Abstract
In this report we will present a case on a seventy-six-year-old female patient with bilateral hydronephrosis, sepsis, and insulinoma that died due to septic shock. Necropsy findings showed that the cause of bilateral hydronephrosis was the swelling of the urinary bladder mucosa that occurred due to purulent cystitis which is rare. Interestingly, no obvious predisposing factor in the patient was found. Diabetes mellitus, which is a risk factor for the development of an infection, was not found in our patient, but on the contrary, insulinoma.

INTRODUCTION
Causes of hydronephrosis can be obstructive and non-obstructive. Causes of urinary tract obstruction include kidney stone, congenital blockage, blood clot, scarring of tissue, benign tumor or cancer (bladder, cervical, colon, or prostate), pregnancy and urinary tract infection (or other diseases that cause urinary tract inflammation) [1].

CASE REPORT
A seventy-six-year-old woman with anasarca was transferred to our intensive care unit from the general hospital upon the insisting of her family. The patient suffered from arterial hypertension, permanent atrial fibrillation, ischemic cardiomyopathy, and chronic kidney disease stage III, microcytic anemia and insulinoma. Patient medical history and discharge summary were deficient therefore, based on available data; it was possible to conclude that the patient had an infection, probably of the urinary tract, with developing heart failure and sepsis with anasarca. Two days before the patient’s transport to our institution, she was given a temporary pacemaker because of symptomatic bradycardia.

Performed abdominal ultrasound showed bilateral hydronephrosis. At the time of admission her erythrocytes were 2.78 x 10¹²/L (normal range 3.96 - 5.08 x 10¹²/L), hemoglobin 70 g/L (119 - 157 g/L), MCV (mean corpuscular volume) 85.3 fL (83.0 - 97.2 fL), leukocytes 4.54 x 10⁹/L (3.4 - 9.7 x 10⁹/L), thrombocytes 46 x 10⁹/L (158 - 424 x 10⁹/L), sodium 137 mmol/L (137 - 146 mmol/L), potassium 2.7 mmol/L (3.9 - 5.1 mmol/L), calcium 1.93 mmol/L (2.14 - 2.53 mmol/L), urea 17.3 mmol/L (2.8 - 8.3 mmol/L), creatinine 135 µmol/L (49 - 90 µmol/L), albumin 28.0 g/L (39.6 - 48.4 g/L), total proteins 51.8 g/L (66 - 80 g/L) and CRP (C-reactive protein) 34.3 mg/L (<5 mg/L). Urine nitrates were negative, leukocyte esterase positive, traces of protein, urine sediment contained 6 leukocytes, 8 erythrocytes, some bacteria and fungus. First urine culture, taken on the second day of the patient’s hospital stay, was sterile, but cytological analyses of urine revealed signs of inflammation containing neutrophils, erythrocytes (91% smooth erythrocytes), fungal parts and degenerately changed epithelial cells. Patient’s diuresis was regular. During the second day of hospitalization the patient was given a temporary pacemaker because of symptomatic bradycardia.

Performing abdominal ultrasound showed bilateral hydronephrosis. She was examined by a urologist who suggested a MSCT (multislice computed tomography) urography be carried out. Past history was negative on vesicoureteral reflux, radiotherapy or abdominal surgery. MSCT urography and MSCT abdoman scan demonstrated turbid fatty tissue due to anasarca and bilateral hydronephrosis grade III/IV (Figure 1). Ureters were not reported due to multiple artefacts. It was not possible

to see inorganic lithiasis. Ascites was present. Based on these findings, which did not describe an obstruction, and given the fact the patient had sufficient diuresis, the urologist did not indicate setting a double J prosthesis. Regarding patient's severe illness and bad medical condition, the urologist did not indicate a cystoscopy as well. Hematological investigations (cytological analyses of bone marrow aspirate, leukogram) were carried out because of anaemia, thrombocytopenia did not reveal any myeloproliferative disorder and anti-thrombolytic antibodies were negative. Diazoxide, which the patient was taking in her chronic therapy, was excluded from therapy because of doubt that a part of her clinic state (heart failure, anaemia, thrombocytopenia) could be connected to adverse effects of the aforementioned drug. Despite excluding diazoxide from therapy, there was not any obvious improvement in the patient’s clinical status, and later due to hypoglycemia, diazoxide was administered again. Values of thyroid hormones and TSH (thyroid-stimulating hormone) were in correlation with non-ges 3.86 - 5.08 x 10^{-12}/L thyroidal illness (regular TSH, decreased values of free-thyroxine and free-triiodothyronine). Gynecological examination showed hyperplasia of endometrium without intraepithelial lesion.

An echocardiogram carried out showed a regular size of the left ventricle with borderline wall thickness (12mm), hypokinesis of the basal one third of the septum, ejection fraction estimated around 55%. The estimation of diastolic function was limited because of atrial fibrillation. Both atria were dilated. Tricuspid regurgitation with systolic pressure in pulmonary artery that estimated around 60mmHg. Mild mitral valve regurgitation was present and a small amount of pericardial effusion without repercussion on hemodynamics.

In the following treatment period the patient developed septic shock with multiple organ failure and was mechanically ventilated and hemodialized. Taking serial roentgenograms bilateral infiltrates were noticed. Tracheal aspirates were continuously positive with isolated *Pseudomonas aeruginosa*, *Acinetobacter baumannii* (carbapenem resistant), *Klebsiella pneumoniae*, *Providencia rettgeri*, *Enterobacter cloacae* and *Escherichia coli*. *Candida albicans* was isolated from two samples of urine culture while *Providencia rettgeri* and *Enterococcus faecalis* were isolated from one samples of urine culture. Respiratory symptoms were dominant. The clinical condition of the patient worsened and following the patient’s death on 34th day after the admission an autopsy revealed acute purulent cystitis (Figures 2,3), acute bilateral pyelonephritis, bilateral hydronephrosis, bilateral pneumonia and serofibrinose pleuritis, anasarca, bilateral hydrothorax, hydropericard, ascites, generalised atherosclerosis, hypertrophy of the left ventricle, myocardial fibrosis and insulinoma (pT1, pN0, pM0 (TNM Classification of Malignant Tumors, in this case, tumor was only present in the pancreas without involving lymph nodes and without further metastases).

The results of post-mortem examination showed that acute purulent cystitis was the cause of hydronephrosis.

**DISCUSSION**

While researching medical literature an occurrence of bilateral hydronephrosis as a result of bladder mucosa swelling because of urinary bladder’s inflammation was noticed, but precise incidence of that occurrence is unknown and probably is very rare [1].

Toyota N et al., report a case of an emphysematous cystitis and bilateral hydronephrosis in a sixty-two-year-old female patient with poor regulated diabetes mellitus type II and recurrent urinary tract infection associated with neuropathy that led to neurogenic bladder. She was treated with antibiotic therapy in addition of urinary catheterization and symptoms as well as radiological findings resigned. The same authors claim that when they consulted with the literature they found 153 similar cases during the last fifty years [2]. Based on clinical findings and
medical data it is unlikely that neurogenic bladder is a cause of bilateral hydronephrosis in our patient.

Biswas A et al., report of an 18-year-old Caucasian male with a hemorrhagic cystitis and hydronephrosis (investigation showed that tuberculosis was the main cause) but without clear cause of obstruction [3]. As a result of ketamine abuse there are some reported cases of cystitis-like changes and decreased cystometric capacity sometimes accompanied by unilateral or bilateral hydronephrosis [4]. Sharma SK et al., reported of a case in which bilateral dilatation of the upper urinary tract in the adult male was a result of inflammatory disease, prostatism, and where the patient positively reacted to conservative therapy that indicated the possibility of these phenomena being of infective rather than of obstructive origin [5].

We presented the patient with bilateral hydronephrosis, sepsis, and insulinoma where the cause of hydronephrosis was not detected during her lifetime. The results of post-mortem examination showed that acute purulent cystitis was the cause of hydronephrosis.

Evidently, a recurrent cystitis accompanied by the swelling of the bladder mucosa led to disturbance in the urine output from ureters to urinary bladder. Ureters were not presented appropriately during MSCT urography, because of multiple artefacts and possibly because of poor and slow secretion of contrast due to already existing kidney failure.

Therefore, we have shown the case of a patient with bilateral hydronephrosis that developed as a result of purulent cystitis. Although there are such cases described in the literature, this was the first such case we came across. Interestingly, we did not find any obvious predisposing factor in the patient. Urine isolates (apart from Candida) were treated with antibiotics according to the antibiogram, and the clinical picture was dominated by signs of lung inflammation. At the time of the diagnosis of the advanced bilateral hydronephrosis, the patient had a normal diuresis. Diabetes mellitus, which is a risk factor for the development of an infection, was not present in our patient but, on the contrary, insulinoma. Also, we believe that diazoxide and other drugs in chronic therapy probably did not have any effect on the aforementioned illness.

Learning points: We have shown a rare example of bilateral hydronephrosis caused by purulent cystitis. As mentioned before, we did not find any obvious predisposing factor in the patient. Diabetes mellitus, which is a risk factor for the development of an infection, was not present in our patient but, on the contrary, insulinoma.

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REFERENCES