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ACUTE KIDNEY INJURY OF THE HUS-LIKE SYNDROME TYPE ON THE BACKGROUND OF SARS-COV-2 IN CHILDREN

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Since the beginning of the pandemic, SARS-CoV-2 has mutated into different variants. In 2025, new sublines of the Omicron variant appeared – “Stratus” and “Nimbus”, which are characterized by the ability to evade existing population immunity. Clinical course with predominant damage to the upper respiratory tract, gastrointestinal tract, nervous system, acute kidney injury. Objective: to analyze the course of COVID-19 coronavirus infection in children, complicated by kidney damage in the form of hemolytic-uremic syndrome. Two clinical cases of hemolytic-uremic syndrome in children with COVID-19 coronavirus infection (subvariants Omicron, 2025) are presented. Conclusions: a feature of the course of COVID-19 was kidney damage in the absence of clinically pronounced respiratory syndrome and with relatively prolonged viremia. Kidney damage occurred in the form of hemolytic-uremic syndrome with the presence of hemolytic anemia in combination with thrombocytopenia and the development of acute renal failure.

Key words: coronavirus infection, hemolytic uremic syndrome, respiratory failure, multisystem inflammatory syndrome, thrombotic thrombocytopenic microangiopathy, complement-associated atypical hemolytic uremic syndrome.

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ГОСТРЕ УРАЖЕННЯ НИРОК ПО ТИПУ ГУС-ПОДІБНОГО СИНДРОМУ НА ТЛІ SARS-COV-2 У ДІТЕЙ

З початку пандемії SARS-CoV-2 видозмінювався у різні варіанти. У 2025 році появилися нові сублінії варіанту Omicron – «Stratus» і «Nimbus», які характеризуються здатністю уникати наявного популяційного імунітету. Клінічний перебіг з переважним ураженням верхніх дихальних шляхів, шлунково-кишкового тракту, нервової системи, гострим пошкодженням нирок. Мета: проаналізувати перебіг коронавірусної інфекції COVID-19 у дітей, яка ускладнилась ураженням нирок за типом гемолітико-уремічного синдрому. Наведено два клінічні випадки розвитку гемолітико-уремічного синдрому у дітей на фоні коронавірусної інфекції Covid-19 (субваріанти Omicron, 2025 рік). Висновки: особливістю перебігу COVID-19 було ураження нирок на фоні відсутності клінічно вираженого респіраторного синдрому та з відносно тривалою вірусемією. Ураження нирок перебігало по типу гемолітико-уремічного синдрому із наявністю гемолітичної анемії в поєднанні з тромбоцитопенією і розвитком гострої ниркової недостатності.

Ключові слова: коронавірусна інфекція, гемолітико-уремічний синдром, дихальна недостатність, мультисистемний запальний синдром, тромботична тромбоцитопенічна мікроангіопатія, комплемент-асоційований атиповий гемолітико-уремічний синдром.

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Since the onset of the pandemic, SARS-CoV-2 has evolved into a wide range of variants characterized by varying degrees of severity and polymorphism of clinical manifestations and complications [18]. Depending on the extent of genomic variation, some variants have acquired the ability to spread rapidly and become more virulent, causing a severe disease course and an unfavorable prognosis. They show low sensitivity to current vaccines and immunotherapy against COVID-19 [10]. Five strains of SARS-CoV-2 are known: Alpha, Beta, Gamma, Delta, and Omicron [13, 19]. The latter strain carries the greatest number of point mutations and marked tropism for human ACE2 receptors, which allows it to penetrate human cells more efficiently and results in higher transmissibility [10].

Recently, new hybrid variants of the Omicron strain of SARS-CoV-2 have been described – XD, XF, and XE. The XE variant is a recombinant of Omicron BA.1 and BA.2; the XD variant results from recombination of Delta and BA.1 (Omicron);

XF is a hybrid of BA.2 (Omicron) and the British Delta strain [10, 13]. To assess their level of risk, variants are divided into three categories: variants under monitoring, variants of interest, and variants of concern. The latter comprise the VUM, VOL, and VOC categories. It is the VOC variants that require continuous monitoring. The Omicron strain and its sublineages (BA.1, BA.2, BA.3, BA.4, and BA.5) are characterized by a marked degree of immune evasion, which contributes to their increased spread [3, 4, 13]. Over these years, the emergent SARS-CoV-2 virus has undergone changes that, in terms of epidemiological and clinical characteristics, bring it closer to ordinary respiratory viruses [4, 8].

A number of studies have examined the efficacy of antiviral therapy for different SARS-CoV-2 strains in relation to disease severity. In particular, patients infected with the Omicron strain had a 61 % lower risk of death and a 56.7 % lower risk of hospitalization and oxygen dependence [14].

The clinical presentation of SARS-CoV-2 infection is characterized by fever (in 80 %), cough (in 40 %), sore throat (in 30 %), and difficulty in nasal breathing with rhinorrhea (in 20 %). Cases involving the gastrointestinal tract, nervous system, and lower respiratory tract with the development of pneumonia, respiratory failure, and kidney failure, as well as shock, have also been described [2, 11]. Rapid secondary bacterial infection and severe electrolyte disturbances contribute to increased mortality [17].

SARS-CoV-2 infection is characterized by the development of a specific pathophysiological phenomenon – post-COVID syndrome (long COVID) – which is marked by clinical polymorphism ranging from pronounced, protracted asthenic syndrome to multisystem inflammatory syndrome (MIS-C) [1, 7].

Kidney injury occupies a special place in the clinical manifestations of SARS-CoV-2 infection. This is explained by the morphology of the nephron and the renal calyceal-pelvic system, the high expression of the ACE2 protein in renal tubular cells – which increases viral tropism for the nephron vascular endothelium – as well as hemodynamic status and the activity of the inflammatory process [2, 6]. Kidney injury in children with SARS-CoV-2 occurs in 13.9 % of cases and is associated with an increased mortality rate [5, 6].

The purpose of the study was to analyze the course of COVID-19 coronavirus infection in children complicated by kidney injury of the hemolytic-uremic syndrome type.

Materials and methods. This publication presents two cases of COVID-19 coronavirus infection in children complicated by kidney injury of the HUS-like syndrome type. The children were treated at the Municipal Enterprise “Khmelnyskyi Infectious Diseases Hospital” of the Khmelnytskyi City Council (ME “KIDH” KCC) and at the Municipal Non-Commercial Enterprise “Khmelnyskyi Regional Children’s Hospital” of the Khmelnytskyi Regional Military Administration (MNE “KRCH” KRMA), under the continuous supervision of staff from the Department of Pediatrics, Obstetrics, and Gynecology of the Faculty of Postgraduate Education of Vinnytsia National Pirogov Memorial Medical University, Ukraine.

For the diagnosis and differential diagnosis of COVID-19 infection and differential diagnosis of the type of kidney injury, a range of laboratory and instrumental investigation methods were used. Laboratory methods included: clinical, biochemical, and immunological tests including the Coombs test, nephelometry for haptoglobin, a colorimetric method for fibrinogen, coagulation tests, bacteriological culture, enzyme immunoassay for D-dimer and IgM/IgG antibodies to SARS-CoV-2, immunochromatography for procalcitonin, and polymerase chain reaction (PCR) for SARS-CoV-2 RNA detection. Instrumental methods: ultrasound examination of the chest, abdominal organs, and kidneys, and chest X-ray.

Glomerular filtration rate was assessed using the Schwartz formula, based on serum creatinine level (in

μmol/L), the child’s height, and a coefficient dependent on the child’s age and sex. Acute kidney injury was diagnosed according to the international KDIGO (Kidney Disease: Improving Global Outcomes) criteria, based on serum creatinine level and urine output.

Inclusion criteria: children with PCR-confirmed SARS-CoV-2 infection presenting the triad characteristic of HUS – hemolytic anemia, thrombocytopenia, and acute kidney injury.

Exclusion criteria: Shiga toxin-associated hemolytic-uremic syndrome, thrombotic thrombocytopenic purpura (TTP), COVID-19-associated multisystem inflammatory syndrome (MIS-C), and sepsis-associated thrombotic thrombocytopenic purpura (TTP).

The study was conducted in accordance with the principles of the Declaration of Helsinki. Written informed consent for the publication of clinical data (full names and telephone numbers of the children’s mothers) was obtained from the legal representatives. The Ethics Committee of Vinnytsia National Pirogov Memorial Medical University approved the publication of the clinical cases (Protocol No. 4 dated 18.03.2026).

Results of the study. Clinical Case 1. A 14-year-old girl was admitted to the intensive care unit of ME “KIDH” KCC in September 2025 with complaints of fever (39.0 °C), nausea, vomiting, loose stools, and jaundice. According to the history, the illness began with fever, general weakness, and nausea. She received outpatient antipyretics and antiemetics; however, on the third day of illness the urine turned sharply darker, becoming almost black, diuresis decreased, and jaundice and a rash appeared, prompting hospitalization. On admission, a rapid COVID-19 test was performed and was positive. On the second day, polymerase chain reaction (PCR) testing detected SARS-CoV-2 RNA.

On clinical examination, the child’s condition was assessed as severe: she was lethargic, with icteric discoloration of the skin and sclera and a petechial rash on the trunk. Auscultation of the chest revealed no pulmonary pathology, while cardiac auscultation revealed bradycardia, with a pulse rate of 52–55 beats per minute, blood pressure of 145/90 mmHg, and StO₂ of 97–99 %. The abdomen was soft and non-tender on palpation, with no hepatosplenomegaly. In the morning, markedly dark urine was noted on voiding (Fig. 1).

On the first day after admission, urine output was 300 mL per day; on the second day, with diuretic stimulation, 650 mL was obtained; from the sixth day of hospitalization, diuresis recovered without stimulation.

Laboratory testing revealed normochromic anemia that progressed over five days (erythrocytes $2.3 \times 10^{12}/L$, hemoglobin 76g/L, color index 0.96), together with a moderately elevated reticulocyte count (22–26‰). In addition, occasional schistocytes – fragments of damaged erythrocytes – were found on blood smear.



Fig. 1. Color of the child's urine at admission.

The urine was turbid and brown in color. Urinalysis showed proteinuria of up to 1.7-6.0 g/L, microscopic erythrocyturia, and high specific gravity. Urine color normalized on the sixth day after admission.

However, on the seventh day of hospitalization, the renal concentrating function began to decline, as indicated by a glomerular filtration rate of 75 mL/min/1.73 m². Table 1 presents the results of the biochemical investigations. The table data show that, on admission, the child had elevated transaminase levels with moderate bilirubinemia and a moderate increase in urea (normal range in children: 1.8–6.4 mmol/L) and creatinine (normal range for this age: 18–62 μmol/L), which normalized by the eighth day of hospitalization.

Table 1

Biochemical blood analysis over time

Date	Bilirubin, μmol/L			Protein, g/L	AST	ALT	Urea	Creatinine
	Total	Direct	Indirect					
07.09.25	55.3	-	55.3	Hemolysis	394	93	9.4	55
08.09.25	49.0	11.0	38.0	57.0	175	42	14.2	117
09.09.25	36.0	11.0	25.0	63.0	350	40	12.0	110
11.09.25	23.7	-	23.7	75.0	124	70	12.0	113
14.09.25	15.0	-	15.0	73.0	44	39	7.5	83

Note: ALT, AST – mmol/L; urea – mmol/L; creatinine – μmol/L.

The direct Coombs test was negative. CRP was negative, procalcitonin (PCT) was 0.57 ng/mL (normal), blood electrolytes: potassium 4.7 mmol/L, sodium 130 mmol/L (mild hyponatremia), chloride 103 mmol/L. The coagulogram, including soluble fibrin monomer complexes, was within normal limits, D-dimer 0.8 μg/mL. Haptoglobin was 0.06 g/L (normal range 0.08–1.70 g/L for this age), consistent with intravascular hemolysis of erythrocytes. Enzyme immunoassay (ELISA) testing showed no markers of hepatitis A, B, or C. SARS-CoV-2 RNA was detected by PCR at admission and up to day 15 from disease onset. No laboratory markers of leptospirosis or intestinal infections were found.

Ultrasound examination (US) of the thoracic and abdominal organs revealed a mild enlargement of both kidneys.

Body temperature normalized on the second day of hospital treatment, which subsequently allowed systemic autoinflammatory conditions such as MIS-C, hemophagocytic syndrome, and macrophage activation syndrome to be excluded; normal dynamics of CRP and procalcitonin allowed sepsis to be ruled out.

Elevated indirect bilirubin levels together with decreased erythrocyte and hemoglobin counts, moderate reticulocytosis, and dark urine color provided grounds to suspect hemolysis.

At the same time, the concurrent decrease in platelet count, the appearance of occasional schistocytes on blood smear, the absence of coagulation abnormalities, rising creatinine and urea, decreased haptoglobin, and transient oliguria supported acute kidney injury of the HUS-like syndrome type associated with SARS-CoV-2.

Given the hepatic and renal injury, it was decided not to administer remdesivir. The child received pathogenetic and symptomatic treatment: oral rehydration, infusion therapy, diuretics, enterosorption, and antibacterial therapy. As the girl's condition worsened, she was given dexamethasone 4 mg four times daily on the first day, followed by prednisolone 30 mg twice daily, intravenous immunoglobulin at a dose of 1 g/kg per course, emetone, and enalapril. A single transfusion of packed red blood cells was performed.

On the sixth day, the child's condition improved: she became more active, appetite returned, jaundice and rash regressed, and spontaneous diuresis resumed without stimulation. After obtaining a negative SARS-CoV-2 test (12 days after admission), she was transferred to MNE "KRCH" KRMA, where she remained for a further 15 days. Examination revealed isosthenuria and a decreased glomerular filtration rate (62 mL/min/1.73 m²), suggesting the development of tubulointerstitial nephritis. She was discharged with a recommendation to continue enalapril and undergo quarterly assessment of glomerular filtration rate. No ultrasound signs of an inflammatory process in the kidneys were found at discharge, and Doppler findings were within the age norm.

Clinical Case 2. A 5-year-old girl was admitted to the anesthesiology and intensive care unit of ME "KIDH" in September 2025 with maternal complaints of fever up to 40.0 °C, lethargy, irritability, vomiting, nasal congestion, anorexia, decreased urination, and darkening of the urine. Admission diagnosis: SARS-CoV-2 infection, toxic nephritis.

According to the history, the child had been ill for 4 days, with fever, nausea, vomiting, mild rhinitis,

and general weakness. On examination by a pediatrician, a rapid COVID-19 test was performed and was positive. She received outpatient treatment, but her condition did not improve; diuresis decreased and the urine darkened sharply, leading to hospitalization. On admission, the condition was severe, attributable to an intoxication syndrome. SpO₂ was 96 %, respiratory rate 22/min, pulse 102/min, blood pressure 85/50 mmHg. Soft-tissue pastiness, subicteric sclerae, fine petechiae on the buttocks, hyperemic pharynx, and clear tonsils were noted. Cardiac and pulmonary auscultation revealed no pathology; palpation revealed hepatomegaly of up to 3 cm. At admission, voiding revealed dark brown urine, raising suspicion of hemolytic kidney injury, HUS, and disseminated intravascular coagulation (DIC) syndrome, potentially induced by SARS-CoV-2. A series of laboratory investigations was performed. The results of the complete blood count showed normochromic anemia (hemoglobin 73–63 g/L, erythrocytes $2.3\text{--}2.4 \times 10^{12}/\text{L}$), thrombocytopenia ($87\text{--}74 \times 10^9/\text{L}$), which changed to thrombocytosis on

recovery, lymphopenia, and occasional schistocytes noted on blood smear on two occasions.

Diuresis was stimulated (due to oliguria) and recovered by the 5th day of hospitalization. Brown urine color and proteinuria persisted for 5 days from admission, while urine microscopy revealed no pathological abnormalities, ruling out nephritis.

Urine ketone bodies were persistently elevated, and diastase levels reached high values of 2927–1700 U/L, indicating the development of acute pancreatitis.

Table 2 shows the dynamics of the biochemical investigations. Elevated urea and creatinine levels persisted for 5 days, transaminases for 4 days, and bilirubin was only mildly elevated for a few days.

The coagulogram showed no abnormalities, ruling out DIC syndrome. D-dimer was 3.46 µg/mL. Haptoglobin (single measurement) was 0.05 g/L (normal range 0.08–1.55 g/L for this age). PCT level was 2.85 ng/mL, which normalized after two days (0.55 g/L); CRP was 24 mg/L; lactate dehydrogenase (LDH) was 1811 U/L.

Table 2

Biochemical blood analysis over time

Date	Bilirubin, µmol/L			Protein, g/L	AST	ALT	Urea	Creatinine
	Total	Direct	Indirect					
13.09.25	26.0	-	26.0	67	289	42	14.1	110
15.09.25	23.0	-	23.0	-	143	33	15.4	114
17.09.25	14.0	-	14.0	61.2	63	28	11.8	83
19.09.25	9.0	-	9.0	52.0	38	40	6.0	62
23.09.25	12.0	-	12.0	58.0	38.5	34	7.0	64

Note: ALT, AST – mmol/L; urea – mmol/L; creatinine – µmol/L.

Markers of viral hepatitis A, B, and C were negative. SARS-CoV-2 RNA was detected for 12 days. No laboratory markers of leptospirosis or yersiniosis were found. Bacteriological examination of throat and stool samples revealed no pathogenic flora.

Ultrasound of the abdominal organs and kidneys revealed mild hepatosplenomegaly and echographic signs of a pronounced inflammatory process in the kidneys. Chest X-ray revealed no pathology.

Based on the disease history, complaints, clinical findings (oliguria), and laboratory results (normochromic anemia, moderate reticulocytosis, presence of schistocytes, thrombocytopenia, decreased haptoglobin, and elevated creatinine, transaminases, and diastase), the child was diagnosed with SARS-CoV-2 infection complicated by pancreatitis, hepatitis, acute kidney injury of the HUS-like syndrome variant, and acetonemic syndrome.

The girl received the following treatment: infusion therapy with normal saline and 10 % glucose (adjusted for diuresis and perspiration losses), 4 % sodium bicarbonate, a single transfusion of packed red blood cells (washed erythrocytes), diuretics, glucocorticosteroids (prednisolone 40 mg/day for 5 days), antibacterial therapy, rivaroxaban, protease and proton pump inhibitors, enterosorbents, antiemetics, and hepatoprotectors.

The child's general condition improved on the seventh day of hospitalization. After obtaining a

negative PCR result for SARS-CoV-2 (on day 12), the child was transferred to the nephrology department of MNE “KRCH” KRMA, where she remained for 2 weeks. A marked decrease in glomerular filtration rate ($44 \text{ mL/min/1.73 m}^2$) was found, with an increase before discharge ($62 \text{ mL/min/1.73 m}^2$; normal range for this age $100\text{--}120 \text{ mL/min/1.73 m}^2$). No ultrasound signs of an inflammatory process in the kidneys were found at discharge. Blood pressure stabilized. She was also discharged with a recommendation to continue enalapril and undergo quarterly nephrologist follow-up with assessment of glomerular filtration rate.

Discussion. Acute kidney injury (AKI) is known to be a common complication in patients with COVID-19; however, precise epidemiological data on its prevalence and incidence are lacking. Several authors report that AKI is associated with an increased risk of mortality in COVID-19 patients [3, 9].

The mechanisms of kidney injury in SARS-CoV-2 infection are diverse. According to various authors, the main causes of AKI include hypovolemia, hemodynamic disturbances, the virus itself – which is capable of damaging vascular endothelium and renal tubules – microthrombotic vascular injury, as well as the development of glomerulonephritis and rhabdomyolysis [8].

Another study describes kidney injury in COVID-19 as the development of collapsing glomerulopathy, which may occur as a complication

of COVID-19. The authors emphasize that the development of collapsing glomerulopathy is linked to the APOL1 genotype, which is common in individuals of African descent and serves as a marker of high susceptibility to this mechanism of kidney injury after viral infections. However, the researchers did not detect the SARS-CoV-2 virus itself in renal tissue, suggesting that collapsing glomerulopathy results from a cytokine storm or a hyperimmune response to the virus rather than direct infection of renal cells by the virus [8, 16].

Another mechanism of kidney damage is the development of thrombotic microangiopathies, which include the following four conditions: HUS, thrombotic thrombocytopenic purpura (TTP), disseminated intravascular coagulation syndrome (DIC syndrome), and aHUS (atypical HUS) [15].

HUS is always a severe complication characterized by the simultaneous development of three features: destruction of erythrocytes (hemolytic anemia), low platelet count (thrombocytopenia), and the development of acute kidney injury. This syndrome has several possible mechanisms of development, but most often (in approximately 90 % of cases) it develops after an intestinal infection caused by Shiga toxin-producing *E. coli* (STEC-HUS); it is less commonly associated with pneumococcus, and atypical HUS, linked to genetic defects of the complement system and other hereditary metabolic defects, is even rarer [15, 17, 19, 20].

In the described clinical cases, both children with SARS-CoV-2 infection had hepatic and renal injury, and one child also had pancreatitis. The kidney injury resembled HUS, as evidenced by the development of hemolytic anemia (normochromic erythrocytes, moderately elevated bilirubin and reticulocytes), thrombocytopenia, elevated urea and creatinine (acute kidney injury), a moderate decrease in haptoglobin, the presence of schistocytes on complete blood count, oliguria, and dark brown urine with proteinuria.

Autoimmune thrombocytopenic purpura is typically characterized by mucosal bleeding, petechiae and ecchymoses on the skin, and a positive direct Coombs test [15, 17], none of which were present in the described clinical cases.

Atypical HUS results from dysregulation of the alternative complement pathway. It is classified as a

rare disease with a severe course and a high mortality rate. Its activation can be triggered by any external factor, including viral infection. Clinically, atypical complement-mediated HUS is characterized by a triad of symptoms: non-immune hemolytic anemia, thrombocytopenia, and signs of organ damage of varying severity, with progression of renal failure that can be fatal. Since the introduction of biological therapy (eculizumab) for aHUS, the prognosis has improved considerably [15]. In the present clinical cases, the anemia, thrombocytopenia, and renal failure did not reach critical levels and resolved rapidly.

Differential diagnosis was performed with autoinflammatory syndromes such as COVID-19-associated MIS-C, hemophagocytic syndrome, and macrophage activation syndrome. The absence of hepatosplenic syndrome, prolonged fever, specific skin involvement (particularly of the palms, soles, oropharyngeal mucosa, and conjunctivae), cardiac and coronary vessel involvement, coagulopathy, and the absence of rising procalcitonin, CRP, and fibrinogen levels helped exclude these conditions.

The absence of multiple organ dysfunction syndrome and coagulopathy, together with normal PCT and CRP levels, excludes sepsis-associated thrombotic thrombocytopenic microangiopathy [15].

Antipyretics could have had an adverse effect on both the liver and kidneys; however, fever in the children was short-lived and responded well to paracetamol and ibuprofen, so there was no indication to increase the dose or frequency of administration.

Limitations. Acute kidney injury associated with COVID-19 coronavirus infection has several possible mechanisms of development. The described clinical cases show kidney injury resembling hemolytic-uremic syndrome; however, the absence of complement system studies and molecular genetic testing did not allow complement-mediated atypical HUS to be excluded. Nevertheless, as researchers studying HUS report, this hereditary variant typically has a severe and protracted course, often with extrarenal manifestations, namely involvement of the central nervous system (CNS) and the cardio-respiratory system (CRS) [20, 30]; however, in the presented clinical cases it resolved rapidly, with no symptoms of CNS or CRS involvement.

Conclusion

The SARS-CoV-2 coronavirus, which caused a pandemic lasting nearly five years, is now regarded as a seasonal virus; however, cases of severe disease or clinically pronounced post-COVID syndrome continue to be reported. This infection predominantly affects the respiratory tract, but it may also manifest as injury to the kidneys, liver, pancreas, or heart in the absence of a pronounced respiratory syndrome, as reflected in this article. Therefore, children with acute kidney injury should be tested for SARS-CoV-2. Indeed, a distinctive feature of the course of coronavirus infection COVID-19 in these cases was injury to the kidneys, liver, and pancreas against a background of the absence of a clinically pronounced respiratory syndrome, together with prolonged viremia, which should be regarded as an unfavorable factor for the development of various organ complications.

Several mechanisms of kidney injury in SARS-CoV-2 infection exist; however, in these cases the coronavirus may have acted as a trigger for kidney injury through the development of thrombotic

microangiopathy with clinical features resembling hemolytic-uremic syndrome. These children should undergo planned follow-up testing for hereditary causes and complement-mediated atypical hemolytic-uremic syndrome.

These cases demonstrate the diverse pathophysiological pathways of kidney injury in SARS-CoV-2 infection, including the HUS type, for the diagnosis and differential diagnosis of which a complete blood count should be examined to detect hemolytic anemia with reticulocytes and schistocytes, thrombocytopenia, haptoglobin level, and renal failure, together with evaluation of the complement system and molecular genetic testing.

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