DIFFERENTIAL DIAGNOSIS OF CHRONIC KIDNEY DISEASE PROGRESSION IN A PATIENT WITH PSORIASIS AND XANTHOGRANULOMATOUS PYELONEPHRITIS

Diagnostyka różnicowa progresji przewlekłej choroby nerek u pacjentki z łuszczycą i żółtakoziarniniakowym odmiedniczkowym zapaleniem nerki

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Abstract

Chronic kidney disease refers to a group of disorders characterised by lasting abnormalities in kidney structure or function – persisting for over three months – and associated with conditions of various aetiologies. The classification of chronic kidney disease is based on two fundamental categories. The first one, marked as G, refers to the glomerular filtration rate, usually estimated using the CKD-EPI formula. The second, marked as A, relates to the amount of albumin excreted in the urine. Identifying the underlying causes of the disorders is essential, as targeted treatment can slow the natural progression of the disease and postpone the onset of the final, fifth stage – end-stage kidney disease – which necessitates the initiation of renal replacement therapy. The paper presents a case of a 50-year-old female patient with chronic kidney disease at stage G5 A3, with multiple comorbidities including suboptimally treated psoriasis, gout, recurrent urinary tract infections, and changes characteristic of xanthogranulomatous pyelonephritis of the right kidney, along with a history of chronic hydronephrosis.

Streszczenie

Przewlekła choroba nerek to zespół nieprawidłowości w zakresie czynności lub budowy nerek, utrzymujących się powyżej 3 miesięcy, występujących w jednostkach chorobowych o różnej etiologii. Klasyfikacja przewlekłej choroby nerek opiera się na dwóch podstawowych kategoriach. Pierwsza z nich – G – odnosi się do wartości przesączania kłębuszkowego, przy czym zwykle posługuje się jego szacowaną wartością, wyliczoną za pomocą wzoru CKD-EPI. Druga wartość –A – dotyczy ilości albuminy wydalanej z moczem. Ustalenie etiologii zaburzeń jest ważne, ponieważ ukierunkowane leczenie może spowolnić naturalny postęp choroby i opóźnić wejście w ostatnie, piąte stadium, zwane schyłkową niewydolnością nerek, które wiąże się z koniecznością wdrożenia leczenia nerkozastępczego. W pracy przedstawiono przypadek 50-letniej pacjentki z przewlekłą chorobą nerek w stadium G5 A3, z licznymi obciążeniami, między innymi nieoptymalnie leczoną łuszczycą, dną moczanową, nawracającymi zakażeniami układu moczowego oraz zmianami o charakterze żółtakoziarniniakowego odmiedniczkowego zapalenia prawej nerki i przewlekłym wodonerczem w wywiadzie.

Keywords: psoriasis; end-stage renal failure; xanthogranulomatous pyelonephritis

Słowa kluczowe: łuszczyca; schyłkowa niewydolność nerek; żółtakoziarniniakowe odmiedniczkowe zapalenie nerek

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Introduction

Chronic kidney disease (CKD) is a condition affecting many patients, very often coexisting with other disorders and contributing to multimorbidity [1]. Despite being relatively easy to diagnose, CKD is an insidious disease because it may not cause noticeable symptoms until advanced stages, and thus go undetected. The aetiology of CKD is highly diverse – ranging from primary and genetic causes to infectious factors and secondary complications of other systemic diseases, as well as more complex origins. The progression of the disease is gradual, and its severity is classified based on two main parameters. The first is a 5-stage classification denoted by the letter G. It defines the glomerular filtration rate (GFR) – though, for practical reasons, an estimated GFR (eGFR) is almost always used, calculated using the CKD-EPI (Chronic Kidney Disease Epidemiology Collaboration) formula. The second is a 3-stage classification described by the letter A, referring to albuminuria. This is assessed using either a 24-hour urine collection or the albumin-creatinine ratio in a spot urine sample. In patients with stage G5 CKD, kidney failure is diagnosed, which, due to the irreversibility of changes, is also referred to as end-stage kidney disease (ESKD). At this point, renal replacement therapy is required, which may take the form of haemodialysis, peritoneal dialysis, or kidney transplantation [2].

The following case involves a patient with multimorbidity, including several conditions that predispose to progressive kidney damage, thereby complicating differential diagnosis.

Case report

A fifty-year-old woman of Ukrainian nationality was admitted to the Department of Internal Medicine, Nephrology and Dialysis, Military Institute of Medicine – National Research Institute in Warsaw, due to progression of CKD. Her medical history included nephrolithiasis, chronic right-sided hydronephrosis, recurrent urinary tract infections, psoriasis, well-controlled type 2 diabetes mellitus previously treated with metformin, hypertension, normocytic anaemia, gout, and hypothyroidism managed with hormone replacement therapy. The patient also reported long-term tobacco use. Approximately 10 years earlier, the patient had undergone right-sided nephrolithotomy for staghorn calculi, with a past history of appendectomy. The woman presented to the Emergency Department in early January 2024 due to an elevated serum creatinine level (4.7 mg/dL), detected during outpatient tests performed as part of the preparatory work-up for a computed tomography (CT) scan. The examination was ordered by a urologist for the diagnosis of a hypoechoic lesion in the right kidney, previously visualised on ultrasound. In the previous measurement, performed more than three years earlier, the creatinine level was 1.2 mg/dl.

On admission, the patient reported dysuric symptoms, for which she had received antibiotic treatment three times in the past two months. Worsened blood pressure control and blurred vision were also observed. In addition, the patient reported an exacerbation of long-standing nausea over the past three months, now accompanied by vomiting occurring two to three times per week. In recent years, psoriasis treatment had primarily relied on topical therapies; however, their effectiveness had been modest. Previous narrowband UVB phototherapy had yielded good clinical response.

On physical examination, numerous confluent psoriatic lesions were observed on the skin of the back, abdomen, thighs, elbows, and the scalp, with sparing of the face and upper half of the trunk. The findings were consistent with a severe course of the disease. Additionally, mild oedema was observed.

Laboratory tests performed upon admission revealed the following: serum creatinine: 4.1 mg/dl (reference range: 0.5–0.9), eGFR CKD-EPI: 13 ml/min/1.73 m², blood urea nitrogen: 194 mg/dl (reference range: 15–43), uric acid: 14.8 mg/dl (reference range: 2.4–5.7). The patient also presented with metabolic acidosis: pH 7.182, with HCO_3^- concentration 15.1 mmol/l, hyperphosphataemia: 6.3 mg/dl (reference range: 2.6–4.5), mildly elevated parathyroid hormone (PTH): 79.4 pg/ml (reference range: 10–60) and erythrocyte sedimentation rate (ESR): 111–109 mm/h. Additional laboratory tests revealed elevated levels of serum amyloid A: 14.3 mg/dl (reference range: <0.64) and beta-2-microglobulin: 13.4 ug/ml (reference range: 1.09–2.53), accompanied by severe mixed dyslipidaemia (total cholesterol 298 mg/dl [<190], LDL 147 mg/dl [<116], triglycerides 456 mg/dl [<150], HDL 38 mg/dl [reference range: >45] and normocytic anaemia: haemoglobin 11 g/dl (reference range: 12–15). C-reactive protein (CRP) and procalcitonin levels remained within the normal range. On admission, urinalysis revealed proteinuria (100 mg/dL), leukocyturia, and haematuria. The urine cultures showed mixed flora. A subsequent culture was negative; however, follow-up testing demonstrated persistent leukocyturia without bacteriuria, along with haematuria. The 24-hour urine collections revealed proteinuria at the level of 2.6–3.2 g/24 h. The glycated haemoglobin level was 5.5%. Total calcium and albumin concentrations were within normal limits. The proteinogram revealed no evidence of monoclonal protein. Infection with HIV, active or past hepatitis B and C, as well as tuberculosis, was excluded. Complement components C3 and C4 were within the normal range. The reticulocyte count was also normal, with no evidence of haemolysis, iron deficiency, or vitamin B_{12} or folic acid deficiency. Antibody tests for ANA, anti-dsDNA, p- and c-ANCA, and rheumatoid factor (RF) were negative.

Echocardiographic examination revealed left ventricular hypertrophy, with no segmental wall motion abnormalities or significant valvular defects; the ejection fraction was 65%.

During hospitalisation, antihypertensive treatment was optimised, metformin was discontinued, and linagliptin was initiated. Metabolic acidosis was corrected through oral administration of bicarbonates, which led to the resolution of nausea and vomiting. In addition, an attempt was made to treat renal failure conservatively by introducing allopurinol and atorvastatin.

During hospitalisation, the patient experienced two episodes of sudden pain, swelling, and redness of the second toe on the right foot, suggestive of a gout flare. A comparative radiological and ultrasound examination of the feet revealed no signs of active inflammation. A good response to colchicine treatment was achieved, which supported the diagnosis of a gout attack. During her stay in the Department, the patient was consulted by a dermatologist, who recommended urgent initiation of UVB phototherapy. Topical psoriasis treatment was also intensified. Ophthalmological consultation revealed no signs of diabetic or hypertensive retinopathy.

In the follow-up ultrasound of the urinary tract, the right kidney showed hypertrophy of the middle renal column, measuring approximately 34×36 mm, with no possibility of excluding an isoechoic focal lesion. In the parenchyma of the right kidney, three hypoechoic lesions were visualised in the vicinity of the pyramids, without detectable vascularity, measuring: 5×15 mm in the upper pole, 12×21 mm in the middle column region, and 15×27 mm in the lower pole. The findings could be consistent with abscesses accompanied by papillary necrosis or neoplastic growth. The parenchyma of both kidneys demonstrated heterogeneous echostructure, increased echogenicity, and hyperechoic areas in the peripyramidal regions. The pelvicalyceal systems appeared slightly dilated bilaterally, with no evidence of calculi. Based on these findings, antibiotic therapy with ceftriaxone and ciprofloxacin was initiated, alongside antifungal treatment with fluconazole.

To verify the nature of the urinary tract abnormalities, after intravenous administration of fluids and acetylcysteine, a CT scan of the abdomen and pelvis was performed before and after the administration of intravenous contrast agent, including the urographic phase. The right kidney showed approximately a dozen hypodense lesions with hair-thin septations, which exhibited irregular contrast filling during the urographic phase and displayed pathological enhancement of both walls and septa in the venous phase. The pelvis and ureter of the right kidney had irregularly thickened walls. Additionally, on the right side, there were areas of increased density in the perirenal and pararenal fat tissue, as well as several enlarged paraaortic lymph nodes. The overall imaging findings raised a suspicion of xanthogranulomatous pyelonephritis of the right kidney. Selected CT scan images with annotated structures described in the text are presented in Figure 1 and Figure 2.

In subsequent phases of the study, normal contrast enhancement of both kidneys was observed, with delayed excretion of contrasted urine into the pelvicalyceal systems of both kidneys. Further passage through the undilated ureters to the urinary bladder, which had uniformly thickened walls, was undisturbed. Additionally, small fat hernias were described in the periumbilical region, with defect diameters up to 10 mm, as well as an oblique inguinal hernia on the left side with a defect diameter of 20 mm, containing normal visceral adipose tissue within the hernia sac. The patient underwent a urological consultation and was initially considered for diagnostic ureterorenoscopy or right-sided nephrectomy; however, after reassessment, outpatient observation was recommended.

Despite preserved diuresis, no significant improvement in kidney function was achieved (creatinine level was 4.3 mg/dl, eGFR CKD-EPI 12 ml/min/1.73 m², urea level 148 mg/dl). Consequently, possible kidney replacement therapy methods were discussed with the patient. She initially preferred peritoneal dialysis; however, due to the presence of hernias, uncertainty regarding the condition of the right kidney, and communication difficulties related to the language barrier, referral was issued for the creation of an arteriovenous fistula for haemodialysis. The patient was placed under

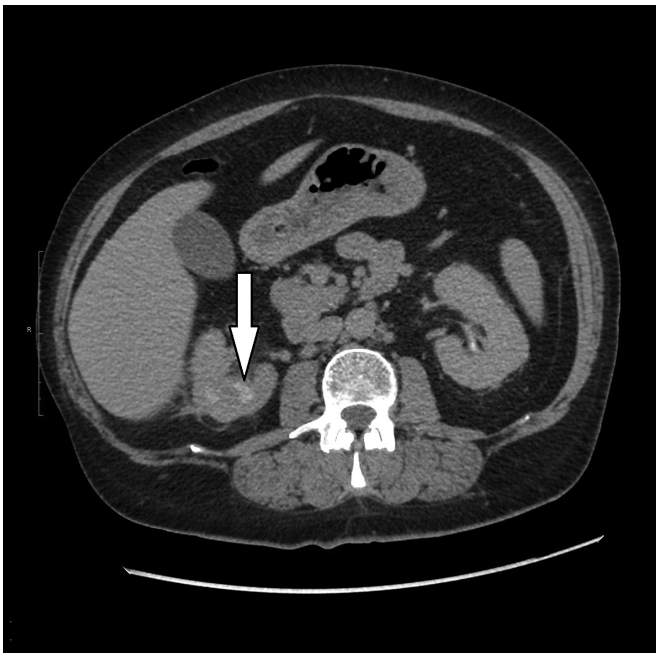

Figure 1. CT scan revealing one of the hypodense lesions in the right kidney

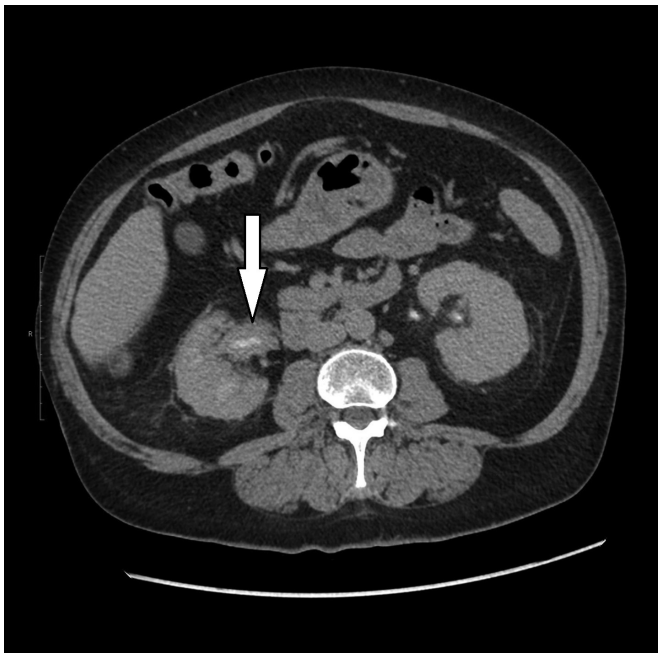

Figure 2. CT scan revealing thickened walls of the right renal pelvis

predialysis care at the Nephrology Outpatient Clinic, where she received darbepoetin alfa for the treatment of renal anaemia.

Discussion

In the described case, CKD was diagnosed at an advanced stage (G5 A3). Due to contraindications to kidney biopsy, it was not possible to determine the exact aetiology of nephron damage. Meanwhile, biopsy – enabling immunopathological evaluation of the collected material – currently constitutes the cornerstone of diagnostic assessment both in Poland and globally [3, 4].

The patient presented with multiple comorbidities, each of which could independently contribute to the development of chronic kidney disease. The most probable leading cause was determined to be glomerulonephritis in its most common form, IgA nephropathy, which may be associated with long-term, suboptimally treated psoriasis, as also indicated by the present haematuria [5, 6]. Other factors that may have contributed to the deterioration of kidney function include chronic tubulointerstitial nephritis secondary to gouty nephropathy, as well as radiologically diagnosed xanthogranulomatous pyelonephritis [7, 8]. An additional comorbidity was arterial hypertension, which, however, was well controlled, and ophthalmological examination did not reveal features of hypertensive retinopathy [9].

Another condition that could have contributed to the patient's poor condition was diabetes mellitus and diabetic nephropathy. However, the lack of retinopathy and a normal glycated haemoglobin level suggested that this aetiology could be excluded [10]. Amyloidosis was considered unlikely because, despite an elevated serum amyloid A (SAA) protein concentration, no amyloid deposits were visualised in the gingival biopsy [11].

Regardless of the aetiology, the advanced stage of kidney disease in this patient may necessitate the initiation of kidney replacement therapy in the near future. The patient initially opted for peritoneal dialysis – a method that is rarely chosen in Poland. According to 2023 data, only 766 patients used this form of treatment, accounting for approximately 4% of the just over 21,000 individuals undergoing dialysis in Poland at that time. This number decreased by nearly 50 patients compared to the previous year [12, 13].

Every form of kidney replacement therapy comes with its own specific inconveniences. For this reason, it is crucial for patients to be able to choose the therapy that best suits their expectations and lifestyle. However, contraindications to specific dialysis methods are also crucial. In the described case, the patient's umbilical and oblique inguinal hernias would complicate peritoneal dialysis. Therefore, and also due to communication difficulties related to the patient's background, a decision was made to prepare her for the initiation of haemodialysis. Based on the nephrologist's assessment, disseminated or advanced xanthogranulomatous pyelonephritis is an indication for nephrectomy [8]. However, while this procedure might expedite the initiation of haemodialysis, it would concurrently eliminate one of the sources of chronic inflammation. Moreover, histopathological examination of the removed kidney would enable assessment of the nature of the changes, including the exclusion of a tumour. This is a prerequisite for considering kidney transplantation; therefore, at present, this treatment option is unavailable to the patient. The optimal approach would be a preemptive transplant from a living related donor. This entails performing the transplantation during the predialysis stage, utilising a kidney graft from a living donor who voluntarily elects to donate one of their healthy kidneys. Most kidney transplants come from deceased donors and are implanted in individuals already undergoing dialysis while awaiting an organ. Nevertheless, such a transplant offers a more favourable prognosis than dialysis therapy [14, 15].

It should be noted that the patient has multiple comorbid conditions. Multimorbidity worsens the prognosis, which is why effective diagnosis and treatment of comorbidities, as well as the prevention of further complications, are so important [16, 17]. Predialysis nephrological care and optimisation of pharmacotherapy, including treatment of anaemia, may delay the need to initiate dialysis therapy. At the same time, coordinated care involving multiple medical specialties is essential for the effective management of the patient.

Summary

This case highlights the diagnostic complexity involved in determining the aetiology of kidney failure, which is likely multifactorial. The patient was diagnosed with glomerulonephritis – one of the most common causes of chronic kidney disease. However, the rapid progression to end-stage kidney disease was most likely driven by a combination of contributing factors including diabetes, hypertension, and gout. In patients with a history of conditions that predispose to nephron damage, it is essential to perform regular laboratory monitoring of renal function, including serum creatinine measurement with glomerular filtration rate estimation, and urinalysis with evaluation for proteinuria and urinary sediment. These tests may facilitate early detection and indicate the need for further diagnostic evaluation and more intensive management of underlying comorbidities contributing to CKD. Early disease identification and implementation of appropriate therapeutic measures may delay the need for renal replacement therapy, thereby improving both prognosis and quality of life.

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