O_26 DOI: https://doi.org/10.5114/reum/219190

Atypical hemolytic uremic syndrome secondary to adjuvant therapy with gemcitabine

Marta Maria Maksimowska[1,2], Stanisław Niemczyk[2]

[1]Faculty of Medicine, Medical University of Warsaw, Poland
[2]Clinic of Internal Medicine, Nephrology and Dialysis, Military Institute of Medicine, Warsaw, Poland

Key words: gemcytabine, aHUS, hemolytic, uremic, HUS

Introduction: Hemolytic uremic syndrome (HUS) usually presents with a triad of symptoms: thrombocytopenia, hemolytic anaemia, and acute kidney injury. Currently, HUS is differentiated into HUS associated with *Escherichia coli* infection and Shiga toxin production, atypical HUS associated with complement system disorders, and secondary HUS associated with infection or drug-induced HUS, etc. During the differential diagnosis, thrombotic thrombocytopenic purpura (TTP), associated with reduced ADAMTS13 (a disintegrin and metalloprotease with thrombospondin type 1 motifs, member 13) activity or deficiency, should always be ruled out.

Case description: A 63-year-old woman with a history of ductal carcinoma of the pancreatic tail, surgically removed in February 2023, was undergoing adjuvant chemotherapy with gemcitabine. Additionally, patient had bronchial asthma, hypertension, and hypothyroidism treated with hormone replacement therapy. The patient was admitted to the emergency department due to severe anaemia requiring red blood cell transfusion and biochemical evidence of kidney damage. On admission, the patient reported weakness, intermittent shortness of breath, and palpitations, and denied nausea, vomiting, or bleeding symptoms. Laboratory tests revealed anaemia, thrombocytopenia, and markers of renal damage. Urinalysis revealed asymptomatic bacteruria, hematuria, and a small amount of protein. Abdominal ultrasound revealed post-resection changes of the pancreatic body and tail, a 19-mm hypoechoic right adrenal nodule, right renal cysts, and a non-dilated pelvicalyceal system. During hospitalisation, progressive deterioration of renal function was observed despite conservative treatment of acute kidney injury, as well as worsening blood pressure control despite gradual intensification of antihypertensive therapy. A suspicion was raised of the development of atypical hemolytic uremic syndrome during the course of adjuvant chemotherapy with gemcitabine. The ADAMTS13 activity was determined at 80.6%, and stool polymerase chain reaction for Shiga-toxigenic *Escherichia coli* was negative. Four therapeutic plasmapheresis sessions were performed, but they were ineffective. An application for eculizumab treatment was submitted under the drug program, which was rejected.

Conclusions: This case report illustrates a rare but severe complication of aHUS associated with gemcitabine treatment. Gemcitabine should be discontinued immediately if characteristic symptoms are observed. Plasmapheresis treatment was found to be ineffective in achieving significant clinical improvement, suggesting a potential need for complement inhibitor therapy, particularly eculizumab.