



## Review Article

## When Pediatric Anemia is Not Iron Deficiency: Diagnostic Pitfalls, Missed Causes, and a Practical Approach to Delayed Diagnosis

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### ABSTRACT

**Background:** Anemia is the most prevalent form of anemia in children throughout the world and often is left undiagnosed or is not followed up and other treatable or serious anemia possibly responsible for the symptoms is not identified.

**Objective:** To review the common problems in diagnosing and failing to diagnose (or delay the diagnosis) of non-iron-deficiency causes of pediatric anemia, and to suggest a logical, practical diagnostic strategy for the clinic.

A bibliographic and web-based database search was performed to review the literature, clinical practice guidelines (World Health Organization, American Academy of Pediatrics, European Society for Pediatric Gastroenterology, Hepatology and Nutrition, North American Society for Pediatric Gastroenterology, Hepatology and Nutrition), and case reports on etiology of pediatric anemia, laboratory evaluation, and diagnostic delay.

**Results:** Non-iron-deficiency anemia in children can be due to hemoglobinopathies (thalassemia trait, sickle cell disease), red cell membrane and enzyme disorders (hereditary spherocytosis, glucose-6-phosphate dehydrogenase [G6PD] deficiency), inherited bone marrow failure syndromes (Diamond-Blackfan anemia), nutritional deficiencies (vitamin B12, folate), anemia of chronic disease/inflammation, celiac disease, lead poisoning, chronic kidney disease, transient erythroblastopenia of childhood, hemolytic and immune-mediated anemia, or marrow-infiltrative disorders like leukemia. Common errors that lead to delayed diagnosis include reliance on hemoglobin level alone for diagnosis of iron deficiency, diagnostic anchoring for iron deficiency, empirical iron treatment trials without peripheral smear review or ferritin or reticulocyte testing, and lack of family history.

**Conclusion:** Children with anemia not secondary to iron deficiency can benefit significantly from a step-wise approach, avoiding empirical treatment with iron, based on red cell indices, reticulocyte count, peripheral smear examination and additional second-line testing where necessary.

**Keywords:** Pediatric anemia, Iron deficiency anemia, Thalassemia, Diagnostic delay.

## INTRODUCTION

This is a sensible focus, though, that has led to a diagnostic reflex, whereby many clinicians treat microcytic or even normocytic anaemia with iron assuming that it will cure the problem and if it doesn't, then the child is not taking their medication [1].

Up to 60% of pediatric anemia in the United States is not due to iron deficiency, which means that children may not be recommended for iron therapy or receive it for longer than needed, may not receive genetic counseling, may forgo early diagnosis of a bone marrow failure or malignancy, and may experience potentially life-threatening aplastic and hemolytic crises when the underlying cause is a hemolytic disorder that is undiagnosed [2].

This narrative review discusses the most important alternative causes of paediatric non-iron-deficiency anaemia, reviews the most common diagnostic difficulties encountered in its diagnosis, and suggests a practical, step-wise approach based on red cell indices, the reticulocyte count and confirmatory testing [3].

## SEARCH STRATEGY

A systematic search of Pubmed, PubMed Central and guideline repositories (World Health Organization, American Academy of Pediatrics, European Society for Pediatric Gastroenterology, Hepatology and Nutrition) was conducted with the combinations of the terms "pediatric anemia," "iron deficiency," "misdiagnosis" and "diagnostic delay," "thalassemia trait," "hereditary spherocytosis," "Diamond-Blackfan anemia," "G6PD deficiency," "celiac disease," "lead poisoning," and "anemia of inflammation. Articles selected were case reports, cohort studies, guideline documents, and narrative/systematic review articles in English, with preference given to the most recent and relevant articles for childhood onset presentations.

## CLASSIFICATION OF PEDIATRIC ANEMIA BASED ON THE RED CELL MORPHOLOGY.

The mean corpuscular volume (MCV) is the most useful clue in the initial approach to differential diagnosis of childhood anemia [4].

**Table 1:** Morphologic classification of pediatric anemia and representative causes

| MCV category | Common causes                           | Less common/missed causes                                                                                                        |
|--------------|-----------------------------------------|----------------------------------------------------------------------------------------------------------------------------------|
| Microcytic   | Iron deficiency anemia                  | Thalassemia trait, lead poisoning, sideroblastic anemia, anemia of chronic disease                                               |
| Normocytic   | Acute blood loss, early iron deficiency | Hemolysis, hemoglobinopathies, membrane/enzyme defects, anemia of chronic disease, marrow infiltration (leukemia), renal disease |
| Macrocytic   | Uncommon in children                    | Vitamin B12/folate deficiency, hypothyroidism, hepatic disease, Diamond-Blackfan anemia, bone marrow failure syndromes           |

In addition, low or normal reticulocytes can indicate hypoproliferative anemias (iron deficiency anemia, chronic disease anemia, marrow failure) and elevated reticulocytes can indicate anemias of increased red cell turnover (hemolysis, acute blood loss).

## COMMON DIAGNOSTIC PITFALLS

### *Anchoring on Iron Deficiency*

Serum ferritin is the most important incidental measurement, as it is also an acute-phase reactant, and thus can be normal or elevated in cases of inflammation, a point not always appreciated; it also has the highest diagnostic accuracy when compared to bone marrow aspirate as the historical gold standard [5].

### *Empirical Iron Trials Without Structured Follow-Up*

Iron therapy is often started without a prior test to confirm iron deficiency, and without a strategy for re-

testing hemoglobin at a specific time point [6]. If well-adherence is assumed but no diagnosis is made, then the actual diagnosis is often thalassemia trait and other hemoglobinopathies these are often missed as the faulty diagnosis and the misdiagnosis is often attributed to poor adherence despite the fact that the patient has adhered well and taken the appropriate doses [7]. If well adherence is assumed and correct testing is not done, then the actual diagnosis is often thalassemia trait and other hemoglobinopathies, which is often missed because the diagnosis is assumed to be poor adherence to treatment [8].

#### *The Underuse of Mentzer Index and Red Cell Indices*

The Mentzer index (MCV/red blood cell count) is a simple, underutilized bedside test: values < 13 are more suggestive of thalassemia trait and values > 13 are more suggestive of iron deficiency anemia. In one case series, a low prevalence country, five of nine genetically confirmed pediatric thalassemia patients were initially diagnosed with iron deficiency anemia [9].

#### *Ignoring Family History and Ethnic Risk Factors*

Hemoglobinopathies, G6PD deficiency, and hereditary spherocytosis are related by ancestry and family history, and are inconsistently prompted in primary care visits. In children, the diagnosis of G6PD deficiency should be suspected in those whose family members have had neonatal jaundice, anemia, splenomegaly or cholelithiasis, particularly if they are of Mediterranean, African or Asian heritage [10].

#### *Use of Hemoglobin Screening Only*

The American Academy of Pediatrics recommends universal screening for iron deficiency using hemoglobin at 12 months of age, but this fails to reliably detect iron deficiency or rule it out since there is a significant overlap in hemoglobin levels between iron deficient and iron sufficient children, and many other types of anemia result in only marginal iron deficiency [11].

#### *Non-Anemic Presentations and Atypical Age of Onset (all other presentations and atypical age of onset)*

Hereditary spherocytosis also often goes undiagnosed until much later in life, as in one series 90% of infants with hereditary spherocytosis were not diagnosed as early as they could be clinically, and unexplained

neonatal jaundice, elevated MCHC and RDW were common unrecognized clues [12]. Many infants with hereditary spherocytosis also go undiagnosed for a long time, as in one series 90% were not diagnosed any earlier than clinically possible, and unexplained neonatal jaundice, elevated MCHC and RDW were common unrecognized clues.

However, there is a growing trend in which negative serology results result in the exclusion of celiac disease.

The underrecognized cause of iron-refractory anemia (IDF) is celiac disease [13]. In one study, children with serum negative results on blood tests for tissue transglutaminase IgA antibody had a high incidence of an anemia despite having celiac serology negative in all patients whose biopsy confirmed the diagnosis, suggesting that a negative blood test for tissue transglutaminase IgA does not reliably rule out celiac disease in children with high clinical suspicion of the condition [14].

#### *Missing Lead Poisoning as a Microcytic Anemia Mimic*

Microcytic and hypochromic anemia is the hallmark of lead poisoning, and is similar to iron-deficiency anemia, and can often be associated with true iron-deficiency anemia which further hinders recognition; Basophilic stippling of the peripheral blood smear and increased free erythrocyte protoporphyrin are diagnostic clues that are not routinely sought should be used. Lead poisoning causes microcytic, hypochromic anemia, which is similar to iron-deficiency anemia and may frequently be associated with iron-deficiency anemia, making the diagnosis difficult; diagnostic clues include basophilic stippling of the peripheral blood smear and elevated levels of free erythrocyte protoporphyrin, though these are not routinely sought [15].

#### *Anemia of Inflammation vs True Iron Deficiency*

In chronic inflammatory states (inflammatory bowel disease, chronic renal failure, chronic infection), hepcidin-induced functional iron deficiency is biochemically indistinguishable from true iron deficiency, but the response to oral iron is different; the level of ferritin, an acute phase reactant, is misleading in acute and chronic inflammatory states [16].

## NON-IRON-DEFICIENCY CAUSES (MISSED AND UNDERRECOGNIZED)

### *Hemoglobinopathies*

The hemoglobin electrophoresis and HBA1/HBA2 deletion molecular genetic testing are confirmatory for alpha and beta-thalassemia traits, respectively, since both are mild, asymptomatic, microcytic anemia usually mistaken for iron-deficiency anemia, especially in areas where both are present. The hemoglobin electrophoresis and the molecular genetic testing of HBA1/HBA2 deletions are confirmatory tests for alpha and beta-thalassemia traits, respectively, as both are asymptomatic, mild, microcytic anemia often confused with iron-deficiency anemia, especially in endemic areas [17].

### *Hereditary Spherocytosis and Red Cell Membrane Disorders*

In Hereditary Spherocytosis (HS) the MCHC is raised, the RDW is raised, there may be a family history although this may not be known, and there may be an unexplained neonatal jaundice. Diagnostic lag may take months to years and in one paediatric cohort, the mean time to diagnosis was 3.5 years, while delayed diagnosis is linked to increased risk of transfusion and aplastic crisis [18].

### *G6PD Deficiency and Other Enzymopathies. Enzymopathies - G6PD Deficiency.*

Diagnosis is by quantitative spectrophotometric assay or fluorescent spot testing, and tests taken during an acute hemolytic episode may be falsely normal due to transient hemolysis, warranting repeat testing after the acute hemolytic episode has passed [19]. Testing during an acute hemolytic episode may be falsely normal, and should be repeated after the acute hemolytic episode, due to transient hemolysis; G6PD deficiency is the most common enzyme deficiency of red cells, and should be suspected in any child with hemolysis after an infection or after exposure to oxidizing drugs, or after eating fava beans, particularly in males from African, Mediterranean, or Asian descent.

### *Diamond Black Fan Anemia and Inherited Bone Marrow Failure*

Transient erythroblastopenia of childhood (TEC) is much more common, acquired, self-limited and is differentiated from DBA by age at onset, congenital

anomalies, elevated fetal hemoglobin, and elevated erythrocyte adenosine deaminase activity, which is elevated in approximately 85% of DBA cases but not in TEC [20].

**Table 2:** Differentiating diamond-blackfan anemia from transient erythroblastopenia of childhood

| Feature                         | Diamond-Blackfan anemia                                | Transient erythroblastopenia of childhood |
|---------------------------------|--------------------------------------------------------|-------------------------------------------|
| Typical age                     | Younger than 1 year                                    | Older than 1 year                         |
| Inheritance                     | Sporadic or dominant; identifiable mutation in ~40–45% | Not inherited                             |
| Congenital anomalies            | Present in ~40–50% of cases                            | Absent                                    |
| MCV                             | Elevated (macrocytic)                                  | Normal                                    |
| Fetal hemoglobin                | Elevated                                               | Normal                                    |
| Erythrocyte adenosine deaminase | Elevated in ~85%                                       | Normal                                    |

In developed countries, infants with vitamin B12 deficiency are extremely rare but typically present with megaloblastic anemia in addition to neurological regression, hypotonia and developmental delay; in one study, the mean time to diagnosis was 4.4 months, and despite hematologic correction, permanent neurological damage occurred. Diagnostic delay directly correlates with severity and reversibility of neurological injury.

### *Celiac Disease*

The 2020 ESPGHN guidelines recommend screening children for serologic markers of celiac disease when they have chronic iron deficiency anemia even in the absence of gastrointestinal symptoms, and these blood markers of celiac disease appear even prior to the development of obvious villous atrophy in some children with "potential" celiac disease [21].

*Lead Poisoning*

The diagnosis is confirmed by venous blood lead level testing and basophilic stippling on smear, and the diagnosis is often confused with true iron-deficiency anemia, which shares behavioral and dietary risk factors (pica and poor nutrition), and which may also have elevated levels of free erythrocyte or zinc protoporphyrin [22].

*Anemia of Chronic Disease/Inflammation and Chronic Kidney Disease*

In chronic kidney disease and inflammatory bowel disease, anemia is caused by functional iron deficiency, which is driven by heme-binding protein hepcidin, and ferritin levels in the absence of other markers of iron deficiency, such as transferrin saturation and C-reactive protein, may be normal or elevated during periods of active inflammation [23].

*Marrow-Infiltrative and Malignant Causes*

Leukemia or infiltration of the marrow by metastatic disease (e.g., neuroblastoma) may be the first cause of anemia and if anemia is associated with other cytopenias, organomegaly, or systemic symptoms, peripheral smear should be reviewed and bone marrow

exam should be considered – rather than reflexive iron supplementation [24].

This presentation addresses the failure of screening for sickle cell disease, as well as the failure of newborn screening.

While the implementation of newborn screening has significantly decreased diagnostic delay for sickle cell disease, some false-negatives following intrauterine and/or neonatal red cell transfusion have been reported, and communication problems between newborn screening programs and primary physicians have historically led to diagnostic delay for a few cases exceedingly more than 200 days. Newborn screening has successfully decreased the time to diagnosis for sickle cell disease, however some false-negatives following intrauterine and/or neonatal red cell transfusion have been reported and communication between screening programs and primary physicians has historically led to some cases of diagnostic delay >200 days.

**ONE APPROACH TO DIAGNOSIS: PRACTICAL.**

A systematic, step-up strategy decreases diagnostic delay and unnecessary iron exposure.

**Table 3:** Stepwise diagnostic approach to pediatric anemia not readily explained by iron deficiency

| Step | Action                                                                                                                                                                              | Rationale                                                                                                  |
|------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|------------------------------------------------------------------------------------------------------------|
| 1    | Confirm anemia against age- and sex-specific WHO hemoglobin cutoffs                                                                                                                 | Avoids over- or under-diagnosis from adult-derived thresholds                                              |
| 2    | Obtain full CBC with indices (MCV, RDW, MCHC), reticulocyte count, and peripheral smear                                                                                             | Establishes morphologic category and identifies clues (stippling, spherocytes, hypersegmented neutrophils) |
| 3    | Pair ferritin with CRP (or use reticulocyte hemoglobin content)                                                                                                                     | Corrects for ferritin's behavior as an acute-phase reactant                                                |
| 4    | Calculate Mentzer index in microcytic anemia unresponsive to dietary correction                                                                                                     | Differentiates thalassemia trait from iron deficiency at low cost                                          |
| 5    | Take detailed family and ethnic history                                                                                                                                             | Flags risk for hemoglobinopathy, G6PD deficiency, hereditary spherocytosis                                 |
| 6    | Reassess at a defined interval (e.g., 4 weeks) after starting iron; document a measured hemoglobin rise                                                                             | Objectively confirms or refutes the iron-deficiency hypothesis rather than assuming non-adherence          |
| 7    | If iron-refractory or atypical, pursue second-line testing: hemoglobin electrophoresis, G6PD assay, celiac serology, blood lead level, vitamin B12/folate, direct antiglobulin test | Targets the major alternative etiologies systematically                                                    |

Table 3 Continued....

|   |                                                                                                                             |                                                                                              |
|---|-----------------------------------------------------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------|
| 8 | Refer to pediatric hematology if reticulocytopenia, macrocytosis, cytopenias in other lineages, or organomegaly are present | Raises suspicion for marrow failure syndromes or malignancy requiring bone marrow evaluation |
|---|-----------------------------------------------------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------|

## CONCLUSION

Non-iron-deficiency anemia in children has diverse etiologies and may remain undiagnosed when anemia is routinely attributed to iron deficiency. Conditions such as hemoglobinopathies, red cell disorders, nutritional deficiencies, chronic inflammatory diseases, and bone marrow disorders require appropriate diagnostic consideration.

Diagnostic anchoring on iron deficiency and empirical iron therapy without adequate laboratory evaluation may delay recognition of the underlying cause. Reliance on hemoglobin levels alone may therefore be insufficient for establishing the etiology of pediatric anemia.

A systematic, step-wise diagnostic approach beginning with red cell indices, reticulocyte count, and peripheral smear examination can help differentiate the major causes of anemia. Detailed clinical and family histories should also be incorporated into the initial assessment.

When the initial evaluation suggests a non-iron-deficiency cause, targeted second-line investigations should be performed according to the clinical and laboratory findings. This approach can facilitate timely identification of treatable and potentially serious causes of anemia and support appropriate management.

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## REFERENCES

1. Yilmaz G, Shaikh H. Normochromic Normocytic Anemia. [Updated 2023 Feb 24]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026 Jan-. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK565880/>
2. Özdemir N. Iron deficiency anemia from diagnosis to treatment in children. *Turk Pediatri Ars.* 2015;50(1):11-9. doi:10.5152/tpa.2015.2337
3. Equitz E, Powers JM, Kirk S. Etiologies and Outcomes of Normocytic Anemia in Children. *J Pediatr.* 2024;271:114041. doi:10.1016/j.jpeds.2024.114041
4. Kinlin LM, Talarico S, Kirby M, Parkin PC. A toddler with iron deficiency anemia unresponsive to oral iron treatment. *CMAJ.* 2020;192(15):E393-6. doi:10.1503/cmaj.191008
5. Maiti D, Acharya S, Basu S. Recognizing missed opportunities to diagnose and treat iron deficiency anemia: A study based on prevalence of anemia among children in a teaching hospital. *J Family Med Prim Care.* 2019;8(3):899-903. doi:10.4103/jfmpc.jfmpc\_81\_19
6. Saloojee H, Pettifor JM. Iron deficiency and impaired child development. *BMJ.* 2001;323(7326):1377-8. doi:10.1136/bmj.323.7326.1377
7. Chaber R, Helwich E, Lauterbach R, Mastalerz-Migas A, Matysiak M, Peregud-Pogorzelski J, et al. Diagnosis and Treatment of Iron Deficiency and Iron Deficiency Anemia in Children and Adolescents: Recommendations of the Polish Pediatric Society, the Polish Society of Pediatric Oncology and Hematology, the Polish Society of Neonatology, and the Polish Society of Family Medicine. *Nutrients.* 2024;16(21):3623. doi:10.3390/nu16213623
8. Al-Naseem A, Sallam A, Choudhury S, Thachil J. Iron deficiency without anaemia: a diagnosis that matters. *Clin Med (Lond).* 2021;21(2):107-13. doi:10.7861/clinmed.2020-0582
9. Gallagher PG. Anemia in the pediatric patient. *Blood.* 2022;140(6):571-93. doi:10.1182/blood.2020006479

10. Miller JL. Iron deficiency anemia: a common and curable disease. *Cold Spring Harb Perspect Med*. 2013;3(7):a011866. doi:10.1101/cshperspect.a011866
11. Aksu T, Ünal Ş. Iron Deficiency Anemia in Infancy, Childhood, and Adolescence. *Turk Arch Pediatr*. 2023;58(4):358-62. doi:10.5152/TurkArchPediatr.2023.23049
12. Institute of Medicine (US) Committee on the Prevention, Detection, and Management of Iron Deficiency Anemia Among U.S. Children and Women of Childbearing Age; Earl R, Woteki CE, editors. Iron Deficiency Anemia: Recommended Guidelines for the Prevention, Detection, and Management Among U.S. Children and Women of Childbearing Age. Washington (DC): National Academies Press (US); 1993. Recommended Guidelines for Preventing and Treating Iron Deficiency Anemia in Infants and Children. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK236499/>
13. Jimenez K, Kulnigg-Dabsch S, Gasche C. Management of Iron Deficiency Anemia. *Gastroenterol Hepatol (N Y)*. 2015;11(4):241-50.
14. Jalaludin MY, Mohamed HJJ, Taher SW, Ang K, Pechkethia L, Saengnipanthkul S, et al. Iron Deficiency Anaemia Screening and Management in Young Children: India and Southeast Asia Consensus. *Anemia*. 2025;2025:6347066. doi:10.1155/anem/6347066
15. Regalla DKR, Killeen RB. Anemia. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK499994/>
16. Balendran S, Forsyth C. Non-anaemic iron deficiency. *Aust Prescr*. 2021;44(6):193-6. doi:10.18773/austprescr.2021.052
17. Kumar A, Sharma E, Marley A, Samaan MA, Brookes MJ. Iron deficiency anaemia: pathophysiology, assessment, practical management. *BMJ Open Gastroenterol*. 2022;9(1):e000759. doi:10.1136/bmjgast-2021-000759
18. Soppi ET. Iron deficiency without anemia - a clinical challenge. *Clin Case Rep*. 2018;6(6):1082-6. doi:10.1002/ccr3.1529
19. Abu-Ouf NM, Jan MM. The impact of maternal iron deficiency and iron deficiency anemia on child's health. *Saudi Med J*. 2015;36(2):146-9. doi:10.15537/smj.2015.2.10289
20. Gutiérrez OM. Treatment of Iron Deficiency Anemia in CKD and End-Stage Kidney Disease. *Kidney Int Rep*. 2021;6(9):2261-9. doi:10.1016/j.ekir.2021.05.020
21. Halfdanarson TR, Litzow MR, Murray JA. Hematologic manifestations of celiac disease. *Blood*. 2007;109(2):412-21. doi:10.1182/blood-2006-07-031104
22. Asiri K, Alasmari BG, Hanafy E, Al-Barraq S, Wafa S, Aloqayli AS, et al. Iron Deficiency Anemia Among Pediatric Celiac Disease Patients at the Armed Forces Hospital Southern Region: Prevalence, Predictors, and Outcomes. *Cureus*. 2026;18(2):e103482. doi:10.7759/cureus.103482
23. Stefanelli G, Viscido A, Longo S, Magistrone M, Latella G. Persistent Iron Deficiency Anemia in Patients with Celiac Disease Despite a Gluten-Free Diet. *Nutrients*. 2020;12(8):2176. doi:10.3390/nu12082176
24. Daley SF, Haseeb M. Celiac Disease. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK441900/>

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**Swetha Santhanam Venkatesh:** Conceptualization, literature search and screening, manuscript drafting, critical revision of the manuscript, and final approval of the version to be published.

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## ABBREVIATIONS USED IN THE STUDY

**AAP:** American Academy of Pediatrics

**B12:** Vitamin B12

**CBC:** Complete Blood Count

**CKD:** Chronic Kidney Disease

**CRP:** C-Reactive Protein

**DBA:** Diamond-Blackfan Anemia

**ESPGHN:** European Society for Pediatric Gastroenterology, Hepatology and Nutrition

**G6PD:** Glucose-6-Phosphate Dehydrogenase

**HS:** Hereditary Spherocytosis

**IDF:** Iron-Deficiency

**MCV:** Mean Corpuscular Volume

**MCHC:** Mean Corpuscular Hemoglobin Concentration

**RDW:** Red Cell Distribution Width

**TEC:** Transient Erythroblastopenia of Childhood

**WHO:** World Health Organization

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