Hemolytic Uremic Syndrome Associated with Paraquat Intoxication

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Hemolytic uremic syndrome (HUS) refers to the triad of renal failure, thrombocytopenia and nonimmune microangiopathic haemolytic anemia (MAHA) demonstrated usually by red cell fragments (schistocytes). Medication-related MAHA sometimes occurred in several clinical conditions. It was well known that paraquat intoxication was very fatal and toxic to several organs. However, there is no report that paraquat is associated with HUS. We report paraquat associated HUS as first case report here. 66-year-old patient was referred to our emergency room due to paraquat intoxication. Gastric lavage was performed, activated charcoal was administrated through oral route and daily hemoperfusion was undergone for three days after admission. Initial laboratory finding showed that his hemoglobin level was 16.8 g/dL, platelet count was 299 x 10^3/mm³, blood urea nitrogen (BUN) and serum creatinine level was 22.7 mg/dL and 1.23 mg/dL. After 3rd hemoperfusion, thrombocytopenia (22 x 10^3/mm³), anemia (9.1 g/dL) and azotemia (creatinine; 4.6 mg/dL) were developed. Ten days after admission, thrombocytopenia and anemia was not recovered and azotemia was aggravated. Platelet count was 45 x 10^3/mm³, hemoglobin level was 7.4 g/dL, peripheral blood smear revealed more than 10 schistocytes (0-3/HPF), LDH level was 948 U/L and creatinine level was 13.4 mg/dL. We diagnosed him as hemolytic uremic syndrome and performed plasma exchange three times consecutively. MAHA and thrombocytopenia was improved and LDH was normalized after plasma exchange. However, his renal function was not recovered immediately. Twelve times hemodialysis was done as renal replacement therapy during his admission. Now his renal function was completely restored. We should suspect paraquat associated with HUS when thrombocytopenia and anemia maintained for a long time after paraquat intoxication.

Penicillamine-induced membranous glomerulonephritis in a patient with Wilson's disease

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Penicillamine has been used for the treatment of a variety of disease. Nephrotic syndrome is a rare complication of the treatment of Wilson's disease with penicillamine. We describe a 49-year-old female with Wilson's disease who developed nephrotic range of proteinuria 13 months after the initiation of penicillamine therapy. She was initially compatible with Wilson's disease showing low serum ceruloplasmin level, high excretion of copper in the urine (109 mg/day [15.0-60.0]), and detection of mutation of ATP7B gene. After diagnosis, 750 mg of D-penicillamine was prescribed and proteinuria of 3,750.6 mg/day developed after 13 months of therapy without no renal insufficiency. She had mild pitting edema on both legs. Serum albumin was 3.9 g/dL and cholesterol was 222 mg/dL, respectively. Renal biopsy was performed. Immunofluorescent finding showed IgG deposit along the glomerular basement membrane and electron-microscopic findings revealed subepithelial deposits, which were consistent with membranous glomerulonephritis (MGN). Penicillamine was switched to zinc (0.025 g of zinc, three times a day). Proteinuria was completely disappeared 9 months after withdrawal of penicillamine. We should keep in mind that penicillamine induce nephrotic range proteinuria and withdraw offending drug for the treatment of proteinuria.