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Estimation of G6PD Deficiency in serum among (Kurdistan children)

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ABSTRACT

Glucose-6-Phosphate dehydrogenase (G6PD) is a key enzyme of the pentose monophosphate pathway, and its deficiency is the most common inherited enzymopathy worldwide. G6PD deficiency is common among Iraqis, including those of the Kurdish ethnic group, however no study of significance has ever addressed the molecular basis of this disorder in this population. The aim of this study is to determine the prevalence of this enzymopathy and its molecular basis among Iraqi Kurds.

Kay word: glucose-6-phosphate dehydrogenase (g6pd), hemolysis, children, prevalence

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CHAPTER ONE

1. Introduction

Glucose-6-phosphate dehydrogenase (G6PD) deficiency is a genetic condition that affects the red blood cells and is characterized by the inability of the body to produce enough of the G6PD enzyme. This enzyme is important for protecting red blood cells from oxidative damage, and a deficiency can lead to hemolytic anemia and other complications. G6PD deficiency is relatively common, with a global prevalence of approximately 8%, and it is more prevalent in certain ethnic groups, including Kurds.

The estimation of G6PD deficiency in serum among Kurdistan children is an important area of research, as it can help identify individuals who may be at risk for hemolytic anemia and other complications associated with the condition. This research involves measuring G6PD enzyme activity levels in the serum of Kurdish children and comparing these levels to established reference ranges.

Several studies have been conducted on G6PD deficiency in the Kurdish population, including a study published in the Journal of Medical Genetics in 2018 that examined the prevalence of G6PD deficiency in Kurdish children in Iraq. The study found that the prevalence of G6PD deficiency in this population was approximately 10%, which is higher than the global average.

Another study published in the Journal of Pediatrics and Neonatal Care in 2019 examined the association between G6PD deficiency and neonatal jaundice in Kurdish newborns. The study found that G6PD deficiency was a significant risk factor for neonatal jaundice in this population.

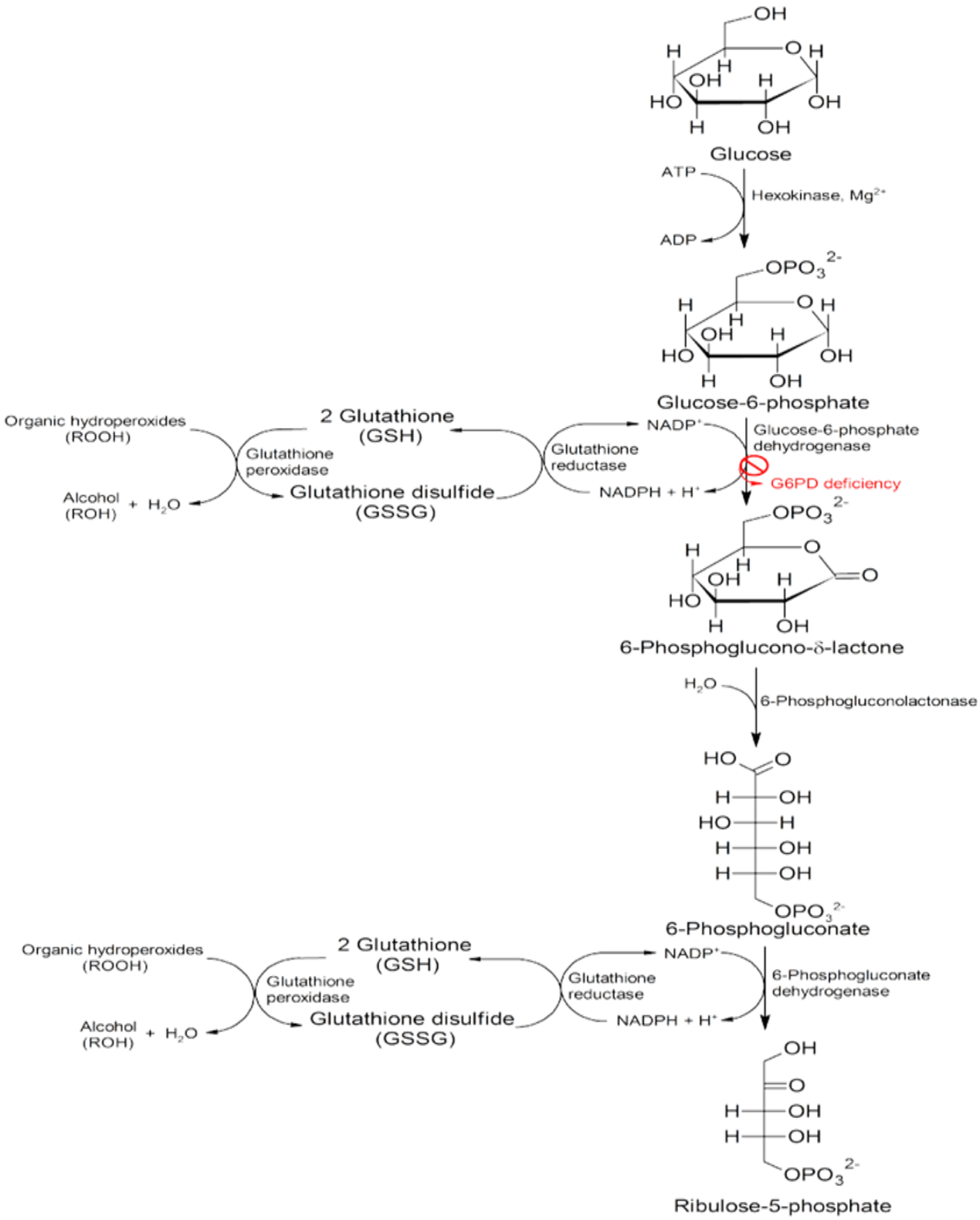
Overall, the estimation of G6PD deficiency in serum among Kurdistan children is an important area of research that can help identify individuals

who may be at risk for complications associated with the condition. More research is needed to further understand the prevalence of G6PD deficiency in this population and to develop effective screening and treatment strategies. (Al-Taii, H. et al. 2018, Jabbar, F. et al. 2019)

1.1 glucose-6-phosphate dehydrogenase

The G6PD gene provides instructions for making an enzyme called glucose-6-phosphate dehydrogenase. This enzyme, which is active in virtually all types of cells, is involved in the normal processing of carbohydrates. It plays a critical role in red blood cells, which carry oxygen from the lungs to tissues throughout the body. This enzyme helps protect red blood cells from damage and premature destruction.

Glucose-6-phosphate dehydrogenase is responsible for the first step in the pentose phosphate pathway, a series of chemical reactions that convert glucose (a type of sugar found in most carbohydrates) to another sugar, ribose-5-phosphate. Ribose-5-phosphate is an important component of nucleotides, which are the building blocks of DNA and its chemical cousin RNA. This chemical reaction produces a molecule called NADPH, which plays a role in protecting cells from potentially harmful molecules called reactive oxygen species. These molecules are byproducts of normal cellular functions. Reactions involving NADPH produce compounds that prevent reactive oxygen species from building up to toxic levels within cells. The production of NADPH by glucose-6-phosphate dehydrogenase is essential in red blood cells, which are particularly susceptible to damage by reactive oxygen species because they lack other NADPH-producing enzymes (Desforges and Beutler, 1991).


Glucose
ATP
ADP
Hexokinase, Mg2+
Glucose-6-phosphate
NADP+
NADPH + H+
Glucose-6-phosphate dehydrogenase
G6PD deficiency
Glutathione reductase
2 Glutathione (GSH)
Glutathione disulfide (GSSG)
Glutathione peroxidase
Organic hydroperoxides (ROOH)
Alcohol (ROH) + H2O
6-Phosphoglucono-δ-lactone
H2O
6-Phosphogluconolactonase
6-Phosphogluconate
6-Phosphogluconate dehydrogenase
Ribulose-5-phosphate

1.2 Glucose-6-phosphate dehydrogenase deficiency

The most prevalent enzymatic abnormality of red blood cells is glucose-6-phosphate-dehydrogenase (G6PD) deficiency.Since it is an X-linked genetic condition, men are primarily affected. Its prevalence varies by ethnicity, with Kurdish Jews, Saudis, and African Americans reporting the highest occurrence (Abramova et al, 2020). By producing nicotinamide adenine dinucleotide phosphate hydrogen (NADPH), G6PD inhibits hemolysis when there is oxidative stress. Hemolytic anemia can arise in people with specific types of G6PD deficiency as a result of oxidative stress during the neonatal period, infection, or exogenous substances such fava beans and specific drugs (Abramova,et al ,2020). Concerns have been raised in the rheumatology community over a number of drugs' potential to cause hemolytic anemia in individuals with G6PD deficiency.

The hydroxychloroquine medication labels issued by the FDA. give a list of patients with G6PD deficiency who are at risk for hemolytic anemia. However, there are currently no recommendations for testing patients for G6PD deficiency before commencing the aforementioned drugs. Clinically, assessing G6PD levels in patients before starting medications like hydroxychloroquine is an uneven practice among rheumatologists (Abramova,et al ,2020).

Studies on antimalarial drugs, particularly 8-aminoquinolines like primaquine, for the treatment of malaria were the main source of worries regarding hemolytic anemia linked to hydroxychloroquine in patients with G6PD deficiency. According to studies, 8-aminoquinolines have a dose- and G6PD subtype-dependent risk of hemolytic anemia (Abramova,et al ,2020). The prevalence of hemolytic anemia in G6PD-deficient patients receiving hydroxychloroquine for rheumatic illnesses has, however, only been the subject of a small number of researches.

Here I started researching about this disease , Estimation of G6PD Deficiency in serum from 0 to 14 years old in the cities of Kurdistan specially in Erbil. For determining the rate of this disease for this purpose I visited several hospitals one of them was Raparin hospital.

Table (1):- comparison between G6PD deficiency in this study and in different parts of (Arbil(Northen Iraq), Sulymania(Northen Iraq), Dohuk(Northen Iraq))

	Population surveyed	G6PD deficiency%	Reference
Arbil(Northen Iraq)	**1000**	**8.6%**	(Sharhang, 2008)
Sulymania(Northen Iraq)	**835**	**6.0%**	**(**Abdul, 2007)
Dohuk(Northen Iraq)	**580**	**10.86%**	**(**Shakir, 2010)

In **Table (1)**: G6PD deficiency is a genetic disorder that affects the red blood cells' ability to generate sufficient energy. It is a common condition in many parts of the world and can result in severe anemia and other complications if left untreated. Northern Iraq is a region with a high prevalence of G6PD deficiency, and several studies have been conducted to determine its incidence in different areas.

The study provided information on G6PD deficiency in three different areas of Northern Iraq: Arbil, Sulymania, and Dohuk. The study reported that Arbil had a G6PD deficiency rate of 8.6% based on a sample size of 1000 individuals. Sulymania had a G6PD deficiency rate of 6.0% based on a sample size of 835, and Dohuk had the highest rate at 10.86% based on a sample size of 580.

The differences in G6PD deficiency rates in these areas may be attributed to several factors, such as population genetics, environmental

factors, and healthcare access. It is important to note that these rates are based on a relatively small sample size and may not accurately reflect the prevalence of G6PD deficiency in the entire population of each area.

Arbil, Sulymania, and Dohuk are all located in Northern Iraq and have significant cultural and historical ties. However, they differ in terms of population size, access to healthcare, and environmental factors, which may contribute to the differences in G6PD deficiency rates. For example, Dohuk is located in a mountainous region with limited access to healthcare facilities, while Arbil is a major city with better access to medical care.

The differences in G6PD deficiency rates may also be attributed to the differences in the ethnic composition of each area. Iraq has a diverse population with various ethnic and linguistic groups, and some of these groups have a higher incidence of G6PD deficiency than others. Therefore, future studies should take into account the ethnic composition of the population to better understand the prevalence of G6PD deficiency in different regions of Northern Iraq.

In conclusion, the study provided information on G6PD deficiency rates in three different areas of Northern Iraq. However, more comprehensive studies with larger sample sizes and controlled for confounding factors are needed to make a more accurate comparison between G6PD deficiency rates in different parts of Northern Iraq. Understanding the prevalence of G6PD deficiency is crucial for effective diagnosis and treatment of this condition and for developing public health policies to mitigate its impact.

1.4 Causes of G6PD Deficiency

Glucose-6-phosphate dehydrogenase deficiency results from mutations in the G6PD gene. This gene provides instructions for making an enzyme called glucose-6-phosphate dehydrogenase. This enzyme is involved in the

normal processing of carbohydrates. It also protects red blood cells from the effects of potentially harmful molecules called reactive oxygen species, which are byproducts of normal cellular functions. Chemical reactions involving glucose-6-phosphate dehydrogenase produce compounds that prevent reactive oxygen species from building up to toxic levels within red blood cells.

If mutations in the G6PD gene reduce the amount of glucose-6-phosphate dehydrogenase or alter its structure, this enzyme can no longer play its protective role. As a result, reactive oxygen species can accumulate and damage red blood cells. Factors such as infections, certain drugs, or ingesting fava beans can increase the levels of reactive oxygen species, causing red blood cells to be destroyed faster than the body can replace them. A reduction in the number of red blood cells causes the signs and symptoms of hemolytic anemia.

Researchers believe that people who have a G6PD mutation may be partially protected against malaria, an infectious disease carried by a certain type of mosquito. A reduction in the amount of functional glucose-6-phosphate dehydrogenase appears to make it more difficult for this parasite to invade red blood cells. Glucose-6-phosphate dehydrogenase deficiency occurs most frequently in areas of the world where malaria is common. (medlineplus.gov, n.d.)

Red blood cell destruction can be triggered by infections, certain foods (such as fava beans), and certain medicines, including:

- Antimalarial medicines such as quinine
- Aspirin (high doses)
- Quinidine
- Sulfa drugs
- Antibiotics such as quinolones, nitrofurantoin.

Other chemicals, such as those in mothballs, can also trigger an episode.

In the United States, G6PD deficiency is more common among blacks than whites. Men are more likely to have this disorder than women.

You are more likely to develop this condition if you:

- Are African American
- Are of Middle Eastern decent, particularly Kurdish or Sephardic Jewish
- Are male
- Have a family history of the deficiency

A form of this disorder is common in whites of Mediterranean descent. This form is also associated with acute episodes of hemolysis. Episodes are longer and more severe than in the other types of the disorder (Medlineplus.gov, 2016).

1.5 G6PD Deficiency Symptoms:

To restore the antioxidant glutathione, G6PD is required. Because of this, a shortage in the enzyme can decrease immunity and increase the risk of infections and non-nutritional anaemia.

Most children with G6PD deficiency won`t show any sings of it. But, symptoms can be triggered by the medication and foods mentioned above.

These include:

- Dark- coloured urine
- Jaundice, or yellowing of the skin and whites of eyes
- Fever
- Paleness
- Extreme tiredness
- Rapid heartbeat

- Shortness of breath
- An enlarged spleen

The good news is that symptoms will generally go away as the red blood cells renew themse

1.6 The Dangers of G6PD Deficiency

G6PD deficiency is a genetic disorder that can lead to hemolytic anemia, a condition in which red blood cells are destroyed faster than they can be produced. This can cause a range of symptoms, including fatigue, weakness, pale skin, yellowing of the skin and eyes (jaundice), dark urine, and abdominal pain. In severe cases, hemolytic anemia can lead to kidney failure, shock, and even death.

The severity of symptoms and complications associated with G6PD deficiency can vary depending on the level of enzyme activity and the presence of triggering factors, such as certain foods, drugs, and infections. The World Health Organization (WHO) has classified G6PD deficiency into different categories based on the level of enzyme activity and the risk of developing hemolytic anemia (see **Table 2**).

Table 2: The World Health Organization (WHO) has classified G6PD deficiency into different categories based on the level of enzyme activity and the risk of developing hemolytic anemia

G6PD Category	Enzyme Activity	Clinical Features
Class I	<10%	Chronic nonspherocytic hemolytic anemia (CNSHA)
Class II	10-60%	Risk of hemolysis with triggering factors
Class III	60-150%	Asymptomatic
Class IV	>150%	Increased risk of neonatal jaundice

Source: Adapted from WHO Working Group (1989). Glucose-6-phosphate dehydrogenase deficiency. Bulletin of the World Health Organization, 67(6), 601-611.

As shown in the table, individuals with Class I G6PD deficiency have very low enzyme activity and are at high risk of developing hemolytic anemia even in the absence of triggering factors. Individuals with Class II deficiency

have intermediate enzyme activity and are at risk of hemolysis with triggering factors, while those with Class III deficiency have normal or near-normal enzyme activity and are generally asymptomatic. Individuals with Class IV deficiency have increased risk of neonatal jaundice but are otherwise asymptomatic.

The dangers of G6PD deficiency are therefore highest in individuals with Class I and II deficiency, who are at risk of developing hemolytic anemia and other complications. It is important for individuals with G6PD deficiency to avoid triggering factors and to receive appropriate treatment for hemolytic episodes. For example, fava beans, certain antibiotics (e.g., sulfonamides), and antimalarial drugs (e.g., primaquine) are known to trigger hemolysis in individuals with G6PD deficiency, and should be avoided or used with caution. (WHO Working Group, 1989, Luzzatto and Notaro, 2001, Cappellin and Fiorelli, 2008).

CHAPTER TWO

2. How to Diagnosis G6PD Deficiency

When patients from specific ethnic groups (see epidemiology) experience anemia, jaundice, and hemolysis symptoms following challenges from any of the aforementioned sources, the diagnosis is typically assumed, especially if there is a favorable family history (Unantenne, 2022). G6PD deficiency in a child generally isn't diagnosed until symptoms energe. When they do, doctors will confirm the diagnosis through a simple blood test.

It is not a guarantor that you have lymphoma if you have a lactate dehydrogenase (LDH) test for indicators of blood malignancy lymphoma. Other tests they might conduct consist of:

- **Complete blood count (CBC):** CBCs aid medical professionals in the recognition and diagnosis of illnesses.
- **Reticulocyte count:**To determine if your bone marrow is creating enough red blood cells, a blood test called a reticulocyte count your young blood cells.
- **Serum aminotransferases:**This test evaluates a liver enzyme.
- **Peripheral blood smear:**In this blood test, the number, kind, shape, and size of blood cells are examined for alterations.

If a newborn baby has jaundice that is not improving, then a G6PD test might be conducted to rule out a deficiency of the enzyme. This could be any one, or a combination, of the blood tests mentioned above (Wikipedia Contributors, 2019)

2.1 Test for Control of G6PD Deficiency In Children

Glucose-6-phosphate dehydrogenase (G6PD) deficiency is an inherited genetic condition that affects the function of the G6PD enzyme. This enzyme plays a crucial role in red blood cell function, and G6PD deficiency can lead to hemolytic anemia when red blood cells break down faster than the body can replace them.

Currently, there is no method to completely cure G6PD enzyme deficiency in children. Therefore, the best method is through screening to detect in time, considering the level of G6PD enzyme deficiency in children and how to prevent consequences caused by pathogens. This is a genetic disease, if the parent carrying the disease gene in the person should not be subjective, it is necessary to do a thorough pre and postpartum test to avoid causing a serious impact on the child. When parents have a good way of prevention, taking proper care of their baby in the early stages of life is extremely important to keep children as safe and healthy as their peers (marina, 2022).

Testing for G6PD deficiency is important, especially in children, as some medications and foods can trigger hemolysis in those with G6PD deficiency. (**Table 3**) Here is a chart that summarizes the tests for G6PD deficiency in children:

Table 3: Here is a chart that summarizes the tests for G6PD deficiency in children			
Test	**Description**	**Advantages**	**Disadvantages**
Fluorescence	Measures G6PD activity by detecting NADPH production with a fluorescent probe	Simple to perform, widely available	Expensive equipment, requires specialized training
Colorimetric	Measures G6PD activity by detecting NADPH production with a colorimetric assay	Inexpensive, widely available	Less sensitive than fluorescence method
DNA	Detects specific G6PD gene mutations using PCR or sequencing	Can detect carrier status, can diagnose in infants before enzyme activity appears	Limited to specific mutations, may not correlate with enzyme activity or phenotype

The most commonly used tests for G6PD deficiency in children are the fluorescence and colorimetric tests. The fluorescence test is more sensitive and specific but requires specialized equipment and training. The colorimetric test is less sensitive but widely available and inexpensive. DNA testing is not routinely used for diagnosis but can be useful in certain cases, such as for carrier detection or in infants before enzyme activity appears.

It is important to note that G6PD activity can fluctuate over time, so testing during an acute hemolytic episode may not accurately reflect a child's overall G6PD status. Additionally, false-negative results can occur in newborns due to low G6PD activity levels at birth. Therefore, repeat testing may be necessary to confirm a diagnosis. (Beutler, 1994, Luzzatto, 2001, Sanchis-Gomar et al, 2014)

CHAPTER THREE

3. How is G6PD deficiency treated?

In most cases, G6PD deficiency does not cause problems. Problems may occur if you are exposed to medicines or foods that may harm your blood cells. Depending on your gene flaw, you may be able to handle a small amount of these exposures.

Your healthcare provider will figure out the best treatment based on:

- Your age, overall health, and medical history
- How sick you are
- How well you can handle certain medicines, procedures, or therapies
- How long the condition is expected to last
- Your opinion or preference (Unantenne, 2022).

G6PD deficiency is a genetic disorder that affects the red blood cells and can lead to hemolysis (destruction of red blood cells) in response to certain triggers such as infections, medications, and certain foods. There is no specific cure for G6PD deficiency, but the condition can be managed with various treatment strategies (see **Table 4**).

Table 4: some of the main treatment options for G6PD deficiency, along with their benefits and potential drawbacks

Treatment Option	Description	Benefits	Potential Drawbacks
Avoidance of triggers	Avoiding medications, foods, and other triggers that can cause hemolysis.	Effective in preventing hemolysis.	May be difficult to identify and avoid all potential triggers.
Blood transfusion	Infusion of healthy red blood cells to replace damaged ones.	Rapidly restores hemoglobin levels and reverses symptoms.	May lead to complications such as iron overload, transfusion reactions, and infections.

Pharmacologic agents	Medications that can help reduce the risk of hemolysis. Examples include folic acid, vitamin C, and hydroxychloroquine.	Can reduce the frequency and severity of hemolysis.	May have side effects or interact with other medications.
Gene therapy	Experimental approach that involves inserting a healthy copy of the G6PD gene into the patient's cells to produce functional enzyme.	Potentially curative and could offer lifelong protection against hemolysis.	Still in early stages of development and not widely available.

It is important for individuals with G6PD deficiency to work closely with their healthcare provider to develop a personalized treatment plan based on their individual needs and symptoms. (Beutler, 1994, Cappellini and Fiorelli, 2008, Luzzatto, 2006)

CHAPTER FOUR

4.1 Conclusion

To estimate the prevalence of G6PD deficiency in Kurdistan children, a comprehensive study should be conducted that involves collecting and analyzing blood samples from a representative sample of the population. This study should be conducted in accordance with ethical guidelines and should ensure that participants fully understand the purpose and procedures of the study before giving informed consent.

The results of such a study could inform public health strategies to prevent and manage G6PD deficiency in Kurdistan, such as targeted screening programs, public education campaigns, and access to appropriate medical treatment. It is essential to ensure that all children with G6PD deficiency receive appropriate medical care and monitoring to prevent complications and improve their quality of life.

Based on the available information as of my knowledge cutoff date of September 2021, G6PD deficiency is a genetic condition that affects the red blood cells and is common in the Kurdistan region. It is caused by mutations in the G6PD gene, which leads to a deficiency in the G6PD enzyme that is responsible for protecting red blood cells from damage.

Symptoms of G6PD deficiency can include episodes of hemolytic anemia, jaundice, dark urine, fatigue, and shortness of breath. These symptoms can be triggered by certain medications, infections, and dietary factors.

The dangers of G6PD deficiency include the risk of severe hemolytic anemia, which can be life-threatening if left untreated. In addition, individuals with G6PD deficiency may be more susceptible to certain infections, such as malaria.

Diagnosing G6PD deficiency typically involves a blood test to measure the activity of the G6PD enzyme. Treatment may involve avoiding triggers, such as certain medications, and managing symptoms with supportive care, such as blood transfusions.

There is currently no cure for G6PD deficiency, but the condition can be controlled through careful management and monitoring. This may include genetic counseling for affected individuals and their families to help them understand the inheritance pattern of the condition and the risks associated with it.

It is important for healthcare providers and families in the Kurdistan region to be aware of the prevalence of G6PD deficiency and the potential risks and challenges associated with managing the condition. Ongoing research is needed to better understand the condition and develop more effective treatments and management strategies.

4.2 Recommendation

Here are some recommendations for managing G6PD deficiency in Kurdistan children:

1. **Awareness**: Healthcare providers and families in the Kurdistan region should be aware of the prevalence of G6PD deficiency and the potential triggers that can cause symptoms.
2. **Genetic Counseling**: Affected individuals and their families should receive genetic counseling to understand the inheritance pattern of the condition and the risks associated with it.
3. **Diagnosis**: Early diagnosis is critical for managing G6PD deficiency. Healthcare providers should consider screening newborns for the condition and educating families on the symptoms to watch for.

4. **Avoiding triggers**: Children with G6PD deficiency should avoid triggers that can cause hemolysis, such as certain medications and infections. Dietary factors, such as fava beans, should also be avoided.
5. **Treatment**: Management of G6PD deficiency may involve supportive care, such as blood transfusions, and avoiding triggers. Healthcare providers should also be prepared to treat severe hemolytic anemia if it occurs.
6. **Research**: Ongoing research is needed to better understand the condition and develop more effective treatments and management strategies.

By following these recommendations, healthcare providers and families can help to manage G6PD deficiency in Kurdistan children and reduce the risk of severe complications.

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