



**CR61 Obstipation as a manifestation of bilateral hydronephrosis**Marina Andrešić<sup>a</sup>, Nikolina Novak<sup>a</sup>, Josipa Živko<sup>a</sup>, Maša Sorić<sup>a</sup><sup>a</sup> *Emergency Medicine Department, University Hospital Dubrava, Zagreb, Croatia*DOI: <https://doi.org/10.26800/LV-145-supl2-CR61> Marina Andrešić 0000-0002-3753-778X, Nikolina Novak 0000-0001-7416-7805, Josipa Živko 0000-0001-7297-5366, Maša Sorić 0000-0002-5002-9800**KEYWORDS:** Hydronephrosis; Ultrasound; Intestinal Obstruction**INTRODUCTION/OBJECTIVES:** Hydronephrosis is a progressive dilatation of the renal calyces due to obstruction of urine outflow. If left untreated, the disease progresses to kidney failure and death.**CASE PRESENTATION:** A 59-year-old man presented to the emergency department with a 5-day history of obstipation and bloating. He had a permanent urinary catheter and had no complaints regarding it. Vital signs were normal. Physical examination revealed abdominal distension and diffuse abdominal tenderness without peritoneal guarding. Laboratory tests revealed elevated C-reactive protein (208.6mg/L), high creatinine (639µg/L), urea (30.5mmol/L), high white blood cell count (37.8x10<sup>9</sup>/L) with normal urinalysis. Immediately, point-of-care ultrasound was performed which revealed bilateral hydronephrosis (grade IV) and dilated small bowel loops with wall thickening, keyboard sign, and “to-and-fro” peristalsis, indicating small bowel obstruction. Abdominal computed tomography (CT) without contrast confirmed nephrolithiasis and bilateral ureterolithiasis without ileus due to a widespread prostatic neoplasm. A urologist was consulted and a bilateral percutaneous nephrostomy was performed and the patient was admitted for further treatment.**CONCLUSION:** The purpose of this case report is to accentuate the importance of considering wide differential diagnoses in patients with abdominal symptoms and the benefit of point-of-care ultrasound in making the diagnoses, especially in patients not fit for contrast CT imaging.**CR62 Personalized approach to patients with statin intolerance based on pharmacogenomics (function of OATP1B1 protein)**Anja Ćuk<sup>a</sup>, Doroteja Đekić<sup>a</sup>, Dunja Leskovar<sup>b</sup>, Livija Šimičević<sup>c</sup><sup>a</sup> *School of Medicine, University of Zagreb, Zagreb, Croatia*<sup>b</sup> *Division for Metabolic Diseases, Department of Internal Medicine, University Hospital Centre Zagreb, Zagreb, Croatia*<sup>c</sup> *Division for Pharmacogenomics and Therapy Individualization, Department of Laboratory Diagnostics, University Hospital Centre Zagreb, Zagreb, Croatia*DOI: <https://doi.org/10.26800/LV-145-supl2-CR62> Anja Ćuk 0000-0002-5992-8327, Doroteja Đekić 0000-0002-2811-4398, Dunja Leskovar 0000-0002-2342-4450, Livija Šimičević 0000-0002-2491-1920**KEYWORDS:** dyslipidemia; hmg coa statins; OATP1B1 protein, human; pharmacogenomics**INTRODUCTION/OBJECTIVES:** Statins, HMG CoA reductase inhibitors, are effective at lowering blood cholesterol levels and protecting against cardiovascular incidents. Their uptake into hepatocytes depends on the activity of the SLCO1B1 gene which codes for the OATP1B1 transporter protein. Pharmacogenomic testing provides us information about the activity level of a certain protein. Lowered activity level of OATP1B1 protein can prolong the bioavailability of some drugs, such as statins. This greater systemic exposure to statins carries a higher risk of intolerance symptoms when compared to normal function of the OATP1B1 protein.**CASE PRESENTATION:** We present two patients with dyslipidemia and statin intolerance symptoms. Female, 58, with history of multiple cardiovascular incidents presented with elevated liver function tests (GGT 1048, ALP 167, AST 219, ALT 93) while on atorvastatin therapy. After removal of the statin, her LFTs improved (GGT 419, ALP 122, AST 80, ALT 98) which indicated statin-induced liver lesion. Male, 55, also with history of cardiovascular incidents, came with muscle cramping in rest and high creatine-kinase (236 U/L) while on rosuvastatin therapy. In these cases, pharmacogenomic testing was indicated and in both patients the function of SLCO1B1\*5(c.521T>C) was lowered (\*1/\*5). Due to high cardiovascular risk and detected statin intolerance, both were candidates for PCSK9-inhibitors, monoclonal antibodies that lower cholesterol.**CONCLUSION:** The example of these two patients shows us how pharmacogenomic testing can help in choosing the right therapy based on individuals' genetics.