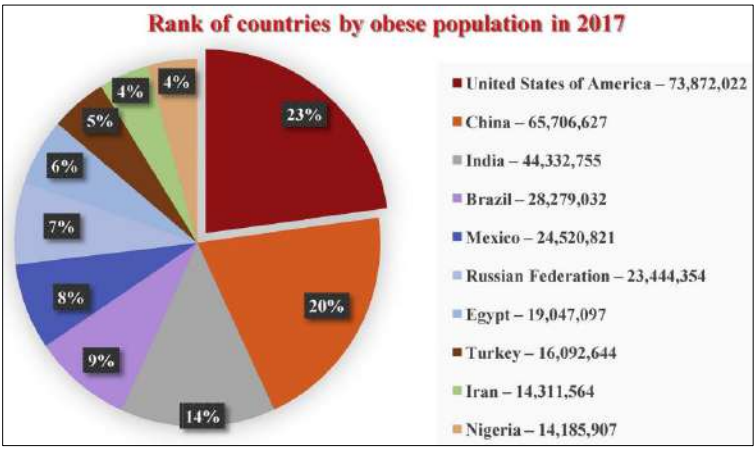

Fig 1.1: Epidemiology of obesity condition worldwide (Karri *et al*., 2019).

Obesity is a state of metabolic dysfunction usually caused by an imbalance of energy intake and expenditure, in addition to genetic background. It is a cluster of risk factors associated with the development of several other metabolic complications including insulin resistance, type 2

diabetes, hyperlipidemia and cardiovascular disease. Obesity associated hyperlipidemia is mainly characterized by increased level of serum total cholesterol, low-densitylipoprotein cholesterol (LDL-C) and triglycerides (TG) along with that decreased level of high density lipoprotein cholesterol (HDL-C). Obesity contributes to fat accumulation in the liver which is a major risk factor for the development of non-alcoholic fatty liver disease (NAFLD). The prevalence of obesity and its associated metabolic complications have considerably increased worldwide. The substantial increment in sedentary lifestyle and unhealthy diet pattern instigated the significant rise in the incidence of obesity condition (Das *et al*., 2022). Various traditional drugs are used to treat obesity associated complications. These include statins, ezetimibe, cholestyramine and fibrates which have been vividly used to treat hyperlipidemia condition and thiazolidinediones (TZD) to treat obesity associated type 2 diabetes. But these drugs are often associated with different harmful side effects (Chen *et al*., 2014). To overcome this, the scientific community constantly looks for the natural resources to treat obesity-associated metabolic dysfunctions without any harmful side effects. The prevalence of obesity is one of the most serious health problems worldwide. Therefore, there has been increasing interest in searching for novel nutraceuticals and functional foods to be utilized as dietary intervention strategies for the management of obesity related complications (Das *et al*., 2022).

Prevalence in India

India as a developing country is considered in a transition state of under nutrition mainly due to poverty and obesity because of rapid urbanization and changes in the life style. A recent study by Ahirwar and Mondal (2019), reported that the prevalence of obesity in India is higher among the urban populations with high socioeconomic status. During the period of 1998 to 2018, there has been a drastic change in the lifestyle of the Indian population towards physical inactivity and consumption of calorie-dense foods. In India, more than 135 million individuals are affected by obesity in the current scenario. Rising rates of obesity has led to a drastic increase in the prevalence of obesity associated comorbidities. Assessment of obesity status can be done by various anthropometric tools. Some of the important methods are summarized as follows.

Body Mass Index (BMI): BMI is a calculation based on weight and height. It provides a standardized way to categorize weight status. You can calculate your BMI using online tools or a simple formula**:** BMI = weight (kg) / height (m^2)

Interpreting the BMI Spectrum

The World Health Organization (WHO) classifies weight status based on BMI ranges**:**

- **Underweight:** BMI less than 18.5
- **Normal Weight:** BMI 18.5 to 24.9
- **Overweight:** BMI 25 to 29.9
- **Obese:** BMI 30 or greater

While BMI is a widely used tool, it has limitations. So, it is crucial to consider other factors such as follows**:**

- **Muscle Mass:** Muscle weighs more than fat. An individual with a high amount of muscle mass might have a high BMI despite a healthy body composition.
- **Body Frame:** Individuals with larger frames naturally weigh more than those with smaller frames.
- **Age:** It is an important factor for different individuals, BMI might vary among adolescent and pregnant women.

Beyond BMI: A More Holistic Approach

For a more accurate picture, healthcare professionals may consider these additional factors**:**

- **Waist Circumference:** Excess fat around the waist is linked to a higher risk of health problems.
- **Body Composition:** Techniques like bioelectrical impedance analysis can estimate muscle mass and body fat percentage.
- **Medical History and Family History:** Certain medical conditions and family history can influence weight.

Aetiology of obesity

The aetiology of obesity is multifactorial and includes a complex interplay between several factors like genetic, environmental, psychosocial and behavioural (Yilmaz & Younossi, 2014). Obesity is defined on the basis of body mass index (BMI) above 30 kg/m^2, BMI in the range of 27 to 30 kg/m^2 is described as over-weight condition. Although BMI is not considered as a predictor of adiposity or distribution of fat, higher level of BMI does enhance the risk of metabolic complications. Genetic and environmental factors majorly impact the body weight of an individual. Additionally, various

socioeconomic factors strongly influence the obesity incidence. Progressive changes in lifestyle associated with urbanization, physical inactivity, access to easily available calorie dense and refined food items are the major contributing factors in the increasing prevalence of obesity and its associated complications. Maternal obesity is considered to be strongly associated with the development of obesity and type 2 diabetes in off-spring (Sarma *et al*., 2021).

Pathogenesis of obesity and associated complications

Pathogenesis of obesity mainly involves either increased appetite or reduced utilization of calories occurs due to physical inactivity, cellular dysfunctions etc. Development of obesity is fundamentally associated with excess accumulation of fats in adipocytes resulting in adipocyte dysfunction, abnormal secretion of cytokines and chronic low-grade inflammation. Adipose tissue remodeling is one of the elementary defects in obesity and may lead to further metabolic diseases (Yilmaz & Younossi, 2014). Adipose tissue is known for secreting various molecules called adipokines. Impaired adipose tissue function results in secretions of a number of proinflammatory and atherogenic adipokines and raises the risk of developing vascular complications (Karri *et al*., 2019). Adipokines are known for modulating glucose as well as lipid metabolism, thus their altered secretion might adversely impact the insulin sensitivity and secretion. Obesity contributes to arise comorbidities for type 2 diabetes, dyslipidaemia, fatty liver diseases, chronic inflammation, hypertension and cardiovascular diseases (Blüher, 2013). Figure 1.2 represents the metabolic progression and major complications of obesity.

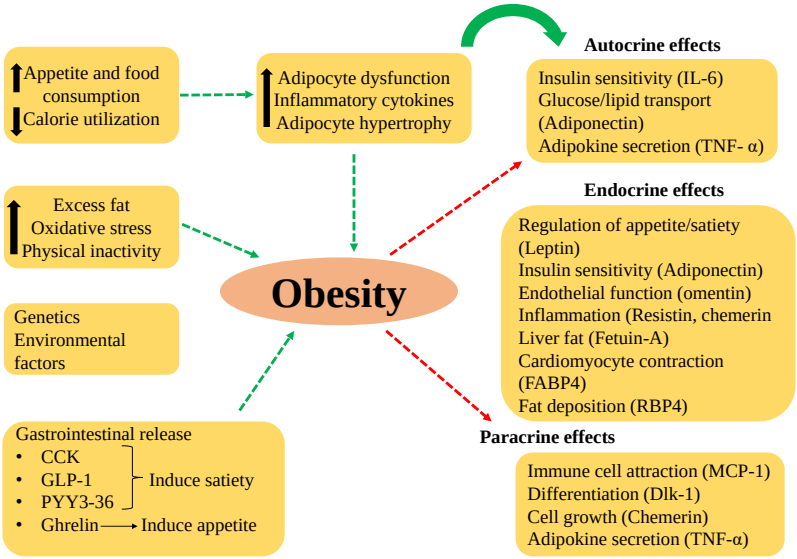

Fig 1.2: Schematic representation of metabolic progression and complications of obesity. (Modified from Blüher, 2013 and Karri *et al.*, 2019).

Obesity is linked with concomitant rise in the risk of several other chronic conditions. In obesity, deposition of excess circulating lipids as triglycerides in non-adipose organs like liver, skeletal muscle, heart and pancreas can adversely affects local and systemic insulin sensitivity (Longo *et al.*, 2019). This ectopic fat deposition is the primary cause of obesity induced insulin resistance in white adipose tissue (WAT), liver and skeletal muscle. Obesity associated insulin resistance further arises a wide cluster of metabolic abnormalities. It is strongly corelated with the development of insulin resistance and type 2 diabetes, dyslipidaemia, non-alcoholic fatty liver disease, hypertension, cardiovascular disease, and malignancy. Obesity related metabolic dysfunction is characterized by visceral adiposity, hypoadiponectinemia, hyperresistinemia, glucose intolerance and dyslipidaemia condition. Obesity can also significantly impact the mental health of people which in turn influence the quality of human life (Sarma *et al.*, 2021).

White adipose tissue (WAT)

White adipose tissue is primarily responsible for controlling energy homeostasis. Adipocytes reserve energy and utilise it according to the need of body. Additionally, adipocytes secrete paracrine factors to modulate the metabolism of other tissues by sensing the energy requirement of body. Dysfunction of WAT in obesity induces ectopic fat accumulation in other

tissues (related with glucose metabolism) developing lipotoxicity. This metabolic condition further leads to systemic insulin resistance in body.

Liver

Liver is the central organ that plays a crucial role in lipid metabolism and maintains energy balance through numerous metabolic pathways including *de novo* lipogenesis, uptake and oxidation of fatty acids and exporting triacylglycerol. In obesity condition, liver becomes susceptible to ectopic lipid accumulation which further lead to the development of non-alcoholic fatty liver disease (NAFLD). Increase in *de novo* fatty acid biosynthesis might enhance the fatty acids accumulation in the hepatocytes (Yilmaz & Younossi, 2014).

Skeletal muscle

Skeletal muscle is one of the main tissues in the body primarily responsible for homeostasis of glucose and lipid utilization. Ectopic lipid accumulation in skeletal muscle has been considered to be crucial to instigate insulin resistance in the body particularly in obesity condition (Sarma *et al*., 2021).

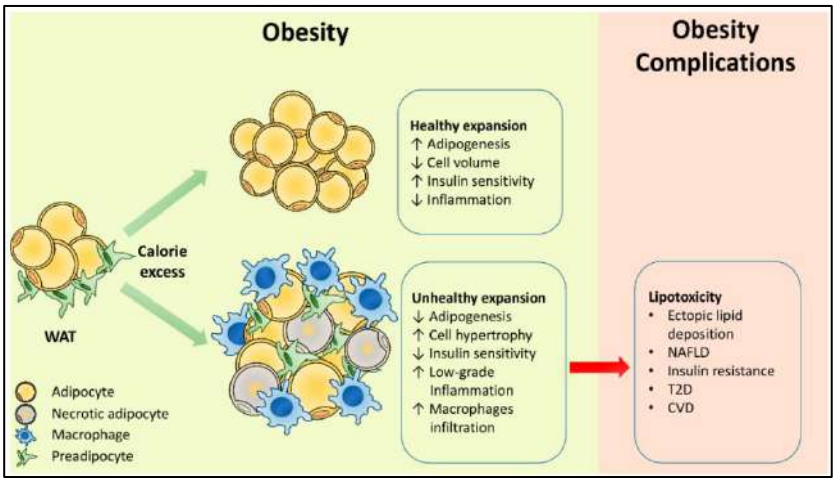

Fig 1.3: Healthy and unhealthy expansion of white adipose tissue in obesity condition and its association with metabolic complications. Figure was adapted from Longo *et al*., (2019).

Prevention and treatment of obesity

Obesity leads to the development of cardiovascular diseases worldwide including developing and developed countries. Many commercial drugs like statins, ezetimibe, cholestyramine and fibrates etc., have been widely utilized

to treat obesity associated dyslipidaemia condition (Thompson *et al*., 2016; Kashani *et al*., 2008). The conventional drugs used to treat obesity condition and their major adverse impacts are shown in table 1.1. The prescriptions of these molecules have been restrained by virtue of chronic side effects correlated with long term intake (He *et al*., 2018; Chen *et al*., 2014). So, the main focus of scientific community is to consistently look for natural resource which can be beneficial for the prevention and treatment of obesity without any harmful side effects. Limited success has been observed in the therapeutic interventions for obesity treatment, therefore there is an immediate need for identification and utilization of novel nutraceuticals from various underutilized sources. This might provide optimal medical nutritional therapy in the management of obesity and its associated metabolic complications.

Table 1.1: Currently available long-term anti-obesity drugs for the treatment of obesity, their mechanism of action and adverse effects

Drug	Mechanism of action	Common adverse effects
Orlistat	Pancreatic lipase inhibitor	Loose oily stools, flatulence, fecal incontinence
Phentermine/ Topiramate ER	Sympathomimetic amine (appetite suppressant) Anti-convulsant agent	Dry mouth, paresthesia, insomnia, depression, anxiety
Naltrexone SR/ Bupropion SR	Norepinephrine/ dopamine reuptake inhibitor and opioid receptor antagonist	Nausea, dry mouth, constipation, headache, dizziness
Liraglutide	GLP-1 analogues	Nausea, vomiting, constipation, diarrhea
Ezetimibe	Inhibit the absorption of biliary and dietary cholesterol	Mild to moderate gastrointestinal discomfort
Cholestyramine	Bind bile acid in gut, prevent their enterohepatic circulation, promote their fecal excretion	Abdominal pain, heart burn, bloating, constipation
Statins	HMG-CoA reductase inhibitor	Myalgia, cramp and muscle weakness, central nervous system complaints

Many studies depicted the effects and safety efficacy of the use of pharmacological strategies for treating obesity condition (Son and Kim, 2020; Kang and Park 2012). In table 1.1, some of the most commonly used drugs for treating obesity and related dyslipidaemia in patients is described. The major mechanism of action and adverse side effects for long term use has been investigated by several studies in the past decade (Zodda *et al*., 2018; Nutescu and Shapiro, 2003).

Nutraceuticals to combat obesity associated complications

Fig 1.4: The nutraceuticals reported to mitigate obesity and associated metabolic complications.

Embracing a diet rich in natural, unprocessed whole foods is a cornerstone strategy for preventing obesity. These foods are packed with essential nutrients that promote satiety, regulate metabolism, and provide sustained energy. In the recent years there are ample of studies on modulatory role of nutraceuticals or functional ingredients in management of obesity and associated complications. Emerging activity of omega-3-fatty acids, soluble fibres, quercetin, resveratrol, pre and pro-biotics etc in nutrition therapy of obesity has gained attention of the scientific community. These nutraceuticals are reported in clinical trial to exert several beneficial properties in obesity condition. Green tea, ginger, oates, mushrooms are rich in functional bioactive ingredients to help in reducing complications related to obesity and overweight. The mechanism of those nutraceuticals has been proposed by various studies but the study on bioavailability remains elusive. The challenges that science could seek to overcome this can be the substantially studied on the application of bioactive components for development of functional food formulations to address many limitations related to the production of nutraceuticals enriched foods. Further the evaluation of the food products with respect to its nutritional quality, efficacy of bioavailability, stability as well sensorial effectiveness are the urgent requirement. Most importantly extensive scientific clinical trials should confirm the health benefits and safety of such sterol rich food formulations.

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Chapter - 6
Gastrointestinal Disorders

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Chapter - 6

Gastrointestinal Disorders

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Introduction

Gut health has now become one of the most important health criteria among other health conditions. In this chapter, the diseases and conditions associated with gastrointestinal tract is elaborated. The conditions and diseases associated with GI tract infections are very commonly occurring in everyday life. The factors like poor sanitation, hygienic practices, changes in eating pattern, drug side-effects, consumption of contaminated foods are mainly associated with these diseases and conditions. The side-effects may vary among individuals causing nausea, vomiting, anorexia, weakness, bloating to severe abdominal discomfort, gas, flatulence, delayed gastric emptying and diarrhoea. Depending upon the severity of the disease, syndromes like dumping syndrome, malabsorption syndrome etc. may also occur (Patterson *et al*, 2022).

It is very important to know about one's gut health to prevent the occurrence of these diseases and for normal GI functioning otherwise it will affect nutrient absorption at various points causing malnutrition.

Gastrointestinal (GI) Tract Physiology

There are various organs that form the GI tract and are involved in its functioning (Figure 1). The main steps involved in digestion and assimilation of the food include (Wardley, 2007)**:**

- Gulp the food.
- **Digestion:** The food is mixed with the enzymes that are released in different parts of the of the gastrointestinal tract. The complex food is broken down into simpler forms.
- **Motility:** The food mixture is propelled through mouth, oesophagus, stomach, duodenum, small and large intestine to the anus.
- **Absorption:** The various nutrients are absorbed into the blood mainly from small intestine and other parts of the body.

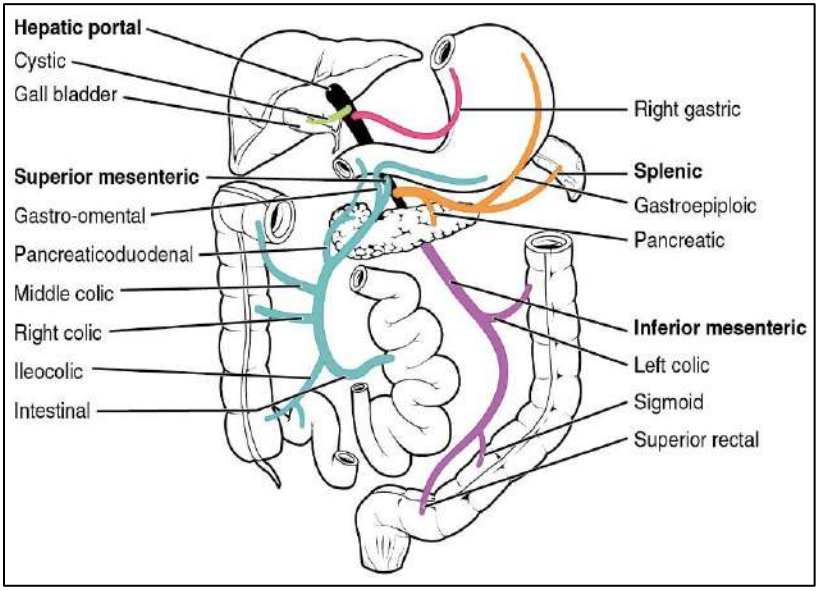
Any disturbance at any point of the GI tract may affect storage,

propulsion, digestion and absorption leading to malnutrition due to nutrient deficiencies.

Based on location of the organs from the centre of the body, GI tract is divided into two sections:

Upper GI Tract

- Mouth
- Esophagus
- Stomach
- Parts of small intestine



Source**:** Maleta *et al*, 2019

Fig 1: Organs Involved in Gastrointestinal Tract

Lower GI tract

- Large intestine
- Rectum
- Anus

Gastrointestinal Disorders

There are various disorders associated with gastrointestinal tract. Some of them are mentioned below

a. Dysphagia (Augustyn, 2024)

It is a condition that includes difficulty in moving food or liquid through the mouth, pharynx and esophagus (Figure 2). The patient could sense the swallowed material sticking along its path. Dysphagia is caused by two main mechanisms

- Mechanical obstruction
- Motor dysfunction

Source: Wen *et al*, 2023

Fig 2: Patient having difficulty in swallowing

Depending upon the mechanism involved, dysphagia can be sub-divided into two types-

- **Oropharyngeal dysphagia:** The patient has difficulty in swallowing. The conditions of nasopharyngeal regurgitation and aspiration may be present. It is caused due to diseases like advanced stage of Alzheimer's disease, developmental disabilities, thyroid disorders, multiple sclerosis, muscular dystrophy, Parkinson's disease, stroke etc.
- **Esophageal dysphagia:** In this disorder the food gets stuck in middle or lower sternal area. It can be associated with regurgitation, aspiration etc. The difficulty may arise both for solid or liquid food. It is caused due to diseases like Achalasia, enlarged atrium (right side of the heart), esophageal cancer, esophageal spasm, scleroderma, lung cancer etc.

Complications of dysphagia

- If food consumption is decreased, malnutrition and weight loss may occur.

- Individuals who could not swallow liquids are at high risk of dehydration.
- Aspiration (inhalation of food, liquid into lungs) may cause airway obstruction, choking or respiratory infections, including pneumonia. If a person does not have a normal cough reflex, aspiration is more difficult to diagnose and may go unnoticed.

Diagnosis of Dysphagia

- **Fluoroscopic Barium Swallow study:** This reveals the nature of a problem. In this study, the patient consumes food or liquid containing barium (a metallic element visible on X-rays) and the swallowing process is monitored using a video X-ray technique known as video fluoroscopy.
- **Endoscopy:** It is a thin, flexible tube to examine the oesophageal lumen directly.
- **Monomeric assessment:** Measurement of esophageal sphincter pressure and peristalsis.
- **Neurological examination:** To evaluate mental status, physical reflexes and cranial nerves are associated with swallowing.

Nutritional Management for Dysphagia

Nutrition Assessment

For assessment of anthropometric parameters, dietary intake, weight loss and development of nutritional deficiencies should be done.

Nutritional Diagnosis

Based on the assessment, dietary deficits and stage of malnutrition to be identified and nutritional deficiency should be diagnosed based on the biochemical parameters.

Nutritional Management

- **Modification of physical properties:** The physical properties of foods and beverages could be modified using alternative feeding methods to reduce the level of difficulty in swallowing. Foods as a part of diet should have easy-to-manage textures and consistencies. This includes soft, cohesive foods rather than hard or crumbly foods.

The American Dietetic Association, 2007 international guidelines have specified the consistency levels of foods as per the tolerance of ability to swallow the food as part of medical nutrition therapy. These are mentioned in the Table 1 below.

Table 1: Foods to be included and avoided in Dysphagia pureed

S.No.	Level 1	Level 2	Level 3	
	Dysphagia pureed	**Dysphagia mechanically altered**	**Dysphagia advanced**	**Liquid consistencies**
1.	Well mashed or pureed food	Soft textured food; easily form a bolus	Mixed texture	Only those tolerated are allowed in the diet
2.	Homogenous and cohesive consistency	Moist, cohesive	Moist and bite sized pieces when swallowed	Thin**:** watery fluids- milk, tea, coffee, juices, carbonated beverages
3.	For patients with moderate or severe dysphagia and poor oral or chewing ability.	For patients with mild to moderate dysphagia. Some chewing ability is required.	For patients with mil dysphagia and adequate chewing ability.	Nectar like**:** Thicker than water that can be sipped through straw- butter milk, unstrained juices (tomato, grape, watermelon)
4.	Foods include- Mashed banana, potato, peaches, soups, gravies, Vegetable stocks, puddings and scrambled eggs	Foods include- moist oatmeal, cornflakes, white bread, rice flakes with milk, soft tender cooked vegetables, fruit pies, minced-tender cooked meat and scrambled eggs	Foods include- poached or scrambled eggs, cheese, soft-fresh juicy fruits, moistened bread, shredded, tender cooked, finely chopped vegetables, coffee, tea (if thin liquids are tolerated)	Spoon thick**:** Fluids that can be eaten with spoon and can hold their shape- milk pudding, thickened milkshakes
5.	Foods avoid- Cheese, oatmeal, rice, fat rich sources, peanut butter, pulpy beverages, fruit chunks and seeds	Foods avoid- dry foods, hard cooked eggs, sprouts, sandwiches, slied cheese, dried fruits, raw vegetables and crackers	Foods avoid- dry foods, vegetable skins, raw vegetables, hard textured fruits or fruit skin, chewy candies, nuts and seeds, chunky peanut butter	

b. Indigestion (Augustyn, 2024)

Indigestion is a term that includes variety of upper abdominal conditions like heartburn, regurgitation and dyspepsia (non-specific, upper abdominal discomfort or pain) (Figure 2). The causes of indigestion are as follows**:**

- Gastro Esophageal Reflux Disease (GERD)
- Gastritis
- Peptic ulcer
- Gall Bladder disease
- Motility disorders
- Malabsorption disorders
- Tumours in oesophagus or stomach
- Conditions like diabetes mellitus, heart disease and hypothyroidism
- Iron and potassium supplements

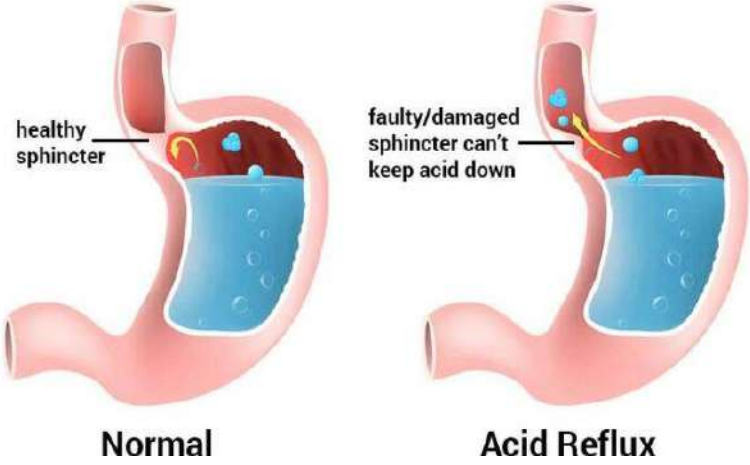
Source: Wen *et al*, 2023

Fig 2: Upper abdominal pain

Signs and symptoms: Epigastric discomfort following meals, abdominal pain, bloating, nausea, vomiting/ regurgitation

- **Gastro Oesophageal Reflux Disease (GERD) (Johnson, 2023):** GERD is one of the most prominent symptoms related to upper GI tract infections. It may be defined as "mucosal damage produced by abnormal reflux of gastric contents into oesophagus". It is a condition that occurs due to acid reflux into oesophagus from stomach due to gastric motor dysfunction. There is a backward flow (regurgitation) of the gastric acid and pepsin or duodenal contents into oesophagus (Figure 3). The reflux occurs when pressure inside the stomach is higher than that maintained by the muscles at the stomach and oesophagus junction. There are many factors associated with oesophageal reflux as mentioned in Table 2 (Nelms and Sucher, 2011). The causes of GERD may be**:**

- Reduced lower oesophageal sphincter (LES) pressure
- Inadequate oesophageal tissue defence
- Direct mucosal irritants
- Decreased gastric motility
- Increased intra-abdominal pressure
- Oral contraceptives



Source: Johnson, 2023

Fig 3: Acid Reflux into Oesophagus from stomach

Table 2: Factors associated with Oesophageal Reflux

Conditions	Substances
Ascites (accumulation of fluid in abdomen)	Alcohol; anticholinergic agents
Delayed gastric emptying	Antihistamines
Eating large meals	Caffeine
Lying flat after eating	Calcium channel blockers
Obesity	Chocolate
Pregnancy	Smoking
Wearing clothes that fit directly across the waist or abdomen	Diazepam, garlic, high fat foods, onion
Hiatal hernia	Peppermint and spearmint oils
Scleroderma	Progesterone
Increased secretion of hormones gastrin, estrogen, progesterone	Theophyline, antidepressants

Complications of GERD

- Untreated GERD may lead to impaired swallowing, aspiration of

gastric contents into lungs, ulceration and perforation or stricture of oesophagus.

- Oesophageal ulcers due to severe and chronic inflammation.
- Consequent bleeding, healing and scarring or ulcerated tissue may narrow in the inner diameter of oesophagus. This may lead to oesophageal stricture.
- Occurrence of pulmonary disease due to aspiration of gastric content in lungs.
- Occurrence of eroded tooth enamel, sore throat and laryngitis.
- Barrett's oesophagus- a severe condition in which normal squamous epithelium of distal oesophagus is replaced by cells that resemble gastric or intestinal tissue.

Diagnosis of GERD

- Endoscopy
- Esophagogastroduodenoscopy (EGD)- This visualizes esophagus, stomach and duodenum
- Barium radiology studies
- Oesophageal manometry
- pH monitoring
- Bernstein test

Nutritional Management for Indigestion, GERD and other associated complications

Nutritional Assessment

- Anthopometry
- 24 hr recall
- Diet history/ food diary- to focus consumption of foods that lower LES pressure and increase gastric acidity
- Lifestyle factors including smoking, physical activity patterns or others which may challenge LES competence

Nutritional Diagnosis: Intake of inadequate food/ oral beverage, excessive fat intake, difficulty in swallowing, obesity, inadequate iron-calcium intake, impaired nutrient utilization, undesirable food choices, food and nutrition related knowledge.

Dietary guidelines: Intake of balanced diet with more alkaline foods. The prescribed nutritional parameters are-

- **Energy:** As per REE and ER (1500-1800 Kcal for adults). For obese person (20-25kcal/Kg/day)
- **Carbohydrates:** 55-60% of total energy
- **Protein:** 15-20% of total energy (0.8-1.0 g/kgBW/day)
- **Fats:** 15-20% of total energy
    - Meals should be consumed at least 2-3 hours before the bed time.
    - Bed time meals and snacks should be avoided.
    - Foods that may weaken LES pressure and increase acid production should be avoided- carbonated beverages, tea, coffee, cocoa, alcohol, high fat milk and milk products, meat products, scrambled eggs, sweets and high sugar content products, preserved or canned products.
    - In case of oesophagitis, avoid consumption of citrus fruits and juices, tomato products, pepper, spicy foods etc.

Patient counselling

- Elevate the head end of the bed by 6-8 inches to reduce night time reflux.
- Consumption of small and frequent meals.
- Cigarette smoking and alcohol should be discontinued.
- Wearing tight fitting garments to be avoided.
- For obese people, weight loss is recommended.
- Avoid long-term use of Non-steroidal Anti-Inflammatory Drugs (NSAIDs) like aspirin, ibuprofen etc.

c. Gastritis (Augustyn, 2024)

Gastritis is a broad term that include symptoms resulting from inflammation of the stomach lining and symptoms of burning or discomfort. The mucous membranes of the stomach are inflamed and irritated due to excess of acid production in the stomach. The main causative factor of gastritis is the infection caused by *Helicobcater pylori*.

Depending on the severity of the infection, gastritis can be classified as

- Acute gastritis
- Chronic gastritis

Acute gastritis: It is the sudden inflammation of lining of the stomach which may result from irritating substances mainly bacteria or treatments that damage the gastric mucosa (high dose radiation). The symptoms include pain, nausea and vomiting. Symptoms like tissue erosions and ulcer may also occur. If these symptoms are not treated on time they may lead to chronic gastritis.

Chronic gastritis: This is caused by long term bacterial infections or autoimmune diseases. The symptoms include loss of appetite and abdominal discomfort for more than a week. There may be changes occurring in enzymatic activity of gastric mucosal cells that may lead to recurrent inflammation.

Signs and symptoms: nausea, vomiting, anorexia, heart burn, pain in upper abdomen, headache, bleeding, malaise, haemorrhage, dark stools, hiccups, low blood pressure, tachycardia and rapid pulse.

Complications of Chronic gastritis

- Hypochlorhydria- Abnormally low acid (HCl) secretion
- Achlohydria- Absence of HCl secretion
- Impaired absorption of non-heme iron and vitamin B12.
- Pernicious anemia- This occurs due to destruction of stomach cells which produce intrinsic factor that help in the absorption of vitamin B12.

Diagnosis: Gastritis can be diagnosed through physical examination and some diagnostic tests as mentioned below**:**

- Upper endoscopy
- Blood tests
- Stool test (faecal occult blood test)

Nutritional management of gastritis

- Liquid diet for one to two days, thereafter switch to semi-solid diet.
- Non-citrus fruits and alkalizing fruits like papaya.
- Preference for whole grains than refined flour. Soft cereals, milk, egg, fruit juices, vegetables, sugar puddings, porridge and khichdi may be given.
- Poorly tolerated foods should be excluded.
- Bland diet to be preferred with small frequent feedings.
- Adequate hydration- water and electrolyte (oral rehydration fluid).

About 150 ml liquid to be given per hour.

- Avoid fat intake and alcohol.
- Lactose tolerance to be monitored.

Summary

In this chapter, some common gastrointestinal disorders and their nutritional management is discussed. The pathophysiology and related diagnosis of each disorder is emphasized in detail. When adequately followed, these disorders can be prevented with utmost care through proper dietary intake.

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Chapter - 7
Nutrition Care and Dietary Approach in Diabetes Mellitus

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Chapter - 7

Nutrition Care and Dietary Approach in Diabetes Mellitus

Paromita Mukherjee

1. Introduction

A major public health concern, diabetes mellitus affects millions of people globally, says the International Diabetes Federation (IDF). One characteristic that sets diabetes apart is hyperglycemia, or consistently elevated blood glucose levels. Diabetes is a metabolic disease that progresses over time as a result of insufficient insulin production or activity. (American Diabetes Association [ADA], 2021a). The quality of life and the risk of morbidity and death can be greatly affected by the consequences that might develop from uncontrolled diabetes, such as cardiovascular disease, nephropathy, neuropathy, and retinopathy (Fowler, 2008).

Nutrition and dietary management are cornerstone components of diabetes care.According to Evert *et al*. (2019), a balanced diet significantly helps with optimal glycemic control, weight management, and preventing problems associated with diabetes. Personalised medical nutrition treatment for all diabetics is recommended by the American Diabetes Association (2021b) as an essential intervention. (MNT). A thorough comprehension of diabetes, its pathogenesis, and the function of food and nutrition in the treatment of the disease is the goal of this review. We will also discuss current dietary recommendations, sample diet plans, and emerging dietary strategies for diabetes care.

2. Diabetes Definition

High blood sugar levels (hyperglycemia) that do not go down due to normal insulin production or action—or both—are symptoms of diabetes mellitus, a metabolic condition that can last for years. Pancreatic beta cells secrete the hormone insulin, which promotes glucose storage in the muscles and liver and helps cells absorb glucose into the bloodstream (Kahn *et al*., 2014). When a person has diabetes, their blood glucose levels are consistently high because their bodies are unable to either create enough insulin or properly respond to the insulin they do produce. (DeFronzo *et al*., 2015).

3. Classification of Diabetes Mellitus

The two most prevalent types of diabetes are type 2 and type 1.

3.1. Type 1 Diabetes (T1D)

A total inability to produce insulin is a hallmark of type 1 diabetes, which goes by a few other names**:** IDDM, juvenile-onset diabetes, and hypoglycemia (Atkinson *et al*., 2014). Although it can happen at any age, type 1 diabetes usually affects young people and children (Maahs *et al*., 2010). It accounts for around 5-10% of all diabetes occurrences. No one knows for sure what causes type 1 diabetes, however environmental variables such viral infections and genetic predisposition are considered to have a role (Rewers & Ludvigsson, 2016).

3.2. Type 2 Diabetes (T2D)

The majority of instances of diabetes are type 2, often known as adult-onset diabetes or non-insulin-dependent diabetic mellitus (NIDDM) (Chatterjee *et al*., 2017). Insulin resistance is the failure of cells to respond to insulin as it should. and beta-cell failure becomes more severe during the course of type 2 diabetes (DeFronzo, 2009). According to research by Kolb and Martin (2017), there is a robust correlation between being overweight, not exercising consistently, and having a family history of type 2 diabetes.

3.3. Gestational Diabetes Mellitus (GDM)

Gestational diabetes mellitus is defined as a glucose intolerance of any severity that starts or is first detected during pregnancy, according to the American Diabetes Association (2021c). Gestational diabetes mellitus (GDM) affects around 7% of pregnancies globally and is associated with an increased risk of macrosomia, preeclampsia, and caesarean delivery (Buchanan *et al*., 2012). Research by Bellamy *et al*. showed that women with GDM had a greater chance of acquiring type 2 diabetes in old age (2009).

3.4. Other Types of Diabetes

Leslie *et al*. (2016) listed many less frequent forms of diabetes, including secondary diabetes caused by underlying disorders such pancreatic diseases, endocrinopathies, or drug-induced diabetes, maturity-onset diabetes of the young (MODY), and latent autoimmune diabetes in adults (LADA).

4. Pathophysiology of Different Types of Diabetes Mellitus

4.1. Pathophysiology of Type 1 Diabetes

The immune system mistakenly targets and destroys the beta cells in the pancreas, which are responsible for producing insulin, in type 1 diabetes, an

autoimmune illness. (van Belle *et al*., 2011). Environmental and genetic variables are believed to interact intricately to set off an autoimmune response, although the precise mechanisms by which it occurs remain unknown (Pociot & Lernmark, 2016). Beta cell death causes insulin production to gradually decrease until total insulin insufficiency is reached (Katsarou *et al*., 2017). Hyperglycemia and the necessity for exogenous insulin replacement treatment result from cells' inability to efficiently absorb glucose when insulin is absent. (Atkinson & Eisenbarth, 2001).

4.2. Pathophysiology of Type 2 Diabetes

According to what Kahn *et al*. (2006) said, the symptoms of type 2 diabetes include beta-cell malfunction and insulin resistance. Because insulin is not efficiently absorbed and used by cells throughout the body, particularly in the liver, muscles, and fat, glucose absorption and utilisation are impaired in insulin resistance (Samuel & Shulman, 2016). Hyperinsulinemia occurs when pancreatic beta cells secrete more insulin than the body needs to deal with insulin resistance (Prentki & Nolan, 2006). Progressive beta-cell malfunction and relative insulin shortage result from beta cells' inability to keep up with the body's rising insulin demand (Weyer *et al*., 1999). Hyperglycemia and type 2 diabetes are the outcomes of insulin resistance and beta-cell malfunction together (DeFronzo, 2009).

Insulin resistance and beta-cell dysfunction in type 2 diabetes are associated with a range of risk factors, such as obesity, low physical activity, genetics, and advanced age (Kahn *et al*., 2014). Obesity and insulin resistance are known to be associated with increased adipokine and pro-inflammatory cytokine production by adipose tissue, particularly in central obesity. (Guilherme *et al*., 2008). Chronic low-grade inflammation develops as a result of these variables and worsens insulin signalling and beta-cell function (Donath & Shoelson, 2011).

4.3. Pathophysiology of Gestational Diabetes Mellitus

From a pathophysiological standpoint, gestational diabetes mellitus (GDM) is comparable to type 2 diabetes. Human placental lactogen, prolactin, and cortisol are among the hormones produced by the placenta during pregnancy that might cause insulin resistance (Lain & Catalano, 2007). Typically, as insulin demand increases, the pancreatic beta cells respond by secreting more insulin (Buchanan & Xiang, 2005). On the other hand, hyperglycemia and the onset of GDM can occur in women whose beta cells fail to effectively compensate (Ploughs *et al*., 2018). Factors that increase the likelihood of gestational diabetes include a mother's age, her weight, her

family's diabetes history, and her ethnic heritage (Chiefari *et al*., 2017).

5. Signs and Symptoms of Diabetes Mellitus

The classic symptoms of diabetes mellitus include

1. Polyuria (increased urination)**:** An increase in urine output is caused by the osmotic action of blood glucose levels that pull fluid out of tissues (Chiasson *et al*., 2003).
2. Polydipsia (increased thirst)**:** As a result of fluid loss through increased urination, individuals with diabetes often experience excessive thirst (Kitabchi *et al*., 2009).
3. Polyphagia (increased hunger)**:** Because their bodies can't use glucose for energy efficiently, people with diabetes may lose weight even when they consume more. (Kitabchi *et al*., 2006).
4. Fatigue**:** Weakness and exhaustion might result from hyperglycemia and the body's inefficiency in using glucose as an energy source. (Morbach *et al*., 2012).
5. Blurred vision**:** Blurred vision might occur as a result of fluid being drawn from the eye lenses due to high blood glucose levels(Bourne *et al*., 2013).
6. Slow-healing wounds**:**Diabetes, by reducing blood flow and damaging nerves, can hinder the body's wound-healing abilities (Boulton *et al*., 2005).
7. Tingling or numbness in hands or feet**:** Chronic hyperglycemia can lead to nerve damage (diabetic neuropathy), causing tingling, numbness, or pain in the extremities (Tesfaye *et al*., 2010).

Some people with diabetes, especially tpe 2 diabetics, may not have any symptoms at all when the condition is first starting to progress. In high-risk groups, this emphasises the significance of diabetes screening on a frequent basis.

6. Diagnosis of Diabetes Mellitus

Plasma glucose indicators used to diagnose diabetes mellitus include fasting plasma glucose (FPG), 2-hour plasma glucose (2-h PG) following a 75-g oral glucose tolerance test (OGTT), and glycated haemoglobin (A1C) criteria (ADA, 2021a).

6.1. Fasting Plasma Glucose (FPG)

Blood glucose levels are determined by FPG after an 8-hour fast. The criteria for diagnosing diabetes based on FPG are as follows**:**

- Normal**:** FPG < 100 mg/dL (5.6 mmol/L)
- Impaired Fasting Glucose (IFG)**:** FPG 100-125 mg/dL (5.6-6.9 mmol/L)
- Diabetes**:** FPG ≥ 126 mg/dL (7.0 mmol/L)

6.2. 2-Hour Plasma Glucose (2-h PG) after 75-g Oral Glucose Tolerance Test (OGTT)

A 75-gram glucose load is consumed two hours before and two hours after the two-hour post-test, which is known as the oral glucose tolerance test (OGTT). Criteria for a diabetes diagnosis based on a 2-hour PG are as follows.**:**

- Normal**:** 2-h PG < 140 mg/dL (7.8 mmol/L)
- Impaired Glucose Tolerance (IGT)**:** 2-h PG 140-199 mg/dL (7.8-11.0 mmol/L)
- Diabetes**:** 2-h PG ≥ 200 mg/dL (11.1 mmol/L)

6.3. Glycated Hemoglobin (A1C)

The OGTT is a blood sugar measurement that is taken before and two hours after a 75-gram glucose load. Diabetes can be diagnosed using the following 2-hour PG-based criteria**:**

- Normal**:** A1C < 5.7%
- Prediabetes**:** A1C 5.7-6.4%
- Diabetes**:** A1C ≥ 6.5%

A second test done on a different day can confirm diabetes if hyperglycemia is not evident.It was the World Health Organisation [WHO], 2006).

7. Dietary Treatment of Diabetes Mellitus

The foundation of diabetes care is medical nutrition therapy (MNT), which should be tailored to each patient's specific dietary requirements, cultural preferences, and treatment objectives. (Evert *et al*., 2019). The primary objectives of MNT in relation to diabetes are to**:**

1. Aim for and maintain consistent, healthy blood glucose levels.
2. Improve and maintain healthy levels of blood lipids.
3. Make sure there are enough calories to keep or gain a healthy weight.
4. Reduce the likelihood of diabetic complications or put them off till later

7.1. Carbohydrate Management

Carbohydrate management is a key component of MNT in diabetes. The amount and type of carbohydrates consumed have a direct impact on postprandial blood glucose levels (Franz *et al*., 2002). Strategies for carbohydrate management include**:**

7.1.1. Carbohydrate Counting

Carbohydrate counting involves quantifying the amount of carbohydrates consumed at each meal and snack. Carbohydrate counting allows patients more freedom in their dietary choices by allowing them to adjust their insulin dosages to the quantity of carbs taken (Gillespie *et al*., 1998).

7.1.2. Glycemic Index and Glycemic Load

The glycemic index (GI) is a measure of the rate of increase in blood glucose levels relative to a reference diet, as discovered by Jenkins *et al*. (1981), which is often glucose or white bread. Blood glucose levels rise more slowly after eating low GI meals because of their slower digestion and absorption. The quick digestion and absorption of high GI foods causes blood glucose levels to increase quickly, according to Atkinson *et al*. (2008). According to Salmerón *et al*. (1997), the glycemic load (GL) considers both the GI and the carbohydrate content of a serving. People with diabetes can better regulate their blood sugar levels by eating meals that are low in glucose and glycemic index (Thomas & Elliott, 2009).

7.1.3. Fiber Intake

The absorption of glucose is slowed, postprandial blood glucose levels are reduced, and insulin sensitivity is improved by dietary fibre, especially soluble fibre (Chandalia *et al*., 2000). Vegetables, fruits, whole grains, legumes, and other naturally high-fiber foods should make up the bulk of a diabetic's diet, as the American Diabetes Association (ADA) suggests 14 g of fibre per 1, 000 kcal (ADA, 2021b).

7.2. Protein Intake

When it comes to building and sustaining bodily structures, protein is a macronutrient that is absolutely necessary. Age, weight, and renal function are some of the variables that should inform a diabetic's personalised protein consumption plan (Hamdy & Horton, 2011). Foods high in protein from plants, such as eggs, lean meats, poultry, and fish (such as lentils and soy products) should make up 15-20% of total daily calories, according to the ADA (ADA, 2021b).

7.3. Fat Intake

The type and amount of fat that people with diabetes consume may affect their risk of cardiovascular disease. According to Franz *et al*. (2002), the American Diabetes Association recommends a diet rich in monounsaturated and polyunsaturated fats, with twenty to thirty-five percent of daily calories coming from fat. Some examples of healthy fats are sardines, salmon, nuts, seeds, avocados, and olive oil. Cut back on processed foods, fatty meats, and high-fat dairy to reduce your risk of cardiovascular disease.(Sacks *et al*., 2017).

7.4. Sodium Intake

The development of cardiovascular disease and nephropathy can be accelerated in diabetics due to their hypertension (Ritz & Orth, 1999). Diabetics and those with hypertension should both cut back on salt consumption to below 2, 300 milligrammes daily, according to the American Diabetes Association (ADA, 2021b). To cut back on salt, try eating more fresh, unprocessed foods, checking nutrition labels, and seasoning dishes with herbs and spices instead of salt.

7.5. Alcohol Consumption

The effects of alcohol on blood glucose levels are well-documented, and they include an increased risk of obesity and other health problems.People with diabetes may be more or less likely to develop cardiovascular disease depending on the kind and quantity of fat they eat. According to Franz *et al*. (2002), the American Diabetes Association recommends a diet rich in monounsaturated and polyunsaturated fats, with twenty to thirty-five percent of daily calories coming from fat. Some examples of healthy fats are sardines, salmon, nuts, seeds, avocados, and olive oil. Cut back on processed foods, fatty meats, and high-fat dairy to reduce your risk of cardiovascular disease. people with diabetes should drink alcohol with food and keep an eye on their blood glucose levels as they drink (Emanuele *et al*., 1998).

8. Sample Diet Plans for Diabetes Management

The following sample diet plans are based on a 1, 600-calorie diet and can be adjusted to meet individual caloric needs and preferences. If you want a tailored eating regimen, it's best to go to a doctor or certified dietician.

8.1. Sample Diet Plan for Type 1 Diabetes

Breakfast

- 1 cup cooked oatmeal (30 g carbohydrate)

- 1/2 cup berries (7.5 g carbohydrate)
- 1 hard-boiled egg
- 1 tsp olive oil

Snack

- 1 small apple (15 g carbohydrate)
- 1 oz almonds

Lunch

- 2 slices whole-grain bread (30 g carbohydrate)
- 2 oz turkey breast
- 1 slice low-fat cheese
- 1 cup mixed greens
- 1 tbsp light salad dressing

Snack

- 1 cup raw carrots (12 g carbohydrate)
- 2 tbsp hummus

Dinner

- 3 oz grilled salmon
- 1 cup cooked quinoa (30 g carbohydrate)
- 1 cup steamed broccoli
- 1 tsp olive oil

Snack

- 1 cup low-fat Greek yogurt (10 g carbohydrate)
- 1/4 cup sliced strawberries (3 g carbohydrate)

8.2. Sample Diet Plan for Type 2 Diabetes

Breakfast

- 1 whole-grain English muffin (25 g carbohydrate)
- 1 tbsp almond butter
- 1 small banana (15 g carbohydrate)

Snack

- 1 cup raw bell pepper slices (5 g carbohydrate)
- 1/4 cup guacamole

Lunch

- 1 cup lentil soup (30 g carbohydrate)
- 1 small whole-grain roll (15 g carbohydrate)
- 1 cup mixed greens
- 1 tbsp balsamic vinaigrette

Snack

- 1 cup low-fat cottage cheese
- 1/2 cup sliced peaches (7.5 g carbohydrate)

Dinner

- 3 oz grilled chicken breast
- 1 small baked sweet potato (15 g carbohydrate)
- 1 cup roasted Brussels sprouts
- 1 tsp olive oil

Snack

- 1 small pear (15 g carbohydrate)
- 1 oz low-fat string cheese

Total carbohydrates**:** 127.5 g

8.3. Sample Diet Plan for Gestational Diabetes

Breakfast

- 1 slice whole-grain toast (15 g carbohydrate)
- 1/4 avocado, mashed
- 1 hard-boiled egg
- 1 cup low-fat milk (12 g carbohydrate)

Snack

- 1 small orange (15 g carbohydrate)
- 1 oz walnuts

Lunch

- 2 oz grilled chicken breast
- 1 cup cooked brown rice (30 g carbohydrate)
- 1 cup steamed green beans
- 1 tsp olive oil

Snack

- 1 cup low-fat Greek yogurt (10 g carbohydrate)
- 1/4 cup blueberries (5 g carbohydrate)

Dinner

- 3 oz baked cod
- 1 small baked potato (15 g carbohydrate)
- 1 cup roasted asparagus
- 1 tsp butter

Snack

- 1 small apple (15 g carbohydrate)
- 1 tbsp peanut butter

Total carbohydrates**:** 117 g

Table 1: Blood sugar levels measured following a 75-gram oral glucose tolerance test (OGTT), glycated haemoglobin (A1C), and fasting plasma glucose (FPG) are the diagnostic criteria for diabetes mellitus.

Test	Normal	Prediabetes	Diabetes
FPG	<100 mg/dL	100-125 mg/dL	≥126 mg/dL
2-h PG	<140 mg/dL	140-199 mg/dL	≥200 mg/dL
A1C	<5.7%	5.7-6.4%	≥6.5%

Table 2: Sample diet plan for type 1 diabetes (1, 600 calories).

Meal	Food	Carbohydrate (g)
Breakfast	1 cup cooked oatmeal	30
	1/2 cup berries	7.5
	1 hard-boiled egg	-
	1 tsp olive oil	-
Snack	1 small apple	15
	1 oz almonds	-
Lunch	2 slices whole-grain bread	30
	2 oz turkey breast	-
	1 slice low-fat cheese	-
	1 cup mixed greens	-
	1 tbsp light salad dressing	-
Snack	1 cup raw carrots	12
	2 tbsp hummus	-

Dinner	3 oz grilled salmon	-
	1 cup cooked quinoa	30
	1 cup steamed broccoli	-
	1 tsp olive oil	-
Snack	1 cup low-fat Greek yogurt	10
	1/4 cup sliced strawberries	3
Total		137.5

Table 3: Sample diet plan for type 2 diabetes (1, 600 calories).

Meal	Food	Carbohydrate (g)
Breakfast	1 whole-grain English muffin	25
	1 tbsp almond butter	-
	1 small banana	15
Snack	1 cup raw bell pepper slices	5
	1/4 cup guacamole	-
Lunch	1 cup lentil soup	30
	1 small whole-grain roll	15
	1 cup mixed greens	-
	1 tbsp balsamic vinaigrette	-
Snack	1 cup low-fat cottage cheese	-
	1/2 cup sliced peaches	7.5
Dinner	3 oz grilled chicken breast	-
	1 small baked sweet potato	15
	1 cup roasted Brussels sprouts	-
	1 tsp olive oil	-
Snack	1 small pear	15
	1 oz low-fat string cheese	-
Total		127.5

Table 4: Sample diet plan for gestational diabetes (1, 600 calories).

s	Food	Carbohydrate (g)
Breakfast	1 slice whole-grain toast	15
	1/4 avocado, mashed	-
	1 hard-boiled egg	-
	1 cup low-fat milk	12
Snack	1 small orange	15
	1 oz walnuts	-
Lunch	2 oz grilled chicken breast	-
	1 cup cooked brown rice	30

	1 cup steamed green beans	-
	1 tsp olive oil	-
Snack	1 cup low-fat Greek yogurt	10
	1/4 cup blueberries	5
Dinner	3 oz baked cod	-
	1 small baked potato	15
	1 cup roasted asparagus	-
	1 tsp butter	-
Snack	1 small apple	15
	1 tbsp peanut butter	-
Total		117

9. Emerging Dietary Strategies for Diabetes Management

In light of the potential benefits of several alternative dietary approaches in assisting with the management of diabetes, these are being considered alongside more traditional techniques.

9.1. Mediterranean Diet

The Mediterranean diet has been associated with improved glycemic control and a reduced risk of cardiovascular disease in diabetics. This diet is characterised by a moderate intake of red meat, eggs, nuts, seeds, and olive oil, as well as a high intake of legumes, fresh produce, whole grains, and olive oil. (Esposito *et al*., 2015; Salas-Salvadó *et al*., 2011).

9.2. Low-Carbohydrate Diets

Snorgaard *et al*. (2017) found that some people with type 2 diabetes were able to lose weight, improve their glycemic control, and decrease their medication needs by following a low-carbohydrate diet, which generally restricts carbohydrate consumption to less than 130 g per day. Additional study is necessary to determine the significance of low-carbohydrate diets in diabetic therapy, as there is ongoing dispute on their long-term safety and effectiveness. (van Zuuren *et al*., 2018).

9.3. Intermittent Fasting

People with type 2 diabetes have taken to intermittent fasting, a novel dietary strategy, to help them lose weight and keep their blood sugar levels in control (Corley *et al*., 2018). Increasing insulin sensitivity, decreasing inflammation, and promoting fat loss may be possible with intermittent fasting, according to certain studies (Aranason *et al*., 2017). To find out how to fast most effectively and if intermittent fasting is safe and beneficial for

diabetes therapy in the long run, more study is needed.(Mattson *et al*., 2017).

10. Conclusion

Prevention and management of diabetes mellitus rely heavily on nutrition and dietary control. To better regulate blood sugar levels, encourage weight loss, and lessen the likelihood of diabetes-related problems, individualised medical nutrition therapy should centre on managing carbohydrates, establishing good eating habits, and including certain nutrients. Emerging dietary strategies, such as the Mediterranean diet, low-carbohydrate diets, and intermittent fasting, show promise in diabetes management but require further research to establish their long-term safety and efficacy.

As the global burden of diabetes continues to rise, it is essential for healthcare professionals and researchers to stay informed about the latest developments in diabetes nutrition and to work collaboratively to develop evidence-based dietary guidelines and interventions. By providing comprehensive and individualized nutrition care, we can empower individuals with diabetes to achieve optimal health outcomes and improve their quality of life.

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