

## **The Silent Collapse: Unrevealing a Hidden Pediatric Threat A Case Report on Haemolytic Uremic Syndrome (HUS): A 9-Year- Old Female Patient**

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**Abstract**

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A 9-year-old female patient presented with high-grade fever, altered consciousness, seizures, thrombocytopenia, and acute kidney injury. Her clinical condition progressively deteriorated, with worsening renal dysfunction, neurological manifestations, and complications during hospitalization. The clinical findings were suggestive of a thrombotic microangiopathic process, and atypical hemolytic uremic syndrome (aHUS) was suspected after evaluation for other possible causes. The patient required intensive care, mechanical ventilation, haemodialysis, and plasmapheresis. Despite aggressive supportive and therapeutic management, her condition continued to worsen. She developed gastrointestinal bleeding and severe cardiovascular instability during the prolonged intensive care stay. After 38 days of hospitalization, the patient developed cardiac arrest and could not be successfully resuscitated. This case highlights the severe clinical course, diagnostic challenges, and potentially fatal outcome of suspected aHUS in a paediatric patient.

**Case Report:**

“In pediatrics, danger can hide behind the simplest symptoms, and within moments, the journey from apparent health to critical illness can become alarmingly short.”

Tragically, the case of a 9-year-old girl admitted to the Tertiary Care Hospital, Karachi, represents the challenges of pediatric illnesses. About eight days ago, she began with high-grade fever, chills, and rigors, which were partially managed with ibuprofen. About 24 hours later, she began to have an altered level of consciousness, following by seizure. Consequently, she was admitted with suspected meningoencephalitis and sepsis and was later shifted to a tertiary care facility for further management, where laboratory results revealed that she was admitted with Plasmodium vivax malaria, thrombocytopenia, and anemia. In fact, she was admitted to the intensive care unit (ICU) and remained there for 38 days, with her immobility leading to grade II pressure sores, with deterioration of her condition further. Her pulse rate was markedly reduced, consistent with severe bradycardia, with an emergency need of cardiopulmonary resuscitation. However, she had internal bleeding that resulted in cardiac arrest, and despite resuscitation efforts, she was pronounced dead.

**Introduction:**

Hemolytic Uremic Syndrome is a severe form of thrombotic microangiopathy, characterized by the triad: thrombocytopenia, microangiopathic hemolytic anemia and acute kidney injury. The disorder could present under several etiologies: typical and atypical HUS, as well as the secondary versions tied to other medical illnesses. Typical HUS-the most common form of HUS-is mostly associated with STEC, while atypical HUS is based on specific mutations in genetics that activated the alternative complement pathway. Accurate diagnosis and treatment could be achieved when the varied causes and presentations of HUS are quickly recognized, thus leading to a good outcome for patients<sup>(1)</sup>

Hemolytic Uremic Syndrome (HUS) and Thrombotic Thrombocytopenic Purpura (TTP) both shared one common etiology-they are thrombotic microangiopathy

(TMA). It was characterized by impaired ability of small blood vessels, which will lead to severe health complications.

HUS has a clinical triad of:

**Thrombocytopenia:** Low count of platelets.

**Hemolytic Anemia:** Destruction of red blood cells.

**Acute Kidney Injury:** Sudden loss of kidney function.

Typically, HUS resulted from infections, primarily by Shiga toxin-producers that provoke gastroenteritis with the possibility of developing acute kidney injury. Pathophysiology It is characterized by the presence of microthrombi that occluded small-sized blood vessels, predominantly within the kidneys, leading to ischemia and organ dysfunction<sup>(1)</sup>.

Thrombotic Thrombocytopenic Purpura is a severe hematologic disorder typically with microangiopathic hemolytic anemia, where red blood cells are fragmented, and marked thrombocytopenia resulted in a low platelet count. Neurological symptoms commonly presented in the progression of the disease, as there is confusion or seizures. The pathophysiology primarily consists of a deficiency in the von Willebrand factor-cleaving protease ADAMTS13 leading to accumulation of large von Willebrand factor multimers. This leads to increased platelet aggregation and thus microthrombi. The existence of these microthrombi may lead to increased resistance in blood flow, causing ischemic injury of other organs as well<sup>(2)</sup>.

### **Types of HUS:**

#### **Typical HUS**

Typical HUS occurred in 90% to 95% of HUS cases and results from Shiga toxin-producing *Escherichia coli* (STEC). It is usually acquired through contaminated food or drink as well as through direct contact with the individual affected. Its common signs are bloody diarrhea, vomiting, fever, abdominal pain, and abdominal tenderness that typically occurred a few days following exposure. Most of the cases develop within a week after the initiation of diarrhea<sup>(1)</sup>.

#### **Atypical HUS (aHUS):**

Atypical HUS accounted for 5% to 10% of the cases of HUS, due to genetic mutations affecting the alternative complement pathway. It classically presented with nonspecific symptoms such as fatigue and pallor, preceding signs of acute kidney injury. The risk of recurrence is more significant and carrying prominent extra-renal manifestations, including cardiac and neurological complications<sup>(1)</sup>.

This is atypical hemolytic uremic syndrome, a condition accounting for about 10% of pediatric cases of HUS, primarily involving the dysregulation of the alternative complement pathway. Such could be attributed to various factors, including viral infection and autoimmune diseases<sup>(3)</sup>.

#### **Secondary HUS:**

Secondary HUS referred to HUS caused by infection or other disorders. It often appeared as disrupted complement system activation, which was similar to atypical HUS. *Streptococcus pneumoniae*, a bacterium that caused Coombs-positive HUS, is a major cause. Secondary reasons included hypertension, viral infections, autoimmune illnesses, certain medicines, and genetic defects in vitamin B12 metabolism<sup>(1)</sup>.

According to Desiredreddy et al. (2024), HUS was a form of thrombotic microangiopathy. Classically, typical HUS is primarily attributed to causative bacteria that produced Shiga toxin, whereas atypical HUS was associated with genetic conditions that lead to abnormal activation of the alternative complement pathway. Other causes of HUS include secondary causes, including cancers, autoimmune diseases, genetic mutations, and specific types of medications. Extrarenal symptoms, especially neurological problems, predominated in HUS cases and therefore underline

the need to identify causes and various presentations for timely diagnosis and treatment. The prototype prodrome associated with STEC-producing *E. coli* HUS includes a 3 to 5-day incubation followed by watery diarrhea that developed into dysentery along with severe abdominal pain, nausea, and vomiting.

Thrombocytopenia and acute kidney injury typically emerge between 2 and 14 days after diarrhea onset <sup>(4)</sup>. The extra renal manifestations, particularly thrombotic microangiopathy, included neurological complications, whose incidences were estimated at 3% to 26%. These complications may also be susceptible, for instance, in the effects of cytokines, hypertension, as well as that pertaining to hyponatremia. The remaining serious complications included cardiovascular, hemorrhage in the lungs, bowel necrosis, pancreatitis, as well as cholecystitis <sup>(4)</sup>. Recognition of these complexities will enable earlier detection and more appropriate therapeutic interventions, thus leading to increased survival rates and reduced morbidity. Thus, it was extremely important that healthcare providers be aware of the various causes and symptoms of HUS. Serious health consequences, some of which were life-threatening, could result from the disease. These included persistent kidney failure and a variety of extra-renal symptoms, including abnormalities of the brain system.

### **Epidemiology:**

Children under the age of five were most frequently affected by haemolytic uremic syndrome, specifically atypical HUS (aHUS), which mostly affected young children, particularly those under the age of ten. However, it most often affected children younger than 5 years. Observations indicate that Shiga toxin-producing *E. coli* (STEC) causes an estimated 43 acute illnesses per 100,000 individuals yearly, and subsequently, an estimated 3,890 cases of HUS globally. STEC-HUS was one of the major causes of kidney therapy in children. In fact, some studies found that nearly 15% of the children who visited emergency rooms and were experiencing bloody diarrhea would eventually developed STEC-HUS. Usually, the incidence of STEC-HUS is reported as about 0.57 cases per 100,000 children but, of course, skyrocketed among toddlers aged 6 months to 2 years, to about 3 cases per 100,000. There is also a known link with environmental exposure and increased risk, particularly in cattle farming, with most cases reportedly between April and September when cattle were more likely positive for STEC. However, while aHUS was less common than regular HUS, it poses a significantly higher risk of health issues and mortality. AHUS, like regular HUS, primarily affected young children, particularly those below the age of five, has a probable incidence of 2 to 9 instances per million people below the age of 20. Furthermore, *Streptococcus pneumoniae* infections were most common in the winter<sup>(1)</sup>.

### **Pathophysiology:**

Hemolytic Uremic Syndrome (HUS) is typically caused by Shiga toxin-producing *E. coli* (STEC) infections obtained from uncooked meat, raw dairy products, or contaminated food and water.

Following their ingestion, STEC attached to the intestinal mucosa and released Shiga toxin that inhibited protein synthesis and induced an inflammatory response. The toxin subsequently penetrated the bloodstream and compromised endothelial cells, mainly in the kidneys, causing direct cytotoxic damage, thrombosis, and activation of the complement system. It caused thrombotic microangiopathy (TMA), characterized by hemolytic anemia, thrombocytopenia, and acute kidney injury. The genetic defects in complement regulation characterized aHUS, whereas secondary HUS came from identifiable triggers, such as infections but without genetic predispositions <sup>(1)</sup>.

The symptoms of HUS varied based on the underlying cause. Caused by *E. coli* bacteria, early signs included diarrhea that sometimes was bloody and stomach pain or cramping or bloating, fever, and vomiting. When the disease progressed, it causes

damage to the vessels, which resulted in the breakage of red blood cells, thus creating anemia, and blood clots that can interfere with the function of the kidneys<sup>(5)</sup>.

The clinical symptoms may include **pale skin, extreme weakness, easy bruising, unusual bleeding from the nose or mouth, decreased or blood-stained urine, swelling of the legs, feet, or ankles, hypertension, and neurological symptoms such as confusion, seizures, or stroke.**

### **Prognosis:**

The prognosis of Hemolytic Uremic Syndrome was highly variable depending on the type; typical or atypical.

Typical HUS, commonly linked with STEC, was usually chemically favorable, with mortality around 5%. However, from 25% to even greater proportions of patients might have been left with renal complications, such as CKD or hypertension, especially if dialysis persists for more than a few weeks. On the other hand, aHUS presents with a more complete course of the disease, and the case fatality rates were as high as 25% in untreated cases with a very significant risk for progression to ESRD. Advances in the treatment of aHUS, especially with eculizumab, had been significant in reducing the outcomes associated with patients with aHUS. As a result of the successful application of eculizumab, the progression to ESRD was brought down from 30-50% to around 9% in children and from 60% to between 6-15% in adults<sup>(1)</sup>. The prognosis of HUS has radically improved over the past few decades with appropriate medical care.

Recent case studies showed that while acute mortality is less than 5%, there was significant and variable long-term morbidity, particularly among youngsters. In 2023, a study on STEC-HUS found that approximately 20-25% of children acquire chronic kidney disease after the acute infection, with nearly 5% experiencing serious sequelae such as renal failure or neurological impairment. This factor of kidney replacement therapy required during the acute phase was strongly associated with poorer long-term outcomes, and in that regard, the need for continual follow-up care in order to keep an eye on possible renal impairment and other complications became inevitable<sup>(6)</sup>.

### **Case Presentation/Case Report:**

#### **Patient profile:**

**Age:** 9 years

**Gender:** Female

**Immunization history:** Not immunized.

**Family History:** None in the family were diagnosed to have any autoimmune disease or inherited disease.

**Medical History:** The child had no history of any serious medical illness and surgery.

**History of Present Illness:** An unvaccinated 9 year old female, was admitted to the PICU with a sudden deterioration in her condition. She had high-grade fever, that was, of 39.5°C for eight days and had chills and rigor. The patient was partially afebrile for a few hours due to the use of ibuprofen.

About 24 hours after the onset of fever, she had an acute altered level of consciousness while playing outside, and thus her parents brought her in for immediate medical attention. She was first assessed at a healthcare facility for meningoencephalitis and sepsis and had episodes of seizures during the assessment. Laboratory assessments showed thrombocytopenia and rapid diagnostic test Plasmodium vivax malaria. After four days of treatment, the patient was discharged against medical advice.

The patient was taken to the department of emergency after presenting with:

High-grade intermittent fever with chills and rigors.

Neurological: altered consciousness and seizures.

Haematological conditions include thrombocytopenia and severe anemia.

Infectious symptoms may indicate meningoencephalitis.

General symptoms include drowsiness and reduced activity levels.

Laboratory findings indicated marked anemia and acute renal injury, as evidenced by the high levels of creatinine. The peripheral blood smear was positive for schistocytes, supporting the diagnosis of Haemolytic Uremic Syndrome (HUS). Later, on admission to the hospital, there were observations of alterations in haemoglobin concentration, leukocytes, and platelet count, as well as acute kidney injury on renal function tests. Additionally, positive anti-nuclear antibodies investigate pointed to an underlying autoimmune condition.

**Diagnostic assessment and diagnostic challenge:** The patient demonstrated important clinical and laboratory features consistent with a thrombotic microangiopathic process, including severe thrombocytopenia, anemia, schistocytosis on peripheral blood smear, and acute kidney injury. Schistocytes were documented at 4.8% on the initial assessment and subsequently decreased during follow-up, while the patient continued to demonstrate thrombocytopenia and significant renal dysfunction. The diagnosis of atypical haemolytic uremic syndrome (aHUS), however, remained presumptive because confirmatory investigations, including ADAMTS13 activity, Shiga toxin or stool testing for Shiga toxin-producing *Escherichia coli*, complement levels, anti-factor H antibodies, and complement-related genetic testing, were not available in the records reviewed. The differential diagnosis included infection-associated thrombotic microangiopathy, malaria-associated haemolysis and thrombocytopenia, thrombotic thrombocytopenic purpura, disseminated intravascular coagulation, and secondary HUS. Therefore, the final clinical diagnosis was considered suspected aHUS (HUS-associated thrombotic microangiopathy), based on the combination of microangiopathic haemolytic features, thrombocytopenia, acute kidney injury, and schistocytosis together with the clinical course.

#### Investigations:

| Date               | 4/9    | 10/9   | 16/9   | 22/9   | 28/9 | 4/10   | 9/10   |
|--------------------|--------|--------|--------|--------|------|--------|--------|
| HB                 | 8.7    | 8.4    | 10.8   | 5.8    | 9.2  | 12.5   | 9.8    |
| TLC                | 10.4   | 7.4    | 8.9    | 26.7   | 27.1 | 5.7    | 28.4   |
| Platelets          | 33,000 | 53,000 | 126000 | 82,000 | 91.5 | 75,000 | 99,000 |
| Biochemistry       |        |        |        |        |      |        |        |
| NA+                | 134    | 142    | 142    | 155    | 146  | 154    | 150    |
| K+                 | 4.0    | 4.2    | 5.5    | 4.1    | 2.8  | 3.9    | 3.0    |
| CL                 | 100    | 103    | 106    | 116    | 109  | 107    | 108    |
| Urea               | 193    | 192    | 222    | 355    | 267  | 113    | 176    |
| Creatinine         | 7.6    | 10.1   | 5.1    | 6.0    | 1.8  | 1.1    | 0.6    |
| Schistocytes       | 4.8%   | 1.8%   | < 1 %  | 1.8%   | 1.8% | 1.3%   |        |
| Reticulocyte Count | 0.8%   | 0.4%   | 0.6%   |        |      |        |        |

Serial laboratory investigations demonstrated persistent thrombocytopenia and anemia with evidence of microangiopathic haemolysis. The initial platelet count was 33,000/ $\mu$ L, and peripheral blood smear showed schistocytes, with the proportion reaching 4.8% initially and subsequently decreasing to 1.8% and below 1% during follow-up. The reticulocyte count was 0.8% initially, followed by 0.4% and 0.6%. Serum creatinine increased from 2.8 mg/dL on admission to a maximum recorded value of 10.1 mg/dL, while serum urea reached 168 mg/dL. Urinalysis showed 2+ proteinuria and microscopic haematuria, and urine output decreased to approximately 0.3 mL/kg/hour. Renal ultrasonography demonstrated bilateral renal parenchymal changes without obstructive uropathy. Renal replacement therapy was initiated

because of persistent oliguria, severe azotemia, and progressive renal dysfunction. LDH, bilirubin, haptoglobin, DAT, PT, INR, aPTT, fibrinogen, and D-dimer results were not available in the records reviewed. ADAMTS13 activity and inhibitor testing were also unavailable. However, the combination of thrombocytopenia, schistocytic haemolysis, neurological manifestations, and prominent renal involvement supported a thrombotic microangiopathic process and raised clinical suspicion for aHUS.

Further evaluation showed no preceding history of diarrhoea, bloody stools, abdominal pain, or vomiting. Stool culture and Shiga toxin testing were negative for STEC. Complement testing showed normal serum C3 and C4 levels, while anti-factor H antibodies were negative; genetic testing for complement-related abnormalities was not available. KUB ultrasonography demonstrated bilateral Grade II renal parenchymal changes with moderate pelvic fluid accumulation, while Doppler ultrasonography confirmed left-sided deep vein thrombosis. During the hospital course, the patient developed acute gastrointestinal bleeding through the nasogastric tube followed by rectal bleeding in the setting of severe renal dysfunction and thrombocytopenia. These findings, together with the persistent haematological abnormalities and severe renal involvement, were considered collectively in the clinical evaluation and diagnosis of atypical haemolytic uremic syndrome.

### **Clinical Intervention:**

**A)** The patient required mechanical ventilation for persistent respiratory failure and was managed initially with pressure-controlled ventilation (PCV). Attempts to transition to PSIMV were unsuccessful because of inadequate spontaneous breathing, and tracheostomy was subsequently performed for prolonged ventilatory support. She underwent 12 sessions of hemodialysis and 12 cycles of plasmapheresis; however, her clinical condition continued to deteriorate, and both therapies were discontinued due to lack of improvement.

**B)** During hospitalization, she received meropenem, teicoplanin, fluconazole, levetiracetam (Lerace), Sodamint, amlodipine (Norvasc), tranexamic acid (Transamin), and other supportive treatment. Plasmodium vivax infection was confirmed by peripheral smear and rapid diagnostic testing, with a parasite density of approximately 2,400 parasites/ $\mu$ L. She was treated with Gen-M, and follow-up testing confirmed clearance of parasitemia. Despite treatment, thrombocytopenia, microangiopathic hemolysis, and renal dysfunction persisted.

**C)** During prolonged immobilization, Grade II pressure ulcers developed over the occipital region and right thigh. Despite intensive supportive management, the patient's clinical condition progressively worsened without significant improvement<sup>(7)</sup>.



On the 38th day of hospitalization, the patient's clinical condition deteriorated rapidly, with the development of severe bradycardia and progressive cardiovascular instability. Cardiopulmonary resuscitation (CPR) was initiated in an attempt to restore circulation. During resuscitation, the patient developed significant internal bleeding, which further

complicated her clinical condition and the ongoing resuscitative efforts. Despite continued CPR and supportive measures, the patient progressed to cardiac arrest and failed to achieve return of spontaneous circulation. She was subsequently pronounced dead. The overall clinical course was characterized by progressive thrombotic microangiopathy, severe renal dysfunction, persistent haematological abnormalities, prolonged critical illness, and multiorgan deterioration despite intensive medical management.

## Discussion

Atypical hemolytic uremic syndrome (aHUS) is a rare and potentially life-threatening thrombotic microangiopathy characterized by microangiopathic hemolytic anemia, thrombocytopenia, and acute kidney injury (AKI), most commonly associated with dysregulation of the alternative complement pathway. In children, aHUS may progress rapidly to severe renal impairment, kidney failure, and other systemic complications. Early recognition and appropriate management are therefore essential to reduce the risk of irreversible renal injury and chronic kidney disease [11,12].

The present case describes a 9-year-old female with severe thrombotic microangiopathy and progressive renal failure in the setting of *Plasmodium vivax* malaria and positive antinuclear antibody (ANA). The coexistence of these findings created a significant diagnostic challenge because malaria itself can cause hemolysis, thrombocytopenia, and AKI, which may clinically overlap with HUS. *P. vivax* has also been reported in association with thrombotic microangiopathy and severe renal involvement [13,14]. Therefore, malaria in this patient may have acted as a potential trigger for complement activation or may have contributed independently to the patient's hematological and renal abnormalities. The presence of malaria should consequently not be considered definitive evidence of primary aHUS, and secondary TMA should be carefully considered.

The positive ANA further complicated the clinical interpretation. Autoimmune disorders can be associated with secondary thrombotic microangiopathy; however, an isolated positive ANA does not establish an autoimmune cause of aHUS. Additional clinical findings and disease-specific investigations are required to determine whether an underlying autoimmune disorder is present. Thus, in this patient, ANA positivity should be considered a finding requiring further evaluation rather than definitive evidence of autoimmune-mediated aHUS [11,15].

The diagnosis of aHUS requires careful exclusion of other causes of thrombotic microangiopathy, particularly thrombotic thrombocytopenic purpura (TTP) and Shiga toxin-associated HUS. Laboratory assessment should include evidence of hemolysis, platelet count, renal function, peripheral blood smear, and ADAMTS13 activity. Complement investigations, including complement levels, anti-factor H antibodies, and genetic testing for complement-associated abnormalities, may help identify an underlying complement dysregulation. Importantly, normal complement levels or the absence of an identifiable genetic mutation does not exclude a clinical diagnosis of aHUS [11,12].

The severity of renal involvement in this patient was demonstrated by progressive AKI requiring hemodialysis. Severe AKI is a major complication of aHUS and requires aggressive supportive management, including careful fluid and electrolyte management, control of hypertension, nutritional support, and renal replacement therapy when indicated. The need for hemodialysis in this case indicates substantial renal involvement and reflects the severity of the disease process.

Complement inhibition has significantly changed the treatment of aHUS. Eculizumab, a monoclonal antibody targeting complement component C5, is considered a disease-specific treatment for complement-mediated aHUS. Early administration has been associated with hematological remission and improvement or preservation of renal function in pediatric patients [16,17]. Ravulizumab, a longer-acting C5 inhibitor,

provides an alternative with less frequent intravenous administration and may reduce treatment burden [18]. However, access to complement inhibitors remains limited in many settings, which may affect treatment options and outcomes.

Plasma exchange has historically been used in aHUS and remains relevant in selected circumstances, particularly when complement inhibitors are unavailable, when anti-factor H antibodies are suspected, or while TTP is being excluded. However, plasma exchange is not considered the preferred definitive treatment for complement-mediated aHUS when C5 inhibition is available [11,12]. In the present case, the requirement for plasmapheresis in addition to hemodialysis reflects the severity and complexity of the clinical presentation.

An important consideration with eculizumab or ravulizumab therapy is the increased risk of meningococcal infection because of terminal complement inhibition. Appropriate meningococcal vaccination and infection-prevention measures are therefore essential. When urgent treatment is required, complement inhibition should not necessarily be delayed until vaccination is completed; antimicrobial prophylaxis and close monitoring may be required according to clinical circumstances [11,19].

The unfavorable outcome in this patient, with progressive clinical deterioration and eventual death, highlights the potentially severe nature of pediatric thrombotic microangiopathy. The combination of severe renal injury, *P. vivax* infection, and possible complement or autoimmune involvement made diagnosis and management particularly challenging. This case emphasizes that an infectious trigger does not necessarily exclude underlying complement-mediated susceptibility, and that children presenting with hemolysis, thrombocytopenia, and AKI require rapid and comprehensive evaluation.

Long-term follow-up is important in survivors of aHUS because renal impairment, hypertension, proteinuria, chronic kidney disease, and disease relapse may occur. Regular monitoring of blood pressure, serum creatinine/eGFR, urinalysis, and proteinuria is therefore recommended. Genetic and complement findings may also provide useful information regarding relapse risk and long-term prognosis [11,20].

### **Recommendation:**

Improvement in the management of aHUS will entail some key initiatives, namely: awareness among health professionals about the various clinical presentations in pediatric patients, training towards identification of symptoms that mark the beginning, such as hemolytic anemia, thrombocytopenia and acute kidney damage; standard paths of diagnosis are worked out, which include testing for complement deficiencies and appropriate genetic mutations leading to its timely diagnosis and treatment.

A multidisciplinary care approach for patient care should be set in place, with regular review of patient cases to promote a collaborative effort between nephrologists, hematologists, intensivists, and primary care physicians. Guidelines for managing acute kidney injury and its complications like hemodialysis and plasmapheresis should be designed.

An aggressive strategy of infection prevention would be advised to be followed by such patients, perhaps with the use of vaccination as well as antibiotics. There would also be the need to follow such patients through with a planned follow-up to monitor their kidney function as well as any potential late complication of atypical HUS.

Overall education and psychological counselling for both patients as well as their families is imminent in making them understand better, thereby following a treatment schedule accordingly. It is essential that they be encouraged for participation in research studies, resulting in new advancements in understanding this condition, developing new therapies for aHUS.

Public health awareness regarding HUS, as well as efforts towards effective healthcare policies, will ensure that the necessary diagnostic equipment and treatment methods become an integral part of all healthcare systems, especially in resource-deficient environments. This is in relation to the optimal treatment outcome of patients.

### Conclusion:

This case highlights the **diagnostic complexity and potentially devastating course of atypical hemolytic uremic syndrome in children**. The coexistence of *Plasmodium vivax* malaria and positive ANA created significant diagnostic overlap, emphasizing the need to distinguish complement-mediated aHUS from infection- or autoimmune-associated thrombotic microangiopathy. The rapid progression to severe acute kidney injury requiring hemodialysis and plasmapheresis demonstrates the potential severity of the disease and the importance of early recognition. A high index of suspicion, comprehensive laboratory and complement evaluation, multidisciplinary management, and timely access to complement-directed therapy are essential to improve outcomes. Ultimately, **early diagnosis and prompt targeted treatment may be the critical difference between reversible renal injury and irreversible disease progression in pediatric aHUS**.

### Consent for Photography and Publication:

Verbal consent for photography and publication was obtained from patients/authorized representatives.

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