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Anemia Across the Spectrum: A Holistic Review of Its Causes, Classification and Control Measures

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Abstract

Background: Anaemia affects more than 1.6 billion individuals worldwide and remains a considerable public health issue, especially in low and middle-income nations. Anaemia, characterized by its diverse causes and clinical manifestations, necessitates a comprehensive diagnosis and therapeutic strategy. This study provides a thorough examination of the types and origins of anaemia, highlighting the importance of combining morphological and etiological categories for improved diagnosis and treatment.

Methodology: A thorough literature review was performed utilizing PubMed, Scopus, and Embase, employing search phrases like “anemia,” “types of anemia,” “etiology of anemia,” “iron deficiency anemia,” “megaloblastic anemia,” and “sickle cell anemia.” Articles published in English from 2010 to 2024 were included.

Results: The results categorize anaemia into seven principal types: iron deficiency, pernicious, hemolytic, sickle cell, thalassaemia, aplastic, and anaemia of chronic disease. Iron deficiency anaemia is the most extensively researched, although non-nutritional variants are inadequately represented, particularly in low-resource environments. Pernicious anaemia is common in elderly people with autoimmune disorders. Genetic disorders such as sickle cell anaemia and thalassemia profoundly impact areas like sub-Saharan Africa. The review examines the ramifications of hemolytic, aplastic, and inflammation-associated anaemia. Diagnostic instruments, such as complete blood counts, iron tests, and vitamin assays, are essential for precise classification and treatment.

Conclusion: The review emphasizes the complex etiology of anaemia, highlighting the necessity of diagnostic instruments such complete blood counts and iron investigations for precise identification of underlying causes. Public health initiatives, such as iron supplementation, deworming, and maternity care, are recommended to alleviate the effects of anaemia. The analysis continues by advocating additional research into non-nutritional factors and region-specific strategies to successfully diminish worldwide anaemia prevalence. This comprehensive approach seeks to inform clinical practices and public health policy in tackling the multifaceted difficulties of anaemia.

Keywords: Anemia; Iron Deficiency; Public Health Strategies; Global Health; Anemia Diagnosis

1. Introduction

The term "anaemia" refers to a significant decrease in the number of red blood cells or a lower-than-normal concentration of haemoglobin, leading to diminished oxygen affinity [1]

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Anaemia is not a singular disease but rather the clinical manifestation of various underlying health conditions. This is a pervasive global public health concern, impacting pregnant women, children under five, and those with weakened immune systems, with increased prevalence noted among residents of low-and middle-income countries[2]. Research indicates that anaemia impacts more than 1.6 billion individuals worldwide and is a major factor in morbidity and mortality among pregnant women and children [3].

Despite extensive research over several decades, anaemia persists as a multifaceted condition characterised by various forms and a wide array of aetiologies, including nutritional deficiencies, chronic illnesses, genetic problems, and haemorrhage. This complexity is a considerable obstacle to precise diagnosis, effective treatment, and strategic prevention, particularly in regions with constrained medical resources[4]. Although prior research has investigated anaemia, a considerable amount of the existing literature is fragmented, concentrating on certain demographics, geographic areas, or forms of anaemia, such as iron deficiency or megaloblastic anaemia. A lack of comprehensive evaluations exists that combine the morphological and etiological classifications of anaemia with contemporary worldwide data. In the absence of a cohesive comprehension of the diverse types of anaemia and its aetiologies, healthcare professionals are prone to misdiagnosing patients, while public health officials may neglect essential risk factors when formulating interventions [5]

This study is justified by the urgent need to unify current knowledge into a singular, coherent resource. This review seeks to address a significant gap in the literature by synthesising data on the kinds and causes of anaemia across various locations and populations. It will facilitate clinical decision-making, guide public health strategies, and establish a basis for future study and policy formulation. The review aims to improve global comprehension of anaemia and the efficacy of initiatives to mitigate its prevalence worldwide.

2. Materials and Methods

A comprehensive systematic literature search was performed in databases such as PubMed, Scopus, Web of Science, National Library of Medicine and Cochrane Library. Search terms encompass "anaemia," "types of anaemia," "causes of anaemia," "iron deficiency anaemia," and "anaemia classification." Boolean operators were employed to enhance search precision. Included were articles published in English and peer-reviewed from 2010 forward. Studies were rejected if they were case reports, non-English, or deficient in methodological rigour. Data was retrieved and thematically synthesised. Figure 1.0 shows the flow chart of the inclusion and exclusion criteria.

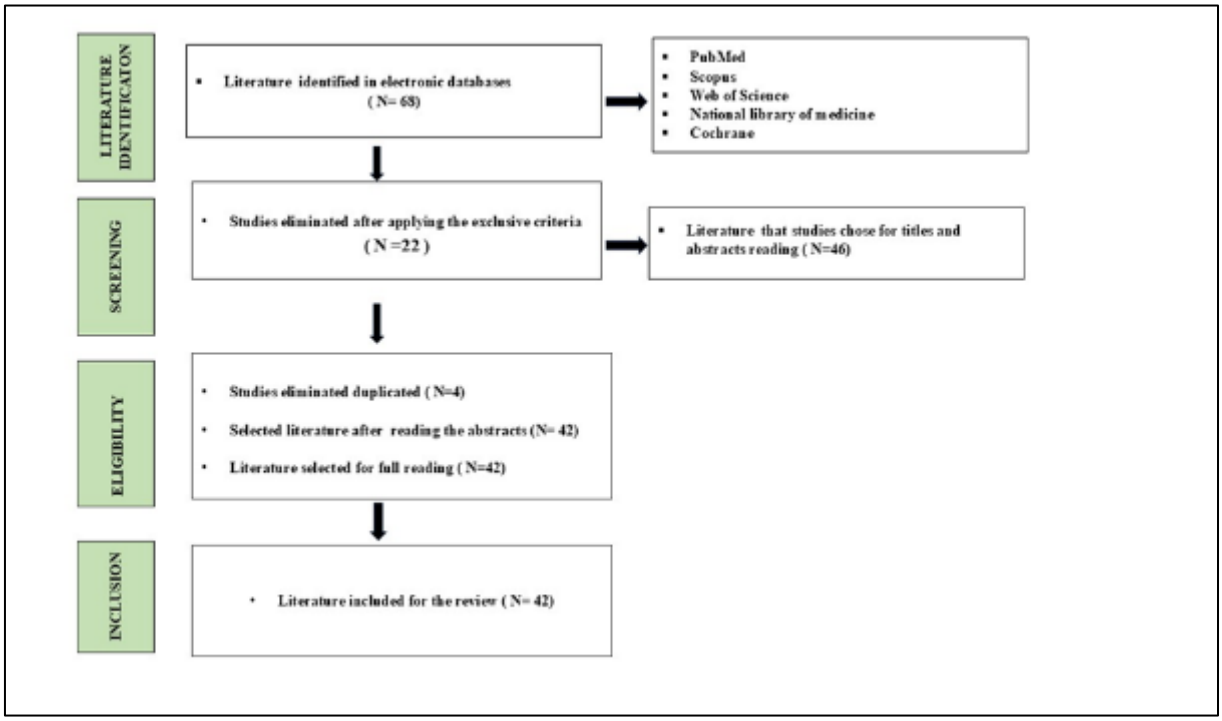

Figure 1 The inclusion and exclusion criteria employed in the studies

2.1. Iron Deficiency Anemia

Despite the abundance of iron on Earth, significant iron deficiency remains one of the foremost contributors to anaemia in humans, which is the leading cause of anaemia globally. Iron is widely recognised for its critical role in various physiological processes in the human body, particularly in haemoglobin synthesis. Iron deficiency anaemia is a condition characterised by subnormal amounts of iron in the blood. This form of anaemia is more common among adolescents and premenopausal women [6]. This condition may result from significant menstrual blood loss, internal gastrointestinal bleeding, or frequent blood donation [7].Anaemia resulting from iron deficiency in the body can occur owing to several factors, as noted by [8] .Iron-deficiency anaemia can be attributed to pregnancy or childhood growth spurts, excessive menstrual bleeding, inadequate iron absorption, gastrointestinal bleeding, dietary insufficiencies (such as an iron-deficient or restricted diet), pharmacological agents (including aspirin, ibuprofen, naproxen, and diclofenac), erythrocyte abnormalities, renal haemorrhage, hookworm infestation, vitamin deficiencies (notably folic acid and vitamin B12), and bone marrow disorders [9]. Prominent symptoms of iron deficiency anaemia encompass fatigue, lethargy, headaches, arrhythmias (palpitations), changed taste perception, oral soreness, and tinnitus. Iron deficiency Anaemia during pregnancy elevates the likelihood of difficulties for both the mother and the infant, including low birth weight, preterm delivery, and postnatal depression. Insufficient iron reserves in the infant may result in anaemia in the newborn [10].

2.2. Pernicious anaemia (PA)

Pernicious anaemia is an autoimmune disorder marked by chronic atrophic gastritis and a deficiency of cobalamin. Pernicious anaemia is the predominant cause of cobalamin deficiency globally. The prevalence significantly differs by age, ranging from 0.1% in the general population to 1.9% among individuals over 60 years old. PA constitutes roughly 20%-50% of the aetiology of CD in adults. The prevalence of PA escalates with age and differs by geographic location. It is often considered quite prevalent in Scandinavian nations[11].

Pernicious anaemia is the predominant cause of vitamin B12 deficiency. Vitamin B12 is vital for survival. New cells must be generated in the body; this is mostly sourced from meat, fish, eggs, and dairy products. A shortage in vitamin B12 can lead to anaemia and other medical disorders. Anaemia may arise from a deficiency of vitamin B12. Pernicious anaemia typically manifests in adults over the age of fifty; it is less prevalent in males than in women and often has a familial tendency. It occurs more often in individuals with other autoimmune disorders [9], [12].

Furthermore, many medications may inhibit the absorption of vitamin B12. Metformin, colchicine, neomycin, and other anticonvulsants employed in epilepsy management are the most quintessential examples. Nervous complications, such as paraesthesia, visual distortions, and instability, and psychological disorders like depression, confusion, memory impairment, or even dementia, may occur. Consequently, chronic or severe vitamin B12 insufficiency may lead to enduring cerebral or neural impairment [13].

2.3. Hemolytic Anemia

Haemolytic anaemia is defined by the premature destruction of red blood cells by the body prior to the completion of their natural lifespan and subsequent removal from the bloodstream. Haemolytic anaemia can impact individuals of any age, race, or gender[14], [15]. The consequences of haemolytic anaemia encompass weariness, discomfort, arrhythmia, cardiomegaly, and the potential for heart failure if the condition remains untreated. Traditionally, haemolytic anaemia is categorised into two classes: inherited or genetic abnormalities that impact the erythrocyte cytoskeleton, resulting in a spectrum of diseases with diverse genetic and phenotypic traits[14]. These diseases can vary from asymptomatic states that may only manifest during a haemolytic or aplastic crisis induced by a viral infection to severe congenital haemolytic anaemias necessitating regular blood transfusions. Diseases resulting from abnormalities in the erythrocyte cytoskeleton exhibit considerable variability in genetics and phenotypes, ranging from complete asymptomatic states until a haemolytic or aplastic crisis is provoked by a viral infection, to severe congenital forms necessitating lifelong transfusions[16].

Sickle cell anaemia, thalassaemia, hereditary spherocytosis, hereditary elliptocytosis, stomatocytosis (HSt), glucose-6 phosphate dehydrogenase (G6PD) deficiency, and pyruvate kinase insufficiency are the most common erythrocyte cytoskeleton disorders[9]. Acquired haemolytic anaemias encompass immunological haemolytic anaemia, autoimmune haemolytic anaemia, alloimmune haemolytic anaemia, and drug-induced haemolytic anaemia. Mechanical haemolytic anaemias, paroxysmal nocturnal haemoglobinuria, specific diseases, and various chemicals can also impair red blood cells and result in haemolytic anaemia [17]. Fatigue is the principal symptom of haemolytic anaemia, resulting from insufficient oxygen supply. Additional symptoms encompass jaundice, upper abdominal pain, leg ulcers and tenderness, as well as a pronounced reaction to a blood transfusion. Therapeutic interventions for haemolytic anaemia encompass

blood transfusions, pharmacological agents, plasmapheresis, surgical procedures, haematopoietic stem cell transplants, and modifications in lifestyle[14].

2.4. Sickle cell anaemia

Sickle cell anaemia is a hereditary illness resulting from haemoglobin abnormalities. Haemoglobin diseases are hereditary blood conditions resulting from mutations in haemoglobin genes inherited from both parents [18]. Nwabulo and colleagues have argued that sickle cell disease (SCD) is an autosomal recessive genetic illness of red blood cells that is inherited from parent carriers. It is a prevalent hemoglobinopathy of significant public health concern. Sickle Cell Disease is mostly caused by a single nucleotide mutation that impacts the production of glutamic acid. The specified mutation leads to the substitution of glutamic acid with valine at the sixth position in the beta-globin amino acid sequence.

In its deoxygenated form, the sickle red blood cell exhibits increased stickiness and forfeits the physiological characteristics of a normal red blood cell, resulting in a series of issues that can trigger sickle cell crises and consequences [19].

Annually, around 300,000 infants worldwide are documented to have developed serious haemoglobin abnormalities [20]. Sub-Saharan Africa accounts for approximately 75% of the global burden of sickle cell disease (SCD). In Ghana, 2% (about 15,000) of neonates are affected by sickle cell disease (SCD), with 55% exhibiting the homozygous variant inherited from both parents [20] In Nigeria, 100,000 to 150,000 infants are diagnosed with SCD each year, representing 33% of the global burden of the disease. In percentage terms, Ghana's SDC load exceeds that of Nigeria[18].

Sickle cell anaemia is a hereditary chronic condition, notably prevalent in individuals of African, South or Central American, Caribbean, Mediterranean, Indian, and Saudi Arabian ancestry. The clinical manifestations of sickle cell disease encompass acute pain crises, anaemia, recurrent infections, and persistent end-organ damage [20]

2.5. Thalassemia

Thalassemia is an inherited blood disorder characterized by the reduced production of healthy red blood cells and hemoglobin. It results from genetic mutations that impair the synthesis of one or more globin chains, which are essential components of hemoglobin [21]. The disorder is broadly classified into two major types: alpha-thalassemia and beta-thalassemia. Alpha-thalassemia major, also known as hydrops fetalis, represents the most severe form of alpha-thalassemia, while the severe form of beta-thalassemia is referred to as beta-thalassemia major or Cooley's anemia. These conditions affect both males and females and are more commonly observed in individuals of Mediterranean, Middle Eastern, Asian, and African descent.

Hemoglobin is composed of two types of protein chains: alpha and beta globin. Inadequate production of these chains leads to abnormal red blood cell formation and reduced oxygen-carrying capacity. The synthesis of these globin chains is regulated by genes, and thalassemia is passed from parents to their offspring through autosomal recessive inheritance [22][23] Symptoms arise due to insufficient oxygen delivery to tissues and vary depending on the severity of the disorder. Individuals with mild forms of alpha or beta thalassemia may experience minimal symptoms, while those with intermediate or major forms can suffer from moderate to severe anemia, bone deformities, delayed growth and puberty, and an enlarged spleen[23].

More severe cases, such as beta-thalassemia major or hemoglobin H disease, often present with additional complications, including jaundice, dark urine, poor appetite, hepatosplenomegaly, and cardiac abnormalities [24]. Standard treatment options for moderate to severe thalassemia include regular blood transfusions, iron chelation therapy to prevent iron overload, and folic acid supplementation to support red blood cell production.

2.6. Aplastic anaemia

Aplastic anemia is a blood disorder in which the body's physiological mechanism does not allow the bone marrow to produce enough new blood cells. This condition may result in a number of health-related problems, including arrhythmias, cardiomegaly, heart failure, infections, and bleeding. Impairment to the bone marrow's stem cells causes aplastic anemia[14].

A number of acquired diseases, conditions, and factors contribute to the cause of aplastic anemia, including toxins such as pesticides, arsenic, and benzene; radiation and chemotherapy; medicines such as chloramphenicol; infectious diseases such as hepatitis and Epstein-Barr virus, cytomegalovirus, parvovirus B19, and HIV; and autoimmune

disorders such as lupus and rheumatoid arthritis [9]. Inherited conditions such as Fanconi anemia, Schwachman-Diamond syndrome, dyskeratosis, and Diamond-Blackfan anemia may also cause aplastic anemia [15].

Results reported by Escalante *and others* have suggested that the most common symptoms of aplastic anemia are fatigue, breathing weakness, dizziness, headaches, cold hands and feet, soft skin, mouth, and nails, and chest pain.

Treatment of aplastic anemia includes blood transfusion, blood and bone marrow stem cell transplantation, and medications. These therapies can prevent or limit complications, alleviate symptoms, and improve the quality of life. Transplantation of blood and bone stem cells can cure aplastic anemia disorders[25].

2.7. Anemia caused by chronic diseases

Anemia of chronic disease (ACD) or anemia of inflammation develops during an inflammatory process, which triggers the immune system's activation, the release of cytokines, and an increase in hepcidin. This process causes a drop in plasma iron levels and the suppression of erythropoiesis, which leads to anemia of chronic disease (ACD) or anemia of inflammation [26]. It is often characterized as either normocytic, normochromic, or hypoproliferative, with a hemoglobin level between 8 and 12 g/dL. ACD is widely recognized as the second most widespread anemia, following iron deficiency anemia, and it is particularly prevalent among the elderly population. Approximately one-third of the elderly population who exhibit symptoms of anemia are diagnosed with anemia of chronic disease (ACD). According to [27], this type of anemia is commonly observed in hospitalized patients and individuals with chronic conditions. The global prevalence of anemia is believed to be as high as 40%, with a significant proportion attributed to anemia of chronic disease (ACD) either as a standalone condition or in conjunction with other forms of anemia. The role of ACD in this overall prevalence is of considerable significance[26].

Rheumatoid arthritis, systemic lupus erythematosus, vasculitis, sarcoidosis, inflammatory bowel disease, neoplastic diseases, chronic kidney disease (CKD), acute/chronic bacterial infections, fungal, viral, and parasitic diseases, as well as chronic rejection of organ transplants, respiratory failure, heart failure, obesity, and other chronic processes, are all potential generators of ACD. [26], [27]. Elevated hepcidin levels in infections represent a host defense mechanism against infections because it limits the availability of iron to the microorganisms [28].

3. Causes of Anemia

3.1. Genetic

Anemia can be brought on by genetic red blood cell diseases that affect the generation, structure, or function of red blood cells. According to [29], genetic red blood cell disorders such as thalassemia and the thalassemia trait, sickle cell disorders and the sickle cell trait, glucose-6-phosphate deficiency, other hemoglobinopathies and hemolytic anemias, and Krüppel-like factor 1 variant are responsible for about 11% of anemia globally[30].

Even though all populations have inherited red blood cell abnormalities, the frequency of anemia varies substantially within and between various countries, even across small geographical [22], [29]. Populations in or originating from Africa, the Middle East, and Asia have the greatest incidences. Sickle cell disease, hemolytic anemias, and G6PD deficiency all enhance the destruction of red blood cells through various mechanisms, whereas thalassemias result in the production of red blood cells that are inefficient and have a shorter lifespan[31].

Genetic red blood cell abnormalities are immutable risk factors for anemia, yet thalassemia prevention and treatment are relatively well-developed in many Asian nations. Although the knowledge and resources for managing hereditary red blood cell abnormalities are severely lacking in many nations, collaborations are being formed to enhance management and therapy[22].

3.2. Environmental

Exposure to air pollutants, such as NO_2 and $PM_{2.5}$ (fine particulate matter), is one possible factor in the development of anemia. Systemic inflammation has been demonstrated to rise in response to exposure to $PM_{2.5}$ and NO_2 [32], [33] and affect bone marrow stimulation, particularly in those individuals who have conditions associated with chronic inflammation, such as diabetes or obesity [34]. Many of these studies have reported on the short-term changes in circulating inflammatory marker levels, but new research suggests that exposure to air pollutants over an extended period may also result in chronic and self-perpetuating systemic inflammation [33].

These results suggest that air pollution, through its effects on systemic inflammation, may cause a cascade of events including downregulation of erythropoietin production, exacerbation of hematopoietic precursors' refractoriness to endogenous erythropoietin, and chronic and sustained upregulation of hepcidin, an iron regulatory protein, each of which can result in decreased hemoglobin and anemia. Similar trends have been reported by Das & Chatterjee in their work on the assessment of the hematological profiles of adult male athletes from two different air pollutant zones in West Bengal.

Das and Chatterjee have indicated that an individual's reaction to air pollutants depends on the type of pollutant to which an individual is exposed, the degree of exposure, and the concentration of the chemicals [35]Prolonged exposure to air pollution at low concentrations may induce anemia in people [36]. The study further indicated that many components of airborne pollutants have the propensity to reach the blood rapidly without previously being biotransformed; moreover, the hematopoietic system is extremely vulnerable to air pollutants since its cells undergo constant restructuring. Anemia may result from red blood cell destruction from toxic airborne pollutants [36]. The biological processes through which air pollution may limit the formation of red blood cells are evident in the synthesis of heme, the formation of red blood cells, and their lifespan [33], [35].

3.3. Physiological

3.3.1. Heavy Menstrual Bleeding (HMB)

Studies have reported that there is a relationship between heavy menstrual bleeding and iron deficiency anemia [37], [38]. Heavy menstrual bleeding is a contributing factor to significant blood loss during the menstrual period, which in turn can cause the body's levels of iron to be depleted, as indicated by [38] Iron is necessary for the production of red blood cells, and when these levels are below the normal range, it can result in an individual being diagnosed with iron deficiency anemia [39]. In a study by[40], they objectively defined heavy menstrual bleeding as the loss of 80 ml or more blood during every menstrual cycle. In contrast, [39] have reported that the National Institute for Health and Care Excellence in the United Kingdom has given a more refined definition, which defines HMB as excessive menstrual blood loss that physically, emotionally, socially, and financially affects the quality of life of women and can be seen by itself or with other accompanying symptoms[41].

3.3.2. Hereditary red blood cell enzymopathies

Hereditary non-spherocytic hemolytic anemia (HNSHA) is a group of rare disorders that affect the red blood cells. These disorders are caused by mutations in genes that encode enzymes involved in the metabolism of red blood cells. The enzymes help to maintain the integrity and function of the red blood cells, and their deficiency leads to premature destruction of the cells [42]. This results in anemia, which is a condition of low red blood cell count or hemoglobin level. In comparison to other hereditary conditions affecting red blood cells, such as membrane disorders or hemoglobinopathies, the shape of the red blood cell does not exhibit any distinct abnormalities. The diagnosis relies on detecting reduced activity of specific enzymes and characterizing the abnormality at the DNA level. Deficiencies in glucose-6-phosphate dehydrogenase (G6PD) and pyruvate kinase (PK) are the most frequent enzyme diseases. Hereditary non-spherocytic hemolytic anemia can also be caused by a number of different enzyme abnormalities, albeit these are often far less well-publicized.

Some examples of these rare enzyme conditions are deficiencies of hexokinase, phosphofructokinase, triosephosphate isomerase, aldolase A, glyceraldehyde-3-phosphate dehydrogenase (GAPDH), phosphoglycerate kinase (PGK), diphosphoglycerate mutase (BPGM), enolase (ENO), pyrimidine 5'-nucleotidase (P5N), adenylate kinase (AK), and glutathione reductase (GR). The clinical manifestations, inheritance patterns, and treatment options may vary depending on the type and severity of the enzyme defect. Some patients may have mild or asymptomatic anemia, while others may experience severe hemolysis, jaundice, gallstones, splenomegaly, and chronic transfusion dependency[42].

3.4. Nutritional

A primary cause of anemia is nutritional deficiency, referring to the body's inability to obtain necessary nutrients from the diet. Nutrient deficiency and insufficient bioavailability of hemopoietic nutrients for adequate Hb concentration as well as for erythrocyte synthesis can result in nutritional anemia. The bioavailability of hemopoietic nutrients such as iron, vitamin B12, folic acid, and ascorbic acid gets affected in contact with heat and light [43]. The bioavailability of elemental non-heme iron can be reduced by a number of different variables, including polyphenols, cinnamon, phytase in whole grains and legumes, and calcium. Some of the commonly known nutrients that are needed for the production and function of red blood cells are iron, vitamin B12, and folate [44].

Iron deficiency anemia is the most common type of anemia worldwide. Iron is an essential component of hemoglobin, the protein that carries oxygen in the blood. When there is not enough iron in the body, hemoglobin production is reduced and the red blood cells become small and pale.

Iron deficiency can result from inadequate intake of iron-rich foods, such as meat, eggs, and leafy green vegetables, or increased loss of iron due to bleeding, menstruation, pregnancy, or parasitic infections [45], [46].

Vitamin B12 deficiency anaemia is also known as pernicious anemia. Vitamin B12 is a cofactor for several enzymes that are involved in the synthesis of DNA and the maturation of red blood cells. Vitamin B12 is primarily found in animal products, such as meat, eggs, and dairy products. Therefore, vegans and vegetarians are at risk of developing this type of anemia if they do not supplement their diet with vitamin B12 or consume fortified foods. Vitamin B12 deficiency can also occur due to malabsorption of the vitamin in the digestive tract, which can be caused by diseases such as celiac disease, Crohn's disease, or atrophic gastritis [47].

Folate deficiency anemia has similarities to vitamin B12 deficiency anemia in relation to its impact on the production of red blood cells and synthesis of DNA. Folate is another vitamin B that is essential for the formation and growth of red blood cells. Folate is found in various plant foods, such as fruits, vegetables, legumes, and nuts. Folate deficiency can result from inadequate intake of these foods or increased demand for folate due to pregnancy, lactation, or rapid growth. Folate deficiency can also be caused by certain medications that interfere with folate metabolism, such as methotrexate, sulfasalazine, or phenytoin[48], [49].

However, Shubham *et al.* share the view that nutritional causes of anemia can be prevented and treated by consuming a balanced diet that includes foods rich in iron, vitamin B12, and folate, or by taking supplements as prescribed by a physician. Nutritional anemia can have serious consequences on health and quality of life if left untreated. Therefore, it is important to recognize the signs and symptoms of anemia and seek medical attention if they occur [44].

4. Diagnostic Approaches for anemia

Anemia is diagnosed with a complete blood count, reticulocyte count, peripheral smear, and iron tests. Depending on the suspected cause, additional tests such as vitamin B12 and folate levels, hemoglobin electrophoresis, and a bone marrow biopsy may be necessary.

5. Public Health and Clinical Implications

Understanding the complex nature of anemia is critical for effective treatment. Iron supplements, deworming, nutrition education, and maternal care are all critical methods. Clinicians must distinguish between various types for effective management, while policymakers require integrated methods customized to regional needs.

6. Discussion

The review emphasizes the variability of anemia and the importance of thorough diagnostic and therapeutic approaches. While much is known about iron deficiency anemia, there is less focus on non-nutritional causes in low-resource settings. Further research should investigate context-specific risk variables and intervention outcomes. Anemia is a complex and pervasive condition with many root causes and types. Comprehensive understanding and context-specific interventions are critical for reducing the global burden. This study underlines the importance of comprehensive diagnostic approaches and evidence-based public health strategies.

7. Conclusion

The review emphasizes the complex etiology of anaemia, highlighting the necessity of diagnostic instruments such complete blood counts and iron investigations for precise identification of underlying causes. Public health initiatives, such as iron supplementation, deworming, and maternity care, are recommended to alleviate the effects of anaemia. The analysis continues by advocating additional research into non-nutritional factors and region-specific strategies to successfully diminish worldwide anaemia prevalence. This comprehensive approach seeks to inform clinical practices and public health policy in tackling the multifaceted difficulties of anaemia.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

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