Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

need HD, was treated with eculizumab and had partial recovery of renal function.

Conclusions: In conclusion, different types of TMA may occur during the course of COVID-19, including aHUS. COVID-19 likely represents a second hit of primary aHUS that manifests in genetically predisposed individuals (e.g., those with an underlying complement risk factor). Early identification of COVID-19-associated primary aHUS is needed in order to promptly start treatment with eculizumab.

No conflict of interest

POS-012

SCLERODERMA RENAL CRISIS AS A TRIGGER FOR ATYPICAL HEMOLYTIC UREMIC SYNDROME: A RARE CASE

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Introduction: Atypical hemolytic uremic syndrome (aHUS) may remain undiagnosed in patients with normal renal function. Precipitating factors for aHUS vary widely and may include infections, drugs, autoimmune conditions, transplants, pregnancy, and metabolic conditions. Scleroderma renal crisis can also act as a trigger for aHUS via sharing of common complement activation pathways. We here present a case of scleroderma renal crisis triggering underlying aHUS.

Methods: We searched our electronic database to identify the details of the case. aHUS was diagnosed with anti-factor H level antibodies and genetic analysis for exome sequencing. AFH levels >100 AU/ml were considered diagnostic for aHUS.

Results: A 26-year-old male was diagnosed with chronic kidney disease in the year 2018. In March 2021, he was referred to us for hypertension, nausea, generalized weakness, and reduced appetite. His serum creatinine level was 19.27 mg/dl for which he was advised hospitalization and immediate dialysis initiation. His evaluation revealed positive results with significantly elevated levels of anti-nuclear antibody and anti-centromere B antibody. After 3 months of the first visit, his blood pressure remained uncontrolled despite being treated with 4 antihypertensives. With a strong clinical suspicion of aHUS, we assessed AFH levels that were highly elevated (419.5 AU/ml). Genetic analysis revealed heterozygous deletion of CFH (exon 17), duplication of CFHR1 (exon 5,6 and intron 1,3) as well as CFHR3 (exon 1,2,3,6 and intron 4). His mother, the prospective donor for renal transplant, also showed the same genetic mutation. He has been continued on maintenance hemodialysis. At 12 months follow-up, he is now planned for transplant and is under work-up.

Conclusions: In a young, hypertensive patient with end-stage renal disease, a scleroderma renal crisis can be a trigger for aHUS. Both scleroderma renal crisis and aHUS can have similar clinical manifestations. It is important to ascertain the exact cause of renal dysfunction before renal transplant as aHUS has a high rate of recurrence in a renal allograft.

No conflict of interest

POS-013

THROMBOTIC MICROANGIOPATHY AFTER COVID-19: LACK OF EVIDENCE OF COMPLEMENT ACTIVATION? A CASE REPORT

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Introduction: Evidence regarding thrombotic microangiopathy related to covid-19 is reported in the literature, particularly in severe cases. We describe a case recovered from previous asymptomatic covid-19, presenting with acute renal failure, hemolytic anemia, and low platelets. Thrombotic microangiopathy (TMA) was confirmed by renal biopsy, without immunofluorescence staining for C3c and C1q, suggesting this case is not complement-mediated. Anticoagulant therapy led to kidney function improvement.

Methods: Case report.

Results: A 72-year-old women with a past medical history of primary hypertension was referred to the hospital for the diagnosis of acute renal failure. Three days prior to admission, she suffered abdominal pain, decreased urine output, her blood test revealed elevated serum creatinine of 393 umol/L, and low platelets of 43.6 K/uL. She denied history of hematologic or renal disorders, yet mentioned that she found asymptomatic covid-19 one month before admission. On admission, the vital signs was significant for a blood pressure of 140/80 mmHg. Physical examination was noted with facial oedema, upper abdominal pain, otherwise unremarkable. Laboratory test confirmed acute renal failure with the ongoing increase of serum urea 30.4 mmol/L and creatinine 575 umol/L. Her total blood count discovered thrombocytopenia and anemia, with the platelet count of 50 k/uL, and the hemoglobin of 94 g/L. Lactate dehydrogenase was in upper limit of 434 U/L, and the bilirubin level was in normal range. The peripheral blood smear showed "fragmented" RBCs. Coombs' test was negative for both direct and indirect method. Stool examination failed to detect either red or white blood cell. Haptoglobin level was 1.14 g/L, which was in normal range (0.41-2.58 g/L). Ddimer was elevated 1376 ng/mL, and the fibrinogen 6.37 g/L. Immunology investigation was conducted with the result of normal level for both complement C3 and C4, negative reaction for anti-cardiolipin IgM and IgG, anti MPO, anti PR3, RF, anti-streptolysin O. Bone marrow aspiration did not show abnormalities. There were Forrest III gastric ulcers found by gastric endoscopy (two ulcers with diameter of 9mm and 10mm, with pseudo-membrane covered). Initially she was treated symptomatically with amlodipin, intravenous PPI, and IV furosemide. As the kidney function was getting worse, hemodialysis was initiated at day 1, day 3, day 6, and day 10 of admission. Renal biopsy was performed and showed active thrombotic microangiopathy. Given the normal complement profile, and negative C3c staining on immunofluorescence of renal biopsy investigation, complement mediated TMA may not be the pathogenesis of this case. The patient was started for anticoagulant therapy, initially subcutaneous low molecular weight heparin and then oral anti-vitamin K. She obtained dramatic recovery with dialysis off, increased urine output, normalized platelets and red cell count, and serum urea and creatinine back to nearly normal range at discharge.

Conclusions: Complement related thrombotic microangiopathy is a rare and severe condition. This case of TMA after covid-19 reveals a non-complement mediated pathogenesis, with different treatment. Anticoagulation is an effective therapy in hypercoagulation induced TMA.

No conflict of interest

POS-014

USE OF ECULIZUMAB FOR ATYPICAL HEMOLYTIC UREMIC SYNDROME AND SECONDARY THROMBOTIC MICROANGIOPATHY: CASE SERIES

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Introduction: Eculizumab is a humanized monoclonal antibody that prevents the formation of the terminal complement complex. It is associated with hematologic and renal recovery along with cessation of immunological damage in the TMA patient. Eculizumab has been approved by the regulatory authorities for the treatment of aHUS in both, native and transplanted kidneys. However, there is not such approval for patients with secondary TMA and use of eculizumab is based solely on a limited number of case reports.

Methods: This is a single-center report of three patients who received eculizumab either for sTMA (n=1) or aHUS (n=2). The diagnosis of HUS was made by the presence of microangiopathic hemolytic anemia, thrombocytopenia, and acute kidney injury (AKI). ADAMTS13 level was checked for TTP exclusion. All patients failed to respond to standard treatment. Mutations in genes encoding complement factors were checked using PCR. Data on demographic and clinical characteristics, treatment modalities before eculizumab therapy were collected. Patients were followed for 3-12 weeks.

Results: **Case 1:** a 26-year-old female was admitted to the hospital due to leukopenia, anemia, thrombocytopenia, and AKI. Anuria and azotemia progressed, therefore hemodialysis was initiated. Arthralgia,